

the concept	notes
<p>EMBOLISM(moving in the circulation)</p>	<ul style="list-style-type: none"> • 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% (from dislodged thrombus) • 2. Fat embolism • 3. Air/Nitrogen embolism • 4. Amniotic fluid embolism <p>→2.3.4 makes 1%.</p>
<p>Read slide 5</p>	<p>Read slide 5</p>
<p>THROMBOEMBOLISM</p>	<ul style="list-style-type: none"> • Types (according to site of origin): 1- venous Origin→lower limbs(DEEP VEINS THROMBI - dvt-) Target→lungs 2- arterial (systemic) emboli Origin→heart chambers (intracardiac mural thrombi) Target→lower limbs(75%)→other→Brain ; Intestine; Kidneys; Spleen; etc... (any organ that has arterial supply!) <p>causes :</p> <ul style="list-style-type: none"> -2/3 Lt. ventricular failure - ¼ Lt. atrial dilatation - Ulcerated atherosclerotic plaque - Aortic aneurysm - valve vegetationetc
<p>Pulmonary Thromboembolism</p>	<ul style="list-style-type: none"> • Saddle embolus→LARGE EMBOLUS OCCLUDING THE BIFURCATION OF PULMONARY ARTERY TRUNK (FATAL) • Paradoxical embolus→Passage of embolus from venous to systemic circulation through PFO, ASD or VSD • CONSEQUENCE READ SLIDE 14

Fat embolism
(FAT GLOBULES + HEMATOPOIETIC CELLS)

- Causes:
 1. **Skeletal injury: (long bones fractures)**
note → In skeletal injury, fat embolism occurs in 90% of cases, but only 10% or less have clinical findings = Fat embolism syndrome
 2. Adipose tissue Injury :(e.g. fat necrosis in acute pancreatitis)
- Results:
 - 1- Mechanical obstruction of vessels
 - 2- Free fatty acid release: toxic injury to endothelium + systemic immune response

Fat embolism 'syndrome'

- characterized by:
- Pulmonary Insufficiency (rapid breathing; shortness of breath)
 - Neurologic symptoms (mental confusion; lethargy; coma)
 - petechial rash (pinpoint rash, found on chest, head, and neck area due to bleeding under skin)
 - Fever
 - Anemia
 - Thrombocytopenia
 - Death in 10% of cases

Therapy

- no specific treatment
- Supportive care is the mainstay of therapy

read slide 21

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

Read slides 22-24

Amniotic fluid embolism
(KERATIN AND FETAL SQUAMOUS CELLS IN
PULMONARY ARTERIOLES)

see slides 27-28

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

Symptoms

sudden severe dyspnea, cyanosis, ARDS, and hypotensive shock, followed by seizures, DIC and coma

Microscopic Findings upon autopsy
fetal squamous cells, lanugo hair, fat, mucinetc within the maternal pulmonary microcirculation

INFARCTION

- infarct = **an area of ischemic necrosis** caused by occlusion of arterial supply or venous drainage
- 99% result from thrombotic/ embolic events
- other mechanisms: **local vasospasm**, **expansion of atheroma**, **extrinsic compression of vessel** (e.g., by tumor); **vessel twisting** (e.g. testicular torsion; bowel volvulus); and **traumatic vessel rupture**
- types : red (hemorrhagic) or white (anemic) and may be either septic or bland
- morphology : wedge-shaped (occluded vessel at the apex and periphery of organ forming the base)
- histologic hallmark : ischemic coagulative necrosis (ultimately replaced by scar) **but The brain is an exception (liquefactive necrosis)].**

note →margins of infarcts become defined with time

Read slides 31-35

Read slides 31-35