SHOCK

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PATHOPHYSIOLOGY

- **Shock** is a state of circulatory insufficiency that creates an imbalance between tissue oxygen supply (delivery) and demand (consumption), resulting in end-organ dysfunction.
- The mechanisms that can result in shock are frequently divided into four categories:
- (1) hypovolemic,
- (2) distributive,
- (3) cardiogenic,
- (4) obstructive.

CATEGORIES OF SHOCK

- **Hypovolemic shock** occurs when decreased intravascular fluid or decreased blood volume causes decreased preload, stroke volume, and cardiac output (CO).

CATEGORIES OF SHOCK

- In **distributive shock**, there is relative intravascular volume depletion due to marked systemic vasodilatation. This is most commonly seen in **septic shock.**
- Compensatory responses to decreased SVR may include increased **CO** (increased contractility and heart rate) and tachycardia.
- The concurrent decreased SVR results in a decreased preload and may hinder CO overall.
- **Anaphylaxis**, **Adrenal Insufficiency**, and **Neurogenic** shock are additional causes of **Distributive Shock**.

CATEGORIES OF SHOCK

- In **cardiogenic shock**, the left ventricle fails to deliver oxygenated blood to peripheral tissues due to variances in contractility, as well as preload, afterload, and right ventricular function.
- **Myocardial infarction** is the most common cause of cardiogenic shock.
- **Dysrhythmias** are another common cause because they can lead to a decreased CO.

CATEGORIES OF SHOCK

- **Obstructive shock** is uncommon (1%) and is due to a decrease in venous return or cardiac compliance due to an increased left ventricular outflow obstruction or marked preload decrease.
- **Cardiac Tamponade**, **Pulmonary Embolism**, and **Tension Pneumothorax** are causes of obstructive shock.

FACTORS AFFECTING CARDIAC OUTPUT

- **CO** is determined by heart rate and stroke volume.
- **Stroke Volume** is dependent on preload, afterload, and contractility.
- The **Mean Arterial Pressure** is dependent on CO and the SVR.
- This is important because there is a **Mean Arterial Pressure** threshold below which oxygen delivery is decreased.
- **SVR** directly impacts mean arterial pressure, but also impacts afterload and thus CO.
- Patients in **SHOCK** may initially have normal blood pressures (**Cryptic Shock**), yet have other objective signs of shock.

FACTORS AFFECTING CARDIAC OUTPUT

- **CO** is dependent on the interplay of cardiac inotropy (speed and shortening capacity of myocardium), chronotropy (heart contraction rate), and lusitropy (ability of heart muscle to relax and heart chambers to fill).
- Determinants of **Inotropy** include autonomic input from **sympathetic** activation, **parasympathetic** inhibition, circulating catecholamines, and short-lived responses to an increase in afterload (Anrep effect) or heart rate (Bowditch effect).

FACTORS AFFECTING CARDIAC OUTPUT

- During **SHOCK** states, higher levels of **Epinephrine** will be produced and reinforce adrenergic tone.
- **Norepinephrine** interacts with cardiac **β1-receptors**, resulting in increased **Cyclic Adenosine Monophosphate**.
- This leads to a process of intracellular signaling with an increased **chronotropy** and sequestration of **calcium**, leading to myocardial relaxation.

LACTIC ACID

- When **compensatory mechanisms** fail to correct the imbalance between tissue supply and demand, **Anaerobic** metabolism occurs and results in the formation of **Lactic Acid**.
- **Lactic Acid** is rapidly buffered, resulting in the formation of measured serum **Lactate**. Normal venous **lactate** levels are <2.0 mmol/L.
- Most cases of **Lactic Acidosis** are a result of inadequate oxygen delivery, but **Lactic Acidosis** occasionally can develop from an excessively high oxygen demand (e.g., status epilepticus).
- In other cases, **Lactic Acidosis** occurs because of impaired tissue oxygen utilization (e.g., septic shock or the postresuscitation phase of cardiac arrest).
- Elevated **lactate** is a marker of impaired oxygen delivery or utilization and correlates with short-term prognosis of critically ill patients in the ED.
- Serial **lactate** assessments may be indicated because lactate clearance is associated with improved outcomes in septic shock and may assist with resuscitation.

COMPENSATORY MECHANISMS AND THEIR FAILURE

- **SHOCK** provokes a myriad of autonomic responses to maintain perfusion pressure to vital organs.
- Stimulation of the **Carotid Baroreceptor** stretch reflex activates the **sympathetic** nervous system, triggering:
- (1) **Arteriolar Vasoconstriction**, resulting in redistribution of blood flow from the skin, skeletal muscle, kidneys, and splanchnic viscera;
- (2) an increase in **heart rate** and **contractility** that increases **CO**;
- (3) constriction of **venous capacitance** vessels, which augments **venous return;**
- (4) release of the vasoactive hormones **Epinephrine**, **Norepinephrine**, **Dopamine**, and **Cortisol** to increase arteriolar and venous tone; and
- (5) release of **Antidiuretic Hormone** and activation of the **Renin-angiotensin** axis to enhance **water** and **sodium** conservation to maintain **intravascular volume.**

COMPENSATORY MECHANISMS AND THEIR FAILURE

- These compensatory mechanisms attempt to maintain **Oxygen** delivery to the most critical organs (**heart** and **brain**), but blood flow to other organs, such as the **kidneys** and **GI** tract, may be compromised.
- The **Cellular Response** to decreased **Oxygen** delivery (**adenosine triphosphate** depletion) leads to ion-pump dysfunction, influx of **Sodium**, efflux of **Potassium**, and reduction in membrane resting potential.
- These pathologic events give rise to a cascade of metabolic features including **Hyperkalemia**, **Hyponatremia**, **Azotemia**, **Hyper-** or **Hypoglycemia**, and **Lactic Acidosis**.

COMPENSATORY MECHANISMS AND THEIR FAILURE

- As **Global Tissue Hypoxia** progresses, shock ensues, followed by the **Multiorgan Dysfunction** syndrome, which is manifested by renal failure, respiratory failure, myocardial depression, liver failure, and then disseminated intravascular coagulation.

CLINICAL FEATURES

HISTORY AND COMORBIDITIES

- Although the clinical presentation of a patient in shock and the underlying cause may be quite apparent (e.g., **Acute Myocardial Infarction, Anaphylaxis**, or **Hemorrhage**), it may be difficult to obtain a history from patients in shock.
- Assistance with medical history from EMS, family, or other sources may help determine the cause of shock.

PHYSICAL EXAMINATION

- **SHOCK** is usually associated with **systemic arterial hypotension—Systolic Blood Pressure** <90 mm Hg.
- **Blood Pressure** may not drop if there is an increase in **Peripheral Vascular Resistance** in the presence of decreased **Co** with inadequate tissue hypoperfusion. For this reason, **Blood Pressure** is an insensitive marker for global tissue hypoperfusion.
- **Shock may occur with a normal blood pressure, and hypotension may occur without shock.**
- No single vital sign is diagnostic of shock, and **Blood Pressure** is particularly insensitive in the presence of peripheral vascular disease, tachycardia with a small pulse pressure, or cardiac dysrhythmias. Composite physical findings are useful in the assessment of shock

Composite Physical Examination Findings in Shock	
Temperature	**Hyperthermia** or **Hypothermia** may be present.
Heart rate	Usually elevated; however, **Paradoxical Bradycardia** can be seen in shock states due to hypoglycemia, β-blocker use, and preexisting cardiac disease.
Systolic blood pressure	May actually increases lightly when cardiac contractility increases in early shock and then fall as shock advances.
Diastolic blood pressure	Correlates with **Arteriolar Vasoconstriction** and may rise early in shock and then fall when cardiovascular compensation fails.
Pulse pressure	Increases early in shock and decreases before systolic pressure begins to drop.
Mean arterial blood pressure	Often low, < 65 mmHg

Composite Physical Examination Findings in Shock	
CNS	Acute delirium, restlessness, disorientation, confusion, and coma secondary to decrease in cerebral perfusion pressure.
Skin/capillary refill	pallor, pale, cyanosis, sweating, cool, and capillary refill time >2–3s.
Cardiovascular	Neck vein distention or flattening depending on the type of shock. Tachycardia and arrhythmias.
Respiratory	Tachypnea, increased minute ventilation, bronchospasm, and with progression to respiratory failure.
Renal	Reduced glomerular filtration. leading to **oliguria**.
Metabolic	**Lactic Acidosis**, **Hyperglycemia**, **Hypoglycemia**, and **Hyperkalemia**. As shock progresses, **Metabolic Acidosis** occurs with concurrent respiratory compensation

DIAGNOSIS

LABORATORY EVALUATION

- **Arterial Blood Gases** are useful to assess acid-base status and ventilation and oxygenation concerns, whereas a **Venous Blood Gas** is limited to acid-base information.
- A rise in serum **Lactate** correlates with **mortality** in many shock states; typically this is due to **anaerobic** metabolism.

IMAGING

- **Chest Radiograph** The portable anteroposterior view chest radiograph is often used in the evaluation of unstable patients to avoid transporting the patient during resuscitation. While limitations exist, evaluation of the heart size, presence of pulmonary edema, free air under the diaphragm, pneumothorax, infiltrates, or effusions may provide useful clinical information.

IMAGING

- **US** Bedside US assessment is an important tool for developing a differential diagnosis, assessing volume status, defining cardiac function, and assisting with procedures.
- Various US methods are described to determine overall volume status by assessing right-sided filling pressures, including measuring **Inferior Vena Cava** respiratory variation or end-expiratory **Vena Cava** respiratory variation, and other methods.
- Bedside cardiac US to assess left ventricular **Ejection Fraction** can assist with determining the cause of shock.
- US may also be used to assess for **Vascular** emergencies. Identifying an **Abdominal Aortic Aneurysm** on US may lead to further evaluation. Findings of a **Deep Vein Thrombosis** may increase the suspicion for a **Pulmonary Embolism**.
- The **Rapid Ultrasound in Shock exam** involves a three-part bedside physiologic assessment simplified as the **Pump** (cardiac), the **Tank** (volume status), and the **Pipes** (arterial and venous)

IMAGING

- **CT** Although CT is an accurate and noninvasive approach for detecting internal pathology, patients must travel from the ED to the radiology suite, which may be unadvisable in unstable shock.

TREATMENT

- The ***ABCDE*** tenets of shock resuscitation are establishing ***a*irway**, controlling the work of ***b*reathing**, optimizing the ***c*irculation**, ensuring adequate oxygen ***d*elivery**, and achieving ***e*nd** points of resuscitation.

ESTABLISHING THE AIRWAY

- Airway control is best obtained through **Endotracheal Intubation**.
- **Sedatives** used to facilitate intubation may cause **arterial vasodilatation**, **venodilation**, or **myocardial** suppression and may result in **hypotension**.
- **Positive-pressure Ventilation** reduces **Preload** and **Co**. The combination of **Sedative** agents and **Positive-pressure Ventilation** will often lead to **Hemodynamic Collapse**.
- To avoid this unwanted situation, initiate **Volume Resuscitation** and **Vasoactive Agents** before intubation and positive-pressure ventilation.

CONTROLLING THE WORK OF BREATHING

- **Respiratory muscles** are significant consumers of oxygen during shock and contribute to lactate production.
- **Mechanical Ventilation** and **Sedation** allow for adequate oxygenation, improvement of hypercapnia, and assisted, controlled, synchronized ventilation.
- All of these treatments decrease the work of breathing and improve survival.

OPTIMIZING THE CIRCULATION

- The **Trendelenburg Position** does not improve cardiopulmonary performance compared with the supine position. It may worsen pulmonary gas exchange and predispose to aspiration.
- **Passive leg raising** above the level of the heart with the patient supine may be effective.

OPTIMIZING THE CIRCULATION

- Most patients in **SHOCK** have either an absolute or relative volume deficit.
- The exception is the patient in **Cardiogenic Shock** with pulmonary edema.
- Administer **Fluid** rapidly (over 5 to 20 minutes), in set quantities of 500 or 1000 mL of normal saline, and reassess the patient after each bolus.

OPTIMIZING THE CIRCULATION

- For large fluid volumes, consider using **Lactated Ringer's** to avoid **Hyperchloremic Metabolic Acidosis** associated with **0.9% Sodium Chloride** solution.
- In clinical situations where **Hypochloremia** can be predicted, such as from GI losses due to vomiting or from urinary excretion due to diuretics, there may be an advantage to **0.9% Sodium Chloride** rehydration.
- **Central Venous** access may aid in assessing volume status (preload) and monitoring Scvo2.
- It is also the preferred route for the long-term administration of certain vasopressor therapy.
- However, there is no need for universal central access in patients with septic shock, and the need for central access should be individually determined.

Vasopressors

- **Vasopressors** are used when there has been an inadequate response to volume resuscitation or if there are contraindications to volume infusion.
- **Vasopressors** are most effective when the **vascular space** is "full" and least effective when the **vascular space** is depleted.

ENSURING ADEQUATE OXYGEN DELIVERY

- A **Hyperadrenergic** state results from the compensatory response to shock, physiologic stress, pain, cold treatment rooms, and anxiety. Pain further suppresses myocardial function, impairing oxygen delivery and increasing consumption.
- Providing analgesia, muscle relaxation, warm covering, anxiolytics, and even paralytic agents, when appropriate, decreases this inappropriate systemic oxygen consumption.
- Sequential examination of **lactate** and **Scvo2** is a method to assess adequacy of a patient's resuscitation.

END POINTS OF RESUSCITATION

- Noninvasive parameters, such as **Blood Pressure, Heart Rate**, and **Urine Output**, may underestimate the degree of remaining hypoperfusion and oxygen debt, so the use of additional physiologic end points may be informative.
- A goal-directed approach of **Mean Arterial Pressure >65** mm Hg, **Central Venous Pressure Of 8 To 12** mm Hg, **Scvo2 >70**%, and **Urine Output >0.5** mL/kg/h during ED resuscitation of septic shock has been shown to decrease mortality.

Fluid Therapy

- **Rapid restoration of fluid deficits** modulates inflammation and, if the condition progresses to shock, decreases the need for subsequent **vasopressor** therapy, **steroid** administration, and **invasive** monitoring.

Fluid Therapy

Normal saline (NS)

- Slightly hyperosmolar containing 154 mEq/L of both sodium and chloride.
- Risk of inducing hyperchloremic metabolic acidosis when given in large amounts due to relatively high chloride concentration.

Lactated Ringer's (LR)

- Sodium 130 mEq/L, potassium 4 mEq/L, calcium 3 mEq/L, chloride 109 mEq/L, lactate 28 mEq/L. LR results in a buffering of the acidemia that is advantageous over NS.
- Theoretical risk of inducing hyperkalemia in patients with renal insufficiency or renal failure due to small potassium content (very small amount).

Fluid Therapy

- **Colloids** are high-molecular-weight solutions that increase **plasma oncotic pressure**.
- **Colloids** can be classified as either natural (albumin) or artificial (starches, dextrans, and gelatins).
- Due to their higher molecular weight, **Colloids** stay in the intravascular space significantly longer than **crystalloids**. The intravascular half-life of **Albumin** is 16 hours versus 30 to 60 minutes for **Normal Saline** and **Lactated Ringer**'s solution.
- Resuscitation with **crystalloids** requires two to four times more volume than **colloids**.
- The outcome advantage between **crystalloid** and **colloids** continues to remain unresolved in sepsis, despite multiple studies.
- Due to the equivalency and the higher cost of **colloids**, **crystalloids** would seem to be a better choice for resuscitation in the ED.

Fluid Therapy

- **Sodium Bicarbonate** shifts the **oxygen-hemoglobin dissociation** curve to the left, impairs tissue unloading of hemoglobin-bound oxygen, and may worsen intracellular acidosis. However, despite no definitive clinical trials supporting benefit but perhaps harm, many clinicians remain uncomfortable withholding bicarbonate if the pH is <7.00.