EMBOLISM:

An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.

Types according to composition of emboli:

1. Thromboembolism: 99% result from dislodged thrombus
2. Fat embolism
3. Air embolism
4. Nitrogen embolism
5. Amniotic fluid embolism

1%
2 TYPES / SIDES OF CIRCULATION: VENOUS & ARTERIAL (SYSTEMIC)
Origin of most venous thrombi = lower limbs

Target of most venous thrombi = lungs
Origin of most arterial thrombi = heart chambers

Target of most arterial thrombi = lower limbs (75%)
Types according to site of origin: venous and arterial (systemic) emboli

Emboli result in partial or complete vascular occlusion.

The consequences of thromboembolism include ischemic necrosis (infarction) of downstream tissue
Embolus derived from a lower extremity deep venous thrombosis and now impacted in a pulmonary artery branch.
PULMONARY THROMBOEMBOLISM

- 95% originate from deep veins of Lower Limbs

- Special terms:

  1. **Saddle embolus**: large embolus occluding at the bifurcation of Pulmonary artery trunk

  2. **Paradoxical embolus**: Passage of an embolus from venous to systemic circulation through interatrial septal defect or interventricular septal defect (AD or IVD)
Clinical consequence of pulmonary thromboembolism:

- Most pulmonary emboli (60% to 80%) are clinically asymptomatic because they are small
- If large → Pulmonary infarction
- When more than 60% of pulmonary vessels are obstructed → RVF, CV collapse → Sudden death
- Obstruction of medium sized arteries → Pulmonary hemorrhage
- If multiple emboli (showers of emboli) over a long time → Pulmonary Hypertension and right ventricular failure
Systemic (arterial) thromboembolism

- Emboli traveling within the arterial circulation
- 80% due to intracardiac mural thrombi (origin)
  
  causes: 
  - $\frac{2}{3}$ Lt. ventricular failure
  - $\frac{1}{4}$ Lt. atrial dilatation
  - Ulcerated atherosclerotic plaque
  - Aortic aneurysm
  - valve vegetation

- The major targets are:

  Lower limbs 75%; Brain 10%; Intestine; Kidneys; Spleen; etc... (any organ that has arterial supply!)
Fat embolism

- Causes:
  1. Skeletal injury (fractures of long bones)
  2. Adipose tissue Injury (massive fat necrosis like acute pancreatitis, etc...)

- Results:
  1. Mechanical obstruction of vessels
  2. Free fatty acid release from fat globules → local toxic injury to endothelium.

- In skeletal injury, fat embolism occurs in 90% of cases, but only 10% or less have clinical findings = Fat embolism ‘syndrome’
**Fat (Bone Marrow) Embolus** = Fat Globules + Hematopoietic Cells
Fat embolism ‘syndrome’ is characterized by:
A. Pulmonary Insufficiency
B. Neurologic symptoms
C. Anemia
D. Thrombocytopenia
E. Death in 10% of the case

- Symptoms appears 1-3 days after injury: Tachypnea, Dyspnea, Tachycardia and Neurological symptoms
Air Embolism

causes:
1. Obstetric procedures
2. Chest wall injury
3. Decompression sickness: in Scuba deep-sea divers ((nitrogen ))

Note: More then 100ml of air is required to produce clinical effect!
Air Embolism - Clinical consequence

1. **Painful joints**: rapid formation of gas bubbles within Skeletal Muscles and supporting tissues.

2. **Focal ischemia in brain and heart**

3. **Respiratory distress** (chokes) → Lung edema, Hemorrhage, atelectasis, emphysema

4. **caisson disease**: in scuba divers; gas emboli in the bones leads to multiple foci of ischemic necrosis, usually the heads of the femurs, tibias, and humeri
Amniotic fluid embolism

- **High Mortality Rate** = 20%-40%
- **Very rare** complication of labor
- infusion of amniotic fluid into maternal circulation via tears in placental membranes and rupture of uterine veins.

- **Symptoms:** sudden severe dyspnea, cyanosis, and hypotensive shock, followed by seizures, DIC and coma

- **Microscopic** Findings upon autopsy:
  fetal squamous cells, languo hair, fat, mucin .....etc within the maternal pulmonary microcirculation
AMNIOTIC FLUID EMBOLUS: KERATIN AND FETAL SQUAMOUS CELLS IN PULMONARY ARTERIOLES
INFARCTION

- infarct = an area of **ischemic necrosis** caused by occlusion of arterial supply or venous drainage in a particular tissue

- 99% of all infarcts → result from thrombotic or embolic events

- other mechanisms include: local **vasospasm**, expansion of an atheroma, **extrinsic compression** of a vessel (e.g., by tumor); vessel **twisting** (e.g., in testicular torsion or bowel volvulus; and traumatic vessel **rupture**
MORPHOLOGY OF INFARCTS

- Infarcts may be either red (hemorrhagic) or white (anemic) and may be either septic or bland.
- All tissue infarcts tend to be wedge shaped, with the occluded vessel at the apex and the periphery of the organ forming the base.
- The margins of both types of infarcts tend to become better defined with time.
- The dominant histologic characteristic of infarction is ischemic coagulative necrosis.
- Most infarcts are ultimately replaced by scar. Note: The brain is an exception, it results in liquefactive necrosis.
**Red infarcts:**

- occur in any of the following scenarios:
  1. **venous** occlusions (such as in ovarian torsion)
  2. **loose** tissues (like lung) that allow blood to collect in the infarcted zone;
  3. tissues with **dual** circulations (lung and small intestine),
  4. previously congested tissues because of **sluggish venous** outflow
  5. when flow is **re-established** to a site of previous arterial occlusion and necrosis
**White infarcts**

- occur with:
  1. *arterial* occlusions
  2. *solid* organs (such as heart, spleen, and kidney).

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**Septic infarctions;** occur when:

1. *bacterial vegetations* from a heart valve embolize
2. microbes seed an area of necrotic tissue.

- infarct is converted into *abscess* with a greater inflammatory response
Red and white infarcts.

A → lung
B → spleen
KIDNEY INFARCT

Kidney infarct replaced by a large fibrotic scar.
FACTORS THAT INFLUENCE DEVELOPMENT OF AN INFARCT

- **nature of the vascular supply**
- **rate of development of the occlusion** (collateral circulation)
- **tissue vulnerability to hypoxia**
  - Neurons undergo irreversible damage $\Rightarrow$ 3 to 4 minutes of ischemia.
  - Myocardial cells die after only 20 to 30 minutes of ischemia
- **the oxygen content of blood**