



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.





ARDS

Acute respiratory distress syndrome · 2023 Global Definition · protect the lung, find the trigger

4–8 mL/kg
predicted body weight

DIAGNOSE *all four criteria, and what the 2023 Global Definition changed*

Criterion	Detail
Timing	Within 1 week of a known insult, or of new or worsening respiratory symptoms.
Imaging	Bilateral opacities on chest radiograph, CT or ultrasound , not explained by effusion, collapse or nodules.
Origin	Not fully explained by cardiac failure or fluid overload. Get an echo if there is no obvious ARDS risk factor, because the whole treatment diverges if this is really decompensated heart failure.
Oxygenation	Hypoxemia on PEEP or CPAP ≥ 5 . New in 2023: a non-intubated category for patients on high-flow nasal oxygen ≥ 30 L/min or CPAP/NIV , and SpO₂/FiO₂ ≤ 315 may substitute for PaO ₂ /FiO ₂ when SpO ₂ is ≤ 97%. Lung ultrasound is accepted, and the Kigali pathway allows diagnosis without arterial gases in resource-limited settings.

SEVERITY BY P/F ON PEEP ≥ 5

Mild 201 to 300 (mortality ~27%) · **Moderate 101 to 200** (~32%) · **Severe ≤ 100** (~45%). **Severity is not just prognostic:** it is what selects patients for proning, neuromuscular blockade and ECMO referral.

ARDS IS A SYNDROME, NOT A DIAGNOSIS

Always name the trigger. **Pulmonary:** pneumonia, aspiration, contusion, inhalation, near-drowning. **Extrapulmonary:** sepsis, pancreatitis, massive transfusion or TRALI, trauma, burns. **Lung-protective ventilation supports the patient; treating the precipitant is what ends the illness.**

LUNG-PROTECTIVE VENTILATION *the non-negotiable core, and why each number exists*

Target	Value	Why
Tidal volume	4 to 8 mL/kg PBW <i>start 6, drop to 4</i>	Dose on predicted body weight, calculated from height, never actual weight. Lung size tracks height, so dosing an obese patient on actual weight delivers a grossly injurious breath. Men = $50 + 0.91 \times (\text{height cm} - 152.4)$; women = $45.5 + 0.91 \times (\text{height cm} - 152.4)$.
Plateau pressure	≤ 30 cmH₂O	Measured on an inspiratory hold . It reflects alveolar distension , which is what injures lung; peak pressure includes airway resistance and misleads.
Driving pressure	< 15 cmH₂O	Plateau minus PEEP , which is tidal volume normalized to the compliance of the remaining aerated lung. It may matter more than any single tidal volume or PEEP value , because it describes the strain actually applied to the "baby lung".
pH floor	≥ 7.20 <i>permissive hypercapnia</i>	Accept the CO₂. Chasing a normal PaCO ₂ with bigger breaths or a much higher rate re-creates the injury you are trying to avoid.
PEEP / FiO ₂	Titrate to PaO₂ 55 to 80 or SpO₂ 88 to 95%	Use an ARDSNet table (FiO ₂ 0.3 → PEEP 5, rising to FiO ₂ 1.0 → PEEP 18 to 24). Higher PEEP in moderate to severe disease , but avoid routine recruitment maneuvers: the ART, JAMA 2017 strategy of aggressive recruitment with decremental PEEP increased mortality .

PRONE POSITIONING *the adjunct with the clearest mortality benefit, and it is under-used*

WHO AND HOW LONG

Moderate to severe ARDS with P/F below 150 on FiO₂ ≥ 0.6 and PEEP ≥ 5. **At least 16 consecutive hours per session, started early.** PROSEVA, NEJM 2013 reported **28-day mortality of 16% versus 33%.** **Brief or late proning under-delivers the benefit**, which is the commonest way this intervention is wasted.

WHY IT WORKS

Proning **recruits dorsal lung that was compressed by the heart and abdomen**, makes ventilation more homogeneous, and improves V/Q matching. **The benefit is not just better oxygenation but less ventilator-induced injury**, because strain is distributed across more open lung. That is why it improves survival when transient oxygenation tricks do not.

ADJUNCTS AND RESCUE *in order, for moderate to severe or refractory disease*

Intervention	Where it fits
Conservative fluids	Once shock has resolved. FACTT, NEJM 2006 gave more ventilator-free days with a conservative strategy, without increasing renal failure. Do not apply it while the patient is still in shock.
Neuromuscular blockade	SCCM 2026 suggests it when P/F stays < 150 with persistent hypoxemia or unmet ventilator targets after optimization, for 48 h at most. Pooled trials show lower mortality and barotrauma; ROSE found no gain from routine early paralysis, so it is targeted, not a default. Pair with deep sedation, and remember it masks seizures.
Corticosteroids	Dexamethasone in moderate to severe ARDS (DEXA-ARDS, Lancet Respir Med 2020), and steroids for the specific trigger where indicated, such as COVID-19 or pneumocystis.
Inhaled nitric oxide or prostacyclin	Rescue oxygenation only. It improves the number on the monitor with no mortality benefit , so treat it as a bridge, not a treatment.
VV-ECMO	P/F below 80, or pH below 7.25 with uncorrectable hypercapnia , at an ECMO center (EOLIA, NEJM 2018). Refer early rather than after multi-organ failure , because transport of a crashing patient is the limiting step.

REFRACTORY HYPOXEMIA, STEPWISE

Optimize sedation and synchrony → increase PEEP → prone for 16 h → neuromuscular blockade up to 48 h → inhaled pulmonary vasodilator → VV-ECMO. **Work down the list rather than jumping to rescue therapy**, since the early steps are cheaper, faster and carry the better evidence.

PEARLS & PITFALLS

- **Predicted body weight, from height.** Actual weight over-ventilates and causes lung injury.
- **Do not chase a normal PaCO₂.** Accept permissive hypercapnia to a pH of 7.20.
- **Prone early and for long sessions.** Short or late proning wastes the one adjunct with a clear survival benefit.
- **Always name the trigger.** ARDS is a syndrome, and the precipitant is what you actually treat.





Mechanical Ventilation

Modes · initial settings · alarms and troubleshooting · bag if in doubt

6–8 mL/kg
predicted body weight

HOW A BREATH WORKS *four ideas that explain every setting on the machine*

TRIGGER, LIMIT, CYCLE

Every breath has a **trigger** (what starts it: the patient or the timer), a **limit** (the target it aims at), and a **cycle** (what ends it). Naming these three for a given mode tells you exactly how it will behave.

VOLUME VERSUS PRESSURE

Volume control: you set the tidal volume and **the pressure varies**, so watch for barotrauma. **Pressure control:** you set the pressure and **the volume varies**, so watch for a falling tidal volume as compliance worsens. **Whichever you set is guaranteed; the other one is the thing that can hurt you.**

THE TWO CIRCUITS

CO₂ is cleared by minute ventilation (tidal volume × rate). **Oxygenation is driven by FiO₂ and mean airway pressure**, which mostly means PEEP. **They are separate knobs:** a hypoxic patient does not need a faster rate, and a hypercapnic one does not need more PEEP.

MODES AND INITIAL SETTINGS

Mode	How it works	Typical use
A/C (volume or pressure)	Every triggered breath is fully supported , whether the patient or the timer starts it	The initial mode for almost everyone. Full rest for the respiratory muscles. Watch for breath stacking in a tachypneic patient
PSV (pressure support)	The patient triggers every breath ; you set the support pressure and PEEP	Weaning and spontaneous breathing trials. There is no backup rate, so it needs an intact respiratory drive
SIMV	Set mandatory breaths plus the patient's own, with or without pressure support	Largely superseded. Weaning by SIMV is slower than by daily spontaneous breathing trials

STARTING POINT, TYPICAL ADULT

A/C volume · tidal volume 6 to 8 mL/kg predicted body weight · rate 12 to 16 · FiO₂ 100% then wean to SpO₂ 92 to 96% · PEEP 5 cmH₂O · I:E 1:2 at a flow of about 60 L/min. Use 6 mL/kg if there is any suspicion of ARDS.

PREDICTED, NEVER ACTUAL, BODY WEIGHT

Lung size tracks height, not weight. Dosing tidal volume on actual weight in an obese patient delivers a massively injurious breath. **PBW depends only on height and sex**, so calculate it once at intubation and write it on the chart.

PEAK VERSUS PLATEAU *the single most useful bedside discrimination on a ventilator*

Pattern	What it means, and what to do
Peak high, plateau normal	An airway resistance problem: secretions, bronchospasm, a kinked or bitten tube, or a mucus plug. Suction, bronchodilate, unkink, place a bite block. The lung itself is fine.
Peak high, plateau high	A compliance problem: pneumothorax, pulmonary edema, ARDS, mainstem intubation, abdominal distension or compartment syndrome. Treat the cause and check tube depth , and reduce tidal volume.
Keep plateau ≤ 30 cmH ₂ O	Plateau pressure reflects alveolar distension, which is what injures lung. Measure it with an inspiratory hold. Peak pressure alone is misleading , because it includes resistance through the tube and airways.
Auto-PEEP	Breath stacking from incomplete exhalation. Check with an expiratory hold . Suspect it in COPD, asthma, and any high respiratory rate. It raises intrathoracic pressure, drops venous return, and is an under-recognized cause of hypotension and PEA arrest .

SUDDEN DETERIORATION *disconnect and bag with 100% oxygen first, then work through DOPES*

WHY BAG FIRST

Taking the patient off the ventilator and hand-bagging separates patient problems from machine problems immediately, relieves auto-PEEP, and tells you about compliance through your own hands. **Do it before troubleshooting the screen.**

DOPES

Displacement of the tube (too deep into a mainstem, out, or esophageal) · **O**bstuction (secretions, kink, biting, mucus plug) · **P**neumothorax · **E**quipment failure (circuit, oxygen supply, ventilator) · **S**tacking of breaths, meaning auto-PEEP.

THE LEVERS

Raise the PaO₂: increase FiO₂ or PEEP. **Lower the PaCO₂:** increase minute ventilation (rate or tidal volume). **Raise the PaCO₂:** reduce rate or tidal volume. **Watch out:** raising the rate shortens expiration and can create auto-PEEP, which makes things worse.

OBSTRUCTIVE DISEASE *asthma and COPD, where the enemy is expiration, not inspiration*

SETTINGS

Low rate of 8 to 10, tidal volume 6 mL/kg, high inspiratory flow with a short inspiratory time to lengthen expiration (I:E 1:4 to 1:5), and minimal PEEP. Accept **permissive hypercapnia** rather than chasing a normal CO₂, since a faster rate worsens the trapping that is causing the problem.

SUDDEN HYPOTENSION

Disconnect the circuit and let the patient exhale for 30 to 60 s. If the pressure recovers it was auto-PEEP; if it does not, suspect **tension pneumothorax** and decompress. **Reaching for fluid and vasopressors first misses both** and wastes the minutes that matter.

ARDS AND WEANING *the two settings changes with the clearest mortality evidence*

Situation	What to set	Why
ARDS	Tidal volume 6 mL/kg PBW, plateau ≤ 30, PEEP by an ARDSNet table, and permissive hypercapnia	ARMA, NEJM 2000 found lower tidal volume reduced mortality versus 12 mL/kg. This is the intervention on this sheet with the strongest evidence, and it is under-applied because tidal volume is dosed on the wrong weight.
Severe ARDS	Prone positioning for 16 h or more per day	Improves survival in P/F below 150 . Consider neuromuscular blockade early, and refer for ECMO if refractory.
Weaning	Daily awakening trial paired with a spontaneous breathing trial, then check cuff leak, secretions and cough	The pairing liberates patients sooner than either alone, and the commonest cause of excess ventilator days is nobody asking daily . An SBT tests the lungs, not the airway: a weak cough predicts reintubation despite perfect gas exchange.

PEARLS & PITFALLS

- **When in doubt, take them off the vent and bag.** It separates patient problems from machine problems.
- **Dose tidal volume on predicted body weight**, calculated from height, never on actual weight.
- **Rising peak with a stable plateau is resistance:** suction and bronchodilate, do not add PEEP.
- **Auto-PEEP causes hypotension and PEA.** Disconnect before you escalate treatment.

References: ARDS Network, ventilation with lower tidal volumes, *NEJM* 2000 · ATS / ACCP clinical practice guideline on liberation from mechanical ventilation · SCCM

PADIS guidelines · standard references on dynamic hyperinflation and ventilator waveform interpretation.

Educational aid; verify settings against local protocol and patient factors.





Weaning & SBTs

Liberation from mechanical ventilation · wake up and breathe, every day

Pair them
SAT then SBT

SCREEN EVERY PATIENT, EVERY DAY

The **commonest cause of excess ventilator days** is **nobody asking the question**. Protocolized daily screening liberates patients faster than clinician judgment alone, because the default otherwise is to leave a stable patient ventilated. **Every ventilated day carries its own risk**: pneumonia, delirium, weakness and sedation exposure all accumulate, so the question is not "are they ready?" but "why are they still on it?"

READINESS TO WEAN *all five should be trending the right way before you trial*

Domain	What you need
Cause	The underlying respiratory failure is resolving , not merely stable.
Oxygenation	PaO₂/FiO₂ above 150 to 200 on FiO₂ ≤ 0.4 with PEEP ≤ 5 to 8 . A patient needing high FiO ₂ or PEEP has not fixed the lung problem yet.
Hemodynamics	Stable, on minimal or no vasopressor , and no active ischemia . Weaning increases work of breathing and cardiac demand, so an unstable circulation fails the trial for cardiac reasons.
Airway	Adequate cough and manageable secretions . This is the criterion most often skipped, and the one that predicts reintubation.
Neurologic	Awake or easily rousable, initiating breaths, pH ≥ 7.25 . A patient who cannot protect an airway will fail after extubation regardless of gas exchange.

THE LIBERATION SEQUENCE *the pairing is what produces the benefit, not either trial alone*

- 1 Spontaneous awakening trial. Stop sedation and assess.** Girard, Lancet 2008 showed that **pairing the awakening trial with the breathing trial produced more ventilator-free days and lower one-year mortality** than the breathing trial alone. **A sedated patient cannot pass an SBT**, so an unpaired SBT frequently fails for the wrong reason and buys another day of ventilation.
- 2 Spontaneous breathing trial. Pressure support 5 to 8 with PEEP 5, or CPAP, or a T-piece. 30 min is enough for most patients** and is as good as 120 min; a longer trial mainly tires them out. **RSBI (respiratory rate divided by tidal volume in liters) under 105** predicts success, but **treat it as a screen and not a verdict**: a borderline RSBI with an otherwise passing trial should not block extubation.
- 3 Assess the airway separately, then extubate. A passed SBT tests the lungs, not the airway.** Confirm the patient is **alert, protecting the airway, coughing well and not drowning in secretions** before the tube comes out.

STOP THE SBT IF

RR above 35 or climbing · SpO₂ below 90% or PaO₂ below 60 on FiO₂ ≥ 0.5 · HR above 140 or a 20% change · new arrhythmia · SBP above 180 or below 90 · agitation, anxiety or diaphoresis · accessory muscle use or paradoxical abdominal breathing. A failed trial is information, not a setback: rest the patient fully overnight, find the reason, and retry in 24 h rather than repeatedly re-challenging a fatigued patient.

EXTUBATION, CUFF LEAK AND WHAT COMES AFTER

CUFF-LEAK TEST

Only in high-risk patients: prolonged or traumatic intubation, a large tube, or previous reintubation. **A leak under 110 mL, or under 10 to 15% of tidal volume, suggests laryngeal edema**. If it fails, give **methylprednisolone at least 4 h before extubation** rather than cancelling outright. **Do not test everyone**, since false positives delay extubation without benefit.

PROPHYLACTIC SUPPORT, NOT RESCUE

Apply NIV immediately after extubation in high-risk patients: ventilated over 24 h, COPD or hypercapnia, heart failure, obesity. **High-flow nasal oxygen** suits hypoxemic and high-risk patients. **NIV used as a rescue for established post-extubation failure delays reintubation and worsens outcomes**, so the timing is the whole point.

WHY THEY FAIL

Respiratory: secretions, bronchospasm, atelectasis, weak muscles. **Cardiac**: weaning is a stress test, and negative intrathoracic pressure raises venous return and left ventricular afterload, unmasking **ischemia or pulmonary edema**. **Neuromuscular**: ICU-acquired weakness, oversedation, delirium. **Metabolic**: low potassium, phosphate or magnesium, acidosis, malnutrition, poor sleep.

WHEN IT KEEPS FAILING *WIND categories, and what changes with each*

Category	Definition	What to do
Simple	Liberated on the first SBT	Nothing further. Most patients.
Difficult	Up to 3 SBTs, or within 7 days of the first	Work through the failure list above systematically rather than simply retrying. Correct electrolytes, treat cardiac contribution, mobilize.
Prolonged	More than 3 SBTs or beyond 7 days	Use a formal weaning protocol, fix every reversible cause, and prioritize nutrition and rehabilitation. Consider early tracheostomy , which improves comfort, reduces sedation need and eases the work of breathing, though it does not by itself shorten ventilation.

AFTER THE TUBE COMES OUT *the highest-risk window is the next 48 hours*

WATCH FOR EARLY FAILURE

Reintubation carries substantially higher mortality than the original intubation, and most failures declare within 48 h. **Watch for stridor, rising respiratory rate, accessory muscle use, a falling saturation and a rising CO₂**. Sit the patient up, give chest physiotherapy, suction, and treat bronchospasm and fluid overload early rather than waiting.

DO NOT LET FEAR OF FAILURE DELAY

A small reintubation rate is the sign of a correctly calibrated unit: a rate near zero means patients are being left intubated too long, which carries its own pneumonia, delirium and weakness burden. **Extubate when the criteria are met and manage the risk afterwards**, rather than waiting for certainty that never arrives.

PEARLS & PITFALLS

- **Pair the SAT with the SBT.** Sedation interruption is what most often unlocks a successful trial.
- **30 minutes is enough.** Longer trials tire the patient without improving the prediction.
- **A passed SBT tests the lungs, not the airway.** Assess mentation, cough and secretions separately.
- **Use NIV prophylactically in high-risk patients.** Waiting to rescue with it delays reintubation and costs lives.





Sedation & Delirium

Analgesia first · light sedation · the ABCDEF bundle · deeper is not safer

RASS 0 to -2
the target

RASS *Richmond Agitation-Sedation Scale, scored at every assessment*

Score	Term	Description
+4	Combative	Violent, an immediate danger to staff
+3	Very agitated	Pulls at or removes tubes and lines, aggressive
+2	Agitated	Frequent non-purposeful movement, fights the ventilator
+1	Restless	Anxious, but movements are not aggressive
0	Alert and calm	The goal in most patients
-1 to -2	Drowsy, light sedation	Awakens to voice, with eye contact sustained more than (-1) or less than (-2) 10 s. This is the target range
-3	Moderate sedation	Movement to voice but no eye contact
-4 to -5	Deep, unarousable	Responds only to physical stimulus, or not at all. Reserve for a specific indication such as severe ARDS with paralysis, refractory status, or raised intracranial pressure

ASSESS PAIN AND DELIRIUM SEPARATELY *agitation is a symptom, and the reflex to sedate it treats the wrong thing*

PAIN FIRST

Score pain before adding any sedative, using BPS or CPOT in a patient who cannot self-report. **Agitation is far more often untreated pain than inadequate sedation**, and treating it with propofol leaves the patient in pain and unable to say so.

CAM-ICU FOR DELIRIUM

Assess only if RASS is -3 or above. Delirium = (1 and 2) plus (3 or 4): 1. acute onset or fluctuating course · 2. inattention · 3. altered level of consciousness (any RASS other than 0) · 4. disorganized thinking. Screen **every shift**.

THE COMMONEST SCORING ERROR

Do not run CAM-ICU on a deeply sedated patient at RASS -4 or -5. The result is meaningless, because you are testing the sedation rather than the brain. **Lighten first, then assess**, which is precisely what the daily awakening trial is for.

THE ABCDEF BUNDLE *higher compliance is associated with lower mortality, and it works as a bundle, not a menu*

Element	What it means in practice
A · Assess and manage pain	Score it, treat it, and reassess. Analgesia precedes sedation , always.
B · Both SAT and SBT	Pair the spontaneous awakening trial with the spontaneous breathing trial daily . Paired is better than either alone, because a patient who is still sedated cannot pass a breathing trial.
C · Choice of sedation	Light sedation with a non-benzodiazepine . Propofol or dexmedetomidine over benzodiazepines, which are the most modifiable risk factor for delirium .
D · Delirium	Screen every shift, then treat causes and use non-pharmacologic measures first .
E · Early mobility	Mobilize even intubated patients. Reduces delirium duration and ICU-acquired weakness , and is among the highest-yield elements.
F · Family engagement	Family at the bedside reorients the patient better than any drug, and improves their own outcomes too.

DRUG DOSES *adult infusions, titrated to the lowest effective dose*

Agent	Dose	Notes
Propofol	5 to 50 mcg/kg/min	Fast on and off, which makes daily awakening easy. Watch hypotension, hypertriglyceridemia , and propofol infusion syndrome at high dose and long duration. Count its calories in the nutrition plan .
Dexmedetomidine	0.2 to 1.4 mcg/kg/h	Light, rousable sedation with no respiratory depression , so it can continue through extubation. Associated with less delirium than benzodiazepines . Causes bradycardia and hypotension , and it is not adequate for deep sedation.
Fentanyl	25 to 100 mcg/h, bolus 25 to 50	The analgesia-first workhorse. Accumulates in prolonged infusion because it redistributes into fat.
Morphine	1 to 5 mg/h, bolus 2 to 4 mg	Avoid in renal failure , where the active metabolite accumulates and causes prolonged sedation and myoclonus.
Ketamine	0.1 to 0.5 mg/kg/h	Opioid-sparing adjunct that preserves respiratory drive and bronchodilates.
Midazolam	0.02 to 0.1 mg/kg/h	Reserve for alcohol withdrawal, status epilepticus, or deliberate deep sedation . Accumulates badly in renal and hepatic impairment, and is the drug most implicated in ICU delirium.
Haloperidol	2 to 5 mg IV as needed	For dangerous agitation only, never as prophylaxis . It does not prevent or shorten delirium. Watch the QTc .

MANAGING DELIRIUM *almost everything that works is not a drug*

TREAT THE CAUSES

Pain, hypoxia, sepsis, sodium and glucose derangement, withdrawal, urinary retention and constipation. Then **stop the culprits**: benzodiazepines and anticholinergics. Delirium is a symptom of something, and the something is usually on this list.

NON-PHARMACOLOGIC FIRST

Reorient, restore the day-night cycle, protect sleep, give back glasses and hearing aids, mobilize early, involve family, and remove every line and tube you no longer need. These outperform any drug.

MULTIMODAL, OPIOID-SPARING ANALGESIA

Scheduled acetaminophen, ketamine, regional or neuraxial blocks, dexmedetomidine, and gabapentinoids for neuropathic pain, reserving opioids for boluses. **Less opioid means less delirium, less ileus and fewer ventilator days**.

PEARLS & PITFALLS

- **Pain first.** Agitation is often untreated pain, not inadequate sedation.
- **Deeper is not safer.** Over-sedation prolongs ventilation, delirium and ICU stay.
- **Do not run CAM-ICU at RASS -4 or -5.** Lighten first, then assess.
- **Antipsychotics neither prevent nor shorten delirium.** Benzodiazepines are the most modifiable risk.

References: SCCM clinical practice guidelines for the prevention and management of pain, agitation / sedation, delirium, immobility and sleep disruption in adult ICU patients (PADIS) · SCCM ICU Liberation and the ABCDEF bundle · Sessler et al, Richmond Agitation-Sedation Scale, 2002 · Ely et al, CAM-ICU, 2001.

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.





VASOPRESSORS

Squeeze the pipes · receptor pharmacology, dosing and the escalation ladder

NOREPINEPHRINE FIRST
then vasopressin, then epinephrine

WHICH RECEPTOR, WHICH EFFECT *what you are actually turning on*

α_1

Vascular smooth muscle.
Vasoconstriction, raises SVR and blood pressure. The main effect you want.

β_1

Myocardium. **Modest inotropy and chronotropy.**
Useful, but also the source of tachyarrhythmia.

β_2

Vessels and bronchi.
Vasodilation — unwanted here, and why epinephrine can drop the diastolic early.

V_1

Vasopressin receptor. **Non-adrenergic, so it still works in acidosis** when catecholamine receptors are failing.

AT_1

Angiotensin II receptor. **A third, renin-axis route to vasoconstriction** when catecholamines and V_1 are maxed.

AGENT ACTIVITY & DOSING *receptor strength +++ strong to – none*

Agent	α_1	β_1	Other	Dose	First choice for
Norepinephrine	+++	++	–	0.05–0.5 mcg/kg/min (no ceiling; titrate)	Septic and most vasodilatory or undifferentiated shock. Start here unless you have a specific reason not to.
Vasopressin	–	–	V_1 +++	0.03 U/min fixed — do not titrate	Catecholamine-sparing add-on to norepinephrine. Fixed because higher rates were not studied and cause digital and mesenteric ischemia.
Epinephrine	+++	+++	β_2 ++	0.01–0.5 mcg/kg/min	Anaphylaxis, cardiac arrest, refractory septic shock. Raises lactate independently of perfusion , so lactate clearance stops being a usable target.
Phenylephrine	+++	–	–	0.5–9 mcg/kg/min; push-dose 50–200 mcg	When tachyarrhythmia limits norepinephrine, and for transient hypotension. Pure vasoconstriction, so it can drop cardiac output — avoid in low-output states.
Angiotensin II	–	–	AT_1 +++	start 20 ng/kg/min	Catecholamine-refractory vasodilatory shock. Raises MAP reliably; watch for thrombosis and give VTE prophylaxis.
Dopamine	dose-dep	++	D_1 (low)	2–20 mcg/kg/min	Rarely. Only select bradycardic shock. Twice the arrhythmia rate of norepinephrine (24.1% vs 12.4%); no overall mortality difference, but worse mortality in the cardiogenic subgroup.
Methylene blue	–	–	SGC inhib	1–2 mg/kg IV over 20–30 min	Rescue for refractory vasoplegia , classically post-cardiopulmonary bypass. Inhibits soluble guanylate cyclase , blocking the nitric oxide–cGMP pathway that keeps vessels dilated. Avoid in G6PD deficiency and with serotonergic drugs.

THE SEPTIC SHOCK LADDER *for MAP below 65 despite adequate fluid*

- 1 Norepinephrine, first-line.** Titrate to **MAP $\geq$ 65**. **Start it peripherally through a good proximal line rather than wait for central access** — the delay costs more than the line does. Surviving Sepsis 2026 makes this a **strong recommendation over dopamine, epinephrine and selexpressin**; only the comparison against vasopressin or angiotensin II is a conditional suggestion, so this is not a general downgrade of norepinephrine.
- 2 Add vasopressin 0.03 U/min** once norepinephrine reaches roughly **0.25–0.5 mcg/kg/min**. It is added to spare catecholamine load, not to rescue a falling pressure — **if the patient is still deteriorating, escalate a different agent rather than turning the vasopressin up.**
- 3 Add epinephrine** if MAP remains inadequate on norepinephrine plus vasopressin. **Angiotensin II is the alternative** at this rung for catecholamine-refractory vasodilatory shock.
- 4 Hydrocortisone 200 mg/day** (50 mg q6h or by infusion) for shock with an ongoing vasopressor requirement. **Then stop and reassess:** is the source controlled, is there an unrecognized adrenal insufficiency, tamponade, acidosis or hypocalcemia? Reassess perfusion daily and de-escalate.

VASOPRESSOR LOAD *norepinephrine equivalents — get the vasopressin conversion right*

THE CONVERSION

NEE (mcg/kg/min) = norepinephrine + epinephrine + phenylephrine/10 + dopamine/100 + (vasopressin U/min $\times$ 2.5). So **vasopressin at its fixed 0.03–0.04 U/min contributes only about 0.075–0.1 mcg/kg/min of norepinephrine equivalent.** It is a small, fixed contribution — **do not read 0.04 U/min as anything like 1 mcg/kg/min of norepinephrine.**

WHAT COUNTS AS REFRACTORY

NEE above roughly **0.5 mcg/kg/min** marks refractory shock and should trigger a deliberate stop: escalate the next agent, add steroid, and **re-examine the reversible contributors — uncontrolled source, severe acidosis, hypocalcemia, and adrenal insufficiency.** Rising requirement is a prompt to re-diagnose, not simply to titrate harder.

AT THE BEDSIDE

PERIPHERAL & EXTRAVASATION

Norepinephrine may run short-term through a **good proximal peripheral line** while central access is arranged. **Watch the site hourly.** The antidote for extravasation is **phentolamine** infiltrated locally (topical nitroglycerin is an alternative).

MAP TARGET

$\geq$ 65 mmHg, and **60–65 is acceptable in patients over 65**, in whom permissive hypotension avoided excess vasopressor exposure. Individualize upward in chronic hypertension. **Titrate to perfusion, not to the number:** lactate clearance, urine output, mentation.

MONITOR & COMPLICATIONS

Arterial line for accurate pressure once you are escalating. Watch for **tachyarrhythmia, digital and mesenteric ischemia, and hyperglycemia.** **Reassess the need daily and wean deliberately** — wean norepinephrine first and stop vasopressin last.

PEARLS & PITFALLS

- Vasopressin 0.04 U/min is worth about 0.1 mcg/kg/min of norepinephrine**, not 1. Overstating it by tenfold makes a patient look far better supported than they are.
- Vasopressin is fixed-dose.** Do not titrate it up as a rescue — escalate a different agent instead.
- Start peripherally rather than wait for a central line.** Withholding pressors for access is the commoner harm.
- A pure vasoconstrictor in a low-output state can make things worse** by raising afterload — if the problem is the pump, an inotrope belongs alongside the pressor.
- Norepinephrine is first-line for nearly all vasodilatory and undifferentiated shock** — reach for it early rather than loading more fluid.
- Epinephrine raises lactate directly** via aerobic glycolysis, so you lose lactate clearance as a resuscitation target. Use ScvO₂, echo or capillary refill.
- Refractory shock is a prompt to re-diagnose:** source, acidosis, calcium, adrenal axis — before simply titrating higher.
- Wean norepinephrine first and stop vasopressin last.** Coming off the fixed-dose agent first is a common cause of an avoidable rebound in requirement.





Inotropes

Drive the pump · contractility, cardiac output and the inodilator trade-off

Perfusion
not a number

BEFORE YOU START ONE

An inotrope is a bridge, not a treatment. No inotrope has been shown to improve survival, and several increase mortality with prolonged use, because **every one of them buys contractility at the cost of myocardial oxygen demand and arrhythmia. The question is always what the inotrope is bridging to:** revascularization, valve surgery, recovery from a reversible insult, mechanical support, or transplant. **Use the lowest effective dose for the shortest time, and confirm the patient is actually volume-replete and adequately perfused in terms of pressure first,** because an inotrope cannot fix an empty ventricle or a vasoplegic circulation.

HOW THEY WORK *four different routes to the same intracellular calcium*

Class	Mechanism	Agents, and what it means clinically
Beta-1 agonist	Raises cAMP, so more calcium enters and contractility rises	Dobutamine, epinephrine, dopamine. Blunted in patients on chronic beta-blockers, because the receptor is occupied, and blunted again by receptor downregulation in chronic heart failure.
PDE-3 inhibitor	Blocks cAMP breakdown, so it works downstream of the beta receptor	Milrinone. An inodilator : it raises output and lowers both systemic and pulmonary vascular resistance. Still effective in the beta-blocked patient, which is its main advantage.
Calcium sensitizer	Increases myofilament sensitivity to calcium without raising calcium itself	Levosimendan. Not approved by the FDA and unavailable in the United States, though widely used in Europe. Effects persist for days through an active metabolite.
Na/K-ATPase inhibitor	Raises intracellular sodium and therefore calcium	Digoxin. Mild inotropy with AV nodal slowing. Useful in AF with heart failure, and it does not drop the blood pressure, but it is slow in onset and accumulates in renal impairment.

AGENTS AND DOSING

Agent	CO	SVR	Dose	Best for
Dobutamine <i>first-line</i>	↑↑	↓	2 to 20 mcg/kg/min	Cardiogenic shock with adequate pressure, septic myocardial dysfunction once the MAP is restored. Quick on and off, which makes it forgiving.
Milrinone	↑	↓↓	0.125 to 0.75 mcg/kg/min	RV failure and pulmonary hypertension, because it dilates the pulmonary vasculature and unloads the RV, and the beta-blocked patient. Accumulates in renal failure.
Epinephrine	↑↑	↑	0.01 to 0.5 mcg/kg/min	Refractory low output and post-cardiac-surgery. Beta effects predominate at low dose, alpha at higher dose. Causes lactate rise and hyperglycemia through beta-2 effects, which is expected and not by itself a sign of worsening perfusion.
Dopamine	↑	~	2 to 20 mcg/kg/min	Rarely used now. Caused more arrhythmias than norepinephrine, and higher mortality in the cardiogenic shock subgroup of a large randomized trial. "Renal-dose" dopamine does not protect the kidney and should not be used.
Levosimendan	↑	↓	0.05 to 0.2 mcg/kg/min	Decompensated or beta-blocked heart failure. Not available in the US. Its long-acting metabolite means a dosing error keeps acting for days.

WHICH ONE, AND WHEN *decide from the blood pressure and the ventricle that is failing*

Situation	Choice, and why
Low output, adequate MAP	Dobutamine first-line, or milrinone. There is room to accept some vasodilation, and the priority is flow.
Low output with hypotension	Norepinephrine to restore the MAP first, then add an inotrope. Starting an inodilator into a hypotensive patient drops the pressure further and worsens coronary perfusion of the very ventricle you are trying to help.
RV failure or pulmonary hypertension	Milrinone or dobutamine, paired with norepinephrine. The RV is perfused throughout the cardiac cycle and is exquisitely sensitive to systemic pressure, so maintaining the MAP is as important as the inotrope. Add inhaled pulmonary vasodilators where available.
On chronic beta-blockade	Milrinone, which bypasses the occupied beta receptor entirely. Escalating dobutamine against a blocked receptor mostly produces tachycardia.
Septic myocardial dysfunction	Dobutamine, only after the MAP is restored with fluid and norepinephrine and evidence of low output persists. Most septic shock does not need an inotrope.

HANDLE WITH CARE

INODILATORS DROP THE PRESSURE

Milrinone and dobutamine both lower systemic vascular resistance, so in a marginal patient the MAP falls even as cardiac output rises. **Pair them with norepinephrine** rather than abandoning the inotrope.

ARRHYTHMIA AND OXYGEN DEMAND

Every beta-agonist raises heart rate, is arrhythmogenic, and increases myocardial oxygen consumption. In ischemic cardiogenic shock that is a real trade-off. **Monitor the rhythm continuously and titrate to the lowest effective dose.**

TITRATE TO PERFUSION

Reassess with lactate clearance, urine output, mentation, capillary refill and ScvO₂, not with a target cardiac index. **Driving the number rather than the patient is how inotrope-related harm happens,** and a good number with cold peripheries and a rising lactate is not success.

PEARLS & PITFALLS

- **Fix pressure and volume before adding an inotrope.** It cannot fill an empty ventricle.
- **Milrinone for the beta-blocked patient,** because it works downstream of the receptor.
- **Milrinone accumulates in renal failure,** and levosimendan keeps acting for days.
- **An inotrope is a bridge.** If you cannot name what it is bridging to, reconsider starting it.





Acute Kidney Injury

KDIGO staging · find the cause · protect what is left · when to start RRT

Indications
not a stage number

DEFINE AND STAGE *KDIGO: any one criterion defines it, and you stage by whichever is worst*

Stage	Serum creatinine	Urine output
Definition	≥ 0.3 mg/dL rise within 48 h, or ≥ 1.5 × baseline known or presumed within 7 days	< 0.5 mL/kg/h for 6 h
Stage 1	1.5 to 1.9 × baseline, or a ≥ 0.3 mg/dL rise	< 0.5 mL/kg/h for 6 to 12 h
Stage 2	2.0 to 2.9 × baseline	< 0.5 mL/kg/h for ≥ 12 h
Stage 3	≥ 3.0 × baseline, or ≥ 4.0 mg/dL, or RRT started	< 0.3 mL/kg/h for ≥ 24 h, or anuria ≥ 12 h

WHY URINE OUTPUT IS THE EARLY SIGNAL

Creatinine lags injury by 24 to 48 h, so a normal value never excludes evolving AKI, and **the urine output criterion is usually met first**. **Baseline matters**: without one, a single "normal-looking" creatinine under-stages a patient whose true baseline was 0.5. **Fluid overload dilutes creatinine and falsely reassures** while independently worsening outcomes, so a rising weight with a flat creatinine is not good news.

FIND THE CAUSE *pre-renal, intrinsic, post-renal: the urine tells you which*

Category	Causes, and how to confirm
Pre-renal <i>commonest in ICU</i>	Hypovolemia, sepsis with vasodilation, cardiorenal and hepatorenal syndromes, abdominal compartment syndrome. FENa < 1% and bland sediment. Remember venous congestion : a congested, high-CVP patient is pre-renal from poor renal <i>outflow</i> , and that one needs diuresis rather than fluid.
Intrinsic	ATN (ischemic or septic, the common endpoint), nephrotoxins , AIN , glomerulonephritis, rhabdomyolysis, tumor lysis. FENa > 2% . Urine microscopy is the cheapest test that changes management : muddy-brown granular casts mean ATN, RBC casts mean glomerulonephritis, white cells and eosinophils suggest AIN, and a bland sediment points back to pre-renal.
Post-renal	Obstruction: a blocked catheter first, then stones, prostate or pelvic mass. Flush or replace the catheter and do a bladder scan before anything else , because it is the one cause that reverses in minutes. Ultrasound if still unclear.
Caveats	FENa is invalid on diuretics : use FEurea < 35% instead. Also send CK if rhabdomyolysis is plausible, review every drug level, and get a renal ultrasound when the cause is not obvious.

PROTECT WHAT IS LEFT *there is no drug that treats AKI, so prevention is the treatment*

DO

Optimize volume and perfusion, targeting **MAP ≥ 65** and higher in chronic hypertension, whose autoregulation is shifted. **Stop nephrotoxins**: NSAIDs, aminoglycosides, and ACE inhibitors or ARBs while shocked. **Use balanced crystalloids over high-chloride saline**, since a chloride load causes renal vasoconstriction. **Renally dose everything** and monitor vancomycin and aminoglycoside levels.

DO NOT

No renal-dose dopamine, which does not protect the kidney and adds arrhythmia. **No diuretics to prevent or treat AKI**: converting oliguric to non-oliguric AKI makes the chart look better without changing the outcome. **Avoid starches. Do not withhold contrast for a genuinely indicated scan**: the risk of contrast nephropathy is smaller and more reversible than the risk of missing the diagnosis.

AND DE-RESUSCITATE

Fluid overload is an independent predictor of death in AKI, and it accumulates quietly once the initial resuscitation is over. **Once shock has resolved, shift from giving fluid to removing it**. Watch for **abdominal compartment syndrome** in the distended, oliguric patient.

RENAL REPLACEMENT THERAPY *start for an indication, not for a stage*

Question	Answer	Why
Start now if	Refractory hyperkalemia · refractory acidosis · refractory fluid overload or pulmonary edema · uremic pericarditis or encephalopathy · selected dialyzable poisons	These are emergencies that will not wait for a trend. The mnemonic is AEIOU : Acidosis, Electrolytes, Intoxication, Overload, Uremia.
Timing otherwise	Do not start early just because the stage is high	START-AKI, NEJM 2020 and AKIKI, NEJM 2016 found no benefit from an accelerated start , and many patients recover without ever needing it. But do not over-delay either : AKIKI 2, Lancet 2021 found harm from waiting beyond a strict threshold. Watch for an indication, and act when one appears .
Modality	CRRT for hemodynamic instability, raised ICP, or when slow steady clearance matters. IHD or SLED when stable, or when rapid clearance is needed	CRRT is gentler on the blood pressure and avoids the osmotic swings that worsen cerebral edema. Regional citrate anticoagulation is preferred for CRRT circuits, since it prolongs filter life without systemically anticoagulating the patient.

AFTER THE AKI *an episode of AKI is a permanent change to the patient's risk, not a resolved event*

IT DOES NOT END AT DISCHARGE

AKI substantially raises the long-term risk of CKD, recurrent AKI and death, even when the creatinine returns to baseline. Document the episode explicitly in the discharge summary, because it is routinely lost and the next clinician then prescribes as if the kidneys were never injured.

WHAT TO ARRANGE

Recheck creatinine within 1 to 3 months to establish the new baseline. **Review every drug held during the admission** and restart deliberately rather than by default: an ACE inhibitor or metformin stopped for AKI is frequently never restarted, or restarted unmonitored. **Refer to nephrology** if renal function has not recovered, there is significant proteinuria, or the cause remains unexplained.

PEARLS & PITFALLS

- **Creatinine lags by 24 to 48 h**. A normal value does not exclude evolving AKI; follow the urine output.
- **Flush the catheter and scan the bladder first**. Obstruction is the one cause that reverses immediately.

- **Start RRT for an indication, not a stage**. Early initiation has repeatedly failed to help.
- **Do not use diuretics or dopamine as a strategy**. More urine is not better kidneys.

References: KDIGO clinical practice guideline for acute kidney injury · START-AKI Investigators, *NEJM* 2020 · Gaudry et al, AKIKI, *NEJM* 2016 and AKIKI 2, *Lancet* 2021 ·

Zarbock et al, ELAIN, *JAMA* 2016 · Semler et al, SMART, *NEJM* 2018.

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





Acid-Base Interpretation

A reproducible stepwise read of any blood gas · six steps, every time

6 steps
every gas

NORMAL VALUES *arterial*

pH	PaCO ₂	HCO ₃ ⁻	Anion gap
7.35 to 7.45	35 to 45 mmHg	22 to 26 mEq/L	3 to 11 mEq/L

THE STEPWISE APPROACH *run all six in order, and a mixed disorder cannot hide from you*

- 1 **Look at the pH.** Below 7.35 is an acidemia, above 7.45 an alkalemia. **A normal pH does not mean a normal gas:** it is the classic appearance of a mixed disorder in which two processes cancel out.
- 2 **Identify the primary driver.** Which of PaCO₂ or HCO₃ has moved in the direction that **explains** the pH? That one is primary; the other is compensating.
- 3 **Check compensation against the expected value** from the formulas below. **Compensation is never complete and never overshoots**, so if the measured value differs from expected, **a second disorder exists**. This step is what converts a gas from a label into a diagnosis.
- 4 **Calculate the anion gap on every gas**, whatever the pH, and **correct it for albumin**. A raised gap can be the only clue to a serious second process in a patient whose pH looks fine.
- 5 **If the gap is raised, calculate the delta ratio** to expose a coexisting non-gap acidosis or metabolic alkalosis.
- 6 **Correlate with the patient.** Match the pattern to the history: vomiting, DKA, sepsis, COPD, toxins, diuretics. **A gas that does not fit the story means you have the wrong story or an extra disorder.**

COMPENSATION FORMULAS *measured off expected means a second disorder*

Primary disorder	Expected compensation
Metabolic acidosis	Winter's formula: expected PaCO₂ = (1.5 × HCO₃) + 8, ± 2. A PaCO ₂ higher than expected means a coexisting respiratory acidosis (they are tiring); lower means a coexisting respiratory alkalosis.
Metabolic alkalosis	expected PaCO ₂ = (0.7 × HCO ₃) + 21
Acute respiratory acidosis	HCO ₃ rises 1 per 10 mmHg rise in PaCO ₂
Chronic respiratory acidosis	HCO ₃ rises 3.5 to 4 per 10 mmHg rise in PaCO ₂
Acute respiratory alkalosis	HCO ₃ falls 2 per 10 mmHg fall in PaCO ₂
Chronic respiratory alkalosis	HCO ₃ falls 4 to 5 per 10 mmHg fall in PaCO ₂
Acute versus chronic	Acute is minutes to hours and reflects buffering alone. Chronic means renal compensation is complete, which takes 3 to 5 days and produces a much larger bicarbonate shift. Using the acute formula on a chronic retainer invents a metabolic alkalosis that is not there.

ANION GAP AND DELTA RATIO

ANION GAP

AG = Na – (Cl + HCO₃), normal **about 3 to 11** on modern ion-selective analyzers, which read chloride higher than older methods; the classic **12 ± 4** anchors the delta ratio. It represents unmeasured anions: **lactate, ketones, toxins, urate and sulfate. Always correct for albumin: add 2.5 mEq/L for every 1 g/dL the albumin sits below 4.** Uncorrected, a hypoalbuminemic ICU patient with a genuinely raised gap will read as normal, the commonest way a lactic acidosis is missed on the numbers.

DELTA RATIO

Use only when there is a high-anion-gap metabolic acidosis. (AG – 12) / (24 – HCO₃). **< 0.4** pure non-gap acidosis · **0.4 to 1** high-AG plus a non-gap acidosis · **1 to 2** pure high-AG acidosis · **> 2** high-AG acidosis plus a metabolic alkalosis, or a chronic respiratory acidosis. It works because a pure gap acidosis consumes one bicarbonate for each unmeasured anion added.

CAUSES BY DISORDER *acidosis by mnemonic, alkalosis by urine chloride*

Pattern	Causes
High-AG acidosis <i>MUDPILES</i>	M ethanol · U remia · D KA and other ketoacidoses · P ropylene glycol · I ron, Isoniazid · L actic acidosis · E thylene glycol · S alicylates. Lactate and ketones account for most of what you will actually see.
Non-gap acidosis <i>HARDUPS</i>	H yperalimentation · A ddison, Acetazolamide · R TA · D iarrhea, the commonest cause · U reteral diversion · P ost-hypocapnia, P ancreatic fistula · S aline, S pironolactone. Urine anion gap separates renal from GI losses: negative in diarrhea, positive in RTA.
Alkalosis <i>saline-responsive</i>	Urine chloride below 20. Vomiting or NG suction, diuretics (between doses), contraction, post-hypercapnia. Treat with 0.9% saline plus potassium chloride , since the deficit is chloride and volume.
Alkalosis <i>saline-resistant</i>	Urine chloride above 20. Hyperaldosteronism (Conn syndrome), Cushing syndrome, severe hypokalemia, Bartter and Gitelman syndromes, licorice. Saline will not fix these; treat the cause.

MIXED AND TRIPLE DISORDERS *you can have two metabolic disorders at once, but only ever one respiratory*

Setting	High-AG acidosis	Alkalosis	Respiratory
Vomiting alcoholic	Alcoholic ketoacidosis	Vomiting	Respiratory alkalosis (withdrawal or sepsis)
DKA with vomiting and sepsis	Ketoacidosis	Vomiting, NG losses	Respiratory alkalosis (sepsis)
Salicylate overdose	Salicylate	Vomiting	Respiratory alkalosis (direct central stimulation)
COPD with sepsis on diuretics	Lactic acidosis	Loop diuretics	Respiratory acidosis (CO ₂ retention)
The common doubles	Salicylate = high-AG acidosis + respiratory alkalosis · DKA or AKA with vomiting = high-AG acidosis + metabolic alkalosis · COPD on diuretics = respiratory acidosis + metabolic alkalosis.		

PEARLS & PITFALLS

- **Calculate the anion gap on every gas.** A normal pH with a raised gap signals a hidden mixed disorder.
- **A chronic respiratory disorder shifts bicarbonate far more than an acute one.** Do not mislabel that as metabolic.
- **Urine chloride splits metabolic alkalosis** into the kind saline fixes and the kind it will not.

References: Berend et al, physiological approach to assessment of acid-base disturbances, *NEJM* 2014 · Kraut & Madias, serum anion gap and its uses and limitations · Albert, Dell & Winters, quantitative displacement of acid-base equilibrium in metabolic acidosis, *Ann Intern Med* 1967 · Emmett, approach to mixed acid-base disorders.
Educational aid; verify doses against local protocol, renal function, and patient factors.





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), euvolemic (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.





DKA & HHS

The two hyperglycemic emergencies · ADA/EASD 2024: beta-hydroxybutyrate in, the anion gap out

BHB, NOT THE GAP
for diagnosis and resolution

WHAT CHANGED IN 2024 *the first major revision since 2009*

THE ANION GAP IS OUT

Quantitative beta-hydroxybutyrate replaces the anion gap for both diagnosis and resolution. **Large-volume saline produces a hyperchloremic normal-gap acidosis** that keeps the gap open after the ketosis has cleared, which delays transition to subcutaneous insulin. The gap retains a role **only where ketone testing is unavailable**.

THE GLUCOSE BAR IS LOWER

Glucose ≥ 200 mg/dL or a prior history of diabetes now satisfies the hyperglycemia criterion. This deliberately captures **euglycemic DKA**, which the old threshold of 250 excluded, and which is increasingly common with **SGLT2 inhibitors**.

BALANCED CRYSTALLOID PREFERRED

Where available, balanced crystalloid is now favored over 0.9% saline, being associated with **faster DKA resolution, less hyperchloremic acidosis and shorter length of stay**. Saline remains acceptable and is still what most units reach for first.

DIAGNOSE *three criteria for DKA, four for HHS*

Parameter	DKA	HHS
Glucose	≥ 200 mg/dL (11.1 mmol/L) OR a prior history of diabetes	≥ 600 mg/dL (33.3 mmol/L)
Ketones	BHB ≥ 3.0 mmol/L, or urine ketones 2+ or more	BHB < 3.0 mmol/L, or urine ketones under 2+
Acid-base	pH < 7.3 and/or bicarbonate < 18 mmol/L	pH ≥ 7.3 and bicarbonate ≥ 15 mmol/L
Osmolality	Variable	Effective osmolality > 300 mOsm/kg ($2 \times \text{Na} + \text{glucose}$, in mmol/L), or total > 320

Overlap is common — a substantial minority present with mixed features and are managed as DKA. **Precipitants, in rough order:** infection, **insulin non-adherence or interruption**, new-onset diabetes, myocardial infarction or stroke, pancreatitis, and drugs including steroids, SGLT2 inhibitors and antipsychotics. **Look for the precipitant in every case;** treating the metabolic picture alone is why people relapse.

GRADE THE SEVERITY *it decides the route of insulin and the level of care*

Severity	BHB	pH	Bicarbonate	Mental state and route
Mild	≤ 6 mmol/L	> 7.25	≥ 15 mmol/L	Alert. Suitable for subcutaneous rapid-acting insulin with close nursing supervision, in the emergency department or a step-down unit.
Moderate	≤ 6 mmol/L	7.0–7.25	10 to < 15 mmol/L	Alert or drowsy. Uncomplicated cases can also be managed outside intensive care if monitoring is adequate.
Severe	> 6 mmol/L	< 7.0	< 10 mmol/L	Stupor or coma. Intravenous infusion and a higher level of care.

Not every variable has to be met to assign a grade, and the placement decision remains clinical. **Ketones alone do not separate mild from moderate** — use the pH, bicarbonate and mental state.

THE THREE PILLARS *fluids, potassium, then insulin — in that order*

- Fluids first.** Isotonic saline or balanced crystalloid at 500–1,000 mL/h for the first 2 to 4 hours, aiming to replace about half the deficit in 8 to 12 hours. Use smaller 250 mL boluses in older patients and in heart or kidney disease. **A rising sodium as the glucose falls is expected and is not by itself a reason to give hypotonic fluid** — 0.45% saline is indicated only if osmolality fails to decline despite adequate positive fluid balance and appropriate insulin.
- Check potassium before insulin.** Below 3.5 mmol/L: withhold insulin and replace at 10–20 mmol/h until above 3.5. 3.5 to 5.0: start insulin and add potassium to the fluids. Above 5.0: start insulin, give no potassium, and recheck in 2 hours. Insulin drives potassium into cells, and giving it into an unrecognized hypokalemia can cause fatal arrhythmia.
- Insulin.** DKA: fixed-rate infusion at 0.1 units/kg/h — a bolus is not required. Mild DKA can be treated with subcutaneous rapid-acting analogue (about 0.1 units/kg every 2 hours) where supervision allows. HHS: 0.05 units/kg/h, targeting a glucose of 200 to 250 mg/dL and a controlled fall.
- Add dextrose when glucose falls below 250 mg/dL** — 5% or 10% alongside the crystalloid — and reduce insulin to 0.05 units/kg/h. In euglycemic DKA, start the dextrose at the same time as the insulin, from the outset. **Never stop the insulin to correct the glucose:** insulin is what clears the ketosis, so you feed the patient glucose and keep the insulin running.
- Bicarbonate and phosphate are not routine.** Consider bicarbonate only if pH is below 7.0. Phosphate replacement has not improved outcomes in randomized trials; give it only for a level under 1.0 mmol/L with muscle weakness or cardiac or respiratory compromise, and **replace slowly, since rapid correction causes hypocalcemia**.

RESOLUTION AND TRANSITION *the criteria that were rewritten*

WHEN IS IT RESOLVED?

DKA: BHB below 0.6 mmol/L, together with a venous pH of 7.3 or above or a bicarbonate of 18 or above; ideally the glucose is also under 200 mg/dL. **Do not use the anion gap, and do not use urine ketones** — as the acidosis improves, beta-hydroxybutyrate is converted to acetoacetate, so **urine ketones can persist or even rise in a patient who is recovering**. HHS: osmolality below 300 mOsm/kg, glucose under 250, urine output above 0.5 mL/kg/h, and improved cognition.

GETTING OFF THE INFUSION

Give subcutaneous basal insulin 1 to 2 hours before stopping the intravenous infusion and wait until the patient is eating. **Stopping the drip without that overlap causes rebound ketosis**, because intravenous insulin has a half-life of minutes. Estimate the total daily dose from 0.3 to 0.6 units/kg/day, the pre-admission regimen, or the infusion requirement, and use a **basal-bolus** regimen. **Before discharge, establish why it happened**, check injection technique and supply, and arrange diabetes follow-up.

PEARLS & PITFALLS

- Check potassium before the first unit of insulin. Below 3.5 you replace first — this is the commonest avoidable death.
- Do not chase the anion gap. Saline-induced hyperchloremic acidosis keeps it open and delays transition; follow the BHB.
- A rising sodium is expected as glucose falls. It is not a trigger for 0.45% saline on its own.
- Never stop the insulin when the glucose normalizes. Add dextrose and keep clearing the ketones.
- Euglycemic DKA is diagnosed on ketones and acidosis, not glucose. Ask about SGLT2 inhibitors and pregnancy.
- Overlap basal insulin 1 to 2 hours before stopping the drip, or the patient goes straight back into ketosis.

References: Umpierrez et al. Hyperglycemic Crises in Adults With Diabetes: A Consensus Report. ADA/EASD/JBDS/AACE/DTS,

Diabetes Care

2024;47:1257–1275 (and

Diabetologia

2024) · JBDS-IP guidance on DKA and HHS · trials of balanced crystalloid versus saline in DKA.

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





Nutrition in Critical Illness

When · what route · how much · and why less is more early on

Feed the gut
within 24–48 h

WHEN AND BY WHAT ROUTE *enteral first, early, and at a modest rate*

Decision	What to do, and why
Start early	Enteral nutrition within 24 to 48 h of admission in the hemodynamically stable patient. The point is not the calories: early feeding maintains gut mucosal integrity and reduces bacterial translocation, which is why it lowers infectious complications even at trophic volumes.
Delay or withhold	Uncontrolled shock on escalating vasopressors, bowel ischemia, obstruction, or a high-output fistula. Feeding a gut that is not being perfused risks non-occlusive mesenteric ischemia, so stabilize first. A stable patient on a modest fixed dose of norepinephrine can still be fed.
Enteral first	Gastric is the default, and works in most patients. Post-pyloric for high aspiration risk or repeated gastric intolerance. Do not delay feeding to arrange post-pyloric access.
Parenteral	Only when enteral fails or is contraindicated. Add supplemental PN around day 3 to 7 if enteral targets cannot be met. Early full PN increases infection without improving survival, which is why it is a fallback rather than a parallel option.

HOW MUCH *the modern answer is less than instinct suggests in the first week*

ENERGY

20 to 25 kcal/kg/day as the eventual target, but cap delivery at about 70% of that in the first few days and advance over 48 to 72 h. **Why:** critically ill patients generate substantial endogenous energy from catabolism, so full early feeding produces **overfeeding, hyperglycemia and impaired autophagy** on top of it.

PROTEIN

About 1.3 g/kg/day (ASPEN allows 1.2 to 2.0). **Protein is the component to protect when calories are restricted**, because muscle catabolism, not calorie deficit, is what drives ICU-acquired weakness. **Higher targets have not consistently improved outcomes**, and may harm in acute kidney injury.

TROPHIC FEEDING

A low-volume "trophic" rate is acceptable in the early phase, particularly in ARDS, where trophic and full feeding gave equivalent outcomes. **It preserves the gut without the metabolic cost**, and is a reasonable compromise when tolerance is marginal.

REFEEDING SYNDROME *the complication caused by the treatment, and it is predictable*

WHO IS AT RISK

Little or no intake for more than 5 days, low BMI, alcohol use disorder, chronic malnutrition, or recent significant weight loss. **Identify them before the first feed**, because the syndrome is entirely predictable from the history.

WHAT HAPPENS

Feeding triggers insulin release, which drives phosphate, potassium and magnesium into cells alongside glucose. **The hallmark is a falling phosphate after feeding starts**, with arrhythmia, weakness, respiratory failure and seizures if unrecognized.

WHAT TO DO

Start at 25 to 50% of goal, or 10 to 20 kcal/kg in very high risk, and advance slowly. **Give thiamine 200 to 300 mg before or with the first feed**, since refeeding drives thiamine-dependent metabolism. **Replace phosphate, potassium and magnesium aggressively and monitor them daily.** Do not stop feeding; slow it down and replace.

MONITORING AND SPECIAL SITUATIONS

Issue	What to do	Why
Gastric residual volumes	Routine measurement is not recommended, and do not stop feed for a GRV under 500 mL	GRV correlates poorly with aspiration and stopping feeds for it is a major cause of under-nutrition. Reserve it for the patient who is actually vomiting or distended.
Intolerance	Prokinetics first (metoclopramide or erythromycin), then post-pyloric access	Most intolerance is gastroparesis from illness, opioids and sedation, all of which are modifiable before abandoning the enteral route.
Aspiration risk	Head of bed 30 to 45 degrees, and good oral care	The single most effective mechanical measure, and it also reduces ventilator-associated pneumonia.
Obesity	Hypocaloric, high-protein: about 11 to 14 kcal/kg actual weight, protein up to 2.0 to 2.5 g/kg ideal body weight	Preserves lean mass while mobilizing fat stores, and avoids the overfeeding that dosing on actual weight would produce.
CRRT	Increase protein to 2.0 to 2.5 g/kg/day	Continuous filtration removes substantial amino acids, so standard targets under-deliver in exactly the patients least able to afford it.
Watch	Glucose, electrolytes, phosphate, and triglycerides if on PN	Propofol is a lipid emulsion, so count its calories in the daily total; it is a common hidden cause of overfeeding and hypertriglyceridemia.

DO NOT FORGET *the parts of feeding that are not calories*

MICRONUTRIENTS AND VITAMINS

Thiamine, folate and trace elements in the malnourished and in alcohol use disorder. **High-dose vitamin C, selenium and glutamine are not recommended** as routine immunomodulation; several trials found no benefit and some found harm.

GLYCEMIC CONTROL AND BOWELS

Target glucose 140 to 180 mg/dL; tighter control causes hypoglycemia without benefit. **Prescribe a bowel regimen from the start**, since opioids and immobility make constipation near-universal and it worsens feed tolerance and delirium.

KEEP FEEDING THROUGH INTERRUPTIONS

Cumulative fasting for procedures, imaging and extubation attempts is a major hidden cause of under-nutrition. **Use the shortest safe fasting window and restart promptly**, and consider a volume-based catch-up protocol rather than a fixed hourly rate.

PEARLS & PITFALLS

- **Feed the gut early, even at trophic volumes.** The benefit is mucosal integrity, not calories.
- **Thiamine before the first feed** in anyone at refeeding risk, and watch the phosphate daily.
- **Do not stop feeds for a gastric residual under 500 mL.** It is a leading cause of under-nutrition.
- **Count the propofol calories.** It is a lipid emulsion and a hidden source of overfeeding.

References: ASPEN / SCCM guidelines for nutrition support therapy in the adult critically ill patient · ESPEN guideline on clinical nutrition in the intensive care unit · EDEN

trial of trophic versus full enteral feeding in ARDS, *JAMA* 2012 · NICE guidance on identifying refeeding risk.

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





ICU Prophylaxis

VTE · stress ulcer · the daily checklist that catches what rounds miss

Every day
review, don't assume

VTE PROPHYLAXIS *critically ill patients are at high risk, and omission is the commonest preventable harm here*

PHARMACOLOGIC

Enoxaparin 40 mg SC once daily when CrCl is above 30. Reduce to 30 mg daily if CrCl is below 30, or use unfractionated heparin 5000 units SC BID to TID, which is not renally cleared and is easier to reverse. In obesity, consider a higher or twice-daily dose with anti-Xa guidance, since standard dosing under-anticoagulates at high body weight.

MECHANICAL

Intermittent pneumatic compression when drugs are contraindicated: active bleeding, platelets below 50, or an imminent procedure. Combine drug and mechanical prophylaxis in the highest-risk patients, such as major trauma and orthopedic surgery. Graduated stockings alone are not adequate and cause skin breakdown in the poorly perfused.

REASSESS, DO NOT SET AND FORGET

Contraindications change daily. A patient held for a procedure on Monday is frequently still not receiving prophylaxis on Thursday, because nobody restarted it. Prescribe a restart time at the same moment you hold it.

STRESS ULCER PROPHYLAXIS *genuinely indicated in a minority, and given to almost everyone*

Question	Answer
Who needs it	Mechanical ventilation expected beyond 48 h, or coagulopathy (platelets below 50, or INR above 1.5). Also consider in shock, high-dose corticosteroids, chronic liver disease and acute kidney injury. Being in an ICU is not itself an indication.
Which agent	Pantoprazole 40 mg IV daily is first-line, with famotidine 20 mg IV BID as an alternative. REVISE, NEJM 2024 found pantoprazole reduced clinically important upper GI bleeding to 1.0% from 3.5% in ventilated patients, without increasing pneumonia or C. difficile, which had been the long-standing concern.
When to stop	Deprescribe once the patient is extubated and tolerating enteral feeding, since enteral nutrition is itself protective and the indication has gone. The real harm is continuation: a prophylactic PPI started in the ICU is frequently still being taken a year after discharge, which is how a 5-day intervention becomes a permanent medication.

THE DAILY CHECKLIST *FAST HUGS BID, run on every patient at every round*

	Element	What you are actually checking
F	Feeding	Enteral nutrition started and at target? Enteral beats parenteral and protects the gut barrier.
A	Analgesia	Adequate and not excessive. Analgesia precedes sedation, always.
S	Sedation	Light target (RASS 0 to -2), with a daily awakening trial unless there is a specific reason not to.
T	Thromboprophylaxis	Prescribed, given, and restarted after any hold.
H	Head of bed	Elevated 30 to 45 degrees, which reduces aspiration and ventilator-associated pneumonia.
U	Ulcer prophylaxis	Indication reviewed, and stopped if it has gone.
G	Glucose	Target 140 to 180 mg/dL. Tighter control causes hypoglycemia without benefit.
S	Spontaneous breathing trial	Paired with the awakening trial. Every ventilated day is a decision, not a default.
B	Bowels	A regimen in place. Constipation from opioids and immobility is near-universal and worsens delirium and feeding tolerance.
I	Indwelling lines	Every catheter reviewed and removed if not needed. Risk of CLABSI and CAUTI accrues per device-day.
D	De-escalate	Narrow the antibiotics, set a stop date, and screen for delirium with CAM-ICU.

DRUG DOSES

Agent	Dose	Note
Enoxaparin	40 mg SC once daily	CrCl above 30. 30 mg daily if CrCl below 30.
Unfractionated heparin	5000 units SC BID to TID	Preferred in renal impairment (CrCl below 30) and when reversibility matters.
Dalteparin	5000 units SC daily	Alternative LMWH; does not require renal dose adjustment at prophylactic doses.
Pantoprazole	40 mg IV once daily	First-line stress ulcer prophylaxis.
Famotidine	20 mg IV BID	Alternative. Reduce the dose in renal impairment, where accumulation causes confusion.

PREVENT THE OTHER ICU-ACQUIRED HARMS *bundles, because each is a complication the ICU itself creates*

Harm	What prevents it	The active ingredient
Ventilator-associated pneumonia	Head of bed 30 to 45 degrees, daily sedation interruption with a breathing trial, oral care, subglottic secretion drainage	Extubating sooner. The tube is the risk factor, so everything that shortens ventilation prevents VAP.
Central line infection	Full barrier precautions, chlorhexidine, avoid the femoral site, scrub the hub, daily review	Removing the line. Risk accrues per catheter-day.
CAUTI	Insert fewer catheters, closed drainage, nurse-driven removal protocols	Not having a catheter. Incontinence is not an indication.
Delirium and weakness	Light sedation, non-benzodiazepine agents, early mobility, sleep and day-night cues, family presence	Early mobility, which reduces both delirium duration and ICU-acquired weakness more than any drug.
Pressure injury	Regular repositioning, a risk assessment on admission, heel offloading, nutrition	Documented turns. Highest risk in the hemodynamically unstable, who are the hardest to turn and need it most.

PEARLS & PITFALLS

- Prescribe the restart time when you hold VTE prophylaxis, or it will not be restarted.
- Deprescribe ulcer prophylaxis at extubation, before it follows the patient home for years.
- Being in an ICU is not an indication for a PPI. Check for a real one.
- Run the checklist out loud. Its value is that it catches the thing nobody was thinking about.

References: Cook et al, REVISE, NEJM 2024 · CHEST guideline on prevention of VTE in hospitalized medical and critically ill patients · Vincent, "Give your patient a FAST HUG (at least) once a day", Crit Care Med 2005 · SCCM ICU Liberation and the ABCDEF bundle.

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





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.





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.

