Hemodynamic disorders

Normal fluid homeostasis

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

Homeostasis vs Hemostasis

Circulatory Disturbances (Disturbances of Blood and Body Fluids)

The health of cells and tissues depends ***not only*** on an **intact circulation** to deliver oxygen and remove wastes ***but also*** on **normal fluid balance.**

Normal fluid homeostasis includes ***maintenance of vessel wall integrity (intact circulation) as well as intravascular pressure, blood volume, and protein content (osmolarity) within certain physiologic ranges***. Therefore any change in one of these factors will affect the tissue homeostasis and may result in ***oedema*** or ***congestion***.

Normal fluid homeostasis also means ***maintaining blood as a liquid until such time as injury necessitates clot formation.*** Clotting at inappropriate sites ***(thrombosis)*** or migration of clots ***(embolism)*** obstructs blood flow to tissues & leads to cell death ***(infarction).*** Conversely inability to clot after injury results in ***haemorrhage***. Extensive haemorrhage can result in ***shock.***

Hemodynamic disorders

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

Hyperaemia (congestion)

- Both these terms denote an excess of blood in the blood vessels of a given part.
- Too much blood being brought in by the arteries (hypereamia) or too little blood being drained out by the veins (Congestion).

Hyperemia

- Hyperemia is an increased volume of blood in a tissue.

– Active hyperemia occurs due to dilation of arterioles & capillaries (exercise, inflammation)

– Passive hyperemia (congestion) occurs due to increased venous pressure that occurs with impaired outflow of blood from the area

- Cyanosis is a bluish discoloration of the lips and skin indicating a lack of oxygen

Active hyperaemia

Increase in the amount of blood in the arterial side of the circulation

General

- Accompanies diseases
- Renal diseaes
- Intravenous medication

Local

- First stage of inflammation
- Increased physiological activity (stomach - intestine udder- muscles)

Local Passive Hyperaemia

Accumulation of blood due to obstruction of the venous drainage in any part of the body

* Pressue by torniquet or bandage
* Pressure induced by tumour, enlarged lymph node , abscess, etc.
* Intestinal obstruction, torsion, or strangulation
* Venous thrombosis

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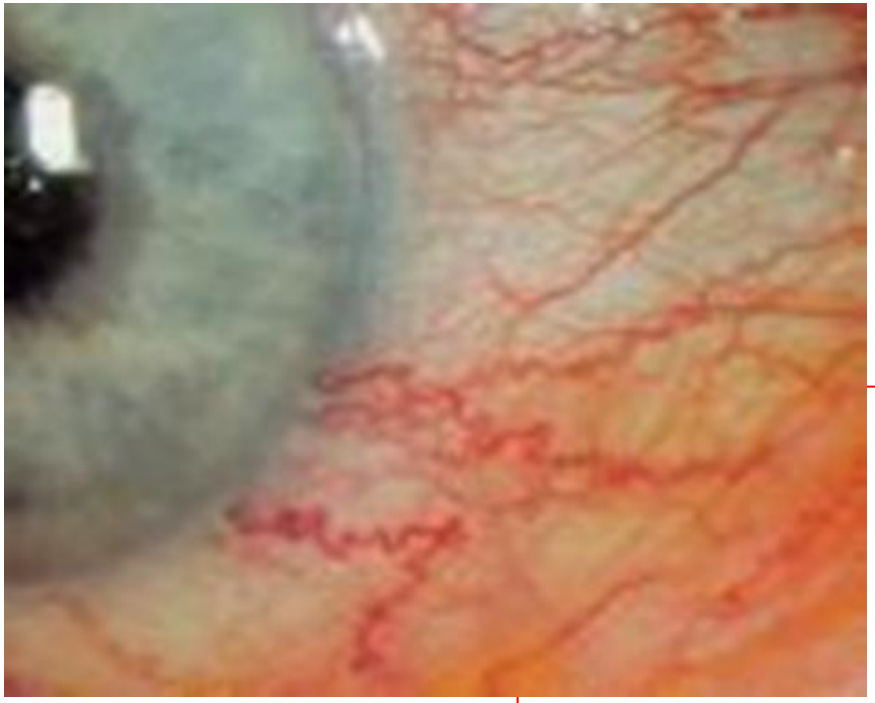
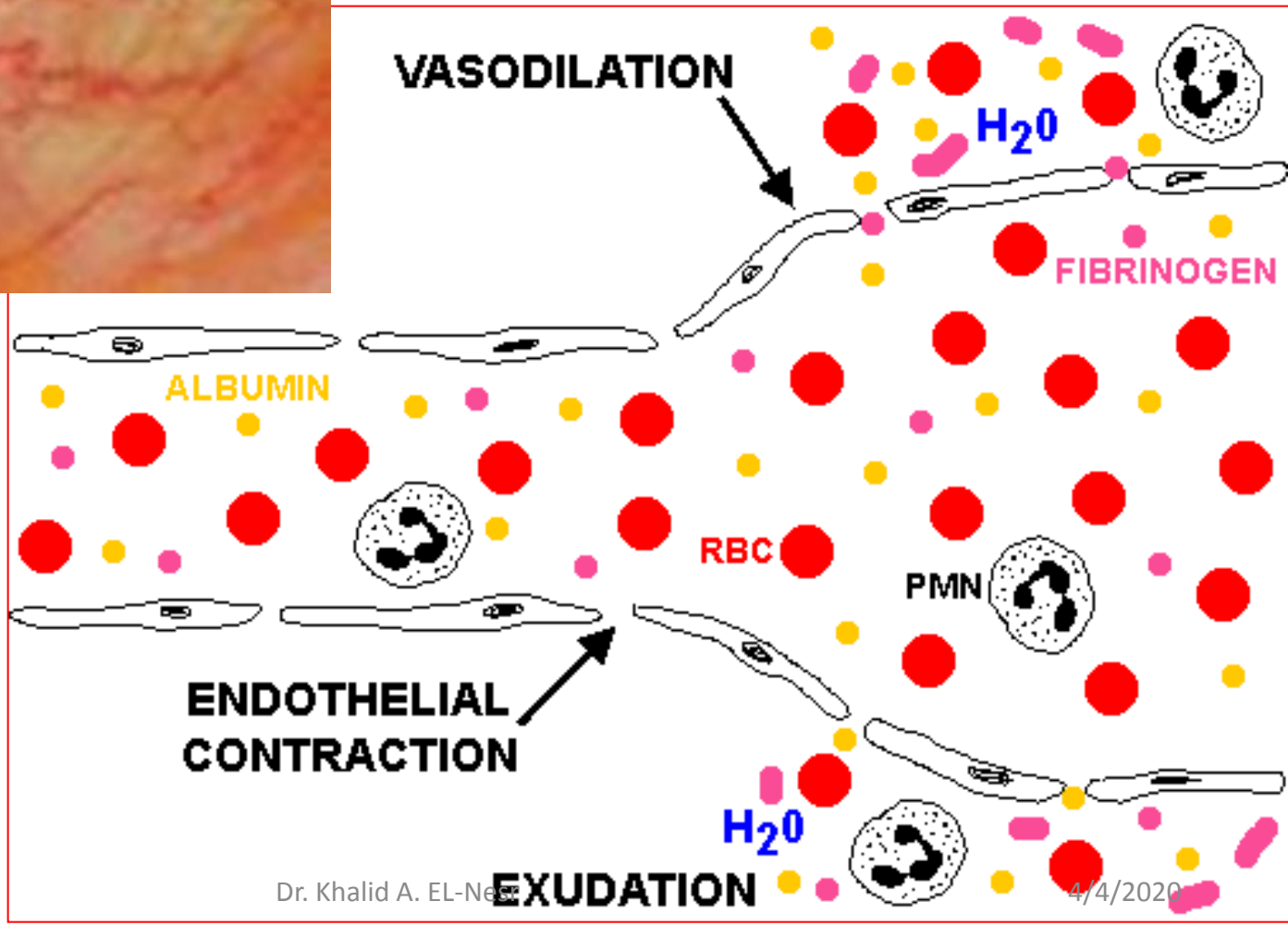



VASODILATION
H_20
FIBRINOGEN
ALBUMIN
RBC
PMN
ENDOTHELIAL
CONTRACTION
H_20
EXUDATION

Consequences of chronic congestion

• Systemic

– 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, fibrosis with time (Induration)

– Skin: cyanosis, anasarca

– Lungs: heavy, firm, heart failure cells on microscopy, (Induration) (lung induration)

• Local

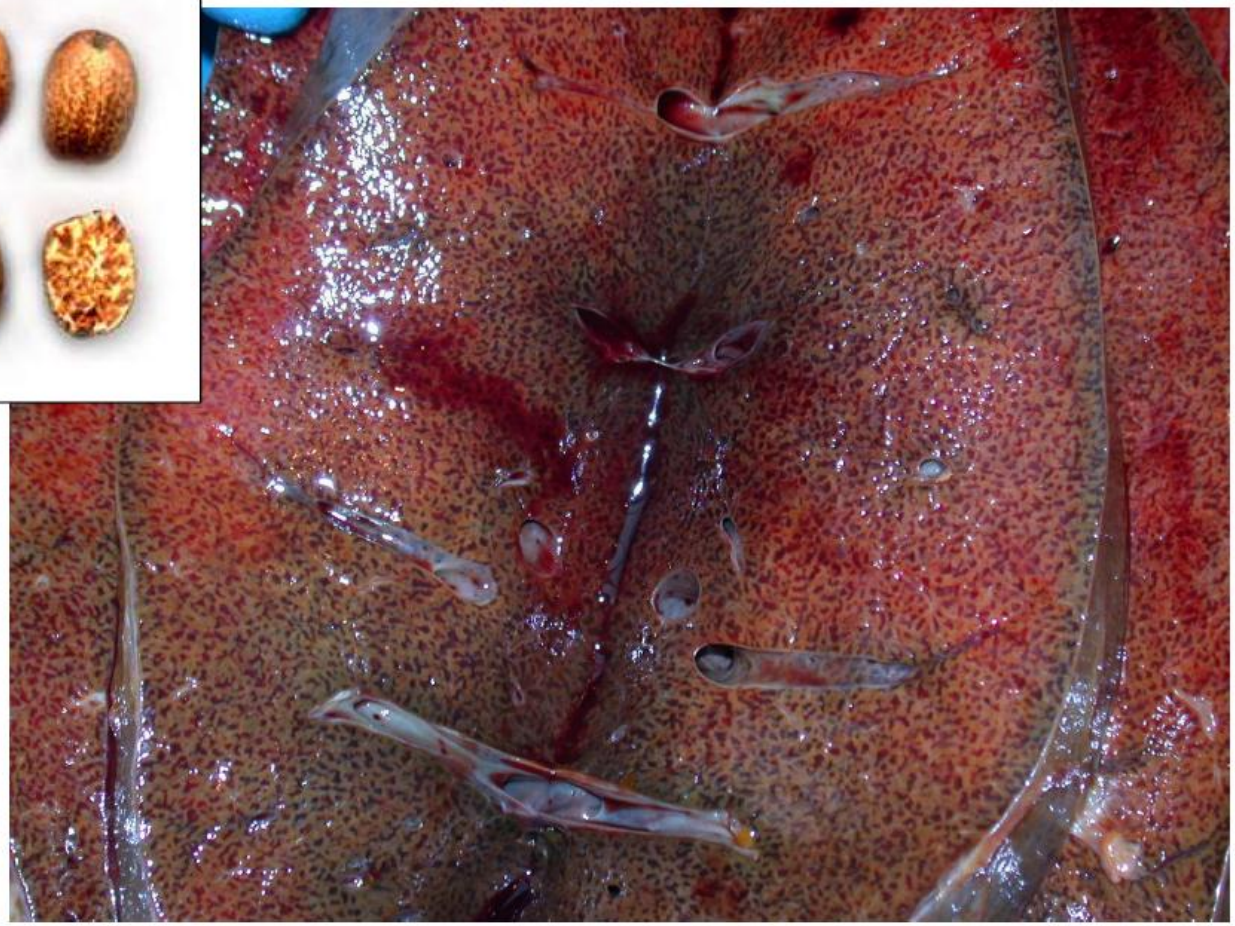
– May occur in every organ, extremities ,……etc.

• Stasis

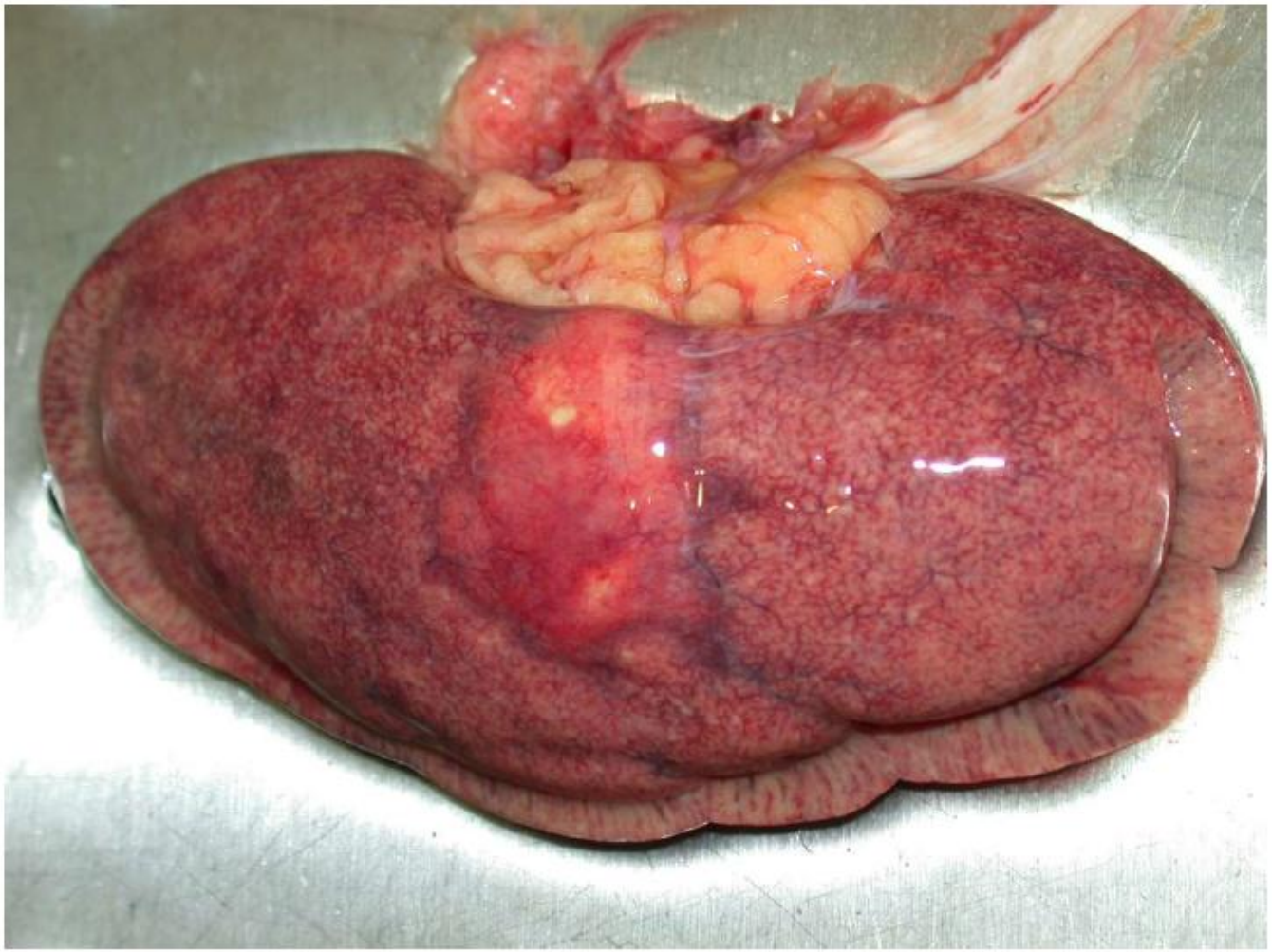
– Arterial supply maintained, venous outflow stopped

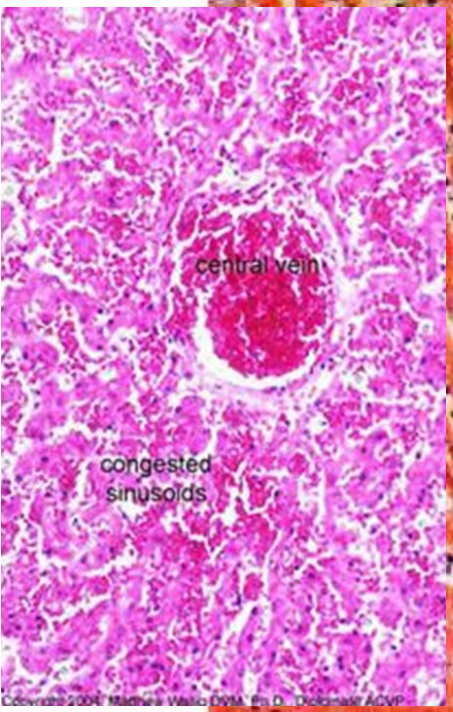
– Consequence: necrosis

Nutmeg liver



Kidney-congestion (+?)





Congestion (liver)

Dilatation of central vein and sinusoids with pressure atrophy on the hepatic cells

Oedema

- An excessive accumulation of fluid (water) in the intercellular spaces or body cavities.

Oedema

- The presence of excessive fluid in tissue or organs is usually indicated by the prefix "hydro", e.g.,
- hydrothorax,
- hydroperitoneum (also called "ascites"), hydrocephalus,
- hydrocele (accumulation of serous fluid in tunica vaginalis of the testis) ,
- "Anasarca" is a generalized oedema (subcutaneous tissue).

Oedema

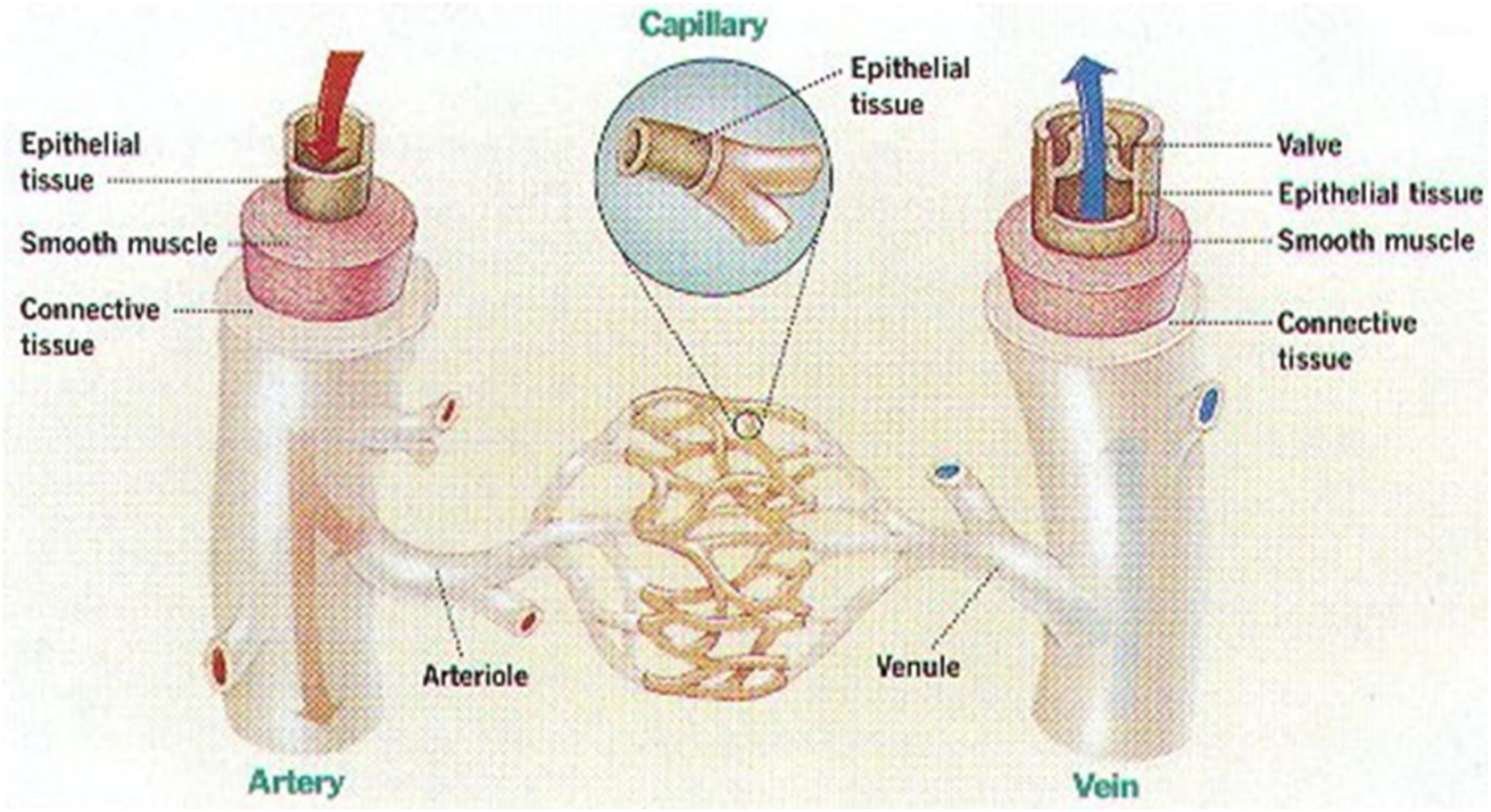
Accumulation of fluid in the intercellular spaces or body cavities

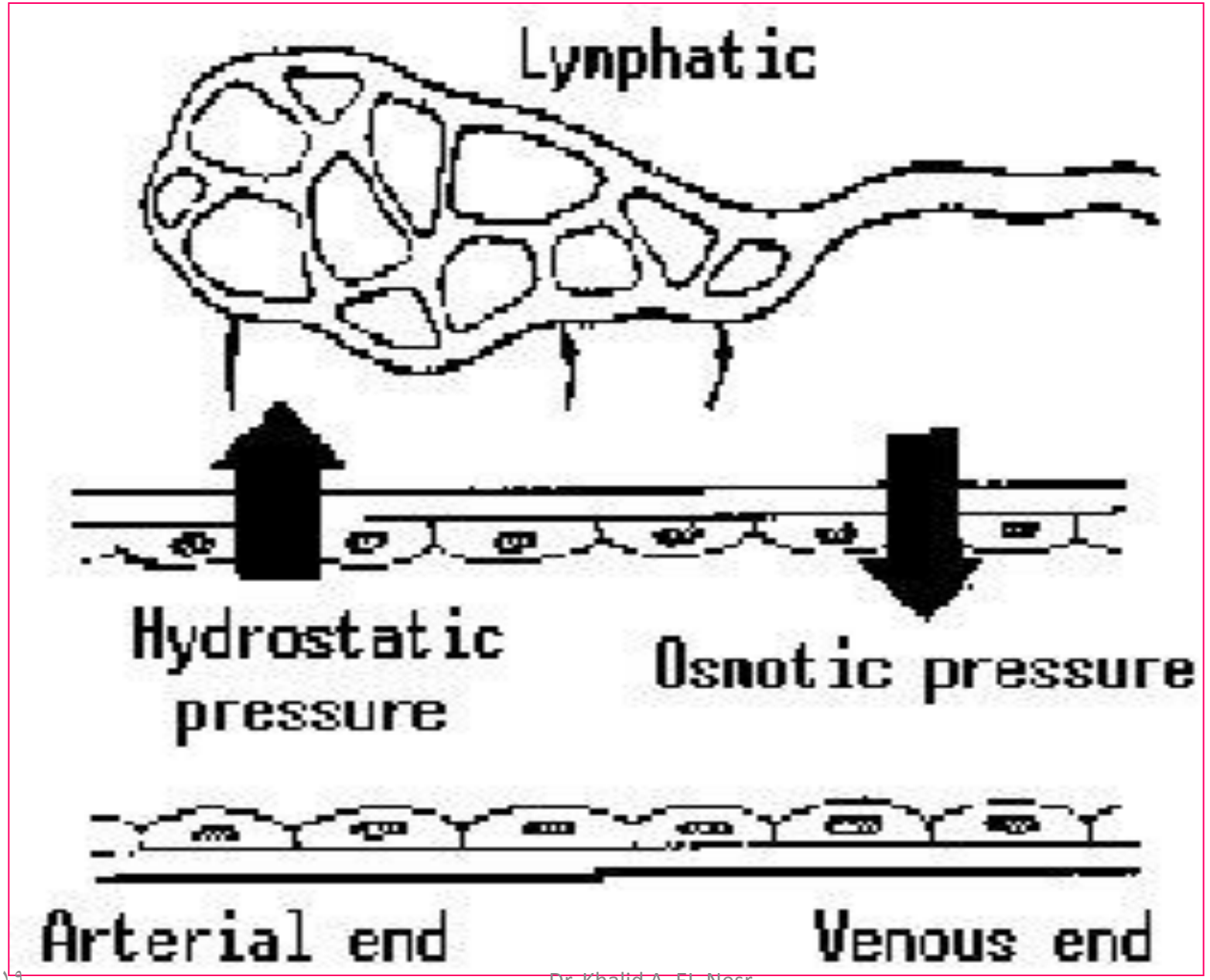
Inflammatory	Non inflammatory

Types of oedema

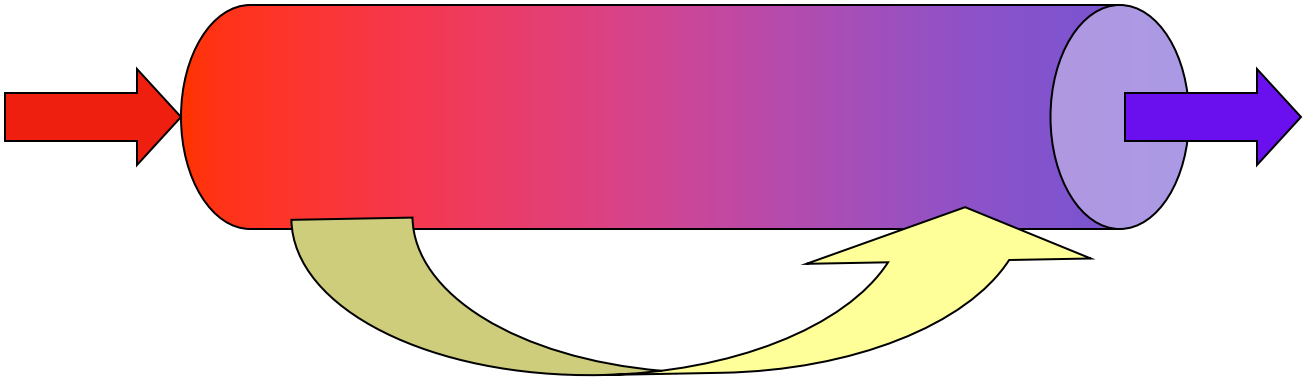
- There are three types, these are
  1- General non-inflammatory oedema.
  2- Local non-inflammatory oedema.
  3- Local inflammatory oedema.

Pathogenesis of oedema




Lymphatic
Hydrostatic pressure
Osmotic pressure
Arterial end
Venous end

Normal Microcirculation



Capillary	Arterial	Venous
Hydrostatic Pressure	45	15
Oncotic Pressure	30	30
Net filtration Pressure	+ 15 mmHg (leak-out)	- 15 mm Hg (Reabsorb)

General Non-inflammatory Oedema

Pathogenesis:

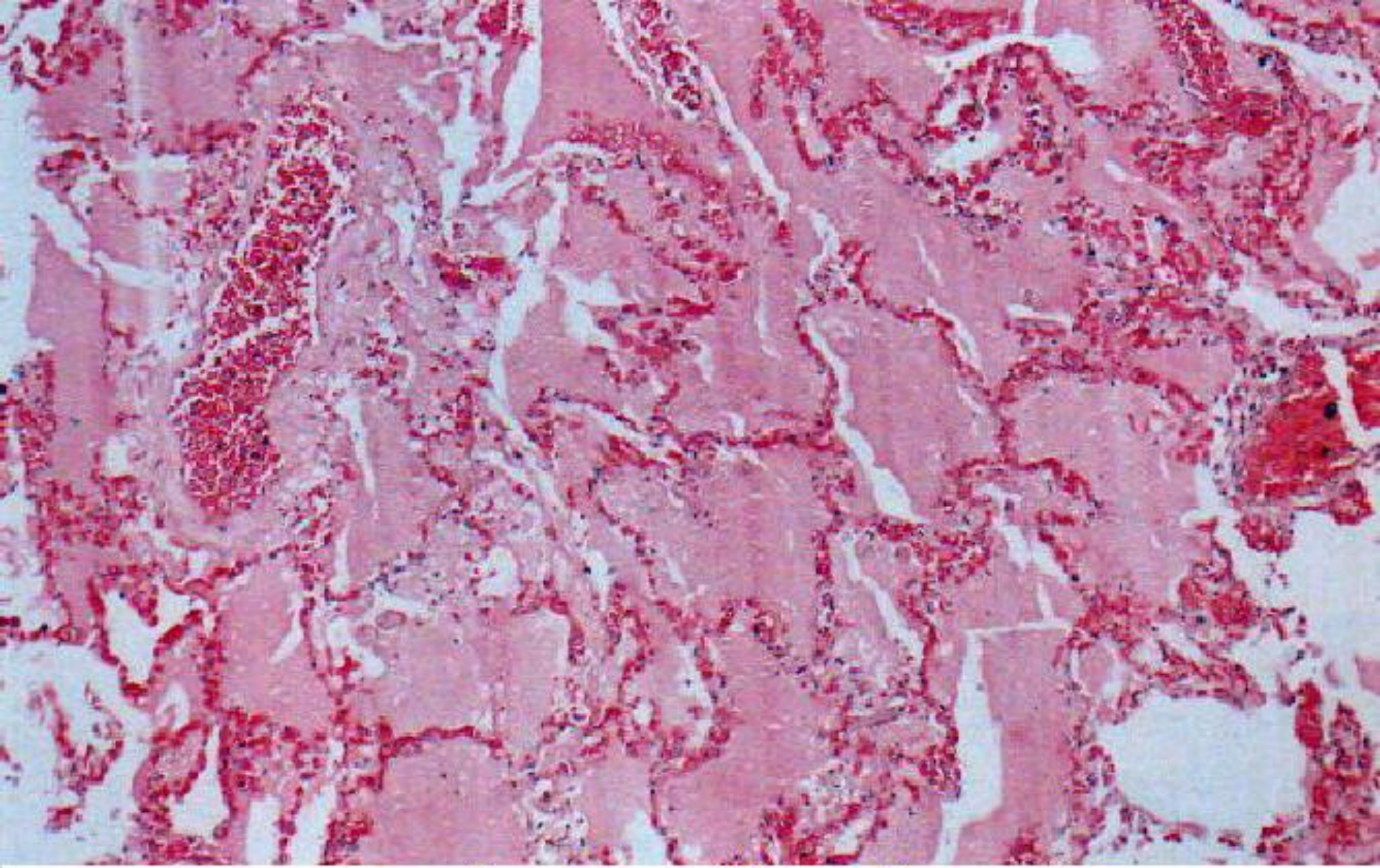
High hydrostatic pressure at the arterial end or low cellular osmotic pressure at the venous end or both

Causes:

- Renal failure (renal oedema)
- Hepatic failure as in cirrhosis (hepatic oedema)
- Low protein intake (nutritional oedema)
- Cardiac failure and passive congestion (cardiac oedema)
- Parasitic infestation (parasitic oedema)
- High level of mineralocorticoids or estrogen (hormonal oedema)
- Histamine shock (allergic oedema)

Microscopical structure

- The intercellular spaces are large (wide). Faint-pink esoinophilic materials may or may not be seen in the section.



Oedema (lung)

The alveoli are filled with proteinaceous oedematos fluid

Hemorrhage

- **Hemorrhage is the loss of blood**

Types of hemorrhage:

1- according to location:

- **external hemorrhage:** blood exits the body (**ext. hem.**)
- **internal hemorrhage:** blood remains in the body (**int.hem.**)

2- according to cavities:

- **hemothorax** is blood in thoracic cavity
- **hemoperitoneum** is blood in peritoneal cavity
- **hemopericardium** is blood in pericardial cavity

3-According to size:

- **a hematoma** is coagulated blood in tissue
- **petechiae** is a pinpoint hemorrhage
- **purpura** is a patch >3mm in size
- **echymoses** are larger hemorrhagic spots on skin and mucosa

Hemorrhage

• Terminology

– hemoptysis refers to coughing up blood from lungs
– hematemesis refers to vomiting blood from upper GI tract
– hematochezia refers to passing bright red blood per rectum
– melena refers to passage of dark (black) stools
– hematuria refers to passage of blood (intact RBCs) in urine
- hemoglobinuria refers to passage of blood (Hb) in urine
– metrorrhagia refers to excessive menses

• Symptoms depend on amount, site, duration of blood loss
– rapid loss of less than 20 % of blood volume is compensatable
– massive loss (>1500 ml) results in hypovolemia and shock
– chronic loss results in anemia

Haemorrhage - 1

Mechanism

Rhexis

Break in the wall of the blood vessel

Diapedesis

Oozing through unbrocken intact vessels

Classification

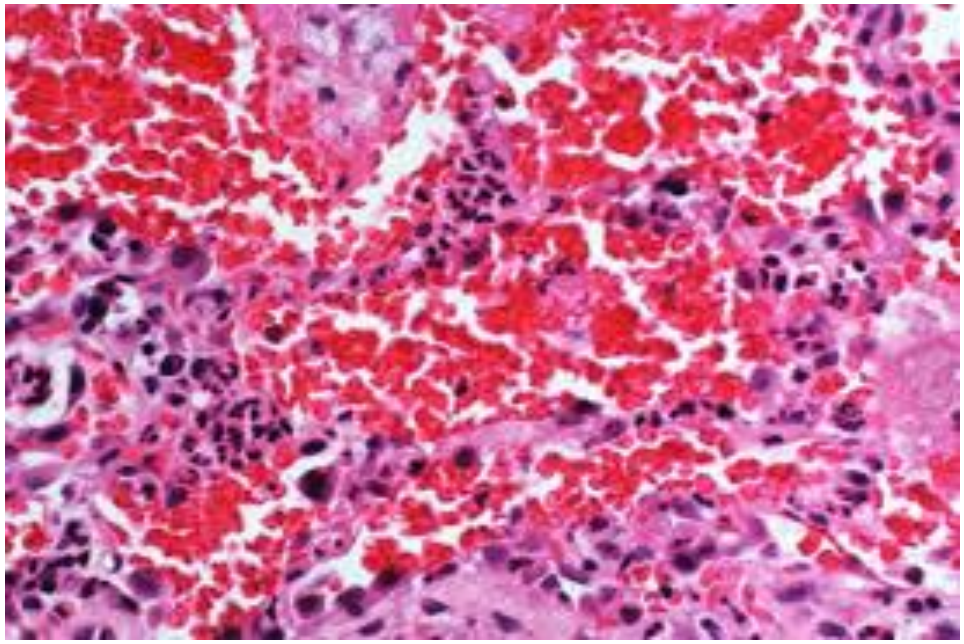
Source Cardiac - arterial - venous - capillary

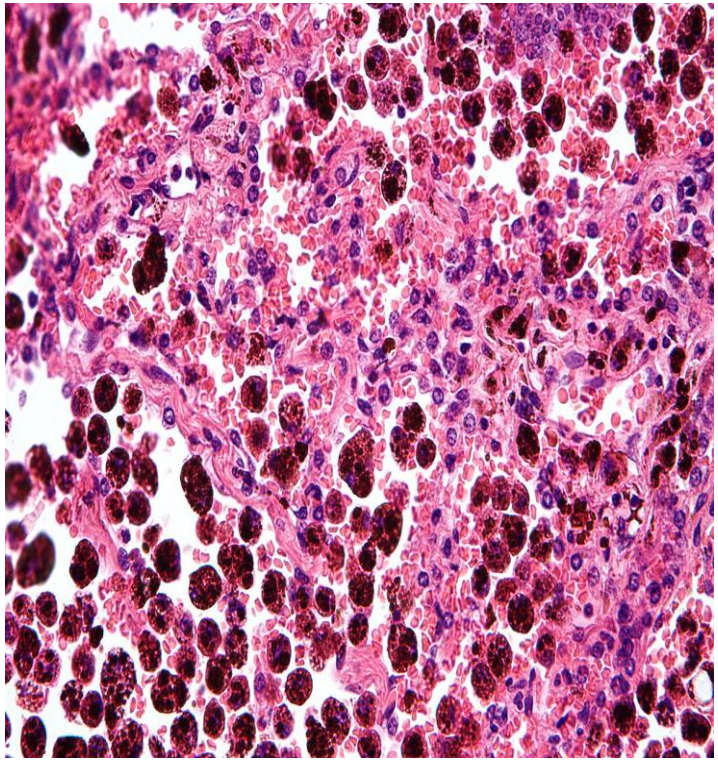
Size Petechiae (1mm) - echymosis (1 cm) - spots

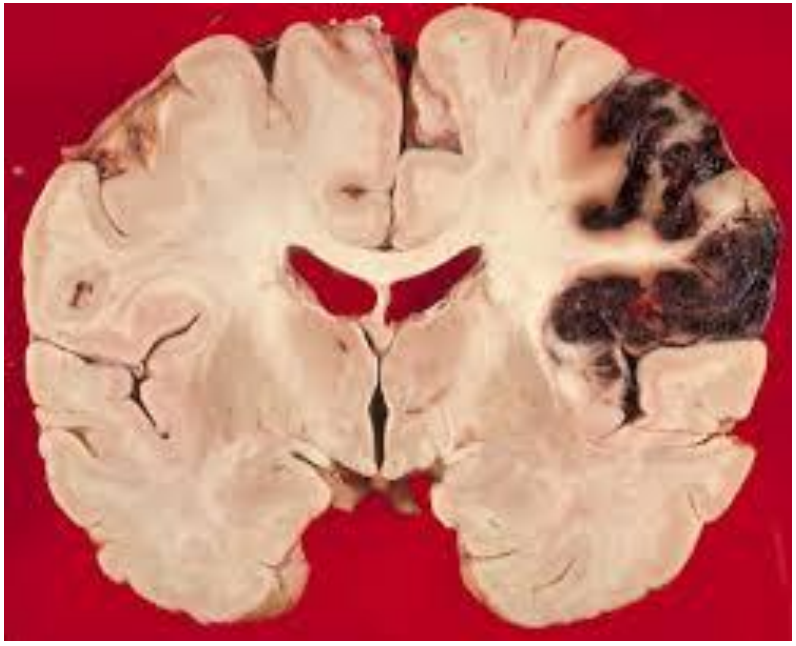
Location Submucous - subcapsular-subserous

From body openings Epistaxis (nose)

haemoptysis (mouth) haematemesis (stomach)
enterrhagia (intestine) metorrhagia (uterus)
haematuria (blood in urine)







clot formation

- **Blood clots in order to prevent loss of blood**

(if endothelium is damaged then a "plug" is made to fill hole)

- **vessel constriction, platelet plug, reinforced by fibrin**
- **Clotting requires platelets, endothelium, plasma proteins**

– normally there is a balance of clot formation and clot lysis

Thrombosis

- **Thrombus is formation of clot within vessel during life**

– formation of a thrombus may cause complications

– certain factors predispose to thrombus formation:

- **stasis of blood (CHF, dehydration)**
- **hypercoaguable states (inherited, malignancy)**
- **endothelial injury (thrombogenic surface revealed)**

Thrombosis

Types:

According to location

Cardiac - arterial - venous - capillary - lymphatic

According to shape

Lateral - occluding - sadle - canalized

According to colour

White(pale) - red - mixed - laminated

According to infectivity

Aseptic - septic - parasitic

Mechanism

Thrombocytes accumulate on the endotheium

*

Liberation of thromboplastin

*

Stimulation of fibrin formation

*

Erythrocytes and leucocytes become embedded into the fibrin network

Thrombosis - 2

Causes and predisposing factors

Inflammation
(phlebitis, migration of strongulus vulgaris larvae, pyogenic infection)

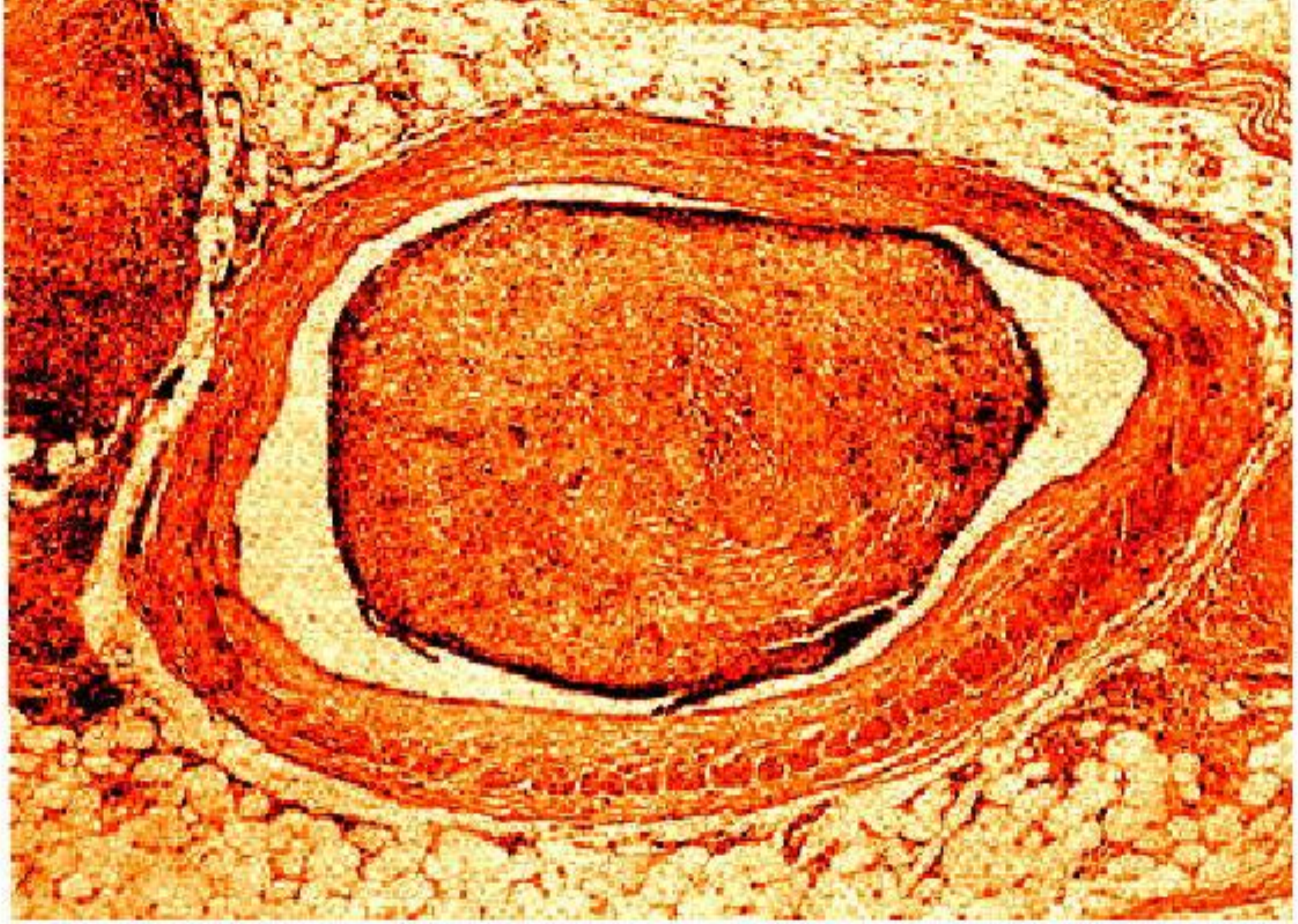
Slowing or stasis of blood flow
(cardiac insufficiency, passive congestion)

Disruption of laminar flow of the blood stream
(aortic arch, bifurcation of vessels, anurysm, varicose thrombosis, external pressure)

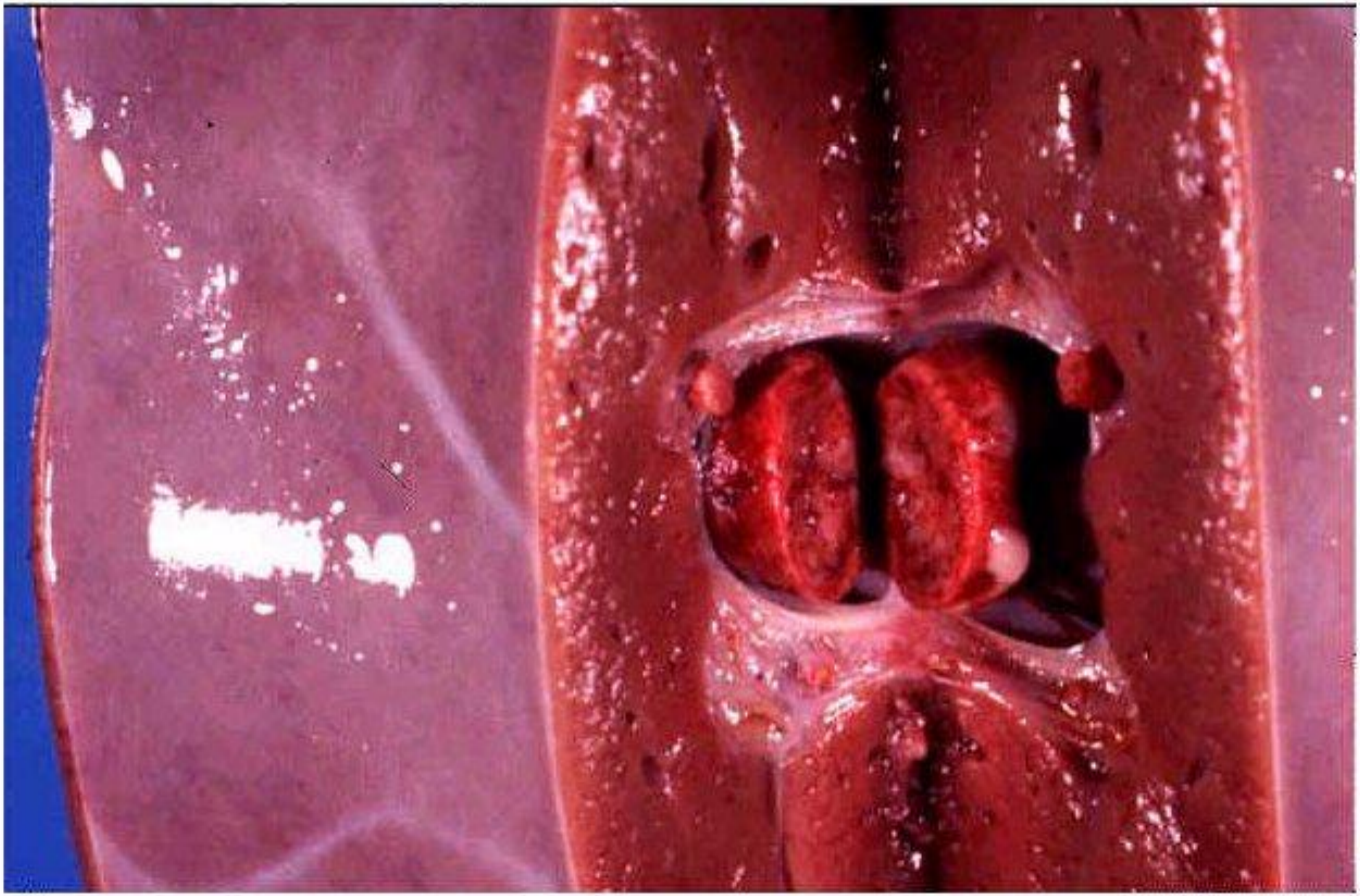
Changes in the composition of blood
(surgery, parturition, trauma)

Fate

Contraction - fragmentation - liquefaction - organization - canalization - mineralization



Thrombosis of an artery



Liver (Cow): Thrombosis of the portal vein

Thrombosis (clot formation)

- **Fate of thrombus :**

– Lysis of the thrombus removal of thrombus by fibrinolysis

– Organization and recanalization replacement of the thrombus by granulation tissue and creation of new channels through thrombus

– Propagation is complete occlusion of a vessel with extension of the thrombus proximal in vein

– Embolus formation is detachment of a thrombus and impaction lodge distally

Embolus

- An embolus is a thrombus or other movable intravascular mass that circulate and may cause obstruction of a vessel

- Types of emboli
  - thromboemboli (99%)
  - fat emboli
  - gas emboli
  - solid emboli
  - liquid emboli

Embolism

Lodgement of foreign bodies circulating in the blood in small arteries and capillaries

Types

* Pieces of thrombi (simple emboli)
* Fat globules
* Gases
* Parasites
* neoplastic cells
* foreign bodies
* clumps of blood cells
* coagulated protein
* clumps of bacterial colonies

Effect

Infarction - abscess - metastatic tumour

Infarction

- **Infarction is irreversible ischemic necrosis of cells usually due to occlusion of arterial supply**

Factors influencing outcome of vessel occlusion include

– pattern of vascular supply (presence of dual blood supply)
– rate of development of occlusion
– vulnerability of tissue to hypoxia
– oxygen content of blood

End result of an infarct depends on tissue's ability to repair

– heart heals an infarct by fibrosis
– liver is able to replace damaged tissue over time
– brain is unable to regenerate or create a scar and forms

Infarction - 1

Ischaemia and necrosis in a local area of the body due to obstruction of its blood supply

Pathogenesis

Obstruction of an end-artery

*

engorgement of the capillaries in the area with blood forced from the collateral circulation

(stage of red infarction)

*

haemorrhage by diapedesis in the area

(stage of haemorrhagic infarction)

*

Autolysis of erythrocytes and coagulative necrosis of tissue

(stage of pale infarction)

The area of infarction is surrounded by hyperaemic inflammatory zone containing many leucocytes

Infarction - 2

Causes of obstruction of blood vessels in infarction

Thrombosis

Embolism

Substances that cause vasoconstriction

Compression of the blood vessel (abscess, tumour, cyst)

Effect

No effect if the necrotic area is small

*

Organisation and scar formation

*

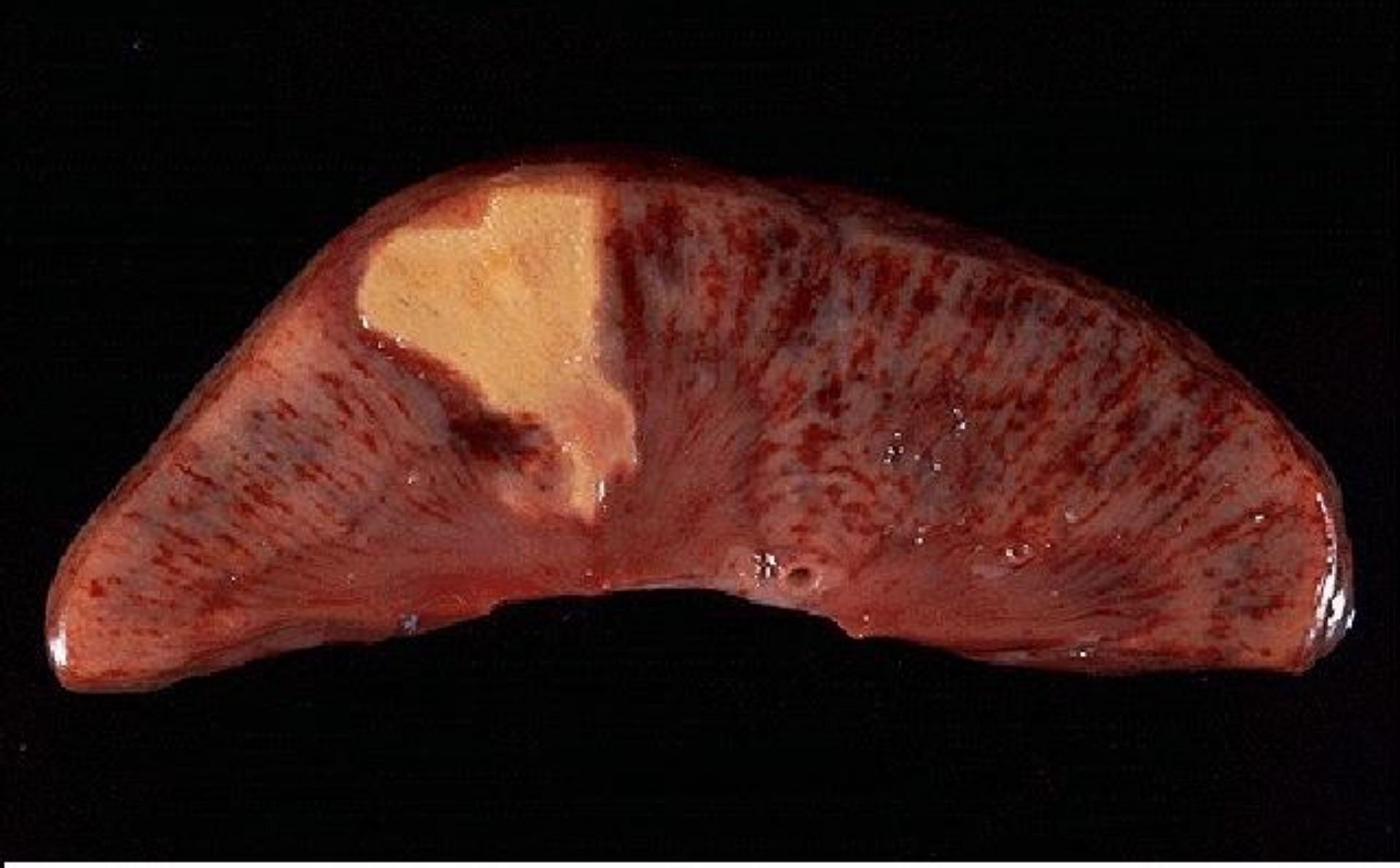
Shock due to absorption of necrotic material

*

Bacterial invasion and abscess formation or gangrene

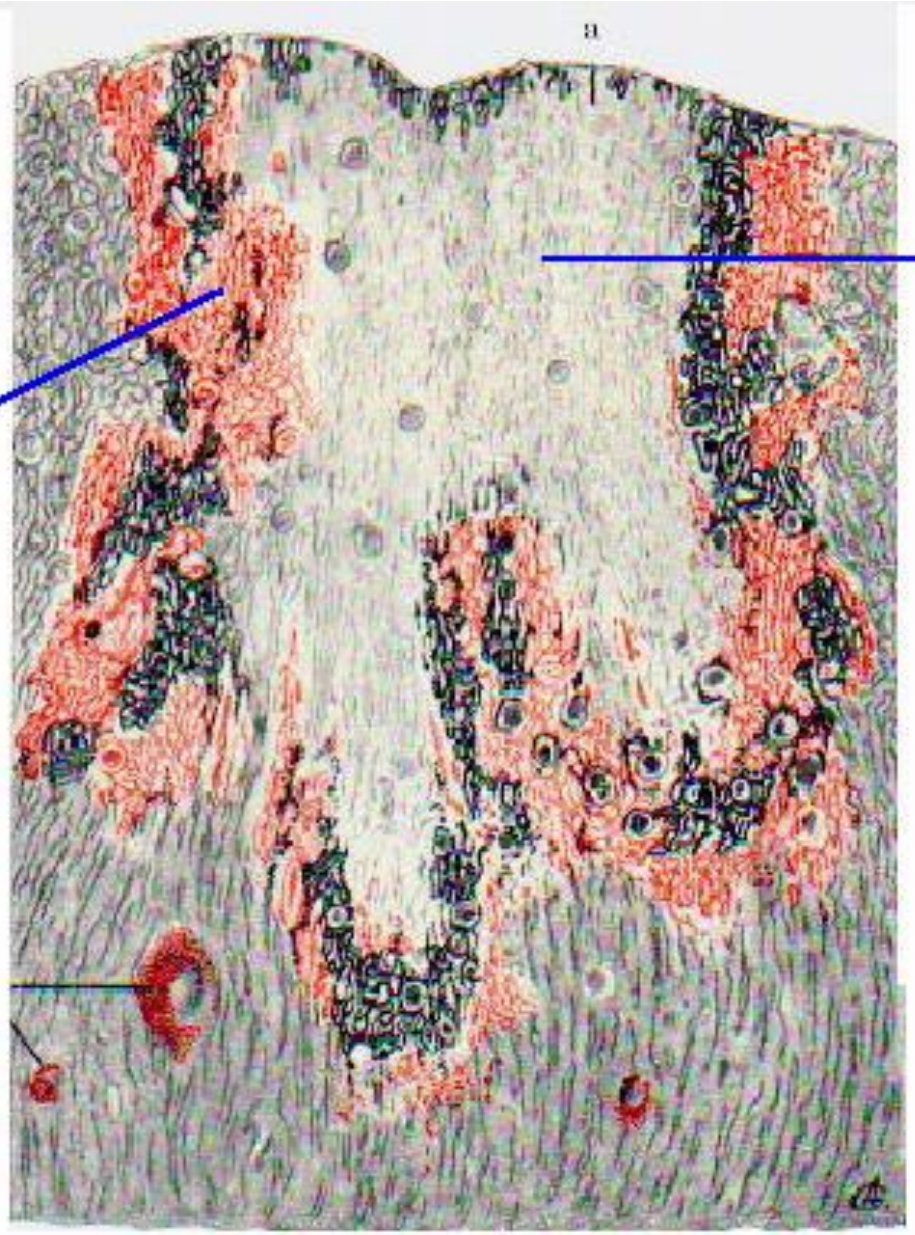
*

Death when infarction occurs in a vital organ (heart, brain)



Kidney: Infarction

Wedge-shaped pale area of coagulative necrosis



Infarction (kidney)

Shock

- **Wide spread hypoperfusion of tissue due to reduction in the bloods volume or cardiac output, or redistribution of blood, resulting in an inadequate effective circulatory volume.**

Shock

Inadequate supply of the brain with blood leading to clinical abnormalities
(subnormal temp., unconsciousness)

Types of shock according to the cause

Hypovolaemic
Cardiogenic
Traumatic
Infectious
Nervous

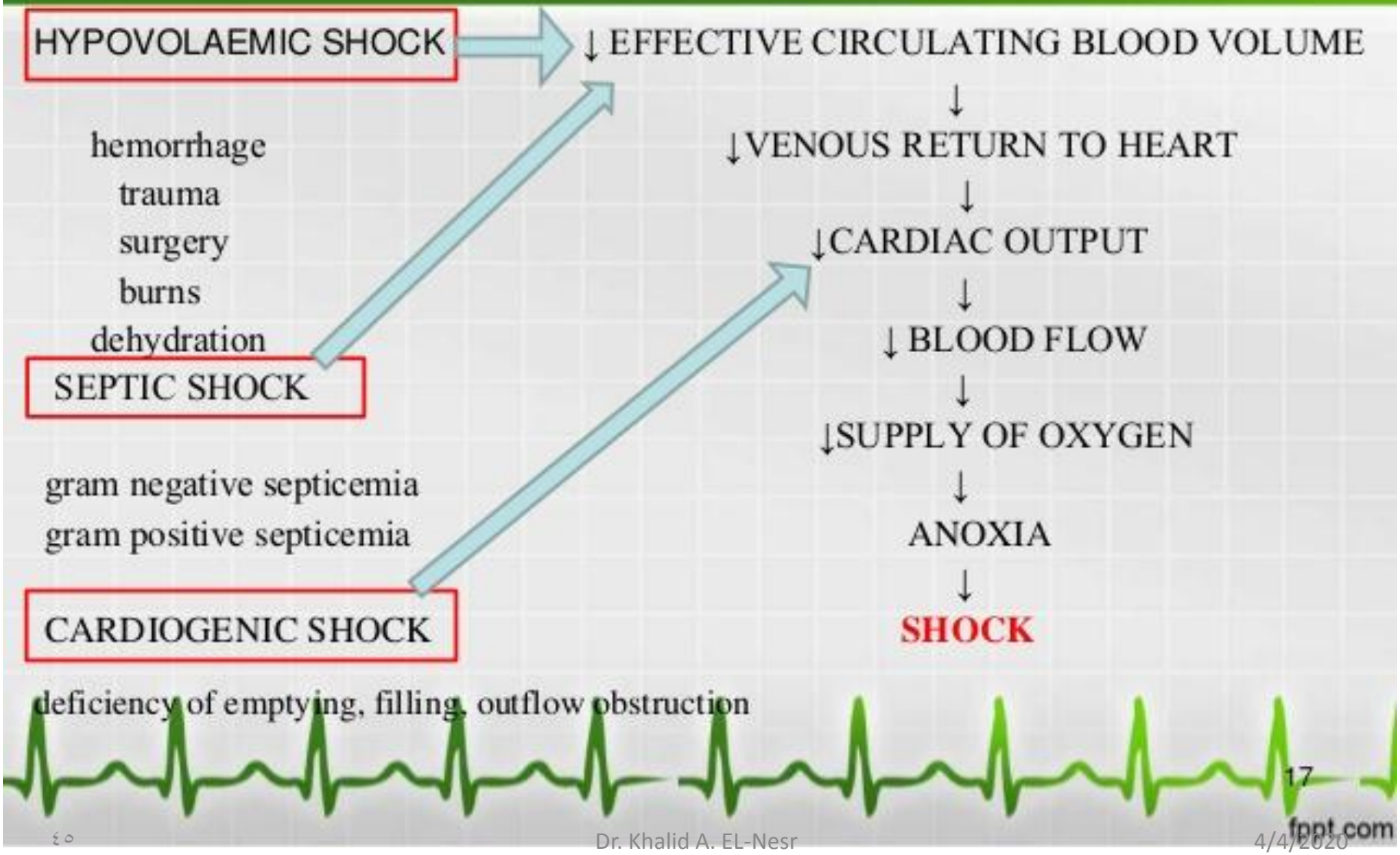
Types according to duration

Primary or Secondary

Shock

- **Hypovolemic (hemorrhagic) shock:**
  - Due to inadequate blood or plasma volume caused by hemorrhage, fluid loss from severe burns or trauma (traumatic shock)
- **Cradiogenic shock:**
  - Caused by failure of the myocardial pump due to myocardial damage, ext. pressure, outflow obstruction
- **Septic shock:**
  - Caused by severe bacteremic infections, most commonly by Gr – ve bacteria (endotoxic shock) and occasionally by Gr +ve and fungi
- **Nervous shock:**
  - Associated with anesthetic accidents, spinal cord injury and caused by massive peripheral dilatation.

PATHOPHYSIOLOGY



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shock

- **Grossly:**
  - **Hyperemia**
  - **Hemorrahge, oedema (lung –intestine)**
  - **Bood tinged fluid in the body cavities**
  - **Spleen is smaller in size (empty)**
- **Histopathologically:**
  - **Deg. And necrosis**
  - **Dilated bvs**
  - **Hemorrhage**
  - **Oedema**