

SHOCK

Physiology of Cardiovascular System (Hemodynamics)

$$BP = CO \times SVR$$

$$CO = SV \times HR \text{ (Normal = 4.0 - 8.0 l/min)}$$

$$\text{Thus, } BP = SV \times HR \times SVR$$

$$\begin{aligned} MAP &= \text{Dist. Pressure} + \frac{1}{3} \text{ Pulse Pressure} \\ &\text{- (Normal = 60 - 110 mmHg)} \end{aligned}$$

Stroke volume is a function of :-

Preload (reflects patients volume status, CVP)

After load (SVR)-resistance to ventricular ejection (vascular tone and pressure)

Contractility - Many factors including coronary perfusion, baseline myocardial function, use of cardiotonic medications

Cardiovascular Changes in Shock

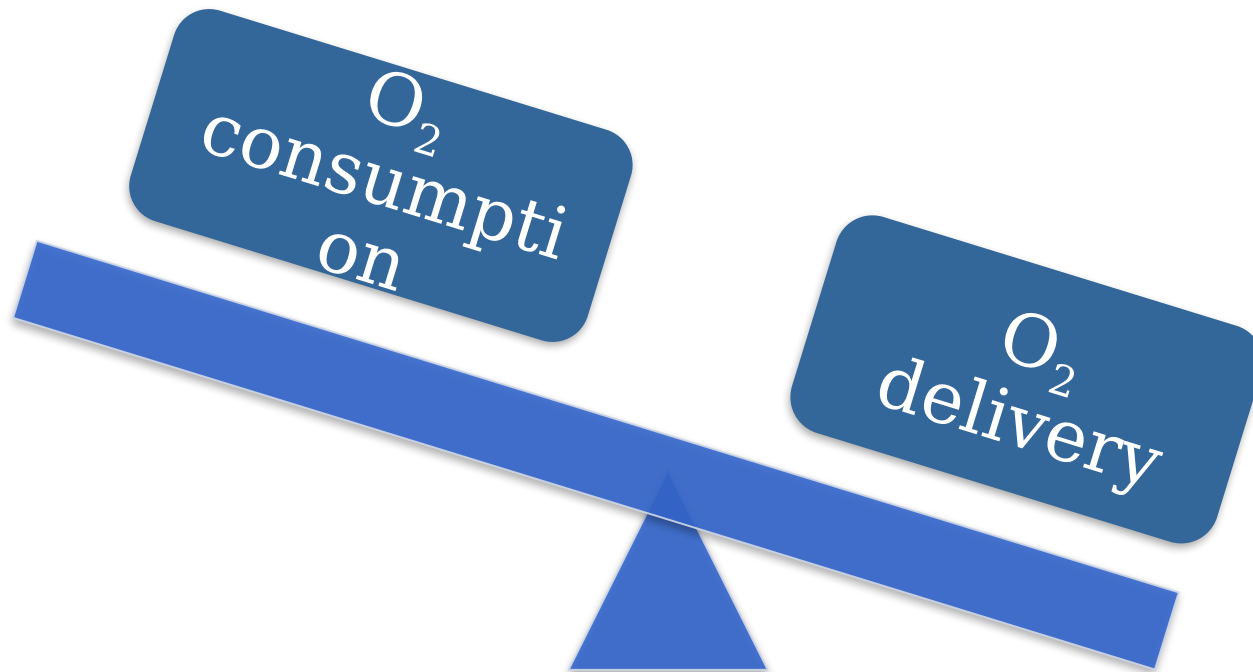
<u>Type</u>	<u>Preload</u>	<u>Afterload</u>	<u>Contractility</u>
Cardiogenic	□	□	□
Hypovolemic	□	□	No change
Distributive	□	□	□
Septic			
early	□	□	□
late	□	□	□

Definition

- Shock is the clinical syndrome that results from inadequate tissue perfusion to meet the tissue's oxygenation demand
- Or it is circulatory insufficiency that creates an imbalance between tissue oxygen supply and oxygen demand.
- The result of shock is global tissue hypoperfusion and is associated with a decreased venous oxygen content and metabolic acidosis (lactic acidosis).

Dema
nd

Suppl
y



Severity of Shock

- Compensated shock: patient is developing shock but body still able to maintain perfusion.
- Decompensated shock: patient developed shock but body no longer can compensate.
- Irreversible shock: patient developed shock but body is unable to maintain perfusion to organs.

Types of shock

- Shock is classified into four categories:-
 - (1) hypovolemic caused by inadequate circulating volume
 - (2) cardiogenic caused by inadequate cardiac pump function
 - (3) distributive caused by peripheral vasodilatation and maldistribution of blood flow
 - (4) Obstructive caused by extra cardiac obstruction to blood flow

Hypovolemic Shock

- It is the most common cause of shock in trauma patients.
- Causes:
 - External: bleeding (trauma), GI bleeding, ruptured aneurysms, hemorrhagic pancreatitis, vomiting or diarrhea, dehydration and burn.
 - Internal: third spacing: intestinal obstruction, pancreatitis, cirrhosis.

Categories of Hemorrhagic Shock

	Class I	Class II	Class III	Class IV
Blood Loss %	< 750 ml < 15 %	750-1500 ml (15-30 %)	1500-2000 ml (30-40 %)	> 2000 ml > 40 %
HR (b/min)	Normal or minimally ↑	> 100	> 120	> 140
Ventilatory R. (breath/min)	Normal	20 - 30	30 - 40	> 40
SBP	Normal	Normal	Decreased	Greatly Decreased
Urine output	Normal	20 - 30	5 - 15	Minimal

Types of Shock cont'd

- **Cardiogenic** - impaired tissue perfusion as a result of cardiac dysfunction (most common cause of death from MI)
 - Low CO and high systemic vascular resistance
 - A compensatory increase in SVR occurs to maintain vital organ function
 - Subsequent increase in LV afterload, LV work, and cardiac oxygen consumption
 - CO decreases and ultimately results in volume retention, pulmonary edema, and RV failure
 - MI (usually 40% of LV is damaged)
 - Cardiomyopathy
 - Arrhythmias
 - Heart failure
 - Cardiac tamponade
 - Acute valvular dysfunction (acute mitral regurgitation, aortic insufficiency)
 - Other severe forms of myocardial injury (trauma)

Types of Shock cont'd

- **Distributive shock -**

- the blood vessels can dilate enough that the blood within them, even though it is of normal value, is inadequate to fill the system and provide efficient perfusion.
- CAUSES: Infection, drug overdose (e.g narcotic), and spinal cord injury .

Types of Shock cont'd

3 categories

- a) Anaphylactic Shock - impaired tissue perfusion resulting from antigen-antibody reaction that releases histamine into the blood stream. Capillary permeability increases and arteriolar dilatation occurs. Blood return to the heart is decreased dramatically.**

Causes:-

- Contrast media**
- Drug reactions**
- Blood transfusion reactions**
- Food allergies**
- Insect bites or stings**
- Snake bites**

Types of Shock cont'd

- b) Septic Shock (systemic inflammatory response syndrome) - impaired tissue perfusion caused by widespread infection and invasion of microorganisms in the body, causing vasodilatation**
- C) Neurogenic shock - impaired tissue perfusion caused by damage or dysfunction of the sympathetic nervous system (rare)**
 - Trauma**
 - Anesthesia**
 - Spinal Shock**

Types of Shock cont'd

- **Obstructive Shock** - impaired tissue perfusion resulting from obstruction to blood flow
 - Low CO secondary to a physical obstruction to flow
 - Compensatory increased SVR

Causes :-

- Pulmonary Embolus
- Aortic dissection
- Pericardial tamponade
- Tension pneumothorax
- ❖ Initial clinical presentation can be identical to hypovolemic shock

Pathophysiology of Shock

- **A mean arterial pressure (MAP) of 60 to 110 mmHg is needed for cells to receive the oxygen and nutrients needed to metabolize energy in amounts sufficient to sustain life.**
- **The body has compensatory mechanisms to assist in maintaining this MAP in response to changes in volume, pumping ability of the heart and changes in the vascular system.**
- **As long as these mechanisms are effective, the body can survive the changes. When these mechanisms fail, tissues are inadequately perfused and shock begins**

Compensatory Mechanisms

- **Baroreceptors (pressure receptors) - located in the carotid sinus and aortic arch.**
 - **Decrease in MAP causes decreased stretching of the baroreceptors (they lose their inhibitory effect on the vasomotor center)**
 - **Sympathetic efferent activity is stimulated. Brain sends impulse to the adrenal glands to release catecholamines (epinephrine and norepinephrine)**
 - **Catecholamines cause an increase in heart rate and vasoconstriction**
 - **Parasympathetic activity is decreased at the same time**

Compensatory Mechanisms cont'd

- **Chemoreceptors - located in the aortic arch and carotid arteries**
 - **receptive to oxygen changes in the blood**
 - **regulates blood pressure and heart rate**
- **Kidneys - release renin which leads to the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor**
 - **leads to release of aldosterone from the adrenal cortex which results in retention of sodium and water**
 - **increase in sodium triggers release of ADH (antidiuretic hormone)**
 - **ADH causes kidneys to retain water to raise blood volume and blood pressure**

Chain of Events

- **Diminished tissue perfusion deprives cells of oxygen, nutrients, and therefore energy.**
- **Cells make ATP (energy) and store it for later use.**
- **If cells have to do this in an oxygen poor environment, then not as efficient. Anaerobic metabolism results in production of lactic acid.**
- **The increased acidity causes normal cell functions to cease.**
- **Cellular dysfunction is reversible at first but leads to organ damage if untreated**

Chain of Events cont'd

- **Hopefully, with the drop in blood pressure and the decrease in oxygenated blood, the baroreceptors and chemoreceptors will be able to compensate.**
- **If the compensatory mechanisms cannot restore tissue perfusion the syndrome of shock begins**
- **The cell swells, the cell membrane becomes more permeable and fluid and electrolytes seep from and into the cell. Mitochondria and lysosomes are damaged and the cell dies.**

Chain of Events cont'd

- **Platelets and white blood cells clump together and obstruct the microvasculature**
- **Major organs begin to malfunction as they are deprived of oxygen, as a result of hypoxemia and metabolic acidosis**
- **Respiratory failure, renal failure, decreased cerebral perfusion, and disseminated intravascular coagulation (DIC) may also be seen**
- **The earlier that medical management and interventions can be initiated, the greater the chance of survival for the patient**

Clinical pictures

Hemorrhagic Shock

Early Signs and symptoms:

- Mental status: lethargy, sleepy
- Skin: clammy, pale, mottling. Cyanosis around the nose and mouth first, spreads to extremities
- Blood pressure: begins to fall, capillary refill delayed
- Pulse: rapid and weak
- Respirations: rapid and shallow
- Other: decreased urination

Clinical Pictures con't

Late signs and symptoms

- Mental status: decreased LOC, to unresponsive
- Skin: gray, mottled, cyanotic, waxen, sweating stops
- Blood pressure: decreases, becomes undetectable
- Pulse: slows then disappears
- Respiration: agonal
- Other: bradycardia, leads to asystole

- ❖ **differs with the amount of fluid loss**
- **Class I 750 mL (15%)**
 - **Slightly anxious**
 - **Normal blood pressure**
 - **Heart rate < 100 / min**
 - **Respirations 14-20 / min**
 - **Urinary output 30 mL / hour**
 - **Warm skin, Normal Cap Refill**

Class II 750-1500 mL (15-30%)

- **Anxious**
- **Normal blood pressure**
- **Heart rate > 100 / min**
- **Decreased pulse pressure**
- **Respirations 20-30 / min**
- **Urinary output 20-30 mL / hour**
- **Pale, Cool, Cap Refill Delayed**

Class III 1500-2000 mL (30-40%)

- **Confused, anxious**
- **Decreased blood pressure**
- **Heart rate > 120 / min**
- **Decreased pulse pressure**
- **Respirations 30-40 / min**
- **Urinary output 5-15 mL / hour**
- **V. Pale, Sweaty, Cap refill V Delayed**

Class IV >2000 mL (>40%)

- **Confused, lethargic**
- **Hypotension**
- **Heart rate > 140 / min**
- **Decreased pulse pressure**
- **Respirations >35 / min**
- **Urinary output negligible**

Pulses

- Radial 70-80 mmHg
- Femoral 60-70 mmHg
- Carotid ≤ 60 mmHg

Cardiogenic Shock: Signs and Symptoms

- Drop in cardiac output
 - Skin: cyanosis
 - Pulse: bradycardia, tachycardia, or within normal limits
 - Respirations: diminishing breath sounds progressing to wheezing and crackles. Patient complains of increasing dyspnea.
 - Other: pulmonary edema and left heart failure

Signs and Symptoms of Obstructive Shock

- Mental status: anxiety, feeling of impending doom
- Skin: pallor to cyanosis around the mouth and the nose
- Other: chest pain, lung sounds may be clear, possible syncope, cardiac dysrhythmias (PVC's, A-Fib common) can lead to sudden cardiac arrest

Signs and symptoms of septic shock

- Skin: varies from flushed pink (if fever is present) to pale and cyanotic.
- Blood pressure: early—cardiac output increases but toxins prevent increase in BP. Late --- drop in BP, hypotension
- Respiratory: dyspnea with altered lung sounds
- Other: high fever, (except in elderly and very young), Late sign is pulmonary edema

Neurogenic Shock S/S

- Mechanism: vasodilation
- Skin: areas of vasodilation, at first become warm, pink and dry. Later with pooling: mottling of dependent areas, pallor and cyanosis to the upper surfaces
- Pulse: highly variable depending on injury or action of drug/poison: May be abnormally slow or abnormally fast, usually not normal
- Respiration: severely compromised: becoming slow, shallow, with abnormal patterns. Patient may lose stimulus to breathe
- Other: hypothermia. Pulmonary edema with drug or poisoning

Clinical Signs		Hypovolemic Shock	Distributive Shock	Cardiogenic Shock	Obstructive Shock
A	Patency	Airway open and maintainable/not maintainable			
B	Respiratory rate	Increased			
	Respiratory effort	Normal to increased		Labored	
	Breath sounds	Normal	Normal (± crackles)	Crackles, grunting	
C	Systolic blood pressure	Compensated Shock → Hypotensive Shock			
	Pulse pressure	Narrow	Wide	Narrow	
	Heart rate	Increased			
	Peripheral pulse quality	Weak	Bounding or weak	Weak	
	Skin	Pale, cool	Warm or cool	Pale, cool	
	Capillary refill	Delayed	Variable	Delayed	
	Urine output	Decreased			
D	Level of consciousness	Irritable early Lethargic late			
E	Temperature	Variable			

Fluid management of Shock

- Crystalloid Solutions
 - Normal saline
 - Ringers Lactate solution
- Colloid Solutions
 - Pentastarch
 - Blood products (albumin, RBC, plasma)

Fluid management con't Crystalloid Solutions

- Normal Saline
- Lactated Ringers Solution
- Require 3:1 replacement of volume loss
- e.g. estimate 1 L blood loss, require 3 L of crystalloid to replace volume

Fluid management con't

Colloid Solutions

- Albumin 5%
- Red Blood Cells
- Fresh Frozen Plasma
- Replacement of lost volume in 1:1 ratio

Colloids vs Crystalloids

- Stay in circulation
- Plasma Expand
- May disrupt Clotting Direct and Dilutional
- Anaphylaxis
- Lesser Volume
- All fluid compartments
- No direct effect on Clotting
- Cellular function better preserved
- Greater volume c. X3

Oxygen Carrying Capacity

- Only RBC contribute to oxygen carrying capacity (hemoglobin)
- Replacement with all other solutions will
 - support volume
 - Improve end organ perfusion
 - Will NOT provide additional oxygen carrying capacity

Oxygen Carrying Capacity con't

- ❑ It is given as packed RBCs, which should be cross-matched, but in an urgent situation, 1 to 2 units of type O Rh-negative blood are an acceptable alternative.
- ❑ When > 1 to 2 units are transfused (eg, in major trauma), blood is warmed to 37°C .

Fluid use in others

- 1) Fluid Resuscitation in burn patient :-
 - IV LR 1L/hr in adult
20ml/kg/hr in children
 - Foley catheter – UO/hr.:
 - 30ml/hr in adults
 - 1ml/kg/hr in children
 - After ascertaining the extent of the burn estimate the fluid needs:
 - Parkland formula: 4ml/kg/% of TBSA burn (LR)
 - <50% TBSA □ use 2 large bore IV line
 - >50% or other medical problems □ CVP
 - >65% TBSA--> burn center

Fluid use con't

2) Pediatric patient

- Except for myocardial failure use 10-20ml/kg every 2-10 minutes. Reassess after every bolus.
- At 60ml/kg consider: ongoing losses, adrenal insufficiency, intestinal ischemia, obstructive shock. Get CXR. May need inotropes.

General Treatment of Shock

- Remember your **ABC's**
- Administer airway
- 100% O2 via a non- re-breather mask
- Assist ventilations if necessary
- Position patient to assist perfusion
- Keep patient warm
- Perform focused assessment
- Monitor and adjust O2, gain IV access, cardiac monitor, pulse oximetry
- Fluid replacement of LR or NS
- Need 3 liter of fluid to replace 1 liter of blood loss
- Apply pressure to IV or blood to facilitate faster infusion

Management of Shock Emergencies Flowchart

- Oxygen
- Pulse oximetry
- ECG monitor
- IV/IO access
- BLS as indicated
- Bedside glucose

Hypovolemic Shock

Specific Management for Selected Conditions

Nonhemorrhagic

- 20 mL/kg NS/LR bolus, repeat as needed
- Consider colloid after 3rd NS/LR bolus

Hemorrhagic

- Control external bleeding
- 20 mL/kg NS/LR bolus repeat 2 or 3x as needed
- Transfuse PRBCs as indicated

Distributive Shock

Specific Management for Selected Conditions

Septic

- Management Algorithm:
- Septic Shock

Anaphylactic

- IM epinephrine (or auto-injector)
- Antihistamines
- Corticosteroids
- Epinephrine infusion
- Albuterol

Neurogenic

- 20 mL/kg NS/LR bolus, repeat PRN
- Vasopressor

Cardiogenic Shock

Specific Management for Selected Conditions

Bradyarrhythmia/Tachyarrhythmia

- Management Algorithms:
- Bradycardia
 - Tachycardia with poor perfusion

Other (eg, CHD, Myocarditis, Cardiomyopathy, Poisoning)

- 5 to 10 mL/kg NS/LR bolus, repeat PRN
- Vasoactive infusion
- Consider expert consultation

Obstructive Shock

Specific Management for Selected Conditions

Ductal-Dependent (LV Outflow Obstruction)

- Prostaglandin E₁
- Expert consultation

Tension Pneumothorax

- Needle decompression
- Tube thoracostomy

Cardiac Tamponade

- Pericardiocentesis
- 20 mL/kg NS/LR bolus

Pulmonary Embolism

- 20 mL/kg NS/LR bolus, repeat PRN
- Consider thrombolytics, anticoagulants
- Expert consultation

Monitoring intervention

- Monitoring for patients in shock include:-
 - ✓ ECG
 - ✓ Pulse oximetry
 - ✓ Blood pressure
 - ✓ Urine output
 - ✓ CVP
 - ✓ Base deficit
 - ✓ Serum lactate - <2 mmol/L

Adequacy of resuscitation

- Capillary refill < 2 sec
- Adequate pulses
- Warm limbs
- Normal mental status
- Urine output > 1 mL/kg/hr
- Adequate blood pressure
- Improved base deficit
- Decreased lactate

Summary

- Goal of therapy is identification, evaluation, and treatment of shock in its earliest stage
- Initial priorities are for the ABC's (universal immediate management of shock)
- Fluid, fluid, fluid - Administer adequate amounts of fluid rapidly. Remember ongoing losses.
- Fluid resuscitation begins with 20cc/kg of crystalloid or 10cc/kg of colloid
- Subsequent treatment depends on the etiology of shock and the patient's hemodynamic condition
- Successful resuscitation depends on early and judicious intervention