



CHEST PAIN

The five that kill · exclude them first, then risk-stratify with the right score

ECG IN 10 MINUTES
before anything else

THE FIVE THAT KILL *exclude these before you reach for a differential*

ACS

Pressure, exertional, radiating to jaw or arm, diaphoresis. **ECG within 10 minutes** and serial troponin. **A normal first ECG does not exclude it** — repeat it if the pain continues or changes.

PULMONARY EMBOLISM

Pleuritic pain, dyspnea, tachycardia, hypoxia. **Wells or Geneva first, then D-dimer only if probability is low or intermediate**; CTPA if the probability is high or the D-dimer is positive.

AORTIC DISSECTION

Abrupt, maximal at onset, tearing, radiating to the back. Pulse or blood-pressure differential, new aortic regurgitation, focal deficit. **CT angiography** of the aorta.

TAMPONADE

Beck's triad (hypotension, raised JVP, muffled sounds), **pulsus paradoxus over 10 mmHg**, electrical alternans. **Bedside echo makes the diagnosis**; pericardiocentesis treats it.

TENSION PNEUMOTHORAX

Sudden pain, severe dyspnea, absent breath sounds, tracheal shift, shock. **A clinical diagnosis — do not wait for the film.** Needle decompression, then a chest drain.

THE FIRST TEN MINUTES *the order that catches the time-critical causes*

- 1 ECG within 10 minutes of arrival**, and repeat at 15–30 minutes if pain persists or the picture changes. **Compare with an old ECG if one exists.** Look specifically for **ST elevation, new left bundle branch block, and the posterior MI pattern** (tall R and ST depression in V1–V3, which needs posterior leads V7–V9). **Right-sided leads in inferior MI** before you give any nitrate.
- 2 Vital signs in both arms.** A **systolic differential, a pulse deficit, or a new diastolic murmur** moves aortic dissection to the front of the queue and changes everything downstream, because anticoagulating a dissection is catastrophic.
- 3 High-sensitivity troponin at 0 and 1–2 hours.** Use your laboratory's assay-specific thresholds; **the change between samples matters more than a single absolute value.** Troponin marks myocardial injury, not infarction — PE, sepsis, heart failure, myocarditis, tachyarrhythmia, chronic kidney disease and dissection all raise it.
- 4 Bedside ultrasound and a chest film.** Echo for effusion, right ventricular strain and regional wall-motion abnormality; lung ultrasound for pneumothorax. **Widened mediastinum on the film supports dissection but its absence does not exclude it.**
- 5 Then, and only then, risk-stratify.** Scores are for patients in whom you have already excluded the immediately lethal causes — **they are not a substitute for that exclusion.**

RIGHT SCORE, RIGHT DECISION *each score answers one question only*

Score	Population	Bands	The one decision it answers
HEART	Undifferentiated chest pain in the emergency department	0–3 low (MACE roughly 2%) · 4–6 moderate (roughly 15–20%) · 7–10 high (roughly 50%)	Disposition. Low with negative serial troponins can go home with follow-up. It does not tell you when to take an admitted patient to the catheter laboratory — that is the commonest misuse of this score.
GRACE	Confirmed NSTE-ACS, already admitted	≤ 108 low · 109–139 intermediate · ≥ 140 high	Timing of the invasive strategy. GRACE ≥ 140 is the guideline trigger for an early invasive approach, within 24 hours. Intermediate risk is managed invasively during the admission. This is the score for the cath-timing question, not HEART.
TIMI	Unstable angina or NSTEMI	Seven equally weighted variables; 0–1 low, 6–7 high (14-day event rate roughly 5% rising to over 40%)	Prognosis, and who gains most from an invasive strategy and more intensive antithrombotics. Simpler than GRACE but less discriminating, so where guidelines specify a threshold for invasive timing they use GRACE, not TIMI.
Wells / PERC	Suspected pulmonary embolism	Wells sets pre-test probability; PERC applies only once probability is already low	Whether to test at all. PERC is a rule-out for low-probability patients, not a probability score. D-dimer belongs in low or intermediate probability, never as a screen in high probability — there, image regardless.
ADD-RS	Suspected aortic dissection	One point each for high-risk condition, pain feature, and examination finding	Whether to image now. Two or three features means immediate CT angiography. Zero or one with a negative D-dimer makes dissection unlikely.

The trap: these scores were derived in different populations to answer different questions, and their numbers are not interchangeable. **Applying a HEART band to a cath-timing decision, or a GRACE band to an emergency-department discharge, is the classic error.** Check that the score named actually matches the decision in front of you.

DON'T FORGET THESE

ESOPHAGEAL RUPTURE

Boerhaave: forceful vomiting, then severe pain, then subcutaneous emphysema. Mortality climbs steeply with delay. **CT with oral contrast**; it is a surgical emergency and is missed because it mimics ACS.

THE COMMON, NON-LETHAL CAUSES

Musculoskeletal, reflux, anxiety, herpes zoster (pain can precede the rash by days), pneumonia and pericarditis. **Reproducible tenderness lowers but does not exclude ACS** — a meaningful minority of infarcts have a reproducible chest wall.

WHO PRESENTS ATYPICALLY

Women, people with diabetes, the elderly, and postoperative patients present with dyspnea, fatigue, nausea or confusion rather than pain. **Keep the threshold for an ECG and a troponin low in these groups**; this is where infarcts are missed.

PEARLS & PITFALLS

- ECG within 10 minutes, and repeat it.** A single normal tracing in ongoing pain is not reassurance, it is an incomplete test.
- Check the blood pressure in both arms before you anticoagulate.** Treating a dissection as ACS is the catastrophic version of this miss.
- Troponin means myocardial injury, not infarction.** Interpret the delta and the clinical context, never a lone number.
- HEART is for disposition; GRACE is for cath timing.** Swapping them is the classic score error on this presentation.
- Tension pneumothorax is diagnosed at the bedside.** If you are waiting on the chest film, you have already waited too long.
- Reproducible chest wall tenderness does not exclude ACS,** and neither does relief with a gastrointestinal cocktail.





AF with RVR

Atrial fibrillation with rapid ventricular response · treat the trigger, not just the number

< 110

lenient rate target

UNSTABLE? CARDIOVERT NOW

Hypotension or shock, ischemic chest pain, acute pulmonary edema, or altered mentation caused by the rate means **immediate synchronized DC cardioversion at 120 to 200 J biphasic**, with sedation if time allows. **The judgment is whether the rate is causing the instability or merely accompanying it**: a septic patient at 140 is usually tachycardic because of the sepsis, and cardioverting them treats the wrong problem and rarely holds.

TREAT THE TRIGGER FIRST *new AF in an acutely ill patient is usually a symptom, and it often reverts when the cause is fixed*

FIND THE DRIVER

Sepsis, hypovolemia, pain, PE, hyperthyroidism, alcohol, post-operative state, and myocardial ischemia. Rate-controlling a patient whose driver is untreated means escalating doses against a physiologic tachycardia, and the drugs then cause the hypotension you were trying to avoid.

CORRECT POTASSIUM AND MAGNESIUM

Low magnesium sustains RVR and blunts every rate-control drug you give. Replace it empirically in the acutely ill: **magnesium 2 g IV over 15 min** both slows AV conduction directly and improves the response to everything else.

WORK IT UP

12-lead ECG (not just the monitor), **electrolytes**, **TSH**, **troponin if ischemia is plausible**, and **an echo**. The ECG is also how you catch the one presentation that changes every drug decision, below.

RATE CONTROL *lenient is as good as strict, and the choice of agent is decided by the ejection fraction*

Drug	Dose	When to use it, and when not to
Metoprolol <i>first-line</i>	2.5 to 5 mg IV over 2 min , repeat q5 min to a max of 15 mg , then convert to oral	Preferred when there is no decompensated heart failure , and the better choice when the driver is adrenergic (post-operative, thyrotoxic, ischemic). Avoid in acute decompensated HF , severe asthma , and bradycardia .
Diltiazem	0.25 mg/kg IV over 2 min , may repeat at 0.35 mg/kg , then infusion 5 to 15 mg/h	Equally effective and often faster. Contraindicated in HFrEF , because its negative inotropy can precipitate cardiogenic shock, and in pre-excitation .
Digoxin	0.25 to 0.5 mg IV load , then 0.25 mg q6h , total ≤ 1.5 mg in 24 h	The add-on for heart failure or hypotension , since it does not drop the blood pressure. Slow onset over hours , so it is never the answer to an urgent rate. It controls the rate at rest but poorly with exertion, and toxicity rises in renal impairment.
Amiodarone	150 mg IV over 10 min , then 1 mg/min for 6 h , then 0.5 mg/min	Useful when heart failure or hypotension rules out the others, and gives rate and rhythm control together . Give by central line where possible (phlebitis). Treat it as chemical cardioversion : it may restore sinus rhythm, so the same anticoagulation rules apply.

LENIENT BEATS STRICT

Target a resting rate below 110. RACE II, NEJM 2010 found lenient control non-inferior to a strict target of under 80, with **fewer drugs, fewer visits and fewer side effects**. Chase a tighter number only if the patient remains symptomatic or the ventricle is failing from the rate.

RHYTHM CONTROL AND ANTICOAGULATION *the 48-hour rule governs when you may cardiovert safely*

- Under 48 h of clearly documented onset**: cardioversion is reasonable without prior anticoagulation, electrically or chemically. Use **flecainide or propafenone only if there is no structural heart disease**, and **amiodarone** if there is. **Anticoagulate at the time of cardioversion regardless**, since the atrium is stunned afterwards and the stroke risk is highest in the days that follow.
- Over 48 h or unknown duration**: either **3 weeks of therapeutic anticoagulation before** elective cardioversion, or a **TEE to exclude left atrial appendage thrombus** and cardiovert immediately. Either way, **anticoagulate for at least 4 weeks afterwards**, because mechanical atrial function recovers long after the rhythm looks normal.
- Consider early rhythm control in newly diagnosed AF**. EAST-AFNET 4, NEJM 2020 found that starting rhythm control **within a year of diagnosis** reduced cardiovascular death, stroke and hospitalization. In critical illness the opposite applies: **treat the cause and rate-control**, because most of these patients revert on their own.

STROKE PREVENTION *decided by the score, never by the rhythm strategy*

CHA₂DS₂-VASc

C heart failure 1 · **H** hypertension 1 · **A**₂ age ≥ 75 2 · **D** diabetes 1 · **S**₂ prior stroke, TIA or thromboembolism 2 · **V** vascular disease 1 · **A** age 65-74 1 · **Sc** female 1.
Anticoagulate at ≥ 2 in men and ≥ 3 in women; at 1 and 2 respectively it is a discussion. **The score does not change because you restored sinus rhythm**.

DOAC DOSING

Apixaban 5 mg BID, reduced to **2.5 mg BID** if **2 of 3**: age ≥ 80, weight ≤ 60 kg, creatinine ≥ 1.5. **Rivaroxaban 20 mg daily with the evening meal** (15 mg if CrCl 15-50). **Edoxaban 60 mg daily**, 30 mg if CrCl 15-50 or weight ≤ 60 kg, and **avoid if CrCl > 95**. **Dabigatran 150 mg BID** (75 mg if CrCl 15-30).

WHEN A DOAC IS WRONG

Moderate-to-severe mitral stenosis or a mechanical valve: warfarin only. DOACs were harmful in the mechanical-valve trial. **Antiphospholipid syndrome: warfarin**. In severe renal impairment use apixaban or warfarin, and **never combine a DOAC with an antiplatelet** without a specific, time-limited indication.

THE TRAP: PRE-EXCITED AF *the one situation where standard rate control can kill the patient*

RECOGNIZE IT

An irregularly irregular, broad-complex tachycardia, often very fast (above 200) with beat-to-beat variation in QRS width. This is AF conducting down an accessory pathway in **WPW**. Look for a delta wave on any previous ECG.

WHY AV-NODAL BLOCKERS KILL

Blocking the AV node removes the only competing route and pushes all conduction down the accessory pathway, which has no decremental properties. The ventricular rate accelerates and degenerates into **ventricular fibrillation**. This applies to **adenosine, beta-blockers, diltiazem, verapamil and digoxin**.

WHAT TO GIVE INSTEAD

Procainamide, or **synchronized DC cardioversion**, which is the safest choice in anyone unstable or when you are unsure. Amiodarone is **not** a safe default here. Refer for accessory pathway ablation afterwards.

PEARLS & PITFALLS

- New AF in critical illness is usually the symptom**. Treat sepsis, pain, hypovolemia and the PE, and the rate often follows.
- Replace magnesium early**. It aids rate control and is nearly always low in these patients.
- A rate above 150 that is perfectly regular is probably not AF**. Reconsider flutter with 2:1 block or SVT.
- Irregular and broad means no AV-nodal blocker**. Procainamide or shock.
- Diltiazem is the wrong drug in HFrEF**, where digoxin or amiodarone is safer.
- Anticoagulation follows CHA₂DS₂-VASc, not the rhythm**. Restoring sinus rhythm does not remove the stroke risk.





Sepsis & Septic Shock

Recognize it · antibiotics and cultures · resuscitate · control the source

First hour
time is mortality

DEFINE AND RECOGNIZE *Sepsis-3, and why the screening tool is not the diagnosis*

THE DEFINITIONS

Sepsis: life-threatening organ dysfunction from a dysregulated host response to infection, operationalized as an acute rise in **SOFA of 2 or more**.
Septic shock: vasopressors needed to keep MAP $\geq$ 65 AND lactate $>$ 2 mmol/L despite adequate fluid. That combination carries **hospital mortality above 40%**, which is why it is named separately.

qSOFA IS NOT A SCREEN

RR $\geq$ 22, altered mentation, SBP $\leq$ 100. Two or more should escalate care, but **qSOFA has poor sensitivity and misses many septic patients**, so **never use it alone to rule sepsis out**. Pair it with **NEWS2 or MEWS and clinical judgment**. Its real value is prognostic, not diagnostic.

LACTATE

Above 2 mmol/L signals hypoperfusion, and it rises before the blood pressure falls, which is why a normotensive patient with a lactate of 4 is already in trouble. **Recheck at 2 to 4 h: clearance matters more than the single value**, and a falling lactate is the most useful early sign that resuscitation is working.

THE FIRST HOUR *each element, and the reason it is in the bundle*

Action	Why, and the practical catch
1. Measure lactate	Identifies occult hypoperfusion in a patient who still looks stable. Remeasure within 2 to 4 h if above 2.
2. Blood cultures before antibiotics	Two or more sets , because cultures drawn after the first dose lose yield and you lose the chance to de-escalate later. But never delay antibiotics beyond about 45 min to obtain them.
3. Broad-spectrum antibiotics	Immediately in septic shock. Time to antibiotics is the single most consistent modifiable driver of mortality here , so give them before imaging, before the ICU bed, and before the lactate returns. Cover the likely source and the local resistance pattern.
4. Crystalloid 30 mL/kg	For hypotension or lactate $\geq$ 4. Use a balanced crystalloid rather than 0.9% saline , which delivers a supraphysiologic chloride load and causes hyperchloremic acidosis and worse renal outcomes. Treat 30 mL/kg as a starting point, not a mandate: reassess as you go in heart failure, cirrhosis and dialysis patients.
5. Vasopressors if MAP $<$ 65	Start during or after fluids, not once they are finished , since prolonged hypotension while a bag runs is itself organ injury. A peripheral line is acceptable to avoid delay while central access is obtained.

RESUSCITATE *fluids until they stop helping, then vasopressors*

FLUIDS, THEN STOP GUESSING

After the initial volume, guide further fluid by dynamic measures: passive leg raise with a stroke-volume response, or pulse-pressure or stroke-volume variation. **Roughly half of hemodynamically unstable patients are not fluid responsive**, so reflexive repeat boluses simply cause edema, worsen oxygenation and prolong ventilation. **Avoid starches and gelatins**; consider albumin when large volumes are needed.

THE VASOPRESSOR LADDER

1. Norepinephrine first-line, titrated to MAP $\geq$ 65; it raises pressure with **fewer arrhythmias than dopamine**. **2. Add vasopressin 0.03 units/min** once norepinephrine reaches about **0.25 to 0.5 mcg/kg/min**, which is **catecholamine-sparing** rather than simply additive. **3. Add epinephrine** if the MAP is still low. **Angiotensin II** in refractory shock.

THE MAP TARGET IS A FLOOR

MAP $\geq$ 65 is a floor, not a ceiling, and **60 to 65 is reasonable in patients over 65**, where a higher target adds arrhythmia without benefit. **Individualize upward in chronic hypertension**, whose autoregulation is shifted right. **Follow perfusion, not the number:** lactate clearance, urine output, mentation and capillary refill.

SOURCE CONTROL, STEROIDS AND STEWARDSHIP *what determines the second 24 hours*

Element	What to do	Why
Source control	Identify and control the anatomic source as soon as possible, ideally within 6 to 12 h. Drain, debride, or remove the device	No antibiotic sterilizes an undrained collection or an infected line. Failure to respond is usually a source problem, not a drug problem.
Corticosteroids	Hydrocortisone 200 mg/day (50 mg q6h or by infusion) for shock on ongoing vasopressors	The evidence is split. ADRENAL, NEJM 2018 found no mortality benefit but faster shock reversal ; APROCCHSS, NEJM 2018, adding fludrocortisone , did show one. Both shortened shock reversal , which is the defensible reason to give it.
De-escalate	Narrow on culture results, review daily, and set a stop date	Prolonged broad therapy drives C. difficile, resistance and candidemia . Procalcitonin can support stopping, though not starting.

EMPIRIC ANTIBIOTICS BY SOURCE *default is vancomycin + cefepime; add anaerobic cover only for an anaerobic source*

Source	First-line empiric	Why
Unknown <i>the usual starting point</i>	Vancomycin + cefepime 2 g q8h	Covers MRSA, Pseudomonas and GNR without needless anaerobic spectrum , which is associated with higher mortality, C. difficile and VRE . Meropenem if prior ESBL.
Anaerobic source <i>intra-abdominal, aspiration with abscess, necrotizing, pelvic, bite</i>	Vancomycin + piperacillin-tazobactam, or vancomycin + cefepime + metronidazole 500 mg q8h	Anaerobic cover is essential here. ACORN was neutral on AKI, so do not withhold pip-tazo on that basis. Also necrotizing soft tissue: add clindamycin, call surgery.
Lung	Community: ceftriaxone 2 g q24h + azithromycin. HAP/VAP: vancomycin + cefepime	Pneumonia is not an anaerobic source unless aspiration with abscess. Never a beta-lactam alone in severe CAP.
Urine	Ceftriaxone 2 g q24h; cefepime if Pseudomonas risk or complicated; carbapenem if ESBL	The urinary tract is not an anaerobic source. Decompress obstruction and replace the catheter , or antibiotics fail.
CNS	Ceftriaxone 2 g q12h + vancomycin + dexamethasone	Dexamethasone with or before the first dose. Add ampicillin if over 50, pregnant or immunosuppressed, for Listeria.
Neutropenic fever	Cefepime 2 g q8h monotherapy	Add vancomycin only for instability, line infection, severe mucositis or known MRSA. Not routine.

PEARLS & PITFALLS

- **Never delay antibiotics** for a lactate, imaging or an ICU bed.
- **A normal qSOFA does not exclude sepsis.**
- **Half of unstable patients are not fluid responsive.** Reassess dynamically.
- **Not improving? Hunt an undrained source** before broadening.
- **Do not forget the rest:** VTE and ulcer prophylaxis, glucose 140–180, early enteral feed.

References: Surviving Sepsis Campaign international guidelines (SCCM / ESICM) · Singer et al, Sepsis-3 definitions, *JAMA* 2016 · Venkatesh et al, ADRENAL, *NEJM* 2018 ·

Anname et al, APROCCHSS, *NEJM* 2018 · Semler et al, SMART, *NEJM* 2018.

Educational aid; verify doses against local protocol, renal function, and patient factors.





Delirium

Acute brain failure · Find the cause, fix the environment, and avoid the sedatives

PREVENTION WORKS

drugs largely do not

DIAGNOSE IT *the hypoactive form is commoner and routinely missed*

THE CORE FEATURES

Acute onset and fluctuating course · **inattention** — the cardinal feature · **disorganized thinking** · **altered level of consciousness**. Inattention plus acute fluctuation is the combination that defines it.

SCREEN FORMALLY

Use a validated tool — **4AT** on the wards, **CAM**, or **CAM-ICU** in ventilated patients. **Screen at least daily** in at-risk patients. Informal impression misses the majority of cases, particularly overnight.

THREE MOTOR SUBTYPES

Hypoactive — withdrawn, drowsy, quiet. **Commonest and most missed**, and carries the **worst prognosis**. **Hyperactive** — agitated, the one that gets referred. **Mixed** — fluctuating between the two.

FIND THE CAUSE *delirium is a symptom — the diagnosis is what caused it*

Category	Look for
Drugs start here, every time	Benzodiazepines, opioids, anticholinergics, steroids, antihistamines . Also withdrawal from alcohol, benzodiazepines or nicotine. Review every medication started or stopped in the last 72 hours.
Infection	Urinary, respiratory, skin, intra-abdominal. But treat asymptomatic bacteriuria as a red herring — a positive urine culture without urinary symptoms does not explain delirium and treating it causes harm.
Metabolic	Sodium, calcium, glucose, urea, liver failure, hypoxia, hypercapnia, thyroid disease, thiamine deficiency .
Structural & neurological	Stroke, subdural hematoma, seizure or non-convulsive status , meningitis or encephalitis. Image if there are focal signs, head injury, anticoagulation, or no other explanation.
The simple things	Pain, urinary retention, constipation , dehydration, and sleep deprivation. These are common, easily missed, and easily fixed — check a bladder scan and the bowel chart before escalating.

MANAGE IT NON-PHARMACOLOGICALLY *this is the treatment, not an adjunct*

- 1 Treat the precipitant.** Everything else is supportive. If you have not identified a cause, you have not finished looking.
- 2 Reorient and normalize.** Clock and calendar visible, curtains open by day, **lights off and quiet at night**, familiar objects, and **family present as much as possible**. Consistent staff where you can.
- 3 Restore the senses.** **Glasses on, hearing aids in, dentures fitted**. Sensory deprivation is a potent and completely reversible contributor that is forgotten daily.
- 4 Mobilize, hydrate, and feed.** Get them out of bed and walking. **Remove tethers** — catheters, telemetry leads, infusions — as soon as they are not needed. Protect sleep by clustering observations.
- 5 Deprescribe.** Stop or reduce the culprit drugs, particularly benzodiazepines and anticholinergics, and use **non-opioid analgesia** where possible — though **untreated pain itself causes delirium**, so do not leave patients in pain to avoid opioids.

WHEN TO USE MEDICATION *a narrow and reluctant indication*

THE EVIDENCE

Antipsychotics do not prevent delirium and do not shorten it. The large **MIND-USA** trial found haloperidol and ziprasidone did not improve outcomes in ICU delirium, and guidance recommends against their routine use for prevention or treatment. **Benzodiazepines make delirium worse** and should be avoided except for **alcohol or benzodiazepine withdrawal**, where they are the treatment.

IF YOU MUST

Reserve for **severe agitation causing genuine risk** to the patient or others, or distress that non-pharmacological measures have failed to settle. Use the **lowest effective dose of a single agent for the shortest time**, review daily, and **document a stop date**. Check the **QTc** and the electrolytes. **Never continue an antipsychotic to discharge by default** — this is a major source of long-term inappropriate prescribing.

PREVENT IT *the one intervention with strong evidence — about a third of cases are preventable*

Target	The intervention
Cognitive impairment	Orientation, cognitively stimulating activity, and familiar faces. Identify pre-existing dementia on admission — it is the strongest single risk factor and changes your threshold for everything else.
Sleep deprivation	Non-pharmacological sleep protocol: cluster observations, reduce noise and light, avoid overnight labs and vital-sign checks where safe. Do not use sedatives to induce sleep — they cause the problem you are trying to prevent.
Immobility	Early and repeated mobilization , range-of-motion exercises, and minimizing catheters, lines and restraints that physically tether the patient to the bed.
Vision and hearing	Ensure glasses and hearing aids are present and working . Ask the family to bring them in — they are frequently left at home on an emergency admission and never replaced.
Dehydration	Early recognition and correction, with encouragement of oral intake and a proactive approach to volume in the frail.

WHO IS AT RISK *identify them on admission and apply the bundle proactively*

PREDISPOSING

Age over 65 · **pre-existing cognitive impairment or dementia** — the dominant risk factor · frailty and multimorbidity · sensory impairment · previous delirium · depression · alcohol excess · polypharmacy · malnutrition.

PRECIPITATING

Acute illness and surgery (especially hip fracture and cardiac surgery) · ICU admission · **new psychoactive drugs** · uncontrolled pain · urinary catheterization · physical restraint · multiple ward moves. **The frailer the patient, the smaller the insult needed** — in advanced dementia a urinary catheter alone can precipitate it.

PEARLS & PITFALLS

- Hypoactive delirium is the commonest form and the most dangerous.** A quiet, withdrawn patient is not "settled" — screen them.
- Delirium is not a diagnosis.** Name the precipitant, or keep looking.
- Do not treat asymptomatic bacteriuria** as the cause. It is an extremely common false explanation that stops the real workup.
- Antipsychotics do not treat delirium.** They sedate agitation, at the cost of QTc, falls, and inappropriate long-term prescriptions.
- Check for retention and constipation** before escalating. A bladder scan solves a surprising number of cases.
- Delirium predicts long-term cognitive decline and mortality.** Document it, communicate it at discharge, and arrange follow-up.



SET THE TARGET *tight control has no benefit and real harm on the wards*

WARDS (NON-ICU) · NEW 2025

100–180 mg/dL (5.6–10.0 mmol/L) if reached without significant hypoglycemia (ADA 2025, kept in 2026). A **fasting value below 100** predicts hypoglycemia within 24 h: trim that day's basal dose.

ICU

140–180 mg/dL (7.8–10.0 mmol/L) on an insulin drip, started for glucose persistently ≥ 180. **Avoid targets below 110:** tight control (81–108) raised mortality in NICE-SUGAR. 110–140 for selected patients (cardiac surgery) **only without hypoglycemia**.

RELAX IT

Up to 250 mg/dL may be acceptable with terminal illness or short life expectancy, advanced kidney failure or dialysis, high hypoglycemia risk, or labile glucose. State the individualized target in the notes.

BUILD THE REGIMEN *three components, prescribed deliberately*

Component	What it does	How to set it
1 · Basal glargine, detemir, degludec, or NPH	Covers hepatic glucose output between meals and overnight	Roughly 50% of the total daily dose . Starting total is about 0.3–0.5 units/kg/day , reduced to 0.2–0.3 in renal impairment, the elderly, or the underweight.
2 · Prandial (bolus) rapid-acting analogue	Covers the carbohydrate in each meal	The other 50% split across meals . Hold the prandial dose if the patient does not eat , and give it with or just after the meal in unreliable eaters.
3 · Correction	Corrects the glucose that is already high	A supplemental scale on top of basal-bolus, not instead of it. Choose a sensitivity based on total daily dose and weight.

Sliding-scale insulin used alone is discouraged: it treats hyperglycemia only after it has happened, produces wide swings, and is associated with worse control. It remains reasonable only as a short-term bridge while you determine requirements, or in a patient with mild, transient hyperglycemia.

WHAT TO DO WITH THE HOME MEDICATIONS *most oral agents come off*

GENERALLY STOP ON ADMISSION

Metformin: hold with AKI, hypoxia, sepsis, contrast, or before surgery (lactic acidosis risk). **SGLT2 inhibitors:** hold in acute illness and **before surgery**, because of **euglycemic ketoacidosis**, in which the glucose looks reassuringly normal. **Sulfonylureas:** hold, as unpredictable intake causes hypoglycemia. **Thiazolidinediones:** hold with heart failure or edema.

OFTEN CONTINUE

DPP-4 inhibitors are generally well tolerated and weight-neutral. **GLP-1 receptor agonists** may be continued in stable patients, but cause nausea and **delayed gastric emptying**, which matters around procedures and sedation. **Never stop insulin in type 1 diabetes**, even when NPO: withholding basal precipitates ketoacidosis.

SPECIAL SITUATIONS *where regimens fail*

Situation	Approach
NPO / surgery	Continue basal (often at 75–80% of the usual dose), hold prandial , and keep correction doses. Check glucose every 4–6 hours . Use a variable-rate insulin infusion for prolonged fasting or major surgery.
Enteral or parenteral feeding	Match insulin to the feed. Continuous feed needs basal cover plus regular correction; bolus feeds need rapid-acting insulin with each. If the feed stops unexpectedly, the insulin must be reviewed immediately , as this is a common cause of severe hypoglycemia.
Glucocorticoids	Cause a predominantly post-prandial and afternoon rise. Prednisone given in the morning often needs additional intermediate-acting insulin (NPH) in the morning . Anticipate the rise, and taper the insulin as the steroid tapers .
Transitioning off an infusion	Give the first dose of subcutaneous basal 2–4 hours before stopping the drip . Stopping an infusion without overlap is one of the commonest causes of rebound hyperglycemia and ketoacidosis.

HYPOGLYCEMIA *the complication your regimen is most likely to cause*

Level	Definition and response
Level 1: alert	< 70 mg/dL (3.9 mmol/L). Treat and, importantly, review the regimen : this is the signal to act before it becomes severe.
Level 2: clinically significant	< 54 mg/dL (3.0 mmol/L). Requires immediate treatment and a documented change to the insulin orders.
Level 3: severe	Any level with altered mental status requiring assistance . A patient-safety event that should trigger formal review.
Treat it	Conscious: 15–20 g fast-acting oral carbohydrate, recheck in 15 minutes, repeat as needed, then give a complex-carbohydrate snack if the next meal is distant. Unable to swallow: IV dextrose , or glucagon if there is no access, noting that glucagon is less effective in liver disease or malnutrition.
Prevent it	Commonest causes are an interrupted feed or meal , a missed insulin adjustment after steroids are tapered , improving renal function (insulin clearance rises), and overlapping correction doses . Review insulin daily against actual readings rather than continuing yesterday's orders.

HbA1c AND DISCHARGE PLANNING *the admission is a chance to reset the outpatient regimen*

CHECK AN HbA1c

Measure in every patient with diabetes or hyperglycemia **if it has not been done in the previous 3 months**. It distinguishes **stress hyperglycemia** from previously undiagnosed diabetes and tells you whether the home regimen was working: a high value means discharge on the same regimen will fail.

PLAN THE HANDOVER

Provide **written instructions, a glucose meter and supplies**, and confirm the patient or carer can demonstrate the technique. Arrange **follow-up within 1–2 weeks** of a regimen change. Consider **cost and access** explicitly: insulin the patient cannot afford is the commonest silent cause of readmission.

PEARLS & PITFALLS

- **Never withhold basal insulin in type 1 diabetes**, including when NPO. Basal covers hepatic glucose output, not food.
 - **Overlap subcutaneous basal with the insulin infusion by 2–4 hours**, or the patient will rebound.
 - **SGLT2 inhibitors cause euglycemic ketoacidosis**. Hold them in acute illness and before surgery, and check ketones even when the glucose looks fine.
- **Sliding scale alone is reactive**. Prescribe basal-bolus with correction and adjust it daily against the actual readings.
 - **Review insulin the moment a feed is interrupted:** this is a leading cause of inpatient hypoglycemia.
 - **Plan the discharge regimen and the follow-up**, and make sure the patient can actually administer and afford it. An elegant inpatient regimen that fails at home has achieved nothing.



Inpatient Hypoglycemia

Treat it now, then find out why · It is almost always something you can prevent

< 54 mg/dL
clinically significant

DEFINE AND TREAT *do not wait for a laboratory glucose*

THE LEVELS

Level 1 — < 70 mg/dL (3.9 mmol/L): an alert value. Treat and **review the regimen**. **Level 2** — < 54 mg/dL (3.0 mmol/L): clinically significant. **Level 3** — **severe**: any level with **altered mental status requiring assistance** — a patient-safety event.

CONSCIOUS AND SWALLOWING

15–20 g fast-acting oral carbohydrate — glucose tablets, juice, or gel. **Recheck in 15 minutes and repeat if still low**. Once above target, give a **complex carbohydrate snack or the next meal**, or it will simply fall again.

UNCONSCIOUS OR NPO

Intravenous dextrose, then recheck. **Glucagon only if there is no IV access**. Doses below.

THE DOSES *because “give dextrose” is not an order*

IV DEXTROSE

D50W 25 g (50 mL) IV push through a good vein, or **D10W 100-250 mL**, which is gentler on the vein and causes less rebound hyperglycemia. **Recheck at 15 min**. Follow with a complex carbohydrate or a **D10 infusion** if the cause is still acting.

GLUCAGON, ONLY WITHOUT ACCESS

1 mg IM or SC, or 3 mg intranasal. It mobilizes **glycogen, so it fails where there is none**: liver disease, malnutrition, alcohol. Often causes vomiting. **Get access and give dextrose**.

OCTREOTIDE FOR SULFONYLUREA

50-100 mcg SC every 6 to 8 h. It suppresses the insulin release that dextrose keeps re-triggering, which is why repeat glucose boluses fail here. **Admit; do not discharge after one correction**.

FIND THE CAUSE *in hospital it is nearly always iatrogenic or situational*

Cause	Detail
The feed or meal stopped	The single commonest inpatient cause. Enteral feed interrupted for a procedure, vomiting, nil-by-mouth orders, or a missed meal — while the insulin continued. Any interruption to nutrition must trigger an immediate insulin review .
Insulin or sulfonylurea	Wrong dose, wrong timing, duplicated prescriptions, or a correction scale stacked on top of basal-bolus. Sulfonylureas cause prolonged, recurrent hypoglycemia and are a common culprit in the elderly.
Improving renal function	Insulin and sulfonylureas are renally cleared . As an acute kidney injury recovers, clearance rises and the previously appropriate dose becomes an overdose. Frequently missed.
Steroid taper	Glucose falls as steroids are reduced or stopped, but the insulin regimen built around the steroid stays in place. Reduce insulin as the steroid comes down .
Illness itself	Sepsis, liver failure, adrenal insufficiency, alcohol, malnutrition and cachexia . New unexplained hypoglycemia in a non-diabetic patient should prompt thought of adrenal insufficiency and of occult sepsis.

TWO SITUATIONS THAT NEED MORE THAN DEXTROSE *where a single correction is not enough*

SULFONYLUREA-INDUCED

Do not discharge after a single correction. The effect is long, and **rebound hypoglycemia recurs for many hours to days**, particularly with long-acting agents and renal impairment. **Admit for observation and repeated glucose monitoring**, and consider **octreotide**, which suppresses the insulin secretion driving it and reduces the need for repeated dextrose boluses. Beware overdose and deliberate self-harm.

HYPOGLYCEMIA WITHOUT DIABETES

If there is no obvious cause, **the diagnostic samples must be taken during the hypoglycemia**, before treating — take them as you give the dextrose. Send **glucose, insulin, C-peptide, proinsulin, beta-hydroxybutyrate, and a sulfonylurea screen**, plus **cortisol**. **High insulin with high C-peptide** suggests insulinoma or a sulfonylurea; **high insulin with low C-peptide** indicates exogenous insulin administration. Missing that window means repeating the whole episode.

PREVENT THE NEXT ONE *this is a systems problem more than a clinical one*

Action	Detail
Review insulin daily	Against the actual glucose readings , not yesterday's orders. Adjust proactively rather than only reacting to a low.
Link nutrition to insulin	Make it explicit that a stopped feed or a nil-by-mouth order requires an insulin review the same hour . Hold prandial doses if the patient does not eat, and time rapid-acting insulin with or just after the meal in unreliable eaters.
Individualize the target	Relax targets in the frail, elderly, those with hypoglycemia unawareness, advanced renal or liver disease, and limited life expectancy. Tight control on the wards causes harm without benefit .
Treat it as an incident	A level 3 event should trigger a documented review of the regimen and, where relevant, an incident report. Recurrent hypoglycemia in the same patient is a prescribing failure, not bad luck.
Plan the discharge	Reassess the home regimen — the admission often reveals it was already too aggressive. Check the patient can recognize and self-treat , and address driving advice and hypoglycemia unawareness.

HYPOGLYCEMIA UNAWARENESS *the patients who stop feeling it*

WHY IT HAPPENS

Repeated hypoglycemia blunts the counter-regulatory and adrenergic warning response, so the patient loses the tremor, sweating and palpitations that would normally prompt them to eat. The first sign then becomes **confusion or collapse**. Commonest in **long-standing type 1 diabetes, tight control, autonomic neuropathy**, and in those on **beta-blockers**, which mask the adrenergic symptoms.

WHAT TO DO

Deliberately relax targets and scrupulously avoid further hypoglycemia for several weeks — awareness often recovers. Consider **continuous glucose monitoring with alarms**, structured education, and involvement of the diabetes team. **Ask about it directly**: "Do you still get warning symptoms before your sugar drops?" It changes the target, the regimen and the driving advice.

PEARLS & PITFALLS

- **Take the diagnostic labs before you give the dextrose** in unexplained hypoglycemia — the window closes the moment you treat.
- **Sulfonylurea hypoglycemia recurs**. One correction and discharge is a recognized cause of death; admit and consider octreotide.
- **Always follow the sugar with food**. Fast-acting carbohydrate alone will be followed by another fall.
- **Recovering renal function unmasks insulin overdose**. Reduce the dose as the creatinine improves.
- **An interrupted feed is an insulin emergency**. This single link prevents a large share of inpatient hypoglycemia.





Electrolyte Emergencies

Potassium · sodium · magnesium · phosphate · calcium

The rate
kills, not the number

POTASSIUM *hyperkalemia is the one that arrests the patient during your assessment*

Step	Aim	Agent and dose
1. PROTECT	Stabilize the myocardium	Calcium gluconate 10%, 10 mL IV over 2 to 3 min , for $K \geq 6.5$ or any ECG change. It does nothing to the potassium level: it raises the threshold potential and buys 30 to 60 min, so repeat it if the ECG changes persist . Use calcium chloride via central access if peripheral access is poor.
2. SHIFT	Drive potassium into cells	Insulin 10 units with 25 g dextrose IV , the most reliable shifter; recheck glucose hourly for 6 h , since late hypoglycemia is common. Albuterol 10 to 20 mg nebulized , roughly 8 times the asthma dose, and additive to insulin. Bicarbonate only if genuinely acidotic ; it is a weak shifter otherwise.
3. REMOVE	Eliminate potassium from the body	Hemodialysis is definitive , particularly in acute kidney injury. Loop diuretic if the patient still makes urine, and binders (patiromer or sodium zirconium cyclosilicate) for subacute control. Stop the culprits : potassium supplements, ACE inhibitors, ARBs, spironolactone, NSAIDs, trimethoprim.

THE POINT EVERYONE MISSES

Shifting is temporary and wears off in 2 to 6 h. Insulin and albuterol move potassium into cells; they do not remove a single milliequivalent from the body. **If you have not arranged removal, the potassium comes back**, usually overnight. **ECG progression: peaked T waves → PR prolongation → wide QRS → sine wave → arrest**, though the ECG is unreliable and a normal one never excludes dangerous hyperkalemia.

SODIUM *the rate of correction, not the target, is what causes the harm*

HYPONATREMIA

Correct by no more than 8 to 10 mEq/L in 24 h, and no more than 8 in high-risk patients: sodium at or below 105, hypokalemia, alcohol use, malnutrition or liver disease. Overcorrection causes **osmotic demyelination**, which appears days later and is often irreversible. **Acute symptomatic (seizure, coma): 3% saline 100 to 150 mL over 10 min, repeated up to 3 times** to raise the sodium 4 to 6 mEq/L and stop the seizure. **If you overshoot, give 5% dextrose with desmopressin** to relower it, the DDAVP clamp.

HYPERNATREMIA

Chronic: **lower by no more than 0.5 mEq/L per hour and 8 to 10 per 24 h**, to avoid cerebral edema as water re-enters brain cells that have adapted. **Free water deficit = total body water $\times ((Na / 140) - 1)$** , where TBW = weight $\times 0.6$ in men and 0.5 in women. **Replace with enteral water where possible**, otherwise 5% dextrose or 0.45% saline, and **remember ongoing losses**, which the formula does not include.

HYPOKALEMIA *and the magnesium rule that governs it*

REPLACE SAFELY

Peripheral: maximum 10 mEq/h at a concentration no greater than 40 mEq/L, since faster or stronger causes phlebitis and pain. **Central: 10 to 20 mEq/h** with continuous monitoring. **Mix in saline, not dextrose**, because dextrose triggers insulin release and drives potassium further into cells.

FIX THE MAGNESIUM FIRST

Refractory hypokalemia is hypomagnesemia until proven otherwise. Low intracellular magnesium unblocks the ROMK channel, so the kidney secretes every dose of potassium you give straight into the urine. **Give magnesium and potassium together**, not in sequence.

AIM HIGHER IN CARDIAC PATIENTS

Target $K \geq 4.0$ in heart failure, on digoxin, and after myocardial infarction or arrest, because hypokalemia lowers the threshold for ventricular ectopy and potentiates digoxin toxicity at otherwise therapeutic levels.

THE FORGOTTEN THREE *magnesium, phosphate and calcium, which explain a great many unexplained ICU problems*

Electrolyte	Low, and high
Magnesium <i>normal 1.7–2.2 mg/dL</i>	Low (common) : torsades and other arrhythmia, refractory hypokalemia and hypocalcemia , seizures, weakness. Causes: diuretics, GI losses, alcohol, PPIs, refeeding. Rx: $MgSO_4$ 1 to 2 g IV over 15 to 60 min; 2 g IV push for torsades regardless of the level . High (renal failure plus a load): loss of reflexes, hypotension, respiratory depression. Rx: IV calcium, stop the magnesium, dialysis .
Phosphate <i>normal 2.5–4.5 mg/dL</i>	Low : diaphragmatic weakness that makes a patient impossible to wean , plus hemolysis, rhabdomyolysis and confusion. Causes: refeeding , DKA treatment, alcohol, sepsis. Rx: IV sodium or potassium phosphate over 6 to 12 h , oral if mild. High : renal failure, tumor lysis, rhabdomyolysis. Rx: binders and treat the cause ; watch the calcium-phosphate product.
Calcium <i>check ionized</i>	Low : perioral tingling, Chvostek and Trousseau signs , tetany, prolonged QT , seizures. Causes: hypomagnesemia (functional hypoparathyroidism) , post-thyroidectomy, pancreatitis, massive transfusion (citrate), rhabdomyolysis. Rx: IV calcium gluconate for symptoms or a long QT, and correct the magnesium or it will not respond . High : see hypercalcemia of malignancy; treat with saline, then bisphosphonate or denosumab.

BEFORE YOU TREAT THE NUMBER *three checks that change what you do*

IS IT REAL?

Pseudohyperkalemia from hemolysis, a tight tourniquet, fist clenching, or extreme leukocytosis or thrombocytosis. **Pseudohyponatremia** from severe hyperlipidemia or paraproteinemia. **Repeat the sample before treating an isolated abnormal value in a well patient with a normal ECG**, but **never delay treatment in a sick one**.

ACUTE OR CHRONIC?

This decides the permitted rate of correction, and it is the single most important question in sodium disorders. Acute derangements (under 48 h) can be corrected faster because the brain has not adapted; chronic ones must be corrected slowly. **If the duration is unknown, treat it as chronic**.

WHAT IS THE VOLUME STATUS?

In hyponatremia it separates hypovolemic (saline), euvoletic (SIADH, fluid restrict) and hypervolemic (heart failure or cirrhosis, restrict and diurese) causes, which need opposite treatments. **Send paired serum and urine osmolality and a urine sodium before giving any fluid**, since fluids destroy the diagnostic information.

PEARLS & PITFALLS

- **Calcium protects the heart, it does not lower the potassium**. Always follow it with shift and removal.
- **Shifting wears off**. If you have not arranged removal, the potassium returns overnight.
- **The rate, not the target, causes osmotic demyelination**. Cap correction at 8 mEq/L per 24 h in high-risk patients.
- **Check magnesium in every refractory hypokalemia and hypocalcemia**. Neither corrects until it does.





Hyperkalemia

Protect · shift · remove · then find out why it happened

$K \geq 6.5$

or any ECG change

TREAT NOW IF $K \geq 6.5$, OR AT ANY LEVEL WITH ECG CHANGES OR RAPID RISE

Get a 12-lead and put the patient on a monitor. The classic sequence is **peaked T waves** → **flat P and long PR** → **wide QRS** → **sine wave** → **arrest**, but the ECG is insensitive and around half of dangerous hyperkalemia shows no classic change at all, so treat the number and the trend rather than the tracing. A **bradycardic or wide-complex rhythm** in a dialysis patient is hyperkalemia until proven otherwise.

THE THREE STEPS *protect the myocardium, shift potassium inward, then actually remove it*

Step	Agent and dose	Why, and what to watch
1 · PROTECT <i>onset minutes, lasts 30–60 min</i>	Calcium gluconate 10%, 10 mL IV over 2–3 min. Repeat if the ECG has not improved in 5–10 min. Calcium chloride 10 mL gives 3× the elemental calcium but needs central access.	Raises the threshold potential and stabilizes the myocardium without lowering the potassium at all. It buys minutes, so it must always be followed by shift and removal. Peripheral calcium chloride causes tissue necrosis if it extravasates. Never run calcium and bicarbonate through the same line, they precipitate.
2 · SHIFT <i>onset 15–30 min, lasts 2–6 h</i>	Insulin 10 units IV with 25 g dextrose. Consider 5 units if eGFR is low. Albuterol 10–20 mg nebulized (4–8× the bronchodilator dose). Bicarbonate only if genuinely acidotic.	Drops K by roughly 0.6–1.2 mEq/L , then it comes back. Hypoglycemia is the main harm, in 20–30% of patients, and 5 units halves severe events in renal impairment while lowering K a little less. Check glucose at 1 h and every 1–2 h for 6 h. Up to a third of patients do not respond to albuterol at all.
3 · REMOVE <i>the only step that changes the balance</i>	Hemodialysis is definitive, especially in AKI or oliguria. Loop diuretic if the patient still makes urine. Oral binder (SZC or patiomer). Stop the culprit drugs and all potassium.	Shifting is temporary, so K rebounds within 2–6 h unless it leaves the body. Call nephrology early rather than after the third rebound. Binders act too slowly for the acute emergency but prevent the next one.

FIND THE CAUSE *almost always reduced excretion, a drug, or cells spilling their contents*

REDUCED EXCRETION

AKI and advanced CKD are the commonest causes. Then **adrenal insufficiency, type 4 RTA** (see below), and **effective volume depletion**, where poor distal sodium delivery leaves nothing to exchange potassium for.

DRUGS

ACE inhibitors and ARBs, spironolactone and eplerenone, amiloride, NSAIDs, trimethoprim (blocks ENaC exactly as amiloride does), **heparin** (suppresses aldosterone synthesis), **calcineurin inhibitors**, non-selective beta-blockers, digoxin toxicity, succinylcholine, pentamidine, and potassium supplements or salt substitutes.

CELL RELEASE AND SPURIOUS

Rhabdomyolysis, tumor lysis, hemolysis, burns, crush injury, acidosis. Then rule out **pseudohyperkalemia**: a hemolyzed sample, a clenched fist under the tourniquet, a delayed specimen, or marked **leukocytosis or thrombocytosis**, where potassium leaks out during clotting. **Recheck on fresh plasma if the result does not fit the patient.**

TYPE 4 RTA *the cause people forget: mild kidney disease with unexplained, persistent hyperkalemia*

Type 4 renal tubular acidosis (hyporeninemic hypoaldosteronism)	
The lesion	Too little aldosterone, or a distal tubule that cannot respond to it. Aldosterone normally drives sodium reabsorption in exchange for potassium and hydrogen, so losing it impairs excretion of both . That is why the picture is hyperkalemia and acidosis together.
The picture	Normal anion gap (hyperchloremic) metabolic acidosis with hyperkalemia , in a patient whose creatinine is only mildly to moderately raised. Urine pH is usually below 5.5 , which separates it from type 1 RTA, where acidification fails and the urine stays inappropriately alkaline. The hyperkalemia is out of proportion to the degree of kidney impairment, and that mismatch is the clue.
Who gets it	Diabetic nephropathy is the classic setting , then chronic interstitial disease, obstructive uropathy, HIV, sickle cell, and lupus. Drug causes overlap with the list above: ACE inhibitors and ARBs, spironolactone, NSAIDs, trimethoprim, heparin, calcineurin inhibitors.
Treatment	Stop or reduce the offending drug first , since that alone often fixes it. Then a low-potassium diet , a loop or thiazide diuretic to increase distal sodium delivery, and sodium bicarbonate if the acidosis persists, which also shifts potassium. Fludrocortisone 0.1 mg daily is a last resort, because the mineralocorticoid load worsens hypertension, edema, and heart failure in exactly the population that has this diagnosis.

ONGOING CONTROL *the acute fix lasts hours; this is what stops the readmission*

Option	Dose	Where it fits
Sodium zirconium cyclosilicate	10 g PO three times daily for up to 48 h, then 5–10 g daily	Onset within about an hour, so it is the binder of choice when you want potassium gone rather than hidden. Carries a sodium load , so watch for edema in heart failure and cirrhosis.
Patiomer	8.4 g PO daily , titrated weekly	Onset around 7 h , so it is a maintenance drug, not a rescue. Binds magnesium as well , so check magnesium, and separate it from other oral drugs by 3 h.
Sodium polystyrene sulfonate	Largely superseded	Slow, poorly evidenced, and associated with colonic necrosis , particularly with sorbitol and in postoperative or ileus patients.
Drugs and diet	Review at every visit	Do not reflexively stop the ACE inhibitor, ARB, or MRA in heart failure or CKD , where they carry a mortality benefit. Dose-reduce, add a binder, and correct acidosis instead. Ban salt substitutes , which are potassium chloride.

MONITOR AND DISPOSITION *the rebound is the part that catches people out*

AFTER TREATMENT

Cardiac monitor until $K < 6.0$ and the ECG has normalized. **Recheck potassium at 1 h and again at 2–4 h**, because insulin and albuterol wear off together and the level climbs back. Recheck **glucose** on the same schedule. **Do not discharge on the strength of a single post-treatment potassium** if nothing has been removed from the body.

WHEN TO CALL RENAL EARLY

Refractory or rapidly rising potassium, oliguric AKI, dialysis dependence, or hyperkalemia driven by ongoing tissue breakdown such as rhabdomyolysis or tumor lysis, where potassium arrives faster than you can shift it. **Shifting only postpones the dialysis, so make the call at the start.**

PEARLS & PITFALLS

- **Calcium protects but does not lower potassium.** Giving it and stopping there is the classic error; it must always be followed by shift and removal.
- **Insulin and albuterol only move potassium into cells.** Unless it leaves the body, the level rebounds in 2–6 h.
- **Recheck glucose after insulin**, since hypoglycemia is common and late.
- **A normal ECG does not exclude danger.** Sensitivity is poor, so treat the number and the trend.
- **Unexplained hyperkalemia in a diabetic with mild CKD is type 4 RTA** until proven otherwise.
- **Check the sample before the guidelines.** Hemolysis explains a lot of this.

References: UK Kidney Association Treatment of Acute Hyperkalemia in Adults · KDIGO and standard critical care references · trials of insulin dosing in renal impairment · patiomer and sodium zirconium cyclosilicate prescribing information.

Educational aid; verify doses against local protocol, renal function, and patient factors.





Hypertensive Emergency

Severe blood pressure with acute organ damage · controlled reduction, never a crash

≤ 25%
SBP drop, first hour

THE DAMAGE DEFINES IT, NOT THE NUMBER

Emergency is severe hypertension, usually above 180/120, with **acute target-organ damage**, and it needs **titratable IV therapy in a monitored setting**. **Urgency** is a high number **without** acute damage, and it needs **oral therapy and gradual reduction over days**. A patient at 230/130 who feels well and has clean labs, ECG and exam is an **urgency**, and treating them with IV drugs causes the strokes and infarcts this sheet exists to prevent. **Look for the damage before you reach for the pressure.**

FIND THE TARGET-ORGAN DAMAGE *a focused search, because each site changes the target and the drug*

Organ	What to look for
Brain	Hypertensive encephalopathy (headache, confusion, seizures, visual loss, often with PRES on MRI), ischemic or hemorrhagic stroke . Encephalopathy improves as the pressure falls; a stroke does not, which is why the two get opposite targets.
Heart	Acute coronary syndrome and acute pulmonary edema , including SCAPE (sympathetic crashing acute pulmonary edema): sudden severe dyspnea, hypertension and diffuse crackles from afterload mismatch rather than volume overload.
Aorta	Acute aortic dissection . Tearing pain, pulse or pressure differential, widened mediastinum. The most time-critical target on this page , and the only one where you lower both rate and pressure aggressively.
Kidney and eye	Acute kidney injury with active urine sediment, and retinal hemorrhages, exudates or papilledema . Fundoscopy is the cheapest organ-damage test you have and is routinely skipped.
Pregnancy	Preeclampsia with severe features, or eclampsia , at ≥ 20 weeks and up to 6 weeks postpartum. Different thresholds and different drugs entirely.

HOW FAST, AND HOW LOW *the target depends on the organ, and getting this backwards causes harm*

Scenario	Target	Why
Most emergencies	Reduce SBP by no more than 25% in the first hour , then below 160/100 by 2 to 6 h, then 130 to 140 over 24 to 48 h	Chronic hypertension shifts cerebral autoregulation to the right , so a "normal" pressure is under-perfusion for this patient. Crashing the pressure causes watershed stroke, myocardial ischemia and acute kidney injury.
Aortic dissection	SBP 100 to 120 and HR < 60, within minutes	Rate first, then pressure . Give the beta-blocker (esmolol or labetalol) before any vasodilator , because a vasodilator alone causes reflex tachycardia that raises dP/dt and propagates the dissection.
Ischemic stroke	Permissive . Treat only above 220/120 , or below 185/110 if thrombolysing	The penumbra is perfused by pressure-dependent collaterals, so lowering the BP extends the infarct.
Intracerebral hemorrhage	SBP to about 140, smoothly; avoid dropping below 130	Reduces hematoma expansion. Large or rapid swings are associated with harm , so a smooth infusion beats intermittent boluses.
SCAPE / pulmonary edema	High-dose nitroglycerin , plus NIV	The problem is afterload, not volume . Doses far above the usual anginal range are needed, and heavy diuresis in a patient who is not fluid-overloaded is counterproductive.

THE IV AGENTS *short-acting and titratable, so a mistake can be undone in minutes*

Drug	Dose	Where it fits, and the caution
Labetalol	10-20 mg IV over 2 min, repeat or double q10 min ; or infusion 0.5-2 mg/min . Cumulative max 300 mg .	Combined alpha and beta blockade; the workhorse for most emergencies including dissection and pregnancy . Avoid in asthma, bradycardia, heart block and decompensated heart failure .
Nicardipine	Start 5 mg/h , increase 2.5 mg/h every 5 min , max 15 mg/h .	Smooth, predictable titration; preferred in stroke and encephalopathy where minute-to-minute control matters. No effect on heart rate, so pair with a beta-blocker in dissection .
Clevidipine	Start 1-2 mg/h , double every 2 min toward target, max about 32 mg/h .	Shortest-acting of the dihydropyridines and the most truly titratable. Lipid emulsion , so watch triglycerides and count the calories in prolonged infusions.
Esmolol	Load 500-1000 mcg/kg over 1 min , then 50 mcg/kg/min , titrate to max 200 mcg/kg/min .	Half-life about 9 min , which makes it ideal in dissection and in anyone in whom you may need to stop quickly.
Nitroglycerin	5-200 mcg/min . In SCAPE, high doses well above the anginal range are needed.	Pulmonary edema, SCAPE and ACS . Venodilator at low dose, arterial at high dose. Tachyphylaxis within 24 h . Avoid with recent PDE5 inhibitor use.
Hydralazine	5-10 mg IV over 2 min, repeat q20-40 min .	Common in pregnancy . Elsewhere a poor choice: unpredictable, long-acting and not titratable , with reflex tachycardia.
Nitroprusside	0.3 mcg/kg/min , titrate; avoid above 2-4 mcg/kg/min or beyond 24-48 h.	Specialist, refractory use only. Cyanide and thiocyanate toxicity , especially in renal or hepatic failure, and it raises intracranial pressure , so avoid it in neurologic emergencies.

SPECIAL SCENARIOS *each one has a drug that is actively dangerous*

PREECLAMPSIA AND ECLAMPSIA

Labetalol, IV hydralazine or oral nifedipine, plus **magnesium sulfate** for seizure prophylaxis and treatment. **Definitive treatment is delivery**. **Avoid ACE inhibitors and ARBs**, which are fetotoxic.

PHEOCHROMOCYTOMA

Alpha-blockade first (phentolamine), and never a beta-blocker alone. Blocking beta-2 mediated vasodilation while alpha stimulation continues produces **unopposed alpha vasoconstriction and a hypertensive crisis**. The same trap applies to **MAO-inhibitor and tyramine reactions**.

COCAINE AND STIMULANTS

Benzodiazepines first, since they treat the sympathetic drive that is causing everything. Add **phentolamine or nitroglycerin** if the pressure persists. **Avoid pure beta-blockers** for the same unopposed-alpha reason.

PEARLS & PITFALLS

- Organ damage, not the number, defines an emergency.** A very high BP with no acute damage is urgency, and IV therapy harms it.
- Look at the fundi and the ECG** before deciding which one you are treating.
- Workup is a search for damage:** ECG, troponin, BMP against baseline, urinalysis for protein, blood and casts, and a pregnancy test in anyone of childbearing potential.
- In dissection, the beta-blocker goes in first.** A vasodilator alone propagates the tear.
- Stroke gets the opposite target to everything else.** Leave the pressure alone unless you are lysing.
- Use an arterial line** for any true emergency on an infusion, and recheck the neurologic exam and urine output hourly. Falling output or new confusion means you dropped it too far, too fast.

References: 2025 AHA/ACC guideline for the prevention, detection, evaluation and management of high blood pressure in adults · AHA / ASA guidelines on spontaneous intracerebral hemorrhage and on acute ischemic stroke · ACC / AHA guideline on aortic disease · ACOG guidance on gestational hypertension and preeclampsia.

Educational aid; verify doses against local protocol, renal function, and patient factors.





Alcohol Withdrawal

From tremor to delirium tremens · treat early, dose to symptoms, give thiamine first

Thiamine
before any glucose

THE TIMELINE FROM THE LAST DRINK *knowing where the patient sits tells you what is coming next*

Syndrome	From last drink	What it looks like, and what it means
Minor withdrawal	6 to 24 h	Tremor, anxiety, sweating, nausea, insomnia, mild tachycardia and hypertension. The window in which treatment is easy and prevents everything below.
Withdrawal seizures	12 to 48 h	Generalized tonic-clonic, usually brief and self-limiting , often a single seizure or a short cluster. A third of untreated patients then progress to delirium tremens , so a seizure mandates escalation rather than reassurance. Focal seizures or a prolonged postictal state means image the head.
Alcoholic hallucinosis	12 to 48 h	Visual or tactile hallucinations with an intact sensorium: the patient knows where they are and that the hallucinations are not real. That preserved orientation is what separates it from DT , and it is not by itself a marker of severity.
Delirium tremens	48 to 96 h	Global confusion and clouded consciousness with severe autonomic instability: fever, marked tachycardia, hypertension, diaphoresis. A medical emergency with meaningful mortality even when treated , and much higher when it is not. Belongs in an ICU.

FIRST MOVES *two of these are done before you write for a single benzodiazepine*

THIAMINE BEFORE GLUCOSE

Give IV thiamine before any glucose-containing fluid. Thiamine is the cofactor for the enzymes that metabolize glucose, so a glucose load in a depleted patient consumes the last of it and can **precipitate Wernicke encephalopathy**. Use **500 mg IV three times daily for 2 to 3 days** if Wernicke is suspected, and 100 to 250 mg daily as prophylaxis. **Wernicke's triad of confusion, ophthalmoplegia and ataxia is complete in only a minority**, so treat on suspicion.

REPLACE AND EXCLUDE

Replace **magnesium, potassium and phosphate**, and watch for **refeeding syndrome** once nutrition restarts. Then **exclude the mimics**, because they are common in exactly this population and all present as agitation: **infection, head injury with subdural hematoma, GI bleeding, hepatic encephalopathy, hypoglycemia, and other drug withdrawal**. **Anchoring on withdrawal is how the subdural gets missed**.

PREDICT WHO WILL GET SEVERE

The strongest predictors are **a previous seizure or episode of DT**, a high blood alcohol level while appearing sober, prolonged heavy drinking, and older age with comorbidity. **PAWSS** can formalize the prediction on admission and identify who needs prophylaxis rather than watchful waiting.

BENZODIAZEPINES, DOSED TO SYMPTOMS *the mainstay, because they are cross-tolerant with alcohol at the same GABA-A receptor*

- 1 **Use a symptom-triggered protocol.** Dosing to **CIWA-Ar** rather than a fixed schedule gives **less total benzodiazepine and shorter treatment** without more complications. **The catch is that CIWA requires a communicative patient:** it is invalid in the intubated, the delirious, and anyone who cannot report nausea and anxiety, where it must be replaced by a sedation-scale or front-loaded protocol.
- 2 **Pick by liver function.** **Diazepam or chlordiazepoxide** are long-acting, and their active metabolites give a self-tapering effect that smooths the course. **Lorazepam is preferred in cirrhosis, in the elderly, and in anyone with respiratory compromise**, because it is glucuronidated rather than oxidized and does not accumulate in liver disease.
- 3 **Escalate fast and front-load in severe cases.** Give **repeated doses at short intervals until the patient is calm and lightly sedated**, then let the long half-life do the tapering. **Under-dosing early is the commonest error:** it lets the syndrome escalate into DT, which then needs vastly more drug.
- 4 **Treat and prevent seizures with benzodiazepines, not phenytoin.** **Phenytoin does not work here**, because withdrawal seizures arise from GABA-A downregulation and NMDA upregulation rather than a cortical focus. **Levetiracetam is equally inappropriate as monotherapy.** A benzodiazepine treats the underlying receptor problem and is both the treatment and the prophylaxis.

DELIRIUM TREMENS AND BENZODIAZEPINE RESISTANCE

when the requirement climbs, change strategy rather than repeating the same dose

MANAGE DT IN AN ICU

Escalating IV benzodiazepines to light sedation remain first-line. Add **aggressive fluid and electrolyte replacement, continued thiamine, airway monitoring, and treatment of hyperthermia**, which is a marker of severity. **Look again for a concurrent infection or bleed**, since DT and sepsis frequently coexist.

RECOGNIZE RESISTANCE

Requirements beyond roughly 200 mg of diazepam-equivalent in the first few hours define benzodiazepine resistance, caused by profound receptor downregulation. **Add phenobarbital**, which acts at a different site on GABA-A, **prolongs channel opening independently of an endogenous ligand, and also antagonizes glutamate**. It works where more benzodiazepine will not.

ADJUNCTS ARE NOT SUBSTITUTES

Dexmedetomidine and propofol control the autonomic drive and the agitation, but neither is anticonvulsant and neither treats the receptor problem. Never use them as monotherapy: keep a benzodiazepine or phenobarbital on board, or you will suppress the visible signs while the patient remains at risk of seizing.

TYPICAL STARTING DOSES

Diazepam 10 to 20 mg IV or PO, repeated every 5 to 10 min until lightly sedated. **Chlordiazepoxide 25 to 100 mg PO**, tapering over days. **Lorazepam 2 to 4 mg IV or PO** when the liver is the concern. **Phenobarbital** is given as a loading dose then titrated, ideally under a defined institutional protocol. **Titrate to the patient, not to the table:** requirements vary several-fold.

MONITOR FOR OVER-SEDATION TOO

The target is **calm and rousable, not unconscious**. Watch the respiratory rate and consciousness at each dose, particularly in **cirrhosis, the elderly, and anyone who has also taken opioids**. **Long-acting agents accumulate**, so the patient who looks right at hour 6 can be over-sedated at hour 18.

PEARLS & PITFALLS

- **Thiamine before glucose, every time.** A glucose load in a depleted patient can precipitate Wernicke encephalopathy.
- **Hallucinations with a clear sensorium are hallucinosis, not DT.** Confusion is what defines DT.
- **Do not treat withdrawal seizures with phenytoin.** It does not work on this mechanism.
- **CIWA is invalid in a patient who cannot talk to you.** Use a sedation scale instead.
- **Look for the subdural, the infection and the GI bleed.** Agitation is not proof of withdrawal.
- **Start relapse prevention before discharge:** naltrexone or acamprosate plus counseling, which is where the long-term benefit lies.





Blood Transfusion

Thresholds and products · Restrictive beats liberal, and one unit at a time beats two

Hb < 7 g/dL
most stable inpatients

RED CELLS — THE THRESHOLD *a lower trigger is not a compromise, it is the better outcome*

THE DEFAULT

Transfuse at a hemoglobin below 7 g/dL in the hemodynamically stable hospitalized adult, including in critical care. **Restrictive strategies match or beat liberal ones** across a wide range of populations while using far less blood, and every avoided unit avoids its risks.

ONE UNIT, THEN REASSESS

One unit raises the hemoglobin by roughly 1 g/dL. Prescribe a **single unit and recheck** rather than reflexively ordering two — the second unit is frequently unnecessary. **Transfuse for symptoms and physiology, not to reach a round number.**

WHEN NOT TO TRANSFUSE

Chronic deficiency anemia is not a transfusion indication in a stable patient. Iron, B12 or folate deficiency is corrected with replacement, and the patient has usually compensated. **Transfusing instead delays the diagnosis and exposes them to risk for no benefit.**

THE EXCEPTIONS TO 7 *a few groups have a higher trigger*

Population	Threshold and rationale
Stable inpatients, including critical care	7 g/dL. The evidence base is broad and consistent, and includes septic shock and gastrointestinal bleeding.
Cardiac surgery	7.5 g/dL. A specific, slightly higher trigger — not the same as the 8 used for the groups below, which is a distinction routinely blurred at the bedside.
Orthopedic surgery, or pre-existing cardiovascular disease	8 g/dL. Reduced capacity to compensate for anemia justifies the higher trigger.
Acute myocardial infarction	Genuinely unsettled. Trial evidence has not shown a clear benefit for a liberal strategy, but the point estimates have favored it, and many now accept a higher trigger around 8 to 10 g/dL in active ischemia. Discuss with cardiology rather than applying 7 reflexively.
Active major hemorrhage	Thresholds do not apply. Activate the major hemorrhage protocol and give balanced ratios of red cells, plasma and platelets while achieving surgical or radiological source control. The hemoglobin lags badly during acute bleeding and must not be used to decide.

THE OTHER PRODUCTS *each has a distinct indication, and each is over-used*

Product	When it is indicated
Platelets	Prophylactically at 10 ×10⁹/L or below in hypoproliferative thrombocytopenia, and below 50 for major elective non-neuraxial surgery. Higher targets are used by convention for neurosurgery, ocular surgery and neuraxial procedures . Routine prophylaxis is not indicated in non-thrombocytopenic patients having cardiac surgery.
When platelets harm	Avoid in TTP, HIT and hemolytic uremic syndrome unless there is life-threatening bleeding — adding platelets fuels the thrombotic process. Look at the film before you transfuse platelets: schistocytes change the answer completely.
Modified products	Irradiated components for Hodgkin lymphoma, after stem cell transplant, and on purine analogues, to prevent transfusion-associated graft-versus-host disease. Anti-D is required for RhD-negative women of childbearing potential exposed to RhD-positive components.

HOW MUCH, AND WHAT IT SHOULD DO *know the expected increment, because a failed one is a finding*

Product	Dose	Expected effect
Red cells	1 unit , then reassess	Raises Hb by about 1 g/dL (hematocrit ~3%) in a 70 kg adult who is not bleeding. A smaller rise than expected means ongoing loss or hemolysis , not an inadequate dose.
Platelets	1 apheresis unit (equivalent to a pool of 4-6 whole-blood units)	Raises the count 30-50 ×10⁹/L . A flat post-transfusion count suggests refractoriness from consumption, splenomegaly, sepsis or HLA alloimmunization.
Fresh frozen plasma	10-15 mL/kg , roughly 3-4 units in an adult	Small doses achieve nothing. For warfarin reversal, 4-factor PCC is faster, smaller in volume and preferred ; plasma is for multi-factor deficiency with bleeding.
Cryoprecipitate	1 pool (about 5 units)	Raises fibrinogen by roughly 50-100 mg/dL . Recheck the fibrinogen rather than assuming.

GIVING IT SAFELY *the fatal errors happen at the bedside, not in the laboratory*

- Consent and document the indication.** Record why, what and how much, and tell the patient what to report. **An unexplained transfusion on a drug chart is a governance failure and a clinical one.**
- Positive patient identification at the bedside, at sampling and at administration.** **Wrong blood in tube is the leading route to an ABO-incompatible transfusion**, and it is a bedside identification error, not a laboratory one. **Label the sample at the patient, never in advance and never at the desk.**
- Observations before starting, at 15 minutes, and at completion**, with the patient **in view during the first 15 minutes**, when severe reactions declare themselves. **Complete each unit within 4 hours** of leaving controlled storage.
- If a reaction occurs, stop the transfusion first** and keep the line open with saline. Then assess, recheck identity against the unit, and inform the laboratory. **Do not restart the same unit.**
- Distinguish overload from lung injury.** **TACO** gives hypertension, a raised JVP and a raised natriuretic peptide, and **responds to diuresis** — it is the commonest cause of transfusion-related death and is preventable by slowing the rate and reviewing the volume. **TRALI** gives hypoxemia with bilateral infiltrates **within 6 hours**, normal filling pressures, and **does not respond to diuretics**.

PEARLS & PITFALLS

- Prescribe one unit and reassess.** The reflex two-unit order is the single commonest over-transfusion.
- Cardiac surgery is 7.5, not 8.** Orthopedic surgery and pre-existing cardiovascular disease are the 8 g/dL groups.
- Iron deficiency needs iron, not blood**, in a stable patient — and blood obscures the workup for weeks.
- Label the sample at the bedside.** Wrong blood in tube is how ABO-incompatible transfusions actually happen.
- Do not give platelets in TTP or HIT** unless the bleeding is life-threatening. Check the film first.
- Think TACO before TRALI.** Circulatory overload is far commoner, is the leading transfusion death, and is largely preventable.





Anticoagulation Reversal

Warfarin · DOACs · heparins · reverse only for bleeding that actually threatens the patient

Which drug?

and when was the last dose

EVERY MAJOR BLEED · DO THIS FIRST reversal is an adjunct; none of it substitutes for stopping the bleeding

STABILIZE AND CONTROL THE SOURCE

ABC, direct pressure, packing. Urgent surgery, endoscopy or interventional radiology is the definitive treatment. A reversed patient who is still bleeding from an uncontrolled source will simply bleed again.

TRANSFUSE TO TARGETS

Platelets above 50, or above 100 for intracranial bleeding · fibrinogen above 1.5 g/L · correct ionized calcium and temperature · TXA 1 g. Cold, acidotic, hypocalcemic blood does not clot whatever reversal agent you give.

ASSESS THE DRUG

Which agent, what dose, the exact time of the last dose, and renal and hepatic function. Time and renal function predict the remaining drug effect better than any coagulation test, particularly for dabigatran, which is renally cleared. Send a drug-specific assay if available.

WARFARIN vitamin K antagonist: give both PCC and vitamin K, for different reasons

Situation	What to give
Life-threatening or critical-site bleed	4-factor PCC 25 to 50 U/kg by INR and weight, PLUS IV vitamin K 5 to 10 mg. Both are needed and they act on different timescales: PCC replaces the missing factors within minutes but wears off in hours, while vitamin K restores the liver's own synthesis over 12 to 24 h. Give PCC alone and the INR rebounds once it is cleared.
High INR, no bleeding	Hold warfarin, with or without oral vitamin K 1 to 5 mg. Do not reverse a number. Reversing an asymptomatic high INR exposes the patient to thrombosis for no benefit, and makes re-anticoagulation harder.
Why not FFP	Only if PCC is unavailable. FFP is slower to prepare, needs thawing and cross-matching, requires a large volume that risks TACO, and corrects the INR less completely.

DOACs specific agent first, PCC if it is not available

Drug	Specific reversal	Dose, and the alternative
Dabigatran direct thrombin inhibitor	Idarucizumab	5 g IV (two 2.5 g vials). Works within minutes. Dabigatran is also dialyzable, unlike the factor Xa inhibitors, which is a useful backup in renal failure.
Apixaban Rivaroxaban	4F-PCC (off-label)	50 U/kg (some protocols use a fixed 2,000 units). Andexanet alfa was withdrawn from the US market in December 2025 after the FDA judged its thrombotic risk to outweigh its benefit.
Edoxaban	4F-PCC (off-label)	4F-PCC 50 U/kg.
Why andexanet is gone	ANNEXA-I, NEJM 2024 studied andexanet in intracranial hemorrhage on a factor Xa inhibitor. It improved hemostatic efficacy but caused more thrombotic events, including ischemic stroke, and was stopped early. Reversal is not free: you are trading a bleeding risk for a clotting one, so reserve it for genuinely life-threatening bleeding.	

HEPARINS AND THE LABS protamine works well for one and poorly for the other

PROTAMINE

Unfractionated heparin: 1 mg per 100 units given in the last 2 to 3 h, to a maximum of 50 mg. Near-complete reversal. Enoxaparin: 1 mg per 1 mg if within 8 h, but only about 60 to 75% reversal, because protamine binds the long chains and LMWH is mostly short ones. Fondaparinux has no antidote; consider activated PCC in extremis. Give protamine slowly: rapid infusion causes hypotension, bradycardia and anaphylactoid reactions.

THE LAB TRAP

A normal PT and aPTT do NOT exclude a DOAC effect, which is the single most dangerous misreading on this page. Use drug-calibrated anti-Xa for apixaban, rivaroxaban and LMWH, and dilute thrombin time or ecarin clotting time for dabigatran. If assays are unavailable, decide on the last dose and renal function rather than on a reassuring screen.

WHEN TO REVERSE, AND WHEN TO RESTART

REVERSE FOR

Life-threatening bleeding, or bleeding into a critical site: intracranial, spinal, pericardial, airway, intraocular, or a compartment. Emergency surgery that cannot be delayed. Not for minor bleeding, and not for a raised number alone.

RESTART DELIBERATELY

Restarting is as important as reversing. The thrombotic risk of prolonged interruption is real, and a mechanical valve or recent VTE tolerates it poorly. Agree a restart date at the time of reversal, and document it, since anticoagulation stopped in an emergency is frequently never restarted.

DO NOT FORGET

Ask about antiplatelets too, which are often taken alongside and are not reversed by any of the above. Platelet transfusion does not help in antiplatelet-associated intracerebral hemorrhage and may cause harm. Check for a reversible contributor: renal failure, liver disease, or an interacting drug.

PERIPROCEDURAL INTERRUPTION most reversal questions are really planning questions asked too late

Agent	Stop before an elective procedure	Notes
DOACs	24 to 48 h for low bleeding risk; 48 to 72 h for high risk, and longer as CrCl falls, especially for dabigatran	Routine bridging is not needed and increases bleeding without reducing thrombosis. No preoperative coagulation test reliably confirms clearance, so time and renal function are the plan.
Warfarin	5 days, then check the INR	Bridge only for genuinely high thrombotic risk: mechanical mitral valve, recent VTE or stroke, or high CHA ₂ DS ₂ -VASC with prior event. BRIDGE, NEJM 2015 found bridging in AF increased major bleeding with no reduction in thromboembolism.
Heparins	LMWH: last dose 24 h before. UFH infusion: stop 4 to 6 h before	Short half-lives make these the easiest to time, which is why they were used for bridging in the first place.

PEARLS & PITFALLS

- PCC and vitamin K together for warfarin. PCC works in minutes and wears off; vitamin K holds the correction.
- A normal PT and aPTT do not exclude a DOAC. Use the last dose and renal function.
- Reverse for the bleed, not for the number. An asymptomatic high INR is held, not reversed.
- Protamine only partly reverses LMWH, and does nothing for fondaparinux.

References: ACC expert consensus decision pathway on management of bleeding in patients on oral anticoagulants · ASH guidelines on management of anticoagulant-related bleeding · Connolly et al, ANNEXA-I, NEJM 2024 · Pollack et al, RE-VERSE AD (idarucizumab), NEJM 2017 · Neurocritical Care Society reversal guidance.

Educational aid; verify doses against local protocol, renal function, and patient factors.

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