**Embolism**

**Definition:** An embolus is a detached intravascular solid, liquid or gaseous mass that is carried by blood to sites distant from its point of origin. After traveling via the blood, the embolus can obstruct a vessel.

**Causes of embolism:**

An embolus can arise from:

1. Thrombus (99% of emboli arise from a thrombus. Such an embolus is called thromboembolus)
2. Platelets aggregates
3. Fragment of material from ulcerating atheromaus plaque
4. Fragment of a tumor
5. Fat globules
6. Bubbles of air
7. Amniotic fluid
8. Infected foreign material
9. Bits of bone marrow

The consequences of thromboembolism include ischemic necrosis (infarction) of downstream tissue.

Depending on the site of origin, emboli may lodge anywhere in the vascular tree; the clinical outcomes are best understood from the standpoint of whether emboli lodge in the pulmonary or systemic circulations.

**Type of emboli**

1. Thromboembolism
2. Fat embolism
3. Air embolism
1- Thromboembolism

Based on its sites of origin & impaction, thromboembolism can be divided into:

a) Pulmonary thromboembolism (PTE)

95% of PTE arise from thrombi in the deep leg veins. The thromboembolism will travel along with the venous return & reach the right side of the heart. From there, it will go into the pulmonary trunk & pulmonary arteries. Depending on the size of the embolus and on the state of pulmonary circulation, the pulmonary embolism can have the following effects:

1. If the thrombus is large, it may block the outflow tract of the right ventricle or the bifurcation of the main pulmonary trunk (saddle embolus) or both of its branches, causing sudden death by circulatory arrest. Sudden death, right side heart failure (cor pulmonale), or cardiovascular collapse occurs when 60% or more of the pulmonary circulation is obstructed with emboli.

2. If the embolus is very small (as in 60-80% of the cases), the pulmonary emboli will be clinically silent. Embolic obstruction of medium sized arteries manifests as pulmonary hemorrhage but usually does not cause infarction because of dual blood inflow to the area from the bronchial circulation.

3. If the cardiorespiratory condition of the patient is poor (i.e., if the patient previously had cardiac or pulmonary disease), then obstruction of a medium sized pulmonary artery by a medium-sized embolus can lead to pulmonary infarction.

4. Recurrent thromboembolism can lead to pulmonary hypertension in the long run. A patient who has had one pulmonary embolus is at high risk of having more.

b) Systemic thromboembolism

Systemic thromboembolism refers to emboli travelling within arterial circulation & impacting in the systemic arteries.

- Most systemic emboli (80%) arise from intracardiac mural thrombi. In turn, two thirds of intracardiac mural thrombi are associated with left ventricular wall infarcts and another quarter with dilated left atria secondary to rheumatic valvular heart disease.

- The remaining (20%) of systemic emboli arise from aortic aneurysm, thrombi on ulcerated atherosclerotic plaques,

- Unlike venous emboli, which tend to lodge primarily in one vascular bed (the lung), arterial emboli can travel to a wide variety of sites. The major sites for arteriolar embolization are the lower extremities (75%) & the brain (10%), with the rest lodging in the intestines, kidney, & spleen. The emboli may obstruct the arterial blood flow to the tissue distal to the site of the obstruction. This obstruction
may lead to infarction. The infarctions, in turn, will lead to different clinical features which vary according to the organ involved.

2- Fat Embolism

Fat embolism usually follows fracture of bones and other type of tissue injury. After the injury, globules of fat frequently enter the circulation. Although traumatic fat embolisms occur usually it is as symptomatic in most cases and fat is removed. But in some severe injuries the fat emboli may cause occlusion of pulmonary or cerebral microvasculature and fat embolism syndrome may result. Fat embolism syndrome typically begins 1 to 3 days after injury during which the raised tissue pressure caused by swelling of damaged tissue forces fat into marrow sinosoid & veins. The features of this syndrome are a sudden onset of dyspnea, blood stained sputum, tachycardia, mental confusion with neurologic symptoms including irritability & restlessness, sometimes progress to delirium & coma.

3 - Air embolism

Gas bubbles within the circulation can obstruct vascular flow and cause distal ischemic injury almost as readily as thrombotic masses. Air may enter the circulation during:

• Obstetric procedures
• Chest wall injury
• In deep see divers & under water construction workers.
• Neck wounds penetrating the large veins
• Cardio thoracic surgery.
• Arterial catheterization& intravenous infusion.
Infarction

**Definition:** infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply or venous drainage in a particular tissue.

Nearly 99% of all infarcts result from thrombotic or embolic events. Other mechanisms include [almost all of them are arterial in origin]:

- Local vasospasm
- Expansion of atheroma due to hemorrhage in to athermatous plaque.
- External compression of the vessels. e.g trauma
- Entrapment of vessels at hernial sacks etc.

**The development & the size of an infarct are determined by the following factors:**

A. **The nature of vascular supply**

The following organs have a dual blood supply.

- Lung → pulumonary artery
  → Bronchial artery
- Liver → hepatic artery
  → Portal vein
- Hand & forearm
  → Radial arteries
  → Ulnar arteries.

The effect of such a dual blood supply is that if there is obstruction of one of the arterial supplies, the other one may offset the rapid occurrence of infarction in these organs unlike the renal & splenic circulations which have end arterial supply. Infarction caused by venous thrombosis is more likely to occur in organs with single venous outflow channels, such as testis &ovary.

B. **Rate of development occlusion**

Slowly developing occlusions are less likely to cause infraction since they provide time for the development of collaterals.
C: Tissue susceptibility to hypoxia:

The susceptibility of a tissue to hypoxia influences the likelihood of infarction. Neurons undergo irreversible damage when deprived of their blood supply for only 3 to 4 minutes. Myocardial cells die after 20-30 minutes of ischemia. Fibroblasts are more resistant, especially those in the myocardium.

D: Oxygen content of blood

Partial obstruction of the flow of blood in an anemic or cyanotic patient may lead to tissue infarction.

E: The severity & duration of ischemia.

Types of infarcts

Infarcts are classified depending on:

A) The basis of their color (reflecting the amount of hemorrhage) into:

1. Hemorrhagic ((Red) infarcts
2. Anemic (White) infarcts

B) The presence or absence of microbial infection into:

1. Septic infarcts
2. Bland infarcts

1. Red infarcts occur in:

a) Venous occlusions as in ovarian torsion
b) Loose tissues such as the lung which allow blood to collect in infarct zone.
c) Tissues with dual circulations (eg. the lung), permitting flow of blood from unobstructed vessel in to necrotic zone.
d) In tissues that were previously congested because of sluggish outflow of blood.
e) When blood flow is reestablished to a site of previous arterial occlusion & necrosis.

2. White infarcts occur in:

a) Arterial occlusion in organs with a single arterial blood supply.
b) Solid organs such as the heart, spleen, & kidney, where the solidity of the tissue limits the amount of hemorrhage that can percolate or seep in to the area of ischemic necrosis from the nearby capillaries.
Morphology of infarcts

Gross:

<table>
<thead>
<tr>
<th>Red infarction</th>
<th>Pale infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newly formed with in (2-3) days</td>
<td>After (2-3) days</td>
</tr>
<tr>
<td>Red in color RBCs not lysis</td>
<td>Pale because of RBCs lysis</td>
</tr>
<tr>
<td>Protruded</td>
<td>Depressed</td>
</tr>
<tr>
<td>occur in spongy tissue ex: lung ,brain</td>
<td>Occur in solid tissue ex: kidney.</td>
</tr>
</tbody>
</table>

Microscopy: All infarcts are triangular -shaped with the occluded vessel at the apex and the periphery of the organ forming the base of the wedge. The infarction will induce inflammation in the tissue surrounding the area of infarction. Following inflammation, some of the infarcts may show recovery, however, most are ultimately replaced with scars except in the brain. The dominant histologic feature of infarction is ischemic coagulative necrosis. The brain is an exception to this generalization, where liquifactive necrosis is common.

Clinical examples of infarction:

A. Myocardial infarction
- usually results from occlusive thrombosis supervening on ulcerating atheroma of a major coronary artery.
- is a white infarct.
- can cause sudden death, cardiac failure, etc...

B. Cerebral infarcts
- May appear as pale or hemorrhagic
- increase in intracranial pressure may occur due to swelling of large cerebral infarction, as recent infarcts are raised above the surface since hypoxic cells lack the ability to maintain ionic gradients & they absorb water & swell.
- Is one type of cerebrovascular accidents (CVA) or stroke which has various clinical manifestations.
C. Lung infarcts

- Are typically dark red & conical (wedge-shaped).
- Can cause chest pain, hemoptysis, etc…

D. Splenic infarcts

- Conical & sub capsular
- Initially dark red later turned to be pale.
Shock

**Definition:** Shock is a state in which there is failure of the circulatory system to maintain adequate cellular perfusion resulting in widespread reduction in delivery of oxygen & other nutrients to tissues. In shock, constitutes systemic hypoperfusion due to reduction either in cardiac output or in the effective circulating blood volume. The end results are hypotension followed by impaired tissue perfusion and cellular hypoxia.

**Clinical course of shock**

- Patient with shock may manifest as having a weak and rapid pulse, tachypnea, & cool, clammy, cyanotic skin. In septic shock, the skin will initially be warm & flushed because of peripheral vasodilation. The patient may present with confusion, restlessness, decreased urine output, coma, and death.

- Adequate organ perfusion depends on arterial blood pressure (BP) which, in turn, depends on:
  1. Cardiac output (CO)
  2. Peripheral vascular resistance (PVR)

**Classification of shock**

Shock can be divided into:

A. Hypovolemic shock
B. Cardiogenic shock
C. Distributive shock

**A. Hypovolemic shock**

**Definition:** This is shock caused by reduced blood volume. Reduction in circulating blood volume results in the reduction of the preload which leads to inadequate left ventricular filling, reflected as decreased left & right ventricular end diastolic volume and pressure. The reduced preload culminates in decreased cardiac out put which leads to widespread tissue perfusion (shock).

**Causes of hypovolemic shock include:**

a) Hemorrhage (blood loss).

b) Diarrhea & vomiting (water lose, cholera, diabetic, coma).

c) Burns

d) Trauma

e) Loss of plasma
The effect of hemorrhage depends on the rate and amount of blood loss. Hypovolemic shock is the most common type of shock in clinical medicine. A normal healthy adult can lose 550ml (10% of blood volume) without significant symptoms. But loss of 25% or more of the blood volume (N=1250ml) results in significant hypovolemia.

B. Cardiogenic shock

**Definition:** This is shock that results from severe depression of cardiac performance. It primarily results from pump failure [myocardial failure].

**Causes of cardiogenic shock can be divided into:**

1. Acute myocardial infraction.
2. Myocarditis
3. Dilated cardiomyopathy/hypertrophic cardiomyopathy
4. Myocardial depression in septic shock
5. Rupture of aortic artery, aneurism of myocardium
6. Left ventricle outflow obstruction E.g. Aortic stenosis, hypertrophic cardiomyopathy
7. Reduction in forward cardiac output E.g. Aortic or mitral regurgitation
8. Arrhythmia

This can be called obstructive shock. The extra cardiac causes of cardiogenic shock can be caused by:

a) Pericardial tamponed (gross fluid accumulation in the pericardial space) results in a decreased ventricular diastolic filling → ↓CO

b) Tension pneumothorax (gas accumulation in pleural space)

This decreases the venous return by creating a positive pressure.

c) Acute massive pulmonary embolism occupying

d) Severe pulmonary hypertension
C. Distributive shock

**Definition:** Distributive shock refers to a group of shock subtypes caused by profound peripheral vasodilatation despite normal or high cardiac output.

**Causes of distributive shock**

1) Septic shock – the commonest among the group & clinically very important.
2) Neurogenic shock
   - Usually occurs in the setting of an aesthetic or spinal cord injury
3) Anaphylactic shock
   - Initiated by generalized IgE – mediated hypersensitivity response, associated with systemic vasodilatation & increased vascular permeability.
4) Endocrine shock
   - This is a type of shock that typically occurs in adrenal insufficiency.

**Bacteremia:** is the presence of viable bacteria in the blood as evidenced by blood culture. **Septicemia:** is systemic infection due the presence of microbes and their toxin the blood. **Sepsis:** is a systemic response to severe infection mediated via macrophage-derived cytokines that target end organ receptors in response to infection. It is also called SIRS.

**Septic shock**

**Definition:** This is a kind of shock caused by systemic microbial infection, most commonly by gram – negative infection (endotoxic shock) but can also occur with gram – positive or fungal infections.or can be defined as sepsis with

1. Hypotention,
2. Organ dysfunction, &
3. Unresponsiveness to fluid administration.

**Stages of shock**

Uncorrected shock passes through 3 important stages:

1) **An initial nonprogressive phase**
   
   It is also called a period of early compensatory period, during which compensatory mechanisms are activated & perfusion of vital organs maintained.
2. **Progressive stage (Established shock)**

- This is characterized by tissue hypoperfusion with onset of worsening circulatory & metabolic imbalances including acidosis.
- There is a widespread tissue hypoxia.
- Anaerobic glycolysis results in excessive lactic acid production.

3. **An irreversible stage**

- A stage at which, even if hemodynamic disorders are corrected survival is not possible.
- Transition to irreversible damage is mediated via various mechanisms.

**Morphology of septic shock:**

- All organs are affected in severe shock. In shock, there is widespread tissue hypoperfusion involving various organs such as the heart, brain, & kidney. This leads to widespread hypoxic tissue necrosis. The widespread tissue necrosis manifests as multiple organ dysfunction. Various organs may fail to perform their normal functions.