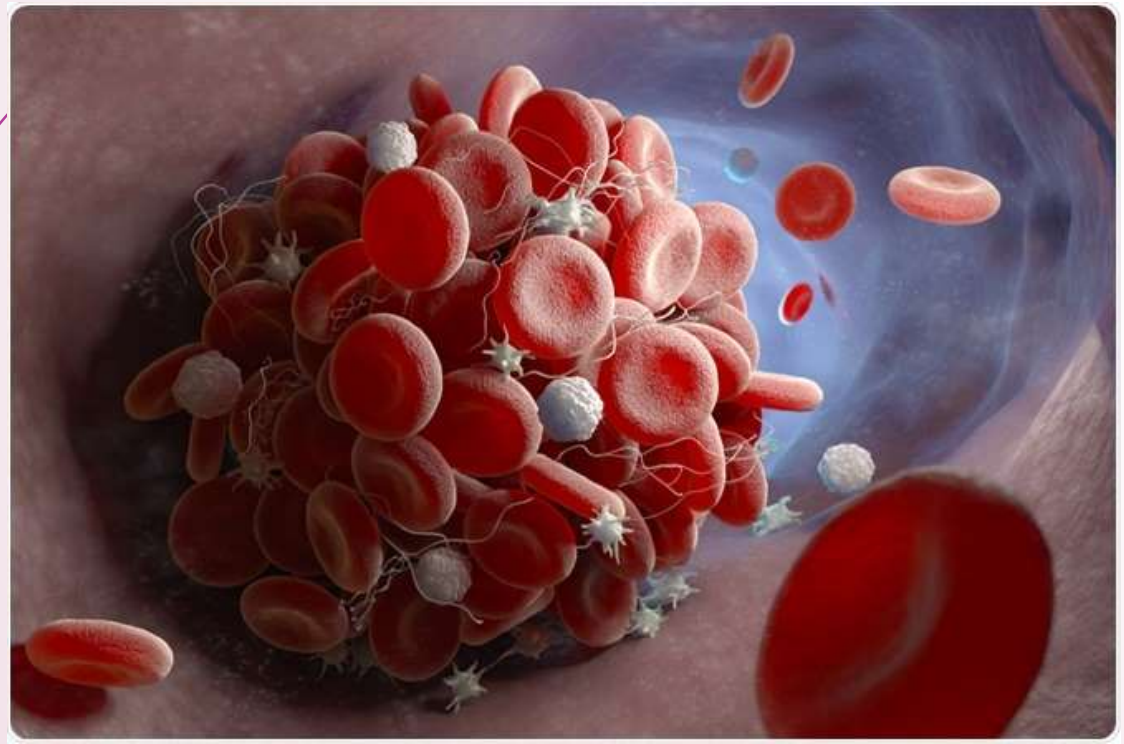
* chapter 4
↪ Robbins

Hemodynamic Disorders, Thromboembolism, and Shock



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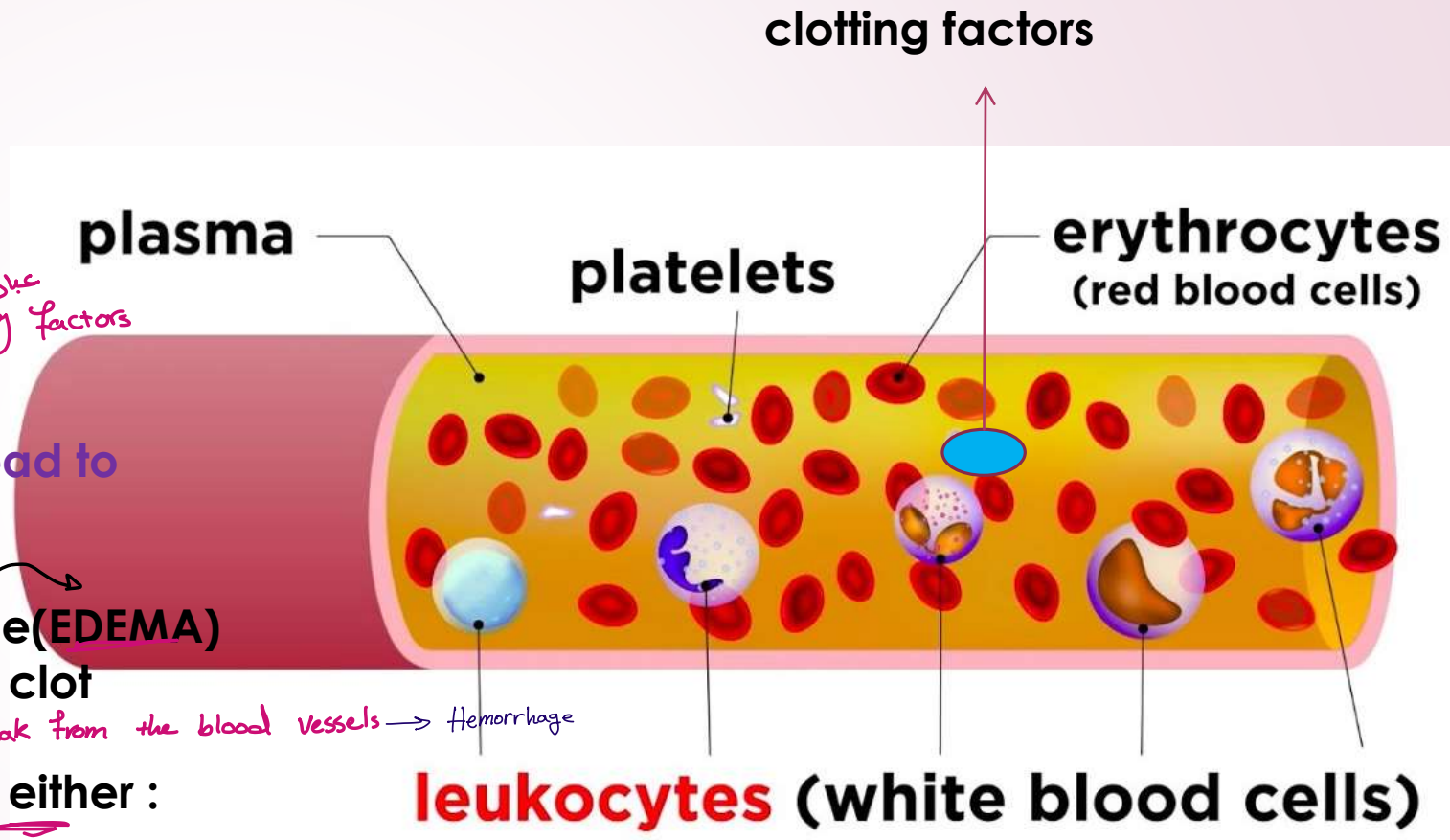
* Hemodynamic disorders :
↳ disorders that affecting the circulation.

Composition of blood

1. **plasma protein (**Fluid and electrolyte).
2. **RBC.** + WBC → plasma protein بتسبح بـ
3. **Clotting pathway.** → عبارة عن مجموعة platelets + clotting factors



So any disturbances (disease) in these processes lead to pathological conditions: e.g

1. Defect in Fluid and electrolyte balance(EDEMA)
2. Damage to blood vessels or defective clot formation (HEMORRHAGE) → if the RBCs leak from the blood vessels → Hemorrhage
3. Disturbance in clotting pathway led to either :

- ① Hemorrhage. ✓
- ② thromboembolism ✓ ↓ جلطات.

* Clotting pathway :
↳ Group of proteines help us to stop bleeding.

← واذا هاي البروتينات من وظيفتها إن الجسم يخلق كميات كبيرة من RBCs

thromboembolism ← يعني جلطات (clott) واذا صار العكس (جسمي كل شوي بكون) ويصير (Hemorrhage).

So clinically we have: (will) the following catogeries :

1. fluid and electrolytes disturbance : ((first component of the blood))

* So, any disturbance in this component in form of :

1. increased volume : HYPEREMIA AND CONGESTION (clinical items)
2. abnormal distribution : EDEMA
3. Decreased volume:
   - INFARCTION. (1 or 2) → single tissue (decrease in blood supply to a single organ)
   - Shock → generilized decrease in the effecting circulating volume either because the blood is low or the heart is not pumping good.

2. Inadequate hemostasis : (2nd component)

- HEMORRHAGE (No clotting factor) — يا عندي في clotting pathway
- THROMBOSIS and EMBOLISM (excess)

3. disturbance in RBC:

- extravasation from vessels: HEMORRHAGE.

(يا اطلع RBCs ↓ vessels)

** first component

1. HYPEREMIA AND CONGESTION (increased volume)

- **Hyperemia and congestion both refer to an increase in blood volume within a tissue.**
- **Hyperemia is an active process resulting from arteriolar dilation and increased blood inflow, as occurs at sites of inflammation or in exercising skeletal muscle.**

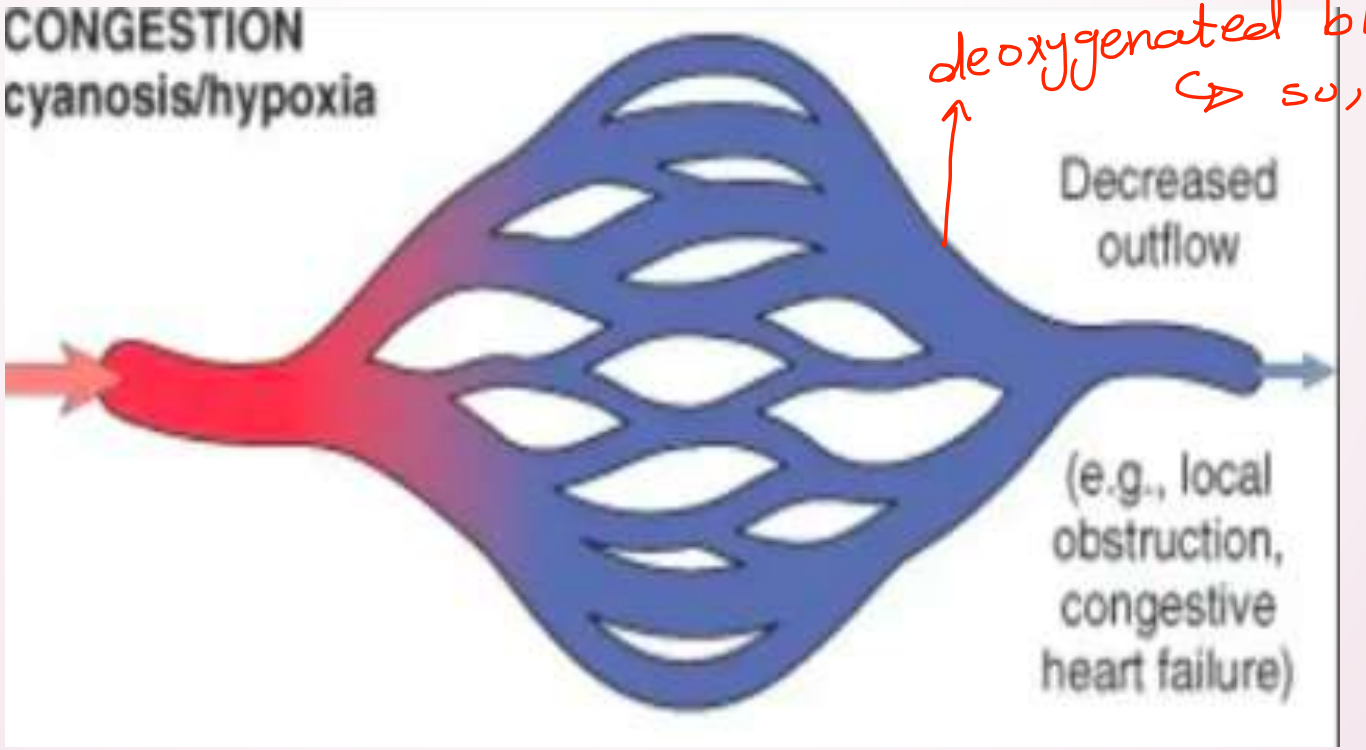
excess Blood

(1)

(2)

deoxygenated Blood

- Congestion is a passive process resulting from impaired outflow of venous blood from a tissue.
- It can occur systemically, as in cardiac failure, or locally as a consequence of an isolated venous obstruction.



Clinically

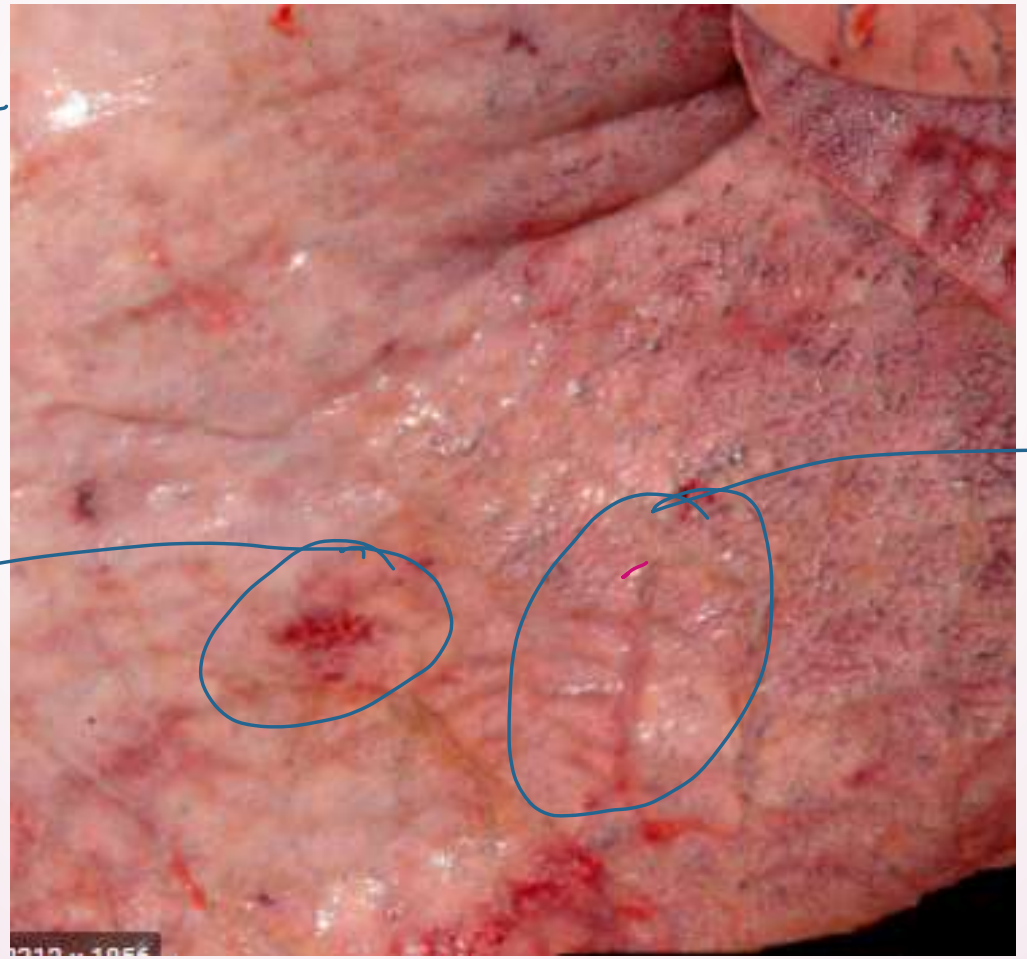
Hyperemic tissues are redder than normal because of engorgement with oxygenated blood

Congested tissues have an abnormal blue-red color (cyanosis) that stems from the accumulation of deoxygenated hemoglobin in the affected area.

they are not occuring only in skin or external organs, they are occur also in internal organs.

I. LUNG CONGESTION.

Cut surfaces of hyperemic or congested tissues feel wet and typically ooze blood



lung tissue

site of rupture

مع الوقت نتيجة زيادة pressure بصير Rupture

prominent BV

excess blood

capillary bed مع الوقت، اذا هنا ظل فيان كم بصير

consequence

Rupture

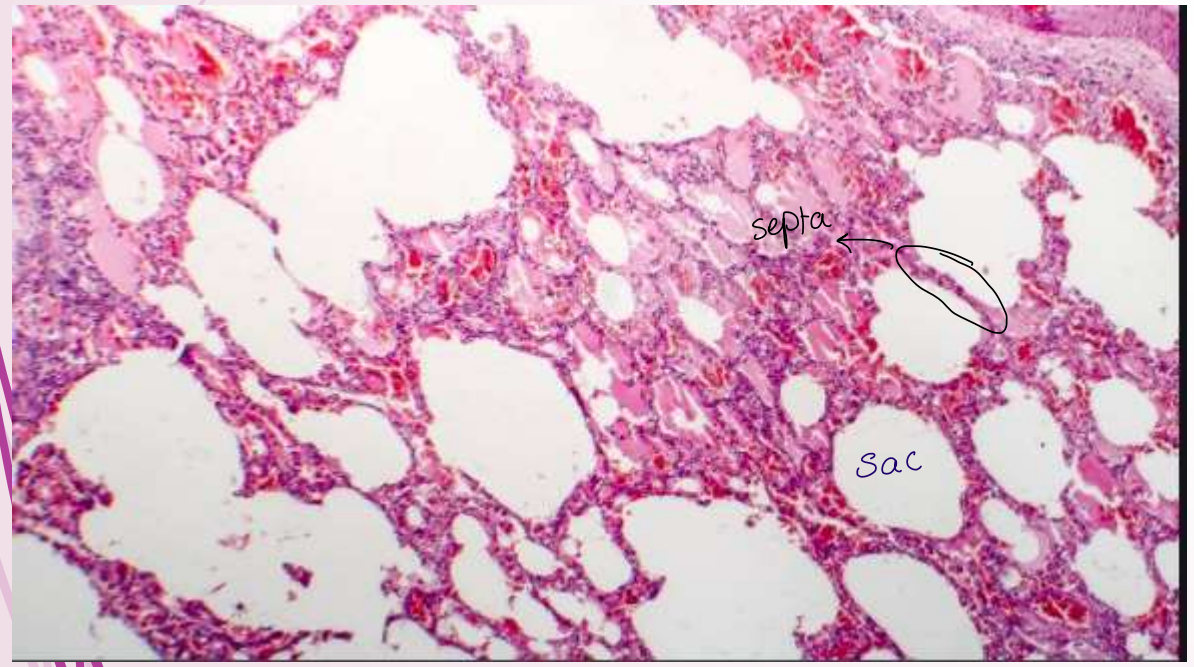
BV مع الوقت بصير فارج بطلع كمية كبيرة من الدم بطلع كمية صغيرة من الدم (oozing)

Microscopic examination:

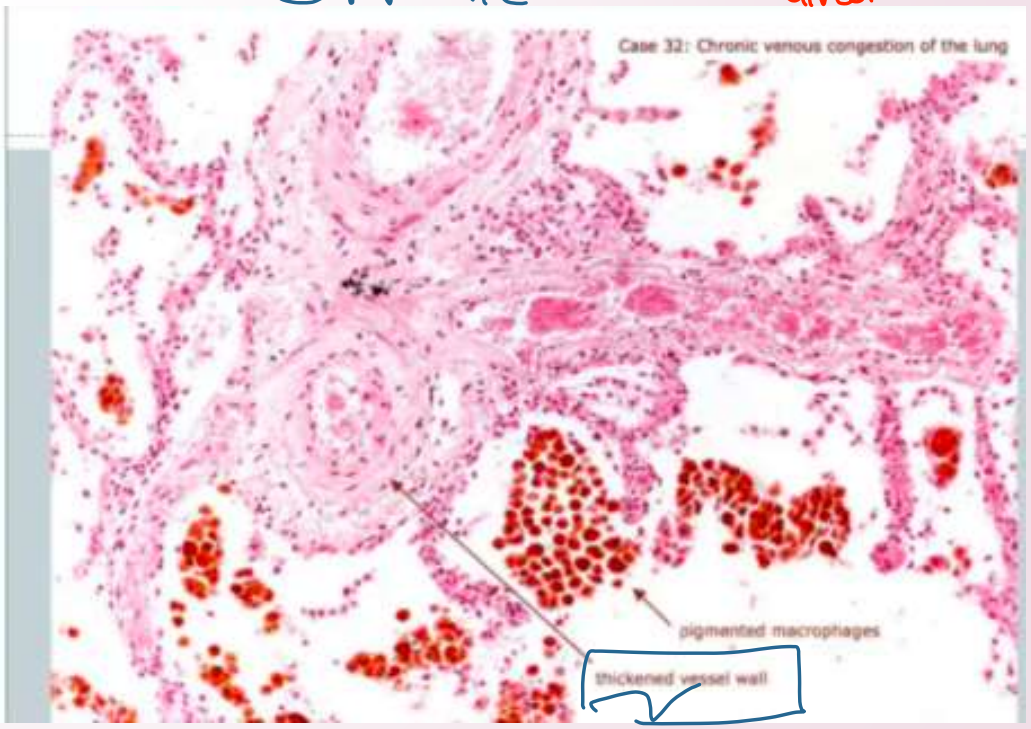
1. acute pulmonary congestion is marked by blood-engorged alveolar capillaries and
2. variable degrees of alveolar septal edema and intraalveolar hemorrhage.
3. (hemorrhage + RBCs inside the sacs.)

2. chronic pulmonary congestion, the septa become thickened and fibrotic, and the
2. alveolar spaces contain numerous macrophages laden with hemosiderin ("heart failure cells") derived from phagocytosed red cells.

acute

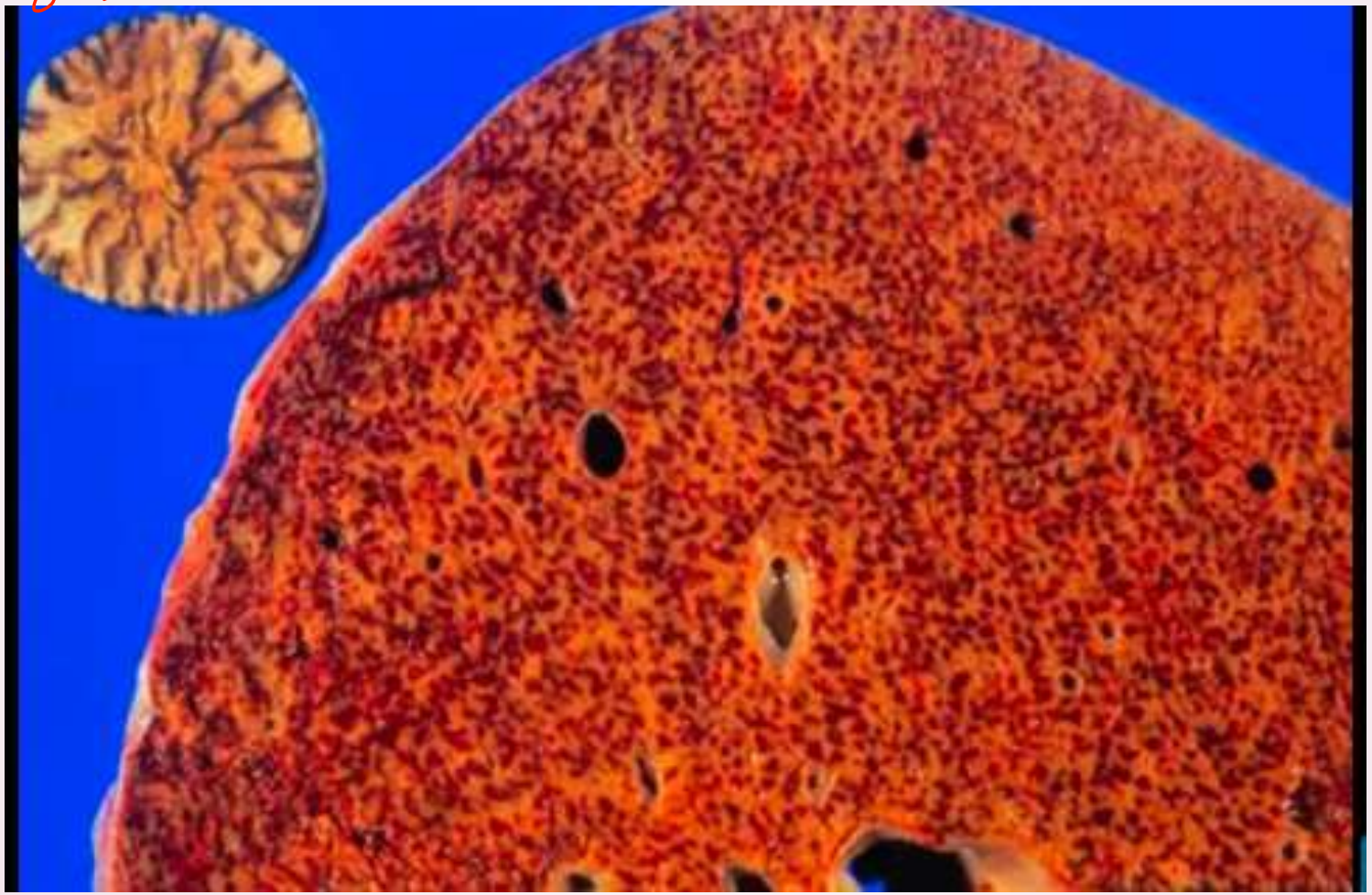


Chronic



II. HEPATIC CONGESTION.

central areas are red and slightly depressed compared with the surrounding tan viable parenchyma, creating “nutmeg liver”



centrally located hepatocytes are prone to necrosis more than t he periportal hepatocytes which is better oxygenated because of their proximity to hepatic arterioles

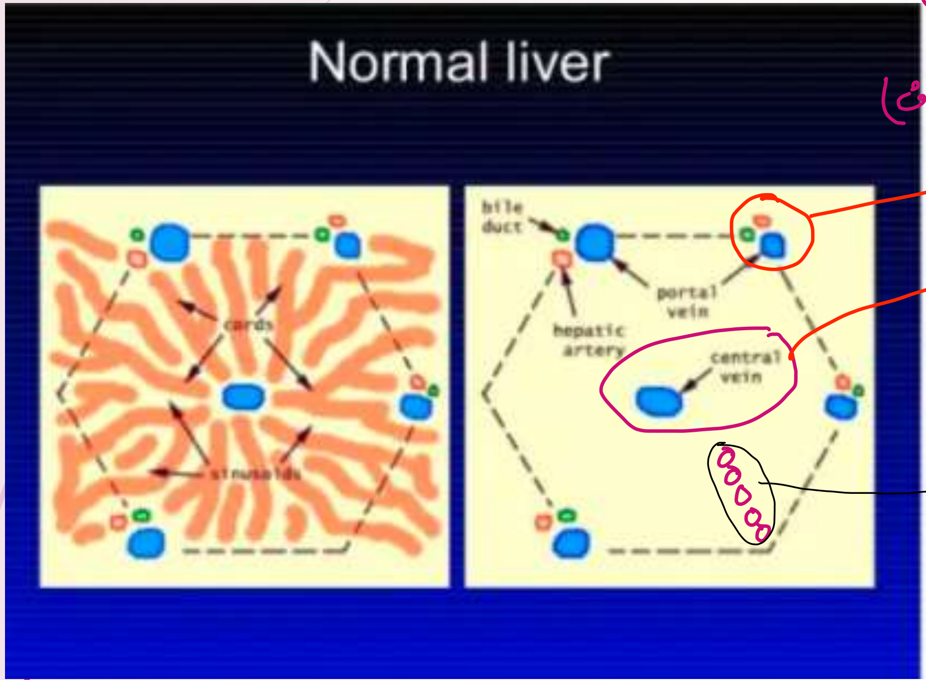
O_2 ↑↑

* Basic cells found in liver → hepatocyte

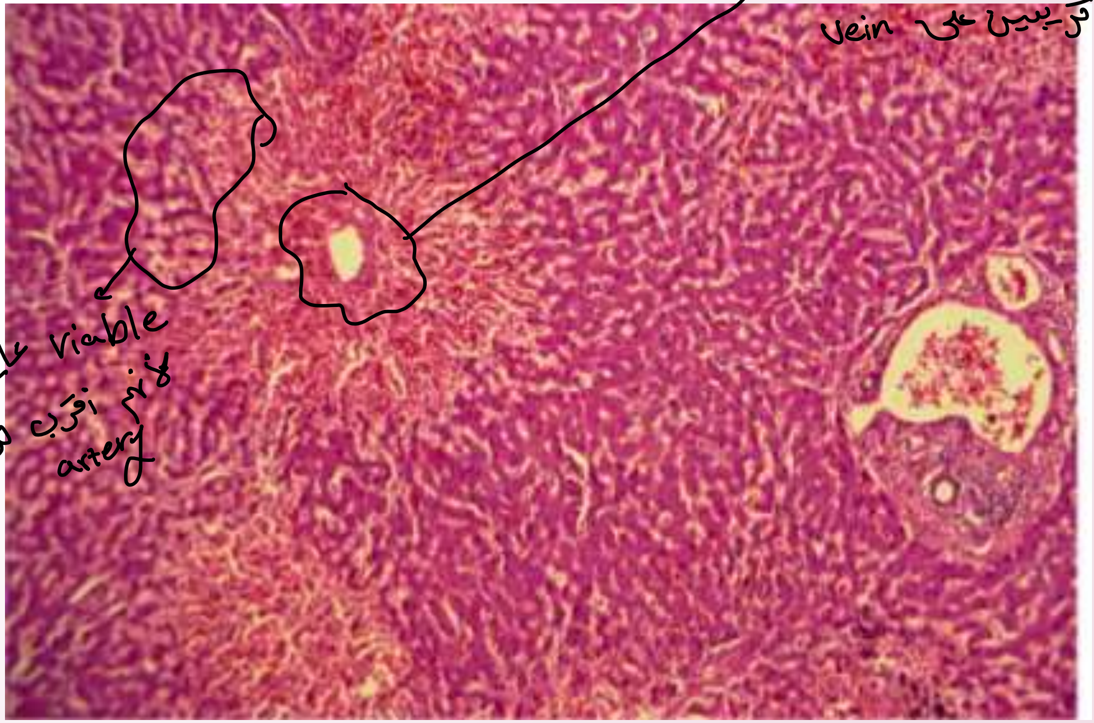
مرتبة جوا الـ liver ترتيب معين جوا → hexagonal unit (ست أطراف)

(على الأطراف) → portal triad (three structure inside this portal area) → 1- hepatic artery. 2- portal vein. 3- bile duct.

central vein (بالوسط)



خلايا الكبد مرتبة زي هيك تقريبا



necrotic hepatocyte
vein لأنه قريبين على

viable عايشين
لأنه أقرب للـ artery

(hepatocyte A) جالسة بجانب الـ central vein

(hepatocyte B) جالسة بجانب الـ portal triad

صار عندي (insult in liver) وصار عندي ischemia → أكثر خلية رح تتأثر بالـ ischemia → central one ← why?! لأنه موجود حوالين vein ورح يعاني لأنه ما في ٪ O_2 كافي

سؤال * Who are the hepatocyte that are more prone to have ischemia ?

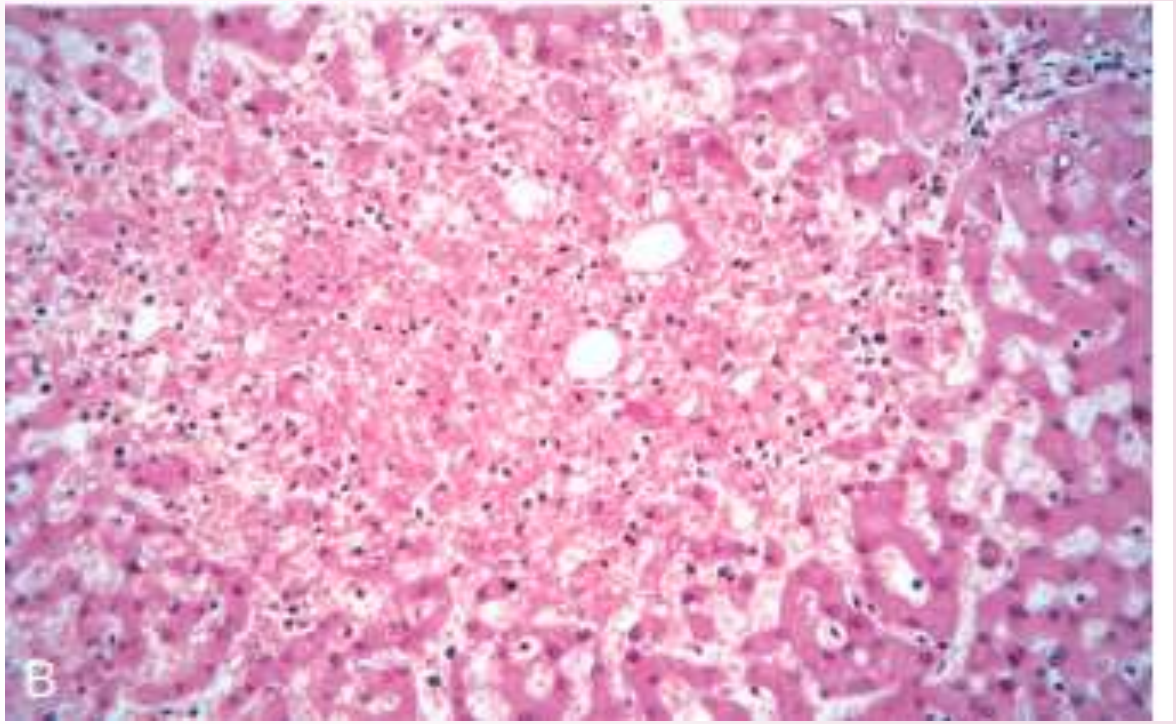
↳ the ones that are located near to central vein.

او periportal

يعني بشكل أقل

Microscopic findings include :

centrilobular hepatocyte necrosis.
Hemorrhage.
hemosiderin-laden macrophages



2. EDEMA (abnormal distribution)

إذا ال fluid طلع من جوا ال vessel برا

- Is an accumulation of interstitial fluid within **tissues** and **subcutaneously**.
- Extravascular fluid can also collect in **body cavities** and such accumulations are often referred to collectively as effusions.

- Examples include:
- effusions in the pleural cavity (hydrothorax). lung
- the pericardial cavity (hydropericardium).
- the peritoneal cavity (hydroperitoneum, or ascites). clinical term

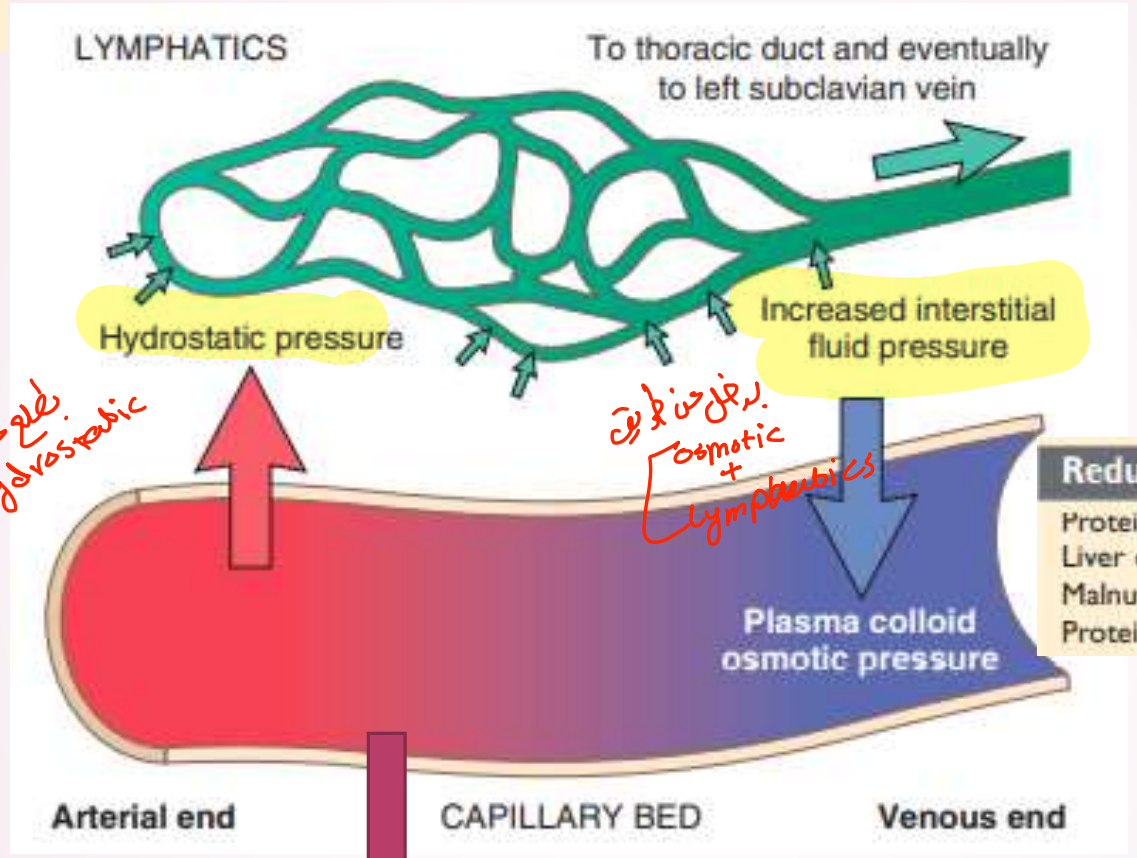
- Anasarca is severe, generalized edema marked by profound swelling of subcutaneous tissues and accumulation of fluid in body cavities. edema بكل جسم

Anasarca is a medical condition that leads to general swelling of the whole body

❖Causes of Edema

Lymphatic Obstruction

- Inflammatory
- Neoplastic
- Postsurgical
- Postirradiation



Reduced Plasma Osmotic Pressure (Hypoproteinemia)

- Protein-losing glomerulopathies (nephrotic syndrome)
- Liver cirrhosis (ascites)
- Malnutrition
- Protein-losing gastroenteropathy GIT

Arteriolar Dilation

- Heat
- Neurohumoral dysregulation

Sodium Retention

- Excessive salt intake with renal insufficiency
- Increased tubular reabsorption of sodium
- Renal hypoperfusion
- Increased renin-angiotensin-**aldosterone** secretion

Impaired Venous Return

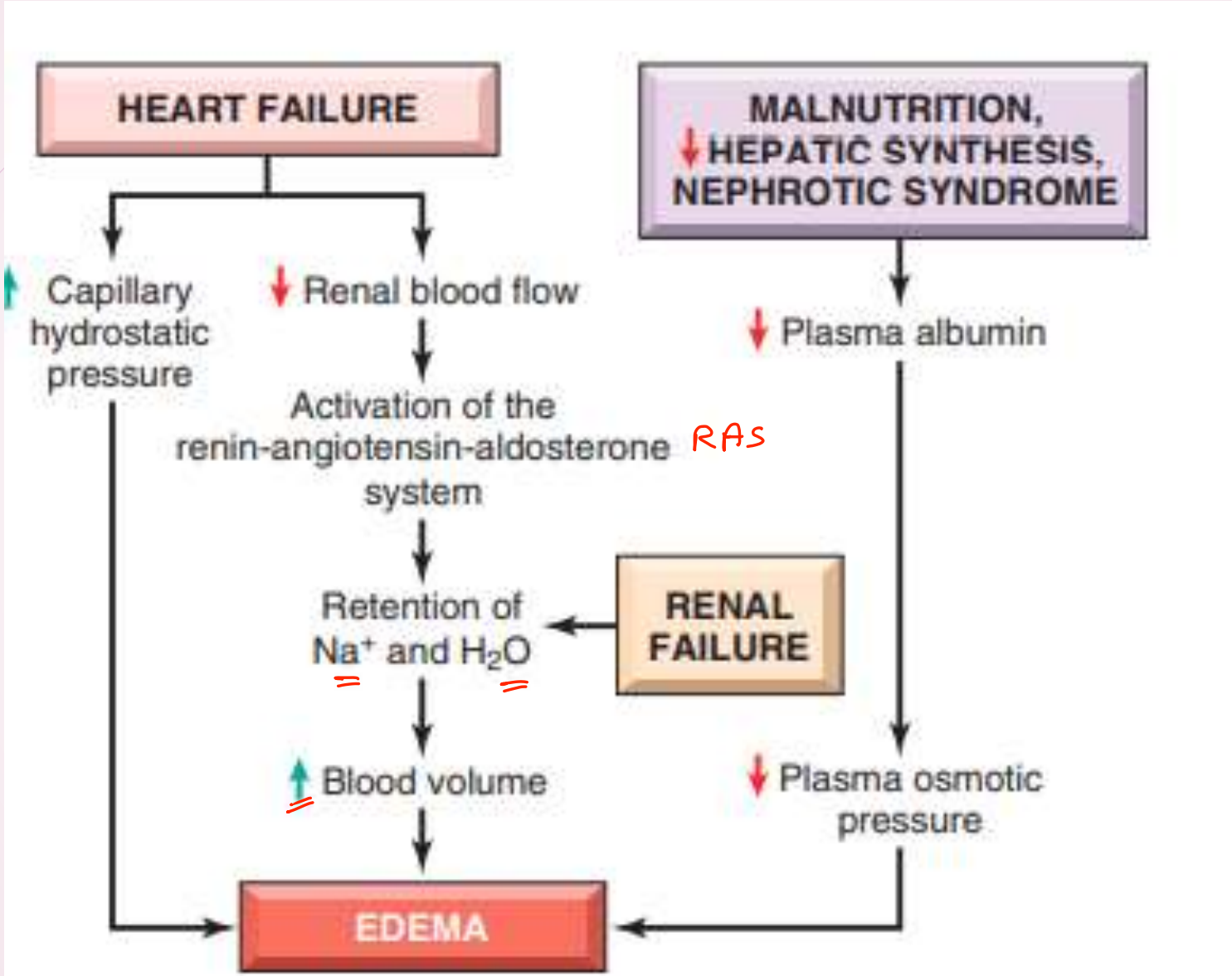
- Congestive heart failure
- Constrictive pericarditis → No adequate pumping
- Ascites (liver cirrhosis)
- Venous obstruction or compression
- Thrombosis
- External pressure (e.g., mass)
- Lower extremity inactivity with prolonged dependency

Table 4.1 Causes of Edema

Increased Hydrostatic Pressure
Impaired Venous Return
Congestive heart failure
Constrictive pericarditis
Ascites (liver cirrhosis)
Venous obstruction or compression
Thrombosis
External pressure (e.g., mass)
Lower extremity inactivity with prolonged dependency
Arteriolar Dilation
Heat
Neurohumoral dysregulation
Reduced Plasma Osmotic Pressure (Hypoproteinemia)
Protein-losing glomerulopathies (nephrotic syndrome)
Liver cirrhosis (ascites)
Malnutrition
Protein-losing gastroenteropathy
Lymphatic Obstruction
Inflammatory
Neoplastic
Postsurgical
Postirradiation
Sodium Retention
Excessive salt intake with renal insufficiency
Increased tubular reabsorption of sodium
Renal hypoperfusion
Increased renin-angiotensin-**aldosterone** secretion
Inflammation
Acute inflammation
Chronic inflammation
Angiogenesis

Mechanisms of edema

- **1. Increased Hydrostatic Pressure:**
- Increases in hydrostatic pressure are mainly caused by disorders that impair venous return, either :
- Localized: e.g deep venous thrombosis.
- Generalized increases in venous pressure: e.g congestive heart failure.


HEART FAILURE
MALNUTRITION,
HEPATIC SYNTHESIS,
NEPHROTIC SYNDROME
Capillary hydrostatic pressure
Renal blood flow
Plasma albumin
Activation of the renin-angiotensin-aldosterone system
RAS
Retention of Na+ and H2O
RENAL FAILURE
Blood volume
Plasma osmotic pressure
EDEMA

2. **Reduced Plasma Osmotic Pressure**

- Reduction of plasma albumin concentrations leads to decreased colloid osmotic pressure of the blood and loss of fluid from the circulation.
- albumin accounts for almost half of the total plasma protein.
- common causes of reduced plasma osmotic pressure:
  - lost from the circulation: e.g Nephrotic syndrome renal disease
  - synthesis of inadequate amounts: e.g severe liver disease (e.g., cirrhosis) and protein malnutrition.

3. Lymphatic Obstruction

- Edema may result from lymphatic obstruction that compromises resorption of fluid from interstitial space.

- results from a localized obstruction caused by an inflammatory or neoplastic condition.

Infiltration and obstruction of superficial lymphatics by breast cancer may cause edema of the overlying skin; the characteristic finely pitted appearance of the skin of the affected breast is called peau d'orange (orange peel).

Not only tumors

the parasitic infection filariasis can cause massive edema of the lower extremity and external genitalia (so-called "elephantiasis.

lymphatic obstruction

4. Sodium and Water Retention

- Excessive retention of salt lead to edema by increasing hydrostatic pressure (because of expansion of the intravascular volume) and reducing plasma osmotic pressure.
- Excessive salt and water retention are seen in a wide variety of diseases that compromise renal function, including poststreptococcal glomerulonephritis and acute renal failure.

- Subcutaneous edema :
- can be diffuse but usually accumulates preferentially in the legs with standing and the sacrum with recumbency, a relationship termed dependent edema.
- Finger pressure over edematous subcutaneous tissue displaces the interstitial fluid, leaving a finger-shaped depression; this appearance is **called pitting edema.**
- **Under microscope:** skin shows clearing and separation of the extracellular matrix

Edema is easily recognized on gross inspection;

unilateral (parasitic)

Bilateral (systimatic condition like heart failure)

- Edema resulting from renal dysfunction or nephrotic syndrome often manifests first in loose connective tissues (e.g., the eyelids, causing periorbital edema).

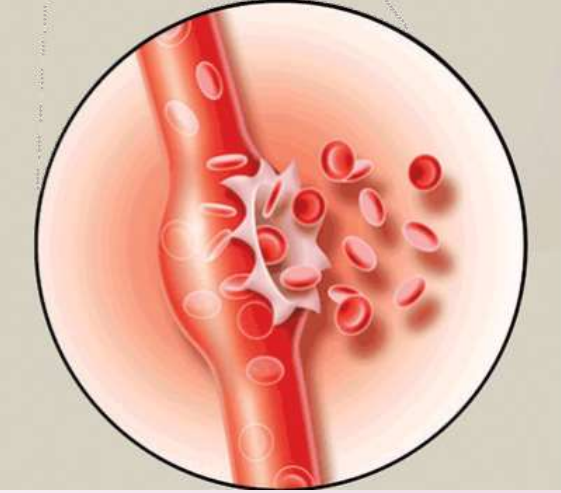
Clinical Features

- **Subcutaneous edema :**
  - is important to recognize primarily because it signals potential underlying cardiac or renal disease.
  - when significant, it also can impair wound healing and the clearance of infections.
- **Pulmonary edema:**
  - It can cause death by interfering with normal ventilatory function; besides impeding oxygen diffusion, alveolar edema fluid also creates a favorable environment for infections..

Brain edema:

- Is life threatening; if the swelling is severe, the brain can herniate (extrude) through the foramen magnum pressure, the brain stem vascular supply can be compressed, leading to death due to injury to the medullary centers controlling respiration and other vital functions .

II. HEMORRHAGE

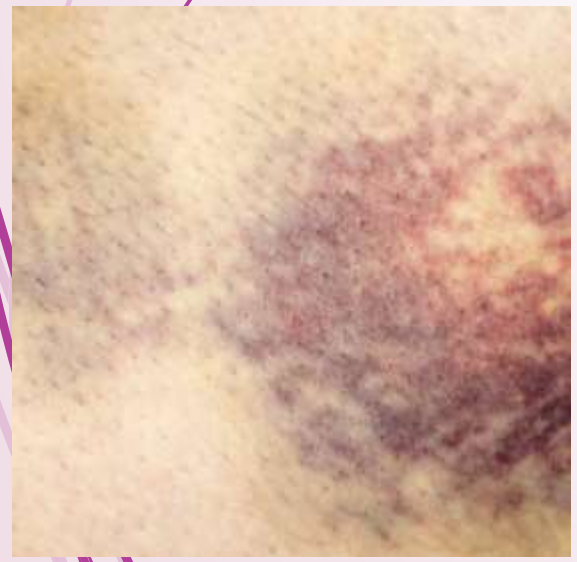


- extravasation of blood from vessels, is most often the result of damage to blood vessels or defective clot formation.
- Trauma, atherosclerosis, or inflammatory or neoplastic erosion of a vessel wall also may lead to hemorrhage,

- hemorrhagic diatheses: tendency to have bleeding than others.

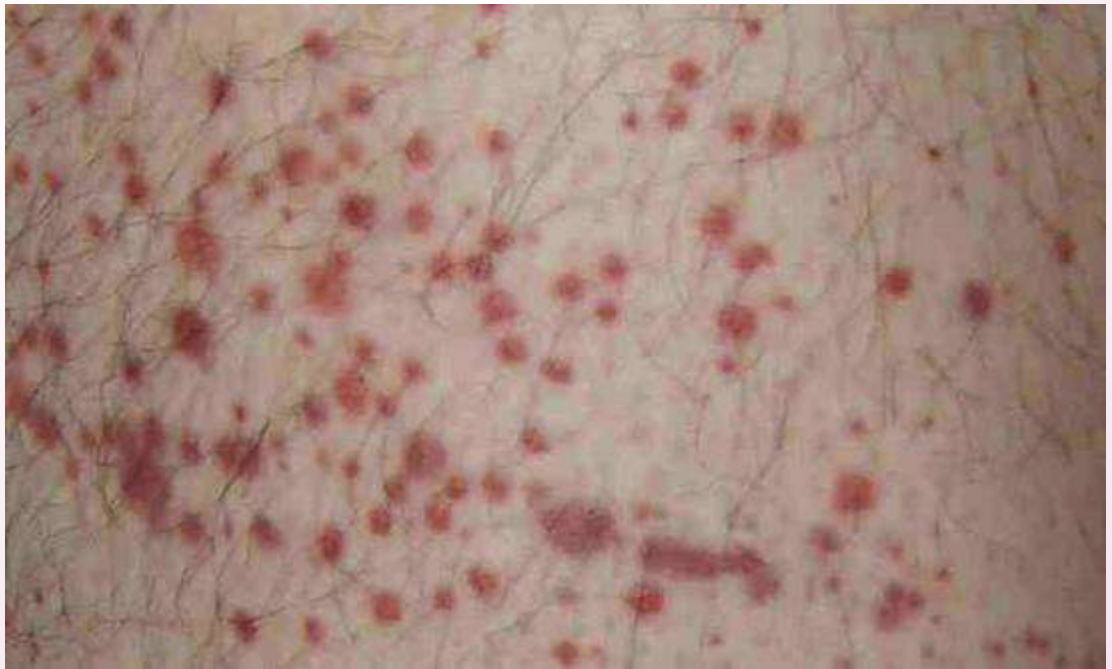
- **Hemorrhage may be manifested by different appearances and clinical consequences**.

- Hemorrhage may be external or accumulate within a tissue as a hematoma,
- May ranges in significance from trivial (e.g., a bruise) to fatal (e.g., a massive retroperitoneal hematoma resulting from rupture of a dissecting aortic aneurysm.
- Extensive hemorrhages can occasionally result in jaundice from the massive breakdown of red cells and hemoglobin.



Subcutaneous bleeding my present as

- **1. Petechiae :**
- are minute (1 to 2 mm in diameter) hemorrhages into skin, mucous membranes, or serosal surfaces
- Causes
  1. low platelet counts (thrombocytopenia).
  2. defective platelet function.
  3. loss of vascular wall support, as in vitamin C deficiency.



2. Purpura

- are slightly larger (3 to 5 mm) hemorrhages.
- Purpura can result from the same disorders that cause petechiae, as well as:
- trauma.
- vascular inflammation (vasculitis).
- increased vascular fragility.

3.Ecchymoses:

- are larger (1 to 2 cm) subcutaneous hematomas (also called bruises).
- Extravasated red cells are phagocytosed and degraded by macrophages; the characteristic color changes of a bruise result from the enzymatic conversion of hemoglobin (red-blue color) to bilirubin (blue-green color) and eventually hemosiderin (golden-brown)

- The clinical significance of any particular hemorrhage depends on:
  - ✓ ① the volume of blood that is lost.
  - ✓ ② the rate of bleeding.