

# **EMBOLISM AND INFARCTION**

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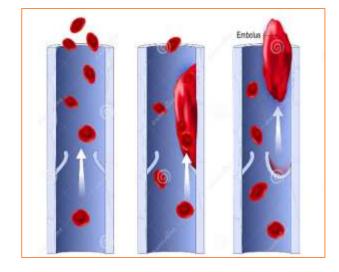
# **EMBOLISM**:

• 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

• <u>Types (according to composition of emboli):</u>

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

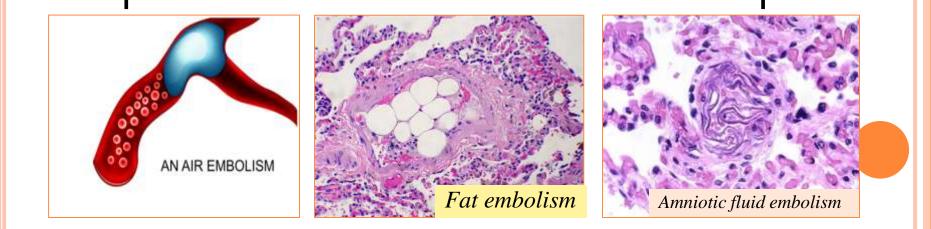
#### **Emboli Types (according to composition)**



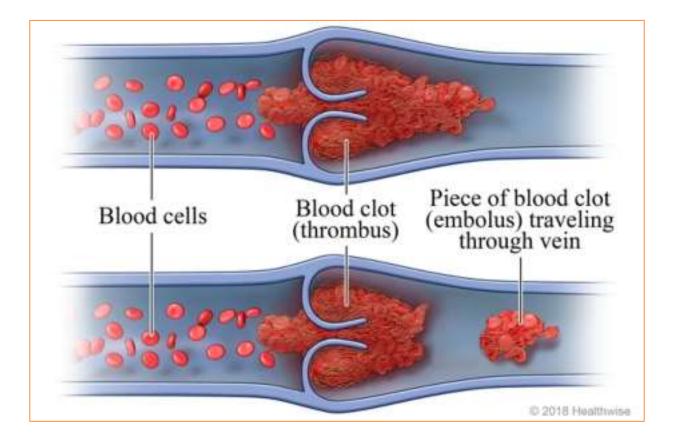
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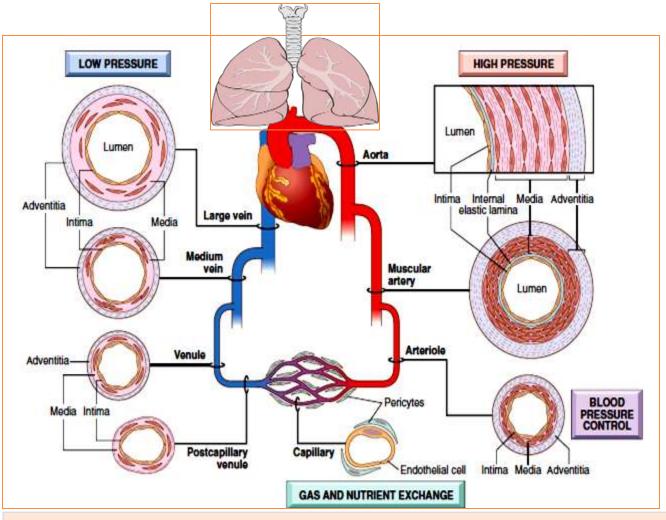




# THROMBUS VS EMBOLUS ....?



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



Emboli Types (according to site of origin):

- 1- venous
- 2- arterial (systemic) emboli

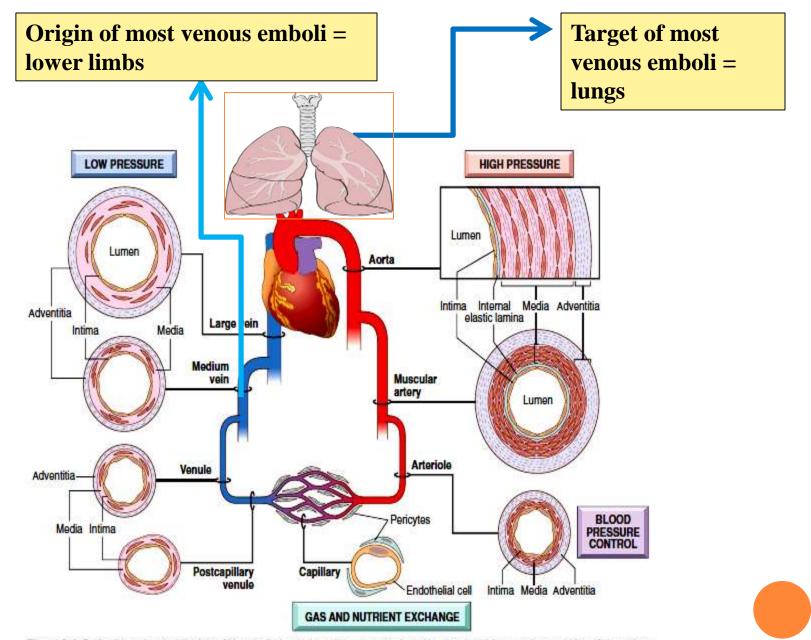


Figure 9-1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.

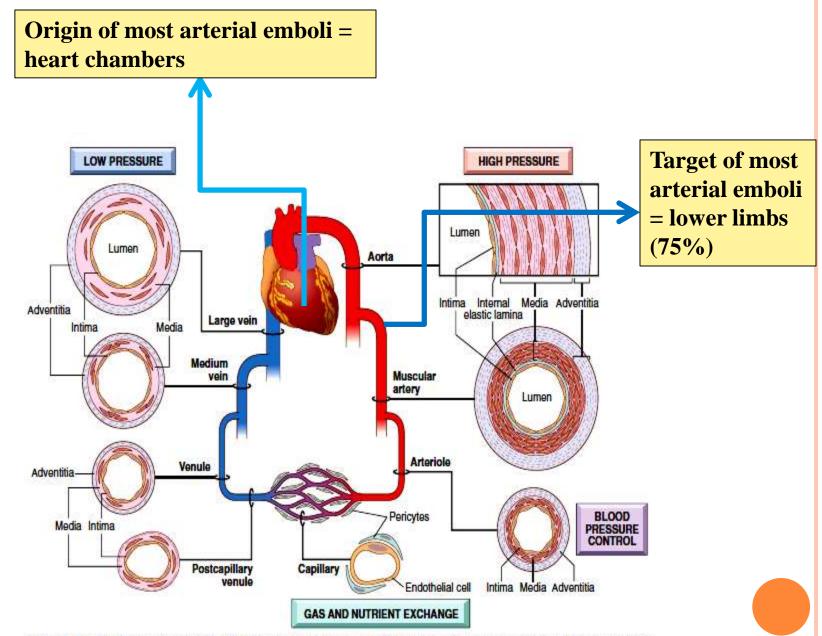
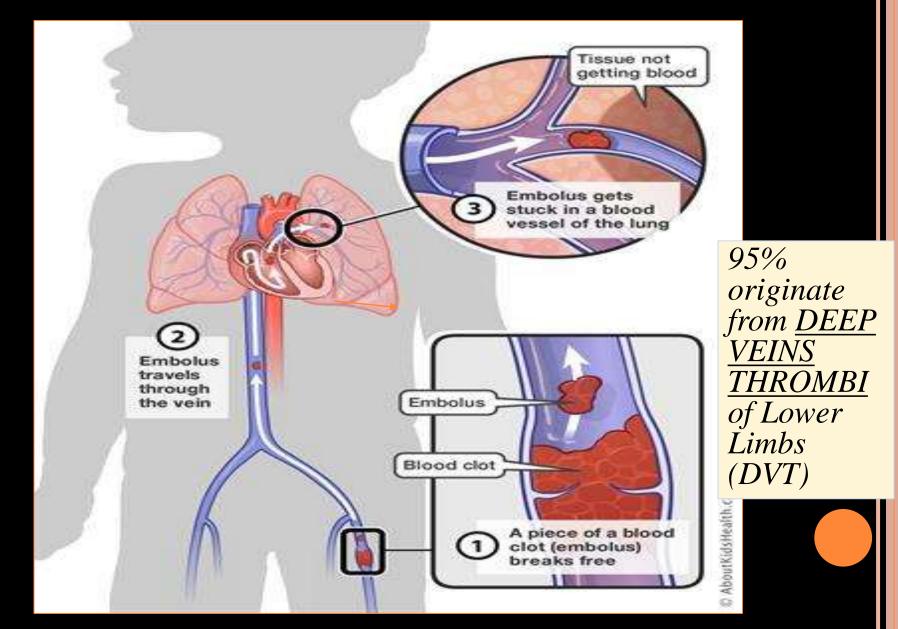


Figure 9-1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.

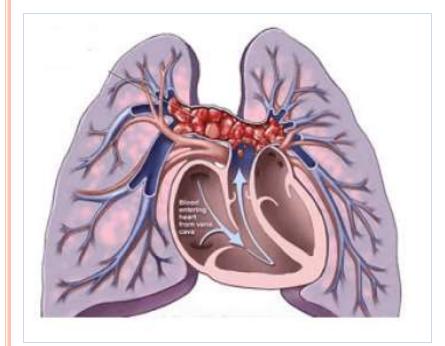
• Emboli result in partial or complete vascular occlusion.

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

## **VENOUS THROMBI**



#### **Pulmonary Thromboembolism-** Special terms

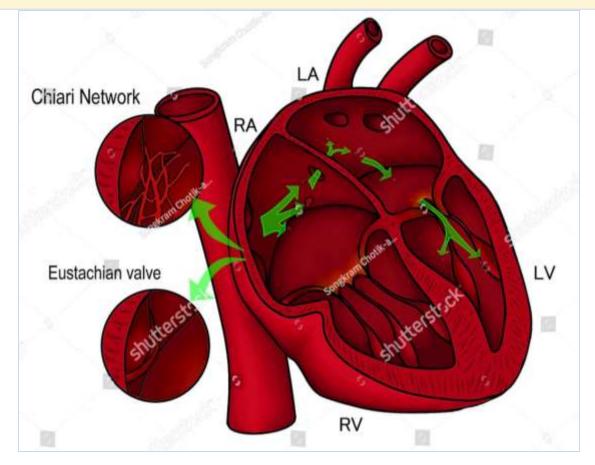


Saddle embolus Large embolus Occluding the Bifurcation of Pulmonary artery TRUNK (FATAL) Embolus derived from a lower extremity deep venous thrombosis and now impacted in a pulmonary artery branch



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

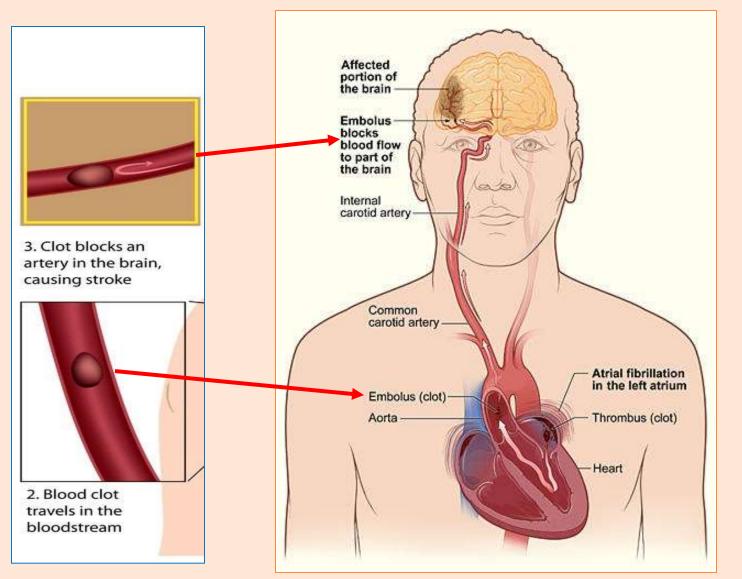


**CLINICAL CONSEQUENCE OF PULMONARY** 

THROMBOEMBOLISM :

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

- $\frac{1}{4}$  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!)

# oFat embolism

- <u>Causes:</u>

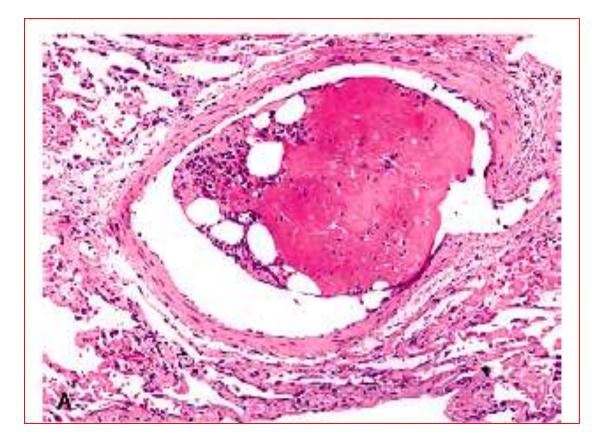
1. Skeletal injury (fractures of long bones)

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  $\rightarrow$  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:

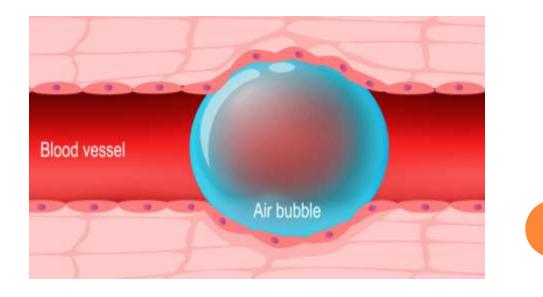
- *Pulmonary Insufficiency* (rapid breathing; shortness of breath)
- Neurologic symptoms (mental confusion; lethargy; coma)
- *petechial rash* (pinpoint rash, found on chest, head, and neck area due to bleeding under skin)
- Fever
- Anemia
- Thrombocytopenia
  Death in 10% of the case

- Symptoms appear 1-3 days after injury

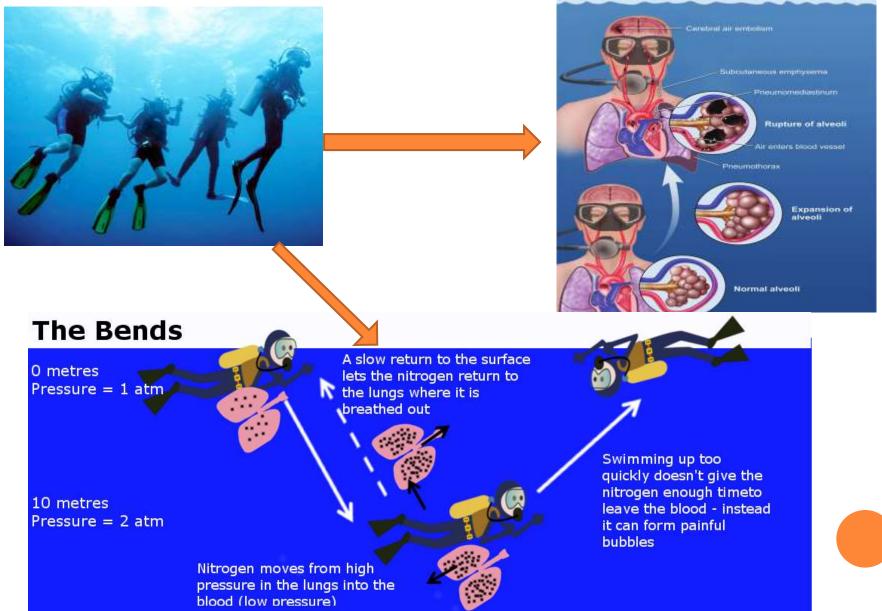
## **Air Embolism**

#### o causes:

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



#### **DECOMPRESSION SICKNESS**



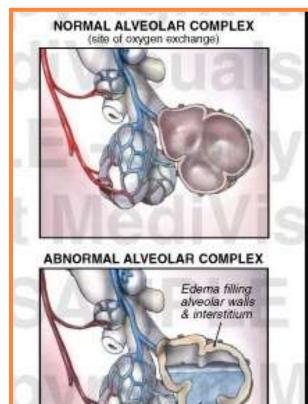
# **AIR EMBOLISM- CLINICAL CONSEQUENCE**

- 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

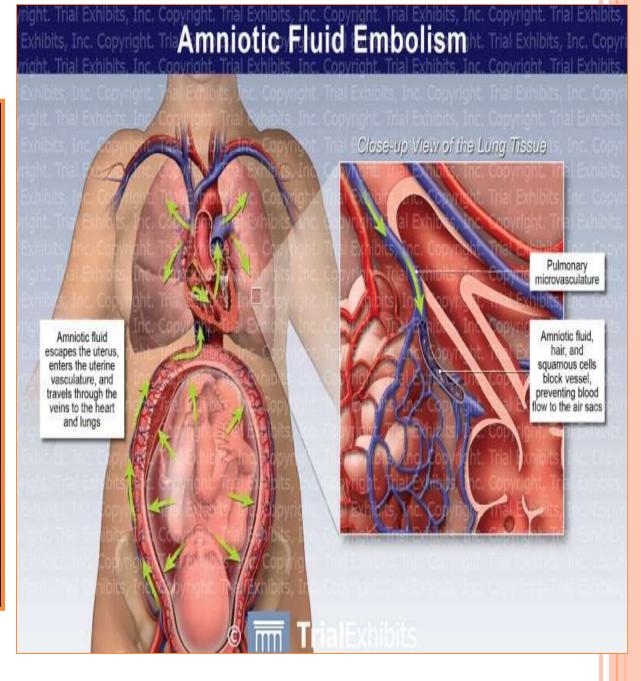
## • Amniotic fluid embolism

- 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:

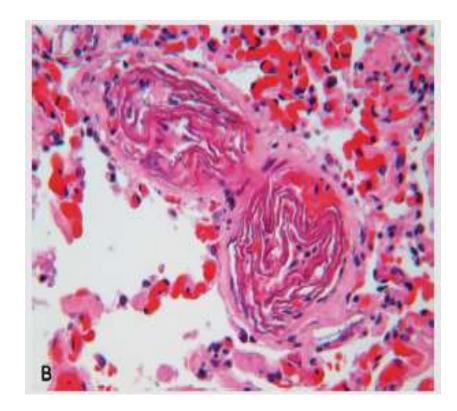
fetal squamous cells, lanugo hair, fat, mucin .....etc within the maternal pulmonary microcirculation



Alveoli fill with fluid: / little oxygen exchange result: ARDS



#### **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: <u>The brain is an **exception**</u> (liquefactive necrosis)
- most infarcts are ultimately replaced by scar

### **RED INFARCTS:**

## o occur in any of the following scenarios:

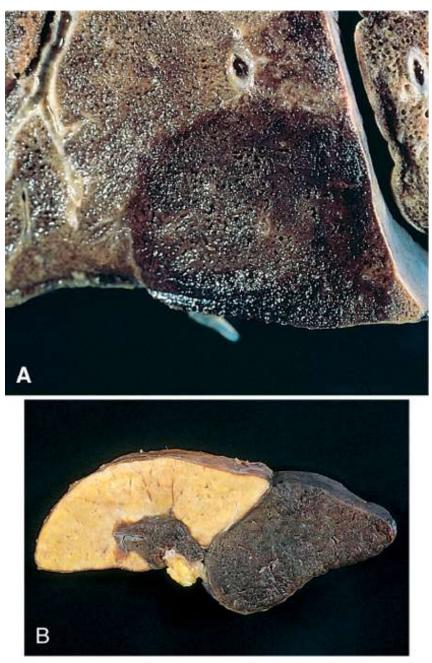
- (1) **venous** occlusions (e.g. ovarian torsion)
- (2) **loose** tissues (e.g. lung) that allow blood to collect in the infarcted zone
- (3) tissues with **dual** circulations (e.g. lung and small intestine)
- (4) previously congested tissues because of sluggish venous outflow
- (5) when flow is **re-established** to a site of previous arterial occlusion and necrosis

# WHITE INFARCTS

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

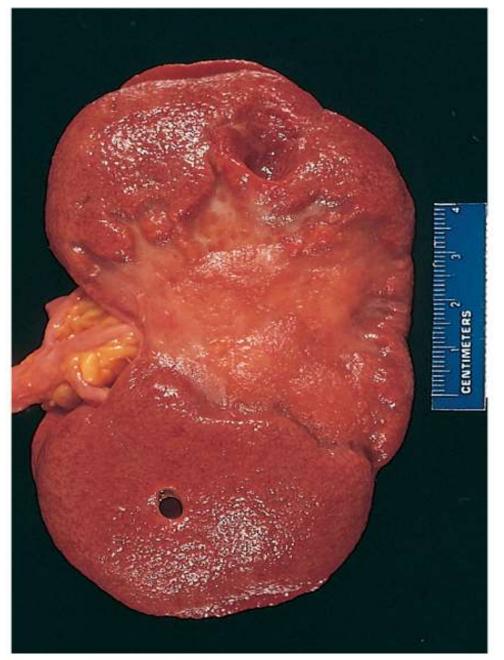
# **Septic infarctions:**

- 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



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Red and white infarcts. A→lung B→spleen



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### kidney **DNEY WH** infarct replaced by a large fibrotic TE scar INFARCT

# 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
- o 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 types venous and arterial embolism. 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.