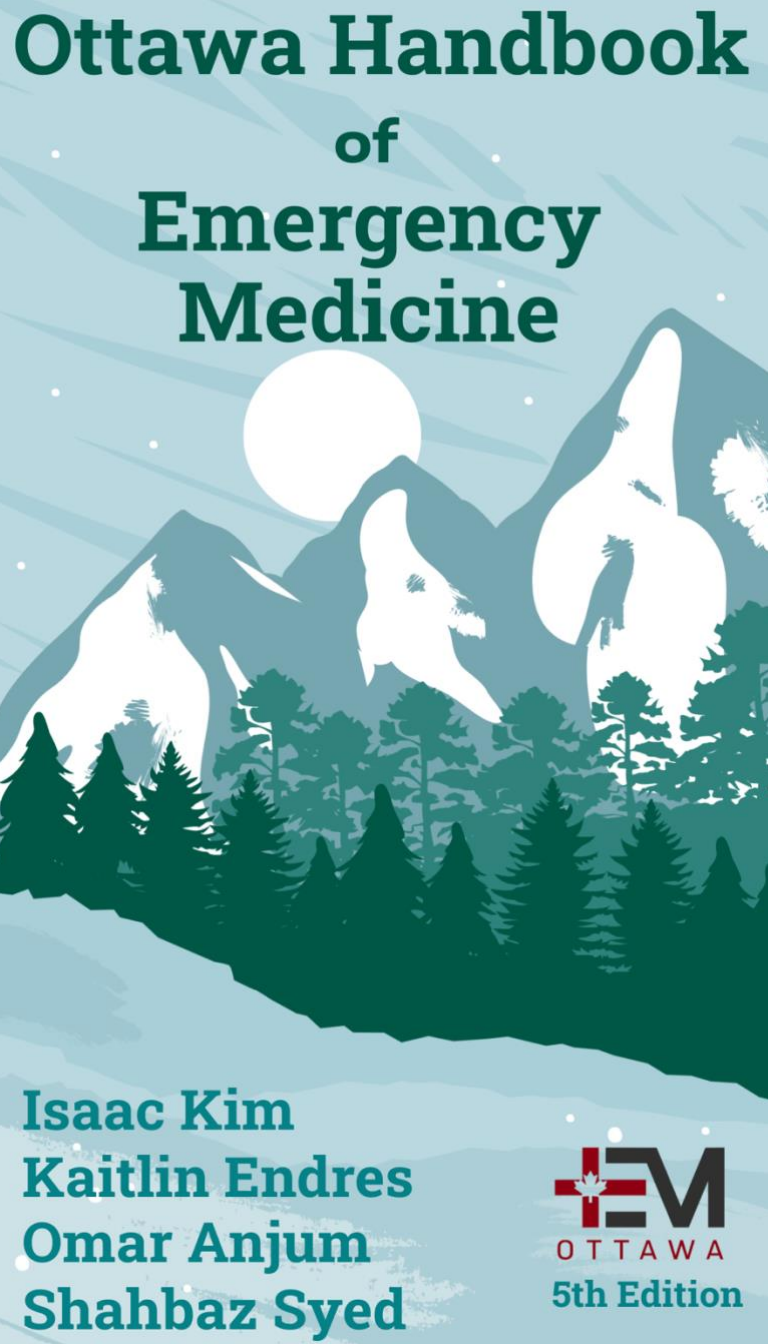


Ottawa Handbook of Emergency Medicine



Isaac Kim
Kaitlin Endres
Omar Anjum
Shahbaz Syed


OTTAWA
5th Edition

Authors and Editors

Omar Anjum, MD, MEng, FRCPC

Author and Editor

Department of Emergency Medicine, University of Ottawa

Isaac Kim, BSc, MD

Co-Author and Associate Editor

Department of Emergency Medicine, University of Ottawa

Kaitlin Endres, BSc, MD

Co-Author and Associate Editor

Department of Emergency Medicine, University of Ottawa

Shahbaz Syed, MD, FRCPC

Faculty Lead and Associate Editor

Department of Emergency Medicine, University of Ottawa

Current Contributors

Nick Costain MD, FRCPC

Jesse McLaren MD, CCFP-EM

Eric Mutter MD, FRCPC

Previous Contributors

Laura Olejnik MD

Celina DeBiasio, BSc

Taylor Young, BHSc, MD

Warren Cheung, MD, FRCPC

Miguel Cortel-Leblanc, MD, FRCPC

George Mastoras MD, FRCPC

Brandon Ritcey MD, FRCPC

Brian Weitzman MDCM, FRCPC

Daniel James, MD, FRCPC

Jim Yang MD, FRCPC

Sarah Addleman MD, CCFP-EM

Luke Edgar, MD

Stella Yiu MD, CCFP-EM

Rebecca Seliga, BSc, MD

Nathalie Côté, MD, FRCPC

Lisa Fischer, MD, FRCPC

Peter Johns MD, FRCPC

David Gruber MD, CCFP-EM

K. Jean Chen MD, CCFP-EM

Michael Ho, MD, FRCPC

Jennifer Leppard MD, CCFP-EM

Matthew Lipinski MD, FRCPC

Fifth Edition: Published December 2023

This book can be downloaded from <https://emottawablog.com>

All rights reserved. This work may not be copied in whole or in part without written permission of the authors. While the information in this book is believed to be true and accurate at the time of publication, neither the authors nor the editors nor the contributors can accept any legal responsibility for any errors or omissions that may be made. Readers are advised to pay careful attention to drug or equipment information provided herein. The primary intended readers are medical students, junior residents and paramedics; as such it is expected that a supervising physician is consulted prior to initiation of treatment and management discussed in this handbook.

Preface

Introduction

Dear readers,

This handbook is a student-driven initiative developed in order to help you succeed on your emergency medicine rotation. It provides concise approaches to key patient presentations you will encounter in the emergency department. This guide has been peer-reviewed by staff physicians to ensure evidence is up-to-date and accurate. Based out of Ottawa, our hope is that this resource will benefit clerkship students and help bridge the emergency medicine knowledge gap from pre-clerkship to clinical practice.

Sincerely,

Omar Anjum, MD, MEng, FRCPC

Author and Editor

How to Use this Guide

Topics are subdivided according to **background**, **assessment**, **investigations**, and **management**.  indicates there are images.

[EMOttawa Blog Post](#): click here on applicable pages to be hyperlinked to a related EMOttawa Blog post for more info on this topic.

Background
This section provides common definitions, pathophysiology, etiology or risk factors for certain conditions. Differential diagnoses are also discussed (“Symptoms Approach” section).
Assessment
Common historical and physical exam features are mentioned here. Diagnostic criteria or techniques/methods used to aid in diagnosis may also be noted.
Investigations
Relevant labs, radiological evaluation and adjunctive tests are mentioned for consideration of diagnostic workup.
Management
General and disease-specific management approaches are discussed. Disposition and discharge criteria may also be noted.

Key References: Used for further reading. Some sources are provided because they are deemed useful.

Table of Contents

Resuscitation

- Airway
- Breathing
- Circulation
- Trauma

Symptoms Approach

- Syncope
- Altered Mental Status
- Headache
- Head Trauma
- Seizures
- Shortness of Breath
- Chest Pain
- Chest Pain Risk Stratification
- Abdominal Pain
- Pelvic Pain
- Back Pain
- Vaginal Bleeding
- Red Eye
- Monoarthritis & Polyarthritis

Selected Emergencies

- Anaphylaxis
- Asthma
- Chronic Obstructive Pulmonary Disease
- Myocardial Infarction
- Congestive Heart Failure
- Cardiac Dysrhythmias
- Pericarditis & Myocarditis
- Deep Vein Thrombosis
- Pulmonary Embolism
- Gastrointestinal Bleeding
- Stroke
- Transient Ischemic Attack
- Wound Management
- Diabetic Emergencies
- End-of-Life Care
- Sepsis
- Acid-Base Disorders
- ENT Emergencies
- Electrolyte Disturbances
- Urologic Emergencies
- Environmental Injuries
- Orthopedic Emergencies
- Pediatric Orthopedic Emergencies
- Pediatric Fever
- General Surgery Emergencies
- Toxicological Emergencies
- Vascular Emergencies
- Psychiatric Emergencies
- Thyroid Emergencies

Pain Management

- Clinical Decision Rules
- Risk Stratification Scales
- Advanced Cardiac Life Support
- Point of Care Ultrasound
- Approach to the Chest X-Ray
- Approach to the ECG

Resuscitation

Airway

Decision to Intubate

Failure to maintain or protect airway (e.g. low GCS, airway trauma)

Failure to ventilate/oxygenate (e.g. low or declining SpO₂, rising pCO₂)

Anticipatory (e.g. trauma, overdose, inhalation injury, anaphylaxis, inc. WOB)

Assessment

Difficult Bag-Valve Mask Ventilation “BOOTS”
B = Beard; O = Obese; O = Older; T = Toothless; S = Snores/Stridor
Difficult Laryngoscopy
Look for gestalt signs. Evaluate the 3-3-2 rule. Check for signs of obstruction, swelling, trauma. Assess neck mobility. Upper lip bite test: Concern if patient cannot bite past vermilion border
Difficult Supraglottic Device “RODS”
R = Restricted mouth opening; O = Obstruction, Obese D = Disrupted or Distorted anatomy; S = Stiff lung or cervical Spine

Airway Techniques

Temporizing Measures
Chin lift/jaw thrust, BVM, suctioning, nasal airway, oral airway, LMA
Definitive Airway
Orotracheal/nasotracheal intubation, surgical airway (percutaneous or open cric)

Airway Methods

Rapid Sequence Intubation (RSI)
Awake intubation w/ topicalization
Ketamine-facilitated intubation
Crash airway without meds

Rescue Airways

LMA
Cricothyroidotomy

Rapid Sequence Intubation (6Ps)

Preparation
Prepare equipment and medications, use checklist if available
Pre-Oxygenation
100% FiO ₂ , employ PEEP valve to improve recruitment
Pre-Treatment (Optional)
Increased ICP: fentanyl 3µg/kg Hypotension: fluids/vasopressors (infusion or push-dose) Acidosis: bicarb (controversial), consider maintaining spontaneous respiration Anxiolysis: midazolam 2-4mg
Positioning
Sniffing position, ramped position if obese, adjust bed height
Paralysis with Induction
Administration of sedative (i.e. Ketamine, Propofol, Etomidate) followed by muscle relaxant if indicated (i.e. Succinylcholine or Rocuronium)
Place Tube with Proof
Intubate patient and confirm tube placement (continuous waveform EtCO ₂)
Post-Intubation Management
Post-intubation analgesia, ongoing sedation, ventilator management, further resuscitation.

Airway Checklist

Breathing

Definitions

Acute respiratory failure = $pO_2 < 50\text{mmHg}$ +/- $pCO_2 > 45\text{mmHg}$
Hypoxic Respiratory Failure
Diffusion problem: pneumonia, ARDS V/Q mismatch: PE, Asthma, COPD Shunt Low ambient FiO_2 : high altitude Alveolar hypoventilation
Hypercarbic Respiratory Failure, Normal Lungs
Disorder of respiratory control: overdose, brainstem lesion, CNS disease Neuromuscular disorders: muscular dystrophy, GBS, Myasthenia Gravis, ALS Anatomic: trauma, ankylosing spondylitis, kyphosis/severe scoliosis
Hypercarbic Respiratory Failure, Abnormal Lungs
Increased airway resistance: AECOPD, asthma exacerbation Decreased gas exchange: scarring, IPF

Assessment

Look	Listen	Feel
Mental status, colour, chest wall movement, accessory muscle use Paradoxical abdominal movement	Auscultate for breath sounds Signs of obstruction Air entering or escaping Wheeze and stridor	Tracheal deviation, crepitus, flail segments, chest wounds

Investigations

Labs: CBC, electrolytes, cardiac enzymes +/- D-dimer +/- BNP, VBG

Tests: POCUS, CXR +/- CT Chest

Management of Breathing

Spontaneously Breathing Patient
Nasal prongs Face mask, Non-rebreather face mask High flow nasal oxygenation (i.e. MaxTech)
Temporizing Measures for Inadequate Ventilation
Bag-valve mask +/- nasal airway CPAP/BiPAP: acute exacerbations of CHF, COPD, asthma
Definitive Measures for Inability to Maintain/Protect Airway
Oro-tracheal intubation Surgical airway
Additional Modalities
Needle or finger thoracostomy for tension pneumothorax Chest tube to drain pleural effusion/hemothorax/pneumothorax

Circulation

Causes of Shock

Hypovolemic Shock	Hemorrhage GI losses	Third spacing Dehydration Over diuresis
Obstructive Shock (Intra-Thoracic)	Pulmonary embolism Cardiac tamponade Tension pneumo	Valvular dysfunction Congenital heart disease Air embolism
Distributive Shock (Vasodilation)	Septic shock Anaphylactic shock Neurogenic shock	Drug overdose Adrenal crisis
Cardiogenic Shock	ACS Cardiomyopathy	Cardiac structural damage Dysrhythmias

Assessment

Clinical symptoms and signs suggestive of shock	
Vitals: ↑HR, ↓BP, ↑RR	High initial lactate
Urine Output <0.5mL/kg/hr	Skin mottling
Capillary refill time >3 secs	Altered mental status

Investigations

Labs: CBC, electrolytes, BUN, Cr, LFTs, Tnl, VBG, lactate

Tests: CXR, ECG, POCUS - RUSH exam (cardiac, IVC, lungs, aorta)

Management

Perfusion Goals
Urine Output >0.5mL/kg/h, MAP >65mmHg, improved mentation, improved cap refill time, lactate clearance
Hemorrhagic Hypovolemic Shock: fill the tank
Control hemorrhage (tourniquets, direct compression, pelvic binders) Fluids until blood available, balanced transfusion (1:1:1 of pRBCs:platelets:FFP)
Obstructive Shock: alleviate obstruction
Tension pneumothorax: needle decompression then chest tube Cardiac tamponade: IV crystalloids, pericardiocentesis PE: IV crystalloid, inotropes, thrombolysis
Distributive Shock: source control, squeeze the pipes
Anaphylaxis: Epinephrine IM, IV fluids, antihistamines if symptomatic, corticosteroids Sepsis: Broad-spectrum antibiotics, IV fluids +/- norepinephrine
Cardiogenic Shock: support forward flow
Norepinephrine 0-20µg/min, dobutamine 0-5µg/kg/min Treat underlying cause: cath lab, mechanical circulatory support (IABP, Impella, VAD, ECMO), heart transplant
Cellular Toxins
Antidotes for various toxins (see Toxicology)

Trauma Resuscitation

Primary Survey

1. Airway	3. Circulation
Assess patency of airway, look for obstruction (blood, emesis, teeth, foreign body), ensure C-spine precautions, airway management	Assess LOC, signs of shock (HR, BP, skin color, urine output, base deficit), sources of bleeding (external, chest, abdomen, pelvis, femur)
2. Breathing	4. Disability
Expose chest, assess breathing, auscultate for breath sounds, rule out tension pneumothorax	GCS assessment Neurological evaluation including pupils
5. Exposure/Environment	
Fully expose and assess patient Logroll patient to inspect for injuries, spinous tenderness and rectal exam for high-riding prostate and tone Keep patient warm to prevent hypothermia	
Adjuncts	
eFAST Exam: subxiphoid pericardial window, perisplenic, pelvic/retrovesical, bilateral anterior lung Portable X-ray: chest, pelvis, grossly deformed limbs ECG: evaluate for dysrhythmias	

Investigations

Bloodwork: CBC, lytes, BUN, Cr, glucose, lactate, INR/PTT, fibrinogen, B-hCG, tox bloodwork, T+C, U/A

Imaging: CT (selective vs. pan-scan) for stable patients; unstable patients may require emergent OR

Trauma Triad of Death

Coagulopathy
Hypothermia
Acidosis

Management

General Resuscitation
Immediate hemorrhage control (Stop the Bleed) Blood transfusion: balanced resuscitation to avoid dilutional coagulopathy Tranexamic acid: 1g IV bolus then 1g IV over 8 hours Consider permissive hypotension
Head Trauma
Seizure management/prophylaxis, treat suspected raised ICP, neurosurgical intervention for severe head injury/bleeds
Spinal Cord Trauma
Immobilization, treat neurogenic shock (goal MAP >85), consult spine service
Chest Trauma
Airway management, chest decompression, resuscitative thoracotomy in arrest, surgery for life-threatening injuries
Abdominal Trauma
Laparotomy for hemodynamically unstable and hollow organ injuries
Orthopedic Injuries
Reduce and immobilize when possible, irrigate open fractures, assess for neurovascular and skin compromise, adequate analgesia, consult ortho

Symptoms Approach

Syncope

Definition: sudden and transient loss of consciousness and loss of postural tone accompanied by a rapid return to baseline

Pathophysiology: dysfunction of both cerebral hemispheres or the brainstem (reticular activating system) usually from hypo-perfusion

Differential Diagnosis

Cardiac	Rhythm Disturbances: dysrhythmias, pacemaker issues Structural: outflow obstruction (aortic stenosis, HOCM), MI Other CV diseases: dissection, cardiomyopathy, PE	
Non-Cardiac	Reflex (neurally mediated)	Vasovagal: sensory or emotional reactions Orthostatic: postural, dehydration Situational: coughing, straining Carotid sinus pressure: shaving Subclavian steal: arm exercises
	Medications	CCBs, B-blockers, digoxin, insulin QT prolonging meds Drugs of abuse
	Focal CNS Hypoperfusion	Hypoxia, epilepsy, dysfunctional brainstem

Assessment

History: syncope character (ask about exertion!), cardiac risk factors, comorbidities, medication/drug use, family history, orthostatic symptoms
 Rule out seizure/stroke/head injury

Physical Exam: cardiac exam (murmurs, rate), CNS exam

Investigations

Labs: CBC, glucose, lytes, extended lytes, BUN, Cr, CK/Tnl, B-hCG

Heart rate/rhythm	Tachyarrhythmia, bradyarrhythmia
Electrical conduction	PR (WPW, AV block), BBB and bifasicular block, long QT
Axis	Bifasicular block, new right axis from PE
R-wave progression	Early from WPW type A, late from cardiomyopathy
Tall/small voltages	Small (pericardial effusion), large (HCM, AS)
ST/T changes	PE, STEMI, Brugada, ARVD

Management

General
ABCs, monitors, oxygen, IV access
Cardiogenic Syncope
Consult cardiology for workup +/- permanent pacemaker
Non-Cardiogenic Syncope
Benign causes or low-risk syncope: discharge with GP follow-up Consider outpatient cardiac workup
Risk Stratification Prediction Rules
Canadian Syncope Risk Score

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 15. CMAJ 2011; 183(15): 1694-1695. CMAJ 2016; 188(12): E298.

[Canadian Syncope Risk Score](#)

Altered Mental Status

Definition: decrease in LOC caused by either diffuse CNS dysfunction (toxic/metabolic causes) or primary CNS disease

Differential Diagnosis

Drugs
Abuse: Opiates, benzodiazepines, alcohol, illicit drugs Accidental: Carbon monoxide, cyanide Prescribed: β -blockers, TCAs, ASA, acetaminophen, digoxin Withdrawal: Benzodiazepines, EtOH, SSRIs
Infection
CNS: meningitis, encephalitis, cerebral abscess Systemic: sepsis, UTI, pneumonia, skin/soft tissue, bone/joint, intraabdominal, iatrogenic (indwelling lines or catheter), bacteremia
Metabolic
Kidneys: electrolyte imbalance, renal failure, uremia Liver: hepatic encephalopathy Thyroid: hyper or hypothyroid Pancreas: hypoglycemia, DKA, HHS
Structural
Bleeds: ICH, epidural hematoma, subdural hematoma, SAH Brain: Stroke, seizures, surgical lesions, hydrocephalus Cardiac: ACS, dissection, arrhythmias, shock

Assessment

History: collateral from family/friends/EMS, onset and progression, preceding events, past medical history, medications, history of trauma, baseline functional status

Physical Exam: ABCs, primary survey, vital signs including temp and glucose, neuro exam (GCS and focal neurological deficits)

Investigations

Labs: CBC, lytes, glucose, BUN, Cr, LFTs, INR/PTT, serum osmolality, VBG, troponin, urinalysis, toxicology panel, septic workup as needed

Tests: ECG, CXR, CT head

Management

General
Monitors, oxygen, vitals, IV access Airway management for declining GCS and inability to protect airway
Treatment
Treat underlying cause, universal antidotes (Dextrose, Oxygen, Naloxone, Thiamine), broad-spectrum antibiotics, warm/cool, BP control
Disposition
Consider admission for working up underlying cause

Headache

Common Types

Migraine: POUND (Pulsatile, Onset 4-72hrs, Unilateral, N/V, Disabling intensity), photophobia/phonophobia, chronic, recurrent, +/- aura

Cluster: unilateral sudden sharp retro-orbital pain, <3 hours usually at night, pseudo-Horner's symptoms, precipitated by alcohol/smoking

Tension: tight band-like pain, tense neck/scalp muscles, precipitated by stress or lack of sleep

Differential Diagnosis

Intra-cranial	Extra-cranial
Bleed: epidural, subdural, subarachnoid, intracerebral hemorrhage Infection: meningitis, encephalitis, brain abscess Increased ICP: mass, cerebral venous sinus thrombosis	Acute angle closure glaucoma Temporal arteritis Carotid artery dissection CO Poisoning Pregnancy-related headaches

Assessment

History: red flags (sudden onset, thunderclap, exertional onset, meningismus, fever, neurological deficit, AMS), symptoms of increased ICP (persistent vomiting, headache worse lying down and in AM)

Physical Exam: vitals, detailed neuro exam (cranial nerves, gait, coordination, motor/sensory, reflexes), neck for meningeal irritation, eye exam (slit lamp, IOP), temporal artery tenderness

Investigations

Neuroimaging to rule out deadly causes. Most benign headaches do NOT need further investigation. Refer to Ottawa SAH Rule.

LP: if CT head negative (>6h from onset) but strong suspicion of SAH

ESR/CRP: if suspect temporal arteritis

Management

Common Benign Headache Regimen
Fluids: No clear evidence but consider in dehydrated patient Antidopaminergic: Metoclopramide 10mg IV Antihistamine: Diphenhydramine 25mg IV Analgesic: Acetaminophen 1g PO NSAIDs: Ketorolac 10-30mg IV or Ibuprofen 600mg PO Steroids: Dexamethasone 10mg PO/IV (rebound migraine prophylaxis)
Non-Traditional Uses
Cluster Headaches: oxygen, Sumatriptan, Verapamil Refractory Headaches: Magnesium, Lidocaine, Propofol, Ketamine, Valproate Nerve Blocks: greater occipital nerve, sphenopalatine block, trigger points

EMOttawa Blog Posts: [Migraine](#), [Subarachnoid Hemorrhage](#), [Podcast](#)

Head Trauma

Common Injuries

Epidural Hematoma (EDH): Often temporal bone fracture resulting in middle meningeal artery injury; primarily a disease of the young; rare in elderly or children <2 due to tight attachment of dura to skull; temporary improvement in condition with subsequent worsening is highly suggestive

Subdural Hematoma (SDH): most common; disruption of bridging veins; most commonly during acceleration-deceleration injuries; common in pts with brain atrophy (elderly, alcohol) as bridging veins transverse greater distances; slow venous bleeding delays symptoms; 20% present with bilateral SDH

Mild Traumatic Brain Injury (aka Concussion): most often due to direct contact, but also acceleration-deceleration injuries; defined as GCS13-15.

Assessment

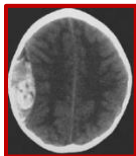
History: LOC, retrograde or anterograde amnesia, alteration in mental state at time of accident (feeling dazed, disoriented or confused), headache, photophobia, dizziness, N/V.

Physical Exam: vitals (assess for Cushing Triad of increased ICP: $\uparrow$ BP, $\downarrow$ HR, irregular breathing), c-spine for midline boney tenderness, neuro exam for focal neurological deficits

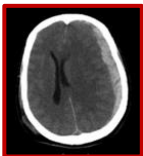
Investigations

Refer to Canadian CT Head Rule in patients with minor head injury to assess appropriate application of the rule.

EDH



SDH



Depending on size, may show midline shift (i.e. uncus herniation, ventricle shift)

EDH
Bi-convex, lentiform Blood pools superior (epi) to dura, unable to cross suture lines
SDH
Crescent shaped Blood pools between dura and arachnoid and is not limited by suture lines, only by dural reflections (i.e. falx cerebri, tentorium, falx cerebelli)

Management

Concussion
Outpatient management for uncomplicated cases: GCS 15, normal exam and CT head 24 hr period of cognitive rest with gradual return to work/play pending resolution of symptoms
Epidural and Subdural Hematomas
Consult neurosurgery for management (i.e. burr hole, craniotomy, decompressive craniectomy) Reduce ICP: raise head of bed to 30° ; IV Mannitol 1g/kg over 20 mins or HTS 1.25-5mL/kg over 5 mins; hyperventilation; diuretics Reverse anticoagulation (i.e. give prothrombin complex concentrate if on Warfarin or Xa inhibitors; Praxbind for Dabigatran)

Seizures

Definitions

Seizure: Excessive abnormal neuronal activity associated with alternations in sensory, motor, autonomic and/or cognitive function

Status Epilepticus: unremitting seizure activity >5 mins in duration or recurrent seizure activity without intervening return to baseline

Refractory Status Epilepticus: no termination after 1st-or 2nd-line agents

Psychogenic Non-Epileptic Seizures: functional convulsions not associated with abnormal neuronal activity (usually variable convulsions, pelvic thrusting, forced eye-closure, responding or maintaining normal LOC, no post-ictal phase)

Common Causes:

Metabolic Disturbances	Infections
Hepatic encephalopathy Hypoglycemia or hyperglycemia Hyponatremia Uremia	CNS abscess Encephalitis Meningitis
CNS Lesions	Intoxication*/Withdrawal
Brain metastases Anoxia/hypoxia Stroke Arteriovenous malformations CVST Epilepsy Bleeds: SAH, SDH, EDH, ICH	Bupropion* TCAs* Lithium* Alcohol/benzos Anti-epileptic drugs

Assessment

History: triggers, aura, memory before and after incident, appearance of convulsions, post-ictal phase, urinary incontinence, tongue biting, infectious symptoms, sensorimotor symptoms, med non-compliance, recent trauma, pregnancy, EtOH, immunocompromised, H/A, other injuries

Physical Exam: vitals (esp pupils), neuro exam (GCS, nystagmus, tone, reflexes)

Investigations

Blood work: CBC, lytes, BUN, Cr, B-HCG; if post-ictal confusion, status or first-time seizure add: LFTs, lactate, VBG, drugs of abuse screen, EtOH level, extended lytes, anti-epileptic drug levels (if applicable)

Tests: ECG, non-contrast CT head if first-time seizure, status, persistent focal deficits, change in seizure pattern, or prolonged post-ictal state

Management of Status

1 st -Line	IV Lorazepam 0.1mg/kg (up to 4mg x2 doses) IM Midazolam 0.2mg/kg (up to 10mg x2 doses)
2 nd -Line	IV Keppra 60mg/kg IV OR IV Phenytoin or Fosphenytoin 20mg/kg OR IV Valproate 40mg/kg (contraindicated in pregnancy)
Refractory Status	RSI intubation, call ICU IV Propofol 2-5mg/kg (infusion 3-5mg/kg/hr) IV Midazolam 0.2mg/kg (infusion 0.05-2mg/kg/hr)

[EMOttawa Blog Post](#)

Shortness of Breath

Definitions

Tachypnea: RR >18 in adults

Hyperpnea: high minute ventilation to meet metabolic demands

Orthopnea: dyspnea lying flat

Paroxysmal Nocturnal Dyspnea: sudden dyspnea at night

Differential Diagnosis

Pulmonary	Cardiac
Airway obstruction Respiratory failure (refer to Type 1 vs Type 2 in "Breathing" section) Anaphylaxis Pulmonary embolism Tension pneumothorax	Pulmonary edema Myocardial infarction Cardiac tamponade Pericardial effusion Arrhythmias
Toxic-metabolic	Neuro-endocrine
Toxin ingestion (ASA, organophosphates, CO poisoning) Sepsis Acidosis (DKA, lactic, etc.)	Thyrotoxicosis Guillain-Barre syndrome Amyotrophic lateral sclerosis Multiple sclerosis

Assessment

History: OPQRST, recent travel, trauma, PE risk factors (Wells Criteria, PERC rule), sick contacts

Physical Exam: appearance, signs of respiratory distress, cardiac/resp exam

Investigations

Blood work: CBC, lytes, BUN/Cr, VBG, cardiac enzymes +/- D-dimer

Tests: ECG, POCUS, CXR (portable if unstable)

Management

General
Monitors, oxygen, vitals, IV access, ABCs
Intubate
If not protecting airway or significant respiratory distress
Empiric Treatment
Trauma: ATLS guidelines, decompress tension pneumo Anaphylaxis: Epinephrine, fluids, +/- antihistamines, +/- steroids Cardiac Causes: see various cardiac sections below Asthma/COPD: oxygen, bronchodilators, corticosteroids +/- antibiotics PE: DOACs as outpatient, LMWH, tPA for massive PE Infection: antibiotics, steroids of obstructive lung disease

Chest Pain

Differential Diagnosis

Deadly Six (PET MAC)	Cardiac
Pulmonary embolism Esophageal rupture/mediastinitis Tension pneumothorax Myocardial infarction Aortic dissection Cardiac tamponade	Pericarditis Myocarditis Endocarditis
Respiratory	Gastrointestinal
Pneumonia Pleural effusion Acute chest syndrome (sickle cell) Lung or mediastinal mass	Esophagus - Mallory-Weiss tear, esophageal spasm Stomach - GERD, dyspepsia/PUD Pancreas - pancreatitis Gallbladder - biliary colic, cholecystitis, cholangitis
Musculoskeletal	Other
Intramuscular pain Rib pathology	Panic attack Herpes Zoster

Assessment

History: character of pain, cardiac risk factors (see HEART score), PE risk factors (see PERC rule), recent trauma, neuro symptoms

Physical Exam: appearance, cardiac exam, resp exam, neuro screen, vitals + pulse deficits

Investigations

Tests: ECG, CXR +/- CTPA

Labs: CBC, lytes, abdo panel, troponin +/- D-dimer

Management

General	ABCs, monitors, oxygen, vitals, IV access, equipment
ACS	ASA, Nitro (avoid in RV infarct), Clopidogrel/Ticagrelor, UFH, STEMI protocol (PCI vs. thrombolytics)
PE	Anticoagulation +/- thrombolysis for massive PE
Esophageal rupture	Urgent thoracics consult, IV antibiotics, NPO, endoscopy and further imaging
Tension pneumothorax	Needle decompression then chest tube (4 th or 5 th ICS anterior axillary line)
Tamponade	Pericardiocentesis
Dissection	Urgent vascular consult, reduce BP and HR with IV labetalol, surgery vs. medical management
Disposition	Diagnosis and risk stratification dependent

Chest Pain Risk Stratification

HEART Score

Inclusion Criteria	Exclusion Criteria
Patients ≥ 21 years old presenting with symptoms suggestive of ACS	New STEMI >1 mm or other new ECG changes, hypotension, life expectancy <1 year, non-cardiac medical/surgical/psychiatric illness
H = History	
0 = slightly suspicious +1 = moderately suspicious +2 = highly suspicious	
E = ECG	
0 = normal +1 = No ST depression but LBBB, LVH, repolarization changes +2 = ST depression/elevation not due to LBBB, LVH, or digoxin	
A = Age	
0 = age <45 +1 = age 45-64 +2 = age ≥ 65	
R = Risk Factors	
Risk factors = HTN, hypercholesterolemia, DM, obesity (BMI >30), smoking (current or smoking cessation ≤ 3 months), positive FHx (parent/sibling with CVD <65 yo), atherosclerotic disease (prior MI, PCI/CABG, CVA/TIA, or PVD) 0 = No known risk factors +1 = 1-2 risk factors +2 = ≥ 3 risk factors or history of atherosclerotic disease	
T = Troponin (initial)	
0 = initial troponin $\leq$ normal limit 1 = initial troponin 1-2X normal limit 2 = initial troponin >2 X normal limit	
Interpretation	
Scores 0-3: 0.9 - 1.7% risk of MACE within 6 wks Score 4-6: 12-16.6% risk of MACE Score ≥ 7 : 50-65% risk of MACE	Use the HEART Pathway (HEART score + delta Tnl) to further lower risk of MACE (not prospectively validated but 1% risk of MACE in retrospective data)

PERC Rule

Inclusion Criteria	Exclusion Criteria
Patients where pre-test probability of PE is considered to be low risk ($<15\%$)	Moderate to high risk for PE
Patients can be safely ruled out and do not require further workup if no criteria are positive:	
Age ≥ 50 , HR ≥ 100 , SpO ₂ $<95\%$, hemoptysis, hormone use (OCs, hormone replacement, estrogen), recent (≤ 4 weeks) surgery/trauma, prior PE/DVT or unilateral leg swelling	

[EMOttawa Blog Post](#)

Abdominal Pain

Differential Diagnosis

RUQ	Epigastrium	LUQ
Hepatitis Biliary colic Cholecystitis/Cholangitis* Pancreatitis* Pneumonia Pleural effusion PE*	Gastritis Dyspepsia/PUD Duodenitis Pancreatitis* Cardiac - ACS*	Pancreatitis* Gastritis Pneumonia Pleural effusion PE*
Right Flank	Umbilicus	Left Flank
Colitis Perforation* Obstruction* Renal colic Pyelonephritis AAA*	Colitis Perforation* Obstruction* Aortic dissection* AAA* Early appendicitis	Colitis Perforation* Obstruction* Renal colic Pyelonephritis AAA*
RLQ	Hypogastric	LLQ
Appendicitis Ectopic pregnancy* PID, TOA Testicular torsion, epididymitis, orchitis Ovarian torsion Renal colic	UTI (Cystitis) Renal colic Obstruction	Diverticulitis* Ectopic pregnancy* PID, TOA Testicular torsion, epididymitis, orchitis Ovarian torsion Renal colic

Can't-Miss Diagnoses*	Risk Factors
Ruptured Ectopic	Hx of STI/PID, recent IUD, previous ectopic, smoking, fallopian tube surgery, tubal ligation
Ruptured AAA	Elderly, hx HTN/DM, smoking, trauma hx
Pancreatitis	Alcohol use, biliary pathology
Cholangitis	Charcot's Triad: fever, RUQ pain, jaundice
Mesenteric Ischemia	Elderly, CAD, CHF, dehydration, infection
Obstruction	Operative or malignant history, elderly
Perforated Viscus	Risk factors for diverticulitis or PUD, malignancy or instrumentation (i.e. colonoscopy)
Comp. Diverticulitis	Elderly, low-fibre diet, Western population

Assessment

History: OPQRST, associated symptoms (N/V, fever, chills, bowel movement, urinary symptoms, pelvic discharge/bleeding)

Physical Exam: abdominal exam +/- pelvic exam, cardiac/resp exam

Investigations

Labs: CBC, lytes, BUN, Cr, LFTs, lipase, lactate, B-hCG +/- CK/TnI

Tests: ECG, CXR, POCUS

Radiology performed U/S (biliary pathology, ectopic, AAA), CT abdo/pelvis

Management

ABCs, NPO, analgesics, antibiotics, anti-emetics, consult surgery as needed

Pelvic Pain

Differential Diagnosis

Gynecological		
Ovaries: Ruptured cyst, abscess, torsion Fallopian tubes: Salpingitis, tubal abscess, hydrosalpinx Uterus: PID, endometriosis, fibroids Pregnancy related (1st trimester): Ectopic pregnancy, threatened abortion, ovarian hyperstimulation Pregnancy related (2nd-3rd trimester): Placental abruption, round ligament pain, Braxton-Hicks contractions Other: Bartholin abscess		
Urinary Tract	Urological	Other
Urolithiasis Pyelonephritis Cystitis	Testicular torsion Prostatitis	Sexual or physical abuse

Assessment

History: OPQRST, associated symptoms (vaginal bleeding, discharge, dyspareunia, bowel or bladder symptoms), pregnancy and sexual history

Physical Exam: vitals, abdominal exam

Pelvic exam (assess cervical motion tenderness, adnexal tenderness)

Speculum exam (look for discharge, blood, take samples as needed)

Investigations

Labs: CBC, lytes, BUN/Cr, B-hCG, +/- vaginal and cervical swabs

Tests: PoCUS - rule out ectopic, free fluid assessment

Formal abdo/pelvic ultrasound

Management

General
ABCs, IV access, analgesia, antiemetics, +/- admit and consult For STIs/PID: safe sex practices, partner testing
Ovarian Cyst
Uncomplicated: analgesia with follow-up Hemoperitoneum or hemodynamically unstable: surgery
Ovarian Torsion/Testicular Torsion
Surgical detorsion or removal
Pelvic Inflammatory Disease
Severe infection: admit with IV antibiotics (Cefoxitin 2g IV q6h IV + Doxycycline 100mg IV q12h x 24 hrs then switch to PO) Mild-moderate infection: Ceftriaxone 500mg IM x 1 + Doxycycline 100 PO BID x 14 days

Back Pain

Deadly Differential Diagnosis

Spinal	Vascular
Cauda equina and spinal cord compression: Spinal metastasis Epidural abscess/hematoma Disc herniation Spinal fracture with subluxation Meningitis Vertebral osteomyelitis Transverse myelitis	Aortic Dissection Ruptured AAA Pulmonary Embolism Myocardial Infarction

Assessment

History: fracture history, cancer risk, infection risk, steroid use, **red flags (BACK PAIN):** Bowel/Bladder dysfunction, Anesthesia (saddle), Constitutional symptoms (night pain, weight loss, fever/chills), Chronic disease, Paresthesias, Age >50, IVDU/infection, Neurological deficits

Physical: vitals + pulse deficits, inspect skin for infection/trauma, abdo exam for AAA, cardiac exam (aortic murmur), MSK lower back exam, neuro exam (lower extremity, reflexes, rectal tone), post void residual

Investigations

Bloodwork: usually not indicated unless suspected infection (CBC, ESR, CRP)

Bedside U/S : rule out AAA, look for bladder distention post-void

PVR: cauda equina syndrome (PVR >200cc has 90% sensitivity for CES)

Management

Cauda Equina Syndrome
Urgent MRI, spine consult, analgesia, IV Dexamethasone
Aortic Dissection
Immediate specialist consultation, IV Labetalol to control HR and BP
Ruptured AAA
Blood resuscitation, immediate OR if unstable
Epidural Abscess or Vertebral Osteomyelitis
MRI to definitively diagnose +/- bone scan (osteomyelitis), broad spectrum antibiotics, orthopedics consult
MSK Back Pain
Analgesia: Acetaminophen, NSAIDs Multidisciplinary approach with GP follow-up

Vaginal Bleeding

Differential Diagnosis

Non-Pregnant (PALM COEIN)	Pregnant <20w
Polyps	Cannot Miss: ectopic pregnancy
Adenomyosis	Other: spontaneous abortion, molar pregnancy, subchorionic hematoma, implantation bleeding
Leiomyoma (fibroids)	
Malignancy	Pregnant >20w
Coagulopathy	Cannot miss: uterine rupture, placenta previa, placental abruption, vasa previa,
Ovulatory dysfunction	Other: 'bloody show', cervical lesion, post-partum hemorrhage
Endometrial	
Iatrogenic (i.e. drugs, surgery)	
Not classified	

Assessment

History: LMP, pregnancy complications, GTPAL, time and duration of bleeding, clots, signs/symptoms of blood loss (ie. anemia sx: fatigue, dyspnea, syncope, soaking; #pads/hr), trauma, past menstrual, reproductive & sexual hx

Physical Exam: Vitals (BP, HR), abdominal exam (localize pain, peritoneal S&S, size of uterus), speculum & bimanual exam (must r/o placenta previa first in 2nd/3rd trimester; cervical os, products of conception, lacerations/trauma, cervical motion and adnexal tenderness), fetal HR monitoring (if >20 wks)

Diagnosis

Labs: Most important: determine if pregnant (qualitative as screening, if positive pursue quantitative b-hCG). CBC, type & screen, INR, PTT, Rh status, U/A

Transabdominal Ultrasound (TAUS): first step to attempt to confirm intrauterine pregnancy (IUP), if unable, then considered no definitive IUP (NDIUP)

Transvaginal Ultrasound (TVUS): if NDIUP on TAUS, used to confirm IUP

Discriminatory Zone: if b-hCG > 1,500 or b-hCG > 6,000, IUP should be visible on TVUS & TAUS, respectively. If not, suspect ectopic pregnancy.

Management

General
If unstable: resuscitate with blood (O- until crossmatched) and crystalloid
Non-Pregnant
Most can be discharged home with close Gyne follow-up
If unstable: high dose IV conjugated estrogen q4-6h for 24h + Gyne consult + consider TXA
If stable: NSAIDs, short course of TXA during bleeding episodes, OCPs
Pregnant
Rho-GAM in all Rh (-) pts
Spontaneous Abortion:
- If os is open: attempt to remove products of conception to relieve pain, if unsuccessful consult OB - If os is closed, patient febrile, uterus is tender: suspect Septic Abortion - empiric IV Gentamycin & Clindamycin and consult OB - If os is closed and uterus is non-tender: Threatened Abortion - expectant management, d/c home with close OB/GP F/U vs. Missed Abortion - Misoprostol 800 mcg vaginally or PO or surgical (D+C) - Emotional Support: recognize trauma/grief accompanying miscarriage
Ectopic Pregnancy (consult OB):
- Expectant if stable and reliable - F/U in 2 days with repeat b-hCG - Medical: Methotrexate 50 mg/m² IM if meets indications - F/U with OB - Surgical (Dilation and curettage, evacuation, oophorectomy, etc.)

[EMOttawa Blog Post](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 9th ed, 2018; Chapter 31. EM:RAP C3, 2016; Non-pregnant Vaginal Bleed & Pregnant First Trimester Vaginal Bleeding

Red Eye

Causes of Red Eye

Traumatic	Foreign body Corneal abrasion/ulcer Subconjunctival hemorrhage Caustic contamination Blunt or penetrating trauma	Retrobulbar hematoma Hyphema Orbital compartment syndrome Globe rupture
Non-Traumatic	Acute angle closure glaucoma Retinal ischemia Temporal arteritis Conjunctivitis Keratitis	Episcleritis Scleritis Uveitis Endophthalmitis Cellulitis (orbital or periorbital)

Assessment

History: Check for red flags such as severe pain, persistently blurred vision, proptosis, reduced pupillary light reflex, corneal epithelial defect, ciliary flush, pupils unreactive to light, soft contacts, neonate, immunocompromise

VVEPPS plus Fundoscopic Exam	
Visual acuity (with correction)	Pupillary evaluation
Visual fields	Pressure determination (IOP)
External exam	Slit lamp exam (consider fluorescein)
Extraocular muscle movement	Fundoscopic exam

Investigations

Labs: CBC, ESR/CRP in monocular vision loss (r/o temporal arteritis)

Imaging: CT and US as needed to rule out foreign bodies, orbital cellulitis, fracture, globe pathology. Most red eyes do NOT need further investigation.

Management

Critical Diagnosis - Immediate Intervention
Caustic Contamination: Irrigation with Morgan Lens until tear-film pH = 7.4, outpatient Ophtho Orbital Compartment Syndrome: Lateral canthotomy and cantholysis, Ophtho Acute Angle Closure Glaucoma (Goal: IOP <35 or 25% reduction): Timolol 0.5% 1gtt, Apraclonidine 1% 1gtt, Pilocarpine 1% 1gtt q15 mins, Prednisolone 1% 1gtt, Acetazolamide 500mg IV, Mannitol 1g/kg IV, Ophtho
Emergent Diagnosis - Ophthalmology Consult
Penetrating Trauma or Corneal Ulcer or Open Globe: Protect eye, analgesia, IV abx, tetanus prophylaxis. Avoid contact lenses Keratitis: Topical anesthetic, remove foreign material, discuss abx/antivirals Episcleritis: Artificial tears and ketorolac drops Scleritis: NSAIDs, discuss steroids Anterior Uveitis: 2 drops cyclopentolate 1%, discuss steroids Endophthalmitis: Admit for IV vancomycin and ceftazidime Orbital Cellulitis: IV abx with 2 nd generation cephalosporin
Urgent Diagnosis - Manage in ED Before Discharge
Foreign Body: Topical anesthetic, removal under slit lamp with swab or 27-gauge needle. Outpatient Ophtho for rust ring removal for metallic objects Corneal Abrasion: topical anesthetic Periorbital Cellulitis: Amox/Clav 875 mg PO BID x 10 days

Monoarthritis & Polyarthritis

Differential Diagnosis:

Infectious	
Septic Arthritis RFs: young or elderly, low SES, IVDU, alcohol use disorder, DM, skin infections, chronic arthritis, recent intraarticular injections, prosthetic joint, immunocompromised Gonococcal Septic Arthritis RFs: young, sexually active, STI hx (poly, asymm) Lyme Arthritis (poly, asymm)	
Peri-Articular	Crystal
Bursitis, tendinitis, muscle strain	Gout, pseudogout (CPPD)
Musculoskeletal	Other
Fractures, injury, osteoarthritis	Hemarthrosis: spontaneous or traumatic, malignancy

Involves multiple joints: RA, OA (knees, hips, spine), SLE, scleroderma, seronegative arthritis, polymyalgia rheumatica, viral arthritides (Gonococcal and Lyme arthritis)

Assessment

History: PMHx, characterize pain, duration of symptoms, associated sx, hx of trauma, septic arthritis RFs

Physical Exam: Systemic: S&S of rheumatic disease; joints: warmth, redness, pain, effusion, deformity, ROM, pain with ROM

Gout	1 st MTP, hx of kidney stones
OA	DIPs > PIPs, MCPs, worse with activity and better with rest
Inflammatory (i.e. RA)	MCPs and PIPs > DIPs, pain/stiffness that improves with activity, low grade fever, PMHx or FHx
Septic	Classic triad: fever (only 50% cases), joint pain, effusion

Investigations

Labs: Unable to rule out or diagnose specific etiologies, ↑CRP + ESR or ↑WBC may suggest inflammatory or septic arthritis, ↑serum urate suggests gout

XR: 4 signs of OA: joint space narrowing, subchondral sclerosis, subchondral cysts, osteophytes

Arthrocentesis: Can be diagnostic or therapeutic. Send for cell count & diff, culture, crystal analysis. Avoid if # suspected. Consult ortho if prosthetic joint.

Management

Gout	Do NOT start Allopurinol in acute flares NSAIDs: Naproxen 500 mg BID, Ibuprofen 800 mg TID, Indomethacin 50mg TID Colchicine: 1.2 mg PO loading dose, then 0.6 mg PO one hour later, then 0.6 mg PO daily until two days after resolution of gout flare. Can use in combo with NSAIDs or standalone Steroids (2nd Line): Prednisone 20 mg BID with a tapering dose x 7-10d (rebound gout flare is common with steroid treatment)
OA	Non-Pharmacological: weight loss, exercise Pharmacological: Tylenol 1 g PO TID, Celecoxib 100mg PO BID Topical: NSAIDs, Diclofenac, Capsaicin Intra-Articular: steroid or hyaluronic acid (equivocal evidence) Ortho Referral: for consideration of joint replacement if failing conservative management
Septic	Requires admission. Empiric IV abx (Ceftriaxone & Vanco) later narrowed based on gram stain and cultures. Often requires arthroscopic joint irrigation or often I&D required for shoulders/hips

Selected Emergencies

Anaphylaxis

Definition: life-threatening immune hypersensitivity systemic reaction leading to histamine release, vascular permeability, and vasodilation

Common Triggers: foods (egg, nuts, milk, fruits), meds (antibiotics, NSAIDs), insect bites, local anesthetics, occupational allergens, aeroallergens

Differential Diagnosis: shock (of any etiology), angioedema, flush syndrome, asthma exacerbation, vancomycin infusion reaction

Diagnostic Criteria:

Acute onset (minutes to hours) + ANY of the following three:
Involvement of skin +/- mucosa WITH EITHER respiratory difficulty or low blood pressure
Exposure to likely allergen with 2 of 4 signs: Skin-mucosal involvement (urticarial, angioedema, flushing, pruritis) Respiratory difficulties (dyspnea, wheezing, stridor, hypoxemia, rhinitis) Low BP (hypotonia, syncope, pre-syncope, headache, collapse) GI symptoms (abdo pain, cramps, N/V)
Low blood pressure after exposure to known allergen

Assessment

General: TREAT FIRST, ABCs, monitors, oxygen, vitals, IV access appearance, respiratory distress, visualize swelling (lips, tongue, mucous membrane)

History: exposure to any known or likely allergen, co-morbidities, recent medication use, family history, atopy

Management

General Management
If need to protect airway: Ketamine as induction agent Epinephrine: 0.3-0.5mg IM (1:1000 conc.) to anterolateral thigh q5-10 mins Antihistamines: Benadryl 50mg IV/PO (controversial) Steroids: Methylprednisolone 125mg IV/Prednisone 50mg PO (controversial)
Refractory Hypotension
Epinephrine drip 1-10µg/min IV (titrate to desired effect) Consider Norepinephrine 0.05-0.5µg/kg/min
Patients with β -Blockers
If Epinephrine unsuccessful, Glucagon 1-5mg IV over 5-10 mins followed by 5-15µg/min infusion
Disposition
May discharge as early as 2 hours if stable. Education to avoid allergen, consider allergy testing, Epi-pen prescription Meds at discharge: may consider 2 nd generation antihistamines, steroids not necessary

EMOttawa Blog Posts: [Anaphylaxis](#), [Podcast Part 1](#), [Podcast Part 2](#)

Asthma

Definition: chronic inflammatory airway disease with recurrent reversible episodes of bronchospasm and variable airflow obstruction

Exacerbation Triggers: URTIs, environmental allergens, smoking, exercise

Classification (CAEP/CTS Asthma Severity):

Respiratory Arrest/Fatal
Appearance: altered mental status, cyanotic, decreased resp. effort Vitals: low HR, high RR, low O ₂ sat <90% despite oxygen Exam: Silent chest - consider preparing for advanced airway intervention
Severe
Appearance: agitated, diaphoretic, labored respirations, difficulty speaking Vitals: high HR, high BP, O ₂ sat 90-95% Exam: worsening resp. distress, exp/insp. wheezing, FEV1 <40% predicted
Moderate
Appearance: SOB at rest, cough, congestion, nocturnal symptoms Vitals: O ₂ sat >95% Exam: exp. wheezing, FEV1 40-60% predicted
Mild
Appearance: SOBOE, chest tightness Vitals: O ₂ sat >95% Exam: exp. wheezing, FEV1 >60% predicted

Assessment

History: triggers, recent infection, thorough asthma hx including prior exacerbations, hospitalizations + interventions/ICU stays, family history

Asthma Control Criteria: daytime symptoms <4/week, no exercise limitation, no absenteeism, no nocturnal symptoms, rescue puffer <4/week, normal PFT, exacerbations mild/infrequent

Physical Exam: vitals, sign of distress, accessory muscle use, respiratory exam

Investigations: CXR, ECG +/- VBG, +/- PEFR (to estimate FEV1), bloodwork (CBC - infection, lytes - potassium)

Management

Treat Exacerbation ("0.5 - 5 - 50")
Ipratropium bromide 0.5mg neb OR 4-8 puffs via MDI + spacer q20mins x 3 Salbutamol 5mg neb OR 4-8 puffs via MDI + spacer q20mins x 3 Prednisone 50mg PO NOTE: MDIs are superior to nebs unless patient too tachypneic to use MDI
Severe Asthma
MgSO ₄ 2g IV over 30 mins Epinephrine 0.3mg IM then 5µg/min IV infusion Ketamine 1mg/kg (in conjunction with BiPAP)
Respiratory Failure
Consider NIPPV first (BiPAP) Intubate (LAST RESORT): Ketamine 1mg/kg IV + Succinylcholine 1.5mg/kg IV Involve ICU early

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 73. CMAJ 1996; 155(1): 25-37.

Chronic Obstructive Pulmonary Disease

Classifying Severity: based on GOLD Classification

GOLD 1	Mild	$FEV_1 \geq 80\%$ predicted
GOLD 2	Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3	Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4	Very Severe	$FEV_1 < 30\%$ predicted

Risk Factors: smoking (#1), occupational dust, chemical exposure

AECOPD Triggers: viral URTI, pneumonia, environmental allergens or pollutants, smoking, CHF, PE, MI $\geq 80\%$ predicted

Assessment

Cardinal Symptoms: $\uparrow$ SOB, $\uparrow$ sputum production, $\uparrow$ sputum purulence.

Key Elements on History: duration of symptoms, severity of airflow limitation, number of previous episodes (total/hospitalizations), co-morbidities, premorbid functional status, present treatment regimen, previous use of mechanical ventilation, use of home oxygen.

Clinical Signs of Severity: rapid shallow pursed-lip breathing, use of accessory muscles, paradoxical chest wall movements, worsening or new onset central cyanosis, peripheral edema, hemodynamic instability, decreased LOC or confusion, decreased O_2 sat.

Consider PE if deteriorating & not improving with standard COPD therapy.

Investigations

Labs: CBC, electrolytes, VBG, lactate, serologies (triplex, COVID, etc)

Tests: CXR, ECG, pulse oximetry

Management

Follow **GOLD Guidelines** for best practices

Oxygen
Venturi masks (high-flow devices) preferred over nasal prongs Target SaO_2: $>88\%$ Goal $PaO_2 = 60-65\text{mmHg}$
Bronchodilators
SABA: Salbutamol 2.5-5mg via nebulizer or 4-8 puffs via MDI with spacer q15mins x3 PRN Anticholinergic: Ipratropium bromide 500 μg via nebulizer or 4-8 puffs q15mins x3 PRN
Systemic Corticosteroids
Oral is equivalent to IV in most exacerbations Oral Prednisone 40-60mg for 5-10 days IV Methylprednisolone 125 mg BID-QID (for severe exacerbations or not responding to oral steroids)
Antibiotics
Indication: ≥ 2 of: 1) $\uparrow$ sputum production 2) $\uparrow$ sputum purulence 3) $\uparrow$ SOB Simple exacerbation: Amoxicillin, 2 nd /3 rd gen Cephalosporin, Macrolide, Doxycycline or TMP/SMX Complicated exacerbation: Fluoroquinolone or Amoxicillin/Clavulanate
Ventilation
NIPPV such as CPAP or BiPAP (in respiratory acidosis, severe dyspnea/distress)
Intubation
For life-threatening exacerbations, failed NIPPV, altered LOC, severe hypoxemia, cardiovascular instability, respiratory or cardiac arrest

[EMOttawa Blog Post](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 74. Am J Respir Crit Care Med 2013; 187(4):347-365.

Myocardial Infarction

Definition: evidence of myocardial ischemia on the spectrum of ACS (unstable angina, NSTEMI and STEMI). Diagnosed by cardiac marker abnormalities and one of: ECG changes or HPI consistent with ACS.

Differential Diagnosis

Stable Angina
Transient episodic chest discomfort secondary to myocardial ischemia Precipitated by exertion or emotion, lasts <15 mins, relieved by rest or nitro
Unstable Angina
Angina with minimal exertion or at rest, new-onset angina, angina post MI/PCI/CABG, worsening change from baseline angina, increased duration of pain or threshold, or decreased response of previously effective angina meds
NSTEMI (non-occlusive)
Infarction without ST elevation (note: 25% NSTEMI have totally occluded artery)
STEMI (acute coronary occlusion)
Infarction with primary ST elevation: $\geq 1\text{mm}$ in 2 contiguous leads, except for V2-3 $\geq 1.5\text{mm}$ in females, $\geq 2\text{mm}$ in males over 40, and ≥ 2.5 in males under 40 - Other signs of occlusion: hyperacute T waves, minor ST elevation with reciprocal ST depression, posterior MI (primary ST depression V1-4, often with 0.5mm STE on posterior leads), LBBB + Modified Sgarbossa, DeWinter sign

Assessment

History: character of pain, associated symptoms (diaphoresis, radiating pain, vomiting, and exertional pain have highest likelihood ratios for acute MI)

Classic Risk Factors: male, smoking, diabetes, HTN, FHx, dyslipidemia

Varied Presentations in: women, elderly, diabetics, dementia

Complications of AMI: arrhythmias, cardiogenic shock, papillary muscle rupture, pericarditis, stroke

Physical Exam: vitals, cardiac exam, resp exam, pulses, signs of complications

Investigations: ECG (ST-T changes, new BBB, pathological Q waves), CXR

Labs: CBC, lytes, cardiac enzymes

Management

General
ABCs, monitors, oxygen, vitals, IV access Pain control: NTG (avoid for RV infarcts) or Morphine if resistant to NTG
ACEi, B-blockers, Statins
Atorvastatin 80mg PO in STEMI. Do NOT delay transfer to cath lab for statin No role for initiating ACEi or B-blocker in the ED ACEi, B-blocker + statins likely to be initiated during hospital admission (<24-48 hrs from time of presentation)
Antiplatelet Therapy
ASA 325mg chewed Clopidogrel 300mg PO OR Ticagrelor 180mg PO (if going for primary PCI)
Antithrombotic Therapy
Primary PCI: UFH 4000 Units (max) then 12 U/kg/hr Fibrinolytics: Enoxaparin or Fondaparinux IV bolus then SC dose daily
Goals
Primary PCI: within 90 mins of hospital arrival Lytics: <12 hours of symptoms or cannot get to PCI centre within 120 mins, given within 30 mins of hospital arrival

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 78. Circulation 2013; 127:00-00. JACC 2022 Nov,80(20):1925-1960 (ACC Expert Consensus on Chest Pain in the ED).

Congestive Heart Failure

Etiology: CAD, HTN, valve abnormalities, cardiomyopathy, infarction, pericardial disease, myocarditis, cardiac tamponade, metabolic disorders (i.e. hypothyroidism), toxins, congenital

Precipitants of CHF Exacerbation

Cardiac	Medications
Ischemia, dysrhythmias, mechanical complications (i.e. papillary muscle rupture)	Forgot meds, negative inotropes (CCB, β -blocker), NSAIDs, steroids
High Cardiac Output	Other
Anemia, infection, pregnancy, hyperthyroidism	Lifestyle (high salt intake), renal failure, PE, HTN

Assessment

Symptoms	Signs
Left-sided: SOB, PND, fatigue, orthopnea, angina, syncope, altered mental status, cough + wheeze (pulmonary congestion) Right-sided: fatigue, abdominal distension, leg swelling, weight gain, nocturia	General: tachypnea, tachycardia, hypertension, hypotension, weak pulses Left-sided: hypoxia, crackles, wheezes, S3 or S4 Right-sided: pitting edema, JVP elevation, hepatomegaly, ascites

Investigations

Labs: CBC, electrolytes, AST, ALT, BUN, Cr, Troponin, BNP (or NT-proBNP)

Tests: CXR, ECG, PoCUS (systolic function, pulmonary edema)

Management

General
ABCs, monitors, 100% O ₂ non-rebreather facemask, vitals, IV access, position upright, +/- Foley catheter, treat precipitating factor Morphine 1-2mg IV prn
First-line
Nitroglycerin 0.4mg sl q5min (if sBP>100) +/- topical nitroglycerin patch (0.2-0.8mg/h) Furosemide: double home dose, 20mg IV if furosemide naive
Second-line
Double furosemide dose Think Sympathetic Crashing Acute Pulmonary Edema (SCAPE): Nitroglycerin infusion if hypertensive (start 100 μ g/min & titrate) Think Cardiogenic Shock if hypotensive (sBP<90): Norepinephrine 2-12 μ g/min or Dobutamine 2.5 μ g/kg/min

Cardiac Dysrhythmias

Causes:

Enhanced Automaticity: MI, drugs, toxins, lyte imbalances

Triggered Activity: Torsades de Pointes, post-MI reperfusion

Re-entry: VT and SVT

Main Classifications

Bradydysrhythmias and AV Conduction Clocks
1° = prolonged PR interval 2° (Mobitz I) = gradual PR interval prolongation then QRS drop 2° (Mobitz II) = PR interval constant with QRS drop 3° = P wave and QRS complex unrelated, PP and RR intervals constant
Supraventricular Tachydysrhythmias (Narrow QRS)
Regular rhythm: Atrial: sinus tachycardia, atrial tachycardia, atrial flutter AV: SVT (AVNRT > AVRT), junctional tachycardia Irregular rhythm: Atrial: a fib, multifocal atrial tachycardia, SVT w/ aberrancy
Ventricular Tachydysrhythmias (Wide QRS)
Regular rhythm: Ventricular tachycardia, SVT w/ aberrancy Irregular rhythm: Ventricular fibrillation, polymorphic VT, Afib w WPW

Assessment

Unstable Patient: altered mental status, respiratory distress, hypotension, syncope, chest pain with acute MI, signs of CHF, shock

Stable Patient: light-headedness, SOB, palpitations, mild anxiety

Management

General: monitors, oxygen, continuous monitoring, IV access

Initial Approach: ABCs, treat symptomatic & unstable patients immediately

*See detailed **ACLS Algorithms**

Bradycardia Algorithm
1. Treat Reversible Causes: O ₂ if hypoxia, Ca if hyperK, STEMI cath lab 2. Treat Unstable Primary Bradyarrhythmia: Atropine 0.5mg IV q3-5 mins +/- Dopamine 2-10µg/kg/min OR Epi 2-10µg/min If ineffective: transcutaneous pacing, prepare for IV pacing Type II 2oAV block OR 3oAV block: transcutaneous pacing
Tachycardia Algorithm
Synchronized cardioversion (with premedication) if unstable
Atrial Fibrillation/Atrial Flutter
1. Treat secondary causes: sepsis, GI bleed, CHF, PE, EtOH withdrawal 2. Rate vs. Rhythm control
VF/pVT
Shock-CPR-pulse rhythm check cycles, Epinephrine 1mg IV q3-5mins, consider Amiodarone 300mg IV bolus with 2 nd dose 150mg IV
PEA/Asystole
CPR, airway support, IV access, Epinephrine 1mg IV q3-5mins

[EMOttawa Blog Post](#)

Pericarditis & Myocarditis

Definitions

Pericarditis: Inflammation of the pericardial sac surrounding the heart

Myocarditis: Inflammation of the cardiac muscle

Causes

Idiopathic	Most common, often presumed viral
Infectious	Viral, bacterial including TB, fungal
Post-injury	Trauma, surgery, MI, radiation, aortic dissection
Iatrogenic	Post-vaccine, drugs
Systemic disease	Uremia, RA, SLE, scleroderma, malignancy

Assessment

History: **Pericarditis:** non-exertional, pleuritic pain, worse when supine and improves leaning forwards/sitting up, fevers, myalgias, dyspnea, diaphoresis, syncope or presyncope, recent viral illness. **Myocarditis:** presents mainly with CHF symptoms including dyspnea on exertion, bilateral leg swelling

Physical Exam: vitals (fever, BP, HR, RR), pericardial friction rub in pericarditis

Diagnosis of Pericarditis: ≥ 2 of: characteristic chest pain, friction rub, ECG changes, pericardial effusion (PCE) on ultrasound

Investigations

Pericarditis	Myocarditis
Labs	
$\uparrow$ CRP, ESR, WBC though not needed for diagnosis	Highly suggestive with elevated troponin, though negative troponin does not rule it out.
ECG	
Diffuse concave ST elevation II>III PR depression, ST depression aVR only Note: any Q wave, convex ST elevation, ST depression other than aVR or hyperacute T wave = acute coronary occlusion until proven otherwise	May have tachycardia/arrhythmia, conduction abnormality (PR, QRS, QT), Q wave, or ST/T changes Note: can mimic STEMI, and myocarditis is diagnosis of exclusion
Ultrasound	
Used to identify the presence of a pericardial effusion	Reduced LVEF, global hypokinesis, regional wall motion abnormalities, may see PCE
CXR	
May see cardiomegaly if a pericardial effusion is present	

Management

General
Treat underlying cause
Pericarditis
Most can be managed as outpatient NSAID: Ibuprofen 600mg PO TID (+ PPI) until symptom free, taper over 2-4 wks Colchicine 0.6mg BID x 3 months ($\downarrow$ risk of recurrence = NNT 5) Steroids: autoimmune, uremic diseases (consult cardiology)
Myocarditis
Admission under cardiology Supportive: treat arrhythmias and CHF symptoms Complications: ventricular dysrhythmias, LV aneurism, CHF, dilated CM

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 9th ed, 2018; Chapter 72.
Tintinalli's Emergency Medicine - 9th ed, 2020. Chapter 55.

Deep Vein Thrombosis

Definition: formation of a blood clot in a deep vein, most commonly in the legs or pelvis

Massive Iliofemoral DVT: Phlegmasia Alba Dolens (PAD) or “painful white/milky leg”: venous thrombosis progresses to massive DVT, but without ischemia as collateral veins are spared. **Phlegmasia Cerulea Dolens (PCD)** or “painful blue leg”: occurs following PAD when ischemia ensues, worsening congestion and edema, eventually progressing to gangrene

Risk Factors: venous stasis (surgery or trauma), vessel injury (surgery or trauma), hypercoagulability (inherited thrombophilia, active malignancy, pregnancy, OCPs, prior PE/DVT)

Differential Diagnosis: chronic venous insufficiency, cellulitis, muscle strain/tear, Baker's cyst, hematoma, claudication/ischemia, intra-abdominal compression, unrecognized trauma

Assessment

Hallmarks of DVT include unilateral erythema, swelling, warmth and limb tenderness (especially with palpation of posterior calf and popliteal fossa)

Modified Wells Criteria for DVT	
+1 Active malignancy (treatment within 6 months or palliative) +1 Paralysis, paresis, or recent immobilization of lower limb +1 Bedridden >3 days or major surgery in last 12 weeks +1 Tenderness along deep venous system +1 Entire leg swollen +1 Calf swelling >3cm compared to asymptomatic side +1 Pitting edema in symptomatic leg +1 Superficial non-varicose veins +1 Previous DVT -2 Alternative diagnosis as or more likely than DVT	
Interpretation and Further Workup	
DVT unlikely (low risk) Score <2	Order D-Dimer: If negative (<500) = no DVT If positive (≥500) = obtain leg Doppler US
DVT likely (high risk) Score ≥2	Obtain leg Doppler US ± D-dimer* *D-dimer still useful in case of negative US, as negative US with positive dimer in this subgroup warrants repeat US to ensure below knee clot has not progressed to above knee

Investigations: CBC, BUN, Cr, electrolytes, D-dimer, INR/PTT
Leg doppler - standard 3-point compression tests

Management

DVT
Preferred Outpatient Therapy: DOACs (Apixaban 10mg BID x7 days then 5mg BID or Rivaroxaban 15mg BID x3 weeks then 20mg daily) DVT + Cancer or Pregnancy: LMWH (Enoxaparin 1mg/kg SC) Renal Impairment: Unfractionated Heparin 70-80 U/kg Consult Thrombosis for outpatient management

Pulmonary Embolism

Definition: results most commonly after a clot formed hours-weeks earlier in deep veins of leg dislodges and travels through venous system into right ventricle and then into pulmonary vasculature. Occasionally, a clot originates spontaneously in pulmonary vasculature

Risk Factors: see DVT risk factors above

Differential Diagnosis: ACS, costochondritis, rib fracture, spontaneous pneumothorax, pneumonia, pericarditis, COPD/asthma

Assessment

Hallmark symptoms include sudden onset of dyspnea and pleuritic chest pain

Massive PE: hypotension, cardiac arrest, bradycardia (HR <40) with shock

Submassive PE: no hypotension, evidence of RV strain (on POCUS, CT or elevated BNP, ECG changes), or myocardial necrosis (elevated troponin)

Wells Criteria for PE	Interpreting Wells Criteria	
+3 Signs + symptoms of DVT +3 PE = #1 diagnosis +1.5 HR >100 +1.5 Immobilization ≥3 days OR surgery in past 4 weeks +1.5 Hx DVT/PE +1 Hemoptysis +1 Active cancer (treatment within 6 months or palliative)	Two-Tier Low: ≤4 High: >4	PE unlikely Order D-Dimer: If negative (<500) = no PE If positive (≥500) = CTPA PE likely Obtain CTPA
PERC (PE Rule-out Criteria)	Age-Adjusted	
Essentially rules out PE (i.e. <2% chance of PE) if PERC Negative (NO high-risk features below) and low pre-test probability. High-Risk Features: Age ≥50, HR ≥100, SpO ₂ <95%, hemoptysis, hormone use, recent (≤4 weeks) surgery/trauma, prior PE/DVT or unilateral leg swelling If PERC positive, apply Wells	D-dimer levels rise naturally with age, even in otherwise healthy patients. For patients >50, Age x 10 validated as safe D-dimer cut-off. For example, for a 62-year-old patient, 620 (62 x 10) is validated as the D-dimer threshold. Therefore D-dimer <620 rules out PE.	
ECG Signs of Acute RV Strain	YEARS Algorithm	
Heart rate/rhythm: sinus tach, new A fib Electrical conduction: new RBBB Axis: new right axis, or S1 ST/T changes: primary T wave inversion anterior and inferior, S1Q3T3	Simplifies Wells score into 3 features most predictive of PE: 1. Clinical signs of DVT 2. Hemoptysis 3. PE as most likely diagnosis 0 YEARS items: D-dimer threshold <1,000 excludes PE ≥1 YEARS item: D-dimer <500 required to exclude PE	

Management

PE
See DVT management above; tPA reserved for massive PE (50mg IV over 20 minutes, followed by another 50mg IV if no improvement)

EMOttawa Blog Posts: [PE in Pregnancy](#), [D-Dimer in PE](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 9th ed, 2017; Chapter 78. JAMA 2014;311(11):1117-1124. Lancet 2017; 390(10091):289-297.

Gastrointestinal Bleeding

Risk Factors: medications (NSAIDs, anticoagulants), excessive vomiting, bleeding disorders, malignancy, alcohol use, ulcer history, H. pylori

Differential Diagnosis

Upper GI bleed (proximal to Ligament of Treitz)

Peptic ulcer disease (gastric > duodenal)
Gastritis/esophagitis
Esophageal varices
Mallory-Weiss tears
Gastric cancer

Lower GI bleed (distal to Ligament of Treitz)

Colitis (inflammatory, infectious, ischemic)
Anorectal pathology (hemorrhoids, fissures, proctitis)
Angiodysplasia
Diverticulosis
Malignancy

Assessment

History: blood quantity/quality, symptoms of anemia (fatigue, SOB, chest pain), Hx liver disease, medication review, smoking/EtOH, bleeding disorders, constitutional symptoms

Beware mimics: Pepto-Bismol, iron ingestion can cause dark stools

UGIB: hematemesis, coffee ground emesis, melena, BRBPR if brisk UGIB

LGIB: hematochezia, BRBPR

Physical Exam: ABCs, vitals, inspect nasal-oral cavity, abdominal exam, DRE

Investigations

Labs: CBC, lytes, INR/PTT, BUN, Cr, lactate, VBG, T+S/T+C

Tests: ECG, CXR +/- CT if indicated for LGIB

Management

General

ABCs, monitors, oxygen, vitals, 2 large bore IVs, reverse any anticoagulation, GI consult
Intubate early if suspect unprotected airway or risk of aspiration
Transfusion threshold: Hb <70, Plt <50, or hemodynamically unstable or with active bleeding

UGI Bleed

Pantoloc 80mg IV bolus then 8mg/h infusion (or intermittent BID dosing)
Octreotide 50µg IV bolus then 50µg/h infusion - for suspected variceal bleeding
Ceftriaxone 2g IV: for suspected variceal bleeds, prevention of SBP
Tranexamic acid: hemodynamically unstable patients (no clear evidence)
Balloon tamponade: crashing GI bleeding patient

LGI Bleed

NPO, IV fluids, manage underlying etiology (i.e. Abx, steroids)
Colonoscopy to evaluate cause of bleeding

EMOttawa Blog Posts: [Upper GI Bleeds](#), [Podcast](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 30.

Stroke

Common Syndromes

ACA Stroke
Contralateral motor + sensory deficits (Leg > face/arm) Bowel and bladder incontinence Impaired judgement/insight
MCA Stroke
Contralateral motor + sensory deficits (Face/arm > leg) Contralateral hemianopsia; gaze preference towards lesion Aphasia (dominant) or neglect (non-dominant)
PICA Stroke (Wallenberg Syndrome)
Pain/temperature loss on contralateral side + ipsilateral face Ipsilateral Horner's-like syndrome "Deadly Ds": dysphagia, diplopia, dysarthria, dysphonia

Assessment

History: time of onset (usually abrupt), LOC (usually normal or non-significant decrease), focal symptoms, headache (pain more suggestive of hemorrhagic stroke or dissection), functional baseline (dictates treatment)

Stroke Mimics: seizure, migraine, syncope, metabolic derangements, sepsis, tumor, functional neurological disorder (conversion disorder), seizure (i.e. Todd's paralysis)

Physical Exam: vitals (close attention to BP), neuro (NIHSS scale), cardiovascular exam (dissection, arrhythmias, valvular pathology), look for comorbidities

Investigations

Labs: CBC, lytes, extended lytes, glucose, BUN, Cr, INR, PTT

ECG: rule out Afib

Neuroimaging: acute stroke protocol (CT/CTA immediately)

Management

General
ABCs, monitors, oxygen, vitals, IV access +/- intubation (declining GCS, evolving symptoms, or presumed hemorrhagic transformation) BP control: lower if HTN severe (>220/120), BP <185/110 if giving tPA Consult neurology, admission to stroke unit
Antiplatelet Therapy
Don't give acutely, start ASA +/- Clopidogrel daily once discharged
Stroke Time Windows (consult FAST-ED app)
<4.5 hrs: tPA 0.9mg/kg (max 90mg, 10% bolus, 90% over 1 hr) <6-24 hrs: Endovascular Therapy (EVT) for large vessel occlusion (i.e. carotid, MCA, basilar) Discuss with stroke team
Stroke Prevention
Primary: stratify based on CHADS ₂ (stroke risk with Afib), Ottawa TIA Risk Score, prescribe ASA vs. DOACs Secondary: 28 days of ASA + Clopidogrel in minor stroke (NIHSS 0-3), oral anticoagulation started 1-2 weeks post-stroke if Afib, ASA otherwise

[EMOttawa Blog Post](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 101. NEJM 1995; 333:1581-1588. AMJ Neuroradiol 2001; 22:1534-1542. Int J Stroke 2020; 15(6): 689-698. NEJM 2018; 378(1):11-21. NEJM 2018; 378: 708-718.

Transient Ischemic Attack

Definitions

TIA Definition (Canadian Stroke Best Practices)
A brief episode of neurological dysfunction caused by focal brain, spinal cord or retinal ischemia without imaging evidence of acute infarction, typically resolving within one hour.
Very High-Risk TIA
All TIAs with symptom onset within 48 hours
High-Risk TIA
Symptom onset between 48 hours and 2 weeks with the following symptoms: Motor or speech disturbance
Moderate-Risk TIAs
Symptom onset between 48 hours and 2 weeks with the following symptoms: Sensory loss, vision loss Posterior circulation stroke: binocular diplopia, dysarthria, dysphagia, ataxia
Low-Risk TIAs
All TIAs with symptom onset greater than 2 weeks

Assessment

History: time of onset, differentiate between motor, speech, vision disturbances and posterior circulation symptoms

Physical Exam: vitals, neuro exam, cardiovascular exam (dissection, arrhythmias, valvular pathology), look for comorbidities

Investigations

Labs: CBC, lytes, extended lytes, glucose, BUN, Cr, INR, PTT

ECG: rule out Afib

Neuroimaging: all TIAs (non-contrast CT head in ED), moderate-risk ($\pm$ CTA head & neck), high- to very high-risk (+ CTA head & neck)

Management

General
ABCs, monitors, oxygen, vitals, IV access
High-Risk TIA features
Loading dose: Clopidogrel 300mg PO + ASA 160mg PO Dual antiplatelet therapy: Clopidogrel 75mg PO + ASA 81mg PO daily x 21-28 days Monotherapy (after 21 days): ASA 81mg PO daily
Low-Risk TIA features
ASA 81mg PO daily
Disposition
Stroke clinic follow-up within 3 weeks Risk stratification: CHADS ₂ (stroke risk with Afib), Ottawa TIA Risk Score Outpatient carotid dopplers or CT angiogram +/- endarterectomy

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 101. Canadian Stroke Best Practices, 2018. Stroke 2018; 49:2278-2279. Int J Stroke. 2019; 14(7) 756-751.

Wound Management

Definitions:

Primary Closure: closure immediately (via sutures or staples)

- Leads to faster healing and best cosmetic result
- Best for wounds within 8 hrs of presentation or face wounds within 24 hrs

Secondary Closure: wound heals naturally, without surgical closure

- Best for small partial thickness avulsions or fingertip amputations.

Delayed/Tertiary Closure: initial wound cleansing/dressing followed by packing, then primary closure after 72 hrs

- Best when patients present late (>24 hrs) or contaminated crush wounds

Closure Techniques: simple interrupted/running suture is most common method of closure in the ED. Other methods include: staples; horizontal mattress; hair apposition (all good for scalp lacs) and vertical mattress (gaping, deep wounds); tape or glue.

Assessment

Suture Types:

Absorbable	Non-Absorbable (more common in ED)
Braided: • Vicryl • Vicryl Rapide Monofilament: • Monocryl • Fast absorbing gut • Chromic gut	Braided: • Ethibond • Silk Monofilament: • Ethilon (Nylon)

Wound Preparation:

1) Debridement 2) Wound Cleansing 3) Irrigation

Suture Sizes:

3-0 sutures on **Three** places: chest, abdomen, back

4-0 sutures on **Four** extremities: arms and legs

5-0 sutures where you have **Five** fingers: hands

6-0 sutures (**S**)ensitive areas: face, genitals

Timing of Removal:

Face: 3-5 days

Scalp & arms: 7-10 days

Tunk, legs & dorsal hands/feet: 10-14 days

Palms & soles: 14-21 days

Management

Antibiotic Prophylaxis	Indicated in gross contamination, severe crush injuries, open fractures, wounds involving joints (including MCP, i.e. "fight bites") or cat bites, intra-oral lacs, immunocompromised (DM, CKD, chronic steroid use) Ideal Abx: Keflex or Clavulin (bites)
Tetanus Prophylaxis	Give DTaP booster unless last booster was within 10 yrs Given ASAP but can be given days-weeks following injury

Diabetic Emergencies

Definitions

DKA	HHS
Predominantly Type 1 DM Insulin deficiency + stressor → counter-regulatory hormone excess → ↑ lipolysis (ketoacidosis) and osmotic diuresis (dehydration) Serum glucose: >16 mmol/L Other labs: $\text{HCO}_3^- < 15$, pH <7.3 Onset: hours to days Features: dehydration, often young	Predominantly Type 2 DM Relative insulin deficiency + stressor → counter-regulatory hormone excess → osmotic diuresis (dehydration) Serum glucose: >30 mmol/L Onset: days to weeks Features: severe dehydration, hyper-osmolality, often elderly with AMS
Stressor (7 Is): Infection, Infarction, Intoxication, Insulin (dose changed/missed), Incision (surgery), Initial (diagnosis), Impregnated	

Assessment

History: N/V, abdominal pain, polyuria/polydipsia, weakness, anorexia

Physical Exam: rapid, deep breathing (Kussmaul) respirations, tachycardia, ileus, acetone breath

Investigations

Labs: glucose, urine/serum ketones, β-hydroxybutyrate, CBC, lytes, extended lytes, glucose, BUN, Cr, VBG, lactate +/- cultures, β-HCG, cardiac enzymes (if indicated)

Management

Fluid Resuscitation
NS 1-2 L over 1 hours Change to D5½NS when BG <16
Insulin
Short acting insulin Regular Infusion of 0.1 U/kg/h (goal = lower BG by 4-5) Once gap closed: continue infusion x 1 hr but overlap + switch to SC insulin
Electrolyte Replacement
Potassium K <3.3 mmol/L: hold insulin and give 40mmol/L KCl K 3.3-5 mmol/L: give 20-30mmol/L KCl K >5 mmol/L: re-check K in 1-2 hours Phosphate: Low phosphate can be replaced if severe levels or metabolic disturbances (muscle weakness, paralysis, rhabdomyolysis) Sodium: Pseudohyponatremia common due to dilutional decrease
Disposition
Admission if: first time presentation, comorbidities, unable to close gap, iatrogenic complications (ARDS, cerebral edema, fluid overload), or DKA/HHS due to stressors listed above (i.e. need to manage MI or sepsis in hospital) Education: diet, insulin administration, fluid replacement

[EMOttawa Blog Post](#)

End-of-Life Care

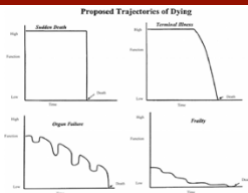
Trajectories of Dying:

Sudden death (15%): i.e. cardiac arrest, trauma

Terminal illness (30%): predictable decline in 6 months or less i.e. cancer or terminal AIDS

Organ failure (30%): gradual decline with intermittent exacerbations i.e. COPD, CHF

Frailty (30%): gradual decline, lingering course over many years i.e. dementia



Palliative Performance Scale (PPS) & Eastern Cooperative Oncology Group (ECOG) Performance Status:

Stable	PPS 70-100%
Transitional	PPS 40-60%
End-of-Life	PPS <30%, bedbound
Grade	ECOG
0	No restrictions, able to carry on pre-disease performance
1	Restricted physically strenuous activity, able to carry out light/sedentary work
2	Ambulatory, able to carry out self-care, unable to carry out working activities, up >50% waking hours
3	Capable of only limited self-care, confined to bed/chair >50% waking hours
4	Completely disabled, cannot carry out self-care, totally confined to bed/chair
5	Death

Investigations

Consider discontinuation of cardiac monitoring, vital signs and bloodwork and minimization of IV fluids. Given presenting symptoms of the patient and their goals of care, further investigations are at times appropriate. Discontinue only meds and devices (i.e. catheters, NG tubes) not contributing to patient comfort.

Symptom Management

Agitation/Delirium	Haloperidol 0.5mg-1mg SC/IV q2h PRN - if severe add Methotrimeprazine 12.5-25mg SC q4h PRN - if severe add Midazolam 0.5-1mg SC/IV q30min PRN
Pain/Dyspnea	Opioid Naive: SC route preferred over IV (due to longer $t_{1/2}$) - Morphine 1-2mg SC/IV q30min PRN OR - Hydromorphone 0.2-0.4mg SC/IV q30min PRN If patient on regular oral opioid medication, convert current regime to SC dosing: Morphine: 10mg (PO) = 5mg (SC/IV) equals Hydromorphone: 2mg (PO) = 1mg (SC/IV) O2 - if hypoxic or for patient's comfort Fan blowing - improves patient comfort
Secretions	Re-position, reassure family that secretions not causing distress Glycopyrrolate 0.4mg SC/IV q4h PRN
Nausea/Vomiting	Metoclopramide 5-10mg SC/IV q4h PRN (contraindicated in bowel obstruction) OR Haloperidol 0.5-1mg SC/IV q4h PRN

EMottawa Blog Posts: [Palliative Care Part 1](#), [Part 2](#), [GOC Discussion](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 9th ed, 2017; Chapter e9. J Palliat Med 2018. 21(11):1651-1661. J Palliat Care. 1996; 12(1):5-11. CJEM 2020 22(5) 626-628.

Sepsis

Definitions

Sepsis	
Life threatening organ dysfunction caused by a dysregulated host response to infection with a qSOFA score ≥ 2	
Septic Shock	
Vasopressor requirement to maintain a MAP ≥ 65 Serum lactate ≥ 2 mmol/L in the absence of hypovolemia	
Clinical tools to aid in sepsis recognition	
SIRS	2 or more of: T <36 or >38.3 HR >90 RR >20 or CO ₂ <32 WBC <4 or >12
qSOFA	2 or more of: GCS <15 RR ≥ 22 sBP <100

Assessment

History: associated symptoms, full review of systems, comorbidities

Physical Exam: vitals, volume status, look for a focus (respiratory, urine, abdomen, skin, blood, brain, permanent lines)

Investigations

Full Septic Workup: CBC, lytes, extended lytes, BUN, Cr, LFTs, VBG, lactate, INR/PTT, blood C+S, urine C+S, ECG, CXR

RUSH Exam: heart (PSL, 4 chamber), IVC view, Morrison's and splenorenal views, bladder window, aorta, pneumothorax

Management

General
Monitors, oxygen, vitals, 2 large bore IVs Early antibiotics (within 1 hour), crystalloids (RL > NS) Endpoints: MAP >65 , capillary refill time, lactate clearance, urine output
Resuscitation
Crystalloids: Ringer's Lactate for patients with hypotension or lactate ≥ 4 Vasopressors: Norepinephrine 5-10 μ g/min (if not fluid responsive), Vasopressin 0.04 U/min (if moderate doses of NE being used) Steroids: if refractory to fluids + pressors or on chronic steroids
Antibiotics
Empiric treatment: Pip-Tazo 3.375g IV +/- Vancomycin 1-1.5g IV Respiratory: Ceftriaxone 2g IV + Azithromycin 500mg IV Urinary: Ceftriaxone 2g IV + Tobramycin 3-5mg/kg (single dose) Meningitic doses: Ceftriaxone 2g IV + Vancomycin 2g IV + Dexamethasone 10mg IV (within 1 hr of Abx) +/- Acyclovir 1g IV (for HSV encephalitis)
Disposition
Admission to medicine +/- ICU (if requiring vasopressors or intubated)

[EMOttawa Blog Post](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 6. NEJM 2001; 345(19): 1368-77. JAMA 2016; 315(8): 801-10. Crit Care Med 2017; 45(3): 486-552. JAMA 2019; 321(7): 654-664. AJRCCM 2019; 199(9): 1097-1105.

Acid-Base Disorders

Etiology of Acid-Base Disorders

Respiratory Acidosis (Acute)	Airway obstruction Pulmonary disease Thoracic trauma	CNS depression Neuromuscular disorders Toxicologic (opioid OD)
Respiratory Alkalosis	Hyperventilation Panic attack Pulmonary disease Hyperthyroidism	Pregnancy Sepsis Sympathomimetics Salicylate toxicity
Elevated Anion Gap Metabolic Acidosis (CATMUDPILES)	CO poisoning Aminoglycosides Theophylline Methanol Uremia DKA	Paraldehyde Iron, Isoniazid Lactic acidosis Ethanol, Ethylene glycol Salicylate toxicity
Normal Anion Gap Metabolic Acidosis (HARDUP)	Hyperalimentation Acetazolamide use Renal tubular acidosis	Diarrhea Uretero-enteric fistula Pancreatic fistula
Metabolic Alkalosis	Vomiting, NG suction Diuretics Massive transfusion	Hyperaldosteronism Hypercortisolemia Hypoalbuminemia Hypercalcemia

Investigations

Labs: CBC, Cr, lytes, extended lytes, TSH, VBG, lactate, tox screen. For suspected: DKA/AKA, add add B-hydroxybutyrate; ASA overdose, add salicylate level; ingestion, add toxic alcohol screen

Imaging: Consider based on type of acid-base disturbance and suspected cause, i.e. CXR for suspected pulmonary disease

Disorder	pH	PCO ₂	HCO ₃ ⁻	Expected Compensation*
Resp. Acidosis	↓	↑	↑	↑ HCO ₃ ⁻ = 0.1 ΔPCO ₂ (acute) ↑ HCO ₃ ⁻ = 0.4 ΔPCO ₂ (chronic)
Resp. Alkalosis	↑	↓	↓	↓ HCO ₃ ⁻ = 0.2 ΔPCO ₂ (acute) ↓ HCO ₃ ⁻ = 0.5 ΔPCO ₂ (chronic)
Metab. Acidosis	↓	↓	↓	↓ PCO ₂ = ~1 ΔHCO ₃ ⁻
Metab. Alkalosis	↑	↑	↑	↑ PCO ₂ = ~1 ΔHCO ₃ ⁻

*Inappropriate compensation indicates a second acid-base disorder

Anion Gap: $\text{Na} - (\text{Cl}^- + \text{HCO}_3^-)$; Normal AG = 8 - 12; ↑ likely metabolic acidosis
If metabolic acidosis is present, calculate a delta gap and use Winter's formula
Winter's Formula: $\text{pCO}_2 = 1.5\text{HCO}_3 + 8 \pm 2$ (for metabolic acidosis)

Management

Acute Respiratory Acidosis = PCO₂ > 40 mm Hg, pH < 7.35
Relieve airway obstruction, treat underlying cause, supplemental O ₂ if hypoxic Non-invasive or mechanical ventilation as needed
Respiratory Alkalosis = PCO₂ < 38 mm Hg, pH > 7.45
Find and treat underlying cause
Metabolic Acidosis = HCO₃⁻ < 24 mmol/L, pH < 7.35
Find and treat underlying cause
Metabolic Alkalosis = HCO₃⁻ > 40 mmol/L, pH > 7.45
Rarely causes dangerous alkalemia. Find and treat underlying cause

ENT Emergencies - Vertigo

Important Causes

Benign Paroxysmal Positional Vertigo (BPPV)

Short lived (20-30 secs) vertigo brought on by lying down, turning over or getting out of bed. Resolves when still. **No spontaneous nystagmus.**

Vestibular Neuritis

Hours or days of constant severe vertigo, worse with head movements. Difficult with gait. **Spontaneous or gaze evoked nystagmus** in first few days. Resolves over a few weeks.

Posterior Circulation Stroke

Can present similar to vestibular neuritis. May have focal paresthesia, weakness, headache or neck pain, and Deadly Ds: dysarthria, diplopia, dysmetria, dysphonia, dysphagia.

Vestibular Migraine

Often under-diagnosed. Multiple episodes of dizziness lasting minutes to days. History of migraines. Half of the episodes have either typical migraine headache, and/or associated photophobia/phonophobia.

Other less common causes: Meniere's, Multiple Sclerosis, labyrinthitis, other central causes (cerebellar hemorrhage, PICA stroke, head trauma)

Assessment

Positional Testing (Dix-Hallpike or Roll Test): if short episodes initiated with head movement and without spontaneous or gaze evoked nystagmus

HINTS Exam: if constant vertigo and nystagmus present

Central Cause: neuro exam, gait and coordination exam

Dix-Hallpike Test (diagnose posterior-canal BPPV)

Head turned 45° to one side while patient sitting. Patient moved to supine position with head hanging over edge of bed. Observe for vertical upward or rotatory nystagmus. Repeat with patient looking 45° in other direction.

Roll Test (diagnose horizontal-canal BPPV)

Patient initially supine, head on bed. Turn head 90 ° to one side, observe for horizontal nystagmus. Both sides will show horizontal nystagmus.

HINTS Exam (differentiate vestibular neuritis vs. posterior stroke)

Must have all three to be diagnosed vestibular neuritis:

Head Impulse: corrective saccade as examiner turns head away from direction of spontaneous nystagmus

Nystagmus: unidirectional horizontal/rotatory nystagmus

Test of Skew: no vertical or slanted eye movements on cover-uncover test

Management

Peripheral

Epley's Maneuver for PC BPPV, Gufoni for HC BPPV

Consider steroids for vestibular neuritis (evidence poor)

Central

Neuroimaging, neuro consult +/- stroke management

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 19.

ENT Emergencies

Epistaxis

Causes: trauma (nasal, digital, facial), URI, allergies, low humidity, polyps, foreign body, idiopathic causes (familial), systemic causes (atherosclerosis, anticoagulation, pregnancy, coagulopathies, diabetes, liver disease)

Assessment: visualize nares + oropharynx for active bleeding

Labs: CBC, INR/PTT +/- cross+type

Management

General
ABCs, vitals, volume assessment Initial step: compress cartilaginous part of nose x 20 mins Next step: compress with Lidocaine/Epinephrine/decongestant-soaked pledget +/- topical TXA +/- Silver nitrate if able to identify site
Anterior Bleeds (90% Little's area/Kesselbach's plexus)
Anterior packing: nasal tampon, rhino rockets or Vaseline gauze pack Apply anterior pack to active side first, if ineffective, pack both nares
Posterior Bleeds
Epistat or Foley catheter. Apply traction once inserted. Keflex x 5 day course or until pack removal to prevent TSS

Pharyngitis

Etiology: viruses (rhinovirus, adenovirus), bacterial (Group A Strep)

Assessment

History: odynophagia, URI symptoms, complications are rare (ie. rheumatic fever)

Physical Exam: vitals, ABCs, red flags

Can't Miss Diagnoses
Peritonsillar abscess: muffled voice, uvular deviation Retropharyngeal abscess: drooling, airway compromise Tracheitis: may be confused with croup, stridor, labored breathing Epiglottitis: fever, stridor, rapidly progressive swelling

Modified Centor Criteria	
Age 3-14 years old = +1 15-44 years old = 0 >44 years old = -1	Tonsillar exudates = +1 Tender anterior cervical lymph nodes = +1 Temp >38°C = +1 Absent cough = +1

Management: fluids, antipyretics, single dose Dexamethasone may reduce pain/duration.

Antibiotics reduce symptoms by 16 hours. They do NOT reduce incidence of suppurative complications.

Electrolyte Disturbances

History: review of systems, neurologic symptoms (headache, lethargy, weakness, muscle cramps, ↓ LOC, personality changes, seizures), comorbidities, infection, intake + losses, past history of electrolyte disturbances

Hyperkalemia: [K] >5.5 mmol/L

Causes
Pseudohyperkalemia (#1), chronic renal failure, acute acidosis, medications* (ACEi, NSAIDs, K-sparing diuretics, Digoxin, Septra), cell death (rhabdo, burn/crush injuries, hemolysis, tumour lysis syndrome)
ECG Changes
Rate (brady, junctional), conduction (wide PR/QRS), axis change, peaked Ts
Management
Protect: 1 amp CaCl or 3 amps Ca gluconate (*if ECG changes noted) Shift: 1-2 amps D50W + 10 U regular insulin, albuterol nebs +/- bicarbonate (if acidotic) Excrete: fluids, Lasix, PEG3350 +/- dialysis if critical K or unable to excrete

Hypokalemia: [K] <3.5 mmol/L

Causes
Renal losses (diuretics), non-renal losses (vomiting, diarrhea), metabolic alkalosis
ECG Changes
Rhythm (ectopy, TdP), conduction (U wave), Diffuse ST depression, flat/inverted T
Management
Replace: KCl 10-20mmol/hr IV or KCl 40-60mmol PO q2-4hrs HypoMg: MgSO ₄ 500mg/h IV to ensure K being driven into cells

Hyponatremia: [Na] <135 mmol/L

Causes
Hypo-osmolar most common - hypervolemic (CHF, cirrhosis, nephrotic syndrome), euvoletic (SIADH), hypovolemic (adrenal insufficiency, vomiting, diarrhea, diuretics, poor PO fluid intake)
Management
Known acute (<24-48 hrs) [Na]<120 or symptomatic (↓ LOC, focal neurological symptoms): max Na 8mmol/L in 24 h to prevent central pontine myelinolysis Dose option: IV 3% saline 100cc IV over 10 mins (if seizing)

Hypercalcemia: [Ca] >2.6 (corrected for albumin)

Causes
Malignancy (breast, lung, kidney, multiple myeloma), hyperPTH, granulomatous diseases, meds (thiazides, Li, estrogen, vitamin A/D toxicity)
ECG Changes
Short QT, ST elevation, bradyarrhythmias, AV block
Management
Bolus NS until normal perfusion, then infusion to 200cc/hr with goal of urine output 2L/day. Lasix to promote diuresis, bisphosphonates and calcitonin.

Urologic Emergencies

Renal Colic

Risk Factors: hereditary (RTA, G6PD deficiency, cystinuria, oxaluria), lifestyle (minimal fluid intake, excess vit C, oxalation, purines, calcium), meds (loop diuretics, acetazolamide, topiramate), medical conditions (UTI, IBD, gout, DM, hypercalcemia), obesity

Assessment

History: unilateral flank pain +/- radiating to groin, "writhing" in pain, N/V, trigonal irritation (frequency, urgency)

Physical Exam: vitals (fever, HR, RR), abdominal exam, CVA tenderness

Investigations: CBC, urinalysis, B-hCG (females)

CT
Vast majority do NOT need CT imaging Relative indications: first presentation of renal colic, complicated renal colic, elderly patients, suspicion of a serious alternative diagnosis
Ultrasound 🔍
Most helpful in detecting hydronephrosis (98% sensitivity)
KUB
Plain X-rays are neither sensitive nor specific for detection of renal stones. KUB may be used to follow stone progression.

Management

General	IV NS if clinically dehydrated
N/V	Zofran 4-8mg IV
Analgesia	Morphine 6mg IV + Ketorolac 30mg IM/IV or Naproxen 500mg PO
MET	Tamsulosin 0.4mg PO OD x3 weeks (large stone >4mm or distal stones)
Disposition	Can be safely discharge with appropriate GP/urology follow-up
Urology consult	Intractable pain, infected stone, compromised renal function (single kidney, transplanted kidney, bilateral obstruction)

UTI and Pyelonephritis

Causes: E. coli (85%), Klebsiella, Proteus, Saprophyticus

Assessment

History: UTI (frequency, urgency, dysuria, hematuria), pyelo (fever/chills, flank pain, N/V), associated vaginitis/cervicitis symptoms, sexual history

Investigations: urine dipstick, urine R+M, urine C+S +/- CBC, BUN, Cr

Management

Uncomplicated UTI
Septra DS PO BID x 3 days Macrobid 100mg BID x 5 days If suspected STI: Levofloxacin 500mg PO daily x 7d + CTX 250mg IM x1
Complicated UTI/Uncomplicated Pyelonephritis
Ciprofloxacin 500mg PO BID or Septra DS PO BID x 10-14 days Consider US/CT imaging for complicated UTI
Complicated Pyelonephritis
Ceftriaxone 1g IV q24h

Environmental Injuries

Hypothermia (T <35°C)

Causes: ↑ heat loss (EtOH, environmental), ↓ thermogenesis (hypothyroidism, hypoglycemia, adrenal insufficiencies), impaired thermogenesis (toxins, CNS lesions, SC injury)

Risk Factors: low SES, age extremes, drug OD, psych comorbidities

Assessment

Mild (32° - 35°C): excitation response (↑ HR/BP/RR, shivering)

Moderate (28° - 32°C): physiologic slowing, NO shivering, AMS, ataxia

Severe (24° - 28°C): dysrhythmias (brady>slow Afib>Vfib>asystole), irritable myocardium (avoid invasive heart procedures), fixed/dilated pupils

Investigations

Labs: CBC, lytes, BUN, Cr, VBG, lactate, INR/PTT, glucose

Tests: ECG (Osborne waves), pCXR (aspiration, pulmonary edema)

Management

General
Monitors, O ₂ , IV access, vitals, esophageal or Foley temp Remove wet clothes, rewarming strategies
Cardiac Arrest
Modified ACLS protocol (pulse checks 60 mins, shock x3 cycles then wait >30°C, withhold Epi until >30°C then double interval until >35°C)
Passive Rewarming (T >32°C)
Cover patients with insulating blanket, let body generate heat
Active Rewarming (T <32°C)
Active External: warming blankets, radiant heat, heating pads Active Internal: warm IV fluids, warmed O ₂ , dialysis, ECMO, heated cavity lavage (pleural, gastric, peritoneal, bladder)

Heat Stroke (T >40.5°C)

***Differentiated from heat exhaustion by AMS/elevated LFTs**

Classic/Non-Exertional: elderly, heat waves, indoors with no AC

Exertional: young athletes, runners

Assessment

Classic: dry/hot skin, not always dehydrated, HIGHER mortality

Exertional: diaphoretic, profound dehydration, more morbidities (liver failure, renal failure, DIC, lactic acidosis)

Management

General
Monitors, cooled IV fluids, rapid evaporative cooling Antipyretics NOT effective (as not a hypothalamus problem, can also make DIC/liver failure worse)
Treat Symptoms
Shivering: Midazolam 2mg IV Rhabdomyolysis: IVF, Lasix, NaHCO ₃ Seizures: Lorazepam 2mg IV Hyperkalemia: protect, shift, eliminate

EMOttawa Blog Posts: [Hypothermia Part 1](#), [Part 2](#)

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 138+139. Resuscitation 2015;95:148-201.

Orthopedic Injuries - Upper Limb

Assessment

History: mechanism of injury, associated neurological symptoms, blood loss

Exam: ABCs + vitals, look + feel, active and passive ROM, neurovascular status, assess bleeding/open fractures, skin tenting, complications of compartment syndrome, examine joint above and below fracture

Investigations: radiographs as clinically indicated

Upper Limb

Distal Radius Fracture🔍: FOOSH. Several fracture patterns. Colle's fracture is most common (distal radial fracture with dorsal displacement, volar apex angulation, and is extra-articular).

Exam: "dinner fork deformity" if dorsally angulated as in Colle's fracture.

Management: hematoma block, reduction to restore radial length and correct dorsal angulation. Success of reduction depends on several factors (intra- vs. extra-articular, comminution, quality of cast mold).

Colle's Fracture



Scaphoid Fracture🔍: 15-40yo with FOOSH. High complication rate (5-40% with AVN/non-union).

Exam: limited wrist/thumb ROM, snuff box tenderness, axial loading of 1st MC, pain to scaphoid tubercle volarly.

Management: thumb spica splint for suspected fractures (even if negative X-ray) x 6-12 weeks, repeat imaging in 10 days.

Scaphoid Fracture



Proximal Humeral Fracture🔍: high energy trauma (young), FOOSH (elderly). Neer classification to determine 1/2/3/4 part fracture. Separate part if displaced > 1cm or >45° angulation.

Management: minimally displaced (sling or cuff-and-collar immobilization), displaced GT or 2/3/4 part in younger patients (ORIF)

Proximal Humeral Fracture



Boxer's Fracture🔍: blow on distal-dorsal aspect of closed fist. Volar angulation of neck of 5th metacarpal into palm.

Management: closed reduction if angulation >40°. If stable, ulnar gutter splint for 4-6 weeks.

Boxer's Fracture



Orthopedic Injuries - Lower Limb

Assessment

History: mechanism of injury, associated neurological symptoms, blood loss

Exam: ABCs + vitals, look + feel, active and passive ROM, neurovascular status, assess bleeding/open fractures, skin tenting, complications of compartment syndrome, examine joint above and below fracture

Investigations: radiographs as clinically indicated, use decision rules for ankle/foot/knee to guide assessment

Lower Limb

Ankle Fracture🔍: inversion/eversion injury. Risk-stratification based on Weber's classification.

Weber A: below syndesmosis - typically stable

Weber B: at level of syndesmosis - can be unstable

Weber C: above level of syndesmosis - always unstable

Management: non-operative (non-WB BK cast), operative (most of Weber Type B/all Type C)

Weber A Fracture



Jones Fracture🔍: Stress injury. Midshaft 5th MT fracture (>15mm from proximal end of 5th MT). High incidence of non-union.

Management: non-WB BK cast x6 weeks.

Pseudo-Jones Fracture: Traumatic injury. Proximal tubercle of 5th MT (<15mm from proximal end of 5th MT). Non-union is uncommon.

Management: protective weight bearing in stiff soled shoe or boot.

Jones Fracture



Hip Fracture🔍: fall (elderly), direct force to hip, rotational force.

Garden I: incomplete, valgus impacted

Garden II: complete, non-displaced

Garden III: complete, partially displaced

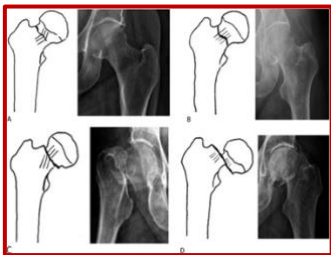
Garden IV: complete, fully displaced

Exam:

shortened/abducted/externally rotated leg, painful ROM

Management: Elderly may get hemi or total hip arthroplasty. Young adults get ORIF.

Garden Classification



Pediatric Orthopedic Emergencies

General:

History: mechanism of injury, associated neuro symptoms, blood loss, consider non-accidental trauma

Exam: Vitals, look + feel, active & passive ROM, neurovascular status, assess bleeding/open #s, skin tenting, signs of compartment syndrome (tight compartments, pain w passive stretch), examine joint above & below fracture

Investigations: X-ray, rarely MRI

Physeal Fractures:

Use Salter-Harris classification with **SALTER** mnemonic

Type 1	Straight through physis
Type 2	Above physis
Type 3	Lower than physis
Type 4	Through Everything (above & below)
Type 5	Rammed (erasure of physis)

Supracondylar Fractures:

Most common elbow fracture in peds. Can mimic ossification centres. Remember with **CRITOE** mnemonic (average age of appearance)

Capitulum	1 year
Radial head	3 years
Internal (medial) epicondyle	5 years
Trochlea	7 years
Olecranon	9 years
External (lateral) epicondyle	11 years



Ossification Centres

Atraumatic Pediatric Hip Pain:

Septic Arthritis	Bacterial joint infection of the hip, M > F, S. Aureus (most common)
Transient Synovitis	Most common cause of atraumatic hip pain, self-resolving inflammatory process, often post-viral or minor trauma, peak 3-6 yrs, M > F
Legg-Calve-Perthes Disease (LCPD)	Idiopathic necrosis of capital femoral epiphysis, M > F, peak 3-11 yrs, mostly unilateral but can be bilateral
Slipped Capital Femoral Epiphysis (SCFE)	Slippage of the proximal femoral epiphysis, M > F, peak 8-15 yrs, frequently bilateral, most common hip disorder in adolescents RFs: obesity, M > F, endocrinopathies

Assessment

Septic Arthritis	Typically febrile, unable to weight bear, erythema, limited passive ROM, swelling/warmth/erythema, irritability/lethargy/malaise, hip in flexion, external rotation, and abduction
Transient Synovitis	Typically afebrile, able to weight bear with pain, hip in flexion, external rotation, and abduction

LCPD	Limp, Trendelenburg sign, limited internal rotation and abduction of hip, pain can be referred to knee or groin, worse with activity, lower limb length disparity in severe cases
SCFE	Poorly localized pain, often referred, antalgic gait, limited hip internal rotation, Drehmann sign (external rotation with hip flexion)

Investigations

Septic Arthritis	CBC, ESR, CRP, blood cx, X-rays, US, synovial fluid cell count, differential, gram stain, and cultures Kocher criteria (see below)
Transient Synovitis	Same as septic arthritis (must exclude septic arthritis)
LCPD	Hip x-rays (AP pelvis, frog leg lateral)
SCFE	Hip x-rays (AP pelvis, frog leg lateral +/- cross-table lateral)

Kocher Criteria (Walk FEW):

Elements (+1pt each)	Interpretation (%Risk)
Inability to Walk/Weight bear	0 pts (0.2%)
Fever >38.5°C	1 pt (3%)
ESR >40	2 pts (40%)
WBC >12	3 pts (93%)
	4 pts (99%)

Management

Septic Arthritis	IV antibiotics Consult Ortho
Transient Synovitis	Symptom management (NSAIDs, joint rest) Dispo home Re-assessment if symptoms persist or worsen
LCPD	Consult Ortho
SCFE	Non-weight bearing Consult Ortho

Common Forearm Injuries:

Nightstick Fracture: Isolated ulnar shaft fracture by direct blow to forearm.

Buckle Fracture: Linear compression resulting in buckling of bone without cortical disruption. Common at metaphyseal-diaphyseal junctions.

Greenstick Fracture: Fracture disrupting the cortex unilaterally.

Nursemaid's Elbow: Radial head subluxation. Radial head displaced from annular ligament typically due to axial traction on forearm. Clinical dx treated with supination/flexion or hyperpronation maneuvers.



Nightstick

Greenstick

Pediatric Fever

Definitions

Fever: Temp $>38.0^{\circ}\text{C}$, rectal most accurate

Fever without a source: <3 yo, no identifiable source of fever after initial history/exam

Fever of unknown origin: fever lasting 2+ weeks, without known cause after initial investigations

Serious Bacterial Infection (SBI): includes UTI, meningitis, bacteremia

Invasive Bacterial Infection (IBI): includes meningitis, bacteremia

Differential Diagnosis

Respiratory tract infections (viral = most common or pneumonia), UTI, otitis media, skin infections, GI (i.e. gastroenteritis), surgical (i.e. appendicitis), environmental (i.e. hyperthermia), rheumatologic (i.e. JIA), oncologic (i.e. ALL), Kawasaki disease ($\geq 5\text{d}$ fever)

Assessment

Pregnancy and Birth Hx: Gestational hx (i.e. U/S, maternal immunization status, screening), birth hx (fever, prolonged labour, GBS status), NICU stay

PMHx: Immunization status, previous infections or surgeries

Fever Hx: duration (clarify if daily fever or breaks between fevers), route of measurement

Important Notes

1. Treat tactile fever equally as a documented fever in neonates
2. Rectal = most reliable

History: Localizing symptoms

(cough/congestion/sore throat, ear pain, headache/neck stiffness, N/V/D, dysuria/frequency/urgency/hematuria, rash), lethargy (i.e. somnolence), irritability (i.e. inconsolable), decreased po intake, urine output, aLOC, seizures, sick contacts, travel history, exposure to genital/oral herpes

Physical Exam: Vitals, pediatric triangle (see below)

CV/Resp: tachycardia, crackles, wheezing

Abdo: organomegaly, masses, guarding

Eyes: conjunctivitis, discharge

ENT: tympanic membrane, external ear canal, oropharynx (Koplik spots in measles, pharyngitis, strawberry tongue, herpetic lesions, mucositis)

GU: candida, diaper dermatitis

Neuro: nuchal rigidity, bulging fontanelle, lethargy, focal neuro signs

Skin: petechiae, viral exanthems

Pediatric Triangle (ABC):

Appearance	TICLS: Tone, Interactiveness, Consolability, Look and gaze, Speech and cry
Breathing	Tripod position, tachypnea, hot potato voice, stridor, wheezing, retractions (suprasternal, supraclavicular, intercostal, subcostal), grunting, nasal flaring, head bobbing, seesaw breathing
Circulation	Delayed cap refill, extremity mottling, cyanosis

Important Bacterial Pathogens

0-28d	GBS, E. coli, Listeria
29-60d	H. influenza, S. pneumo, N. meningitides, E. coli
>60 d	S. pneumo, N. meningitides, E. coli

Investigations

IMs = inflammatory markers (procalcitonin > 0.5ng/mL, CRP >20 mg/L, ANC > 5.2x10⁹/L, T>38.5°C if procalcitonin not available)

0-60 days	
Everyone	Urinalysis (U/A), urine cx if +U/A, blood cx, IMs*
0-21d	LP, urine cx always with U/A
22-28d	-ve IMs: shared-decision making regarding LP +ve IMs: LP
29-60d	-ve IMs: no LP +ve IMs: shared decision-making regarding LP
60+ days	
U/A (cx if +ve), others (i.e. CBC, CXR) rarely needed unless fever ≥5 d	

Urine: bag sample or midstream to screen, catheterization or midstream urine for culture if starting Abx

Considerations for CXR: persistent tachypnea despite fever treatment, abnormal SpO₂ i.e. <95%, increased work of breathing, focal findings on auscultation, persistent fever x5 days, chest pain

CSF studies: Tube 1 (culture + sensitivity), Tube 2 (protein + glucose), Tube 3 (cell count + diff), Tube 4 (+/- HSV PCR, hold for other studies)

Management

Initial Doses:

Ampicillin = 50mg/kg IV q6h (200mg/kg/day)

Tobramycin = 7mg/kg IV q24hr (7mg/kg/day); monitor trough levels

Acyclovir = 15-20mg/kg IV q8h (45-60mg/kg/day)

Cefotaxime = 50 mg/kg IV q6h (200mg/kg/day)

Age	Antimicrobials		Disposition
0-21d	Amp & Tobra, +/- Acyclovir (if HSV risk factors/unwell)		Admit
22-28d	Positive IMs	+ve UA or +ve IMs with -ve CSF: Amp & Tobra +ve CSF: Cefotaxime +/- Amp	Admit
	Negative IMs	Consider Ceftriaxone -ve CSF: shared decision making (admit vs discharge)	Admit if no LP
29-60d	Positive IMs	-ve CSF: Amp & Tobra +ve CSF: Cefotaxime +/- Amp	Admit
	Negative IMs	D/C home Abx if +ve UA: PO Keflex or Cefixime +/- IV dose x1	Discharge
>60d	Treatment based on specific cause		

General Surgery Emergencies

Biliary Pathologies:

Cholelithiasis: Presence of gallstones (cholesterol pigmented or mixed)

Risk Factors (5F's): Forties, Female, Fertile, Fatty tissue (obese), FHx

Biliary Colic	Transient cystic duct obstruction
Cholecystitis	Gallbladder (GB) inflammation due to persistent cystic duct obstruction
Choledocholithiasis	Common bile duct (CBD) gallstone obstruction
Gallstone Pancreatitis	Gallstone blocking pancreatic duct leading to obstruction and inflammation
Cholangitis	Infection of the biliary tract due to choledocholithiasis

Assessment

Biliary Colic	RUQ pain, N/V Post-prandial (fatty meals), w/in hrs of last meal Intermittent, should dissipate Radiation to right shoulder/scapula Entire episode lasts approx 1-6 hrs
Cholecystitis	RUQ pain, N/V Fever +ve Murphy's sign Often previous hx of biliary colic
Choledocholithiasis	RUQ pain, N/V Jaundice Can lead to cholangitis or gallstone pancreatitis
Ascending Cholangitis	N/V, hypotension Charcot's Triad: Fever, jaundice, RUQ pain Reynold's Pentad: Charcot's triad, hypotension, aLOC

Investigations

Labs: CBC, BUN, Cr, lytes, LFTs, total & direct bilirubin (direct $> \frac{1}{2}$ total with GB obstruction), lipase (or amylase depending on centre) +/- VBG, blood cx

Imaging: RUQ US or CT (if broader Ddx); if dx remains unclear → MRCP or ERCP
+/- EUS, oral cholecystography, hepatobiliary iminodiacetic acid (HIDA scan)

Pathology	WBC	ALP	GGT	ALT	AST	Bili
Biliary Colic	N	N	N	N	N	N
Cholecystitis	↑	N / ↑	N / ↑	N / ↑	N / ↑	N
Choledocholithiasis	N	↑	↑	N / ↑	N / ↑	↑
Ascending Cholangitis	↑	↑	↑	↑	↑	↑

Management

Biliary Colic
Correct fluid and electrolyte imbalances, symptom management Outpatient Gen Sx referral: Cholecystectomy considered for ≥ 2 episodes or crescendo biliary colic (i.e. symptoms impacting daily function/unable to eat) Dispo: Home
Cholecystitis
Correct fluid and electrolyte imbalances, symptom management, NPO Abx: Ceftriaxone (CTX) Dispo: Inpatient Gen Sx for cholecystectomy (chole)
Choledocholithiasis
Correct fluid and electrolyte imbalances, symptom management, NPO Dispo: Inpatient GI for ERCP, Gen Sx for eventual chole
Cholangitis

Correct fluid and electrolyte imbalances, symptom management, NPO
 Hemodynamic stabilization
Abx: CTX & Flagyl; Pip-Tazo if severe
Dispo: Often sick and require ICU or stepdown, GI for ERCP, Gen Sx for percutaneous drain (temporizing in critically ill) and eventual chole

Acute Appendicitis:

Pathophysiology: Obstruction, inflammation, and infection of appendix

Background: Most common surgical cause of acute abdo pain in pts <50

Assessment

History	Progressively worsening periumbilical pain Migrates to RLQ within 12h N/V, fever, anorexia
Physical Exam	McBurney's point tenderness, Psoas & Obturator signs, Rovsing's sign, rebound tenderness, Jump test (peds) Focal peritonitis (guarding, rigidity) suggests perforation
Pitfalls	<50% pts have classic migratory pain Acute appendicitis can be very difficult to exclude/diagnose

Investigations

Labs: CBC, Cr, lytes, LFTs, bili, lipase (amylase), CRP/ESR (peds), U/A, blood cx

Imaging: Abdo US, CT (larger body habitus or -'ve US), MRI (pregnancy)

Scores: Alvarado score, Pediatric appendicitis score, Appendicitis inflammatory response score

Management

Supportive: NPO, IV fluids, analgesia, anti-emetics

Abx: CTX & Flagyl

Dispo: Admit, Gen Sx for appendectomy

Diverticular Disease:

Diverticulosis	Colonic diverticula
Uncomplicated	Diverticulosis + peri-colonic fat inflammation +/- micro abscess/perforation
Complicated	Diverticulitis + significant abscess/perforation

Assessment

History: LLQ pain (80% patients in North America), change in bowel habits, N/V/D or constipation, fever, anorexia, dysuria (colovesical fistula), vaginal discharge (colovaginal fistula)

Physical Exam: Fever, LLQ tenderness, focal peritonitis (guarding, rigidity) suggests perforation, palpable mass (complicated/abscess)

Investigations

Labs: CBC, BUN, Cr, lytes, CRP/ESR (may indicate more complicated disease)

Imaging: CT abdo w contrast (esp. in 1st time presenters), US if limited access

Management

Supportive: analgesia, antiemetics, antipyretics

Diet: Clear liquid/soft diet initially, advance to diet as tolerated

Antibiotics: Routine Abx may not be necessary in select patients for uncomplicated diverticulitis. Follow local institution practices.

Uncomplicated: Clavulin 875/125mg PO BID or Cipro 500mg PO BID + Flagyl 500mg PO TID x 5-10 days

Complicated: Admit to Gen Sx, NPO, CTX 1g IV q24h + Flagyl 500mg IV q8h

Toxicological Emergencies

Differential Diagnosis

“Hot and Crazy” (DIMES)
Drug-related: sympathomimetics (cocaine, amphetamines, caffeine, PCP, ketamine), anticholinergics, ASA, SS/NMS/MH, EtOH withdrawal Infection: meningitis, encephalitis, sepsis Metabolic: hypoglycemia, uremia, electrolytes, thyrotoxicosis, pheo Environmental: heat stroke Structural: ICH
“Low and Slow” (ABCD0)
ADHD tablets (clonidine) B-blockers Calcium-channel blockers Digoxin Opiates/Organophosphates

Common Toxidromes

Anticholinergics	
Vitals: hyperthermia, tachycardia Signs: mydriasis, dry skin Symptoms: agitation, hallucination, constipation, urinary retention “dry as a bone, red as a beet, blind as a bat, mad as a hatter, hot as a hare”	Antidepressants Antihistamines Antipsychotics Antispasmodics Atropine Carbamazepine
Cholinergics	
Vitals: hypotension, bradycardia Signs: miosis, diaphoresis, seizures Symptoms: urination, bronchospasm, vomiting, diarrhea	Organophosphates Nerve gas Mushroom Anticholinesterase
Sympathomimetics	
Vitals: hyperthermia, tachycardia, HTN Signs: mydriasis, diaphoresis, seizures Symptoms: agitation, anxiety	Amphetamines Cocaine LSD Ephedrine
Sedative/Hypnotics	
Vitals: hypothermia, hypotension, bradypnea Signs: respiratory depression, miosis (opioids), altered LOC	EtOH, BZDs, GHB Opioids (morphine, heroin, fentanyl) Barbiturates

Basic Approach (ABCDE)

Airway	Intubate early if impending airway compromise
Breathing	Think metabolic derangements if low RR
Circulation	Ensure patient is well perfused
Detect and Correct	Consider universal antidotes (D extrose, O xygen, N aloxone, T hiamine), correct vitals, correct signs (i.e. seizure), consider decontamination/enhanced elimination
Emergency Antidotes	Specific antidotes and treatments

Vascular Emergencies

Ruptured AAA

Risk Factors: FHx, HTN, PVD/CAD, DM, connective tissue disease, smoking

AAA <5cm	AAA 5cm - 7cm	AAA >7cm
0.3% risk of rupture/yr	10% risk of rupture/yr	20% risk of rupture/yr

Assessment

Classic Triad: acute onset back/abdo/flank pain + hypotension (with or without syncope) + pulsatile abdominal mass

Other Presentations: syncope, UGIB/LGIB, high output CHF, ureteral colic, bowel obstruction symptoms, neurological symptoms

Tests: PoCUS^Q to detect AAA (>3cm), ECG, CT aortogram (if stable)

Management

General
ABCs, monitors, oxygen, vitals, IV access STAT vascular surgery consult
Resuscitation
IV crystalloids, blood (aim for systolic BP 90 - 100mmHg) Massive transfusion protocol
Urgent Surgical Intervention
Open surgery with graft replacement or endovascular aneurysm repair
Post-op Complications
Infection: graft contamination or hematogenous seeding Ischemia: Spinal cord ischemia, CVA, visceral ischemia, erectile dysfunction Aortoenteric fistula: commonly presents as GI bleeding Endo Leak: blood flow outside of the graft lumen

Acute Arterial Occlusion

Definition: acute embolus or arterial thrombosis, true emergency as irreversible damage can occur within 6-8 hours

Risk Factors: atherosclerosis, MI with LV thrombus, Afib, valve stenosis, stent/grafts

Assessment

History (6Ps): Pain, Paresthesia, Pallor, Polar (cold), Pulselessness, Paralysis (late finding)

Investigations: Doppler probe to leg with proximal BP cuff - perfusion pressure <50mmHg, ABI <0.5

Management

STAT vascular surgery consult
Immediate heparinization with 4000 IU bolus Revascularization (i.e. embolectomy) vs. CT angiogram (depends on if emboli from Afib vs. secondary to PVD)

Psychiatric Emergencies

Background: ask every patient **MOAPS:** Mood (depression, mania), Organic (EtOH, drug use), Anxiety (worries, obsessions, phobias), Psychosis (hallucinations or delusions) and Suicidal/homicidal thoughts

Suicide Risk Factors: **SAD PERSONS:** Sex (male); Age (<19 or >45); Depression or hopelessness; Previous attempts or psychiatric care; EtOH/substance use (excessive); Rational thinking loss; Social supports lacking; Organized suicidal plan or serious attempt; No spouse; Sickness

Assessment

Depression	Schizophrenia
≥5 sx (≥1 must be ↓ mood or interest) for 2+ wks. MSIGECAPS: Mood low Sleep increased or decreased Interest decreased Guilt or worthlessness Energy decreased Concentration decreased Appetite increased or decreased Psychomotor retardation / agitation Suicidal ideation	≥2 sx (of which ≥1 must be a, b or c) each present for >1 month; 6 months altered behaviour required to make dx a) Hallucinations b) Delusions (bizarre or non-bizarre) c) Disorganized speech (i.e. frequent derailment or incoherence) d) Grossly disorganized/catatonic behaviour e) Negative symptoms (i.e. flat affect, avolition, alogia)
Generalized Anxiety	Mania
≥3 of 6 sx majority of days for 6+ months. BE SKIM: Blank mind/difficulty concentrating Easily fatigued Sleep disturbance Keyed up/on-edge/restless Irritability Muscle tension	≥3 sx with euphoria; ≥4 sx with irritable mood for 1+ wk. GST PAID: Grandiosity Sleep (decreased need for) Talkative/pressured speech Pleasurable activities with painful consequences Activity ↑ (goal-directed)/agitation Ideas (flight of) Distractable

Investigations

Labs: CBC, lytes, B12, Vit D, TSH, BUN, Cr, LFTs, serum EtOH & Tylenol level, urine tox

Safety Assessment
1. Do you have any thoughts that life isn't worth living? 2. Do you have a plan to take your life? 3. Imminence/Mean: When do you plan to complete this plan? Do you have the means available to complete this plan? 4. Protective Factors What prevents you from carrying out this plan? 5. Future Orientation: What are your plans after you leave hospital? 6. Hopefulness: Do you think you can get better? Are you improving? 7. Assess Previous Attempts: organization/lethality, no remorse after, attempt isolated location, affairs in order (will, belongings given away)

Management

Depression	1 st line: SSRIs, SNRIs, NDRIs, CBT, Behavioural Activation
Anxiety	1 st line: SSRIs, SNRIs, CBT, Pregabalin (GAD),
Schizophrenia	1 st line: atypical antipsychotics (Paliperidone, Aripiprazole, Olanzapine, Risperidone, Quetiapine)
Mania	1 st line: mood stabilizers (Lithium, Valproic Acid), atypical antipsychotics (Quetiapine, Aripiprazole)

[EMOttawa Blog Post](#)

Thyroid Emergencies

Types of Thyroid Emergencies

HYPERthyroidism	HYPOthyroidism
Graves' disease Toxic multinodular goiter Toxic adenoma Thyroiditis (autoimmune, postpartum, amiodarone, infectious, traumatic)	Hashimoto's thyroiditis Drug-induced (lithium, amiodarone) Insufficient dietary iodine Central hypothyroidism (hypothalamic or pituitary dysfunction, brain injury, mass)

Thyroid Storm: Severe thyrotoxicosis. Pyrexia (40-41°C), tachycardia, altered mental status, cardiovascular collapse, hypotension, abdominal pain, and signs of hyperthyroidism (lid lag, goiter, ophthalmopathy, tremor)l **Burch and Wartofsky Score** can help distinguish between true Thyroid Storm vs Thyrotoxicosis

Myxedema Coma: Life-threatening event precipitated by stress in untreated hypothyroidism. Hypothermia (<36 °C), altered mental status, hypotension, hypoventilation, bradycardia, acute precipitating illness, and signs of hypothyroidism (dry skin/hair, reduced reflexes, bradykinesia, facial edema).

Assessment

History: constitutional symptoms, thyroid disease, recent acute illness, altered mental status, skin and hair changes, palpitations or bradycardia, abdominal pain, dyspnea, cold or heat intolerance, medications, recent surgery

Physical Exam: vitals, cardiac exam, ophthalmologic exam, neck exam for thyroid enlargement, dysphagia, dysphonia

Investigations:

Labs: can help identify underlying trigger: CBC, TSH, free T3 & T4, ECG, glucose, VBG, troponin, b-hCG (pregnancy can trigger thyrotoxicosis), septic workup (common trigger for myxedema coma)

Tests: ECG; POCUS: assess cardiac fn, rule out pericardial effusion; consider CT head if signs of trauma or focal neuro deficits

TSH	Free T ₄	Free T ₃	Disease
Low	High	High	Hyperthyroidism
Low	Normal	Normal	Subclinical hyperthyroidism
Low	High	Normal	Thyroiditis
Low	Low	Low	Central hypothyroidism
High	Low	Low	Hypothyroidism
High	Normal	Normal	Subclinical hypothyroidism

Management

Thyroid Storm
β-adrenergic blockade: Propranolol 60-80mg PO or 1mg IV, Esmolol infusion (if CHF) Inhibit thyroid synthesis: PTU 1g PO or Methimazole 30mg PO Block thyroid release: SSKI 1-2 drops PO TID, Lugol's 5-7 drops PO TID (1hr after PTU/Methimazole) Inhibit T₄ to T₃ conversion: Hydrocortisone 300 mg IV Supportive: volume resuscitation, cooling measures, anxiolytics, consider bile acid sequestrants, treat underlying precipitant Consult: ICU and Endo
Myxedema Coma
Resuscitation: fluid resuscitation, broad spectrum antibiotics, airway support Thyroid Hormone Replacement: T ₄ 400 mcg IV and T ₃ 20 mcg IV Steroids: Hydrocortisone 100 mg IV Supportive: passive rewarming

Pain Management

Reflex Responses to Pain: ↑ HR, RR and BP, ↑ metabolic rate and O₂ consumption, ↓ urinary and gastric tract tone (urinary and bowel retention)

General Approach: patient-centred, target specific pain syndromes, and use non-pharmacological and pharmacological approaches

Non-Opioid Analgesics

Acetaminophen	
First line foundational analgesia Combine with NSAIDs to improve efficacy	Tylenol 975mg PO
NSAIDs	
Ibuprofen and Naproxen: best safety profile and least side effects Ketorolac: helpful in acute painful condition Topical NSAID preparations: added analgesia for acute MSK and joint pain If prescribing NSAIDs, consider concomitant PPI therapy in patients with higher risk of GI bleeds	Ibuprofen 400 - 800mg PO Naproxen 500mg PO Ketorolac 10 - 30mg IM/IV
Ketamine	
Sub-dissociative doses are effective for analgesia Administer over 20 minutes to minimize risk of emergence reaction	Ketamine 0.1 - 0.3mg/kg IV Over 20 mins
Regional and Local Nerve Blocks	
Useful in lacerations, acute fractures requiring reduction or operation, and headaches (i.e. occipital neuralgia)	Lidocaine + Epi Max 7mg/kg Lidocaine - Epi Max 5mg/kg Bupivacaine Max 2.5-3mg/kg

Opioid Analgesics

Morphine	
Pros: less abuse potential, palliative care pain, cancer pain, dosing range well known Cons: active metabolites may accumulate in renal insufficiency	PO: 0.5mg/kg IV/SC: 0.1mg/kg
Hydromorphone	
Pros: easier titration, more equipotent, better tolerated in renal insufficiency Cons: initial dosing range unclear, more side effects	PO: 1-2mg IV/SC: 0.5-1mg
Fentanyl	
Pros: most effective for acute pain (fractures, trauma) Cons: abuse potential, overdose potential, long ½ life	IV/SC: 25-50µg

Gabapentinoids

Pregabalin & Gabapentin	
Ideal for neuropathic pain	Pregabalin: 50mg PO TID upto 300mg/day Gabapentin: 300mg PO TID upto 2400mg/day

Key References: Rosen's Emergency Medicine: Concepts and Clinical Practice - 8th ed, 2014; Chapter 3. 2017 Canadian Guideline for Opioids for Chronic Non-Cancer Pain. Source: <http://nationalpaincentre.mcmaster.ca/guidelines.html>

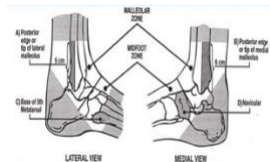
Clinical Decision Rules

Canadian CT Head Rule for Minor Head Injury

Inclusion Criteria	Exclusion Criteria
Head injury resulting in witnessed LOC/disorientation or definite amnesia; initial ED GCS >13; injury within 24hrs	Minimal head injury, obvious penetrating skull injury, acute neurological deficits, unstable vital signs assoc. with major trauma, seizure prior to ED assessment, bleeding disorder, pregnant
High Risk Criteria (neurological intervention)	
GCS <15 at 2 hrs after injury, suspected open or depressed skull fracture, signs of basal skull fracture, vomiting >2 episodes, age >65	
Medium Risk Criteria (for brain injury on CT)	
Amnesia before impact >30 mins, dangerous mechanism	

Ottawa Ankle Rules

Inclusion Criteria
Adult patient (ALSO been validated in pediatrics), any mechanism of blunt ankle injury
Exclusion Criteria
Age <18, pregnant, isolated skin injury, injury >10 days, reassessment of same injury



Ankle X-ray only required if
Bony tenderness at A OR B OR inability to take 4 complete steps in ED
Foot XR only required if
Bony tenderness at C OR D OR inability to take 4 complete steps in ED

Ottawa Knee Rules

Inclusion Criteria	Exclusion Criteria
Adult patient, blunt knee injury, "knee" = patella, head/neck of fibula, proximal 8cm of tibia and distal 8cm of femur	Age <18, pregnant, isolated skin injury, injury older than 7 days, return for reassessment, AMS, paraplegic, multi-trauma
Knee X-ray only required if	
Age >55 OR isolated patellar tenderness OR fibular head tenderness OR inability to flex 90° OR inability to take 4 complete steps in ED	

Ottawa SAH Rule

Inclusion Criteria	Exclusion Criteria
Alert patients >15, new severe atraumatic headache, max intensity within 1 hr	New neurological deficits, prior aneurysm, prior SAH, known brain tumors, chronic recurrent headaches (>3 headaches of same character/intensity for >6 months)
CT is indicated if any criteria are present	
Neck pain/stiffness, witnessed LOC, age >40, onset during exertion, thunderclap headache, limited neck flexion on examination	

Risk Stratification Scales

Canadian Syncope Risk Score

Inclusion Criteria		Exclusion Criteria	
Age >16, present to ED with syncope within 24 hours		Prolonged (>5 min) LOC, AMS, witnessed seizure, major trauma, intoxication, language barrier, head trauma	
Clinical Evaluation		Investigations	ED Diagnosis
-1 Vasovagal predisposition +1 Hx heart disease +2 sBP<90 or sBP>180		+2 Elevated Tnl +1 QRS axis <-30° or >100° +1 QRS >130ms +2 Corrected QT>480ms	-2 Vasovagal syncope +2 Cardiac syncope
Interpretation		Total score = -3 to 11 Score of 0 = 1.9% risk of serious adverse event within 30d Score of 11 = 83.6% risk of serious adverse event within 30d	

Ottawa Heart Failure Risk Scale

Inclusion Criteria		Exclusion Criteria	
Age >50, symptoms consistent with CHF (acute SOB, fluid retention, underlying cardiac abnormality) and/or response to diuretics		O ₂ <85%, HR>120, sBP<90, confusion, ischemic chest pain, acute STEMI on ECG, prognosis of weeks (due to chronic disease), arrival from LTC	
Initial Assessment		Investigations	Walk Test
+1 Hx of stroke or TIA +2 Hx of intubation for respiratory distress +2 HR >110 on ED arrival +1 SaO ₂ < 90% on EMS or ED arrival		+2 New ischemic changes on ECG +1 BUN>12mmol/L +2 HCO ₃ >35mmol/L +2 Elevated Tnl +1 ProBNP>5µg/L	+1 SaO ₂ <90%, HR>110 during 3-min walk test, or too ill to walk
Interpretation		Total score = 0 to 15 Score of 0 = 2.8% risk of serious adverse event within 14d Score of 9 = 89% risk of serious adverse event within 14d	

Ottawa TIA Risk Score

Inclusion Criteria		Exclusion Criteria	
Age >18, ED diagnosis of TIA		Confirmed stroke, decreased LOC, presentation >7 days following onset of most recent TIA	
Clinical Findings		Investigations	
+2 First TIA (in lifetime) +2 Symptoms >10 min +2 History of carotid stenosis +3 Already on antiplatelet therapy +1 History of gait disturbance +1 History of unilateral weakness -3 History of vertigo +3 Initial triage diastolic BP >110mmHg +1 Dysarthria or aphasia (history of examination)		+2 Afib on ECG +1 New or old infarction on CT +2 Platelet count >400 +3 Glucose >15	
Interpretation		Total score = -3 to 14 Score of 0 = 0.04% risk of stroke within 7d Score of 14 = 27.6% risk of stroke within 7d	

Advanced Cardiac Life Support

Electrical Cardioversion

Indications
Paroxysmal SVT Atrial fibrillation/Atrial flutter Ventricular Tachycardia
Pre-medication
Midazolam 1-5mg +/- fentanyl 50-200µg Propofol 50-150mg IV Ketamine 0.25-1.5mg/kg IV Etomidate 20mg IV
Synchronized Cardioversion
pSVT/Aflutter: 150J biphasic or 300J monophasic Vtach/Afib: 200J biphasic or 360J monophasic

Atrial Fibrillation or Atrial Flutter

General
Assess ABCs, monitors, O2, IV access, ECG to confirm Afib
1. Rapid AF/AFL secondary to medical causes?
Treat underlying causes (eg sepsis, bleeding, PE, CHF, ACS, alcohol withdrawal)
2. Unstable from primary AF/AFL? (rare except WPW)?
Synchronized cardioversion
3. Stable?
a) Rhythm Control: Cardioversion or Procainamide 15 mg/kg IV over 60 min: safe if anticoagulated 3 weeks OR no valvular disease/prior TIA and <12hr or <48hr if CHADS-65 <2
b) Rate Control: target <100: Diltiazem 0.25mg/kg IV or Metoprolol 5 m; second line: Digoxin 0.25-0.5mg IV

Ventricular Fibrillation/Pulseless Ventricular Tachycardia

General
Intubate, ventilation, early IV/IO access to administer medications Treat reversible causes: hypovolemia, hypoxia, acidosis, hyper/hypokalemia, hypothermia, toxins, ischemia
Shock-CPR-shock cycles
1. Shock first (200J biphasic or 360J monophasic) If defibrillator not immediately available start CPR, then shock ASAP 2. High quality CPR for 2 min Push hard (2-2.4 inches) and fast (100-120/min), complete chest recoil, minimize interruptions, avoid excessive ventilations (10/min), change compressors q2min, monitor end-tidal CO ₂ 3. Shock
Drugs provided during CPR
Epinephrine: 1mg IV q3-5min Amiodarone: 300mg IV bolus (preferred), 150mg IV (2 nd dose) Lidocaine for refractory VF: 1.5mg/kg IV q3-5min (max 3mg/kg) Magnesium sulfate for polymorphic VT: 2g IV

Key Reference: 2021 CAEP AF/AFL Best Practice Checklist.

Advanced Cardiac Life Support

Wide Complex Tachycardia (85-95% = VT)

General	
Assess ABCs if stable, monitors, O ₂ , vitals, IV access, ECG, CXR	
Unstable	Chest pain, SOB, LOC, low BP, CHF, AMI
Prepare for synchronized cardioversion (200J biphasic or 360J monophasic) Consider premedication	
Stable	Consider cardioversion (as meds only revert VT 30% time)
Procainamide: 20-50mg/min (max 17mg/kg) Amiodarone: 150mg over 10 mins (repeat x2 PRN) Magnesium sulfate for polymorphic VT: 2g IV *Avoid multiple antidysrhythmics sequentially (to prevent proarrhythmic effects). If one fails, go to electrical cardioversion.	

Paroxysmal Supraventricular Tachycardia (AVnRT, AVRT)

Unstable	Chest pain, SOB, LOC, low BP, CHF, AMI
Synchronized cardioversion (150J biphasic or 300J monophasic) Consider premedication	
Stable	
Vagal Manoeuvres Adenosine: 6mg IV over 3 secs (1 st dose), 12mg IV (2 nd dose) Diltiazem: 20mg IV over 2 min (1 st dose), 25mg IV (2 nd dose)	

Pulseless Electrical Activity or Asystole

General	
Intubate, ventilation, early IV/IO access to administer medications, PoCUS	
Management	
1. Ongoing CPR 2. Treat reversible causes: 5Hs (Hypovolemia, Hypoxia, Hydrogen acidosis, Hyper/hypokalemia, Hypothermia) and 5Ts (Toxins, Tamponade, Tension pneumothorax, Thrombosis - coronary, Thrombosis - pulmonary) 3. Epinephrine 1mg IV q3-5mins	

Bradycardia (HR <60)

General	
ABCs, monitors, O ₂ , vitals, IV access, ECG	
Identify and treat reversible causes	
i.e. O ₂ for hypoxia, Ca for hyperkalemia, reperfusion for STEMI	
Unstable from bradyarrhythmia	
Atropine 0.5mg q3-5min (max 3mg) Not effective for 3 ^o heart block Transcutaneous pacing → Transvenous pacing Consider infusions: Dopamine 2-10µg/kg/min OR Epinephrine 2-10µg/min	
Stable	
1 ^o AV block or Type I 2 ^o AV block: Observe Type II 2 ^o AV block or 3 ^o AV block: transcutaneous pacing → transvenous pacing	

Point of Care Ultrasound

Definitions

Hyperechoic: object is more echogenic (brighter) than surrounding tissue

Hypoechoic: object is less echogenic (less bright) than surrounding tissue

Isoechoic: object has same echogenicity than surrounding tissue

Anechoic: object has absence of echoes within it

Near field: area closer to probe

Far field: area farther from probe

Recommended Probe Selection

	Abdo	Cardiac	Lung	Gyne	Soft Tissue	MSK
Curvilinear	+	-	+	+	-	-
Phased Array	+	+	+	-	-	-
Linear	-	-	+	-	+	+
Intracavitary	-	-	-	+	-	-

Ultrasound Artifacts

Acoustic shadowing: shadow distal to reflective surface (i.e. bone, gallstone)

Acoustic enhancement: posterior enhancement due to transmission through a fluid filled structure

Edge artifact: refraction of U/S waves due to two different propagation speeds

Reverberation artifact: sound bouncing between highly reflective surfaces and probe (i.e. metal needle)

Cardiac

Clinical questions: Is there a pericardial effusion? Is there cardiac activity? Is the LV function reduced? Is there RV strain?

Exam:

Parasternal Long Axis: assess LV function
Fractional shortening <30% indicates reduced LV function Assessed by the difference in LV diameter between end diastole and systole.
E-point septal separation >7mm indicates reduced LV function Assessed by looking at the septal slap between anterior mitral valve leaflet and septum.
Fractional shortening ~100% or EPSS ~0mm indicates hyperdynamic LV Can be seen when ventricles are “kissing” or empty
Parasternal Short Axis: assess LV function, some indication of RV function
Fractional shortening <30% indicates reduced LV function RV larger than LV or “D-shaped” LV may indicate RV pressure overload
Apical 4 Chamber View: assess RV function
RV = LV or RV > LV suggests right heart strain
Subxiphoid View: assess pericardial effusion
Anechoic area first appearing between pericardium and RV, can expand to encompass all 4 chambers

AAA

Clinical questions: Is there an abdominal aneurysm?

Exam: Transverse view using curvilinear probe, max AP diameter >3cm may indicate AAA. Does not necessarily provide information of whether it is ruptured or not.

Point of Care Ultrasound

eFAST

Clinical questions: Is there evidence of pneumothorax or free fluid in the abdomen?

Exam: Curvilinear probe to look at RUQ, LUQ, and suprapubic areas. Free fluid will appear anechoic, collects early near the caudal edge of liver (RUQ).

Right and left thorax views using cardiac or curvilinear probe. Absence of lung sliding in pneumothorax.

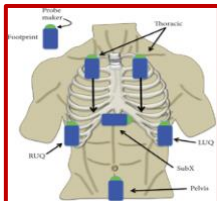
Positive LUQ



Positive RUQ



eFAST Views



Lung

Clinical questions: Is there pneumothorax? Is there hemothorax or pleural effusion?

Exam: Curvilinear probe along mid axillary line at the level of diaphragm
Mirror sign - interface between liquid and air-filled thorax will create a highly reflective surface for sound. Absent mirror sign in fluid-filled thorax (i.e. in hemothorax or pleural effusion).

Spine sign - visualized when anechoic or hypoechoic fluid is present in the pleural space.

Lung sliding - shimmering appearance of pleura, "ants marching on a log". Absent lung sliding may indicate pneumothorax.

Spine Sign



First Trimester

Clinical questions: Is there an intrauterine pregnancy?

Exam: Curvilinear probe along midline sagittal view

Diagnosis of IUP on POCUS
Confirm bladder-uterine juxtaposition to identify the uterus
Gestational sac within the uterus Circular anechoic intrauterine area surrounded by thickened echogenic rim
Yolk sac OR fetal pole visualized within the gestational sac Yolk sac is circular structure with hypoechoic centre within the gestational sac Fetal pole is a small mass at the margin of yolk sac present between 5-6 weeks
Myometrial mantle >8mm Uterine tissue surrounding gestational sac

[EMOttawa Blog POCUS Manual](#)

Key References: Boyd, Jeremy S., et al. "EMERGENCY ULTRASOUND." *The Atlas of Emergency Medicine*, 4e Eds. Kevin J. Knoop, et al. New York, NY: McGraw-Hill.

Approach to the Chest X-Ray

Step 1: Confirm details: patient name, date of birth, MRN, date/time image was taken, previous imaging available (for comparison)

Step 2: Assess image quality using the mnemonic **RIFE**

Rotation: medial aspect of each clavicle should be equidistant from spinous processes & spinous processes vertically oriented against vertebral bodies

Inspiration: 8-9 posterior and 5-6 anterior ribs both lung apices, costophrenic angles and lateral rib edges bilaterally

Projection: note if the film is AP or PA (more common). AP films magnifies heart and widens mediastinum

Exposure: spinous processes should be visible posterior to the heart

Step 3: Assess the CXR for pathology using the **ABCD** approach

Airway	Trachea: deviation (suggests tension pneumo) Carina: NG tube should bisect carina if correctly placed Bronchi: right bronchus is wider, shorter, more vertical Hilar structures: major pulmonary vasculature & major bronchi, left hilum usually superior to right, assess for hilar masses (hilar asymmetry), lymphadenopathy (LNs should not be visible in healthy pts)
Breathing/ Bones	Lungs: ensure lung markings are present from hilum to chest border (absence indicates pneumothorax), pulmonary edema (peribronchial cuffing, Kerley B lines, septal lines), increased airspace opacification (consolidation/malignant lesion) Pleura: if visible indicates pleural thickening (mesothelioma), increased opacity when fluid (hydrothorax), blood (hemothorax) or air and fluid (hydropneumothorax) accumulates in pleural space which appears as area of opacification Ribs: fractures
Cardiac	Heart size: cardiomegaly (PA cardiothoracic ratio >0.5) due to valvular heart disease, cardiomyopathy, pulmonary HTN or pericardial effusion Heart borders: well defined, silhouette sign suggests pneumonia, RA makes up majority of right & LV majority of left heart borders Mediastinum: widening can indicate aortic dissection
Diaphragm	Right hemidiaphragm: usually higher than left Silhouette sign: indicates pneumonia Costophrenic angles: blunting suggests pleural effusion or consolidation Flattening: of diaphragm in hyperinflation (COPD/asthma) Free air: indicates pneumoperitoneum and suggests bowel perforation, best seen under right hemidiaphragm Gastric bubble: best seen under left diaphragm, don't confuse with pneumoperitoneum

Approach to the ECG

HEARTS: a systematic approach to ECG interpretation, which puts ST/T in context

Step 1: Heart rate/rhythm - how is heart beating? Rhythm strip: rate (300 divided by number large boxes), regularity, QRS width, and whether QRS is driven by sinus rhythm (P wave upright in II, biphasic in V1)

Tachycardia (>100)	Narrow Complex: <i>Regular:</i> sinus (P wave), SVT (no P wave), AFL (flutter wave) <i>Irregular:</i> PAC (p wave), AF (no P wave), MAT (different P wave) Wide Complex: <i>Regular:</i> sinus p wave), VT >> SVT with aberrancy (no p wave) <i>Irregular:</i> AF with aberrancy, AF with WPW, polymorphic VT
Bradycardia (<60)	Narrow Complex: <i>Regular:</i> sinus (p wave +/- 1° AV block), junctional (no p wave) <i>Irregular:</i> 2° AV block (dropped beat preceded by PR prolongation in Mobitz 1 vs fixed PR in Mobitz 2) Wide Complex: <i>Regular:</i> 3° AV block (p wave dissociated), idioventricular (no p wave)

Step 2: Electrical conduction - how is conduction flowing? Rhythm strip: look at the three intervals

PR Interval	Represents AV nodal conduction <i>Normal:</i> 120-200ms (3-5 small boxes) <i>Short:</i> accessory pathway (WPW) if delta wave <i>Long:</i> AV delay, including 1° (fixed long) and 2° Type 1 (prolonged PR until dropped beat)
QRS Interval	Represents ventricular conduction <i>Narrow:</i> <120ms (three small boxes) <i>Wide:</i> ventricular rhythm, or atrial rhythm with aberrant conduction (i.e. WPW, BBB, hyperkalemia, sodium channel blockade)
QT Interval	Represents total ventricular depolarization/repolarization time <i>Normal:</i> <1/2 RR interval, correct for rate: $QTc = QT / \sqrt{RR}$ <i>Prolonged:</i> >500ms risk Torsades (polymorphic VT with long QT)

Step 3: Axis - what is the main direction of depolarization in the frontal plane? Limb leads: look at direction of QRS in lead I, aVF, +/- II

	Represents left/right myocardium and conduction <i>Normal:</i> positive I, positive aVF (going left/down) <i>Right:</i> negative I, positive aVF (going right/down) DDx: RV strain, lateral Q, left post. fascicular block, lead misplaced <i>Left:</i> positive I, negative aVF and II (going left/up) DDx: LVH, inferior Q, left ant. fascicular block, lead misplaced
--	--

Step 4: R-wave progression: main direction of depolarization in horizontal plane? Chest leads: look at R/S ratio as it progresses from V1 to V6

	Represents anterior/posterior myocardium and conduction <i>Normal:</i> R<S in V1, R>S by V3-4 <i>Early:</i> R>S in V1 or V2 • DDx: RBBB, WPW, RVH, posterior MI <i>Late:</i> no R>S by V4 • DDx: anterior MI, LVH, left anterior fascicular block
--	---

Step 5: Tall/small voltages - what is overall amplitude of depolarization? Limb and chest leads: look at overall QRS amplitude

Represents viable myocardium and barrier between ECG leads and heart

Tall: normal in young/healthy

- DDx LVH ($R > 11$ in aVL, S in V1-2 + R in V5-6 > 35), RVH ($R > S$ in V1, right axis)

Small: all limb leads $< 5\text{mm}$ or all chest leads $< 10\text{mm}$

- DDx: breast/adipose tissue, COPD, pericardial effusion, cardiomyopathy

Step 6: ST/T changes - any repolarization changes, secondary vs primary?

Look at all leads, interpret ST/T changes in context of preceding QRS (abnormal depolarization will produce secondary repolarization abnormalities).

Consider regional/reciprocal leads:

- **Limbs Leads:** inferior lead III reciprocal to high lateral lead aVL; apex lead II reciprocal to aVR
- **Chest Leads:** anterior leads (V3-4) reciprocal to posterior leads (V8-9); septal leads V1-2 reciprocal to lateral leads (V5-6)

ST Elevation (STE)	Normal: isoelectric, except for 1 mm STE in V2-3 ST elevation secondary to abnormal depolarization: • LBBB/LVH with anterior deep S followed by ST elevation • LV aneurysm: old anterior Q and persisting ST elevation Primary ST elevation: • STEMI: regional STE, hyperacute T, reciprocal change • Pericarditis: diffuse concave STE, II>III, reciprocal aVR • Brugada Syndrome: coved or saddleback STE V1-2 Normal variant: • Early repolarization: STE proportional to large voltage
ST Depression (STD)	ST depression secondary to abnormal depolarization • Tachy-arrhythmia with diffuse ST depression • RBBB/RVH with anterior tall R followed by STD • LBBB/LVH with lateral tall R followed by STD Primary ST depression • Acute coronary occlusion: STD reciprocal to STE (i.e. posterior MI), deWinter T wave (STD + hyperacute T) • Subendocardial ischemia: diffuse ischemic STD with reciprocal STE in aVR • Hypokalemia: diffuse STD with, flat T, large U wave
Tall T Wave	Normal: asymmetric, small relative to QRS, concordant to QRS (upright except aVR/V1, sometimes aVL/III) Tall T wave secondary to abnormal depolarization • LVH/LBBB with deep anterior S followed by tall T wave Primary tall T wave • Hyperacute (acute coronary occlusion): symmetric, wide base, large relative to QRS, regional distribution +/- reciprocation • Peaked (hyperkalemia): symmetric, narrow base, sharp peak, diffuse distribution Normal variant • Early repolarization: tall asymmetric T proportional to large voltage
T Wave Inversion (TWI)	Secondary T wave inversion • BBB: RBBB anterior TWI, LBBB lateral TWI • Hypertrophy: RVH anterior TWI, LVH lateral TWI Primary T wave inversion • PE: anterior/inferior TWI • Coronary occlusion: TWI reciprocal to hyperacute T wave, or from refractory occlusion • Coronary reperfusion (Wellens): resolved chest pain with reperfusion TWI • Stress cardiomyopathy: diffuse TWI with long QT

Key References: Life in the Fast Lane. ECG Library Basics. 2020 <https://litfl.com/ecg-library/>
ECG Cases Blog: emergencymedicinecases.com/blogs/ecg-cases.

End