PATHOPHYSIOLOGY OF SHOCK

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DEFINITION

- Shock is a systemic state of **low tissue perfusion** that is inadequate for normal cellular respiration.
- With **insufficient delivery of oxygen** and glucose, cells switch from aerobic to anaerobic metabolism. If perfusion is not restored in a timely fashion,cell death ensues.

The end result is **hypotensio**n and **cellular hypoxia** and, if uncompensated, may lead to impaired cellular metabolism and death.

TYPES OF SHOCK

Shock is classified as follows:

- **1. Hypovolaemic shock**
- **2. Cardiogenic shock**
- **3. Obstructive shock**
- **4. Distributive shock**
- **5. Endocrine shock**

1.HYPOVOLAEMIC SHOCK

- Hypovolaemic shock is due to **a reduced circulating volume.**

  Hypovolaemia may be due to haemorrhagic or non-haemorrhagic causes.

  i)**Haemorrhagic** causes :

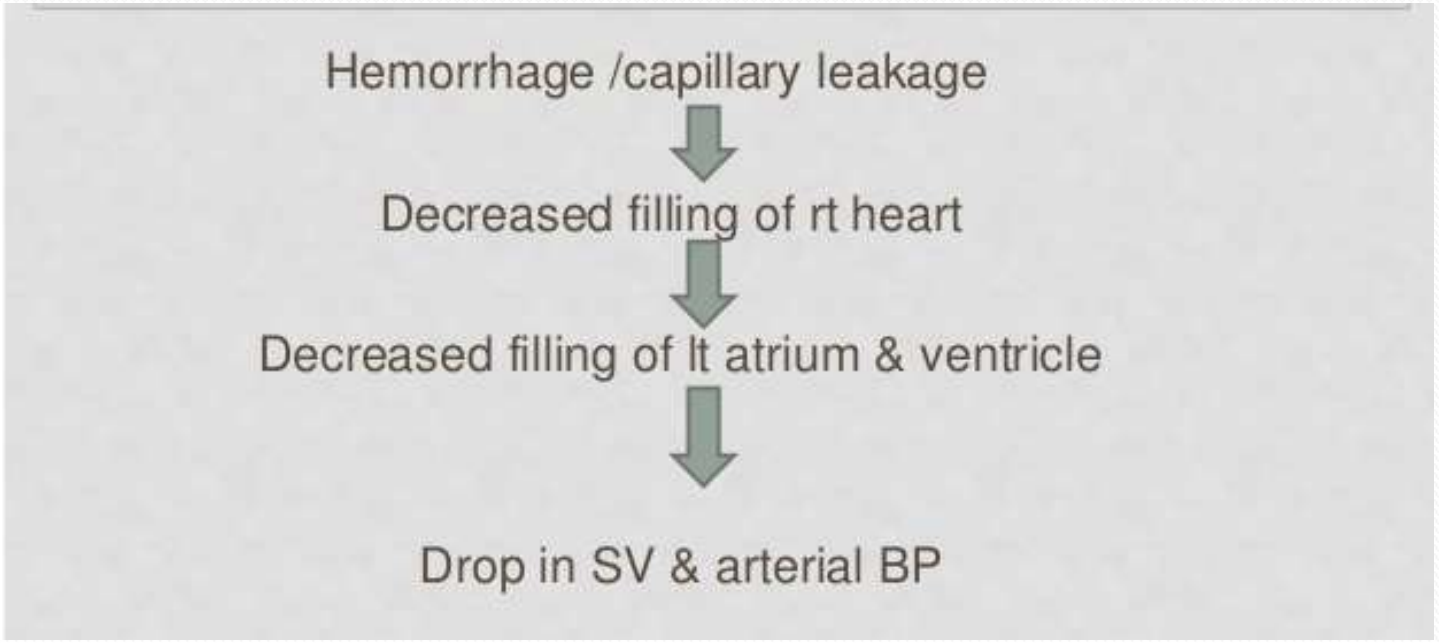
  eg; in trauma,surgery

  ii) **Non-haemorrhagic** causes:

  eg; poor fluid intake (dehydration), excessive fluid loss due to vomiting, diarrhoea,urinary loss (e.g. diabetes), evaporation, or 'third-spacing' where fluid is lost into the gastrointestinal tract and interstitial spaces, as for example in bowel obstruction or pancreatitis.

HYPOVOLAEMIC SHOCK

PATHOPHYSIOLOGY OF HYPOVOLAEMIC SHOCK



CLASSIFICATION OF HYPOVOLAEMIC SHOCK

	Class I	Class II	Class III	Class IV
Blood loss (ml)	750 ml	750–1,500 ml	1,500–2,000 ml	>2,000 ml
Blood loss (% blood volume)	<15%	15–30%	30–40%	>40%
Heart rate	<100	>100	>120	>140
SBP	No change	No change	Reduced	Very low
DBP	No change	Raised	Reduced	Unrecordable
Resp Rate	<20	>20	>30	>40
Urine output (ml/h)	>30	20–30	10–20	<10
Extremities	Normal	Pale	Pale	Cold
Mental state	Alert	Anxious	Aggressive/ drowsy	Confused/ unconscious

SBP systolic blood pressure; *DBP* diastolic blood pressure

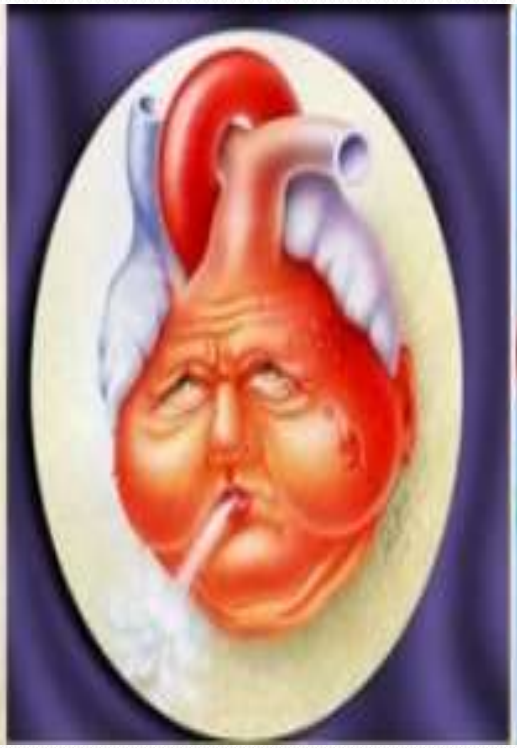
2. CARDIOGENIC SHOCK

- Cardiogenic shock is due to primary **failure of the heart to pump** blood to the tissues.

  Causes of cardiogenic shock include;

  - **myocardial infarction**
  - **cardiac dysrhythmia**
  - **valvular heart disease**
  - **blunt myocardial injury and**
  - **cardiomyopathy**

CARDIOGENIC SHOCK



3. OBSTRUCTIVE SHOCK:

- In obstructive shock there is **a reduction in preload** due to **mechanical obstruction** of cardiac filling.
- Common causes of obstructive shock include;

  - cardiac tamponade

  - tension pneumothorax

  -massive pulmonary embolus or **air embolus**
- In each case, there is reduced filling of the left and/or right sides of the heart leading to reduced preload and a fall in cardiac output.

4. DISTRIBUTIVE SHOCK

- Distributive shock describes the pattern of cardiovascular responses characterising a variety of conditions, including;

  A) Septic shock

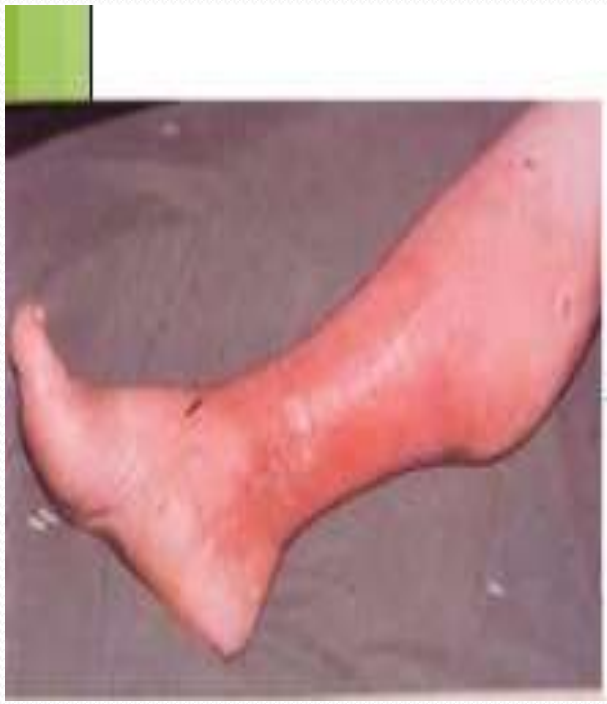
  B) Anaphylaxis and

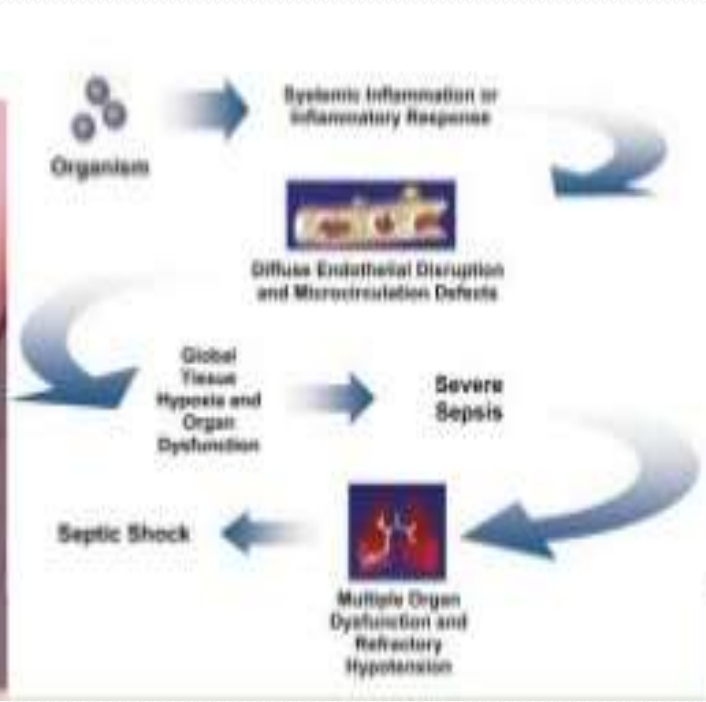
  C) Spinal cord injury (Neurogenic shock).

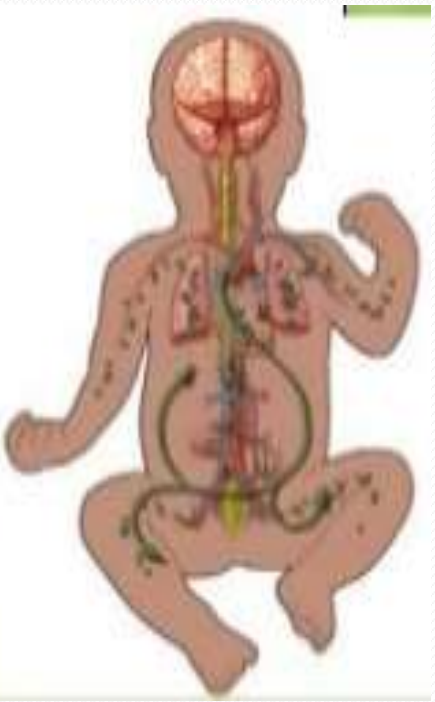
A) SEPTIC SHOCK

- The cause in sepsis is less clear but is related to the release of bacterial products (**endotoxin**) and the activation of **cellular and humoral components of the immune system.**
- There is **maldistribution of blood** flow at a microvascular level with arteriovenous shunting and dysfunction of cellular utilization of oxygen.
- In the later phases of septic shock there is hypovolaemia from fluid loss into interstitial spaces and there may be concomitant myocardial depression.

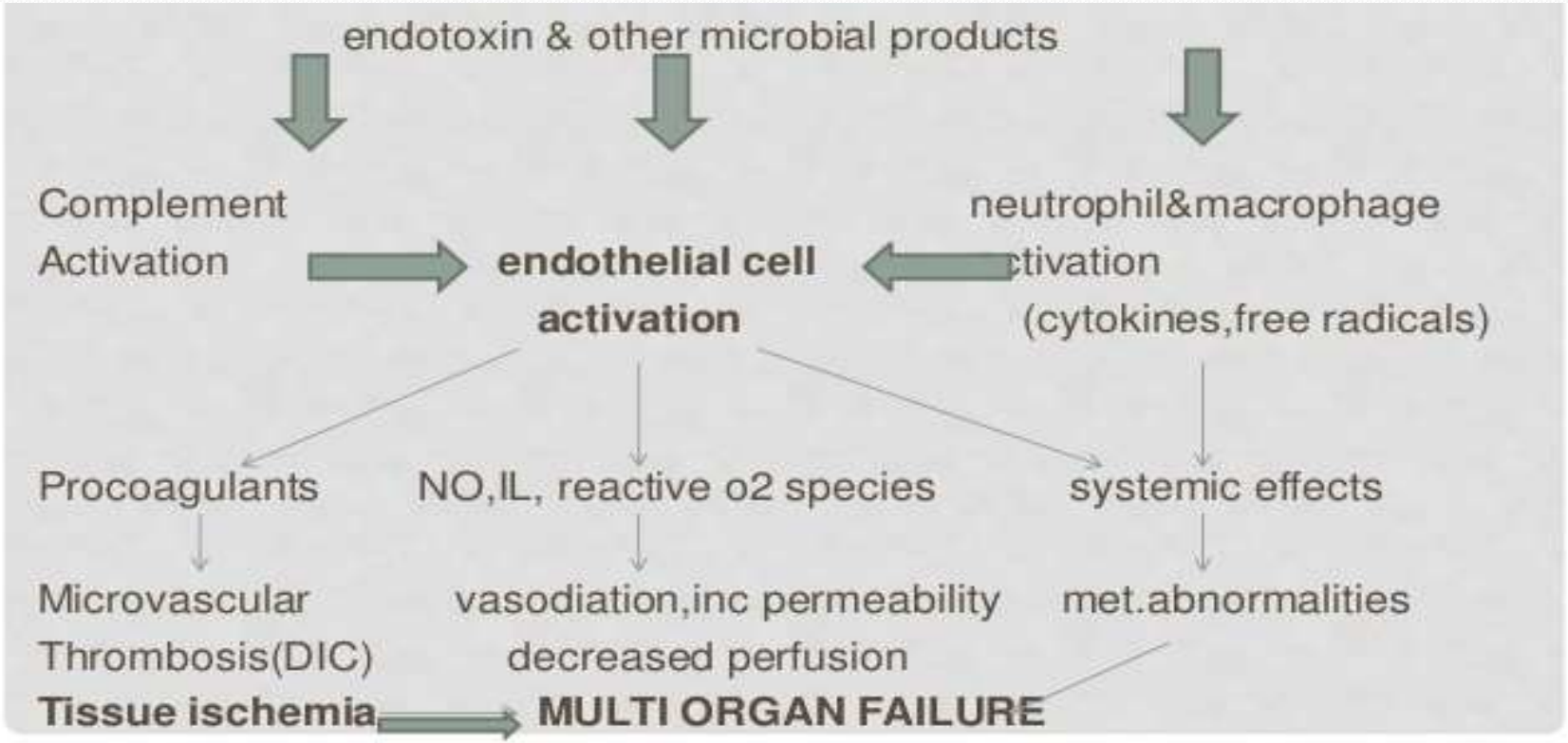
SEPTIC SHOCK



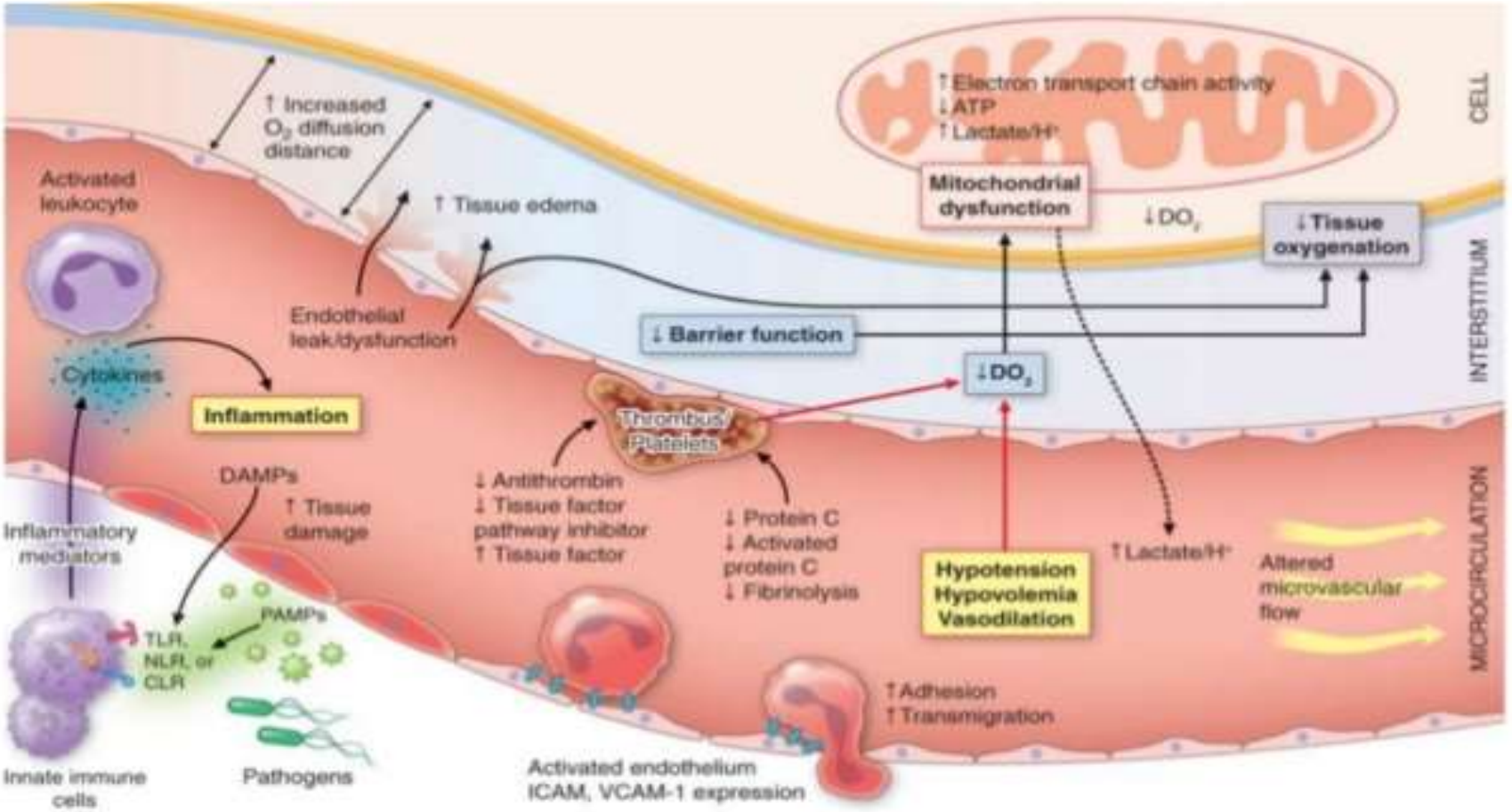




PATHOPHYSIOLOGY OF SEPTIC SHOCK



PATHOGENESIS



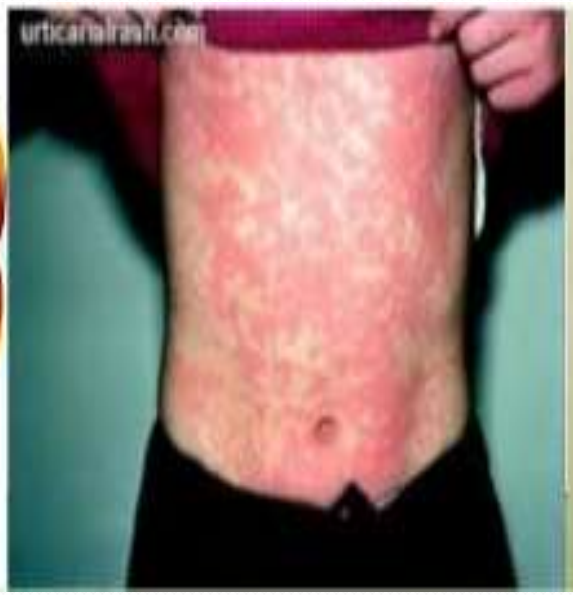
B) ANAPHYLAXIS SHOCK:

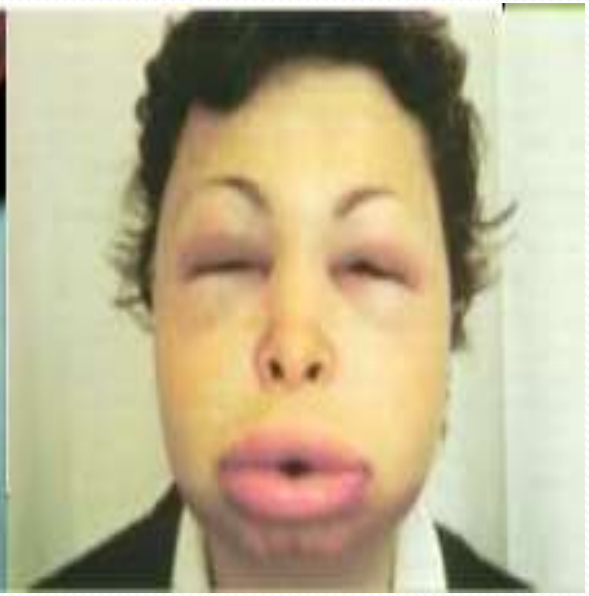
It is a type of severe **hypersensitivity** or **allergic** reaction.

- Its causes include allergy to insect stings, medicines or foods (nuts, berries, sea foods) etc.

- It is caused by a severe reaction to an allergen, leading to the release of **histamine** that causes widespread **vasodilation** and **hypotension.**

ANAPHYLACTIC SHOCK





C) NEUROGENIC SHOCK

- It is caused by **spinal cord injury** usually as a result of a traumatic injury or accident
- There **is failure of sympathetic outflow** and adequate vascular tone.
- Damage to CNS impairs cardiac function by reducing heart rate and loosening the blood vessel tone resulting in severe hypotension.

PATHOPHYSIOLOGY OF SHOCK

Pathophysiology of shock can be discussed under 3 headings

- **A) Cellular level**
- **B) Microvascular level**
- **C) Systemic level**

A) CELLULAR LEVEL

- As perfusion to the tissues is reduced, cells are deprived of oxygen and must switch from **aerobic to anaerobic** metabolism.
- The product of anaerobic respiration is not carbon dioxide but **lactic acid**. When enough tissue is underperfused, the accumulation of lactic acid in the blood produces systemic **metabolic acidosis**.
- As glucose within cells is exhausted, anaerobic respiration ceases and there is **failure of the sodium/potassium pumps** in the cell membrane and intracellular organelles.

  Intracellular lysosomes release autodigestive enzymes and cell lysis ensues.
- Intracellular contents, including potassium, are released into the bloodstream.

B) MICROVASCULAR LEVEL

- As tissue ischaemia progresses, changes in the local milieu result in **activation of the immune and coagulation systems.**
- Hypoxia and acidosis activate complement and prime neutrophils,resulting in the generation of **oxygen free radicals** and **cytokine** release.
- These mechanisms lead to **injury of the capillary endothelial** cells. These, in turn, further activate the immune and coagulation systems. Damaged endothelium loses its integrity and becomes 'leaky'.
- Spaces between endothelial cells allow **fluid to leak out** and tissue oedema ensues, exacerbating cellular **hypoxia**.

C) SYSTEMIC LEVEL

1)CARDIOVASCULAR:

- Hypovolemia leads to **decreased ventricular preload** that, in turn reduces the stroke volume.
- As preload and afterload decrease, there is a **compensatory baroreceptor response** resulting in increased sympathetic activity and release of catecholamines into the circulation.
- This results in **tachycardia** and systemic **vasoconstriction**

2)RESPIRATORY

- The response of the pulmonary vascular bed to shock parallels that of the systemic vascular bed, and the relative increase in pulmonary vascular resistance, particularly in septic shock, may exceed that of the systemic vascular resistance, leading to right heart failure.
- The **metabolic acid**osis and increased sympathetic response result in an **increased respiratory rate** and minute ventilation to increase the excretion of carbon dioxide.
- It produce a **compensatory respiratory alkalosis**.

3) RENAL

- The physiologic response of the kidney to hypoperfusion is to **conserve salt and water.** In addition to decreased renal blood flow, increased afferent arteriolar resistance accounts for **diminished glomerular filtration rate** (GFR) that together with increased aldosterone and vasopressin, is responsible for **reduced urine formation**.
- Decreased perfusion pressure in the kidney leads to reduced filtration at the glomerulus and a decreased urine output.
- The **renin–angiotensin–aldosterone axis is stimulated**, resulting in further vasoconstriction and increased sodium and water reabsorption by the kidney.

4)ENDOCRINE

- Hypovolemia, hypotension, and hypoxia are sensed by baroreceptors and chemoreceptors that contribute to an autonomic response that attempts to restore blood volume, maintain central perfusion, and mobilize metabolic substrates.
- Activation of the **adrenal and renin–angiotensin systems**, **vasopressin** (antidiuretic hormone) is released from the hypothalamus in response to **decreased preload** and results in vasoconstriction and resorption of water in the renal collecting system.
- **Cortisol** is also released from the adrenal cortex, contributing to the sodium and water resorption and sensitising cells to catecholamines.

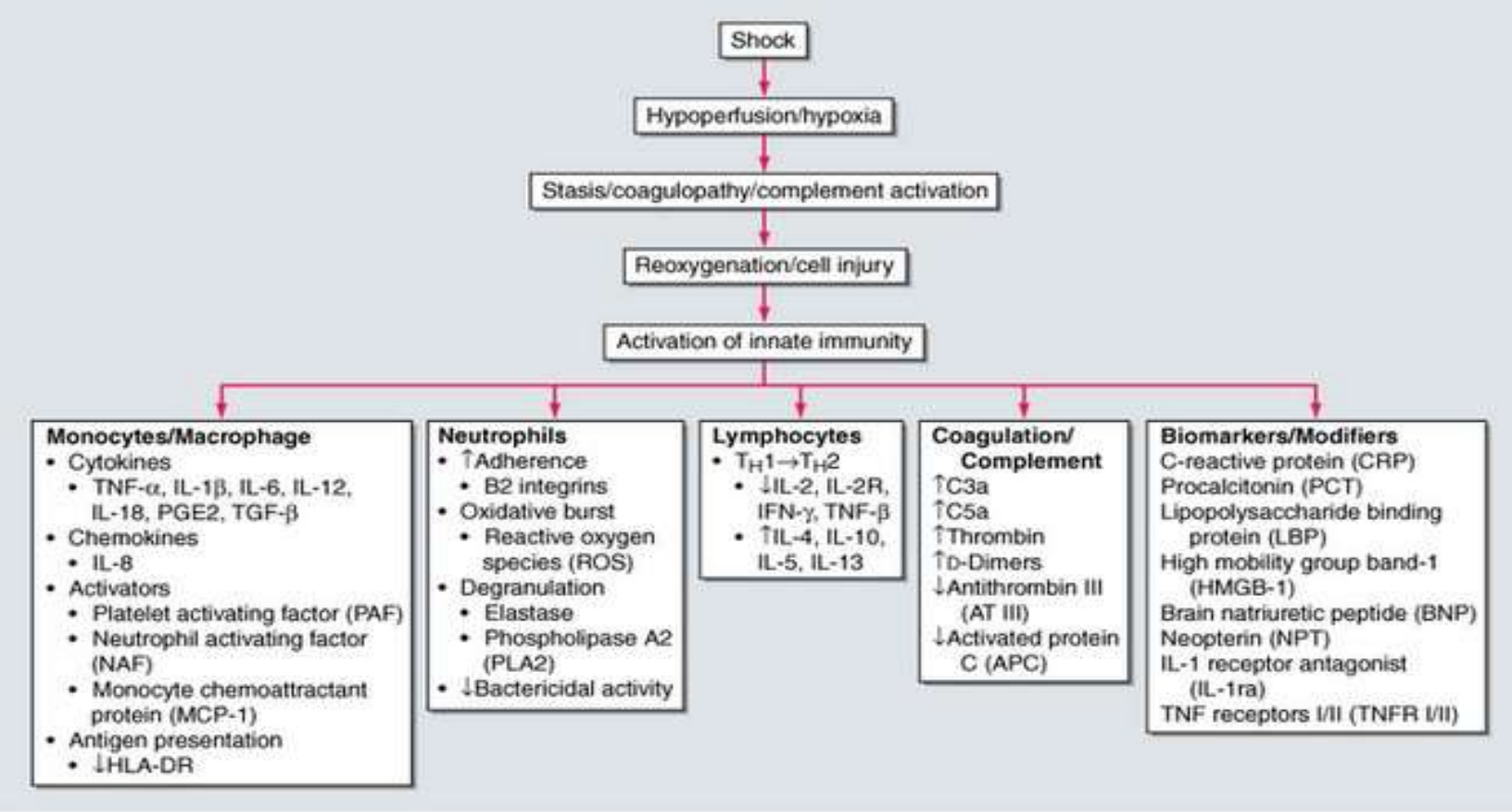
5) METABOLIC

- During shock, there is disruption of the normal cycles of carbohydrate,lipid, and protein metabolic.
- Through the citric acid cycle alanine in conjunction with lactate, which is converted from pyruvate in the periphery in the presence of oxygen deprivation, enhances the hepatic production of glucose.
- With reduced availability of oxygen, the breakdown of glucose to pyruvate, and ultimately lactate, represents an inefficient cycling of substrate with minimal net energy production.

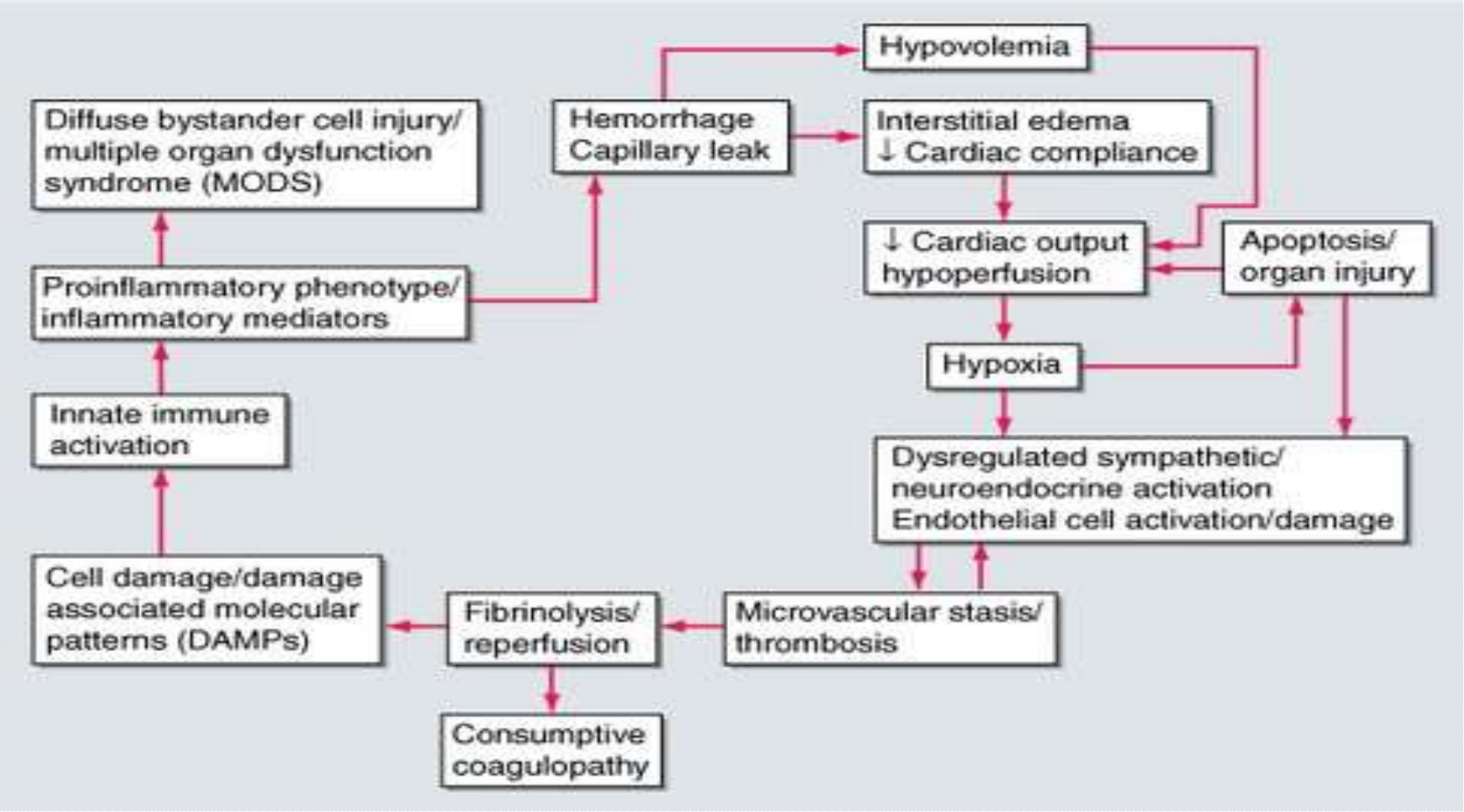
Cardiovascular and metabolic characteristics of shock

	Hypovolaemia	Cardiogenic	Obstructive	Distributive
Cardiac output	Low	Low	Low	High
Vascular resistance	High	High	High	Low
Venous pressure	Low	High	High	Low
Mixed venous saturation	Low	Low	Low	High
Base deficit	High	High	High	High

A schematic of the host immunoinflammatory response to shock.



Shock-induced vicious cycle.



There are **two basic** features in the mechanism of shock:

- 1. Reduced effective circulating volume.
- 2. Reduced supply of oxygen to the cells and tissues with resultant anoxia (**Tissue anoxia**).

1. <u>Reduced effective circulating volume</u>: It may result by either of the following mechanisms:

- i) By actual loss of blood volume as occurs in hypovolaemic shock.
- ii) By decreased cardiac output without actual loss of blood (normovolaemia) as occurs in cardiogenic and septic shock.

2. Tissue anoxia:

Following reduction in the effective circulating blood volume, there is **decreased venous return** to the heart resulting in decreased cardiac output. This consequently causes reduced supply of oxygen to the organs and tissues and hence tissue anoxia and shock ensues.

SEQUENTIAL EVENTS IN THE MECHANISM OF SHOCK:

- Decreased effective circulating blood volume.
- Decreased venous return to the heart.
- Decreased cardiac output.
- Decreased blood flow.
- Decreased supply of oxygen.
- Anoxia.
- Shock.

STAGES OF SHOCK:

Table 2.2 Clinical features of shock.

	Compensated	Mild	Moderate	Severe
Lactic acidosis	+	++	++	+++
Urine output	Normal	Normal	Reduced	Anuric
Conscious level	Normal	Mild anxiety	Drowsy	Comatose
Respiratory rate	Normal	Increased	Increased	Laboured
Pulse rate	Mild increase	Increased	Increased	Increased
Blood pressure	Normal	Normal	Mild hypotension	Severe hypot

STAGES OF SHOCK:

Deterioration of the circulation in shock is a progressive phenomenon and can be divided into 3 stages:

- 1. **Non-progressive** (initial, compensated, reversible) shock
- 2. **Progressive decompensated** shock
- 3. **Decompensated** (irreversible) shock

1. NON-PROGRESSIVE (INITIAL, COMPENSATED, REVERSIBLE) SHOCK:

In the early stage of shock, an attempt is made to maintain adequate cerebral and coronary blood supply by redistribution of blood so that the vital organs (brain & heart) are adequately perfused and oxygenated. This is achieved by activation of various neurohumoral mechanisms causing “wide spread vasoconstriction” and by “fluid conservation by the kidney”. If the condition that caused the shock are adequately treated, the compensatory mechanism may be able to bring about recovery & re-establish the normal circulation; this is called **compensated or reversible shock**. These compensatory mechanisms are:

i) Wide spread vasoconstriction

ii) Fluid conservation by the kidney

iii) Vascular autoregulation

i) WIDE SPREAD VASOCONSTRICTION

- In response to reduced blood flow (hypotension) and tissue anoxia, the neural and humoral factors (e.g. Baroreceptors, chemoreceptors, catecholamines, renin & VEM/vasoexcitor material from hypoxic kidney) are activated.
- All these bring about vasoconstriction.Wide spread vasoconstriction is a protective mechanism as it causes increased peripheral resistance, increased heart rate (tachycardia) and increased BP.
- Clinically cutaneous vasoconstriction is responsible for cool and pale skin in initial stage of shock.

ii) FLUID CONSERVATION BY THE KIDNEY:

- In order to compensate the actual loss of blood volume in hypovolaemic shock, the following factors may assist in restoring the blood volume and improve venous return to the heart:
  - Release of **aldosterone** from hypoxic kidney.
  - Release of **ADH** due to decreased effective circulating blood volume.
  - Reduced GFR due to arteriolar constriction.
  - Shifting of tissue fluids into the plasma due to lowered capillary hydrostatic pressure (hypotension).

iii) VASCULAR AUTOREGULATION:

- In response to hypoxia and acidosis, regional blood flow to the heart and brain is preserved by vasodilation of the coronary and cerebral circulation.

2. PROGRESSIVE DECOMPENSATED SHOCK:

- This is a stage when the patient suffers from other stress or risk factors (e.g. Pre-existing cardiovascular and lung disease) besides persistence of the shock so that there is a progressive deterioration.
- The effects of progressive decompensated shock due to tissue hypoperfusion are as follows:

  a) Pulmonary hypoperfusion with resultant tachypnoea and adult respiratory distress syndrome.

  b) Tissue anoxia causing anaerobic glycolysis results in metabolic lactic acidosis.

3. DECOMPENSATED (IRREVERSIBLE) SHOCK:

- When the shock is so severe that inspite of compensatory mechanisms and despite therapy and control of etiologic agent which caused the shock, **no recovery takes place,** it is called decompensated or irreversible shock. It's effects due to widespread cell injury include the following:

  a) Progressive fall in the blood pressure due to deterioration in cardiac output attributed to release of myocardial depressant factor (MDF).

  b) Severe metabolic acidosis due to anaerobic glycolysis.

  c) Respiratory distress due to pulmonary oedema, tachypnoea and adult respiratory distress syndrome (ARDS).

  d) Ischemic cell death of brain, heart and kidneys due to reduced blood supply to these organs.

- Clinically, at this stage the patient has features of **coma**, **worsened heart function** and progressive **renal failure** due to acute tubular necrosis.

DIFFERENTIATION

Type of Shock	CVP and PCWP	Cardiac Output	Systemic Vascular Resistance	Venous O_2 Saturation
Hypovolemic	↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Septic				
Hyperdynamic	↓↑	↑	↓	↑
Hypodynamic	↓↑	↓	↑	↓↑
Traumatic	↓	↓↑	↑↓	↓
Neurogenic	↓	↓	↓	↓

MANAGEMENT OF SHOCK:

General principles of shock management:

1. The overall goal of shock management is to **improve oxygen** delivery/utilisation in order to prevent cellular and organ injury.
2. Effective therapy requires treatment of underlying etiology.
3. Restoration of adequate perfusion, monitoring and comprehensive supportive care.
4. Interventions to restore perfusion centre on achieving an adequate blood, increasing cardiac output and optimising oxygen content of the blood.
5. Oxygen demand should also be reduced.

MANAGEMENT OF HYPOVOLAEMIC SHOCK:

- Maximise **oxygen delivery**-completed by ensuring adequacy of ventilation, increasing oxygen saturation of the blood and restoring blood flow.
- Control further blood loss.
- **Fluid resuscitation**.
- Drugs like anti-secretory agents have vasoconstrictive properties and can reduce blood flow to portal systems. E.g. **Somatostatin**.

MANAGEMENT OF SEPTIC SHOCK:

- The most important aspects of medical therapy for patients with septic shock/sepsis include:

  i. Adequate **oxygen delivery**.

  ii. Crystalloid fluid administration-normal saline (0.9% NaCl), lactated ringer, plasmylate.

  iii. Broad spectrum **antibiotics**-cefotaxim, clindamycin, ceftriaxone, ciprofloxacin, metronidazole, cefepime, levofloxacin, vancomycin.

  iv. Alpha and beta **adrenergic agonists**-nor epinephrine, dopamine, dobutamine, epinephrine, vasopressin, phenylephrine.

  v.**Corticosteroids** like hydrocortisone, dexamethasone.

MANAGEMENT OF CARDIOGENIC SHOCK:

- Management of cardiogenic shock includes **supportive** measures:
- **Oxygen therapy**, judicious use of fluids with careful monitoring of central venous pressure or pulmonary wedge pressure.
- Other monitoring should include continuous ECG, 12 lead ECG, urine output, urea and electrolytes & blood gases.
- Patient should be preferably managed in **coronary care unit.**
- Drugs like **morphine** 5-10mg i.v. helps to relieve pain and anxiety associated with myocardial infarction. Inotropic support, vasodilators and mechanical support may be needed.

MANAGEMENT OF NEUROGENIC SHOCK:

- Restoring **sympathetic tone**: it would be either through the stabilisation of a spinal cord injury or in the instance of spinal anaesthesia by positioning the patient appropriately.
- **Immobilization:** if the patient has a suspected case of spinal cord injury, a traction may be needed to stabilize the spine to bring it to proper alignment.
- **IV fluids**: administration of i.v. 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 given to anaphylactic shock includes:

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

2.**Oxygen**, which helps in breathing.

3.Intravenous (i.v.) **antihistamines** and **cortisone** to reduce inflammation of air passages and improve breathing.

4.A beta agonist (e.g. Albuterol) to relieve breathing symptoms.

THANK YOU