EMBOLISM AND INFARCTION

Dr. Nisreen Abu Shahin

Associate Professor of Pathology

Pathology Department

University of Jordan

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% (from dislodged thrombus)***
2. ***Fat embolism***
3. ***Air /Nitrogen embolism***
4. ***Amniotic fluid embolism***

} **1%**

2 TYPES /SIDES OF CIRCULATION: VENOUS & ARTERIAL (SYSTEMIC)

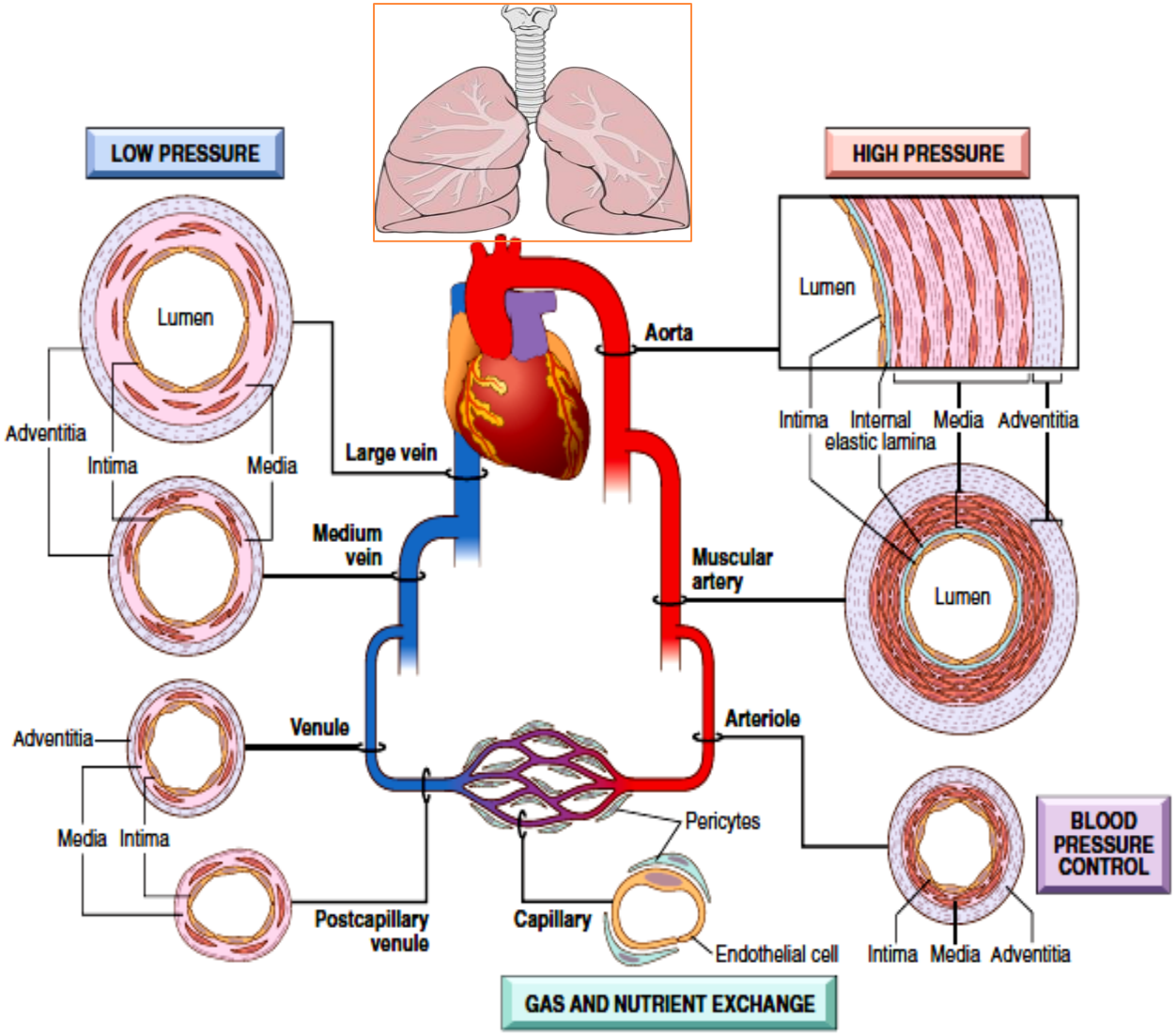


Figure 9–1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.

Origin of most venous thrombi = lower limbs

Target of most venous thrombi = lungs

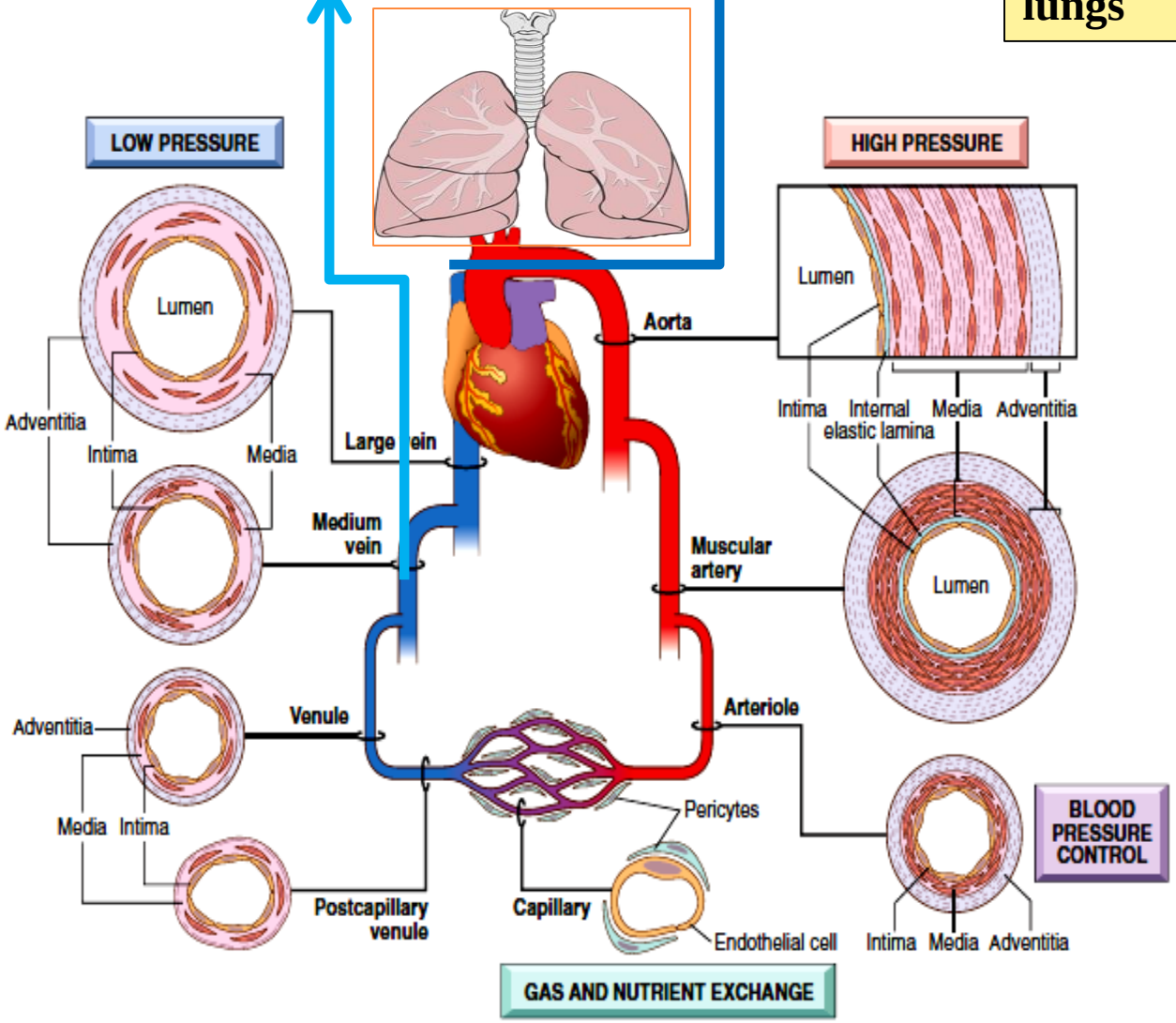


Figure 9–1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.

Origin of most arterial thrombi = heart chambers

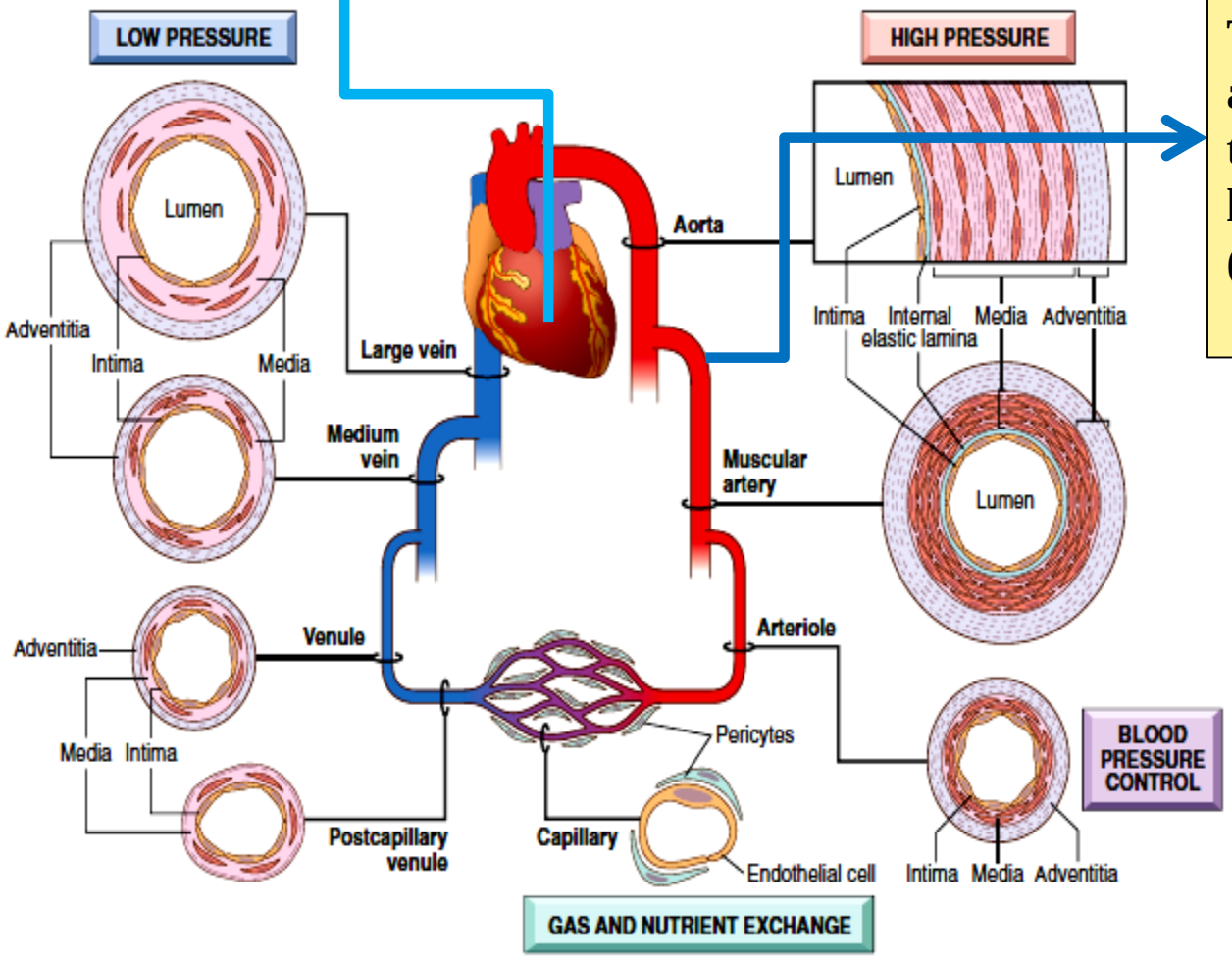


Figure 9–1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.

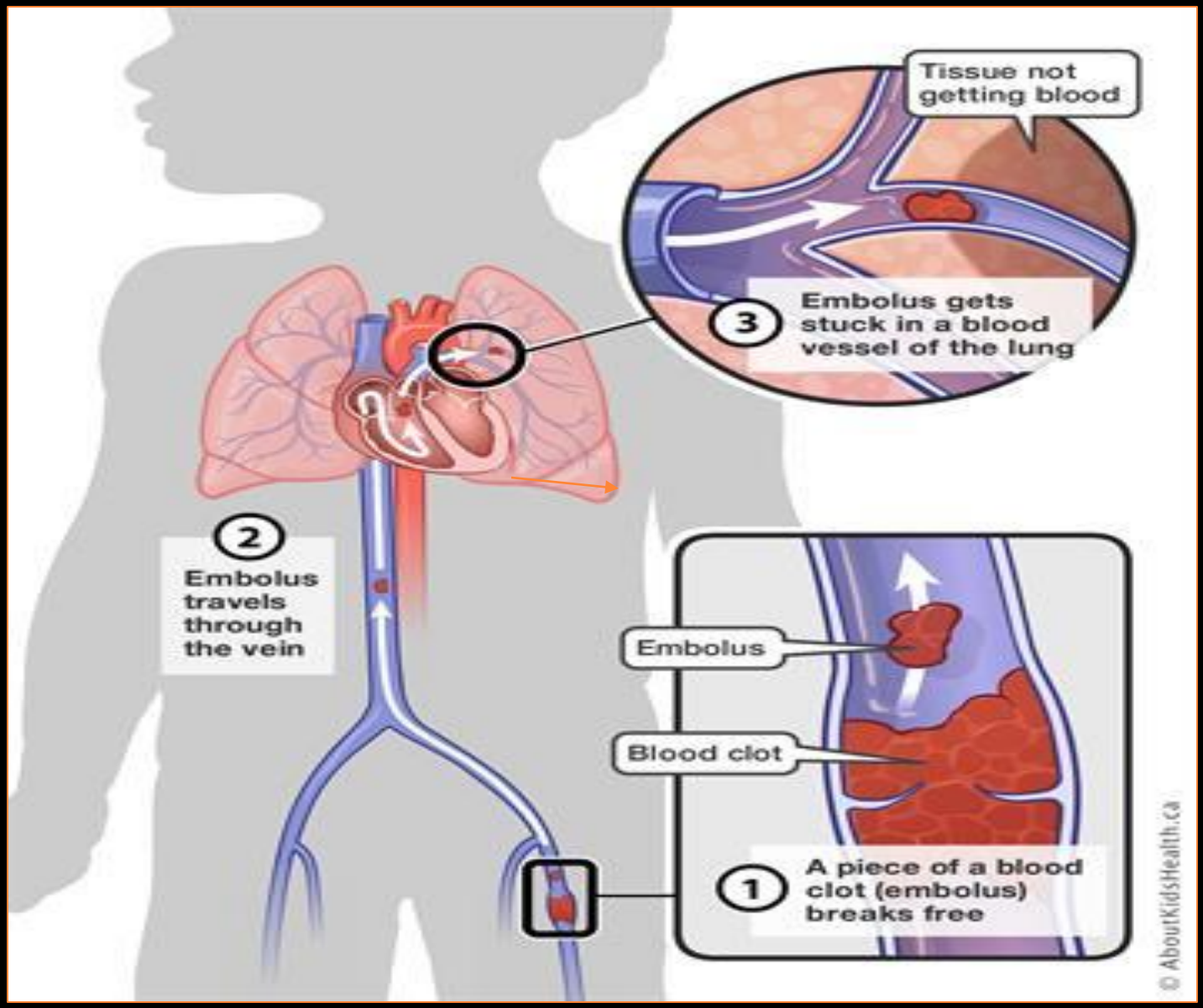
Target of most arterial thrombi = lower limbs (75%)

- Types according to site of origin: **venous** and **arterial** (systemic) emboli

- Emboli result in partial or complete vascular occlusion.

- The consequences of thromboembolism: ischemic necrosis (***infarction***) of downstream tissue

VENOUS THROMBI



PULMONARY THROMBOEMBOLISM

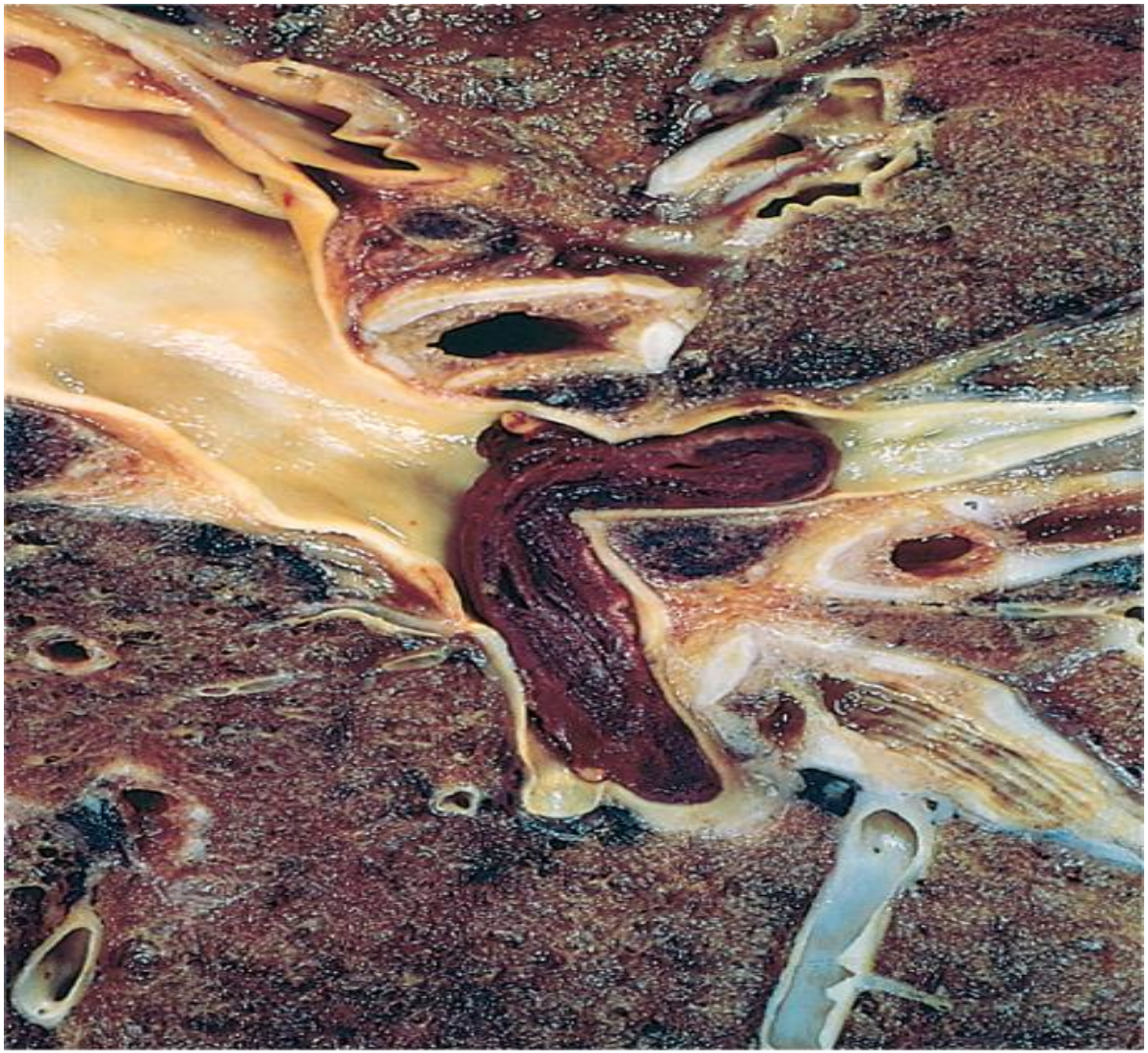
- *95% originate from deep veins of Lower Limbs (DVT)*

- ***Special terms:***

*1- **Saddle embolus**: large embolus occluding the bifurcation of pulmonary artery trunk (fatal)*

*2- **Paradoxical embolus**: Passage of embolus from venous to systemic circulation through ASD or VSD*

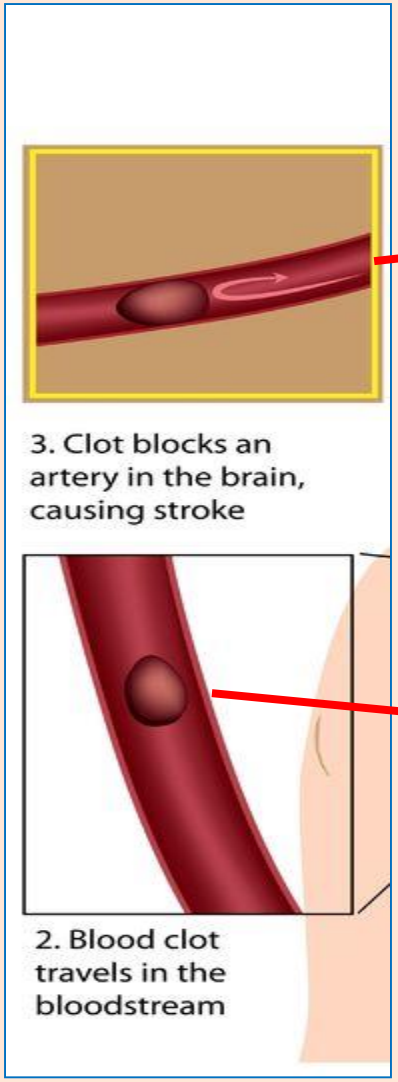
Embolus derived from a lower extremity deep venous thrombosis and now impacted in a pulmonary artery branch

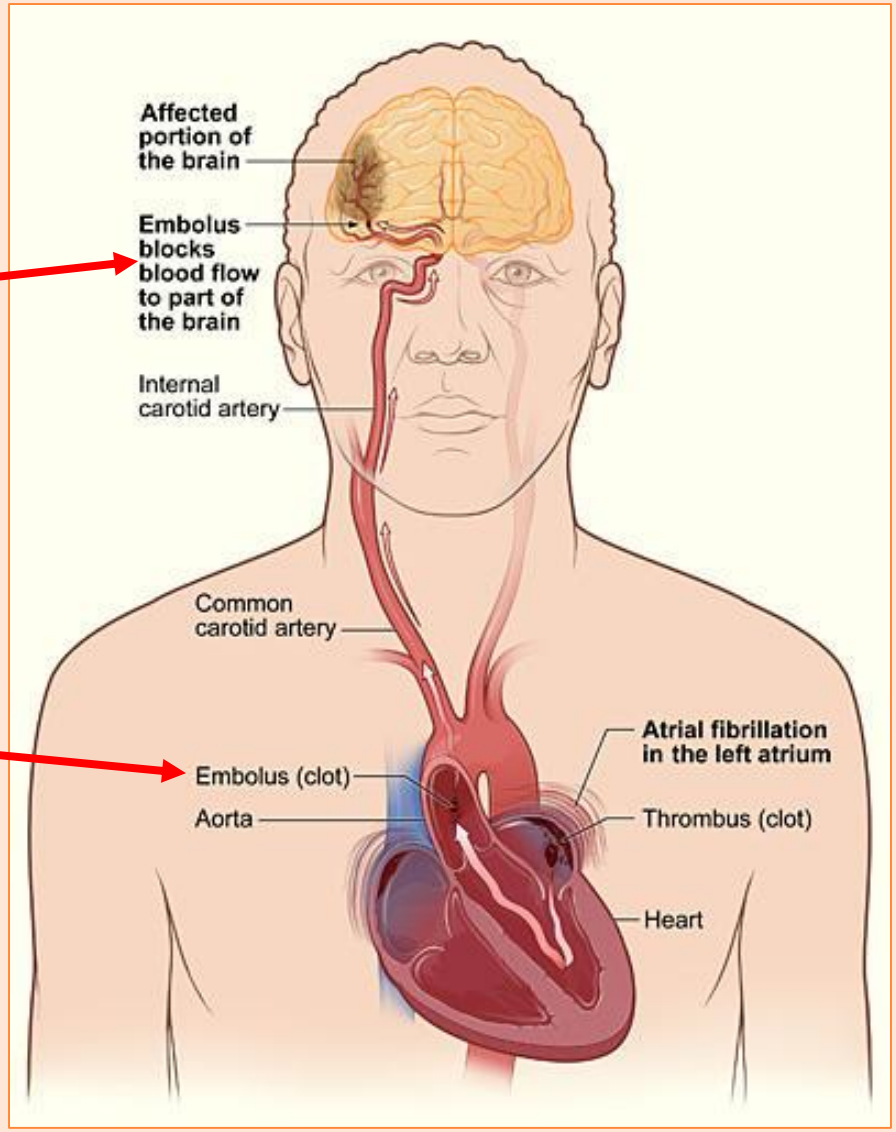


Clinical consequence of Pulmonary thromboembolism :

- 60%- 80% pulmonary emboli are clinically **asymptomatic** (small) → *Organization*
- If large→ *Pulmonary infarction*
- > 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*

Arterial emboli





Systemic (arterial) thromboembolism

- Emboli traveling within the arterial circulation
- 80% due **to intracardiac mural thrombi (origin)**

causes: -2/3 Lt. ventricular failure

- ¼ Lt. atrial dilatation
- Ulcerated atherosclerotic plaque
- Aortic aneurysm
- valve vegetation ….etc

- 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…)*

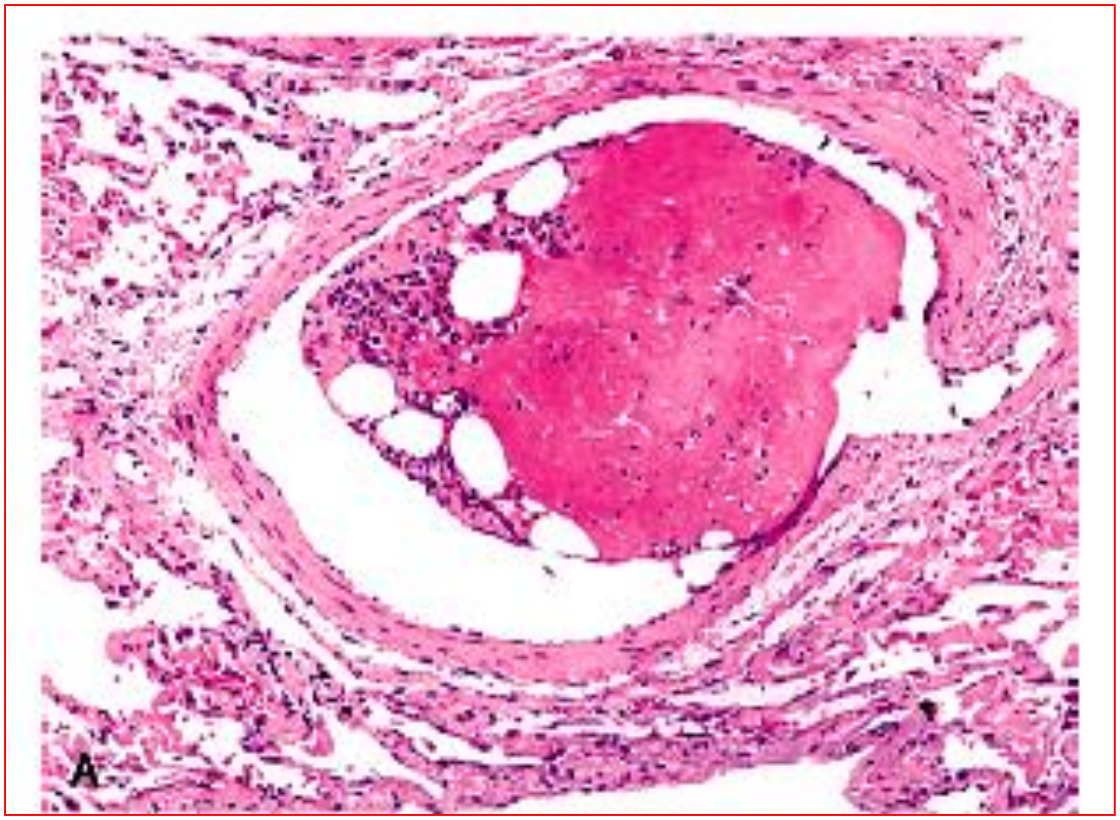
Results:

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 embolus= fat globules + hematopoietic cells



- **Fat embolism 'syndrome'** is 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 the case*

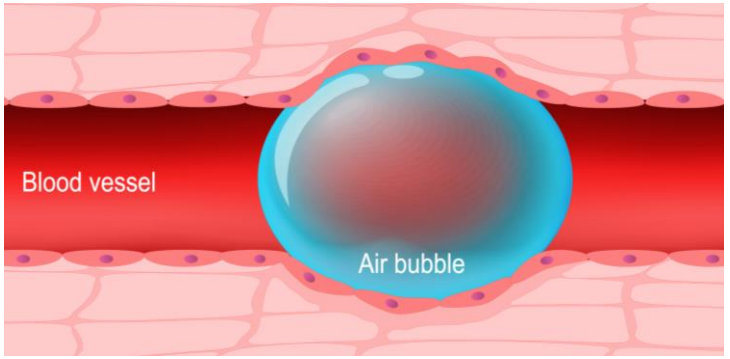
- Symptoms appears 1-3 days after injury

Air Embolism

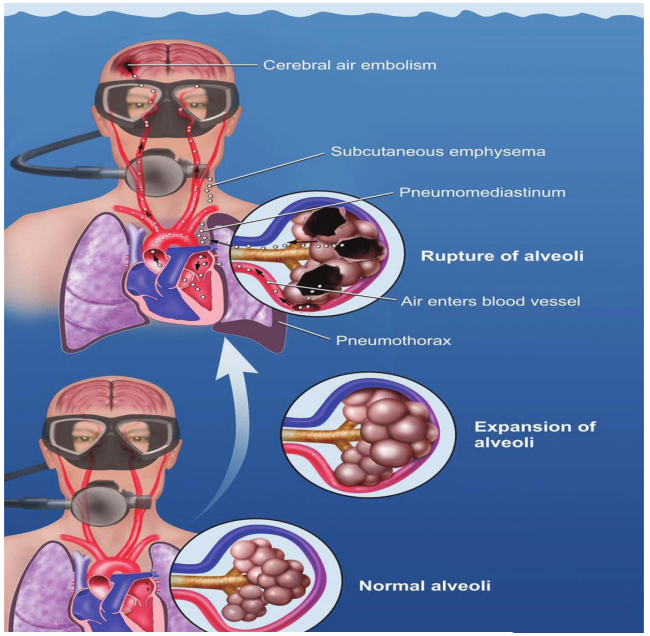
- causes:

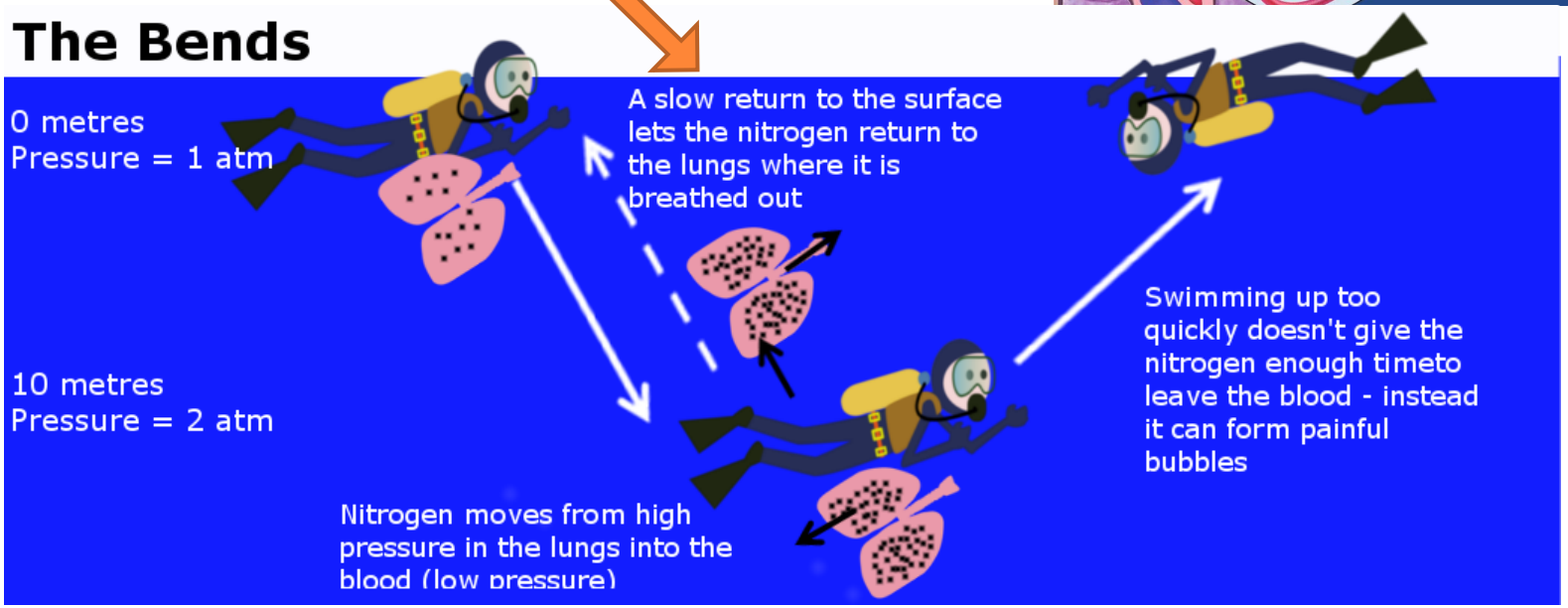
1. *Obstetric procedures*
2. *Chest wall injury*
3. *Decompression sickness: in* Scuba deep-sea divers ((nitrogen))

- *Note: More then 100ml of air is required to produce clinical effect!*



Decompression sickness





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

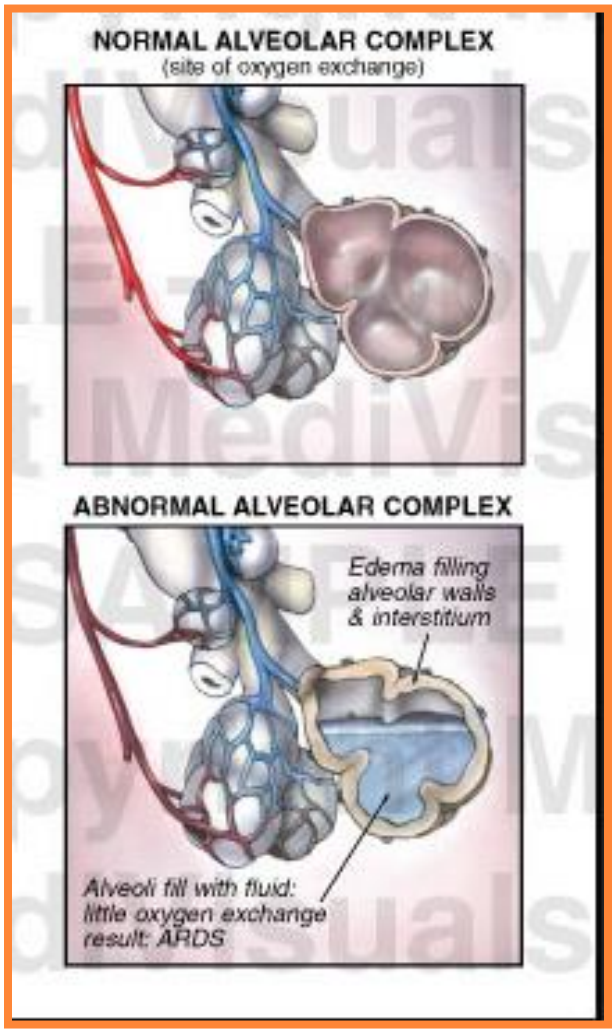
4. **Caisson disease**: in scuba divers; gas emboli in the bones leads to multiple foci of ischemic necrosis, usually the heads of the femurs, tibias, and humeri

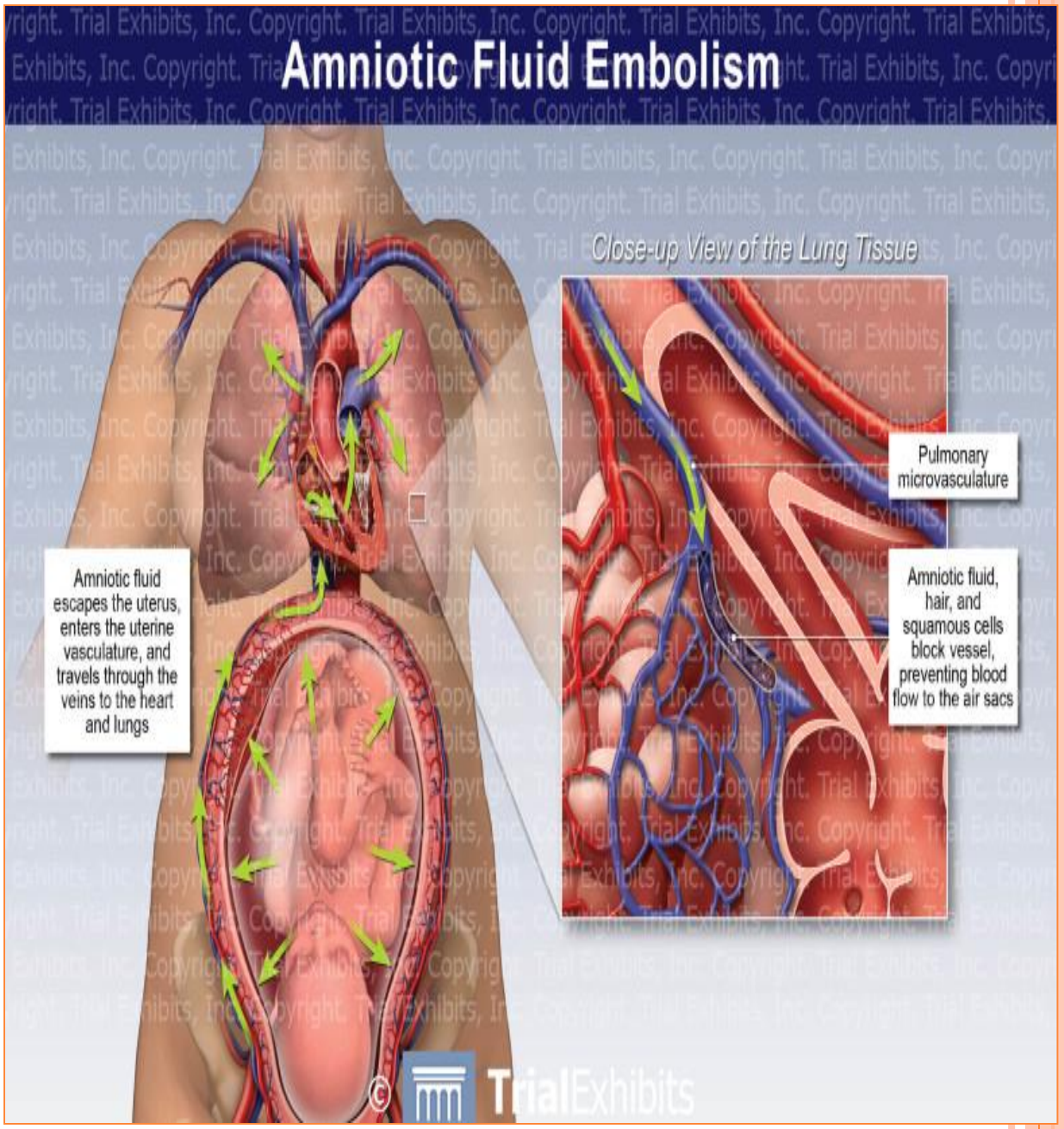
Amniotic fluid embolism

- 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, ARDS, and hypotensive shock, followed by seizures, DIC and coma**

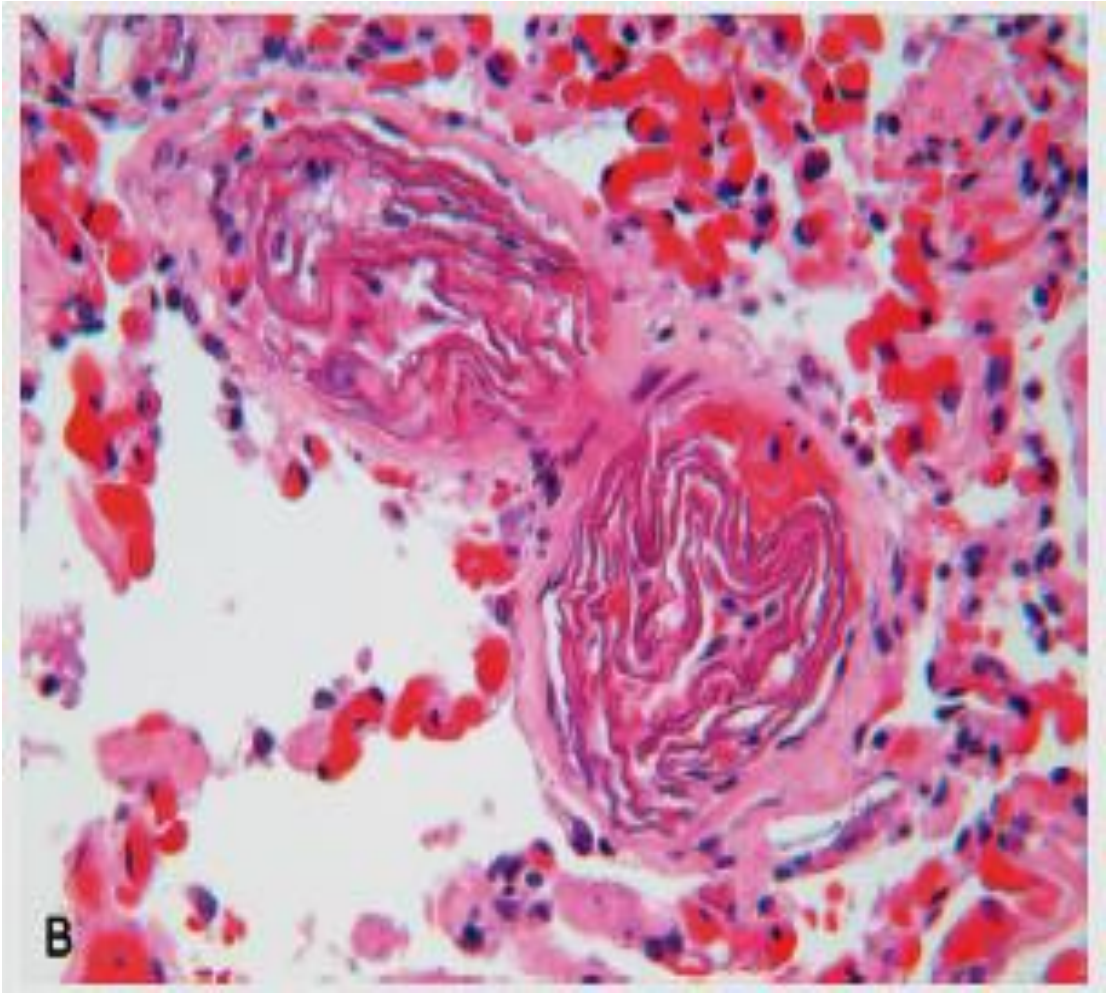
- **Microscopic** Findings upon autopsy:

  fetal squamous cells, languo hair, fat, mucin …..etc within the maternal pulmonary microcirculation


NORMAL ALVEOLAR COMPLEX
(site of oxygen exchange)
ABNORMAL ALVEOLAR COMPLEX
Edema filling alveolar walls & interstitium
Alveoli fill with fluid: little oxygen exchange result: ARDS


Amniotic Fluid Embolism
Close-up View of the Lung Tissue
Amniotic fluid escapes the uterus, enters the uterine vasculature, and travels through the veins to the heart and lungs
Pulmonary microvasculature
Amniotic fluid, hair, and squamous cells block vessel, preventing blood flow to the air sacs
TrialExhibits

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 tissue
- 99% of 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
  - tend to be wedge shaped (occluded vessel at the apex and the periphery of the organ forming the base)
  - margins of infarcts tend to become better defined with time
  - histologic hallmark of infarction is **ischemic coagulative necrosis**
  - note: The brain is an **exception** (liquefactive necrosis)
  - most infarcts are ultimately replaced by scar

RED INFARCTS:

- **occur in any of the following scenarios:**

(1) **venous** occlusions (e.g. ovarian torsion)

(2) **loose** tissues (e.g. lung) that allow blood to collect in the infarcted zone

(3) tissues with **dual** circulations (e.g. 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).

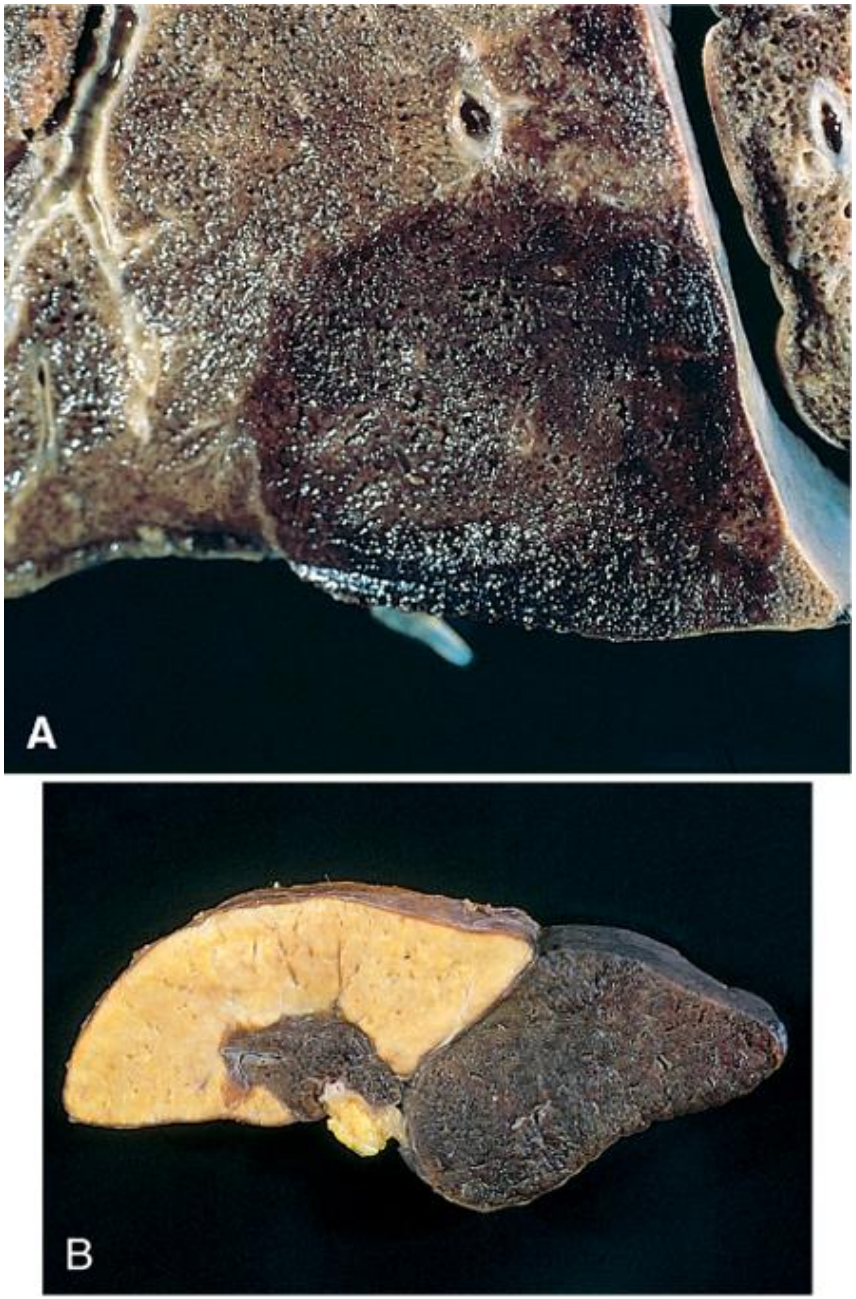
Septic infarctions:

- occur when infarct is superimposed by infection;
- examples:

1- **infected vegetations**

2- **microbes seed an area of necrotic tissue**

- infarct is converted into **abscess** with a greater inflammatory response



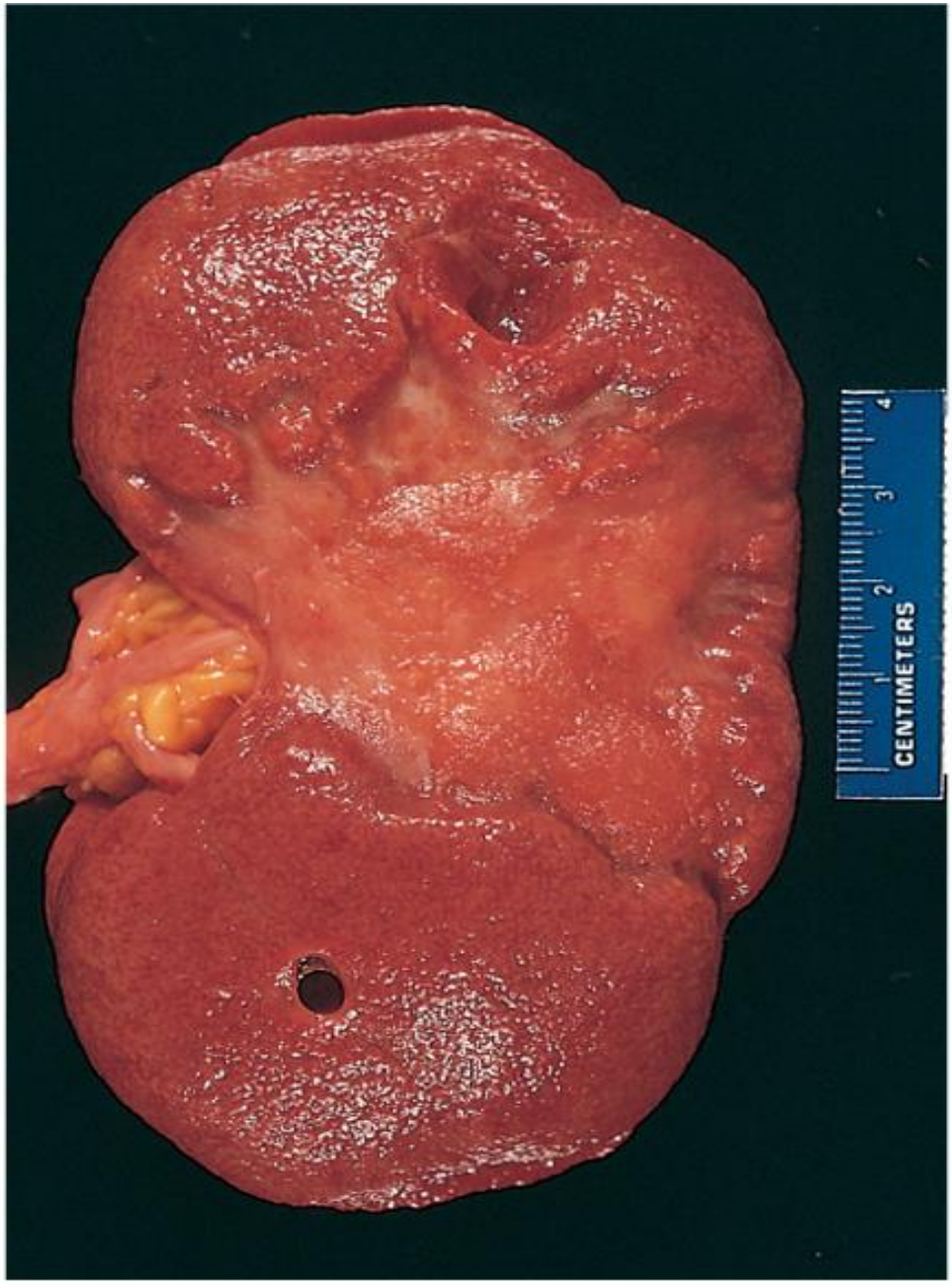
Red and white infarcts.

A→lung

B→spleen

Kidney white infarct

kidney infarct replaced by a large fibrotic scar



Factors That Influence Development of an Infarct

- ***nature of vascular supply***
- ***rate of occlusion development (***collateral circulation)
- ***tissue vulnerability to hypoxia***

- Neurons undergo irreversible damage → 3 to 4 minutes of ischemia.
- Myocardial cells die after only 20 to 30 minutes of ischemia

- ***oxygen content of blood***