



Cardiac Arrest · ACLS

Adult advanced cardiovascular life support · compressions and early defibrillation are the treatment

Defib
early, and often

THE FIRST 60 SECONDS

Start high-quality compressions · attach the monitor or defibrillator · give oxygen · obtain IV or IO access. Everything that changes survival happens in this minute, and it is compressions and defibrillation, not drugs. **Do not delay compressions for access, an airway, or a rhythm interpretation**, and get the pads on the moment the machine arrives, because the single strongest predictor of survival in a shockable rhythm is time to first shock.

RHYTHM CHECK *every 2 minutes, and the whole algorithm forks here*

SHOCKABLE · VF or pulseless VT

1. Shock at 120 to 200 J biphasic, escalating to maximum. 2. CPR for 2 min, obtain access. 3. Rhythm check, and **shock again if still shockable**. 4. Epinephrine 1 mg after the second shock, then every 3 to 5 min. 5. Shock, CPR 2 min. 6. Amiodarone 300 mg, or lidocaine 1 to 1.5 mg/kg. 7. Repeat cycles; second dose of amiodarone 150 mg or lidocaine 0.5 to 0.75 mg/kg.

NON-SHOCKABLE · asystole or PEA

1. CPR for 2 min, obtain access. 2. Epinephrine 1 mg as soon as possible, then every 3 to 5 min. 3. **Do not shock**: there is no organized electrical activity to reset, and the attempt only interrupts compressions. 4. Rhythm check every 2 min. 5. **Actively work through the reversible causes**, since this pathway has no defibrillation to fall back on. 6. If it becomes shockable, switch pathways.

WHY EPINEPHRINE TIMING DIFFERS BETWEEN THE TWO

In a shockable rhythm, defibrillation is the definitive treatment and epinephrine is secondary, so it waits until after the second shock. In a non-shockable rhythm there is nothing to shock, so epinephrine is given as early as possible, where earlier administration is associated with better outcomes. Its role is to raise coronary and cerebral perfusion pressure during compressions, which is also why it is worthless without good compressions.

DRUGS AND ENERGY

Agent	Dose	Note
Epinephrine	1 mg IV or IO every 3 to 5 min	Both pathways. Early in non-shockable , after the second shock in shockable. There is no role for high-dose epinephrine.
Amiodarone	300 mg, then 150 mg	For refractory VF or pulseless VT , meaning after shocks have failed. Improves survival to admission; the effect on survival to discharge is less certain.
Lidocaine	1 to 1.5 mg/kg, then 0.5 to 0.75 mg/kg	An acceptable alternative to amiodarone, with broadly comparable outcomes.
Magnesium	1 to 2 g IV	Torsades de pointes only . It is not a routine arrest drug and does not help undifferentiated VF.
Calcium and bicarbonate	Only for specific indications	Not routine , and routine use is associated with worse outcomes. Reserve for hyperkalemia, calcium channel blocker overdose, and known severe acidosis .
Defibrillation	Biphasic 120 to 200 J, escalating; monophasic 360 J	Charge the defibrillator during compressions and resume immediately after the shock. Minimize the pre-shock and post-shock pause , since each second of no-flow costs coronary perfusion that takes many compressions to rebuild.

HIGH-QUALITY CPR *the only intervention on this page with an unambiguous survival benefit*

THE NUMBERS

Rate 100 to 120 per minute. Depth 5 to 6 cm (2 to 2.4 inches) with full chest recoil, since leaning prevents the venous refilling that generates the next compression's output. **Interruptions under 10 s**, and a chest compression fraction above 60%. Rotate the compressor every 2 min, because quality degrades measurably before the provider feels tired.

VENTILATION

10 breaths per minute with an advanced airway, otherwise 30:2. **Do not hyperventilate**. Excessive rate raises intrathoracic pressure and reduces venous return and cardiac output during compressions. A bag-mask or supraglottic airway is acceptable; intubation should not interrupt compressions.

CAPNOGRAPHY GUIDES YOU

EtCO₂ below 10 mmHg means compressions are not generating enough output: change the compressor, check depth and recoil, look for a cause. An abrupt rise signals ROSC, and it also confirms tube placement.

REVERSIBLE CAUSES *in PEA and asystole this is the treatment, not an afterthought*

The Hs	The Ts
Hypoxia → oxygenate, confirm the tube	Tension pneumothorax → needle or finger thoracostomy, before imaging
Hypovolemia → fluids, and blood if bleeding	Tamponade → bedside ultrasound, pericardiocentesis
Hydrogen ion (acidosis) → ventilate; bicarbonate only if severe	Toxins → the specific antidote (bicarbonate, lipid emulsion, glucagon, naloxone)
Hypo- and hyperkalemia → calcium, insulin with dextrose, or replace potassium	Thrombosis, coronary → PCI, and consider ECPR in a suitable candidate
Hypothermia → active rewarming, and continue resuscitation while cold	Thrombosis, pulmonary → thrombolysis, then continue CPR for 60 to 90 min to circulate the drug

WHEN IT WILL NOT STOP *and how the arrest ends*

REFRACTORY VF

Check pad position first and switch to anterior-posterior. Double sequential external or vector-change defibrillation improved survival to hospital discharge in DOSE-VF, NEJM 2022. Consider ECPR in a witnessed shockable arrest with a reversible cause.

AFTER ROSC

Secure the airway, get a 12-lead ECG, treat the cause, avoid hyperoxia and hypocapnia, target temperature, and admit to ICU. Do not hyperventilate the post-arrest brain: hypocapnia causes cerebral vasoconstriction. See Post-Cardiac Arrest Care, sheet #16.

WHEN TO STOP

Consider termination after adequate ACLS with persistent asystole, no reversible cause, and a sustained EtCO₂ below 10 mmHg after 20 min. Not in hypothermia, drowning, or after thrombolysis, where prolonged resuscitation is appropriate.

PEARLS & PITFALLS

- Compressions and early defibrillation save lives; drugs are secondary.
- Do not hyperventilate. It lowers venous return at the worst moment.
- Charge during compressions and minimize every pause around the shock.
- In PEA the algorithm is the H's and T's, not more epinephrine.

References: American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, adult advanced cardiovascular life support ·

Cheskes et al, DOSE-VF, NEJM 2022 · Kudenchuk et al, ALPS amiodarone versus lidocaine versus placebo, NEJM 2016 · Perkins et al, PARAMEDIC2, NEJM 2018.

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





Post-Cardiac Arrest Care

Protect the brain · prevent fever · treat the cause · delay prognosis · 2025 AHA

≤ 37.5 °C
prevent fever

TARGETS AFTER ROSC *the brain injury continues after the heart restarts, and these are how you limit it*

Parameter	Target	Why this target
SpO ₂	90 to 98% PaO ₂ 60 to 105	100% oxygen only until a saturation reads reliably, then titrate the FiO₂ down. Hyperoxia generates free radicals that worsen reperfusion injury; hypoxemia extends the ischemic one. 2020 said 92 to 98%.
PaCO ₂	35 to 45 mmHg	Normocapnia. Hypocapnia causes cerebral vasoconstriction and worsens ischemia in an already injured brain, and it is easy to produce by leaving the ventilator on arrest settings. Hypercapnia raises intracranial pressure.
MAP	At least 65 mmHg	Autoregulation is impaired after arrest, so cerebral perfusion becomes pressure-dependent . Higher MAP targets did not improve outcome in four trials. No vasopressor is proven better ; norepinephrine is the usual choice.
Temperature	One constant target, 32 to 37.5 °C	Fever after arrest is clearly harmful , and preventing it is the part of temperature control with the strongest evidence. Hold the target at least 36 h .
Glucose	140 to 180 mg/dL	Avoid below 70 and above 180: both worsen neurologic outcome. Tight control adds hypoglycemia without benefit.

IMMEDIATE PRIORITIES *the first hour after ROSC*

- 1 **Confirm ROSC and get a 12-lead ECG**, because it drives the catheter lab decision. **Repeat a tracing taken within minutes of ROSC**: early ECGs over-call STEMI. Confirm the airway with **waveform capnography**.
- 2 **Turn the oxygen down and the ventilator to normal settings**. Hyperoxia and hyperventilation are both harmful and both are the default state of a patient who has just been resuscitated.
- 3 **Support the pressure** with fluid and norepinephrine, and **treat the cause**: work back through the H's and T's, since the thing that caused the arrest is usually still present.
- 4 **Not following commands: start temperature control, admit to ICU, and get an EEG promptly**. Treat clinical and EEG seizures; **no prophylaxis**. **Do not treat myoclonus that has no EEG correlate** (new 2025): the sedatives needed drop the pressure and cloud the exam.

TEMPERATURE CONTROL *the target changed: prevent fever rather than chase deep cooling*

WHAT TO DO NOW

Every adult who does not follow commands, any rhythm, any location: **one constant target between 32 and 37.5 °C, held with a feedback device for at least 36 h** (2020 said 24 h; BOX, NEJM 2023 found 36 h no worse than 72 h, and 72 h remains acceptable). **Routine cooling to 33 °C is no longer the default**: TTM2, NEJM 2021 found **no mortality or functional benefit over normothermia with fever prevention**, and more arrhythmia.

MANAGE SHIVERING

Shivering defeats temperature control and raises metabolic demand at exactly the wrong time. Escalate through **analgesia and sedation, magnesium, surface counter-warming, and neuromuscular blockade** if needed. **Paralysis masks seizures**, so pair it with EEG monitoring.

AND AVOID REBOUND

Rewarm no faster than 0.5 °C per hour (trials used 0.25 to 0.5), and **keep preventing fever afterwards**: rebound hyperthermia is common and is associated with worse outcomes. **No rapid cold IV saline before hospital arrival**.

CORONARY ANGIOGRAPHY & SUPPORT *immediate for some, and safely deferred for the rest*

Situation	What to do (2025 AHA)
Go immediately	Persistent ST elevation, regardless of coma (Class 1). Without ST elevation: cardiogenic shock, recurrent ventricular arrhythmia, or significant ongoing ischemia (Class 2a).
Defer safely	Stable, comatose, no ST elevation: emergency angiography is not recommended (Class 3, no benefit). COACT, NEJM 2019 and TOMAHAWK, NEJM 2021 found no survival benefit . Angiography before discharge if a cardiac cause is suspected (Class 1).
In shock	Multivessel disease: open the culprit artery only (CULPRIT-SHOCK, NEJM 2017). Temporary mechanical support is not routine (Class 3); consider it only in highly selected refractory shock (Class 2b).
Look elsewhere too	Head-to-pelvis CT and echo or POCUS may be reasonable (Class 2b) when the cause is unclear: intracranial hemorrhage, massive PE, dissection, tamponade, and CPR injuries.

NEUROPROGNOSTICATION *multimodal, never one test, and never early*

THE RULE

Wait at least 72 h after return to normothermia AND after sedatives have cleared (cold slows their clearance). **No finding counts alone** (Class 1); ERC / ESICM asks for at least two concordant predictors. The reason for the caution is the **self-fulfilling prophecy**: withdrawing care on a single early finding produces the poor outcome it predicted.

Modality	Supports a poor outcome (each Class 2b, never alone)	Timing
Clinical exam	Bilaterally absent pupillary or corneal reflexes . An absent or extensor motor response, and myoclonus alone, are of uncertain value	≥ 72 h, off sedation
SSEP	Bilaterally absent N20 cortical response , the modality least affected by sedation	≥ 48 h
NSE or NfL	Very high values ; NfL (added 2025) is more sensitive. No universal cutoff. Hemolysis falsely elevates NSE	Within 72 h
EEG	Burst suppression off sedation, or persisting status epilepticus . Do not use absent reactivity	≥ 72 h
CT or MRI	Reduced gray-white ratio on CT; extensive restricted diffusion on MRI	MRI 2 to 7 d
Favorable signs	New in 2025 : withdrawal or better motor response, normal NSE, a continuous EEG background without discharges, no restricted diffusion on MRI. Reasons to continue full support.	

PEARLS & PITFALLS

- **Prevent fever for at least 36 h**. That, not deep cooling, is what the evidence supports.
- **Never prognosticate before 72 h** after normothermia, off sedation, or on one finding.

References: 2025 AHA Guidelines for CPR and ECC, Part 11: Post-Cardiac Arrest Care, *Circulation* 2025 · Dankiewicz et al, TTM2, *NEJM* 2021 · Hassager et al, BOX, *NEJM* 2023 · Lemkes et al, COACT, *NEJM* 2019 · Desch et al, TOMAHAWK, *NEJM* 2021 · Thiele et al, CULPRIT-SHOCK, *NEJM* 2017.

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





Shock Differentiation

Four mechanisms · four hemodynamic fingerprints · the type decides the first move

Warm vs cold
the 10-second triage

THE FOUR TYPES *shock is inadequate tissue perfusion, and only four things cause it*

Type	Mechanism	Causes, and the thing that defines it
Distributive <i>commonest</i>	Vasodilation with maldistribution of flow	Sepsis above all, then anaphylaxis, neurogenic and adrenal crisis . The output is normal or high, but the resistance has collapsed and oxygen is not extracted where it is needed.
Cardiogenic	Pump failure	MI , myocarditis, arrhythmia, acute valve failure, decompensated chronic heart failure. The ventricle cannot generate output against a normal load.
Hypovolemic	Loss of circulating volume	Hemorrhage , and non-hemorrhagic losses through the GI tract, kidney, skin or into a third space. There is not enough volume to fill the ventricle.
Obstructive	Mechanical block to filling or ejection	Tension pneumothorax, tamponade, massive PE , and auto-PEEP in a ventilated patient. The pump and the volume are both fine , which is why fluid and inotropes fail and only relieving the obstruction works.

HEMODYNAMIC FINGERPRINTS *learn the pattern and the diagnosis usually falls out*

Parameter	Distributive	Cardiogenic	Hypovolemic	Obstructive
Preload (CVP)	↓ / N	↑	↓	↑
Wedge (PCWP)	↓ / N	↑	↓	↓ *
Cardiac output	↑ / N	↓	↓	↓
SVR (afterload)	↓	↑	↑	↑
ScvO ₂	↑	↓	↓	↓

READING THE TABLE

* **Tamponade raises the wedge**, unlike PE and tension pneumothorax, because it equalizes intracardiac pressures. **The ScvO₂ row is the most useful single line**: it is **high in distributive shock**, where flow bypasses the tissues and oxygen is not extracted, and **low in the other three**, where a struggling circulation forces the tissues to extract more. **Normal values**: CI 2.5 to 4.0, SVR 800 to 1200, CVP 2 to 8, PCWP 6 to 12, ScvO₂ at least 70%.

THE BEDSIDE PHENOTYPE *you can halve the differential before any monitor is attached*

WARM

Flash capillary refill, bounding pulses, warm peripheries, wide pulse pressure. The vessels are dilated, so this is **distributive**: early sepsis, anaphylaxis, neurogenic or adrenal.

COLD

Cool mottled skin, capillary refill beyond 3 s, thready pulse, narrow pulse pressure. The vessels are clamped down to defend the pressure, so this is **cardiogenic, hypovolemic or obstructive**. **A narrow pulse pressure is an early sign** and often precedes hypotension.

MIXED IS COMMON

Real patients have more than one mechanism: septic shock with a septic cardiomyopathy, hemorrhage with a tension pneumothorax, cardiogenic shock that becomes vasoplegic. **Reassess the phenotype after every intervention**, since a patient who stops responding has usually acquired a second mechanism.

BEDSIDE WORKUP *phenotype in minutes with ultrasound and a leg raise*

Tool	What it answers
POCUS, the RUSH exam	Pump : LV function, RV size and strain, pericardial effusion. Tank : IVC diameter and collapsibility, lung B-lines for pulmonary edema, free fluid in the abdomen. Pipes : aorta for dissection or aneurysm, and the legs for DVT. This single exam separates all four types in a few minutes and finds tamponade and pneumothorax at the same time.
Fluid responsiveness	Passive leg raise with a measured stroke-volume response , or dynamic indices such as stroke-volume or pulse-pressure variation above 12 to 13%. Give fluid only if the patient is actually responsive : static measures like CVP predict poorly, and about half of hemodynamically unstable patients are not fluid responsive at all, so a reflex bolus simply causes edema.
Perfusion markers	Serial lactate and its clearance, ScvO₂, urine output, mentation and capillary refill . Target a MAP of at least 65 , individualized upward in chronic hypertension. Capillary refill guided resuscitation performs at least as well as lactate and is available everywhere.

FIRST MOVES BY TYPE *the wrong first move is actively harmful in three of the four*

DISTRIBUTIVE

Fluids plus early norepinephrine. Septic: **antibiotics within the hour and source control**. Anaphylaxis: **IM epinephrine 0.3 to 0.5 mg** into the thigh. **Do not delay the vasopressor** waiting to finish a fluid target.

CARDIOGENIC

Avoid fluid boluses, which worsen congestion. **Norepinephrine for pressure, dobutamine for output**, and **revascularize the culprit artery** in MI. Escalate to mechanical support early rather than late.

HYPOVOLEMIC AND OBSTRUCTIVE

Hypovolemic: stop the loss, balanced crystalloid, and in hemorrhage **blood in balanced ratio plus TXA and definitive hemostasis**, not crystalloid. **Obstructive**: **decompress**. Needle or finger thoracostomy, pericardiocentesis, thrombolysis for massive PE. **Fluids and pressors are only a bridge to the decompression**.

PEARLS & PITFALLS

- **Starting doses when you commit to a pressor**: norepinephrine 0.05-0.1 mcg/kg/min titrated to a MAP of 65, **vasopressin 0.03 units/min as a fixed non-titrated dose** added second, and **dobutamine 2.5-5 mcg/kg/min** when the problem is output rather than tone. **Start peripherally rather than waiting for central access**.
- **Warm versus cold splits the differential in ten seconds**, before any monitor.
- **A high ScvO₂ points to distributive shock**; the other three drive it down.
- **Do not give fluid to a patient who is not fluid responsive**. Half of unstable patients are not.
- **Obstructive shock does not respond to drugs**. Find the obstruction and relieve it.
- **Reassess the phenotype after every intervention**. A patient who stops responding has usually acquired a second mechanism.





Tachyarrhythmias

The tachycardia-with-a-pulse algorithm · unstable means electricity, stable means read the QRS

Unstable?
cardiovert

STEP 1 · IS THE PATIENT UNSTABLE? *this question comes before any rhythm diagnosis*

UNSTABLE MEANS ANY OF

Hypotension or shock · ischemic chest pain · acute heart failure · altered mental status, caused by the rate. Rate-related instability generally needs a rate above 150: below that, look for another explanation rather than blaming the rhythm. **The judgment is whether the rate is causing the instability or merely accompanying it,** because cardioverting a septic patient at 140 treats the wrong problem and rarely holds.

THE ENERGY, AND SYNC OR NOT

Synchronized cardioversion: narrow regular (SVT) **50 to 100 J** · narrow irregular (AF) **120 to 200 J** · wide regular (VT) **100 J. Wide and irregular is treated as VF: use UNSYNCHRONIZED defibrillation.** **Why:** the machine cannot reliably find an R wave in a chaotic polymorphic rhythm, and attempting to sync simply means it never discharges. **Sedate if there is time.**

STEP 2 · STABLE? READ THE QRS *narrow under 0.12 s versus wide at or above 0.12 s, then regular or irregular*

Pattern	Likely rhythm	Treatment
Narrow & regular	SVT (AVNRT or AVRT), or atrial flutter with fixed block	Vagal maneuvers first , using the modified Valsalva (strain, then lie flat with legs raised), which is markedly more effective than the standard technique. Then adenosine . If refractory, diltiazem or a beta-blocker.
Narrow & irregular	AF, flutter with variable block, or multifocal atrial tachycardia	Rate control with diltiazem or a beta-blocker , targeting under 110. Anticoagulate by CHA ₂ DS ₂ -VASC. MAT responds to treating the underlying lung disease, not to cardioversion.
Wide & regular	VT until proven otherwise	Assume VT, especially with structural heart disease or prior infarct , because treating VT as SVT is far more dangerous than the reverse. Amiodarone 150 mg over 10 min , or procainamide. Adenosine is reasonable only if regular and monomorphic, as a diagnostic maneuver.
Wide & irregular	Pre-excited AF, AF with aberrancy, or torsades	Get expert help. Pre-excited AF: no AV-nodal blockers (below). Torsades: magnesium 2 g. Defibrillate if unstable.

THE TWO THAT KILL IF YOU TREAT THEM NORMALLY

PRE-EXCITED AF (WPW)

Irregular, wide and often very fast, with beat-to-beat variation in QRS width. Avoid every AV-nodal blocker: adenosine, verapamil, diltiazem, beta-blockers and digoxin. **Why they kill:** blocking the AV node removes the only competing pathway, so conduction shifts entirely to the accessory pathway, which has **no decremental properties**. The ventricular rate accelerates and degenerates into **ventricular fibrillation**. **Use procainamide, or synchronized cardioversion**, which is the safest choice if you are unsure.

TORSADES DE POINTES

Polymorphic VT on a background of long QT. Magnesium 2 g IV regardless of the serum magnesium level, because it stabilizes the membrane rather than correcting a deficiency. **Correct potassium and magnesium, and stop every QT-prolonging drug.** If it recurs, **increase the heart rate** with overdrive pacing or **isoproterenol**, since a faster rate shortens the QT and suppresses the pauses that trigger it. **Defibrillate if pulseless.**

DRUG DOSES *adult IV*

Drug	Dose and note
Adenosine	6 mg rapid push into a large proximal vein with an immediate flush, then 12 mg if needed. Its half-life is under 10 seconds, so a slow push or a distal line simply fails. Warn the patient about the brief awful chest sensation. Avoid in pre-excited AF and severe asthma. It is also diagnostic: transient AV block unmasks flutter waves even when it does not terminate the rhythm.
Diltiazem	0.25 mg/kg (about 15 to 20 mg) over 2 min , then an infusion. Rate control for AF and flutter. Avoid in HFrEF , where its negative inotropy can precipitate shock, and in pre-excitation.
Metoprolol	2.5 to 5 mg IV over 2 min, repeated every 5 min to a maximum of 15 mg. Preferred when the driver is adrenergic. Avoid in decompensated heart failure and severe asthma.
Amiodarone	150 mg over 10 min, then 1 mg/min for 6 h. Useful in VT and when heart failure limits the alternatives. Treat it as chemical cardioversion in AF , so the same anticoagulation rules apply.
Magnesium	2 g IV over 10 to 15 min for torsades, irrespective of the level.

BEFORE YOU TREAT, AND AFTERWARDS

TREAT THE DRIVER

Sinus tachycardia is a response, not a diagnosis. Before slowing any fast rhythm, look for **sepsis, hypovolemia, pain, anemia, PE, hyperthyroidism, alcohol withdrawal and drugs.** **Blunting a compensatory tachycardia removes the patient's own compensation** and can precipitate collapse.

AF SPECIFICS

Lenient rate control under 110 is as good as strict. **Cardiovert without prior anticoagulation only if onset is clearly under 48 h;** otherwise **3 weeks of anticoagulation or a TEE** to exclude atrial thrombus first, then **4 weeks of anticoagulation afterwards** because the stunned atrium is at highest risk in the days after.

GET A 12-LEAD, AND KEEP IT

Record a 12-lead during the tachycardia and after it terminates. The rhythm strip after adenosine is often the only diagnostic evidence anyone will ever have, and it determines the referral for ablation. **Correct potassium and magnesium in everyone.**

PEARLS & PITFALLS

- **Procainamide dose**, since it is the drug named for pre-excited AF: **20-50 mg/min IV** until the arrhythmia stops, hypotension develops, the **QRS widens by more than 50%**, or a **maximum of 17 mg/kg** is reached. Stop at whichever comes first.
- **Adenosine fails on technique more often than on pharmacology.** Give it into a **large proximal vein with an immediate 20 mL flush and the arm elevated:** its half-life is seconds. Warn the patient about chest tightness and a sense of doom, and **run a continuous rhythm strip**, which is diagnostic even when it does not convert.
- **Unstable and rate-related means electricity, not drugs.** Sedate if there is time.
- **Wide and regular is VT until proven otherwise**, particularly with prior infarct.

- **Irregular and wide means no AV-nodal blocker.** Procainamide or shock.
- **Adenosine needs a big vein, a fast push and a flush.** Its half-life is seconds.





Bradycardia & Heart Block

AV conduction disease · Is it symptomatic? Is it reversible? Is it infranodal?

SITE DECIDES FATE
nodal vs infranodal

CLASSIFY *where the block sits predicts whether it will progress*

Degree	ECG	Site & behavior
First degree	PR > 200 ms, every P conducts	Usually nodal . Benign in isolation. Marked delay (> 300 ms) can cause pseudo-pacemaker symptoms.
Mobitz I (Wenckebach)	Progressive PR lengthening, then a dropped beat; grouped beating	Nodal (AV node) . Often vagal or drug-related, frequently benign, rarely progresses. Improves with exercise or atropine.
Mobitz II	Constant PR , then a sudden dropped beat; often wide QRS	Infranodal (His-Purkinje) . Unstable, progresses to complete block without warning. Escape rhythm is slow and unreliable .
2:1 block	Every other P conducts — cannot classify by PR pattern alone	Ambiguous. Narrow QRS + PR long favors nodal; wide QRS favors infranodal. Exercise or atropine improves nodal, worsens infranodal .
Third degree (complete)	AV dissociation, atrial rate > ventricular, regular P-P and R-R	Escape site sets the risk: junctional (narrow, 40–60) is more stable than ventricular (wide, 20–40), which is unreliable.

ACUTE MANAGEMENT *treat the patient, not the number*

- 1 **Is the patient unstable?** Hypotension, altered mentation, ischemic chest pain, or acute heart failure. A rate of 38 in a well athlete needs nothing; a rate of 48 with shock needs everything.
- 2 **Atropine 1 mg IV**, repeat every 3–5 min to a maximum of 3 mg. Works on the **AV node** — so it helps nodal block but is **often ineffective in Mobitz II or complete block**, and by speeding the atria it can **worsen** infranodal block.
- 3 **Transcutaneous pacing** if atropine fails — sedate, set rate 60–80, increase output until **electrical and mechanical capture** (confirm with a pulse, not the monitor).
- 4 **Chronotropic infusion** as a bridge: dopamine 5–20 mcg/kg/min or epinephrine 2–10 mcg/min.
- 5 **Transvenous pacing** for persistent unstable bradycardia, then decide on a permanent device once reversible causes are excluded.

FIND THE REVERSIBLE CAUSE *before anyone implants a device*

DRUGS — check the chart first
Beta-blockers, non-dihydropyridine **calcium channel blockers**, digoxin, amiodarone, clonidine, acetylcholinesterase inhibitors. **Glucagon** for beta-blocker toxicity, **IV calcium** for CCB toxicity, **digoxin Fab** for digoxin.

METABOLIC & ISCHEMIC
Hyperkalemia (widening QRS, peaked T) · **hypothyroidism** · hypothermia · **inferior MI** (RCA supplies the AV node; often transient and atropine-responsive) · **anterior MI** with block, which signals extensive infarction and is ominous.

INFILTRATIVE & INFECTIOUS
Lyme carditis — young patient, endemic exposure, high-grade block that usually **resolves with antibiotics**, so do not implant early. Also sarcoidosis, amyloidosis, endocarditis with **root abscess**, myocarditis, post-TAVR or post-surgical.

PERMANENT PACING *symptoms that match, or a block that will not recover*

Indication	Detail
Symptomatic bradycardia	Pace when symptoms correlate temporally with the bradycardia and no reversible cause is found. Correlation is the whole game.
Mobitz II, high-grade, or complete block	Permanent pacing when not attributable to a reversible cause — regardless of symptoms, because progression is unpredictable.
Do not pace	Sleep-related sinus bradycardia or transient sinus pauses during sleep should not be paced unless another indication exists. Asymptomatic Mobitz I generally needs no device.
Choose the right pacing mode	If LVEF 36–50% with AV block and > 40% ventricular pacing expected, use more physiologic activation (CRT or His-bundle pacing) rather than RV pacing, to prevent pacing-induced heart failure.

BLOCK IN ACUTE MI *the territory tells you the prognosis*

INFERIOR MI — usually benign
The **RCA supplies the AV node** in most people. Block is **nodal**, with a narrow escape, often **atropine-responsive**, and typically **resolves within days** of reperfusion. Temporary pacing may bridge; permanent devices are usually unnecessary.

ANTERIOR MI — ominous
Block reflects **extensive septal infarction** destroying the His-Purkinje system. **Infranodal**, wide escape, **atropine will not help**, and it signals a large infarct with high mortality. Prepare for pacing early and expect a permanent device.

AFTER THE DEVICE *what to check and what can go wrong*

Issue	Recognize it
Failure to capture	Pacing spike with no following complex . Causes: lead displacement, rising threshold, hyperkalemia, ischemia, or metabolic derangement.
Failure to sense	Spikes falling where they should not, risking R-on-T . Undersensing means the device is blind to intrinsic beats.
Oversensing	The device inhibits inappropriately (myopotentials, electromagnetic interference) and the patient becomes symptomatically bradycardic.
Early complications	Pneumothorax , hematoma, lead perforation with tamponade , and infection. Get a post-procedure chest X-ray; fever with a device demands blood cultures and an echo.
Pacing-induced cardiomyopathy	Falling EF with a high RV pacing burden — the reason physiologic pacing is preferred when heavy pacing is anticipated.

PEARLS & PITFALLS

- **Atropine can worsen infranodal block**. It speeds the atria while the diseased His-Purkinje system cannot follow, dropping the ventricular rate. Have pads on before you push it.
- **Wide QRS escape = infranodal = unstable**. Narrow junctional escape is comparatively reassuring.
- Always check **potassium and TSH**, and review every rate-slowing drug, before calling it primary conduction disease.
- **Lyme carditis usually recovers** — ask about travel and rash in the young patient with high-grade block before implanting.
- Confirm pacing **capture by palpating a pulse**. Electrical spikes on the monitor without mechanical capture is a trap.
- Block with an **anterior MI** implies a large infarct and carries far worse prognosis than block with an inferior MI.





Airway & RSI

Rapid sequence intubation · oxygenate, do not just intubate · plan for failure before you start

Oxygenation
beats intubation

THE 7 Ps *preparation is the intervention; the laryngoscopy is the easy part*

Step	What it means
1. Prepare	Equipment, suction, drugs drawn, monitoring, and a stated plan A to D that the whole team has heard aloud. Assign roles, and have a vasopressor drawn up before induction, not after the pressure drops.
2. Preoxygenate	3 min of tidal breathing, or 8 vital-capacity breaths, by HFNC or NIV (ATS 2026, strong recommendation). This washes nitrogen out of the lungs and fills the functional residual capacity with oxygen, which is the reservoir that buys apnea time. A non-rebreather cannot reach FiO_2 1.0 and fails in shunt (pneumonia, ARDS, edema): NIV halved peri-intubation hypoxemia in PREOXI.
3. Optimize	Resuscitate before you induce. Correct hypotension and hypovolemia, and consider a push-dose or infusion vasopressor first. Apneic oxygenation: leave a nasal cannula at 15 L/min running throughout laryngoscopy, which meaningfully extends safe apnea time at no cost.
4. Paralyze and induce	Give the induction agent and the paralytic in rapid succession. Do not under-dose the paralytic: an inadequately paralyzed patient is the commonest cause of a difficult first attempt.
5. Position	Ear-to-sternal-notch alignment, with ramping in obesity, and head-up 20 to 30 degrees, which improves both the view and preoxygenation.
6. Prove	Waveform capnography is mandatory and is the only reliable confirmation. Absent waveform means the tube is not in the trachea until proven otherwise.
7. Post-care	Secure, sedate, ventilate lung-protectively, and anticipate hypotension.

DRUGS *adult IV doses, and every one of them is reduced in shock*

INDUCTION

Ketamine 1 to 2 mg/kg (use 1 mg/kg if shocked): the agent of choice in shock, since it preserves sympathetic drive, and it bronchodilates in asthma. **Etomidate 0.3 mg/kg** (0.15 in shock): cardiostable, with transient adrenal suppression after a single dose that has not been shown to change outcome. **Propofol 1.5 to 2.5 mg/kg**: avoid or greatly reduce in shock, since it vasodilates and is negatively inotropic.

PARALYSIS

Rocuronium 1.2 mg/kg: onset 45 to 60 s, lasting 45 to 70 min, reversible immediately with **sugammadex 16 mg/kg**. **Succinylcholine 1 to 1.5 mg/kg**: onset 45 s, lasting 6 to 10 min. The shorter duration is not a rescue plan, because a patient who cannot be oxygenated will desaturate long before it wears off.

DO NOT GIVE SUCCINYLCHOLINE IF

Hyperkalemia or renal failure with a high potassium · burns beyond 24 to 48 h · denervation, stroke or spinal cord injury beyond 72 h · crush injury and rhabdomyolysis · myopathy such as Duchenne · prolonged immobility · personal or family history of malignant hyperthermia. All but the last cause hyperkalemic cardiac arrest through upregulated extrajunctional acetylcholine receptors. Use rocuronium instead.

PREDICT DIFFICULTY BEFORE YOU COMMIT *the airway you did not expect to be difficult is the dangerous one*

LEMON

Look externally: beard, facial trauma, obesity, small mandible, large tongue. Evaluate 3-3-2: three fingers mouth opening, three between chin and hyoid, two between hyoid and thyroid notch. Mallampati 3 or 4. Obstruction or obesity: stridor, mass, angioedema, radiation change. Neck mobility restricted by collar, arthritis or fixed flexion.

AND THINK PHYSIOLOGY, NOT JUST ANATOMY

MACOCHA is validated for ICU intubation, where a score of 3 or more predicts difficulty. But the patient who arrests during intubation usually had a physiologically difficult airway, not an anatomically difficult one: hypoxemia with no reserve, hypotension, severe acidosis needing a high minute ventilation, and right ventricular failure. Fix the physiology first, or accept that induction may be the terminal event.

THE FAILED AIRWAY *declare it out loud and move down the plan; oxygenation always beats intubation*

Plan	Action	The rule
A · Intubate	Direct or video laryngoscopy	Maximum 3 attempts, plus one by the most experienced operator. Change something every attempt: reposition, external laryngeal manipulation, bougie, video laryngoscope, smaller tube. Repeating an identical attempt is not a plan, and each one causes trauma and bleeding that makes the next worse.
B · Supraglottic	Second-generation supraglottic airway	Maximum 3 attempts, then stop and think. A supraglottic airway that oxygenates is a success, not a failure, and it buys time for a definitive plan.
C · Facemask	Two-person bag-mask ventilation	Two-person technique, oral and nasal adjuncts, and full paralysis, which improves mask ventilation rather than worsening it. This is the last chance to reoxygenate before the front of the neck.
D · Front of neck	Scalpel-bougie-tube cricothyroidotomy	Cannot intubate, cannot oxygenate: declare it early and cut. The recurring lesson from airway deaths is delay, not technical failure. Abort and reoxygenate whenever the saturation falls to 90 to 93%, rather than pressing on with one more look.

AFTER THE TUBE *the period in which post-intubation arrest happens*

CONFIRM AND SECURE

Waveform capnography, bilateral breath sounds, and a chest radiograph with the tip 3 to 5 cm above the carina. Record the depth at the lips so migration is detectable. Place an orogastric tube and sit the patient head-up 30 to 45 degrees.

SET THE VENTILATOR

Analgesia first, then sedation, so the patient is not paralyzed and aware. Lung-protective tidal volume 6 to 8 mL/kg predicted body weight, plateau pressure under 30, and titrate PEEP and FiO_2 together. Match the minute ventilation to the pre-intubation state in severe acidosis, or the pH will fall further.

EXPECT HYPOTENSION

Three mechanisms combine: the induction agent removes sympathetic drive, positive pressure reduces venous return, and the patient was usually already underfilled. Have fluid and a vasopressor running or immediately available, and consider reducing the induction dose rather than treating the consequences.

PEARLS & PITFALLS

- Resuscitate before you induce. The physiologically difficult airway kills more often than the anatomically difficult one.
- Leave nasal oxygen running through laryngoscopy. It is free apnea time.
- Change something every attempt, and cap it at three.
- No waveform means no tube until proven otherwise. Take it out and oxygenate.





Status Epilepticus

The time-critical seizure emergency · treat by the clock, and do not under-dose

5 min
is already status

DEFINE IT, AND WHY THE CLOCK MATTERS SO MUCH

Five minutes of continuous seizure, or two or more seizures without full recovery in between, is status epilepticus: treat it now. Refractory means failure of first- and second-line therapy; super-refractory means continuing or recurring 24 h or more despite anesthesia. The reason for the strict timing is receptor biology: as a seizure continues, GABA-A receptors are internalized off the synaptic membrane while NMDA receptors are recruited to it. Benzodiazepines therefore become progressively less effective the longer you wait, which is why the same drug that would have worked at 5 min often fails at 45. Note the start time and time every drug from it.

0 TO 5 MIN *do all of this while the benzodiazepine is being drawn up, not before*

AIRWAY AND OXYGEN

Position, high-flow oxygen, suction, and be ready to support ventilation. Most seizures stop before an airway is needed, so prepare for intubation without rushing into it.

ACCESS AND GLUCOSE

IV access and a capillary glucose in every patient, because hypoglycemia is instantly reversible and a common cause. Give thiamine 100 to 250 mg before glucose in the alcohol-dependent or malnourished: a glucose load consumes the last thiamine and can precipitate Wernicke encephalopathy.

MONITOR AND TIME IT

SpO₂, ECG, blood pressure, and write down the start time. Send electrolytes, calcium, magnesium, renal function and antiseizure drug levels if already treated, since subtherapeutic levels from non-adherence are among the commonest triggers.

STAGED THERAPY *escalate on the clock; the commonest error at every stage is giving too little*

- 1 5 to 20 min · BENZODIAZEPINE, at the full weight-based dose. Lorazepam 0.1 mg/kg IV (max 4 mg), repeated once, or midazolam 10 mg IM, or diazepam 0.15 to 0.2 mg/kg IV. Under-dosing is the single commonest error, usually from fear of respiratory depression, yet ongoing seizure causes far more respiratory compromise than an adequate benzodiazepine dose. If there is no IV, give IM midazolam: it is at least as effective as IV lorazepam and avoids the delay of chasing access.
- 2 20 to 40 min · SECOND-LINE, and the choice does not matter much. One of: levetiracetam 60 mg/kg (max 4500 mg), fosphenytoin 20 mg PE/kg, or valproate 40 mg/kg (max 3000 mg), all IV. ESETT, NEJM 2019 found the three equivalent, each stopping seizures in roughly half of patients, so pick on side-effect profile and availability: levetiracetam is easiest and interaction-free, fosphenytoin risks hypotension and arrhythmia, valproate is avoided in hepatic disease and pregnancy.
- 3 Beyond 40 min · REFRACTORY, go to anesthesia. Intubate and start continuous EEG, then a midazolam or propofol infusion (thiopental or ketamine if needed), titrated to seizure suppression or burst-suppression. Do not keep cycling through more second-line agents: each additional failed drug costs time during which neuronal injury continues.

FIND AND FIX THE CAUSE *several causes have a specific antidote, and the seizure will not stop without it*

Cause	Specific treatment
Metabolic	Hypoglycemia → glucose (after thiamine) · hyponatremia → hypertonic saline, which will not respond to any antiseizure drug · low calcium or magnesium, uremia, hypoxia.
Drugs	Alcohol or benzodiazepine withdrawal · non-adherence to antiseizure medication, so check levels · toxins · isoniazid overdose → pyridoxine, which is the only thing that works.
Brain	Stroke, hemorrhage, tumor, trauma, and infection. Meningitis or encephalitis needs antimicrobials and acyclovir immediately, not after imaging.
Pregnancy	Eclampsia → magnesium sulfate 4 to 6 g IV, then delivery. Magnesium, not a benzodiazepine, is the first-line drug here, and this is the one setting where the standard ladder does not apply.

NON-CONVULSIVE STATUS *the diagnosis you will miss unless you deliberately look for it*

WHEN TO SUSPECT IT

A patient who does not wake up after the convulsion stops. Also unexplained coma, or a fluctuating conscious level with no other explanation. Subtle signs: eye deviation, rhythmic twitching of face or limbs, automatisms, aphasia, nystagmus.

HOW TO CONFIRM AND TREAT

Urgent or continuous EEG is the only way to diagnose it, so a normal exam does not exclude it. Treat exactly like convulsive status and hunt the same causes. A meaningful proportion of unexplained ICU coma is non-convulsive status, and it is missed because nobody orders the EEG.

AFTER CONTROL

KEEP THEM SUPPRESSED, THEN WEAN SLOWLY

Start a maintenance antiseizure drug before the infusion is weaned, and wean the anesthetic slowly under continuous EEG, watching for recurrence. Image the brain and treat the underlying cause.

AND DO NOT FORGET

Aspiration is common: check a chest radiograph and protect the airway. Consider functional or dissociative seizures in the differential, particularly with a normal gas, preserved awareness and prolonged asynchronous movements. Look for rhabdomyolysis after a prolonged convulsion.

PRACTICAL CAUTIONS *the drug problems that catch people out*

WHEN TO SECURE THE AIRWAY

Do not intubate reflexively for the seizure itself, since most stop and the paralytic then hides ongoing electrical activity. Do intubate for a failing airway, persistent hypoxia, or the move to anesthesia. If you paralyze, you must have continuous EEG, or you are treating a patient you can no longer assess.

DRUG-SPECIFIC TRAPS

Fosphenytoin causes hypotension and arrhythmia if infused too fast, so rate-limit it and monitor. Valproate is contraindicated in hepatic failure and pregnancy. Levetiracetam needs renal dose adjustment and can cause agitation. Propofol infusion syndrome limits prolonged high-dose use in refractory cases.

PEARLS & PITFALLS

- Under-dosing the benzodiazepine is the commonest error. Give the full weight-based dose, then repeat once.
- No IV? Give IM midazolam rather than losing minutes hunting for access.
- Benzodiazepines work worse the longer you wait, because GABA-A receptors are internalized as the seizure continues.
- Not waking after the fit stops means non-convulsive status until an EEG says otherwise.
- Glucose in everyone, and thiamine before glucose in the alcohol-dependent or malnourished.
- Eclampsia gets magnesium, not a benzodiazepine. It is the one setting where the standard ladder does not apply.





Acute Ischemic Stroke

Time is brain · last known well · glucose, CT, then reperfuse

4.5 h / 24 h
lysis / thrombectomy

THE FIRST TEN MINUTES *three things decide everything that follows*

LAST KNOWN WELL

Not "when were they found". The clock starts at the last moment the patient was verifiably at baseline, and it is the single number that determines eligibility. **Call the family and the last person who spoke to them**, because getting this wrong either denies a treatable patient or lyses an ineligible one.

CHECK THE GLUCOSE

Hypoglycemia is the classic stroke mimic and is instantly reversible. A point-of-care glucose takes seconds and should precede everything else. Other mimics: **seizure with Todd paralysis, migraine with aura, functional deficit, and old deficits unmasked by infection.**

IMAGE IMMEDIATELY

Non-contrast head CT to exclude hemorrhage, plus **CT angiography** to find a large-vessel occlusion, and **CT perfusion**, if immediately available, when outside 6 h or the time is unknown. **Do not wait for labs before lysing**, except a glucose, unless you have specific reason to suspect coagulopathy.

THROMBOLYSIS *within 4.5 h of last known well, extended by imaging in selected patients*

Step	What to do, and why
Choose the agent	Tenecteplase 0.25 mg/kg (max 25 mg) as a single IV bolus, or alteplase 0.9 mg/kg (max 90 mg), 10% as a bolus then the rest over 60 min. Both are Class 1 in 2026, and tenecteplase is one bolus, which matters most when the patient is being transferred for thrombectomy, since there is no infusion to run during transport.
Control the pressure first	BP must be below 185/110 before you give it. Use labetalol or nicardipine. Above that threshold the risk of symptomatic hemorrhage rises steeply, and this is one of the few genuinely absolute preconditions.
Know the window	≤ 4.5 h from last known well as standard. Extended to 9 h, and in wake-up stroke, when perfusion or DWI-FLAIR mismatch imaging shows salvageable tissue. Benefit falls steeply with time in every window, so door-to-needle is the metric, not eligibility.
Then hold everything	No antiplatelet or anticoagulant for 24 h, and repeat the CT at 24 h before starting aspirin. Keep BP below 180/105 for 24 h. This is the window in which hemorrhagic transformation happens.

DO NOT LYSE IF

Hemorrhage on CT · BP uncontrolled above 185/110 · platelets < 100,000, INR > 1.7, or a DOAC within 48 h · recent intracranial surgery, prior intracerebral hemorrhage, or a large established infarct · recent major surgery or active internal bleeding. **When it is borderline, call the stroke physician while imaging is happening**, rather than after.

POSTERIOR STROKE HAS A LOW NIHSS

The NIHSS is weighted toward the anterior circulation, so a basilar or cerebellar stroke can score 2 or 3 while the patient is about to deteriorate catastrophically. **Vertigo, ataxia, diplopia, dysarthria or reduced consciousness deserve a CTA regardless of the score.** A low NIHSS is not a reason to withhold reperfusion.

THROMBECTOMY *for large-vessel occlusion, and the window is far longer than most people assume*

- Find the occlusion.** The target is a **proximal anterior large-vessel occlusion, ICA or M1**, with salvageable brain. This is why **CTA belongs in the initial scan**, not as an afterthought once thrombolysis is running.
- Do not stop at 6 hours.** 2026: up to 24 h is Class 1 with ASPECTS ≥ 6 on plain CT (perfusion adjunctive), large cores (ASPECTS 3 to 5) qualify, and so does basilar occlusion with NIHSS ≥ 10. DAWN, NEJM 2018 extended thrombectomy to **6 to 24 h** and DEFUSE 3, NEJM 2018 to **6 to 16 h**, both selected by a **mismatch between a small core and a large deficit or penumbra**. In late windows the imaging, not the clock, decides. Benefit is large: DAWN reported a number needed to treat of roughly 3 for reduced disability.
- Give thrombolysis as well if the patient is eligible.** Do not skip lysis to go straight to the angiography suite, since some clots resolve before the catheter arrives and access can fail. Lyse and transfer simultaneously.
- Activate the team before you have all the answers.** Transfer and neurointervention activation should run in parallel with imaging review, because **the delay that costs tissue is almost always organizational, not diagnostic.**

SUPPORTIVE CARE AND THE FIRST 24 HOURS

Parameter	Target	Why
Blood pressure	No lysis: permissive, treat only above 220/120. After lysis: below 180/105 for 24 h. After thrombectomy: 180/105 or lower, never a target below 140	The penumbra is perfused by collaterals that depend on systemic pressure, so lowering the BP in an untreated stroke extends the infarct. Once lysed, the balance flips toward bleeding risk.
Glucose	140 to 180 mg/dL	Hyperglycemia worsens infarct growth and hemorrhagic transformation; hypoglycemia mimics and injures. Tight control offers no benefit and causes hypoglycemia.
Temperature	Normothermia, treat fever	Fever raises metabolic demand in tissue that is already marginally perfused, and independently worsens outcome. Look for the source.
Swallow and mobility	Swallow screen before any oral intake; stroke unit	Aspiration pneumonia is a leading preventable complication. Stroke-unit care itself reduces death and dependency more than most drugs here.
VTE prophylaxis	Intermittent pneumatic compression	Proven to reduce DVT in immobile stroke patients. Avoid early therapeutic anticoagulation , which increases hemorrhagic transformation without reducing recurrence.

WATCH FOR THESE COMPLICATIONS

HEMORRHAGIC TRANSFORMATION

Sudden drop in consciousness, new headache, vomiting, or a rising blood pressure means urgent repeat CT. Stop the lytic, reverse with cryoprecipitate and an antifibrinolytic, correct any coagulopathy, and call neurosurgery.

MALIGNANT MCA EDEMA

Peaks at 2 to 5 days in large territory infarcts. **Decompressive hemicraniectomy within 48 h reduces mortality substantially**, with the clearest functional benefit under age 60. Identify candidates early rather than after herniation. Cerebellar infarcts can obstruct the fourth ventricle and need surgery too.

OROLINGUAL ANGIOEDEMA

Occurs with alteplase, **far more often in patients on ACE inhibitors**. Usually mild and unilateral. **Stop the infusion, secure the airway if progressive**, and treat with antihistamine and corticosteroid.

PEARLS & PITFALLS

- Glucose before anything else.** Hypoglycemia is fully reversible and looks exactly like a stroke.
- Leave the pressure high unless you are lysing.** The collaterals need it.
- A low NIHSS does not exclude a large-vessel occlusion**, especially in the posterior circulation.
- Secondary prevention starts at 24 h:** aspirin after the repeat CT, high-intensity statin, anticoagulation for atrial fibrillation, carotid imaging, BP and diabetes control.

References: AHA / ASA guidelines for the early management of patients with acute ischemic stroke · Nogueira et al, DAWN, NEJM 2018 · Albers et al, DEFUSE 3, NEJM 2018 · trials of tenecteplase versus alteplase in acute ischemic stroke · pooled analyses of decompressive hemicraniectomy in malignant MCA infarction.

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





Anaphylaxis

Epinephrine first · intramuscular · anterolateral thigh · now

0.3–0.5 mg
epinephrine IM

EPINEPHRINE IS THE ONLY FIRST-LINE TREATMENT

Epinephrine 0.01 mg/kg IM, up to 0.5 mg in adults and 0.3 mg in children, using the 1 mg/mL concentration, into the anterolateral thigh. Repeat every 5 to 15 min if there is no improvement. Nothing comes before it, and there is no absolute contraindication in anaphylaxis. The thigh is specified because vastus lateralis absorption is faster and more reliable than deltoid or subcutaneous. Delay in giving epinephrine is the single factor most consistently associated with fatal anaphylaxis, and the commonest reason for delay is reaching for an antihistamine first.

RECOGNIZE IT *sudden onset plus an airway, breathing or circulation problem, with or without a rash*

AIRWAY

Swelling of the tongue, lips or throat, hoarse voice, stridor, difficulty swallowing, a sense of throat closure. This is the one that kills quickest and the one that becomes untubatable if you wait.

BREATHING

Wheeze, tachypnea, increasing work of breathing, fatigue, cyanosis, SpO₂ below 94%, progressing to respiratory arrest. In a known asthmatic this is easily mislabeled as an asthma attack.

CIRCULATION

Hypotension, tachycardia, pallor, clamminess, reduced consciousness, cardiac arrest, from massive capillary leak and vasodilation. Up to a third of the circulating volume can shift into the tissues within minutes.

NO RASH DOES NOT MEAN NO ANAPHYLAXIS

Skin and mucosal changes support the diagnosis but are absent in a meaningful minority, and are least likely in the most rapidly progressive drug- and venom-triggered reactions. Their absence never excludes it, and anaphylaxis can present with hypotension alone after a known exposure.

IMMEDIATE MANAGEMENT *five actions, in this order, within the first minutes*

- 1 Remove the trigger and give epinephrine. Stop the drug, infusion, blood product or latex exposure, but **do not delay treatment while you hunt for the cause**. If an insect stinger is present, remove it. **Note the time of each epinephrine dose**, since the decision to escalate depends on how many have been given and when.
- 2 Lie the patient flat and elevate the legs. **Sitting or standing a hypotensive anaphylaxis patient up can be fatal**: with a vasodilated, volume-depleted circulation, the venous return collapses and the heart empties, and sudden deaths have occurred at the moment patients were sat upright or walked. **Sit them up only if breathing is the dominant problem**, and use **left lateral tilt in pregnancy** to keep the gravid uterus off the vena cava.
- 3 High-flow oxygen and aggressive fluid. **15 L/min via a non-rebreather**, and a **rapid crystalloid bolus of 1 to 2 L in adults, or 20 mL/kg in children**, repeated as needed. **Large volumes are frequently required**, because the underlying problem is distributive shock with profound capillary leak rather than pump failure.
- 4 Call for airway help early. **Anesthesia or critical care should be summoned before the airway looks difficult, not after**. Progressive angioedema converts a routine intubation into a surgical airway within minutes, so the threshold for early definitive airway control is low.
- 5 Move to a monitored area and reassess continuously. Recheck airway, breathing, circulation and mental status after every intervention, and **repeat the epinephrine at 5 to 15 min if the patient has not clearly improved**. Most patients who need a second dose need it because the first was given late, not because it failed.

REFRACTORY ANAPHYLAXIS *no better after two IM doses, which is when the plan has to change*

Step	What to do, and why
Epinephrine infusion	Start an IV epinephrine infusion, titrated, by someone experienced with it and with continuous monitoring. This replaces further IM dosing rather than supplementing it. IV push epinephrine is a common source of serious dosing error and should be reserved for cardiac arrest or peri-arrest, where the arrest dose applies.
More fluid	Keep giving crystalloid. Refractory hypotension here is usually under-resuscitation, and several liters may be needed before the pressure responds.
Add a vasopressor	Norepinephrine or vasopressin if shock persists despite epinephrine and fluid. Vasopressin is particularly useful when the vasoplegia is profound.
On a beta-blocker?	Give glucagon 1 to 5 mg IV, followed by an infusion. Beta blockade makes the patient relatively unresponsive to epinephrine, and glucagon bypasses the beta receptor entirely by stimulating adenylate cyclase directly . Warn about vomiting, and protect the airway.

SECOND-LINE, AND WHAT NO LONGER APPLIES *after epinephrine, never instead of it*

STEROIDS AND ANTIHISTAMINES ARE NOT PREVENTIVE

The 2023 AAAAI / ACAAI practice parameter suggests against giving glucocorticoids or antihistamines to prevent biphasic anaphylaxis, because they do not reliably do so. Antihistamines relieve itch and urticaria and nothing else: they do not treat airway swelling or shock, and giving one first is the classic fatal delay.

FOR PERSISTENT SYMPTOMS

Nebulized albuterol for bronchospasm that persists after epinephrine, and **nebulized epinephrine** for upper airway stridor, as a temporizing measure while definitive airway help arrives. **Neither replaces intramuscular epinephrine**.

CONFIRM WITH TRYPTASE

Serum tryptase, ideally within 15 min to 3 h of onset, plus a baseline sample at least 24 h later once recovered. The comparison between acute and baseline is what makes it informative, and an elevated baseline points to an underlying mast cell disorder.

BEFORE THEY LEAVE *observation is risk-stratified, and discharge is an intervention in itself*

HOW LONG TO OBSERVE

There is no single mandated period. Biphasic reaction is unlikely if the episode was not severe and the patient stays symptom-free for an hour after full resolution. Observe longer after a severe reaction, more than one dose of epinephrine, a delayed first dose, or a previous biphasic reaction.

PRESCRIBE AND TEACH

At least two epinephrine auto-injectors, particularly for anyone who needed multiple doses or has had a biphasic reaction, plus **hands-on training with a trainer device** and a **written anaphylaxis action plan**. A prescription without a demonstration is frequently never used correctly.

REFER AND DOCUMENT

Refer every patient to allergy for trigger confirmation and long-term planning. **Document the trigger prominently in the allergy field**, because an undocumented drug trigger is how the same reaction happens again in hospital.

PEARLS & PITFALLS

- Intramuscular thigh, not subcutaneous and not deltoid. Absorption is faster and more reliable.
- Do not sit them up. Lie flat with legs raised; sudden deaths have followed being sat upright.
- An antihistamine first is the classic fatal delay. It treats the rash, not the reaction.
- Glucagon for the beta-blocked patient who is not responding to epinephrine.

References: Golden et al, Anaphylaxis: a 2023 practice parameter update, *Ann Allergy Asthma Immunol* 2023 · Wang et al, Anaphylaxis in practice: a guide to the 2023 practice parameter update, *J Allergy Clin Immunol Pract* 2024 · World Allergy Organization anaphylaxis guidance.

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





Major Hemorrhage

Massive transfusion · damage-control resuscitation · stop the bleeding, do not just replace it

TXA < 3 h
and blood, not saline

ACTIVATE EARLY, AND ON CLINICAL GROUNDS

Loss of one blood volume in 24 h, 50% in 3 h, bleeding faster than 150 mL/min, or transfusion needed to stay alive. Do not wait for a hemoglobin: the initial value is normal in brisk bleeding because the patient has not yet hemodiluted, so a reassuring number early means nothing. **Activating the protocol is what gets balanced products to the bedside** instead of sequential single units, and under-activation is far more damaging than over-activation.

FIRST MOVES *control the source while you resuscitate; these run in parallel*

STOP THE BLEEDING

Direct compression, tourniquet, hemostatic packing, pelvic binder. Definitive control by surgery, interventional radiology or endoscopy is the actual treatment: transfusion only buys the time to get there. Call surgery, IR and hematology at activation, not once products are running.

TXA WITHIN 3 HOURS

Tranexamic acid 1 g IV over 10 min, then 1 g over 8 h (CRASH-2, Lancet 2010; CRASH-3, Lancet 2019 for head injury). Earlier is better and the benefit decays fast. Given beyond 3 h it is associated with harm, so the time of injury, not the time of arrival, is what counts.

RESTRICT CRYSTALLOID

Give blood, not liters of saline. Crystalloid dilutes clotting factors, worsens acidosis and drops the temperature, feeding the lethal triad. Permissive hypotension to a systolic of about 80 to 90 until bleeding is controlled, so you do not pop a fresh clot. The exception is traumatic brain injury, where the injured brain needs perfusion: target a MAP of at least 80.

TRANSFUSE *empiric ratio first, then goal-directed once numbers are back*

Target	Aim	If not met
Empiric ratio	RBC : FFP : platelets = 1 : 1 : 1 in fixed packs until labs return	PROPPR, JAMA 2015 compared 1:1:1 with 1:1:2. Its primary endpoints, 24-h and 30-day mortality, were negative. The 1:1:1 group achieved more hemostasis and fewer deaths from exsanguination at 24 h, which were secondary findings, and that is the honest basis for the balanced ratio.
Hemoglobin	7 to 9 g/dL during active bleeding	Transfuse red cells. Do not chase a normal hemoglobin while still bleeding.
Platelets	> 50, and > 100 for CNS or eye injury, or ongoing bleeding	One adult pool.
Fibrinogen	> 1.5 to 2.0 g/L	Cryoprecipitate or fibrinogen concentrate. Fibrinogen falls first and fastest of all the clotting factors, so it is the one most often left uncorrected.
PT / aPTT	< 1.5 × normal	FFP 15 to 20 mL/kg, or PCC if on warfarin.
Ionized calcium	> 1.1 mmol/L	IV calcium chloride or gluconate. Stored blood is citrated and citrate chelates calcium, so massive transfusion reliably causes hypocalcemia. Calcium is required for the clotting cascade and for myocardial contractility, making this a cause of both refractory coagulopathy and refractory hypotension. Check it early and repeatedly.

THE LETHAL TRIAD *each element worsens the other two, so all three are treated at once*

HYPOTHERMIA

Clotting enzymes are temperature-dependent and slow markedly below 34 °C, so a cold patient is coagulopathic whatever the numbers say. Warm the patient, the fluids and the blood, use forced-air warming, and stop exposing them once the survey is done.

ACIDOSIS

Acidosis impairs the clotting cascade and blunts the response to vasopressors. The fix is restoring perfusion and stopping the bleeding, not bicarbonate. Follow lactate and base deficit as resuscitation markers.

COAGULOPATHY

Trauma-induced coagulopathy is present on arrival in a substantial minority, before any dilution. Balanced ratio plus TXA plus calcium, then viscoelastic testing (ROTEM or TEG) to target the specific defect once available.

BY SETTING, AND WHAT TO REVERSE

Setting	Additional measures
Trauma	Damage-control resuscitation and damage-control surgery: control contamination and bleeding, then return once resuscitated rather than completing a long operation on a cold, acidotic patient. Pelvic binder, early CT or straight to the operating room, TXA early.
Obstetric	Uterotonics, bimanual compression, balloon tamponade, and the operating room. TXA early. Obstetric hemorrhage is frequently underestimated because young patients compensate until they crash.
GI	Endoscopy or interventional radiology; in variceal bleeding add octreotide and ceftriaxone. Surgery if uncontrolled.
Reverse anticoagulants	Warfarin: 4-factor PCC plus vitamin K. DOACs: idarucizumab for dabigatran, 4-factor PCC for factor Xa inhibitors (andexanet alfa was withdrawn in the US in 2025). Ask what they take at activation, since this changes the products you need.

MONITOR, AND KNOW WHEN TO STOP *over-transfusion has its own harms*

WHAT TO REPEAT, AND HOW OFTEN

Full blood count, coagulation screen, fibrinogen, blood gas (lactate and base deficit) and ionized calcium every 30 to 60 min during active bleeding. Numbers drawn an hour ago describe a patient who no longer exists, which is why viscoelastic testing at the bedside is preferred once available.

DE-ESCALATE ONCE CONTROLLED

Stand the protocol down deliberately and tell the blood bank, or products keep arriving. Over-transfusion causes TACO, TRALI, hyperkalemia and thrombosis, and the balanced ratio that was right during exsanguination is wrong once bleeding has stopped. Then switch to VTE prophylaxis, which these patients need sooner than instinct suggests.

PEARLS & PITFALLS

- Blood in a balanced ratio, and TXA within 3 hours. Crystalloid dilutes clotting factors and cools the patient.
- A normal initial hemoglobin means nothing in brisk bleeding. Activate on clinical grounds.
- Check ionized calcium early and repeatedly. Citrate chelates it, and low calcium causes both coagulopathy and hypotension.
- Definitive control is the treatment. Transfusion only buys time to reach the operating room, IR suite or endoscope.





Pulmonary Embolism

Diagnose · risk-stratify on the right ventricle · treat by severity

The RV
decides everything

DIAGNOSE *pretest probability first, because the D-dimer is only interpretable once you have it*

Step	What to do, and why
Estimate probability	Use Wells or the Geneva score . In a low-risk outpatient in whom you are already doubtful, PERC rules PE out without any test if all eight criteria are negative, which spares the D-dimer that would otherwise generate a needless CT.
Use D-dimer correctly	Only in low or intermediate probability , where a negative result excludes PE. It is worthless in high probability , where a negative does not exclude and a positive was expected. Use an age-adjusted threshold ($\text{age} \times 10 \mu\text{g/L}$ above 50), which reduces unnecessary imaging without missing more events.
Image	CT pulmonary angiography is the test. Use V/Q scanning if contrast is contraindicated by anaphylaxis or severe renal failure, or in pregnancy where a normal chest radiograph makes V/Q attractive. If the patient is too unstable to travel, a bedside echo showing RV strain justifies treating without waiting for the CT.
Do not wait to anticoagulate	Start heparin on clinical suspicion when probability is high and imaging will be delayed. The risk of a few hours of anticoagulation is far smaller than the risk of an untreated large PE.

RISK-STRATIFY *2026 AHA/ACC Acute PE Clinical Categories, which retire “massive / submassive”*

Category	Definition	Disposition & therapy
A	Subclinical: incidental, asymptomatic PE	Discharge home from the ED. Hospitalization is not required.
B	Symptomatic, low severity score (PESI I-II, sPESI 0, Hestia 0)	Early discharge is generally recommended , given follow-up and social circumstances.
C1 C2 C3	Symptomatic, elevated severity score (PESI III-V, sPESI ≥ 1 , Hestia ≥ 1), then subdivided: C1 normal RV <i>and</i> normal biomarkers C2 abnormal RV <i>or</i> ≥ 1 abnormal biomarker C3 abnormal RV <i>and</i> ≥ 1 abnormal biomarker	Hospitalize. Anticoagulate and monitor. C3 is the old “intermediate-high”: watch closely for deterioration; the value of systemic thrombolysis here is uncertain (2b) .
D1-D2	Incipient cardiopulmonary failure: normotensive shock, or transient hypotension (SBP < 90 or a drop > 40 mmHg lasting < 15 min or fluid-responsive)	Hospitalize, activate PERT. Advanced therapy may be considered (2b): systemic or catheter-directed thrombolysis, or mechanical thrombectomy.
E1-E2	Cardiopulmonary failure: persistent hypotension with cardiogenic shock, or cardiac arrest	Reperfuse. Systemic thrombolysis (2a) if bleeding risk acceptable; catheter-directed thrombolysis, mechanical thrombectomy or surgical embolectomy each 2a in E1 .

Class 3 HARM: do not give systemic thrombolysis in Categories A1-C2, where bleeding and intracranial hemorrhage outweigh any benefit. **PERT activation is Class 1 for Categories C-E.** Add “R” (eg, C3R) for the respiratory modifier. **Shock markers:** lactate > 2 , AKI, urine output < 0.5 mL/kg/h, altered mentation, cardiac index < 2.2 , MAP < 60 .

ANTICOAGULATE *the treatment for the overwhelming majority, and the choice is driven by comorbidity*

DOAC FIRST-LINE

Apixaban or rivaroxaban with a loading phase and no heparin bridge, or edoxaban and dabigatran after 5 days of heparin. Preferred for most stable PE: **equally effective with less major bleeding** than warfarin. **Avoid in severe renal impairment, antiphospholipid syndrome and pregnancy.**

PARENTERAL: LMWH OVER UFH

2026 recommends LMWH over unfractionated heparin when parenteral therapy is needed. **Reserve UFH for when thrombolysis is likely, in hemodynamic instability, or in severe renal failure**, where its short half-life and reversibility matter. LMWH in pregnancy; in active cancer both LMWH and DOACs are acceptable, with more GI bleeding from DOACs in luminal tumors.

HOW LONG

Provoked by a major transient risk factor: 3 months, then stop. **Unprovoked or a persistent risk factor: extended, indefinite therapy** with periodic review, because the recurrence risk after stopping is high. **Reduced-dose apixaban or rivaroxaban is an option for the extended phase.**

CATEGORY E · THE RV IS FAILING *and the usual resuscitation instincts make it worse*

- 1** Thrombolyse. Alteplase **100 mg IV over 2 h** is standard, with **50 mg** used where bleeding risk is high. **In cardiac arrest with suspected PE, give alteplase 50 mg as a bolus and continue CPR for 60 to 90 min**, because the drug needs circulation to reach the clot and survival with good outcome is reported despite prolonged resuscitation.
- 2** Be careful with fluid. Give at most **500 mL**, and only if the patient is genuinely underfilled. **Over-filling a failing right ventricle distends it further, bows the septum into the left ventricle and reduces cardiac output**, which is the opposite of what you intended. Reach for **norepinephrine** early instead.
- 3** Avoid intubation if you possibly can. **Induction drugs drop preload and systemic pressure, and positive pressure raises RV afterload**, a combination that precipitates arrest in a preload-dependent, afterload-sensitive ventricle. If it is unavoidable, use **a vasopressor before induction, ketamine or etomidate, and the lowest airway pressures that work**.
- 4** Know the alternatives. **Catheter-directed thrombolysis or thrombectomy** where lysis is contraindicated or has failed, and **surgical embolectomy or VA-ECMO** as a bridge in selected centers. **An IVC filter is only for a patient in whom anticoagulation is genuinely contraindicated**, and should be retrieved once it is safe to anticoagulate.

BEFORE AND AFTER DISCHARGE

LOOK FOR THE CAUSE

Take an occult malignancy seriously in unprovoked PE, but limit the search to **age-appropriate cancer screening plus a history and examination**: extensive routine CT screening finds little and causes harm. Test for **antiphospholipid syndrome** if it would change the anticoagulant, since APS mandates warfarin over a DOAC.

FOLLOW UP FOR CTEPH

Ask about PE symptoms and functional limitation at every visit for at least 1 year (2026, Class 1). **Persistent dyspnea beyond 3 months warrants evaluation for CTEPH**, with V/Q as the screening test of choice. It is **potentially curable by pulmonary endarterectomy**, so it must never be dismissed as deconditioning.

PEARLS & PITFALLS

- **Never order a D-dimer in high pretest probability.** It cannot exclude and will only delay the CT.
- **Fluid is not the answer in RV failure.** Cap it at 500 mL and start norepinephrine.
- **Intubation can kill a high-risk PE.** Support the pressure first, and avoid it if you can.
- **PEITHO:** upfront lysis in intermediate-high PE cut decompensation but doubled major bleeding, with a tenfold rise in intracranial hemorrhage and no mortality benefit.

References: 2026 AHA/ACC/Multisociety Acute PE Guideline and its ACC Guideline-at-a-Glance (JACC 2026;87:13) · ESC 2019 acute PE guideline · Meyer et al, PEITHO, NEJM 2014 · CHEST guideline on antithrombotic therapy for VTE disease · Righini et al, age-adjusted D-dimer, JAMA 2014 · Kline et al, PERC rule · Hestia criteria for outpatient management.

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





Poisoning & Antidotes

General approach · toxidromes · specific antidotes · treat the patient, not the poison

Support

then reverse

SUPPORTIVE CARE IS THE MAINSTAY

Resuscitate ABCDE, correct the airway, glucose, temperature and rhythm, and treat the patient rather than the drug name. Antidotes exist for only a minority of poisonings, and almost every good outcome comes from meticulous supportive care rather than a reversal agent. **Call Poison Control early on 1-800-222-1222**, which is staffed nationally and will give an agent-specific plan. **Check a glucose and an ECG in everyone**, since hypoglycemia mimics every toxidrome and QRS or QTc changes silently predict the arrest.

TOXIDROMES *recognize the pattern and you have the class before any level returns*

Toxidrome	Signs	Typical agents
Anticholinergic	Hot, dry, flushed, dilated pupils, delirium, urinary retention, tachycardia. "Mad as a hatter, dry as a bone"	Antihistamines, TCAs, atropine, jimson weed
Cholinergic	SLUDGE or DUMBELS : salivation, lacrimation, diarrhea, miosis, bronchorrhea. Bronchorrhea and bradycardia are what kill	Organophosphates, carbamates, nerve agents
Sympathomimetic	Agitation, sweating (which separates it from anticholinergic), mydriasis, tachycardia, hypertension, hyperthermia	Cocaine, amphetamines, MDMA
Opioid	Pinpoint pupils, low respiratory rate, depressed consciousness . The respiratory rate is the vital sign to follow	Heroin, morphine, oxycodone, fentanyl
Sedative-hypnotic	Depressed consciousness, slurred speech, normal pupils , respiratory depression	Benzodiazepines, alcohol, barbiturates
Serotonin	Clonus and hyperreflexia, greatest in the legs , agitation, hyperthermia, rigidity	SSRIs plus an MAOI, tramadol, linezolid

ACETAMINOPHEN *the commonest serious overdose, and the one where the antidote genuinely works*

WHO NEEDS A LEVEL

Acute ingestion above 150 mg/kg, or roughly 7.5 to 10 g in an adult. Draw the level at 4 h after ingestion, and not before, because earlier values cannot be plotted. Treat if the level is above the Rumack-Matthew treatment line, which starts at 150 mcg/mL at 4 h. The nomogram applies only to a single acute ingestion with a known time, and is invalid for staggered ingestion, extended-release preparations, or an unknown time.

N-ACETYLCYSTEINE

IV 21-hour protocol: 150 mg/kg over 1 h, then 50 mg/kg over 4 h, then 100 mg/kg over 16 h, totaling 300 mg/kg. NAC replenishes glutathione so the toxic metabolite NAPQI is conjugated before it injures hepatocytes. It is essentially fully protective when started within 8 h, which is why you start it empirically rather than waiting for a level if 8 h have nearly passed. Give it regardless of timing in late presentation, staggered ingestion, or established liver injury.

ANAPHYLACTOID REACTIONS

Flushing, urticaria, wheeze and hypotension occur in a meaningful minority, almost always during the high-rate first bag, and are a rate-related histamine release rather than true allergy. Slow or pause the infusion, give an antihistamine, and restart. Do not abandon NAC, because the alternative is untreated hepatotoxicity.

SPECIFIC ANTIDOTES *give alongside supportive care, never instead of it*

Poison	Antidote and note
Acetaminophen	N-acetylcysteine, as above
Opioids	Naloxone, starting 0.04 to 0.4 mg IV and titrating to respiratory rate, not to consciousness. Larger doses in the opioid-dependent precipitate florid withdrawal. Its half-life is shorter than most opioids, so watch for re-sedation and consider an infusion, especially with methadone or fentanyl analogues.
Beta-blockers	Glucagon, plus high-dose insulin with dextrose (euglycemic hyperinsulinemia)
Calcium channel blockers	Calcium, plus high-dose insulin with dextrose, which is the mainstay in severe poisoning
TCAs	Sodium bicarbonate for a QRS above 100 ms, which both narrows the QRS and prevents arrhythmia by overcoming sodium channel blockade
Digoxin	Digoxin-specific Fab fragments. Avoid calcium in digoxin-associated hyperkalemia
Methanol, ethylene glycol	Fomepizole (or ethanol) plus hemodialysis. Fomepizole blocks alcohol dehydrogenase so the parent alcohol is not converted to its toxic acid
Organophosphates	Atropine titrated to drying of secretions, not to heart rate, plus pralidoxime. Very large atropine doses are often needed
Others	Iron → deferoxamine · Cyanide → hydroxocobalamin · Local anesthetic → lipid emulsion 20% · Sulfonyleurea → octreotide plus dextrose · Methemoglobinemia → methylene blue · Warfarin → vitamin K plus PCC · Heparin → protamine · Benzodiazepines → flumazenil, rarely justified because it precipitates seizures in co-ingestion or dependence

DECONTAMINATION AND ELIMINATION *narrow indications, and easy to do more harm than good*

ACTIVATED CHARCOAL

1 g/kg within about 1 h of ingestion, in a patient with a protected airway. Does not bind metals, alcohols, hydrocarbons, lithium or caustics, and aspiration of charcoal causes a severe pneumonitis, so it is not given to the drowsy or vomiting patient.

URINE ALKALINIZATION

For salicylates, and for phenobarbital. Bicarbonate raises urinary pH so the weak acid becomes ionized and cannot be reabsorbed, trapping it in the tubule. It requires aggressive potassium replacement, since hypokalemia prevents the urine from alkalizing at all.

HEMODIALYSIS

For salicylate, lithium, methanol, ethylene glycol, metformin and valproate: small, water-soluble, poorly protein-bound molecules. Call nephrology early in severe salicylate poisoning, where deterioration is abrupt. Never intubate a salicylate patient casually: losing their compensatory hyperventilation can be fatal.

PEARLS & PITFALLS

- Glucose and an ECG in every poisoned patient. Hypoglycemia mimics everything, and the QRS predicts the arrest.
- Start NAC empirically if 8 h are nearly up, and stop it later if the level is below the line.
- Anaphylactoid reactions to NAC are rate-related. Slow it, treat it, restart it.
- Titrate naloxone to breathing, not to waking. Full reversal buys agitation and withdrawal.

References: American Association of Poison Control Centers and US Poison Control (1-800-222-1222) guidance · Rumack & Matthew, acetaminophen poisoning and toxicity nomogram · FDA-approved acetylcysteine (Acetadote) 21-hour intravenous protocol · extracorporeal treatments in poisoning (EXTRIP) workgroup recommendations.

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

