Shock, Electrolytes, and Fluid

Clinical Principles and Surgical Judgment

Based on Sabiston Textbook of Surgery, 22nd edition
Chapter 33, beginning on printed page 221

Source chapter authors
Paige Farley, Sawyer G. Smith, Jason L. Sperry, and Martin Allan Schreiber

Foolad Eghbali, MD
Associate Professor of Surgery
Iran University of Medical Sciences
Tehran, Iran

Teaching chapter and surgical judgment assessment

An integrated account of shock, perfusion, hemorrhage control, fluid prescribing, electrolyte disorders and acid-base interpretation, with original clinical applications.

Component	Contents
Sections 1-3	Shock physiology, clinical differentiation and hemodynamic assessment.
Sections 4-6	Hemorrhage, hemostatic resuscitation, fluid selection and daily prescriptions.
Sections 7-9	Electrolytes, acid-base interpretation, integrated algorithms and review tables.
Section 10	100 extended-case MCQs with option analysis and an answer key.
Section 11	10 structured oral stations with model answers and 20-point rubrics.

Based on Sabiston Chapter 33, with the source passages and contemporary supplementary guidance identified in each teaching section. Cases, calculations, tables and assessments are original educational synthesis. This is not a verbatim translation or a substitute for monitored clinical pathways.

Cumulative teaching and assessment edition. The question bank and oral stations are designed for formative discussion; difficulty, pass marks and examiner reliability have not been formally validated.

1. Foundations of shock

1.1 Shock is inadequate tissue perfusion

Sabiston defines shock in terms of inadequate tissue perfusion. Oxygen delivery is central, but perfusion also supports delivery of other substrates and removal of metabolic products. The clinically important failure occurs at the tissue level; it cannot be defined by a blood-pressure reading alone. [1]

Circulatory failure can arise through inadequate effective circulating volume, pump dysfunction, impaired vascular tone or distribution, or obstruction to flow. These mechanisms can coexist. Detailed differentiation belongs to the next section; the first task is to recognize threatened tissue function and seek the mechanism.

1.2 Compensation can preserve the pressure

Sympathetic responses can increase heart rate, contractility, and vascular resistance. Neuroendocrine responses support intravascular volume. Redistribution of flow helps maintain central circulation, but does not guarantee adequate flow to every tissue. A patient may therefore retain a plausible arterial pressure while peripheral or organ-level signals worsen. [1]

Concept	Meaning	Clinical implication
Pressure	The driving pressure within the circulation.	Necessary to consider, but does not alone describe flow or tissue function.
Flow	Blood reaching the systemic circulation and individual organs.	Can be inadequate despite a pressure maintained by increased resistance.
Oxygen content	Oxygen carried per volume of arterial blood.	Depends predominantly on hemoglobin and its saturation.
Tissue utilization	The ability of tissues to extract and use delivered oxygen and substrates.	Adequate global delivery does not prove normal utilization or distribution.

Clinical pearl
Normal blood pressure does not exclude shock. Conversely, a low pressure requires assessment of cause and perfusion rather than an automatic assumption of volume depletion.

1.3 Recognize a dynamic process

Compare the patient with baseline and the preceding hours. The rate of deterioration, mechanism of injury or illness, treatments already given, and capacity to compensate all influence how an apparently reassuring observation should be interpreted.

Pressure, flow, and oxygen delivery

1.4 Use the equations to identify the missing component

CO = heart rate x stroke volume
MAP - right atrial pressure is approximately CO x systemic vascular resistance

A pressure can be maintained by resistance when flow falls. The pressure-flow relationship is conceptual at bedside: it does not permit precise diagnosis of vascular resistance from a cuff pressure alone.

$CaO_2 = (1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)$
$DO_2 = CO \times CaO_2 \times 10$

Use Hb in g/dL, SaO_2 as a fraction, PaO_2 in mm Hg, and CO in L/min. CaO_2 is then in mL O_2/dL, and the factor 10 converts delivery to mL O_2/min. The constants are approximations. [1]

Worked physiological comparison

Illustrative state	CaO_2	DO_2
Hb 12 g/dL, SaO_2 0.98, PaO_2 100, CO 5 L/min	16.06 mL/dL	803 mL/min
Hb 6 g/dL, same saturation, PaO_2 and CO	8.18 mL/dL	409 mL/min
Hb 12 g/dL, same saturation and PaO_2, CO 2.5 L/min	16.06 mL/dL	401 mL/min

These original calculations show why a normal saturation cannot compensate fully for severe anemia or low flow. The dissolved oxygen contribution is small at the stated PaO_2. The numbers are physiological examples, not transfusion thresholds or individualized treatment targets.

Delivery is not the same as consumption

Oxygen consumption can be expressed as CO x the arterial-to-mixed-venous oxygen content difference, with consistent units. Central venous and mixed venous samples are not interchangeable. A normal or high venous saturation does not by itself establish adequate tissue extraction or regional perfusion. [1]

Clinical pearl
A normal SpO_2 describes saturation of available hemoglobin, not how much hemoglobin is present or how much blood reaches the tissues.

Global measurements and regional perfusion

1.5 A favorable global value can conceal a regional problem

Global arterial pressure, cardiac output, and oxygen-delivery estimates summarize the circulation. They do not directly show whether every organ bed receives adequate flow. Microvascular distribution, regional obstruction, venous congestion, and impaired utilization can produce discordant findings. [1]

Reassuring observation	What it cannot establish alone	What to assess alongside it
Normal cuff pressure	Adequate flow or absence of compensated blood loss.	Trajectory, perfusion examination, mechanism, and relevant tests.
Normal arterial saturation	Adequate oxygen content or delivery.	Hemoglobin, flow, respiratory status, and clinical context.
Improved pressure after treatment	Durable correction of the underlying cause.	Support required, perfusion response, and source control.
Normal/high cardiac output	Uniform regional tissue perfusion.	Organ function, peripheral findings, and possible regional pathology.
Normal/high venous saturation	Normal extraction and absence of cellular dysfunction.	Sampling site, delivery-consumption balance, and other perfusion evidence.

1.6 Hypotension is important, but baseline matters

A major pressure decline from a patient's usual level may be concerning before a conventional hypotension threshold is crossed. Older patients with baseline hypertension may have apparently normal absolute values despite a clinically important change. Conversely, isolated low pressure may have several explanations and requires verification and assessment.

A vasopressor-supported pressure should be interpreted with the dose and trajectory of support. A pressure achieved only with increasing support is not equivalent to the same pressure during spontaneous recovery. Detailed pressure targets and vasoactive choices will be addressed later.

Clinical pearl
Do not choose between pressure and perfusion as though only one matters. Assess both, and explain why they agree or disagree in this patient.

Clinical recognition at the bedside

1.7 Combine signals and follow their direction

Domain	Concerning findings	Important limitations
Brain / behavior	New confusion, restlessness, reduced interaction, or somnolence.	Medication, pain, neurologic disease, and metabolic disorders can contribute.
Skin / periphery	Delayed capillary refill, cool extremities, mottling, or altered skin perfusion.	Temperature, lighting, vascular disease, and examination conditions affect interpretation.
Kidney / output	New low urine output or a downward trend.	Check catheter patency, baseline renal disease, obstruction, and other causes.
Respiration	Tachypnea, increasing effort, or rising oxygen requirements.	Pulmonary disease, pain, acidosis, and other mechanisms may coexist.
Circulation	Pulse changes, narrowing pressure trends, or increasing circulatory support.	Heart rate and pressure can be altered by drugs, pacing, arrhythmia, or compensation.
Overall course	Failure to recover, new weakness, or a cluster of worsening observations.	No single nonspecific finding establishes the mechanism.

A warm periphery or lack of tachycardia does not exclude shock. Medication effects and differing shock mechanisms can alter the classic picture. Examine the patient and reconcile the observations with baseline rather than requiring every textbook sign to appear.

1.8 Shock index is an adjunct

Shock index = heart rate / systolic blood pressure. For example, HR 108/min and SBP 120 mm Hg give an index of 0.90. Sabiston discusses its usefulness and the advantage of considering multiple variables together. It is not a universal rule-out test or an automatic treatment prescription. [1]

A blunted heart-rate response can make a reassuring index misleading. A raised index can also have explanations other than hemorrhage. Interpret the index with the mechanism, perfusion examination, trends, and investigations.

Clinical pearl
Use measurements to strengthen bedside judgment. Do not wait for a score or a complete classic pattern when the patient's course is concerning.

Lactate, base deficit, and hemoglobin

1.9 Lactate is a warning signal, not a diagnosis

Lactate reflects the balance between production and removal. Hypoperfusion can contribute, but adrenergic stimulation, altered metabolism, medications, and impaired clearance may also raise the concentration. Sabiston emphasizes that lactate is an active metabolite and cannot be interpreted simply as proof of anaerobic metabolism. [1]

Finding	Reasonable interpretation	Unsafe shortcut
Elevated lactate	Assess perfusion, likely mechanism, clearance, and the trend.	All elevation proves hemorrhage or demands more fluid.
Normal early lactate	One reassuring component at that time.	Early bleeding or regional ischemia is excluded.
Falling lactate	May support improvement when other findings agree.	The source is controlled and all organs are adequately perfused.
Persistent elevation	Reassess ongoing illness, production, clearance, and treatment effects.	Continue fluid until the number is normal regardless of tolerance.

1.10 Base deficit needs an acid-base context

A larger base deficit can accompany metabolic acidosis and physiological stress, but chloride-rich fluid administration and renal dysfunction can alter it. Interpret the blood gas with chloride, lactate, renal status, and the clinical course. Laboratories may display negative base excess rather than a positive deficit: BE -6 mmol/L corresponds to a base deficit of 6 mmol/L under that convention. [1]

1.11 A normal initial hemoglobin does not exclude bleeding

Hemoglobin is a concentration, not a direct measurement of total blood lost. Timing, redistribution, and administered fluids affect it. Do not delay assessment of suspected bleeding because the initial value is normal; serial findings and the clinical picture are more informative. [1]

Clinical pearl
Current sepsis guidance supports serial lactate and capillary refill as adjuncts, and cautions against continuing fluids solely to normalize lactate. This is a supplement, not a universal lactate-based fluid rule. [3]

Occult hypoperfusion: recognize discordance

1.12 When the pressure looks acceptable but the patient does not

Occult hypoperfusion describes clinically important perfusion impairment not obvious from conventional pressure or pulse measurements alone. It is a recognition problem rather than a separate cause of shock. Preserved pressure with abnormal perfusion signals or metabolic findings should prompt an integrated assessment, not automatic reassurance or a single predetermined therapy.

Pattern	Judgment task
Acceptable pressure plus new oliguria, cool skin, and rising lactate	Consider compensated circulatory failure while evaluating alternative contributors and the likely cause.
Acceptable pressure plus blunted heart rate on a beta blocker	Do not require tachycardia to assess possible blood loss or low flow.
Improved pressure but persistent pain and metabolic deterioration	Reassess regional pathology and whether the underlying problem has been corrected.
Lactate elevation with improving perfusion and impaired clearance	Assess the whole course; avoid treating the laboratory number in isolation.

1.13 An initial surgical assessment pathway

• Recognize the change from baseline and assess immediate threats; escalate to the appropriate expertise.

• Verify observations and evaluate mental status, peripheral perfusion, urine output, and the trajectory of support.

• Consider hemorrhage, infection, pump dysfunction, obstruction, and mixed mechanisms.

• Obtain targeted laboratory tests and bedside imaging when they inform timely decisions; do not delay emergency treatment for complete data.

• Provide cause-directed support while pursuing definitive management of the underlying problem.

• Reassess both response and tolerance. Persisting abnormalities require a revised explanation, not automatic repetition of fluid.

Serial tissue-perfusion assessment, targeted fluid use, and echocardiography to help determine shock type are emphasized in current ESICM guidance. These are identified as supplements to the textbook foundation. [2]

Clinical pearl
Finding possible hypoperfusion is the start of a mechanism-based decision. It is not proof that every patient needs the same fluid or the same pressure target.

Extended case: the apparently stable patient

1.14 Preserved pressure after major surgery

A 76-year-old man undergoes major abdominal surgery. His usual systolic pressure is around 155 mm Hg, and he takes a beta blocker. During the evening, pressure is 118/72 mm Hg and pulse 82/min. SpO2 is 98% on unchanged low-flow oxygen. A junior clinician considers these values reassuring. Over several hours, however, the patient becomes less interactive, his hands become cool, urine output decreases, and abdominal discomfort increases. The operative drain contains little fluid. Initial hemoglobin is 11.1 g/dL, close to the early postoperative value. Lactate is 4.1 mmol/L. The patient has no known severe chronic liver disease, but the mechanism of deterioration has not been established.

One team member proposes waiting for hypotension or a hemoglobin fall. Another wants repeated crystalloid solely until lactate normalizes. The senior surgeon is asked to assess whether the current information can exclude significant bleeding and whether the patient's preserved pressure reflects adequate tissue perfusion. The team can obtain urgent bedside assessment, appropriate investigations, and anesthesia or critical-care support.

Model judgment

Recognize discordance: Pressure and pulse do not explain the worsening mental status, peripheral findings, and urine output. Baseline hypertension and beta blockade further limit reassurance from absolute values.

Do not rule out hemorrhage: A normal initial hemoglobin or little drain output does not exclude clinically important concealed blood loss. Assess the operative context and possible bleeding sites alongside other shock mechanisms.

Use lactate appropriately: It strengthens concern but does not establish the source. Investigate and support the patient urgently without waiting for every classic sign.

Reassess therapy: Treatment should address the mechanism, response, and tolerance. Repeated fluid purely to chase lactate may delay definitive care or add harm.

Clinical pearl
The most important signal is the patient's deteriorating trajectory, not whether one conventional threshold has been crossed.

Extended case: a persistent lactate

1.15 Avoid turning a biomarker into a fluid prescription

A 59-year-old woman with intra-abdominal infection receives initial treatment and a source-control intervention. She has chronic liver disease. During subsequent review, pressure is maintained on decreasing support, interaction improves, capillary refill and peripheral temperature improve, and urine output is recovering. Lactate remains elevated at 3.6 mmol/L after previously reaching 5.2 mmol/L. She has already accumulated a positive fluid balance and now has increasing edema and oxygen requirements. A resident proposes further fluid boluses until lactate is normal. Another clinician suggests ignoring lactate altogether because liver disease may impair its removal.

The surgeon has not yet determined whether source control is complete, whether any further fluid would improve flow, or what explains the new respiratory findings. The patient requires continued review. The decision must incorporate both possible persistent illness and the potential harm of indiscriminate treatment. A falling biomarker and improved peripheral examination are encouraging but cannot establish that every regional problem has resolved.

Model judgment

Keep the differential open: Persistent lactate may reflect ongoing production, reduced removal, or residual perfusion abnormalities. Chronic liver disease is relevant but does not prove the entire explanation.

Do not chase normalization with fluid: Review the likelihood of benefit and the evidence of intolerance before additional administration. Worsening oxygen requirements and edema make a routine repeat bolus particularly questionable.

Check the underlying cause: Reassess the source, physiology, and response to the overall treatment. Seek decision-relevant information rather than treating either the lactate or an apparently improved pressure as a complete answer.

Use trends together: Continue serial examination, relevant laboratory assessment, and support review. Document which findings support recovery and which remain unexplained.

Clinical pearl
Neither persistent lactate nor its plausible alternative explanation should end the clinical assessment. Treat the patient and mechanism, with repeated evaluation of benefit and harm.

Section 1: final review and sources

1.16 High-yield distinctions

Do not confuse	Practical distinction
Blood pressure and perfusion	Pressure is one component; flow, regional distribution, and tissue function also matter.
Saturation and oxygen delivery	Normal saturation does not correct low hemoglobin or low cardiac output.
Lactate elevation and proven hypoxia	Production, removal, stress, drugs, and perfusion all influence lactate.
Normal hemoglobin and absent hemorrhage	Concentration and total blood loss are different quantities.
Temporary response and definitive control	Support may improve observations while the cause persists.
Possible hypoperfusion and automatic fluid need	Choose treatment from the mechanism, expected response, and tolerance.
Global recovery and regional safety	Persistent focal findings require explanation despite improved global variables.

The surgeon's questions

Is perfusion adequate now? What is the likely mechanism? What must be treated before further delay? What finding would support or refute my explanation? Has the patient improved, and has treatment created harm?

Sources

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. In: Sabiston Textbook of Surgery. 22nd ed. Chapter 33. Core passages reviewed: printed pp. 221-222, 228-230, 235-238. Calculations and cases in this section are original educational applications.

[2] ESICM. Guideline on circulatory shock and haemodynamic monitoring in adult ICU patients, 2025. Official society overview: https://www.esicm.org/esicm-guideline-circulatory-shock-haemodynamic-monitoring/. Used to supplement serial perfusion assessment, targeted fluids, and echocardiographic assessment.

[3] Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2026. Official SCCM recommendations: https://www.sccm.org/clinical-resources/guidelines/guidelines/surviving-sepsis-campaign-international-guidelines-for-management-of-sepsis-and-septic-shock-2026. Serial lactate and capillary refill are conditional recommendations with low-certainty evidence; the guidance emphasizes individualized reassessment rather than fluid solely to normalize lactate. Accessed 2 November 2026.

Next section: Types of shock and clinical differentiation.

2. Types of shock and clinical differentiation

2.1 Classify the mechanism, then identify the cause

The useful bedside question is which failure currently limits effective tissue perfusion: insufficient circulating volume, primary pump dysfunction, loss of vascular tone and maldistribution, or mechanical obstruction to circulation. These four mechanisms provide a working differential rather than mutually exclusive labels. The same patient can move between phenotypes during illness and treatment. [1,2]

The modern four-mechanism framework below is an educational synthesis. Sabiston also reproduces historical classifications; the placement of tamponade within an older pump-failure category should not obscure its modern classification as obstructive shock.

Mechanism	Primary failure	Representative surgical settings
Hypovolemic	Inadequate venous return from loss of effective circulating volume.	Hemorrhage; gastrointestinal losses; severe fluid sequestration.
Cardiogenic	Primary ventricular or other cardiac dysfunction reduces forward flow.	Perioperative infarction; acute ventricular failure; severe valve dysfunction or arrhythmia.
Distributive	Reduced vascular tone and abnormal distribution of flow; relative hypovolemia often coexists.	Sepsis; anaphylaxis; neurogenic shock; postoperative vasoplegia.
Obstructive	Mechanical limitation of filling or ejection outside primary myocardial failure.	Tamponade; tension pneumothorax; high-risk pulmonary embolism.

2.2 Begin with time, context, and trajectory

Ask what changed immediately before deterioration: an operation, injury, medication, line insertion, positive-pressure ventilation, new arrhythmia, or interruption of prophylaxis. Determine whether the patient is worsening despite support. Examine for bleeding, infection, pulmonary asymmetry, venous congestion, and cardiac dysfunction in parallel with resuscitation.

Clinical pearl
A shock label must produce a useful action: identify the threat, select a mechanism-directed intervention, and define how its effect will be reassessed.

Hemodynamic patterns: clues, not diagnostic rules

2.3 Interpret a pattern in its treatment context

Phenotype	Cardiac output	SVR	Filling pressure / examination
Hypovolemic	Usually reduced	Usually increased	Filling pressures often low; cool skin and narrow pulse pressure may occur.
LV cardiogenic	Reduced	Usually increased	Left-sided filling pressure often elevated; pulmonary congestion may be present.
RV cardiogenic	Reduced	Usually increased	Systemic venous congestion may predominate; lungs may be clear.
Distributive	Often high early; can be low	Usually reduced	Filling pressure variable; warm skin may occur but is not required.
Obstructive	Usually reduced	Usually increased	Upstream venous pressure may rise; pattern depends on the obstruction.

These are typical untreated or predominant patterns, not mandatory criteria. Fluids, vasopressors, ventilation, pre-existing pulmonary hypertension, and mixed mechanisms can alter every column. Early distributive shock can coexist with low output from volume depletion or myocardial dysfunction. Severe sepsis may be cool rather than warm.

2.4 Separate congestion, preload, and fluid responsiveness

An elevated central venous pressure indicates pressure at the measurement site; it does not establish adequate effective circulating volume or exclude a potential response to fluid. A low value does not prove that more fluid is appropriate. Neither a single CVP nor an isolated inferior vena cava image is a reliable stand-alone prescription for fluid.

Fluid responsiveness means that stroke volume or cardiac output increases with a preload challenge. It is different from a clinical need for fluid and different from fluid tolerance. When applicable, dynamic assessment is preferable to static preload markers. Interpret a passive leg raise with a timely flow measurement, not blood pressure alone. [2]

Clinical pearl
The patient can be edematous, congested, and still have inadequate effective forward flow. Repeated fluid may worsen the problem even when hypotension persists.

Hypovolemic versus cardiogenic shock

2.5 Hypovolemia: find the loss and its consequences

Hemorrhage removes both circulating volume and oxygen-carrying capacity. Nonhemorrhagic losses primarily reduce circulating volume, although the clinical consequences depend on duration and associated electrolyte disturbances. Review the operative field, chest, abdomen, pelvis, retroperitoneum, gastrointestinal tract, and external losses as the context requires. Low drain output cannot exclude concealed bleeding.

Tachycardia, cool extremities, delayed capillary refill, reduced pulse pressure, oliguria, and a small hyperdynamic ventricle may support hypovolemia. None is obligatory or specific. Older patients, beta-blocked patients, and patients with pacing may lack tachycardia. A normal early hemoglobin does not exclude important acute hemorrhage. [1]

A transient improvement with volume support does not prove bleeding has stopped. In suspected major hemorrhage, expedite hemorrhage control and appropriate blood-based resuscitation through the local pathway; serial crystalloid challenges must not substitute for definitive care.

2.6 Cardiogenic shock: identify the cardiac lesion

Consider ischemia, new arrhythmia, ventricular dysfunction, acute valve disease, or a mechanical complication. Obtain ECG and focused echocardiography promptly, with appropriate specialist involvement. Troponin elevation can also occur with sepsis or supply-demand injury; it does not independently identify the shock mechanism.

Observation	Interpretation that helps	Important limitation
Pulmonary edema with poor LV function	Supports a left-sided cardiac contribution.	Edema also occurs with fluid overload and permeability injury.
Venous congestion with clear lungs	Consider RV failure or obstruction.	Does not prove pulmonary embolism or identify a single cause.
Small, vigorously contracting LV	May indicate underfilling or low afterload.	Does not independently establish volume depletion.
Reduced ejection fraction	Supports systolic dysfunction in context.	Chronic dysfunction may not explain the new deterioration.

Clinical pearl
Do not treat every hypotensive patient as volume depleted, and do not attribute every postoperative deterioration to an old low ejection fraction.

Distributive shock: distinguish the cause

2.7 Septic and vasoplegic phenotypes

Sepsis can produce vasodilation, capillary leak, altered microcirculatory flow, and myocardial dysfunction. Fever and warm extremities may be absent. In a postoperative patient, investigate an anastomotic leak, collection, ischemic tissue, infected device, and other plausible sources. Recognition of distributive physiology does not remove the need to search for hemorrhage or obstruction.

Vasodilation after anesthesia or major surgery is a physiological possibility, not a safe default explanation for persistent shock. Reassess the timing, medication exposure, source-control status, volume losses, and cardiac findings. More than one mechanism may require treatment.

2.8 Anaphylaxis: clinical recognition takes priority

Abrupt airway, breathing, or circulation compromise after a plausible exposure should prompt consideration of anaphylaxis. Skin or mucosal changes can be absent. Stop the suspected trigger when feasible, call for help, and initiate the anaphylaxis pathway. Intramuscular adrenaline is first-line treatment in the general emergency pathway; antihistamines must not delay it. Specialist perioperative teams may use monitored intravenous adrenaline according to their dedicated protocol. [3]

2.9 Neurogenic shock is not spinal shock

Neurogenic shock is a circulatory consequence of impaired sympathetic tone, commonly with high spinal cord injury. Vasodilation and relative bradycardia may be present. Spinal shock describes acute loss or depression of neurological reflex activity below the lesion; it is not the name for circulatory failure.

In an injured patient, do not use bradycardia, warm skin, or a spinal lesion to exclude hemorrhage. Assess for associated bleeding and other immediately reversible causes while protecting the spine and supporting perfusion. A neurological mechanism and hemorrhagic shock can coexist.

Clinical pearl
Warm shock does not prove sepsis; absent rash does not exclude anaphylaxis; bradycardia does not exclude traumatic hemorrhage.

Obstructive shock and the bedside sequence

2.10 Recognize a mechanically correctable threat

Cause	Decision-relevant findings	Priority and pitfall
Tension pneumothorax	Sudden respiratory and circulatory deterioration; unilateral air-entry reduction; rising ventilator pressures in a compatible setting.	If clinically suspected in an unstable patient, immediate decompression by a trained clinician; do not await radiography. Tracheal deviation is late and unreliable.
Cardiac tamponade	Pericardial fluid plus compatible hemodynamic/echo physiology; postoperative collections can be loculated.	Urgent drainage strategy appropriate to cause and setting. Effusion alone is not tamponade; classic physical signs can be absent.
High-risk pulmonary embolism	Abrupt dyspnea or collapse; RV pressure overload and reduced LV filling in context.	Urgent reperfusion assessment and expert pathway. RV dilation alone is nonspecific; definitive imaging depends on stability and feasibility.

2.11 A practical differentiation sequence

1. Recognize the threat: Assess airway, breathing, circulation, perfusion, monitoring, and need for immediate help. Treat a strongly suspected immediately reversible cause while evaluation continues.

2. Search in parallel: Examine for hemorrhage and infection, review exposures and procedures, obtain ECG, and use focused cardiac and lung ultrasound with appropriate expertise. Echocardiography is a first-line imaging tool for shock phenotype. [2]

3. Ask whether imaging will change the next action: An unstable patient should not undergo an unsafe transfer merely to complete a diagnostic checklist. Conversely, uncertainty in a sufficiently stable patient may justify definitive imaging before a hazardous intervention.

4. Reassess after each intervention: Track tissue perfusion, flow when available, congestion, oxygen requirement, and support needs. If the response is absent or brief, reconsider the mechanism and unresolved source.

Clinical pearl
The sequence is not “finish classification, then treat.” Diagnosis, support, and correction of a time-critical cause proceed together.

Extended case: bleeding with a failing heart

2.12 Resolve competing explanations

A 74-year-old man is reviewed four hours after major abdominal surgery. He has ischemic cardiomyopathy with a documented ejection fraction of 30%, takes a beta blocker, and received substantial crystalloid during a prolonged operation. His pressure has fallen from 136/78 to 92/58 mm Hg, heart rate is 86/min, and he is drowsy with cool extremities and minimal urine output. Oxygen requirement has increased. There are bilateral crackles and the abdomen is becoming distended. The drain contains only 40 mL of blood-stained fluid. Hemoglobin is 10.8 g/dL compared with 11.5 g/dL shortly after surgery; lactate has increased from 1.9 to 4.4 mmol/L. A limited bedside echo shows poor LV systolic function similar to the previous report; image quality prevents reliable assessment of new regional abnormalities. The right ventricle is not clearly enlarged.

One clinician attributes the shock entirely to cardiomyopathy and recommends diuresis. Another proposes repeated crystalloid boluses until urine output recovers. The surgeon must decide whether either explanation accounts for the trajectory, whether imaging transfer is safe, and what action can establish or control an ongoing surgical cause. There is no single reassuring measurement that resolves the conflicting signs.

Model judgment and analysis

Working diagnosis: Mixed physiology is plausible: concealed hemorrhage or another postoperative source of underfilling may coexist with poor cardiac reserve and fluid-related pulmonary congestion. Chronic low EF does not establish the new cause; modest hemoglobin change, beta blockade, and low drain output do not exclude bleeding.

Immediate priorities: Escalate to senior surgical, anesthesia, and critical-care assessment; support oxygenation and perfusion; review operative events and losses; evaluate ECG and targeted echo findings; rapidly investigate the abdomen and other plausible bleeding sites. Activate the hemorrhage pathway if clinical evidence supports major ongoing bleeding.

Choose the next definitive step: Determine whether the patient can safely undergo diagnostic imaging or needs urgent operative or interventional control. Do not allow serial laboratory sampling or an incomplete echo to delay necessary source control. Base the route on stability, likely source, and available expertise.

Reject both automatic prescriptions: Routine diuresis may worsen effective circulating volume before hemorrhage is addressed. Repeated crystalloid may aggravate lung congestion without correcting blood loss. Select support from the current mechanism, response, and tolerance, then reassess.

Clinical pearl
A pre-existing cardiac diagnosis changes tolerance of resuscitation; it must not become an explanation for every new episode of shock.

Extended case: sudden collapse after a line

2.13 Let timing change the differential

A 63-year-old woman is ventilated after emergency surgery for a perforated viscus. She has received antibiotics and initial circulatory support, and her pressure and peripheral perfusion were improving. A right subclavian central line is inserted. Ten minutes later, pressure falls to 62/38 mm Hg, oxygen saturation drops, and peak airway pressure rises. The monitor shows sinus tachycardia. Air entry is much quieter on the right, although auscultation is difficult in the crowded room. A resident asks for a portable chest radiograph to confirm the line position. Another proposes increasing vasopressor support because she already has septic shock. The abdomen is unchanged and there is no new external bleeding. The ultrasound machine is in another room; a definitive image will take several minutes.

The team must decide whether the established diagnosis remains sufficient, whether the new event is a complication of treatment, and whether waiting for confirmation is justified. The new deterioration occurs on top of a real pre-existing septic illness, so correcting the acute problem will not necessarily eliminate every requirement for support.

Model judgment and analysis

Recognize the new threat: The timing, respiratory asymmetry, rising airway pressure, and profound instability strongly raise concern for tension pneumothorax after line placement. Prior sepsis does not explain away this pattern.

Act without harmful delay: Call for immediate help and arrange emergency decompression and definitive pleural management by an appropriately trained clinician under the local protocol. Do not wait for a chest radiograph when clinical suspicion and instability warrant emergency treatment. Rapid available ultrasound can assist but must not create delay.

Maintain a parallel safety check: Assess the airway and ventilator circuit and consider tube displacement, other procedural complications, and bleeding. These checks accompany action rather than becoming a reason to defer treatment of a likely lethal obstruction.

Reassess the remaining physiology: Improvement after decompression supports the working mechanism but does not prove full recovery. Reevaluate ventilation, perfusion, line complications, source control, and residual distributive or myocardial dysfunction.

> **Clinical pearl**
> An existing shock diagnosis must be reopened when a new procedure is followed by an abrupt, mechanistically different deterioration.

Extended case: a spinal lesion and two sources of shock

2.14 Avoid premature diagnostic closure

A 28-year-old man arrives after a high-speed collision. He is awake but has weakness in all four limbs and reduced sensation below the upper chest. Blood pressure is 84/46 mm Hg and heart rate 58/min. His hands are relatively warm. Oxygen saturation is 97% on supplemental oxygen. He has pelvic pain, lower abdominal tenderness, and bruising over the flank. Initial hemoglobin is 13.2 g/dL. A focused abdominal ultrasound does not show free intraperitoneal fluid, and the chest examination is symmetric. The first clinician concludes that the low pressure and slow pulse establish isolated neurogenic shock. During preparation for further imaging, the patient becomes less interactive, capillary refill worsens, and lactate returns at 4.8 mmol/L. The pelvic injury has not yet been characterized and the retroperitoneum has not been evaluated.

The surgeon must decide whether the clinical pattern provides a complete explanation, what the negative findings actually exclude, and whether the proposed transfer remains safe. The neurological injury is credible, but the mechanism of trauma creates a separate risk of life-threatening hemorrhage.

Model judgment and analysis

Maintain a mixed-shock hypothesis: Loss of sympathetic tone can explain relative bradycardia and vasodilation, but it does not exclude bleeding. Neurogenic and hemorrhagic mechanisms may coexist; reflex loss from spinal shock is a separate neurological concept.

Interpret the negatives correctly: Early normal hemoglobin is not a blood-loss exclusion test. A negative focused abdominal examination does not rule out pelvic or retroperitoneal hemorrhage and does not establish safety for transport in a deteriorating patient.

Address immediate priorities: Continue trauma resuscitation, spinal precautions, rapid reassessment of airway and breathing, and urgent evaluation and control of possible hemorrhage through the trauma pathway. Use pelvic stabilization when indicated by the suspected injury and local protocol. Coordinate surgical, anesthesia, and spine expertise.

Reevaluate transport and treatment response: Choose imaging versus direct hemorrhage-control intervention from current stability and the likely source. Circulatory support for sympathetic failure may be necessary, but a pressure response to a vasopressor cannot certify that bleeding has stopped.

Clinical pearl
A distinctive sign can identify one component of shock while concealing another. The mechanism of injury and the evolving perfusion examination retain diagnostic weight.

Section 2: final comparison and decision checks

2.15 Common errors that change outcomes

Premature conclusion	Better judgment
Warm extremities mean sepsis.	Consider other vasodilatory causes; assess infection, exposures, and the full trajectory.
Clear lungs exclude cardiac shock.	RV failure may present with venous congestion and clear lungs.
RV dilation proves pulmonary embolism.	Interpret with prior disease, ventilation, clinical probability, and appropriate confirmatory evidence.
A fluid response proves hypovolemia is the only problem.	Several shock states are preload responsive; a temporary response does not identify the cause or establish fluid tolerance.
Persistent hypotension requires more fluid.	Reassess flow, vascular tone, obstruction, ongoing losses, and congestion.
Normal initial hemoglobin excludes bleeding.	Concentration may initially remain normal despite acute blood loss.
A new shock phenotype replaces the original disease.	An added obstructive, hemorrhagic, or cardiac component may coexist with the earlier process.

The surgeon's final bedside statement

State the predominant mechanism, the dangerous alternatives still under evaluation, the intervention that cannot wait, and the response expected if your explanation is correct. Document unresolved findings rather than hiding them inside a single diagnostic label.

Sources and scope

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. Sabiston Textbook of Surgery. 22nd ed. Chapter 33, beginning p. 221. Mechanistic framework, tables, decision sequence, and cases here are original teaching synthesis based on the chapter foundations reviewed in Section 1; not a verbatim reproduction.

[2] Monnet X, Messina A, Greco M, et al. ESICM guidelines on circulatory shock and hemodynamic monitoring 2025. Intensive Care Medicine. doi:10.1007/s00134-025-08137-z. Official summary: https://www.esicm.org/esicm-guideline-circulatory-shock-haemodynamic-monitoring/.

[3] Resuscitation Council UK. Special circumstances guidelines, 2025; and Emergency treatment of anaphylactic reactions, 2021. https://www.resus.org.uk/professional-library/2025-resuscitation-guidelines/special-circumstances-guidelines. Guidance supplements the textbook; accessed 2 November 2026.

Next section: Hemorrhagic shock: assessment, hemorrhage control, and resuscitation priorities.

3. Hemodynamic assessment and response to resuscitation

3.1 Replace isolated targets with a coherent physiological assessment

Hemodynamic assessment should establish whether tissue perfusion remains inadequate, identify the dominant mechanism, and determine whether the next intervention is likely to improve flow without unacceptable harm. A monitor provides observations; the surgeon must connect them to the illness, source-control status, and response to treatment. Sabiston emphasizes the limitations of global measurements and the failure of historical strategies that pursued supranormal oxygen delivery as an end in itself. [1]

Use four linked domains: pressure, flow, tissue perfusion, and treatment tolerance. A favorable change in one domain does not guarantee improvement in the others. For example, vasoconstriction can raise arterial pressure without correcting low forward flow, while a fluid bolus can increase stroke volume and simultaneously worsen pulmonary congestion.

Domain	Useful observations	Question it answers
Pressure	Arterial pressure trend; support required; waveform quality.	Is driving pressure plausibly adequate in this patient?
Flow and mechanism	Focused echo; stroke volume or cardiac output when indicated; rhythm.	What limits forward circulation, and did an intervention improve it?
Tissue perfusion	Mental status; capillary refill; skin temperature; urine trend; lactate context.	Are organs and peripheral tissues showing recovery?
Tolerance and harm	Oxygen requirement; lung findings; venous congestion; fluid balance.	What is the physiological cost of continuing treatment?

3.2 Establish the baseline before interpreting change

Record time, position, rhythm, ventilator conditions, vasoactive support, recent fluid or blood administration, and the method used to measure flow. A change that follows several simultaneous interventions cannot confidently be attributed to one of them. Do not delay urgent treatment merely to obtain a perfect baseline; document the uncertainty.

Clinical pearl
Successful resuscitation is sustained recovery of perfusion with control of the cause and acceptable treatment burden. A better blood pressure alone is an incomplete endpoint.

Bedside endpoints and discordant signals

3.3 Use serial examination as a measurement system

Signal	Evidence of improvement	Why interpretation may fail
Mental status	Improved interaction relative to baseline.	Sedation, delirium, neurologic injury, or metabolic disease can dominate.
Capillary refill / skin	Improved refill, warming, or less mottling using a consistent technique.	Ambient temperature, vasopressors, peripheral vascular disease, and technique alter findings.
Urine output	Recovery over a clinically relevant interval.	Obstruction, chronic kidney disease, established AKI, and diuretics confound it; response may lag.
Lactate	A favorable trajectory in the clinical context.	Production, adrenergic stimulation, hepatic clearance, and sampling affect it.
Arterial pressure	Adequate pressure with stable or decreasing support.	A higher number achieved with escalating support may conceal deterioration.

In many adults, urine output below approximately 0.5 mL/kg/h is a warning signal, not an automatic indication for a fluid bolus. Check catheter patency, duration, baseline kidney function, congestion, and the overall perfusion pattern. Do not force a short-term urine response with repeated fluid when flow does not improve or harm is appearing.

3.4 Read a trajectory rather than a single lactate

A decreasing lactate can support recovery but does not establish complete source control or exclude regional ischemia. Persistent elevation requires explanation rather than automatic fluid. A rising value with worsening examination and escalating support is more concerning than an isolated stable elevation with otherwise improving physiology. Serial lactate and capillary refill are adjuncts in current sepsis guidance, with conditional recommendations and low-certainty evidence. [3]

Percentage lactate reduction = (initial lactate - later lactate) / initial lactate x 100. A fall from 6 to 4 mmol/L is a 33% reduction over the stated interval. This calculation describes concentration change, not directly measured metabolic clearance, and is not a universal treatment target.

Clinical pearl
When signals disagree, identify the plausible confounder and seek the measurement that can resolve the next decision. Do not discard the inconvenient observation.

Choose the monitor to answer a question

3.5 Escalate monitoring when initial assessment is insufficient

Verify cuff size and position, signal quality, and rhythm before acting on an unexpected pressure. An arterial catheter permits beat-to-beat assessment and repeated sampling in persistent or supported shock; check leveling, zeroing, and damping. A technically poor waveform can create a false physiological explanation. Monitoring procedures must accompany immediate care rather than delay control of hemorrhage or another time-critical cause.

Focused echocardiography can rapidly examine LV and RV function, pericardial pathology, and filling patterns. Lung ultrasound and clinical examination add information about respiratory tolerance. Repeat imaging after an important change. A normal-looking ejection fraction does not prove normal cardiac output, and an isolated inferior vena cava diameter does not prescribe fluid. [1,2]

Tool	Decision it can support	Limitations that matter
Arterial catheter	Confirm pressure trajectory and response to support.	Position and waveform artifacts; pressure is not flow.
Echo / LVOT VTI	Identify cardiac or obstructive contributions; compare forward-flow changes.	Operator skill, acoustic window, alignment, rhythm, and beat selection.
Pulse contour / other CO method	Track stroke volume or flow during intervention.	Accuracy depends on device, calibration, vascular tone, rhythm, and clinical setting.
Central venous access	Deliver support; obtain venous samples; assess venous pressure in context.	CVP is not a stand-alone volume or responsiveness test; catheter complications.
Pulmonary artery catheter	Selected complex or refractory cases requiring specialist hemodynamic clarification.	Not routine for every patient; invasive risks and interpretation requirements.

3.6 Precision determines whether a change is believable

For serial LVOT velocity-time integral measurements, use a consistent window, sample position, and careful beat selection. If the outflow-tract area is treated as unchanged, percentage VTI change approximates percentage stroke-volume change. Cardiac output also depends on heart rate. A tiny apparent improvement within measurement variability should not trigger more fluid.

Clinical pearl
Use a device when its result can alter management. More numbers do not compensate for an unresolved surgical source or an invalid measurement.

Fluid responsiveness, need, and tolerance

3.7 Three separate decisions

Need: Is there a clinically important perfusion problem that volume expansion might address? **Responsiveness:** Will increasing preload increase stroke volume or output? **Tolerance:** Can volume be added without excessive pulmonary, venous, or tissue congestion? A positive response test answers only the second question.

A patient on the ascending portion of the Frank-Starling relationship may increase stroke volume with preload. This can occur in several shock mechanisms and does not diagnose isolated hypovolemia. Conversely, a nonresponse should prompt review of test validity and alternative mechanisms rather than reflexively giving a larger bolus.

3.8 Passive leg raising: a reversible preload challenge

When safe and technically feasible, move from a semirecumbent position to a supine trunk with elevated legs, while measuring the transient change in flow promptly. Measure before, during, and after return to baseline. Keep other treatment and ventilation changes stable when possible. A pulse-pressure or cuff-pressure response alone is less reliable than a direct stroke-volume or output assessment.

An increase around 10% in reliably measured cardiac output or stroke volume is a commonly used indicator of responsiveness, but the threshold depends on method and precision; borderline results are uncertain. PLR can be useful during spontaneous breathing and arrhythmia if the flow method and beat averaging are suitable. Pain, movement, unsafe positioning, and intra-abdominal hypertension can compromise interpretation; a negative test under these conditions may be misleading. [2]

Worked response assessment

Observation	Interpretation
VTI increases from 14 to 16 cm during a technically sound PLR; heart rate remains stable.	Increase = 14.3%. Supports preload responsiveness if measurement is reliable.
The same patient has worsening oxygenation and venous congestion.	Responsiveness does not establish that further volume has a favorable benefit-harm balance.
VTI increases from 14 to 14.4 cm with variable Doppler alignment.	The 2.9% change may be measurement noise; do not label this a definite response.

Clinical pearl
Give fluid for a plausible perfusion benefit, supported by a valid test when appropriate, and constrained by tolerance. A positive test is not a standing order for repeated boluses.

Dynamic indices and a controlled fluid challenge

3.9 Know when PPV and SVV are interpretable

Pulse pressure variation and stroke volume variation use heart-lung interactions. They perform best under appropriate controlled mechanical ventilation with a regular rhythm, no significant spontaneous effort, and sufficient cardiopulmonary interaction. A numeric display does not verify these conditions. Low tidal volume, poor lung compliance, arrhythmia, spontaneous breathing, RV dysfunction, and altered thoracoabdominal pressures can reduce reliability. Do not change lung-protective ventilation merely to make a routine index appear usable.

A conventional PPV threshold in the low teens is not a universal cutoff. Treat intermediate values and invalid conditions as uncertainty, not a binary answer. A high value can reflect RV afterload effects rather than beneficial LV preload recruitment. A low value with low tidal volume cannot reliably exclude responsiveness. [2]

3.10 Design a fluid challenge before administering it

A fluid challenge is a time-limited therapeutic test, not an open-ended infusion. Specify the indication, fluid appropriate to the mechanism, amount and rate, measurement of benefit, and stopping criteria. A small crystalloid challenge, often 250 mL in adult practice, is an illustrative approach rather than a universal prescription. Major hemorrhage requires hemorrhage control and blood-based resuscitation as indicated, not a sequence of crystalloid tests.

Before	During and immediately after	What to do with the result
Define the perfusion deficit; note flow, pressure, oxygenation, and congestion.	Recheck stroke volume / output using the same reliable method and examine tolerance.	If flow and perfusion improve without important harm, reassess whether any further volume is actually needed.
Select the dose, rate, and stop conditions for this patient.	Stop for meaningful worsening such as respiratory distress or rapidly increasing congestion.	If there is no useful benefit, stop repeated empirical boluses and reconsider mechanism and source.

3.11 Alternative functional tests require expertise

An end-expiratory occlusion test can assess flow change in suitable ventilated patients without significant spontaneous effort. Its small signal demands adequate measurement precision. Do not interpret a subtle VTI fluctuation as a reliable response if the method cannot distinguish it from noise. No functional test should delay treatment of an immediately reversible obstruction or active bleeding.

Venous oxygenation, pressure targets, and stopping rules

3.12 Use venous signals to explain, not certify, perfusion

ScvO2 from a central venous catheter is not identical to mixed venous SvO2 from the pulmonary artery. A low value may reflect inadequate flow or arterial oxygen content, or increased oxygen demand. A normal or high value does not exclude maldistribution, impaired extraction, or regional hypoperfusion. Check sampling site, arterial saturation, hemoglobin, demand, and flow before selecting a treatment. [1]

An elevated central venous-to-arterial carbon dioxide partial-pressure difference may support concern for inadequate effective flow in context. It is not a direct tissue hypoxia meter or an independent indication for fluid. Use correctly paired samples and interpret alongside venous oxygenation and clinical findings. Avoid treating a single cutoff as a substitute for mechanism assessment.

3.13 Individualize pressure and avoid supranormal goals

MAP near 65 mm Hg is a common initial reference in adult septic shock, not a universal target for all shock states. Baseline pressure, cerebral or coronary disease, trauma context, venous pressure, organ response, and treatment adverse effects can change the appropriate goal. A target is provisional: verify that reaching it improves the patient. Do not pursue supranormal cardiac output or oxygen delivery solely to normalize a calculated number. [1,3]

3.14 A practical reassessment loop

Question	Action
Is the cause controlled?	Revisit hemorrhage, infection, ischemia, obstruction, and the need for definitive intervention.
Is perfusion improving?	Integrate examination, urine trend, lactate context, pressure, and support needs.
Would another intervention improve flow?	Use mechanism and a valid functional assessment where appropriate.
Is treatment tolerated?	Review lung findings, oxygen needs, venous congestion, and cumulative balance.
Should resuscitation be stopped or changed?	Stop ineffective boluses; adjust mechanism-directed support; escalate diagnostic or definitive care.

Clinical pearl
Stopping additional fluid is not stopping treatment. It may be the decision that permits appropriate vasoactive support, correction of pump failure, or definitive source control.

Extended case: a positive test with poor tolerance

3.15 Decide whether responsiveness justifies treatment

A 67-year-old woman remains in intensive care after surgery for perforated diverticulitis. She has chronic hypertension and mildly reduced LV function. After initial fluids, antibiotics, and source control, her MAP is 67 mm Hg on a stable norepinephrine infusion. She is more interactive, capillary refill has improved, and lactate has fallen from 5.8 to 3.2 mmol/L over four hours. Urine output remains 0.25 mL/kg/h. She has accumulated a positive fluid balance, and oxygen requirement has risen from 2 to 6 L/min. Lung ultrasound shows increasing bilateral B-lines. Focused echo excludes a large pericardial effusion and obvious acute RV dilation. During a carefully performed PLR, reliably measured stroke volume rises by 13%, with little pressure change, and returns to baseline afterward. The resident recommends repeated 500-mL boluses because the test is positive and urine output is low.

The surgeon must decide which findings indicate continuing hypoperfusion, which indicate treatment intolerance, and whether a preload response predicts a worthwhile clinical benefit. The source-control procedure was technically difficult; a favorable global trend cannot by itself guarantee that the abdominal source has been completely controlled.

Model judgment

Interpret the test correctly: The result supports preload responsiveness, not a mandatory need for more fluid. The small pressure change does not invalidate a genuine flow response. At the same time, worsening respiratory findings make repeated large boluses potentially harmful.

Interpret oliguria in context: Check catheter patency, kidney-function trend, baseline disease, drug exposure, venous congestion, and the duration of oliguria. Urine output may lag recovery or reflect established injury. Do not use it alone to override improving perfusion and emerging intolerance.

Select the next action: Reassess respiratory deterioration, cardiac function, residual hypoperfusion, and source control with the critical-care team. Additional fluid requires a specific expected benefit that outweighs the current risk; the available information does not justify an automatic series of boluses. Continue appropriate pressure support and mechanism-directed treatment.

Define follow-up: Track oxygenation, examination, support needs, renal trajectory, and lactate. If recovery stalls or the abdomen becomes concerning, pursue the unresolved source rather than attributing everything to insufficient preload. Fluid removal is a later individualized decision after considering stability and ongoing resuscitation needs.

> **Clinical pearl**
> The most difficult decision may be to decline more fluid in a patient who would increase stroke volume with it.

Extended case: the monitor suggests fluid, the patient does not

3.16 Identify an invalid inference

A 58-year-old man is ventilated after emergency small-bowel resection. He develops atrial fibrillation with a ventricular rate varying between 110 and 145/min. Ventilation uses a low tidal volume, and he intermittently triggers breaths. MAP is 61 mm Hg on increasing norepinephrine. His hands are cool, capillary refill is prolonged, and lactate has risen from 2.4 to 4.6 mmol/L. A pulse-contour monitor displays PPV of 18% and falling cardiac output. The nurse notes that the arterial waveform became unusually narrow after the patient was turned; the pressure bag and tubing have not been checked. There is increasing abdominal distension and little drain output. Hemoglobin is 9.9 g/dL, only modestly lower than earlier. The resident proposes giving a liter of crystalloid because PPV is above the usual threshold. A second clinician proposes cardioversion as the sole explanation for the deterioration.

The surgeon must distinguish a real clinical emergency from uncertain device outputs, decide whether arrhythmia is the primary cause or a consequence, and prevent a monitoring debate from delaying investigation of a surgical complication. Multiple explanations remain possible and can coexist.

Model judgment

Validate the measurement: Check the arterial system, leveling, zero, damping, connections, and a corroborating pressure promptly. PPV is unreliable here because of arrhythmia and spontaneous effort, with additional limitations from low tidal volume. The displayed percentage cannot justify a liter of fluid.

Do not dismiss the clinical shock: Worsening examination, increasing support, and rising lactate remain concerning even if the device is flawed. Obtain targeted echo and reliable flow assessment when feasible, while evaluating blood loss, abdominal pathology, and other causes. A small hemoglobin change and low drain output do not exclude concealed hemorrhage.

Assess the arrhythmia urgently: Determine whether it is causing hemodynamic instability and treat according to the appropriate emergency rhythm pathway. Correct contributory triggers. Rhythm treatment and surgical assessment proceed together; neither should become a reason to ignore the other.

Choose a safe next test: A suitable PLR with reliable flow measurement may help if positioning is safe, but marked intra-abdominal hypertension can reduce its reliability. If deterioration suggests a time-critical surgical source, prioritize definitive evaluation and control over repeated functional tests.

Clinical pearl
An invalid dynamic index cannot establish fluid need. A faulty monitor also cannot explain away a deteriorating patient.

Section 3: final review and sources

3.17 A decision table for the next intervention

Pattern	Judgment
Pressure improves; perfusion does not.	Reassess flow, oxygen content, regional ischemia, and the unresolved cause; avoid treating pressure as sufficient.
Flow rises after preload challenge; congestion worsens.	Responsiveness is present but tolerance is limited. Reassess the net benefit before further fluid.
No flow response to a valid challenge.	Stop repeating the same empirical bolus; reconsider pump, vascular tone, obstruction, and ongoing source.
Lactate remains high; examination and support improve.	Continue surveillance and explain production/clearance; do not automatically chase normality with fluid.
Urine remains low after other markers improve.	Evaluate kidney injury, obstruction, congestion, and time course; do not prescribe fluid from urine output alone.
All measured global targets improve; focal findings worsen.	Investigate regional pathology or an unresolved surgical complication.
A monitor changes after movement or a procedure.	Check technical validity and procedural complications while assessing the patient.

Minimum documentation after a resuscitation decision

Record the suspected mechanism, unresolved alternatives, intervention and timing, expected benefit, measured response, adverse effects, and the next reassessment trigger. Include whether the definitive cause has been controlled. These entries make a fluid or support decision reviewable rather than merely numerical.

Sources and educational scope

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. Sabiston Textbook of Surgery. 22nd ed. Chapter 33. Printed pp. 235-237: oxygen delivery, cardiac filling assessment, and historical optimization; integrated with foundational passages cited in Section 1. Cases, calculations, and tables are original educational synthesis.

[2] Monnet X, Messina A, Greco M, et al. ESICM guidelines on circulatory shock and hemodynamic monitoring 2025. Intensive Care Med. 51:1971-2012. doi:10.1007/s00134-025-08137-z. Contemporary monitoring and functional-test guidance supplements the textbook.

[3] Prescott H, Antonelli M, Alhazzani W, et al. Surviving Sepsis Campaign: International guidelines for management of sepsis and septic shock 2026. doi:10.1097/CCM.0000000000007075. Official recommendations: sccm.org, guideline of 23 March 2026. Conditional recommendations on dynamic assessment, serial lactate, and capillary refill carry low-certainty evidence. Accessed 2 November 2026.

Next section: Hemorrhagic shock and priorities of hemorrhage control and resuscitation.

4. Hemorrhagic shock and hemostatic resuscitation

4.1 Resuscitation and hemorrhage control are simultaneous tasks

Hemorrhagic shock combines loss of circulating volume with loss of oxygen-carrying capacity. Ongoing bleeding can also produce early coagulation dysfunction, compounded by hypothermia, acidosis, dilution, and hypocalcemia. The surgeon must restore a viable circulation while interrupting the source of loss. Transfusion cannot compensate indefinitely for uncontrolled hemorrhage. [1]

The most consequential decision is often where definitive control should occur and how quickly it can be achieved. A patient who briefly improves after blood but then deteriorates has not demonstrated stability. Repeated temporary responses may reveal ongoing loss and an urgent need for operative, endovascular, or endoscopic treatment.

Immediate question	Practical implication
Is hemorrhage plausible and clinically important?	Use mechanism, operative history, examination, trajectory, and ongoing support needs; do not wait for a low hemoglobin.
Where is blood being lost?	Search external sites, chest, abdomen, pelvis, retroperitoneum, gastrointestinal tract, and operative field as appropriate.
Can the patient safely undergo further diagnostic imaging?	Judge current physiology, transport risk, time to imaging, and whether the result changes the route to control.
What resources must be mobilized now?	Senior surgeon, anesthesia, blood bank, operating room, interventional radiology or endoscopy, and critical care.
What is the plan if the current intervention fails?	Name the next control strategy, transfer threshold, and responsible team before deterioration.

4.2 Recognize the limitations of familiar signs

A normal initial hemoglobin, absence of tachycardia, or low drain output does not exclude serious hemorrhage. A negative focused abdominal ultrasound does not exclude retroperitoneal, pelvic, or early intraperitoneal bleeding. Use serial perfusion findings and the speed of deterioration rather than a single threshold. [1]

Clinical pearl
The purpose of early resuscitation is to preserve life while obtaining control. A temporary rise in pressure must not reset the urgency of definitive hemostasis.

Control first: select the route without avoidable delay

4.3 Match the intervention to the source and physiology

Setting	Useful control measures	Judgment point
External / extremity bleeding	Direct pressure, wound packing, and a tourniquet when indicated.	Control immediately; record tourniquet timing and arrange definitive care.
Suspected unstable pelvic injury	Correctly placed pelvic stabilization; coordinated packing and/or embolization pathway.	A negative abdominal ultrasound does not make pelvic bleeding unlikely.
Intra-abdominal or postoperative bleeding	Operative exploration or targeted endovascular control when appropriate.	Persistent instability may make an imaging detour unsafe.
GI bleeding	Urgent endoscopic, endovascular, or surgical control according to the source and response.	Airway risk, anatomy, available expertise, and failed prior treatment determine the sequence.
Major vascular injury	Immediate vascular control or repair strategy; selected endovascular options.	Mobilize expertise early; do not rely on transfusion to bridge an indefinite delay.

4.4 Damage-control surgery is a physiological decision

When severe physiological derangement makes a prolonged definitive procedure dangerous, an abbreviated operation can control major hemorrhage and contamination, permit temporary closure, and allow staged reconstruction after physiological recovery. This is a deliberate plan with defined reassessment and reoperation, not simply an unfinished operation.

Ongoing bleeding, worsening acidosis, hypothermia, coagulopathy, escalating transfusion, injury burden, and the time required for reconstruction all matter. No isolated pH or temperature should replace judgment. Conversely, an unnecessarily prolonged operation can convert recoverable shock into irreversible deterioration.

4.5 Selected adjuncts are not substitutes for control

Aortic balloon occlusion is a specialist bridge for selected patients in an established pathway. It requires appropriate anatomy, expertise, rapid access to definitive control, and attention to ischemic and access complications. It is not a routine step for every hypotensive trauma patient or a reason to delay an available operation.

Clinical pearl
Ask which action shortens time to durable hemostasis. The best diagnostic route is the one that remains safe and changes the control strategy.

Activate and run a major hemorrhage protocol

4.6 Activate from anticipated need, not a retrospective definition

Activate the local major hemorrhage or massive transfusion protocol when the clinical trajectory suggests life-threatening ongoing bleeding and rapid blood-product need. Do not wait until a historical threshold such as ten red-cell units in 24 hours has already been reached. Scores can support recognition, but should not overrule a convincing clinical emergency.

Communicate the patient identifier, location, suspected source, urgency, products already given, estimated ongoing need, and the responsible clinician. Obtain suitable samples promptly, but do not delay emergency-release blood when required. Blood bank compatibility and emergency-release policies govern product selection and transition to group-specific blood.

4.7 Begin organized replacement, then make it targeted

Initial balanced component packs or an established whole-blood pathway reduce the delay between red-cell delivery and hemostatic support. A common component pack approximating a 1:1:1 plasma:platelets:red-cell strategy contains about 6 plasma units, one adult platelet dose (roughly 6 single-donor-equivalent units), and 6 red-cell units. The ratio does not mean one adult apheresis platelet dose per red-cell unit. Use the blood bank's defined component equivalents and pack composition.

Low-titer group O whole blood is an option in validated programs with compatibility, inventory, and population-specific safeguards. Do not assume all whole-blood products are interchangeable or universally superior. Additional targeted products may still be required. Record whole blood and components together to avoid duplicated or unrecognized replacement.

As current laboratory or viscoelastic results become available, transition from empiric packs to goal-directed treatment of the demonstrated deficits. Continue to account for ongoing bleeding and the delay between sampling and the current physiological state. Protocol organization is valuable; fixed-ratio transfusion should not continue automatically after control.

Evidence interpretation

PROPPR compared early 1:1:1 with 1:1:2 plasma:platelets:red-cell strategies in severe trauma. Overall mortality differences at the primary time points were not statistically significant; 1:1:1 achieved more hemostasis and fewer deaths from exsanguination. This supports early balanced delivery without establishing that one exact ratio is optimal for every surgical bleed. [4]

Clinical pearl
Tell the blood bank when the emergency begins, when requirements change, and when it ends. An unclosed protocol can cause avoidable transfusion and product waste.

Repair the physiology that prevents clot formation

4.8 The lethal triad and the calcium problem

Hypothermia impairs platelet and coagulation function. Acidosis reflects severe physiological stress and contributes to hemostatic dysfunction. Coagulopathy perpetuates bleeding. Hypocalcemia adds a further threat to clotting and cardiovascular function, particularly during rapid citrate-containing transfusion. Address these processes while obtaining mechanical control. [1,2]

Problem	Assessment	Treatment principle
Hypothermia	Measure core temperature; review exposure and cold infusions.	Prevent heat loss, use active warming and approved blood/fluid warmers, and control shock.
Acidosis	Blood gas, lactate/base deficit trend, ventilation, and perfusion.	Restore circulation and control the cause; routine bicarbonate is not hemorrhage control.
Hypocalcemia	Serial ionized calcium during rapid transfusion.	Replace calcium under the local protocol and recheck; total calcium can mislead.
Coagulation dysfunction	Bleeding pattern, platelet count, fibrinogen, PT/aPTT, and/or viscoelastic testing.	Treat the deficit while considering temperature, dilution, anticoagulants, and the surgical source.

Maintain ionized calcium in the local normal range rather than waiting for profound derangement. Calcium chloride and calcium gluconate contain different amounts of elemental calcium and are not interchangeable gram for gram; select the preparation, access, and dose through the transfusion protocol. Extravasation risk and repeat measurement matter. [2]

4.9 A normal laboratory result does not exclude surgical bleeding

Conventional coagulation tests are performed under laboratory conditions and do not reproduce every in-vivo effect of hypothermia or platelet dysfunction. A “normal INR” cannot certify adequate hemostasis. Diffuse oozing should prompt physiological correction and coagulation assessment, while a discrete vessel still requires direct control.

Do not give liters of fluid simply to warm the patient. Warming transfusions prevents further cooling; active warming and hemorrhage control remain essential. Equally, correcting a laboratory pH without restoring perfusion can create false reassurance.

Clinical pearl
Surgical and coagulation bleeding frequently coexist. Treating one does not establish that the other has been resolved.

Goal-directed coagulation support

4.10 Match the product to the defect

Finding in ongoing major bleeding	Likely response	Caution
Low fibrinogen / weak fibrin-based clot	Cryoprecipitate or fibrinogen concentrate according to availability and protocol.	Plasma alone may require excessive volume to correct a substantial fibrinogen deficit.
Low platelet count or relevant clot-strength deficit	Platelet replacement when indicated.	Clot strength depends on fibrinogen as well as platelets; interpret the whole test.
Coagulation factor deficiency	Appropriate plasma or selected factor therapy under a validated algorithm.	PCC is not routine replacement for all traumatic coagulopathy.
Clinically relevant anticoagulant effect	Urgent agent-specific reversal with specialist/pharmacy support.	A normal INR does not exclude important direct oral anticoagulant activity.
Hyperfibrinolysis in the appropriate clinical setting	Antifibrinolytic treatment within the applicable pathway.	Interpret assay type, timing, prior treatment, and the cause of bleeding.

For actively bleeding trauma patients, the European guideline recommends fibrinogen replacement when the level is at or below 1.5 g/L or there is viscoelastic evidence of functional deficiency. Common platelet goals are above 50 x 10^9/L in ongoing bleeding and above 100 x 10^9/L with traumatic brain injury. These are context-specific guideposts, not instructions to transfuse every nonbleeding patient to those values. [2]

4.11 Use viscoelastic tests as an algorithm, not an isolated trace

TEG and ROTEM examine clot development and breakdown in whole blood. Prolonged initiation may suggest factor-related impairment; low fibrin-specific firmness supports a fibrinogen problem; low overall clot strength can reflect fibrinogen or platelets; excessive breakdown suggests fibrinolysis. Assays and activators differ, so numeric thresholds are not interchangeable. [1]

Use a locally validated interpretation and treatment pathway, including heparin effects and prior drugs when relevant. Send conventional tests in parallel when useful. A test obtained before several additional transfusions may not describe the current patient; time-stamp samples and repeat after major changes.

Clinical pearl
Empiric balanced replacement bridges the early information gap. Targeted support should narrow that gap, not become another reason to postpone operative hemostasis.

Pressure strategy and tranexamic acid

4.12 Avoid indiscriminate normalization before control

In selected adults with uncontrolled traumatic bleeding and no brain injury, restricted volume replacement with a temporarily lower pressure can limit over-resuscitation while definitive control is expedited. A systolic pressure around 80-90 mm Hg is a guideline reference for that specific context, not an acceptable target for every patient. [2]

Traumatic brain injury, spinal cord ischemia risk, chronic hypertension, myocardial ischemia, pregnancy, and other vulnerable states require a different balance. Do not transfer permissive-hypotension targets from selected trauma populations to all postoperative or GI hemorrhage. Treat worsening perfusion and individualize support. A vasopressor can be an adjunct in selected profound shock, but cannot replace blood, volume restoration when needed, or control.

4.13 TXA: indication and time are part of the prescription

For trauma patients bleeding or at risk of significant bleeding, give tranexamic acid as early as possible within 3 hours of injury through the trauma pathway; do not delay eligible treatment while waiting for a viscoelastic result. A widely used adult regimen is 1 g IV over 10 minutes followed by 1 g over 8 hours. The clock starts at injury, not hospital arrival. Routine initiation beyond that window is not supported. [1,2]

Do not extrapolate trauma benefit to every cause of hemorrhage. HALT-IT found no reduction in death due to bleeding with its high-dose TXA regimen in acute GI bleeding and identified increased venous thromboembolic events and seizures. Routine TXA for acute GI bleeding is therefore not justified by the trauma evidence. [3]

4.14 Stop resuscitation packs when the indication ends

After control and physiological improvement, stop automatic packs and reassess ongoing losses, hemoglobin, coagulation, temperature, calcium, respiratory status, and fluid burden. Restrictive red-cell thresholds used in stable patients should not be applied mechanically during exsanguination; conversely, an earlier massive bleed does not justify continued transfusion after the emergency resolves.

Monitor for transfusion reactions, hemolysis, electrolyte disturbances, pulmonary complications, and circulatory overload. New hypoxemia or hypotension during transfusion requires urgent assessment and the transfusion-reaction pathway rather than an assumption that more blood is always the answer.

Clinical pearl
The early phase is guided by life-threatening loss and anticipated need; the later phase requires explicit de-escalation and renewed attention to transfusion harm.

Extended case: a transient responder with pelvic bleeding

4.15 CT, angiography, or immediate control?

A 36-year-old man arrives 50 minutes after a high-speed collision. He is pale and restless, with a systolic pressure of 72 mm Hg and heart rate of 126/min. The pelvis is painful and clinically suspicious for instability; a binder is applied correctly. There is no major external hemorrhage, breath sounds are symmetric, and focused ultrasound shows no definite intraperitoneal fluid. Hemoglobin is 12.1 g/dL. After emergency blood, systolic pressure rises to 98 mm Hg and he becomes more coherent, but within minutes pressure falls again to 76 mm Hg. Lactate is 6.5 mmol/L, core temperature 34.7 C, ionized calcium 0.92 mmol/L, and fibrinogen 1.2 g/L. Blood products continue to be required. The CT scanner is available, but transport and scanning will separate him from the resuscitation area. Interventional radiology estimates a 35-minute mobilization time; an operating team can begin earlier.

A resident argues that a negative ultrasound and normal hemoglobin make major hemorrhage unlikely and that the brief pressure response makes CT safe. Another recommends waiting for angiography without a bridging plan because pelvic arterial bleeding may be treatable by embolization. The surgeon must integrate the likely source, physiological trajectory, and actual time to control.

Model judgment

Recognize ongoing life-threatening loss: The response is transient, not sustained stability. Pelvic or retroperitoneal hemorrhage remains plausible despite the ultrasound and initial hemoglobin. Continue the search for concurrent sources without allowing negative tests to close the differential.

Mobilize a parallel pathway: Activate major hemorrhage support and coordinate trauma surgery, anesthesia, blood bank, and interventional radiology. Maintain stabilization and plan rapid hemorrhage control using the institution's pelvic pathway, which may require packing and/or embolization. Waiting without a viable bridge is unsafe.

Choose from real timing and capability: Decide whether CT can be tolerated and will change the immediate strategy. Recurrent profound instability and ongoing transfusion argue against a routine imaging detour. The fastest appropriate available control may take priority; angiography may still be necessary afterward.

Correct coexisting hemostatic failure: Warm actively, provide balanced support, correct measured hypocalcemia, and replace fibrinogen through the protocol. The patient is within the trauma TXA window. These steps accompany control rather than replacing it. Reassess perfusion and requirements after each major intervention.

Clinical pearl
A negative test that does not examine the bleeding compartment cannot override a convincing trajectory of ongoing hemorrhage.

Extended case: diffuse oozing after postoperative hemorrhage

4.16 Decide when to abbreviate and when to stop transfusing

A 71-year-old woman returns to the operating room for hemorrhage after complex hepatobiliary surgery. She has coronary disease and chronic kidney impairment. During re-exploration, a bleeding arterial branch is controlled, but raw surfaces continue to ooze. She has already received multiple red-cell units, less plasma than planned, and substantial crystalloid before protocol activation. Core temperature is 34.1 C, pH 7.19, ionized calcium 0.86 mmol/L, fibrinogen 0.9 g/L, and platelet count 64 x 10^9/L. Oxygen requirements are increasing. A viscoelastic sample taken before the latest products shows reduced clot strength with a low fibrin-specific contribution. One clinician requests PCC because the INR is elevated. Another proposes continuing the same fixed-ratio packs until the INR becomes normal. The surgeon estimates that definitive reconstruction would add another two hours.

The team must separate remaining surgical hemorrhage from impaired clot formation, decide whether the patient can tolerate definitive reconstruction, and define how to avoid continuing a protocol after its benefit ends. Her cardiac and renal disease increase the cost of unnecessary volume, but they do not eliminate the immediate need for appropriate hemostatic support.

Model judgment

Reassess the operative field: Confirm that focal sources are controlled and that no additional concealed source remains. Diffuse oozing with these physiological abnormalities strongly supports a coagulopathic contribution, but does not certify complete surgical hemostasis.

Treat demonstrated deficits: Correct hypothermia and ionized calcium and provide fibrinogen replacement with appropriate targeted component support. Interpret platelet need alongside ongoing bleeding and the full assay. Repeat current tests after the recent products; the earlier trace is not a complete description of the present circulation.

Avoid an isolated INR prescription: PCC is not a universal solution for dilutional or surgical coagulopathy and does not replace fibrinogen or platelets. Review anticoagulant exposure and use factor concentrates only for a defined indication under the relevant pathway. Further plasma should account for factor deficiency and volume tolerance.

Consider staged surgery: Persistent derangement and a prolonged reconstruction may favor abbreviated control and a planned return after recovery, if anatomically feasible. Once hemorrhage is controlled, explicitly deactivate automatic packs, continue targeted reassessment, and monitor respiratory and circulatory complications.

Clinical pearl
The decision to abbreviate surgery and the decision to de-escalate transfusion both require an explicit plan for reassessment, rather than a single laboratory endpoint.

Section 4: final review and sources

4.17 The surgeon's major hemorrhage checklist

Decision	Required reasoning
Activate	Is rapid ongoing blood loss anticipated? Activate before a retrospective unit threshold is reached.
Control	Name the probable source, fastest appropriate route to hemostasis, and fallback if it fails.
Resuscitate	Provide organized blood-based support; limit unnecessary crystalloid and monitor perfusion.
Protect clotting	Track temperature, calcium, fibrinogen, platelets, and factor function; treat demonstrated deficits.
Individualize pressure	Balance ongoing hemorrhage against brain, spinal, coronary, and other organ-perfusion requirements.
Use TXA selectively	Apply the correct indication, injury-based timing, regimen, and pathway; do not generalize to GI bleeding.
Reassess	A transient response or reassuring hemoglobin cannot establish bleeding control.
De-escalate	Stop automatic packs when the indication ends; reassess residual deficits and transfusion harm.

Sources and educational scope

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. Sabiston Textbook of Surgery. 22nd ed. Chapter 33. Printed pp. 228-234, with coagulation, hypothermia, and TXA passages. Tables, decision sequence, and cases are original educational applications. Historical claims are interpreted in the context of contemporary evidence.

[2] Rossaint R, Afshari A, Bouillon B, et al. The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition. Crit Care. 2023;27:80. doi:10.1186/s13054-023-04327-7. https://pmc.ncbi.nlm.nih.gov/articles/PMC9977110/. This open-access guideline is licensed CC BY 4.0 (https://creativecommons.org/licenses/by/4.0/); recommendations are adapted and condensed here, with original clinical interpretation. Trauma recommendations are identified as such and are not universal postoperative prescriptions.

[3] HALT-IT Trial Collaborators. Effects of a high-dose 24-h infusion of tranexamic acid on death and thromboembolic events in patients with acute gastrointestinal bleeding. Lancet. 2020;395:1927-1936. doi:10.1016/S0140-6736(20)30848-5.

[4] Holcomb JB, Tilley BC, Baraniuk S, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA. 2015;313:471-482. doi:10.1001/jama.2015.12.

Practice boundary: Exact product packs, emergency-release compatibility, calcium dosing, viscoelastic thresholds, and anticoagulant reversal require the local major hemorrhage pathway and appropriate expertise. Sources verified 2 November 2026.

5. Fluid selection, vasoactive support, and avoiding excess

5.1 Prescribe for a mechanism and a phase

Fluid and vasopressor decisions should follow the physiological problem: inadequate effective circulating volume, loss of vascular tone, impaired pump function, or obstruction. These therapies can be needed together. The indication must be renewed as the patient moves from emergency rescue to stabilization and recovery; an early lifesaving intervention can become harmful when continued automatically. [1]

Prescription category	Purpose	Required review
Resuscitation	Rapidly improve a threatened circulation.	Mechanism, response, tolerance, and unresolved source.
Replacement	Replace measured or reasonably estimated abnormal losses.	Volume, electrolyte composition, timing, and change in losses.
Maintenance	Meet ongoing water and electrolyte needs when oral/enteral intake is inadequate.	All intake sources, organ function, glucose, electrolytes, and daily need.
Drug and nutrition carrier	Deliver medications, feeds, and flushes.	Count this volume; it is not physiologically invisible.
De-escalation	Stop unnecessary input and consider removal of accumulated fluid.	Resolution of acute shock, congestion, organ function, and ability to tolerate removal.

5.2 State the intended effect before choosing the bag

A resuscitation prescription should identify what is expected to improve, how soon it will be assessed, and what will stop further administration. A replacement order for gastric losses answers a different question from a bolus for circulatory failure. A maintenance order should not continue unchanged simply because it was appropriate before surgery.

Choose the fluid composition from the indication and the patient's sodium, chloride, potassium, acid-base state, brain injury status, and ongoing losses. Choose the volume from likely benefit and tolerance. A physiologically attractive solution can still cause harm in an excessive amount.

Clinical pearl
Every liter needs an indication. The right fluid, at the wrong dose or in the wrong phase, remains the wrong prescription.

Crystalloid selection and important exceptions

5.3 Compare solutions by composition, not by name alone

Fluid	Useful role	Main limitations
Balanced isotonic crystalloid	Common resuscitation choice in many surgical and septic settings.	Products differ in sodium, potassium, buffers, and effective tonicity; not a universal choice for brain injury.
0.9% saline	Useful for selected chloride-depletion states and when a brain-injury pathway favors it.	Large volumes can produce hyperchloremic metabolic acidosis; reassess chloride and renal function.
Dextrose-containing water / hypotonic fluid	Selected maintenance or free-water correction after assessment.	Not suitable as the principal rapid intravascular resuscitation fluid; glucose and sodium effects require monitoring.
Hypertonic saline	Specific neurocritical or severe symptomatic sodium indications under protocol.	Not routine replacement for ordinary shock resuscitation; sodium correction limits and close monitoring matter.
Blood products	Restore oxygen-carrying capacity and hemostatic support during major hemorrhage.	Compatibility, reactions, calcium, temperature, and targeted component needs must be managed.

Balanced crystalloids reduce chloride exposure compared with saline, but do not eliminate edema or fluid overload. A small potassium content is not by itself proof that a balanced solution will worsen potassium in every patient with kidney disease; assess the actual electrolyte disturbance, acid-base state, formulation, and clinical need. Severe hyperkalemia requires its own urgent treatment pathway.

5.4 Do not misread a treatment-induced acidosis

After large saline exposure, a rising chloride and normal-anion-gap acidosis may coexist with improving perfusion. Do not interpret every persistent base deficit as evidence that another bolus is needed. Review lactate, ventilation, kidney function, chloride, and the bedside trajectory together.

Lactated solutions do not automatically imply worsening lactic acidosis. Buffer metabolism and disease context matter, and lactate values should be interpreted clinically. Avoid drawing a diagnostic blood sample from a line contaminated by an ongoing infusion.

Clinical pearl
A deteriorating acid-base measurement can reflect the composition of treatment as well as ongoing shock. Resolve the mechanism before prescribing more of the same fluid.

Albumin, synthetic colloids, and volume-sparing claims

5.5 A colloid is not a remedy for an undefined problem

Albumin can expand the circulating compartment, but capillary permeability, distribution, concentration, and clinical setting affect its behavior. Hypoalbuminemia alone does not prove that albumin infusion will improve outcomes or resolve edema. Consider a specific indication, anticipated benefit, cost, and tolerance rather than treating a serum number. [1]

Clinical situation	Reasonable interpretation
Routine septic resuscitation	Crystalloids remain the starting point; albumin is not automatically required.
Large prior crystalloid exposure or cirrhosis	Selected patients may merit supplemental albumin; use a defined indication and reassess.
Traumatic brain injury	Avoid supplemental albumin for resuscitation; use the neurotrauma fluid pathway.
Interstitial edema with low serum albumin	Look for ongoing inflammation, venous congestion, cardiac/renal disease, and excess input; replacing albumin is not a complete plan.
A proposal to use starch to save volume	Do not infer safety from a smaller infused volume; synthetic starches carry important harm concerns.

5.6 Separate physiological effects from demonstrated benefit

Current sepsis guidance favors crystalloids alone for routine resuscitation, with selected exceptions for albumin, and advises against starches and gelatins. Its balanced-crystalloid preference has a brain-injury exception. These are population-specific recommendations; they do not establish one product for every surgical condition. [2]

Hyperoncotic albumin and dilute albumin are not interchangeable prescriptions. A proposed volume-sparing strategy must still show a plausible clinical benefit and acceptable pulmonary and circulatory effects. Concentrated preparations do not remove the need to monitor perfusion and fluid balance.

Before adding a colloid

Document the remaining perfusion problem, why crystalloid or another treatment is insufficient, whether responsiveness and tolerance have been assessed, and what will be measured afterward. Do not use albumin to postpone control of bleeding, infection, obstruction, or cardiac dysfunction.

Clinical pearl
A lower infusion volume is a surrogate effect. It does not by itself establish better survival, kidney outcomes, or freedom from congestion.

Vasopressors: restore pressure while treating the cause

5.7 Do not make fluid completion a prerequisite in profound vasodilation

Severe hypotension from loss of vascular tone may require early vasoactive support while appropriate volume replacement and source treatment proceed. Conversely, a vasopressor cannot replace missing blood or correct a mechanical obstruction. Reassess the shock phenotype whenever requirements rise.

Agent / class	Typical role	Judgment and monitoring
Norepinephrine	Usual initial vasopressor for septic vasodilatory shock.	Titrate to an individualized pressure/perfusion goal; watch rhythm, ischemia, and flow.
Vasopressin	Adjunct in selected septic shock with escalating norepinephrine.	Not a substitute for source control; follow the ICU protocol and review ischemic risk.
Epinephrine	Selected additional support; separate first-line role in anaphylaxis.	Can increase heart rate and lactate; interpret trends in the treatment context.
Predominantly alpha-agonist support	Selected circumstances requiring specialist assessment.	A higher pressure can coexist with lower stroke volume; not a routine answer to all shock.
Inotropic support	Selected low-output states with cardiac dysfunction and persistent hypoperfusion.	May cause tachyarrhythmia or hypotension; identify the lesion and assess response.

In septic shock, current guidance retains norepinephrine first and suggests vasopressin as needs escalate, with epinephrine for selected persistent hypotension. Cardiac dysfunction and rhythm can alter selection. Exact concentrations and titration require a monitored local protocol. [2]

5.8 Start safely without creating an access-related delay

Where trained staff and an established protocol are available, short-term peripheral vasopressor administration can bridge urgent treatment rather than waiting for central access. Use a suitable well-functioning vein and inspect the site frequently; arrange central access when ongoing treatment warrants it. Pain, blanching, swelling, or suspected extravasation demands immediate action through the local pathway. [2]

Clinical pearl
Escalating pressor requirement is a clinical alarm. Verify delivery, pressure measurement, volume status, pump function, obstruction, and the unresolved source before simply increasing the next dose.

Preventing excess and beginning de-escalation

5.9 Count total exposure and distinguish congestion from depletion

Record boluses, maintenance, replacement, blood products, nutrition, drug carriers, and flushes. Review cumulative balance with serial weight and examination, recognizing inaccuracies and unmeasured losses. Peripheral edema indicates interstitial accumulation but does not establish adequate forward flow. Venous congestion can itself impair organ function.

Warning sign	Reassessment before more fluid
Rising oxygen requirement / new lung findings	Examine for edema, permeability injury, cardiac dysfunction, aspiration, infection, and other causes.
Oliguria despite repeated boluses	Check obstruction, established AKI, venous congestion, intra-abdominal pressure, and actual flow response.
Increasing abdominal distension and airway pressures	Consider intra-abdominal hypertension; assess bladder pressure when indicated and seek urgent surgical review.
Increasing CVP without useful flow improvement	Stop interpreting pressure accumulation as effective resuscitation; review RV function and obstruction.
Lactate persists while perfusion improves	Consider production/clearance and treatment effects; do not chase normalization with fluid alone.

5.10 Use a phased removal decision

First stop unneeded input and adjust ongoing replacement. After acute resuscitation has ended, consider active removal when accumulated fluid is clinically harmful and perfusion can tolerate it. Individualize diuretic use, monitoring, and the desired trajectory; edema alone does not justify aggressive removal during worsening shock.

Review pressure and support needs, examination, oxygenation, urine/electrolyte trends, and the response to each step. Reduce or stop removal if perfusion deteriorates. Renal replacement requires its own indications and expert assessment; do not initiate routine extracorporeal removal solely to make the balance chart negative. ESICM supports de-escalation after the acute phase while recognizing uncertainty and subgroup limits. [3]

Clinical pearl
Prevention starts before overload occurs: stop ineffective boluses, remove duplicate fluid orders, and revisit every carrier and maintenance infusion.

Extended case: low urine output after apparent recovery

5.11 Avoid reversing recovery with another automatic bolus

A 69-year-old man is on the second day after emergency colectomy for feculent peritonitis. Initial shock required blood-pressure support and several liters of crystalloid. His vasopressor requirement is now falling, interaction and capillary refill have improved, and lactate has decreased from 5.4 to 2.3 mmol/L. Urine output remains low and creatinine has risen. The chart shows a cumulative positive balance of 7 L, but does not count several medication carriers. Weight has increased, oxygen requirements are rising, and there is peripheral edema. The abdomen is more distended and ventilator pressures are higher. A technically acceptable preload challenge produces no convincing increase in forward flow. A junior clinician proposes another liter because creatinine is rising; another recommends immediate aggressive diuresis because the balance is positive. The surgeon has not yet reassessed abdominal pressure or excluded a residual source.

The next decision must account for established kidney injury, possible congestion, abdominal pathology, and the possibility that improved global markers do not describe every organ. Neither the creatinine nor the balance chart independently identifies the correct treatment.

Model judgment

Decline an unsupported bolus: The absent useful flow response and emerging intolerance argue against routine additional volume. Check catheter patency, kidney injury contributors, medications, and the accuracy of intake/output data.

Investigate the abdomen and lungs: Distension, higher airway pressures, and oliguria raise concern for intra-abdominal hypertension as one possibility. Measure and evaluate when indicated; also assess residual infection, obstruction, pulmonary complications, and cardiac function. Do not assume fluid accumulation is the sole explanation.

Stop preventable input: Reconcile maintenance, replacement, carriers, flushes, and nutrition. Establish which abnormal losses still require replacement and in what composition. A lower urine output does not create an indefinite replacement order.

Plan removal only after assessing readiness: If the acute shock phase has resolved and congestion is harmful, a monitored individualized diuretic strategy may be appropriate. Ongoing instability or an urgent abdominal problem may change the sequence. Reassess perfusion and electrolytes rather than pursuing a fixed negative balance at any cost.

Clinical pearl
An organ can deteriorate from congestion, established injury, or a surgical complication despite adequate arterial pressure. More fluid is only one hypothesis, and must earn its place.

Extended case: pressor escalation with persistent lactate

5.12 Interpret treatment effects without overlooking failure

A 60-year-old woman undergoes drainage of an intra-abdominal collection after an anastomotic leak. She remains hypotensive after initial resuscitation. Norepinephrine is started through a monitored peripheral catheter while central access is arranged. Subsequent echo shows reduced LV function without a large effusion or clear RV pressure overload. Her extremities are cool and forward-flow assessment suggests low output as well as vasodilation. Epinephrine is added by the critical-care team. Pressure and measured flow improve, but heart rate rises to 135/min and lactate increases from 3.5 to 5.0 mmol/L. She is already edematous and oxygenation is worsening. The abdominal drain has little output, and the adequacy of source control remains uncertain. A resident calls the lactate rise proof of persistent volume depletion and proposes serial fluid boluses. Another attributes it entirely to epinephrine and recommends no further evaluation.

The surgeon must integrate drug effects, cardiac physiology, source-control uncertainty, and fluid tolerance. Both explanations contain a plausible observation but make an unsupported leap: lactate is neither a direct volume test nor a reliable certificate that the remaining problem is pharmacological.

Model judgment

Reassess mechanism and delivery: Confirm pressure measurement and infusion function, repeat perfusion assessment, review echo/flow findings and rhythm, and inspect the peripheral site until the infusion is transferred appropriately. A higher pressure cannot alone establish recovery.

Interpret lactate with the whole trajectory: Epinephrine can increase lactate through adrenergic effects, but that possibility does not exclude ongoing hypoperfusion or failed source control. Compare examination, flow, acid-base status, organ function, and the timing of the drug change.

Avoid automatic fluid: Edema and worsening oxygenation demand assessment of tolerance. Give more volume only for a defined likely benefit supported by an appropriate evaluation; do not use lactate alone as the indication.

Revisit definitive treatment and support: Evaluate whether the leak and collection are adequately controlled, and whether intervention is required. Coordinate vasoactive/inotropic adjustment with critical care, balancing tachycardia, cardiac function, pressure, and perfusion. Define the expected response and trigger for escalation.

Clinical pearl
A plausible drug explanation should refine the assessment, not end it. The unresolved surgical source retains priority even when the monitor improves.

Section 5: final review and sources

5.13 The next prescription should answer these questions

Decision	Practical test
Choose fluid	What deficit am I treating, and does this composition fit the patient?
Choose volume	What benefit is likely, how will I measure it, and what harm would stop me?
Start a pressor	Is inadequate tone/pressure part of the problem, and must support begin while volume assessment continues?
Escalate support	Have delivery failure, bleeding, obstruction, pump dysfunction, and failed source control been reconsidered?
Stop fluid	Has the expected benefit disappeared or intolerance emerged?
Remove fluid	Has acute resuscitation ended, is accumulation harmful, and can perfusion tolerate removal?
Review the next shift	Are replacement losses, maintenance, carriers, nutrition, and all support orders still indicated?

Distinctions to retain

Responsiveness is not need. Edema is not proof of adequate flow. Hypotension is not proof of volume depletion. Low albumin is not a complete indication for albumin. A positive balance is not an automatic indication for dialysis. A better pressure is not proof of source control.

Sources and scope

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. Sabiston Textbook of Surgery. 22nd ed. Chapter 33. The physiological framework is integrated with preceding sections. Tables and cases are original educational applications; contemporary recommendations are identified separately below.

[2] Prescott H, Antonelli M, Alhazzani W, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2026. doi:10.1007/s00134-026-08361-1; companion Critical Care Medicine doi:10.1097/CCM.0000000000007075. Official SCCM guideline, 23 March 2026. Recommendations summarized selectively here are population-specific; certainty varies by intervention.

[3] ESICM clinical practice guideline on fluid therapy in adult critically ill patients. Part 3: fluid removal at de-escalation phase. 2025. doi:10.1007/s00134-025-08058-x. Official society summary: https://www.esicm.org/new-esicm-guideline-fluid-therapy-part-3/. De-escalation and removal recommendations apply after the acute resuscitation phase.

Current sources checked 2 November 2026. Exact infusion concentrations, dose titration, peripheral vasopressor safeguards, and removal protocols require the monitored local pathway. This section develops judgment rather than providing a stand-alone drug-dosing manual.

6. Body water and maintenance / replacement prescriptions

6.1 Start with compartments, not an infusion rate

Total body water varies with age, lean mass, and adiposity. A common teaching model for a lean 70-kg adult uses total water of approximately 60% of body weight: 42 L, with about 28 L intracellular and 14 L extracellular. The extracellular compartment comprises roughly 10.5 L interstitial fluid and 3.5 L plasma. These are illustrative estimates, not measured targets for resuscitation. Older adults and people with greater adiposity generally have a lower water fraction. [1]

Compartment	Dominant physiological features	Clinical implication
Intracellular	Most body water; potassium is the major cation.	Changes in effective extracellular tonicity move water across cell membranes.
Interstitial	Extracellular water outside the vascular space.	Can accumulate substantially during inflammation or venous congestion.
Plasma	The intravascular extracellular compartment.	Its effective filling and forward flow cannot be inferred from total body water alone.
Transcellular	Specialized fluids such as CSF and gastrointestinal secretions.	Abnormal external losses may require specific replacement.

Plasma volume is not the same as whole-blood volume: red cells contribute to blood volume but their water is intracellular. Do not mix a blood-volume percentage with a plasma-water fraction in a compartment calculation.

6.2 Volume and concentration answer different questions

Serum sodium reflects the relationship of exchangeable sodium and potassium to body water; it does not measure total-body sodium or effective circulating volume. A patient losing sodium-rich intestinal fluid may remain normonatremic while becoming markedly volume depleted. Another can be edematous with poor effective arterial filling.

Clinical pearl
Normal serum sodium does not establish normal extracellular volume. Edema does not establish adequate circulating flow.

Osmolality, tonicity, and movement of infused water

6.3 Distinguish all particles from effective osmoles

Osmolality counts dissolved particles per kilogram of water. Tonicity describes the effect of relatively nonpenetrating solutes on cell water. Sodium salts are major determinants of extracellular tonicity; glucose becomes important when markedly elevated. Urea contributes to measured osmolality but usually contributes little to sustained transcellular tonicity because it crosses most cell membranes. Rapid urea shifts, as during dialysis, require separate consideration.

For conventional units, estimated serum osmolality is approximately 2 x sodium + glucose/18 + BUN/2.8, with sodium in mmol/L and glucose and BUN in mg/dL. Estimated effective osmolality omits the BUN term. These are teaching approximations; use the local laboratory method and context. BUN is not interchangeable with a urea concentration in another unit.

6.4 Predict distribution without treating the model as a guarantee

Infusion	Simplified distribution after equilibration	Prescribing implication
Isotonic sodium-containing crystalloid	Primarily expands extracellular water; much distributes outside plasma.	A liter infused is not a liter of sustained plasma expansion.
Dextrose 5% in water	After glucose uptake/metabolism, behaves mainly as electrolyte-free water distributed through total body water.	Not the principal fluid for rapid volume resuscitation; can lower sodium.
Hypertonic sodium solution	Raises extracellular tonicity and recruits intracellular water.	Use only for a defined indication with sodium and clinical monitoring.
Albumin	Distribution depends on concentration, permeability, and the clinical state.	No universal fixed crystalloid-to-albumin equivalence guarantees a clinical effect.

In the simplified 42-L model, 1 L of retained electrolyte-free water would distribute approximately two-thirds intracellularly and one-third extracellularly, with only about 80 mL in plasma. Real distribution changes with renal excretion, capillary permeability, time, and illness. Do not use a compartment ratio to prescribe an automatic resuscitation volume.

Clinical pearl
A bag can be approximately iso-osmolar before infusion yet become effectively hypotonic after its glucose is metabolized.

Maintenance: estimate a need, then subtract existing intake

6.5 Maintenance applies to a stable patient without major abnormal losses

Maintenance supplies expected water and electrolyte needs when oral or enteral intake is inadequate. It does not automatically correct a prior deficit, replace ongoing abnormal losses, or treat shock. Prefer oral or enteral provision when feasible, and stop IV maintenance when it is no longer needed.

For many stable adults, an initial water estimate is 25-30 mL/kg/day, with approximately 1 mmol/kg/day each of sodium, potassium, and chloride and 50-100 g/day glucose to limit starvation ketosis. Older or frail adults and patients with cardiac or renal impairment often need a lower starting water estimate, such as 20-25 mL/kg/day. These are planning references, not fixed requirements. Severe comorbidity, diabetes, brain injury, burns, pregnancy, and critical illness require specific pathways. [2]

6.6 Compare daily estimation with the historical hourly rule

Sabiston presents the 4-2-1 hourly rule: 4 mL/kg/h for the first 10 kg, 2 for the next 10 kg, and 1 for each kilogram thereafter. Above 20 kg it gives weight + 40 mL/h. In adults this can exceed a contemporary daily maintenance estimate; familiarity does not make it an appropriate default. [1]

70-kg teaching example	Calculated water
25-30 mL/kg/day	1750-2100 mL/day, approximately 73-88 mL/h.
20-25 mL/kg/day	1400-1750 mL/day, approximately 58-73 mL/h.
4-2-1 hourly calculation	110 mL/h = 2640 mL/day, approximately 37.7 mL/kg/day.

For obesity, choose an appropriate ideal-weight approach rather than multiplying a large actual weight by a maintenance factor. Severe obesity warrants individualized review. Count oral intake, feeds, blood, drug carriers, and flushes before deciding how much additional IV water remains necessary.

Clinical pearl
The maintenance calculation estimates total provision, not an extra infusion to add on top of all other sources.

Build and audit a 24-hour prescription

6.7 A worked accounting example

A stable 70-kg adult has no abnormal gastrointestinal losses, no significant sodium disturbance, and adequate kidney function. The clinician selects an initial total water plan of 1750 mL/day. Oral intake is expected to provide 600 mL, medication carriers 400 mL, and flushes 150 mL. The remaining IV maintenance water budget is 1750 - 600 - 400 - 150 = 600 mL/day, or 25 mL/h if delivered evenly. This arithmetic does not determine the correct bag composition by itself.

Suppose the existing sources deliver 55 mmol sodium, 15 mmol potassium, and 10 g glucose. Against illustrative planning amounts of 70 mmol sodium, 70 mmol potassium, and 50 g glucose, the residual amounts are 15 mmol sodium, 55 mmol potassium, and 40 g glucose. It may be impossible to meet all residual needs in one standard bag at a safe concentration. Reassess enteral provision and use the approved pharmacy/prescribing pathway rather than improvising concentrated mixtures.

6.8 Audit actual contents, not the label “maintenance”

Example	What it actually supplies	Interpretation
2 L of 0.9% saline	308 mmol sodium and 308 mmol chloride; no potassium or glucose.	May be appropriate replacement for a particular deficit, but is not a neutral routine daily prescription.
2 L of 5% dextrose in water	100 g glucose; no sodium or potassium.	Supplies water and limited energy, but cannot meet all electrolyte needs.
1 L fluid with 20 mmol/L potassium	20 mmol potassium in that liter.	Concentration and daily delivered amount are different quantities.

For monovalent sodium and potassium, mmol and mEq are numerically equal. This equivalence does not apply to every ion. Distinguish sodium mass from sodium chloride mass. Prescribe standardized premixed potassium-containing fluids or approved preparations; never give IV potassium as a push or add it casually at the bedside.

Assess renal function, current potassium, relevant drugs, acid-base state, and urine trajectory before potassium provision. Oliguric kidney injury can make a routine weight-based potassium amount unsafe. Glucose in maintenance fluid is not nutritional support and does not supply protein or adequate calories.

Clinical pearl
The complete prescription has a volume, composition, rate, duration, and monitoring plan. A rate alone is incomplete.

Replacement: match abnormal losses and changing deficits

6.9 Keep replacement separate from ordinary maintenance

Identify the source, measure output, review its likely composition, and replace over a defined interval. Reassess frequently when output changes. An established deficit requires a separate correction plan based on perfusion, duration, electrolyte abnormalities, and tolerance. If the patient is in shock, begin the resuscitation pathway rather than waiting for the next scheduled replacement interval.

Loss	Likely disturbance	Replacement judgment
Vomiting / nasogastric suction	Water, chloride, hydrogen, and variable potassium losses; chloride-depletion alkalosis may develop.	Chloride-containing isotonic replacement may be appropriate; replace potassium only with safe monitoring.
High-output small-bowel stoma / proximal fistula	Substantial sodium and water loss; potassium and magnesium deficits may coexist.	Replace sodium-containing losses, monitor magnesium and renal function, and address the cause of high output.
Diarrhea / distal intestinal loss	Water and electrolytes including bicarbonate and potassium; metabolic acidosis may occur.	Use clinical and biochemical assessment; do not copy a gastric-loss prescription without review.
Biliary or pancreatic drainage	Sodium-rich fluid; bicarbonate loss varies by secretion.	Quantify loss and consider composition-specific assessment and acid-base trends.
Blood loss	Volume, red-cell mass, and hemostatic components.	Use hemorrhage control and appropriate blood-based support, not ordinary drain replacement alone.

A temporary mL-for-mL order for measured gastrointestinal losses can be useful when linked to frequent review, but is not a universal rule for every drain. Do not replace urine routinely mL for mL: it already contributes to ordinary water output and can reflect treatment, diuresis, or disease. Polyuria requires cause-specific assessment.

6.10 Avoid the double count

Do not add normal urine output and ordinary insensible loss to a maintenance estimate that already includes them. Fever, rapid breathing, open wounds, and unusual environmental conditions can change insensible loss, but automatic large supplements are unreliable. “Third-space loss” should not become an unmeasured recurring order; evaluate perfusion, redistribution, and tolerance.

Clinical pearl
Replace an identified loss and treat its cause. Do not replace a vague label indefinitely.

Extended case: gastric loss with edema and alkalosis

6.11 Separate deficit, ongoing loss, and accumulated excess

A 76-year-old woman weighing 56 kg remains nil by mouth after surgery for bowel obstruction. She has heart failure with preserved ejection fraction and moderately impaired kidney function. Nasogastric output has reached 1200 mL over the preceding day and increased during the last six hours. She receives a standing “maintenance” order of saline at 100 mL/h plus medication carriers and intermittent potassium replacement. Weight has risen by 3 kg, her ankles are edematous, and oxygen requirement has increased. Sodium is 139 mmol/L, potassium 3.0 mmol/L, chloride 91 mmol/L, bicarbonate 34 mmol/L, and creatinine is rising. She is alert, with borderline pressure but no clear deterioration in peripheral perfusion. The fluid chart does not distinguish earlier resuscitation, current maintenance, and gastric replacement. One resident proposes stopping all fluid because she is edematous; another proposes increasing saline to replace the entire previous day's output immediately.

The surgeon must determine whether current effective filling is inadequate, whether congestion is contributing to kidney dysfunction, and which part of the prescription addresses the evolving gastric loss. Neither edema nor a normal sodium resolves these questions.

Model judgment

Reconstruct the account: Review all intake, weight, current losses, timing of prior replacement, and the reason for continued suction. The standing infusion supplies 2400 mL/day before carriers, a substantial amount for this patient. Do not assume it was all maintenance or all replacement.

Identify the biochemical pattern: Gastric loss with chloride depletion and hypokalemia plausibly contributes to metabolic alkalosis. Evaluate magnesium and acid-base context. A normal sodium does not rule out sodium/chloride depletion or establish normal circulating volume.

Assess perfusion and tolerance: Examine for pulmonary and venous congestion and use targeted cardiac/flow assessment when needed. Urgent instability would require a different sequence. Avoid both indiscriminate withdrawal of necessary replacement and rapid catch-up infusion based solely on a historical output.

Rewrite the prescription: Individualize a smaller maintenance component after accounting for carriers; prescribe measured ongoing gastric replacement with an appropriate chloride-containing solution and safe potassium provision. Renal impairment and respiratory findings constrain the rate and demand early reassessment. Correct the underlying obstruction/ileus issue and reassess suction necessity.

Clinical pearl
An edematous patient can still have a specific electrolyte deficit. Correcting that deficit requires a composition-aware plan, not an unlimited volume prescription.

Extended case: high-output stoma with a normal sodium

6.12 Do not equate oral water intake with effective replacement

A 48-year-old man is readmitted ten days after ileostomy formation. He reports drinking about 3 L of plain water daily because he was advised to stay hydrated. Stoma output is watery and approximately 2.5 L/day. He is thirsty, dizzy on standing, and 4 kg below discharge weight. Resting pressure is 108/68 mm Hg and heart rate 98/min; on standing he becomes symptomatic. Serum sodium is 137 mmol/L, creatinine has increased substantially, potassium is 3.4 mmol/L, and magnesium is low. He has no edema. A resident concludes that normal sodium and the reported intake exclude important depletion, and prescribes routine maintenance alone. Another proposes unrestricted additional plain water. The surgical team has not yet assessed stoma anatomy, medications, infection, or partial obstruction. The patient has no convincing features of circulatory collapse, but his losses and biochemical trajectory demand prompt attention.

The task is to distinguish a sodium-concentration result from sodium and water balance, identify the ongoing loss, and build a plan that does more than repeat an average maintenance formula.

Model judgment

Recognize extracellular depletion: Weight loss, postural symptoms, high output, and kidney deterioration support clinically important sodium/water loss despite normal serum sodium. Assess perfusion and determine whether initial isotonic volume restoration is required before a longer replacement plan.

Measure and investigate: Confirm output and intake, examine the abdomen and stoma, review anatomy and drugs, and evaluate reversible causes of high output. Infection and obstruction can require specific treatment. Check renal and electrolyte trends, including magnesium, rather than focusing on sodium alone.

Replace the right material: Routine maintenance does not cover liters of additional sodium-rich intestinal loss. Use appropriate IV replacement initially when indicated, with safe potassium/magnesium correction. Do not treat unrestricted plain water as equivalent to sodium-containing replacement; excessive hypotonic drinking can worsen net sodium loss in some high-output stoma settings.

Plan a sustainable transition: Coordinate a stoma/nutrition pathway with an appropriate glucose-saline oral rehydration strategy, output control, and individualized intake advice once clinically suitable. Specify review timing, weight/output targets, biochemical monitoring, and escalation triggers; do not discharge with a generic instruction to drink more.

Clinical pearl
Normal sodium is a concentration finding. High-output intestinal losses require a balance and composition assessment.

Section 6: final review and prescribing worksheet

6.13 A practical sequence for the next 24 hours

Step	Decision to document
1. Assess urgency	Shock, severe symptoms, or a dangerous electrolyte abnormality requires an immediate pathway.
2. Estimate ordinary needs	Use a suitable weight and clinical context; reduce for limited tolerance and adapt for specialized conditions.
3. Count existing provision	Oral/enteral intake, carriers, flushes, nutrition, blood, and other infusions all count.
4. Identify separate problems	Existing deficit or excess, measured abnormal losses, and redistribution require explicit plans.
5. Select composition	Water, sodium/chloride, potassium, glucose, and other deficits must be considered independently.
6. Specify delivery	Use available standardized preparations, safe concentrations, rate, duration, and review triggers.
7. Reassess and stop	Review examination, balance, losses, renal function and electrolytes; stop IV provision once unnecessary.

A useful conceptual account is: additional IV provision = individualized maintenance need minus other usable intake, plus separately planned replacement/correction, adjusted for excess and redistribution. This is a reasoning framework rather than a formula that makes every component additive without clinical judgment.

Monitoring that changes the next order

Review fluid status, balance, and renal/electrolyte trends at least daily in a stable patient receiving IV fluid; reassess sooner with major losses, evolving kidney injury, significant sodium/potassium abnormalities, or treatment intolerance. Track weight when feasible and clinically useful. Specify who will review results and how the order changes when oral intake or losses change.

Sources and scope

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. Sabiston Textbook of Surgery. 22nd ed. Chapter 33, printed pp. 255-256 (supplied PDF pp. 595-596): body water and maintenance fluids. The simplified compartment model and calculations are original teaching applications. Historical rules are compared with current prescribing guidance rather than treated as automatic adult orders.

[2] NICE. Intravenous fluid therapy in adults in hospital. CG174, recommendations 1.4-1.5 and general assessment/monitoring principles. https://www.nice.org.uk/guidance/cg174/chapter/recommendations. Published 2013, updated 2017; current recommendations checked 2 November 2026. General adult maintenance estimates are summarized selectively; specialized disease pathways may differ.

Cases, tables, and worksheet are original. Electrolyte correction limits, high-output stoma management, and disease-specific prescriptions require their dedicated pathways. The formulas are educational and do not replace reassessment.

7. Sodium, potassium, calcium, magnesium, and phosphate

7.1 Determine urgency before completing classification

Electrolyte disorders can cause neurological injury, arrhythmia, weakness, ileus, and respiratory failure. They can also be markers of a continuing surgical problem. Begin with symptoms, ECG when indicated, rate of change, kidney function, treatment exposures, and the reliability of the result. A number outside the reference range is not automatically an emergency; a dangerous clinical pattern must not wait for a complete etiologic workup. [1]

Threat	First clinical priority
Seizure or marked neurological deterioration with hyponatremia	Stabilize and treat suspected hyponatremic encephalopathy through a monitored hypertonic-saline pathway.
Severe hyperkalemia or compatible ECG abnormalities	Protect the heart when indicated, shift potassium, arrange elimination, and monitor for rebound.
Symptomatic hypocalcemia / arrhythmia	Measure ionized calcium where appropriate; urgent monitored calcium treatment and cause assessment.
Severe hypokalemia or hypomagnesemia with arrhythmia	Monitored replacement, medication review, and assessment of associated deficiencies.
Severe hypophosphatemia with organ dysfunction	Recognize possible respiratory/cardiac effects and refeeding; replace safely with renal and calcium monitoring.

7.2 Validate without delaying necessary treatment

Hemolysis, sampling from an infusing line, delayed processing, and assay method can mislead. Repeat a suspicious sample promptly. In a high-risk patient with convincing severe hyperkalemia or ECG changes, repeat testing accompanies treatment rather than postponing it. Check units: calcium, magnesium, and phosphate are often reported in either mmol/L or mg/dL, and the values are not numerically interchangeable.

Interpret extracellular concentration separately from total-body stores. Insulin, beta-adrenergic activity, acid-base shifts, cell injury, and refeeding can change a measured concentration without a proportional external gain or loss. Conversely, significant depletion may be present before the serum value falls.

Clinical pearl
Treat the immediate threat, verify the measurement, and identify the mechanism in parallel. Normalizing a value does not establish that the cause has ended.

Hyponatremia: establish tonicity and mechanism

7.3 A sodium below 135 mmol/L requires context

First distinguish hypotonic hyponatremia from hyperglycemia-related translocational hyponatremia and pseudohyponatremia associated with some indirect assays in severe hyperlipidemia or hyperproteinemia. Measured osmolality, glucose, and the laboratory method can resolve a misleading sodium result. Corrected-sodium estimates in hyperglycemia are approximate and should not replace serial assessment during treatment.

In hypotonic hyponatremia, obtain urine osmolality and urine sodium when feasible before major treatment changes, without delaying emergency care. A dilute urine suggests suppressed antidiuresis; an inappropriately concentrated urine indicates that water excretion is limited. Interpret urine sodium with recent saline, diuretics, kidney disease, and the clinical examination.

Pattern	Causes to consider	Treatment implication
Reduced effective circulating volume	GI loss, diuretics, or other sodium loss.	Restore appropriate volume and treat the cause; watch for rapid water diuresis afterward.
Apparently euvolemic hypotonic state	SIAD, medication effects, cortisol deficiency, and selected endocrine disorders.	Do not diagnose SIAD until important mimics are excluded; restriction is not treatment for all hyponatremia.
Edema / low effective arterial filling	Heart failure, cirrhosis, and kidney disease.	Treat the underlying state and individualize water/sodium management.
Postoperative water retention	Pain, nausea, stress, drugs, and hypotonic intake.	Review every water source and the persistence of antidiuretic stimuli.

Primary adrenal insufficiency may combine hyponatremia with hyperkalemia; secondary adrenal insufficiency can produce hyponatremia without hyperkalemia. Hypotension or suspected adrenal crisis requires urgent assessment and treatment. Do not label a postoperative concentrated urine as SIAD on that finding alone.

Clinical pearl
The same sodium concentration can require very different therapy. Tonicity, symptoms, time course, and effective circulating volume determine the next action.

Hyponatremia: rescue safely, then control correction

7.4 The emergency goal is symptom relief, not immediate normalization

Severe symptoms such as seizure or profound neurological impairment plausibly caused by hyponatremia require monitored hypertonic saline and senior/critical-care involvement. A commonly used European approach is 150 mL of 3% saline over 20 minutes, followed by clinical and sodium reassessment, with protocol-directed repetition when needed. Other validated pathways use different bolus volumes; do not combine regimens. An initial rise of approximately 4-6 mmol/L is usually sufficient for urgent neurological benefit. [2]

7.5 Distinguish an intended rise from a maximum limit

For chronic or uncertain-duration hyponatremia, avoid excessive correction because of osmotic demyelination risk. European guidance limits the rise to 10 mmol/L in the first 24 hours and 8 mmol/L in each subsequent 24 hours. High-risk patients, including those with very low sodium, malnutrition, alcohol use disorder, hypokalemia, or advanced liver disease, require a more conservative ceiling, commonly no more than 8 mmol/L in any 24 hours, with a lower intended rise. These are ceilings, not goals to reach. [2]

Measure sodium after rescue boluses and frequently during active correction, often every 2-4 hours in unstable correction, with close urine-output surveillance. Potassium replacement can contribute to the sodium rise. A sudden large dilute urine output after volume restoration or withdrawal of an antidiuretic stimulus can accelerate correction even after saline has stopped.

7.6 Have an overcorrection plan before starting

If the sodium rise is approaching or exceeding the safe trajectory, stop the correction-driving intervention and obtain urgent specialist review. Desmopressin and carefully controlled electrolyte-free water may be used to halt aquaresis and relower sodium under an expert protocol. Do not wait for delayed neurological signs. Repeat sodium and account for all fluid and potassium sources.

A sodium-deficit or predicted-infusion formula cannot anticipate an abrupt change in renal water excretion. Use calculations to estimate a starting plan, then let measured response govern the next prescription. Fluid restriction alone is inadequate rescue treatment for severe hyponatremic encephalopathy.

Clinical pearl
The greatest danger can emerge after apparently successful treatment, when antidiuresis resolves and the patient begins excreting large volumes of dilute urine.

Hypernatremia: restore circulation and replace water

7.7 Separate a free-water problem from immediate shock

Hypernatremia, usually sodium above 145 mmol/L, reflects hypertonicity. Review access to water, thirst, neurological status, gastrointestinal and skin losses, sodium administration, osmotic diuresis, and impaired urinary concentration. A hospitalized patient who cannot drink depends on the team to recognize and replace water loss. [1]

If circulation is severely compromised, restore perfusion with appropriate isotonic resuscitation first while planning subsequent water correction. Isotonic support alone does not reliably correct the free-water deficit. Once perfusion is addressed, choose enteral water when safe or an appropriate IV hypotonic strategy and treat the ongoing cause.

Worked estimate, with limitations

Estimated water deficit = total body water x [(measured sodium / desired sodium) - 1]. With estimated total body water of 30 L and sodium 160 mmol/L, the deficit relative to 140 is 30 x (160/140 - 1) = 4.29 L. This is a model estimate, not an order to infuse 4.29 L promptly. It excludes ongoing losses, other intake, renal changes, and errors in estimated body water.

7.8 Correct according to duration and measured response

For chronic or uncertain-duration hypernatremia, a conventional cautious plan limits reduction to roughly 0.5 mmol/L/h and about 10 mmol/L/day, with serial sodium monitoring and adjustment. Adult outcome evidence is less definitive than the evidence linking excessive hyponatremia correction to harm; avoid both uncontrolled rapid correction and prolonged undertreatment. Clearly acute sodium loading or symptomatic acute disease requires specialist management. [3]

Polyuria with inappropriately dilute urine raises concern for arginine vasopressin deficiency or resistance, but hyperglycemia, diuretics, and postobstructive or recovering kidney states also matter. Central deficiency may require desmopressin; water replacement and antidiuretic treatment must be coordinated because urine losses can stop abruptly. Do not simply copy a previous hourly replacement order after that change.

Clinical pearl
The water-deficit equation describes a starting estimate. The current urine output and serial sodium describe what is actually happening.

Potassium: protect, shift, eliminate, and prevent rebound

7.9 Hyperkalemia is a physiological emergency, not just an ECG diagnosis

Severe hyperkalemia, commonly potassium at or above 6.5 mmol/L, warrants urgent monitored assessment and treatment. ECG abnormalities or rapid progression can make lower values dangerous. A normal ECG does not establish safety. Review kidney failure, drugs, potassium provision, insulin deficiency, acidosis, cell injury, and sample artifact. [4]

Task	Mechanism and limitation
Protect the myocardium	IV calcium when indicated by ECG toxicity or the emergency pathway; it does not lower potassium.
Shift potassium intracellularly	Insulin with glucose and selected adjunct beta-agonist treatment; the effect is temporary.
Remove potassium	Stop inappropriate input; select elimination measures and urgently assess dialysis when severe/refractory or renal failure makes medical clearance inadequate.
Check for harm and rebound	Repeat potassium, ECG/clinical assessment, and glucose surveillance after insulin; hypoglycemia can be delayed.

A common adult emergency regimen uses 10 units soluble insulin with 25 g glucose IV, with additional glucose and prolonged monitoring according to baseline glucose and protocol. Insulin dosing and hypoglycemia prevention require particular care in kidney impairment. Calcium chloride and gluconate differ in elemental calcium and access risks; use the correct formulation-specific emergency dose. Do not assume a hypocalcemia regimen and a hyperkalemia regimen are interchangeable. [4]

7.10 Hypokalemia: identify ongoing loss and magnesium deficiency

Potassium below 3.5 mmol/L may follow GI or renal losses, alkalosis, insulin, beta-agonists, or refeeding. Marked reduction, especially below 2.5 mmol/L, symptoms, or ECG abnormalities requires urgent monitored management. Assess rhythm, drugs, magnesium, renal function, and the continuing source. A serum result does not precisely quantify the total-body deficit.

Use oral/enteral replacement when suitable; IV therapy is reserved for appropriate urgency or inability to use the gut. Use premixed preparations and a pump, never IV push. Standard ward rates are often limited to 10 mmol/h; higher rates require a specified monitored protocol, appropriate access, and frequent reassessment. Correct magnesium deficiency and account for potassium in phosphate replacement and all other sources.

Clinical pearl
A transient potassium reduction after insulin does not equal elimination. Plan the next measurement and definitive clearance before the first treatment wears off.

Calcium: use the biologically relevant measurement

7.11 Total calcium and ionized calcium are not interchangeable

Albumin affects total calcium, while pH changes alter binding and can change ionized calcium without a similar total-calcium change. Alkalosis lowers the ionized fraction. Albumin-correction equations can be inaccurate in critical illness and kidney disease; use directly measured ionized calcium when the clinical decision depends on biologically active calcium. Handle the sample correctly. [1]

Disorder	Surgical context / findings	Judgment
Hypocalcemia	After neck surgery, massive transfusion, pancreatitis, magnesium deficiency, or renal disease; tetany, seizure, QT prolongation.	Symptomatic disease needs urgent monitored calcium and cause treatment; do not wait for a corrected-total formula.
Hypercalcemia	Hyperparathyroidism, malignancy, drugs, or other causes; dehydration, altered cognition, renal dysfunction.	Assess severity and volume tolerance; treat the cause and use specialist-directed calcium-lowering therapy when indicated.

7.12 Acute hypocalcemia and definitive endocrine treatment

For severe symptomatic hypocalcemia, a commonly used adult approach is 10-20 mL of 10% calcium gluconate diluted and given over about 10 minutes with ECG monitoring, followed by reassessment and further treatment under protocol. Persistent need may require an infusion. Formulation, access, renal function, and compatibility determine safe delivery. Calcium chloride carries greater extravasation risk and is not volume-equivalent. [5]

After thyroid or parathyroid surgery, evaluate PTH, magnesium, phosphate, kidney function, and vitamin D context. Oral calcium and active vitamin D may be required for persistent hypoparathyroidism; acute IV rescue does not substitute for a continuing plan. Low magnesium can impair PTH secretion and action.

In severe hypercalcemia, restore appropriate volume when depleted but avoid a fixed large saline load in cardiac or renal disease. Calcitonin can provide a rapid temporary effect in selected emergencies; antiresorptive or other therapy depends on cause and renal considerations. Routine loop diuresis solely to lower calcium is not a substitute for appropriate assessment and treatment.

Clinical pearl
A low total calcium with hypoalbuminemia may not require IV calcium; a symptomatic low ionized calcium with alkalosis may require urgent treatment despite a less dramatic total result.

Magnesium and phosphate: linked deficiencies and organ effects

7.13 Magnesium can determine whether other correction succeeds

Hypomagnesemia may follow GI loss, renal wasting, drugs, or malnutrition. It can cause arrhythmia and neuromuscular symptoms and can sustain hypokalemia and hypocalcemia. A normal serum value does not exclude depleted stores, but replacement should still account for kidney function and the clinical indication. Oral therapy suits selected stable cases; severe symptoms or arrhythmia require monitored IV treatment.

Hypermagnesemia is especially important in renal impairment with magnesium-containing laxatives, antacids, or infusions. Weakness, depressed reflexes, hypotension, bradycardia, and respiratory depression suggest toxicity. Stop the source, support ventilation/circulation, use calcium for significant toxicity under protocol, and consider dialysis when severe or clearance is inadequate. Dose magnesium in mmol or the clearly specified salt preparation; grams of magnesium sulfate are not grams of elemental magnesium.

7.14 Phosphate participates in energy and muscle function

Pattern	Mechanism / consequence	Clinical response
Low phosphate	Poor intake, refeeding, insulin, respiratory alkalosis, or renal loss; severe depletion can cause weakness, respiratory failure, rhabdomyolysis, or hemolysis.	Choose oral or IV replacement from severity, symptoms, renal function, and gut access.
High phosphate	Kidney failure, cell breakdown, phosphate administration, or other causes.	Stop excess input; assess calcium and cause; consider renal replacement when the overall emergency requires it.

Phosphate below about 0.32 mmol/L (1 mg/dL) is commonly considered severe. IV replacement requires a validated regimen with reassessment of phosphate, calcium, potassium, magnesium, and renal function. Sodium-phosphate and potassium-phosphate preparations provide different accompanying cation loads. Excessive or rapid replacement can cause hypocalcemia and other complications; do not mix phosphate and calcium in an unverified line or solution.

Clinical pearl
Refractory hypokalemia or hypocalcemia should prompt a magnesium check. New respiratory weakness in a malnourished patient should prompt a phosphate and refeeding assessment.

Extended case: successful rescue, dangerous sodium trajectory

7.15 Recognize overcorrection before neurological injury appears

A 64-year-old man is admitted before surgery for an obstructing upper-GI lesion. He has lost substantial weight, has eaten little for two weeks, and takes a thiazide. Sodium is 112 mmol/L, potassium 2.7 mmol/L, and measured osmolality is low. He becomes confused and has a generalized seizure. The team provides monitored hypertonic-saline rescue, stops the thiazide, and begins appropriate electrolyte treatment. Neurological symptoms improve as sodium reaches 117 mmol/L. Volume depletion is also treated. Over the next several hours, urine output abruptly rises and urine becomes dilute. Sodium is 121 mmol/L eight hours after the initial value, although the hypertonic infusion has already stopped. Potassium replacement is continuing. A resident calls the improvement a success and recommends waiting until the next morning for another sodium result; another proposes continuing saline until sodium is normal so surgery can proceed.

The surgeon must distinguish rescue success from correction safety and decide whether the absence of current symptoms provides reassurance. The duration of hyponatremia is uncertain, and malnutrition and hypokalemia increase vulnerability to osmotic demyelination.

Model judgment

Identify the new emergency: The rise is already 9 mmol/L in eight hours, exceeding a conservative high-risk daily ceiling and continuing during aquaresis. Stopping hypertonic saline did not stop the correction mechanism. A normal examination now does not exclude delayed injury.

Escalate immediately: Seek endocrine/renal and critical-care support, stop avoidable correction-driving inputs, measure sodium frequently, and monitor urine closely. Use an expert desmopressin/electrolyte-free-water relowering strategy when indicated rather than passively observing continued rise.

Count coupled treatments: Potassium repletion can contribute to the sodium increase; it must be coordinated with the sodium plan. Continue necessary treatment through a controlled prescription rather than abandoning the hypokalemia problem.

Reconsider timing and cause: Review medications, losses, nutrition and refeeding risk, and the urgency of surgery. A nonurgent operation should not drive rapid normalization. If intervention is truly urgent, coordinate perioperative treatment and monitoring with the relevant teams.

Clinical pearl
Symptoms can improve while treatment risk increases. A sudden water diuresis changes the prescription even when no new saline is being given.

Extended case: refractory potassium and respiratory weakness

7.16 Treat the linked syndrome rather than three isolated numbers

A 57-year-old woman undergoes surgery after prolonged poor intake from an intestinal fistula. She has marked weight loss and low muscle mass. Feeding and dextrose-containing infusions are advanced rapidly after surgery. Within 36 hours she develops weakness, a new oxygen requirement, edema, and ventricular ectopy. Potassium is 2.6 mmol/L despite repeated replacement, magnesium 0.42 mmol/L, and phosphate 0.22 mmol/L; ionized calcium is mildly reduced. Creatinine is rising and urine output is falling. Her prefeeding phosphate had been within the laboratory reference range. A resident proposes only escalating potassium because ectopy is the most visible threat. Another assumes the normal baseline phosphate excludes nutritional depletion and attributes the weakness entirely to postoperative atelectasis. The medication record shows additional carbohydrate delivery that was not counted by the nutrition prescription.

The surgeon must assess an immediate rhythm and respiratory threat, identify the temporal relationship to calorie delivery, and coordinate replacement when declining renal clearance increases the risk of overshoot. Alternative postoperative causes remain possible and require assessment.

Model judgment

Recognize probable refeeding physiology: The timing, nutritional history, and combined fall in phosphate, magnesium, and potassium strongly support a refeeding-related syndrome. A normal initial serum phosphate did not establish adequate stores. Evaluate other contributors, including sepsis, respiratory alkalosis, medications, and losses. [6]

Stabilize and correct together: Use cardiac monitoring and assess ventilation and respiratory muscle weakness. Severe symptomatic phosphate and magnesium depletion require urgent protocol-based treatment alongside potassium. Magnesium deficiency may explain the refractory potassium; correcting one number alone is insufficient.

Modify the nutrient exposure: Obtain nutrition/critical-care input, review all carbohydrate sources, provide thiamine promptly, and reduce or pause calorie advancement according to severity and the specialist pathway. Do not continue full escalation unchanged while replacing recurrent deficits.

Control replacement risk: Account for potassium in phosphate preparations, kidney impairment, calcium changes, line compatibility, and repeat results. Reassess frequently rather than applying a standard deficit dose repeatedly. Review fluid accumulation and the underlying surgical disease at the same time.

Clinical pearl
The diagnosis is a trajectory linked to feeding and organ dysfunction, not a single low phosphate result. The treatment must coordinate calories, vitamins, fluid, and electrolytes.

Section 7: final review and source map

7.17 Cross-checks that prevent avoidable harm

Finding	Do not miss
Hyponatremia with a concentrated urine	Volume depletion, cortisol deficiency, drugs, and renal disease before diagnosing SIAD.
Rapid sodium rise after treatment	Water diuresis and potassium replacement; reassess the entire correction plan.
Hypernatremia with polyuria	Osmotic diuresis, AVP deficiency/resistance, and changing renal function.
Hyperkalemia improves after insulin	Rebound and delayed hypoglycemia; arrange elimination and repeated measurements.
Potassium or calcium resists correction	Magnesium deficiency and continuing losses.
Low total calcium in critical illness	Direct ionized measurement may be more useful than an albumin formula.
Weakness after feeding begins	Phosphate, magnesium, potassium, thiamine, and fluid shifts.
Electrolyte infusion in kidney injury	Reduced clearance, accompanying cation load, accumulation, and repeat-dose risk.

Sources and scope

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. Sabiston Textbook of Surgery. 22nd ed. Chapter 33, printed pp. 258-262 and electrolyte physiology. The clinical framework, tables, and cases are original synthesis; contemporary emergency guidance refines treatment safety.

[2] Spasovski G, et al. Clinical practice guideline on diagnosis and treatment of hyponatraemia. Eur J Endocrinol. 2014;170:G1-G47. doi:10.1530/EJE-13-1020. Society for Endocrinology emergency guidance on severe symptomatic hyponatraemia, 2016. Correction targets and limits differ by risk and protocol; high-risk ceilings must be conservative.

[3] Society for Endocrinology. Inpatient management of cranial diabetes insipidus. Endocr Connect. 2018;7:G8-G11. https://pmc.ncbi.nlm.nih.gov/articles/PMC6013691/. Hypernatremia correction requires clinical and biochemical reassessment.

[4] Resuscitation Council UK. Special circumstances guidelines, 2025: hyper/hypokalaemia. https://www.resus.org.uk/professional-library/2025-resuscitation-guidelines/special-circumstances-guidelines. UK Kidney Association hyperkalaemia guideline landing page, current download labelled updated July 2026; exact local implementation should be checked.

[5] Society for Endocrinology. Emergency management of acute hypocalcaemia in adult patients. Endocr Connect; emergency guidance and addendum. https://pmc.ncbi.nlm.nih.gov/articles/PMC8117371/. Formulation-specific IV dosing is essential.

[6] da Silva JSV, et al. ASPEN consensus recommendations for refeeding syndrome. Nutr Clin Pract. 2020;35:178-195. doi:10.1002/ncp.10474. NICE CG32: nutrition support and refeeding-risk management. Current sources checked 2 November 2026.

Adult educational guidance. Drug concentrations, infusion rates, monitoring frequency, correction ceilings, and dialysis decisions require the appropriate monitored local pathway. Laboratory reference ranges and units must be verified.

8. Acid-base disorders and clinical interpretation

8.1 Describe the process, not just the pH

Acidemia means blood pH below 7.35; alkalemia means pH above 7.45. Acidosis and alkalosis describe processes that move pH downward or upward. Opposing processes may coexist and produce a nearly normal pH despite serious disease. The surgeon must identify the processes, connect them to the clinical cause, and decide which intervention cannot wait. [1]

The Henderson-Hasselbalch relationship is pH = 6.1 + log[HCO3 / (0.03 x PaCO2)], with bicarbonate in mmol/L and PaCO2 in mm Hg. It describes the ratio of the metabolic component to dissolved carbon dioxide; it does not identify the cause of either abnormality.

Primary process	Direction of primary change	Expected adaptive direction
Metabolic acidosis	Bicarbonate falls.	PaCO2 falls through increased ventilation.
Metabolic alkalosis	Bicarbonate rises.	PaCO2 rises, within physiological constraints.
Respiratory acidosis	PaCO2 rises.	Bicarbonate rises; renal adaptation takes time.
Respiratory alkalosis	PaCO2 falls.	Bicarbonate falls; the magnitude depends on duration.

8.2 Obtain interpretable measurements

Use a blood gas with contemporaneous electrolytes, albumin, lactate, renal function, and glucose as indicated. Blood-gas bicarbonate is calculated from measured pH and CO2; chemistry total CO2 is measured by a different method and approximates bicarbonate. Investigate a major discrepancy rather than combining unrelated samples. Note time, oxygen, ventilation, hemodynamics, and recent therapy.

Venous gases can support assessment of pH and metabolic trends in selected settings, but venous CO2 is not interchangeable with arterial CO2 for precise compensation calculations, and venous oxygen tension does not measure arterial oxygenation. Obtain an arterial sample when the decision depends on arterial ventilation or oxygenation.

Clinical pearl
A normal pH is not a normal acid-base assessment. Calculate the gap and test compensation whenever the clinical picture or other values are abnormal.

A reproducible interpretation sequence

8.3 Six questions before prescribing a correction

Step	Question and action
1. Clinical urgency	Is there shock, respiratory failure, arrhythmia, severe electrolyte disturbance, or a toxic exposure? Stabilize while interpreting.
2. pH and direction	Acidemia, alkalemia, or near-normal pH? Which changes in CO2 and bicarbonate explain the direction?
3. Expected compensation	Does the observed second variable fall within the expected range? If not, identify an additional primary process.
4. Anion gap	Calculate from contemporaneous chemistry; account for albumin and laboratory reference range.
5. Additional metabolic process	When the gap is high, compare its rise with the bicarbonate fall, with appropriate limitations.
6. Cause and next action	Link the pattern to losses, fluids, drugs, ventilation, perfusion, kidney function, and the surgical source.

8.4 Compensation is an estimate, not a second diagnosis

An adaptive change usually moves pH toward normal and does not overshoot into the opposite disturbance in a simple disorder. Observed values outside an expected range suggest a mixed process, but timing, chronic disease, and measurement error must also be considered. Renal adaptation to respiratory disease is slower than immediate buffering; do not apply a chronic rule to a new event.

A low bicarbonate can represent metabolic acidosis or adaptation to respiratory alkalosis. A high bicarbonate can represent metabolic alkalosis or adaptation to chronic hypercapnia. Labeling the disorder from bicarbonate alone can reverse the clinical interpretation.

A useful bedside statement

“There is a primary ___ process, with appropriate / inappropriate compensation, an albumin-adjusted gap of ___, and evidence for an additional ___ process. The most likely causes are ___; the next priority is ___.”

Clinical pearl
The interpretation is complete only when it changes a clinical decision or specifies the next investigation.

Expected compensation: calculations and limits

8.5 Use consistent reference values and units

Primary disorder	Common bedside estimate
Metabolic acidosis	Expected PaCO2 = 1.5 x bicarbonate + 8, plus or minus 2 mm Hg (Winter's formula).
Metabolic alkalosis	Expected PaCO2 approximately 40 + 0.7 x (bicarbonate - 24), with a broad tolerance of about 5 mm Hg.
Respiratory acidosis, acute	Bicarbonate rises about 1 mmol/L per 10-mm Hg PaCO2 rise above 40.
Respiratory acidosis, chronic	Bicarbonate rises approximately 3.5-4 mmol/L per 10-mm Hg PaCO2 rise.
Respiratory alkalosis, acute	Bicarbonate falls about 2 mmol/L per 10-mm Hg PaCO2 fall below 40.
Respiratory alkalosis, chronic	Bicarbonate falls approximately 4-5 mmol/L per 10-mm Hg PaCO2 fall.

These physiological approximations vary among references and are most useful for identifying meaningful discordance. Use bicarbonate in mmol/L and arterial CO2 in mm Hg. Baseline chronic hypercapnia, kidney impairment, and evolving illness can complicate a simple rule.

Worked example: failing respiratory compensation

With bicarbonate 12 mmol/L, Winter's estimate is 1.5 x 12 + 8 = 26 mm Hg, with an expected range of 24-28. A measured PaCO2 of 40 is not reassuringly “normal”: it indicates an additional respiratory acidosis in this context. The patient may be tiring, sedated, or unable to ventilate adequately. A PaCO2 of 20 instead supports an additional respiratory alkalosis.

Why this matters during intubation

A patient with severe metabolic acidosis may depend on very high spontaneous minute ventilation. Apnea or inadequate postintubation ventilation can rapidly worsen acidemia. Involve experienced airway/critical-care staff, prepare circulation, minimize avoidable interruption of ventilation, and match support to physiological demand within lung-protective constraints. Treating the metabolic cause remains essential.

Clinical pearl
A CO2 within the laboratory reference range can be profoundly inappropriate for the patient's metabolic state.

Anion gap, albumin correction, and mixed metabolic disease

8.6 Calculate what the pH can conceal

Anion gap, excluding potassium = sodium - (chloride + bicarbonate). Use the laboratory reference interval and the same convention throughout. Low albumin reduces the expected gap. A common correction is: adjusted gap = measured gap + 2.5 x (4 - albumin in g/dL). This is an approximation, not a substitute for measuring lactate or ketones. [2]

Example: sodium 138, chloride 105, bicarbonate 18 gives a measured gap of 15 mmol/L. With albumin 2 g/dL, adjusted gap is 20. An apparently modest unadjusted gap can therefore conceal clinically important unmeasured anions.

8.7 Compare the gap rise with the bicarbonate fall

Using a teaching normal gap of 12 and bicarbonate of 24, delta gap = adjusted gap - 12, and bicarbonate fall = 24 - measured bicarbonate. If the gap rise substantially exceeds the bicarbonate fall, consider associated metabolic alkalosis or a high baseline bicarbonate. If the fall exceeds the gap rise, consider an additional normal-gap acidosis. The local normal gap, baseline values, timing, and renal excretion all affect interpretation.

A delta ratio is the gap rise divided by the bicarbonate fall. It becomes unreliable when the denominator is small or bicarbonate is normal/high. Do not force a ratio into a case where a direct comparison is clearer. The relationship is approximate; not every deviation proves another disease.

High-gap causes	Normal-gap causes
Lactate accumulation, ketoacids, retained acids in kidney failure, and selected toxins or drugs.	Bicarbonate loss from diarrhea or fistulas; chloride-rich fluid exposure; renal tubular or other renal acid-excretion problems.
Measure the suspected substance and assess production, clearance, and the clinical source.	Review losses, chloride exposure, potassium, renal function, and medication effects.

Clinical pearl
A normal or alkalemic pH does not exclude a high-gap acidosis. Hypoalbuminemia can make the measured gap deceptively small.

Metabolic disorders: choose cause-directed treatment

8.8 Metabolic acidosis does not automatically mean more fluid

In the surgical patient, consider ongoing hemorrhage or sepsis, ischemic bowel or limb, ketoacidosis including SGLT2-associated disease with modest glucose, kidney failure, bicarbonate-rich GI losses, and chloride exposure. Lactate is one contributor, not a complete explanation for every gap. Severe pain, focal findings, or an unexplained trajectory warrant regional ischemia assessment even when global pressure improves.

Treat hypoperfusion according to mechanism and tolerance, secure source control, treat ketoacidosis through its pathway, and replace appropriate losses. A persistent base deficit after saline can reflect hyperchloremia. Do not use it alone to prescribe further crystalloid or to declare failed resuscitation.

8.9 Bicarbonate: a selected intervention with physiological costs

Bicarbonate can generate CO2, add sodium and volume, lower ionized calcium, and produce overshoot alkalosis. Adequate ventilation and repeat gases/electrolytes matter. It does not repair hemorrhage, infected tissue, or bowel ischemia. Current sepsis guidance advises against bicarbonate solely to improve hemodynamics or reduce pressors in hypoperfusion-related lactic acidemia, but conditionally supports it in severe metabolic acidemia with significant AKI; certainty for that selected indication is very low. [3]

Severe refractory acidemia, dangerous hyperkalemia, volume overload, or selected intoxications may warrant renal-replacement assessment according to the full clinical context. Do not start dialysis solely because an isolated bicarbonate is below normal.

8.10 Metabolic alkalosis: ask what generated and sustains it

Gastric losses, diuretics, chloride/potassium depletion, mineralocorticoid effects, and correction of chronic hypercapnia are common clues. Urine chloride can help: a low value, often below 20 mmol/L, supports chloride depletion, while a higher value may reflect active diuretics or other renal mechanisms. Interpret timing and kidney function.

Treat the cause and replace chloride/potassium when indicated and tolerated. An edematous patient may not tolerate routine saline. Selected congested patients may need a different specialist strategy, such as adjustment of diuretics or acetazolamide with electrolyte surveillance. Do not use one alkalosis prescription for every volume state.

Clinical pearl
Correcting pH is a supporting action. Correcting the process that generated the disorder is the definitive task.

Respiratory disorders and treatment-related patterns

8.11 Respiratory acidosis: identify inadequate alveolar ventilation

Consider opioid or sedative effect, airway obstruction, neuromuscular weakness, fatigue, severe lung disease, inadequate ventilator support, and increased dead space. Assess airway, respiratory effort, oxygenation, consciousness, medications, and the gas trajectory. Oxygen therapy may correct saturation while CO2 continues to rise; it does not establish adequate ventilation.

Acute hypercapnia with neurological deterioration or inability to sustain ventilation requires prompt respiratory support assessment. Chronic hypercapnia has a different baseline bicarbonate and tolerance. Rapid correction can uncover posthypercapnic metabolic alkalosis; review the whole trajectory rather than targeting a single familiar CO2.

8.12 Respiratory alkalosis: do not assume anxiety

Pain and anxiety are possibilities, but so are sepsis, pulmonary embolism, hypoxemia, CNS disease, salicylates, and excessive ventilator support. A combined respiratory alkalosis and high-gap metabolic acidosis should raise concern for specific causes including salicylate toxicity. Evaluate the clinical setting rather than treating hyperventilation as the diagnosis.

Observed pattern	Potential implication
Low CO2 and low bicarbonate	Compensated metabolic acidosis, respiratory alkalosis with renal adaptation, or a mixed process.
High CO2 and high bicarbonate	Chronic respiratory acidosis, compensated metabolic alkalosis, or mixed disease.
Nearly normal pH with a high adjusted gap	A metabolic acidosis may be counterbalanced by alkalosis.
Alkalemia with paresthesias or tetany	Check ionized calcium and the cause of alkalosis; total calcium may mislead.

8.13 Reassess after the intervention

Repeat testing on a timeline suited to severity and treatment. Record ventilation changes, fluid type and dose, electrolytes, perfusion, and source interventions. A rising pH after intubation does not certify improved tissue perfusion; a falling lactate does not certify that respiratory compensation is adequate.

Clinical pearl
The blood gas is a physiological snapshot. Treatment can change the dominant process within minutes, while a second process persists.

Extended case: alkalemia concealing ischemic disease

8.14 Follow the gap despite an apparently favorable pH

A 72-year-old man is reviewed after surgery for adhesive bowel obstruction. He has ongoing nasogastric losses, worsening abdominal pain, and new confusion. He is tachypneic and has received opioids, saline, and potassium at different times. Blood pressure is 110/66 mm Hg on modest support, and urine output is falling. Arterial pH is 7.46, PaCO2 29 mm Hg, and calculated bicarbonate approximately 20 mmol/L; contemporaneous chemistry bicarbonate is 20. Sodium is 140, chloride 94, albumin 2 g/dL, and lactate 6.2 mmol/L. The abdomen is tender without dramatic rigidity. A resident calls this respiratory alkalosis from pain and recommends reassurance because the patient is not acidemic. Another proposes more saline until the base deficit resolves. The surgeon must decide whether the gas excludes clinically important hypoperfusion and whether the abdominal findings require urgent action.

Calculation and model judgment

Calculate the gap: Measured gap = 140 - (94 + 20) = 26. Albumin-adjusted gap = 26 + 5 = 31. A clinically important high-gap metabolic acidosis exists despite alkalemia. Lactate contributes, but does not automatically explain all unmeasured anions.

Test compensation: With bicarbonate 20, Winter's expected PaCO2 is 38 plus or minus 2. The actual 29 supports an additional respiratory alkalosis. Comparing the adjusted gap rise of 19 with the bicarbonate fall of only 4 also suggests a concurrent metabolic alkalosis, plausible with gastric chloride loss; baseline and sampling limitations still matter.

Act on the surgical problem: Worsening pain, lactate, confusion, and kidney trajectory warrant urgent evaluation of bowel viability, persistent obstruction, leak, or another source. A maintained pressure and absence of acidemia do not exclude regional ischemia. Choose imaging versus intervention from stability and the likely pathology, with immediate senior involvement.

Avoid treating one number: Do not attribute all hyperventilation to pain or prescribe repeated fluid solely for base deficit. Assess perfusion, flow, tolerance, gastric replacement needs, and the time to source control. Reevaluate after definitive treatment and each major support change.

Clinical pearl
The dangerous interpretation is “no acidosis because the pH is high.” Three primary processes can coexist in one plausible postoperative scenario.

Extended case: normal CO2 during severe metabolic acidosis

8.15 Recognize loss of compensation and plan the airway

A 61-year-old woman is in intensive care after source control for intra-abdominal sepsis. She has AKI and receives increasing circulatory support. Earlier she was alert and breathing rapidly, with pH 7.25, bicarbonate 10 mmol/L, and PaCO2 about 24 mm Hg. After analgesia and several hours of illness, she becomes drowsy and her respiratory rate falls. The new arterial gas shows pH 7.02, bicarbonate 10, and PaCO2 40. Chemistry confirms low bicarbonate, with a raised albumin-adjusted gap. Lactate remains elevated. Oxygen saturation is 97% with supplemental oxygen. A resident calls the new CO2 normal and suggests a bicarbonate bolus while waiting for morning review. Another prepares routine intubation without discussing her preceding minute ventilation. The surgeon must distinguish worsening metabolic disease from a superimposed ventilatory failure and anticipate the physiological consequences of airway intervention.

Calculation and model judgment

Identify the mixed disorder: Winter's expected PaCO2 is 1.5 x 10 + 8 = 23, with a range of 21-25. PaCO2 of 40 is markedly inappropriate and establishes an additional respiratory acidosis in this setting. The fall in respiratory effort and consciousness is concerning; preserved saturation does not establish adequate ventilation.

Escalate support urgently: Involve experienced airway and critical-care clinicians. Evaluate reversible medication effects, airway and respiratory muscle function, lung pathology, and ongoing shock. Do not wait for the next routine review.

Prepare for intubation physiology: If airway control is needed, plan hemodynamic support, minimize avoidable apnea, and provide sufficient postintubation ventilation within safe constraints. A standard ventilator prescription may fail to reproduce the necessary compensatory ventilation. Obtain an early reassessment gas.

Continue cause-directed treatment: Revisit source control, perfusion, renal failure, and other gap contributors. Bicarbonate may be considered for selected severe metabolic acidemia with significant AKI under the critical-care pathway; it is not a substitute for ventilation or definitive treatment. Review calcium, sodium/volume load, and the need for renal replacement based on the complete emergency.

Clinical pearl
A “normal” respiratory variable can reveal impending failure when the patient requires an abnormal value to compensate.

Section 8: final review and source map

8.16 Minimum interpretation checklist

Checkpoint	Question
Clinical status	Is the patient failing perfusion, ventilation, or neurological function now?
Sampling	Are gas and chemistry contemporaneous and technically credible?
Primary processes	Which changes drive the pH, and could opposing processes conceal severity?
Compensation	Is CO2 or bicarbonate appropriate for the primary process and its duration?
Gap and albumin	Is there hidden high-gap acidosis despite a small measured gap or normal pH?
Mixed metabolism	Does the gap rise match the bicarbonate fall, allowing for baseline and uncertainty?
Cause and action	Which source, loss, infusion, drug, renal, or respiratory problem must be treated?
Reassessment	What clinical response and repeat measurements will verify that the intervention helped?

Sources and educational scope

[1] Farley P, Smith SG, Sperry JL, Schreiber MA. Shock, Electrolytes, and Fluid. Sabiston Textbook of Surgery. 22nd ed. Chapter 33. Integrated with the chapter discussion of hypoperfusion, acidosis, chloride exposure, electrolytes, and resuscitation reviewed in preceding sections. The structured interpretation method, calculations, tables, and cases here are original educational synthesis rather than a translation of a dedicated textbook subsection.

[2] Jung B, et al. Diagnosis and management of metabolic acidosis: guidelines from a French expert panel. Ann Intensive Care. 2019;9:92. doi:10.1186/s13613-019-0563-2. Used for the diagnostic role of blood gases, expected respiratory response, albumin-adjusted anion gap, and cause-directed assessment. Compensation rules are approximate bedside physiological estimates, not treatment targets.

[3] Prescott H, Antonelli M, Alhazzani W, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2026. doi:10.1007/s00134-026-08361-1. Official SCCM recommendations: avoid bicarbonate solely for hemodynamic improvement in hypoperfusion-related lactic acidemia; consider selected severe metabolic acidemia with AKIN stage 2-3. Conditional recommendations; certainty low and very low, respectively.

Current recommendations checked 2 November 2026. Numbers in cases are internally consistent teaching approximations. Severe disturbances, airway intervention, bicarbonate therapy, and renal-replacement decisions require appropriate monitored care and local protocols.

9. Integrated algorithm and final review tables

9.1 One framework, repeated as physiology changes

Use this sequence to integrate the chapter. Assessment, support, and definitive treatment proceed together in an unstable patient. A completed classification is not a prerequisite for treating a strongly suspected immediately reversible threat. The algorithm is an original teaching synthesis of Sections 1-8, supported by the sources cited there.

Decision point	Action / branch	Reassessment trigger
1. Threatened perfusion or organ function?	Assess airway, breathing, circulation, neurological status and monitoring. Call for senior surgical/anesthesia/critical-care help as indicated.	Deterioration, rising support, altered consciousness, or new respiratory failure.
2. Cause that cannot wait?	Control major bleeding; treat suspected tension physiology; manage anaphylaxis or dangerous arrhythmia through its emergency pathway.	Do not wait for a complete test panel or unsafe imaging transfer.
3. Dominant mechanism?	Evaluate losses, vascular tone, pump function, and obstruction; use focused echo and lung assessment where appropriate.	Mixed physiology, discordant findings, absent or transient response.
4. Appropriate next intervention?	Select blood-based support for hemorrhage, targeted fluid for plausible volume benefit, vasoactive/inotropic support when indicated, and definitive source treatment.	Define expected improvement and stopping conditions before repetition.
5. Has treatment helped without harm?	Compare pressure, flow, tissue perfusion and tolerance. Validate measurements.	Worsening oxygenation, congestion, persistent source, or no useful flow response.
6. What phase now?	Continue rescue when needed; stabilize; stop unnecessary input; consider individualized de-escalation only when ready.	Reopen the algorithm after each important change.

Clinical pearl
At every loop, state: What is failing? What must be corrected now? What evidence would change my explanation? What harm will stop this treatment?

Emergency priorities: what must not be delayed

9.2 Parallel action takes priority over perfect classification

Clinical pattern	Priority	Do not be reassured by
Probable major ongoing hemorrhage	Activate major hemorrhage support and expedite an appropriate control route.	Normal early hemoglobin, low drain output, or a transient pressure response.
Abrupt respiratory/circulatory collapse after a procedure	Evaluate airway, ventilation and obstruction; treat a strongly suspected lethal reversible cause promptly.	An existing sepsis diagnosis or a pending chest radiograph.
Sudden airway/breathing/circulation compromise after exposure	Anaphylaxis pathway and adrenaline treatment as indicated.	Absent rash.
Severe metabolic acidosis with falling respiratory effort	Urgent ventilation/airway assessment and cause treatment; prepare for loss of compensation.	Normal saturation or a CO2 inside the usual reference interval.
Seizure with hypotonic hyponatremia	Monitored hypertonic rescue, then controlled correction and cause assessment.	A plan to correct slowly without first addressing severe symptoms.
Severe hyperkalemia / ECG toxicity	Protect when indicated, shift, eliminate, and monitor potassium/glucose.	A normal ECG in a high-risk severe case or a short-lived response to insulin.

9.3 Choose a safe route to definitive care

Ask whether the patient can tolerate transport and delay, whether the test changes the next intervention, and what control option is actually available. The most technically elegant option is not automatically the fastest safe option. Record a fallback for failed treatment or deterioration during transfer.

Clinical pearl
A monitor can guide support, but cannot establish that bleeding, infection, ischemia, or obstruction has been controlled.

Shock phenotype: compact differential

9.4 Use patterns to generate hypotheses

Mechanism	Typical clues	Main trap
Hypovolemic	Loss history, reduced filling, often cool skin and low flow.	Normal hemoglobin or lack of tachycardia does not exclude blood loss.
LV cardiogenic	Low flow, compatible cardiac lesion, often pulmonary congestion.	Chronic low EF may not explain the new event; troponin alone does not identify the mechanism.
RV cardiogenic	Venous congestion and low forward flow; lungs may be clear.	Clear lungs do not exclude a cardiac contribution.
Distributive	Vasodilation/maldistribution; output and peripheral temperature vary.	Warm skin does not prove infection; cold skin does not exclude sepsis.
Obstructive	Compatible tamponade, tension pneumothorax or pulmonary embolism physiology.	Effusion is not automatically tamponade; RV dilation alone is not proof of embolism.
Mixed	Features of more than one process or changing response during treatment.	A second diagnosis does not erase the first.

9.5 Four domains for response assessment

Domain	Useful question
Pressure	Is pressure appropriate for this patient, with what amount of support?
Flow	Did reliable stroke-volume/output assessment improve, and what limits it?
Tissue perfusion	Are examination, organ function and contextual biomarkers recovering?
Tolerance	Is treatment worsening lung function, venous congestion or tissue edema?

A favorable result in one domain is insufficient if the others deteriorate. If measurements disagree, verify technique, identify confounders, and obtain the information that changes the next action. Reassessment is a clinical decision, not merely another set of numbers.

Fluid and vasoactive decisions: final matrix

9.6 Separate need, responsiveness, and tolerance

Finding	Next judgment
Likely volume-responsive hypoperfusion without important intolerance	Use a defined, appropriate challenge or replacement intervention, then reassess actual benefit.
Positive preload test with worsening congestion	Do not equate responsiveness with a favorable fluid prescription. Reconsider dose, alternatives, and cause.
No response to a valid challenge	Stop repeating empirical boluses; reconsider pump function, tone, obstruction and ongoing losses.
PPV/SVV displayed during arrhythmia or spontaneous breathing	Treat validity as uncertain; do not prescribe from the displayed threshold alone.
Marked vasodilatory hypotension	Support pressure promptly while assessing volume and treating the source; do not require completion of arbitrary fluids first.
Escalating pressor requirement	Check delivery and pressure measurement, then reopen the mechanism and source differential.
Acute phase resolved with harmful accumulation	Stop unnecessary input; consider monitored removal if perfusion tolerates it.

9.7 Product selection in context

Major hemorrhage: blood-based resuscitation and control. **Many nonhemorrhagic resuscitation settings:** an appropriate isotonic crystalloid, with balanced solutions often suitable. **Brain injury or specific chloride deficits:** follow the relevant exception. **Albumin:** a selected indication, not an automatic response to a low serum value. **Hypotonic water:** a defined water/maintenance indication, not principal rapid shock resuscitation.

In septic vasodilatory shock, norepinephrine is generally initial support; adjuncts and inotropes depend on persistent physiology and cardiac function. Do not copy this sequence into anaphylaxis or every cardiogenic/obstructive scenario. Use formulation-specific local protocols and monitored access safeguards.

Clinical pearl
More fluid, more pressure support, and more monitoring are different interventions. Choose the one that addresses the current failure.

Hemorrhage and daily fluid prescription: review cards

9.8 Major hemorrhage: control plus clot-preserving support

Task	Review point
Activate early	Anticipated rapid ongoing blood need matters more than a retrospective 24-hour threshold.
Control the source	Use operative, endovascular, endoscopic or direct methods appropriate to physiology and anatomy.
Deliver organized support	Use the local component or whole-blood pathway, then target current deficits.
Protect clot formation	Warm; monitor ionized calcium, fibrinogen, platelets and coagulation function.
Apply exceptions	Pressure targets, TXA indications and time windows are context-specific.
De-escalate explicitly	Stop automatic packs when their indication ends; reassess ongoing loss and transfusion harm.

9.9 Daily prescription: a separate accounting problem

Component	What belongs in the plan
Maintenance	Individualized ordinary needs after subtracting other provision.
Existing deficit or excess	A separate correction plan with timing, tolerance and biochemical limits.
Abnormal loss	Measured source, expected composition, replacement interval and reassessment.
Redistribution	Assess effective filling and congestion; do not replace an unmeasured “third space” indefinitely.
Hidden intake	Count medication carriers, nutrition, flushes, blood and oral/enteral fluids.

A stable 70-kg adult starting estimate of 25-30 mL/kg/day gives 1750-2100 mL/day before adjustment. It is total provision, not extra IV fluid. The 4-2-1 hourly rule would give 2640 mL/day in that example, illustrating why an inherited rate must be reviewed. Kidney/cardiac disease, frailty, obesity and specialized conditions modify the plan.

Electrolyte emergencies: integrated comparison

9.10 Treat danger and mechanism together

Disorder	Priority	Critical safety check
Hyponatremia	Establish tonicity and symptoms; rescue severe neurological disease, then treat the cause.	Prevent overcorrection, especially with malnutrition, hypokalemia or dilute water diuresis.
Hypernatremia	Assess circulation, free-water loss, sodium input, urine concentration and access to water.	A deficit estimate excludes ongoing losses; coordinate water and antidiuretic treatment.
Hyperkalemia	Protect if indicated; shift and arrange elimination.	Normal ECG does not guarantee safety; repeat potassium and monitor delayed hypoglycemia/rebound.
Hypokalemia	Replace safely and address losses, shifts and magnesium deficiency.	Never IV push; count potassium in every preparation and adapt to kidney function.
Hypocalcemia	Use ionized calcium when clinically important; rescue symptomatic disease.	Total/albumin-adjusted values can mislead; correct magnesium and consider neck surgery or citrate.
Hypercalcemia	Assess symptoms, cause, renal function and appropriate volume restoration.	Avoid a fixed large fluid prescription in limited cardiac/renal tolerance.
Magnesium disorders	Recognize linked potassium/calcium problems or toxicity with impaired clearance.	Salt mass is not elemental magnesium; monitor renal function and accumulation.
Phosphate disorders	Assess energy/muscle effects, cell breakdown and nutrition-related shifts.	Choose sodium versus potassium preparation deliberately; monitor calcium and compatibility.

Refeeding cross-check

After prolonged poor intake, new weakness, respiratory difficulty, arrhythmia or edema following calorie delivery should prompt review of phosphate, magnesium, potassium, thiamine, glucose and all nutrient sources. A normal prefeeding serum concentration does not establish adequate stores. Coordinate nutrition changes and replacement rather than treating one isolated value.

Clinical pearl
A repeat dose is a new prescription. Recheck current results, renal clearance, ongoing loss and all accompanying electrolytes before giving it.

Acid-base and calculation reference

9.11 A compact calculation sheet

Calculation	Expression / interpretation
Cardiac output	Heart rate x stroke volume.
Oxygen content	CaO2 = 1.34 x Hb x SaO2 + 0.003 x PaO2. Hb in g/dL; saturation as a fraction; oxygen tension in mm Hg.
Oxygen delivery	DO2 = CO x CaO2 x 10 when CO is L/min and content is mL/dL.
Estimated osmolality	2 x sodium + glucose/18 + BUN/2.8 in conventional units; effective osmolality omits BUN.
Anion gap, no potassium	Sodium - (chloride + bicarbonate); use the local reference range.
Albumin-adjusted gap	Measured gap + 2.5 x (4 - albumin in g/dL), an approximation.
Winter's estimate	Expected PaCO2 = 1.5 x bicarbonate + 8, plus or minus 2 mm Hg.
Free-water deficit estimate	Estimated TBW x [(sodium / desired sodium) - 1]; not an immediate infusion order.
Percent flow change	(Post-test flow - baseline flow) / baseline flow x 100.

9.12 Interpret before treating

Read pH, identify metabolic/respiratory direction, test expected compensation, calculate the albumin-adjusted gap, consider mixed metabolic processes, then identify cause and treatment. A “normal” CO2 may be excessive during metabolic acidosis; alkalemia can conceal a high-gap process. Gas and chemistry should be contemporaneous.

Do not use a formula to override an urgent bedside threat. Deficit calculations do not predict a sudden change in renal excretion. A favorable global oxygen calculation cannot exclude regional ischemia. A monitor change smaller than measurement error cannot certify therapeutic benefit.

Clinical pearl
Units, timing and measurement validity are part of the calculation. A mathematically correct number can still support the wrong clinical decision.

Final synthesis: decisions that demonstrate surgical judgment

9.13 A concise handover that remains actionable

State: the current perfusion problem; predominant and possible mixed mechanisms; source-control status; interventions already given; measured benefit and harm; unresolved findings; the next action and its trigger; and who will reassess. Include the trajectory of support, not only the latest pressure.

Before accepting the current plan	Ask
A reassuring number	What important disease can this number fail to exclude?
Another bolus	What benefit is expected, and what would make me stop?
More vasopressor	Is the mechanism still correct and is the drug reaching the patient?
A better lactate or pH	Has the source been controlled, and are regional or respiratory problems being missed?
A maintenance order	What other intake, abnormal loss or clearance change has occurred?
Electrolyte replacement	What coupled deficiency, accompanying cation or kidney problem changes the dose?
An imaging transfer	Can the patient tolerate the delay, and will the result change immediate treatment?
De-escalation	Has the acute phase ended, and how will deterioration be detected?

9.14 Evidence and use of this review

This section consolidates the original algorithms, tables and cases in Sections 1-8. The base text is Farley, Smith, Sperry and Schreiber, Sabiston Textbook of Surgery, 22nd edition, Chapter 33. It integrates the cited ESICM 2025 shock-monitoring and fluid guidance; Surviving Sepsis Campaign 2026; European trauma bleeding guidance 2023; NICE CG174/CG32; and the electrolyte and acid-base sources listed in the relevant sections. Current society guidance was rechecked on 2 November 2026.

Recommendations differ in evidence certainty and patient population. Selected sepsis, trauma, brain-injury, renal, endocrine and nutrition pathways are not interchangeable. Exact emergency drug doses, correction limits, infusion concentrations and monitoring requirements remain linked to the detailed section and local protocol; this review does not replace those details.

Clinical pearl
The strongest judgment combines a defensible working explanation with a clear plan for recognizing when it is wrong.

10. Surgical judgment: 100 extended-case questions

How to use this bank

Each item asks for one best answer from the information and decision point stated. Attempt the question before reading the explanation. A full bedside plan may include several simultaneous actions; the requested answer identifies the most defensible interpretation or priority among the four alternatives, not every step of care.

The bank contains 100 original extended cases, designed to test reasoning across shock mechanisms, monitoring validity, hemorrhage control, fluid prescribing, electrolyte safety, and mixed acid-base disease. Clinical trajectories and competing treatment proposals are included deliberately. Difficulty and discrimination have not been psychometrically validated; faculty review and learner performance should inform summative use.

Questions	Main emphasis
1-10	Occult hypoperfusion and competing shock mechanisms.
11-20	Response assessment, test validity and tolerance.
21-30	Hemorrhage control and hemostatic support.
31-44	Fluid selection, vasoactive decisions and daily prescribing.
45-68	Sodium, potassium and linked electrolyte emergencies.
69-80	Acid-base interpretation and respiratory compensation.
81-100	Integrated and evolving bedside decisions.

The blocks describe dominant themes; several questions cross themes. All answer positions are balanced across A-D, with no intended content cue from position. The answer key follows the questions. Each explanation identifies the key reasoning and why the other choices do not fit this case.

Source map: use the numbered teaching section shown beneath each explanation and its full references. The source framework is Sabiston 22nd edition, Chapter 33, supplemented by the explicitly identified contemporary society guidance in Sections 1-9. These questions and oral scenarios are original educational applications, not copied examination items. Emergency doses and protocols remain subject to local implementation.

Question 001

A 77-year-old man is reviewed six hours after pancreatic resection. His blood pressure is 116/70 mm Hg, heart rate 84/min on chronic beta blockade, and oxygen saturation 98%. He is increasingly inattentive, his hands are cool, and urine output has fallen over three hours. Lactate has risen from 1.8 to 4.6 mmol/L; hemoglobin is 10.7 g/dL compared with 11.1 immediately after surgery. The drain contains little blood, but abdominal girth is increasing. He received substantial intraoperative crystalloid and now needs more oxygen. The resident proposes waiting for hypotension or a larger hemoglobin fall before investigating bleeding. The surgeon must select the most defensible response while avoiding indiscriminate additional fluid. The operative note describes difficult dissection near major vessels, and the last fluid challenge did not improve measured forward flow. Blood bank and anesthesia support can be mobilized immediately; the decision should not depend on waiting for the next routine blood count.

What is the best next approach?

A. Observe until systolic pressure falls below 90

B. Give repeated crystalloid until lactate normalizes

C. Escalate assessment for concealed bleeding while supporting perfusion

D. Exclude hemorrhage because the drain is nearly empty

Answer and option analysis

A - Why this option does not fit: Waiting for profound hypotension delays recognition in a compensated patient.

B - Why this option does not fit: Oxygen deterioration suggests limited tolerance; lactate alone cannot prescribe volume.

C - Best answer: Clinical trajectory supports occult hypoperfusion; neither beta-blocked heart rate nor early hemoglobin excludes blood loss.

D - Why this option does not fit: A drain does not sample every potential concealed bleeding compartment.

Review link: teaching Section 1. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 002

A 65-year-old woman has undergone emergency vascular surgery. Initial shock improves after treatment: MAP is 72 mm Hg, lactate decreases from 5.2 to 2.4 mmol/L, and peripheral temperature improves. She nevertheless develops worsening diffuse abdominal pain, new distension, and blood-stained stool. Analgesia makes the examination difficult, and she has no rigid abdomen. Kidney function is deteriorating. The ICU team considers the falling lactate evidence that tissue perfusion is adequate everywhere and recommends waiting until morning for abdominal imaging. Transport currently appears feasible with monitoring. The surgeon must interpret the global trend against the evolving focal findings rather than decide from a single biomarker. Her earlier resuscitation corrected systemic pressure but did not establish intestinal viability. The surgeon must decide whether the improving global trend justifies delay when anatomical findings are worsening, and whether a safely obtainable study can change the immediate intervention rather than merely document deterioration later.

Which judgment is most appropriate?

A. Defer investigation because lactate is approaching normal

B. Give bicarbonate to prevent further intestinal ischemia

C. Diagnose benign postoperative ileus without further evaluation

D. Urgently investigate possible intestinal ischemia despite improving global markers

Answer and option analysis

A - Why this option does not fit: Global biomarker recovery cannot exclude an important regional vascular problem.

B - Why this option does not fit: Buffer administration does not establish bowel viability or correct the source.

C - Why this option does not fit: Blood-stained stool and worsening pain make premature benign attribution unsafe.

D - Best answer: Regional ischemia can progress while systemic pressure and lactate improve; the new focal findings require explanation.

Review link: teaching Section 1. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 003

A 58-year-old woman develops weakness and poor peripheral perfusion after a difficult pelvic operation. Oxygen saturation is 100% on supplemental oxygen and PaO2 is 150 mm Hg. Hemoglobin is 6.1 g/dL, cardiac output is estimated at 3 L/min, and lactate is rising. The team is evaluating ongoing bleeding. A resident suggests that oxygen delivery must be adequate because saturation is maximal, and proposes increasing inspired oxygen further as the principal treatment. Her lungs already show congestion after several crystalloid boluses. No transfusion decision should depend on saturation alone, and the surgeon must distinguish oxygen tension, oxygen content, and blood flow in choosing the next mechanism-directed action. The estimated flow measurement is being checked, but the anemia is confirmed on a reliable sample. Further oxygen can affect dissolved content slightly, yet the treatment plan still needs to address the suspected loss, red-cell mass, and the patient's limited tolerance of additional nonblood volume.

Which explanation best guides management?

A. PaO2 above 100 guarantees adequate tissue delivery

B. Low hemoglobin and flow can markedly limit delivery despite maximal saturation

C. Dissolved oxygen becomes the dominant carrier at this PaO2

D. Additional crystalloid reliably restores oxygen-carrying capacity

Answer and option analysis

A - Why this option does not fit: Oxygen tension does not measure hemoglobin mass, cardiac output, or regional distribution.

B - Best answer: Most arterial oxygen is hemoglobin-bound; normal saturation cannot compensate for severe anemia and low output.

C - Why this option does not fit: At this tension dissolved oxygen remains a small component of total content.

D - Why this option does not fit: Crystalloid does not replace red cells and may worsen dilution and congestion.

Review link: teaching Section 1. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 004

A 62-year-old man with abdominal sepsis receives source treatment and vasoactive support. After epinephrine is added for low-output physiology, measured flow and capillary refill improve and pressure stabilizes. Lactate nevertheless rises from 3.1 to 4.8 mmol/L. He has accumulated edema and requires increasing oxygen. The abdominal source was technically difficult to control, so residual infection remains possible. One clinician recommends serial fluid boluses until lactate falls; another says the entire elevation is pharmacological and no reassessment is needed. The surgeon has access to repeat focused echo, serial examination, and imaging if current stability permits. The decision must account for both drug effects and unresolved illness. The team has not established whether the new respiratory findings reflect permeability injury, cardiac dysfunction, or accumulated volume. These uncertainties influence tolerance but do not remove the obligation to reassess a potentially persistent abdominal process and the response to each support change.

What is the best interpretation?

A. Stop evaluating the source because pressure improved

B. Immediately remove all circulatory support because lactate rose

C. Treat every lactate rise as proof of preload deficiency

D. Reassess perfusion and source control while considering an adrenergic lactate contribution

Answer and option analysis

A - Why this option does not fit: Improved support variables do not certify complete surgical control.

B - Why this option does not fit: Abrupt withdrawal can worsen shock; therapy should follow the full physiological assessment.

C - Why this option does not fit: Lactate is not a direct measure of volume responsiveness.

D - Best answer: Epinephrine can increase lactate, but this does not exclude persistent disease or justify automatic fluids.

Review link: teaching Section 1. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 005

A 70-year-old woman remains critically ill after drainage of abdominal infection. MAP is maintained with increasing norepinephrine. Her central venous oxygen saturation is 82%, but she is confused, mottled, oliguric, and increasingly acidemic. Hemoglobin is stable, oxygen saturation is satisfactory, and the sampling catheter position is known. The resident interprets the high ScvO2 as proof that oxygen delivery and extraction are adequate and recommends reducing surveillance. The surgeon is concerned about maldistribution, impaired extraction, and an incompletely treated source. A repeat flow assessment and abdominal review are available, but each requires coordination. The next plan must explain the discordant bedside and venous findings. The value was obtained from a correctly identified central venous port rather than a pulmonary artery. This improves sampling confidence but does not make the result a direct measure of tissue extraction everywhere; the clinical trajectory remains a separate source of evidence.

Which conclusion is defensible?

A. High ScvO2 excludes shock when arterial saturation is normal

B. High ScvO2 does not exclude ongoing tissue or regional hypoperfusion

C. High ScvO2 independently indicates a red-cell transfusion

D. ScvO2 and pulmonary-artery SvO2 are identical measurements

Answer and option analysis

A - Why this option does not fit: The worsening examination and escalating support remain evidence of circulatory failure.

B - Best answer: Venous saturation reflects delivery-demand-extraction relationships and can remain high despite serious distributive illness.

C - Why this option does not fit: Transfusion requires a clinical indication; this isolated measurement does not provide one.

D - Why this option does not fit: Sampling sites differ and their values cannot be assumed interchangeable.

Review link: teaching Section 1. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 006

A ventilated 54-year-old man with improving septic shock undergoes right-sided central venous catheter placement. Within minutes pressure falls to 60/35 mm Hg, oxygen saturation decreases, peak airway pressure rises, and right-sided air entry becomes markedly reduced. The abdomen and surgical drains are unchanged. A portable radiograph has been ordered but will take several minutes, and ultrasound is not immediately at the bedside. One resident proposes more norepinephrine because sepsis was the established diagnosis; another wants radiographic confirmation before any pleural intervention. The surgeon must decide whether the abrupt timing and respiratory asymmetry warrant treatment before completion of the usual diagnostic sequence. No previous unilateral chest abnormality was documented before insertion. The team can assess the tube and ventilator circuit in parallel, but the surgeon must decide whether those checks and a delayed image should postpone treatment of a strongly suspected mechanical emergency.

What is the best immediate priority?

A. Treat strongly suspected tension pneumothorax through emergency decompression

B. Give a large crystalloid bolus as definitive treatment

C. Attribute the collapse entirely to recurrent vasodilation

D. Wait for the radiograph before treating the chest

Answer and option analysis

A - Best answer: In this unstable compatible presentation, waiting for radiography risks fatal delay to reversible obstruction.

B - Why this option does not fit: Fluid does not relieve the mechanical intrathoracic obstruction.

C - Why this option does not fit: The new procedural and unilateral respiratory pattern requires a reopened differential.

D - Why this option does not fit: Confirmation should not delay emergency treatment of convincing tension physiology.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 007

A 67-year-old man develops hypotension and dyspnea after major colorectal surgery. Focused echo shows a dilated right ventricle with reduced LV filling. He has longstanding pulmonary hypertension, is ventilated with substantial airway pressures, and received a large fluid load. There is no baseline bedside image, but prior records mention chronic RV enlargement. Oxygenation and pressure permit cautious monitored investigation. A resident considers the image diagnostic of acute pulmonary embolism and proposes immediate thrombolysis despite the recent operation. The surgeon must balance a potentially lethal embolus against alternative explanations and the bleeding risk of reperfusion treatment. A more complete echo and appropriate definitive imaging can be arranged promptly. The proposed treatment could cause substantial bleeding at the fresh operative site, while missing an acute embolus would also be dangerous. The decision therefore requires the most discriminating information that can be obtained safely within the patient's current stability and available local pathway.

Which approach best reflects judgment?

A. Integrate clinical probability and prior disease before selecting definitive imaging and treatment

B. Administer thrombolysis solely because the RV is large

C. Give repeated fluid until the RV becomes smaller

D. Exclude embolism because RV disease was documented previously

Answer and option analysis

A - Best answer: RV dilation is nonspecific; stable investigation can distinguish acute embolism from chronic or ventilator-related RV stress.

B - Why this option does not fit: The image alone does not establish acute embolism or justify major bleeding risk.

C - Why this option does not fit: Additional volume can worsen RV congestion and does not identify the cause.

D - Why this option does not fit: Chronic disease does not prevent a superimposed acute embolus.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 008

A 29-year-old man arrives after a motorcycle collision with quadriparesis, pressure 82/44 mm Hg, and heart rate 56/min. His hands are warm. He also has pelvic tenderness and flank bruising. Initial hemoglobin is 13.0 g/dL and focused abdominal ultrasound shows no free fluid. During preparation for CT he becomes less interactive and capillary refill worsens. The resident labels isolated neurogenic shock and suggests vasopressors without further hemorrhage assessment. The surgeon must decide what the characteristic neurological pattern establishes and what it does not exclude. Pelvic stabilization and major hemorrhage resources are available, while the safety of transport is changing with the clinical trajectory. The patient also has a mechanism capable of causing more than one injury, and no complete assessment of the retroperitoneum has occurred. The surgeon must evaluate the evolving state rather than equate a recognizable autonomic pattern with exclusion of all concurrent sources of circulatory loss.

What is the best working approach?

A. Call circulatory failure spinal shock and stop reassessment

B. Exclude bleeding because tachycardia is absent

C. Use negative abdominal ultrasound to exclude pelvic hemorrhage

D. Assess and treat possible combined neurogenic and hemorrhagic shock

Answer and option analysis

A - Why this option does not fit: Spinal shock describes neurological reflex depression, not a complete circulatory diagnosis.

B - Why this option does not fit: Autonomic injury and other factors can prevent the expected heart-rate response.

C - Why this option does not fit: That examination does not reliably exclude retroperitoneal or pelvic bleeding.

D - Best answer: Sympathetic failure can coexist with concealed pelvic or retroperitoneal loss despite bradycardia and normal early hemoglobin.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 009

A 46-year-old woman on the surgical ward receives an antibiotic infusion. Within minutes she develops wheeze, a hoarse voice, hypotension, and marked distress. No urticaria is visible. She has no history of cardiac failure, and the deterioration followed the new exposure closely. The infusion is stopped and emergency help is called. A junior clinician recommends an antihistamine first because the absence of rash makes anaphylaxis uncertain; another wants to wait for a confirmatory blood test. This is not a specialist anesthetic environment using an established IV-adrenaline protocol. The surgeon must choose the treatment priority while airway and respiratory support are prepared. The onset is rapid and the first exposure-related symptoms involved more than one vital system. A later diagnostic sample may support the event review, but the immediate bedside decision must reflect the threat to airway and circulation rather than the availability of confirmatory testing.

Which intervention should be prioritized?

A. Antihistamine before adrenaline

B. Intramuscular adrenaline through the emergency anaphylaxis pathway

C. Wait for serum testing to establish the diagnosis

D. Treat only with a fluid infusion

Answer and option analysis

A - Why this option does not fit: Antihistamines do not treat the immediately threatening airway or circulatory problem.

B - Best answer: Airway, breathing, and circulation compromise after exposure warrants prompt treatment even without skin findings.

C - Why this option does not fit: Anaphylaxis is a clinical emergency; confirmatory investigations must not postpone treatment.

D - Why this option does not fit: Fluid can support circulation but does not replace first-line adrenaline and airway assessment.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 010

A 71-year-old man deteriorates after cardiac surgery. He has rising venous pressure, low output, oliguria, and escalating circulatory support. A limited transthoracic study shows only a small anterior effusion, but posterior views are poor. He has no striking pulsus paradoxus and heart sounds are difficult to assess. The resident concludes that tamponade is excluded because the effusion looks small and the classic physical triad is incomplete. The surgeon knows postoperative collections may be loculated and that ventilation changes the examination. Further targeted imaging and immediate surgical review are available. The patient is too unstable for a prolonged diagnostic transfer away from definitive support. Positive-pressure ventilation and postoperative anatomy can alter familiar diagnostic signs. An incomplete image should therefore be treated as incomplete evidence, and the next test or intervention should be chosen according to its ability to identify and relieve the current hemodynamic threat safely.

What should happen next?

A. Exclude tamponade because anterior fluid depth is small

B. Send the patient for a routine distant CT without reassessment

C. Assume primary myocardial failure and stop looking for obstruction

D. Urgently reassess for postoperative localized tamponade with the surgical team

Answer and option analysis

A - Why this option does not fit: Location and pressure effects matter more than one limited size measurement.

B - Why this option does not fit: An unstable patient needs a safe, decision-directed route to diagnosis and control.

C - Why this option does not fit: Pump dysfunction and postoperative obstruction can produce overlapping low-output findings.

D - Best answer: Limited views and absent classic signs do not exclude a hemodynamically important loculated collection.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 011

A 66-year-old woman remains on low-dose norepinephrine after surgery for peritonitis. Capillary refill and interaction are improving, but urine output is low. She is 6 L positive, oxygen requirement is rising, and lung ultrasound shows increasing B-lines. A technically reliable passive leg raise increases stroke volume by 14%, returning to baseline afterward. The resident proposes repeated 500-mL boluses because she is a responder. Echo shows no new obstruction, and the source-control result remains under review. The surgeon must decide whether a physiological ability to increase flow proves that additional volume will provide enough benefit to outweigh worsening respiratory tolerance. No new severe hypotension has occurred. The earlier fluid prescription was appropriate during rescue, but the patient has entered a different trajectory. The team needs to specify what additional clinical benefit it expects now and what respiratory or venous findings would make the next infusion unacceptable.

Which judgment is best?

A. Responsiveness is present but additional fluid still requires a favorable benefit-harm assessment

B. Give repeated boluses until PLR becomes negative

C. Ignore the test because pressure changed little

D. Begin aggressive removal solely from the positive balance

Answer and option analysis

A - Best answer: A positive test does not establish need or tolerance; emerging pulmonary harm can change the treatment choice.

B - Why this option does not fit: A negative test is not a universal resuscitation endpoint and may be reached through harmful overfilling.

C - Why this option does not fit: A genuine flow response can occur without a large arterial-pressure change.

D - Why this option does not fit: Readiness for removal requires perfusion and phase assessment, not balance alone.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 012

A 59-year-old man after emergency laparotomy develops atrial fibrillation and intermittent spontaneous ventilator triggering. Tidal volume is low for lung protection. The monitor displays pulse pressure variation of 19%, while MAP falls and oxygenation worsens. He has already received several liters of fluid. The resident cites a conventional PPV threshold and orders another liter. There is no valid baseline stroke-volume comparison, and the arterial waveform has not been checked since repositioning. The surgeon must separate true deterioration from uncertain dynamic measurements, without delaying assessment of recurrent abdominal pathology or arrhythmia-related low flow. Reliable targeted echo and waveform review can be performed at the bedside. The displayed percentage looks precise, but precision of display does not verify physiological validity. The question is whether the current measurement can separate a volume-responsive process from arrhythmia, pump dysfunction or continuing surgical disease well enough to justify a potentially harmful intervention.

What is the best next assessment strategy?

A. Validate monitoring and use a suitable alternative flow-based assessment

B. Dismiss shock because the dynamic index is unreliable

C. Raise tidal volume permanently to improve the monitor display

D. Give the liter because PPV exceeds the usual cutoff

Answer and option analysis

A - Best answer: Arrhythmia and spontaneous effort undermine conventional PPV interpretation; the displayed value cannot independently justify fluid.

B - Why this option does not fit: An invalid index does not negate worsening clinical perfusion and support needs.

C - Why this option does not fit: Ventilator changes should protect the patient rather than serve an isolated index.

D - Why this option does not fit: A threshold is meaningful only when the validity conditions are satisfied.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 013

A 63-year-old woman has borderline pressure after a prolonged operation but no immediate life-threatening deterioration. A resident performs a preload maneuver and reports that LVOT VTI increased from 14.0 to 14.4 cm. The Doppler angle and sample position changed between measurements, and heart rate was not recorded. She has worsening edema and oxygen needs. The resident calls the result definitely positive and proposes more fluid. Another clinician calls the result definitely negative and proposes diuresis. The surgeon must determine whether the available measurement can resolve the treatment question, rather than force a binary label from a small and technically inconsistent change. The observed pressure fluctuates with respiration, and different beats were selected in the two acquisitions. These technical issues do not establish a fluid prescription in either direction; the immediate need is an interpretation consistent with the method's actual precision and the patient's tolerance.

Which conclusion is most defensible?

A. Convert VTI directly to cardiac output without heart rate

B. The test proves that immediate fluid removal is safe

C. The apparent change is too uncertain to establish a useful flow response

D. The test proves clinically important responsiveness

Answer and option analysis

A - Why this option does not fit: Cardiac output depends on heart rate as well as stroke volume.

B - Why this option does not fit: An uncertain preload result does not establish readiness for removal.

C - Best answer: A 2.9% change with inconsistent acquisition can lie within measurement error; neither definite label is justified.

D - Why this option does not fit: The magnitude and technical inconsistency do not support that certainty.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 014

A 72-year-old man in the ICU has low cardiac output, elevated central venous pressure, and worsening renal function after abdominal surgery. His lungs are relatively clear, but the liver is congested and the right ventricle appears poorly functioning. He is receiving positive-pressure ventilation and has accumulated fluid. A resident interprets high CVP as proof that effective circulation is adequate and recommends no further mechanism assessment. Another interprets oliguria as evidence of underfilling and proposes repeated boluses. The surgeon must distinguish pressure at a venous measurement site from forward flow and identify whether RV failure, obstruction, ventilation, or another contributor is driving the deterioration. The patient is receiving a level of ventilatory support that can influence venous pressure independently of effective filling. The team must decide whether additional volume would improve forward circulation or predominantly raise upstream pressure and worsen renal and hepatic congestion.

What should guide the next decision?

A. Give repeated fluid solely because urine output is low

B. Exclude cardiac disease because the lungs are clear

C. Assess RV function, obstruction, flow, and congestion together before more volume

D. Treat high CVP as a direct measure of normal circulating flow

Answer and option analysis

A - Why this option does not fit: This may worsen RV congestion without improving output.

B - Why this option does not fit: Predominantly RV failure can occur without pulmonary edema.

C - Best answer: High venous pressure does not certify adequate forward flow, and oliguria may reflect congestion rather than simple depletion.

D - Why this option does not fit: Venous pressure and effective arterial delivery are different physiological quantities.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 015

A 52-year-old man with severe pancreatitis has a tense abdomen, rising airway pressure, oliguria, and escalating circulatory support. The team performs a passive leg raise that shows no output increase. The resident declares absolute fluid unresponsiveness and excludes all possibility of underfilling. Bladder pressure has not yet been measured, and the ultrasound window is difficult. The surgeon is concerned that intra-abdominal pressure, measurement limitations, and mixed physiology may affect both the test and the kidney findings. Additional fluid also carries obvious risk. The next step should neither prescribe volume reflexively nor accept an unqualified conclusion from a potentially compromised maneuver. The tense abdomen could be both a consequence of prior fluid exposure and a contributor to the present organ dysfunction. The surgeon must investigate that possibility while interpreting any functional test cautiously, rather than let a single negative maneuver end the assessment.

Which interpretation is best?

A. Assess abdominal pressure and test validity while evaluating perfusion and tolerance

B. Diagnose renal failure unrelated to the abdominal process

C. Use the negative test to exclude every volume-related contribution

D. Give fluid until the abdomen becomes less tense

Answer and option analysis

A - Best answer: Intra-abdominal hypertension can compromise PLR interpretation and itself contribute to organ dysfunction.

B - Why this option does not fit: The combination of pressure-related findings requires a linked physiological evaluation.

C - Why this option does not fit: A compromised maneuver cannot support such a broad exclusion.

D - Why this option does not fit: Extra volume may aggravate edema and abdominal pressure.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 016

A 68-year-old man has recurrent hypotension after a vascular operation. A 250-mL fluid challenge raises MAP from 57 to 67 mm Hg for several minutes, but the pressure then falls, lactate continues to rise, and the abdomen enlarges. Stroke volume was not measured. Hemoglobin has not changed dramatically since the previous sample. A resident considers the short response proof that repeated fluid is the definitive treatment. The surgeon must decide whether the temporary pressure effect identifies the mechanism, whether ongoing loss remains plausible, and what action can establish durable recovery. The operating team and blood bank can be mobilized while the assessment continues. The previous operation created a plausible concealed bleeding site, and a brief numerical improvement does not show that loss has stopped. The next intervention must aim for sustained perfusion and source control rather than a series of short-lived pressure responses.

What is the most appropriate conclusion?

A. Repeat saline indefinitely because every rise represents recovery

B. The unchanged hemoglobin excludes important new bleeding

C. The response proves that hemorrhage has stopped

D. A transient response does not establish control; urgently evaluate ongoing hemorrhage

Answer and option analysis

A - Why this option does not fit: This delays control and can increase dilution and congestion.

B - Why this option does not fit: Concentration can lag acute loss and be affected by fluid administration.

C - Why this option does not fit: A temporary pressure rise does not show durable hemostasis.

D - Best answer: Brief support effects can occur while the underlying loss persists; the trajectory demands definitive-source assessment.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 017

A 64-year-old woman on stable support suddenly appears to have a MAP of 42 mm Hg after being turned. She remains interactive, peripheral perfusion is unchanged, and a cuff reads 69 mm Hg. The arterial tracing is damped and its baseline level changed relative to the transducer. There is no new respiratory or surgical finding. The resident starts escalating vasopressor doses before checking the system. The surgeon must avoid undertreating a genuine event while recognizing a likely technical cause. The line can be inspected immediately and independent measurements repeated. She already has distal ischemic risk from substantial vasoconstrictor exposure earlier in the admission. The patient has no other new finding suggesting an immediate independent shock mechanism, but continuous assessment must accompany technical verification. A reliable pressure should be restored quickly so necessary support is neither withheld in error nor escalated in response to an artifact.

What is the best first response?

A. Remove all monitoring because the line may be inaccurate

B. Escalate support solely from the displayed arterial value

C. Promptly validate leveling, zeroing, waveform and corroborating pressure while assessing the patient

D. Assume the cuff must be wrong because invasive measurements are always superior

Answer and option analysis

A - Why this option does not fit: The patient still needs reliable surveillance and an appropriately functioning system.

B - Why this option does not fit: A technically invalid pressure can lead to harmful overtreatment.

C - Best answer: The timing and clinical discordance strongly suggest artifact; verification prevents unnecessary pressor escalation.

D - Why this option does not fit: An arterial catheter can also generate important measurement error.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 018

A 75-year-old woman recovering from shock has improved mentation, warm hands, falling lactate, and decreasing vasopressor needs. Urine output remains 0.2 mL/kg/h over several hours and creatinine has risen. She has received multiple boluses without a useful flow response and now has increasing oxygen needs. The urinary catheter has not been checked. The resident requests another liter to reach a urine target, while another clinician proposes dialysis solely from the urine rate. The surgeon must determine which remaining assessment changes management and whether either automatic intervention is justified. There is no dangerous potassium value, severe refractory acidemia, or other established dialysis indication. No recent examination has determined whether abdominal pressure, venous congestion or catheter obstruction contributes to the low output. The surgeon must use the renal finding to trigger a focused assessment rather than assume it can be corrected by repeatedly increasing total water administration.

What is the best next approach?

A. Give fluid until urine output exceeds a fixed target

B. Ignore renal findings because lactate improved

C. Start dialysis solely because several hours of urine output are low

D. Evaluate catheter patency, AKI and congestion while withholding unsupported repeat boluses

Answer and option analysis

A - Why this option does not fit: An isolated urine target does not justify treatment despite emerging intolerance.

B - Why this option does not fit: Improved systemic markers do not eliminate a continuing organ problem.

C - Why this option does not fit: Renal replacement requires the full clinical indication and trajectory.

D - Best answer: Oliguria can lag recovery or reflect established injury, obstruction or congestion rather than ongoing fluid-responsive depletion.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 019

A 57-year-old man with cirrhosis has undergone source treatment for peritonitis. His pressure is maintained with decreasing support, capillary refill and interaction improve, and urine output is recovering. Lactate falls from 6.0 to 4.0 mmol/L but remains elevated. He is edematous and develops increasing oxygen requirement. A resident proposes additional fluid until lactate is normal; another proposes stopping all surveillance because liver disease explains the result. The surgeon must weigh impaired clearance against possible continuing production or regional pathology, and determine whether more volume has a defensible indication. No current flow assessment suggests a clear need for expansion. The current lactate trend is encouraging but does not map individual tissue beds. An integrated plan should identify which signs support recovery, which remain unexplained, and what specific new finding would prompt repeat imaging or intervention for residual disease.

Which response is best?

A. Assume all persistent lactate is caused by liver disease

B. Declare source control complete from the downward trend

C. Continue integrated reassessment without using lactate normalization as an automatic fluid target

D. Give more fluid regardless of pulmonary findings

Answer and option analysis

A - Why this option does not fit: A plausible confounder does not exclude ongoing pathological production.

B - Why this option does not fit: A falling systemic marker does not certify anatomical control.

C - Best answer: Clearance may contribute, but persistent disease remains possible and treatment tolerance limits empirical fluid.

D - Why this option does not fit: This treats a concentration rather than a demonstrated volume-responsive problem.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 020

A 61-year-old man remains in shock after initial treatment for presumed abdominal sepsis. Several fluid challenges have produced no sustained benefit. Pressor needs rise, skin becomes cooler, and oxygenation deteriorates. No reliable cardiac-output assessment has been obtained and the last echo preceded the deterioration. The resident proposes a larger bolus because earlier doses may have been insufficient. The surgeon recognizes that additional volume might worsen lung function and that low output, obstruction, or an unresolved source could now dominate. Bedside echocardiography and more reliable flow monitoring can be arranged without postponing urgent surgical review. The next decision must explain failure of the current strategy. The patient has moved beyond the initial trial of therapy without a coherent explanation for failure. The surgeon must preserve emergency support while gathering decision-relevant information, not require prolonged ineffective treatment before reconsidering the working diagnosis and anatomical source.

What is the best next step?

A. Reassess phenotype and flow while urgently revisiting source control

B. Increase bolus size without reassessing the mechanism

C. Use maintained MAP to exclude low output

D. Wait for lactate normalization before evaluating the source

Answer and option analysis

A - Best answer: Persistent nonresponse justifies mechanism-directed information and definitive-source review rather than repeated unchanged treatment.

B - Why this option does not fit: Repeated failure and intolerance weaken the rationale for more of the same therapy.

C - Why this option does not fit: Pressor-supported pressure does not guarantee adequate forward flow.

D - Why this option does not fit: This reverses the priority between treating the cause and observing its effects.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 021

A 34-year-old man with suspected unstable pelvic injury repeatedly loses his initial pressure response to emergency blood. Ultrasound shows no intraperitoneal fluid and hemoglobin is 12.5 g/dL. A binder is in place. He remains restless, with rising lactate and ongoing rapid product need. CT is available, but transport would remove him from the immediate resuscitation and control area. Angiography will take longer to mobilize than the operating team. The resident recommends waiting for CT because it may localize the source precisely. The surgeon must select the next route from actual physiology and local capability rather than a fixed preference for one technology. The institutional pelvic pathway can combine temporary stabilization, operative measures and later endovascular control. The team must use actual response and resource timing, with a fallback if the first control strategy is incomplete, rather than assume one modality is universally preferred.

Which plan is most appropriate?

A. Coordinate the fastest suitable hemorrhage-control pathway with ongoing blood-based support

B. Defer protocol activation until ten red-cell units have been given

C. Treat solely with escalating pressors until angiography is ready

D. Wait for routine CT because negative ultrasound establishes transport safety

Answer and option analysis

A - Best answer: Recurrent instability makes a routine imaging delay hazardous; pelvic control may require staged packing and/or embolization.

B - Why this option does not fit: A retrospective threshold should not delay anticipated major-hemorrhage support.

C - Why this option does not fit: Pressors do not replace missing blood or provide definitive hemostasis.

D - Why this option does not fit: The ultrasound does not exclude pelvic loss or show that recurrent instability has resolved.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 022

A 48-year-old woman develops brisk intraoperative bleeding during complex liver surgery. Only three red-cell units have been given, but the field is still filling, pressure responses are brief, and anesthesia expects rapid further need. Plasma and platelets will be delayed unless the blood bank receives a protocol activation. The resident argues that massive transfusion cannot be declared until ten red-cell units have been administered within 24 hours. The surgeon must decide whether the protocol is a retrospective diagnostic label or an organizational response to anticipated need. Surgical control is being pursued, and the team can communicate ongoing requirements and later deactivate the pathway explicitly. The blood bank can deliver an organized product sequence once activated and can adjust to laboratory results later. The surgeon must communicate anticipated need now and define how the pathway will be stopped, rather than use a historical definition as a prerequisite for logistics.

What should be done now?

A. Continue red cells alone until laboratory coagulopathy is extreme

B. Use crystalloid instead to postpone protocol activation

C. Activate the local major-hemorrhage protocol from the anticipated rapid ongoing need

D. Wait until the historical unit threshold is met

Answer and option analysis

A - Why this option does not fit: This can worsen the early imbalance between oxygen-carrying and hemostatic replacement.

B - Why this option does not fit: Crystalloid does not replace red-cell mass or adequate hemostatic components.

C - Best answer: Early activation organizes timely support before a retrospective unit threshold has already been reached.

D - Why this option does not fit: That delay can leave hemostatic support behind the rate of loss.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 023

A 73-year-old woman undergoes re-exploration for postoperative hemorrhage. A discrete arterial source is controlled, but raw surfaces continue to ooze. Temperature is 34.0 C, ionized calcium 0.85 mmol/L, fibrinogen 0.9 g/L, and pH 7.18. She has received red cells, less plasma than planned, and large crystalloid volumes. Her oxygen requirement is rising. The resident requests PCC solely because INR is elevated; another says the surgeon should keep searching for an artery and ignore the laboratory results. The surgeon must recognize overlapping surgical and physiological bleeding, choose targeted support, and decide whether prolonged reconstruction is tolerable during persistent derangement. There is no evidence of a specific anticoagulant indication for reversal in the history provided. The decision must therefore address the demonstrated deficiencies and physiological impairment while continuing inspection for a residual source, rather than convert a single prolonged test into a universal factor prescription.

Which approach is best?

A. Ignore coagulation because one vessel has been controlled

B. Reassess surgical hemostasis while correcting temperature, calcium and demonstrated coagulation deficits

C. Continue crystalloid until all visible oozing stops

D. Give PCC as the universal solution to the INR

Answer and option analysis

A - Why this option does not fit: Physiological failure can perpetuate hemorrhage after focal surgical control.

B - Best answer: Diffuse oozing and severe derangement support a coagulopathic contribution without proving every focal source is controlled.

C - Why this option does not fit: Additional dilution and cooling can worsen hemostasis and respiratory tolerance.

D - Why this option does not fit: PCC does not replace fibrinogen or platelets and needs a defined indication.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 024

A 60-year-old man remains actively bleeding during pelvic surgery. Fibrinogen is 1.0 g/L, platelet count 120 x 10^9/L, and a fibrin-specific viscoelastic measure is markedly reduced. He is already congested after substantial fluid and blood exposure. The resident proposes large additional plasma volumes as the sole response to the fibrinogen deficit. Cryoprecipitate and fibrinogen concentrate are available through the local protocol. The surgeon must choose a targeted hemostatic intervention while continuing mechanical control and avoiding the assumption that every low clot-strength result is primarily a platelet problem. Current calcium and temperature also require surveillance during ongoing transfusion. The available fibrin-specific assay is interpreted through a validated local algorithm rather than an arbitrary threshold copied from another machine. The surgeon should use the result alongside the measured concentration, current bleeding, and the limitations of further volume in this patient.

What is the best targeted response?

A. Give platelets alone because any weak clot means thrombocytopenia

B. Use plasma alone regardless of the volume required

C. Stop investigating bleeding because a biochemical cause is identified

D. Replace fibrinogen through the local cryoprecipitate or concentrate pathway

Answer and option analysis

A - Why this option does not fit: The platelet count and fibrin-specific result point to a fibrinogen contribution.

B - Why this option does not fit: This may be inefficient for a marked fibrinogen deficit and worsen congestion.

C - Why this option does not fit: A coagulation deficit can coexist with a persistent focal surgical source.

D - Best answer: The measured and functional fibrinogen deficit is clear; targeted replacement avoids relying on excessive plasma volume.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 025

A 42-year-old man arrives with major traumatic bleeding 3 hours and 40 minutes after injury. He has received some blood in transit and still requires urgent control and hemostatic support. A resident proposes starting routine trauma tranexamic acid because only 25 minutes have elapsed since hospital arrival. The exact injury time is reliable. The surgeon must distinguish the treatment window used in trauma evidence from the administrative time of admission, while ensuring that uncertainty about one adjunct does not delay the major hemorrhage pathway. There is no separate indication that would independently justify TXA under another specialist protocol. The team still needs immediate product coordination, warming, calcium surveillance and definitive control. Missing the routine trauma window changes the adjunct decision; it does not justify delay or withdrawal of the other interventions that directly address the active threat.

Which judgment is most appropriate?

A. Do not initiate routine trauma TXA based on the arrival clock; prioritize control and support

B. Stop blood support because the TXA window has passed

C. Wait for viscoelastic testing before continuing hemorrhage control

D. Start TXA because every actively bleeding patient benefits at any time

Answer and option analysis

A - Best answer: The usual trauma window is within three hours of injury, not three hours after hospital arrival.

B - Why this option does not fit: Missing an adjunct window does not remove the need for definitive resuscitation and control.

C - Why this option does not fit: Coagulation information accompanies, rather than replaces, urgent mechanical hemostasis.

D - Why this option does not fit: Benefit and risk depend on indication and timing, not bleeding alone.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 026

A 69-year-old man has major upper-GI bleeding with recurrent hypotension despite initial blood support. Endoscopic and interventional teams are coordinating source control. He has no trauma. A resident proposes routine high-dose tranexamic acid because the drug reduced bleeding deaths in traumatic injury, and suggests that this should precede endoscopy. Another clinician worries that changing the cause of bleeding may change both benefit and harm. The surgeon must choose an evidence-aware adjunct plan while maintaining the urgency of definitive control, airway assessment, and organized resuscitation. The patient has additional thrombotic risk from malignancy and immobility, but no separate specialist indication for TXA. The endoscopic pathway remains the appropriate route to investigate and treat the likely lesion, with rescue alternatives if it fails. Evidence from a different bleeding population cannot by itself establish that an additional drug will improve this patient's outcome or should precede control.

Which response is best?

A. Delay endoscopic control until the infusion is completed

B. Do not routinely extrapolate trauma TXA benefit to acute GI bleeding

C. Use TXA routinely because all hemorrhage shares the same evidence

D. Stop all transfusion because TXA is unsuitable

Answer and option analysis

A - Why this option does not fit: An adjunct must not postpone definitive management of ongoing bleeding.

B - Best answer: HALT-IT did not show a bleeding-death benefit and identified harms; definitive control and support remain priorities.

C - Why this option does not fit: A mechanistic similarity does not establish equal outcome benefit across indications.

D - Why this option does not fit: The lack of an adjunct indication does not remove the need for blood-based support.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 027

A 31-year-old man has major traumatic abdominal bleeding and a severe head injury with impaired consciousness. He briefly improves after emergency blood, but remains at risk of recurrent hypotension. The resident recommends maintaining systolic pressure around 80 mm Hg because this target is sometimes used before traumatic hemorrhage control. The surgeon must balance rebleeding risk against secondary cerebral injury and coordinate immediate operative control. The exact intracranial situation is not yet fully defined, and anesthesia is preparing support. The patient cannot be treated as an isolated penetrating torso injury without brain involvement simply because the abdominal source is the most visible hemorrhagic problem. Cerebral vulnerability makes the consequences of hypotension different from those of isolated torso injury. The operative and anesthesia teams need a joint goal based on both injuries, with serial neurological and circulatory assessment as far as the emergency allows.

What is the best pressure strategy?

A. Delay hemorrhage control until pressure is fully normalized

B. Avoid blood products because they could raise pressure

C. Use a brain-injury-aware perfusion goal while expediting hemorrhage control

D. Apply the same low systolic target regardless of head injury

Answer and option analysis

A - Why this option does not fit: Definitive control and appropriate support should proceed together.

B - Why this option does not fit: Appropriate blood support remains essential while pressure and bleeding are managed.

C - Best answer: Permissive-hypotension targets from selected trauma populations should not be applied indiscriminately when cerebral perfusion is threatened.

D - Why this option does not fit: The neurological injury materially changes the balance of pressure-related risks.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 028

A 55-year-old woman receives rapid transfusion during re-exploration for hemorrhage. She develops worsening hypotension and diffuse oozing despite progress in surgical control. Core temperature is low, total calcium appears only mildly reduced, but ionized calcium is 0.78 mmol/L. The resident argues that the total calcium value excludes a clinically important deficit. Another orders equal volumes of calcium chloride and calcium gluconate as though the preparations contain the same elemental amount. The surgeon must interpret the biologically active measurement, recognize the transfusion context, and ensure safe formulation-specific treatment while continuing to monitor temperature, coagulation, and the ongoing source. The team must also distinguish the dose that protects the hyperkalemic myocardium from a replacement regimen for hypocalcemia. A shared concentration label does not guarantee equivalent elemental delivery, and treatment must remain linked to the correct indication and preparation.

What is the most appropriate response?

A. Wait for a much lower total calcium before treatment

B. Assume calcium replacement alone will stop all bleeding

C. Use chloride and gluconate interchangeably by volume

D. Correct ionized hypocalcemia using the appropriate formulation-specific monitored protocol

Answer and option analysis

A - Why this option does not fit: Ionized calcium better reflects the relevant active fraction in this setting.

B - Why this option does not fit: Hemostasis also depends on source control, temperature and coagulation deficits.

C - Why this option does not fit: Their elemental calcium concentrations and access risks differ.

D - Best answer: Rapid citrate exposure can reduce active calcium; total calcium and equal formulation volumes can mislead.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 029

At a trauma review meeting, a team discusses a patient who received early balanced component packs and later targeted treatment. A resident states that PROPPR proved a universal mortality advantage of 1:1:1 over 1:1:2 at both primary time points, and proposes that every major surgical bleed must continue the same fixed ratio until discharge. Another notes that hemostasis and exsanguination outcomes differ from overall mortality and that treatment should adapt after control. The surgeon is asked to choose the interpretation that should guide the next protocol revision. The question concerns the evidence and its application, not whether early organized hemostatic support is valuable. The proposed protocol would otherwise affect patients with very different causes, timing and ongoing needs. A revision should preserve rapid availability while making the transition to current-result-guided therapy explicit, rather than treat an early ratio as an indefinite endpoint.

Which interpretation is most accurate?

A. The findings justify withholding early plasma and platelets entirely

B. Early balanced delivery helps organize support, but the trial did not establish universal mortality superiority

C. The absence of primary mortality significance proves all ratios are identical

D. The trial requires fixed-ratio transfusion after bleeding has stopped

Answer and option analysis

A - Why this option does not fit: That conclusion ignores early hemostatic support and the trial's relevant secondary outcomes.

B - Best answer: Primary overall mortality differences were not statistically significant; hemostasis and exsanguination findings support a more nuanced interpretation.

C - Why this option does not fit: Failure to demonstrate superiority is not proof of equivalence in every context.

D - Why this option does not fit: Postcontrol treatment should follow current deficits and avoid unnecessary products.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 030

A 64-year-old man has undergone successful control of intraoperative vascular bleeding. Pressure and perfusion improve, product need has stopped, and recent coagulation testing shows no major remaining deficit. He nevertheless continues receiving fixed-ratio packs because the protocol was never formally deactivated. Oxygen requirement rises and weight is increasing. The blood bank asks whether another cooler should be prepared. The resident recommends continuing until the next morning to prevent possible recurrence. The surgeon must distinguish vigilance for rebleeding from automatic ongoing transfusion, while preserving a clear plan for restarting support if loss recurs. There is no active hemorrhage or separate transfusion indication at present. The patient still needs surveillance for recurrent loss, potassium and calcium changes, respiratory complications and hemodynamic deterioration. Ending automatic packs is a deliberate treatment decision with restart criteria, not a declaration that the admission no longer requires close observation.

What is the best next action?

A. Stop all clinical and laboratory observation after deactivation

B. Continue packs solely because recurrence is possible

C. Replace every remaining pack with an equal crystalloid volume

D. Explicitly deactivate automatic packs and continue targeted surveillance

Answer and option analysis

A - Why this option does not fit: Deactivation does not establish immunity from rebleeding or complications.

B - Why this option does not fit: Potential recurrence warrants monitoring, not prophylactic uncontrolled product administration.

C - Why this option does not fit: No current deficit has been identified to justify that additional input.

D - Best answer: Once the indication ends, product delivery should be reassessed to limit transfusion harm and waste.

Review link: teaching Section 4. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 031

A 56-year-old woman is recovering after emergency abdominal surgery. She has received 6 L of saline. Pressure, interaction, and peripheral perfusion are improving and lactate falls to 1.7 mmol/L, but bicarbonate remains 17 mmol/L with chloride 116 mmol/L and no important albumin-adjusted gap elevation. Kidney function is under review and oxygen requirement is rising. A resident interprets the persistent base deficit as proof that resuscitation has failed and orders further saline. The surgeon must determine whether the biochemical pattern can reflect treatment exposure and whether additional volume has demonstrated benefit. There are no new focal abdominal findings, but continued surveillance remains necessary. The team has not demonstrated a new flow response that would justify more volume. The surgeon should compare the metabolic pattern with actual chloride exposure, lactate and organ trajectory before concluding that the remaining acid-base disturbance represents continued oxygen debt from inadequate filling.

What is the best interpretation?

A. Declare every remaining metabolic abnormality harmless

B. Treat the base deficit as a direct intravascular volume measurement

C. Give bicarbonate instead of evaluating the cause

D. Consider hyperchloremic acidosis and reassess fluid need rather than repeating saline automatically

Answer and option analysis

A - Why this option does not fit: Clinical recovery still requires ongoing assessment and verification of alternative causes.

B - Why this option does not fit: Base deficit has multiple causes and cannot prescribe a bolus alone.

C - Why this option does not fit: A buffer does not resolve the exposure, volume state or other possible contributors.

D - Best answer: The chloride-rich normal-gap pattern may persist despite improving perfusion and does not independently indicate more volume.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 032

A 70-year-old man has hypoalbuminemia after prolonged sepsis and abdominal surgery. Albumin is 1.9 g/dL, legs are edematous, oxygen requirement is increasing, and venous congestion is evident. Perfusion is currently improving on decreasing support. A resident proposes concentrated albumin solely to normalize the serum concentration and assumes its small bottle guarantees freedom from overload. Another proposes a synthetic starch to save infused volume. The surgeon must decide whether either product addresses a defined remaining problem rather than treating the laboratory label. There is no cirrhosis-specific albumin indication, large-volume paracentesis, or other established reason for replacement in the information provided. There is no evidence that the low serum concentration alone identifies an intravascular deficit that will respond beneficially to colloid. The decision must distinguish a possible selected volume indication from an attempt to correct a biomarker and make edema disappear.

Which approach is best?

A. Use starch because a smaller volume proves renal safety

B. Normalize albumin routinely regardless of perfusion and congestion

C. Assess the cause of edema and a specific treatment indication before prescribing colloid

D. Assume edema proves that forward circulation is adequate

Answer and option analysis

A - Why this option does not fit: Volume sparing does not establish safety and starch has important harm concerns.

B - Why this option does not fit: The serum number is not an independent mandate for colloid therapy.

C - Best answer: Low albumin alone does not establish outcome benefit from infusion; concentrated volume does not remove physiological risk.

D - Why this option does not fit: Interstitial accumulation can coexist with impaired effective arterial flow.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 033

A 45-year-old man with traumatic brain injury develops septic circulatory failure after surgery. The team plans initial crystalloid while treating infection and supporting pressure. A resident insists that the general preference for balanced crystalloid in sepsis must apply without exception; another suggests albumin to minimize the infusion volume. The neurocritical team is concerned about the brain-injury context and the characteristics of the available solutions. The surgeon must select a defensible approach that recognizes the population-specific exception rather than applying one product rule to every patient. Volume, perfusion, sodium, and intracranial concerns will be monitored alongside the chosen fluid. The available balanced product and albumin each have different composition and distribution characteristics. The choice therefore belongs within an integrated neurocritical and circulatory plan, rather than follow the usual sepsis preference without considering the specific cerebral injury and sodium context.

Which approach is most appropriate?

A. Follow the brain-injury-aware crystalloid pathway, commonly favoring 0.9% saline

B. Use albumin because reduced bottle volume guarantees neurological safety

C. Avoid all volume assessment because a pressor can replace fluid

D. Apply balanced crystalloid preference as an exception-free rule

Answer and option analysis

A - Best answer: Brain injury modifies routine sepsis fluid selection; albumin and general balanced-fluid assumptions should not be applied automatically.

B - Why this option does not fit: A volume-sparing claim does not establish safety in traumatic brain injury.

C - Why this option does not fit: Pressure support does not eliminate the need to evaluate effective circulating volume.

D - Why this option does not fit: Guidance specifically recognizes a brain-injury exception.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 034

A 63-year-old woman has profound vasodilatory hypotension after urgent abdominal source treatment. Appropriate initial fluid assessment is underway, but pressure remains dangerously low. A functioning peripheral vein is available with trained nursing surveillance, while central access will take time. The resident recommends withholding norepinephrine until a central catheter has been inserted, despite worsening mentation and mottling. Another proposes an unmonitored peripheral infusion of indefinite duration. The surgeon must choose a practical immediate support strategy and preserve safeguards around drug concentration, site assessment, access planning, and repeated perfusion review. The hospital has an established peripheral-vasopressor protocol for this situation. The catheter site can be observed directly, and the infusion concentration is standardized by protocol. The surgeon must arrange escalation of access and respond promptly to suspected extravasation while avoiding a preventable delay in supporting a severely inadequate arterial pressure.

What is the best plan?

A. Treat central access as proof that the underlying source is controlled

B. Delay all vasopressor treatment until central access is complete

C. Start protocol-based monitored peripheral support as a bridge while arranging ongoing access

D. Use peripheral infusion indefinitely without site inspection

Answer and option analysis

A - Why this option does not fit: Access enables therapy but does not establish definitive disease treatment.

B - Why this option does not fit: The delay can prolong severe hypotension and organ hypoperfusion.

C - Best answer: Urgent pressure support need not await central access when appropriate peripheral safeguards and expertise are available.

D - Why this option does not fit: Extravasation and delivery failure require active surveillance and an access plan.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 035

A 68-year-old man had been improving after surgery for perforation but now needs rapidly increasing norepinephrine. He is cooler, urine output falls, and a new abdominal drain becomes cloudy. The infusion pump and catheter have not yet been checked. A resident proposes adding another vasopressor as the sole response, without reopening the diagnosis because septic shock was already established. The surgeon must recognize that delivery failure, recurrent infection, bleeding, obstruction, or myocardial dysfunction can change the current physiology. Bedside examination, waveform review, targeted echo, and urgent abdominal reassessment are feasible in parallel with maintaining necessary support. The patient is not stable enough for an unattended transfer. New requirements could reflect disease progression or a technical delivery problem, and both can be assessed quickly. Additional support may be needed during the assessment, but it should accompany a renewed explanation and a specific plan for definitive treatment.

What is the best next approach?

A. Give fluid solely because pressor dose increased

B. Escalate drugs alone because the diagnostic label is already known

C. Maintain needed support while checking delivery and reassessing mechanism and source

D. Stop norepinephrine abruptly to reveal the untreated pressure

Answer and option analysis

A - Why this option does not fit: Increased dose does not by itself establish volume responsiveness or tolerance.

B - Why this option does not fit: The established label cannot explain every new deterioration without reassessment.

C - Best answer: Rising requirements are an alarm for failed treatment or a new process, not merely a request for another drug.

D - Why this option does not fit: This can worsen organ perfusion and is unnecessary for diagnostic evaluation.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 036

A 74-year-old woman remains in active shock after complex abdominal surgery. She is markedly edematous and several liters positive, but pressor doses are escalating, the source is not controlled, and perfusion remains poor. A resident proposes aggressive diuresis to make the balance negative before further intervention. Another argues that edema should never influence management. The surgeon must distinguish a real treatment burden from readiness for active removal, while arranging definitive source care and reviewing unnecessary ongoing inputs. Kidney function is deteriorating and no urgent dialysis indication has yet been established. The question concerns the immediate phase of care rather than a later recovery strategy. The clinical team can immediately stop duplicate and unnecessary infusions without assuming that the patient is ready for active removal. These are different decisions, and the presence of harmful accumulation should guide vigilance while urgent source control and effective circulation remain unresolved.

Which plan is best?

A. Start extracorporeal removal solely to normalize recorded balance

B. Address active shock and the source while stopping unnecessary input; reassess removal readiness

C. Ignore all input because edema is inevitable in sepsis

D. Remove fluid aggressively solely from the balance chart

Answer and option analysis

A - Why this option does not fit: Renal replacement requires appropriate clinical indications and a tolerance assessment.

B - Best answer: Harmful accumulation matters, but aggressive removal during worsening shock may further impair perfusion.

C - Why this option does not fit: Preventable carriers and duplicate orders can still contribute substantial harm.

D - Why this option does not fit: A positive balance does not establish that acute resuscitation has ended.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 037

A stable 70-kg man is nil by mouth after surgery but can take 500 mL enterally. The team selects a provisional total water plan of 1750 mL/day. Antibiotic carriers provide 450 mL and flushes another 200 mL. There are no abnormal GI losses, sodium is stable, and there is no shock. A resident prescribes an additional 1750 mL as IV maintenance because the calculated daily requirement has that value. Another argues that carrier fluids do not count because they are medications. The surgeon must determine the remaining water budget before separately auditing electrolyte and glucose composition. The plan will be reassessed when oral intake changes. This is a calculation of water remaining after current provision, not a complete bag prescription. Sodium, potassium, glucose and other components still need a separate audit, and the entire plan must change if oral intake or losses differ from the estimate.

What additional IV maintenance water follows from this provisional account?

A. 600 mL/day before composition and clinical adjustment

B. 1050 mL/day because medication carriers are excluded

C. 1750 mL/day regardless of other intake

D. Zero because any enteral intake eliminates maintenance need

Answer and option analysis

A - Best answer: 1750 minus 500 minus 450 minus 200 equals 600; all existing sources count toward water provision.

B - Why this option does not fit: Medication diluent water is physiologically relevant and must be counted.

C - Why this option does not fit: That treats total need as an extra infusion and double-counts existing provision.

D - Why this option does not fit: The available enteral volume does not meet the provisional remaining requirement.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 038

A 51-year-old woman with severe obesity is admitted for elective surgery. She weighs 140 kg, has no abnormal losses, and will be reassessed for early oral intake. The resident applies a weight-based maintenance rule to actual weight and leaves the resulting infusion unchanged for several days. Medication carriers and feeds are not included in the account. By the second day she gains weight and requires oxygen. The surgeon must decide how obesity, current intake, and tolerance affect the maintenance prescription. She has no current shock that would justify treating the ordinary daily estimate as a rapid resuscitation requirement, and exact ideal weight can be established from her height. The patient's current actual weight includes substantial adipose mass, which changes its relationship to total water and ordinary requirements. A safe plan also needs to recognize that the reason for IV provision may disappear once adequate oral or enteral intake resumes.

Which approach is best?

A. Use actual weight without adjustment because water distributes equally into adipose tissue

B. Continue the same infusion until discharge regardless of oral intake

C. Treat new oxygen need with another routine bolus

D. Individualize using an appropriate ideal-weight method and subtract all other provision

Answer and option analysis

A - Why this option does not fit: Adiposity changes the relation between body mass and water requirements.

B - Why this option does not fit: Maintenance need must change with intake, losses and clinical state.

C - Why this option does not fit: Emerging intolerance requires assessment rather than automatic expansion.

D - Best answer: Actual-weight multiplication in severe obesity and an unchanged inherited rate can overestimate ordinary needs.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 039

A 79-year-old woman with cardiac disease has persistent nasogastric output after bowel surgery. She is edematous but has low chloride, potassium 3.0 mmol/L, bicarbonate 35 mmol/L, and worsening renal function. The standing infusion is labeled maintenance but its relationship to measured gastric replacement is unclear. A resident proposes stopping every fluid because of edema; another proposes rapidly infusing the entire preceding day's output without assessing what has already been replaced. The surgeon must separate ordinary needs, accumulated excess, ongoing chloride-rich losses, and effective filling. Her oxygen requirement is rising, so the volume and rate of any intervention have real consequences. Current heart and renal function make a rapid volume-based response risky even when a specific chloride deficit is plausible. The surgeon must specify the remaining deficit, ongoing loss and rate of delivery with close assessment of oxygenation and effective circulation.

What is the best prescribing strategy?

A. Stop all replacement because edema excludes electrolyte depletion

B. Treat alkalosis solely with additional glucose water

C. Rapidly replace all historical output without reviewing earlier intake

D. Reconstruct the account and separately plan tolerated gastric replacement and maintenance

Answer and option analysis

A - Why this option does not fit: Interstitial excess does not rule out clinically important chloride or potassium loss.

B - Why this option does not fit: This does not directly address a chloride-depletion mechanism and may worsen sodium-water imbalance.

C - Why this option does not fit: This can double-count replacement and worsen cardiorespiratory burden.

D - Best answer: Specific chloride/potassium deficits can coexist with edema; composition, prior replacement and current tolerance must guide treatment.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 040

A 49-year-old man is readmitted after ileostomy creation with 2.7 L/day watery output, postural dizziness, weight loss, and AKI. Serum sodium is 138 mmol/L. He drinks large quantities of plain water and believes this excludes dehydration. Magnesium is low and potassium is borderline reduced. A resident proposes routine maintenance only, while another recommends unrestricted additional water. The surgeon must interpret a normal concentration against a clear negative balance and evaluate the causes of high output, including anatomy, drugs, infection, or obstruction. The patient is awake but symptomatic on standing and requires a plan for both immediate correction and sustainable outpatient intake. The recorded oral volume does not show the amount of sodium absorbed or lost, and creatinine change supports a clinically consequential imbalance. The surgeon must arrange a plan the patient can sustain after IV treatment rather than provide only an instruction to increase water.

Which approach is most appropriate?

A. Assess sodium-water depletion and provide source-specific replacement with a stoma pathway

B. Ignore magnesium until creatinine normalizes

C. Exclude depletion because sodium is normal

D. Treat all output with unlimited plain water alone

Answer and option analysis

A - Best answer: Normal sodium does not exclude extracellular depletion; plain water is not equivalent to replacement of sodium-rich intestinal losses.

B - Why this option does not fit: Associated deficiencies affect symptoms and replacement response and need safe coordinated treatment.

C - Why this option does not fit: Serum concentration does not measure total-body sodium or circulating volume.

D - Why this option does not fit: Hypotonic intake may be ineffective or worsen net sodium loss in high-output settings.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 041

A 62-year-old woman has hypotension and poor perfusion after substantial nonhemorrhagic GI losses. Sodium is elevated, and the team identifies both extracellular volume depletion and a water deficit. A resident proposes using only 5% dextrose water as the initial rapid circulatory rescue because it will eventually distribute throughout total body water. She is drowsy, with delayed capillary refill and a falling pressure. The surgeon must sequence restoration of effective circulation and subsequent water correction rather than choose one solution for both purposes without considering distribution. There is no current pulmonary congestion, but repeated assessment will be required as volume is restored. The patient needs both urgent perfusion support and a subsequent measured sodium trajectory. The first intervention should be selected for its immediate circulatory effect; later fluid composition and rate should address the remaining water problem as renal handling changes.

What is the best sequence?

A. Use dextrose water alone because total-body distribution guarantees plasma rescue

B. Restore threatened circulation appropriately, then correct water deficit with monitored adjustment

C. Ignore hypernatremia after pressure improves

D. Infuse the entire calculated water deficit immediately

Answer and option analysis

A - Why this option does not fit: Most equilibrated water leaves the plasma compartment.

B - Best answer: Electrolyte-free water provides little sustained plasma expansion after distribution and should not be the sole rapid rescue fluid.

C - Why this option does not fit: The remaining water deficit and ongoing losses still require treatment.

D - Why this option does not fit: The equation does not determine a safe correction rate or include changing losses.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 042

A 65-year-old man is stable after surgery without drains or unusual losses. A resident calculates routine maintenance, then adds his entire measured urine output and an extra estimated ordinary insensible loss as replacement. Drug carriers are ignored. Over two days weight rises and ankle edema appears. Kidney function was initially normal, and the resident explains that replacing every output must produce neutral balance. The surgeon must identify whether the method has counted expected physiological losses more than once. There is no polyuric disorder, fever, large open wound, or other unusual condition that would independently require a separate cause-specific replacement strategy at this time. The maintenance estimate was intended to cover expected physiological output, so its components must be understood before adding new terms. A genuine abnormal polyuria or extra loss would change the calculation, but it has not been established in this case.

Which correction to the plan is best?

A. Avoid adding ordinary outputs already included in the maintenance estimate

B. Stop monitoring because normal kidneys prevent every fluid error

C. Continue because every urine milliliter always needs separate replacement

D. Ignore drug carriers because their volume is prescribed for another purpose

Answer and option analysis

A - Best answer: Routine maintenance incorporates expected urine and insensible losses; separate additions can create systematic overprescription.

B - Why this option does not fit: Renal adaptation is not a guarantee against iatrogenic accumulation.

C - Why this option does not fit: Ordinary urine output is not an abnormal drain loss requiring automatic matching.

D - Why this option does not fit: Their water and electrolyte content still enters the patient.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 043

A 76-year-old man develops oliguric AKI after abdominal surgery. A pre-existing maintenance order contains potassium based on a weight-derived daily estimate. Potassium has risen to 5.8 mmol/L, and he is also receiving potassium-containing replacement for a drain whose output has nearly stopped. The resident wants to continue both orders because the original calculation was correct. The surgeon must recognize that maintenance references describe a starting plan, not a requirement that survives changing clearance and loss. The ECG is being assessed and repeat chemistry is available. There is no rationale to treat the historical output as though it remains unchanged. The current serum result is not yet at the most extreme range, providing an opportunity to prevent progression through medication and infusion reconciliation. Review must occur now rather than after an old theoretically reasonable prescription creates a new emergency.

What should happen now?

A. Wait for an arrhythmia before reviewing potassium input

B. Stop inappropriate potassium provision and reassess the current electrolyte and renal plan

C. Continue the original dose because daily potassium needs are fixed

D. Continue drain replacement at the previous output rate

Answer and option analysis

A - Why this option does not fit: Preventing progression is part of appropriate hyperkalemia management.

B - Best answer: Falling excretion and disappearing losses invalidate the previous replacement and maintenance assumptions.

C - Why this option does not fit: Renal clearance and serum concentration materially alter safe provision.

D - Why this option does not fit: Replacement must follow current measured losses rather than an obsolete value.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 044

A stable 70-kg patient has a small remaining daily water budget after feeds and carriers are counted, but substantial residual potassium and glucose needs. A resident tries to force all deficits into the remaining small infusion volume by adding concentrated electrolytes at the bedside. The patient has moderate kidney impairment, and the ward lacks monitoring for a highly concentrated infusion. The surgeon must distinguish arithmetic completeness from a safe deliverable prescription. Standard premixed options, enteral provision, and pharmacy review are available. No severe symptomatic deficit requires bypassing those safeguards, but the next order still needs to address each actual requirement rather than omit the problem. The ward can provide standard formulations within approved limits and arrange an alternate route for residual needs. The physician's task is to reconcile safety and requirements, not treat mathematical completion as permission for an improvised preparation.

Which approach is best?

A. Ignore residual electrolytes because the water budget is small

B. Concentrate all electrolytes until every calculated target fits

C. Add potassium by IV push to avoid extra fluid

D. Reconcile needs with standardized preparations and pharmacy-supported delivery

Answer and option analysis

A - Why this option does not fit: A limited water allowance does not eliminate real electrolyte needs.

B - Why this option does not fit: Dose, concentration, compatibility and clearance impose separate safety constraints.

C - Why this option does not fit: IV potassium push is unsafe and not an acceptable volume-saving method.

D - Best answer: A mathematical residual does not justify unsafe concentration, mixing or access; route and monitoring must fit the prescription.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 045

A malnourished 58-year-old woman has been nil by mouth for several days after surgery. Her maintenance infusion provides 75 g glucose daily, and a resident describes this as adequate nutritional support because it limits starvation ketosis. She has lost muscle mass and will soon begin enteral feeding. Baseline potassium, magnesium, and phosphate are not yet checked. The surgeon must distinguish a limited glucose provision from energy, protein, vitamin and electrolyte needs, and recognize that restarting nutrition can create new physiological risk. There is no current shock, but the duration of poor intake and weight loss make the feeding plan a clinical decision requiring coordination. Even the maintenance carbohydrate contributes to the total exposure that matters during refeeding. A nutrition plan must include all sources, risk-adapted advancement and biochemical monitoring rather than assume that tolerating a small glucose infusion proves nutritional adequacy.

What is the best interpretation?

A. Treat 75 g glucose as complete daily nutrition

B. Maintenance glucose does not meet nutritional needs; assess refeeding risk and plan support

C. Advance full feeding without electrolyte assessment because maintenance was tolerated

D. Withhold all nutrition indefinitely to avoid electrolyte shifts

Answer and option analysis

A - Why this option does not fit: It omits essential protein and other nutrient requirements.

B - Best answer: Limited carbohydrate can reduce ketosis without supplying adequate calories, protein or micronutrients, and may contribute to refeeding shifts.

C - Why this option does not fit: Tolerance of small carbohydrate exposure does not exclude refeeding vulnerability.

D - Why this option does not fit: Risk requires a controlled nutrition and monitoring plan, not indefinite starvation.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 046

A 43-year-old woman receives hypotonic maintenance fluid after elective surgery while experiencing persistent nausea and pain. Over the next day sodium falls from 138 to 124 mmol/L. She is mildly confused, and the chart includes additional water from medications. The urine is concentrated. A resident labels renal sodium failure and proposes more of the same fluid because urine output is low. The surgeon must assess symptoms and tonicity, recognize postoperative antidiuretic stimuli, and review all intake. She has no documented severe neurological emergency yet, but the trajectory is active and requires closer monitoring than the original once-daily laboratory schedule. Pain and nausea can maintain antidiuresis even without a structural endocrine disorder. The surgeon must decide whether the current neurological change is attributable to sodium, whether severe rescue is needed, and what intake and monitoring changes are necessary immediately.

What is the best next approach?

A. Diagnose SIAD solely from concentrated urine without excluding mimics

B. Wait for a seizure before changing monitoring

C. Reassess symptomatic hypotonic hyponatremia and stop inappropriate water exposure

D. Continue the same fluid solely to increase urine output

Answer and option analysis

A - Why this option does not fit: Volume depletion, drugs, cortisol deficiency and other causes must be assessed.

B - Why this option does not fit: Active symptoms and a substantial fall require prompt reassessment before severe deterioration.

C - Best answer: Postoperative antidiuresis plus hypotonic input can lower sodium; low urine output is not a mandate for more water.

D - Why this option does not fit: This may aggravate water retention and hyponatremia.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 047

A 55-year-old man presents after surgery with dehydration, glucose 650 mg/dL, and measured sodium 124 mmol/L. Osmolality is elevated rather than low. He is alert but weak and has osmotic urine losses. The resident calls this severe hypotonic hyponatremia and orders hypertonic saline solely from the sodium concentration. The surgeon must distinguish a glucose-related water shift from true hypotonic disease, while recognizing that substantial total water and electrolyte losses may coexist. There is no seizure or evidence of a separate hyponatremic encephalopathy. Appropriate serial glucose, sodium, potassium and osmolality measurements are available through the hyperglycemic-emergency pathway. The measured sodium may rise during glucose treatment as water moves back intracellularly, even while total deficits remain. The team therefore needs an integrated hyperglycemic-emergency plan with serial osmolality and electrolytes rather than interpret every sodium change as an independent treatment failure.

Which interpretation best guides treatment?

A. Manage the hyperglycemic hypertonic state with serial sodium and volume assessment

B. Ignore potassium until glucose is normal

C. Assume no total-body water deficit because water moved out of cells

D. Give hypertonic saline simply because sodium is below 125

Answer and option analysis

A - Best answer: Glucose can lower measured sodium through translocation; the measured concentration alone does not justify hypotonic-hyponatremia rescue.

B - Why this option does not fit: Insulin and changing renal losses can rapidly alter potassium and require early monitoring.

C - Why this option does not fit: Osmotic diuresis can cause major external losses despite the transcellular shift.

D - Why this option does not fit: Tonicity and symptoms must be established before selecting sodium-specific rescue.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 048

A 67-year-old woman has hypotonic hyponatremia after surgery with nausea, low pressure, and concentrated urine. She recently stopped chronic glucocorticoids. Potassium is normal. A resident calls the pattern definitive SIAD because urine remains concentrated and argues that normal potassium excludes adrenal insufficiency. The surgeon must consider whether impaired cortisol reserve could mimic the biochemical pattern and whether hypotension changes treatment urgency. There are no major GI losses, but fluid status remains difficult to determine clinically. The patient is becoming less interactive, so endocrine investigation must not become a reason to postpone necessary emergency support if adrenal crisis is suspected. A complete volume assessment is difficult, and the medication history supplies an additional mechanistic clue. The clinician should preserve appropriate urgent treatment when crisis is suspected while using endocrine investigations to refine the diagnosis rather than delay it.

Which approach is most appropriate?

A. Assess cortisol deficiency and other mimics before accepting SIAD

B. Diagnose SIAD from urine osmolality alone

C. Use water restriction as the sole response to possible adrenal crisis

D. Exclude adrenal disease because potassium is normal

Answer and option analysis

A - Best answer: Secondary adrenal insufficiency can cause hyponatremia without hyperkalemia; the medication history and hypotension matter.

B - Why this option does not fit: Concentrated urine shows antidiuresis but does not establish its cause.

C - Why this option does not fit: Potential circulatory endocrine failure requires urgent cause-directed assessment and treatment.

D - Why this option does not fit: Normal potassium does not exclude secondary cortisol deficiency.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 049

A 59-year-old woman becomes obtunded and has a generalized seizure after prolonged nausea, low-solute intake, and postoperative hypotonic fluids. Sodium is 114 mmol/L and measured osmolality is low. The airway and seizure are being managed, and senior help is present. A resident recommends fluid restriction alone because rapid sodium correction can cause demyelination. Another proposes normalizing sodium immediately to 140. The surgeon must distinguish urgent partial neurological rescue from the later maximum correction limit, while reviewing duration, risk factors, urine output and all ongoing infusions. Hypertonic saline and frequent sodium measurements can be provided in a monitored setting. The danger of later overcorrection does not negate the immediate neurological threat. Rescue and subsequent controlled treatment are separate phases with different endpoints, and the surgeon must ensure that a safe monitored pathway connects them without pursuing immediate normalization.

What is the best initial sodium strategy?

A. Raise sodium to normal immediately with repeated unrestricted boluses

B. Delay treatment until the full cause is proven

C. Use protocol-based hypertonic rescue for a limited initial rise, then control further correction

D. Use restriction alone until the next daily blood sample

Answer and option analysis

A - Why this option does not fit: Normalization is not the emergency goal and risks excessive correction.

B - Why this option does not fit: Etiologic testing should not postpone lifesaving symptomatic rescue.

C - Best answer: Severe neurological symptoms warrant prompt partial correction rather than restriction alone or immediate normalization.

D - Why this option does not fit: This is inadequate rescue for severe hyponatremic neurological compromise.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 050

A malnourished 66-year-old man has sodium 111 mmol/L and potassium 2.8 mmol/L after prolonged GI losses. Symptoms improve after appropriate initial treatment. Sodium reaches 116, then rises to 120 within eight hours of baseline as urine output suddenly increases and becomes dilute. Hypertonic saline has already been stopped. The resident assumes correction is now self-limited because no sodium infusion continues. The surgeon recognizes uncertain chronicity and several demyelination risk factors. Potassium replacement and other inputs are still active. The next decision must address the current trajectory before delayed neurological injury appears, rather than judge safety solely from the improvement in mental status. There is no assurance that spontaneous water diuresis will stop when the intended initial rise has been achieved. The treatment team must act on the measured rate and the patient's vulnerability rather than wait for an overt neurological complication that may be delayed.

What is the best response?

A. Urgently control overcorrection with specialist-guided measures and frequent sodium/urine review

B. Continue saline until sodium is normal because symptoms improved

C. Wait because stopping hypertonic saline guarantees safe correction

D. Ignore potassium replacement in sodium accounting

Answer and option analysis

A - Best answer: A rising sodium during aquaresis can continue without saline; this high-risk trajectory needs immediate intervention.

B - Why this option does not fit: Early symptomatic improvement does not remove demyelination risk.

C - Why this option does not fit: Renal free-water loss can remain a powerful correction mechanism.

D - Why this option does not fit: Potassium repletion can contribute to the sodium rise.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 051

A 63-year-old woman with chronic severe hyponatremia is undergoing controlled correction. Sodium begins at 115 mmol/L and potassium at 2.4 mmol/L. The team gives substantial potassium replacement while a separate clinician calculates a sodium-only infusion plan. Over several hours sodium rises more than anticipated despite stable saline delivery. Urine output has also begun to increase. The resident says potassium cannot affect sodium because the laboratory reports different ions. The surgeon must coordinate two necessary treatments and avoid overcorrection without abandoning the arrhythmia risk of hypokalemia. Frequent chemistry and senior renal/endocrine review are available, and the total correction trajectory can still be adjusted. Several clinicians are prescribing in parallel, creating a risk that each assumes the other's treatment has no effect on sodium. A coordinated order set and named reassessment responsibility are needed so necessary potassium correction and sodium safety remain part of one plan.

Which statement should guide the revised plan?

A. Potassium has no relevance to sodium correction safety

B. Trust the original formula over repeated measured values

C. Potassium replacement and changing water excretion must be included in sodium correction assessment

D. Stop all potassium permanently despite severe deficiency

Answer and option analysis

A - Why this option does not fit: This ignores the physiological coupling and can underestimate the rise.

B - Why this option does not fit: Changing physiology requires adjustment from the observed trajectory.

C - Best answer: Exchangeable cations and body water jointly influence sodium concentration; isolated sodium-infusion arithmetic is incomplete.

D - Why this option does not fit: A necessary treatment should be coordinated safely rather than abandoned indefinitely.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 052

A 78-year-old woman unable to drink has sodium 160 mmol/L after several days of poor intake and ongoing water losses. Estimated total body water is 30 L. The resident calculates a 4.29-L deficit relative to sodium 140 and orders that entire volume rapidly, assuming the formula supplies the rate. She is not in shock but has cardiac disease and unknown-duration hypernatremia. Urine output and ongoing losses are not included in the calculation. The surgeon must distinguish a starting estimate from the amount and speed that can safely be delivered, and establish a plan that changes with serial sodium and tolerance. The patient also needs assessment of access to drinking water, the cause of ongoing loss and the chosen total-body-water estimate. Serial observed response can differ from the prediction, making the next sample and tolerance assessment more important than completing a calculated bag volume.

What is the best use of the calculation?

A. Ignore all ongoing urine loss until the deficit is replaced

B. Infuse the full amount immediately because the arithmetic is correct

C. Use the value as a direct measure of plasma-volume deficit

D. Use it as an approximate deficit within a monitored staged replacement plan

Answer and option analysis

A - Why this option does not fit: Current losses can materially change both need and sodium trajectory.

B - Why this option does not fit: A correct estimate does not establish safe delivery timing.

C - Why this option does not fit: A free-water estimate is not an intravascular filling measurement.

D - Best answer: The formula excludes ongoing losses and does not determine correction rate, distribution or cardiac tolerance.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 053

A 47-year-old man with central vasopressin deficiency develops hypernatremia after missing desmopressin while unable to drink. He produces large volumes of dilute urine and is initially volume depleted. After appropriate support and specialist-directed desmopressin, urine output falls sharply. A resident continues replacing urine at the earlier high hourly rate and also continues a separate free-water infusion. Sodium begins falling faster than planned. The surgeon must recognize that treatment changed the loss mechanism, not simply the numerical sodium value. Clinical perfusion has improved, but ongoing monitoring and intake reconciliation are essential to avoid overshooting into a new water-excess problem. The earlier prescription was tied to an excretion rate that no longer applies. The team needs a new integrated plan for water and antidiuretic therapy, with clear review intervals, rather than let two independently reasonable orders continue together after the physiology changes.

What is the best next action?

A. Assume desmopressin eliminates the need for sodium surveillance

B. Give additional saline solely to reverse every sodium fall

C. Recalculate current water delivery and replacement from the new urine and sodium trajectory

D. Continue the original hourly rate until the old deficit estimate is fully delivered

Answer and option analysis

A - Why this option does not fit: Combined water and antidiuretic treatment can produce rapid changes.

B - Why this option does not fit: The correct response depends on rate, volume status and the intended correction plan.

C - Best answer: Once antidiuresis takes effect, the prior high-loss replacement rate can become excessive.

D - Why this option does not fit: The estimate does not override the abrupt change in renal excretion.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 054

A 72-year-old man has severe diarrhea, sodium 157 mmol/L, and marked circulatory depletion. Pressure is 70/40 mm Hg with confusion and delayed capillary refill. The resident is concerned about hypernatremia and refuses any isotonic resuscitation because its sodium concentration is above normal serum values. Another wants to correct the entire water deficit before supporting circulation. The surgeon must address the immediate perfusion threat while planning subsequent controlled correction and investigation of ongoing losses. There is no pulmonary edema, but repeated assessment is available and important because rapid changes in renal function may follow restoration of circulation. The immediate circulation and the later sodium target must not be treated as competing alternatives. Once shock improves, continued isotonic input should be reconsidered so the remaining free-water deficit is corrected through an individualized and monitored plan rather than ignored.

Which sequence is best?

A. Assume isotonic treatment alone guarantees complete water correction

B. Withhold circulatory support until sodium is normal

C. Use only a slow maintenance infusion despite profound shock

D. Restore threatened circulation first with appropriate resuscitation, then adjust water correction

Answer and option analysis

A - Why this option does not fit: A substantial free-water deficit and ongoing losses can remain afterward.

B - Why this option does not fit: This allows ongoing organ hypoperfusion while treating a secondary numerical target.

C - Why this option does not fit: Maintenance is not adequate emergency resuscitation.

D - Best answer: Immediate shock management and subsequent free-water correction address different urgent physiological needs.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 055

A 69-year-old woman with oliguric AKI after surgery has a confirmed potassium of 6.9 mmol/L. The ECG has no dramatic abnormality, but she is weak and potassium has risen rapidly. Potassium-containing fluids and an implicated drug remain prescribed. A resident recommends observation until ECG changes appear. Another proposes calcium alone as the complete treatment. The surgeon must identify the need for monitored potassium-lowering treatment, assess the appropriate cardiac-protection indication, and arrange elimination when renal clearance is inadequate. Repeat potassium and glucose surveillance can be provided, while renal and critical-care teams are contacted promptly. The team should remove inappropriate sources while planning repeat measurements, glucose surveillance and the safest elimination strategy. The absence of a dramatic tracing does not turn a confirmed rapidly rising severe potassium value into a routine daily-review problem.

What is the best approach?

A. Treat severe hyperkalemia urgently despite the nondiagnostic ECG and plan definitive clearance

B. Wait for ECG toxicity before treating a confirmed severe value

C. Use calcium alone to remove potassium from the body

D. Continue potassium input because it was a maintenance requirement

Answer and option analysis

A - Best answer: A normal ECG does not guarantee safety, and shifting measures do not replace elimination or repeat monitoring.

B - Why this option does not fit: Potential lethal progression is not excluded by the current tracing.

C - Why this option does not fit: Calcium stabilizes excitability when indicated but does not lower total potassium.

D - Why this option does not fit: The current clearance and concentration invalidate that prescription.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 056

A 61-year-old man with advanced AKI has potassium 7.0 mmol/L and compatible ECG changes. He receives appropriate calcium and insulin-glucose treatment, and potassium decreases to 5.6 after an hour. The resident declares the emergency resolved and cancels repeat testing and renal review. Urine output remains minimal, ongoing cell breakdown is suspected, and no effective elimination plan has been established. His glucose is falling, although the initial insulin treatment was accompanied by glucose. The surgeon must interpret the distinction between intracellular redistribution and potassium removal and protect against delayed treatment complications while arranging further management. The initial interventions have bought time rather than established durable recovery. The surgeon must ensure that the next phase deals with the total potassium burden, continuing release and low excretion while detecting delayed adverse effects of the temporary shifting treatment.

Which plan is most appropriate?

A. Continue potassium and glucose surveillance and arrange adequate elimination

B. Give more insulin automatically without checking glucose and trajectory

C. Repeat calcium as the sole potassium-elimination strategy

D. Cancel monitoring because one repeat value improved

Answer and option analysis

A - Best answer: Insulin has a temporary shifting effect; persistent low clearance creates rebound risk and hypoglycemia may be delayed.

B - Why this option does not fit: Repeat therapy requires reassessment and hypoglycemia prevention.

C - Why this option does not fit: Calcium does not remove potassium.

D - Why this option does not fit: A single early reduction cannot establish durable control.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 057

A 58-year-old woman develops severe hyperkalemia with QRS widening after surgery. The team prepares IV calcium under the emergency pathway. A junior clinician substitutes the same volume of 10% calcium gluconate for a prescribed volume of 10% calcium chloride, assuming both names indicate identical elemental delivery. The patient has fragile peripheral access and requires prompt monitored treatment. The surgeon must ensure that formulation-specific dosing and access risks are respected without postponing cardiac protection. Potassium shifting and elimination are being prepared in parallel, because the team recognizes that calcium does not correct the underlying total-body potassium excess. The dose should be checked against the actual stocked concentration and emergency indication. The team also needs an explicit reassessment point for ECG response, ongoing potassium treatment and access safety, rather than assume one calcium administration ends the emergency.

What is the most appropriate correction?

A. Treat the preparations as interchangeable by volume

B. Use the correct formulation-specific dose and access precautions under the emergency protocol

C. Delay all cardiac protection until potassium is removed

D. Stop elimination measures once calcium has been given

Answer and option analysis

A - Why this option does not fit: This can produce inadequate or inappropriate calcium delivery.

B - Best answer: Equal volumes of chloride and gluconate are not elemental-calcium equivalents and have different tissue-injury considerations.

C - Why this option does not fit: Cardiac stabilization and potassium-lowering measures should proceed in the appropriate sequence.

D - Why this option does not fit: Membrane protection does not reduce potassium burden.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 058

A 44-year-old man recovering uneventfully from elective surgery has an unexpected potassium of 6.2 mmol/L on a visibly hemolyzed sample. Previous values and kidney function were normal, he has no relevant symptoms, and the ECG is unremarkable. There is no potassium infusion or cell-injury history. A resident prepares insulin-glucose without repeating the sample, while another wants to ignore the result permanently. The surgeon must decide how the pretest context and sample quality affect immediate verification without adopting a policy that would delay treatment in a genuinely high-risk severe case. A carefully collected repeat sample can be obtained promptly at the bedside. The context lowers the probability of a true emergency but does not make verification optional. The appropriate approach should avoid both unnecessary insulin-related harm and an unrecognized genuine elevation, with prompt results and an escalation plan if the repeat is confirmed.

What is the best next step?

A. Treat every hemolyzed result as proven severe hyperkalemia

B. Promptly repeat a reliable sample and assess the patient while maintaining appropriate vigilance

C. Use a normal ECG to exclude hyperkalemia definitively

D. Ignore the result and never recheck

Answer and option analysis

A - Why this option does not fit: Unnecessary insulin can cause harm when the result is spurious.

B - Best answer: Low-risk clinical context and hemolysis justify urgent verification rather than automatic treatment or dismissal.

C - Why this option does not fit: The ECG alone is not a reliable exclusion test.

D - Why this option does not fit: A possible artifact still requires confirmation.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 059

A 65-year-old woman has prolonged gastric drainage and ventricular ectopy. Potassium remains 2.7 mmol/L despite several replacement doses. Magnesium has not been measured, chloride is low, and alkalosis persists. The resident proposes doubling potassium delivery without checking kidney function or accounting for the current infusion concentration. The surgeon must identify ongoing loss and coupled deficiency rather than treat the serum value as an isolated reservoir. She remains on a monitored bed, the gut cannot currently be used reliably, and a protocol-based IV replacement pathway is available. The next plan must specify both safe administration and evidence that the underlying mechanism has changed. Continuing gastric losses and alkalosis can coexist with a missing magnesium contribution. The replacement strategy therefore needs current concentrations, preparation checks and repeat observations, so treatment addresses the persistent mechanism rather than merely increase the amount on the next order.

What is the best next approach?

A. Check and correct magnesium while addressing ongoing losses and safe potassium delivery

B. Escalate potassium indefinitely without reviewing other factors

C. Wait for magnesium correction before assessing the arrhythmia

D. Give potassium by IV push because ectopy is present

Answer and option analysis

A - Best answer: Magnesium deficiency can sustain renal potassium loss; continuing gastric and alkalosis-related mechanisms also require treatment.

B - Why this option does not fit: This risks overshoot or infusion harm without correcting the cause.

C - Why this option does not fit: The immediate rhythm threat and linked deficits require coordinated care.

D - Why this option does not fit: Urgency does not make IV potassium push safe.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 060

A 57-year-old man with renal impairment has phosphate 0.25 mmol/L and marked respiratory weakness. Potassium is 5.9 mmol/L. A resident selects potassium phosphate because it is the familiar IV preparation and orders additional potassium-containing maintenance. Calcium is borderline low. The surgeon must arrange urgent treatment of severe symptomatic phosphate depletion while recognizing that the replacement salt delivers another clinically important ion. The patient needs frequent reassessment and renal-adjusted delivery; there is no justification for treating phosphate as though its accompanying sodium or potassium content were invisible. Pharmacy can supply an alternative appropriate preparation through the local protocol. The choice also affects the total sodium and potassium account, not only the phosphate concentration. The surgeon must coordinate replacement with renal function, symptoms, repeat calcium and infusion compatibility rather than treat the pharmacy product name as a complete prescription.

Which plan is best?

A. Choose phosphate replacement with deliberate cation selection and renal/calcium monitoring

B. Infuse calcium and phosphate together without checking compatibility

C. Withhold all treatment because any replacement has risk

D. Use potassium phosphate regardless of the current potassium

Answer and option analysis

A - Best answer: Potassium phosphate can worsen existing hyperkalemia; preparation and dose must fit the whole electrolyte state.

B - Why this option does not fit: Precipitation and calcium disturbances make unverified coadministration unsafe.

C - Why this option does not fit: Severe symptomatic depletion still requires a safe targeted plan.

D - Why this option does not fit: The accompanying potassium load is clinically relevant.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 061

A 39-year-old woman becomes anxious and markedly hyperventilates after surgery. She develops perioral tingling and carpal spasm. Total calcium is within the laboratory reference interval, but the blood gas shows alkalemia. The resident excludes calcium-related symptoms from the total value and proposes repeated sedative doses without reviewing airway, pain, pulmonary disease or other triggers. The surgeon must assess the cause of hyperventilation and whether altered protein binding has changed biologically active calcium. The patient is not assumed to have a benign anxiety episode simply because symptoms began after a stressful event, and direct ionized calcium can be measured promptly. Symptoms developed during a rapid change in ventilation, which makes timing especially important. The surgeon should avoid automatically assigning a benign emotional cause and should use clinical examination and the relevant active-ion measurement to determine whether specific treatment is required.

Which interpretation is most appropriate?

A. Assume all postoperative hyperventilation is anxiety

B. Normal total calcium excludes all calcium-mediated symptoms

C. Assess ionized calcium and the cause of alkalosis despite normal total calcium

D. Treat the total calcium number without assessing ventilation

Answer and option analysis

A - Why this option does not fit: Hypoxemia, embolism, sepsis and other serious causes remain possible.

B - Why this option does not fit: Total concentration does not fully describe the ionized fraction.

C - Best answer: Alkalemia increases calcium binding and can reduce the active fraction without an equivalent total-calcium change.

D - Why this option does not fit: Cause-directed assessment is essential and the relevant active measurement may differ.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 062

A 52-year-old woman develops tingling, tetany, and QT prolongation after total thyroidectomy. Ionized calcium is low and magnesium is also reduced. She can speak but is increasingly distressed. A resident recommends waiting for an albumin-corrected total calcium calculation before treating, while another proposes one rescue dose with no follow-up plan because the operation is over. The surgeon must address symptomatic acute hypocalcemia and anticipate persistent hypoparathyroid physiology or magnesium-related impairment. ECG monitoring and IV therapy are available, and endocrine review can help establish oral calcium, active vitamin D, and subsequent biochemical surveillance once the immediate threat is controlled. The patient requires an immediate monitored response and a continuing explanation for recurrence. Correcting the acute value does not establish recovery of parathyroid function, and unaddressed magnesium deficiency may make subsequent calcium management ineffective.

What is the best approach?

A. Treat once and omit further surveillance

B. Delay rescue until a correction formula confirms severity

C. Provide monitored acute calcium treatment and assess magnesium and ongoing endocrine needs

D. Correct magnesium alone while ignoring symptomatic calcium instability

Answer and option analysis

A - Why this option does not fit: Postoperative hypocalcemia may persist or recur and needs reassessment.

B - Why this option does not fit: Direct ionized measurement and clinical findings already establish the urgent problem.

C - Best answer: Symptoms and low ionized calcium warrant rescue; a continuing cause and replacement plan remain necessary afterward.

D - Why this option does not fit: Linked deficiency treatment does not replace immediate care of the acute calcium threat.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 063

A 71-year-old man in intensive care has total calcium 7.3 mg/dL and albumin 1.8 g/dL after prolonged sepsis. He has no tetany, new QT abnormality, or unexplained circulatory change. A resident orders repeated IV calcium solely to normalize total calcium, using an albumin formula as a definitive measurement. Kidney function is impaired and phosphate is elevated. The surgeon must distinguish altered protein binding from true biologically important hypocalcemia and avoid unnecessary treatment that could worsen complications. A properly handled direct ionized sample can be obtained, while the cause of his hypoalbuminemia and other biochemical changes is reviewed. The proposed treatment must demonstrate a physiological indication in the present patient. A low total value is a prompt for interpretation, not a mandate to administer calcium repeatedly without considering the active fraction and the associated renal-phosphate state.

What is the most appropriate next step?

A. Assess ionized calcium and clinical indication before routine IV replacement

B. Normalize total calcium with repeated IV doses regardless of ionized level

C. Assume low albumin makes every calcium disturbance harmless

D. Ignore kidney function and phosphate when selecting treatment

Answer and option analysis

A - Best answer: Total calcium and correction formulas can mislead in critical illness; treatment should address active deficiency and symptoms.

B - Why this option does not fit: This risks treating a binding-related laboratory finding rather than a clinical deficit.

C - Why this option does not fit: True ionized hypocalcemia can still coexist and must be assessed.

D - Why this option does not fit: Both alter the risks and interpretation of calcium therapy.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 064

A 74-year-old woman with malignancy develops confusion, AKI, and marked hypercalcemia. She is clinically depleted but has limited cardiac reserve and a history of pulmonary edema. The resident applies a fixed large saline regimen without a tolerance assessment and adds a loop diuretic purely to lower calcium. The surgeon must restore appropriate circulation while planning cause-specific calcium-lowering treatment and avoiding a new respiratory emergency. Endocrine and renal expertise are available, and the rate of replacement can be adjusted from examination, output and monitoring. The patient does not have a universal volume requirement simply because hypercalcemia commonly causes dehydration. The initial saline rate should be adjusted from response and congestion rather than copied from an otherwise healthy patient. The team must also identify the cause and choose sustained calcium-lowering treatment, because restoring volume alone may not resolve the underlying process.

Which plan is best?

A. Individualize volume restoration and arrange appropriate calcium-lowering therapy with close reassessment

B. Use a fixed large volume regardless of respiratory findings

C. Rely on loop diuresis alone before addressing depletion

D. Wait for spontaneous correction because confusion is nonspecific

Answer and option analysis

A - Best answer: Depletion needs treatment, but cardiac/renal tolerance and the underlying cause determine the safe strategy.

B - Why this option does not fit: This can precipitate harmful congestion in limited cardiac reserve.

C - Why this option does not fit: Diuresis can worsen effective volume and does not replace cause-specific management.

D - Why this option does not fit: Marked symptomatic disease and AKI warrant prompt assessment and treatment.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 065

A 77-year-old man with CKD and postoperative constipation receives repeated magnesium-containing laxatives. He becomes weak, bradycardic, hypotensive, and hyporeflexic, then develops shallow breathing. Magnesium is markedly elevated. The resident attributes the reflex loss to sedation and proposes another laxative because ileus persists. The surgeon must recognize a drug-related electrolyte emergency and understand that impaired clearance can sustain toxicity. Calcium, respiratory support, and urgent renal assessment can be arranged. Further magnesium exposure is avoidable, and the decision about extracorporeal clearance should reflect severity and kidney function rather than waiting for the bowel problem to resolve first. Slowed intestinal transit can prolong absorption from an oral source, while impaired kidneys reduce clearance. The surgeon must treat the respiratory and cardiac threat and stop further exposure rather than assume a constipation treatment cannot be responsible for systemic deterioration.

What is the best next approach?

A. Wait for spontaneous excretion despite advanced impairment and severe symptoms

B. Continue laxatives because oral magnesium cannot cause systemic toxicity

C. Treat only with more sedative for discomfort

D. Stop magnesium and provide urgent supportive, calcium-directed and renal assessment as indicated

Answer and option analysis

A - Why this option does not fit: Inadequate clearance may require urgent dialysis assessment.

B - Why this option does not fit: Oral exposure can produce severe toxicity, especially with renal impairment and slowed transit.

C - Why this option does not fit: This may worsen respiratory compromise and ignores the biochemical mechanism.

D - Best answer: The pattern is compatible with magnesium toxicity; respiratory/cardiac compromise and poor clearance make delay dangerous.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 066

A malnourished 60-year-old woman remains difficult to wean from ventilation after prolonged abdominal illness. Oxygenation and lung imaging have improved, but respiratory muscle strength is poor. Feeding was recently advanced and phosphate is 0.24 mmol/L, with low magnesium and potassium. A resident attributes the entire problem to deconditioning and sees no need to change the nutrition or electrolyte plan because prefeeding levels were normal. The surgeon must assess a potentially reversible energy and muscle-function problem without assuming it is the only cause of respiratory failure. Kidney function, ionized calcium, and replacement compatibility will influence the next prescription. Other causes of weaning difficulty still need assessment, but the severe measured phosphate deficit deserves immediate attention. A sustainable response also requires the feeding and linked-electrolyte plan to change rather than replace one value while continuing the same driving exposure.

Which approach is most appropriate?

A. Exclude depletion because baseline phosphate was normal

B. Treat severe phosphate depletion and assess linked refeeding physiology while evaluating other causes

C. Escalate calories unchanged and replace only potassium

D. Infuse phosphate rapidly without renal or calcium monitoring

Answer and option analysis

A - Why this option does not fit: Serum concentration before feeding does not establish adequate total stores.

B - Best answer: The timing and combined deficiencies can contribute to diaphragmatic weakness despite improved lung oxygenation.

C - Why this option does not fit: This leaves severe phosphate/magnesium problems and the feeding mechanism unaddressed.

D - Why this option does not fit: Replacement can cause important biochemical and infusion complications.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 067

A 54-year-old man with major weight loss begins nutrition after weeks of poor intake. Within two days he develops edema, tachycardia, weakness and ventricular ectopy. Phosphate, magnesium and potassium fall substantially, while glucose rises. The feeding prescription appears modest, but medication and maintenance infusions deliver additional carbohydrate that was not counted. The resident proposes treating each low value independently while continuing to advance calories. The surgeon must integrate the temporal pattern, organ effects, thiamine risk and fluid exposure rather than treat refeeding as a synonym for one low phosphate result. Nutrition and critical-care teams can coordinate the response. The patient may have more than one reason for weakness and edema, so the diagnosis needs clinical integration. The striking nutrient-linked electrolyte trajectory nevertheless requires action now, with safe replacement adjusted for clearance and repeat results rather than unchanged full advancement.

What is the best plan?

A. Treat only phosphate because other changes are unrelated

B. Stop nutrition indefinitely after correcting the first laboratory value

C. Use the written feed volume as the entire carbohydrate exposure

D. Address probable refeeding syndrome with coordinated nutrient, thiamine, fluid and electrolyte management

Answer and option analysis

A - Why this option does not fit: Potassium, magnesium, vitamins and fluid shifts are integral to the syndrome.

B - Why this option does not fit: A controlled sustainable refeeding plan is preferable to unplanned prolonged starvation.

C - Why this option does not fit: Dextrose-containing drugs and fluids can add meaningful calories.

D - Best answer: The linked trajectory and organ findings require adjustment of the driving exposure as well as safe replacement.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 068

A 64-year-old man develops rhabdomyolysis and AKI after prolonged limb ischemia. Phosphate and potassium are markedly elevated, total calcium is low, and he has oliguria. A resident proposes large phosphate-containing nutritional supplements and routine calcium normalization without considering symptoms or ionized values. The surgeon must prioritize dangerous potassium and renal complications, assess calcium-related symptoms, and understand that the released intracellular phosphate is not simply an isolated dietary problem. There is no tetany or current calcium-mediated arrhythmia, but ECG and ionized assessment are being obtained. Renal consultation is available for the combined clearance problem and possible dialysis indications. The calcium decision should reflect symptoms, ionized results and the risk of calcium-phosphate deposition in the clinical setting. The combination of severe potassium excess, ongoing cell injury and poor clearance also demands a durable elimination plan rather than isolated laboratory correction.

Which approach is best?

A. Manage potassium only once and ignore ongoing cell release

B. Treat the cell-breakdown and renal emergency while individualizing calcium and stopping excess phosphate

C. Normalize total calcium automatically without contextual assessment

D. Replace phosphate because weakness implies every patient is depleted

Answer and option analysis

A - Why this option does not fit: Continuing release and poor excretion create recurrent danger.

B - Best answer: Hyperphosphatemia, potassium excess and impaired clearance are linked; indiscriminate replacement can add harm.

C - Why this option does not fit: In phosphate excess, calcium treatment needs a clear indication and careful evaluation.

D - Why this option does not fit: The measured phosphate and cell-breakdown mechanism show the opposite problem.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 069

A 68-year-old man has persistent gastric drainage and worsening abdominal pain after surgery. Arterial pH is near normal, bicarbonate is 22 mmol/L, and contemporaneous sodium and chloride yield a measured gap of 25. Albumin is 2 g/dL and lactate is elevated. The resident considers the near-normal pH proof that there is no serious acid-base problem and attributes pain to ileus. The surgeon must determine whether an acidosis is offset by another process and whether the abdominal source requires urgent assessment. The patient's pressure is supported and therefore cannot independently establish adequate regional perfusion. There is enough information to begin interpretation before repeating every test. The degree of gap elevation should be judged against albumin and the local reference convention. A supported pressure and a normal final pH cannot replace assessment of tissue perfusion or the anatomical process causing new abdominal findings.

What is the most appropriate conclusion?

A. Low albumin makes the gap less important

B. A high-gap acidosis can be masked by concurrent alkalosis and needs cause-directed evaluation

C. Treat pain alone without reviewing the surgical source

D. Near-normal pH excludes metabolic acidosis

Answer and option analysis

A - Why this option does not fit: Low albumin lowers the expected gap, so an elevated value remains concerning.

B - Best answer: Normal pH reflects the net effect, not absence of opposing processes; low albumin can further conceal gap severity.

C - Why this option does not fit: The evolving focal findings and abnormal gap warrant a broader assessment.

D - Why this option does not fit: Opposing disorders can normalize the final pH.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 070

A 63-year-old woman with septic illness and AKI has bicarbonate 12 mmol/L and arterial PaCO2 40 mm Hg. She becomes drowsy after analgesia and breathes less vigorously than earlier. Saturation remains 98% on oxygen. The resident calls the CO2 normal and interprets the disorder as fully compensated metabolic acidosis. The surgeon must compare the value with expected compensation rather than a laboratory reference interval and recognize a potential airway/ventilation problem. The patient requires urgent bedside review, while source control and the metabolic cause continue to be assessed. A repeat gas should follow any major respiratory intervention. The change in respiratory effort provides a clinical explanation for the additional process, but other causes still require assessment. Immediate support planning should preserve the patient's needed ventilation and continue treatment of the metabolic and surgical problem.

What is the correct interpretation?

A. There is isolated respiratory alkalosis

B. Compensation is appropriate because PaCO2 is in the usual normal interval

C. Normal saturation excludes ventilatory failure

D. There is additional respiratory acidosis; expected PaCO2 is approximately 26 plus or minus 2

Answer and option analysis

A - Why this option does not fit: The observed CO2 is higher, not lower, than expected for the metabolic acidosis.

B - Why this option does not fit: The appropriate value depends on the metabolic disturbance, not the reference interval alone.

C - Why this option does not fit: Supplemental oxygen can preserve saturation despite inadequate CO2 elimination.

D - Best answer: Winter's formula gives 1.5 x 12 + 8 = 26; a value of 40 is inappropriately high.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 071

A 71-year-old woman with prolonged surgical sepsis has sodium 138, chloride 105 and chemistry bicarbonate 18 mmol/L. Albumin is 2 g/dL. The resident reports that the measured gap of 15 is too modest to justify checking for lactate, ketones or retained acids. The patient has worsening perfusion and recent medication changes. The surgeon must account for the main unmeasured protein anion and use the calculation as a prompt for direct cause-specific tests, not as a substitute for them. Samples are contemporaneous and reliable, and the hospital uses a gap convention that excludes potassium with an illustrative normal value near 12. The adjusted result is a screening interpretation rather than a measurement of a particular acid. It should prompt appropriate direct testing and source review, with the patient's clinical deterioration retaining importance even if repeat arithmetic remains only moderately abnormal.

What is the approximate albumin-adjusted gap?

A. 20 mmol/L, supporting evaluation for unmeasured anions

B. 15 mmol/L because albumin never affects the gap

C. 35 mmol/L because albumin is multiplied directly by sodium

D. 10 mmol/L because albumin correction is subtracted from the measured gap

Answer and option analysis

A - Best answer: Measured gap is 138 - 105 - 18 = 15; adding 2.5 x (4 - 2) gives 20.

B - Why this option does not fit: Albumin materially contributes to the expected unmeasured anions.

C - Why this option does not fit: That expression has no valid role in the stated gap calculation.

D - Why this option does not fit: That reverses the usual correction for low albumin.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 072

A 73-year-old man has gastric losses, sepsis concern and worsening abdominal tenderness. Arterial pH is 7.46, PaCO2 29 mm Hg and bicarbonate about 20 mmol/L. Chemistry shows sodium 140, chloride 94 and albumin 2 g/dL. Lactate is high. A resident attributes the entire pattern to pain-related hyperventilation and suggests reassurance because the blood is alkalemic. The surgeon must calculate the adjusted gap and compare respiratory response with the expected value before deciding whether a surgical ischemic or infectious source remains urgent. Historical baseline bicarbonate is unavailable, so mixed-metabolic interpretation must retain appropriate uncertainty rather than imply mathematical proof of every cause. The multiple processes have plausible different causes that need parallel treatment and confirmation. The surgical team should not let an alkalemic pH prevent urgent assessment of ischemia or infection, nor use a calculated pattern as proof of one anatomical diagnosis.

Which interpretation best fits?

A. Pure metabolic alkalosis with normal regional perfusion

B. Isolated normal-gap acidosis with appropriate compensation

C. Isolated respiratory alkalosis with no metabolic concern

D. High-gap metabolic acidosis with respiratory alkalosis and likely additional metabolic alkalosis

Answer and option analysis

A - Why this option does not fit: Neither the gap nor the clinical trajectory supports that reassuring conclusion.

B - Why this option does not fit: The gap is elevated and PaCO2 is lower than the expected compensatory range.

C - Why this option does not fit: The markedly elevated adjusted gap cannot be explained away by pH direction.

D - Best answer: Adjusted gap is 31; PaCO2 is below Winter's estimate, and the gap rise greatly exceeds the bicarbonate fall.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 073

A 58-year-old man with severe metabolic acidosis is tiring despite very rapid spontaneous breathing. Bicarbonate is 8 mmol/L, PaCO2 20 mm Hg and pH about 7.22. He is hypotensive and requires urgent experienced airway assessment. A resident prepares routine induction followed by a standard low minute-ventilation setting, assuming that mechanical ventilation automatically improves every gas. The surgeon must anticipate what apnea and loss of compensation could do before the metabolic process is corrected. Source treatment, circulatory preparation and critical-care support are being mobilized. The decision is not whether to tolerate a failing airway, but how to avoid a preventable physiological collapse during the necessary intervention. The airway team must consider whether the patient can tolerate the induction sequence and how quickly adequate ventilation can be reestablished. A plan that corrects saturation while allowing CO2 to rise toward a routine normal target can worsen the metabolic emergency substantially.

Which planning principle is most important?

A. Treat oxygen saturation as proof of sufficient ventilation

B. Delay all airway support until the acidosis is fully corrected

C. Minimize avoidable apnea and plan adequate postintubation ventilation with hemodynamic support

D. Use a normal PaCO2 target immediately regardless of bicarbonate

Answer and option analysis

A - Why this option does not fit: Saturation does not measure CO2 clearance or compensatory minute ventilation.

B - Why this option does not fit: A failing airway or exhaustion may require urgent intervention before definitive metabolic recovery.

C - Best answer: The patient depends on high ventilation; losing it can rapidly worsen acidemia and circulation.

D - Why this option does not fit: A normal value may be dangerously high relative to metabolic compensation needs.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 074

A 70-year-old man with chronic hypercapnic lung disease has baseline PaCO2 near 60 mm Hg and bicarbonate near 32 mmol/L. After surgery he receives diuretics and prolonged gastric suction. The new gas shows PaCO2 60, bicarbonate 42 and alkalemia. Chloride and potassium are low. A resident attributes all bicarbonate elevation to chronic respiratory compensation and continues the same loss-producing therapy without review. The surgeon must compare the current result with the known baseline and determine whether a second process now contributes. Oxygenation is stable, but weakness and ectopy are increasing. Volume tolerance is limited by cardiac disease, so any chloride replacement must be individualized rather than automatic. The current alkalemia can affect potassium and ionized calcium, adding a reason to review symptoms and rhythm. The clinician must account for ongoing losses and limited tolerance while avoiding a simple assumption that all high bicarbonate is benign compensation.

What is the best interpretation?

A. All bicarbonate elevation must be respiratory compensation

B. The patient has isolated acute respiratory acidosis

C. A superimposed metabolic alkalosis is likely beyond the chronic respiratory adaptation

D. Give unlimited saline without checking congestion

Answer and option analysis

A - Why this option does not fit: The known baseline and new loss history argue against that single explanation.

B - Why this option does not fit: CO2 has not newly risen and the blood is alkalemic.

C - Best answer: Stable CO2 with bicarbonate substantially above baseline and chloride/potassium loss supports an additional metabolic process.

D - Why this option does not fit: The mechanism may need chloride treatment, but volume tolerance still determines delivery.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 075

A 46-year-old woman becomes tachypneic and restless after surgery. PaCO2 is 25 mm Hg, bicarbonate is mildly reduced, and pH is alkalemic. She has new pleuritic discomfort, increasing oxygen requirement and a faster pulse. The resident calls this anxiety because she is frightened and proposes sedation without further review. She also has malignancy and reduced mobility. The surgeon must distinguish a physiological description from its cause and determine whether pulmonary embolism, hypoxemia, sepsis or another process requires urgent evaluation. She remains sufficiently stable for a monitored diagnostic pathway, but the trajectory is new and cannot be explained solely by her emotional reaction to it. The emotional response may be a consequence of the respiratory problem rather than its cause. The decision should preserve the chance to diagnose a treatable pulmonary or systemic event before sedation suppresses a useful warning sign and delays definitive evaluation.

What is the best response?

A. Give bicarbonate to correct the mildly low bicarbonate

B. Sedate first and stop assessment if the respiratory rate falls

C. Exclude embolism because pH is high rather than low

D. Evaluate serious causes of hyperventilation rather than accepting anxiety as the diagnosis

Answer and option analysis

A - Why this option does not fit: The lower bicarbonate may be adaptive and does not establish an alkali deficit.

B - Why this option does not fit: Suppressing a sign can mask or worsen the underlying problem.

C - Why this option does not fit: Alkalemia is compatible with acute pulmonary vascular disease.

D - Best answer: Respiratory alkalosis can accompany embolism, hypoxemia or sepsis; the new respiratory findings require investigation.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 076

A 50-year-old man with a high-output distal intestinal fistula has bicarbonate 15 mmol/L, chloride 113, sodium 138 and albumin near normal. Lactate is normal and perfusion is stable, though he has weight loss and electrolyte depletion. The resident assumes every low bicarbonate reflects occult septic lactic acidosis and proposes more pressor support. The surgeon must interpret the approximately normal gap, the loss source, potassium and renal function, while still assessing infection clinically rather than excluding it from one lactate result. The next prescription should replace the appropriate abnormal losses and address the fistula, not merely change pressure in a currently stable circulation. The clinical stability changes the immediate support need, but does not make ongoing fistula output harmless. Composition, replacement interval, renal function and the underlying anatomy must remain linked to the prescription and its planned reassessment.

Which explanation is most useful?

A. Treat the acidosis solely by increasing norepinephrine

B. Bicarbonate-rich intestinal losses can produce a hyperchloremic metabolic acidosis

C. Low bicarbonate proves elevated lactate despite the measured result

D. Normal pressure eliminates all need for fluid and electrolyte assessment

Answer and option analysis

A - Why this option does not fit: Vasoconstriction does not replace lost bicarbonate-rich fluid or treat the fistula.

B - Best answer: The source and normal-gap pattern support external alkali loss, requiring composition-aware replacement and cause management.

C - Why this option does not fit: Several metabolic mechanisms lower bicarbonate without lactate accumulation.

D - Why this option does not fit: Stable pressure does not exclude significant ongoing abnormal losses.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 077

A 66-year-old woman remains in septic shock with severe metabolic acidemia, pH 7.12, and significant AKI. Ventilation and source-control status are being reassessed. She is already sodium and volume loaded, and ionized calcium is low. A resident argues that bicarbonate is always prohibited in every lactic-acidosis setting; another argues it reliably improves survival and should replace source treatment. The surgeon must distinguish selected guideline-supported consideration from a universal benefit claim, and account for CO2 generation, calcium and fluid effects. Renal-replacement indications are being reviewed from the entire clinical picture rather than an isolated bicarbonate value. The team needs a specific therapeutic aim and review schedule if bicarbonate is chosen. The decision remains conditional and context-dependent; a treatment that changes pH can still add sodium, CO2 and calcium-related hazards without controlling the cause.

Which statement is most defensible?

A. Bicarbonate is absolutely forbidden regardless of AKI and severity

B. Consider bicarbonate under the monitored severe-acidemia/AKI pathway while continuing definitive treatment

C. Bicarbonate guarantees survival and complete hemodynamic recovery

D. Give it without considering ventilation or ionized calcium

Answer and option analysis

A - Why this option does not fit: That ignores the selected conditional indication.

B - Best answer: Guidance permits selected use with very low certainty; it does not replace ventilation, perfusion or source care.

C - Why this option does not fit: The evidence does not justify that certainty or replace cause-directed care.

D - Why this option does not fit: CO2 generation and calcium effects can create important treatment hazards.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 078

A 62-year-old man has a blood gas during severe shock and a chemistry panel collected several hours later after transfusion, saline and a ventilator change. The resident combines the earlier CO2 with the later bicarbonate and chloride to calculate compensation and a mixed-disorder diagnosis. The calculated pattern appears internally inconsistent with the current examination. The surgeon must decide whether to act on a complex interpretation or first obtain contemporaneous reliable measurements. The patient remains monitored, and urgent source treatment is continuing independently. A repeat sample can be obtained promptly without delaying control, while the timing and composition of treatment are documented. The original samples can still help describe the trajectory if their times are kept separate. They should not be combined as though they describe one steady state, especially after major changes in ventilation, perfusion and chloride exposure.

What is the best next step?

A. Trust every formula despite mismatched sampling times

B. Assume chemistry bicarbonate and calculated gas bicarbonate must always be identical

C. Interpret current contemporaneous gas and chemistry with the treatment timeline

D. Stop definitive treatment until all previous samples agree

Answer and option analysis

A - Why this option does not fit: The arithmetic cannot repair an invalid temporal comparison.

B - Why this option does not fit: Different methods and timing can produce discrepancies requiring evaluation.

C - Best answer: Combining measurements from changing physiological states can manufacture a misleading mixed-disorder conclusion.

D - Why this option does not fit: Urgent source care should continue while measurements are clarified.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 079

A 57-year-old woman taking an SGLT2 inhibitor has poor intake and undergoes urgent surgery. The next day she is nauseated, tachypneic and acidemic with an elevated albumin-adjusted gap. Glucose is 175 mg/dL and lactate is not high enough to explain the gap. The resident excludes ketoacidosis because glucose is not markedly elevated and proposes more saline for presumed generic shock. The surgeon must identify a medication-associated possibility while evaluating perfusion, infection and other causes. Ketone testing is available, and the patient needs a coordinated metabolic treatment pathway if confirmed, including potassium surveillance rather than an unstructured fluid-only response. The medication and fasting history make direct ketone assessment particularly useful. If the diagnosis is confirmed, treatment must account for glucose delivery, insulin, potassium and the surgical precipitant rather than assume a relatively modest glucose value excludes an important metabolic emergency.

What should be prioritized diagnostically?

A. Treat only with repeated fluid until bicarbonate normalizes

B. Measure ketones and evaluate euglycemic ketoacidosis in the medication context

C. Exclude ketoacidosis solely from glucose below 200

D. Assume every gap is lactate even when measured lactate is modest

Answer and option analysis

A - Why this option does not fit: Cause-specific insulin/glucose and electrolyte management may be required through the relevant pathway.

B - Best answer: SGLT2-associated ketoacidosis can occur without marked hyperglycemia, especially around fasting and surgery.

C - Why this option does not fit: Glucose magnitude alone is not an adequate exclusion in this setting.

D - Why this option does not fit: The gap requires evaluation for additional unmeasured acids.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 080

A 69-year-old man with longstanding CO2 retention is ventilated after surgery. PaCO2 is rapidly reduced from his chronic level near 70 to 40 mm Hg, while bicarbonate remains around 36 mmol/L. He becomes alkalemic and develops hypokalemia. The resident interprets the high bicarbonate as a new indication for fluid restriction alone and considers the respiratory history irrelevant. The surgeon must understand the different timing of respiratory correction and renal adaptation, while assessing diuretic exposure, chloride losses and volume tolerance. The next ventilator and electrolyte plan should be coordinated rather than target one normal gas value at the expense of a coherent overall physiological state. The team's routine target may conceal the importance of the chronic baseline and the speed of change. A coordinated respiratory and metabolic plan should use the current pH, electrolytes and volume state rather than simply normalize one variable and ignore the rest.

Which mechanism best explains an important contribution?

A. Persistent renal bicarbonate adaptation after rapid correction of chronic hypercapnia

B. New acute respiratory acidosis because CO2 is 40

C. The pattern proves that all patients need immediate large-volume saline

D. A normal CO2 guarantees normal acid-base status

Answer and option analysis

A - Best answer: CO2 can change faster than chronic renal compensation resolves, producing posthypercapnic alkalosis.

B - Why this option does not fit: That value is lower than baseline and does not explain the alkalemia.

C - Why this option does not fit: Additional chloride and volume treatment depends on actual losses and tolerance.

D - Why this option does not fit: Bicarbonate and the preceding physiological state remain essential.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 081

A 64-year-old man remains in shock after an emergency operation. At shift change, MAP is 68 mm Hg, but vasopressor dose doubled during the last hour, urine output has fallen, and the surgeon is unsure whether source control was complete. The outgoing resident proposes reporting only the latest pressure because it meets the target. Several fluid challenges and product exposures have occurred, but their responses are poorly documented. The incoming team must decide what to do next without repeating ineffective therapy. The surgeon is asked which handover structure best preserves clinical judgment, unresolved uncertainty and accountability rather than presenting a reassuring isolated number. An incoming team needs to know what would falsify the current explanation and who will respond to it. The support trajectory, observed response and anatomical uncertainty are therefore part of the clinical handover, not optional narrative details.

Which handover is most useful?

A. Report MAP alone because it is the principal resuscitation endpoint

B. State mechanism, support trajectory, source status, intervention responses and explicit reassessment triggers

C. List product totals without responses or current questions

D. Declare the diagnosis final so the next team will not reconsider it

Answer and option analysis

A - Why this option does not fit: It omits escalating support and worsening tissue/organ findings.

B - Best answer: This makes the current explanation reviewable and allows the next team to recognize failure or change.

C - Why this option does not fit: Exposure matters, but does not explain benefit, harm or the next action.

D - Why this option does not fit: Ongoing deterioration requires the ability to reopen the differential.

Review link: teaching Section 9. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 082

A 71-year-old woman develops hypoxemia after several liters of postoperative fluid. She is edematous and lung ultrasound shows bilateral B-lines. However, she also has fever, focal chest findings and a recent aspiration episode. A resident attributes all respiratory deterioration to volume and recommends aggressive removal without assessing perfusion or infection. Another insists that because several diagnoses are possible, none should be investigated until the next day. The surgeon must recognize that a marker of extravascular lung water does not identify the entire cause and that fluid tolerance and pulmonary pathology can coexist. Current pressure support and renal function will determine whether removal is safe. Several contributors may be active at once, and therapy directed at one should not erase the others from consideration. The surgeon must select the next assessment and intervention from the whole trajectory rather than force every respiratory finding into a single fluid label.

Which approach is best?

A. Ignore fluid burden because an aspiration occurred

B. Diagnose pure overload from B-lines alone

C. Evaluate the pulmonary differential and congestion together while assessing removal readiness

D. Delay all reassessment because the differential is mixed

Answer and option analysis

A - Why this option does not fit: An additional cause does not eliminate a harmful volume contribution.

B - Why this option does not fit: The finding is not sufficiently cause-specific for that conclusion.

C - Best answer: Edema and B-lines support fluid intolerance but do not exclude aspiration, infection or permeability injury.

D - Why this option does not fit: Mixed physiology demands parallel evaluation rather than diagnostic paralysis.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 083

A 60-year-old man has persistent hypoperfusion after surgery. The team agrees to test whether a small fluid intervention improves forward flow, but the resident writes an open-ended rapid infusion without a defined dose, measurement method or stopping condition. He already has a rising oxygen requirement. Another clinician plans to judge success only from a brief pressure rise. The surgeon must convert the intention into a test whose result can guide the next decision. Reliable stroke-volume assessment is available, and alternative mechanisms are being considered in parallel. The question is how to prescribe and evaluate the challenge, not whether every patient with shock needs it. The intended challenge is a test of useful hemodynamic benefit under current conditions. Defining the result and harm criteria beforehand makes it possible to stop ineffective treatment and explain the next step, even if one pressure number briefly looks better.

What is the best modification?

A. Specify indication, amount/rate, flow endpoint, tolerance checks and a review time

B. Use urine output within one minute as the only response measure

C. Declare the challenge therapeutic regardless of measured response

D. Leave the infusion open until pressure is normal

Answer and option analysis

A - Best answer: A controlled challenge requires a measurable expected benefit and stopping conditions rather than an unbounded infusion.

B - Why this option does not fit: Renal output can lag and is not an immediate direct stroke-volume measurement.

C - Why this option does not fit: The result must determine whether repetition remains justified.

D - Why this option does not fit: Pressure alone does not establish useful flow or acceptable treatment burden.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 084

A 58-year-old man deteriorates after a thoracic procedure. He has low flow, elevated venous pressure and rapidly increasing support. A resident concludes that the high CVP excludes any obstructive mechanism and favors more vasoconstrictor support without imaging. The lungs are asymmetrical, and a focused cardiac assessment has not been repeated since the procedure. The surgeon must decide whether pressure upstream of an obstruction can rise while forward circulation fails, and whether a time-critical cause warrants immediate action. Bedside ultrasound can be brought quickly, but the patient may require treatment from clinical findings before complete imaging if instability and suspicion are compelling. The procedural context changes the probability of a mechanically correctable event. The surgeon should choose the fastest safe assessment or treatment that can address that possibility, not treat a high upstream pressure as evidence that circulation through the whole system is adequate.

Which interpretation is correct?

A. Vasopressors provide definitive mechanical correction

B. Elevated upstream venous pressure is compatible with obstructive shock

C. High CVP proves normal cardiac filling and excludes obstruction

D. The preprocedure assessment excludes a new procedural complication

Answer and option analysis

A - Why this option does not fit: They do not relieve a tension pneumothorax or drain tamponade.

B - Best answer: Obstruction can impair filling or ejection while venous pressure rises; current clinical and imaging assessment is essential.

C - Why this option does not fit: Pressure accumulation does not certify effective forward flow.

D - Why this option does not fit: The abrupt change requires a renewed differential.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 085

A 75-year-old man with known EF 30% develops hypotension after abdominal surgery. He has pulmonary congestion, but also increasing abdominal distension and a brief response to emergency blood. The focused echo shows no clear change from baseline, and hemoglobin is only modestly lower than earlier. The resident attributes the entire episode to the old cardiomyopathy and requests diuresis before surgical reassessment. The surgeon must determine whether poor cardiac reserve changes resuscitation tolerance without necessarily explaining the new loss of perfusion. Concealed hemorrhage and mixed physiology remain plausible, and immediate senior review can coordinate appropriate support and the safest route to source evaluation. The existing cardiac diagnosis is important because it changes the patient's reserve and risk from additional volume. It should refine the treatment plan while the new deterioration is explained, rather than become a reason to postpone assessment of a fresh surgical problem.

What is the best working diagnosis and plan?

A. Treat the old EF as proof of isolated acute pump failure

B. Use pulmonary congestion to rule out effective volume loss

C. Give diuresis before evaluating any surgical cause

D. Consider mixed hemorrhagic and cardiac physiology and urgently evaluate the new source

Answer and option analysis

A - Why this option does not fit: An unchanged chronic finding cannot exclude a new hemorrhagic process.

B - Why this option does not fit: Congestion can coexist with inadequate circulating volume or ongoing bleeding.

C - Why this option does not fit: This may worsen effective filling and delay control of a life-threatening source.

D - Best answer: Pre-existing dysfunction increases vulnerability and limits tolerance but does not establish the cause of new postoperative shock.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 086

A 72-year-old woman with CKD receives insulin-glucose for severe hyperkalemia. Potassium falls appropriately at one hour. Three hours later she becomes sweaty and confused, but the resident assumes the earlier glucose administration prevents hypoglycemia and plans another insulin dose from an old potassium result. There has been little urine output and no repeat glucose since treatment. The surgeon must recognize a delayed adverse effect while maintaining concern for recurrent potassium excess. Bedside glucose and chemistry are readily available, and the team can arrange definitive clearance assessment. The immediate neurological change should not be assigned automatically to sepsis or the original electrolyte disorder. Confusion after insulin may have several causes, but the readily testable treatment complication needs prompt attention. At the same time, persistent renal failure means a new glucose problem must not distract the team from repeat potassium assessment and definitive clearance planning.

What is the best immediate response?

A. Exclude hypoglycemia because glucose was coadministered

B. Check and treat glucose urgently and reassess potassium before further shifting therapy

C. Ignore rebound potassium because the first repeat improved

D. Repeat insulin automatically because the initial potassium was severe

Answer and option analysis

A - Why this option does not fit: Coadministration reduces risk but does not eliminate delayed events.

B - Best answer: Delayed hypoglycemia is possible, particularly with renal impairment; repeat treatment requires current measurements and prevention safeguards.

C - Why this option does not fit: Temporary intracellular shift does not establish durable elimination.

D - Why this option does not fit: An old value cannot justify unassessed repeat therapy during possible hypoglycemia.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 087

A 55-year-old man had 1.5 L/day biliary drainage earlier in the week. Output now falls to 100 mL/day after a procedure, oral intake improves, and medication carriers remain substantial. The old replacement order continues unchanged in addition to full maintenance. Weight rises, sodium and chloride increase, and oxygen needs worsen. The resident calls the order obligatory because it was initially reasonable. The surgeon must determine whether a historical loss is still a current indication and whether reduced output itself also requires explanation in the anatomical context. The patient needs a new account and source review, not merely a copied prescription or an assumption that every change is recovery. Reduced drain output can be a marker of improvement or of impaired drainage, depending on the procedure and anatomy. The prescription and surgical interpretation must therefore be updated together rather than consider fluid accounting separate from the reason the drain was placed.

What is the best plan?

A. Continue replacement at the historical peak until discharge

B. Assume reduced output always proves anatomical healing

C. Reassess drain function and current losses, then rewrite replacement and maintenance

D. Stop monitoring because oral intake has resumed

Answer and option analysis

A - Why this option does not fit: This ignores current balance and creates preventable excess.

B - Why this option does not fit: Obstruction or displacement may also reduce drainage.

C - Best answer: The earlier indication cannot justify unchanged delivery after output and other intake change; reduced output may also need explanation.

D - Why this option does not fit: New excess and changing drainage still require clinical and biochemical review.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 088

A 59-year-old man with bowel ischemia concern has rising lactate, severe metabolic acidosis and very rapid breathing. Supplemental oxygen keeps saturation at 99%, but he becomes exhausted and less responsive. The resident proposes increasing inspired oxygen as the principal response and assumes no airway concern exists while saturation remains high. The surgeon must distinguish oxygenation from ventilation and circulatory delivery, and continue urgent anatomical assessment. The abdomen is increasingly painful and support requirements rise. Experienced airway and critical-care teams are available to coordinate support, but loss of spontaneous compensation during any intervention could be hazardous if the underlying metabolic demand is ignored. The required respiratory adaptation can be substantial and may not remain sustainable as illness progresses. The surgeon must ensure that an airway intervention replaces the lost ventilation appropriately while continuing urgent investigation of the abdominal cause and support of circulation.

Which priority best reflects the physiology?

A. Increase oxygen alone until lactate falls

B. Assess impending ventilatory failure while treating perfusion and the suspected ischemic source

C. Use saturation to defer source assessment

D. Suppress tachypnea without planning replacement ventilation

Answer and option analysis

A - Why this option does not fit: Oxygen does not correct inadequate ventilation or mechanical/anatomical source problems.

B - Best answer: Normal saturation cannot exclude CO2-retention risk, low flow or regional ischemia; respiratory compensation may be failing.

C - Why this option does not fit: Arterial saturation cannot certify regional tissue perfusion.

D - Why this option does not fit: This can remove a necessary compensatory response and worsen acidemia.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 089

A 67-year-old man has suspected postoperative bleeding but is currently maintaining perfusion after initial support. The surgical team is debating CT angiography versus direct exploration. The latest pressure is acceptable, yet the resident has not reviewed whether support is escalating or whether the last response was sustained. Imaging can change the choice of endovascular versus operative control, but transport removes him from immediate surgical access. The surgeon must decide what additional trajectory and logistical information matters rather than apply an automatic rule that every suspected bleed either always or never goes to CT. A clear deterioration threshold and contingency team can be arranged. The decision should include the resources available during transport and the response if deterioration occurs in the scanner. A test that helps anatomical planning is valuable only when its expected benefit is balanced against the time and physiological risk it adds.

What is the best decision principle?

A. Send every patient because CT always improves outcome regardless of delay

B. Never image any postoperative bleed even when stable and the result changes treatment

C. Use sustained stability, transport risk and the test's effect on immediate control strategy

D. Base transport safety solely on one cuff reading

Answer and option analysis

A - Why this option does not fit: Physiology and access to definitive control can make the detour dangerous.

B - Why this option does not fit: Selected stable patients can benefit from anatomical localization.

C - Best answer: Imaging is justified by a safe decision-changing pathway, not a single acceptable pressure or a universal rule.

D - Why this option does not fit: Support trajectory and tissue perfusion are necessary parts of the judgment.

Review link: teaching Section 9. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 090

A 62-year-old woman is recovering after severe sepsis. She is fluid positive and edematous, but perfusion is improving, kidney function is stable, potassium is acceptable and there is no severe refractory acidosis. Oxygen needs are modest. The resident requests dialysis solely to produce a negative fluid chart, without first reviewing maintenance, carriers or an appropriate diuretic strategy. The surgeon must recognize the distinction between a harmful accumulation problem that deserves management and a universal indication for extracorporeal treatment. Any removal plan needs clinical goals, tolerance review and reassessment rather than pursue a chart number independently of the patient's course. Fluid accumulation should be linked to actual clinical harm and phase of illness rather than a desired chart appearance. The team can stop unnecessary input immediately, then select a monitored removal strategy if the patient is ready and an indication exists.

Which approach is best?

A. Remove fluid rapidly without monitoring perfusion

B. Continue all input because renal function is stable

C. Reduce unnecessary input and individualize de-escalation before routine extracorporeal removal

D. Start dialysis automatically whenever cumulative balance is positive

Answer and option analysis

A - Why this option does not fit: The rate must reflect hemodynamic and organ tolerance.

B - Why this option does not fit: Preventable fluid accumulation can remain harmful despite stable creatinine.

C - Best answer: A positive balance alone does not establish a dialysis indication; treatment should address actual harm and tolerance.

D - Why this option does not fit: This ignores indications, risks and less invasive options.

Review link: teaching Section 5. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 091

A 56-year-old man with surgical sepsis has a substantial albumin-adjusted anion gap. Lactate is only mildly elevated, glucose is modestly raised and kidney function has deteriorated. He has prolonged poor intake and several medication exposures. The resident labels all of the gap lactic acidosis because infection is present and sees no reason to measure ketones or consider other retained acids. The surgeon must decide whether the measured contributor adequately accounts for the pattern and whether a second process changes treatment. Source assessment remains necessary, but the metabolic differential should not close merely because one plausible disease has already been identified. The diagnostic work remains parallel to urgent support and source control. A familiar disease can explain part of the pattern without explaining all of it, so the next test should address the residual gap rather than repeat an unsupported assumption.

What is the best approach?

A. Exclude ketoacidosis whenever glucose is only modestly elevated

B. Use bicarbonate as a diagnostic test for the cause

C. Assume infection makes every unmeasured anion lactate

D. Investigate additional gap contributors alongside lactate and the surgical source

Answer and option analysis

A - Why this option does not fit: Fasting and certain drugs can produce ketoacidosis without extreme hyperglycemia.

B - Why this option does not fit: A pH response to buffer does not identify the source of unmeasured acids.

C - Why this option does not fit: The measured lactate and broader context do not justify that conclusion.

D - Best answer: A high gap is not synonymous with lactate; ketones, renal retention, drugs or toxins may coexist.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 092

A 73-year-old woman has metabolic alkalosis after gastric drainage and loop diuretic exposure. Urine chloride is elevated in a sample collected soon after the latest diuretic. Potassium is low and volume status is mixed, with peripheral edema but possible reduced effective filling. The resident interprets the single urine value as proof that chloride depletion cannot contribute and proposes a mineralocorticoid diagnosis without reviewing timing. The surgeon must use the urine finding as contextual evidence rather than an infallible classifier, and select treatment from losses, medications, renal function and tolerance. Repeating assessment after relevant changes may alter the interpretation. The sample does not become useless because a diuretic was given, but its inference changes. The surgeon should use timing, repeated assessment and the actual loss history to decide whether the next treatment should provide chloride, change drugs or address another mechanism.

Which interpretation is best?

A. The single value definitively excludes a chloride-related mechanism

B. All alkalosis should be treated with unlimited saline regardless of congestion

C. Ignore potassium because it cannot sustain alkalosis

D. Recent diuretic exposure can confound urine chloride and must be integrated with the clinical pattern

Answer and option analysis

A - Why this option does not fit: Timing and drugs materially change urinary electrolyte handling.

B - Why this option does not fit: Composition and volume treatment must still fit tolerance.

C - Why this option does not fit: Potassium depletion can help maintain the disorder and requires safe correction.

D - Best answer: A high urine chloride soon after a loop dose does not exclude relevant gastric or diuretic-related chloride depletion.

Review link: teaching Section 8. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 093

A 60-year-old man with postoperative shock receives an initially defensible combination of fluid and norepinephrine while the source is investigated. After two hours, pressure is higher but mentation, skin perfusion and urine output worsen; oxygen need increases. The resident argues that the original plan was evidence-based and therefore should simply be repeated unchanged. The surgeon must distinguish an appropriate starting choice from proof that its continued repetition remains correct. New focused echo suggests a cardiac contribution, and abdominal source control remains uncertain. The next plan needs a revised explanation with measurable response targets rather than loyalty to the first diagnostic label. A documented expected response and stop criterion would make the next plan reviewable. The clinician should update the explanation from new findings and preserve a clear escalation path, rather than confuse evidence-based starting therapy with a requirement for endless repetition.

What is the best next decision?

A. Stop all treatment because the first plan failed

B. Declare recovery because pressure improved

C. Repeat the same therapy because the initial choice was defensible

D. Reassess the mechanism and source and change treatment from the observed benefit-harm profile

Answer and option analysis

A - Why this option does not fit: Failure requires revised support and definitive assessment, not therapeutic withdrawal by default.

B - Why this option does not fit: Flow and tissue perfusion can remain inadequate despite a supported pressure.

C - Why this option does not fit: Clinical response must update the prescription.

D - Best answer: A reasonable initial intervention can become inappropriate when response reveals mixed disease or intolerance.

Review link: teaching Section 9. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 094

A stable 68-kg man has adequate total water delivery from feeds, carriers and a small IV infusion. The resident declares the daily prescription complete without examining sodium, potassium or glucose content. Chemistry later shows falling potassium and increasing chloride, while the recorded volume remains close to the intended budget. Kidney function is stable and there are modest ongoing GI losses. The surgeon must explain why achieving a volume target does not necessarily meet composition needs and why new deficits require a safe route-specific plan. The solution is not automatically more liters; the existing sources may need adjustment, substitution or separate approved supplementation. Solute content can be changed through substitution and safe supplementation without necessarily increasing total volume. The next order should state which deficiency or excess it addresses, with a review time and a plan for ongoing measured gastrointestinal loss.

What is the best revision?

A. Increase total volume until every electrolyte normalizes

B. Audit actual electrolyte content and losses independently of the water total

C. Give concentrated bedside mixtures without compatibility review

D. Treat the recorded daily volume as proof of adequate replacement

Answer and option analysis

A - Why this option does not fit: This may worsen excess without correcting the composition mismatch.

B - Best answer: Water balance and composition are separate requirements; a correct volume can still deliver excess chloride and insufficient potassium.

C - Why this option does not fit: Safe concentrations, preparations and access remain necessary.

D - Why this option does not fit: It does not identify what solutes entered or left the patient.

Review link: teaching Section 6. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 095

A 65-year-old man is receiving potassium and magnesium replacement after high-output losses. As treatment progresses, the losses decrease but renal function worsens and urine output falls. The resident continues a standing replacement schedule because several earlier results were low. The latest chemistry is delayed, and additional potassium is also present in maintenance and phosphate therapy. The surgeon must determine whether historical depletion justifies automatic repeat dosing when both external loss and excretory capacity have changed. He has no current malignant arrhythmia requiring blind emergency administration, and a repeat sample with medication reconciliation can be obtained promptly. The previous low results remain relevant to the history but cannot determine the next dose in isolation. A repeat-dose decision must account for cumulative administration, current excretion and the disappearance of the earlier loss, with clear responsibility for reviewing the new result.

What is the best next step?

A. Stop all electrolyte surveillance once the original deficit was identified

B. Reassess current concentrations, clearance and all sources before repeat replacement

C. Ignore potassium delivered with phosphate

D. Continue automatically because low stores prevent overshoot

Answer and option analysis

A - Why this option does not fit: Treatment and changing physiology require ongoing reassessment.

B - Best answer: Changing losses and kidney function can convert a previously appropriate schedule into an accumulation risk.

C - Why this option does not fit: The accompanying ion contributes to the total prescribed dose.

D - Why this option does not fit: Reduced excretion and multiple sources can still produce dangerous excess.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 096

A 58-year-old woman with a high-output ileostomy becomes dizzy, oliguric and hyponatremic after drinking mostly water. Urine is concentrated. A resident diagnoses SIAD from the urine and orders restriction as the sole intervention, without assessing weight loss, postural findings or sodium-rich external output. The surgeon must distinguish appropriate antidiuresis from true inappropriate secretion and evaluate whether effective volume depletion is limiting water excretion. She has no seizure or coma, but the ongoing loss and AKI require prompt treatment. Replacement composition and subsequent correction speed must be monitored because restoring volume can abruptly change renal water handling. The patient's sodium trajectory can change quickly once appropriate volume is restored. A safe plan therefore includes the initial loss correction and surveillance for later aquaresis, rather than choosing restriction or saline from a concentrated urine value alone.

Which approach is best?

A. Diagnose SIAD from concentrated urine alone

B. Ignore correction speed after restoring volume

C. Evaluate and treat likely sodium-water depletion while monitoring the sodium response

D. Replace all losses with plain water

Answer and option analysis

A - Why this option does not fit: That finding does not distinguish the cause of antidiuresis.

B - Why this option does not fit: A water diuresis can accelerate sodium rise once the stimulus resolves.

C - Best answer: Concentrated urine can be appropriate in hypovolemia; restriction alone may fail to address the actual loss.

D - Why this option does not fit: The intestinal sodium loss and hyponatremia require composition-aware treatment.

Review link: teaching Section 7. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 097

A 64-year-old woman with septic shock initially has warm extremities and a hyperdynamic LV. Later, pressor needs rise, hands cool, and a repeat echo shows new depressed LV function. She is fluid positive and lung congestion has increased. The resident insists that the earlier distributive classification excludes a cardiac component and orders more fluid from the initial plan. The surgeon must recognize that phenotype can evolve during illness and treatment, while also reassessing ischemia, rhythm and source control. The new finding does not prove one specific myocardial diagnosis, but it should materially change the next flow and support assessment. The new bedside findings indicate that the old model no longer explains the whole circulation. Further information should identify the current flow limitation and guide support while evaluating the myocardial cause and the ongoing abdominal source in parallel.

What is the best working interpretation?

A. Give fluid solely because sepsis usually causes vasodilation

B. The first phenotype permanently determines all later therapy

C. New LV dysfunction proves acute coronary occlusion without further assessment

D. Evolving mixed distributive and cardiac dysfunction requires reassessment and tailored support

Answer and option analysis

A - Why this option does not fit: The current flow limitation and tolerance, not the general label, determine benefit.

B - Why this option does not fit: Circulatory physiology is dynamic and must be reevaluated.

C - Why this option does not fit: Several causes can depress function during severe illness.

D - Best answer: Shock mechanisms can coexist or change; new pump findings and intolerance invalidate automatic continuation of the early plan.

Review link: teaching Section 2. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 098

A 61-year-old man has recovered from an admission for high-output stoma-related depletion, hypomagnesemia and AKI. Symptoms and chemistry improve after IV treatment. The resident proposes discharge with only the instruction to drink more water, without reviewing current output, medication changes or follow-up laboratories. The surgeon knows the external loss has not fully resolved and that treatment success in hospital may not persist after IV support stops. The patient can understand a written plan and has access to a stoma service. The next decision should translate physiological recovery into a sustainable strategy rather than equate one normal sample with permanent correction. The discharge decision should verify that the plan remains effective after the final IV infusion is stopped. The patient needs practical instructions and timely access to review, with objective output and biochemical monitoring that can detect recurrence before severe depletion returns.

Which discharge plan is most defensible?

A. Recommend unrestricted plain water as the complete replacement plan

B. Continue the hospital IV prescription indefinitely without reassessment

C. Provide output-specific intake advice, medication review, monitoring and clear escalation criteria

D. Use one normal chemistry panel as proof no further review is needed

Answer and option analysis

A - Why this option does not fit: Water alone may not replace sodium-rich losses effectively.

B - Why this option does not fit: Long-term provision must fit current needs, feasible routes and ongoing monitoring.

C - Best answer: Persistent losses require a sustainable replacement and follow-up plan after IV treatment ends.

D - Why this option does not fit: The original mechanism can recur outside the hospital.

Review link: teaching Section 9. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 099

A 67-year-old woman has a carefully selected fluid challenge for possible preload-responsive hypoperfusion. Stroke volume does not improve, oxygen requirement rises, and venous congestion becomes more evident. Pressure increases slightly for a few minutes, which the resident calls success and uses to justify another identical challenge. The surgeon must decide whether the observed endpoint fulfilled the intended benefit and whether a pre-agreed stopping condition has been reached. The source has not yet been definitively controlled, and focused assessment suggests more than one mechanism. The decision should preserve necessary support and source treatment while preventing an ineffective therapy from continuing through habit. The current test provides evidence about both benefit and harm, and both should influence repetition. The surgeon should communicate why the bolus is being stopped and what alternative assessment or definitive intervention will address the unresolved perfusion problem next.

What is the best response?

A. Stop repeating the ineffective challenge and reassess mechanism and source

B. Stop all source evaluation because the challenge was completed

C. Use the small pressure rise as sufficient evidence of success

D. Repeat until venous pressure reaches a fixed target

Answer and option analysis

A - Best answer: Absent useful flow benefit plus worsening tolerance meets a reasonable stop rule despite a brief pressure rise.

B - Why this option does not fit: A therapeutic test does not provide definitive anatomical treatment.

C - Why this option does not fit: The intended flow benefit and emerging harm matter more than one transient number.

D - Why this option does not fit: A static pressure target does not establish beneficial flow or tolerance.

Review link: teaching Section 3. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

Question 100

A 70-year-old man after emergency bowel surgery has MAP 66 mm Hg on increasing support, a falling but still elevated lactate, oliguria, new hypoxemia and a distended tender abdomen. He has received fluid, blood and multiple electrolyte preparations. The handover contains no clear account of responses, source-control certainty, cumulative carriers or a plan for the next deterioration. One resident wants another bolus, another wants diuresis, and a third wants only a higher pressor dose. The surgeon must select the process that can resolve these competing prescriptions without assuming that one numerical endpoint defines recovery or that every unresolved problem has the same mechanism. The immediate plan should specify what must happen now, what information will discriminate competing mechanisms, and what change will trigger escalation. Each proposed therapy should have a measured purpose and stop condition so the team can learn from the next response.

Which immediate management framework is best?

A. Wait for all biochemical values to normalize before source review

B. Reassess source, mechanism, flow, perfusion and tolerance in parallel and define the next action

C. Choose whichever prescription most quickly raises MAP

D. Follow every proposed treatment simultaneously without endpoints

Answer and option analysis

A - Why this option does not fit: This delays the cause-directed action that may be needed for recovery.

B - Best answer: The competing observations require an integrated updated explanation with explicit treatment and reassessment priorities.

C - Why this option does not fit: A pressure-only goal can conceal low flow, intolerance or persistent surgical pathology.

D - Why this option does not fit: Conflicting unmeasured interventions can add harm and obscure their effects.

Review link: teaching Section 9. Reconsider the mechanism, timing, and treatment response before generalizing this answer to another patient.

100-question answer key

1-20	21-40	41-60	61-80	81-100
001: C	021: A	041: B	061: C	081: B
002: D	022: C	042: A	062: C	082: C
003: B	023: B	043: B	063: A	083: A
004: D	024: D	044: D	064: A	084: B
005: B	025: A	045: B	065: D	085: D
006: A	026: B	046: C	066: B	086: B
007: A	027: C	047: A	067: D	087: C
008: D	028: D	048: A	068: B	088: B
009: B	029: B	049: C	069: B	089: C
010: D	030: D	050: A	070: D	090: C
011: A	031: D	051: C	071: A	091: D
012: A	032: C	052: D	072: D	092: D
013: C	033: A	053: C	073: C	093: D
014: C	034: C	054: D	074: C	094: B
015: A	035: C	055: A	075: D	095: B
016: D	036: B	056: A	076: B	096: C
017: C	037: A	057: B	077: B	097: D
018: D	038: D	058: B	078: C	098: C
019: C	039: D	059: A	079: B	099: A
020: A	040: A	060: A	080: A	100: B

One point per best answer is suitable for a simple formative total out of 100. There is no validated pass mark. For teaching, record the reason for each error: mechanism, measurement validity, timing, treatment tolerance, calculation, or failure to reassess. A score alone does not measure operative competence.

11. Ten structured oral examination stations

Administration and scoring

Suggested format: 2 minutes to read and 8-10 minutes to discuss each station. The candidate page contains the opening scenario. The examiner page contains a new development, a model response and a case-specific rubric. Release the development after the candidate states an initial assessment and plan; ask neutral clarification questions rather than supplying missing decisions.

Each station has five domains worth 0-4 points each, for a total of 20. Apply the anchors below to the station-specific behaviors. Accept clinically defensible alternatives when justified by physiology, available resources and a clear reassessment plan. Exact wording is not required.

Points	Behavioral anchor
0	Omitted, materially incorrect, or unsafe despite neutral clarification.
1	Recognizes the issue only after substantial prompting; plan remains incomplete.
2	Identifies the main issue and a reasonable action but misses an important link, safeguard or contingency.
3	Independent, coherent response with minor omissions that do not undermine the immediate plan.
4	Independent, prioritized and justified response that integrates uncertainty, safety and reassessment.

Record critical safety concerns separately from the numerical score. A critical concern should trigger faculty review of the response and context; it is not an automatic psychometrically validated fail rule. The proposed concerns describe dangerous reasoning to explore, not a substitute for examiner judgment.

Total suggested formative score: 200 across 10 stations. These stations have not undergone standard setting, examiner reliability testing or outcome validation. Use the model answers as review guides and calibrate examiners before any high-stakes assessment.

Station 01: candidate brief

Occult hemorrhage with limited cardiac reserve

A 76-year-old man is six hours after complex abdominal surgery. He has ischemic cardiomyopathy, takes a beta blocker, and received substantial crystalloid. Pressure is 112/68 mm Hg, heart rate 82/min, and saturation is 98% on increasing oxygen. He is becoming inattentive, cool, and oliguric. Lactate rises from 1.8 to 4.5 mmol/L. Hemoglobin is 10.5 g/dL, only modestly lower than the initial postoperative result. The abdomen is enlarging and drain output is small. Focused echo shows poor LV function similar to baseline; the study cannot exclude new regional disease. The resident proposes diuresis because of crackles, while anesthesia asks whether another crystalloid bolus is needed. Imaging is available but requires transfer. The operating team and blood bank can mobilize immediately. You are the senior surgeon. State your immediate priorities and the decision that determines the route to definitive evaluation. Explain why the familiar pressure, heart rate, hemoglobin and EF do not resolve the case.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 01: examiner guide

Release after the initial plan

The pressure briefly improves with emergency blood, then falls again; oxygen needs rise and the abdomen becomes more distended. What changes in your assessment of transport and source control?

Model response

Recognize occult hypoperfusion and possible concealed hemorrhage with mixed cardiac limitation. Call senior anesthesia/critical-care and surgical support, verify monitoring, obtain targeted information and activate major hemorrhage resources when anticipated need warrants it. Normal pressure, beta-blocked heart rate and modest early hemoglobin change cannot exclude loss. Baseline poor EF affects tolerance but does not identify the new cause. Repeated transient responses favor ongoing disease; choose urgent control versus imaging from current stability, likely source, resource timing and safe transport. Avoid reflexive diuresis or repeated crystalloid without a defined benefit. Reassess perfusion, flow, respiratory tolerance, calcium, temperature and coagulation during support.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Recognition of compensated/mixed shock	Names at least two independent perfusion abnormalities and explains why apparently reassuring variables do not exclude hemorrhage.	0-4
Parallel resuscitation and escalation	Mobilizes surgical/anesthesia/blood resources promptly and supports circulation without delaying cause assessment.	0-4
Control-route judgment	Uses sustained stability and actual timing to justify imaging versus urgent intervention and supplies a fallback.	0-4
Tolerance and hemostatic review	Recognizes cardiac/respiratory limits and includes warming, calcium and goal-directed coagulation support when transfusion is substantial.	0-4
Reassessment and communication	States expected responses, stop conditions, unresolved source status and named reassessment responsibility.	0-4

Critical safety concerns: Waiting for profound hypotension before escalating; excluding bleeding from the drain or early hemoglobin; prescribing unlimited fluid without source review.

Teaching cross-reference: Section 1-4. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 02: candidate brief

An obstructive event during treatment of sepsis

A 58-year-old woman is ventilated after source treatment for abdominal sepsis. She had been improving, with lower support needs and better peripheral perfusion. A right subclavian catheter is inserted. Ten minutes later pressure falls markedly, oxygen saturation decreases, peak airway pressure rises and right-sided air entry becomes quieter. The ventilator alarm is sounding and the room is crowded. A radiograph has been requested but is several minutes away; the ultrasound machine is not at the bedside. A resident proposes escalating norepinephrine because the established diagnosis is septic shock. Another insists that pleural treatment cannot occur without a radiograph. You must direct the first minutes of care, explain what makes the new mechanism plausible and decide which checks should occur in parallel. The abdominal findings have not changed, but the pre-existing septic illness remains real. Explain how you would interpret improvement after a successful emergency intervention and what residual problems still require review.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 02: examiner guide

Release after the initial plan

After decompression, pressure and airway pressures improve but the patient remains on some support. The resident says the persistent requirement proves the intervention failed. Respond.

Model response

Recognize a new probable tension pneumothorax in an unstable procedural context. Call immediate help, check airway/tube/circuit in parallel, and arrange prompt decompression and definitive pleural management by a trained clinician under protocol without waiting for delayed radiography. Rapid available ultrasound can assist but must not create harmful delay. Preserve needed support and assess other procedural complications. A response supports the obstructive contribution, but residual sepsis, pump dysfunction or another problem may remain. Reassess ventilation, perfusion, imaging/pleural status, catheter function and source control. Do not interpret incomplete normalization as proof that the original action was wrong.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Reopens the mechanism	Connects procedure timing, unilateral findings and ventilator change to a new obstructive component.	0-4
Emergency action	Prioritizes timely trained decompression when clinical suspicion and instability justify it.	0-4
Parallel checks	Assigns airway/circuit checks and monitoring without using them to postpone the likely lifesaving action.	0-4
Mixed-physiology interpretation	Explains residual support after improvement without discarding either the obstruction or the septic illness.	0-4
Follow-up plan	Specifies definitive pleural care, reassessment and review of procedural complications and ongoing source status.	0-4

Critical safety concerns: Waiting for radiography despite convincing unstable tension physiology; attributing every new sign to sepsis; treating pressor escalation as definitive relief of obstruction.

Teaching cross-reference: Section 2-3. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 03: candidate brief

A positive preload test in a congested patient

A 67-year-old woman remains in intensive care after surgery for peritonitis. She is more interactive, capillary refill has improved and lactate is falling, but urine output remains low. She has gained weight, is several liters positive and needs increasing oxygen. Lung ultrasound shows more B-lines. A reliable passive leg raise increases stroke volume by 13%, returning to baseline afterward; pressure changes little. There is no obvious new pericardial or RV obstruction. The resident proposes repeated 500-mL boluses until the test becomes negative. Another recommends immediate aggressive diuresis solely from the balance chart. You must explain the three distinct questions of fluid need, responsiveness and tolerance, decide whether the current information supports automatic fluid, and identify what else would change the next prescription. Include the unresolved surgical source, catheter patency, renal injury and the possibility that urine recovery can lag other improvements.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 03: examiner guide

Release after the initial plan

The patient later has stable low support, persistent congestion and no continuing abnormal losses. How do you decide whether active removal is now appropriate?

Model response

The test supports responsiveness but does not establish a clinical need or acceptable tolerance. Do not repeat fluid solely from PLR positivity or oliguria. Reconcile all input, assess kidney trajectory/catheter, cardiac function, pulmonary differential and source-control adequacy. Select another intervention only for a defined expected benefit and stopping condition. Stop unnecessary maintenance and carriers. Removal readiness requires that acute resuscitation needs have resolved sufficiently, congestion is harmful and perfusion can tolerate the plan. Consider monitored diuretics where appropriate; do not begin routine extracorporeal removal from positive balance alone. Reassess pressure/support, examination, oxygenation, urine and electrolytes during any change.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Conceptual separation	Clearly distinguishes need, responsiveness and tolerance and interprets the little-changed pressure appropriately.	0-4
Rejects automatic prescriptions	Explains why neither repeat boluses nor aggressive removal follows from a single test or balance value.	0-4
Investigates oliguria and oxygen change	Includes catheter, AKI, congestion, cardiac and pulmonary causes rather than a one-cause assumption.	0-4
Phase-aware de-escalation	Stops unneeded input and states physiological readiness criteria before active removal.	0-4
Measurable review	Defines benefit, harm and reassessment for the chosen plan and preserves source-control review.	0-4

Critical safety concerns: Using PLR positivity as a mandatory series of boluses; forcing urine output despite intolerance; aggressive removal during escalating shock without reassessment.

Teaching cross-reference: Section 3-5. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 04: candidate brief

Pelvic hemorrhage with imperfect resource timing

A 35-year-old man arrives after a high-energy collision. Pressure is 74/42 mm Hg, heart rate 128/min, and he is restless. The pelvis is painful and suspicious for instability; stabilization is applied. No major external bleeding is visible. Focused abdominal ultrasound is negative and hemoglobin is 12.3 g/dL. Emergency blood produces only a transient improvement. Lactate rises, core temperature is 34.5 C, ionized calcium is low and fibrinogen is 1.1 g/L. The CT scanner is available, interventional radiology needs 35 minutes to mobilize and the operating team can begin earlier. The resident wants CT before any decision. You must lead resuscitation, explain the negative tests, choose a route based on current physiology and actual resources, and describe how initial product organization will transition to targeted therapy. State the information needed before applying a permissive-hypotension strategy.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 04: examiner guide

Release after the initial plan

New information establishes a severe associated brain injury. How does this modify your pressure plan without delaying bleeding control?

Model response

Treat probable ongoing major hemorrhage; negative abdominal ultrasound does not exclude pelvic/retroperitoneal loss and early hemoglobin is not an exclusion test. Activate protocol and coordinate trauma, anesthesia, blood bank, operating and interventional teams. Use the fastest appropriate local pelvic-control pathway with fallback and staged embolization if needed; recurrent instability argues against a routine imaging detour. Warm, monitor and correct calcium, replace fibrinogen and other demonstrated deficits, and assess trauma TXA timing from injury. Pressure goals must account for brain injury and other organ vulnerability rather than copy a low target indiscriminately. Control and support proceed together; deactivate automatic packs when their indication ends.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Recognizes ongoing hemorrhage	Explains transient response and the limitations of ultrasound and hemoglobin.	0-4
Resource-based control plan	Chooses and justifies a timely pelvic pathway, including contingency rather than indefinite waiting.	0-4
Organized hemostatic support	Includes protocol activation, warming, calcium, fibrinogen and transition to current-result-guided treatment.	0-4
Population exceptions	Uses injury-based TXA timing and modifies permissive hypotension for cerebral injury.	0-4
Communication and deactivation	Provides blood-bank updates, response checks and explicit stop/restart criteria.	0-4

Critical safety concerns: Waiting for a retrospective transfusion threshold; assuming a negative ultrasound excludes pelvic loss; applying a low pressure target unchanged with severe brain injury.

Teaching cross-reference: Section 4. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 05: candidate brief

Gastric replacement in a patient with edema

A 78-year-old woman weighing 56 kg has prolonged nasogastric drainage after surgery for obstruction. She has heart failure and CKD. Output was 1.2 L over the last day but is now increasing. A standing saline infusion at 100 mL/h is labeled maintenance, with additional medication carriers and intermittent potassium. Weight has risen, ankles are edematous and oxygen need is increasing. Sodium is 139 mmol/L, potassium 3.0, chloride 91 and bicarbonate 34; creatinine has worsened. The chart does not separate rescue fluid, maintenance and replacement. One resident wants to stop all fluids, another wants to replace the full prior day's output immediately. You must reconstruct the account, explain how depletion and edema can coexist and write the components of a safe revised plan. Include what the normal sodium does and does not establish.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 05: examiner guide

Release after the initial plan

After the underlying obstruction is treated, gastric output falls sharply and oral intake improves. What orders must be revisited immediately?

Model response

Separate ordinary needs, prior deficit/excess and current measured losses. Count all intake and determine what output has already been replaced. Normal sodium does not establish normal extracellular sodium or filling. Assess perfusion, congestion and kidney function; the alkalosis/hypochloremia and hypokalemia fit a gastric-loss contribution. Individualize a lower maintenance budget after subtracting carriers and enteral intake. Prescribe composition-aware replacement over defined intervals with safe potassium and magnesium assessment, renal adjustment and early review of respiratory tolerance. Treat the cause and review suction need. When output falls and oral intake rises, stop obsolete replacement and reduce unnecessary maintenance rather than copy yesterday's rate.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Accounting	Separates maintenance, correction, current replacement and hidden intake without double counting.	0-4
Physiological interpretation	Explains normal sodium, chloride-depletion alkalosis and coexistence of edema with specific deficits.	0-4
Safe prescription	Specifies composition, interval, renal/potassium safeguards and individualized volume rather than a fixed large catch-up dose.	0-4
Cause-directed care	Reassesses obstruction/ileus, drain function and suction necessity.	0-4
Dynamic revision	Updates orders from falling losses and increasing oral intake with explicit monitoring.	0-4

Critical safety concerns: Equating edema with absence of electrolyte deficit; replacing historical output without checking prior replacement; leaving potassium unchanged during worsening renal clearance.

Teaching cross-reference: Section 6. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 06: candidate brief

Hyponatremia rescue and overcorrection

A malnourished 64-year-old man has sodium 112 mmol/L and potassium 2.6 after prolonged poor intake and diuretic exposure. He develops a seizure and reduced consciousness. Emergency airway/seizure care is underway. Explain the immediate sodium treatment goal, why it differs from normalization, how you determine correction safety when duration is uncertain, and what variables must be monitored. After initial rescue he improves at sodium 117. Volume restoration and potassium treatment continue. Several hours later urine becomes copious and dilute, and sodium reaches 121 within eight hours of baseline despite stopping hypertonic saline. The resident recommends waiting until morning because the patient is now alert. You must identify the new threat, decide how quickly to act and explain why the lack of continuing saline does not guarantee safety.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 06: examiner guide

Release after the initial plan

The next sodium is still rising. Describe the principles of a specialist relowering strategy and how you coordinate necessary potassium correction.

Model response

Provide monitored protocol-based hypertonic rescue for severe neurological symptoms, aiming for a limited initial rise. Treat uncertain duration cautiously and recognize malnutrition/hypokalemia as high-risk features. Set an intended rise below the conservative ceiling and monitor sodium and urine closely. The observed nine-point rise in eight hours with aquaresis is dangerous in this context. Stop avoidable correction-driving inputs and obtain urgent endocrine/renal/critical-care help. Expert use of desmopressin and controlled electrolyte-free water may halt and reverse excessive correction, with frequent reassessment. Potassium contributes to the sodium trajectory and must be coordinated, not ignored or indefinitely abandoned. Investigate and treat the underlying cause.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Rescue judgment	Treats severe symptoms promptly while avoiding immediate normalization.	0-4
Risk-adjusted limits	Recognizes uncertain chronicity and high-risk factors and distinguishes target from maximum ceiling.	0-4
Trajectory recognition	Identifies aquaresis and overcorrection despite stopped saline and improved consciousness.	0-4
Expert response	Escalates promptly and describes controlled desmopressin/water principles with frequent testing.	0-4
Coupled therapy	Includes potassium, total inputs, urine and underlying cause in one correction plan.	0-4

Critical safety concerns: Restriction alone for seizure; aiming directly for sodium 140; waiting overnight despite rapid high-risk correction; ignoring the sodium effect of potassium replacement.

Teaching cross-reference: Section 7. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 07: candidate brief

Hyperkalemia, rebound and delayed hypoglycemia

A 70-year-old woman with oliguric AKI has confirmed potassium 7.0 mmol/L and QRS widening after surgery. Potassium-containing fluids and an implicated medication are still ordered. Describe the first actions, the purpose of calcium, shifting treatment, elimination and the monitoring plan. She receives appropriate initial therapy and potassium falls to 5.6 at one hour. Urine remains minimal and cell injury may be ongoing. Three hours later she becomes sweaty and confused. The resident says glucose given with insulin excludes hypoglycemia and proposes another insulin dose from the old potassium result. You must respond to the new symptoms while maintaining a durable potassium strategy. Include how preparation-specific calcium dosing and access safety differ from simply ordering an equal volume of another calcium salt.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 07: examiner guide

Release after the initial plan

Repeat potassium later rises again despite early improvement. What determines the urgency of renal replacement assessment?

Model response

Use continuous monitoring and urgent appropriate cardiac protection for ECG toxicity; calcium stabilizes but does not lower potassium. Stop inappropriate sources, provide protocol-based insulin/glucose and suitable adjunct shifting therapy, and arrange elimination. Check potassium repeatedly and monitor glucose for delayed hypoglycemia, especially with kidney impairment. New sweating/confusion needs immediate glucose assessment and treatment, not an automatic repeat insulin dose. Ongoing severe or refractory potassium excess with poor clearance requires urgent renal/dialysis review. Verify current results, continuing release, access and preparation-specific elemental calcium. Early improvement buys time; it does not prove total-body removal or durable control.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Emergency sequence	Differentiates myocardial protection, intracellular shift and elimination and acts urgently.	0-4
Source control	Reconciles potassium input, drugs and ongoing cell release.	0-4
Monitoring	Plans repeated potassium/ECG review and sustained glucose surveillance.	0-4
Responds to evolution	Checks new hypoglycemia symptoms and anticipates rebound instead of using an old result.	0-4
Definitive clearance and preparation safety	Escalates renal replacement appropriately and distinguishes calcium formulations/access.	0-4

Critical safety concerns: Using calcium as elimination; stopping surveillance after one improved potassium; repeating insulin without glucose review; delaying severe refractory hyperkalemia care for absent urine recovery.

Teaching cross-reference: Section 7. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 08: candidate brief

Refeeding with linked electrolyte failure

A 55-year-old woman has severe weight loss after weeks of intestinal illness. Feeding and dextrose-containing infusions are increased after surgery. Within 36 hours she develops weakness, edema, ventricular ectopy and difficulty sustaining ventilation. Potassium is 2.6 mmol/L despite replacement, magnesium 0.40 and phosphate 0.21; ionized calcium is reduced. Creatinine is rising. Before feeding, phosphate was in the reference interval. The resident attributes weakness to deconditioning and proposes potassium alone while continuing full calorie advancement. You must identify the syndrome and alternatives, explain why the baseline result does not exclude depleted stores, and coordinate urgent organ support with nutrition and electrolyte treatment. Include the contribution of carriers, thiamine, renal clearance and the accompanying ions in replacement products.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 08: examiner guide

Release after the initial plan

The stocked phosphate preparation contains potassium, but the next potassium result rises rapidly as urine falls. How does the prescription change?

Model response

Recognize probable refeeding-related electrolyte shifts and organ dysfunction, while evaluating concurrent sepsis, respiratory and cardiac causes. Provide monitoring and respiratory assessment. Correct severe phosphate/magnesium deficits alongside potassium through safe renal-adjusted protocols; magnesium can sustain refractory potassium/calcium abnormalities. Review all nutrient sources, provide thiamine promptly and reduce or pause advancement according to severity with specialist input. A normal prefeeding concentration does not establish normal stores. Count potassium in phosphate and other sources; change preparation and dose when potassium or clearance changes. Monitor calcium, compatibility, fluid accumulation and repeated results. A sustainable feeding plan is essential after stabilization.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Syndrome recognition	Links timing, poor intake, combined deficiencies and organ effects while preserving a differential.	0-4
Immediate support	Addresses arrhythmia and respiratory weakness with appropriate monitoring and escalation.	0-4
Integrated replacement	Treats phosphate/magnesium/potassium together with renal, calcium and compatibility safeguards.	0-4
Nutrient plan	Includes thiamine, all carbohydrate sources and severity-adapted advancement.	0-4
Evolving prescription	Reassesses accompanying ions and repeat doses when potassium and urine output change.	0-4

Critical safety concerns: Continuing full feeding unchanged during severe evolving derangement; potassium-only correction; assuming baseline normal phosphate excludes risk; ignoring potassium in phosphate preparations.

Teaching cross-reference: Section 7. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 09: candidate brief

Alkalemia masking a high-gap surgical emergency

A 72-year-old man after surgery for obstruction has worsening abdominal pain, tenderness and declining urine output despite supported pressure. He has persistent gastric suction. Arterial pH is 7.46, PaCO2 29 mm Hg and bicarbonate approximately 20 mmol/L. Contemporaneous sodium is 140, chloride 94 and albumin 2 g/dL; lactate is high. A resident calls this isolated anxiety-related respiratory alkalosis and proposes observation because the patient is not acidemic. Calculate the measured and albumin-adjusted gap, test respiratory compensation and assess whether another metabolic process is plausible. Explain the limitations of the inference and how it changes the urgency of anatomical assessment. You must decide whether imaging is safe and useful or whether the trajectory requires another route to intervention.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 09: examiner guide

Release after the initial plan

The pressure improves further with support while abdominal pain worsens. Does this reduce concern about regional ischemia?

Model response

Measured gap is 26 and adjusted gap approximately 31. With bicarbonate 20, Winter's expected PaCO2 is 38 plus or minus 2; actual 29 indicates an additional respiratory alkalosis. The large gap rise relative to the small bicarbonate fall suggests concurrent metabolic alkalosis, plausible with gastric losses, while baseline and timing limit certainty. Alkalemia does not exclude serious high-gap acidosis or ischemia. Escalate surgical assessment, evaluate bowel viability, obstruction, leak and other sources, and select safe imaging versus intervention from current stability and likely benefit. Improved global pressure cannot certify regional perfusion. Treat the sources and reassess gas, electrolytes and response.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Arithmetic	Calculates measured gap 26 and albumin-adjusted gap about 31 with correct units/convention.	0-4
Compensation	Calculates expected CO2 38 plus or minus 2 and identifies extra respiratory alkalosis.	0-4
Mixed metabolism	Explains likely additional alkalosis and limitations rather than overclaiming a proven cause.	0-4
Clinical priority	Connects the pattern and focal findings to urgent source/ischemia assessment.	0-4
Decision and reassessment	Justifies the control/imaging route and does not use improved pressure as regional reassurance.	0-4

Critical safety concerns: Declaring no acidosis from alkalemia; attributing all findings to anxiety; delaying source assessment until laboratory normalization; prescribing fluid solely from base deficit.

Teaching cross-reference: Section 8. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.

Station 10: candidate brief

Loss of respiratory compensation during severe acidemia

A 62-year-old woman with abdominal sepsis and AKI was initially alert and breathing rapidly. Bicarbonate was 10 mmol/L and PaCO2 24. After analgesia and progressive illness, she becomes drowsy with less respiratory effort. The new gas shows pH 7.02, bicarbonate 10 and PaCO2 40. Oxygen saturation is 98% on supplemental oxygen. The resident calls CO2 normal and proposes bicarbonate followed by routine morning review. Another prepares a standard induction and routine ventilator settings without discussing the prior minute ventilation. You must interpret the mixed disorder, direct urgent assessment and describe an airway plan that avoids a preventable physiological collapse. Include hemodynamic preparation, the role and limitations of bicarbonate, continuing source review and renal-replacement assessment when indicated.

Your task

Present a prioritized assessment and immediate plan. Explain the competing mechanisms, justify the next decision, identify what must not be delayed, and specify how benefit and harm will be reassessed. State assumptions and resource-dependent alternatives explicitly.

The examiner will then provide additional information. Adapt the plan and explain which previous assumptions remain valid and which must change.

Station 10: examiner guide

Release after the initial plan

After intubation the pressure improves, but lactate and abdominal findings do not. How do you prevent the airway success from closing the source-control question?

Model response

Expected PaCO2 is 23 plus or minus 2; a value of 40 shows superimposed respiratory acidosis, supported by falling effort and consciousness. Saturation does not establish adequate ventilation. Call experienced airway/critical-care help, assess reversible drug and respiratory contributors, prepare circulation, minimize avoidable apnea and provide sufficient safe postintubation ventilation with early gas reassessment. Bicarbonate may be considered in selected severe metabolic acidemia with significant AKI, but cannot replace ventilation or source treatment; consider sodium/volume, calcium and CO2 effects. Review definitive renal indications and the unresolved abdominal source. A successful airway corrects one component, not every cause of shock.

Case-specific rubric: 20 points

Domain	Evidence for full credit	Points
Interpretation	Correctly derives expected CO2 and identifies an additional respiratory acidosis.	0-4
Urgency	Escalates drowsiness/falling effort promptly despite normal saturation.	0-4
Airway physiology	Plans circulation, minimized interruption and adequate safe ventilation rather than a routine normal-CO2 target.	0-4
Adjunct judgment	Describes selected bicarbonate and renal-replacement considerations without universal claims.	0-4
Continuing source care	Separates respiratory success from regional perfusion/source control and states repeat assessment triggers.	0-4

Critical safety concerns: Calling CO2 40 appropriate compensation; suppressing breathing without replacement ventilation; delaying airway review until morning; treating bicarbonate or improved pressure as definitive source control.

Teaching cross-reference: Section 8-9. Apply the shared behavioral anchors; document any accepted alternative and the candidate's justification.