Vade Mecum 2024

"There are, in truth, no specialties in medicine,
since to know fully many of the most important diseases
a man must be familiar with their manifestations in many organs."

~ William Osler

Foreword

Dear Colleagues,

Thank you in advance for the dedication and service you are about to provide our community. You represent a source of hope for our profession, and the values you will bring to your new role will have more impact than you may realize.

You are going to learn so much about yourself during this time. Take pride in your strengths and embrace the parts of you that motivate you to try. I encourage you to celebrate your victories, however small they may feel. You might not always feel like you are meeting your expectations, but there will be countless moments where you will be the right person for a patient or peer at the right time.

Be kind to each other. We are stronger and more effective as a group than we could ever be as individuals. Our time and effort are precious and require discretion to use them well.

The details of your experiences matter. Successes and failures are a collective effort, and if we do not stop to recognize the steps, we can lose sight of what we are working towards.

Enjoy your time together! The connections you make during this time will leave a lasting impression on you.

Mirna Attalla (Editor)

SEOHSC Resident/Clerk Dictation System Access

Dictation ID: (Your PCS ID)

1. Dial ext. **2700** @KGH or **5100** @HDH
2. Enter your **Dictation ID**, then **#**
3. Enter **Password** (your ID), then **#**
4. Enter **Work type**: See table on left (ask your attending for RELEVANT WORK TYPE NUMBER)
5. Enter **Patient CR**, then press #
6. Enter **Attending ID**, then press #

Work Type
2: Discharge
4: Consult
5: Clinic
11: Perioperative Clinic

7. You will hear "Beep", then press **2** to start dictation 8. When the dictation is complete, choose: **a. High Priority Transcription:** Press 6. **b. Finalize report, then starting another:** Dictate "End of Report", then press 8. **c. Finalize report and end:** 5.	**Functions** 1: Pause (up to 60min) 2: Resume dictation 3: Short rewind 7: Longer rewind 77: Complete rewind 4: Fast forward; 44 to fast forward to the end

Diet Order Guidelines

House Diets (Do not require therapeutic diet): Regular, Vegetarian, Kosher, Halal	
Therapeutic Diets: Gluten free, lactose free	
Renal Diets	
AKI	35-70g protein, 50-80mmol K^+, 45-87mmol Na^+, 800-1200mg PO_4^{-3}
CKD	40-70g protein, 100mmol Na^+, 800-1000mg PO_4^{-3} daily.
HD	80-85g protein, 50-80mmol K^+, 100mmol Na^+, 800-1000mg PO_4^{-3}
PD	70-90g protein, 80-100mmol K^+, 100mmol Na^+, 800-1000mg PO_4^{-3}
Transplant	75-105g protein, 100mmol Na^+, 800-1200mg PO_4^{-3} daily.
Fluid Diet	
Clear Fluids	Only clear liquids allowed– designed for <48rs in duration.
Full Fluids	For patients requiring a short-term fluid diet
Diabetic Diets	
Diabetic	Consistent level of carbs and 1 snack provided throughout day.
Maternal Diabetic	Diet suitable for pre/post-natal women with gestational diabetes
Sodium Restricted Diet: 80mmol Na^+/day	
Fiber Diet	
Low Fiber	Total insoluble fiber <10g/day.
High Fiber	Total insoluble fiber 25-38g/day.
Texture Modification	
Purred	Mod-Severe chewing and/or swallowing difficulty
Minced	Mild –Mod chewing and/or pharyngeal swallowing difficulty.
Soft	Mild swallowing problems. Adequate dentition required.
Chopped	For self-feeding patients who can only use one hand.
Fluid Consistency	
Nectar, Honey, Pudding	Reduced tongue control, delay swallow, or ability to protect airway.

Allergy

Author: Dr. Sherwin Wong *Updated May 2024*

Penicillin (or Drug X) Allergy

Pearl: DO NOT label the patient with an allergy before confirming, they are difficult to remove. Adverse reaction ≠ allergy. 0.5-1% of reported allergies are true rxns.

Important Questions to Elicit:

- **What was the rxn:** Distinguish adverse rxn from an allergic rxn
- **When did the rxn occur:** if >10yrs ago, 20% will remain allergic
- **Account of the rxn:** Does the patient remember, or informed by other sources?
- **Rxn to mechanism congruence:** Is the drug known to cause IgE mediated rxn
- **Why was the drug prescribed:** Symptoms may be related to medical condition
- **How fast did the rxn occur:** within hours, days, etc…
- **Were there any other drugs taken at the time:** confounder
- **What intervention was given:** intubation, epinephrine, steroid, etc…
- **Any allergy testing performed:** Skin testing correlation with clinical response
- **What other antibiotic the patient has tolerated in the past.**

What antibiotic can be used if there is a risk of penicillin allergy:

a. **Penicillin:**
- **High risk** (anaphylaxis) → use another class, consult Allergy, consider desensitization
- **Moderate risk** (IgE but not anaphylaxis) → graded challenge test
- **Low risk:** administer medication and monitor
- **Resensitization:** ~3% w/ enteral, after 2-3 courses; possibly higher w/ parental

b. **Cephalosporin:** cross rxn low (98% of +ve skin test to penicillin will tolerate)
- **High risk** (Anaphylaxis) → use another class, consult Allergy
- **Moderate risk** (Hx suggestive of IgE rxn) → graded challenge test
- **Low risk** (Hx not suggestive of IgE rxn) → routine administration

c. **Carbapenem:** cross rxn low (99% of +ve skin test to penicillin will tolerate)

Patient Education: explain the difference between an allergy vs an adverse rxn. *Document: (1) Discharge Summary: separate paragraph with an individualized subheading (2) Write in the Orders – Pharmacy adds it in the Adverse Reaction Icon or removes documented "allergic reaction to Drug X".

Anaphylaxis

Diagnostic Criteria: any of the following. *Mucosa: Respiratory, GI, GU, Eyes

I. Acute onset upon skin, mucosal tissue*, or both manifested as urticaria, pruritus, erythema, etc. **AND** either (a) or (b): (a) Respiratory symptoms (dyspnea, stridor, wheeze); (b) Hypotension

II Acute syndrome onset after exposure to a **likely allergen** manifesting with ≥2 of the following: Skin/mucosa, respiratory manifestation, hypotension, GI symptoms (abdominal cramps, N&V)
III Acute ↓BP: SBP <90mmHg or ↓ >30% from baseline after exposure to **known allergen** for the patient

Investigations: ONLY an allergen challenge test is considered as the gold standard.

- **Mast cell tryptase level:** Collect within 1st 3hrs of episode → repeat in 24hrs after all features resolved. Special way to collect sample (call Core Lab for instructions).
- **Skin test:** Refractory period of ~4wks post severe anaphylaxis: false –ve.

Management for Anaphylaxis:

1. **ABC** (call anesthesia/ICU if airway is threatened), O_2, monitor, IV access
2. Primary Medication:

a. **Epinephrine IM:** 0.5mL of 1:1000 (1mg/mL) solution IM q5-15min PRN
b. **Epinephrine IV push if in shock:** 1mL of 1:10 000 (0.1mg/mL) IV q5-15min PRN with cardiac monitor 67

3. Remove the allergen – food in the mouth, latex on skin, medication, infusion, etc
4. Adjunct Medications:

a. Cetirizine 10mg PO QD (preferable); or Diphenhydramine (Benadryl) 50mg PO/IM/IV q6hr
b. Ranitidine 50mg IV (preferable); or Ranitidine 150mg PO q8h
c. Methylprednisolone 125mg IV q6h → prednisone 50mg PO OD once stable (↓ incidence & severity of delay biphasic rxn)
d. Salbutamol 5mg neb q20min PRN dyspnea
e. Consider Glucagon 1-5mg IV over 5 mins for patients on B-blocker or ACE-i

Disposition:

- Admit for 24hrs for observation post-anaphylaxis (**Biphasic anaphylaxis**: 2nd phase may occur within 48hrs and more severe in 1/3 cases)
- Who to D/C: Stable, Adequate supervision, Able to rapidly access emergency Tx
- Referral to the Allergy/Immunology

Patient Education: DO NOT FORGET

- What is an allergic rxn and specify the culprit. ALLERGEN AVOIDANCE.
- Medic Alert bracelet/necklace
- Prescribe an EpiPen. Teach the patient AND family (have them practice with a demo). Print a pamphlet with diagrams. Inform the patient to note the expiratory date on the EpiPen and replace accordingly.

Contrast Reactions and Pre-Medication

Clinical Pearls:

- Contrast reactions have an event rate of 0.15-0.7% with >98% being self-limiting
- Gadolinium-based reactions occur in 0.02-0.09% with >96% being self-limiting
- Iodine is non-allergenic, shellfish allergies are NOT associated with increased risk of contrast reaction

History:

Allergic-Like Reaction Features	Physiologic-Like Reaction Features
• Urticaria or Pruritus	• Transient warmth / chills • Nausea / vomiting

• Facial edema, sneezing, conjunctivitis, rhinorrhea • Hoarseness or stridor • Wheezing + coughing	• Hypertension • Chest pain, arrhythmia • Pulmonary edema • Seizure

Management of Allergic-Like Reactions: (see Anaphylaxis section)

Management of Physiologic-Like Reactions:

- **Hypotension & Bradycardia** - bolus with 1-2L of IV fluids, consider Atropine 0.6-1mg IV q3-5min (max dose 3mg)
- **Hypertension –** continue IV hydration, treat as hypertensive emergency if needed
- **Cardiac symptoms** – monitor for chest pain, arrhythmia & pulmonary edema

Prevention of Contrast Induced Nephropathy:

Could give IV crystalloid administration for those with GFR <60mL/min
  - Example RL at 1mg/kg/hr 6-12hrs pre and 6-12 hrs post-procedure
- hold nephrotoxics on day of procedure and restart if Cr stable after 48 hours
- Risk is higher with intra arterial contrast compared to intravenous.

Pre-medication for Documented Contrast Allergy:

- **Elective Pre-medication**
  - Prednisone 50mg po at 13hr, 7hr and 1hr pre-contrast AND Diphenhydramine 50mg IV/IM/PO 1hr pre-contrast
  - OR Methyprednisolone 32mg po / hydrocortisone 200mg IV at 12hr, 2hr, pre-contrast can add diphenhydramine as above
- **Emergency Pre-medication**
  - Methylprednisolone 40mg OR hydrocortisone 200mg IV q4h until contrast AND diphenhydramine 50mg IV 1hr pre-contrast
  - Dexamethasone 7.5mg OR betamethasone 6.0mg IV q4h AND diphenhydramine 50mg IV 1hr pre-contrast

Drug Challenge

Clinical Pearls: Only perform in patients who are **unlikely** to be allergic in a monitored environment. Drug challenges do not rule out future adverse reactions.

I. Immediate Reactions (IgE reactions)

a. Setting: Monitored setting with ability to treat anaphylaxis with IV access
b. Patient optimization: remove beta-blockers for 24 hours and optimize respiratory status
c. Placebo doses: recommended to exclude false positive reactions
d. Starting dose: 1/10,000 or 1/1000 of therapeutic dose – preferably oral when applicable
e. Escalation: Tenfold increases q30-60min until full dose

Authors: Dr. Laura Scott, Dr. Jeffrey Lam, Dr. Julia Milden, Dr. Joshua Durbin, Dr. Pardis Balari

Updated May 2024

Approach to Chest Pain

Approach: In order of precedence, but can occur simultaneously in reality.

1. **1º Survey** – ABCD: ↓↑BP? ↓/↑HR? ↓LOC?
2. **1º Resus:** ACLS for unstable arrhythmia? Tx for ↓BP/↑BP
3. **1º Adjuncts:** IV, O2, Cardiac Monitor +/- crash cart with pads. STAT tests: ECG, CXR, Trop/CK
4. **Survey:** Rapidly assess for relevant clinical features to direct management
5. **Reassess patient:** look for evolution with serial ECG & Trop

Etiologies: (Focusing on the common causes that tend occur overnight)

Cardiac – Serial ECGs, don't remove lead stickers to compare to previous

- Stable angina: (a) retrosternal chest discomfort, (b) exacerbation with exertion, eating, emotional stress, and (c) relieved with rest and/or NTG.
- 3/3 = typical; 2/3 = atypical; 1/3 = non-cardiac
- Unstable angina: (a) new rest angina > 20 min, (b) new angina of ≥ CCS class III severity, or (c) crescendo pattern (more frequent, severe, lasts longer with less exertion) of increasing angina increased by ≥ CCS class I

 Hx: quality, onset, duration, exacerbating/relieving factors (e.g. pleuritic/positional), radiation, associated symptoms (SOB, diaphoresis, N&V, lightheadedness, palpitations)

 Usually crescendo onset (not max intensity at onset), pressure-like and diffuse

 Radiation to both arms (+LR 2.6), neck/jaw (+LR 1.5), one arm (+LR 1.3)

 Is the pain identical to prior ischemic angina i.e. previous MI? (+LR 2.2)

 NTG relief is non-discriminatory for acute chest pain (+LR 1.1)

- **Pericarditis**: 2 of (1) typical sharp, pleuritic and positional chest pain, (2) pericardial friction rub, (3) ECG changes (diffuse concave up ST elevation with PR depression), and (4) new or worsening pericardial effusion

Pulmonary – Rule out pneumothorax and hemothorax

- **PE:** pleuritic chest pain occurs in ~40% but could be any pain.
- **Pneumonia, Parapneumonic effusion, Empyema:** not uncommon
- **Lung cancer:** ~20% has pain –dull/ache and persistence quality.

Aorta –sudden tearing pain have low predictive values. VERY painful.

- **Aortic dissection:** high misdiagnosis rates, rare to occur *de novo* whilst in hospital. Features: sudden onset, tearing pain radiating to back, pulse deficit/BP differential (SBP difference ≥20mmHg), CXR with widen mediastinum (>2 have high PLR), & focal neurological deficit. Pain can extend to abdomen/limbs as it progresses.
- New diastolic murmur (AI), hemothorax, hemopericardium/tamponade, ECG suggestive of inferior infarct (STEMI) if type A ascending dissection.

GI – Pain exacerbated/relieved w/ PO intake is the most indicative.
Esophageal origin (-itis, spasm, reflux): response to "Pink Lady" (viscous lidocaine + antacid) does not reliably distinguish cardiac vs esophageal spasm
Cholecystitis: *de novo* incidence is uncommon in inpatients, usually of the acalculous subtype. Suspect in patients more unwell or 3rd gen cephalosporin.

MSK – Usually focal, must be verified with the patient whether identical to chief complaint pain. Pain on palpation can occur with the above etiologies. Can mimic "pleuritic" pain. Check for skin lesions (e.g. shingles rash)

Acute Coronary Syndrome (ACS)

Definitions:

	Unstable Angina	NSTE-ACS	STE-ACS
ECG	ST ↓, or non-specific ST-TΔ	ST ↓, TWI or non-specific ST-TΔ	ST ↑ in two contiguous leads
Trop	Negative	Positive	Positive
Mx	Similar in risk stratification and revascularization		Immediate revascularization

Approach to Ischemic ECG: Systematically analyze; compare to previous

1. ST↑: Does the ST ↑ fit a vascular territory? Are there expected reciprocal changes? ST↑ in AVR is not a STEMI (may represent LM/pLAD/or diffuse disease).
2. ST↓: Ensure these are not reciprocal changes: (PAIL: "Posterior ST↑ → Anterior ST↓; Anterior ST↑ → Interior ST ↓; Interior ST↑ → Lateral ST↓). ST↓ is not localizing unless it represents reciprocal change.
3. Look for other cues: hyperacute T wave, T wave flattening/inversions, pathologic Q waves or poor R-wave progression for previous infarcts.
4. Dysrrhythmia: Dangerous rhythm present? VT/VF, heart blocks (Mobitz II or 3°) that may require temporary pacer wire. Always look for AV block in inf. STEMI.
5. **Serial ECG:** One ECG is a snapshot in time only ∴ taken serially can yield dynamic change/evolution over time.

ST Elevation Territory

I - Lateral	**aVR**	**V1** - Septal	**V4** - Anterior
II - Inferior	**aVL** - Lateral	**V2** - Septal	**V5** - Lateral
III - Inferior	**aVF** - Inferior	**V3** - Anterior	**V6** - Lateral

Special cases to consider:

- Suspect RV infarct with inferior STEMI if: (a) ST ↑ in lead III > II, (b) ST ↑ in V1 → GET right-sided lead (V4R)
- Suspect posterior infarct often with inferior STEMI if: (a) horizontal ST ↓ in V1-3, (b) upright T waves, (c) R > S in V1-2 → GET posterior leads (15 lead ECG) though absence of ST ↑ does not rule out posterior infarct.
- ST ↑ in aVR with diffuse ST ↓: consider (a) left main disease, (b) multi-vessel disease, or (c) global ischemia secondary to secondary process (e.g. sepsis)
- Wellens syndrome: deeply inverted or biphasic T waves in V2-3 (indicates critical LAD stenosis)
- De Winter's T wave: upsloping ST ↓ with tall T waves in precordial leads

Differential for ST Elevation

STEMI	Pericarditis	LVH
LBBB	Paced rhythm	LV aneurysm
Hyper-K/Ca	Brugada syndrome	Early repolarization

Approach to ↑ Troponin: New assay has ↑ Sen & Spec, but doesn't always = ACS, myocardial injury vs. myocardial infarction.

1. Concurrent Hx & ECG(s) suggests ACS?
2. Clinical context of the ↑ Troponin – eg. CHF, Sepsis – hypotension, Tachyarrhythmia
3. Serial Troponin – q6h x 24hrs, or until peaks; UA can evolve into NSTE-ACS

Important Information to Gather:

ϒ Coronary Anatomy: Angiograms, PCI, CABG Hx – the details:

- When & where the procedure was performed – request notes if done elsewhere
- Anatomy and lesions: may elucidate the vessel(s) at risk
- PCI: Angioplasty/BMS/DES
- CABG Grafts: what type (vein, arterial), connections

ϒ ECHO: Cardiac function, wall motion abnormalities, valvular lesion

ϒ Non-Invasive Tests: stress test/echo, nuclear scans, CT coronary angiography

ϒ CAD Risk Factors: hypertension, dyslipidemia, diabetes, smoking, FHx of premature ACS (1st degree males <55 yo, females <65 yo), sedentary lifestyle, inflammatory disease

ϒ Calculate TIMI or GRACE score for risk-stratification and management plan. **TIMI ≥ 2** or **GRACE > 140** in NSTE-ACS are considered higher-risk prompting early invasive strategy rather than ischemia-guided therapy.

Acute Management of NSTE-ACS

1. **Supportive:** ABC, monitor, serial ECG until pain free, O_2, IV access
2. **Medications – Urgent Ones:**
   a. **Antiplatelet:** Aspirin + either Clopidogrel/Ticagrelor

Aspirin 160mg chewable PO x1 → ASA EC 81mg PO OD
Clopidogrel 600mg x1 (if urgent cath) or 300 mg x1 (otherwise), then 75mg PO OD
Ticagrelor 180mg x 1, then 90mg PO BID.

   *Ticagrelor preferred if no contrainidications
   * HOLD P2Y12 inhibitor if awaiting CABG, continue ASA.
   b. **Anticoagulant:** One of the following:

Unfractionated Heparin REDUCED dose EntryPoint Orderset if plan for urgent cath or proceduralization.
LMWH (e.g. enoxaparin 1mg/kg SC BID)
Fondaparinux 2.5 mg SC OD

   c. **Anti-anginal: BB, CCB** (amlodipine), Nitrates → Remember, CCB C/I in low LVEF, BB contraindicated in acute congestion from CHF.

NTG spray 0.4 mg SL q5min x 3 PRN	
NTG patch 0.4/0.8/1.2mg/hr q12h on, q12h off	Mild Angina
NTG infusion 10mcg/min, ↑ by 10mcg/min q5min till pain-free as BP tolerates (Max 200mcg/min) – in CSU!	Persistent angina or CHF

3. **Monitoring:** Depends on the clinical circumstances. If admitted under:
   a. Cardiology: Unstable → CSU; Stable → Davies 3 + telemetry **ongoing pain is an indication for critical care (CSU/D4) admission.
   b. Internal Medicine: Unstable → D4ICU; Stable → Ward+telemetry.
   c. Serial ECGs and serial Troponin
4. **Medications (non-urgent):** high-intensity statin (**atorvastatin 80 mg** or **rosuvastatin 40 mg**), beta-blocker, ACEi/ARB

** Make sure to review the platelet count and hemoglobin, as many medicine patients will experience CP with trop rise in the context of ++anemia, which resolves with correction of underlying anemia.

Acute Management of STE-ACS

STE-ACS in Intra-Hospital Setting: protocol not well-defined – can be chaotic.

1. **Stabilize**
2. **Crash Cart**: Pads on ASAP. May develop sudden dysrhythmia.
3. **Get Help: Consult Cardiology** (Cardio fellow), Senior Resident, RACE.
4. **Medications ASAP**: Aspirin load + Ticagrelor/Plavix load + heparin + nitrates/Anti-anginal
5. **Get Ready to Transport**: DON'T wait for meds – get to PCI ASAP. DAPT should be given prior to PCI. Aim for presentation to balloon time of < 90 min.
6. **Non-urgent medications:** B-blocker, high-dose statin, ACEi/ARB

Chronic Management of ACS

1. Non-pharmacologic: **smoking/EtOH cessation**, weight loss, **cardiac rehab**
2. Antiplatelets: typically **ASA lifelong**, **clopidogrel/ticagrelor for 1 year**
- Recommended for DAPT to be extended beyond 1 year (up to 3 years) in patients who underwent PCI and are not at high risk of bleeding. Ticagrelor is recommended over clopidogrel in patients who underwent PCI, if no C/I.
- If need for interruption for elective surgery, **DES requires at least 3 months** and **BMS requires at least 1 month of DAPT**. ASA always continued if possible.
- If concomitant A fib requiring anticoagulation (CHADS2 ≥ 1 or age ≥ 65): triple therapy (ASA+P2Y12i+OAC) for one month then OAC + Plavix

3. ACE inhibitor/ARB: Titrate up as BP tolerates
4. B-blocker: useful for LVEF < 40%, anti-anginal, rate control (e.g. A fib), rhythm control (e.g. ventricular arrhythmias). Titrate up as HR and BP tolerate.
5. Anti-anginal: B-blocker, **long-acting CCB** (amlodipine), **nitrates**, ***ranolazine, Ivabridine (if in sinus and despite max BB, not at target HR).***
6. Cholesterol-lowering agents: **statin**, **ezetimibe**, **PCSK9 inhibitor**
* Consider reassessment of LV function with repeat ECHO, consider primary prevention ICD if repeat LVEF < 30-35% (see *Heart Failure* section)
7. Refer for Cardiology follow up when appropriate
8. Driving restrictions: For STEMI/NSTEMI with significant LV damage: 1 month restriction for private driving and 3 month restriction for commercial driving. For NSTEMI with PCI and minor LV damage: 48 hour restriction for private driving and 7 day restriction for commercial driving. For NSTEMI without PCI or UA: 7 day restriction for private driving and 30 day restriction for commercial driving. ** SEE CCS drive and fly guidelines. Popular exam question.

Heart Failure

Key Info to Gather:

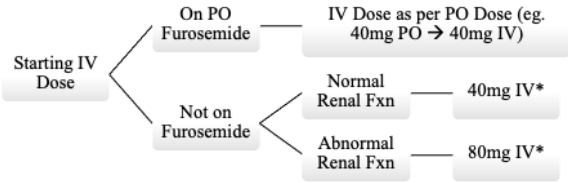
ϒ **NYHA Class:** Past and Present? Class I (ø Symptom), Class II (Slight limitation of ordinary activity), Class III (Marked limitation with min exertion), Class IV (Symptom at rest or with any exertion)
ϒ **Type and Cause?** HFrEF (reduced EF< 40%), HFmEF (mid-range EF 41-49%) or HFpEF (preserved EF >50%). Underlying etiology (e.g. ischemic cardiomyopathy).
ϒ **Previous Investigations:** ECHO, Angiogram, Non-invasive tests
ϒ **Dry weight?** Ask the Patient, or find in the HF Clinic Notes
** MAKE SURE TO ASK RE TRIGGERS (Diet, meds, new LV dysfunction, etc)

Categories	Common Etiology	Diagnosis
CAD	STEMI, NSTEMI, UA	Hx, ECG
Valvular Dx	Stenosis, Regurg	Hx+Exam
Dysrhythmia	Tachy (Rapid AFib), Brady	ECG

Myocardium	Diastolic, Systolic dysfxn	ECHO
↑Preload	↑Na^+ intake, med compliance	Hx
↑Impedance	Hypertensive emergency	BP trends

Acute Mx of "Wet and Warm" State:

1. **ABC**: Sit up, O_2. Assess airway. CPAP if hypoxemic-start at 5cmH_2O, then titrate ↑ as BP tolerates.
2. **Etiology**: elucidate & Tx.
3. **General Acute Mx:**
   - **Furosemide** (Total Volume Depletion):



Note on Furosemide Prescription and Titration:

1. **Diuresis Induction** is dose dependent; double dose if urine output inadequate (NOTE: higher doses is necessitated for low eGFR.)
2. **Total Diuresis Volume** is based on the amount of time spent above diuresis threshold (QD, BID, TID). The decision should be based on reassessment of diuretic response.
3. **Target 0.5-1kg weight loss per 24 hours**

- **Morphine**: Do not routinely use - mostly only for palliation for dyspnea
- **Nitrates**: NTG patch/infusion as BP tolerates
- **Oxygen**
- **Position**: sitting upright
- **Positive pressure ventilation**
- **Salt and fluid restrictions, daily weight, ins and outs.**

Acute Management of "Wet and Cold" State – Cardiogenic shock

- Do NOT strictly need to have low blood pressure – early cardiogenic shock can have a NORMAL BP!
- Clues: cold to touch, signs of end-organ damage (e.g. worsening pulmonary edema, LOC, AKI, ischemic liver, lactic acidosis)
- Call senior for consideration of ionotropic agent +/- vasodilator therapy (often needs concurrent vasopressor therapy)

Chronic Mx – Non-Pharmacological:

1. **Na^+ and fluid restriction** counselling; we have CHF handouts for this
2. **Weight:** DOCUMENT the "dry weight" in the D/C summary and tell the patient that is the weight to aim for.
3. **Warning Signs of Decompensation:** ↑NYHA, Wt > 2kg in 2 days.
4. **Smoking/EtOH cessation**
5. **Flu/pneumococcal vaccination**

Chronic Mx – Pharmacological:

- **HFrEF (EF <40%)**

1. Guideline-Directed Medical Therapy (GDMT) includes ARNI or ACEi/ARB + beta-blocker + MRA + SGLT2i

2. NYHA II-IV on good medical therapy: recommended to switch ACEi/ARB to **ARNI (i.e. Entresto).** A washout period of 36 hours is required when switching from ACEi (not ARB) to ARNI (risk of angioedema). Also recommended to switch to ARNI if admitted to hospital with an exacerbation.
3. If sinus rhythm and HR > 70 on maximal BB therapy: consider adding **ivabradine**
4. Hydralazine/ISDN: recommended in HFrEF who cannot tolerate ARNI/ACEi/ARB or in patients with NYHA IV on optimal medical
5. Digoxin: recommended in HFrEF with poorly controlled atrial fibrillation on maximal BB and in sinus rhythm with ongoing symptoms on optimal medical therapy
6. Vericiguat now considered in HFrEF patients with symptoms and hospitalizations on optimal therapy

- **HFpEF (EF >50%)**

1. Control systolic and diastolic HTN
2. Candesartan can be considered to reduce hospitalizations
3. Lasix for symptom management
4. Reasonable to consider MRA

- Palliative care for intractable dyspnea

**** if chronic HF medications are held on admission for another cause, should be resumed prior to D/C.**

Chronic Mx – Device Therapy:

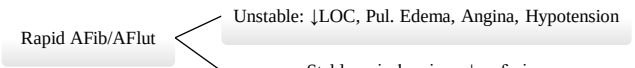
1. ICD for primary prevention:
(1) Ischemic cardiomyopathy (> 40 days post-MI, 30 days post-PCI, 3-6 months post-CABG): **LVEF < 35%** (NYHA II-III) or **LVEF < 30%** (NYHA I)
(2) Non-ischemic cardiomyopathy: **LVEF < 35%** after **3 months of optimal medical therapy**
Note: if changing goals of care to DNR, consider discussing ICD deactivation

2. Cardiac resynchronization therapy (CRT)
(1) **LVEF < 35%** (NYHA II-III or ambulatory IV) PLUS
(2) Sinus rhythm with **QRS > 130 ms + LBBB** (strong) or **QRS > 150 ms + RBBB** (weak) OR
(3) Consider if **chronic RV-pacing** (weak)

Atrial Fibrillation and Atrial Flutter

Diagnosis:
1. Atrial Fibrillation: i. absent p-waves + ii. fibrillating baseline + iii. irregularly irregular QRS
2. Atrial Flutter: i. absent p-waves + ii. Saw-tooth 'f' waves + iii. f-waves at 300, QRS rate follows a block, which may vary (usually 2:1 at 150)
See algorithm in next section under 'tachycardia' for differential diagnosis.

Approach to Rapid AFib and AFlutter:



Acute Management - Unstable: *urgent cardioversion* *to sinus*

1. **Crash cart/monitor:** pads on patient, ACLS algorithm, O_2, adequate IV access

2. **Call for Help:** senior resident and RACE
3. **Pre-medicate:** call senior +/- anesthesia to assist with procedural sedation. Skip if patient at risk for cardiac arrest.
4. **Synchronized cardioversion** at max 200J if unclear

Note: *Often, rapid atrial fibrillation is a response to an underlying physiologic stressor. In that case, the answer is to treat the underlying cause (sepsis, pulmonary embolism, etc). If the patient was previously well, and it's clear that the instability is rising from the rapid rhythm, the treatment is* ***cardioversion*****. Many times these patients will improve with diuresis or fluids in addition to general electrolyte management and pharmacologic rate control.**

Acute Management – Stable

1. Cardioversion (Electrical)

1. Low risk of stroke (clear onset <48h, anticoagulation >3 weeks): cardiovert
2. High risk of stroke (onset >48h or unknown, other high risk features such as recent CVA, mechanical/rheumatic valve, high CHADS2, etc.)
- Option 1: **pre-procedural anticoagulation for 3 weeks** then cardiovert
- Option 2: **TEE-guided cardioversion** (start IV UFH and keep NPO)

3. Post-procedural anticoagulation for 4 weeks

2. Cardioversion (Chemical)

Medication	Notes
Amiodarone 150mg IV x 10min →1mg/min x 6hrs → 0.5mg/min x 18hrs	Chemical cardioversion (same risk of stroke – should know the onset is <48 hours, or pt must be AC)

3. Pharmacological (Rate Control) *(safest option when onset not known)*

1. **Call for Help:** senior resident and RACE
2. **Common IV Meds:**

Medication	Notes
Beta-Blockers: **Metoprolol** 2.5 - 5mg IV q5min x 3. Max 15 mg.	Give over 2 minutes to avoid hypotension. Onset <5min. Last 3-4hrs. Caution in active CHF, bronchospasm. May also put the metoprolol into a mini-bag and run over a longer period of time to avoid hemodynamic swings.
Calcium-Channel Blockers (Non-DHP): **Diltiazem** 10mg IV x 1; repeat 20mg in 15min → 5-15mg/hr infusion	*DO NOT use this in low EF.* Onset <5min. Last 1-3hrs.
Digoxin 0.5mg IV, then 0.25mg IV q6-8h x 3-4 (Need to weight/renal dose) → A load is between 1-1.5mg.	Onset 15-30min, useful if ↓BP. ~↑Parasympathetics.

3. **Start PO formulation** of the effective IV Med ASAP to prevent relapse.

Long Term Management:

1. Rate vs Rhythm Control

Rate Control Agents		Rhythm Control Agents
Metoprolol	25-200mg PO BID	**Amiodarone** 100-200 mg PO QD
Bisoprolol	2.5-10mg PO QD	Consult Cardio/EP for consideration of ablation – particularly if new, uncontrollable, young patient, paroxysmal.
Diltiazem CD	120-360mg PO QD	
Digoxin	0.0625-0.25mg PO QD	

2. Thromboembolic Stroke Prophylaxis: Educate: Risk & benefit discussion is KEY.

CHADS2-65: CHF, HTN, Age ≥ 65-75, DM, Stroke/TIA

HAS-BLED: HTN, Age ≥ 65, Stroke, Bleeding Hx, Liver/Kidney Disease, Elevated INR, Drugs (anti-platelets), EtOH Use

CHADS2	0	1	2	3	4	5	6
Stroke%/yr	1.9	2.8	4.0	5.9	8.5	12.5	18.2
HASBLED	0	1	2	3	4	5	
Bleed%/yr	1.1	1.0	1.9	3.7	8.7	12.5	

ECG: Heart Block

AV Block	ECG Characteristics
1st degree	(1) PR≥0.20s; (2) Each P followed by a QRS; (3) Constant PR
2nd degree	**Mobitz I (Wenckebach Phenomenon):** (1) Progressive ↑ PR interval; (2) Progressive ↓ RR interval; (3) "Group" beating; (4) RR interval containing the non-conducted P wave < than the sum of RR interval prior to the pause
	Mobitz II: (1) PR intervals stay constant; (2) Intermittent non-conducted P waves; (3) RR interval containing the non-conducted P wave is equal to two PP intervals
	2:1 AV block: cannot distinguish between Mobitz I and II * Clues: Mobitz II can worsen with atropine or exercise; improve with vagal maneuvers; often associated with wide QRS
3rd degree (Complete)	(1) Regular PP interval; (2) Regular RR interval; (3) PR intervals variable, no relationship between P & QRS (AV dissociation); (4) Atrial rate > ventricular rate. * If some P conduct into QRS, considered **high-grade AV block** **** in CHB QRS width is very important to determine if the escape rhythm is junctional or ventricular.**

ECG: Bradycardia

I. Rhythm Identification

Narrow complex bradycardia

Regular: sinus bradycardia, junctional bradycardia, complete AV block with junctional escape, atrial flutter with high degree block

Irregular: sinus arrhythmia, sinoatrial exit block, atrial fibrillation with slow ventricular response, atrial flutter with variable block, second degree AV block type I and II

Wide complex bradycardia

Regular: sinoventricular rhythm, complete AV block (ventricular escape), sinoventricular rhythm, regular bradycardia with aberrancy

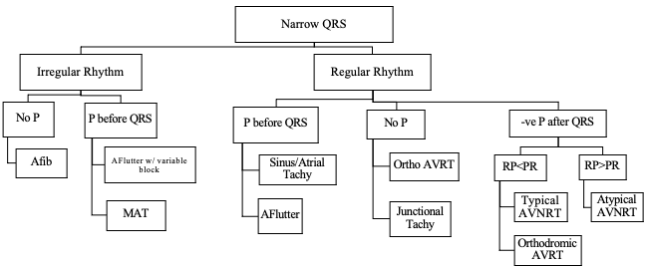
Irregular: sinoatrial exit block with aberrancy, irregular bradycardia with aberrancy, second degree AV block type I and II

II. Acute approach to bradycardia

Stable vs unstable?
- Vitals and ECG. Review medications. Look for reversible causes (i.e. ischemia, hypoxia, electrolyte disturbances, etc.)
- If unstable (hypotensive, aLOC, ischemic CP, shock): consider atropine and/or transcutaneous pacing. May require higher level of care for pressors (i.e epi or dopamine), isoproterenol, and/or temporary pacer.
- Consider a referral to Cardiology for consideration of PPM if second degree type II AV block or higher, or symptomatic bradycardia without a reversible cause.

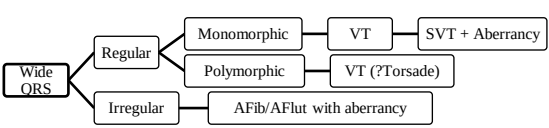
ECG: Tachycardia

I. Rhythm Identification Algorithm – Narrow Complex Tachycardia



Rhythm:	Characteristics:
AFib:	No p-waves. Fibrillating baseline (prominent at V1).
A Flutter:	F-waves at ~300, QRS at 2:1, 3:1, etc.
MAT (Multi-focal atrial tachycardia):	≥3P's with variable morphologies. Variable PR & PP. Commonly seen with lung disease (e.g., COPD).
Atypical AVNRT (10%):	Retrograde P after QRS. Rates >140bpm.
Typical AVNRT (90%):	No P, P in QRS, or P after QRS as Pseudo-r' in V1, Pseudo-S in II, III, aVF. RP<80ms. Rate >140bpm.
Ortho AVRT:	If no P, indistinguishable from AVNRT. Rate >140bpm. RP>80ms.
Junctional Tachy:	No P, or occasional retrograde P. HR <140bpm.

II. Rhythm Identification Algorithm – Wide Complex Tachycardia



Regular WCT: if unstable/unclear, **TREAT AS VT** unless proven otherwise.
Clinical factors that raise the risk of VT: age > 35, CAD, MI, FHx of sudden death

If stable (i.e., you have time to figure it out), features that favour VT over SVT include:

Absence of Block	No signs of LBBB or RBBB
Axis Deviation ('Northwest')	+QRS in aVR, extreme R axis
Broad QRS	>160 ms, R-S interval >100 ms
AV Dissociation	P and QRS at different rates, P waves may be superimposed on to QRS complexes
Capture Beats	P followed by conducted QRS complex of normal duration (narrow)
Fusion Beats	Hybrid complex/morphology
Precordial Leads Concordance	V1-6 QRS either all +ve or -ve

** Refer to Brugada criteria or Vereckei algorhythms.

Syncope

Definition: (a) Complete loss of consciousness & (b) Rapid onset though short duration & (c) Loss of postural tone during episode & (d) Spontaneous full neurological recovery
** Remember that syncope = Cerebral hypoperfusion!

History: Essential Elements to be Elicited for ALL the events

- **Frequency:** How many times has this happened?

- **Prior to the Syncope:**

Circumstance	What were the events & setting prior to the event?
Prodrome	Did the patient feel anything abnormal prior to the event?
Provokers	Positional, exertional, and/or situational?
Relievers	Has patient learned a way to abort these events?

- **During the Syncope:**

Onset acuity	Any recollection of falling or hitting the ground?
Duration	Period of unconsciousness? How do they know?
Witnesses	Witnesses to how the event unfolds or behaviour?

- **After the Syncope:**

Recovery	Time to achieve full neurological recovery?
Associated Features	Anything after the event? Any post-ictal features?

- **Any Injury:** Any injury, or risk for future injury during such events?

- **Non-cardiac features:** features of PE, seizure, sleep-disordered breathing

Physical Examination: Special Maneuvers

- **Orthostatic BP**

- **Respiratory distress** (e.g hyperventilation in PE)

- **Carotid sinus massage** – diagnose carotid sinus hypersensitivity: recommended for >40yo with syncope NYD. 10s on each side in both

supine/standing position with cardiac monitoring. +ve Test: symptoms with asystole >3s, and/or ↓SBP >50mmHg.

High Risk Features: Syncope on exertion, in supine, or a/w new angina, history of heart disease, ECG changes (use OESIL score)

ICD & Pacemaker for the Non-Cardiologist

Pacemaker (Single Chamber, or Dual Chamber):
Function: Coding system (5 letter system – will only discuss the 1st 3 here)

1st Letter	**Chamber paced** (V: ventricle, A: atrial, D: dual, O: none)
2nd Letter	**Chamber sensed** (V: ventricle, A: atrial, D: dual, O: none)
3rd Letter	**Response to a sensed event** (**I: Inhibit** –will not pace in response to a sensed event. **T: Trigger** –will pace in response to a sensed event. **D: Dual (T+I)** – will pace/inhibit based on circumstances in atrium/ventricle. **O: None**)
4th Letter (optional)	**Rate responsive/adaptive pacing** (R: present)

Common Modes and Common Indications:

AOO, VOO, DOO (Asynchronous Mode): ventricle chamber is being paced regardless of sensing
VVI (On Demand Mode): will not depolarize the ventricle if it already is depolarizing.
DDD (Dual Chamber): Will pace atrium if no sensed atrial event, then will pace ventricle if no sensed ventricular event.

CRT (Biventricular Leads):
- **CRT-P:** synchronizes ventricular pacing to improve cardiac output
- **CRT-D:** combination of CRT-P + ICD

Magnet Application – What is the Response:
In general (however, it depends on the propriety model and programming):

ICD CRT-D → Apply Magnet → Defibrillation Deactivation (ie no shocks or ATP pacing)

Pacemaker CRT-P → Apply Magnet → Turns off sensing Asynchronous Pacing (VOO/DOO)

Authors: Dr. Justin Boyle, Dr. Ian White *Updated May 2024*

Non-Invasive Ventilation

Non-invasive positive pressure ventilation (CPAP and BIPAP)

Indications	Contraindications
- Increased work rate of breathing (to avoid fatiguing) - Hypercapnic resp acidosis - Acute cardiogenic pulm edema Conditions likely to benefit: COPD, heart failure, Obesity hypoventilation, neuromuscular weakness. Potentially asthma. Conditions less likely to benefit: hypoxic resp failure, ARDS, pneumonia	**Absolute Contraindications** - Facial trauma or surgery - Inability to protect airway (no gag/cough, low GCS) **Relative contraindications** - Increased respiratory secretions/vomiting - Inability to remove mask (Decreased GCS, physical restraint) - Hemodynamic instability/hypotension, RV failure (NIPPV decreases preload) - Recent gastric/esophageal surgery, bowel obstruction, Pneumothorax **DO NOT USE PHYSICAL RESTRAINTS W/ NIPPV**

HFNC (High Flow Nasal Cannula) Optiflow/Airvo:

Setting: 20-60L flow, 21-100% Fio2.
Effect: Provides substantially more o2 than typical nasal cannula and provides some PEEP (although minimal). ↑ oxygenation, and some support with ventilation.
Indications: Parenchymal lung disease, hypoxic resp failure (ie pneumonia, ILD). Benefits include use over prolonged periods of time, allows patient to eat, well tolerated.
Follow up: Monitor pulse oximetry and clinical status.

CPAP (Continuous Positive Airway Pressure):

Initial setting: min. of 5cmH_2O – Pts with high BMI require higher pressures
Effect: Elevates PEEP = recruit collapsed alveoli to ↑ oxygenation
Indications: Heart Failure (equal to BIPAP), Sleep apnea
Follow up: SpO_2 and clinical status, titrate based off this.

BiPAP (Bilevel Positive Airway Pressure):

Initial setting: usually 10/5 initially (IPAP must be >EPAP by at least 5 to be effective); back up rate of 8 breaths/min
Effect: assists inspiration and provides ↑ positive pressure during expiration. Improves ventilation, decreases resp muscle effort
Indications: COPD w/ hypercapnic resp acidosis, Heart failure, OHS, neuromuscular weakness
Follow up: Within 2hrs of starting NIPPV: Assess VBG/ABG, clinical status (o2, RR, GCS). Increase pressures by 2-3 cmH2O every 5-10 mins for optimal response. If no response, consider ICU consult/call staff if within GOC.

Predictors of NIPPV:

Success
- Improve in <2hrs: stabilizing HR, RR, ↓ $PaCO_2$ >8mmHg, ↑pH >0.06, improving mental status
- Mild-moderate acidemia (pH 7.25-7.35), RR<30, GCS 15, minimal air leak

Failure
- Severely sick patient, presence of pneumonia of chest radiograph

- GCS < 11, RR > 30, Copious resp secretions, poor mask fit

NIPPV Weaning Criteria:

- Hemodynamically stable
- Wean EPAP once: (1) SaO_2 >92%, (2) FiO_2 <0.50
- Wean IPAP once: (1) pH>7.3, (2) RR<22, (3) $PaCO_2$<50 or compensated

Complications:

- Delayed feeding, decline in mobility (predictors of poor outcomes),
- ↓ preload → hypotension
- Gastric distension → ↑ aspiration risk
- Hyperinflation, impaired sleep, dry nose/throat, pooled secretions

Sepsis

Definition: Organ dysfunction due to dysregulated host response to infection

Steps: In practice, the following are concerted simultaneously per resources available

1. **Vitals:** destabilization from sepsis manifests in different ways – isolated to a system (eg. ↓BP), or in combination (eg. ↓BP + respiratory failure).
2. **Stabilization:**

- Airway: If not protecting airway adequately, consult ICU +anesthesia for intubation
- Breathing: supplemental O2+NIPPV/intubation if inadequate oxygenation, and/or ventilation
- **Circulation:**
  a. **What is the endpoint?** A blood pressure that perfuses the end organs. Usually achieved with MAP 65mmHg. correlate end organ manifestations (LOC, chest pain, renal function)
  b. **How much fluid to give?** 500mL to 1L crystalloid IV bolus at a time; **When to start vasopressors?** As a general rule, if the MAP does not ↑ by with 2-3L crystalloid. Can temporize with vasopressors if unstable.

3. **Adequate access:** A CVC maybe required eventually, or immediately if there is inadequate peripheral access.
   DO NOT WASTE TIME trying to put a central line in place for resuscitation. Run vasopressors peripherally or intra-osseous if needed.
4. **Help:** inform senior and call Code 99 – support from RACE team. Sepsis Mx is a complex– the severity and rate of progression are dynamic
5. **Early Antibiotics and Source Control:** administer appropriate antimicrobial(s) within 1hr of recognizing sepsis. Factors to consider: (a) Source; (b) Allergies; (c) Dose adjustment; (d) Co-morbidities; (e) Immune status; (f) Recent exposure to: antibiotics, nosocomial settings, invasive procedures. Broad-spectrum antibiotics if the source is not obvious – do not delay.
6. **Transfer patient to monitored setting** (i.e. D4ICU, ICU)
7. **Ongoing monitoring and evaluation**:

(a) Parameters: LOC, Vital signs, Urine output, SpO_2, lactate, CvO_2
(b) ? Improvement: Y/N
(c) If NO improvement, consider the following:

 Factors affecting tissue oxygenation: respiratory status, hemodynamics

 Infection: adequate antibiotics; definitive source control

 Localizing: require further imaging to define the source of problem

*See Surviving Sepsis Guidelines for full management

Acute Respiratory Distress Syndrome (ARDS)

Timing	• Within one week of known clinical insult or new/worsening respiratory symptoms
Clinical features	• Not explained by cardiac failure or volume overload • Bilateral opacities that are not explained by effusions, lobar/lung collapse or nodules
Oxygenation	• Mild: PaO2/FiO2 = 200-300mmHg with PEEP or CPAP at or greater 5cm H2O • Moderate: PaO2/FiO2 = 100-200mmHg with PEEP 5cm H2O • Severe: PaO2/FiO2 less than 100mmHg with PEEP 5cm H2)

Massive Transfusion Protocol

**Activate if the patient is in hemorrhagic shock (shock index >1.4), mechanism is compatible with need for a massive transfusion (e.g., high speed car accident, gunshot, postpartum hemorrhage), and is expected to require blood component support (plasma, platelets, fibrinogen) in addition to red blood cells

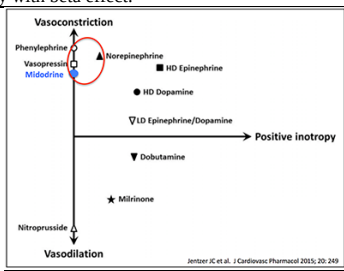
Ensure at least 2 large bore IVs present. Consider using Level-1 rapid infuser

Components of massive transfusion protocol **RBCs:** Transfuse as needed for hemodynamic support **FFP:** Transfuse 1 plasma for every 3 red cells until results of INR is available Platelets: Transfuse if platelet count <50 (<100 with head injury, spinal trauma) **Fibrinogen concentrate:** Remember to check fibrinogen levels. Transfuse fibrinogen concentrate (4 grams per dose) when fibrinogen <1.5 g/L Current evidence is to give products in a 1:1:1 ratio
Complications Monitor for hypocalcemia (pRBCs stored with citrate that binds Ca), hypothermia (can warm blood), hyperkalemia and acid-base derangements
Priority: Stop the bleeding (e.g GI, IR embolization, surgery, tourniquet)

Vasopressors and Inotropes

Vasopressor
Norepinephrine 2-20mcg/min "Ionopressor" Indication: 1st line for sepsis No "Max" dose. Consider adding vasopressin if >20mcg/min needed. Run with central access as can cause tissue ischemia with extravasation.
Epinephrine 0.01-0.05mcg/kg/min "Ionopressor" Indication: sepsis, cardiogenic shock, bradycardia, anaphylaxis Potent but proarrhythmogenic and increase O_2 demand. Consider in ↓CO+SVR Push dose: Take 10ml syringe with 9 ml normal saline and draw up 1 ml cardiac epi (vial is 100mcg/ml). Dose 5-20 mcg as push prn.
Phenylephrine 0.5-9mcg/kg/min "Pure vasopressor" Indication: Distributive shock Take 10mg (Each vial is 10mg/ml) of phenylephrine and inject into a 100mL NS/D5W bag → 100mcg/mL. Can be given as bolus 0.5-2ml (50-200mcg q5-10min PRN), or an infusion (0.5-9mcg/kg/min) for temporization. Tachyphylaxis when used for long periods.
Vasopressin 0.01-0.04units/min "Pure vasopressor" Indication: Distributive shock Very potent. Helpful in RV failure/pulm HTN as it reduces PVR. Usually an adjunct to norepinephrine. High risk tissue ischemia with extravasation.

Inotropes
Dobutamine 2-20mcg/kg/min "Ionodilator" Indication: Cardiogenic shock Direct inotropic effect due to β-agonism with +ve chronotropy, ∴ O_2 consumption. Vasodilatory effect counteracts the inotropic effect and can cause hypotension. Usually used in combination with Norepinephrine to balance this effect. Does have tachyphylaxis.
Milrinone 0.375-0.75mcg/kg/min "Ionodilator" Indication: Cardiogenic shock Short-term inotropic agent (<48hrs) that ↑CO+SVR without ↑HR & O_2 demand. Mainly used as a pulmonary vasodilator (i.e. shock in someone with pulmonary HTN. Onset is ~6 hrs and should not be used as first-line agent (until RV failure confirmed and peripheral vasodilation managed with peripheral vasoconstrictor. Long half life so it can accumulate with renal failure.
Isoproterenol 2-10mcg/min "Ionodilator" Indication: Bradycardia Potent chronotropy with beta effect.



PEARLS:

- ****FLUIDS FIRST** before vasopressors, which can cause organ hypoperfusion if used before adequate fluid resuscitation (except in cardiogenic shock, where fluids are used cautiously)
- **All of the above meds can be given via a peripheral IV.** The concern is with the risk of extravasation causing local ischemia-necrosis. Most risk with norepinephrine and vasopressin.
- **Does the measured BP correlate with other clinical information?** Measure BP from both upper limbs– be wary of titrating against a falsely low BP due to peripheral arterial stenosis. Certain clinical states have a lower resting BP (cirrhosis, ESRD, etc). Ask the patient, or check records for baseline BP.
- **Arterial lines:** can deceive one with falsely low BP when in a small artery.

Rule out obstructive shock (cardiac tamponade, tension pneumothorax, PE)

Dermatology

Authors: Dr. Sherwin Wong, Dr. Kit Birkness *Updated May 2024*

SJS/TEN (Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis)

Clinical features:

- Clinical diagnosis with pathological correlation
- Painful mucocutaneous reaction preceded by fever and dark red spots +/- blistering
- Body surface area (BSA): SJS < 10%; SJS/TEN overlap 10-30%; TEN > 30%
- Nikolsky's sign may be present (blister sloughs when rubbed)
- Common causative agents: allopurinol, NSAIDs, sulfonamide antibiotics and anticonvulsants **1-3 weeks** after exposure.

Investigation: lesion skin biopsy

Management: Remove offending agent, supportive measures. Ophthalmology for ocular involvement. Controversy for IVIG, steroids, cyclosporine.

Varicella Zoster Virus (Shingles)

Clinical Pearls:
- Varicella zoster causes chickenpox (primary infection) and shingles (reactivation)
- Diagnosis is mostly clinical with characteristic rash
- Determine if uncomplicated or disseminated
- Identify if patients meet criteria for oral vs. parenteral antiviral therapy

Clinical Features:
- Reactivation of VZV within sensory ganglia, typically in adults >60yrs
- 70-80% present with prodromal pain 2-3 days prior to drug eruption
- Rash: macular → papules → grouped vesicles → pustulation → ulceration → crusting, typically confined to one dermatome that does not cross midline
- Uncomplicated – one to three adjacent dermatomes
- Cutaneous Dissemination – >20 lesions beyond primary/adjacent dermatomes
- Visceral Dissemination – visceral organ involvement (e.g., pneumonia, encephalitis)
- Complications (associated nerve)
  - Aseptic meningitis (CN V)
  - Bacterial superinfection (any)
  - Bell's Palsy (CN VII)
  - Ocular involvement (CN II, III, V1)
  - Post-herpetic neuralgia (any)
  - Ramsay Hunt Syndrome (triad of ipsilateral facial paralysis, ear pain and vesicles in auditory canal)

Diagnosis: PCR is the most sensitive (>95%) within 1 day

Management:

Indications for treatment*: >50 years old, moderate or severe pain, severe rash, facial/ ocular involvement, other complications of zoster, immunocompromised patients

*Less effective in patients presenting >72h after rash onset
  - Oral therapy: acyclovir 800mg po 5 times per day for 7-10 days OR famicyclovir 500mg TID x 7 days OR valacyclovir 1g po TID x 7 days
  - IV therapy*: acyclovir 10mg/kg IV q8h x 7-10 days (parenteral therapy is required for immunocompromised patients or severe neurologic complications)
- Corticosteroid therapy controversial, but if used must be with antiviral therapy
- Pain: opioids, anticonvulsants (gabapentin/pregabalin), topical lidocaine, TCAs
- Immunocompetent patients require standard precautions.
- Immunocompromised patients require airborne and contact precautions (as in chickenpox)

Endocrinology

Authors: Dr. Samantha Bruzzese, Dr. Robyn Houlden, Dr. Kit Birkness *Updated May 2024*

DKA and HHS

Diagnostic Pattern:

	DKA	HHS
Prominent feature(s)	Ketoacidosis	Volume depletion & hyperosmolality
Glucose (mmol/L)	>14 (usually)	Typically >30
Arterial pH	<7.30	>7.30
Bicarbonate (mmol/L)	<15	>15
Urine ketones	Positive	Negative
Beta-hydroxybutyrate	>3.0	< 3.0
Serum Osm (mOsm/kg)	Variable	>320

Anion gap	>12	<12
Insulin needed	Always	Usually required

Initial investigations:

1. **Blood gas:** ABG or VBG; can't assess mixed acid/base disorders with VBG
2. **Ketones:** urine dip acetoacetate and serum b-hydroxybutyrate (BHOB)
3. **Basic panel**: blood glucose, lytes, extended lytes, Cr, serum osmolality, albumin, lactate, A1C:
   - PseudohypoNa: for every ↑10mmol/L serum glucose >5.6mmol/L, add 3mmol/L to the serum [Na^+]; accounts for ↓ [Na^+] due to hyperglycemia)
   - Calculate anion gap (correct for albumin if low)
4. **Other**: depending on the underlying trigger – CBC, CXR, troponin, ECG, lipase, liver enzymes, blood cultures, urinalysis, TSH, pregnancy test, etc.

Pitfalls to be Beware of:

- Euglycemic DKA: with SGLT inhibitors (-gliflozin), pregnancy, chronic liver disease, prolonged vomiting or diarrhea, sepsis, low calorie diet
- Mixed acid/base: DKA can be precipitated by "stress" factor including toxins or other acid/base altering etiologies
- In HHS, more prolonged duration of relative insulin insufficiency and inadequate fluid intake results in higher blood glucose levels (>34 mmol/L), serum osmolality (>320 mOsm/kg) but minimal acid-base disturbance
- Negative urine ketones do not rule out DKA (better to check serum ketones)

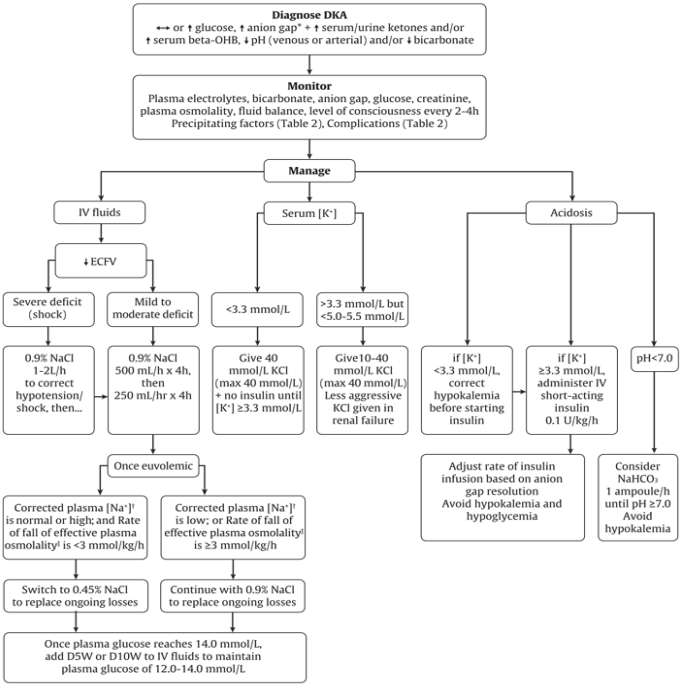
Precipitants (Must Investigate):

- I's: intoxication, infection, insulin omission, initial presentation, infarction/ischemia, intra-abdominal process, intra/peri-operative, iatrogenic (like steroids)
- Consider why insulin was omitted: disordered eating, psychosocial factors, cost, technique, etc.

Mx Principles:

- **Objectives:**
  - Restoration of normal ECFV and tissue perfusion
  - Resolution of ketoacidosis
  - Correction of electrolyte imbalance and hyperglycemia
  - Diagnosis and treatment of coexistent illness
  - Recognize and manage complications (e.g. hyper/hypokalemia, ECFV over-expansion, cerebral edema, hypoglycemia)
- **Tx Order, "DKA": (1) D**ehydration, **(2) K**$^+$ Potassium, **(3) A**nion Gap
- **Complexity:** constant physiological flux during treatment. DKA/HHS protocols can fail due to the dynamics, therefore keep monitoring & adjusting the mx PRN
- **Monitor**:
  - Clinical status: vitals, neuro vitals, volume status
  - 'Lytes and blood glucose q2h until AG < 12 and K+ WNL
  - Capillary glucose q1h until blood glucose < 14mmol/L, then q2h
  - VBG q4h until DKA corrected - Serum Osm - Track urine output
- **[PO_4^{-3}]:** replete if severe losses
- **pH<6.9:** dilute 3amps/150mmol $NaHCO_3$ in 850mL D5W, infuse over 3hrs, r/a
- **Tx End-Goal**: DKA/HHS Resolution Criteria – Anion gap < 12

***** Should know how to write out DKA/HHS orders without order sets



Bridging to Subcutaneous Insulin:

- Consider when: AG <12, patient able to eat a meal + precipitant improved
- Overlap insulin infusion with subcut insulin; can d/c infusion 2h after sc dose or after patient's own insulin pump is reinitiated
- Be vigilant of anion gap widening again

Management pitfalls to beware of:

- Rapid reduction in osmolality can cause cerebral edema – especially in HHS should be lowered no faster than 3mOsm/kg/hr
- Insulin is used to stop ketoacid production in DKA – adjust insulin wrt AG
- Glucose reduction in HHS is due to ECFV re-expansion and osmotic diuresis (therefore, IV insulin isn't necessarily required as no ketoacid production)
- In HHS, individualize IV fluids based on clinical picture, not protocol
- Patients with euglycemic DKA still require insulin to suppress ketosis
-

Inpatient Diabetes Management

Key Points:

- Avoid sliding scales as the sole modality – use scheduled basal bolus insulin therapy with correction dose insulin
- Review capillary blood glucose (CBG) results and antihyperglycemic medication records daily
- Allow knowledgeable patients to participate in self-management

- Consult Diabetes Consult Service for patient diabetes education, complex diabetes management (e.g. parenteral/enteral feeds, pulse steroids, insulin pumps), diabetes discharge planning – involve early in hospitalization
- Glucocorticoid therapy: hyperglycemia is common
  - Institute CBG monitoring for 48hrs min in all patients receiving high-dose glucocorticoid therapy
  - In patients already treated for hyperglycemia, early adjustment of insulin doses is recommended
  - During tapers, adjust insulin doses proactively to avoid hypoglycemia

DMI and DMII Inpatient Management:
- Pt home regimen may not be appropriate during acute illness, AKI, or diet changes
- Reassess non-insulin antihyperglycemics (AHA) and insulin often
- Analyze CBG record daily to look for: (1) hypo-, (2) hyper-, (3) recurrence/pattern

Medication Reconciliation

	Can Eat	NPO
Non-Insulin Agents	Continue (Hold SADMANS in AKI)	Can continue DPP4s and GLP1s, d/c others
Insulin	Continue (Consider 20-30% reduction if low po intake)	D/c short-acting Continue basal (with 20-30% reduction)

CBG Frequency

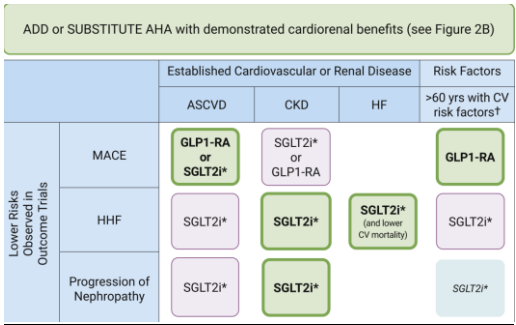
Clinical Scenario	CBG Monitoring
People who are eating	Before meals and bedtime
NPO, Enteral Feeds	q4-6hr
Critically Ill	q1-2hr

CBG Target

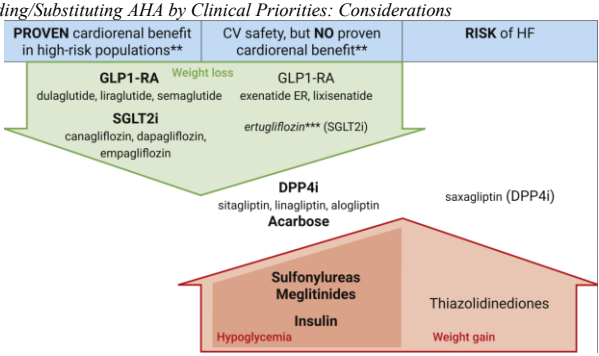
Clinical Scenario	CBG Target
Non-Critically Ill	Pre-prandial 5-8 mmol/L Random <10 mmol/L
Critically Ill	6-10 mmol/L
CABG intraoperatively	5.5-11.1 mmol/L
Noncardiac surgery periop.	5-10 mmol/L

Type 2 Diabetes Agents:
- Assess for micro- and macrovascular complications, review medication side effects and patient's current medication practices, support behavioural interventions
- Regularly review oral agents; if A1C not at target, adjust or advance therapy
- Consider starting basal insulin if symptomatic hyperglycemia or metabolic decompensation

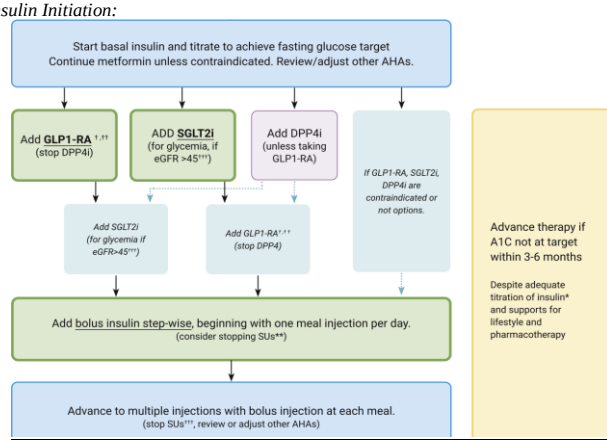
See *Diabetes Canada Clinical Practice Guidelines* for full management support doi: 10.1016/j.jcjd.2020.08.001. PMID: 32972640.
ASCVD, CKD or HF OR Age > 60 with 2 CV RFs (smoking, DLPD, HTN):



Adding/Substituting AHA by Clinical Priorities: Considerations



Insulin Initiation:



Insulin

Insulin Initiation for DMII:

- Calculate total daily requirements based on insulin used in daily sliding scale
- Basal insulin: give total daily requirement as basal dose, or 0.2units/kg long-acting
- Insulin on continuous feeds: complex; consult DM Consult Service

Insulin Pumps: Always Consult the Diabetes Consults/Endocrinology

Functional pump: If the patient can operate, may continue using the machine. Do not use if the patient is not capable. Patient may use their CGM but Nursing staff can only dose insulin based on hospital meter.
Malfunctioned pump: Discontinue the pump & adjuncts, then convert to SC injection regimen; basal dose can be found by accessing pump info, clinic notes, and patient/family; don't worry about bolus dose if not known, use sliding scale

Insulin Adjustment:

Insulin type	Injected	Has Major Effect	Effect Shown
Rapid acting	Before breakfast	Breakfast-lunch	Before lunch
Rapid acting	Before lunch	Lunch-supper	Before supper
Rapid acting	Before supper	Supper-bedtime	At bedtime
Long acting	Morning or qhs	Evenly over 24 hrs	Before breakfast

Insulin Types: (underlined = covered under ODB)
Useful tool at guidelines.diabetes.ca/reduce-complications/insulin-prescription-tool

Insulin Type (Trade Name)	Onset	Peak	Duration
BOLUS (prandial or mealtime) insulins			
Rapid-acting insulin analogues			
Insulin Aspart (NovoRapid®, Trurapi®, Kirsty®)	9–20min	1–1.5h	3–5h
Insulin Glulisine (Apidra®)	10–15min	1–1.5h	3.5–5h
Insulin Lispro (Humalog® U100, U200, Admelog®)	10–15min	1–2h	3–4.75h
Faster-acting insulin aspart (Fiasp®)	4min	0.5-1.5h	3-5h
Short-acting insulins			
Insulin regular (Humulin®-R, Novolin® ge Toronto)	30min	2–3h	6.5h
Insulin regular U-500 (Entuzity® U-500)	15min	4-8h	17-24h
Basal Insulins			
Intermediate-acting (cloudy)			
Insulin Neutral Protamine Hagedorn (Humulin® N, Novolin® ge NPH)	1–3 h	5–8h	Up to 18h
Long-acting insulin (clear) Insulin Detemir (Levemir®) Insulin Glargine U-100 (Lantus®, Basaglar®, Semglee®) Insulin Glargine U-300 (Toujeo®) Insulin Degludec (Tresiba®)	90 min	N/A	Levimir: 16–24h Lantus®: 24h Toujeo®: >30h Tresiba®: 42h

Example of Writing Diabetes Prescription at Discharge:

Glargine prefilled pen 25 units sc q2200 M: 3 boxes Repeat x 10	Glucose meter test strips of choice M: 200 strips, Repeat x 10
Insulin pen needle tips 4 mm M: 1 box Repeat x 10	Libre sensors M: 2 sensors, Repeat x 10
Lancets M:100. Repeat x 10	Baqsimi nasal glucagon 3 mg Use as directed for severe hypoglycemia M: 1 unit, Repeat x 10 L

Hypoglycemia

Clinical Manifestations:

Autonomic: tremor, palpitations, sweating, anxiety, hunger, nausea, tingling
Neuroglycopenic: difficulty concentrating, confusion, weakness, drowsiness, vision changes, difficulty speaking, headache, dizziness
Hypoglycemia unawareness: Lack of autonomic symptoms
*Only ascribe the symptoms to hypoglycemia if simultaneous with blood glucose <4.0mmol/L and symptoms resolve with glucose normalization

Severity:

Mild: Autonomic symptoms present. Individual is able to self-Tx
Moderate: Autonomic + neuroglycopenic symptoms. Individual can self-Tx.
Severe: Individual requires assistance. Unconsciousness/seizure may occur. Blood glucose is typically <2.8 mmol/L.

Acute Mx:

1. **Vitals:** Neurological Status, ABC – capillary glucose
2. **Acute Intervention:**

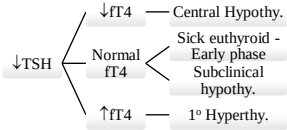
Mild to Moderate	Severe
Patients who can eat: Give 15-16g PO fast acting: • Dex4 liquid 1 bottle (59mL) • Juice 150 mL • 1 package of jam • 4 glucose tablets po	**Conscious patient:** Give 20g PO of fast acting: • Dex4 liquid 1 1/3 bottles (80mL) • Juice 175 mL • 1 package of jam • 5 glucose tablets po
Patients receiving enteral nutrition who can't eat: Give 15-16g of fast acting glucose: • Dex4 liquid 1 bottle (59mL) • Juice 150 mL	**Conscious patient receiving enteral nutrition who can't eat:** Give 20g of fast acting glucose: • Dex4 liquid 1 1/3 bottles (80mL) • Juice 175 mL
Patients who are strictly NPO: • Give 10-25 g (20-50 mL) D50W IV over 1-3 minutes STAT • If no IV access: Give 1mg glucagon IM/SC STAT	**Patient can't safely take PO Tx:** • Notify MRP ASAP • 25 g (50 mL) of D50W IV push over 1-3 minutes STAT • If no IV access: Give 1mg glucagon IM/SC STAT

Note for patients taking acarbose**:** Give glucose tablets (as advised above) or Dex4 liquid (as advised above) **OR** milk 250mL milk (1 cup) **OR** honey 15 mL (1 tblsp)

3. **Recheck blood glucose 15 minutes later, repeat t**x & monitor as needed.
4. **Etiology:** Diabetic vs Non-diabetic
   a. **Diabetic:** Usually related to drug-dose changes/error in administration, renal failure (accumulation of insulin, or other DM meds), skipped meals/NPO
   b. **Non-Diabetic** (uncommon):
   - Low caloric intake: rare – seen in severe starvation
   - Low gluconeogenesis: (a) Advance liver failure; (b) EtOH intoxication; (c) Adrenal insufficiency
   - Glucose mobilization: (a) Drugs (insulin, oral hypoglycemics, salicylate – neuroglycopenia, fluoroquinolone – in elderly); (b) Refeeding syndrome
   - Endogenous hyperinsulinism: insulinoma
   - Nasal glucagon (Baqsimi) is the preferred form of glucagon for use outside the hospital.

Thyroid Panel Interpretation

Key Message: Acute inpatient thyroid function tests are difficult to interpret in isolation without clinical manifestations. Repeat wks after acute illness resolved.



Hyperthyroidism

Chronic Mx: Goal to normalize TSH. TSH may remain low for wks during initial tx

Thioamides	*Initial Dose – severity based*	*Maintenance*
Methimazole (MTZ)	15-60 mg QD	5-15 mg QD
Propylthiouracil (PTU)*	300-900 mg QD	100 mg QD

*PTU only in 1st trimester and thyroid storm, otherwise use MTX

Beta-blocker: attenuating sympathetic-driven discomforts		
Propanolol	10-40mg po q6-8h	Alt: Metoprolol, Atenolol

Thyroid Storm:
- Clinical fts: ↑HR, ↑T, CHF, agitation, psychosis, liver F
- Burch-Wartofsky score: ≥45 highly suggestive, 25-44 supportive, <25 unlikely
- Acute management: consult Endo ASAP, don't wait for TSH/fT4 to treat

General	*Storm-Specific*
1. IV fluids and correct low 'lytes 2. Tx T': cooling, acetaminophen 3. Propanolol 40-80mg PO q6h or 1mg IV q10-15min, OR Esmolol 250-500mcg/kg IVx1, then 50mcg/kg/min infusion until HR controlled 4. Find precipitant & tx	1. PTU* 200 mg PO/NG q6h, when stable convert to usual doses *Preferred over Methimazole because blocks conversion of T4 to T3 2. Hydrocortisone 100 mg IV q8h x 24-48hrs 3. Lugol's solution (Iodine) 10 drops orally q8h – start 1hr after PTU 4. Extra measures: Cholestyramine 4 g orally qid, plasma exchange, thyroidectomy

Hypothyroidism

Chronic Mx: Goal to target N. TSH for 1º Hypothy. & N. fT4 for Central Hypothy.

- Young & healthy: Levothyroxine start at 1.6mcg/kg/day
- Elderly, cardiac hx: Levothyroxine start at 0.4mcg/kg/day (~25mcg PO OD), increase q3-6 weeks until TSH normal

Myxedema: ↓HR, ↓BP, ↓Temp, ↓LOC, ↓ [Na^+], ↓Glucose, ↓[HCO_3^-]. ↑CK.
Acute Mx: **Consult Endo. ASAP.** If features fit, tx. Don't wait for TSH/fT4.

General	**Myxedema**
1. IV fluids +/- pressors 2. Re-warm 3. IV dextrose if needed	1. Hydrocortisone 100mg IV q8h – as tx may unmask co-existing adrenal insufficiency 2. Levothyroxine 200-400 mcg IV, then 50-100 mcg IV daily until able to take PO

4. Find precipitant & tx	**3.** May require T3 tx if severe (cannot convert T4 to T3) but IV T3 not on hospital formulary (If were able, give T3 5-20mcg IV x 1 dose, then 2.5-10mcg IV q8h until clinically improved

Adrenal Insufficiency (AI)

Approach to Diagnosis

1. *Suspicious Features:*
- Early sx: fatigue, weakness, anorexia, weight loss, N/V
- Advanced sx: hypoglycemia, orthostatic hypotension, ↓Na, ↑K, salt-craving
- Physical Exam: pigmentation (skin, mouth), postural hypotension
- Investigations: hypoglycemia, ↓Na, ↑K, ↑Ca

2. *Is patient adrenally insufficient**?***
- AM cortisol vs. ACTH stimulation test

(a) Serum cortisol: draw random serum cortisol at 8am

Cortisol	**Interpretation** (based on early am values)
<80nmol/L	Diagnostic if multiple symptoms correlate with dx (if symptoms do not correlate, must do ACTH stim test)
>415nmol/L	Diagnosis is unlikely though cannot rule out

(b) 8am ACTH stimulation test (non-emergent diagnostic test)

(1) Measure pre-test serum cortisol and ACTH
(2) Cosyntropin® 250mcg IV/IM x 1
(3) Serum cortisol at 30 or 60min post-stim

Cortisol Post-Stim	**Interpretation**
>500-550 nmol/L + Normal ACTH	No AI
<500-550 nmol/L + -ACTH	1º AI
500-550 nmol/L + ↓ACTH	2º/3º AI, then MRI pituitary
Indeterminate stimulation	Insulin Tolerance Test (Consult Endo)

3. Etiology: Elucidate the cause for the 1º vs 2º adrenal insufficiency.

Acute Adrenal Insufficiency Insufficient basal/stress glucocorticoid needs. Precipitated by acute stress, lack of stress dosing, or D/C chronic glucocorticoids. Mx depends on: (1) Clinical acuity; (2) Known or undiagnosed adrenal insufficiency:

Acuity	**AI Diagnosed**	**AI yet to be Diagnosed**
Mild	2-3x chronic PO steroid dose OR Hydrocortisone 50mg IV q8h if N&V	Depends on the clinical situation. If fairly stable, empirical tx can wait.
Severe	1. Hydrocortisone 100mg IV q8h 2. NS+D5W IV	1. Dexamethasone 4mg IV q6h – Doesn't interfere w/ serum cortisol, but suppresses HPA axis. 2. NS + D5W IV

Discharge - Patient Education:
- Educate the patient on what adrenal insufficiency is
- Direct to Canadian Addison Society (http://www.adisonsociety.ca)
- Emphasize adherence – dangerous to abruptly stop or reduce steroids
- Instruct the patient "Sick Day" Rules – 2-3x usual dose until illness resolves
- If unable to maintain PO intake, seek medication attention ASAP for IV mx
- Medical Alert Bracelet – "Adrenal Insufficiency": can present with aLOC

Authors: Dr. Michael K. Parvizian, Dr. Rana Kandel, Dr. Lawrence Hookey, Dr. Michael Scaffidi *Updated May 2024*

GI Bleed

Upper or Lower?
UGIB: originates proximal to the ligament of Treitz – presents as melena, hematemesis, coffee-ground emesis or hematochezia (brisk upper GI bleed)
LGIB: originates from below ligament of Treitz – presents as hematochezia/BRBPR, occult bleeding or even melena – long term occult blood loss can present as anemia

Melena?

Verify if it is melena: direct visualization or ask the patient, "Is it as BLACK as this (pointing to something black- dark brown doesn't count!)?" - Confirm with a DRE – do NOT do FOBT since this is screening, not for patients with symptoms.

History/Exam:

1. Vitals: check hemodynamic stability (tachycardia, orthostatic changes, presyncope, reduced urine output, JVP) – tachycardia might be blunted by BB
2. History: ask about risk factors - NSAIDs, antiplt/anticoag drugs, EtOH, smoking, PUD, cirrhosis, surgery (GI or aortic), coagulopathies, critical illness
- *Previous Procedures:* EGD, C-scope, GI surgeries
- *Signs of end-organ ischemia:* angina, dyspnea, presyncopal
- Examine: look for stigmata of chronic liver disease (hepatosplenomegaly, ascites, jaundice, spider nevus) i.e. signs of portal HTN and risk of variceal bleed

Lab Investigations

1. CBC (Q6H), -lytes, Cr, urea, AST/ALT/ALP/bili, PT/aPTT/INR, Type & Cross
2. **Elevated Urea to Creatinine Ratio** suggests UGIB
3. Troponin, lactate and VBG
4. ECG look for signs of ischemia

Initial Management:

1. Airway & Breathing: triage patient - consider calling ICU or D4 if unstable. Does this patient require intubation for airway protection & emergent EGD?
2. Circulation: 2x large bore IVs stat. IV fluids bolus and/or blood transfusions (transfuse if overt hemorrhage, do not wait for CBC) - NEED to STABILIZE for ANY definitive tx.
3. Monitored Setting: Frequent vitals if unstable and cardiac monitoring
4. Transfusion: Type & Cross. Use unmatched RBC for profound hypotensive shock unresponsive to crystalloids. **Transfusion:** Target Hb>70g/L OR Hb>80g/L **IF active ischemia** (angina, ischemic ECG change or significant cardiac hx). 1unit should raise Hb by 10 g/L in absence of hemorrhage.
5. Coagulopathies: Hold antiplt/anticoags. Vit K (10mg) IV for reversing warfarin; Prothrombin Complex Concentrates for rapid reversal (see *Hematology*). Transfuse plts if <50.
6. Follow up: Frequent vitals, CBC Q6H, stool charting. Monitor ins/outs.

Upper GI Bleed Management:

a. Medications:

- **Non-variceal bleed suspected:** Pantoprazole 80mg IV then 8mg/hr or 40mg IV BID x 72 hours.
- **Patient has cirrhosis add-on:** All GI bleeds in patients with cirrhosis should get Ceftriaxone 1g IV Q24H x 5 days even if not variceal.
- **Variceal bleed suspected add-on:** Octreotide 50mcg IV bolus then 50mcg/h infusion x 5 days
- **Consult GI:** EGD to identify source + therapeutic intervention.
- **Consult IVR for Embolization:** if endoscopy fails/cannot perform endoscopy.

Lower GI Bleed Management:

a. Stable Lower Bleed: Consult Gastroenterology- without bowel preparation, c-scope/flex sig can't visualize anything; NOT urgent interventions. Stabilize first.

b. Brisk Lower Bleed Suspected:

1. Identify the source:

- **CT Angiogram:** the patient needs to be bleeding FAST for this scan to work
- **Tagged RBC Scan:** if CT angiogram failed, or bleed is slower rate

2. Consult IVR for Embolization: discuss feasibility & risks (e.g distal ischemia).

3. Consult General Surgery: if unable to intervene yet continues to hemorrhage

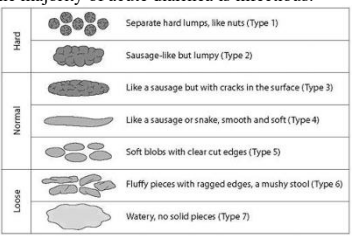
Diarrhea

Approach:

1. What is meant by "Diarrhea"? Clarify stool volume/weight and use Bristol Chart.

2. Even if the etiology is not apparent on hx, certain features can differentiate causes

3. Acute <4wks vs chronic >4wks: Acute maybe the 1st presentation of a chronic diarrhea illness. *The majority of acute diarrhea is infectious.



Features	Notes
Baseline	"Good days" can be perceived as "normal".
Duration	Acute vs Chronic. Beware of normalization.
Course	Acuity of onset; periodicity of exacerbation and relief.
Stool Characteristics	Description can be unreliable. Bristol Stool Chart is objective. Blood: inflammation. Food particles: ↑motility, ↓absorb. Steatorrhea: ↓absorb
Volume	"Small" & "large" volumes are usually suggestive of large & small bowel origin, respectively.
Frequency	NPO relieves diarrhea: osmotic, ↓absorb, motility; Nocturnal diarrhea despite NPO: secretory
Exacerbate	(1) PO; (2) Travel; (3) Source of water; (4) GI sx
Relieve	NPO, ↑ Fiber

Associated Symptoms	Nutrition Deficiency: Wt. loss, edema, micronutrient deficiency manifestations, incontinence and urgency

Chronic Diarrhea:

Class	Features	Common Causes
Inflam	Hematochezia, fever; high CRP.	IBD, chronic ischemia, radiation; Campylobacter, Salmonella
Osmotic	Stops when osmotic agent D/C	Meds: Osmotic laxatives; Gastric bypass
↓Absorb	Stops when NPO, Steatorrhea	Pancreatic insufficiency, Celiac Dx; Gastric bypass
Secretory	Nocturnal diarrhea despite NPO	Neuroendocrine CA; Infection: V. cholera, Giardia
Motility		**Overflow**, IBS, DM neuropathy, short gut, ↑thyroid

Investigations:
- Stool charting: record frequency and characteristics, colour
- Stool fat test: Tedious and unpleasant – collection and special diet required. Inform the lab & patient. Send for fecal elastase.

Unintentional Weight Loss

Unintentional weight loss: significant when > 5% of body weight over 6-12mo
Focused history:
- Pattern of weight loss: progressive vs. stable vs. fluctuating
- Use the subjective global assessment (gold standard for malnutrition diagnosis) & calculate BMI
- Functional factors: obtaining food, preparing meals, feeding self, chewing, swallowing

Causes	Features	Pertinent Investigation
Malignant	1. GI: abdo pain/bloating, early satiety, N&V, dysphagia 2. Lung: SOB, cough, hemoptysis 3. Lymphoma/Leukemia: constitutional sx, lumps 4. Renal, prostate etc.	CBC, lytes (hypercalcemia), Renal function & U/A, AST/ALT/ALP, Albumin, PT/NR, age appropriate cancer screening (not FIT/FOBT) Consider: ESR, CRP and imaging (CXR, CT scan)
Non-Malignant	1. Malabsorption: steatorrhea, diarrhea, bloating, N&V 2. Peptic ulcer disease: abdominal pain, dyspepsia, UGIB 3. IBD: diarrhea +/-blood, extraintestinal manifestations	Iron studies, Folic acid, B12, celiac testing, 72hr fecal fat, fecal elastase. Fecal calprotectin if suspecting IBD and consult GI for possible scope.
Endocrine	1. Hyperthyroidism: 2. T1DM usually normal or ↑appetite, rarely in T2DM 3. Adrenal Insufficiency 4. Pheochromocytoma	TSH +/- fT4, glucose & HbA1c AM cortisol Urine metanephrines
Infections	HIV, TB, Hep C, helminths	Travel Hx., HIV & Hepatitis serology

		TST
Chronic disease	CHF, asthma, COPD, bronchiectasis, CF, CKD	Detailed PMHx and severity, glucocorticoid treatments.
Neuro disease	Stroke, Parkinson's, ALS, altered cognition, dementia	Consider SLP consult to assess swallowing.
Rheum	1. RA: arthritis, extra-articular manifestations 2. GCA: headaches, proximal myopathy, fever, jaw claudication	RF, anti-CCP, ANA ESR & CRP as screening, temp a. bx.
Meds	Diabetes or thyroid medications, AEDs, cholinesterase inhibitors	Medication history.
Substances	EtOH, cocaine, amphetamines	Social hx, EtOH level, Tox screen
Psych disorders	1. Eating disorders 2. Depression 3. Bipolar disorder, ADHD (secondary to medications)	Psych hx and medication review.

Management:
- Consider the need for enteral feeds (NG etc.) Start low and go slow
- Dietician consult for assistance with feeding formulations and monitoring
- Monitor closely for **refeeding syndrome:** ↓Ph, ↓K, ↓Mg & ↑glucose, pulm edema

Celiac Disease

Pathophysiology: Autoimmune rxn to gluten causes small bowel inflammation, malabsorption & diarrhea

Clinical presentation:

1. Malabsorption: weight loss, fatigue, folate and iron deficiency
2. GI: abdo pain, bloating, steatorrhea, oral ulcers, dyspepsia, ↑ALT&ST
3. Skin: Dermatitis herpetiform
4. Nutritional deficiency: Folate/Vit D/iron deficiency & osteoporosis
5. Complications: increased risk of GI malignancies

Diagnosis on gluten-rich diet:
- Always send TTG with IgA level because of IgA deficiency
- If IgA deficient send IgG TTG and DGP (direct gliadin protein antibody)
- Serology and histology are dependent on gluten RICH diet

1. High prob: Get a TTG antibody and IgA level + duodenal biopsy
Both positive = celiac disease *Both negative* = celiac unlikely
Biopsy/serology disagreement = send genotype (HLA-DQ2/8), if IgA deficient send IgG TTG +/- DGP antibodies and w/u for other causes of villous atrophy

2. Low prob: Get TTG + IgA level
Positive TTG: Get a duodenal biopsy *Negative TTG with normal IgA*: Celiac unlikely
Negative TTG and low IgA: Send IgG TTG/DGP antibodies and if negative celiac is unlikely and if positive should have duodenal bx.

** Other causes of villous atrophy: giardiasis, small bowel overgrowth, CVID

Diagnosis on gluten-free diet (GFD):
- Patients on GFD may have normal serology and histology
- Genotype analysis (HLA-DQ2/8) does NOT depend on gluten intake

- If genotype negative then celiac disease is ruled out but if positive need to do gluten challenge (3 g gluten daily for 6 weeks) as tolerated

Celiac disease vs. non-celiac gluten sensitivity:
Symptom response with GF is not diagnostic and may represent non-celiac gluten sensitivity, which is only considered when celiac disease is ruled out.

Management of Celiac Disease:
- Most effective treatment is a gluten-free diet.
- Gluten containing foods → BROW (Barley, Rye, Oats, Wheat)
- Monitor for nutritional deficiencies
- Refer to Dietician & consider GI follow up if complex patient

Dysphagia

Oropharyngeal dysphagia: Difficulty initiating swallow
Esophageal dysphagia: Sensation of food being stuck in the neck or chest

Focused History:

If chronic:
1. Difficulty swallowing liquids or/and solids? Was it solids first then liquids?
2. Difficulty with initiating swallowing? Does food feel stuck and if so, where (higher up or lower down)?
3. Associated sx: pain, regurgitation, cough, drooling, hematemesis, wt. loss, GERD
4. Is dysphagia intermittent vs constant vs progressively worsening

Oropharyngeal	
Structural (usually solids>liquids)	*Neuromuscular (usually solids & liquids)*
Zenker's diverticulum: a/w aspiration	Stroke: Residual neuro deficits
Malignancy or radiation injury	ALS
Infection (HSV, Oral candidiasis)	CNS tumors
Goiter	Myopathy
Proximal stricture/web/ring	Parkinson's disease

Esophageal	
Structural (EGD ± barium swallow)	*Neuromuscular (dx with manometry)*
Rings & webs: -Schatzki ring: usually near GE jxn -Esoph. web: usually proximal	**Distal Esophageal Spasms:** -Uncoordinated peristalsis & hypercontractile, intermittent sx
Eosinophilic esophagitis: -dx with EGD + biopsy -often in younger patients	**Achalasia:** prog. Sx (solids --> liquids). Distal "birds beak" on barium swallow
Peptic or malignant stricture	**DM, scleroderma, amyloid:** -hypomotility
Infectious esophagitis: -esp. immunocompromised patients -CMV, Candida *a/w odynophagia	**Ineffective esoph. motility:** - >50% swallows are weak or failed
Pill esophagitis: -(±) odynophagia -NSAIDs, bisphosph., tetracyclines	**Absent contractility:** -Idiopathic or assoc. with systemic disease -Persistent or intermittent dysphagia

Investigations & Mgmt:
- Barium swallow +/- EGD +/- Manometry testing- Cine esophagram supplies much more info than regular swallow
- Mgmt depends on diagnosis and may warrant GI specialty input +/- intervention (ie. dilation, stents)

Inflammatory Bowel Disease

Ulcerative Colitis (UC): Superficial Inflammation of colonic mucosa starting at rectum and extending proximally in contiguous manner
Crohn's disease (CD): Transmural inflammation that can occur anywhere in GI tract (mouth to anus) and is not contiguous (ie. Skip lesions)
- Diagnosis of IBD is established by combining history/physical exam, imaging, endoscopic and histological assessments.
- Although classically contiguous inflammation is indicative of UC, patients on steroid therapy can have patchy inflammation on endoscopic assessment as different areas can start the healing process, which can mimic CD endoscopically.

	Ulcerative Colitis	Crohn's Disease
Location	Continuous colonic inflamm. With rectal involvement. Proctitis, left-sided (up to the splenic flexure), or pancolitis.	Any portion of the GI tract, most commonly the terminal ileum and colon.
Symptoms	Bloody diarrhea, abdo pain, urgency/tenesmus, nocturnal symptoms, incontinence	Luminal inflammation (diarrhea +/- blood), abdo pain, fever/sepsis could be suggestive of penetrating complication, obstructive symptoms if stricture present
Endoscopic features	Friable mucosa with diffuse ulceration, often difficult to differentiate based on endoscopic features	
Histological features	Mucosal distribution, continuous disease (no skip lesions), architectural distortion, gland disruption, crypt abscess	Transmural inflammation with skip lesions +/- noncaseating granulomas (present 30% of time but pathonomonic)
Gastroenterology Complications and Associations	Toxic megacolon, perforation, stricture, CRC	Perianal disease (fissures, fistulae, abscesses); strictures, abscess, malabsorption, aphthous ulcers, CRC (if Crohn's Colitis)
Extra-intestinal	Dermatologic: pyoderma gangrenosum, erythema nodosum Ocular: episcleritis, uveitis, Rheumatologic: peripheral arthritis, ank spond, sacroiliitis Renal: Nephrolithiasis (i.e. calcium oxalate) Other: VTE, vasculitis, Vitamin deficiencies, assoc. with PSC	

Investigations:
- CBC, lytes/Cr, AST/ALT/ALP/bilirubin, iron panel/B12
- CRP often elevated (useful for monitoring) & Fecal calprotectin (indicator of luminal inflammation)
- Exclude other causes: stool C&S, O&P, C. difficile, celiac testing, ±CMV
- Colonoscopy ± small bowel assessment (e.g. MR enterography; for CD)

Active flare Management:
- Determine if patient needs to be admitted (i.e. dehydrated, pain or diarrhea)

- IV fluids and pain 41gmt.. as needed & send w/u to rule out other causes like infection
- IV solumedrol 40-60 mg total daily dose with close monitoring for complications (e.g. toxic megacolon) once infectious causes ruled out.
- VTE prophylaxis even with hematochezia given high risk of VTE
- Stool charting
- GI consult +/- general surgery consult in severe or refractory disease

Acute Severe UC:

- 10-15% of flares. Important to recognize given high chance of M&M
- Requires multidisciplinary approach (GI, Gen Sx) as failure of initial medical management by day 3 of corticosteroids should prompt escalation to rescue biologic therapy ± referral to colectomy if no response to medical therapy by day 7
- Infliximab is the agent of choice for rescue/salvage therapy (for both CD and UC). Consideration can be made for Tofacitinib (JAK inhibitor) prior to proceeding with colectomy.

Classification of disease severity (Truelove and Witts' Criteria)

Severity	Characteristics
Mild	< 4 stools/day +/- blood, normal ESR, no signs of toxicity
Moderate	4-6 stools/day, occ. Blood, minimal toxicity, CRP < 30 mg/L
Severe	> 5 stools/day AND any of: o Fever (> 37.8˚C) o Tachycardia (>90 bpm) o Anemia (Hgb < 105 g/L) o ESR >30 mm/hr, CRP >30 mg/L
Fulminant	10 stools/day, continuous bleeding, toxicity, abdominal tenderness/distention, requiring pRBC transfusion, colonic dilation

Maintenance therapy:

1. 5-ASA can be used to induce remission & maintenance (esp. in UC: acts topically)
2. Immunosuppressant (6-MCP, Azathioprine, MTX): effective steroid-sparing agents
3. Check TMPT first and monitor for bone marrow suppression
4. Biologics: Infliximab, Adalimumab, Vedolizumab, Ustekinumab, Tofacitinib
5. Pre-biologic work-up includes (IGRA/TST, Hep B and C serologies) to exclude TB and viral hepatitis prior to starting tx as can be reactivated with immunosuppression
6. If patient is not responding to current regimen, can increase dose, decrease interval between injections/infusions, or change to another biologic

Common Biologic Therapy in IBD:

Agent	Mechanism of Action	Considerations
Infliximab (Remicade)	Anti-TNF	Assoc. skin cancers, drug induced SLE, systemic effects from immunosuppression Can be used for IBD and Ank Spond
Adalimumab (Humira)	Anti-TNF (subQ)	Similar side effect profile to Infliximab Can be used for IBD and Ankylosing Spondylitis
Ustekinumab (Stelara)	IL-12, -23 inhibitor	Can be used to treat both IBD and psoriasis
Vedolizumab (Entyvio)	α4-β7 inhibitor (prevents lymphocytes from entering colon)	Gut specific, safest profile Does not act on current inflammation, takes weeks
Tofacitinib	JAK inhibitor (oral small molecule)	Strong immunosuppressive effect Oral therapy

Cirrhosis

Diagnosis: (1) Definitive: complete exam of liver (autopsy or after transplant);
(2) Biopsy: 80-100% sensitivity;
(3) Inferred: clinical, laboratory, and radiological features of portal hypertension
Predictors:

NAFLD Score (http://nafldscore.com/): Use this score for the NAFLD group to estimate the severity of fibrosis. F3-4: significant fibrosis to result in cirrhosis.
FIB-4 Score
APRI Score: this score has been validated for Hepatitis C to estimate severity of fibrosis.

Etiology: *Common* - Alcohol, NAFLD/MASLD, chronic Hep B/C, NAFLD/MASLD. *Infrequent* - PBC, PSC, AIH, HH. *Rare* - A1AT, Wilson, others.
History: (1) Etiology (2) Evidence of decompensation (variceal bleed, ascites, encephalopathy, jaundice)
Laboratory:
- CBC (↓Plt), lytes (↓Na^+), Cr; PT/aPTT; AST, ALT, ALP, TBili, Alb, glucose
- Etiology workup is case dependent: Do not just order everything

Cirrhosis Complications:

(a) Hepatic Encephalopathy

Diagnosis: A clinical diagnosis. NH_4^+ level has poor correlation with HE.
Severity: Grade 0: Normal **Grade 1:** Δ sleep, ↓ attention **Grade 2:** confused, asterixis, **Grade 3:** stupor, clonus, Babinski +ve **Grade 4:** coma
MOST Common Precipitant: ↓ lactulose compliance, GI bleed, ***SBP**, drug toxicity, acute fulminant hepatic failure (*always check for ascites)
Management:
1. Intubation? If too stuporous, too agitated for any tx.
2. Lactulose
- **Retention Enema**: lactulose 200g (300mL) in 700mL H_2O retention enema for 30min q6h, or 20g NG q1h until BM, then QID.
- **Chronic:** lactulose 20g PO TID-QID for 3 soft stools/day (When precipitant treated, 80% no need for further lactulose. Slowly taper to see if needed.). Add PEG 3350 if not tolerating lactulose or inadequate response. If persistent encephalopathy after meeting target BMs can add rifaximin.

3. Address underlying precipitant(s)

(b) Hepatorenal Syndrome (HRS)

Suspect When: AKI in a cirrhotic **with** ascites. A diagnosis of exclusion.
Steps to Diagnosis of Exclusion: Be quick to suspect, workup, and treat.
1. Rule out causes of AKI <u>rapidly</u>: $FENa^+$ <1% in pre-renal and HRS, but $FENa^+$ improves with volume challenge in pre-renal
2. D/C diuretics, nephrotoxic drugs, and non-selective beta-blocker
3. Vol. challenge with albumin: usually 25% albumin 100mL IV BID x 2 days
4. Seek and Tx Precipitant: SBP, GI bleed, infections
5. Consult GI and Nephrology early once you have completed step 1-4
6. Temporizing Measures:
- Vol. Expansion: 25% Albumin 100mL IV BID (50g albumin/day)
- ↓Portal HTN: Octreotide 50mg/hr IV infusion
- ↑MAP by 10mmHg: Midodrine 7.5mg PO q8h, up to 15mg q8h or norepinephrine

(c) Ascites 2º Portal HTN

General Tx:
1. Na^+ restrict: <88mmol (<2g)/day.
2. H_2O restrict: when Na^+<120mmol/L
3. Medications: start Spironolactone 100mg OD <u>+</u> Furosemide 40mg OD. Titrate to max Spironolactone 400mg OD + Furosemide 160mg OD.

4. Therapeutic Paracentesis: If the patient is hypotensive, just drain enough to relieve the tension. For large volume paracentesis (more than 5 L removed) replace with 8 g of albumin per litre (see paracentesis or albumin orderset).

Recurrent Ascites:

[Na^+] restriction non-compliance is most common cause for difficult to control ascites. Consider when: >1 paracentesis in 2 weeks if each tap removes ~10L.

Spot Urine [Na^+]>[K^+] + weight (fluid) loss: diuretic sensitive and compliant to [Na^+] restriction

Spot Urine [Na^+]>[K^+] + no weight (fluid) loss: diuretic sensitive yet NOT compliant to [Na^+] restriction

Spot Urine [Na^+]<[K^+] + no weight (fluid) loss: diuretic resistance

(d) Spontaneous Bacterial Peritonitis

Diagnosis: PMN>250x10^{6}cell = (Total WBC in x10^{6}cells under *Fluid Cell Count*) x (Proportion of Neutrophils under *Fluid Differential*). <30% of cultures will be +ve. Clinical features nonspecific, may be asymptomatic or have hepatic encephalopathy or abdo pain

1. Antibiotic Mx and Source Control: Ceftriaxone 2g IV OD x 5 days

2. Albumin Infusion if AKI: 1.5g/kg Day 1→ 1g/kg Day 3

3. SBP Prophylaxis: after 1st SBP**.** Norfloxacin 400mg OD (drug of choice); Ciprofloxacin 750mg PO Qweekly; Septra DS 1tab PO OD.

Acute Pancreatitis

Diagnosis: >=2 of the following

(a) Typical abd. pain, (b) ↑ Lipase/amylase >3xULN, (c) Radiographic evidence

Types:

1. Interstitial Edematous (90%): Local or diffuse pancreatic edema. May be complicated by a local peripancreatic fluid collection early, which may later become a pancreatic pseudocyst.

2. Necrotizing (10%): Local or diffuse pancreatic and/or peripancreatic necrosis. May be complicated by a local acute necrotic collection early, which may become an area of walled off necrosis later.

Causes: I-GET-SMASHED

Idiopathic (15%); **Gallstones** (40% – including microlithiasis); **EtOH** (30%); **T**rauma; **S**moking; **M**umps; **A**utoimmune (2 subtypes); **S**corpion (Trinidad species); **H**yperCa^{+2}/**H**ypertriglyceridemia/**H**ypothermia; **E**RCP (3% diagnostic ERCP 5%, therapeutic ERCP); **D**rugs (1%): in wks to months (highest risk: furosemide, metronidazole, valproate, simvastatin, ozempic style drugs)

Investigations:

1. Lipase; Ca^{+2}T; LFT; Triglyceride (>11.2mM a/w pancreatitis 2º ↑TG but TG ↑ during acute episode and after fatty meals); IgG4 (autoimmune: "sausage" pancreas on CT)

2. US: Spe. 10% for CBD stone/dilatation. CBD diameter: 6mm but up to 1cm with ↑age & cholecystectomy.

3. CT w/ contrast: ?complications. If obtained <72hrs from onset underestimates severity. (use CTSI prognosis scale)

4. Post-ERCP pancreatitis: 75% ↑ lipase post-ERCP but 5% develops clinical pancreatitis. Lipase <1000U/L 2hrs post-procedure: NPV 98%.

Mx: Mainly supportive for SIRS

1. Analgesia: Morphine 2.5-5mg SC/IV q2-4h PRN pain

2. IV Fluid: Maintain intravascular euvolemia. 10ml/kg bolus if hypovolemic and 1.5ml/kg/hour with frequent volume status reassessment. Aggressive vol. repletion out of favour as could increase morbidity. Consider RL as NS yields NAGMA.

3. Nutrition: Early enteric feeding. Low Fat Diet for PO. If unable to tolerate PO, Peptamen® NG. Consider TPN if enteric feeding will be greatly delayed.
4. Gallstone Pancreatitis: ERCP (consult GI). Cholecystectomy (consult GenSx). (If deferred, ~18% for recurrent pancreatitis, or biliary related complications within next 3mths).

Prognosis: Various options available (BISAP, Ranson's Criteria, etc.)

BISAP Score – various = performing scoring systems

1 point for each in Admission: •ΔLOC, •>60 years old, •Pleural effusions, •SIRS +ve, •Urea>8.9mmol/L

Total Score (% mortality): 0-2 (~2%), 3 (3.6%), 4 (7.5%), 5, (9.5%)

CRP at 48hr >150mg/L – predicts severe course (Sen 80%, Spec 76%)

Complications:

1. SIRS: 3rd spacing, multi-organ failure, ARDS, etc…
2. Necrotizing Pancreatitis: About 1/3 will develop infection.
- Consider in those who deteriorate or do not improve for >7-10 days.
- CT abd. to diagnosis pancreatic bed necrosis (usually takes 48-72hrs)
- Infected necrosis: clinically difficult to distinguish SIRS from sepsis.
- If working diagnosis is infected necrosis → empiric meropenem 1g IV x 1 (consult ID for approval). Consult GenSx & GI: possible surgical or endoscopic debridement vs IVR drainage.

3. Pancreatic pseudocysts: consider if abdominal pain persists ~4 weeks after resolution of acute pancreatitis episode.

Elevated Liver Enzymes

Hepatocellular pattern: ↑AST&ALT +/- Conjugated Bili

1. Viral hepatitis: Hepatitis (A, B, C, D, E), CMV, EBV, VZV, HSV
2. Drugs & toxins: Alcohol, acetaminophen, salicylate, prescriptions, OTCs
3. Autoimmune hepatitis: ANA, Anti-SMA, Anti-LKM, anti-SLA, QIgs
4. Vascular: Budd-Chiari, SOS (sinusoidal obstructive syndrome)
5. Hereditary: Wilsons, A1AT deficiency, hemochromatosis
6. NAFLD: Diabetes Mellitus and hypercholesterolemia
7. Malignancy

Cholestatic pattern: Initial rise in AST/ALT then Tbili and ALP

All patients require an abdominal ultrasound
1. Extrahepatic Cholestasis (Biliary dilation on ultrasound): CBD stone, biliary stricture, malignancy, PSC. Consult GI for ERCP
2. Intrahepatic Cholestasis (No biliary dilation on ultrasound): Drugs, PBC, PSC, TPN, sepsis, pregnancy, congenital. Consider MRCP if no cause is found.

Infiltrative pattern: ↑ALP/GGT +/- Bili, AST&ALT

All patients require a GGT to confirm a hepatic source
1. Malignancy: HCC, metastatic disease, lymphoma
2. Infectious: TB, Histoplasmosis
3. Other: amyloidosis, sarcoidosis

Acute Liver Failure

Definition: Acute liver injury + hepatic encephalopathy + coagulopathy (INR>1.5) in those w/o cirrhosis or preexisting liver disease. Acute liver failure defined as duration <26wks. Poor prognosis with high mortality rate.

Etiology:

1. **Drugs/toxins:** Acetaminophen (most common), anti-TB drugs, AEDs
2. **Toxins:** Amanita phalloides (mushroom sp.)
3. **Viral:** Hepatitis, HSV, EBV, CMV, HHV6
4. **Vascular:** Budd-Chiari, ischemic hepatitis, SOS
5. **Other:** Wilsons, HELLP, autoimmune hepatitis

Clinical features:

1. **Neurological:** Encephalopathy
2. **CVS:** Hypotension, shock
3. **Resp:** Resp alkalosis, pulm edema, ARDS
4. **GI:** bleed (diathesis), jaundice
5. **Renal:** HRS, ATN, electrolyte abn (↓K,Na,Ph)
6. **Heme:** ↓Plt, ↓Fbrinogen,↑PT/PTT, DIC
7. **Endo:** Hypoglycemia, met. Acidosis (↑ lactate)
8. **Infections:** SBP, bacteremia

Treatment:

1. **ABCs**: assess whether ICU is needed for close monitoring of vitals, sugars, ICP
2. **Coagulopathy**: give Vit K 5-10mg IV, FFP 2-4U if active bleeding
3. **Infection:** start broad spectrum antibiotics – don't wait for blood cx
4. **AKI/HRS**: albumin/midodrine/octreotide (see above)
5. Consider **NAC** if acetaminophen related
6. Consider **liver transplant** (consult GI)

Colorectal Cancer Screening

Average-Risk Screening:

Who: No personal or family history of CRC, IBD, or familial CRC syndromes that is currently ages 50-74.
How: Fecal Immunochemical Test q2years
What to do if positive: Colonoscopy within 8 weeks
Future Screening modality/time: Based on polyp type (if any) found. See CancerCareOntario's Post-Polypectomy Guidelines

Common High-Risk Screening Groups:

1. **First-degree relative with CRC:** Start 10 years before diagnosis or 10 years before onset of relatives CRC. Colonoscopy q5 years (if onset < 60 years) or q10 years (onset >10 years).
2. **IBD:** If UC or Crohn's Colitis start 8-10 years post-diagnosis if pancolitis or 15 if left-sided (up to splenic flexure). Frequency based on time-since diagnosis.
3. **Lynch Syndrome:** Start at ages 20-25 with colonoscopy q1-2 years.
4. **FAP:** Start at ages 10-12 with colonoscopy every year.

Types of polyps: Each will be described by site, morphology, size, and degree of dysplasia (if any).

1. **Adenomas** (>50%): Tubular (45% of total, 2% malignancy risk), Tubulovillous (6%, 20-25% malignancy risk), villous (1% of total, 15-40% malignancy risk)
2. **Hyperplastic** (35%): No malignant potential.
3. **Serrated adenomas:** May be sessile serrated or traditional serrated.
4. **Inflammatory:** Benign foci of inflamed mucosa/scar tissue. Seen in conditions such as IBD.
5. **Hamartoma**: Benign growth of a disorganized mixture of cells/tissue.

Irritable Bowel Syndrome (IBS)

Diagnostic Criteria: All of

1. Abdominal pain >=1 day per week in the last 3 months (on average)
2. Pain associated with >=2 of: defecation, change in stool frequency, change in stool consistency.
3. Criteria fulfilled for the last 3 months with symptom onset >=6 months prior to diagnosis

Subtypes: Based on Bristol Stool Chart

IBS-C: >25% of stools are Bristol Stool 1-2
IBS-D: >25% of stools are Bristol Stool 6-7
IBS-M: >25% of stools are Bristol Stool 1-2 and another >25% Bristol Stool 6-7
IBS-U: Meets diagnostic criteria for IBS but not for a subtype.

Treatment: All patients will need individualized therapy regimens

General: Exercise (3-5 times weekly), fiber (dietary or supplement), food diary (to identify personal triggers and avoid), FODMAP diet (esp IBS-D), psychotherapy, antidepressant (TCA, SSRI)
IBS-C: Osmotic laxatives (PEG), Chloride channel activator (lubiprostone), antibiotics (neomycin), 5-HT4 agonists (has risk of severe adverse effects so only use if severe/refractory).
IBS-D: Opiates (loperamide), antibiotics (rifaximin), 5-HT3 antagonists (has risk of severe adverse effects so only if severe/refractory. Evidence only in females).
Pain: Antispasmodics (hyoscyamine), psychotherapy, antidepressant

Gastroparesis

Definition: Syndrome of delayed gastric emptying in the absence of mechanical obstruction. Most common symptoms include nausea, vomiting, early satiety, belching, bloating +/- epigastric abdominal pain.

Etiologies:

1.Diabetes Mellitus: Usually more pronounced in T1DM. Typically in those who have had DM for >5 years and if poorly controlled DM. Secondary to autonomic dysfunction.
2. Rheumatological: amyloidosis and scleroderma
3. Autoimmune: autoimmune gastrointestinal dysmotility which can be idiopathic or associated with neoplasm such as SCLC.
4. Neurological: parkinsonism, multiple sclerosis, brainstem CVA and tumours.
5. Medications: narcotics, TCAs, alpha 2 agonists (clonidine), CCB, GLP-1 agonists, octreotide etc.
6. Viral infections: Norwalk virus and rotavirus. Can also develop post viral gastroparesis.
7. Post surgical: any surgery that poses a risk to the vagus nerve.

Investigations

1. Exclude mechanical obstruction: EGD or imaging such as CT or MR enterography can be used to exclude mechanical obstruction.
2. Assess gastric motility: Most common test is a **scintigraphic gastric emptying study** – low fat egg white meal with imaging immediately after ingestion and again at 1, 2, and 4 hours following ingestion. Delayed gastric emptying is defined as:

1. Gastric retention >10% at four hours and/or
2. >60% at two hours

Treatment/Management:

1. Lifestyle modifications: Eat frequent small sized meals. Avoid carbonated drinks, smoking and alcohol intake. Avoid high fat content meals and nondigestible fiber.
2. Diabetes: optimizing glycemic control.
3. Antiemetics & prokinetics: Options include metoclopramide and domperidone which are taken three times daily before meals. Other options include antiemetics to use as PRN such as ondansetron, diphenhydramine or prochlorperazine.
*Monitor QTc regularly.
4. Macrolide antibiotic: Erythromycin but maximum use is four weeks. Prone to tachyphylaxis and side effects such as ototoxicity, bacterial resistance and QT prolongation.
5. Refractory gastroparesis: Gastric electrical stimulation, G-POEM etc.(all experimental treatment options at this point)
*Often, improving gastric emptying does not correlate with a noted change in sx.

Geriatrics

Authors: Dr. Kit Birkness, Dr. Chris Frank *May 2024*

5 M's: *Mind, Mobility, Medications, Multi-Complexity, Matters Most*
Focuses on deprescription, mgmt of delirium/dementia, community resources, rehab

Geriatric Assessment

- *Collateral* should be taken in every Geriatrics assessment
- *Cognitive status*: current, baseline, cognitive concerns (duration, onset, progression, fluctuation), MMSE, MoCA, chief lifelong occupation, education (years)
- *Communication*: speech, hearing, vision
- *Emotional*: mood, fear of falling
- *Perceptions*: hallucinations, delusions
- *Socially engaged* (often/not)
- *Sleep* (normal/disrupted/daytime sleepiness)
- *Substances:* alcohol, smoking
- *Social*: married, lives with, lives in, formal/informal supports, caregiver stress
- *Mobility*: walking, transfers, gait aid, balance, falls with number and timeline
- *Continence*: bowels, bladder
- *Nutrition:* weight, appetite
- *ADLs* (ind/assist/dep): feeding, bathing, dressing, toileting
- *iADLS* (ind/assist/dep)*:* cooking, cleaning, grocery, medication, finances, driving status
- *Medications*: organize by medical indication, consider role in presenting illness
- *Clinical frailty scale rating* (1-9) using status two weeks prior to acute illness onset
- *Code Status* and focus of care

Agitation

Agitation can be an imprecise term: ask about specific behaviours, psychotic fts, physical/environmental precipitants, whether it is in the context of delirium/dementia
- *Assess for triggers*: urinary retention (bladder scan), pain, dyspnea, constipation, hypoglycemia, medications/toxins, infection or metabolic cause, environment
- *Management*: If not aggressive and no psychotic features, consider:
Trazadone 6.25mg (if frail) OR 12.5mg-25mg po q6h PRN
- For severe distress, risk of harm to self or others:
Quetiapine 6.25mg-25mg po bid (x1 OR short-term then taper) OR Haloperidol 0.5mg IM/IV (2mg max) OR Loxapine 6.25mg IM OR Lorazepam 0.5mg IM/IV (2mg max)

Delirium

Definition: acute change in conscious state characterized by inattention, hypo- or hyperactivity (or mixed), and disorganized thinking. Can have fluctuations in LOC.
Assessment: attention (do WORLD forward and backward, months backward, days of the week backward, or serial subtraction from 100 by 7), orientation, + collateral
- Physical: GCS, full neurological exam, signs of infection incl skin breakdown, CVA tenderness, and evidence of constipation/urinary retention
- Screening Tools: Confusion Assessment Method (CAM), 4A's Test
- Ddx: DIMS-PLUS5

Drugs: new, changes, anticholinergics, benzos, opiates, steroids, sedatives, toxins
Infection: source from urinary tract, skin/soft tissue, GI system, lungs, meningitis
Metabolic: electrolytes, dehydration, thyroid, hypoxia/hypercarbia, liver/kidney
Structural: neurologic (stroke, seizure), other
Pain: Poorly controlled pain
Liquids/Solids: dehydration, malnutrition
Urine/bowels: retention, constipation
Senses: vision, hearing, communication deficits
Sleep: disrupted sleep, napping
Stasis: restraints (including IV lines, catheters)
Stress: psychological distress

Management
- DOS charting to identify patterns and precipitating factors
- Supportive care: hearing aids, glasses, frequent reorientation, family to bring in familiar objects, don't wake throughout night and do rouse throughout the day
- Treat underlying cause of delirium

Dementia (Major Neurocognitive Disorder)

Definition: chronic cognitive decline which interfere with independence in everyday activities, which do not occur exclusively in the context of delirium

Stage	Memory Impairment (**CURE**)	Functional Impairment (**IRAN**)
Mild	**C**urrent events/news	**I**ADLS
Moderate	**U**S President/PM	**R**ewears clothes (needs cuing for ADLs)
Severe	**R**elatives (1st degree)	**A**DLs
Very Severe	**E**verything	**N**on-verbal, **N**on-ambulatory

Orthostatic Hypotension

Pathophysiology: deficit in maintaining intracranial pressure
Definition: sustained reduction in sBP by 20mmHg or in dBP by 10mmHg within 3min of standing
Ddx: 4D-AID. Causes with compensatory tachycardia: 4D. Without: AID.
4Ds: Deconditioning, Dysfunctional heart (CHF, AS), Dehydration, Drugs (anti-HTN)
AID: Autonomic dysfunction (diabetic autonomic neuropathy, Parkinsonism, low B12, hypothyroid, ethanol), Idiopathic (pure autonomic failure), Drugs (B-blockers)
Treatment: Address underlying cause, then consider: Abdo binders (at KGH)
Midodrine 2.5mg po bid (9am and 4pm), contraindicated in active ischemia
OR Fludrocortisone 0.05-0.1mg po daily, contraindicated in CHF

Falls

Ddx: presyncope/syncope (cardiac, neuro), OH, sensory impairments (vision, vestibular, neuropathy, proprioception), neurologic/psych (stroke, PD, cognition, mood), MSK (arthritis, pain), performance measures (weakness, balance, gait abnm), drugs, environmental factors

Authors: Dr. Nicole Relke, Dr. Paula James, Dr Pallavi Ganguli, Dr. Jeannie Callum, Dr. Kerstin Dewitt, Dr. Bethany Monteith, Dr. Kit Birkness *Updated May 2024*

Blood Products

Consent: obtain from patient or SDM with signed form. You can initiate transfusions without consent if life-threatening emergency but must get consent as soon as possible. *Note: explicitly ask Jehovah's Witnesses if they would accept specific blood products

RBC Transfusion: (see below for Transfusion Reactions)

RBC Transfusion Threshold for Adult Inpatients:

- Hb<50 g/L: for uncomplicated vaso-occlusive pain crisis in sickle cell disease
- Hb<50 g/L for chronic iron deficiency without serious symptoms (+ IV Iron)
- Hb<70g/L: hemodynamically stable pts, including those with active bleeding
- Hb<70g/L: pts with hematological/oncologic disorders
- Hb<75g/L: pts undergoing cardiac surgery
- Hb<80g/L: pts undergoing hip fracture surgery, pts with acute myocardial infarction
- Hb<90g/L if hemodynamic symptoms (tachycardia, pre-syncope, etc.)

- In absence of active hemorrhage, order 1 unit (300mL) at a time
- 1 unit of RBCs should increase Hb by 10g/L

Orders:

- *1 unit RBCs over 2h (*max *4h* if cardiac hx, volume overload)
- Group and screen is automatic when you order pRBCs
- Use online order set when time allows (includes indications for irradiation, etc.)
- Consider *Lasix 20 (*or *40mg) IV x1* prior to infusion if volume overload, hx CHF
- *Repeat CBC post-transfusion* if active bleed, otherwise following AM
- RARELY[1] order *type and cross x units RBCs (do not transfuse)*[2] for complicated pts requiring full IAT crossmatch (e.g. Hx multiple antibodies, Sickle cell disease)

[1]Clearly tell nursing that the order is not for immediate transfusion

[2]Rare population going to OR needs crossmatched units, e.g. re-do cardiac surgery

Acute GIB Principles of Care:

- The more blood you give, the more bleeding there is: onboard RBC transfusions in patients who are actively bleeding unless very unstable
- Frequent monitoring: vitals, lay eyes, CBCs until scope
- Do not need to activate code transfusion to get unmatched RBCs
- Consider activating massive hemorrhage protocol if 2-4 units uncrossmatched RBCs given, but ongoing poor hemodynamic response and continued hemorrhage
- Guidelines recommend against plasma transfusion for variceal bleed (even if ↑INR)

Principles of Massive Transfusion Protocol:

- Target transfusion ratio 1:1:2 of plasma : platelets : RBCs by end of code
- Permissive hypotension: allow BP below normal so long as mentating well
- Reverse anticoagulants
- Blood work q30-60min: maintain hb>70, platelets>50, fibrinogen>1.5
- Monitor for ↓Ca, ↑K q1h; consider giving IV calcium q4units RBCs
- Terminate Code Transfusion by calling Blood Bank when bleeding source controlled and rate of transfusion has slowed

Platelet Transfusion

Indication: **Plt (x10^9/L) Threshold**	
Active Bleeding	<50 (<80-100 if CNS bleed)
Neurosurgery	<80

Major Surgery/High risk invasive procedure	<50 (<30 cirrhosis)
Low risk invasive Procedure	<20
Prophylaxis	<10

Order: *1 unit platelets transfused over 1 hour (max 4h)*
- Repeat CBC within 1 hour of transfusion (1 adult dose - plt by $>7x10^9$/L
- Platelets should only be transfused to patients with ITP, PTP, or HIT after consultation with hematology or transfusion medicine (may increase mortality rate for PTP and HIT, and rarely needed for ITP)

Fresh Frozen Plasma (FFP): 1 unit is 200-290mL, dose is 10-15 mL/kg

Indications: Bleeding or high-risk invasive procedure with INR/PTT > 1.8 (except cirrhosis where plasma should rarely be administered), massive transfusion, coagulopathy post-cardiac surgery, TTP for plasma exchange
Order: *Transfuse 1000 ml FFP over 1-4 hours*
- Typically, 600-800ml for small adults; 1000-1200ml for larger adults
- Max rate over 4 hours; depends on bleeding, CV status
- Use order set
- *Repeat INR immediately post-transfusion*

Fibrinogen Concentrate: 1-gram vials

Indications: (1) Bleeding w/ fibrinogen <1g/L (2) Massive hemorrhage, APML w/ fibrinogen <1.5-2.0g/L (3) ICH 2° TPA w/ fibrinogen <2.0g/L
Order: *Transfuse 4 grams over 5 minutes by IN push or minibag infusion*
Each dose should increase fibrinogen by ~0.5-1.0g/L

Albumin

Indications: (1) Large volume paracentesis (>5L), admin 25g/4L fluid taken off, (2) SBP for HRS prophylaxis day 1&3, (3) Hepatorenal syndrome diagnosis
Order: *Transfuse "X" mL of 25% albumin IV*
- 25% albumin in 100mL bottle, see Order Set for dosage for each indication
- Each 100ml of 25% is a 500ml volume expansion
- Albumin is associated with a risk of TACO
- 5% Albumin almost never indicated except for plasma exchange

Prothrombin Complex Concentrate (Octaplex®): Vol. 40mL per 1000IU
Heparinized (Contraindicated for HIT): lasts: ~6hrs

Definition: Factor concentrate of vitamin K-dependent factors, II, VII, IX, X
Indication: Emergency reversal of warfarin or vitamin K deficiency when:
(1) INR>1.5 AND (2) life threatening bleeding or emergency surgery
Contraindication: PCC should not be given when vitamin K alone sufficient

Situation	Vitamin K
INR >8-10, no bleeding	2mg PO
Surgery >6 hours later	10mg IV
Non-critical bleeding	1mg IV

Orders: *Octaplex X IU, each 1000 IU over 5min, ± Vitamin K 10mg IV x1*
- *Repeat INR immediately post-transfusion*

INR	PCC Dose (*X*)
<3	1,000 IU
3-5	2,000 IU
>5	3,000 IU
NOAC	2,000 IU

- PCC for NOACs: can give second dose of 2000IU if ongoing bleeding at 2h
- For life-threatening bleeding, do not wait for INR: give PCC 2,000IU, rpt INR
- See Order Set for Anticoagulant Reversal

Acute Transfusion Reactions

Risks of Transfusion from RBC per unit:

Febrile reaction	1: 300	Anaphylaxis	1: 40,000
Allergic Reaction	1: 100	HBV	1: 2 million
TACO	1: 100	HCV	1: 27 million
TRALI	1: 10,000	HIV	1: 13 million
RBC Antibody	1: 13	DHTR	1: 2,500

- Mandatory (by Health Canada) to report ALL transfusion reactions (even mild urticarial reactions or mild TACO) to Blood Bank
- Stop transfusion, perform bedside assessment, check for clerical errors
- Send blood and tubing back to lab for testing for more severe reactions

Febrile Reactions

a. Acute Hemolytic Transfusion Reactions: fever, hemoglobinuria, dyspnea, hypotension, DIC, renal failure, nausea/ vomiting.
Etiology: Usually due to ABO incompatibility and incorrect patient identification.
Management: Stop transfusion, disconnect transfusion tubing. Send group and screen and DAT to blood bank. Send hemolytic, DIC, and renal labs. Send U/A.

b. Acute Febrile, Non-Hemolytic Transfusion Reactions: fever ≤ 4 hours from transfusion
Management: Stop transfusion. Order IgA and haptoglobin levels. Acetaminophen. If any symptoms or temp >39ºC, send group and screen and DAT to blood bank and blood cultures to microbiology
Prevention: washed RBC if significant and recurrent

c. Bacterial Contamination: Sepsis (hypotension, fever, tachycardia, rigors)
Management: Stop transfusion. Gram stain and culture of blood product and patient from different IV site. Start antibiotic therapy.

Allergic Reactions

a. Anaphylaxis: urticaria, hypotension, hypoxia, stridor/wheeze, dyspnea, N & V
Management: Stop transfusion. See *Anaphylaxis and Anaphylactoid Reaction* for further management.
Prevention: IV steroids, diphenhydramine.

b. Allergic Reactions: urticaria, pruritis
Management: Stop transfusion if severe, slow infusion if <2/3 of body surface area. Diphenhydramine 25-50mg PO/IV.

Dyspnea

a. Transfusion related acute lung injury (TRALI): acute hypoxia <90% RA, bilateral infiltrates on CXR, no evidence of circulatory overload
Management: Stop transfusion. Respiratory supportive measures (oxygen supplementation/NIPPV). Send samples to blood bank for donor-recipient HLA antibody testing.

b. Transfusion associated circulatory overload (TACO): dyspnea, orthopnea, tachycardia, JVP, hypertension, cardiogenic pulmonary edema (CXR, BNP)
Management: Slow/stop transfusion. Respiratory supportive measures (oxygen supplementation/NIPPV). Furosemide

Hypotension

a. Bradykinin mediated hypotension: isolated hypotension, associated with ACE inhibitors
Management: Fluid and inotropic support. Consult transfusion medicine before

providing any further transfusions

Massive Transfusion-Related

a. Coagulopathy - Hemorrhagic: tends to occur after 4-8 units RBCs due to dilution of coagulation factors (RBC doesn't contain clotting factors or platelets)
Prevention: Transfuse 4 units RBC, with 2 units plasma (2:1 ratio) only in the context of a Code Transfusion. Order platelets if platelet count drops below 50 $x10^9$/L. Code Transfusion and 2:1 is only for the first 30-60 minutes of hemorrhagic shock until the results of lab testing can guide component therapy.

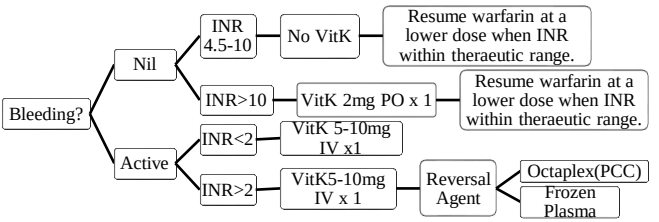
b. Citrate toxicity: exacerbated in those with liver dysfunction. Results in:
- **Metabolic Alkalosis:** citrate metabolism generates bicarbonate
- **Hypocalcemia:** citrate binds to calcium. Can give calcium prophylactically every 4 units

c. Hyperkalemia: due to RBC lysis during storage

d. Hypothermia: RBC is cold!
Management: Warm saline and blood infusion, Bair Hugger
Prevention: Blood Warmer device during transfusion

Anticoagulation and Antiplatelet Reversal

Warfarin



DOACs

1. Discontinue DOAC. Supportive Mx.
2. Improve renal function – many of the DOACS are primarily renally cleared.
3. Dabigatran has a reversal agent – idarucizumab
4. For other DOACs and life-threatening bleeding – Octaplex (PCC)

Antiplatelets

1. Discontinue anti-platelet (if risk of hemorrhage outweighs thrombotic risk)
2. Supportive management (platelet transfusion appears to be of no benefit)

Venous Thromboembolism – DVT, PE

Risk Factors for VTE: Older age, prior history of PE or DVT, hospitalization, immobilization, surgery, trauma, estrogen therapy, pregnancy (especially postpartum period), malignancy. Most patients with VTE have no risk factors!

Deep Vein Thrombosis

Approach

1. Symptoms: Unilateral calf pain, unilateral swelling, erythema, palpable cord
2. Diagnosis for outpatients - Wells score for DVT:
Score ≤ 1: do D-dimer. Score ≥ 2: do D-dimer + doppler U/S. If doppler U/S negative, need either a Wells score of 1 or less or a negative D-dimer to rule out DVT. Otherwise, rule out DVT with a second doppler U/S in 7 days.

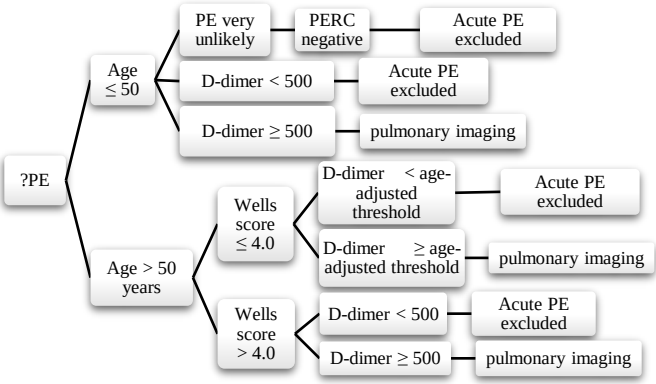
Mx

1. Anticoagulation (**See Below: Anticoagulation Mx**)
2. Phlegmasia cerulea dolens (suspect with cyanosis): consult IVR - cath-directed TPA

Pulmonary Embolism

Approach

1. HPI: Dyspnea, pleuritic chest pain, fatigue, presyncope with activity, hemoptysis
2. Physical findings: ↑RR, ↑HR, often no abnormal findings
3. PE Testing: Exclude without imaging in outpatients or patients being admitted through the emergency department by:
   - 3.1. PE very unlikely and PERC negative, or
   - 3.2. D-dimer < 500, or
   - 3.3. D-dimer < age-adjusted threshold (if patient over age 50 AND Wells score for PE is 4.0 or less)
4. Imaging: CTPA or V/Q if contrast allergy.
5. Diagnosis of hospital-acquired PE in inpatients. D-dimer < 500 make PE very unlikely. Otherwise, patients require imaging.



Mx

1. **ABC:** Attention to hemodynamics – obstructive pathophysiology.
2. **Thrombolytic:** Indication – hemodynamic shock**.** ICH risk: 3%.
3. **Anticoagulation:** Start while investigating for PE if suspicious and no contraindications

Risk Stratification of PE

European Society of Cardiology Guidelines:

Low-risk = sPESI score of 0
Intermediate-low risk = ↑ troponin **or** right ventricular dilation/dysfunction seen on CT/echo **or** sPESI > 0
Intermediate-high risk = ↑ troponin **and** right ventricular dysfunction
High-risk = hemodynamic shock secondary to PE

Disposition

Home treatment: *Low-risk* patients who have necessary home supports, can mobilize, and have no other indication for hospital admission.

Intermediate-low risk patients: may also be treated at home, vitals normal, safe mobilization possible, and no other indications for hospitalization
Intermediate-high and high-risk patients: require admission to carefully monitored environment. For intermediate-high risk patients, document the BOVA score.

VTE Anticoagulation Mx

For high-risk patients (hemodynamic shock caused by PE): if do not have contraindication, treat with thrombolytic:
- Alteplase, dose may vary between 50 mg and 100 mg IV
- Tenecteplase 0.5mg/kg to a maximum of 50 mg IV
Use KHSC thrombolysis order set. Follow with either Dalteparin 100 units/kg SC q12h or IV unfractionated heparin.

For intermediate-high risk patients: Dalteparin 100 units/kg SC q12h
- Patients > 95 kg: Dalteparin 100 units/kg bid, max dose 12,500 units bid
- Plan ahead in case of deterioration to high risk (risk 1/20): assess for thrombolysis contraindications (see order set), determine if within pt GOC to receive thrombolysis should they develop shock/deteriorate
- 1 in 10 risk of major bleeding with thrombolysis, risk is higher in older patients.
Intermediate-low and low-risk patients: Give the first dose of anticoagulation immediately (if not already given). See below.

Are anticoagulants high-risk for this patient? Consider the following:
1. Is their eGFR <20?
2. Is their platelet count <80?
3. Have they had serious bleeding (such as GI bleed or intracranial bleed) within the past 6 months?
4. Were they on another anticoagulant medication at the time of their VTE diagnosis?
If 'YES' to ANY questions above, do not prescribe anticoagulation. Call Hematology for consultation.
If 'NO' to ALL questions above, then they are safe to be anticoagulated.

To decide which agent is indicated, consider the following:
1. Does the patient have a luminal gastrointestinal tumour?
2. Is the patient already prescribed any of:
carbamazepine (Tegretol), phenytoin (Dilantin), phenobarbital, primidone, rifampin, isoniazid, fluconazole (or other antimycotic ----azole), anti-HIV meds?
3. Is the patient pregnant or breast feeding?
If 'YES' to any of Questions 1-3, then prescribe Dalteparin
If 'NO' to all of Questions 1-3, then prescribe Apixaban or Rivaroxaban

Prescriptions:
- Apixaban 10 mg bid x 7 days, then 5 mg bid x 90 days (LU code 444) OR
- Rivaroxaban 15 mg bid x21 days, then 20 mg daily x 90 days (LU code 444) OR
- Dalteparin 200 units/kg daily x 14 days, with one repeat. (LU code 188).
Round to nearest prefilled syringe dose: 7,500/ 10,000/ 12,500/ 15,000/ 18,000U
- Patients > 95 kg: Dalteparin 100 units/kg bid, max dose 12,500 units bid
- Always weigh patient
Prior to discharge: Teach patient to self-inject Dalteparin. Fax Dalteparin prescription to patient's pharmacy in advance or check whether pharmacy has Dalteparin in stock (pharmacy will order for next day delivery).

Note: do not use IV unfractionated heparin unless creatinine clearance is < 20 ml/min or the patient has a high risk of imminent bleeding.

Follow up:

- Refer patient to the Thrombosis clinic.
- If the patient is co-prescribed carbamazepine, phenytoin, phenobarbital, primidone, rifampin, isoniazid, fluconazole (or other ----azole antimycotic), anti-HIV meds, refer patient to anticoagulation management clinic for warfarin mgmt.

IVC Filter:

Only indication: Bleed risk so high that patient cannot be anticoagulated.
Team who inserts filter is responsible for ensuring removal as soon as possible.
Complications of leaving IVC in situ: filter fracture, migration, thrombosis; IVC perforation; increased risk of DVT.

Multiple Myeloma

Plasma cell neoplasm producing monoclonal immunoglobulin "M-protein"

Presentation: "CRAB" features

- **C**alcium (↑), **R**enal insufficiency, **A**nemia (usually normocytic), **B**one lytic lesions. Other: infections, fatigue, weight loss, radiculopathy.

Diagnostic Criteria:
- SLiM CRAB + clonal plasma cells (PC) (BM>10% or plasmacytoma);
- SLiM = BM ≥60% PC, light chain ratio >100, > 1 MRI lytic lesion ≥ 5mm;
- CRAB = Ca >2.75mmol/L, Cr >177 umol/L, Hgb <100g/L, boney lesions

Initial Investigations: CBC, Cr, ionized calcium, total protein, albumin, quantitative immunoglobulins (IgG, IgA, IgM), SPEP and UPEP with immunofixation, serum free light chain. Skeletal survey.

Management:

- Hematology consultation for bone marrow biopsy
- **NOTE: Spinal cord compression** is a medical emergency. Timely diagnosis + treatment prevent irreversible neurological damage.
  See *Spinal Cord Compression Secondary to Malignancy*.
- Assess need for Nephrology consult/urgent dialysis in renal failure. Early steroids and chemotherapy may improve rate of renal recovery.

Heparin Induced Thrombocytopenia

Type 1: Non-immune mediated. Mild thrombocytopenia on day 1-2 after exposure to heparin. Returns to normal in 2-5 days without discontinuing heparin. No tx necessary. Not associated with thrombosis.
Type 2: Immune-mediated (antibodies to PF4-heparin complex). Causes thrombocytopenia and thrombosis. Timing tends to occur during Day 5-14 after exposure to heparin or its derivatives.
When to Consider HIT: Trend of platelet count reduction, thrombosis (venous or arterial), + recent heparin exposure

4T Score: Provides Pre-Test Probability of HIT ↓↑

Parameters	2 pt	1 pt	0 pt
Platelet count	>50% ↓Plt **and** Nadir ≥$20x10^9$/L	30-50% ↓Plt; **or** Nadir 10-$19x10^9$/L	<30% ↓Plt; **or** Nadir <$10x10^9$/L
Timing of platelet count fall	Clear onset 5-14 days; **or** day 1 if	Onset 5-14 days but not clear; **or** onset after day	Onset < 4 days (no recent heparin)

	heparin exposure within 30 days	14; **or** day 1 if heparin exposure >30 days ago	
Thrombosis	New thrombosis; Skin necrosis; anaphylactoid reaction to IV heparin bolus	Progressive or recurrent VTE; Suspected VTE; Red skin lesions	None
Other Causes?	Nil	Possibly	Yes
Interpretation – Total Score: ≤3pts: <1% for HIT**;** 4-5pts: ~15%**;** ≥6pts: ~65%			

Laboratory Investigations: Do not order HIT assay if 4T score is low (≤ 3)

(a) Immunoassay – ELISA: detects the presence of PF4-heparin Antibody

Sensitivity	Specificity	PPV	NPV*
91-97%	74-86%	50-93%	**95%**

*High NPV. If the ELISA assay is negative, functional assay will not be pursued.

(b) Functional assay – Serotonin release assay: detect antibodies that induce heparin-dependent platelet activation– takes about 1-2 weeks for results

Management:

- Discontinue all heparin indefinitely! (Including heparin locks or flushes)
- Start non-heparin anticoagulant: DOAC, fondaparinux, argatroban, bivalirudin
- Avoid warfarin in acute HIT (worsening prothrombotic state)
- Warfarin can be started when platelets ≥ 150 with overlapping non-heparin anticoagulation ≥5 days and until INR therapeutic.
- Note: Use order set when transitioning from argatroban to warfarin
- All patients with HIT should have bilateral leg doppler U/S to rule out DVT
- Platelet transfusion **not** recommended due to thrombosis (unless sig bleeding)
- If heparin strongly indicated/needed in the future (i.e. cardiopulmonary bypass), consult Hematology for evaluation and management plan

Infectious Disease

Authors: Dr. Andrew McNaughton, Dr. Alison Sumner, Sue McKenna, Dr. Evan Wilson, Dr. Michael Scaffidi

Updated May 2024

Approach to Infectious Disease and Common Antibiotics

Approach to ID: Questions to Consider

- Vitals and clinical stability
- Risk factors for infection
- Immune status
- Typical vs atypical pathogens
- Community vs hospital-acquired
- Source and source control
- Prev culture and antibiotic history
- Culture validity – contamination, colonization, antibiotic tx
- Correlation of cultures with symptoms, signs, or imaging
- Local pathogen, susceptibility and resistance patterns
- Indications for antibiotics

PEARLS

- Presence of bacteria does not always signify infection
- Fever in a sick patient does not always signify infection
- Antibiotics are not benign
- Consider renal function prior to prescribing and double check for dose adjustments!
- Not all inpatient antibiotics need to be administered via IV route
- American guidelines are NOT applicable to everywhere

Kingston Health Sciences Centre Antimicrobial Stewardship
(https://khscnow.kingstonhsc.ca/cr/antimicrobial-stewardship-program, from KHSC terminal**):** local antibiograms, and recommendations tailored to KHSC

	Cocci	**Bacilli**
Gram positive (G+ve)	Staphylococci, Streptococci Enterococci, Peptostreptococci (anaerobe)	*Clostridioides* (an anerobe formerly Clostrida), Corynebacteria, Listeria, Bacillus, Nocardia, Actinomyces (anerobe)
Gram negative (G-ve)	*Neisseria meningitidis, Neisseria gonorrheae, Moraxella catarrhalis*	Bacteroides (anaerobe) *E. coli, Klebsiella, Enterobacter Proteus, Salmonella, Shigella, Pseudomonas, Acinetobacter, Haemophilus influenzae Helicobacter, Campylobacter, Legionella*

Class	**Body flora Site**	**Potential Disease**
Gram positive (G+ve)	skin, mucous membranes	cellulitis, respiratory tract infection (RTI), osteomyelitis, line infection
Gram negative(G-ve)	gastrointestinal (GI), genitourinary (GU)	biliary, urinary tract infection (UTI), pelvic inflammatory disease (PID)
Atypicals	respiratory tract, GU	pneumonia, UTI, PID
Anaerobes	mouth, throat, sinus, female GU, distal bowel	abscess, dental infections, appendicitis

Four Moments of Prescribing Antibiotics

1. *Make the diagnosis*. Viral? Non-infectious? Antibiotics necessary?
2. *Appropriate cultures and empiric therapy.* Assess RFs, check severity, choose broad-spectrum vs narrow-spectrum
3. *Revise therapy.* Change antibiotics? IV to oral? D/c?
4. *Duration.* Shortest effective duration? Changes?

Central Line-Associated Bloodstream Infections

Diagnostic criteria: any of the following

a. **Blood culture from CVC lumen and peripheral vein grows same organism**
b. **Catheter tip and peripheral blood grow same organism:** AFTER removing the CVC and cutting the tip of the line for culture

How to Draw the Cultures: 2 sets from each lumen of CVC, 1 set from peripheral vein. 1 set = one aerobic bottle and one anaerobic bottle

Contaminants: Common organisms that are contaminants include coagulase-negative *Staphylococcus spp. Bacillus spp.* and *Corynebacterium spp*.

***S. aureus and Candida albicans* is never treated as a contaminant**. In general, growth from a SINGLE blood culture in a patient with no other features of infection (normal vitals, afebrile, normal WBC) is typically a contaminant

Pathogens:

a. Coagulase negative *Staphylococcus*: (e.g., S. *epidermidis, S. hemolyticus, S. intermedius*, etc.). Note: S. *lugdunensis* causes endocarditis and metastatic infections; treat like *S. aureus*)

- **Antibiotics:** vancomycin 15 mg/kg IV q12h pending susceptibilities; cefazolin 2 grams IV q8h or cloxacillin 2 grams IV q4-6h if methicillin-susceptible
- **Duration: 7 days**
- **CVC Removal:** if CVC cultures remain positive, or sepsis secondary to CVC infection

b. *Staphylococcus aureus -MR/SSA*

- cefazolin 2 grams IV q8h or cloxacillin 2g IV q4h (MSSA); vancomycin 15 mg/kg IV q8-12h (MRSA)
- **Duration:** 14 days following first negative repeat blood cultures if no infective endocarditis
- **CVC Removal:** Yes

c. *Enterococcus faecalis, E. faecium*

- **Antibiotic:** start with vancomycin 15 mg/kg IV q12h pending susceptibilities and transition to ampicillin 2 grams IV q4h if susceptible
- For Vancomycin-resistance (VRE); contact asK/ID team for approval to use linezolid 600 mg PO/IV q12h or Daptomycin 6 mg/kg IV q24h
- Duration: 14 days if no infective endocarditis
- **CVC Removal:** Yes

d. Gram Negative Bacilli [includes extended-spectrum beta-lactamase producing (ESBL) *E. coli or Klebsiella* species*]*

- **Antibiotic:**
- Not septic/no healthcare exposure: ceftriaxone 1 gram IV q24h
- Not septic/with healthcare exposure/ known GNB colonization: ceftazidime 1-2 grams IV q8h or gentamicin 6 mg/kg IV q24h (if normal renal function, ESBL GNB colonization)
- Septic: meropenem 1 gram IV q8h or gentamicin 6 mg/kg IV q24h
- **Duration**:7 to 14 days, tailor narrowest agent according to susceptibility

CVC Removal: Yes

e. Candida

- **Antifungal:** Fluconazole 6 mg/kg (round to nearest 100 mg) po/IV q24h (C. *albicans, C. parapsilosis, C. tropicalis etc.*)
- Caspofungin 70 mg IV once then 50 mg IV q24h (C. *glabrata*, C. *krusei*)
- Duration: 14 d following first negative blood cultures if no infective endocarditis
- **CVC Removal:** Yes

PEARLS Does the patient need the CVC? Establish IV with POCUS

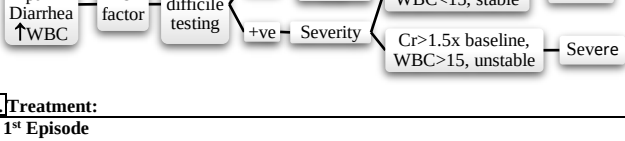
Clostridiidoides (formerly Clostridium) Difficile Infection (CDI)

Risk Factors for *C. difficile* Infection: (1) Antibiotic exposure (increased risk with prolonged therapy, multiple antibiotics); (2) Hospitalized in acute center; (3) Long-term care facility; (4) Elderly; (5) History of CDI (6) PPI or H2 blocker (7) GI surgery (8) Chemotherapy

Antibiotics Risk: ALL antibiotics have the potential to trigger CDI

- **High Risk:** second and third generation cephalosporins (cefuroxime, ceftriaxone, ceftazidime etc.), fluoroquinolones, clindamycin
- **Medium Risk:** penicillins, beta-lactam + beta-lactamase inhibitors (pip-tazo, amox-clav), vancomycin, carbapenems, macrolides, metronidazole
- **Low:** aminoglycosides, tetracyclines, sulfonamides, rifampin

1. Diagnosis and Classification:



2. Treatment:

1st Episode	
Mild/Severe	vancomycin 125mg PO QID x 10-14 days
Severe **with** sepsis, megacolon, ileus	General surgery consultation AND vancomycin 125 - 500mg PO/NG QID x 14 days AND metronidazole 500mg IV q8h. In ileus, vancomycin 500mg PR q6h
First recurrence	Mild/Severe, uncomplicated: vancomycin 125mg PO QID x 14 d
2nd or subsequent recurrences	vancomycin taper: 125mg PO QID x 14 days → 125mg TID x 7 days → 125mg BID x 7 days → 125mg QD x 7 days → 125mg q2 days x 2 weeks

3. Stop other antibiotic if possible, or use an antibiotic with a narrower spectrum
4. Discontinue PPI or H2 blocker therapy if possible
5. Avoid anti-peristaltic medications

If severe allergy to oral vancomycin, give fidaxomicin 200 mg PO BID x 10 days
Repeat *C.difficile* testing for "cure" not recommended

Infective Endocarditis (IE)

Important Info:

- Symptoms: non-specific, especially subacute IE – fever, night sweats, weight loss
- Valvular lesion: history of valvular heart disease, prosthetic valve
- Sources: recent dental procedures, surgery, intravenous drug use, IV catheter, intra-cardiac device
- Complications: valvular regurgitation, heart failure, conduction block, emboli
- Most common pathogens: Viridians streptococci (*S. milleri, anginosus, mitis/oralis, etc.), S. aureus*, *Enterococcus spp.* HACEK organisms
- Bacteremia ≠ endocarditis, & ECHO "abnormalities" ≠ endocarditis

Modified Duke Criteria	
Major:	
Microbiologic	Typical organism (Viridans streptococcus spp., *S. aureus*, HACEK, *Enterococcus spp.*) identified in 2 separate BC drawn 12hrs apart
	1+ve BC for *Coxiella burnetii* (not routinely done), **or** IgG antibody titer for Q fever phase 1 Ag >1:800
Echocardiographic	ECHO +ve for vegetation, abscess, new partial dehiscence of prosthetic valve

	ECHO evidence of new valvular regurgitation
Minor:	Predisposing heart condition or IDU
	Fever >38ºC
	Vascular phenomena: major arterial emboli, septic pulmonary infarct, mycotic aneurysm, ICH, Janeway's lesions
	Immune phenomena: Osler's nodes, Roth's spots, GN, RF+ve
	Microbiologic evidence: +ve BC with no major criteria met, or serologic evidence of infection with an organism consistent with IE
"Definite" if 2 major criteria, or 1 major and 3 minor criteria, or 5 minor criteria or pathologic evidence; "Possible" if: 1 major criteria and 1 minor criteria, or 3 minor criteria	

Investigations: Echocardiogram – TTE may be adequate but if negative and high index of suspicion for IE remains, obtain TEE. Prosthetic valve, cardiac device, concern for abscess should warrant TEE. Imaging for embolic events as appropriate (CT head / CT chest/abdo / MRI spine). Serology for blood-borne viruses if IDU (HIV, Hep B, Hep C)

Management: (2015 ESC Guidelines; 2015 IDSA Guidelines)
Empiric Antibiotic Therapy: Empiric therapy should only be started after **at least two sets of blood cultures** are drawn. It should be reserved for acutely ill patients with signs and symptoms that are strongly suggestive of IE. An empiric regimen should cover MRSA/MSSA, streptococci, and Enterococci, (HACEK)

Native valve	vancomycin 15-20mg/kg IV q12h AND ceftriaxone 2g IV q24h
Prosthetic valve	vancomycin 15-20mg/kg IV q12h AND gentamicin 1mg/kg IV q8h AND rifampin 300 mg PO q8h (make sure to double check for drug interactions)

Targeted Antibiotic Therapy:
Consult ID for expert input especially when the organism is identified, Enterococcal infection, or a pathogen with complex resistance pattern.
Duration of Therapy:
Should be calculated from the first day of negative blood cultures. Treatment duration for native valve IE ranges from 4-6 weeks, and for prosthetic valve IE is generally 6 weeks. Refer to consensus guidelines/ID consult service if unclear.
What Should be Removed?:
- IV catheters (consult with Nephro regarding HD line removal)
- Cardiac devices (TEE with lead/valve vegetation; staph/candida infection; high grade bacteremia with coag-neg staph/Cutibacterium; CIED pocket infection)
- AV fistula (consult Nephro/ID)

Indications for Surgical Intervention: (1) heart failure refractory to medical management; (2) Perivalvular extension – abscess; (3) Progressive heart block; (4) Fungal pathogen; (5) Large vegetation (>1-1.5cm); (6) Persistent bacteremia despite optimal antibiotics; (7) Recurrent emboli on optimal antibiotics; (8) Prosthetic valve endocarditis.

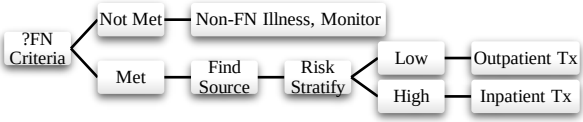
Febrile Neutropenia (FN)

Definition

- **Fever:** ≥38.3ºC x1, or sustained ≥38ºC >1hr (Be wary of fever suppression: acetaminophen, prednisone, elderly)
- **Neutropenia:** ANC<$1.0x10^9$/L (ASCO 2012), or expected to ↓ <$0.5x10^9$/L in next 48hr if current ANC<$1x10^9$/L.

Approach

1. Overarching Schema



2. Finding the Source: Pan-culture not recommended. Inves. should be based on Hx+Exam. (a) Mandatory: CBC+diff., renal panel, liver panel, blood culture x2 sets. (b) Adjunct: CXR, urine culture.

Pathogens: (a) Occult: 60%; (b) Source found: 30%. Bacterial: (a) G+ve (70%): S. aureus, S. epi., Strep; (b) G-ve (30-40%); (c) Anerobe (<5%): if +ve, consider abscess. Fungal: occurs usually after 1wk of FN+antibiotic Tx.

3. Risk Stratification:

Low Risk (all met): (a) No/mild symptoms; (b) ∞ hemodynamics; (c) No major comorbidities; (d) Non-heme cancer; (e) Age<60; (f) Neutropenia expected <7d; (g) Tolerate PO; (h) Close to hospital for follow up (<30min away); (i) MASCC score >21

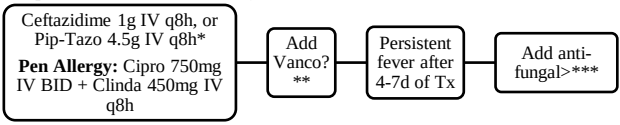
High Risk: (a) Failure to meet Low Risk Criteria; (b) Heme Transplant; (c) Infectious focus identified; (d) Febrile for >48hr; (e) FN DESPITE on Neupogen®/Neulasta®.

4. Mx: (ASCO 2019 – Outpatient Mx; IDSA 2010 – Inpatient Mx)

a) Outpatient:

No Penicillin Allergy	Cipro 750mg BID + amoxicillin/clavulanate 875/125mg q12h
Penicillin Allergy	Cipro 750mg BID + Clinda 450mg q8h

b) Inpatient (Refer to PCS EntryPoint order set for FN)



*Hx of highly Ω pathogens – eg. ESBL: use meropenem 1g IV QD instead
**Add vanco if (a) suspected catheter-related sepsis; (b) Grade 3-4 Mucositis; (c) MRSA colonized; (d) Unstable; (e) Pneumonia on radiography.
*** Consider add anti-fungal if persistent fever after 4-7d Tx + neutropenia duration expected to be >7d, in which case: Consult ID.

c) Duration of Empirical Tx: (a) If afebrile+ANC >1.0: D/C antibiotics when afebrile x2 days; (b) If afebrile+ANC<1.0, can stop if cultures negative, no focus of infection found, vitals normal and afebrile >72h.

d) Caveats of empirical Tx: (a) Clinically Stable: rarely need to alter empirical Tx even if fever persists; (b) If vanco, or other specific abx was added initially for clinical reasons, it should be d/c'd if susceptible bacteria is not isolated.

Infectious Meningitis and Encephalitis

Clinical Suspicion:

- Headache, neck stiffness, fever, altered mental status: 2 of 4 present in 95%
- Rash: viral exanthem (enterovirus), non-blanchable petechiae/purpura (*N.meningitidis*), genital/orolabial vesicles (HSV1/2)
- Exam maneuvers: NOT sufficiently sensitive to rule out either disease – if you suspect meningitis/encephalitis, an LP is mandatory

Pathogens – Clues:

- Sexual History: high risk sexual activity, history of STI, infectious genital symptoms
- Local infection: sinusitis, otitis media, mastoiditis, recent NeuroSx
- Immunosuppression: HIV (toxoplasmosis., Cryptococcus spp., TB), asplenia (*S. pneumoniae*, *N.meningitidis*, *Haemophilus influenzae*), HypoIg (meningococcus)
- Time of year: summer and late fall – viral predilection (eg. enterovirus, West Nile Virus) also Lyme disease
- Geography: CDC website http://wwwnc.cdc.gov/travel/ for disease activity
- HSV: orbitofrontal lobe (confusion, psychosis, somnolence), temporal lobe (seizures preceded by olfactory, gustatory hallucinations, *déjà vu*, corticospinal tract signs, upper quadrant visual field loss, aphasia if dominant hemisphere affected)

Age 2-50	*N. meningitidis*, *S. pneumonia*
Age >50	*S. pneumoniae*, *N. meningitidis*, *L monocytogenes*,
CSF shunt	Coag -ve Staph, *S. aureus*, *Cutibacterium acnes*, *P. aeruginosa*
Post-NeuroSx	*S. aureus*, Coag -ve Staph., *P. aeruginosa*, *Cutibacterium acnes*

Investigations

General: Peripheral BCx x 2, CBC, Cr, PT/INR, lactate – if appropriate: Lyme, WNV, HIV, Syphilis serologies and Cryptococcal antigen.

Lumbar Puncture:

- Tube 1: Cell count + differential
- Tube 2: Culture and sensitivity
- Tube 3: Glucose and protein
- Tube 4: Cell count (compare with Tube 1), cytology, storage of special test Ask for viral PCR (HSV/VZV, enterovirus, CMV and EBV if immunocompromised), AFB, cryptococcal ag, VDRL – if RFs for any

Imaging: Indications for CT before LP – papilledema, significant immunocompromise (AIDS, transplant, high dose steroids, chemotherapy), new onset seizures, GCS <12

CSF Analysis

	Normal	**Bacterial**	**Aseptic (viral / Lyme)**	**Fungal/TB**
Appearance	Clear	Turbid	Clear	Fibrin web
Protein (g/L)	0.18-0.45	>1	<1	>1
Glucose (mmol/L)	2.5-3.5	<2.2	Normal	1.6-2.5
Gram stain	Normal	60-90% positive	Normal	-

CSF:Serum glucose	0.6	<0.4	>0.6	<0.4
CSF lactate	<3	>6	<3	No data
WCC	<3	>500	<1000	100-500
Other		Neutrophil predominance	Lymphocyte predominance	Lymphocyte predominance

Mx

1. **DO NOT DELAY Antibiotics for CT! Though attempt LP before Abx**
2. **ABC –** LOC may be compromised; intubation may be required
3. **Isolation:** Contact and droplet precautions
4. **Empiric Antibiotics:**

	Regimen
Community	ceftriaxone 2g IV q12h
Community – age >50, imm comp.	ceftriaxone 2g IV q12h AND ampicillin 2g IV q4h
Recent international travel	As above and ADD vancomycin 20-25mg/kg IV q12h; consider lower dose and more frequent if younger and ok renal function
Nosocomial/ Recent NeuroSx	vancomycin 20-25mg/kg IV q12h AND ceftazidime 2g IV q8h
Severe beta-lactam Allergy	vancomycin 20-25mg/kg IV q12h AND moxifloxacin 400mg IV q24h AND TMP/SMX 5 mg/kg IV q6-8h

5. **Steroids:** dexamethasone 10mg IV q6h x 4 days (give before antibiotics; Discontinue if *S.pneumoniae* or *H.influenzae* ruled out)
6. **Empirical antiviral:** acyclovir 10mg/kg IV q8h (If suspect viral encephalitis – discontinue if HSV/VZV CSF PCR is negative)
7. **Consult: ID**; NeuroSx, or Neuro depending on the situation
8. **Prophylaxis of close contacts:** Community contacts are the responsibility of Public Health and HCWs are responsibility of Occupational Health.

Pneumonia

Approach:

Key Questions:

- **Antibiotic Hx:** Review Abx use in <u>past 3mths</u> – consider using a different class
- **Culture Hx:** Pathogen and sensitivity/Ω, and notable <u>colonization</u>
- **Patient is from:** Community acquired – home, retirement home; Nosocomial – >48hrs after admission, nursing home, extended care, recent admission
- **Risks:** Seasonal (eg. Influenza); Recreational: Fungal (eg. Histoplasmosis); Immunosuppression (eg. PCP)
- **Extrapulmonary signs/symptoms:** Confusion, abdominal pain, diarrhea, bradycardia, rash, arthritis

 Recurrence: frequent – consider risk factors and immunodeficiency

 Allergy: CLARIFY the Rxn

Risk Stratification:
- Clinical predication tool (MDCalc!) – Pneumonia Severity Index (PSI) recommended over CURB-65 in most recent CAP guidelines.
- **Respiratory Failure, Sepsis** → Inpatient Mx.
- **Complications:** empyema, ARDS, pulmonary cavitation → Inpatient Mx.

Empiric Antibiotic Regimen:

CAP Outpatient	amoxicillin 1g PO q8h OR doxycycline 100 mg PO q12h OR azithromycin 500 mg PO q24h
CAP Outpatient w/ co-morbidities*	amox/clav 875mg PO q12h OR cefuroxime 500 mg PO q12h AND doxycycline 100 mg PO q12h OR azithromycin 500 mg PO q24h
CAP Inpatient	ceftriaxone 1g IV q24h AND azithromycin 500mg PO q24h x 3 days OR levofloxacin 750mg PO q24h
CAP requiring ICU admission	ceftriaxone 1g IV q24h AND azithromycin 500mg PO/IV q24h x 3 days OR ceftriaxone 1g IV q24h AND levofloxacin 750 mg PO q24h
HAP/VAP	Pip-Tazo 4.5g IV q6h OR Ceftazidime 2g IV q8h OR levofloxacin 750mg IV q24h AND vancomycin 15mg/kg IV q12h OR (if vanco allergy) linezolid 600mg IV q12h
Aspiration	ceftriaxone 1 g IV q24h OR amox/clav 875 mg PO q12h
MRSA	Vancomycin 15mg/kg IV q12h, consider loading dose of 20-25mg/kg if severe
Pseudomonas	Pip-tazo 4.5g IV q6h

*Co-morbidities: chronic heart, lung, liver, or renal disease; diabetes mellitus; EtOH; malignancy; or asplenia.

Failure to Respond: (a) non-infectious (b) drug/bug mismatch (c) complications/source control (eg. empyema)

Duration of Tx: Uncomplicated pneumonia: 5 days. MRSA / Pseudomonas pneumonia: 14-21 days. Complicated pneumonia (necrotizing, complicated effusion/empyema): until **clinical** (will occur first)+ radiographic resolution – 4-6 wks

Post-Tx CXR: Radiographic resolution takes 2-6wks. CXR in 4wks for age>50 to assess complications/underlying mass.

Prevention: Annual influenza vaccination for <u>all</u>.; Pneumococcal polysaccharide 23 valent vaccine for ≥65yo and those with diseases; Smoking cessation.

COVID-19 Pneumonia

When to Suspect COVID-19:
Any patient presenting with infectious symptoms including:
- Fever
- Cough
- Fatigue
- Anorexia
- Dyspnea
- Myalgias

Key Questions:

- **Vaccine status:** Review vaccination status
- **Exposure history:** place of residence (nursing home?), travel from outside Canada or from community in outbreak (can refer to Public Health Ontario for updated tracking map), sick contacts
- **Risk factors for severe disease:** See below
- **Extrapulmonary signs/symptoms:** Confusion, abdominal pain and diarrhea, signs of thromboembolism (VITT), arrhythmia and cardiac injury
- **Progression of disease:** important to establish onset of and acuity of progression of symptoms (patients with severe COVID-19 pneumonia can progress very quickly to requiring high flow oxygen and/or intubation).

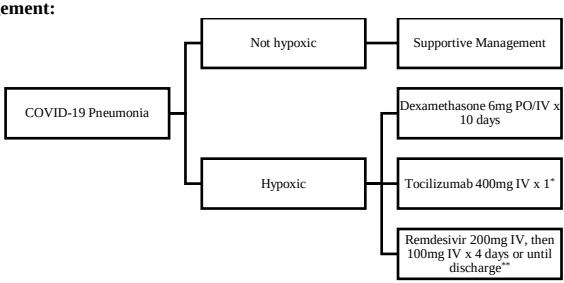
Risk Stratification for Severe Disease:

High Risk	Moderate Risk	Low Risk
• Age ≥60 years • Nursing home resident • Immunocompromised • Chronic lung disease (incl. asthma) • Cardiovascular disease (incl. HTN) • Morbid obesity (BMI ≥ 40kg/m2 • Diabetes • CKD – dialysis • CVD • Liver disease • Tobacco use	• Age 20-64 with no high risk factors • Age <20 years with underlying comorbidity	• Age <20 years without underlying comorbidity

Investigations:

- **Swab all admitted patients for COVID-19!** Please refer to the criteria listed on the COVID-19 Risk Straitification Order Set on PCS
  - Surveillance swabs, or
  - Diagnostic swabs
- **Bloodwork:** lymphopenia, elevated ferritin, CRP, LDH, liver enzymes, PT, D-dimer, trop/BNP
  and CK all typical
- **Cardiac:** ECG for QT monitoring and signs of acute cardiac event
- **Diagnostic imaging: a NORMAL CXR/CT Scan CANNOT exclude diagnosis of COVID-19**
  - CXR: if needed, portable recommended for ease of cleaning and limiting exposure to staff
  - CT Chest: should be used sparingly and reserved for specific indications (e.g. rule out PE, empyema, abscess, etc.)

Management:



* for patients requiring mechanical ventilation, or have increasing O2 requirements despite 2-3 days of dex, or CRP >75mg/L
** only for patients requiring oxygen but NOT non-invasive or invasive ventilation; reduces length of stay, but not mortality
Tocilizumab and Remdesivir should only be administered after consultation with the Infectious Diseases consult service

Failure to Respond: (a) PE! (b) bacterial/viral co-infection (c) complications/source control (eg. empyema)

When to Involve K2ICU: (a) If you're concerned! You are not alone. (b) worsening respiratory distress (c) O2 requirements
> 5L/min via nasal cannula (especially if acute and progressive worsening)

When to Involve ID: (a) From the get-go! While the ID service does not need to formally follow every COVID patient,
many of the ID staff are involved in COVID research (b) in the use of "off label" or investigational therapies (see above)

Prevention: No organized effort for the vaccination of inpatients. Consider discussing with pharmacy to determine if your
patient is eligible. If so, can order: Pfizer-BioNTech vaccine 0.5mL IM x 1; vaccine to be administered by COVID
vaccination clinic.

Skin and Soft Tissue Infection

Approach

1. **Infectious or Not?** Common cellulitis mimics – stasis dermatitis, dependent rubor, acute lipodermatosclerosis, DVT (femoral vein), eczema, gout, lymphedema, erythema migrans
2. **Classify the infection:**

Diagnosis	Erythema	Supportive Features	Mx
Cellulitis	Less defined. May appear suddenly overnight.	±Pain but not out of proportion. ±Superficial necrosis (if severe).	Medical ± source control
Necrotizing Fasciitis	Poorly defined border. Rapid progression (in hrs).	Septic. Pain out of proportion to exam. Crepitus. Bullae. Recent surgery, elevated CK, CRP	**Surgery + antibiotics**

3. Localize 1º source: Cracked skin, trauma, abrasion, ulcer, foreign body, abscess
4. Spread? Lymphatics, deep structures (eg. bone), distal sites (endocarditis)
5. Investigations:

- Superficial wound swab C+S: DO NOT – colonized and usually polymicrobial.
- Deep tissue aspirate/biopsy C+S: useful for purulent collections – abscess, necrotizing fasciitis
- Laboratory findings nonspecific. LRINEC score – poor sensitivity, ?specificity – use with caution

6. Mx:

Nec. Fascitis	
	(1) **EMERGENTLY Consult Ortho**. If **Fournier's gangrene, consult Urology**. Source control is the ONLY intervention that matters. Surgical debridement, fasciotomy ± amputation.
	(2) Empiric Antibiotics: ceftriaxone 2g IV q24h AND metronidazole 500 mg PO/IV q12h OR piperacillin-tazobactam 3.375 g IV q6h OR meropenem 1 g IV q8h ADD vancomycin 15-20mg/kg IV q12h if MRSA coverage indicated
	(3) Consult Infectious Disease: Narrow antibiotics based on culture results

Cellulitis	(1) Empirical Antibiotic Mx – a Guide			
	Risks	Severity	Antibiotic	Route	Duration
	Nil	Mild	cephalexin 500-1000 mg PO q6h	PO	5 days*
	Nil	Mod-Severe	cefazolin 1-2g IV q8h	IV	5-14 days
	DM	Mild-Mod	cephalexin 500-1000 mg PO q6h OR cefazolin 1-2g IV q8h	PO/IV	5 days
	DM	Severe	piperacillin-tazobactam 3.375g IV q6h	IV	5-14 days
	(2) Source Control: drain abscess, debride/amputation necrotic tissue. Foreign body: remove. Cracked skins: emollient, hygiene, and treat *Tinea pedis* – clotrimazole cream 1%				

* Duration of therapy should always be guided by clinical response, regardless of severity/route

*****NB –** erythema may progress over first 24-48h of abx, NOT indication to broaden!!

Ulcer: Diabetic foot is most common. Sacral ulcer is also common*.	(1) Empirical Antibiotic Mx – a Guide			
	Severity	Antibiotic	Route	Duration
	Mild-Mod	**cephalexin, amox/clav, cefazolin	PO/IV	1-wk, if no response consider extended coverage
	Severe	*PipTazo+/-MRSA	IV	≥2wks
	(2) General Care: Debride, Wound care, Nutrition, Pressure relief.			
	Types	**Mx**		
	Arterial	CV optimization. Vascular Sx?		
	Venous	Meds causing edema? Leg elevation (above heart level). Stockings (if arterial sufficient).		
	Neuropathic	Frequently check feet. Podiatry. DM control.		

**** Abx in Sacral Ulcers?** Evidence suggests that there is little role for antibiotic therapy in the treatment of stage IV sacral ulcers if the wound will not be closed. If the would will be closed and there is evidence of OM on bone bx, then appropriate abx.

Goal should always be excellent local wound care. (Clinical Infectious Diseases, Volume 68, Issue 2, 15 January 2019, Pages 338–342, https://doi.org/10.1093/cid/ciy559).

***MRSA Risk Factors: Low prevalence of community acquired and nosocomial MRSA in Kingston**. Consider coverage if: (1) Previous MRSA Infection, (2) MRSA Colonization, (3) Significantly Immunosuppressed, (4) Severe Sepsis, (5) Purulence

Pseudomonas Risk Factors: Low prevalence in Kingston. Consider coverage if: (1) If in high prevalence area, (2) Frequent exposure of foot to water, (3) Previously Pseudomonas infected ulcer.

Osteomyelitis (OM)

Definition: Infection of the bone (Cortex or Marrow)

- **Complications:** Chronic OM, limb threatening, nidus for seeding
- **Source: Localize** (i) Hematogenous (CVC, IHD, IDU, immunocompromised, hardware, sickle cell) (ii) Contiguous (DM, peripheral neuropathy, PVD, peripheral ulcers, hardware) iii) Direct inoculation (iatrogenic, trauma)

JAMA. 2008. 299(7):806:813.		
Does this diabetic have OM of the lower limb?		
Parameter	**PLR**	**NLR**
Probe to bone	**6.4**	0.39
Bone exposure	**9.2**	0.70
Ulcer area >2cm^2	7.2	0.70
ESR≥70mm/hr	11	0.34
X-Ray	2.3	0.63
MRI	3.8	**0.14**

Investigations:

- Labs: CBC+diff, ESR, CRP
- **Superficial wound, or sinus tract swab culture:** limited value, poor correlation with deep space organisms
- Blood culture: useful in hematogenous spread (a positive blood culture for *S.aureus* may obviate the need for bone biopsy!)
- Bone biopsy culture: open, or percutaneous – obtain two samples (C+S and histopathology); most specific of all investigation; cultures are accurate if sample taken through uninvolved tissue; can also be taken at time of irrigation and debridement (intra-op)

Imaging:

- X-ray: 10-14d for radiographic manifestations; low SN/SP; Findings: periosteal elevation, cortical/medullary lucencies, bony destruction
- MRI: high SN+NPV; added value as can assess adjacent tissues (eg. abscess, spinal cord – highly useful in **vertebral OM**); may have limitations if hardware in situ
- CT: high SN+NPV; esp. useful in setting of prosthesis or hardware
- Bone scan: high SN; higher rate of false +ve if suspected site has other inflammatory processes (eg. septic arthritis, DM foot, healed osteomyelitis)
- WBC scan:

Mx:

1. **Consult Orthopedics/Neurosurgery service ASAP** if prosthetic in situ at the area of involvement, or evidence of structural instability (bony instability – pathological fracture, neurovascular compromise)
2. **Empiric antibiotics (only in setting of sepsis or focal neuro deficit):** *S. aureus* and GN aerobic bacilli coverage, and narrow based on culture results.
3. **Clinical Infectious Diseases**, Volume 68, Issue 2, 15 January 2019, Pages 338–342 https://doi.org/10.1093/cid/ciy559

Pathogen	Risk Factors	Empiric Regimen
G+ve	*S. aureus* (general, IDU), Coag-neg staph (CoNS) (hardware), GBS (DM)	Cloxacillin/Cefazolin Vancomycin – MRSA or CoNS
G-ve	GN aerobic bacilli (IDU), *Pseudomonas* (IDU, DM), *Pasteurella spp.* (cat bite), *Salmonella spp.* (sickle cell)	Cephalosporin ($3^{rd}/4^{th}$), FQ
Poly-microbial	Contiguous – chronic ulcers, diabetic ulcers	PipTazo Clinda+Cipro

4. **Tx Duration**: 4-6weeks*, monitor and reassess for response (**depends on extent of surgical mgmt., wound closure, hardware, etc.)*
5. **Consult**: ID/Orthopedics/Neurosurgery/Vascular PRN
6. **Follow up**: Clinical signs/symptoms, ESR +/- CRP; follow-up imaging should be ordered only if evidence of ongoing infection (clinically, or inflam markers)
   *Have the appropriate lab work and imaging scheduled **PRIOR** to the respective clinic follow up.*

Septic Joint

Golden Rule: Monoarthritis = septic arthritis until proven otherwise. A septic joint is a **rheumatologic emergency** given risk of rapid joint erosion and disability without treatment. Presence of crystal does not r/o infection.

Differential Diagnosis of Monoarthritis:
- Infectious: bacterial, viral, fungal, osteomyelitis/osteonecrosis extending to joint
- Crystal: gout, pseudogout, hydroxyapatite, basic calcium phosphate
- Rheumatologic: seropositive, or seronegative, sarcoidosis, polymyalgia rheumatica
- Neoplastic: chondrosarcoma, osteoid osteoma, metastasis
- Unclassified: trauma, osteoarthritis, hemarthrosis, non-arthritis (bone, soft tissue)

Risk Factors:
- **22% of native joint infections occur in the absence of identifiable RF**
- Major: RA (and any abnormal joint structure); prior joint surgery, endocarditis, IDU, immunocompromised, adv age, chronic renal failure, DM
- Minor: Skin and soft tissue disease, other joint disease, joint injection, sys disease (CvD, cancer, liver disease, sickle cell, EtOH), hypogammaglobulinemia, low SES

Complications: osteomyelitis (30%), permanent joint damage, sepsis, endocarditis

History:
(1) **Prosthetics**; (2) **Previous septic arthritis**: pathogen & sensitivity, and surgical Tx; (3) **Antibiotics**: did they received treatment BEFORE arthrocentesis; (4) **Other joints**: septic polyarticular arthritis is uncommon (consider disseminated gonococcal); (5) **Infectious focus (ROS)**

Investigations:
Labs: CBC+diff, CRP, ESR
Arthrocentesis (3C's) – Cell count + diff, Culture and Gram stain, Crystal
* WBC<50k does not r/o septic arthritis,

Imaging: X-ray, CT scan/MRI if prolonged symptoms

Interpretation	WBC ($x10^6$cells/L)	PMN (%)
N	<200	<25
Non-Inflam	200-2000	<25
Inflam	2000-50000	25-50
Septic	>50000*	>90

Joint Aspirations:

1. Diagnostic and sometimes therapeutic reasons
2. Absolute contraindication is infection overlying site of injection
3. Relative contraindications include significant hemostasis defects and bacteremia

JAMA. 2007. 297(13):1478-88.			
Aspirate		PLR	NLR
WBC ($x10^9$cells/L)	>25	2.9	0.32
	>50	**7.7**	0.42
	>100	**28**	0.71
PMN%	>90%	3.4	0.34
	<90%	0.34	
Serum			
ESR (mm/hr)	>30	1.3	**0.17**
CRP (mg/L)	>100	1.6	0.44

Mx:

1. Empiric antibiotics: Cefazolin 2g IV q8h (+/- vancomycin 15-20mg/kg IV q12h)
2. Urgent Ortho consult for arthroscopic irrigation and debridement.

Urinary Tract Infection

Definitions:

- **Asymptomatic Bacteriuria:** positive urine culture but no UTI symptoms/signs. Common in elderly patients (F>M), indwelling catheter (100%)
- **Uncomplicated UTI:** Acute uncomplicated**;** Acute non-obstructive pyelonephritis; Female, without genitourinary abnormalities
- **Complicated UTI**: Male UTI; Genitourinary functional, or anatomical abnormality – PCKD, nephrolithiasis, neurogenic bladder, diabetes (poorly controlled), immunosuppression (including renal transplant), pregnancy, indwelling urinary catheter, recent urinary tract instrumentation
- **Pyelonephritis:** infection of the renal parenchyma
- **Pyuria:** increased PMN leukocytes in urine – **KEY presence of pyuria is not specific to infection**. Absence of pyuria rules out UTI

Investigations:

- **Blood tests:** CBC and diff, lytes, Cr, **lactate, blood cultures** (if pyelo/sepsis suspected)
- **Urine tests:** urinalysis; urine C+S > 10^5 cfu/ml in clean catch, midstream sample suggests UTI
- **Imaging:** In male pts, consider PVR to rule out retention +/- abdominal US; non-contrast CT abdo for stones; contrast CT abdo for pyelo/abscess

Mx:

1. **Asymptomatic Bacteriuria:** screening (urine culture) and treatment indicated in the following groups ONLY – pregnant women, patients undergoing endoscopic urologic procedures (with risk of mucosal trauma) Minimal evidence for/against screen/tx within 1 month renal transplant, neutropenic patients, Patients with chronic indwelling catheters, spinal cord injury managed with intermittent catheterized, elderly in nursing homes, diabetic female should not be treated in the absence of signs/symptoms of UTI. **Unwarranted treatment will result in increased risk of infection with resistant organisms.**

2. Acute Uncomplicated Cystitis (females)

Antibiotic	Dose	Notes:
Nitrofurantoin	100mg PO BID x 5d	Cystitis in females only
TMP-SMX	1 DS tab PO BID x 3d	
Ciprofloxacin	250mg PO BID x 3d	NOT first line
Levofloxacin	250mg PO OD x 3d	

3. Acute Pyelonephritis

Antibiotic	Dose	Notes:
Ceftriaxone	1g IV OD	Reasonable GN enteric coverage
Levofloxacin	750mg IV/PO OD	Bioavailability is ~100% Use PO unless otherwise indicated
Ciprofloxacin	400mg IV or 500mg PO BID	

*Duration of Tx: typically 48hrs of parenteral therapy, or until afebrile then switch to PO therapy. 5-7 d of a fluoroquinolone suffice, total of 10-14 d for beta-lactams.

Specific Considerations:

a) Catheter-associated bacteriuria. Common organisms: E.coli and Enterococci; responsible for 80% of sepsis w/ urinary source. **Treatment:** replace catheter when appropriate, antibiotics for symptomatic or complicated bacteriuria.

b) Pregnancy and UTI: Tx regardless of symptom**. Treatment:** amoxicillin or nitrofurantonin ($1^{st}/2^{nd}$ trimester only) x 4-7 days.

c) Male UTI: Typically treat for longer duration than uncomplicated UTI in female. Consideration of prostatitis, no good evidence re: appropriate regimen. If can tolerate PO – Septra as above, if IV consider fluoroquinolone or CTX.

PEARLS:

- Sample contamination: often the sample is taken from Foley.
- Culture correlation: correlate with symptoms, signs, and/or urine analysis/dip.
- Source control: remove the nidus of infection.
- Prevention: don't Foley everyone – few indications; use urinal if possible; measure daily weight instead of ins/outs.

Nephrology

Authors: Dr. Sunchit Madan & Dr. Caitlyn Vlasschaert, Dr. Ian White *Updated May 2024*

Acute Kidney Injury

AKI is defined as any of the following:

1. Serum creatinine (SCr) increase by ≥27 μmol/l) within 48 hours; or
2. Increase in SCr to ≥1.5 times baseline, known/thought to have occurred within the past 7 days; or
3. Urine volume <0.5 ml/kg/h for 6 hours

AKI can be staged by severity based on degree of rise in SCr, or by the degree and duration of reduction in urine output.

	Etiology	Basic Work up
Pre-renal	-Absolute ↓ in circulatory vol. – blood, GI, urine -Effective ↓ in circulatory vol: CHF, cirrhosis, nephrotic syndrome, sepsis -Renal artery Stenosis, dysplasia, emboli	• Urine: hyaline casts, urine Na^+<20, $FENa^+$<1%, FEUrea<35% (if on diuretics within 3 days) • Serum Urea:Cr >20:1 (Convert Cr from μmol/L to mmol/L) • Renal artery stenosis: MRA
Intra-renal	**Glomerular:**	**Glomerular:** • Urine: RBC casts, RBC acanthocytes

	-Nephrotic: > 3.5g protein/24h, edema, hypoalbuminemia, hyperlipidemia, hyperCoag -Nephritic: AKI, hypertension, hematuria and/or proteinuria **Interstitial:** • Acute: AIN drug hypersensitivity (penicillin, cephalosporin, PPI), pyelonephritis • Chronic: fibrosis, sarcoid, metals **Tubular:** • Acute (ATN): Ischemic; Nephrotoxic: Exogenous (Contrast IV, acyclovir, aminoglycoside, ethylene glycol); Endogenous (myoglobulin – rhabdo; hemoglobulin – massive hemolysis; light chain – MM, urate –TLS) • Chronic: PCKD **Vascular:** • Acute: HUS/TTP; vasculitis Chronic: HTN, DM	• Blood: C3, C4, ANA, ENA, RF, ANCA, cryoglobulins, anti-GBM, ASO titer, HBV, HCV, HIV (these are expensive tests and decisions to order specific ones are based on patient's history and exam) **Interstitial:** • Urine: WBC cast; eosinophil (low utility) **Tubular:** • ATN: granular casts, urine Na^+>20, $FENa^+$>1%, FEUrea>35% (if on diuretics within 3 days) • ↑CK; hemolysis work up; SPEP&UPEP, immunoglobulins; uric acid **Vascular:** Blood smear (schistocytes)
Post-renal	**Renal pelvis to urethral meatus:** • Intraluminal**:** stone, BPH, bladder cancer Extraluminal**:** abd/pelv mass, lymphadenopathy, AAA	• Urine: RBC, WBC, crystal or bland, uric acid (tumour lysis syndrome) • Foley Renal US

Approach:

1. Is there any acute indication for dialysis?

- **A**cid/base disturbance: severe metabolic acidemia refractory to med management
  - Consider giving IV Sodium Bicarbonate to temporarily correct metabolic acidosis whilst treating the cause.
- **E**lectrolytes disturbance (hyperK^+) refractory to medical management
  - Kaliuresis is key for sodium excretion, consider Lasix +/- other diuretics if patient produces urine. Intracellular shifting of potassium using insulin and IV dextrose, and/or salbutamol (*see Hyperkalemia section*) is a temporary measure to reduce serum potassium. Labs should be re-assessed regularly and nephrology should be involved early.
  - Consider other causes of hyperK which may be present simultaneously with AKI, and may change management (ex. rhabdomyolysis, TLS)
- **I**ntoxications: methanol, ethylene glycol, salicylate, lithium
- **O**verload of volume (pulmonary edema) refractory to medical management
- **U**remia: encephalopathy (confusion, asterixis), pericarditis (chest pain, rub)

2. **Cr Trend?** (Check outpatient blood work): (a) Baseline Cr, (b) Acute vs Chronic vs Acute on Chronic, (c) Progression Rate.

3. **Post-Renal? U/S and foley catheter** – rapid & definitive. Definitive Tx requires Urology/IVR intervention. No urine drainage post-foley insertion does not mean that post-renal AKI is ruled out. If 2 functional kidneys, extremely unlikely to be nephrolithiasis.

4. **Pre-Renal? Hx+Exam usually suffice**. Renal artery etiologies are less apparent.
5. **Renal?** Not obvious. Start with **Urine dip + microscopy.** The most devastating acute causes are **RPGN, HUS/TTP** – rare, therefore a high index of suspicion is required.

Approach to AKI in the renal transplant patient:

Kidney transplant recipients are a special population, as the differential diagnosis for an AKI in these patients is slightly more complicated. Have a low threshold to consult Nephrology for this patient group. A very brief summary of the causes of AKI in this group is as follows:

1. Rejection (ALWAYS SUSPECT THIS!) – may have proteinuria, hematuria, WBCs in urine
2. Pre-renal or ATN – just like native kidneys
3. Calcineurin inhibitor toxicity (ciclosporin, tacrolimus) – need to get trough levels
4. Infections (bacterial, CMV, EBV, BK virus) – knowing the CMV and EBV status of the donor can be helpful.
5. Recurrence of primary disease
6. Renal artery/vein thrombosis or hydronephrosis – get renal US of transplant kidney with doppler. Usually an early post-operative complication

Chronic Kidney Disease

Etiology:

- The leading causes of CKD are:
  1. Diabetes – by far the commonest, with about 40% of CKD patients having this as cause
  2. HTN
  3. Glomerulonephritis – IgA Nephropathy is the commonest type of GN (*remember - not all GNs present as AKI*)
  4. Cystic Kidney Disease

There are many others - beyond the scope of this book - but the above are most common you will encounter.

Determining Cause:

History is much more valuable than any investigation you can order:

- Hematuria, rash, joint swelling, frothy urine, signs of obstruction, Rheumatological conditions such as SLE, Sjogren's, or scleroderma
- Decreased PO intake, illness/infectious hx
- History of diabetes and determine severity
- Kidney stones, abdominal malignancies, bladder outlet obstruction, Polycystic Kidneys, dialysis dependence in family
- NSAID use (naming generic and brand names), ACE-I/ARB, diuretics, other nephrotoxic meds.

Investigations:

- First test should be Urinalysis + urine microscopy – protein, blood
- Urine ACR – to quantify degree of proteinuria
- Kidney ultrasound – size of kidneys, obstruction.
- Serum and urine electrophoresis – looking for monoclonal proteins
- GN work-up if indicated – ANA, ANCA, hepatitis serology, anti-DS DNA
- Kidney biopsy (in consultation with nephrology)

Prevention of progression

1. Blood pressure control: ACE-I/ARB first line
2. SGLT2: 1st line for all CKD GFR > 20 even in absence of diabetes.
3. Proteinuria: ACE-I/ARB 1st line for proteinuria
4. Dyslipidemia: Statin +/- ezetimibe
5. Optimize glycemic control: (GLP-1, SGLT2, insulin, metformin, etc)
6. Smoking cessation
7. Prevention of acute on chronic kidney injury: *Hold SICK day meds when ill*

CKD Complications:

Electrolytes: Hyperkalemia

- Keep K < 5: Renal diet (low potassium), Diuretics (non potassium sparing). Unclear benefit for Kayexelate®

Metabolic Acidosis:

- Sodium bicarbonate 500mg -1g PO BID-TID. Sodium load will complicate BP and volume control Mx.
- Target normal bicarbonate level

Anemia

- Reticulocyte count, ferritin, transferrin saturation, Vitamin B12, and folate
- In non-dialysis patients If anemic with TSAT <30%, and ferritin <500 - given oral/parenteral iron first.
- If Hb remains <90g/L consider adding erythropoietin stimulating agent (ESA), use with caution in those with hx active malignancy, stroke, and VTE
- Target Hb should be 90-115g/L. Targeting normal Hb can be harmful.

Bone Disease $\downarrow Ca^{+2}$, $\uparrow PO_4^{-3}$, $\uparrow$PTH ---> vascular calcification. Occurs eGFR < 60.

Hyperphosphatemia

- Strict renal/dialysis diet
- PO_4^{-3} Binders: binds to ingested PO_4^{-3} and prevents absorption. Includes Ca^{+2} carbonate 500mg (TUMS) PO w/ meals: may result in $\uparrow Ca^{+2}$If refractory $\uparrow PO_4^{-3}$, or in the context of $\uparrow Ca^{+2}$ use sevelemer or lanthanum

Hypocalcemia: Due to decreased 1-alpha hydroxylase activity

- Ca^{+2} binders + Vitamin D. More problematic if $\uparrow Ca^{+2}$ is secondary to calcium-based PO_4^{-3} binder

2º hyperparathyroidism: Due to decreased Ca^{+2}

- Suppress with VitD though may result in $\uparrow PO_4^{-3} + \uparrow Ca^{+2}$. If refractory, consider calcitriol followed by calcimimetic ex. cinacalcet.
- Target PTH 2-9x ULN in dialysis patients.

Fluid overload:

- Diuretics, will require escalating doses as renal function ↓
- If ESRD, may require dialysis

Cardiovascular:

- HTN: Optimize BP to target <130/80. ACEi/ARB can be used if renal fxn is followed closely. As CKD advances, fluid retention is 1º cause of HTN.
- Dyslipidemia: Statin +/- ezetimibe

Renal Replacement Therapy Related Complications

Common Complications:

Hypotension: usually seen in those initiating HD, titrating in dry weight.

- Look for ischemic injury ie chest pain, dyspnea, diaphoresis. Can occur when taking off too much fluid

Hemodialysis Specific:

Line infection: suspect when septic, tender along HD line, discharge at site
Bleed at insertion site: check coagulation, apply pressure, contact Nephro.

Peritoneal Dialysis Specific

Peritonitis: Painful but not surgical abdomen-like; cloudy fluid (ask pt). **Diagnostic criteria:** WBC>100x10^6 cells/L (after at least 2hrs) + >50%PMN. **Management:** Call dialysis unit for nursing staff to drain effluent for analysis (Cell count, Diff., C+S) and give IP antibiotics: vancomycin 1g dwell + Ceftazidime 1g dwell. If fungal peritonitis (usually WBC>200x10^6 cells/L), contact Nephro ASAP as PD catheter removal is usually warranted.
Exit/Tunnel Site Infection: purulence, inflammation. **Management:** C+S. Antibiotic to cover G+ve>G-ve.

AV Fistula Specific:

Rupture – consult VascSx ASAP: apply pressure or clamp to halt hemorrhage.

Approach to Fluids

***Dextrose Calory:** 4 kcal/gram, so a 5% glucose solution provides 0.2 kcal/mL.

	Na	K	Lactate	Dextrose (g/100mL)	Tonicity	Indication
	(mmol/L)					
0.9% NaCl	154	0	0	0	"Iso"	Vol. expand
Ringer's Lactate	130	3	28	0	"Iso"	Vol. expand
5% Dextrose	0	0	0	5.0	"Iso"/Hypo	↑[Na^+]
Isotonic Bicarb (3 amps + 850ml D5W)	130	0	0	4.25	"Iso"	NAGMA Uremic acidosis
0.45% NaCl	77	0	0	0	Hypo	Maintenance fluid, ↑[Na^+]
3.3% Dextrose + 0.3% saline	45	0	0	3.3	Hypo	Maintenance fluid
10% Dextrose	0	0	0	10.0	Hyper	Persistent ↓glucose
20% Dextrose	0	0	0	20.0	Hyper	
0.45% NaCl + D5W	77	0	0	5.0	Hyper	Maintenance fluid
NS + D5W	154	0	0	5.0	Hyper	Vol. expand + ↓glucose
3% saline	513	0	0	0	Hyper	Symptomatic ↓[Na^+]

****Grams of salt in NS:** 9g NaCl is in 1L NS – exceeds recommended daily salt intake

Colloids: BLOOD PRODUCTS (Obtain Consent): Expensive and limited evidence

Production (Formulation)	Infusion Rate	Indications	Notes
Albumin 5% (25g albumin in 500mL vial)	5-10mL/min max	Consider if 3rd spacing and hypotensive	Not very different from NS

Albumin 25% (25g albumin in 100mL vial)	2-3mL/min max	Paracentesis >5L; Hepatorenal syndrome; SBP	Increases IV oncotic pressure and drawing 3rd spaced fluids

Fluid Tips:

1. Use a balanced crystalloid for resuscitation, preferably Ringers Lactate.
2. RL typically 1st line EXCEPT: Hypercalcemia, metformin-associated lactic acidosis, Elevated ICP (slightly hypotonic). RL okay to use in hyperkalemia.
3. Albumin only indicated in SBP, HRS, and after large volume paracentesis.

Approach to Acid and Base

Interpretation Steps:

1. Acid/Base? Acidemia (pH<7.35) vs Alkalemia (pH>7.45)

2. ABG #s make sense?

- Calculate $[H^+] = 24 \times (P_aCO_2)/HCO_3$ with the values obtained from the ABG.
- If there are major discrepancies, ? sample error (not stored in ice or processed late)

3. Dominant process? Acidosis vs Alkalosis (See Table 2)

4. Compensation appropriate? Any other concurrent acid/base disorders.

Expected Compensation for Respiratory Acid/Base Disorder		
	Acute	Chronic
RespAc: q10mmHg ↑CO_2, ↑HCO_3^- by	1	3
RespAlk: q10mmHg ↓CO_2, ↑HCO_3^- by	2	5

Expected Compensation for Metabolic Acid/Base Disorder	
MetAc: q1mmol/L ↓HCO_3, ↓CO_2 by	1
MetAlk: q1mmol/L ↑HCO_3^-, ↑CO_2 by	0.7

5. Elucidate the **Etiology** of each acid/base disorder(s) discovered

Metabolic Acidosis

Calculate Anion Gap = $[Na^+] - [Cl^-] - [HCO_3^-]$. Normal AG: 12±2. CORRECT for ↓ albumin by adding: for every ↓10g/L albumin, add 2 to the calculated AG.

(a) Anion Gap MetAc (AGMA): AG>12 → Dangerous Etiologies

- Calculate ΔΔ ratio = (Calc.AG – ꭤAG)/(ꭤ$HCO3^-$ - measured $HCO3^-$)
- Interpret: ΔΔ=1-2: pure MetAc; ΔΔ>2: MetAc + MAlk; ΔΔ<1: MetAc + NAGMA
- Is CO_2 compensation appropriate: whether concomitant RespAc present
- Causes: KULT – Ketones (DKA), Urea (uremia), Lactate and Toxins. Ketones (urine dip+β-hydroxybutyrate), urea, lactate "T": ASA, Toxic alcohols – Methanol, Ethylene Glycol, Isopropyl alcohol.
- Calculate Serum Osmol gap = (Measured serum Osmol) – (Calculated serum Osmol); Calculated serum Osmol = $2[Na^+]$ + [glucose] + [urea] + [EtOH]; "Normal Osmol gap" <10; Accounting for presence of toxic alcohols)

b) Non-Anion Gap MetAc (NAGMA): AG<12. The etiologies are subacute/chronic. Common DDx on a day to day basis: Normal saline; Diarrhea; RTA; Renal Failure

- Urine AG = (U_{Na} + U_K) – U_{Cl} (Can't use U_{AG} if hypovol. & AGMA present)
- Interpret: -ve UAG: GI loss, RTA II; +ve UAG: RTA I, IV, renal failure

Respiratory Acidosis: Consider the causes by mechanisms:

1) Hypoventilation:
(a) ↓LOC (most common); (b) Airway obstruction; (c) Chest wall restriction; (d) Nerve/ Muscle weakness
2) increased dead space (e.g PE)
3) Intrapulmonary shunt: e.g. pulmonary edema

Hypernatremia

Approach

1. **Severely Symptomatic** (i.e., seizure, coma): **Treat STAT** with D5W.
2. **Acute vs Chronic**? Most often chronic (developed over >48 hours). If symptomatic, suspect acute.
3. **Cause**? History usually sufficient to establish cause. Order urine osmolality.

Common Causes

1. ↓Free H_2O intake (dehydration): ↓ access (elderly, confusion, mobility issue), ↓ retention (N&V)
2. Free H_2O loss: Renal (diuresis, diabetes insipidus); Extra-renal (diarrhea, laxatives, burns)
3. Excess Na^+ intake: Exogenous ($NaCO_3$ given during resus); Endogenous (1^o hyperaldost., Cushing)

Three Most Common Causes and Distinguishing Features:

Condition	Urine Output	Urine Osmol	Notes
Dehydration	↓	↑	↓ access to free H_2O or free H_2O loss
Diabetes Insipidus	↑↑	↓↓ (<100)	Suspect with polyuria (>3L/day). **Central** (pituitary Sx – responds to DDAVP) *vs.* **Nephrogenic** (due to intrinsic kidney dz, hypoK^+, hyperCa^{2+}, Li^+).
Diuresis	↑	↑	**hyperglycemia** in DKA/HHS, **mannitol** used to Tx ↑ICP, **loop diuretics**

Management

1. **Correction Goal:** can calculate *free H_2O deficit* & replace this by ~50% per day
   Free H_2O Deficit (L) = 0.6×BW(kg) × [1 – (Serum [Na^+] /140mmol/L)]
   Practically speaking, we usually aim to decrease [Na^+] by 10 mEq/L/day.
2. **Free H_2O Replacement:**
   - Enteral: po is usually sufficient for mild ↑[Na^+]. Consider NG route if more required.
     (Sample NG replacement regimen: 300mL NG q4-6h, or 50mL/hr NG continuous infusion)
   - Parenteral: if hypovolemic, isotonic to ½NS (or D5W if ↑↑↑[Na^+]). if euvolemic, D5W.
3. **Monitor:** [Na^+] q6h during correction.
4. **Consult Nephro/Endo:** if ↑ [Na^+] and/or polyruia are resistant to H_2O replacement
   May require specific Tx for ***Diabetes Insipidus***:
   i. **Central:** DDAVP* (if urine output >300mL/hr and urine Osmol <200).
      *Risk of quick drop in [Na^+].
   ii. **Nephrogenic:** Discontinue offending meds. Thiazide diuretics may help.

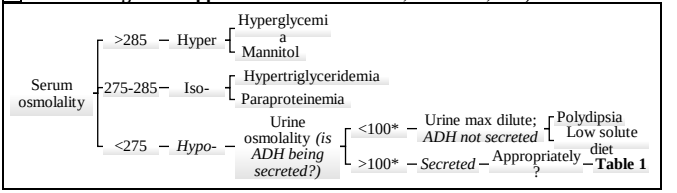
Hyponatremia

Approach:

1. **Severely Symptomatic** (i.e., seizure, coma): **Treat STAT** with 3% saline.

2. **Acute vs Chronic?** Most often chronic (developed over >48 hours).

3. **Cause – Diagnostic Approach:** Order serum osm, urine osm, & 3) urine Na



* in patients with CKD, a higher cutoff (~200) may be used (due to impaired urine diluting ability)

Table 1: Is ADH being secreted ***appropriately***?

	Yes		No
Urine [Na^+]	<20		>40
Vol Exam	Hypovolemic	Hypervolemic	Euvolemic
Cause	Hypovolemia	Edema-forming states (CHF, cirrhosis, nephrotic syndrome)	Hypothyroidism Glucocorticoid deficiency **SIADH**: assoc. w/ malignancies, CNS or lung disorders, medications (*diagnosis of exclusion)*
NS challenge	[Na^+]: Rapidly ↑	[Na^+]: no Δ or further ↓	

Management

1. If seizure or coma: 3% saline 100mL IV over 10min ≈ ↑[Na^+] by ~3mmol/L. Repeat bolus × 2 prn to ↑[Na^+] by ~5mmol/L. Once stable, 3% saline at 1 ml/kg/hr.

2. For stable patients, treat hyponatremia according to suspected origin:

- **Hypovolemic hyponatremia** (most common): give **isotonic** fluids (RL, NS)
  Rationale: Volume expansion halts ADH secretion; Once ADH off, [Na^+] will ↑rapidly 2^o aquauresis
- **Hypervolemic hyponatremia**: water restriction, loop diuretics (caution: not thiazide)
  in extreme cases, ADH receptor antagonists (vaptans) may be used, but must be monitored closely
- **Euvolemic hyponatremia** (most likely to actually be hypo- or hypervolemic):
  if suspected SIADH, trial water restriction
  if suspected other endocrine cause, Tx underlying cause

3. Correction Rate:

- If severe neurologic symptoms, ↑ by 5 mEq/L then rapid normalization
- If acute (<48 hours): rapid normalization. e.g., 3% saline at 1 ml/kg/hr
- **If chronic**, goal: 4-8 mEq/L per 24 hrs (max: 8 mEq/L per 24 hrs)
  Closely monitor serum [Na^+] q2-4h and urine output

* above 8-12 mEq/L in 24 hrs (**overcorrection**), risk of central pontine demyelinosis
symptoms: UMN findings, pseudobulbar palsy, spastic quadriplegia, ophthalmoparesis, locked-in syndrome; presentation may be delayed by > 1 week
If overcorrection: Stop current Tx. Re-lower [Na^+] with D5W IV ≥200mL/hr ± DDAVP.

Hypokalemia

Approach:

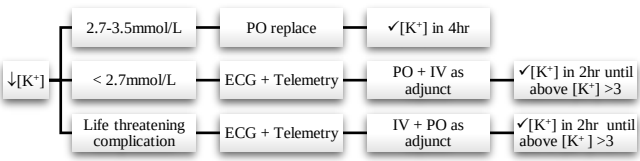
1. **Emergency symptoms?** Obtain ECG to detect and **treat STAT**.
   - A. Arrhythmias: VT/VF, atrial tachycardia, sinus bradycardia, AV blocks
     other ECG changes: prolonged QTc, ST depression, ↓ T wave/↑ U wave amplitude
   - B. Muscle weakness: generalized +/- GI, respiratory muscle paralysis

2. **Cause – Diagnostic Approach:**

	Source	Etiology	Elicit Clues From:
1	GI loss	Diarrhea, vomiting, SPSS (Kayexelate®)	Hx, Meds
2	K^+ shifting into cells	Na^+/K^+-ATPase pump activation: • **β-agonist** (e.g., salbutamol) • **insulin** (e.g., refeeding syndrome, exogenous insulin administration)	Hx, Meds
		Alkalosis (respiratory or metabolic) *also causes renal wasting of K^+*	Exam (resp rate), VBG **usually transient hypoK**
		↑Blood cell production (↑ uptake)	CBC, retic count
		Hypokalemic periodic paralysis syndrome Thyrotoxic periodic paralysis syndrome	Exam (objective weakness/paralysis) Check thyroid panel
3	Renal K^+ loss	Diuretics (**loop & thiazide diuretics**)	Hx, Exam, Meds
		Hyperaldosteronism	↑ urine [K^+]
		Hypomagnesemia	Serum ↓[Mg^{+2}]
		Loss of bicarbonate (e.g. compensation for metabolic alkalosis, Type II RTA)	Serum ↓[HCO_3^-] +/- NAGMA

Management

1. Treat the underlying cause
2. Replace K^+:
   - A. Approximate Rule: For every 10mmol KCl given, serum [K^+] ↑ by ~0.1mmol/L
   - B. ↓[Mg^{+2}] renders K^+ replacement less efficacious – replace Mg^{+2} concomitantly



Oral: 40-100 mmol/day in 2 doses to minimize GI side effects.

Formulations	Dose	Notes
KCl slow release (Slow- K®): 8mmol K^+/tablet	2 tabs PO QD	For maintenance therapy (e.g., with chronic diuretics)
KCl sln. (K-Elixir®): 1.33mmol K^+/mL	40 mEq PO × 1	Tastes horrible.
K citrate (K-lyte®): 25mmol K^+/tablet	25-50 mEq PO × 1	

IV

Access	Max Dose	Max Infusion Rate	Notes
Peripheral (regular IV) or Central Line	KCl 10mmol in 100mL sterile H_2O ("mini-bag")	10 mmol/hr over 1hr	**Do not exceed:** periph: 10 mEq/hr central: 20 mEq/hr → above this, risk of arrhythmias or phlebitis.
	NS 1L with KCl 40mmol	Periph: over 4hrs Central: over 2 hrs	
Central Only	KCl 40mmol in 100mL (sterile H_2O)	Give over 2hrs ***For life threatening*** ↓ $[K^+]$, ↑rate to 40mmol/hr.	For aggressive replacement: monitor on **telemetry**.

Hyperkalemia

Approach:

1. Emergency sx? Re-draw $[K^+]$, obtain ECG and **treat STAT if any ECG changes**.

- ECG changes: peaked T waves (taller than QRS), flat P waves widening of PR interval and QRS complex → sine wave → VF
- Muscle weakness: generalized +/- GI, respiratory muscle paralysis

2. Cause – Diagnostic Approach:

First, confirm that it is ***true*** hyperkalemia (ask lab if sample hemolyzed, re-draw lytes).
Pseudohyperkalemia is a phlebotomy artifact:

- **Cell lysis**: fragile cells (CLL; consider ABG *(larger bore, slower flow = less shearing).*
- **Technical problem** ("hard poke", sample taken from arm with IV K^+ running)

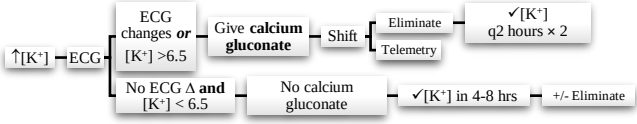
Causes of true hyperkalemia:

	Source	Etiology	Elicit Clues From:
1	↑ K^+ intake (any route)	Supplements (IV, Oral)	Meds
2	K^+ shifting out of cells	↓ Na^+/K^+-ATPase pump activity: • Insulin deficiency (**DKA**) • b-blocker toxicity • digoxin toxicity	Hx, Meds
		Metabolic acidosis (NAGMA)	Serum lytes, VBG
		Cell lysis: Rhabdomyolysis, TLS	Serum CK, uric acid
3	↓ excretion (GI or kidney)	Decreased GFR – in **AKI, CKD, ESRD** (can be exacerbated by **NSAIDs**)	Serum Cr, urea

		↓ Aldosterone activity: • **MRAs** (spironolactone, eplerenone), **ACEi/ARBs** • Hypoaldosteronism (e.g., in **adrenal insufficiency**) • **Heparin** (inhibits aldost. synthase)	Hx, Meds
		Inhibition of ENaC (in collecting duct): • **TMP-SMX** (Septra®) • Amiloride • Calcineurin inhibitors (tacrolimus, cyclosporine)	Meds

Management

1. Approach:



2. Treatments:

IMMEDIATE STEPS (stabilize patient)
1. **Calcium gluconate** 1g (1 amp) IV over 2 mins • Indicated **ONLY** when there is ECGΔ or [K^+] >6.5mmol/L • Role: stabilizes the cardiac membrane • Onset <1 min, peaks immediately, lasts ~60min.
2. **Regular Insulin** 10 Units IV (a.k.a. "shifting") • Give 1amp D50W **before** giving insulin if hypo- or euglycemic. • Role: Shifts K^+ into cells • Onset <10 min, peaks ~60min, lasts ~4hrs.
3. **Salbutamol** (Ventolin®) 10 mg* neb q4h PRN • Onset <5 min, peaks ~90min, lasts ~3hrs. • Role: Shifts K^+ into cells • Note: this is a 2-4x higher dose than what you give for asthma exacerbation, only suitable for patients who can tolerate side effects (tachycardia)
LATER STEPS (permanently eliminate extra potassium)
4. **Diuretics** (e.g., furosemide): for patients who produce urine **AND/OR** **Fluids if hypovolemic:** Crystalloid fluids, RL okay to use. If renal failure + hyperkalemia can give bolus of isotonic bicarbonate.
5. **Hemodialysis:** for refractory hyper[K^+] or anuric patients
6. **Potassium binders** (e.g., SPSS (Kayexelate®), patiromer): GI elimination • use SPSS with caution in patients with recent kidney transplant (increased risk of colonic necrosis in this patient population) • monitor all lytes + extended lytes after initiation • Takes > 24 hours to work, limited clinical utility acutely

Hypercalcemia

Approach:

1. What's the difference between total (tCa) and ionized (iCa^{2+}) calcium?

A. **tCa:** measures free Ca^{2+} + Ca bound to albumin (inert)
- *clinically, we care about the **free** (ionized) calcium portion*
- Free [Ca] underestimated when Alb ↓, overestimated when Alb ↑ (pseudohypercalcemia)
- corrected [tCa] = measured [tCa] + [(40-serum albumin) x 0.02]

B. **iCa^{2+}:** directly measures free Ca^{2+} only, but more expensive to run ($$$)
- must be kept on ice during transport for accurate analysis

→ *Order one or the other; not both!*

2. Symptoms: "*Stones, Bones, Groans, Thrones, Psychiatric overtones"*
- Commonly: weakness, confusion, abdominal pain/constipation, polyuria, polydipsia

Acute Emergencies:

Complications	Features to Elicit	Action
Stupor, Coma	Δ in LOC, Sz	Get help; consider intubation, **Tx ASAP**
Arrhythmia (*uncommon*)	ECG Δ: bradycardia or heart block	Get help; ACLS; **Tx ASAP**

3. Cause – Diagnostic Approach:

Initial bloodwork: total Ca + albumin (or ionized Ca^{2+}), PO_4^{3-}, PTH and ALP.
After this, add Vitamin D (25-OH VitD2 & 1,25-OH VitD) and PTHrP if needed.

Disorder	Serum Ca^{2+}	Serum PO_4^{3-}	PTH	Urine Ca^{2+}
1˚ hyperparathyroidism	high	low	high	high
3˚ hyperparathyroidism	high	high	high	high
Familial hypocalciuric hypercalcemia (FHH)	high	low	high	low
Lithium	high	low	high	low
Thiazide diuretics	high	low to normal	low or high* *may unmask 1˚ hyperPTH	low
Vitamin D excess (including non-renal Vitamin D activation, as seen in lymphoma, TB, sarcoidosis & other granulomatous dz)	high	high	low	high
Malignancy-associated (↑PTHrp)	high	low	low	high
Osteolysis	high	high	low	high

Note: **2˚ hyperparathyroidism** (seen in CKD) causes high PTH but **hypo**calcemia.
(lack of Vitamin D activation by the kidneys → hypo[Ca] → overstimulation of parathyroid gland)

4. Management

1. **IV fluids**: NS 1-2L IV bolus, then 100-200mL/hr. *USE NORMAL SALINE*
   - Rationale: hypervolemia induces calciuresis
   - Monitor volume status; loop diuretics prn for pulmonary edema.
2. Specific treatments by underlying cause:
   - ***A. PTH-mediated hypercalcemia:***
     - **Parathyroidectomy** (definitive treatment)
     - **Cinacalcet –** for patients who are not surgical candidates
   - ***B. Malignancy-associated hypercalcemia:***
     - **Bisphosphonates** (e.g., pamidronate, zoledronic acid)
       - Dose: 60mg IV for tCa < 3.5, 90 mg IV for tCa > 3.5
       - Indication: acute -[Ca^{+2}] (<48hr). Be wary in renal failure.
       - [Ca^{2+}] normalizes in ~4 days, lasts ~1 mth.
     - **Calcitonin**
       - Dose: 200-400U sc q12h × 2d max
       - Beware: risk of tachyphylaxis (use small sc test dose first).
       - Modest lowering of [Ca^{2+}] within hours.
   - ***C. Granulomatous disease-associated hypercalcemia***
     - **Prednisone**
       - Dose: 20-40 mg po daily
       - [Ca^{2+}] normalizes in ~4 days
3. **Dialysis:** for refractory hypercalcemia or patients unable to tolerate Na^+ load of NS

Hypomagnesemia

Approach

1. **Emergency symptoms?** Ask for help, ABCs and give **$MgSO_4$ 2g IV over 2 mins STAT**.
   - Neurologic: tremors & delirium → seizures & coma
   - Cardiac: wide QRS → atrial and ventricular tachycardias (including Torsades)
   - Other electrolyte Δs: leads to concomitant hypoK & hypoCa → replete prn

2. **Cause – Diagnostic Approach**

	Source	Etiology – Major	Elicit Clues From:
1	GI Mg^{2+} loss	diarrhea, N&V	Hx
		acute pancreatitis	Hx, lipase
		PPI	Meds
2	Renal Mg^{2+} loss	Diuretics (**loop** and **thiazide**)	Meds
		Gitelman syndrome *(mimics chronic thiazide diuretic use)*	↓ serum [Na^+] & [K^+] ↓ urine [Ca^+] metabolic alkalosis
		Bartter syndrome *(mimics chronic loop diuretic use)*	↓ serum [Ca^+] & [K^+] ↑ urine [Ca^+] metabolic alkalosis
		Other drugs (aminoglycosides, calcineurin inhibitors, cisplatin)	Meds
		Chronic EtOH use	Hx
		Hypercalcemia	↑ serum [Ca^+]

		GI Loss	Renal wasting
Tests to help differentiate between GI and renal losses:	24hr urine Mg collection	< 10 mg	> 10-30 mg
	Fractional excretion of Mg *(analogous to FENa):* $FE_{Mg} = \frac{U_{Mg} \times S_{Cr}}{(0.7 \times S_{Mg}) \times U_{Cr}}$	$FE_{Mg} < 2\%$	$FE_{Mg} > 4\%$

3. Management

Mg Replacement

Severity	Action	Monitoring
↓$[Mg^{2+}]$ with emergency symptoms *(seizure, arrhythmia)*	**$MgSO_4$ 2g IV over 2min** then $MgSO_4$ infusion (4-8g IV over 24 hrs).	Telemetry while replacing. Check $[Mg^{2+}]$ 4hrs after infusion ✓ for hyporeflexia q1h after boluses of IV $MgSO_4$
Severe but stable	**$MgSO_4$ 2g IV** over 2-4hrs *plus* oral replacement	Check $[Mg^{2+}]$ 4hrs after infusion
Mild to Moderate	**Oral replacement**: – Mg oxide 1-2 tabs po daily/BID (400mg/tab) – $Mg(OH)_2$ 7.5-15 mL po daily/BID (80 mg/mL) ***Note***: diarrhea is a limiting side effect (Mg hydroxide is *aka* milk of magnesia)	Check $[Mg^{2+}]$ 24 hrs post-replacement

Hypophosphatemia

Approach:

1. Emergency symptoms? Ask for help, ABCs and give IV phosphate replacement* **STAT**.

- Neurologic: irritability & delirium → seizures & coma
- Muscle weakness +/- rhabdomyolysis and hemolysis (cells unable to form ATP)

***Note:** Dangerous side effect profile for IV phosphate as it binds calcium:

- rapid drop in serum Ca^{2+} → arrhythmias and other sequelae of hypocalcemia
- $PO_4^{3-} + Ca^{2+}$ = precipitation → kidney stones & kidney failure

***Safe doses of IV phosphate:* 15 mmol over 2 hours, 30 mmol over 6 hours**

2. Cause – Diagnostic Approach

	Source	Etiology	Elicit Clues From:
1	↓GI absorption	Anorexia, chronic diarrhea	Hx
		Antacid use (binds PO_4^{3-})	Meds
2	PO_4^{3-} shifting into cells	Na^+/K^+-ATPase pump activation: • **β-agonist** (e.g., salbutamol) • **insulin** (e.g., **refeeding syndrome**, exogenous insulin administration)	Hx, Meds
		Respiratory alkalosis	Exam (RR), VBG
		Hungry bone syndrome *(Immediately post-parathyroidectomy. Drop in PTH = rebound PO_4^{3-} & Ca^{2+} uptake into bone)*	Surgical History

3	Renal PO_4^{3-} losses	**1° hyperparathyroidism** *(PTH stimulates PO_4^{3-} & Ca^{2+} leeching from bone, then PO_4^{3-} renal loss & Ca^{2+} renal reabsorption*. Result: hypo[PO_4^{3-}] & hyper[Ca^{2+}])	Serum Ca^{2+}, PTH
		Deficiency of active Vitamin D (as in **2° hyperparathyroidism**)	Serum Ca^{2+}, PTH
		Proximal tubule dysfunction (as in **Fanconi syndrome** and some genetic disorders)	Urine PO_4^{3-}, glucose, amino acids Lytes, VBG

3. Management

PO_4^{3-} Replacement

Severity	Action	Monitoring
↓[PO_4^{3-}] with emergency symptoms *(e.g., seizure)*	30 mmol IV phosphate (*choose*: as KPhos ***or*** NaPhos) over 6 hrs *plus* oral replacement	Telemetry Check [PO_4^{3-}] 4hrs after infusion
Severe but stable	15 mmol IV phosphate (*choose*: as KPhos ***or*** NaPhos) over 2-3 hrs *plus* oral replacement	Check [PO_4^{3-}] 4hrs after infusion
Mild to Moderate	**Oral replacement**: – Sodium phosphate solution 4mL PO BID (4mmol/mL) ***Note***: diarrhea is a limiting side effect	Check [Mg^{2+}] 24 hrs post-replacement

Neurology

Authors: Dr. Lauren Mak, Dr. Pardis Balari *Updated May 2024*

Status Epilepticus (SE), Seizure (Sz)

Status Epilepticus: Continuous or repetitive Sz activity for >30min. In practice, intervene if Sz lasts >5min, or incomplete recovery of consciousness between ≥2 discreet Sz.

Epidemiology: Yearly incidence of 1.3-74/100,000. High in <1yr and >60yrs.

Ictal Manifestations: If possible, record a video of the event – helps with diagnosis.
- Generalized Convulsive SE: highest complications, morbidity and mortality. Impaired LOC, bilateral tonic stiffening, followed by symmetric rhythmic jerking.
- Focal Motor SE: manifestation depends on epileptogenic brain region. Progression of focal jerking OR widespread unilateral jerking muscle activity ± impaired LOC
- Myoclonic SE: generalized, myoclonic jerks rhythmic/arrhythmic.

Management: Stabilize & TERMINATE the Sz ASAP.
- t1=ongoing Sz activity regarded as abnormally prolonged, unlikely to spontaneously stop, initiate tx
- t2=time after which ongoing Sz activity poses significant risk of LT complications (Epilepsia. 2015;56(10):1515)

1. Rapid Assessment & Support (ASAP)	ABCs, cardiac monitoring, O2 sats, frequent vitals. Rapid neurologic exam. Two large bore IVs. Lytes, extended lytes, glucose, LFTs, CBC, tox screen.	
2. Initial Pharmacologic Tx (within 10-20 min)	1st IV: benzodiazepine **Lorazepam** 0.1mg/kg (max 2mg/min) No IV: **Midazolam** 10mg IM (>40kg)	2nd IV: anti-seizure med **Levetiracetam** 60 mg/kg (max 4500 mg) OR **Phenytoin** 20mg/kg (25-50mg/min) OR **Fosphenytoin** 20mg PE/kg (100-150mg PE/min) No IV: can give IO
	Correct metabolic abnormalities if present.	
3. Secondary Tx	Repeat anti-seizure med OR choose other 1st line drugs not already given. Refractory SE: prepare for RSI and continuous **midazolam** or **propofol** infusion. Third-line drugs: phenobarbital, lacosamide, topiramate, clobazam	
4. Secondary Assessment (Postictal Recovery)	Repeat full neurologic exam. Head CT or MRI. LP if infectious etiology suspected.	

Investigations

- **Electrolytes:** Na, Ca, Mg
- **Metabolic:** glucose, TSH, Cr, LFTs, tox screen
- **Non-contrast CT head** (*once stable): for ALL 1st time seizure – hyperdensities (acute hemorrhage), hypodensities (ischemia, mass lesion)
- **EEG**
- **LP**: suspicious of acute infectious process, leptomeningeal cancer, chronic meningitis

Diagnosis: Clinical dx, confirmed by observation of sustained and rhythmic generalized tonic and clonic motor activity. EEG is critical to diagnose more subtle forms of SE, differentiating myoclonus SE from nonepileptic myoclonus and to exclude ongoing nonconvulsive Sz.

Etiology: In adults, usually due to structural lesion or a metabolic disturbance

Genetic	Fam Hx of Sz, epilepsy, sudden deaths. Personal Hx of Sz.
Acquired	
Metabolic	↑/↓Glucose, ↓Na, ↓Ca, ↓Mg, anoxia, hepatic encephalopathy, uremia, thyroid storm, sepsis.
Toxin/Meds	EtOH/barbiturates/benzo withdrawal. Recreational drugs. Non-compliance. Δ to anti-epileptic regimen. Drug interaction. ↓ Sz threshold (e.g. penicillin G, isoniazid, clozapine).
Vascular	Hemorrhage, stroke (*most common in older adults), ↓brain perfusion, AVM, venous thrombosis, SAH.
Cancer	Mass effect from tumour ± surrounding vasogenic edema.
Infection	Meningitis, encephalitis, cerebral abscess.
Trauma	Birth injury, head trauma, intracranial operation, intracranial device, cerebral radiation.
Autoimmune	Paraneoplastic: ANNA 1 (anti-Hu), anti-NMDA, anti-GAD.

Patient Education

- **Common Triggers**: sleep deprivation, alcohol, infection/systemic illness, certain medications.

- **Activities**: avoid activities where sudden loss of consciousness would be dangerous e.g. swimming, working at heights, etc.
- **Driving**: restrictions must be put in place, must have discussion with patient that you must report to the Ministry of Transportation of Ontario (MTO).

Stroke

Overarching Schema: (1) Is this a stroke? (2) When was patient last known to be/seen without deficits? (3) Contraindication to thrombolysis?

Initial Assessment (Stroke. 2019;50(12):e344. Epub 2019 Oct 30.)

	Action to be Taken		Note
1	ABCs and activate **Stroke Protocol**		• Call Operator: Stroke team, Radiologist will be contacted. • Two large bore IV access.
2	**RAPID** assessment	Establish onset	Patient/Collateral Hx: Was onset <4.5hrs ago? Determine eligibility for tPA vs. thrombectomy.
		Rule out mimics	↓/↑Glucose, Sz, re-emergence of previous stroke symptoms, complex migraine.
		NIHSS	Quick exam for deficit pattern and severity.
		tPA contraindication	The only absolute contraindication is cerebral hemorrhage. Elicit: recent Sx, intra-cranial cancer, INR, anticoag, antiplatelet.
3	**CT head w/o contrast**		Don't wait for the porter!
4	**Labs**		Glucose, CBC, Troponin, Lytes, PTT/INR.

Management:

1) TIA:

No known cardioembolic source
- $ABCD^2$ score <4: Start Aspirin (162-325 mg OD)
- $ABCD^2$ score ≥4: DAPT, Aspirin (162-325 mg OD) + Clopidogrel (300-600 mg loading dose then 75 mg OD) for 21 days

Already on anticoagulation or indication (e.g. AFib, mechanical heart valve)
- Anticoagulation over antiplatelet therapy

2A) Ischemic – tPA candidate (<4.5 hrs):

Hyperacute Phase

For Thrombolysis: up to 4.5hrs from onset and no C/I

Alteplase	Dynamics & Kinetics	Notes
0.9mg/kg (max: 90mg): 10% of total over 1min, then infuse rest over 1hr.	Duration: -lysis activity persists for 1hr after infusion. Clearance: hepatic, >50% cleared in 5min post-infusion.	Intracranial hemorrhage risk: 6%. ↓BP<185/110 prior to lysis.

Be wary of (i) Hemorrhagic conversion; (ii) Angioedema (acute to subacute). ASA post-tPA Day 1. Repeat CT in 24hrs to r/o reperfusion hemorrhage.

Acute Phase

Admit to Stroke Unit (proven mortality benefit) – D4ICU.
- BP Target: Usually transiently ↑(will resolve in 1-2 days on its own).
  - if sBP>220 or dBP>120 if no lysis, after -lysis: maintain BP <180/105
- Maintenance: NPO, head to 45º, glycemic control, antipyretic, NG tube, maintain hydration (↓serum viscosity), DVT prophylaxis (not given on Day 1 post-lysis)

- Investigations: telemetry, ECG, ECHO, repeat CT on day 5-7, MRI if needed

Prevention

Risk factor modification:

- Anti-platelet: ASA/clopidogrel/Aggrenox®. Ø superior. ASA 81mg QD least $.
- DAPT in the ST (21-90 days) depending on intervention and severity.
  - CAS-30d, intracranial large artery NIHSS ≤3-90d, small vessel NIHSS ≤3-21d
- LT DAPT has no benefit for stroke reduction but increases risk of bleeding.
- Carotid stenosis: anti-platelet, endarterectomy.
- AFib: anti-coagulation when "safe" (no hemorrhagic transformation).

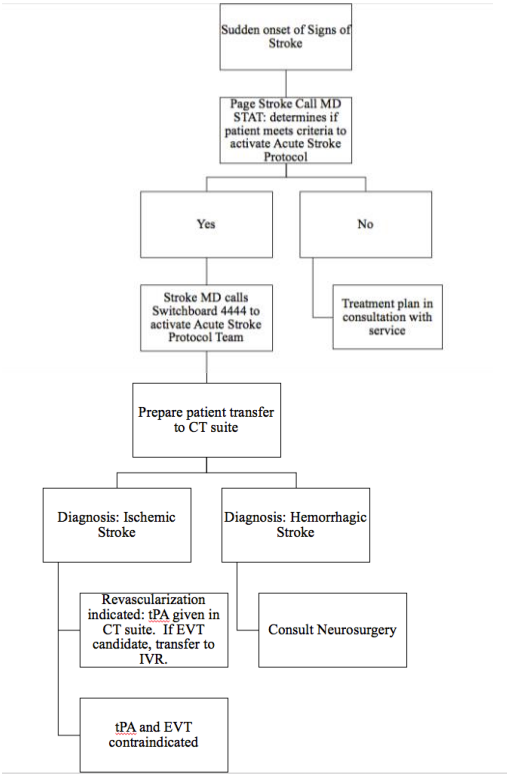
2B) Ischemic – thrombectomy candidate (4.5-24 hrs):

- Treatment can be initiated within timeframe of last known to be well
- Small infarct core and no hemorrhage, clot burden not in distal branches
- Imaging-target mismatch ratio >1.8, volume >15mL and ischemic core >70mL OR clinical-ASPECTS mismatch (NIHSS ≥10 + ASPECTS ≥6)

3) Hemorrhagic: SAH and Intracerebral Hemorrhage

- Stabilize patient. May need to transfer to D4ICU/K2ICU especially with SAH.
- Manage -ICP: elevate bed 30º, NS (Ø hypotonic soln), Ø glucocorticoids, consider – osmotic tx (IV mannitol/hypertonic saline), CSF drainage.
- Reverse any coagulopathies and D/C all anticoagulants/anti-platelets.
- BP target: sBP 140-160.
- Intermittent pneumatic compression to prevent venous thromboembolism.
- Treat hyperglycemia (7.8-10 mmol/L).
- Monitor for and treat seizures (more common in lobar hemorrhages).
- Consult Neurosurgery ASAP for surgical or IVR intervention.

KHSC Internal Activation of Acute Stroke Protocol



Headache

Low Risk	High Risk
• ≤50 yrs • Features of 1º headache (migraine, tension, cluster) • Hx of similar episodes • No pattern change • No high risk comorbidities • No neurological findings on px • No new/concerning findings on px/hx	• **S**ystemic symptoms (fever)* • **N**eoplasm hx • **N**eurologic deficit* • **O**nset sudden/abrupt* • **P**attern change • **P**ositional • **P**recipitated (sneezing/coughing, exercise) • **P**apilledema* • **P**rogressive • **P**regnancy/Puerperium • **P**ainful eye/autonomic features* • **P**ost-traumatic

	• **P**athology of immune system (HIV) • **P**ainkiller overuse

Guillain-Barré Syndrome

Pathogenesis: believed to result from an immune response preceding a triggering event (infection, immunization, surgery, trauma etc.) resulting in cross-reaction with peripheral nerve components because of molecular mimicry.

Epidemiology: 1-2/100,000 per year. All age groups affected but incidence increases ~20% with every decade. M>F.

Presentation: (Brain. 2014;137(Pt 1):33. Epub 2013 Oct 26.)
- Progressive (over ~2wks), symmetric muscle weakness with hyporeflexia/areflexia
- Most common presentation: Usually starts in legs, 90% hypo-/areflexia in affected limbs, 80% paresthesia in hands and feet, 70% dysautonomia, 50% facial palsies.

Diagnostic Criteria: Progressive weakness of legs and arms AND hypo-/areflexia in affected limbs.

Investigations:
- **LP**: ↑protein, normal WBC (albuminocytologic dissociation)
- **EMG**: helps to delineate demyelinating (↓motor nerve conduction, ↑F-wave latency, conduction blocks and temporal dispersion) vs. axonal (↓distal motor and/or sensory amplitudes, transient motor nerve conduction block) (Ann Neurol. 1998 Nov;44(5):780-8)
- **Ab**: IgG to GQ1b
- **MRI Spine**: may show thickening and enhancement of the intrathecal spinal nerve roots and cauda equina. (AJNR Am J Neuroradiol. 2004;25(4):645)

Management:

Supportive Care	Approach	
Respiratory Failure	• ICU or step-down admission for close monitoring • 15-30% of pts will need ventilatory support • Consider intubation: FVC <20ml/kg, max IP <30cmH_2O, max EP <40cmH_2O	
Autonomic Dysfunction	Paroxysmal fluctuations in BP	• Intraarterial monitoring • HYPOtension: IV fluids phenylephrine 40 mcg bolus • HYPERtension (MAP>125): labetolol, esmolol or nitroprusside
	Tachyarrhythmias bradyarrhythmias	• Closely monitor • Atropine or cardiac pacing may be required
	Adynamic ileus	• Erythromycin 3mg/kg IV over 45 min q8h
	Urinary retention	• Neostigmine 0.5-1mg IM q3h up to 5x
Pain Management	• Gabapentin 100-300 mg PO OD-TID • Carbamazepine 200-400 mg PO OD increasing slowly over wks	

	• Epidural morphine • Avoid narcotics, but if used monitor due to autonomic dysfunction

Oncology

Authors: Dr. Grace Zhang, Dr. Kit Birkness *Updated May 2024*

Screening Guidelines

Site	Screening
Breast	The following do not apply to women with higher risk (personal history of breast cancer, history of breast cancer in 1st degree relative, known BRCA1/BRCA2 mutation, prior chest wall radiation): (CMAJ 2011;183(17):1991-2001.) • **Age 40-49**: routine screening not recommended • **Age 50-74**: routine screening mammography q2-3yrs
Cervical	The following applies to screening asymptomatic women who are or have been sexually active: (CMAJ 2013;185(1):35-45.) • Age <24: no routine screening • Age 25-69: routine screening q3yrs • Age ≥70: if have 3 successive negative Pap tests in the last 10yrs, may cease screening; otherwise suggest continuing screening until 3 negative screening results obtained.
Colorectal Cancer (CRC)	*≥50-74 years old with average risk for CRC* (i.e., no first-degree relatives with CRC, no IBD, no pre-cancerous CR polyps): (CMAJ 2016. DOI:10.1503/cmaj.151125) • FOBT (gFOBT or FIT) q2yr OR flexible sigmoidoscopy q10yr *FamHx of colorectal CA* in ≥1st degree relative: (Cancer Care Ontario) • Routine screening with colonoscopy q10yrs beginning at age 50, or 10 years earlier than the age of the relative was diagnosed, whichever occurs first. *Higher risk individuals*: (Can J Gastroenterol 2004;18(2):93-99.) • AAPC: colonoscopy q1yr beginning at age 16-18yrs • FAP: sigmoidoscopy q1yr beginning at age 10-12yrs • HNPCC: colonoscopy q1-2yrs beginning at age 20, or 10yrs younger than the earliest case in the family (whichever was first) • IBD (UC, or CD): based on British Society of Gastroenterology
Lung	Low dose CT chest every year up to three consecutive years is recommended in high-risk populations defined as: • Age of 55-74 • Smoking history of ≥30pack years • Active smoker OR quit less than 15 years ago (CMAJ 2016. DOI:10.1503/cmaj.15142)
Prostate	Controversial issue: (CUAJ 2011;5(4)235-240.) • Decision to use PSA testing for early detection of prostate cancer should be individualized – discuss the risk (false negative and false positive; biopsy; risk of treatment with sx & radiation) • Screening: male >50 years old with at least 10yr life expectancy • Screening interval not specified (evidence suggests ~q2-4yrs) • Age of screening discontinuation: 75yrs

Cerebral Edema (Vasogenic) Secondary to Malignancy

Clinical Features:
Headache – dull, mild at onset and progressively worse, usually with focal neuro deficit, ↑ICP, **aLOC.** Causes nighttime awakenings, nausea & vomiting. Valsava exacerbates.
Vomiting – with/without nausea; projectile vomiting suggests posterior fossa tumors & obstructive hydrocephalus.
Papilledema – visual acuity usually spared though may report transient blurred vision.
Investigations: CT/MRI – distinguishes vasogenic vs. cytotoxic edema
Management:

1. **Glucocorticoids (Vasogenic only):** Dexamethasone 10mg IV x 1, then 4mg IV/PO BID – max effect in <72hrs. Slowly wean steroids when symptoms & signs controlled – monitor closely for relapse.
2. **Consult:** Rad Onc. Consider NeuroSx and/or Med Onc if indicated.

Spinal Cord Compression Secondary to Malignancy

Location: (a) Thoracic: 60%; (b) Lumbosacral: 30%; (c) Cervical: 10%
Note: 50% have more than 1 area of compression
Clinical Features: Variable.
Cancers most often associated are breast, multiple myeloma, lymphoma, lung, prostate

- **Back pain:** (a) Localized & progressive ≈ spine lesion.
  (b) Radicular, worse with recumbency/Valsalva ≈ nerve root compression/ invasion;
  Band-like sensation around body ≈ thoracic radiculopathy.
- **Neurological:** Motor – clumsiness, weakness, may not be ambulatory.
  Sensory – ask about saddle anesthesia, *check sensory level from back* and anal tone.
  Reflexes – increased = cord; low/absent = cauda equina.
- **Bladder & bowel dysfunction:** urinary retention, fecal incontinence – late stage

Investigations: Urgent MRI of the entire spine
Mx:

1. **Dexamethasone**: 10mg IV/PO x1 then 4mgPO/IV BID for duration of radiation
2. **Emergent Consultation:**

- **NeuroSx ASAP if**: (a) able to tolerate surgery; (b) for tissue diagnosis; (c) single site for decompression; (d) pathological fracture; (e) unstable spine; (f) intractable pain; (g) cord has received maximum dose of radiation
- **Rad/Onc**

Pearls: *Do a THOROUGH Neuro exam with documentation – can evolve rapidly!
*Time is of the essence – low threshold to contact attending if delays/uncertainty!

Superior Vena Cava Syndrome

Definition: venous return to the heart impaired due to SVC obstruction
Clinical Features: all 2° to venous congestion in areas drained by SVC.

- Symptoms: fullness in head exacerbated with bending forward, facial swelling; arm swelling; dyspnea, cough, dysphagia – ?laryngeal edema; headache, confusion – ?cerebral edema
- Signs: facial edema, distention of superficial veins in region drained by SVC (upper thorax and above); facial plethora, stridor; Pemberton's sign

Investigations: CT with contrast to define the anatomy; biopsy
Mx:

1. Cerebral/Laryngeal edema: emergent – may require intubation & empirical tx
2. Empirical treatment may obscure the diagnosis of malignancy (only treat if unstable)*: Dexamethasone 10mg IV x 1 then 4mg IV/PO BID.
3. Consult: RadOnc, MedOnc

4. If recurrent, obstruction may benefit from intravascular stenting.
5. Monitor for acute CHF after relieving obstruction

Pearls: * Can defer dexamethasone for a satisfactory biopsy if the patient is stable.
* IV fluids: IV fluid via arm veins will exacerbate SVC syndrome
* ONLY emergency if cerebral edema or laryngeal edema causing airway compromise

Tumour Lysis Syndrome

Definition: Laboratory and Clinical
- **Laboratory (≥2 parameters):** ↑[urate] >475.8umol/L, ↑[PO_4^{-3}] >1.5mmol/L, ↑[K^+] >6.0mmol/L, ↓[Ca^{+2} corrected]<1.75mmol/L, [Ca^{+2}_i]<0.3mmol/L.
- **Clinical (Laboratory + ≥1 parameter):** AKI, arrhythmia, seizure, or death

Risk Factors
- Hematologic malignancy (especially acute leukemias and high-grade lymphomas)
- Dehydration
- Pre-existing kidney disease
- Treatment for cancer in last 7 days (although TLS can occur spontaneously in settings of high tumour burden even WITHOUT treatment)

Investigations: Regularly measure: LDH, lytes, Ca^{+2}, PO_4^{-3}, & uric acid* – frequency depends on severity (q6-12hrs). Auxiliary: rhythm monitoring, or regular ECG.
*[Uric acid]: after rasburicase tx, transport sample on ice (to halt enzyme action)

Mx:

1. **Volume expansion to induce diuresis:** high infusion rates (200-250mL/hr) for urine output (~2mL/kg/hr). Choice: crystalloids – normal saline. (Urine alkalinization attenuates urate crystallization, but ↑[PO_4^{-3}] → $Ca_3(PO_4)_2$ precipitation. (↑[PO_4^{-3}] is more difficult to manage than ↑[urate])).
2. **Urate Mx:**
   - **Allopurinol:** preventative therapy – **renal dosing**
   - **Rasburicase:** indicated when urate >470umol/L, or AKI 2º to urate nephropathy. Be wary of anaphylaxis, & it is contraindicated in G6PD (methemoglobin). At KGH, only the hematologist can order it (Rasburicase 6mg IV x 1).
3. **Renal replacement (dialysis):** standard indications. Commence earlier if anticipating complicated course because of the rapid accumulation of electrolytes. Consult nephrology especially with high-risk TLS.

Other Oncologic Emergencies

Metabolic Complications:
- Hypercalcemia of malignancy – see "Hypercalcemia"
- SIADH – see "Hyponatremia"

Hematologic
- Hyperviscosity syndrome
- Febrile neutropenia – see "Febrile neutropenia"
- Pulmonary embolism – see "Pulmonary embolism"

Structural: Malignant pericardial effusion

Adverse Effects of Chemotherapy

Further information relating to specific chemotherapeutic medications can be found on:
- https://www.cancercareontario.ca/en
- http://www.bccancer.bc.ca/

Complications of Immunotherapy

Definition: inflammation to any organ system secondary to immunologic (cytokines, adoptive cell therapy, checkpoint inhibitors, etc.) cancer treatments
Clinical Features: dependent on organ system involved, can vary greatly in severity from mild to life-threatening. Common presentations requiring admission:
- Colitis (diarrhea, abdo pain) - Hepatitis - Pancreatitis
- Myocarditis - Uveitis
- Pneumonitis: cough, fever (CXR may mimic pneumonia). Can cause ARDS, resp F
- Endocrinopathies: hypo/hyperthyroidism, hypophysitis, DMI, adrenal insufficiency

Investigations: As appropriate depending on organ involvement
Mx:

1. Hold immunotherapy
2. Consider high dose steroids (Prednisone 1-2mg/kg /day) if severe or life-threatening immune-mediated toxicities
3. Early consultation of medical oncology for ongoing management.

Palliative Care

Authors: Dr. Kit Birkness, Dr. Danielle Kain *May 2024*

Approach to Palliative Patient

- *History*: pain, dyspnea, appetite/thirst, ability to swallow, nausea, dry mouth/need for mouth care, constipation, sleep, mood, confusion/hallucinations, hiccups, burping, itchiness, fatigue/drowsiness, patient priorities
- *Physical (of actively dying patient)*: level of consciousness, breathing (rate, pattern, apneas, secretions, work of breathing), comfort (fidgeting, groaning, grimacing), perfusion (temperature dysregulation, colour of lips/nails), skin (breakdown, mottling, edema)
- Complete physical and investigations for non-dying patient only if changes mgmt
- *Assessment*: Palliative Performance Scale score (PPS, see below), ESAS score

PPS %	Ambulation	Activity; Disease	Self-care	Intake	LOC
100	Full	Normal; No disease	Full	Normal	Full
90	Full	Normal; Some	Full	Normal	Full
80	Full	N with effort; Some	Full	Normal/reduced	Full
70	Reduced	Can't do normal job or work; Some	Full	As above	Full
60	Reduced	Can't do hobbies or housework; Some	Occasional assistance needs	As above	Full/confusion
50	Mainly sit/ lie	Can't do any work; Extensive disease	Considerable assistance needs	As above	Full/confusion
40	Mainly in bed	As above	Mainly assistance	As above	Full/confusion/ drowsy
30	Bed bound	As above	Total care	Reduced	As above
20	Bed bound	As above	As above	Minimal	As above
10	Bed bound	As above	As above	Mouth care	Drowsy/coma
0	Death	-	-	-	-

- *Plan*: Update family daily, assess goals of care, add symptomatic management

Basic Comfort Care Order Set

(may include some/all of the following):
1. DNR/DNI, comfort-directed care 2. Discontinue routine vitals
3. RR bid and PRN 4. Discontinue bloodwork
5. Discontinue feeds, daily weights, ins and outs, telemetry, NG tube, etc.

6. Hydromorphone 0.5-1mg subcut OR morphine 2.5-5mg q1h PRN pain/dyspnea (in opioid-naïve patients)
7. Haldol 0.5-1mg subcut q4h PRN, first line for agitation & nausea
8. Nozinan 5mg subcut q4h PRN, second line for agitation & nausea
9. Scopolamine 0.4mg subcut q4h PRN secretions OR Glycopyrrolate 0.4mg subcut q4h PRN secretions (use the latter if patient awake/alert as Scopolamine is sedating)
10. Midazolam 5-10mg IV/IM q5min PRN dyspnea crisis, seizure, or catastrophic hemorrhage; page MD if used
11. Midazolam 1-2mg subcut q30min PRN, third-line for agitation
- Review medication list and discontinue medications not for comfort/symptom control
- Consider: reposition patient q2h, mouth care qid and PRN, O2 for comfort if severe dyspnea + hypoxia, insertion or removal Foley catheter for comfort, removing IVs

Symptom Management

Pain: Consider scheduled opioids or acetaminophen if able to tolerate po intake
Dyspnea: Scheduled opioids
Constipation: Senna 17.2mg po daily PRN; PEG 17g po daily PRN; Bisacodyl suppository PR daily PRN
Nausea (depends on cause): Metoclopramide 10mg po/subcut/IV q4h PRN first-line; Olanzapine 2.5-5mg subcut bid; Dexamethasone 4-8mg po/IV/subcut daily; Ondansetron 4mg po/subcut/IV bid-tid (best for nausea from chemo, otherwise 3rd-line)
Anxiety: Ativan 0.5mg subcut q8h PRN, low-dose Olanzapine also anxiolytic
Sore throat/mouth pain: Lidocaine viscous 2%10-15cc qid PRN, swish and swallow

A Brief Serious Illness Conversation Guide

1. *Prepare* by reviewing PMHx, understanding intent of treatment and prognosis
2. *Introduce the conversation and ask permission*: "I'd like to discuss your illness and what might lie ahead, so that we might better plan for the future. Is that okay?"
3. *Assess patient's understanding*: "What is your current understanding of your illness?" "What have you noticed that has been different in recent weeks (or months?)"
4. *Share prognosis* in "hope/wish, worry, wonder" framework:
E.g. "I want to share with you my understanding of where you are in your illness. **I hope** (that you will continue to feel as well as you have been/ that things improve), but **I worry** that (time may be as short as (weeks, months, years)/you might get sick quickly/this is as well as you will feel). **I wonder** if it might be time to focus on maximizing your quality of life in the time that you have left.
5. *Allow silence, explore emotion:* "How do you feel when I say time may be short?"
6. *Explore*: goals, location of care (home vs other), critical abilities, fears and strengths.
7. *Summarize, make recommendations, affirm*. "Given that X is important to you, I recommend that we do Y."
8. *Plan*: Discuss practicalities of what this decision means, including code status, any changes to medications, bloodwork, vital signs, supports that can be set up at home.
9. *Document* the conversation

Pronouncing a Death

1. *Before entering room*: check code status, determine whether death was expected, and if family is in room, screen for eligibility for organ donation
2. *Enter the room*: introduce yourself, convey regrets, elicit names and relation to deceased, explain your need to perform an end-of-life exam and pronounce the patient, invite family to remain in the room

3. *Examine*: check pulses and listen for heart sounds x1min, listen for breath sounds and look for chest rise x1min (can stop here if palliative pt). Can also assess neurological function by attempting to elicit pain (squeeze nail bed or sternal rub), pupil reactivity (direct pupillary light reflex), can add corneal reflex, then close the patient's eyes.
4. *Pronounce*: note time and date of death.
5. *Discuss next steps:* invite family to stay in room to say goodbye for as long as they'd like. When they're ready, the patient will be brought down to the hospital morgue and the funeral home will collect them from there. Ask which funeral home they will use (for death certificate). It is not necessary to routinely offer autopsy.
6. *Deliver news, document:* Call next of kin if not present in room, otherwise admitting cannot release the body to the funeral home. Fill out death certificate.

Palliative Pearls

- *Secretions*: Very prevalent at end of life. These are often more distressing to families than to patients, can liken to snoring as a comparison: it bothers the person next to you, but not the person sleeping. Most important treatment is reassurance of family. There is low evidence for anti-secretory agents.
- *Pain and nausea*: Treat these symptoms based on their underlying etiology for optimal control.
- *Agitation*: Should be treated in a stepwise fashion from least sedating (Haldol) to more sedating (Nozinan).

Perioperative Medicine

Authors: Dr. Ryan Peters, Dr. Kristen Marosi, Dr. Michael Scaffidi *Updated May 2024*

General Approach: Pre-op assessment checklist

1. **Cardiac risk assessment (**including endocarditis ppx)
2. **Delirium risk and frailty**
3. **Medications and withdrawal** (especially anticoagulants and antiplatelet agents)
4. **Hematologic consideration (**VTE ppx, anemia optimization, coagulopathies tx)
5. **Metabolic (**diabetes, steroids)

Cardiac Risk Assessment

Approach:

1. **Is it emergent/urgent surgery?** Do not delay…EXCEPT if active ACS, decompensated HF, unstable arrhythmia, or anything that requires emergent optimization
2. **Calculate Revised Cardiac Risk Index (RCRI) score to assess for risk of MACE,** if your patient is ≥ 45 years or, is 18-44 years with known significant cardiovascular disease (CAD, CVD, PAD, CHF, PHTN, severe obstructive intracardiac abnormality)

RCRI:

History of ischemic heart disease	+ 1
History of congestive heart failure	+ 1
History of cerebrovascular disease	+ 1
Insulin-dependent diabetes	+ 1
Preoperative serum creatinine > 177 umol/L	+ 1
High risk surgery (intraperitoneal, intrathoracic, suprainguinal, vascular)	+ 1

3. **For elective surgeries,** order BNP if:
   - **i.** Age ≥ 65 years

ii. RCRI ≥1
iii. Age 45 to 64 years with significant CV disease

4. If BNP ordered and it is:

i. **Normal:** no routine post-op routine monitoring
ii. **BNP abnormal or unavailable:** monitoring for myocardial injury after noncardiac injury (MINS) postop trop x 48 to 72 hours, PACU ECG, "shared care management"

5. If MINS present (i.e. asymptomatic post-op troponin elevation), then:

i. **ASA**
ii. **Statin**
iii. **Optimize cardiac risk factors**

Medication Management

	Medication	Action	Notes
Chronic medications	ASA	Hold at least 3 days before surgery; restart post-op when risk of bleeding decreased	-if recent stent (6 weeks bare metal, 3 to 12 months drug-eluting), then continue -if remote stents >12 months, continue if acceptable bleeding risk with surgeon
	P2Y12 platelet receptor inhibitor	Elective surgery: Hold clopidogrel/ticagrelor 5-7 days before surgery; hold prasugrel 7-10 days before surgery	For urgent/emergent surgery: talk to patient, cardio, anesthesia, and surgeon; restart DAPT as soon as safe post-op
	Beta-blocker	Continue, unless SBP is low	
	Statin	Continue	
	SGLT2 inhibitors	Hold 3 to 4 days before surgery	
	Other oral antihyperglycemics (including metformin)	Hold the day of surgery	
	Long acting basal (e.g. glargine)	Once nightly: Reduce dose by 25%	
		Twice daily: Reduce am dose the morning of surgery by 25%	
	Type 1 Diabetics on Basal Insulin	80% of basal insulin dose evening before surgery and 80% the morning before surgery	Prevent hyperglycemia/diabetic ketoacidosis
	Short acting insulins	Hold day of surgery	

	Diuretics	Hold, unless concern of volume overload is high	
	Steroids	Consider stress dose with signs of relative adrenal insufficiency	Relative adrenal insufficiency: *Prednisone >20 mg x 3 weeks *Cushingoid appearance *Confirmed primary or secondary adrenal insufficiency

Anticoagulation

Approach: Balance both thromboembolic risk and procedural bleeding risk
1. **Procedural risk** Does anticoagulation need to be held?
2. **Patient risk:** Is bridging needed?
3. **Bleeding risk:** Surgery specific? Patient specific (i.e. HAS-BLED score)?
4. **How to bridge:** Timing

	Procedural Examples	Anticoagulation
Low bleeding risk	• Minor dental procedure • Minor derm procedure • Cataracts • Minor procedures (e.g. paracentesis) • Endoscopic procedures without biopsy	Continue
High bleeding risk	• Any procedure w/ neuraxial anesthesia • Neurosurgery (intracranial, spine) • Cardiac surgery (e.g. CAGB) • Major vascular surgery (e.g. aortic aneurysm repair) • Major lower limb orthopedic surgery (e.g. hip replacement) • Lung resection surgery • Intestinal anastomosis surgery • Selected procedures (e.g. polypectomy)	Hold

Indications for Bridging:
- Mechanical mitral valve repair or old aortic valve repair (e.g. ball-cage)
- DVT, PE, or arterial TE <3 months
- Chronic afib, CHADS 5-6
- Rheumatic mitral stenosis
- Prior thrombosis when warfarin held
- Severe thrombophilia (APLA, protein C,S ATIII deficiency)

Bridging Warfarin and LMWH:

Day	Action
-5	Stop warfarin
-3	Start full dose LMWH
-1	Last dose of LMWH before OR; administer ½ the dose
Day of OR/POD 1	Start warfarin at 1.5x the usual dose at least 12 hours after OR and full dose LMWH after OR
POD 2	Warfarin 1.5x usual dose + LMWH

POD 3	Warfarin at usual dose + LMWH. Check INR and continue LMWH until INR therapeutic, then stop

Diabetes

Targets: In the perioperative setting, glycemic targets are as follows:

Hospitalized Patient with Diabetes	Blood Glucose Targets (mmol/L)
CABG intraoperatively	5.5-11.1
Perioperatively for other surgeries	5.0-10.0

Management of Insulin Therapy:

- Consider insulin infusion for OR cases >4hrs and patients on large doses of insulin or DM1 on BBI.
- NOTE: KGH has a diabetes pre and post procedure entry point order set.

Thromboprophylaxis

Approach: Thromboprophylaxis in the postoperative setting must balance the risk of VTE with post-operative surgical bleeding risk.

Patient Group	Thromboprophylaxis Options	Duration
Hip or knee arthroplasty	Rivaroxaban 10mg po daily Apixaban 2.5mg PO BID Dabigatran 220mg PO daily Enoxaparin 30mg SC BID or 40mg SC daily Dalteparin 5000 U SC daily Tinzaparin 4500 U or 75 U/kg SC daily Fondaparinux 2.5mg SC daily Nadroparin 38 U/KG daily (day 1-3 post op), 57 U/kg SC daily thereafter ASA: 81mg daily, after receiving rivaroxaban 10mg daily for the first 5 post op days	14-35 days
Hip fracture surgery	Enoxaparin 30 or 40mg SC daily Dalteparin 2500 or 5000 U SC daily Tinzaparin 4500 U SC daily Fondaparinux 2.5mg SC daily Nadroparin 38 U/kg SC daily (day 1-3 post op) then 57U/kg SC daily	14-35 days
Major orthopedic trauma	Enoxaparin 30mg SC BID Dalteparin 5000 U SC daily Tinzaparin 4500 U SC daily *once hemostasis evident	Until discharge (including rehabilitation)
Spine surgery	Uncomplicated-mobilization alone Complicated (cancer, spinal cord injury with leg weakness or paralysis, prior VTE, combined anterior/posterior approach)—LMWH once daily starting day after surgery	Until discharge (including rehabilitation)
Isolated below knee fracture	No prophylaxis if outpatient or overnight hospital stay LMWH once daily if inpatient	Until discharge (including rehabilitation)
Knee arthroscopy	Low risk—None	5-30 days

	Higher risk (major knee reconstruction, prior VTE, cancer, other VTE risk factors)—LMWH once daily or direct oral anticoagulant	
Lower extremity amputation	LMWH once daily	Until discharge (including rehabilitation)
Other (bedrest, incision and drainage, etc.)	LMWH once daily	Until discharge

Respirology

Updated May 2024

Approach to Dyspnea

Approach

- **1º Survey – ABCD:** Airway threatened? Stat vitals! Possible Hypercarbia (↓LOC, signs of fatigue). **Rapid Cardiac and Resp Exam 1st** (narrows DDx)
- **1º Resus: Intubation?** Suction? O_2 (Venturi, NRB) + NIPPV?
- **1º Adjuncts: IV**, Monitor, Crash Cart. STAT tests: **ECG, CXR**, VBG (ABG if have time & hands), Bloodwork.
- **2º Survey:** Assess for high yield features that will narrow the differential – DO NOT spend too much time on Hx & Exam if the patient has yet to be stabilized. Do bedside POCUS if within skillset: B lines? Line sliding? IVC+JVP? Pleural effusion?
- **Baseline Level of Dyspnea:** Ask about multiple activities to be certain. Use mMRC to quantify – standardized scale for communication.
- **Progression:** Rate of deterioration. Rapid? Or else ask how was the breathing half a year ago, a year ago, etc…
- **Walk Test:** Verify (1) Degree of dyspnea (mMRC) – complaint-to-objectivity correlation; (2) O_2: Any desaturation? Does it correlate with dyspnea?

Etiologies: (Focusing on the common causes that tend occur overnight call)

Respiratory

- **AECOPD:** Smoker + wheeze + ↑CO_2 on ABG/VBG is quite suggestive e of the diagnosis. AECOPD can coexist with other conditions such as CHF.
- **Pneumonia:** Usually diagnosed on admission but deteriorate due to natural Hx, Tx failure, secretion Mx issues, resp fatigue/airway compromise. Or hospital acquired.
- **Pleural Effusion:** CXR: AP film can "hide" substantial vol. if the patient does not sit upright and inspire fully. Look for other signs of volume overload or asymmetry on auscultation.
- **Aspiration:** Stroke, dementia, delirium, GERD are common causes. Hx clues (nursing/PSW). CXR may not show anything initially as the pneumonitis has yet to manifest radiographically; RLL iniltrate may be seen.
- **PE:** Difficult to diagnose– note risk factors for DVT/PE. There is no diagnostically useful parameter for PE itself though +ve exam for DVT may point towards PE. Consider when other causes are less likely – usually in setting of relatively normal CXR (compared with past films) and cardiac causes have been ruled out.
- **Pneumothorax: Almost all iatrogenic if developed during hospital stay** (thoracentesis, bronchoscopy, biopsy). Be vigilant of tension pneumothorax – ↓BP, distended neck veins, trachea deviates AWAY FROM the hemithorax with less/no breath sound.

- **Mucus plug:** Hx (weak cough), ↓breath sounds, or atelectatic changes on CXR.
- **Non-cardiogenic pulmonary edema:** ARDS closely resembles CHF but asymmetrically distributed and not responsive to furosemide.

Cardiac

- **CHF:** Orthnopnea, ↑JVP, bilateral crackles, leg swelling. Elucidate cause: volume overload, ACS, Dysrhythmia, Valvulopathy. B lines + distended IVC.
- **Pericardial tamponade:** requires **HIGH INDEX OF SUSPICISION**. ↓BP, ↑JVP, muffled heart sounds, low voltage/electrical alternans, cardiomegaly, recent cardiac procedure.

Metabolic

- **Bleed:** Shortness of breath or tachypnea due to insufficient tissue oxygen delivery
- **Acidosis:** DKA, RTA etc.

Airway Compromise

- **Obstruction:** less common on the internal medicine ward – Think if stridor, supraclavicular in drawing, anaphylaxis (usually antibiotics, IV ferrous dextran). Most importantly, do they require intubation or is there anaphylaxis?

Asthma Exacerbation

Approach to Acute Exacerbation Mx

1. ABCD: Vitals, mental status, accessory muscle use. Blood gas to assess ventilation – a normal CO_2 is concerning, should be hypocapneic unless tiring.

2. Classify and Management based on Severity:

Severity	Characteristics	Principles of Mx	Dispo.
Mild	Mild effort, Good and rapid response to β-agonist, low CO_2	Bronchodilators + inhaled steroids	D/C if improved
Moderate	Strenuous effort to breath, Partial response to β-agonist, normal CO_2	Bronchodilators + Glucocorticoid + $MgSO_4$	Ward, D4ICU
Severe	Struggling, Diaphoretic, Difficult to speak, N/↑CO_2	Get Help, Intubation	ICU
Near Death	Exhausted, ↓LOC, ↓RR/HR, Silent Chest, N/↑CO_2	Bronchodilators + Glucocorticoid + $MgSO_4$	ICU

3. Medications:

Salbutamol	**MDI:** 4puffs inh q4h regular and q1h PRN dyspnea **Nebs:** 2.5-5mg neb q4h regular and q1h PRN dyspnea (severe: q20min x3 to start; use higher dosage)
Ipratropium	**MDI:** 4puffs inh q4h regular and q1h PRN dyspnea **Nebs:** 250-500mcg neb q4h regular and q1h PRN dyspnea (severe: q20min x3 to start)
Prednisone	50mg PO QD x 10-14d
Methylprednisolone	125mg IV QD x 5d then transition to PO for total 14 d
Magnesium sulphate	2g IV over 20min
Ketamine	Call anesthesia

4. History: Previous visits/admissions, intubations, steroids use, triggers, chronic control adequacy, Respirologist who follows?

Pearls of Mx:

- Be very **aggressive** with the treatment and do not treat like AECOPD
- **Differences in asthma and COPD treatment:**
    - Longer prednisone course for asthma

- NIPPV in asthma is controversial: May help WRB but may increase Auto-PEEP. Consult staff/ICU prior.
- A normal CO_2 is concerning in asthma (pt is tiring)

- Low threshold to consult Critical Care
- Frequent and close monitoring: clinically and via ABG (reassess in 30min)

Criteria for Discharge: Clinical stabilization with resolution of respiratory distress, No evidence of respiratory failure, and PEF>70%

Acute Exacerbation of COPD

Approach to Acute Exacerbation Mx

1. ABCD: Vitals, mental status, accessory muscle use. Obtain blood gas to assess.

2. Classify and Management based on Severity:

Severity	Characteristics	Principles of Mx
Mild	Mild effort, Good and rapid response to β-agonist	Bronchodilators
Moderate	Strenuous effort to breathe, Partial response to β-agonist	Bronchodilators + Glucocorticoid ± BiLevel
Severe	Struggling, Diaphoretic, Difficult to speak, N/↑CO_2	Triall BiLevel with low threshold to intubate; Bronchodilators + Glucocorticoid

3. BiLevel NIPPV Indication: acute respiratory acidosis with pH<7.30, respiratory distress. No utility for chronic ↑ [CO_2] with normal pH.

4. Medications:

Bronchodilator	Dose	Notes
Salbutamol	**MDI:** 4puffs inh q4h regular and q1h PRN dyspnea **Nebs:** 2.5-5mg neb q4h regular and q1h PRN dyspnea (severe: q20min x3 to start)	**MDI:** always with **aerochamber** – ensure the patient uses it properly and able to hold breath for ~10s. **Nebs:** avoid if requiring NIPPV (need to remove NIPPV for 20 min).
Ipratropium	**MDI:** 4puffs inh q4h regular and q1h PRN dyspnea **Nebs:** 250-500mcg neb q4h regular and q1h PRN dyspnea (severe: q20min x3 to start)	
Home inhalers	Continue regular home inhalers	
Steroids		
Prednisone	50mg PO QD x 5d	Consider in moderate to severe AECOPD.
Methylprednisolone	40mg IV divided in 1-2 doses	

DO NOT HOLD BRONCHODILATORS FOR NIPPV – nurses may do this, and you must ensure that they don't. NIPPV must be stopped briefly to give bronchodilators (MDIs can be given quickly with an aerochamber) then resume NIPPV. **Inadequate bronchodilation leads to intubation.**

5. Antibiotics: NOT ALL AECOPD requires empiric antibiotic treatment
- Indication: purulent sputum, ↑sputum volume, severe exacerbation, radiographic evidence of pneumonia

6. Treat the Precipitant: resp. infection, CHF, PE

Important to elicit: (1) Baseline Fxn – define with mMRC and DALs; (2) FEV1 severity by GOLD criteria; (3) Home O_2; (4) CO_2 retainer; (5) Home NIPPV and settings; (6) Annual # exacerbations; (7) Recent antibiotic use, previous confirmed pathogens; (8) Vaccination; (9) Patient is from: home, retirement home; nursing home,

or other institutions; (10) Smoking Hx: habits and attempts to quit; (11) PFT: most recent; (12) Respirologist: ? following; (13) COPD rehab: enrolled?

Chronic Mx:

1. **Smoking cessation:** Council upon every encounter. Offer pharmacological agent.
2. **Prevention:** Annual Influenza and pneumococcal q5-10yrs vaccination.
3. **COPD Action Plan**
4. Regular long LAMA/LABA therapy for mod to severe COPD
5. Regular ICS+LABA/LAMA for mod/severe COPD with >1 AECOPD in the last yr
6. Frequent exacerbations: Azithromycin 250mg PO 3 times/week.
7. **Pulmonary Rehab:** improves QoL, dyspnea & exercise endurance (6MWT)
8. **O_2 therapy:** If PaO_2<55mmHg, or PaO_2<60mmHg with bilateral ankle swelling, cor pulmonale or hematocrit >56%; Target: long term continuous O_2 for >15hr/d.
9. **NAC-** useful in some patients for reducing secretions.

Inhalers

Short Acting β2-Agonist (SABA)			Short Acting Muscarinic Antagonist (SAMA)
	**Ventolin® MDI®** Salbutamol 100 mcg		**Ventolin® Diskus®** Salbutamol 200 mcg		**Atrovent® MDI®** Ipratroprium 20 mcg
	**Airomir™ MDI®** Salbutamol 100 mcg		**Bricanyl® Turbuhaler®** Terbutaline 0.5 mg		

Long Acting β2-Agonist (LABA)			
	**Serevent® Diskus®** Salmeterol 50 mcg BID		**Oxeze® Turbuhaler®** Formoterol 6, 12 mcg BID		**Foradil®** Formoterol 12 mcg BID
	**Onbrez® Breehaler®** Indacterol 75 mcg QD				

Long Acting Anti-Cholinergic (LAAC)			
	**Spiriva® HandiHaler®** 18 mcg QD		**Spiriva® Respimat®** 2.5 mcg, 2 puffs QD		**Tudorza™ Genuair™** Aclidinium 400 mcg BID
	**Seebri® Breehaler®** Glycopyrronium 50 mcg QD		**Icruse™ Ellipta®** Umeclidinium 62.5 mcg QD		

Inhaled Corticosteroids (ICS)		
 **Flovent® MDI®** Fluticasone 50, 100, 250 mcg BID	 **Flovent® Diskus®** Fluticasone 50, 100, 250, 500 mcg BID	 **Arnuity™ Ellipta®** Flurticasone 100, 200 mcg OD
 **Alvesco®** Ciclesonide 100, 200 mcg OD/BID	 **Asmanex™ Twisthlaer™** Mometasone 200, 400 mcg QD/BID	 **Pulmicort® Turbuhaler®** Budesonide 100, 200, 400 mcg BID
 **QVAR® MDI®** Beclomethasone 50, 100 mcg BID		

ICS + LABA		
 **Advair® Diskus®** Salmeterol/ Fluticasone 50/100 mcg, 50/250 mcg, 50/500 mcg one puff BID	 **Advair® MDI®** Salmeterol/ Fluticasone 25/125 mcg, 25/250 mcg BID 1-2 puffs BID	 **Symbicort® Turbuhaler®** Budesonide/ Formoterol 100/6, 200/6 mcg BID
 **Zenhale™ MDI ®** Mometasone/ Formoterol 50/5, 100/5, 200/5 mcg BID	 **Breo™ Ellipta™** Fluticasone/ Vilanterol 100/25 mcg or 200/25 mcg QD	

LABA + LAAC		SABA + SAMA
 **Anoro™ Ellipta™** Umeclidinium/ Vilanterol 62.5/25mcg QD (30 doses per device)	 **Ultribro® Breehaler®** Indacterol/ Glycopyrronium 110/50mcg (6 or 30 capsules per box) QD	 **Combivent® Respimat®** 20/100mcg QID (120 actuation per cartridge)
 **Spiolto™ Respima®** Tiotproprium/ Olodaterol 2.5/2.5mcg QD (28 or 60 doses per device)	 **Duaklir™ Genuair®** Aclidiniumm/ Formoterol 400/12mcg BID (60 doses per device)	 **Combivent® Respimat®** 20/100mcg QID (200 actuation per cartridge)

ICS + LABA +LAMA		
 **Trelegy** Fluticasone furoate/Umeclidinium Vilanterol 100 *or* 200/62.5/25 mcg	 **Breztri** Budesonide/glycopyrrolate/formoterol fumarate 160/9/4.8 mcg	

Pleural Effusion

Thoracentesis

Indication:
- **Diagnostic**
- **Therapeutic:**
  - Mitigating respiratory failure
  - Reliving symptoms: shortness of breath, chest pain

Contraindications -- All Relative:
- Localized infection over the proposed site for thoracentesis.
- Coagulopathy:
  - Depends on the comfort of the operator
  - Anticipated difficulty with pleural space entry (eg. large habitus)
  - Warfarin: most are comfortable with INR < 1.7
  - DOACS (relative): most are comfortable after withholding for 1-2 days
- Positive Pressure Ventilation: inflates lung - may increase risk of pneumothorax

Transudate or Exudate? narrows the diagnosis. The most common methods are:

a. Pleural Fluid Analysis
- **Light's Criteria:** Exudative if 1/3 +ve: (a) fluid/serum protein >0.5; (b) LDH ratio >0.6; (c) LDH fluid is more than 2/3 of upper limit of normal of serum LDH level. Caveats: pleural fluid can be concentrated via diuresis or other modalities of fluid removal resulting in a pseudo-exudate.
- **pH <7.2** → empyema is most concerning cause (other causes: malignancy, rheumatoid arthritis, esophageal rupture)
- Cell count + differential: the differential may be clue to diagnosis
- Culture and Gram stain
- Cytology: Sen 60% on 1st tap, addition of 15% with 2nd tap
- Glucose
- Amylase: when considering pancreatitis related pleural effusion
- Triglycerides, chylomircons: when considering chylothorax
- Flow cytometry: if concerns for lymphoma
- Other: adenosine deaminase, fluid hematocrit, AFB

Order all the underlined tests, additional tests depending on clinical scenario

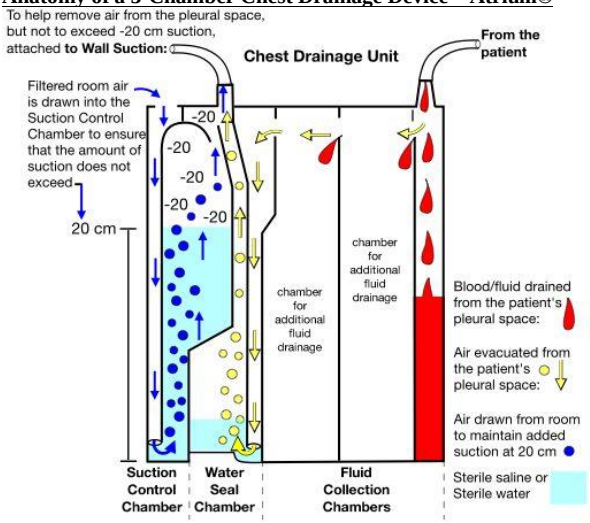
b. Pleural Ultrasound, CT Thorax: inferring the diagnosis based on the complexity of the pleural space and fluid characteristics: loculation, fibrinous strands, echogenic material in the pleural fluid.

Etiology: the clinical context is absolutely crucial to elucidate the cause

Transudate		Exudate	
↑capillary hydrostatic pressure	↓plasma oncotic pressure	↑permeability of pleural capillaries	Lymphatic dysfunction
• CHF • Constrictive pericarditis	• Nephrotic syndrome • Cirrhosis • Hypo albuminemia	• **Inflammatory – infectious:** pneumonia, empyema, lung abscess • **Inflammatory - non-infectious:** Malignancy, collagen vascular disease (SLE, RA), subdiaphragmatic irritation (pancreatitis), PE with infarction, Chronic CHF, Trauma	

Chest Tube and Drainage System

Anatomy of a 3-Chamber Chest Drainage Device – Atrium®



1. **Fluid Collection Chambers:** collect fluid via gravity & overflow.
2. **H_2O Seal Chamber:** When this chamber is attached to suction, air from Chamber 1 is suctioned through the H_2O seal & bubbles up into a sub-chamber where it will be removed by the negative pressure.
3. **Chamber 3:** Suction Control

Daily Chest Tube Mx:

Observation	Problem	Solution(s)
Tube and/or Water Seal Chamber fluid does not oscillate with respiration	Absent oscillation: (1) the drain is blocked, or (2) out of the pleural cavity.	ABCs. Check if tube is displaced. Unkink tube. Ask patient to cough to see if it oscillates again. Re-position patient. CXR to evaluate tube position. DO NOT ADVANCE THE CHEST TUBE INTO THE THORAX.
Unexpected bubbling in the water seal chamber, or Excessive bubbling on suction	Air is leaking from the (1) lung, (2) insertion site, or (3) tubings and drains.	ABCs. CXR to evaluate pneumothorax. Check if system is disconnected. Reconnect & secure with tape. Check tube insertion site for loose dressing, or "hissing" sound. Reinforce with occlusive dressing. If tube is dislodged, see below.
Bloody drainage	Pre-existing, or Iatrogenic hemothorax	ABCs. Check CBC. CXR to evaluate chest tube position and effusion size. Call Thoracic Surgery ASAP if gross hemothorax.
Chest tube dislodged	Incomplete system – dangerous if the chest tube was inserted for pneumothorax	ABCs. CXR. call Resp. Check insertion site for "hissing" sound. Apply a flutter valve dressing – seal ONLY 3 edges. The unsealed 4th edge allows air to escape during expiration. The dressing collapses during inspiration → preventing air entering the pleural space.
Clamping	Dangerous if there is an air leak proximal to the clamp.	NEVER clamp without supervision if there is bubbling in the Water Seal Chamber. Otherwise, there is no escape for the air & a tension pneumothorax can develop.

Chest Tube Discontinuation

Effusion	When daily drainage is <100 - 150mL.
Pneumothorax	When there is no bubbling in the water seal chamber + pneumothorax is resolved radiographically. Clamp and repeat CXR before removing.

Approach to PFT

Clinical Questions the PFT Answer: (1) Is there an obstruction? (2) Is there a restriction? (3) Is there hyperinflation/gas trapping? (4) Is there diffusion abnormalities?

Components of PFT (Needs to be ordered individually on the requisition as per indicated): (1) Spirometry: FEV1, FVC, flow-volume loops; (2) Post-bronchodilator effects; (3) Lung Volumes (Measures TLC, FRC, RV); (4) Diffusion Capacity; (5) ABG and/or Pulse Oximetry; (6) Respiratory Pressures (MIP, MEP); (7) Peak Expiratory Flow Rate.

Contraindications: There are no absolute contraindications though the following are conditions that one should be wary of due to the physiology generated by the respiratory efforts during the procedure.

Interpretation:

	Question?	What to look for?			
1	Comparable? Patient vs Reference	Are the entered characteristics as per the patient's? (Clerical error) **Reference standard:** Predicted "ℕ" spirometry values are generated via regression equations based on a population without physiologic lung impairment. Although seldom need to be done, ensure that the reference standard is appropriate, usually ethnically, for the patient especially when there is a clinical/imaging to PFT discrepancy.			
2	Acceptable?	Good expiratory effort made. Either commented or illustrated by the curve/loop.			
	Reproducible?	Tests are repeated 3x. Must meet ATS criteria for reproducibility.			
3	Upper airway obstruction?	Flow volume loop characteristics			
	Question?	**FEV_1/ FVC**	**FVC**	**TLC**	**Adjuncts**
4	Obstructive?	↓	N or ↓	N or ↑	Reversibility? (↑FEV_1 post-bronchodilator by ≥12% + ↑FEV_1 by 200mL.)
5	Restrictive?	N or ↑	↓	↓	Lung Vol: TLC, RV, VC, FRC tend to ↓ though not all at once. Parenchymal involvement? ↓ DLCO Extraparenchyma? N DLCO, ↓ MIP/MEP
6	Combined?	↓		↓	
	Question?	**What to look for?**			
7	Isolated diffusion abnormality?	If ↑DLCO and airspace disease on CXR, then suspect alveolar hemorrhage. If ↓DLCO and normal lung volumes, suspect anemia, PE, pulmonary HTN, R-to-L shunt, early ILD or emphysema			

PEARLS:
*****Step 2 cannot be further emphasized.
*****The morphology of the flow-volume curve is usually indicative of the process.

Rheumatology

Authors: Dr. Simone Ten Kortenaar, Dr. Pardis Balari *Updated May 2024*

Rheumatological Blood Tests

PEARLS:
*****no single antibody test is diagnostic without accompanying history and physical exam findings.
*avoid screening asymptomatic patients.

Anti-Nuclear Antibody (ANA): non-specific and often used as a screening test. It may be elevated in those with rheumatoid arthritis, Sjogren's syndrome, systemic sclerosis, inflammatory myositis, or mixed connective diseases. A negative ANA makes SLE very unlikely.

Interpretation: if positive, it is reported as a titre (number of dilutions after which it is still positive).
*A titre of 1:40 will be read positive, but in a healthy individual it is rarely significant.
* titres >1:640 are very high and suggestive of autoimmune disease
* titres >1:320 are a "middle-ground" and warrants assessment for disease activity

Extractable Nuclear Antigen (ENA) panel: assesses levels of autoantibodies to several different proteins. ENA is ordered for initial diagnosis in those suspected of having a connective tissue disease, or as a follow up to a positive ANA. As with any rheumatologic test, the interpretation of the ENA panel is not always straightforward, and there is often overlap with other conditions. The panel typically includes anti-dsDNA, anti-chromatin, anti-SSA, anti-SSB, anti-centromere, anti-Sm, anti-RNP, anti-Scl-70, and anti-Jo-1.

Anti-Neutrophil Cytoplasmic Antibodies (ANCA): classically associated with small-vessel vasculitides: granulomatosis with polyangiitis (GPA, previously Wegener's Granulomatosis), microscopic polyangiitis (MPA), and eosinophilic granulomatosis with polyangiitis (EGPA, previously Churg-Strauss Syndrome). There is the potential for overlap. Polyarteritis nodosa (PAN) is NOT associated with ANCAs.

Stain Pattern	Antigen	Vasculitides
c-ANCA (cytoplasmic)	PR3	GPA
p-ANCA (perinuclear)	Multiple, MPO	Non-specific (MPA, EGPA)

Complement (C3, C4): complement can be consumed in SLE, cryoglobulinema, and vasculitides. They can also be low in endocarditis, sepsis, and GN.

Rheumatoid Factor (RF): moderately non-specific! It can be positive in RA, Sjogren's syndrome, MCTD, SLE, poly/dermatomyositis, hepatitis B or C, sarcoidosis, malignancy (particularly B-cell neoplasm), and primary biliary cholangitis.

Anti-Cyclic Citrullinated Peptide (Anti-CCP): used in conjunction with RF and has better specificity for RA than RF (96% specificity, 67% sensitivity). There is cross-reactivity in those with TB, Sjogren's syndrome, and chronic lung disease, etc.

Anti-dsDNA: specific for SLE. Negative dsDNA does not rule out SLE and interpretation should always be made in the context of history, physical exam, and lab investigations. It can also be used to monitor SLE activity.

Arthropathies

- **Seropositive** – polymyositis/dermatomyositis, systemic lupus erythematosus (SLE), scleroderma, rheumatoid arthritis (PSSR).
- **Seronegative diseases** – pondyloarthropathies including ankylosing spondylitis, reactive arthritis, psoriatic arthritis, and 110nteropathic (IBD-associated) arthritis. Seronegative rheumatoid arthritis.
- **Vasculitides** – particularly ANCA vasculitis.
- **Crystal-induced** – gout, pseudogout (calcium pyrophosphate deposition or CPPD), hydroxyapatite deposition disease.
- **Infectious** – also known as septic joints. Can be further classified as gonococcal, non-gonococcal (S. aureus, GAS, S. pneumonia, GNB, anaerobes), viral, fungal, or atypical (Lyme disease associated).
- **Traumatic** – fracture, hemarthrosis, foreign body.

- **Non-arthritis** – osteomyelitis, avascular necrosis, tendonitis, ligamental injury, bursitis.
- **Degenerative** – osteoarthritis.
- **Other** – sarcoidosis, Still's disease, Familial Mediterranean fever, TB.

Number and Patterns of Joint Involvement

Monoarthritis (1 joint) – you should always rule out a ***septic joint*** with a new monoarthritis. Differentials: crystal-induced, traumatic, spondyloarthropathies, degenerative conditions. Particular joint involvement varies and may range from an isolated knee to an ankle or MCP/MTP for example.

Oligoarthritis (2-4 joints) – asymmetric oligoarthritis often involves a solitary finger and toe, knee, or elbow, etc, but can still be seen in RA, vasculitis, infectious (don't forget about Lyme disease!), or crystal-induced arthropathies.

Polyarthritis (≥5 joints) – the classic presentation of seropositive arthropathies such as RA and SLE involves symmetrical small joint involvement (for example, MCPs, wrists, MTPs, ankles). SLE arthritis is non-erosive while RA causes true joint erosions. Large joints can be involved in RA, SLE, or spondyloarthropathies in addition to small joint involvement.

Synovial Fluid Analysis

Synovial fluid should always be sent for analysis of the following:

- Cell count
- Culture
- Gram stain
- Crystals
  - Gout: yellow, needle shaped, negatively birefringent crystals
  - Pseudogout: blue, rod/rhomboid shaped, positively birefringent crystals

Synovial fluid type	**Cell count per mm^3, ($2x10^9/L$)**
Normal	<200
Non-inflammatory (OA, trauma, avascular necrosis, etc)	<2000
Inflammatory (crystal-induced, RA, spondyloarthritis, etc)	>2000
Septic joint	>50,000

Systemic Lupus Erythematosus (SLE)

Not all positive ANA tests indicate SLE! Before you order an ANA, think about what features on history or physical exam could persuade you that this patient may have SLE. If the ANA is negative, it is *unlikely* to be SLE.

- SLE is classified using the Systemic Lupus International Collaborating Clinics (SLICC) criteria. However, the risks of untreated SLE outweigh the need to always check all the boxes of the SLICC criteria.
- Your suspicion for SLE should be peaked if 4 or more criteria are met, which includes at least 1 clinical and at least 1 immunological/lab feature.

- Criteria are also met if a patient has biopsy-proven SLE nephritis with a positive ANA or anti-dsDNA.
- Whenever SLE patients are unwell, rule out disease flares as a cause. C3, C4, and anti-dsDNA may guide etiology + UA and renal function.

Clinical Criteria	Immunological Criteria
Acute cutaneous changes	ANA
Chronic cutaneous changes	Anti-dsDNA
Oral or nasal ulcers	Anti-Sm antibody
Non-scarring alopecia	Antiphospholipid antibody
Arthritis (synovitis or tenderness with morning stiffness in 2 or more joints)	Low complement (C3, C4) levels
Serositis (pleuritis, pleural effusion, pericardial effusion)	Direct Coombs test (without presence of hemolytic anemia)
Renal involvement (proteinuria or RBC casts)	
Neurological changes (seizures, psychosis)	
Hemolytic anemia	
Leukopenia (without an identifiable cause)	
Thrombocytopenia (without an identifiable cause)	

Systemic Sclerosis/Scleroderma

Systemic sclerosis is then further subdivided into **limited cutaneous systemic sclerosis** (lcSSc) and **diffuse cutaneous systemic sclerosis** (dcSSc). You may recognize limited cutaneous systemic sclerosis as "CREST" syndrome: calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasias. LcSSc tends to affect the face and extremities distal to the elbows/knees, while dcSSc affects extremities proximal to the the elbows/knees.

Scleroderma renal crisis (SRC) can be life-threatening without timely diagnosis and management. It affects 5-20% of patients with dcSSc and 1-2% of patients with lcSSc. It almost always occurs within the first five years of disease onset. It features:

- AKI
- Moderated to marked hypertension (BP >140/80 mmHg)
- Proteinuria (while SRC can also be associated with microscopic hematuria it does not manifest as GN)
- New-onset thrombocytopenia or hemolysis

Treatment: Aim to return patient to their previous baseline blood pressure within 72 hours (captopril – titrate to BID or TID). AVOID GLUCOCORTICOIDS – can be a potential trigger for SRC.

Vasculitis

Large vessel – Giant Cell Arteritis (GCA)

- Age >50, F>M
- Fronto-temporal headache, scalp tenderness, jaw claudication

- Visual changes are the most severe manifestation of GCA (with a risk of blindness if left untreated). Stroke and TIA are other serious potential complications of large vessel ischemia.
- Fatigue, malaise, fever, weight loss.
- Symptoms of polymyalgia rheumatica (severe shoulder/hip girdle stiffness)
- ESR >50
- Temporal artery biopsy is the gold standard for diagnosis and should be done urgently in suspected patients (done by opthalmology in Kingston). Keep in mind that the sensitivity of this diagnostic modality is relatively low (40-60%).

Treatment: 1 mg/kg/day (up to 80 mg) prednisone for 2-4 weeks with a gradual taper over 6 months.

Small vessel – ANCA Vasculitides

- May present acutely with alveolar hemorrhage or GN
- In patients with both pulmonary and renal involvement, always rule out Goodpasture's syndrome/anti-GBM. While ANCA vasculitis can affect other organ systems as well, anti-GBM is limited to lung and renal involvement.
- Patients can develop asymmetric sensory/motor neuropathy from damage to at least two separate nerve areas (**mononeuritis multiplex**).

	GPA	**MPA**	**EGPA**
ANCA	Mainly PR3	Mainly MPO	Mainly MPO
Lung	Alveolar hemorrhage, lung nodules	Alveolar hemorrhage	Pleural effusion, patchy infiltrates
Renal	Frequent glomerulonephritis	Frequent glomerulonephritis	Rare glomerulonephritis
Skin	Purpura	Purpura	Purpura
Mononeuritis multiplex	Sometimes	Sometimes	Often

Toxidromes

Updated May 2024

Approach to Toxidromes

Principles of Mx:

1. **Stabilize**: ABCDE
2. **CALL POISON CONTROL ASAP (Phone: 1-800-268-9017)**
3. **Elucidate the Toxin(s)**
4. **Decontaminate:** Remove yet to be absorbed toxin. Remove what you can from mouth & skin WITH protective gear. Consult Experts for indication for invasive methods (activated charcoal, irrigation, gastric/whole bowel, surgical removal).
5. **Antidote:** NAC (acetaminophen overdose), Digibind® (digoxin OD), ethanol (toxic alcohol), fomepizole (toxic alcohol), O_2 (CO2)
6. **Eliminate:** Urine alkalinization (useful for weak acids ie, ASA). Hemodialysis (small molecules). Hemofiltration (mid-sized molecules).

Elucidating the Toxin(s):

a. **History:** mandatory information you should elicit
- **Source:** Patient, **collateral (anyone at the scene)**, note/diary

- **Substance:** Elicit what was taken (prescribed meds, EtOH co-ingestion, other illicit drugs), what was found on the scene, history of overdoes, preparation (ie, fast acting vs sustained release)
- **Amount:** dose on the container, pill count, any regurgitated
- **Timing:** one-time bolus vs interval ingestion

b. Syndromic Pattern Recognition:

- **Anticholinergic** (ie, atropine, scopolamine, TCAs, diphenhydramine): "Hot as a hare, Blind as a bat, Dry as a bone, Red as a beet, Mad as a hatter", urinary retention and decreased GI motility
- **Cholinergic** (ie, organophosphates, carbamates): diarrhea, urination, miosis, bronchospasm, bronchorrhea, emesis, lacrimation, salivation
- **Sympathomimetic** (ie, cocaine, amphetamines, caffeine, nicotine, ASA): CNS excitation, ↑HR, ↑BP, fever, mydriasis, diaphoresis
- **Narcotic and Sedative/Hypnotic** (ie, alcohols, opioids, benzo, barbiturate): ΔLOC, ↓RR, ↓HR, ↓BP, ↓GI motility, miosis for opiates

c. Investigations: Preliminary yet mandatory

- CBC, Lytes, Cr, Glucose, serum osmol, **ABG**, ALT, AST, $Bili_T$, INR, lactate
- Blood Drug Screen: ASA, acetaminophen, ethanol
- Urine Drug Screen (lots of caveats and cross reactivity)
- ECG

Raised Anion Gap & Osmolar Gap: Is there a "hidden toxin"?

Anion Gap = $[Na^+] - [Cl^-] - [HCO_3^-]$

- Normal AG <12

↑**Anion Gap**: use "**KULT"** to rapidly determine whether the ↑AG is due to a toxin – **K**: ketones; **U**: urea (when >40mmol/L); **L**: lactate; **T**: toxin (methanol, ethylene glycol, paraldehyde, salicylates)

Osmol Gap = serum osmol – calculated osmol

- Calculated osm = $2[Na^+]$ + [Glu] + [urea]
- Normal Osmol Gap <10

↑**Osmol Gap:** EtOH, Methanol, Ethylene Glycol, Isopropyl alcohol, Osmotic agents (eg. mannitol)

Aspirin (ASA) Overdose

Pharmacokinetics/dynamics:
Absorption: rapid in stomach and small intestine. Peak level: in 1hr though delayed for enteric coated/sustained release formulation (hrs to days). Vol. of distribution: 0.1-0.3L/kg (- with academia) as ~90% protein bound. Elimination ½ life: 2-4hrs (unsaturated) and up to 30hrs (saturated and tissue bound). Metabolism: hepatic. Excretion: renal. During overdose, absorption & elimination alter significantly which delays peak level being reached by several hours, or more.

Toxicity: Fatal dose for adult ~10-30g. Clinically manifests when serum salicylate >2.9-3.6mmol/L.

Clinical Features: Early: hyperventilation, tachycardia, GI irritation, and tinnitus. Late: clinical instability, altered LOC (cerebral edema), pulmonary edema (non-cardiogenic), hyperthermia. Acute intoxication: usually follows the described Early to Late features. Chronic: non-specific – confusion, metabolic acidosis – though cerebral and pulmonary edema are more common

Investigations:
- Acid/base (overtime): Respiratory alkalosis (early)→Mixed→Metabolic acidosis (late)
- Serum salicylate levels do not adequately assess seriously poisoned patients as it does not reflect tissue fraction of the drug. Severe poisoning occurs at lower (or normal) serum levels for chronic intoxication.

Mx:

1. **Principles of intoxication Mx**
2. **GI decontamination:** activated charcoal 1g/kg (max 50g) orally if alert and cooperative
3. **Indications for dialysis:**
   (a) Severe Clinical Syndromes: altered LOC (cerebral edema), pulmonary edema
   (b) Renal insufficiency that interferes with salicylate excretion
   (c) Fluid overload that prevents the administration of sodium bicarbonate
   (d) Serum salicylate >7.2mmol/L
   (e) Clinical deterioration despite aggressive and appropriate supportive care
4. **Glucose supplementation:** neuroglycopenia may result in aLOC despite N serum glucose.
5. **Serum and urine alkalinization**:** (Alkalemia from respiratory alkalosis is not a contraindication as often have base deficit)

- Bolus $NaHCO_3$ 1-2 amps then maintenance infusion 3 amps $NaHCO_3$ (1 amp = 50mEq $NaHCO_3$) mixed into 1L of D5W & infused at 150-250mL/hr → target urine pH 7.5-8.0 (urine dip stick to measure).
- Add 20-40mEq KCl to each liter of IV fluids if renal fxn allows as $\downarrow[K^+]$ counteracts the alkalinization process
- **Close monitoring:** salicylate levels and ABG frequently. Lytes and ext lytes.

PEARL:
***Intubation:** Mechanical Ventilation has difficulty generating a similar magnitude of hyperventilation in comparison to a conscious and adequately ventilating patient. The brief hypoventilation during intubation process, slow bagging, and ventilator-patient asynchrony can exacerbate the acidemia. If you do intubate, bag deep & fast.
****Urine alkalinization:** turns salicylate into an anion → prevents diffusion into CNS & tubular reabsorption
*****Chronic salicylate poisoning**: may have N serum levels though end organ manifestation.

Acetaminophen Overdose

Pharmacokinetics/dynamics: Absorption: ~100% absorbed in GI. Peak level: within 2hrs though delayed for extended-release. Vol. of distribution: 0.8-1L/kg. Metabolism: hepatic (90%) via conjugation mainly though some by CYP 2E1 & 1A2 which produces NAPQI (toxic metabolite), and renal. Elimination ½ Life: 2-4hrs though increase with hepatotoxicity. Excretion: renal.

Toxicity: ingestion >150 mg/kg

Clinical Manifestations: <24hrs: non-specific → 24-72hrs: RUQ pain, AbN labs → 72-96hrs: acute hepatic failure, thrombocytopenia → >5 days: partial clinical resolution of hepatotoxicity or progression to multi-organ failure

Investigations:
- Acetaminophen level (q2-4h until peak, then repeat q12h)
- Liver enzymes and synthetics.
- King's College Criteria (MDCalc)

Mx:

1. **Principles of intoxication Mx**
2. **GI decontamination:** activated charcoal 50g PO if <4h from ingestion
3. **Indication for N-acetylcysteine antidote as per Rumack-Matthew nomogram*:** Get Help from Poison Control
   (a) APAP level at ≥4th hr from ingestion is above "Tx line" on nomogram (If you know pt ingested >10g start before waiting 4hr)
   (b) Hx of APAP ingestion and evidence of hepatoxicity
   (c) Serum level >66umol/L and unknown time of ingestion
   (d) Delayed (>24hrs) presentation and hepatic injury**
4. **NAC Protocol:** Protocol for NAC infusion under "KGH Parenteral Pharmacy" website; or ask Poison Control to fax one over
   - **Anaphylactoid rxn (10%):** If rash & mild symptoms, hold infusion & Tx with Benadryl® 50mg IV + Ranitidine 150mg IV x1 ± methylprednisone 125mg IV x1. Restart at slower rate. If true anaphylactoid rxn, stop NAC & substitute with methionine (only effective if given in <10hrs from ingestion).
- Continue treating until APAP level undetectable, AST/ALT ≤100IU/L (or falling and <50% peak) and INR <2.0.

5. **Consider referral for liver transplantation (King's College Criteria)**
6. **Close monitoring:** APAP level and hepatic panel.

PEARLS:

* Nomogram: If pt has multiple ingestions, cannot use nomogram to determine if toxic.
**If patient presents late, serum levels may be undetectable, but they are still at risk for toxicity.

Ethanol Toxicity/Withdrawal (Acute)

Pharmacokinetics/dynamics: Absorption: rapid in upper GI. Peak level: <2hr. Vol. of distribution: 0.5L/kg. Metabolism: hepatic. Elimination: zero order kinetics for hepatic oxidation. Average ~7-10g/hr, but it varies significantly → EtOH naive: 3-4.5mmol/L/hr; Chronic use: 5.5-8mmol/L/hr.

Note: 0.6oz EtOH = 13.6g EtOH = 1 standard drink

Toxic Dose: >66mmol/L – coma & resp. depression (though higher levels may be tolerated in chronic use)

Withdrawal is a diagnosis of exclusion – exclude or treat comorbid illnesses

Stage	Timing	Symptoms and Signs
Withdrawal	Within 6hrs, lasting 24-48hrs	↑HR, ↑BP, tremulousness
Hallucinations	Within 12-24hrs, lasting 24-48hrs	Primarily visual hallucination (may have auditory/tactile) with NO altered MS
Seizures	Within 48hrs	Generalized tonic clonic seizure – usually brief in duration
Delirium Tremens	Within 48-96hrs, lasting 1-5 days	Marked confusion, agitation, hallucination, sympathetic overdrive

EtOH Withdrawal Mx:

1. **ABC.** Determine where to admit the patient based on withdrawal severity and likelihood of complications. CIWA >20 should be in a monitored setting – D4ICU/ICU.
2. **Benzodiazepines (BZD):** Cornerstone of treatment – Sedate and suppress the sympathetic drive without resulting in intubation if possible.
   CIWA Protocol: Validated withdrawal severity assessment tool whereby BZD is given depending on overall severity. 10 withdrawal features are assessed and scored routinely – max score 67 (severity: mild <15, moderate 16-20, severe >20). If total score ≥8, BZD is given. If total score is <8, no BZD is given.

BZD	Dose	Notes
Diazepam	IV: 10-20mg IV PO: 20mg PO	Rapid onset with IV though slow with PO/PR; Diazepam has long half-life (120 hours with active metabolites).
Lorazepam	IV: 2mg IV PO: 2-4mg PO	IV lorazepam is dissolved in propylene glycol. Hepatic metabolism does not result in active metabolites, ∴ safer with elderly & liver dysfunction.

*__Dosing:__ Be ready to give high doses of BZD. Patients who consume high quantities of EtOH chronically and/or in moderate to severe withdrawal are likely to require high doses of BZD for any meaningful tranquilizing effect. Note that sedation is a lesser "evil" than sequelae of severe withdrawal.

Opioid Toxicity

Clinical (Acute): Hallmark – Bradypnea, Miosis. ΔLOC – confusion, drowsy, stuporous. Seizure is not common.

Mx:

1. **Principles of intoxication Mx**
2. **Naloxone (Antidote):** indicated for ↓LOC or hypoventilation

- **OD in patients without severe pain syndromes:** Naloxone 0.4-2mg IV/IM/SC/intra-nasal q2-3min (start with 0.4mg) until max total of 10mg. Infusion: rate of infusion is 2/3 of initial effective bolus dose in mg/hr. A bolus of ½ the initial effective bolus should be given 15min after the continuous infusion has started.
- **OD in patients with severe pain syndromes:** Dilute 1mL of 0.4mg/mL Naloxone into 9mL NS → final concentration of 0.04mg/mL → give Naloxone 0.04-0.08mg IV push q30-60s until improvement (wary of precipitating acute pain crisis from reversing opioid analgesia).

PEARLS:

*Diagnosis can commonly be elucidated with Hx & Meds review (newly started, dose Δ, renal Fxn).

**Commonly seen in surgical patients, titrating opioid rapidly, and new AKI.

***If no reversal effect noted (especially for respiratory depression) with a total of 2mg naloxone given in rapid succession, consider another etiology.

Lithium Toxicity

Pharmacokinetic/dynamics: Peak serum concentration: <4hrs for immediate release (Extended release formulation and Lithium Carbonate form concretion in GI tract – delays peak [serum] substantially to days); Vol of Distribution: low, poor protein binding, VERY SLOW equilibration between extracellular & intracellular space; Excretion: renal though 80% excreted is actively reabsorbed in the tubules

Clinical Features: Acute, Acute-on-Chronic, Chronic: dissimilar pharmacokinetic and clinical presentation – elucidate

- **Acute:** usually non-specific GI symptoms; **Acute-on-chronic:** quick to develop neuro sequelae; **Chronic:** commonly due to AKI, usually presents with Neuro issues
- **Specifics: (a) Neuro: acute** – confusion, agitation, seizure, ataxia; **chronic** – coarse tremor, fasciculation, myoclonic jerks; **(b) Cardiac:** arrhythmia, AV block, ↑QTc, ST-T change; **(c) AKI**; **(d) -[Na^+]**

Mx:

1. **Principles of toxicity Mx**
2. **Indications for dialysis:** (a) Serum [Li^+] >5mmol/L; (b) Serum [Li^+] >4mmol/L with renal insufficiency; (c) Decreased LOC, sz, life threatening cardiac or neuro complications
3. **Hydration**: IV NS at high infusion rates - aggressive (2-3L/day)

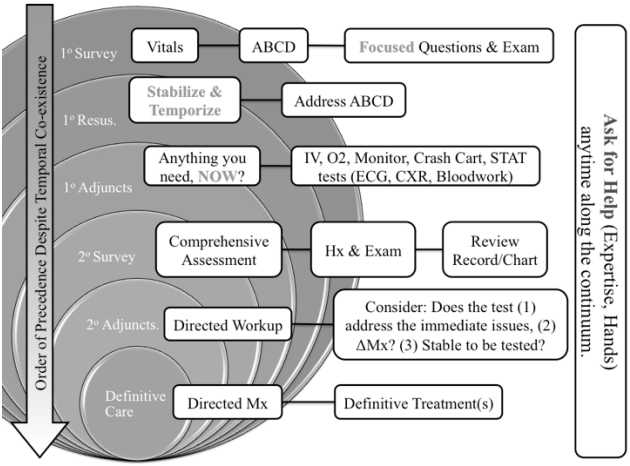
PEARLS:

*Serum [Li^+] DOES NOT correlate with tissue level; watch for rebound due to slow equilibration

General Approach to an Unstable Patient

Updated May 2024

1. **Recognition (Most important and often overlooked):** "Look Sick, Sound Sick, Are Sick until proven otherwise"
2. **Resource Evaluation:** Expertise, Equipment, Manpower (RACE Team), Setting (lack of bed in more monitored setting should not delay management, but important to alert Charge Nurse/Senior Resident/ICU to plan for accommodation)
3. **Get Help:** to address resource deficit – current and anticipated.
4. **Manage: Basic Framework when Approaching an Unstable Patient**



*__Choosing Wisely®:__ Not the time during acute

RACE (Rapid Assessment of Critical Event) Team

What do they do: If you are seeing a sick patient and need help, call. The team will come with a crash cart, medications, antibiotics, and experience.

Members:
- RACE Physician (during daytime) – acute situational expertise.
- Specially Trained Nurse – expertise with high acuity and familiar with the use of more advanced drugs.
- Respiratory Therapist

When to activate the RACE team:

Airway:
- Threatened
- Stridor
- Excessive secretions

Breathing:
- Resp Rate <8 or >30
- Distressed breathing
- O_2 sat <90 on >50% O_2 or 6L/min

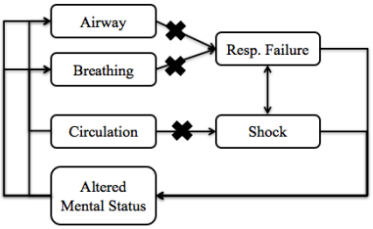
Circulation:
- sBP <90 or >200 (or a decrease of >40 mmHg)
- Heart rate <40 or >130

Disability:
- Decreased level of consciousness

Approach to Altered Mental Status

Overarching Schema

Altered Mental Status: characterize the "altered" in detail & compare to baseline via notes and individuals who know the patient

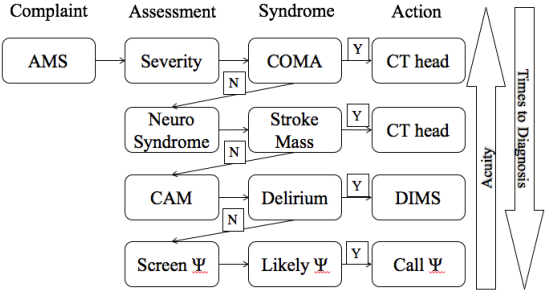


1. **1º Survey:** ABCD.
2. **1º Resus:** Fix ABC 1st as they kill and can cause AMS.
   - ABC: Whether the ABC problem is/are caused by or resulting in AMS, fix them 1st. Reasons: (1) They kill; (2) They cause AMS; (3) May reverse AMS.
   - Non-ABC: Hypoglycemia (D50W), Opiate (Naloxone), Benzo (flumazenil).
3. **Resource Evaluation:** Expertise, Equipment, Manpower – Get Help
4. **2º Survey:** DETAILED Hx, Exam, & Data Review
5. **2º Adjuncts (Workup)**

Etiology to Consider

- **Drug:** New, discontinued, dose Δ, Δ to metabolism, Δ to excretion, toxin.
- **Infection:** Focal source, sepsis.
- **Metabolic:** O_2, CO_2, temp, glucose (neuroglycopenia), electrolyte (Na^+, Ca^{+2}), pH, uremia, hepatic encephalopathy, SIRS (thyroid, Cushing's Syndrome, paraneoplastic).
- **Structural:** Stroke, seizure.

Algorithm



Pitfalls:

- **Hx Helps:** especially collateral history – CALL.
- **"Quick" Exam:** The etiology will be missed. Perform a detailed neuro exam, skin for ulcer (decubitus) and cellulitis.

Approach to Hypoxia and Dyspnea

Overarching Schema:

1. General Appearance? Look sick? Is sick until proven otherwise.

2. 1º Survey: ABCD Assessment

A. Stridor? (remove obstruction; anaphylaxis?) Gurgling? (suction)

B. Oxygen supplementation with 50%VM or NRM, then titrate down.

C. Hypotensive – resuscitate accordingly.

D. Awake & talking? Confused? Stuporous? (Tolerate Guedel/LMA + Bagging?)

3. Resource Evaluation: Expertise, Equipment, Manpower – Get Help!

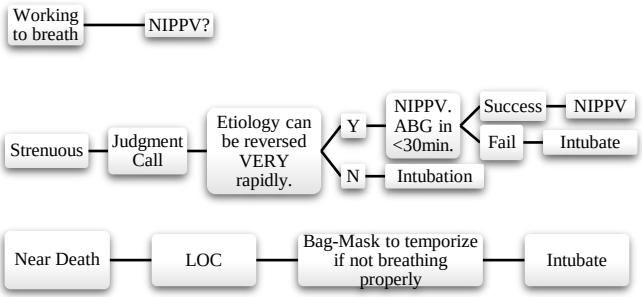
4. Severity: Quickly determine which category the patient fits in

Working to Breath
- **Appearance:** Effortful, but not in major distress >5 word sentence
- **Vitals:** ↑HR
- **Resp:** ↑RR, ↑tidal vol., ↓CO_2
- **Work:** Accessory muscles

Strenuous "Adrenergic driven"
- **Appearance:** Vigorously breathing, diaphoretic, <5 word sentence
- **Vitals:** ↑HR, ↑BP
- **Resp:** ↑↑↑RR, ↓/↑tidal vol., ↓CO_2
- **Work:** Accessory muscles, intercostal in-drawing, tripoding

Near Death "Tiring Out"
- **Appearance:** ↓LOC - drowsy, stuporous, "tired out"
- **Vitals:** ↓HR, ↓BP
- **Resp:** ↓RR, ↓tidal vol., ↓ breath sound intenisty, normal/↑CO_2
- **Work:** Ineffective to minimal effort

5. 1º Resus:



6. 2º Survey and Directed Work up: Investigations – Minimal
- Blood Work: CBC, -lytes + Cr, VBG (ABG ASAP when there are free hands), Trop
- Imaging: CXR (PoCUS if you able to employ)
- Auxiliary: ECG

7. Directed and Definitive Mx

Approach to Bradypnea

Overarching Schema:

1. **General Appearance?** ↓LOC? "Tired out" look? (2º to ↑ work of breathing?) Miosis? (think opioids)
2. **1º Survey:** ABCD Assessment
   - **A.** Tongue – obstructing? (oropharyngeal airway - OPA) Gurgling? (suction secretion) Protecting airway?
   - **B.** Cyanosis? Hypoxic? Shallow breathing? (↓ Tidal vol.)
   - **C.** Hypotensive – resuscitate accordingly
   - **D.** Drowsy? Stuporous? (Tolerate OPA/LMA + Bagging?)
3. **Resource Evaluation:** Expertise, Equipment, Manpower – Get Help
4. **Severity:** quickly determine which category does the patient fit

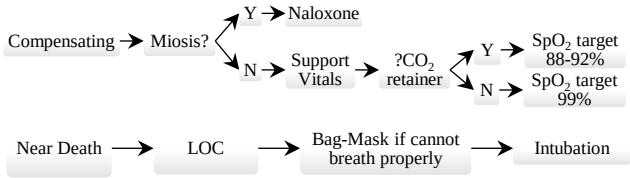
Compensating

- **Appearance:** ↓LOC
  Vitals: Hemodyanmically stable
- **Resp:** ↓RR, Tidal vol. ~normal, SpO_2 mildly ↓ to normal
- **Work:** Adequate tidal volume, but just slow to breath

Near Death "Tiring Out"

- **Appearance:** Stuporous, "tired out" - diaphoretic
- **Vitals:** ↓HR, ↓BP
- **Resp:** ↓RR, Tidal vol. ↓, ↓ breath sound intenisty
- **Work:** Ineffective to minimal effort

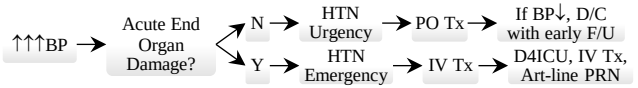
5. **1º Resus:**



6. **Directed Workup: Investigations – Minimal**
   - Blood Work: VBG (ABG ASAP when there are free hands) for ↑CO_2, -lytes + Cr,
   - Imaging: CXR (PoCUS if you able to employ)
   - Auxiliary: ECG
7. **Directed and Definitive Mx:**
   - **Opioid toxicity** (a common cause in hospital): naloxone for reversal
   - **Any cause of respiratory failure:** tiring out from work of breathing

Approach to Hypertensive Emergency

Overarching Schema:



Approach: 3 Questions to Answer

1. Is there Acute End-Organ Damage?
- Neuro: visual Δ, focal deficit, convulsion, coma; Intracranial bleed
- Cardiovascular: aortic dissection, ACS, HF
- Retinopathy: flame hemorrhage, papilledema
- Nephropathy: AKI, hematuria/proteinuria
- Investigations: CBC, Cr, urine analysis, ECG, CXR, CT head PRN

2. What is the BP target?
- Aortic dissection: ↓ SBP<120
- Ischemic stroke: No thrombolysis: use anti-HTN only if >220/120; For thrombolysis ↓ <185/<110 PRIOR to lysis
- Intracranial hemorrhage: SBP<140-160

3. How Quick to ↓BP?
- Generally: 1st hr of Tx ↓ MAP <15% (lest risk of hypoperfusion) → ↓ MAP by ~25% in next 24-48hrs
- Aortic dissection: ASAP to target

IV Medications:

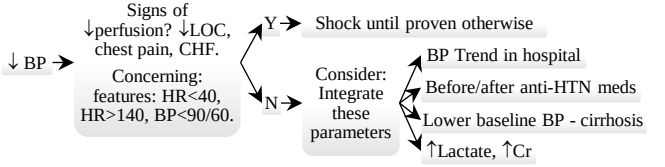
Hydralazine 10-40mg q4h	Onset 5-20min; Last 1-4hrs	Avoid in HTN encephalopathy
Labetalol Bolus 10-20mg q10min **Labetalol Infusion** 1-8mg/min (start at 2mg/min)	Onset 2-5min; Last 2-18hrs	**POTENT**; Avoid in acute HF; Has little chronotropic effect
Nitroglycerin start 5mcg/min (Max 200mcg/min)	Onset 1min; Last 2-5min	Easy to titrate; Reflex ↑HR.
Nitroprusside 0.25-10mcg/kg/min (>3mcg/kg/min for days – check thiocyanate)	Onset 1-2min; Last 1-10min	**POTENT**; Wary of cyanide, thiocyanate tox; C/I in pregnancy

Drug of Choice: As per your comfort though some for specific conditions.
- **Stroke (ischemic/hemorrhagic):** labetalol, hydralazine. Avoid nitroprusside & NTG – venodilators can ↑ICP + ↓cerebral perfusion.
- **Aortic dissection:** labetalol, nitroprusside
- **Acute MI:** NTG
- **Pulmonary edema:** NTG
- **Scleroderma renal crisis:** Enalaprilat IV

Approach to Shock

Definitions:
Hypotension – BP <90/60 or 30 mmHg drop in SBP from baseline
Shock – inadequate perfusion of and oxygen delivery to tissues
Hypoxemia – decreased oxygen in blood
Hypoxia – decreased oxygen in tissues

Overarching Schema:



1. **General Appearance?** ↓LOC, Chest pain → end-organ ↓ perfusion
2. **Survey:** ABCD Assessment
3. **Resource Evaluation:** Expertise, Equipment, Manpower – Get Help
4. **1º Resus:** NS 1L IV bolus ASAP – wide open with pressure bag around.
5. **1º Adjuncts:** IV access – large bore peripheral IV. If nil, intra-osseous access.
6. **2º Survey and Directed Workup:** Elucidate the shock mechanism – Focused Physical Exam 1st (& PoCUS) → Order ECG ± CXR → Hx: Focused. Passive leg raise to check if volume responsive.
7. **Directed and Definitive Mx**

Categories of Shock: Definitions and Examples

Hypovolemic	Decreased intravascular blood volume → decreased preload → diminished stroke volume
Distributive	"Relative" hypovolemia from excessive vasodilation + microvascular dysfunction (ie, sepsis, anaphylaxis, spinal injury, drugs)
Obstructive	Impairment of venous return to right atrium (ie, tension pneumothorax, PE, pericardial tamponade)
Cardiogenic	Primary cardiac insult causes decreased cardiac output (ie, heart failure, ACS, valvular heart disease, obstructive causes of shock)
Neurogenic	Form of distributive shock; may have hypotension AND bradycardia

Categories of Shock: Fundamental Mechanisms

	Hypovol	**Distributive**	**Cardiogenic**	**Obstructive**
Periphery	Cool	Warm	Cool	Cool
Pul Edema	No	Yes/No	Yes	No
JVP	↓	↑/↓	↑	↑

Classes of Hypovolemic Shock:

Class	**I**	**II**	**III**	**IV**
Blood Loss	0-15%	15-30%	30-40%	>40%
BP	N	N	Decreased	Decreased
HR	N	>100	>120	>140
RR	N	20-30	30-40	>35
CNS	N to Anxious	Anxious	Confused	Lethargic
Urine Output (ml/hr)	N	20-30	5-15	Negligible

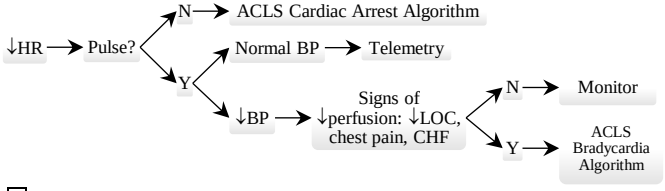
Directed Mx: based on mechanism

Mechanism	**Goal of resus: ↑O_2 delivery** = ↑CO (cardiac output) x ↑CaO_2 (arterial O_2 content)
Hypovolemic	(1) Volume Expand: Crystalloid (and/or pRBC)

Distributive	(1) Vasopressor	
Cardiogenic (refers to cardiogenic shock 2º causes intrinsic to the ♥)	**Arrhythmia**	Unstable ↑/↓HR → ACLS
	ACS	Vasopressor + Inotropic (CALL FOR HELP)
	Valve	Vasopressor + Inotropic (CALL FOR HELP)
Obstructive (refers to cardiogenic shock 2º to causes extrinsic to the heart → usually results in ↓ LV preload)	(1) Volume Expand: once satisfied with response or if no response, stop fluid (PE: interventricular dependence can paradoxically ↓ BP with too much fluid). (2) Inotropic (CALL FOR HELP)	

Approach to Bradycardia

Overarching Schema:



1. **General Appearance?** ↓LOC? Chest pain? Dyspnea?
2. **1º Survey:** ABCD Assessment – signs of hypoperfusion
   - **A.** Tongue obstructing? (OPA) Gurgling? (suction secretion)
   - **B.** Cyanosis? Hypoxic? Signs of CHF?
   - **C.** Hypotensive?
   - **D.** Drowsy? Stuporous? (Tolerate OPA/LMA + Bagging?)
3. **Resource Evaluation:** If unstable, get the crash cart, place the pads on the patient. Call Code 99 and inform your senior.
4. **1º Resus:**
   - **See ACLS Algorithm for Bradycardia** (includes pharmacological options like atropine, dopamine infusion, or epinephrine infusion, as well as transcutaneous pacing)
5. **1º Adjuncts:** IV access.
6. **2º Survey and Directed Workup:**
   - ECG ASAP: Mobitz II 2º HB, 3º HB, ischemia
   - Lytes, Cr, Ca, Mg, P, Trop, CK, Lactate
   - Meds review: amiodarone, β-blocker, CCB (diltiazem), digoxin
   - Hx: Active ischemia?
   - **Consult Cardio** for further Mx. Indication for invasive pacing device?
7. **Directed and Definitive Mx**

Notes on Transcutaneous Pacer Function:

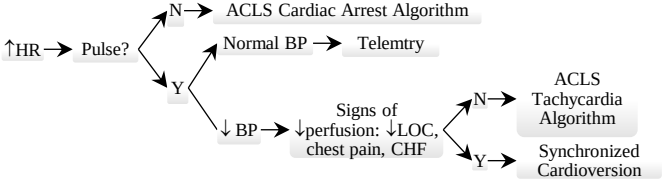
- Achieve both **electrical capture** (paced QRS complex visible) & **mechanical capture** (corresponding pulse) → signs of ↑ cardiac output (raised BP, ↑LOC).
- **Capturing current:** usually 60-100mA suffices to electrically capture the myocardium with transcutaneous pacing. However, during the initial setup, ↑ the

current (mA) to a higher magnitude to 1st successfully achieve electrical (& mechanical) capture, then slowly dial down in tandem of 10mA to find the minimal current necessary to achieve capture. The ideal setting is ~1.25x the minimal current required for captures.

- **Discomfort:** transcutaneous pacing will result in skeletal muscle contraction – can be quite uncomfortable. Sedation and analgesics may be required (ie, Midazolam 2mg IV x1, Fentanyl 25-50mcg IV x1).

Approach to Tachycardia

Overarching Schema:



1. General Appearance? ↓LOC (hypoperfusion)? Chest pain?

2. 1º Survey: ABCD Assessment – signs of hypoperfusion

A. Tongue obstructing? (OPA) Gurgling? (suction secretion) Protecting airway?
B. Cyanosis? Hypoxic?
C. Hypotension?
D. Hypotensive?
E. Drowsy? Stuporous? (Tolerate OPA/LMA + Bagging?)

3. Resource Evaluation: If unstable, get the crash cart, place the pads on the patient. Call Code 99 and inform your senior.

4. 1º Resus:

- Crash cart: Apply pads. Shock Mode. Rhythm: (1) QRS Wide or Narrow? (2) Regular or Irregular?
- **See ACLS Algorithm for Tachycardia**

5. 1º Adjuncts: IV access.

6. 2º Survey and Directed Workup:

- ECG ASAP, ischemic changes
- Lytes, Cr, Ca, Mg, P, Trop, CK, Lactate
- **Consult Cardio** for further Mx.

7. Directed and Definitive Mx

Pearl: Nodal blockade maneuvers may have (1) no effect, (2) transient effect – slows the ventricular rhythm, (3) terminate the SVT.

A Primer on Post-Intensive Care Syndrome (PICS)

Definition:

Constitutes new or worsening function in >1 of cognitive, psychiatric, or physical function after critical illness.

Domain	Risk Factors	Clinical Manifestations	Diagnosis	Treatment	Prevention
Cognitive	Delirium, prior cognitive deficit, sepsis, ARDS, shock, acute brain insult	Poor attention, memory, processing speed, executive function	MoCA, MMSE, Mini-Cog	Cognitive exercises, memory aids; possible role for pharmacotherapy	**A**wakening and **B**reathing **C**oordination with daily sedative interruption **D**elirium monitoring **E**arly ambulation **F**amily empowerment ***ABCDEF approach**
Psych	Preexisting anxiety/depression/PTSD, cognitive insults	Anxiety, depression, PTSD, sexual dysfunction	Short Form-26 Scale, Hospital Anxiety and Depression Scale, Impact of Events Scale	CBT, counselling/interpersonal interventions, Pharmacotherapy	
Physical	Preexisting functional disability, frailty, prolonged mechanical ventilation	ICU-acquired weakness including poor mobility, falls, paralysis	Assessment by PT/OT/dietitian	Multidisciplinary programs with PT/OT	

*important to rule out alternative causes of symptoms/mimickers of PICS (i.e. delirium, stroke, etc.)

Outcomes:

Health-related quality of life is significantly lower in survivors of critical illness/their family members. There may be mild improvements in cognitive, psychiatric, and physical impairments within 12 months of discharge. There is an elevated risk of death in patients after critical illness, particularly in the first 3-6 months after ICU admission.

Common Overnight Pages - QIM

Authors: Dr. Priscilla Matthews, Dr. Stephen Gauthier *May 2024*

Low Blood Pressure

1. SBP < 90, MAP < 60, decrease > 40 in SBP or >30% from patient's baseline MAP
2. Important to place in the context of individual patient
3. Assess the following 4 aspects:
- **A.** HR → ECG
- **B.** Volume status → Physical exam & US for IVC/LV volume
  - DDx = hypovolemia, hemorrhage, tamponade, massive PE, new RV infarction, tension PTX, air trapping
- **C.** Cardiac function → ECG & US for LV function
- **D.** Systemic vascular resistance → peripheral vasodilation
  - DDx = cervical spinal cord compression, anaphylaxis, fulminant liver failure, sepsis

Pearls:
- Ask for repeat blood pressure within 15-30 minutes and see if it is still low
- Treat the patient, not the # (but take all low BPs seriously)
- Consider the patient's average BP and how the hypotensive value compares
- If the life-threatening causes are ruled out, consider other benign causes, i.e., the patient may have been lying down/sleeping
- If the patient has good cardiac function and has features consistent with volume depletion, consider giving a fluid bolus and reassessing their blood pressure

Managing a Code – The Basics

1. Know the differences at KHSC:
- **A.** Code BLUE = cardiac arrest
- **B.** Code 99 = medical emergency other than cardiac arrest (i.e., collapse, injury, etc.)

2. When a code is called for a medicine patient, the D4, RT, and sometimes RACE staff will attend
3. Use the ABCDE IV
- **A.** Airway → assess, manage, and anticipate airway complications
  - Can the patient talk?
  - Position patient appropriately such that the airway can be protected with head-tilt chin-lift maneuver
  - If patient not protecting airway (i.e., intoxicated, seizing, etc.), recognize the need for intubation
- **B.** Breathing → ensure adequate ventilation & oxygenation
  - Ensure that RT can come
  - Start the patient on appropriate oxygen delivery method (nasal prongs, non-rebreather mask, Venturi mask, etc.)
  - If they aren't doing well with these or on max delivery methods (i.e., 60% FiO2 on a Venturi mask), consider D4 admission +/- ICU consultation
- **C.** Circulation → assess for hemodynamic stability and volume status
  - If patient is hypotensive/in shock, try to figure out what type as this is critical to guide management

- Crystalloid (but be cautious → fluids can push a patient into volume overload)
- Does the patient need pressors? → D4/ICU

D. Disability
- Evaluate their neurological status, manage the decreased LoC (again based on the underlying cause, i.e., increased ICP, intoxication, hypoglycemia, etc.)

E. Environment
- Screen for clues to identify underlying etiology
- Examine the body → signs of infection?
- Temperature
- Remove inciting/aggravating items

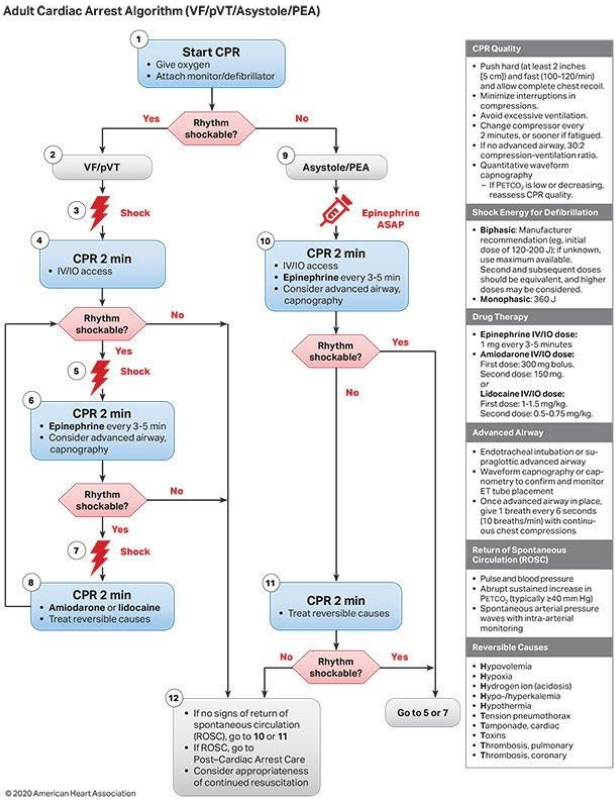
F. IV access/IO access if unable to obtain IV access - 2 large bore IVs (one for meds, one for bloodwork)
- Draw bloodwork immediately (general workup - CBC, electrolytes + extended electrolytes, glucose, creatinine/urea, VBG + lactate, liver panel, coagulation panel, BNP/troponin, CRP/ESR, blood cultures)

G. Other investigations
- ECG
- PoCUS can be very helpful!
- CXR
- Urine studies, bladder scan
- CT (depending on what your clinical suspicion is)

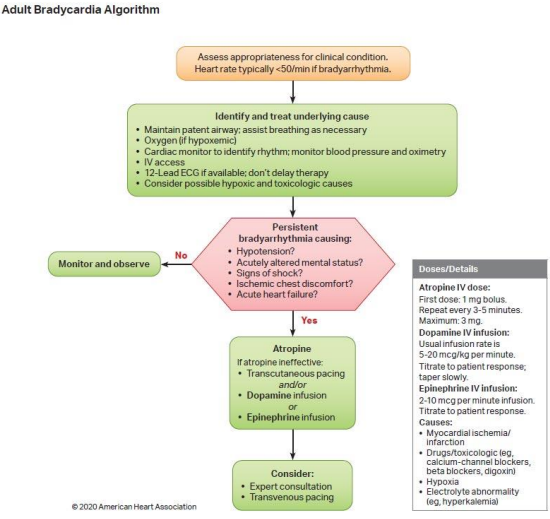
H. Other tips for running a code:
- Delegate tasks when appropriate
- When senior staff/residents come, it's helpful to give them a brief summary of the patient (why they were admitted, recent events (i.e., med changes or position changes), events leading up to current situation, and what you've done so far)

ACLS Algorithms

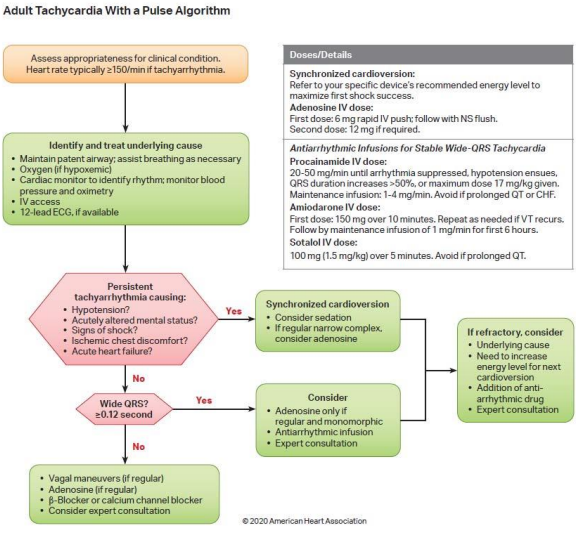
Cardiac arrest



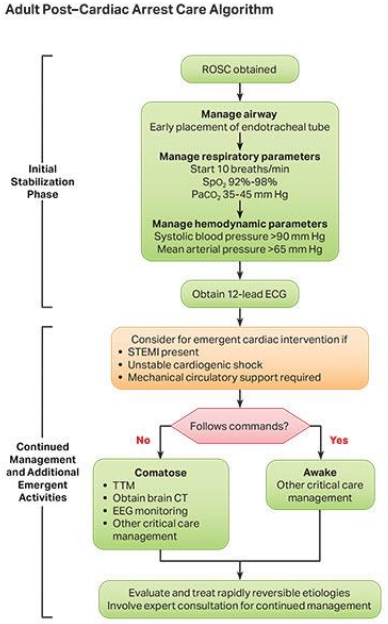
Bradycardia



Tachycardia with a pulse



ROSC and Post-Cardiac Arrest Care



Initial Stabilization Phase

Resuscitation is ongoing during the post-ROSC phase, and many of these activities can occur concurrently. However, if prioritization is necessary, follow these steps:

- Airway management: Waveform capnography or capnometry to confirm and monitor endotracheal tube placement
- Manage respiratory parameters: Titrate FiO_2 for SpO_2 92%-98%; start at 10 breaths/min; titrate to $PaCO_2$ of 35-45 mm Hg
- Manage hemodynamic parameters: Administer crystalloid and/or vasopressor or inotrope for goal systolic blood pressure >90 mm Hg or mean arterial pressure >65 mm Hg

Continued Management and Additional Emergent Activities

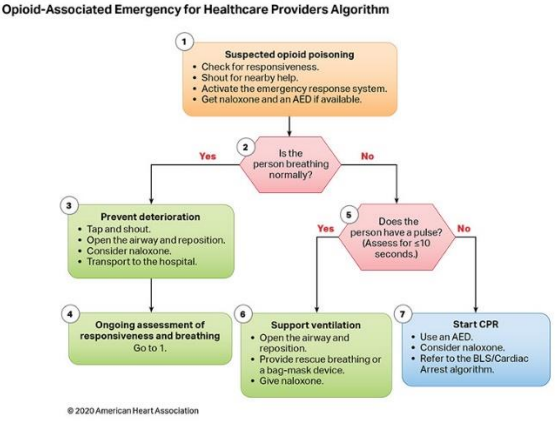
These evaluations should be done concurrently so that decisions on targeted temperature management (TTM) receive high priority as cardiac interventions.

- Emergent cardiac intervention: Early evaluation of 12-lead electrocardiogram (ECG); consider hemodynamics for decision on cardiac intervention
- TTM: If patient is not following commands, start TTM as soon as possible; begin at 32-36°C for 24 hours by using a cooling device with feedback loop
- Other critical care management
  - Continuously monitor core temperature (esophageal, rectal, bladder)
  - Maintain normoxia, normocapnia, euglycemia
  - Provide continuous or intermittent electroencephalogram (EEG) monitoring
  - Provide lung-protective ventilation

H's and T's

Hypovolemia
Hypoxia
Hydrogen ion (acidosis)
Hypokalemia/**h**yperkalemia
Hypothermia
Tension pneumothorax
Tamponade, cardiac
Toxins
Thrombosis, pulmonary
Thrombosis, coronary

Opioid-Associated Emergencies



Transferring a Patient to the Ward and Admissions to Subspecialty Services

Transfer to ward from D4/ICU:

- Assess the patient and document a focused H&P
  - History - What was their course so far and the period immediately prior to transfer? What are their current symptoms?
  - Physical Exam - Vitals (stable vs. unstable), relevant physical exam maneuvers
  - Investigations - Recent labs and/or imaging that is relevant and needs to be acted on
  - Plan - Usually orders are pre-specified in the D4/K2 ICU order set and you can sign off on them; however, you are ultimately responsible for these orders so make sure to review them. Changes would include:
    - Frequency of vitals monitoring (change from q1-2h → q4h) and labs
    - Restarting certain medications, i.e., sleep aids, bowel prep, nausea meds
  - If you are uncertain about anything, contact your D4 resident

Admissions to subspecialty service:

- Similar to doing an ER consult. It's good to ask the resident handing over to you if there are any expected overnight admissions so you're aware in advance. Common services that have overnight admissions include Cardiology and Heme-Onc.
- History:
  - PMHx, medications, relevant social history
  - HPI relevant to the service patient is being admitted to
- Physical Exam - Vitals (stable vs. unstable), complete physical exam with attention to reason for admission
- Investigations

- Check Connecting Ontario or PCS for labs/ imaging/ pathology/ micro results

- Plan
  - Do the relevant admission orders (i.e., for a Heme-Onc patient, the Oncology Inpatient Order Set on EntryPoint)
  - This is just like admitting a patient to medicine but be aware of service-specific requirements and medication adjustments
  - Review with the fellow/subspecialty senior resident
  - The patient is under your care so make sure to follow up on pertinent investigations

Leaving Against Medical Advice (AMA)

1. Review the patient's chart and ensure they don't have a condition that impairs his/her capacity to make decisions (i.e., psych issues, encephalopathy, delirium)
2. Talk to the patient. Most patients want to leave because of:
- **A.** Dissatisfaction with their care
- **B.** Dissatisfaction with staff caring for them
- **C.** Feeling uninvolved or out of the loop
- **D.** Inadequate symptom management
- **E.** Personal problems

3. Attempt to answer questions directly about their diagnosis/management plan and alleviate concerns/symptoms
4. If the patient is capable and wants to leave, explain the consequences and ensure they understand the risks. Make sure to document everything well!
5. If they decide to leave, make sure you consider if there is anything you can do to reduce the risk of a bad outcome (i.e., give them a prescription for antibiotics, refer them for follow up).
6. There is an AMA form that should be signed; however, it is critical that you also document the discussion you had and your impression related to 1) capacity, and 2) that they understand and appreciate the potential consequences of their decision.
7. Let your D4/senior know and hand this information over in the morning.

Agitated/Aggressive Patient

1. This is a very common page overnight!
2. Information to obtain from nursing:
- **A.** Acuity: new vs ongoing
- **B.** Is the patient altered or presenting disorganized thinking (i.e., more in keeping with delirium)
- **C.** Vitals
- **D.** Physical violence or harm to self/others
- **E.** Recently administered medications

3. Address factors that might be contributing including:
- **A.** Vitals
- **B.** Pain, constipation, nausea
- **C.** Cardiorespiratory status, abdominal exam
- **D.** Delirium workup if appropriate

4. Non-pharmacological interventions
- **A.** Reorient and target stimuli causing discomfort (i.e., pain/ constipation/ nausea)
- **B.** Involve known faces if possible

C. De-escalation techniques

5. Pharmacological options (chemical restraints)

A. Limit to when it's absolutely necessary, and a short term option

B. Patient tolerating oral intake:

- 1st gen antipsychotic - can be used in undifferentiated agitation, violent patients needing immediate sedation
  - Haloperidol 0.5/1mg PO
- 2nd gen antipsychotics - beneficial in patients with known psychotic disorder
  - Seroquel 12.5mg/25mg PO
  - Risperidone 0.5/1mg PO
  - Olanzapine 2.5/5mg PO
- Benzodiazepine - good for agitation related to drug intox/withdrawal or violent patient needing immediate sedation
  - Ativan 0.5-1mg SL

C. Patient not tolerating oral intake:

- Ativan 0.5mg IM

D. Make sure to check ECG before prescribing antipsychotics that could prolong QTc

E. We tend to stay away from benzodiazepines especially in the elderly population for their risk of diminishing respiratory drive and inducing delirium. Preferably start with 1st gen antipsychotic in geriatric patients.

6. Physical restraints

A. These are medical grade restraints that attach to the patient at 4-5 points

B. **Last resort option, have a high threshold**

C. Posey mitts are a softer physical restraint option when the patient is pulling out IVs/lines

Insomnia

1. This is a very common page overnight!

2. Figure out **why** the patient can't sleep – pain, delirium, withdrawal of home med, etc. – and address these underlying causes first

3. Try to ensure the best possible sleep hygiene in hospital although that isn't always possible in a busy, noisy environment (avoid caffeine/ CNS stimulants in the evening, take meds causing insomnia in the morning, give earplugs, limit vital checks while sleeping if not medically necessary, etc.)

4. If the patient needs a sleep aid, consider some of the following agents:

5. Great first line option and well tolerated by most:

- Melatonin 3-10mg po qhs

- Sleep maintenance:
  - Zopiclone 3.75-7.5mg po qhs (consider avoiding in elderly)
- Insomnia in older adults/with chronic pain:
  - Trazodone 25-100mg po qhs (not first line though! consider when other indications are present)
- Other options:
  - Mirtazapine 7.5-15mg po qhs (not first line though! consider when other indications are present)

6. Reasons to avoid benzos for sleep: older age, cognitive dysfunction, opioid use, addictive properties, and increased risk of delirium

Low Urine Output (Oliguria)

1. What is oliguria? Defined as < 400 mL in 24h or < 20 mL per hour
2. Etiology:
   - **A.** AKI/CKD
   - **B.** Pre-renal (most common) - hypovolemia, third-spacing, renal artery stenosis
   - **C.** Renal - ATN, systemic disorders
   - **D.** Post-renal - BPH/other prostatic disorders, tumors, kidney stones, foley catheter obstruction
3. Approach:
   - **A.** Further assessment: acuity, duration, associated symptoms, fluid status, meds review, pain, constipation, recent labs (i.e., Cr, urea), volume status, urine output (ins & outs), mental status, orthostatic vitals
   - **B.** If > 200 mL → in and out catheter to drain urine
   - **C.** If > 400 mL → insert foley catheter
     - If nursing having trouble with foley catheter, call urology
   - **D.** If patient already has foley, ensure it is draining and ask nursing to flush to confirm patency
   - **E.** Volume status = dry → fluids
   - **F.** Volume status = intravascularly overloaded → investigate for underlying cause (i.e., cardiorenal, renal failure, etc.) and consider whether diuresis would help
   - **G.** Consider stopping contributory medications (i.e., anticholinergics), treating other causes (i.e., constipation)

Difficulty Obtaining IV Access

1. A lot of times, nursing staff will call us because they are unable to get IV access or the IV continuously goes interstitial
2. First, get nursing to try again if they've only tried 1-2x
3. If still struggling, assess whether IV access is imminently needed?
   - **A.** Is the patient unstable? Medications can only be delivered intravenously? Cannot take medications orally?
   - **B.** If IV access is not imminently needed → translate all IV meds to oral
4. IV access imminently needed → call the following people who might be able to help:
   - **A.** Anesthesia resident
   - **B.** RACE nurse

New Rash

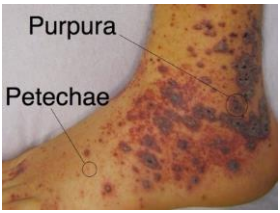
1. Assess whether the rash is life-threatening or not. Key information includes:
   - **A.** Is the patient hemodynamically stable?
   - **B.** When/ where is the rash? Is it spreading or localized?
   - **C.** How fast did it develop?
   - **D.** Were there recent exposures?
2. Red flag appearance of rashes:
   - **A.** Fever
   - **B.** Toxic appearance
   - **C.** Hypotension
   - **D.** Mucosal involvement

E. Severe pain
F. Elderly, immunosuppressed, or new medication

3. Classify the rash as described below:

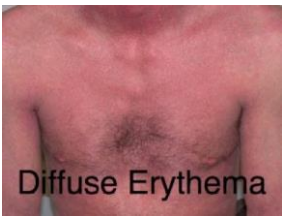
Petechial / purpureal rash:
Description:
1. Petechiae = small, red lesions caused when capillaries leak; purpura = petechiae > 0.5cm
2. Non-blanching
3. Usually start in dependent areas (i.e., legs)
4. Palpable → vasculitis/infection; non-palpable → low platelets (DIC, ITP)

Approach:
1. Palpable → Meningococcemia, DIC, endocarditis
2. Non-palpable → DIC, TTP, purpura fulminans



Erythematous rashes:
Description:
1. Diffuse red skin from capillary congestion secondary to inflammatory/ infectious conditions

Approach:
1. Assess for sloughing (Nikolsky's sign, i.e., "popping the blister")
A. Positive → think about TEN
B. Negative → think about anaphylaxis



Maculopapular rashes:
Description:
1. Most common type with broadest ddx. Combination of 2 types of lesions: macules (flat, red splotches) + papules (solid, raised lesions)

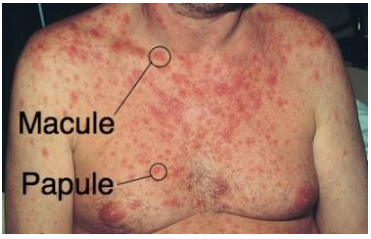
Approach:

1. Central vs peripheral distribution?

- **A.** Central + toxic appearing/ fever → erythema migrans (Lyme dz), viral exanthem
- **B.** Central + non-toxic → drug reaction
  - Stop the agent!
- **C.** Peripheral + toxic appearing
  - Target lesions? (cutaneous eruption of well-marked, round, erythematous macules, papules, vesicles, and/or bullae)
    - Yes → Consider SJS or erythema multiforme
    - No → meningococcemia, syphilis, erythema migrans
- **D.** Peripheral + non-toxic → psoriasis, eczema, scabies

2. SJS and erythema multiforme

- **A.** EM → usually self-limited but if it starts involving mucous membranes can be life-threatening
- **B.** SJS (<10% BSA; TEN >30% BSA) → mucocutaneous lesion often triggered by medications



Vesiculobullous rashes:

Description:

1. Fluid-filled lesions with small vesicles (<1cm) or bullae (>1cm)

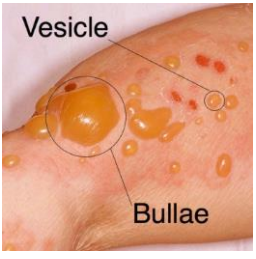
Approach:

1. Central Febrile with diffuse distribution → varicella, disseminated gonococcal dz, purpura fulminans, DIC

2. Febrile with localized distribution → necrotizing fasciitis, hand/foot/mouth disease

3. Afebrile with diffuse distribution → bullous pemphigoid, pemphigus vulgaris

4. Afebrile with localized distribution → contact dermatitis, herpes zoster, burns



Culturally Safe and Sensitive Care

Authors: Andrew McNaughton, Justin Boyle

Culturally safe and sensitive care is fundamental for providing patient-centred care. Healthcare practitioners must understand how their values, perspectives, beliefs, and biases can impact their ability to care for patients.

Health Equity is achieved when people are able to reach their full health potential and receive high-quality care that is fair and appropriate to them and their needs, no matter where they live, what they have, or who they are (Health Quality Ontario).

What is **cultural safety/sensitivity**?

- The concept of cultural safety has its origins with the Maori People (NZ).
- **Cultural safety** refers to a state whereby a provider embraces the skill of self-reflection as a means to advancing a therapeutic encounter with First Nations, Inuit, Métis peoples and other communities including but not limited to visible minorities, gay, lesbian, transgendered communities, and people living with challenges. Self-reflection in this case is underpinned by an understanding of power differentials (IPAC-AFMC, 2009).
- **Cultural competence** is the ability of healthcare providers to self-reflect on their own cultural values and how these impact the way care is provided. It includes each HCP's ability to assess and respect the values, attitudes, and beliefs of persons from other cultures and to respond appropriately when planning, implementing and evaluating a plan of care that incorporates health-related beliefs and cultural values, knowledge of disease incidence and prevalence, and treatment efficacy (Lavizzo-Mourey & MacKenzie, 1996).
- **Cultural safety** is the **goal** and outcome of **practicing** in a **culturally competent environment**.
- Cultural safety surpasses cultural sensitivity, which recognizes the importance of respecting difference. Cultural safety is predicated on the understanding of power differentials inherent in health-service delivery and redressing these inequities through educational processes (CINA, 2011, p. 2).
- Patients define what culturally safe means to them and how their cultural location, beliefs, and values are or were considered.
- Medicine has an obligation to respond to the effects of past and present colonial ideology embedded in the systems in Canada (such as the educational, political and health-care systems) that affect Canada's Indigenous peoples.

The Practice Environment

- There are physical, social, cultural, relational and systemic barriers to health and accessibility before, during and after encounters with the health-care system and health-care providers.
- There are a variety of practice environments, including on- and off-reserve, community health centres, nursing stations, urban and rural, and primary, secondary and tertiary health care.
- The environment supports a collaborative approach and the development of reciprocal relationships with the family and interprofessional team that are respectful regardless of professional beliefs and values.
- Health is interconnected with the environment (e.g., health and well-being of the land, access to healthy foods, clean drinking water).

The Health Status of Indigenous Individuals in Canada

- Health inequities and inequalities are shaped by history and ongoing systemic racism and have a profound impact on Indigenous peoples' health.

- The distal determinants of health (colonialism, racism, self-determination, and social exclusion) account for the oppressive historical and contemporary context in Canada and have resulted in direct impacts on the health of Indigenous peoples.
- Historical trauma has intergenerational impacts on the health and well-being of Indigenous peoples.
- Some legislation and health policies have a direct impact on the health and well-being of Indigenous peoples (e.g. the Indian Act specifies who is considered to have First Nations status and non-status. Status determines access to federal versus provincial services).
- Indigenous women carry a greater burden for health and social disparities.
- Culture provides protective factors in the health of Indigenous peoples.
- To provide culturally competent and culturally safe care in Canada, it is imperative that the Truth and Reconciliation Commission of Canada's Calls to Action for Health (18, 19, 21, 22, 23, 24) are addressed.

How can we be culturally safe and sensitive in the care we offer patients?

- Reflective Practice
- Allyship
- Participating in cultural safety/sensitivity training
- Institutional Advocacy
- Medical Education

Resources:

- San'yas Indigenous Cultural Safety Training
- https://sanyas.ca/
- https://www.queensu.ca/hreo/education
- https://www.cna-aiic.ca/-/media/cna/page-content/pdf-en/cultural-competence-and-safety-competencies.pdf?la=en&hash=B133D82B2F659E65CC171B9F41253DBF8BFEBA52
- The "paradox of well-intentioned physicians providing inequitable care. (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4169280/)

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