



Hemodynamic Disorders, Thromboembolic Disease and Shock (Part 1)

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*250 years of EXCELLENCE
in medical education,
research & innovation
and healthcare*

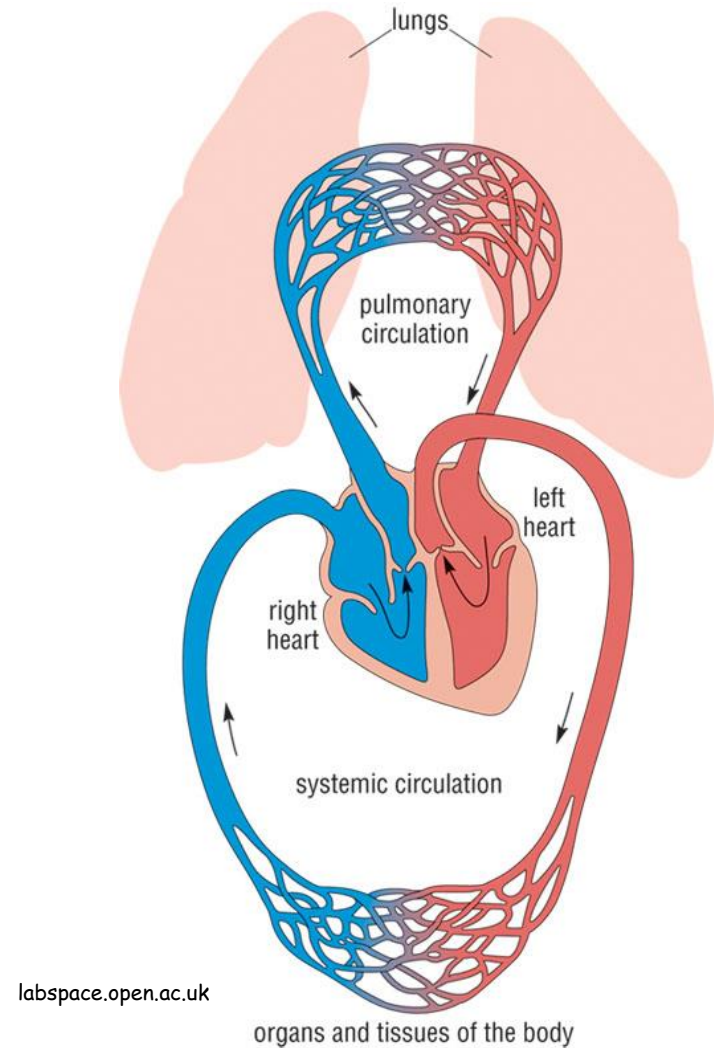
13th September 2021

Normal fluid homeostasis

- Vessel wall integrity
- Intravascular pressure and osmolarity in physiologic ranges
- Maintaining blood as a liquid

Protagonists

- Heart
- Blood vessels and lymphatic vessels
- Blood



Hemodynamic disorders

- Hyperemia (active and passive)
- Edema
- Hemorrhage
- Thrombosis
- Embolism
- Infarction
- Shock

HYPERAEMIA

HYPEREMIA I.

- **Definition:** locally increased blood volume
- **Forms:**
 - active,
 - passive/congestion



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- **ACTIVE HYPEREMIA**
 - Active dilation of arteriae, arterioles, capillaries
 - erythema
 - Forms: physiological, pathologic (inflammation, fever, chemical and physical injury)



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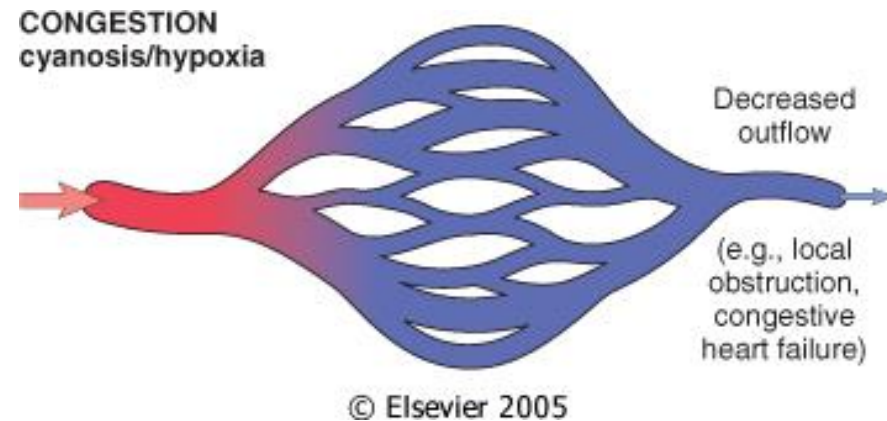
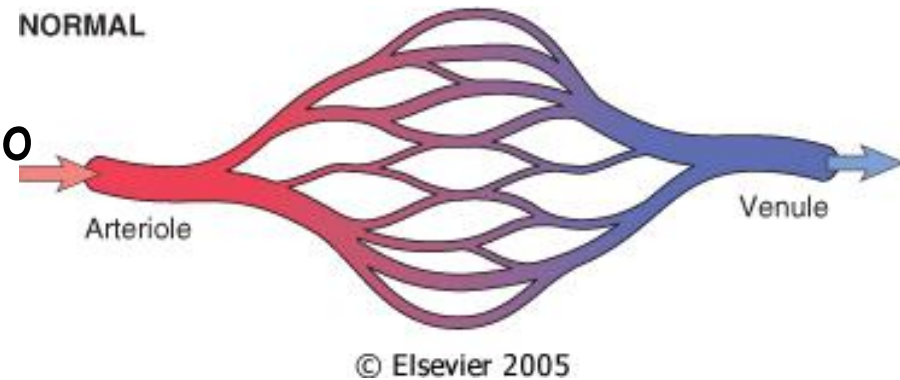
HYPEREMIA II.

- **PASSIVE HYPEREMIA**
(congestion)

- dilation of venous side due to decreased outflow
- cyanosis, hypoxia

- **Causes:**

- **Local:** thrombosis, obstruction
 - Deep venous thrombosis-legs
 - Pylethrombosis- portal congestion
 - V.cava sup. syndrome
- **Systemic:** congestive heart failure



Consequences of systemic chronic congestion

Left sided heart failure

Right sided heart failure

Lungs

heavy, firm, heart failure cells on microscopy (Induratio brunea pulmonum)

Liver

- nutmeg liver (hepar moschatum)
- centrilobular necrosis
- cardiac fibrosis (cirrhosis-misnomer!)

Kidneys

- stellate veins accentuated
- cortex widened
- sharp separation of medulla and cortex

Spleen

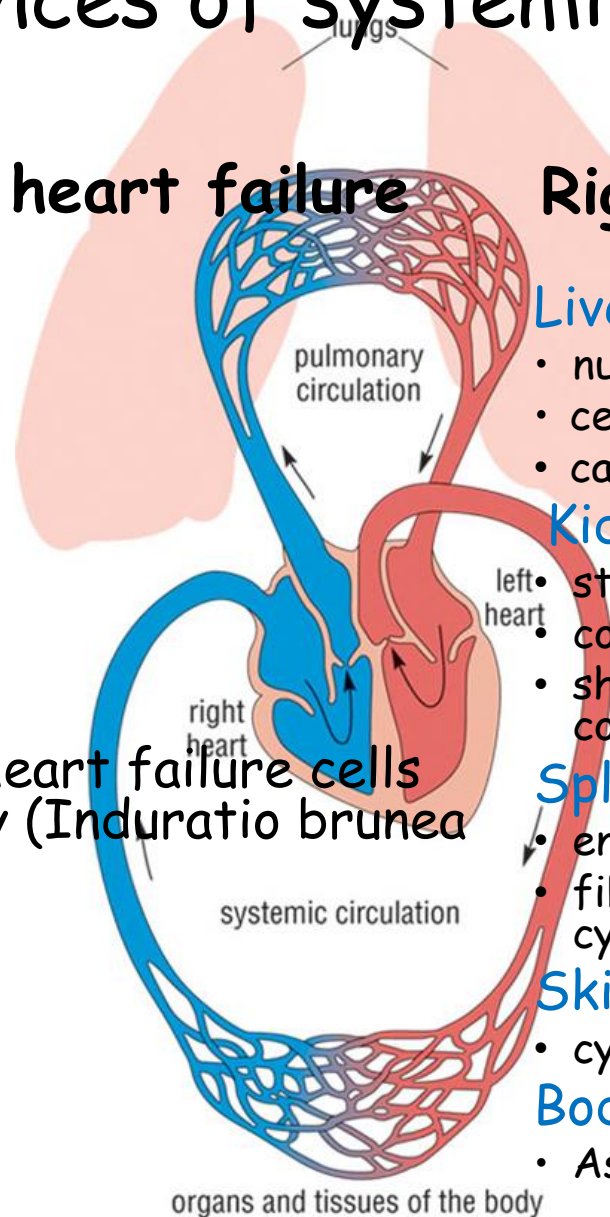
- enlarged, livid
- fibrosis with time (Induratio cyanotica lienis)

Skin

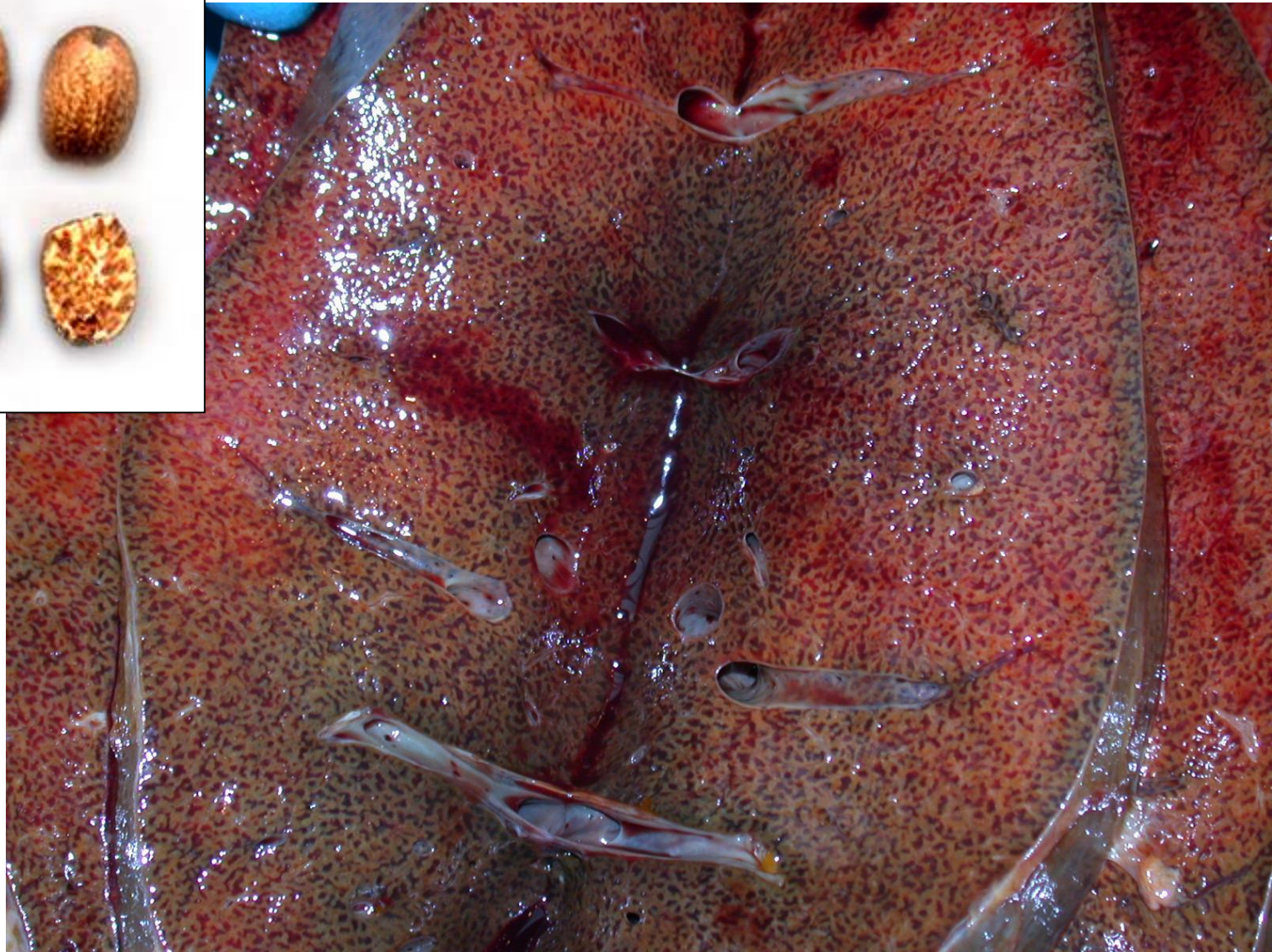
- cyanosis, anasarca

Body cavities

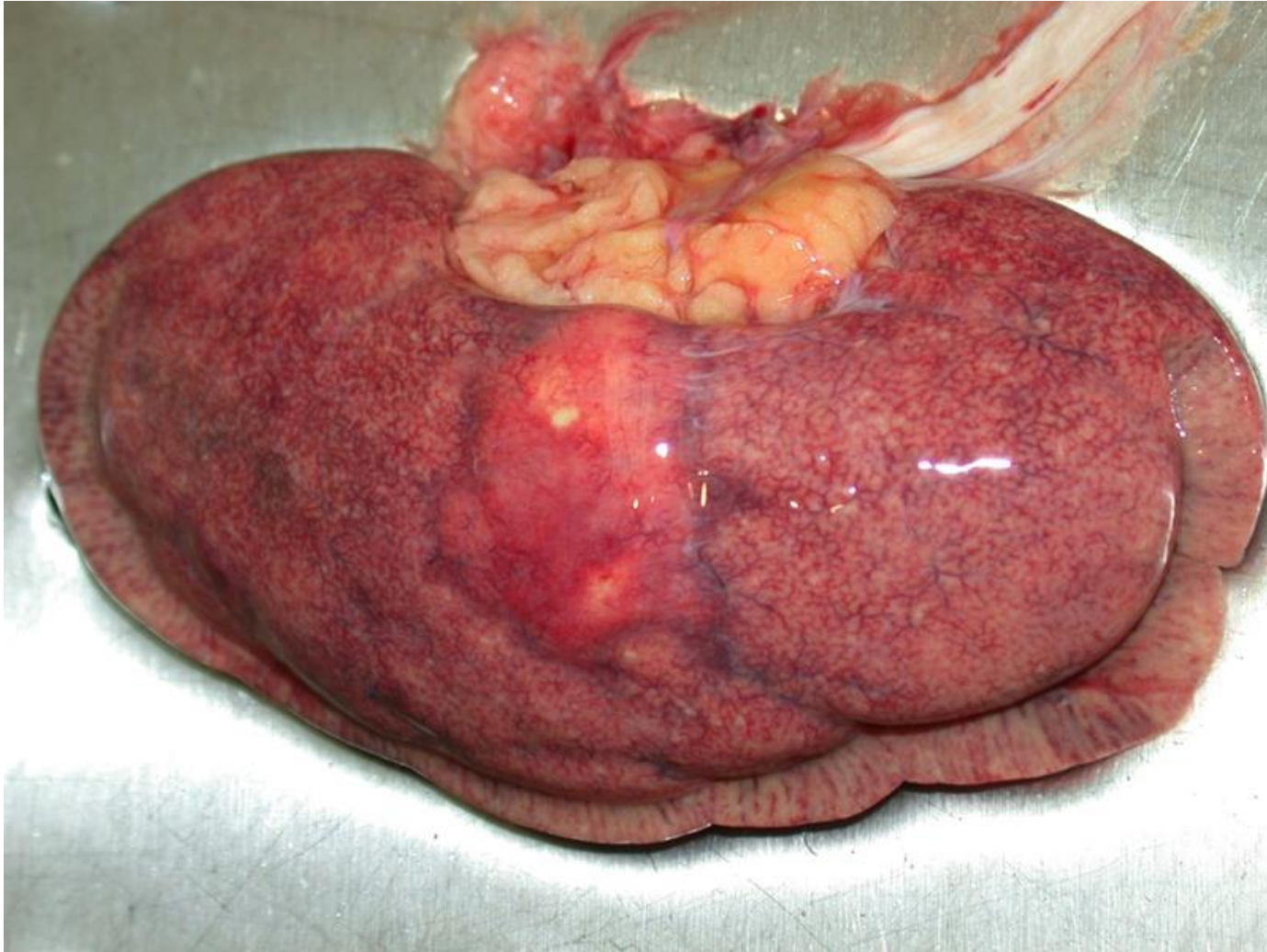
- Ascites, hydrothorax



Systemic congestion-Nutmeg liver



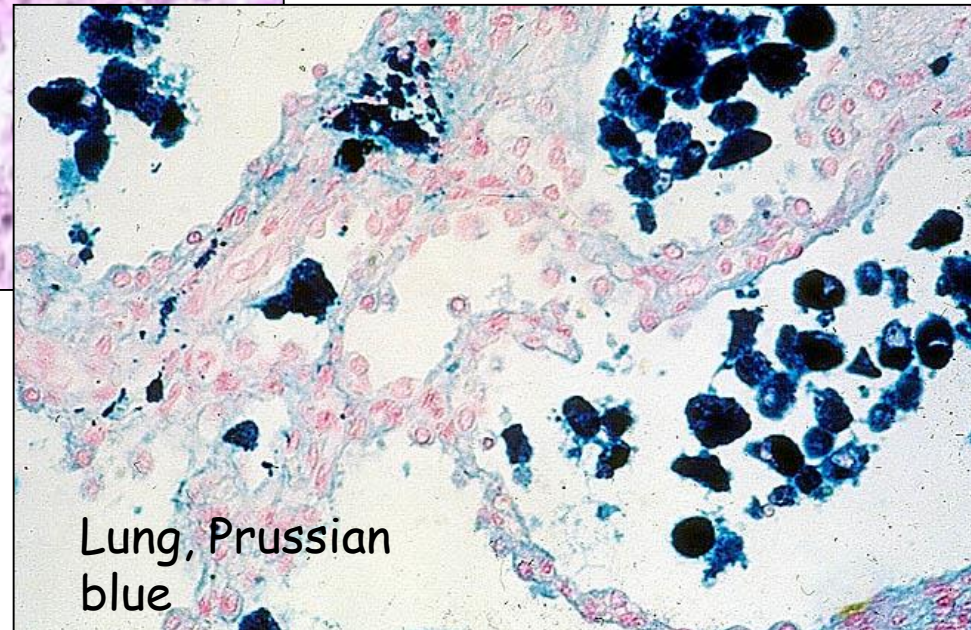
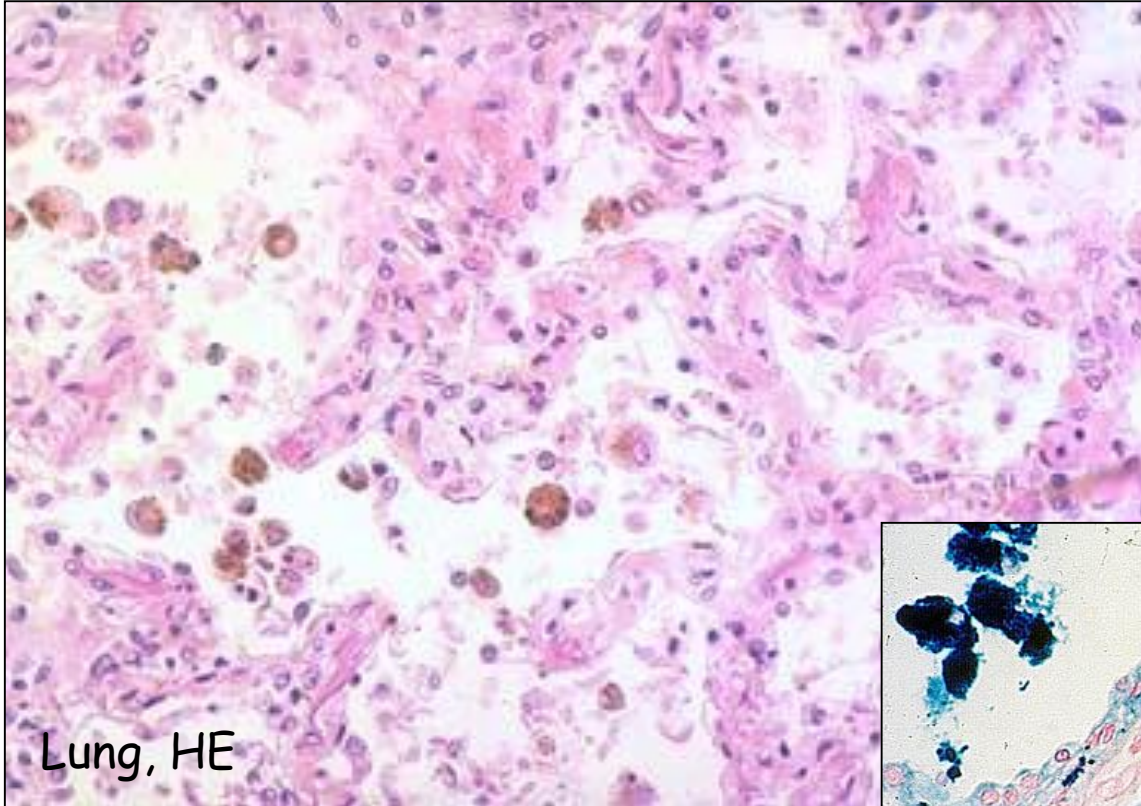
Systemic congestion (+?) - kidney



Lung, chronic passive hyperemia/ congestion



Hemosiderin in heart failure cells



Local chronic congestion

- May occur in every organ
 - E.g. vena cava superior syndrome,
 - Budd-Chiari sy (hepatic vein thrombosis),
 - extremities etc.

Local congestion



EDEMA

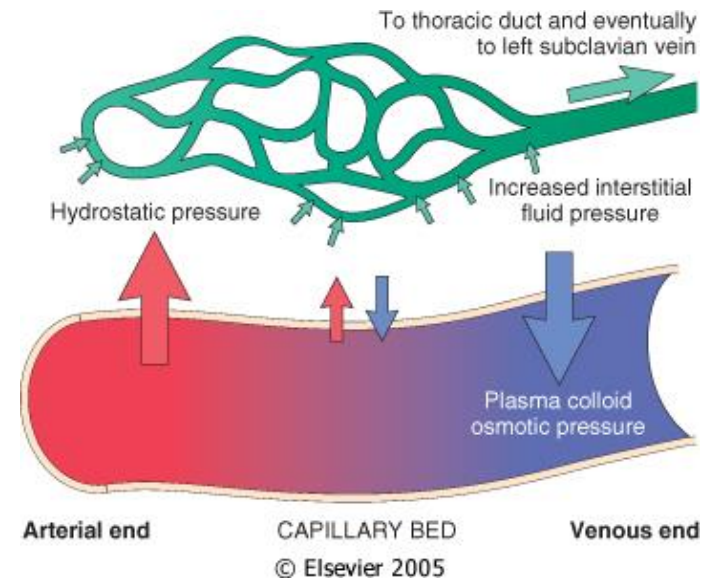
EDEMA

- Definition: increased fluid content in the interstitium (transudate or exudate)
- Causes:
 - **INCREASED INTRAVASCULAR HYDROSTATIC PRESSURE**
(arteriolar dilation or impaired venous return)
(**LOCAL**- eg. Deep venous thrombosis of the legs,
SYSTEMIC- eg. Congestive heart failure)
 - **DECREASED PLASMA COLLOID OSMOTIC PRESSURE**
(eg. nephrosis sy, cirrhosis, protein malnutrition)
 - **LYMPHATIC OBSTRUCTION**
(lymphedema, elephantiasis)
 - **SALT AND WATER RETENTION**
(GN, Acute renal failure)
 - **INFLAMMATION (EXUDATE)**

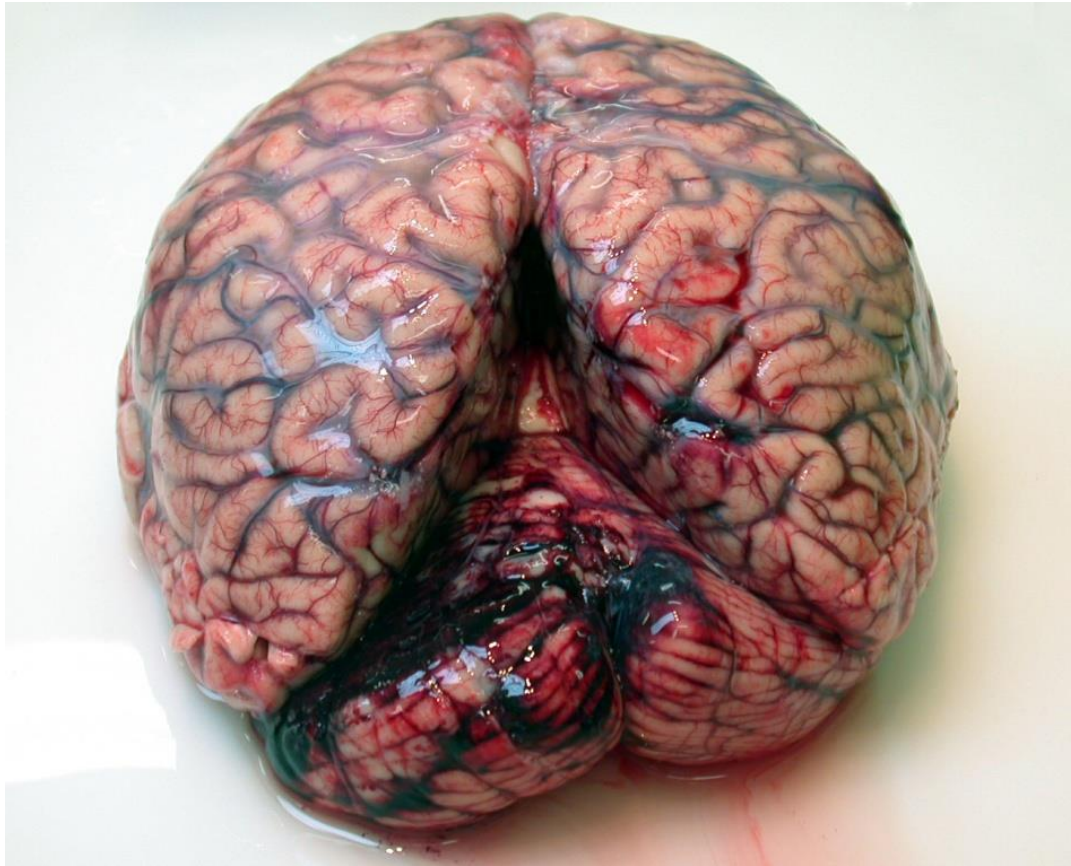
Normal fluid homeostasis:

60% of body weight is water

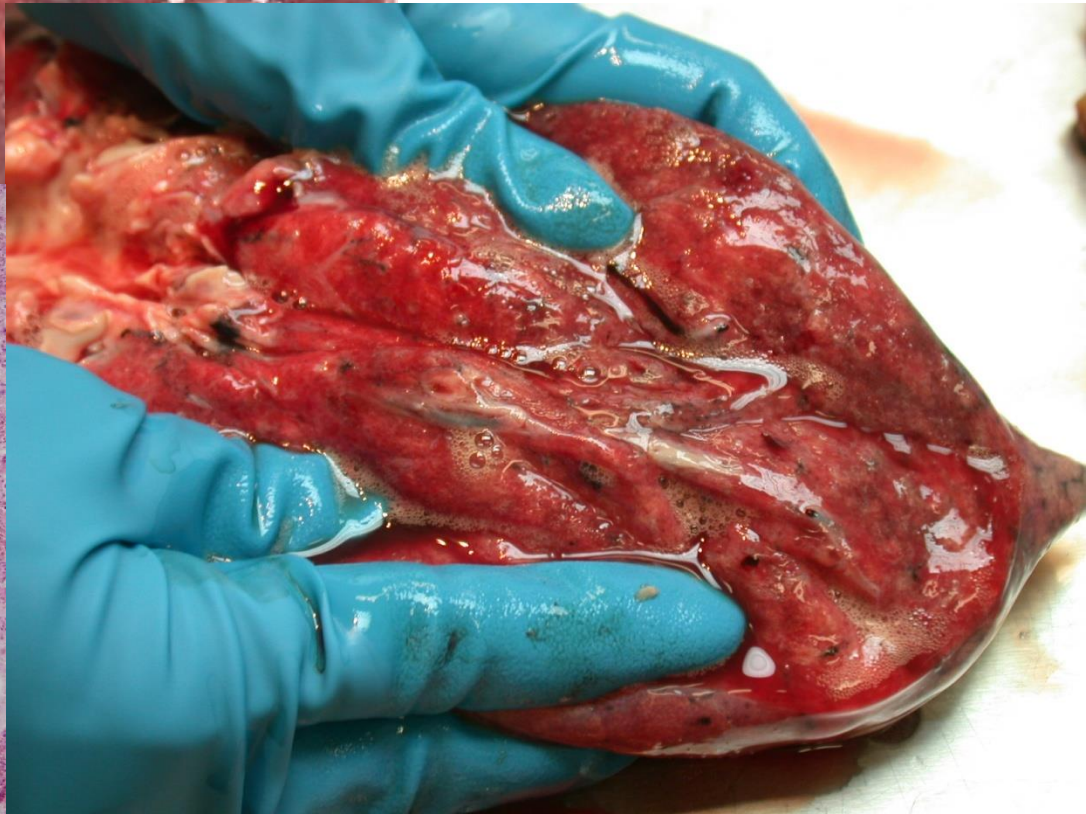
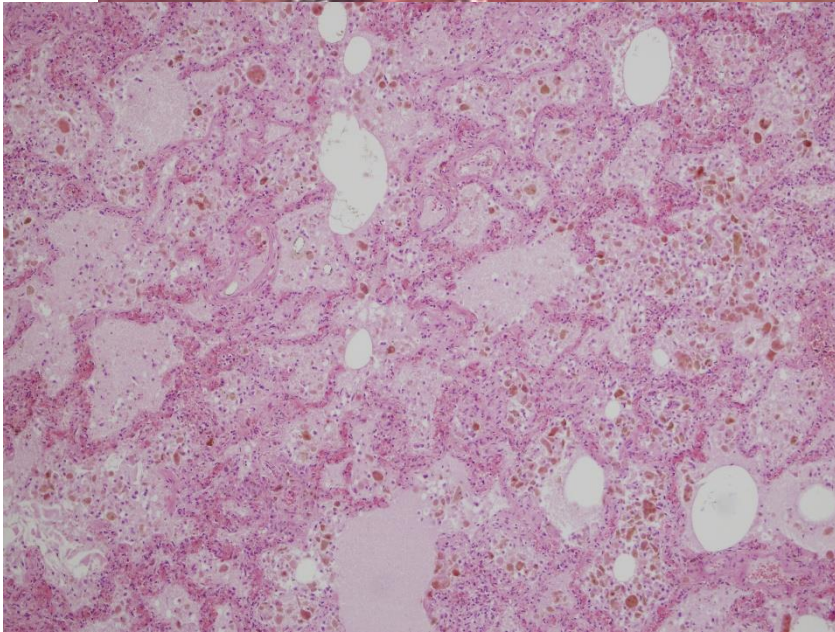
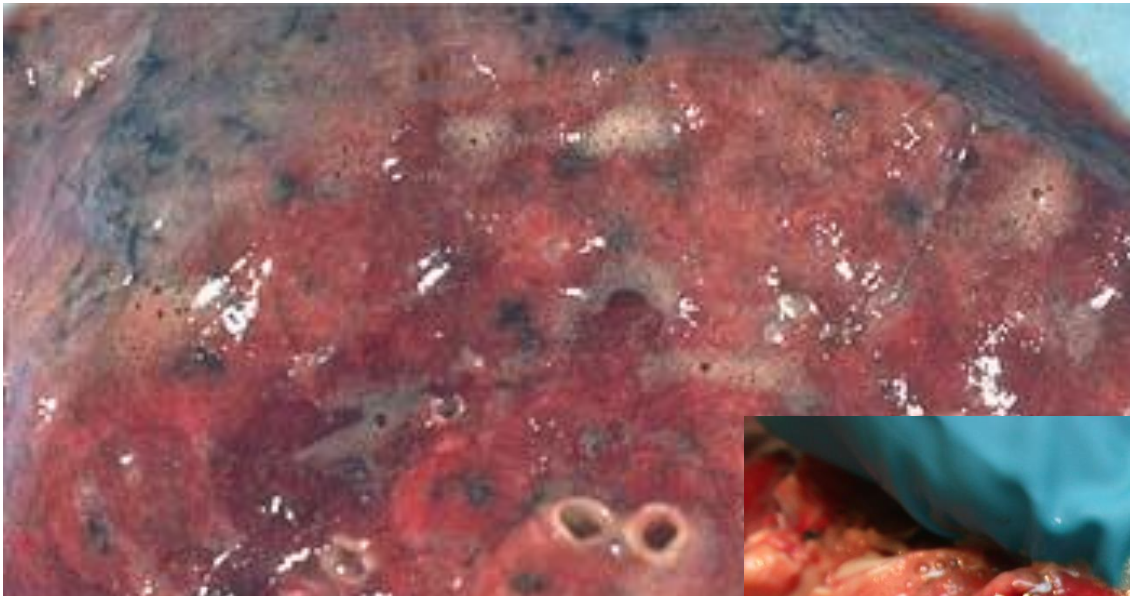
2/3 intracellular, 5% blood, remainder within interstitium

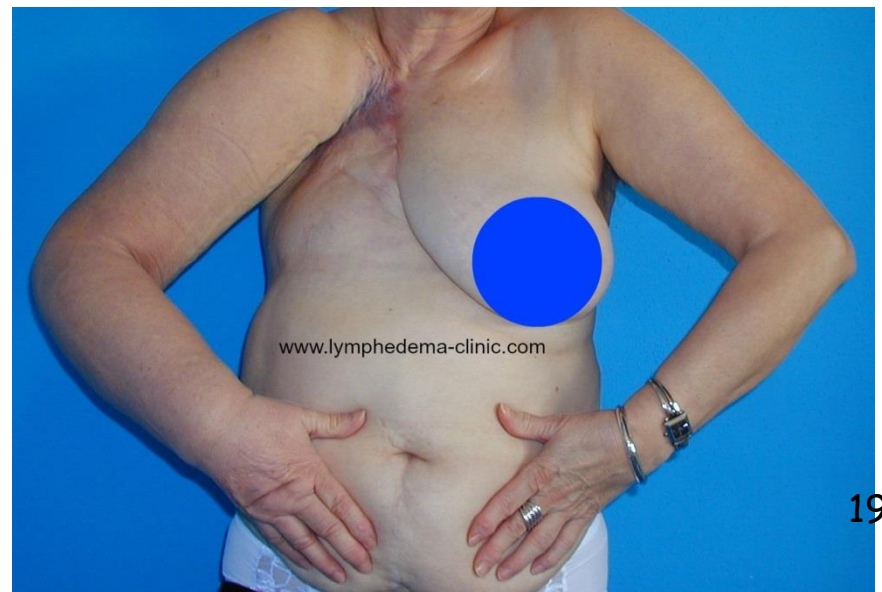


Cerebral edema- tonsillar herniation



Pulmonary edema





HEMORRHAGE

Hemostasis-protagonists

- Vascular wall (endothelium)
- Platelets
- Coagulation cascade

Normal hemostasis—sequence of events

- **Arteriolar vasoconstriction** (major regulator: endothelin)
- Platelet adherence, activation and aggregation  **Primary hemostasis** (major regulator: ECM)
- Fibrin meshwork creation, additional platelet recruitment  **Secondary hemostasis** (major regulator: Tissue factor)

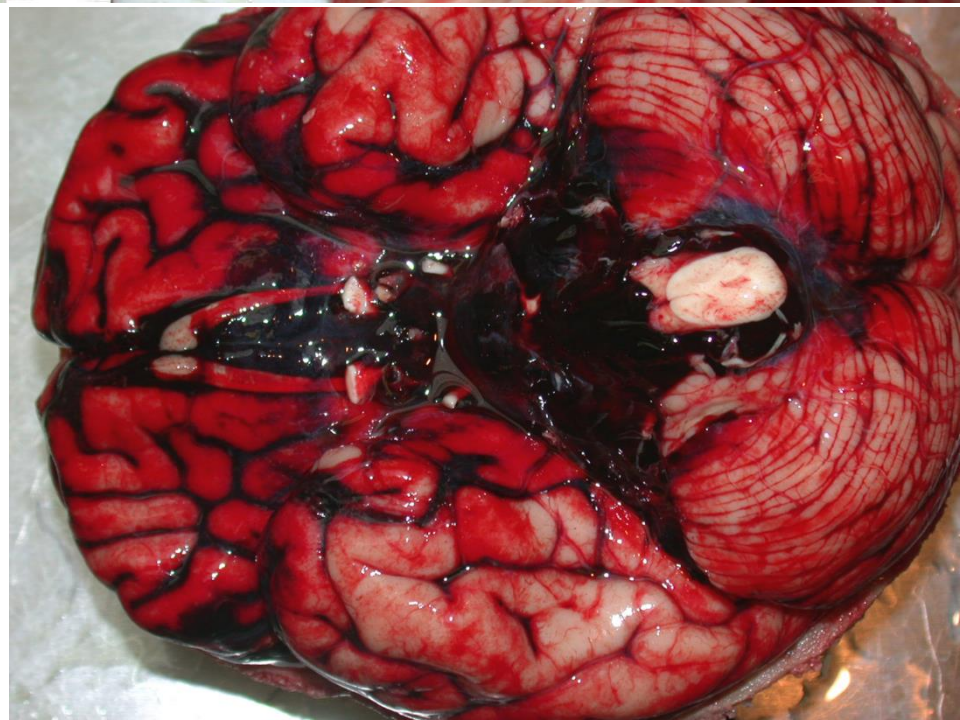
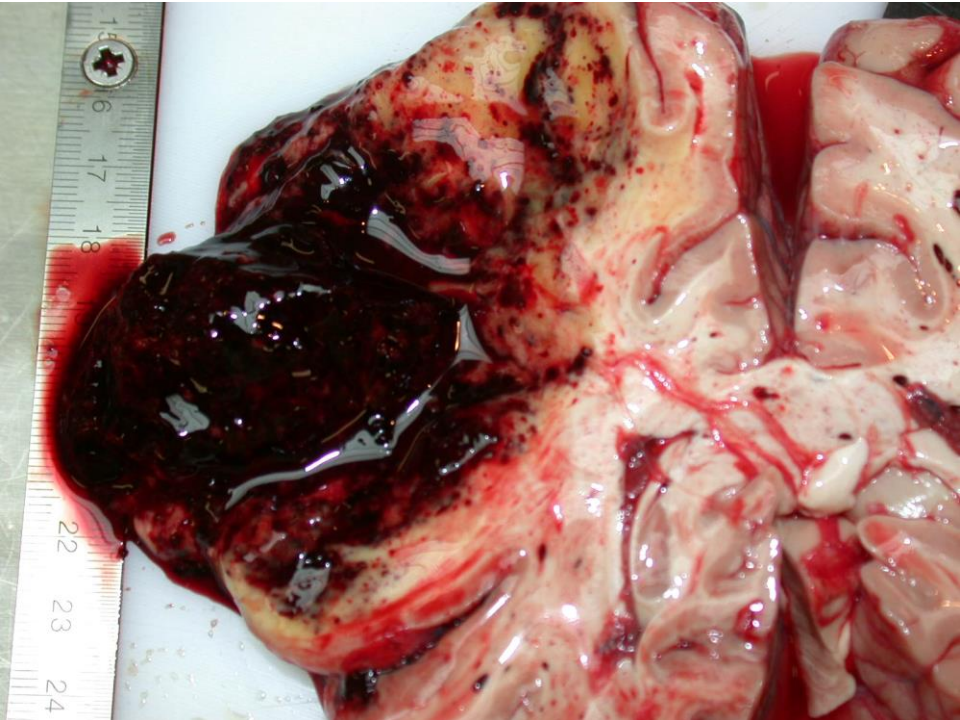
Hemorrhage

- **Definition:** Extravasation of blood
- **Pathogenesis:**
 - Rupture of vessel wall (**haemorrhagia per rhexim**)
 - Erosion of vessel wall (**haemorrhagia per arrosionem**)
 - Vascular wall disturbances and other hemorrhagic diatheses (**haemorrhagia per diapedesim**)
 - **Vessel wall abnormalities:** due to - hypoxia, infections, drugs, impaired collagen synthesis, Henoch-Schönlein purpura, Hereditary hemorrhagic teleangiectasia etc.
 - **Other hemorrhagic diatheses**
 - **Thrombocytopenia** (low platelet count)
 - Decreased platelet production
Bone marrow diseases, bone marrow infiltration, drug induced (Heparin-induced thrombocytopenia), infections (HIV associated!) etc.
 - Decreased platelet survival
 - ⊗ Immune thrombocytopenic purpura (ITP, autoimmune)
 - ⊗ Thrombotic microangiopathies (TTP: thrombotic thrombocytopenic purpura, HUS: Hemolytic- uremic syndrome)
 - **Thrombasthenia (defective platelet function):** primary, secondary (aspirin!!!)
 - **Abnormalities in clotting factors**
 - ⊗ Primary, or Secondary (acquired - eg. In hepatic diseases!)
 - ⊗ Von Willebrand disease
 - ⊗ Hemophilia A (factor VIII deficiency)
 - ⊗ Hemophilia B (Factor IX deficiency- Christmas disease)
 - ⊗ Hemophilia C (Factor XI deficiency)
 - Disseminated intravascular coagulation (DIC, consumption coagulopathy)**
 - » Causes: obstetric complications, infections, neoplasms, excessive tissue injury
 - » Hemorrhage and thrombosis

Categories

- By extent
 - Exsanguination, hematoma, suffusion, ecchymosis, purpura, petechia
- By localization
 - eg: hemothorax, hemopericardium, hemascos, hemarthrosis, epistaxis, hematemesis, melena, hematochesia...

By pathomechanism...



Haemorrhagia per rhexim (by rupture)- cerebral



Haemorrhagia per arrosionem
(by erosion)
(hypopharynx tumor)



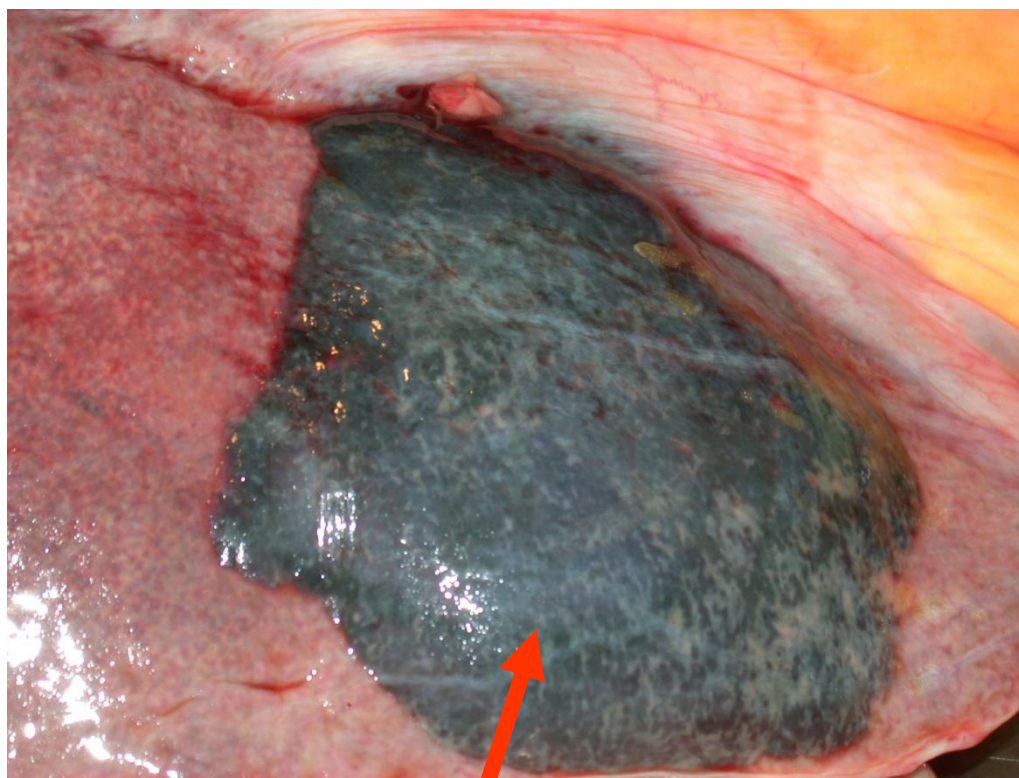
Petechiae- pericardium
(thrombocytopenia)



Petechiae- fat embolism due to trauma

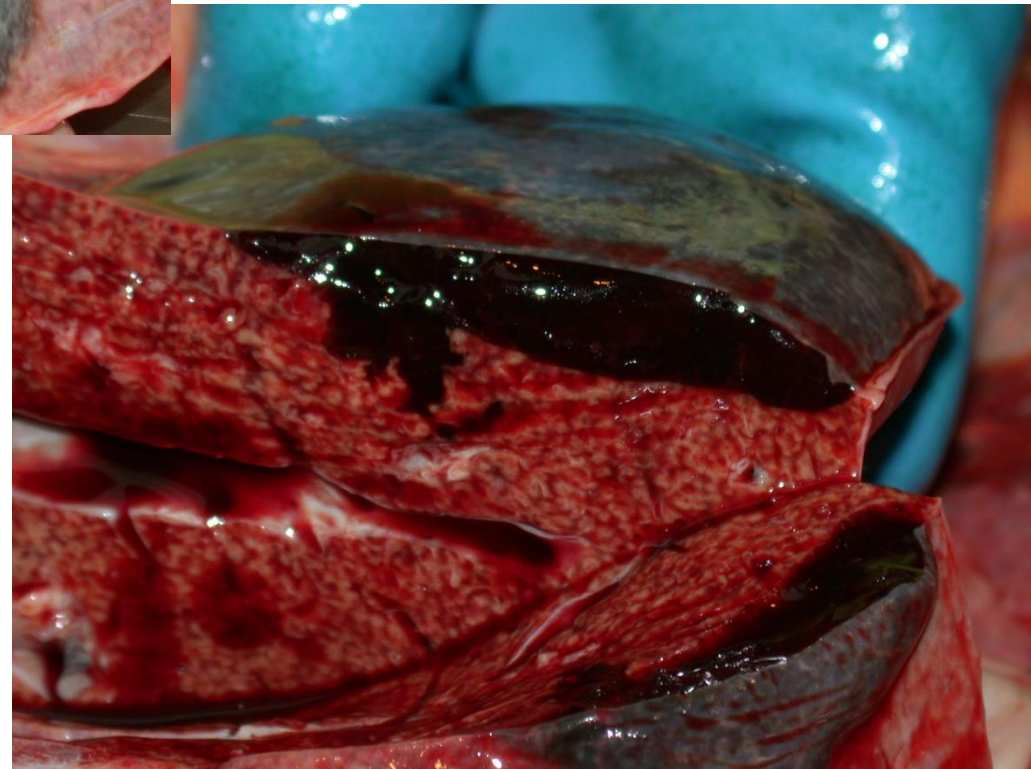
**Haemorrhagia per diapedesim
(by diffusion)**

Categories by extent...



**Subcapsular
hematoma -
liver**

hematoma

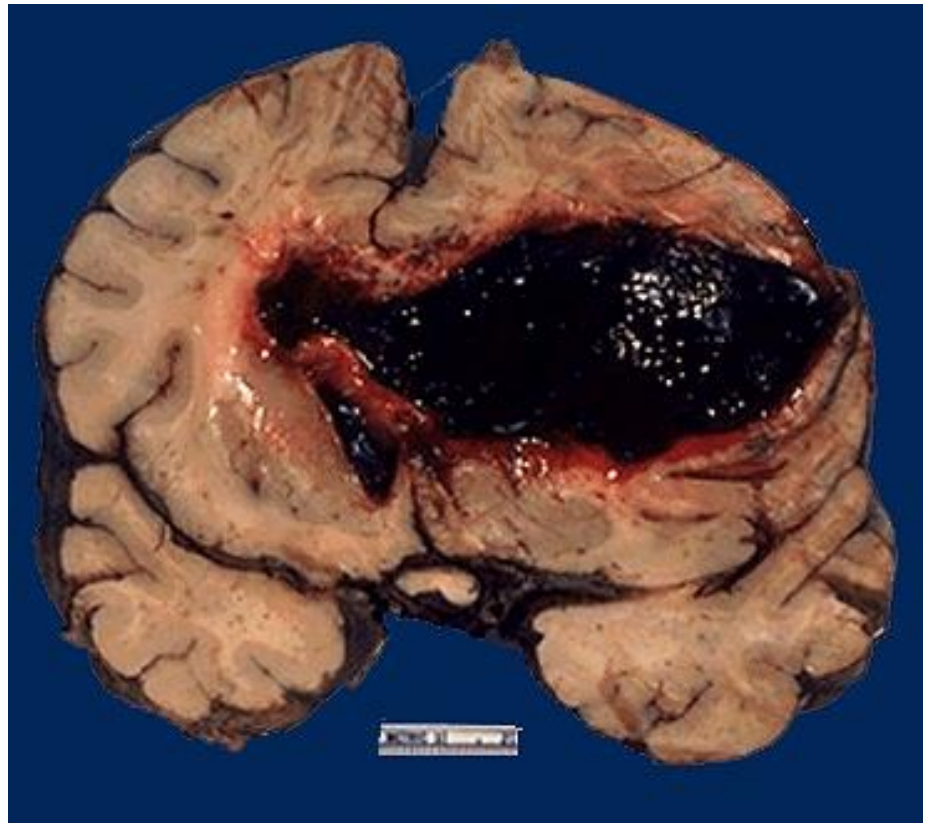


Suffusion, hematoma

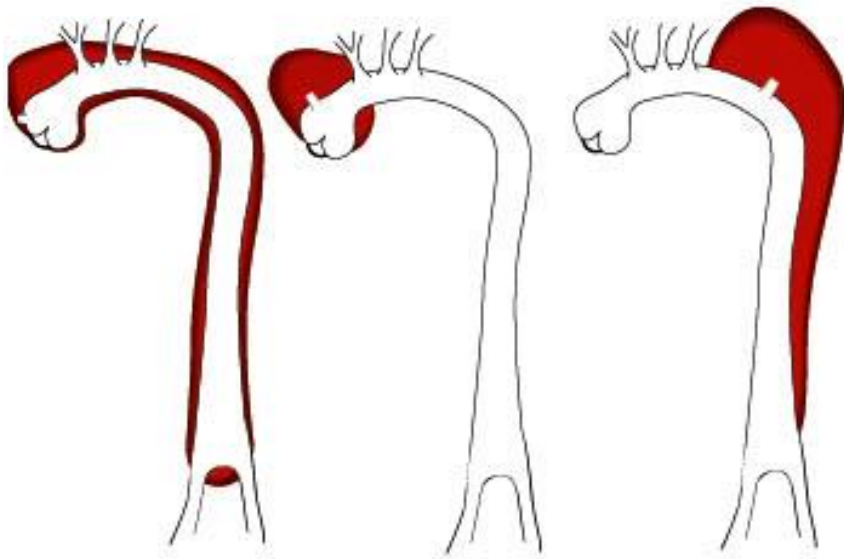




Thalamus -
hemorrhage-
hypertension



Apoplexia cerebri-
hypertension



Hemopericardium- due to aortic dissection 33

THROMBOSIS

THROMBOSIS

- **Definition:** coagulation of blood within vessels or heart chambers
- **Pathogenesis (Virchow's triad)**
 1. **Endothelial injury** (inflammation, hypertension, atherosclerosis etc.)
 2. **Stasis or turbulence** of blood flow
 3. **Hypercoagulability**
 - **Primary** (Genetic predisposition) (e.g. Leyden-mutation in factor V. (resistant to cleavage by protein C)
 - **Secondary (Acquired):** smoking, obesity, drugs, pregnancy, malignant tumors (e.g. pancreas cc- Trousseau phenomenon), antiphospholipid antibody syndrome

Classification by localization I.

Arterial thrombosis

Pathogenesis: endothelial injury, turbulent blood flow (due to atherosclerosis, vasculitis)

Complications:

- **Ischemia** (non occlusive thrombus)
- **Infarction** (occlusive thrombus)

a.coron.-AMI, angina pectoris

Cerebral arteries- TIA, stroke, status lacunaris

a. mesenterica sup. or inf.- bowel infarction

Classification by localization II.

Venous thrombosis (phlebothrombosis)

Pathogenesis: stasis (varicositas, immobilization), thrombophlebitis

Localization: 90% legs

Periprostatic, parametrial plexus, dura sinuses, v. portae, vv.hepaticae, Trousseau phenomenon: migratory thrombophlebitis

Complications: Ulcus cruris

Postthrombotic sy

Vena cava superior sy

Vena cava inferior sy- congestion in lower extremities, pelvis

Classification by localization III.

Thrombosis in heart chambers (like arterial thrombosis)

Pathogenesis: endothelial injury (eg. AMI), turbulent blood flow (dilatation, AMI)

Complications: atrial, ventricular, valvular (endocarditis maranthica) thrombi may be the source of **embolism**

Fibrin (Hyalin) thrombi

- In arterioles, capillaries, venules (disordered microcirculation)
- Composed of platelets, fibrin
- DIC

Secondary hypercoagulable states

High risk for thrombosis

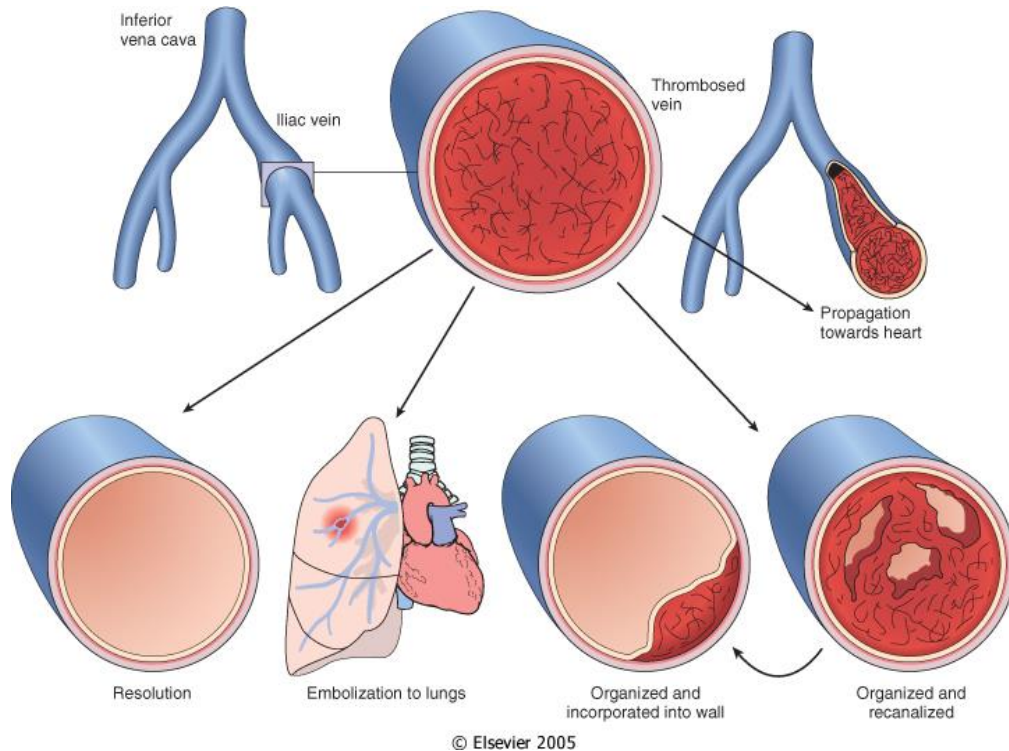
- Prolonged bed rest or immobilization
- Myocardial infarction
- Atrial fibrillation
- Tissue damage
 - (including surgery fractures, burns etc)
- Prosthetic cardiac valves
- Disseminated intravascular coagulation
- Heparin-induced thrombocytopenia
 - HIT (unfractionated heparin)
- Cancer
- Antiphospholipid antibody syndrome
 - („lupus anticoag.sy“)

Secondary hypercoagulable states

Lower risk for thrombosis

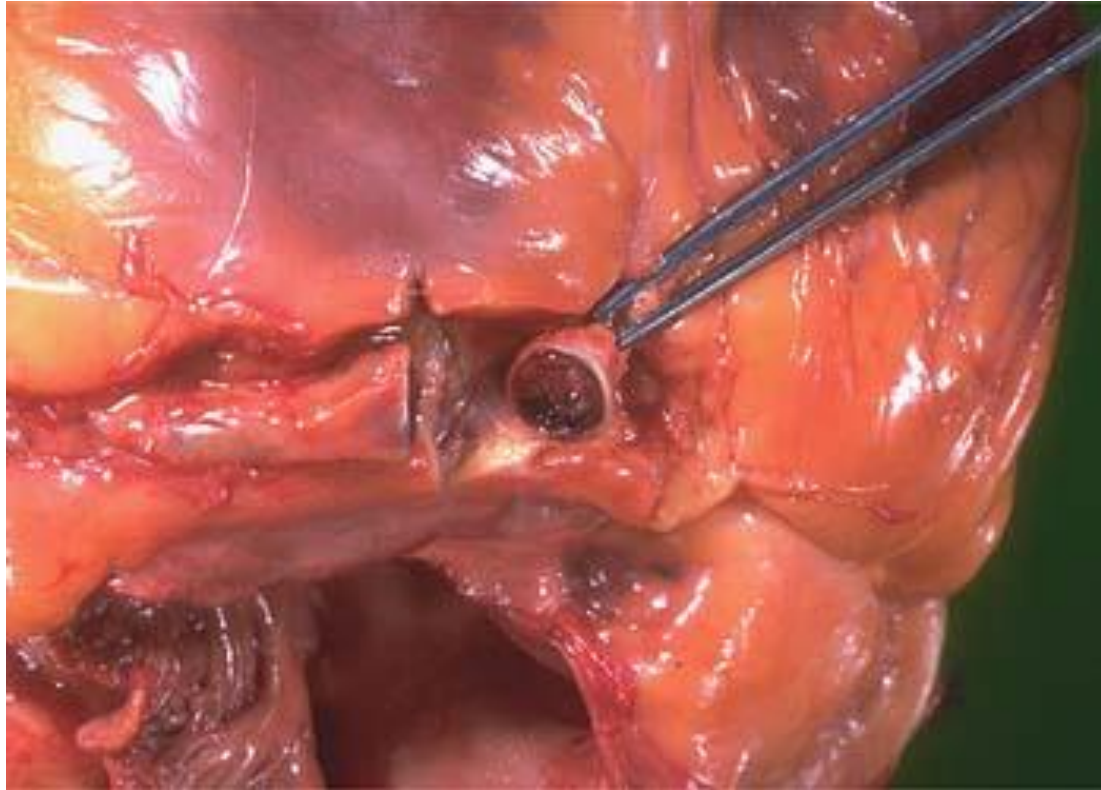
- Cardiomyopathy
- Nephrotic syndrome
- Hyperestrogenic states
(Pregnancy, postpartum)
- Oral contraceptives
- Hyperlipidaemia
- Sickle cell anaemia
- Smoking

FATE OF THROMBI



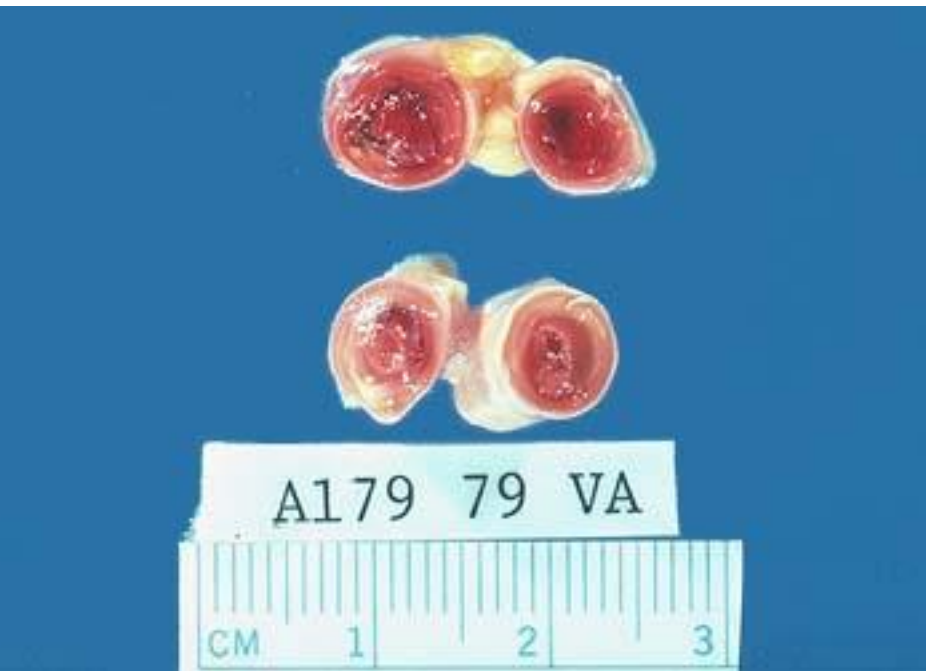
- Resolution
- Propagation
- Embolization
- Organization, recanalization

Arterial thrombus



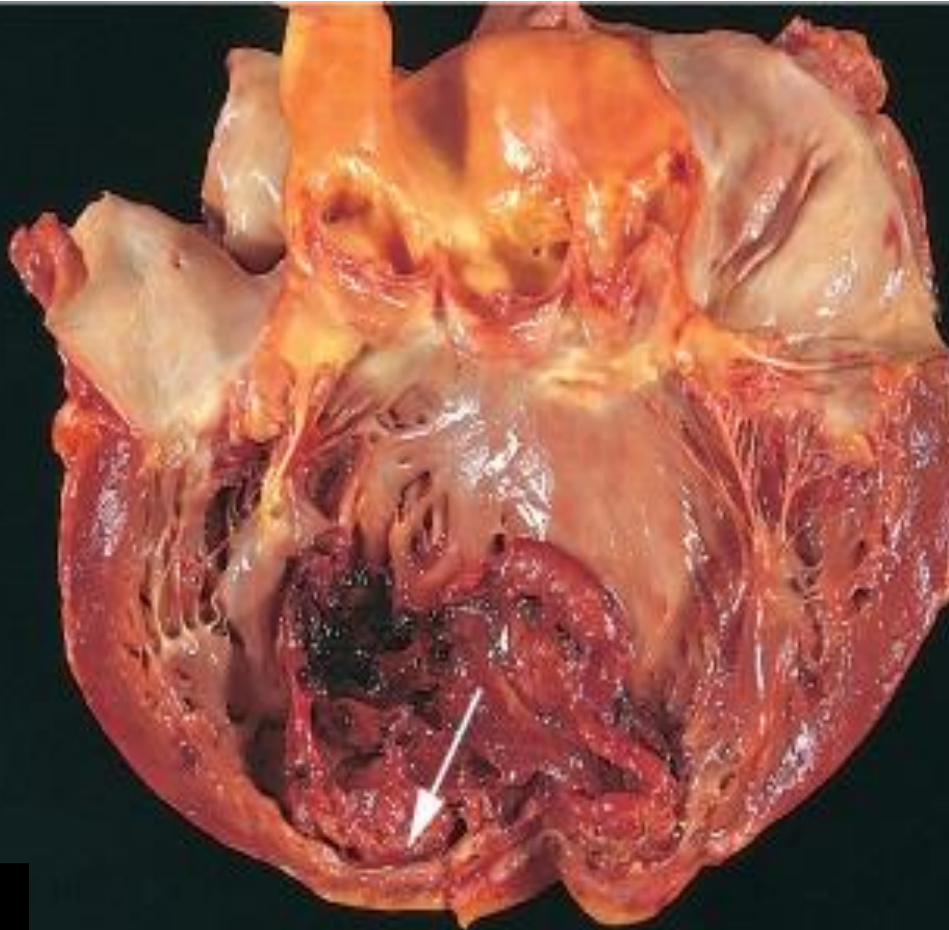
Coronary artery thrombosis

Venous thrombosis

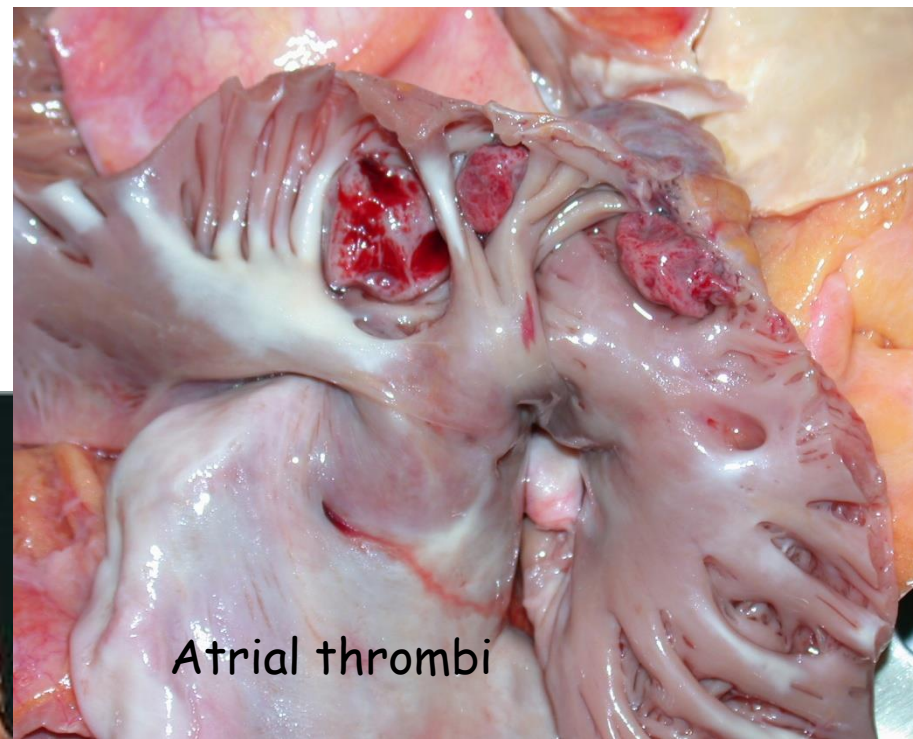


Thrombus in iliac vein

Cardiac thrombi



Mural thrombus- left ventricle



Atrial thrombi



Endocarditis (thrombotic vegetations)

EMBOLISM

- **Embolus:** detached intravascular gaseous, liquid or solid mass carried by the bloodstream from its site of origin to another site where it causes vascular obstruction and subsequent tissue damage (necrosis)
- **Forms:**
 - Thromboembolism
 - Fat embolism
 - Air embolism
 - Amniotic fluid embolism-pulmonary edema, ARDS, DIC
 - Cholesterol embolism- kidney
 - Bacterial embolism- e.g. infective endocarditis
 - Foreign body embolism- i.v. drug abusers

Thromboembolism I.

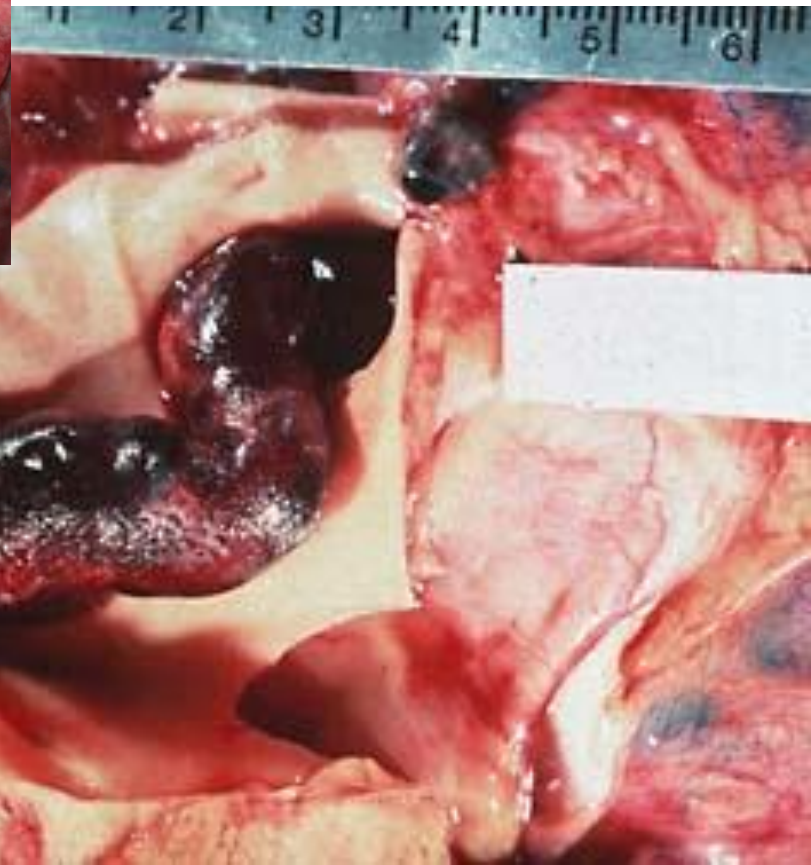
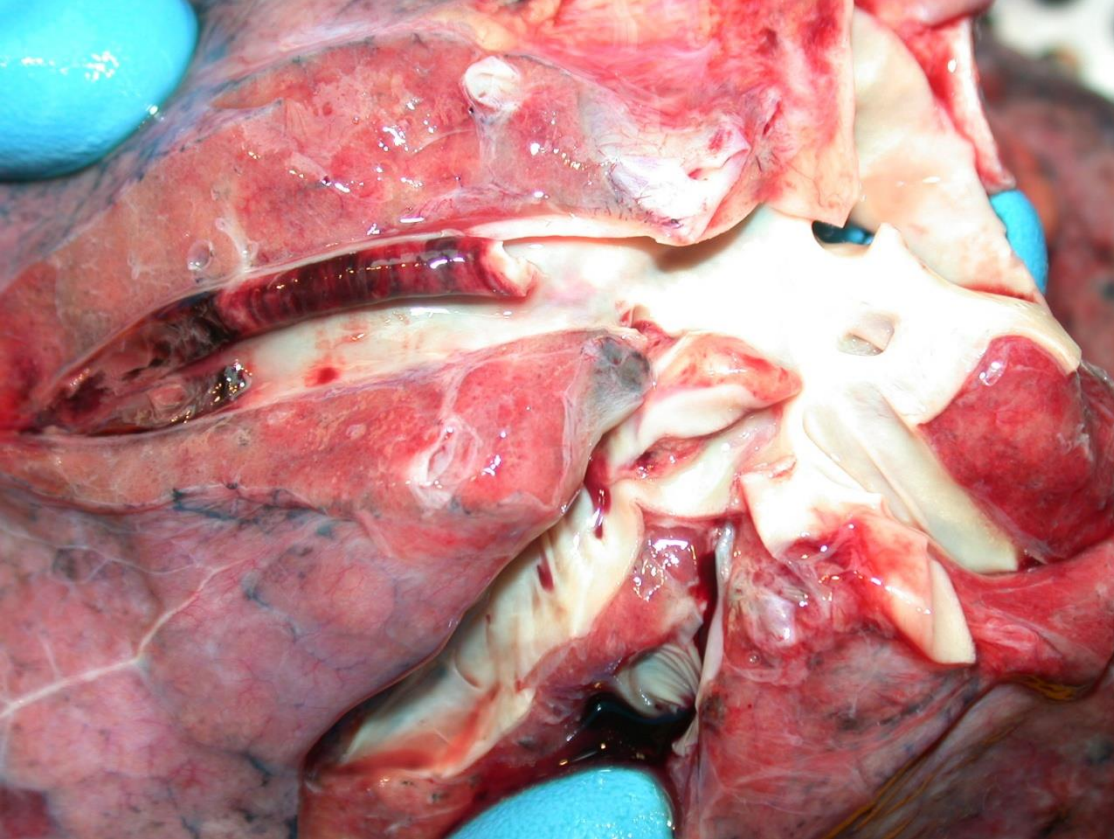
Pulmonary thromboembolism

Source: deep venous thrombosis of lower extremities

Possible complications:

- sudden death (saddle embolus)
- pulmonary hypertension, cor pulmonale
- hemorrhage, hemorrhagic infarction

Pulmonary embolism



Thromboembolism II.

Systemic thromboembolism

Source: -80% mural thrombi within heart chambers (AMI, left atrial thrombi)

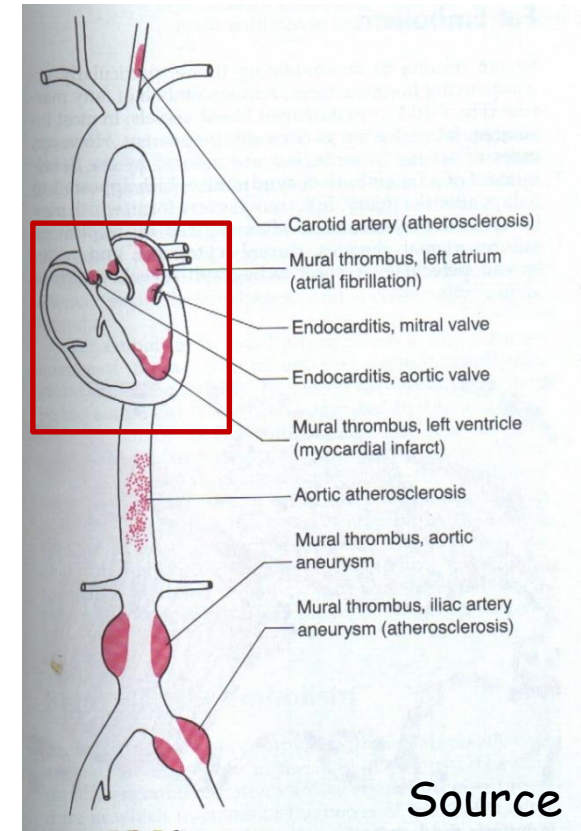
-others: aortic atherosclerosis, aortic aneurysm, vegetations (in endocarditis), unknown origin

Paradoxical embolism!

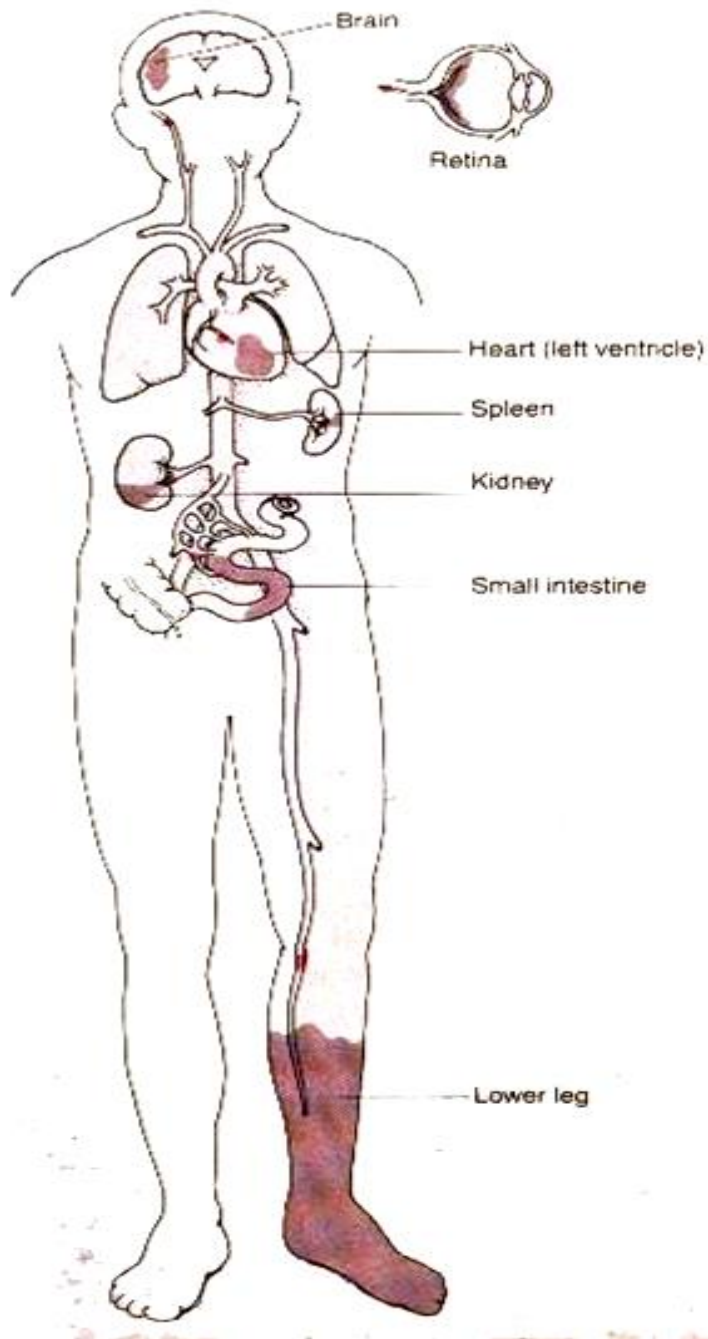
Complications: arterial embolization

- in lower extremities (75%)
- in the brain (10%),
- in arteries of bowel, kidney, spleen, upper extremities

→ INFARCTION



Source



Systemic arterial embolization

lower extremities
brain
retina
heart
spleen
kidney
small bowel

Fat embolism

- After fractures of long bones
- **Clinical signs:** respiratory distress, neurologic symptoms (restlessness, irritability, delirium, coma), anemia, thrombocytopenia
- Mechanic and toxic injury

Air embolism

Causes:-Trauma (chest wall injury)

- obstetric complications

- decompression disease (eg. scuba divers)

- caisson's disease

Complications: neurological symptoms, pulmonary hemorrhage and edema, atelectasis, epiphyseal necrosis of long bones