INTERNAL MEDICINE RESIDENCY PREP COURSEPACK

TABLE OF CONTENTS

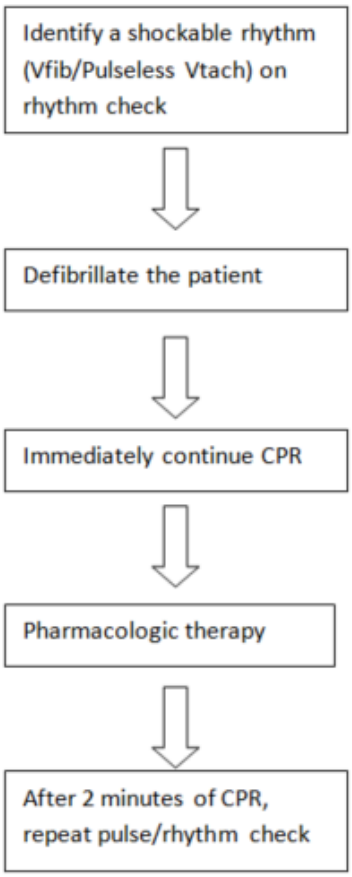
CARDIOVASCULAR

- True code blue: unresponsive & no pulse

ACLS-HOW TO RUN A CODE

- Get code page → walk quickly to room → Is it a real code? (or LOC, seizure, etc…) → If unresponsive, check for a central pulse (carotid or femoral) → Ask what patient's code status is (assume full code if info unavailable) → If no pulse… start chest compressions → Get 3 things (**monitor, O2, IV**) → assign roles! (see below) & continue CPR until algorithm says to stop CPR (not even for intubation ease)
- As soon as monitor is on do you first pulse and rhythm check - need to check pulse WHILE you look at rhythm
- If VF or pulseless VT, then SHOCK (defibrillate and follow below algorithm)!
    - Abnormal rhythms you can shock into a normal rhythm. THINK PEA looks like a normal rhythm and asystole is no rhythm, so no shock…
    - After shock, immediately CPR and follow below
    - AHA recommends charging 15s before the end of 2 minutes so the machine is ready to shock, but this makes Dr. Chang nervous in case a provider gets shocked by accident

Ventricular Fibrillation/Pulseless Ventricular Tachycardia algorithm

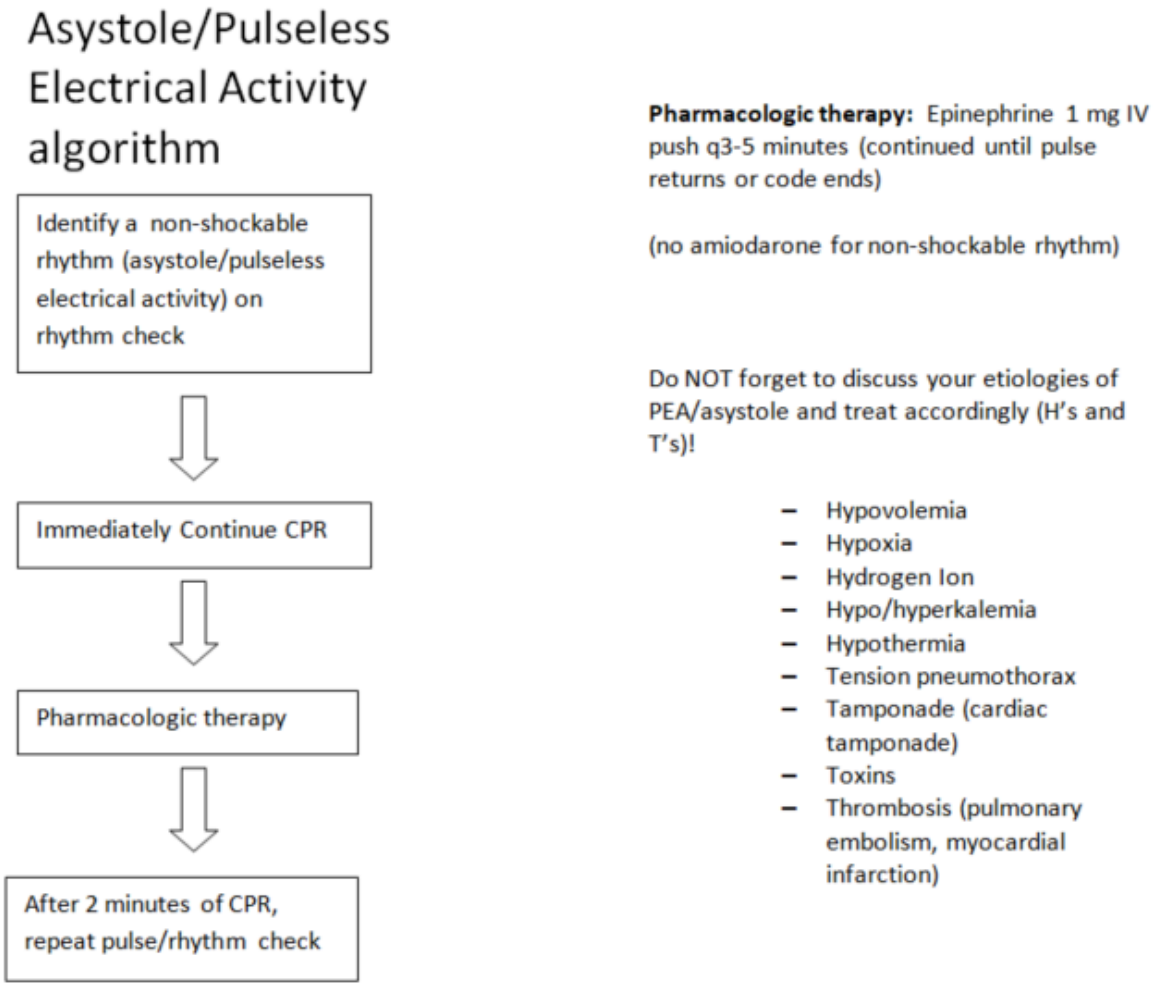


Monophasic defibrillator: 360 J
Biphasic defibrillator: 120-200 J for 1st defibrillation, then 360 J for all subsequent defibrillations

Main pharmacologic therapy, started after 2nd defibrillation: Epinephrine 1 mg IV push q3-5 minutes (continued until pulse regained or code ends)

After 3rd defibrillation: Amiodarone 300 mg IV bolus x1 OR Lidocaine 1-1.5 mg/kg
After 4th defibrillation: Amiodarone 150 mg IV bolus x1 (no more amio after this dose) OR Lidocaine 0.5-0.75 mg/kg

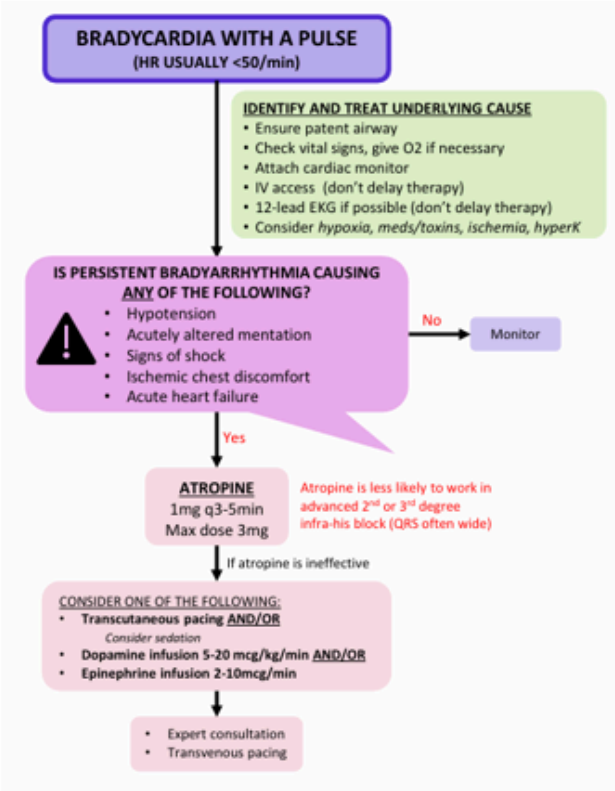
- If PEA or asystole… continue CPR and begin meds
- After 2 minutes of CPR, repeat rhythm check
- Don't forget to discuss reversible causes (Hs&Ts - see below)
- Switch back to VT/VF algorithm if indicated



- Roles
  - Code leader
  - Time keeper
  - Pulse checker
  - Rotation of CPR people - usually rotate every 2 minutes - always next person available in line
  - +/- CPR coach to assess whether compressions are high quality
  - Messager to pharmacy - b/c pharmacist usually stands outside of the room
  - Chart checker
  - Family caller
  - Call the ICU
- When to end the code?
  - Return of spontaneous circulation (ROSC), *or*
  - You decide to end the code - usually 20-30 minutes is reasonable depending on your discretion
  - Family can ask to end the code
- If ROSC achieved…
  - Obtain vitals and respond accordingly
  - EKG and respond accordingly (if MI → immediate cath lab)
  - If patient can't follow meaningful commands → targeted temperature management (induced hypothermia) for 24 hours…though recent study showed no difference in outcomes for ROSC from outpatient cardiac arrest treated with TTM

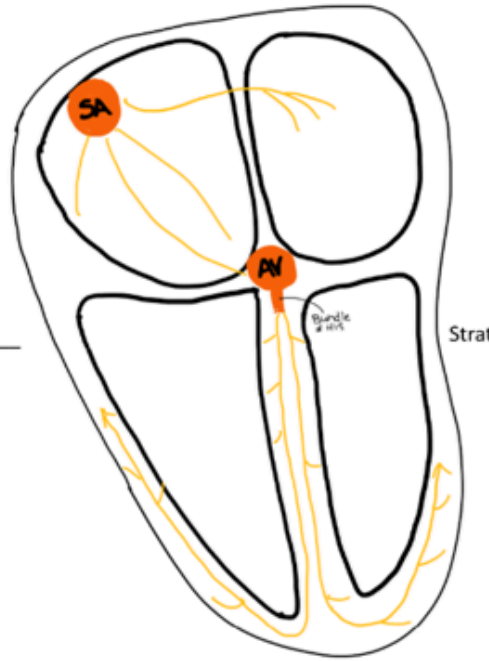
ARRHYTHMIAS

BRADYCARDIA WITH A PULSE



What are **strategies** for speeding up the heart rate?

Strategy _____



Strategy: _____

Strategy: _____

HR > 150/min

TACHYCARDIA WITH A PULSE
(HR USUALLY >150/min)

IDENTIFY AND TREAT UNDERLYING CAUSE
- Ensure patent airway
- Check vital signs, give O2 if necessary
- Attach cardiac monitor
- IV access (don't delay therapy)
- 12-lead EKG if possible (don't delay therapy)

IS PERSISTENT TACHYARRHYTHMIA CAUSING ANY OF THE FOLLOWING?
- Hypotension
- Acutely altered mentation
- Signs of shock
- Ischemic chest discomfort
- Acute heart failure

Yes → **SYNCHRONIZED CARDIOVERSION**
- Consider sedation
- If regular AND narrow, consider adenosine IV push (1st dose-6mg, 2nd dose- 12mg)

No

Is it
- Narrow vs. Wide
- Regular vs. Irregular

IS QRS WIDE?
>0.12 seconds

No:
If rhythm is **REGULAR**:
- Vagal maneuvers (valsalva, carotid massage)
- Adenosine 6mg rapid IV push followed by NS flush. Second dose 12mg
- B-Blocker or Calcium Channel Blocker

If rhythm is **IRREGULAR**
- B-Blocker or Calcium Channel Blocker

Consider expert consultation

6 Blow into a syringe

Yes:
- Consider antiarrhythmic infusion
  - **Amiodarone**
  - **Procainamide**
  - **Sotalol 100mg**
- Consider expert consultation
- Consider adenosine ONLY if regular AND monomorphic (e.g. suspected SVT with aberrancy)

TACHYCARDIA WITH A PULSE

What are **strategies** for ~~speeding up the heart rate~~ slowing down?

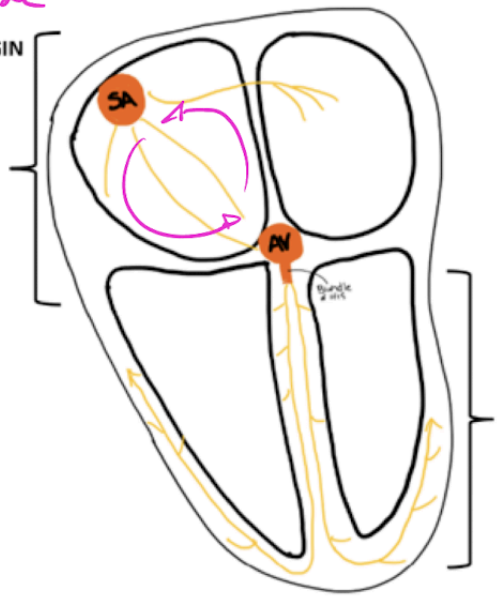
Strategy: Block AV Node

SUPRAVENTRICULAR IN ORIGIN

Origin AV Node (AVNRT) — vagal Adenosine

Origin is NOT the AV node — CCB BB

If they have a pulse, synch the shock



VENTRICULAR IN ORIGIN

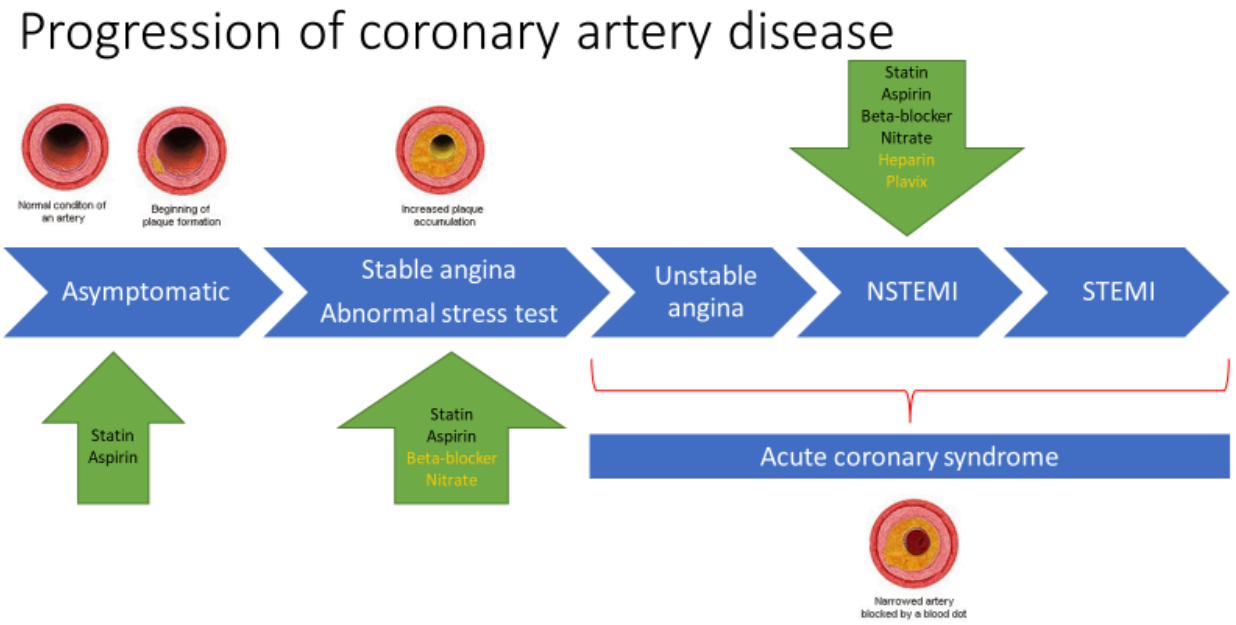
Strategy: Amiodarone

Strategy: Suppress ventricular ectopy → shock

Wide Complex Tachycardia

- Ventricular tachycardia
- Ventricular fibrillation
- Any "narrow complex tachycardia" plus a bundle branch block

Internal Medicine Residency Preparation Course 2025



Key questions:

What are the goals of stress testing?

- Detection of CAD in patients with chest pain
  - Intermediate risk patients!
- Evaluate anatomic and functional severity of CAD
- Exercise Studies (gold standard) can assess:
  - evaluation of functional capacity
  - evaluation of exercise related symptoms
  - assessment of chronotropic incompetence and arrhythmia

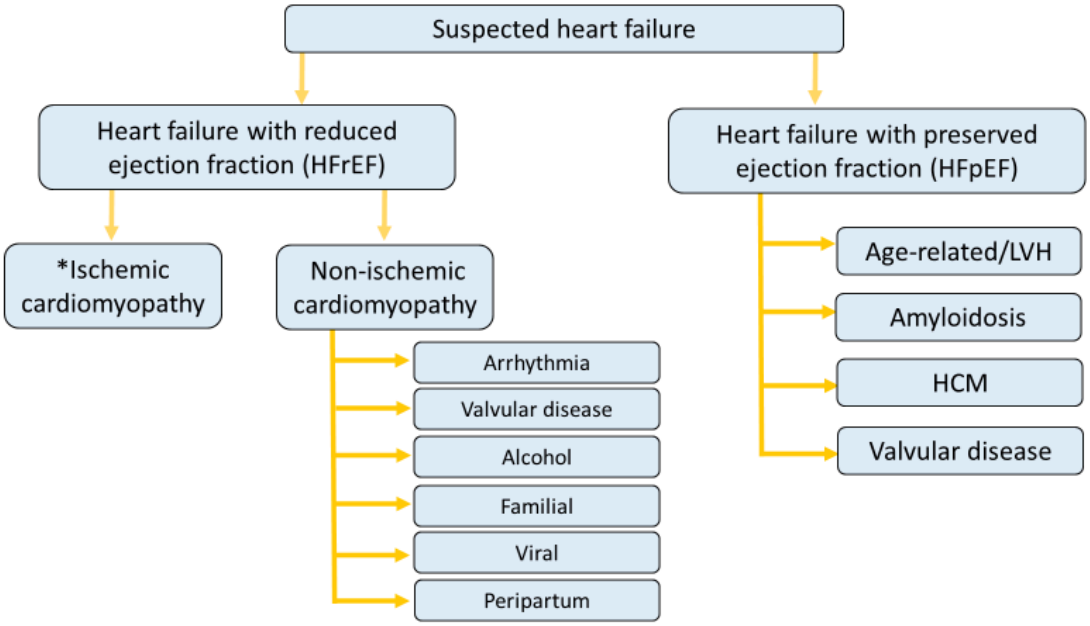
What are the key components of medical therapy in a NSTEMI?

- Statin - lowers cholesterol and lowers risk of MI (LDL < 50 if high CAD risk)
- Aspirin - reduces platelet clumping which can prevent thrombus formation, improving blood flow
- Beta-blocker - reduce myocardial oxygen demand by decreasing heart rate and contractility
- Nitrate - relaxes blood vessels and improves blood flow
  - Caution if hypotensive, severe aortic stenosis, or RV infarct
  - contraindicated if recent use of PDE inhibitor
- Heparin - prevent clot extension and reformation to improve blood flow
- Plavix - antiplatelet to prevent thrombus formation

What are indications for a more urgent cardiac catheterization for unstable angina/NSTEMI?

- more urgent catheterization if:
  - uncontrolled chest pain
  - ventricular tachychardia
  - dynamic ekg changes
  - rapidly rising troponin
- Should consider whether the patient can take dual antiplatelet therapy safely for 6-12 months

CONGESTIVE HEART FAILURE



What are the medications with a morbidity and mortality benefit in patients with HFrEF?

- **Beta Blockers** (bisoprolol, carvedilol, metop succinate)
  - Side effects: can trigger cardiogenic shock, can ↓ BP
- **ACEi/ARB/ARNI**
  - Side effects: ↓ BP, ↑ K+, ↑ creatinine
- **SGLT-2 inhibitors** (dapagliflozin, empagliflozin, canagliflozin)
  - Side effects: ↑ creatinine, ↑ risk of UTI
- **Aldosterone antagonists** (spironolactone, eplerenone)
  - Side effects: ↓ BP, ↑ K+, ↑ creatinine

***Note Loop diuretics do not decrease mortality, but do help with sx management

- Loop diuretics (furosemide, torsemide, bumetanide)
  - Side effects: ↓ BP, ↓ K+ and ↓ Na+, ↑ Creatinine

What are two new therapies for treating HFrEF?

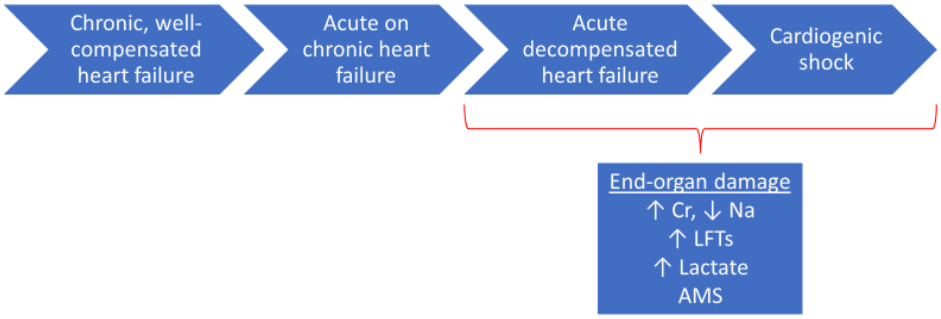
1. **ARNI:** Subcutril-valsartan (entresto) = Neprilysin inhibitor + ARB: consider in patients with HFrEF, NYHA class II-III sx, and BP tolerates ACE inhibitor/ARB
   a. Before starting, you must stop ACE inhibitor for at least 36 hours before (risk of angioedema)
   b. Can cause hypotension and hyper-K+
   c. Contraindicated if hx of angioedema with ACE inhibitors
   d. BNP levels will be falsely elevated on this med
2. **SGLT-2 inhibitors** = dapagliflozin, empagliflozin, canagliflozin: start in patients with T2D and CV disease

When is an ICD recommended for primary prevention in patients with HFrEF?

Recommended in patients with:

- Non-ischemic dilated cadriomyopathy OR ischemic cardiomyopathy at least 40 days post MI
- LVEF </= 35%
- NYHA class II-III sx on optimal guideline-directed medical therapy
- Life expectancy > 1 year (patients need to be healthy enough)

Spectrum of Heart Failure



ECG OVERVIEW

Learning Objectives:

- Develop a systematic approach to interpreting ECGs
- Translate the description of individual components of an ECG into a diagnosis
- Become familiar with common and important patient presentations requiring ECGs

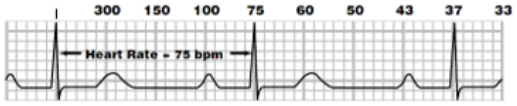
*****The majority of the time will be spent on working through examples and cases*****
*****Please review the quick reference below and prior M1 lectures before this session*****

Quick Reference on Structured ECG Interpretation:
(Figures from The Complete Guide to ECGs, O'Keefe, available online through THSL)

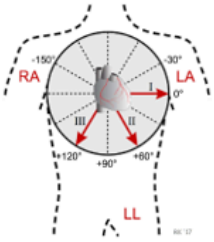
Remember, this reference is a just a quick overview of structured ECG interpretation and is not meant to be complete. You could spend countless hours trying to go through all the details of ECGs! Instead, develop a framework like this to help you get to the answers you want.

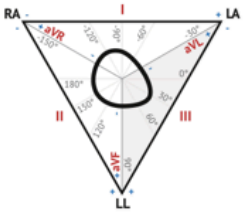
<table>
<tr><td>Ventricular Rate</td><td>Technical notes:
1. One small horizontal box is 0.04 seconds.
2. One big horizontal box (5 small boxes) is 0.20 seconds.
3. One standard ECG is 10 seconds long.

Method 1: Count the total QRS complexes in the whole ECG. Multiple by 6 to get ventricular rate since the ECG is 10 seconds long.

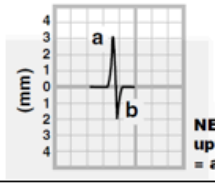
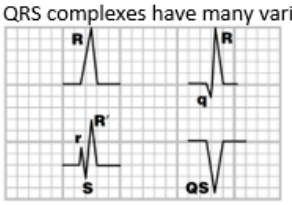
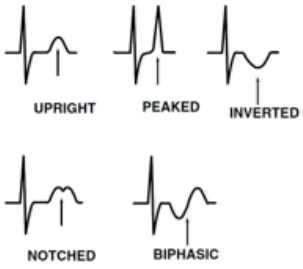
Method 2: Only works for regular rhythms. Count the number of big boxes between QRS complexes. The ventricular rate is 300 divided by this number.
 
</td></tr>
<tr><td>Rhythm</td><td>Start by identifying if the QRS complexes are regular or irregular.

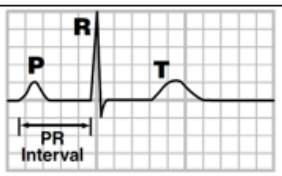
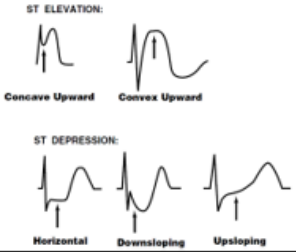
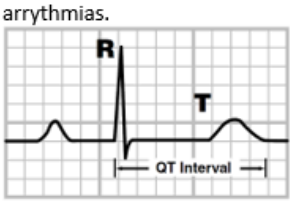
If regular, then ask if it is a sinus rhythm (impulse originating from the SA node to activate the atria) with subsequent AV node conduction to activate the ventricles. For this to be true, there needs to be:
1. A P wave before every QRS complex.
2. A QRS complex after every P wave.
3. P wave morphology must appear sinus in origin (see below in P wave).

If irregular or not sinus rhythm with normal AV conduction, there is likely some arrhythmia or heart block occurring. Determining the exact abnormality may require more information!</td></tr>
<tr><td>Axis</td><td>Represents the overall direction of ventricular activation. Normally this is between -30 and +90 degrees. Looking at leads I and aVF (sometimes II) will determine the axis.
 

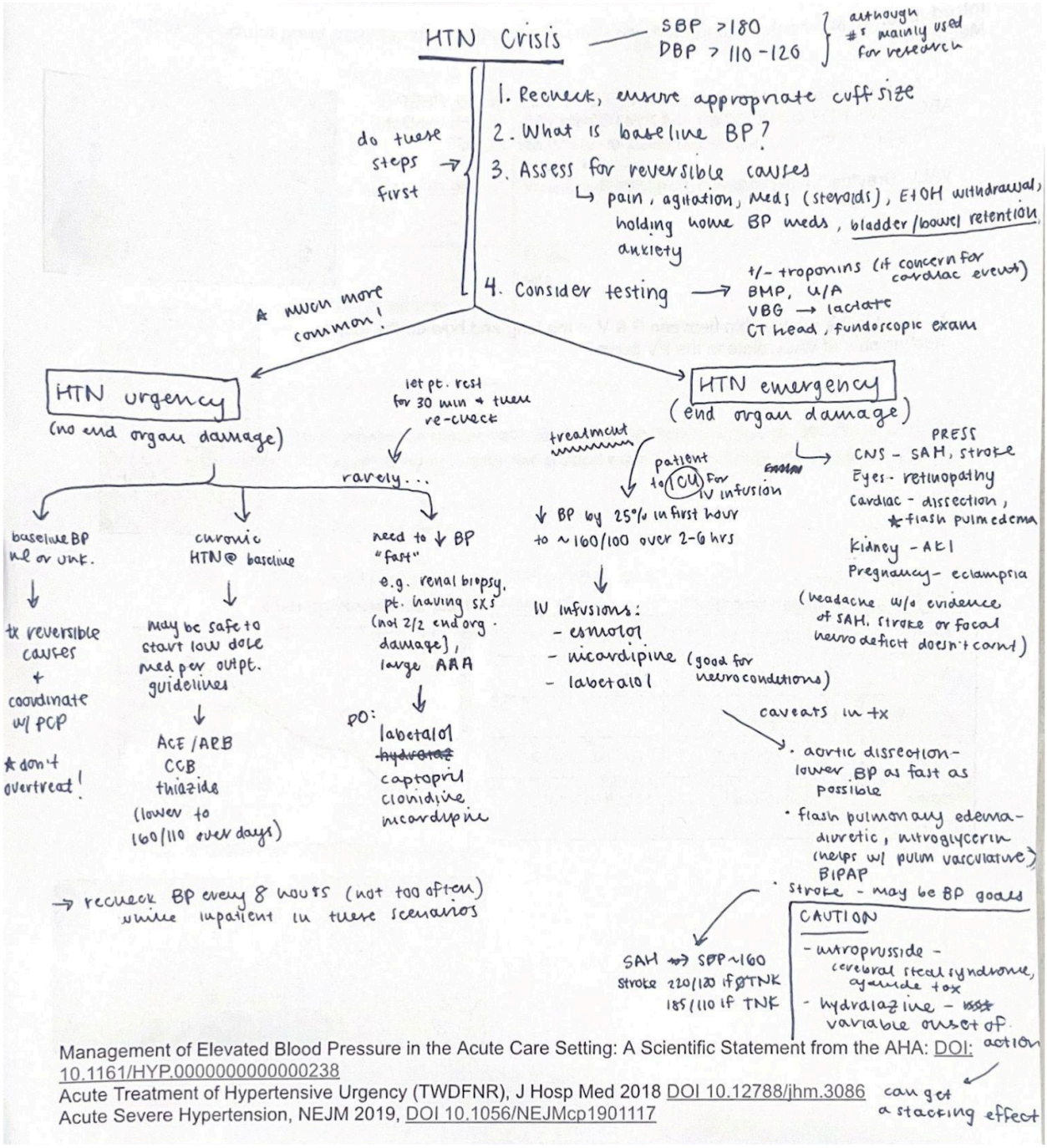


<table>
<tr><th rowspan="2">Axis</th><th colspan="3">Net QRS Voltage</th></tr>
<tr><th>Lead I</th><th>aVF</th><th>Lead II</th></tr>
<tr><td>Normal axis (0° to 90°)</td><td>+</td><td>+</td><td></td></tr>
<tr><td>Normal variant (0° to -30°)</td><td>+</td><td>-</td><td>+</td></tr>
<tr><td>Left axis deviation (-30° to -90°)</td><td>+</td><td>-</td><td>-</td></tr>
<tr><td>Right axis deviation (> 100°)</td><td>-</td><td>+</td><td></td></tr>
<tr><td>Right superior axis (-90° to +180°)</td><td>-</td><td>-</td><td></td></tr>
<tr><td colspan="4">"+" represents positive (> 0) net QRS voltage
"–" represents negative (< 0) net QRS voltage</td></tr>
</table></td></tr>
</table>

	 **NET QRS VOLTAGE =** **upward – downward deflection (mm)** **= a – b = 3 – 2 = 1 (positive)**
P wave	Represents atrial depolarization. Since the SA node is in the RA, the RA activates first and this spreads to the LA. This is why the first part of the P wave better reflects the RA and the second part better reflects the LA. This is helpful when thinking about right and left atrial enlargement. A normal P wave is: 1. Upright in I, II, aVF (because the vector of depolarization points down since the SA node is at the top of the heart). 2. Usually not more than 2 small boxes wide (may indicate atrial enlargement). 3. Usually not more than 2 small boxes tall (may indicate atrial enlargement).
QRS complex	Represents duration of ventricular depolarization. Normal duration is < 0.120 seconds. If longer than this, it indicates that there is some ventricular conduction delay (bundle branch block) or independent ventricular activation (pacing, PVC, VT). QRS complexes have many variations. In this example, the QRS duration is all the same.  **QRS duration = 1.5 small boxes = 0.06 sec.** The height/depth (voltage) of a QRS complex matters too. High voltage may indicate some degree of ventricular hypertrophy. (Technical note: one small vertical box is 1 mm and represents 0.1 mV.)
T wave	Represents ventricular repolarization. The size, shape, and direction of the T wave may help to understand the underlying disease process. For example, peaked T waves are commonly associated with hyperkalemia. 
PR interval	Represents the periods between the beginning of atrial depolarization and the beginning of ventricular depolarization. Normal duration is 0.12-0.20 seconds. An abnormally long duration (known as first degree heart block) commonly indicates delay in AV conduction.

	 **PR INTERVAL = 4 small boxes = 4 x 0.04 = 0.16 sec.**
ST segment	Represents the end of ventricular depolarization and the beginning of the ventricular repolarization. Essentially the part of the ECG between the QRS complex and the T wave. Depressions and elevations of the ST segment are concerning for myocardial ischemia. Concerning ST elevations are > 1 mm (one small vertical box). They should occur in at least two contiguous leads to be consistent with myocardial ischemia from coronary artery blockages. Some sources suggest having higher ST elevation cutoffs for leads V2-V3 as there can be some elevations there as a normal variant (> 2 mm in men age > 40, > 2.5 mm in men age < 40, > 1.5 mm in women of any age). 
QT interval	Represents the entire duration of ventricular systole, including depolarization and repolarization. Corrected QT interval (QTc) is discussed often. It is corrected for heart rate since QT duration varies due to heart rate. There are many ways to do this but the most common is to take the QT divided by the square root of the R-R interval duration. Do not worry so much about this right now. Just know a prolonged QTc is > 0.440 seconds and this can predispose you to having ventricular arrythmias.  **QT interval = 8 small boxes = 8 x 0.04 sec. = 0.32 sec.**
Diagnosis	Putting it all together in the right clinical context.

HYPERTENSIVE CRISIS



Management of Elevated Blood Pressure in the Acute Care Setting: A Scientific Statement from the AHA: DOI: 10.1161/HYP.0000000000000238
Acute Treatment of Hypertensive Urgency (TWDFNR), J Hosp Med 2018 DOI 10.12788/jhm.3086
Acute Severe Hypertension, NEJM 2019, DOI 10.1056/NEJMcp1901117

VENTILATOR 101

Lecture Notes:

- **Basic ventilator settings**
  - PPV: Three sets of waveforms we see when looking at the vent (conventionally)
    - Top curve: Pressure-time curve
      - Between the breaths, the pressure curve will return to PEEP (positive end expiratory pressure) at baseline
    - Middle curve: Flow-time curve
    - Bottom: Volume-time curve
  - Example Case: ABG: pH 7.24/ PaO2 55 / PaCO2 70, **O2 sat 70%** at FiO2 50%, PEEP 5, RR 20, TV 500
    - Normal ABG: pH 7.35-7.45 / PaO2 at least >80, many will be >95 / PaO2 35-45
    - Improve the Oxygenation: increase **FiO2** or **PEEP**
      - Increase FiO2: if you increase the FiO2 and the O2 sat does not improve, you want to consider if you have shunt physiology
      - Increase PEEP: benefit of increasing PEEP is that you get alveolar recruitment
        - In ARDS: we often will try to up the PEEP and go for lower tidal volumes
        - Negative outcomes of PEEP: barotrauma and too much pressure in chest that can compromise venous return
        - Auto-PEEP (dynamic hyperinflation): Problem that occurs in patients with underlying obstructive airway disease due to insufficient exhalation time that causes patients to retain a large volume of air in the lungs
          - PEEP total = PEEP set + PEEP auto
    - Improve the Ventilation: RR and TV (Minute ventilation = TV x RR)
      - Increase the TV: people will be very hesitant to do this because of the evidence for lung-protective ventilation in ARDS
        - ARDS → use “lung protective ventilation” where we use 6 cc/kg of ideal body weight, so people will hesitation to increase TV
      - Increase the RR: can increase, but be careful that you do not decrease the between breath time and leave insufficient time for exhalation
        - You want to know the between breath time (BBT) for a RR
          - Example: for RR = 10, BBT = 6 → BBT = time inspiration (1) + time exhalation (5), but if you increase to RR = 30, your BBT = 2, which decreases your time exhalation to 1 → leading to auto-PEEP
- **VALI: 4 major types**
  - **(1) Micro-barotrauma**
    - Cannot see that this is happening but there is so much shearing it’s creating free radical release and damage to the lungs
  - **(2) Macro-barotrauma (big concern!) / Volu-trauma**
    - Pneumothorax
      - X-Ray: absent vascular markings
      - Ultrasound: absence of lung sliding → the visceral and parietal pleura are NOT sliding up against each other because there is air in the space between them
      - If untreated → will continue to get larger and larger, leading to tension physiology and obstructive shock
    - Pneumomediastinum
      - X-ray: air in the mediastinum where air is not normally found (in the axilla, in between fascial planes in the neck, by great vessels)
      - Exam: crepitus when feeling the chest
  - **(3) FiO2 Toxicity**
    - Goal: keep FiO2 <60%
    - If FiO2 is too high can lead to free radical release and damage

- **(4) RACE Injury (Repeated Alveolar Collapse and Expansion) → atelectrauma**
  - **Cause**: injury from the shearing forces created when the alveoli open/close if there is not enough PEEP to keep them open
- **Other: VAP (ventilator-associated pneumonia)**

- **Peak and plateau pressures**

Who?	Peak	**Plateau (MOST impt!)**	PEEP(t)
What?	Maximal airway pressure during inspiration	Maximal alveolar pressure at the end of inspiration (when the lungs are most full)	Pressure in lung when most empty
Where ?	Airway pressure	Alveolar pressure	
When?	Inspiration	During Inspiration	Expiration
Why?	Worried if the peaks are going up that there is a resistance problem	Correlates with compliance, increased Pplat means decreased compliance → this gives you information about barotrauma	
How?	Look at the vent	End inspiratory breath hold (usually <30)	Expiratory breath hold

- Key Equations
  - (1) Ohm's Law: V= IR → change in pressure = F x R → Pairway - P alveolar = Flow x Resistance
    - End inspiratory hold → if you stop flow and airway and alveolar equilibrate, so P airway = P alveolar
    - You HAVE to ensure there is NO FLOW on the machine during your inspiratory hold → if there is airflow the plat is NOT REAL, and this is likely to actually UNDERESTIMATE your plat
  - (2) Compliance = delta V/ delta P = TV / (Pplat - PEEP)
    - Rearrange the equation: Pplat = TV/C + PEEP → P(alveolar) = TV/C + PEEP
  - (3) Equation of Motion: combines (1) and (2)
    - P(airway) = F x R + VC + PEEPtotal
  - Example Case: 65 yo F, severe COPD, PNA, ?ARDS
    -

Scenario	Peak	Plat	DDx
Base	30	20	
1	50	40	Pneumothorax, flash pulmonary edema, severe mucus plugging with lobar collapse, DAH (diffuse alveolar hemorrhage), abdominal compartment syndrome
2	50	20	probably a flow or resistance problem → biting the tube, secretions, anaphylaxis, post-bath if tube is kinked
3	10	~	leak!
4	30	20	New severe respiratory distress → massive PE!

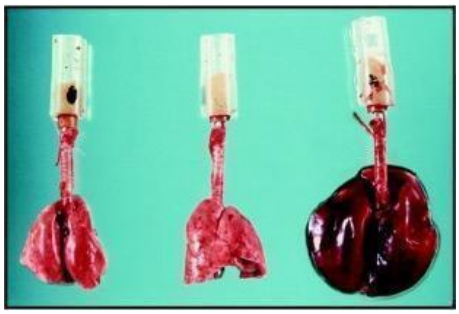
  - Text Summary of Scenarios:
    - 8 am: Peak 30, Plat 20

- Scenario 1: 8:15 am: not doing well → Peak 50, Plat 40 → this is a problem with compliance or total PEEP (they are on VC, so not a volume problem)
    - What in 15 minutes could have changed? → Pneumothorax, flash pulmonary edema, severe mucus plugging with lobar collapse, DAH (diffuse alveolar hemorrhage), abdominal compartment syndrome
- Scenario 2: 8:15: Peak 50, Plat 20 →probably a flow or resistance problem → biting the tube, secretions, anaphylaxis, post-bath if tube is kinked
- Scenario 3: 8:15 am, Peak 10, Plat ~: leak
- Scenario 4: 8:15 am, Peak 30, Plat 20, respiratory distress → MASSIVE PE!
    - In the acute setting, doesn't impact flow, resistance, volume, compliance, etc
    - Look for someone with unchanged vent setting with SEVERE respiratory distress

- PEEP Titration
    - Goal: gradually increase PEEP as you move along the volume pressure loop → When your PEEP goes up and your PPlat goes up by a disproportionate amount, then you start to be concerned that your compliance has gone down

Introduction to Mechanical Ventilation (Session 1) – Ventilator Associated Lung Injury, Ventilator Mechanics

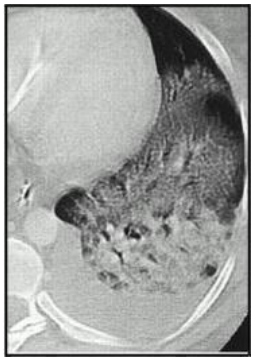
1. A patient is on AC/VC mode, 50% FIO2, TV 0.5L, RR 20, PEEP 5. ABG shows PO2 40, PCO2 60, pH 7.24, O2Sat 70%. Which ventilator settings have the most significant effect on the PO2? PCO2?

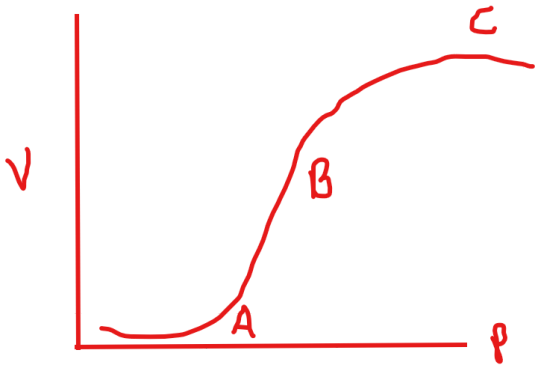


VALI. Dreyfus. ARRD 1988; 137: 1159-64.

PO2: increasing the FiO2 and PEEP (see details above)
PCO2: increasing the RR and TV (see details above)

2. What is the relationship between P & V in the lung and how do the major mechanisms of VALI relate to the PV curve?





A: Danger of RACE injury!
B: GOAL to ventilate here → the slope of the volume pressure is constant
C: Danger of barotrauma!

3. What is a plateau pressure?

What?	Pressure	Peak "hold"
Where?	Alveoli	airway

When?	End inspiration	
Why?	lungs “most full”	Resistance
How?	End-insp hold button	Look at vent

Introduction to Mechanical Ventilation (Session 1, Part 2) – Ventilator Modes, Breath Types

1. Modes: Describe the differences between Assist Control (AC), Synchronized Intermittent Mandatory Ventilation (SIMV), and Spontaneous ventilation?

	AC (usually on AC in the ICU!)	SIMV (more so a pediatric one)	Spontaneous (used for pressure support)
Mandatory Breaths	**Mandatory = spontaneous**	Not a focus of lecture	No set mandatory breaths (you don’t set a RR!)
Spontaneous Breaths			

2. Breath types: Describe the differences between Pressure Control (PC), Volume Control (VC), and Pressure Regulated Volume Control (PRVC)

	PC = tends to be more comfortable	VC (most ARDs may be put on this so you can control the TV)	PRVC (pressure regulated volume control) Aka: VC+ , volume-targeted, auto-flow
Universal Settings (for AC!)	PEEP, FiO2, and RR → these need to be programed for EVERYONE		
Clinician Controlled	-Setting the peak pressure (specifically the pressure above PEEP), PEEP, and inspiratory time	-Volume and flow (shape of the flow as well as the maximum inspiratory flow) - Time is indirectly set because you are controlling the others	-Pressure control (with a volume TARGET)
Patient Controlled	-Volume -Flow	-Pressure	

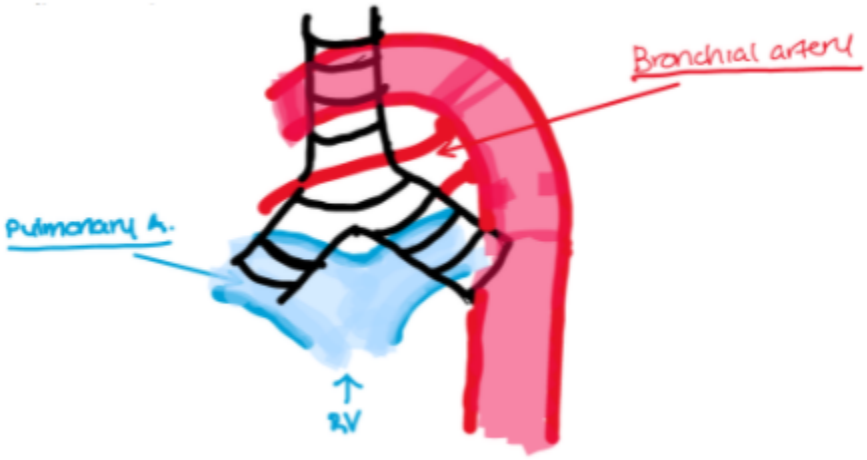
3. Three identical patients are intubated and sedated for Acute Respiratory Distress Syndrome (ARDS). You are rounding in the morning and start a Spontaneous Awakening Trial (SAT). What do you expect will happen when you wean sedation?

- SAT in Pressure Control Patient: patient is going to take deeper breaths (more volume and more flow) → so we are now no longer in lung protective strategy
- SAT in Volume Control Patient: flow and volume cannot change, so they are breathing at the opposite vector and can double-trigger a breath leading to breath stacking (breathing double what they are intended to breath)

HEMOPTYSIS

Helpful Schema for Causes of Hemoptysis:

Circulation:



Treatment:

Ref: Managing Massive Hemoptysis, Chest 2020 PMID 31374211

PULMONARY EMBOLISM

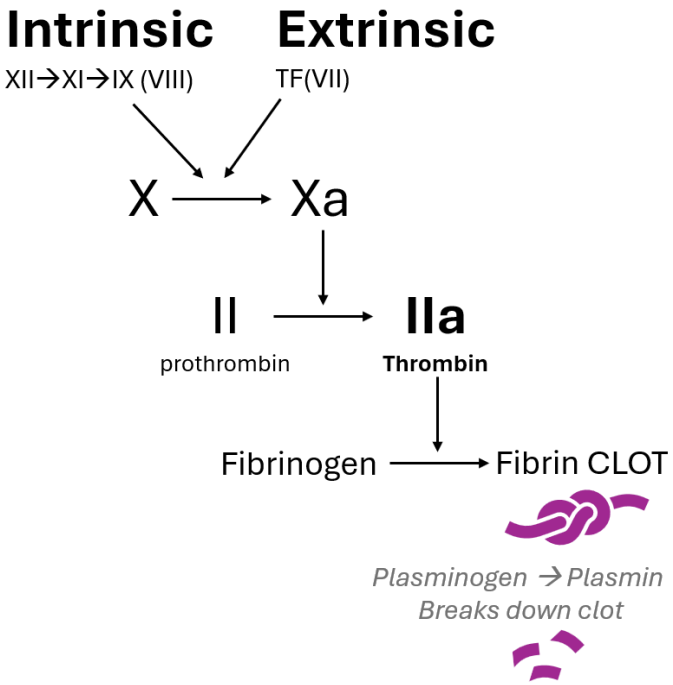
RISK STRATIFICATION

LOW	INTERMEDIATE aka "submassive"	HIGH aka "Massive"
(<10% 30d mortality)		*(>15% 30d mortality)*
<PESI =0 or clinical judgment	Increased BNP Increased troponin Right heart strain Hypoxia	Persistent hypotension
Home vs hospital (pending shared decision-making)	Needs telemetry	Needs ICU admission

TREATMENT

	Thrombolysis	**Unfractionated Heparin** (UFH, "heparin drip")	**Low molecular weight heparin** (LMWH, enoxaparin/Lovenox)	**Warfarin** (Coumadin)	**DOACs** Apixaban and rivaroxaban are the most commonly used
Dosing	Systemic or catheter-directed (dependent upon IR) **infusion**	Institution-specific. Typically bolus then **infusion** (e.g. 80U/kg IV bolus followed by 18U/kg/hr)	Enoxaparin 1mg/kg **subQ** q12hrs **OR** 1.5mg/kg **subQ** q24hrs	Typically start at 2.5mg -5.0mg **PO** QHS. Takes several days to reach therapeutic state.	Apixaban (Eliquis) – 10mg **PO** BID x 7 days then 5mg **PO** BID Rivaroxaban (Xarelto) – 15mg **PO** BID x21 days then 20mg **PO** Qday
Mechanism	Binds to fibrin and activates plasminogen which turns into plasmin (degrades fibrin clots)	Binds antithrombin inhibits IIa (thrombin)>> Xa	Binds antithrombin inhibits primarily Xa >>IIa (thrombin)	Inhibits vitamin K synthesis of factors II, VII, IX, and X	ApiXaban and rivaroXaban both directly inhibit Factor Xa
Indication	PERT TEAM!!!! (PERT = PE response team)	-Upcoming procedure -Anticipate possible thrombolysis -Cr clearance < 15-30 -Active bleeding??	-Logistics (easier to give Lovenox than Heparin gtt) -Luminal GI cancer -Bridge to warfarin Contraindicated with Cr Cl < 15!!!	-alternate indication (eg. mechanical valve) -Anti-phospholipid syndrome -D-D interaction (carbamazepine, rifampin, phenytoin)	Start here if you can!!!

Reversal	Antifibrinolytic agents such as tranexamic acid or aminocaproic acid.	Protamine	Protamine	*Typically do not reverse unless bleeding. If INR ≥10 without bleeding can consider low dose PO vitamin K (1-2mg PO). If bleeding give Vit K 10mg IV + Prothrombin Complex Concentrate (KCentra).*Use FFP transfusion if no KCentra available.	Andexanet alpha (Andexxa) can be used to reverse apixaban and rivaroxaban (idarucizumab/Praxbind for dabigatran) If not available, can use PCC.



Ref: Kearon C et al. Antithrombotic Therapy for VTE Disease. *Chest*. 2016, doi:10.1016/j.chest.2015.11.026

CIRRHOSIS: TOP 4 COMPLICATIONS

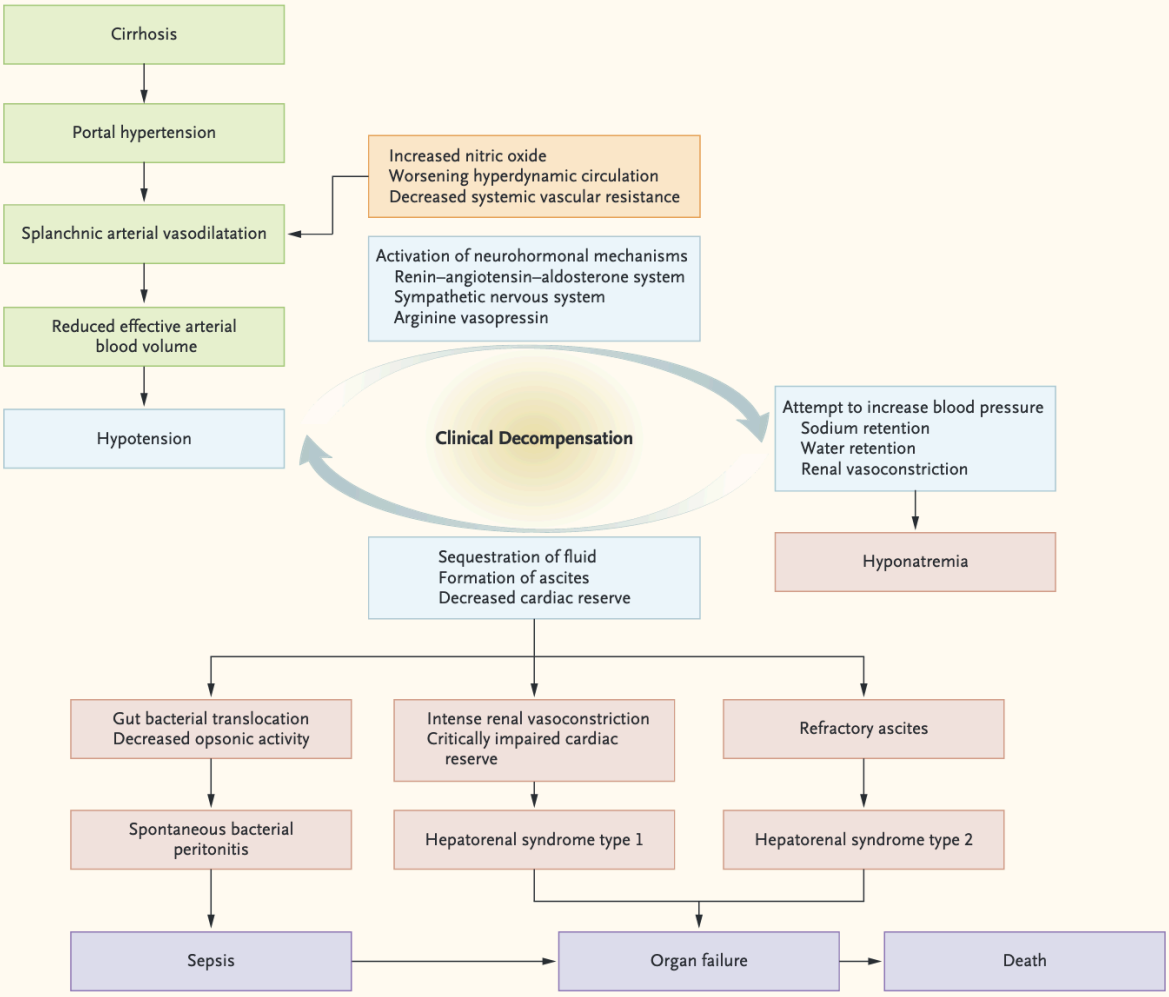


Figure 2. Pathophysiological Features of Hypotension in Patients with Cirrhosis.

The peripheral arterial vasodilatation hypothesis states that as cirrhosis progresses, systemic vasodilatation from reduced systemic vascular resistance and the sequestration of fluid into the peritoneal cavity result in arterial underfilling and activation of salt-retaining neurohormonal mechanisms such as the sympathetic nervous system and the renin–angiotensin–aldosterone system to counteract low arterial blood pressures. Consequently, although plasma and blood volume are increased, effective arterial blood volume is decreased. These circulatory changes, along with the development of sodium and water retention and the formation of ascites, are an adaptive compensatory response aimed at maintaining adequate cardiac output and organ perfusion.

CASES

Case 1

Mr. M is a 56 yo man with NAFLD cirrhosis who woke up in the middle of the night and had a dark bowel movement last night. This morning he had another large dark bowel movement.

Past medical history: DM, HTN, dyslipidemia

Past surgical history: cholecystectomy

Family history: Mother DM, father has DM and HTN

Variceal bleed

Management:
-ABCs
-Labs
-CTX, PPI (cover PUD empirically), Octreotide
-EGD once stable (banding, Minnesota tube [only stays in for 24 hours to prevent ischemia])

Case 2

Ms. P is a 39 yo female with no medical problems. She has increasing abdominal girth and weight gain for 2 months

Past medical history: none

Past surgical history: appendectomy

Family history: no liver disease

Social history: smokes 1ppd, drinks 35 servings of alcohol a week

Ascites

*****2:1 AST:ALT also seen in end stage liver disease (not just alcohol)**

Management:
-2g Na+ restricted diet (*no fluid restriction necessary - can cause AKI)**
-Diuretics
-Paracentesis (give albumin if over 5L)
-Monitor weight

Case 3

Mr. V is a 57 yo man who presents with confusion

Past medical history: chronic hepatitis C cirrhosis complicated by ascites and leg swelling

Past surgical history: splenectomy

Family history: unknown

Hepatic encephalopathy

Management:
-Blood ammonia, neuro-imaging are not helpful
-Lactulose, Miralax, or Rifaximin once showing symptoms (don't need to use as primary prophylaxis)
-Dietitian consult (need high protein diet)
-Caution with TIPS

Case 4

Ms. A is a 23 yo female who is unresponsive by her family. She was found to have an empty bottle of alcohol and extra-strength acetaminophen near her. Family last saw her 2 days ago.

Past medical history: none

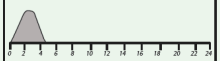
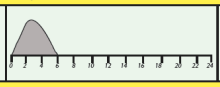
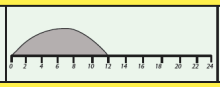
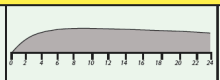
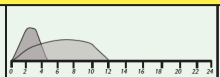
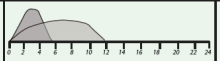
Past surgical history: none

Family history: no liver disease

Acute liver failure
-HE within 8 weeks of first symptoms without previous liver disease
-Leading cause of death = increased ICP/brain herniation
-Treat with N-Acetylcysteine for APAP overdose
-Discuss with liver transplant center
-Prognosis: use King's College Criteria

DIABETES: INPATIENT MANAGEMENT

UMHS Inpatient Formulary Insulin Products

BRAND NAME	GENERIC NAME	AVAILABLE INPATIENT FORMULATIONS	APPEARANCE	ACTION CURVE	ONSET	PEAK	DURATION	NOTES
RAPID-ACTING INSULIN (MEAL TIME and CORRECTION SCALE COVERAGE)								
Humalog	Lispro	Vial, Pen	Clear		10 to 20 minutes	1 to 3 hours	4 to 5 hours	Can be administered from time of meal to 15 minutes post-meal. Do not administer until meal tray is in front of patient.
SHORT-ACTING INSULIN (MEAL TIME and CORRECTION SCALE COVERAGE)								
Humulin R	Regular	Vial	Clear		30 to 60 minutes	2 to 3 hours	6 hours	
INTERMEDIATE-ACTING INSULIN (BASAL COVERAGE)								
Humulin N	NPH	Vial	Cloudy		2 to 4 hours	6 to 10 hours	10 to 12 hours	
LONG-ACTING INSULIN (BASAL COVERAGE)								
Lantus	Glargine	Vial	Clear		Begins working in 1 hour. Reaches full activity within 4 to 5 hours and continues at a constant level for 24 hours.			DO NOT MIX WITH ANY OTHER INSULIN. Usually given once a day, but may be given twice a day.
Levemir	Detemir	Vial	Clear		50 to 120 minutes	6 to 8 hours	16 hours	
COMBINATION INSULINS								
Humalog 75/25	75% Lispro Protamine (like NPH) and 25% Lispro	Vial	Cloudy		10 to 20 minutes	1 to 3 hours AND 6 to 10 hours	10 to 12 hours	DO NOT MIX WITH ANY OTHER INSULIN. Mixing would disrupt the ratio and could alter insulin action.
Humulin 70/30	70% Human NPH and 30% Human Regular	Vial	Cloudy		30 to 60 minutes	2 to 3 hours AND 6 to 10 hours	10 to12 hours	

*Insulin vials in use are stable for 28 days at room temperature

*Endorsed by UMHS Glycemic Management Subcommittee *Aug. 2010*

PERIOPERATIVE and PERIPROCEDURE DIABETES MANAGEMENT
For Adult Inpatients and Outpatients NOT ON INSULIN PUMP
PROVIDER DOCUMENT

DAY(s) BEFORE PROCEDURE	
1) Patient takes oral diabetes medications EXCEPT	Take usual dose
SGLT-2 inhibitors (listed below)	Hold for 2 days before procedure
2) Patient takes evening or bedtime insulin	
- **NPH**	Take usual dose
- **Mixed insulins**	Take usual dose
- **Glargine/GlargineU-300/Detemir /Reg U-500** (with no other insulin)	Take 50% of usual dose
- **Degludec** (with no scheduled meal insulin)	Take 50% of usual dose for 2 day before procedure
- **Glargine /Glargine U-300/Detemir** (as part of a regimen which includes scheduled meal insulin)	Take 70% of usual dose
- **Degludec** (as part of a regimen which includes scheduled meal insulin)	Take 70% of usual dose for 2 days before procedure
- **Regular or aspart or lispro or glulisine or fiasp**	Take usual dinner dose for regular meal Take 50% of usual dose for sugary beverage diet for bowel prep
3) Patient takes non-insulin injectables	Take usual dose
4) Patient uses insulin pump* **- See Periprocedure and Perioperative Insulin Pump Guidelines.**	Continue basal rate- **unless frequent hypoglycemia** Then reduce to temporary basal rate of 70%
MORNING OF PROCEDURE	
1) Patient takes oral diabetes medications	HOLD dose
2) Patient takes am insulin	
- **NPH**	Take 50% of usual dose
- **Mixed insulins**	Take 50% of usual dose
- **Glargine/Glargine U-300/ Detemir /Reg U-500/Degludec** (with no scheduled meal insulin)	Take 50% of usual dose
- **Glargine/Glargine U-300/Detemir/Degludec** (as part of a regimen which includes scheduled meal insulin)	Take 70% of usual dose
- **Regular or aspart or lispro or glulisine or fiasp**	HOLD doses
3) Patient takes non-insulin injectables	HOLD dose
4) Patient uses insulin pump* **- See Periprocedure and Perioperative Insulin Pump Guidelines.**	Reduce to temporary basal rate of 70%

Intraoperative and postoperative use of pump should be addressed on an individual basis in consultation with patient's endocrinologist
MIXED INSULIN INCLUDE (75/25,70/30 or 50/50)
ORAL SGLT2 INHIBITORS -Canagliflozin; Dapagliflozin; Empagliflozin; Ertuglifloxin, Sotagliflozin; alone or in combination pills
NONINSULIN INJECTABLES- Bydureon®, Byetta®, Victoza®, Symlin®, Ozempic®, Xultophy®, Soliqua®, or Trulicity®.
On days of bowel prep, use adjusted doses of Glargine/Glargine U-300/ Detemir /Reg U-500/Degludec as above

Michigan Medicine Insulin Sliding Scales

Insulin Requirement			
Blood Glucose mg/dL	❑ Low	❑ Moderate	❑ High
< 70			Follow hypog
70 - 150	0 units	0 units	0 units
151 - 200	0 units	2 units	4 units
201 - 250	2 units	4 units	6 units
251 - 300	4 units	6 units	10 units
301 - 350	6 units	8 units	12 units
351 - 400	8 units	10 units	14 units
> 400	10 units Contact M.D.	12 units Contact M.D.	16 units Contact M.D.

Inpatient Diabetes Management

Know your insulins!

- Majority are renally cleared
- 1000 units in vial, 300 units in pen

Insulin Preparations	Onset	Peak	Action
Bolus Insulin			
Short Acting Regular (“R”)	30 minutes	2-4 hours	6-10 hours
Rapid Acting Aspart (Novolog) Glulisine (Apidra) **Lispro (Humalog**, Admelog) Ultra Rapid Acting Aspart (Fiasp)	5-15 minutes 2.5-4 minutes	1-2 hours 30-90 minutes	4-6 hours 4-6 hours
Basal Insulin			
Intermediate Acting NPH (“N”)	1-2 hours	4-8 hours	10-20 hours
Long Acting			
Determir (Levemir)	3-4 hours	Nearly flat	Approx. 24 hours
Glargine (Lantus)	3-4 hours	Nearly flat	Approx. 24 hours
Glargine U300 (Toujeo)	6 hours	Nearly flat	24-30 hours
Degludec U100/U200 (Tresiba)	1 hour	Nearly flat	24-50 hours
Other			
Mixed Humulin or Novolog 70/30 Humalog 75/25 Humulin 50/50	5-15 minutes	1-2 hours	10-20 hours

How do you convert from one insulin to another?

- All insulins convert 1:1 but timing matters

You are admitting a patient to the inpatient service who has diabetes. What history do you need to take in order to decide how to manage their blood sugars during the hospitalization?

- How long has the patient had diabetes?
- What is the patient’s current diabetes regimen?

- What is the patient's adherence to their home regimen
- Does the patient check his/her BG, if so how often?
- What are the patient's BGs generally at home?
- What is the patient's previous A1c?
- Who manages the patient's diabetes as an outpatient?
- Can interventions in the hospital, including education, supplies, meds, or social interventions, improve DM care at home?

What are the inpatient blood sugar targets?

- Pre-meal <140mg/dl
- Random <180g/dl (but generally okay as long as <200)
- Try to avoid hypoglycemia

What should I do with the patient's oral or non-insulin diabetes medications?

- Typically hold non-insulin diabetes medications while inpatient

What is a basal-bolus regimen? What is the classic ratio of basal:bolus insulin?

- Total Daily Dose (TDD) = 50% Basal + 50% Bolus (meal + correction)
- Bolus = ⅓ Breakfast + ⅓ Lunch + ⅓ Dinner

What do we mean when we say someone is on a basal-heavy regimen?

- If a T2 diabetes patient only uses basal at home, in the hospital give them 75-100% of home basal dose and no mealtime coverage. Give correction qac (with meals)/qhs based on TDD.
- If a T2 diabetes patient uses basal bolus at home, in the hospital give them 75-100% of home basal and bolus dose. Give correction qac (with meals)/qhs based on TDD.
- If a T1 diabetes, in the hospital give them 75-100% of home dose. Do NOT hold basal insulin due to the risk of DKA. They always need mealtime insulin. Use custom correction (1:50 > 150) or home correction.

What is the difference between carb coverage and correction?

- Carb coverage is a preventive measure and calculated from the carb content of meal
- Correction is a corrective measure and dosed on the pre-meal blood glucose
    - Correction is not based on post-meal blood glucose, which is hard to interpret

Who should never be on a basal-only regimen?

- T1 diabetics! Even if they are not eating, because the body is always generating glucose and has a risk of DKA.

Weight-based insulin dosing

Patient characteristics	Units/kg/day
Insulin sensitive: lean, malnourished, elderly, chronic kidney disease (on dialysis), or insulin naïve	0.3
Moderately insulin sensitive: Type 1 diabetes	0.4
Average weight T2 diabetes	0.5
Insulin resistant: obese, postoperative stress, infected, or receiving high doses of corticosteroids	0.5 to 1

What is an estimated weight-based insulin regimen for a 60kg patient with Type 1 Diabetes?

60kg * (0.4 u/kg/day) = 24 units/day

How do I pick the right sliding scale?

- Low, Moderate, and High correctional scales refer to their TDD, which is a proxy of their insulin sensitivity. Pick the correctional scale according to the patient's TDD.

Correctional Scale at MM

Blood glucose mg/dl	Low TDD <40 units/day	Moderate TDD 40-80 units/day	High TDD >80 units/day	Custom low dose (very sensitive)
<70	********Follow hypoglycemic protocol*********			
100-150	0 units	0 units	0 units	0 units
151-200	0 units	2 units	4 units	1 unit
201-250	2 units	4 units	6 units	2 units
251-300	4 units	6 units	10 units	3 units
301-350	6 units	8 units	12 units	4 units
351-400	8 units	10 units	14 units	5 units
>401	10 units	12 units	16 units	6 units

How often can I give sliding scale insulin?

NPO Status

Inpatient Insulin Guidelines for NPO patients

	T2 Basal at Home	T2 Basal Bolus at Home	T1
Basal	50%	70%	70%
Mealtime	None	Hold	Hold
Correction	q6	q6	q6

Let's try adjusting insulin for NPO Status:

A patient is on 80units of Glargine QHS and is made NPO at MN.
How would you adjust his/her insulin?

A patient is on 40units of Glargine plus 13units Lispro TID with meals and is made NPO at MN. How would you adjust his/her insulin?

A patient is on NPH 30units in the morning and 20units before bed and is made NPO at MN.
How would you adjust his/her insulin?

Stress hyperglycemia
- Any BG > 140 in patients with A1c < 6.5%
- Initiate correction insulin qAC/qHS
- Add glargine 0.1-0.2units/kg if remains high

Steroids

Steroids disproportionately affect _____prandial (mealtime)_____ blood sugars

The ratio of basal:bolus in patients on steroids is: 60/40

What are options for managing steroid-induced hyperglycemia?

Patient not on insulin at home taking 60mg prednisone for COPD exacerbation
- add correctional insulin and escalate to scheduled insulin if needed

Patient on Lantus only at home taking dexamethasone for chemo
- add meal coverage

Patient on basal-bolus regimen already at home on solumedrol for Lupus flare
- increase mealtime insulin

Steroid tapers pose a particular challenge because the steroids dose can change daily.
- Can use a NPH trick. Nph is a roughly 12 hour insulin. Patients on prednisone have higher daytime insulin needs. So, can dose NPH at the same time as prednisone in am and taper down daily as steroid is reduced. (Lantus can be continued)

Type 1 DM

Never HOLD basal insulin in a Type 1 diabetic patient!!!

What is a carb ratio?

What is a sensitivity factor?

Insulin Pumps

- Uses a rapid-acting insulin administered via a pump with a subcutaneous cannula.
- There is a steady amount of rapid-acting insulin infusing continuously which acts as the basal insulin
- When the patient eats they enter in the number of carbs and pump gives a bolus of insulin
- The patient enters their blood sugar into the pump and additional correctional insulin is given. If their blood sugar is below target insulin may be subtracted from the meal dose.
- The site is changed every 3 days. The patient must bring their own supplies to change their site.

Who can use an insulin pump in the hospital?

- Only patients who are willing and able to manage the pump themselves
- Dosing they give themselves must be charted. Use pump order set in Michart so nursing can chart doses
- If pump comes off, need to start insulin immediately!!

Insulin Drips

- An insulin drip is regular insulin that is given via IV
- Treatment of choice for DKA/HHS
- Sometimes used for severe hyperglycemia or patient with rapidly changing insulin needs
- Insulin drip do NOT account for meal coverage. If a patient is eating on an insulin they still need to get subcutaneous insulin with meals
- Most hospitals require an ICU bed to use an insulin drip due to q1 hours blood sugar checks. UMHS is an exception.
- When you stop an insulin drip the insulin wears off very quickly. You generally are going to transition someone from an insulin drip onto a basal-bolus regimen. Remember to give Lantus 2 hours before shutting off someone's insulin drip to avoid escape hyperglycemia.

  To transition off an insulin drip, sum up all the insulin used in the drip over 24 hours. Give 50% of the TDD as basl and overlap with drip by 24 hours. Give 50% of TDD as a bonus. Add a correctional scale.

U500 insulin

What is U-500 insulin?

- Pharmacy needs to approve U-500, endocrine needs to approve

Treatment of Hypoglycemia:

15-15 rule:

Who should be given D50 IV?

Glucagon:

<u>Discharging Your Patient with Diabetes:</u>

- Restart oral agents if appropriate
- Determine if they need insulin and if so, determine types and dosing
- Make sure they have the supplies they need
- Have a plan for follow-up

What supplies might a patient need to manage their diabetes at home?

- Insulin vials/pens 1 vial = 1000units insulin. 1 pen = 300units insulin.
- Needles = 6mm, 31 gauge (if pens use ultrafine or nano pen needles)
- Syringes 30, 50, 100units
- Meter and glucose test strips
- Lancets and lancing device
- Alcohol wipes

Glycemic Management Scenarios

Scenario #1
A 52yo man is admitted to Michigan Medicine for chest pain. He has a history of Type 2 diabetes on metformin 1000mg BID, glipizide XL 10mg per day, and Lantus 90units QHS at home with A1c 8.2%. He is going to be NPO at MN. Please suggest a diabetes regimen for him while NPO.

- Basal: 50% of home dose, no mealtime, correction q6h. Hold home diabetes meds!

Scenario #2
An 86yo woman is admitted to Michigan Medicine for confusion, UTI, and acute on chronic kidney disease. She is on metformin and Lantus at home. On admission her blood sugar is 456mg/dl. You hold the metformin and order her home Lantus dose plus a moderate lispro correctional scale. Nursing gives 12units lispro for the 456. One hour later nursing calls you her sugar is still 321mg/dl. What do you do next?
-Give 4units lispro
-Give 8units lispro
-Give 12units lispro
-**Give nothing, recheck in 3 hours - otherwise would start stacking given lispro duration of action**

Scenario #3

A 44yo man is admitted to Michigan Medicine for pneumonia. He takes metformin and glipizide at home as well as Novolin 70/30 insulin 32units with breakfast and 20units with dinner. His A1c is 7.2% but he sometimes get afternoon lows. You are concerned about using 70/30 in the hospital setting and you want to convert him to Lantus and Lispro. HINT – calculate a total daily dose and split it up 50% basal and 50% bolus

- 80% of home dose is 42
- Then split 50% basal and 50% bolus. Basal is 21 and mealtime bolus is 7 with a moderate sliding scale.

Scenario #4
A 62yo (85kg) women is admitted to Michigan Medicine for hypertensive urgency. She takes metformin, glipizide, pioglitazone, and dulaglitide at home with A1c 7.1%. Non-insulin agents are initially held on admission and she is started on low dose humalog correctional scale but on hospital day #2 she has multiple blood sugar readings >250mg/dl. Please suggest a regimen using 0.3units/kg dosing as an estimate for insulin naïve patient.

- 24 units for daily dose (weight based) -> 23 units basal, 4 units mealtime. low dose correctional scale qAC/qHS. Or go with “Ten of Lantus never hurts”

Scenario #5
A 43yo (65kg) female is admitted to Michigan Medicine for lupus flare. At home she takes Lantus 15units per day plus metformin and glipizide with A1c 7.2%. Her home prednisone dose of 5mg per day is bumped to 60mg on admission. By hospital day #2 her blood sugars are running around 270-280’s on her home Lantus plus she has received 16 units correctional humalog. What are two strategies you could use to optimize glycemic control in the setting of high-dose steroids.

- 4-6 units lispro qAC plus escalate to moderate scale OR add NPH 16units qAM with morning prednisone

ONC EMERGENCIES

Tumor Lysis Syndrome	•
Hypercalcemia of Malignancy	•
Spinal cord compression	•
Superior Vena Cava Syndrome	○
Immunotherapy toxicities	

ANTIBIOTICS OVERVIEW

List 4 broad classes of penicillin antibiotics.

OG penicillin, amino-penicillin, anti-staph penicillin,

What group of beta-lactams will not treat enterococci?

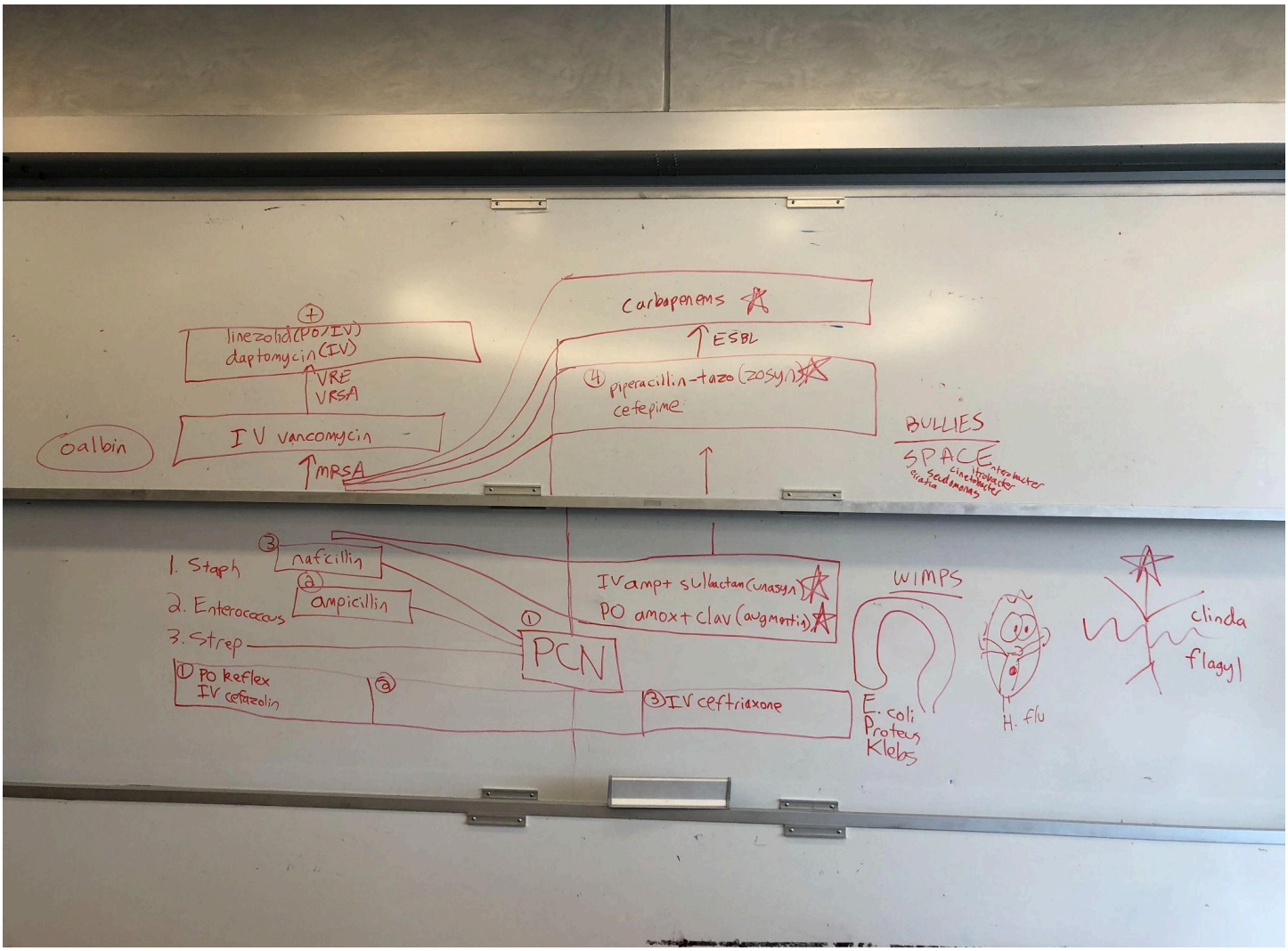
cephalosporins

List the SPACE bugs:

1. Serratia
2. Pseudomonas
3. Acinetobacter
4. Citrobacter
5. Enterobacter

What three organisms are covered by meropenem but not ertapenem?

Acinetobacter, Pseudomonas, and Enterococci



(+)

VRE - linezolid, PO or IV (causes low Plt ~2 wks into tx)
- IV daptomycin (doesn't work in lungs or CNS)

MRSA - IV vancomycin (very renal toxic -> must get peaks and troughs)

1. Staph - anti-staph penicillin (nafcillin)
2. Enterococcus - amino-penicillin (ampicillin/amoxicillin)
3. Strep - OG penicillin

B-lactams: penicillins, cephalosporins, carbapenem

(-)

ESBL - carbapenems (meropenem, ertapenem)
^ also treat 1-3(+), NOT MRSA b/c beta-lactam

BULLIES = healthcare-associated infections
1. Serratia
2. Pseudomonas
3. Acinetobacter
4. Citrobacter
5. Enterobacter

→ extended-spectrum penicillin = piperacillin (+tazo = Zosyn), cefepime
^ also treat 1-3(+)

WIMPS = commensal organisms, part of our microbiome, cause community-acquired infections
1. Gut - enterobacteria (E. coli, Proteus, Klebs)
2. Children - "naso-pharygneal-oto-laryngeal access of doom" (H flu)

→ IV amp + sulbactam (Unasyn)
→ PO amox + clav (Augmentin)
^ also treat 1-3(+)

Cephalosporins
(1st gen) PO Keflex (cefalexin), IV cefazolin (Ancef) → great for staph and strep (SSTI)
(3rd gen) IV ceftriaxone (Rocephin) → treats wimps and strep pneumo (good for CAP)

Anaerobes
Above diaphragm - clindamycin
Below diaphragm - metronidazole (Flagyl)
Anywhere - carbapenems + beta-lactamase penicillins (Zosyn, Augmentin, Unasyn)

NEPHROLOGY: TOP 5 CONSULTS

Serum creatinine is limited, but the best that we have.
- needs to be steady state
- based on muscle mass
- 3 variables matter for calculating eGFR: creatinine, age, sex

1. "False Positive" Elevations:
a. Decreased tubular secretion. **Bactrim**, H2 blockers. Correlate clinically. If mild elevation, wait until off Bactrim and recheck
b. Increased Cr release. Rhabdomyolysis. (Correlate clinically)
c. Increased Cr ingestion. Cooked meat, creatine supplements
d. Lab assay interference. Cefotoxin (very very rare)

Alternatives to SCr (only if suspect inaccuracy of SCr)
- Cystatin C – very muscular, or very cachectic (like in heme-onc where renal dosing is important)
- Timed urine collections for Cr clearance
- GFR measurement (inject tracer, then do serial measurements) [consider in a potential kidney donor]

2. ACEi/ARB initiation or discontinuation
a. Top reasons for clinicians NOT to use (but not always necessary to stop)
    i. Hyperkalemia. If do not want to stop ACE/ARB, can modify diet, can add diuretic (thiazide, loop), and can consider potassium binders
    ii. Rise in serum Cr. Can tolerate up to 30% rise (appropriate physiological effect)
    iii. Held in setting of AKI which is fine, but beware not restarting
    iv. Advanced renal failure. However, never too late to start it and can still have benefit!
    v. When to stop: not ready for dialysis, or not interested in dialysis
b. SGLT2. Also are great, and underused! Can prevent ESRD by 30-40%. Mechanism not understood

3. Hematuria (most are urologic in origin, but not all!)
a. r/o urological disorders
b. Recognize glomerulonephritis (hematuria, HTN, acute renal insufficiency; often there is some proteinuria which points to the filter/glomerulus)
    i. THIS IS A NEPHROLOGY EMERGENCY
    ii. needs a biopsy
    iii. refer to neph
    iv. RED FLAGS: new proteinuria, new or worsening HTN, new or worsening renal insufficiency

4. Secondary causes of HTN
a. Resistant HTN (BP > 140/90 despite 3 meds), young age of onset
    i. renal artery stenosis: intervention is not better than medical management (ACEi/ARBs)
        1. Revascularize if: fibromuscular dysplasia, progressive renal failure, refractory HTN

5. Medication nephrotoxicity
a. Prescribed or OTC or illicit
    i. NSAIDs - increase risk of AKI and CKD. Better to avoid NSAIDs in HTN, CHF, or CKD of all causes
    ii. PPIs - can cause acute interstitial nephritis (AIN). However, very much a risk-benefit decision. "Luck of the draw," not dose-dependent

6. Bonus: always get a urinalysis for CKD/AKI
a. Pyuria/WBC: infectious, AIN
b. Hematuria: GN

HYPONATREMIA

Algorithm for Hyponatremia: What 3 labs do you need?

3 labs needed in hyponatremia workup: serum Osm, urine Osm, urine Na [*ensure patient is not on a diuretic]
Note: most cases are multifactorial

Hyponatremia (Na<135)
- Mid: 130-135
- Moderate: 121-129
- Severe: <=120 --> consider ICU, nephrology consult

Step 1: Acute or symptomatic?
[Na 120s-130s not impressive - you have some time]
Acute = a drop in Na within 48 hours
Symptomatic: altered mental status, seizures [cerebral edema and herniation risk]

If acute/symptomatic --> empirically treat
- Goal to increase Na to stop seizing/less altered
  - Want it to increase by 4-6 mEq (goal is not to necessarily return to normal immediately)
- Rx entails ICU, 100cc 3%NS using peripheral IV over 10 min (can give this a couple times) [*exception to where would want peripheral IV for a hypertonic solution given risk to brain outweighs extravasation risk]

If not -> Step 2

Step 2: Further diagnostics
[Serum Osm = 2{Na} + Glucose/18 + BUN/2.8 + Ethanol/x]
[nl serum Osm ~ 290]

*Na and Glucose are effective osmoles, BUN and ethanol are ineffective (i.e. does not pull water across cell)

Na contributes significantly so if Na decreases, would expect serum Osm to decrease

Question 1: Is this hyperosmolar hyponatremia?

Yes: Osm>280

Etiologies: hyperglycemia (for every 100 increase in Gluc, Na decreases by 1.6), pseudohyponatremia (caused by significant hyperlipidemia, paraproteinemia (ex. multiple myeloma))

No: Osm<280 --> Question 2

Question 2: Is renal water excretion intact?

(If Na is low, body's natural response is to get rid of extra free water)
If yes: Uosm<100-300
Etiologies:
- Primary polydipsia
- Poor Na intake: "Tea and toast" diet, chronic alcohol use

Treatment: Fluid restriction
If no: i.e. Uosm>300 --> Question 3

Question 3: Is there adequate circulating volume?

RAAS involves aldosterone which increase Na reabsorption in DCT of nephron
ADH promotes aquaporin channels for free water reabsorption

If no: RAAS activated, ADH activated
UNa < 20-40 (low)
Etiologies: dehydration or intravascular volume deplete seen in CHF, cirrhosis, CKD
Treatment: Fluids if dehydration, diuresis if CHF/cirrhosis/CKD
If yes: RAAS not activated
UNa>20-40
Etiologies:
SIADH (RAAS is off, ADH through the roof)
- Causes of SIADH: small cell lung cancer, head trauma, pneumonia, SSRIs, MDMA, carbamazepine, cerebral salt wasting, significant stress like surgery [ultimately any pulmonary or brain condition]
- Treatment of SIADH: fluid restriction +/- salt/urea tabs (to cause an osmotic diuresis)

Adrenal insufficiency
Hypothyroidism
Diuretics (loop)

Treatment for hyponatremia (non-acute/asymptomatic)
Risk of correcting too quickly: osmotic demyelination syndrome (central pontine myelinolysis)
- Higher risk if cirrhosis, chronic malnutrition, chronic alcohol use

Goal: correct <6-8 mEq within 24 hours

Acute ONSET (<48hrs)
see above

Acute OFFSET (corrected too fast)
see above

AGITATION APPROACH

Agitation: A state of excessive psychomotor activity accompanied by increased tension and irritability.

Framework to approaching the agitated patient:

Verbal De-Escalation

- Assess Situation & Establish Leadership
- Approach with Empathy
- Accommodate & Ensure Safety
- Set Boundaries & Give Options
- Send for Backup
- Keep Safe Distance & Use Open Body Language
- Know What Your End Goal Is

IF FAILS…

Offer Medication

- Offer PO before IM
- Use minimum effective dose
- Choose medication based on patient profile and agitation etiology

IF FAILS AND VIOLENCE IMMINENT…

Restraints as a Last Resort

- **Restraint = any procedure or device employed to limit a person's mobility**
- Tell patients why they are being restrained and criteria for release
- Restrain for **shortest time** with the **least amount of restriction** and should be **continuously reassessed**
- Document a restraint note
- Debrief afterward; discuss ways to avoid future use of restraints
- Review your hospitals' restraint policy

Helpful **video demonstrating verbal de-escalation** from CU/Denver Health**:** Uses a different framework than AAASSKK however principals are the same

Medical treatment of agitation

For **undifferentiated agitation:** If there is no psychosis event would treat the same as substance intoxication/withdrawal. If there is a psychosis event, treat the same as primary psychiatric disorder.

Medical Illness/ Delirium	Neurologic and Neurocognitive	Substance intoxication & withdrawal	Primary psychiatric disorder
Examples: infection, uremia, metabolic and electrolyte disturbances, medications/poly-pharmacy, ANY critical illness	Examples: dementia, brain tumors, focal seizures, Wilson's	Examples: suspect alcohol or benzodiazepine intoxication or withdrawal, or CNS stimulant use	Examples: schizophrenia, bipolar, MDD, anxiety, personality disorders

• Identify and address underlying medical conditions
• Optimize non-pharmacologic management!
• Choose small doses of less anticholinergic antipsychotics (in order of preference depending on clinical situation and side effect profile):
  • PO olanzapine, PO risperidone
  • Low dose PO haloperidol
  • IM olanzapine or IM ziprasidone
  • IM Haldol (preferred over IV)
• Avoid benzos
	• Choose small doses of less anticholinergic antipsychotics as in medical illness/delirium
	• Risk of EPS significantly higher for patients with parkinsonism as well as lewy body dementia. would avoid Haldol in these circumstances. Prefer quetiapine
	• Optimize non-pharmacologic management!
		• **For ETOH withdrawal and CNS Stimulant use:** Use benzodiazepines (PO, IM, or IV depending on circumstances) for ETOH withdrawal and stimulant intoxication
		• **For CNS depressants** (e.g. ETOH intoxication): haloperidol is often effective
			• Try to anticipate PRN needs in advance
			• Consider psychiatry consultation
			• Order of preference depending on clinical situation and side effect profile:
			  • PO olanzapine,PO risperidone
			  • PO haloperidol **plus benzodiazepine**
			  • IM olanzapine or IM ziprasidone
			  • IM Haldol (preferred over IV) **plus benzodiazepine**

Modified from the following: Wilson, M., Pepper, D., Currier, G., Holloman, G., Feifel, D., 2012. The Psychopharmacology of Agitation: Consensus Statement of the American Association for Emergency Psychiatry Project BETA Psychopharmacology Workgroup. Western Journal of Emergency Medicine 13, 26–34.. doi:10.5811/westjem.2011.9.686

Medication	Dosing Considerations	Notes
Olanzapine (Zyprexa) PO 2.5-10mg (tablets, disintegrating tablets) IM 10mg MAX 30mg /day	Can be used in hepatic or renal failure, but use lower doses (5 mg/day initially)	**Lowest risk of QTc prolongation amongst antipsychotic meds (~6 msec)** Slight inc risk hypotension Do NOT use IM olanzapine with IM/IV benzodiazepines (can cause resp. depression)
Ziprasidone (Geodon) IM: 10mg Q2hrs or 2mg Q4hrs (max 40mg/day)	No renal dose adjustment in renal insufficiency. Cautious with hepatic impairment as drug is extensively metabolized by the liver.	**High risk QTc prolongation - Dose-dependent**
Risperidone (Risperdal) PO 0.5-2mg (tablets, disintegrating tablets, liquid) Not available IM/IV MAX 4mg/day	Do not exceed 0.5 mg BID in pts with moderate/severe renal disease (given clearance reduced by 60%)	Elevated risk of EPS compared with other SGA Moderate risk of hypotension **Minimal anticholinergic side effects** **First line agent in pregnant patients (no known teratogenic effects)** **Prolongs QTc (~10 msec)**
Quetiapine (Seroquel) PO 12.5-100mg BID PRN (tablets, liquid) Not available IM/IV MAX 200mg/day	Use reduced doses in liver disease or renal impairment	**Minimal anticholinergic side effects** **Lowest risk of EPS – good choice for elderly patients, though not generally considered 1st line for agitation** Prolongs QTc (~15 msec)
Haloperidol (Haldol) 1-5 mg PO or IM TID PRN MAX 30mg/day Caution with IV -increased risk arrhythmias	Good choice in pts w/ severe liver disease; use lower doses in renal failure	Elevated risk of EPS (especially in delirious/medically ill patients) Risk of QTc prolongation, especially with IV formulation Bonus: May help with nausea Can give with diphenhydramine OR benzodiazepine to reduce risk of acute dystonia. Typically don't give both at once as this is not likely to give additional benefit and risks more complications *(Ref: Jeffers et al, J Emerg Med, 2022)* **Second-line agent in pregnant patients**
Lorazepam (Ativan) 1-2mg PO, IM, IV **Midazolam (Versed)** 2.5-5mg PO, IM, IV		Benzodiazepine. Paradoxically worsens delirium (except if DTs/ ETOH) Inc risk resp depression, sedation, and confusion. Do not give concomitantly with opioids or IM olanzapine due to higher risk of respiratory depression. **Does not prolong QTc or cause EPS**
Ketamine IM 4-6 mg/kg, IV 1-2mg/kg	Avoid doses lower than 1mg/kg as this may cause dissociative effects without sedation	Used more in Emergency Dept. setting Highly sedating. Can cause respiratory depression and laryngospasm. Emergence reactions (psychotic symptoms as medication wears off) can occur - sometimes given with benzo for this reason **Does not prolong QTc**

References used to help inform table creation:

- Wilson MP, Pepper D, Currier GW, et al: The psychopharmacology of agitation: consensus statement of the American Association for Emergency Psychiatry Project BETA Psychopharmacology Workgroup. West J Emerg Med 2012; 13:26–34
- Garriga M, Pacchiarotti I, Kasper S, et al: Assessment and management of agitation in psychiatry: expert consensus. World J Biol Psychiatry 2016; 17:86–128
- Jeffers, T., Darling, B., Edwards, C., Vadiei, N., 2022. Efficacy of Combination Haloperidol, Lorazepam, and Diphenhydramine vs. Combination Haloperidol and Lorazepam in the Treatment of Acute Agitation: A Multicenter Retrospective Cohort Study. The Journal of Emergency Medicine 62, 516–523.. doi:10.1016/j.jemermed.2022.01.009

Cases and answers to be uploaded later!

Stroke – Acute Management and Work-up
Vignette: 65 yoM, hx of CAD, HTN, BMI 35 admitted to IM at the VA for PNA, develops difficulty talking and weakness after dinner. You are paged to bedside by nursing.

When should acute stroke be in the ddx?
1.) *focal neuro deficit*
2.) *sudden change*

What is the MAIN OBJECTIVE in an ischemic stroke code? Why?

Work-up and Traige for Possible Treatment
#3 Key Questions that define stroke treatment options

1.) last known normal

2.) barriers to treatment (anticoagulation, active bleed, recent surgery, etc.)

3.) symptoms

#Who should be involved when managing acute stroke?
- Neuro (stroke team), RRT

#What labs are needed?
1. POC glucose**
2. CBC (platelets)
3. Coags
4. Creatinine (least important)

Need IV access (contrast)

#What imaging is needed?
- Non-contrast head CT
    - Call CT tech and ask for next available scanner
- CT angio head and neck
    - Need vessel imaging to determine candidacy for neuro IR
    - Consider combining CTA w/ head CT for larger strokes
- CT perfusion (CTP)
    - identify penumbra (salvageable tissue) and infarct core (irreversible damage)

#What vital sign needs special attention?
- Blood pressure
    - Goal for TNK: ≤ 185/110
    - Add drip if needed (nicardipine) or labetalol

Acute Treatment Options:

NIH Stroke Scale (NIHSS) - https://www.mdcalc.com/calc/715/nih-stroke-scale-score-nihss
- 4 points ⇒ TNK
- 6 points ⇒ Neuro IR

#TNK
- NIHSS score of **at least 4**
- TNK contraindications - https://www.mdcalc.com/calc/1934/tpa-contraindications-ischemic-stroke

THROMBOLYSIS CONTRAINDICATIONS BY TYPE*		
Not an Ischemic Stroke or no tissue is salvageable	**Significant Risk of Intracranial Hemorrhage**	**Significant Risk of Systemic Bleeding**
- ICH - SAH - Extensive hypodensity (a completed stroke)	- BP > 185/110 - Bacterial endocarditis - Intra-axial intracranial neoplasms - Unruptured >10mm aneurysm - Intracranial or intraspinal surgery within 3 months	- INR > 1.7 (if on warfarin but INR<=1.7, tPA appears safe and may be beneficial) - Treatment dose of LMWH w/in 24 hours - Current use of direct thrombin inhibitor or factor Xa inhibitors with a dose w/in 48 hours - Plt count <100K - Aortic dissection - GI or GU hemorrhage within 21 days - Elevated PTT
RELATIVE CONTRAINDICATIONS		
- Seizure at onset and high concern that deficits are post-ictal - Blood glucose <50 or >400 mg/dL	- Other intracranial vascular abnormalities	- Major surgery or trauma within previous 14 days - Pregnancy

-
- TNK is 1 dose
- Treatment window: within 4.5 hours of symptom onset
    - more effective earlier

#NeuroIR
- NIHSS score of **at least 6**
- Potential treatment window: up to 24 hours
    - Coagulopathy is not a contraindication to neuro IR

Stroke level of care or ICU care w/ frequent monitoring

Subsequent Work-up and Secondary Prevention of Ischemic Stroke

#Post-TNK consideration

#General Medications and Labs

- Baby aspirin and high intensity statin
- Lipid panel and A1c
- Echo (look for LA dilation, MV disease)
    - If < 60 yo, look for PFOs
    - PFOs are not closed for patients > 60.
- Vessel imaging (CTA or MRA)
    - Symptomatic carotid stenosis 70-99% → urgent surgery (carotid endarterectomy or stenting)
    - Asymptomatic carotid stenosis >70% → consider surgery though less urgent. benefit uncertain
- PT, OT speech and language

#Special Considerations on Specific Categories/Mechanisms of Ischemic Stroke

SEIZURE IN THE SICK INPATIENT

Mrs. Jones is a 71 yoF admitted for PNA to IM. 4 days of productive cough, new 4L oxygen requirement, intermittently febrile, admitted overnight, now on ceftriaxone. Overnight labs reveal WBC 15, Cr 1.5 up from 0.8. CXR with focal opacity in left lobe c/w PNA. You are paged to bedside by RN "please come assess, patient is having seizure"

#Consideration on Lorazepam for Acute Seizure
-If patient is in status epilepticus (2.5-3 mins of seizing can count as this, not always 5 mins), give lorazepam
-If patient is actively seizing, give lorazepam
-If patient has multiple seizures in a day, consider giving Ativan
-Consider drug risks:
    -Respiratory depression
    -Impaired mentation (risk of not being able to protect airway)

#Are They Still Seizing?
-Not seizing if they don't have rhythmic motor activity OR if they are having conscious responses to external stimuli (movement, speech, etc)

#Should I Order Head Imaging?
-Order urgent non-contact Head CT if first seizure of life, especially if patient not recovering quickly
-MRI Brain if patient has suspected metastatic cancer or bacteremic
    -Look out for structural abnormalities, bleed, etc. i.e. acute dangerous issues
    -No need to order if other overt risk (like hypoglycemia)
-No need to order if patient has had seizures before

#Epilepsy vs Medically Provoked Seizure?
-Infection, hypoxia, electrolyte issues, trauma, sleep issues, substance use, hypoglycemia all lower seizure threshold and can be synergistic/additive in nature
    -This is NOT epilepsy. Medically provoked seizure, even if repeat seizures

#Should I Order an EEG?
-If trying to differentiate traditional seizure vs. PNES vs. something else, i.e. if unsure
-EEG can capture changes from previous seizure EVEN IF NOT actively seizing
-No need if pretty sure this is a provoked seizure (in this case, pretty sure this was medically provoked)

#Should I Start a Seizure Medication?
-If medical provoked seizure, consider Levetiracetam if treatment of provoking factors will take time ("bridging" treatment): to avoid falls, aspiration, etc.
-Consider side effects of levetiracetam (Keppra): better than Ativan
    -Not sedating
    -Agitation (Keppra rage)
-Give as loading dose + maintenance dose:

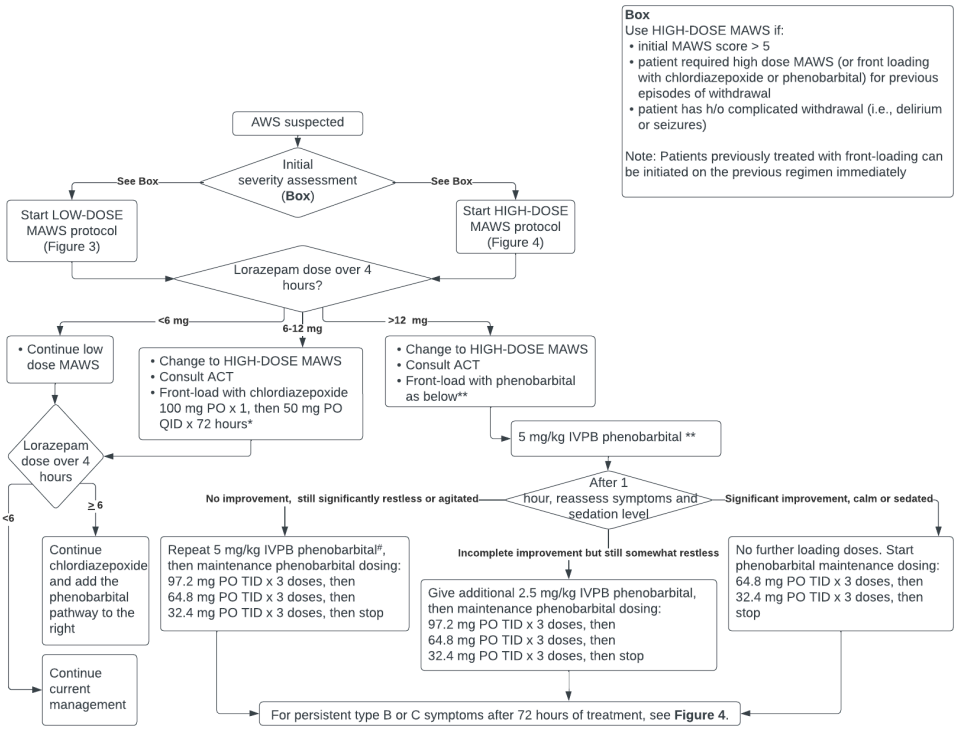
#Can They Drive?
-NO. NO DRIVING W ANY TYPE OF SEIZURE IN MICHIGAN. Pay attention to state law where you are.
#If Time…. PNES
-N/A

ALCOHOL USE DISORDER AND WITHDRAWAL

1. Prevalence, Risks, and Consequences of Alcohol Use
    a. Less than 10% of individuals with AUD receive any treatment in the past year
    b. COVID-19 led to increased rates of alcohol use and heavy alcohol use that started in 2020 and extended beyond into 2022
    c. More adolescents use alcohol compared to tobacco, nicotine, vaping, marijuana
    d. But alcohol use in adolescents may be down trending according to Michigan study
    e. Alcohol causes organ damage with >20K deaths from alcohol-related cancer per year
2. Definitions of Alcohol Use
    a. Dietary guidelines is 1 drink or less per day for women and 2 drinks or less per day for men
    b. Blood alcohol concentration is determined by how much and how quick a person drinks and body's rates of alcohol absorption, distribution, and metabolism
    c. BAC of 0.08% is federal legal limit with typical adult reaching this BAC after consuming 4 or more drinks in women or 5 or more drinks in me in about 2 hours
3. Diagnosis of Alcohol Use Disorder
    a. AUDIT-C is screening tool to detect substance use disorder
    b. Diagnosis of AUD is based on DSM guidelines
    c. Four Cs of addiction include compulsion, craving, consequences, and control (or lack thereof)
4. Mechanism of Alcohol
    a. Alcohol binds to GABA-A receptors (inhibitory)
    b. Alcohol blocks glutamate receptors via NMDA receptor (excitatory)
    c. This causes down regulation of inhibitory receptors and up regulation of excitatory receptors
    d. During withdrawal these compensatory changes are no longer opposed by alcohol
5. Alcohol Withdrawal
    a. Amount used, chronicity of use, and time since last use impact withdrawal
    b. Minor symptoms include autonomic and motor symptoms including tachycardia, tachypnea, diaphoresis, tremor
    c. Moderate symptoms include psychiatric symptoms including hallucinations
    d. Severe symptoms include psychiatric symptoms and delirium tremens
    e. Delirium tremens peaks around 72-96 hours
    f. Prediction of alcohol withdrawal severity is done using PAWSS which includes patient interview questions and clinical evidence (according to this JAMA study history of DT and SBP>140 were more likely to result in severe alcohol withdrawal)
6. Testing for Alcohol Use
    a. Direct tests: BAC, PEth, EtG or EtS (both urine)
    b. Indirect tests: CDT (good marker of relapse)
    c. General workup labs: CMP, CBC, nutritional deficiencies (B12, folate), hepatitis panel, HIB, TB, pregnancy test

d. PEth tests for abnormal phospholipid that is formed only in the presence of ethanol with high specificity and sensitivity (detectable for 3-4 weeks of daily intake of 50g ethanol and up to approximately 2 weeks after ceasing alcohol intake)

7. Supportive Care for Alcohol Use Disorder
    a. Replete electrolytes (Mg, K, phos, Ca)
    b. Correct nutritional deficiencies (B1, folate, B12)
    c. Thiamine to prevent Wernicke's encephalopathy (lack of consensus on dosing but instructor orders 250mg IV daily for 3-5 days for prophylaxis and 500mg IV TID for 3-5 days for suspected)
8. Management of Alcohol Withdrawal
    a. Benzodiazepines are the most extensively studied class
    b. Only benzodiazepines show consistent efficacy across outcomes
    c. Too few trials comparing efficacy and safety of benzodiazepines to other agents like phenobarbital and ketamine
    d. Benzodiazepines bind to GABA-A receptors and increase the frequency of Cl- channel opening but requires endogenous GABA
    e. Phenobarbital binds to GABA-A receptors and prolongs the duration of Cl- channel opening but does not require endogenous GABA (also binds to NMDA receptors)
    f. Lorazepam, oxazepam, and temazepam are safe to use in liver disease
9. MAWS (at Michigan), CIWA (elsewhere)
    a. Scale which includes type A symptoms (CNS excitation), type B symptoms (adrenergic hypersensitivity), and type C symptoms (delirium) scored from 0-10
    b. There is low dose protocol that gives Ativan 1mg for MAWS=1-3 and Ativan 2mg for MAWS>3 OR high dose protocol that gives Ativan 2mg for MAWS=1-6 and Ativan 4mg for MAWS>6



PSYCHIATRIC EMERGENCIES

Lecture Outline:

1. Introduction

- Overview of common psychiatric emergencies and basic management principles
- Challenges in diagnosing psychiatric conditions in medically ill patients (and vice versa)
- The role of Consultation-Liaison (CL) Psychiatry

2. Common Psychiatric Emergencies in Medical Settings

- **Suicidal Ideation and Behavior**:
    - Risk assessment: clinical evaluation and available tools
    - Safety planning and next steps
- **Psychosis/Mania**
    - Assessment and differential diagnosis
    - Management
- **Catatonia**:
    - Recognition and differentiation from other conditions.
    - Treatment approach
- **Substance Withdrawal Syndromes**:
    - Alcohol, benzodiazepines, and opioids.
    - Management protocols (e.g. MAWS/CIWA, COWS)
- **Mood and Anxiety Disorders**:
    - Depression, panic attacks and acute anxiety in medical settings.
    - Short-term interventions and referral to psychiatry
- **NMS and Serotonin Syndrome**
    - Differences and similarities, differential diagnosis
    - Assessment and work-up
    - Management
- **Legal and Ethical Considerations**:
    - Capacity assessment
    - Leaving AMA and patient rights

3. Case-Based Discussion

- 2 cases
- Discussion about diagnosis, workup, and management.

4. When to Consult Psychiatry

- Indications and how to communicate with the team

4. Q&A and Wrap-Up

CODE STATUS DISCUSSIONS

- *Goals of Care: Understanding expectations of hospitalization*
- *Code Status: what would you like to be done in this case*
    - *“If your heart were to stop”*
    - *“If you were to die in the hospital what would you like…”*
    - *“What would your wishes be if…”*

1. *Normalize discussion - talk to every patient about this!*
2. *Align with them - My goal is to learn what is important to you so I can provide care aligned to your wishes*
3. *Gather Information - what do you enjoy doing outside of the hospital? What does your day to day look like?*
4. *Summarize - “It sounds like” your QOL has been acceptable to you…*

- *If overwhelming, take break/pause and ask them if they want to continue discussing it*
- *Can communicate risks and benefits of CPR - “given what we know about your illness and comorbidities doing chest compressions and putting them on a breathing machine would do more harm than good”*
    - *If many comorbidities cna phrase it as “allow a natural death”*
- *Most patients default to full code*
- *You can make recommendations - “this doesn't mean we won't treat what brought you to the hospital”*
- *“Are you ok with a prolonged road to recovery?”*

SPIKES - for serious/bad news but also works well for code discussions!
S - Setting
P - Perception of their health and their goals
I - Invite patient to share what they know about CPR
K - Knowledge - provide info in small pieces
E - Empathy - recognize their emotions
S - Summarize - go over plan and what you've discussed

**** MDCALC GO-FAR (Good outcome following attempted resuscitation) ****

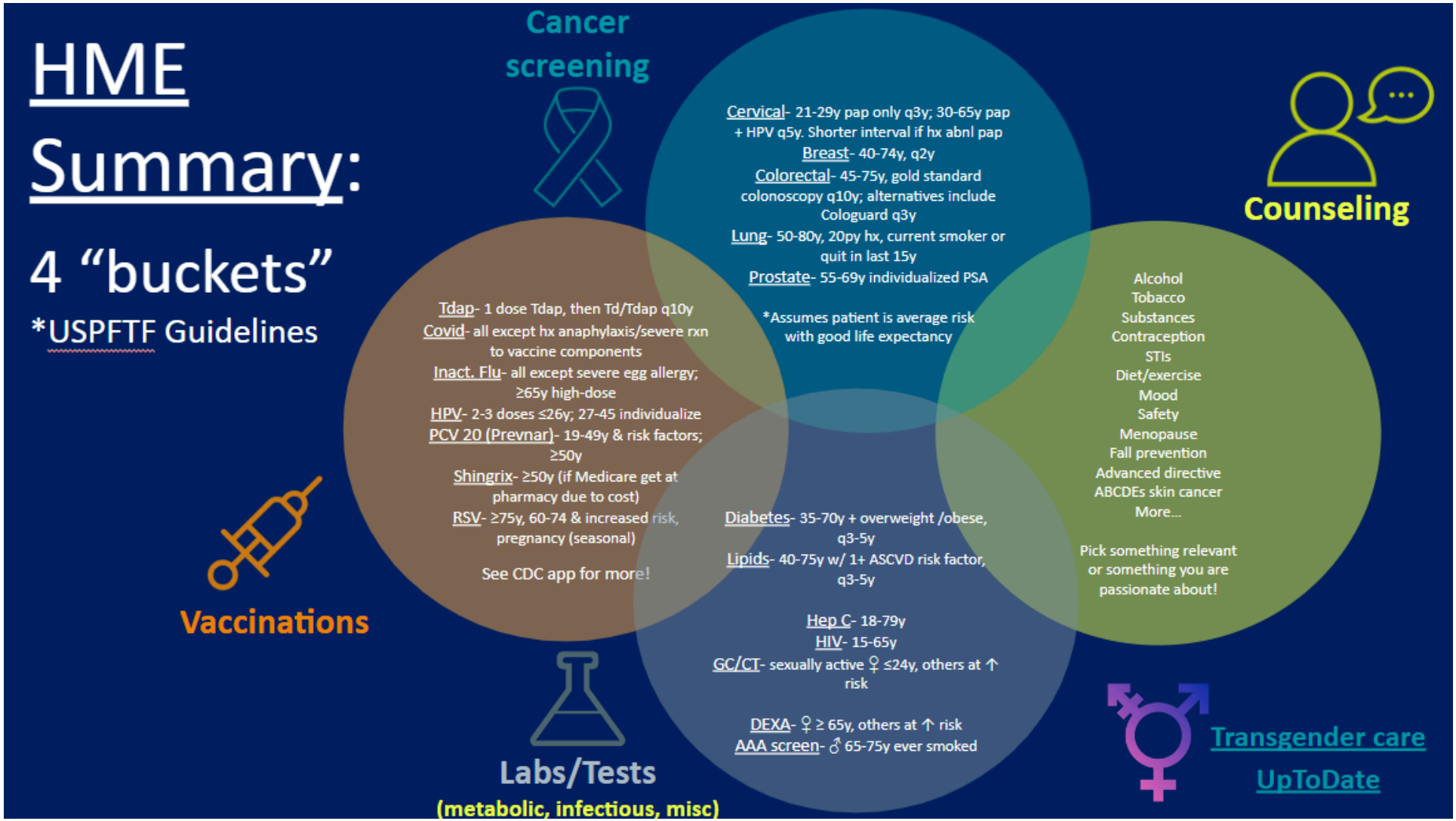
PATIENT-CONTROLLED ANALGESIA

Content will be added just prior to session

OUTPATIENT

4 "Buckets" of USPFTF Guidelines

Link to small group activity (summarized below)



- **What is an HME?**
  - A visit focused on preventative care that can help save lives; it does not have to be annual
  - Younger patients can come every few years, whereas older patients usually come annually, which may help build rapport
  - Medicare coverage: You do not have to do a physical exam but still useful to do so

- Focus on high yield physical exams for every HME (heart, lungs, abdomen, check for edema, check ears, neck)

Helpful apps/websites

- Vaccine schedule: https://www.cdc.gov/vaccines/hcp/imz-schedules/index.html; Pneumococcal Vaccine: https://www.nfid.org/protecting-adults-against-pneumococcal-disease/
- Cancer screening (USPSTF): https://www.uspreventiveservicestaskforce.org/uspstf/search_results?searchterm=cancer%20screening%20
- Labs and imaging (USPSTF): https://www.uspreventiveservicestaskforce.org/uspstf/recommendation-topics/uspstf-a-and-b-recommendations
- ASCCP Pap App (follow routine and abnormal pat results, but it is not a free app; website is).
- MD calc (good for inpatient calculation of ASCVD risk for instance): https://www.mdcalc.com/
- Caution with AI: It quickly generates a list of suggested screening tests. It works better if you clarify where you want screening guidelines from. It is not updated for the newest screening recommendations (ie 2024 USPSTF for breast cancer starting at age 50 is not there yet, and it may cite old references

Strategies to help with efficiency in the clinic

Before the visit

- Develop a framework ie based on age, organ system, type of screening
- Electronic chart tools can be helpful (OPAs)

- Be prepared for commonly asked questions - ie what tests should I do for colon cancer screening? // develop a script!
- Apply the sample principles you use for efficiency on inpatient, ie pre-charting = pre-rounding for outpatient visits
  - Start a note with template
  - Choose one preventative health issue to address if time
- Run the list with your team (ie MA, RN, preceptor)
  - What can be done before you step into the room?
  - Ask clarification questions that don't need to wait until you see the patient

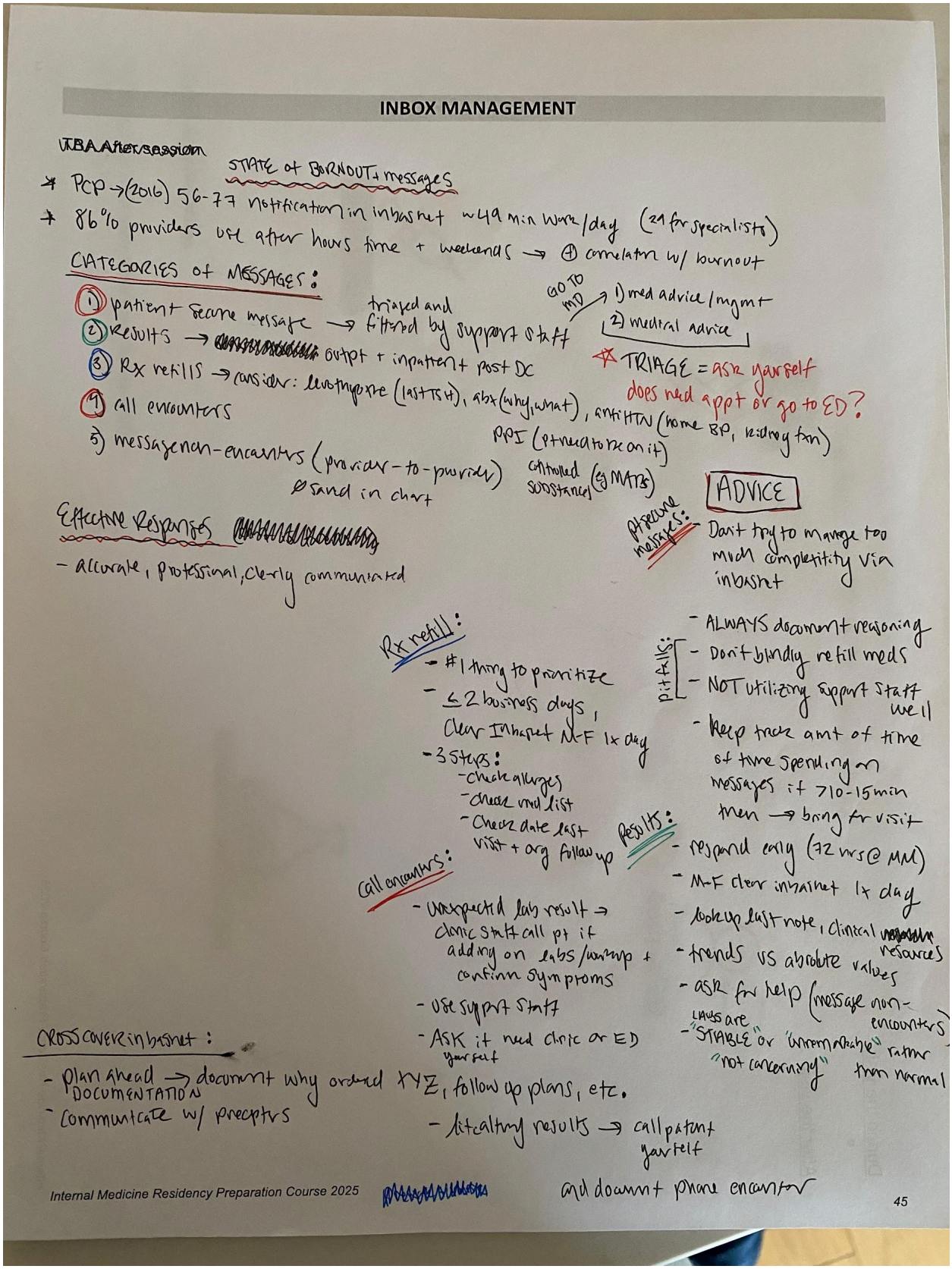
During the visit

- Acute concerns take precedence during a patient visit. You can switch from an HME to a problem-focused visit and bill for a problem-focused visit
- Set an agenda early (for every visit, not just HME)
  - Ask what the patient wants to talk about
  - Negotiate as needed for time, and don't be afraid to set up a follow-up visit
- Stick the dismount
  - "I'm going to place the orders, then we'll review the plan, and then you'll be all set for the day."
  - Don't forget to do teach-back! ("Tell me in your words what your understanding is for the plan from today.")
  - "What questions do you have about what we discussed today?"

- Regarding counseling, pick a topic that is impacting health in multiple ways (ie smoking, something age-related, or a topic you are passionate about to help engage the patient

<u>After the visit</u>

- Finish notes asap
    - Consider dictation to improve efficiency
- Write a to-do list for the next visit at the end of the note


INBOX MANAGEMENT
TBA After session
STATE of BURNOUT + messages
PCP → (2016) 56-77 notification in inbasket w 49 min work/day (21 for specialists)
86% providers use after hours time + weekends → correlation w/ burnout
CATEGORIES of MESSAGES:
1) patient secure message → triaged and filtered by support staff
2) Results → outpt + inpatient + post DC
3) Rx refills → consider: last TSH, abx (why, what), antiHTN (home BP, kidney fxn), PPI (pt need to be on it), controlled substances (eg MATS)
4) all encounters
5) message non-encounters (provider-to-provider) saved in chart
go to MD → 1) med advice/mgmt 2) medical advice
TRIAGE = ask yourself does need appt or go to ED?
ADVICE
Effective Responses
- accurate, professional, clearly communicated
proactive messages:
- Don't try to manage too much complexity via inbasket
Rx refill:
- #1 thing to prioritize
- ≤ 2 business days, clear Inbasket M-F 1x day
- 3 steps:
-Check allergies
-Check med list
-Check date last visit + arg follow up
pitfalls:
- ALWAYS document reasoning
- Don't blindly refill meds
- NOT utilizing support staff well
- keep track amt of time of time spending on messages if >10-15 min then → bring for visit
Call encounters:
- unexpected lab result → clinic staff call pt if adding on labs/imaging + confirm symptoms
- use support staff
- ASK if need clinic or ED yourself
Results:
- respond early (72 hrs @ MM)
- M-F clear inbasket 1x day
- look up last note, clinical resources
- trends vs absolute values
- ask for help (message non-encounters)
- Labs are "STABLE" or "unremarkable" rather than normal "not concerning"
CROSS COVER inbasket:
- plan ahead → document why ordered XYZ, follow up plans, etc. DOCUMENTATION
- communicate w/ preceptrs
- critical/any results → call patient yourself and document phone encounter
Internal Medicine Residency Preparation Course 2025
45

10 Inbox Management Highlights/Pearls

1. Check your Inbox at least once per day Monday-Friday

2. Respond to abnormal results at least within 72 hours, refill requests at least within 2 business days.

3. DynaMed is a great resource for outpatient medicine—use it if you cannot find a good answer to work up an abnormal lab on UpToDate.

4. Three steps to always take before refilling a medication:

    - Check allergies
    - Check medication list
    - Check date of last visit and organize follow up

5. Use "call encounters" to delegate inbox workload (i.e. nursing staff to call patients with results)

6. Always ask yourself if a clinic appointment or emergency evaluation is indicated before providing remote care.

7. For life-altering results (i.e. new cancer diagnosis), call the patient yourself and document your conversation as a "phone encounter."

8. Use "Message (Non-Encounter)" to discuss basic questions with your preceptor.

9. Make sure your responses to patient messages are accurate, professional and have a clear plan outlined—this will save you time later and make life easier for your patients.

10. Many clinics have different support staff available (dieticians, social workers, pharmacists, etc)—ask your preceptor what clinic resources are available and utilize them when indicated.

Example Responses to Sample Patient Messages (There are many appropriate responses—just a few ideas)

Atrial Fibrillation Case:

Hello Mr. ZW,

I am sorry that you are having difficulty with refilling your apixaban! If the cost is too high, I can offer you another blood thinner called "warfarin". You will have to follow a special diet while on this medication and get your labs checked regularly; however, it is much cheaper than the blood thinner you are currently taking and works very well to prevent strokes. I will have our pharmacy team call you to go over this medication in more detail.

In the meantime, please do not participate in kickboxing or any other contact-sports, because these sports will increase your risk of bleeding, Please go to the emergency department if you hit your head or notice blood in your stool—with your blood thinner, this could be life-threatening. You could try running or yoga instead if you are interested in staying active!

Take care,

Dr. Lemon

Levothyroxine Refill Case→Nursing Staff Message:

BF has had a persistently elevated TSH on two checks this year, and she has missed her annual appointment. I will increase her levothyroxine to 100mcg daily and order repeat TSH to be drawn in 6 weeks. She should go to the emergency department if she has severe lethargy, unintended loss of consciousness, or confusion. Could you call her to see how her symptoms are, inform her of the above plan, and assist with re-scheduling her annual appointment?

Thank you,

Dr. Lemon

Levothyroxine Refill Case→Direct Patient Message:

Hello Mrs. BF,

It looks like your thyroid lab is still a bit too high, which means you are not getting enough of your thyroid medication (levothyroxine). I have increased the dose of your levothyroxine to 100mcg daily—please pick this up from your pharmacy as soon as you are able. I will also order another thyroid lab, which you can have drawn 6 weeks after you start the new dose. When you are able, please re-schedule your annual appointment too so I can see you in-person. If you feel extremely tired (cannot do your daily activities), are passing out, or feel confused--please call an ambulance to take you to the emergency department, because these symptoms may be because of your thyroid.

Could you also message me back to say how you are feeling now?

Thank you,

Dr. Lemon

WEIGHT: INCLUSIVITY, STIGMA & APPROACHES TO TREATMENT

Why care about weight stigma?

-

Examples of weight stigma in healthcare:

-

Actions to reduce weight stigma:

-

Best practices for discussing behavioral changes with patients

-

Key takeaways when discussing medications for weight loss with patients

-

Medications FDA approved for long-term use for weight loss

PINK = discussed in talk; **BLUE** = additional information on common medications for your knowledge

Medication	Mechanism	Anticipated Weight Loss	Side Effects and Contraindications	Other Considerations
Tirzepatide (Zepbound) (FDA approved 2023)				
GLP-1-RAs: focus on **semaglutide (Wegovy)** (2021)	Glucagon-like peptide-1 receptor agonist	15% weight loss	- Similar to Zepbound	- Similar to Zepbound - Also treats T2DM, HFpEF, used off-label to treat MASLD - Other benefits: reduces cardiovascular disease in patients with AND without T2DM
Phentermine-topiramate (Qsymia) (2012)	Stimulant + enhanced GABA activity	8-10%	- Paresthesias, change in taste, dry mouth, constipation, insomnia, anxiety - Contraindications: hyperthyroid, glaucoma, pregnancy (teratogenic)	- Oral medication - Potential for misuse - $$ (can prescribe components separately off-label) - May help with headache prevention - Taper if stopping medication to avoid seizures
Bupropion-naltrexone (Contrave) (2014)	Appetite suppressant	5-6%	- Nausea, dizziness, changes in bowel habits, insomnia, HA, dry mouth - Contraindications: seizures, eating disorder, uncontrolled HTN, opioid use, MAOI use, pregnancy	- Oral medication - $$ (can prescribe components separately off-label) - May help with smoking cessation, alcohol intake, mood

Key takeaways when discussing bariatric surgery options with patients

-

Bariatric Surgery

Procedure	Mechanism	Outcomes	Adverse Effects
Roux-en-Y gastric bypass 	Restricts food intake, causes partial malabsorption, changes appetite regulating hormones, changes bile acid and gut microbiota	- 31% weight loss at 1 year - 26% weight loss at 5 years - For patients w/ T2DM: 29% with A1c <6%	- Major 30 day adverse events: 5% - Late adverse effects: micronutrient deficiencies, dumping syndrome, anastomotic ulcers, cholelithiasis, nephrolithiasis, abdominal pain
Gastric sleeve 	Restricts food intake, may have benefit from removing endocrine-rich gastric tissue	- 25% weight loss at 1 year - 19% weight loss at 5 years - 23% with A1c <6%	- Major 30 day adverse events: 2.6% - Late adverse effects: possible micronutrient deficiencies, sleeve stenosis, cholelithiasis

Arterburb et al. Annals of Internal Medicine, 2018; Schauer et al. NEJM, 2017.

Key citations:

1. Phelan SM, Burgess DJ, Yeazel MW, Hellerstedt WL, Griffin JM, van Ryn M. Impact of weight bias and stigma on quality of care and outcomes for patients with obesity. Obes Rev. 2015;16(4):319-326. doi:10.1111/obr.12266
2. Nutter S, Russell-Mayhew S, Alberga AS, et al. Positioning of Weight Bias: Moving towards Social Justice. J Obes. 2016;2016:3753650. doi:10.1155/2016/3753650

OUTPATIENT CHRONIC DISEASE MANAGEMENT

BP goals (per 2017 AHA/ACC guidelines)

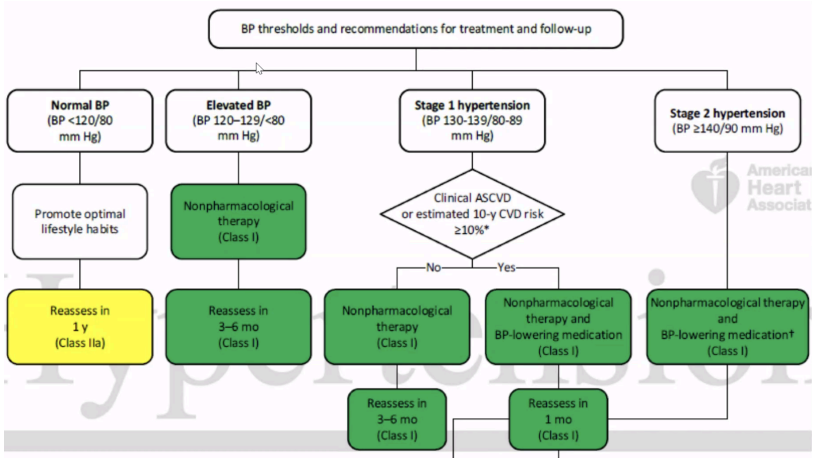
- Existing vascular disease (PAD, prior MI/stroke, etc) or ascvd >10% — <130/80
- Everyone else — <140/90

Choosing initial antihypertensive

1. >20/10 over goal? -> Combo agent (usually CCB + ACEi/ARB)
2. Specific indication for ACEi/ARB (CKD, DM with proteinuria, HF)?
3. High concern side effects of ACEi/diuretic (very frail elderly, poor follow-up)? -> Consider CCB
4. If no to the above, start with chlorthalidone 12.5mg (ALLHAT trial)

Hypertension: 2017 ACC/AHA guidelines

- **Diagnosis**
  - 1) >180/120 any visit, 2) average home readings >130/80 over 10 readings in a week, 3) average clinic >130/80 over at least 3 visits
- **When to start treatment** (see flow sheet) SPRINT trial = more benefit to lower BP target if higher risk
  - >140/80 start meds
  - 130-139/80-89 start meds depending on ASCVD



  - PREVENT new alternative to ASCVD that tends to predict lower risk
- **Treatment**
  - Non-pharmacologic:
    - 1) low salt diet (<2000 mg Na/day -5/2 mmHG), 2) DASH diet (-6/4 mmHg), 3) weight loss (/1mmHG/kg)
  - Medications:
    - >20/10 over goal? → combo agent with CCB + ACEi/ARB
    - Special indication for ACEi/ARB? → CKD, DM with proteinuria, HF
    - High concern for side effet of ACEi/aiuretic? → CCB
    - If no to above, consider starting with chlorthalidone 12.5mg (ALLHAT trial)

Class	Starting Dose	Side Effect	Specific Indications	Notes
Calcium channel blocker	Amlodipine 5mg	Leg swelling	-	-
ACE inhibitor	Lisinopril 5mg	Hyperkalemia, cough	Heart failure, chronic kidney disease, diabetic w/ proteinuria	Check BMP 7-14 days after starting
Thiazide diuretic	Chlorthalidone 12.5mg	Renal failure, dehydration	-	Check BMP 7-14 days after starting

- **Descalation of medications:** think about descalation 20-30 mmHG below goal with monitoring

Diabetes: goal A1C <7, someites goal <8 frail, limited life expectancy patients

- **Medications**

Treatment Class	Most common agent	Starting dose	Max dose	Contraindications	Side effects	Decrease in a1c
Diet and exercise		-	-	-	-	0-3%
Biguanide	Metformin	500mg daily	1000mg twice daily	eGFR < 30	Diarrhea, nausea	1.5%
GLP-1 Agonists	Exenatide	2mg weekly (SQ)	2mg weekly (SQ)	History of medullary thyroid cancer, CrCl < 30	Nausea	1%
SGLT-2 Inhibitor	Empagliflozin	10mg daily	25mg daily	ESRD	Yeast infxn, UTI	1%
Sulfonylurea	Glipizide	5mg daily	10mg twice daily	-	Hypoglycemia, weight gain	1.5%
Thiazolidinediones	Pioglitazone	15mg daily	45mg once daily	Class III/IV heart failure, severe liver disease	Hypoglycemia, edema	1%
DPP-4 Inhibitor	Sitagliptin	100mg daily	100mg daily	-	Hypoglycemia	1%

- **Treatment steps**

- 1: Lifestyle (weight loss)
- 2: Metformin (titrated up to 2000mg daily as tolerated)
- 3: Existing vascular disease + (CKD or CHF)? -> SGLT-2
- 4: Existing vascular disease without CKD/CHF -> SGLT-2 or GLP-1
- 5: No existing vascular disease: Sulfonylurea or SGLT-2 or GLP-1 or other

- Logistics & Counselling for starting insulin
  - Long-acting or NPH, depending on insurance
  - Counsel on hypoglycemia and sx
  - Needles, glucometer, test strips
  - Fasting AM blood glucose
  - Insulin injection chart
- Notes about starting/managing insulin
  - GOAL <140 fasting blood glucose
  - Think insulin initially A1C >10, can also start metformin
  - Consider CGM for patients on multiple daily doses of insulin
  - If need post-prandial insulin, likely start with one meal

Unipolar Major Depression- Diagnosis

- DSM-V definition:
  - At least two weeks of at least 5 of:

Must have at least one of these

- **Depressed mood** most of the day, nearly every day.
- **Markedly diminished interest or pleasure** in all, or almost all, activities most of the day, nearly every day.
- Significant weight loss when not dieting or weight gain, or **decrease or increase in appetite** nearly every day.
- **Insomnia or hypersomnia** nearly every day
- **A slowing down of thought and a reduction of physical movement** (observable by others, not merely subjective feelings of restlessness or being slowed down).
- **Fatigue** or loss of energy nearly every day.
- Feelings of **worthlessness or excessive or inappropriate guilt** nearly every day.
- **Diminished ability to think or concentrate**, or indecisiveness, nearly every day.
- Recurrent thoughts of death, **recurrent suicidal ideation** without a specific plan, or a suicide attempt or a specific plan for committing suicide.

Generalized Anxiety Disorder- Diagnosis

DSM-V definition:

- Excessive anxiety and worry occurring more days than not for at least six months, about a number of events or activities
- The individual finds it difficult to control the worry.
- The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past six months):
- •1. Restlessness or feeling keyed up or on edge
- •2. Being easily fatigued
- •3. Difficulty concentrating or mind going blank
- •4. Irritability
- •5. Muscle tension
- •6. Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep)

OUTPATIENT RESPIRATORY INFECTIONS ***see comments for further notes

OTC Symptomatic Treatment for URI's

INGREDIENT	EFFECT	MAX DOSE	Trade name
Dextromethorphan	Cough suppressant	1200mg / day	Delsym, Robitussin
Guaifenesin	Expectorant/mucolytic	2400mg / day	Mucinex
Ibuprofen	Pain reliever	3200mg / day	Advil, Motrin
Acetaminophen	Pain reliever	4000mg / day	Tylenol
Phenylephrine	Decongestant	60mg / day	Sudafed PE
Pseudoephedrine	Decongestant	240mg / day	Sudafed
Codeine w/ guaifenesin	Cough suppressant and mucolytic	120mg codeine / day	Cheratussin AC
Codeine w/ promethazine	Cough suppressant and antihistamine	120 mg codeine / day	?
Benzonatate	Cough suppressant	600mg / day	Tessalon Perles

*Nasal sprays- steroid (nasacort, flonase) for post-nasal drip and congestion; decongestant (Afrin) to help with nasal congestion, don't use more than 3 days given rebound rhinorrhea; saline (helps with congestion)
**Limited or no benefit= oral antihistamines, antibiotics, zinc (some report reduces duration of sx)??
***Counsel patients on how many combo meds might include Tylenol, should be mindful to avoid >3-4g APAP per day

Common Combination meds:
Dayquil = Acetaminophen + dextromethorphan + phenylephrine
Nyquil = Acetaminophen + dextromethorphan + doxylamine (antihistamine/mild sedative)
Mucinex DM = Guaifenesin + dextromethorphan **Dr. Walford likes this one bc less "stuff" in there
Coricidin HBP Cough and congestion = Guaifenesin + dextromethorphan
Mucinex D = Guaifenesin + pseudoephederine
Tylenol Cold and Flu Severe = Guaifenesin + dextromethorphan + acetaminophen + phenylephrine

Take home points:
-hypoxia, labored breathing, high suspicion for MI/PE most common reasons to send pt with COVID-19/URI to ED; look for conditions that require antibx (AOM, PNA, bacterial sinusitis, strep pharyngitis, AECOPD); dextromethorphan + guaifenesin have shown most benefit for common cold

OUTPATIENT MUSCULOSKELETAL CONCERNS

Focus on the Knee and Shoulder exam
Recommended resources or reading:

- Orthobullets
- AAFP
- U of M Videos:
    - https://open.umich.edu/find/open-educational-resources/medical/musculoskeletal-m1
    - NEJM videos

Musculoskeletal complaints:

- it is important to take a detailed history, including detailed mechanism, time frame, previous injuries and treatment plans, provoking activities and also what improves symptoms as well as what sports/activities patient participates in
    - important to inquire about rheumatologic symptoms or history (family and personal), STIs (Reiter's), infection, gout
    - For knee specifically as about mechanical symptoms (locking, catching), falls, swelling
    - For shoulder specifically ask 'handedness", difficulty with range of motion
- Important to understand patient goals:
    - for athletes what sport, time of year, etc
    - for older individuals what activities they participate in, work requirements, etc

Exam:

- Palpation, ROM, strength testing
- Special tests
    - Knee
        - Meniscal: Thessaly is better than McMurray testing. Can also do Apley grind
        - ACL: Lachman is better than Anterior and Posterior drawer
        - Collateral: Valgus and varus stress
    - Shoulder
        - Neer's
        - Hawkin's
        - Scapula- evaluate movement and rhythm, scapula retraction and assistance test
        - Apprehension/Relocation testing
- Joint above and below
    - Hip for the knee: int/ext ROM
    - Neck for the shoulder: ROM, spurlings, intrinsic strength testing

Imaging:

- Knee
    - Ottawa knee rules
    - Views: Standing AP and lateral, merchant, notch
    - Indications for MRI
        - ACL tear, collateral ligament injury, meniscus injury (not degenerative), cartilage/chondral injury/lesion, insufficiency fracture

- Shoulder
    - Views: Grashey, internal/external, axillary (scapula Y if unable to abduct)
    - Indications for advanced imaging
        - US: Rotator cuff tendinopathy or tear (not massive), biceps tendinopathy or tear
        - MRI: massive rotator cuff tear, occult fracture

        - MRA: Dislocation. should only be ordered by specialist

Differential Diagnosis: can be delineated by age, location, injury or exam finding.

- Knee:
    - Anterior knee pain (PFPS, patella subluxation, patella tendionpathy, quad tendinopathy)
    - Medial knee pain (meniscus, MCL, pes anserine bursitis, OA, insufficiency fracture)
    - Lateral knee pain (meniscus, LCL, ITB, OA, insufficiency fracture)
    - Posterior knee pain (popliteal cyst, OA, meniscus, gastroc/hamstring)
    - Traumatic (ACL, acute meniscus, patella/quad tendon rupture, patella dislocation, MCL tear)
    - Degenerative (OCD, OA, meniscus,

- Shoulder:
    - Traumatic (biceps rupture, dislocation, fracture, shoulder separation, clavicle fracture, rotator cuff tear)
    - Degenerative (rotator cuff tendinopathy/tear, glenohumeral OA, AC OA)
    - Chronic (OA, rotator cuff pathology- tendionpathy, chronic tear, biceps tendinopathy)

Treatment: based on diagnosis, age, function, goals

- Knee: PT, bracing, injections, surgery

Shoulder: PT, injections, surgery

HOW TO GIVE A GREAT LECTURE

Notes on 'How to Give a Great Lecture'

Robert Dickson

What's wrong with most lectures?

- Lack of **empathy**: failure to imagine the experience of the lecture from the audience's perspective
- Lack of **synthesis**: mere reporting of data without meaningful interpretation
- Lack of **preparation**: little thought or time given to optimizing presentation

Grab 'em by the throat:

- The **stakes**: why this matters *to your audience*
- The **crisis**: the problem that you're going to remedy
- The **fix**: how you're going to resolve the crisis in your talk

Three compelling story structures (from Little 2016):

- **Story A**: Facts about the world; existing work explains it poorly; we explain it better.
- **Story B**: Topic is heavily studied; but something is missing or wrong; we fill that gap.
- **Story C**: Theories and facts seem contradictory; we resolve the contradiction.

Further resources on effective communication:

- *Trees, Maps, and Theorems* by Jean-luc Doumont: phenomenal book on clarity in communication (writing, presentations, figures); Doumont speaks at UM every fall, well worth attending.
- *The Sense of Style* by Steven Pinker: thoughtful and practical "style guide" on writing by a lucid cognitive scientist
- *Authority and American Usage* by David Foster Wallace: wonderful essay on code-switching and adaptive grammar
- *The Visual Display of Quantitative Information* and *The Cognitive Style of PowerPoint* by Edward Tufte: seminal books on achieving clarity via data visualization (and obscuring it via PowerPoint)

Further resources on effective storytelling:

- *Three Templates for Introductions to Political Science Articles* by Andrew Little: www.andrewtlittle.com/papers/little_intros.pdf
- *The Hero with a Thousand Faces* by Joseph Campbell
- *Story* by Robert McKee
- *Aaron Sorkin Teaches Screenwriting*: www.masterclass.com/classes

Further resources for choosing color palettes:

- *ColorBrewer*: colorbrewer2.org/
- *Pictalicious.com*: make a palette from a photograph:
- *Wes Anderson palettes*: wesandersonpalettes.tumblr.com/

Advice on using PowerPoint:

- Teach more by teaching less: prune slides to a single coherent point.
- Stop dividing the audience's attention: don't display text you don't want the audience to read.
- Avoid the passive voice trap.
- Be concrete with your slide title.
- Use hierarchies coherently.
- Use light text on a dark background.
- Use a small number of themed colors.
- Use a sans serif font (Arial, Helvetica, Gill Sans, etc.).
- Never use Comic Sans.
- Use animations to control your audience's attention.
- Consider avoiding bullet lists altogether.
- Use coherent heuristics.
- Use vivid, self-explanatory figures to improve clarity.
- Don't use unnecessary animations.
- Don't use unnecessary clip art.
- Show radiology images on a black background.
- End strong by bringing it home.
- Consider a handout.
- Invest in a laser pointer/slide advancer.
- Bring your own laptop and adapter.
- Show up early, run through your slides.
- Practice! Time yourself.

Extra notes from lecture:

Communication is crucial to success in academic medicine

1) What's wrong with most lectures
    - Information vs message (meaningful interpretation)
    - Prepared/thankful for the audience's attention. Need to fight for attention and hang on to it.
2) How to grab audience by the throat
    - Protagonist with intention and an obstacle (early and effectively)
    - Need different structure for academic talks = stakes, crisis, fix
    - Act like you care about your talk, if you care the audience will care
    - Subtraction is key (get rid of parts that don't progress the story)
    - substance/rigor vs triviality/fluff (can be combo of these)
    - Meet audience at their level
3) Powerpoint
    - anchoring bias and halo effect - use to advantage, good slides might make people assume you'll have a good talk too
    - teach more by teaching less, stop dividing audience's attention
    - be more concrete/specific
    - 8% males are red/green colorblind, can create palate from figures, remember eyedropper tool
    - avoid bullets and convert to a figure if you can
    - show and tell figures (add figures when possible), show article, table highlighting what's important or make your own figure)

End it by bringing it back to the stakes/why this matters

RESIDENTS AS TEACHERS: TEACHING IN THE CLINICAL SPACE

KEY TAKE HOME POINTS:

What are at least 3 qualities of good clinical teachers?

- Positive relationships with learners
- Good communication skills
- Can diagnose and treat patients and learners
    - Strong knowledge base and clinical reasoning
- Seems to have Perfect talk at perfect time
- Enthusiasm

What are at least 3 tools to help promote knowledge retention for your adult learners?

- Mnemonic, algorithm, tables, white board, list, spaced repetition
- Advanced organizer: Example from clinical problem solvers site

What is a teaching script? How is this different/similar to a chalk talk?

- Pre-planned script to deliver high yield teaching in an organized way
- Can include Chalk Talks, worksheets, interactive discussions

What are the four steps to create a teaching script?

1. Planning: Identify a commonly encountered teachable moment or clinical trigger
    a. Symptom,exam finding, test result, clinical scenario, management question
2. Target High Yield teaching point
    a. How to approach, a schema, pathophysiology, EBM, physical diagnosis
3. Identify Evidence-based sources to develop your teaching script
    a. Clinical care guidelines, ABIM choosing wisely, clinical exam series
4. Organize
    a. Audience, where, plan questions, use tool to promote knowledge retention, keep it short

PREPARING A TEACHING SESSION

LINK TO ASSIGNMENT/PLANNING SHEET

RADIOLOGY REVIEW

INTERVENTIONAL RADIOLOGY - WHEN TO CONSULT

- DR does lots of procedures in the hospital, basic biopsies, paras, etc, BUT anything more complex is likely IR
- commonly consulted for:
    - bleeds: GI, RP, bronchial
    - tubes/drains/caths: PCNs, PTBDs, PCTs
- if IR says no to a procedure, it's d/t significant concern for bad complication
- **Bleed Consult**
    - **LOCALIZATION is everything** - CTA or endoscopy
        - drastically changes clinical & technical approach, end organ or not? organ ischemia? etc
        - imaging a bleed: sensitivity depends on speed of bleed
            - **conventional angiography** if **>1 cc/min**
            - **CTA** if **>0.5cc/min**
                - i.e. if you can't see extrav on CTA, you won't see it on conventional angio (bleed is too slow)
                - if bleed <0.5cc/min, not arterial
            - **tagged RBC** if **>0.1cc/min**
        - if pt with c/f hemorrhage, get CTA!
- **CONTRAST AGENTS DO NOT HURT KIDNEYS**
    - modern contrast agents are not nephrotoxic, this is based on outdated contrast agents
    - if the patient needs contrast then give them contrast (DR most likely to push back on this)
    - concerns about contrast agents delay care
    - do you need to check GFR first? **NO**
    - evidence-based
- **GI bleed**
    - urgent, unstable bleed + GI can't scope → **CTA** unless imminently about to code
    - cannot prophylactically embolize SMA/IMA given high risk of bowel ischemia
- **RP bleed**
    - small arterial branch in RP ruptures → potential spaces opens up → RP vessels rupture → expansion of hematoma → vicious cycle
    - if IR says no to embo, it is bc there is no point, we reached a point of no return and bleeding is mostly venous now
    - typically pts get conservative management
    - if iatrogenic etiology, IR most likely to take pt because then there is 1 to a few culprit arteries to target
    - if spontaneous bleed, get CTA, IR may or may not take pt depending on localization
- **BAE**
    - CTA in massive hemoptysis - localize
        - confirm bronchial vs pulmonary artery
        - confirm laterality
        - identify take-off of the bronchial arteries/variants
        - do NOT see extrav on CT or conventional angio if bronchial bleed
- **Tubes/Drains**
    - drains are more involved than they seem, take hours, not simple or easy
    - many times need advanced procedures like lithotripsy, balloon ureteroplasty, etc
    - think about what the drain means for pt in terms of QOL, change pt's life significantly

    - seriously consider whether the drain in question can be capped
- **Biliary Interventions (PTBDs)**
    - biliary tubes are the worst for IR
    - massive QOL hit for pts - painful, bile leakage, q4w exchanges, prone to clogs
    - responsible for getting them tube-free after placed, hard to take out, most have them for rest of their lives
    - just because there is a drain in, does not mean there needs to be a bag, goal is to cap it if internal/external biliary drain
- **PCT** (perc cholecystostomy tube)
    - gallbladder drain for patients with chole but not surgical candidate
    - VERY hard to get pts tube-free, need compelling reason
    - equivocal imaging findings + leukocytosis is not a good enough reason
- G-tube is highest risk, lowest reward procedure in IR
- *"simple" IR procedures can always go wrong*, be mindful when consulting

NEURO IMAGING AND EXAM

DIZZINESS – PEARLS

- Physical exam – Cranial nerves, drift, coordination (finger-nose-finger, heel-knee-shin), gait
    - Episodic? Add orthostatics and Dix-Hallpike
    - Nystagmus present? Add HINTS—Head impulse, Nystagmus type, Test of Skew
    - Head impulse: corrective saccades indicates peripheral abnormality on the side you turn towards
    - nystagmus: vertical and changing directional nystagmus are concerning for central pathology
    - test of skew: cover eyes
- Acute Vestibular Syndrome (AVS) – continuous, persistent, eg vestibular neuritis, posterior circulation stroke
- Episodic Vestibular Syndromes (EVS)
    - Triggerable – t-EVS, eg BPPV, Orthostatic hypotension
    - Spontaneous – s-EVS, eg vestibular migraine, TIA

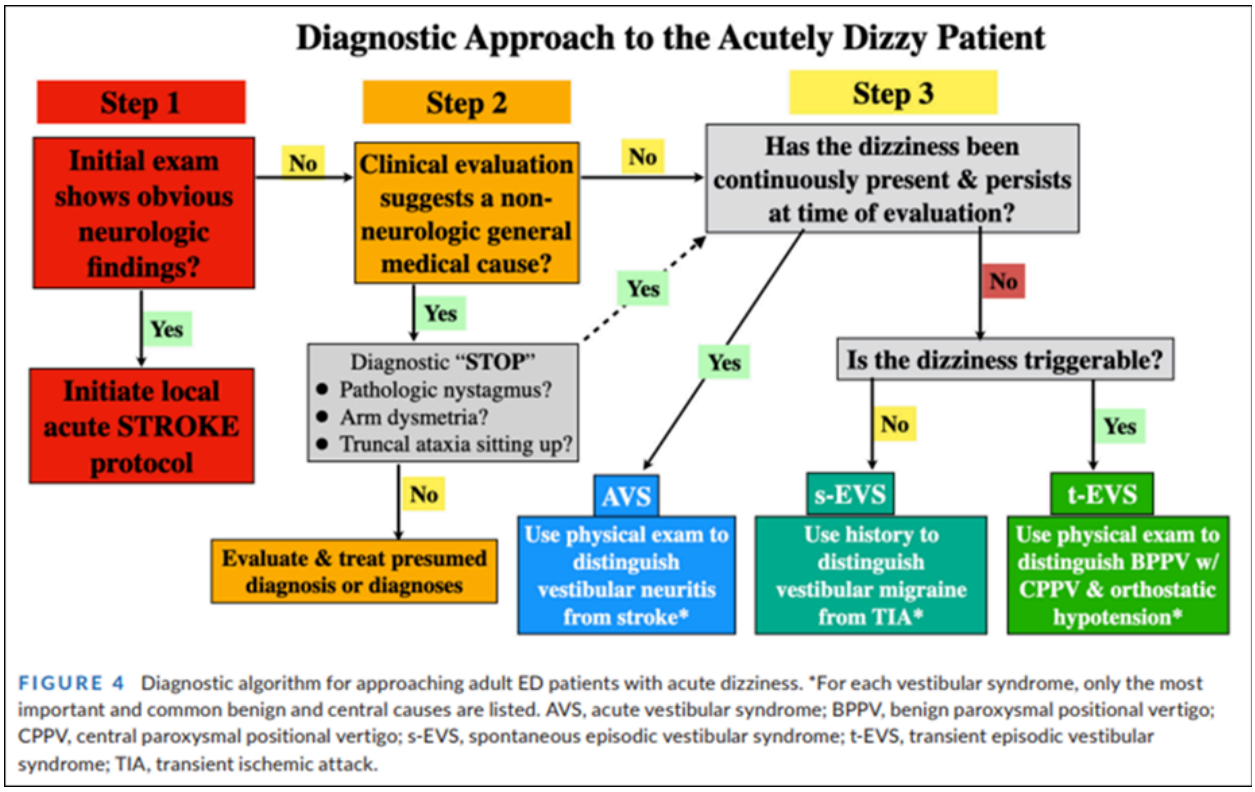


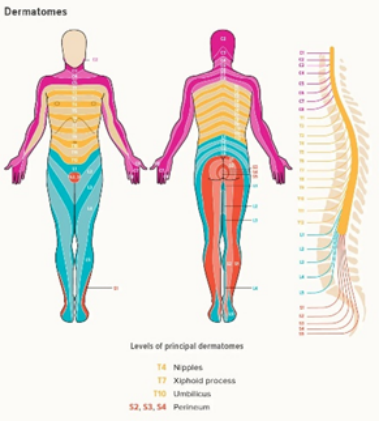
FIGURE 4 Diagnostic algorithm for approaching adult ED patients with acute dizziness. *For each vestibular syndrome, only the most important and common benign and central causes are listed. AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional vertigo; CPPV, central paroxysmal positional vertigo; s-EVS, spontaneous episodic vestibular syndrome; t-EVS, transient episodic vestibular syndrome; TIA, transient ischemic attack.

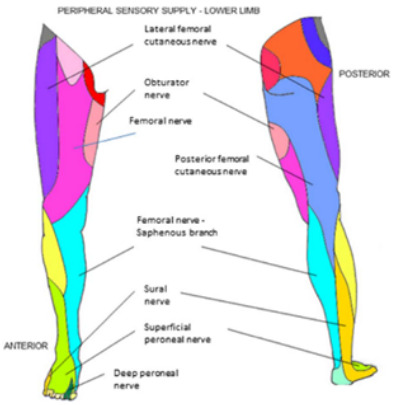
Cost effective care:

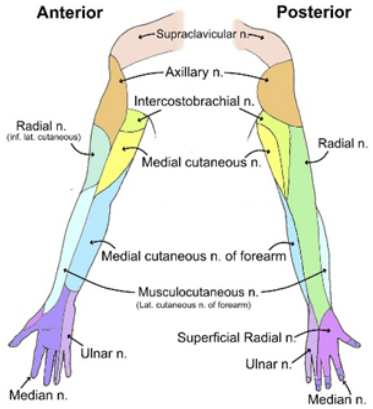
- Orthostatic VS and Dix-Hallpike for all pts with EVS
- If you can diagnose peripheral cause by physical exam you DO NOT need CT, MRI or hospital admission!!
    - BPPV – Dix-Hallpike finds upbeat torsional nystagmus; downward ear = symptomatic,
        - § tx w/ Epley in ED; give home Epley handout, consider Ca/VitD supp
    - Vestibular neuritis – Horizontal nystagmus beats away from sx ear and *never changes direction*;
        - § abnormal head thrust toward sx ear, otherwise reassuring HINTS
        - § consider steroid if within 3d onset

Approaching a patient with BACK PAIN

- Physical Exam: sensory, motor (including tone), and reflexes
    - When checking sensation, always go in with a hypothesis.







- If there is weakness, use reflexes and tone to differentiate an upper and lower motor neuron process.

Recognize RED FLAGS requiring emergent imaging:

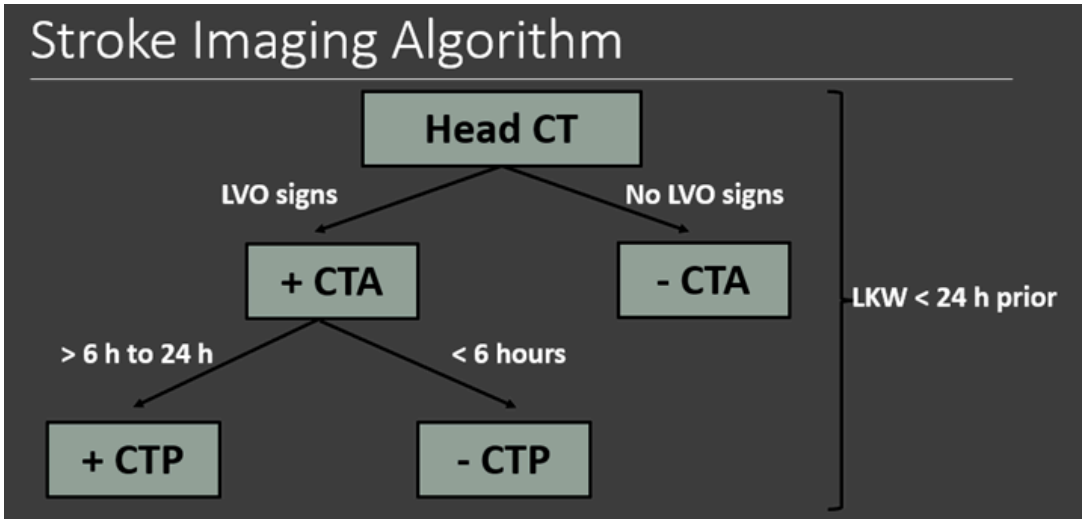
History:	Physical Exam:
Severe or progressive neurologic deficits • **Bowel / bladder changes** • **Weakness** • **Saddle anesthesia**	Abnormal Neuro Exam: • Weakness • Saddle anesthesia • Loss of sphincter tone
Significant trauma	Vertebral tenderness / limited spinal ROM
Unexplained fever	Fever
Sudden back pain with spinal tenderness	
Serious medical condition • **History of cancer** • **Immunosuppression / prolonged steroids** • **IV drug use** • **Unexpected weight loss**	
Worse when supine / at night	

In patients with back pain, choose your imaging wisely!
–X-ray or CT if musculoskeletal
–MRI if concern for disc disease (with / without neurologic symptoms)

Consider EMG to differentiate radiculopathy from other causes of weakness / numbness.

STROKE - TIME IS BRAIN!

- Quickly figure out the following:
  - Last known well
  - Screen for mimic: BP, glucose, seizure, migraine
  - Consider contraindications to thrombolysis: anticoagulation, recent surgery, severe head trauma 3 mo, prior intracerebral hemorrhage, brain tumor, GI malignancy ro recent bleed.



Thrombolysis:
IV tenecteplase (TNK)
–Given within 4.5 hours
–Dosing 0.25 mg/kg (max 25 mg) push in 5 sec

Endovascular Therapy:
—Give thrombolysis first if within 4.5 hours.
–If stroke is small on imaging (CT Perfusion or ASPECT score) then consider from 6-24 hours.

HEADACHE (HA) – Exam and imaging tips

- Check the following on physical exam: cranial nerves (inc pupils and fundi), neck, and basic neuro
- Migraines are the most common reason to present to the ED. Review the diagnostic criteria through the International Headache Soc online – ichd-3.org – is worth your time!
- Anyone who presents to the ED with a headache should be screened for a secondary headache. Imaging should be obtained if **SNNOOP10** is positive.

Table 2.—The American Headache Society "Choosing Wisely" Recommendations

1. Don't perform neuroimaging studies in patients with stable headaches that meet criteria for migraine
2. Don't perform CT imaging for headache when MRI is available, except in emergency settings
3. Don't recommend surgical deactivation of migraine trigger points outside of a clinical trial
4. Don't prescribe opioid or butalbital-containing medications as first-line treatment for recurrent headache

Imaging Recommendations:

- MRI brain w/wo contrast is best unless presentation is emergency.
- CT Head Non-contrast in an emergency
  - 95% sensitivity for SAH within 48h

- MR Veno head or CT Veno head – consider if thrombophilia, thunderclap onset, pregnant/postpartum, positional, papilledema
    - MRV head *without contrast* if pregnant.
- MR Angio or CT Angio head/neck – Consider if HA onset after trauma, thunderclap onset, or stroke-like symptoms

SNNOOP10
(aka indications to get a head CT for a patient with headache)

S	**S**ystemic symptoms (such as fever, rash, etc)
N	History of malignancy/tumor (**n**eoplasm)
N	**N**eurologic exam abnormality or focal deficit (including decreased consciousness)
O	Sudden or abrupt **o**nset
O	**O**lder age (> 65)
P1	Change in **p**attern
P2	**P**ositional features
P3	**P**recipitated by Valsalva maneuvers
P4	Presence of **p**apilledema
P5	**P**rogressive nature/worsening
P6	Onset in **p**regnancy or post-partum
P7	**P**ainful eye movements or autonomic features
P8	**P**ost-traumatic onset
P9	Immune system disorder or **p**athology
P10	Overuse of analgesics/'**p**ainkillers' or new medication use at onset

- **DDX for headache with loss of consciousness**
    - SAH (often due to aneurysm rupture)
    - EDH
    - seizure
    - venous sinus thrombosis
    - RCVS (reversible cerebral vasoconstriction syndrome)
    - PRES
    - stroke
    - Colloid cyst (rare)

*** general principle: to lose consciousness, you need to knock out both hemispheres, the brainstem, or both thalami

- cranial nerve exam in a comatose patient
    - 2: pupillary responses and blink to threat

    - 3 and 6: doll's head

- GCS scoring: out of 15.
    - pneumonic to remember categories: **4** eyes + v**6** motor (and 5 for the remaining category)
    - 3 is lowest score, 15 is highest

Glasgow Coma Scale		
Response	Scale	Score
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points; **Severe Brain Injury** = 3-8 points		

-

ACUTE UPPER GI BLEED

Causes of UGIB:

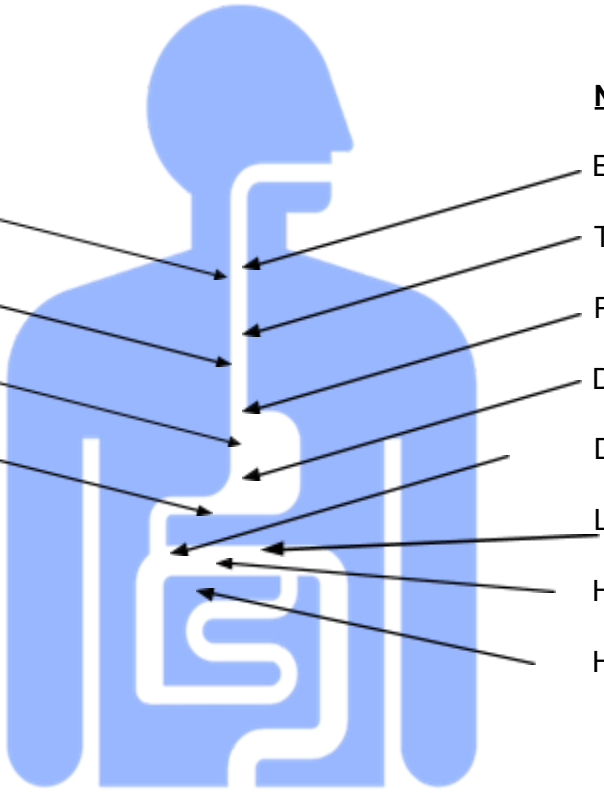
Liver-related causes:

Esophageal varices

Gastric varices

Portal HTN gastropathy

GAVE



Non-liver related causes:

Esophagitis

Tear: MW or Boerhaave

PUD

Dieulafoy

Duodenal ulcer

Ligament of Treitz

Hemosuccus pancreaticus

Hemobilia

*Cancer, ulcer, AVMs can occur anywhere along GI tract and cause bleeding

Approach/Treatment to Acute UGIB

Immediate	Urgent	Empiric Treatment	
		Suspected cirrhosis	No suspected cirrhosis
• ABC/Vitals • Brief exam • IV access → 2 large bore • Resuscitate (IV fluids) • NPO status	• Call GI (+/- IR) • Review history and MAR (discontinue appropriate meds (i.e. prophy heparin, BP meds)) • Supportive transfusion (Hgb goal is usually 7 and for cardiac patients, goal might be 8; platelet goal >50k; INR < 1.5	Only 50% of variceal bleeds stop spontaneously • Ceftriaxone reduces mortality, bacterial infection, rebleeding rates, and hospitalization days by -1.9 days • Octreotide • EGD within 12 hours • ICU level care • If refractory, IR	90% of non-variceal bleeds stop spontaneously • Start IV PPI • EGD within 24 hours usually

	but not reliable in cirrhosis)	can do emergent TIPS vs. other procedures	

Notes on lines:

- Usually patients in the hospital have a 20-22G
- To give a 1L bolus, 16G takes 5.5 minutes; 18G takes 11 minutes, 20G takes 17 minutes

Blood transfusion thresholds:

- NEJM 2013 found that less is more after studying 921 patients severe UGIB and randomizing to restrictive (<7) vs. liberal (<9)
    - Liberal group had higher mortality, death 2/2 bleeding, adverse and serious adverse events, and rates of further bleeding
    - ½ of the restrictive group required no transfusions
    - HR significant in those with cirrhosis particularly because more blood products increase portal pressure
    - **Conclusion: Hgb threshold of 7 is recommended**

Gold standard mock page:

- Items in red were commonly missed
- **Assessment:** Conduct a physical exam and ask for vitals
- **Lab workup:** CBC, T&S, INR
- **Treatment:** IV fluid bolus (having hemodynamic effects), NPO, 2 large bore IVs, ceftriaxone, octreotide, Consult GI for EGD
- **Level of care:** Move to ICU
- Anticipatory guidance
- **Communication:** Close loop, notify SMR, see the patient, speak with the family

SEPSIS

1. What is sepsis?

2. What evaluation is recommended?

3. What is an appropriate “Fluid challenge?”

4. When to pull the trigger on antibiotics?

5. How do I monitor patients?

ANEMIA

Vitals
None
Have you checked your blood pressure or heart rate? I don't know how to check this.

Medications:

Do you take any medications? I don't take any prescription medications.

Do you take anything over-the-counter? Yes, ibuprofen for my back pain that flared up last week.

How much ibuprofen were you taking? I had some 600 mg tablets that I was taking every 3-4 hours

Are you on any blood thinners? No

If the student says the word "hemoglobin" or "CBC" please ask them, "What is that?"

Assessment
Why were you in clinic today? I was feeling lightheaded.
What did you and your doctor discuss? She told me to drink more water, and that she was going to check some blood tests.
How are you feeling? Somewhat tired but otherwise OK.
How long have you been lightheaded? One day

Do you have any chest pain, palpitations shortness of breath? No
Do you have any other symptoms? What do you mean?
Have you had any nausea or vomiting? No
Have you noticed any changes in your bowel movements? Yes, I had three black bowel movements today, most recently an hour ago.
Do you have any abdominal pain? Yes, I have had some cramping since last night

Have you ever had stomach ulcers? No
Have you ever been told you had low blood counts? No. I have never had my bloodwork checked before.

Do you have anyone who could take you to the ER? Yes, but they won't be able to for another 6 hours.
Do you feel like you could drive yourself to the ER? Not really with how bad I feel.

- When to send patients to the ER?
    - Acute chest pain, acute focal deficits, confusion, seizure, unstable vitals, profound symptoms
    - Ok to communicate uncertainty
    - If going to ER, how will get there?
        - Are they physically impaired due to symptoms?
        - Do they have transportation?
        - Are they in danger of becoming seriously ill in next 15 minutes?
- What you can prescribe?
    - Antibiotics, symptom management (inhaler)
- Guidance and next steps
    - Review precautions, home care advice, next steps
- Grey areas - how to triage:
    - Leg is painful and mildly swollen for past 2 days
        - Ask about shortness of breath, spreading rashes, DVT risk factors
    - 5 episodes of vomiting and diarrhea in last 4 hours
        - Bloody? Fevers?
    - Week of cough, sinus pain, purulent rhinorrhea. Symptoms have worsened in last day
        - Fevers, confusion, eye involvement
    - Painful arm rash that spread despite starting antibiotic
        - Fever, nausea, chills, animal bites, blisters

AFIB WITH RVR

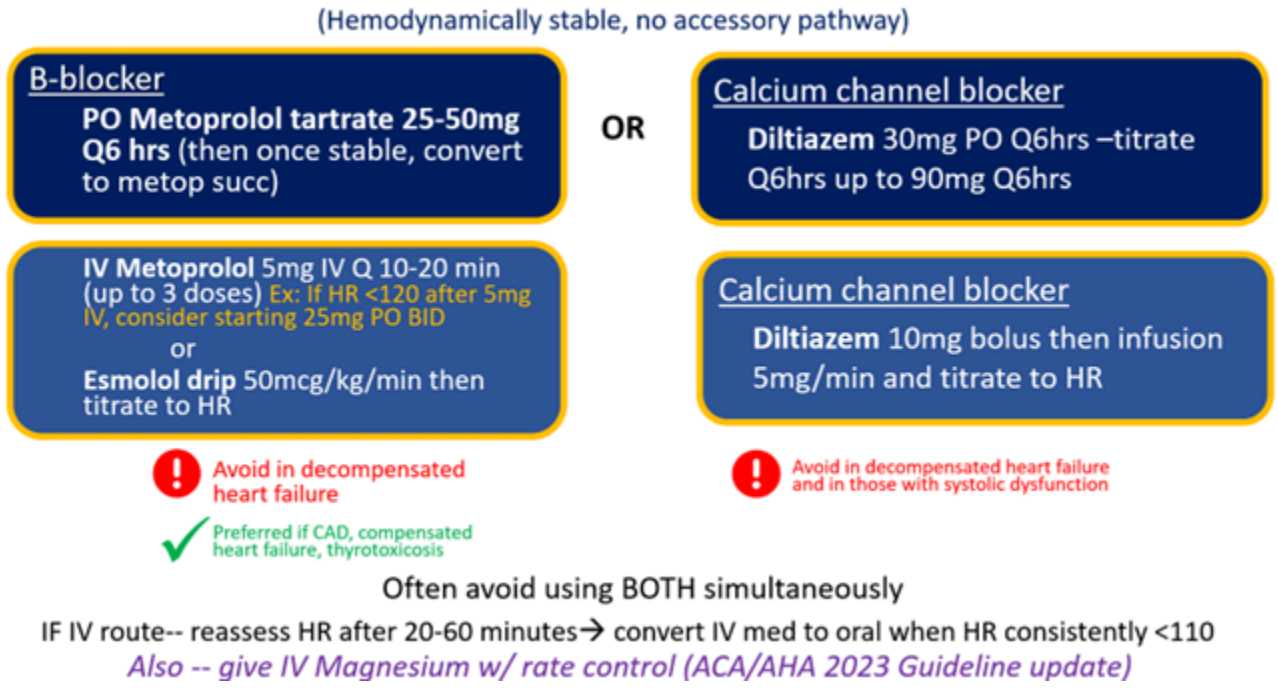
Assessment

- Symptoms to ask about
- Physical exam to perform:

Diagnostic work-up:

What are common triggers for afib?

Treatment (hemodynamically stable, no accessory pathway):

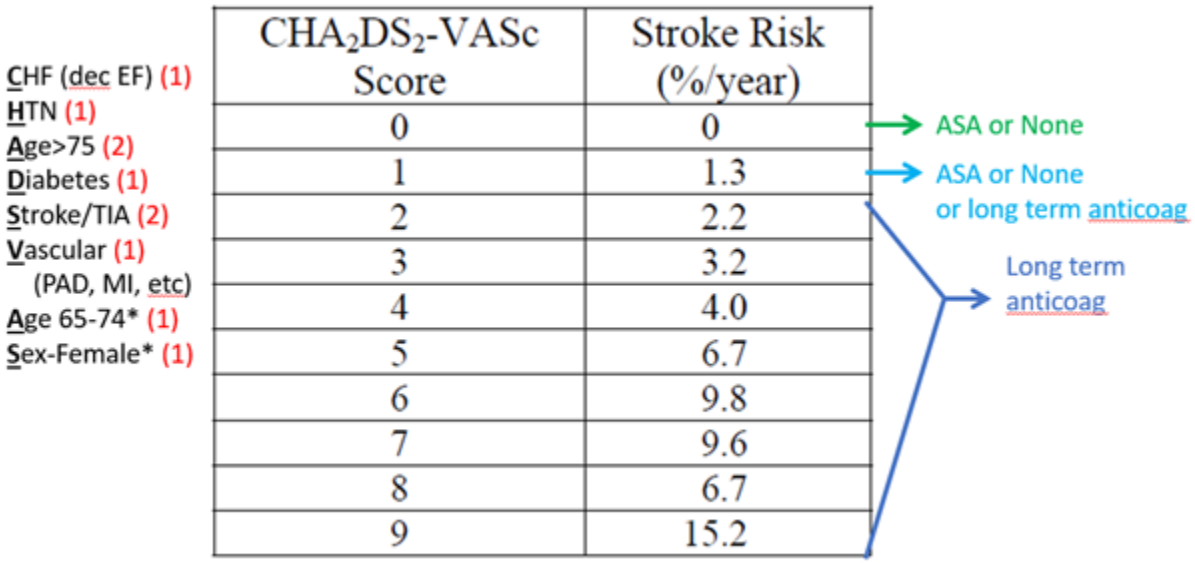


Rate control (*What is the goal HR in the hospital?)*

Rhythm Control: Especially ideal for first occurrence of symptomatic afib/flutter, occurrence due to reversable cause (e.g. post-op), hospital readmissions for afib, symptoms despite adequate rate control, inability to achieve adequate rate control, tachycardia-induced cardiomyopathy, or younger patients <65.

- Chemical cardioversion
- Electrical cardioversion

When is anticoagulation needed?



2 caveats to think about with anticoagulation:

When do you BRIDGE in the hospital?

Level of Care: Don't forget to transfer to telemetry!

UTI

Framework to approach outpatient pages

1. Overview
    a. Expectations
        i. Answer page within 15 minutes
        ii. Don't have to have computer access
        iii. In training, always have senior/attending oversight
        iv. Document note after phone call and route ot PCP
    b. Limitations
        i. No controlled substance refills
        ii. Can defer non-urgent topics to regular business hours
2. Framework triage steps
    a. Introduce yourself and role
    b. Gather history (HPI, PMH, meds, allergies - often can reference EMR remotely)
    c. Develop plan
        i. Recommendations - OTC meds/home care, prescriptions
        ii. Disposition - ER, urgent care, clinic in AM/Monday, home care
    d. Anticipatory guidance/reasons to seek additional care
        i. If prescribing abx, counsel about diarrhea/C diff risk
    e. Closed loop communication/teach back
    f. Document in EMR

Approach to UTI symptoms on call:

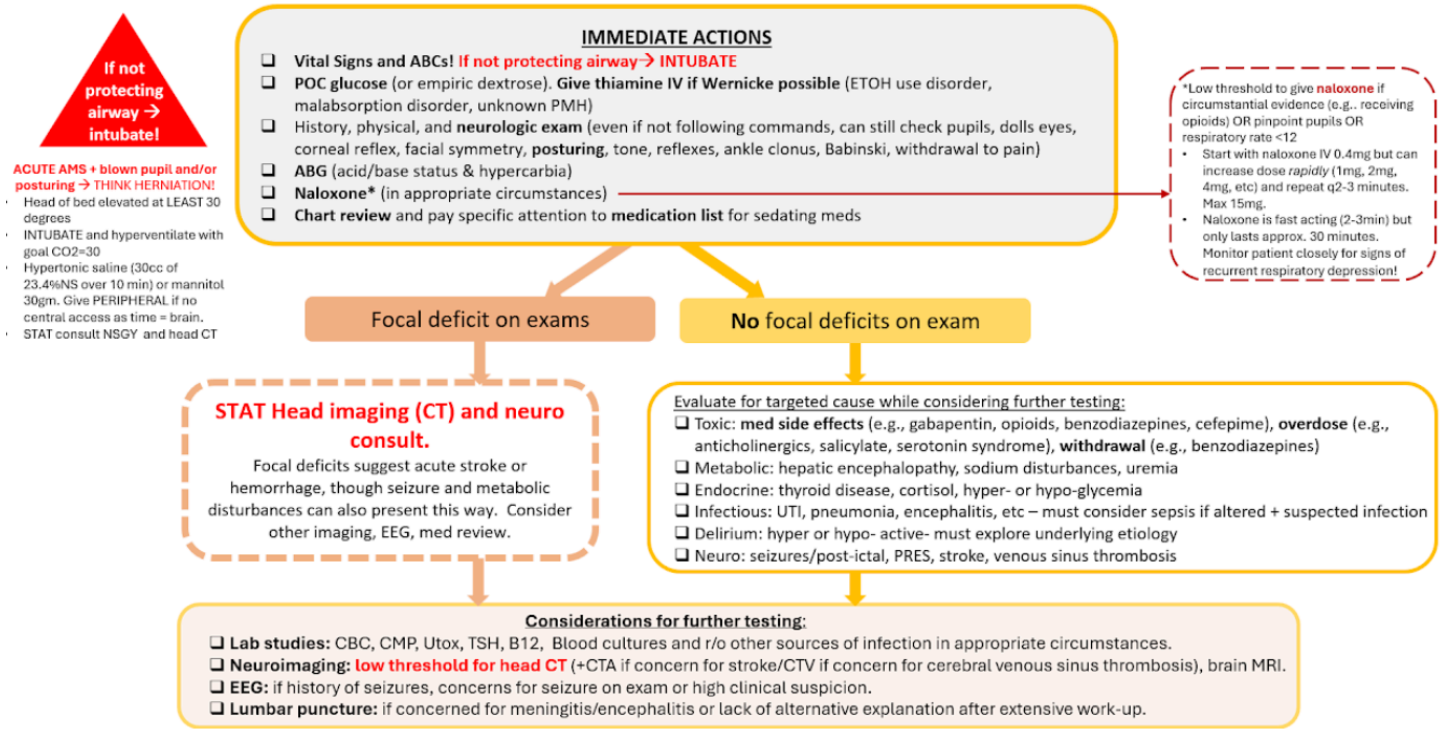
- Sxs of dysuria + frequency + no vaginal d/c or irritation in female = high positive likelihood ratio (>24) for UTI
    - Uncomplicated UTI = prescribing an abx is appropriate
- Must do
    - Intro name and title
    - Inquire about other sxs
    - Inquire about allergies
    - Call in abx
        - Bactrim, nitrofurantoin, fosfomycin, cephalexin
    - Tell patient reasons to go to ER
    - Ask pt to confirm plan moving forward
- Should do
    - Inquire about current meds
    - Ask about h/o UTI
    - Obtain patient's pharmacy info
    - Ask pt if they have any questions

General outpatient page Q&A:

- Things that are ok to prescribe
    - Topical abx, certain antivirals (Tamiflu, paxlovid)
    - Potentially abx for bacterial sinusitis, animal bite
- Things to recommend seeing a clinic/physician
    - New medications, inhaler, abdominal diagnoses (ex. diverticulitis)

ALTERED MENTAL STATUS

Click here for cross-cover guide which has AMS slide:



Naloxone

When do you give naloxone?

How much naloxone can you give?

There are at least THREE things you must think about after giving naloxone:

What is half life of naloxone?

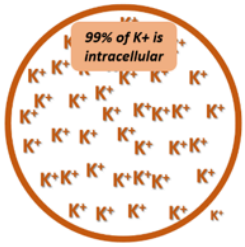
When do you need to admit to ICU for monitoring after naloxone successfully administered?

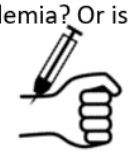
HYPERKALEMIA

Acute Hyperkalemia

Mild 5.5-5.9mEQ
Moderate 6-6.5 mEQ
Severe >6.5mEQ *or* EKG changes

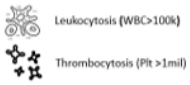
(1) Is this *REALLY* hyperkalemia? Or is it "Pseudohyperkalemia"?





Hemolyzed specimen

Always ask the lab if the specimen is hemolyzed!



(2) If this is real, what is the cause? Look at BMP, POC glucose, and EKG (if K>6) to start

Potassium load	Transcellular shift/ Cell breakdown	Renal excretion
K+ rich foods *(also think about TPN, tube feeds)*	Rhabdomyolysis Tumor Lysis Syndrome Hemolysis **Hyperglycemia**/DKA Acidosis Meds (i.e. BB, Digoxin)	Acute kidney injury CKD Urinary obstruction Meds (i.e. ACEI/ARB, TMP/SMX) Hypoaldosteronism Type 4 RTA

(3) How do I treat true hyperkalemia?

(often done simultaneously while determining cause)

Rapid onset
Short acting

Calcium gluconate (1 amp)
*can give Ca chloride –prefer central access
Give if EKG changes or severe high K (>6.5 reasonable)

Stabilize cardiac membranes

RECHECK EKG after giving calcium! Repeat dose if no improvement

B 2 Agonist (Ex: albuterol 10-20mg over 10 min)
Bicarbonate

Insulin (10 units) **PLUS**

Glucose (1amp of D50 = 25gm)

SHIFT the K+ back in the cells (takes minutes-hours)

RECHECK GLUCOSE frequently

Kayexalate/SPS (15gm PO) **or**
other binding agent (patiromer, zirconium cyclosilicate)

Diuretics
Diet (low K)
Discontinue contributing meds
Dialysis (in certain cases)

Decrease total body potassium (takes hours-days)

Later onset
Longer acting

(4) Continue to monitor

EKG: if EKG changes initially seen→ monitor for resolution
POC glucose: recheck hourly x 3 hours to ensure no hypoglycemia, then can space out
BMP: recheck at ~2hrs initially, then base frequency of monitoring on K+ trend