# Shock

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# What is Shock?

- Shock is a condition in which the metabolic needs of the body are not met because of an inadequate cardiac output.
- If tissue perfusion is quickly restored, cellular injury may be limited, but if treatment is delayed shock may become irreversible with cellular injury and metabolic dysfunction becoming widespread.
- Shock can occur with a normal blood pressure and hypotension can occur without shock

# AETIOLOGY OF SHOCK

**(1) *Inadequate circulating blood volume***

- Develops from intravascular loss of salt and water, loss of plasma or loss of blood.
- Results in depletion of various capacitance bed and decreased ventricular-end-diastolic
- volume stroke volume and cardiac output.

# AETIOLOGY OF SHOCK (Contd)

(2***) Loss of Autonomic control of the vasculature***.

- Develops from spinal cord injuries, regional anaesthetics or administration of autonomic blocking agents.
- Results in expansion of the vasculature, which leads to inappropriate perfusion causing pooling of blood in vascular beds that do not need it at the expanse of those that do.

# AETIOLOGY OF SHOCK (Contd)

***(3) Impaired cardiac function***

- Develops from compression of the heart or intrinsic abnormality of the heart or obstruction of either the pulmonary or systemic vasculature.
- Results in decreased ventricular end-diastolic volume and cardiac output.

# AETIOLOGY OF SHOCK

***(4) Vascular Impairment***.

- Develops when endogenous or exogenous vasoactive metabolites are released into the circulation.
- This causes a reduced arteriolar and venous vasomotor tone
- Included in this category are shock associated with the Systemic Inflammatory Response syndrome (SIRS) or sepsis, anaphylaxis, adrenocortical insufficiency, and traumatic injuries.

# Understanding Shock

- Inadequate systemic oxygen delivery activates autonomic responses to maintain systemic oxygen delivery
  - Sympathetic nervous system
    - Norepinephrine, epinephrine, dopamine, and cortisol release
      - Causes vasoconstriction, increase in HR, and increase of cardiac contractility (cardiac output)
  - Renin-angiotensin axis
    - Water and sodium conservation and vasoconstriction
    - Increase in blood volume and blood pressure
  - ADH
    - Water and sodium conservation and retention

**Goal is to maintain cerebral and cardiac perfusion:**

- Vasoconstriction of splanchnic, musculoskeletal, and renal blood flow

# Understanding Shock

- Cellular responses to decreased systemic oxygen delivery
  - ATP depletion → ion pump dysfunction
  - Cellular edema
  - Hydrolysis of cellular membranes and cellular death

# Understanding Shock

Global Tissue Hypoxia

- Endothelial inflammation and disruption
- Inability of O2 delivery to meet demand

- Result in:
  - Lactic acidosis
  - Cardiovascular insufficiency
  - Increased metabolic demands

Leads to systemic metabolic lactic acidosis that overcomes the body's compensatory mechanisms

# Understanding Shock

**Multiorgan Dysfunction Syndrome (MODS)**

- Terminal phase of Shock
- Occurs when effective treatment is not instituted immediately, and failure of homeostatic mechanism ensues
  - Cardiac depression
  - Respiratory distress
  - Renal failure
  - DIC
- Result is end organ failure

# TYPES OF SHOCK
# (1) Inadequate circulating blood volume

**Hypovolemic shock**

- The losses are primarily external
- Can be due to haemorrhage (haemorrhagic shock), loss of fluid into the gut, or as a result of vomiting/diarrhoea and dehydration (Non-haemorrhagic hypovolemic shock).

# TYPES OF SHOCK
# (1) Inadequate circulating blood volume

**Vascular Impairment**.

- **(i) Traumatic shock**
  - Initial loss of blood or plasma externally.
  - Later, there is internal loss of fluid arising from wound produced inflammatory mediators and disrupted endothelium.

# TYPES OF SHOCK
# (1) Inadequate circulating blood volume

**Vascular Impairment**.

- **(iii) Septic shock**
  - Due to inflammatory mediators and disruption of endothelium, there is a loss of plasma into the tissue primarily. The early stages are characterized by fever and cutaneous vasodilatation.
  - Later followed by the classical signs of shock.

# TYPES OF SHOCK
# (2) Loss of autonomic control of vasculature.

- Neurogenic shocks may be due to spinal cord injury, severe head injury, or spinal anesthesia leading to a loss of vascular tone in the tissues supplied by the nerves.

# TYPES OF SHOCK
# (3) Impaired cardiac Function

**This can be intrinsic or extrinsic.**

- **Intrinsic** mechanisms of cardiac dysfunction leading to shock (***Cardiogenic shock)*** include myocardial infarction, cardiomyopathy, valvular heart disease, cardiac rhythm disturbances, and myocardial depression from drug toxicity or trauma.

# TYPES OF SHOCK
# (3) Impaired cardiac Function

- **Extrinsic** mechanisms of cardiogenic shock produce cardiac dysfunction by compressive or obstructive means.
- *Cardiac compressive shock* may be due to pericardiac tamponade or tension  pneumothorax which may cause external compression of the heart, which impedes diastolic filling and decreases cardiac output.
- *Cardiac obstructive shocks* are due to pulmonary embolism or systemic arteriolar constriction causing an increased vascular resistance, thereby limiting systolic ejection.

# CLINICAL FEATURES.

**HYPOVOLEMIC SHOCK**

- The symptoms and signs are progressively due to:
  - (i) Sign of adrenergic discharge to the skin.
  - (ii) Oliguria
  - (iii) Cardiac or/and Neurologic dysfunction.

# CLINICAL FEATURES.

## HYPOVOLEMIC SHOCK

**Mild Shock (<20% of blood volume)**

- Clinical features are those of increased adrenergic discharge to the skin.
  - Cool and clammy skin
  - Collapsed subcutaneous veins
  - Sweating (occasionally)
- Thirst.
- BP is normal in the supine position.
- Urinary output is normal, but urine  may be concentrated

# CLINICAL FEATURES.

## HYPOVOLEMIC SHOCK

- **Moderate Shock** (20-40%) (due to effect of vasopressin and aldosterone).
  - Oliguria
  - Plus the other signs of mild shock
  - Supine B.P. is usually normal
- **Severe Shock** (>40%) due to cardiac and neurologic dysfunction
  - Low B.P.
  - Change in the sensorium (Restlessness, stupor or unconsciousness.

# CLINICAL FEATURES.

## TRAUMATIC SHOCK

- Initial signs and symptoms are due to external blood loss as in hypovolemic shock.
- But the major consequences are not shown until 24-48hrs by which the inflammation mediated vascular permeability would have developed.
- This aggravates pre-existing hypovolemia due to a generalized increase in systemic vascular permeability.

# CLINICAL FEATURES.

## SEPTIC SHOCK

- Initial manifestation is due to attempt by the body to dissipate excessive heat produced by the hypermetabolic state viz a viz warm, pink and sweaty skin (early or red shock)
- Later hypoperfusion of vital organs activate cutaneous pressure mechanisms leading to the classical hypovolemic signs. There is a fall in skin temp which progresses to chills.

# CLINICAL FEATURES.

## SEPTIC SHOCK

- The cutaneous vasoconstriction compromises the body's inability to dissipate heat and loads to another rapid rise in body temperature, which in turn leads to cutaneous vasodilatation.
- Terminally, there is widespread disruption of vascular endothelium leading to hypovolemic and reduction in cardiac output ("white or late septic shock).

# CLINICAL FEATURES.

**NEUROGENIC SHOCK**

- History of spinal injury, regional anaesthetist or autonomic blocker administration.
- The skin of the denervated skin is warm and pink.
- BP is low.
- Heart Rate is rapid (or else slow if the adrenergic supply to the heart is block
- Heat loss can be considerable

# CLINICAL FEATURES.

## CARDIAC COMPRESSIVE SHOCK

- Distended neck veins in injured patients with features consistent with hypovolemic shock are diagnostic of cardiac compressive shock.
- Clinical features of cardiac tamponade or tension pneumothorax.

# CLINICAL FEATURES.

## CARDIAC OBSTRUCTIVE SHOCK

- Produces mechanical or functional obstruction that results in increased left ventricular or right ventricular after loading causing failure.
- Coarctation of the aorta and malignant hypertension are examples of obstructive causes of increased left ventricular afterload, whereas pulmonary embolism and various other causes of pulmonary hypertension led to increased right ventricular afterload and right ventricular failure.

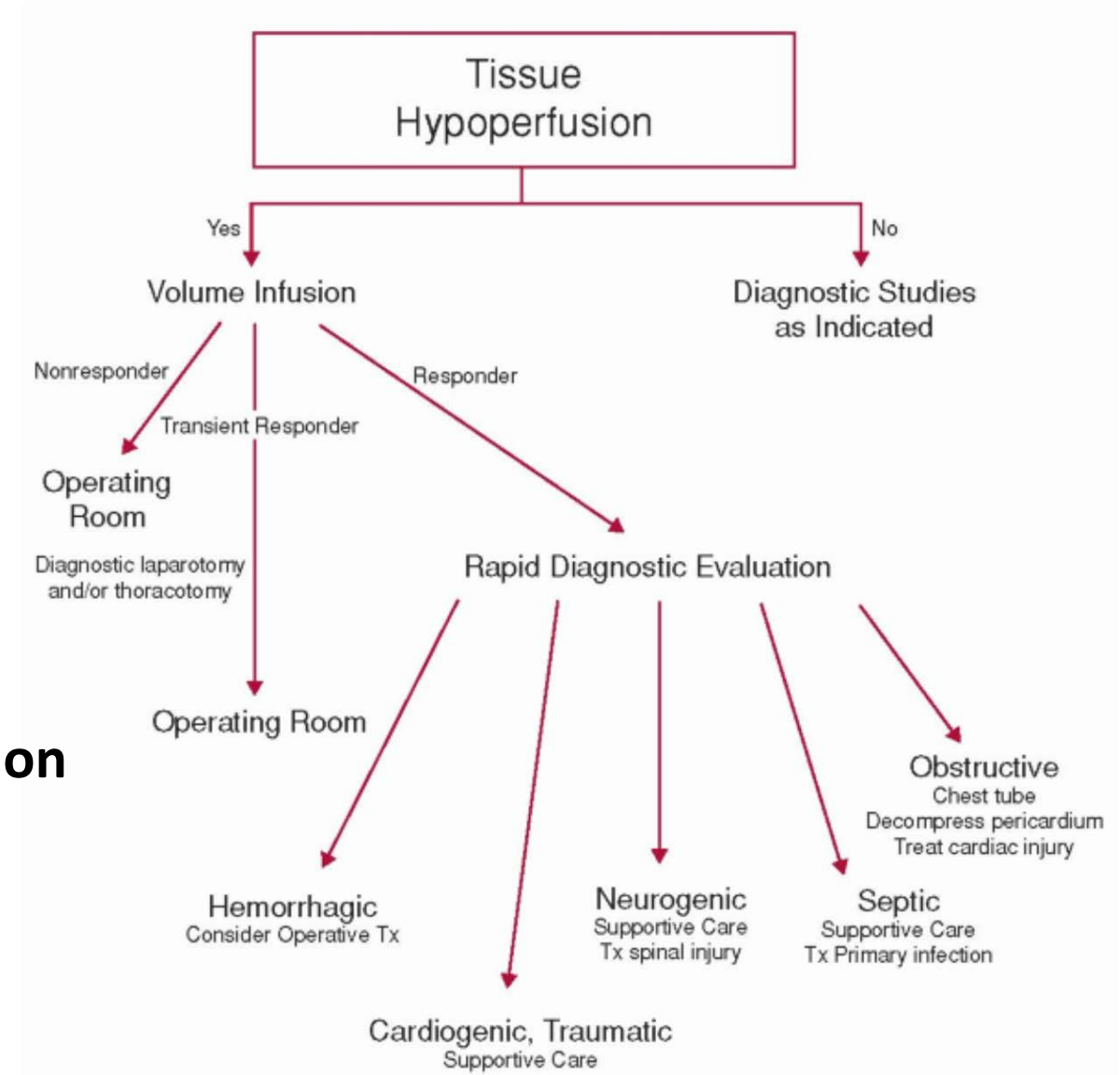
# CLINICAL FEATURES.

## CARDIOGENIC SHOCK

- Feature of cardiac dysfunction co-existing with shock symptoms.
- Common in the elderly because of increasing incidence of cardiac diseases in this age group

# TREATMENT.

**An Algorithm for Managing Shock**




**Note the types of responders to Resuscitation**

1. Responder
2. Transient Responder
3. Non-Responder

# TREATMENT.

**General:**

**RESUSCITATE!**

**ABC of Resuscitation**

- Airways maintenance
- Breathing
- Analgesia
- Cover the patient with blanket to prevent hypothermia.
- Supplementary oxygen therapy

# SPECIFIC TREATMENT: Hypovolemic Shock

- Infusion of crystalloids
- Blood infusion in haemorrhagic shock.
- Identify ongoing blood loss
- Stop ongoing blood loss
- Monitor!

# SPECIFIC TREATMENT: Septic Shock

- Best prevented.
- Infusion of crystalloid
- Antibiotics.
- Monitor

# SPECIFIC TREATMENT: Neurogenic Shock

- Infusion of crystalloids
- Vasoconstrictor drugs e.g., Norepinephrine
- Elevation of the part of the body that is involved.

# SPECIFIC TREATMENT: Cardiogenic Shock

- Optimize the heart rate
- Vasodilators e.g., dopamine
- May require cardiac surgery.
- **For Cardio-obstructive shock**: Treat the Underline Cause

# SPECIFIC TREATMENT: Monitoring

- Serial haematocrit
- Hourly urinary output monitoring
- Intra-arterial catheters to monitor arterial BP and arterial blood gases.
- Swanz-Ganz catheters to monitor the arterial wedge pressure

# Organ failure syndromes of shock
# (1) LUNG

- Most of the late deaths after an initial successful resuscitation from shock are caused by respiratory failure.
- Usually due to respiratory distress syndrome.
- May become manifested within 24hrs of injury.
- Early signs are tachypnea and increased respiratory effort.
- Underlying cause is due to widespread inflammation of coagulopathy leading to microvascular damage.

# Organ failure syndromes of shock
# (2) OTHERS

- **KIDNEYS**
  - In shock, blood shunts to the medulla and bypasses the cortex resulting in cortical necrosis leading to renal failure if prolonged.
- **LIVER**
  - Shock induced liver failure is usually manifested by elevation of serum bilirubin and liver enzymes.

# Organ failure syndromes of shock
# (3) OTHERS

- **GIT**
  - Ischaemic damage of the mucosa occurs (called stress ulcer in the stomach).
  - This may allow bacterial endotoxin to reach the circulation.
- **ADRENALS**
  - Acute adrenal insufficiency can result from haemorrhagic necrosis of the adrenals in leading to refractory shock.

# The End

THANK YOU