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 L.L

- Special variants:
  - Saddle embolus: at bifurcation of Pulmonary artery
  - Paradoxical embolus: Passage of an embolus from venous to systemic circulation through IAD, IVD
Clinical consequence of Pulmonary Thromboembolism:

- Most pulmonary emboli (60% to 80%) are clinically silent because they are small.
  - **Organization:** 60 – 80 %
  - **Sudden death, Right ventricle failure, CV collapse** when more than 60 % of pulmonary vessels are obstructed.
  - **Pulmonary hemorrhage:** obstruction of medium sized arteries.
  - **Pulmonary Hypertension and right ventricular failure** due to multiple emboli over a long time.
Systemic thromboembolism

- Emboli traveling within the arterial circulation
- 80% due to intracardiac mural thrombi
  - 2/3 Lt. ventricular failure
  - ¼ Lt. atrial dilatation
    - Ulcerated atherosclerotic plaque;
    - Aortic aneurysm;
    - valvular regurgitation

- The major targets are:
  1. Lower limbs 75%
  2. Brain 10%
  3. Intestines
  4. Kidneys
  5. Spleen
**Fat embolism**

- **Causes**
  1. *Skeletal injury* (fractures of long bones)
  2. *Adipose tissue Injury*

- Mechanical obstruction is exacerbated by free fatty acid release from the fat globules, causing 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** 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 and deep-sea divers ((nitrogen ))

More then 100ml of air is required to produce clinical effect.

Clinical consequence
1. Painful joints: due to rapid formation of gas bubbles within Sk. Muscles and supporting tissues.
2. Focal ischemia in brain and heart
3. Lung edema, Hemorrhage, atelectasis, emphysema, which all lead to Respiratory distress. (chokes)
4. caisson disease: gas emboli in the bones leads to multiple foci of ischemic necrosis, usually the heads of the femurs, tibias, and humeri
Amniotic fluid embolism
- Mortality Rate = 20%-40%
- Very rare complication of labor
- due to infusion of amniotic fluid into maternal circulation via tears in placental membranes and rupture of uterine veins.
- sudden severe dyspnea, cyanosis, and hypotensive shock, followed by seizures, DIC and coma
- Findings:
  Squamous cells, languo hair, fat, mucin .....etc within the pulmonary microcirculation
INFARCTION

- An infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage in a particular tissue.
- Nearly 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 aseptic.
- All 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 histological characteristic of infarction is ischemic coagulative necrosis.
- Most infarcts are ultimately replaced by scar. The brain is an exception, it results in liquefactive necrosis.
RED INFARCTS:

- occur in
  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).

Septic infarctions

- occur when bacterial vegetations from a heart valve embolize or when microbes seed an area of necrotic tissue.
- the infarct is converted into an abscess, with a correspondingly greater inflammatory response
Red and white infarcts.

A → lung

B → spleen
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)
- 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