جامعة الملك سعود
King Saud University

# Shock and Metabolic Response to Surgery

Surgery Team
MED 433

MED 433

# Objectives :

1. **Definition of Shock**
2. **Causes of Shock**
3. **Pathophysiology of Shock** (Microcirculation, Microcirculation, Cellular function )
4. **Effects on Individual Organ Systems** (Nervous system, Kidneys, Respiratory system, Heart, Gut, Liver, Neuro humoral response )
5. **Principles of Management** (Hypovolemic shock, Septic shock, Cardiogenic shock, Anaphylaxis )
6. **Advanced Monitoring Organ Support** (Cardiovascular support, Respiratory support, Renal support)
7. **Nutrition**

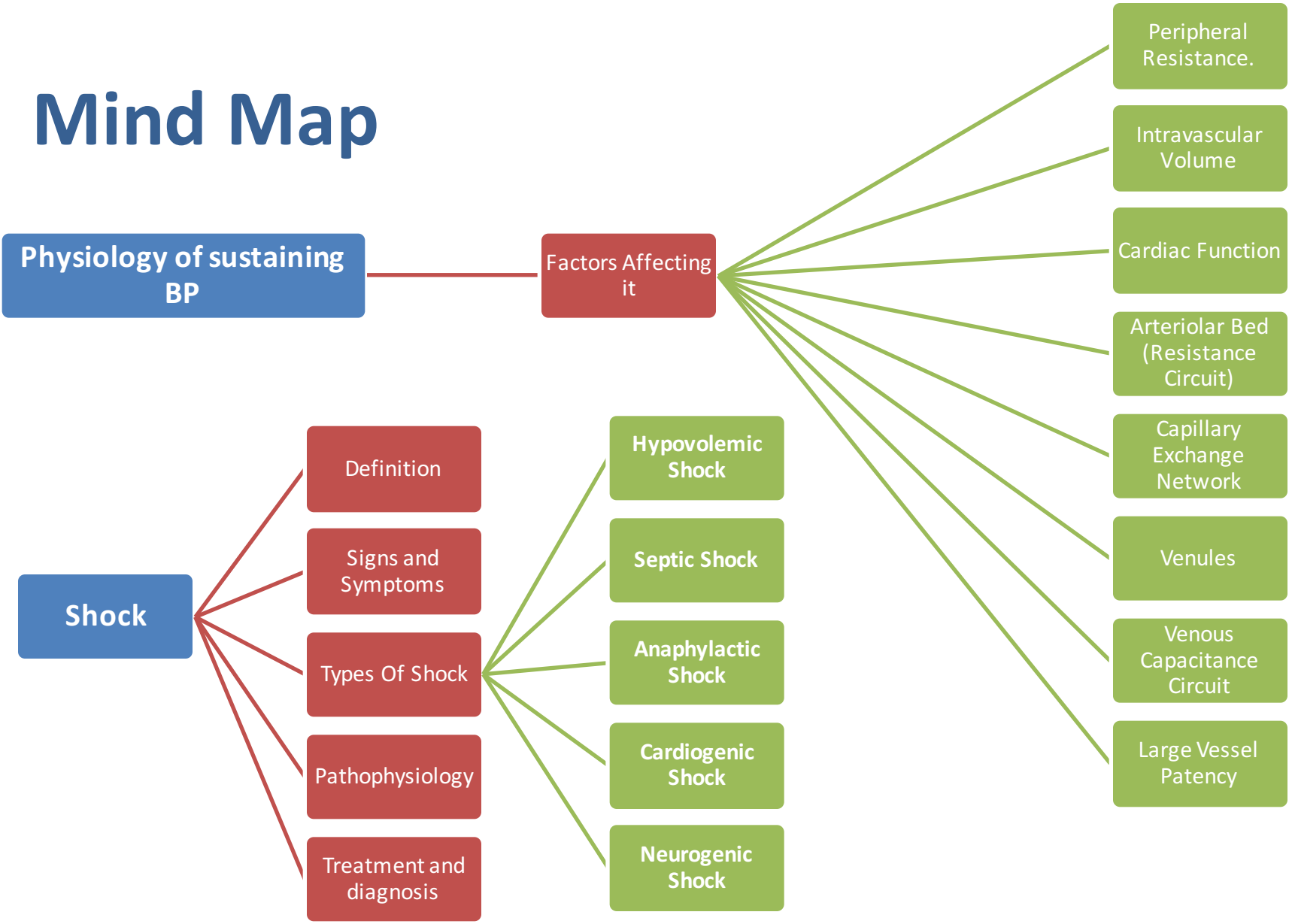
**The Metabolic Response To Injury:**

1. **Features of the response when not modified by medical intervention**
2. **Factors mediating the metabolic response to injury** (The acute inflammatory response, The endothelium and blood vessels, Afferent nerve impulses and sympathetic nervous system activation, The endocrine response of surgery)
3. **Consequences of the metabolic response to injury** (Hypovolaemia, Increased energy metabolism and substrate cycling, Catabolism and starvation, Changes in red blood cell synthesis and blood coagulation)
4. **Factors modifying the metabolic response to injury** (Control of blood glucose, Manipulation of inflammation and coagulation in severe infection)
5. **Anabolism**

**Sources :** Slides, Raslan's Notebook, Principles & Practice of Surgery by: O. James Garden, Step Up To Medicine.

**Color Index** : Slides & Raslan's | Textbook | Doctor's Notes | Extra Explanation

# Mind Map




**We truly recommend that you go back to physiology lectures of CV Block, especially:**
**L7&9-** Regulation Of Stroke Volume
**L11-12-** Regulation Of Blood Pressure

# 1st :Blood Pressure Regulation

- **Changes In the following Elements Regulate BP And Perfusion:**

1. **Peripheral Resistance.**
2. **Intravascular Volume**
3. **Cardiac Function** (heart)
4. **Arteriolar Bed (**Resistance Circuit)
5. **Capillary Exchange Network**
6. **Venules**
7. **Venous Capacitance Circuit**
8. **Large Vessel Patency**

**MAP = CO X TPR** — **CO*** = HR x Stroke volume

## 1-Total Peripheral Resistance (TPR)

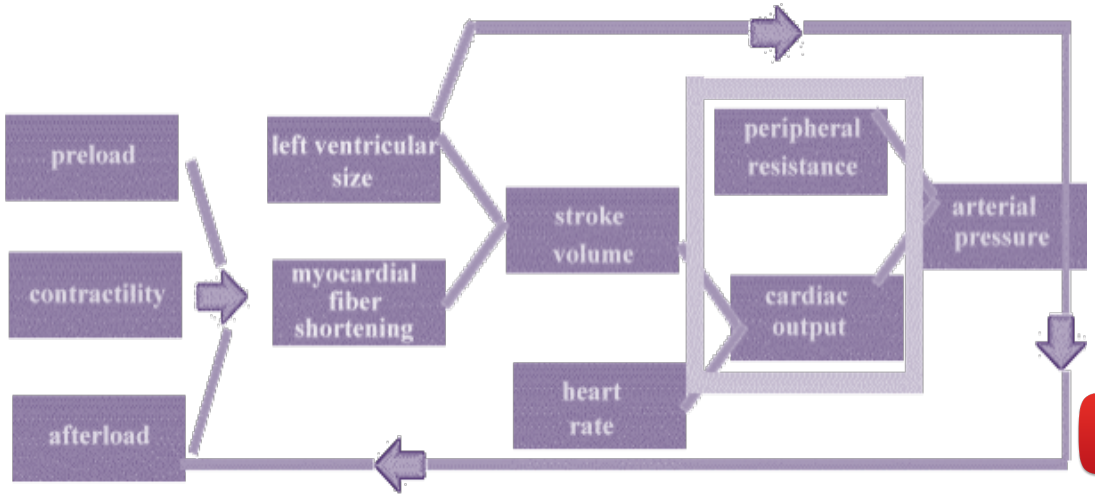
It's the resistance that must be overcome to push blood through the circulatory system and create flow.

★ **TPR is determined by:**

1. Diameter Of Blood Vessel (Change in blood vessels' diameter will affect blood pressure)
2. Blood viscosity.

If it decreases:
**Decreased arterial blood pressure**

If it increases:
- ✓ **Decreased venous return.**
- ✓ **Decreased EDV**
- ✓ **Decreased SV**
- ✓ **Decreased CO** (CO = HR X SV)
- ✓ **Decreased arterial blood pressure (MAP=CO X PR)**




*Heart Rate (HR) X Stroke Volume (↓ intravascular volume, ↓ EDV) = **Cardiac Output** → Cardiac Output X Peripheral Resistance = **Arterial Pressure.**

**MAP** =**M**ean **A**rterial blood **P**ressure.

**Stroke Volume (SV)** = Volume of blood ejected by left ventricle of the heart per beat.

**Venous Return**: the flow of blood from the periphery to the right atrium, it is equal to cardiac output.

**EDV = E**nd-**D**iastolic **V**olume: Amount of blood remaining in the heart by the end of diastole.

**TPR** = Total peripheral resistance.

**Khan Academy:-** Putting It All Together: Pressure, Flow, And Resistance (very helpful)

# 2- Intravascular Volume :

## What is intravascular volume ?

It is the volume of blood in a patient's circulatory system

## Loss of intravascular volume is usually caused by :

| | | |
|---|---|---|
| Bleeding | Failure to Rehydrate | Loss of third space fluids (sweating) |

## The effect of intravascular volume on BP and perfusion:

| | |
|---|---|
| **Decrease in intravascular volume=decreased Blood pressure** | **Decrease in intravascular volume = Decrease venous return =Decreased end diastolic volume.** |

# 3- Cardiac Function:

| Cardiac output is the result of: | Examples of changes that can alter cardiac output |
|---|---|
| **Heart Rate** | 1. **Bradycardia:** ↓ **heart Rate** (lead to ↑ **SV** ↓ **CO,** longer time for ventricle filling but not enough to give normal CO)<br>2. **Tachycardia:** ↑ **heart rate** (lead to ↓ **SV&CO,** Because the time of filling ventricle is shorted) |
| **Contractility** | MI or cardiomyopathy (pump failure) |
| **Loading Conditions** | **Vasodilator Agents:** eg. Histamine (Vasodilation: decreased end-diastolic volume) |

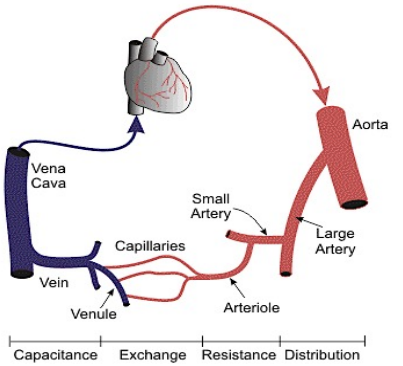
# 4- Resistance Circuit (Arteriolar Bed):

***What determine the BP in the arterioles?**
Increase in the permeability and the oncotic pressure. Oncotic pressure will increase due to the presence of proteins in the blood vessel which lead to fluid shift from extravascular to intravascular space, leading to volume expansion 

- **Decreases In Arteriolar* Tone Produce:**
  - Hypotension.
  - Decreased perfusion to vital organs.
- **Increases In Arteriolar Tone:** will prevent optimal cardiac performance (increased afterload=decreased contractility).

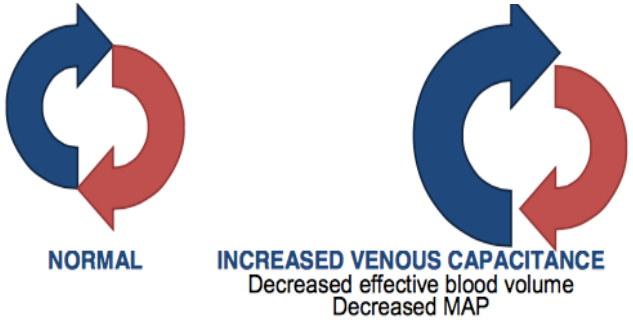
# 5- Capillary Exchange Network:

- Largest area of the vascular tree.
- Site of exchange of nutrients, electrolytes and fluids.
- Alterations in microvascular integrity (e.g., capillary leak syndrome) result in loss of intravascular volume.
- Blockage of or shunting away from small vessels leads to decreased tissue perfusion.



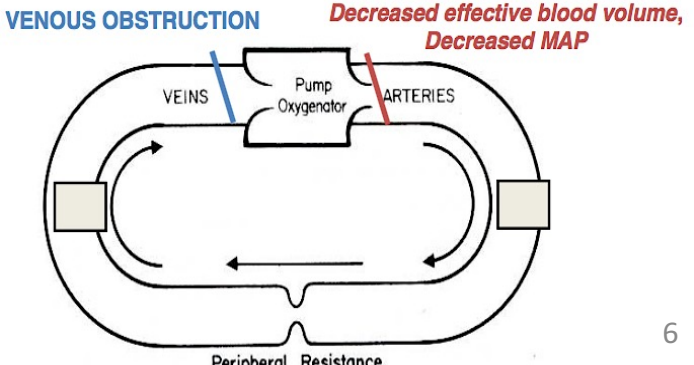

# 6- Venous Capacitance :

- ✓ Portion of the circulatory system contains 80% of the intravascular volume.
- ✓ **Decrease in effective circulating blood volume and MAP caused by:**
  - Decreases in venous tone.
  - Increases in venous vascular capacitance




# 7- Large Vessel Patency:

- ✓ Obstruction of the systemic or pulmonic circuit will decrease ventricular ejection and systemic perfusion.
- ✓ Venous obstruction will decrease venous return.
- ✓ **Examples of obstructive shock:**
  - Massive pulmonary embolism.
  - Venous occlusion.



# 2nd :Shock

## Definition

**Shock: State of altered tissue perfusion severe enough to induce derangements in normal cellular metabolic function** (shortly: low perfusion that causes tissue hypoxia)

**Shock can also be defined as** an imbalance between oxygen delivery and oxygen demand that results in cell dysfunction and ultimately cell death and multiple organs failure.

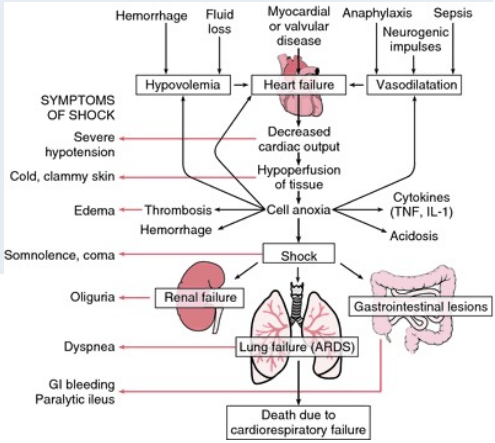
## Signs and Symptoms

### Clinical Signs And Symptoms Of Shock Relate To Decreased Organ Perfusion :

1. **Mental Status Changes:** decreased cerebral perfusion
2. **Decreased urine output:** decreased renal perfusion
3. **Cold Clammy Extremities:** Decreased perfusion to the skin due to diverted blood flow
4. **EKG Changes:**
   - May indicate myocardial ischemia
   - May be primary event (cardiogenic shock) or due to decreased myocardial perfusion due to shock from other causes

### Hemodynamic Parameters That May Indicate Shock:

- **Heart Rate:** Initial tachycardia (attempt to increase CO)
- **Rhythm:** Regular and tachycardic
- **Blood Pressure:** Low
- **Cardiac Output:** Usually low




## Types Of Shock

Each type will be detailed in the upcoming slides

### Types of Shock

1- Hypovolemic Shock

2- Septic Shock

3- Anaphylactic Shock

4- Cardiogenic Shock

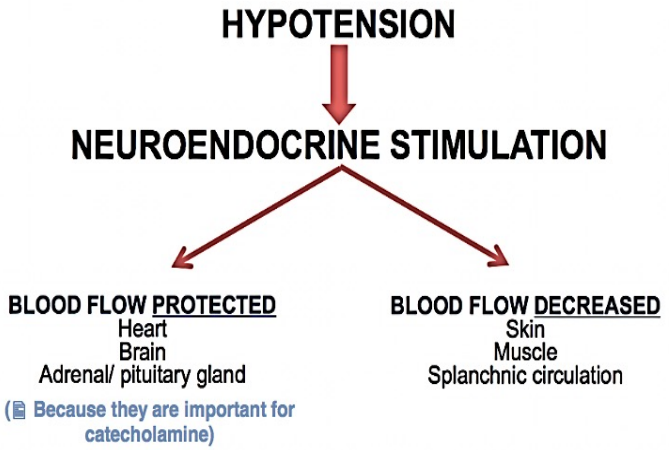
5- Neurogenic Shock

# Hemodynamic Response To Shock

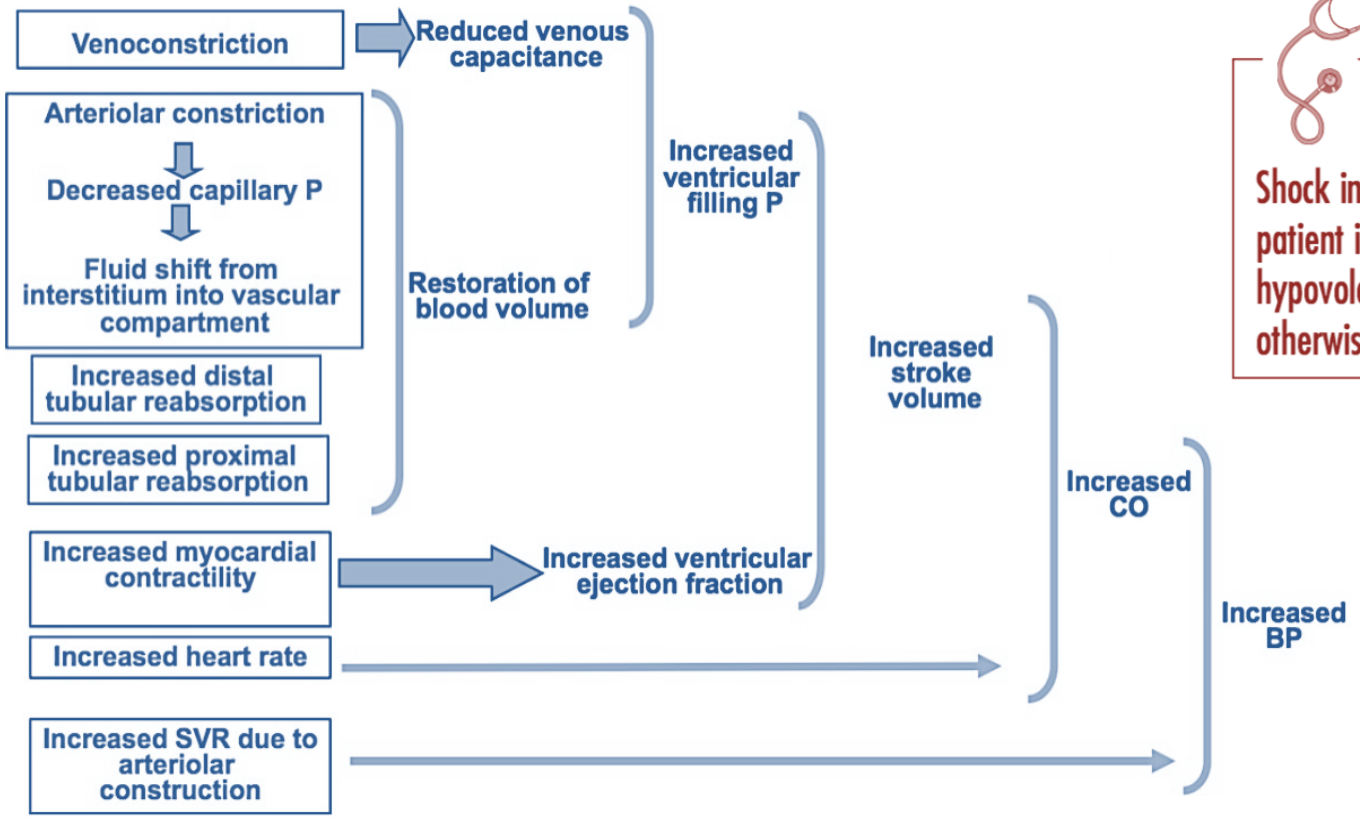
★ **Mechanisms For Restoring Cardiovascular Homeostasis:**

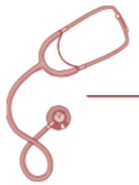
1. **Redistribution Of Blood Flow:** Attempt to preserve perfusion to vital organs.
2. **Augmentation Of Cardiac Output:** Increased heart rate, Increased peripheral resistance .
3. **Restoration Of Intravascular Volume.**

**1- Redistribution Of Blood Flow**




## HEMODYNAMIC RESPONSE TO SHOCK



Shock in trauma or postop patient is assumed to be hypovolemic until proven otherwise.

# Pathophysiology

This slide is not mentioned in the lecture but included in the objectives.

In clinical practice there is often significant overlap between the causes of shock; for example, patients with septic shock are frequently also Hypovolaemic. While differences can be detected at the level of the macrocirculation, most shocks (**exception neurogenic**) is associated with increased sympathetic activity and all share common pathophysiological features at the **cellular level.**

**Macrocirculation** — will be detailed in each type

## Microcirculation

- Changes in the microcirculation (arterioles, capillaries and venules) have a central role in the pathogenesis of shock .
- **Arteriolar vasoconstriction**: seen in early hypovolemic and cardiogenic shock.
- If shock remains uncorrected, local **accumulation of lactic acid and carbon dioxide**, together with the release of vasoactive substances lead to vasodilation .
- **Capillary permeability increases** with the loss of fluid into the interstitial space and haemoconcentration within the capillary.
- The resulting increase in blood viscosity, which lead to to platelet aggregation and the formation of microthrombi.

## Cellular Function

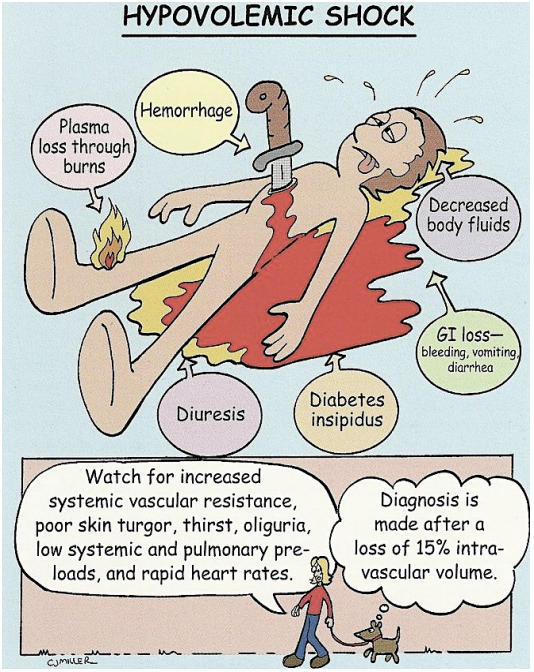
- ✓ Under normal aerobic conditions glucose is metabolism is oxidative (energy efficient; 38 moles of ATP per mole of glucose)
- ✓ Tissues extract about 25% of oxygen delivered to them, when the oxygen delivery falls cells are able to increase the amount of oxygen extracted as a compensatory mechanism, however this is limited with a maximum oxygen extraction ratio of 50%, at this point further reduction in oxygen delivery will cause critical reduction in oxygen consumption and anaerobic metabolism takes place (Dysoxia)
- ✓ Anaerobic respiration -> rise in lactic acid concentration in the circulation

- ✓ Because of ATP depletion sodium does not actively move against its concentration gradient, instead it accumulates inside the cell.
- ✓ Other effects secondary to the depletion of ATP is an osmotic gradient across the cell membrane, dilation of the endoplasmic reticulum and cell swelling
- ✓ Accumulation of lactic acid inside the cell reduces the PH -> disruption of protein synthesis, damage lysosomal and mitochondrial membranes -> necrosis

# Types Of Shock

## 1- Hypovolemic Shock:

- This is the **commonest** and most readily **corrected** cause of shock .
- It results from a **reduction in intravascular volume** secondary to :
  - **Loss of Blood**{most common cause}(e.g. trauma, gastrointestinal hemorrhage and Surgery)
  - **Loss of Plasma** (e.g. burns)
  - **Loss of Water And Electrolytes** (e.g. vomiting, diarrhea, diabetic ketoacidosis)
- Decrease in intravascular blood volume lead to **decrease** in **cardiac output** and tissue perfusion.




### Pathophysiology (Macrocirculation):

In hypovolemic shock there is **catecholamine** release from the adrenal medulla and sympathetic nerve endings, lead to **tachycardia and increased myocardial contractility act to preserve cardiac output.**

### Clinical Signs and Symptoms

| Decrease In Intravascular Blood Volume | Blood Diverted From Skin To Maintain Organ Perfusion | Blood Diverted Preferentially To Heart And Brain | Decreased Blood Flow To Brain And Heart | End-stage Shock |
|---|---|---|---|---|
| • **Due to any cause** of those listed above | • **Pale and cool skin**<br>• **Postural hypotension**<br>• **Tachycardia** | • **Oliguria**<br>• **Tachycardia**<br>• **Hypotension** | • **Restless**<br>• **Agitated**<br>• **Confused**<br>• **Hypotension**<br>• **Tachycardia**<br>• **Tachypnea** | • **Bradycardia**<br>• **Arrhythmias**<br>• **Death** |

- **Hypovolemic shock is** associated with peripheral vasoconstriction (cool skin)
- **Septic shock** is associated with severe peripheral vasodilatation (flushing, warm skin).

**Khan Academy: Hypovolemic Shock (9:37)**

# Management of Hypovolemic Shock:

The severity of hemorrhagic shock is frequently classified according to percentage of Estimate Blood Volume lost :

| Severity of Blood Loss | Blood Loss | | Heart Rate | CNS |
|---|---|---|---|---|
| | (%EBV) | ml | | |
| Class I | < 15% ( like in blood donation) | <750 | <100 | Normal |
| Class II | 15–30% | 750-1500 | >100 | Anxious |
| Class III | 30–40% | 1500–2000 | >120 | Confused |
| Class IIII | >40% | >2000 | >140 | Lethargic |

**Treatment: replace volume + treat underlying cause.**

1. **Airway and Breathing**: patients in **severe** shock and circulatory collapse generally require intubation and mechanical ventilation.
2. **Circulation:**

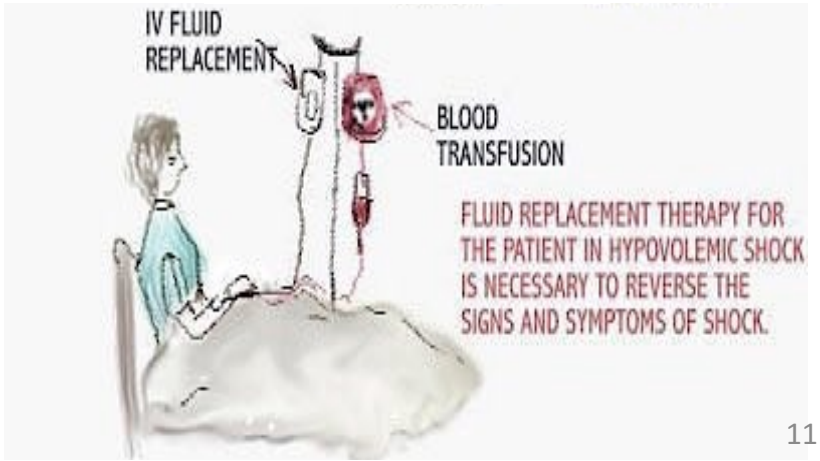
A. **If hemorrhage is the cause**: **stop the bleeding** by applying direct pressure.

B. **IV hydration:**

- Patients with class I shock usually do not require fluid resuscitation. Patients with class II shock benefit from fluids, and patients with classes III and IV require fluid resuscitation.
- Give fluid bolus followed by continuous infusion and reassess.
- The hemodynamic response to this treatment guides further resuscitative effort.

C. **For non-hemorrhagic shock**: blood is not necessary. Crystalloid solution with appropriate electrolyte replacement is adequate.

Think ABCs with any patient in "shock": Secure airway, breathing, and circulation.



# 2- Septic Shock:

## General Characteristics

- **Definition :** Septic shock is defined as hypotension induced by sepsis that persists despite adequate fluid resuscitation. This results in hypo-perfusion and can ultimately lead to multiple organ system failure and death.
- **Causes:** include (but are not limited to) **Pneumonia, Pyelonephritis, Meningitis, Abscess Formation, Cholangitis, Cellulitis, and Peritonitis.**
- **Complications :** Adult Respiratory Distress Syndrome, Acute Tubular Necrosis (ATN) , DIC (Disseminated Intravascular Coagulation), multiple organ failure, or death .

## Pathophysiology

**Severe Infection with Release Of Microbial Products**

- Release of vasoactive mediators

**Hyperdynamic State**

- Peripheral vasodilation
- Increased cardiac output
- Fever, tachycardia, tachypnea, warm skin

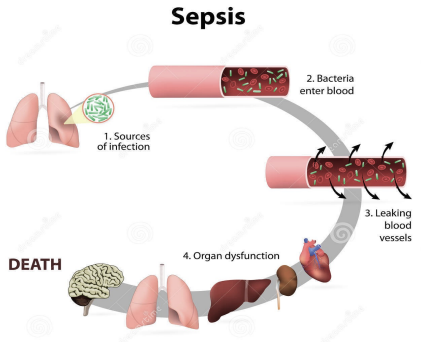
**Maintenance Of Intravascular Volume**

- Hyperdynamic shock

OR

**Failure To Maintain Intravascular Volume**

- Hypodynamic shock
- Cool skin, tachycardia, hypotension, oliguria




## Clinical Features

1. **Manifestations related to cause of sepsis** (e.g., pneumonia, urinary tract infection, peritonitis)
2. **Signs of SIRS** (see the next slide)
3. **Signs of shock** (hypotension, oliguria, lactic acidosis).
4. Patient may have a **fever** or may be **hypothermic** (hypothermia is more common in the very young, elderly, debilitated, and immuno-compromised).

## Diagnosis

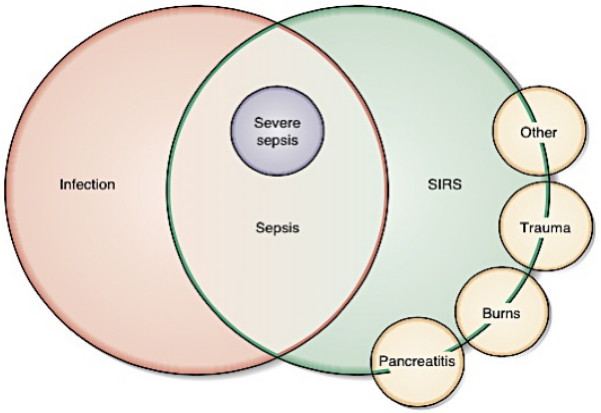
- Septic shock is essentially a clinical diagnosis.
- Confirmed by positive blood cultures, but negative cultures are common.
- A source of infection can aid in diagnosis, but there may be no confirmed source in some cases.

## Management

1. **Initially, IV antibiotics (broad spectrum) at maximum dosages**. Antibiotics for more rare organisms or antifungal medications may be required if there is no clinical response or if suspicion for an atypical organism (i.e., immuno-compromised). If cultures are positive, antibiotics can be narrowed based on sensitivity testing.
2. Surgical drainage if necessary.
3. **Fluid administration:** to increase mean BP.
4. Vasopressors may be used if hypotension persists despite aggressive IV fluid resuscitation.
   a. Dopamine is typically the initial agent.
   b. If dopamine does not increase the BP, norepinephrine may be given.

## ★ Systemic Inflammatory Response Syndrome (SIRS):

- ✓ The patients demonstrate a similar response as sepsis but **without infective agents**.
- ✓ The criteria are : (two or more to call it SIRS)
  - Temperature >38 or < 36 **( could be hypothermia or hyperthermia)**
  - Heart rate >90
  - RR > 20 or a pco2 < 34 mmHg (4.3 kpa)
  - WBC > 12,000 0r < 4,000 with more than 10% bands




# 3- Anaphylactic Shock:

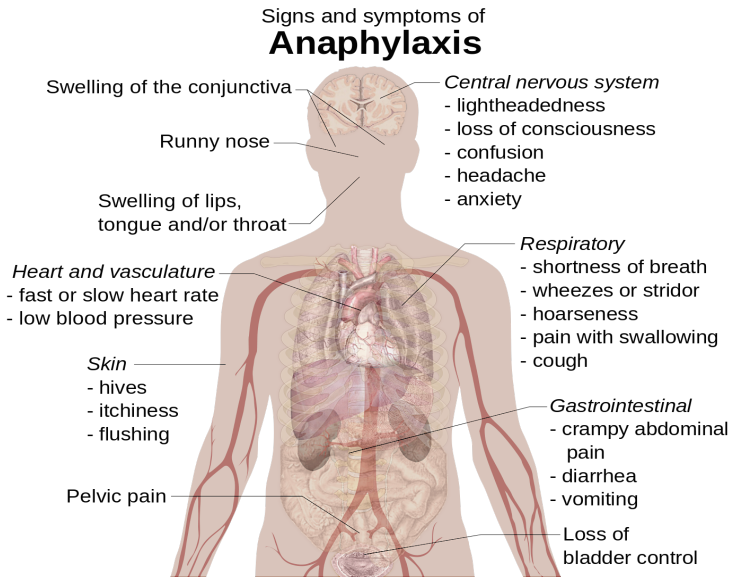
- This is a severe systemic hypersensitivity reaction following exposure to an agent (allergen) triggering the release of vasoactive mediators (Histamine, kinins, prostaglandins)
- **Anaphylactic shock results from vasodilation, intravascular volume redistribution, capillary leak and a reduction in cardiac output.**

★ **Prominent Physical Findings:**

- Urticaria and anigoedema (especially around lips).
- Laryngeal edema (stridor), wheezing.

## ✸ Management Of Anaphylaxis :

1. Stop administration of causative agent (drug/fluid)
2. Lie patient flat, feet elevated.
3. **ABCs**—Secure the airway; intubation may be necessary .
4. **Give Adrenaline (epinephrine):**
   - 0.5-1.0 mg IM, experienced doctors could use IV adrenaline
5. **Give Antihistamines** (both H1 and H2 blockers) and **Corticosteroids** (although they have a minimal effect in hyper-acute condition).
   - **Antihistamines:** Chlorphenamine
   - **Corticosteroid:** Hydrocortisone
6. **Supportive Care :** IV fluids (crystalloid or colloid), oxygen (100% O2)
7. Second-line therapy.



# 4- Cardiogenic Shock:

## General Characteristics

- When the total amount of myocardium affected reaches a critical point, myocardial function begins to deteriorate.
- While stroke volume decreases, the heart rate increases in an effort to maintain cardiac output (CO = SV x HR) But increased HR is limited and CO falls to levels that are inadequate to support end-organ function
- Coronary perfusion decreases and this in turn causes progressive myocardial ischemia with progression of myocardial injury.
- unlike hypovolemic shock circulating volume is typically normal or increased with increased circulating Angiotensin-II and aldosterone

## Pathophysiology (Macrocirculation)

- Typically presents with signs of a **low- output state**.
- If associated with left ventricular failure, there may be **Pulmonary Edema**.

**How do you know if it is cardiogenic or not?**
1. SOB
2. Raised JVP?
3. Lower limb edema

## Causes

★ **Caused by the progressive loss of myocardium, because of:**

1. **After Acute Myocardial Infarction**: (most common cause)
2. Pericardial Tamponade (compression of heart)
3. Tension Pneumothorax (compression of heart)
4. Primary arrhythmias (Ineffective cardiac contraction)
5. **Mechanical Abnormalities** (valvular defects, ventricular septal defect)
6. Massive Pulmonary Embolism leading to RVF.
7. **Myocardial Disease** (cardiomyopathies, myocarditis)

## Clinical Features

- **Typical findings seen in shock** (altered sensorium, pale cool skin, hypotension, tachycardia, Tachypnea, Oliguria)
- **Engorged Neck Veins**—Venous pressure is usually elevated.
- **Pulmonary congestion.**

## Management

1. **ABCs .**
2. **Identify And Treat Underlying Cause :**
   a. **Acute MI:** Standard treatment with aspirin, heparin • or, emergent revascularization with PCI (or CABG)
   b. **Cardiac Tamponade, Pericardiocentesis, Valvular Abnormalities :** surgery
   c. **Treatment of arrhythmias .**
3. **Vasopressors** (Dopamine is often the initial drug used and Norepinephrine used in severe or resistant cases)
4. **IV fluids are likely to be harmful if left ventricular pressures are elevated,** the patient might develop pulmonary edema . .

# 5- Neurogenic Shock :

- **Neurogenic shock results from a failure of the sympathetic nervous system to maintain adequate vascular tone (sympathetic denervation).**

★ **Causes include**: spinal cord injury, severe head injury, spinal anesthesia, pharmacologic sympathetic blockade.

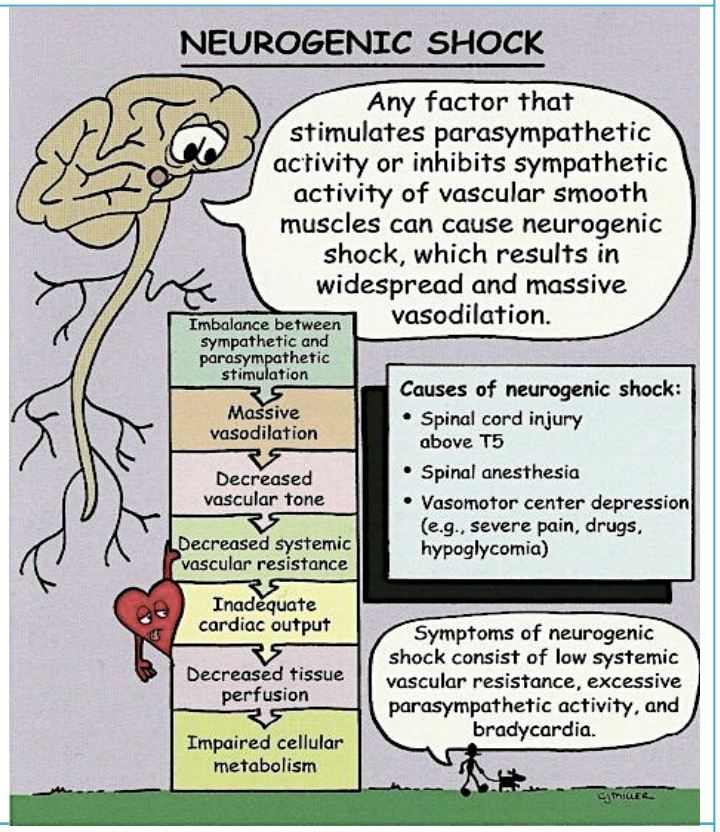
## Pathophysiology

**Traumatic disruption of sympathetic efferent nerve fibers results in:**

- **Loss of vasomotor tone.**
- Peripheral vasodilation.
- Fall in systemic vascular resistance.

## Clinical Features

- **Hypotension** (caused by peripheral vasodilation)
- **Bradycardia:**
  - As a result of loss of sympathetic stimulation (unopposed vagal tone)
  - **It is the only type of shock that lead to Bradycardia.**
- **Warm, well-perfused skin.**
- **Cardiac output is decreased, SVR** (Systemic Vascular Resistance) **low.**
- **Urine output low or normal.**




## Management

1. **Assessment Of Airway** (Hypoxemia must be prevented and, if present, rapidly corrected by maintaining a clear airway (e.g. head tilt, chin lift) and administering high flow oxygen)
2. **Stabilization of the entire spine** (Supine or Trendelenburg position)
3. **Volume Resuscitation** (**circulation :** Initial resuscitation should be targeted at arresting haemorrhage and providing fluid (crystalloid or colloid) to restore intravascular volume and optimize cardiac preload)
4. Role Out other causes of shock.
5. High dose corticosteroids.
6. Maintain body temperature.

## Diagnosing Shock State Based On Hemodynamic Parameters:

| Type | Central Venous Pressure | Cardiac Output | Systemic vascular resistant (SVR) |
|---|---|---|---|
| Hypovolemic | Decreased | Decreased | Increased |
| Cardiogenic | Increased | Decreased | Normal or Increased |
| Septic | Decreased or increased | Increased | Decreased |
| Traumatic | Decreased | Decreased or increased | Decreased or increased |
| Neurogenic | Decreased | Decreased | Decreased |
| Hypoadrenal | Decreased or increased | Decreased or increased | Decreased or increased |

## Principles Of Resuscitation

- **Maintain ventilation:** ensure oxygen delivery.
- **Enhance perfusion.**
- **Treat underlying cause.**

## How to correct each of the following:

| | |
|---|---|
| **Heart Rate(HR) & Stroke Volume(SV)** | Inotropes + fluids (fluids increase SV) |
| **Hemoglobin** | Transfuse |
| **SO2 + PaO2** | Partially dependent on FIO2 (fractional inspired oxygen) and pulmonary status. |

## Effects Of Shock At The Organ Level

| | |
|---|---|
| **Renal** | • **Renal Hypoprefusion** → activation of Rennin–Angiotensin system.<br>• **Oliguria** → could progress to anuria<br>• **Acute Renal Failure** → ↑ urea, ↑ creatinine, ↑ K+ & Metabolic Acidosis. |
| **Respiratory** | • Tachypnea<br>• Hypoxia<br>• **Pulmonary Edema** (Common in cardiogenic shock).<br>• Acute lung injury & Adult Respiratory Distress Syndrome. |
| **Liver** | • Liver failure.<br>• Acute ischemic hepatitis. |
| **GI tract** | Failure of intestinal barrier (sepsis, bleeding) |

# 3rd :Metabolic Response to Surgery

## ✸ Features Of The Metabolic Response To Injury:

- Historically, the response to injury was divided into two phases: 'ebb' and 'flow'. In the ebb phase during the first few hours after injury patients were cold and hypotensive (shocked). When intravenous fluids and blood transfusion became available, this shock was sometimes found to be reversible and in other cases irreversible. If the individual survived the ebb phase, patients entered the flow phase which was itself divided into two parts. The initial catabolic flow phase lasted about a week and was characterized by a high metabolic rate, breakdown of proteins and fats, a net loss of body nitrogen (negative nitrogen balance) and weight loss. There then followed the anabolic flow phase, which lasted 2–4 weeks, during which protein and fat stores were restored and weight gain occurred (positive nitrogen balance). Our modern understanding of the metabolic response to injury is still based on these general features.

## ✸ Factors Mediating The Metabolic Response To Injury:

**The Acute Inflammatory Response :**

1. Inflammatory cells (macrophages, monocytes, neutrophils)
2. Pro-inflammatory cytokines and other inflammatory mediators
3. Endothelium

**Endothelial cell activation**

1. Adhesion of inflammatory cells.
2. Vasodilatation .
3. Increased permeability.

**Nervous System**

Afferent nerve stimulation and sympathetic nervous system activation

**Endocrine**

1. Increased secretion of stress hormones
2. Decreased secretion of anabolic hormones

**Bacterial Infection**

The topic in this slide and the upcoming slides were NOT mentioned in the lecture, yet they are required according to Dr. Adnan's objectives

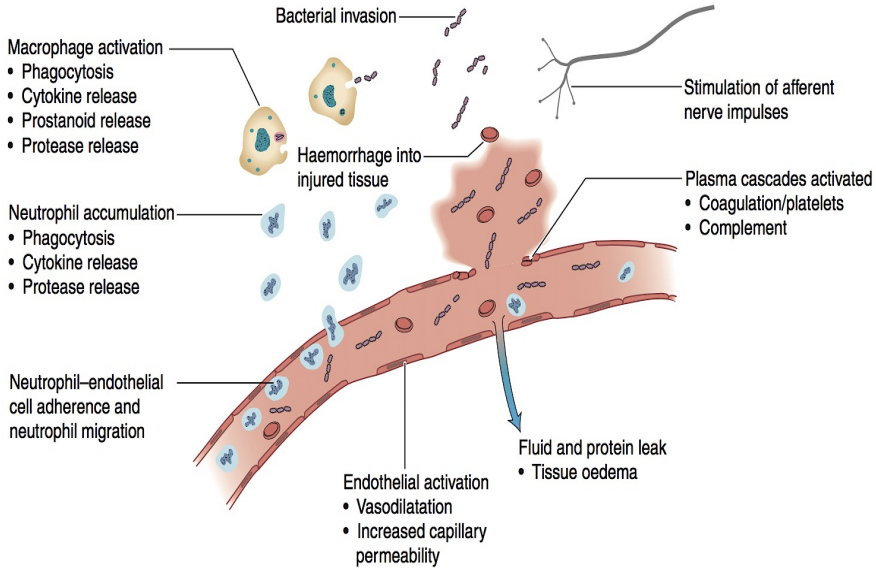
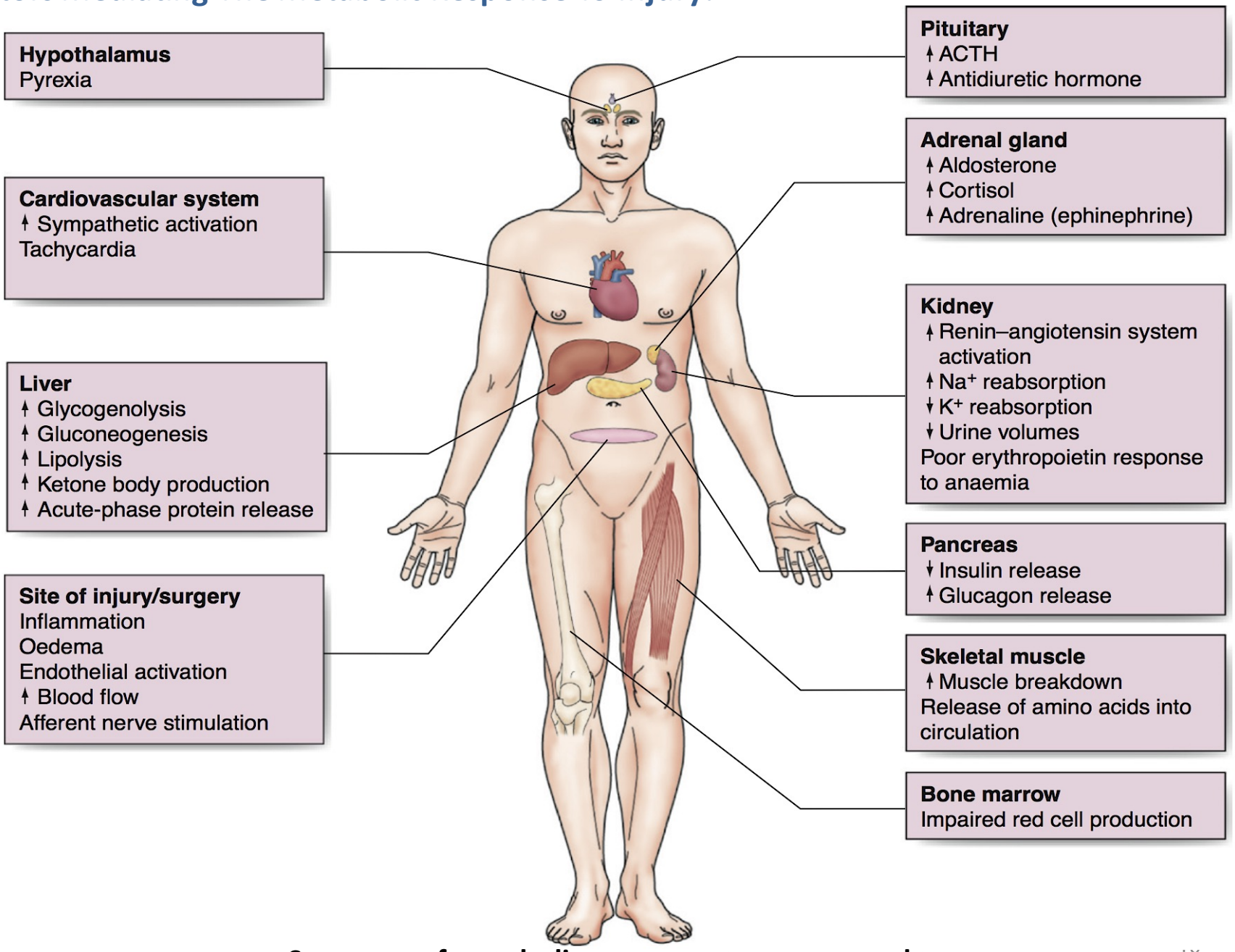



Fig. 1.1 Key events occurring at the site of tissue injury.

# Factors Mediating The Metabolic Response To Injury:




**Summary of metabolic responses to surgery and trauma**

# SUMMMARY

**Table 1.16 Clinical assessment of shock**

| | |
|---|---|
| **Conscious level** | Restlessness, anxiety, stupor and coma are common features and suggest cerebral hypoperfusion |
| **Pulse** | Low volume, thready pulse consistent with low-output state; high volume, bounding pulse with high-output state |
| **Blood pressure** | Changes in diastolic may precede a fall in systolic blood pressure, with ↓ diastolic in sepsis and ↑ in hypovolaemic and cardiogenic shock |
| **Peripheral perfusion** | Cold peripheries suggest vasoconstriction (↑ SVR); warm peripheries suggest vasodilation (↓ SVR) |
| **Pulse oximetry** | Hypoxemia common association of all forms of shock and ↓tissue $O_2$ delivery |
| **ECG monitoring** | Myocardial ischaemia commonest cause of cardiogenic shock but common in all forms of shock |
| **Urine output** | < 0.5 ml/kg/h suggestive of renal hypoperfusion |
| **CVP measurement** | Low CVP with collapsing central veins consistent with hypovolaemia |
| **Arterial blood gas** | Metabolic acidosis and ↑ lactate consistent with tissue hypoperfusion |

In isolation, single measurements are not helpful. Measurements are far more useful when used in combination with the findings of a detailed clinical examination. Observation of trends over time, together with the response to therapeutic interventions (e.g. a fluid challenge) is key to the successful management of shock.

SUMMARY BOX **1.12**

**Clinical effects of shock**

**Nervous system**
- Restlessness, confusion, stupor, coma
- Encephalopathy and/or delirium common in sepsis

**Renal**
- Renal hypoperfusion → activation of rennin–angiotensin system
- Oliguria (< 0.5 ml/kg/h urine) → anuria
- Acute renal failure → ↑ urea, ↑ creatinine, ↑ $K^+$ & metabolic acidosis

**Respiratory**
- Tachypnoea
- ↑ Ventilation/perfusion (V/Q) mismatch & ↑ shunt → hypoxia
- Pulmonary oedema (common in cardiogenic shock) → hypoxia
- Acute lung injury and acute respiratory distress syndrome → hypoxia

**Cardiovascular**
- ↓ Diastolic pressure → ↓ coronary blood flow
- ↓ Myocardial oxygen delivery → myocardial ischaemia → ↓ contractility & ↓ CO
- Acidosis, electrolyte disturbances and hypoxia predispose to arrhythmias
- Widespread endothelial cell activation → microcirculatory dysfunction

**Gastrointestinal**
- Splanchnic hypoperfusion → breakdown of gut mucosal barrier
- Stress ulceration
- Translocation of bacteria/bacterial wall contents into blood stream → SIRS
- Acute ischaemic hepatitis.

SUMMARY BOX **1.11**

**Sepsis – definitions**

**Systemic inflammatory response (SIRS)**

SIRS is defined as 2 or more of the following criteria:
- Temperature > 38C° or < 36°C
- Heart rate > 90 beats per minute
- Respiratory rate > 20 per minute or $P_aCO_2$ < 4.5 kPa
- White cell count > 12 or < 4 × $10^9$/l or > 10% immature neutrophils

**Bacteraemia**
- The presence of viable bacteria in the blood. The presence of other pathogens in the blood is described in a similar way i.e. viraemia, fungaemia and parasitaemia.

**Sepsis**
- The systemic response to infection. Defined as SIRS with confirmed or presumed infection.

**Severe sepsis**
- Sepsis with evidence of organ dysfunction.

**Septic shock**
- Sepsis-induced hypotension and/or tissue hypoperfusion (e.g. oliguria, lactic acidosis) despite adequate fluid resuscitation.


# Questions

- **What are the causes of shock?**
  1. Reduced oxygen delivery
  2. Maldistribution of blood flow
  3. Impaired oxygen utilization
  4. Increased tissue oxygen requirement

- **What is the most common type of shock in surgical setting?**

Hypovolemic

- **How does sepsis usually arise?**

Localized infection

- **What is the common sites of infection leading to sepsis?**

Lung > Abdomen > urinary tract > skin

- **List some causes of cardiogenic shock?**

MI, Arrhythmia, valvular disease, cardiac tamponade, PE, tension pneumothorax

- **What are the best indicators of tissue perfusion?**

Urine output & mental status

- **During first 24-48 hours after surgery, there is no drop in potassium levels (hypokalemia), why?**

Because there is shifting of potassium from intracellular compartment

- **Pancreatitis can lead to which type of shock?**

Hypovolemic

- **What happens to the lung in systemic inflammatory response (SIR)?**

ARDS (adult respiratory distress syndrome)

- **Why does decreased pulse pressure occur early in hypovolemic shock?**

Pulse pressure = systolic pressure – diastolic pressure
Pulse pressure is reduced due to vasoconstriction which leads to elevated diastolic BP

- **What are the signs and symptoms of neurogenic shock?**
  1. Hypotension + bradycardia
  2. neurological deficits


# MCQs

**Q1** Which one of these parameters will appear first and can be diagnostic for shock :

- A) Hypotension
- B) Bradycardia
- C) Decreased tissue perfusion
- D) Tachycardia

**Q2** The most sensitive tissue to ischemia is :

- A) Muscle
- B) Nerve
- C) Skin
- D) Adipose tissue

**Q3** Which one of the following does not cause hypovolemic shock :

- A) Hemorrhage
- B) Trauma
- C) Surgery
- D) Myocardial infarction
- E) Burns

**Q4** A 25 y/o driver sustained a car accident presented to the ER with flaccid paralysis, bradycardia, and hypotension. The most likely diagnosis is :

- A) Neurogenic shock
- B) Cardiogenic shock
- C) Hypovolemic shock
- D) None of the Above

**Q5** The commonest cause of the previous case is :

- A) Massive external bleeding
- B) Ischemic heart disease
- C) Injury to the high thoracic spine
- D) Internal bleeding

**Q6** Which of the following is the only type of shocks that we don't give fluid as a treatment :

- A) Cardiogenic shock
- B) Neurogenic shock
- C) Septic shock
- D) Hypovolemic shock
- E) Anaphylactic shock

**Q7** Which of the following hormonal changes in NOT TRUE during metabolic response to injury :

- A) Increase Growth Hormone
- B) Increase Glucagon
- C) Increase Insulin
- D) Decrease Testosterone

**Q8** Which of the following is a negative acute phase protein :

- A) Albumin
- B) Plasminogen
- C) C-reactive
- D) Haptoglobin

**Q9** During metabolic response to injury, most of the proteins are lost through which of the following pathways :

- A) Stool
- B) Skin
- C) Enterohepatic circulation
- D) Urine

Answers : 1.D 2.B 3.D 4.A 5.C 6.A 7.C 8.A 9.D

# Cases Mentioned By the Doctor:

**Case 1: Circulatory Shock**

10 y/o female fell off bike riding down a hill. Initially well but 4 hrs later complained of abdominal pain and left shoulder pain.

- On examination:
  - Vital signs (VS): BP 90/60, P 120 (tachycardic), RR 30 (tachypneic), T 100.1, O2 sat 95% (low)
  - General (GEN): pale, anxious
  - Lung: clear to auscultation
  - Precordium (COR): tachycardic with murmur best heard at base
  - Abdomen (ABD): diffuse tenderness without peritonitis or mass
- Labs: Hb 7.5 (low)
- Hemodynamics:

| | |
|---|---|
| **Central venous pressure** | Decreased |
| **Cardiac output** | Decreased |
| **Systemic vascular resistance** | Decreased |

- Abdominal CT: splenic laceration with free peritoneal fluid
- Patient is in **respiratory failure**:


- **Treatment** of respiratory failure:
  1) Primary resuscitation
  2) Oxygen
  3) Mechanical ventilation if necessary

**Case 2: Septic Shock**

15 y/o male with a 4 day history of abdominal pain, N/V and anorexia

- On examination:
  - VS: BP 70/60 (low), P 130 (high), RR 28 (high), T102.4, O2 sat 99%
  - GEN: moderate distress from abdominal pain
  - COR: tachycardic
  - ABD: diffuse tenderness w peritonitis
- Labs:
  - WBC 19,600 (high), 90% segments
  - Hb 14.2
- Hemodynamics:

| | |
|---|---|
| **Cardiac output** | Increased |
| **Systemic vascular resistance** | Decreased |

- **Dx**: perforated appendicitis

# Continue..

**Case 3: Neurogenic Shock**

17 y/o male, diving into water

- On examination:
  - VS: BP 90/60 (low), P 110 (high), RR 24 (high)
  - Paralysis below C5
- Hemodynamics:

| | |
|---|---|
| **Central venous pressure** | Decreased |
| **Cardiac output** | Decreased |
| **Systemic vascular resistance** | Decreased |

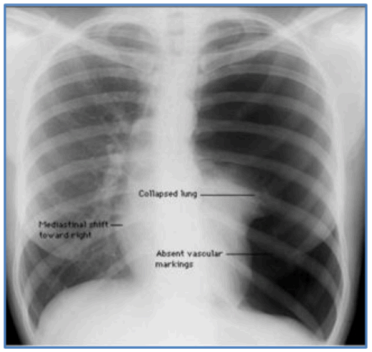
- Cervical X-ray: C5 fracture

**Case 4: Cardiogenic Shock**

17 y/o male, training for track team

- On examination:
  - VS: BP 70/50 (low), P 140 (high), RR 35 (high), $O_2$ sat 88%
  - Absent breath sounds in left lung field, distended neck veins
- Dx: tension pneumothorax
- Hemodynamics:

| | |
|---|---|
| **Central venous pressure** | Increased |
| **Cardiac output** | Decreased |
| **Systemic vascular resistance** | Normal |



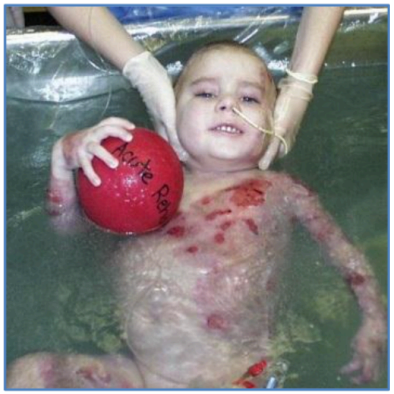

**Case 5: Capillary Leak Syndrome**

3 y/o male, clothes ignited from roaster

- On examination:
  - VS: BP 60/60 (low), P 170 (high), RR 35 (high), T102.4, O2 sat 89%
  - GEN: moderate distress
  - LUNG: tachypneic, clear to auscultation
  - COR: tachycardic, regular
  - SKIN: 60% TBSA partial and full thickness burn
- Hemodynamics:

| | |
|---|---|
| **Cardiac output** | Decreased |
| **Systemic vascular resistance** | Increased |

- **Dx**: 60% of total body surface area (TBSA) burn; hypovolemic shock (loss of fluid into interstitium, called "third spacing")
- **Rx**: MAINTAIN VENTILATION



Increased O2 demand (sepsis, hypovolemia, trauma etc) → Hyperventilation → Repiratory fatigue → Respiratory failure (=acidosis, hypoxia, coma) → Death

**What is capillary leak syndrome?**

1. Vasodilatation
2. A-V shunting
3. Maldistribution of flow
4. Increased capillary permeability + interstitial edema
5. Decreased oxygen extraction
6. Primary defect of oxygen utilization at cellular level

# Thank You..

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