Shock

By

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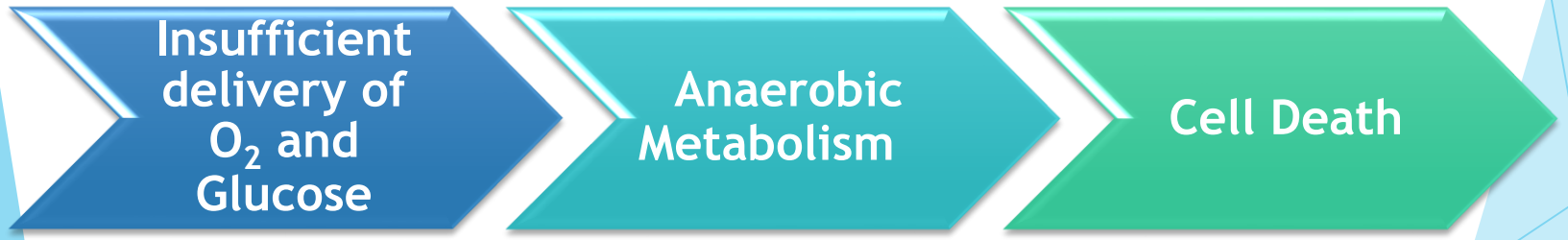
MBChB FIBMS

SHOCK

- a systemic state of low tissue perfusion, which is inadequate for normal cellular respiration.

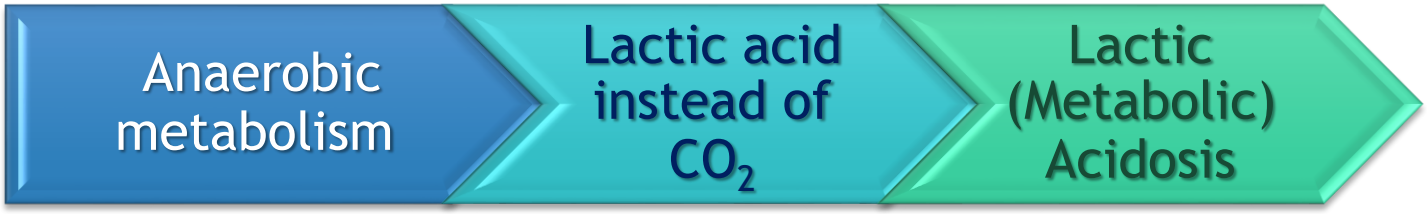


SHOCK

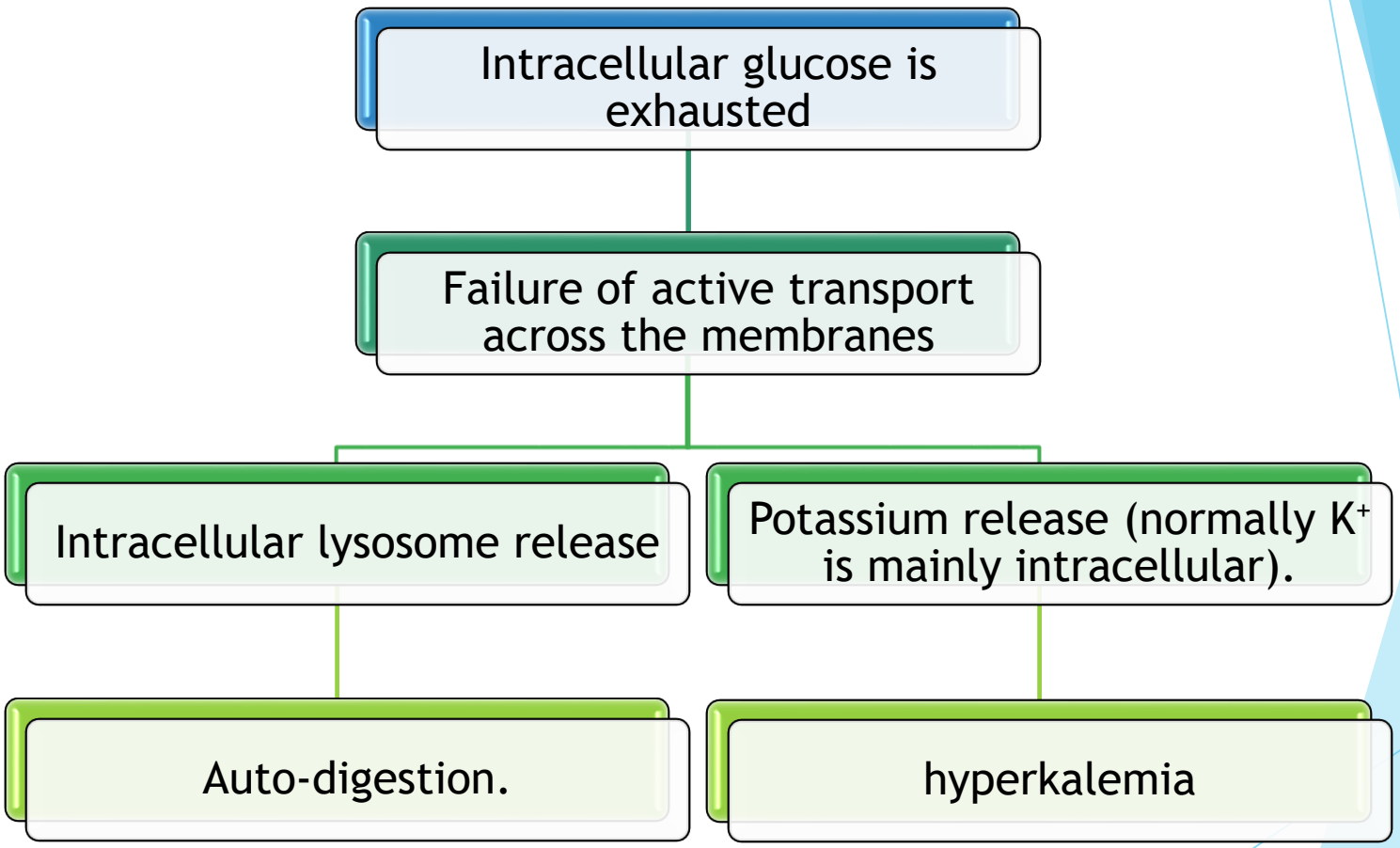
- The most important cause of death in surgical patients.
- Death in shock could be:
    1. Rapid death > profound shock.
    2. Delayed death >
        - Consequences of organ ischaemia.
        - Ischaemia-Reperfusion injury.

PATHOPHYSIOLOGY

Cellular

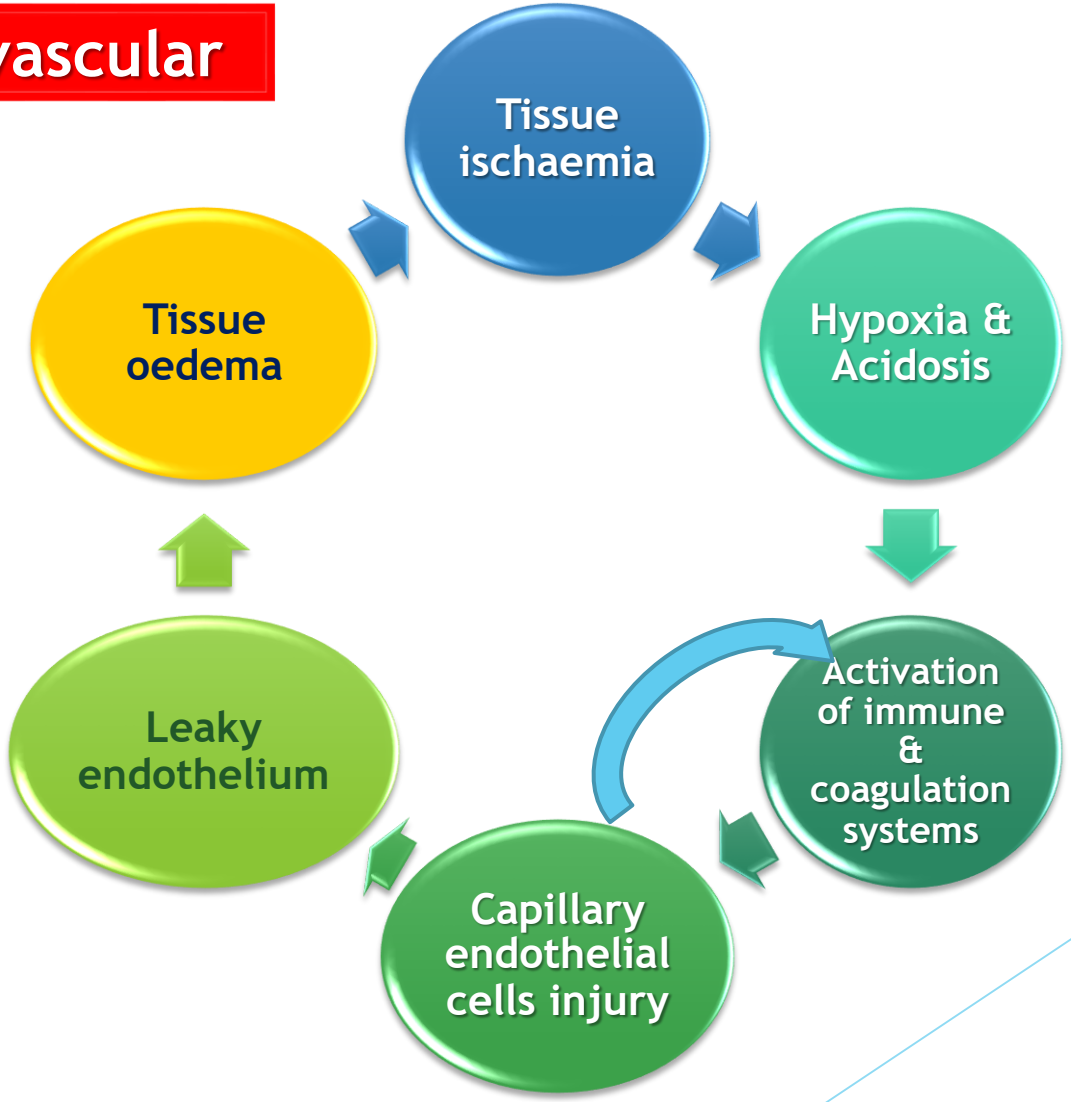


PATHOPHYSIOLOGY



PATHOPHYSIOLOGY

Microvascular



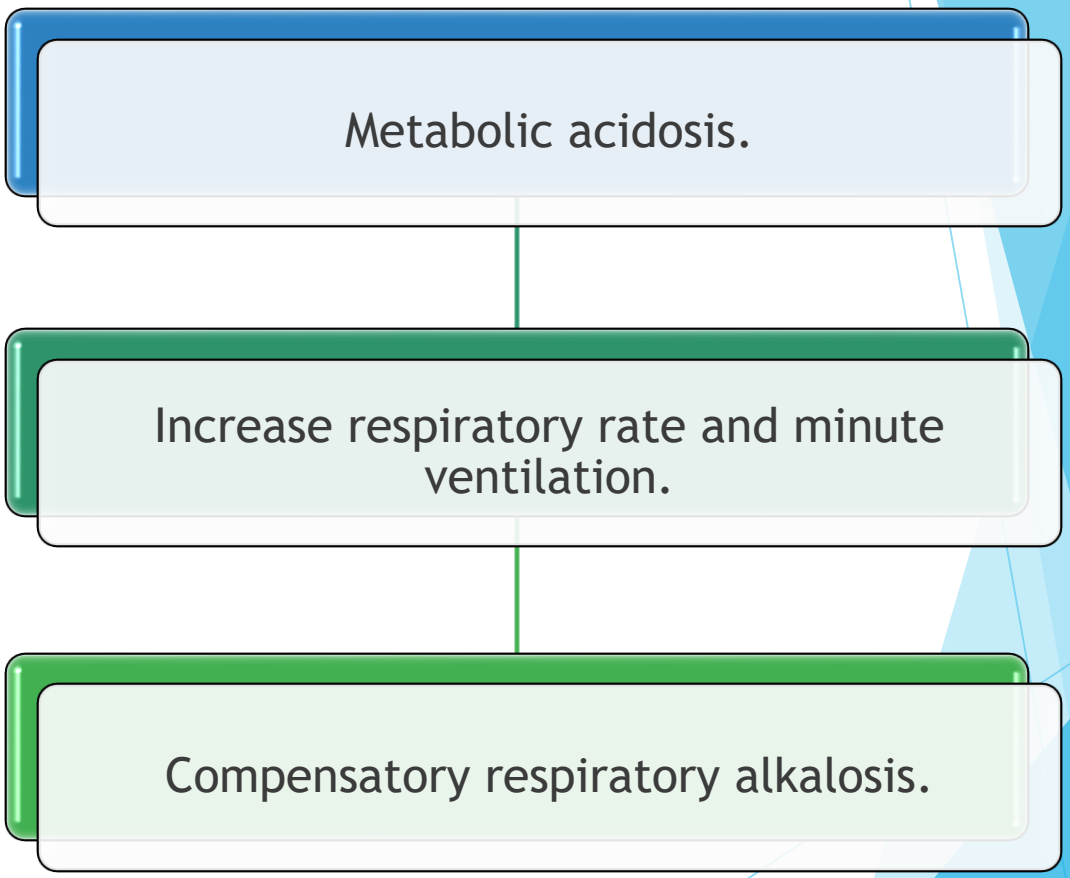
SYSTEMIC IMPACT OF SHOCK

CARDIOVASCULAR

- Decrease pre and after load
- Baroreceptor response.
- Sympathetic activity and Catecholamines
- Tachycardia and systemic vasoconstriction (except septic shock).

SYSTEMIC IMPACT OF SHOCK

RESPIRATORY



SYSTEMIC IMPACT OF SHOCK

RENAL

- Decrease renal perfusion → decrease urine output.
- Renin Angiotensin Aldosterone axis stimulation → more vasoconstriction and increase Sodium and water reabsorption.

SYSTEMIC IMPACT OF SHOCK

ENDOCRINE

- Adrenal gland medulla → adrenaline and noradrenaline.
- Cortisol from adrenal cortex ▫ sodium and water reabsorption.
- Renin Angiotensin system activation.
- Vasopressin → vasoconstriction and increase water reabsorption.

ISCHAEMIA-REPERFUSION SYNDROME

Ischaemia-reperfusion syndrome

- Systemic hypoperfusion → Cellular and organ damage progresses.
  - Direct effect of tissue hypoxia.
  - Local activation of inflammation.
- Further injury occurs once normal circulation is restored.
- The acid and potassium → direct myocardial depression, vascular dilatation and further hypotension.
- The cellular and humeral elements (complements, neutrophils, microvascular thrombi) → more endothelial injury to organs

Ischaemia-reperfusion syndrome

To attenuate the reperfusion injury:

1. Reduce the extent of tissue hypoxia.
2. Reduce the duration of tissue hypoxia.

Prevention is better than treatment

CLASSIFICATION

CLASSIFICATION OF SHOCK

- Depending on the initiating mechanism of shock.
- Different states may coexist within the same patient.

1. HYPOVOLAEMIC SHOCK
2. CARDIOGENIC SHOCK
3. OBSTRUCTIVE SHOCK
4. DISTRIBUTIVE SHOCK
5. ENDOCRINE SHOCK

HYPOVOLAEMIC SHOCK

- Hypovolaemia is the most common form of shock.
- Hypovolaemia is to some degree a component of all other forms of shock.
- A reduced circulating volume.

1. Haemorrhagic hypovolaemia (bleeding)

HYPOVOLAEMIC SHOCK

1. **Non-haemorrhagic hypovolaemia:**
   - Poor fluid intake
   - Excessive fluid loss
     - Vomiting
     - Diarrhea
     - Urinary loss (Diabetes)
     - Evaporation
     - **"Third-spacing" loss** (fluid is lost into the gastrointestinal tract and interstitial spaces e.g. bowel obstruction, pancreatitis)

CARDIOGENIC SHOCK

Primary failure of the heart to pump blood to the tissues.

Causes:

1. Myocardial infarction (M.I.)
2. Cardiac dysrhythmias.
3. Valvular heart disease.
4. Blunt myocardial injury.
5. Cardiomyopathy.
6. Myocardial depression:

CARDIOGENIC SHOCK

Evidence of venous hypertension with pulmonary or systemic oedema may coexist with the classic signs of shock.

OBSTRUCTIVE SHOCK

A reduction in preload because of mechanical obstruction of cardiac filling > fall in cardiac output.

Causes:

1. Cardiac tamponade.
2. Tension pneumothorax
3. Massive pulmonary embolus.
4. Air embolus.

DISTRIBUTIVE SHOCK

An inadequate organ perfusion, accompanied by:

- Vascular dilatation with hypotension.
- Low systemic vascular resistance.
- Inadequate afterload → abnormally high cardiac output.

There is maldistribution of blood flow at microvascular level with arteriovenous shunting and dysfunction of the cellular utilization of oxygen.

DISTRIBUTIVE SHOCK

Causes:

1. **Septic shock**: release of bacterial endotoxins the activation of cellular and humoral components of immune system.
2. **Anaphylaxis**: vasodilation is caused by histamine release.
3. **Neurogenic shock**, spinal cord injury: failure of sympathetic out flow and adequate vascular tone.

In the late phases of septic shock there is hypovolaemia from fluid loss into the interstitial spaces and there may be concomitant myocardial depression, which complicates the clinical picture.

ENDOCRINE SHOCK

Endocrine shock may present as a combination of Hypovolaemic, cardiogenic and distributive shock.

Causes:

1. Hypothyroidism: disordered vascular and cardiac responsiveness to circulating catecholamines → fall in cardiac output.
2. Hyperthyroidism: may cause a high-output cardiac failure.
3. Adrenal insufficiency: caused by adrenal pathology (Addison's disease).

SEVERITY OF SHOCK

SEVERITY OF SHOCK

Compensated shock vs. Decompensated shock

Mild, moderate and severe shock

Compensated shock

- Cardiovascular and endocrine responses
- Reduce flow to non-essential organs (skin, muscle and GIT)
- Preserve preload and flow to kidneys, lungs and brain.

SEVERITY OF SHOCK

Compensated shock

- Clinically, tachycardia and cool peripheries, but normal vital signs and urine output.
- There is occult metabolic acidosis and activation of humoral and cellular elements within the underperfused organs.
- This state will lead to multiple organ failure and death if prolonged (ischaemia-reperfusion effect)

SEVERITY OF SHOCK

If a patient has this state uncorrected for 12 hours, then he is liable for:

1) A significant high mortality rate.

2) High infection rate.

3) High incidence of multiple organ failure.

SEVERITY OF SHOCK

Decompensation

- In general, loss of around 15% of the circulating blood volume is within normal compensatory mechanisms.
- Blood pressure is usually well maintained and only falls after 30-40% of the circulating volume has been lost.

Mild shock

- Patient may exhibit mild anxiety.
- Tachycardia and tachypnea.
- Mild reduc
- Normal blo
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Moderate shock

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Severe shock

- Patient is unconscious.
- Profound tachycardia and laboured respiration.
- Hypotension.
- Urine output falls to zero.

Pitfalls

Shock

Pitfalls: Capillary refill

- Its not a specific marker of shock.
  - May be in the early stages of shock.
  - In distributive (septic) shock, the peripheries will be warm and capillary refill will be brisk despite profound shock.

Pitfalls: Tachycardia

- Tachycardia may not always accompany shock.
  - Patients on beta blockers.
  - Patients who have implanted pacemakers.
  - In some young patients with penetrating trauma, when there is haemorrhage but little tissue damage, there may be a paradoxical bradycardia rather than tachycardia accompanying the shock state.

Pitfalls: Blood pressure

- Hypotension is one of the **last** signs of shock.
  - Children and fit young adults are able to maintain blood pressure until the final stage of shock (i.e. profound shock but normal blood pressure).
  - Elderly patients who are already hypertensive may present with a 'normal blood pressure' but be hypovolaemic and hypotensive relative to their usual blood pressure.

CONSEQUENCES UNRESUSCITATABLE SHOCK

- Patients who are in profound shock for a prolonged period of time become '**unresuscitatable**'.

Prolonged profound shock

Cellular ischaemia

Cell death

loss of the body ability for compensation

UNRESUSCITATABLE SHOCK

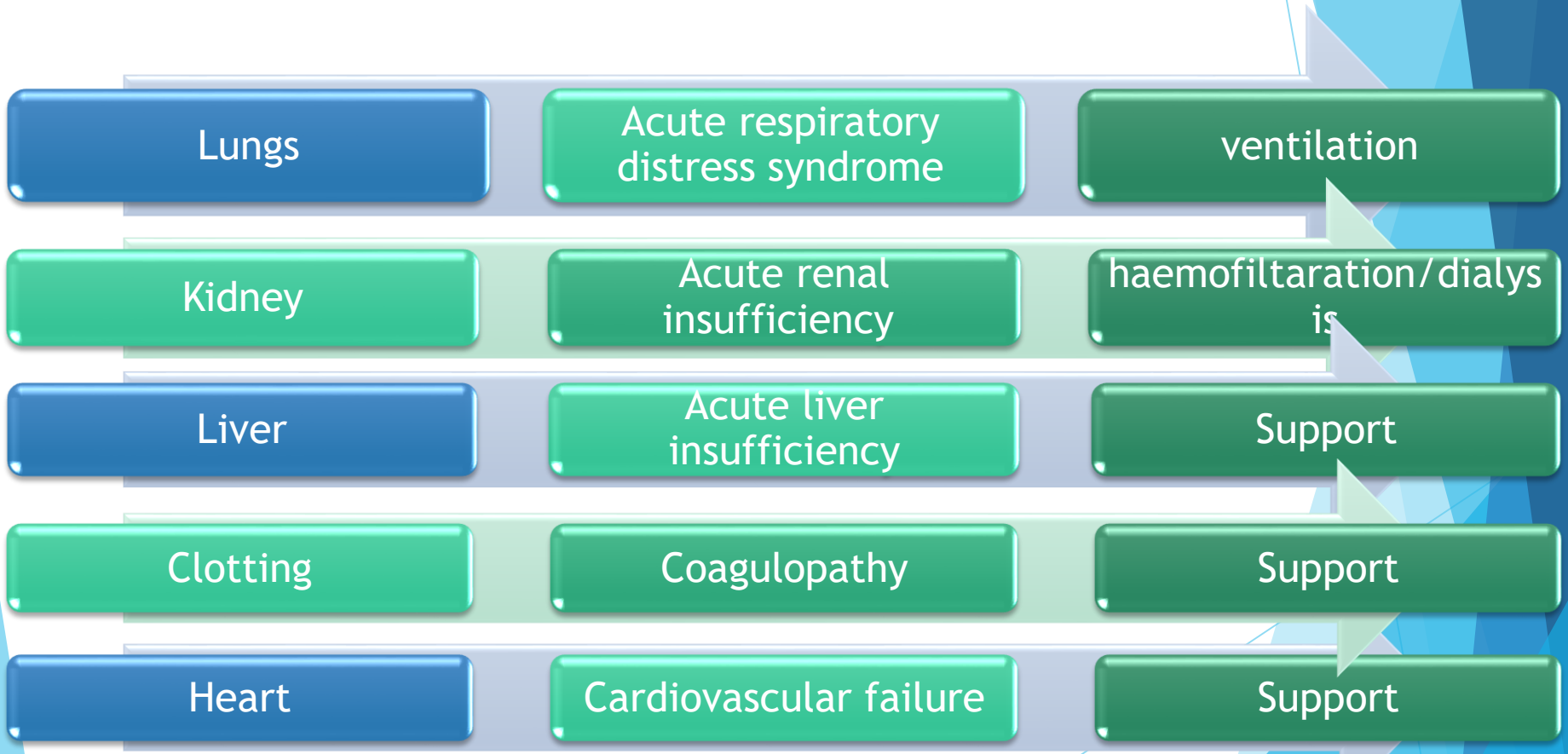
- Myocardial depression.
- Loss of responsiveness to fluid or inotropic therapy.
- Peripherally, there is loss of the ability to maintain systemic vascular resistance and further hypotension ensues.
- Death is the inevitable result.

MULTIPLE ORGAN FAILURE

Multiple organ failure is defined as two or more failed organ systems.

- When intervention is timely and the period of shock is limited, patients may make a rapid, uncomplicated recovery.
- It carries a mortality rate of 60%.
- No specific treatment for multiple organ failure, so the management is by supporting organ systems.

Multiple organ failure



THANX