EMBOLISM AND INFARCTION

Dr. Nisreen Abu Shahin

Professor of Pathology

Pathology Department

University of Jordan

EMBOLISM:

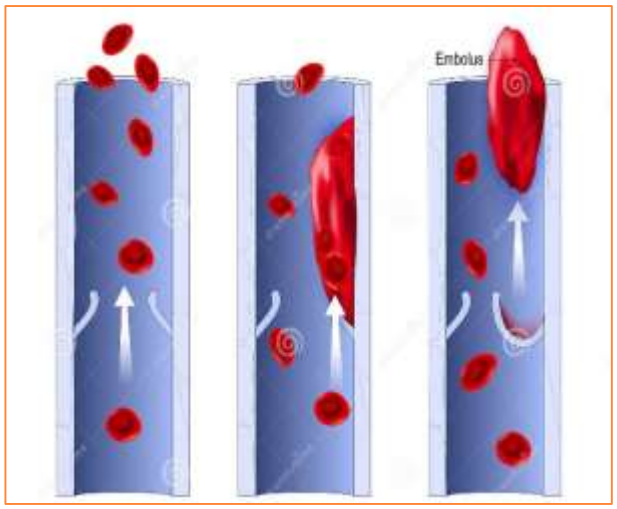
- **An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin**

- ***Types (according to composition of emboli):***

1. ***Thromboembolism: 99% (from dislodged thrombus)***
2. ***Fat embolism***
3. ***Air /Nitrogen embolism***
4. ***Amniotic fluid embolism***

} **1%**

Emboli Types (according to composition)



99%

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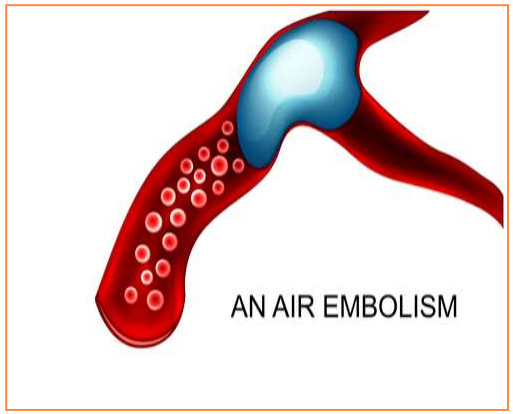
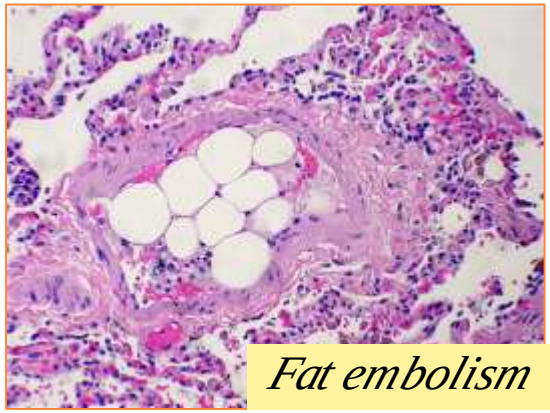
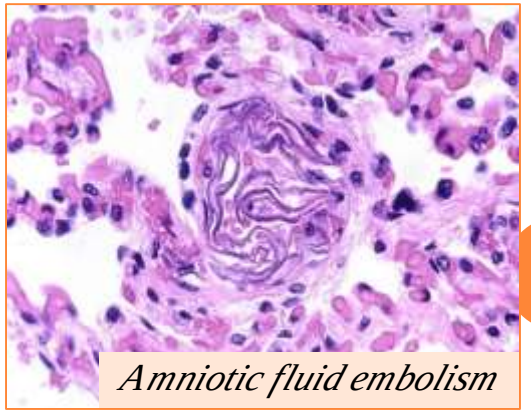
1%





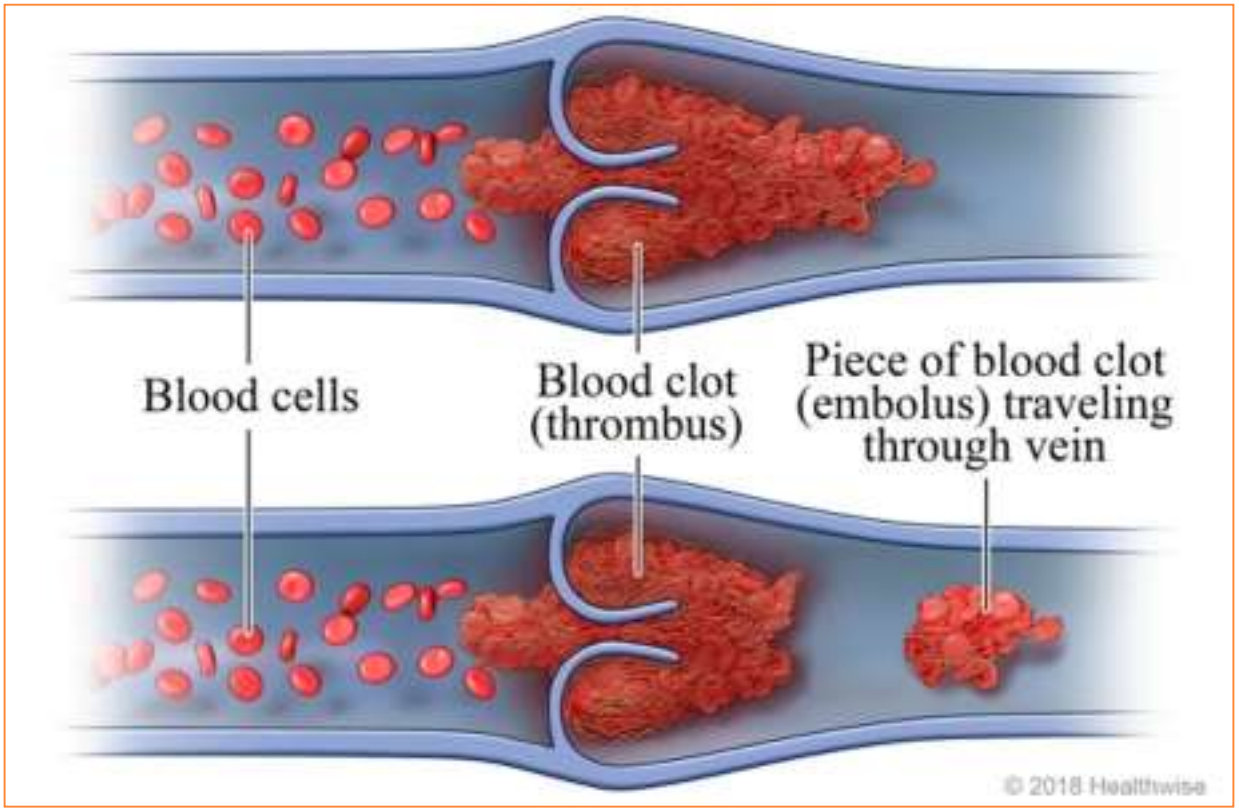
Fat embolism

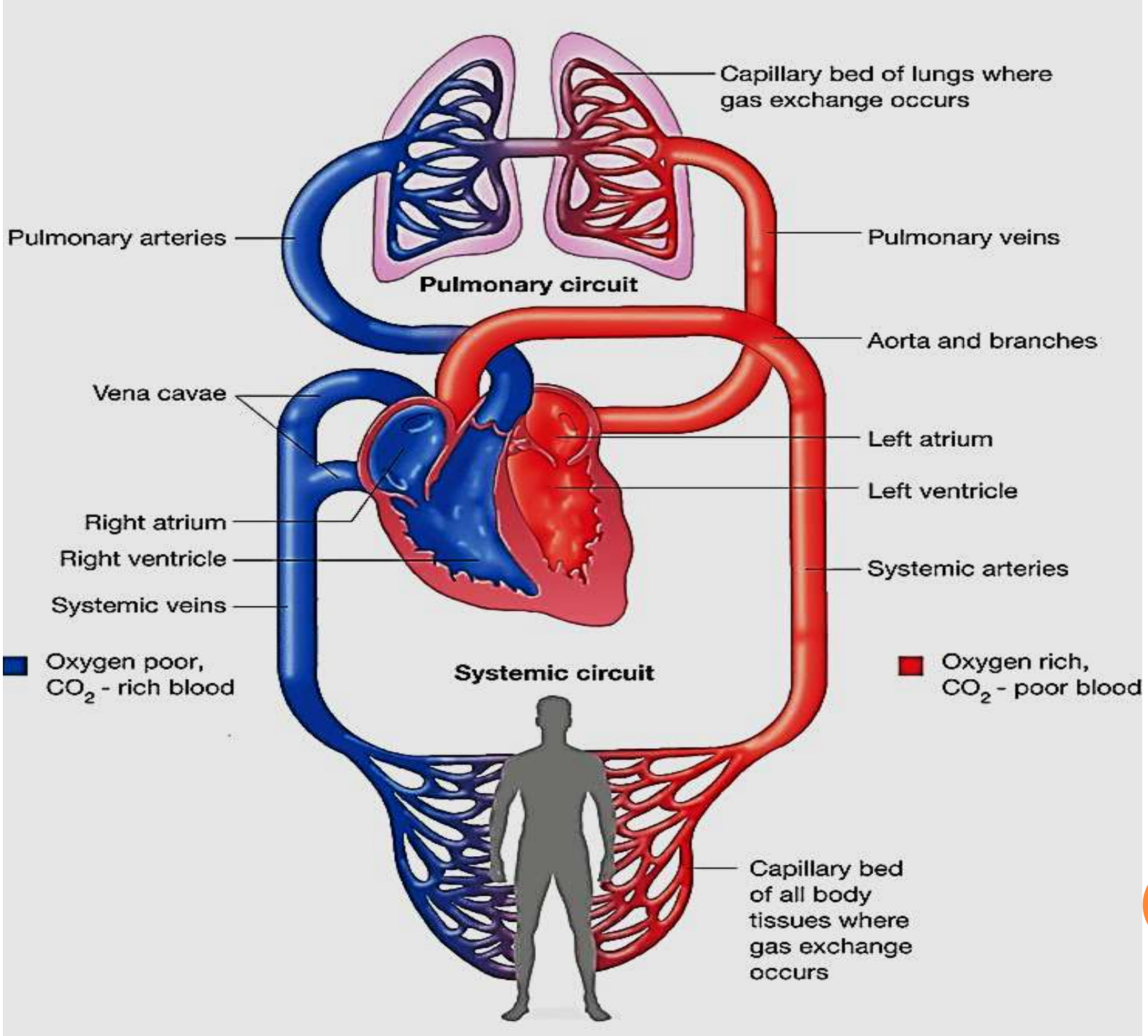


Amniotic fluid embolism

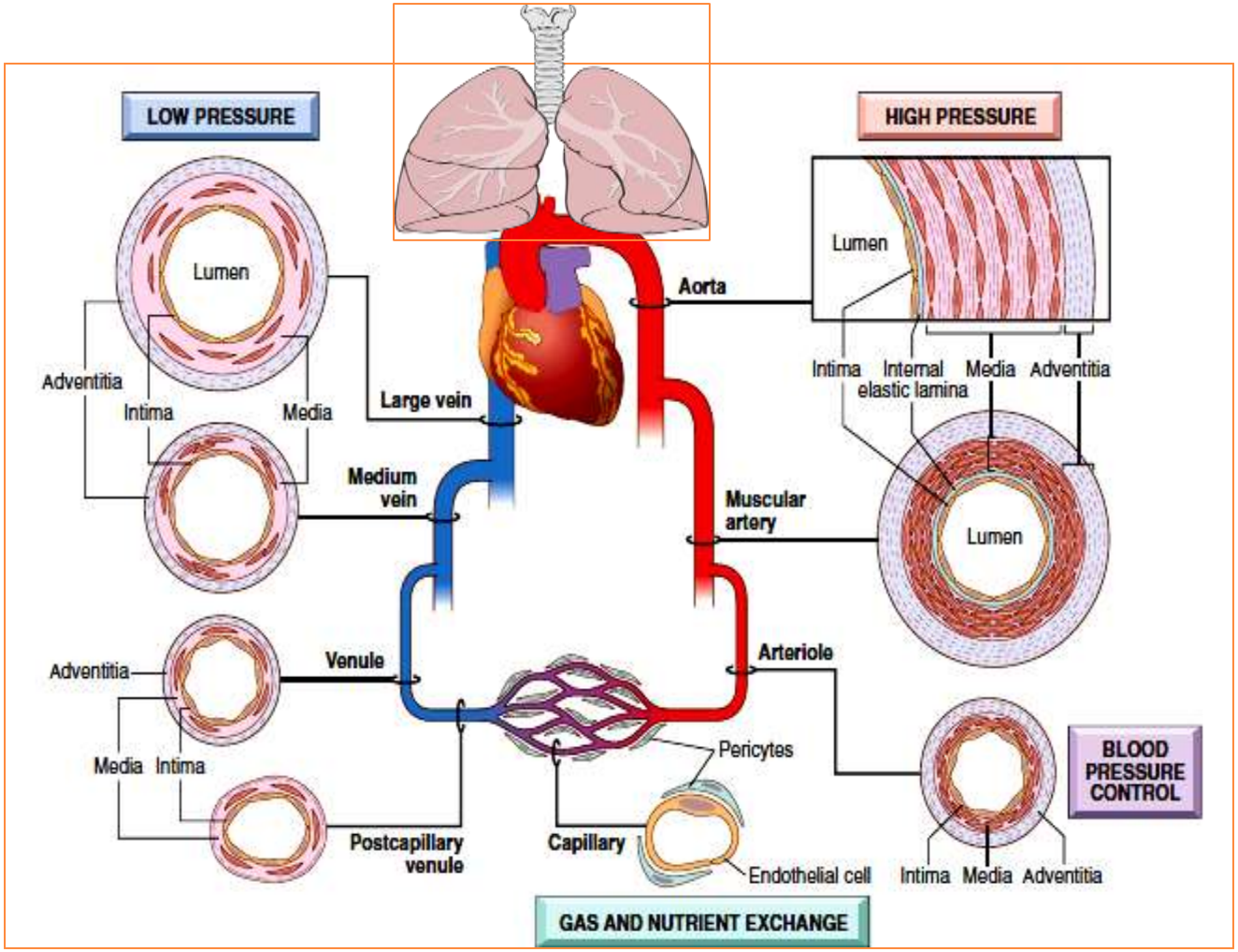
THROMBUS VS EMBOLUS ….?

THROMBOEMBOLISM




Capillary bed of lungs where gas exchange occurs
Pulmonary arteries
Pulmonary veins
Pulmonary circuit
Aorta and branches
Vena cavae
Left atrium
Left ventricle
Right atrium
Right ventricle
Systemic arteries
Systemic veins
Oxygen poor, CO_2 - rich blood
Systemic circuit
Oxygen rich, CO_2 - poor blood
Capillary bed of all body tissues where gas exchange occurs

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



Emboli Types (according to site of origin):

1- venous

2- **arterial** *(systemic) emboli*

Origin of most venous emboli = lower limbs

Target of most venous emboli = lungs

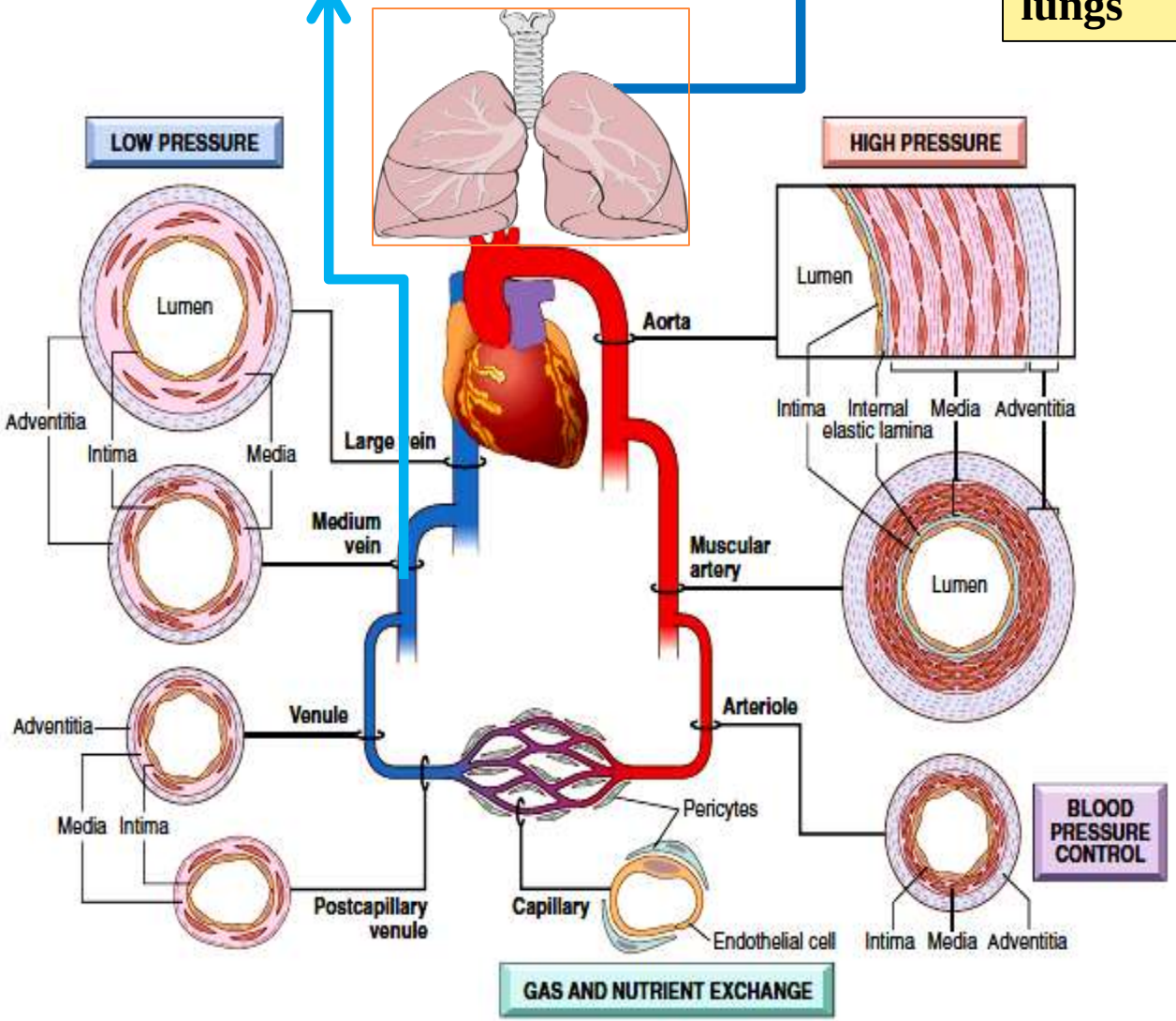
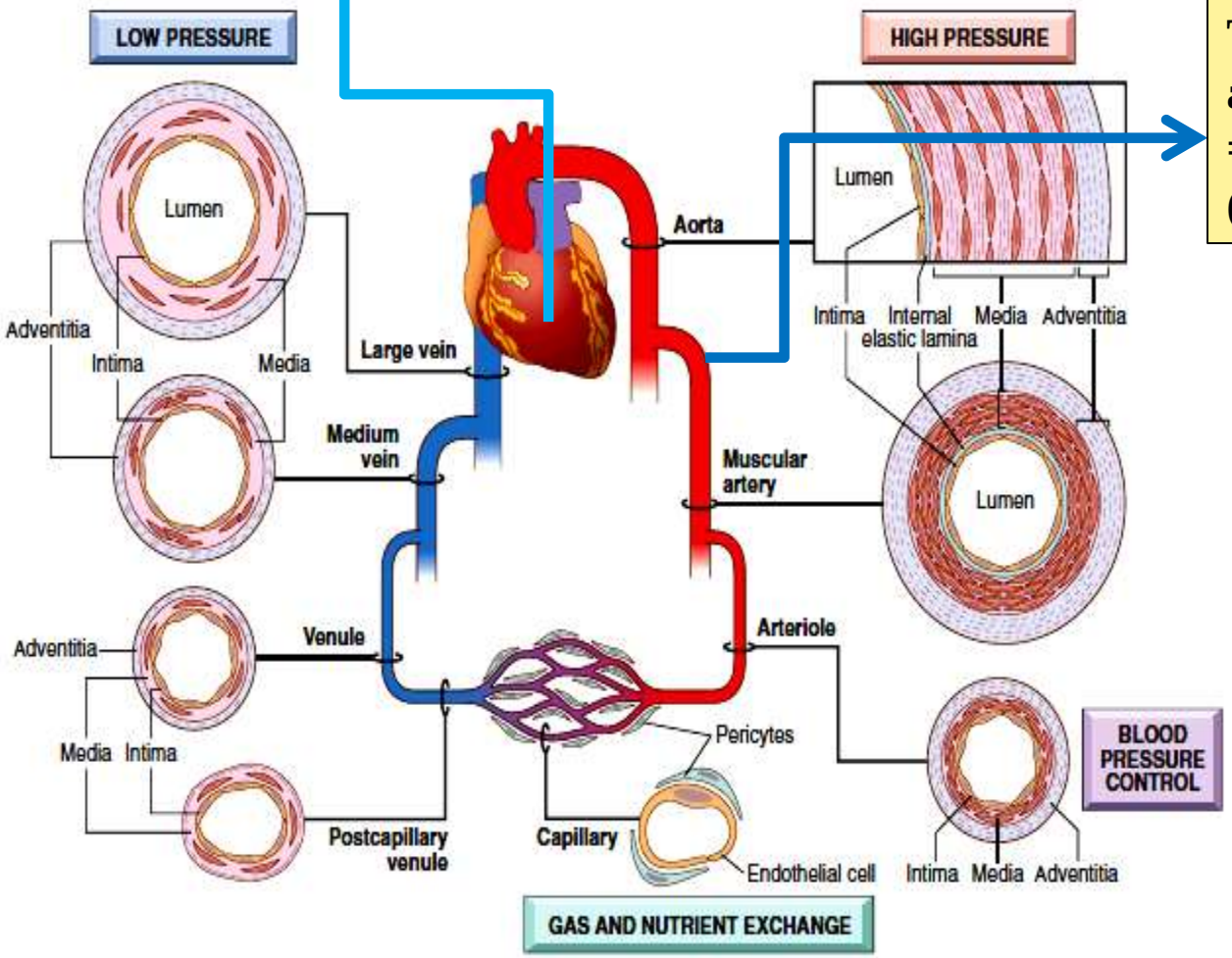


Figure 9–1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.

Origin of most arterial emboli = heart chambers



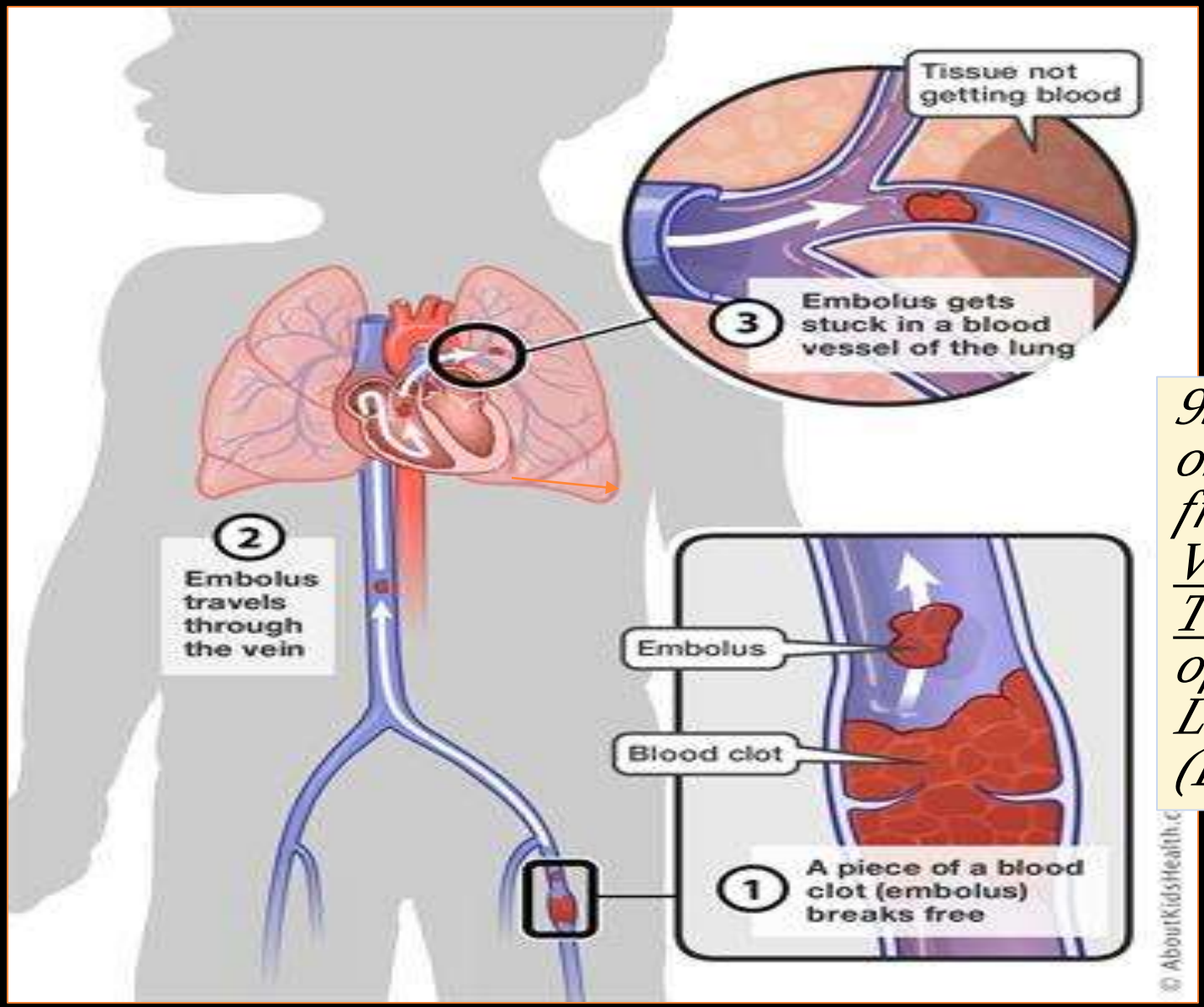
Target of most arterial emboli = lower limbs (75%)

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.

- *Emboli result in partial or complete vascular occlusion.*

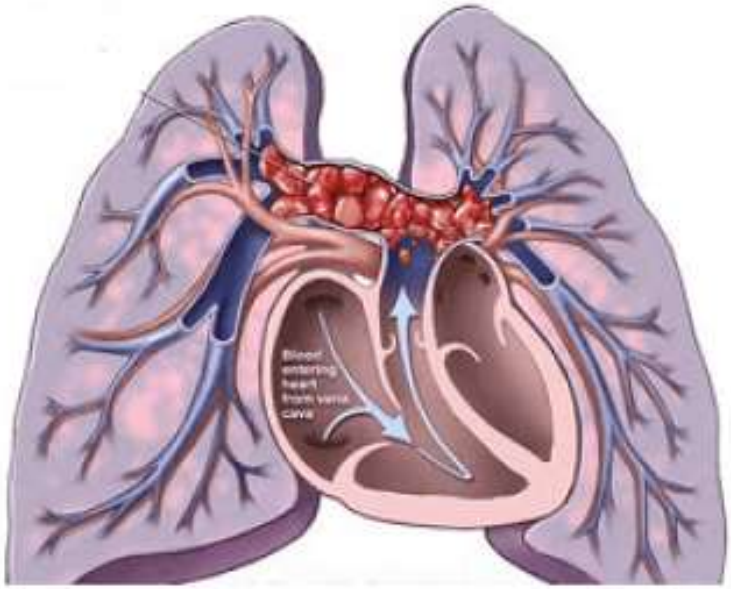
- *consequences of embolism: ischemic necrosis (**infarction**) of downstream tissue*

VENOUS THROMBI



95% originate from <u>DEEP VEINS THROMBI</u> of Lower Limbs (DVT)

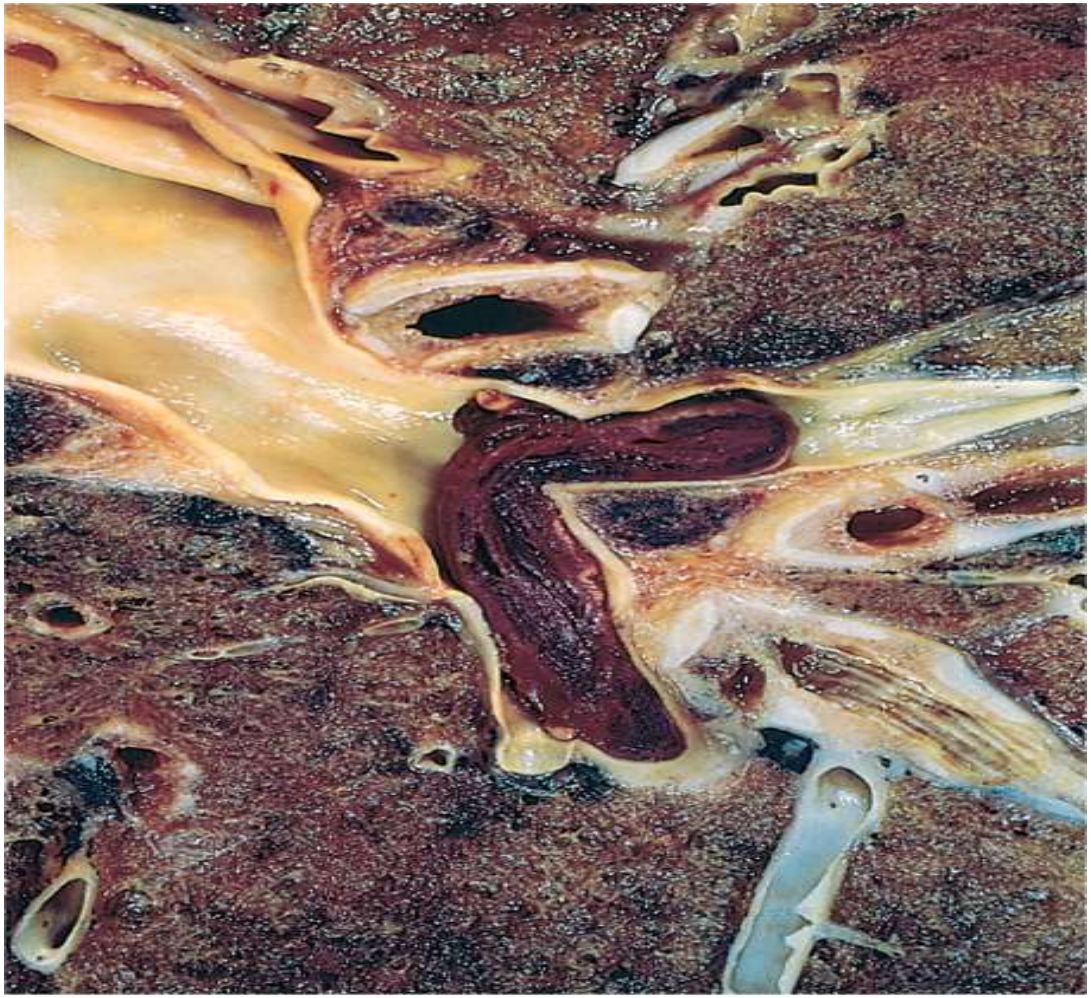
Pulmonary Thromboembolism- *Special terms*



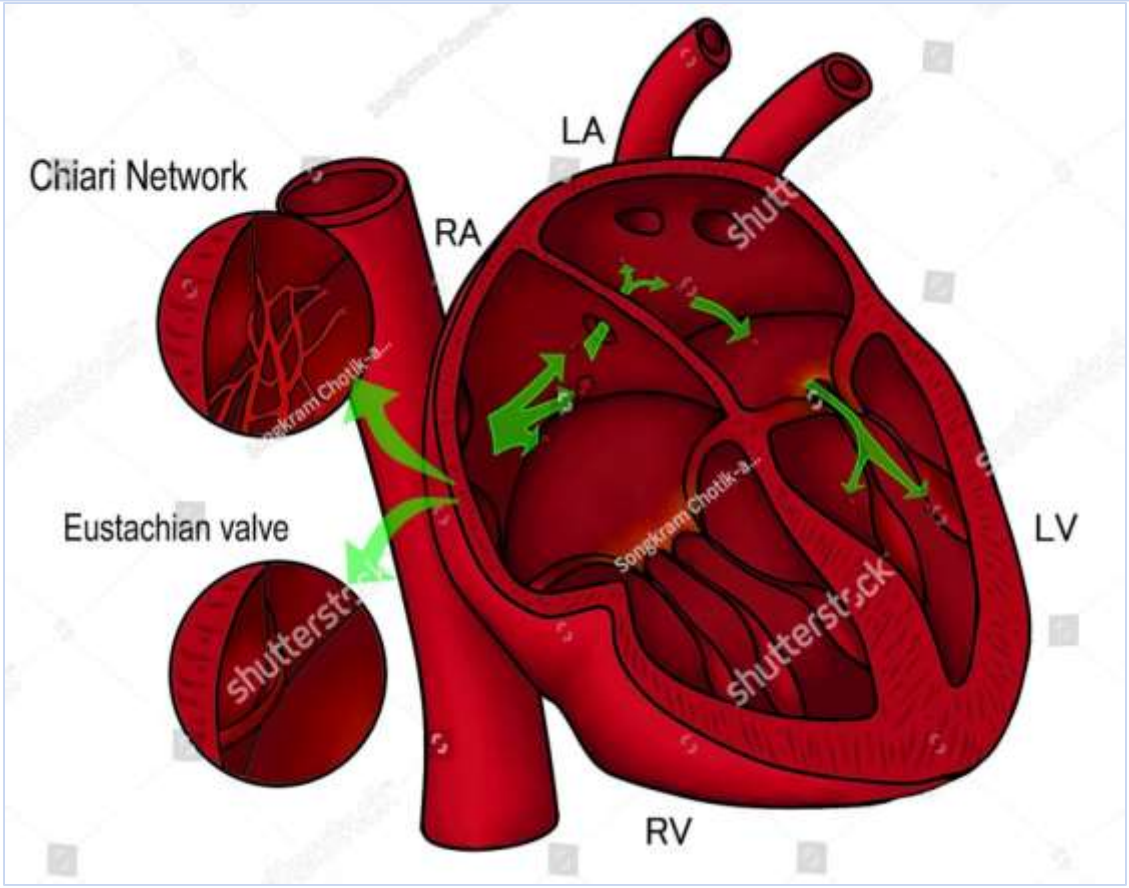
SADDLE EMBOLUS

LARGE EMBOLUS OCCLUDING THE BIFURCATION OF PULMONARY ARTERY TRUNK (FATAL)

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

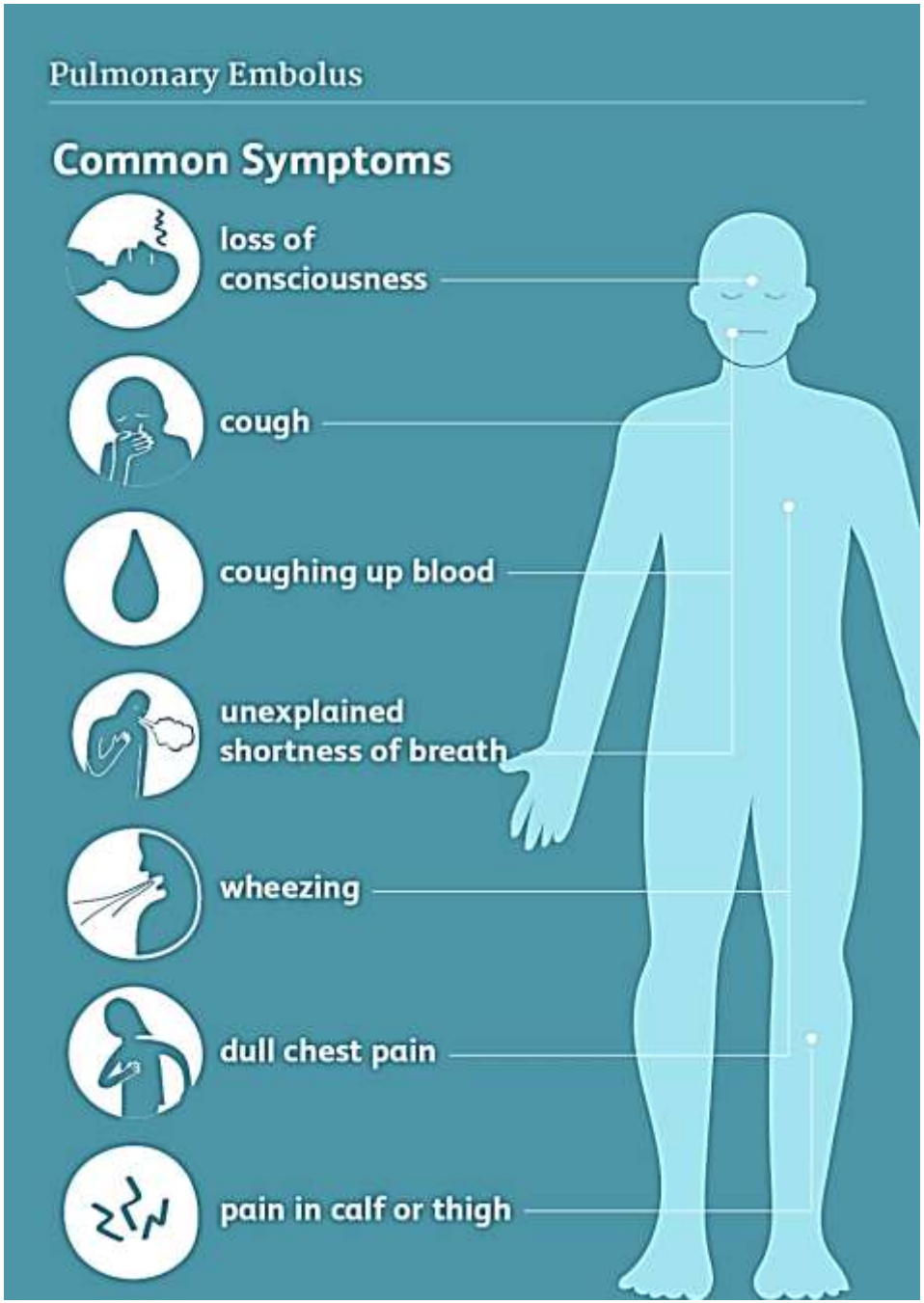


***Paradoxical embolus**: Passage of embolus from venous to systemic circulation through PFO, ASD or VSD*

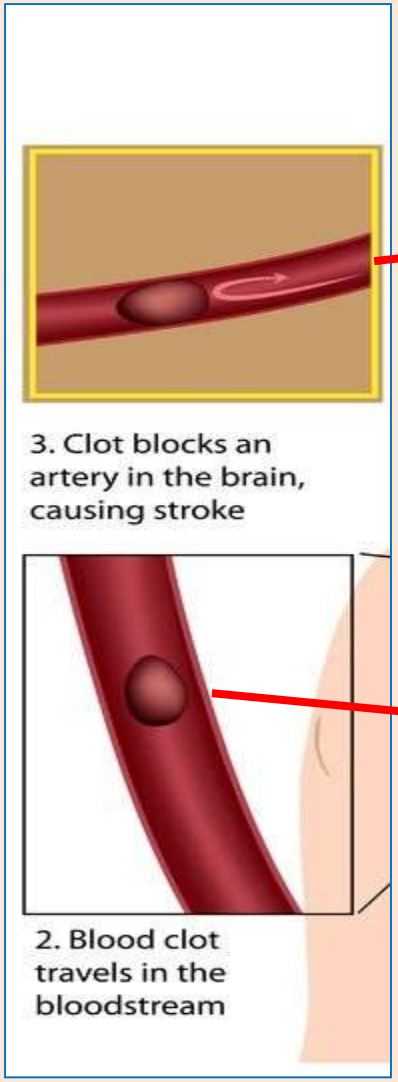


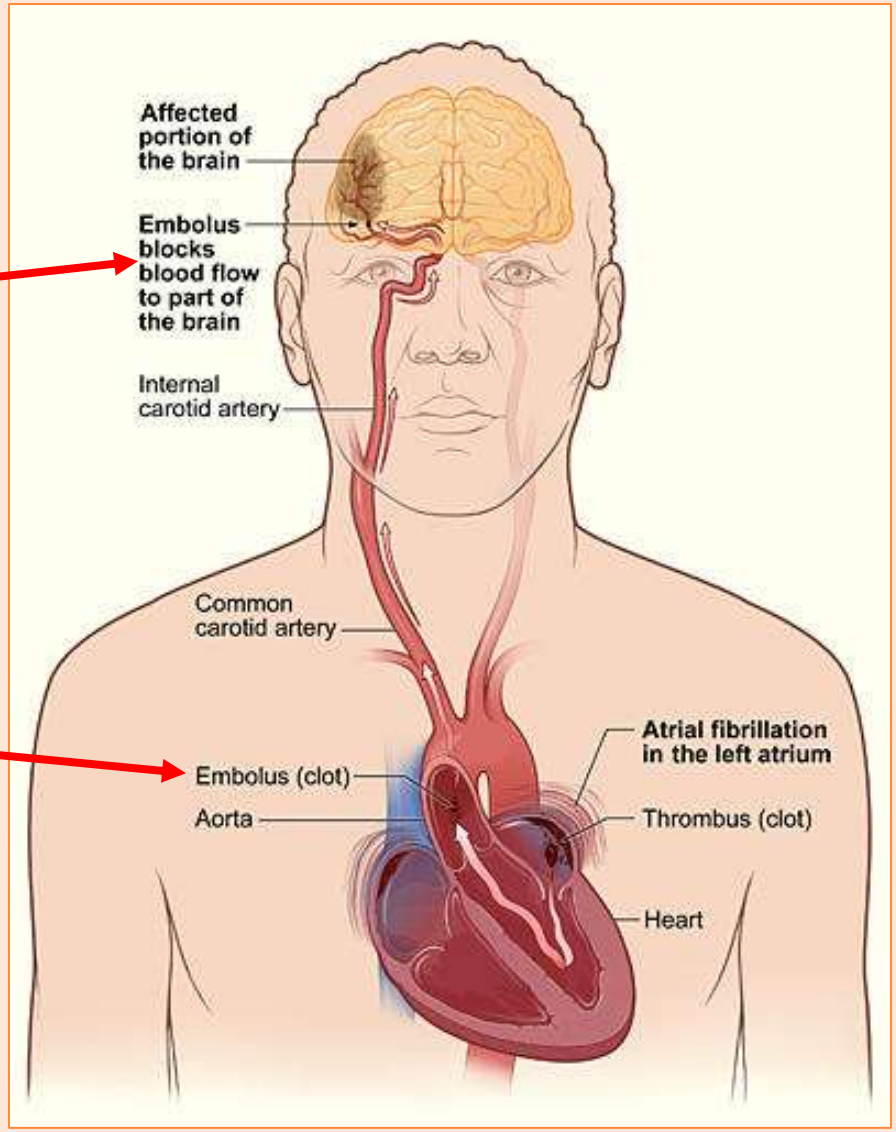
Clinical consequence of Pulmonary thromboembolism :

- **Asymptomatic** *(60%- 80%; small)*
- *Pulmonary infarction (large)*
- *Pulmonary hemorrhage*
- *Pulmonary Hypertension* and *right ventricular failure: (showers of emboli over a long time)*
- *Sudden death (RVF, CV collapse): > 60 % of pulmonary vessels are obstructed*


Pulmonary Embolus
Common Symptoms
loss of consciousness
cough
coughing up blood
unexplained shortness of breath
wheezing
dull chest pain
pain in calf or thigh

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 ; Brain ; Intestine; Kidneys; Spleen; etc…
(any organ that has arterial supply!)

Fat embolism

- *Causes:*

1. *Skeletal injury: (long bones fractures)*
2. *Adipose tissue Injury :(e.g. fat necrosis in acute pancreatitis)*

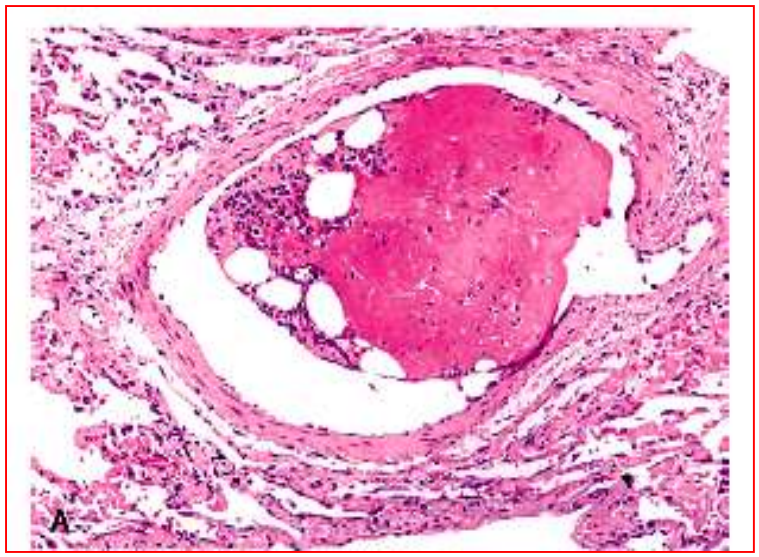
- *Results:*

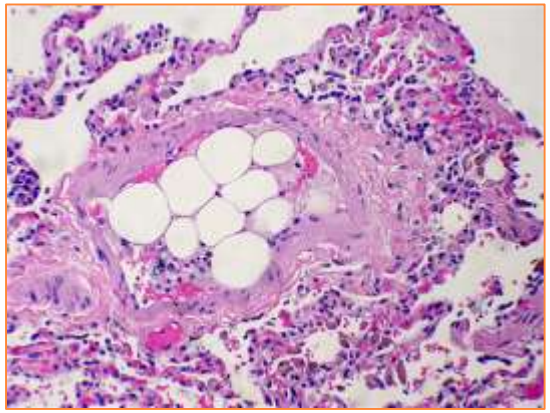
1- Mechanical obstruction of vessels

2- Free fatty acid release: toxic injury to endothelium + systemic immune response

- *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 cases*

Note: Symptoms appear 1-3 days after injury

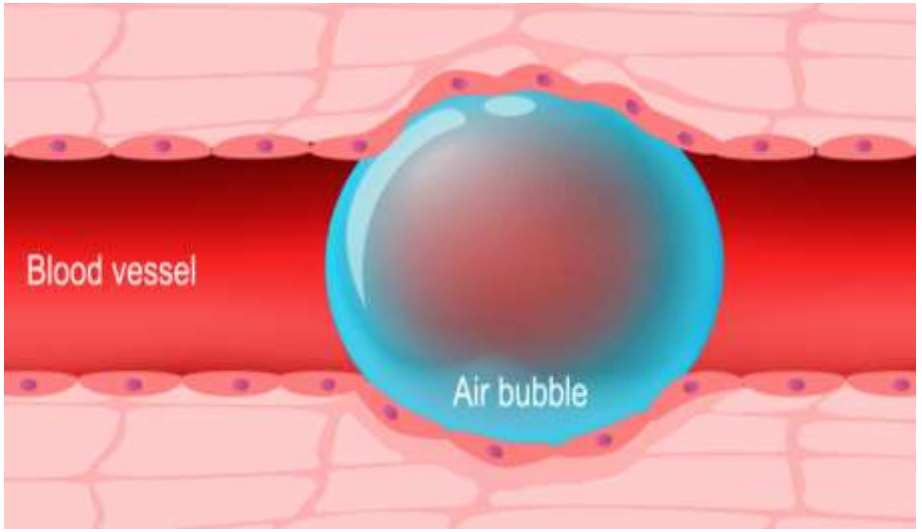
THERAPY FOR FAT EMBOLISM SYNDROME

- *no specific treatment*
- *prevention, early diagnosis, and adequate symptomatic treatment are of paramount importance.*
- **Supportive care** *is the mainstay of therapy*
- *Includes: maintenance of adequate oxygenation and ventilation, stable haemodynamics, blood products as clinically indicated, hydration, prophylaxis of deep venous thrombosis and stress-related gastrointestinal bleeding, and nutrition.*

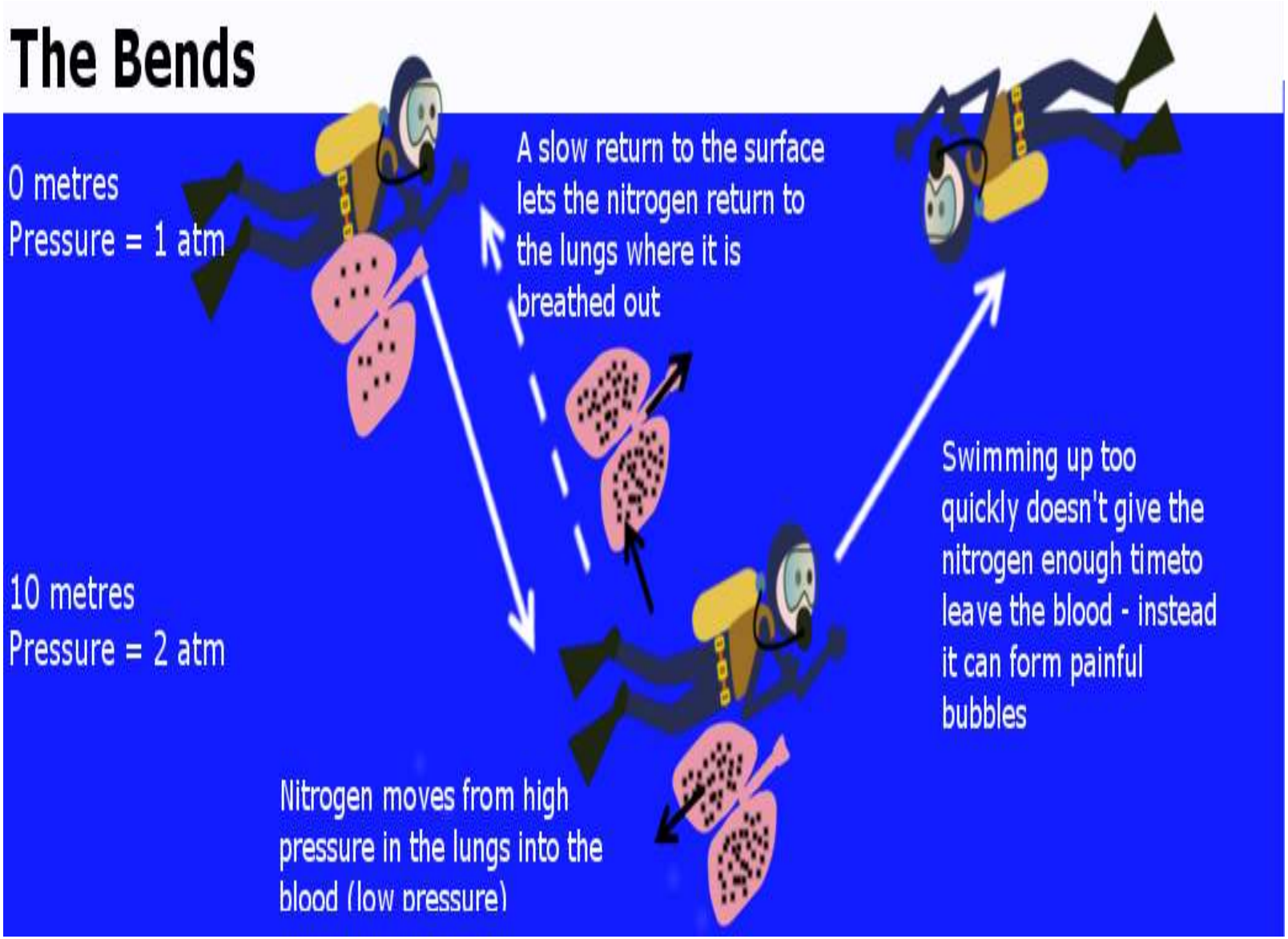
Air Embolism

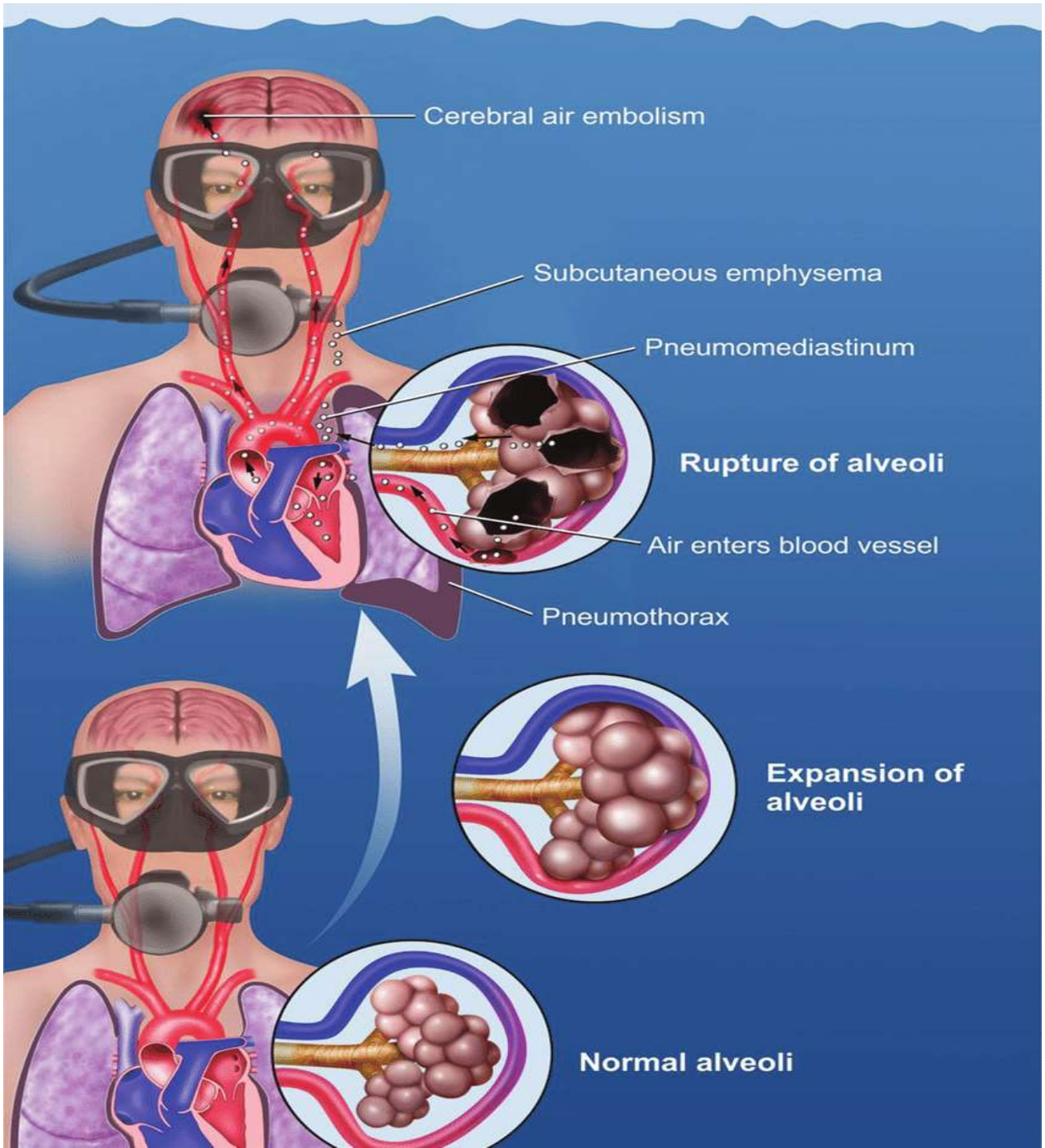
- *Causes:*

1. *Surgical & obstetric procedures*
2. *Traumatic chest wall injury*
3. *Decompression sickness: in Scuba deep-sea divers ((nitrogen))*



Decompression sickness (the bends)




Cerebral air embolism
Subcutaneous emphysema
Pneumomediastinum
Rupture of alveoli
Air enters blood vessel
Pneumothorax
Expansion of alveoli
Normal alveoli

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*

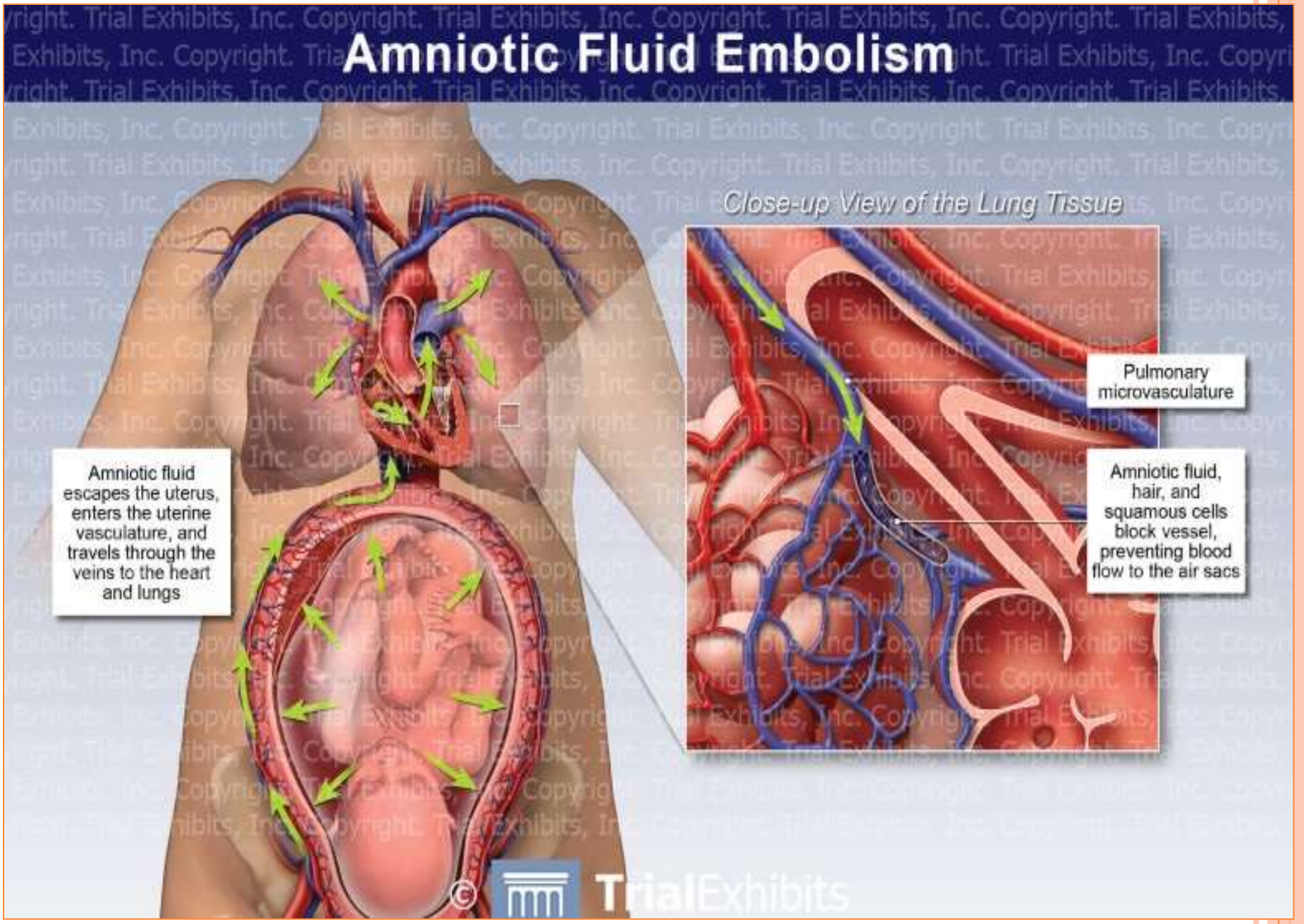
Amniotic fluid embolism

- *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**

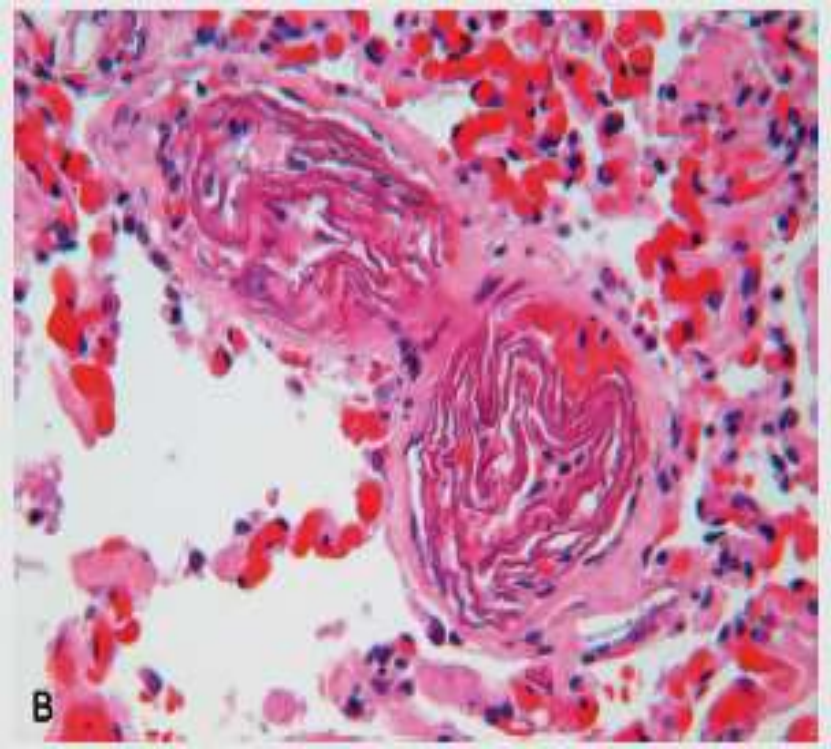
- **Microscopic** *Findings upon autopsy:*

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

Amniotic Fluid Embolism



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*
- *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**

MORPHOLOGY OF INFARCTS

- *infarcts may be either* **red** *(***hemorrhagic***) or* **white** *(***anemic***) and may be either septic or bland*

- *wedge-shaped (occluded vessel at the apex and periphery of organ forming the base)*

- *margins of infarcts become defined with time*

- *histologic hallmark :* **ischemic coagulative necrosis** **(***ultimately replaced by scar)*

[note: The brain is an **exception** *(liquefactive necrosis)].*

RED INFARCTS:

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

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

(2) **loose** *tissues (e.g. lung)*

(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:* **arterial** *occlusions in* **solid** *organs (such as heart, spleen, and kidney).*

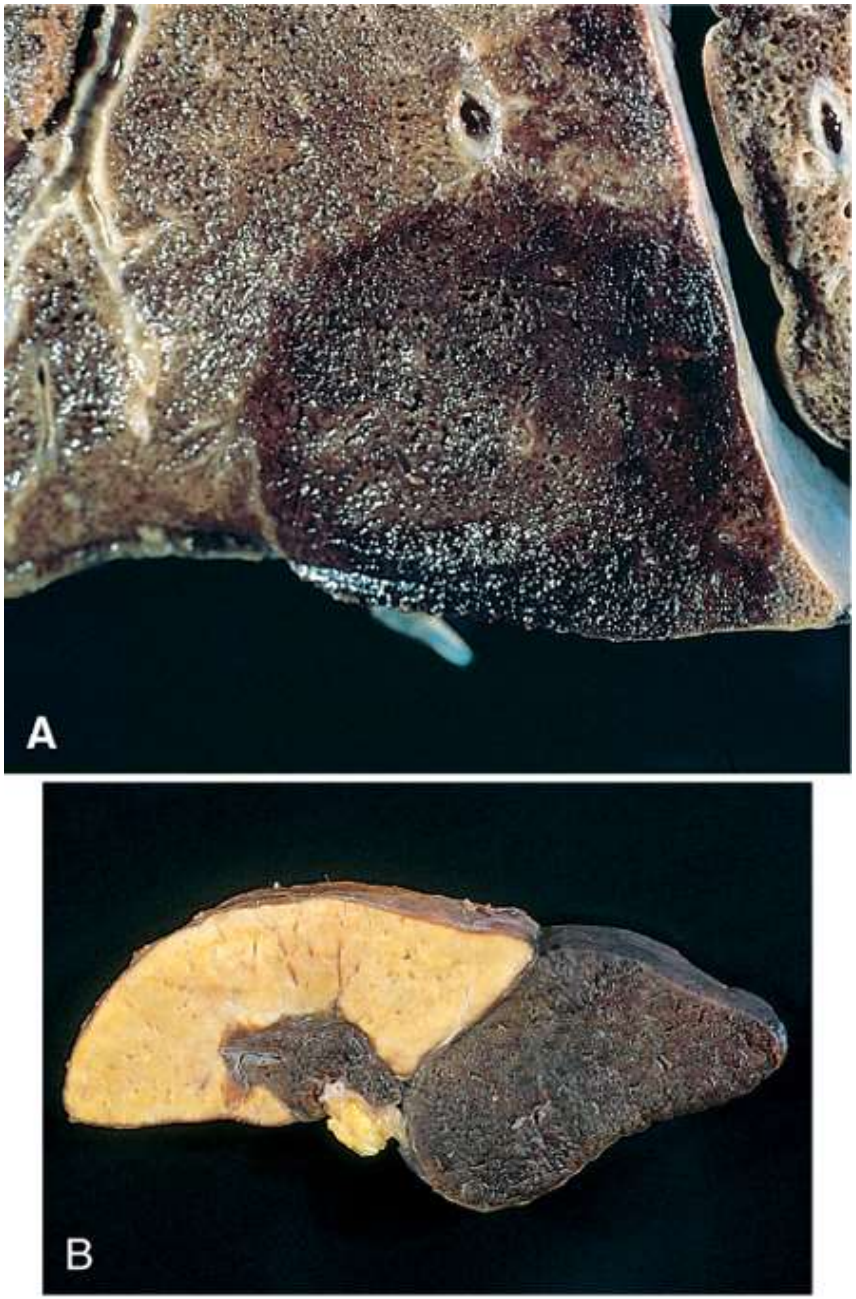
Septic infarctions:

- *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*



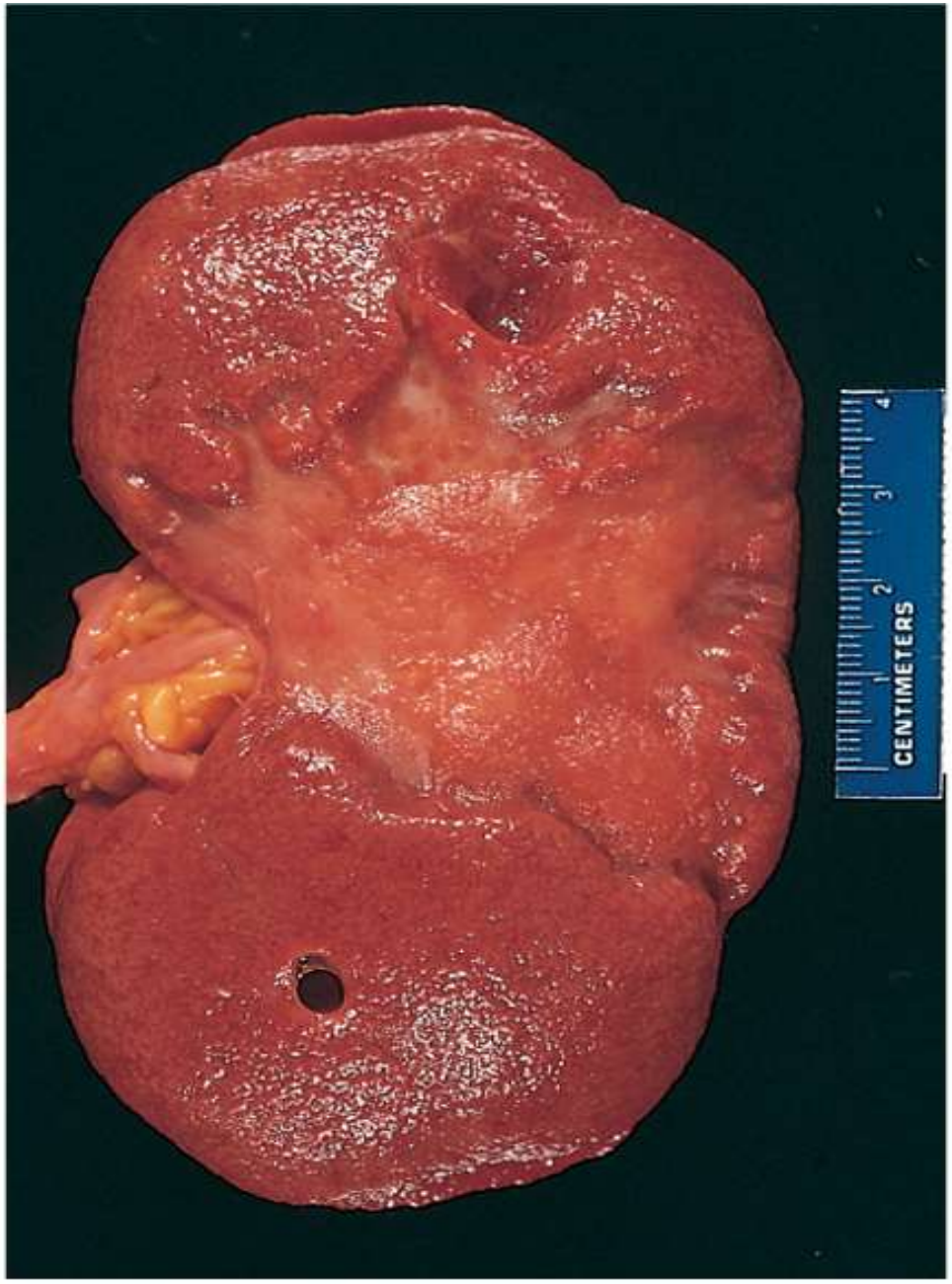
Red and white infarcts.

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 and*** *irreversible damage*
  - *Neurons →only 3 minutes*
  - *Myocardial cells →20 to 30 minutes*
- ***oxygen content of blood***

Q: If we have an embolus in the pulmonary artery will the embolus be considered of venous or arterial origin and will its final target be the lungs ?

A: Embolism in the pulmonary arteries belongs to venous embolism and the main target is the lung itself .
It is a bit confusing but remember that pulmonary artery carries venous blood from the right side of the heart to the lungs, so it's quite the opposite to what do arteries do.

- *Q: Can pulmonary embolism be of an arterial origin?*
- *A: Yes, the lung can be a target of both venous and arterial embolism types. It depends on the side of the circulation it originated from. So if the embolus is coming from venous circulation and is reaching the lung through the right side of the heart it is a venous embolus. On the other hand, if the embolus is coming out of the left side of the heart and reaching the lung through bronchial circulation, then it is an arterial embolus.*