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A Review on Treatment Diagnosis and Prevention of Shock

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Abstract: *The purpose of this chapter is to educate nurses, residents, and medical students about emergency medicine. Shock has been discussed, along with its definitions. An introduction to shock has been provided at the start of the chapter. With comparison, several shock categories have been developed. Every shock type has been covered, along with its handling and unique characteristics. There has been much discussion about septic shock, and various terms and meanings have been employed. Guidelines for the most recent surviving sepsis campaign have been written down. Hemorrhagic shock has also been extensively discussed, and a table that distinguishes its stages has been presented. All of the references utilized to complete this chapter are finally included at the end of the chapter.*

Keywords: hypovolemia, sepsis, distributive shock, obstructive shock, cardiogenic shock

I. INTRODUCTION

A clinical syndrome known as shock is characterized by oliguria (a urine output of less than 20 mL/hr or 0.3 ml/kg/hr for two consecutive hours), hypotension (a systolic blood pressure of less than 90 mmHg or a mean arterial pressure of less than 60 mmHg or reduced by greater than 30%, for at least 30 minutes), and poor peripheral perfusion (e.g. cool and clammy skin which demonstrates poor capillary refill). Disorders that result in an underlying hemodynamic defect of a low intravascular volume and a decrease in myocardial contractility are linked to hypovolaemic shock and cardiogenic shock, respectively[1]

Over the past 50 years, there has been very little change in the understanding and treatment of hypovolaemic shock. The treatment consists of replacing the intravascular volume by infusing blood and/or 0.9% sodium-containing colloid or crystalloid fluids, as well as managing the causative lesion through surgery to correct blood loss[2]

An abrupt circulatory collapse resulting in insufficient tissue perfusion and end organ damage is referred to as shock. Fluid loss from the circulating blood is the primary characteristic of circulatory shock. Volume , making imposible for all areas to receive enough circulation[3]

The clinical manifestation of shock results from a pathophysiologic change at the cellular level, which is caused by an insufficient supply or consumption of oxygen and metabolic substrates (such as glucose) to meet the energy requirements of the tissue .[4]

Shock is categorized as a pathophysiological state in which peripheral tissues fail to supply enough metabolic substrate (especially oxygen) or use it inappropriately. It can also be distributive, obstructive, cardiogenic, or hypovolaemic

Pathophysiology of shock

A state of circulatory insufficiency known as shock causes an imbalance between the tissues' demand for oxygen and its delivery, which can lead to end organmalfunction.Cellular shock initially affects the mitochondria The majority of aerobic energy is produced via substrate burning.

(fats and carbs) and oxygen, resulting in the formation of carbon dioxide and water. But stunned Cellular hypoxemia results in the tissues going into anaerobic state and building up lactic acid. Acidosis sets , and lactate begins to accumulate in the blood.

Serum lactic acid measurement can identify tissue hypoxemia. It is a trustworthy resource for prognostic and outcome prediction

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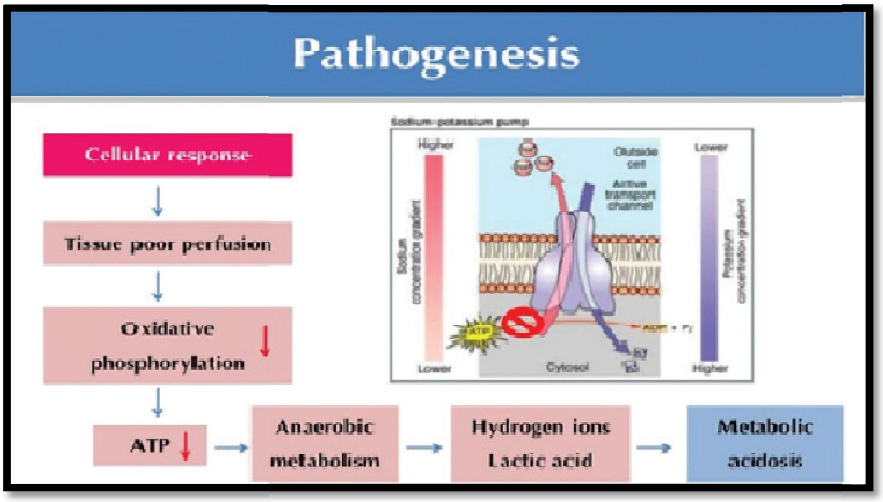

ETIOLOGY

The final manifestation of a complex list of etiologies, shock can be fatal if not treated promptly. There are four main broad categories of shock: distributive, hypovolemic , cardiogenic , and obstructive. The wide range of etiologies can contribute to each of these categories and are manifested by the final outcome of shock. Undifferentiated shock is the diagnosis of shock made, but the underlying etiology has not been found.

EPIDEMIOLOGY

After hypovolemic and cardiogenic shock, distributive shock is the most prevalent kind of shock. Shock that is obstructive occurs less frequently. Septic shock is the most prevalent kind of distributive shock, with a death rate ranging from 40 to 50%.[6]

Types of shock

- Hypovolemic Shock
- Cardiogenic Shock
- Obstrective Shock
- Distributive Shock

[1] Hypovolemic Shock

Hypovolemic shock is a state of insufficient organ perfusion resulting from an abrupt decrease of intravascular volume. Reduced macro- and microcirculation and a significant decline in ventricular preload have detrimental effects on tissue metabolism and can set off an inflammatory response.

A loss of intravascular fluid, typically whole blood or plasma, results in hypovolaemic shock. Whole blood loss: One clear reason for hypovolaemic shock is blood loss from an open wound

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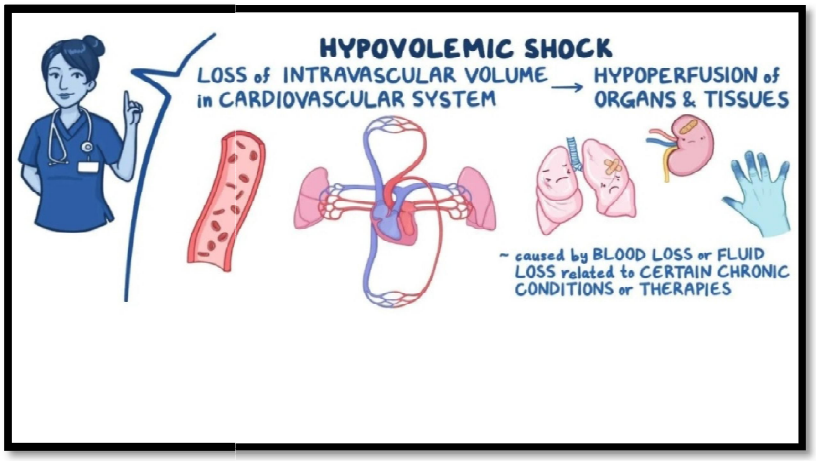


Symptoms

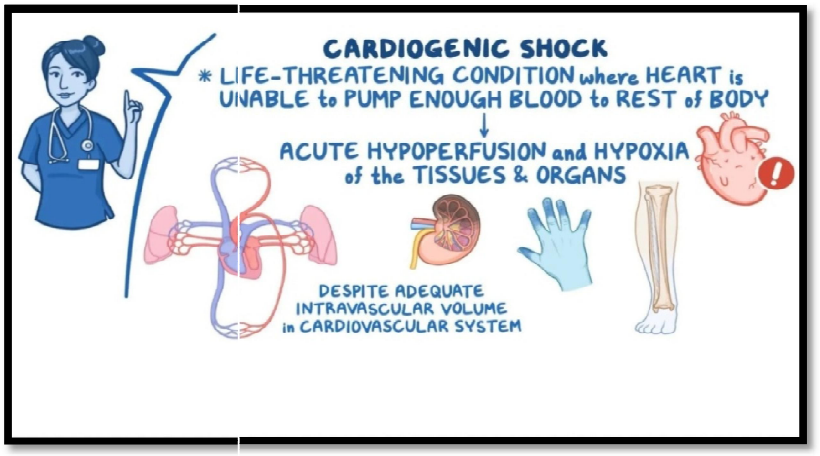
Abdominal or chest pain may arise from mesenteric and coronary ischemia caused by severe hypovolemic shock. Brain malperfusion can also cause agitation, sluggishness, or confusion.[9]

CAUSES

- Hemothorax, hemoperitoneum,
- long-bone fracture, pelvic fracture break
- bleeding in the retroperitoneu

[2] Cardiogenic Shock

A major loss in the heart's pumping ability, resulting from either diastolic or systolic dysfunction that lowers the ejection fraction or impairs ventricular filling, is the main cause of cardiogenic shock. Cardiogenic shock may occur with any disease that causes direct myocardial damage or otherwise inhibits the cardiac contractile meSchanism.[1]



Symptoms

- chest pen or pressure
- Coma
- Decreased urination
- Fast pulse [10]

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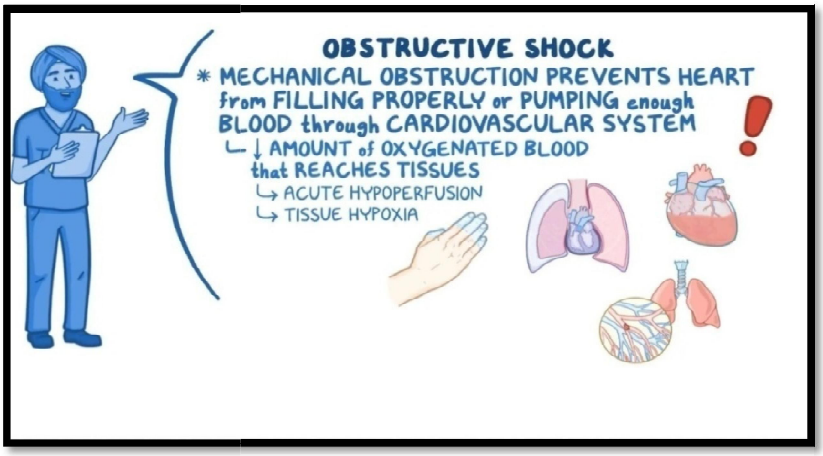
CAUSES

- cardiac bruising
- Inflammation of the heart muscle
- Weakened heart from any cause
- Infection of the heart muscle

[3] Obstrective Shock

The illness known as obstructive shock is brought on by blockage of the heart or major arteries. Obstructive shock must be separated from cardiogenic shock despite their similar symptoms since the two require different approaches to treatment.

Either a significant drop in preload or an increase in left ventricular outflow blockage causes obstructive shock. By reducing cardiac compliance and squeezing the inferior or superior vena cava, extracardiac processes that raise intrathoracic pressure can cause obstructive shock.



Symptoms

- Unusually fast breathing.
- Hypotension (low blood pressure).
- Tachycardia (fast heart rate).
- Altered consciousness.
- Very little pee output [11]

CAUSES

- Traumatic diaphragmatic breathing
- cardiac tamponade;
- tension pneumothoraxaa hernia physical assessment
- Heart and bilateral breath sounds lung imaging using thoracic ultrasonography swaying QUICK assessment

[4] Distributive Shock

Distributive shock is a state of relative hypovolemia resulting from pathological redistribution of the absolute intravascular volume and is the most frequent form of shock.[8]

Significant systemic vasodilation is a hallmark of distributive shock, which is frequently linked to relative intravascular volume depletion. the main compensatory reactions to lower systemic vascular resistance in distributive shock.[2]

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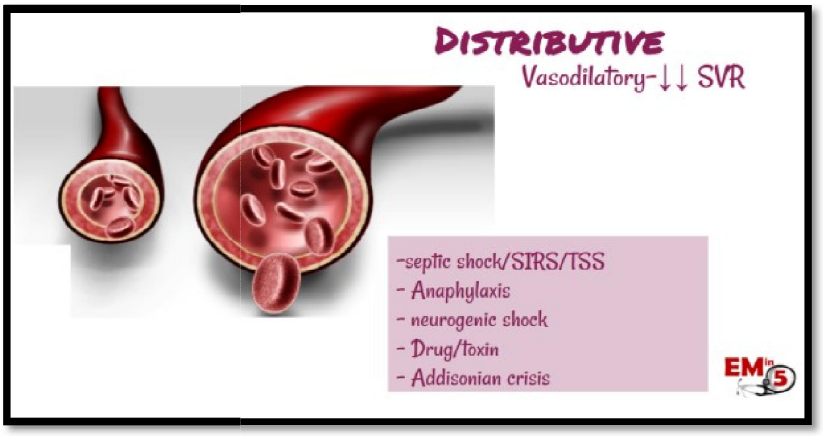

Symptoms

- Skin rash
- Fast heart rate and breathing
- Low blood pressure
- Warm arms and legs
- Chills
- Pain in your belly[11]

CAUSES[7]

- SIRS brought on by bleeding;
- Fat emboli syndrome;
- Neurogenic shock

DRUG USE IN THE TREATMENT OF SHOCK

[1] **Norepinephrine**

Most widely used vasopressor.
Potent α 1 agonist causing vasoconstriction in tissue beds.
Resultant increase in SVR causes rise in blood pressure.
Standard dose: 4 mg in 50 ml (0.08 mg/ml).

[2] Epinephrine

Nature's vasopressor.
Most commonly used during resuscitation cardiac arrest and anaphylaxis.
α1: Increases SVR. β1: Increases HR and myocardial contractility
β2: Bronchial smooth muscle relaxation.
Standard dose: 10 mg in 50 ml (0.2mg/ml).

[3]Dopamine

Vasopressor agent
Use in septic and cardiogenic shock.
Receptor activation varies according to dosage.
It is recommended for treating chronic CHF, sepsis, MI, trauma, and hypotension.

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[4] Doubutamine
An inodilator;
Synthetic catecholamine.
β1 stimulation: Boost cardiac contractility and heart rate.
Vasodilatation mediated by β2.
Dobutamine frequently causes MAP to decrease.
Vasodilatation normally requires NE to counteract it.

[5] Vasopressin
A peptide hormone secreted by the posterior pituitary that enhances water retention and DCT and CT permeability.(Receptor V2).
The smooth muscle of the arterial wall contains the V1 receptor, which when stimulated results in vasoconstriction and smooth muscle contraction.

[6] Adrenaline
When this medication is administered, it raises total peripheral resistance (TPR), lowers renal blood flow, and increases cardiac output.
Anaphylactic shock is managed with the use of adrenaline.

[7] Isoprenaline
It works by activating the β adrenergic receptor.
Both the heart and the periphery are affected
.Unlike adrenaline, it does not cause kidney vasoconstriction
It is beneficial for shock patients with elevated peripheral vascular resistance.

[8] Metaraminol
Its haemodynamic actions are similar to those of nor-adrenaline
Its duration of action is longer than that of nor-adrenaline. [5]

Treatment of shocks:
COMMON PATHWAY
Pathological process in which extreme failure of circulatory system by decrease blood volume cardiac output.
↓
Systemic hypotension decrease blood pressure tissue perfusion
↓
Decrease the oxygen supply (hypoxia)
↓
Reversible cellular injury
↓
Irreversible tissue injury
↓
Organ failure
↓
Death

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Hypovolemic shock

Decrease the volume
↓
Lose of blood/plasma/fluid
↓
Decrease circulating blood volume
↓
Decrease cardiac output
↓
Extreme and widespread failure of c.s.
↓
Continue

Clinical e,g
Burn,diarrhea,vomiting, accident
Hemorrhage

Cardiogenic shock :-

Abnormality of heart
↓
Myocardial damage/mesh abnormality of heart
↓
Low cardiac output
↓
Extreme
↓
Clinical example
↓
MI, artery vein, injury ,
↓
ventricular rupture
↓
ventricular arrhythmia

Anaphylactic shock:-

A release histamine whole body, form mast cells.
↓
Cause vasodilatation
↓
Decrease blood pressure
↓
Systemi hypotension

Clinical example:
IgE mediated hypersensitivity reactions
Histamine – chen – body – immune system,
– WBC – allergic reaction

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Neurogenic shock

Spinal cord injury neuronal damage
↓
Acute vasodilatation
↓
Decrease blood pressure

Septic shock

Due to serve sepsis with hypotension which can't be correct by infusing fluid
↓
Caused by gram positive bacteria
↓
Staphylococcus aurous
↓
Staphylococcus pneumonia
↓
Gram negative bacteria, fungi
↓
Ricketisiae
↓
Pathophysiology
↓
Bacterial infections
↓
Release of toxin
↓
Myocardial problem
↓
Decrease contraction
↓
Inadequate blood flow
↓
Tissue hypoxia
↓
Cell death

Mangement of shock:-

General principles of shock management:-

The overall goal of shock management is to improve oxygen delivery / utilisation in order to prevent cellular and oxygen injury.

Effective therapy requires treatment of underlying etiology.

Restoration of adequate perfusion , monitoring and comprehensive supportive care.

Interventions to restore perfusion centre, increasing cardiac output and optimisinf oxygen content.

Oxygen demand should also be reduced.

Physical Examination:-

Site of untreated infection

GI Hemorrhage on rectal examination.

Pulsus paradox and elevated JVPseen in cardiac tamponade

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Paucity of breath sounds, deviation of trachea away from the affected side, subcutaneous emphysema seen in tension pneumothorax

Diagnostic testing

BLOOD TESTS

Blood urea nitrogen (BUN),creatimine, transaminase evaluation show extent of end organ damage

Urine electrolytes, FE Na, FE urea indicate hypovolemia status

Increased WBC show infective process

Increased cardiac enzymes show primary cardiac problem

Blood cultures, urine cultures and sputum cultures should be obtained.

Lactate measurement

MONITORING

Echocardiography :- an echocardiogram shouldbe obtained in patients with suspected caediogenic shock.

Advanced hemodynamic monitoring :- recently new central venous catheter system linked with computer based algorithms provides continuous monitoring of hemodynamic parameters.

Cardiac catheterization and coronary angiography indicated in all patients with cardiogenic shock.

RESUSCITATION :-

Resuscitation should not be delayed in order to definitively diagnose the source of the shocked state.

If there is initial doubt of cause of shock, it is safer to assume the cause is hypovolemia. And begin fluid resuscitation.

Intravenous fluids should be given through short, wide bore catheters.

The oxygen carrying capacity of crystalloid and colloids is zero.

If blood is being lost, the ideal replacement is blood, although crystalloid blood products.

END POINDS OF RESUSCITATION :-

Traditionally, patients have been resuscitated until they have a normal pulse, BP, and urine output.

Occult hypoperfusion: state of normal vital signs and continued

With current monitoring techniques, occult hypoperfusion is manifested only by a persistent lactic acidosis and low mixed venous oxygen saturation.

Resuscitation algorithms directed at correcting global perfusion end points (base deficits, lactate, mixed venous oxygen saturation) rather than traditional end points have been shown to improve mortality and morbidity in high risk surgical patients.

MANAGEMENT OF NEUROGENIC SHOCK

Immobilization: if the patient has a suspected case of spinal cord injury.

High dose steroid to reduce inflammation

IV fluids: administration of IV fluids is done to stabilize the patient's blood pressure.

Inotropic agents such as dopamine may be infused for fluid resuscitation.

Atropine is given intravenously to manage severe bradycardia.

MANAGEMENT OF ANAPHYLACTIC SHOCK

During an anaphylactic attack, cardiopulmonary resuscitation (CPR) may be needed if breathing or heart beat is stopped.

Medications in Anaphylactic shock includes:

Epinephrine (adrenaline) to reduce body's allergic response.

Oxygen, which helps in breathing.

Intravenous anti histaminics and cortisone to reduce inflammation of air passages and improve breathing.

Beta agonist (e.g. Albuterol) to relieve breathing symptoms.

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MANAGEMENT OF HYPOVOLEMIC SHOCK

Fluid resuscitation

Maximise oxygen delivery ventilation

MANAGEMENT OF CARDIOGENIC SHOCK

Oxygen therapy

Morphine 5 to 10 mg

Reduce pain and anxiety associated with MI

Inotropic agent

Vasodilators

MANAGEMENT OF SEPTIC SHOCK

Corticosteroids

Dexamethasone

Hydrocortosone

Adrenergic agonist

Dopamine

dobotamine

nor epinephrine

epinephrine

Antibiotics

ciprofloxacin

Clindamycin

Leofloxacin

Diffrencial Diagnosis

Complete blood counts, basic serum chemistries (including renal function), and other tests (liver function tests, lipase/amylase, cardiac biomarkers, etc.) as indicated by the specific patient's circumstances are among the initial laboratory studies performed on patients in shock.

Most patients with suspected infections should have blood cultures performed; if clinically warranted, cultures of the urine, cerebral spinal fluid, pleural, ascitic, and/or other fluid compartments should also be sent.

Base deficit

A base deficit does not reliably indicate shock or an increased serum lactate content, even if it is linked to hypovolemia and poor tissue perfusion.. A retrospective trial of 16,305 trauma patients demonstrated that worsening base deficit was linearly associated with worsening injury severity scor

Chest x ray

Chest x-ray can assist in diagnosis of the etiology of shock. Special attention is paid to the heart size, presence of edema, infiltrates or effusions, and free air.While a chest x-ray can provide useful clinical data, it has limitations. For example, the absence of congestion on an initial chest x-ray does not exclude the diagnosis of acute decompensated heart failure.Furthermore, anteroposterior chest x-rays are particularly limited in that the posterior lungs are poorly visualized compared to posterior-anterior films.

Compound Tomography

Computed tomography (CT) is a generally accurate and noninvasive means of detecting internal pathology from various infections, vascular processes, or trauma. While CT imaging can be performed relatively rapidly, patients must

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travel from the ED to the CT scanner, which may be hazardous for unstable patients in shock. The potential benefits of CT, including diagnosing the etiology of shock and facilitating attempts at source control, must be weighed against possible risks associated with travel.

Echocardiography :-

Formal echocardiography may be useful in patients for whom cardiogenic shock is suspected, and who are not taken immediately to the catheterization laboratory or for patients with suspicion for aortic or pulmonary embolism. However, due to the time delays typically associated with formal echocardiography, it is generally not a component of the front-line evaluation of patients in shock presenting to the ED.

Other Diagnostics :-

Further diagnostic studies may also be indicated based on a patient's presentation. For example, lumbar puncture is indicated in patients with suspected meningitis after antibiotics have been administered; diagnostic paracentesis is indicated for patients with suspected spontaneous bacterial peritonitis; and magnetic resonance imaging is appropriate for suspected epidural abscesses.[7]

II. CONCLUSION

Shock is a state of global tissue hypoperfusion. After initial resuscitation, detailed physical examination is important to determine the cause of shock. Patients in shock have to be kept on monitored bed. Urine output and central venous pressure would need to be monitored in such patients

In trauma patients, hypovolemia is the main reason for shock. Control of hemorrhage and blood replacement are necessary. Hypovolemia can also develop due to gastroenteritis, heat stroke, febrile illness, etc. Septic shock needs early administration of antibiotics, after drawing a full septic workup. Cardiogenic shock needs to be treated meticulously and monitored closely. Inotropes and fluids have to be administered, cautiously. Neurogenic shock needs good vasopressor support. Obstructive shock whether it is tension pneumothorax or pericardial tamponade, both, need decompression. Pulmonary embolism needs to be treated with

anticoagulants. Anaphylactic shock can be managed by administration of parenteral epinephrine,crystalloids, steroids, and antihistamines

Management of shock is often complicated especially in extremes of age, pregnancy, and patients with multiple comorbidities

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