STEM-S

NEET-SS

CRITICAL CARE MEDICINE

GROUP

LMR | NOTES

LAST MINUTE REVISION

VERSION 2.0

PRESENTED BY STEM-S

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SEPSIS

- Sepsis = infection + life-threatening organ dysfunction due to dysregulated host response.
- Operationally: acute SOFA ↑ ≥2 in the setting of suspected/confirmed infection. (Sepsis 3)
- Septic shock: sepsis + vasopressor requirement to maintain MAP ≥65 mmHg + lactate >2 mmol/L, despite adequate volume resuscitation.
- "Severe sepsis" used in Sepsis 2 definition, now obsolete terminology in sepsis 3.

SOFA 2 Scoring System

Organ system	0 points	1 point	2 points	3 points	4 points
Brain	GCS 15 (or thumbs-up, fist, peace sign)	GCS 13–14 OR localizing to pain OR drugs to treat delirium	GCS 9–12 OR withdrawal to pain	GCS 6–8 OR flexion to pain	GCS 3–5 OR extension to pain / no response to pain / generalized myoclonus
Respiratory	PaO_2/FiO_2 >300 mmHg (>40 kPa)	≤300 mmHg (≤40 kPa)	≤225 mmHg (≤30 kPa)	≤150 mmHg (≤20 kPa) + advanced ventilatory support	≤75 mmHg (≤10 kPa) + advanced ventilatory support OR ECMO
Cardiovascular	MAP ≥70 mmHg, no vasopressor/ inotrope	MAP <70 mmHg, no vasopressor/ inotrope	Low-dose vasopressor: NE + Epi ≤0.2 µg/kg/min, OR any dose of another vasopressor/ inotrope	Medium-dose NE + Epi >0.2–≤0.4 µg/kg/min, OR low-dose NE/Epi ≤0.2 + another vasopressor/ inotrope	High-dose NE + Epi >0.4 µg/kg/min, OR medium-dose >0.2–≤0.4 + another vasopressor/inotrope, OR mechanical circulatory support
Liver	Bilirubin ≤1.2 mg/dL	≤3.0 mg/dL	≤6.0 mg/dL	≤12.0 mg/dL	>12 mg/dL
Kidney	Creatinine ≤1.2 mg/dL	Creatinine ≤2.0 mg/dL OR urine output <0.5 mL/kg/h for 6–12 h	Creatinine ≤3.5 mg/dL OR urine output <0.5 mL/kg/h ≥12 h	Creatinine >3.5 mg/dL OR urine output <0.3 mL/kg/h ≥24 h OR anuria ≥12 h	Receiving RRT OR fulfills criteria for RRT
Hemostasis	Platelets >150 $\times 10^3/\mu L$	≤150 $\times 10^3/\mu L$	≤100 $\times 10^3/\mu L$	≤80 $\times 10^3/\mu L$	≤50 $\times 10^3/\mu L$

SURVIVING SEPSIS CAMPAIGN 2026

6 domains | major recommendations in a nutshell

1 SCREENING & EARLY MANAGEMENT

- Use **NEWS/NEWS2, MEWS or SIRS** rather than qSOFA alone.
- Obtain **blood cultures** as soon as possible, ideally before antibiotics.
- Give crystalloids **≥30 mL/kg within 3 h** for sepsis-induced hypoperfusion/septic shock (individualize and reassess).
- Start **early vasopressors** if hypotension persists; can start peripherally and in parallel with fluids in unstable shock.
- Target **MAP ~65 mmHg** (60–65 mmHg in age ≥65 yr).
- ICU admission within **6 h** if required.

2 INFECTION

≤1 h

- Give **antibiotics immediately, ideally within 1 h** in septic shock or probable/definite sepsis.
- If possible sepsis without shock: rapid investigation; if concern persists, **antibiotics within 3 h**.
- Rapidly identify and perform source control when indicated, **ideally within 6 h**.
- Use **prolonged-infusion β-lactams** after a loading dose.
- **De-escalate** antibiotics and favor **shorter duration** when appropriate.

3 HEMODYNAMICS

MAP

- Crystalloids first-line; **balanced crystalloids** preferred over 0.9% saline.
- Use **dynamic measures** for fluid responsiveness (e.g., PLR, SVV, PPV).
- Target **MAP ~65 mmHg** (60–65 mmHg in age ≥65 yr).
- **Norepinephrine** first-line vasopressor. Add **vasopressin** if requirements escalate; add **epinephrine** if MAP remains inadequate.
- Consider **active fluid removal** after acute resuscitation when appropriate.

4 RESPIRATORY SUPPORT

- In acute hypoxemic respiratory failure, **HFNC preferred** over conventional oxygen and initially over NIV.
- Consider **awake proning** (if not intubated).
- **ARDS: VT 6 mL/kg IBW**; plateau pressure **≤30 cmH_2O**.
- Moderate–severe ARDS: **higher PEEP** and **prone >12 h/day**.
- Use **intermittent neuromuscular blocker boluses** (rather than continuous infusion).
- Consider **VV-ECMO** for severe refractory ARDS at experienced centers.

5 ADJUNCTIVE & ADDITIONAL THERAPIES

- Use **IV corticosteroids** in septic shock.
- **Do not use** IV vitamin C, IVIG or probiotics.
- Use a **restrictive transfusion** strategy.
- Start **enteral nutrition within 72 h**.
- Initiate **insulin** at glucose **≥180 mg/dL** (10 mmol/L).
- **LMWH preferred** over UFH for VTE prophylaxis.
- **Do not start RRT** for AKI without a definitive indication.
- Consider **bicarbonate only if pH ≤7.2** with AKI stage 2/3.

6 GOALS, TRANSITIONS & LONG-TERM OUTCOMES

- Discuss prognosis and goals of care early, ideally **within 72 h**.
- Use a structured **ICU-to-ward transition** and handover.
- Perform **medication reconciliation** at transitions.
- Provide **sepsis education** before discharge.
- Assess **physical, cognitive and emotional** sequelae.
- Support **mental health** and **rehabilitation** after discharge.

SHOCK

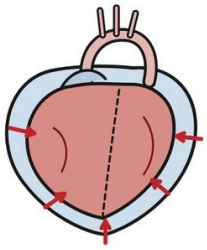
SHOCK

↓ CIRCULATING VOLUME	PUMP FAILURE	VASCULAR DILATATION		OBSTRUCTION
HYPOVOLEMIC SHOCK	**CARDIOGENIC SHOCK**	**SEPTIC SHOCK**	**ANAPHYLACTIC SHOCK**	**OBSTRUCTIVE SHOCK**
Bleeding / Dehydration *Bleeding* *Dehydration*	**Myocardial infarction + Pulmonary edema** *MI* *Pulmonary edema*	**Fever + warm skin + vasodilation**	**Urticaria / angioedema ± airway involvement**	**Hypotension + raised JVP** **Think obstructive** JVP ↑
↓ Preload ↓ Cardiac output	↓ Cardiac output ↑ Systemic vascular resistance (vasoconstriction)	↓ Systemic vascular resistance	↓ Systemic vascular resistance + ↓ circulating volume (capillary leak)	**Pulmonary embolism (PE)** **Cardiac tamponade** **Tension pneumo-thorax**
Clinical features • ↓ JVP • Cold, clammy skin • Tachycardia • Narrow pulse pressure	**Clinical features** • ↑ JVP • Crackles (pulmonary edema) • Cold, clammy skin • Narrow pulse pressure	**Clinical features** • Fever • Warm, flushed skin • Bounding pulse • Wide pulse pressure • Often high cardiac output (early phase)	**Clinical features** • Urticaria / angioedema • Bronchospasm / airway edema • Hypotension • Warm skin (early) • May progress to cold skin (late)	

CARDIAC TAMPONADE

Pericardial pressure → impaired diastolic filling → ↓ cardiac output → obstructive shock

PATHOPHYSIOLOGY



- Distended neck veins (JVP ↑)
- Non-compliant pericardium
- Compression of cardiac chambers (especially RA and RV) → ↓ diastolic filling → ↓ stroke volume → ↓ cardiac output
- **Pericardial effusion increases intrapericardial pressure**

CLINICAL FEATURES

Symptoms

- Dyspnea (most common)
- Chest pain (pleuritic, retrosternal)
- Palpitations
- Weakness, fatigue
- Early satiety, abdominal discomfort

Signs

- **Beck triad** (may be absent): hypotension; muffled heart sounds; distended neck veins (JVP ↑)
- Tachycardia
- **Pulsus paradoxus** (>10 mmHg fall in SBP on inspiration)
- Kussmaul's sign (↑ JVP on inspiration)
- Signs of obstructive shock (cold extremities, altered sensorium)

DIAGNOSIS

1. Clinical suspicion

Hypotension + raised JVP ± muffled heart sounds ± pulsus paradoxus

2. ECG (low voltage, electrical alternans)

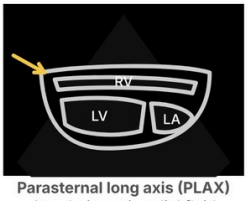
Electrical alternans (beat-to-beat variation in QRS amplitude)

- Low voltage QRS complexes
- Sinus tachycardia common

3. Chest X-ray

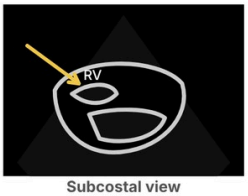
- Enlarged, globular (water-bottle) cardiac silhouette (especially in chronic effusion)
- May be normal in acute tamponade

ULTRASOUND (POCUS) FINDINGS

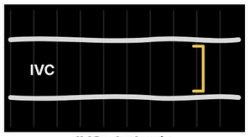


Parasternal long axis (PLAX)
Anechoic pericardial fluid surrounding heart

Apical 4 chamber
Right atrial diastolic collapse (>1/3 of RA free wall)



Subcostal view
Right ventricular diastolic collapse



IVC plethoric
Dilated IVC with < 50% inspiratory collapse

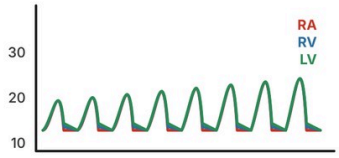
HEMODYNAMIC FEATURES

(classic but not always present)

- ↑ Right atrial pressure (↑ JVP)
- ↓ RV and LV diastolic filling
- = Equalization of diastolic pressures (RA ≈ RV diastolic ≈ LV diastolic)
- ↓ Stroke volume and cardiac output
- ↓ Pulsus paradoxus (>10 mmHg)

CARDIAC CATHETERIZATION (rarely needed)

Equalization of diastolic pressures



MANAGEMENT IN ICU

1 Initial stabilisation

- Airway, oxygen, monitoring
- Avoid positive pressure ventilation if possible (may worsen tamponade)
- IV fluids (small bolus) to maintain preload
- Vasopressors (norepinephrine) if hypotension persists

2 Definitive treatment: Pericardiocentesis (emergent if unstable)

- Ultrasound-guided pericardiocentesis (**subxiphoid approach preferred**)
- Send fluid for analysis (cell count, culture, cytology, biochemistry)
- Continue drainage if catheter placed

3 Treat underlying cause

- Post-MI (free wall rupture), malignancy, TB, uremia, viral pericarditis, aortic dissection, etc.
- Surgical pericardial window if recurrent or loculated effusion

SPECIAL SITUATIONS IN STATUS EPILEPTICUS

TOXIN-INDUCED STATUS (Benzodiazepine-resistant SE)

Benzodiazepine-resistant seizures with history of toxin exposure

↓

Think of
- Isoniazid (INH)
- Gyromitra mushroom (false morel)
- Rocket fuel / Hydrazine

↓

Treatment
Pyridoxine (vitamin B6)

Why?
Hydrazines inhibit GABA synthesis
→ ↓ GABA
→ ↑ glutamatergic excitation
→ severe seizures

When to consider empiric pyridoxine
- Suspected toxin-induced seizure
- History of TB / residence in TB-endemic area
- Unexplained SE

WHY PHENYTOIN CAN FAIL IN TOXIC SE

Toxin-induced seizures

↓

Benzodiazepines first

↓

Certain poisonings
(phenytoin may be ineffective / harmful)
- Cocaine
- Tricyclic antidepressants (TCA)
- Theophylline

↓

Avoid phenytoin
- May be ineffective
- Can worsen toxicity (e.g. TCA, cocaine)

TCA overdose with seizures
- Wide QRS / hypotension

↓

Sodium bicarbonate

NON-CONVULSIVE STATUS EPILEPTICUS (NCSE)

Suspect when

Persistent altered consciousness

±
- Fluctuating mental status
- Subtle facial/ocular movements
- Unexplained coma after convulsive SE
- Unexplained neurological deterioration

↓

Confirm with EEG

Continuous EEG is preferred when suspicion persists.

Suggested duration of monitoring
- **24 hours** in non-comatose patients
- **48 hours** in comatose patients

STATUS EPILEPTICUS IN SAH / ACUTE BRAIN INJURY

SAH or acute brain injury with altered consciousness

↓

Think of NCSE

↓

Why important?
- Seizure activity → ↑ cerebral metabolic demand
- ↑ risk of cerebral ischemia

↓

Management
- Early EEG (continuous if needed)
- Treat seizures as per standard protocol
- Optimize cerebral perfusion
- Treat underlying cause

Airway: consider intubation when
- GCS < 8
- Inability to protect airway
- Anticipated neurological deterioration

DRUG TABLE

Treatment	Role	Key exam trap
Lorazepam	First-line benzodiazepine	Adequate early dosing is crucial
Midazolam	First-line, especially IM if no IV	Useful prehospital
Diazepam	Benzodiazepine option	Rapid redistribution
Levetiracetam	Second-line	Few interactions
Fosphenytoin	Second-line	Cardiac/BP monitoring
Valproate	Second-line	Avoid/caution in significant hepatic disease
Propofol	Refractory SE anesthetic	Hypotension; PRIS with prolonged/high-dose therapy
Midazolam infusion	Refractory SE	Accumulation/hypotension
Phenobarbital/barbiturate	Refractory/super-refractory	Respiratory/cardiovascular depression
Pyridoxine	Hydrazine/isoniazid SE	**Specific toxicologic antidotal therapy**

COMMON LOCATIONS — PATTERN RECOGNITION

Location	Classic association
Putamen/basal ganglia	Chronic hypertension
Thalamus	Hypertension
Pons	Hypertension; often severe
Cerebellum	Hypertension; rapid deterioration/hydrocephalus
Lobar	CAA, anticoagulation, tumor/AVM etc.

IMPORTANT SCORING SYSTEMS IN INTRACEREBRAL HEMORRHAGE (ICH)

ICH SCORE

Predicts 30-day mortality

Component	Findings	Points
GCS score	13–15	0
	5–12	1
	3–4	2
Hematoma volume (on CT)	< 30 mL	0
	≥ 30 mL	1
Intraventricular hemorrhage (IVH)	Absent	0
	Present	1
Infratentorial origin	No	0
	Yes	1
Age	< 80 years	0
	≥ 80 years	1

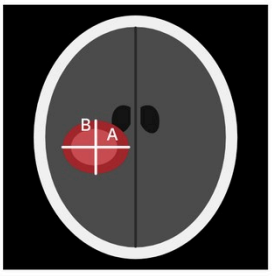
Total score: 0 – 6

30-day mortality (%)

0	1	2	3	4	5	6
0	13	26	72	97	100	100

ICH VOLUME CALCULATION

ABC/2 method (mL)



Volume (mL) = **A × B × C / 2**

A = largest diameter (cm)
B = diameter perpendicular to A (cm)
C = number of slices × slice thickness (cm)

Hematoma volume ≥ 30 mL = 1 point in ICH score

FUNC SCORE

Predicts functional outcome at 90 days (favorable: mRS 0–3)

Component	Findings	Points
Age	< 60 years	0
	60–79 years	1
	≥ 80 years	2
ICH volume	< 30 mL	0
	30–60 mL	1
	> 60 mL	2
Infratentorial origin	No	0
	Yes	1
Pre-ICH cognitive impairment/dementia	No	0
	Yes	1
GCS score	13–15	0
	5–12	1
	3–4	2

Total score: 0 – 8

Probability of favorable outcome at 90 days (%)

0	1	2	3	4	5	6	7	8
88	75	62	46	29	16	7	3	1

ICH-GS (GLASGOW COMA SCALE) SCORE

Predicts 30-day mortality

Component	Findings	Points
Age	< 80 years	0
	≥ 80 years	1
ICH volume	< 30 mL	0
	≥ 30 mL	1
GCS score	13–15	0
	5–12	1
	3–4	2
IVH	Absent	0
	Present	1

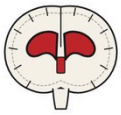
Total score: 0 – 5

30-day mortality (%)

0	1	2	3	4	5
1	6	17	45	73	90

GRAEB SCORE (INTRAVENTRICULAR HEMORRHAGE)

Quantifies ventricular blood volume



Ventricle	Points (each side)
Lateral ventricle (anterior horn)	0 – 4
Lateral ventricle (posterior horn)	0 – 4
Third ventricle	0 – 4
Fourth ventricle	0 – 4

Total score: 0 – 12

Semiquantitative grading for each ventricle

0 = no blood
1 = < 25% filled
2 = 25 – 50%
3 = 50 – 75%
4 = > 75% filled

SPOT SIGN (CTA)

Predicts hematoma expansion

Contrast extravasation within the hematoma on CTA

↓

Increased risk of hematoma expansion and poor outcome

CAUSES OF COMA

Category	Important causes	Clinical clue
A — Alcohol / acidosis	Toxic alcohols, severe metabolic acidosis	Osmolar or anion gap, acid–base disturbance
E — Epilepsy / electrolytes	Nonconvulsive status, Na/Ca abnormalities	Subtle twitching, gaze deviation; EEG needed
I — Insulin	Hypoglycemia, severe hyperglycemia	Bedside glucose
O — Opiates / oxygen	Opioids, hypoxia, hypercapnia	Respiratory depression, abnormal blood gas
U — Uremia	Uremic encephalopathy	Renal failure, metabolic abnormalities
T — Trauma / temperature	Intracranial hemorrhage, TBI, hypothermia, heat illness	Trauma history, abnormal temperature
I — Infection	Meningitis, encephalitis, sepsis-associated encephalopathy	Fever, meningism, systemic infection
P — Poisoning / psychiatric mimics	Sedatives, toxic drugs; psychogenic unresponsiveness as a diagnosis of exclusion	Toxidrome, medication history
S — Stroke / seizure / shock	Brainstem stroke, ICH, SAH, postictal state, shock	Focal deficits, pupil abnormalities, hypotension

Other key causes: hepatic encephalopathy, adrenal crisis, thiamine deficiency, cerebral venous thrombosis and posterior reversible encephalopathy syndrome (PRES).

13. IMPORTANT CAUSE PATTERNS

Pattern	Diagnosis to consider
Coma + **hypoglycemia**	Hypoglycemic encephalopathy
Coma + **pinpoint pupils**	Opioid / pontine lesion
Coma + **anisocoria**	Mass effect/uncal herniation
Coma + **fever + meningism**	CNS infection
Coma + **asterixis/flapping**	Metabolic encephalopathy
Coma + **seizure activity**	SE/NCSE
Coma + **trauma**	TBI/ICH
Coma + **SAH**	Hydrocephalus/ICP/early brain injury
Coma + **ICH**	Hematoma expansion/IVH/ICP
Coma + normal CT + persistent unexplained state	Metabolic/toxic/NCSE → MRI/EEG

RAISED INTRACRANIAL PRESSURE (ICP) / IMPENDING HERNIATION

Emergency treatment

Immediate escalation

Elevate head of bed ~30°
- Keep neck midline
- Optimize venous drainage

Prevent hypoxemia and hypotension
(SpO_2 ≥94%, MAP ≥65 mmHg)
- Maintain adequate oxygenation
- Maintain adequate blood pressure
- Secure the airway if necessary

Treat underlying lesion urgently
- Involve neurosurgery early

Hypertonic saline or mannitol
OR
- Use in suspected acute herniation
- Choose based on clinical context

$PaCO_2$ 30–35 mmHg

Brief controlled hyperventilation
- Temporary rescue bridge for impending herniation
- Not for routine prolonged use

IMPORTANT DRUG AND TREATMENT CONSIDERATIONS IN COMA

Hypoglycemia
- Give IV dextrose immediately
- Give thiamine promptly when deficiency is plausible
- Do not delay glucose

Suspected opioid toxicity
- Provide ventilatory support first
- Give titrated naloxone

Suspected meningitis / encephalitis
- Do not delay appropriate empiric antimicrobials (ceftriaxone) or acyclovir for imaging or lumbar puncture when treatment is urgent

Nonconvulsive status epilepticus
- Unexplained persistent coma after a seizure requires urgent EEG assessment

Traumatic coma

Avoid secondary brain injury
(MAP ≥65 mmHg)
- Prevent hypoxia
- Prevent hypotension

Brain death
✓ Prerequisites
✓ Exclude confounders
✓ Neurological examination
✓ Required testing

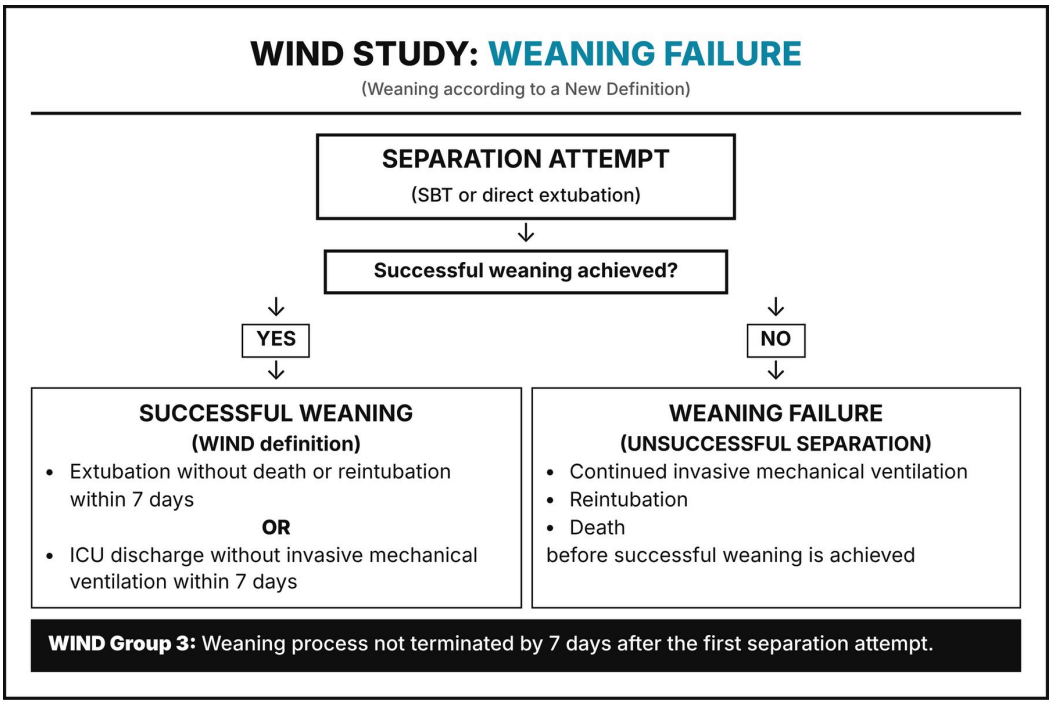
- **Coma alone is insufficient**
- Ensure formal prerequisites, exclusion of confounders and the required neurological examination/testing are satisfied

RAPID DRUG TABLE

Intervention	Role	Exam trap
Norepinephrine	Neurogenic shock / MAP augmentation	Often preferred in cervical/high thoracic SCI
Phenylephrine	Vasopressor	Pure α → does not treat bradycardia
Atropine	Symptomatic bradycardia	Does not correct vasodilation
LMWH	VTE prophylaxis	IVC filter ≠ routine prophylaxis
Methylprednisolone	Not routinely recommended	Major exam trap
Early decompression/stabilization	Mechanical compression/instability	Timing individualized

NEUROMUSCULAR DISORDERS IN THE CRITICALLY ILL

- ICU-acquired weakness (ICUAW) = new diffuse weakness developing during critical illness.
- Major entities: CIP + CIM + critical illness neuromyopathy.
- Classic setting: sepsis/MODS + prolonged ICU stay + mechanical ventilation + immobilization + corticosteroids/neuromuscular blockers.
- Major clinical consequence → prolonged ventilator dependence + difficult weaning + prolonged rehabilitation.
- Always distinguish from GBS, myasthenia, spinal cord disease, residual sedation/NMB and electrolyte disorders.



• RAPID TREATMENT TABLE

Intervention	Main mechanism	Primary use	Exam trap
FiO_2	↑ inspired O_2	Hypoxemia	Doesn't recruit alveoli
PEEP	Prevents alveolar collapse	Hypoxemic failure/ARDS	Excess → hypotension/RV strain
↓ VT	↓ alveolar strain	ARDS	Use PBW, not actual weight
↑ RR	↑ minute ventilation	Hypercapnia	Can worsen auto-PEEP
Proning	Better V/Q + more homogeneous stress	Severe ARDS	>12 h/day
NIV	Positive-pressure support without ETT	Selected respiratory failure	Not for inability to protect airway
SBT	Tests spontaneous breathing	Liberation	RSBI not mandatory

WHEN TO INITIATE RRT: THE AEIOU ALGORITHM

Critically ill patient with AKI

↓

Assess complications, trajectory and reversibility

↓

A	E	I	O	U
Acidosis	**Electrolytes**	**Intoxications**	**Overload**	**Uremic complications**
Severe metabolic acidosis refractory to appropriate treatment.	Refractory/ life-threatening hyperkalemia; ECG changes.	Selected dialyzable toxins, e.g. lithium, methanol, ethylene glycol, salicylates when indicated.	Refractory fluid overload, especially pulmonary edema.	Encephalopathy, pericarditis, bleeding attributable to uremia or other serious symptomatic uremia.

Also consider: persistent oliguria/anuria, rapidly worsening biochemical abnormalities, inability to meet fluid/nutritional needs safely, and selected dialyzable metabolic disorders.

⚠ **Exam trap:** There is **no universal creatinine, BUN or potassium number** that alone determines RRT initiation. Interpret the result **alongside ECG, symptoms, trajectory, response to medical treatment and the patient's overall condition.**

5. MECHANICAL COMPLICATIONS

Complication	Classic clue	Key diagnostic test
Papillary muscle rupture → acute MR	Acute pulmonary edema + shock + new murmur	Echo
Ventricular septal rupture	Acute pulmonary edema + loud pansystolic murmur	Echo + L→R shunt
LV free-wall rupture	Shock/tamponade picture	Echo → pericardial effusion

VSD

AMI → acute pulmonary edema + loud pansystolic murmur

↓

urgent echo

↓

RV overload + L→R shunt

↓

Ventricular septal rupture

VSD rupture is rare, often developing within 24 h after infarction, and requiring urgent surgical evaluation.

6. DIFFERENTIAL — AMI SHOCK

Feature	LV pump failure	RV infarction	VSD	Acute MR	Tamponade
Pulmonary edema	+++	Usually absent	+++	+++	Variable
Murmur	—	—	Loud pansystolic	New systolic	—
Echo	↓ LV function	RV dysfunction	L→R shunt	MR	Pericardial effusion
JVP	↑	↑↑	↑	↑	↑
Key trap	Don't miss mechanical cause	Fluids may help	Post-MI acute deterioration	Papillary muscle rupture	Free-wall rupture

Echocardiography is key for identifying pump failure and excluding tamponade, massive PE and valvular regurgitation.

CRITICAL CARE MEDICINE

Feature	Type 1 – Hypoxemic	Type 2 – Hypercapnic	Type 3 – Perioperative	Type 4 – Shock-associated
Primary defect	Oxygenation failure	Ventilation failure	Atelectasis → V/Q mismatch	Respiratory muscle hypoperfusion/fatigue
PaO_2	↓↓↓	↓ / variable	↓	↓ / variable
$PaCO_2$	Normal/↓ initially	↑	Usually normal/ ↓ initially	Variable; may ↑
pH	Usually normal/alkalotic initially	↓ in acute	Usually normal initially	Metabolic acidosis may coexist
A–a gradient	↑	Normal in pure hypoventilation	↑	Usually ↑
Typical causes	Pneumonia, ARDS, pulmonary edema, PE	COPD, CNS depression, neuromuscular disease, severe asthma	Postoperative atelectasis, anesthesia, pain/splinting	Septic/cardiogenic shock, severe systemic illness
Main problem	O_2 can't get into blood	CO_2 can't get out	Reduced lung volume/recruitment	Demand > respiratory muscle perfusion
Typical clinical clue	Severe hypoxemia	Hypercapnia + acidemia	Post-op hypoxemia	Shock + tachypnea/work of breathing
Major treatment principle	O_2 + treat lung pathology	Ventilatory support + treat cause	Recruitment, analgesia, mobilization, O_2	Treat shock + respiratory support
NIV relevance	Selected patients	Very important	Selected	Selected; failure threshold low
Intubation trigger	Refractory hypoxemia	Severe acidosis/exhaustion/altered sensorium	Progressive failure	Persistent shock + respiratory failure

Severe hypoxemia with poor improvement despite high FiO_2 → think SHUNT.

Alveolar ventilation: $VA=(\text{Tidal Volume}-\text{Dead Space})\times RR$

Exam pearl: Increasing RR does not necessarily correct hypercapnia if dead-space ventilation is very high.

4. ARDS vs CARDIOGENIC PULMONARY EDEMA

Feature	ARDS	Cardiogenic pulmonary
Primary mechanism	↑ permeability	↑ hydrostatic pressure
LV dysfunction	Not required	Often present
PCWP	Usually ≤18 mmHg historically	Usually >18 mmHg
Edema fluid	Protein-rich	Relatively protein-poor
CXR	Bilateral opacities	Bilateral edema
Kerley lines	Less typical	May occur
Cardiomegaly	Usually absent	Often present
Response to diuresis	Variable	Usually improves
Hypoxemia	Often severe	Variable

DRUG / TREATMENT TABLE

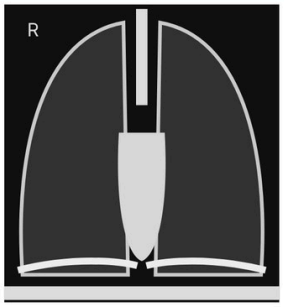
Intervention	Purpose	Exam trap
Low VT ventilation	↓ volutrauma	Use **PBW**
PEEP	Prevent alveolar collapse	Excessive PEEP can cause overdistension/hemodynamic compromise
Proning	Improve oxygenation + reduce VILI	Most important in severe ARDS
Conservative fluid strategy	Limit pulmonary edema	Don't compromise perfusion
Neuromuscular blockade	Facilitate controlled ventilation in selected patients	Not mandatory for every ARDS patient
Corticosteroids	May be used in selected ARDS protocols	Regimen/timing matters
VV-ECMO	Rescue severe refractory hypoxemia	Requires appropriate selection and expertise

CHEST X-RAY IN ACUTE EXACERBATIONS: ASTHMA vs COPD

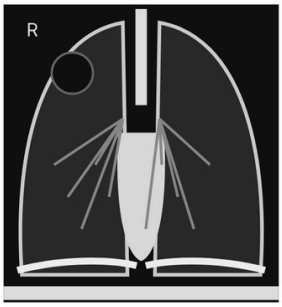
ASTHMA EXACERBATION

Hyperinflated lungs →

Flattened diaphragm →



COPD EXACERBATION



← Hyperinflated lungs with increased lucency

← Prominent bronchovascular markings

← Flattened diaphragm

Feature	Asthma exacerbation	COPD exacerbation
Lung volumes	Hyperinflated	Hyperinflated (often more marked, emphysematous)
Diaphragm	Flattened (due to air trapping)	Flattened and low (often more pronounced)
Lung fields	Increased lucency, usually clear	Increased lucency with prominent bronchovascular markings
Cardiac silhouette	Narrow/vertical (due to hyperinflation)	Narrow/vertical (often more marked)
Bronchovascular markings	Usually normal or mildly increased (peribronchial thickening)	Prominent, coarse markings; may have bullae
Focal opacities	Absent (unless complication e.g. pneumothorax)	Usually absent; look for pneumonia or pneumothorax as common triggers
Other findings	Air trapping; rule out pneumothorax	Emphysematous changes (bullae), diaphragmatic flattening, occasional atelectasis/infection

Treatment	Asthma	COPD	Exam pearl
SABA	⭐⭐⭐	⭐⭐⭐	Rapid bronchodilation
Ipratropium	Severe exacerbation	⭐⭐⭐	Particularly important in acute COPD
Systemic steroids	⭐⭐⭐	⭐⭐⭐	Give early in significant exacerbations
ICS	Foundation of controller therapy	Selected patients	ICS monotherapy is not COPD's usual cornerstone
LABA	Use with ICS-containing regimen	⭐⭐⭐	Long-acting bronchodilation
LAMA	Add-on	Major COPD therapy	Tiotropium = classic LAMA
$MgSO_4$ IV	Severe acute asthma	Not routine	Bronchodilator adjunct
Antibiotics	Not routine	Selected exacerbations	Think bacterial features
NIV	Selected	Major role	Especially COPD + acute respiratory acidosis

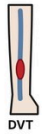
PULMONARY EMBOLISM

Pathophysiology, Diagnosis, Echocardiography and Management

KEY POINTS

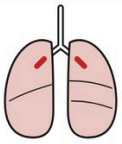
- PE = pulmonary arterial obstruction, usually thromboembolism from DVT.
- Acute PE increases pulmonary vascular resistance → acute RV pressure overload → RV failure → ↓ LV preload → shock.
- Echocardiography helps identify RV strain but is **NOT** a substitute for lung imaging.
- In patients with pulmonary hypertension, even a small PE can cause critical elevations in PA pressure and acute RV failure.

1. PATHOPHYSIOLOGY



DVT

— Embolization →



Pulmonary embolus in pulmonary artery

Pulmonary arterial obstruction → ↑ Pulmonary vascular resistance (PVR)

↓

Acute RV afterload ↑

↓

RV dilation + hypokinesis → Septal shift toward LV

↓

↓ LV preload
↓ Cardiac output

↓

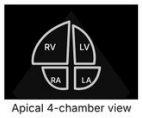
OBSTRUCTIVE SHOCK

Additional effects

- V/Q mismatch
- Impaired pulmonary perfusion
- Hypoxemia
- Tachypnea
- ↑ Sympathetic drive

2. ECHOCARDIOGRAPHIC FINDINGS OF ACUTE PE

A. RV DILATATION

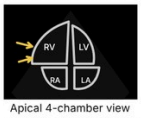


Apical 4-chamber view

RV size ≥ LV size

(Normally RV is 50–60% of LV)

B. McCONNELL SIGN



Apical 4-chamber view
Hypokinesis of RV free wall with
apical sparing (McConnell sign)

C. D-SIGN (SEPTAL FLATTENING)

Parasternal short-axis view

Interventricular septal flattening due to increased right-sided pressures (D sign)

NORMAL HEART (PSAX)

- RV is 50–60% of LV size
- Normal septal position

ACUTE PE (PSAX)

- Dilated RV (≥ LV size)
- Flattened septum (D sign)
- Small, underfilled LV

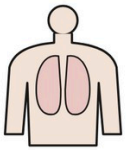
CHRONIC RV STRAIN (e.g. PH)

- Enlarged and thick-walled RV
- Septal changes (chronic)
- Consider chronic pressure overload

Echocardiography is NOT a substitute for lung imaging in the diagnosis of PE because no sonographic finding is sufficiently specific for the disorder.

3. CLINICAL PRESENTATION

- Sudden dyspnea
- Pleuritic chest pain
- Tachypnea
- Tachycardia
- Hypoxemia
- Syncope
- Hemoptysis (less common)
- Hypotension/shock (in high-risk PE)



4. DIAGNOSTIC ALGORITHM (STABLE PATIENT)

Clinical suspicion of PE

↓

Assess pre-test probability (clinical decision rule)

↓

Low / Intermediate probability

↓

D-dimer

↓

Negative PE excluded (in appropriate low-risk patients)

Positive CT pulmonary angiography (CTPA)

High probability

↓

CT pulmonary angiography (CTPA) (when feasible)

↓

If CTPA contraindicated or unavailable

↓

V/Q scan (in appropriate patient)
OR
Lower extremity compression ultrasound (if DVT suspected)

5. UNSTABLE PATIENT (SUSPECTED PE)

Suspected PE + Hypotension / Shock

↓

ABC, oxygen, monitor, supportive care

↓

Bedside echocardiography (FoCUS)

↓

Exclude:

- Tamponade
- Tension pneumothorax
- Major LV failure

↓

Findings suggestive of PE
(RV dilation/strain ± DVT)

↓

If CT is unsafe/unavailable and clinical probability is high

↓

Treat as hemodynamically significant PE + consider thrombolysis

6. MANAGEMENT

A. STABLE PE

- Anticoagulation (DOAC, LMWH, or VKA based on patient factors)
- Treat underlying cause
- Routine systemic thrombolysis is **NOT** indicated

B. HIGH-RISK / HEMODYNAMICALLY UNSTABLE PE

- Oxygen and supportive care
- Anticoagulation
- Systemic thrombolysis if appropriate (and no contraindication)
- If thrombolysis contraindicated or fails → catheter-directed therapy or surgical embolectomy (at specialized centers)

7. RV FAILURE MANAGEMENT (CRITICAL CARE PEARLS)

- **Correct hypoxemia aggressively** (hypoxemia → pulmonary vasoconstriction → ↑ PVR → ↑ RV afterload).
- **Avoid excessive fluids** (overdistension of RV can worsen LV filling and reduce cardiac output).
- **Vasopressors:** Norepinephrine (support systemic pressure).
- **Inotropes:** Dobutamine or milrinone (improve RV contractility/output).
- Use echocardiography to guide fluid resuscitation and therapy.

8. DIFFERENTIAL DIAGNOSIS (SUDDEN DYSPNEA + SHOCK)

Diagnosis	Key bedside clue (echo/clinical)
Massive PE	RV dilation/strain; LV underfilled
Tamponade	Pericardial effusion + tamponade physiology
Tension pneumothorax	Absent lung sliding
Acute MI	LV regional/global dysfunction
Aortic catastrophe	Aortic pathology ± pericardial effusion
Severe sepsis	Distributive physiology ± infection source

9. KEY TAKE-HOME POINTS

1. PE kills primarily through acute RV failure.
2. Classic echo signs: RV dilation, McConnell sign, D-sign.
3. Echo does **NOT** definitively diagnose PE.
4. Hemodynamic instability → consider thrombolysis.
5. Normotensive + RV strain = higher risk PE, but not an automatic indication for thrombolysis.
6. In patients with pulmonary hypertension, even a small PE can cause major hemodynamic consequences.

Clinical setting	Common bacterial causes	Empiric treatment
Community-acquired meningitis	S. pneumoniae; N. meningitidis	Ceftriaxone + Vancomycin
Elderly / immunocompromised	S. pneumoniae; Listeria monocytogenes; Gram-negative bacilli	Ampicillin + 3rd-generation cephalosporin ± Vancomycin
Nosocomial / post-neurosurgical meningitis	Gram-negative bacilli, including *Pseudomonas*; S. aureus; coagulase-negative staphylococci	Vancomycin + ceftazidime

Meningococcal meningitis
Clinical pattern: Fever + headache + neck stiffness ± Petechiae/purpura → meningococcemia

Progression:
Endotoxin → cytokines → endothelial injury → capillary leak → shock → DIC → multiorgan failure

Diagnostic clue
CSF Gram stain (sensitivity up to 90%)
→ Gram-negative diplococci
→ highly suggestive of N. meningitidis

Treatment
Adult 18–50 yr: Ceftriaxone 2 g IV q12h (first-line empiric therapy)
>50 yr: Add ampicillin → covers Listeria.
Once meningococcemia is confirmed, switch to penicillin G when appropriate.

Complications: Waterhouse–Friderichsen syndrome
→ endothelial injury
→ DIC + shock + multiorgan failure

Meningococcemia
→ purpura fulminans + adrenal hemorrhage + multiorgan failure

4. IMPORTANT DIFFERENTIALS

Feature	Encephalitis	Meningitis	Brain abscess	Autoimmune encephalitis
Altered sensorium	Prominent	Variable	Variable	Prominent
Seizures	Common	Possible	Common	Very common
Focal deficit	Possible	Less typical	Common	Possible
MRI	Parenchymal lesions	Often normal	Ring-enhancing	Limbic/other patterns
CSF	Inflammatory	Inflammatory	Variable	Often mild/normal
Key clue	Brain dysfunction	Meningeal syndrome	Mass lesion	Psychiatric + seizures

5. PATHOPHYSIOLOGY

Virus enters CNS
→ neuronal/glial infection
→ innate immune activation
→ cytokine-mediated inflammation
→ cerebral edema + neuronal injury
→ seizures + altered consciousness + focal deficits
→ severe cases → raised ICP/herniation

6. ETIOLOGY — RAPID PATTERN RECOGNITION

Infectious

- HSV-1 → classic sporadic encephalitis
- VZV → encephalitis ± vasculopathy
- Enteroviruses
- Arboviruses
- Rabies
- Japanese encephalitis
- HIV/CMV/HHV-6 in immunocompromised

HSV can cause life-threatening encephalitis, particularly in immunocompromised patients; VZV can also cause encephalitis and disseminated CNS disease.

Noninfectious

- Anti-NMDA receptor encephalitis
- LGI1
- CASPR2
- GABA-B
- Paraneoplastic encephalitis
- ADEM

PATTERNS OF DIARRHEA: IMPORTANT CAUSES AND CLUES

Pattern	Important causes	Clue
Watery, high-volume	Cholera, enterotoxigenic bacteria, secretory tumours	Large volume
Watery + recent antibiotics	*C. difficile*	Fever, leukocytosis
Watery + fasting improves	Osmotic diarrhea	Decreases with fasting
Watery persists during fasting	Secretory diarrhea	Continues overnight
Blood + fever	*Shigella, Campylobacter, Salmonella*, STEC, IBD	Inflammatory
Blood + severe abdominal pain	Ischemic colitis	Pain out of proportion / vascular risk
Greasy + weight loss	Pancreatic insufficiency, celiac, Giardia	Malabsorption
Nocturnal + chronic	IBD, microscopic colitis, secretory causes	Organic disease
Post-transplant + voluminous diarrhea	Acute GVHD	± rash / liver dysfunction
ICU + tube feeds	Enteral-feed related diarrhea	Temporal relation

Organism	Classic presentation	Treatment principle
Vibrio cholerae	Profuse “rice-water” diarrhea	Aggressive rehydration ± antibiotic in severe disease
ETEC	Traveler's watery diarrhea	Usually supportive; antibiotic in selected severe cases
Shigella	Fever + bloody diarrhea/tenesmus	Antibiotic if indicated
Campylobacter	Fever + inflammatory/bloody diarrhea	Usually supportive; antibiotics for severe/high-risk disease
Salmonella	Fever ± diarrhea	Supportive; antibiotics for invasive/high-risk disease
STEC/EHEC	Bloody diarrhea, often little fever	Avoid antibiotics + antimotility drugs; monitor HUS
C. difficile	Antibiotic-associated watery diarrhea	Specific CDI therapy
Giardia	Foul-smelling, greasy diarrhea	Antiprotozoal therapy
Entamoeba histolytica	Dysentery	Tissue-active + luminal therapy

7. Management

Intervention	Mechanism / role	Exam trap
Oral vancomycin	Luminal therapy for CDI	IV vancomycin alone does not adequately treat colonic CDI
Fidaxomicin	Inhibits bacterial RNA polymerase; reduces recurrence	Preferred for initial nonfulminant CDI when feasible; not established for fulminant disease
IV metronidazole	Adjunct in fulminant CDI	Not preferred as sole treatment for severe CDI
Bezlotoxumab	Monoclonal antibody against toxin B; recurrence prevention adjunct	Not an antibiotic; caution in congestive heart failure
FMT / microbiota-based therapy	Restores microbiome function	Consider for appropriately selected patients with recurrent CDI, not routine initial disease
Stop the inciting antibiotics		

Prevention and infection control

- Contact precautions: gloves and gown; isolate or cohort according to institutional policy.
- Hand hygiene: soap and water is particularly important when spore contamination is suspected or during outbreaks; alcohol alone does not kill spores.
- Environmental cleaning
- Antimicrobial stewardship: minimize unnecessary clindamycin, fluoroquinolones, cephalosporins and broad-spectrum antibiotics.

9. PYOMYOSITIS / INFECTIOUS MYOSITIS

Think: Fever + focal deep muscle pain/tenderness
→ CK may be elevated
→ MRI = best anatomical test
→ blood cultures
→ look for abscess.
Common pathogen: S. aureus (classic organism)
Treatment Antibiotics → targeted to organism
If intramuscular abscess→ drainage + antibiotics
If septic/necrotizing process→ surgical source control.

10. IMPORTANT DIFFERENTIALS

Mimic	Distinguishing clue
DVT	Swelling; no primary infectious skin changes
Stasis dermatitis	Usually bilateral, chronic hyperpigmentation/scaling
Contact dermatitis	Itch > pain; exposure pattern
Erysipelas	Sharply demarcated raised erythema
Cellulitis	Poorly demarcated erythema/warmth
NF	Pain out of proportion + rapid progression + toxicity
Pyomyositis	Deep muscle pain ± abscess
Ischemia	Severe pain + vascular findings, cool/pale limb
Compartment syndrome	Pain with passive stretch + tense compartment

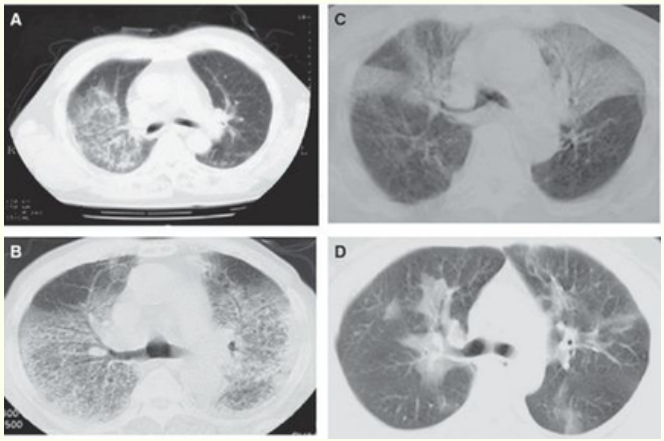
Invasive fungal infections

- IFI = fungal invasion of normally sterile tissue, blood vessels or organs; distinguish it from colonization.
- Major ICU syndromes: invasive candidiasis (candidemia/intra-abdominal candidiasis), invasive aspergillosis and mucormycosis.
- Risk factors: broad-spectrum antibiotics, central venous catheters, TPN, abdominal surgery, neutropenia, transplantation and immunosuppression.
- Blood cultures can be negative in invasive candidiasis, especially deep-seated disease.
- Early recognition + appropriate antifungal therapy + source control are key.

High-yield pearls and traps

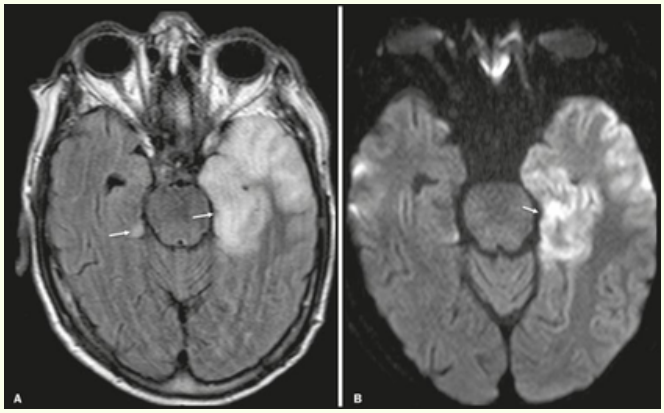
1. CD4 below 200/µL: classic association with PJP in HIV; non-HIV patients may develop PJP at different immune thresholds.
2. CD4 below 100/µL: risk of toxoplasma encephalitis in susceptible patients, especially with positive Toxoplasma IgG.
3. CD4 below 50/µL: classic risk for disseminated MAC in untreated advanced HIV.
4. CMV PCR positivity alone does not always establish CMV end-organ disease.
5. Candida isolated from sputum or BAL usually represents colonization rather than Candida pneumonia.
6. EBV after transplantation → consider post-transplant lymphoproliferative disorder (PTLD).
7. BK virus after kidney transplantation → consider viral nephropathy with unexplained graft dysfunction.
8. VZV may disseminate in immunocompromised hosts, causing pneumonitis, hepatitis or encephalitis.
9. Persistent fever despite antibiotics should prompt a systematic reassessment, not automatic addition of every antimicrobial class.
10. Immunosuppressive drugs may themselves cause cytopenias, liver abnormalities and drug fever that mimic infection.

4. Viral pneumonia and ARDS — pattern recognition



Diffuse bilateral ground-glass opacities

- Influenza, COVID-19, RSV and other viral pneumonias.
- Also consider PJP, pulmonary edema, diffuse alveolar hemorrhage and drug toxicity.



Temporal-lobe abnormalities: Classic HSV encephalitis pattern.

4. IMPORTANT ICU DIFFERENTIALS

Diagnosis	Pattern recognition
Sepsis-associated cholestasis	Sepsis + direct bilirubin ↑ ± relatively modest enzyme elevation
Ischemic hepatitis	Shock/hypoxemia + **massive AST/ALT**
Acute liver failure	Acute liver injury + **INR ↑ + encephalopathy**
Hemolysis	Indirect bilirubin ↑ + LDH ↑ + haptoglobin ↓ + reticulocytosis
Biliary obstruction	Direct bilirubin + ALP/GGT disproportionately ↑
SOS/VOD	Post-HCT + **jaundice + tender hepatomegaly + rapid weight gain**

Treatment
Mild/moderate → supportive care + careful fluid/sodium balance
Severe SOS with renal/pulmonary dysfunction → defibrotide

7. DRUG/CAUSE TABLE

Cause	Bilirubin pattern	Key clue
Gilbert	Indirect	Mild, intermittent; otherwise well
Crigler–Najjar	Indirect	Severe inherited conjugation defect
Hemolysis	Indirect	↑LDH, ↓haptoglobin
Hepatitis	Mixed/direct	AST/ALT predominant
Sepsis cholestasis	Direct	Critically ill/septic
CBD obstruction	Direct	ALP/GGT ↑, ductal dilation
SOS	Direct	Post-HCT + weight gain/hepatomegaly
Cyclosporine/methotrexate/TPN	Hyperbilirubinemia	Post-HCT iatrogenic causes

NON-VARICEAL UPPER GI BLEEDING

Common causes

Cause	Clue	Endoscopic appearance (typical)
Peptic ulcer disease (gastric or duodenal)	Most common major cause	**Ulcer** with visible vessel or adherent clot
Gastritis/ duodenitis	NSAIDs, alcohol, critical illness	Erythematous, friable mucosa
Mallory-Weiss tear	Forceful retching → hematemesis	Linear mucosal tear at gastroesophageal junction
Esophagitis	Reflux/odynophagia	Erosions/ulcers in esophagus
Malignancy	Weight loss, chronic symptoms	Ulceroproliferative mass
Dieulafoy lesion	Massive bleeding with minimal mucosal lesion	Protruding artery with minimal surrounding mucosal change

PEPTIC ULCER BLEED — MANAGEMENT

1 Resuscitation

- ABC, oxygen as needed
- Two large bore IV access
- Crystalloids ± blood transfusion (restrictive strategy, Hb target ~7 g/dL in most patients)
- Correct coagulopathy, stop antithrombotics if possible
- High-dose IV PPI (e.g., pantoprazole 80 mg bolus followed by 8 mg/h infusion)

↓

2 Upper GI endoscopy

- Perform within 24 hours (earlier if hemodynamic instability or ongoing bleeding).

↓

3 Endoscopic hemostasis

Depending on lesion:

- **Injection therapy** (e.g., dilute epinephrine)
- **Thermal therapy** (heater probe, bipolar, APC)
- **Mechanical therapy** (clips)

Injection | Thermal | Hemoclip

⚠ Epinephrine injection alone is **NOT** adequate definitive therapy for ulcer bleeding; combine with another hemostatic modality.

↓

4 Rebleeding

- Repeat endoscopy

↓

5 Endoscopic failure

- Angiographic embolization

↓

6 Failure of endoscopic + angiographic therapy

- Emergency surgery

6. DRUG TABLE

Treatment	Role	Exam trap
Octreotide	↓ splanchnic blood flow → variceal bleeding	**Not definitive therapy**
Terlipressin	Vasoconstriction → ↓ portal pressure	Watch ischemic complications
PPI	Acid suppression → ulcer clot stabilization/healing	Central in non-variceal UGIB
Erythromycin IV	Promotes gastric emptying before endoscopy	Useful with large blood burden
Lactulose	HE treatment	Not a hemostatic drug
Antibiotics	Infection prophylaxis in cirrhotic variceal bleed	Do not omit in acute variceal hemorrhage

CAUSES — PATTERN RECOGNITION

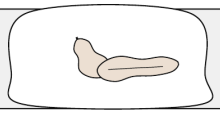
Cause	Typical clue
Diverticular bleeding	Sudden, painless, often brisk hematochezia
Angiodysplasia	Elderly; recurrent/intermittent bleeding
Ischemic colitis	Abdominal pain + bloody diarrhea
Colitis	Diarrhea + pain/fever
Colorectal cancer/polyps	Occult/chronic ± overt bleeding
Hemorrhoids	Bright-red blood, usually coating stool
Anal fissure	Severe pain during defecation + small-volume blood
IBD	Bloody diarrhea + systemic/inflammatory symptoms
Post-polypectomy	Delayed bleeding after colonoscopy
CMV colitis	Immunosuppressed/transplant patient

CRITICAL CARE MEDICINE

1

1 DIAGNOSTIC ALGORITHM

Acute severe epigastric pain ± vomiting



Suspect acute pancreatitis

Diagnosis requires ≥ 2 of 3:

1. Typical abdominal pain
2. Serum lipase or amylase ≥ 3 × ULN
3. Characteristic imaging findings (CT / ultrasound / MRI)

Determine etiology

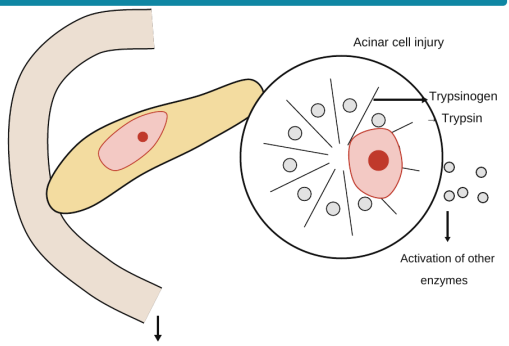
- Gallstone (US abdomen)
- Alcohol
- Hypertriglyceridemia
- Hypercalcemia
- Drug-induced
- ERCP / post-procedure
- Trauma
- Infection
- Autoimmune / other

Assess severity

- Organ failure
- SIRS
- BUN / hematocrit trend
- Renal function
- Oxygenation
- Imaging for complications (when indicated)

2 PATHOPHYSIOLOGY

Pancreatic acinar injury
↓
Premature activation of trypsinogen → trypsin
↓
Activation of other pancreatic enzymes
↓
Autodigestion + pancreatic inflammation
↓
Cytokine release
↓
Systemic inflammatory response
↓
Capillary leak + third spacing
↓
Hypovolemia → shock + AKI



Severe inflammation

- Microvascular injury
- Pancreatic / peripancreatic necrosis
- Sterile or infected necrosis

3 SEVERITY — REVISED ATLANTA CLASSIFICATION

Category	Key feature
Mild	No organ failure, no local or systemic complications.
Moderately severe	Transient organ failure (< 48 h) and/or local complications.
Severe	Persistent organ failure > 48 h.

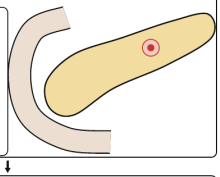
Management of Acute Pancreatitis

1 DIAGNOSIS

Acute severe epigastric pain ± vomiting

Diagnosis (≥ 2 of 3)

1. Typical abdominal pain
2. Serum amylase / lipase ≥ 3 × ULN
3. Characteristic imaging (CECT / USG / MRI)



Determine etiology

- Gallstone (USG)
- Alcohol
- Drugs
- Hyper-triglyceridemia
- Hyper-calcemia
- Others (autoimmune, post-ERCP, etc.)

2 SEVERITY ASSESSMENT

Assess severity (within 24–48 hours)

Clinical criteria (e.g. SIRS, organ failure)

- Shock
- Respiratory failure
- Renal failure
- Persistent organ failure (> 48 h)

Scoring systems

- BISAP
- Ranson score
- APACHE II

Morphological severity (imaging at 48–72 h)

- CECT severity index
- Pancreatic and peripancreatic necrosis

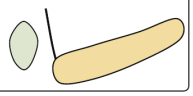
3 ETIOLOGY-SPECIFIC MANAGEMENT

Gallstone pancreatitis

Cholangitis or persistent biliary obstruction?

- YES → **Urgent ERCP**
- NO → **No routine urgent ERCP**

Mild gallstone pancreatitis → Cholecystectomy during same admission after clinical stabilization



Other etiologies

- Alcohol cessation
- Stop offending drug
- Treat hypertriglyceridemia (if severe)
- Treat autoimmune (consider steroids)

4 EARLY MANAGEMENT (FIRST 24 HOURS)

ABC + assess perfusion

Fluid resuscitation
Early, goal-directed IV crystalloid

Monitor

- MAP
- HR
- Urine output
- BUN/creatinine
- Hematocrit
- Lactate
- Respiratory status

Analgesia
Opioid / non-opioid as required

Oxygen
If hypoxemic

Nutrition
Early oral feeding when tolerated
If unable to eat → enteral nutrition (preferred over parenteral)

5 ANTIBIOTICS

Sterile pancreatitis

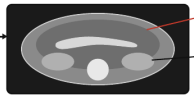
No prophylactic antibiotics

Suspected / confirmed infected necrosis

- Antibiotics with pancreatic penetration
- Multidisciplinary management
- Drainage / debridement when indicated

6 NECROTIZING PANCREATITIS

Severe pancreatitis (consider CECT / MRI when indicated)



- Pancreatic necrosis
- Peripancreatic necrosis

Sterile necrosis

- No infection
- Conservative / supportive management

Infected necrosis

- Clinical deterioration ± gas in collection / culture evidence
- Antibiotics
- Step-up approach

Percutaneous / endoscopic drainage
↓
If inadequate Minimally invasive necrosectomy
↓
Selected refractory cases Surgery

7 FOLLOW UP AND PREVENTION

- **Cholecystectomy** for gallstone pancreatitis (during same admission for mild disease)
- Alcohol abstinence (alcohol-related)
- Control hypertriglyceridemia
- Avoid offending drugs
- Treat underlying autoimmune cause
- Manage complications and prevent recurrence

HYPERMAGNESEMIA & MAGNESIUM SULFATE TOXICITY

SEVERE HYPERMAGNESEMIA — EMERGENCY ALGORITHM

HYPERMAGNESEMIA

↓

STOP ALL MAGNESIUM SOURCES

↓

ABC + ECG + continuous monitoring

↓

Symptoms or ECG abnormalities?

↓

YES:
IV CALCIUM GLUCONATE
(temporary reversal of cardiotoxicity and neuromuscular depression)

↓

Assess renal function

↓

Adequate renal function
Isotonic IV fluids
Consider loop diuretic if appropriate and maintain volume

Severe AKI/CKD, refractory toxicity or life-threatening manifestations
HEMODIALYSIS

↓

Serial serum Mg, ECG and clinical monitoring

MAGNESIUM SULFATE TOXICITY

Especially important during eclampsia treatment.

MONITOR DURING MAGNESIUM INFUSION

- Respiratory rate
- Deep tendon reflexes
- Urine output

WARNING SIGNS OF TOXICITY

- Absent patellar reflexes
- Respiratory depression
- Oliguria

IF SIGNIFICANT TOXICITY OCCURS

STOP $MgSO_4$
↓
IV calcium gluconate
↓
Supportive care ± dialysis

HYPOMAGNESEMIA vs HYPERMAGNESEMIA

Feature	Hypomagnesemia	Hypermagnesemia
Reflexes	Hyperreflexia	Hyporeflexia / areflexia
Neuromuscular manifestations	Tremor, tetany, seizures	Weakness, paralysis
ECG	QT prolongation	PR/QRS prolongation, AV block
Arrhythmia	Torsades de pointes	Bradyarrhythmia / heart block
Treatment	Magnesium replacement	Stop magnesium + IV calcium
Severe renal failure	Not typical	Major risk

High-yield exam pearls

1. Hypermagnesemia + renal failure + Mg-containing medication → classic scenario.
2. Hyporeflexia/areflexia + respiratory depression after $MgSO_4$ → Mg toxicity.
3. First step → STOP magnesium.
4. Symptomatic toxicity → IV calcium gluconate.
5. Severe toxicity with renal failure → hemodialysis.
6. Calcium reverses the effects of Mg but does not eliminate Mg.
7. Monitor DTRs, respiratory rate and urine output during therapeutic $MgSO_4$ infusion.

Hypermagnesemia is usually caused by magnesium administration in the setting of impaired renal excretion; severe toxicity produces hyporeflexia, muscle weakness, respiratory depression, hypotension and cardiac conduction abnormalities, and is managed by stopping magnesium, IV calcium as a physiological antagonist, enhancing renal elimination when appropriate, and hemodialysis for severe toxicity or renal failure.

7. Drugs and treatment traps

Intervention	Role	Exam trap
KCl	Preferred replacement in most cases	Never IV push
Magnesium sulfate	Correct associated Mg^{2+} deficiency	K^+ may remain low until Mg^{2+} is corrected
Spironolactone/eplerenone	Selected renal K^+ wasting/mineralocorticoid excess	Not a substitute for emergency K^+ replacement
Amiloride	Selected renal K^+ wasting states	Monitor for hyperkalemia, especially with renal impairment
Acetazolamide	Selected alkalosis-related scenarios	Can worsen potassium depletion
Insulin / β_2-agonists	Shift K^+ intracellularly	Can precipitate or worsen hypokalemia

8. High-yield INI-SS pearls

1. Refractory hypokalemia → check magnesium first.
2. Hypertension + hypokalemia + metabolic alkalosis → suspect mineralocorticoid excess.
3. Hypokalemic metabolic alkalosis with low urine chloride → vomiting/chloride depletion is likely.
4. Hypokalemia with metabolic acidosis → think diarrhea or renal tubular acidosis.
5. Prominent U waves are a classic ECG clue.
6. Correcting potassium does not necessarily correct the cause; ongoing GI or renal losses must be controlled.
7. In thyrotoxic periodic paralysis, profound hypokalemia may result mainly from an intracellular shift; aggressive replacement can cause rebound hyperkalemia.
8. Digoxin-treated patients with hypokalemia have increased susceptibility to digoxin toxicity.
9. During high-output diuresis, monitor potassium and magnesium closely; the uploaded critical-care text specifically highlights hypokalemia following brisk post-transplant diuresis and recommends cardiac monitoring when replacement requirements exceed 0.3 mEq/kg/hour.

"ECG → K^+ → Mg^{2+} → Urine K^+ → Acid–base/BP → Cause."
Hypokalemia is serum K^+ <3.5 mmol/L; management requires assessment of ECG, severity, renal function and Mg^{2+}, identification of renal/GI losses or intracellular shifts, and potassium replacement—preferably oral when mild, IV KCl with monitoring when severe or symptomatic, with simultaneous correction of hypomagnesemia.

High-yield table

Pattern	Diagnosis
Alkalosis + urine Cl^- <20	Vomiting/NG suction/volume depletion
Alkalosis + urine Cl^- >20 + hypertension	Mineralocorticoid excess
Alkalosis + urine Cl^- >20 + hypomagnesemia	Gitelman
Alkalosis + urine Cl^- >20 + hypercalciuria	Bartter
Alkalosis after rapid correction of chronic hypercapnia	Post-hypercapnic alkalosis
Diuretic + hypokalemia + alkalosis	Diuretic-induced alkalosis
Severe alkalosis + volume overload	Consider acetazolamide
Severe refractory alkalosis + renal failure	Consider RRT

Exam Pearls

1. Urine chloride is the key bedside discriminator.
2. Urine Cl^- <20 → chloride-responsive.
3. Urine Cl^- >20 → chloride-resistant or ongoing renal chloride loss.
4. Vomiting/NG suction → H^+ + Cl^- loss → metabolic alkalosis.
5. Hypokalemia perpetuates metabolic alkalosis → aggressively correct K^+.
6. Hypertension + hypokalemic metabolic alkalosis → think aldosterone/mineralocorticoid excess.
7. Gitelman → hypomagnesemia + hypocalciuria.
8. Bartter → hypercalciuria.
9. Post-hypercapnic alkalosis is common after rapid correction of chronic hypercapnia.
10. Acetazolamide promotes renal HCO_3^- loss and is useful when saline is undesirable.
11. Severe alkalemia can cause arrhythmias, neurological dysfunction and reduced ionized calcium.
12. Never interpret an elevated $PaCO_2$ in metabolic alkalosis as compensation without calculating the expected value.

Metabolic alkalosis is a primary elevation of serum bicarbonate; urine chloride classifies it into chloride-responsive (<20 mmol/L, usually volume/chloride depletion) and chloride-resistant (>20 mmol/L, ongoing renal losses/mineralocorticoid excess), with treatment directed at chloride and potassium repletion, correction of the underlying cause, and acetazolamide or renal replacement therapy in selected refractory/volume-overloaded patients.

Thyroid disorders

Disorder	TSH	FT4	Key feature
Primary hypothyroidism	↑	↓	Hashimoto
Subclinical hypothyroidism	↑	Normal	Biochemical only
Primary hyperthyroidism	↓	↑	Graves/toxic nodules
Subclinical hyperthyroidism	↓	Normal	Biochemical only
Central hypothyroidism	↓/normal	↓	Pituitary/hypothalamic
NTIS	↓/normal	Normal/↓	Critical illness
Thyroid storm	↓	↑	Fever + CNS/CV/GI
Myxedema coma	↑ usually	↓	Hypothermia + CNS depression

Thyroid emergencies — side-by-side

	Myxedema coma	Thyroid storm
Temperature	Low	High
HR	Bradycardia	Tachycardia
Mental status	Depressed	Agitated/delirious
BP	Hypotension	Hypertension → shock
GI	Ileus	Vomiting/diarrhea
Na^+	Low	Variable
Glucose	Low	Variable
Treatment	T4 + hydrocortisone	β-blocker + thionamide + iodine + steroid