Internal Medicine QuickStart Guide

Last Update: Mar 2020
This QuickStart Guide has been created by the MGH Internal Medicine Residency as a primer to providers not trained in internal medicine who may be called to help on general internal medicine floors during the COVID-19 pandemic. This resource is not intended to be a comprehensive guide to all the things one may see on the general medicine inpatient services nor does it contain the totality of information that one could know about the topics discussed herein; rather, this is meant to cover the highlights of the topics that we – the internal medicine residents – feel are most important in the day-to-day inpatient management of internal medicine patients. This guide draws heavily on a backbone created by the core-educator faculty, to whom we are incredibly grateful; it also draws upon many previously created resources – including the MGH White Book (which is referenced within for further information), UpToDate, among others.

As with any other medical reference, this manual is NOT intended to provide specific clinical care decisions in an individual case, and should NOT substitute for clinical judgment. Every clinical care decision must be made by the exercise of professional judgment by the individual responsible for the care of a patient based on the facts of that individual case, which may differ from the facts upon which entries in this manual are based. You should consult other references and your fellow residents, fellows, and attendings whenever possible. We have carefully inspected every page, but errors may exist.

We appreciate all feedback (including messages about errors that may exist) to help make this a more useful resource; please reach out to Yousef Badran (yousef.badran@mgh.harvard.edu), Sneha Kannan (sneha.kannan@mgh.harvard.edu), or Brian Mugo (bmugo@mgh.harvard.edu) with any comments/questions/suggestions.

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**OTHER RESOURCES:**
2019-2020 MGH Medicine White Book: Access the app [here](#) OR Adobe [here](#) OR PDF [here](#)
Click the Whitebook hyperlinks on each page throughout the document for topic specific information (desktop compatible, NOT mobile compatible)
What are the can’t-miss diagnoses of my patient’s chest pain?

- **Cardiac:**
  - Acute MI (central pressure, +/- SOB; assess for known CAD, PAD, carotid disease; history of similar prior pain; note that symptom chest pain may not be classic in (1) elderly, (2) women, (3) diabetics). If you have suspicion for a acute MI (as discussed below) then call a RAPID RESPONSE
  - Myocarditis (similar character to acute MI but classically in patient with viral prodrome)

- **Pulmonary:**
  - Pneumothorax (sudden onset, recent procedure, unilateral absence of breath sounds)
  - Pulmonary embolism (sudden onset, pleuritic, +/- hypoxemia; risk factors for clot – malignancy, surgery, immobility)

- **Vascular:**
  - Acute aortic syndromes (sudden tearing pain, known aneurysm, BP differential between arms)

What are alternative diagnoses of my patient’s chest pain?

- **Cardiac:** pericarditis, aortic stenosis, stress cardiomyopathy (Takostubo’s)
- **Pulmonary:** Pneumonia, pneumonitis, pleurisy
- **GI:** GERD, esophageal spasm, PUD, pancreatitis
- **Pysch:** panic attack
- **Derm:** varicella zoster

What do I do when my patient develops acute-onset chest pain?

- Focused history (quality, onset, severity, radiation, relieving factors, on ASA, history of MI/CAD?)
- Focused exam (general appearance, cardiac exam, chest wall palpation, respiratory exam)
- Check vitals (low threshold to call Rapid Response if hypotension or severe hypoxemia)
- EKG (repeat at 10-15 min intervals until chest pain is resolved and monitor for any changes)
- Consider CXR if associated hypoxemia (order STAT and call 63050)
- Consider STAT labs (CMP, CBCd, VBG/ABG, lactate, TnT, D-dimer in appropriate setting)
- Ensure adequate IV access
- Low threshold to involve attending or Senior On (p22337) for any unstable patient
- Review labs, imaging & White Book – additional imaging/treatment as indicated

How do I work-up a suspected STEMI (refer to MGH inpatient STEMI protocol)?

1) If your patient has:
   - Concerning symptoms of chest pain, acute or worsening SOB, unexplained diaphoresis or nausea OR
   - Acute clinical deterioration (hypotension, hypoxemia, altered mental status) OR
   - ST changes on telemetry or new arrhythmia

2) THEN get an EKG to look for signs of STEMI:
   - New ST elevation ≥ 1mm in two consecutive leads (see Redbook p115 for details about leads; N.B. different criteria for leads V2-V3)
   - New ST depression in at least two leads V1-V4
   - New multi-lead ST depression with ST elevation in aVR
   - New LBBB with acute symptoms

3) IF EKG changes concerning for STEMI are present, then call RAPID RESPONSE to mobilize resources AND obtain IV access, give ASA 324mg immediately and refer to White Book for additional evidence-based adjuncts to reperfusion therapy (bottom of page)

   See White Book p14-18 for more details on ACS and Chest Pain
What is Heart Failure?
• Inefficient pumping > reduced blood flow to kidneys > salt/water retention > even worse pumping
  o Systolic or HF with reduced ejection fraction (HFrEF); EF <40%; ventricle can’t pump like normal
  o Diastolic or HF with preserved EF (HFpEF); EF > 50%; ventricle can’t relax and fill like normal

How do I know if my patient is having a Heart Failure exacerbation (acute decompensated HF)?
• May report: Dyspnea (+/- on exertion), lower extremity edema, weight gain, orthopnea, abd swelling
• Exam: crackles (unlike atelectasis, crackles don’t clear with coughing), elevated JVP, pitting edema (check up to buttocks), and weight (check Cardiology notes, last d/c summary for “dry weight”; ”lastwt[10] smart phrase can give you a trend); check extremities @ thighs (cold extremities = concern for cardiogenic shock)
• Studies: Elevated NT-proBNP (>450 for <50y/o, >900 for >50y/o; artificially low in obesity), elevated Cr, elevated LFTs, pulmonary edema on CXR

Why do they have acute decompensated Heart Failure?
• ~ 1/2 unknown, ~ 2/5 dietary/med non-compliance
• Can’t miss/should know causes: ischemia/infarction, arrhythmia, comorbid illness (GI, pulmonary), toxins (EtOH, cocaine), endocrinopathy, meds (excessive beta-blockade, NSAIDs, steroids, Ca-channel blockers)
• Work up: EKG (check for arrhythmia, q waves, ST changes, etc) +/- pacemaker interrogation, telemetry (24h), TSH, TTE (NOT ROUTINE; consider if none in last ~ year AND/OR you suspect a new/worsening valve issue (e.g. new/worsening murmur) or ischemia)
• If new diagnosis, consider Heart Failure consult & referring to HF Transitions clinic on discharge

How do I manage/treat decompensated HF?
• Basic orders: telemetry, Na (2g) and fluid (<2L/d) restricted diet, daily weights, strict I/Os, VTE ppx
• Meds: NO NSAIDs, NO Ca channel blockers, IF IN SHOCK/ VERY DECOMPENSATED, HOLD beta-blockade; otherwise continue; IF IN SHOCK/HAS WORSENING AKI, HOLD ACEi/ARB; otherwise continue
• Removing fluid and volume management
  o Diuretic conversion: Lasix 80 PO = Lasix 40 IV = Torsemide 40 PO = Bumex 2 IV/PO
  o Start with 2x the home dose of diuretic, give in IV form; so if 20mg PO Lasix at home, give 20 IV. If no home dose, start with ~20x their Cr (often Cr increased from baseline due to cardiorenal cause)
  o If they haven’t peed in 1-2h, then double dose (still IV). If they do pee, repeat this dose 1-2 times daily
  o Switch to oral diuretics when you think they have peed enough to be back to their “dry weight”
  o Carefully monitor Ins/Outs (place Foley if needed) & daily STANDING weights (bed weights unreliable)
• Special medication considerations:
  o If HFrEF: if BP/HR/lytes tolerate, they should be on BB, ACEi/ARB and Spironolactone prior to D/C
  o If anemia, screen for iron deficiency (TSat < 20%); give IV iron if needed (200mg Venofer qOD x5 doses)
• Discharge preparation
  o Please note discharge weight and discharge NT-proBNP in hospital course
  o If the patient has an MGH cardiologist, consider HF Transitions Clinic (J. Ruckel, NP; D. Valladane)

How do I know when to ask for help?
• Patient is not peeing to increasing amounts of diuretic
• Patient has signs/symptoms of cardiogenic shock such as hypotension (MAP < 60mmHg), increasing lactate/Cr/LFTs, or worsening mental status
• Consider consulting HF if very low EF (<25%)

See Whitebook p25 for more details on Inpatient Heart Failure
**Where do I start?**

- Obtain vitals & EKG and do an exam
  - Is the patient pulseless? >> **CODE BLUE**
  - Is the patient unstable (low blood pressure, altered mental status, chest pain, SOB) >> **RAPID RESPONSE**
    - Instability requires ELECTRICITY (cardioversion or defibrillation; may need to sedate patient)
- If the patient is stable, you can direct your management by figuring out if this is a:
  - Wide Complex Tachycardia (WCT): QRS > 120 (3 small boxes on ECG)
  - Narrow Complex Tachycardia (NCT): QRS < 120

**What do I do if this is a stable wide-complex tachycardia (WCT)?**

- If lasting < 30 seconds (non-sustained), check BP, electrolytes & replete; continue to monitor
- If lasting > 30 seconds (sustained), make sure the patient has a pulse, then **CALL RAPID RESPONSE & PLACE DEFIBRILLATOR PADS**; then
  - Confirm code status & IV access
  - Ask for a baseline EKG (prior to abnormal rhythm) & check the QTc
  - Place BP cuff on patient and set to cycle every 2-3 minutes

**Could this wide complex tachycardia be a supraventricular tachycardia (SVT) with aberrancy instead of VT?**

- Always err on the side of caution and assume first that all WCTs are ventricular tachycardia (VT)
- Suspect SVT with aberrancy in young patients who do not have prior cardiac history if normal baseline QTc

See [Whitebook p9](#) for more details on Wide Complex Tachycardia

**What do I do if this is a stable narrow complex tachycardia?**

- Determine if the patient is tachycardic but in sinus rhythm
  - Sinus rhythm = P before every QRS; QRS after every P; P wave is upright in leads I & II
  - Sinus tachycardia is often compensatory – find & treat the underlying cause(s), e.g. pain, fever/infection, hypovolemia, substance use & withdrawal, etc. No need to beta-block unless symptomatic or lasting for days
- If the patient is not in sinus rhythm AND has normal or high BP, consider treating with:
  - Metoprolol: IV 2.5-5mg q5min for 2-3 times
  - Diltiazem (if baseline EF on Echo is normal): IV 5-10mg q5min for 2-3 times
  - ALWAYS repeat BP before giving the next dose
  - REMEMBER to chase with oral doses afterwards (metoprolol 25mg q6h or diltiazem 25mg q6h)
- If the patient is not in sinus rhythm AND has borderline BP (Systolic BP 100-120), **ASK FOR HELP**
  - You can consider a trial of amiodarone 150mg x 1

**What about adenosine?**

Adenosine can help terminate a narrow complex tachycardia or help you figure out what it is. Because adenosine can cause prolonged pauses & asystole, you will need to **CALL A RAPID RESPONSE**

See [Whitebook p8](#) for more details on Narrow Complex Tachycardia
What do I do about a slow HR?

What next?
- Review telemetry
  - Bradyarrhythmias: complete heart block, type II second degree AV block
- Review recent medications, procedures, hospital events
- Ensure beta-blockers and calcium channel blockers have holding parameters for heart rate and blood pressure
- Labs: CMP (check electrolytes, renal function), lactate, +/- troponin if concern for ischemia/ACS

What if I am concerned about beta-blocker or calcium channel blocker overdose?
- Give IV glucagon (3-10mg bolus, followed by infusion at 3-5mg/hr)

See Whitebook p3 for more details on Bradycardia
Severe Asymptomatic Hypertension and Hypertensive Emergency:

<table>
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<tr>
<th>Severe asymptomatic HTN</th>
<th>Hypertensive Emergency</th>
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<tr>
<td>Definition</td>
<td>BP &gt; 180/120 with end-organ damage</td>
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<tr>
<td></td>
<td>Examples: encephalopathy (severe headache, seizure, AMS), TIA, papilledema, retinal hemorrhage, pulmonary edema, MI, angina, hemolytic anemia, AKI/hematuria</td>
</tr>
</tbody>
</table>
| Time Course             | 1) Reduce MAP 10-20% in first hour (e.g. target <180/120) but by no more than 25% in first 24 hours  
2) Reduce to normal over days (target < 130/80) |
|                         | Exceptions (place EMERGENT CONSULT): Ischemic stroke (neuro), Aortic dissection (cards, cardiac surg, vascular surg), ICH (neurosurg) |
| PO vs. IV               | PO: Short-acting -> Convert to long-acting prior to discharge |
|                         | Short-acting IV -> Convert to PO when improving |
| Meds                    | Captopril (6.25mg q8, avoid if AKI), Labetalol (100mg q8, avoid if HR < ~65-70), Hydralazine (10mg q6 or q8), Isordil (5-10 mg TID) |
|                         | IV: Labetalol (start with 20mg, can give increased dose every 10min), or Hydralazine (start with 5-10mg, can give increased dose every 15-30min)  
Topical: Nitropaste (rapid on/offset, check BP often and add another med early)  
Infusion/gtt: Cannot be used on normal medicine floors |

Routine Hypertension:
If asymptomatic and BP < 180/120, in general no urgency to treatment.
Can work on normalizing BP to < 130/80 over days.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosing:</th>
<th>Adverse Effects:</th>
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<tbody>
<tr>
<td>Amlodipine</td>
<td>5-10mg daily, can take 24-48h to take effect</td>
<td>Lower extremity edema</td>
</tr>
<tr>
<td>Lisinopril (ACEi)</td>
<td>5-40mg daily</td>
<td>Hyperkalemia, AKI (tolerate up to 30% increase in Cr), Cough, Angioedema, Contra-indicated in pregnancy. If initiating, BMP within 1 week</td>
</tr>
<tr>
<td>Preferred in CHF, DM, CKD</td>
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<tr>
<td>Losartan (ARB)</td>
<td>25-100mg daily</td>
<td>Hyperkalemia, AKI, NO cough/angioedema. If initiating, BMP within 1 week</td>
</tr>
<tr>
<td>Preferred in CHF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carvedilol</td>
<td>6.25-25mg BID (can do 50mg BID if wt &gt; 85kg). Preferred in CHF</td>
<td>Bradycardia (avoid if baseline HR &lt; 70), Consider holding in CHF exacerbation</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>12.5-50mg daily</td>
<td>Hypokalemia, hyponatremia, hyperuricemia, hypercalcemia, hypomagnesemia (seen with &gt;25mg daily). If initiating, BMP within 1 week</td>
</tr>
<tr>
<td>Isosorbide Dinitrate</td>
<td>5-40mg TID. Long acting is isosorbide mononitrate (Imdur) Preferred in angina/CAD</td>
<td>Headaches, Rebound hypertension</td>
</tr>
</tbody>
</table>

What if the hypertension still isn’t controlled?
Once the patient is on 3 agents of different classes at max doses (including 1 diuretic), consider sending work-up for secondary hypertension (AM serum aldosterone and renin levels; renal duplex/vascular US; TSH; OSA -> outpt sleep study; 24h urine free cortisol). Consider nephrology consult vs. outpatient referral.

See Whitebook p38 for Hypertensive Urgency & Emergency or p40 Outpatient HTN for more details
What do I do about hypotension?

Hypotension
(SBP < 90 or ↓ SBP > 40 mmHg from baseline)

Obtain vitals (confirm BP on manual check), H&P (chest pain?, SOB?, lightheaded?; mental status, warm/cool extremities – check at medial knee)

Asymptomatic (N.B. many elderly patients and young women have baseline SBP 80s-100s, especially while sleeping)

1. Compare BP to recent values to assess trend
2. Send labs (lactate, ABG/VBG, CBC, CMP, TnT, EKG/CXR)
3. Ensure IV access in case need fluids (two 18G)
4. Review meds for potential causes
5. Review DDx (below) and treat appropriately
6. Repeat BP q30-60min x1-2 hours

Symptomatic: new altered mental status, chest pain, cold on exam

1. Call a rapid response
2. Order IVF (250-1000cc depending on risk of heart failure and pulmonary edema)
3. See steps 1-5 for “asymptomatic”
4. Repeat BP q5-15 min to assess improvement with fluids
5. Other tools include vasopressors if headed to ICU

Why is my patient hypotensive?

- Mean arterial pressure = Cardiac output x systemic vascular resistance + central venous pressure
- To be hypotensive, must be an insult to heart’s ability to squeeze (like in cardiogenic shock/heart failure, the vasculature (globally vasodilated like sepsis) or fluid status (volume depleted like in a bleed)

<table>
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<tr>
<th>HYPOTENSION</th>
<th>↑ CO</th>
<th>↓ CO</th>
</tr>
</thead>
<tbody>
<tr>
<td>ETIOLOGY</td>
<td>DISTRIBUTIVE (66%)</td>
<td>HYPOVOLEMIC (16%)</td>
</tr>
<tr>
<td>PATHOPHYS</td>
<td>↓ ↓ SVR CO → ↑ CO</td>
<td>↓ ↓ CO → ↑ SVR</td>
</tr>
<tr>
<td>EXAMPLES</td>
<td>Sepsis/SIRS, anaphylaxis, liver failure, adrenal insuff, meds</td>
<td>Bleed (GI, retroperitoneal, thigh, abdominal), diarrhea, emesis</td>
</tr>
<tr>
<td>TREATMENT PRINCIPLES</td>
<td>Antibiotics (vanc/cefepime) + fluids (CALL RAPID RESPONSE, GET HELP if not improving to above)</td>
<td>Volume resuscitation (IVF +/- blood) (CALL RAPID RESPONSE, GET HELP if not improving to above)</td>
</tr>
</tbody>
</table>

See Whitebook p60 for more details on Hypotension
What is hypoxemic respiratory failure?

- Inability to oxygenate tissues (deliver O\textsubscript{2}), know by the oxygen saturation < 90% (88% if COPD)
  - **Main causes of hypoxemia**: 1. Hypoventilation (low FiO\textsubscript{2})=decreased O\textsubscript{2} delivery to lungs 2. V/Q mismatch: imbalance of oxygen delivery and blood flow in the lung 3. Shunt: blood flowing through the lung doesn’t encounter oxygenated air 4. Impaired diffusion: oxygen isn’t able to diffuse from the air to the blood
  - **Common causes**: Pneumonia, mucus plugging, pulmonary edema from heart failure, COPD, asthma, atelectasis **Must not miss**: PE, pneumothorax, ARDS

What do I if a patient is hypoxemic?

- First: Supplement their oxygen: place a nasal cannula (NC) on (can increase to 6L/min flow), a facemask, or non-rebreather mask (up to 10L).
  - **COVID-19 era considerations**: Many normal supports for oxygen are considered aerosolizing and need airborne precautions: NRB >10L, BiPap, CPAP, nebulizers, high-flow nasal cannula. These should be avoided unless you have a discussion with respiratory, Medicine Senior, and or critical care attending.
- Do you need a rapid?: What is the oxygen saturation? What is their work of breathing (Are they tachypneic? Are they using accessory muscles to breathe?)? If the oxygen saturation is <90 and doesn’t improve with basic supplementation or they are working hard → CALL RAPID RESPONSE. **COVID consideration**: If rapidly escalating NC requirement >4L, contact Medicine Senior, RICU for consideration of early intubation.
- Diagnostics: Chest x-ray (STAT order & x6-3050), sputum culture, labs: BMP, CBC with differential, ABG or VBG, Legionella and Strep pneumococcal urinary antigen, respiratory viral panel (restricted because of COVID) and flu/RSV (will need droplet and strict contact precautions), CT-PE protocol, Coronavirus PCR
- Management: Depends on etiology
  - If suspect Pneumonia: start antibiotics (see Pneumonia page); pulmonary edema: diuretics; COPD or asthma flare: inhalers, steroids (see COPD page); PE: anticoagulation, PERT (DVT/PE);

What is hypercarbic respiratory failure?

- Hypercarbia is a build-up of CO\textsubscript{2}
- Caused by:
  - Decreased respiratory rate: secondary to sedatives, central sleep apnea, obesity, brainstem stroke
  - Can’t breathe: OSA, asthma/COPD
- Clinical presentation: sedated, decreased RR>> **think about hypercarbia when a patient with COPD is somnolent!**

What do I do if someone has hypercarbic respiratory failure?

- Diagnostics: Stat ABG preferred (VBG if ABG challenging), BMP, CBC with differential, CXR, consider toxicology screen
- Treatment: Prior to placing on non-invasive positive pressure ventilation, consult Medicine Senior, respiratory therapy as to what kind of airborne precautions the patient may need. See COPD (p11 COPD) management. If etiology sedation from opiates, give Narcan (0.4-2mg IV/IM q2 min, no upper limit, can start drip at 0.2-0.6mg/hour)

How do I get help if someone is in respiratory distress (hypoxemic, working hard to breathe, or hypercarbic)?

- Call a rapid (Ask the floor coordinator or a RN to have it paged out)
- Page respiratory (can ask floor coordinator to overhead a stat page if needed) from paging directory
- Page the medicine senior on (p22337)
- Call RICU (stat if needs emergent intubation or non-urgent) x6-3333
- Call CODE BLUE if severe respiratory distress and you anticipate emergent intubation
  - See the **Whitebook p44-48** for more details on Oxygenation and Ventilation
How do I figure out if my patient really has COPD to begin with?

- If they have PFTs (pulmonary function tests) in the system, these usually reveal obstruction
  - Look for FEV1/FVC absolute ratio (NOT % predicted) of < 0.7
- If they do not have PFTs, a history of current/former smoking OR a diagnosis of asthma in adulthood (sometimes this can be mistaken for COPD) can raise suspicion

How do I know that my patient is having a COPD exacerbation?

- A COPD exacerbation is defined clinically by 2 or more of:
  - Increased shortness of breath or oxygen requirement
  - New cough or worsening of baseline cough
  - Increased sputum production or change in sputum color

How do I assess a patient I am admitting with a COPD exacerbation?

- Take a good history
  - How much do they smoke? How far can they walk (or # stairs climbed) at baseline compared to now?
  - Do they use BiPAP at home? Nocturnal CPAP? What are their settings?
- Do a thorough respiratory physical exam
  - Examine for increased respiratory effort (accessory muscle use, belly breathing)
    - Increased work of breathing may require BIPAP OR intubation; DISCUSS WITH YOUR TEAM
  - Wheezing or poor air movement may be present; crackles should make you think of alternative dx
- Obtain labs/imaging
  - VBG +/- ABG >> specifically, assess pH and pCO2
    - Patients with severe COPD will often have baseline elevated pCO2 (but with normal pH)
    - Acidosis from high CO2 & altered mental status may require BIPAP OR intubation; discuss as team
    - ABG necessary if concern for acidosis more than hypoxemia—which we typically monitor on pulse ox
  - CBC + diff >> leukocytosis may indicate concomitant or alternative diagnosis of pneumonia
  - Flu/RSV +/- respiratory viral testing (restricted now for COVID) >> viral infections can trigger COPD
  - Other tests to consider: NT-proBNP, troponin, EKG >> help rule out heart failure or other cardiac cause

How do I treat a patient with a COPD exacerbation?

- Bronchodilators
  - HOLD home inhalers; START DuoNeb (albuterol/ipratropium) q4h standing + q2h PRN
  - NOTE: albuterol may cause tachycardia; in patients w/ high HR, may need to use ipratropium alone
  - As patient improves, SPACE out DuoNebs & RESTART home inhalers (if the patient was not on home inhalers, you may also START one; see here for guidance)
- Steroids: can give prednisone 40mg qD x 5 days (NOTE: steroids can worsen leukocytosis and can cause a persistent or new increase in neutrophils and bands)
- Antibiotics:
  - Typically, azithromycin 250mg qD x 5 days to help decrease inflammation
  - If you are concerned clinically about a pneumonia, see here for community acquired pneumonia or here for hospital-acquired pneumonia or aspiration pneumonia

What do I do when my patient is nearing discharge?

- When the patient’s oxygen requirement is back to their baseline at rest, have RN or PT walk the patient to get ambulatory O2 sats; if the pt desaturates below 88%, may need more treatment vs. go home with (more) O2

See Whitebook p54 for more details on COPD
Venous Thromboembolism (DVT/PE)

When do I order DVT prophylaxis?:
Typically ordered for everyone without contra-indications (upcoming procedure, severe thrombocytopenia)
• LMWH (Lovenox) if the patient has good kidney function (40mg SC daily)
• Heparin if the patient has renal failure or changing kidney function ex: significant AKI (5000U bid)
• Consider Sequential Compression Devices / SCDs (very few contra-indications!); okay to order with above

When do I worry about a DVT and what do I do?
• Asymmetric edema in single extremity, swelling > pain, erythema (venous congestion in extremity)
• Patient has risk factors: immobility, coagulopathy, malignancy, spinal cord injury, obesity, OCPs, lines
• Order venous duplex ultrasound of the concerning extremity >> Study is positive for a DVT ONLY if it’s in a deep vein; superficial veins don’t count (NB: superficial femoral vein is a DEEP vein)
• If the study is positive, start anti-coagulation (details at end of the page)

When should I worry about PE and what do I do?
• The patient has dyspnea, sinus tachycardia, pleuritic chest pain, hypoxemia, hemoptysis w/o other cause
• The patient has increased risk factors for thromboembolism (immobility, malignancy, OCPs, etc.)
• Determine and order best imaging study (usually CT-PE if renal function allows), can do bilateral LE ultrasounds to search for DVT (but remember sensitivity and specificity of this for PE is poor)

What do I do when a patient has a PE?
• Data: Monitor vitals (HR, BP, SpO2 closely), obtain ECG, troponin x 2, NT-proBNP. Consider TTE
• Complete PE Risk Stratification (Note that clot burden is important but risk stratification is ONLY by hemodynamic stability and signs of right heart strain (ECG shows new right bundle branch block or T-wave inversions in V2-V4, elevated troponin or NT-proBNP, CT or TTE evidence of right heart strain)

<table>
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<tr>
<th>MASSIVE PE (right heart strain and hypotension; unstable patient)</th>
<th>SUB-MASSIVE PE (right heart strain, but hemodynamically stable)</th>
<th>NON-MASSIVE PE (no right heart strain and hemodynamically stable)</th>
</tr>
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<tbody>
<tr>
<td>- Immediately consult PERT (Pulmonary Embolism Response Team) by calling x47378</td>
<td>- Initiate anti-coagulation</td>
<td>- Initiate anti-coagulation. Consider surveillance alone if subsegmental PE with no proximal DVT, low risk for recurrent VTEs (venous thromboembolisms)</td>
</tr>
<tr>
<td>- Will require thrombolysis and anti-coagulation &gt;&gt; ICU patient</td>
<td>- Consider thrombolysis or catheter-directed thrombolysis via urgent PERT consult (Pulmonary Embolism Response Team) by calling x47378</td>
<td></td>
</tr>
</tbody>
</table>

Starting Anti-coagulation
• Start anti-coagulation (per flow below): Please check with pharmacy when dosing ANY anti-coagulation
• Do NOT start anti-coagulation if absolute contra-indications exist (ex: Plts < 50, or recent intracranial hemorrhage). In this case, call the PERT team.
• If patient needs thrombolysis or ability for quick cessation of anti-coagulation for procedure etc, use heparin gtt with bolus, PTT in goal for VTE
• Patient has an underlying malignancy: Use LMWH (Lovenox)
• Patient has APLAS (Antiphospholipid Antibody Syndrome): start Warfarin (pharmacy helps dose) with heparin or lovenox until goal INR (>2) is achieved for 48 hours
• Otherwise use a Direct Oral Anti-coagulant (DOAC) with load, ie: apixaban (BID, preferred in patients with renal disease, 1 week of 10mg BID followed by 5mg BID) or rivaroxaban (qD, 3 weeks of 15mg BID followed by 20mg qD)
• Length of anti-coagulation treatment: (consider hematology consult)
• 3 months if provoked, potentially indefinitely if underlying malignancy exists

See Whitebook p57 & p58 for more details on DVT/PE diagnosis and management
How do I know if my patient has sepsis?
• Patient may look SICK; may or may not have clear source of infection
• Evaluate for 2/3 SIRS criteria (HR>90, RR>20, temperature >38°C/<36°C, or WBC>12k/<4k) + Infectious source; criteria does miss 1 in 8 septic patients
• Evaluate for 2/3 qSOFA (Sequential Organ Failure Assessment): 1) RR > 20, 2) AMS, 3) SBP< 100
• If hypotensive, evaluate for other causes of hypotension (see Hypotension page and ACCESS below)

How should I work up my potentially septic patient?
• Examine for a source: Cough, low SpO2, crackles (pneumonia), abdominal pain, CVA tenderness (pyelonephritis), skin, extremities (SSTI - if cool, think about cardiogenic shock)
• Studies: blood culture x 2 sets, UA/Urine Culture, CXR, MRSA nasal swab & Resp Cx if concern for PNA, EKG, BMP, LFTs, CBC w/ Diff, Lactate, VBG
• Monitor closely: Continuous Telemetry and pulse Ox, HR, MAP (goal >65mmHg), UOP (consider Foley, goal 0.5cc/kg/hr), O2 requirement (if going up with IVF, may need vasopressors sooner)

How do I treat Sepsis?
Sepsis/septic shock are emergencies, so early recognition is critical. USE THE “ADULT SEPSIS ORDER SET”.

1. Antibiotics – FAST and tailored to source and patient
   • Must be IN the patient within one hour. Get blood and urine cultures FIRST if possible
   • Use sepsis order set and past micro data to guide antibiotic choice (worry about resistant organisms if antibiotics < 30d ago, hospitalization < 90d ago, admitted > 5d, chronic care facility resident, HD, or immunocompromised
   • Attempt to narrow antibiotics in 48h with improvement, ideally guided by culture data, usually course of 7-10d sufficient if source addressed.

2. ACCESS AND Resuscitation – FAST (<3 hours) and with re-evaluation
   • Guarantee access with at least 2 large-bore IVs (18G or greater)
   • Start fluids 500mL-1L at a time (goal 30mL/kg initially of LR). Can stop if improving. If low ejection fraction on Echo or history of heart failure, reduce size of boluses to 250-500mL, but still run fast (no cont IVF)
   • Examine the patient frequently (at least every 15-20 minute). Goal to keep MAP > 65mmHg. Monitor lactate, VBG (ph), creatinine, urine output
   • If not improving, ASK FOR HELP (Med Senior or Attending). May need unit transfer for vasopressors.

3. Source control
   • Evaluate early for conditions that require emergent source control (e.g., necrotizing soft tissue infections, peritonitis, cholangitis, intestinal infarctions, abscess, obstructive renal stone w/ hydro)
   • Consider removing lines if bacteremia (PICCs, Ports, etc) but make sure you have other access first
   • Consider imaging if not improving with appropriate antibiotics

What are some special cases I don’t want to miss?
• Chronic Steroids – give sick dose steroids up front (3x home dose) or if persistent shock/hypotension, give stress dose steroids 50mg hydrocortisone IV q6h
• Immunosuppressed: Send 1,3 Beta D- glucan, Galactomannan, CryptoAg to assess for fungemia
• Special consults: Endocarditis –ID, Cirrhosis –early Med Senior or attending
  See Whitebook p61 for more on sepsis and Whitebook p103 for more on empiric antibiotics.
When should I suspect my patient is having an upper GI bleed?
Clinical symptoms a patient may report include hematemesis, melena, or hematochezia (if brisk UGIB).
Perform rectal exam; it will be important to know if patient is having melenic stools if/when you speak with GI

My patient has a GI bleed. What should I do?
• If your patient is unstable or actively bleeding frequently/large amounts, CALL RAPID RESPONSE AND:
  o ENSURE APPROPRIATE ACCESS: 2 LARGE BORE IVs (this means 16G or 18G). If a patient has a PICC line, you still must have two 18G PIVs as a PICC does not infuse as quickly
    ▪ 16G = gray
    ▪ 18G = green
    ▪ 20G = pink
    ▪ 22G = blue
  o START RESUSCITATION:
    ▪ Transfuse to the following goals: Hgb > 7 (NOTE: in an acutely bleeding patient, Hgb drop will lag by several hours on CBC) or 8 (if active cardiac pain, Platelet > 50k, INR < 2, fibrinogen > 100. If transfusing more than 3-4 units of pRBC, call blood bank to activate massive transfusion protocol
    ▪ How to get blood products to the room STAT:
      • Emergent blood product form is at the front desk of every unit
      • Delegate runner to take patient sticker and form to blood bank (Jackson 2)
      • Important: call blood bank to let them know someone is coming for blood
        ▪ Run LR (or any available IVF) wide open while waiting for pRBC
  o STAT page RICU if concerned for airway compromise
  o STAT page GI for potential endoscopic intervention (may need to call IR depending on source of bleed and need for embolization)
  o Obtain labs:
    ▪ CBC q8h (depending on rate of bleeding may make less frequent)
    ▪ BMP
    ▪ H pylori Ab
    ▪ Active type and screen
    ▪ PT/INR (does not need to be daily but on admission and then if patient appears to be getting sicker)
• Once/if your patient is stable:
  o Continue to ensure appropriate access, adequate resuscitation, and appropriate lab orders AND/OR consults as noted above
  o Also make sure to order:
    ▪ IV PPI BID (pantoprazole 40 mg IV BID) – can switch to PO if good PO intake
    ▪ For cirrhotic patients with suspected variceal bleed:
      • Ceftriaxone 1g q24h x 7 days for bacterial translocation prophylaxis
      • IV Octreotide: 50 mcg x 1 IV, followed by infusion x 3-5 days

What causes an acute upper GI bleed?
Some causes include peptic ulcer disease (caused by alcohol, H.pylori, NSAIDs), gastritis/esophagitis, varices (think in relation to cirrhosis), Mallory-Weiss tear (think in relation to alcohol use), neoplasm

See Whitebook p66 for more details on upper GI bleed
What does a lower GI bleed look like?
Most commonly it’s maroon colored stools, bright red blood per rectum, or blood clots in the stool.

Important note: patients frequently presents with a lower Hgb than their baseline and can’t tell if they have blood in their stools or not

Where is the blood coming from?
• Usually from the distal parts of the small bowel, colon, or rectum.
• If the patient has no abdominal pain, the cause is either diverticulosis (most common), arteriovenous malformation, or internal hemorrhoids. If the patient has abdominal pain, causes include ischemic colitis, IBD, or infection (especially if the patient is febrile).

How do I manage a lower GI bleed?
• Check vital signs and make sure patient is not hypotensive or orthostatic. If patient is hypotensive then manage as an upper GI bleed to be safe regardless of the history. Brisk upper GI bleeds can look like lower GI bleeds and warrant emergent management
• ACCESS: 2 LARGE BORE PIVs (this means 16G or 18G). If a patient has a PICC line, you still must have two 18G as a PICC does not infuse as quickly
• Always perform a rectal exam to assess for hemorrhoids (do NOT do this if the patient has neutrophil count <500)
• Orders: always have an active type and screen, CBC, BMP, PT/INR (does not need to be daily but on admission and then if patient appears to be getting sicker).
• Check a CBC at least twice daily or more frequently if Hgb dropping quickly. Be conscious that checking q6-8 may also cause iatrogenic drop in Hgb.
• Transfuse to keep hemoglobin >7 (or >8 in active CAD), Platelets >50, INR<2.
• Consult GI (and/or IR); they will provide you with further instructions. If you are asked to “prep” the patient for colonoscopy, look for the colonoscopy order set in epic. Other studies that GI may request may be a video capsule endoscopy, CT angio, or tagged red blood cell scan
• The Unstable/Actively Bleeding Patient: Get help > RAPID RESPONSE
  o STAT page GI for potential endoscopic intervention (may need to call IR depending on source of bleed and need for embolization)

See Whitebook p67 for more details on lower GI bleeds
How do I know my patient has pancreatitis?

Patient must present with any two of the following three signs/symptoms:

- Abdominal pain or nausea/vomiting (typically epigastric radiating to the back)
- Lipase or amylase > 3x ULN
- Findings on CT (radiologist says its pancreatitis)

What tests do I order when the patient is admitted?

- On admission: CTAP, lipase, triglycerides
- Daily: CMP; no need to trend lipase (not prognostic)

What are the most common causes of pancreatitis?

1. Gallstones/sludge> eval with RUQUS (right upper quadrant ultrasound-order via limited abdominal us) v CT
2. Alcohol
3. Hypertriglyceridemia: check triglycerides, (may be elevated due to pancreatitis itself)
4. Post-ERCP
5. Refer to this [diagnostic schema](#) if none of the above

How do I manage the patient when they get to the floor?

1. **Pain control**:
   - OK to use IV opioids (hydromorphone preferred); remember bowel regimen

2. **Reverse precipitants (not always applicable)**:
   - Treat ↑Ca or ↑TG
   - If there is a stone on CTAP > urgent (24-72H) ERCP >>page GI
   - Talk to pharmacy about possible meds that cause pancreatitis

3. **IV Fluids** (severe hypovolemia from 3rd spacing)
   - Start LR: Give a 2L bolus + start gtt (150-250cc/hr for 48 hours); LR >> NS
   - If history of heart failure or pulmonary hypertension, check respiratory status (needing O2 or not); may need to DECREASE fluid rate if they have increasing oxygen requirements

4. **Nutrition**:
   - Start PO (low-fat diet) once no n/v or abd pain
   - At 96h if patient still not tolerating feeds >> think about starting tube feeds (not TPN) & repeat CT scan

What complications should I watch out for?

- **If hypotension**
  - think sepsis, give more fluids; start ceftepime + metronidazole (talk to pharmacy about dosing) repeat CT
  - think pseudoaneurysm if signs of bleeding (Hb drop, hematochezia), STAT CTA and IR consult

- **If new/worsening oxygen requirement**
  - think either volume overload or developing ARDS (known complication)
  - Get a CXR and ASK FOR HELP

- **If severe non-resolving abdominal pain (after 3 days in house)**
  - consider repeat CTAP, starting cefepime and metronidazole, and GI consult

- **Daily:** check the electrolytes: replete the magnesium and calcium if needed

- If a patient is >10 days out from an episode of pancreatitis, think about late complications and refer to the **White Book** (link below)

See [Whitebook p77](#) for more details on pancreatitis
What are some basics I should know about liver function tests?

- **LFTs** – AST, ALT, Alkaline Phosphatase (ALP), Bilirubin (bili), Albumin
  - PT-INR and platelets (plt) are also markers of true liver “function”
- **Acute Liver Injury (ALI)** – ↑AST, ALT, ALP, bili, +/- PT-INR. indicative of damage to the liver
- **Acute Liver Failure** – ALI + encephalopathy + INR >1.5 in pt w/o pre-existing liver disease (often needs ICU)
- **Cirrhosis** – chronic, related to repeated ALI from a variety of causes. Pts w/ cirrhosis will not necessarily have ↑AST, ALT, ALP, or bili; will often have ↑PT-INR, ↓platelets, ↓albumin. See End-Stage Liver Disease

How do I start to figure out what’s going on?

1) Run medication list through Liver Tox (see below for common culprits) and take history for toxins
2) Consider non-hepatic disease (rhabdo, HF, hemolysis, bone turnover) and send relevant tests below
3) Order RUQUS (Right upper quadrant ultrasound; order in epic under limited abdominal ultrasound) with Doppler, serum and urine toxicology screen
4) Consider basic hepatitis panel, iron studies, MRCP (only if cholestatic pattern of liver injury, below)
5) Consult GI if these studies are unrevealing or are suggestive of less common liver disease

Is there a more systematic way to think about what’s going on?

YES! Use the R-Ratio to determine pattern and then see below (causes) for a differential diagnosis.

- **Hepatocellular** (↑AST & ALT relative to ALP +/- ↑bili). **Cholestatic** (↑ALP relative to AST & ALT +/- ↑bili).
- **Infiltrative** pattern has ↑ALP with nl bili. Often, the picture is MIXED.
- **Special Patterns:**
  1) AST, ALT >1000: DDx includes shock, TYLENOL toxicity, acute viral hepatitis, Budd-Chiari syndrome, AIH, or rhabdomyolysis. 2) AST:ALT ≈ 2:1: commonly seen with EtOH-associated ALI

Very common causes of LFT abnormalities and additional workup (*indicates definite need for GI c/s):

- **Medications.** ALWAYS check Liver Tox; Common culprits include antibiotics, anti-epileptics, antidepressants, statins, amiodarone, TPN. Tylenol in ↑doses. Sometimes requires GI c/s.
- **Toxins.** Unregulated OTC supplements, EtOH, cocaine. Sometimes requires GI c/s.
- **Congestive Hepatopathy.** ↑R-sided pressures in heart cause congestion of liver and damage. Order TTE.
- **Biliary obstruction*.** Cholangitis or choledocholithiasis. RUQUS can show CBD dilation, MRCP if negative.
- **Rhabdomyolysis/muscle damage.** ↑AST, ALT in muscle. Send CK to determine if muscle injury present.
- **Hemolysis.** RBCs contain bili (mostly ‘indirect’) and AST. Special slide, haptoglobin, LDH are helpful.
- **NAFLD*.** No additional action needed, but somewhat a diagnosis of exclusion. RUQUS can show liver fat.
- **Viral illness or viral hepatitis*.** Nonspecific viral illnesses may cause ↑LFTs. Specific viruses include Hep A, B, C (D&E are very rare), EBV, CMV (more common), HSV, VZV (more rare). Can send viral serologies.
- **Malignancy.** Metastases or primary liver cancer (Hepatocellular carcinoma). RUQUS, CT, or MRI helpful.
- **Bone.** ↑ALP due to bone turnover. Other LFTs normal. ↑GGT correlates with ↑ALP if from liver.

Less common causes of LFT abnormalities (most managed with GI c/s):

- **Autoimmune hepatitis (AIH)**
- **Budd-Chiari Syndrome**
- **Celiac disease**
- **Hemochromatosis**
- **Wilson’s Disease**
- **Alpha-1-Antitrypsin Deficiency (A1AT)**
- **Primary biliary cholangitis (PBC)**
- **Primary biliary cirrhosis (PSC)**
- **Sarcoidosis**
- **Amyloidosis**

See WhiteBook p78 for more details on LFT abnormalities
Where do I start if I think/know my patient has cirrhosis?

• Chart biopsy for diagnosis/etiology of cirrhosis and recent EGD (to look for varices)
• Evidence of a cirrhotic liver on CT scan, ultrasound, or biopsy
• On history: why do they have cirrhosis? (alcohol, hepatitis B/C, non-alcoholic steatohepatitis (NASH), autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis)
• On exam: may see jaundice, pruritus, abdominal distention, lower systolic BP, splenomegaly, ascites, spider angiomata (>3), gynecomastia, testicular atrophy, palmar erythema, asterixis
• Labs: ↑TBili, ↑INR, ↓Alb, ↓Na, ↓platelets, LFTs may be normal. If unknown etiology of cirrhosis, can send Hep B/C serologies, ANA, ASMA, AMA, α1AT, ceruloplasmin

How do I know if their cirrhosis is decompensated?

• Look for the following signs/symptoms: new/worsening hepatic encephalopathy (sleep/wake disturbance, poor attention), asterixis, worsening jaundice/LFTs, new or worsening ascites/LE swelling, new GI bleed
• Look for cause of decompensation: infection (spontaneous bacterial peritonitis vs other systemic infection, acute alcohol use, medication/dietary noncompliance (should adhere to low salt diet)

How do I manage decompensated cirrhosis?

Use the VIBES Framework below:

• Volume (ascites, edema, hepatic hydrothorax, hepatorenal syndrome)
  o Treatment of volume overload: 2g Na restriction, diuretics (if initiating, 50mg/day spironolactone + 20mg/day furosemide is usual starting dose (5:2 ratio)), therapeutic LVP: indicated for tense or refractory ascites; if > 4L, transfuse 1 bottle of 25% albumin for every liter removed
  o Monitor Cr for hepatorenal syndrome (HRS). Click HRS for more info. Consult Renal if concerned
• Infection (Spontaneous Bacterial Peritonitis-SBP)
  o Must rule out acute SBP in all patients with ascites (even if not reason for admission) -> obtain diagnostic paracentesis (SBP diagnosis is >250 PMNs in the ascites fluid)
  o Treatment of SBP: CTX 1g q24h x 5d. If Cr >1, BUN >30, or TBili >4, also give 25% Albumin (1.5 g/kg on day 1 & then 1.0 g/kg on day 3). Discontinue beta-blockade indefinitely.
  o Prophylaxis: IV CTX 2g q24 x 7 days if GI bleed; secondary prophylaxis with cipro 500mg daily after
• Bleeding (esophageal/gastric varices, portal hypertensive gastropathy, coagulopathy)
  o Acute upper GI bleeding: see Upper GI Bleeding & call GI for urgent EGD if concerned for variceal bleed
• Encephalopathy (portosystemic encephalopathy)
  o Identify current severity, trend, precipitant >> check asterixis and attention (days of week backward)
  o Treatment: Treat precipitant, anti-delirium precautions, Lactulose (30mL q2h until BM → titrate to ~ 3-4 soft BM qD- increase/decrease based on mental status, consider adding rifaximin if not improving
• Screening/Surgery (transplant)
  o Vaccinations: HAV, HBV, Influenza, Pneumovax, Prevnar (and up-to-date on all other vaccines)
  o HCC screening with q6m RUQUS/MRI + AFP

What serious things should I look out for?

• Worsening altered mental status – if not protecting airway > CALL RAPID RESPONSE + RICU
• Hypotension with worsening mentation > CALL RAPID RESPONSE, may need ICU transfer for pressors, can give fluid/albumin gingerly
• Worsening Cr or oliguria/anuria > consult Renal

See Whitebook p83 for more details on ESLD
What is an acute kidney injury (AKI)?
AKI is defined as:
- An increase in serum creatinine (SCr) ≥ 0.3 mg/dL within 48 hrs
- An increase in SCr ≥ 1.5x baseline within 7 days
- Urine output (UOP) < 0.5 mL/kg/hr for 6 hrs

OK; my patient has an AKI; what could have caused it?

<table>
<thead>
<tr>
<th>PRE-Renal (21%)</th>
<th>INTRINSIC RENAL</th>
<th>POST-Renal (10%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Due to inadequate flow to kidneys</td>
<td>Due to damage to the kidney structures</td>
<td>Poor outflow from the kidney or bladder</td>
</tr>
<tr>
<td>True hypovolemia:</td>
<td>Tubulointerstitial ATN: (45%)</td>
<td>Vascular Microvasc (&lt;4%)</td>
</tr>
<tr>
<td>- Dehydration (poor PO)</td>
<td>- Sepsis</td>
<td>- TTP/HUS</td>
</tr>
<tr>
<td>- Bleeding</td>
<td>- Ischemia</td>
<td>- APLAS</td>
</tr>
<tr>
<td>Effective hypovolemia (3rd spacing):</td>
<td>- Toxins (contrast, rhabdo, meds)</td>
<td>- Malig HTN</td>
</tr>
<tr>
<td>- Sepsis / SIRS</td>
<td></td>
<td>- Meds</td>
</tr>
<tr>
<td>- CHF / cardiorenal</td>
<td>AIN: (2%)</td>
<td>Macrovasc</td>
</tr>
<tr>
<td>- Cirrhosis / hepatorenal</td>
<td>- Meds, infxn</td>
<td>- RAS</td>
</tr>
<tr>
<td>Impaired blood flow:</td>
<td>Crystallization:</td>
<td>- Dissection</td>
</tr>
<tr>
<td>- NSAIDs, ACEi / ARBs</td>
<td>- TLS, acyclovir</td>
<td>- Thrombosis</td>
</tr>
</tbody>
</table>

What do I do and how do I figure out what is causing this AKI?
- First, check to make sure that your patient does not need renal replacement (dialysis) due to:
  - Volume overload > hypoxemia
  - Bad/worsening hyperkalemia – look at BMP
  - Bad/worsening acidosis – send VBG
- Obtain a good history focused on risk factors for pre-renal losses (poor PO intake, high GI losses, infections, diuretic use/lack thereof), trauma history, as well as a good medication history
  - STOP/AVOID meds that could be causing AKI (see above/ask pharmacy) & dose medications renally
- Do a good exam focused on volume status
  - If hypovolemic, give back fluids (UNLESS also hypoxemic; send labs below BEFORE IV fluids. Caution with patients with very low EFs on Echo (<40%)-can give back fluid gingerly)
  - If hypervolemic, consider diuresis. Can ask hepatology/cardiology for help PRN
  - If euvolemic:
    - Rule out obvious post-renal issues – bladder scan; consider renal ultrasound
      - NOTE: you have to have bilateral ureteral blockage to cause AKI
    - Send urine studies (sodium, urea nitrogen, creatinine) and calculate a FENa OR FEUrea (for patients on diuretics) to confirm your suspicion
    - If FENa > 2% or FEUrea > 35% (intrinsic cause), obtain UA and ask for help with spinning urine
      - See here for help in interpreting UA or how to spin urine

What do I do to manage this AKI?
- Treat underlying cause; consult renal if cause is still not clear or help with intrinsic causes (other than ATN)
- Monitor creatinine daily; follow urine output and volume status closely (give back fluid if dry, avoid hypotension). Consider placing Foley if needed.

See Whitebook p89 for more details on AKI
What do you mean when you say IV Fluids?

- General types:
  - Crystalloid (NS or LR)
  - Free Water (D5W)
  - Colloid (Albumin or Blood Products) - NOT superior to crystalloid for volume resuscitation in sepsis

When and how do I give IV fluids?

  - **Fluid Choice**: in general, we prefer LR over NS
  - **Rate**: 500cc-1L over 30 min-2 hours; can trial smaller volume if risk of volume overload (i.e. HF history)

- **As a maintenance** to replace daily fluid loss (~1.6L/d in normal adults; in pts w/ fever, pancreatitis, etc)
  - **If a patient is taking POs, typically no need for maintenance IV fluids**
  - **Fluid of Choice**: D5 1/2NS; NOTE: provides insufficient caloric replacement (~170 kcal/L)
  - **Rate**: 60 ml/hr + 1 ml/kg/hr for every kg above 20 kg. I.E. for a 60 kg adult = ~100 ml/hr
  - **Duration**: Always set an end time or timed duration; indefinite fluids are dangerous

When can I use albumin?

- Use only to replace serum oncotic pressure, NOT to give volume
- Indications include spontaneous bacterial peritonitis, large volume paracentesis, hepatorenal syndrome, etc; see Whitebook p100 for dosing & more details on albumin

When and how do I replete electrolytes?

- **Potassium**: in general, 10 mEq K increases serum K by 0.1
  - **Goal**: >4 for CAD/Arrhythmias; >3.5 for everyone else. NO repletion if on hemodialysis, unless <3.0
  - **Options**: Use PO > IV. Packets = immediate release (q4-6hr). Pills = extended release; give x 1, recheck
  - **Max dosing**: 80 mEq at once before rechecking K. Remember to correct hypoMg
  - **Rate**: max by PIV: 10 mEq/hr; by central line: 20 mEq/hr (requires telemetry)

- **Magnesium**: 2 g increases serum Mg by 0.5 (note that hypoMg can cause hypoK, hypoCa)
  - **Goal**: CAD/Arrhythmia: >2.0-2.2, >1.7 for everyone else
  - **Options**: Use IV > PO (PO may cause diarrhea). Mag sulfate 2g IV OR Mag oxide 400 mg PO TID x 1 day

- **Phosphorus**: 
  - **Goal**: Replete if patient is symptomatic, phos < 1 OR < 2 if patient is at risk for refeeding syndrome
  - **Options**: PO > IV. K-Phos 1 packet QID or Neutra-Phos 1 packet QID.
    - For IV, K-Phos (1.5 mEq K/mmol phos) OR Na-Phos (1.3 mEq Na/mmol phos); give 15-45 mmol phos at a time. NOTE: IV dosing can precipitate calcium and cause hypoCa

- **Calcium**: 1 g of Ca gluconate increases serum Ca by 0.5
  - **Goal**: Replete if patient is symptomatic or has prolonged QTc (> 500), Ca < 7.5, or iCa < 1
  - **Options**: IV if severe; PO if mild. Ca gluconate 1-2 gm IV or Ca Carbonate 1250 mg PO BID
  - **NOTE**: phosphate binds calcium; consider management of hyperphosphatemia

See Whitebook p100 for more details on IVF and electrolytes
## INPATIENT EMPIRIC ANTIMICROBIAL THERAPY

**THINK ABOUT:**
- **Host** (e.g. immunocompetent vs. neutropenic vs. recent exposure to antibiotics)
- **Prior micro data** (did they have multi-drug resistant organisms in the past?)
- **Local susceptibilities** (see MGH antibiogram [here](#))
- **Clinical syndrome** (e.g. CAP vs. VAP vs. meningitis)

The goal is to eventually go from EMPIRIC to TARGETED therapy based on culture results

For more information click on each syndrome (e.g. meningitis) to access the specific white book page

<table>
<thead>
<tr>
<th>Suspected Clinical Syndrome</th>
<th>Microbiology</th>
<th>Empiric Antimicrobial Therapy</th>
<th>Additional Info</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Meningitis</strong></td>
<td>- Viral, HSV, S. pneumo &gt; N. meningitis. - Listeria for &gt;50 y/o or immunocompromised - If hardware or nosocomial: Staph, PsA; P. acnes if VP shunt</td>
<td><strong>General:</strong> Vanc AND CTX 2g Q12 Age ≧50 yo: add ampicillin <strong>Immunocompromised:</strong> Vanc + cefepime (or meropenem) + amp <strong>If concern for HSV:</strong> add Acyclovir</td>
<td>Dex 10 mg PO/IV q6h x 4 days w/ initial abx dose if S. pneumo</td>
</tr>
<tr>
<td><strong>Community Acquired</strong></td>
<td>- Viral (most common), S. pneumo, H. flu, Moraxella, S. aureus, Legionella, Mycoplasma, Chlamydia, Klebsiella,</td>
<td><strong>Ceftriaxone + Azithromycin OR Levofloxacin</strong></td>
<td>Consider COVID 19 or flu testing + Oseltamivir</td>
</tr>
<tr>
<td><strong>Pneumonia (CAP)</strong></td>
<td>- CAP organisms (as above) + S. aureus + GNRS including PsA</td>
<td>Vanc + Cefepime</td>
<td>IF MRSA nasal swab negative can consider stopping vanc</td>
</tr>
<tr>
<td><strong>Hospital-acquired &amp;</strong></td>
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<tr>
<td><strong>Ventilator-Associated</strong></td>
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<tr>
<td><strong>Pneumonia (HAP/VAP)</strong></td>
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<tr>
<td><strong>Endocarditis</strong></td>
<td>- Native valve: S. aureus, Strep, Enterococcus, few GNRS, HACEK &lt;5% -Prosthetic: S. aureus, S.epi</td>
<td><strong>Native valve:</strong> Vanc + CTX <strong>Prosthetic:</strong> Vanc + Gent (or Vanc + CTX if prosthetic valve &gt;1 year)</td>
<td>For MSSA: β-lactam &gt;&gt; Vanc</td>
</tr>
<tr>
<td><strong>Cholecystitis/Ascend.</strong></td>
<td>- Often polymicrobial; broad abx for 48h even if BCx growing 1 org. - Common bugs: E. coli, Klebsiella; less likely Enterococcus, anaerobes.</td>
<td><strong>Ceftriaxone + Metronidazole OR Pipercillin-tazobactam</strong></td>
<td>If nosocomial consider cefepime+ flagyl. Source control (ERCP/per chole) is critical for treatment</td>
</tr>
<tr>
<td><strong>Cholangitis</strong></td>
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</tr>
<tr>
<td><strong>Other Intra-abdominal (e.g. abscess/diverticulitis)</strong></td>
<td>GNRS, anaerobes, Enterococ, Candida; S. aureus, Strep rare</td>
<td>[Ceftriaxone or Cipro] + Metronidazole OR Pipercillin-tazobactam</td>
<td>If nosocomial consider cefepime+ flagyl. Source control (drainage) is critical for treatment</td>
</tr>
<tr>
<td><strong>Spontaneous</strong></td>
<td>- Enteric GNR, includ Enterobacter, Strep, Enterococcus; rarely anaerobes</td>
<td>Ceftriaxone</td>
<td>Cipro reserved for beta-lactam allergies and for prophylaxis</td>
</tr>
<tr>
<td><strong>Bacterial Peritonitis</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>UTI / urosepsis</strong></td>
<td>E.coli, Klebsiella, S.saprophyticus, Proteus (check prior culture data for history of ESBL in urine)</td>
<td><strong>General:</strong> Ceftriaxone <strong>Cefepime (if c/f pseudomonas)</strong></td>
<td>- Carbapenem if hx of ESBL - ADD Vanc if recent instrumentation</td>
</tr>
<tr>
<td><strong>Catheter-Associated</strong></td>
<td></td>
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</tr>
<tr>
<td><strong>UTI (CAUTI)</strong></td>
<td>- GNR’s, Enterococcus -Prior culture data useful</td>
<td><strong>General:</strong> CTX AND Vanc; consider PsA if risk MDRO, hosp. acquired</td>
<td>Tx only if sx; repeat UA/UCx 48 hrs after removal or replacement</td>
</tr>
<tr>
<td><strong>Osteomyelitis</strong></td>
<td>- S aureus&gt; Strep, PsA (diabetic), GNR, Enterococ, Eikenella (human bites), Pasteurella (animal bites)</td>
<td><strong>No abx until after bone bx+cx unless HD unstable/ severe neuro symp</strong> <strong>General:</strong> Vanc</td>
<td>ADD CTX or Cefepime if DM/PVD/ Ulcer</td>
</tr>
<tr>
<td><strong>Septic Arthritis</strong></td>
<td>- Staph, Strep, N. gonorhea (sex. active), E. coli; Salmonella (sickle cell); PsA (IVDU); Lyme, viruses (poly-articular)</td>
<td><strong>Blood + joint aspirate cx prior to abx unless hemodynamically unstable</strong> <strong>General:</strong> Vanc AND CTX</td>
<td>Switch CTX to Cefepime if IVDU, other risk factor for PsA</td>
</tr>
<tr>
<td><strong>Skin/Soft Tissue (SSTI)</strong></td>
<td>- Impetigo: S. aureus &gt; Strep -Cellulitis/Erysipelas: Strep &gt; Staph -Nec Fas: Strep, C. perfringens, MRSA</td>
<td><strong>PURULENT:</strong> Vanc NON-PURULENT: Cefazolin</td>
<td>Consult ortho for joint washout</td>
</tr>
<tr>
<td><strong>Septic shock, no source</strong></td>
<td>- GNRs, S. aureus, Strep, PsA, anaerobes. Consider toxic shock syndrome (TSS)</td>
<td>Vanc + [CTX if Cefepime or Ceftaz] + Metronidazole OR Vanc + Pip-tazo (avoid if AKI or CKD)</td>
<td>-If TSS: Add Clinda 900 IV q8h -MDRO: Meropenem/Impimen</td>
</tr>
</tbody>
</table>
What multi-drug resistant organisms should I know?

Although the list of MDROs is growing, here are 4 important MDROs to know:

- Extended Spectrum Beta Lactamases (ESBL)
  - Any **gram negative rod (GNR)** that is resistant to **ceftriaxone**. Most commonly Klebsiella or E.coli
- AmpC Beta-Lactamases (Cephalosporinases)
  - A type of ESBL; these organisms can easily become resistant to cephalosporins through activation of a gene encoding the AmpC beta-lactamase
  - They may initially appear susceptible to third-generation cephalosporins but then become resistant
  - So what bugs are included in this category
    - The list can be remembered using the mnemonic **SPICE**-M or **SPACE**-M
      - Serratia
      - Providencia
      - Indole-positive Proteus (non-mirabilis species) OR Acinetobacter
      - Citrobacter
      - Enterobacter
      - Morganella
  - In summary, an AmpC-inducible organism is a **SPICE**-M organisms that is resistant to **cefazolin**
- Vancomycin resistant enterococcus (VRE)
- Methicillin resistant staph aureus (MRSA)

How do I figure out if my patient has had an MDRO in the past?

For every patient presenting with any infection, open the **60 DAY MICRO** tab in Epic and review the patients previous culture data to determine if they have had a MDRO

If they have had an MDRO in the past, and are presenting clinically with a similar infection, it is always safest to **treat for the MDRO** until you receive new culture data (in which case you can de-escalate antibiotics)

What antibiotics do I use?

For ESBLs and AmpC organisms:
- If critically ill, empirically treat with **meropenem** AND **consult ID**
- Otherwise, may consider high dose cefepime (2g q8h) if the MIC for cefepime is < 2

For VRE:
- First make sure it is a true infection (often a colonizer in the urine and skin; it is a true pathogen if it is isolated in the blood, abdomen or pelvis)
- If it is a true infection, start **linezolid** or daptomycin AND **consult ID**

For MRSA:
- Treat with **vancomycin** for serious infections (e.g. bacteremia, endocarditis) AND **consult ID**
- Bactrim, clindamycin or doxycycline can be used in simple skin infections

See **Whitebook p105** for more details on drug resistant organisms
Which bugs cause community acquired pneumonia (CAP)?
- **Bacteria:** Strep pneumo (most common for immunocompetent AND immunosuppressed), Haemophilus influenzae, mycoplasma, Staph aureus, Moraxella catarrhalis
- **Viruses**

How do I know if my patient has a pneumonia?
- **Clinical signs/symptoms:** Cough, fever, leukocytosis, hypoxemia + new consolidation on CXR
- **Exam:** New oxygen requirement, crackles on exam (listen anteriorly and posteriorly to all lung fields)
- **CXR:** can be negative or lag behind clinical presentation

If my patient aspirated, do they necessarily have a pneumonia?
- Aspiration itself can lead to inflammation in the lung (pneumonitis) or pneumonia. Both present with same signs/symptoms especially in elderly patients with prior stroke or dementia
- Would start antibiotics either way and if patient improves rapidly <24 hours, likely pneumonitis and may stop Abx (discuss with the team first)

How do I manage a community acquired pneumonia?
- **Orders:** CXR, sputum culture and gram stain, +/- MRSA nasal swab
- If patient has been in the hospital for >48h, cover for hospital acquired pneumonia (HAP)
- Under the “micro tab” review “all available abnormal micro” to see what patients may have grown from a sputum culture in the past
- Culture-directed therapy is always preferred but should certainly not delay treatment (cover empirically as below, and narrow if possible)
- Risk Factors for MDRO: Hospitalized in past 30d; IV antibiotics in past 90d, frequent hospitalizations
- Risk factors for Pseudomonas: GNR on gram stain, history of Pseudomonas, bronchiectasis, COPD with frequent exacerbations
- Risk factors for MRSA: GPC clusters on gram stain, recent flu-like illness, necrotizing/cavitation/empyema

<table>
<thead>
<tr>
<th>Pneumonia</th>
<th>Treatment (5-7d)</th>
<th>Additional Information</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CAP</strong></td>
<td>CTX 1g QD <strong>AND</strong> Azithro 500 mg on day 1 then 250mg x4d OR Levofoxacin (750mg) QD</td>
<td>Amp/sulb (Unasyn) can replace CTX <strong>Duration:</strong> 5 days</td>
</tr>
<tr>
<td><strong>CAP with MDRO risk factors</strong></td>
<td>Anti-pseudomonal (cefipime)+ anti-MRSA (vancomycin)</td>
<td><strong>Duration:</strong> 5-7 days</td>
</tr>
<tr>
<td>Or HAP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspiration</td>
<td>Standard CAP/HAP treatment (+/- anaerobic coverage (metronidazole) only if abscess or empyema is suspected)</td>
<td>Aspiration pneumonitis will resolve rapidly after transient pneumonia-like symptoms and does not need to be treated with antimicrobials for full course (see above)</td>
</tr>
</tbody>
</table>

- If no response to therapy after 72h: consider chest CT (+/- BAL) to eval for empyema, abscess, fungal infxn

**Do NOT recommend:** Corticosteroids or follow up imaging to see if pneumonia has “cleared”. Can get repeat CXR 4-6 weeks after discharge.

See Whitebook p106 page for more on CAP and Whitebook p107 for more on HAP.
Cellulitis/SSTI

**What skin findings should make me think about cellulitis?**
- Erythema, warmth, tenderness, edema, induration (+/- purulence or fever); usually NOT bilateral

**What labs or imaging should I send?**
- Blood and wound culture typically not recommended; obtain if: evidence of systemic toxicity, extensive skin involvement, immunosuppressed, special exposures (bites, water), recurrent/persistent cellulitis
- Consider ultrasound to assess for presence of abscess

**What antibiotics should I start? What bugs should I target?**

<table>
<thead>
<tr>
<th>Mild</th>
<th>Purulent (MRSA &gt; MSSA)</th>
<th>Non-purulent (Strep &gt;&gt; S. aureus)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I&amp;D only</td>
<td>cephalixin OR clindamycin (PCN allergy)</td>
<td></td>
</tr>
</tbody>
</table>

| Mod | (systemic signs, abscess > 2cm or abscess w/ overlying infxn) | I&D + culture (Cx) + TMP-SMX OR doxycycline OR clindamycin | cefazolin OR ceftriaxone (both are IV) OR clindamycin (PCN allergy) |

| Severe | (systemic signs AND > 1 of immunocomp, low BP, rapid spread) | I&D + Cx + vancomycin OR IV linezol +/- clinda (for toxic shock) | vancomycin + ceftriaxone +/- clindamycin |

- Consider MRSA coverage in pts w/ IVDU, previous MRSA infection/colonization, abx in last 8 weeks, DM
- See here for other Abx to cover pts with cirrhosis, immunocompromise, neutropenia, DM foot infections
- Consider early surgical +/- ID consult if c/f necrotizing fasciitis (calculate LRINEC); start vanc/zosyn/clinda

**How long do I treat?**
- 5-14 days, draw margins to track progress; consult dermatology if not improving (should improve in 3d)

UTI

**When should I think about a UTI?**
- Patient has frequency, urgency, dysuria, malaise, incontinence, nocturia, suprapubic tenderness

**What labs/imaging should I send?**
- BMP, CBC, UA, urine sediment; obtain imaging (CT + contrast > ultrasound) if patient is severely ill, has persistent clinical symptoms after 48-72h of antibiotics, or there is concern for obstruction

**What antibiotics should I start?**
- First off, don’t treat asymptomatic bacteriuria unless the pt is pregnant, 3-6 mo s/p renal transplant, or as prophylaxis for urologic procedures; also fungi are common colonizers; see here about when to treat fungi
- Use prior microbiology data to guide empiric management prior to sensitivities; or empirically:

<table>
<thead>
<tr>
<th>Empiric Antibiotics</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>NFT 100mg BID x 5d OR T/S DS BID x 3d OR Fosfomycin 3 x 1</td>
<td>Avoid NFT if CrCl&lt;40 &amp; empiric T/S (E. Coli is 28% resistant @ MGH)</td>
</tr>
<tr>
<td>[CTX OR CEFE] +/- vanc/linezolid if c/f GPC infection &gt;&gt; Narrow to FQ if improving; Duration: 5-7d for FQ; 7-10d for T/S &amp; beta-lactam</td>
<td>Remove/replace urologic devices e.g. stents</td>
</tr>
<tr>
<td>[CTX OR FQ] AND VANC (risk of MRSA and Enterococcus) Duration: 7d if improving; 10-14d otherwise Alternatives: P/T (if c/f PsA); CBPN if c/f ESBL</td>
<td>Remove catheter ASAP, obtain repeat UA/UCx from new catheter PRIOR to abx</td>
</tr>
</tbody>
</table>

**KEY:** NFT—nitrofurantoin; T/S—TMP/SMX; CTX—ceftixime; FQ—fluoroquinolone; P/T—piperaclillin/tazobactam; CEF—cefepeime; CBPN—carbapenem; AMG—aminoglycoside; CPO—ciprofloxacin; LVO—levofloxacin; FLUC—fluconazole; AmB—amphotericin B; R—resistance

See Whitebook p110 (SSTI) or p109 (UTI) for more details
What should I look for in a patient with suspected bacteremia?

- Fever (> 38 C) or hypothermia (< 36 C), rigors, hemodynamic instability (hypotension, tachycardia), leukocytosis (high % bands), elevated CRP
- Do a head to toe exam focused on identifying source and complications of bacteremia:
  - Sources (Where did the bacteria come from?): Lines (PIV, central line, port-a-cath, Foley catheter), recent procedures/implanted hardware, cellulitis/skin abscess/wounds, poor dentition / dental procedures, PNA, UTI/prostatitis, intra-abdominal infections
  - Complications of bacteremia (Where did the bacteria go?): Meningitis/mycotic aneurysm, endocarditis, osteomyelitis/septic joint, epidural abscess, hardware infections, septic emboli (lungs, spleen, kidney, Janeway lesions)

How do I manage a patient with bacteremia?

- Obtain two sets of blood cultures prior to starting antibiotics: 1st set from a peripheral stick, 2nd set from port or indwelling line if concern for line infection (or another peripheral).
- If patient appears sick, also send UA/urine culture, CXR if pulmonary symptoms.
- Review allergies: If pt has PCN allergy, review test dose protocol (Ellucid) vs consult Allergy.
- Review patient’s history under “Micro” tab – look for a MRSA, VRE, any multi-drug resistant organisms.

<table>
<thead>
<tr>
<th>Organism in blood culture</th>
<th>Empiric antibiotic treatment</th>
<th>Other considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram positive cocci: Clusters/chains (Staph), Pairs/short chains (Strep/Enterococcus)</td>
<td>Start Vancomycin initially If MSSA, switch to β-lactam (e.g. nafcillin)</td>
<td>For Staph bacteremia: Consult ID, consider need for TTE/TEE, think about hardware infection</td>
</tr>
<tr>
<td>Gram positive rods/bacilli: (e.g. Actinomyces, Clostridia species)</td>
<td>Consult ID as resistance patterns vary</td>
<td>More likely true infection in immunocomp. pts, indwelling lines, multiple bottles positive.</td>
</tr>
<tr>
<td>Gram negative organisms: (e.g. E. coli, Klebsiella, Pseudomonas)</td>
<td>Start ceftriaxone (2g Q24h) vs cefepime (if septic or c/f Pseudomonas).</td>
<td>If hx MDRO, consider meropenem.</td>
</tr>
<tr>
<td>Anaerobes (e.g. if intra-abdominal infection, empyema, post-obstructive PNA)</td>
<td>Add metronidazole to regimen (or could use piperacillin/tazobactam monotherapy)</td>
<td>Caution using pip/tazo in pts w/CHF, CKD, or AKI</td>
</tr>
<tr>
<td>Candida (or other fungal infxns)</td>
<td>Start micafungin and consult ID.</td>
<td>Toxic shock = fever, hypotension, rash, multiorgan dysfunction</td>
</tr>
<tr>
<td>If concern for toxic shock</td>
<td>Add clindamycin to regimen above</td>
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</tbody>
</table>

When should I suspect that my patient has endocarditis?

- Risk factors: Bacteremia, IV drug use, poor dentition/recent dental procedures, prosthetic or structurally abnormal heart valves (ask specifically if valve replacement w/in prior 12 months)
- History: Fever, malaise, chest pain, dyspnea, wt loss, night sweats
- Exam: New murmur, septic spots (Janeway/Osler), conjunct hemorrhage, focal back (epidural abscess) or abdominal pain (spleenic/renal infarct)

How do I diagnose and manage endocarditis?

- Obtain a TTE if pt has + blood cultures and/or high suspicion based on exam and risk factors
- Formally diagnose by Duke Criteria: need 2 major criteria OR 1 major + 3 minor OR 5 minor criteria
- Check serial EKGs (lengthening PR interval c/f valvular abscess), daily blood cx until negative
- Antibiotics: Vanc and CTX (2g Q24h). Gentamicin if valve replacement in last 12 months AND normal GFR.
  See Whitebook p112 for more details on bacteremia/endocarditis
HEMATOLOGY

Anemia

How do I figure out what is causing the anemia?

*NB: All labs should be drawn or added on to samples obtained prior to transfusion

**Anemia = low H/H**

Send CBC: if pancytopenic, send B12, folate, ANA, CCP/RF, HIV, HCV, HBV; consider med effect (NSAIDs, PPIs, anti-epileptics, chemotherapy)

Send reticulocyte count: use this to calculate the reticulocyte index; [click here](#)

Send type and screen, PT/INR, PTT

**Low reticulocyte index (<2)**

*Check CBC for MCV*

- **Microcytic (MCV < 80)**
  - Send iron, TIBC, ferritin
  - *Iron deficiency*: ↓ iron, ↑ TIBC, ↓ ferritin (<30)
  - *Anemia of chronic illness*: ↓ iron, ↓-nl TIBC, ↑ ferritin
  - *Thalassemia*: all normal

- **Normocytic (MCV 80-99)**
  - *Early iron deficiency*: send iron studies
  - *Vitamin deficiency*: send folate, B12
  - *Renal disease*: check BMP
  - *Hypothyroidism*: send TSH
  - *Marrow disorder*: SPEP, SFLC (serum free light chains), discuss bone marrow bx

- **Macrocytic (MCV > 100)**
  - *Vitamin deficiency*: send folate, B12
  - *Cirrhosis*: get EtOH history, LFTs consider HCV/HBV serologies
  - *Hypothyroidism*: send TSH
  - *Marrow disorder*: SPEP, SFLC
  - *Consider medication effect*: HIV meds, chemotherapeutics

**Normal reticulocyte index (>2)**

*Assess for hemolysis*: LDH (high), bilirubin (high), haptoglobin (low), UA (+urobili; - bil); if hemolysis labs +, send DAT/Direct Coombs

*Obtain special slide* (blood smear); someone will need to examine at Gray 5 Heme lab

*Consider acute blood loss*: GI bleed, hematoma

What do I do about it?

- Transfuse pRBC to a goal Hgb of 7 (8 for patients with severe coronary artery disease).
  - Patients who are listed for solid organ transplant should receive leukoreduced blood products.
  - Patients with a history of recent treatment with chemotherapy or hematologic malignancy should receive leukoreduced + irradiated blood products.

- Treat the underlying cause:
  - *Iron deficiency anemia*: consider ñ loss (menses, chronic GIB e.g. cancer, hemolysis) or ñ intake/absorption (IBD, celiac, PPI use). If severe anemia or CKD/IBD/CHF/PO intolerance: iron sucrose 300 mg QOD x 3 doses. If mild: PO repletion with 975 mg iron sulfate QOD x 3-6 months.
  - Anemia of chronic disease: treat underlying disease (CKD, CTD, infection)
  - Folate deficiency: 1-5 mg PO folate QD
  - B12 deficiency: 1-2 mg PO vitamin B12 QD
  - Hemolytic anemia, concern for marrow disorder: Consult hematology

- If no acute precipitant (like hemolysis) and H/H are stable, consider deferring work-up to outpatient

See [Whitebook p125-126](#) for more details on anemia
What is syncope?
• Transient loss of consciousness and postural tone due to cerebral hypoperfusion with spontaneous, complete recovery of consciousness without intervention.

What are the most common causes of syncope?
• Nervous system (60%): vasovagal, carotid sinus syncope, situational
• Orthostasis (15%): autonomic failure, drug-induced, volume depletion
• Cardiogenic (15%): arrhythmia, valvular disease (i.e. Aortic Stenosis), PE, tamponade, dissection
• Neurologic (<10%): seizure, stroke, TIA, subclavian steal
• Notably, the cause of syncope is not found in 30% of cases.

What history is helpful for distinguishing the different types of syncope?
• Nervous system
  o Vasovagal: prodrome of dizziness, nausea, warmth, diaphoresis in the setting of stress, pain, urination, defecation
• Orthostasis
  o Prodrome of dizziness, nausea, warmth, diaphoresis in the setting of known volume depletion, medication effect, or autonomic failure (i.e. Parkinson’s Disease, Lewy Body dementia)
• Cardiogenic
  o Arrhythmia: no prodrome, prior intermittent palpitations associated with dizziness or lightheadedness. Syncope while sitting or supine. Falling forwards causing facial injuries, “drop attack”
  o Valvular: progressive dyspnea on exertion or angina on exertion.
  o Pulmonary embolism: pleuritic chest pain, history of DVT/PE
• Neurologic
  o Seizure: urinary or fecal incontinence, seizure like movements, prior seizure history
  o Stroke: focal neurologic deficits, slurred speech, facial droops

What work up should I perform for syncope?
Put everyone on telemetry unless confident in orthostasis (i.e.-patient faints in the hospital while using the bathroom)
• Nervous system - Vasovagal: history is often sufficient for diagnosis
• Orthostasis - Check orthostatic vital signs, evaluate for new anti-hypertensives, and physical exam for volume depletion and signs of undiagnosed neurologic conditions such as cogwheel rigidity
• Cardiogenic
  o Arrhythmia: EKG, continuous telemetry, consider TTE
  o Valvular: EKG, TTE
  o Pulmonary embolism: troponin, BNP, D-Dimer, Well’s score, and consider CT-PE (can obtain V/Q scan if renal function is poor)
• Neurologic
  o Seizure: EEG
  o Stroke: CT, MRI brain, MRA head/neck

What should I think about on discharge?
• Consider placing a ziopatch prior to discharge to monitor for arrhythmias
• Establish close outpatient follow up, especially if blood pressure medications were changed

See the Whitebook p40 for more details
What is delirium?
It is an acute (hours to days) change from the patient’s neurocognitive baseline leading to deficits in attention and awareness that tend to fluctuate over the course of the day.

Who is at greatest risk for delirium?
Patients with prior delirium, underlying neurological disease (e.g. dementia, stroke, Parkinson’s) hearing or visual impairment, age >65, multiple comorbidities, alcohol use are at highest risk.

How can I prevent my patient from getting delirium?
• Reorient: Encourage use of glasses and hearing aids, orient patient to date/location/reason for hospitalization, encourage communication with family/friends, maintain same care team when possible
• Minimize tethers and alarms: DC unnecessary telemetry, pulse ox, IV medications, lines, catheters
• Avoid/limit delirium causing medications (discuss with pharmacy); common culprits include opiates, benzodiazepines, steroids, antihistamines, antibiotics
• Support normal sleep wake cycle:
  ◦ Daytime: Lights on, raise shades, OOB to chair, work with PT/OT
  ◦ Nighttime: Lights/TV off, minimize unnecessary vitals, labs, diuresis, melatonin 5 mg q6PM

How do I know my patient has delirium?
• You have to know their baseline mental status (resources: prior notes, family, friends, nursing staff, PCP)
  ◦ Once you know the baseline mental status, then you can know how it has changed
    § NOTE: Delirium can be HYPOACTIVE OR HYPERACTIVE
  ◦ Clearly document neurocognitive baseline and prior focal neurological deficits for future provides
• Make sure to think about other causes of acute altered mental status (see next page)

What could have caused my patient to have delirium?
• Medication/Toxins – review MAR & home meds, ask pharmacy, consider ETOH/Drug withdrawal
• Infection – review CBC, FEVER tab in Epic, consider sending basic infectious workup
• Metabolic – check pulse ox, review BMP, LFTs, glucose, consider VBG for hypercarbia
• Undertreated Pain – consider recent procedures, review PAIN tab

What do I do about delirium?
• FIRST address precipitating factors AND institute prevention strategies listed above
• For hyperactive delirium (dangerous behavior only):
  ◦ Escalating interventions: reorientation > 1:1 sitter > medications > restraints
  ◦ Important medication considerations:
    ▪ Use short term PRNs only, continuous or prophylactic dosing should be avoided
    ▪ Onset of action may be 5 -20 minutes with IV or longer with IM/PO
    ▪ Consider dosing medications in the early evening to permit sleep, but avoid daytime sedation
    ▪ Avoid quickly stacked doses of antipsychotics as this can lead to profound sedation
    ▪ Check daily EKG, change treatment if QTc > 500, increases by 25% new T wave flattening or U wave
  ◦ Initial medication options:
    Haloperidol 2.5-5 mg IV q3h PRN
    Olanzapine 2.5-10 mg SL/PO/IM q4-12h PRN
    For continued severe agitation can consider scheduling nightly or standing doses OR psychiatry consult

See Whitebook p184 for more details on delirium
How do I make sure nothing serious is going on in the first couple of minutes?

- **ABCs & Vitals:**
  - If unresponsive/pulseless, **CALL CODE BLUE.** If hypoxemic & GCS < 8, **CALL RICU & RAPID RESPONSE**
  - Vitals can give you a clue to etiology; correct vital sign abnormalities as able

- **Bedside Exam:**
  - Establish arousal (AVPU or GCS), ability to follow commands, attention (days of week forward & backwards); examine for focal weakness
  - If not following commands, check for pupillary/corneal reflexes, withdrawal to pain in all extremities and posturing
  - Examine C-spine if trauma; check for asterixis/myoclonus (metabolic cause), tongue biting +/- incontinence (seizure), neck rigidity (meningitis)

- **STAT Orders:** ALWAYS check fingerstick glucose & EKG
  - If patient fell, order non-con head CT
  - If patient has focal deficits or findings c/f stroke, **CALL CODE STROKE** & order STAT non-con head CT

What do I do next?

- **Review current & home meds for:**
  - Diabetes medications
  - Sedatives e.g, benzos & opiates
  - Antibiotics – cefepime, FQs (-floxacins)
  - Anti-epileptic drugs
  - TCAs (-triptylines)
  - Anti-histamines (e.g. Benadryl, famotidine)
  - Anti-dopaminergics – antipsychotics and some anti-emetics (Reglan, Compazine)
  - Other considerations: LFTs/NH3, B12, TSH +/- T4, cultures, serum/urine toxicology, drug levels (e.g. AEDs), and UA. Consider substance use/withdrawal and other drug toxicities (digoxin, lithium, valproate)

What else should I think about?

- Consider a neuro consult
- Consider EEG w/ LTM (for intermittent seizure, non-convulsive status) and MRI (+ contrast if CrCl OK)
- Consider LP if concern for CNS infection or malignancy; image first to r/o herniation
  - See this [page](#) – section on Secondary/Subacute MS Workup – about what LP studies to consider
  - Ask lab to save CSF for further studies if needed

But what is actually causing this person to act so weird?

1) Metabolic 2) Infectious 3) Drugs/toxins/medication 4) Primary CNS 5) Delirium

**Acute** (minutes-hours): trauma, vascular, ↑ ICP, meds/toxins, metabolic

**Subacute** (hours-days): infectious, AI, neoplastic, metabolic

**Chronic** (days-months+): neurodegenerative, psych

How do I handle this situation?

Treat the underlying cause

For acute agitation, consider antipsychotics – Haldol (IV/IM/PO, check QTc, if dystonic rxn give Benadryl 25-50 IM/IV), olanzapine (SL/PO/IM: QTc less affected), quetiapine (PO, check QTc)

See [Whitebook p183](#) for more details on AMS
Where do I start?

• **All patients**: attempt non-pharmacologic measures (e.g., PT/exercise/activity, heat or ice, treating comorbid psych dx, massage, acupuncture, etc.) and consider adjuvant meds (see below)

• **Mild-moderate pain**: use non-opioids in addition to above
  - Acetaminophen (Tylenol) – max dose 3g daily (2g is safe in liver disease)
  - NSAIDs – cautious use in pts with high bleed risk, cardiovascular or renal disease; give w/ meals very useful in inflammatory (think swelling, erythema, warmth) pain or bony pain
    - Oral: Ibuprofen, naproxen, or celecoxib (decreased risk of GI bleeding with this)
    - IV: Ketorolac (Toradol) 15mg dose equivalent to 30mg – can use q6h x 3 days

• **Moderate-severe pain**: consider short acting opioids in addition to above

• **Severe pain requiring around the clock short-acting opioids** – consider adding extended-release opioids
  - If pain source expected to resolve (fracture, hematoma), avoid extended-release opioids

Are there other medications I should be thinking about?

• **Steroids** – can be useful in bony pain, inflammatory pain, or pain associated with visceral distention (eg. hepatic capsular stretch from metastases); caution in cancer pts (may interfere w/ treatment)

• **Topical lidocaine/Lidocaine patch** – can be useful in neuropathic pain as well as muscle spasm

• **Muscle relaxants (benzos, tizanidine, cyclobenzaprine, baclofen)**: useful in spasm; watch for sedation

• **Gabapentin, pregabalin, SNRIs (duloxetine), TCAs (amitriptyline, nortriptyline)**: use for neuropathic pain

How do I use opiates appropriately?

• In patients on suboxone/methadone for opiate use disorder, consult ACT for assistance

• AVOID morphine and hydrocodone in patients with renal disease; fentanyl and methadone are safest in patients with renal disease but hydromorphone (Dilaudid) or oxycodone are acceptable alternatives

• Beware the following adverse effects:
  - Constipation: ALWAYS start standing senna & miralax when initiating opioids
  - Respiratory depression – hold opioid, consider low doses (0.04 to 0.08mg) of naloxone q1-2min
  - Nausea/vomiting – avoid Zofran (worsens constipation); can use Reglan, Compazine, Haldol
  - Other: pruritis, myoclonus, delirium, true allergic reaction (rare)
  - NOTE: hypotension from low-dose opiates is unlikely. If someone is in pain but has lower systolic BPs (90s), okay to still give narcotics with monitoring

What do I do if I want to switch between opiates?

**Opioid Equianalgesic Doses**

<table>
<thead>
<tr>
<th>Drug</th>
<th>PO (mg)</th>
<th>IV (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>20</td>
<td>n/a</td>
</tr>
<tr>
<td>HYDROcodone</td>
<td>20</td>
<td>n/a</td>
</tr>
<tr>
<td>HYDROMorphine</td>
<td>7.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Fentanyl*</td>
<td>n/a</td>
<td>0.1 (100 mcg)</td>
</tr>
</tbody>
</table>

**Fentanyl patch (mcg/hr)**

<table>
<thead>
<tr>
<th>Morphine PO (mg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
</tr>
<tr>
<td>50</td>
</tr>
<tr>
<td>75</td>
</tr>
</tbody>
</table>

*Use caution converting to Fentanyl - short duration of action

Click for Online Equianalgesic Calculator

NOTE: Switching requires a 25-50% reduction to account for incomplete cross-tolerance

The online calculator also uses slightly discrepant equianalgesic dosing from the chart to the left

See Whitebook p153-154 for more details on pain control
GENERAL MEDICINE

Inpatient Diabetes Management

What types of insulin are there?

<table>
<thead>
<tr>
<th>Type (Onset)</th>
<th>Name (bold = on MGH formulary)</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid (10 min)</td>
<td>lispro (Humalog), aspart (Novolog), glulisine (Apidra)</td>
<td>0.5-2.5 hr</td>
<td>&lt; 5 hr</td>
</tr>
<tr>
<td>Short (30 min)</td>
<td>regular (Humulin R, Novolin R)</td>
<td>2.5-5 hr</td>
<td>4-12 hr</td>
</tr>
<tr>
<td>Intermediate (1-2 hr)</td>
<td>NPH (Humulin N, Novolin N) BID</td>
<td>4-12 hr</td>
<td>12-18 hr</td>
</tr>
<tr>
<td>Long (3-4 hr)</td>
<td>glargine (Lantus)QD, detemir(Levemir)BID/QD, degludec (Tresiba)QD</td>
<td>none</td>
<td>24 hr</td>
</tr>
</tbody>
</table>

**Basal insulin:** fixed intermediate/long-acting; for basic metabolic requirements

**Prandial/Nutritional insulin:** fixed rapid/short-acting; to cover meals

**Correctional insulin:** sliding scale rapid/short-acting; to correct hyperglycemia (not intended to cover meals)

What should I do when I admit this patient with diabetes mellitus (DM)?

1. Hold home non-insulin DM meds (**NEVER hold basal insulin for T1DM**); write for consistent carb diet.
2. Open the “Adult Basal/Bolus Insulin” Order Set in Epic.
   a. FSBG AC & QHS will be auto-selected; change to q6h for patients who are NPO or on tube feeds
   b. If on home insulin, continue home insulin regimen with dose reduction (~25-50% reduction)
   c. If not on home insulin AND well controlled (A1c < 7%), start with sliding scale if age>75/frail/ESLD/ESRD; else, moderate-dose; transition to basal-prandial based on needs
   d. If not on home insulin AND not well controlled (A1c > 7%), start with basal (0.2 U/kg) & correctional (moderate dose). Then add a prandial/nutritional insulin in 1-2 days based on needs.
3. Check A1c in all patients with hyperglycemia if not done in last 3 months

What should I do while the patient is in the hospital?

Review blood glucose (BG) trends (use “Gluc” tab in Epic) and adjust insulin dosing daily:

- Fasting or AM BG high (w/ other BGs in range) → ↑ basal insulin dose*
- Fasting BG high + HS BG high (w/ other BGs in range) → ↑ pre-dinner prandial insulin dose
- Pre-lunch or dinner BG high (w/ other BGs in range) → ↑ prandial insulin dose of preceding meal
- BG rising steadily over course of day → ↑ prandial insulin dose at each meal

In general, increase by no more than 20% of total daily insulin requirement every day

What if my patient becomes hypoglycemic on insulin?

If awake and alert, PO juice is best – 4 ounces. If unable to take PO, give IV D50W (auto-selected in “Adult Basal/Bolus Insulin” order set as a PRN). If BG<50 or altered mental status, give 1 amp (25 g). If BG 50-69 with normal mental status (but unable to take PO), give 1/2 amp (12.5 g). ALWAYS recheck FSBG in 15 min. Review BG and insulin trends; adjust insulin dosage as necessary.

What if my patient is NPO?

Put NPO instructions in the order administration instructions on ALL insulin orders. 50% dose reduction or 0.1 U/kg/day for basal insulin. HOLD prandial/mealtime insulin. Change sliding scale & FSBG to q6h.

What about DKA/HHS?

**DKA/HHS is a medical emergency; ASK FOR HELP.** Open “DKA/HHS” order set; see here (DKA) or here (HHS)

What should I do when the patient is getting discharged?

If new to home insulin → place nutrition consult & arrange for floor RN teaching + outpatient f/u. Using discharge order set, send prescriptions for glucometer, test strips, lancets, syringes/vials or pens/needles to MGH outpatient pharmacy and bring to floor for RN teaching

See **Whitebook p171** for more details on Inpatient Diabetes

For patients w/ DM starting steroids, tube feeds or TPN, please see bottom of p171 (Special Situations)
What’s the dangerous stuff that I should look out for?

• **ANAPHYLAXIS**
  - An acute, life-threatening, multi-organ syndrome of systemic vasodilation + bronchospasm
  - How do I know it’s anaphylaxis?
    - Low BP (SBP<90 or >30% drop from baseline)
    - Skin/mucosal swelling: hives, angioedema
    - Respiratory symptoms: stridor, bronchospasm
    - GI symptoms: nausea/vomiting/diarrhea/abdominal pain
  - What do I do?
    - **CALL A RAPID RESPONSE**; in the meantime:
      - ABCs: Secure airway, administer O2 (for SpO2 > 94%) and IVF (if hypoTN)
      - Give EPINEPHRINE: 0.3mg dose works no matter what dilution
      - Symptoms can recur within 8-72 hrs; to prevent this, give methylprednisolone 125mg IV QD x 2d
      - Adjunctive agents: Diphenhydramine 50mg IV/IM (H1 blocker), albuterol (nebs q15 mins x3 doses)
  - What caused it? Could be a lot of things; common ones include antibiotics, foods, latex

• **SJS/TEN (Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis)**
  - What is it?
    - Severe drug reaction that leads to blisters/bullae & desquamation/peeling of skin & mucosa
    - Usually happens 1-3 weeks after exposure to the trigger (Bactrim is the most common trigger)
    - See [here](#) (under Skin section) for other triggers and what to do.

• **DRESS (Drug Reaction with Eosinophilia & Systemic Symptoms)**
  - What is it?
    - A drug-specific immune response causing multisystem organ damage
    - Often presents w/ fever, facial edema, rash, lymphadenopathy, 2-8 weeks after drug exposure
    - Labs typically show eosinophilia (>700/microL), among other findings. See [here](#) (bottom of page) for other findings and see [here](#) to calculate a RegiSCAR score (to determine likelihood of DRESS)

My patient has a penicillin/cephalosporin allergy; can I still use a penicillin/cephalosporin?
Please see [MGH PCN and Cephalosporin Hypersensitivity Pathway](#) or the app [here](#)

My patient has a CT contrast allergy; what do I do to get them this CT study with IV contrast?
Call & coordinate with radiology. Please see bottom half of [this page](#)

What about Vancomycin Hypersensitivity (Red Man Syndrome)?

• What is it?
  - An idiopathic hypersensitivity reaction associated with rapid IV administration > flushing, erythema, and pruritis of upper torso, neck, and face
  - Difficult to distinguish from early anaphylaxis; if in doubt, treat as anaphylaxis!

• What do I do?
  - See the [MGH Vancomycin Hypersensitivity Pathway](#)

What if my patient just has a rash?
This is the most common drug reaction. Look for a erythematous macular/papular diffuse rash that may or may not be itchy. Cephalosporins and penicillins are common offenders. For rashes about which you are uncertain, consider a dermatology consult for help

See [Whitebook p178-181](#) for more details on Allergies and Drug Reactions
What should I ask a patient with AUD on admission? What should I review in the chart?
- Drinks/day, type of alcohol, time of last drink, hx of complicated withdrawal (seizures/intubation/DTs)
- Review MassPAT, recent tox screens, previous Addiction Consult Team (ACT) notes, other drug use

What labs abnormalities should I watch out for in pts with AUD?
- Abnormal LFTs (alcoholic hepatitis, cirrhosis), low K/Mg/Ca/phos, high anion gap + low HCO₃ (aka anion gap metabolic acidosis- could be 2/2 alcoholic ketoacidosis and/or elevated lactate from poor PO intake/hypovolemia), sometimes pancytopenia (especially thrombocytopenia), elevated INR/low albumin suggest possible cirrhosis

When should I be concerned about alcohol withdrawal? What are the symptoms?
- Time of onset after last drink is variable depending on how much alcohol a person normally drinks. In some patients with severe AUD, withdrawal can start even if blood alcohol level is still elevated. In general, symptoms can start 6 hours after last drink
- Minor withdrawal signs: 6-48h after last drink-> tremors, sweats, ↑ HR, headache, anxiety
- Alcoholic hallucinosis: 24h-6d after last drink-> visual/tactile >auditory hallucinations
- Withdrawal seizures: highest risk of seizures 6-48h after last drink
- Delirium tremens (“DTs”): DANGEROUS! Mortality 1-4%. Delayed onset 48h-5d after last drink-> tremors, sweats, ↑HR, HTN, fever, inattention, hallucinations, agitation, disorientation is key distinguishing feature

How do I treat alcohol withdrawal?
- Make sure K/Mg/Phos/Ca2+ are repleted, Order folate 1mg PO QD and daily multivitamin
- Give IV thiamine 500mg TID x 5 days (first dose BEFORE glucose/D5NS)-> PO 100mg QD x 10 days (if IV thiamin shortage can do PO)
- Consider D5NS (AFTER thiamine) to treat anion gap metabolic acidosis and hydrate
- Consult Addictions Consult Team
- Open “Alcohol Withdrawal Order Set” in Epic. Under “Disease Specific Med” select Ativan (CIWA) OR Phenobarbital
  - Ativan: Use for mild/moderate sx’s, no h/o complicated withdrawal, and/or when phenobarb is contraindicated.
    - Usually choose Standard Dose Regimen (0-4mg q4h PRN). Consider Low Dose (0-2mg q4h PRN) if increased risk of sedation or respiratory depression. Default to PO unless more severe symptoms or unable to take PO, then use IV PRN. Consider making doses standing if hx of severe withdrawal
    - Make sure to also order “Alcohol Withdrawal Assessment” order under “Nursing Orders”
    - If CIWA scores consistently >16 (and total Ativan dose received <30mg), consider switch to phenobarb
  - Phenobarb: Use for moderate/severe signs or in patients with history of severe or complicated withdrawal. If patient had success with phenobarb in past, can try this again. Contraindications include history of frequent AMA discharges, patient already received >30mg ativan since arrival, unstable respiratory status. Use “Phenobarbital” order set in Epic.
    - DO NOT give additional benzos after giving phenobarb!!

What if patient is not doing well (e.g. somnolence, agitation, disorientation, unstable VS) despite treatment?
- Call RAPID RESPONSE or Med Senior for seizures, hypoxia, mental status change, airway concern
- Severe cases of alcohol withdrawal do sometimes require transfer to ICU for intubation, IV gtts

See Whitebook p197 for more details on Alcohol Withdrawal
My patient’s chart says he/she has opiate use disorder (OUD); what should I do on admission?

- Ask the patient if he/she is currently withdrawing? See below for treatment of symptoms.
- Take a good history to assess severity of OUD & withdrawal risk
  - WHAT substances does he/she use?
  - WHEN did he/she last use? HOW often?
  - What complications of use has he/she had (overdoses, infectious complications)?
  - What goals does he/she have? Any interest in treatment? Help with safer use?
- Review MASSPAT, toxicology screens, previous Addiction Consult Service (ACT) & PCP/psych notes
- Order the following labs (** = order if patient has used IV drugs since last check or not checked recently):
  - Serum toxicology
  - Urine toxicology
  - Urine VPAIN
  - LFTs
  - HIV**
  - HBV/HCV serologies**
  - Syphilis Screen**
  - TSPOT**
  - EKG for QTc

**NOTE:** For help with managing withdrawal or overdose, initiating OR adjusting suboxone or methadone, further motivational interviewing, discharge planning & connection to resources/treatment, consult the ADDICTIONS CONSULT TEAM (ACT) by placing an order in Epic; page 29667 if urgent.

My patient is not withdrawing and not sedated; can I restart home methadone or suboxone?

- If on home methadone and QTc < 500, generally safe to restart home dose
- If on home suboxone AND denies recent use of other opioids, generally safe to restart home dose
- If on home suboxone AND has used other opioids recently, wait to restart home dose until Clinical Opioid Withdrawal Scale (COWS) >10 (~12h s/p last heroin/short acting opioid use) to avoid causing withdrawal

My patient appears to be withdrawing from opiates; what do I do?

- If on home methadone and QTc < 500, can restart methadone
- If on home suboxone and COWS > 10, can restart home suboxone
- If on neither home, consult the ACT team manage symptoms
  - Autonomic symptoms (sweating, runny nose, tearing, etc): clonidine 0.1-0.2mg TID PRN (due to possible hypotension, monitor BPs & avoid giving with 1st suboxone/methadone dose)
  - Abdominal cramps: bentyl 10-20mg Q6H PRN
  - Nausea: promethazine 25-50mg IM PRN
  - Diarrhea: loperamide 2mg PRN
  - Anxiety: hydroxyzine 25mg Q8H PRN or trazodone 50-100mg q8h PRN

My patient overdosed; what do I do?

CALL RAPID RESPONSE for altered mental status; ALSO CALL RICU if pt is apneic/hypoxic/not protecting airway
GIVE NALOXONE 0.4mg IV, if no response increase dose q2 min->0.5mg->2mg->4mg; ASK FOR HELP Once patient is better, monitor mental status, start continuous O2 monitoring & get a CXR. Consider asking pharmacy for a naloxone gtt as naloxone is very short acting.

Can I use opiates to treat pain in patients with current/past OUD? What about if on suboxone/methadone?

In brief, YES, you can. Keep in mind patients may require high doses due to increased tolerance Consult the ACT team for help. Consider a Pain Service consult.

I am about to discharge my patient with OUD. What should I keep in mind?

- Work with floor CM/ACT SW to ensure pts have insurance, PCP, +/- suboxone prescriber/methadone clinic
- For pts on methadone, give last dose letter (date/time/dose/location(MGH) of last dose > seal envelope)
- Prescribe Intranasal Narcan (Choose option in Epic to Add Narcan Instructions to Discharge Paperwork)
- Provide harm reduction counseling. See PCOI document “Harm Reduction Conversation Guide”
- Remind patient MGH Bridge Clinic (Founders 8, 617-643-8281) is always available for treatment See Whitebook p198 for more details on OUD and Opioid Withdrawal