

Hemorrhagic Shock: Pathophysiology, Diagnosis, and Contemporary Management

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How to cite this article:

Dinesh K. Evolving Hemorrhagic Shock: Pathophysiology, Diagnosis, and Contemporary Management. J of Emer and Trauma Nurs. 2025; 6(1): 25–29.

ABSTRACT

Hemorrhagic shock is a life-threatening condition characterized by severe blood loss leading to inadequate tissue perfusion and cellular oxygenation. It represents a significant cause of morbidity and mortality in trauma, obstetrics, and various surgical settings. This article provides a comprehensive overview of hemorrhagic shock, encompassing its complex pathophysiology, the clinical stages of presentation, modern diagnostic approaches, and the principles of contemporary management. Key emphasis is placed on rapid assessment, fluid and blood product resuscitation, control of hemorrhage, and the evolving concepts of damage control resuscitation and permissive hypotension. Understanding the mechanisms and implementing timely, evidence-based interventions are paramount to improving patient outcomes.

KEYWORDS

• Blood loss • Hypovolemia • Shock • Hemorrhage • Tissue perfusion • Organ failure • Resuscitation • Trauma

INTRODUCTION

Hemorrhagic shock, a subset of hypovolemic shock, arises from an acute loss of circulating blood volume, leading to a critical reduction in effective tissue perfusion. This cellular hypoxia, if uncorrected, culminates in anaerobic metabolism, lactic acidosis, multi-organ dysfunction, and ultimately, death.

Globally, hemorrhage remains a leading cause of preventable death in trauma, accounting for upto 30-40% of all trauma fatalities, and is also a major contributor to mortality in non-traumatic contexts such as gastrointestinal bleeding, ruptured aneurysms, and obstetric emergencies.¹

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➤ Received: 30.03.2025 Accepted: 16.05.2025



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The human body possesses sophisticated compensatory mechanisms to maintain vital organ perfusion in the face of hypovolemia. However, these mechanisms are finite, and beyond a certain threshold of blood loss, decompensation rapidly ensues. Early recognition and aggressive management are critical for survival, necessitating a thorough understanding of the physiological responses, clinical indicators, and the structured approach to resuscitation. This article aims to synthesize current knowledge regarding the pathophysiology, diagnostic criteria, and contemporary therapeutic strategies for hemorrhagic shock, drawing from established guidelines and recent advancements in critical care medicine.^{2,3}

METHODS AND MATERIAL

This article synthesizes current understanding of hemorrhagic shock by reviewing peer-reviewed literature, established clinical guidelines from professional organizations (e.g., American College of Surgeons Advanced Trauma Life Support - ATLS, European Society of Intensive Care Medicine - ESICM, Eastern Association for the Surgery of Trauma - EAST), and authoritative medical textbooks. The selection of material focused on providing a comprehensive overview of the condition, emphasizing advancements in pathophysiology, diagnostic modalities, and contemporary management strategies, particularly those related to damage control resuscitation, permissive hypotension, and the evolving role of blood product ratios. Priority was given to studies and guidelines published predominantly within the last two decades to ensure the inclusion of current evidence-based practices. Information was systematically extracted regarding the definition, classification, physiological responses, clinical presentation, laboratory and imaging diagnostics, and therapeutic interventions for hemorrhagic shock.⁴

RESULT

Pathophysiology of Hemorrhagic Shock

The core pathophysiological event in hemorrhagic shock is a reduction in circulating blood volume, leading to a decrease in cardiac preload, stroke volume, and ultimately,

cardiac output. The body initially responds with a series of compensatory mechanisms:

1. **Catecholamine Release:** Adrenaline and noradrenaline are released, causing systemic vasoconstriction, increased heart rate, and myocardial contractility to shunt blood to vital organs (brain and heart). This maintains blood pressure initially.
2. **Renin-Angiotensin-Aldosterone System (RAAS) Activation:** Angiotensin II causes further vasoconstriction and aldosterone promotes sodium and water retention by the kidneys to increase blood volume.
3. **Antidiuretic Hormone (ADH) Release:** ADH promotes water reabsorption in the kidneys.
4. **Fluid Shifts:** Fluid shifts from the interstitial space into the intravascular space, a process known as autotransfusion, to restore circulating volume.

As blood loss continues (typically >30-40% of total blood volume), these compensatory mechanisms become overwhelmed. Tissue hypoperfusion leads to a critical reduction in oxygen delivery, forcing cells to switch from aerobic to anaerobic metabolism. This generates lactic acid, leading to metabolic acidosis. The "vicious triad" of trauma-induced coagulopathy, hypothermia, and acidosis further exacerbates bleeding, impairs organ function, and contributes to the "lethal triad" which is difficult to reverse. Endothelial damage, systemic inflammatory response, and microvascular thrombosis further compromise perfusion and can lead to multiple organ dysfunction syndrome (MODS).⁵

Clinical Presentation and Stages of Hemorrhagic Shock

The clinical presentation of hemorrhagic shock varies depending on the volume of blood lost, as classified by ATLS guidelines:

- **Class I (up to 15% blood loss, ~750 ml):** Minimal symptoms. Heart rate usually normal, blood pressure normal, slight anxiety.
- **Class II (15-30% blood loss, ~750-1500 ml):** Tachycardia (>100 bpm) is the earliest significant sign. Respiratory rate slightly increased, pulse pressure narrowed, cool and clammy skin, increased capillary refill time, mild anxiety.

- **Class III (30-40% blood loss, ~1500-2000 ml):** Marked tachycardia (>120 bpm), tachypnea, significant hypotension, altered mental status (confusion, agitation), decreased urine output. This stage requires blood transfusion.
- **Class IV (>40% blood loss, >2000 ml):** Profound hypotension, bradycardia (ominous sign), weak or absent peripheral pulses, lethargy/unresponsiveness, anuria. Immediate and aggressive resuscitation is critical.

It is crucial to note that these classifications are based on healthy adults. Patients on beta-blockers, athletes, or the elderly may have blunted physiological responses, making diagnosis more challenging.⁶

Diagnosis of Hemorrhagic Shock

Diagnosis is primarily clinical, based on a history of potential blood loss (e.g., trauma, GI bleed) and the physical signs of hypovolemia.

- **Clinical Assessment:** Rapid initial assessment (primary survey: Airway, Breathing, Circulation, Disability, Exposure) is paramount. Vital signs, mental status, skin perfusion, and external evidence of bleeding are key.
- **Laboratory Investigations:**
 - **Hemoglobin/Hematocrit:** Initial levels may be normal due to compensatory mechanisms or prior to fluid resuscitation, but will drop as fluid is administered or blood loss continues. Not a reliable early indicator.
 - **Lactate:** Elevated serum lactate is a sensitive marker of tissue hypoperfusion and an excellent prognostic indicator. Serial measurements guide resuscitation.
 - **Base Deficit:** Reflects metabolic acidosis and tissue hypoperfusion; an increasing deficit indicates ongoing shock.
 - **Coagulation Profile (PT, aPTT, INR, Fibrinogen):** Essential for diagnosing and monitoring trauma-induced coagulopathy.
 - **Type and Crossmatch:** Expedite blood product availability.

- **Imaging:**

- **Focused Assessment with Sonography for Trauma (FAST) exam:** Rapid, non-invasive ultrasound to detect free fluid (blood) in the pericardial, perihepatic, perisplenic, and pelvic spaces. Critical in trauma.
- **Computed Tomography (CT) Scan:** Used to localize bleeding sources, especially in stable or stabilized patients. Angiography can identify active extravasation.

Contemporary Management of Hemorrhagic Shock

The management of hemorrhagic shock follows a structured approach with simultaneous assessment and intervention:

1. **Airway and Breathing:** Ensure a patent airway and adequate ventilation, often requiring intubation in severe shock to reduce the work of breathing and optimize oxygenation.
2. **Circulation and Hemostasis:** This is the cornerstone of management.
 - **Control of Hemorrhage:** The definitive treatment. This involves direct pressure, tourniquets for extremity bleeding, surgical intervention (e.g., laparotomy, thoracotomy), interventional radiology (e.g., angioembolization), or external fixation for pelvic fractures.
 - **Fluid Resuscitation:** Initial resuscitation typically involves intravenous crystalloids (e.g., Lactated Ringer's). However, excessive crystalloid administration can worsen coagulopathy, hypothermia, and acidosis. The concept of "permissive hypotension" advocates for maintaining a systolic blood pressure target (e.g., 80-90 mmHg) in actively bleeding patients without head injury until definitive hemorrhage control is achieved, to prevent dislodging clots and worsening bleeding.
 - **Blood Product Transfusion:** Timely administration of blood products is critical. Current guidelines emphasize balanced resuscitation with plasma, platelets, and red blood cells (RBCs) in ratios approaching 1:1:1 to mitigate

coagulopathy (Damage Control Resuscitation - DCR).

- **Red Blood Cells (RBCs):** To restore oxygen-carrying capacity.
 - **Fresh Frozen Plasma (FFP):** To replace clotting factors.
 - **Platelets:** To improve platelet count and function.
 - **Cryoprecipitate:** For fibrinogen replacement.
 - **Tranexamic Acid (TXA):** An antifibrinolytic agent, given early (within 3 hours of injury) in trauma patients with significant bleeding, has shown to reduce mortality.
 - **Calcium Replacement:** Hypocalcemia is common during massive transfusions due to citrate toxicity and can impair myocardial function and coagulation; calcium should be monitored and replaced.
 - **Vasoactive Agents:** Vasopressors (e.g., norepinephrine) may be used as an adjunct in refractory shock after adequate volume resuscitation and hemorrhage control, but should not replace volume.
3. **Monitoring:** Continuous monitoring of vital signs, fluid balance, urine output, and serial laboratory values (lactate, base deficit, coagulation profile, Hb) guides resuscitation efforts.
 4. **Prevention of the "Lethal Triad":** Active measures to prevent and treat hypothermia (warming blankets, warm fluids), acidosis (adequate perfusion, bicarbonate in severe cases), and coagulopathy (balanced transfusion, TXA, specific factor replacement).

DISCUSSION

The management of hemorrhagic shock has evolved significantly, moving from aggressive crystalloid resuscitation to a more nuanced approach centered on damage control resuscitation (DCR) and hemostatic resuscitation. The recognition that indiscriminate crystalloid use can dilute clotting factors, worsen acidosis, and cause hypothermia (the "lethal triad") has shifted the paradigm towards early and balanced transfusion of blood products. The 1:1:1

ratio of plasma:platelets:RBCs, while not universally applied or proven superior in all contexts, reflects this emphasis on correcting coagulopathy from the outset.⁷

Permissive hypotension, another critical advancement, acknowledges that rapidly increasing blood pressure before hemorrhage control can disrupt forming clots and exacerbate bleeding. This strategy, however, is contraindicated in patients with traumatic brain injury, where maintaining cerebral perfusion pressure is paramount. The role of point-of-care viscoelastic hemostatic assays (e.g., ROTEM, TEG) is also gaining traction, allowing for real-time assessment of individual patient coagulopathy and guiding targeted transfusion of specific blood products rather than fixed ratios.

Despite these advancements, challenges remain. Pre-hospital care, rapid transport, and seamless transition to definitive care are crucial. Identifying occult bleeding sources can be difficult, particularly in non-traumatic cases. Furthermore, special populations, such as the elderly, children, and pregnant women, present unique physiological responses and management considerations. The importance of a multidisciplinary team approach, involving emergency physicians, surgeons, anesthesiologists, interventional radiologists, and critical care specialists, cannot be overstated in optimizing outcomes for patients with hemorrhagic shock. Continuous education, protocol development, and research into novel hemostatic agents and resuscitation strategies are vital to further reduce the morbidity and mortality associated with this devastating condition.

CONCLUSION

Hemorrhagic shock is a medical emergency requiring rapid recognition, aggressive resuscitation, and definitive control of bleeding. Understanding its intricate pathophysiology, recognizing the subtle and overt signs of decompensation, and applying contemporary management principles are fundamental to improving patient survival. The paradigm shift towards damage control resuscitation, emphasis on balanced blood product ratios, and the judicious application of permissive hypotension represent significant strides in care. A coordinated, multidisciplinary effort, guided by continuous monitoring

and evidence-based protocols, remains the cornerstone of effective management for this life-threatening condition.

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