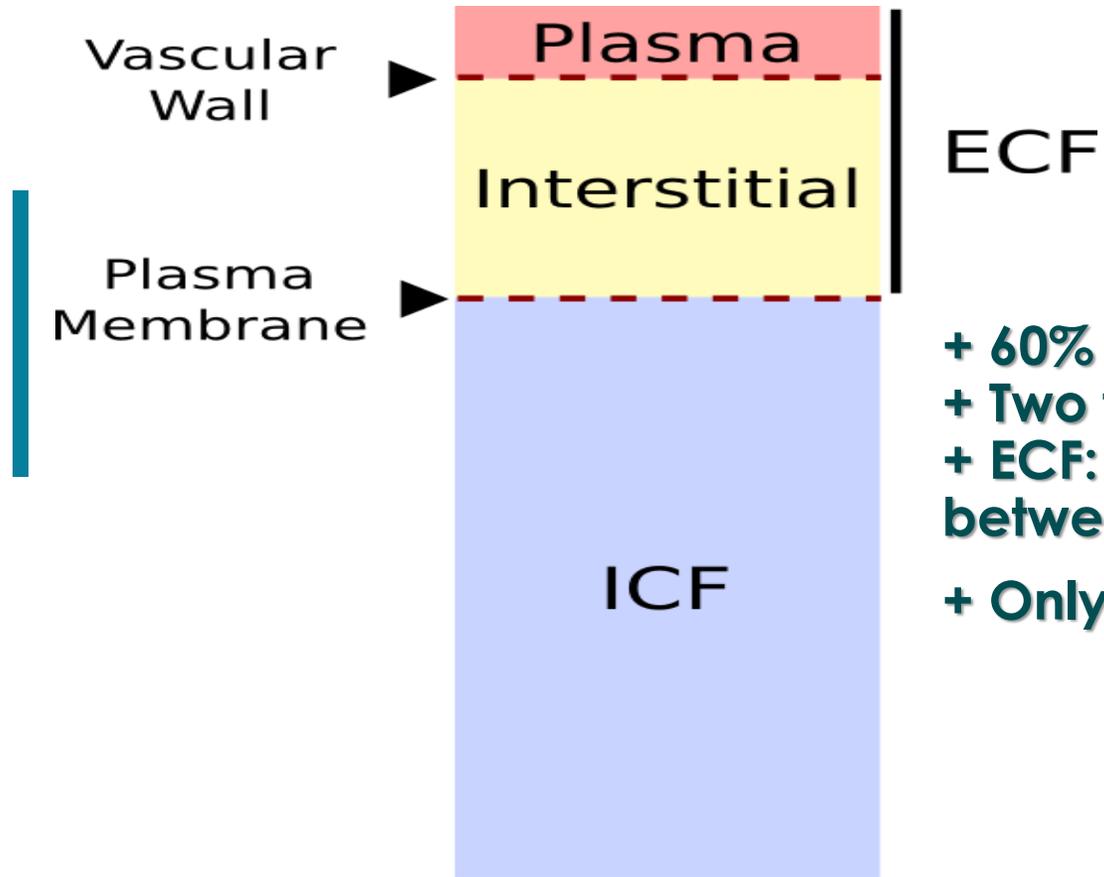




Hemodynamic Disorders 1

Edema, hyperemia and congestion

Ghadeer Hayel, M.D.
Assistant Professor of Pathology
Consultant Hematopathologist
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- + 60% of lean body weight is water.
- + Two thirds is intracellular.
- + ECF: most of the extracellular fluid is in the that lies between cells
- + Only 5% of the body's water is in blood plasma.

Edema

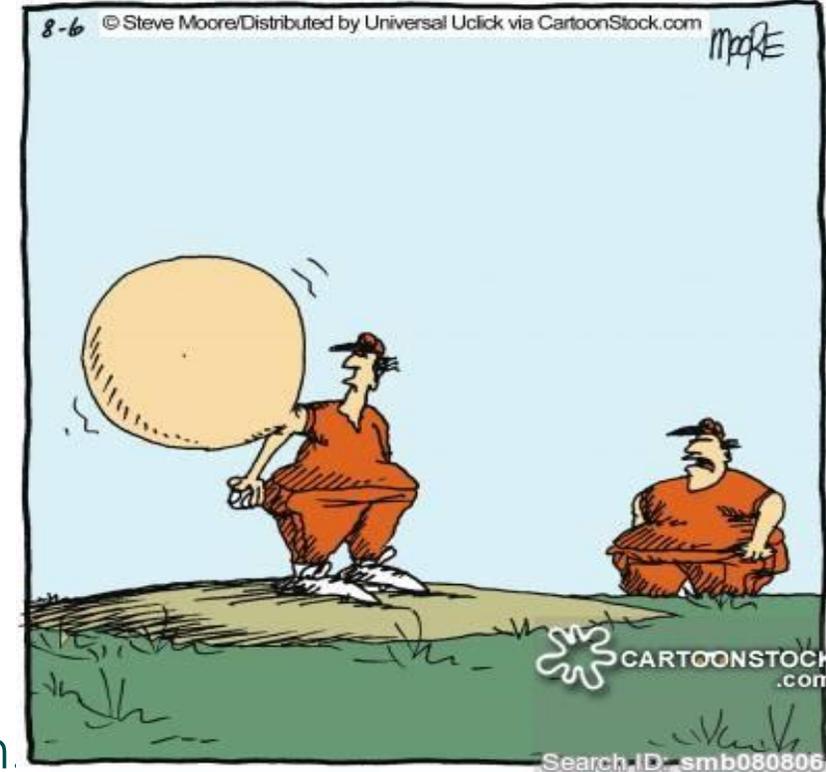
- + Localized or generalized accumulation of fluid in interstitial spaces.
- + Extravascular fluid can also collect in body cavities & such accumulations are often referred to as effusions.

Effusions into body cavities:

Hydrothorax: within thorax, around lungs; also pleural effusion.

Hydropericardium: Fluid in the pericardial sac.

Hydroperitoneum or ascites: Fluid in the peritoneal cavity.

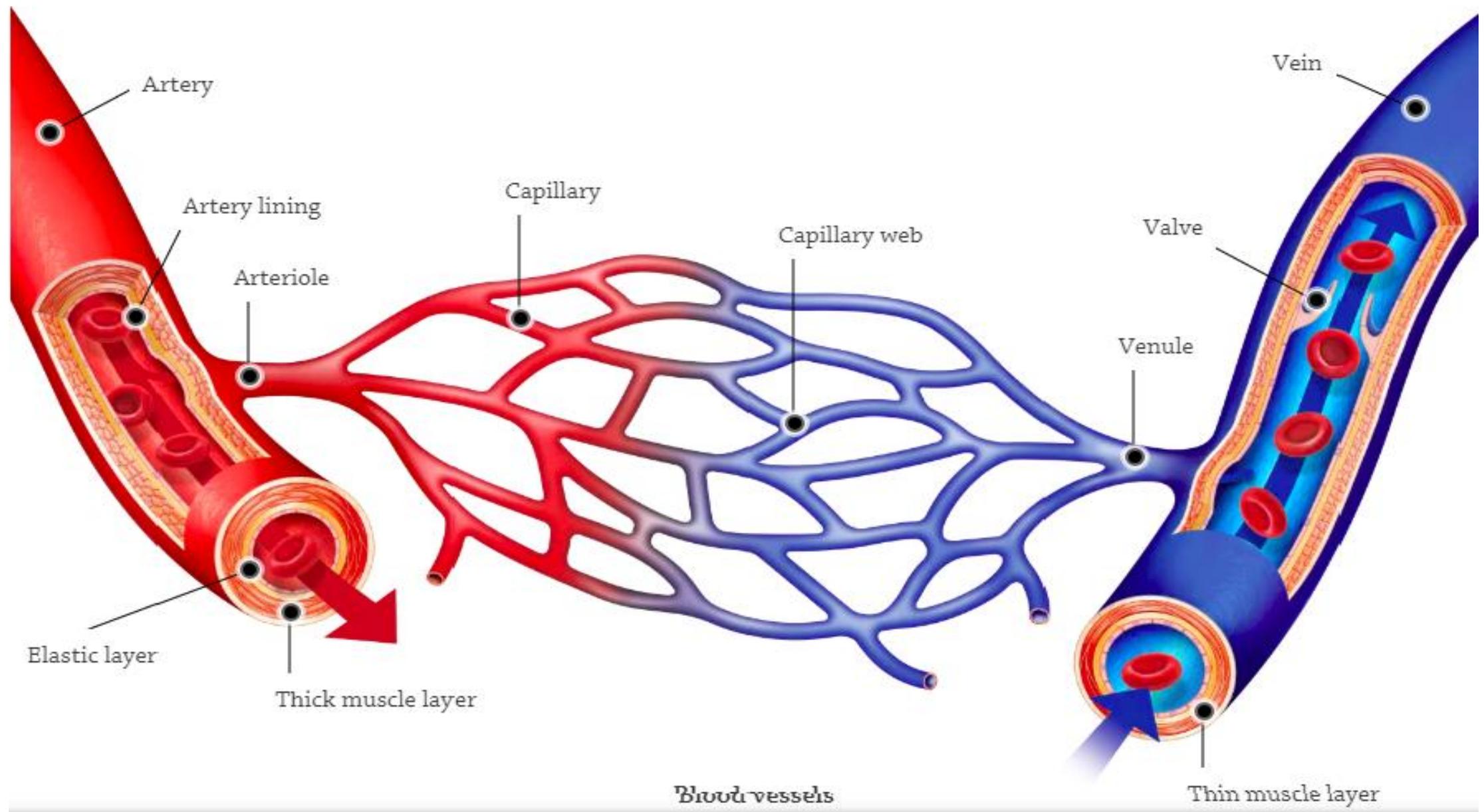


"Swelling, Babcock, is our body's way of telling us that something's wrong. But if you insist on staying in the game, hey, it's your elbow."

Anasarca

- severe, generalized edema
- *ana*= throughout, *sark*= flesh
- Most commonly used to describe fetal or neonatal whole-body, subcutaneous swelling.





Normally..

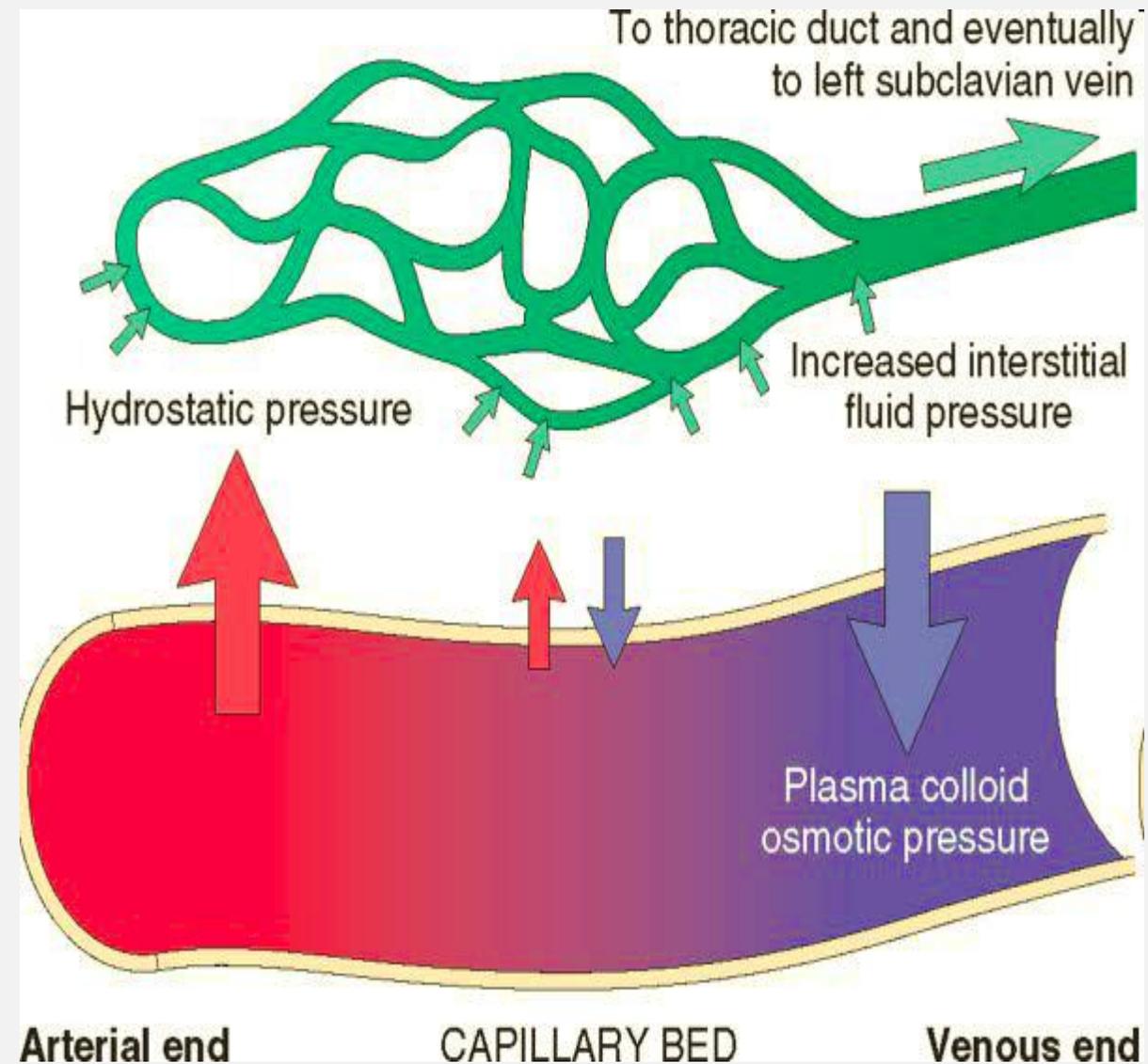
Fluid movement between the vascular & interstitial spaces is governed mainly by two opposing forces:

+Vascular hydrostatic pressure: the blood pushing against the walls of the capillaries:

- * Higher on arteriolar side
- * Lower on venular side
- * Lowest in interstitium

+Colloid osmotic pressure: produced by plasma proteins (mainly albumin)

Only a small net outflow of fluid into the interstitial space, which is drained by lymphatic vessels.



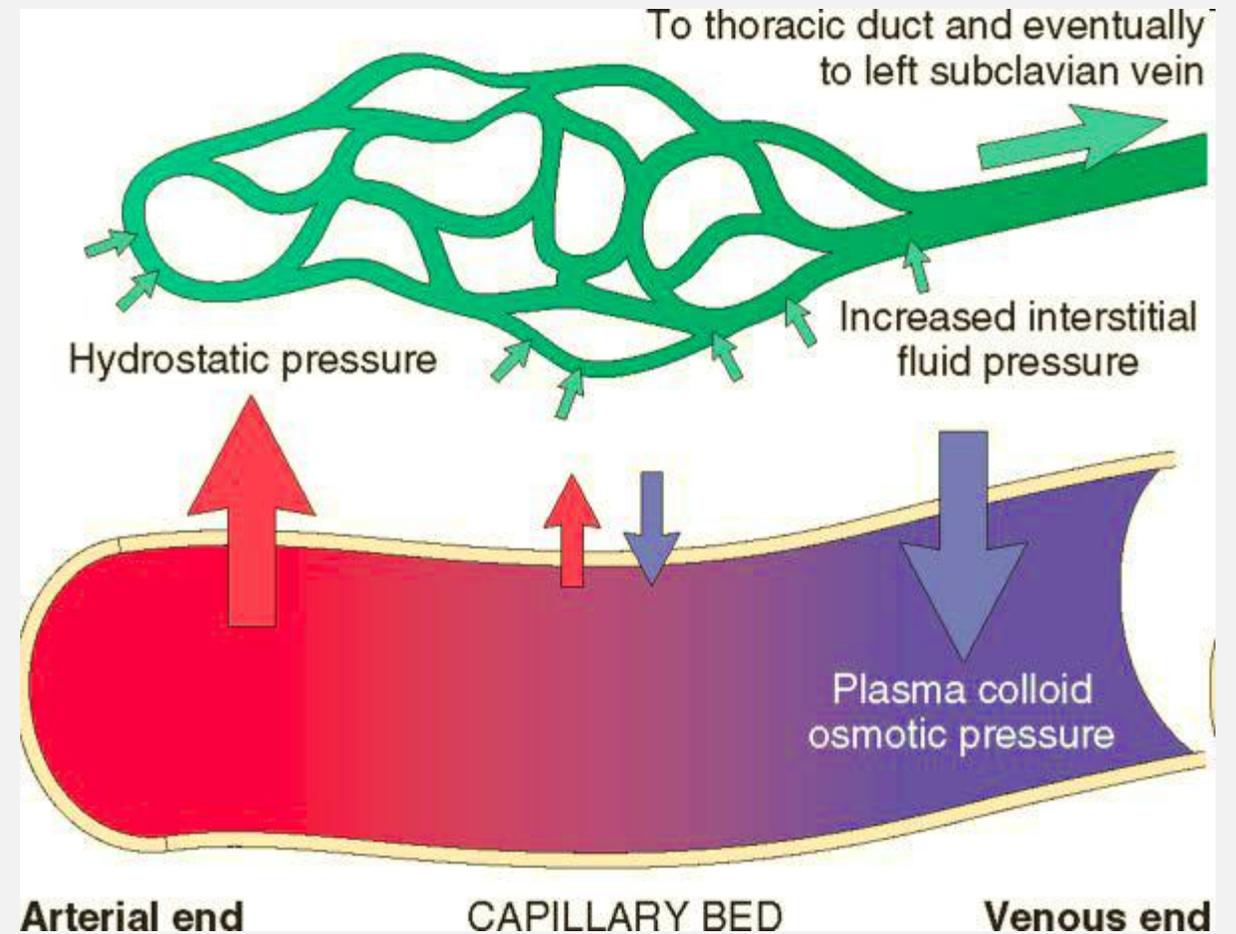
In edema/effusion..

- Either increased hydrostatic pressure or diminished colloid osmotic pressure causes increased movement of water into the interstitium.

- Extravascular fluid collections can be classified as follows:

+Transudate: poor in protein and/or cells, fluid grossly clear, low specific gravity (<1.012). Increased hydrostatic pressure or reduced intravascular colloid pressure.

+Exudate: rich in protein and/or cells, fluid grossly cloudy, high specific gravity (>1.012). Inflammatory edema (increased vascular permeability).



Main Causes of edema..

Increased Hydrostatic Pressure

Impaired Venous Return

Congestive heart failure

Constrictive pericarditis

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)

Reduced protein synthesis (e.g., advanced liver disease)

Malnutrition

Protein-losing gastroenteropathy

Lymphatic Obstruction

Inflammatory

Neoplastic

Postsurgical

Postirradiation

Sodium Retention

Excessive salt intake with renal insufficiency

Decreased renal excretion of sodium

Renal hypoperfusion

Increased renin-angiotensin-aldosterone secretion

Inflammation

Acute inflammation

Chronic inflammation

Angiogenesis

We will discuss the following causes of edema..



- 1- Increased Hydrostatic Pressure.
- 2- Reduced Plasma Osmotic Pressure.
- 3- Lymphatic Obstruction.
- 4- Sodium and Water Retention

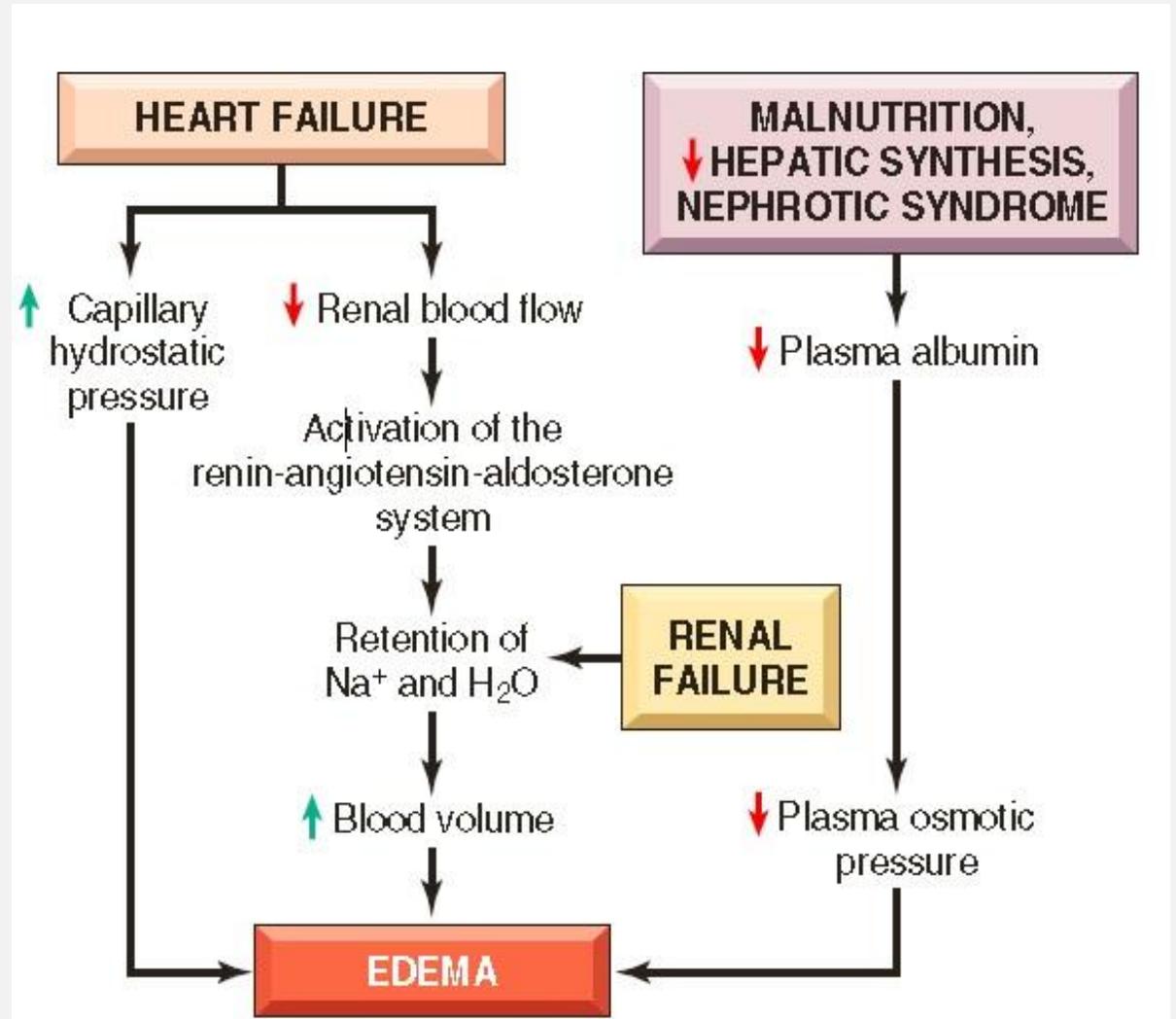
Increased Hydrostatic Pressure.

- mainly caused by disorders that impair venous return.

1. Localized: Deep vein thrombosis (DVT), Localized edema in affected leg.

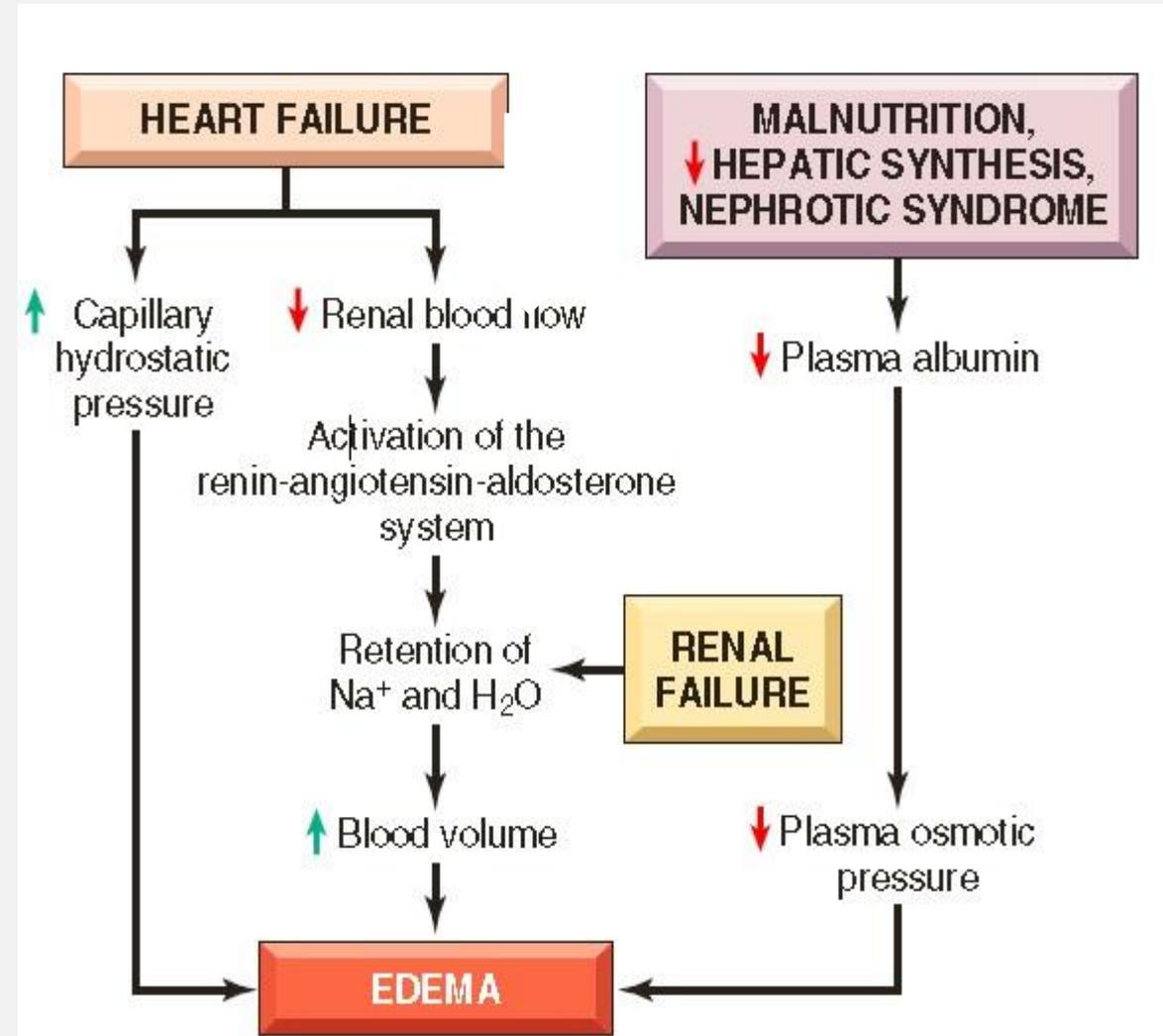
2. Generalized: Congestive heart failure (CHF) → Systemic Edema.

- Unless cardiac output is restored or renal water retention is reduced (e.g., by salt restriction or treatment with diuretics or aldosterone antagonists) this downward spiral continues.



Reduced Plasma Osmotic Pressure.

- Reduction in plasma albumin concentrations → ↓↓ colloid osmotic pressure of the blood & loss of fluid from the circulation.
- Albumin accounts for almost half of the total plasma protein.
- common causes:
 - 1) albumin is **lost** from the circulation (**nephrotic syndrome**).
 - 2) albumin synthesized in **inadequate** amounts (**severe liver disease & malnutrition**)
- Unfortunately, increased salt & water retention by the kidney fails to correct the plasma volume deficit & worsen edema, because the primary defect → low serum protein, persists.



Lymphatic Obstruction

- Lymphatic obstruction can compromise resorption/clearance of fluid from interstitial spaces.
 - Trauma, fibrosis, invasive tumors, & infectious agents can all disrupt lymphatic vessels
- + **Filariasis:** A parasitic infection that can cause massive edema of the lower extremity (**elephantiasis**), & external genitalia by producing inguinal lymphatic & lymph node **fibrosis**.



Lymphatic Obstruction

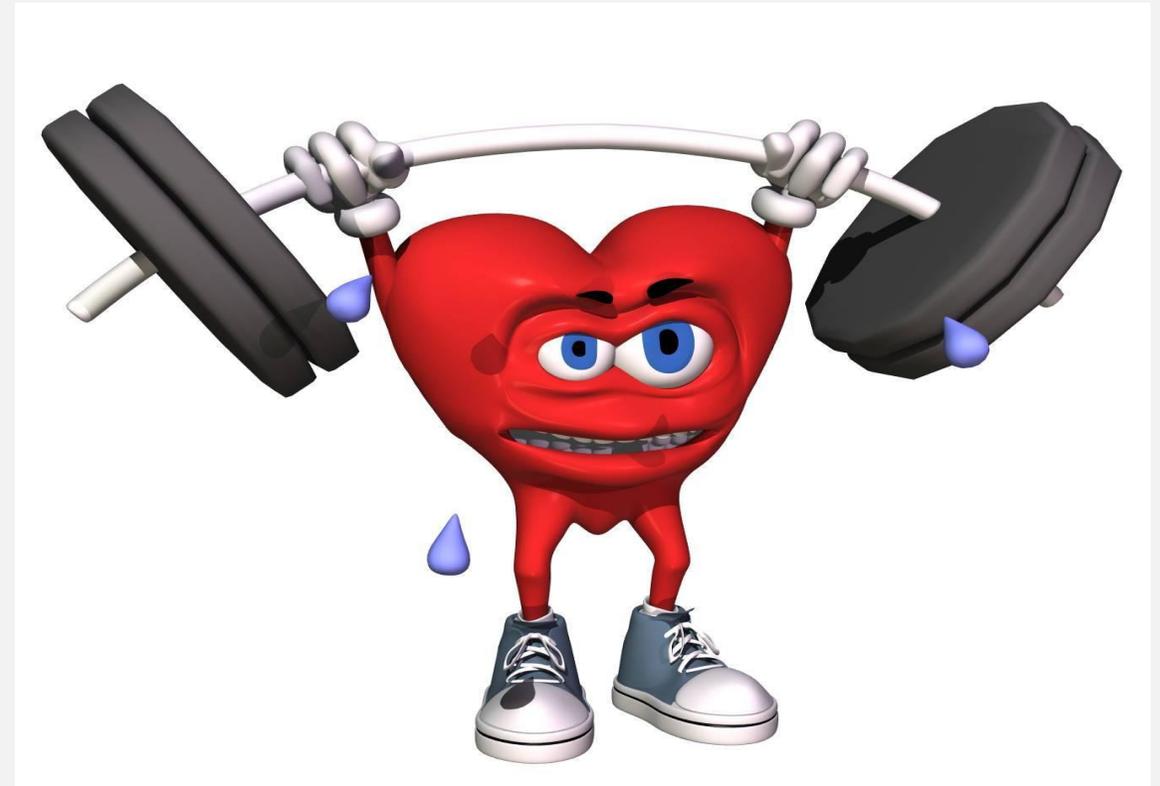
+ Infiltration & obstruction of superficial lymphatics by breast cancer may cause edema of the overlying skin; finely pitted appearance of skin of **peau d'orang**.

+ Women with breast cancer who undergo axillary lymph node resection and/or irradiation-both of can disrupt & obstruct lymphatic drainage, resulting in severe lymphedema of the arm.



Sodium and Water Retention

- Excessive retention of salt (& its obligate associated water) can lead to edema by increasing hydrostatic pressure (because of water cause expansion of the intravascular volume) and reducing plasma osmotic pressure.
- Conditions that compromise renal function, including **poststreptococcal glomerulonephritis & acute renal failure**



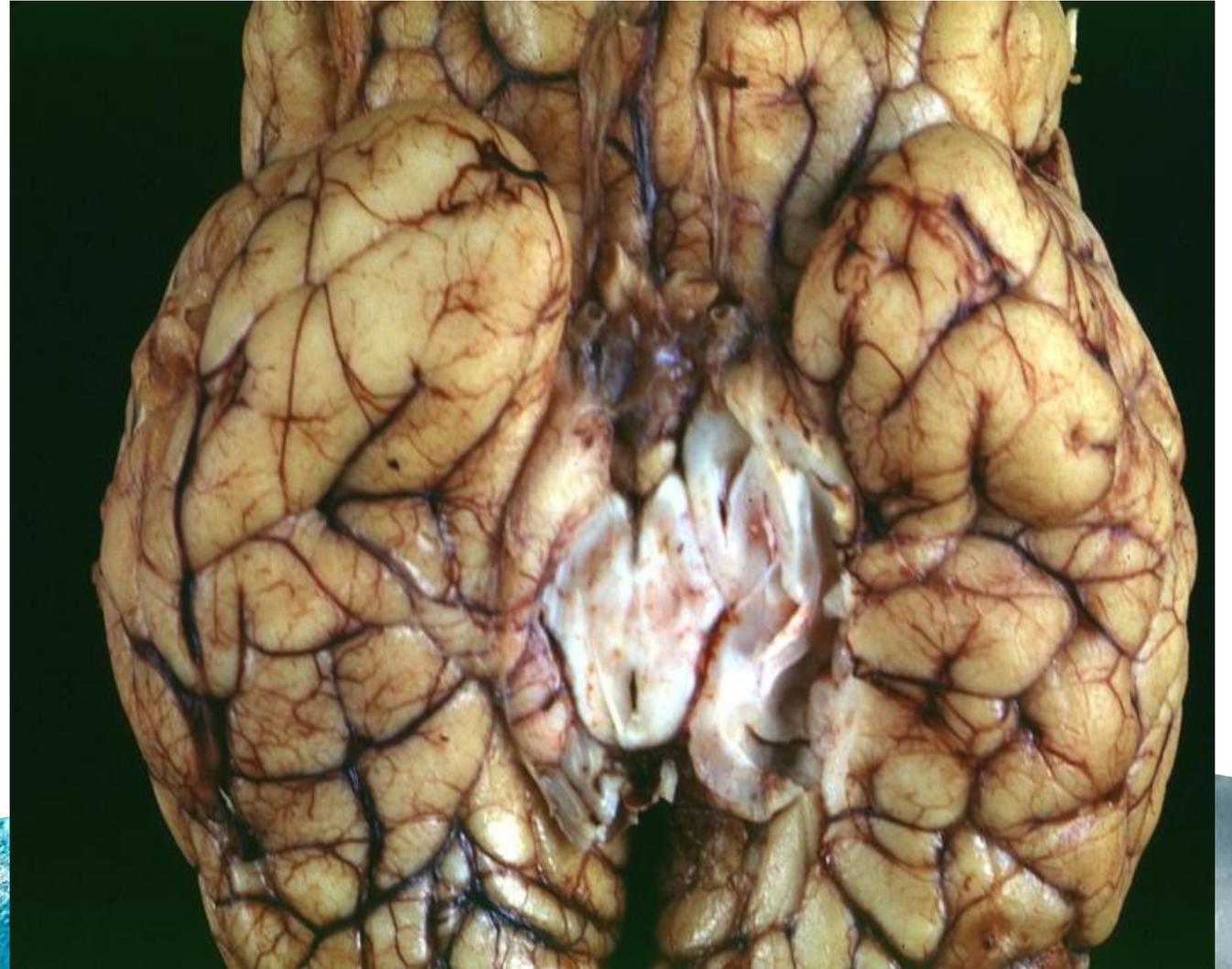
- **Subcutaneous edema** can be diffuse or localized.
- More in parts of the body positioned the greatest distance below the heart, where hydrostatic pressures are highest. **Most pronounced in the legs with standing and the sacrum with recumbency (dependent edema)**
- Finger pressure over edematous subcutaneous tissue leaving a finger-shaped depression; **pitting edema**.
- Edema from **renal dysfunction** or **nephrotic syndrome**
- often manifests first in parts with loose connective tissues (e.g., the eyelids (**periorbital edema**)).



+ **pulmonary edema:** the lungs often are two to three times their normal weight, sectioning shows frothy, or blood-tinged fluid (mix of air, edema fluid, extravasated red cells).

+ **Brain edema** can be localized (abscess or tumor) or generalized, depending on the pathologic process or injury.

With generalized edema, the **sulci are narrowed as the gyri swell and become flattened against the skull.**



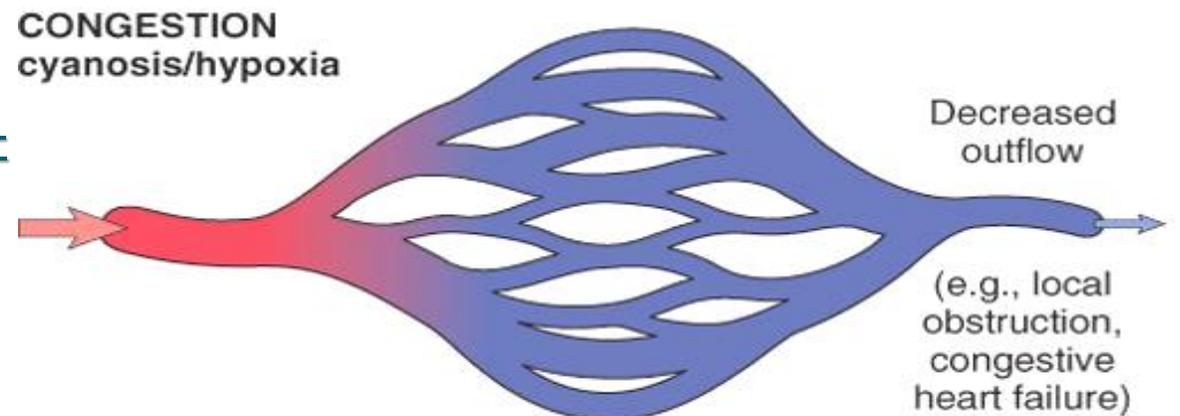
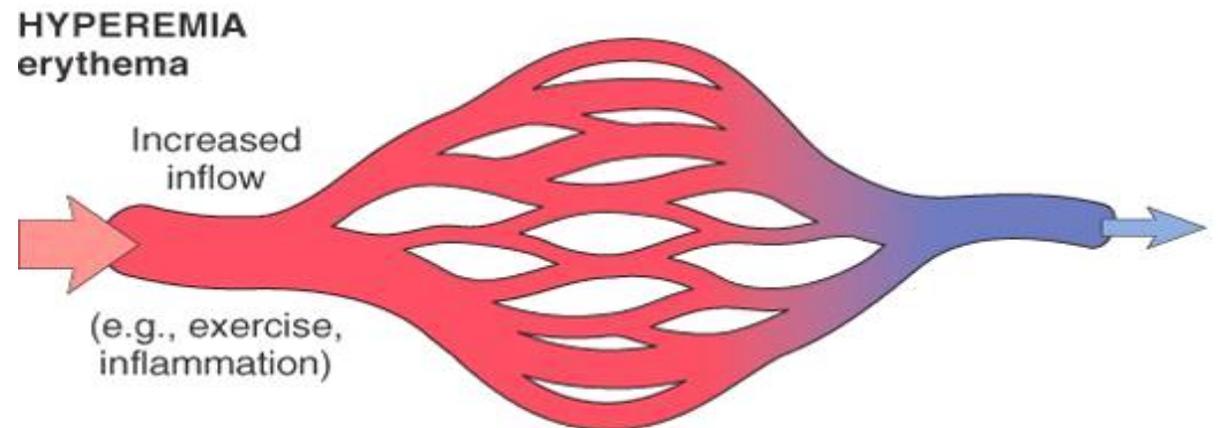
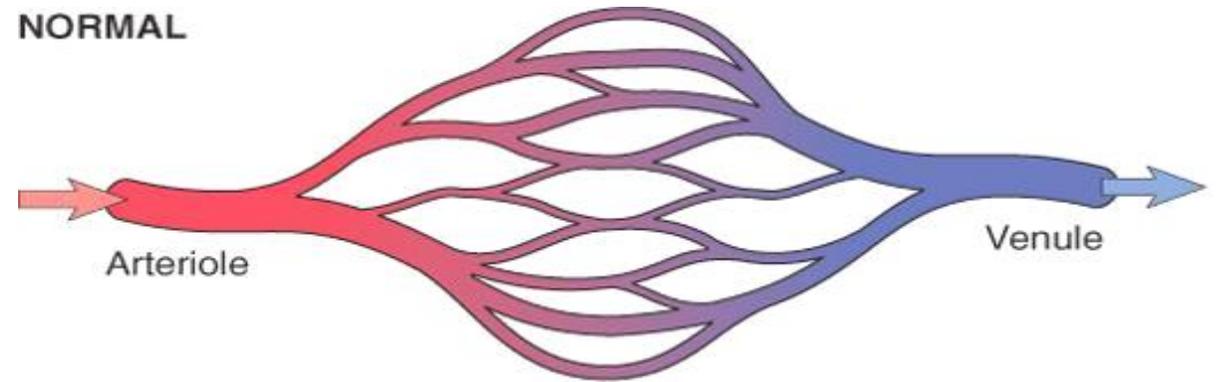


HYPEREMIA AND CONGESTION

Both refer to an **increase in blood volume within a tissue** but have different underlying mechanisms & consequences.

+ **Hyperemia** is an active process → arteriolar dilation & increased blood inflow, (in inflammation or in exercising skeletal muscle), tissues are redder than normal coz of oxygenated blood.

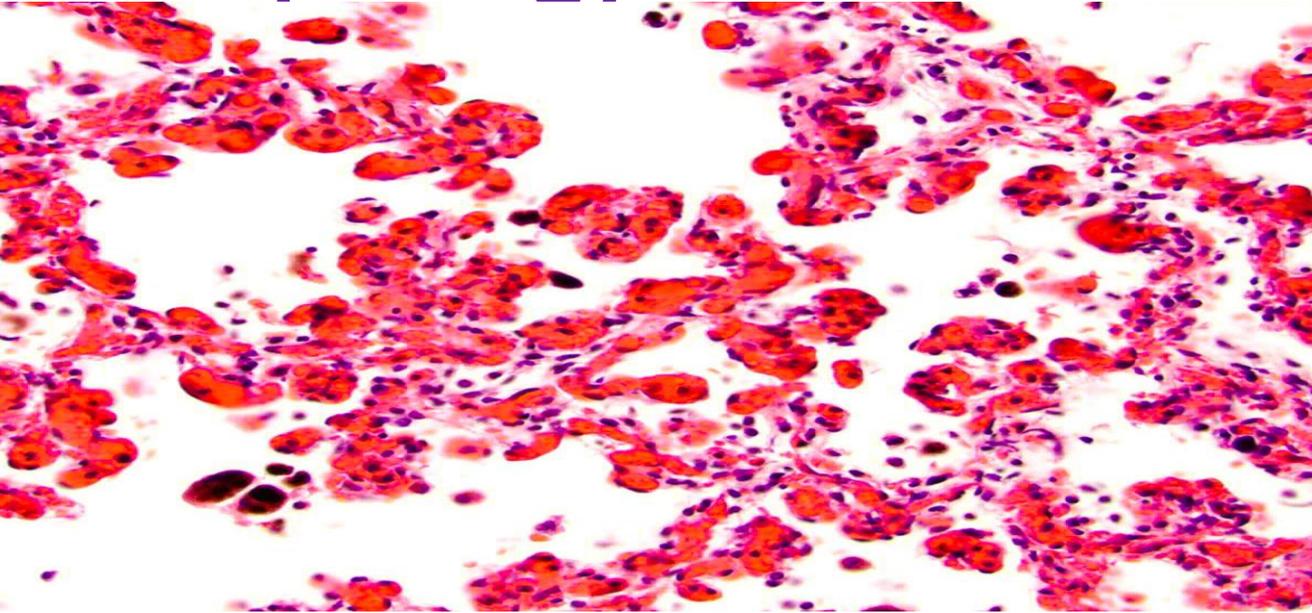
+ **Congestion** is a passive process → impaired outflow of venous blood from a tissue. It can occur systemically (cardiac failure) or locally (isolated venous obstruction), tissues have an abnormal blue-red color (**cyanosis**) due to accumulation of deoxygenated hemoglobin.



Complications of long standing chronic congestion

- **Long standing congestion** → **Stasis of poorly oxygenated blood** → **chronic hypoxia** → **death of cells** → **Fibrosis**
- **Increased capillary pressure** → **rupture** → **Foci of hemorrhages** → **hemosiderin laden macrophages.**

Morphology

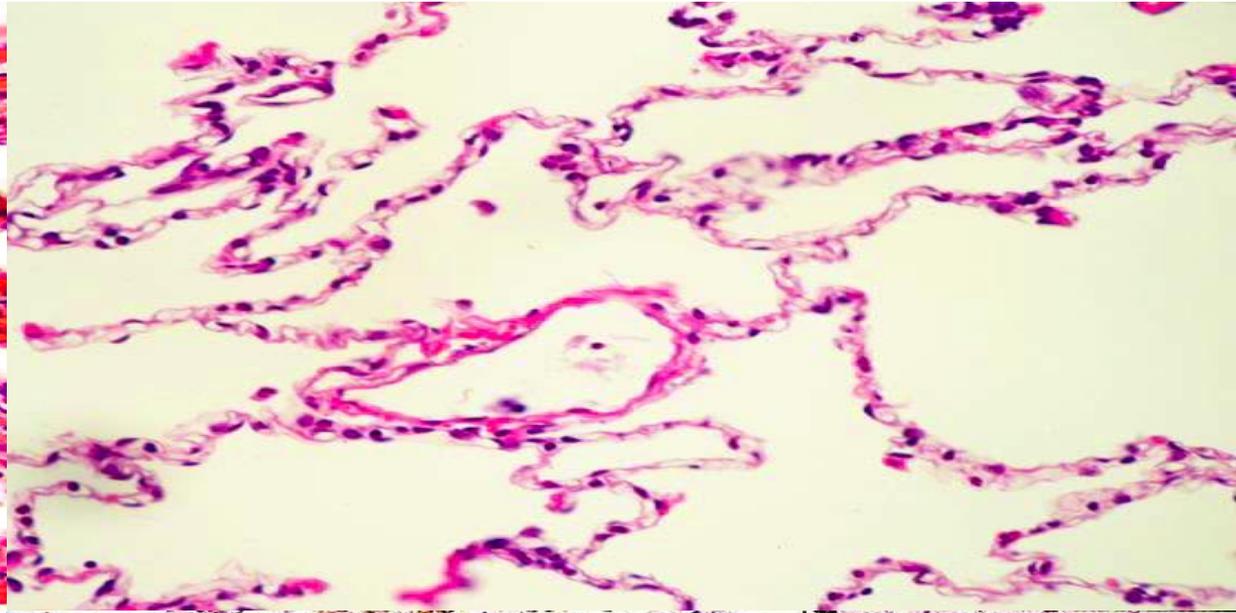


Acute pulmonary congestion

Marked blood-engorged alveolar capillaries.

variable degrees of alveolar septal edema.

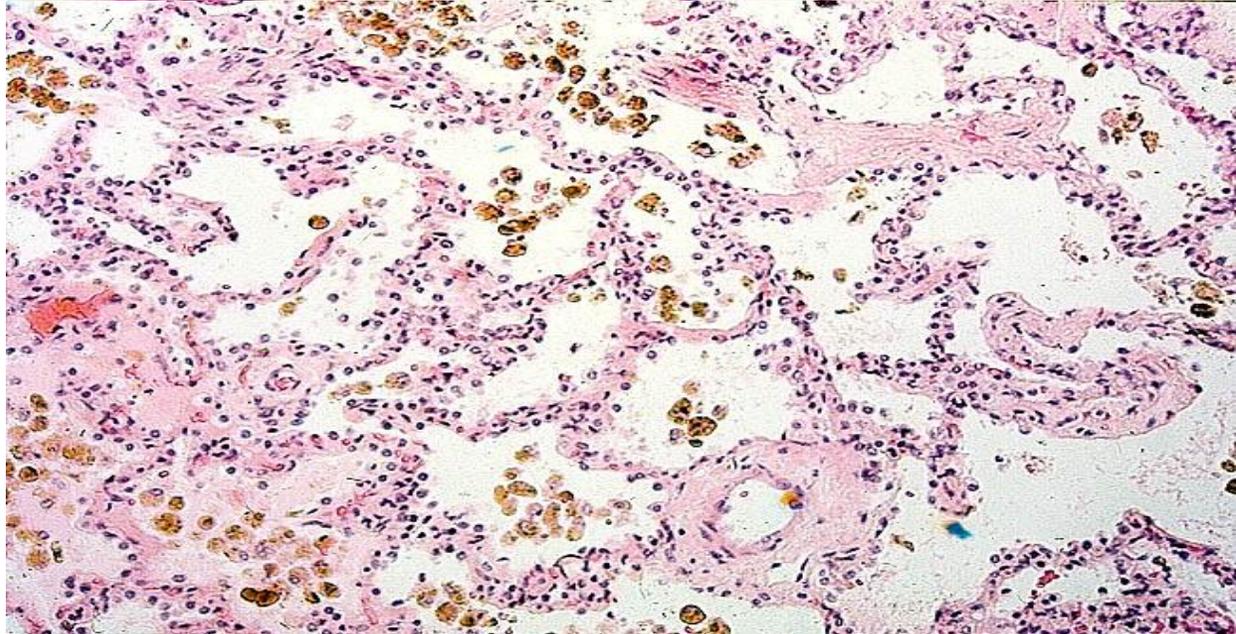
Intraalveolar hemorrhage



chronic pulmonary congestion

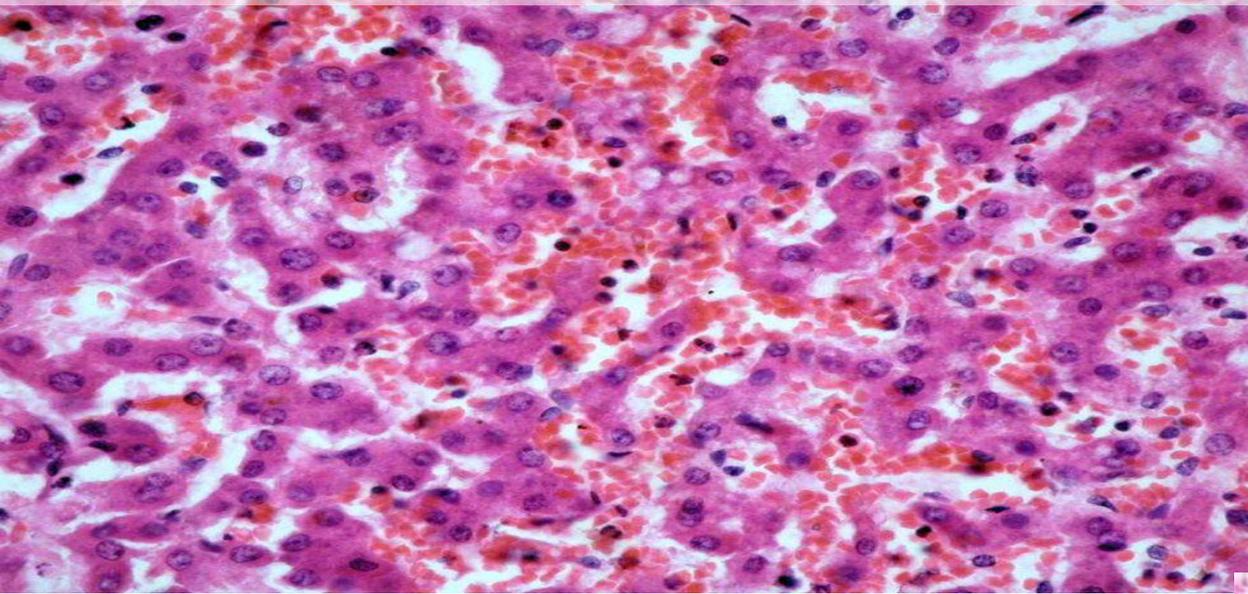
septa become thickened & fibrotic.

Alveolar spaces contain numerous macrophages laden with hemosiderin "**heart failure cells**" derived from phagocytosed red cells.



Morphology

Acute hepatic congestion



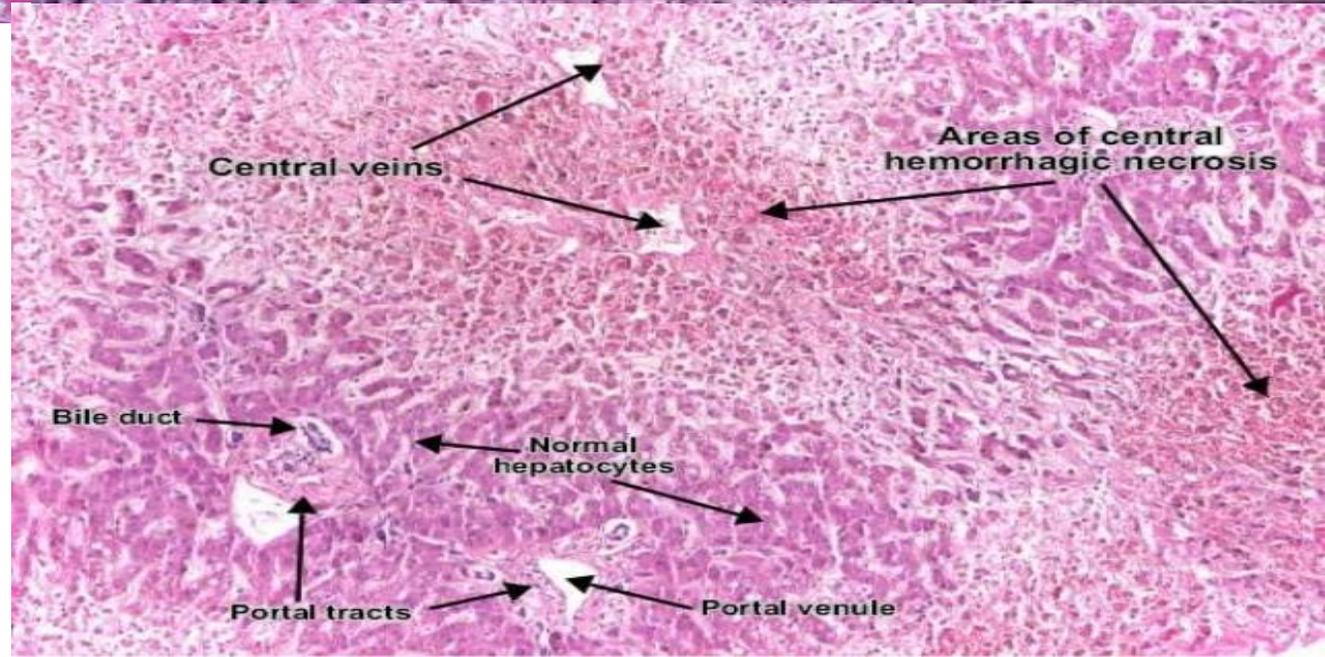
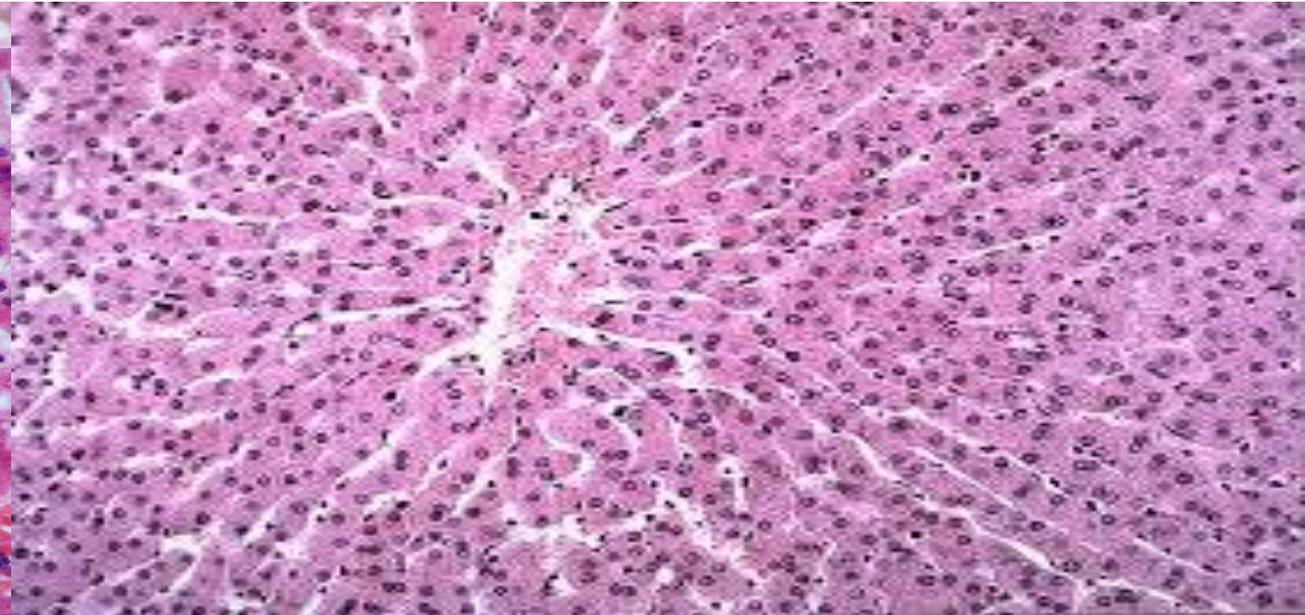
Acute hepatic congestion

the central vein & sinusoids are distended with blood.

The **periportal** hepatocytes, better oxygenated (proximity to hepatic arterioles), develop only reversible fatty change.

Chronic passive hepatic congestion

Centrilobular hepatocyte necrosis, hemorrhage, and hemosiderin-laden macrophages



Chronic Liver congestion(Nutmeg liver)

The central regions of hepatic lobules are red-brown and slightly depressed (because of a loss of cells) and are accentuated against surrounding zones of uncongested area, sometimes fatty liver



Chronic Passive Congestion, "nutmeg liver"

Nutmeg

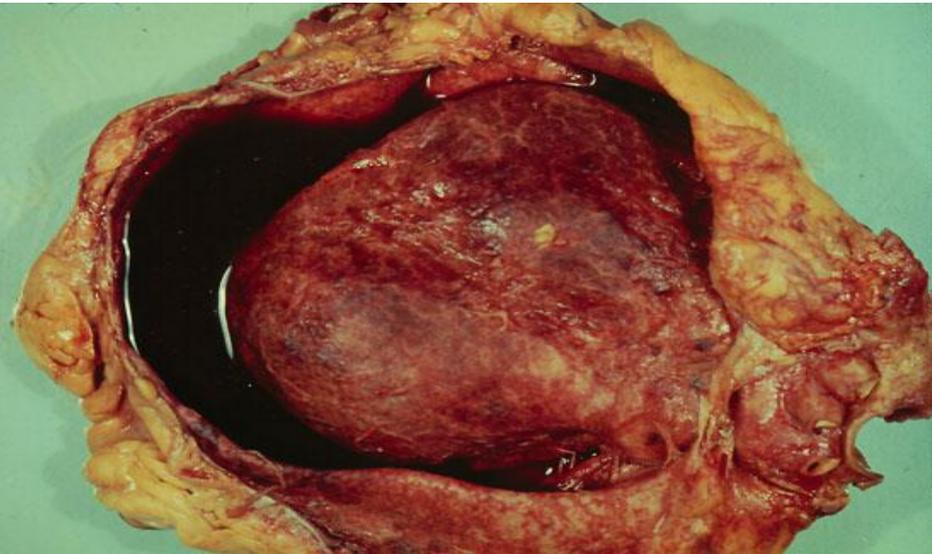


Hemorrhage

Hemorrhage ?

- **The Extravasation of blood from vessels, often the result of damage to blood vessels or defective clot formation.** (Extravasate : to move out of the vasculature)
- **Hemostasis** is the process of blood clotting that prevents excessive bleeding after blood-vessel damage.
- **Inadequate hemostasis may result in hemorrhage.** Conversely, **inappropriate clotting (thrombosis) or migration of clots (embolism) can obstruct blood vessels causing ischemia.**
- The risk of hemorrhage (after insignificant injury) is increased in some clinical disorders collectively called **hemorrhagic diatheses**. These have diverse causes, including inherited or acquired defects in **vessel walls, platelets, or coagulation factors**, all of which must function properly to ensure hemostasis.

Clinical features of hemorrhages..



Into Body cavities

Large bleeds into body cavities are described according to location: hemothorax, hemopericardium, hemoperitoneum, or hemarthrosis (in joints).



Hematoma

Accumulation of blood within a tissue.

ranges in significance from trivial to fatal.



Jaundice

Extensive hemorrhages can occasionally result in jaundice from the massive breakdown of red cells and hemoglobin.



Ecchymosis

+ Large 1-2 cm subcutaneous hematomas (bruises), red cells phagocytosed & degraded by macrophages.

+ color changes result from enzymatic conversion of hemoglobin (red-blue color) to bilirubin (blue-green color) & eventually hemosiderin (golden-brown).



Petechiae

+ minute 1-2 mm hemorrhages into skin, mucous membranes, or serosal surfaces.

+ Causes: low platelet counts (thrombocytopenia), defective platelet function, or loss of vascular wall support, as in vitamin C deficiency



Purpura

+ Purpura is 3-5 mm hemorrhages.

+ can result from the same disorders that cause petechiae, as well as trauma, vascular inflammation (vasculitis), & increased vascular fragility.

The clinical significance of hemorrhage..



- ❖ It depends on the **volume** of blood lost & the **rate** of bleeding;
 - + Rapid loss up to 20% of volume, or slow losses of even larger amounts, may have little impact in healthy adults.
 - + greater losses, can cause hemorrhagic (hypovolemic) shock.
- ❖ The site of hemorrhage also is important; bleeding that would be **trivial in the subcutaneous tissues**, can cause **death if located in the brain**
- ❖ Chronic or recurrent **external** blood loss (e.g., due to peptic ulcer or menstrual bleeding), **culminates in iron deficiency anemia** due to loss of iron in hemoglobin. By contrast, iron is efficiently recycled from phagocytosed red cells, so **internal** bleeding (e.g., a hematoma) **does not lead to iron deficiency**



THANK YOU