Stethopedia

CLINICAL REASONING

Approach to…

20 presentations · one flowchart each

Differential, workup and management on a single page. Every number comes from current guidelines and primary studies, with PubMed-verified citations.

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Approach to Chest Pain

CARDIOVASCULAR

1.7%

KEY DECISION RULE
HEART score 0–3[5]
6-wk MACE incl. revasc at 0–3; 16.6% at 4–6, 50.1% at 7–10

Acute chest pain or an anginal equivalent (dyspnea, jaw, arm, epigastric) → 12-lead ECG within 10 min vs prior; BP both arms; hs-troponin 0 and 1–3 h; CXR[1,2,12] → **Shock, STEMI or a bedside red flag?**

Yes →

Emergency: cath lab, decompress, echo or CTA[2,6,11]

- New STE ≥1 mm ×2 leads (V2–3: ≥1.5–2.5 mm)[4]
- Posterior STEMI; Sgarbossa+ LBBB/paced[4,12]
- Hypotension, hypoxemia, VT/VF
- Tearing pain + pulse deficit/BP Δ >20 mmHg[6,12]
- Absent breath sounds, tracheal shift, ↓BP
- ↑JVP, muffled sounds, pulsus >10 mmHg[12]

→ **Cath lab: STEMI, unstable ACS; decompress; echo; CTA/TEE aorta**

If not: which system?

	Myocardial ischemia ~10% in ED	Aortic & pericardial	Pulmonary	GI & chest wall
CAUSES	• **STEMI** • **NSTEMI, unstable angina** • Angina, AS, HCM, spasm, Takotsubo	• **Acute aortic syndrome** • **Cardiac tamponade** • Pericarditis, myopericarditis	• **Pulmonary embolism** • **Tension pneumothorax** • Pneumonia, pleuritis	• **Esophageal rupture** • GERD, spasm, PUD, biliary, pancreatitis • Costochondritis, zoster, panic
CLUES	• Radiates to both arms (LR 2.6)[3] • Prior abnormal stress test (LR 3.1)[3] • ST depression on ECG (LR 5.3)[3]	• Abrupt severe or tearing pain to back[6] • Sharp, pleuritic, better sitting forward[10] • Friction rub; diffuse STE, PR depression[10]	• Pleuritic pain, dyspnea, hypoxemia[8] • Leg swelling; cancer, surgery, immobile[8] • Fever, cough, crackles, dullness	• Focal tenderness reproduces the pain[3] • Burning, meal-related, epigastric • Pain after retching; neck crepitus
WORKUP	• hs-Tn >99th %ile + rise/fall at 0, 1–3 h[2,4] • HEART score if not ruled in or out[5] • CCTA or stress test if intermediate[1]	• Out if ADD-RS ≤1 + D-dimer <500 µg/L[7] • Otherwise CT angiography of the aorta[6] • Echo: effusion, tamponade, AR[6,10]	• Wells + PERC, then D-dimer or CTPA[8,9] • CXR: pneumothorax, consolidation • Bedside echo: RV strain[8]	• Only once ACS is reasonably excluded[1] • Lipase, LFTs; RUQ ultrasound if biliary • CT with oral contrast if rupture suspected
MANAGE	• ASA 162–325 mg chewed; DAPT, heparin[2] • STEMI <12 h: PCI ≤120 min, else lytics[2,11] • NSTE-ACS: cath <24 h if GRACE >140[2]	• AAS: IV β-blocker; HR 60–80, SBP <120 mmHg[6] • Type A: emergency surgery; no lytics[6] • Pericarditis: NSAID + colchicine[10]	• PE: anticoagulate; lyse if shock[8] • Tension PTX: needle, then chest tube[12] • Pneumonia: antibiotics by severity	• GERD: PPI trial • Chest wall: NSAID, reassurance • Rupture: NPO, antibiotics, surgery

Stethopedia
stethopedia.ca/approach/chest-pain

Evidence [1] Gulati 2021, Circulation [2] Rao 2025, Circulation [3] Fanaroff 2015, JAMA [4] Thygesen 2018, J Am Coll Cardiol [5] Backus 2013, Int J Cardiol [6] Isselbacher 2022, Circulation [7] Nazerian 2018, Circulation [8] Creager 2026, Circulation [9] Kline 2008, J Thromb Haemost [10] Schulz-Menger 2025, Eur Heart J [11] Wong 2019, Can J Cardiol [12] Manual 2024–25, pp. 13–14, 32, 43

Approach to Syncope

CARDIOVASCULAR

97.8% KEY DECISION RULE
Canadian Syncope Risk Score[3,4]
sensitivity for 30-day serious outcomes at a score of −1 or more

Transient loss of consciousness with full spontaneous recovery → Eyewitness history, orthostatic vitals, 12-lead ECG[1,2] → **Any high-risk feature?**

Yes → **High risk: admit for telemetry and echo**[1,2]

- Exertional or supine; no prodrome
- Palpitations or chest pain beforehand
- Structural heart disease or heart failure
- Abnormal ECG: block, long QT, Brugada, WPW
- SBP <90, HR <40 awake, or GI bleeding
- Family history of young sudden death

If not: which mechanism?

	Reflex ~60%	**Orthostatic** ~15%	**Arrhythmia** cardiac ~15%	**Structural** cardiac	**Mimics** not syncope
CAUSES	• Vasovagal • Situational: cough, micturition • Carotid sinus hypersensitivity	• Volume loss or bleeding • Drugs: diuretics, vasodilators • Autonomic failure: PD, diabetes	• **VT/VF: scar, long QT, Brugada** • **High-grade AV block** • SVT, WPW, sinus node disease	• **Aortic stenosis, HCM** • **Pulmonary embolism** • **MI, tamponade, dissection**	• Seizure • Hypoglycemia, intoxication • TIA, psychogenic
CLUES	• Trigger: standing, pain, emotion • Prodrome: warmth, nausea, sweating • Normal heart and ECG	• Within 3 min of standing • Older; new or changed drugs	• Abrupt, no prodrome; supine • Palpitations; family sudden death • First event at ≥35 y (LR 3.3)[5]	• Syncope on exertion; murmur • Chest pain, dyspnea, hypoxemia	• Tongue bite, postictal state[6] • Focal deficit; LOC >5 min
WORKUP	• History alone if typical[1] • Tilt test if recurrent, unclear[2]	• SBP ↓≥20 or DBP ↓≥10 in 3 min[7] • CBC, creatinine; drug review	• Telemetry if high risk • Holter, patch or loop recorder[1] • EP study in selected cases[2]	• Echo if heart disease suspected[1] • Troponin; CTPA if PE likely[8]	• Glucose; EEG if seizure likely • CT or MRI if focal signs
MANAGE	• Explain; avoid triggers • Counter-pressure manoeuvres[9] • Salt and fluids if no hypertension[1]	• Stop culprit drugs; give volume • Compression; rise slowly • Midodrine or fludrocortisone[1]	• Pacemaker for AV block[1] • ICD or ablation for VT[1] • Stop QT-prolonging drugs; fix K, Mg	• Treat cause: AVR, anticoagulation, PCI • Avoid vasodilators in AS or HCM	• Treat cause; neurology referral

Stethopedia
stethopedia.ca/approach/syncope

Evidence [1] Shen 2017, J Am Coll Cardiol [2] Brignole 2018, Eur Heart J [3] Thiruganasambandamoorthy 2016, CMAJ [4] Thiruganasambandamoorthy 2020, JAMA IM [5] Albassam 2019, JAMA [6] Sheldon 2002, J Am Coll Cardiol [7] Freeman 2011, Clin Auton Res [8] Costantino 2018, JAMA IM [9] van Dijk 2006, J Am Coll Cardiol [10] Manual 2024–25, p. 34

Approach to Palpitations

CARDIOVASCULAR

43%

KEY DECISION RULE
Modified Valsalva (REVERT)[8]
of SVTs converted to sinus rhythm, vs 17% with standard Valsalva

Awareness of a fast, forceful or irregular heartbeat → 12-lead ECG in symptoms: QRS width, regularity; delta wave, long QT, Brugada[1,10] → K, Mg, CBC, TSH, glucose; review drugs, caffeine, stimulants[3,10] → **Arrhythmia causing instability?**

Yes →

Unstable: synchronized cardioversion now[9,10]

- Hypotension or signs of shock
- Acutely altered mental status
- Ischemic chest pain
- Acute heart failure, pulmonary edema
- Sinus tach: treat cause, no shock

→ **Pads, sedation; pulseless or polymorphic VT: defibrillate**

If stable: what does the ECG show?

	Narrow, regular SVT	**Narrow, irregular** mostly AF	**Wide QRS (≥120 ms)** treat as VT	**Sinus or ectopy** find the cause
CAUSES	• AVNRT, orthodromic AVRT • Atrial flutter 2:1 (rate ~150) • Focal atrial tachycardia	• Atrial fibrillation • Flutter with variable block • MAT (COPD), frequent PACs	• **VT; less often SVT with aberrancy** • **Pre-excited AF (WPW)** • **Torsades: long QT, low K or Mg**	• PACs, PVCs • **Sinus tach: bleed, PE, sepsis, thyroid[10]** • Psychiatric, e.g. panic (31% overall)[6]
CLUES	• Abrupt onset and offset[1] • Neck pounding: AVNRT (LR 177)[5] • Delta wave on baseline ECG: WPW[1]	• Irregularly irregular; no P waves • Alcohol, thyroid, OSA, infection[3]	• Prior MI or low EF: VT likely[4] • AV dissociation, fusion beats[7] • Syncope; FHx of sudden death[4]	• Skipped beat, then a thump • Episodes <5 min: LR 0.38 for arrhythmia[5] • Syncope, exertion, FHx SCD: high risk[4]
WORKUP	• Find P waves; compare with baseline • Adenosine unmasks flutter, AT[1]	• TSH, creatinine, CBC; echo[3] • CHADS-65 for stroke risk[3]	• If any doubt, it is VT[4] • Echo, ischemia workup; QTc[4] • Rule out high K, TCA, Na blockers[10]	• Holter, patch or loop by frequency[5] • Echo: murmur, syncope, frequent PVCs[4]
MANAGE	• Modified Valsalva, legs raised[8] • Adenosine 6 mg rapid push, then 12 mg[1] • Fails: IV β-blocker or diltiazem; ablation[1]	• β-blocker; diltiazem if EF >40%[2,3] • OAC if CHADS-65 positive; DOAC first[3] • <12 h, no stroke/TIA: cardiovert, no OAC[3]	• Stable VT: procainamide; HFrEF: amio[4,10] • Pre-excited AF: procainamide; no nodal/amio[1,2] • Torsades: IV Mg; stop QT drugs[4]	• Sinus tach: treat cause, not rate[1,10] • Symptomatic PVCs: β-blocker[4] • Reassure once rhythm–symptom match

Stethopedia
stethopedia.ca/approach/palpitations

Evidence [1] Page 2016, J Am Coll Cardiol [2] Joglar 2024, Circulation [3] Andrade 2020, Can J Cardiol [4] Al-Khatib 2018, Circulation [5] Thavendiranathan 2009, JAMA [6] Weber 1996, Am J Med [7] Brugada 1991, Circulation [8] Appelboam 2015, Lancet [9] Wigginton 2025, Circulation [10] Manual 2024–25, pp. 4, 8–10

Approach to Dyspnea

RESPIRATORY

LR 0.11 — KEY DECISION RULE
BNP <100 ng/L (pg/mL)[2]
for HF in ED dyspnea; NT-proBNP uses its own, higher cutoffs

Shortness of breath, new or acutely worse

→ Look yourself: RR, SpO2, work of breathing, speech, mentation; ABCs, monitor, IV[12]

→ ECG, chest X-ray, blood gas, BNP, hs-troponin; lung and cardiac POCUS[2,5,12]

→ **Respiratory distress?**

Yes →

Distress: call for help; secure the airway[12]

- Stridor, angioedema: IM epi[10]
- Can't speak; quiet chest
- Drowsy, tiring, or rising PaCO2
- Hypoxemia despite high-flow O2
- Tension PTX signs: decompress
- Shock: PE, tamponade[3,12]

→ **O2 to SpO2 ≤96% (88–92% if CO2 retainer); intubate early[7,8]**

If not: where is the problem?

	Heart & vessels	Airways	Lung & pleura	Outside the chest
CAUSES	• **Acute heart failure, ACS** • **Pulmonary embolism** • Arrhythmia, valve disease, tamponade	• **Anaphylaxis, angioedema, foreign body** • Asthma or COPD exacerbation • Mucus plug; vocal cord dysfunction	• Pneumonia, aspiration • **Tension pneumothorax** • Effusion, ARDS, ILD, hemorrhage	• **Metabolic acidosis: DKA, sepsis** • Anemia; toxins: CO, salicylate, opioids • GBS, MG, ALS; obesity; anxiety
CLUES	• Prior HF (LR 5.8); PND (LR 2.6)[2] • S3 (LR 11); CXR congestion (LR 12)[2] • PE: sudden, pleuritic; clear CXR; DVT[3]	• Stridor, urticaria after an exposure • Wheeze, prolonged expiration • A-lines + sliding, no DVT: asthma/COPD[5]	• Fever, cough, focal crackles • Unilateral ↓ breath sounds • Hypoxemia worse on exertion: ILD[12]	• Clear lungs; SpO2 normal (falsely in CO) • Kussmaul breathing; low HCO3 • Weak cough, ↓FVC; PaCO2 rises late[12]
WORKUP	• ECG, hs-troponin, (NT-pro)BNP; echo[1] • Diffuse B-lines: edema (sens 97%)[5] • PE: Wells, then D-dimer or CTPA[3]	• Peak flow or FEV1 if able • Blood gas if drowsy or tiring[12] • Spirometry once stable	• CXR; POCUS: pneumonia sens 89%[5] • No sliding + lung point: PTX[5] • Cultures; tap a new effusion[12]	• VBG, COHb, lactate, glucose, CBC, salicylate • Normal A-a, ↑PaCO2: hypoventilation[12] • Bedside FVC or NIF if weak[12]
MANAGE	• Edema: CPAP, IV diuretic; nitrate if SBP OK[1,6] • PE: anticoagulate; lyse if shock[3] • ACS: chew ASA; STEMI: cath lab[4]	• Anaphylaxis: IM epi 0.5 mg, thigh[10] • Nebs, steroids; asthma: IV Mg[9,12] • COPD, pH ≤7.35 + ↑PaCO2: bilevel NIV[6,9]	• Tension PTX: needle, then chest tube[12] • Pneumonia: cultures, antibiotics • Hypoxemic: HFNC rather than NIV[11]	• Opioid: naloxone; treat DKA, sepsis[12] • Weak: serial FVC; ICU early[12] • Anxiety only after the rest excluded[12]

Stethopedia
stethopedia.ca/approach/dyspnea

Evidence [1] Heidenreich 2022, Circulation [2] Wang 2005, JAMA [3] Creager 2026, Circulation [4] Rao 2025, Circulation [5] Lichtenstein 2008, Chest [6] Rochwerg 2017, Eur Respir J [7] Siemieniuk 2018, BMJ [8] O'Driscoll 2017, Thorax (BTS) [9] Wedzicha 2017, Eur Respir J [10] Cardona 2020, World Allergy Organ J [11] Oczkowski 2022, Eur Respir J [12] Manual 2024–25, pp. 22, 43–44, 51

Approach to Abdominal Pain

ABDOMINAL

99% — KEY DECISION RULE
Alvarado score, cut-point 5[3]
sensitivity for appendicitis; a score below 5 helps rule it out

Acute abdominal pain in an adult → β-hCG if could be pregnant; vitals; abdominal, pelvic or genital and rectal exam[2] → CBC, lytes, creatinine, LFTs, lipase, lactate, UA; ECG if upper pain[2] → **Red flags of an acute abdomen?**

Yes →

Acute abdomen: resuscitate, call surgery/gyne[1,2]

- Hypotension, tachycardia or sepsis
- Rigidity, guarding or rebound
- Pain out of proportion to the exam[11]
- Bilious vomiting, obstipation[12]
- Hematemesis or hematochezia
- β-hCG + with shock or peritonism[8]

→ Sepsis: fluids, antibiotics; AAA: permissive hypotension, OR[10]

If not: where is the pain?

	Upper abdomen RUQ/epigastric	Lower abdomen, pelvis RLQ, LLQ	Diffuse often surgical	Image-negative outside gut
CAUSES	• **Acute cholangitis** • Biliary colic, cholecystitis • Pancreatitis, peptic ulcer	• **Ectopic pregnancy** • **Ovarian or testicular torsion** • Appendicitis, diverticulitis, stone, PID	• **Ruptured AAA or aortic dissection** • **Mesenteric ischemia** • **Perforation, obstruction, volvulus**	• **Inferior MI** • **DKA, adrenal crisis** • Hypercalcemia, pneumonia, zoster, IBS
CLUES	• Fever, jaundice; full triad often absent[5] • Murphy sign weak: LR 2.8 (CI 0.8–8.6)[6] • Epigastric pain boring to the back	• Pain migrating to RLQ; fever • Vaginal bleeding; missed period[8] • Sudden one-sided pain with vomiting	• Older smoker; back pain, pulsatile mass[10] • Pain out of proportion; AF[11] • Colicky pain, prior surgery or hernia[12]	• Cardiac risk with upper abdominal pain • Carnett sign: abdominal wall pain[2] • Normal imaging; recurrent pain
WORKUP	• RUQ US first; MRCP if CBD stone possible[4] • Lipase >3× ULN + typical pain[7] • CT only if pancreatitis unclear[7]	• β-hCG +: transvaginal ultrasound[8] • CT with IV contrast; noncontrast if stone[1,2] • Doppler US if torsion; US first if pregnant[9]	• CTA if ischemia or aortic cause[10,11] • Bedside ultrasound for AAA if unstable • CT with IV contrast, not plain films[1,12]	• ECG ± troponin[2] • Glucose, calcium, gas, cortisol • CXR if cough or hypoxemia
MANAGE	• Cholangitis: antibiotics, ERCP[2] • Cholecystitis: early cholecystectomy[2] • Pancreatitis: moderate fluids, early PO[7]	• No IUP: PUL; serial β-hCG, gynecology[8] • Torsion: urgent surgical detorsion • Surgery; antibiotics if uncomplicated[9]	• Rupture: door-to-repair <90 min[10] • Ischemia: heparin, revascularize[11] • SBO: NG tube, fluids; OR if strangulated[12]	• Treat cause: ACS, DKA, hypercalcemia • Adrenal crisis: IV hydrocortisone 100 mg[2] • Functional: TCA or SNRI, not opioids[2]

Stethopedia
stethopedia.ca/approach/abdominal-pain

Evidence [1] Scheirey 2018, J Am Coll Radiol [2] Manual 2024–25, pp. 69, 82, 85, 182 [3] Ohle 2011, BMC Med [4] Russo 2023, J Am Coll Radiol [5] Kiriyama 2018, J Hepatobil Pancreat Sci [6] Trowbridge 2003, JAMA [7] Tenner 2024, Am J Gastroenterol [8] Po 2021, J Obstet Gynaecol Can [9] Di Saverio 2020, World J Emerg Surg [10] Chaikof 2018, J Vasc Surg [11] Bala 2022, World J Emerg Surg [12] Ten Broek 2018, World J Emerg Surg

Approach to GI Bleeding

ABDOMINAL

98.6% KEY DECISION RULE
Glasgow-Blatchford score ≤1[1,5]
sensitivity for intervention or death; score ≤1 suits outpatient care

Hematemesis, coffee-ground emesis, melena or hematochezia

→ Two large-bore IVs; shock index, rectal exam; CBC, INR, urea, creatinine, crossmatch[12]

→ **Unstable, cirrhosis or antithrombotic?**

Yes →

Unstable: resuscitate, ICU, then EGD or CTA[1,3,12]

- Shock (SI >1 or SBP <90): RBC now[3,12]
- Hb <70 g/L (<80 if CV disease): transfuse[1,2,12]
- Large hematemesis or ↓LOC: intubate[7,12]
- Cirrhosis: octreotide, ceftriaxone, EGD ≤12 h[7]
- Anticoagulant: hold; warfarin: PCC, not FFP[9]
- Cardiac ASA: keep; no vit K, no TXA[9,10]

→ **Hematochezia + shock: EGD if upper possible, else CTA**[3,12]

If stable: where is the source?

	Upper, nonvariceal most UGIB	**Variceal** cirrhosis	**Lower GI** colon	**Small bowel** EGD, colo –
CAUSES	• Peptic ulcer: NSAIDs, H. pylori • Esophagitis, gastritis, Mallory-Weiss • **Aortoenteric fistula (aortic graft)**	• **Esophageal varices** • **Gastric varices** • Portal hypertensive gastropathy	• Diverticular bleed (30–65%)[12] • Hemorrhoids, angioectasia, colitis • Colorectal cancer; post-polypectomy	• Angioectasia; Heyde (with AS) • Crohn, NSAID ulcers, tumour • Meckel diverticulum if young
CLUES	• Melena on exam (LR 25)[4] • Urea:Cr >0.12 (BUN:Cr >30): LR 7.5[4] • NSAIDs or aspirin; epigastric pain	• Known cirrhosis or heavy alcohol use • Ascites, spider angiomas, low platelets	• Painless hematochezia; older adult • Clots in stool: not upper (LR 0.05)[4] • Hematochezia + shock: 10–15% upper[12]	• Normal EGD and colonoscopy[11] • Iron deficiency; recurrent melena
WORKUP	• Blatchford ≤1: outpatient EGD[1,5] • Others: EGD within 24 h[1,2] • Aortic graft: CTA, vascular surgery[12]	• EGD within 12 h of presentation[7] • INR, bilirubin, albumin: Child-Pugh	• Oakland ≤8: outpatient colonoscopy[3,8] • Admitted: colonoscopy after prep[3] • Brisk bleed: CTA, not urgent colo[3]	• Second-look EGD or colonoscopy[11] • Capsule endoscopy first line[11] • CT enterography if stricture risk[11]
MANAGE	• Pre-EGD: erythromycin ± IV PPI[1,12] • High-risk ulcer: scope, IV PPI ×3 d[1,2] • PPI BID ×2 wk; H. pylori, retest if neg[1,2]	• Octreotide 2–5 d; ceftriaxone 1 g/d[7] • Band ligation; avoid over-transfusing[6,7] • TIPS ≤72 h: Child C10–13 or B+active[7]	• Most stop on their own • Endoscopic clips or coagulation[3] • CTA positive: embolize; surgery last[3]	• Deep enteroscopy to treat lesion[11] • Iron; octreotide for angioectasia[12]

Stethopedia
stethopedia.ca/approach/gi-bleeding

Evidence [1] Laine 2021, Am J Gastroenterol [2] Barkun 2019, Ann Intern Med [3] Sengupta 2023, Am J Gastroenterol [4] Srygley 2012, JAMA [5] Stanley 2017, BMJ [6] Villanueva 2013, N Engl J Med [7] de Franchis 2022, J Hepatol [8] Oakland 2017, Lancet Gastroenterol [9] Abraham 2022, Am J Gastroenterol [10] HALT-IT 2020, Lancet [11] Gerson 2015, Am J Gastroenterol [12] Manual 2024–25, pp. 67–68

Approach to Abnormal Liver Tests

ABDOMINAL

29–33

KEY DECISION RULE

Healthy ALT upper limit (ACG)[1]

U/L in men and 19–25 U/L in women; assess any ALT above this

Abnormal ALT, AST, ALP or bilirubin on routine or acute testing

→ Repeat tests; history: alcohol, drugs, supplements, metabolic and viral risk[1,2]

→ R = (ALT/ULN) ÷ (ALP/ULN); bilirubin, INR, albumin, platelets[1,12]

→ **Liver failure, cholangitis or overdose?**

Yes →

Emergency: admit; hepatology, GI or toxicology

- ALF: INR ≥1.5 + confusion, no cirrhosis[3,12]
- Possible acetaminophen excess[5]
- Fever, RUQ pain, jaundice[11]
- Drug + ALT >3×, bilirubin >2× ULN (Hy's law)[2]
- Shock, hypoglycemia, ↑lactate[3]
- Pregnancy: HELLP, acute fatty liver[12]

→ **NAC if acetaminophen risk or early ALF; ALF: ICU, transplant[3,4,5]**

If not: which pattern?

	Hepatocellular, mild R >5, <5× ULN	**Hepatocellular, marked** R >5, ≥5× ULN	**Cholestatic or mixed** R <5	**Isolated bilirubin** enzymes normal
CAUSES	• MASLD; alcohol-related disease • Drugs, herbal supplements[2] • HBV, HCV, iron overload, Wilson, autoimmune	• **Acetaminophen toxicity** • **Ischemia, HSV, Budd-Chiari, Wilson** • Acute hepatitis A, B, E; drugs; autoimmune	• **Cholangitis** • Stone; pancreatic or biliary cancer • PBC, PSC, drugs, infiltration	• Gilbert syndrome • Hemolysis; hematoma resorption • Direct: sepsis, TPN, drugs; Dubin-Johnson[1]
CLUES	• Obesity, diabetes, dyslipidemia[7] • AST:ALT ≥2:1 suggests alcohol[1,12] • AST ≫ ALT, high CK: muscle, not liver[12]	• Overdose, or repeated excess + alcohol[5] • ALT:LDH <1.5: ischemia, acetaminophen[6] • HSV: fever, anicteric; pregnant, immunosupp.[3,12]	• Fever, RUQ pain, jaundice: Charcot[11] • Painless jaundice, weight loss: cancer • Itch, middle-aged woman: PBC; IBD: PSC[1,10]	• Indirect; rises with fasting, illness[1] • Anemia, high LDH, low haptoglobin
WORKUP	• HBsAg, anti-HBc, HCV Ab; iron studies[1] • ANA, anti-smooth muscle, IgG; ceruloplasmin[1] • Ultrasound; FIB-4 ≥1.3 → elastography[7]	• Acetaminophen level even if no history[5] • HAV IgM, HBsAg, anti-HBc IgM, HSV PCR[3] • Doppler ultrasound; trend INR, glucose[3]	• High GGT confirms liver source of ALP[1] • Ultrasound: dilated ducts → MRCP/ERCP[1] • Normal ducts: AMA (PBC); MRCP (PSC)[1,10]	• Fractionate: direct vs indirect[1] • CBC, smear, reticulocytes, haptoglobin • Direct: ultrasound, as for cholestasis[1]
MANAGE	• Stop alcohol and culprit drugs[2] • MASLD: weight loss, treat metabolic risk[7] • HBV, HCV: refer for antivirals[8,9]	• NAC now if acetaminophen possible[5] • Stop the drug; autoimmune: steroids[2,12] • Suspect HSV: empiric IV acyclovir[3]	• Cholangitis: antibiotics, then drainage[11] • Stones: ERCP, then cholecystectomy[12] • PBC: ursodiol 13–15 mg/kg/d[10]	• Gilbert: reassure; no further tests[1] • Hemolysis: find and treat the cause

Stethopedia
stethopedia.ca/approach/abnormal-liver-tests

Evidence [1] Kwo 2017, Am J Gastroenterol [2] Chalasani 2021, Am J Gastroenterol [3] Shingina 2023, Am J Gastroenterol [4] Lee 2009, Gastroenterology [5] Dart 2023, JAMA Netw Open [6] Cassidy 1994, J Clin Gastroenterol [7] Rinella 2023, Hepatology [8] Osiowy 2025, Can Liver J [9] Bhattacharya 2023, Clin Infect Dis [10] Lindor 2019, Hepatology [11] Miura 2018, J Hepatobiliary Pancreat Sci [12] Manual 2024–25, pp. 84–87

Approach to Diarrhea

ABDOMINAL

≤1%

KEY DECISION RULE
Fecal calprotectin ≤40 µg/g[9]
probability of IBD in adults with IBS-type symptoms

≥3 loose stools/d: acute <14 d, chronic >4 wk

→ Duration, blood, fever, travel, food, new drugs, antibiotics, immune status; volume status[1,11]

→ Electrolytes, creatinine if more than mild; stool tests only if they change care[1,2]

→ **Severe, bloody or high-risk?**

Yes →

High risk: rehydrate and send stool tests[1,11]

- Hypovolemia: orthostasis, AKI[1,11]
- Fever ≥38.5 °C or >6 stools/d[11]
- Sepsis: blood cultures, antibiotics[1,11]
- Bloody, afebrile: no antibiotics[1,5]
- Shock, ileus, megacolon: fulminant CDI[3]
- Immunosuppressed, ≥70, pregnant[11]

→ **Fulminant: PO vancomycin 500 mg QID + IV metronidazole; surgery[3]**

If not: how long, and which type?

	Acute infectious most; viral	**C. difficile** antibiotic use	**Chronic watery** >4 wk	**Chronic fatty** malabsorptive	**Chronic inflammatory** bloody
CAUSES	• Norovirus; traveller's E. coli • Campylobacter, Salmonella, Shigella • **Shiga toxin E. coli O157 (HUS)[1,5]**	• Antibiotics within 3 mo[3] • Hospital stay, age ≥65, PPI[3,11] • **Fulminant colitis, megacolon**	• IBS-D, microscopic colitis • Drugs, bile acid; lactose, sorbitol • Hyperthyroidism, neuroendocrine tumour	• Celiac disease • Pancreatic insufficiency • Bacterial overgrowth, short bowel	• **Colorectal cancer** • Crohn disease, ulcerative colitis • Ischemic, radiation, CMV colitis
CLUES	• Large, watery: small bowel[11] • Blood, tenesmus, fever: colonic[11] • Travel, food, sick contacts	• ≥3 unformed stools in 24 h[3] • Fever, ↑WBC, abdominal pain	• Osmotic gap >125 mOsm/kg: osmotic[11] • Gap <50, persists fasting: secretory[11]	• Greasy, foul, floating stools • Weight loss; low iron or B12	• Onset >50 y, blood, nocturnal[8,11] • Weight loss, anemia, fever
WORKUP	• None if mild and not high-risk[1,2] • Stool culture or PCR if severe[1] • Shiga toxin if bloody[1]	• Test only if not on laxatives[3] • Severe: WBC ≥15 ×10⁹/L[3] • Severe: Cr >133 µmol/L (1.5 mg/dL)[3]	• CBC, TSH, tTG, Giardia[8,10] • Calprotectin ≤40 µg/g: IBD unlikely[9] • Colonoscopy + biopsy if alarm[8,11]	• tTG-IgA with total IgA[8,10] • Fecal elastase[8]	• Stool culture, C. diff; calprotectin, CRP[9,11] • Colonoscopy with biopsies[8]
MANAGE	• Oral rehydration[1,2] • Loperamide if no fever or blood[1,2] • Azithromycin: febrile dysentery, travel[1]	• Stop antibiotics; fidaxomicin 200 mg BID[3,4,6] • Or PO vancomycin 125 mg QID; each ×10 d[3,4] • Recurrent: fidaxomicin; FMT after 2nd[3,4,7]	• Stop culprit drugs and sugars • Budesonide: microscopic colitis[8] • Bile acid binder for bile acid[8]	• Duodenal biopsy, then gluten-free diet[8] • Pancreatic enzymes with meals[11]	• GI referral; treat IBD • Cancer: staging and surgery

Stethopedia
stethopedia.ca/approach/diarrhea

Evidence [1] Shane 2017, Clin Infect Dis [2] Riddle 2016, Am J Gastroenterol [3] McDonald 2018, Clin Infect Dis [4] Johnson 2021, Clin Infect Dis [5] Wong 2000, N Engl J Med [6] Louie 2011, N Engl J Med [7] van Nood 2013, N Engl J Med [8] Arasaradnam 2018, Gut [9] Menees 2015, Am J Gastroenterol [10] Smalley 2019, Gastroenterology [11] Manual 2024–25, pp. 74, 121

Approach to Acute Kidney Injury

RENAL & VOLUME STATUS

89%

KEY DECISION RULE
FEUrea ≤35% (pre-renal AKI)[3,12]
sensitivity on diuretics (24/27); later study: specificity 33%

Creatinine rising or urine output falling → KDIGO: Cr ↑≥26.5 µmol/L (0.3 mg/dL) in 48 h, ≥1.5× in 7 d, or UOP <0.5 mL/kg/h ×6 h; stage 1–3[1] → K, VBG, ECG; volume; hold nephrotoxins; bladder scan; UA, sediment; urine Na, Cr, urea[1,11] → **Any renal emergency?**

Yes →

Renal emergency: treat now; call nephrology[2,11]

- ECG change, K ≥6.0 mmol/L, or rising fast
- Refractory pulmonary edema
- pH <7.1 despite bicarbonate
- Uremia: pericarditis, confusion
- Lithium or toxic alcohol
- RPGN: hemoptysis, RBC casts[10,11]

→ **ECG change: Ca gluconate; K ≥6.5: shift K; dialyze if refractory**[2,8,11]

If not: where is the injury?

	Pre-renal commonest	**Tubulointerstitial** intrinsic	**Glomerular & vascular** intrinsic	**Post-renal** quick to fix
CAUSES	• Volume loss: GI, bleeding, diuretics • Low EABV: heart failure, cirrhosis, sepsis • NSAIDs, ACEi/ARB	• ATN: ischemia, sepsis, contrast • AIN: β-lactams, PPIs, NSAIDs • **Rhabdomyolysis; crystals (TLS)**	• **RPGN: anti-GBM, ANCA vasculitis** • Lupus, IgA, post-infectious GN • **TTP/HUS, scleroderma crisis, atheroemboli**	• **Bladder outlet: BPH, anticholinergics** • Blocked urinary catheter • Bilateral stones, pelvic mass
CLUES	• Orthostasis, dry mucosa; or congestion • Bland sediment, hyaline casts[11]	• Muddy brown casts, tubular cells: ATN[4] • New drug; fever, rash, sterile pyuria • Heme+ dipstick, no RBCs: rhabdo[11]	• RBC casts, dysmorphic RBCs • Hemoptysis, rash, sinusitis • Schistocytes, low platelets, high LDH	• Anuria; palpable bladder • Prostatism, recurrent UTIs[6]
WORKUP	• FENa <1% if off diuretics[3,12] • FEUrea ≤35% on diuretics: weak[3,12]	• Fresh urine microscopy[4] • CK; urate, phosphate, LDH if TLS • Skip urine eosinophils (LR ~1)[5]	• ANCA, anti-GBM, C3/C4, ANA[10] • Smear, LDH, haptoglobin, ADAMTS13 if TMA • Biopsy if it will change treatment[10]	• Bladder scan for retention • Renal US: hydronephrosis[6]
MANAGE	• Dry: balanced crystalloid; hold diuretics[7,11] • HF congestion: diurese; Cr rise OK[11] • Cirrhosis: stop diuretics; albumin challenge[11]	• ATN: supportive; avoid nephrotoxins[1] • AIN: stop drug; steroids per nephrology • Rhabdo: early isotonic fluids; watch K[9]	• RPGN: steroids ± PLEX, nephrology today[10,11] • TTP: hematology, PLEX[11] • Scleroderma crisis: captopril[11]	• Foley; flush or replace blocked catheter • Fever + hydro: urgent decompression[11] • Watch for post-obstructive diuresis

Stethopedia
stethopedia.ca/approach/acute-kidney-injury

Evidence [1] Kellum 2013, Crit Care (KDIGO) [2] James 2013, Am J Kidney Dis (CSN) [3] Carvounis 2002, Kidney Int [4] Perazella 2008, Clin J Am Soc Nephrol [5] Muriithi 2013, Clin J Am Soc Nephrol [6] Licurse 2010, Arch Intern Med [7] Semler 2018, N Engl J Med [8] STARRT-AKI 2020, N Engl J Med [9] Bosch 2009, N Engl J Med [10] Rovin 2021, Kidney Int (KDIGO GN) [11] Manual 2024–25, pp. 94, 96–97, 101, 108, 142 [12] Pépin 2007, Am J Kidney Dis

Approach to Hyponatremia

RENAL & VOLUME STATUS

≤10

KEY DECISION RULE

Limit to avoid osmotic demyelination[2,4]

mmol/L max in 24 h; ≤8 if Na ≤105, low K, EtOH, poor diet, cirrhosis

Serum Na <135 mmol/L, from no symptoms to seizure or coma

→ Osm ≥275 mOsm/kg = not hypotonic: glucose (Na ↓2.4 per 5.6 mmol/L), lipids, paraprotein, glycine[1,8]

→ Send urine Osm/Na, TSH, AM cortisol (never delay 3%); volume status; drugs[1,9]

→ **Severe symptoms, or acute with symptoms?**

Yes →

Severe symptoms: give 3% NaCl bolus now[1,2,5,9]

- Seizure, coma, or somnolence[1,4]
- Vomiting or cardiorespiratory distress[1,4]
- Acute (<48 h) + symptoms: marathon, MDMA[1]
- Symptoms resolve or Na ↑5: stop 3%[1,9]
- Na rise over limit: relower, D5W ± DDAVP[1,9]

→ 3% NaCl 100–150 mL/10–20 min; Na after each; ≤3×; aim ↑4–6[1,2,9]

If not: volume status + urine Osm/Na?

	Hypovolemic UNa ≤30 if GI	**Euvolemic** UNa >30	**Hypervolemic** UNa ≤30	**Low urine Osm** UOsm ≤100
CAUSES	• Vomiting, diarrhea, third-spacing • Thiazides; cerebral salt wasting • **Primary adrenal insufficiency**	• **SIADH: lung, CNS, cancer** • Drugs: SSRIs, carbamazepine; pain • **Low cortisol; severe hypothyroid**	• Heart failure • Cirrhosis • Nephrotic syndrome; advanced CKD	• Primary polydipsia • Low solute: beer, tea and toast
CLUES	• Orthostatic, dry mucosa, low JVP • UNa ≤30: extrarenal loss[1] • UNa >30: diuretic, adrenal, renal[1]	• UOsm >100, UNa >30, no diuretic[1] • FE urate >12% even on diuretics[7] • Na falls after normal saline[9]	• Edema, ascites, raised JVP • UNa ≤30 unless diuretics or CKD[1]	• UOsm ≤100 mOsm/kg[1] • Psychiatric illness; large intake • Beer, poor diet: high demyelination risk[6]
WORKUP	• Urine Na before giving fluids[1] • AM cortisol, K; ACTH stim if low	• TSH, AM cortisol before SIADH label[1] • CXR; CT head if neuro signs	• BNP, echo; liver tests, albumin • Creatinine, urine protein	• Diet and fluid intake history • UOsm ≈ last 2 digits of SG × 30[9]
MANAGE	• Isotonic saline; stop thiazide[1] • Adrenal crisis: IV hydrocortisone 100 mg[9] • UOP >100 mL/h: DDAVP ± D5W[1,9]	• Fluid restrict 1–1.5 L/d; treat cause[9] • Urea 0.25–0.5 g/kg/d, or NaCl + loop[1,3] • Avoid normal saline in SIADH[9]	• Fluid restriction[1] • Loop diuretic if overloaded[1] • Treat the heart, liver or kidney	• Restrict water; add solute slowly[9] • Corrects fast: watch Na and UOP[6,9] • DDAVP clamp if rising too fast[9]

Stethopedia
stethopedia.ca/approach/hyponatremia

Evidence [1] Spasovski 2014, Eur J Endocrinol [2] Verbalis 2013, Am J Med [3] Hoorn 2017, J Am Soc Nephrol [4] Adrogué 2022, JAMA [5] Baek 2021, JAMA Intern Med [6] George 2018, Clin J Am Soc Nephrol [7] Fenske 2008, J Clin Endocrinol Metab [8] Hillier 1999, Am J Med [9] Manual 2024–25, pp. 104, 107, 182

Approach to Hyperkalemia

RENAL & VOLUME STATUS

13% — KEY DECISION RULE
IV insulin for hyperkalemia[5]
of insulin doses in dialysis inpatients caused glucose <3.3 mmol/L

Serum potassium above the upper limit of normal → STAT 12-lead ECG and monitor; creatinine, glucose, gas, CBC; drug review[1,10] → Hemolyzed or unexpected with normal ECG: repeat K on a blood gas before treating[1,10] → **ECG change, K ≥6.0, or rising fast?**

Yes →

Emergency: stabilize, shift, then remove K[1,10]

- ECG: peaked T, long PR, wide QRS, brady
- ECG change: Ca gluconate 10% 30 mL IV[1]
- K ≥6.5: shift K even if ECG normal[1,3]
- K 6.0–6.4: monitor, binder, recheck K[1,10]
- After insulin: K in 1–2 h; glucose ≥3 h[5]

→ **Shift: insulin 5–10 U + dextrose 25–50 g; salbutamol 10–20 mg[1,4,6]**

Then: why is K high?

	Low GFR or distal flow commonest	**Low aldosterone effect** drugs	**Shift out of cells** redistribution	**Pseudohyperkalemia** not real
CAUSES	• **AKI, especially oliguric** • Advanced CKD; missed dialysis • Low distal flow: HF, hypovolemia[9]	• ACEi/ARB, MRA, K-sparing diuretic • NSAIDs, trimethoprim, heparin, CNIs • **Adrenal insufficiency: low Na, low BP**	• **Rhabdomyolysis, tumour lysis** • Insulin deficiency: DKA, HHS • Acidosis; digoxin, β-blockers	• Hemolysis; fist clenching, tourniquet[1] • Very high platelets or WBC[10] • Drawn above a K-containing IV
CLUES	• Low urine output; rising creatinine • Edema, or dry and orthostatic	• New drug or dose, with low eGFR[1,2] • Diabetic CKD, non-gap acidosis: type 4 RTA	• Crush, found down, recent chemo • High glucose; anion-gap acidosis	• Hemolysis flagged; no risk factors[1] • Implausible: no CKD, culprit drug or lysis[1]
WORKUP	• Creatinine, urea; bladder scan • Renal ultrasound if AKI is new	• Medication and supplement review[1] • Renin, aldosterone if unclear • Cortisol, ACTH if adrenal suspected[10]	• CK, urate, phosphate, LDH • Blood gas; digoxin level if on it	• Repeat without tourniquet; gas K[1,10] • Platelets, WBC on the CBC[10]
MANAGE	• Dialysis if oliguric, ESKD or refractory[1] • Making urine: loop diuretic[1] • Volume-depleted: isotonic fluid first[1]	• Hold culprit; restart RASi ± binder later[11] • Binder: SZC 10 g TID ≤48 h; SPS GI risk[1,7,8] • Adrenal crisis: IV hydrocortisone 100 mg[10]	• Treat cause: DKA insulin; digoxin Fab[12] • Bicarbonate only if acidemic[1] • Serial K: lysis keeps releasing it	• No treatment once repeat K is normal

Stethopedia
stethopedia.ca/approach/hyperkalemia

Evidence [1] Lindner 2020, Eur J Emerg Med [2] Clase 2020, Kidney Int [3] Montague 2008, Clin J Am Soc Nephrol [4] Harel 2016, PLoS One [5] Apel 2014, Clin Kidney J [6] Allon 1989, Ann Intern Med [7] Noel 2019, JAMA Intern Med [8] Kosiborod 2014, JAMA [9] Gumz 2015, N Engl J Med [10] Manual 2024–25, pp. 65, 103, 105, 107, 182 [11] KDIGO 2024, Kidney Int (CKD) [12] Chan 2014, Clin Toxicol

Approach to Acid–Base Disorders

RENAL & VOLUME STATUS

+2.5

KEY DECISION RULE

Albumin-corrected anion gap[2,12]

mmol/L added to the AG per 10 g/L albumin below 40 g/L (4 g/dL)

Abnormal pH, HCO3 or PaCO2 on a blood gas or chemistry panel

→ Gas + lytes: pH <7.35 acidemia, >7.45 alkalemia; primary process; VBG pH ~0.03 below ABG[1,3]

→ Winter's PaCO2 = 1.5×HCO3 + 8±2; anion gap = Na − (Cl + HCO3), correct for albumin[1,2,12]

→ **Shock, toxin, acute ↑CO2 or extreme pH?**

Yes →

Emergency: ABCs, ICU, poison centre[1,12]

- pH <7.20 or >7.60: arrhythmia, shock[12]
- Drowsy or acute ↑PaCO2: NIV/intubate[1]
- Shock or sepsis + lactate >2 mmol/L: fluids[7,9]
- Toxic alcohol suspected: fomepizole ± HD[6]
- Salicylate: IV NaHCO3; intubated: keep RR high[4,10,12]
- Salicylate HD: AMS, >7.2 mmol/L (>6.5 if AKI)[10,12]

→ **Acidemia: NaHCO3 only if able to ventilate; recheck gas, K, iCa**

Then: which primary disorder?

	High-AG acidosis AG ↑	Normal-AG acidosis Cl ↑	Metabolic alkalosis HCO3 ↑	Respiratory PaCO2 ↑ or ↓
CAUSES	• **Lactate: shock, ischemia, metformin** • **Ketoacidosis: DKA (incl. SGLT2i), alcohol** • **Toxic alcohols, salicylate; uremia**	• Diarrhea, GI fistula, ileal conduit • Large-volume 0.9% saline • RTA; acetazolamide	• Vomiting, NG suction • Diuretics; low K or Mg • Mineralocorticoid excess	• **High PaCO2: opioids, COPD, weakness** • **Low PaCO2: sepsis, PE, hypoxemia** • Pain, anxiety, pregnancy, cirrhosis
CLUES	• Osm gap >10: toxic alcohol; can be normal[6] • Respiratory alkalosis + tinnitus: salicylate • ΔAG vs ΔHCO3: hidden second process[1]	• Diarrhea or saline load in history • High K, diabetic CKD: type 4 RTA[12]	• Volume-depleted: vomiting, diuretics • Hypertension with low K	• PaCO2 +10: HCO3 +1 acute, +4 chronic[1,12] • PaCO2 −10: HCO3 −2 acute, −4 chronic[1,12]
WORKUP	• Lactate, β-hydroxybutyrate, creatinine • Salicylate, ethanol, measured Osm[6]	• UAG neg: GI or proximal RTA; pos: distal[5,12] • Urine pH, K; Ca/Cr for type of RTA[12]	• Urine Cl: low = saline-responsive[11] • Renin, aldosterone if hypertensive[11]	• ABG: venous PCO2 is unreliable[3] • CXR, drug screen; CTPA if PE likely
MANAGE	• Lactate: restore perfusion; DKA protocol[7] • Fomepizole ± HD for toxic alcohol[6] • NaHCO3 if pH ≤7.20 + AKI 2–3 (not DKA)[8,9]	• Stop saline; balanced crystalloid[9] • Alkali for RTA 1–2; type 4: lower K[12]	• 0.9% saline + KCl if Cl-responsive[12] • Overloaded: acetazolamide[12] • Aldosterone excess: amiloride or MRA[12]	• NIV or ventilate; reverse opioids • No NaHCO3 for respiratory acidosis[12] • Alkalosis: treat hypoxemia, pain, cause

Stethopedia
stethopedia.ca/approach/acid-base

Evidence [1] Berend 2014, N Engl J Med [2] Figge 1998, Crit Care Med [3] Byrne 2014, Respirology [4] Stolbach 2008, Acad Emerg Med [5] Batlle 1988, N Engl J Med [6] Kraut 2018, N Engl J Med [7] Kraut 2014, N Engl J Med [8] Jung 2025, JAMA [9] Prescott 2026, Crit Care Med [10] Juurlink 2015, Ann Emerg Med [11] Emmett 2020, Clin J Am Soc Nephrol [12] Manual 2024–25, pp. 102–103

Approach to Anemia

HEMATOLOGY

<70 g/L

KEY DECISION RULE

Restrictive transfusion (AABB 2023)[1]

Hb trigger, stable inpatients; <80 g/L if CV disease or ortho surgery

Hb <130 g/L (men) or <120 g/L (women), or a fall from baseline

→ Bleeding, diet, drug, alcohol history; CBC with MCV, RDW, other cell lines vs prior; smear[11]

→ Reticulocyte index; draw iron, B12, folate, hemolysis labs and T&S before transfusing[11]

→ **Unstable, bleeding or hemolyzing?**

Yes →

Unstable: resuscitate; urgent GI or hematology[8,11]

- Shock: low BP, tachycardia
- Angina or dyspnea at rest
- Pancytopenia or blasts on smear
- Melena, hematemesis or BRBPR
- Schistocytes, low platelets: TTP?
- Fast Hb fall, jaundice, dark urine

→ **Bleed/shock: transfuse now, not by Hb; MI: consider <100 g/L[9,11]**

If not: reticulocyte index, then MCV?

	Microcytic (RI <2%) MCV <80 fL	**Normocytic (RI <2%)** MCV 80–99 fL	**Macrocytic (RI <2%)** MCV ≥100 fL	**Hemolysis or blood loss** RI >2%
CAUSES	• Iron deficiency: blood loss, malabsorption • **Occult GI cancer causing iron loss** • Inflammation; thalassemia trait; lead	• Inflammation, CKD, endocrine • **Acute bleed before retics rise** • **Marrow failure, myeloma, infiltration**	• **B12 deficiency: pernicious anemia** • Folate lack; alcohol; liver disease • MTX, hydroxyurea, MDS, thyroid	• **Bleeding: GI, retroperitoneal** • **TTP/HUS/DIC; autoimmune hemolysis** • G6PD, sickle cell, spherocytosis, valve
CLUES	• Pica, koilonychia, restless legs[4] • Menorrhagia, melena, NSAIDs; celiac • Mentzer (MCV/RBC) <13: thalassemia[11]	• Anticoagulant, procedure, falling Hb • High RDW: mixed deficiency[11] • Other cytopenias, bone pain, high Ca	• Paresthesias, ataxia, low vibration[6] • Hypersegmented neutrophils[11] • Vegan, gastrectomy, PPI, metformin[6]	• Jaundice, dark urine, splenomegaly[11] • Schistocytes + low platelets: TMA[11] • Spherocytes: AIHA or hereditary[7,11]
WORKUP	• Ferritin <45 µg/L (<100 if inflamed)[2,4] • tTG; men/postmeno: EGD/colonoscopy[2,3] • Fe replete: Hb electrophoresis, lead[11]	• Serial Hb, creatinine, TSH, CRP, iron[11] • SPEP + free light chains if unexplained[11] • Marrow biopsy if cytopenias or blasts[11]	• B12, folate, TSH, LFTs; drug review[11] • Borderline B12: MMA ± homocysteine[6,11] • Low B12: anti-intrinsic factor Ab[11]	• LDH, bilirubin, haptoglobin, DAT, UA[11] • Schistocytes: PLASMIC, ADAMTS13[8,11] • Bleed: endoscopy or CT angiography
MANAGE	• Elemental Fe 50–100 mg daily/QOD[3,5] • IV iron if intolerant, malabsorption[3,11] • Treat source; iron 3 mo past Hb normal[3]	• Treat cause; iron if also deficient[11] • CKD: nephrology for iron ± ESA[11] • Marrow failure: urgent hematology[11]	• B12 1 mg IM if neuro, else 1–2 mg PO/d[6] • Folate 1–5 mg/d once B12 replete[6,11] • MDS suspected: hematology referral	• TTP: PLEX, steroids; avoid platelets[10,11] • Warm AIHA: steroids; call blood bank[7,11] • Bleed: stop source; transfuse by need[11]

Stethopedia
stethopedia.ca/approach/anemia

Evidence [1] Carson 2023, JAMA [2] Ko 2020, Gastroenterology [3] Snook 2021, Gut [4] Camaschella 2015, N Engl J Med [5] Stoffel 2017, Lancet Haematol [6] Stabler 2013, N Engl J Med [7] Brodsky 2019, N Engl J Med [8] Bendapudi 2017, Lancet Haematol [9] Carson 2023, N Engl J Med [10] Zheng 2020, J Thromb Haemost (Tx) [11] Manual 2024–25, pp. 133–134, 136, 141, 144

Approach to Thrombocytopenia

HEMATOLOGY

99.8% — KEY DECISION RULE
4Ts score 0–3 (low probability)[1,2]
NPV for HIT (13 studies, 3068 patients): no PF4 test, keep heparin

Platelets <150 ×10⁹/L, or a fall from baseline → Bleeding history and exam; heparin and drug review; prior counts; smear, citrate tube if clumps[8] → CBC, creatinine, LFTs, PT/aPTT, fibrinogen, HIV, HCV, β-hCG, type and screen[8] → **Bleed, HIT, TMA, DIC, blasts or HELLP?**

Yes →

Emergency: treat now; call hematology[8]

- Major bleed; headache: CT head
- Heparin and fall >50%: suspect HIT
- Schistocytes: TTP until excluded
- Long PT/aPTT, low fibrinogen: DIC
- Blasts or pancytopenia on smear
- Pregnant: HELLP or preeclampsia

→ 4Ts ≥4: non-heparin AC; high PLASMIC: PLEX; major bleed: plt[1,3,8]

If not: which mechanism?

	Low production	Immune destruction	Consumption	Sequestration, dilution
CAUSES	• **Leukemia, MDS, marrow infiltration** • Sepsis; viral: HIV, HCV, EBV, CMV • Alcohol, B12 or folate lack, chemo	• **HIT: any heparin, even flushes** • Drugs: quinine, antibiotics, ASMs • ITP (exclusion); SLE, APS, lymphoma	• **TTP; HUS (Shiga toxin, complement)** • **DIC: sepsis, cancer, trauma, obstetric** • HELLP; ECMO, balloon pump, valves	• Hypersplenism: cirrhosis, portal HTN • Massive transfusion; hypothermia • Gestational thrombocytopenia
CLUES	• Other cytopenias; blasts on smear[8] • Alcohol use, poor diet; macrocytosis • Fever, bone pain, B symptoms	• Heparin d5–10; <24 h if ≤100 d prior[1,8] • New drug; sudden, deep fall[8] • Well; isolated; large platelets[8]	• Schistocytes, high LDH, low haptoglobin[8] • TTP: plt <30 ×10⁹/L, neuro signs, fever[8] • Bloody diarrhea, then AKI: ST-HUS[8]	• Splenomegaly; stigmata of liver disease • Pregnant, well, normal BP; plt ≥100[10]
WORKUP	• B12, folate, HIV, HCV[8] • Marrow biopsy if other lines low[8]	• 4Ts; anti-PF4 only if ≥4[1,2] • ANA, aPL if SLE or APS suspected[8]	• PLASMIC; ADAMTS13 before PLEX[3,4] • DAT, D-dimer; stool Shiga toxin if diarrhea[8]	• Ultrasound: spleen, liver • Pregnant: BP, urine protein, LFTs
MANAGE	• Treat cause; stop marrow toxins • No bleed, chemo: platelets if <10 ×10⁹/L[7] • Marrow disease: urgent hematology[8]	• Stop drug; HIT: argatroban/fonda/DOAC[1,8] • ITP <30: steroids + ritux or TPO-RA[6] • ITP critical bleed: plt, IVIG, steroid, TXA[9]	• TTP: PLEX, steroid, ritux, caplacizumab[5,11] • DIC: fix cause; plt <10, <50 if bleeding[7,12] • TTP: no platelets unless major bleed[8]	• Treat cause; usually observe • Major surgery: platelets if <50 ×10⁹/L[7]

Stethopedia
stethopedia.ca/approach/thrombocytopenia

Evidence [1] Cuker 2018, Blood Adv [2] Cuker 2012, Blood [3] Bendapudi 2017, Lancet Haematol [4] Zheng 2020, J Thromb Haemost (Dx) [5] Zheng 2020, J Thromb Haemost (Tx) [6] Cuker 2026, Blood Adv [7] Metcalf 2025, JAMA [8] Manual 2024–25, pp. 136, 138, 144 [9] Chowdhury 2026, Blood Adv [10] Reese 2018, N Engl J Med [11] Zheng 2025, J Thromb Haemost [12] Levi 2009, Br J Haematol

Approach to Fever of Unknown Origin

INFECTIOUS DISEASE

58% — KEY DECISION RULE
FDG-PET/CT in classic FUO[6]
pooled diagnostic yield (sensitivity 86%, specificity 52%)

Fever ≥38.3 °C on several occasions for ≥3 wk, still undiagnosed

→ Confirm fever; repeat full history (travel, animals, drugs) and exam; stop non-essential drugs[1,2]

→ CBC, smear, CMP, LDH, ESR, CRP, ferritin, 3 BCx, UA, HIV, IGRA, CXR; CT C/A/P, then PET/CT[2,6,12]

→ **Unstable, high-risk host, GCA or HLH?**

Yes →

High risk: culture, treat, admit, consult[2,12]

- Hypotension or sepsis
- Neutropenia: ANC <0.5 ×10⁹/L
- Transplant, biologics, AIDS
- Vision loss, jaw claudication: GCA
- Ferritin >3000 µg/L, cytopenias, spleen: HLH

→ **Cultures first; GCA: prednisone 40–60 mg now; rheum ≤24 h[8,12]**

If stable: which category?

	Infection ~38%	Inflammatory ~21%	Neoplasm ~12%	Other
CAUSES	• **Endocarditis (incl. culture-negative)** • **TB (extrapulmonary), occult abscess** • Malaria; EBV, CMV, HIV; brucella, Q fever	• **Giant cell arteritis, PMR** • Adult-onset Still disease • SLE, vasculitis, sarcoid, IBD	• **Lymphoma** • Leukemia, myeloproliferative, myeloma • Renal cell, liver, colon cancer	• Drug fever; VTE, hematoma • **HLH** • Thyroiditis, periodic fever, factitious
CLUES	• Murmur, emboli; valve, device, IDU[7] • Travel, animals, raw milk, TB birthplace • Night sweats; dental or surgical history	• Age >50: headache, jaw claudication • Daily spikes, salmon rash, arthritis[1] • High ESR/CRP; very high ferritin (Still)	• Drenching sweats, weight loss, nodes • High LDH, cytopenias, splenomegaly	• β-lactam, ASM; rash, eosinophilia uncommon[12] • Ferritin >3000 µg/L, cytopenias, ↑TG[12] • Episodic fever, well between[4]
WORKUP	• 3 culture sets before antibiotics; echo[7] • IGRA; malaria smears; serology[1,12] • Tissue biopsy: histology, AFB culture[12]	• ANA, RF, ANCA, CK, ferritin, SPEP[12] • Temporal + axillary ultrasound first[9] • Temporal biopsy if ≥55 y, no lead[4]	• SPEP, free light chains; smear review[12] • PET/CT unhelpful if ESR, CRP normal[5] • Excisional node, liver or marrow biopsy	• Stop drug: fever settles in ≤1 wk[12] • HScore; sIL-2R, marrow biopsy[10,11] • Leg Doppler; TSH; fever diary
MANAGE	• Treat organism; drain abscess • No empiric antibiotics if stable[1,12]	• GCA: prednisone 40–60 mg now; rheum ≤24 h[8] • Otherwise no empiric steroids[1]	• Tissue before any steroids[1] • Hematology or oncology referral	• HLH: dexamethasone ± etoposide[11] • No diagnosis in 23–51% of FUO[3,4] • Then observe: most resolve[12]

Stethopedia
stethopedia.ca/approach/fever-of-unknown-origin

Evidence [1] Haidar 2022, N Engl J Med [2] Wright 2024, Open Forum Infect Dis [3] Fusco 2019, BMC Infect Dis [4] Bleeker-Rovers 2007, Medicine [5] Bleeker-Rovers 2007, Eur J Nucl Med [6] Takeuchi 2016, J Nucl Med [7] Fowler 2023, Clin Infect Dis [8] Mukhtyar 2026, Ann Rheum Dis [9] Dejaco 2024, Ann Rheum Dis [10] Fardet 2014, Arthritis Rheumatol [11] La Rosée 2019, Blood [12] Manual 2024–25, pp. 129, 134

Approach to Altered Mental Status

NEUROLOGY & MENTAL STATUS

LR 9.6 — KEY DECISION RULE
Confusion Assessment Method[1]
for delirium (any cause); negative CAM has LR 0.16

Acute change from baseline in arousal, attention or thinking

→ ABCs, vitals, glucose; collateral: baseline, last known well, med changes[12]

→ Exam: GCS, attention, pupils, focal signs, meningismus; CAM or 4AT[1,2]

→ **Any emergency feature?**

Yes →

Emergency: treat now, then find the cause[12]

- Glucose <4 mmol/L: dextrose now ± thiamine[3,4,9]
- Airway at risk or GCS ≤8: call ICU
- Focal deficit, unequal pupils: code stroke
- Fever, stiff neck: cultures, dex + antibiotics[6]
- Seizure ≥5 min: benzodiazepine[5]
- Miosis, slow RR: naloxone[12]

→ **Lorazepam 4 mg IV or midazolam IM; naloxone 0.04 mg IV, titrate[5,12]**

If not: which cause category?

	Metabolic	Infection	Drugs & toxins	Primary CNS	Delirium, multifactorial
CAUSES	• **Hypoglycemia, hypoxia, hypercapnia** • **Wernicke (thiamine deficiency)** • Na, Ca, uremia, liver, endocrine	• **Meningitis, encephalitis** • **Sepsis** • Pneumonia; UTI only if GU symptoms[10]	• **Opioid or sedative toxicity** • **Alcohol or benzo withdrawal** • Serotonin syndrome, NMS, anticholinergics	• **Stroke, ICH, herniation** • **Nonconvulsive status epilepticus** • Post-ictal, subdural, PRES	• Age, dementia, sensory loss + insult[12] • Retention, constipation, pain
CLUES	• Asterixis, multifocal myoclonus[12] • Eye palsy, ataxia, EtOH, poor diet[4] • Hypoglycemia can mimic stroke	• Fever, headache, stiff neck, rash • New seizure; immunosuppressed	• Drugs started, stopped or missed[12] • Toxidrome: pupils, skin, HR, temp	• Focal deficit, gaze deviation, pupils • Unexplained coma; subtle twitching[7]	• No single cause once 1–4 excluded[12] • Hypoactive: quiet, sleepy, missed
WORKUP	• Lytes, Ca, creatinine, LFTs, VBG[12] • TSH, ammonia, cortisol if needed	• Blood cultures, CXR, lactate[10] • CT 1st: focal, seizure, GCS <10, immunosupp.[6]	• Med review; tox screen, levels • Acetaminophen, salicylate; ECG (QTc)	• CT ± CTA head and neck[12] • Urgent EEG if coma unexplained[7] • MRI if CT negative or subacute[12]	• Rescreen daily: CAM or 4AT[1,2] • Bladder scan; bowel and pain review[12]
MANAGE	• Thiamine 200 mg IV TID if Wernicke[4] • Low Na + symptoms: 3% NaCl[11] • Lactulose for liver; NIV for CO2	• Meningitis: dex + antibiotics before CT[6] • Acyclovir if encephalitis possible	• Stop deliriogenic drugs[8] • Benzos only for withdrawal, seizure[8]	• Code stroke; bleed: neurosurgery[12] • Status: benzo, then ASM load[5,12] • HOB 30°; reverse anticoagulant[12]	• Reorient, glasses, sleep, walk[8,12] • Antipsychotic only if unsafe[8,12] • Avoid benzos and restraints[8]

Stethopedia
stethopedia.ca/approach/altered-mental-status

Evidence [1] Wong 2010, JAMA [2] Tieges 2021, Age Ageing [3] Lega 2023, Can J Diabetes [4] Galvin 2010, Eur J Neurol [5] Glauser 2016, Epilepsy Curr [6] van de Beek 2016, Clin Microbiol Infect [7] Towne 2000, Neurology [8] Marcantonio 2017, N Engl J Med [9] Schabelman 2012, J Emerg Med [10] Nicolle 2019, Clin Infect Dis [11] Spasovski 2014, Eur J Endocrinol [12] Manual 2024–25, pp. 193, 198–199, 204, 212

Approach to Headache

NEUROLOGY & MENTAL STATUS

100% KEY DECISION RULE
Ottawa SAH Rule[3]
sensitivity if any of 6 (spec 15.3%): nontraumatic, peak ≤1 h, intact

Headache that is new, changed, or unlike the usual pattern

→ Onset, tempo, pattern vs usual; vitals, fundi, neuro exam, temporal arteries[1,12]

→ Pregnant: BP (preeclampsia); red painful eye: glaucoma; housemates ill: CO level[1]

→ **Any red flag (SNNOOP10)?**

Yes →

Red flag: same-day workup; treat the emergency[1,12]

- Sudden: peak ≤1 h, or on exertion
- Fever, stiff neck, rash, confusion
- Focal signs, seizure, papilledema
- New after age 50; jaw claudication
- Cancer, immunosupp., pregnancy
- Positional, Valsalva, post-trauma

→ **Meningitis or GCA: cultures/ESR, then treat before LP or biopsy**[7,11]

Then: primary or secondary cause?

	Primary most common	**Vascular** thunderclap	**Infection, inflammation** fever or >50 y	**Mass or pressure** progressive
CAUSES	• Migraine ± aura • Tension-type; medication overuse • Cluster, other autonomic types	• **Subarachnoid hemorrhage** • **ICH, venous thrombosis, dissection** • **Pituitary apoplexy; also RCVS, PRES**	• **Bacterial meningitis** • **Encephalitis, brain abscess** • **Giant cell arteritis**	• **Tumour, abscess, hydrocephalus** • Idiopathic intracranial HTN • Low pressure: post-LP, CSF leak
CLUES	• POUND 4 of 5: migraine (LR 24)[6] • Band-like, mild, no nausea: tension • Brief orbital pain, tearing: cluster	• Ottawa: ≥40 y, neck pain, witnessed LOC[3] • Exertion, instant peak, ↓neck flexion[3] • Horner: dissection; OCP, puerperium: CVT[12]	• Fever, neck stiffness, confusion[11] • Jaw claudication (LR 4.2), diplopia[8] • Normal ESR: GCA less likely (LR 0.2)[8]	• Worse with Valsalva (LR 2.3)[6] • Worse lying down; morning vomiting[1] • Papilledema, diplopia, ↓LOC[12]
WORKUP	• Migraine/tension, normal exam: no scan[6] • New cluster-type: MRI (LR 10.7)[6,12] • Headache ≥8 d/mo: overuse risk; diary[4]	• Alert, intact, CT ≤6 h negative: no SAH[2] • CT >6 h negative: LP or CTA[2] • CTA head/neck; CT or MR venography[12]	• Blood cultures, then LP[11] • CT 1st: focal, seizure, GCS <10, immunosupp.[11] • ESR, CRP; temporal artery US or biopsy[9]	• Abnormal exam: image (LR 5.3)[6] • MRI with gadolinium[1] • LP opening pressure after imaging
MANAGE	• ED: IV prochlorperazine or GON block[10] • Ketorolac, metoclopramide; no opioids[10] • Acute drugs ≤2 d/wk; prevent if ≥4 bad d/mo[4,12]	• Neurosurgery; reverse anticoag; lower BP[5,12] • aSAH: oral nimodipine; secure aneurysm[5] • CVT: anticoagulate even if bleed[12]	• Antibiotics + dexamethasone ± acyclovir[11] • GCA: prednisone 40–60 mg now; rheum ≤24 h[7] • GCA with vision loss: emergency[7]	• HOB 30–45°, osmotherapy, neurosurgery[12] • Tumour edema: dexamethasone[12]

Stethopedia
stethopedia.ca/approach/headache

Evidence [1] Do 2019, Neurology [2] Godwin 2019, Ann Emerg Med [3] Perry 2013, JAMA [4] Medrea 2024, Can J Neurol Sci [5] Hoh 2023, Stroke [6] Detsky 2006, JAMA [7] Mukhtyar 2026, Ann Rheum Dis [8] Smetana 2002, JAMA [9] Dejaco 2024, Ann Rheum Dis [10] Robblee 2026, Headache [11] van de Beek 2016, Clin Microbiol Infect [12] Manual 2024–25, pp. 195, 197–198

Approach to Weakness

NEUROLOGY & MENTAL STATUS

NNT 2.6

KEY DECISION RULE
Thrombectomy for LVO (HERMES)[4]
to cut 90-day disability by ≥1 mRS level in anterior LVO stroke

Weakness: true loss of power, not fatigue or pain-limited effort

→ Tempo, last known well; UMN vs LMN signs, pattern, sensory level, bulbar signs[11]

→ Glucose, K, Ca, Mg, PO4, CK, ECG; serial VC and NIF if LMN, NMJ or bulbar[5,11]

→ **Stroke, cord or breathing threat?**

Yes →

Emergency: code stroke, spine MRI or ICU[2,10,11]

- Sudden face, arm, speech deficit[1]
- Sensory level, saddle loss, retention
- VC <20 mL/kg or weak NIF[5]
- Bulbar signs, dyspnea, weak cough
- Rapid ascending weakness
- Flaccid; peaked T or U waves

→ **Stroke: glucose, CT + CTA; cord: stat MRI spine; failing VC: ICU[2,10]**

If not: where is the lesion?

	Brain (UMN) sudden, focal	**Spinal cord, cauda equina** level, saddle	**Horn, root and nerve** LMN signs	**Junction and muscle** proximal
CAUSES	• **Ischemic stroke** • **Intracerebral hemorrhage** • Tumour, abscess, MS; Todd paresis	• **Compression: tumour, abscess, bleed** • **Cauda equina syndrome** • Myelitis, infarct, B12/copper, spondylosis	• **Guillain-Barré syndrome** • Polyneuropathy: DM, alcohol, B12 • Radiculopathy, CIDP; ALS (UMN + LMN)	• **Myasthenic crisis, botulism** • **Hypo- or hyperkalemia; rhabdomyolysis** • Myositis; statin or steroid myopathy
CLUES	• Face, arm or speech: any one, LR 5.5[1] • Aphasia, neglect, field cut • Hemiparesis; brisk reflexes, Babinski	• Sensory level or saddle loss; retention[11] • Back pain worse lying down; cancer[10] • Fever, injection drug use: abscess	• Ascending, areflexic after infection[7] • Atrophy, fasciculations, low tone • Radiating pain; distal sensory loss	• Fatigable ptosis, diplopia, dysphagia • Proximal, symmetric; sensation normal • Better with effort, dry mouth: LEMS
WORKUP	• Glucose; CT rules out bleed; CTA for LVO[2] • MRI brain if CT unrevealing	• Stat MRI whole spine with contrast[10] • B12, copper, HIV, syphilis; LP if myelitis	• LP: ↑protein; may be normal in wk 1[7] • NCS/EMG; MRI if radicular; A1c, B12, SPEP[11] • mEGRIS: ventilation risk[6,7]	• AChR, then MuSK Ab; CT chest[8,11] • EMG with repetitive stimulation • CK, K, TSH; review drugs
MANAGE	• ≤4.5 h: TNK 0.25 mg/kg (max 25 mg) or tPA[2,3] • LVO: thrombectomy ≤24 h by imaging[2,12] • BP ≤185/110 pre-lysis; ICH: reverse anticoag[2,11]	• Tumour: dexamethasone; RT ± surgery[10] • Abscess: drainage, IV antibiotics • Neurosurgery or spine surgery now	• Can't walk ≤4 wk: IVIG 0.4 g/kg ×5 d or PLEX[7] • No corticosteroids for GBS[7] • Intubate: VC <20 mL/kg or >30% fall[5]	• MG crisis: IVIG or PLEX, ICU; avoid FQs, AGs[8,11] • High K: Ca gluconate if ECG change; shift K[9] • Low K: replace on monitor; rhabdo: fluids

Stethopedia
stethopedia.ca/approach/weakness

Evidence [1] Goldstein 2005, JAMA [2] Heran 2024, Can J Neurol Sci [3] Menon 2022, Lancet [4] Goyal 2016, Lancet [5] Lawn 2001, Arch Neurol [6] Luijten 2023, JNNP [7] van Doorn 2023, Eur J Neurol [8] Sanders 2016, Neurology [9] Lindner 2020, Eur J Emerg Med [10] Lawton 2019, J Clin Oncol [11] Manual 2024–25, pp. 196–198, 200 [12] Heran 2025, Can J Neurol Sci

Approach to Arthritis

MUSCULOSKELETAL (MSK)

LR 28 — KEY DECISION RULE
Synovial WBC >100 ×10⁹/L (>100,000/µL)[1]
for septic arthritis; <25 ×10⁹/L lowers it (LR 0.32) but can't exclude

Painful, swollen joint(s) → Confirm synovitis; count joints; tap any effusion: cells, Gram stain, culture, crystals[1,10] → **Could this be a septic joint?**

Yes → **Possible septic joint: tap before antibiotics[1,2]**

- Acute hot, swollen joint(s)
- Older age, DM, RA, prosthesis, skin infection
- Fever in only 57%: absence doesn't exclude
- Gram stain positive or WBC >50 ×10⁹/L
- Pustules and tenosynovitis: gonococcal
- Crystals seen but patient looks septic

→ **Joint and blood cultures, IV antibiotics, ortho to drain[10,11]**

Then: synovial WBC and joint pattern?

	Septic usually >50	Crystal 10–100 ×10⁹/L	Inflammatory 2–100 ×10⁹/L	Noninflammatory <2 ×10⁹/L
CAUSES	• **S. aureus > strep > gram-negatives** • **Gonococcal (DGI)** • **Prosthetic joint infection: ortho/ID[10]**	• Gout (monosodium urate) • CPPD (pseudogout)	• Rheumatoid arthritis • SpA: psoriatic, reactive, IBD, axial • Viral (parvovirus, HBV, HCV), Lyme, SLE	• Osteoarthritis • **Fracture, hemarthrosis** • AVN, internal derangement
CLUES	• Mono (knee ~50%); >1 joint in ~20%[10] • PMN ≥90% (LR 3.4)[1] • Lower WBC: DGI, PJI, immunosupp.[10,11,12]	• 1st MTP; peaks within 24 h; tophi[3] • Diuretics, alcohol, CKD, meat, seafood • Older; knee or wrist; chondrocalcinosis	• Morning stiffness >30 min[10] • Symmetric MCP, PIP, wrist, MTP: RA[5] • Dactylitis, psoriasis, uveitis, GU/GI: SpA	• Age >45, bony swelling, stiff <30 min • Trauma or anticoagulant: bloody tap • Locking or giving way: mechanical
WORKUP	• Gram stain, culture; blood cultures ×2 • GC NAAT: urine, throat, rectum • X-ray; CT or MRI if deep joint[10]	• Needles, negatively birefringent: gout • Rhomboids, positively birefringent: CPPD • Culture anyway: 1.5% also septic[2]	• RF, anti-CCP, ESR/CRP; hand/foot X-ray[5] • HLA-B27, SI imaging; GC/chlamydia NAAT[10] • Parvo IgM, HBV, HCV, HIV, Lyme; ANA[10]	• X-ray: joint space loss, osteophytes • MRI if AVN or derangement suspected • Bloody tap: X-ray for fracture; INR, CBC
MANAGE	• Cefazolin + ceftriaxone ± vancomycin (MRSA)[11] • Drain: serial taps or washout[10,11] • 3–4 wk; 2 wk if hand/wrist washout[9,10]	• Colchicine, NSAID or steroid (oral/IA)[4] • Tophi/≥2 flares/y: allopurinol, <360 µmol/L[4] • CPPD, 1–2 joints: aspirate, IA steroid[7]	• NSAID or short prednisone course[10] • Early rheum; methotrexate for RA[6] • Reactive: treat GU infection[10]	• Exercise, weight loss, tai chi[8] • Topical or oral NSAID; IA steroid[8] • Hemarthrosis: hold anticoagulant; ortho

Stethopedia
stethopedia.ca/approach/arthritis

Evidence [1] Margaretten 2007, JAMA [2] Shah 2007, J Emerg Med [3] Neogi 2015, Ann Rheum Dis [4] FitzGerald 2020, Arthritis Care Res [5] Aletaha 2010, Arthritis Rheum [6] Fraenkel 2021, Arthritis Care Res [7] Zhang 2011, Ann Rheum Dis [8] Kolasinski 2020, Arthritis Care Res [9] Gjika 2019, Ann Rheum Dis [10] Manual 2024–25, pp. 169–171 [11] Ravn 2023, J Bone Jt Infect (SANJO) [12] McNally 2021, Bone Joint J (EBJIS)

Approach to Shock

CRITICAL CARE

LR 11

KEY DECISION RULE

Passive leg raise[4]

fluid responsive if cardiac output rises; LR 0.13 if it doesn't

Hypotension or signs of poor tissue perfusion

→ Access, monitor; mentation, UOP, skin, lactate; ECG, CXR, labs, cultures, POCUS[1,10]

→ **Hypoperfusion or MAP <65?**

Yes →

Shock: resuscitate while you find the cause

- Failing airway: intubate, pressors ready[10]
- Hypovolemia: fluid bolus; bleeding: blood[2,9]
- MAP <65: peripheral norepinephrine[2]
- Possible septic shock: antibiotics ≤1 h[2]
- Anaphylaxis: IM epi (1 mg/mL), thigh[8]
- High lactate: repeat to guide resuscitation[2]

→ **Anaphylaxis: epi 0.5 mg IM; rising NE: add vasopressin, then epi**[2,8]

Then: which type (skin, JVP, POCUS)?

	Distributive ~66%	**Hypovolemic** ~16%	**Cardiogenic** ~16%	**Obstructive** ~2%
CAUSES	• **Septic shock** • **Anaphylaxis** • Adrenal crisis, neurogenic, drugs	• **Hemorrhage: GI, trauma, AAA, ectopic** • GI losses: vomiting, diarrhea • Renal losses, burns, pancreatitis	• **Acute MI** • Decompensated HF, myocarditis • Acute MR/AR; arrhythmia	• **High-risk PE** • **Tension pneumothorax** • **Cardiac tamponade**
CLUES	• Warm skin, wide pulse pressure[1,10] • Sepsis + pressors + lactate >2 mmol/L[3] • Exposure; urticaria, wheeze, stridor[8]	• Cold, pale; flat neck veins[1,10] • Small, collapsing IVC on POCUS • Melena, trauma, anticoagulants	• Cold, wet: high JVP, crackles, S3[1] • Chest pain; new murmur • Inferior MI: right-sided ECG for RV infarct	• High JVP, clear lungs (or RV infarct)[1] • No breath sounds; pulsus paradoxus
WORKUP	• Blood cultures before antibiotics[2] • Image for a source needing control[2] • Cortisol, ACTH if adrenal crisis possible[11]	• CBC, INR, fibrinogen, iCa; crossmatch[10] • FAST, rectal exam; CTA if stable[10] • β-hCG if pregnancy possible	• Any MI + shock → emergent cath[10,12] • Echo: LV, RV, valves[6] • Troponin, lactate	• POCUS: effusion, big RV, no sliding[1] • CTPA only if stable enough[7]
MANAGE	• Sepsis: crystalloid ≥30 mL/kg in 3 h[2] • Hydrocortisone 50 mg q6h (sepsis)[2,10] • Adrenal crisis: IV hydrocortisone 100 mg[11]	• Bleed: blood not crystalloid; GI/IR/OR[9,10] • Trauma: SBP 80–90 (no TBI), TXA ≤3 h[9] • Reverse anticoag; losses: LR > NS[10]	• Revascularize MI; cardiovert or pace[6,10] • NE ± dobutamine; avoid dopamine[5,6] • Shock team; mechanical support[6]	• Tension PTX: needle, then chest tube[10] • Tamponade: pericardiocentesis[10] • PE with shock: systemic thrombolysis[7]

Stethopedia
stethopedia.ca/approach/shock

Evidence [1] Vincent 2013, N Engl J Med [2] Prescott 2026, Crit Care Med [3] Singer 2016, JAMA [4] Bentzer 2016, JAMA [5] De Backer 2010, N Engl J Med [6] Sinha 2025, J Am Coll Cardiol [7] Creager 2026, Circulation [8] Cardona 2020, World Allergy Organ J [9] Rossaint 2023, Crit Care [10] Manual 2024–25, pp. 61–64 [11] Beuschlein 2024, J Clin Endocrinol Metab [12] Thiele 2017, N Engl J Med