# Management of Hypovolemic Shock in Emergency Settings: Overview

Waleed Hussain Mohsin Alharthi <sup>1</sup>, Salem Khalifah Gh Aldhafeeri <sup>2</sup>, Faisal Ali Yahia Hakami <sup>3</sup>, Salem Hussain Moadi Alanazi <sup>4</sup>, Alanazi , Anwar Jazio <sup>5</sup>, Mohammed Hamoud Furaykh Albanaqi <sup>6</sup>, Abdullah Awadh Aziz Almutairi <sup>7</sup>, Abdulaziz Ayed Alenezi <sup>8</sup>, Mohammed Blibes H Alenezi <sup>9</sup>, Zaid Dakhel B Alhazmi <sup>10</sup>

- 1- Emergency Medical Specialist, Children's Hospital, Taif, Saudi Arabia
- 2- Emergency medical services technician, Health Crisis and Disaster Management Center, Hafr Al-Batin, Saudi Arabia.
- 3- Emergency medical services technician, Emergency Coordination and Care Department, Jazan Health Cluster, Saudi Arabia
  - 4- Emergency medical services technician, Red Crescent Authority Branch, Tabuk, Saudi Arabia.
  - 5- Emergency medical services technician, Ministry of Health Branch- Northern Border Region, Saudi Arabia
- 6- Emergency medical services technician, Emergency Management and Medical Coordination, Turaif, Saudi Arabia.
- 7- Emergency medical services technician, Ministry of Health Branch, Hafr Al-Batin, Saudi Arabia.
- 8- emergency medical services, Health Crisis and Disaster Management Center, Hafr Al-Batin, Saudi Arabia.
  - 9- Emergency medical services, Prince Abdulaziz bin Musaed Hospital Arar, Saudi Arabia
  - 10- Technicians and Health Assistants, Eradah Complex for Mental Health- Arar, Saudi Arabia

### **Abstract:**

Hypovolemic shock is a life-threatening condition resulting from a significant loss of blood volume, leading to inadequate tissue perfusion and potential organ failure. Immediate recognition and intervention are critical, especially in emergency settings where the patient's survival depends on rapid treatment. Initial management includes assessing the severity of shock, securing the airway, and establishing intravenous (IV) access for fluid resuscitation. The primary goal is to restore intravascular volume using crystalloid solutions (e.g., normal saline or lactated Ringer's) and, if necessary, administering blood products for severe hemorrhage. Monitoring vital signs, urine output, and mental status helps gauge the effectiveness of resuscitation efforts and guide further interventions. Beyond initial stabilization, managing the underlying cause of hypovolemic shock is crucial. Once hemostasis is achieved through control of bleeding (e.g., surgical intervention or other techniques), additional therapies such as vasopressors may be considered to support systemic circulation if fluid resuscitation alone is insufficient. Continuous reassessment of blood volume status and organ function is paramount to avoid complications associated with aggressive fluid therapy. Collaboration among emergency medical personnel, trauma surgeons, and critical care providers ensures a multi-faceted approach to optimize patient outcomes in this critical condition.

**Keywords:** Hypovolemic shock, Emergency management, Fluid resuscitation, Fluid resuscitation, Blood volume, Crystalloids, Blood products, Hemostasis, Vital signs monitoring, Organ perfusion, Vasopressors, Trauma care.

#### Introduction:

Hypovolemic shock is a life-threatening condition characterized by a significant reduction in blood volume, leading to inadequate tissue perfusion and oxygen delivery. It commonly results from various causes, including traumatic injuries, gastrointestinal hemorrhage, and severe dehydration. The rapid identification and management of hypovolemic shock in emergency settings are crucial for improving patient outcomes and minimizing the risk of morbidity and mortality. The significance of understanding the pathophysiology, clinical presentation, diagnostic approaches, and evidence-based management strategies cannot be overstated, particularly in high-pressure environments like

emergency departments (EDs) where time is of the essence [1].

The physiological consequences of hypovolemic shock stem from decreased intravascular volume, which results in reduced venous return to the heart, lowered cardiac output, and impaired organ perfusion. Compensation mechanisms—such as increased heart rate, peripheral vasoconstriction, and activation of neurohormonal pathways—are often insufficient to maintain hemodynamic stability. The cascade of failed compensatory responses leads to cellular dysfunction and, eventually, multi-organ failure if not adequately treated. Research has shown that vital organs, including the brain and kidneys, are particularly vulnerable to the consequences of inadequate perfusion, highlighting the importance of early recognition and intervention during the initial phases of shock [2].

The clinical presentation of hypovolemic shock can vary significantly depending on the severity of blood volume loss and the underlying cause. Patients may present with a range of symptoms, from mild tachycardia and hypotension to altered mental status and cold, clammy extremities. Emergency care providers must be vigilant in assessing the patient's clinical status, including vital signs, skin perfusion, and urine output, to identify the degree of shock and correlate it with potential underlying etiologies. Valuable tools such as the shock index (heart rate/systolic blood pressure ratio) and the use of standardized assessment scales can aid healthcare providers in making timely and accurate clinical decisions [3].

In the emergency setting, the management of hypovolemic shock involves a multidisciplinary approach focusing on rapid diagnosis, aggressive resuscitation, and continuous monitoring. Early fluid resuscitation with intravenous (IV) crystalloid fluids is often the cornerstone of initial management, with the goal being to restore perfusion pressure and optimize oxygen delivery to tissues. Recent studies have sparked discussions over the type and volume of fluids used, with a growing interest in balanced crystalloids compared to normal saline, particularly in critically ill patients. Additionally, the use of blood products dictates a pivotal aspect of care, especially when dealing with trauma gastrointestinal hemorrhage. Blood transfusions must be judiciously administered based on clinical scenarios and laboratory findings,

hemoglobin levels and the patient's hemodynamic response [4].

The adoption of a "massive transfusion protocol" in trauma centers has been suggested as a means of expediting the delivery of necessary blood products while minimizing the risk of coagulopathy and other complications associated with massive transfusion. The use of guidelines for early transfusion of red blood cells, plasma, and platelets has demonstrated improved outcomes in patients suffering from blunt trauma and hemorrhagic shock. Furthermore, emerging therapies, including the use of vasopressors for those who do not respond adequately to fluid resuscitation, continue to evolve and require careful consideration in the management protocols for hypovolemic shock [5].

Effective teamwork and communication among healthcare professionals are essential components in the emergency management of hypovolemic shock. The utilization of standardized protocols and checklists helps mitigate errors and optimize treatment in high-stress situations. Furthermore, monitoring tools, such as point-of-care ultrasound and continuous hemodynamic monitoring, provide clinical teams with real-time data to guide therapeutic decisions and monitor the patient's response to interventions [6].

# Pathophysiology of Hypovolemic Shock:

Hypovolemic shock is a severe clinical condition characterized by the inadequate perfusion of tissues and organs due to a significant reduction in blood volume. This state of shock is often a result of acute blood loss, fluid loss, or a combination of both, leading to a critical reduction in effective circulating volume. Understanding the pathophysiology of hypovolemic shock involves an exploration of the hemodynamic changes, physiologic responses, and subsequent organ dysfunction that ensue following significant volume depletion [7].

## **Causes of Hypovolemic Shock**

Hypovolemic shock can be triggered by various events that lead to substantial fluid loss. The most common causes include:

1. **Hemorrhage**: Acute blood loss from trauma, surgical procedures, or gastrointestinal bleeding can rapidly decrease blood volume. Losses exceeding 20% (approximately 1 liter in an adult) can precipitate shock [8].

- 2. Fluid Loss: Conditions such as severe dehydration from vomiting, diarrhea, or excessive diuresis, often seen in diabetic patients, can lead to hypovolemic shock. Additionally, burns can result in plasma loss through damaged skin.
- 3. **Thirdspacing of Fluids**: Pathological conditions like sepsis can result in fluid being sequestered in the interstitial spaces, effectively reducing the circulating blood volume [8].

#### **Hemodynamic Changes**

The initial stages of hypovolemic shock involve a series of compensatory mechanisms intended to maintain organ perfusion despite the reduced blood volume. The body's response can be divided into several phases:

- 1. Compensatory Phase: As blood volume decreases, the body initiates compensatory mechanisms to preserve blood flow to vital organs. The baroreceptors in the carotid sinus and aortic arch detect decreased blood pressure and stimulate the sympathetic nervous system. This leads to increased heart rate (tachycardia), peripheral vasoconstriction to redirect blood flow to essential organs (like the heart and brain), and increased myocardial contractility [9].
- 2. Renin-Angiotensin-Aldosterone System (RAAS) Activation: The decrease in renal perfusion triggers the release of renin, leading to the formation of angiotensin II. This potent vasoconstrictor further raises systemic vascular resistance and blood pressure. Additionally, aldosterone release increases sodium and water retention in the kidneys, aiming to restore blood volume.
- 3. Fluid Shifts and Electrolyte Imbalance: As intravascular volume decreases, fluid may shift from the cells and interstitial space to maintain circulatory volume, but this process is limited by the severity of the hypovolemia. Electrolyte imbalances, particularly hyponatremia and hyperkalemia, can occur due to renal compensation and tissue breakdown in severe cases [9].
- 4. **Progressive Phase**: If volume depletion continues past a critical threshold,

- compensatory mechanisms fail, progressing to further hemodynamic instability. Cardiac output decreases due to lower preload resulting from reduced venous return. The persistent hypoperfusion leads to cellular hypoxia and metabolic acidosis, as anaerobic metabolism takes over, producing lactic acid and compounding the shock state.
- 5. **Refractory Shock**: Ultimately, if not addressed promptly, persistent hypovolemic shock can lead to irreversible organ damage. Multi-organ failure can ensue as cells lose their ability to generate ATP, leading to cellular necrosis and apoptosis [10].

#### **Organ Dysfunction**

The state of hypovolemic shock severely impacts multiple organ systems:

- 1. Cardiovascular System: Myocardial ischemia may occur due to reduced coronary perfusion, further exacerbating cardiac dysfunction. Hypoperfusion can lead to arrhythmias and potentially, myocardial infarction [11].
- 2. **Renal System:** The kidneys are highly susceptible to ischemia and are often one of the first organs to demonstrate dysfunction in shock. Acute kidney injury (AKI) can develop as glomerular filtration rate decreases due to decreased renal perfusion.
- 3. **Neurological System**: The brain is particularly vulnerable to hypoxia. Prolonged hypoperfusion can lead to altered mental status and, if untreated, may result in varying degrees of cerebral hypoxia or infarction.
- 4. **Gastrointestinal System**: Compromised perfusion can lead to mucosal ischemia, translocation of bacteria, and gastrointestinal bleeding due to necrosis of the intestinal mucosa.
- 5. **Hematologic System**: Coagulopathy may occur due to consumption coagulopathy as platelets and clotting factors become depleted in the setting of shock, leading to disseminated intravascular coagulation (DIC) [11].

#### **Diagnosis and Management**

The diagnosis of hypovolemic shock is primarily clinical, supported by history, examination, and

laboratory investigations. Patients typically present with tachycardia, hypotension, altered mental status, and signs of decreased perfusion (such as cold extremities and delayed capillary refill). Point-of-care ultrasound and laboratory tests, including lactate and hemoglobin levels, can assist in evaluating the severity of shock and guiding treatment.

Immediate management includes the initiation of fluid resuscitation with crystalloids and/or blood products, depending on the cause of hypovolemia. In some cases, medications such as vasopressors may be needed if blood pressure fails to respond adequately to fluid therapy. Furthermore, addressing the underlying cause, such as controlling hemorrhage or managing fluid losses, is paramount for effective recovery from hypovolemic shock [12].

# **Clinical Presentation and Diagnosis:**

Hypovolemic shock represents a critical state of diminished perfusion to tissues resulting primarily from inadequate circulating blood volume. It is a clinical emergency that arises from various causes, including significant blood loss due to trauma, gastrointestinal bleeding, or severe dehydration. Understanding the clinical presentation and the approach to diagnosing hypovolemic shock is essential for timely management and intervention [13].

# **Understanding Hypovolemic Shock**

Hypovolemic shock occurs when there is a significant decrease in the intravascular volume, leading to reduced cardiac output and subsequent insufficient tissue perfusion. The volume loss can be classified as either absolute or relative. Absolute hypovolemia involves direct losses from the vascular system, such as hemorrhage due to trauma or surgery, while relative hypovolemia can occur due to conditions such as sepsis, where vasodilation leads to pooling of blood in the periphery without actual loss of fluid volume [13].

#### **Clinical Presentation**

The clinical presentation of hypovolemic shock is characterized by a spectrum of signs and symptoms that reflect the body's compensatory mechanisms attempting to counteract diminished blood volume and inadequate perfusion:

- 1. Vital Sign Changes: **Patients** with hypovolemic shock typically exhibit tachycardia (increased heart rate) as the body attempts to maintain cardiac output. Blood pressure may be hypotensive or remain within the normal range initially due to compensatory mechanisms. However, as the shock worsens, hypotension usually becomes apparent [14]. Respiratory rates often increase (tachypnea) as a compensatory response to metabolic acidosis and tissue hypoxia.
- Skin Changes: Upon examination, the skin may display signs of pallor, coolness, and clamminess due to peripheral vasoconstriction as blood is redirected from the skin to vital organs. Meanwhile, capillary refill time may exceed two seconds.
- 3. Altered Mental Status: Patients may demonstrate confusion or altered levels of consciousness due to decreased cerebral perfusion. Anxiety, agitation, or restlessness often precede significant mental alterations such as lethargy or unresponsiveness [14].
- 4. **Gastrointestinal Symptoms**: The gastrointestinal tract may be affected, leading to symptoms such as nausea, vomiting, or abdominal pain. Decreased perfusion can also lead to ileus.
- 5. **Urinary Output**: Oliguria (reduced urine output) or anuria (absence of urine output) can be notable findings due to renal hypoperfusion. Urine output is a key indicator of overall perfusion status and kidney function [15].

# Diagnostic Criteria

The diagnosis of hypovolemic shock is primarily clinical, although additional testing can assist in confirming the diagnosis and determining the underlying cause. Key diagnostic criteria include:

1. Clinical Assessment: A thorough history and physical examination are essential. Clinicians must evaluate for possible causes of volume loss, such as trauma, gastrointestinal bleeding, or conditions causing dehydration. Additionally, understanding a patient's medical history, medications, or potential sources of fluid loss is crucial [16].

- 2. **Laboratory Tests**: Blood tests can support the diagnosis of hypovolemic shock. Important laboratory assessments may include:
  - Complete Blood Count (CBC): Provides insights into hemoglobin levels, which help assess organizational loss and hydration status.
  - Electrolytes: Assessing sodium, potassium, and bicarbonate levels can reveal electrolyte imbalances often associated with fluid loss.
  - Metabolic Panel: Evaluates renal function and metabolic status through blood urea nitrogen and creatinine levels [17].
- 3. **Imaging Studies**: Depending on the suspected source of blood loss, imaging studies may be employed. For instances of trauma, ultrasound, CT scans, or X-rays can help identify internal bleeding or other sources of hypovolemia.
- 4. Hemodynamic Monitoring: In some cases, especially for critically ill patients, invasive hemodynamic monitoring may be indicated. Measurements like central venous pressure (CVP) or pulmonary artery catheterization can help evaluate the volume status and cardiac output, although these techniques carry more risk and are generally reserved for complex cases.
- 5. **Point-of-Care Ultrasound (POCUS)**: Increasingly, POCUS is being utilized in emergency settings to rapidly assess for free fluid in the abdomen or identify pericardial effusion, which can aid in differentiating hypovolemic shock from other forms of shock [18].

# Initial Assessment and Triage in Emergency Settings:

Hypovolemic shock is a critical condition characterized by a significant reduction in circulating blood volume, leading to inadequate oxygen delivery to tissues and organs. This state often arises from acute blood loss due to trauma, hemorrhagic events, or severe fluid depletion caused by dehydration, burns, or other medical conditions. The rapid identification and treatment of hypovolemic shock are essential for improving patient outcomes [19].

Before delving into the assessment and triage process, it is essential to understand the physiological underpinnings of hypovolemic shock. The body is reliant on a certain volume of blood to maintain adequate perfusion of tissues. When blood volume diminishes by more than 15%, or approximately 750 milliliters in an average adult, compensatory mechanisms may initially preserve perfusion. However, if blood loss continues and adequate volume is not restored, cellular metabolism falters, leading to multisystem organ failure [19].

#### **Initial Evaluation**

# A. Primary Survey

The initial evaluation of a patient suspected of hypovolemic shock follows the principles of the Advanced Trauma Life Support (ATLS) algorithm, often referred to as the "ABCs" of emergency care: Airway, Breathing, Circulation, Disability, and Exposure [20].

- 1. **Airway (A)**: Ensure that the patient's airway is clear. In severe cases, alterations in consciousness may threaten airway patency. Supplemental oxygen should be provided early to improve oxygenation [21].
- 2. **Breathing (B)**: Assess the effectiveness of breathing. Look for signs of respiratory distress, such as tachypnea, use of accessory muscles, or cyanosis. Administer oxygen as needed and consider advanced airway management in cases of respiratory failure.
- 3. Circulation (C): Initiate circulation assessments promptly. Check pulse rate, blood pressure, and capillary refill time. Tachycardia (typically over 100 beats per minute) and hypotension (systolic blood pressure under 90 mmHg) are hallmark signs of hypovolemic shock. Central and peripheral pulse quality can indicate the severity of volume loss. External bleeding should be controlled if present, and internal bleeding may require imaging studies or surgical intervention [21].
- 4. **Disability (D)**: Evaluate neurological status using the Glasgow Coma Scale (GCS). decreased level of consciousness or confusion may suggest inadequate perfusion affecting the brain.

5. **Exposure (E)**: Fully expose the patient to assess for additional injuries or signs of trauma while maintaining normothermia to prevent hypothermia [22].

#### **B.** Secondary Survey

Following the primary survey, a more thorough secondary survey should be conducted to identify potential sources of blood loss or contributing factors to the hypovolemic state:

- Patient History: Obtain a comprehensive history, including the mechanism of injury, medical background, and medications. This information can provide clues regarding preexisting conditions that may exacerbate the shock, like anticoagulant therapy or coagulopathies.
- 2. **Physical Examination**: Conduct a complete head-to-toe examination. Look for signs of trauma, such as contusions, abrasions, or deformities. Abdominal distension may indicate intra-abdominal hemorrhage.
- 3. **Diagnostic Tests**: Laboratories should initiate studies such as complete blood count (CBC), electrolytes, and type and crossmatching for potential blood transfusion. Imaging studies like ultrasound (FAST exam) can also help identify intra-abdominal bleeding [23].

#### **Emergency Triage**

Triage in the context of hypovolemic shock is crucial for prioritizing care based on severity. The following stratification outlines the key components of effective triage:

- 1. Classifying Shock Severity: The classification of shock severity typically revolves around the percentage of blood volume lost:
  - Class I Shock: 15% blood volume loss (up to 750 mL); pulse rate may be slightly elevated but blood pressure may remain normal.
  - Class II Shock: 15-30% blood volume loss (750-1500 mL); mild tachycardia and hypotension start to emerge.
  - Class III Shock: 30-40% blood volume loss (1500-2000 mL); marked

- tachycardia (over 120 bpm) and hypotension become prominent.
- Class IV Shock: Greater than 40% blood volume loss (more than 2000 mL); severe hypotension, bradycardia, altered mental status, and decreased urination [24].

# 2. Immediate Actions Based on Triage Levels:

- Class I and II: Establish venous access, initiate fluid resuscitation with crystalloids (e.g., normal saline or lactated Ringer's solution) and monitor vital signs closely. Blood transfusion might not be immediately necessary at this stage.
- Class III and IV: Rapidly initiate fluid resuscitation with isotonic fluids and prepare for potential blood product administration (packed red blood cells, plasma). Surgical intervention may be required based on internal bleeding.
- 3. Monitoring and Reassessment:
  Continuous monitoring of vital signs, urine output, and mental status is essential to guide the therapeutic approach. A reassessment should occur every 5-15 minutes during resuscitation [25].

#### Fluid Resuscitation Strategies:

Hypovolemic shock is a critical condition characterized by a significant reduction in circulating blood volume, leading to inadequate tissue perfusion and oxygenation. It can result from various causes, primarily including hemorrhage (due to trauma, surgery, or gastrointestinal bleeding) and severe dehydration (from vomiting, diarrhea, or excessive fluid loss in burns). Swift and effective intervention is essential; among these interventions, fluid resuscitation plays a pivotal role [26].

The human body relies on a delicate balance of components that ensure adequate perfusion to vital organs. When fluid volume decreases significantly, the body can no longer maintain this equilibrium, paving the way for a series of pathological changes. The decrease in intravascular volume hampers cardiac output, which, combined with vasodilation and increased vascular permeability, leads to tissue hypoxia. Regions of the body are deprived of oxygen, leading to cellular injury and potentially

contributing to irreversible damage if not addressed promptly [27].

Hypovolemic shock is clinically identifiable by several signs including tachycardia, hypotension, altered mental status, and decreased urine output. Establishing a precise diagnosis quickly is necessary for effective treatment planning [28].

Fluid resuscitation aims to restore circulating volume and improve tissue perfusion. The primary goals include normalizing hemodynamic parameters (blood pressure and heart rate), optimizing oxygen delivery to tissues, and enhancing renal perfusion. The two main approaches for fluid resuscitation in hypovolemic shock are the 'EFAST' (Early Fluid Administration for Shock Treatment) principles, which advocate for early aggressive resuscitation especially in trauma patients, and a more cautious strategy which prefers the assessment of the patient's response to initial fluid administration before further resuscitation efforts [29].

Fluid resuscitation can be categorized into two phases: initial and maintenance. The initial phase involves rapid infusion of fluids to restore circulation and improve organ function, while the maintenance phase ensures ongoing restoration and correction of any fluid or electrolyte imbalances [30].

# **Types of Fluid Solutions**

Fluid resuscitation employs various types of intravenous (IV) fluids categorized primarily into crystalloids and colloids.

- 1. Crystalloids: These are aqueous solutions containing electrolytes, such as normal saline (0.9% sodium chloride), lactated Ringer's solution, and dextrose solutions. Crystalloids are often preferred in the initial resuscitation phase due to their safety profile, ready availability, and low cost. Studies have demonstrated that isotonic crystalloids like normal saline and lactated Ringer's solution are effective in restoring intravascular volume. However, excessive use may lead to dilutional coagulopathy and electrolyte imbalances [31].
- Colloids: Colloid solutions, typically containing larger molecules, aim to maintain oncotic pressure and increase intravascular volume more effectively than crystalloids. Common examples include hydroxyethyl

starch (HES), dextran, and albumin. However, their clinical use has been a subject of controversy. While some studies suggest that colloids can enhance hemodynamic stability, others point towards potential adverse effects, including kidney injury, coagulopathy, and increased mortality in certain populations [31].

The choice of fluid type may depend on the etiology of shock, clinical context, and the patient's specific needs. For example, in cases involving severe hemorrhage, rapid crystalloid resuscitation is often employed initially to quickly restore intravascular volume, followed by targeted transfusion of blood products as necessary [32].

#### **Monitoring and Response Assessment**

Effective fluid resuscitation is not just about the volume of fluids administered; it is equally about carefully monitoring the patient's response. Parameters to monitor include vital signs, urine output, mental status, and laboratory values like lactate levels and hemoglobin concentration [32].

Fluid responsiveness can be assessed using dynamic measures such as stroke volume variation or pulse pressure variation, especially in mechanically ventilated patients. Early identification of fluid overload, indicated by signs such as elevated jugular venous pressure or pulmonary edema, is critical to guide fluid resuscitation efforts and avoid complications.

Recent literature has begun to emphasize a more personalized approach to fluid resuscitation. Individualized treatment algorithms assess patient-specific characteristics such as age, comorbidities, and the underlying cause of hypovolemic shock [32].

The concept of 'goal-directed therapy' has emerged, focusing on maintaining specific hemodynamic targets (e.g., mean arterial pressure and central venous pressure) during resuscitation. This precision medicine approach also incorporates advanced monitoring technologies like echocardiography or minimally invasive cardiac output devices to optimize fluid administration [33].

Additionally, there is growing interest in the 'early goal-directed therapy' (EGDT) paradigm, particularly in septic shock. Research continues to investigate the optimal timing, volume, and type of fluids to administer during resuscitation, suggesting

that timing and precision are vital for reducing mortality [34].

# **Management of Underlying Causes:**

Hypovolemic shock is a life-threatening condition resulting from inadequate circulating blood volume, leading to reduced tissue perfusion and insufficient oxygen delivery to essential organs. This physiological state can arise from various underlying causes, including hemorrhage, severe dehydration, and fluid loss due to burns, vomiting, or diarrhea. The effective management of hypovolemic shock necessitates a comprehensive understanding of its underlying causes, timely identification of the etiology, and appropriate medical intervention strategies that target these root causes to restore hemodynamic stability and mitigate the risk of multi-organ failure [35].

Before delving into management strategies, it is crucial to understand the pathophysiology of hypovolemic shock. When there is a significant loss of blood volume—typically defined as more than 15% or 750 mL in an adult—the body's compensatory mechanisms are activated. These compensatory responses aim to maintain blood pressure and ensure adequate perfusion to vital organs. The body starts to retain sodium and water through hormonal pathways, primarily mediated by the renin-angiotensin-aldosterone system (RAAS), and increases heart rate and contractility in an effort to tighten peripheral blood vessels [36].

However, these compensatory mechanisms have their limits. As hypovolemia progresses, clinical signs such as tachycardia, hypotension, altered mental status, oliguria, and cold clammy extremities become evident. If not promptly addressed, hypovolemic shock can culminate in multi-organ dysfunction, leading to irreversible damage and eventual mortality [36].

# **Identification of Underlying Causes**

The management of hypovolemic shock begins with the identification of its underlying cause. Accurate diagnosis is crucial for targeting interventions effectively. In clinical practice, this typically involves a thorough history-taking, physical examination, and relevant laboratory and imaging studies.

1. Hemorrhagic Causes: One of the most common causes of hypovolemic shock is

hemorrhage, which can result from trauma, surgical complications, gastrointestinal bleeding, or obstetrical issues. Rotational trauma in accidents can lead to splenic or liver lacerations, while ruptured ectopic pregnancies can be a significant source of internal bleeding in women [37].

- Fluid Loss: Non-hemorrhagic causes of fluid loss can occur due to severe dehydration, often resulting from gastrointestinal losses due to vomiting or diarrhea or from excessive sweating. Fluid loss can also occur via the skin in the case of extensive burns or from thirdspacing in conditions such as pancreatitis or sepsis.
- Anaphylaxis and Sepsis: Severe allergies can instigate systemic reactions that lead to vascular leakage, resulting in significant intravascular fluid depletion. Similarly, septic shock can spur a considerable inflammatory response, leading to increased permeability of blood vessels and resultant hypovolemic shock [38].

#### **Management Strategies**

Once the underlying cause has been identified, management should proceed through targeted interventions aimed at addressing these factors while simultaneously ensuring immediate resuscitation of the patient [39].

In all cases of hypovolemic shock, immediate and aggressive fluid resuscitation is paramount. This often starts with the administration of crystalloid solutions such as Normal Saline or Lactated Ringer's. The Parkland Formula can be utilized in cases of burn-induced shock to estimate the volume of fluids required. In major hemorrhagic cases, transfusion of packed red blood cells (PRBCs), fresh frozen plasma, and platelets may be necessary to correct coagulopathy and restore blood volume.

The "massive transfusion protocol" may be enacted in cases where substantial blood loss has occurred, typically defined as the rapid administration of more than 10 units of PRBCs within 24 hours. However, clinicians must balance the risks of transfusion-related complications with therapeutic benefits [39].

In cases of hemorrhagic hypovolemic shock, controlling the source of bleeding is essential. This may involve surgical intervention to ligate blood

vessels, repair traumatic injuries, or address gastrointestinal bleeds through endoscopy. In instances of acute trauma care, damage control surgery may be necessary to stabilize the patient before definitive repairs can be safely performed.

For patients suffering from hypovolemic shock due to dehydration, efforts should focus on rehydration strategies, which may involve the administration of oral rehydration solutions or intravenous fluids. Electrolyte imbalances should be corrected by monitoring serum electrolytes and adjusting fluid therapy accordingly [40].

In cases of significant vomiting or diarrhea, antiemetic and antidiarrheal medications may be warranted, and the underlying etiology, such as infection or toxicity, should be evaluated for specific treatment strategies.

When hypovolemic shock is secondary to sepsis or anaphylaxis, the management should include the use of broad-spectrum antibiotics in the case of sepsis, along with vasopressors for hemodynamic support if fluid resuscitation alone does not improve blood pressure. Anaphylactic shock necessitates immediate administration of epinephrine, along with antihistamines and corticosteroids [41].

# **Monitoring and Reassessment:**

Hypovolemic shock is a life-threatening condition that arises from a significant loss of blood volume, leading to inadequate perfusion of tissues and organs. This medical emergency requires immediate recognition and management to prevent multi-organ failure and reduce mortality rates. An effective intervention involves a continuous process of monitoring and re-evaluation, which ensures that treatment protocols are tailored to the evolving needs of the patient. Hypovolemic shock is characterized by a decrease in intravascular volume, which compromises the ability of the heart to pump blood efficiently. It can result from various factors, including:

- 1. **Hemorrhage**: This is the most common cause and may occur due to trauma, surgical complications, gastrointestinal bleeding, or obstetric emergencies [42].
- Fluid Loss: Conditions like severe dehydration from vomiting, diarrhea, or burns can lead to significant fluid loss

without a corresponding decrease in blood volume.

3. **Third Space Loss**: Situations where fluid shifts from the intravascular space to the interstitial space, such as in severe sepsis or pancreatitis, contribute to hypovolemia without direct blood loss [42].

Each of these scenarios can precipitate a cascade of physiological changes that, without intervention, leads to decreased organ perfusion, cellular dysfunction, and potentially irreversible consequences.

#### **Clinical Manifestations**

Patients in hypovolemic shock exhibit a range of clinical signs and symptoms. These can be categorized into early and advanced manifestations:

- Early Signs: Increased heart rate (tachycardia), mild hypotension, altered mental status (anxiety), and cool, clammy skin [43].
- Advanced Signs: Persistent hypotension, weak or thready pulse, significantly altered consciousness (confusion or lethargy), mottled skin, and decreased urine output.

Recognizing these changes is crucial for prompt intervention. The severity of the shock can often be gauged by the degree of tachycardia and hypotension, with more pronounced symptoms indicating a more critical state.

# **Initial Management Strategies**

The cornerstone of treating hypovolemic shock is rapid fluid resuscitation to restore hemodynamic stability. The initial management protocol typically includes:

- 1. Intravenous (IV) Fluid Replacement: Isotonic crystalloids, such as Normal Saline or Lactated Ringer's solution, are administered to replenish lost volume. In cases of significant hemorrhage, packed red blood cells might be used.
- Control of Cause: Identifying and controlling the source of volume loss is critical. For example, in cases of hemorrhage, surgical intervention may be necessary [44].

- 3. **Monitoring**: Continuous monitoring of vital signs, urine output, and level of consciousness is imperative to guide treatment decisions.
- 4. **Medications**: In some cases, vasopressors may be required to manage persistent hypotension, particularly during advanced stages of shock [44].

# **Monitoring Techniques**

Effective monitoring is essential in the management of hypovolemic shock, providing real-time feedback on the patient's response to treatment. This includes:

- Vital Signs: Continuous tracking of heart rate, blood pressure, respiratory rate, and temperature provides immediate insight into the patient's hemodynamic status [45].
- 2. **Urine Output**: Monitoring urine output provides a direct measure of renal perfusion and can alert the healthcare team to inadequate fluid resuscitation.
- 3. **Pulse Oximetry**: Assessment of oxygen saturation levels helps evaluate the adequacy of oxygen delivery to tissues.
- 4. **Laboratory Tests**: Regular blood tests, including complete blood count, electrolyte levels, and lactate, provide insight into the patient's metabolic state and overall fluid balance.
- 5. Central Venous Pressure (CVP): Invasive monitoring techniques, such as CVP measurements, can offer further detail regarding fluid status and cardiac function, guiding fluid resuscitation strategies [45].

# **Re-evaluation Protocol**

Re-evaluation is a critical component of the management plan for hypovolemic shock. This involves a systematic reassessment of the patient's clinical status after interventions have been initiated. A few key strategies include:

- 1. **Frequent Assessment Intervals**: Regular reassessment (every 15-30 minutes initially) is crucial for early detection of deterioration or improvement [46].
- Response to Treatment: Evaluating the patient's response to fluid resuscitation and other interventions. Improvement in blood

- pressure, heart rate normalization, and urine output is indicative of effective management.
- 3. Utilization of Scoring Systems: Employing clinical scoring systems like the Shock Index (heart rate/systolic blood pressure ratio) can help gauge the severity of shock and guide subsequent treatment decisions.
- 4. **Multidisciplinary Approach**: Engaging various specialists, including intensivists, trauma surgeons, and nursing staff, facilitates comprehensive care and timely intervention in response to evolving clinical scenarios [46].

#### **Complications and Long-Term Considerations:**

Hypovolemic shock is a critical condition that arises from a significant reduction in the intravascular volume, leading to inadequate perfusion of organs and tissues. It is commonly precipitated by severe blood loss, dehydration, or fluid loss due to conditions such as burns or severe diarrhea. Understanding the complications and long-term considerations that follow hypovolemic shock is essential for clinicians, patients, and caregivers, as it can significantly impact health outcomes and quality of life [47].

# Pathophysiology of Hypovolemic Shock

Before delving into the complications and long-term considerations, it is crucial to grasp the underlying pathophysiology of hypovolemic shock. When the body experiences a loss of volume, it initiates compensatory mechanisms such as increased heart rate and peripheral vasoconstriction. However, if fluid loss is not promptly addressed, these compensatory mechanisms can be overwhelmed. The reduced cardiac output results in inadequate oxygen delivery to tissues, leading to cellular dysfunction and ultimately multi-organ failure if the shock state persists [48].

# **Immediate Complications**

1. Organ Dysfunction: One of the most alarming complications of hypovolemic shock is the potential for organ dysfunction. Organs such as the kidneys, liver, and heart are particularly susceptible to ischemic injury due to prolonged inadequate perfusion. Renal failure, for instance, can occur as a result of acute tubular necrosis stemming from sustained hypoxia. Severe cases may lead to the need for dialysis [49].

**2.** Coagulation Disorders: The state of hypovolemic shock can also predispose patients to coagulopathy. The body's clotting mechanisms may be disrupted due to reduced blood volume and alterations in platelet function, leading to an increased risk of both bleeding and thrombosis.

- **3. Metabolic Disturbances:** The metabolic derangements that accompany hypovolemic shock may include lactic acidosis due to anaerobic metabolism. Prolonged acidosis can impair cardiac function and lead to further hemodynamic instability [50].
- **4.** Acute Respiratory Distress Syndrome (ARDS): In severe cases of hypovolemic shock, there is an increased risk of developing ARDS, a life-threatening condition characterized by sudden pulmonary inflammation and edema, leading to impaired gas exchange [51].

#### **Long-Term Considerations**

The ramifications of hypovolemic shock can extend well beyond the acute episode, influencing both physical and psychological aspects of a patient's health [52].

- 1. Psychological Impact: The psychological ramifications of experiencing a traumatic event that leads to hypovolemic shock can be profound. Patients may develop post-traumatic stress disorder (PTSD), anxiety, or depression due to the event's life-threatening nature. Mental health support is crucial to address these concerns and facilitate healing.
- 2. Persistent Fatigue and Weakness: Many survivors of hypovolemic shock report prolonged periods of fatigue and generalized weakness. This fatigue may stem from a combination of factors, including muscle deconditioning due to immobility, nutritional deficiencies, and the psychological toll of the traumatic experience [53].
- **3. Risk of Recurrence:** There exists a concerning potential for recurrence in cases of hypovolemic shock precipitated by an underlying condition, such as gastrointestinal bleeding or severe dehydration. Therefore, long-term management of any contributing factors is crucial to mitigate the risk of experiencing subsequent episodes. [54]
- **4. Impaired Quality of Life:** Survivors of hypovolemic shock may also experience a decline in

their overall quality of life. This could be due to physical limitations, persistent pain, psychological distress, or changes in lifestyle. Ongoing rehabilitation may be necessary to help patients regain their functional status [55].

# Rehabilitative Strategies

A multidimensional approach to rehabilitation is crucial for addressing the acute and long-term consequences of hypovolemic shock. Such approaches may involve:

- 1. Physical Therapy: Targeted physical therapy can aid in restoring strength, mobility, and endurance. Gradual reintroduction of physical activity tailored to the patient's specific needs allows for a monitored, safe recovery [56].
- **2. Nutritional Support:** Patients recovering from hypovolemic shock often face nutritional deficits due to their underlying condition or prolonged hospitalization. Nutritional assessment and intervention should be prioritized to support recovery and improve overall health [57].
- **3. Psychological Support:** As noted, the psychological impact of hypovolemic shock can be significant. Offering access to mental health resources, including counseling or support groups, can facilitate the emotional healing process, minimizing the risk of long-term mental health issues [58].
- **4. Patient Education:** Educating patients about their condition, recognizing signs of future complications, and understanding the importance of adherence to any follow-up care recommended by healthcare providers is a crucial step in promoting long-term health outcomes [59].

#### **Conclusion:**

The effective management of hypovolemic shock in emergency settings is critical for improving patient outcomes and reducing mortality. Timely recognition, prompt intervention, and a systematic approach to fluid resuscitation are essential components of care. Emphasizing a thorough initial assessment allows healthcare providers to identify the underlying causes of fluid loss, enabling targeted treatments that can stabilize the patient and restore hemodynamic balance. The use of appropriate fluid therapy, alongside continuous monitoring and reassessment, ensures that healthcare teams can

effectively adapt treatment plans based on the patient's response.

In addition to immediate resuscitative efforts, addressing the root causes of hypovolemic shock is vital to prevent recurrence and complications. among emergency personnel, Collaboration surgeons, and critical care teams is paramount to delivering comprehensive care. As protocols continue to evolve, ongoing education and adherence to established guidelines will enhance the ability to manage hypovolemic shock effectively, ensuring that patients receive the best possible outcomes during these critical moments. Continued research into emerging practices and technologies will further contribute to advancements in care, ultimately improving survival rates and quality of life for individuals experiencing this life-threatening condition.

#### **References:**

- Giannoudi M., Harwood P. Damage control resuscitation: lessons learned. Eur J Trauma Emerg Surg. 2016;42(3):273–282. doi: 10.1007/s00068-015-0628-3.
- 2. Mutschler M., Paffrath T., Wölfl C. The ATLS(®) classification of hypovolaemic shock: a well established teaching tool on the edge? Injury. 2014;45(Suppl 3):S35–S38. doi: 10.1016/j.injury.2014.08.015.
- Abhilash K.P., Chakraborthy N., Pandian G.R., Dhanawade V.S., Bhanu T.K., Priya K. Profile of trauma patients in the emergency department of a tertiary care hospital in South India. J Family Med Prim Care. 2016;5(3):558–563. doi: 10.4103/2249-4863.197279.
- Ciechanowicz D., Samojło N., Kozłowski J., Pakulski C., Żyluk A. Incidence and etiology of mortality in polytrauma patients: an analysis of material from Multitrauma Centre of the University Teaching Hospital no 1 in Szczecin, over a period of 3 years (2017-2019). Pol Przegl Chir. 2020;92(4):1–6. doi: 10.5604/01.3001.0014.1127.
- Ibrahim I., Chor W.P., Chue K.M. Is arterial base deficit still a useful prognostic marker in trauma? A systematic review. Am J Emerg Med. 2016;34(3):626–635. doi: 10.1016/j.ajem.2015.12.012.

- Eastridge B.J., Holcomb J.B., Shackelford S. Outcomes of traumatic hemorrhagic shock and the epidemiology of preventable death from injury. Transfusion. 2019;59(S2):1423–1428. doi: 10.1111/trf.15161.
- 7. Paffrath T., Lefering R., Flohé S., TraumaRegister D.G.U. How to define severely injured patients? an Injury Severity Score (ISS) based approach alone is not sufficient. Injury. 2014;45(Suppl 3):S64–S69. doi: 10.1016/j.injury.2014.08.020.
- 8. Mutschler M., Nienaber U., Brockamp T. A critical reappraisal of the ATLS classification of hypovolaemic shock: does it really reflect clinical reality? Resuscitation. 2013;84(3):309–313. doi: 10.1016/j.resuscitation.2012.07.012.
- Wise R., Faurie M., Malbrain M.L.N.G., Hodgson E. Strategies for intravenous fluid resuscitation in trauma patients. World J Surg. 2017;41(5):1170–1183. doi: 10.1007/s00268-016-3865-7.
- Roy N., Kizhakke Veetil D., Khajanchi M.U. Learning from 2523 trauma deaths in Indiaopportunities to prevent in-hospital deaths. BMC Health Serv Res. 2017;17(1):142. doi: 10.1186/s12913-017-2085-7.
- 11. Fox E.E., Holcomb J.B., Wade C.E., Bulger E.M., Tilley B.C., Proppr Study Group. Earlier endpoints are required for hemorrhagic shock trials among severely injured patients. Shock. 2017;47(5):567–573. doi: 10.1097/SHK.00000000000000788.
- Trajano A.D., Pereira B.M., Fraga G.P. Epidemiology of in-hospital trauma deaths in a Brazilian university hospital. BMC Emerg Med. 2014;14:22. doi: 10.1186/1471-227X-14-22.
- 13. Wen Y., Yang H., Wei W., Shan-shou L. The outcomes of 1120 severe multiple trauma patients with hemorrhagic shock in an emergency department: a retrospective study. BMC Emerg Med. 2013;13 Suppl 1:S6. doi: 10.1186/1471-227X-13-S1-S6.
- 14. Guly H.R., Bouamra O., Spiers M., Dark P., Coats T., Lecky F.E. Trauma Audit and Research Network. Vital signs and estimated

- blood loss in patients with major trauma: testing the validity of the ATLS classification of hypovolaemic shock. Resuscitation. 2011;82(5):556–559. doi: 10.1016/j.resuscitation.2011.01.013.
- Weber C.D., Lefering R., Dienstknecht T. Classification of soft-tissue injuries in open femur fractures: relevant for systemic complications? J Trauma Acute Care Surg. 2016;81(5):824–833. doi: 10.1097/TA.0000000000001216.
- Petrosoniak A., Hicks C. Resuscitation resequenced: a rational approach to patients with trauma in shock. Emerg Med Clin North Am. 2018;36(1):41–60. doi: 10.1016/j.emc.2017.08.005.
- 17. Squara P, Hollenberg S, Payen D: Reconsidering vasopressors for cardiogenic shock: Everything should be made as simple as possible, but not simpler. Chest 156: 392–401, 2019.
- 18. Kim IY, Kim JH, Lee DW, Lee SB, Rhee H, Seong EY, Kwak IS, Song SH: Fluid overload and survival in critically ill patients with acute kidney injury receiving continuous renal replacement therapy. PLoS One 12: e0172137, 2017.
- Biancofiore G, Bindi L, Romanelli AM, Bisà M, Boldrini A, Consani G, Danella A, Urbani L, Filipponi F, Mosca F: Renal failure and abdominal hypertension after liver transplantation: Determination of critical intraabdominal pressure. Liver Transpl 8: 1175– 1181, 2002.
- Dalfino L, Tullo L, Donadio I, Malcangi V, Brienza N: Intra-abdominal hypertension and acute renal failure in critically ill patients. Intensive Care Med 34: 707–713, 2008.
- 21. Woodward CW, Lambert J, Ortiz-Soriano V, Li Y, Ruiz-Conejo M, Bissell BD, Kelly A, Adams P, Yessayan L, Morris PE, Neyra JA: Fluid overload associates with major adverse kidney events in critically ill patients with acute kidney injury requiring continuous renal replacement therapy. Crit Care Med 47: e753– e760, 2019.

- 22. Malbrain MLNG, Van Regenmortel N, Saugel B, De Tavernier B, Van Gaal PJ, Joannes-Boyau O, Teboul JL, Rice TW, Mythen M, Monnet X: Principles of fluid management and stewardship in septic shock: It is time to consider the four D's and the four phases of fluid therapy. Ann Intensive Care 8: 66, 2018.
- 23. Ostermann M, Liu K, Kashani K: Fluid management in acute kidney injury. Chest 156: 594–603, 2019.
- 24. Prowle JR, Echeverri JE, Ligabo EV, Ronco C, Bellomo R: Fluid balance and acute kidney injury. Nat Rev Nephrol 6: 107–115, 2010.
- 25. Zarbock A, Küllmar M, Ostermann M, Lucchese G, Baig K, Cennamo A, Rajani R, McCorkell S, Arndt C, Wulf H, Irqsusi M, Monaco F, Di Prima AL, García Alvarez M, Italiano S, Miralles Bagan J, Kunst G, Nair S, L'Acqua C, Hoste E, Vandenberghe W, Honore PM, Kellum JA, Forni LG, Grieshaber P, Massoth C, Weiss R, Gerss J, Wempe C, Meersch M: Prevention of cardiac surgerykidney injury associated acute implementing the KDIGO guidelines in highrisk patients identified by biomarkers: The PrevAKI-multicenter randomized controlled trial. Anesth Analg 133: 292-302, 2021.
- Chen KP, Cavender S, Lee J, Feng M, Mark RG, Celi LA, Mukamal KJ, Danziger J: Peripheral edema, central venous pressure, and risk of AKI in critical illness. Clin J Am Soc Nephrol 11: 602–608, 2016.
- 27. Sharfuddin AA, Molitoris BA: Pathophysiology of ischemic acute kidney injury. Nat Rev Nephrol 7: 189–200, 2011.
- 28. Lim HS: Cardiogenic shock: Failure of oxygen delivery and oxygen utilization. Clin Cardiol 39: 477–483, 2016.
- 29. Mullens W, Abrahams Z, Skouri HN, Francis GS, Taylor DO, Starling RC, Paganini E, Tang WH: Elevated intra-abdominal pressure in acute decompensated heart failure: A potential contributor to worsening renal function? J Am Coll Cardiol 51: 300–306, 2008.
- 30. Vincent JL, De Backer D: Circulatory shock. N Engl J Med 369: 1726–1734, 2013.

- De Backer D, Orbegozo Cortes D, Donadello K, Vincent JL: Pathophysiology of microcirculatory dysfunction and the pathogenesis of septic shock. Virulence 5: 73–79, 2014.
- 32. Beaubien-Souligny W, Bouchard J, Desjardins G, Lamarche Y, Liszkowski M, Robillard P, Denault A: Extracardiac signs of fluid overload in the critically ill cardiac patient: A focused evaluation using bedside ultrasound. Can J Cardiol 33: 88–100, 2017.
- 33. Winton FR: The influence of venous pressure on the isolated mammalian kidney. J Physiol 72: 49–61, 1931.
- 34. Saito S, Uchino S, Takinami M, Uezono S, Bellomo R: Postoperative blood pressure deficit and acute kidney injury progression in vasopressor-dependent cardiovascular surgery patients. Crit Care 20: 74, 2016.
- Cruces P, Salas C, Lillo P, Salomon T, Lillo F, Hurtado DE: The renal compartment: A hydraulic view. Intensive Care Med Exp 2: 26, 2014.
- 36. Kim IY, Kim JH, Lee DW, Lee SB, Rhee H, Seong EY, Kwak IS, Song SH: Fluid overload and survival in critically ill patients with acute kidney injury receiving continuous renal replacement therapy. PLoS One 12: e0172137, 2017.
- 37. Yealy DM, Kellum JA, Huang DT, Barnato AE, Weissfeld LA, Pike F, Terndrup T, Wang HE, Hou PC, LoVecchio F, Filbin MR, Shapiro NI, Angus DC; ProCESS Investigators: A randomized trial of protocolbased care for early septic shock. N Engl J Med 370: 1683–1693, 2014.
- 38. Legrand M, Dupuis C, Simon C, Gayat E, Mateo J, Lukaszewicz AC, Payen D: Association between systemic hemodynamics and septic acute kidney injury in critically ill patients: A retrospective observational study. Crit Care 17: R278, 2013.
- 39. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M; Early Goal-Directed Therapy Collaborative Group: Early goal-directed therapy in the treatment of severe sepsis and

- septic shock. N Engl J Med 345: 1368-1377, 2001.
- 40. Myles PS, Bellomo R, Corcoran T, Forbes A, Peyton P, Story D, Christophi C, Leslie K, McGuinness S, Parke R, Serpell J, Chan MTV, Painter T, McCluskey S, Minto G, Wallace S; Australian and New Zealand College of Anaesthetists Clinical Trials Network and the Australian and New Zealand Intensive Care Society Clinical Trials Group: Restrictive versus liberal fluid therapy for major abdominal surgery. N Engl J Med 378: 2263–2274, 2018.
- 41. Hjortrup PB, Haase N, Bundgaard H, Thomsen SL, Winding R, Pettilä V, Aaen A, Lodahl D, Berthelsen RE, Christensen H, Madsen MB, Winkel P, Wetterslev J, Perner A; CLASSIC Trial Group; Scandinavian Critical Care Trials Group: Restricting volumes of resuscitation fluid in adults with septic shock after initial management: The CLASSIC randomised, parallel-group, multicentre feasibility trial. Intensive Care Med 42: 1695–1705, 2016.
- 42. Langenberg C, Bellomo R, May C, Wan L, Egi M, Morgera S: Renal blood flow in sepsis. Crit Care 9: R363–R374, 2005.
- 43. Mouncey PR, Osborn TM, Power GS, Harrison DA, Sadique MZ, Grieve RD, Jahan R, Harvey SE, Bell D, Bion JF, Coats TJ, Singer M, Young JD, Rowan KM; ProMISe Trial Investigators: Trial of early, goal-directed resuscitation for septic shock. N Engl J Med 372: 1301–1311, 2015.
- 44. Jansen JR, Maas JJ, Pinsky MR: Bedside assessment of mean systemic filling pressure. Curr Opin Crit Care 16: 231–236, 2010.
- 45. Verbrugge FH, Mullens W, Malbrain M: Worsening renal function during decompensated heart failure: The cardio-abdomino-renal syndrome. In: Annual Update in Intensive Care and Emergency Medicine 2012, edited by Vincent J-L, Berlin, Springer, 2012, pp 577–588.
- 46. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, Bellomo R, Bernard GR, Chiche JD, Coopersmith CM, Hotchkiss RS, Levy MM, Marshall JC, Martin GS, Opal SM, Rubenfeld

- GD, van der Poll T, Vincent JL, Angus DC: The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA 315: 801–810, 2016.
- 47. Damman K, van Deursen VM, Navis G, Voors AA, van Veldhuisen DJ, Hillege HL: Increased central venous pressure is associated with impaired renal function and mortality in a broad spectrum of patients with cardiovascular disease. J Am Coll Cardiol 53: 582–588, 2009.
- 48. Peake SL, Delaney A, Bailey M, Bellomo R, Cameron PA, Cooper DJ, Higgins AM, Holdgate A, Howe BD, Webb SA, Williams P; ARISE Investigators; ANZICS Clinical Trials Group: Goal-directed resuscitation for patients with early septic shock. N Engl J Med 371: 1496–1506, 2014.
- 49. Hjortrup PB, Haase N, Bundgaard H, Thomsen SL, Winding R, Pettilä V, Aaen A, Lodahl D, Berthelsen RE, Christensen H, Madsen MB, Winkel P, Wetterslev J, Perner A: Restricting volumes of resuscitation fluid in adults with septic shock after initial management: The CLASSIC randomised, parallel-group, multicentre feasibility trial. Intensive Care Med 42: 1695–1705, 2016.
- 50. Wheeler AP, Bernard GR, Thompson BT, Schoenfeld D, Wiedemann HP, deBoisblanc B, Connors AF Jr., Hite RD, Harabin AL; National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network: Pulmonary-artery versus central venous catheter to guide treatment of acute lung injury. N Engl J Med 354: 2213–2224, 2006.
- 51. Pinsky MR: Functional haemodynamic monitoring. Curr Opin Crit Care 20: 288–293, 2014.
- 52. Bradley RD: Diagnostic right-heart catheterisation with miniature catheters in severely ill patients. Lancet 2: 941–942, 1964.
- 53. Monnet X, Marik P, Teboul JL: Passive leg raising for predicting fluid responsiveness: A systematic review and meta-analysis. Intensive Care Med 42: 1935–1947, 2016.
- 54. Connors AF Jr., Speroff T, Dawson NV, Thomas C, Harrell FE Jr., Wagner D, Desbiens

- N, Goldman L, Wu AW, Califf RM, Fulkerson WJ Jr., Vidaillet H, Broste S, Bellamy P, Lynn J, Knaus WA; SUPPORT Investigators: The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 276: 889–897, 1996.
- 55. Sugimoto M, Takayama W, Murata K, Otomo Y: The impact of lactate clearance on outcomes according to infection sites in patients with sepsis: A retrospective observational study. Sci Rep 11: 22394, 2021.
- 56. Marik PE, Baram M, Vahid B: Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest 134: 172–178, 2008.
- 57. Godin M, Murray P, Mehta RL: Clinical approach to the patient with AKI and sepsis. Semin Nephrol 35: 12–22, 2015.
- 58. Bednarczyk JM, Fridfinnson JA, Kumar A, Blanchard L, Rabbani R, Bell D, Funk D, Turgeon AF, Abou-Setta AM, Zarychanski R: Incorporating dynamic assessment of fluid responsiveness into goal-directed therapy: A systematic review and meta-analysis. Crit Care Med 45: 1538–1545, 2017.
- 59. Pinsky MR: Functional hemodynamic monitoring. Crit Care Clin 31: 89–111, 2015.