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Therapeutic I, 5th stage
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SHOCK

Shock is an acute state of inadequate perfusion of critical organs that can lead to death if therapy is not optimal.

The principal function of the circulatory system is to supply oxygen and vital metabolic compounds to cells throughout the body, as well as removal of metabolic waste products.

Classification of shock

Shock can be effectively categorized by etiology into four major types

A – **Hypovolemic shock** : results from decreased intravascular volume secondary to loss of blood or fluids and electrolytes. Occur in trauma , GI bleeding , Dehydration (Vomiting and diarrhea)

B- **Cardiogenic Shock :** results from cardiac failure with the resultant inability of the heart to maintain adequate tissue perfusion.

C- **Obstructive shock** : an acute decrease in cardiac output resulting from , e.g , Cardiac tamponade, tension pneumothorax, and massive pulmonary embolism

D- **Distributive or vasodilatory shock** : The reduction in systemic vascular resistance results in inadequate cardiac output and tissue hypoperfusion despite normal circulatory volume. It is categorized to(according to the cause)

1- **Septic shock** : it is typically secondary to bacteremia caused by such gram-negative organisms as *Escherichia coli, Klebsiella,*

Proteus, and *Pseudomonas*

2- Systemic inflammatory response syndrome (SIRS) . Defined as a systemic response to a nonspecific infectious or noninfectious insult—such as from burns, pancreatitis, ischemia or trauma.

3- Neurogenic shock : is caused by traumatic spinal cord injury or effects of an epidural or spinal anesthetic. This results in loss of sympathetic tone with a reduction in systemic vascular resistance and hypotension without a compensatory tachycardia.

4- Anaphylactic: shock : inappropriate vasodilatation triggered by an allergen (e.g. bee sting), often associated with endothelial disruption and capillary leak.

Pathophysiology

Shock results in failure of the circulatory system to deliver sufficient oxygen (O_2) to tissues despite normal or reduced O_2 consumption.
Shock may be caused by **intravascular volume deficit** (hypovolemic shock), **myocardial pump failure** (cardiogenic shock), or **peripheral vasodilation** (septic, anaphylactic, or neurogenic shock).

Fall in blood pressure (BP) is compensated by increased sympathetic outflow, activation of the renin–angiotensin system, and other factors that **stimulate peripheral vasoconstriction**. Compensatory vasoconstriction redistributes blood away from skin, skeletal muscles, kidneys, and gastrointestinal (GI) tract toward vital organs (eg, heart and brain) in attempt to maintain oxygenation, nutrition, and organ function.

Severe lactic acidosis often develops secondary to tissue ischemia and causes localized vasodilation, which further exacerbates the impaired cardiovascular state.

systemic inflammatory
response syndrome (SIRS) is characterized by inflammatory cytokines production by the body in response to ischemia, injury, or infection .

SIRS is clinically characterized by profound vasodilation, which impairs perfusion, and increased capillary permeability, which can lead to reduced intravascular volume. It is the hallmark of septic shock , a late manefistation of hypovolemic shock and uncommon in cardiogenic shock.

Regardless of etiology, the most distinctive clinical manifestations of hypovolemic shock are arterial hypotension and metabolic acidosis. Metabolic acidosis is a consequence of an accumulation of lactic acid resulting from tissue hypoxia and anaerobic metabolism. If the decrease in arterial blood pressure (BP) is severe and protracted, such hypotension will inevitably lead to severe hypoperfusion and organ dysfunction.

Clinical Presentation

Patients with hypovolemic shock may have, thirst , anxiousness, weakness , lightheadedness , dizziness , scanty urine output, dark yellow urine.

Signs of more severe volume loss include

Tachycardia (>120 beats/min) , **Tachypnea** (>30 breaths/min), **Hypotension** (SBP <90 mm Hg) , mental status changes or unconsciousness ,agitation. , Normal or low body temperature and cold extremities.

Biochemical changes include

A- Elevated Serum **Na^{+2}** and **Cl^{-}** concentrations

B- **(BUN): creatinine** ratio may be elevated initially but creatinine increases with renal dysfunction.

C- **Lactic acidosis** with decreased **bicarbonate** and **pH.**

D- **(CBC)** is normal in absence of infection.

E- In hemorrhagic shock, the **red cell count**, **hemoglobin** , and **hematocrit** will **decrease**.

Diagnosis and Monitoring

1- Noninvasive and invasive monitoring

2- Evaluation of medical history.

3- Clinical presentation.

4- Laboratory findings.

Pulmonary artery (Swan–Ganz) catheter is an invasive method can be used to determine central venous pressure (CVP) , pulmon ary artery pressure (PAP) , cardiac output (CO) and pulmonary artery occlusion pressure (PAOP).

Renal function can be assessed grossly by hourly measurements of urine output.

O_2 delivery and consumption

In normal individuals, O_2 consumption (Vo_2) is dependent on O_2 delivery (Do_2) (Vo_2 flow dependency).(1)

At certain level , increases in Do_2 will not alter Vo_2 (flow independency). In critically ill patients there is a continuous, pathologic dependence relationship of Vo_2 with Do_2.

These indexed parameters are calculated as

$$\boldsymbol{Do_2 = CI \times (CaO_2)}$$

$$\boldsymbol{Vo_2 = CI \times (CaO_2 - CvO_2)}$$

where CI = cardiac index, Cao_2 = arterial O_2 content, and Cvo_2 = mixed venous O_2 content.

The **VO_2:DO_2 ratio** (O_2 extraction ratio) can be used to assess adequacy of perfusion and metabolic response.

Patients who can **increase** Vo2 when Do2 is increased are more likely to **survive. Low** Vo2 and O2 extraction ratio values indicate **poor** O2 utilization and lead to **greater mortality.**(

Treatment

Goals of Treatment: The goal during resuscitation from shock is

1- To **achieve** and **maintain** mean arterial pressure (MAP) above ***65 mm Hg*** while ensuring adequate perfusion to critical organs.

2- **Prevent** further disease progression with subsequent organ damage.

3- **To reverse** organ dysfunction that has already occurred.

General Approach

1- Initiate supplemental O_2 at the earliest signs of shock.

2- Fluid resuscitation to maintain circulating blood volume .

If fluid administration does not **achieve desired end points**, pharmacologic support is necessary with **inotropic** and **vasoactive** drugs.

3- Supportive care measures include assessment and management of pain, anxiety, agitation, and delirium.

Fluid resuscitation for hypovolemic shock

1-Crystalloid solutions (**0.9% sodium chloride** or **lactated Ringer solution**) are the initial fluids of choice.

The choice between normal saline and lactated Ringer solution is based on **clinician preference** and **adverse effect concerns.**

Advantage of Crystalloids are :

A- Can be rapidly and easily administered

B- Compatible with most drugs

C- Have low cost.

Their disadvantages include :

The need to use large fluid volumes , the possibility that dilution of oncotic pressure may lead to pulmonary edema.

Crystalloids are administered at a rate of **500 to 2000 mL/h**, depending on **severity of the deficit**, **degree of ongoing fluid loss**, and **tolerance to infusion volume**.

Usually **2 to 4 L** of crystalloid normalizes intravascular volume.

Colloids

Such as : Hydroxyethyl starch , **dextran**, **albumin**

Colloids possess the theoretical advantage of prolonged intravascular retention time .It have been associated with fluid overload, renal dysfunction , and bleeding. Most of colloids are expensive.

In 2013 , FDA concluded that hydroxyethyl starch is associated with increased mortality and renal injury.

Blood products

(**whole blood**, **packed RBC**, **fresh frozen plasma**, **platelets**)

Maintain O_2-carrying capacity, as well as clotting factors and platelets for blood hemostasis.

Blood products may be associated with

A- Transfusion-related reactions

B- **virus transmission**

C- Hypocalcemia resulting from added citrate

D- Increased blood viscosity from supranormal hematocrit elevations

E- **Hypothermia** from failure to appropriately warm solutions before administration.

Pharmacological therapy

Hypovolemic shock:

Body increase CO and peripheral resistance to maintain BP.If fluid therapy is inadequate , Inotropic agent is given , e.g **Dobutamin** .

Vasopressors (e.g **Norepinephrine**) are only used if the above measures failed

Septic shock

Initial hemodynamic therapy is administration of IV fluid **(30 mL/kg of crystalloid).**

Norepinephrine is the preferred initial vasopressor if no responding to fluid administration.

Epinephrine may be added in cases where there is suboptimal hemodynamic response to norepinephrine.

Phenylephrine may be tried as the initial vasopressor in cases of severe tachydysrhythmias.

Dobutamine is used in low CO states despite adequate fluid resuscitation pressures.

Vasopressin may be considered as adjunctive therapy in patients who are refractory to catecholamine vasopressors despite adequate fluid resuscitation.

Potent vasoconstrictors such as norepinephrine and phenylephrine should be given through **central veins** because of possibility of **extravasation** and **tissue damage** with peripheral administration.

1-Norepinephrine : first-line therapy for septic shock . It effectively increases MAP. It has strong α1-agonist activity and less potent β1-agonist effects , weak vasodilatory effects of β2-receptor stimulation.

Norepinephrine infusions are initiated at 0.05 to 0.1 mcg/kg/min and rapidly titrated to preset goals of MAP (usually at least 65 mm Hg)

2 - **Phenylephrine** : it is pure α1-agonist, improves MAP by increasing cardiac index , Enhance venous return to the heart (increase in CVP and stroke index) and by acting as a positive inotrope.

Phenylephrine 0.5 to 9 mcg/kg/min, used alone or in combination with dobutamine or low doses of dopamine.

Tachydysrhythmias, are infrequent with phenylephrine, particularly when it is used as a single , because it does **not have β1-adrenergic agonist activity**

3 – **Epinephrine** : It has combined α- and β-agonist effects.It combine vasoconstrictor and inotropic effects , associated with tachydysrhythmias and lactate elevation.

Younger patients appear to respond better to epinephrine, possibly because **of greater β-adrenergic reactivity**

4 **-Dopamine**

It is not as effective as norepinephrine and epinephrine for achieving goal MAP. Improving contractility and heart rate, primarily from its β1 effects. It increases MAP and SVR as a result of both increased CO and, at higher doses, its α1 agonist effects.

The clinical utility of dopamine is limited **because large dosages are frequently necessary** to maintain CO and MAP. Its clinical use frequently is hampered by **tachycardia** and **tachydysrhythmias**

5- Dobutamine : is an inotrope with vasodilatory properties. It should be started at dosages ranging from 2.5 to 5 mcg/kg/min

Evaluation of therapeutic outcome (1)

1- Vital signs, urine output, mental status, and physical examination.

2- Reserve pulmonary artery catheterization for complicated cases of shock not responding to conventional fluid and medication therapies.

3- Laboratory tests : CBC , BUN: s.creatinin , PT &PTT , s. Lactate

4- Successful fluid resuscitation should increase **SBP (>90 mm Hg), CI (>2.2 L/min/m2),** and **urine output (0.5–1 mL/kg/h).**

5- MAP of **greater than 65 mm Hg** should be achieved to ensure adequate cerebral and coronary perfusion pressure.

6- Patient receiving blood : PT, INR