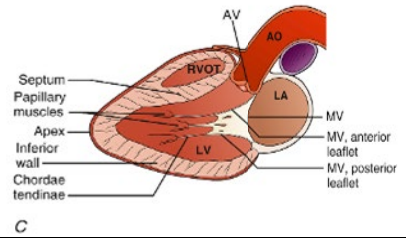
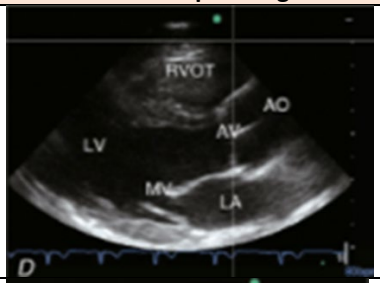
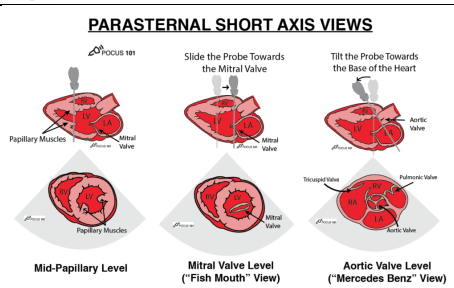
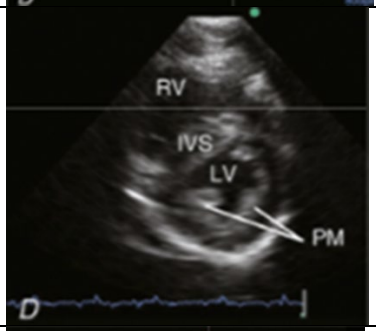
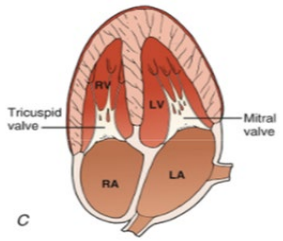
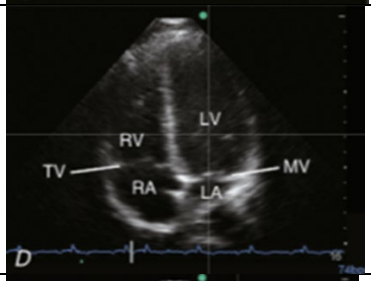
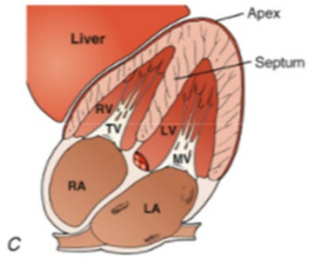
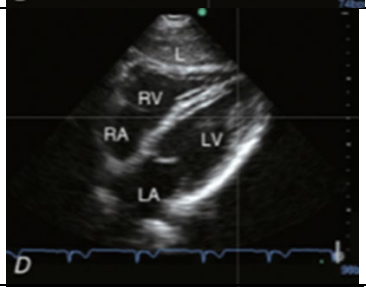
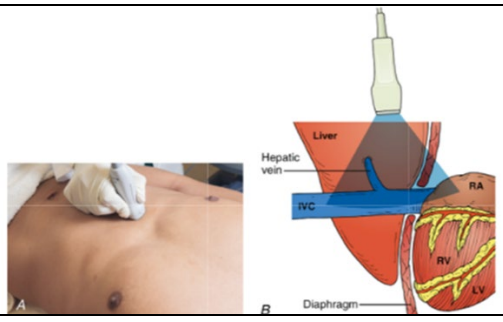
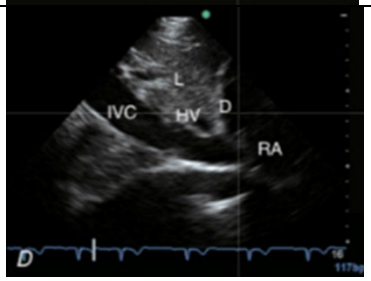
AY 2024-2025 Systems-Based Cumulative Academic Summaries

Contents

Cardiology

Cardiac POCUS

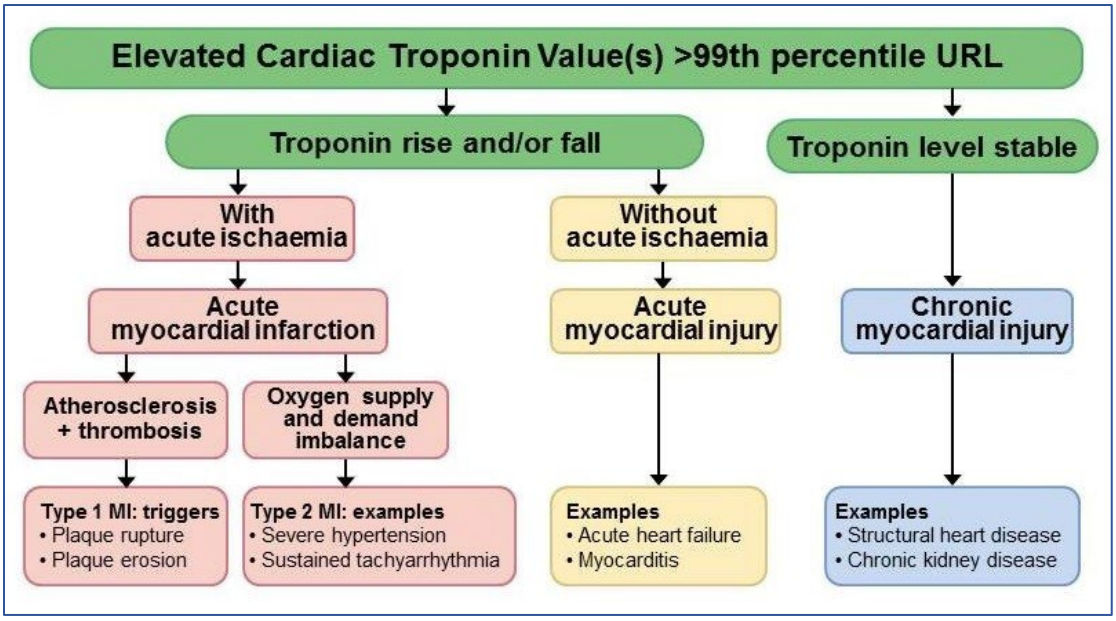
Indication	Limitation	Basic US probe maneuvers
- Evaluate/rule out life-threatening conditions quickly at bedside (Trauma, Tamponade, PTX, Shock, Resus, e.g.)	Time constraints (don't delay care for perfect views) Must understand artifacts, potential risks (thermal injury in eye POCUS, e.g.)	Slide – to move probe vertically or horizontally on pt skin Rock – probe moves along long-axis (tail moves side-to side, probe still) Fan/Tilt – probe moves along short-axis (tail moves up and down, probe still)

View	Anatomy Observed	Sample Image
1. Parasternal Long Axis (PLAX) At 3rd or 4th ICS along LSB with probe indicator towards pt R shoulder		
2. Parasternal Short Axis (PSAX) At 3rd or 4th ICS along LSB with probe indicator towards pt L shoulder (turn 90 degree clockwise from PLAX)		
3. Apical 4 Chamber (A4C) At PMI with probe indicator towards pt L shoulder to L side (decubitus positioning can be helpful!)		
4. Subcostal / subxiphoid Below xiphoid with probe indicator towards pt L side		
5. BONUS – IVC Below xiphoid with probe indicator towards head, MUST visualize IVC into RA – consider transhepatic view if subcostal poor		

Telemetry Monitoring Indications

24 Hours	48 Hours	Indefinite (until resolution or definitive intervention)
Post-pacemaker/ICD/ablation S/p AICD firing Chest pain syndrome (excluding very low-risk patients) Uncontrolled atrial tachyarrhythmia Initiation of drug known to cause Torsades de Pointes	Acute/subacute HF ACS (NSTE-ACS, STEMI) Myocarditis/pericarditis Syncope of unknown etiology Post-noncardiac surgery, high-risk Acute neurologic event (stroke/TIA) Chest pain (intermediate/high risk)	Post-cardiac arrest Temporary pacing High-grade AV block Post-cardiac surgery, high risk Arrythmias (incl LQTS & WPW) Intensive care unit New-onset bradyarrhythmia Overdose of pro-arrhythmic agent Severe HypoK or HypoMag **Consider: severe sepsis, severe EtOH w/d

Elevated Troponins



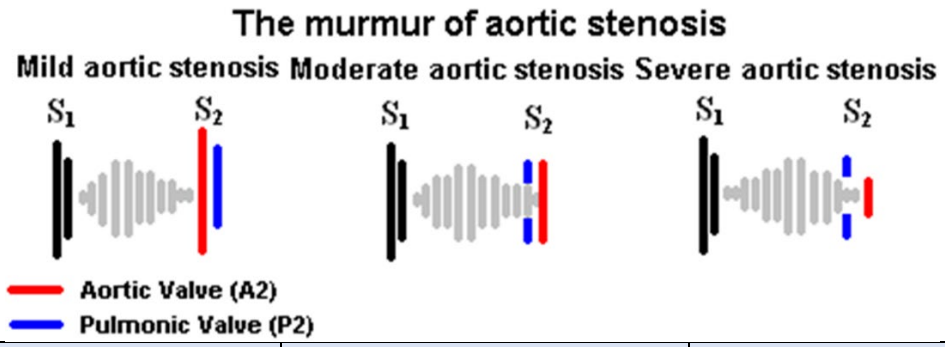
Chest Pain

	Less likely cardiac / ischemic	Higher likelihood cardiac/ ischemic
History	Sharp, fleeting, pleuritic, positional, tear, ripping Lasts for seconds	Central, pressure, squeezing, heaviness, exertional, retrosternal, radiating Lasts >5 minutes
	NSTE-ACS	**STEMI**
On EKG	Non-specific ST segment changes ST depressions / T-wave inversion (not geographic!) If non-diagnostic, repeat in 5-10 minutes or with change in pain!	Geographic distribution (at least 2 contiguous leads with >=1 mm ST elevation) - V2-V3 criteria = 1.5 mm in women, 2+ mm in men >40 yo, 2.5+ mm in men <40 yo - Reciprocal depressions expected (PAILS) New LBBB may be suggestive iso ACS symptoms (Sgarbossa Criteria useful)
Immediate Treatment	Call Cardiology Nitro SL/ODT 0.4 mg q5min x3, then gtt if persists NO NITRO for SBP<90, HR<50, suspect RV infarct ASA 325 mg SL (NOT enteric coated) Plus/minus P2Y12 per your Cards team (Clopidogrel, ticagrelor, prasugrel) Anticoagulant (Fondaparinux! Or heparin bolus then gtt OR LMWH, weight-based) Oxygen for SpO2 <90%	Activate the cath lab / STEMI system Nitro SL/ODT 0.4 mg q5min x3, then gtt if persists NO NITRO for SBP<90, HR<50, suspect RV infarct ASA 325 mg SL (NOT enteric coated) Plus/minus P2Y12 per your Cards team (Clopidogrel, ticagrelor, prasugrel) Anticoagulant (heparin bolus then gtt OR LMWH, weight-based) Oxygen for SpO2 <90%
When to cath	<u>Within <2 hours:</u> - VTach, Shock, refractory angina on max dose nitro <u>Within 24 hours of presentation</u> - Elevated Grace (140+) or TIMI (>2) scores - New ST depression - >20% in biomarkers <u>Within 72 hours</u> - Moderate risk (Grace 109-140) or (TIMI <=2)	<u>At PCI-capable hospital:</u> For STEMI within 24 hrs of symptom onset, 90 minutes from first medical contact ➔ ED to PCI <30 minutes <u>At hospital where nearest PCI is >120 min away:</u> Push tPA within 30 minutes, transfer to PCI center!
Complications	<u>Mechanical</u> LV/RV failure Ventricular wall rupture Septal rupture Papillary muscle rupture Ventricular aneurysm or pseudoaneurysm	<u>Electrical</u> VTach, VFib, AVB, Fascicular block <u>Inflammatory</u> Dressler's syndrome (pericarditis)
Follow-on management	Beta-blocker ACEi or ARB if intolerant Statin (goal LDL <70) TTE (repeat TTE at 40d for low EF. If EF is still reduced -> ICD)	

Pericarditis

<table>
<tr><th colspan="5">Pericarditis</th></tr>
<tr><td colspan="2">Historical clues</td><td colspan="2">Exam</td><td>EKG Findings</td></tr>
<tr><td colspan="2">Chest pain: sharp, pleuritic, may be sudden or insidious depending on etiology/chronicity, positional - improves with leaning forward, radiation to trapezius ridge</td><td colspan="2">Pericardial rub (often triphasic & loudest at LSB) – may be elicited with pressure on diaphragm with pt leaning forward
POCUS may demonstrate pericardial effusion</td><td>4 phases: Diffuse ST elevations with reciprocal ST depressions in aVR and V1, NOT in a specific anatomic distribution (Phase 1)</td></tr>
<tr><td colspan="5">Etiologies</td></tr>
<tr><td colspan="2">Infectious</td><td>Infarction</td><td>Iatrogenic</td><td>Other</td></tr>
<tr><td colspan="2">Viral (Coxsackie, adeno, EBV, CMV, Flu, HIV, Mumps, COVID)
Bacterial (TB – most common in developing world!, GPC, GNR)
Fungal (Histo, blasto, cocci, aspergillus)
Parasitic (Toxo)</td><td>Post-infarct (1-3d)
Dressler (wks to mos)</td><td>Post-operative/cath
Radiation-induced (Hodgkin, Breast, e.g.)
Drugs (procainamide, hydralazine, INH, AC, phenytoin)</td><td>Idiopathic (>50% in US)
Traumatic
Autoimmune (RA, SLE, Scleroderma, Sjogren)
Neoplasm (1˚ or met)
Renal failure (Uremic or chronic iHD with nl BUN)</td></tr>
<tr><td>Evaluation</td><td colspan="4">HPI – evaluate for infectious exposures, recent surgery or occult/known MI, potential autoimmune illness, meds…
Initial – EKG, CXR, Troponin, TSH, ESR/CRP, urgent TTE
(r/o tamponade, r/o other causes of chest pain / effusion; troponin may suggest myopericarditis or ACS)
Subsequent – if refractory/severe; per primary suspected etiology, may include chest CT, CMR (LGE), serologies
Diagnosis: 2 out of 4 of classic chest pain, pericardial rub, EKG changes, and/or pericardial effusion</td></tr>
<tr><td>Disposition</td><td colspan="4">Inpatient Management for High-Risk patients with any of:
- Fever, Subacute onset, Tamponade, moderate-to-large pericardial effusion (>20 mm in end-diastole), immunosuppression, warfarin/DOACs, acute trauma, failure to respond to 7d of appropriate therapy, OR elevated troponins == c/f myopericarditis
Otherwise, outpatient management ok for patients without these features (low-risk) – most patients!</td></tr>
<tr><td>Treatment</td><td colspan="4">Goals are to control pain, reduce inflammation, and prevent recurrence
Options (will vary based upon initial vs recurrent episodes):
- Exercise restriction for all!
- First-line: Aspirin OR Naproxen OR Indomethacin (1-2 weeks) PLUS colchicine (3 months)
- Second-line if NSAID contraindicated: Prednisone (1-2 weeks) PLUS colchicine (3 months)
- Third-line: IVIG OR Anakinra OR Azathioprine
- Fourth-line: Pericardiectomy
Pericardiocentesis ONLY for hemodynamically significant OR large/refractory effusions (can be diagnostic)</td></tr>
</table>

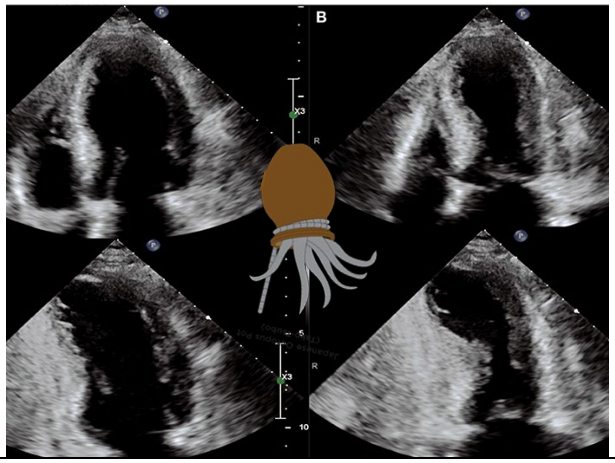
Aortic Stenosis

Epidemiology	2nd most common valvular disease in the US Prevalent in 5% of adults >65 yo, 12.4% of adults age >75 Age-related calcification most typical, but can occur 2/2 bicuspid or rheumatic disease		
Presentation	HF, Angina, Syncope, Fatigue all possible presentations		
Physical Exam	"Pulsus parvus et tardus" Systolic crescendo-decrescendo ejection murmur at RUSB +/- radiation to carotids Later murmur = more severe Loss of S2 = likely severe 		
Echo Findings – Gauging severity	**Mild** – usually Asx US every 3-5 y	**Moderate** – usually Asx US every 1-2 y	**Severe** – can be asx US every 6-12 mo
	2.0-2.9 cm^2 valve area, 2.0-2.9 m/s jet velocity <20 mmHg pressure gradient	<1 cm^2 valve area, 3.0-3.9 m/s jet velocity 20-39 mmHg pressure gradient	<1 cm^2 valve area, 4 m/s jet velocity 40 mmHg pressure gradient
Treatment	Observation	TAVR	SAVR
Decision of who and when to replace / perform valvuloplasty is complex but in general, treat symptomatic patients or asx pts with reduced LVEF	**Most asymptomatic patients** unless Stage C with LVEF<50%, other cardiac surgery Serial US at frequency per AS severity	Less risk, but may be less durable ?better in older patients Anticoagulate!	Higher risk, but may be more durable Better in younger, more robust patients Anticoagulate!

GDMT for HFrEF

Classify	ACC/AHA Stages	NYHA Classes
	A: At-risk (HTN, CVD, DM, Obesity, FH, etc) **B**: No current sxs but structural heart dz, evidence of increased filling pressure, or elevated BNP/trop in absence of alt dx **C:** Current or previous HF sxs **D**: End-Stage (Recurrent hospitalization, having to decr GDMT)	**1:** Structural myocardial changes without limitation to ordinary physical activity **2**: Symptoms with moderate activity **3**: Symptoms with mild activity **4**: Symptoms at rest
BB (Class 1 recc)	Metoprolol Succinate (XL) (25 mg daily start, 200 mg daily goal), Carvedilol (3.125 mg BID start, 25-50 mg BID goal), Bisoprolol (1.25 mg daily start, 10 mg daily goal)	
RAASi (1)	ARNI (Entresto = sacubitril/valsartan) preferred (24-26 mg BID start, 97-103 mg BID goal) ARB or ACEi acceptable	
SGLT2i (1)	Empagliflozin or Dapagliflozin (10 mg start = goal)	
MRA (1)	Spironolactone (12.5-25 mg start, 25-50 mg goal), eplerenone (25 mg start, 50 mg goal)	
Bidil (1*)	Hydralazine-isosorbide dinitrate (1 tab TID start) for NYHA III-IV sxs in African Americans	
Diuretic PRN (1)	As needed or daily torsemide/furosemide/bumetanide PO for patients with Stage C-D HF	
NOTE	Having patients on one agent from each class is better than having them on max dose of one agent but not being able to add other agents due to symptoms or HoTN.	

Takotsubo Cardiomyopathy

Disease	Stress-induced cardiomyopathy (SCM; or apical ballooning syndrome) leading to reduced LV systolic function in the absence of obstructive CAD, pheochromocytoma, or myocarditis. - May present with chest pain/SOB/syncope on the spectrum from ADHF to cardiogenic shock
Epi.	Predominantly affects older females; may represent 1-3% of all STEMIs Usually precipitated by stressful physical/emotional event (e.g. spousal death, surprise)
Eval	Troponin: Elevated EKG: Non-specific or ischemic-appearing (STE) TTE: **"Octopus Pot" appearance** == apical dyskinesia/ballooning with normal base motion. Wall motion abnormalities not following a coronary artery territory. **↓LVEF** LHC**:** pts often get ischemia eval to rule out ACS CMR: may help distinguish b/w myocarditis, infiltrative disease as SCM often lacks late gad enhancement) 
Tx	Supportive physical/mental health care; as per presentation (ADHF, cardiogenic shock) => GDMT - If LV outflow obstruction present, treat similar to HOCM Repeat TTE in 3-6 months to evaluate EF recovery (most regain normal function within wks to mos) Recurrence rate 1-2% per year

Advanced Heart Failure

Indicators	"I NEED HELP" mnemonic ~ essentially EF<35% with recurrent HF exacerbations and hospitalizations despite escalating diuretics, intolerance or de-escalation of GDMT, or need for inotropes == ACC Stage D	
Specialty Care	Refer to HF specialist EARLY (Class I)	
Inotropes	Palliative for patients not candidates for advanced therapies OR Bridge to Therapy Dobutamine (B1 agonist - faster/shorter acting – better at first in ICU – arrhythmia, tachycardia, HTN) Milrinone (PDE3i – slower/longer acting – good for home therapy – C/I in renal dysfxn - arrhythmia, hoTN, HA) ** side note: no significant difference in outcomes in cardiogenic shock between these agents (Mathew, et al, 2021)	
Therapy	Heart Transplantation (Class I) Younger <65-70. Extensive work-up for candidacy is institution-specific but may include end-organ function labs, RHC, RUQUS w/doppler, PFT, UTD immunizations and cancer screenings, & much more. No TUD or substance use; need good social support and medical adherence ~4000 transplants / yr (roughly half of eligible pts) Complications include CMV/infection, rejection, drug AE (HTN, DM, skin ca, etc) Mean survival of 11 years post-transplant	Durable Mechanical Circulatory Support (Class I) Left Ventricular Assist Device (LVAD) most common Bridge to Transplant or Transplant Candidacy vs. Destination Therapy (depends on comorbidities). Similar evaluation as transplant pre-MCS insertion. Need anticoagulation and continued GDMT. Less functional due to external wires/driveline, carrying around the VAD battery pack, etc Complications include stroke, skin or pump infection, pump thrombosis, arrhythmia, and GIB Mean survival around 5 years post-VAD

HFpEF

Diagnosis

1. **Signs/symptoms of HF caused by a structural and/or functional cardiac abnormality**
   - With EF >= 50%
2. **Corroborated by at least one evidence of elevated filling pressures**
   - Elevated proBNP
   - Cardiogenic pulmonary or systemic congestion (JVD, peripheral edema, pulmonary edema, e.g.)

H2FPEF score

H2 = Heavy (BMI>30) = 2pts
On >= 2 antiHTN meds = 1 pt
F = Atrial fibrillation = 3 pts
P = Pulmonary hypertension (PASP >35 mm HG on Echo) = 1 pt
E = Elder age (>60 yo) = 1 pt
F = Filling pressure (E/e' >9 on doppler Echo) = 1 pt
Sum >= 6 points = highly diagnostic of HFpEF

Initial Evaluation

Initial Work-Up (All HF Pts)
EKG + TTE
ProBNP
CBC
CMP
Iron Panel
Lipids
UA with microalbumin panel

Reduced EF (<40%)
Ischemic Eval (MPS, LHC, etc) - most common cause

Nonischemic eval (SPEP/UPEP/IFE/FLC, HIV, EtOH hx, Chagas eval, CMR)

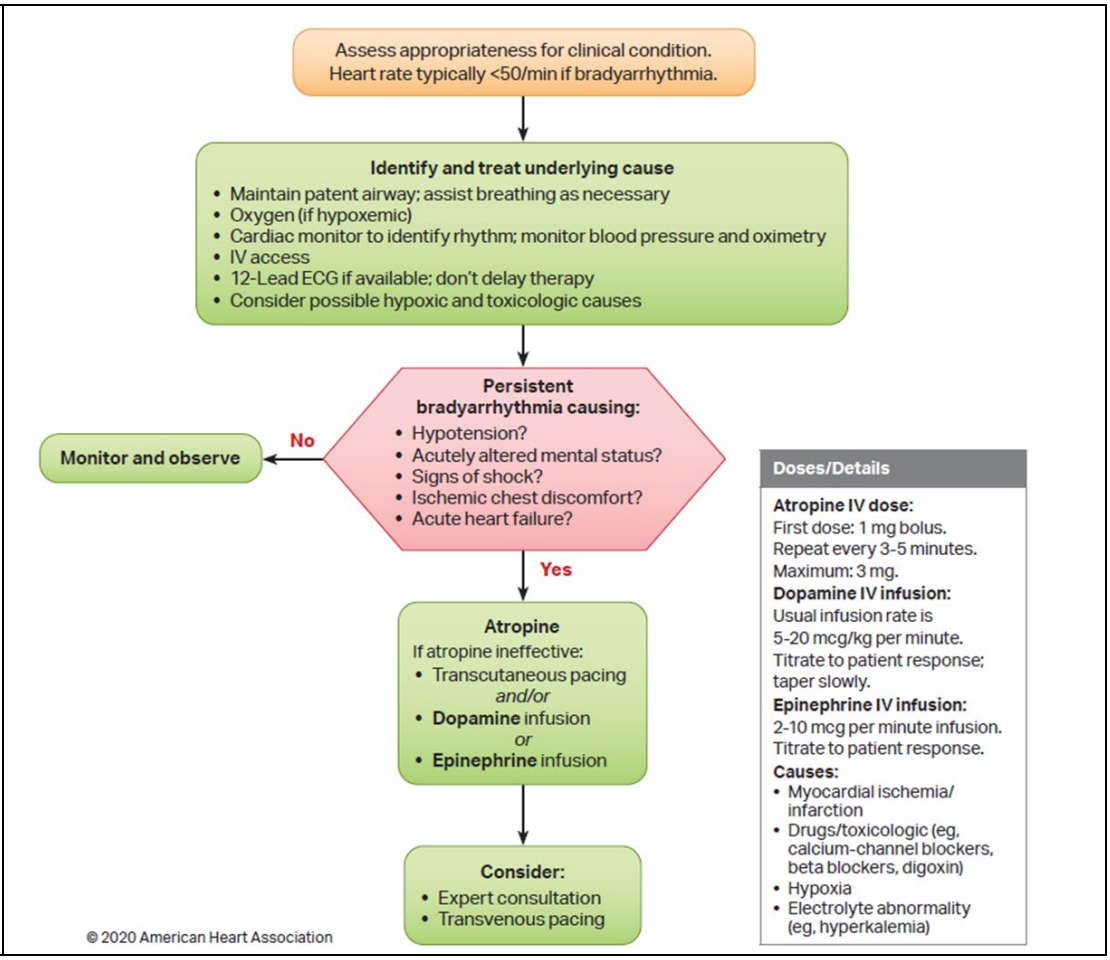
Preserved EF (>=50%)
Rule out non-cardiac mimics (CKD, Liver dz, CVI)
Eval cardiac mimics (Infiltrative or Hypertrophic CM, Pericardial or Valve dz, HOHF)
Eval risk factors (HTN, DM, Afib, Obesity, CAD, CKD)

GDMT for chronic Mgmt

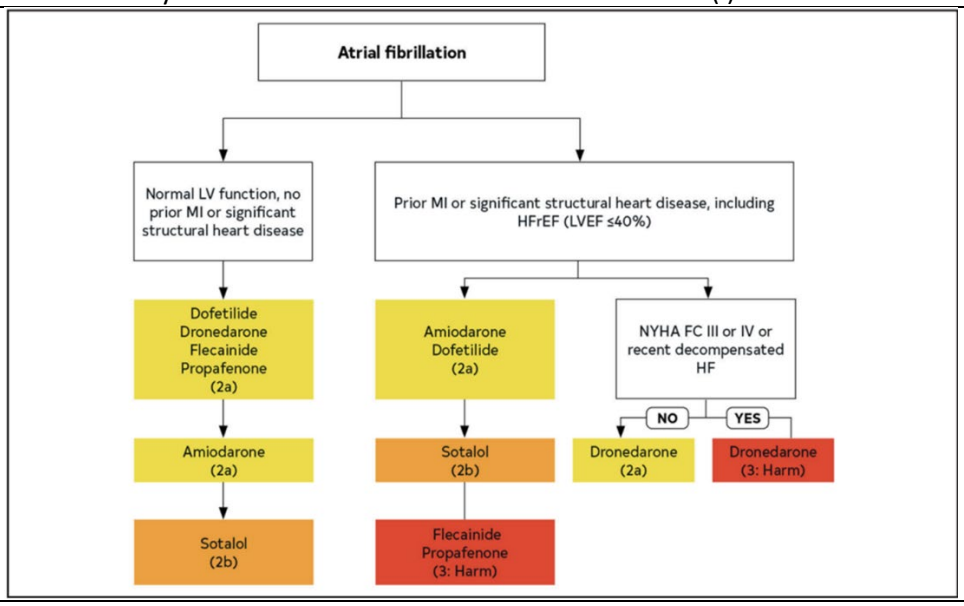
PRN Diuretic (Class 1 rec) – furosemide, bumetanide, torsemide
SGLT2i (2a) – empagliflozin, dapagliflozin, canagliflozin
MRA (2b) – spironolactone, eplerenone, finerenone
RAASi (2b) – ACEi, ARB, or ARNI
GLP-1… not formally recc'ed but coming soon? – dulaglutide, liraglutide, semaglutide, tirzepatide, etc

Bradycardia

Diagnostics:
- Obtain BMP, TSH, Trop (if ACS concern)
- Dig level PRN
- Consider Lyme serology for AVB
- TTE
- **Complete Med Rec!!**



Rhythm control in atrial fibrillation

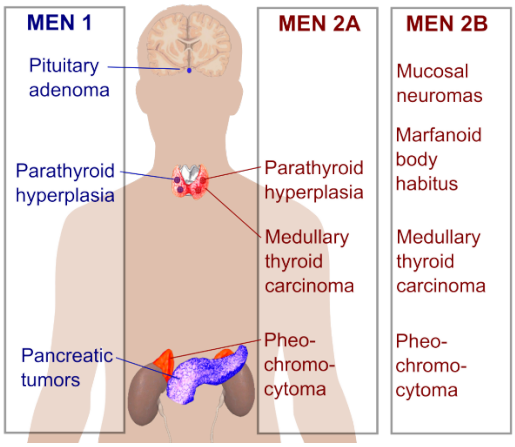
Classifications	At risk – presence of modifiable/non-modifiable AF risk factors (obesity, OSA, DM, EtOH, male, age) Pre-AF – structural or electrical evidence that predispose to AF (Large atria, PAC, AT, Aflutter) Paroxysmal AF – intermittent AF for <=7d from onset Persistent AF – continuous AF for >7d and requires intervention Long-standing persistent AF – continuous AF for >12 mo Successful AF ablation – freedom from AF after percutaneous/surgical intervention Permanent AF – AF without further attempts at rhythm control
Rhythm vs Rate	When to choose rhythm control: - "Everyone deserves a trial of NSR" – ultimately a discussion of patient goals with Cards - Younger, shorter period with A-fib diagnosis, symptomatic When to choose rate control: - Older, longer history of A-fib dx (harder to convert to NSR), less or asymptomatic
Electrical Intervention	Electrical cardioversion – recommended as initial strategy OR after medication conversion fails (I) - 3 weeks AC before, 4 weeks afterwards – if in afib for >-48 hrs for ALL patients - Perform TEE to rule out LA thrombus if urgent cardioversion needed (unstable) (I) First-line ablation is effective for symptomatic, paroxysmal A-fib (Class I) - Favor for younger patients with fewer comorbidities - Better than medication to keep patient in NSR, especially EARLY in disease course - Need AC and rhythm control for at least 3 months afterwards (I)
Rhythm control agent selection	
Drug Monitoring	Amiodarone – effective but toxic - Thyroid function (baseline, T+3mo, annual); LFT, eye exam, PFT & CXR (baseline, annual) Dofetilide – can be used for inpatient chemical cardioversion then maintenance - BMP, Mg/Phos, EKG q3-6 mo Fleicanide + Propafenone – contraindicated in structural or ischemic heart disease - Can be used for pill-in-pocket at-home treatment - Must take beta-blocker ~30 min before use - First use should be monitored in ED for hemodynamic stability; if stable, ok for home use
Other considerations	Don't forget anticoagulation (stroke ppx) per CHA2D2SVASC risk! (balanced with HAS-BLED) Obesity management, OSA diagnosis and treatment, Alcohol reduction or cessation OK to continue caffeine consumption if patient reports caffeine not a symptom trigger

Endocrinology

Endocrine Emergencies

Organ / Derangement	Condition	
Hyperglycemia	DKA	HHS
	Hyperglycemia (>250) Arterial pH <7.3, Bicarb <15 Moderate ketonuria/emia (bHB) Often T1DM, but can be T2DM May have faster onset TX: INSULIN GTT PROTOCOL	Hyperglycemia (>600) Serum Osm >320 Volume depletion Less marked acidosis Often T2DM after recent/protracted illness TX: INSULIN GTT PROTOCOL
Adrenals	Pheochromocytoma	Adrenal Crisis
	May be etiology for recurrent severe HTN 6P's: Pounding HA, Perspiration, Palpitations/Tachycardia, Paroxysmal HTN panic, pallor DX: 24hr Ur metanephrines / catecholamines TX: Surgical resection! Phenoxybenzamine pre-op or Phentolamine in acute HTN crisis (Nicardipine, nitroprusside, etc also)	Shock > Orthostatic HoTN Severe N/V/D Dehydration, hypoNa, hyperK, hypoGlyc Pain in back, abdomen, legs AMS/LOC, fever/hypothermia Primary vs Secondary vs Tertiary etiologies TX: Collect ACTH and Cortisol up front, then give hydrocortisone 100 mg STAT, then q6-8h
Thyroid	Thyroid Storm (SEVERE HYPER)	Myxedema Coma (SEVERE HYPO)
	Thermoregulation dysfxn, AMS/Seizure/Psychosis/hyperreflexia, lid lag, Afib/Tachycardia/CHF, Dys/Tachypnea, N/V/D MANY possible precipitants (infection, trauma, DKA, overreplacement, e.g.) DX: Burch-Wartofsky score >45 TX: PTU, Propranolol, SSKI, Hydrocortisone	AMS!!, Alopecia, HoTN, delayed reflexes, dry skin, abd distension/ileus, general edema MANY precipitants (infxn, cold, stroke, meds) Dx scoring systems available (not in MDCalc) TX: IV Levothyroxine +- IV T3, Hydrocortisone
Pituitary	Pituitary Apoplexy	
	Sx: Sudden onset, severe HA, N/V, photophobia, visual deficit (diplopia, ophthalmoplegia), CN palsy (3^{rd}> 4/5/6), AMS/Seizure, Collapse, sudden death Dx: MRI (or CT) will confirm, assess pituitary function and basic labs, fluid status Tx: Get Endo/NSGY/Neuro on board, may need OR, IV steroids, supportive care in ICU	
Parathyroid	Hypercalcemia	Hypocalcemia
	Stones, bones, groans, psychiatric overtones! Severe >14 Identify etiology! PTH dependent? Get iCa, 25-OH Vit D, PTH, PTHrp, Mg/Phos Tx: Dialysis for ESKD/ARF; otherwise - Aggressive IV hydration (diuresis if overload occurs), IV bisphosphonate w/wo calcitonin, denosumab if refractory	Sx: HoTN, Torsades, heart block, bradycardia, anxiety/AMS, paresthesias, seizure, cramps. Post-surgical complication of thyroid/parathyroidectomy PPX: Oral calcium, calcitriol Tx: if hypoPTH, PO calcium, calcitriol; if sxs/progression, increase oral doses, 1-2g Ca Gluconate ONLY over 10 min followed by continuous IV infusion; replete other lytes

Endo Potpourri

Syndrome	Clinical Findings	Etiology	Evaluation
Cushing Syndrome	Hypercortisolism: Mood disturbance, moon facies, osteoporosis, HTN, central obesity, facial plethora, skin wrinkling/bruising/striae/ulcers, amenorrhea,	Excess cortisol ACTH-dependent: pituitary adenoma, carcinoid tumors ACTH-independent: adrenal adenoma, exogenous steroids	1 mg dexamethasone suppression test + 24 hr urine free cortisol OR Late-night salivary cortisol If 2/3 positive -> ACTH Low ACTH -> MRI/CT Abdomen for adrenal tumor Hi ACTH -> High-dose suppression test HDST neg -> CT pan scan for ectopic ACTH source HDST pos -> MRI/CT for Cushing's dz
Multiple Endocrine Neoplasia, Type 1		MEN1 gene mutation (though not required for clinical diagnosis) Pituitary: adenoma (Cushing/Acromegaly), prolactinoma Parathyroid: adenoma Pancreatic: gastrinoma, insulinoma, VIPoma, carcinoid	Diagnosis requires 2 or more primary MEN1 tumor types OR 1 MEN1 associated tumor in family members Genetic testing if suspected as above, multiple pancreatic NETs, FH or PH of other endocrinopathies, or pts <40 with gastrinomas/insulinomas/etc
Hypoglycemia in non-T2DM	Whipple's Triad: Fasting BG < 55, neuroglycopenia, resolution of sxs with PO glucose OR Fasting BG < 45 + neuroglycopenia (neuro sxs, e.g. confusion)	Exogenous insulin use Insulinoma Diminished PO intake	72-hour fast in those for whom cause isn't obvious (protocols vary) BMP, C-peptide, Insulin, Proinsulin, bHB
Euglycemic DKA	HAGMA + elevated bHB + urinary ketones T1DM or T2DM Taking an SGLT2i	Increased glycosuria and diuresis + Increased glucagon -> maintained decreased insulin state & increased lipolysis/ketogenesis Decreased renal ketone clearance Euglycemia maintained via kidneys	BMP Beta-Hydroxybutyrate UA Med Rec Managed like usual DKA and discontinuation of SGLT2i (update EMR)

Adrenal Masses

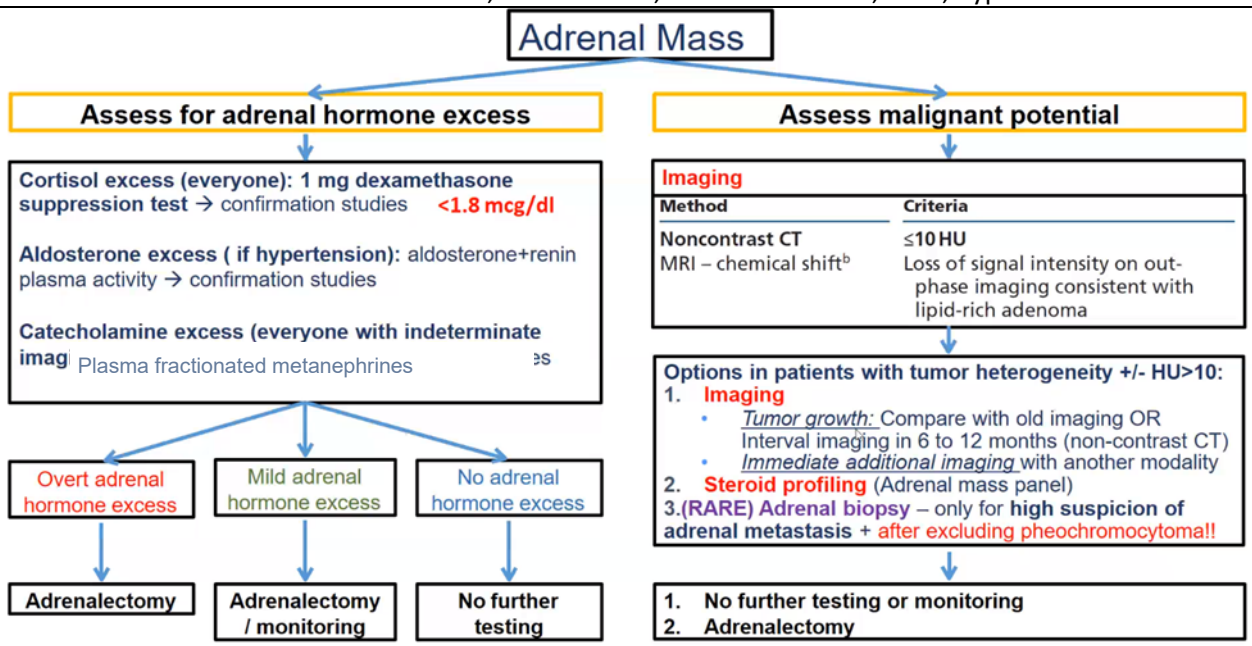
Adrenal Mass etiology → 4% pheo, 4% carcinoma, 2% other malignancy, otherwise benign

- 5-15% are functional (adenoma, aldosteronoma, pheo)
- 5-10% are malignant (carcinoma, pheo)
- Adrenal Incidentalomas are common! 85% of all adrenal tumors, found in 6% of all adults
- ALL adenomas ≥1cm should be evaluated

Evaluation consists of 2 steps:

1. Is this cancer?
    a. Need good Hx (weight loss, prior cancer, lymphoma, virilization) + dedicated CT scan w/wo contrast
    b. ≥10 HU density, irregular shape/content, size ≥4cm, ≥1 cm annual growth = incr risk of cancer

2. Is this secreting excess hormones?
    a. Need good Hx (episodic palpitations/panic/pallor, cushingoid sxs, virilization) + labwork + CT scan
    b. Pheo – plasma fractionated metaphrines (24hr urine metanephrines later) – NOT catecholamines
        i. TCA, dopaminergics, amphetamines, reserpine, withdrawal, EtOH can alter levels
    c. Cortical adenoma – 1 mg ON DST, Plasma aldosterone concentration, Plasma Renin activity
    d. Adrenocortical carcinoma – DHEA-S, testosterone, androstenedione, BMP, hyperaldo eval



**Remember, evaluate cortisol secretion in ALL patients with an adrenal mass – use 1 mg dex suppression test!

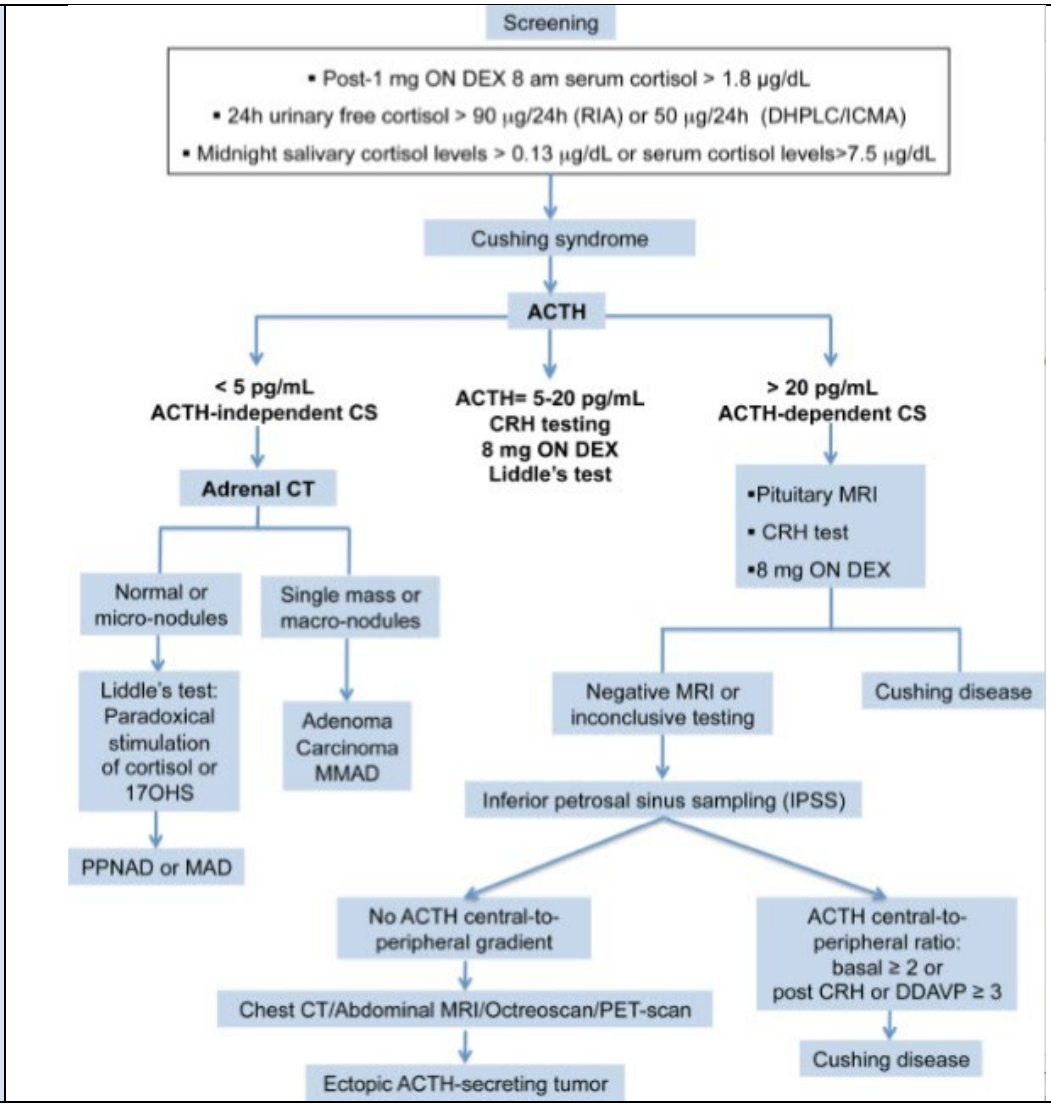
Hyperandrogenism

Definition	Excess androgen levels in females
Symptoms	Hirsutism (most common – male pattern terminal hair growth) Virilization (deeper voice, laryngeal enlargement, enlargement, refractory acne vulgaris, male-pattern baldness) Only occurs with SEVERE hyperandrogenism (e.g. androgen-producing tumor or ovarian hyperthecosis)
Differential	- **PCOS = 95% of cases – can be at any point after menarche, but often younger women** - Congenital adrenal hyperplasia (CAH) = 1 to 2% - childhood to early adult onset - Adrenal tumor, Ovarian Tumor, Cushing's Syndrome, ovarian hyperthecosis = each <1% of cases
Clinical Evaluation	History: **menstrual history**, onset of hirsutism, exogenous testosterone exposure, family history Exam: BP and weight, skin exam for acanthosis nigricans, facial/body hair, acne vulgaris
Diagnostic Evaluation	All patients: Total testosterone + SHBG ± morning, follicular-phase 17-hydroxyprogesterone (CAH eval – some reserve this just for oligomenorrheic patients) - Add TSH w/reflex FT4, Prolactin, HCG, and FSH if pt has oligomenorrhea/amenorrhea eval - Add DHEAS for severe sxs, rapidly progressive hirsutism or virilization – high levels not c/w PCOS For DHEA >700 ug/dL or testosterone >150 ng/dL: - Adrenal CT/MRI for adrenal tumor eval - TVUS for ovarian tumor eval (not needed for PCOS dx if all other hyperandrogen causes ruled out)

Cushing Syndrome

Suspicious Sxs:
Truncal obesity
Buffalo hump
Moon facies
Acne
DM or Hyperglycemia (polyuria/dipsia)
Purple striae
Impaired wound healing
Easy bruising
Facial plethora

1. Confirm hypercortisolism
↓
2. Assess ACTH dependence
↓
3. Localize Lesion



Pituitary Potpourri

Condition	Definition	Evaluation	Management
Pituitary incidentaloma	Incidentally identified pituitary mass, microadenoma <10mm, macroadenoma if >10mm, commonly non-functioning	1. Rule out hypersecretion (prolactin, IGF-1; add cortisol/ACTH if indicated) 2. Rule out hypopituitarism (FSH/LH, cortisol, TSH/FT4, total testosterone in M; menstrual history in F, Presence of menses = no FSH/LH)	Treat per findings; if benign then nothing; if sx of non-prolactinoma lesion (homonymous hemianopsia, e.g.) can surgerize
Hyperprolactinemia	Elevated prolactin level; Physiologic in pregnancy, lactation or nipple stimulation, stress, sleep, high protein meals, Pathologic in pituitary adenoma, medication, hypothyroid, decreased protein clearance	Repeat prolactin level, TSH/FT4 History addressing physio/pathologic causes (new antipsychotic, SSRI/TCA, verapamil, reglan, e.g. Visual field testing, reflexes Pituitary MRI	Asx microadenoma = none Macroadenoma = give dopaminergic (cabergoline) Sx not tolerating dopaminergic = surgery
Pituitary Apoplexy	Apoplexy = sudden hemorrhage or infarct of a pituitary adenoma Sheehan syndrome = pituitary infarct from postpartum hemorrhage	Query HA, diplopia, vision loss, AMS, hypocortisol sxs Obtain hypopituitarism labs, BMP Pituitary MRI NSGY consult	Vision loss = NSGY emergency!! Hydrocortisone for ACTH deficiency (life threatening!)
Medication-induced pituitary dysfunction	Multiple medications alter pituitary function and can lead to changes in size or function (e.g. Immune checkpoint inhibitor hypophysitis, opioid-induced hypogonadotropic hypogonadism, psych meds)	Obtain hypopituitarism labs (ACTH, LH, TSH, GH) Med rec, looking for: estrogens, testosterone, antipsychotics, somatostatin/ocreotide, reglan, GnRH agonist (leuprolide),	Replace hormones, give steroids or sex hormones if needed, discontinue the offending medication if able
Diabetes insipidus More in Nephro!	Central = posterior pituitary cannot make ADH – often 2/2 trauma/anoxia/surgery Nephrogenic = kidneys cannot respond to ADH	Urine and serum osms Water deprivation challenge Desmopressin challenge (PO, nasal, subQ)	Central = desmopressin (consider TZD or NSAID) Nephrogenic = drink to thirst, TZD, <2g/day Na restriction, consider NSAIDs

Post-TBI Hypopituitarism

Epi

Direct and indirect insults to the pituitary gland/stalk (infarct, hypotension, ischemia, shearing forces, etc)
Risk increases with: diffuse brain swelling, prolonged ICU admission, incr ICP, hypotension, hypoxia, basal skull fracture, increased age, blast injury
Acute: Anterior hormone dysfxn (GH and LH/FSH most common) as high as 53-78%
Posterior hormone dysfxn – DI in 3-51%, SIADH in 3-37%
Chronic hypopituitarism: 13-56% at 3-6 months, 5-76% at >12 months

Symptoms

ACTH Deficiency:
- Weakness, fatigue, muscle and joint pain
- Hyponatremia
- Hypoglycemia
- Hypotension (especially if co-existing DI)

GH Deficiency:
- Increased fat mass
- Lower BMD of L-spine (? Fracture risk)
- Poor quality of life
- Dyslipidemia
- Increased inflammatory markers
- Higher CAC scores
- Increased mortality

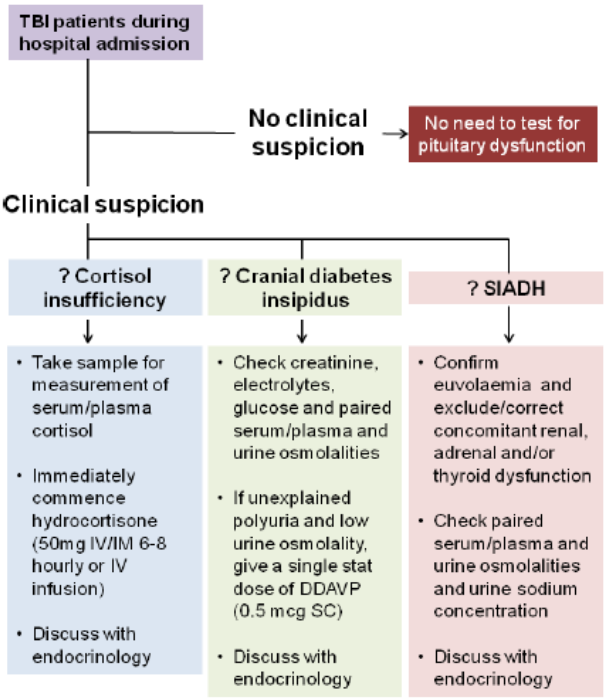
Prolactin: Inability to lactate

TSH Deficiency:
- Fatigue
- Cold intolerance
- Decreased appetite
- Constipation
- Facial puffiness/edema
- Dry Skin
- Bradycardia
- Delayed relaxation phase of the DTRs
- Anemia

Gonadotropin Deficiency:
- Women: Amenorrhea, hot flashes, vaginal dryness, low libido, infertility, decreased BMD
- Men: Infertility, low libido, decreased energy, decreased muscle mass/strength, decreased sexual hair, decreased BMD

Screening

Current recommendations:
- Serum cortisol levels monitored for the first 7 days post-TBI
- Screening for ACTH and TSH deficiencies:
  - Patient is hospitalized for >24hrs
  - CT head shows abnormality (brain swelling, diffuse axonal injury, basal skull fracture, epidural/subdural hematoma, cranial vault fractures)
  - Signs or symptoms of hypopituitarism
  - In general, most agree to treat for AI if clinical suspicion
- DI should be considered with hypernatremia and hypotonic polyuria



Screening tests (performed at 0900):

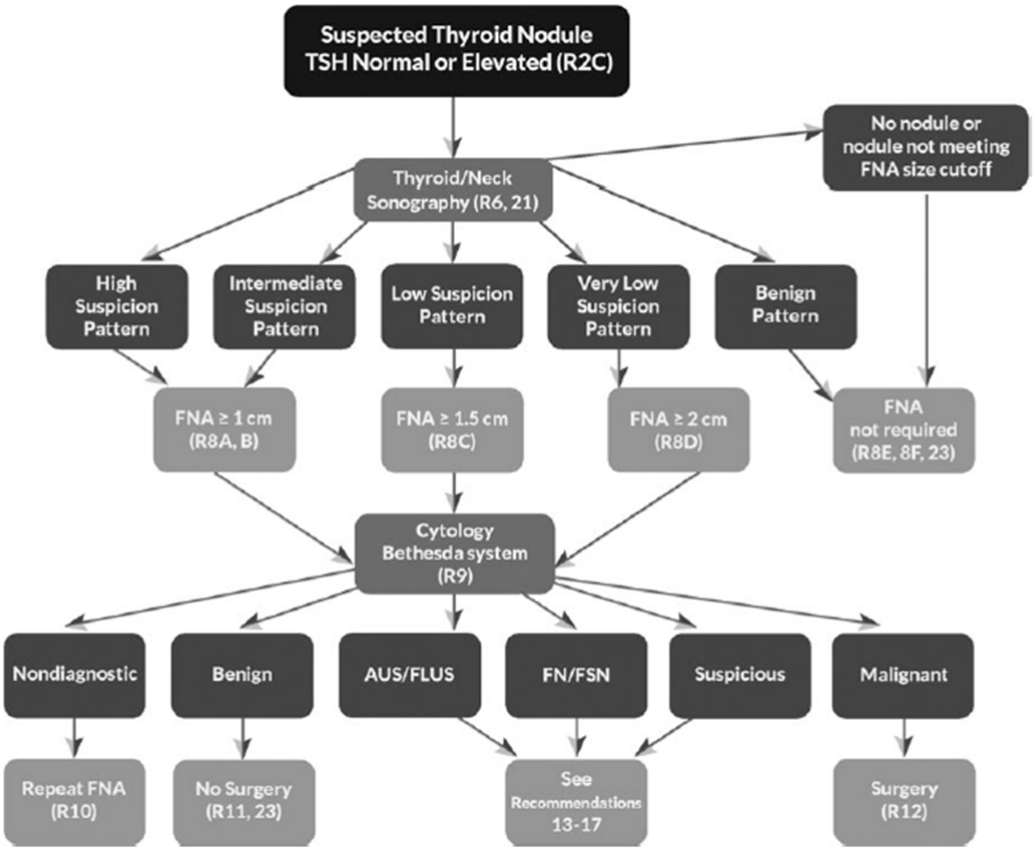
Men:
BMP, TSH and FT4, Cortisol, LH, FSH, Testosterone, SHBG, albumin

Women:
BMP, TSH and FT4, Cortisol
AND
LH, FSH, estradiol (if premenopausal with new cycle irregularity)
OR
FSH (postmenopausal)

Adrenal Insufficiency

		Tertiary	Secondary	Primary
		Hypothalamus	**Pituitary**	**Adrenal**
		↓ Production/ ↓ Response	**↓ Production**	**↓ Production / ↑Metabolism**
Etiology		• Most common = long term steroid use • Space-occupying (Tumors) • Trauma • Infiltrative (infections, granulomatous disease, malignancy, proteins, metals) • Iatrogenic	• Most common = long term steroid use • Congenital/Genetic • Autoimmune • Space-occupying (Tumors) • Trauma • Infiltrative • Vascular (hemorrhage, thrombosis) • Iatrogenic	• Most common = autoimmune (Addison disease) • Congenital/Genetic • Infiltrative (infectious, granulomatous diseases, malignancy, protein, metal) • Vascular (hemorrhage, thrombosis) • Liver disease • Iatrogenic (Surgery, Rads, meds)
Diagnosis		<u>Basal (0600 – 0900) Cortisol</u> Serum (0600 – 0900) < 3 mcg/dL (500 nmol/L) Salivary < 0.18 mcg/dL (5 nmol/L) <u>Cosyntropin Stimulation Test</u> Change in Cortisol < 9 mcg/L – less important Peak cortisol < 14.5 mcg/L		
		Low Plasma ACTH Normal renin & aldo		High Plasma ACTH Elevated renin, Low aldosterone Low Na, High K
		Complete history, Medication reconciliation Targeted laboratory and radiologic diagnostic testing		
Management	**Adrenal Crisis**	• 100 mg IV hydrocortisone • Bolus 1 L isotonic saline +/- 5% dextrose (if hypoglycemic) • 200 mg/day hydrocortisone: 50mg Q6H or daily continuous infusion • Then, 100 mg hydrocortisone daily until stable for transition to maintenance oral regimen		
	Sick Day/ Stress Dosed	Minor: Double or triple hydrocortisone x2-3 days based on the severity of illness Moderate (ex: admitted with CAP): 50 – 75 mg/day hydrocortisone in divided doses (ex: 25mg Q8H) Major: 100 mg IV hydrocortisone & 200 mg/day (50mg Q6H or continuous infusion) until stable for PO		
	Chronic	• 15-20 mg daily hydrocortisone in 2-3 doses (2/3rds in AM) • Females: Consider DHEA 25-50 mg if ↓libido, depression, or persistent fatigue		• 15-25 mg daily hydrocortisone in 2-3 doses (2/3rds in AM) • 0.05 – 0.01mg fludrocortisone (unless > 40 mg hydrocortisone daily) • Females: Consider DHEA 25-50 mg if ↓libido, depression, fatigue

Thyroid Nodule Evaluation



Nodule Types	<u>Benign (90-95%)</u> Adenomatoid nodules (85%) Adenomas (10-15%) Cysts (<1%)	<u>Malignant (5-10%)</u> Papillary (80-85%) Follcular/Hürthle Cell (10%) Medullary (2-3%) Anaplastic (1%) – aggressive! Lymphoma or Met (<1% each)
Thyroid Cancer Risk Factors	<u>Moderate Suspicion</u> Age <20 or >70 yo Male Prior head/neck irradiation >4 cm size Compressive symptoms (dysphagia, dysphonia, hoarseness, SOB, cough)	<u>High Suspicion</u> Family Hx (MTC or MEN) Rapid growth Firm, Hard, or Fixed Vocal cord paresis Cervical LAD Distant metastasis Focal uptake of FDG PET Female (3:1) for PTC
Treatment	<u>Total or Partial Thyroidectomy</u> Mainstay of treatment!! Total preferred unless unilateral, small, without spread Risks: post-op hypocalcemia (parathyroid injury/removal -> hungry bone), SOB or vocal change from RLN injury	<u>Radioactive Iodine (I^{131}) Ablation</u> NOT always indicated Only for Iodine utilizing cancers (follicular, papillary) <u>Use</u>: post-op remnant ablation OR adjuvant therapy with intermediate or high risk of recurrence (extrathyroid extension, lymph node involvement, aggressive subtype, etc) <u>Contraindication</u>: pregnancy, breastfeeding <u>Risks</u>: radiation thyroiditis, neck edema, tumor bleed, Nausea (Also many post-treatment restrictions on contact/travel/etc!!)

Subclinical Hyperthyroidism

Definition	Suppressed TSH <0.5 mU/L (or LLN) with normal-range FT4
Epi & Risks	Affects ~0.7% of adults Increased risk for: Atrial fibrillation, CV events, hip fracture
Initial Eval	Ensure no biotin supplementation around testing (will falsely lower TSH) HPI regarding: sxs of hyperthyroidism (temperature intolerance, swelling, agitation, palpitations, etc), recent neck irradiation, preceding viral illness (de Quervain's), recent childbirth, medications (exogenous synthroid, amiodarone, steroids) Palpate thyroid for tenderness, firmness, increased size, nodularity Repeat TFT in 1-3 months to confirm diagnosis - 25% of patients will have normal TSH within 6 weeks!
Who to Treat	Treatment is generally recommended for: - All symptomatic patients - TSH <0.1 mU/L (though you can observe if asymptomatic with negative testing) - Patients with significant cardiac risk factors or heart disease - Patients with osteoporosis or osteoporosis risk factors Otherwise, observation is usually appropriate for: - TSH 0.1-0.5 mU/L - Low-risk patients (<65 years old, premenopausal, e.g.) without symptoms
Once you Decide to Treat	1. Identify the etiology and possible complications: a. Thyrotropin Receptor Ab (TRAb) and Thyroid stimulating immunoglobulins (TSI) b. Radioactive iodine scan (MNG is the most common cause for subclinical hypothyroidism) i. Thyroid US can be used afterwards to eval nodules or if scintigraphy is contraindicated c. DEXA scan for postmenopausal females 2. Initiate treatment a. Pursuant to underlying etiology as with overt hyperthyroidism b. May include beta-blockers for sxs, observation (thyroiditis, Iodine exposure), or PTU/MMI or RAIU/surgery

Diabetes in Pregnancy

Screening

Patients with T2DM risk factors should be screened at initial prenatal visit
- Dx with T2DM if positive

ALL pregnant patients should be screened at weeks 24-28
- One-step (75g OGTT fasting) or Two-step strategy (50g non-fasting, then 100g OGTT fasting)
- GDM if fasting >92, 1hr > 180, 2h > 155

Why does this matter? Uncontrolled DM == incr risk for mother and fetus!!
- During first 10 wks: incr risk of anencephaly, microcephaly, congenital heart disease, renal anomalies, caudal regression
- During 2nd/3rd trimester: incr risk of macrosomia, preterm delivery, pre-eclampsia

Measurement	Pregnancy Goal	Non-Pregnant Goal
Hgb A1c% (if can be achieved without hypoglycemia)	<6%	<6.5% (females attempting conception), <7% (most adults) <7.5-8.5% (older adults pending comorbidities, PS)
Fasting/Pre-prandial	70-95 (or <95 in GDM)	80-130
1-hr Post-prandial	110-140 (or <140 in GDM)	<180
2-hr Post-prandial	100-120 (or <120 in GDM)	<180

Treatment

Insulin should be used for T1D in pregnancy & is preferred for GDM and T2DM in pregnancy!
- Frequent titration is needed; CGMs are your friend! CGM target range = 63-140 (time in range >70%)

Insulin formulations:
- Detemir, NPH, Aspart, and Lispro (RCT supported!)
- Treat to above targets; requirements increase ~5% per week (often less insulin in first trimester but +35-70% later on)

Oral medications:
- Metformin ok for SOME pts who can't take insulin (contraindications: HTN, preeclampsia, risk for IUGR)

Preeclampsia Prophylaxis:
- ASA 81 mg daily (Week 12 onward) – all pts with DM in pregnancy!

Postpartum considerations:
- Titrate insulin to adjust for nocturnal hypoglycemia 2/2 lactation metabolism
- GDM pts likely won't need tx post-partum but should get 75g OGTT for DM after 4-12 wks!

Gastroenterology

Approach to Abdominal Pain

History	Physical	Labs/Rads
LOCATES Location/radiation Other symptoms Character/Quality Alleviating/aggravating factors Timing – onset/duration – chronicity! Environment at onset Severity PMH, PSH, Meds, bowel movement, LMP, sexual history	Inspect – visible discomfort? distension? bruising? Stigmata? Auscultate – hypoactive/absent BS? Succussion? Percussion – shifting dullness? Tympany? Palpation – organomegaly? Guarding/rebound? Murphy's? McBurney's point? Rectal & GU exams	Labs – per history CBC, BMP, LFT, Lipase, bHCG (acute) Stool studies? Celiac testing? Fecal calpro? STI testing? Add Iron panel (chronic) Rads – per history KUB, RUQUS, TVUS, CT A/P

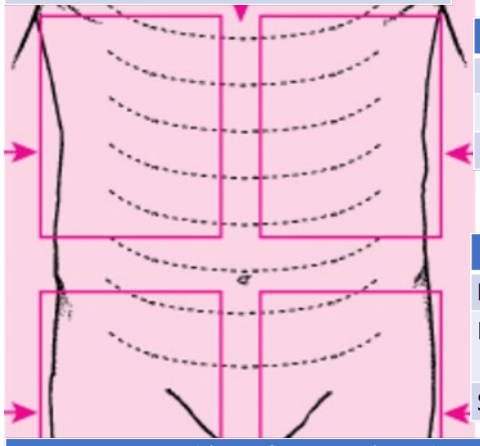
Abdominal Pain Differential

Diffuse Abdominal Pain							
Differential Diagnoses	Acute Pancreatitis	Early appendicitis	Gastroenteritis	Intestinal Obstruction	Mesenteric Ischemia	Peritonitis	• Adrenal insufficiency • Bites/Stings • Diabetic or alcohol ketoacidosis • Heavy Metal Toxicity • Hypercalcemia • Medications (e.g., opioid, anticholinergics) • Sickle cell crisis • Typhoid Fever
Historical Clues	Very severe, Radiation to left shoulder or back	Waves of pain to constant sharp or dull pain	Vomiting precedes pain	Vomiting follows pain, dull, waves of pain	Waves to constant pain, very severe	Very severe, sharp, constant pain	

Right or Left Upper Quadrant Pain	
Differential Diagnoses	**Historical Clues**
Acute Pancreatitis	Very severe, radiation to L shoulder or back
• Costochondritis • Herpes zoster • Lower lobe pneumonia • Myocardial ischemia • Radiculitis	

Right Upper Quadrant	
Differential Diagnoses	**Historical Clues**
Appendicitis w/ Gravid Uterus	Waves of pain to constant sharp or dull pain
Cholecystitis & Biliary colic	Recurrent episodes, waves of pain, Right scapula radiation
Congestive hepatomegaly	
Hepatitis or hepatic abscess	
Perforated duodenal ulcer	Radiation to back, Very sudden

Left Upper Quadrant	
Differential Diagnoses	**Historical Clues**
Gastritis	
Splenic Abscess or rupture	Left shoulder radiation

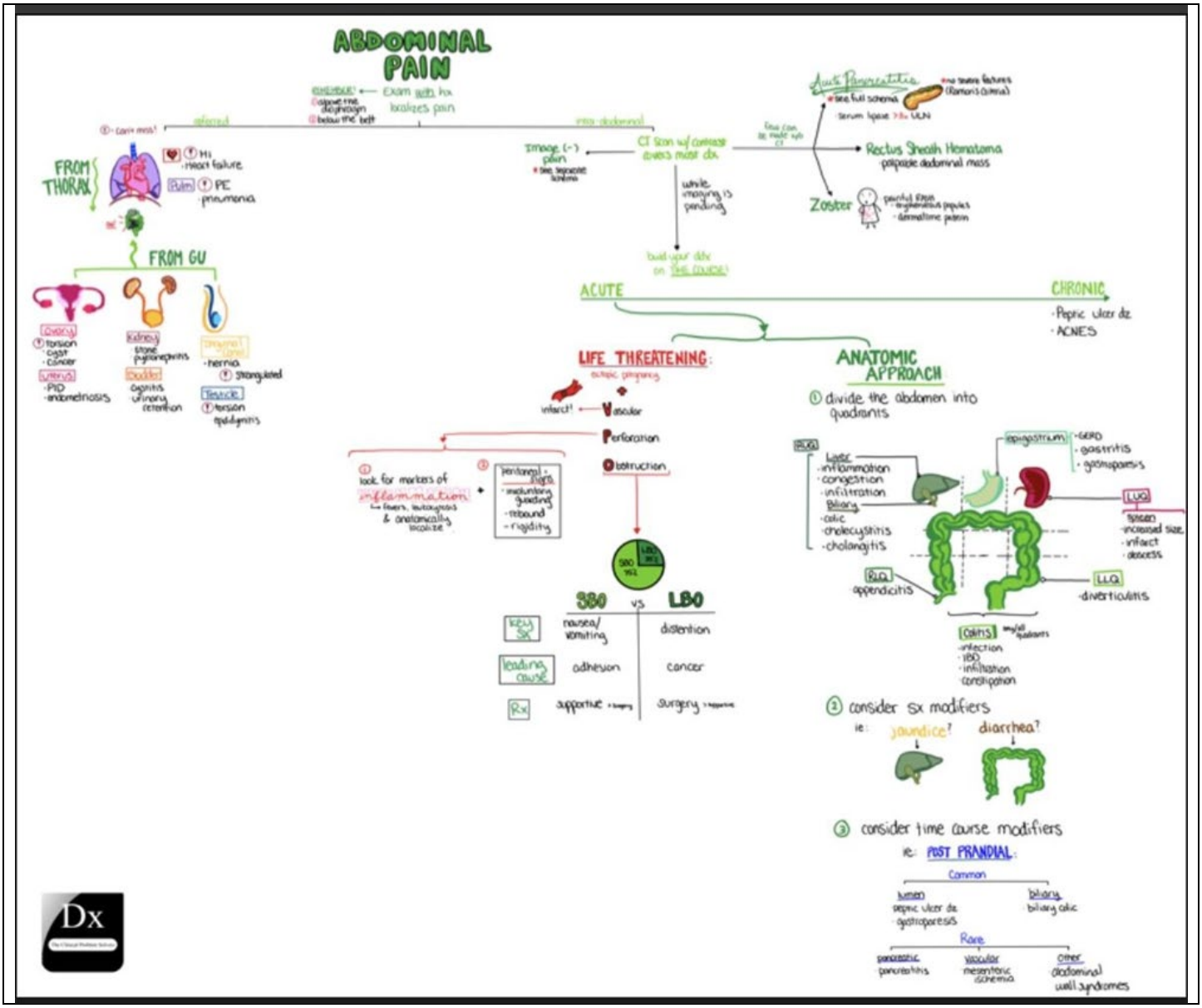


Right Lower Quadrant	
Differential Diagnoses	**Historical Clues**
Appendicitis	Waves of pain to constant sharp or dull pain
Cecal diverticulitis	Recurrent episodes
Meckel diverticulitis	Recurrent episodes
Mesenteric adenitis	

Left Lower Quadrant	
Differential Diagnoses	**Historical Clues**
Ischemic colitis	Bloody bowel movements
Sigmoid diverticulitis	Recurrent episodes

Right or Left Lower Quadrant Pain	
Differential Diagnoses	**Historical Clues**
Abdominal or psoas abscess	Radiates to back
Abdominal wall hematoma	Prior surgery
Cystitis	Suprapubic or groin
Endometriosis	Perimenstrual pain, urinary/defecatory pain
Incarcerated or strangulated hernia	Difficult, painful BM, hematochezia or constipation
Mittelschmerz	Recurrent
Pelvic Inflammatory Disease	Prior untreated STI
Nephrolithiasis	Suprapubic or groin pain, Very sudden, Very severe, waves of pain
Ruptured abdominal Aortic Aneurysm	Very sudden, Tearing, Radiates to back
Ruptured Ectopic pregnancy	Very sudden
Torsion of ovarian cyst or testis	Groin pain, very sudden

Abdominal Pain: Another Approach



Acute Pancreatitis

Diagnosis	**Must have 2 out of 3 of the following:** - Characteristic epigastric or LUQ pain w/wo radiation to back/flank/chest - Lipase (or amylase) >3x ULN (lipase ULN = 60) - Characteristic imaging findings (US/CT/MRI)
Etiology	**I GET SMASHED** **I**diopathic (15-25%) **G**allstones (40-70%) **E**thanol aka Alcohol (25-35%) **T**rauma **S**teroids **M**alignancy / Mumps **A**utoimmune **S**corpion sting **H**ypertriglyceridemia (5%) or Hypercalcemia **E**RCP (post-procedural, esp if multiple wire pass) **D**rugs (Azathioprine, Lasix, sulfa, flagyl, estrogens,5-ASA, valproate, e.g.)
Risk Stratification	Severity - Mild (most common) = no organ failure - Moderately Severe = organ failure resolving within 48 hrs - Severe = organ failure > 48hrs (shock, ARF, GIB, ARDS) or Marshall score >= 2 Scoring systems (sensitive, not specific) - BISAP – can use at 24 hrs and may be more accurate - Ranson's Criteria – need initial and T+48 hr labs - Apache II – estimates ICU mortality
Treatment	1. Supportive care (opiate pain control, anti-emetics) 2. IV Fluids = BOLUS 10 mL/kg up front, then run LR at 1.5 mL/kg/hr **(WATERFALL study)** 3. NPO until vomiting controlled, then start a Low-Fat diet 4. Monitor hemoconcentration (Hct), renal perfusion as surrogate for pancrease (sCr / BUN) at 6-8 hrs after initial resus and daily; consider CRP at 48 hrs 5. Antibiotics ONLY if evidence of necrosis/air or other indication (Pos BCx, PNA, etc) 6. ERCP within 24hrs ONLY if evidence of concurrent cholangitis
Complications	Necrotizing pancreatitis (worse if under-resuscitated) Pancreatic pseudocyst, Walled-off necrosis ARDS

Upper GI Bleeding

<table>
<tr><td>Risk Stratification</td><td colspan="3">Glasgow-Blatchford Score (GBS) – on MDCalc!
- Identifies risk for needing intervention, appropriateness for OP 28gmt.
- Score 0 = low risk
- Score 6+ = high risk (50%)
AIMS65 – on MDCalc! --- identifies risk for mortality – Score 3 (10%), Score 5 (25%)</td></tr>
<tr><td rowspan="2">Buckets</td><td>Ulcerative/Erosive</td><td>Portal Hypertensive</td><td>Vascular Lesions</td></tr>
<tr><td>Duodenal or gastric ulcer
Esophagitis
Gastropathy or Duodenopathy</td><td>Varices (Esophageal, Gastric, or Ectopic)
Portal HTN Gastropathy</td><td>Angiodysplasia / AVM
Dieulafoy’s Lesion
Gastric Antral Vasc Ectasia (GAVE)</td></tr>
<tr><td>Interventions</td><td colspan="3">Always think ABC first
Obtain type & cross (unstable) vs type & screen
Make patient NPO
Hold anticoagulants; if INR > 2.5, need to reverse; partially or completely hold DAPT
Establish two large-bore Ivs (IO or Cordis/MAC also options)
Maintain SpO2 >94%
Treat hypotension (goal SBP > 90 or MAP > 65) – crystalloid/MTP
Consult GI for endoscopy! Consider CTA
IR or Surgery consult if unstable
IV PPI bolus (80 mg) & then BID (40 mg)
SBP ppx with Rocephin</td></tr>
</table>

Lower GI Bleeding

Hematochezia DDX	Evaluation	Treatment
Hemorrhoids/Fissure (most common minor cause) Diverticulosis (often self-limited) Arteriovenous malformation Colorectal polyp or cancer Infectious colitis Inflammatory bowel disease Ischemic colitis Angiodysplasia & Dieulafoy Aorto-enteric fistula Anastomotic dehiscence Post-polypectomy Brisk UGIB (15% of hematochezia)	History: - AC or AntiPlt meds? - Recent surgery or endoscopy? - UGIB risk factors (melena, N/V, liver disease) - Pain, prandial association, # episodes, travel VS: Hemodynamic stability? Exam: melena, hematochezia, DRE, stigmata of cirrhosis, etc CTA – unstable after initial resus OR have ongoing rapid bleeding Colo – HDS, no rapid bleed – finds source in 2/3rd of pts EGD – usually second line but can be first if high suspicion for UGI source	Maintain large bore PIVs RBC/Plt Transfusions as indicated Hold non-ASA NSAIDs, AC, APT, etc Correct coagulopathies Hemodynamically stable: - Colonoscopy if no rapid bleed - CTA or RBC scan if colo neg or cannot tolerate colo Hemodynamically unstable: - Resuscitate - IR and Surgery consults - CTA to identify lesion -- Embolize - UGIB if CTA unrevealing - Colonoscopy thereafter

Chronic Diarrhea Differential

Type of Diarrhea	Watery			Malabsorption and/or Fatty	Inflammatory	
Subtype	Secretory (reduced absorption or increased secretion of water)	Osmotic (poor absorption of osmotically active substances)	Functional (no single underlying mechanism)		Typically Bloody	Atypically Bloody
Presentation	Often nocturnal, unrelated to food intake, fecal osmotic gap < 50 mOsm/kg	Improves with fasting and worse with eating Fecal osmotic gap > 125 mOsm/kg	Loose or watery stools with or without abdominal pain	Steatorrhea, Foul-smelling, floating stool, bloating, gas, frequently with large osmotic gap	Frequent small-volume stools, tenesmus (i.e., urge w/o success), mucous, blood, and/or pus	
Evaluation	• Fecal lytes, Fecal osmolality, TSH, Medication review • Consider: ova & parasites, bile acid diarrhea, endoscopy			Fecal lytes, Fecal osmolality, Stool fat or Sudan stain, IgA tTG, Elastase or chymotrpsin	• Fecal calprotectin, Stool culture/PCR, Ova & Parasites, CRP, ESR, CBC • Consider: Endoscopy	
Differential Diagnosis	• Altered colonic anatomy (e.g., colectomy) • Faster transit time (e.g., stimulants, scleroderma, amyloid) • Medications (e.g., metformin, antibiotics, alcohol) • Endocrine (e.g., VIPoma, hyperthyroidism)	• Lactose intolerance • Overlap with fatty small bowel disease etiologies • Medications (e.g., laxatives, magnesium supplements) • Sugar alcohols	• Irritable bowel syndrome • Functional diarrhea	• Altered bowel anatomy (e.g., Roux-Y) • Small bowel disease (e.g., celiac, small intestinal bacterial overgrowth, Whipple's, some parasites) • Pancreatic insufficiency	• Inflammatory bowel disease (i.e., Crohn's, Ulcerative colitis) • Ischemic colitis • Infectious (e.g., Salmonella, EHEC or shiga toxin E coli, Campy, Shigella, vibrio, yersenia, CMV) • Malignancy	• Microscopic Colitis • Radiation colitis • Infectious (e.g., C. perfingens, ETEC, Cryptosporidium, Cyclospora, C. diff, giardia, non-CMV viruses)

Inflammatory Bowel Disease

	Ulcerative Colitis	Crohn Disease
Clinical Manifestations	Diarrhea, tenesmus, urgency hematochezia, weight loss, fever	Abdominal pain, diarrhea, Inflammatory masses, fever, weight loss, intestinal strictures and fistulas
Extraintestinal Manifestations	Peripheral arthritis, spondylitis, sacroiliitis, ILD, PSC (UC) Oral aphthous ulcers, uveitis, iritis, episcleritis Pyoderma gangrenosum, erythema nodosum	
Workup and Diagnosis	CBC, CMP, ESR, CRP Stool: *C. difficile* toxin, *Shigella, Salmonella, Campylobacter, Escherichia*, O&P, Fecal calpro Quantiferon gold, chronic hepatitis panel, TPMT (pre-medication eval) OC +/- MRE/CTE for CD	
Histologic Features	Contiguous mucosal inflammation from the colorectum, clearly demarcated	Asymmetric transmural inflammation (cobblestone) that skips areas from mouth to anus, Granulomas
Treatment	Mild: Oral/topical 5-ASA, steroid PR/enema, Multimatrix budesonide Moderate: As above, plus AZA/6-mercaptopurine, oral glucocorticoids, Biologics (TNF-a, IL-23, etc), small molecules Severe: PO/IV steroids, cyclosporine, biologics, small molecules (updacitinib, tofacitinib), surgery	Mild: sulfasalazine for colitis, budesonide for IC disease Moderate: PO/IV steroids, AZA/6-mercaptopurine, MTX, biologics (TNF-a, IL-23, etc), small molecule (Upadacitinib) Severe: PO/IV steroids, biologics (TNF-a, IL-23, etc), small molecule (Upadacitinib)
General Health Considerations	CRC Screening: Colonoscopy 8 years after dx, q1-5 years thereafter; Colo for UC screen at time of PSC dx Vax: influenza, COVID-19, pneumonia, HAV/HBV, HPV +/-Shingrix; avoid live vaccines (MMR, varicella, FluMist) if on anti-TNF Smoking cessation, Avoid NSAIDs and opioids, DEXA if on >7.5mg of prednisone >3 months	

Irritable Bowel Syndrome (IBS)

<table>
<tr><td>Diagnosis</td><td colspan="4">Per Rome IV Criteria: Recurrent abdominal pain at least 1 day/wk in the last 3 mo, with ≥2 of the following:
1. Related to defecation (pain improves/worsens with BM)
2. A/w change in the frequency of stooling (more/less)
3. A/w change in the form/appearance of stool (Bristol Scale changes)</td></tr>
<tr><td>Subtypes</td><td>Constipation (IBS-C)
>25% BMs Bristol Types 1 or 2
<25% BMs Bristol Types 6 or 7</td><td>Diarrheal (IBS-D)
<25% BMs Bristol 1 or 2
>25% BMs Bristol 6 or 7</td><td>Mixed (IBS-M)
>25% BMs Bristol 1 or 2
>25% BMs Bristol 6 or 7</td><td>Unspecified (IBS-U)
Meet Rome IV criteria but not one specific subtype</td></tr>
<tr><td>Evaluation</td><td colspan="4">Rule out Red Flags!!
- Age>50, Bleeding, Nocturnal pain/diarrhea, Progressive abdominal pain, weight loss/fever/systemic symptoms, FDR with IBD or CRC
- Further work-up (labs, endoscopy, etc) if any of the above present
Once red flags are ruled out:
- Obtain CBC, TSH, & CRP (+ BMP, Giardia, Celiac panel, and fecal calpro for diarrheal subtype)
- If labs normal & Rome IV met → diagnosis made!</td></tr>
<tr><td rowspan="2">Treatment</td><td colspan="4">All Patients: Create strong, reliable patient-provider relationship with education and reassurance
Lifestyle modifications (exercise, sleep, stress reduction)
Increase soluble fiber intake (target >35g daily – SLOW uptitration!)
Low dose TCA, Gut-directed psychotherapy (CBT-GI, hypnotherapy) – NNT of 4
Peppermint oil (antispasmodic)
Trial exclusion diet (e.g. FODMAP)</td></tr>
<tr><td colspan="2">IBS-C
1. Osmotic Laxatives still first-line (can cause cramping!)
2. Secretagogues (GCA: linaclotide or plecanatide, or Chloride channel activator: Lubiprostone)
3. TCA, SNRI, Biofeedback therapy
4. Tegaserod (5HT4)</td><td colspan="2">IBS-D
1. Loperamide or bile acid sequestrant
2. Eluxadoline (mixed opioid ag/antagonist)
3. Rifaximin 14d trial – can lead to 10mo w/o sxs – can repeat
4. Alosetron (5HT3)</td></tr>
</table>

GERD and Dyspepsia

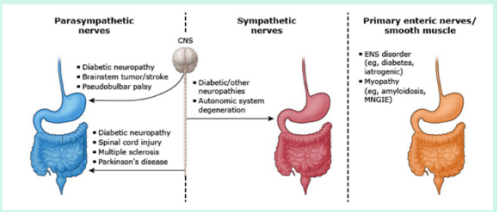
Definitions	GERD – reflux of gastric contents into the esophagus causing symptoms and/or complications Functional dyspepsia – bothersome postprandial fullness, early satiety, and epigastric pain or burning occurring 3d/wk for at least 6 mo WITHOUT evidence of structural disease (PseudoGERD)
Epi	~30% of adults in western society Causes: reduced LES pressure, hiatal hernia, increased intra-abdominal pressure (obesity, pregnancy), abnormal esophageal/gastric motility, Zollinger-Ellison syndrome; consider NSAIDs, other meds
Symptoms	Red Flag (need endoscopy!) – Weight loss, anorexia, GIB, recurrent vomiting, anemia, dysphagia, odynophagia, FH of gastric ca in FDR, New onset age >=60 yo Typical – retrosternal pyrosis (heartburn), regurgitation, epigastric discomfort - For typical symptoms only, manometry, barium swallow, laryngoscopy not needed Atypical esophageal – chest pain, nausea, dysphagia, odynophagia, eructation (belching), globus Extraesophageal – cough, hoarseness, throat clearing, dysphonia, dental caries, acidic taste
Evaluation	60 years or older – Endoscopy (EGD) <60 years old – h pylori stool antigen testing (2 weeks OFF PPI), 8 week PPI trial for typical sxs - If PPI trial effective, reduce dose to lowest effective dose - If PPI trial ineffective, evaluate medication timing and compliance before referring for endoscopy. If endoscopy negative, pursue ambulatory reflux monitoring and/or manometry
Treatment	GERD – 8 week trial of PPI, then as above Functional dyspepsia – EGD for alarm signs or if 60+ yo, rule out h pylori → PPI trial → TCA trial
PPI Risks	Mild documented increased risk of osteoporosis, hip fracture, CKD, CAP, c diff recurrence or index infection, dementia

Functional Dyspepsia: In Depth

Heartburn Differential					
Esophagitis	Gut-Brain	Structural	Motility	Cardiac	Psych
- GERD - Reflux hypersensitivity - Infectious esophagitis - Pill esophagitis - Eosinophilic esophagitis	- Functional dyspepsia	- Achalasia/EGJ outflow obstruction - Strictures / Webs - Esophageal tumor	- Distal esophageal spasm - Hypercontractile esophagus - Absent contraction - Esophageal dysmotility	- ACS - Stable angina	- Anxiety

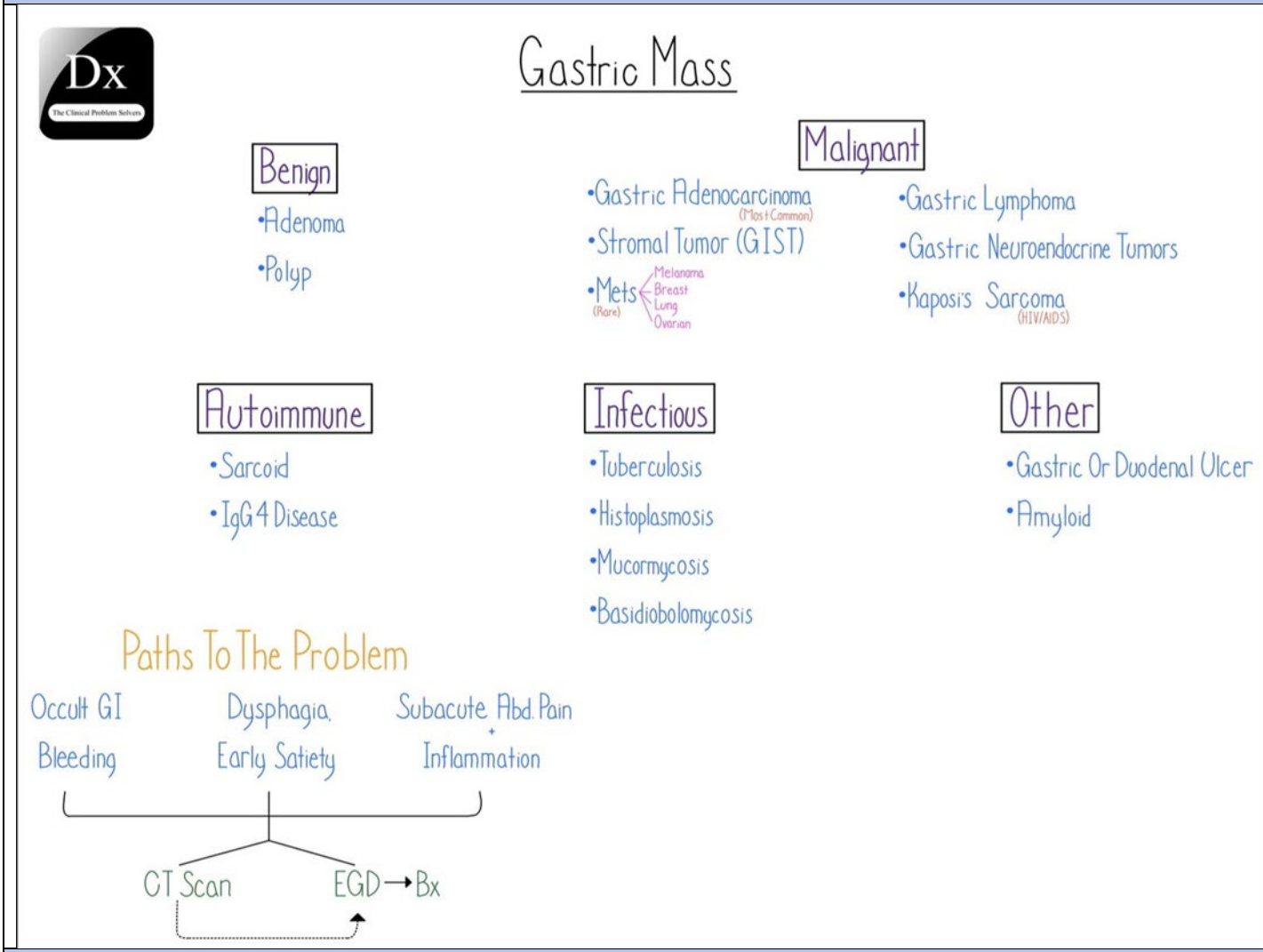
Evaluating Dyspepsia	
For uninvestigated dyspepsia:	1. Send H pylori stool antigen & begin 4-week course of PPI → eradicate H pylori if present 2. If no response at T+4 weeks, increase total daily dose of PPI x 4 weeks 3. If no response at T+8 weeks, obtain EGD (GO STRAIGHT TO EGD IF: alarm sxs, overt GIB, or age≥60!) - Some will trial a TCA prior to EGD → if it works, can avoid further testing → functional heartburn - Biopsies to rule out EoE → treat accordingly if EGD abnormal 4. If EGD is normal, obtain ambulatory pH or pH impedance monitoring (OFF PPI therapy!) 5. If Acid Exposure Time (AET) <4% or Reflux-Symptom Assn (RSA) is negative, get high res manometry 6. If no major motor disorder observed on high resolution manometry → functional heartburn diagnosed!
Functional Heartburn	Diagnosed with: Symptoms at least 2x/week for the last months, with onset ≥6 months ago Must include ALL of the following: - Burning retrosternal discomfort or pain - No symptom relief despite optimal antisecretory therapy - Lack of evidence of GERD or EoE - Absence of major esophageal motor disorders Treatment: - Provider reassurance, validation of symptoms, avoid repeat invasive testing - TCA (Elavil), SSRI (fluoxetine), 5-HT4 serotoninergic (Tegaserod), Melatonin (QoL)

Gastroparesis

Definition	Objectively delayed gastric emptying of solids in the absence of mechanical obstruction Associated with N/V, early satiety, eructation, bloating, upper abdominal pain
Etiology	**IDIOPATHIC** Most common cause! <50% of pts **POST-VIRAL** Norwalk, Rotarvirus Often improves w/i 1 yr (may not if a/w CMV, EBV, VZV) **IATROGENIC** **Meds** Opioids CCB, clonidine Dopaminergics Antimuscarinics GLP1s Cannabinoids TCAs **Post-Surgical** Gastric or Thoracic sx Post-ablation **SYSTEMIC/NEURO DISEASE**  Diabetic neuropathy but many other neurodegenerative dz Systemic sclerosis Autoimmune disease
Diagnosis	1. Exclude mechanical obstruction - Upper endoscopy - CTE or MRE (small bowel mass, SMA s/o) 2. Gastric scintigraphy (preferred) or 13C breath test - Stop gastric motility-affecting agents 48h prior; test should last 3-4 hrs - BG <275 during testing - >10% retained food at 4 hours = diagnostic on emptying study! (>60% at 2 hours suggestive)
Treatment	- Correct nutritional deficiencies, hydration, maintain glycemic control - Small, frequent, low-fat, low-fiber meals - Metoclopramide at lowest effective dose (FDA) – watch for extrapyramidal symptoms - Erythromycin for flare/short-term use

Gastric Masses

Gastric Masses Schema



Gastric Adenocarcinoma

Types:

- Intestinal-type (most common), and diffuse-type

Risk Factors:

- H. pylori infection = primary nongenetic risk factor
- Male, ethnicity, geography, diet (smoked, salted, pickled, nitrates, nitrites), Smoking, obesity
- Prior stomach surgery, chronic atrophic gastritis, hereditary syndromes (Lynch, FAP, e.g.)
- Gastric intestinal metaplasia and dysplasia

Symptoms: Most patients are asymptomatic!

- Presenting symptoms in later stages:
    - Pain (mild early, then severe as disease progresses)
    - Weight loss – secondary to insufficient caloric intake rather than catabolism
    - Nausea, Early satiety – 2/2 tumor mass, inability of stomach to distend
    - GIB w/wo IDA not uncommon (melena, hematemesis <20% of cases)

Physical Exam

- Uncommon but may include: palpable mass, Virchow's (supraclavicular) lymph node, ascites, jaundice.

Elevated LAEs on LFT

Markers of Liver Injury	Markers of Liver Function
AST, ALT, Alk Phos, GGT, Bilirubin	Albumin, Platelets, INR, Clotting Factors

R = Patient's ALT / ULN* ALT / Patient's ALP / ULN* ALP

Initial Evaluation: R-factor

- R ≥ 5: hepatocellular
- R ≤ 2: cholestatic
- R between 2 and 5: mixed (hepatocellular and cholestatic)

Hepatocellular (R ≥ 5)	Mixed (2 < R < 5)	Cholestatic (R ≤ 2)
Alcohol-associated, Viral, or Autoimmune hepatitis; Wilson disease, Alpha-1-antitrypsin, Hemochromatosis, Budd Chiari, MASLD	Drug-induced liver injury, viral hepatitis, AIH	Ductal obstruction (Choledocholithiasis, biliary stricture, pancreaticobiliary tumor) Primary biliary cholangitis (PBC) Primary sclerosing cholangitis (PSC)

Isolated Alk Phos Hi ULN = 105

History & Physical
GGT
→ If GGT abnormal, RUQUS
AMA, ANA, ASMA
→ Negative eval & >2x ULN = bx
Else, observation vs MRCP/ERCP

Isolated Bili Elevation

Fractionated Bili
→ If high direct bili, RUQUS
? Sepsis, TPN, cirrhosis, obstruction
→ MRCP/ERCP (ductal dil.)
Or
AMA, ANA, ASMA (duct nl)

AST/ALT ULN = 35

Borderline <2x ULN
Mild 2-5x ULN
→ H&P
D/c hepatotoxins, EtOH
Assess risk for MASLD, viral hep
→ CBC
LFT
PT/INR
HBV, HCV Ab
Iron panel
RUQUS
→ If persistently elevated at 3-6 mo →
ANA, ASMA, IgG, Ceruloplasmin, A1AT

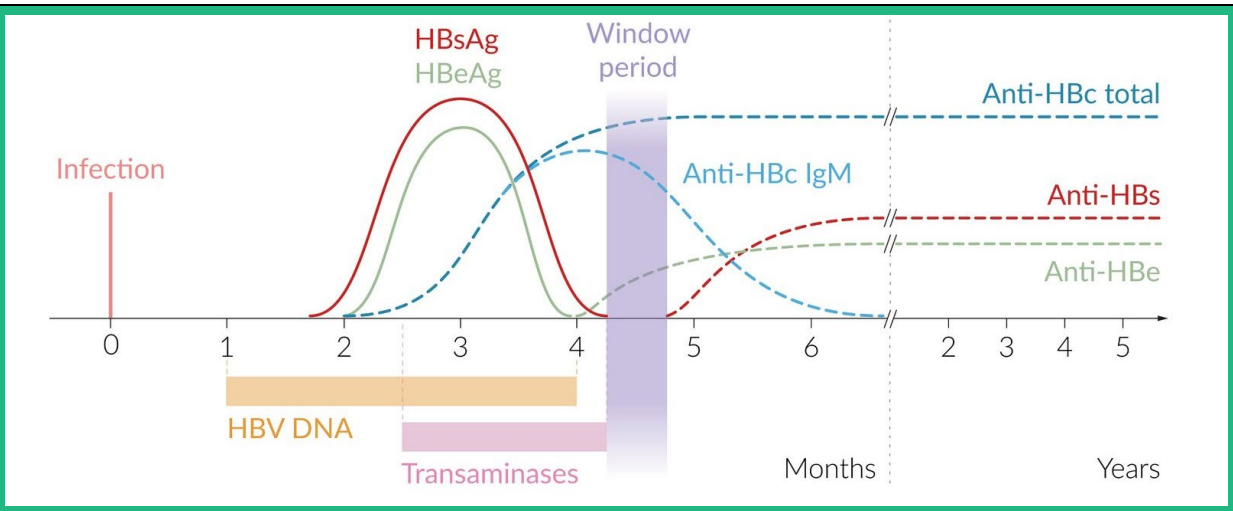
If no dx, consider bx

Moderate 5-15x ULN
Severe >15x ULN
→ H&P
D/c hepatotoxins, EtOH
Eval for signs of ALF
→ As above
HAV IgM/IgG
ANA, ASMA, IgG, Ceruloplasmin
→ If ALF→ transplant eval

Consider biopsy

Massive ALT >10,000
→ H&P, d/c hepatotoxins
Toxic ingestion, ischemia, rhabdo eval
Eval for signs of ALF
→ As above, Anti-LKM, EBV, CMV, HSV, Toxicology, Doppler US

HBV Serology Mastery

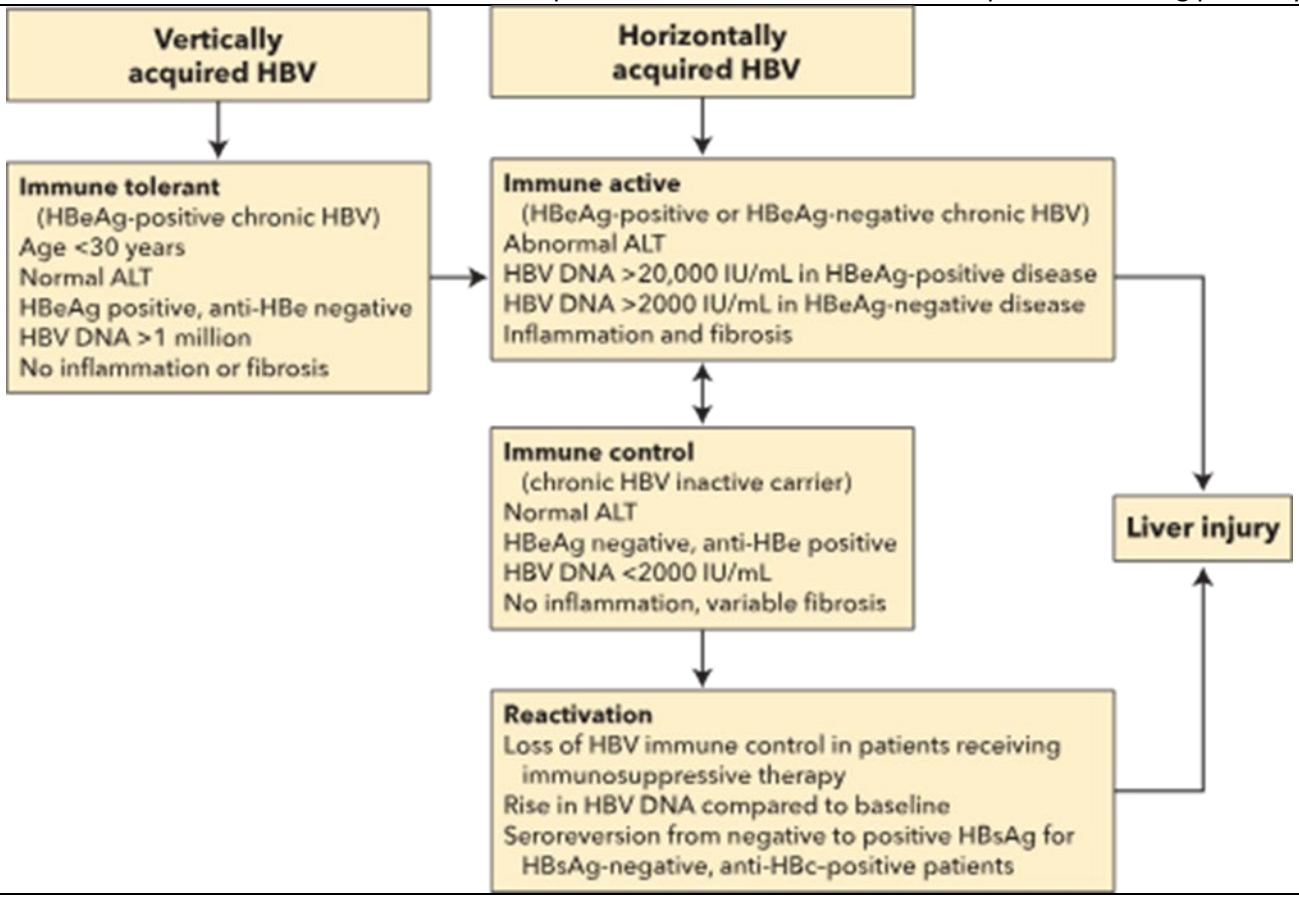


Status	HBsAg	HBsAb	HBcAb IgG	HBeAg	HBeAb	HBV DNA PCR
Susceptible	-	-	-	-	-	-
Immunized	-	+	-	-	-	-
Early Infection	+	-	-	+	-	↑↑↑
Window period (infection)	-	-	+	+/-	+	↑↑
Cleared infection	-	+	+	-	+	-
Chronic infection	+	-	+	+/-	+	↑↑

Hepatitis B Serologies: Alternate

	HBsAg	Anti-HBs	Anti-HBc	HBeAg	Anti-HBe	HBV DNA	ALT
Susceptible	-	-	-	-	-	-	Normal
Immunized	-	+	-	-	-	-	Normal
Acute infection							
Early	+	-	IgM	+	-	+	Elevated
Window	-	-	IgM/IgG	-	-	+	Elevated
Recovery		+	IgG		+	+/-	Normal
Resolved Infection							
Resolved	-	+	IgG	-	-	-	Normal
Chronic Infection Phases							
Immune tolerant	+	-	+IgG	+	-	High DNA	Nl or mild elevation
Immune active	+	-	+IgG	+/-	-	HBeAg + higher levels of DNA	Elevated
Inactive chronic	+	-	+IgG	-	+(or -)	Low DNA	Normal
+Occult HBV							
Occult HBV	-	+/-	Usually +	-	+/-	Liver	Normal

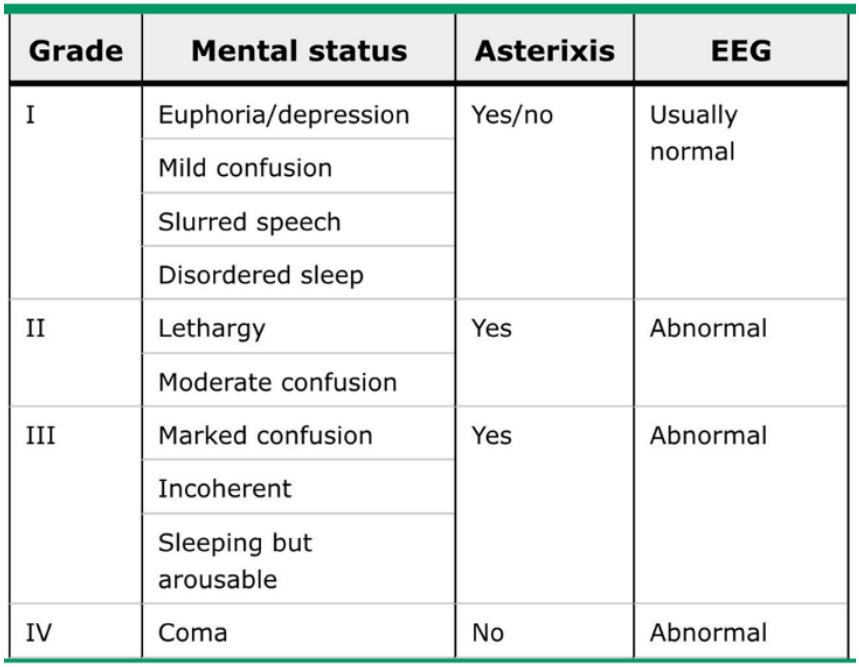
Hepatitis B Infection

Virus	Double stranded DNA para-retrovirus – has reverse transcriptase replication!
Risk Factors	Vertical transmission (pregnancy – highest risk!) High-risk sexual activity Blood borne (IVDU, transfusion, percutaneous) Unvaxxed Incarceration Starting immunosuppression Hemodialysis
Presentation	Acute infection – may be asymptomatic or present as acute hepatitis (few will have ALF) May be spontaneously cleared or be chronic infection Chronic HBV == risk factor for cirrhosis and hepatocellular carcinoma == 6 months persistent HBsAg positivity
Chronic HBV Phases	
Screening	ALL adults x1 and in first trimester of each pregnancy – HbsAg, HBsAb, HBcAb IgG
Treatment	Entecavir or tenofovir (first-line) - Indications: acute liver failure, immune-active or reactivation phases, cirrhosis, select immunosuppression, pregnant patients in 3rd trimester with VL greater than 200,000 U/mL
Prophylaxis	Oral antiviral therapy for patients who are: A. HBsAg-positive or isolated HBcAb–positive AND B. Receiving B-cell–depleting tx (e.g., rituximab), prednisone (≥10 mg x4 wks), or anthracyclines PLUS/MINUS C. Receiving TNF-α or TKIs (consider)

Acute Liver Failure vs Cirrhosis

Acute Liver Failure	Cirrhosis
1. Duration of ≤26 weeks without prior chronic liver dz 2. Elevated LAE, jaundice, thrombocytopenia, etc 3. INR ≥1.5 **4.** Hepatic encephalopathy	Chronic fibrosis of liver parenchyma (imaging or biopsy) Sxs: organomegaly, caput medusae, gynecomastia, testicular atrophy, palmar erythema, spider angiomata, jaundice, fetor hepaticus, parotid hypertrophy, contractures, edema, soft BP 1. Compensated a. Asymptomatic or vague constitutional sxs 2. Decompensated **a.** Ascites, SBP, HE, Varices, Portal HTN, HRS, HPS, HCC
ALF Etiologies	**Cirrhosis Etiologies**
DILI (APAP, antimicrobials, anti-TB, supplements, MDMA) Viral hepatitis (A, B +- D, E) HSV, CMV, VZV, EBV AIH, Wilson's Malignancy, ischemia, Budd-Chiari, HH AFLP, HELLP	Medications (Amio, MTX, INH) HBV, HCV Schistosomiasis, Tick-borne illness AIH, PBC, PSC, IGG4, Wilson's, HH, A1AT EtOH, MASLD Right-sided CHF, HHT, Sarcoid

Hepatic Encephalopathy

Definition	Reversible impairment of neuropsychiatric function a/w impaired hepatic function and increased ammonia concentration
Risk Factors	Drugs (BZD, opiate); Increased ammonia, GIB, Dehydration, Vascular occlusion
Classification	
Treatment	Reverse the underlying cause! Ensure adequate hydration, nutrition, falls precautions Start Lactulose PO or PR (long term therapy) + Rifaximin (at least 3 mo) – Titrate to 2-3 BM per day

Grade	Mental status	Asterixis	EEG
I	Euphoria/depression	Yes/no	Usually normal
	Mild confusion		
	Slurred speech		
	Disordered sleep		
II	Lethargy	Yes	Abnormal
	Moderate confusion		
III	Marked confusion	Yes	Abnormal
	Incoherent		
	Sleeping but arousable		
IV	Coma	No	Abnormal

New Onset Ascites Evaluation

<table>
<tr><td>Diagnosis</td><td colspan="2">Accumulation of free fluid in the abdomen
<u>Grade 1</u>: detectable only by US (as little as 100 mL)
<u>Grade 2:</u> detectable by physical exam (shifting dullness, fluid wave, e.g.)
<u>Grade 3:</u> Marked abdominal distension</td></tr>
<tr><td>Evaluation:

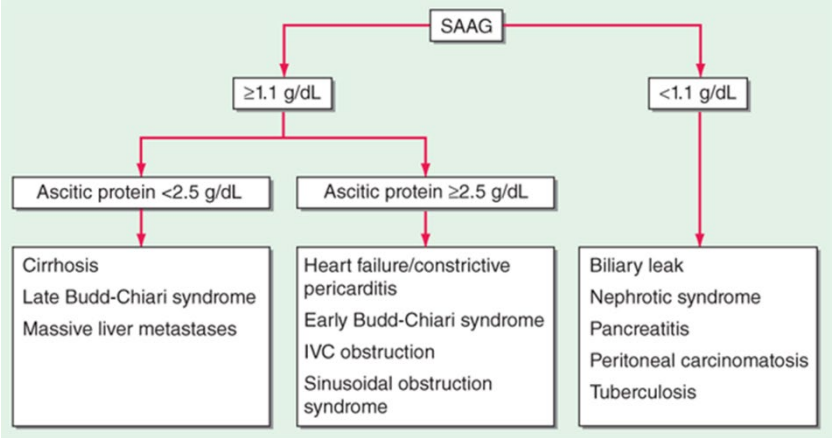
Diagnostic Paracentesis</td><td><u>Appearance:</u>
1. “Coors Light” – Minimal foam, transparent - transudate
2. “Blue Moon” – foam, slightly murky but light – long-standing transudate vs exudate
3. “Amber ale” – exudate (parapneumonic effusion vs Ca vs infarct vs trauma)
4. “Stout” – dark brown – likely exudate (blood, Ca, aspergillus, amoebic liver abscess rupture)
5. “Milk” – chylothorax due to thoracic duct injury</td><td><u>Ascitic Fluid Labs:</u>
Cell count (PMN ≥250 = SBP) with gram stain
Body fluid culture
Albumin, total protein, LDH (order serum equivalents)
± TG (if concern for chylo)

<u>Initial imaging:</u>
POCUS to confirm pocket for tap
Formal complete abdominal US + doppler</td></tr>
<tr><td>Etiologies

#1 cause = decompensated cirrhosis (84%)

Cardiac, peritoneal carcinomatosis, “mixed” (10-15%)</td><td colspan="2">1. Rule in or out SBP based on PMN count → tx with CTX or FQ + IV albumin

2. Calculate a Serum-Ascites Albumin Gradient (SAAG)
= Alb (serum) – Alb (ascites)
Higher gradient ← high hydrostatic pressure (portal HTN) OR low oncotic pressure (decr protein production)

3. Additional evaluation per suspected primary etiology
(e.g. liver imaging/elastography/coags, TTE, 24hr urine protein/lipids, AFB/ADA, laparoscopy, e.g.)
 
</td></tr>
</table>

Budd-Chari Syndrome

Pathophys	<u>Hepatic vein outflow obstruction from</u> A. Thrombus (majority – hypercoagulable state, APLS, OCPs, polycythemia or MPN) OR B. Obstructive mass (HCC, RCC, hepatic cyst, aspergilloma, e.g.)
Suspect	Acute liver failure, acute hepatitis / marked AST/ALT elevation (>1000), or chronic liver disease with risk factors
Diagnosis	<u>RUQUS with doppler diagnostic in majority of cases</u> CT or MRI also options (especially to identify mass)
Tx - Treat underlying d/o!	<u>Prevent propagation:</u> Anticoagulation = Warfarin (LWMH bridge) – need variceal evaluation first! <u>Restore patency</u>: directed thrombolysis vs venous stenting <u>Liver decompression:</u> TIPS or surgical shunt

Hepatorenal Syndrome

Diagnosis

Cirrhosis with ascites

Diagnosis of AKI according to ICA-AKI criteria: acute increase in sCr ≥0.3 mg/dL (≥26.5 μmol/L) within 48 h; or, a percentage increase in sCr ≥50% from baseline that is known, or presumed, to have occurred within the prior 7 d[a]

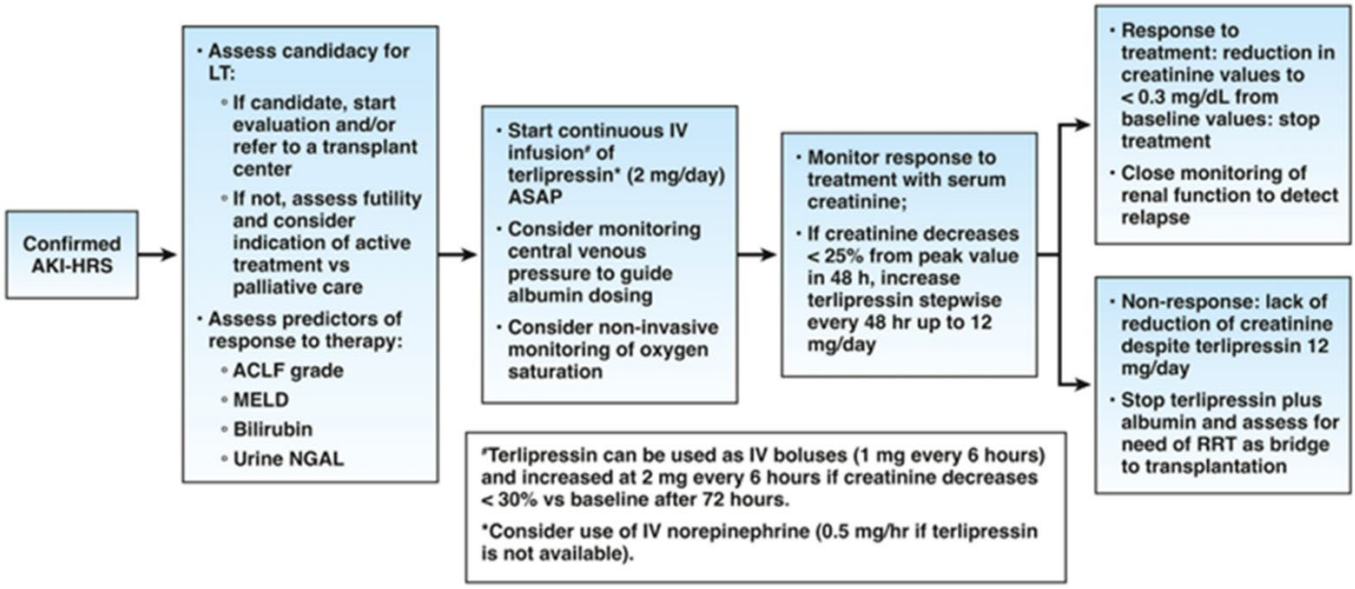
No response after 2 consecutive days of diuretic withdrawal and plasma volume expansion with albumin (1 g/kg of body weight, with a maximum of 100 g/d)

Absence of shock

No current or recent use of nephrotoxic drugs (NSAIDs, aminoglycosides, iodinated contrast media, etc)

No macroscopic signs of structural kidney injury, defined as:
Absence of proteinuria (>500 mg/d)
Absence of microhaematuria (>50 RBCs per high-power field)
Normal findings on renal ultrasonography

Management



General Internal Medicine

On Duty Determination

Duty Determination	Duty fitness or limitations that are individualized to patient's branch, duty location, job requirements/MOS/rate, and special duties (flight, dive, e.g.) -- NUANCED
Importance	Protecting patients and their units from unnecessary harms, provides formal communication between medical and the command/employer
Where can I find more info?	DoDI 1332.45 DoDI 6130.03 OPNAVINST 1300.20 USA - Guide for Physical Profiling, MOS/Medical Retention Boards, MEB, PEB USAF - AFI 48-133 USAF - AAM Guide

Patient Medical Decision Making

	Capacity	Competency
Definition	A person's ability to make an informed decision	The mental soundness to make decisions or act
Domain	Case-by-case decisions Patients may have capacity on one issue but not another (e.g. lab draws vs. surgical consent)	Global – ALL decisions
Determined by	YOU! Any physician, not just psych can evaluate Some consult psych to defer medicolegal risk	Legal system (i.e. a judge) Typically protracted (e.g. guardianship)
How Determined	"4 C's" in the clinical setting Choice – clearly indicated a preferred tx option Case Comprehension – pt understands their medical scenario Consequences – pt knows pro/con of all tx options Consideration – pt can reason through relevant information	Complex legal proceedings in court - May include financial decision making, power of attorney, residency,

Medical Genetics

When to Refer to Genetic Counseling
• If you are suspicious of a heritable disease, based on: • Strong family history (3-2-1-1 Rule = ≥3 family members, in 2 generations, 1 FDR of the other 2, ≥1 before 50 yo) • Birth defects or developmental delay • Cancer • Concern for potential genetic risk for offspring • Reproductive health concerns

Annual Exams

Cancer			
	Age	Frequency	Modality
Breast	Pts with breasts 40-74 years	Q2year	Mammogram
Cervical	Pts with cervices 21-29 yo* Pts with cervices 30-65 yo	Q3year* Q3yr Pap Q5yr HPV or co-test	Pap*, HPV, or Co-testing
Colon	Adults 45 – 75 yo (average risk) Adults at 40 or 10 yrs prior to first degree relative diagnosis age*	Q1yr (FIT) q5yr (VC) q10yr (OC)*	FIT, Cologuard, Virtual or Optical Colonoscopy*
Lung	Adults 50-80 yo with 20 pk-yrs, currently smoking or quit within last 15 yrs	Annual	Low-dose lung CT
Prostate	Grade C – Pts with prostates 55-60 yo	Annual	PSA
Immunizations			
	Age	Frequency	Modality
Pneumococcal	Age >65, OR <65 if comorbidities (DM, Tobacco/EtOH, immunocompromise, eg)	One time OR 1 year apart ->	PCV-20 OR PCV-15, PPSV23
Meningococcal	All adults (Men B for 19-23), esp with immunocompromise/asplenia	Multiple doses	MenACWY x 1-2 MenB x 2-3
HPV	Age 18-45	3 doses	Gardasil (9-valent vax)
Tdap	Age 18+	Q10 years, qPregnancy	Td or Tdap
Herpes Zoster	Age >= 50 yo	2 doses	Shingrix
RSV	Age >=60 yo (or 32-36 wk preg. Sep-Jan)	One time	RSV
Infectious Disease Screening			
	Age	Frequency	Modality
HIV	Patients 15-65 yo	At least once, then by sexual activity/risk level	HIV Ab immunoassay/p24 Antigen
HBV	All adults >18 yo	One time, then by risk level	HBV Surface Antigen and Ab, Anti-HBV Core Ab
HCV	Patients 18-79 yo	One time	Anti-HCV Ab
Latent TB	At risk adults	One time	TST or Quantiferon
STI (Syphilis, GC/CT)	Sexually active adults	Q3-12mo depending on risk level, PrEP Rx, etc	RPR, GC/CT NAAT at receptive sites
Other			
	Age	Frequency	Modality
AAA	Males age 65-75 who have smoked >100 cigarettes (= 5 packs)	One time	Abdominal Ultrasound
Osteoporosis	Women age >65 yo at average risk Women <65 yo at high risk (parental frx, steroid use>3mo, Current smoker, heavy EtOH, low BMI)	At least once	DEXA scan
Depression & Anxiety	All adults	Annually	PHQ-2 GAD-2
Lipids	Adults age >35 yo	Q10years	Lipid panel
Pre-DM and T2DM	Adults 35-70 yo with overweight or obesity	Q3years	Hgb A1c
Weight loss	Adults with BMI>=30	Annually	Behavior/Meds Rx

Sample Screening Tools for SDOH

Financial Burdens	Comprehensive Score for Financial Toxicity – Functional Assessment of Chronic Illness Therapy (COST-FACIT) - 11-question survey
Housing Insecurity	Housing Stability and Crisis Response (HSCR) – VA-developed tool, evaluates last 2 months and next 2 mo - 2-question survey
Low Health Literacy	Short Assessment of Health Literacy (SAHL) – language-specific versions available - 18-question test Brief Health Literacy Screen (BHLS) - 3-question survey
Social Isolation	Patient-Reported Outcomes Measurement Information System (PROMIS) – Social Isolation - Multiple versions, including a 4-question short form

Diabetic Foot Exam

When to screen for neuropathy	T1DM: 5 years after diagnosis, then annually T2DM: At time of diagnosis & annually
Necessary Exam Components	DERM - Skin thickness, color, sweating, infection, ulceration, calluses MSK - Derformity - Muscle wasting NEURO - Monofilament test + 1 of the following: - Vibration OR - Pinprick sensation OR - Ankle reflexes OR - Vibration-Perception Threshold (VPT) VASCULAR - Pulses - ABI if indicated
What to do if abnormal	ALL patients with diabetes should receive counseling on foot hygiene/care Referral/further diagnostics per abnormal issue, low threshold to send to Podiatry
Receiving credit for your work	Order "Foot Examination Performed 2028F"

Colorectal Cancer Screening

Who	**Average Risk =** Begin screening at 45 years old (suggested) to 50 years old (recommended) – variety of modalities/frequencies - CRC or advanced polyps in a second-degree relative does NOT confer elevated risk **Higher Risk =** Begin screening at 40 years old or 10 years prior to diagnosis of CRC/advanced polyp in first-degree relative (whichever is earlier) – q5year colonoscopy Consider stopping at 75 years old, especially if multiple comorbidities/lower life expectancy
How	Tier 1: • Colonoscopy every 10 years • Fecal immunohistochemical testing (FIT) annually – cheap, easy – necessitates colo if positive Tier 2: • Flexible sigmoidoscopy every 5-10 years • Multitarget stool DNA test (AKA Cologuard) every 3 years • CT Colonography every 5 years – requires bowel prep but no sedation – necessitates colo if positive • Colon capsule every 5 years **Reminder:** abnormal findings will turn CRC screening into polyp surveillance, e.g. and follow a different frequency per GI recommendations

Cardiometabolic Health

<table>
<tr><th colspan="5">Chronic Disease Development Pathway</th></tr>
<tr><td>Genetics & Development</td><td>Unhealthy Behaviors
- Poor Nutrition
- Physical inactivity
- Poor Sleep
- Chronic stress

Energy Balance Model vs Carbohydrate Insulin Model →</td><td colspan="2">Metabolic Dysfunction
- Impaired glucose tolerance
- Increased waist circumference
- HTN
- Dyslipidemia (HDL<50 in F, <40 in Men, TG >150)

Metabolic syndrome →
Hyperinsulinemia/resistance →</td><td>Development of Chronic Disease
MASLD T2DM CAD Gout OSA Infertility CHF Cancer OA PCOS CKD Chronic pain Fatigue Stroke Dementia</td></tr>
<tr><td>Paradigm</td><td colspan="4">Shifting upstream into behaviors and metabolic dysfunction == reducing chronic disease burden
BUT “Not as easy as move more, eat less” !</td></tr>
<tr><td>Analytes</td><td colspan="4">
<table>
<tr><th>Cardiometabolic Component</th><th>Optimal</th><th>Intermediate</th><th>Poor</th></tr>
<tr><td>Adiposity[b]</td><td>BMI <25 kg/m^2 AND WC ≤88 cm (women)/WC ≤102 cm (men)</td><td>BMI 25-30 kg/m^2 AND WC ≤88 cm (women)/WC ≤102 cm (men)</td><td>BMI >30 kg/m^2 OR WC >88 cm (women)/WC>102 cm (men)</td></tr>
<tr><td>Blood glucose[c,d]</td><td>FPG <100 mg/dL AND HbA_{1c} <5.7% AND NOT taking diabetes medication</td><td>FPG 100-125 mg/dL OR HbA_{1c} 5.7%-6.4% OR FPG <100 mg/dL AND HbA_{1c} <5.7% AND taking diabetes medication</td><td>FPG ≥126 mg/dL OR HbA_{1c} ≥6.5%</td></tr>
<tr><td>Blood lipids[e,f]</td><td>TC:HDL <3.5:1 AND NOT taking lipid-lowering medication</td><td>TC:HDL 3.5-5:1 OR TC:HDL <3.5:1 AND taking lipid-lowering medication</td><td>TC:HDL >5:1</td></tr>
<tr><td>Blood pressure[g]</td><td>SBP <120 AND DBP <80 AND NOT taking BP-lowering medications</td><td>(SBP 120-139 OR DBP 80-89) OR (SBP <120 AND DBP <80 AND taking BP-lowering medications)</td><td>SBP ≥140 OR DBP ≥90</td></tr>
<tr><td>History of CVD[h]</td><td>None of the listed conditions</td><td>Angina only</td><td>One or more of CHD, myocardial infarction, heart failure, stroke</td></tr>
</table>
</td></tr>
<tr><td>Targeted Assessment</td><td colspan="4">
<table>
<tr><th>Nutrition</th><th>Physical Activity</th><th>Sleep</th><th>Stress & Mental Health</th></tr>
<tr><td>▪ Meal/snack pattern (timing and composition)
▪ Liquid calories (juice, sodas, alcohol)
▪ Cravings and food noise
▪ Eating behaviors (emotional/stress eating, night eating, binging, purging, etc.)</td><td>▪ Non-Exercise Activity Thermogenesis (NEAT) – i.e. low intensity walking, taking the stairs, light yardwork, fidgeting, etc.
▪ Exercise – aerobic exercise, strength/resistance training, balance/flexibility</td><td>▪ Quantity
▪ Quality
▪ Consistency</td><td>▪ Past or present chronic stress from job, family situation, health, etc.
▪ Any signs or symptoms consistent with a primary mood disorder?
▪ Other social/contextual factors</td></tr>
</table>
*Can use SCOFF questionnaire for eating disorders
Body Composition – BMI (different, lower cutoffs for Asian patients), DEXA or InBody analysis
Labs – TSH w/ reflex FT4, CBC, CMP, fasting lipids, HgbA1c (consider ApoB, Lp(a), fasting insulin, cortisol, IGF-1)
</td></tr>
<tr><td>Interventions</td><td colspan="4">Nutrition – focus on food quality (unprocessed and minimall processed), eliminate liquid calories, mindful/intuitive eating, caloric restriction or tracking with app, dietitian referral!
Activity – Decrease sedentary time & increase non-exercise activity (aka get your steps in!), mix of aerobic and strength training, start slow and incorporate more, can refer to Exercise physiologist (Mr Combest at WR)
Sleep – CBT-I, other sleep trackers, Mind-body medicine, yoga/stress management (Calm, PCBH)
Tactical Medication Lever – many meds may work, not just GLP1! Consider comorbidities, potential AEs, lab monitoring needs, titration schedule feasibility – encourage protein intake and strength training! Consider dedicated IM referral for Weight Management, Clinical Pharmacy, GI clinic for endoscopic tx or Bariatric Surgery
■ See Google Drive from Taylor Neuman for full resources list including educational handouts for pts</td></tr>
</table>

Anti-Obesity Medications

Agent (Trade Name)	MOA & Route	%Body Weight Loss	Contraindications	Caution/Monitor	Side Effects
Phetermine (Apidex-P, Lomaira) 8mg up to TID 15mg 30mg 37.5mg	NE-releasing agent; CNS stimulant acting on hypothalamus PO	5-7%	CVD Hyperthyroidism Uncontrolled HTN Glaucoma Pregnancy Agitated state	Anxiety disorder Seizure history Primary pHTN *FDA approved 3 months but can use longer*	HA, insomnia, dry mouth, anxiety, constipation, palpitations
Phentermine+ topiramate (Qsymia) 3.75/23mg 7.5/46mg 11.25/69mg 15/92mg *migraines, seizure ppx*	NE-releasing agent; CNS stimulant + GABA receptor agonist PO	7.8-10.9%	Hyperthyroidism Glaucoma Recent MAOi use Pregnancy GFR <30	Fetal cleft lip/palate Suicidal ideation Depression/anxiety Increased HR, BP Cognitive impairment Kidney stones ASCVD, HTN GFR 30-49	Paresthesia (1/3 pts), word recall issues, dizziness, dysgeusia, insomnia, constipation, dry mouth
Orlistat (Alli, Xenical) 60mg TID w/ meals 120mg TID w/ meals *Relieve constipation, esp a/w GLP-1 use*	Lipase inhibitor PO	3.3% at 120mg dose	Chronic malabsorption Cholestasis Pregnancy	Liver dysfunction Renal impairment Malabsorption of fat-soluble vitamins & medications (Vit D) Kidney stones, IBS-D	Steatorrhea, flatulence with discharge, fecal urgency + incontinence, increased stools
Naltrexone SR/Bupropion SR (Contrave) 8/90mg 1-4tabs/d *MDD, TUD, food cravings*	Opiate antagonist + DA and NE reuptake inhibitor PO	6.4%	Uncontrolled HTN Sz disorder Anorexia or Bulimia Alcohol w/d Opioid use d/o Pregnancy GFR<30	Suicidal Ideation Depression Anxiety Concomitant MAOi use Liver dysfunction Glaucoma GFR 30-49	N/V, HA, constipation, dizziness, insomnia, dry mouth, diarrhea, ↑ BP & HR
Liraglutide (Saxenda) 0.6-3mg titrate weekly to 3mg daily *Pre-DM, T2DM, CVD*	GLP1 receptor agonist SubQ daily	~8%	PMH or FMH of MEN2 or medullary thyroid cancer Pregnancy	Pancreatitis Acute gallbladder disease Cholelithiasis	fatigue, N/V/D abdominal pain, constipation (hypoglycemia in T2DM pts)
Semaglutide (Wegovy) 0.25-2.4mg titrate every 4 weeks *Pre-DM, T2DM, CVD, NAFLD*	GLP1 receptor agonist SubQ weekly	12-15%	PMH or FMH of MEN2 or medullary thyroid cancer Pregnancy	Pancreatitis Acute gallbladder disease Cholelithiasis	fatigue, N/V/D, abdominal pain, constipation (hypoglycemia in T2DM pts), sinus tach
Tirzepatide (Zepbound; Mounjaro for T2DM) 2.5-15mg titrate every 4 weeks by 2.5mg *T2DM, CVD, NAFLD, less GERD than GLP-1*	GIP + GLP1 receptor agonists SubQ weekly	16-25% on avg	PMH or FMH of MEN2 or medullary thyroid cancer Pregnancy	Pancreatitis Acute gallbladder disease Cholelithiasis	fatigue, N/V/D, abdominal pain, constipation, (hypoglycemia in T2DM pts), sinus tach, ↓OCP effectiveness

Somatic Symptom and Related Disorders

Disorder	Classic Symptom	Associated Symptoms	Epidemiology	Time Course
Somatic Symptom Disorder	Somatic symptoms affecting any body system without identifiable etiology (not beholden to exhaustive work-up once clinical suspicion wanes)	Excessive thoughts, feelings, or behaviors about sxs	~4% prevalence; F>M? 50% remit w/i 1 year Increased risk for iatrogenic harm 2/2 unnecessary testing or procedures	≥6 mo
Illness Anxiety Disorder	Preoccupation with having/acquiring illness with minimal/no sxs	Excessive health-related behavior OR care avoidance	?a/w childhood illness or trauma, M predominant	≥6 mo
Functional Neurologic Disorder *(prev. Conversion Disorder)*	Voluntary motor or sensory neurological symptoms incompatible with medical findings	Seizure-like activity, weakness, paresthesias, often function limiting	4-12 per 100K pts F>M; often with comorbid Neuro dz PNES <1/3 of EMU admits	No specific time frame (dx often takes mos)
Factitious Disorder	Intentional feigning of symptoms for psychological gain	Deceptive behavior without clear external reward	Unknown, est. 1% Often 30-40 yo F>M? HCW, Psych hx also	Single episode vs recurrent
Treatment Paradigm	• First do no harm == follow appropriate work-up algorithm for patient's symptomatology, involve specialty care as needed – however, these are NOT diagnoses of exclusion per se • **Communication, reassurance, and consistent follow-up with the same provider** • Education about the condition • Treat underlying behavioral health conditions (SSRI/SNRI, etc) • Early involvement of behavioral health – with patient consent (CBT, ACT, Mindfulness)			

Anxiety Disorders

Disorder	Key/Differentiating Factors	Time Course	Screening Tools	Work-Up to Consider	Psychopharmacology*	When to Refer
GAD	• Uncontrollable worry/fear about several different things • Somatic sxs: Muscle tightness, holding breath, fatigue, restlessness, and sleep disturbance	≥ 6 months	• GAD-2 • GAD-7	• Screen for MDD • Screen for SUD • Consider medical work-up in select cases	• Sertraline (Zoloft) 100-200 mg PO daily • Escitalopram (Lexapro) 15-20 mg PO daily • Venlafaxine (Effexor) 75-225 mg PO daily • Duloxetine 60 mg PO daily • Paroxetine+ (Paxil) 20-60 mg PO daily	• Refractory to multiple medication trials • Patient interested in therapy
SAD	• Anxiety pertaining social situations with risk of scrutiny or judgment • Avoidant of or has marked distress due to social situations	≥ 6 months	• Mini-SPIN • SPIN	• Screen for MDD • Screen for SUD • Consider medical work-up in select cases	• Sertraline (Zoloft) 100-200 mg PO daily • Venlafaxine (Effexor) 75-225 mg PO daily • Performance anxiety: Propranolol IR 10-20 mg PO 30-60 minutes prior • Paroxetine+ (Paxil) 20-60 mg PO daily	• Refractory to multiple medication trials • Patient interested in therapy
PD	• Recurrent panic attacks described as unexpected waves of anxiety • Persistent concern about additional attacks • Significant maladaptive behavior related to attacks	≥ 1 month	Panic Disorder Severity Scale	• Screen for MDD • Screen for SUD • Screen for other anxiety disorders • Medical work-up for somatic symptoms as appropriate	• SSRI • Augment: ○ Hx of SUD: Gabapentin ○ Not at increased risk for SUD: Long-acting BZD (e.g., clonazepam)	• Immediately for intensive CBT if patient is motivated
OCD	• Recurrent, unwanted intrusive thoughts (obsessions) • Repetitive behaviors or mental acts to try to quell intrusive thoughts	N/A	• MINI35 • Y-BOC35 • PRIME-MD	• Screen for MDD • Screen for SUD • Screen for other anxiety disorders • Careful observation for sequelae of compulsions	• Sertraline (Zoloft) 50-200 mg PO daily • Fluoxetine (Prozac) 10-20 mg PO daily • Fluvoxamine (Luvox) 50-100 mg PO daily; doses > 100 mg should be divided into bid dosing; up to 300 mg/day • Paroxetine+ (Paxil) 20-60 mg PO daily • Venlafaxine (Effexor) 75-225 mg PO daily	• Immediately for CBT if patient is motivated
PTSD	• Exposure to actual or threatened death, serious injury, or sexual violence • Intrusion symptoms: Flashbacks, dissociative reactions, and marked psychological/physical responses to triggers • Persistent avoidance of stimuli • Negative alterations in cognition or mood • Marked alterations in arousal/reactivity	≥ 1 month	PCL-5	• Screen for MDD • Screen for SUD • Screen for other anxiety disorders	• Venlafaxine (Effexor) 75-225 mg PO daily • Sertraline (Zoloft) 50-200 mg PO daily • Paroxetine+ (Paxil) 20-60 mg PO daily • Other antidepressants considered off-label use • Nightmares: Prazosin 3-15mg at night	• Immediately if patient amenable

Affective Disorders

Adjustment Disorders	Depressive sxs in response to a specific stressor Distress out of proportion to severity of the stressor WITH significant functional impact Does not persist longer than 6 months
Depression (Unipolar)	Screening Tools: PHQ-2; if 3 or higher -> PHQ-9 (mild 5-9, moderate 10-14, mod severe 15-19, severe = 20+) Geriatric Depression Scale (10+ = positive) Cornell Scale for Depression in Dementia Must explore Fam Hx, PMH and meds, Social Hx **S** – Sleep (decreased or increased) **I** – Interest in usual activities decreased (Anhedonia) **G** - Guilt **E** – Energy decreased **C** – Concentration issues **A** – Appetite (decreased or increased) **P** – Psychomotor agitation/slowing **S** – Suicidal ideation Dx: >= 5 symptoms from SIGECAPS + hopelessness for >= 2 weeks, causing significant distress NOT attributable to another medical condition (e.g. hypothyroid, anemia, vitamin deficiency, chronic infection, pain, sleep d/o, meds like steroids or BZD) or substance use Tx: **Mild = psychotherapy only usually ok** **Moderate – Severe = Psychotherapy + SSRI or SNRI** (first line; choose based upon comorbidities, prior personal or family response) – Rule out bipolar spectrum FIRST to avoid triggering mania! **Bupropion** CAN be first line depending on comorbidities (e.g. obesity) **Mirtazapine** **Adjunctive therapies** may include TCAs, ASDs, Atypical antipsychotics – refer these refractory cases to BH or Med-Psych Liaison Clinic!
Prolonged Grief Disorder	Yearning/longing and thoughts of the deceased (death at 12+ months ago) Dysphoria - Intermittent with triggers Guilt – centered around deeds done / not done wrt deceased Psychomotor changes – mild **Leading to significant distress/impairment**
Bipolar Spectrum	Screening: Mood Disorder Questionnaire (7+ predictive); Ask if periods of intense energy, decreased sleep, behavioral changes; Family history? Hx of Postpartum mood disorder? **D -** Distractibility **I –** Irritability / Impulsivity **G -** Grandiosity **F –** Flight of ideas **A –** Activity increased **S –** Sleep (decreased total sleep time without fatigue) **T –** Talkativeness

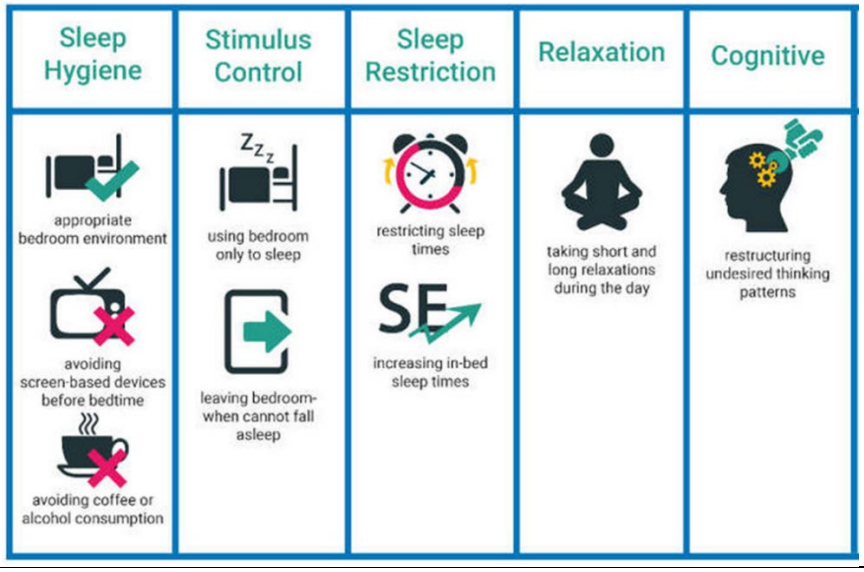
PCM Antidepressants

Group	Relative Benefits	Relative Risks
Escitalopram (Lexapro) Citalopram (Celexa)	Fewer Drug-Drug Interactions Great first line agents	QTc Prolongation (dose dependent), sexual side effects
Fluoxetine (Prozac)	Long half-life = Self-tapering, very well studied, most weight-neutral SSRI	Drug-Drug Interactions, sexual side effects, insomnia
Sertraline (Zoloft)	Best studied during pregnancy, drug of choice for post-MI, best for co-morbid anxiety and ADHD	GI side effects, sexual side effects, insomnia
Paroxetine (Paxil)	FDA approved for vasomotor symptoms of menopause (but would choose an SNRI instead…)	Sexual dysfunction, weight gain, **discontinuation syndrome – withdrawal!!** Risk of teratogenicity, Drug-Drug Interactions
Duloxetine (Cymbalta) Venlafaxine (Effexor) Desvenlafaxine (Pristiq)	Treatment of pain (OA and neuropathic)	GI distress, discontinuation syndrome, sexual dysfunction, Cymbalta & effexor can be very activating, SIADH
Bupropion (Wellbutrin)	Helpful for smoking cessation, weight neutral, minimal sexual a/e, can be helpful for ADHD First-line OR augmentation	Lowers seizure threshold, risk of overdose, contraindicated if history of anorexia, Drug Drug Interactions, Blood Pressure Effects, activating (especially immediate release, may worsen anxiety)
Mirtazapine (Remeron)	Dose dependent sedation and weight gain -- First-line OR augment!	Sedation and weight gain

Fatigue Mitigation in Residency

Avg number of needed sleep hours per night	7-9 hrs (very few can do well on fewer hrs!) – PRIORITIZE for global health improvements
Fatigue	Lack of sleep combined with physical & mental exertion *Cannot be overcome by motivation, training, or will-power* Performance WILL decline (but self-assessment will not change) Susceptibility is individual & varies
Insufficient sleep leads to:	Impaired mental effectiveness and alertness (felt after just 1 night of insufficient sleep!), insidious (you may not notice) Mood and interpersonal dysregulation, executive/hygiene lapses; ACCIDENTS, CASUALTIES, and PSR Events!!; chronic – behavioral health issues (PTSD), weight gain, T2DM, CV disease
Fighting Fatigue <u>ONLY effective counter-measures:</u>	**Adequate sleep** (including sleep banking before fatigue periods) **Napping** (10-20 min optimal to avoid entering deep sleep) **Caffeine** (200 mg just before a catnap, more effective if no sleep restriction or recent daily use/tolerance) **Education**
PSA: Behaviorally-Induced Insufficient Sleep Syndrome	Avoid staying up later to reclaim “Me Time” where possible, you’ll feel more refreshed and function better if you continue to get your needed avg hours!
Bottom Line	Don’t become or cause a sleep casualty! The WR Sleep Medicine Clinic is great at helping struggling residents with Insomnia, ISS, BIISS, or OSA (CPAP not necessary!)

Insomnia

<table>
<tr><td>Definition</td><td colspan="2">Must meet 6 criteria:
1. Sleep sxs: difficulty with sleep onset, maintenance, early awakening, etc
2. Leading to: fatigue, impaired attention/concentration, performance, mood disturbance, etc
3. Not explained just by inadequate sleep opportunity or environment
4. Not explained by another sleep, medical, or mental disorder or substance/medication use
5. & 6. Must occur 3x / week for at least 3 months</td></tr>
<tr><td>Evaluation Includes:</td><td colspan="2">- Predisposing factors: Biology, personality/temperament, adverse childhood events
- Environmental stressors: Illness, Divorce, Loss, Shift work, Job loss
- Compensatory behaviors: Earlier bed time, sleeping in, naps, caffeine, sleep aids, decr activity</td></tr>
<tr><td rowspan="2">Medication Therapy</td><td>Recommended for Sleep Onset
- BZRA: Zolpidem (Ambien), Eszopiclone (Lunesta), Zaleplon (Sonata)
- MLT-2: Ramelteon
- BZD with caution: Temazepam, Triazolam
- Dual Orexins: Suvorexant (Belsomra), e.g.
- Chronic insomnia requiring BZD, BZRA, antipsychotic x3 mo, waiver for combat AO</td><td>Suggested for Sleep Maintenance
- Doxepin
- BZRA: Ambien, Lunesta, Sonata
- BZD with caution: Temazepam, Triazolam
- Dual Orexins: Belsomra, e.g.</td></tr>
<tr><td colspan="2">NOT recommended for Sleep Onset or Maintenance
- Anticholinergics: Diphenhydramine (Benadryl), hydroxyzine (atarax/visteril)
- Melatonin, Tiagabine, L-tryptophan, Valerian
- Antidepressants: Trazodone
- Note: Magnesium products not discussed in guidelines
Then how do I use melatonin?
- Warn pt not FDA regulated (OTC products vary WIDELY in active ingredient dose)
- Dose at 0.5 – 1 mg taken 4 hrs before bedtime (not good for that 2300 inpatient call!)
- Can shift sleep/wake cycle – good or bad depending on pt's disease
- Reasonable to prescribe instead of encouraging OTC use</td></tr>
<tr><td>CBT-i</td><td colspan="2">CBT-I is individualized to the pt & takes WORK (must use 4 out of 7 nights) from the pt (decreased sleep up front can be unpleasant)
- Consider contraindications to sleep restriction (high risk job, mania, seizure, untreated OSA, acute illnesses)
Access to CBT-I is limited! (VA, IHW, WR Sleep Medicine, Self-Pay, Telemynd available)
The VA "Insomnia Coach" app is one excellent resource for at-home use
 
</td></tr>
</table>

Parasomnias & Hypersomnia

	Parasomnia		Hypersomnia
Define	**Abnormal sleep behavior or disturbances**		**Excessive sleepiness**
Type	Slow Wave (NREM)	REM Sleep	
Disorders	**Sleep eating; sleep sexual activities, confusional arousal; bruxism** **Somnambulism** - Sleep-walking, which may last several minutes, with little or no recollection upon awakening - First 3rd of sleep **Night terror / Sleep terrors** - Frantic movement, screaming followed by intense anxiety/arousal - First 3rd of sleep - Abrupt partial arousal from delta-wave sleep without later recollection - Often pediatric, many outgrow **Periodic Limb Movement Disorder (PLMD)** - Sleep-related myoclonus, jerking during sleep (usually NREM) - May occur due to hyperactive motor pathways, often from UMN lesion	**Sleep paralysis** **REM Sleep Behavior Disorder (RBD)** - Loss of usual atonia leading to "dream enactment" - Repeated loud, emotional vocalization & gross motor movement during sleep - Alert, oriented upon arousal **Nightmare disorder** - Repeated, dysphoric, vivid dreams often related to survival, well-being, security – with recollection! - Second half of sleep cycle - No movement or vocalization - Leads to mood disturbance, sleep resistance, and/or negative impact personal or family functioning	**Narcolepsy** Features of REM sleep intruding upon wakefulness and vice versa (possibly due to hypocretin/orexin deficiency) -Type 1: cataplexy present -Type 2: cataplexy absent Often presents as excessive daytime sleepiness (EDS); also a/w sleep paralysis, cataplexy (muscle atonia with intense emotion), and hypnagogic/ hypnopompic hallucinations **Idiopathic hypersomnia** **Kleine-Levin syndrome** - Relapsing/remitting episodes with cognitive disturbances (apathy, derealization, e.g.)
Risk Factors	Often younger demographic Poor quality sleep (deprivation, OSA, alcohol, e.g.), sedative-hypnotics, fever	RBD often older; EtOH w/d, anti-depressants, Neurogenerative disease (Alpha synucleinopathies) a/w RBD	Narcolepsy: Genetic - DQB1*0602 haplotype, prior infection (GAS, flu), certain structural or inflammatory dz
Eval	Eval of medical (cardiac, thyroid, diabetes), neurologic (epilepsy, Parkinsonian dz), or psych comorbidities (MDD, PTSD) – may include brain imaging Sleep history (determining usual patterns) Medications and recreational drug use (SSRI, TCA, SNRI, MAOi, BZD, BZRA, etc) Refer to Sleep, Neuropsych, etc! Sleep diary (patient/family track bedtime, sleep latency, events, etc) Gold standard = polysomnography (PSG, aka sleep study) – evaluates sleep architecture and underlying medical conditions (arrhythmia, epilepsy, OSA, etc)		Eval of all comorbidities Sleep history (rule out chronic deprivation) Med history (eval sedative impacts) PSG (must rule out OSA) Multiple sleep latency test (MSLT)
Mgmt	Manage any underlying medical or neuropsychiatric disease. Targeted therapy (relaxation skills, coping skills, mind-body therapy) may help Sleepwalking: bedroom & home safety, BZD PLMD: clonazepam, melatonin, or valproate Night terror: avoid meds unless treating a secondary cause (MDD, PTSD)	RBD: Manage any neurodegenerative process, educated pt and bedpartner on bedroom safety (de-clutter, no weapons, lower bed ht). Consider MLT receptor agonist Nightmare Disorder: treat underlying disorders, stress mgmt., imagery rehearsal therapy, prazosin for PTSD	Lifestyle modifications: sleep hygiene, scheduled naps, EtOH and sedative avoidance, work/activity restrictions while uncontrolled. Meds: modafinil 1st line, sodium oxybate (cataplexy); pitolisant (EDS+Cataplexy), solriamfetol (EDS); stimulants

Note: Exploding head syndrome, sleep enuresis, and sleep hallucinations are categorized as "other" parasomnias

Chronic Fatigue

Fatigue Definition

Difficulty in initiating or maintaining activity and/or difficulty with concentration, memory, or mood regulation

Acuity

Acute = 1 month or less	Subacute = 1-6 months	Chronic = 6+ months

Subacute – Chronic Differential (Exhausting but non-exhaustive)

Lifestyle	Medical	Psychological	Sleep
Extremes of activity Frequent travel Home stressors Food/housing insufficiency Work/life imbalance	Anemia Cancer Chronic Liver/Lung/Heart/Renal dz HIV/AIDS/EBV/HCV Fibro, PMR Medication Obesity Thyroid dz	Anxiety Depression PTSD Stress Substance use disorder	Poor sleep hygiene Restless legs OSA Sleep insufficiency Shift work sleep disorder

Evaluation

History & Physical

- Duration
- Preceding factors (lifestyle or medication changes, stressors, etc)
- SOCIAL HISTORY !! including sleep history
- Concomitant symptoms (pain, weakness, SOB, DOE)
- Medications (psychotropics, antihistamines, BZD, BB, Opiates)

Labs (may add clarity in 5% of cases)

- CBC w/diff, CMP, TSH, CK (if myalgia or weakness present), HCV Ab (if one-time screening not performed), HIV (if one-time or risk-related screening not performed)

Diagnostics - Pursuant to above ddx

Systemic Exertional Intolerance Disease (AKA chronic fatigue syndrome)

May be comorbid with central sensitization syndromes (fibromyalgia, IBS, interstitial cystitis, e.g.)

Diagnosis of exclusion, essentially

Diagnosis requires ALL three of the following:

1) Substantial reduction in ability to engage in pre-illness activities for >6 mos
    1) Is accompanied by fatigue of new or definite onset
    2) Not related to ongoing exertion
    3) Not substantially relieved by rest
2) Post-exertional malaise
3) Unrefreshing sleep

AND at least one of:

1. Cognitive impairment
2. Orthostatic intolerance

Management

- Treat the underlying cause!

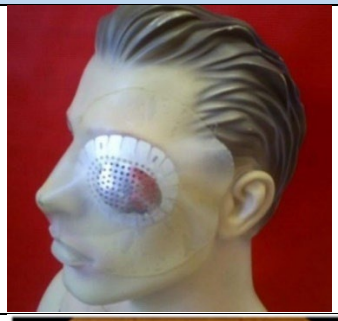
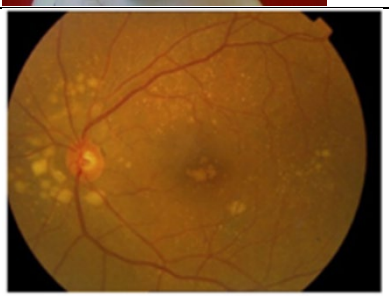
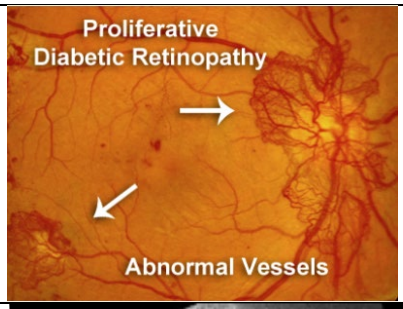
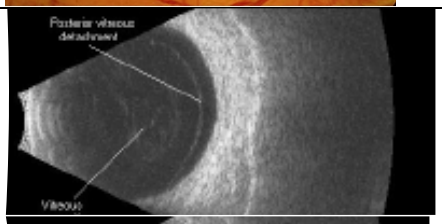
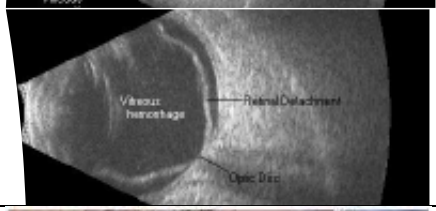
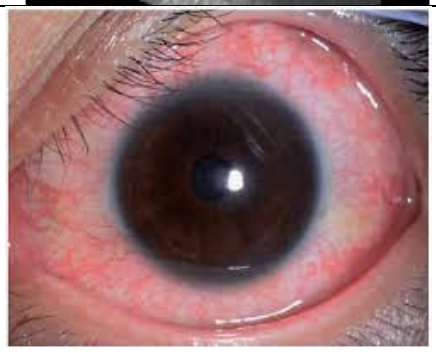
  For SEID:
    - Frequent office visits, reassessment & reassurance
    - Optimization of medical and psych comorbidities
    - No strong evidence for medication therapy
    - Pacing strategies for exertion
    - Auxiliary modalities: PT, OT, Biofeedback, Massage, Acupuncture, Yoga, Tai Chi

Outpatient Alcohol Use Disorder (AUD) Management

Screening	AUDIT-C – Score of 3+ is positive → full 10-question AUDIT screener Single Alcohol Screening Question – Positive if 1+ episode of 4 drinks in a day (women) or 5 in a day (men) 3-Item Screening Tool – 83% sensitivity for AUD within past year **CAGE is no longer USPSTF recommended TIP: Be non-judgmental over use, desire to change, harm reduction vs abstinence as goal, etc
Diagnosis	Pattern of ETOH use leading to ≥ 2 of the following within 12 months: • Alcohol consumed in ↑ amounts or for ↑ duration than intended • Difficulty controlling drinking, with persistent desire or unsuccessful efforts to ↓ or stop drinking • Considerable time spent obtaining, using, or recovering from alcohol and its effects • Craving for alcohol • Alcohol-related interference with one's major responsibilities or obligations • Continued consumption of alcohol despite social or interpersonal difficulties • ↓ Participation in important social, occupational, or recreational activities in order to drink • Consuming alcohol during physically hazardous situations • Continuing to drink despite known persistent or recurrent physical or psychological consequences • Tolerance • Withdrawal MILD = 2-3 symptoms MODERATE = 4-5 symptoms SEVERE = 6+ symptoms

Med	Line?	FDA?	MOA for AUD	Dosage	Contraindications	Side Effects
Naltrexone	1st NNT 12-20	Approved	Mu-opioid antagonist -> craving/drinking reduction	25-100 mg PO daily OR 380 mg IM q4wk	Active OUD, acute hepatitis, ALF (safe in cirrhosis with close monitoring)	Dysphoria, fatigue, HA, N/V/D
Acamprosate	1st NNT ~11	Approved	Unclear (?NMDA/GABA) -> craving/drinking reduction	666 mg (2 tab 333) PO TID	Allergy, GFR<30 (can dose reduce if GFR 30-60)	N/V/D, insomnia, anxiety/depression, dizziness, myalgia, rash
Disulfiram	2nd NNT 20	Approved	Aldehyde dehydrogenase inhibitor -> triggers unpleasant reaction if drinking on it → abstinence	Initial: 250-500 mg PO daily x1-2 wks Maintenance: 250 mg PO daily	Allergy, GFR<30, pregnancy, severe cardiac/CAD,	Diarrhea, insomnia, anxiety, depression, dizziness, bad taste, arrhythmia
Topiramate	2nd	Off-label	Unclear (dopamine reward pathway modulation) → abstinence, reduced cravings	Initial: 25 mg PO daily Maintenance: increase by 25-50 mg per week to max dose of 300 mg per day as tolerated	Metabolic acidosis Pregnancy Category D	GI sxs, dysgeusia, HA, dizziness, parestesia, cognitive impairment, hyponatremia
Gabapentin	2nd	Off-label	Unclear (?GABA modulation) -> reduced cravings and withdrawal sxs	Initial: 300 mg PO daily Maintenance: increase by 300 ng q1-2d to target 600 mg TID	None (Pregnancy cat C)	GI sxs, dizziness, drowsiness, insomnia, fatigue, headache
Others (Off Label)	Baclofen Prazosin Zofran Chantix GLP-1RA		Variable, limited or emerging evidence regarding benefits GLP-1RA promising			
Referral	Refer to Psych or Addiction Specialist if: • Drinking despite consequences, Unsuccessful attempt at ↓ drinking, poor response to primary care intervention, OR complicated comorbid psychiatric conditions					

Ophthalmology for the PCM

Disease	Patient	Exam	Management	Photo
Ocular Trauma	s/p any kind of trauma (MVA, assault, battle injury) to the head/face	Look for open globe, evidence of fracture, slit lamp	CALL OPHTHO Irrigate chemical injury Lateral canthotomy for retrobulbar hematoma Eye shield CT vs US Abx (FQ) pain/nausea ctrl Tetanus shot Fox eye shield →	
Macular Degeneration	Older, white, female, tobacco use, light iris color Dry (80-90%) vs Wet (10-20%)	Dry – drusen, pigment change Wet – choroidal neo-vascularization	Dry – AREDS2 supp. (AREDS supp has lung ca risk in smokers) Wet – Anti-VEGF injections Urgent referral for acute vision loss/distortion	
Diabetic Retinopathy	Long standing patients with diabetes, especially worse glycemic ctrl	Proliferative – new vascularity May/may not have macular edema or cataracts	Yearly Screen! T1DM Begin 5 years s/p diagnosis T2DM Screen at diagnosis	
Posterior Vitreous Detachment vs. Retinal Detachment	PVD – age >50, myopia, prior PVD RD – age >50, prior RD, FH of RD, myopia, prior eye sx or injury	PVD – linear separation including optic disc RD – fluid develops under retina, lifting up from choroid, spares optic nerve	<u>Routine referral</u> for sxs >1 week <u>Urgent referral</u> for sxs <1 week OR curtain/veil, constant blurry vision, hx of driving glass, ocular surg, trauma/DM	 
Uveitis	Non-infectious – pts with sarcoid, Bechets, lymphoma, HLA-B7, RA or reactive arthritis Infectious – Toxo, HSV/VZV/CMV, syphilis, TB, candida	Inflamed uvea (middle layer of eye) Anterior, Intermediate, posterior, or Pan Anterior – non-limbic sparing redness, pain	Optometry screening for HLA-B27, plaquenil, or ethambutol pt Routine uveitis f/u – Ophtho referral annually Uveitis on immune modulators – Uveitis clinic referral Acute new/flare – **urgent** ophtho referral	

Exertional Heat Illnesses

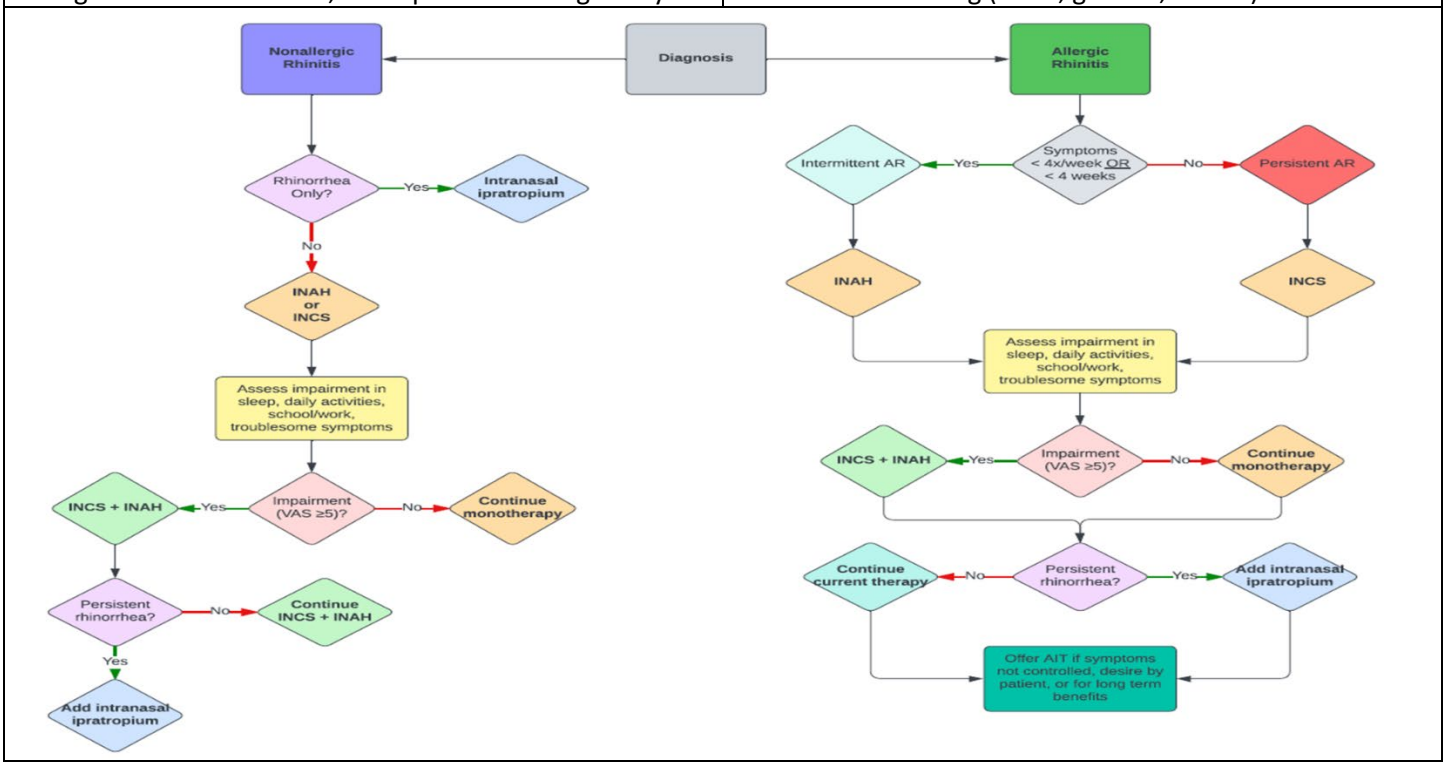
	Heat Exhaustion	Heat Injury	Heat Stroke
Pathophys	Inability to maintain CO due to strenuous activity and heat	Thermoregulatory failure leading to gut endotoxin leakage and inflammatory response	Thermoregulatory failure leading to gut endotoxin leakage and inflammatory response
Symptoms	Profuse sweating, pallor, "prickling", thirst, dizziness HA, cramps, N/V/D, myalgia	As with exhaustion **Absent** CNS dysfunction	As with exhaustion CNS dysfunction
Vitals	+/- Tachycardia, hypotension Core body temp often 101-104F	T may be >104F	Tachycardia, Hypotension T > 104F
Labs	Hypo/ernatremia	Hypo/ernatremia Elevated CK End-organ dysfunction	Hypo/ernatremia Elevated CK End-organ dysfunction
Treatment	Cease activity, shade or AC Supinate, elevate legs Remove excess clothes Rapidly cool to 101F (rectal) ED if not improving	Rapidly cool to 102.2F (rectal) Ice bath or evaporative cool Transfer to ED CBC, CMP, CK, UA, Coags Manage complications	Rapidly cool to 102.2F (rectal) Ice bath or evaporative cool Transfer to ED CBC, CMP, CK, UA, Coags Manage complications

Approach to Back Pain

Disease	Pathophysiology	Symptoms	Exam/Imaging	Treatment
Cervical Radiculopathy	Nerve root compression - Disc herniation - Bone spur (osteophyte) - Rare: tumor, abscess, e.g.	Pain/numbness/ weakness, Unilateral dermatomal distribution	Abduct, Spurling test XR to r/o fracture MRI C-spine	70% resolve without Surgery Ladder: NSAIDs, Gabapentin +/- PT > ESI > Surgery (Fusion, Foraminotomy, Artificial Disk)
Cervical Myelopathy	Compression of the spinal cord, often from spondylosis (arthritis) Don't miss: tumor, trauma, infxn!	Painless! Dexterity Loss Balance loss Bowel/bladder (late) UMN sxs, hyperreflexia	Hoffman sign Tandem Gait Clonus/babinski XR & MRI	No spontaneous improvement without surgery, do NOT send to PT (neck manipulation bad!) 70-90% have some improvement WITH surgery
Lumbar Disc Herniation	Nucleus pulposus herniates (bulge vs extrusion) through annulus, compresses spinal nerve Don't miss: cauda equina!	Pain, numbness, weakness, often unilateral	Straight Leg raise Neuro exam BLE EMG/NCS not necessary XR to r/o frx MRI L-spine	40% better in 1 mo, 70% better in 3 mo <u>Isolated back symptoms:</u> PT or PM&R <u>Radiating leg symptoms:</u> 2-6 wks: Medical mgmt 6-12wk: MRI, Sx referral, ESI Weakness: Will see within 1-2 wks, please have MRI Severe BLE/urinary retention – ED <u>Treatment ladder as above</u>
Lumbar Spinal Stenosis	Arthritic changes Compression of thecal sac	Neurogenic claudication sxs (pain relieve by position e.g. leaning over shopping cart) Proximal pain/cramps	XR and MRI helpful EMG/NCS not so much	<u>Isolated back symptoms:</u> PM&R or Pain clinic <u>Claudicatory symptoms:</u> Typically slowly progressive Ortho will see these pts anytime
Spondylo-listhesis	"Slip" of one vertebral body over another causing pain from instability Antero- top body slides fwd Retro- top body slide back	Low back pain	Get XR first, MRI helpful	Antero = Surgical Retro = non-surgical PM&R
Sacroiliac Joint Dysfunction	Abnormal motion (too much or too little) of the SI joint	Low back pain localizing below PSIS, exacerbated by load bearing/impact	FABER, Gaenslen's, SI Distraction, Fortin's Finger test Difficult to dx with rads. MRI to rule out other dx	SIJ Injection = Gold standard dx NSAIDs, PT, Pelvic Belt, CSI, RFA Surgery not that effective, exhaust everything else first

Chronic Rhinitis

Non-allergic	Allergic
Often adult onset, vasomotor, temperature, spice, non-allergen irritant mediated, often persists throughout year	Often pediatric onset, aeroallergen triggers with seasonal timing (trees, grasses, weeds)



Rhinosinusitis in More Detail

Definitions	Rhinitis: inflammation of nasal passages causing sneezing, rhinorrhea, congestion, itching Rhinosinusitis (aka Sinusitis): inflammation of nasal passages & sinuses causing facial pressure/pain, HA, anosmia

Acute (<4 weeks)	**Chronic (>12 weeks)**
Largely viral or bacterial; mucosal edema, turbinate hypertrophy; rhinorrhea or discharge Antibiotics if: 1. >10 days of symptoms, 2. Severe symptoms (T>102.2F, purulent discharge or facial pain ≥3 days), or 3. Onset after improving viral illness (double sickening) - Augmentin or amoxicillin (doxy or levaquin if PCN allergy)	Has 2 of: nasal drainage (anterior or posterior), nasal blockage or congestion, facial pain/pressure/fullness, reduction or loss of smell Evidence of mucosal inflammation (CT or endoscope) Classified by presence or absence of nasal polyposis (20-33% have polyps) Tx: Intranasal saline spray or irrigation (use before other sprays) Intranasal corticosteroid (2 mo); can use steroids for polyp blockage - Refer to allergy/ENT if no benefit from initial therapy

Allergy Mythology

Myth	Truth	Impact	Recommendations
Severe egg allergies preclude routine flu vax	While there is some egg protein in most flu vaccines, it is still safe for these pts to get the flu vax – no incr risk of anaphylaxis	Preventable influenza hospitalizations and deaths – risk likely higher among those egg allergic pts with comorbid asthma	All eligible patients should get annual flu shot – no need to screen for egg allergy
Shellfish allergy == iodinated contrast allergy	Shellfish allergen is not present in contrast media. Iodine allergy is not real. Betadine allergy IS real but due to povidone component.	Delayed diagnosis and treatment of conditions that require use of IV contrast media (e.g. CTPE, stroke, LHC in MI)	It's ok to use iodinated contrast media in pts with shellfish allergy if no allergy to contrast. Don't say "iodine allergy" for contrast media all
You can't use any beta-lactam antibiotics if pt has a documented PCN allergy	Virtually every patient reporting a history of or who is skin test positive to penicillins may receive a cephalosporin antibiotic as a replacement with the exception of those showing R1 side-chain similarity – even this may be very conservative!	Surgical site infection C diff infection MICU admission Resistant Organism Costs associated with certain antibiotics	For the FEW pts with anaphylaxis to PCN, a non-cross reactive cephalosporin can be given without prior testing – check the R groups! Remove system-generated warnings in the EMR!
"Anaphylaxis treatment consists of Benadryl and steroids"	It's epinephrine. First-line. Up front. Antihistamines can help for cutaneous symptoms & are second-line agents. Steroids lack evidence for clinical benefit.	Far fewer than expected patients (~50%) receive epinephrine first line or at all – especially in the field. This is suboptimal care.	Get comfortable with EpiPen use and teaching for your pts. Institute an anaphylaxis orderset with IM epi in your ED!
"Toxic mold in my house is giving me vague constitutional symptoms!"	Causal association remains weak and unproven for inhaled mycotoxins in homes. AIT can target proven mold allergies but data is not robust. Hypersensitivity pneumonitis and ABPA are valid.	Unnecessary consumer and healthcare spending on mold removal and investigation as causal agent of symptoms	We do not have enough information to say that common households molds cause these symptoms.

Abnormal Uterine Bleeding (AUB)

Definition	Excessive menstrual bleeding in terms of flow, frequency, or duration	
Etiology	**STRUCTURAL** **P –** polyp **A –** adenomyosis **L –** leiomyomata (fibroids) **M –** malignancy/hyperplasia	**NON-STRUCTURAL** **C –** coagulopathy (vWF) **O –** ovarian dysfunction **E -** endometrium **I –** iatrogenic (IUD) **N –** non-specified (scars)
Timing	**OVULATORY** <u>Occurs regularly but flow is excessive</u> - Think Thyroid, bleeding disorders, structural abnormalities	**NON-OVULATORY** <u>Irregular flow and cycle duration</u> Common just after menarche or perimenopause - Think PCOS, Thyroid, Prolactinemia, CKD, Meds (anti-psychotics, chemo, SERMs)
Evaluation	**Good medical, gynecologic, menstrual history & thorough physical (including pelvic/bimanual)!** <u>Labs</u>: Urinary pregnancy test and CBC for ALL; consider PT/PTT/INR, TSH, Iron group, Prolactin/FSH/LH <u>Imaging</u>: TVUS (best look at structural abnormalities, uterine stripe & adnexa) <u>Procedures</u>: Endometrial biopsy (first-line AUB >45 yo OR <45 but with endometrial ca risk factors = obesity, hx of unopposed estrogen, genetic syndromes)	
Treatment	**Address underlying cause (surgical removal of structural issue, PCOS mgmt, etc)** <u>For those desiring contraception</u>: OCPs or levonorgestrel IUD (NOT copper) <u>For those wishing to maintain fertility</u>: medroxyprogesterone acetate NSAIDs or TXA for bleeding control GNRH or IV estrogens for acute/severe bleeding -> ablation, embolization, hysterectomy if refractory	

Premenstrual dysphoric disorder (PMDD)

Diagnosis	1. At least one primary symptom* plus four additional symptoms: - Mood swings* - Irritability or anger* - Hopelessness or depressed mood* - Anxiety* - Appetite change - Anhedonia - Fatigue - Poor concentration - Feelings of loss of control - Sleep disturbance - Physical symptoms: breast pain, bloating, myalgias, weight gain 2. Symptoms typically develop the week before cycles, remit within a week after, and occur with most cycles during a given year

Treatment	• <u>Mild</u>
	  • Exercise, relaxation, chasteberry
	• <u>Moderate – Severe</u>
	  • SSRI (Prozac, Zoloft) - Continuous, Sx-onset, or Luteal dosing
	  • OCP – good for comorbid AUB, contraception, etc
	Some may benefit from CBT; GNRH agonist, acupuncture also options if refractory

Thinking Systems

System 1 – Thinking Fast	System 2 – Thinking Slow
Heuristics AKA mental shortcuts AKA ?intuition – helpful for quick, often correct decisions based on memorized patterns, but error-prone and bias-laden! **Availability Risk –** a prior experience leads you to over-screen or over-test for a specific condition **Representative Risk –** a stereotype or assumption of a patient leads you to under-order or under-treat for them	**Contemplative critical reasoning –** will help you arrive at the correct decision, check your biases, and analyze all available data, but you will sacrifice a lot of time. **Keeps System 1 in check!**

Illness Scripts – a chance to use lots of System 2 to create System 1 patterns

Associated dangers:
Premature closure or "anchoring"
Blind Obedience – unquestioned deference to authority
Availability – the first thing that comes to mind MUST be right! /s
Confirmation Bias – only looking for evidence to support your first thought & ignoring counterfactuals
Left Digit Bias – difference in care with increase in tens-place age (80 vs 79, e.g.)
Win-Stay/Lose-Shift Heuristic/Bias – deviating from best practices after getting burned or not considering risk in absence of safety issue

Risk increases when: you're tired, overworked, or under a time crunch!
You're upset or have negative counter-transference!
You have unchecked implicit or explicit biases!
You're overloaded by complex patient data / history!
You don't have all the right info!
Systems (environment, EMR, team culture, poor/no handoff) & personal issues both contribute…

Presentation Skills

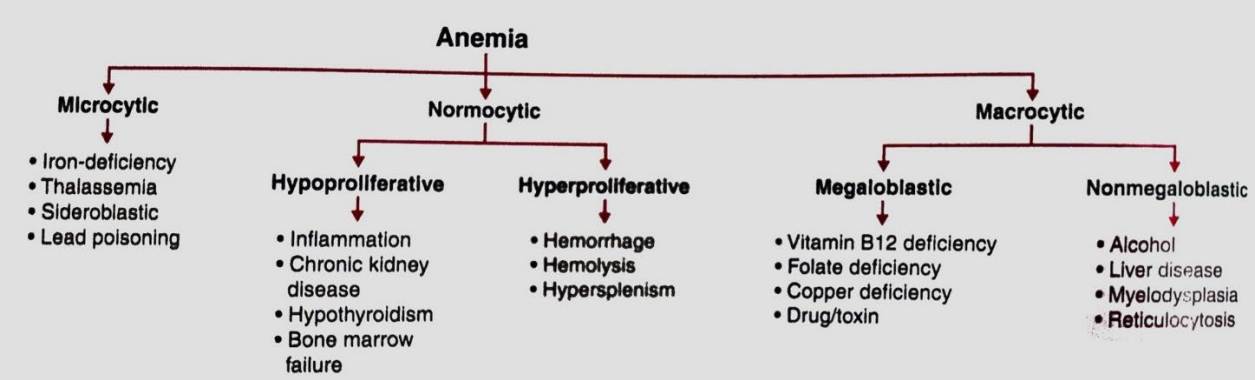
The SOFTEN Approach
S – Smile (warmth, smiling, appearance influence your audience) **O** – Open stance (gesticulate normally, keeping hands uncrossed/not interlaced) **F** – Forward lean (NOT grabbing the podium, standing next to it, leaning into your crowd) **T** – Tone (vary the inflection and volume of your voice) **E** – Eye contact (scan the audience, hold gaze briefly, use names! keep eyes off visual aide) **N** – Nodding (both with your head and with providing affirmation of audience responses) *Keep ppts slides to 6 lines of text, no more than 6 words per slide *Have some audience engaging technique, break, or pause for reflection at least every 15 minutes (e.g. Pair Share)

Managing Talent

Managing Others	<u>3R's of Management</u> 1. Defined **R**oles 2. Clear **R**equirements 3. Established **R**anks (authority) <u>Good Managers</u>: have integrity, respect, commitment, approachability, good comms/listening skills <u>Bad Managers</u>: dishonest, micro-manage, disrespect, emotionally labile, arrogant, unknowledgable, apathetic, aloof Delegating Well - Identify the task > Identify the right person for it (their skills/interests and/or career needs) - Define the task (timeline, expectations, desired end-state) and share any resources - Check-in but don't micromanage! <u>Evaluating your effectiveness as a manager</u> - What's my Style? (Blake Mouton Managerial Quiz) ○ Country Club, Impoverished, Authoritarian, or Team Managers (Team style is more effective) - Does my team know what I need from them? - How often do I communicate with them? - How closely do you follow up on their performance? - Do I give them fair, frequent feedback based on preset expectations?
Managing Yourself!	Set boundaries - Budget your time – manage a good calendar - Carve out protected time for family, hobbies, life maintenance Know when to say no (aka the Disciplined Pursuit of Less) - Success brings more opportunities, but more opportunities dilute our vision and bandwidth, leading to subpar output - Use criteria to evaluate opportunities (Does it match your passion? Your talent? Your or the world's needs?) - Saying no too much == negative perception from others, fewer opportunities offered

Hematology and Oncology

Anemia Framework

Definitions	ASH Guidelines and DODI 6130.03 ◦ Females: Hb < 12 ◦ Males: Hb < 13.5 2024 WHO Guidelines ◦ Females: Hb < 12 ◦ Males: Hb < 13
Differential	

Transfusion Thresholds

Packed Red Blood Cells (pRBC) – Recheck at 15-60 min post transfusion	
<7	Nearly all-comers (reminder: Jehovah's Witnesses may decline!)
<8	Acute Coronary Syndrome (ACS) **8-10 may be reasonable
<10	Active bleeding, trauma
Sickle Cell**	Simple transfusion OR exchange transfusion during pain crises / Acute Chest / Stroke Some will offer when <2 g/dL below baseline (Goal: Hgb 9 or Hb S <30%)
Types of pRBC – Recheck at 15-60 min post-transfusion	
Irradiated	Bone Marrow Transplant, Acquired/congenital immunodeficiency, blood donated from 1st/2nd degree relatives
Leukoreduced	Chronic transfusion, CMV negative at-risk patients (AIDS, transplant), Potential transplant recipients, prior FNHTR
Washed	IgA deficiency, complement-dependent AIHA, continued allergic reactions to pRBC despite anti-histamines
Platelets	
<10K	Always! Risk of spontaneous CNS bleed (less so in ITP)
<20K	Febrile
<50K	Peri-procedure OR active bleeding
<100K	Neurosurgery & Lumbar Puncture (though some will do LP at 50K+)

Transfusion Reactions

Febrile Non-Hemolytic Transfusion Reaction (FNHTR)	Brief, self-limited fever without evidence of hemolysis Dx of exclusion – need BCx, DAT, re-check compatibility Tx: Leukoreduced transfusions in future
Acute Febrile Hemolytic Transfusion Reaction	Patient given wrong ABO/Rh/other antigen-matched blood! Time: Minutes to Hours Sxs: Fevers/chills, rigors, dark urine, AKI, "impending doom" Dx: positive DAT, low haptoglobin, high LDH, bilirubin elevation Tx: IVF, may need pressors, Renal Replacement, coagulopathy mgmt
Delayed Febrile Hemolytic Transfusion Reaction	As above Time: 2d – 1 month Sxs: fatigue, pallor, jaundice Dx: positive Ab screen, DAT, high LDH, indirect Bili, low hapto Tx: Removal of donor from pool, cautious transfusion, may need steroids, IVIG, Rituxan, Epo
Anaphylaxis	Sx: airway closure, angioedema, hypotension/shock Risk: IgA deficiency, allergy to a component of the blood product Tx: Epinephrine, antihistamines, +/- pressors
Transfusion Associated Circulatory Overload (TACO)	Time: 6-12 hrs post transfusion Hypoxia & Pulmonary infiltrates & sxs of overload (JVD, elevated BNP e.g.) Risk: Age>60, CKD, CHF, >1 unit pRBC Tx: Diuresis!
Transfusion Related Acute Lung Injury	Time: 6-12 hrs post transfusion Hypoxia & Pulmonary infiltrates WITHOUT sxs of overload Risk: Critical illness Tx: Supportive care

Thrombocytopenia

Mild	**Moderate**	**Severe**
100K-149K	50K – 99K	0 – 49K
Increased Destruction	**Decreased Production**	**Sequestration or Dilution**
Immune – ITP, SLE/APLS, RA Drugs – HIT, antibiotics MAHA – DIC, TTP, HUS, preeclampsia/HELLP Shearing – CVVH, bypass, IABP, vasculitis	Infection – HIV, HCV, VZV, CMV, EBV, Tickborne (Ehrlichiosis), Parvo, Sepsis Nutrition – EtOH, B12 or Folate deficiency Drugs – Chemotherapy Neoplasm – MDS, liquid tumors Other – Cirrhosis, Aplastic anemia	Hypersplenism Sepsis Massive transfusion/fluid resuscitation Hypothermia Gestational Platelet clumping artifact
Lab Eval		
Can rule out clumping by sending platelet count in a citrate tube ***DO NOT SHOTGUN ALL OF THESE AT ONCE*** CBC with Diff BMP + LFT (need the bili differential) LDH, haptoglobin, DAT, Reticulocyte count PT/PTT/INR Fibrinogen, D-dimer (rule out DIC) HIT Ab, Serotonin Release Assay (SRA) ANA, Lupus anti-coag, anti-cardiolipin, Anti-B2GP1 Pregnancy test BMBx if suspicion for infiltrative/malignancy		

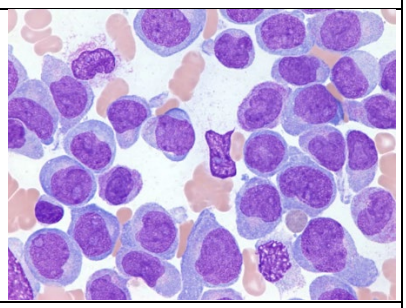
Immune Thrombocytopenic Purpura (ITP)

Cause	Autoantibodies to platelet surface membranes leading to platelet destruction
Etiology	Primary (idiopathic) Secondary – autoimmune (SLE, APS), immunodeficiency (CVID, IgA), malignant (CLL), infectious (HIV, Hep C, H pylori, CMV), meds (MMR vax, Gold, PD-1 inhibitors, etc), & many more!
Findings	Petechiae, purpura, ecchymoses, fatigue, frequent epistaxis/bleeding, +/- splenomegaly Generally risk of severe bleeding is low; but risk increases with prior bleed, Plt<10K, and age >60 yrs old
Diagnosis	Diagnosis of Exclusion: defined as isolated thrombocytopenia with Plt < 100K without other cytopenia or cause Eval should include peripheral smear, HIV, HCV, LFT for all; further work-up per secondary etiology suspected Platelet autoantibody testing is NOT recommended
Treatment	Inpatient: New dx AND plt<20K, regardless of sx/bleeding Outpatient: Plt>20K without bleeding Platelet transfusion: <10K for all; <20K with fever, <50K peri-procedure or active bleed Critical bleed: intracranial/spinal/ocular/RP/pericardial/IM Severe: Hgb falls 2+ g/dL or requires 2+ units First-Line (can be used together, especially in critical bleeding) - Glucocorticoids – pts who are asymptomatic but plt <20-30K OR have minor bleeding with Plt <50K - IVIG – Severe thrombocytopenia and life-threatening bleeding Second-line - Splenectomy, rituximab, thrombopoietin receptor agonists - for refractory or relapse

Thrombotic Thrombocytopenic Purpura (TTP)

TTP
One type of thrombotic microangiopathy = microvascular injury leads to thrombosis in capillaries and arterioles **Epi:** 2-6 per million, more common in younger females **Etiology:** hereditary/congenital deficiency of ADAMTS13 (<5% cases) or autoantibodies against ADAMTS13 (>95% cases) causing large VWF clumps and platelet aggregation **Symptoms:** Fever (20-30%), Anemia, Thrombocytopenia, AKI (10%), Neurologic dysfunction (70-80% - e.g. AMS, HA, TIA, stroke, Sz), Purpura, GI sxs **Dx**: ADAMTS13 & high clinical suspicion (the level takes time to result but you cannot delay treatment) - Use PLASMIC score for risk stratification! Score of 6+ is 72% likely to have severe deficiency **Tx:** If high suspicion, start Therapeutic plasma exchange and corticosteroids ASAP after ADAMTS13 level is sent - You will need Nephro & Heme/Onc consults! In the MICU… - Consider Rituximab infusion if ADAMTS13 level <10 - Consider Caplacizumab for severe cases

Acute Myeloid Leukemia

<table>
<tr><td>Definition</td><td colspan="2">Malignant clonal proliferation of myeloid cells, invading bone marrow</td></tr>
<tr><td>Signs</td><td colspan="2">Pancytopenia
- Anemia: fatigue, dyspnea, weakness
- Neutropenia: fever, infection (low ANC despite high WBC)
- Thrombocytopenia: petechiae, bruising, bleeding (epistaxis, gingival, etc)
Hyperuricemia or other signs of TLS
Lymphadenopathy
Hepatosplenomegaly</td><td>  </td></tr>
<tr><td>Evaluation</td><td colspan="2">Peripheral smear with myeloblasts, but may be absent (Auer Rods == APML / APL, often younger pts)
BMBx with >20% myeloblasts, Flow cytometry, karyotype; HLA typing for HSCT candidates
Molecular genetic testing allows for risk stratification and targeted therapies</td></tr>
<tr><td>Complications

DIC, ICH, etc also worth knowing.</td><td>Leukostasis: accumulation of leukemic cells (usually WBC 50K+) in microvasculature

Sxs: HA, vision changes, seizure, hypoxia, encephalopathy, stroke, CXR infiltrates</td><td>Tumor Lysis Syndrome: metabolic abnormalities from rapid cell death (spontaneous or 2/2 chemo) -> intracellular ions flood extracellular compartment -> hyperK, hyperPhos, hypoCa, hyperuricemia (Pikachuuuu)

Sxs: N/V/D, seizure, arrhythma, cramps, tetany</td></tr>
<tr><td>Treatment</td><td colspan="2">Favorable/Intermediate-Risk Groups: 7+3 == cytarabine x 7d, daunorubicin x3 days (additional agents per molecular targets)
High-Risk Groups: Hematopoietic stem cell transplantation
Curable in 35-40% of healthy pts <60 yo; Cure rate ~5-15% for pts 60+ yo with comorbidities

For Acute Promyelocytic Anemia (APL/APML): all-trans retinoic acid (ATRA) – immediately!
To avoid significant bleeding risk from fibrinolysis & DIC</td></tr>
</table>

Oncologic Emergencies

Emergency	Associated Cancers/ Txs	Key Risk Factors	Expected Timeline	Management (Key Points)
Febrile Neutropenia	Hematologic malignancies, chemotherapy	ANC <500, recent chemo, hx of MRSA, lines, PNA, unstable, SSTI	Within days of neutropenia onset after chemo (usually days 7-14)	Prompt empiric antibiotics (cefepime±vanc), infectious work-up per sxs, consider ID consult
Hypercalcemia of malignancy	PTHrP-producing tumors of the Lung; Osteolytic lesions; Prostate, Breast, Vit D activation, Lymphoma	Small cell lung Ca, Bone mets, dehydration, high tumor burden	Gradual or acute onset; often in late-stage disease	IV NS, bisphosphonates, calcitonin, possible denosumab, treat malignancy (surgery/chemo)
Tumor Lysis Syndrome (TLS)	High-grade lymphoma, AML, usually after cytotoxic therapy (rarely in solid tumors)	High LDH, bulky tumors, high cell turnover	Usually with 12-72 hours of chemo OR spontaneously in high-risk tumors	PiKaChU mnemonic! Aggressive hydration and electrolyte management; Rasburicase if urate>7.5 (can't use with G6PDD), ICU, HD/CRRT if severe
Spinal Cord Compression	Any cancer with vertebral mets (Breast, Prostate, Lung, Myeloma)	Bone mets to spin, rapidly growing tumors	Subacute to acute; days to weeks from met progression	Dexamethasone, MRI spine, urgent radiation or surgical decompression, NSGY + RadOnc consult
Acute Promyelocytic Leukemia (APL)	AML-M3 subtype, t(15:17), PML:RARα	Low: ≤10 WBC, >40 Plt Int: ≤10 WBC, ≤40 Plt High Risk: >10 WBC	High risk of early mortality 2/2 bleed or DIC, infection, differentiation syndrome	ATRA immediately!! Molecular/cytogenetics ASAP, supportive care, manage DIC
Malignant Bowel Obstruction	GI and GYN cancers with peritoneal mets	Peritoneal carcinomatosis, obstruction risk, prior surgeries (adhesions)	Subacute; varies by tumor burden and progression	Bowel rest, NGT decompression, steroids (for ovarian), surgical or palliative approach
Immune-related Adverse Events (irAEs)	Checkpoint inhibitors (e.g. PD-1/PD-L1, CTLA-4 blockers), can affect any system but most often colon, liver, lung	Autoimmune disease history, multi-agent immunotherapy	Typically weeks after starting immunotherapy	Grade-based management: steroids for Grade 2+, hold immunotherapy, eval per affected body system
Cytokine Release Syndrome (CRS) & Immune Effector Cell-Associated Neurotoxicity Syndrome (ICANS)	CAR-T therapy (esp CD19 targeted), biospecific T-cell engagers (BiTE)	High tumor burden, early post-infusion (day 1-3)	Hours to few days after CAR-T	IL-6 blockade (tocilizumab), steroids if severe, ICU admission if unstable or in respiratory failure
Leukostasis	AML, ALL, CML blast crisis (WBC >100k) – may present with stroke or other vasoocclusive sxs (HA, SOB, chest pain, AKI)	High blast count, AML M4/M5 subtype, poor perfusion (baseline vascular disease)	Acute; can present rapidly with high WBC	Hydroxyurea, leukapheresis if symptomatic, avoid RBC transfusion, start chemo
DIC	APL, metastatic cancers – low fibrinogen!	APL, trauma, infection, severe sepsis	Acute; usually during critical illness or initiation of APL therapy	Treat underlying cause! Supportive transfusion (plt, FFP, cryo), avoid antifibrinolytics
SIADH	Small cell lung cancer, head/neck cancers, ectopic ADH secretion	Ectopic ADH, CNS disease, cytotoxic chemo (vincristine, Cyclophosphamide)	Subacute; typically within days of ectopic ADH or drug effect	Fluid restriction; hypertonic saline if sxs, tolvaptan if refractory; treat malignancy
Superior Vena Cava (SVC) Syndrome	Lung cancer, lymphoma, tumor mets to mediastinum	Central venous compression, tumor bulk, thrombosis	Subacute; can present over days to weeks	CT venography, prioritize biopsy, Tx chemo/RT, stent with IR, consider steroids

Pink Ribbon Month: Breast Cancer Basics

Epi	Most common & second deadliest cancer in women (1 in 8 women affected) Sporadic (90%), genetic mutation like BRCA 1/2 (10%) Invasive ductal carcinoma most common; hormone positive (ER) majority of cases
Risk Factors	Older age, prior personal cancer, FDR with breast ca, early menarche, late menopause, OCPs, HRT (longer estrogen exposure), chest wall irradiation <30 years old, obesity, excess EtOH
Exam	Inspect skin for dimpling, color changes, retraction; evaluate nipple discharge, overall breast symmetry, palpate for lumps/nodules and axillary/supraclavicular LAD
Diagnosis	If a mass is identified, keep breast cancer top of mind! Age <30 – obtain US; cysts are aspirated or biopsied, solid masses are biopsied or excised Age >=30 – obtain diagnostic mammo, followed by US (BiRads 1-3) or biopsy/excision (BiRads 4-5)
Treatment	Complex, multiD discussion between medical and surgical oncology, patient, GC, etc depending on stage, pt desires, etc. Options include: Surgery Hormone therapy (usual duration 5 years) - For hormone-positive cancers only - Pre-menopause (tamoxifen); post-menopause (aromatase inhibitor OR tamoxifen then AI) Chemotherapy and/or Immunotherapy
Screening	Annual mammogram age 45-54, then every 1-2 yrs 55-74 (ACS – average and moderate risk) Annual breast MRI and mammo, staggered 6 months apart (high risk = >20% lifetime risk) The GAIL model (Breast Cancer Risk Assessment Tool) – estimate 5 year and ~lifetime risk of developing breast cancer using personal demographic and reproductive info – NOT for BRCA ½ - Can use to guide discussions about chemoprevention, ppx mastectomy, etc… or to refer to Breast Care Center for that
At WR	Breast Care Center on 3rd floor of America Bldg is a fantastic resource; they will handle more advanced diagnostics, multiD care, prophylactic mastectomy discussions, etc AFTER appropriate initial work-up is done in PCMH Pink Ticket – available for overdue patients age 50-75 (last mammo 25+ mo ago) – grab at front desk and take to 3rd floor for same-day mammo

Paraneoplastic Syndromes associated with Lung Cancers

Small Cell	Adenocarcinoma	Squamous Cell	Large Cell
Lamber-Eaton myasthenic syndrome (LEMS) SIADH Ectopic Cushing syndrome SVC Syndrome	Hypertrophic Pulmonary Osteoarthropathy: pain in hands or legs (XR with periosteal elevation) Marantic endocarditis: Non-bacterial thrombotic endocarditis or NBTE	PTHrP: Hypercalcemia Pancoast's Tumor: 1st and 2nd thoracic nerve; shoulder pain + horner syndrome Horner's Syndrome: Ptosis, miosis, anhidrosis	SVC Syndrome Gynecomastia

Infectious Disease

Community-Acquired Pneumonia (CAP)

Dx:

New lung infiltrate plus clinical evidence of infection (fever, purulent sputum, leukocytosis, or hypoxia) not acquired in the hospital setting

Risk strat:

Pneumonia Severity Index (PSI) – more factors, harder to use readily

CURB-65 – fewer factors, easier to use readily

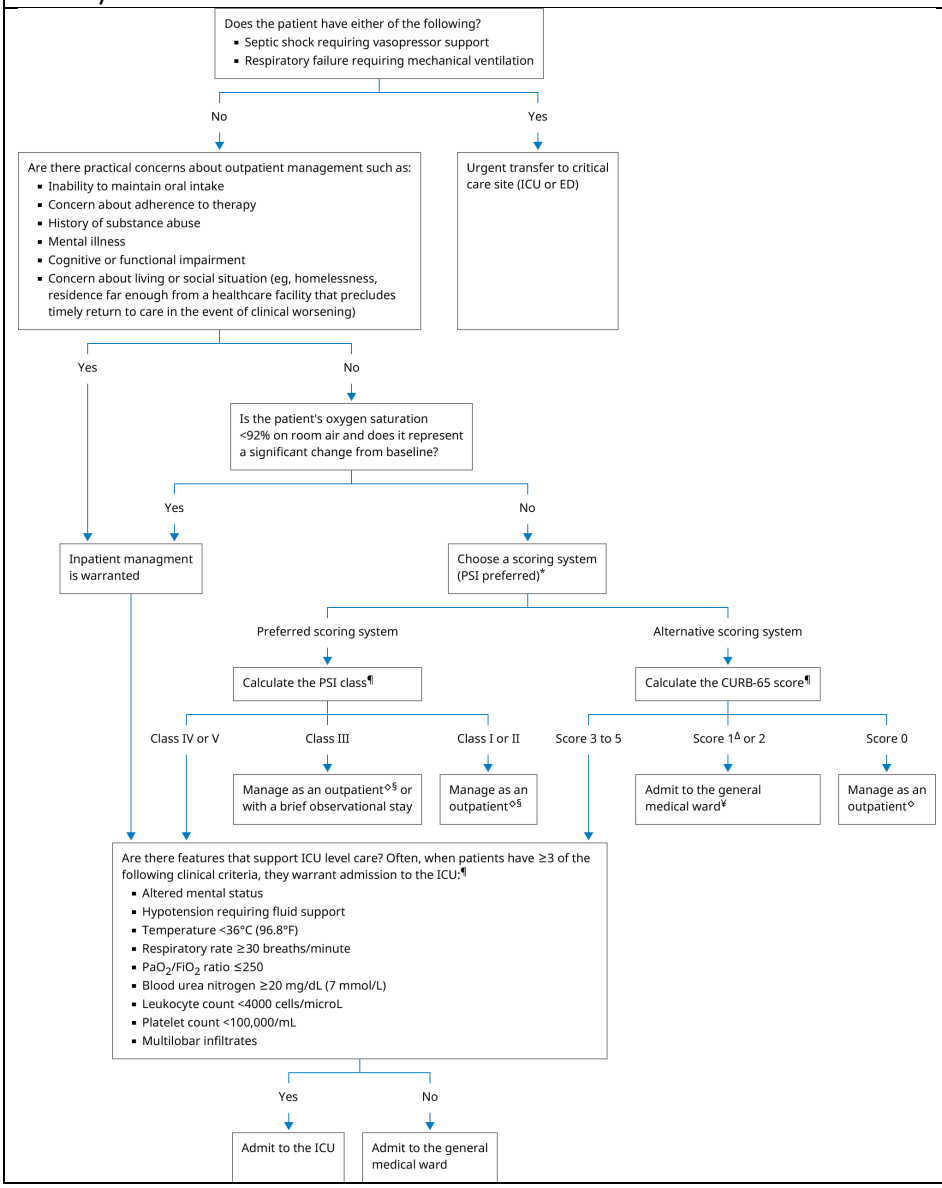
Confusion present?

B**U**N >19

RR >= 30

S**B**P <90 or DBP <= 60

65+ yo old



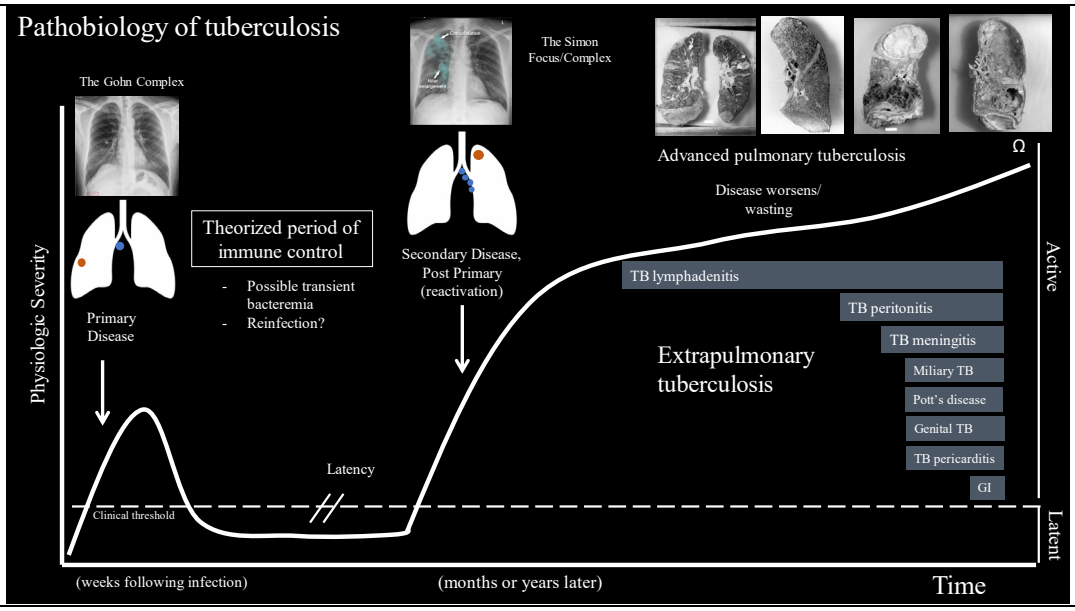
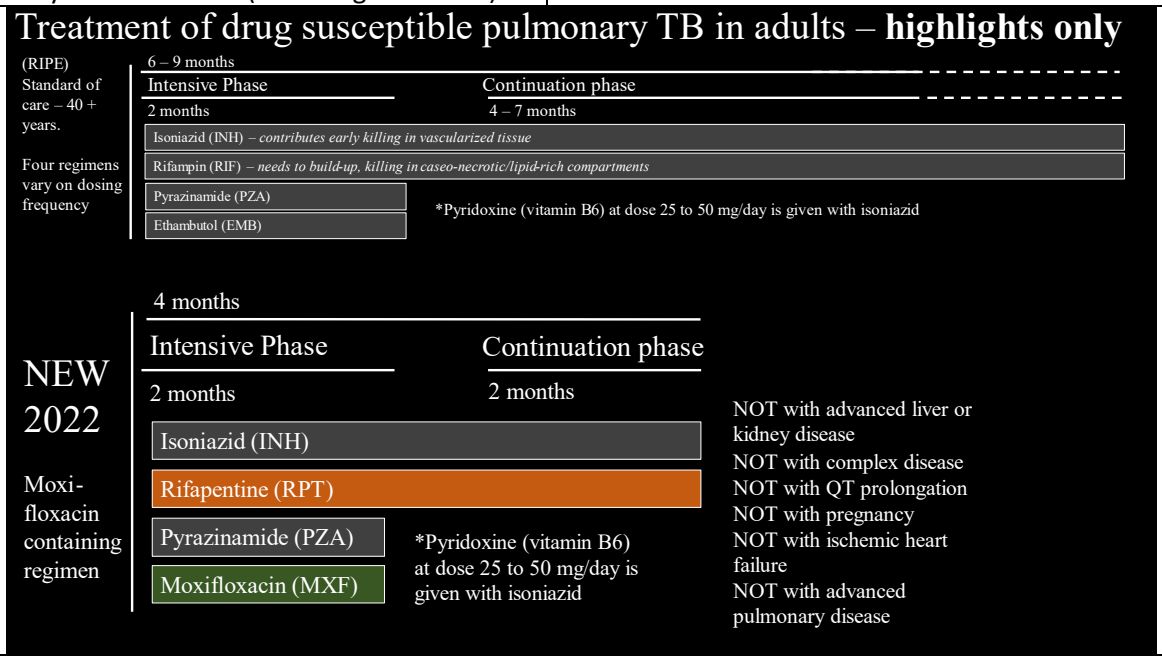
Coccidiomycosis

Coccidiomycosis aka "Valley Fever"
1. Dimorphic fungus – "Mold in the cold, yeast in the heat" • *Coccidioides immitis* (from California) and *Coccidioides posadasii* (all other endemic areas – think SW US!) 2. Acquired by inhalation of airborne arthroconidia 3. Refractory community acquired pneumonia is the most common clinical presentation 4. Fluconazole is the first-line treatment for symptomatic infection 5. Extended periods of therapy and surgical resection possible 6. 6-12 weeks fluconazole in immunocompetent with mild/moderate dz; 12-24 weeks of tri-azole therapy in severe dz

PJP Pneumonia

Causative Organism	*Pneumocystis jirovecii* (previously *carinii*) fungus	
Risk Factors	**HIV/AIDS (CD4 <200)** **Chronic Glucocorticoid use (and other immunosuppressives)** Inherited immune deficiencies, hematopoetic cancers, transplant, severe malnutrition	
Clinical Presentation	(+) HIV ● Subacute infection (weeks to months) ● Fever/night sweats, cough, *progressive* dyspnea, insidious weight loss ● Oral thrush	(-) HIV ● Acute; 1 week from symptom onset to respiratory decompensation ● Fever, non-productive cough, hypoxemic respiratory failure
Prophylaxis	Indications: **CD4 count <200 in HIV** **20+ mg prednisone daily for 4+ weeks** Also – Bone marrow or solid organ transplant, ALL, certain meds, GPA on pred/cyclophosphamide	Regimen: **TMP-SMX** SS or DS tab PO daily (preferred) Alternate - DS tab PO MWF If SJS/TEN/other Bactrim contraindication: pentamidine, dapsone, atovaquone
Diagnosis	Labs **ABG** with disproportionate hypoxemia **Positive PCR** from BAL (99% SN, 89% SP) or induced sputum (99% SN, 82% SP) **Fungitell** (beta-D-glucan) – SN 91%, SP 79% If >200, SN 70%, SP 100%! Many false positives (gauze, HD filters, blood products, e.g.)	Imaging **CXR**: normal or reticular opacities; PTX possible from pneumatoceles **Dry CT Chest**: GGOs, patchy/mosaic; can be nodular pattern less commonly
Treatment	**Start Prednisone 15-30 min before starting anti-PJP therapy** **TMP SMX 2 DS tabs PO Q8hr x 21 days (or IV if unable to take PO)** Prednisone 40 mg PO Q12h x 5 days → 40 mg q24h x5 days → 20 mg Q24h x 11 days Alternatives: Clindamycin + Primaquine OR Atovaquone OR Pentamide If new HIV – should start HAART within 2 weeks of starting PJP treatment – low IRIS risk	

Tuberculosis

Organism	*Mycobacterium tuberculosis* – a pesky, resilient, virulent bacterium – acid-fast staining with thick walls
Clinical Presentation Hallmarks of active disease include weight loss, cough, night sweats, fever, hemoptysis, fatigue, lymph-adenopathy	
Screening	At risk for new infection: close contacts with TB, homeless shelters, prisons, HCWs, drug use Increased risk of progressive LTBI: HIV, transplant, chemo, abnormal chest imaging, ESKD on HD, TNF-alpha, DM, long term glucocorticoids Mild increase in progressive LTBI: BMI<=20, Pack per day TUD, Solitary nodule, Pts from high-risk countries TST/PPD: Inactivated TB extract, place at T0, read at T+48-72hr (FP: BCG vax, prior TB, many PPD, NTM exposure) - If positive -> IGRA IGRA/Quantiferon/T-spot: Assay measuring IFN-gamma response to antigen (No false positive for BCG vax!) - If positive -> chest imaging to determine latent or active TB
Diagnosis	Latent TB Persistent immune response to TB (positive PPD or IGRA) WITHOUT evidence of active infection (sxs or rads findings) Active TB Reasonable risk factors with Signs/symptoms of known or presumed TB infection Chest imaging or Microbiologic evidence (AFB smear, culture, Gene Xpert NAAT)
Treatment	Latent TB falls within PCM wheelhouse INH-RPT weekly x 3 months OR Rifampin daily x 4 months OR INH-RIF daily x 3 months (other regimen exist) Active TB tx does NOT require definitive diagnosis (culture or molecular testing positive for TB) - favored based upon pt factors, lab/imaging findings, clinical suspicion, and public health concerns
Active TB tx Regimens: **RIPE** **Complicated – Consult ID!!** ****Duration and regimen are dependent upon the presence of cavitary lesions, culture positivity, sputum and radiographic monitoring**	

Enteric Infections (Infectious Diarrhea)

First Line Diagnoses (consider in all)	Immunocompromised?	Returning Traveler?
Rotavirus, Norovirus*	**Cryptosporidium***	**Malaria***
ETEC*	**Cyclospora***	**S. Typhi***
C. Diff*	**Cystoisospora***	**Entamoeba Histolytica***
Salmonella*	Microsporidia	**Giardia***
Campylobacter*	Mycobacterial Avium Complex	**Vibrio cholera***
Shigella*	**CMV colitis***	**Vibrio sp.***
Yersinia*		
STEC/EHEC*		
+/-Giardia	RED = Often presents as bloody diarrhea	**BOLD*** = High yield for boards

When Should I use antibiotics?

- Walter Reed Sanford Guide
  - Age <1 or >50 years
  - Immunocompromised
  - iHD
  - Vascular aneurysms, grafts, prosthetic joints
  - Hemoglobinopathy
  - Hospitalized + Fever + 9-10 stools/day

What antibiotic should I use?

- Campy: Azithromycin (most common Rx for travelers), FQ second line
- Salmonella: FQ (if no intl travel)
- Shigella: FQ (2nd line azithromycin or ceftriaxone)
- ETEC: FQ
- Yersinia: FQ (ceftriaxone + gentamicin for severe)

Loperamide is commonly co-prescribed for travelers (despite predominant bacterial etiology), but is **generally NOT recommended for bacterial diarrhea** (incl C diff).

Staph aureus bacteremia

Cultural Clues (or Clangs)

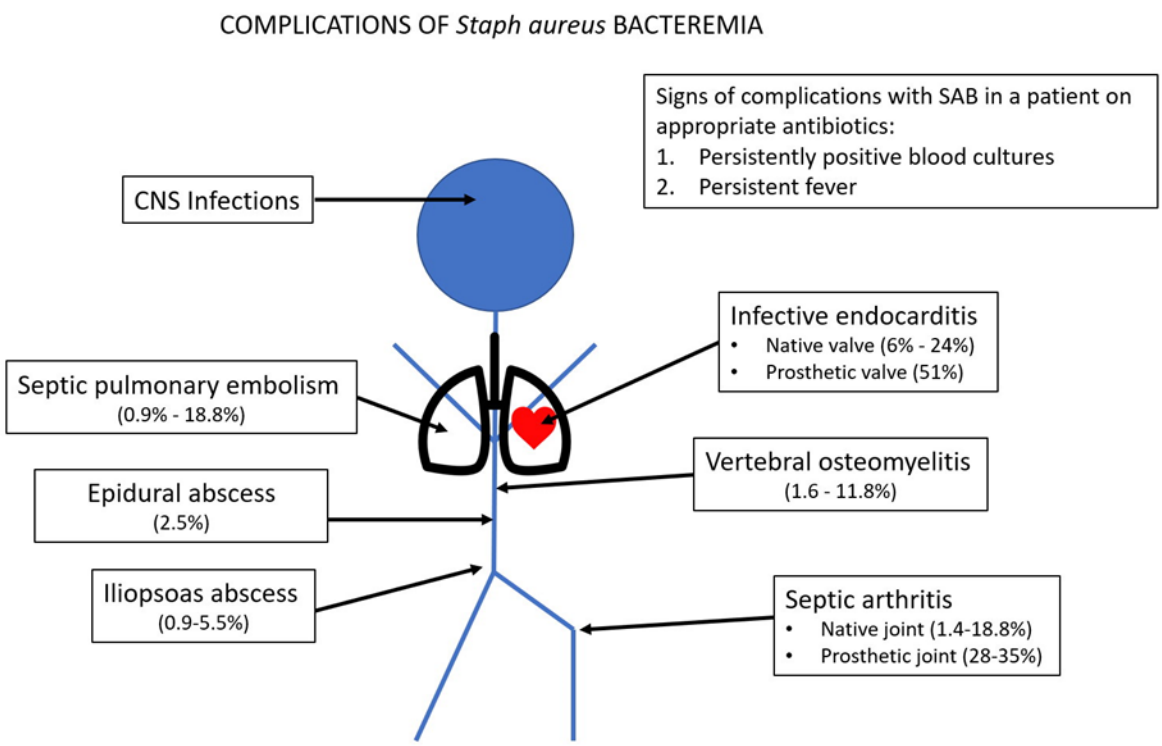
Gram positive cocci in clusters
Gold-yellow colonies with Beta-hemolysis pattern on blood agar
Catalase positive, Coagulase positive, beta-lactamase producing, modified PBPs

BCID (a WR godsend) can help identify MRSA early – mecA positivity!

Risk Factors for Bacteremia

Hemodialysis (RR 257.2) and Peritoneal dialysis (RR 150.0)
HIV (RR 23.7), Solid-organ transplant (RR 20.7), Heart disease, cancer
IV drug use (RR 10.1), EtOH use disorder, DM, Stroke, COPD

Complications



YOU MUST take a thorough history and full-body physical & consult ID !

Grab a TTE – when increased suspicion for IE (cultures not clearing, persistent fever). Prediction scores (VIRSTA eg) can help
Consider PET/CT – to eval for other metastatic sites of infection
Other imaging/diagnostics per suspected site (I&D, spinal MRI, etc)

Antibiotics (Invasive = IV always)

MRSA Meds

(Duration varies with complications: 2 weeks uncomplicated, 4-6 weeks complicated)

- Vancomycin
- Daptomycin (monitor weekly CK, don't use in alveolar-space infection)
- Llinezolid or ceftaroline
- Long-acting: dalbavancin

MSSA Meds:

- Anti-staphylococcal penicillins (nafcillin, oxacillin, e.g.) – NOT vanc
- Cefazolin ("ancef yay" – perioperative colleagues everywhere)
- Salvage: ertapenem + cefazoline

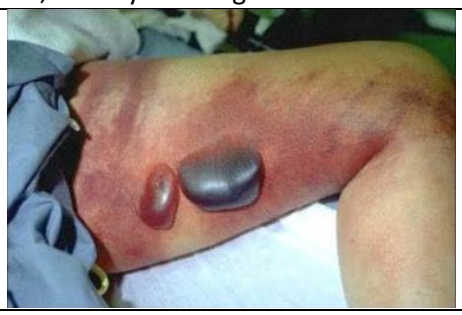
Cellulitis

<table>
<tr><td>Definition</td><td colspan="6">Localized, unilateral infection (dermis), often poorly demarcated and 2/2 microtrauma or barrier disruption
- Differs from Erysipelas (epidermis) in its lack of clear demarcation</td></tr>
<tr><td>Evaluation</td><td colspan="6">History: Identify time course/progression, animal/bite/nature/topical exposures, diabetes status
- MRSA Risk factors: Penetrating trauma? Recent abx use? iHD? Hospitalization or abx within 3 mo? IVDU? LTC/SNF resident?
- Other Red Flags: Toxic shock, gas gangrene, compartment syndrome
- Nec fasc features: pain out of proportion, rapid spread, evidence of necrosis/slough
- Mimics: sxs of VTE or predisposing hx for VTE; contact dermatitis, chronic venous stasis
- Can utilize ALT-70 score (>5 == 85% likelihood!)
Exam: Fluid collection? Well-demarcated borders? Unilateral? Fissuring/maceration/skin breadown? Crepitus?
Labs: CBC, BMP ± Culture (I&D material if drained; BCx ONLY if severe/sepsis)
Imaging: DVUS for lower extremity to r/o clot; CT of affected extremity/area</td></tr>
<tr><td rowspan="2">Class</td><td colspan="2">Mild</td><td colspan="2">Moderate</td><td colspan="2">Severe</td></tr>
<tr><td>Non-Purulent</td><td>Purulent</td><td>Non-Purulent</td><td>Purulent</td><td>Non-Purulent</td><td>Purulent</td></tr>
<tr><td>Features</td><td>Localized, typical without fluid collection or purulence, no sxs sepsis or immune suppression</td><td>Localized with fluid collection, no sxs sepsis or immune suppression</td><td colspan="2">Add systemic signs/symptoms; Often a/w rapid progression, failure of first-line interventions</td><td colspan="2">Add systemic sxs + hoTN, immune compromise, deeper infxn (bullae, slough), or nec fasc</td></tr>
<tr><td rowspan="2">Treatment</td><td>Amoxicillin
Cephalexin</td><td>I&D
Doxy, Bactrim for MRSA</td><td>Cefazolin, CTX; Clinda*</td><td>I&D; Vanc, Dapto, Linezolid – bactrim, doxy</td><td>Vanc/zosyn, carbapenem, Surgical eval</td><td>Surgery; Vanc, Dapto, Linezolid, Ceftaroline</td></tr>
<tr><td colspan="6">If toxin-mediated moderate-severe infection, may add clindamycin!
Treatment course: 5-7d for mild-moderate infection (longer tx for diabetic foot, poor source control, severe infxns)
Treat predisposing factors (tinea pedis, edema, onychomycosis, CVI, stasis dermatitis)
Prophylaxis if ≥3 episodes per year!</td></tr>
</table>

Animal Bites

Common pathogens (typically mixed aerobe/anaerobe)	Pasteurella (50% dog bites, 75% cats), Capnocytophaga canimorsus, Eikenella corrodens (clang: human bites), Streptobacillus moniliformis (rats), Oral anaerobes, human skin flora
Immediate Management	1. Clinical evaluation (find all wounds, determine depth, necrosis, infxn risk) 2. Irrigation and debridement (cultures prn, wash out all, ID/Ophtho/Surg c/s) 3. Primary (less scar, more infxn) or delayed closure
Indications for Abx	If clearly infected → Abx after cultures taken and wound irrigated If not infected, indications for abx prophylaxis: - Mod/severe wound - MOST cat bites - Puncture or crush wound - Face/hand/genital/joint involved - Wound near bone - Wound requiring surgical closure - Immunocompromised (T2DM, asplenia, e.g.) - Venous or lymphatic insufficieny present
Abx regimens	Treatment: Same as PPX but course may extend to 14d or longer per wound severity Prophylaxis: Augmentin 875/125 mg PO q12h x3-5d (Alt: Doxy/Bactrim/Levoquin + Flagyl or Clinda) ** a dose of IV abx ok if c/f severe infection – Talk to ID!
Indications for Primary Closure (Sutures)	Secondary intention ok for many/most after wound washout! EXCEPT: 1. Non-puncture dog bit 2. Non-infected wound 3. Bite <12 hrs old (<24 hrs on face) 4. Not on hand/foot **DO NOT CLOSE**: Cat/human bites (?face ok), puncture/crush wounds, clearly infected wounds, bites >12 hours old or >24 hrs on face, hands/feet, immunocompromised pts
Indications for Vax/Immunoglobulins	Tetanus - Vax: Give Td in adults with <3 or unknown tetanus vax hx OR if >10 yrs since last and clean/minor wound or if >5 years since last and severe/dirty wound - IM IG: Given for pts with severe/dirty wounds AND <3 total Tetanus vax; ALL HIV or severe immunodeficiency regardless of tetanus vax hx Rabies (high concern: bat/bear/beaver/raccoon/fox/otter, medium: cat/cow/dog) - Vax: Pre-bite risk per occupation, call ID/Immz, post-exposure 3-dose vax - IG: call ID/Immz, 20 IU/kg body weight into site (remainder IM bc usually 5-10 mL)

Streptococcal Toxic Shock Syndrome

Definition	Dysregulated immune response to invasive streptococcal infection (Group A > C, G, B), usually skin/soft tissue, but can arise from PNA, septic arthritis, peritonitis, GYN infections, etc
Epi	Rare (2.3 cases per 100K) but occurs 10-30% of patients with invasive Group A *Strep* (GAS) == *strep pyogenes*
Risk Factors	Malignancy, minor trauma, HIV, DM, homelessness, IVDU, immunosuppression, postpartum (48-72h)
Pathophys	Pathologic response to exotoxin or superantigen (usually from GAS but can be from others) => TNF-a, IL-1, IL-6 release => massive cytokine storm, capillary leak, multisystem organ failure
When to Suspect	Rapidly evolving pathology requiring rapid recognition: - Necrotizing fasciitis - Refractory hypotension - Multisystem organ failure (ARF, DIC, ALF, ARDS, e.g.) - Skin/soft tissue source with pain out-of-proportion to exam (20%) - Blanching erythematous rash (macular and/or bullous) – classic strawberry tongue is rare 
DDx	Infectious: Staphylococcal Toxic Shock (think retained gauze/packings/tampons), Typhoid, GNR sepsis, Rocky Mountain Spotted Fever, Leptospirosis, meningococcemia Non-infectious: Kawasaki disease, Heat stroke
Treatment	1. Aggressive sepsis management 2. Start empiric broad spectrum antibiotics (vanc/zosyn, e.g.) + **CLINDAMYCIN** (toxin-binding) - Narrow broad-spectrum antibiotics per sensitivities 3. Surgical debridement of focal nidi (e.g. nec fasc) == survival benefit! 4. IVIG 1 g/kg on day 1 and 0.5 g/kg on days 2 and 3 == another toxin-binder with evidence in Strep TSS

CSF Interpretation

Etiology	WBC	Predominant Cell Type	Glucose	Protein	Opening Pressure
Viral / Aseptic	50-100	Lymphocyte	>45	<200	Normal or Slight Elevation
Bacterial	1000-5000	Neutrophil	<40	100-500	Elevated
Tuberculous	50-300	Lymphocyte	<45	50-300	Variable
Cryptococcal	20-500	Lymphocyte	<40	>45	Elevated

Meningitis and encephalitis

Syndrome	Community-Acquired Bacterial Meningitis	Encephalitis
Pathophys/Site of Infx	Inflammation of the meninges/subarachnoid space, with CSF pleocytosis (>5 wbc, typically far more than this…)	"inflammatory process of the brain in association with clinical evidence of neurologic dysfunction"
Problem Representation →Sxs/Signs/Time Course? →Labs? →Imaging/other Dxs?	Acute fever, HA→AMS as manifestation of severe dz Peripheral leukocytosis often CSF: elevated WBC, L shift, low glucose, elevated protein Imaging: meningeal enhancement (CT in certain pts…)	Acute/subacute AMS, fever, HA Plus/minus peripheral leukocytosis CSF: slightly elevated WBC, lymph predominant commonly (some can have L shift), glucose wnl, protein nl or minimally elevated Imaging: MRI can show localized disease based on infecting organism or be WNL
Top 3-4 Organisms (US adults)	*S pneumonia* > *N meningitidis* Older >50/Immunocompromised: *L monocytogenes* Aerobic GNB	Undiagnosed ~50% of the time HSV-1 (25%), Enteroviruses (25%), VZV (15%), WNV (10%), EBV (10%) Acute immune mediated 21%!!
Empiric Drug(s)	Vanc (trough-based dosing, NOT AUC) + Ceftriaxone 2 gm IV q12H ADD Dexamethasone 10 mg q6H x 4 days (or until not *S. pneumo*) – hearing protection! Age>50/immunocomp: ADD Ampicillin 2 g iv q4h PCN Allergy?? TMP/SMX or Meropenem	Acyclovir 10 mg/kg iv q8h IBW in obesity! (or adjusted in class 3 obesity w/ severe disease!)

West Nile Virus

Transmission	Mosquitoes (*Culex spp*), organ transplant, breastfeeding, blood transfusion Natural reservoir: birds Worldwide distro! First outbreak in US in 1999
Clinical Manifestations	80% asymptomatic! 20% have West Nile Fever - ILI -> fever, HA, nausea, morbilliform rash in 25-50% <1% have Neuroinvasive disease: meningitis (30-40%) or encephalitis (50-60%) - Risk: >60 yo, HTN, DM, Immunocompromised - Common signs unique to WNV: Tremor, Parkinsonism, Myoclonus (esp of face and UEs)
Diagnosis	IgM > PCR (PCR only positive in <60%)
Treatment	Supportive care
Prognosis	Mortality 12%, high rates of long-term neuro sequelae (20% ongoing sxs at 18 mos)

Measles

Virus	Single-stranded, enveloped RNA paromyxovirus (Genus: morbillivirus == morbilliform rash!) Viral transmission via respiratory mucosa or conjunctivae, then spreads via lymphatic tissues

Presentation	• **Incubation (6 – 21 days)**
	• **Prodrome (2-4 days, up to 8) = symptom onset**
	  • Fever, malases, anorexia → conjunctivitis, cough, coryza (rhinorrhea), Koplik spots
	  • Symptoms intensify a few days before the rash appears
	• **Exanthem (2-4 days after fever onset, lasts 3-5 days)**
	  • Diffuse maculopapular, blanching erythematous rash
	  • Starts on face, spreads cephalocaudally and centrifugally
	• **Recovery**
	  • Cough may persist 1-2 weeks
	  • Fever beyond day 3-4 suggest complication
	  • Skin may desquamate
	  • Lifelong immunity (usually)
	 

Complications
 	
Develop in 30%
	• **Death** (1-3 per 1000 cases)
	• Pneumonia
	• Encephalitis (1 in 1000)
	• Subacute sclerosing panencephalitis (SSPE)
	  • Cognitive decline, chorioretinitis, blindness, gait/myoclonus issues, vegetative states – multi-staged dz
	  • 1 in 1000 children
	  • Usually infants <2 yo
	  • Can develop between 7-10 years from initial infection
	  • Many others exist (GI, immune suppression, etc) but are less common

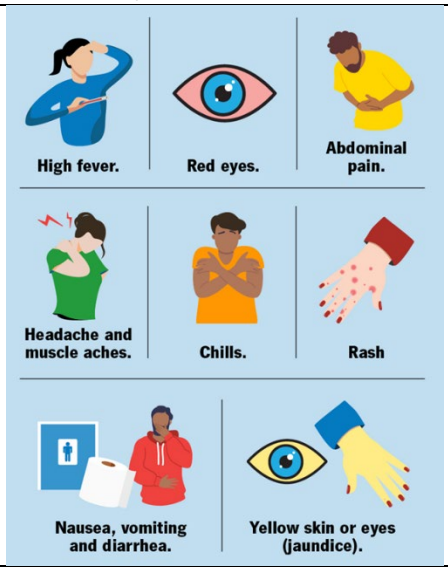
Testing	• **If suspicion is high based on sxs/exposure → Isolate patient**
	  • Airborne precautions!
	• **Call ID + Prev Med (301-400-0075)**
	• **Confirm lack of immunity** (born after 1957, no documented MMR vax or titers)
	• **Oropharyngeal PCR swab >> IgM serologies**
	• Collect
	  • Swab ideally within 3 days of rash onset
	  • Serologies after 3 days increases IgM sensitivity

Treatment	• **Vitamin A** - ALL children; adults if hospitalized
	  • May reduce severity and complication risk incl mortality
	  • Oral administration x2d
	  • Age >=12 mo – 200,000 international units daily
	• **Ribavirin**
	  • For use in measles pneumonia and the immunosuppressed
	  • 15-20 mg/kg per day PO divided in 2 doses (5-7d suggested)
	• **Else, supportive care**

Helminth Infections

Condition	Agent	Transmission	Manifestations	Treatment
Strongyloidosis	*Strongyloides stercoralis* – a nematode	Skin -> travel to lungs -> auto-aspirated into GI tract	Asx up to 50% GI: epigastric pain, pyrosis, occ diarrhea/constipation, early satiety Pulm: wheeze, transient infiltrates Skin: urticaria, larva currens Labs: peripheral eosinophilia	Dx: Stool O/P low sensitivity, Serologies can vary in efficacy, PCR Tx: Ivermectin 0.2 mg/kg/d x 2 days 2nd line – Albendazole 400 mg daily x 3-7d
Hyperinfection Syndrome	*Strongyloides stercoralis*	Immunocompromise (steroids, TNF-a, HTLV-1, Ca, malnutrition) with large burden of parasites	GI: N/V/D, abdominal pain, intestinal erosions Pulm: diffuse infiltrates, SOB, cough, hemoptysis, pneumonitis, edema Neuro: GNR polymicrobial meningitis System: GNR sepsis/shock +/- peripheral Eos	DX: Stool O/P usually more sensitive than in chronic TX: Decrease immunosuppression, Ivermectin 0.2 mg/kg/day until stool O/P neg x 2 weeks PREVENT: Check PCR and tx if Pos OR empirically tx
Cutaneous Larva Migrans	Dog/Cat Hookworms *Ancylostoma caninum* *Anyclostoma braziliense* *Uncinaria stenocephala*	Infected animal feces -> soil or floor -> direct entry into skin (can't get thru basement membrane)	Creeping (<1-2 cm per day) serpiginous eruption under skin, usually 2-8 weeks after exposure; pruritius;	Often self-limited Topical thiabendazole 10-15% BID-TID x5-10d Severe infestation: albendazole 400 mg daily x3-5d OR ivermectin 12 mg PO one time
Enterobiasis (Pinworms)	*Enterobius vermicularis*	Humans only host Enter humans through fecal-oral transmit -> lay eggs around anus at night, very itchy -> fecal-oral continues	Asymptomatic OR perianal itching Appendicitis is rare	Dx: Early AM "scotch tape test" – before shower/BM Tx: OTC pyrantel pamoate x1 OR mebendazole 100 mg PO x1 OR albendazole 400 mg PO x1 Repeat in 2 weeks! Treat all members of household; trim fingernails, wash hands, wash bedclothes
Neurocysticercosis	*Taenia solium*	Consumption of infected raw/undercooked pig meat; humans pass infection via stool to pigs	Seizure (common!), hydrocephalus, HA, focal neuro déficits	Dx: Head imaging with cysts containing scolex, perilesional edema (active), or scattered calcifications (chronic) – bx for definitive; serologies supportive Tx: **Steroids** if diffuse edema If ICP normal: 1-2 cysts -> albendazole >2 cysts -> alb + praziquantel May need NSGY intervention +/- long courses of steroids/antihelminthics if extra-parenchymal

Leptospirosis

Pathogen	Gram-indeterminate, aerobic spirochete *leptospira* – over 64 species recognized - Characteristic "question mark" appearance on microscopy
Epi	Tropical regions worldwide! Over a million cases annually (59,000 deaths ~5.9% mortality) - Areas with US military presence = recent outbreaks in Hawaii, Guam, Honduras, and Japan among ADSM - Resurgent in areas where animal-human interaction increased (urban slums, deforestation, etc) Transmitted in water/soil contaminated by infected mammal urine (primarily rodents, but others implicated too) - Leptospires enter via wounds or mucus membranes - Exposures are often recreational (kayaking, swimming, fishing), professional (harvesting, butchering, clamming), or domestic (cleaning, bathing, etc) Lifecycle – leptospires live in the renal tubules of their host before transmission into soil/water via urine
Condition	2-10d incubation period after transmission <u>Anicteric leptospirosis</u> (90-95% of cases) - Mono or biphasic: Acute and immune phases - Acute phase (4-7d) features abrupt-onset fever, HA, myalgia, nausea. Conjunctival suffusion is classic, but not that common! - Few proceed to immune phase after a period of symptomatic improvement (recurrent fever, uveitis, HA, aseptic meningitis, etc) <u>Icteric leptospirosis (Weil's Disease)</u> 5-10% of symptomatic cases - Fever, jaundice, renal failure classic (can rapidly progress to multisystem organ involvement with DAH/ARDS, myocarditis, liver dysfunction, etc) - 10-15% mortality rate 
Diagnosis	Challenging! Clinical suspicion must be high up front based upon exposure history and symptoms. - Evaluate for other febrile illnesses - Serum PCR – 40-60% sensitive, best earlier in the disease course (by day 7) - IgM/IgG Microscopic agglutination test (MAT) at start (often negative) and day 7 (more likely to be positive) – processed by CDC If you suspect lepto and want to test for it, PLEASE CALL ID!!!
Treatment	<u>Supportive care</u> (fluids, Tylenol, etc) <u>Mild cases</u> - Doxycyline 100 mg BID x7d - Azithromycin 500 mg daily x3d (pregnancy) <u>Moderate/Severe cases</u> - Complication management (ARDS, DAH, ARF, etc) - 7d of either: Doxycycline 100 mg IV q12h **OR** PCN 1.5 million units 16h **OR** Ceftriaxone 1-2g IV q24h **OR** cefotaxime 1g q6h IV **Risk of Jarisch-Herxheimer Reaction by 3rd day of treatment (~10% of cases) – supportive care (IVF, pressors, etc) but DON'T stop the antibiotics!
Prevention	<u>Environmental</u>: - Avoiding contaminated water/soils, clean drinking water wearing appropriate protection (boots, bandaging wounds, etc), washing/hygiene after exposure - Rodent / pest control, systemic fixes for poverty/deforestation/rapid urbanization/overpopulation (easy I know) - No human vaccine, but there are vaccines with limited efficacy for dogs and cows <u>Prophylaxis</u>: - Doxycycline 200 mg PO qWeekly while in endemic zone (e.g. water-based tropical training)

Nephrology

AKI Criteria and Differential

KDIGO Criteria		
1. Increase in SCr by ≥0.3 mg/dL in 48 hours **OR** 2. Increase in SCr to ≥1.5 times baseline in 7 days **OR** 3. Urine output <0.5 mL/kg/hour for six hours		

PRERENAL
- Low volume
  - Hemorrhage
  - Dehydration
  - Burns
- Low cardiac output
  - Heart failure
  - Massive PE
- Low SVR
  - Sepsis
  - Cirrhosis
  - Anaphylaxis
  - Anesthesia
	INTRARENAL
	- Glomerular
	  - Nephrotic/Nephritic
	- Tubules/Interstitial insult
	  - ATN
	  - Nephrotoxins
	  - AIN
	- Vascular
	  - Vasculitis
	  - TTP, HUS, HELLP
	  - Hypertensive emergency
		POSTRENAL
		- Upper tract
		  - Kidney stones
		  - Blood clot
		  - Extrinsic compression
		- Lower tract
		  - BPH
		  - Neurogenic bladder
		  - Cancer
		  - Urethral stricture
		  - Blood clot

Volume status assessment **Review Meds & History**	**Urinalysis & Microscopy** **Review Meds & History**	**Bladder Scan, Renal US** **Review Meds & History**

Exertional Rhabdomyolysis

Suspicion	Post-exertional myalgias, dark urine, UA with blood but no RBCs (myoglobinuria)
Diagnosis	Severe myalgias **AND** CK>5x ULN after exercise
Risk Factors	High intensity exercise unmatched to fitness level Hot and humid climate Dietary supplements (stimulants) Genetics (sickle cell trait, disorders of lipid or glycogen metabolism)
When to Admit (High-Risk Features)	CK > 20,000 U/L Possible compartment syndrome AKI Metabolic derangement (hyperK, hyperPhos, acidosis) Sickle cell trait Limited f/u
Management	Isotonic IVF targeting 200-300 ml/hr UOP until CK decreasing, Rest
Return to Duty	Follow USU CHAMP Guidelines!

Acid-Base Evaluation & Toxic Ingestion

Acid/Base: DDx

$H^+ + HCO_3^- \longleftrightarrow H_2CO_3 \longleftrightarrow H_2O + CO_2$

Anion Gap Metabolic Acidosis

↑ H^+

Lactic acidosis
Tissue hypoxia or impaired mitochondrial oxygen utilization result in mitochondrial dysfunction causing production of lactate to sustain glycolysis. There are three types:

Type A	Type B	Type D
tissue hypoxia	*impaired O2 utilization*	^ *D-Lactate**
hypoperfusion 2/2 shock, hypoxemia, anemia	*sepsis, medications/toxins, ↓ thiamine, malignancy (Warburg effect)*	*short gut syndrome (gut bacteria produce D-lactate)*

Ketoacidosis
Inability to use glucose as an energy source mobilizes fat stores, resulting in production of ketones.
The three primary ketones are ***beta-hydroxybutyrate*** (measured), acetoacetate, and acetone.
Etiologies: DKA, starvation, and chronic alcohol use

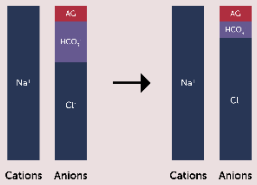
Renal failure
Progressive loss of functional nephrons results in an inability to excrete the daily acid load.
↑ ***AG occurs due to*** ↑ ***unmeasured anions***: urate, phosphate, sulfate, hippurate, etc.

Toxic ingestion
Ingested chemicals are rarely acids, but they may be metabolized into acids or induce acidosis.
Common AGMA-inducing toxins: ***alcohols*** (methanol, ethylene glycol), ***salicylates*** (aspirin)
Some alcohols are metabolized into acids, while aspirin leads to the buildup of lactic acid and ketones.

*The normal lactate lab measures L-lactate; D-lactate is a distinct lab.

Non-Anion Gap Metabolic Acidosis

↓ HCO_3^-



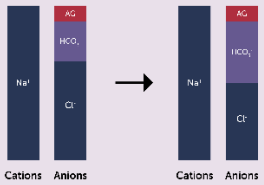
GI loss of HCO_3^-
diarrhea, fistulas, surgical drains, ureteral diversions

Renal loss of HCO_3^-
diuretics (acetazolamide)
↓ mineralocorticoids
renal tubular acidosis (RTA; types I, II, and IV)

HCO_3^- shift
normal saline
(↑Cl^- shifts bicarb intracellularly)

Metabolic Alkalosis

Develop: ↓ H^+ or ↑ HCO_3^- Maintain: ↑ HCO_3^-



GI loss of H^+
vomiting, continuous/prolonged NGT suction

Renal loss of H^+
diuretics (loop, thiazide)
↑ mineralocorticoids
Bartter's, Gitelman's, Liddle's

HCO_3^- retention
↓ volume, K^+, or Cl^-
↑ mineralocorticoids
milk-alkali syndrome (renal insuff. + exogenous alkali)

Respiratory Acidosis

↓ Ventilation ⟶ ↑ CO_2

CNS depression
drugs, neurologic disease or injury

Obstructive lung disease (↑ dead space)
Neuromuscular disease
PE (very early, and late if severe)

Ventilator MV set too ↓ for clinical situation
pts w/ met. acidosis who are unable to overbreathe vent

Respiratory Alkalosis

↑ Ventilation ⟶ ↓ CO_2

CNS stimulation
drugs, disease, fever, anxiety, pain

Compensation for metabolic acidosis
Pregnancy
PE (early, as a response)

Ventilator MV set too ↑ for clinical situation
pts whose met. acidosis is now resolving w/ treatment

Acid/Base: Evaluation

VOCABULARY

-OSIS | ***process*** -EMIA | ***outcome***

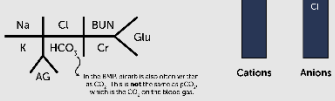
PRINCIPLES

1. Multiple acute metabolic processes **can** coexist
2. Multiple acute respiratory processes **cannot** coexist
3. Metabolic & respiratory processes **can** coexist
4. There can be **many** -oses but only **one** -emia!

APPROACH

1 Start with the *Anion Gap (AG)*. Why?
You may not have a blood gas yet, but you likely have a BMP and ^**AG = AGMA!**

Recall: total cations = total anions.
While it is impractical to measure all ions in the blood, we infer the presence of clinically relevant unmeasured ions (particularly those which cause acidosis) through changes in the balance or concentrations of those we do measure. The **anion gap** is one tool that uses commonly measured ions in the BMP to assess for unmeasured ions.

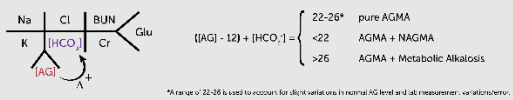


Anion Gap = Na^+ - (Cl^- + HCO_3^-)
Generally, AG >12 is high!

What if it's low (<5) or negative?
↑ **Unmeasured Cations**
(K^+, Ca^{++}, Mg^{++}, Li^+, paraproteinemia)
↓ **Unmeasured Anions**
(hypoalbuminemia)
Chloride Mimics
(salicylate / bromide / iodide toxicities)

2 Next, are they more *acidotic* or *alkalotic* than expected based on the AG?
Compare the *change* in bicarb to the *change* in AG (delta/delta) to see!

In a pure AGMA the ↑ **in AG** should equal the ↓ **in bicarb**.
In other words, the Δ AG + the *measured* bicarb should equal ~24 (the normal value for bicarb)!

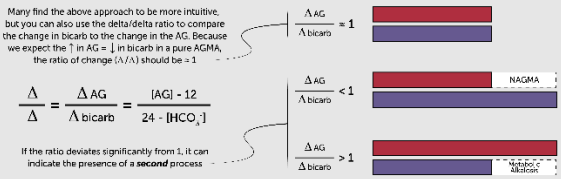


*A range of 22-26 is used to account for slight variations in normal AG level and lab measurement variation/error.

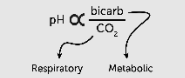
Many find the above approach to be more intuitive, but you can also use the delta/delta ratio to compare the change in bicarb to the change in the AG. Because we expect the ↑ in AG = ↓ in bicarb in a pure AGMA, the ratio of change (Δ/Δ) should be ≈ 1

$$\frac{\Delta}{\Delta} = \frac{\Delta AG}{\Delta bicarb} = \frac{[AG] - 12}{24 - [HCO_3^-]}$$

If the ratio deviates significantly from 1, it can indicate the presence of a ***second*** process

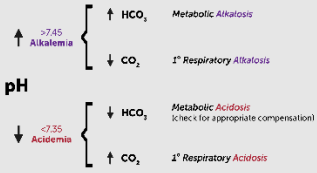


3 Now, look at the gas. First, do you *need* an ABG?
Generally, for ***acid/base***, a VBG approximates an ABG by adding ~0.05 to pH and ~5 mmHg to pCO_2.



1° process is **metabolic** when pH and bicarb go in the **same** direction!

1° process is **respiratory** when pH and pCO_2 go in **opposite** directions!



4 The final step is to assess for the ***chronicity*** (acute, chronic, or acute on chronic) of the disorder and for the appropriate ***compensation*** which can be done by using these formulas:

Metabolic Acidosis
Winter's Formula: $pCO_2 = (1.5 \times [HCO_3^-]) + 8 +/- 2$

Metabolic Alkalosis
$pCO_2 = (0.7 \times ([HCO_3^-] - 24)) + 40 +/- 2$

These formulas give the ***expected pCO2*** if the patient is ***appropriately*** compensating for their metabolic acidosis/alkalosis!
Also, appropriate compensation does **NOT** require the pH to be 7.4 (and it rarely will be)!

Respiratory Acidosis
Acute: ΔpCO_2 ^ 10 → ΔHCO_3^- ^ 1 or ↓ pH 0.08
Chronic: ΔpCO_2 ^ 10 → ΔHCO_3^- ↑ 4 or ⌄ pH 0.03

Respiratory Alkalosis
Acute: ΔpCO_2 ↓ 10 → ΔHCO_3^- ↓ 2 or ^ pH 0.08
Chronic: ΔpCO_2 ↓ 10 → ΔHCO_3^- ⌄ 5 or ↑ pH 0.03

Toxic Ingestion Eval

Methanol (paint thinner, windshield wiper fluid)
- Sx: visual blurring, central scotoma, afferent pupillary defect, AMS
- Lab: HIGH osmolar gap, HAGMA

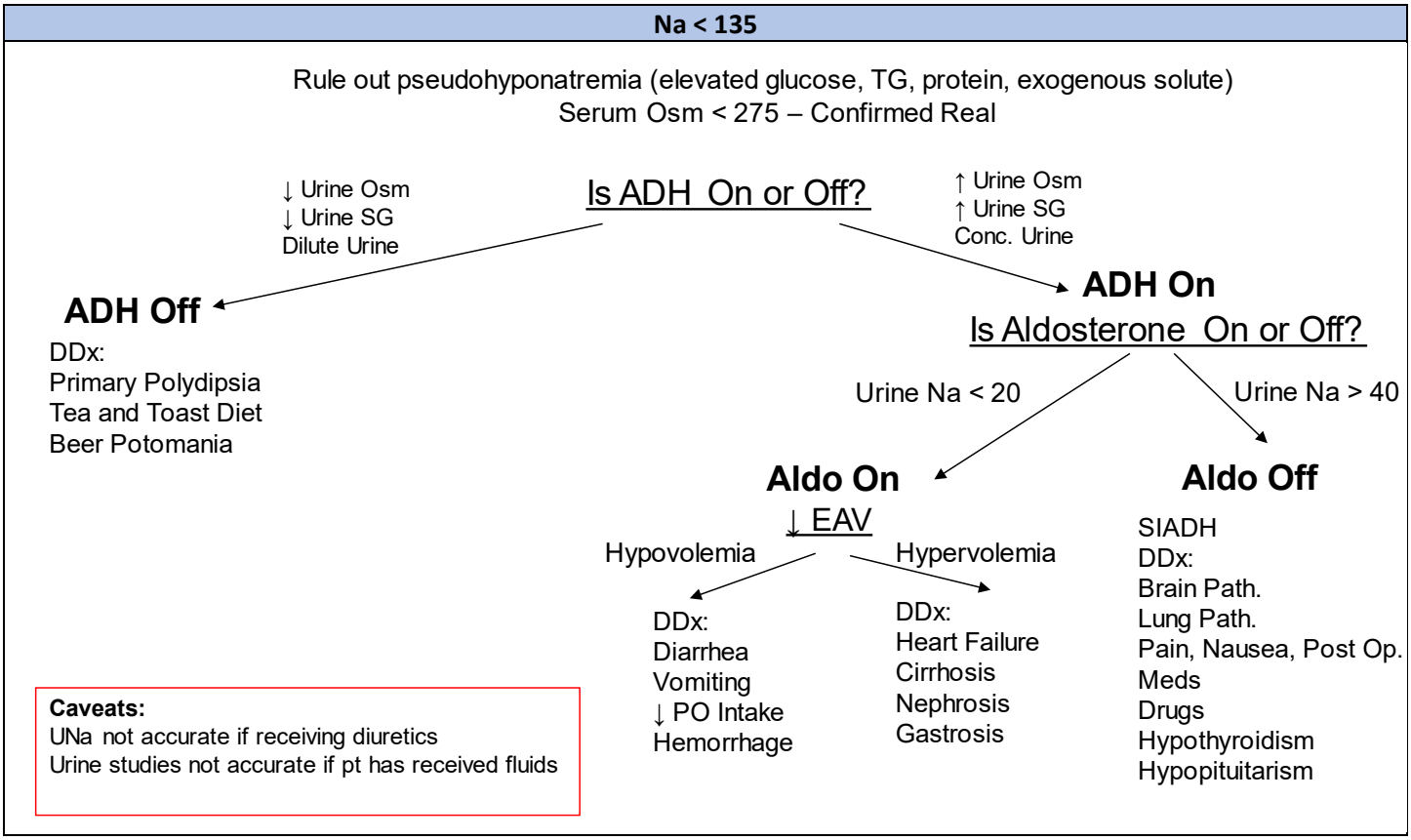
Ethylene glycol (Antifreeze)
- Sx: Flank pain, hematuria, oliguria, CN palsies, tetany
- Lab: HIGH osmolar gap, HAGMA, renal failure, calcium oxalate crystals in urine (and on kidney biopsy)

Isopropyl alcohol (rubbing alcohol, mouthwash)
- Sx: CNS depression, dysconjugate gaze, absent ciliary reflex
- Lab: HIGH osmolar gap, NO anion gap or metabolic acidosis

Hyponatremia

Step 1	Is something wrong with brain or beans?	Brain (recent stroke, injury, etc): cerebral salt wasting, genetic/acquired change in osmostat setpoint Kidney: diuretic use, eGFR<15 (cannot dilute urine)
Step 2	What's the tonicity? - Calculate Sosm = 2*Na + BUN/2.8 + Glucose/18 (Nl = 285-300) - Obtain Uosm, UNa, Sosm	Hypertonic? Consider hyperglycemia Isotonic? Consider lab artifact due to high triglycerides or immunoglobulins Hypotonic? Everything else
Step 3	Assess volume status	Hypervolemic? CHF, Cirrhosis, or Nephrosis Hypovolemic? Expect renal reabsorption of salt/fluid, so CONCENTRATED urine (UOsm >300) with LOW sodium (UNa <20)
Step 4	Assess causes of euvolemic hypoNa Obtain TSH, AM cortisol + ACTH + Urate	Low UOsm – Primary polydipsia, Tea/Toast High Uosm, High UNa – Adrenal insufficiency, Hypothyroidism, Thiazides, SIADH

Hyponatremia: More Traditional Approach



Hypernatremia

Hypernatremia (Na > 145)	
Water losses (MOST cases)	**Intake of salt without water**
Water unavailable or loses access to water Thirst drive is impaired or too altered to drink Loses water via GI, skin, lung, DI, or diuretic	Salt poisoning Iatrogenic (3% NS, Na Bicarb, Valproate tox, e.g.)
1. Evaluate water intake 2. Evaluate potential water losses == Obtain Uosm and Sosm a. If Uosm < Sosm → urine too dilute → think diuretic or diabetes insipidus b. If Usom > Sosm → urine appropriately concentrating → probably GI, skin, or mucous membrane loss 3. Evaluate increased salt intake	
Diabetes Insipidus – ADH aka vasopressin ~ desmopressin aka DDAVP Increase ADH = Increase water reabsorption = increase urine concentration	
Central - ADH deficiency (will concentrate urine with DDAVP) - Brain cannot produce hormone to reabsorb water - **Etiology**: brain trauma/surgery/anoxia/tumor, cerebral hemorrgahe, neurohypophysitis, CNS infxn, Lithium, Toluene, Hereditary or idiopathic - **Treatment:** DDAVP oral or intranasal or subQ first-line; can consider thiazide and/or NSAIDs	**Nephrogenic** - ADH resistance (will not concentrate urine with DDAVP) - Kidney doesn't respond to hormone to reabsorb water - **Etiology:** Medication-induced (lithium, Ampho B, ofloxacin), HyperCa (>12), HypoK (<3), renal dz (ADPKD, sickle cell), Hereditary, Chronic AVP-D - **Treatment:** Thiazide + <2g/day Na restriction, consider NSAIDs

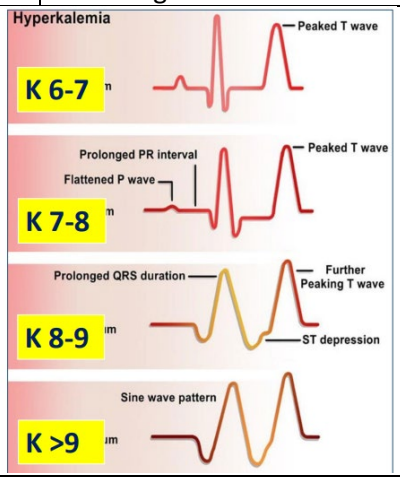
Hypokalemia

K < 3.5				
Causes	Intracellular Shift	Urinary Loss	GI Loss	Other
	Catecholamines Beta-agonist (albuterol) Hypothermia Insulin Increased RBCs	Diuretics, Polyuria RTA Hypomagnesemia Amphotericin B Bartters/Gitelman's	Vomiting/Diarrhea Tube drainage Laxatives	Decreased K intake Increased sweat loss Dialysis Plasmapheresis Spurious lab value
Sxs	Generally asymptomatic! EKG changes: U-waves, QT prolongation, Torsades (worsened if on digoxin, myocardial scarring, QTc meds) Other: Ileus, constipation (moderate); muscle cramps, weakness, arrhythmia, diaphragm weakness (severe)			
Treatment Repletion guide in your handbook! 😊	- Confirm K with repeat BMP & get simultaneous Magnesium level ○ Replete magnesium == will help K correct! - Replete to 3.5 mmol for MOST patients ○ Target ≥4 mmol if higher risk for arrhythmia, significant insulin use, diuretics, etc - Enteral route preferred (Potassium Chloride) ○ 10 mEq will raise by 0.1 (if renal function is normal!) ○ 10 mEq will raise by ~0.2 (if renal function - IV can be adjunct or sole therapy if NPO/unable to swallow/profound shock/severe (<2.5) ○ 10 mEq/hr for PIV (can run multiple in different IVs – may burn!) ○ 20 mEq/hr for central line			

Hyperkalemia

<table>
<tr><th colspan="6">Hyperkalemia Causes</th></tr>
<tr><td colspan="2">Decreased Renal K Excretion</td><td>Excess Endogneous K (Cellular Shift)</td><td>Excess Exogenous K (Intake)</td><td colspan="2">Pseudohyperkalemia</td></tr>
<tr><td>Normal Aldosterone
CKD (GFR<10)
Decr EABV
Chronic Urinary Obstruction
Sickle Cell, Amyloid, AIN
Meds: Bactrim</td><td>Decreased Aldosterone
Primary AI
Adrenal enzyme deficit
Hypoaldo states
RTA Type 4
Drugs: NSAID, Cyclosporine, Heparin, ACEi/ARB, ENaC</td><td>Tissue necrosis
TLS, Rhabdo
Hemolysis
Insulin deficiency
Hyperosmolality
Rigorous exercise
Drugs: B2-blockers, Digoxin, Succ</td><td>Diet (potato, avocado, banana)
Salt substitutes
K-enriched food
Cautopyrophagia
Blood transfusion
Drugs: Oral/IV K repletion, IV PCN</td><td colspan="2">Lab error
Hemolysis
Leukocytosis
Thrombocytosis
Phlebotomy technique (trauma, repeated fist)
Hereditary spherocytosis
Drawing from K-infusion IV</td></tr>
<tr><td>Signs</td><td colspan="3">Mild Hyperkalemia (5 – 5.5)
- Typically asymptomatic without EKG changes
- The most common EKG change in hyperK is NO CHANGE

Moderate Hyperkalemia (5.5-6.4)
- Often asx, but may see muscle weakness/paralysis/nausea

Severe Hyperkalemia (6.5+)
- More likely symptomatic – treat regardless!!
- EKG changes (do not reliably correlate to K level)
- Wide QRS, Prolonged PR, flat P waves, peaked T-waves
- Sinus brady, sinus arrest, sine waves, VT, VF, PEA arrest
- Chronic HD patients will tolerate higher levels</td><td colspan="2"> 
</td></tr>
<tr><td>Treatment</td><td colspan="5">Confirm the K and obtain EKG and other lab eval, but if severe or symptomatic, begin treatment
C (i.e., see) A BIG K Level Decrease
→ Calcium Gluconate (for EKG changes), Albuterol (uncommon), Bicarbonate, Insulin + Glucose, K-binders, Loop Diuretic, Dialysis
- Obtain interval labwork after first treatment
- Handbook has good guide! Moderate (begin with I/G + Lokelma), Severe (I/G + Lokelma, possible early HD)
-
- For severe or emergent hyperkalemia, admit to ICU for treatment!

<table>
<tr><th>Mechanism</th><th>Medication/ Intervention</th><th>Administration</th><th>Dosage</th></tr>
<tr><td rowspan="2">Stabilize myocardium</td><td>Calcium gluconate</td><td>Intravenous (peripheral)</td><td>1.5-3 g over 2-5 min</td></tr>
<tr><td>Calcium chloride</td><td>Intravenous (central)</td><td>0.5 – 1 g over 2-5 min</td></tr>
<tr><td rowspan="3">Drive potassium intracellularly</td><td>Albuterol</td><td>Inhalation</td><td>10-20 mg over 10 min</td></tr>
<tr><td>Sodium bicarbonate</td><td>Intravenous</td><td>50 mEq (~1 Amp) over 5 min</td></tr>
<tr><td>Insulin (regular) w/ glucose</td><td>Intravenous</td><td>10 units bolus, followed by 25 g (1 Amp D50) over 5 min</td></tr>
<tr><td rowspan="3">Prevent potassium absorption</td><td>Kayexalate</td><td>Oral / Rectal</td><td>15 g Q6H PRN / 30-50 g Q6H PRN</td></tr>
<tr><td>Patiromer</td><td>Oral</td><td>8.4 g daily (Max 25.2 g/day)</td></tr>
<tr><td>Sodium zirconium cyclosilicate</td><td>Oral</td><td>10 g Q8H PRN for up to 48 hours</td></tr>
<tr><td rowspan="2">Remove Potassium</td><td>Furosemide</td><td>Intravenous</td><td>~20-40 mg Q6H PRN</td></tr>
<tr><td>Dialysis</td><td>N/A</td><td></td></tr>
</table>
** Kayexalate has fallen out of favor due to intestinal necrosis fears – this exceedingly rare but nonetheless Lokelma (sodium zirconium cyclosilicate) is preferred (if not constipated or obstructed)</td></tr>
</table>

Resistant Hypertension

	JNC 7	AHA
Definition	BP >140/90 On 3 anti-HTN meds INCLUDING diuretic All drugs at or near max dose	Uncontrolled BP despite 3 meds OR BP controlled but requires 4+ meds
Secondary Causes	Etiology	Test
	OSA (25-50%)	Polysomnogram (PSG / Sleep Study)
	Primary hyperaldosteronism (8-20%)	Aldo/renin ratio
	Renal artery stenosis (5-34%)	Renal artery US, CTA/MRA
	Renal parenchymal disease	-
	Drug or EtOH induced	-
	Thyroid disease	TSH
	Pheochromocytoma	Plasma metanephrines
	Cushing's dz	O/N Dex suppression, Late-nite salivary cortisol, 24-hr Ur cortisol

Hypertensive Emergency

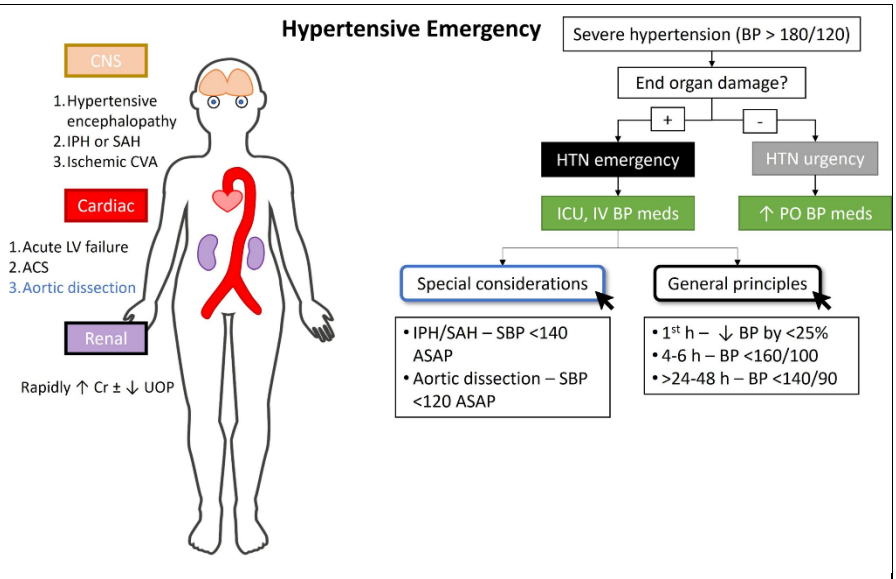
Evaluation:

Looking for Neuro, CV, or Renal emergency

- Labs: BMP, LFT, Troponin, UPT
- Imaging: NCHCT, +/- CTA as indicated

Therapeutics:

- Vasodilators
  - Nicardipine (CCB) – titratable drip, good in Stroke
  - Nitroglycerin, sodium nitroprusside - ADHF
  - Enalaprilat (ACEi)
  - Hydralazine – can cause profound BP drops, rebound tachycardia, also not for ACS!
- Adrenergic Inhibitors
  - Labetalol – good for floor, pregnancy
  - Esmolol – good in Ao dissection
  - Phentolamine - cocaine, +/-pheochromocytoma



Treatment Goals:

- 1st hour (Decrease MAP 10-20% - <180/120)
- Next 23 hrs (Decr MAP another 5-15% - <160/110)
- **Exceptions**: Aortic dissection, eclampsia, pheochromocytoma, acute ischemic stroke (reduce to SBP <140 in first hour; reduce SBP <120 in 20 minutes for dissection; permissive HTN to 220/110 in acute ischemic stroke unless giving tPA)

Nephrotic Syndrome

Diagnosis

Podocyte disruption leading to massive proteinuria

Diagnostic hallmarks = **CAPE**:

- **C**holesterol HIGH
- **A**lbumin LOW (<2.5-3 g/dL)
- **P**rotein excretion in the urine (>3.5g per 24h OR Spot UPCR >3500 mg/g)
- **E**dema (peripheral, periorbital > pulmonary)

Pathophys

Complex, many theories (overfill, underfill, etc). In a nutshell, some damage occurs to glomerulus, triggering:

Urinary protein loss & hypermetabolism → Hypoalbuminemia & Decr oncotic pressure → Renal Na retention → Interstitial edema → hypovolemia

Simultaneous compensatory hepatic protein synthesis → Hyperlipidemia & lipiduria; Hypercoagulability

Etiologies

Minimal Change Disease	Membranous Nephropathy	Focal Segmental Glomerulosclerosis	Diabetic Nephropathy	Renal Amyloidosis	Nephritic Overlap
Most common cause in children. Idiopathic or 2/2 NSAIDs, Hodgkins	2nd most common cause in adults Primary (**PLA2R**) or Secondary (NSAIDs, Gold; HBV, HCV, syphilis), SLE, solid tumors. HIGH clot risk!	Most common cause in adults. Idiopathic or 2ndary to HIV, Sickle Cell Dz, IVDU	Chronic, uncontrolled DM Often comorbid w/ ocular/neural dz	1° (plasma cell dyscrasia) 2° (infxn, inflammation) e.g. MM, RA, TB	Lupus Nephritis, IgA Nephropathy, Membrano-proliferative nephropathy

Evaluation

All patients:

- PMH, medications, PSH, social hx + physical exam
- BMP, LFT, CBC, Coags (AKI = worse prognosis)
- Lipids (Tchol into 300s on avg)
- Urinalysis with micro (proteinuria, fatty casts – Maltese Cross)
- 24hr Urine Protein and/or Spot UPCR
- Renal biopsy (unless etiology evident or PLA2R positive)
- Cancer screenings

Consider:

- PLA2R (~100% SP for 1° MN)
- HIV, Chronic hepatitis
- RPR
- ANA, dsDNA, complement
- SPEP/UPEP
- ESR/CRP
- Talk to Nephro! :)

Management

- **Treat underlying cause!**
- **Hyperlipidemia (ramped up hepatic synthesis 2/2 hypoalbuminemia)**
  - High intensity statin, lifestyle intervention
- **Hypercoagulability (Loss of Protein C/S, ATIII + Hemoconcentration + HLD)**
  - LMWH or warfarin for all low-bleeding risk patients (or intermediate risk if if albumin <2)
- **Infection risk (IgG loss)**
  - Pneumovax!
- **Proteinuria + BP Control**
  - ACEi or ARB x 6-12 mo
  - Refractory: Steroids/CYC, Calcineurin inhibitors, or Rituximab
  - Increased dietary protein intake
  - Proteinuria control == ESKD prevention
- **Edema**
  - TZD or Loop diuretics (TZD may reduce proteinuria)

Neurology

Neuro Exam

1. Assess mental status:
Level of Consciousness: Alert, Drowsy, Obtunded, Stuporous, Comatose
Glasgow Coma Scale (GCS): E4, V5, M6 & Richmond Agitation Sedation Scale (RASS)
Attention, Mood, Orientation, Language, Intellectual Function

2. Assess memory (Recent and Remote-3 objects, corroborated info)

3. Cranial Nerves I-OLFACTORY: Ask pt. to identify a pleasant (not noxious= pain) but common odor? (Each nostril separately test only when a patient complains of difficulty with taste or smell)

4. Cranial Nerves II-OPTIC: Test visual acuity

Notes: -inquire if pt. wears glasses or contacts
-allow patient to hold card at appropriate distance
-test each eye independently in well lit environment

5. Cranial Nerves II-OPTIC: Test visual fields by confrontation, central AND peripheral vision

Notes: -have patient focus on forehead or nose
-test each eye independently- they cover one eye, you don't
-CENTRAL VISION: check each field with finger count (1,2, or 5) in each quadrant
-PERIPHERAL VISION: when first see finger wiggle in outer reaches of each quadrant

6. Perform funduscopic exam of each eye to assess optic nerve head and vessels

7. Test Extraocular Movements

Notes; Cranial Nerve III, IV and VI-: Test ocular motion and convergence by asking pt. to follow finger or pen light with eyes in H pattern without moving their head?

8. CN II and III--Test Direct AND Consensual pupillary reaction to light?

Notes: Shine light in one eye and look at reaction in that eye and the other one

9. Cranial Nerve V-TRIGEMINAL: Test for bilateral light touch (gentle finger or cotton ball tap in discrete location, not rub) and/or temp (tuning fork is cold, warm one side)

Notes: MUST DO ALL THREE: ophthalmic (above eyes), maxillary (on cheeks), mandibular (on sides of chin) divisions

10. Cranial Nerve VII_FACIAL: Test facial muscle strength: (at least 2) Tight eye closure, wrinkle forehead/grimace/show teeth/smile

11. Cranial Nerve VIII-VESTIBULOCOCHLEAR: Test gross hearing acuity in each ear independently? Whisper (while masking the other ear) or finger rub

12. Cranial Nerve IX, X-GLOSSOPHARYNGEAL,VAGUS: Ask the patient to open mouth & say Aah and inspect position of uvula & soft palate or swallow

13. Cranial Nerve XI-SPINAL ACCESSORY: Have patient shrug shoulders and turn head against resistance

14. Cranial Nerve XII-HYPOGLOSSAL: Have patient stick tongue out and push tongue inside check on each side while examiner tests strength by pushing from outside cheek

15. MOTOR EXAMINATION: Bilaterally assess for normal bulk and test muscle tone in arms and legs

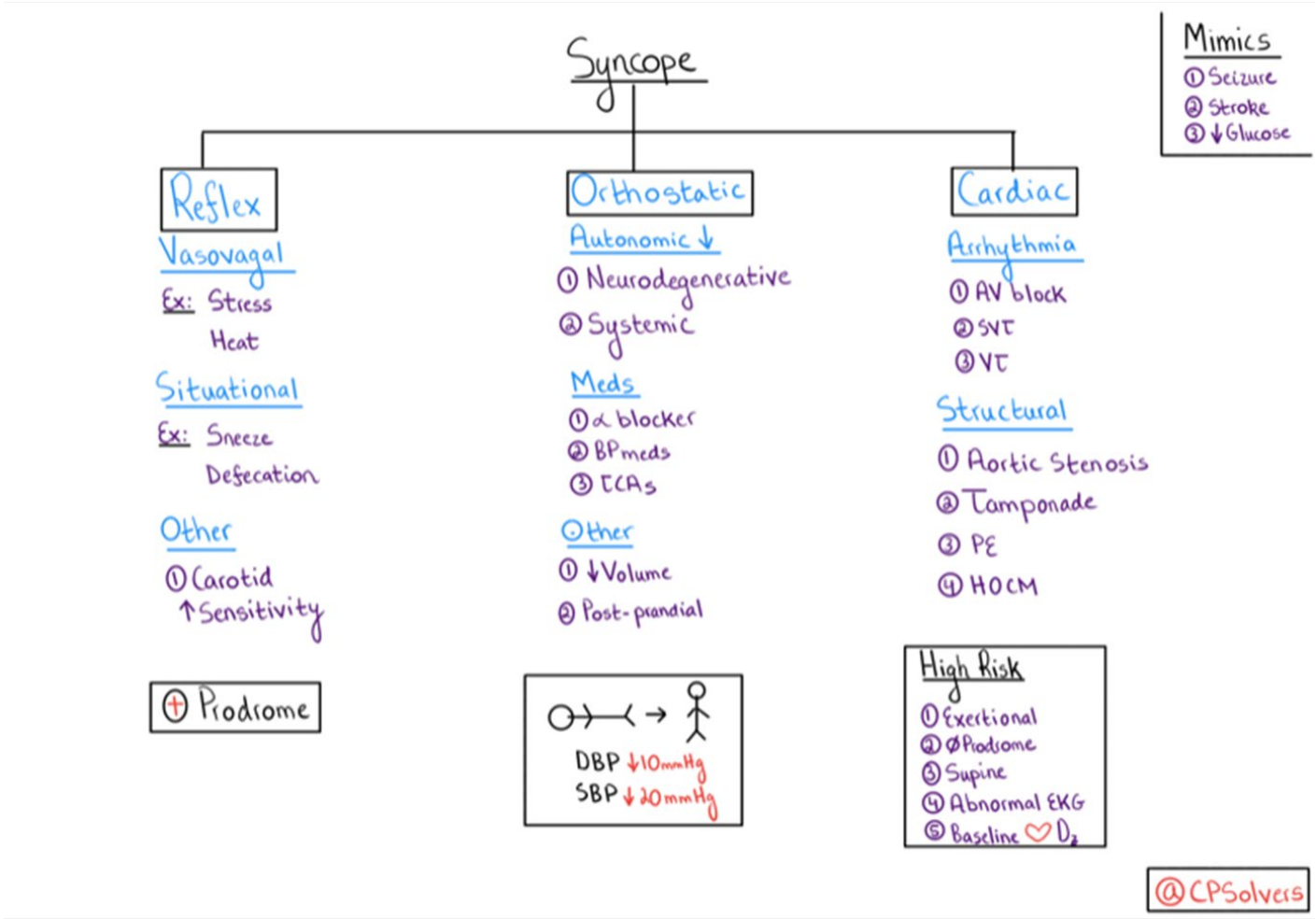
16. STRENGTH EXAMINATION: compare (R to L) shoulder abduction/adduction, wrist extension and flexion, hip flexion and extension, foot dorsiflexion and plantar flexion
17. SENSORY: Test and contralaterally compare sharp vs. dull over the dorsum of the foot and hand WITH PATIENT'S EYES CLOSED Notes: Examiner should provide standard prior to testing with patient's eyes open, to insure pt can discern sharp vs. dull
18. SENSORY: Test and contralaterally compare vibration over great toes WITH PATIENT'S EYES CLOSED
19. SENSORY: Test joint position sense in big toe WITH PATIENT'S EYES CLOSED *Notes:* -grip toe at most distal joint laterally (not on top and bottom); avoid touching nearby digits -ask pt. to identify position (up or down)
20. DEEP TENDON REFLEXES: Biceps, triceps, and brachioradialis, bilaterally
21. DEEP TENDON REFLEXES: Knee jerk/Patellar and Ankle jerk/Achilles, bilaterally
22. DEEP TENDON REFLEXES: Plantar response (Babinski)
23. COORDINATION: Test finger-to-nose and heel-to-shin
24. COORDINATION: Test for dysdiadokinesia -rapid alternating movements in UPPER extremities (one hand turning back and front in the other hand) AND LOWER (tap heel on floor repetitively)
25. GAIT AND STATION: Assess for Romberg (toppling when feet are together and eyes closed) *Notes:* -Stayed close to patient to prevent injury
26. GAIT AND STATION: Observe gait (stride length, base (distance between feet), arm swing, turning (lead with head and shoulders), posture Observe gait: toe-walk, heel-walk, tandem-walk (heel to toe tells balance/coord)

Altered Mental Status

MIST				
Metabolic	**I**nfection	**S**tructural	**T**oxin	Mimic/Distractor
Hypo/hypernatremia Uremia/Renal failure Hypo/hyperglycemia <u>Organ dysfunction</u> Renal or Liver failure Hypo/Hyperthyroid Hypercarbic or Hypoxic Resp Failure <u>Other</u> Thiamine or B12 deficiency Urinary retention Constipation	<u>Extra-CNS</u> Pneumonia UTI ~Sepsis in general <u>CNS</u> Encephalitis	Subdural Hemorrhage	Anti-cholinergic, BZD, or Opiate toxicity Baclofen W/d Ketamine tox or w/d	Aphasia Dysarthria <u>Rx>Dx:</u> Can give D50 or naloxone empirically. Intubate if not protecting airway.

MIST-Negative				
Dementia		Psychiatric	Strategic Stroke	Seizure
<u>Rapidly Progressive</u> Prion dz (Kuru, CJD) Autoimmune Encephalitis Vasculitis	<u>Degenerative w/wo delirium</u> Alzheimers Vascular dementia	Catatonia	Brainstem, Thalamus, Non-dominant Parietal Lobe Frontal Lobe Superior Sagittal sinus	Non-convulsive status

Syncope

Dx: Transient LOC from decreased cerebral perfusion		
		
Eval Review history, meds, risk factors, perform CV and neuro exam, EKG Tilt table testing – reserve for recurrent unexplained syncope Carotid massage – if history convincing for carotid hypersensitivity	Review history, meds, risk factors, perform CV and neuro exam, EKG Orthostatic vitals! Laying to standing (HR>30, SBP drop 20, DBP drop 10 = positive) Neuro c/s for dysautonomia	Review history, meds, risk factors, perform CV and neuro exam, EKG Zio patch (inpt telemetry) EP Study TTE Ischemic eval CTPE
Management Education, hydration, adjustments with positional changes Midodrine (IIa) Counter pressure maneuvers (hand squeeze, leg cross, etc)	Reduce medication doses Hydration, compression garments, counterpressure maneuvers	Treat underlying disorder Medication, LHC, PPM, surgery, PE mgmt

Stroke

Definition	Persistent focal CNS deficits from CNS vascular injury/cause, including infarction or bleeding
Risk Factors	DM, cardiac disease, HLD, HTN, Obesity, Smoking/EtOH/drug use, OSA
Subtypes	Ischemic (Thrombotic vs Embolic vs Hypoperfusive) - A-fib/flutter, vegetation, HFrEF; atherosclerosis/HLD, dissection, vasospasm, arrest/shock Hemorrhagic - Intracerebral (ICH) – HTN, exertion/trauma, coagulopathy, stimulant drugs, AVM - Subarachnoid (SAH) – smoking, HTN, heavy EtOH use, PKD, FH of SAH, stimulant drugs
Symptoms	Sudden focal deficits: - facial or extremity weakness/sensory loss; aphasia; ataxia; visual loss (less common vertigo, brainstem symptoms, AMS) Consider mimics: - seizure, migraine, functional, stroke recrudescence, hyper/oglycemia, toxins/drugs
Diagnostics	Code Stroke! → Identify last known well → NIHSS + Neuro Exam → NCHCT + CTA H/N Labs/Other: - POC Glucose, BMP (rule out hypoglycemia) - EKG (a-fib or arrhythmia?) - CBC (Plts), Coags (Bleeding risk)
Initial Treatment	Thrombolytics - (Tenectplase/TNK or alteplase/TPA) - Within 4.5h from LKW - MUST RULE OUT CONTRAINDICATIONS (Refer to MDCalc) - *Absolute*: acute ICH or SAH, NSGY/TBI/stroke in last 3 mo, BP >185/110, prior ICH, known cerebral AVM or cancer or aneurysm, aortic dissection, active bleeding Endovascular thrombectomy - Within 24h from LKW for non-hemorrhagic, large vessel occlusion (LVO), NIHSS 6+
Subsequent Care **(the VA Stroke orderset is solid)**	Admit to telemetry SLP eval or bedside nursing eval MUST happen before patient eats (NPO otherwise) PT/OT eval TTE +/- bubble study (PFO eval) MRI Brain +/- MRA head/neck Lipids, A1c, TSH, HIV High-intensity statin Hypertension management: - Permissive hypertension to <220/120 for first 24hr (if no TPA or thrombectomy) - <140/90 within 24h to 48h post-stroke - Outpatient goal <130/80 Dual antiplatelet therapy (aspirin + clopidogrel OR ticagrelor) - 21 to 90 days VTE chemoprophylaxis, usually within 24hrs HTN, DM, Obesity, OSA, smoking, CVD mgmt. and mood disorder screening (PCM f/u)

Neurocognitive disorders

Alzheimer's	Frontotemporal Dementia	Dementia with Lewy Bodies	Vascular Dementia
Pathophysiology			
Abnormal amyloid plaque and tau tangle deposition	Tau and TDP-43 protein accumulation within frontal and temporal lobes	Alpha-synuclein protein (Lewy Bodies) deposition	Recurrent cerebrovascular injury, clot, ischemia; prior stroke / TIA; comorbid CV disease, smoking
Symptoms			
Impaired memory as early sx; minimal motor impact until moderate severity	Behavioral-variant type (uninhibited, compulsive, inappropriate, and criminal behaviors with poor insight – slight Male predominant); Language-variant subtype (word-finding difficulty predominates)	Dementia and motor symptoms develop within 1-2 years of each other. REM sleep behavior disorder, Visual hallucinations, sever delusions, falls, orthostatic hypotension, Parkinsonism	Step-wise decrement in function/cognition, early gait impairment & mood changes; a/w emotional lability, apathy, severe cognitive slowing, pronounced gait disorder and repeated falls much earlier than cognitive decline level
Evaluation			
Rule out Mimics! Particularly depression (AKA pseudodementia), using the GDS-15 screener → tx and follow-up; delirium also Obtain thorough history: memory/cognitive domain decline (getting lost, word-finding difficulty, etc), substance use, ADL/IADL completion, work/education history, other sxs (falls, incontinence, AH/VH, etc) Neuropsych screening: SLUMS or MOCA → may refer for formal Neuropsych evaluation Consider sleep study if DLB is suspected Labs: B12, TSH; consider folate, HIV, RPR, iCal if clinically indicated Imaging: NCHCT (brief rule out for SDH, hemorrhage, etc); routine MRI brain			
Treatments			
Targeting cognition Acetylcholinesterase inhibitors (donepezil, rivastigmine, galantamine) - Reasonable to trial for most pts - Benefit is modest - Risk of N/V, loss of appetite, diarrhea, bradycardia, withdrawal syndromes Memantine (targets NMDA) - Added on to cholinesterase inhibitor with moderate Alzheimers OR for those with ACHEi intolerance - No benefit in FTD or vascular subtypes Lecanemab (Leqembi) - mAB targeting amyloid plaque clearance - Expensive		**Neuropsychiatric symptom relief** - Agitation, aggression, delusions, AH/VH, paranoia Non-pharm: Assess safety, create structured routines, reassurance, sleep-wake cycle maintenance, pain management Pharm: atypical antipsychotics (quetiapine, olanzapine) with attempt to taper within 4 months of starting - Caution with QTc prolongation, DLB (high risk for adverse rxn) SSRIs may have some benefit for behavioral-type FTD	

Seizure

Seizure Classifications

Provoked
- MIST (Metabolic, Infectious, Structural, Toxin/Med)

Unprovoked
- Epilepsy (≥2 unprovoked sz >24 hr apart OR 1 unprovoked seizure with high risk of recurrence**)
- Seizure of uncertain significance (doesn't meet above)

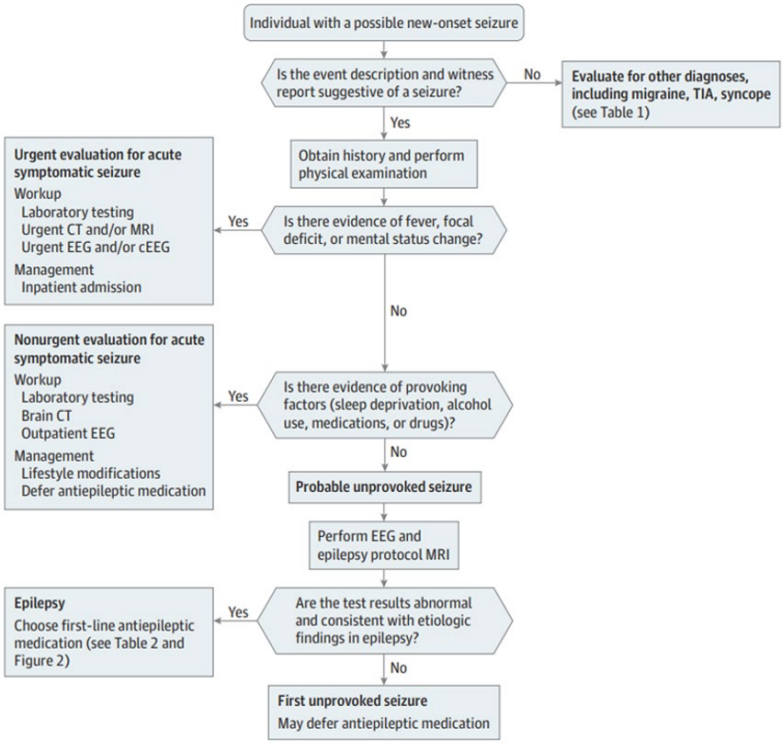
Focal (Origin in single cerebral hemisphere)
- Aware v Impaired; Motor v non-motor

Generalized (origin in bilateral brain network, impaired)
- Motor v non-motor, ±status epilepticus (≥5 min sz activity or recurrent seizure w/o return to baseline)

**High-Risk features
- Occurs ≥1 mo after stroke
- Brain lesion present
- Epileptiform EEG spikes

Rule out Mimics:
- Syncope, Migraines (especially basilar), Psychogenic non-epiletic seizures (PNES), Focal dystonia, Spasticity

New Onset Seizure Evaluation

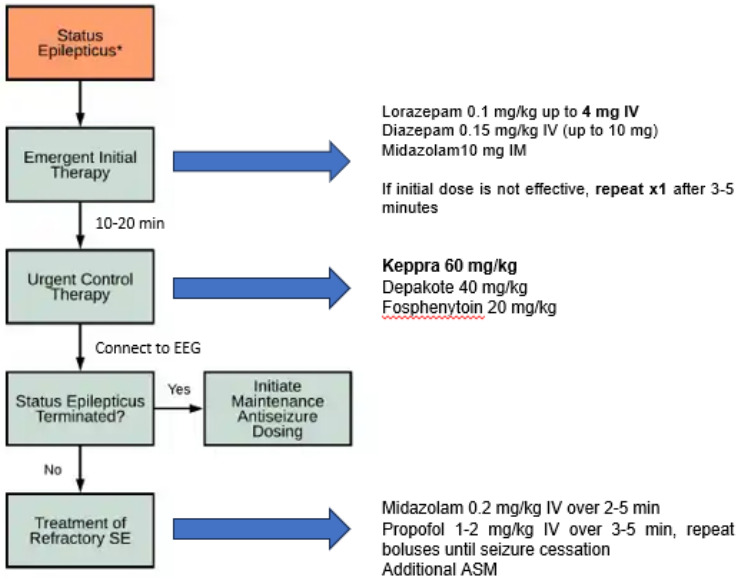


Differentiating Seizure Mimics

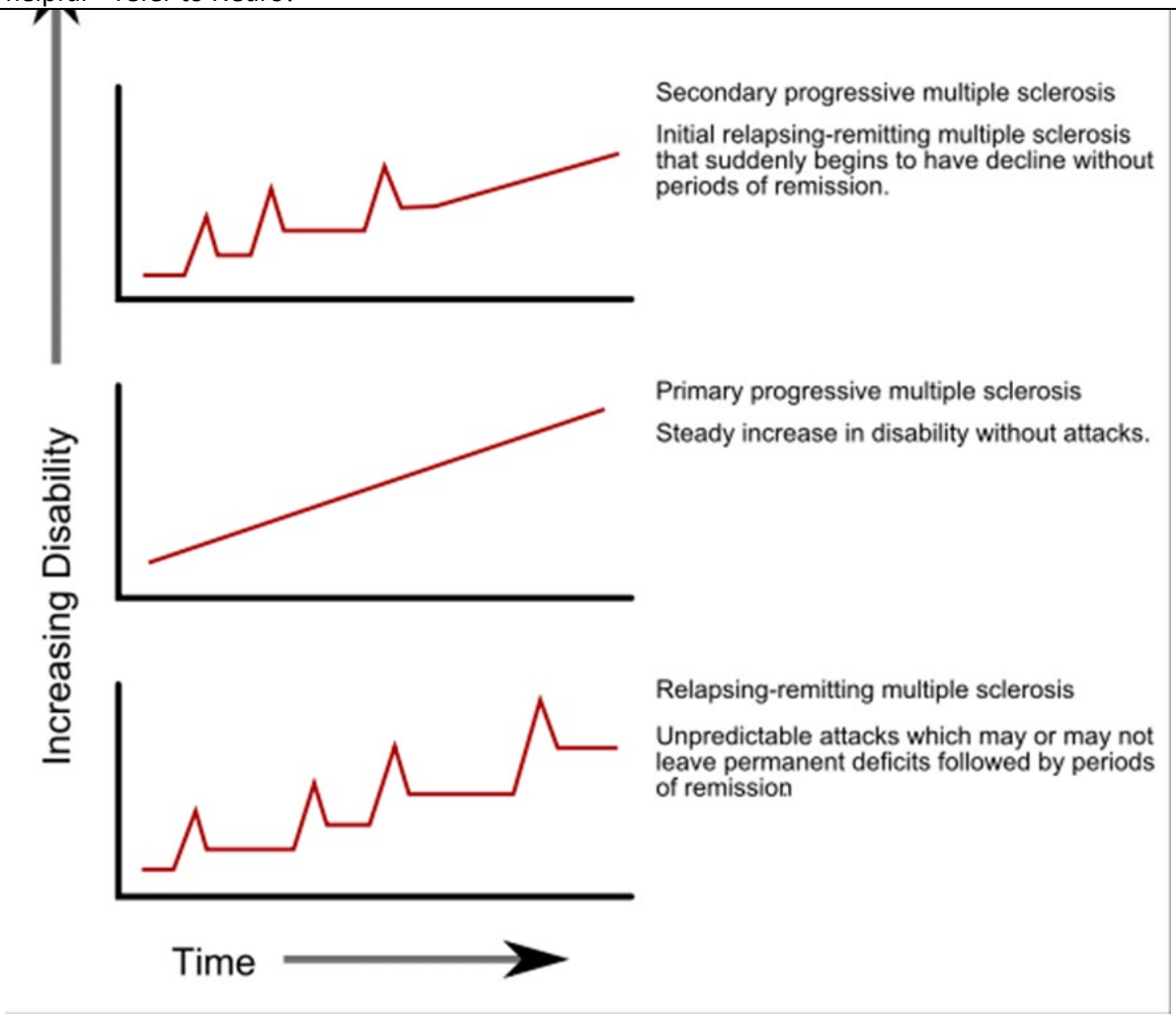
Characteristics	Seizure	Syncope	Migraine Aura
Clinical Features	Sudden LOC Shaking, myoclonic jerking, Tongue biting, Incontinence	Abrupt, transient LOC, Loss of postural tone, spon recovery	Visual/sensory symptoms, evolve over ≥ 5mins, followed by HA
Duration	< 5 mins	1-2 minutes	Up to 1 hour
Post Event Confusion	Yes, Postictal	None	None
Recall of Event	Complete amnesia	Can recall prodrome	Complete
Diagnostic Tools	MRI, EEG	EKG, orthostatic VS	Personal/Fam hx of migraine

Seizure Management

Evaluation: EEG, NCHCT then MRI, refer all seizures to Neurology!
Treatment: Treat provoking factors if provoked, give ASD if provoking factors not easily addressed, otherwise:



Multiple Sclerosis

Diagnosis	Dissemination of demyelinating neurologic symptoms in time and space - Optic Neuritis (classic herald) – painful eye movement, unilateral visual defect, afferent pupillary defect - Lhermitte sign – electric shock sensation - Upper motor neuron signs (spasticity, hyperreflexia, Babinski) - Ataxia or gait disturbance - Sensory changes - Fatigue - Age of onset 15-50 yo, particularly in females MRI of brain, C- and T-spines with periventricular white matter changes Note: CSF testing is NOT required – but may show oligoclonal banding; autoantibody testing may be helpful – refer to Neuro!
Disease Phenotypes	 Activity = clinical relapses or MRI evidence of new/enlarging lesions Progression = gradual accumulation of neurologic deficits independent of relapses
Pseudorelapse	Uhthoff Phenomenon – aggravation of chronic symptoms with increased body temperature (infection/illness, exercise, etc) – may present like actual relapse – treat the underlying cause!

Lambert-Eaton Myasthenic Syndrome

Definition	Autoimmune neuromyopathic disorder with Ab against voltage-gated calcium channels, blocking Ach release and thus preventing muscular contraction in response to neural stimulus -> weakness
Syndrome	Epi: Rare! More often middle-aged adults (earlier in those without cancer) Similar presentation to MG, but weakness that improves with exercise Other sxs: swallow dysfunction, ptosis, hyporeflexia, dry mouth
Evaluation	EMG with augmented motor response to rapid repetitive stimulation Positive serum VGCC Ab (90% of patients) – you will likely get Anti-AchR Ab as well to r/o MG Routine + symptom-driven malignancy work-up
Treatment	**Identify and treat underlying malignancy!** If not paraneoplastic → immunosuppressants, IVIG, or plasmapheresis Amifampridine (symptomatic relief)

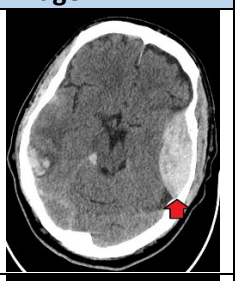
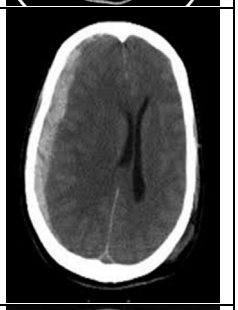
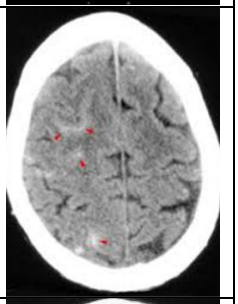
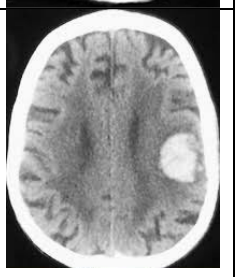
Difference between Lambert Eaton syndrome and Myasthenia gravis

Myasthenia gravis	Lambert Eaton syndrome
Antibody against AchR antibody	Antibody against voltage gated calcium channel
Associated with Thymic tumor	Associated with Small cell lung cancer
Weakness worsen on prolonged exercise	Weakness improves on prolonged exercise
Normal Deep tendon reflex	Decreased or absent deep tendon reflex
Autonomic dysfunction is absent	Autonomic dysfunction is present
On repeated nerve stimulation, there is decremental response	On repeated nerve stimulation, there is incremental response

Acute Spinal Cord Injuries

Spinal Cord Injuries	
Cervical (50%) > Thoracic (35%) > Lumbar (15%) Most commonly from trauma (GLF, MVA, etc) Deaths from SCI usually from: cardiopulmonary failure (C3-C5) vs Paradoxical breathing (like flail chest but 2/2 loss of innervation to intercostals)	
Neurogenic Shock	**Spinal Shock**
Distributive shock sub-type - Loss of sympathetic tone (unopposed parasympathetic) == hoTN + bradycardia May follow injury to T6 or above Onset can be immediate, will resolve over hours to days	Transient, reversible depression of spinal cord function below the level of injury Areflexia, loss of muscle tone below injured level Can occur after injury to ANY level of the spinal cord Onset can be immediate, will resolve over weeks to months
Management	**Management**
Rule out / treat other causes of shock Volume resuscitation and/or Vasopressors Avoid bradycardia-inducing agents (phenylephrine, precedex, propofol even) Monitor for vagal hyperactivity (give atropine)	Address any co-morbid Neurogenic shock, trauma, etc Will follow four phases (Day 0-1, 1-3, Week 1-4, Month 1-12) Reflexes will gradually return with PT During last phase, notable spasticitiy, hyperreflexia, muscle tonicity, and autonomic dysreflexia may occur (Tx PRN)
AUTONOMIC DYSREFLEXIA	
Severe sympathetic response to a **noxious stimulus** Loss of control over sympathetic NS (usually high SCI above T6) Often **weeks to months after initial SCI** Can lead to real, true hypertensive emergencies!!	1. Remove the irritant (bowel, bladder, wound, bandage, eg) 2. Put pt in reverse Trendelenburg 3. Short-acting vasodilator (nitro paste, CCB, hydral, alpha-1) 4. Pain control

Intracranial Hemorrhage

Type	Cause	Treatment	Image
Epidural Hematoma	Middle meningeal artery rupture Head trauma near pterion with brief LOC. "Lucid interval" then rapid deterioration	Sz ppx if GCS<=10 Surgery if: - Volume >30 cc or >15 mm thick - >5 mm midline shift - GCS <9 - Anisocoria	
Subdural Hematoma	Bridging vein rupture Trauma, intracranial hypotension (LP, HD, epidural anesthesia), Structural (post-surgical, cerebral AVM, tumor) Acute, subacute, chronic!	Sz ppx if GCS<=10 <u>Surgery if:</u> - Width > 10 mm, - >5 mm midline shift - GCS <9 or neuro deterioration Chronic can be drained, too.	
Subarachnoid Hemorrhage "Thunderclap HA" Reduced LOC Seizure Photophobia Nuchal rigidity	<u>Aneurysmal</u>: Berry aneurysm rupture - Fisher grade 1-4 based on thickness and intraventricular hemorrhage - Grade I – V surgical risk based on sx severity <u>Trauma:</u> blunt / penetrating	No seizure prophylaxis Maintain normovolemia, Hgb >9, O2>95%, optimal ICP (reduce pain and strain from N/V or BM) Permissive HTN (SBP <160) Nimodipine x21d (vasospasm ppx) **Early clip vs coil**	
Intraparenchymal Hemorrhage	<u>Primary</u>: **HTN, cerebral amyloid angiopathy** <u>Secondary</u>: coagulopathy, AVF, cavernous malformation, tumor, aneurysm, cerebral venous thrombosis, moyamoya, vasculitis, hemorrhagic conversion	ICH Score for risk stratification Specific tx related to etiology (reverse AC, control SBP <140, etc) <u>Surgery if:</u> - Vol. >20 cc or >5 mm midline shift - Cisternal compression - Neuro deterioration - Refractory intracranial HTN	

Subarachnoid Hemorrhage

Pathophys	Ruptured saccular (berry) aneurysm (Most common), Trauma, Intracranial arterial dissection, mycotic aneurysm rupture (less common), Reversible cerebral vasoconstriction syndrome, dural sinus thrombosis, AVM, CAA (rare)
Symptoms	Thunderclap HA, confusion, somnolence, nuchal rigidity, pupillary dilation (CN III compression) Signs of increased ICP: Anisocoria, Loss of brainstem reflexes, posturing, papilledema
Diagnostics	STAT NCHCT (highest sensitivity within 6 hours of symptom onset) LP if suspicion high but NCHCT neg (classically: xanthochromia, non-clearing RBCs, increased opening pressure) Obtain CTA if both negative (occult lesion) or if one positive (identify symptomatic lesion) Angiography is definitive diagnosis
Gauging Severity	<u>Hunt and Hess Classification (higher grade = increased mortality)</u> - Grade I – mild HA, A&O, minimal nucal rigidity = 30% mortality - Grade II - Nuchal rigidity, mod-sev HA, A&O, no neuro deficits (except CN VII) = 40% mortality - Grade III - Lethargy or Confusion, Mild focal Deficits = 50% mortality - Grade IV - Stuporous, more severe focal deficit = 80% mortality - Grade V - Comatose or severe neuro impairment (posturing) = 90% mortality <u>Modified Fisher Scale (assesses risk of cerebral vasospasm in aneurysmal SAH) – On MDCalc!</u> - Grade 0 – No SAH - Grade I – Only focal or diffuse, thin (<1 mm) SAH = 6-24% risk of vasospasm - Grade II – Grade I + intraventricular hemorrhage (IVH) = 15-33% - Grade III – only focal or diffuse thick (1+ mm) SAH = 33-35% - Grade IV - Grade III + IVH = 34-40%
Treatment	- ICU admission with NSGY and neurology consults, q1h neuro checks o Coil aneurysm as needed o External ventricular drain (EVD) placement (ICP monitoring & decompression) - Blood Pressure control (balancing risks of rebleeding from high BP vs ischemic/infarction from low BP) o Goal SBP <160 (or premorbid SBP) or MAP <110 using titratable IV med like cardene gtt o AVOID nitroglycerin or nitroprusside due to cerebrovascular dilation increasing ICP - Reverse coagulopathy (DOAC, warfarin, DAPT, eg) - Pain, nausea, and agitation control (vomiting, agitation, etc raise ICP) - Seizure treatment, consider prophylaxis in higher-risk patients, especially with unsecured aneurysms o Consider EEG for nonconvulsive status epilepticus evaluation - Vasospasm prophylaxis (for ALL aneurysmal SAH patients) o Nimodipine 60mg q6h starting within 48 hours of sxs – continue for 21d post-bleed

Pulmonary and Critical Care Medicine

Classes of Shock

	Distributive	Hypovolemic	Cardiogenic	Obstructive
Etiologies	Sepsis (!!!), Neurogenic, Anaphylaxis	Dehydration, Blood loss	HF, MI, Valvulopathy	Tamponade, PE, PTX
CO	↑	↓	↓	↓
SVR	↓	↑	↑	Nl or ↑
SvO2	↑	↓	↓	Nl or ↓
POCUS	Small IVC (<2.1 cm) >50% Respirophasic collapse Hyperdynamic heart	Small IVC (<2.1 cm) >50% collapse Hyperdynamic heart	Plethoric IVC (>2.1 cm) Minimal collapse Hypodynamic heart Pulmonary B-lines	Normal to big IVC Septal bowing (D Sign) McConnell's sign Pericardial effusion Absent lung sliding
Tx	IVF (30 mL/kg crystalloid), Cx and Abx within 1 hr – septic; Epinephrine, antihistamines - anaphylactic	IVF (30 mL/kg crystalloid), titrate to MAP or SBP, trend lactate, pressors; treat underlying process (stop the bleed! Avoid cold/acidemia/coagulopathy in Trauma, give TXA, activate MTP, etc)	Optimize Preload (diuresis or RRT), Inotropy, Mechanical Support (IABP, Impella) Transplant	Treat underlying pathology (pericardiocentesis, chest tube, thrombolysis/ectomy, e.g.)

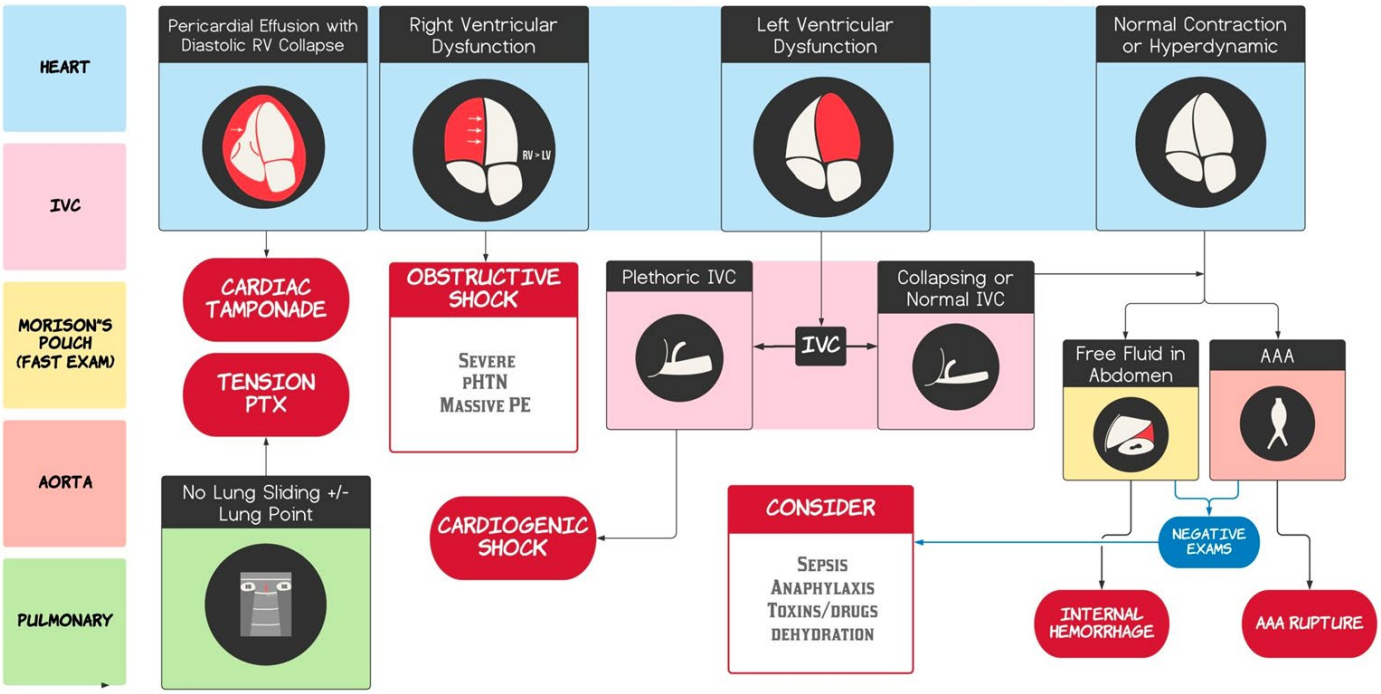
Sepsis & Septic Shock

Definition	Life-threatening organ dysfunction caused by dysregulated host response to infection
Diagnosis	**Requires suspected infectious source** **SIRS** (preferred) = sepsis if meets 2/4 - Temp <36 or >38C, HR>90, WBC <4 or >12 (or >10% bands), RR >20 (or PaCO2 <32) **qSOFA** (less sensitive, more specific/worse prognosis) = sepsis if meets 2/3 - GCS <15 (AMS), RR ≥22, SBP ≤100

Management	**Golden Hour Bundle**
	- IVF: 30 mL/kg crystalloid (LR>NS) within 3 hours
	  ○ Lactate-guided resuscitation helpful (can also use BP, POCUS, cap refill, etc to monitor)
	- Evaluation: Identify source, causative organism, and look for end-organ dysfunction
	  ○ BCx (before abx), CXR, UA/UCx, Lactate (>4 = severe), skin exam, BMP, LFT
	  ○ Cross-sectional imaging, TTE, LP, etc pursuant to clinical picture
	  ○ MUST obtain source control!! (e.g. surgery for nec/fasc, drainage of empyema, etc)
	- Antibiotics: Administer broad-spectrum coverage within 1 hour!
	  ○ Give GNR coverage FIRST (zosyn / cefepime >> vanc)
	- Vasopressors: For septic shock refractory to initial IVF resuscitation (can administer via PIV for brief period for all EXCEPT vasopressin due to skin necrosis without antidote)
	  ○ Norepinephrine (first line) – start here then adjust pressors per patient's needs/comorbidities
	  ○ Vasopressin (second-line) – non-titratable (start at 0.03-0.04 unit/min)
	  ○ Stress-dose steroids (e.g. hydrocortisone IV) - reasonable after second pressor
	  ○ Epinephrine (third) – may be better iso significant cardiac dysfunction

POCUS for Shock

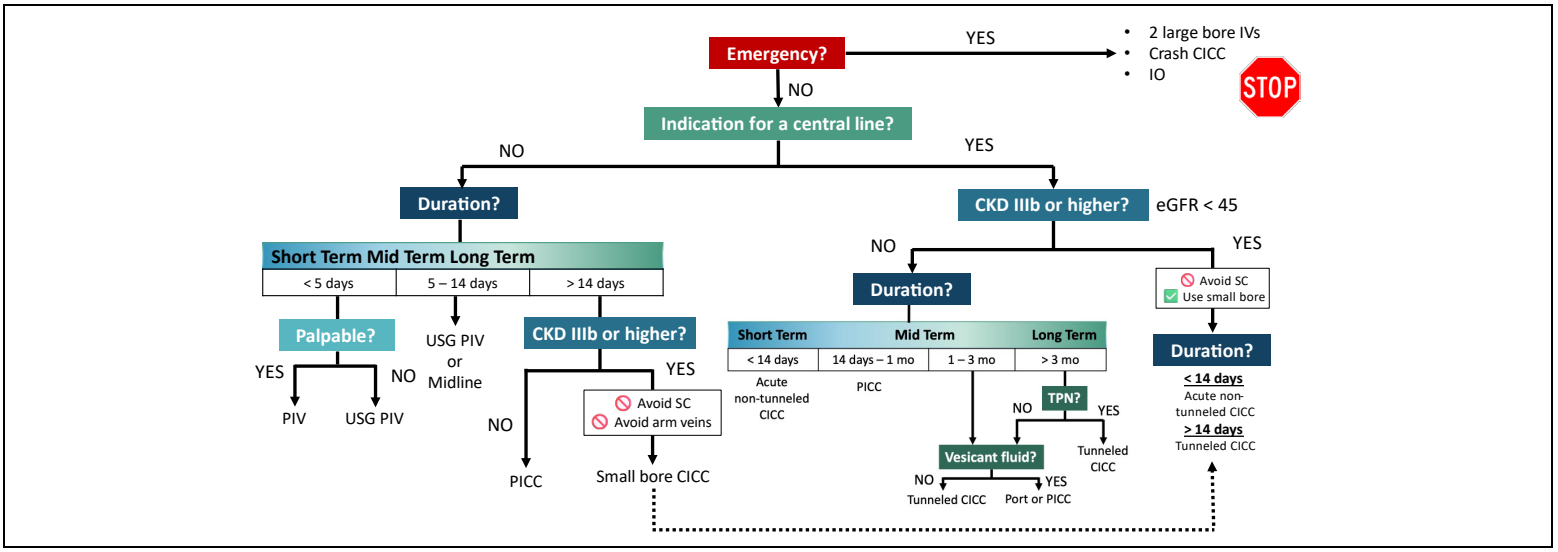
Classic Approaches: RUSH & eFAST



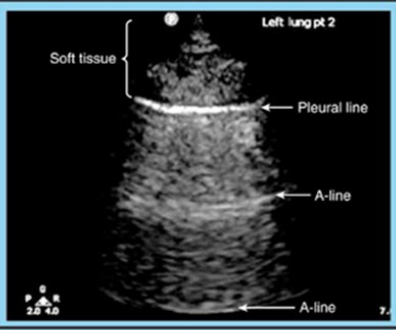
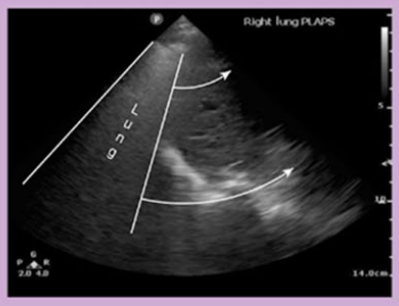
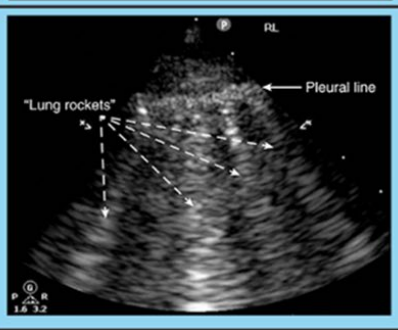
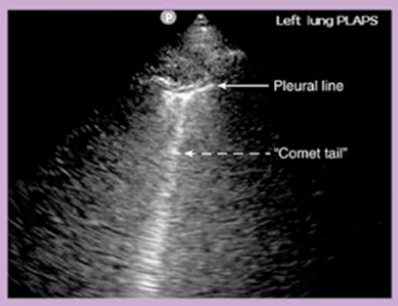
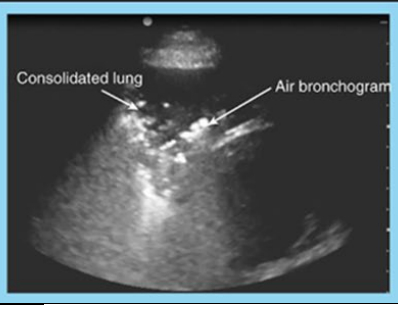
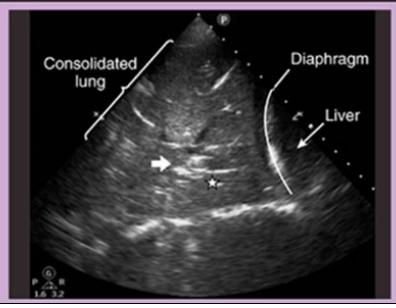
Nancy Maaty's approach (follow the patient's history!)

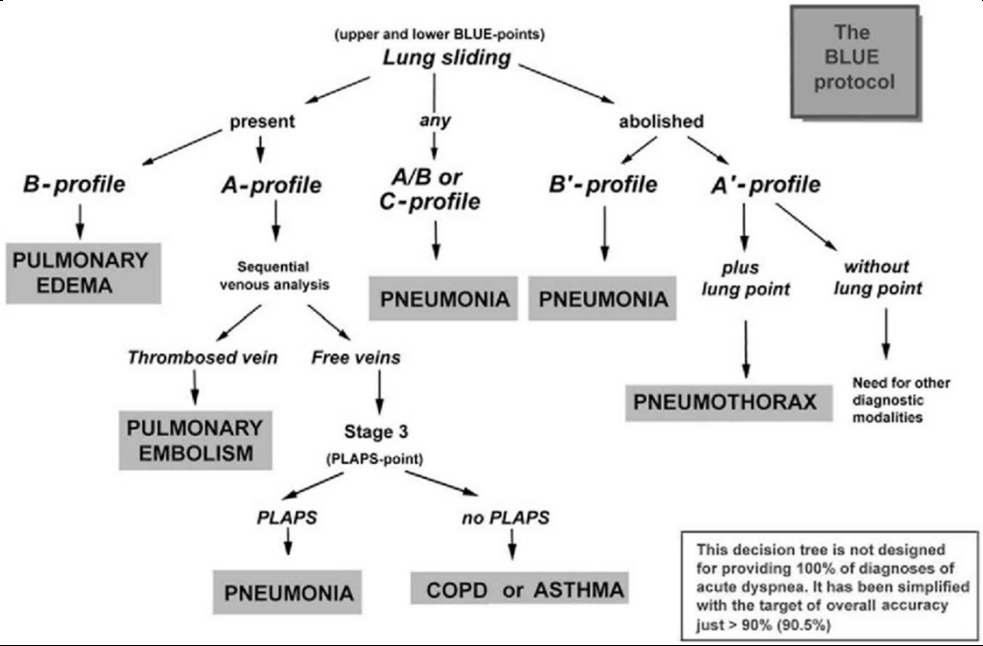
- Rule-Out
  - **Tamponade**
  - **Acute PE**
  - **PTX**
- PLAX
  - LV Eval – EPSS, FS
    - **SAM** vs **LV failure**
  - RV Eval – RVOT Diameter – **PE**
    - RV inversion - **Tamponade**
  - MV/AV color doppler r/o **severe regurg**
- PSAX
  - LV Eval – FS
    - PM kissing - **Hypovolemia**
  - RV - Septal Behavior
- A4C
  - LV – MAPSE
    - → A5C → LVOT VTI
      - PLR → Fluid Responsive?
  - RV – TAPSE
- SC
  - RV – Free Wall thickness
  - IVC view
    - **Tamponade**
- Lung US – r/o **PTX**
- Vascular
  - Run down Aorta, r/o **Dissection**
  - LE 2 point compression, eval for **DVT**

Choosing the Right Line



Pulmonary POCUS

BLUE Exam Points	 Point 1	 Point 2	 Point 3	 Point 4	
A profiles					**A profile** = A-lines + sliding ■ **Normal** ■ If symptomatic, consider: ■ Pulmonry embolism ■ COPD, asthma ■ Nonpulmonary conditions **A' profile** = A-lines + no sliding ■ **Pneumothorax** ■ Pleurodesis (*chemical, infection/inflammatory, fibrosis*) ■ Lung volume loss (*complete atelectasis, mainstem intubation, mucous plug, pneumonectomy*)
B profiles					**B profile** = Bilateral B-lines + sliding ■ **Pulmonary edema** ■ ARDS (*diffuse*) **A/B profile** = Unilateral B-lines + sliding ■ Pneumonia (*mild*) ■ ARDS (*focal*) ■ Scarring **B' profile** = Bilateral B-lines + no sliding ■ Pneumonia (*severe*)
C profile					**C profile** = Consolidation pattern ■ **Pneumonia** ■ Atelectasis ■ Resorptive ■ Compressive



Dyspnea Framework

Bad Heart	Bad Lungs	Bad Blood Between Them
Pericardial effusion **ACS** **Valvular disease** **CHF**	**Pleural effusion** **Parenchymal process (PNA, ARDS)** **Asthma or COPD** **Vocal cord spasm or tracheal obstruction**	**PE** **Acidosis**

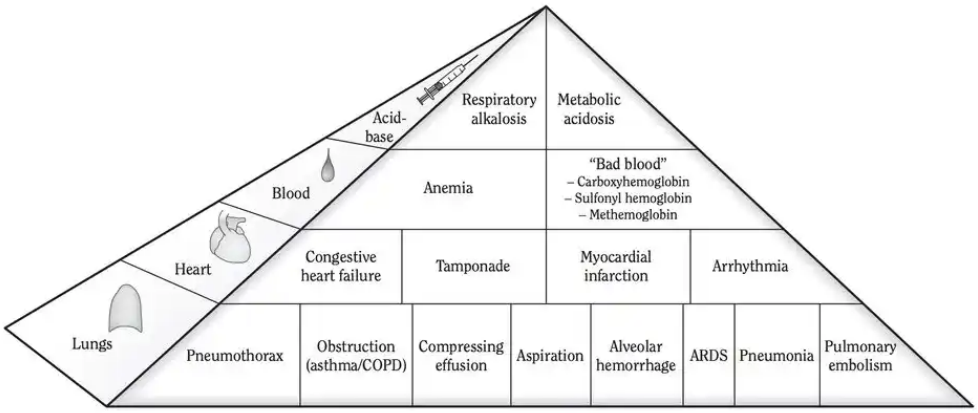
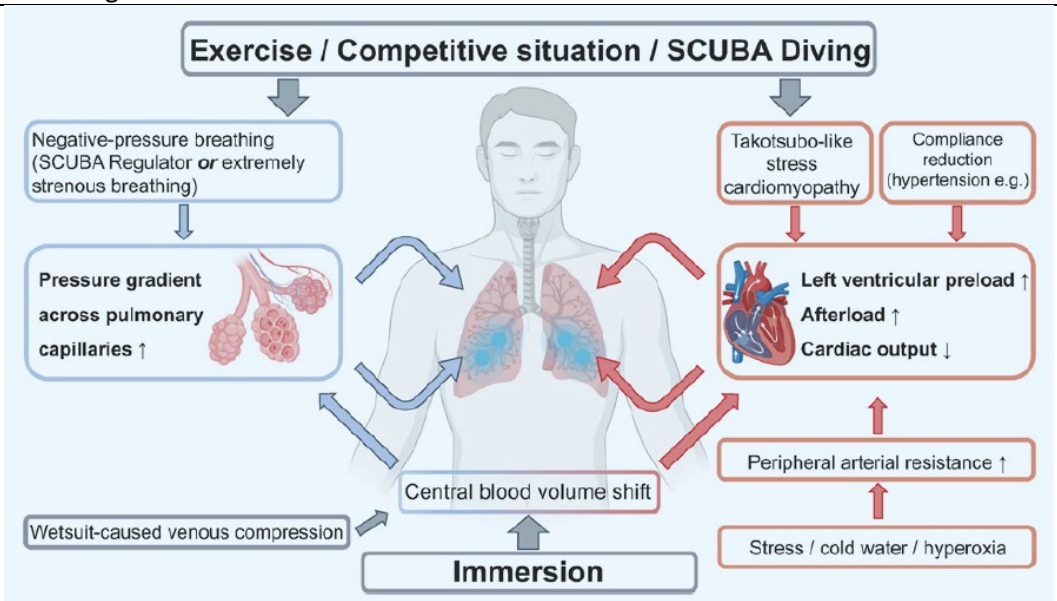


Figure w-3 The dyspnea pyramid. ARDS = acute respiratory distress syndrome; COPD = chronic obstructive pulmonary disease.

Swimming-Induced Pulmonary Edema (SIPE)

Syndrome	Dyspnea and cough after water immersion
Pathophys	
Risk Factors	Open-water swimming (triathlon, SCUBA, military special ops) – prevalence ~1-2% of triathletes and SF Personal: History of SIPE!! Age>50, F>M, Underlying cardiac history Environmental: Cold water (any depth), intense exertion, tight wetsuit, overhydration, poor warm-up
Clinical Findings	Rhonchi / rales on exam; eval for concomitant exercise-induced bronchospasm Pulmonary edema on CXR and/or POCUS
Treatment	Removal from cold/wet environment (incl wetsuit removal, re-warming). Generally good prognosis. Incentive Spirometry. Maintain SpO2 >92%; consider NIPPV.

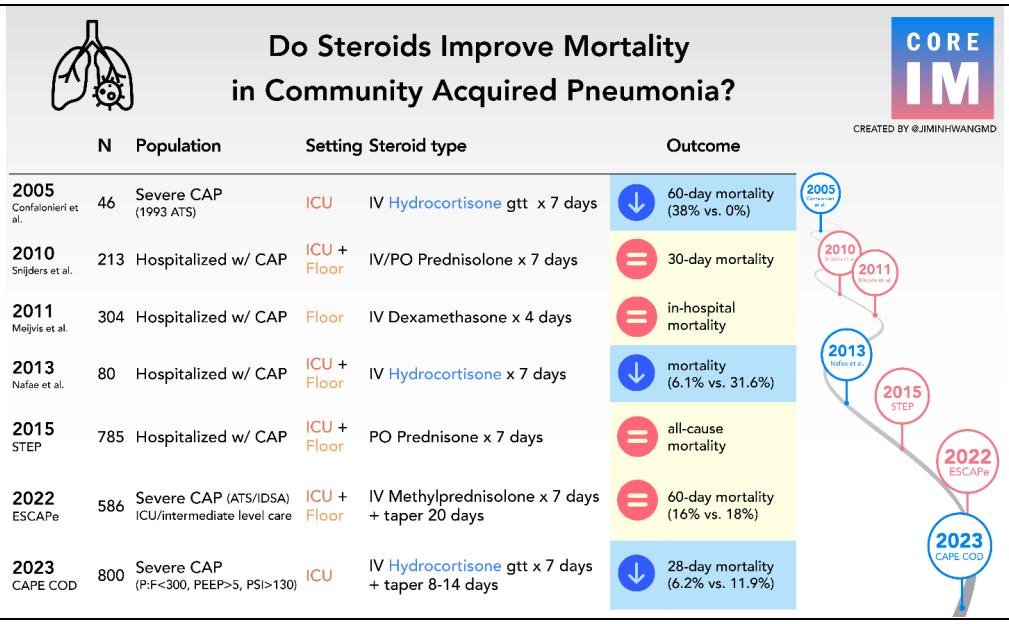
Community-Acquired Pneumonia

Syndrome	New lung infiltrate plus clinical evidence of infection (fever, purulent sputum, leukocytosis, or hypoxia) not acquired in the hospital setting
Risk stratification (on MDCalc!)	Pneumonia Severity Index (PSI) – more factors, harder to use readily (need ABG) CURB-65 – fewer factors, easier to use readily **C**onfusion present? B**U**N >19 **R**R >= 30 S**B**P <90 or DBP <= 60 **65**+ yo old
Bug	Lobar? Think Strep pneumo Interstitial? Think viral or atypicals (legionella, mycoplasma, Chlamydophila) Superinfection? Clang MRSA Less likely – Fungal (PJP, aspergillus, histo/blasto), Parasite (strongy, toxo)
Drug	Outpatient CAP (Healthy Adult) **azithro (Zpak) ineffective at WR due to resistance - Lobar = amoxicillin 1g TID x 5d - Interstitial = doxycycline 100 mg BID x5d Outpatient CAP (Unhealthy AKA our Adults) *think chronic lung/heart/liver dz, T2DM, etc - Augmentin 875/125 mg BID x5-7d !!! - Atypicals: Doxycycline 100 mg BID x5-7d - Respiratory FQ (Levoflox/Moxifloxacin) ok but side effects loom large Inpatient CAP - Standard: Ceftriaxone 1g daily IV x5d, Zithromycin 500 mg daily PO x3d - If abscess or empyema: add anaerobe coverage (e.g. Unasyn IV 3g q6h) - *If MRSA risk*: add vancomycin or linezolid (grab a MRSA nares!) - *If Pseudomonal risk*: cefepime IV 2g q8h (or aztreonam if PCN allergy) + Levaquin 750 mg IV q24h

CAPE COD Trial Summary

Clinical Question	Does IV Hydrocortisone reduce mortality in severe CAP?
Population	*Multicenter RCT conducted in France* Inclusion Criteria - Adults with severe, CAP PSI Score >130 - Invasive or NIPPV (PEEP >=5), HFNC on >=50% FiO2 with P/F ratio <300 - NRB with estimated P/F Ratio <300 Exclusion Criteria - Septic shock - Influenza, TB, or fungal PNA
Intervention	800 pts randomized to hydrocortisone vs placebo (Hydrocortisone delivered as 200 mg infusion daily x4 days) At day 5, steroid taper began if breathing spontaneously, P/F>200, SOFA <= initial SOFA, AND expected discharge from ICU by hospital day 14 If not meeting ALL above, dose continued for addl 3d (7d total) then tapered
Comparison	Saline placebo (both groups received standard of care antibiotics)
Outcomes	Reduced mortality by Day 28 (6.2% death in hydrocort arm vs 11.9 placebo, p=0.006) Reduced mortality by Day 90 (9.3% in hydrocort arm vs 14.7% placebo) Reduced intubation rates (HR 0.59, CI 0.40 – 0.86) No significant difference in Hospital-Acquired infections or GI Bleed
Discussion	Drop in mortality possibly related to reduced pressor requirements, reduced intubation due to decreased lung inflammation; safe intervention though hyperglycemia was at times persistent
Takeaway	Consider early steroid initiation in severe CAP requiring IMC/PCU/ICU level care without septic shock, but with elevated CRP and hypoxia, c/f lack of response to abx **CONTROVERSIAL, NOT CURRENT IDSA GUIDELINE**

Other studies on steroid use in CAP



In general, meta-analysis shows reduced morbidity but NOT reduced mortality. 2019 IDSA guidelines recommend **against** any steroid use for CAP.

Acute eosinophilic pneumonia

Acute eosinophilic PNA
Etiology: often a hypersensitivity reaction to inhalation (dust, fireworks, medications, tobacco smoke) • AKA things common to ADSM (take up/increase smoking on deployment, dust exposure in CENTCOM) **Diagnosis:** • Acute, febrile illness < 5-7 days (cough, chest pain, myalgias) • Hypoxic resp failure – resulting in intubation • CXR = patchy opacities, ARDS • BAL eosinophilia > 25% • Absence of parasitic, fungal, drugs, or asthma **Treatment:** • Rapid and complete response to corticosteroids

Cavitary Lung Lesions

Broad Differential - CAVITY	**Infectious Differential – THANKS-ER**
Cancer	**T**uberculosis/Non-TB mycobateria
Autoimmune	**H**istoplasmosis (and Coccidio/Blasto)
Vascular	**A**spergillus/Anaerobes/Actinomyces
Infection	**N**ocardia
Trauma	**K**lebsiella/GNRs
Youth/Congenital	**S**taph aureus
	Echinococcus
	Rhodococcus

Pulmonary Emboli

Diagnosis

CTA Chest (CTPE) – direct visualization of pulmonary arterial embolus
- Preferred first line study for stable patients with reasonable renal fxn or ESKD

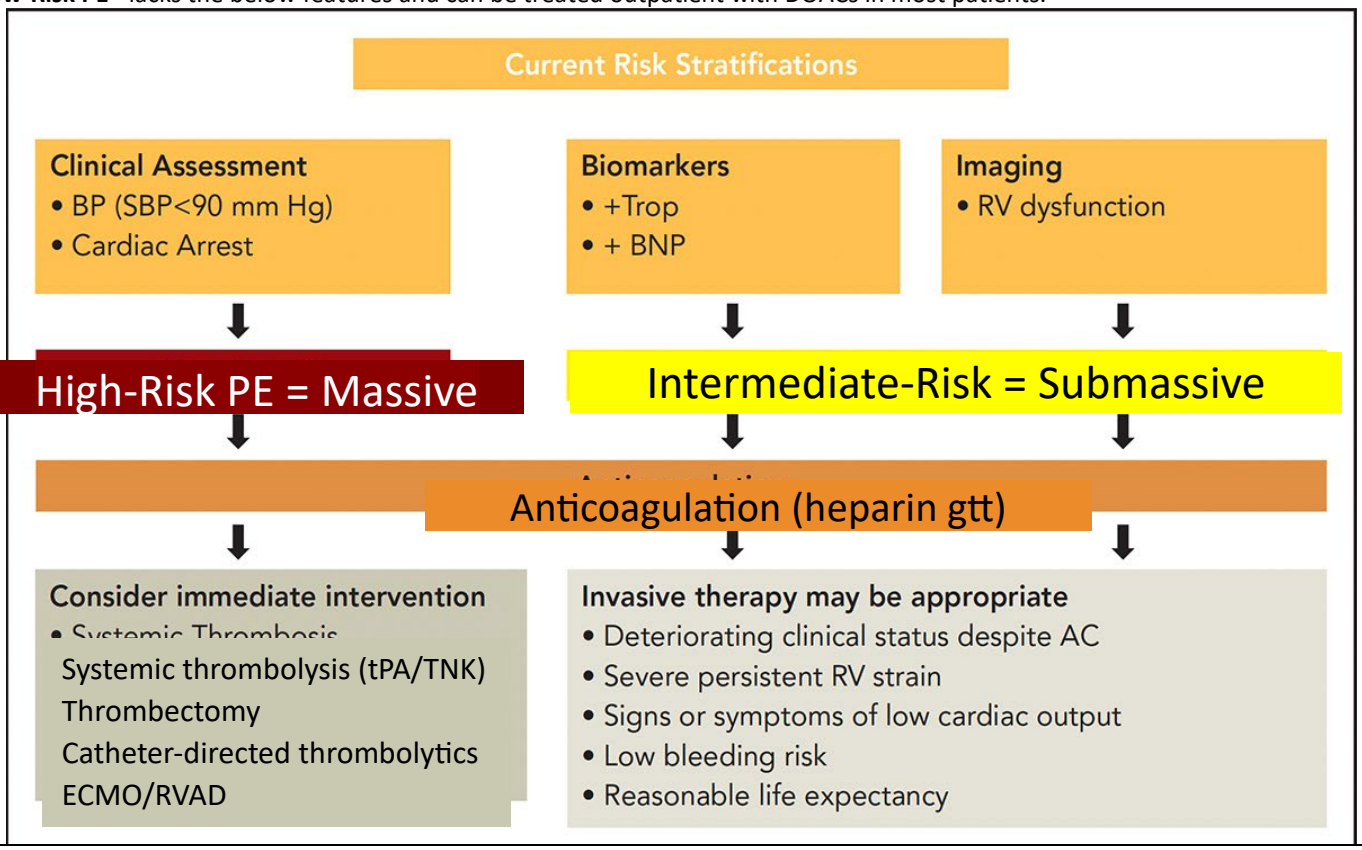
V/Q Scan – preferred in pregnancy, CKD4-5 not ESKD; also preferred for CTEPH (Group 4 Pulm HTN) dx
- Only offers probability estimate

Unable to perform CTPE and V/Q Scan inconclusive – serial lower extremity DVUS, pulmonary angiography, MRA, or V/Q SPECT
- If suspicion is high enough, treat empirically

Risk Stratification with treatment pathways

The **Pulmonary Embolism Severity Index (PESI**) can be used to assist with classification

Low-Risk PE - lacks the below features and can be treated outpatient with DOACs in most patients.



Anticoagulants

Unfractionated Heparin
- Drip is appropriate initial mgmt in submassive and part of appropriate mgmt in massive, will not delay IR
- Can do weight-based subQ dosing but not commonly done

LMWH (preferred in active cancer or pregnancy or for enteral absorption concerns)
- Enoxaparin (Lovenox) 1 mg/kg subQ q12h

Fondaparinux weight-based

DOAC or Thrombin inhibitor
- Apixaban (Eliquis) 10 mg BID x 7d, then 5 mg BID – preferred – do not dose reduce for sCr or age
- Rivaroxaban (xarelto) 15 mg BID x 3 weeks, then 20 mg daily
- Dabigatran (pradaxa) 150 mg BID (after 5-10d parenteral AC)

VKA (Warfarin) – other options are available with lower bleeding risk or bridging requirement

Duration of Anticoagulation for PE

- Provoked (trigger is no longer present): 3 months
- Unprovoked: 3 months
    - HERDOO2 score: guidance for women with their first unprovoked VTE
        - Low risk (score 0-1) can stop after 6 months
- Cancer related: secondary prophylaxis as long as the patient has active cancer
- Recurrent unprovoked: long-term anticoagulation

Acute Exacerbation of COPD

Diagnosis	Cardinal symptoms: Increase in any 2 of dyspnea, cough frequency/severity, or sputum volume/purulence			
Triggers	Infectious (70%, often viral) vs. Other (30%, environmental pollutants, PE, HF, aspiration, MI)			
Eval	CBC w/diff, BMP, Trop, ProBNP, EKG, RVP, CXR +/- CTPE, TTE			
Treatment Setting	OUTPATIENT (80% AECOPD) No sxs of respiratory failure No new or atypical exam findings No serious comorbidities Adequate response to ED/office management Sufficient home support	INPATIENT (no AHRF) RR <=24 No accessory muscle use No change in mental status Hypoxia improves with routine supp O2 PaCO2 at baseline	INPATIENT (non-life threatening AHRF) RR >24 Accessory muscle use No change in mental status Hypoxia improves with routine supplemental O2 PaCO2 50-60 mmHg or above baseline	ICU (life-threatening AHRF) RR>24 Accessory muscle use Acute AMS Hypoxia requiring BiPAP, HFNC, MV PaCO2 above baseline OR pH <=7.25
Acute Treatment	SABA +/- SAMA Cont home LABA, LAMA Consider abx if risk factors present or sxs worsening despite appropriate tx Consider PO steroids x5-7d Antiviral (Tamiflu, Paxlovid, e.g.)	Bronchodilators (nebulized & inhalers) - Increase dose / frequency - Combined SABA/SAMA - Add LABA when stable IV vs PO steroids (5-7 days of PO prednisone 40 mg daily) Consider antibiotics if bacterial infection suspected Consider NIV (esp if pH <7.35) Treat underlying etiologies as able (antivirals, diuresis, e.g.)		Same as inpatient care Consider mechanical ventilation if: Unable to tolerate NIPPV Post-arrest Aspiration/vomiting Hemodynamic instability Unable to protect airway (AMS, e.g.)

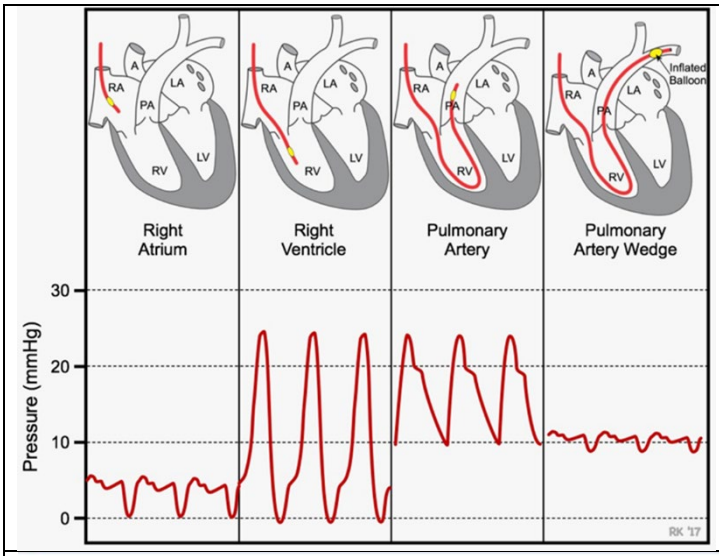
Occupational Lung Diseases

Taking an Exposure History

1. **Work History**
   - Open ended questions of duty descriptions & activity changes, length of time – for EACH job
2. **Risk of Exposure**
   - Work área description; ventilation, visible dust/vapors/gas/fumes, PPE required/provided/worn, SDS data
3. **Temporal Relationship**
   - New projects, changes at work; symptom association with being at work vs home; others at work with sxs?
4. **Non-occupational exposure assessment**
   - Home location, age, length of time living there; humidity/ventilation/vapors/molds/pets/hobbies/travel

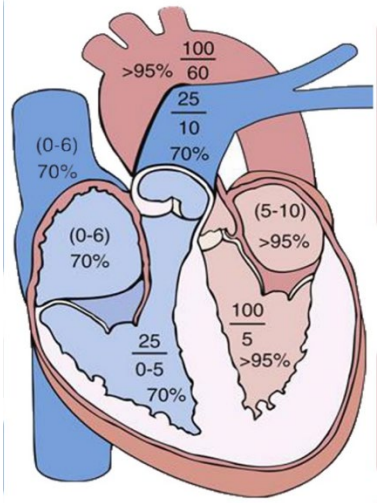
Disease	Exposure	Imaging	Association
Asbestosis	Construction, buildings prior to 70s, ship building	Nodular Pleural based masses and plaques; effusion	Mesothelioma, Small cell and non-small cell lung CA
Silicosis	"Earth Crust" Disturbances. Cutting, grinding, (sand)blasting, hydraulic fracking for natural gases	Ground-glass, nodular, interstitial, and/or fibrotic infiltrates. Pleural effusions less common.	Fibrotic Lung Disease Acute to Chronic course Simple vs complicated
Anthracosis (Coal)	Coal mining, with or without Silica	Honeycombing & groundglass with silica, nodular opacities without silica. Pleural Effusions not seen	Pulmonary Fibrosis without silica, Rapidly progressing ILD with silica.
Cobalt	Hard metal dust & Cobalt Processing. Diamond polishing, Clean energy -> batteries = cobalt cathodes for lithium-ion batteries. Vital to DOD: Munitions, Aerospace alloys, Batteries, Magnets	Nodular & reticular opacities, cystic spaces, pleural effusions uncommon	Pneumoconiosis, Giant Cell Interstitial Pneumonitis
Berylliosis	Aerospace, automotive, nuclear, weapon systems, laser/xray, telecommunication industries	Parenchymal nodules, GGOs in early stages -> hilar lymphadenopathy, interstitial pulmonary fibrosis, and pleural thickening in later stages	Up to 6% of presumed sarcoid is berylliosis Can be diagnosed with serum or BAL beryllium lymphocyte proliferation test (BLPT)

Interpreting Right Heart Catheterization (RHC) Data



Swan-Ganz / PAC / RHC Tracing

What you see shows where you are!



Normal Intracardiac Pressures:
RA = Nickel
RV/PA = Quarter
LA/PCWP = Dime
Systemic/LV = Dollar

PULMONARY VASCULAR RESISTANCE
= (meanPAP – PCWP)/CO

	PCWP ≥ 15	PCWP < 15
PVR ≥ 3 WU‡	*Combined Post- & Pre-capillary PH (CpcPH)*	*Isolated Pre-capillary PH*
PVR < 3 WU	*Isolated Postcapillary PH (IpcPH)*	*Consider high flow state*

Think of it like Ohm's Law:
Resistance = Pressure / Flow or R = V/I
Lung Arterial Resistance = Lung BP gradient / blood flow
Or,
PVR = Trans-Pulmonary Gradient (TPG) / Cardiac Output (CO)
Where TPG = mPAP – PCWP
Thus, **PVR = (mPAP – PCWP) / CO**

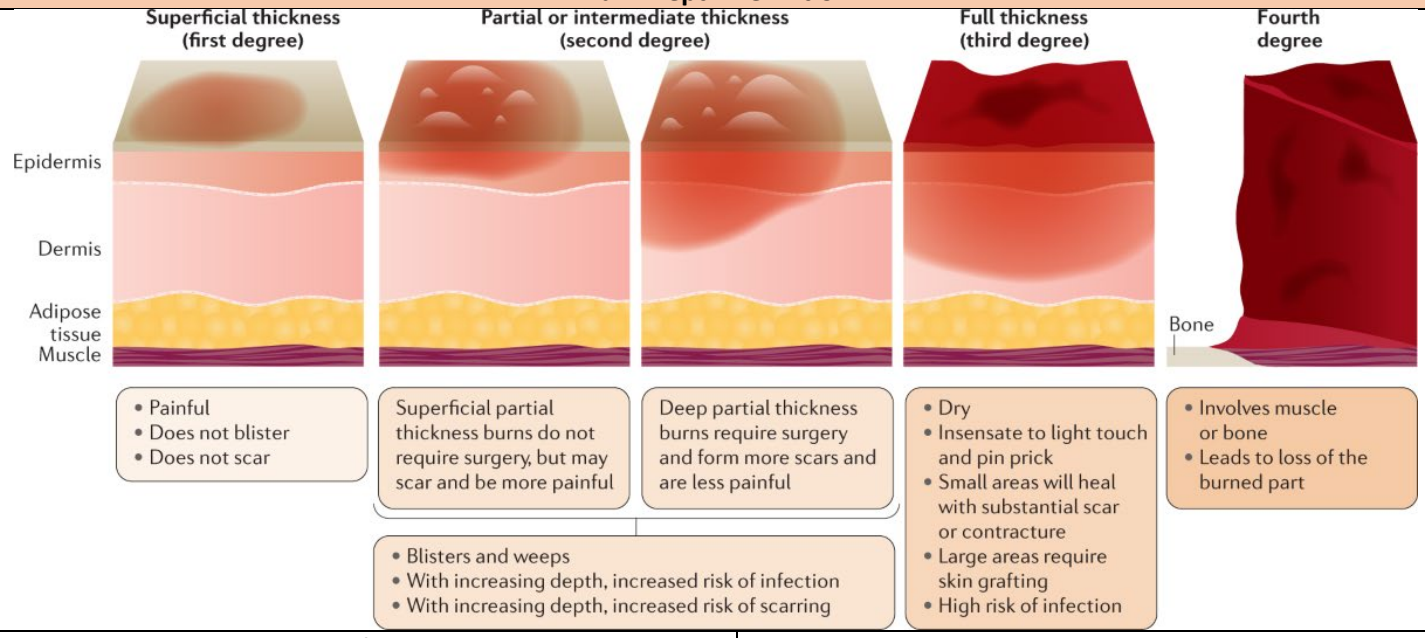
PCWP >3 Woods Unit (WU) is PH
(though truly 2+ WU is very abnormal)
Note: Diagnosis made ideally with patient in euvolemic state. TTE data like elevated RVSP, PASP, TV regurgitant jet velocity, chamber/valvular changes are suggestive if not diagnostic

Pulmonary Hypertension

	Group 1 - PAH	Group 2	Group 3	Group 4 - CTEPH	Group 5
Causes	Idiopathic, Heritable, Drug/Toxin, HIV, CTD, Portal HTN, etc	Left Heart Dz, Valvular dz	Pulm dz / Chronic Hypoxia	Prior or Recurrent PE	Other (Sarcoidosis, Sickle Cell, etc)
Evaluate	HIV, ANA, ANCA, MPO, Scl-70, LFT, RUQUS	TTE	HRCT, PFT, PSG	**V/Q Scan *NOT CTA***	Targeted Labs/Bx
Pre/Post Capillary	Pre	Pre and/or Post	Pre	Pre	Pre and/or Post
Treat	Pulm Vasodilators: CCB, **PCA (Ambrisentan) + PDE-5 (Tadalafil)** GC or ERA	Manage primary	Manage primary Inhaled PCA (Tyvaso) O2 if PaO2 <55	**GC (Riociguat) Thrombo-endarterectomy**	Manage primary Pulm vasodilators

Burn Management Basics

Burn Depth Definition



Indications for Burn Center Care / Consult

- Full thickness burns, Partial thickness burns >= 10% TBSA
- Any deep partial or full thickness burns involving the face/hands/genitals/feets/perineum/joints
- Other comorbidities or concomitant trauma
- Poorly controlled pain
- Inhalational injury
- Chemical or electrical injury (incl. lightning)

Indications for Intubation

- Persistent cough, stridor, hoarseness, or wheezing
- Deep facial or circumferential neck burns
- Nares w/inflammation or singed hair
- Carbonaceous sputum/burnt matter/blisters in oropharynx
- Altered mental status
- Respiratory distress, Hypoxia/hypercapnia
- Elevated carbon monoxide/cyanide levels

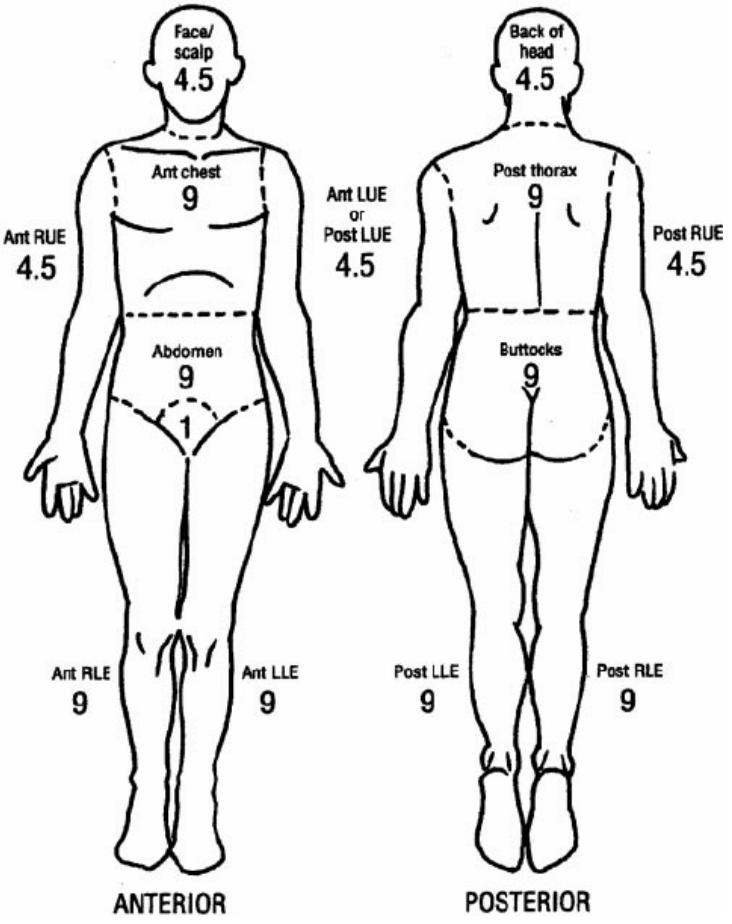
Classifying Burns:

Mild – outpatient or ED care
Moderate – inpatient but not burn center
Severe - Any burn that requires burn center mgmt.

Managing a Severe Burn:

1. Overall stabilization (pressors, airway, trauma/bleed, etc)
2. Fluid Resuscitation – Parkland Formula
   2mL/kg of body weight x %TBSA given IV (half in 8hrs, half over next 16hrs)
3. Early surgical management 24-72h (excision, grafting, etc)
4. WOCN / dressing mgmt for non-op wounds
5. VTE PPX, enteral nutrition, pain control!
6. Abx only for those with proven infection or sepsis

Estimating %TBSA Burned – Rule of 9's



Targeted Temperature Management after ROSC

	TTM1 (2013)	TTM2 (2021)
Premise	Prior trials (HACA) in 2002 had shown survival and neurologic benefit with therapeutic hypothermia aka TTM to 32-34C for patients with out-of-hospital VF or pulseless VT arrest → became standard of care. TTM has complications (decr CO, infxn, electrolyte issues, sedation needs) & is based on small trials. These studies re-evaluated TTM.	
Purpose	Compared outcomes between post-arrest temperature targets of 33C (mild hypothermia) and 36C (normothermia)	Compared therapeutic hypothermia (33C) to targeted normothermia (<37.8C)
Population	939 patients at 36 sites in Australia, Europe who were comatose after out-of-hospital cardiac arrest	1,861 patients at 61 sites in Australia, Europe, US who were comatose post-arrest
Findings	No significant difference in mortality or composite of mortality/poor neuro recovery at 6 months	No significant difference in mortalityat 6 months between groups. Neuro outcomes similar
Takeaway	Cooling to 33C was not associated with reduced all-cause mortality or improved neuro outcomes compared to goal of 36C	**Therapeutic hypothermia is not superior to targeted normothermia for neuroprotection of comatose post-arrest patients**

Rheumatology

Gout

Definition	Impaired metabolism of or excessive purines to process by xanthine oxidase leading to build up and precipitation of monosodium urate crystal deposition in joints, followed by inflammatory phagocytosis	
Risk Factors	Male sex, CV disease, overweight/obesity, smoking, high meat or alcohol consumption, Can be precipitated by illness/infection, surgery, MI, diuretic use, etc	
Evaluation	Uric acid level – crystals begin to precipitate >6.8 in colder peripheral tissues Arthrocentesis – rule out septic joint; identify intracellular negatively refringent crystals XR – look for erosions, tophi	
Treatment	**Acute Flare**	**Chronic**
	Features: First flare often podagra (50%) or monoarticular (feet) Subsequent flares can be oligoarticular (variety) Acute onset (overnight often), will self-resolve within few days without treatment but dz progresses w/ each flare (intercritical phase gets shorter) Colchicine (first-line) - Use caution or avoid in CKD, hepatic dysfunction, risk for med-med interactions - Diarrhea may be intolerable Steroids (PO and/or intrarticular) - Preferred in older adults, ESKD - Avoid with infection, DM, HF NSAIDs - Variety of agents equally effective - Avoid in CKD, PUD, anticoagulation IL-1 inhibitors (anakinra, canakinumab) - If no other options **Can start ULT up front with these if indicated!!**	Lifestyle modifications: - Guidelines conflicted, but consider modest dietary reduction in seafood, meat, alcohol; weight loss, ?increased dairy intake; non-diuretic anti-HTN switches (e.g. to losartan) Indications for Urate Lowering Therapy (12349): 1+ Tophus 2+ flares in 1 year CKD3+ Ne-4-olithiasis Uric acid >9 ULT - Xanthine oxidase inhibitors (XOI) Allopurinol – test HLA-B*5801 for Black/Asian pts to avoid drug-induced hypersensitivity rxns - Dose reduce in CKD - Prescribe daily colchicine w/ start/dose incr Febuxostat – ok for ppl with HLA-B*5801 - Second line due to CV risks Uricosurics (probenecid) not generally used Pegloticase an option in refractory dz - Many develop antibodies limiting efficacy & raising rxn risk Goal: Urate <6 (or Urate <5 if tophi)

Spondyloarthropathies

Inflammatory Arthritis		
Morning stiffness lasting >1 hr Improves with activity Joint swelling, bogginess, erythema, and/or warmth		
By Joint Involvement		
One	<6 joints	6 or more joints
Septic Arthritis Crystalline arthropathies (gout, pseudogout)	Spondyloarthropies: Clues: asymmetric, often axial, enthesitis, dactylitis Psoriasis Ankylosing spondylitis Reactive arthritis Unspecified SpA	Rheumatoid arthritis Systemic rheumatologic diseases: SLE, Systemic Sclerosis, Polymyositis, dermatomyositis, Sarcoidosis, Vasculitides

Idiopathic Inflammatory Myopathies

Suspect…	Objective weakness Muscle pain, fatigue, objective weakness, new rash, elevated LFTs/CK			
Evaluation	First Line labs: BMP, LFT, CK (will correlate to disease activity), LDH, aldolase Second Line labs: myositis panel with antibodies as below – talk to your rheumatologist Histology: EMG (may be falsely normal) or skin (esp for dermatomyositis lesions) and/or muscle biopsy (helpful) ILD evaluation: CT chest & PFT w/DLCO Swallow eval: MBS MRI pelvis/thighs – can demonstrate inflammatory process & guide biopsy site, but NOT required for dx			
	Dermatomyositis	**Polymyositis**	**Necrotizing autoimmune myositis**	**Inclusion body myositis**
Onset	Subacute	Subacute	Acute/subacute	Insidious!
Pattern	Proximal, symmetric	Proximal, symmetric	Proximal	Proximal & Distal, asymmetric
CK	Up to 50x ULN	Up to 50x ULN	>50x ULN	Up to 10x ULN
Auto-Abs	Anti-MDAS, anti-Mi-2, anti-TIF-1, anti-NXP-2	Anti-synthetase	Anti-SRP, anti-HMGCR	Anti-NT5c1A
Associated features	Think occult cancer!! Periorbital heliotrope rash (w/wo edema), shawl sign (upper back), Gottron's papules (MCP/PIP/DIP) May have antisynthetase s/o	Diagnosis of exclusion Muscle biopsy helpful May have anti-synthetase s/o	Weakness is SEVERE, usually worsens after offending med discontinued; fewer extramuscular sxs	Finger & forearm involvement. Develops over years, usually steroid-refractory

Diagnosing Lupus

New EULAR/ACR criteria for the classification of SLE

Clinical domains	Points
Constitutional domain	
Fever	2
Cutaneous domain	
Non-scarring alopecia	2
Oral ulcers	2
Subacute cutaneous or discoid lupus	4
Acute cutaneous lupus	6
Arthritis domain	
Synovitis or tenderness in at least 2 joints	6
Neurologic domain	
Delirium	2
Psychosis	3
Seizure	5
Serositis domain	
Pleural or pericardial effusion	5
Acute pericarditis	6
Hematologic domain	
Leukopenia	3
Thrombocytopenia	4
Autoimmune hemolysis	4
Renal domain	
Proteinuria > 0.5 g/24 hr	4
Class II or V lupus nephritis	8
Class III or IV lupus nephritis	10

Immunologic domains	Points
Antiphospholipid antibody domain	
Anticardiolipin IgG > 40 GPL *or* anti-β2GP1 IgG > 40 units *or* lupus anticoagulant	2
Complement proteins domain	
Low C3 *or* low C4	3
Low C3 *and* low C4	4
Highly specific antibodies domain	
Anti-dsDNA antibody	6
Anti-Sm antibody	6

REFERENCE: Aringer et al. Abstract #2928. 2018 ACR/ARHP Annual Meeting

- ✓ **Classification criteria are not diagnosis criteria**
- ✓ All patients classified as having SLE must have ANA ≥ 1:80 (entry criterion)
- ✓ Patients must have ≥ 10 points to be classified as SLE
- ✓ Items can only be counted for classification if there is no more likely cause
- ✓ Only the highest criterion in a given domain counts
- ✓ SLE classification requires points from at least one clinical domain

@Lupusreference

Diagnosing HLH

Table 2. HLH-2004 diagnostic criteria

The diagnosis of HLH can be established if Criterion 1 or 2 is fulfilled.

1. A molecular diagnosis consistent with HLH

2. Diagnostic criteria for HLH fulfilled (5 of the 8 criteria below)
   - Fever
   - Splenomegaly
   - Cytopenias (affecting ≥2 of 3 lineages in the peripheral blood)
     - Hemoglobin <90 g/L (hemoglobin <100 g/L in infants <4 wk)
     - Platelets <100 × 10^9/L
     - Neutrophils <1.0 × 10^9/L
   - Hypertriglyceridemia and/or hypofibrinogenemia
     - Fasting triglycerides ≥3.0 mmol/L (ie, ≥265 mg/dL)
     - Fibrinogen ≤1.5 g/L
   - Hemophagocytosis in bone marrow or spleen or lymph nodes. No evidence of malignancy.
   - Low or no NK cell activity (according to local laboratory reference)
   - Ferritin ≥500 µg/L
   - sCD25 (ie, soluble IL-2 receptor) ≥2400 U/mL

If hemophagocytic activity is not proven at the time of presentation, further search for hemophagocytic activity is encouraged. If the bone marrow specimen is not conclusive, material may be obtained from other organs. Serial marrow aspirates over time may also be helpful. The following findings may provide strong supportive evidence for the diagnosis: spinal fluid pleocytosis (mononuclear cells) and/or elevated spinal fluid protein and histological picture in the liver resembling chronic persistent hepatitis (biopsy). Other abnormal clinical and laboratory findings consistent with the diagnosis are cerebromeningeal symptoms, lymph node enlargement, jaundice, edema, skin rash, hepatic enzyme abnormalities, hypoproteinemia, hyponatremia, and elevated very low-density lipoprotein (VLDL↑)/low high-density lipoprotein (HDL↓).

Schnitzler Syndrome

Defined	A rare form of chronic urticaria associated with IgM monoclonal gammopathy
Epi	Usually European background, onset around age 51 ± 10 years, no clear causal gene mutation Associated with hematologic malignancy
Symptoms	Arthralgias without arthritis, Chronic urticarial rash, Periodic fevers Lymphadenopathy, Hepatosplenomegaly, Recurrent pericarditis
Diagnostics	CBC: Leukocytosis ESR/CRP: Elevated SPEP/UPEP: MGUS (usually Kappa predominant) FDG/PET: Increased avidity in bone, muscle, lymph nodes LAE, CK, Aldolase, RF, CCP, ANCA often normal
Treatment	IL-1 pathway inhibition - Anakinra (first-line) - Rilonacept or canakinumab (second-line agents)