CRITICAL INTERSECTIONS IN EMERGENCY MEDICINE

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APPROACH TO SHOCK PATIENT IN EMERGENCY DEPARTMENT

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Definition and Pathophysiology of Shock

Shock is a condition characterized by the circulatory system's inability to adequately perfuse tissues, resulting in an oxygen supply that falls short of metabolic demand (Koya & Paul, 2023). Consequently, cells become deprived of oxygen and shift to anaerobic metabolism, leading to lactic acidosis and early-stage, potentially reversible organ dysfunction. If not promptly recognized and treated, this perfusion deficit can cause irreversible cellular damage, culminating in multiple organ failure and death (Koya & Paul, 2023b). Although hypotension (systolic blood pressure <90 mmHg or MAP <65 mmHg) is commonly observed in shock, blood pressure may appear normal in the early stages due to compensatory mechanisms ("Approach to Shock," n.d.).

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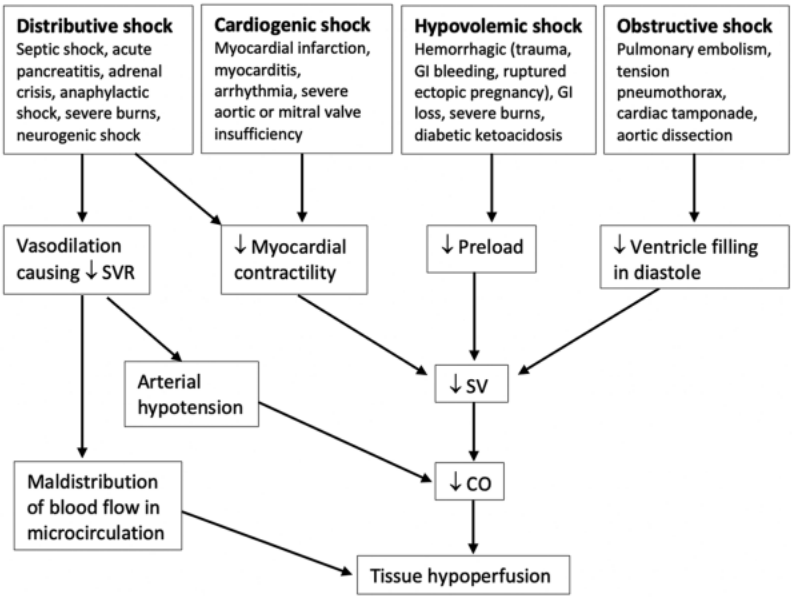
Physiologically, the body responds to shock through activation of the sympathetic nervous system and the hormonal system, manifesting as tachycardia, vasoconstriction, and fluid retention. While these responses initially aim to preserve perfusion of vital organs, unresolved underlying causes can lead to worsening tissue hypoxia, increased capillary permeability, and the release of inflammatory mediators. Consequently, processes such as myocardial depression, vasodilation, and intravascular volume loss further exacerbate the shock state. In the later stages, circulatory collapse and irreversible damage occur, ultimately progressing to refractory shock.

Classification of Shock

Shock is broadly categorized into four main groups based on the underlying pathophysiological mechanism: hypovolemic, cardiogenic, distributive, and obstructive shock (Koya & Paul, 2023c). This classification is grounded in the primary derangement that precipitates the shock state (e.g., volume depletion, cardiac pump failure, loss of vascular tone, or a mechanical obstruction to blood flow). A summary of these shock types and their subtypes is provided below.

Figure: Schematic Overview of the Etiopathogenesis of the Four Main Types of Shock



Hypovolemic Shock

Hypovolemic shock arises from a reduction in the effective circulating volume. The most common cause is hemorrhage (e.g., trauma, gastrointestinal bleeding, ruptured ectopic pregnancy), commonly referred to as hemorrhagic shock. Non-hemorrhagic hypovolemia can occur in settings of severe dehydration (e.g., prolonged vomiting or diarrhea), extensive burns (plasma loss), or diabetic ketoacidosis. The main pathophysiological mechanism is diminished venous return (reduced preload), resulting in a lower stroke volume and subsequently reduced cardiac output (Kislitsina et al., 2018). As compensation, tachycardia and peripheral vasoconstriction ensue, causing the skin to be cool and pale with delayed capillary refill. Central venous pressure (CVP) is typically low (collapsing jugular veins). Clinical indicators include hypotension, tachycardia, fatigue, thirst, and oliguria. Primary

treatment involves rapid replacement of intravascular volume and controlling any ongoing hemorrhage.

Cardiogenic Shock

Cardiogenic shock is caused by impaired cardiac pump function, most commonly due to acute myocardial infarction. Other etiologies include extensive myocarditis, severe arrhythmias (e.g., ventricular tachycardia), acute valvular insufficiencies, or advanced heart failure. The fundamental problem is reduced myocardial contractility, leading to insufficient cardiac output (Kislitsina et al., 2018b). Marked hypotension typically triggers catecholamine release and compensatory vasoconstriction, which rarely restores adequate organ perfusion. Common clinical features are hypotension, narrowed pulse pressure, tachycardia (though bradycardia can sometimes occur), cold and clammy skin, changes in mental status, and oliguria. Pulmonary edema may develop, manifesting as rales on auscultation and decreased oxygen saturation. Jugular venous distension (JVD) is often increased, reflecting elevated right-sided filling pressures. If cardiogenic shock is due to a mechanical factor such as pericardial tamponade (clinical presentation often includes Beck's triad: hypotension, JVD, and muffled heart sounds), immediate relief of the tamponade is crucial (Kislitsina et al., 2018c). In cardiogenic shock resulting from acute myocardial infarction, rapid coronary revascularization (e.g., primary percutaneous coronary intervention) should be performed. Management generally includes cautious fluid administration, inotropic support, and, when required, mechanical support (e.g., intra-aortic balloon pump, ECMO) to augment cardiac output.

Distributive Shock

Distributive shock involves inadequate effective circulating volume due to vasodilation and/or fluid shifts outside the vascular compartment. Systemic vascular resistance is low, and cardiac

output is often high or in the upper-normal range early in the course, leading to a “warm shock” appearance (Kislitsina et al., 2018d). Septic shock is the most prevalent distributive subtype and represents the most common cause of shock overall (Kislitsina et al., 2018e). Other important subtypes include anaphylactic shock and neurogenic shock.

Septic Shock

Septic shock results from a dysregulated host response to severe infection, leading to circulatory and metabolic abnormalities. Sepsis itself is defined as life-threatening organ dysfunction secondary to infection, often identified by a Sequential Organ Failure Assessment (SOFA) score ≥2 in the presence of infection (Guarino et al., 2023). Septic shock is the most advanced stage of sepsis, characterized by persistent hypotension requiring vasopressor support despite adequate fluid resuscitation, along with a blood lactate level >2 mmol/L (Guarino et al., 2023b). The pathophysiology involves excessive release of pro- and anti-inflammatory mediators, widespread vasodilation, capillary leak, and maldistribution of blood flow in the microcirculation (Kislitsina et al., 2018f). Early in septic shock, patients may present with warm extremities due to vasodilation and high cardiac output, but as the condition progresses, myocardial depression and vasoplegia can lead to a “cold” shock phase. Clinically, patients may exhibit fever or hypothermia, tachycardia, tachypnea, hypotension, altered mental status, skin changes (warm or cool), a widened pulse pressure, and signs of progressive organ dysfunction. Immediate initiation of broad-spectrum antibiotics and rapid source control (e.g., drainage of an abscess) are essential.

Anaphylactic Shock

Anaphylactic shock is a subtype of distributive shock triggered by a severe allergic reaction (anaphylaxis). Common

inciting agents include medications, foods, insect stings, and latex, among others. In IgE-mediated anaphylaxis, histamine and other mediators released from mast cells and basophils cause pronounced vasodilation, increased vascular permeability, and bronchospasm. Symptoms usually appear within minutes of exposure and include rash (urticaria), angioedema (swelling of the face, lips, tongue), stridor, wheezing, respiratory distress, tachycardia, and hypotension. The skin often feels warm and appears flushed initially but may cool if perfusion worsens. The first-line treatment is intramuscular epinephrine (adrenaline) at a 1:1000 concentration, typically 0.3–0.5 mg in adults, administered without delay (Administrator, n.d.-b). Additional management includes high-flow oxygen, aggressive fluid resuscitation (with crystalloids), and adjunctive therapies such as antihistamines, corticosteroids, and bronchodilators as needed. Refractory hypotension may require intravenous epinephrine infusion and vasopressor support.

Neurogenic Shock

Neurogenic shock arises when spinal cord injury—particularly above the upper thoracic segments—results in a loss of sympathetic tone (often associated with cervical spinal cord injury). Disruption of sympathetic outflow causes vasodilation and is uniquely associated with bradycardia, making it the only shock type commonly presenting with a slow heart rate ("Neurogenic Shock," 2025). Clinically, hypotension with a normal or low heart rate is observed due to unopposed vagal activity; the skin may appear warm and pink initially but can become cool and clammy as perfusion deteriorates. Additional findings may include motor and sensory deficits and loss of reflexes (spinal shock). Neurogenic shock typically manifests in the acute phase following spinal trauma and can exacerbate secondary spinal cord injury if not properly managed. Treatment does not involve the Trendelenburg position; rather, it includes adequate intravenous fluid administration and vasopressor

therapy (e.g., norepinephrine as a first-line agent) to maintain blood pressure ("Neurogenic Shock," 2025b). Atropine may be used for significant bradycardia. Immobilization of the cervical spine (with a neck collar) is crucial to prevent further injury.

Obstructive Shock

Obstructive shock results from a mechanical impediment to venous return to the heart or to blood flow in large vessels, despite an initially normal cardiac pump function. Major causes include tension pneumothorax, cardiac tamponade, massive pulmonary embolism, and less commonly aortic dissection. These conditions either limit filling of the ventricles or block blood flow in the arterial system, ultimately reducing effective cardiac output. In tension pneumothorax, accumulating intrapleural pressure in one hemithorax pushes the mediastinum to the opposite side, compressing the venae cavae and impeding venous return. Clinically, diminished breath sounds on the affected side, hyperresonance on percussion, tachycardia, hypotension, pronounced jugular venous distension, and contralateral tracheal deviation may be seen. Cardiac tamponade involves fluid accumulation in the pericardium that restricts diastolic filling of the heart; Beck's triad (hypotension, JVD, muffled heart sounds) and pulsus paradoxus are characteristic findings. In massive pulmonary embolism, a large pulmonary artery branch is occluded by a thrombus, causing acute right ventricular overload and failure; the patient typically presents with hypotension, severe dyspnea, tachycardia, increased JVD, and ECG signs of right heart strain. Treatment of obstructive shock centers on rapidly removing the underlying mechanical impediment. For suspected tension pneumothorax, immediate needle decompression and subsequent chest tube placement are mandatory; for tamponade, emergent pericardiocentesis is required; and in massive embolism, thrombolytic therapy or surgical/catheter embolectomy should be

considered. Supportive care includes supplemental oxygen, intravenous fluids, and, when indicated, vasopressor support while the primary cause is addressed.

Clinical Signs and Diagnostic Criteria

Although the clinical presentation of shock can vary depending on the underlying etiology, the hallmark signs typically include hypotension and evidence of compromised organ perfusion. In adults, a systolic blood pressure (SBP) lower than 90 mmHg or a mean arterial pressure (MAP) under 65 mmHg often suggests shock; however, relative hypotension may occur at higher blood pressures in patients with a history of hypertension (Pannu, 2023). Tachycardia (heart rate >100 bpm) is frequently one of the earliest signs, due to compensatory acceleration of the pulse aimed at maintaining cardiac output. Nevertheless, it is important to remember that bradycardia may occur in certain situations, such as neurogenic shock or in patients on beta-blockers. Tachypnea is also common and can serve as a compensatory mechanism for metabolic acidosis. In advanced stages, paradoxical slowing or shallowing of respirations may occur if respiratory failure develops.

Skin findings are critical indicators of tissue perfusion. In hypovolemic and cardiogenic shock, peripheral vasoconstriction leads to cold, pale, and clammy skin with delayed capillary refill (>2–3 seconds). In early-phase septic shock, however, the skin may appear warm or even “hot and flushed” due to high cardiac output and vasodilation; yet as septic shock progresses and perfusion worsens, the skin reverts to a cold, mottled appearance. Mottling (a marbled pattern) is often seen around the knees and is indicative of severe perfusion deficits. Cyanosis (bluish discoloration of the lips and extremities) suggests advanced impairment of oxygenation.

Central nervous system manifestations are also common. Reduced cerebral perfusion may initially present as restlessness,

agitation, or confusion; deeper shock states lead to decreased consciousness and eventual coma. In older adults, changes in mental status may be the first sign of shock.

Renal hypoperfusion manifests as decreased urine output. Oliguria in adults is defined as urine output <0.5 mL/kg/hour and is indicative of shock. If persistent, acute kidney injury may ensue, highlighting the importance of monitoring urine output.

Pulse pressure (the difference between systolic and diastolic pressures) tends to narrow in hypovolemic and cardiogenic shock (e.g., 90/80 mmHg). In septic shock, vasodilation can produce a widened pulse pressure (e.g., 120/50 mmHg). The character of the pulse also differs: in hypovolemic or cardiogenic shock, it is typically weak and rapid, whereas in early septic shock it may be "bounding," signifying a strong, quick upstroke.

Jugular venous distension (JVD) assessment can help distinguish underlying causes of shock. In hypovolemic shock, the jugular veins appear collapsed; in cardiogenic shock and cardiac tamponade, JVD is usually elevated; in distributive shock, JVD is generally normal or low (unless there are additional contributing factors).

Laboratory data provide supportive evidence for the diagnosis. A serum lactate >2 mmol/L reflects global hypoperfusion (Pannu, 2023b). Elevated lactate levels and metabolic acidosis are indicative of cellular hypoxia and anaerobic metabolism. Arterial blood gas (ABG) analysis often shows metabolic acidosis (decreased bicarbonate, base deficit <−4). The complete blood count can reveal low hematocrit (suggestive of blood loss) or hemoconcentration (indicative of severe fluid loss). Leukocytosis or leukopenia, along with elevated C-reactive protein (CRP) or procalcitonin, may point toward sepsis. Abnormalities in renal function tests or elevated liver enzymes suggest organ injury secondary to shock. Coagulation

parameters (PT, aPTT, fibrinogen, D-dimer) warrant monitoring, particularly in septic shock where disseminated intravascular coagulation (DIC) may develop. Cardiac biomarkers (troponin, BNP) are pertinent in cardiogenic shock (e.g., elevated troponin in acute myocardial infarction), though troponin may also rise secondary to myocardial injury in septic shock. Additional targeted tests (e.g., amylase/lipase for suspected pancreatitis, cortisol for adrenal crisis, toxicology screening) may guide further diagnosis.

Although no universal "criteria set" definitively confirms shock, clinical findings combined with low blood pressure can be supplemented by the shock index (heart rate ÷ systolic blood pressure). Normally around 0.5–0.7, a shock index >1 supports the diagnosis of significant hypovolemia/shock. For instance, a heart rate of 120 bpm with an SBP of 80 mmHg yields a shock index of 1.5, suggestive of severe shock. However, factors such as age, pregnancy, or beta-blocker use can alter this index. In trauma, a shock index >1 indicates a high likelihood of massive hemorrhage and necessitates rapid intervention.

According to the Sepsis-3 definition, septic shock is diagnosed when a patient, despite adequate fluid resuscitation, requires vasopressor support to maintain a MAP ≥65 mmHg and has a lactate >2 mmol/L (Guarino et al., 2023c). These criteria imply a severe state with mortality rates exceeding 40%. Anaphylaxis is diagnosed clinically, typically by recognizing sudden onset (within minutes to hours after exposure to an allergen) of hypotension (<90 mmHg or >30% drop from baseline) or respiratory compromise coupled with skin or mucosal findings. Even in the absence of cutaneous manifestations, profound hypotension or bronchospasm may be indicative of anaphylaxis.

In summary, the diagnosis of shock is established through the detection of hypotension and clinical or laboratory evidence of tissue

hypoperfusion. The particular combination of findings points to the specific etiology of shock. Prompt recognition and initiation of treatment are critical to reducing mortality.

Initial Evaluation and Management Algorithm in the Emergency Department

In the emergency department, managing a patient with suspected shock requires a simultaneous approach that encompasses both assessment and treatment steps. Early recognition and immediate initiation of supportive therapy are the most critical factors determining a patient's survival (Pannu, 2023c). In general, the approach can be summarized in four fundamental steps:

1. Rapidly Identify Shock

2. Determine the Shock Type (Differential Diagnosis)

3. Begin Concurrent Resuscitation (Airway, Breathing, Circulation – the "ABC"s)

4. Implement Specific Therapies Targeting the Underlying Cause (Pannu, 2023d)

These steps should proceed in parallel and without interruption.

Primary Survey (Initial Assessment)

A rapid "first look" (primary survey) evaluates the patient's general status, level of consciousness, and vital signs. If the patient is found to be hypotensive, with altered mental status and poor peripheral perfusion, a diagnosis of shock is presumed, and the "ABC" approach is initiated:

1. A (Airway)

• Assess airway patency and security. If the patient has a compromised airway or a condition threatening airway patency (e.g.,

significant secretions, airway obstruction in trauma), secure the airway immediately.

• Suction any obstructing secretions, and consider using an oral or nasal airway device.

• If the airway cannot be reliably maintained, early intubation should be considered. Patients in shock are at high risk of aspiration due to altered consciousness and may tire quickly if they are breathing rapidly to compensate for lactic acidosis.

• When preparing for intubation, be aware that induction and sedation agents can exacerbate hypotension; using a hemodynamically stable agent (e.g., ketamine) is often preferred.

• In anaphylactic shock with angioedema and airway edema, do not delay intubation, as airway compromise can progress rapidly.

2. B (Breathing)

• Patients in shock have an increased demand for oxygen, and tissue oxygenation is often insufficient. Administer oxygen support to maintain an SpO_2 of approximately 94–96% (Pannu, 2023e). Use nasal cannula, a simple face mask, high-flow nasal oxygen, or noninvasive ventilation, depending on severity.

• Carefully assess the chest for symmetric rise and fall, breath sounds, and percussion:

• Absent breath sounds on one side may indicate tension pneumothorax and necessitates urgent decompression.

• Bilateral rales could suggest cardiogenic pulmonary edema or pneumonia.

• Wheezing raises suspicion for anaphylaxis or an asthma exacerbation.

• If the patient is hypoventilating or cyanotic, evaluate the need for intubation and mechanical ventilation. In shock states, patients often exhibit rapid, deep respirations to compensate for metabolic acidosis; if they can no longer sustain this effort or if oxygenation is severely compromised, intubation is indicated.

• Mechanical ventilation can decrease the work of breathing and redirect cardiac output to vital organs. However, remember that positive pressure ventilation may reduce venous return, and sedative drugs can worsen hypotension.

3. C (Circulation)

• Supporting circulation is critical for the initial management of shock. Establish intravenous (IV) access promptly: two large-bore (14–16G) IV catheters are preferably inserted into the antecubital veins. If peripheral venous access is not feasible, consider intraosseous access (particularly in pediatric patients via the proximal tibia).

• Draw blood samples immediately for lab tests (complete blood count, chemistry panel, lactate, blood gas analysis, coagulation profile, blood type and crossmatch, and if indicated, toxicology). In suspected septic shock, obtain cultures (if possible, before antibiotics) at the same time.

• Begin IV fluid resuscitation without delay. In most shock states (with the exception of certain cardiogenic cases), a rapid infusion of crystalloid solution is usually life-saving and typically poses minimal risk (“Approach to Shock,” n.d.-b). Even if the specific shock type is not yet definitively identified, start with a bolus of 500–1000 mL of an isotonic crystalloid to assess hemodynamic response.

• If the patient is truly hypovolemic, there will be a marked improvement in blood pressure and perfusion.

• If there is minimal or no response, or if another shock subtype is suspected, consider initiating vasopressor/inotropic support (detailed in the treatment section).

• Repeatedly evaluate pulse quality, heart rate, capillary refill, and skin temperature. Conduct a thorough physical exam looking for potential internal bleeding (e.g., abdominal distension, subcutaneous bruising), signs of aortic dissection (differential blood pressure between arms), cardiac tamponade (jugular venous distension, muffled heart sounds), or congestive heart failure (bilateral rales, peripheral edema).

Simultaneously, gather a concise and focused history—often from relatives, bystanders, or existing medical records in urgent settings—to identify chronic diseases (e.g., cardiac disease, diabetes, renal failure) and to clarify how and when symptoms began (sudden vs. gradual onset), trauma history, fluid intake/output in the past 24 hours, recent fever or infection, chest pain, possible allergen exposure, and medication use (especially beta-blockers or anticoagulants). This information guides the differential diagnosis:

• **Chest pain + ST elevations:** Highly suggestive of cardiogenic shock (acute myocardial infarction).

• High fever, leukocytosis + suspicion of urosepsis: Points toward septic shock.

• **History of bee sting + angioedema:** Suggestive of anaphylactic shock.

4. E (Exposure)

• Fully expose the patient (while maintaining spinal immobilization if trauma is suspected) to identify concealed bleeding sites (e.g., retroperitoneal hematoma, femur fractures), skin rashes (e.g., purpura fulminans, urticaria), abnormal temperature of

the extremities, or possible infection sites (surgical wounds, catheter entry points, soft tissue infections).

• Measure body temperature. Fever may indicate sepsis, whereas hypothermia can signify severe sepsis or environmental factors.

All primary assessment and initial stabilization interventions should ideally be completed within minutes. Subsequently, proceed with further evaluations targeting the specific shock subtype. At this point, additional diagnostic tools in the emergency department come into play:

Adjunct Diagnostic Tools

Bedside Ultrasound (POCUS)

• Point-of-care ultrasound has become an invaluable tool in evaluating shock. The RUSH protocol (Rapid Ultrasound in Shock) systematically examines the heart, major vessels, abdomen, and lungs to identify the potential cause of shock.

• Cardiac ultrasound may reveal pericardial tamponade, reduced myocardial contractility (low ejection fraction), right ventricular dilation (suggesting a massive pulmonary embolism), or global signs of hypovolemia.

• Abdominal ultrasound (FAST exam) can detect free fluid in the peritoneum (indicating intra-abdominal hemorrhage).

• Lung ultrasound can differentiate pneumothorax (absence of the normal pleural sliding), hemothorax, or pulmonary edema.

• Assessment of inferior vena cava (IVC) diameter and its variability with respiration can offer insight into fluid responsiveness but should not be used as the sole decision-making criterion (Pannu, 2023f).

Monitoring

• All shock patients require close monitoring. Noninvasive blood pressure (NIBP) measurements should be taken frequently or continuously, and the cardiac rhythm should be observed via continuous ECG monitoring.

• Pulse oximetry tracks oxygen saturation; if pulses are difficult to palpate, Doppler examination of peripheral pulses may be performed.

• In advanced management, arterial cannulation (e.g., radial artery) may be necessary for invasive blood pressure monitoring, given that NIBP can be unreliable in shock (Pannu, 2023g). An arterial line also facilitates repeated arterial blood gas and lactate measurements.

• Placement of a central venous catheter can be considered for administering large fluid volumes and vasopressors, and for monitoring central venous pressure (CVP) and central venous oxygen saturation ($ScvO_2$) (Pannu, 2023h). While routine $ScvO_2$ monitoring is no longer mandatory, classic sepsis management protocols have aimed for $ScvO_2$ >70%.

Avoiding Delays in Critical Therapies

• In life-threatening conditions, empiric therapy should begin even before diagnostic confirmation is complete.

• For instance, if septic shock is strongly suspected, broad-spectrum antibiotics should be started within the first hour—ideally after obtaining cultures, but treatment should not be delayed while awaiting results.

• In anaphylaxis, epinephrine administration is initiated immediately upon diagnosis, without waiting for laboratory confirmation.

• In massive hemorrhage, preparation for blood transfusion should be initiated immediately.

Summary of the Emergency Management Algorithm

1. Rapidly Confirm the Presence of Shock

2. Simultaneously Initiate ABC Stabilization

3. Establish Large-Bore IV Access and Obtain Basic Labs

4. Start Prompt Fluid Resuscitation

5. Concurrently Gather Patient History, Conduct Physical Exam, and Perform POCUS to Determine the Underlying Cause

6. Begin Definitive or Etiology-Specific Treatment (surgical intervention, medications, etc.) Without Delay

Throughout this process, the patient should be continuously reassessed and therapy modified based on response. If specialized consultation (cardiology, general surgery, thoracic surgery, etc.) is required, it should be requested at an early stage—for example, cardiovascular surgery in suspected aortic dissection, general surgery for a septic shock case involving an abscess, or cardiology for an acute myocardial infarction.

A structured and parallel approach ensures that all critical interventions are provided within the first 30 minutes. One mnemonic in the literature, "MINUTES," outlines seven steps to be completed in this critical time frame (Hasanin et al., 2024):

• M – Maintain Airway, Breathing, and Circulation (the ABCs).

• IN – Start Infusions (fluids and/or vasopressors if needed).

• IN – Investigate (obtain essential labs urgently).

• U – Use Ultrasound (to differentiate shock types).

• T – Treat the underlying Etiology.

• E – Evaluate the need for advanced interventions to restore organ perfusion.

• S – Stabilize the patient with all these measures in place.

This acronym helps ensure no essential step is overlooked and that care proceeds rapidly in the emergency department setting.

Laboratory and Imaging Studies

The evaluation of a patient in shock relies heavily on laboratory tests and imaging studies to confirm the diagnosis, identify the underlying cause, and detect any organ damage.

Laboratory Studies

Upon suspecting shock in the emergency department, a broad panel of blood tests should be performed. Arterial blood gas (ABG) analysis and serum lactate levels are typically among the first tests ordered; an elevated lactate (>2 mmol/L) indicates tissue hypoperfusion, and higher values carry a poorer prognosis (Pannu, 2023ı). Serial lactate measurements also help assess the effectiveness of resuscitation—if lactate levels decrease over time ("lactate clearance"), it suggests improved tissue perfusion.

•**Complete Blood Count (CBC):** Low hemoglobin and hematocrit values raise the possibility of hemorrhage, whereas elevated hemoglobin may signify hemoconcentration due to hypovolemia. Leukocyte count and differential are also examined; leukocytosis with a left shift is commonly seen in sepsis, whereas neutropenia may point to severe sepsis.

• **Infection Markers:** C-reactive protein (CRP) and procalcitonin are useful indicators of systemic infection in suspected sepsis; significantly elevated levels suggest an infectious etiology.

• **Serum Chemistry Panel:** Elevated blood urea nitrogen (BUN) and creatinine can indicate prerenal azotemia or acute tubular necrosis secondary to shock. Elevated liver enzymes, often coupled with high lactate, may reflect "shock liver" (ischemic hepatitis). Electrolyte imbalances (especially potassium and calcium) can influence management decisions (e.g., severe hyperkalemia urgently affects cardiac function).

• **Coagulation Studies:** Prothrombin time/international normalized ratio (PT/INR), activated partial thromboplastin time (aPTT), fibrinogen, and D-dimer are crucial for detecting disseminated intravascular coagulation (DIC), especially in septic shock. Shock may lead to consumption coagulopathy or, in massive trauma, dilutional coagulopathy, requiring prompt identification.

• **Cardiac Biomarkers:** Elevated troponin T/I confirms acute myocardial infarction as the cause of cardiogenic shock; however, troponin can also rise modestly in septic shock due to myocardial depression. B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) may support a diagnosis of heart failure-related shock.

• **Specialized Testing:** Additional tests are guided by clinical suspicion. For example, in refractory shock possibly related to adrenal insufficiency, serum cortisol should be measured. In anaphylaxis, serum tryptase may be evaluated. In suspected carbon monoxide poisoning, COHb and methemoglobin levels are relevant. For possible acute pancreatitis, amylase and lipase are warranted.

• **Other Considerations:** In all female patients, a pregnancy test (beta-hCG) is essential. A positive result can guide management in cases of suspected ectopic pregnancy or inform decisions about Rh incompatibility in trauma.

Imaging Studies

Imaging studies play a critical role in rapid diagnosis and in clarifying the etiology of shock:

• **Chest X-Ray (CXR):** A readily available study in the emergency department, chest radiographs can reveal findings such as pulmonary edema (cardiogenic shock), unilateral loss of lung volume or hyperlucency (pneumothorax), mediastinal widening (aortic dissection), or pneumonia (potential sepsis source).

• **Bedside Ultrasound:** As previously described, point-of-care ultrasound (POCUS) provides versatile, immediate information. The **Extended Focused Assessment with Sonography in Trauma (E-FAST)** evaluates both the thorax and abdomen for trauma-related injuries, detects pericardial effusions, examines the abdominal aorta for aneurysms, and identifies signs of intra-abdominal bleeding. Ultrasound is also used to guide central venous catheter placement and to detect fluid collections (pleural effusions, ascites) if therapeutic drainage (thoracentesis, paracentesis) is needed.

• **Computed Tomography (CT):** CT scans are invaluable for diagnosing shock etiologies such as aortic dissection (contrast-enhanced CT of the chest), pulmonary embolism (CT pulmonary angiography), and abdominal hemorrhage (contrast-enhanced abdominal CT). However, CT typically requires a stable hemodynamic status; critically unstable patients may need to be stabilized first, and transport to the CT scanner must be carefully managed (e.g., monitoring equipment, portable ventilator).

• **Magnetic Resonance Imaging (MRI):** Owing to its lengthy acquisition times, MRI is rarely used in acute shock management except in highly specific circumstances.

Additional Diagnostic Methods

• **Echocardiography:** In collaboration with cardiology, transthoracic or transesophageal echocardiography can help diagnose specific cardiac pathologies (e.g., papillary muscle rupture, valvular involvement in aortic dissection).

• **Pulmonary Artery (Swan-Ganz) Catheter:** This advanced monitoring tool may be considered in complex shock states (e.g., combined septic and cardiogenic shock) to differentiate hemodynamic parameters more precisely, though it is rarely a first-line option in the acute setting.

By integrating laboratory findings and imaging results into the clinical context, clinicians can more rapidly confirm the etiology of shock, gauge disease severity, and identify associated organ dysfunction. Prompt and accurate diagnostic workup is essential for guiding targeted therapies and improving patient outcomes.

Rapid Diagnostic Scoring Systems

In the emergency department, several quick scoring systems have been developed to aid clinicians in detecting potential shock or sepsis and predicting a patient's risk of clinical deterioration. Two commonly used tools are the **qSOFA** and **NEWS2** scores:

1. qSOFA (Quick Sequential Organ Failure Assessment)

Introduced with the Sepsis-3 definitions, qSOFA focuses on identifying organ dysfunction using three criteria:

1. Respiratory rate ≥22 breaths/min

2. Altered mental status (GCS <15)

3. Systolic blood pressure ≤100 mmHg

The presence of two or more of these criteria suggests a higher probability of sepsis and a greater risk of poor outcome

(Guarino et al., 2023d). qSOFA is intended mainly for rapid assessment outside the hospital or in the emergency department triage area. However, it has limited sensitivity; consequently, the 2021 Sepsis Guidelines recommend not using qSOFA alone as a screening tool, preferring instead NEWS2 or the classic SIRS criteria (Guarino et al., 2023e). A positive qSOFA should prompt a thorough evaluation and possibly an early warning team or intensive care consult.

2. NEWS2 (National Early Warning Score 2)

Widely adopted as a national standard in the United Kingdom, NEWS2 is based on six physiological parameters: respiratory rate, oxygen saturation, systolic blood pressure, heart rate, level of consciousness (AVPU scale), and body temperature. Each parameter is assigned a point value, which is summed to yield the NEWS score. Although initially designed for general in-hospital patient deterioration, NEWS2 has proven more sensitive than qSOFA for detecting sepsis, leading recent guidelines to encourage its use in emergency departments (Guarino et al., 2023f). A score of **5 or higher** is typically regarded as an alarm threshold, indicating a high risk of clinical deterioration. In a patient with suspected infection and a NEWS2 score ≥5, close monitoring and prompt treatment for possible sepsis are warranted.

3. SIRS (Systemic Inflammatory Response Syndrome) Criteria

Although sepsis is no longer defined primarily by SIRS, the presence of **≥2 SIRS criteria** (fever >38°C or <36°C, heart rate >90 bpm, respiratory rate >20 breaths/min or $PaCO_2$ <32 mmHg, leukocyte count >12,000 or <4,000 or >10% band forms) suggests possible sepsis in the context of infection. While SIRS has high sensitivity but low specificity, it can still serve as an early warning sign. Current sepsis definitions focus more on organ dysfunction

markers than on SIRS alone, but the criteria may still be useful in screening.

4. MEWS and Other Scores

Other early warning scores, such as the **Modified Early Warning Score (MEWS)**, function similarly to NEWS by assigning point values to vital signs and mental status. Institutions often use the scoring system that best fits their protocols. Regardless of the chosen tool, these scores are meant to **supplement** rather than replace clinical judgment. A normal score does not exclude the possibility of significant shock or sepsis if clinical suspicion remains high.

In summary, qSOFA is a brief bedside assessment but suffers from lower sensitivity, whereas NEWS2 comprises a more comprehensive set of vital parameters and may be better at identifying early sepsis (Guarino et al., 2023g). These tools provide valuable support, especially for less experienced staff or in high-workload environments, helping prevent critically ill patients from being overlooked. Nonetheless, each patient must undergo a holistic clinical evaluation, and the care team should respond according to the patient's risk level as indicated by the scoring system, ensuring appropriate resources and expertise are provided.

Treatment Approaches

The treatment of shock centers on rapidly achieving hemodynamic stabilization and addressing the underlying cause. Management typically involves a bundle of simultaneous interventions. This section discusses fluid resuscitation, the use of vasoactive agents, source control, antibiotic therapy, and advanced life support measures in sequence.

Rapid Fluid Resuscitation and Fluid Selection

One of the first steps in managing shock is restoring intravascular volume and supporting circulation. In hypotensive, hypoperfused patients, it is critical to begin intravenous (IV) fluid therapy promptly. **Crystalloids** are generally the first choice, with **balanced buffered solutions** (e.g., Ringer's lactate, Plasma-Lyte) being recommended over normal saline (0.9% NaCl) (Guarino et al., 2023h). Studies suggest that balanced crystalloids, which have lower chloride content, may reduce the risk of acidosis and renal injury. The 2021 Surviving Sepsis Campaign (SSC) guidelines also provide a weak recommendation for using balanced solutions (e.g., Ringer's lactate) as the initial fluid of choice (Guarino et al., 2023ı).

Fluid volumes should be titrated according to clinical response. Traditionally, in septic shock, guidelines advised administering at least **30 mL/kg** of crystalloids within the first three hours (Guarino et al., 2023i)—for a 70 kg patient, approximately 2 liters. While the strength of this recommendation was downgraded in 2021 (from strong to weak), it remains in current guidelines (Guarino et al., 2023j). Recent trends emphasize **individualized fluid therapy** rather than a one-size-fits-all approach (Guarino et al., 2023k). Clinicians should assess both the patient's **fluid responsiveness** (e.g., via passive leg-raising tests, inferior vena cava variability) and **fluid tolerance** (e.g., signs of cardiac overload) before administering additional boluses (Guarino et al., 2023l). In practice, many centers administer rapid boluses of 250–500 mL and then re-evaluate clinical parameters (heart rate, blood pressure, capillary refill, etc.) before continuing (Guarino et al., 2023m). If the patient's tachycardia improves, blood pressure rises, and peripheral perfusion recovers, fluid administration can continue; otherwise, clinicians should avoid overhydration and move on to vasopressor support.

Amount of Fluid by Shock Type

• **Hypovolemic Shock:** Non-hemorrhagic hypovolemia often necessitates large volumes of crystalloids. After 1–2 liters are rapidly infused, the patient is reassessed. In **hemorrhagic** hypovolemia, especially with uncontrolled bleeding, the priority is early blood product replacement rather than massive crystalloid infusion, and a strategy of **permissive hypotension** is sometimes employed to avoid exacerbating bleeding.

• **Cardiogenic Shock:** Fluid is administered very cautiously and in small boluses (e.g., 250 mL) only if filling pressures are low, because excessive fluid can worsen pulmonary edema.

• **Distributive Shock (Septic, Neurogenic, Anaphylactic):** Significant vasodilation typically creates a substantial fluid deficit; patients may respond well to rapid fluid boluses. In septic shock, after the initial 30 mL/kg in the first hour, further volumes are guided by hemodynamic monitoring and clinical response. Excessive fluid administration may cause glycocalyx injury and edema, leading to worse outcomes (Guarino et al., 2023n).

Colloid solutions (e.g., albumin) are usually not first-line but may be added if large volumes of crystalloid have already been given without achieving stabilization. Albumin remains in the intravascular compartment longer than crystalloids. Sepsis guidelines suggest that, in patients who have received more than 4–6 liters of crystalloid, adding albumin may help reach mean arterial pressure (MAP) targets with a lower total fluid volume (weak recommendation) (Pannu, 2023i). However, randomized trials have shown no significant mortality benefit with albumin (Guarino et al., 2023o). **Synthetic colloids** (hydroxyethyl starch, dextran, gelatin) are no longer recommended due to the risk of renal failure and coagulopathy, and thus have largely been removed from shock treatment protocols.

Targets for Fluid Resuscitation

Clinically, the aims of adequate fluid therapy include:

• Normalizing heart rate and blood pressure

• Improving peripheral perfusion indicators (warmer extremities, normalizing capillary refill)

• Restoring urine output to >0.5 mL/kg/hr

Where available, **invasive monitoring** can guide therapy by:

• Maintaining a central venous pressure (CVP) of approximately 8–12 mmHg (a classic but not universally applied target in septic shock)

• Raising central venous oxygen saturation ($ScvO_2$) above 70%

• Reducing lactate levels

• Keeping MAP ≥65 mmHg

It is important to note that no single parameter guarantees optimal perfusion, so these endpoints are best interpreted in combination. Normal lactate does not exclude shock, and normal $ScvO_2$ does not always confirm adequate tissue perfusion.

In summary, rapid yet judicious fluid loading constitutes the first step in shock management. Balanced crystalloids in sufficient volumes remain the preferred initial strategy, with continuous re-evaluation to avoid fluid overload. When patients fail to respond to fluid resuscitation, or in situations involving severe vasodilation or underlying cardiac dysfunction, **early vasopressor support** should be initiated without delay.

Vasoactive Medications and Their Indications

When hypotension and hypoperfusion persist despite adequate fluid resuscitation, vasoactive agents become necessary. These drugs fall into two major categories: **vasopressors**, which increase blood pressure by inducing vasoconstriction, and **inotropes**, which boost cardiac output by enhancing myocardial contractility. In practice, many vasoactive agents possess both effects to some degree, but their predominant action guides clinical selection.

The general goal of vasopressor therapy is to maintain a **mean arterial pressure (MAP) ≥65 mmHg** to ensure adequate perfusion of vital organs (Guarino et al., 2023ö). While 65 mmHg is a common initial target, the optimal level may be higher (70–75 mmHg) in patients with coronary artery disease or older adults, whereas it may remain at 65 mmHg for a younger patient. Whenever possible, vasopressors should be administered through a **central venous catheter**. If given peripherally, they must be diluted and closely monitored to avoid extravasation, with transition to central access as soon as feasible.

Key Vasoactive Agents

1. Norepinephrine (NE)

• **Mechanism:** Primarily an α_1-adrenergic agonist, producing significant peripheral vasoconstriction; also exerts mild β_1-adrenergic effects to support cardiac output (Pannu, 2023k).

• Clinical Use:

• **First-line vasopressor** in most shock types, especially septic shock (strong recommendation) (Pannu, 2023l).

• Typically dosed at **0.05–0.5 µg/kg/min** (5–50 µg/min in many adults).

• If targeted MAP is not achieved with moderate to high doses, adding a second agent is preferred over pushing norepinephrine to extremely high rates.

• Combination Therapy:

• **Vasopressin** is the first adjunct recommended once norepinephrine doses approach 0.2–0.3 μg/kg/min in septic shock. Vasopressin (0.03 U/min) provides additional vasoconstriction via V1 receptors, reducing norepinephrine requirements (Pannu, 2023m–o). Doses above 0.04 U/min carry an elevated risk of peripheral ischemia.

• **Epinephrine** may be added if norepinephrine plus vasopressin fail to maintain adequate perfusion.

2. Vasopressin

• **Mechanism:** Acts on V1 receptors, causing vasoconstriction and helping to lower norepinephrine requirements.

• **Dose:** Typically 0.03 U/min (fixed) in septic shock.

• **Cautions:** Doses above 0.04 U/min significantly increase the risk of peripheral ischemia, so vasopressin is usually not titrated beyond this point (Pannu, 2023o).

3. Epinephrine (Adrenaline)

• **Mechanism:** A potent β_1- and β_2-adrenergic agonist with α-adrenergic activity at higher doses (Pannu, 2023ö).

• Clinical Use:

• Often a **second-line** agent in septic shock if norepinephrine plus vasopressin prove insufficient.

• Increases cardiac output but can cause tachyarrhythmias, elevated lactate levels, and splanchnic hypoperfusion (Pannu, 2023p).

• **Guideline Recommendations:** If hypoperfusion persists despite norepinephrine in septic shock, guidelines advocate either adding **dobutamin** or switching to epinephrine infusion (Pannu, 2023r).

4. Dobutamine

• **Mechanism:** A β_1-adrenergic agonist that boosts myocardial contractility and cardiac output; it also lowers afterload slightly through β_2-mediated vasodilation.

• Clinical Use:

• Commonly employed in **cardiogenic shock** once blood pressure is somewhat stabilized, in order to raise cardiac output.

• In **septic shock** with myocardial depression—or if tissue perfusion remains inadequate despite an acceptable MAP—dobutamine can be added to norepinephrine (Pannu, 2023s). A typical dose is **5–20 µg/kg/min**.

• **Cautions:** Dobutamine may cause tachycardia and arrhythmias, and in septic shock, it may not always improve tissue perfusion as desired (Pannu, 2023ş). Nonetheless, it is a core therapy to improve cardiac output in cardiogenic shock.

• Combination Therapy:

• In **cardiogenic shock**, **norepinephrine + dobutamine** is frequently used to support both blood pressure (via norepinephrine) and cardiac output (via dobutamine).

• In acute MI-related cardiogenic shock, if dobutamine alone is insufficient, norepinephrine is added (Pannu, 2023t). Guidelines favor norepinephrine over dopamine in cardiogenic shock.

5. Dopamine

• **Past Uses:** Once a common inotrope/vasopressor, it is no longer routinely recommended for shock management (Pannu, 2023u).

• **Mechanism:** Low doses may enhance renal blood flow (though no proven clinical benefit); moderate doses stimulate β receptors to raise cardiac output; high doses stimulate α receptors with vasopressor effects.

• **Drawbacks:** Dopamine has a high arrhythmogenic potential, and evidence links it to poorer outcomes in septic shock (Pannu, 2023ü).

• **Limited Indications:** May be considered in younger patients with prominent bradycardia, but norepinephrine and/or epinephrine are generally preferred (Pannu, 2023v).

6. Other Vasopressors

• **Phenylephrine:** A pure α-adrenergic agonist used occasionally in severe tachyarrhythmias or in neurogenic shock presenting with hypotension and bradycardia. However, it can reduce cardiac output, so it is not a first-line agent for septic shock.

• **Angiotensin II:** A newer option (AT_1 receptor agonist) approved for refractory septic shock, though its widespread use remains limited.

• **Methylene Blue:** An agent that inhibits nitric oxide–mediated vasodilation, reserved as a last resort for severe vasoplegic states (e.g., refractory septic or anaphylactic shock).

Selecting the Right Vasoactive Agent

• Septic Shock & Distributive Shock:

• **Norepinephrine** is the first-line vasopressor (Pannu, 2023y).

• **Vasopressin** and/or **epinephrine** may be added if norepinephrine at moderate to high doses is inadequate; if cardiac output is low, **dobutamine** is considered (Pannu, 2023z).

• Cardiogenic Shock:

• **Norepinephrine** (weak recommendation) is often initiated if hypotension is severe, followed by an inotrope (dobutamine) (Pannu, 2023aa).

• Anaphylactic Shock:

• First-line therapy is **intramuscular epinephrine**. If shock is refractory, an intravenous epinephrine infusion (approximately 0.1 μg/kg/min, titrated) is the principal agent.

• Neurogenic Shock:

• **Norepinephrine** or **phenylephrine** may be used; in the presence of bradycardia, norepinephrine is typically preferred (its β_1 effect can slightly increase heart rate), and atropine can be added if needed.

• **Obstructive Shock** (e.g., massive pulmonary embolism):

• Norepinephrine with or without dobutamine can provide hemodynamic support, but definitive treatment (e.g., thrombolysis, surgical embolectomy) must rapidly address the obstruction.

In using vasopressors and inotropes, the aim is to **individualize dosing** based on the patient's clinical response. Excessive vasoconstriction can compromise peripheral perfusion, so one should not focus solely on arterial pressure; rather, consider mental status, skin perfusion, and urine output to guide "optimal" dosing. Combination regimens are common—for instance, **NE + dobutamine** is frequently employed in both septic and cardiogenic shock to support blood pressure and cardiac output. If shock persists despite dual or even triple vasopressor therapy, the condition is

deemed “refractory shock,” with a high risk of mortality. In such cases, additional interventions—such as **corticosteroids** (particularly in septic shock) or **advanced mechanical support**—may be warranted (discussed further below).

Source Control (Infection and Other Causes)

The success of shock management depends not only on hemodynamic stabilization but also on rapidly addressing and eliminating the underlying cause. While initiating measures to restore circulation and blood pressure, clinicians must simultaneously pursue source control.

Source Control in Septic Shock

Effective source control is a cornerstone of septic shock management (Juneja, 2012). Once the patient is relatively stable, any infectious focus should be promptly addressed:

• **Intra-Abdominal Infection:** For example, in a patient with septic shock secondary to a perforated appendix or an intra-abdominal abscess, urgent surgical intervention or interventional radiology drainage is crucial.

• **Empyema or Fluid Collections:** Tube thoracostomy (chest tube) placement or percutaneous drainage is indicated.

• **Necrotizing Fasciitis:** Early and aggressive surgical debridement is vital.

• **Intravascular Devices or Prostheses:** Infections related to intravenous catheters, prosthetic heart valves, or joint implants may necessitate removal of the device in addition to appropriate antimicrobial therapy (Juneja, 2012b).

• **Biliary Sepsis:** Endoscopic retrograde cholangiopancreatography (ERCP) can be employed to decompress obstructed bile ducts.

The earlier source control is performed, the greater the likelihood of controlling the infection and reversing shock (Juneja, 2012c). Delays can perpetuate bacteremia and toxin release, undermining therapeutic efforts.

Source Control in Other Shock Types

1. Hemorrhagic Shock

Although infection is not the culprit, the concept of "source control" applies to stopping the bleed. For instance, hemorrhages in trauma may require surgical ligation or angioembolization to control vascular injury, while gastrointestinal bleeds often call for endoscopic hemostasis. In pelvic fractures, external fixation and pelvic binding can reduce ongoing bleeding. Interim measures (tourniquets, compression, resuscitative thoracotomy) may be necessary until definitive hemorrhage control is achieved.

2. Cardiogenic Shock

Source control involves addressing the cardiac pathology precipitating shock. In the case of acute myocardial infarction (MI), early coronary angioplasty or thrombolysis is mandatory; if a complication like severe acute mitral regurgitation or ventricular septal rupture arises, urgent surgical evaluation is required. Rapid cardioversion should be considered for life-threatening arrhythmias causing massive pulmonary edema. In such scenarios, "source control" equates to rectifying the root cardiac problem.

3. Obstructive Shock

• Massive Pulmonary Embolism (PE): Thrombolytic therapy, catheter-directed interventions, or surgical embolectomy represent definitive treatment.

• Cardiac Tamponade or Tension Pneumothorax: Pericardiocentesis or needle decompression/chest tube insertion is required to relieve the obstruction.

4. Anaphylactic Shock

Eliminating further exposure to the offending allergen (e.g., discontinuing the offending medication infusion, removing a bee stinger) is a form of source control.

5. Neurogenic Shock

In spinal cord injuries, early stabilization (spinal immobilization) and surgical decompression (e.g., hematoma evacuation) when necessary can limit further damage and prevent additional deterioration.

Collaboration and Timing

Supportive measures alone (e.g., fluids, vasopressors) are insufficient for definitive shock treatment; addressing the trigger is essential. Close coordination with surgical specialties and other relevant disciplines is crucial for planning and executing source control promptly. In septic shock, the general recommendation is to achieve source control within **6–12 hours** whenever possible (for instance, urgent surgery for a perforation or drainage of an abscess). Each hour of delay can significantly increase mortality, underscoring the importance of **early intervention**.

Initiation and Timing of Antibiotic Therapy

A cornerstone of managing septic shock and severe sepsis is the **early administration of appropriate antimicrobial therapy**. Studies demonstrate that each hour of delay adversely affects survival in infection-related shock (Juneja, 2012d). As a result, administering broad-spectrum antibiotics within the first “golden hour” is strongly recommended (Juneja, 2012e).

Practical Considerations for Early Antibiotic Administration

1. Culture Collection

• In patients suspected of having sepsis or septic shock, blood cultures (and other relevant specimens such as urine, sputum, and cerebrospinal fluid) should be obtained immediately, followed by prompt empirical antibiotic therapy.

• Delaying antibiotics to obtain cultures should be avoided; if obtaining cultures is not feasible, antibiotics must still be administered.

• According to the Surviving Sepsis Campaign, **broad-spectrum empirical regimens**—potentially in combination—should be used initially to cover likely pathogens, followed by de-escalation once the causative organism is identified (Juneja, 2012f). For instance, in a patient with altered mental status and possible meningitis, the empirical regimen should cover not only meningitis but also potential pneumonia or urinary tract infection.

2. Selecting Empirical Regimens

• Antibiotic choice depends on the presumed site of infection (respiratory, urinary, intra-abdominal, etc.) and on risk factors for antimicrobial resistance.

• **Community-Acquired Infections**: Regimens target typical community pathogens.

• **Hospital-Acquired Infections**: Broader coverage for resistant organisms is required.

• Examples include:

• **Urinary Sepsis**: IV extended-spectrum cephalosporin (e.g., cefepime) or piperacillin-tazobactam ± an aminoglycoside.

• **Pneumonia-Related Sepsis**: A combination that covers pneumococci and atypical pathogens (e.g., ceftriaxone plus a macrolide or levofloxacin). In patients requiring ICU admission, piperacillin-tazobactam plus a fluoroquinolone may be considered.

• **Intra-Abdominal Sepsis**: A broad-spectrum β-lactam/β-lactamase inhibitor (e.g., piperacillin-tazobactam) or a carbapenem, sometimes with metronidazole for anaerobic coverage.

• **Skin and Soft-Tissue Sepsis (e.g., Necrotizing Fasciitis)**: A regimen such as carbapenem + clindamycin + vancomycin to cover polymicrobial and Gram-positive organisms.

• When the patient is in shock, coverage for **MRSA** and **resistant Gram-negative** organisms is frequently warranted (e.g., adding vancomycin or linezolid, using a carbapenem for high-risk scenarios).

• If fungal infection is a concern (e.g., neutropenia, TPN use, prior abdominal surgery), empirical antifungal therapy may be considered.

3. Dosing and Route of Administration

• Antibiotic dosing should be **high** (including a proper loading dose) because hypoperfusion in shock may alter drug distribution. Initial doses are generally administered in full, even in renal impairment, with subsequent adjustments based on therapeutic drug levels and renal function (Juneja, 2012g).

• In septic patients, the volume of distribution may increase, prompting higher dosing; extended or continuous infusions of β-lactams are sometimes recommended.

• **Intravenous administration** is standard in shock settings, due to potential absorption issues with oral medications.

4. Timing Goals

• Ideally, antibiotics should be started within **one hour** of recognizing hypotension or shock (Juneja, 2012h).

• If sepsis is identified in triage, ensure antibiotic therapy begins promptly ("hour zero" from triage), which serves as a quality-of-care benchmark.

5. Duration of Antibiotic Therapy

• Treatment duration typically ranges from 7 to 10 days, but it may extend longer in endocarditis or other complicated infections.

• Definitive treatment length is usually determined in the intensive care setting. In the acute phase, the priority is rapid administration of **the correct drugs at the correct doses**. Failure due to improper coverage (e.g., a narrow-spectrum agent alone) or delays can worsen outcomes (Juneja, 2012ı).

Other Shock States

While antibiotic therapy is primarily relevant for sepsis, **prompt etiology-specific therapy** is equally vital in non-infectious shocks:

• **Adrenal Crisis:** Immediate IV hydrocortisone.

• **Anaphylaxis:** Epinephrine administration.

• **Beta-Blocker Overdose:** Glucagon infusion, among other measures.

In all shock scenarios, timely intervention addressing the root cause is imperative for improving survival.

Advanced Interventions (Mechanical Ventilation, RRT, etc.)

In some patients with shock, basic treatments alone are insufficient to stabilize the clinical picture. These individuals may require advanced life support measures—often referred to as **organ**

support therapies—aimed at preserving organ function and improving survival:

1. Mechanical Ventilation (MV)

Mechanical ventilation is indicated in cases of acute respiratory failure, acute respiratory distress syndrome (ARDS), or compromised consciousness resulting from shock. Endotracheal intubation and connection to a ventilator offload the work of breathing, allowing more of the cardiac output to be directed toward other organs.

• Application in Septic Shock (e.g., ARDS):

• Low tidal volume ventilation (6 mL/kg) with appropriate positive end-expiratory pressure (PEEP) helps improve oxygenation and reduce ventilator-induced lung injury.

• In severe ARDS, advanced strategies like veno-venous extracorporeal membrane oxygenation (VV-ECMO) may be considered in an intensive care setting.

• Application in Cardiogenic Shock:

• If pulmonary edema is present, positive-pressure ventilation helps recruit alveoli and enhance oxygenation.

• PEEP can reduce venous return, easing acute pulmonary edema, although excessive PEEP may lower cardiac output and must be carefully titrated.

• Hemodynamic Considerations:

• Patients in shock may experience a further drop in blood pressure upon induction for intubation; vasopressor infusions should be readily available, and a low-dose catecholamine drip is often started prior to or during intubation.

• Oxygen saturation targets typically range around 94–98% to avoid hyperoxia, which may lead to increased oxidative stress.

2. Renal Replacement Therapy (RRT)

Acute kidney injury (AKI) commonly ensues in shock, particularly if accompanied by oliguria/anuria, fluid overload, or life-threatening electrolyte imbalances (e.g., severe hyperkalemia) and metabolic acidosis. Under these circumstances, **dialysis** or **continuous renal replacement therapy (CRRT)** is often required in the intensive care unit.

• CRRT vs. Intermittent Hemodialysis:

• CRRT is preferred in hypotensive patients because the slow and continuous nature of fluid removal is more hemodynamically stable.

• While emergent dialysis is rarely initiated in the emergency department, acute indications (e.g., potassium >7 mEq/L with ECG changes, severe uremia) warrant urgent coordination with nephrology/ICU teams.

• Experimental filters (e.g., high-flow cytokine hemofiltration) have been investigated in septic shock but are not yet standard practice.

3. Transfusion and Blood Product Replacement

• Massive Transfusion Protocol in Hemorrhagic Shock:

• Balanced replacement of packed red blood cells, fresh frozen plasma, and platelets (often in a 1:1:1 ratio) is implemented to maintain hemoglobin at least in the 7–9 g/dL range and prevent coagulopathy.

• Restrictive Transfusion Strategy in Septic Shock:

• Contrary to older protocols advocating higher targets (e.g., hematocrit of 30%), current guidelines advise a restrictive strategy, typically transfusing only when hemoglobin falls below ~7 g/dL (Juneja, 2012i).

• If disseminated intravascular coagulation (DIC) develops, coagulation factors (plasma) and fibrinogen (cryoprecipitate) or platelets should be given according to standard thresholds (e.g., platelet count <10,000–20,000/μL).

4. Steroid Therapy

In **septic shock**, intravenous hydrocortisone is recommended if hypotension persists despite fluids and vasopressors. Typically, a total daily dose of **200 mg** (either continuous infusion or divided doses) is administered. The 2021 Surviving Sepsis Campaign provides a weak recommendation for adding steroids in patients who do not achieve hemodynamic targets with fluids plus vasopressors alone. The rationale is to shorten the duration of vasopressor dependence.

• Additional Considerations:

• In patients at high risk for adrenal insufficiency (e.g., chronic steroid use, adrenal hemorrhage), stress-dose steroids may be started earlier.

• High-dose steroids in **neurogenic shock** or acute spinal cord injury remain controversial due to increased infection risk, with limited evidence of benefit.

5. Mechanical Circulatory Support

If pharmacological therapy proves insufficient in **cardiogenic shock**, temporary mechanical support devices may be required. These advanced interventions typically necessitate specialized intensive care resources:

• Intra-Aortic Balloon Pump (IABP):

• Reduces afterload on the left ventricle and improves coronary perfusion, historically used for acute MI with cardiogenic shock (though recent data show limited benefits in general). It may still prove useful for mechanical complications such as ventricular septal rupture or acute mitral regurgitation.

• Ventricular Assist Devices (VADs) (Impella, TandemHeart):

• Catheter-based pumps that temporarily augment cardiac output.

• Veno-Arterial ECMO (VA-ECMO):

• Provides partial or total cardiopulmonary support in **refractory cardiogenic shock** or massive pulmonary embolism (sometimes combined with surgical embolectomy).

• Requires specialized centers and experienced teams. Early application of ECMO has shown promise in improving survival in certain severe cardiogenic shock cases.

6. Other Supportive Measures

• **Refractory Acidosis**: Intravenous buffers (e.g., trisodium citrate) may be considered in life-threatening acidemia.

• **Severe Hyperkalemia**: Immediate therapies include calcium gluconate, insulin-glucose infusions, and nebulized salbutamol.

• **Neurogenic Shock with Bradycardia**: Temporary pacemaker placement can be considered if refractory to atropine or vasopressors.

Key Takeaways

After initial stabilization in shock (fluids, vasopressors, and etiological treatment), **organ support therapies** should be rapidly deployed where indicated:

• **Mechanical ventilation** can optimize oxygenation and offload work of breathing.

• **Renal replacement therapy** addresses fluid and electrolyte derangements.

• **Transfusion protocols** aim to correct significant anemia or coagulopathy.

• **Steroids** may attenuate prolonged vasopressor requirements in septic shock.

• **Mechanical circulatory support** offers potential rescue in refractory cardiogenic shock.

These interventions typically proceed in an **intensive care unit** setting. Emergency physicians must anticipate such needs and communicate effectively with specialist teams—ensuring a seamless transition to the appropriate level of care.

Shock in Pregnant Patients

Physiological changes during pregnancy can influence both the recognition and management of shock. Because circulating blood volume increases by 30–50% during pregnancy, a similar degree of blood loss may result in delayed deterioration of vital signs compared to a non-pregnant woman. For instance, once tachycardia and hypotension are evident in a pregnant patient, the actual blood loss may be quite significant. Additionally, baseline maternal heart rate is often about 15–20 bpm higher, and systolic blood pressure may be slightly lower; these normal variations must be interpreted cautiously.

Starting in the second trimester, the enlarging uterus can compress maternal blood vessels. In **supine hypotension syndrome**, lying flat leads to vena cava compression by the uterus, reducing venous return and potentially causing profound hypotension or syncope (Fccm, n.d.). Therefore, shock management in pregnancy typically involves positioning the patient with a **30° left lateral tilt** rather than fully supine (Fccm, n.d.b). This position (or manual leftward uterine displacement if supine transport is unavoidable) also optimizes uteroplacental perfusion (Ms, n.d.).

Although the general causes of shock are similar in pregnant and non-pregnant populations, certain obstetric complications warrant special consideration. In trauma, placental abruption or uterine rupture must be suspected. An abrupt onset of shock in the third trimester may indicate **amniotic fluid embolism** (rare but highly lethal). In the postpartum period, hemorrhage (e.g., uterine atony, retained placental fragments, lacerations) is a leading cause of shock and constitutes an obstetric emergency. Conditions such as **eclampsia** or **HELLP syndrome** can present with features of both hypovolemic and distributive shock due to hemorrhage and systemic inflammation.

When treating pregnant shock patients, **maternal stabilization** takes priority, as improving maternal well-being optimally supports the fetus (Albright, McCartney, Hitti, & UW Medicine, 2018). Standard fluid and blood product resuscitation and vasopressor therapy generally apply; however, fetal considerations may influence medication selection. For example, although ephedrine has historically been used in obstetric practice, **norepinephrine** remains first-line in septic shock, with the understanding that it may reduce uterine blood flow but still offers the best chance of maternal survival. Expanded volume of distribution and increased renal clearance in pregnancy can necessitate **higher medication dosages**. While antibiotic safety

profiles for the fetus should be considered, maternal survival in severe infections (e.g., using carbapenems for septic shock) remains paramount (Albright, McCartney, Hitti, & UW Medicine, 2018b).

Fetal monitoring is also important, particularly in pregnancies beyond the threshold of viability (≥24 weeks). Nevertheless, maternal stabilization must take precedence, and an **urgent cesarean section** is not typically performed unless there are separate obstetric indications or maternal status warrants it. In the rare circumstance of maternal cardiac arrest with pregnancy >20 weeks, **perimortem cesarean** within 4–5 minutes of unsuccessful resuscitation is recommended to potentially improve outcomes for both mother and fetus. Aside from such critical scenarios, pregnant patients in shock require **multidisciplinary management**—involving emergency medicine, obstetrics, surgical teams, and the intensive care unit.

Shock in Pediatric Patients

Children exhibit unique features during shock, mainly because they can often maintain blood pressure until very late in decompensation. Thus, hypotension is a **late** finding. Instead, shock in pediatric patients is frequently inferred from **signs of poor peripheral perfusion and tachycardia**. For example, a child presenting with tachycardia (relative to age norms), cool extremities, capillary refill time >3 seconds, lethargy, or somnolence may already be in compensated shock—even if blood pressure remains within the normal range for age. Once a pediatric patient becomes hypotensive, shock is considered decompensated and urgent intervention is needed to prevent cardiac arrest. Blood pressure thresholds should be interpreted according to age (e.g., hypotension in a child aged 1–10 years is roughly SBP < [70 + 2 × age in years] mmHg).

Differences in pediatric anatomy and physiology also affect treatment. Vascular access can be challenging; **intraosseous**

cannulation should be used promptly if peripheral IV lines are difficult to establish. Children have less respiratory reserve, making early intubation more critical in severe cases. Moreover, **hypoglycemia** can exacerbate shock, necessitating diligent glucose monitoring and possible dextrose administration.

Pediatric septic shock is associated with high mortality. The 2020 Surviving Sepsis Campaign guidelines provide specific recommendations for children (Geroteo, Levy, Gotchac, Brissaud, & Dauger, 2022). In settings with ready access to pediatric intensive care, **40–60 mL/kg** of crystalloid is often administered within the first hour (Geroteo et al., 2022b), generally in **20 mL/kg boluses** with frequent clinical reassessment. If shock persists after 40–60 mL/kg, vasopressors/inotropes are introduced. In resource-limited environments where intensive care is unavailable, guidelines recommend giving fluid boluses **only to hypotensive children** (up to 40 mL/kg total), aligning with data from the FEAST trial, which indicated increased mortality from over-resuscitation in settings lacking advanced support (Geroteo et al., 2022c).

Regarding **vasopressors**, pediatric guidelines now discourage **dopamine** use, favoring **epinephrine or norepinephrine** instead (Miranda & Nadel, 2023). Some suggest choosing **epinephrine** in "cold shock" (low cardiac output, vasoconstricted state) and **norepinephrine** in "warm shock" (vasodilated state), but the distinction can be difficult. In practice, **epinephrine** (0.05–1 μg/kg/min) is often the first-line infusion in children (Miranda & Nadel, 2023b), and it can be initiated through a peripheral line before transitioning to central access.

Children are also prone to other causes of distributive shock, such as **severe dehydration** (e.g., from gastroenteritis) or complicated **diabetic ketoacidosis (DKA)**, in which fluid therapy must be carefully balanced to avoid cerebral edema. In traumatic

shock, pediatric patients can rapidly become hypovolemic due to smaller blood volume and robust compensatory mechanisms; by the time vital signs deteriorate, blood loss may be very large. **Massive transfusion** protocols adapted for children (where ~10 mL/kg blood loss approximates one unit of blood) are becoming more standardized.

Overall, management principles mirror adult protocols—secure the airway, provide oxygen, establish IV access, administer fluid boluses, add vasopressors, intubate if needed, and treat underlying causes (e.g., antibiotics for infection). However, pediatric drug **dosing** is weight-based, and medication preparation errors can be minimized using standardized references or tools like the Broselow tape. Frequent reassessment is essential, monitoring **hourly urine output** (>1 mL/kg/hr is desirable), capillary refill, mental status, and blood glucose levels. Thermoregulation is also critical given children's susceptibility to hypothermia.

Shock in Older Adults

Geriatric patients (≥65 years) experience higher mortality rates and pose unique diagnostic and therapeutic challenges in shock. With diminished physiological reserve in cardiac, renal, and hepatic function, older adults exhibit reduced tolerance for hypoperfusion. Multiple comorbidities and polypharmacy (e.g., beta-blockers, diuretics) can mask or modify classic shock responses.

Clinical Presentation:

Older adults may display a muted tachycardic response (Juneja, 2012j). For example, a patient on beta-blockers might appear with a heart rate of 80 bpm yet still be in severe shock. Fever responses are often blunted; an older patient with significant infection may have only a low-grade fever or no fever at all. Consequently, **altered mental status**—ranging from confusion to

drowsiness—often serves as the primary early indicator of sepsis or shock. Peripheral perfusion findings (e.g., delayed capillary refill) may be confounded by peripheral vascular disease or autonomic dysfunction. Thus, clinicians must employ a broad, integrative evaluation and interpret even slight deviations from baseline as potentially significant.

Frequent Causes of Shock:

• **Sepsis** (commonly from urinary, pulmonary, or intra-abdominal infections) is a leading cause of shock in older adults.

• **Myocardial infarction** (with cardiogenic shock) occurs at higher rates in this group.

• Dehydration and gastrointestinal bleeding are also common.

• Polypharmacy can precipitate shock (e.g., excessive warfarin use causing hemorrhage, or overdose of insulin leading to hypoglycemic shock).

Treatment Adjustments:

• **Fluid Resuscitation**: Must be carefully titrated to avoid precipitating heart failure in patients with stiff left ventricles (Juneja, 2012k). A strategy of administering smaller fluid boluses and frequently reassessing hemodynamics is prudent.

• **Vasopressors**: Older adults often have variable vascular responses due to atherosclerosis. While the standard MAP target typically remains ≥65 mmHg, some previously hypertensive patients may require 75–80 mmHg for adequate organ perfusion. Inotrope-induced arrhythmias (e.g., with dobutamine) can be especially dangerous in an older heart (Juneja, 2012l), necessitating cautious dose titration and vigilant monitoring.

• **Antibiotic Therapy**: Renal function may be underestimated by normal serum creatinine levels in older patients with reduced muscle mass (Juneja, 2012m). Renal dosing adjustments should be considered after initial broad-spectrum coverage is administered without delay for suspected sepsis.

• **Comorbidities**: Organ dysfunction progresses more rapidly in older adults. Coronary artery disease can worsen myocardial ischemia under shock conditions, and chronic obstructive pulmonary disease (COPD) can complicate ventilation. Therefore, a lower threshold for **early intubation** and lung-protective ventilation may be beneficial in severe sepsis.

• **Monitoring**: Older adults are frail and require intensive care follow-up, as mortality rates are significantly higher than in younger patients (Juneja, 2012n). Rigorous, guideline-based management—including early antibiotics, appropriate fluid resuscitation, and vasopressor support—remains crucial. Post-shock management often involves rehabilitation, nutritional support, and prevention of secondary complications such as pressure ulcers.

In all special populations—pregnant women, children, and older adults—the overarching principles of shock management remain: **rapid recognition, prompt resuscitation, and definitive treatment of the underlying cause**. However, tailoring these interventions to meet each group's unique physiological needs and risk factors is essential for optimizing outcomes.

Monitoring and Planning for ICU Transport

Once a patient in shock has been stabilized in the emergency department (ED), **close monitoring** must continue, and transfer to a suitable intensive care unit (ICU) should occur as swiftly as possible. Key points include:

Continuous Monitoring in the ED

1. Vital Signs and Hemodynamics

• Maintain continuous monitoring of blood pressure, heart rate, cardiac rhythm, and oxygen saturation.

• Record urine output hourly via an indwelling urinary catheter and urometer.

• If arterial and central venous lines are in place, track **invasive blood pressure**, **central venous pressure (CVP)**, **$ScvO_2$**, and (if available) **intermittent cardiac output** measurements to follow trends in real time.

• Reassess **lactate levels** regularly (e.g., every 1–2 hours, or at least every 4–6 hours); a declining lactate trend suggests improved perfusion, whereas rising levels are worrisome.

2. Laboratory and Clinical Assessments

• Obtain frequent **blood gas analyses** and lab tests (e.g., glucose, electrolytes) to identify issues such as excessive hyperventilation (leading to respiratory alkalosis) or deepening metabolic acidosis, both of which may necessitate changes in ventilatory or metabolic management.

• Perform systematic **clinical evaluations** (hourly or more often): Has mental status improved? Is skin color/perfusion better? Has capillary refill time shortened? Have the extremities warmed? Is the pulse quality stronger? Is respiratory distress diminishing? Each parameter's improvement or deterioration guides potential adjustments to therapy (e.g., addressing new arrhythmias or fever spikes).

Determining ICU Needs

• **All shock patients** generally require ICU admission for advanced monitoring and management.

• The ED focuses only on **initial stabilization**; patients who are mechanically ventilated, on vasopressors, or in need of close observation/advanced therapies must be allocated promptly to the appropriate ICU (e.g., general or coronary).

• **Early notification** of the ICU team helps them prepare the necessary bed and equipment.

• Extended ED stays risk compromising care quality, as ICU staffing ratios and support systems are typically better suited for critically ill patients. **Safe and timely transfer** should be arranged as soon as feasible.

Pre-Transfer Preparation

In-hospital transfer of a critically ill patient is high risk and requires optimal preparation:

1. Hemodynamic Stabilization

• If the patient's shock state remains highly unstable, it may be prudent to delay transport briefly until vital parameters are more controlled, or ensure that an experienced physician escorts the patient.

2. Airway Security

• If intubation is necessary, it should be performed **before** transfer; transporting a patient with an unprotected airway is dangerous.

3. Sedation and Analgesia

• Provide sedation/analgesia to patients who are in pain or intubated to prevent agitation or ventilator dyssynchrony during transport.

4. Medications and Equipment

• Ensure adequate supplies of oxygen (verify portable O_2 tank levels), a **transport ventilator** or bag-valve mask, and that vasopressor infusions are on battery-powered pumps (with enough battery life checked beforehand).

• Keep an **emergency kit** with intubation equipment (laryngoscope, endotracheal tubes, suction), critical medications (epinephrine, atropine, amiodarone, etc.), and basic resuscitation tools.

• Maintain **continuous monitoring** with a portable device (ECG, pulse oximetry, noninvasive blood pressure). If available, a transport monitor/defibrillator monitor is ideal.

5. Transport Team

• Typically, **one physician** (preferably with ICU or emergency experience) and **one skilled nurse or paramedic** is advised (Whiteley, Gray, McHugh, & B O'Riordan, 2001).

• Transporting a mechanically ventilated patient on multiple infusions with only one staff member is unsafe.

• If the patient is on a ventilator, a **respiratory therapist** may also be required.

Communication and Handover

• **Before transport**, communicate the patient's status, treatments provided, and planned interventions to the ICU physician.

• **Upon arrival**, give a concise and systematic handover—an **SBAR** (Situation, Background, Assessment, Recommendation) format is often helpful. For instance:

"A 78-year-old woman with septic shock, likely urosepsis. She is intubated, on norepinephrine at 0.2 μg/kg/min. We have given 3 liters of Ringer's, obtained cultures, started imipenem and vancomycin. MAP is around 65–70 mmHg, her last-hour urine

output was 20 mL. She has arterial and central venous lines in place, and her lactate decreased from 6 to 4. We plan to continue vasopressor support, possibly add hydrocortisone, and perform a renal ultrasound to confirm the source."

• Ensure all medication dosages, administration times, imaging, and lab results are **documented** and transferred with the patient.

Prognosis and Ongoing Care

Shock management initiated in the ED continues in the ICU; **appropriate early intervention** is crucial in saving lives, yet **continued monitoring and advanced care** are equally important. Emergency physicians should:

• Remain involved or at least follow up on the patient's outcomes to refine their practice.

• Foster **multidisciplinary collaboration**, especially in complex shock cases (e.g., trauma combined with sepsis).

Ultimately, caring for a shock patient demands **continuous vigilance** from the first moment of diagnosis through ICU transfer. Once hemodynamic stability is achieved through fluids and vasopressors, addressing the underlying cause and continuing supportive therapies in the ICU are vital. Adherence to current guidelines and evidence-based practices remains **the most effective strategy** to reduce mortality (Pannu, 2023bb).

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METHEMOGLOBINEMIA

SALIH KARAKOYUN[1]

Introduction

Oxygen transportation to tissues is provided by ferrous (Fe^{2+}) iron stored in the hemoglobin protein in erythrocytes. Methemoglobinemia is a clinical picture in which ferrous iron in this structure is converted into ferric (Fe^{3+}) iron by oxidation due to congenital or acquired causes and causes hypoxia by irreversible binding of oxygen (Iolascon et al., 2021). Enzymes and systems such as Cytochrome b5 reductase (CYB5R3) that convert methemoglobin into hemoglobin are involved in the normal functioning of the body, but physiologically methemoglobin levels are below 3%. It has been reported that clinical deterioration increases correlatively as the percentage of methemoglobin in the blood increases, which is more pronounced in the presence of an acute (acquired) etiology. Although methemoglobinemia may be asymptomatic, it starts to show clinical symptoms and signs especially when it exceeds 10% of the total hemoglobin amount. When the methemoglobin level exceeds 1.5 g/dL, which corresponds to 10% for a patient with a hemoglobin

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level of 15 g/dL, cyanosis develops. The presence of cyanosis suggests to the clinician the presence of methemoglobinemia at a level exceeding 10% of the total hemoglobin concentration.

Up to 30-40% of patients with hereditary (chronic) methemoglobinemia can tolerate methemoglobinemia asymptomatically. Both acquired and genetic inherited methemoglobinemias are rare. In the literature, it has been reported that methemoglobinemia caused by external suicidal intake, environmental exposures and drugs used in treatment is proportionally more common than genetically inherited methemoglobinemias and acquired methemoglobinemias have a worse clinical course (Ivek et al., 2022). Vascular collapse, palpitation, dyspnea, central nervous system depression, cyanosis, skin bruising, pallor, petechiae, prolonged capillary refill, acidosis, tonic-clonic seizure, arrhythmia, lethargy, stupor, coma, and death may be observed, as well as ashen skin, headache, weakness, fatigue, rapid fatigue, nausea and vomiting. The only clinical manifestations may be cyanosis and hypoxia on pulse oximetry despite oxygen supplementation. There may be a discrepancy between the partial oxygen in the arterial blood gas (PaO2) and the saturation level (SpO2) measured by peripheral pulse oximetry (saturation gap) (Cefalu et al., 2020). PaO2 may appear higher than it is due to irreversible binding of oxygen to hemoglobin. In some cases, decreased partial pressure of carbon dioxide (pCO2) may be seen in arterial blood gas as a compensatory measure due to severe lactic acidosis. Although blood gas is very valuable in making the diagnosis, co-oximetry device gives us an idea about methemoglobin level as well as carboxyhemoglobin levels and can be used during follow-up. In cases with methemoglobinemia in the literature, chocolate dark brown blood samples with an unusual color have been reported(Ivek et al., 2022; Saleh, Lucyk, & McGillis, 2022).

Preparations containing sodium nitrite, metoclopramide and ranitidine known as suicide kits sold on the internet have been reported (Loiseau et al., 2023). Durao et al. reported diffuse vascular occlusion, cyanosis, livor mortis and petechiae findings, and signs of asphyxia in the autopsy results of a young patient who died with suicide kit (Durao, Pedrosa, Dinis-Oliveira, & medicine, 2020). Methylene blue 1-2mg/kg intravenously can be administered in the treatment of methemoglobinemia. It is recommended that all patients should be treated when methemoglobin levels are above 30% regardless of symptoms. In the presence of comorbidities such as cardiac and pulmonary problems, methemoglobinemia above 10% should be treated. In patients with methemoglobinemia in the range of 10-30%, symptomatic supportive treatment without methylene blue is recommended if the clinical course is asymptomatic. Avoidance of oxidative drugs is recommended in patients with genetic causes of methemoglobinemia such as Hb M disease, G6PD, CYB5R3 reductase deficiency. Since methylene blue is an MAO inhibitor, it is not recommended for use in patients with G6PD disease. In these patient groups and in patients who do not show clinical improvement despite methylene blue, exchange transfusion may be beneficial in cases of acquired methemoglobinemia. In treatment, 1.5-3 g of vitamin C and dextrose solution support are among the recommended symptomatic approaches (Ivek et al., 2022; Matin, Boie, & Moore, 2022; Tucker, Lu, & Zhang, 2018).

Case report

A 29-year-old man was found unconscious at home by his family and white foam was observed in his mouth. This was thought to be a symptom of a tonic clonic seizure at home. When he was brought to the emergency department of Düzce University Faculty of Medicine Hospital by ambulance, his eyes were spontaneously open and fixed in a sitting position looking at a point. He was

localizing the painful stimulus and there was no verbal response. Glasgow coma score was evaluated as 10. Especially the head and neck region was dark gray/black, ash-colored. The rest of the body was cold, mottled skin color similar to cutis marmaratus. Other systemic examination revealed no pathology. Vital findings at the first admission were as follows: Temperature: 35.3 °C, Pulse rate: 144 beats/minute, Blood pressure: 83/48, Spo2: 86% with mask oxygen. Although 100% oxygen was administered at 15 liters/minute with a reservoir mask, this value remained the same. The electrocardiogram at the first admission showed sinus tachycardia of 144 bpm with no ST-T changes and no additional pathology. During blood sampling, the nurse in charge showed unusual chocolate dark brown venous blood. The patient's family found a box labeled 'sodium nitrite' and 3-4 empty blisters of metoclopramide 10 mg tablets next to the patient when they called the emergency assistance center. The patient had no known comorbidities in her history, but he had made one previous unsuccessful suicide attempt and had ordered sodium nitrite from the internet. It was thought that he may have taken metoclopramide to prevent nausea, to increase tolerance and to increase the effect of the poison or he may have learned from the internet that it may increase methemoglobinemia. Since his clinical appearance and general condition were not good at the time of initial presentation, venous blood gas was sent along with routine blood tests. pH: 7.55, pO2: 286 mmHg, pCO2: 9.4 mmHg, SO2: 36.8% HCO3: 14.4 mEql/L, lactate: 14.3 mg/dL cBase(Ecf,ox): -14.3 mmol\L, Glucose: 188 mg/dL, Na+: 137 mEql/L, K+: 5.1 mEql/L, COHb: 1.5%, MetHb level was 83%. Hemoglobin: 12.2 g/dL, WBC: 4.33 10^3/mm3, Platelet (PLT): 230 10^3/mm3, renal, liver function tests, coagulation parameters and cardiac markers with cretin kinase were within normal ranges. The patient's height was 170 cm and weight was 50 kg. Methylene blue was administered at a dose of 2mg/kg.

Hydration and oxygen support were given. Sudden cardiac arrest developed approximately 8 minutes into the follow-up and despite advanced cardiac life support and all interventions, the patient could not be saved and died.

Discussion

Painkillers/antipyretics phenacetin, phenazopyridine, antimicrobial dapsone, Primaquine, anesthetics; benzocaine, prilocaine and nitrate derivatives used by physicians in treatment are the drugs accepted as common causes of acquired methemoglobinemia (Fadah et al., 2022). Household detergents, pesticides, food colorants, aniline dyes (such as diapers), meat curing products, narcotics, laughing gas, dental gel (benzocaine), sulfonamides, nitroglycerin, nitroprusside and silver nitrate and contaminated water have been reported to cause methemoglobinemia. The most commonly accused agents from the drug group are dapsone and benzocaine. Dapsone and benzocaine constitute approximately 45% of the case series reported as side effects of medications administered for therapeutic purposes. Drugs which are more rarely blamed for methemoglobinemia; phenytoin, valproic acid, chloroquine, sulfonamide, amethocaine, tetracaine, quinolone group drugs have been reported as oxidizing agents (Alagha, Doman, Aouthmanyzx, & Medicine, 2022). Sodium nitrite has been reported to be a strong oxidizing agent and mortality rates up to 33% have been recorded in suicide attempts with sodium nitrite. McCann et al. reported that 84% of suicide attempts with sodium nitrite resulted in serious complications including death (Garcia-Galindo et al., 2024; McCann, Tweet, & Wahl, 2021). Acetaminophen is a drug we use very frequently and it is claimed that this drug may cause Sulfhemoglobinemia and methemoglobinemia due to oxidative stress at high doses (Seltzer et al., 2022). Sahu et al. suggested in a letter to the editor that

acetaminophen may cause sulfhemoglobinemia due to its similarity with phenacetin, but the development of methemoglobinemia may be exaggerated and rare (Sahu, Mishra, Lal, & George, 2020). Fadah et al., on the other hand, held hydrocortiazide given for hypertension responsible for methemoglobinemia in one case (Fadah et al., 2022). Since there were no other cases of hydrocortiazide-associated methemoglobinemia reported in the literature, they thought that this may be an exposure that may release free radicals such as heat, light and humidity in the storage conditions of the drug, or it may be related with the expiration date or it may be a manufacturing defect.

Conclusion

The number and frequency of trauma patients admitted to emergency departments is increasing day by day with the increasing population. In a significant number of trauma patients, incision suturing and local anesthetics are given during this procedure. We would like to remind you that there are drugs such as prilocaine and lidocaine with common side effects or rare side effects such as methemoglobinemia. While evaluating blood gas, methemoglobin should be taken into consideration and one should be alert in unexplained hypoxia. It was emphasized that methemoglobinemia can be fatal especially when the level exceeds 70% and the importance of not discharging all patients above 30% without receiving the necessary support/medical treatments and that an impressive suicide method can be obtained by ordering over the internet and that methylene blue antidote should be available in emergencies.

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ELECTRICAL INJURIES IN THE EMERGENCY DEPARTMENT

ERDİNÇ ŞENGÜLDÜR

Introduction

Electrical injuries are traumas caused by exposure to low- and high-voltage electric current and have a wide clinical spectrum ranging from severe burns to cardiac arrhythmias. Electrical accidents can occur at home, at work or in open spaces and are frequently seen in children, construction workers and occupational groups working with electricity (Karray et al., 2025). Electrical injuries can cause both thermal and electrical damage, making their management complex (Zemaitis, Lopez, & Huecker, 2025). Some basic electrical concepts need to be known to understand electrical injuries. Voltage, which refers to the electric potential difference, is generally classified as low voltage for exposures below 1000 V and high voltage for exposures above 1000 V (Dündar, Altın, Aksöz, Sarın, & Özdemir, 2023). Current, which refers to the movement of an electric charge through a conductor, can cause serious damage even in low-voltage but high-current exposures. The resistance, which is the obstacle encountered by the tissues, is high when the skin is dry and low when it is wet, causing more current to pass

through. Alternating current is a current that changes direction continuously and is often used in homes and industry, while direct current travels in one direction and is found in batteries, cars and some medical devices. Alternating current is more dangerous because of the risk of muscle spasms and fibrillation (Isaraj, Xhepa, & Isaraj, 2024; Zemaitis et al., 2025).

Damage to the body caused by electrical injuries occurs through various mechanisms such as the passage of electric current and resistance effect, thermal damage and burns, muscle spasms, cardiac effects, effects on the central nervous system and secondary injuries. When electric current passes through the body, various effects occur due to the different levels of resistance of the tissues. Nerve and muscle tissue easily conduct electric current due to low resistance and muscle spasms and neurological effects are common. In blood vessels, the current can cause endothelial damage and thrombosis, while bone shows the highest resistance, causing thermal damage to the surrounding soft tissues. The skin, on the other hand, shows variable resistance depending on its moisture and integrity; dry skin provides high resistance, while wet or injured skin shows low resistance and allows the current to pass more easily (Chen & Wang, 2024; Dündar et al., 2023; Khor et al., 2023).

Electric current generates heat in the areas it passes through and according to Joule's law, the heat generated increases as the current intensity increases and the duration of the passage increases (Zemaitis et al., 2025). This can cause deep tissue burns and necrosis. Although electrical burns usually appear small on the outside, extensive damage to internal tissues can occur. Alternating current can cause muscle spasms and the duration of exposure may be prolonged because the person is unable to stop the current. When electric current passes through the heart, it can cause serious cardiac arrhythmias. While low-voltage alternating current poses a risk of

ventricular fibrillation, high voltage can lead to cardiac asystole and myocardial necrosis (Arumugam, Thakur, & Sarabahi, 2021; Mobayen & Sadeghi, 2022). Transient changes in electrocardiography (ECG), ST-T wave abnormalities and long QT syndrome may be observed after electric shock. Electrical injuries can also cause serious damage to the brain and nervous system. While loss of consciousness, confusion and seizures may occur during exposure, memory loss, sleep disorders and chronic neuropathies may develop in the long term (Ahmed et al., 2021).

After an electric shock, a person may fall due to muscle spasms, be traumatized or suffer additional injuries such as fractures and head injuries as a result of falling from a high place. Eye and ear damage can also occur due to the electric arc. Electrical injuries can cause serious clinical pictures that vary depending on factors such as the type of current, voltage, duration of exposure and the path it follows in the body. The effects of current on the body are not limited to the skin; it can also have profound effects on the cardiac, neurological and vascular systems (Chen & Wang, 2024). In the next section, we will detail the clinical findings and diagnostic methods of electrical injuries.

Clinical Findings

Clinical manifestations in electrical injuries may vary widely and may vary depending on the severity and duration of exposure, the path of the current and the general health status of the patient. The most prominent clinical manifestations of electrical injuries include burns, musculoskeletal system effects, cardiac disorders, neurologic changes and organ failures (Karray et al., 2025; Zemaitis et al., 2025). Electric current may cause skin lesions at the entry and exit points of the body. Although these burns are usually minor, the underlying deep tissue damage can be serious. High-voltage exposures can cause deep muscle, tendon and bone burns, which

over time can lead to necrotic tissue loss. When electric current affects muscles, it can cause spasms and tetanic contractions. These muscle contractions can result in fractures, dislocations and soft tissue injuries. Especially shoulder dislocations and vertebral fractures are common findings after electrical injuries (Chen & Wang, 2024; Dündar et al., 2023; Zemaitis et al., 2025).

Among its effects on the cardiovascular system, arrhythmias are one of the most common complications. Low-voltage alternating current increases the risk of ventricular fibrillation, while high voltage can lead to asystole. Abnormalities such as ST segment changes, T-wave inversions or prolonged QT syndrome may be observed on ECG. Heart damage may result in myocardial infarction or direct myocardial necrosis (Goyal, Jagne, Dhiman, Patil, & Rattan, 2021). The effects of electric current on the central nervous system may manifest as loss of consciousness, confusion, seizures and neurological deficits. Cognitive disorders, sleep disorders and peripheral neuropathies may develop in the long term after exposure. Respiratory failure may occur as a result of the effect of electric current on respiratory muscles and this may require emergency respiratory support (Ahmed et al., 2021; Goyal et al., 2021).

In laboratory findings, patients with rhabdomyolysis may have elevated creatine kinase (CK) and myoglobin levels, impaired renal function tests and electrolyte imbalances. ECG is an essential diagnostic tool to assess cardiac effects after electrical exposure. Chest radiography and echocardiography can be used to identify cardiac and pulmonary effects. Advanced imaging modalities such as magnetic resonance imaging (MRI) or computed tomography (CT) may be required to assess burns and deep tissue damage (Dündar et al., 2023; Khor et al., 2023; Mobayen & Sadeghi, 2022).

Electrical injuries can produce a wide spectrum of clinical findings and evaluation of each patient requires a multidisciplinary

approach. Rapid assessment of the patient's clinical status, early detection of cardiac and neurologic complications and determination of appropriate treatment strategies are vital.

Diagnosis

The diagnostic process in electrical injuries begins with taking the patient's history of exposure and is supported by a comprehensive physical examination. In the initial evaluation, the duration of exposure to electric current, voltage level, type of current (alternating or direct current), entry and exit points on the body and additional traumas the patient has been exposed to should be questioned. The patient's state of consciousness, respiratory function and hemodynamic stability should be rapidly evaluated. In emergencies, critical interventions should not be delayed by applying the standard ABC (Airway, Breathing, Circulation) approach (Edelson et al., 2020; Soar et al., 2021).

In physical examination, it is important to determine the entry and exit points of electric current. Lesions on the skin may be misleading; a seemingly minor burn may hide serious muscle and bone damages underneath. Muscle spasms, fractures and dislocations should be investigated, with particular attention paid to shoulder dislocations and vertebral fractures. Assessment of the cardiovascular system is critical; if pulse irregularities, hypotension or tachycardia are detected, in-depth evaluation should be performed. Neurologic examination should assess level of consciousness, cranial nerve function and signs of peripheral neuropathy. Patients may require long-term follow-up as both acute and chronic neurologic effects may occur after exposure (Dündar et al., 2023; Mobayen & Sadeghi, 2022; Zemaitis et al., 2025).

Laboratory tests should cover a wide range to determine the patient's systemic involvement. CK and myoglobin levels should be

monitored because of the risk of rhabdomyolysis. Renal function tests (BUN, creatinine) may indicate the development of acute kidney injury due to rhabdomyolysis. Electrolyte imbalances should be evaluated, especially hyperkalemia, because it may lead to serious arrhythmias. Cardiac enzymes (Troponin, CK-MB) should be used to detect myocardial involvement by electric current. Lactate levels may be indicative of severe tissue damage or hypoperfusion (Arumugam et al., 2021; Dechent et al., 2020; Zemaitis et al., 2025).

Imaging modalities play an important role in assessing additional trauma-related injuries and the effect of electric current on internal organs. ECG should be routinely performed in all cases of electrical injury and should be carefully examined for signs such as ventricular fibrillation, bradycardia, ST segment changes or QT prolongation. 24-hour telemetric monitoring is recommended in patients at high risk of cardiac involvement. Chest radiography can be used to detect pulmonary edema or rib fractures. Echocardiography is useful to assess myocardial damage due to electric current. MRI and CT can provide more detailed information in terms of spinal cord damage or deep tissue burns (Arumugam et al., 2021; Isaraj et al., 2024).

Special diagnostic criteria require a more rigorous examination in patients with high-voltage exposure. Patients with high voltage (>1000V) exposure, altered consciousness, chest pain, neurological deficits or significant burn lesions should be hospitalized for further evaluation. Mild electrical injuries can be followed up on an outpatient basis after careful observation. However, long-term follow-up is important in patients at risk of late complications (Karray et al., 2025; Zemaitis et al., 2025).

The diagnostic process of electrical injuries requires a multidisciplinary approach. The clinical status of the patient should be evaluated together with laboratory and imaging findings and an

optimal management plan should be created. In the next section, we will focus on treatment and management strategies in electrical injuries.

Treatment

Rapid and effective intervention is vital in electrical injuries. First, the patient should be moved to a safe environment and contact with the electrical source should be cut off. It is critical that rescuers ensure their own safety. The patient's state of consciousness, breathing and pulse should be quickly assessed. If breathing and circulation have stopped, cardiopulmonary resuscitation (CPR) should be started immediately and emergency medical assistance should be called. Cardiac arrest may develop after electric shock; therefore, patients should be kept under cardiac monitoring when transported to the emergency department (Ahmed et al., 2021; Goyal et al., 2021).

In the emergency department, the patient's airway, respiration and circulation should be carefully evaluated. Since high-voltage electrical injuries may lead to serious arrhythmias, ECG should be performed and continuous monitoring should be ensured. Intravenous fluid therapy should be started due to the risk of hypovolemia and the patient's electrolyte balance should be closely monitored. Damage to muscle tissue due to electrical injuries may lead to rhabdomyolysis. Since this may lead to acute renal failure, the patient's urine output should be monitored and renal function should be protected by providing urine alkalinization. Appropriate analgesics should be administered for pain management and sedation support should be provided according to the patient's need (Karray et al., 2025; Ozdel, Cakıcı, & Sayli, 2019).

The damage seen on the skin in electrical injuries can often be misleading. Although burns appear superficial, deep tissue

damage and muscle necrosis are common conditions. Repeated physical examinations should be performed to evaluate deep tissue involvement and advanced imaging methods should be used when necessary. The risk of compartment syndrome is high in high-voltage injuries; therefore, muscle pressures should be checked at regular intervals and the need for fasciotomy should be evaluated early. In severe cases, early surgical debridement and grafting procedures are necessary. Considering the risk of infection, wound care should be performed regularly and antibiotic prophylaxis should be considered when necessary (Chen & Wang, 2024; Dündar et al., 2023).

Electrical injuries can affect the central and peripheral nervous system. Patients should be monitored neurologically and evaluated for signs such as changes in consciousness, motor deficits and sensory loss. Since spinal cord injuries or brain damage may occur along the route of the electric current, brain imaging tests should be performed when necessary. Since neurological sequelae may develop in the long term, patients should be directed to rehabilitation processes. Electric shock can also cause musculoskeletal injuries. Since muscle spasms and fractures may occur, orthopedic evaluation is important. In addition, patients should be examined in detail for eye and ear injuries (Isaraj et al., 2024; Mobayen & Sadeghi, 2022; Zemaitis et al., 2025).

Long-term consequences of electrical injuries include neurologic disorders, musculoskeletal dysfunction and psychosocial problems. Patients exposed to electric current may often experience chronic pain syndrome, depression and post-traumatic stress disorder. It is important that these patients are followed up with a multidisciplinary team and directed to physiotherapy and psychological support programs. Since permanent disabilities affecting work capacity may develop, patients may need to be

included in vocational rehabilitation processes (Chen & Wang, 2024).

Various complications may occur in electrical injuries. In the acute period, cardiac arrhythmias, respiratory failure, kidney damage and severe burns may occur. In the medium and long term, nerve damage, muscle loss, contractures and psychosocial effects are among the factors that determine the patient's quality of life. The mortality rate may be high due to organ failures and sequelae due to electric shock. Especially in high-voltage injuries, the prognosis is serious and long-term intensive care and rehabilitation may be needed (Goyal et al., 2021; Zemaitis et al., 2025).

Various measures should be taken at individual and community level to prevent such injuries. Regular maintenance of electrical installations, proper insulation of high-voltage lines and the use of occupational safety equipment are critical to prevent accidents. Protective clothing, grounding systems and training programs for people working with electricity play a vital role. Socket protectors should be used for children at home and access to hazardous areas should be restricted. Regular training of workers in high-risk occupational groups and compliance with occupational safety protocols are essential to prevent accidents (Goyal et al., 2021; Zemaitis et al., 2025).

In conclusion, rapid and correct intervention in the treatment of electrical injuries requires a multidisciplinary approach. Appropriate fluid therapy, wound care and cardiac monitoring should be provided in the early period. Prevention of deep tissue damage and systemic complications are the main factors that increase patient survival and quality of life. Long-term follow-up and rehabilitation of patients is also an integral part of the treatment process. Electrical injuries are among the emergencies where serious complications can be prevented with early and effective

management, but can be fatal due to negligence. The incidence of such injuries can be significantly reduced by implementing prevention strategies and increasing public awareness.

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EPILEPTIC SEIZURE AND STATUS EPILEPTICUS

MURAT TASDEMIR[1]

INTRODUCTION

Epilepsy is a chronic neurological disorder characterized by recurrent seizures, affecting millions of individuals worldwide. According to the International League Against Epilepsy (ILAE), epilepsy is defined by recurrent seizures resulting from transient dysfunction in brain activity. The global prevalence of epilepsy ranges approximately between 0.7% and 1.2%, corresponding to around 50–65 million affected individuals worldwide.(Serrano-Castro et al., 2015)

Epileptic seizures significantly impact patients' quality of life and are among the common reasons for emergency department visits. Prompt and accurate diagnosis followed by timely initiation of appropriate treatment is crucial for optimal patient outcomes. Epilepsy should be approached not only as a neurological disorder

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but also as a comprehensive condition with cognitive, psychosocial, and economic dimensions.(Morales et al., t.y.)

Definition

An epileptic seizure is characterized by transient physical and cognitive alterations caused by abnormal and excessive electrical discharges from neurons in the brain. Epilepsy itself refers to a chronic predisposition of the brain to generate recurrent seizures, shaped by genetic factors as well as cognitive, neurological, and psychosocial influences.(Beghi et al., 2010)

The ILAE established the clinical definition of epilepsy in 2014, which includes the following criteria:

- Two or more unprovoked seizures occurring more than 24 hours apart.
- A single seizure with a recurrence risk greater than approximately 60% over the next ten years.
- Diagnosis of an epilepsy syndrome.(Fisher et al., 2014)

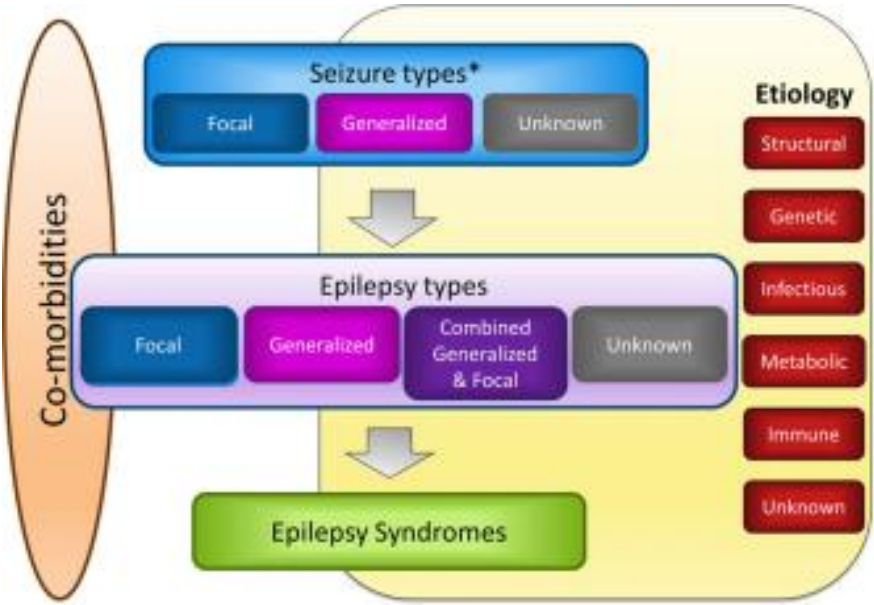
Some seizures arise due to external factors such as metabolic disturbances, exposure to toxins, structural brain anomalies, infections, or inflammatory processes. These are classified as acute symptomatic seizures and typically resolve upon addressing the underlying cause

Classification of Seizures and Epilepsy

The latest classification system developed by the International League Against Epilepsy (ILAE) is designed as a three-level structure, ensuring its applicability across diverse healthcare settings. This approach accommodates various clinical scenarios ranging from regions with limited diagnostic resources to advanced centers equipped with comprehensive diagnostic tools. Ideally,

clinicians should aim to achieve a diagnosis at all three levels and determine the underlying etiology.(Scheffer et al., 2017)

Figure 1:Classification of Seizures and Epilepsy



Reference: (Scheffer et al., 2017)

Level 1: Seizure Type

The foundation of epilepsy classification lies in identifying the seizure type. This level presupposes that the clinician has already established a definitive diagnosis of an epileptic seizure. Therefore, it is not intended as a diagnostic algorithm for differentiating epileptic events from non-epileptic ones.

Seizure types are classified into three primary categories based on ILAE's current nomenclature:

- Focal onset seizures
- Generalized onset seizures

- Seizures of unknown onset

In some clinical environments, particularly those lacking diagnostic resources such as EEG, video EEG, or neuroimaging, classifying epilepsy by seizure type may represent the maximum achievable diagnostic level. Furthermore, a higher level of classification might not be feasible in individuals experiencing only a single seizure due to insufficient clinical information.

Level 2: Epilepsy Type

The second classification level, "Epilepsy Type," assumes that epilepsy has been clinically defined according to the 2014 ILAE criteria. At this stage, epilepsy types are categorized into four main groups:

- Focal Epilepsy
- Generalized Epilepsy
- Combined Generalized and Focal Epilepsy
- Epilepsy of Unknown Type

Level 3: Epilepsy Syndrome

The highest classification level is "Epilepsy Syndrome," encompassing distinct clinical presentations with characteristic seizure patterns, EEG findings, and imaging features that frequently coexist. These syndromes typically demonstrate age-related characteristics, such as typical age at onset, possible remission, seizure triggers, circadian variations, and associated cognitive or psychiatric comorbidities.Epilepsy syndromes provide valuable insights extending beyond seizure type alone; they offer guidance regarding diagnosis, prognosis, and management strategies. However, it is crucial to note that epilepsy syndromes do not

necessarily align directly with specific etiological diagnoses; their primary utility is in guiding clinical management.

Clinically recognized epilepsy syndromes include childhood absence epilepsy, West syndrome, and Dravet syndrome. While the ILAE has not officially classified these syndromes, educational resources such as epilepsydiagnosis.org contribute significantly to understanding their clinical characteristics, EEG patterns, and educational purposes.(Scheffer et al., 2017)

Clinical Approach to Seizures

History Taking

When a patient presents to a healthcare facility following an episode, the primary step involves confirming whether the event was genuinely epileptic. Thus, obtaining a detailed and careful history from both the patient and eyewitnesses is crucial. Reliance solely on the physical description of an episode, even by healthcare professionals or eyewitnesses, can lead to misclassification, causing non-epileptic events to be mistaken for epileptic seizures.Key points to evaluate during history-taking include the presence of an aura preceding the seizure, whether the onset was abrupt or gradual, progression of motor symptoms, loss of bowel or bladder control, oral injuries such as tongue bites, and whether the seizure was unilateral or bilateral. Additionally, the duration of the seizure, as well as postictal symptoms such as altered consciousness or lethargy, must be thoroughly assessed. (Huff & Fountain, 2018, pp. 1153-1155)

Following initial history-taking, clinical features of the seizure should be evaluated. If the patient has an established epilepsy diagnosis, determining whether the current episode matches previous seizure patterns is important. In such cases, clinicians should investigate potential triggering factors, including non-

adherence to antiepileptic medications, recent medication adjustments or substitutions, sleep deprivation, excessive physical exertion, infections, fluid-electrolyte imbalances, and alcohol or substance use and withdrawal.For patients without a known epilepsy history, a more detailed investigation is required. Symptoms such as involuntary nocturnal tongue biting, unexplained injuries, or involuntary urination might suggest previously unrecognized seizures. Additionally, recent head trauma, sudden-onset or severe headaches might indicate an underlying intracranial pathology. In women, recent pregnancy or delivery should prompt consideration of eclampsia. (Huff & Fountain, 2018, pp. 1153-1155)

Furthermore, the patient's medical history should be reviewed comprehensively, evaluating for metabolic abnormalities, hypoxia, malignancies, coagulation disorders or anticoagulant therapy, exposure to industrial or environmental toxins, medication use or discontinuation, and alcohol consumption, to identify possible triggering factors.

Physical Examination

In the initial assessment, vital signs should be rapidly evaluated, along with point-of-care fingerstick blood glucose measurement. Post-seizure physical examination should specifically target seizure-related injuries, with particular attention to head and spinal injuries. Posterior shoulder dislocation is often challenging to diagnose and commonly overlooked; hence, specific attention should be paid. Additionally, oral and tongue lacerations, dental fractures, and pulmonary aspiration of foreign material are common complications to evaluate. (Huff & Fountain, 2018, pp. 1153-1155)

Following initial assessment, a comprehensive neurological examination should be performed. Repeated physical examinations

at regular intervals may be necessary to monitor the patient's clinical status.

Diagnosis

Certain clinical characteristics help differentiate epileptic seizures from non-epileptic events. A hallmark feature of epileptic seizures is their sudden onset and typically brief duration. Some focal seizures may be preceded by an aura lasting 20–30 seconds, but most seizures occur without prior warning. Episodes that progressively evolve over several minutes should prompt suspicion of non-epileptic events and require careful evaluation.Epileptic seizures usually last between 1–2 minutes; prolonged episodes beyond this duration are uncommon except in conditions such as status epilepticus.Memory impairment is an important indicator of epileptic seizures. Apart from simple partial (focal aware) seizures, patients typically cannot recall details of the seizure episode.Purposeless or automatised behaviors are also commonly observed during seizures, including repetitive motor activities such as lip-smacking or hand rubbing.Following the seizure, a characteristic postictal phase typically occurs, marked by confusion, altered consciousness, or lethargy. These clinical features provide significant diagnostic guidance for clinicians evaluating epileptic events. (Huff & Fountain, 2018, pp. 1153-1155)

Laboratory Testing and Neuroimaging

In adults presenting with a first seizure or unclear seizure history, comprehensive laboratory testing is usually necessary. Tests should be tailored according to the patient's clinical context and typically include serum glucose, basic metabolic panel, lactate, calcium, magnesium, pregnancy testing (in appropriate cases), and toxicological screening. Additionally, assessing serum concentrations of antiepileptic medications may be beneficial in

patients known to be on chronic therapy.During epileptic seizures, elevated lactate levels may result in metabolic acidosis with an increased anion gap; however, this condition is generally transient, and lactate levels usually return to normal within approximately 30 minutes.(Lipka & Bülow, 2003) Moreover, prolactin levels tend to transiently increase within 15–60 minutes following epileptic seizures. Measurement of prolactin can thus assist clinicians in distinguishing true epileptic seizures from psychogenic non-epileptic seizures.(Chen, So, Fisher, & Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology, 2005)

Cranial imaging in the emergency department is recommended for patients experiencing a first seizure or changes in the seizure pattern to exclude underlying structural brain lesions. In this context, non-contrast computed tomography (CT) of the brain is preferred due to its rapid accessibility and diagnostic effectiveness.Additionally, cranial CT is strongly indicated if an acute intracranial event (e.g., hemorrhage, tumor, infarction) is suspected based on clinical history, comorbidities, or physical examination findings. Even if a concomitant metabolic disturbance is identified, a CT scan should not be omitted unless intracranial pathology can be confidently excluded.Other traumatic or systemic complications potentially resulting from seizures must not be overlooked. Further imaging studies may be considered based on clinical presentation. For instance, chest radiographs, cervical spine radiographs, or musculoskeletal imaging should be considered if aspiration, cervical trauma, or skeletal injuries are suspected. (Huff & Fountain, 2018, pp. 1153-1155)

Treatment

Typically, active epileptic seizures require minimal intervention beyond protective and supportive measures. During an

ongoing seizure, placing the patient in the lateral (side-lying) position is recommended, if feasible, to reduce the risk of aspiration. Active respiratory assistance is generally not required during the seizure itself and may be practically difficult due to lack of patient cooperation. However, immediately after the seizure ceases, ensuring airway patency and clearing any accumulated secretions are critical steps. Thus, aspiration equipment and appropriate airway management tools must always be readily available.Administration of intravenous anticonvulsant medications is usually unnecessary during uncomplicated, brief seizures. However, clinicians must be prepared for pharmacological intervention if the seizure duration is prolonged. (Huff & Fountain, 2018, pp. 1153-1155)

Benzodiazepines represent the first-line pharmacologic therapy for prolonged or persistent seizures. Diazepam, lorazepam, and midazolam are particularly common agents within this group. Dosages vary depending on patient age, clinical scenario, and route of administration. Established dosing protocols for adults include:

- Lorazepam: Administer 4 mg intravenously; repeat every 5–10 minutes if seizures persist.
- Midazolam: Administer 10 mg intramuscularly or intravenously; repeat dosing after 5–10 minutes as necessary.
- Diazepam: Administer 10 mg intravenously; repeat every 10 minutes if seizures are uncontrolled.(Brophy et al., 2012)

Due to the significant risk of respiratory depression associated with benzodiazepines, clinicians should closely monitor respiratory function and overall clinical status after administration.(Sathe et al., 2019)

Status Epilepticus

Status epilepticus (SE) is defined clinically as a single epileptic seizure lasting longer than five minutes or multiple sequential seizures without full recovery of neurological function between episodes.(Smith et al., 2024)SE can be categorized into stages based on its duration and therapeutic responsiveness. This staging aids clinicians in structuring clinical management and determining appropriate therapeutic interventions.

Stage 1 – Early SE: Defined by seizures responsive to first-line treatments such as benzodiazepines. Typically, initial pharmacologic intervention at this stage is sufficient for seizure cessation.If status epilepticus (SE) does not respond to initial treatment and seizure activity persists beyond 10 to 30 minutes, this condition is classified as **Stage 2 – Established SE**. In this stage, administration of second-line antiepileptic medications is required.When seizure activity continues despite adequate administration of benzodiazepines and second-line antiepileptic medications, the condition is classified as **Stage 3 – Refractory SE**.

Finally, if seizure activity persists for more than 24 hours despite treatment with anesthetic agents, it is classified as **Stage 4 – Super-Refractory SE**, representing the most severe and challenging clinical stage of status epilepticus. (Saygı & Yalçın, 2018, pp. 136-137)

Figure 2: Drugs used in the stage of early tonic–clonic SE (Stage 1)

	Route of administration	Adult dose	Pediatric dose
Fosphenytoin	IV bolus (not exceeding 100 mg PE/min)	15–20 mg PE/kg	n/a
Levetiracetam	IV bolus	Optimal dose not known, most often used 2000–4000 mg	n/a
Phenytoin	IV bolus/infusion (not exceeding 50 mg/min)	15–20 mg/kg	20 mg/kg at 25 mg/min
Phenobarbital	IV bolus (not exceeding 100 mg/min)	10–20 mg/kg	15–20 mg/kg
Valproate	IV bolus	15–30 mg/kg	20–40 mg/kg

PE, phenytoin equivalents; IV, intravenous; n/a, not applicable.

Referance:(Shorvon et al.., 2008)

Figure 3: Drugs used in the stage of established tonic–clonic SE (Stage 2)

	Route of administration	Adult dose	Pediatric dose
Diazepam	IV bolus (not exceeding 2–5 mg/min)	10–20 mg	0.25–0.5 mg/kg
	Rectal administration	10–30 mg	0.5–0.75 mg/kg[a]
Clonazepam	IV bolus (not exceeding 2 mg/min)	1–2 mg at 2 mg/min[a]	250–500 µg
Lorazepam	IV bolus	0.07 mg/kg (usually 4 mg)[a]	0.1 mg/kg
Midazolam	Buccal or intranasal	5–10 mg[a]	0.15–0.3 mg/kg[a]

[a] May be repeated; PE, phenytoin equivalents; IV, intravenous; IM, intramuscular.

Referance:(Shorvon vd., 2008)

Figure 4: Anesthetic drugs used in adults in the stage of refractory tonic–clonic SE (stage 3)

Midazolam	0.1–0.3 mg/kg at 4 mg/min bolus followed by infusion of 0.05–0.4 mg/kg/hour
Thiopentone	100–250 mg bolus over 20 s then further 50 mg boluses every 2–3 min until seizures are controlled. Then an infusion of 3–5 mg/kg/hour to maintain burst suppression
Pentobarbital	10–20 mg/kg bolus at 25 mg/min followed by an infusion of 0.5–1 mg/kg/hour increasing to 1–3 mg/kg/hour to maintain burst suppression
Propofol	2 mg/kg bolus followed by an infusion of 5–10 mg/kg/hour to maintain burst suppression

Referance:(Shorvon vd., 2008)

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