

Doctor.021  
no. 2

# CVS PATHOLOGY



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# **EMBOLISM AND INFARCTION**

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# 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

Circulating fragment of a thrombus in 99% of cases

- Types (according to composition of emboli):

→ Blood clot originated from a thrombus

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

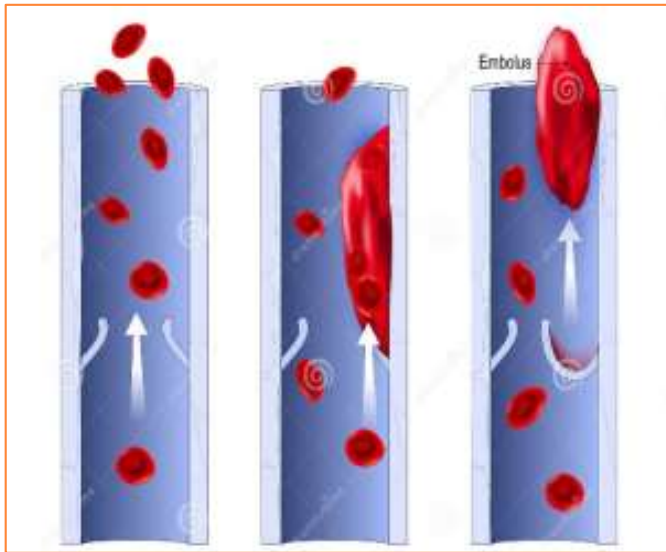
1%

Extremely rare

classify embolism according to their composition



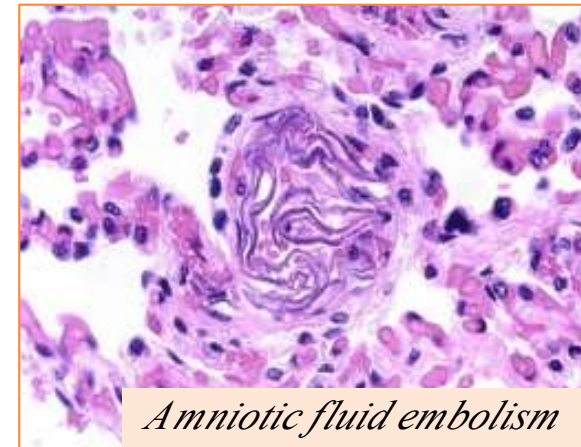
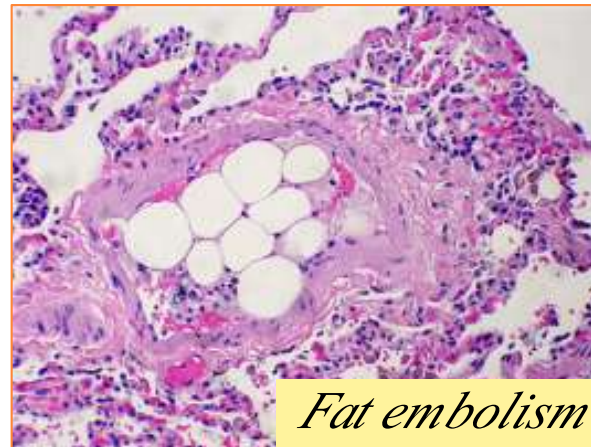
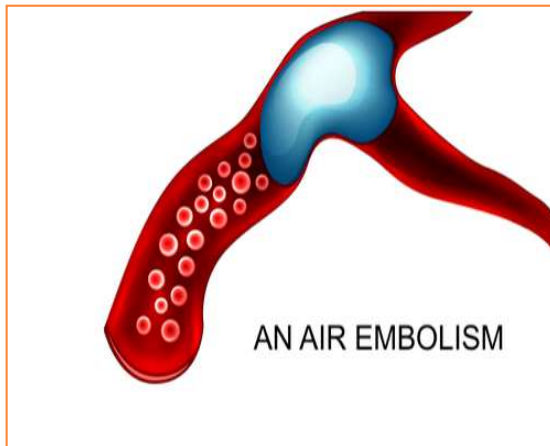
## *Emboli Types (according to composition)*



*99%*

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

**1%**



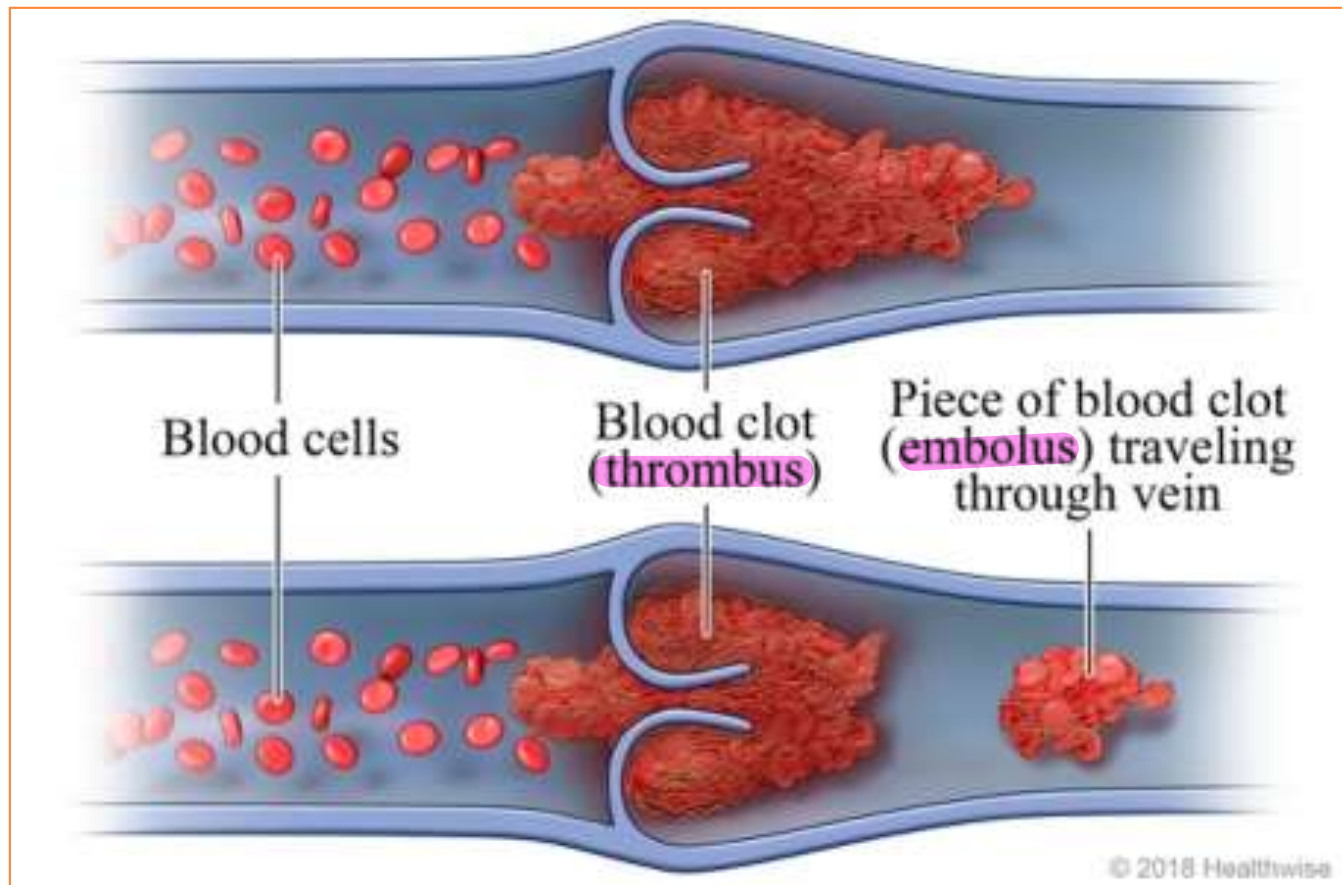
# THROMBUS VS EMBOLUS ....?

## THROMBOEMBOLISM

Thrombus: attached

Embolus: moving

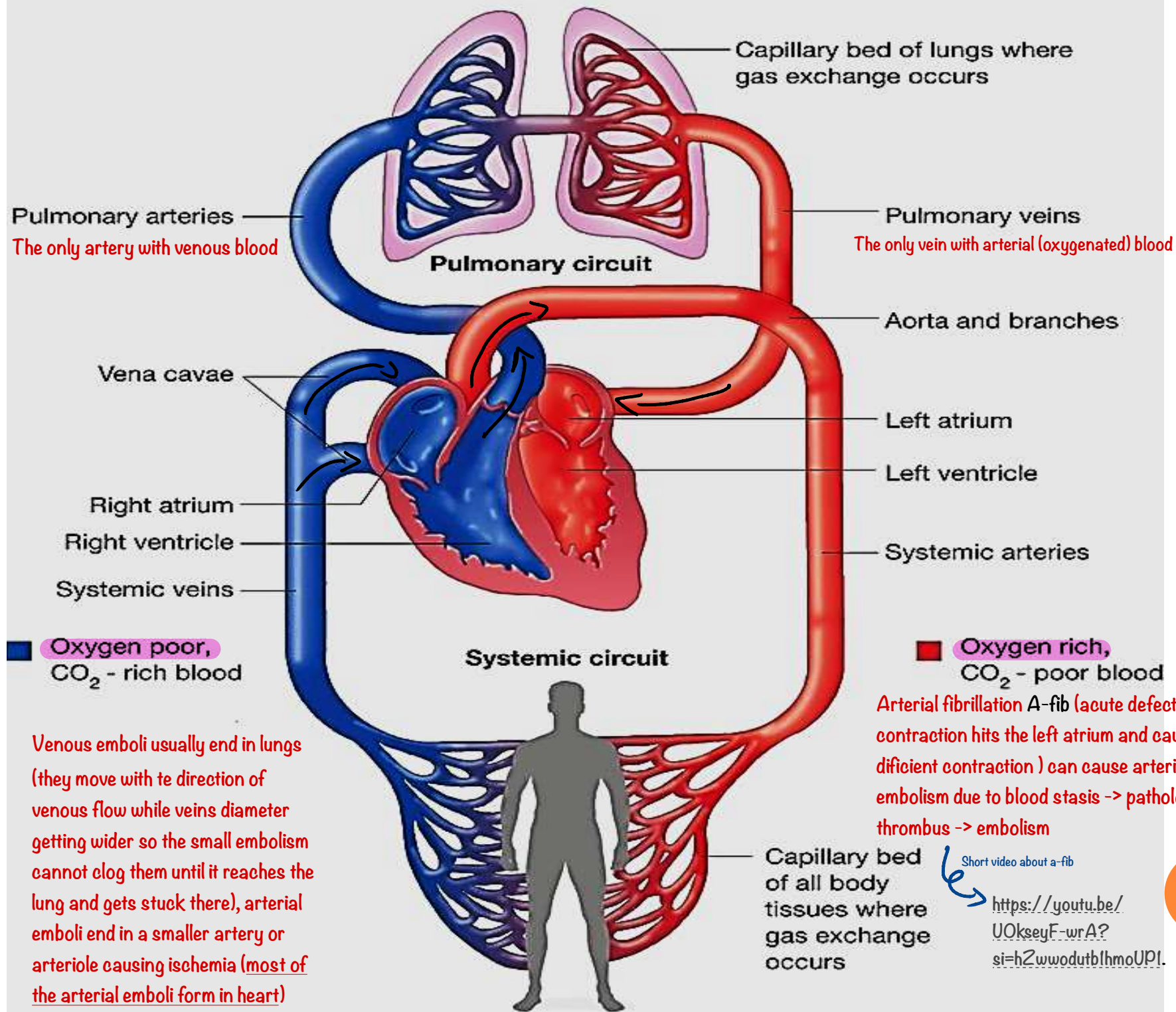
But both are abnormal blood clot



Embolus as you can see in the picture above is relatively small so why it's risky ?

- Because it moves in the circulation and eventually clogs a vessel causing ischemia





Pulmonary arteries  
The only artery with venous blood

Capillary bed of lungs where gas exchange occurs

Pulmonary veins  
The only vein with arterial (oxygenated) blood

Pulmonary circuit

Aorta and branches

Vena cavae

Left atrium

Right atrium

Left ventricle

Right ventricle

Systemic arteries

Systemic veins

■ Oxygen poor, CO<sub>2</sub> - rich blood

Systemic circuit

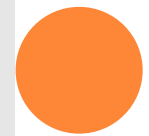
■ Oxygen rich, CO<sub>2</sub> - poor blood

Arterial fibrillation A-fib (acute defect in heart contraction hits the left atrium and causes deficient contraction) can cause arterial embolism due to blood stasis -> pathological thrombus -> embolism

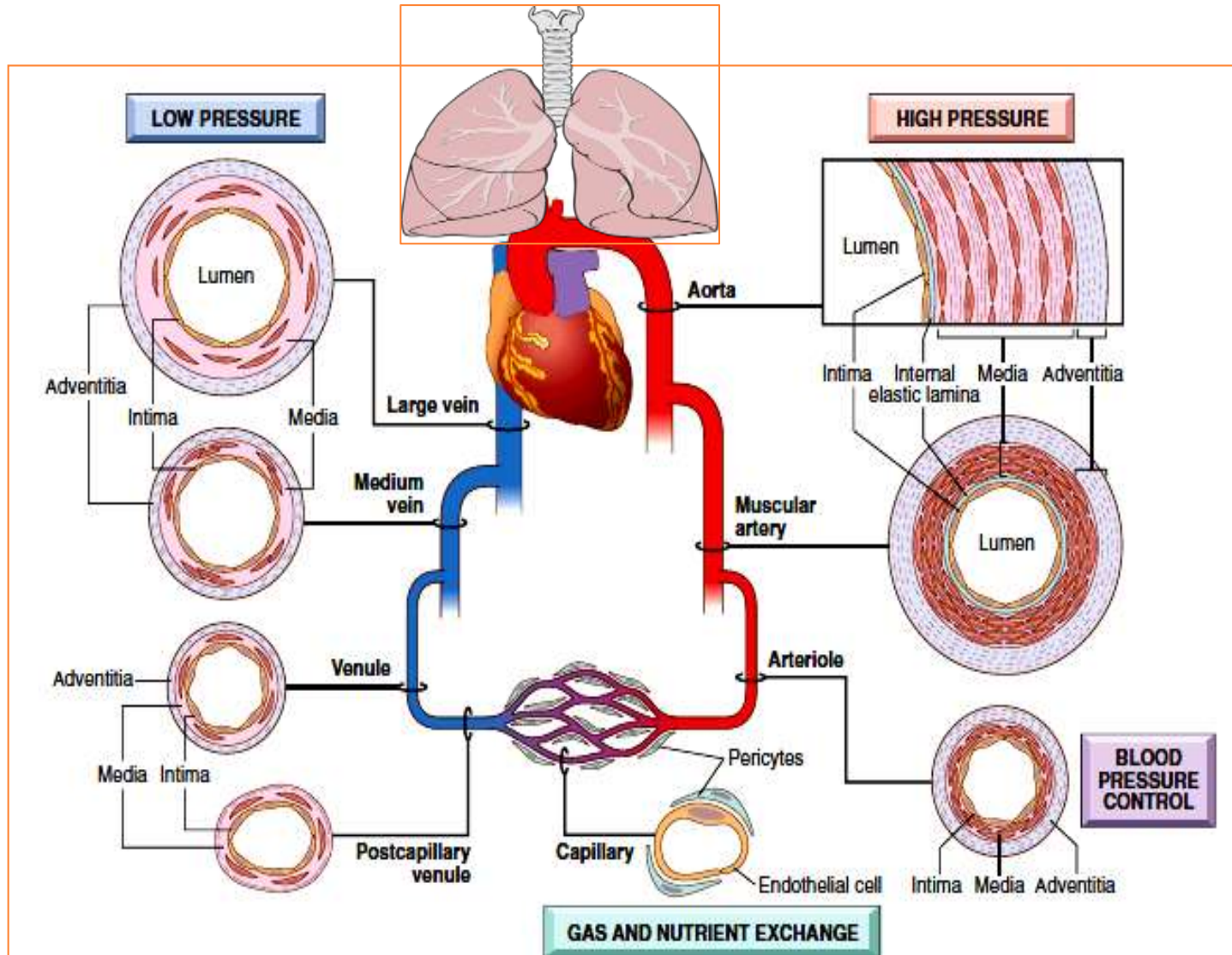
Venous emboli usually end in lungs (they move with the direction of venous flow while veins diameter getting wider so the small embolism cannot clog them until it reaches the lung and gets stuck there), arterial emboli end in a smaller artery or arteriole causing ischemia (most of the arterial emboli form in heart)

Capillary bed of all body tissues where gas exchange occurs

Short video about a-fib  
<https://youtu.be/UOkseyF-wrA?si=hZwwodutb1hmoUPl>



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



*Emboli Types (according to site of origin):*

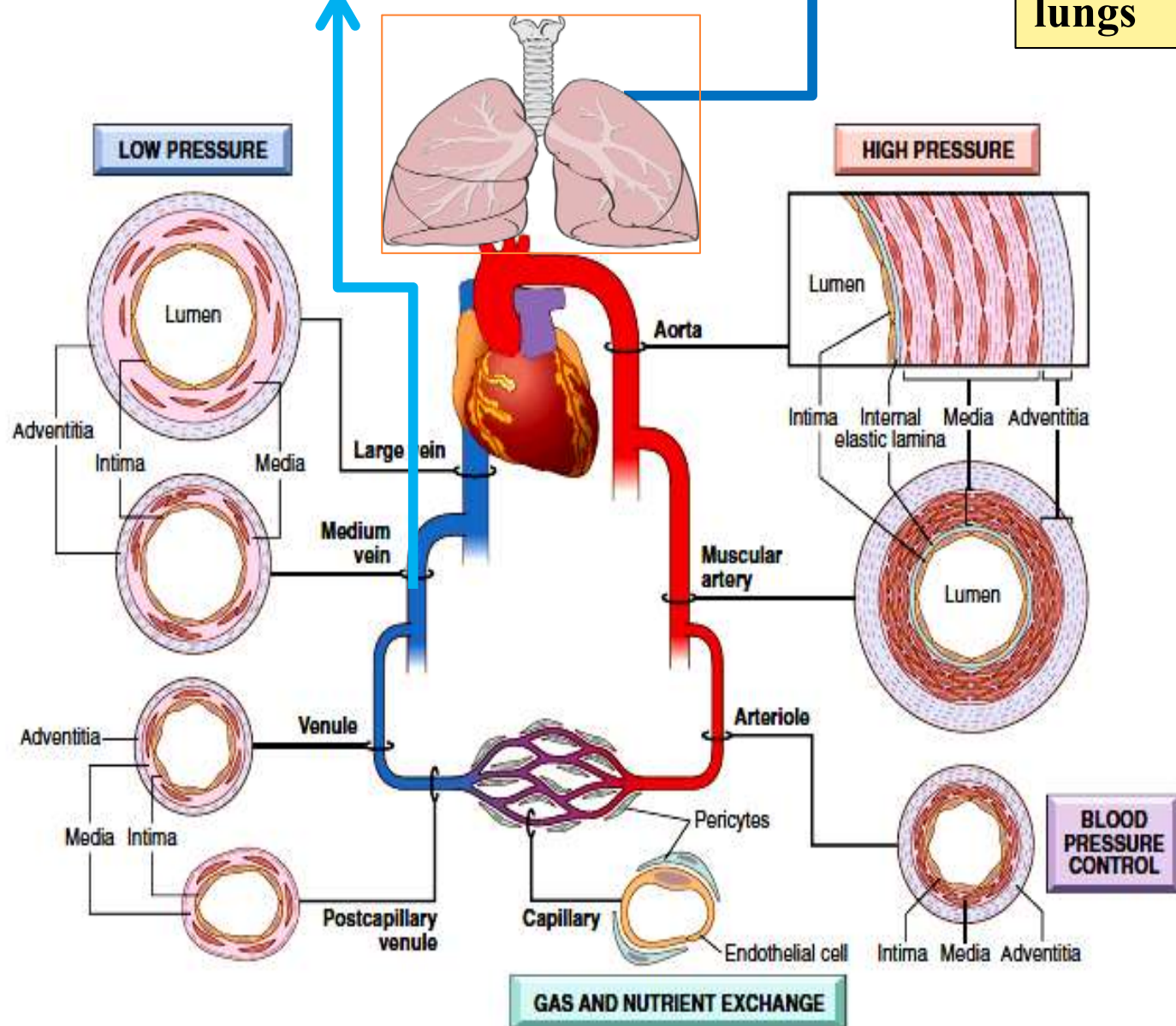
**1- venous**

**2- arterial (systemic) emboli**



**Origin of most venous emboli = lower limbs**

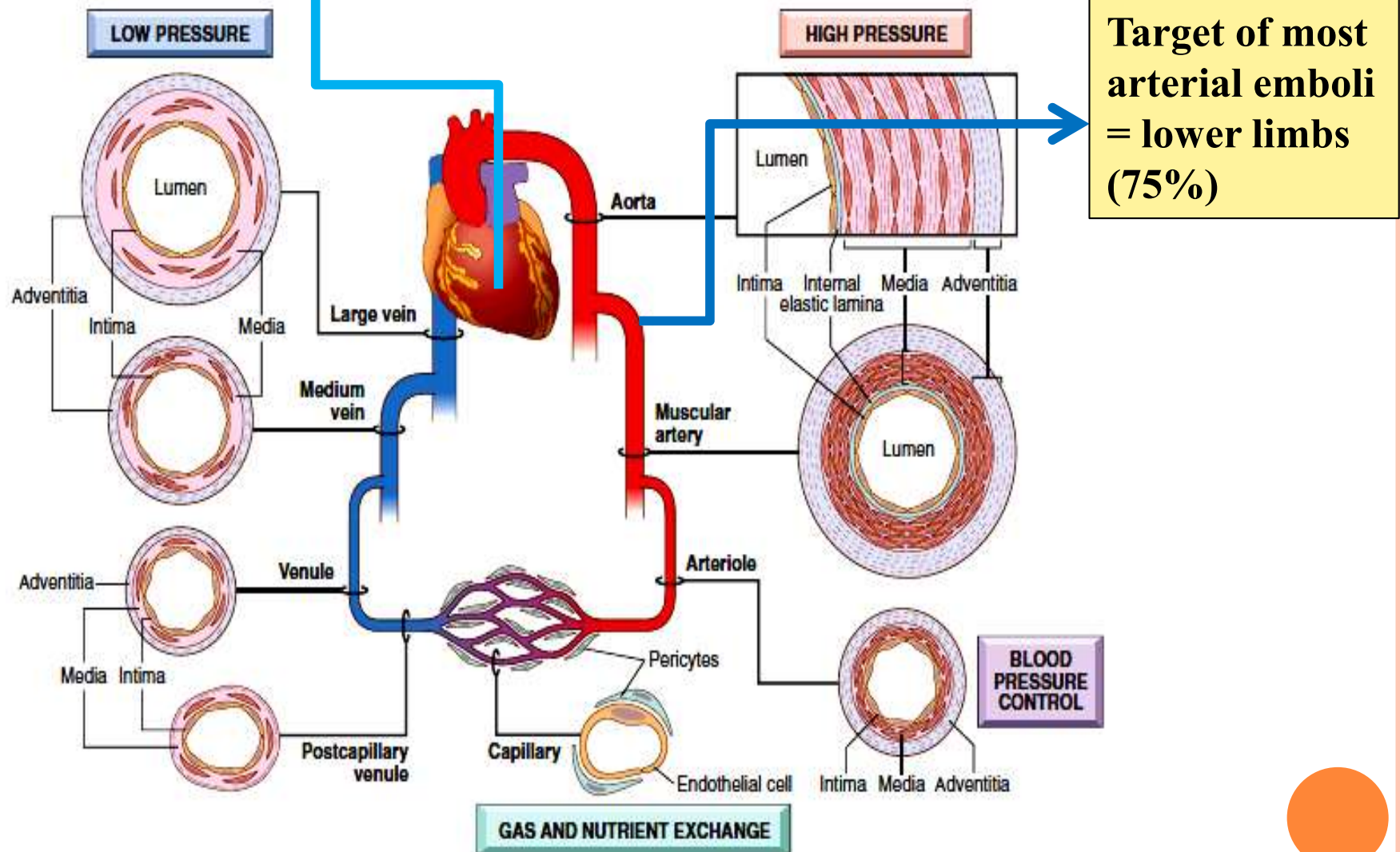
**Target of most venous emboli = lungs**



**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.



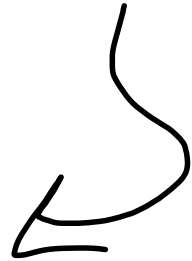
**Origin of most arterial emboli = heart chambers**



**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.

So	Cause.	Target
venous	lower limb(DVT)	Lung
Arterial (systemic)	Heart chambers	Lower limbs(common) Any tissue

- *Emboli result in partial or complete vascular occlusion.*
- *consequences of embolism: ischemic necrosis (**infarction**) of downstream tissue*

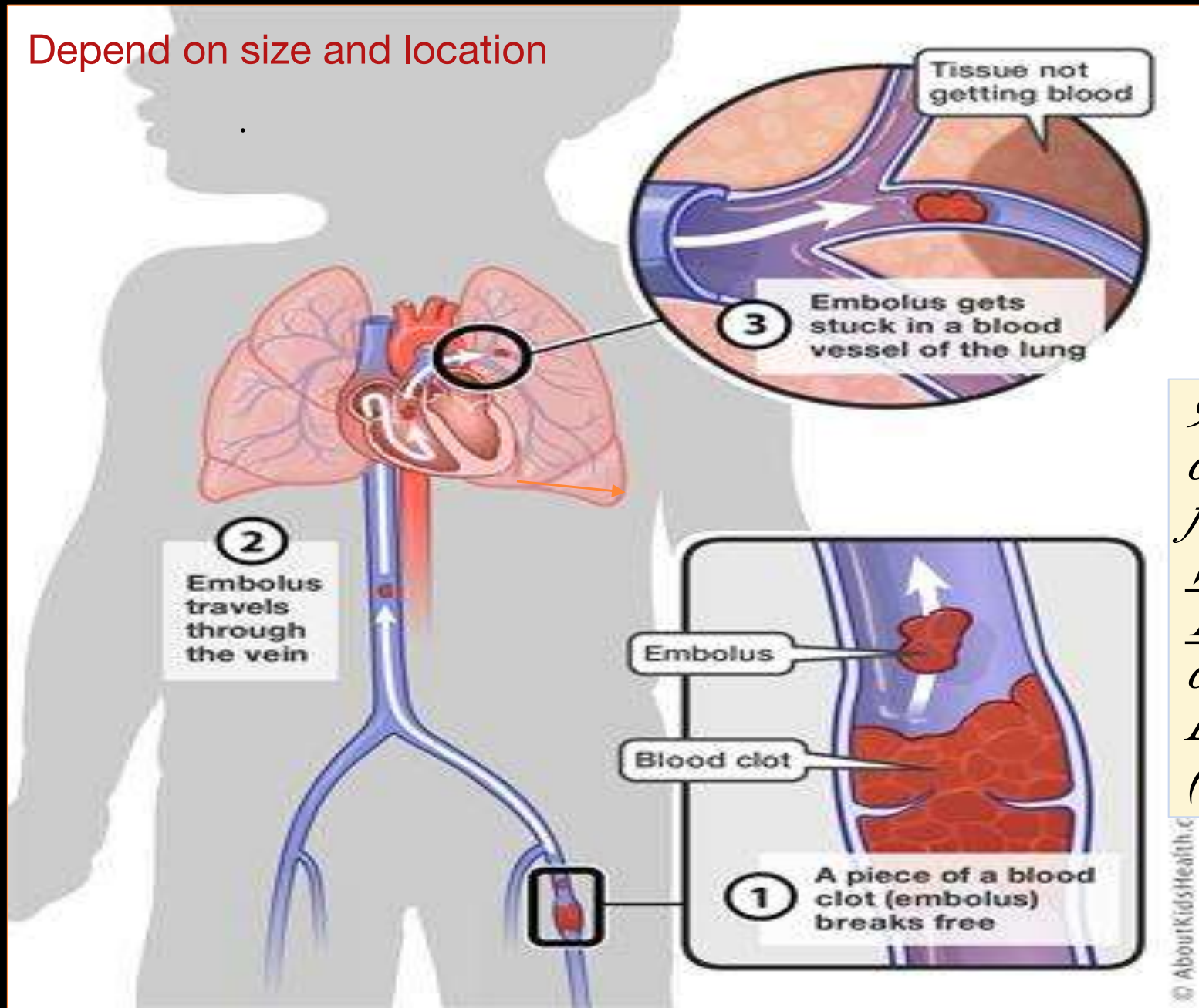


Like thrombus



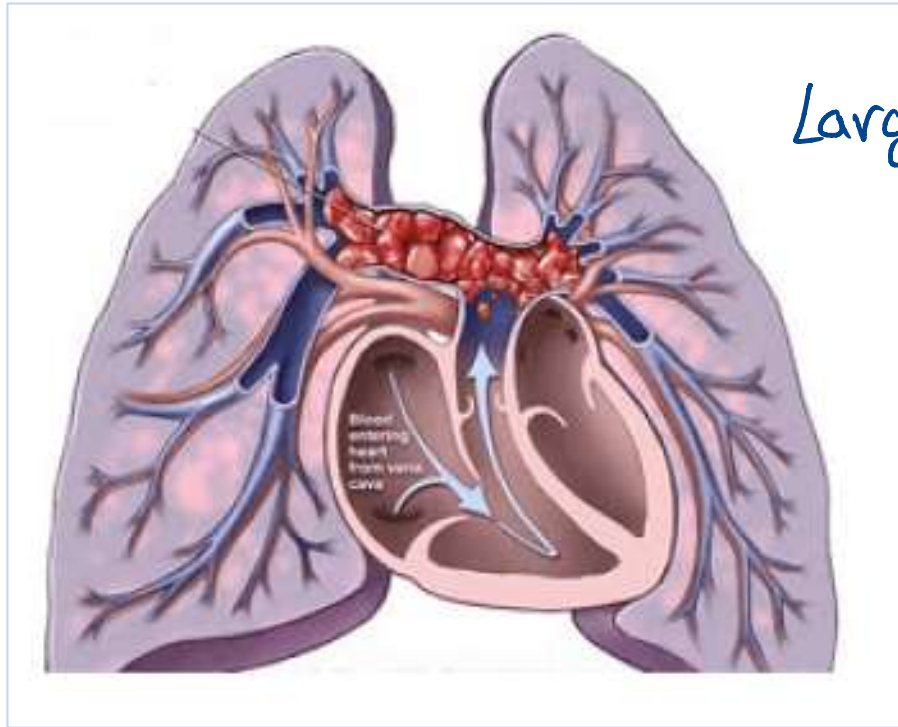
# VENOUS THROMBI

Depend on size and location



*95% originate from DEEP VEINS THROMBI of Lower Limbs (DVT)*





Large

***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*



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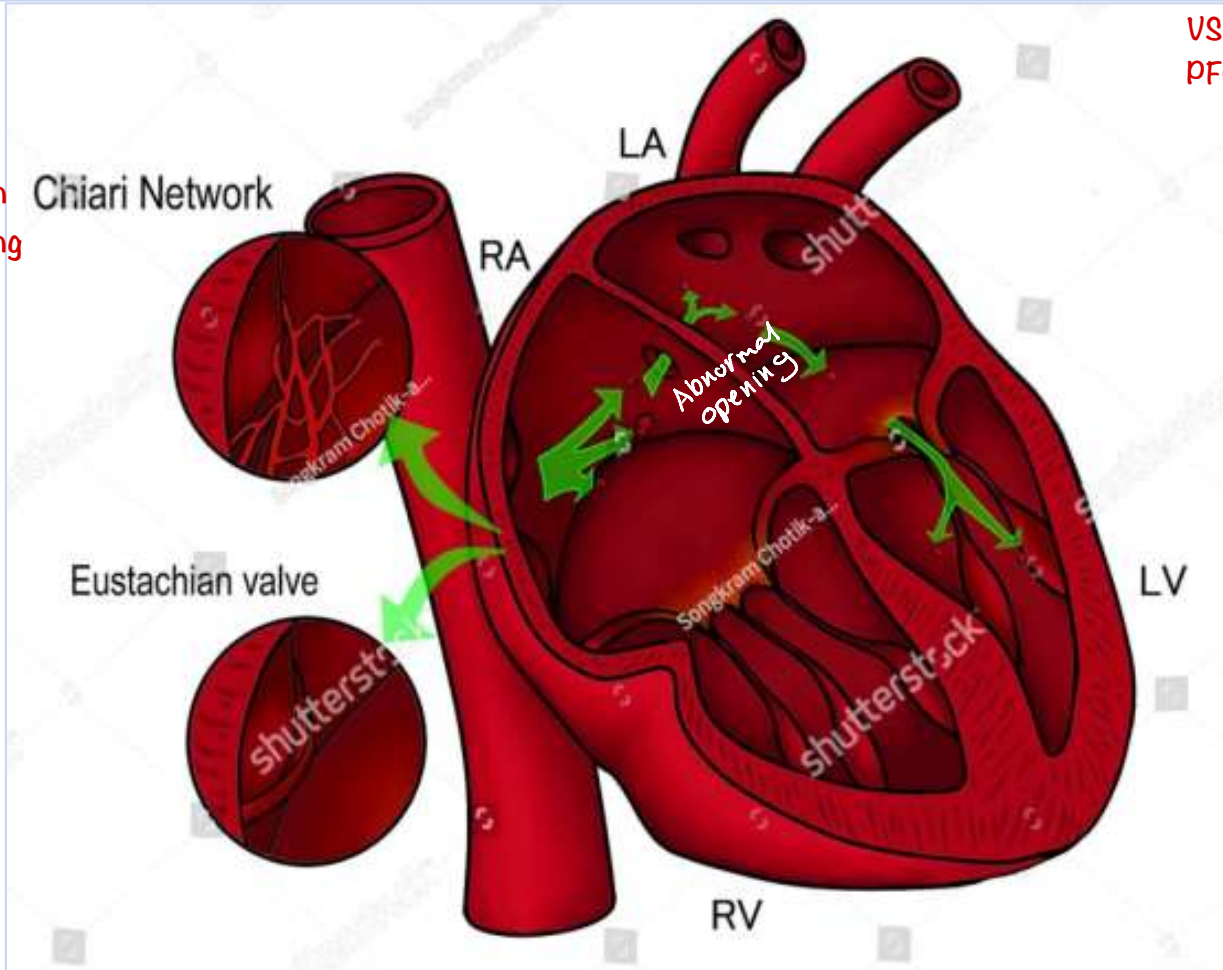


Turns to an arterial embolus

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

Only happens in abnormal conditions when there's a preexisting condition in the heart causing mixing of venous and arterial blood like abnormal openings between the two atria or ventricles

ASD : Atrial septal defect  
VSD : Ventricular septal defect  
PFO : patent foramen ovale



# CLINICAL CONSEQUENCE OF PULMONARY THROMBOEMBOLISM :

○ **Asymptomatic** (60%- 80%; *small*)

○ *Pulmonary infarction* (*large*)

○ *Pulmonary hemorrhage*

The consequences of the embolism depends on 8

- the diameter of the embolus
- The blood vessel being occluded
- The number of emboli (if they were multiple)
- The time of the formation of the emboli

Whith long history and recurrent embolisms

○ *Pulmonary Hypertension* and *right ventricular failure*: (showers of emboli over a long time)

↓ If more than 60% obstructed

○ *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



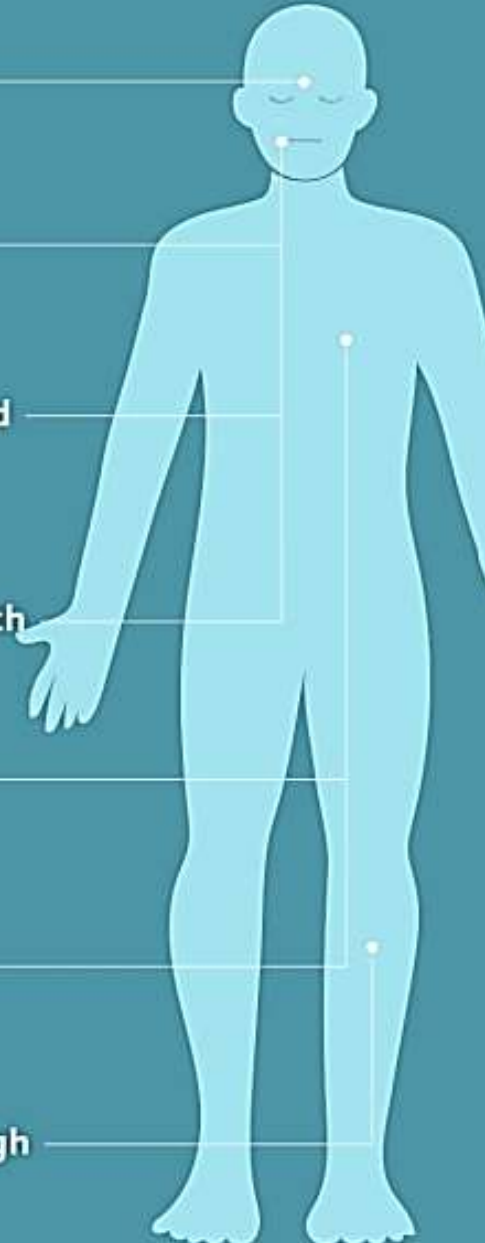
wheezing



dull chest pain



pain in calf or thigh



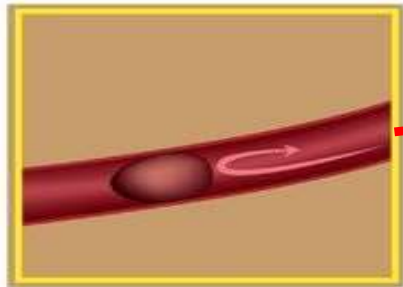
*non-specific symptoms*



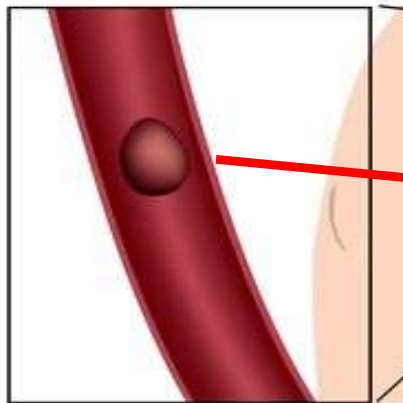


# ARTERIAL EMBOLI

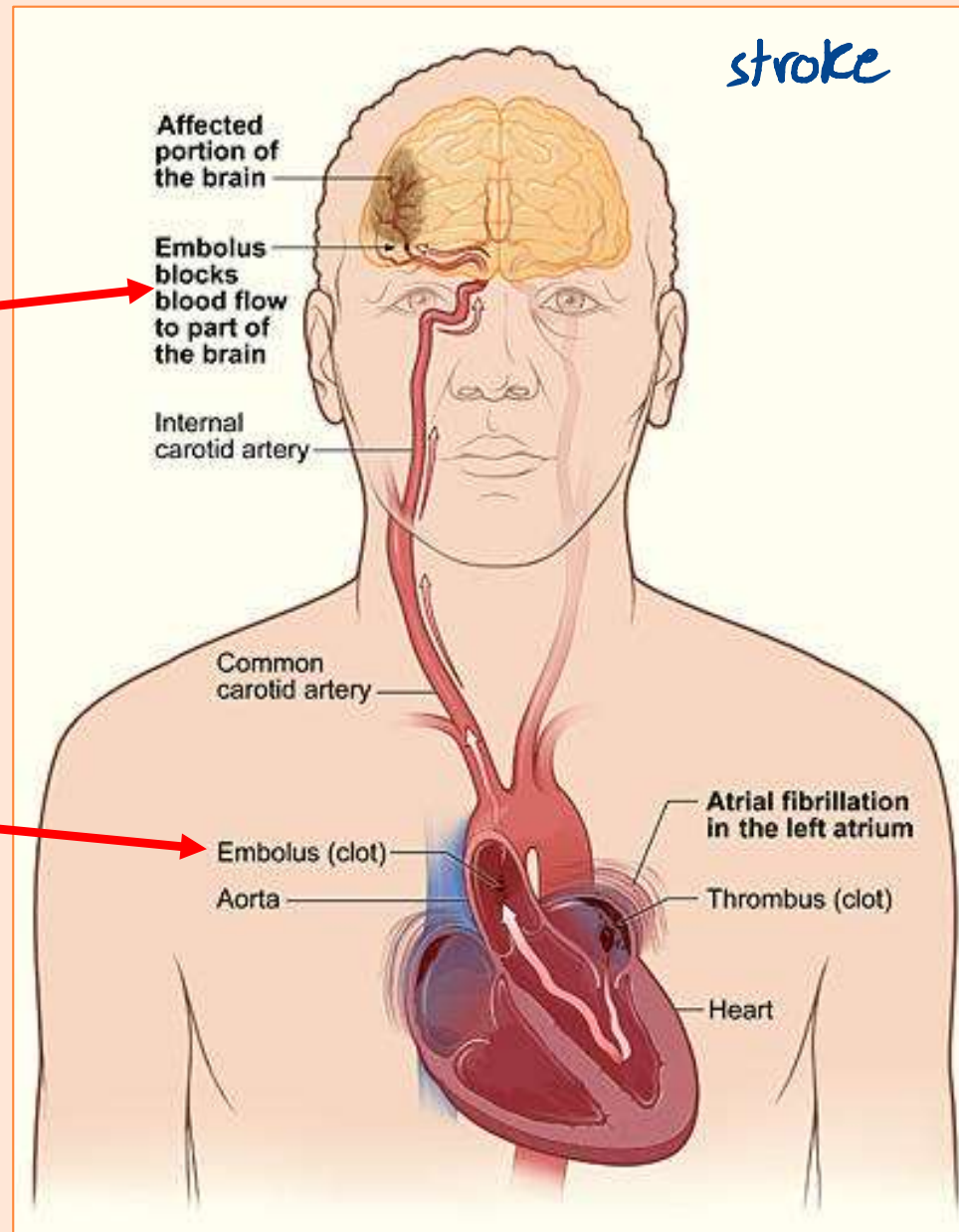
*stroke*



3. Clot blocks an artery in the brain, causing stroke



2. Blood clot travels in the bloodstream



# ○ Systemic (arterial) thromboembolism

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

*causes: -2/3 Lt. ventricular failure*

- $\frac{1}{4}$  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: How can fat (large particles) get into circulation?

why

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

Bones have BM and  
BM have fat

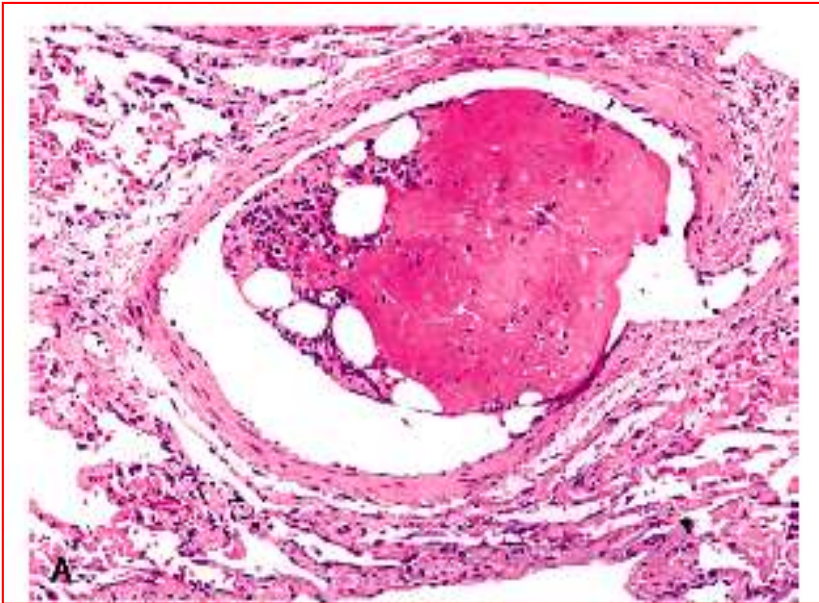
## ○ Results:

- 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

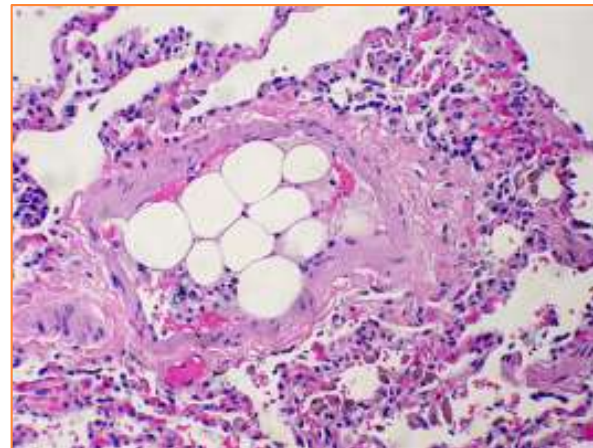


*FAT EMBOLUS = FAT GLOBULES + HEMATOPOIETIC CELLS*



\* Fat looks white due to washing during processing

Blood vessel



- **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* \*1/100 patients may die due to fat embolism
- *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.*

*\* DIC can happen*

*\* Why thrombocytopenia happens?*

*Consumption of platelets*

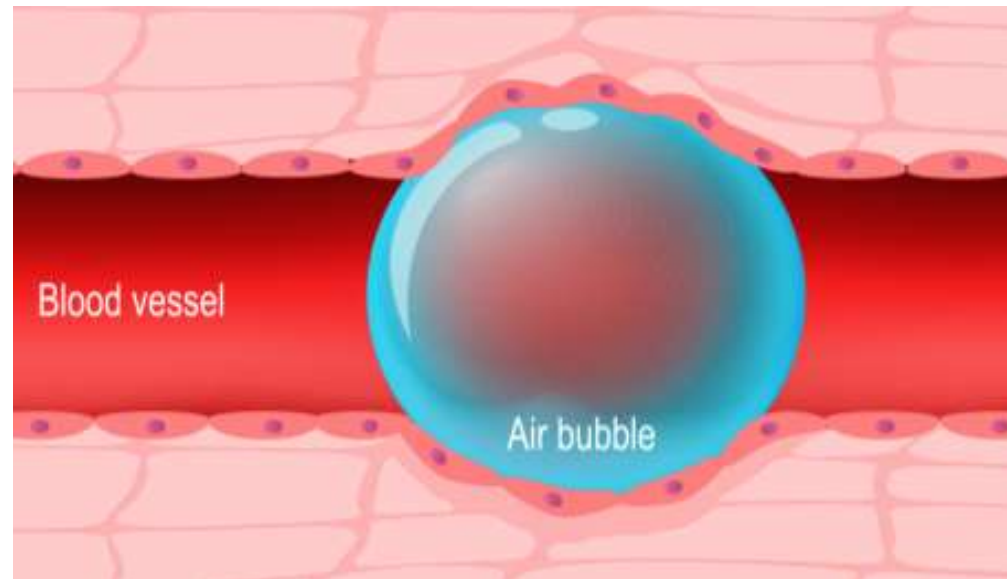
*\* in fat embolism syndrome in addition to mechanical obstructions we will have immune response and it's more important because it's systemic*



# Air Embolism

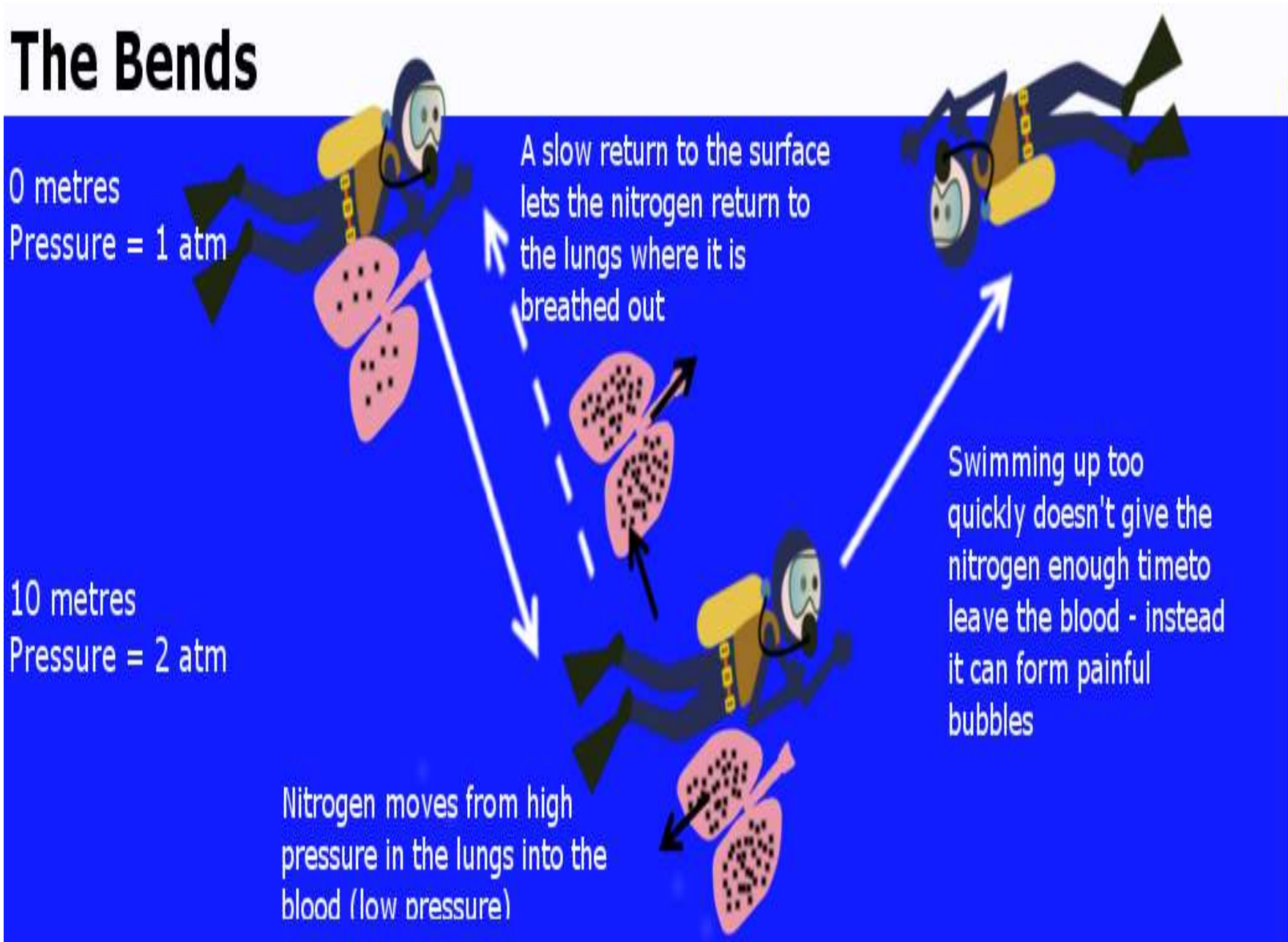
## ○ Causes:

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



# *DECOMPRESSION SICKNESS (THE BENDS)*

## The Bends





\* Why nitrogen?

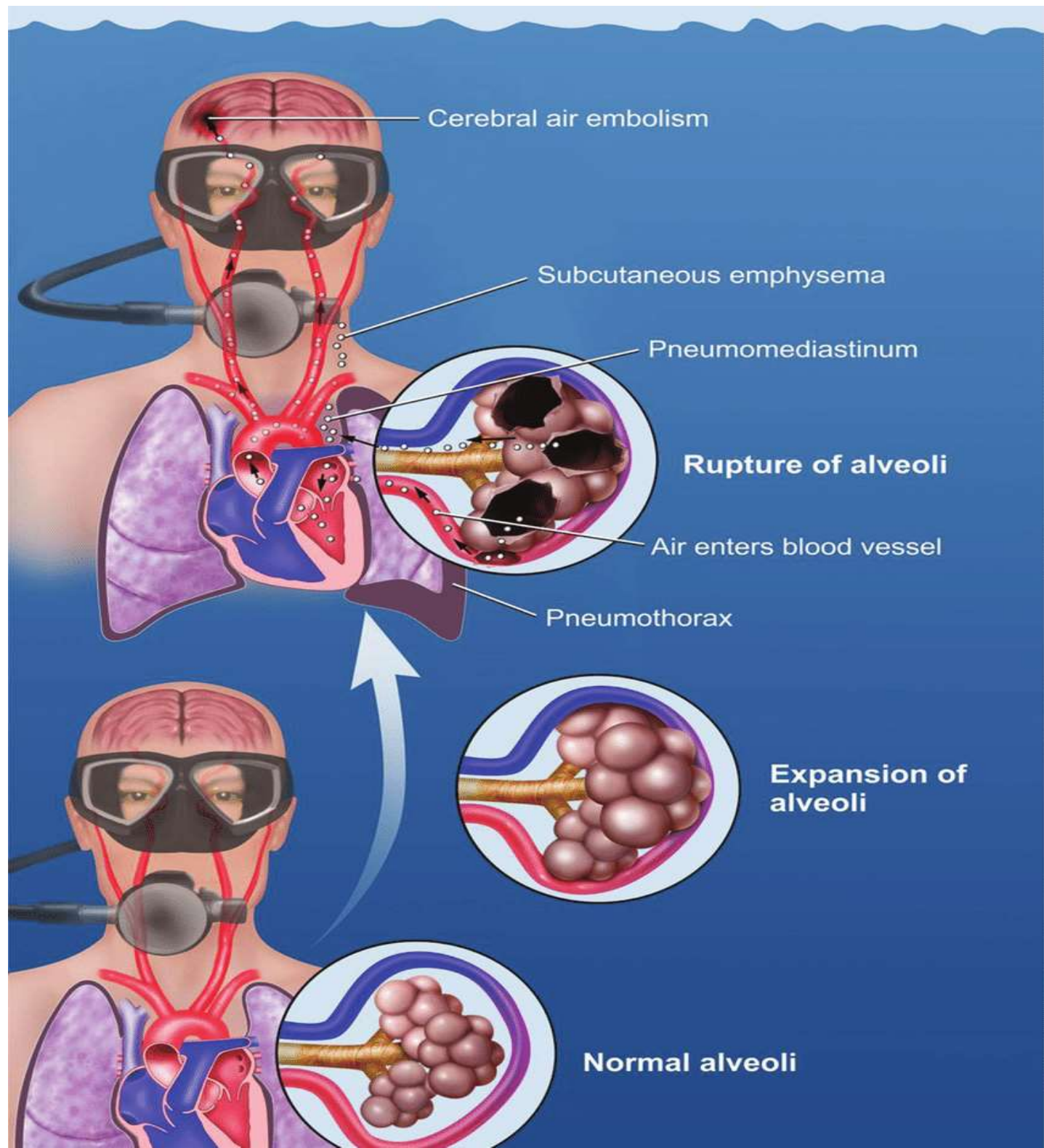
80% of the air in our lungs is nitrogen

Under sea pressure is higher so nitrogen moves from the higher pressure area (lungs) to the lower pressure area (blood) causing air embolism

\* well-trained divers ascend to the sea surface (while diving) gradually and slowly to give enough time for the nitrogen to return slowly to the lungs avoiding air embolisms

\* untrained divers could ascend quickly therefore an air (Nitrogen) bubble forms in their circulation causing mechanical obstruction —> air embolism

++ the rapid expansion of air in alveoli could rupture them leading to pneumothorax, emphysema



# 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 / complicated caesarean section*
- *infusion of amniotic fluid into maternal circulation via tears in placental membranes and rupture of uterine veins.* not in the fetal circulation
- **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 could contains fetal epithelial cells, ceratin or maybe hairs and these components will trigger an immune reaction (more severe than mechanical occlusion)*



# Amniotic Fluid Embolism

Amniotic fluid escapes the uterus, enters the uterine vasculature, and travels through the veins to the heart and lungs

*Close-up View of the Lung Tissue*

Pulmonary microvasculature

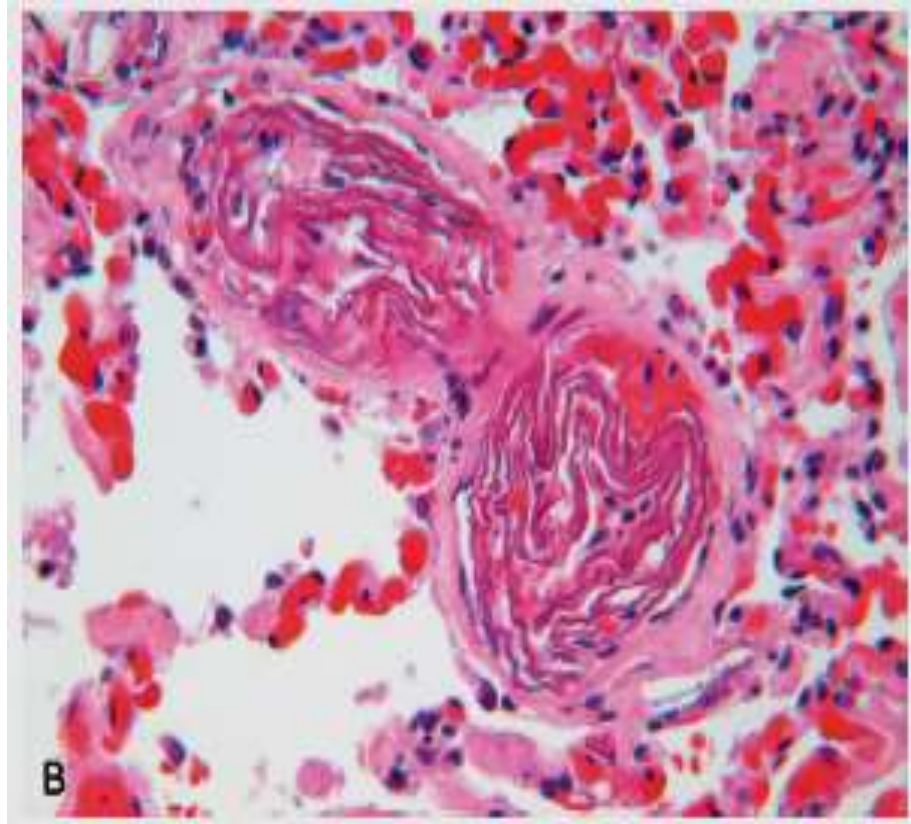
Amniotic fluid, hair, and squamous cells block vessel, preventing blood flow to the air sacs



TrialExhibits

# AMNIOTIC FLUID EMBOLUS. KERATIN AND FETAL SQUAMOUS CELLS IN PULMONARY ARTERIOLES

*Lumen is completely occluded by these cells*



# 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***



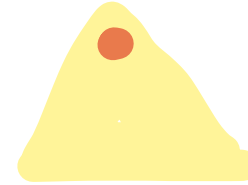
# MORPHOLOGY OF INFARCTS

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

\*Red means there's blood in the infarction site

\*White means there isn't

- *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:

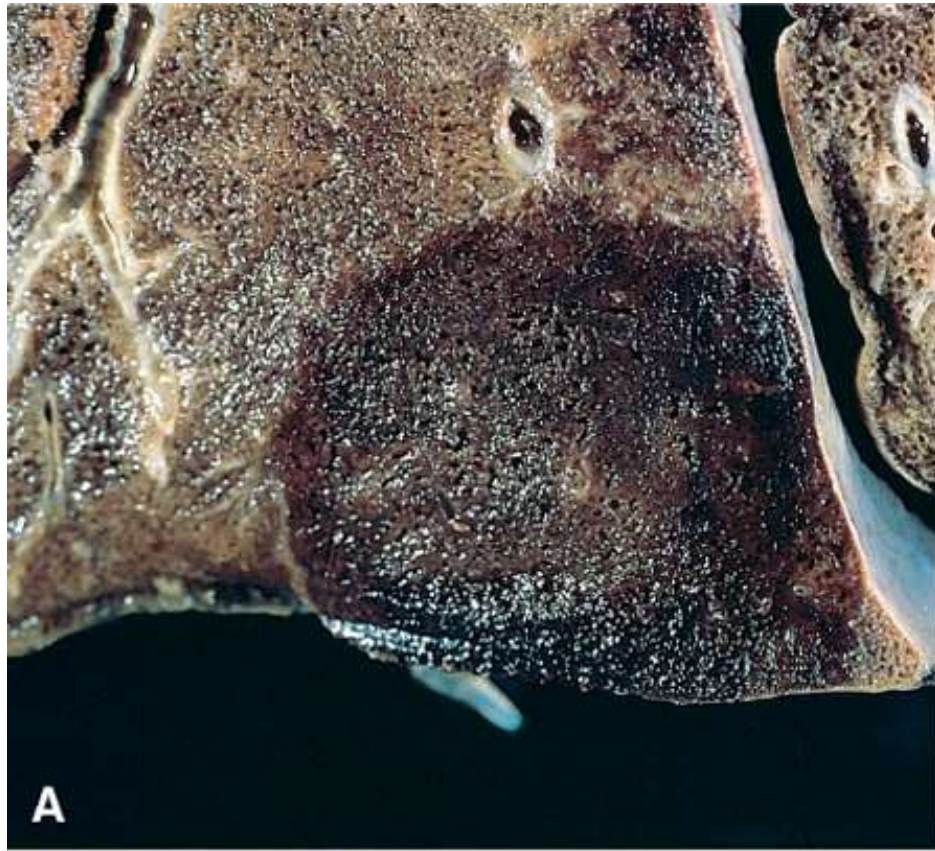
- occur when infarct is superimposed by infection;
- examples:

**1- infected vegetations**

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

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





Red

**Red and  
white  
infarcts.**

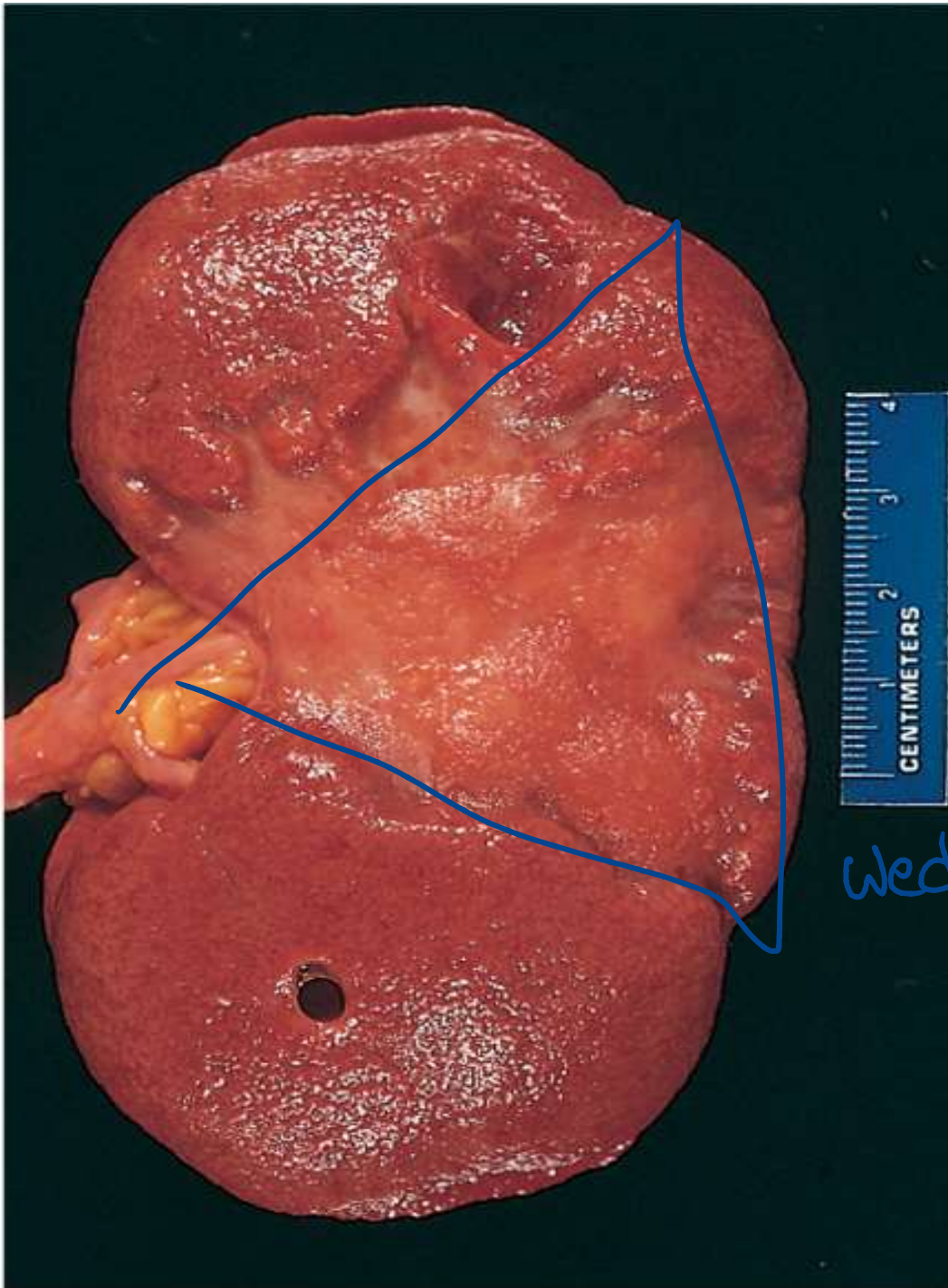
**A → lung**

**B → spleen**



white





wedge shaped

white?  
- solid organ

## 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.*



قالوا قد انتصر الطبيبُ على المُحالِ من الأمور  
زرعَ الجماجمَ والقلوبَ وشدَّ أقفاصَ الصدورِ  
فأجبتهم : ومتى ستُرفعُ رايةُ النصرِ الأخيرِ ؟  
زرعُ الضمائرِ في النفوسِ العارياتِ من الضميرِ

١٢/١٠/٢٠٢٣ يوم استشهد المصابين والرضع في مجمع الشفاء الطبي نتيجة  
إنهاء الوقود، ويوم نهشت الكلاب جثث الشهداء المحاصرة أمام عالم بلا ضمير.