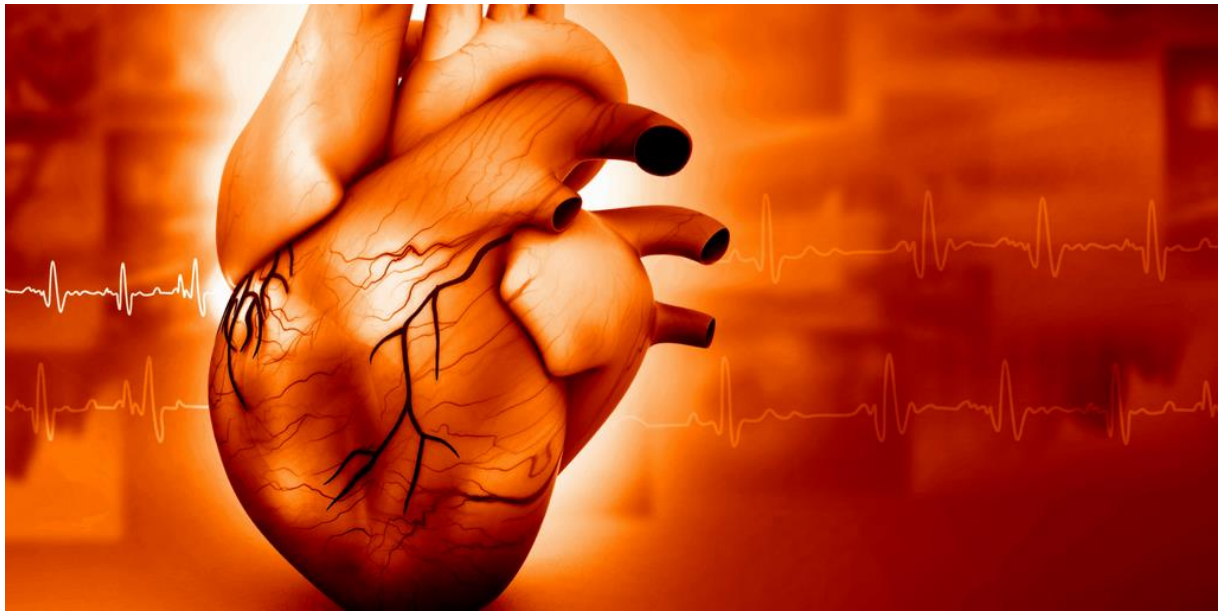
SHOCK



BY SOPHIE NJENGA

BSCN KU

LESSON OBJECTIVES

1. *Define shock*
2. *Identify the types of shock*
3. *Explain the various types of shock by; their cause and pathophysiology*
4. *Explain the management of the various types of shock*

INTRODUCTION

DEFINITION OF SHOCK

Shock can best be defined as *a condition in which widespread perfusion to the cells is inadequate to deliver oxygen.*

- Adequate blood flow to the tissues and cells requires an adequate cardiac pump, effective vasculature or circulatory system, and sufficient blood volume.

- If one of these components is impaired, perfusion to the tissues is threatened or compromised.
- Without treatment, inadequate blood flow to the cells results in poor delivery of oxygen and nutrients, cellular hypoxia, and cell death that progresses to organ dysfunction and eventually death
- Shock affects all body systems.
- It may develop rapidly or slowly, depending on the underlying cause.

During shock, the body struggles to survive, calling on all its homeostatic mechanisms to restore blood flow.

- Regardless of the initial cause of shock, certain physiologic responses are common to all types of shock.
- These physiologic responses include

1. **hypoperfusion of tissues,**
2. **hypermetabolism, and**
3. **activation of the inflammatory response.**

- The body responds to shock states by ***activating the sympathetic nervous system and mounting a hypermetabolic and inflammatory response.***

TYPES OF SHOCK

1. *Cardiogenic shock*
2. *Anaphylactic shock*
3. *Hypovolemic shock*
4. *Neurogenic shock*
5. *Septic shock*

1. CARDIOGENIC SHOCK

- Cardiogenic shock occurs when the ***heart's ability to contract and to pump blood is impaired and the supply of oxygen is inadequate for the heart and tissues.***
- The causes of cardiogenic shock are known as either *coronary or noncoronary*.
- Coronary cardiogenic shock is more common than noncoronary

EPIDEMIOLOGY

- Cardiogenic shock occurs as a serious complication in **5% to 10%** of patients hospitalized with acute myocardial infarction.
- Incidence of cardiogenic shock is more common in men than in women because of their higher incidence of coronary artery disease.

CLASSIFICATION OF CAUSES

The causes of cardiogenic shock are known as either coronary or non-coronary;

1. **Coronary Causes:**

- Coronary cardiogenic shock is more common than non-coronary cardiogenic shock and is seen most often in patients with acute myocardial infarction.

2. **Noncoronary Causes:**

- Noncoronary cardiogenic shock is related to conditions that stress the myocardium as well as conditions that result in an ineffective myocardial function.

PATHOPHYSIOLOGY

This is what happens in cardiogenic shock:

1. **Inability to contract**

- When the myocardium can’t contract sufficiently to maintain adequate cardiac output, stroke volume decreases and the heart can’t eject an adequate volume of blood with each contraction.

2. **Pulmonary congestion**

- The blood backs up behind the weakened left ventricle, increasing preload and causing pulmonary congestion.

4.Diminished stroke volume.

- As a result of the diminished stroke volume, coronary artery perfusion and collateral blood flow is decreased.

5. Increased workload

- These mechanisms increase the heart's workload and enhance left-sided heart failure.

6. End result

- The result is myocardial hypoxia, further decreased cardiac output, and a triggering of compensatory mechanisms to prevent decompensation and death.

CAUSES

Cardiogenic shock can result from any condition that causes significant left ventricular dysfunction with reduced cardiac output.

The causes include;

1. **Myocardial infarction (MI).**

Regardless of the underlying cause, left ventricular dysfunction sets in motion a series of compensatory mechanisms that attempt to increase cardiac output, but later leads to deterioration.

2. Myocardial ischemia.

- Compensatory mechanisms may initially stabilize the patient but later would cause deterioration with the rising demands of oxygen of the already compromised myocardium.

3. End-stage cardiomyopathy.

- The inability of the heart to pump enough blood for the systems causes cardiogenic shock.

CLINICAL MANIFESTATIONS

Cardiogenic shock produces symptoms of poor tissue perfusion.

1. **Clammy skin.**

The patient experiences cool, clammy skin as the blood could not circulate properly to the peripheries.

2. **Decreased systolic blood pressure.**

The systolic blood pressure decreases to 30 mmHg below baseline.

3. **Rapid respirations.**

- The patient experiences rapid, shallow respirations because there is not enough oxygen circulating in the body.

4. **Oliguria.**

- An output of less than 20ml/hour is indicative of oliguria.

5. **Cyanosis.**

- Cyanosis occurs because there is insufficient oxygenated blood that is being distributed to all body systems.

6. Tachycardia.

- Tachycardia occurs because the heart pumps faster than normal to compensate for the decreased output all over the body.

MEDICAL MANAGEMENT

The aim of treatment is to enhance cardiovascular status by:

1. Oxygen.

Oxygen is prescribed to minimize damage to muscles and organs.

2. Angioplasty and stenting.

A catheter is inserted into the blocked artery to open it up.

3. Balloon pump.

- A balloon pump is inserted into the aorta to help blood flow and reduce workload of the heart.

4. Pain control.

- In a patient that experiences chest pain, IV morphine is administered for pain relief.

5. **Hemodynamic monitoring.**

An arterial line is inserted to enable accurate and continuous monitoring of BP and provides a port from which to obtain frequent arterial blood samples.

6. **Fluid therapy.**

Administration of fluids must be monitored closely to detect signs of fluid overload.

NURSING INTERVENTIONS

1. **Fluids**

- IV infusions must be observed closely because tissue necrosis and sloughing may occur if vasopressor medications infiltrate the tissues, and it is also necessary to monitor the intake and output *(reduces risk for fluid overload).*

2. **Enhance safety and comfort**

- Administering of medication to relieve chest pain, preventing infection at the multiple arterial and venous line insertion sites, protecting the skin, and monitoring respiratory and renal functions help in safeguarding and enhancing the comfort of the patient.

3. Prevent Recurrence

- Identifying at-risk patients early, promoting adequate oxygenation of the heart muscle, and decreasing cardiac workload can prevent cardiogenic shock.

4. Administering Medications and Intravenous Fluids

- The nurse plays a critical role in the safe and accurate administration of IV fluids and medications.
- Fluid overload and pulmonary edema are risks because of ineffective cardiac function and accumulation of blood and fluid in the pulmonary tissues.

2. ANAPHYLACTIC SHOCK

- Anaphylactic shock is caused by a ***severe allergic reaction when patients who have already produced antibodies to a foreign substance (antigen) develop a systemic antigen–antibody reaction.***
- It occurs rapidly and is life-threatening.
- An antigen–antibody reaction provokes mast cells to release potent vasoactive substances, such as histamine or bradykinin, causing widespread vasodilation and capillary permeability

PATHOPHYSIOLOGY

Anaphylaxis occurs in an individual after re-exposure to an antigen to which that person has produced a specific IgE antibody.

1. **Re-exposure**

- Upon re-exposure to the sensitized allergen, the allergen may cross-link the mast cell or basophil (***surface-bound allergen-specific IgE)*** resulting in cellular degranulation as well as denovosynthesis of mediators.

2. **Binding**

- Immunoglobulin E (IgE) binds to the antigen (the foreign material that provokes the allergic reaction).

3. **Activation**

- Antigen-bound IgE then activates receptors on mast cells and basophils.

4. **Inflammatory mediators release**

- This leads to the release of inflammatory mediators such as histamine.

5. **Histamine release.**

Many of the signs and symptoms of anaphylaxis are attributable to binding of histamine to its receptors; binding to H receptors mediates pruritus, rhinorrhea, tachycardia, and bronchospasm.

6. **Prostaglandin release**

- Prostaglandin D2 mediates bronchospasm and vascular dilatation, which are principal manifestations of anaphylaxis.

7. **Leukotriene conversion**

- Leukotriene C4 is converted into LTD and LTE , mediators of hypotension, bronchospasm, and mucous secretion during anaphylaxis in addition to acting as chemotactic signals for eosinophils and neutrophils.

CAUSES

1. **Food allergies.**

- The most common anaphylaxis triggers in children are food allergies, such as to peanuts, and tree nuts, fish, shellfish and milk.

2. **Medication allergies.**

- Certain medications, including antibiotics, aspirin and other over-the-counter pain relievers, and the intravenous (IV) contrasts used in some imaging tests.

3. **Insect allergies.**

- Stings from bees, yellow jackets, wasps, hornets and fire ants.

MEDICAL MANAGEMENT

1. Remove antigen.

- Removing the causative antigen such as discontinuing an antibiotic agent could stop the progression of shock.

2. Administer medications.

- Administer medications that restore vascular tone and provide emergency support of basic life functions.

3. Cardiopulmonary resuscitation.

- If cardiac arrest and respiratory arrest are imminent or have occurred, cardiopulmonary resuscitation is performed.

4. Endotracheal intubation.

- Endotracheal intubation or tracheostomy may be necessary to establish an airway.

NURSING INTERVENTIONS

1. **Monitor client's airway.**
- Assess the client for the sensation of a narrowed airway.
2. **Monitor the oxygenation status.**
- Monitor oxygen saturation and arterial blood gas values.
3. **Positioning.**
- Position the client upright as this position provides oxygenation by promoting maximum chest expansion and is the position of choice during respiratory distress.

4. **Activity.**

- Encourage adequate rest and limit activities to within client's tolerance.

5. **Hemodynamic parameters.**

- Monitor the client's central venous pressure (CVP), pulmonary artery diastolic pressure (PADP), pulmonary capillary wedge pressure, and cardiac output/cardiac index.

6. **Monitor urine output.**

- The renal system compensates for low blood pressure by retaining water, and oliguria is a classic sign of inadequate renal perfusion.

3. HYPOVOLEMIC SHOCK

Hypovolemic shock is one of the most common cardiac complications.

- In **hypovolemic shock**, reduced intravascular blood volume causes circulatory dysfunction and inadequate tissue perfusion.
- Vascular fluid volume loss causes extreme tissue hypoperfusion.

Hypovolemic shock usually results from acute blood loss *(about one-fifth of the total volume).*

This leads to:

1. **Internal fluid loss.**

- Internal fluid losses can result from hemorrhage or third-space fluid shifting.

2. **External fluid loss.**

- External fluid loss can result from severe bleeding or from severe diarrhea, diuresis, or vomiting.

3. **Inadequate vascular volume.**

- Inadequate vascular volume leads to decreased venous return and cardiac output.

PATHOPHYSIOLOGY

1. **Fluid loss.**

- Fluid loss can either be internal or external fluid loss.

2. Compensatory mechanism.

- The resulting drop in the arterial blood pressure activates the body's compensatory mechanisms in an attempt to increase the body's intravascular volume.

3. Venous return.

- Diminished venous return occurs as a result of the decrease in arterial blood pressure.

4. Preload.

- The preload or the filling pressure becomes reduced.

5. Stroke volume.

- The stroke volume is decreased.

6. Cardiac output.

- Cardiac output is decreased because of the decrease in stroke volume.

CLINICAL MANIFESTATIONS

Hypovolemic shock requires early recognition of signs and symptoms.

1. **Hypotension.** Hypovolemic shock produces hypotension with narrowed pulse pressure.
2. **Cognitive.** The patient experiences decreased sensorium.
3. **Tachycardia.** The body compensates for the decreased cardiac output by pumping faster than normal, resulting in tachycardia.

4. **Rapid, shallow respirations.** Due to the decrease in oxygen delivery around the body systems, the respiratory system compensates by rapid, shallow respirations.
5. **Oliguria.** There is oliguria or decreased urine output of less than 25ml/hour.
6. **Clammy skin.** The patient develops cool, clammy, and pale skin.

MEDICAL MANAGEMENT

Emergency treatment measures must include prompt and adequate fluid and blood replacement to restore intravascular volume and raise blood pressure.

1. **Volume expansion.** Saline solution or lactated Ringer's solution, then possibly plasma proteins or other plasma expanders, may produce adequate volume expansion until whole blood can be matched.
2. **Antishock garments.** A *pneumatic antishock garment* counteracts bleeding and hypovolemia by slowing or stopping arterial bleeding; by forcing any available blood from the lower body to the brain, heart, and other vital organs; and by preventing return of the available circulating blood volume to the legs.

3. **Treat underlying cause.** If the patient is hemorrhaging, efforts are made to stop the bleeding or if the cause is diarrhea or vomiting, medications to treat diarrhea and vomiting are administered.
4. **Redistribution of fluid.** Positioning the patient properly assists fluid redistribution, wherein a modified Trendelenburg position is recommended in hypovolemic shock.

NURSING INTERVENTIONS

Nursing care focuses on assisting with treatment targeted at the cause of the shock and restoring intravascular volume.

1. **Safe administration of blood.** It is important to acquire blood specimens quickly, to obtain baseline complete blood count, and to type and crossmatch the blood in anticipation of blood transfusions.
2. **Safe administration of fluids.** The nurse should monitor the patient closely for cardiovascular overload, signs of difficulty of breathing, pulmonary edema, jugular vein distention, and laboratory results.

3. **Monitor weight.** Monitor daily weight for sudden decreases, especially in the presence of decreasing urine output or active fluid loss.
4. **Monitor vital signs.** Monitor vital signs of patients with deficient fluid volume every 15 minutes to 1 hour for the unstable patient, and every 4 hours for the stable patient.
5. **Oxygen administration.** Oxygen is administered to increase the amount of oxygen carried by available hemoglobin in the blood.

4. NEUROGENIC SHOCK

- Neurogenic shock is a *distributive type of shock*.
- In neurogenic shock, ***vasodilation occurs as a result of a loss of balance between parasympathetic and sympathetic stimulation.***
- It is a type of shock (a life-threatening medical condition in which there is insufficient blood flow throughout the body) that is caused by the sudden loss of signals from the sympathetic nervous system that maintain the normal muscle tone in blood vessel walls.

PATHOPHYSIOLOGY

The patient experiences the following that results in neurogenic shock:

1. **Stimulation.**

- Sympathetic stimulation causes vascular smooth muscle to constrict, and parasympathetic stimulation causes vascular smooth muscle to relax or dilate.

2. **Vasodilation.**

- The patient experiences a predominant parasympathetic stimulation that causes vasodilation lasting for an extended period of time, leading to a relative hypovolemic state.

3. Hypotension.

- Blood volume is adequate, because the vasculature is dilated; the blood volume is displaced, producing a hypotensive (low BP) state.

4. Cardiovascular changes.

- The overriding parasympathetic stimulation that occurs with neurogenic shock causes a drastic decrease in the patient's systemic vascular resistance and bradycardia.

5. Insufficient perfusion.

- Inadequate BP results in the insufficient perfusion of tissues and cells that is common to all shock states.

CAUSES

Neurogenic shock could be caused by the following:

1. **Spinal cord injury.**

- Spinal cord injury (SCI) is recognized to cause hypotension and bradycardia.

2. **Spinal anesthesia.**

- Injection of an anesthetic into the space surrounding the spinal cord results in a fall in blood pressure because of dilation of the blood vessels in the lower portion of the body and a resultant diminish of venous return to the heart.

3. Depressant action of medications.

- Depressant action of medications and lack of glucose could also cause neurogenic shock.

CLINICAL MANIFESTATIONS

The clinical manifestations of neurogenic shock are as a result of parasympathetic stimulation:

1. **Dry, warm skin.** Instead of cool, moist skin, the patient experiences dry, warm skin due to vasodilation and inability to vasoconstrict.
2. **Diaphragmatic breathing.** If the injury is below the 5th cervical vertebra, the patient will exhibit diaphragmatic breathing due to loss of nervous control of the intercostal muscles (which are required for thoracic breathing).
3. **Hypotension.** Hypotension occurs due to sudden, massive dilation.

4. **Bradycardia.** Instead of getting tachycardic, the patient experience bradycardia.
5. **Respiratory arrest.** If the injury is above the 3rd cervical vertebra, the patient will go into respiratory arrest immediately following the injury, due to loss of nervous control of the diaphragm.

MEDICAL MANAGEMENT

Treatment of neurogenic shock involves:

1. **Restoring sympathetic tone.** It would be either through the stabilization of a spinal cord injury or, in the instance of spinal anesthesia, by positioning the patient appropriately.
2. **Immobilization.** If the patient has a suspected case of spinal cord injury, a traction may be needed to stabilize the spine to bring it to proper alignment.
3. **IV fluids.** Administration of IV fluids is done to stabilize the patient’s blood pressure

NURSING INTERVENTIONS

Nursing interventions are directed towards supporting cardiovascular and neurologic function until the usually transient episode of neurogenic shock resolves.

1. **Elevate head of bed.** Elevation of the head helps prevent the spread of the anesthetic agent up the spinal cord when a patient receives spinal or epidural anesthesia.
2. **Lower extremity interventions.** Applying ***anti-embolism stockings*** and elevating the foot of the bed may help minimize pooling of the blood in the legs and prevent thrombus formation.

3. **Passive range of motion** of the immobile extremities helps promote circulation.
4. **Airway patency.** Maintain patent airway: keep head in neutral position, elevate head of bed slightly if tolerated, use airway adjuncts as indicated.
5. **Oxygen.** Administer oxygen by appropriate method (nasal prongs, mask, intubation, ventilator).

6. **Activities.** Plan activities to provide uninterrupted rest periods and encourage involvement within individual tolerance and ability.
7. **BP monitoring.** Measure and monitor BP before and after activity in acute phases or until stable.
8. **Reduce anxiety.** Assist patient to recognize and compensate for alterations in sensation.

5. SEPTIC SHOCK

One of the most common types of circulatory shock and the incidences of this disease continue to rise despite the technology.

- **Sepsis** is a **systemic response** to infection.
- It is manifested by two or more of the SIRS (Systemic Inflammatory Response Syndrome) criteria as a consequence of documented or presumed infection.
- **Septic shock** is associated with sepsis.

It is characterized by symptoms of sepsis plus hypotension and hypoperfusion despite adequate fluid volume replacement.

PATHOPHYSIOLOGY

- Microorganisms invade the body tissues and in turn, patients exhibit an immune response.
- The immune response provokes the activation of **biochemical cytokines** and mediators associated with an inflammatory response.
- Increased capillary permeability and vasodilation interrupt the body’s ability to provide adequate perfusion, oxygen, and nutrients to the tissues and cells.

- Pro-inflammatory and anti-inflammatory cytokines released during the inflammatory response and activates the coagulation system that forms clots whether or not there is bleeding.
- The imbalance of the inflammatory response and the clotting and fibrinolysis cascades are critical elements of the physiologic progression of sepsis in affected patients.

CAUSES AND RISK FACTORS

There are several factors that can put the patient at risk for septic shock, and these include:

1. Patients with ***immunosuppression*** have greater chances of acquiring septic shock because they have decreased immune system, making it easier for microorganisms to invade the body tissues.
2. **Age:** Elderly people and infants are more prone to septic shock because of their weak immune system.
3. ***Malnourishment*** can lower the body's defenses, making it susceptible to the invasion of pathogens.

4. **Chronic illness:** Patients with a longstanding illness are put at risk for sepsis because the body’s immune system is already weakened by the existing pathogens.
5. **Invasive procedures:** Invasive procedures can introduce microorganisms inside the body that could lead to sepsis.

CLINICAL MANIFESTATIONS

1. Since the ability of the body to provide oxygen and nutrients is interrupted, the heart compensates by pumping faster ***(tarchycardia).***
2. ***Hypotension*** occurs because of vasodilation.
3. To compensate for the decreased oxygen concentration, the patient tends to breathe faster, and also to eliminate more carbon dioxide from the body ***(tarchypnea).***
4. The inflammatory response is activated because of the invasion of pathogens ***(redness, oedema, and fevers).***

5. **Decreased urine output.** The body conserves water to avoid undergoing *dehydration* because of the inflammatory process.
6. **Changes in mentation**. As the body slowly becomes acidotic, the patient's mental status also deteriorates.
7. **Elevated lactate level.** The lactate level is elevated because there is maldistribution of blood.

MEDICAL MANAGEMENT

- The current treatment of septic shock and sepsis include:

❑ *Identification and elimination of the cause of infection.*

NURSING INTERVENTIONS

1. **Infection control**. All invasive procedures must be carried out with **aseptic technique** after careful hand hygiene.
2. **Collaboration**. The nurse must collaborate with the other members of the healthcare team to identify the site and source of sepsis and specific organisms involved.
3. **Management of fever**. The nurse must monitor the patient closely for shivering.

4. **Management of the causative agent:** The nurse should administer prescribed IV fluids and medications including antibiotic agents and vasoactive medications.
5. **Monitor blood levels:** The nurse must monitor antibiotic toxicity, BUN, creatinine, WBC, hemoglobin, hematocrit, platelet levels, and coagulation studies.
6. **Assess physiologic status:** The nurse should assess the patient’s hemodynamic status, fluid intake and output, and nutritional status.


ANY QUESTIONS?
THANK YOU