

- ▣ Physiological Response to Hypovolemic Shock
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- ▣ Objectives

At the end of the session the students should be able to:

- List causes of shock including hypovolemic shock
- Describe compensatory mechanism of shock
- Describe mechanism of non progressive shock
- Describe mechanism of irreversible shock
- Correlate this knowledge to the management of shock.

▣ Definition of shock

Circulatory shock means generalized inadequate blood flow through the body; to the extent that the body tissues are damaged, especially because of too little oxygen and other nutrient delivery to the tissues (Guyton)

- ▣ Inadequate perfusion (oxygen supply) of tissues, resulting in:
 - Organ dysfunction
 - Cellular and organ damageAnd if not quickly corrected...
 - Death

- ▣ Shock is a process that causes the eventual shutdown of all body systems in a systematic order
- ▣ Amount of time for shock to progress varies from patient to patient
- ▣ Is related to the body's overall health and ability to compensate for its deficiencies
- ▣ As the syndrome progresses, the process speeds up
- ▣ The circulatory system fails to provide adequate blood to the tissues, resulting in cellular hypoxia and death

▣ Types of Shock

- Hypovolemic
- Cardiogenic

- Obstructive
- Distributive
- ▣ Causes of Hypovolemic Shock
 - Bleeding (hemorrhage)
 - Loss of plasma
 - burns
 - exfoliative dermatitis
 - Intestinal obstruction
 - Loss of fluid and electrolytes
 - diarrhoea
 - vomiting
 - polyuria
 - excessive sweating
- ▣ Hypovolemic Shock
- ▣ Signs and symptoms:
 - Mental status: lethargy, sleepy,
 - Skin: cold and pale.
 - Blood pressure: begins to fall.
 - Pulse: rapid and weak

- Respirations: rapid and shallow
- Other: decreased urination
- ▣ Cardiogenic Shock

Heart pump failure

- Myocardial infarction
- Cardiac trauma
- Cardiomyopathy
- Congestive heart failure
- Cardiac arrhythmias

▣ Obstructive Shock

It occurs when cardiac output is decreased as a result of mechanical obstruction of left or right ventricular filling

Causes

- Tension pneumothorax with kinking of the great veins
- Massive pulmonary emboli

- Cardiac tamponade (bleeding into the pericardium with external pressure on heart)

▣ Distributive Shock

- Also known as vasogenic shock, low resistance shock or warm shock
- It itself includes three types of shock
 - Neurogenic shock
 - Anaphylactic shock
 - Septic shock
- **Neurogenic shock**
the *vascular capacity* increases so much that even the normal

amount of blood becomes incapable of filling the circulatory system adequately.

One of the major causes of this is *sudden loss of vasomotor tone* throughout the body, resulting especially in massive dilation of the veins.

Causes

- Deep general or spinal anaesthesia
- Brain damage
- Vasovagal syncope

- **Anaphylactic shock**
- It is a rapidly developing severe allergic reaction.

- It occurs when an individual who has previously been sensitive to an antigen is re-exposed to it.
- One of the principal effects is to cause the *basophils* in the blood and *mast* cells in the pericapillary tissues to release *histamine* or a *histamine-like substance*

The histamine causes

- (1) *an increase in vascular capacity*
 - (2) *dilation of the arterioles*
 - (3) *greatly increased capillary permeability,*
- **Septic shock**

This refers to a bacterial infection widely disseminated to many areas of the body, with the infection being borne through the blood from one tissue to another and causing extensive damage. Most of the effects are brought about by endotoxins released by gram negative bacteria.

▣ Stages of Shock

- ▣ Non progressive/Compensated shock ---- body is able to compensate and maintain tissue perfusion
- ▣ Progressive shock---- body begins to lose its ability to compensate--- inadequate perfusion begins

- ▣ Irreversible shock---cell and tissue damage result in multi-system organ failure
- ▣ **Nonprogressive/Compensated shock**
- ▣ Sympathetic reflexes and other factors compensate enough to prevent further deterioration of circulation
- ▣ The factors that cause a person to recover from moderate degree of shock are all negative feedback control mechanisms of circulation. These include the following
 - Baroreceptor reflex
 - CNS ischemic response
 - Reverse stress-relaxation of the circulatory system

- Increased secretion of renin by the kidneys and formation of angiotensin II (constriction of peripheral arteries, and decreased output of salt and water)
- Increased secretion by posterior pituitary gland of vasopressin (ADH) :it constricts the peripheral arteries and veins and greatly increases the water retention by the kidneys.
- Increased secretion by adrenal medulla of epinephrine and norepinephrine

- Compensatory mechanism that return the blood volume back towards normal
 - ▣ *Sympathetic reflexes* and increased secretion of *catecholamine* by adrenal medulla get maximally activated *within 30 seconds* to a few minutes after hemorrhages.
 - ▣ The angiotensin - vasopressin mechanisms and reverse stress relaxation require 10minutes to 1 hour to respond completely.
 - ▣ Readjustment of blood volume may require 1 to 48 hours.

Sympathetic reflex compensations **in shock**

- ▣ Especially important in *maintaining Arterial Pressure*
- ▣ Powerful sympathetic reflexes initiated mainly by arterial baroreceptors and other vascular stretch receptors.
- ▣ *Three important effects*
 - Constriction of arterioles in most of systemic circulation thereby increasing TPR
 - Constriction of venous reservoir increases venous return despite reduced blood loss.
 - Increase in heart rate

Value of the sympathetic nervous reflexes

- ▣ In absence of sympathetic reflexes the body can tolerate only 15-20% of blood loss in contrast to 30-40% when the sympathetic system is intact.
- ▣ Sympathetic reflexes are geared more for maintaining arterial pressure than for maintaining cardiac output.
- ▣ Sympathetic reflexes is brought about by increase of TPR and constriction of veins to keep venous return and cardiac output from falling too low.
- ▣ The second plateau phase of is due to CNS ischemic response.

This leads to extreme stimulation of sympathetic nervous system. This can also be called *last ditch stand* of the sympathetic nervous reflexes to keep arterial pressure from falling too low.

Protection of coronary and cerebral blood flow by reflexes

- ▣ The sympathetic nervous reflexes do not cause significant constriction of either the cerebral or cardiac blood vessel. In addition in these areas local blood flow autoregulation is excellent.
- ▣ In these areas blood flow is maintained until arterial blood pressure falls below 70mmHg.
- ▣ **Progressive shock –vicious circle of cardiovascular deterioration**

- ▣ There are some positive feedbacks that cause shock to become progressive. Some of these are mechanisms are
 - **Cardiac depression:** when arterial blood pressure falls low enough, the myocardium itself gets damaged due to reduced blood supply in the coronaries. This weak heart muscle leads to further reduction in cardiac output.
 - **Blockage of very small blood vessels-Sludged Blood:**
Since tissue metabolism continues to occur despite the sluggish blood flow, large amount of acids like carbonic acid and lactic acid are dumped in to the blood. This along with products from ischemic

tissue cause blockage of small blood vessels.

- **Increased capillary permeability.**

Caused by capillary hypoxia.

- **Release of toxins by ischemic tissues**

Histamine, serotonin, tissue enzymes .

- **Cardiac depression caused by endotoxins:**

Released from dead gram negative bacteria in the intestines. This causes increased cellular metabolism despite inadequate nutrition. Major role in septic shock

- **Generalised cellular deterioration:**

Occurs throughout the body but the organ worst effected is the liver because of its exposure to vascular toxins. The major effects occurring are:

- Na^+/K^+ transport is diminished. Sodium chloride accumulates inside the cell and potassium is lost. The cell begins to swell.
- Mitochondrial activity is depressed
- Lysosomes release hydrolases causing intracellular deterioration.

- **Tissue necrosis in severe shock-**
patchy areas of necrosis
- **Acidosis in shock-**

due to excess lactic acid and retention of carbon dioxide in tissues

All positive feedback does not necessarily lead to vicious cycle. Whether a vicious cycle develops depends on intensity of the positive feedback.

▣ **Irreversible shock**

At this stage of shock, transfusion or any other form of therapy is incapable of saving the person's life.

Also known as refractory shock.

Depletion of the cellular high energy phosphate reserves.

▣ **Physiology of Treatment in Shock**

Replacement Therapy

- Blood and Plasma Transfusion
- Dextran Solution as a Plasma Substitute

Treatment of Shock with

Sympathomimetic Drugs-

Sometimes Useful, Sometimes Not Useful in neurogenic and anaphylactic shock.

Sympathomimetic drugs have not proved to be very valuable in hemorrhagic shock

Other Therapy

- Treatment by the Head-Down Position
- Oxygen Therapy
- Treatment with Glucocorticoids

- Glucocorticoids frequently increase the strength of the heart in the late stages of shock;
- Glucocorticoids stabilize lysosomes in tissue cells
- Glucocorticoids might aid in the metabolism of glucose by the severely damaged cells.

▣ Summary

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THANKS...

