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Circulatory Disturbances- Embolism

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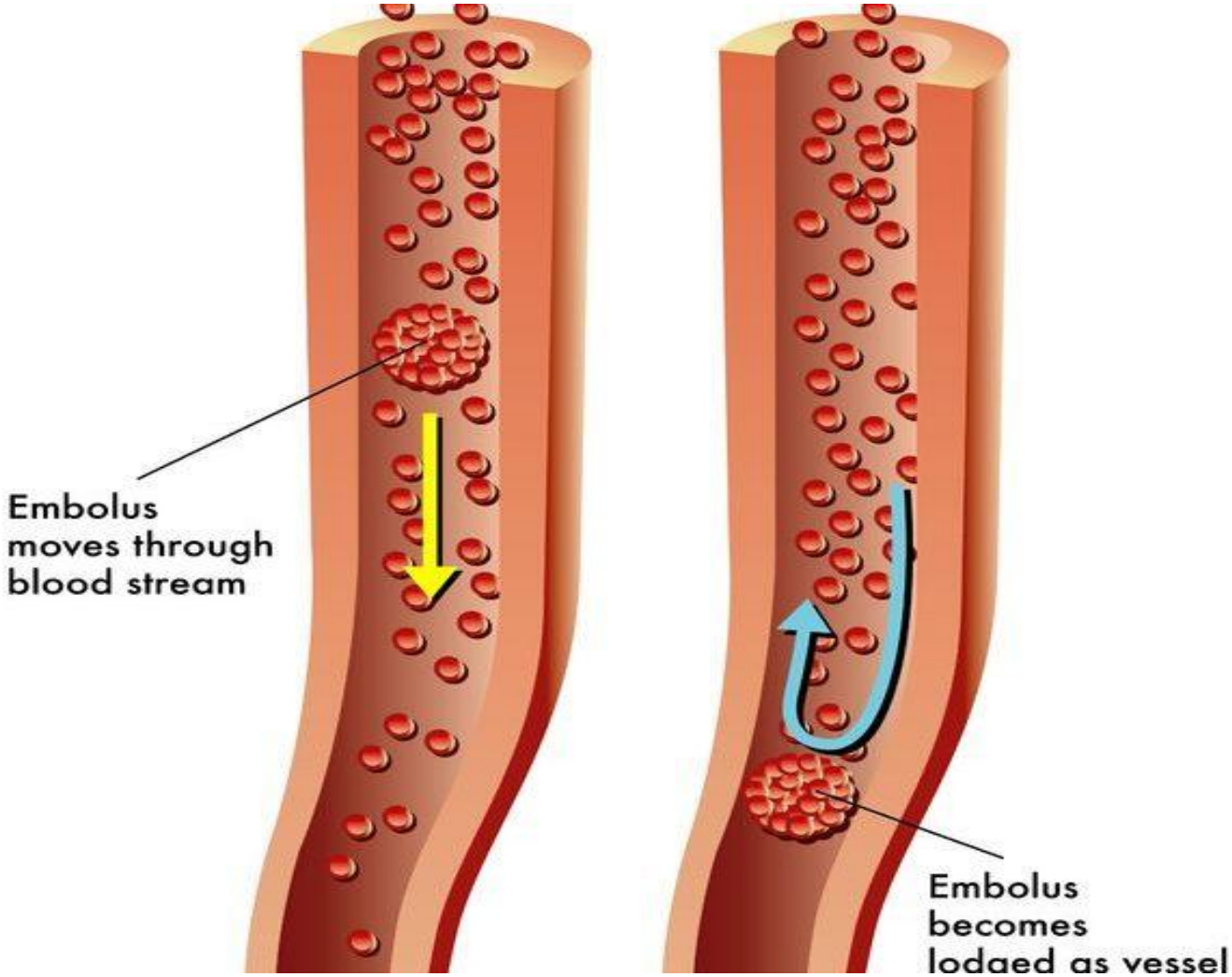
Circulatory Disturbances- Embolism

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

- **Embolism** is the process of partial or complete obstruction of some part of the cardiovascular system by any mass carried in the circulation


Embolus
moves through
blood stream
Embolus
becomes
lodged as vessel

Circulatory Disturbances- Embolism

I) **Depending upon the matter in the emboli**

Solid – Detached thrombi, atheromatous debris (cholesterol emboli, tumor fragments, bone marrow, tissue fragments, bacterial clumps, foreign bodies

Liquid – Fat droplets, oil droplets, amniotic fluid

Gaseous – Air, Nitrogen bubbles

Circulatory Disturbances- Embolism

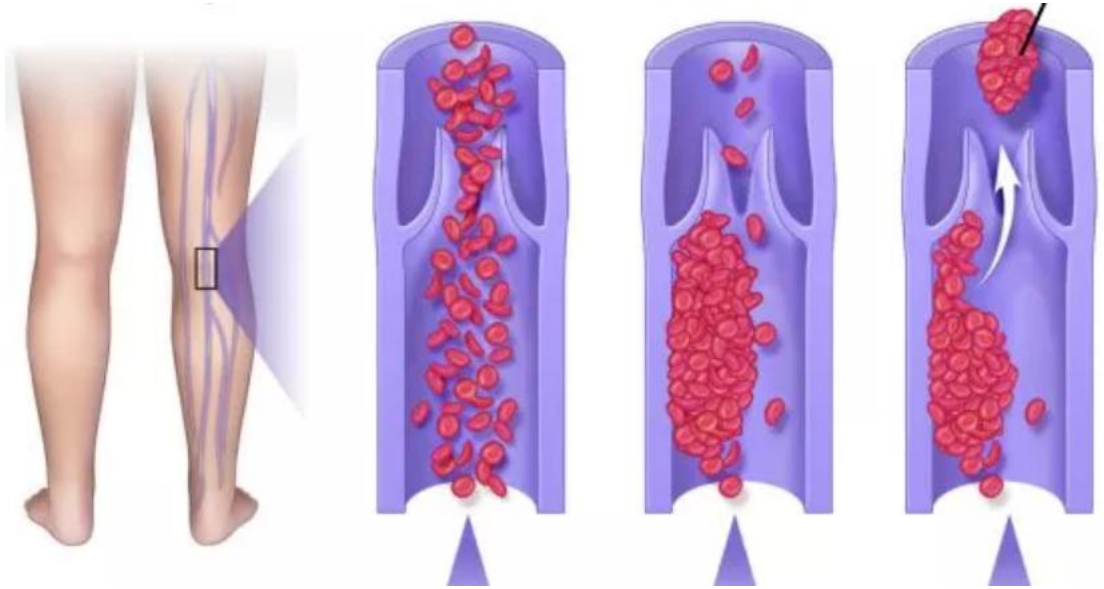
II. Depending upon whether infected or not

Bland when sterile

Septic when infected

Pulmonary Embolism

- Pulmonary emboli - most common form of thromboembolic disease.
- PE is a common and serious causing about 100,000 deaths per year in the US.
- An estimated 20% of individuals with PE die before or shortly after a diagnosis
- > 95% of cases, PE originates from leg DVT. Hence the risk factors are same



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Fate of Pulmonary Embolism

- Most pulmonary emboli (60% to 80%) are clinically silent

Pulmonary Emboli	Clinical outcome
Obstructing > 60% of the pulmonary circulation	Sudden death, acute right heart failure (cor pulmonale)
Obstruction of medium-sized arteries with subsequent vascular rupture	Can result in pulmonary hemorrhage but usually does not cause pulmonary infarction
Embolic obstruction of small end-arterioles	Often produce hemorrhage or infarction
Multiple emboli over time	Pulmonary hypertension and right ventricular failure

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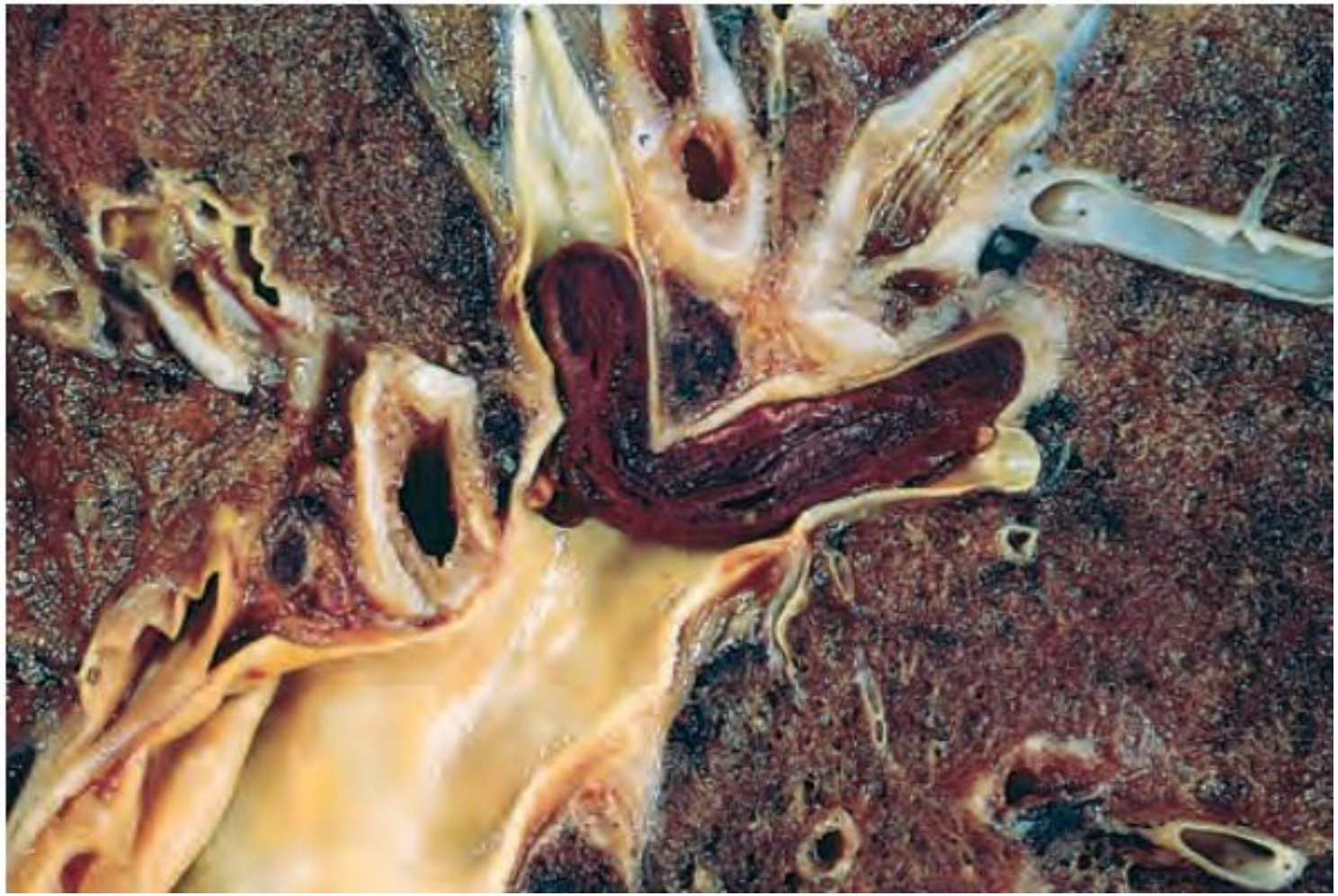
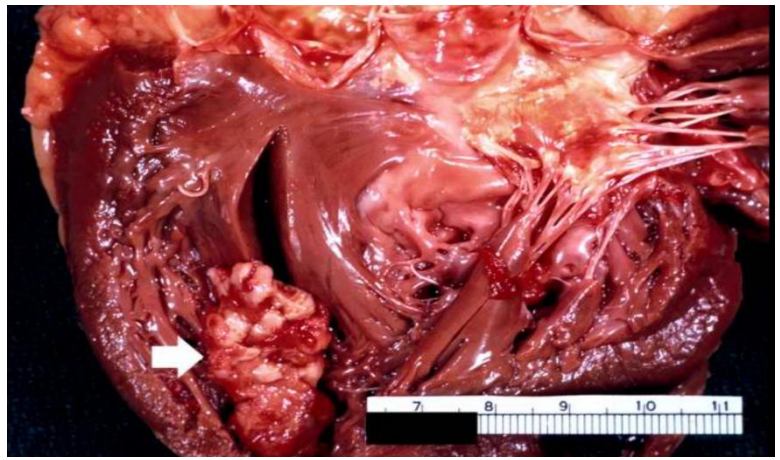
Saddle pulmonary embolus

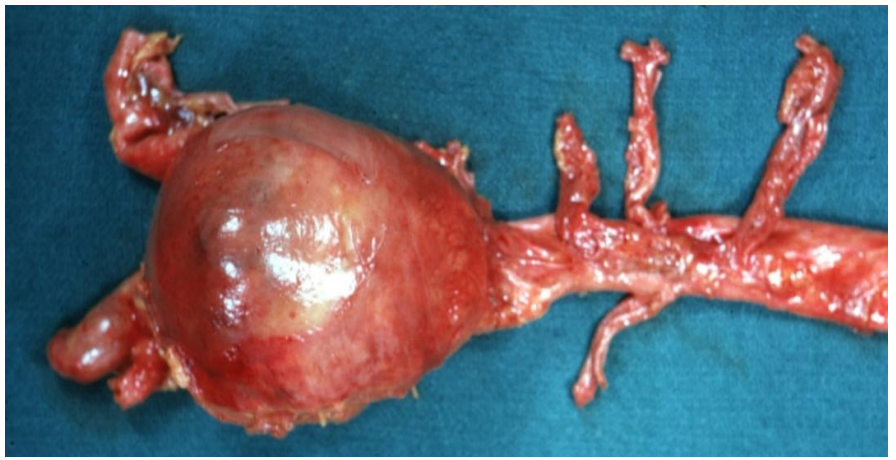


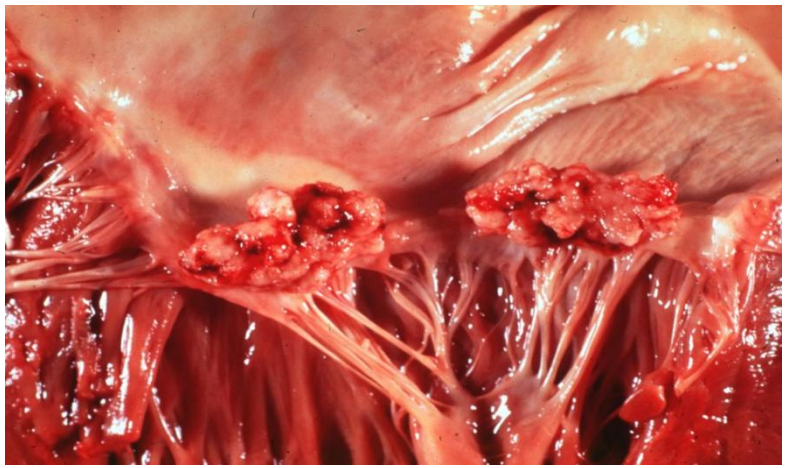
Systemic Thromboembolism

Source

1. 80% intracardiac mural thrombi- 2/3rd left ventricular wall infarct
   1/3rd left atrial fibrillation
2. Rest- aortic aneurysms, atherosclerotic plaques, valvular vegetations, or venous thrombi (paradoxical emboli)
3. 10% to 15% are of unknown origin







Fate of Systemic Embolism

- Lower extremities (75%)
- Brain (10%)
- Intestines,
- Kidneys
- Spleen
- Upper extremities

- Outcome- tissue infarction

(Vulnerability of tissue to ischemia, caliber of vessel, collateral circulation)

Fat Embolism

Fat globules (sometimes bone marrow) (fatal in 5% to 15% of cases)

Clinical Presentation:

Fractures of long bones (90%),

soft-tissue trauma and burns

- 1 to 3 days after injury- tachypnea, dyspnea, and tachycardia; irritability; and restlessness that can progress to delirium or coma, anemia, and thrombocytopenia
- Diffuse petechial rash in these patients with thrombocytopenia

Fat Embolism Pathogenesis

Mechanical obstruction

Fat micro-emboli and associated red cell and platelet aggregates occlude the pulmonary and cerebral microvasculature.

Biochemical injury

Fat globules - free fatty acids - local toxic injury to endothelium, and platelet activation and granulocyte recruitment (with free radical, protease, and eicosanoid release) complete the vascular assault

Diagnosis: Frozen section and special stains (Sudan black B, Oil red O)

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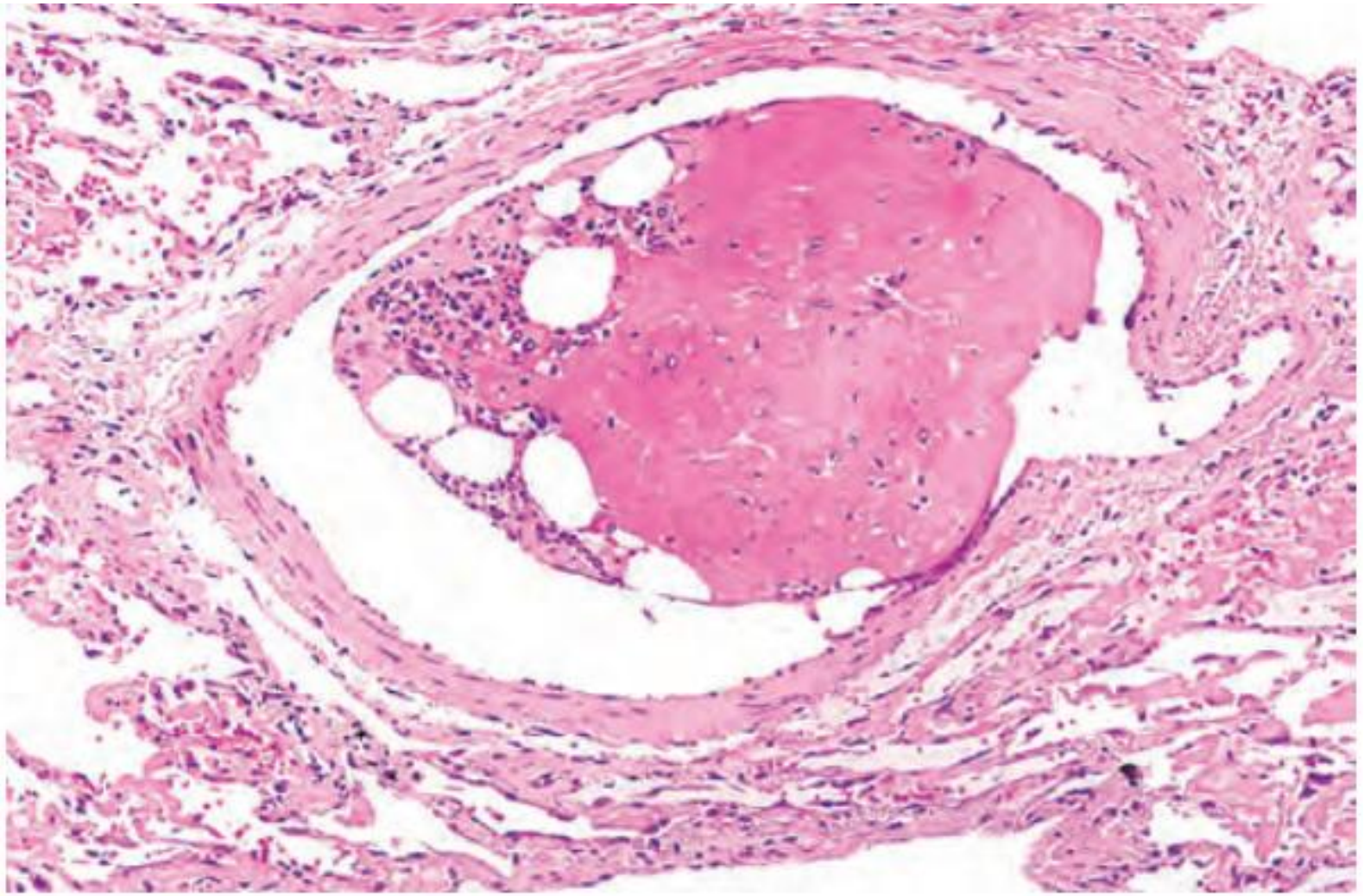
Fat Embolism



Air Embolism

Gas bubbles within the circulation can coalesce to form frothy masses

> 100 mL air volume----produce a clinical effect

300 to 500 mL of air at 100 mL/sec may be fatal.

Communication between the vasculature and outside air e.g.

- neurosurgery in the “sitting position,”
- endovascular and interventional procedures,
- during mechanical ventilation,
- during obstetric or laparoscopic procedures.

Air Embolism Pathogenesis

Mechanical obstruction

Frothy air mass has little effect

Biochemical injury

Microemboli of air trapped in pulmonary capillaries

↓

release of cytokines

↓

induce an intense inflammatory response

↓

Alveoli Injury

Decompression sickness(Specialized form of gas embolism)

- Scuba and deep sea divers, and underwater construction workers

When air is breathed at high pressure (e.g., during a deep sea dive)

Increased amounts of gas (particularly nitrogen) are dissolved in the blood and tissues.

If the diver then ascends (depressurizes) too rapidly

Nitrogen comes out of solution in the tissues and the blood

Decompression sickness (Acute)

- Occurs due to acute obstruction of small blood vessels in the vicinity of joints and skeletal muscles

- **Bends:** Acute pain in joints, ligaments and tendons.
- **Chokes**: Accumulation of bubbles in the lungs resulting in acute respiratory distress.

- Cerebral effects: vertigo, coma and death
- Treated with ‘recompression in a hyperbaric chamber’

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Grecian Bend

Decompression sickness (Chronic)
Caisson disease

- Avascular necrosis of bone- head of femur, tibia, humerus
- Neurological symptoms- paresthesia, paraplegia
- Lung involvement – Haemorrhage & oedema- dyspnea, nonproductive cough and chest pain
- Skin manifestations – Itching, cyanosis, oedema
- Parenchymal cells of liver & pancreas may show fatty vacuoles.

Amniotic Fluid Embolism

- Serious, unpredictable & unpreventable cause of maternal mortality
- The incidence : 2 to 6 in 100,000 deliveries
- Roughly 10% of maternal deaths in the United States
- The mortality rate is up to 80%,
- Permanent neurologic deficit in as many as 85% of survivors

❖Sudden severe dyspnea, cyanosis, and shock, followed by neurologic impairment ranging from headache to seizures and coma, and by DIC

Amniotic Fluid Embolism

1. Biochemical activation of coagulation factors
2. Activation of components of the innate immune system
3. Release of vasoactive substances

❖ Presentation: Acute pulmonary hypertension and right heart failure, which causes hypoxia, left heart failure, pulmonary edema, and diffuse alveolar damage

Diagnosis: Amniotic fluid contents squams, vernix caseosa, lanugo hair, meconium, mucus in pulmonary microcirculation

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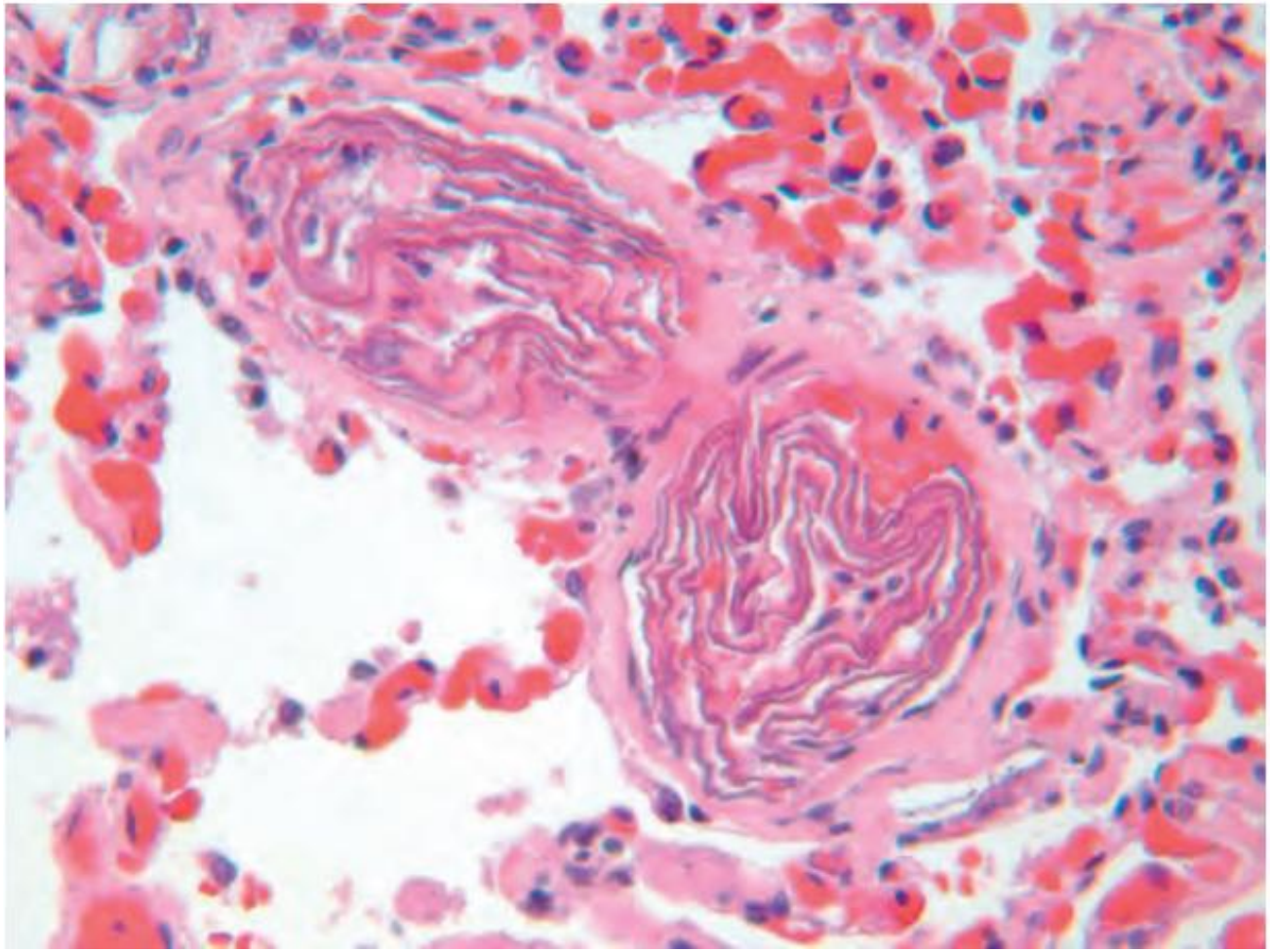
Amniotic Fluid Embolism



Other Embolisms

1. Atheroembolism
2. Tumor embolism
3. Bullet embolism
4. Aeroembolism
   - Seen in those who ascend to high altitudes or air flight in unpressurised cabins
   - They are exposed to sudden decompression from normal atmospheric pressure to low levels

A 28-year-old woman, who is at 33-week gestation, presents to the emergency department with heavy vaginal bleeding. Ultrasound evaluation reveals that she is suffering from abruptio placentae (premature separation of the placenta from the uterus wall). As she is being prepared for delivery, you notice that there is blood seeping from her IV and venipuncture sites and that she has a petechial rash. Concerned, you immediately order several blood tests, which reveal a prolonged PT, prolonged PTT, prolonged bleeding time, prolonged thrombin time, thrombocytopenia, and elevated D-dimer levels. You begin transfusing platelets and fresh frozen plasma in the hope of stabilizing this life-threatening complication.

An 82-year-old woman presents to the emergency department complaining of severe shortness of breath. She tells you that her right calf has been sore as well. On directed history, you discover that she suffered a stroke 6 months ago and has been bedridden ever since. Further evaluation reveals that she is hypoxic and has elevated D-dimer levels. You decide to begin empiric anticoagulant therapy and you order a high-resolution CT scan of the chest with contrast.

4

Ischemia

Circulatory Disturbances- Ischemia

Definition : Inadequate blood supply to a part of organ

Causes :-

Heart: Heart block, Ventricular arrest, Fibrillation

Artery: Thrombosis & embolism, Spasm, Hypothermia,
Arteriosclerosis, Pressure from outside

Vein: Thrombosis & embolism, Ligature, Strangulation, Varicose veins

Capillaries: Frost bite, Sickled red cells, WBCs, Fat globules,
Air bubbles, Pressure from outside

Circulatory Disturbances- Ischemia

Factors determining the extent of damage by ischemia

1] Anatomy of the vasculature in affected area

Lung & Liver- Dual blood supply

Retinal & cerebral- End arteries

Skin, Muscles, Uterus- Abundant anastomosis

2] Nature of the affected tissue: Brain & Myocardium- most vulnerable

3] Rate & degree of obstruction: Sudden obstruction- more severe effect

4) Generalized diseases affecting circulation: Atherosclerosis, Cardiac failure

Circulatory Disturbances- Ischemia

Effects- Ischemia produces effects through

1) Anoxia
2) Deprivation of nourishment
3) Accumulation of waste products of metabolism

Changes at cellular level

Functional disturbance
Atrophy
Degeneration,
Infarction / Sudden death

A 74-year-old man presents to the emergency room, complaining of severe abdominal pain that started acutely 2 hours ago. He also reports that he vomited on his way to the hospital. Upon further questioning, you discover that he has been suffering from episodes of abdominal pain for the last 3 months, which are worse after eating. He had attributed these symptoms to acid reflux but admits that he never had much relief with antacids or proton pump inhibitors. His past medical history is significant for CAD, hypertension, and tobacco use. On physical examination, he is clearly uncomfortable; however, his abdomen is soft and he has only minimal tenderness to palpation. You begin to wonder if his symptoms may be related to vascular disease and you order a CT scan of the abdomen with contrast to confirm your suspicions.

Circulatory Disturbances- Infarction

Definition :-

An infarct is an area of necrosis produced by deprivation of blood supply, the underlying pathological mechanism is known as Infarction

Etiology :-

1) Blockage of an artery
2) Blockage of artery & vein
3) Blockage of a vein

Circulatory Disturbances- Infarction

Sequence of events

1. Localized hyperemia
2. Affected part is swollen due to oedema
3. Necrosis in 12- 48 hrs
4. Progressive proteolysis of necrotic tissue & hemolysis of RBCs
5. Acute inflammatory reaction in the surrounding tissue
6. Pigment liberated by haemolysis are deposited in infarct
7. Ingrowth of granulation tissue

Circulatory Disturbances- Infarction Types

According to their colors

RED :- Hemorrhagic , organs with dual blood supply, organs with loose texture, infarcts produced by obstruction of artery & vein

PALE :- Grayish yellow, opaque, zone of hyperemia around them

According to their age

Recent/ Fresh- reddish, elevated above the surface

Old/ Healed- pale shrunken

According to the presence / absence of Infection

Bland

Septic

Splenic Infarction (Coagulative Necrosis)

Healing of Infarct

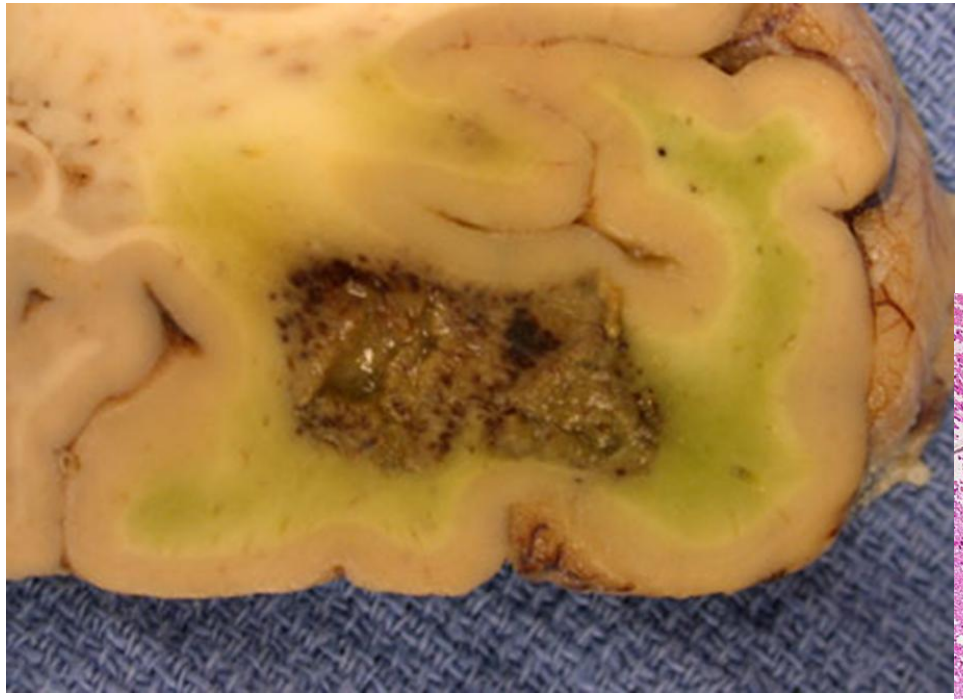
- Removal of necrosed tissue

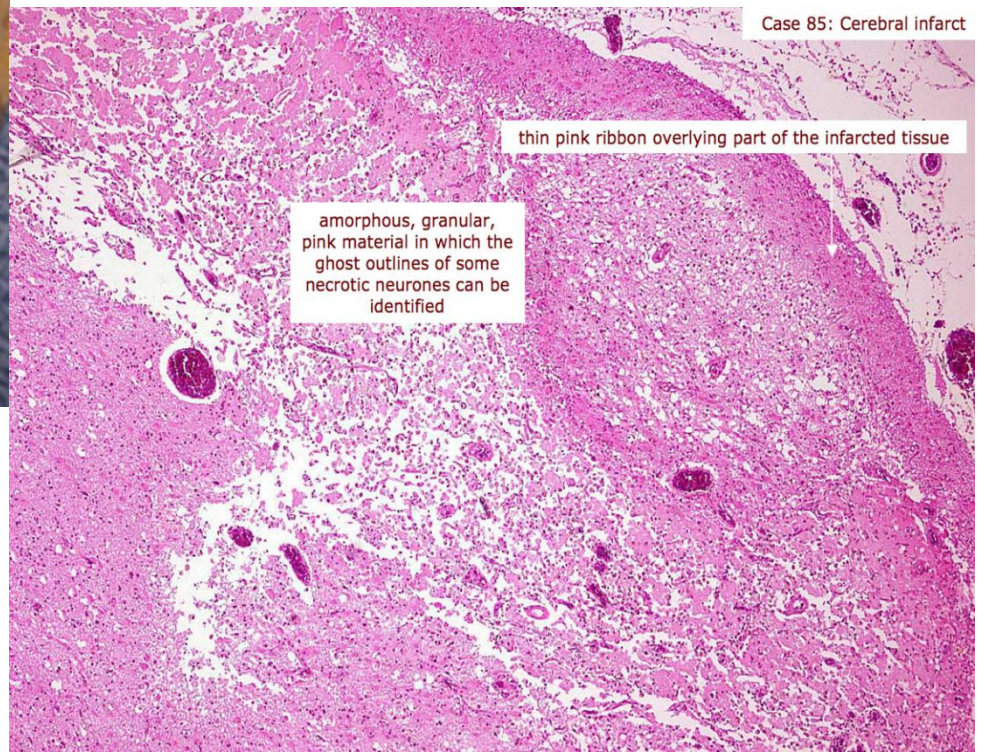
- Regeneration of cells

- Organization of the infarct by granulation tissue

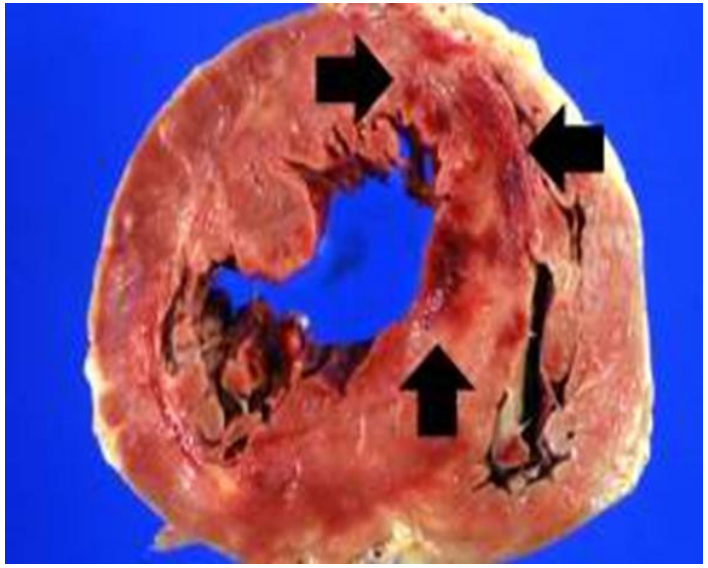
- Fibrosis

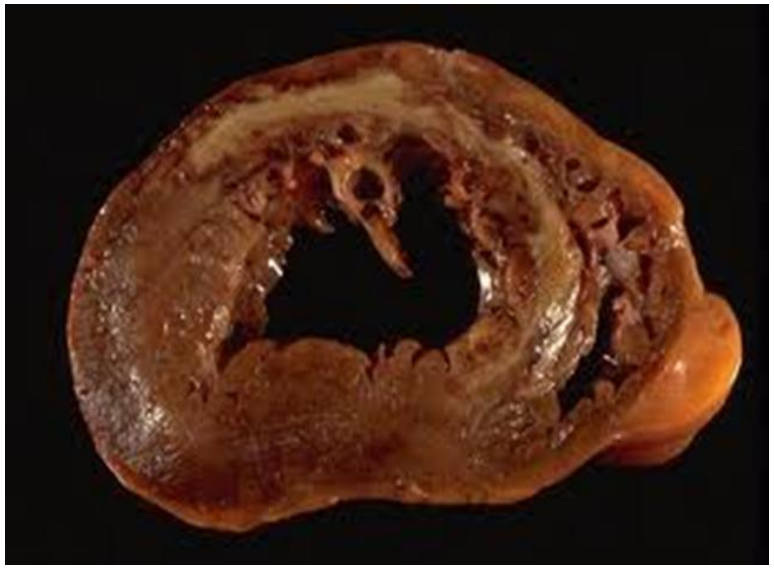
Infarcts in Various organs- Brain

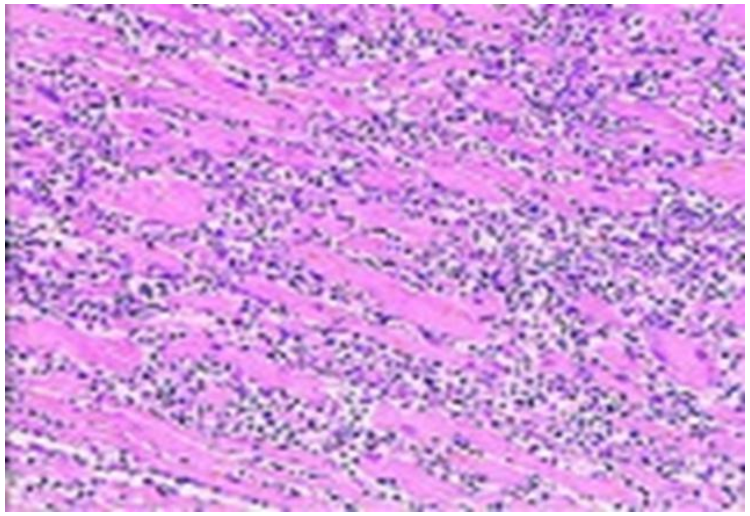


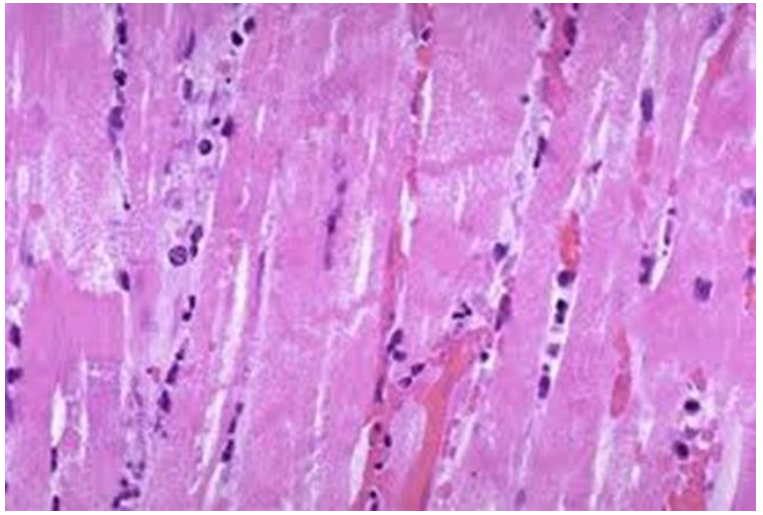


Infarcts in Various organs- Heart

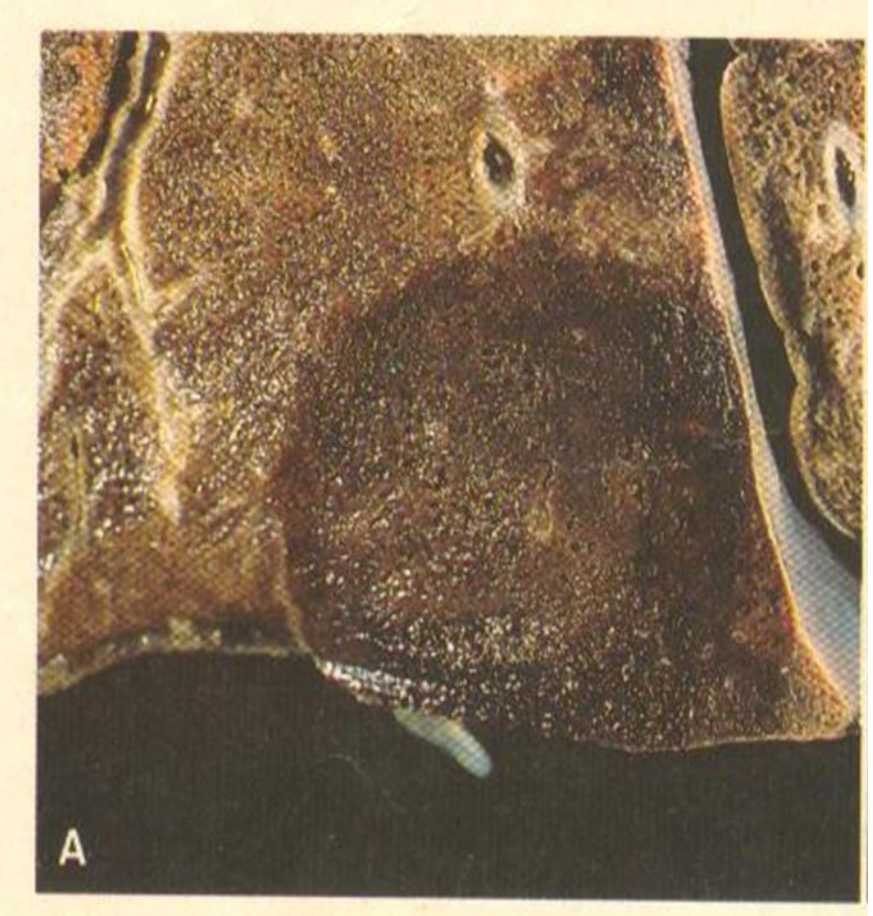


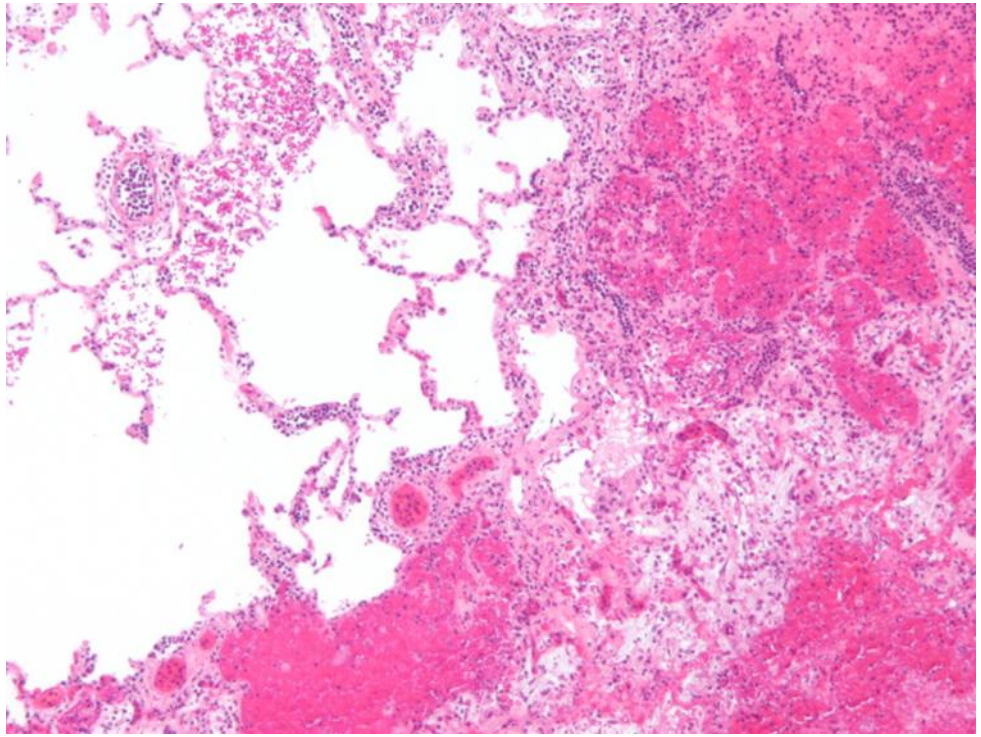




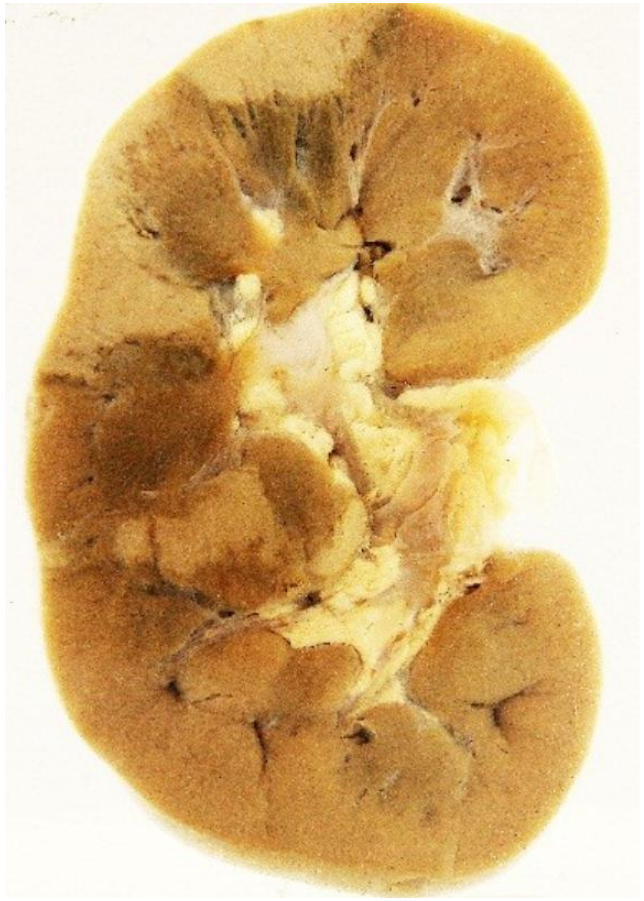


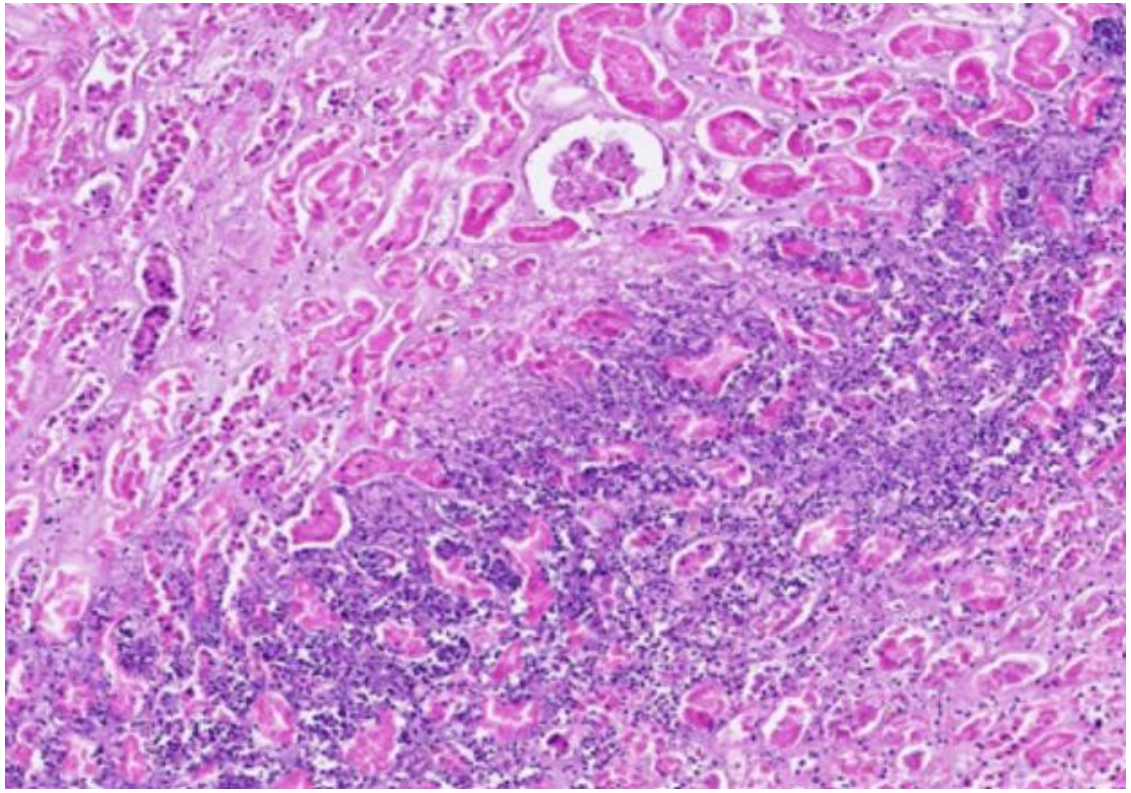
Infarcts in Various organs- Lungs



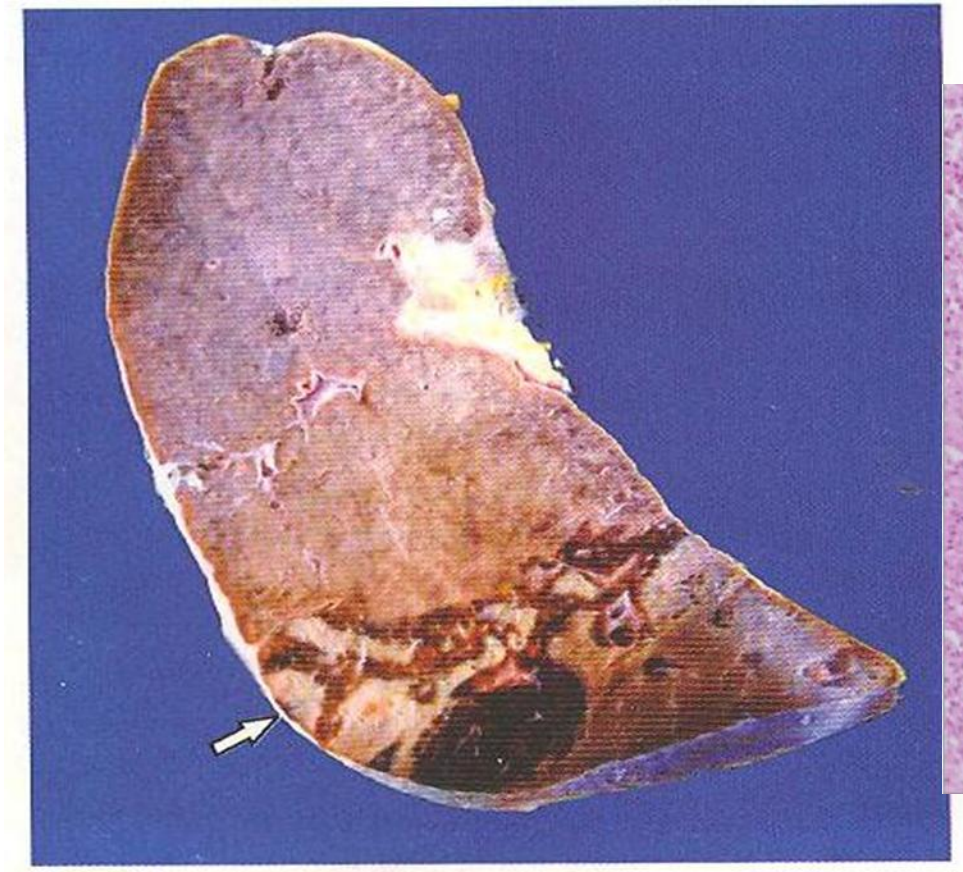


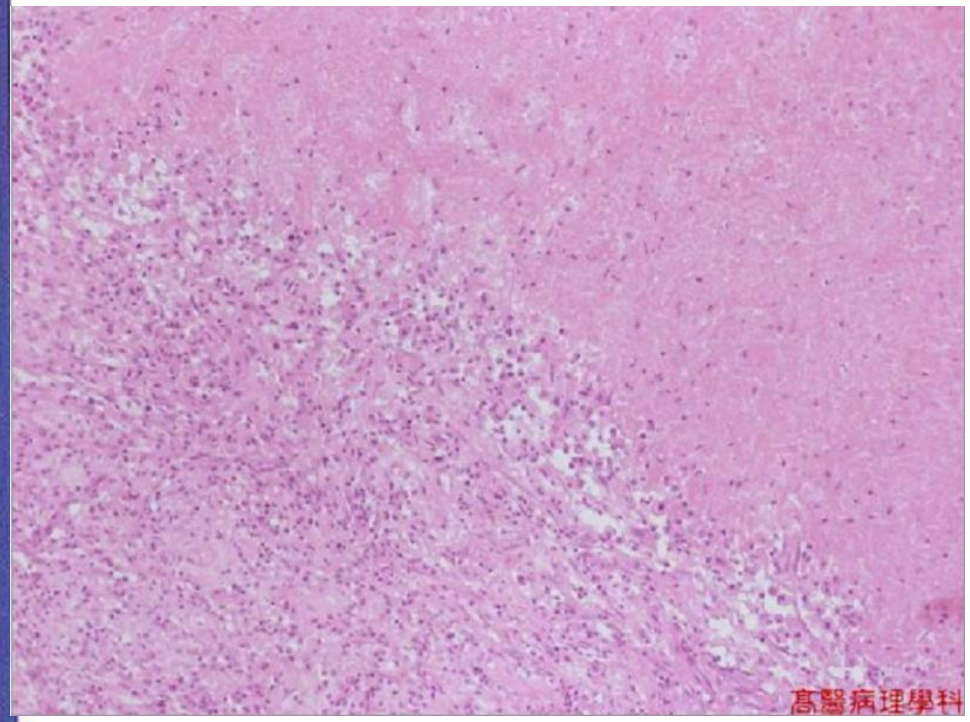
Infarcts in Various organs- Kidney





Infarcts in Various organs- Spleen





Infarcts in Various organs

Organ	Cause	Features
Intestine	Obstruction of superior mesenteric artery & vein,	Haemorrhagic
Lung	Pulmonary Embolism	Wedge shaped, Haemorrhagic coagulative necrosis
Kidney	Thromboembolism	Wedge shaped, Pale, coagulative necrosis
Spleen	Thromboembolism	Wedge shaped, Pale, coagulative necrosis
Brain	Thromboembolism	Liquefactive necrosis
Liver	Rare hepatic vein thrombosis	Dual blood supply

Clinical significance of Infarction

Irreparable damage to organ

- Heart & Brain: Permanent loss of function/ sudden death

Thrombosis, embolism & infarction are inter-related

Thrombosis & embolism give rise to infarction & an infarct of the heart can produce mural thrombosis which might lead to embolism


thank
you