**Introduction**

1. **Definition**
   1. Hypovolemic shock is a life-threatening medical emergency characterized by extensive intravascular volume loss leading to inadequate tissue perfusion, cellular hypoxia, and multi-organ dysfunction. It is categorized as a type of circulatory shock, along with cardiogenic, obstructive, and distributive shock. The hallmark of hypovolemic shock is a severe reduction in effective circulating blood volume and preload that impairs cardiac output, oxygen delivery, and vital organ perfusion. If left untreated, this hypoperfusion state can rapidly progress to irreversible organ injury, metabolic crisis, and death. Thus, prompt recognition and aggressive resuscitation are imperative in hypotensive patients with clinical suspicion for hypovolemic shock.

1. **Epidemiology**
   1. Hypovolemic shock accounts for approximately one-third to 40% of shock cases presenting to acute care settings. This makes it the most common shock state encountered in clinical practice. The overall mortality rate for hypovolemic shock ranges widely from 20% to 60%, depending on the underlying etiology, presence of comorbidities, and time elapsed prior to definitive treatment. Trauma patients with hemorrhagic shock have reported mortality rates around 36-44%. Among elderly patients, the risk of death is significantly higher, with mortality up to 70% in some studies. Delays in diagnosis and therapy also negatively impact outcomes. Each hour elapsed before initiation of treatment is associated with a measurable rise in morbidity and mortality. This underscores the necessity for rapid identification and management of hypovolemic shock across all patient populations.

**C. Etiology**

The precipitating causes of hypovolemic shock can be divided into two major categories: hemorrhagic and non-hemorrhagic etiologies.

1. **Hemorrhagic Causes**
   1. Hemorrhage, or significant blood loss, is the most common cause of hypovolemic shock. Trauma is the leading source of hemorrhagic shock, including both blunt and penetrating mechanisms. Other major etiologies include gastrointestinal bleeding from ulcers, varices, or irritable bowel disease; post-operative hemorrhage; retroperitoneal bleeds; ruptured abdominal aortic aneurysms; obstetric causes such as uterine atony or placental abruption; and coagulopathy disorders leading to increased blood losses.

1. **Non-Hemorrhagic Causes**
   1. Hypovolemia can also develop due to non-hemorrhagic fluid losses. Examples include dehydration from inadequate fluid intake, excessive vomiting or diarrhea, diabetes insipidus, diuretic use, and transcellular fluid shifts as seen in burns, pancreatitis, sepsis, anaphylaxis, and adrenal crisis. The pathophysiologic consequences are similar regardless of whether intravascular depletion occurs through hemorrhage versus non-hemorrhagic mechanisms. However, management strategies may differ depending on the etiology.

**Pathophysiology**

1. **Stages of Hypovolemic Shock**
   1. The body responds to hypovolemia and decreased circulating volume through a series of compensatory mechanisms. These stages of cardiovascular compensation are classically described as follows:
2. **Compensated Shock**
   1. Initially, sympathetic nervous system activation leads to increased heart rate and myocardial contractility to maintain cardiac output. Simultaneously, catecholamine release triggers vasoconstriction of arterioles which raises systemic vascular resistance in an attempt to preserve perfusion pressure. Together, these responses help compensate for reduced intravascular volume to stabilize hemodynamics. Patients may only exhibit mild tachycardia and diaphoresis as the main clinical signs during this stage.
3. **Decompensated Shock**
   1. As hypovolemia worsens and compensatory mechanisms are overwhelmed, signs of cardiovascular decompensation develop. Blood pressure declines as vasoconstriction fails to compensate for decreased preload and cardiac output. Tissue hypoperfusion leads to lactic acidosis and end-organ dysfunction. Patients exhibit hypotension, tachypnea, altered mental status, cool extremities, and oliguria. This decompensated stage of shock is associated with high mortality unless aggressive resuscitation is initiated.
4. **Irreversible Shock**
   1. Prolonged hypotension that is untreated will ultimately result in irreversible shock. Widespread cellular dysfunction and death secondary to hypoxia leads to multi-organ failure. At this stage, even optimal therapy cannot reverse the tissue damage and patient demise rapidly ensues.

**B. Systemic Effects of Hypovolemic Shock**

The reduction in preload, cardiac output, and oxygen delivery associated with hypovolemic shock has detrimental effects on the function of multiple organ systems:

**Cardiovascular**

* Decreased preload reduces ventricular filling pressures and stroke volume
* Cardiac output declines in parallel with falling blood volume
* Myocardial contractility is impaired due to cellular hypoxia
* Baroreceptor-mediated vasoconstriction increases systemic vascular resistance to shunt blood towards vital organs

**Respiratory**

* Tachypnea develops as respiratory compensation for metabolic acidosis
* Decreased pulmonary compliance due to interstitial edema

**Renal**

* Diminished renal arterial perfusion causes decreased glomerular filtration
* Oliguria results from intra-renal vasoconstriction and volume depletion
* Risk of acute tubular necrosis and kidney injury

**Gastrointestinal**

* Splanchnic vasoconstriction leads to reduced mucosal perfusion
* Ileus and gastric dilation due to hypoperfusion

**Neurologic**

* Cerebral hypoperfusion results in anxiety, confusion, and lethargy
* Loss of consciousness and seizures may occur as shock worsens

**Hematologic**

* Hemodilution and dilutional coagulopathy develop from fluid administration
* Impaired platelet function exacerbates coagulation defects

**Metabolic**

* Global tissue hypoxia causes increased anaerobic metabolism and lactic acidosis
* Hepatic hypoperfusion can worsen acidemia
* Hypothermia frequently develops due to heat loss and impaired thermoregulation

**III. Diagnosis of Hypovolemic Shock**

**A. Presentation**

**History**

Key elements of the history in hypovolemic shock patients include recent trauma, GI bleeding, surgery, nausea/vomiting, fever, bloody stools or emesis, uncontrolled diabetes, ingestion of diuretics, burns, allergy symptoms, and orthostatic symptoms. Medication reconciliation should assess for anticoagulants, antiplatelets, and chronic diuretic use

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1. Physical Exam

* Tachycardia, hypotension
* Cool, clammy skin, delayed capillary refill
* Flat neck veins suggestive of volume depletion
* Dry mucous membranes from poor perfusion
* Altered mental status due to cerebral hypoperfusion
* Abdominal pain or distension, absent bowel sounds concerning for mesenteric ischemia
* Focused exam to identify sources of hemorrhage (i.e. trauma, GI bleeding, post-op, obstetric)

**Vital Signs**

Tachycardia and hypotension are classic hallmarks of hypovolemic shock. However:

* Pediatric patients can maintain normal blood pressures despite significant intravascular volume losses >40% due to cardiovascular compensation.
* Elderly patients may not mount an appropriate tachycardic response due to medications or cardiovascular disease.
* Patients on beta-blockers may not manifest tachycardia.
* Baseline hypertension can obscure hypotension. Mean arterial pressure (MAP) should be evaluated relative to a patient's normal values. Any acute drop in MAP >20-30 mmHg should raise suspicion.

**Hemorrhagic Shock Classification**

* Hemorrhagic shock is classified according to four grades of severity based on the volume of acute blood loss (see Table 1). This stratification helps guide appropriate resuscitation efforts.
* Class I hemorrhage involves up to 15% loss of circulating blood volume. Compensatory vasoconstriction maintains normal vital signs without clinical evidence of shock. Class II reflects 15-30% volume loss. Tachycardia develops but blood pressure is typically still maintained. Class III designates loss of 30-40% of blood volume. Significant tachycardia plus hypotension indicates decompensation into significant shock. Immediate fluid and blood product resuscitation is warranted at this stage. Finally, Class IV hemorrhage entails extreme blood losses exceeding 40% of circulation volume. Marked hypotension and end organ damage are evident. Urgent, aggressive intervention is required to avoid demise.
* This classification system underscores the importance of trending compensatory physiologic responses in addition to standard vital signs. Tachycardia precedes hypotension as an early sign of volume loss, while younger patients can maintain blood pressure despite significant hemorrhage volumes. Clinical correlation is essential for rapid triage and management.

**Table 1. Classification of Hemorrhagic Shock**

|  |  |  |  |
| --- | --- | --- | --- |
| Class | Blood Loss (%) | Pulse Rate | Blood Pressure |
| I | <15% | <100 bpm | Normal |
| II | 15-30% | >100 bpm | Normal |
| III | 30-40% | >120 bpm | Decreased |
| IV | >40% | >140 bpm | Decreased |

**B. Diagnostic Evaluation**

1. Laboratory Studies
   1. Complete blood count: Monitor hemoglobin/hematocrit for ongoing losses or hemodilution after resuscitation. Thrombocytopenia may reflect disseminated intravascular coagulation.
   2. Basic metabolic panel: BUN/Cr for renal function. Hyperkalemia can develop from tissue breakdown.
   3. Lactate: Elevated lactate >2-4 mmol/L indicates hypoperfusion and anaerobic metabolism. Trend levels to monitor resuscitation.
   4. Coagulation panel: PT, PTT, and INR to detect dilutional coagulopathy, DIC, or anticoagulant effect.
   5. Arterial or venous blood gas: Evaluate pH for metabolic acidosis. Assess lactate levels.
   6. Type and crossmatch if transfusion anticipated
2. Imaging
   1. Chest x-ray: Evaluate for tension pneumothorax, wide mediastinum concerning for AAA.
   2. CT angiogram: Identify source of hemorrhage if not clinically apparent.
   3. FAST exam: Detect free intraperitoneal or pericardial fluid concerning for ongoing hemorrhage.
3. Thromboelastography
   1. Thromboelastography (TEG) provides rapid assessment of clot initiation, strength, and breakdown to guide blood product needs during massive transfusion. It has advantages over traditional coagulation tests that are slowly returned from the lab. TEG allows goal-directed, real-time treatment of coagulation defects identified at the bedside. Specific parameters that can be trended include R time reflecting enzyme deficiencies, alpha angle showing fibrinogen levels, and MA demonstrative of platelet function. Correction of these abnormalities with the appropriate blood component targeted to TEG results has been shown to reduce transfusions, cost, and improve mortality compared to empiric component therapy. Where available, TEG should be incorporated into massive transfusion protocols to provide precise, patient-specific coagulation management.

**IV. Overview of Management**

**Resuscitation Goals**

Monitor endpoints during resuscitation to assess efficacy and guide interventions:

* Vital signs: MAP >65 mmHg, HR <100 bpm, or SBP >90 mmHg (permissive hypotension)
* Urine output >0.5 mL/kg/hr
* Improving lactate clearance
* Base deficit reduction
* Hemoglobin 7-9 g/dL
* Normalization of PT, PTT, INR

**Permissive Hypotension**

* Permissive hypotension strategies allow blood pressure to be maintained at lower than normal levels in hemorrhagic shock patients until surgical hemostasis can be achieved. This prevents disruption of newly formed blood clots at sites of vascular injury that could occur with the sudden increase in blood flow and pressure induced by aggressive fluid resuscitation. The target blood pressure utilized varies based on patient factors, but commonly a threshold MAP of 50-70 mmHg is used as a guide, as long as the patient has intact neurological function and capacity for end organ perfusion at this lower blood pressure level. After definitive control of bleeding has been obtained, standard resuscitation goals can then be implemented.
* Permissive hypotension can be achieved either by withholding or limiting the volume of resuscitative fluids administered, or by utilizing pharmacological agents such as calcium channel blockers or beta blockers to prevent the compensatory tachycardia and vasoconstriction that would otherwise increase blood pressure. This approach has demonstrated reductions in blood loss, hemorrhage duration, transfusion requirements, and mortality in animal models of hemorrhagic shock. However, human data is limited to small studies showing decreased blood loss intraoperatively when permissive hypotension was utilized. Its use should be restricted to the period prior to surgical hemostasis, and may be most beneficial in trauma centers where rapid control of hemorrhage can be achieved. It is generally not advised in patients with concomitant neurological injury where maintenance of cerebral perfusion pressure is necessary to avoid secondary brain injury.

**V. Management of Hypovolemic Shock**

**A. Control Hemorrhage**

Identify and control any sources of external or internal bleeding as the first priority. This may require emergent surgery, interventional radiology procedures, or endoscopy. Apply direct pressure to external wounds. Avoid overly aggressive fluid resuscitation until bleeding is controlled, as this can worsen hemorrhage.

**B. Airway Protection**

Ensure patent airway. Intubate if necessary to protect airway, provide respiratory support, and enable adequate oxygenation/ventilation.

**C. Fluid Resuscitation**

Administer IV fluids to restore intravascular volume. Choices include:

Isotonic Crystalloids

* Normal saline
* Lactated Ringer's solution

Crystalloids are first-line due to low cost and wide availability. However, only 20-30% of infused volume stays intravascularly. Large volumes may be required, increasing risks of complications like pulmonary edema, abdominal compartment syndrome, and tissue edema.

**Hypertonic Saline**

3-5% NaCl solutions draw interstitial water into the intravascular space via osmosis. This can expand volume with smaller fluid volumes compared to crystalloids. However, hypertonic saline has not shown clear outcome benefits and is not routinely recommended.

Colloids

* Albumin
* Hetastarch

Colloids are suspended in crystalloid solutions but have larger molecular weights that resist extravasation into the interstitium. This allows more efficient plasma volume expansion compared to crystalloids alone. However, colloids are significantly more expensive and have not demonstrated reduced mortality or duration of shock compared to crystalloids. Adverse effects include coagulopathy and acute kidney injury. Routine use is not recommended.

**Restrictive Crystalloid Volumes**

Excessive crystalloid fluid administration is associated with complications such as pulmonary edema, abdominal compartment syndrome, tissue edema, and worsening of coagulopathy due to hemodilution. Restrictive fluid resuscitation seeks to limit these adverse effects by using the minimum volume necessary to maintain perfusion until bleeding can be controlled definitively. This prevents dislodgement of clot formation and reduces the risk of dilutional coagulopathy. Volumes up to 1-2 liters are generally utilized for initial resuscitation until blood products are available. However, the degree of restriction needs to be carefully tailored to each patient’s hemodynamic status. Evidence supporting improved outcomes with restrictive volumes is still lacking, outside of the setting of traumatic brain injury where judicious fluids are clearly beneficial to avoid cerebral edema.

**Blood Products**

Packed red blood cells, fresh frozen plasma, platelets, and cryoprecipitate replenish both volume and oxygen carrying capacity. Transfuse if:

* Hemorrhage results in >30% estimated blood volume loss
* Evidence of hemodynamic instability
* Ongoing bleeding and anticipated continued losses

Monitor for complications such as hypothermia, acidosis, electrolyte abnormalities, and transfusion reactions. Employ strategies to mitigate these adverse effects.

**Damage Control Resuscitation**

Damage control resuscitation employs a strategic approach tailored to the specific needs of patients at high risk for hemorrhagic shock based on injury pattern, degree of blood loss, or unstable vital signs on presentation. It is utilized for trauma patients with massive hemorrhage where conventional resuscitation techniques would be inadequate or potentially detrimental. The overarching goals of damage control resuscitation are to minimize blood loss, restore tissue perfusion, prevent exacerbation of coagulopathy, and reduce the systemic inflammatory response by utilizing permissive hypotension, restrictive crystalloid volumes, early initiation of balanced blood product ratios, and treatment of acidosis.

**Massive Transfusion Protocols**

Massive transfusion protocols help standardize delivery of blood products in appropriate ratios. They improve communication between clinicians and the blood bank to accelerate the provision of blood components. Key triggers include blood loss >1 blood volume within 24 hours or >50% volume loss in 3 hours. Transfusion of >10 units PRBCs in 24 hours is another commonly used indicator to activate institutional massive transfusion protocols.

Most centers also define clinical criteria such as hypotension, tachycardia, and positive FAST exam. Massive transfusion protocols provide pre-defined orders for release of PRBCs, plasma, platelets, and cryoprecipitate in a 1:1:1 or similar ratio for continued transfusion until hemorrhage is controlled. This saves crucial time in a dynamic resuscitation. Compared to conventional component therapy, MTPs effectively increase plasma and platelet administration, reduce crystalloid volumes, correct coagulopathy sooner, and improve survival for massively bleeding patients.

**Early Balanced Blood Product Ratios**

Traditional approaches did not emphasize early use of plasma, platelets, and cryoprecipitate. However, major hemorrhage rapidly depletes both cellular and coagulation factors. Excessive crystalloid resuscitation compounds this effect. Transfusing only packed red blood cells fails to correct the ensuing coagulopathy. This can exacerbate bleeding and increase morbidity and mortality. Current best practices utilize early release of balanced ratios of blood products in conjunction with pRBCs.

Observational data suggests improved survival with plasma-to-pRBC ratios of 1:1 to 1:2 and platelet-to-pRBC ratios approaching 1:1. Although optimal targets are still under investigation, transfusion of all components in a 1:1:1 ratio is now frequently employed by trauma centers. This balanced transfusion helps replenish both oxygen carrying capacity as well as correct coagulation defects. It reduces the severity of trauma-induced coagulopathy and need for massive transfusion. Overall mortality is improved compared to prior practices when only pRBCs were emphasized.

**Treatment of Acidosis**

Metabolic acidosis from hypoperfusion and lactic acid accumulation impairs coagulation and platelet function, while also reducing cardiac contractility. This aggravates shock and worsens outcomes. Buffering agents such as sodium bicarbonate should be administered early in the resuscitative process to help correct acidemia. Adequate ventilation to provide acid-base homeostasis should also be ensured.

**D. Pharmacologic Interventions**

In addition to fluids and blood products, pharmacologic agents can be utilized as adjuncts in the management of hypovolemic shock and coagulopathy:

**Tranexamic Acid (TXA)**

* Tranexamic acid is a synthetic lysine analog that inhibits fibrinolysis by blocking plasminogen activation and binding to fibrin to prevent breakdown of clots.
* Evidence for Use: Two large randomized controlled trials in trauma patients found reduced mortality when 1 gram IV tranexamic acid was administered within 3 hours of injury. The CRASH-2 trial showed a 1.5% absolute reduction in mortality in bleeding trauma patients. The MATTERs study found a 13.7% absolute reduction in mortality in bleeding military patients who required massive transfusion.
* Dosing: The recommended dose is 1 gram IV tranexamic acid over 10 minutes, followed by 1 gram IV infusion over 8 hours. A repeat 1 gram IV dose may be administered if bleeding continues or recurs within 24 hours per some protocols.
* Adverse Effects: There is no significant increased risk of thromboembolic events. Minor potential adverse effects include nausea, vomiting, diarrhea, visual disturbances, hypotension, and headache.
* Considerations: Tranexamic acid is inexpensive and should be incorporated into massive transfusion protocols. It provides maximal benefit when administered in the first 3 hours after injury. Ongoing studies are further evaluating optimal timing, redosing, and safety in TBI patients.

**Prothrombin Complex Concentrates**

* PCCs contain coagulation Factors II, VII, IX, X along with variable amounts of protein C, S, and antithrombin. Four-factor PCCs also contain significant Factor VII.
* Evidence for Use: Compared to FFP, PCCs provide more rapid INR correction and faster attainment of hemostasis. Retrospective studies found reduced transfusion needs, complication rates, hospital length of stay, and improved survival in trauma patients receiving PCCs compared to FFP alone during massive transfusion.
* Dosing: Four-factor PCC dosing is guided by INR and weight. Typical dose is 25-50 units/kg. Three-factor products should only be used for Warfarin reversal per package labeling.
* Adverse Effects: The rate of thromboembolic complications is approximately 1-2%. PCCs should be avoided in patients with history of prior HIT, DIC, PE/DVT, or acute thrombosis.
* Considerations: PCCs provide faster INR normalization than FFP or vitamin K in life-threatening hemorrhage. However, large randomized trials have not been conducted. Exact role in massive transfusion is still being defined.

**Recombinant Factor VIIa**

* rFVIIa promotes localized thrombin generation on activated platelet surfaces independent of factors VIII and IX. It was originally developed for hemophilia patients.
* Evidence for Use: Case reports describe efficacy for refractory hemorrhage, but two RCTs failed to show mortality benefit in trauma. One trial found lower transfusion needs. Concerns exist for increased thromboembolic events with rFVIIa use.
* Dosing: 90 mcg/kg IV was standard dosing extrapolated from hemophilia trials. More recent evidence suggests much lower doses of 10-40 mcg/kg may provide hemostatic effect with less thrombotic risk.
* Adverse Effects: Increased risk of arterial thrombosis resulting in MI, CVA, PE. Also venous thrombosis.
* Considerations: rFVIIa cannot be routinely recommended given lack of proven mortality benefit and concerns for thromboembolic complications. May be considered as "rescue" therapy for life-threatening hemorrhage refractory to all other interventions.

**Vasopressors**

* Agents like norepinephrine and vasopressin raise blood pressure through vasoconstriction. This can help temporarily maintain perfusion before hemorrhage is controlled.
* Evidence for Use: Vasopressor use is associated with increased mortality when utilized before control of bleeding is achieved. Experts recommend they be reserved for patients remaining hypotensive despite adequate volume resuscitation when source control is not immediately possible.
* Dosing: Start with norepinephrine or vasopressin at lowest dose to target MAP ≥65 mmHg. Epinephrine and dopamine may also be considered. Wean pressors once bleeding controlled.
* Adverse Effects: Excessive vasopressor use can reduce cardiac output, worsen lactic acidosis, and decrease tissue perfusion. Use the minimum dose necessary.
* Considerations: Avoid early vasopressor use in hemorrhagic shock. Titrate based on MAP goals and biomarkers of tissue perfusion. Monitor for ischemia and discontinue if patient deteriorates.

**Calcium Salts**

* Ionized calcium is essential for coagulation factor binding, platelet activation, and thrombus stabilization. Hypocalcemia worsens coagulopathy.
* Evidence for Use: Delivery of 1-3 grams IV calcium chloride or 2-6 grams calcium gluconate rapidly corrects ionized calcium levels and treats coagulopathy.
* Dosing: Administer 1 gram IV calcium chloride (or 3 gram calcium gluconate equivalent) and repeat as needed to normalize calcium and improve coagulation function. Avoid in hypercalcemic patients.
* Adverse Effects: Extravasation causes severe tissue necrosis with calcium salts. Other risks include bradycardia and hypotension. Use central access when possible.
* Considerations: Monitor ionized calcium and supplement aggressively with IV calcium salts in the massively bleeding patient to improve coagulation.

**VI. Special Populations**

Management of hypovolemic shock must be tailored to the needs of individual patient populations:

1. Elderly
   1. The elderly are at higher risk for volume depletion due to comorbidities and medication use. They exhibit decreased intravascular volume reserves and impaired cardiovascular compensation to hypovolemia. Shock can progress rapidly with higher associated mortality. Avoid excessive fluid administration due to risks of volume overload.
2. B. Pediatrics
   1. Pediatric patients compensate well for hypovolemia, maintaining normal blood pressures despite significant blood losses. Tachycardia is often the only initial sign. Carefully monitor for decompensation. Utilize clinical signs like delayed capillary refill rather than blood pressure to guide interventions.
3. Pregnancy
   1. Hypovolemic shock has dire implications for both maternal and fetal status. Major causes include obstetric hemorrhage and sepsis. Aggressive, early intervention is warranted to optimize outcomes.
4. Traumatic Brain Injury
   1. Avoid hypotension which reduces cerebral perfusion pressure and risks secondary neurologic injury. Higher blood pressure targets may be warranted.
5. Burns
   1. Burns result in tremendous fluid shifts and resuscitation requires large volumes guided by burn surface area and body weight. Colloids should be avoided in the first 24 hours when capillary leak occurs.

**VII. Key Guidelines and Evidence**

The American College of Surgeons Advanced Trauma Life Support (ATLS) guidelines provide several key recommendations for the management of hemorrhagic shock:

* Administer isotonic crystalloids for initial resuscitation while preparing blood products.
* Initiate blood transfusion if tachycardia and hypotension persist after 2 liters crystalloid infusion.
* Use permissive hypotension before surgical control of hemorrhage.
* Limit crystalloid volumes to decrease risks of coagulopathy, acidosis, hypothermia.
* Employ damage control resuscitation principles including early balanced ratios.

Two landmark randomized controlled trials demonstrating reduced mortality with balanced product ratios are:

* The Pragmatic, Randomized Optimal Platelet and Plasma Ratios (PROPPR) trial, which showed benefit of a 1:1:1 plasma:platelet:RBC ratio compared to 1:1:2 ratio in severely injured trauma patients.1
* The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) study, which found early and higher plasma:RBC ratios decreased mortality.2

**VIII. Clinical Scenarios**

**Scenario 1:**

A 72-year-old female with a history of peptic ulcer disease presents with lightheadedness, fatigue, melena, and orthostatic symptoms. On arrival, her blood pressure is 82/50 mmHg, heart rate is 118 bpm, and respiratory rate is 25 breaths/min. On examination, she has dry mucous membranes, cool extremities, and guaiac positive stool. Laboratory results show hemoglobin of 7 g/dL, creatinine 2.1 mg/dL, and lactate 4.5 mmol/L.

This clinical presentation is consistent with hypovolemic shock secondary to acute gastrointestinal hemorrhage. Immediate fluid resuscitation should be initiated using isotonic crystalloid while preparing packed red blood cells for transfusion. IV pantoprazole and octreotide could be administered concurrently to decrease gastric acidity and reduce variceal blood flow. Blood products will be needed to improve oxygen delivery and intravascular volume. Vasopressors may be required for refractory hypotension if bleeding is not yet controlled. Definitive management involves emergent endoscopy for identification and treatment of the bleeding source.

**Scenario 2:**

A 32-year-old male is brought to the emergency department by EMS after a motorcycle collision. On arrival, his blood pressure is 118/72 mmHg, heart rate is 136 bpm, and GCS is 14. FAST ultrasound is positive for free intraperitoneal fluid concerning for hemorrhage. Initial hemoglobin is 12 g/dL and INR 1.2. One hour after arrival, his blood pressure declines to 82/44 mmHg and repeat hemoglobin has downtrended to 8 g/dL.

This presentation is consistent with class III hemorrhagic shock from presumed splenic laceration. Despite normal initial vital signs, overt evidence of cardiovascular decompensation developed rapidly, indicated by worsening tachycardia and hypotension. Aggressive intravenous fluid resuscitation with crystalloid should be initiated, along with transfusion of packed red blood cells, fresh frozen plasma, and platelets in a balanced ratio. Massive transfusion protocol may be activated depending on clinical course and volume of ongoing hemorrhage. Emergent splenectomy or splenic artery embolization will be needed for definitive hemorrhage control.

Tips for Board Exam Questions

* Know definitions, classification, and diagnostic criteria for hypovolemic shock.
* Understand pathophysiology of different shock subtypes - hemorrhagic, cardiogenic, septic, neurogenic.
* Recognize complications and treatment priorities - address airway, breathing, circulation.
* Identify appropriate fluid, blood product, and pharmacologic therapies based on etiology.
* Apply principles of damage control resuscitation - permissive hypotension, restrictive fluids, early balanced ratios.

**IX. Hypovolemic Shock Summary**

* In summary, hypovolemic shock is characterized by critical intravascular volume depletion leading to tissue hypoperfusion and organ dysfunction. Etiologies include hemorrhage, fluid losses, and transcellular shifts. Diagnosis relies on clinical exam, vital signs, and laboratory trends. Resuscitation priorities are to control bleeding, ensure airway and oxygenation, and restore circulating volume with fluids and blood products. Modern principles of damage control emphasize judicious fluid administration, early balanced transfusions, and prevention of trauma-induced coagulopathy to optimize outcomes. Ongoing research continues to better define optimal resuscitation strategies for hypovolemic shock across diverse patient populations.

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* Understand pathophysiology of different shock subtypes - hemorrhagic, cardiogenic, septic, neurogenic.
* Recognize complications and treatment priorities - address airway, breathing, circulation.
* Identify appropriate fluid, blood product, and pharmacologic therapies based on etiology.
* Apply principles of damage control resuscitation - permissive hypotension, restrictive fluids, early balanced ratios.

**IX. Hypovolemic Shock Summary**

* In summary, hypovolemic shock is characterized by critical intravascular volume depletion leading to tissue hypoperfusion and organ dysfunction. Etiologies include hemorrhage, fluid losses, and transcellular shifts. Diagnosis relies on clinical exam, vital signs, and laboratory trends. Resuscitation priorities are to control bleeding, ensure airway and oxygenation, and restore circulating volume with fluids and blood products. Modern principles of damage control emphasize judicious fluid administration, early balanced transfusions, and prevention of trauma-induced coagulopathy to optimize outcomes. Ongoing research continues to better define optimal resuscitation strategies for hypovolemic shock across diverse patient populations.

**X. References**

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