Vade Mecum 2023

"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 #
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.

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

Death of a Patient

1. What Happened:
- Check code status, was death expected, what happened & who was present

2. Enter the Room:
- Introduce yourself, convey your regrets see if there is anything you can do
- Explain you need to pronounce the patient

3. Pronouncing:
- Check pulses and listen for heart sounds for 1 min, listen for breath sounds for 3 min, check neurological activity (sternal rub, corneal reflex, pupil reactivity)
- Note the Time of Death (pronouncing time)
- Determine if coroner is required

4. After Leaving the Room:
- Fill the Death Certificate
- Call the family if none present.
- Offer to perform an autopsy

Allergy

Author: Dr. Sherwin Wong *Updated June 2020*

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

History: As detailed as possible from the patient and collateral

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 • Facial edema, sneezing, conjunctivitis, rhinorrhea • Hoarseness or stridor • Wheezing + coughing	• Transient warmth / chills • Nausea / vomiting • 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:

IV crystalloid administration for those with GFR <60mL/min
  - 0.9% NaCl at 1mg/kg/hr 12hrs pre and 12 hrs post-procedure OR
- hold nephrotoxics on day of procedure and restart if Cr stable after 48 hours

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.

1. 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

Cardiology

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

Updated June 2021

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 3º) 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:

- 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
- ECHO: Cardiac function, wall motion abnormalities, valvular lesion
- Non-Invasive Tests: stress test/echo, nuclear scans, CT coronary angiography
- CAD Risk Factors: hypertension, dyslipidemia, diabetes, smoking, family history (1st degree males < 55, females < 65), sedentary lifestyle, inflammatory disease
- Calculate TIMI or GRACE score for risk-stratification and management plan. **TIMI ≥ 2** or **GRACE > 140** 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.

   * Clopidogrel > ticagrelor **if**: needing anticoagulation (e.g. A fib, LV thrombus), bleeding risk, thrombolysis, no drug coverage
   * HOLD P2Y12 inhibitor if awaiting CABG, continue ASA.

   b. **Anticoagulant:** One of the following:

Unfractionated Heparin REDUCED dose nomogram 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)	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** (Speak to the most senior person: Cardio fellow/staff), Senior Resident, RACE.
4. **Medications ASAP**: Aspirin load + Ticagrelor/Plavix load + heparin + nitrates/Anti-anginal (***see. Acute Mx of NSTE-ACS***)
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 continue 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:

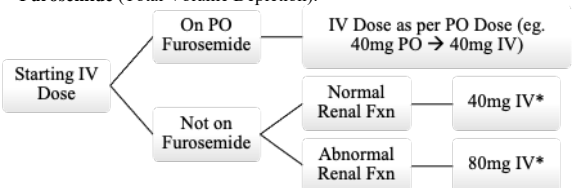
ϒ **NYHA Class:** Past and Present? Class I (ø Symptom), Class II (Slight limitation of ordinary activity), Class III (Marked limitation with min exertion) → Class IV (Symptom at rest or with any exertion)
ϒ **Type and Cause?** HFrEF (reduced EF< 40%), HFmEF (mid-range EF 41-49&) or HFpEF (preserved 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 (**relative contraindication): NIPPV, mechanical 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 inotropic 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 2days.
4. **Smoking/EtOH cessation**
5. **Flu/pneumococcal vaccination**

Chronic Mx – Pharmacological:

▪ **HFrEF < 40%**

1. Triple therapy as tolerated, including **ARNI (now first line for new diagnosis HFrEF) or ACEi/ARB + beta-blocker + MRA**

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. SGLT2 inhibitor: recommended in all patients with HFrEF

4. If sinus rhythm and HR > 70 on maximal BB therapy: consider adding **ivabradine**

5. Hydralazine/ISDN: recommended in HFrEF who cannot tolerate ARNI/ACEi/ARB or in patients with NYHA IV on optimal medical

6. Digoxin: recommended in HFrEF with poorly controlled atrial fibrillation on maximal BB and in sinus rhythm with ongoing symptoms on optimal medical therapy

7. **Vericiguat** now considered in HFrEF patients with symptoms and hospitalizations on optimal therapy

▪ **HFpEF > 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 DC.**

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

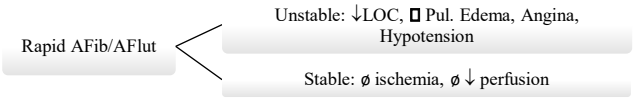
Diagnosis:

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, DM, Stroke/TIA → Anticoagulation

HAS-BLED: HTN, Age ≥ 65, Stroke/TIA, Bleeding Hx, Liver/Kidney Dysf(x), Elevated INR, Drugs (anti-platelets)/Drinks (EtOH)

CHADS2	0	1	2	3	4	5	6
Stroke%/yr	1.9	2.8	4.0	5.9	8.5	12	18

HASBLED	0	1	2	3	4	5	
Bleed%/yr	1.1	1.0	1.88	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 bradyardia

Regular: sinoventricular rhythm, complete AV block (ventricular escape),

sinoventricular rhythm, regular bradycardia with aberrancy

Irregular: Sinoatrial exit block with aberrancy,

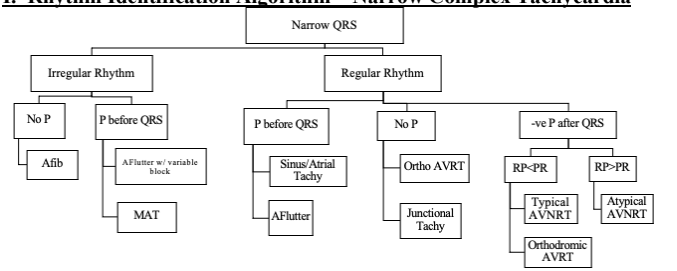
irregular bradycardy with aberrancy, second degree AV block type I and II

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.

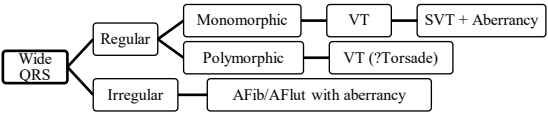
ECG: Tachycardia

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 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)

Author: Justin Boyle *Updated April 2023*

Non- Invasive Positive Pressure Ventilation (NIPPV)

Indications for NIPPV:

- Respiratory acidosis
- Increased work of breathing (to avoid fatiguing)

Absolute C/I for NIPPV:

- Recent (<1month) gastric or esophageal surgery/perforation
- Pneumothorax

Relative C/I for NIPPV (requires close monitoring)

- Cardiac ischemia/arrhythmia, Hemodynamic instability
- Impaired cough/swallow, excessive secretions, vomiting
- Agitated or unable to cooperate, or sedation required, or GCS <13
- Unable to remove mask (DO NOT restrain the pt)

CPAP: ↑ oxygenation and ↑ FRC

Initial setting: min. of 5cmH_2O – liaise with Respirology/RT if unsure about settings (note: pts with high BMI require higher pressures)
Effect: recruit collapsed alveoli
Follow up: SpO_2 and titrate.

BiLevel: ↑ ventilation (and oxygenation)

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
Follow up: ABG within 30-60hr of starting BiLevel. Within 2hrs of starting NIPPV: if pH<7.25 → consider consulting ICU. Monitor tidal volume to assess effect (aim for 400mL)

Predictor of Success on NIPPV:

- Improve in <2hrs: stabilizing HR, RR, ↓ $PaCO_2$ >8mmHg, ↑pH >0.06
- Mild acidemia (pH >7.30), RR<30, GCS 15
- Minimal air leak
- Synchronous breathing

Warning Signs of Impending Intubation:

- Respiratory muscle fatigue, Abdominal paradoxical breathing, Silent chest
- Increasing $PaCO_2$ (or normalizing if initially low)

NIPPV Weaning Criteria:

- Hemodynamically stable
- Wean EPAP once: (1) SaO_2 >92%, (2) FiO_2 <0.50
- Wean IPAP once: (1) pH>7.35, (2) RR<30, (3) $PaCO_2$<50

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 dysfxn 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

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
Complications Monitor for hypocalcemia (pRBCs stored with citrate that binds Ca), hypothermia (can warm blood), hyperkalemia and acid-base derangements
Further considerations Stop the bleeding (e.g GI, IR embolization, surgery)

Vasopressors and Inotropes

Vasopressor
Norepinephrine 2-20mcg/min (first vasopressor for sepsis) No "Max" dose. Consider adding vasopressin if >20mcg/min needed.
Epinephrine 0.01-0.05mcg/kg/min Potent but proarrhythmogenic and increase O_2 demand. Consider in ↓CO+SVR
Phenylephrine 0.5-9mcg/kg/min Take 10mg of phenylephrine and inject into a 100mL NS/D5W bag → 100mcg/mL. Can be given as bolus (100-200mcg 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 Very potent. Helpful in RV failure/pulm HTN as it reduces PVR. Usually an adjunct to norepinephrine.

Inotropes
Dobutamine 2-20mcg/kg/min Direct inotropic effect due to β-agonism with +ve chronotropy, ∴ O_2 consumption. Vasodilatory effect counteracts the inotropic effect. Usually used in combination with Norepinephrine to balance this effect.
Milrinone 0.375-0.75mcg/kg/min 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.
Epinephrine 0.01-0.05mcg/kg/min Consider in hypotension due to ↓CO+SVR – 1st line for ROSC in post-arrest.

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.

- **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

Author: Dr. Sherwin Wong *Updated June 2020*

SJS/ TENs (Steven- Johnson Syndrome/ Toxic Epidermal Necrolysis

Clinical features: Mucocutaneous reaction preceded by fever and dark red spots that may blister. Body surface area (BSA): SJS < 10%, TEN > 30%. Clinical diagnosis with pathological correlation. Nikolsky's sign may be present. Common causative agents: allopurinol, NSAIDs, sulfonamide antibiotics and anticonvulsants **1-3 weeks** after exposure
Investigation:, lesion skin biopsy
Management: Remove offending agent. Supportive measures (similar to burn patients). 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 (eg. Pneumonia, hepatitis, encephalitis)
- Complications
  - 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 those with severe neurologic complications)
- Corticosteroid therapy controversial, but if used must be with antiviral therapy
- Pain: opioids, anticonvulsants (gabapentin/pregabalin), topical lidocaine, TCA's
- Immunocompetent patients require standard precautions. Immunocompromised patients require airborne and contact precautions (as in chickenpox)
-

Endocrinology

Authors: Dr. Samantha Bruzzese, Dr. Robyn Houlden *Updated March 2023*

DKA and HHS

Diagnostic Pattern:

	DKA	HHS
Prominent feature(s)	Ketoacidosis	Volume depletion and hyperosmolality
Glucose (mmol/L)	>14 (not always)	Typically, >30
Arterial pH	<7.30	>7.30
Bicarbonate (mmol/L)	<15	>15
Urine ketones	Positive	Negative
Beta-hydroxybutyrate (mmol/L)	>3.0	< 3.0
Serum osmolality (mOsm/kg)	Variable	>320
Anion gap	>12	<12
Insulin needed	Absolutely needed	Not necessarily needed

Initial investigations:

1. **ABG vs VBG:** Cannot assess mixed acid/base disorders with a VBG.
2. **Blood glucose, lytes, extended lytes, Cr, serum osmolality, albumin, lactate (if hypoxic), A1C:**
   - Pseudo↓[Na^+]: for every 10mmol/L serum glucose >5.6mmol/L, add 3mmol/L to the serum [Na^+] → accounts for ↓[Na^+] due to ↑glycemia)
   - Calculate anion gap
3. **Ketone:** urine dip acetoacetate and serum β-hydroxybutyrate (β-HB)
4. 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 elucidate!

- New diagnosis of diabetes mellitus

- Insulin omission: the most important question is WHY
- Cost, Technique, Knowledge, Psychosocial Factors, Sick Day Rules
- Stressor: Infection, MI, Stroke, Drugs (steroid), Post-operative, etc…

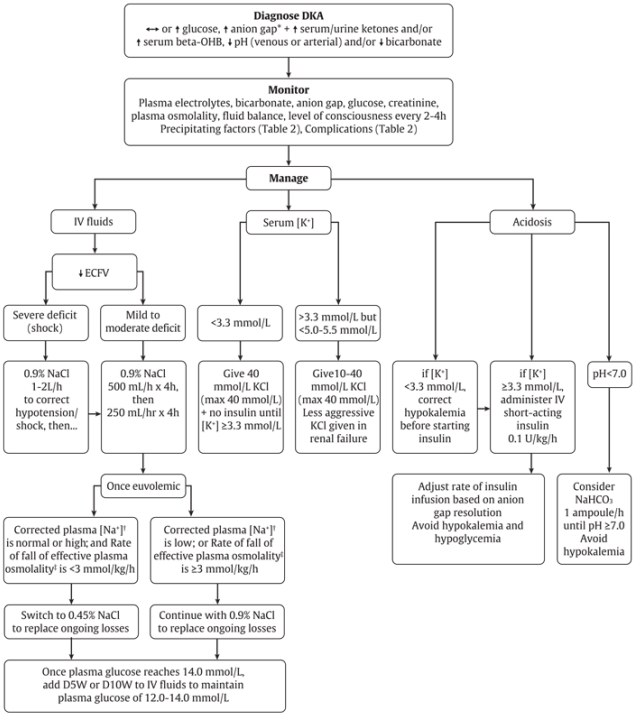
Mx:
Principles:

- **Complex metabolic disorders** – constant physiological flux during Tx. Recognize that the DKA/HHS protocols can fail at times due to the dynamics, therefore **keep monitoring & adjusting** the Mx PRN.
    - **Monitor blood work – lytes, anion gap, osmolality, glucose**
    - **Clinical status – vitals, neuro status, volume status**
- **Tx Order – "DKA": (1) D**ehydration**, (2) K^+ P**otassium**, (3) ↓ A**nion Gap.
- **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 (for example – hyper/hypokalemia, ECFV over-expansion, cerebral edema, hypoglycemia)

Use "Diabetes Management – Diabetic Ketoacidosis (DKA) Order Set (Adult)" on Entry Point (but should know how to write a DKA protocol *de novo*)
Use "Diabetes Management – HHS Order Set (Adult) on Entry Point

Monitoring: (1) -lytes and blood glucose q2h until anion gap is <12 and K^+ is in normal range; (2) capillary glucose q1h until blood glucose <14mmol/L, then q2h; (3) VBG q4h until DKA corrected; (4) Monitor urine output.

Others: ↓$[PO_4^{-3}]$: replete if severe; **pH<6.9**: dilute 3 amps (150mmol) of $NaHCO_3$ in 850mL D5W and infuse over 3hrs, then reassess.



Management pitfalls to beware of:

- Rapid reduction in osmolality can cause cerebral edema – esp in HHS → should be lowered no faster than 3 mOsm/kg/h.
- Insulin is used to stop ketoacid production in DKA – the dose of insulin should be adjusted based on ongoing acidosis (anion gap).
- Glucose reduction in HHS is due to ECFV re-expansion and osmotic diuresis – IV insulin isn't mandatory as there isn't ketoacid production.
- In HHS, IV fluids should be individualized based on clinical picture (not necessarily based on protocol).
- Patients with euglycemic DKA still require insulin to suppress ketosis.

<u>Tx End-Goal – DKA/HHS Resolution Criteria</u>: Anion gap < 12

<u>Bridging to Subcutaneous Insulin</u>:

- Consider when: Tx endpoint is reached <u>+</u> the precipitant is mitigated, and the patient is able to eat a meal
- If SC insulin is ordered, discontinue IV insulin 2 hrs after 1st SC insulin dose is given, or after patient's own insulin pump is reinitiated.
- Be vigilant of anion gap widening, again.

Inpatient Diabetes Management

Refer to Diabetes Management Order sets on Entry Point

- Diabetes Mx – SC insulin therapy patient eating Order Set (Adult)
- Diabetes Mx – SC insulin therapy patient NPO Order Set (Adult)
- Diabetes Mx – Enteral/Parenteral Nutrition Order Set (Adult)
- Diabetes Mx – SC Insulin Pump Therapy Order Set
- Diabetes Mx – DKA Order Set (Adult)
- Diabetes Mx – Pre- and Peri-op Procedure Order Set (Adult)
- Hyperglycemia Mx – Davies 4 ICU IV insulin Therapy Order Set (Adult)

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 – **BUT** involve early in hospitalization
- Glucocorticoid therapy: Hyperglycemia is common
  - Institute CBG monitoring for 48 hrs min in all patients receiving high-dose glucocorticoid therapy → Initiate insulin as appropriate
  - In patients already treated for hyperglycemia, early adjustment of insulin doses is recommended
  - During tapers, adjust insulin doses proactively to avoid hypoglycemia

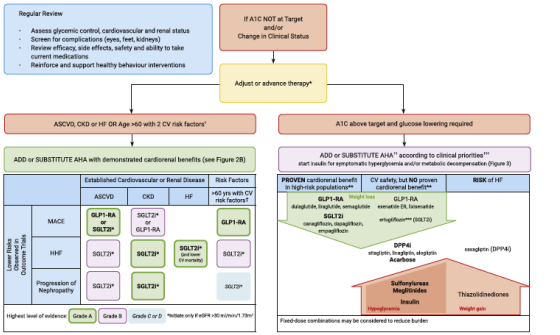
Diabetes Inpatient Management Guide

1	**Eating**	Can Eat		NPO	
2	**Home Meds**	Non-insulin Agents	Insulin	Non-insulin Agents	Insulin
3	**Admission Meds**	As per home regimen (Hold SGLT2i with dehydration)	As per home regimen (consider 20-30% reduct)	D/C	**DM I:** D/C rapid-acting mealtime insulin. Continue basal. **DM II:** D/C rapid-acting insulin. Continue basal.

4	**CBG Frequency**	**Clinical Scenario**	**CBG Monitoring**
		People who are eating	Before meals and bedtime
		NPO, Enteral Feeds	q4-6hr
		Critically Ill	q1-2hr
5	**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
		Acute coronary syndrome	7-10 mmol/L
		Labour and delivery	4-7 mmol/L

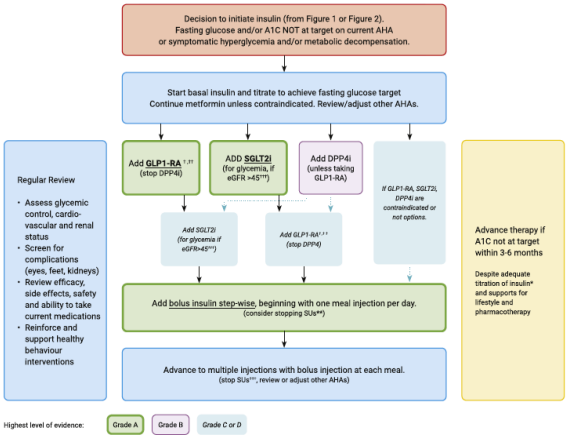
6	**Glycemic Control Regimen Adjustment**	Patient's home regimen may not be appropriate during acute illness, renal injury, and/or dietary restrictions. Noninsulin antihyperglycemics & insulin should be reassessed often. ✓ CBG record daily to look for (1) hypo-, (2) hyper-, (3) recurrence/pattern. Account for these episodes.

Type 2 Diabetes Oral Agents: Reassess oral agents.



* Changes in clinical status may necessitate adjustment of glycemic targets and/or deprescribing.
† Tobacco use, dyslipidemia (use of lipid-modifying therapy or a documented untreated low-density lipoprotein (LDL) ≥3.4 mmol/L, or high-density lipoprotein-cholesterol (HDL-C) <1.0 mmol/L for men and <1.3 mmol/L for women, or triglycerides ≥2.3 mmol/L), or hypertension (use of blood pressure drug or untreated systolic blood pressure [SBP] ≥140 mmHg or diastolic blood pressure [DBP] ≥90 mmHg).
†† All antihyperglycemic agents (AHAs) have Grade A evidence for effectiveness to reduce blood glucose levels.
††† Consider degree of hyperglycemia, costs and coverage, renal function, comorbidity, side effect profile and potential for pregnancy.
** In CV outcome trials performed in people with atherosclerotic cardiovascular disease (ASCVD), chronic kidney disease (CKD), heart failure (HF) or at high cardiovascular (CV) risk.
** VERTIS (CV outcome trial for ertugliflozin) presented at American Diabetes Association (ADA) June 2020 showed noninferiority for major adverse CV events (MACE). Manuscript not published at time of writing.
A1C, glycated hemoglobin; DPP4i, dipeptidyl peptidase-4 inhibitors; eGFR, estimated glomerular filtration rate; GLP1-RA, glucagon-like peptide-1 receptor agonists; exenatide ER, exenatide extended-release; HHF, hospitalization for heart failure; SGLT2i, sodium-glucose cotransporter 2 inhibitors; yrs, years.

Figure 2A. Reviewing, adjusting or advancing therapy in type 2 diabetes.



* Titration of basal insulin to achieve FPG target without hypoglycemia.
† And titrate dose of GLP1-RA, as tolerated.
†† Or fixed-ratio combination.
††† If eGFR >30 ml/min/1.73m², may be used for cardiorenal benefit.
** Sulfonylureas or meglitinides.
AHAs, antihyperglycemic agents; *A1C*, glycated hemoglobin; *DPP4i*, dipeptidyl peptidase-4 inhibitors; *eGFR*, estimated glomerular filtration rate; *GLP1-RA*, glucagon-like peptide-1 receptor agonists; *SGLT2i*, sodium-glucose cotransporter 2 inhibitors; *SU*, sulfonylureas.

Figure 3. Starting or advancing insulin in type 2 diabetes.

Insulin Initiation:

Check insulin requirements on correction dose scale daily and calculate total daily requirements.

Basal Insulin: or 0.2 units/kg glargine U100 or U300 or degludec U100 or U200 once daily; detemir once or twice daily

Insulin on continuous feeds: Complicated, see DM Enteral/Parenteral Nutrition Order set for suggestions, depends on type of feed, Consult Diabetes Consult Service/Endocrinology

Insulin Pumps: Consult the Diabetes Consults/Endocrinology – ALWAYS

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 by blood glucose
Rapid-acting	Before breakfast	Between breakfast and lunch	Before lunch
Rapid-acting	Before lunch	Between lunch and supper	Before supper
Rapid-acting	Before supper	Between supper and bedtime	At bedtime
Long acting	Morning or Bedtime	Evenly over 24 hrs	Before breakfast

Types of Insulin: underlined means covered under ODB, Useful insulin prescription tool available at https://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®) Insulin glulisine (Apidra®) Insulin lispro (Humalog® U100, U200, Admelog®) U-100 Faster-acting insulin aspart (Fiasp®)	 9–20 min 10–15 min 10–15 min 4 min	 1–1.5 h 1–1.5 h 1–2 h 0.5-1.5h	 3–5 h 3.5–5 h 3–4.75 h 3-5 h
Short-acting insulins Insulin regular (Humulin®-R, Novolin® ge Toronto) Insulin regular U-500 (Entuzity® U-500)	 30 min 15 min	 2–3 h 4-8 h	 6.5 h 17-24 h
Basal Insulins			
Intermediate-acting (cloudy) Insulin neutral protamine Hagedorn (Humulin® N, Novolin® ge NPH)	1–3 h	5–8 h	Up to 18 h
Long-acting insulin (clear) Insulin detemir (Levemir®) Insulin Glargine U-100 (Lantus®, Basaglar®, Semglee®) Insulin glargine U-300 (Toujeo®) Insulin degludec U-100, U-200 (Tresiba®)	90 min	N/A	Levimir: 16–24h Lantus®: 24h Toujeo®: >30h Tresiba®: 42h
Premixed Insulins			
Premixed regular insulin – NPH (cloudy) Humulin® 30/70 Novolin® ge 30/70, 40/60, 50/50	A single vial or cartridge contains a fixed ratio of insulin. (% of rapid-acting or short-acting insulin to % of intermediate-acting insulin)		
Premixed insulin analogues (cloudy) Biphasic insulin aspart (NovoMix® 30) Insulin lispro/lispro protamine (Humalog® Mix25 and Mix50)			

Example of Writing Diabetes Prescription at Discharge:

Glargine prefilled pen 25 units sc q2200
M: 3 boxes Repeat x 10

Aspart prefilled pen 8 units sc ac meals.
M: 3 boxes Repeat x 10

Insulin pen needle tips 4 mm
M: 1 box Repeat x 10

Glucose meter test strips of choice
M: 200 strips 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) **OR** • Juice 150 mL **OR** • 1 package of jam; **OR** • 4 Glucose tablets PO	**Conscious patient:** Give 20g PO of fast acting: • Dex4 liquid 1 1/3 bottles (80mL) **OR** • Juice 175 mL **OR** • 1/2 package of jam **OR** • 5 chewable Glucose tabs
Patients receiving enteral nutrition who can't eat: Give 15-16g of fast acting glucose: • Dex4 liquid 1 bottle (59 mL) **OR** • 150 mL of juice	**Conscious patient receiving enteral nutrition who can't eat:** Give 20g of fast acting glucose: • Dex4 liquid 1 1/3 bottle (80 mL) **OR** • 175 mL of juice
Patients who are strictly NPO: • Give 10-25 g (20 to 50 mL) of D50W IV push over 1-3 minutes STAT • If no IV access possible: Give 1 mg glucagon IM/SC (reconstituted) 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: 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** Tx & Monitor as needed.
4. **Etiology:** Diabetic vs Non-diabetic

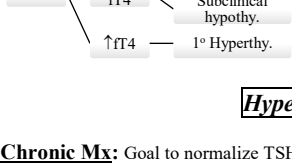
a. Diabetic: Usually related to drug-dose changes/error in administration, renal failure (accumulation of insulin, or other DM meds), skipped meals/NPO

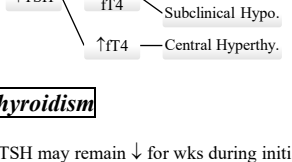
b. Non-Diabetic (uncommon):

- ↓caloric intake: rare – seen in severe starvation
- ↓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 ↓ for wks during initial Tx.

Thioamides	Initial Dose – severity based	Maintenance	PTU is usually only used in 1st trimester & thyroid storm. Otherwise use MTZ Potential adverse effects: pruritic rash, hepatoxicity, agranulocytosis, teratogenic effects
Methimazole (MTZ)	15-60 mg QD	5-15 mg QD	
Propylthiouracil (PTU)	300-900 mg QD	100 mg QD	
Beta-blocker: attenuating sympathetic driven discomforts			
Propanolol	10-40mg q6-8h	May use metoprolol, atenolol in place.	

Thyroid Storm: ↑HR, ↑Temp, CHF, Agitation/Psychosis, Liver failure. **Burch-Wartofsky Score:** ≥45 highly suggestive, 25-44 supportive, <25 diagnosis unlikely.

Acute Mx: Consult Endo. ASAP. If features fit, Tx. Don't wait for TSH/fT4.

General	Storm Specific
1. IV fluids and Correct -lytes 2. Tx ↑temp: cooling, acetaminophen 3. Propanolol 40-80mg PO q6h or 1mg IV q10-15min, OR Esmolol 250-500mcg/kg IVx1 → 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→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 ⍉TSH for 1º Hypothy. & ⍉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 **4.** Find precipitant & Tx	**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 **3.** May require T3 Tx if severe (cannot convert T4 to T3) but IV T3 not on hospital formulary (if was able, give 5 to 20 mcg IV x 1 dose, then 2.5 to 10 mcg IV q8h until clinically improved

Adrenal Insufficiency (AI)

Approach to Diagnosis

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

2. Is patient adrenal insufficient? random cortisol vs ACTH stim test

(a) 8am serum cortisol: highest in early AM – cannot rule in/out.

Cortisol	**Interpretation** (based on early am values)
<80nmol/L	Strongly suggests, but NOT DIAGNOSTIC
>415nmol/L	Diagnosis is unlikely though not confirmatory.

(b) ACTH stimulation test (Non-emergent diagnostic test) - best done in early morning (eg. 8am): (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 → 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 is adrenal insufficiency
- Direct to Canadian Addison Society (http://www.adisonsociety.ca)
- Emphasize adherence – Dangerous to abruptly stop chronic or reduce
- Instruct the patient "Sick Day" Rules – 2-3x usual dose until illness resolves
- If unable maintain PO intake, seek medication attention ASAP for IV Mx
- Medical Alert Bracelet – "Adrenal Insufficiency": Can present with ↓ LOC

Gastroenterology

Authors: Dr. Michael K. Parvizian, Dr. Rana Kandel, Dr. Lawrence Hookey
Updated April 2023

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 - no need for FOBT on DRE samples!

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; and 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 Orderset for dose). 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 the c-scope/flex sig cannot visualize anything – c-scope is NOT an urgent intervention. Stabilize first.
b. Brisk Lower Bleed Suspected:
1. Identify the source:

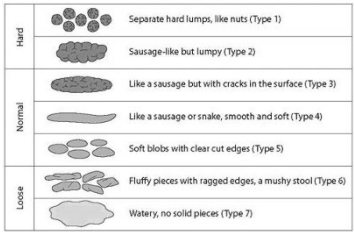
- **CT Angiogram:** the patient needs to be bleeding FAST for this scan to work
- **Tagged RBC Scan:** if CT angiogram failed, or bleed is slower rate

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	Causes: common ones
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 characteristic
- Stool fat test: Tedious and unpleasant – collection and special diet required. Inform the lab & patient. Send for fecal elastase.

Approach to 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 (ie. 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* Consider: ESR, CRP and imaging (CXR, CT scan) *FOBT or FIT are outpatient screening tests.
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: ↑appetite, rarely in T2DM 2.T1DM usually normal or 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 & hyperglycemia, pulmonary 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 a 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

Definitions:

Oropharyngeal dysphagia: difficulty initiating swallowing
Esophageal dysphagia: Sensation of food being stuck in the neck or chest, sternal notch is the borderline

Focused History *If acute onset think of food bolus!

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 (dx with 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

Definition:

- **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 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, associated with PSC	

Investigation:

- 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 mgmt. 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 morbidity and mortality
- 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/dist requiring pRBC transfusion, colonic dilation

Maintenance therapy:

1. 5-ASA can be used to induce remission and maintenance (esp. in UC as it acts topically)
1. Immunosuppressant (6-MCP, Azathioprine, MTX): effective as steroid-sparing agents
2. Check TMPT first and monitor for bone marrow suppression
3. Biologics: Infliximab, Adalimumab, Vedolizumab, Ustekinumab, Tofacitinib
4. 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 in context of immunosuppression.
5. 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 Ankylosing Spondylitis
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, chronic Hep B/C, NAFLD), 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? 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 rapidly: $FENa^+$ <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 ± 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>250x10^6cell = (Total WBC in x10^6cells 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: There are 2 types of acute pancreatitis

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: IGETSMASHED

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

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.

Hematology

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Blood Products

Consent: obtain consent from the patient, or from a SDM; can sign emergency consent if life-threatening emergency but must obtain consent as soon as possible after commencing transfusions
*A word on Jehovah's Witness: EXPLICITLY ASK – some are willing to receive certain blood products (eg. albumin). Document clearly. Transfusion Services | KHSCNow (kingstonhsc.ca)

I. RBC Transfusion: 1 unit RBC is 300mL

Risks of Transfusion from RBC per unit:

Infection	Risk	Reactions	Risk
Sepsis	1 in 250,000	Febrile non-hemolytic rxn	1 in 100
HBV	1 in $2x10^6$	Delayed hemolytic rxn	1 in 2,500
HCV	1 in $27x10^6$	Anaphylaxis	1 in 40,000
HIV	1 in $13x10^6$	ABO incompatibility	1 in 354,000

RBC Transfusion Threshold for inpatients:

- Hb<50g/L for uncomplicated vaso-occlusive pain crisis in sickle cell disease
- Hb<50 g/L for chronic iron deficiency without serious symptoms (along with intravenous iron)
- Hb≤70g/L for inpatients with no cardiac history, including active gastrointestinal bleeding
- Hb≤70-80g/L for inpatients with cardiac disease without hemodynamic symptoms
- Hb≤90g/L if hemodynamic symptoms (tachycardia, pre-syncope, etc.)\
- In absence of active hemorrhage, order 1 unit at a time

[Written Order]: [*Group & Screen, Transfuse "X"unit RBC over Y hours".*]

- "X": 1unit RBC ↑ Hb by ~10g/L. "Y": transfuse over 2hrs if no cardiac history or volume overload (max 4hrs).
- Repeat CBC post-transfusion if bleeding, otherwise perform following AM
- Consider administering with Lasix 20-40mg IV in patients with CHF or volume overload prior to transfusion
- Where time allows always use the EntryPoint orderset to ensure you order irradiated or phenotyped matched RBCs where indicated

Other Transfusion Orders:

- **[Group & Screen]:** ABO, Rh(D) Grouping and Ab Screen – process requires ~60min.
- **[Crossmatch X units RBC]:** Blood Bank will crossmatch units and hold. Only order this for patients with known antibodies that require a full IAT crossmatch. Always write after in brackets "Do not transfuse

II. Platelet Transfusion: 1 adult dose is either a pool (180 mL) or an apheresis unit (223 mL)

Indication: Plt ($x10^9$/L) Threshold	
Active Bleeding	<50 (<80-100 if CNS bleed)
Neurosurgery	<80
Major Surgery or high risk invasive procedure	<50 (<30 for patients with cirrhosis)
Low risk invasive Procedure	<20
Prophylaxis	<10

[Written Order]: *transfuse 1 adult dose over 1 hour*] (Max over 4hrs)

- Repeat CBC within 1 hour of transfusion (1 adult dose ↑ Plt by >7 $x10^9$/L
- Platelets should only be transfused to patients with ITP, PTP, or HIT after approval by hematology or transfusion medicine

III. Frozen Plasma (FP): 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

[Written Order]: [*Transfuse "X" units FFP over "Y units of time"*

- "X": 3units for "small" adult, 4units for "larger" adult. "Y": Transfuse over X hours each (Max over 4hrs, rate dependent on presence of bleeding and cardiovascular status)
- Use EntryPoint orderset if possible.
- Repeat INR immediately post-transfusion.

IV. 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
- **[Written Order]:** [*Transfuse 4 grams over 5 minutes by iv push or minibag infusion*.] Each dose ↑ fibrinogen by ~0.5-1.0g/L

V. Albumin

- **Indications:** (1) Large volume paracentesis (>5L) (2) SBP (3) Hepatorenal syndrome in combination with other agents
- **[Written Order]:** [*Transfuse "X" mL of 25% albumin IV*]
- 25% albumin in 100mL bottle
- See Entrypoint orderset for dosage for each indication

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

- **Indication:** Emergency reversal of warfarin or vitamin K deficiency when (1) INR>1.5 AND (2) life threatening bleeding or emergency surgery
- **[Written Order]:** [*Transfuse Octaplex X IU, each 1000 IU over 5min*] + Vitamin K 10mg IV

INR	PCC Dose
<3	1,000 IU
3-5	2,000 IU
>5	3,000 IU

Note: PCC's should not be given when vitamin K would be sufficient

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

- PCC for NOACs
- Dose is 2000 IU and if still bleeding at 1 hour a second dose of 2000 IU
- Use entrypoint orderset for Anticoagulant reversal

Acute Transfusion Reactions

Acute Transfusion Reactions: Notify blood bank immediately. Send BOTH the blood and tubing back to the lab for testing (exceptions: mild fever <39ºC with no other symptoms, and urticarial reactions). Always check for clerical errors. Always stop the transfusion and perform a bedside assessment.

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 urinalysis.

b. Acute Febrile, Non-Hemolytic Transfusion Reactions: fever up to 4 hours post-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

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 PRBC due to dilution of coagulation factors (PRBC doesn't contain clotting factors or platelets)
Prevention: Transfuse 4 units PRBC, 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.

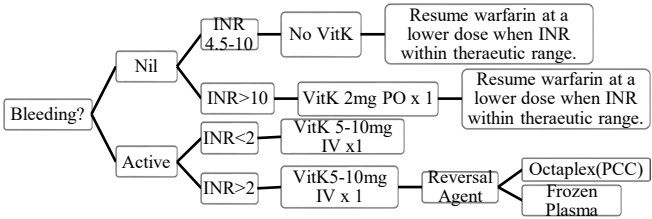
b. Citrate toxicity: exacerbated in those with liver dysfunction. Results in:
- **Metabolic Alkalosis:** citrate metabolism generates bicarbonate
- **Hypocalcemia:** citrate binds to calcium. Can give calcium prophylactically every 4 units

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

Authors: Dr. Kerstin de Wit *Updated March 2023*

Risk factors for VTE:

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

Deep Vein Thrombosis

Approach

1. Symptoms: Unilateral calf pain, ankle swelling, tenderness, pain, erythema
2. Diagnosis for outpatients: Wells score for DVT: Score 1 or less → D-dimer, Score 2 or more → 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.

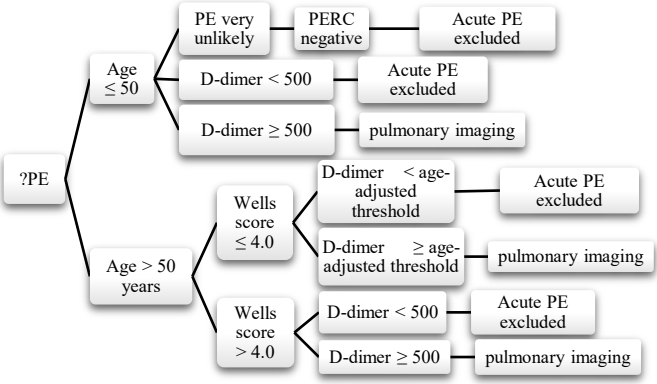
Mx

1. Anticoagulation (**See Below: Anticoagulation Mx**)
2. Phlegmasia cerulea dolens (suspect with cyanosis): Consult IVR for catheter directed tPA

Pulmonary Embolism

Approach

1. HPI: Dyspnea, chest pain, fatigue, presyncope on walking up stairs, 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 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.
Select intermediate-low risk patients may also be treated at home, if they have normal vitals, can mobilize safely and there is no other indication for hospital admission.

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), who do not have a high bleeding risk: Thrombolytic: alteplase, dose may vary between 50 mg and 100 mg IV, or tenecteplase 0.5mg/kg to a maximum of 50 mg IV. Use KHSC thrombolysis order set. Follow with either dalteparin 100 units/kg SC every 12 hours or IV unfractionated heparin.

For intermediate-high risk patients: give dalteparin 100 units/kg SC every 12 hours. Patients > 95 kg should have 100 units/kg twice a day, **maximum dose 12,500** units twice a day. 1 in 20 patients will deteriorate to high-risk so plan ahead: assess patient for thrombolysis contraindications (see thrombolysis order set) and explain plan to patient to check they would consent for thrombolysis if they developed hemodynamic shock. 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? (Check PCS)
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 next steps.
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 for 7 days followed by 5 mg bid for 90 days, LU code 444
or
RIVAROXABAN 15 mg bid for 21 days followed by 20 mg daily for 90 days, LU code 444
or
DALTEPARIN 200 units per kg, once per day for 14 days, with one repeat, LU code 188.
Round to the nearest prefilled syringe dose: 7,500 / 10,000 / 12,500 / 15,000 / 18,000 units.

Patients > 95 kg should have 100 units per kg twice a day, **maximum dose 12,500** units twice a day.
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).

Special note: do not use IV unfractionated heparin unless creatinine clearance is < 20 ml/min or the patient has a high risk of imminent bleeding. For intermediate-high risk PE use LMWH.

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 start / monitoring.

IVC Filter:

Only indication:
Bleed risk so high that patient cannot be anticoagulated.
Team who inserts filter is responsible for ensuring removal at first possible opportunity.

Complications of leaving IVC in situ: filter fracture, migration, thrombosis; IVC perforation; increased risk of DVT.

Multiple Myeloma

Author: Dr. Bethany Monteith ***Updated March 2023***

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, Anemia Hgb <100g/L, Bone 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. Symptoms → severe back pain (85% of pts), weakness, sensory deficits, bowel/bladder dysfunction (late finding). Dx Requires **Urgent MRI / CT.** Consider urgent radiation oncology and/or neurosurgery consultation. Start steroids. 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 in 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). Results in thrombocytopenia and thrombosis. Timing tends to occur during Day 5-14 after exposure to heparin or its derivatives.

When to Consider this Dx: Trend of platelet count reduction, Thrombosis (venous or arterial), Recent heparin exposure

4T Score: Provides Pre-Test Probability of HIT

Parameters	2pt	1pt	0pt
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 heparin exposure within 30 days	Onset 5-14 days but not clear; **or** onset after day 14; **or** day 1 if heparin exposure >30 days ago	Onset < 4 days (no recent heparin)
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 (argatroban, bivalirudin, fondaparinux, or DOAC)
- 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 entry point 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 active 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
Updated by Andrew McNaughton June 2021

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

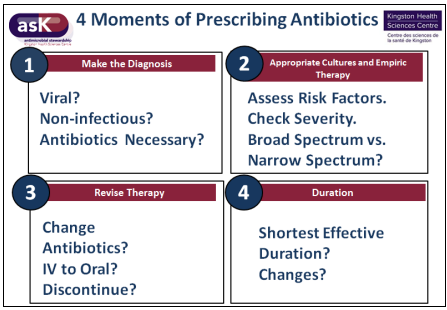
Kingston Health Sciences Centre Antimicrobial Stewardship

(https://khscnow.kingstonhsc.ca/cr/antimicrobial-stewardship-programfrom 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



Central Line-Associated Bloodstream Infections

Diagnostic criteria: any of the following

a. **Blood culture from CVC lumen and peripheral vein grows same organism** and:
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

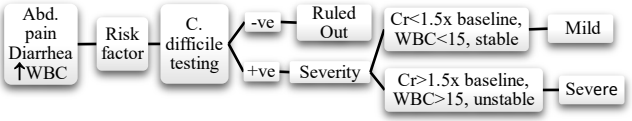
Clostridiidoides (formerly Clostridium) difficile Infection(CDI)

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, IVcatheter, 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

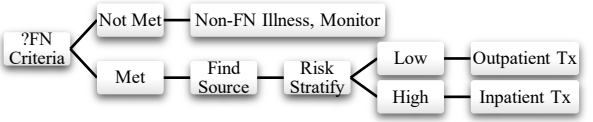
Febrile Neutropenia (FN)

Definition

- **Fever:** $\geq$38.3^{o}C x1, or sustained $\geq$38^{o}C >1hr (Be wary of fever suppression: acetaminophen, prednisone, elderly)
- **Neutropenia:** ANC<1.0x10^{9}/L (ASCO 2012), or expected to $\downarrow$ <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

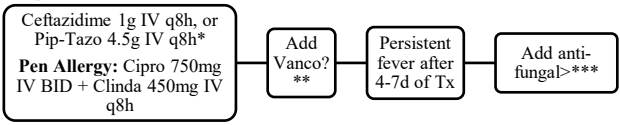
High Risk: (a) Failure to meet Low Risk Criteria; (b) Heme Transplant; (c) Infectious focus identified; (d) Febrile for >48hr; (e) FN DESPITE on Neupogen®/Neulasta®.

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. pneumoniae*
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 past 3mths – consider using a different class
- **Culture Hx:** Pathogen and sensitivity/Ω, and notable colonization
- **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 all.; 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:

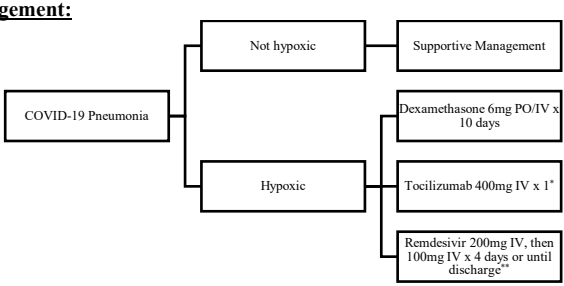
High Risk	Moderate Risk	Low Risk
• Age ≥60 years • Nursing home resident • Immunocompromised • Chronic lung disease (incl. asthma) • Cardiovascular disease (incl. HTN) • Morbid obesity (BMI ≥ 40kg/m2 • Diabetes • CKD – dialysis • CVD • Liver disease • Tobacco use	• Age 20-64 with no high risk factors • 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 1° 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!!

	(1) Empirical Antibiotic Mx – a Guide

Ulcer: Diabetic foot is most common. Sacral ulcer is also common*.	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)

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:

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**

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 (3rd/4th), 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,

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

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

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%
Ciprofloxacin	400mg IV or 500mg PO BID	Use PO unless otherwise indicated

*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 (1st/2nd 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 wt. instead of in's and out's

Nephrology

Authors: Dr. Sunchit Madan & Dr. Caitlyn Vlasschaert *Updated June 2020*

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-	1. Absolute ↓ in circulatory vol. – blood, GI, urine 2. Effective ↓ in circulatory vol: CHF, cirrhosis, nephrotic syndrome, sepsis 3. 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
Renal	**Glomerular:** Nephrotic vs Nephritic Syndrome **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;	**Glomerular:** • Urine: RBC casts, RBC acanthocytes • 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)

	hemoglobulin – massive hemolysis; light chain – MM, urate –TLS) • Chronic: PCKD **Vascular:** • Acute: HUS/TTP; vasculitis Chronic: HTN, DM	• ↑CK; hemolysis work up; SPEP&UPEP, immunoglobulins; uric acid **Vascular:** Blood smear (schistocytes)
Post-	**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
  - 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.
  - It is important to consider other causes of hyperkalemia 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?** (✓Outpatient blood work): (a) Baseline Cr, (b) Acute vs Chronic vs Acute on Chronic, (c) Progression Rate.

3. **Post-Renal? US** – rapid & definitive. Definitive Tx requires Urology/IVR intervention. No urine drainage post-foley insertion does not mean that post-renal AKI is ruled out.

4. **Pre-Renal? Hx+Exam usually suffice**. Renal artery etiologies are less apparent.

5. **Renal?** Not obvious. **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 by far the commonest you will encounter.

Determining Cause:

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

- Hematuria, frothy urine, signs of obstruction (frequency, nocturia, urgency)
- History of diabetes and determine severity
- Rheumatological conditions such as SLE, Sjogren's, or scleroderma
- Kidney stones, abdominal malignancies, bladder outlet obstruction
- NSAID use (naming generic and brand names)
- Polycystic Kidneys, dialysis dependence in family, SLE

Investigations:

- Urinalysis – 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)

Complications:

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. Targetting normal Hb can be harmful.

Bone Disease Ca^{+2}, PO_4^{-3}, PTH, vascular calcification

$\uparrow PO_4^{-3}$:

- Strict renal/dialysis diet
- PO_4^{-3} Binders: binds to ingested PO_4^{-3} and prevents absorption
- Ca^{+2} carbonate 500mg PO ac meals: may result in $\uparrow Ca^{+2}$ which is associated with other harmful effects
- If refractory $\uparrow PO_4^{-3}$, or in the context of $\uparrow Ca^{+2}$: sevelemer or lanthanum

$\downarrow Ca^{+2}$:

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

2° hyperparathyroidism:

Suppress with VitD though may result in $\uparrow PO_4^{-3} + \uparrow Ca^{+2}$. If refractory, consider calcitriol followed by calcimimetic ex. cinacalcet. Consult Nephrology.

Cardiovascular:
- 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.

Drugs:
Renal dosing or contraindicated. Nephrotoxin?

Electrolyte:
- $\uparrow K^+$: Renal diet (low potassium), Kayexelate® (unclear benefit)
- Met Acidosis: Sodium bicarbonate 500mg -1g PO BID-TID. Sodium load will complicate BP and volume control Mx.

Fluid overload: Diuretics

Renal Replacement Therapy Related Complications

Common Complications:
Hypotension: usually seen in those initiating HD, titrating in dry weight. Look for ischemic injury

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.

	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"	$\uparrow[Na^+]$
0.45% NaCl	77	0	0	0	Hypo	Maintenance fluid, $\uparrow[Na^+]$

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^+]

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.
****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

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 – ↻AG)/(↻$HCO3^-$ - 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° 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**

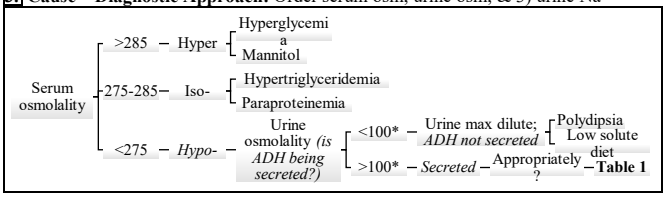
Management

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 2º 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: 8-12 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:**

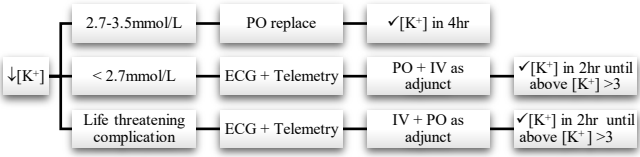
	Source	Etiology	Elicit Clues From:
1	GI loss	Diarrhea, vomiting, SPSS (Kayexelate®)	Hx, Meds
2	K^+ shifting into cells	Na^+/K^+-ATPase pump activation: • **β-agonist** (e.g., salbutamol) • **insulin** (e.g., refeeding syndrome, exogenous insulin administration)	Hx, Meds
		Alkalosis (respiratory or metabolic) *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). **Pseudo**hyperkalemia 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)

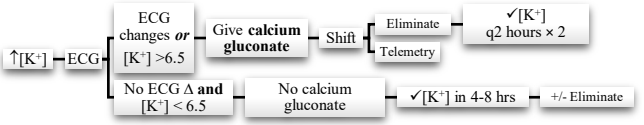
Causes of true hyperkalemia:

	Source	Etiology	Elicit Clues From:
1	↑ K^+ intake (any route)	Supplements (IV, Oral)	Meds
2		↓ Na^+/K^+-ATPase pump activity:	Hx, Meds

	K^+ shifting out of cells	• Insulin deficiency (**DKA**) • β-blocker toxicity • digoxin toxicity	
		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
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

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)

3. Management

1. IV fluids: NS 1-2L IV bolus, then 100-200mL/hr.
- 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 $[Ca^+]$	
Tests to help differentiate			**GI Loss**	**Renal wasting**
		24hr urine Mg collection	< 10 mg	> 10-30 mg

between GI and renal losses:	Fractional excretion of Mg *(analogous to FENa)*: $FE_{Mg} = \frac{U_{Mg} \times S_{Cr}}{(0.7 \times S_{Mg}) \times U_{Cr}}$	$FE_{Mg} < 2\%$	$FE_{Mg} > 4\%$

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. ✓ $[Mg^{2+}]$ 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	✓ $[Mg^{2+}]$ 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)	✓ $[Mg^{2+}]$ 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

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 ✓ [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	✓ [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	✓ [Mg^{2+}] 24 hrs post-replacement

Neurology

Authors: Lauren Mak *Updated June 2020*

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 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 seizure 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 **Phenytoin** 20mg/kg (25-50mg/min) OR **Fosphenytoin** 5mg/kg PE (100-150mg PE/min) No IV: can give IO or IM
	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 continuous **midazolam** or **propofol** infusion Third-line drugs: phenobarbital, lacosamide, topiramate, clobazam	
4. Secondary Assessment (Posticta l Recovery)	Repeat full neurologic exam Head CT or MRI LP if infectious etiology suspected	

Investigations

- **Electrolytes:** [Na^+], [Ca^{+2}], [Mg^{+2}], glucose
- **Metabolic:** TSH, Renal Function, LFTs
- **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	FamHx of Sz, epilepsy, sudden deaths. Personal Hx of Sz.
Acquired	
Metabolic	↓/↑ Glucose, ↓Na^+, ↓Ca^{+2}, ↓Mg^{+2}, Anoxia, Hepatic encephalopathy, Uremia, Thyroid storm, sepsis.
Toxin/Meds	EtOH/barbituates/benzo withdrawal. Elicit drugs. Non-compliance, Δ 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 have to report to driving authorities.

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 or Collateral Hx: Was onset <4.5hrs ago? Determine eligibility for tPA vs. thrombectomy
		Rule out mimics	↓/↑glycemia, Seizure, 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 45°, 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)

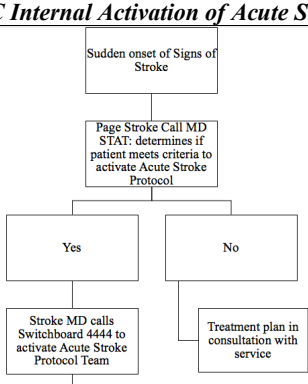
2B) Ischemic – thrombectomy candidate (4.5-24 hrs):

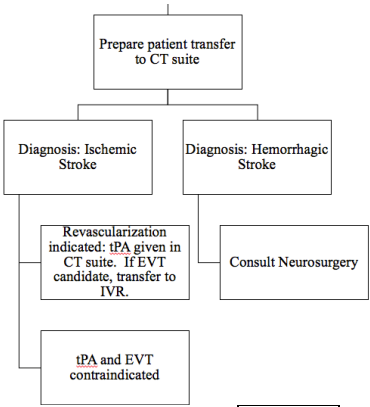
- Treatment can be initiated within timeframe of last known to be well
- Small infarct core and no hemorrhage, clot burden not in distal branches
- Imaging-target mismatch ratio>1.8, volume>15mL and ischemic core>70mL OR clinical-ASPECTS mismatch (NIHSS≥10 + ASPECTS ≥6)

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 1° headache (migraine, tension, cluster) • Hx of similar episodes • ∅ pattern change • ∅ high risk comorbidities • ∅ neurological findings on px • ∅ 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 <40cm$H_2$0	
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

Author: Dr. Grace Zhang *Updated June 2020*

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 continue screening until 3 consecutive screening results obtained.
Colorectal	≥50-74 years old with average risk for colorectal CA: (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, which ever 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; <10% of those with brain tumour presents with headache alone – usually with focal neuro deficit, or ↑ICP: awakens patient at night, exacerbated with Valsalva, and associated with N&V. Δ**LOC. 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

Mx:

1. **Glucocorticoids (Vasogenic only):** Dexamethasone 10mg IV x 1→ 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) Lumbrosacral: 30%; (d) Cervical: 10 – Note: 50% have more than 1 area of compression

Clinical Features*: Variable. Most often associated with breast cancer, multiple myeloma, lymphoma, lung ca, prostate ca

- **Back pain:** (a) Localized & progressive ≈ spine lesion; (b) Radicular, worse with recumbency, or Valsalva ≈ nerve root compression/invasion; **Band-like sensation around body** ≈ thoracic radiculopathy
- **Neurological:** Motor usually proceeds sensory deficit – Complains of weakness, or clumsiness; **Ambulatory?**; Sensory – **CHECK SENSORY LEVEL FROM THE BACK;** Saddle anesthesia, anal tone; Reflex: ↑ (cord), or ↓/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 → 4mgPO/IV BID for duration of radiation
2. **Emergent Consultation:**

- **NeuroSx – ASAP****: (a) able to tolerate surgery; (b) 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:

*THOROUGH Neuro exam & DOCMUMENT THE FINDINGS IN DETAILS FOR COMPARISON The diagnosis is not always clear in the beginning, and it can evolve rapidly within hrs to typical features.

Do not delay – if you cannot contact the consult resident (or not satisfied with their assessment & plan), **INFORM YOUR ATTENDING ASAP.

Superior Vena Cava Syndrome

Definition: venous return to the ♥ impaired due to SVC obstruction

Clinical Features: all 2° to venous congestion to all 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→ 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:

*Deferring dexamethasone for a satisfactory biopsy is acceptable if the patient is stable.

IV fluids: IV fluid via arm veins will exacerbate SVC syndrome

***NOT an emergency **UNLESS presence of cerebral edema, or laryngeal edema resulting in 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 [$Ca^{+2}{}_T$]<1.75mmol/L, [$Ca^{+2}{}_i$]<0.3mmol/L
- **Clinical (Laboratory + ≥1 parameter):** AKI, ♥ arrhythmia, seizure, or death

Risk Factors
- Hematologic malignancy (esp. acute leukemias and high-grade lymphomas)
- Dehydration
- Pre-existing kidney dx
- 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 (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, abdominal pain)
- Hepatitis
- Pancreatitis
- Pneumonitis (cough, fever)
  - Chest x-ray may show consolidation which mimics pneumonia
  - Can lead to ARDS and acute respiratory failure
- Myocarditis
- Uveitis
- Endocrinopathies: hypo/hyperthyroidism,hypophysitis (inflammation of pituitary gland), adrenal insufficiency, T1DM

Investigations
- As appropriate depending on organ involvement

Mx:

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

CARDIAC RISK ASSESSMENT

Authors: Dr. Ryan Peters, Dr. Kristen Marosi

Guidelines pertain to patients undergoing non-cardiac surgery requiring overnight hospital stay

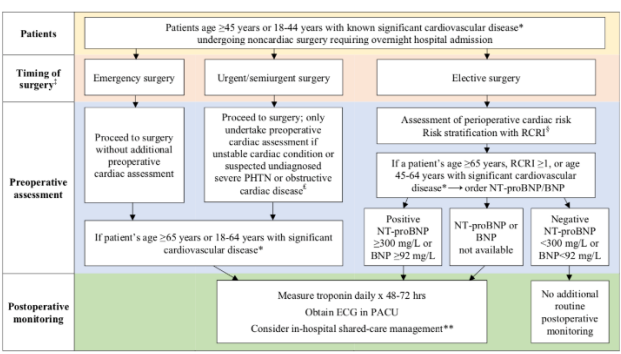
- Age ≥ 45 or
- Age 18-44 with known significant cardiovascular disease (CAD, CVD, PAD, CHF, PHTN, severe obstructive intracardiac abnormality)
-

Revised Cardiac Risk Index (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 surgery)	+ 1

Risk of myocardial infarct, cardiac arrest, or death at 30 days post-op

RCRI Score	Risk estimate (95% CI)
0	3.9 % (2.8% – 5.4%)
1	6.0% (4.9% – 7.4%)
2	10.1% (8.1% – 12.6%)
≥ 3	15.0% (11.1% – 20.0%)



Patients who experience myocardial injury or infarct after non-cardiac surgery (MINS) should be initiated on ASA and statin therapy, and have cardiac risk factors optimized.

NOTE: KGH has an Entrypoint Order Set for MINS.

ANTIPLATELETS

Clinicians must balance the risk of discontinuation of antiplatelet agents against the risk of bleeding. Below is an approach to the management of antiplatelets in the perioperative setting:

Type of Surgery	**Aspirin (81 mg)**	**P2Y12 inhibitors**
Low risk procedures (see below for details)	ASA can be continued	If monotherapy: stop 3-4 d before procedure If DAPT: stop 7-10 d before the procedure
Elective/Non-urgent noncardiac surgery in patients with no coronary stents Based on POISE 2 trial which showed ASA had no benefit on MACE, but increased major bleeding	Stop 3-7 days prior *does not include patients undergoing carotid endarterectomy or with recent coronary stenting (6wk BMS, 3-12mo for DES) Resume 8-10d post-surgery (restart earlier if MINS event)	
Elective/Non-urgent noncardiac surgery in pts with coronary stents		
PCI patients with a BMS -recommend delaying surgery for 1mo	ASA can be continued	Stop 5-7 d before (clopidogrel or ticagrelor) Stop 7-10 d before (prasugrel) Should be restarted as soon as considered safe by surgeon
PCI patients with a DES -recommend delaying surgery for 3 months -if semi-urgent surgery required delay at least 1 month after PCI w DES	ASA can be continued	Stop 5-7 d before (clopidogrel or ticagrelor) Stop 7-10 d before (prasugrel) Should be restarted as soon as considered safe by surgeon
Elective/Semi-urgent CABG after ACS	ASA should be continued	Stop 2-3 d before (ideally 5 d)

		(clopidogrel or ticagrelor) Stop 5 d before (ideally 7) (prasugrel)

ANTICOAGULATION

Thromboembolic risk must be balanced again procedural bleeding risk to determine perioperative management of anticoagulation therapy.

Thromboembolic risk

High	Mechanical mitral valve Stroke or TIA < 3-6 months Atrial fibrillation with CHADS 5-6 or rheumatic valvular disease VTE within 3 months Severe thrombophilia Protein C/S or antithrombin deficiency Antiphospholipid antibodies
Intermediate	Bileaflet AVR Atrial fibrillation with CHADS 3-4 VTE within 3-12 months Recurrent VTE Active cancer
Low	Atrial fibrillation with CHADS 0-2 (no prior stroke or TIA) VTE > 12 months

Procedural bleeding risk

High	Heart valve replacement Coronary artery bypass Abdominal aortic aneurysm repair Neurosurgical/urologic/head and neck/abdominal/breast ca Bilateral knee replacement Laminectomy Transurethral prostate resection Kidney biopsy Polypectomy, variceal treatment, biliary sphincterectomy, pneumatic dilatation PEG replacement Endoscopic guided FNA Multiple tooth extractions Vascular and general surgery Procedure duration > 45 min
Low	Cholecystectomy Abdominal hysterectomy GI endoscopy ± biopsy, biliary/pancreatic stent without sphincterotomy, endosonography without FNA Pacemaker, cardiac defibrillator insertion Simple dental extraction Carpal tunnel repair Knee/hip replacement, shoulder/foot/hand surgery, arthroscopy Dilatation and curettage Skin cancer excision Abdominal hernia repair

	Hemorrhoidal surgery Axillary node dissection Cataract and non-cataract eye surgery Bronchoscopy ± biopsy Central venous catheter removal Cutaneous and bladder/prostate/thyroid/breast/lymph node biopsy

Perioperative anticoagulation

Medication	Pre-operative Management	Post-operative Management
Warfarin	Discontinue Warfarin for 5 days pre-op to allow INR ≤ 1.5 at time of surgery. Consider bridging with LMWH/UFH in patients with high thromboembolic risk or patient with intermediate thromboembolic risk but low bleeding risk. Last dose of LMWH ≥ 12h or UFH ≥ 4h prior to surgery.	Resume Warfarin post-op at usual or twice the maintenance dose on POD0 or 1. If bridging, restart LMWH/UFH on POD1 if low bleeding risk or POD2-3 if high bleeding risk – consult with surgical team. Continue until INR is therapeutic.
Dabigatran	Discontinue 2-3 days before surgery if CrCl > 50 ml/min, 3-4 days before surgery if CrCl 30-50 ml/min.	Resume Dabigatran on POD1 if low bleeding risk or POD2-3 if high bleeding risk – consult with surgical team.
Rivaroxaban	Discontinue 2-3 days before surgery if CrCl > 30 ml/min, 3-4 days before surgery if CrCl 15-30 ml/min.	Resume Rivaroxaban on POD1 if low bleeding risk or POD2-3 if high bleeding risk – consult with surgical team.
Apixaban	Discontinue 2-3 days before surgery if CrCl > 50 ml/min, 3-4 days before surgery if CrCl 30-50 ml/min.	Resume Apixaban on POD1 if low bleeding risk or POD2-3 if high bleeding risk – consult with surgical team.

DIABETES IN THE PERIOP SETTING

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

Acute hyperglycemia affects immune function and wound healing. In general, it can lead to increased postoperative infections (surgical site infections), has been shown to increase allograft rejection, and can increase morbidity and mortality in surgical patients.[2]

The benefits of improved perioperative glycemic control need to be balanced against perioperative hypoglycemia, which can be masked if a patient is receiving sedation. Frequent BG monitoring is recommended.

Management of Oral Antihyperglycemics

Based on the American Diabetes Association 2021 Standards of Medical Care in Diabetes:[3]

Oral Antihyperglycemic	Preoperative Management
SGLT2 inhibitors	Withhold 3-4 days before surgery
Other oral antihyperglycemics (including metformin)	Withhold the day of surgery

*Note, no evidence of GLP-1 agonists on glucose levels in perioperative setting.[3]

Oral antihyperglycemics may be restarted in the postoperative setting when the patient is eating well and there are no other contraindications (i.e. acute renal insufficiency)

Management of Insulin Therapy

Basal insulin is required in all Type 1 diabetics and Type 2 diabetics who are insulin dependent, even when NPO. This is to prevent ketoacidosis.

Insulin therapy in the perioperative setting can be highly individualized. A general approach (assuming NPO at midnight) is provided below:

Type of Insulin	Dose and Regimen	Recommendations
Long acting basal (i.e. Glargine or determir)	Once nightly	Reduce dose by 25%[3,4]
Long-acting basal insulin (i.e. glargine or determir)	Twice daily	Reduce am dose the morning of surgery by 25%[4]
NPH or 70/30 insulin	Twice daily	Reduce am dose on day before surgery by 20%[4] Reduce pm dose the night before surgery by 20%[4] Reduce am dose the morning of surgery by 50% [4]
Type 1 Diabetics on Basal Insulin		Patients should receive 80% of basal insulin dose evening before surgery and 80% the morning before surgery[4] This is done in order to prevent hyperglycemia/ketoacidosis

Short acting insulins		Hold day of surgery[4]

Consider insulin infusion for OR cases >4hrs and patients on large doses of insulin or DM1 on BBI.

Type of Surgery	Recommendations
General Surgery	Suggests pharmacologic prophylaxis (LMWH or UFH) In lap choles, no pharmacologic prophylaxis
Neurosurgical	Suggests against pharmacologic prophylaxis
Trauma	Low-moderate bleeding risk: suggest using pharmacologic prophylaxis High risk of bleeding-suggests against pharmacologic prophylaxis.
Cardiac or Major Vascular	Suggests pharmacologic prophylaxis (LMWH or UFH)
Gynecologic	Suggests using pharmacologic prophylaxis
Urologic	TURP-suggests against pharmacologic prophylaxis Radical prostatectomy-suggests against pharmacologic prophylaxis
ASH Guidelines Notes	-In situations in which pharmacologic prophylaxis is recommended, mechanical prophylaxis is a reasonable alternative -In situations in which pharmacologic prophylaxis is not recommended, mechanical prophylaxis should be used -When possible, mechanical + pharmacologic prophylaxis should be considered

NOTE: KGH has a diabetes pre and post procedure entry point order set.

THROMBOPROPHYLAXIS IN THE POSTOPERATIVE SETTING

Overview

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

If possible, patients should receive pharmacologic prophylaxis.[5] This is done with LMWH or unfractionated heparin, or in the case of orthopedic surgery, DOACs may also be used in select scenarios.[5,6]

In situations in which pharmacologic thromboprophylaxis is contraindicated, the use of mechanical prophylaxis over no prophylaxis is preferred.[5] Of mechanical prophylaxis, intermittent compression devices are preferred over graduated compression stockings.[7]

Guidelines ultimately vary as to use of extended (> 2 weeks) versus short-term (<2 weeks) thromboprophylaxis.[5] Generally speaking, thromboprophylaxis should be continued until the time of discharge, with

extended duration up to 30 days considered in those at high risk for VTE (such as high-risk cancer surgeries, traumas, etc.).[5]

Thromboprophylaxis Approaches in Non-Orthopedic Surgery

Multiple approaches and guidelines exist. Two major guidelines are the 2012 American College of Chest Physicians (ACCP) guidelines[8] and the 2019 American Society of Hematology guidelines

2019 ASH Guideline: Thromboprophylaxis in Orthopedic Surgery

Patients undergoing orthopedic surgeries are at particularly high risk of VTE. Thromboprophylaxis substantially decreases this risk and is recommended in almost all orthopedic surgery scenarios.

Thromboprophylaxis should start about 12 hours after surgery if a pharmacologic approach is being taken. Extended duration (greater than 2 weeks) thromboprophylaxis is recommended for patients with hip or knee arthroplasty, or hip fracture surgery.

The following table from Thrombosis Canada lists current recommendations for thromboprophylaxis after orthopedic surgery.

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 Higher risk (major knee reconstruction, prior VTE, cancer, other VTE risk factors)—LMWH once daily or direct oral anticoagulant	5-30 days
Lower extremity amputation	LMWH once daily	Until discharge (including rehabilitation)
Other (bedrest, incision and drainage, etc.)	LMWH once daily	Until discharge

MEDICATION MANAGEMENT IN THE PERIOPERATIVE SETTING

Certain medications may need to be held, continued, or not initiated within the perioperative period. This is due to a risk-benefit analysis of both adverse and beneficial effects of specific medications within the perioperative period.

The 2017 Canadian Cardiovascular Society Guidelines, *Perioperative Cardiac Risk Assessment and Management for Patients who Undergo Noncardiac Surgery*, provides the following recommendations regarding peri-operative medication management:[9]

Intervention	Management
Management of medications taken chronically and smoking before noncardiac surgery	
ASA	Withhold at least 3 days before surgery[†] and restart ASA when the risk of bleeding related to surgery has passed (ie, 8-10 days after major noncardiac surgery)
β-Blocker	Continue the β-blocker during the perioperative period; however, if a patient's systolic blood pressure is low before surgery, physicians should consider decreasing or holding the dose of the β-blocker before surgery
ACEI/ARB	Withhold ACEI/ARB 24 hours before noncardiac surgery and restart ACEI/ARB on day 2 after surgery, if the patient is hemodynamically stable
Statin	Continue the statin during the perioperative period
Smoking	Discuss and facilitate smoking cessation (eg, nicotine replacement therapy), ideally starting ≥ 4 weeks before surgery
Initiation of new medications and coronary revascularization before noncardiac surgery	
ASA	Do not initiate ASA for the prevention of perioperative cardiac events
β-Blocker	Do not initiate a β-blocker within 24 hours before noncardiac surgery
α_2-Agonist	Do not initiate an α_2-agonist for the prevention of perioperative cardiovascular events
Calcium channel blocker	Do not initiate a calcium channel blocker for the prevention of perioperative cardiovascular events
Coronary revascularization	Do not undertake preoperative prophylactic coronary revascularization for patients with stable coronary artery disease

ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker; ASA, acetylsalicylic acid.

* This applies to patients age 45 years of age or older or 18-44 years of age with known significant cardiovascular disease (ie, history of coronary artery disease, cerebral vascular disease, peripheral vascular disease, congestive heart failure, or a severe obstructive intracardiac abnormality [eg, severe aortic stenosis, severe mitral stenosis, or severe hypertrophic obstructive cardiomyopathy]) undergoing noncardiac surgery requiring hospital admission.

† Except in patients with a recent coronary artery stent and patients undergoing carotid endarterectomy.

Other medication categories:
Diuretics: typically hold unless concern for CHF/volume overload
Steroids: consider stress dose steroids perioperatively for patients where there is a concern for relative adrenal insufficiency

- Prednisone >20mg x 3weeks
- Cushingoid appearance
- Confirmed 1° or 2° adrenal insufficiency

Respirology

Approach to Dyspnea

Updated March 2023

Approach: Ask for Help (Expertise, Hands) anytime along the continuum

1. **1º Survey – ABCD:** Airway threatened? Stat vitals! Possible Hypercarbia (↓LOC, signs of fatigue). **Rapid Cardiac and Resp Exam 1st** (narrows DDx)
2. **1º Resus: Intubation?** Suction? O_2 (Venturi, NRB) + NIPPV?
3. **1º Adjuncts: IV**, Monitor, Crash Cart. STAT tests: **ECG, CXR**, VBG (ABG if have time & hands), Bloodwork.
4. **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.
5. **Baseline Level of Dyspnea:** Ask about multiple activities to be certain. Use mMRC to quantify – standardized scale for communication.
6. **Progression:** Rate of deterioration. Rapid? Or else ask how was the breathing half a year ago, a year ago, etc…
7. **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 infiltrate 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. Elucidate cause: volume overload, ACS, Dysrhythmia, Valvulopathy.
- **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	D/C if improved
Moderate	Strenuous effort to breath, Partial response to β-agonist, normal CO_2	Bronchodilators + Glucocorticoid	Ward, D4ICU
Severe	Struggling, Diaphoretic, Difficult to speak, Ⓝ/↑CO_2	Get Help, Intubation Bronchodilators + Glucocorticoid + $MgSO_4$	ICU
Near Death	Exhausted, ↓LOC, ↓RR/HR, Silent Chest, Ⓝ/↑CO_2		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
  - Do not use NIPPV in asthma – if seriously considering this, pt requires intubation
  - 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, Ⓝ/↑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 for mod to severe COPD with >1 AECOPD in the last yr

6. Frequent exacerbations: Azithromycin 250mg PO QD
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	

Inhale 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: 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

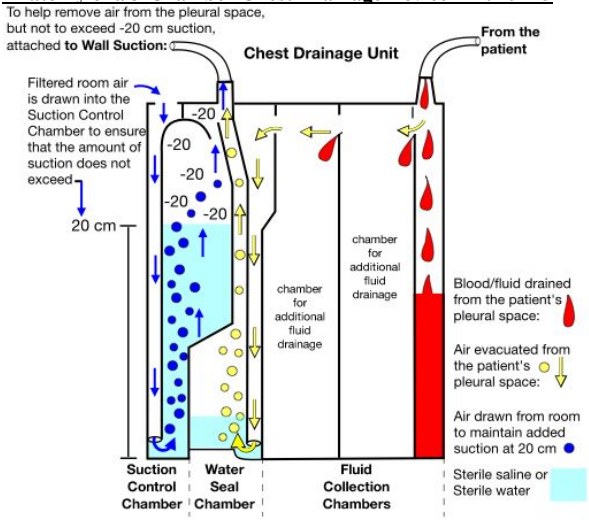
b. Pleural Ultrasound, CT Thorax: inferring the diagnosis based on the complexity of the pleural space and fluid characteristics: loculation, fibrinous strands, echogenic material in the pleural fluid.

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:** collagen vascular disease (SLE, RA), subdiaphragmatic irritation (pancreatitis), PE with infarction, Chronic CHF, Trauma, Cancer	

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. ✓ 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. ✓ 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 "Ⓝ" 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

Author: Dr. Simone Ten Kortenaar *Updated June 2020*

Rheumatological Blood Tests

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

ANA: ANA is non-specific and is 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

ANCA: classically associated with small-vessel vasculitides: granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), and eosinophilic granulomatosis with polyangiitis (EGPA). There is the potential for overlap. Polyarteritis nodosa (PAN) is NOT associated with ANCAs.

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

Anti-CCP: used in conjunction with rheumatoid factor (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.

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

dsDNA: specific for SLE, included in KGH ENA panel. 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.

ENA: The 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 the following:

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), primary biliary cholangitis.

Arthropathies

- **Seropositive** – Polymyositis/dermatomyositis, systemic lupus erythematosus (SLE), scleroderma, rheumatoid arthritis (PSSR)
- **Seronegative diseases** - Spondyloarthropathies including ankylosing spondylitis, reactive arthritis, Psoriatic arthritis, enteric (IBD) arthritis; seronegative rheumatoid arthritis
- **Vasculitides** – particularly ANCA vasculitis
- **Crystal-induced** – Gout, pseudogout (calcium pyrophosphate deposition disease - CPPD), hydroxyapetite deposition disease
- **Infectious** – also known as septic joints. Can be further classified as gonococcal versus non-gonococcal (S. aureus, GAS, S. pneumonia, GNB, anaerobes), 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 – 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 MTP/MCP for example.

Oligoarthritis (2-4 joints involved) 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 (4 or more joints involved) – the classic presentation of seropositive arthropathies such as RA and SLE involves symmetrical small joint involvement (for example, MCPs of the hands, wrists, MTPs of the feet, 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, negatively birefringent crystals
  - Pseudogout: blue, 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

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 dsDNA antibody
- Whenever SLE patients are unwell, rule out disease flares as a cause. C3, C4, and dsDNA may guide etiology + UA and renal function

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

Systemic Sclerosis and 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 of the skin, Raynaud's phenomenon, gut dysmotility, sclerodactyly, and telangectasias. lcSSc tends to affect the face/extremities distal to the elbows and knees, while dcSSc affects the proximal extremities above the knees/elbows.

Scleroderma renal crisis (SRC) can be life-threatening without timely diagnosis and managment. Features:

- AKI
- HTN > 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: captopril, titrate to BID or TID. AVOID GLUCOCORTICOIDS—can be potential trigger for SRC.

Vasculitis

Large vessel – Giant Cell Arteritis Manifestations

- Age >50
- Fronto-temporal heqacache, scalp tenderness, jaw claudication
- Visual changes are the most severe manifestation of GCA. 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 prednisone for 2-4 weeks with a gradual taper over 6 months.

Small-vessel – ANCA vasculitides Manifestations

- 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

Palliative Care

Author: Dr. Hannah O'Neill *Updated June 2020*

The palliative care handbook is an excellent resource for how to manage patients at end of life. This chapter is a brief summary of what to do overnight/if you do not have your handbook on hand.

Goals of care should ideally by addressed by the day team at the time of admission. Unfortunately things can change unexpectedly and you may have to address goals of care overnight. It is important to discuss GOC as early as possible as prognosis is unpredictable.

Signs of End of life include-
Decreased oral intake
Sleeping more
Cheynne-stokes breathing pattern

A way to help answer a family's question about prognosis is that generally things deteriorate in a step wise manner, with the caveat that every patient is different. First in a change in activity month to month, then week to week, day to day and finally hour to hour.

Basic end of life orders may include some of the following;

1. Discontinue Vitals
2. Discontinue bloodwork
3. Discontinue IV fluids/feeds
4. Hydromorphone 1mg po q1h regular/PRN for dyspnea/pain
5. Hydromorphone 0.5-1mg SQ q1h regular/PRN for dyspnea/pain
6. Midazolam 1-2mg PO/SQ q4h regular/PRN for agitation
7. Metoclopramide 10-20mg PO/SQ q4h regular/PRN for nausea/vomiting
8. Scopolamine 0.2-0.8mg SQ q4h Regular/ PRN for secretions
9. Nozinan 2.5-12.5mg q4h PO/SQ for refractory nausea/agitation

Discontinuing Vitals and bloodwork can be discussed with the family, they often worry that this will mean we stop caring for their loved one. It is important to discuss why we check them and what would we do if we found an adverse vital sign/blood work. When the focus is on comfort care we use other signs to guide our management.

Discontinuing feeds and IV fluids can also be a cause for concern with families. It is very human to want to feed those who are sick. There is no evidence that feeds and IV fluids are beneficial in end of life, they have not been shown to prolong life and could potentially worsen secretions. While Scopolamine is used to minimize secretions it can be sedating and does not affect secretions that are already present.

It's important to ask to be informed if the nurse or family members are concerned that the patient is uncomfortable. Escalate the dosing or frequency of medications as required. Involve the palliative care team if the patient is complex and end of life measures need to be escalated. The always have someone on call for palliative care to help.

Toxidromes

Principles of Mx:

1. **Stabilize**: ABCDEFG
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 (CO)
6. **Eliminate:** Urine alkalinization: useful for weak acids (eg. ASA). Hemodialysis: small molecules. Hemofiltration: mid-sized molecule.

Elucidating the Toxin(s): Use ALL of the following lest misdiagnosis

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: fast acting, sustained release
- **Amount:** dose on the container; pill count; any regurgitated
- **Timing:** one-time bolus vs interval ingestion

b. **Syndromic Pattern Recognition:**
- **Anticholinergic:** Atropine, scopolamine, TCAs, Diphenhydramine. Features: "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:** Organophosphates, carbamates. Features: Diarrhea, Urination, Miosis, Bronchospasm, Bronchorrhea, Emesis, Lacrimation, Salivation
- **Sympathomimetic:** Cocaine, amphetamines, caffeine, nicotine, ASA. Features: CNS excitation, ↑HR, ↑BP, fever, mydriasis, diaphoresis
- **Narcotic and Sedative/Hypnotic:** Alcohols, opiate, benzo., barbiturate. Features: 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 – calc. Osmol
- Calc. 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 stability, 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. Severely poisoning occurs at lower (or normal) serum levels for chronic intoxication.

Mx:

1. **Principles of intoxication Mx**
2. **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
3. **Glucose supplementation:** neuroglycopenia may result in ΔLOC despite Ⓝ serum glucose.
4. **Serum and urine alkalinization**:** (Alkalemia from respiratory alkalosis is not a contraindication as often have base deficit)
   - 3 amps of $NaHCO_3$ (each amp: 50mEq $NaHCO_3$) mixed into 1L of D5W & infused at 150mL/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 ↓$[K^+]$ counteracts the alkalinization process
   - **Close monitoring:** salicylate levels and ABG frequently.

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 Ⓝ serum levels though end organ manifestation

Acetaminophen (APAP) 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: Fatal dose for adult >10g.

Clinical Manifestations: <24hrs: non-specific → 24-72hrs: RUQ pain, oliguria → 72-96hrs: acute hepatic failure, AKI (proteinuria, hematuria, ATN)

Investigations:
- APAP level: 4thhr (or anytime after – whichever comes first) and 20hrs after the previous one.
- Liver enzymes and synthetics.
- Modified King's College Criteria (lactate at 4th and 12thhr after rhesus; pH at 12th hr after resus; INR, Cr)

Mx:

1. **Principles of intoxication Mx**
2. **Indication for N-acetylcysteine antidote:** Get Help from Poison Control
   (a) APAP level at ≥4thhr from ingestion is above "Tx line" on nomogram (If you know pt ingested >10g start before waiting 4hr)
   (b) Hx of APAP ingestion with any hepatic injury
   (c) Serum level >66umol/L and unknown time of ingestion
   (d) Delayed (>24hrs) presentation and hepatic injury**
3. **NAC Protocol:** Protocol for NAC infusion under "KGH Parenteral Pharmacy" website; or ask Poison Control to fax one over
   - **NAC IV infusion (20hr protocol)**: 150mg/kg/hr x 1hr → 50mg/kgh/hr x 4hr → 6.25mg/kg/hr x 16hrs
     - **Anaphylactoid rxn (10%):** If rash & mild symptoms, hold infusion & Tx with Benadryl® 50mg IV + Ranitidine 150mg IV x 1 ± methylprednisone 125mg IV x 1. 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 <66umol/L, and AST&ALT normalize, or declining
4. **Consider referral for liver transplantation – Modified Kings College Criteria calculation:** (pH<7.3 or lactate >3mmol/L 12hrs after resus; or Grade 3-4 hepatic encephalopathy PLUS Cr> 300umol/L PLUS INR>6.5; lactate >3.5mmol/L 4hrs after resus)
5. **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 (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 drinker: 5.5-8mmol/L/hr.

Note: 0.6oz EtOH = 13.6g EtOH = 1 standard drink

Toxic Dose: >66mmol/L – coma & resp. depression (though the chronic abuser may tolerate higher levels)

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 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 dysfxn.

***Dosing:** Be ready to give high doses of BZD. Patients who consume high quantities of EtOH chronically and/or in moderate to severe withdrawal is 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 (See B67)
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 see 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:

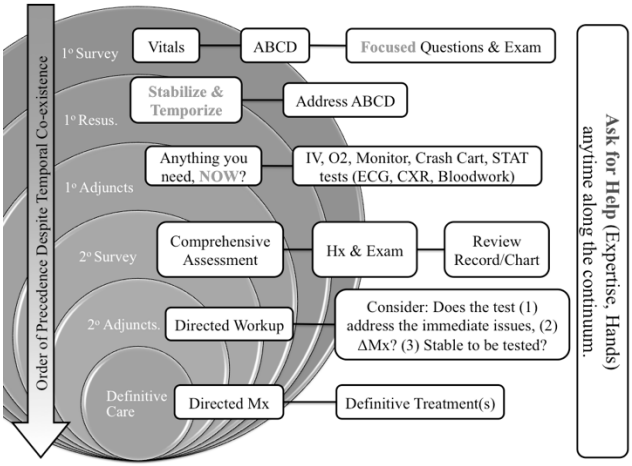
1. Principles of toxicity Mx
2. **Indications for dialysis:** (a) Serum [Li^+] <4mmol/L regardless of clinical status; (b) Serum [Li^+] >2.5mmol/L with neuro sequelae, anticipated renal insufficiency
3. Hydration: render euvolemic with NS at high infusion rates – aggressive; If ↑[Na^+], replace free H_2O.

PEARLS:

* Serum [Li^+] DOES NOT correlate with tissue level; watch for rebound due to slow equilibration

General Approach to an Unstable Patient

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 – 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% 02 or 6L/min

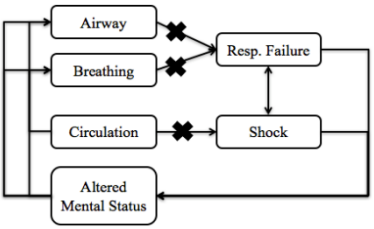
Circulation:
- SBP <90 mmHg or >200 mmHg or a decrease of >40 mmHg
- Heart rate <40 or >130

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 knows the patient

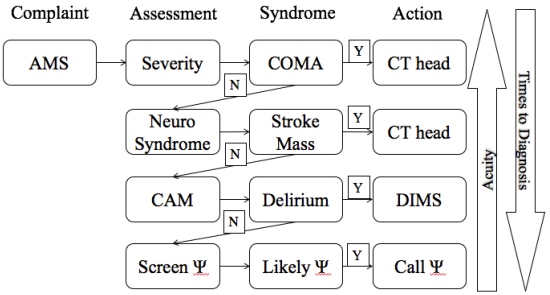


1. **1º Survey:** ABCD.
2. **1ºResus:** Fix ABC 1st as they kill and can cause AMS.
   - ABC: Whether the ABC problem is/are caused by or resulting in AMS, fix them 1st. Reasons: (1) They kill; (2) They cause AMS; (3) May reverse AMS.
   - Non-ABC: Hypoglycemia (D50W), Opiate (Naloxone), Benzo (flumazenil).
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 Metabolism, Δ 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!

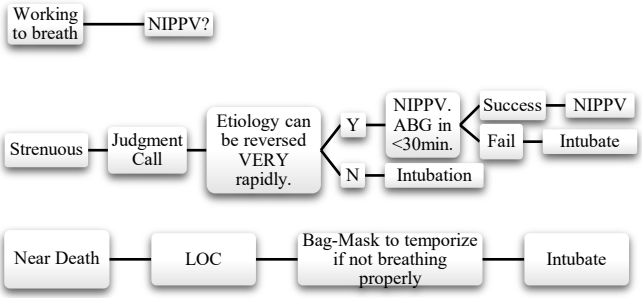
4. Severity: quickly determine which category does the patient fit

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

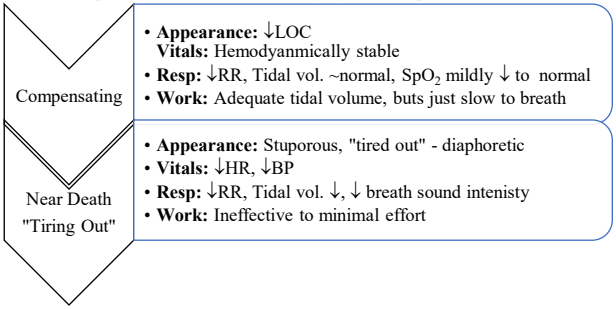
- Blood Work: CBC, -lytes + Cr, VBG (ABG ASAP when there are free hands), Trop
- Imaging: CXR (PoCUS if you able to employ)
- Auxiliary: ECG

7. Directed and Definitive Mx

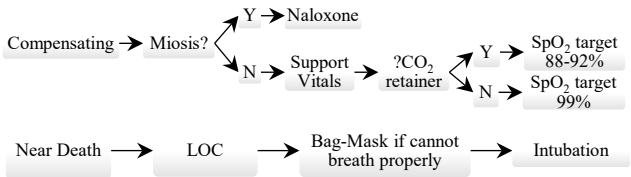
Approach to Bradypnea

Overarching Schema:

1. **General Appearance?** ↓LOC? "Tired out" look? (2o to ↑ work of breathing?) Miosis? (Opiate use?)
2. **1º Survey:** ABCD Assessment
   - **A.** Tongue – obstructing? (Gudedel) Gurgling? (suction secretion) Protecting airway?
   - **B.** Cyanosis? Hypoxic? Shallow breathing? (↓ Tidal vol.)
   - **C.** Hypotensive – resuscitate accordingly
   - **D.** Drowsy? Stuporous? (Tolerate Guedel/LMA + Bagging?)
3. **Resource Evaluation:** Expertise, Equipment, Manpower – Get Help
4. **Severity:** quickly determine which category does the patient fit



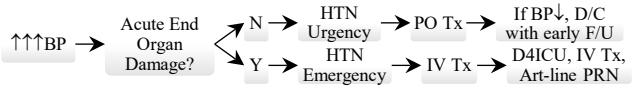
5. **1º Resus:**



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:**
   - **Opiate toxicity** (most common cause in hospital): naloxone 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<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 **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 20mcg/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

DOC: 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:** Enaliprat 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 oxyen in blood
Hypoxia – decreased oxygen in tissues

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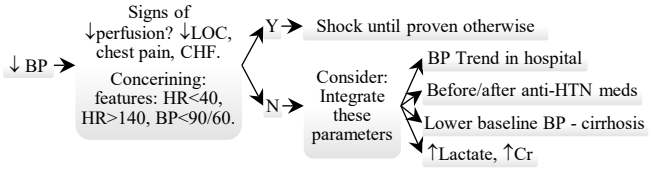

1. **General Appearance?** ↓LOC, Chest pain → end-organ ↓ perfusion
2. **Survey:** ABCD Assessment
3. **Resource Evaluation:** Expertise, Equipment, Manpower – Get Help
4. **1º Resus:** NS 1L IV bolus ASAP – wide open with pressure bag around.
5. **1º Adjuncts:** IV access – large bore peripheral IV. 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 (i.e. sepsis, anaphylaxis, spinal injury (above T4), drugs)
Obstructive	Impairment of venous return to right atrium (i.e. tension pneumothorax, PE, pericardial tamponade)
Cardiogenic	Primary cardiac insult causes decreased cardiac output (i.e. heart failure, ACS, valvular heart disease, obstructive causes of shock)
Neurogenic	Form of distributive shock; may have hypotension AND bradycardia

Endocrine	Form of distributive shock (i.e. myxedema coma, Addisonian crisis, anaphylaxis)

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
Loss	0-15%	15-30%	30-40%	>40%
BP	-	-	Decreased	Decreased
HR	-	>100	>120	>140
RR	-	20-30	30-35	>35
CNS	-	Anxious	Confused	Lethargic
Urine Output(cc/hr)	>30	20-30	5-15	Negligible

Directed Mx: based on Shock Mechanism

Mechanism	**Goal of resus: ↑DO_2 (O_2 delivery)** = ↑CO (cardiac output) x ↑CaO_2 (arterial O_2 content)
Hypovolemic	(1) Volume Expand: Crystalloid (and/or Blood)
Distributive	(1) Volume Expand → (2) Vasopressor
Cardiogenic (refers to ♥genic 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 ♥genic shock 2º to causes extrinsic to the ♥ → 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

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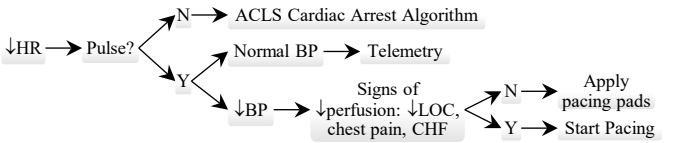


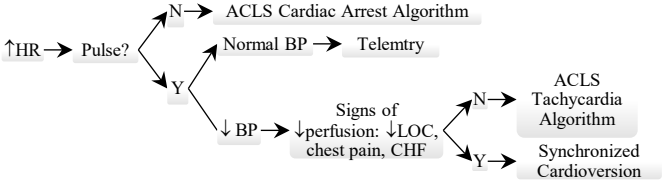
1. **General Appearance?** ↓LOC? Chest pain? Dyspnea?
2. **1º Survey:** ABCD Assessment – Signs of Hypoperfusion
   - **A.** Tongue obstructing? (Gudedel) Gurgling? (suction secretion)
   - **B.** Cyanosis? Hypoxic? Signs of CHF?
   - **C.** Hypotensive?
   - **D.** Drowsy? Stuporous? (Tolerate Guedel/LMA + 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 → Pacing Mode. Analyze the rhythm – any obvious heart blocks? Start pacing if blocks + hypoperfusing (↓LOC).
   - **See ACLS Algorithm for Bradycardia**
5. **1º Adjuncts:** IV access.
6. **2º Survey and Directed Workup:**
   - ECG ASAP: Mobitz II 2º HB, 3º HB, ischemia
   - Lytes+Cr, Ca^{+2}, Mg^{+2}, PO_4^{-3}, 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 maybe required – eg.) Midazolam 2mg IV x 1, Fentanyl 25-50mcg IV x 1.

Approach to Tachycardia

Overarching Schema:



1. General Appearance? ↓LOC (Hypoperfusion)? Chest pain (Hypoperfusion, or cause of bradycardia)?

2. 1º Survey: ABCD Assessment – Signs of Hypoperfusion

A. Tongue obstructing? (Gudedel) Gurgling? (suction secretion) Protecting airway?

B. Cyanosis? Hypoxic?

C. Hypotension?

D. Hypotensive?

E. Drowsy? Stuporous? (Tolerate Guedel/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^{+2}, Mg^{+2}, PO_4^{-3}, Trop, CK, Lactate
- **Consult Cardio.** for further Mx

7. Directed and Definitive Mx

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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 significantly lower in survivors of critical illness/their family members

Mild improvements in cognitive, psychiatric, and physical impairments within 12 months of discharge

Elevated risk of death in patients after critical illness, particularly in first 3-6 months after ICU admission

Culturally Safe and Sensitive Care

Authors: Andrew McNaughton, Justin Boyle

Culturally safe and sensitive care is a 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 **practising** 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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