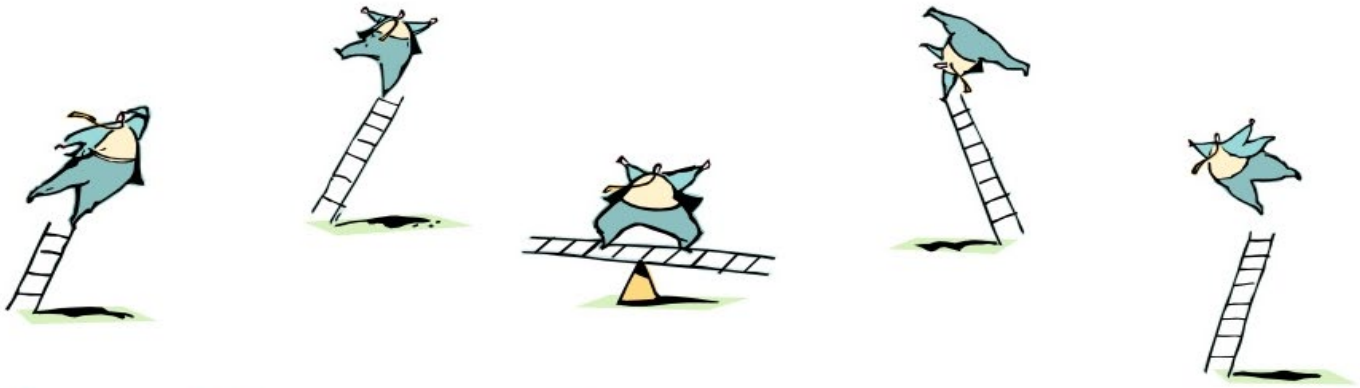
Homeostasis "Hemodynamic" Disorders

Dr. Abdulsalam Al-Ani
3rd Year – Under Graduate
College of Medicine
University of Anbar

Homeostasis



The Great Balancing Act

Homeostasis

Refers to the mechanism of body's ability to maintain a stable internal environment.

Biological systems; regulating hormones, body temp., water balance, …etc.)

Maintaining homeostasis requires that the body continuously monitors its internal conditions in a healthy status, that are optimal for survival.

These mechanisms are necessary for the body to regain its balance when disease or injury occurs and to maintain it as of the healthy one.

Objectives

1. Edema (oedema).
2. Congestion and Hyperemia.
3. Hemorrhage.
4. Thrombosis.
5. Embolism.
6. Infarction.
7. Shock.

Objectives

1. **Edema (oedema).**
2. Congestion and Hyperemia.
3. Hemorrhage.
4. Thrombosis.
5. Embolism.
6. Infarction.
7. Shock.

Normal fluid homeostasis requires :

1. **Vessel wall integrity**
2. **Maintenance of intravascular pressure**
3. **Osmolarity within certain physiologic ranges.**

Edema (Oedema)

The Greek word *odema* means swelling.

Abnormal and excessive accumulation of "free fluid" in the interstitial tissue spaces and serous cavities.

Water constitutes around 60% of our body weight, distributed;

- 2/3 intracellular.
- 1/3 is extra cellular:
  * mainly interstitial
  * intravascular fluid (blood plasma) constitutes only 5% of the total body water.

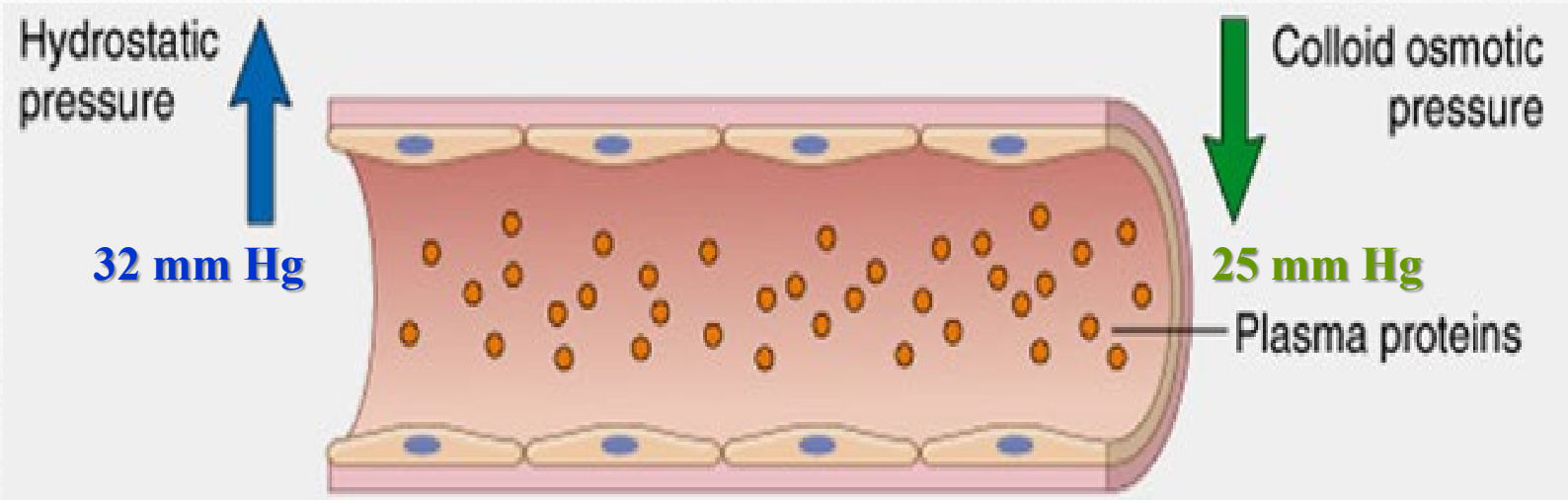
Mechanisms of Edema

The Starling's Law:

Describes fluid movement across capillaries being proportional to capillary permeability, trans-capillary hydrostatic pressure differences, and trans-capillary oncotic pressure differences.

Equation: Filtration = Kf x (Pc – Pif – Oc + Oif).

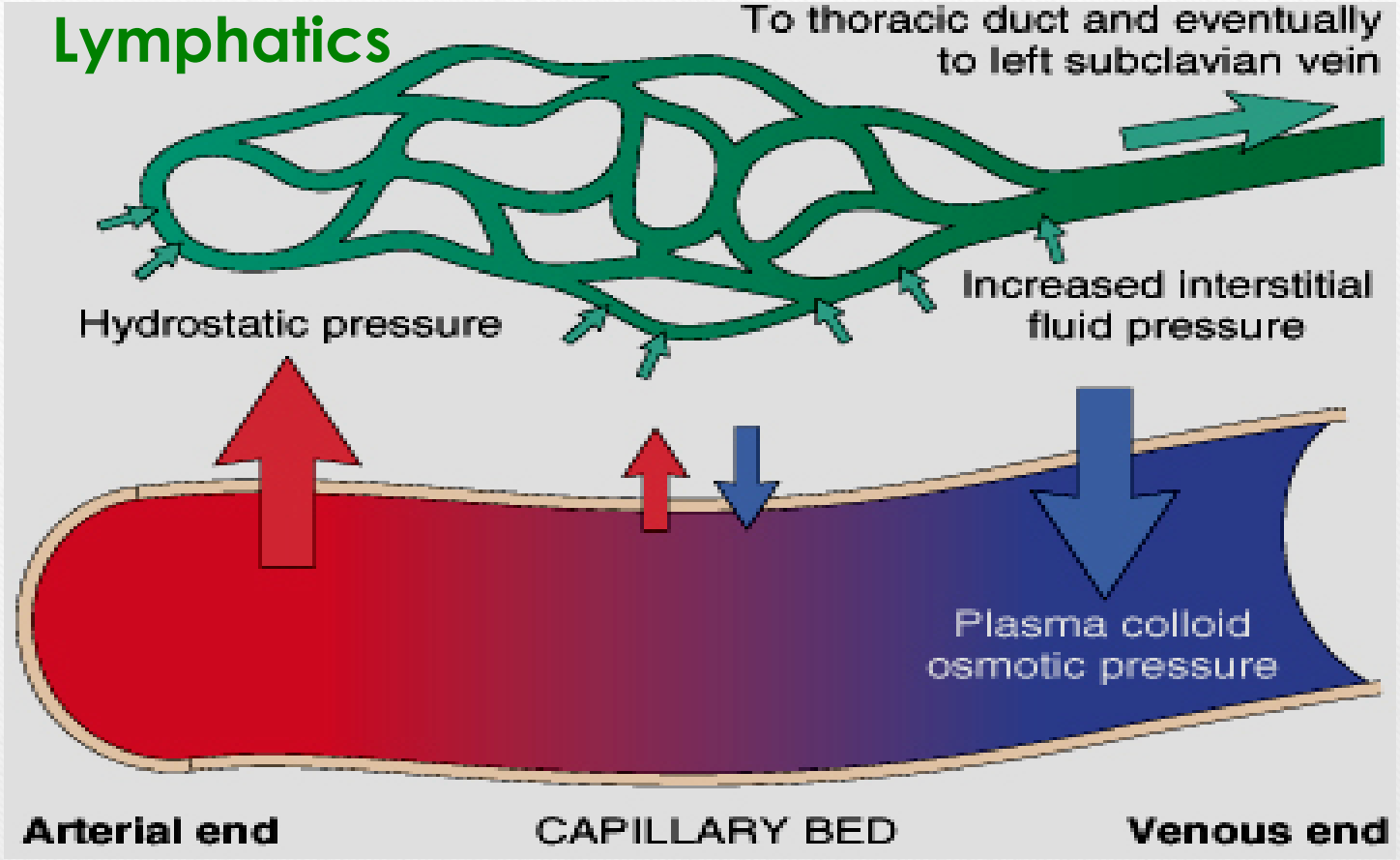
Pc; hydrostatic capillary pressure, **Pif**; interstitial fluid hydrostatic pressure, **Oc**; capillary plasma colloid osmotic pressure, **Oif**; interstitial fluid colloid osmotic pressure, and **Kf**; capillary filtration coefficient (permeability x surface area)



Mean blood hydrostatic pressure (HP) at the arteriolar ends of the capillaries is about 32 mm.Hg, while the colloid osmotic pressure of plasma (OP) (also called oncotic pressure) is 25 mm.Hg.

- The net difference of the two forces causes fluids to escape from intravascular compartment into the tissue interstitial compartment under the influence of the HP.

- As the mean HP at the venous end of the capillaries is lower than that of the arteriolar end (about 12 mm. Hg), the net difference of the two forces here works in opposite direction, i.e. tissue fluids return back to the vessels under the influence of OP.

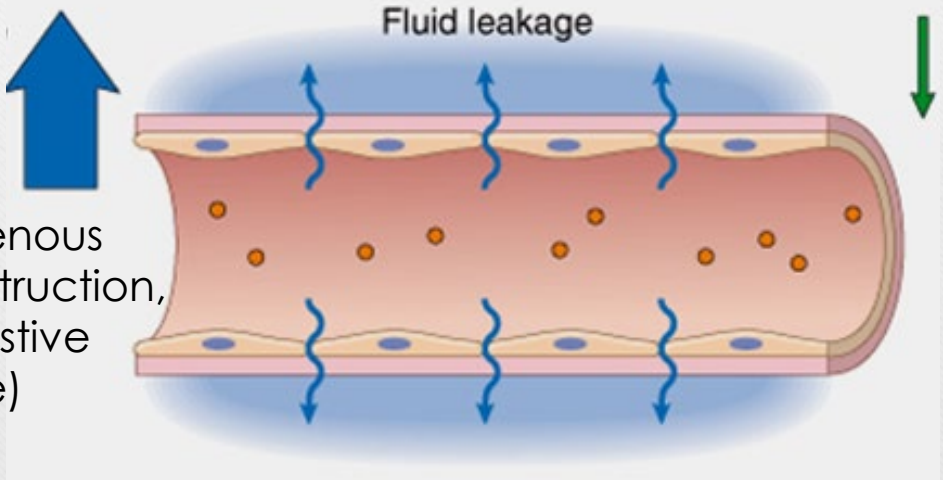


Increased hydrostatic pressure or diminished plasma osmotic pressure leads to extravascular fluid accumulation (edema). Tissue lymphatics drain much of the excess fluid back to the circulation by way of the thoracic duct; however, if the capacity for lymphatic drainage is exceeded, tissue edema results.

Nature of Accumulated Fluid:

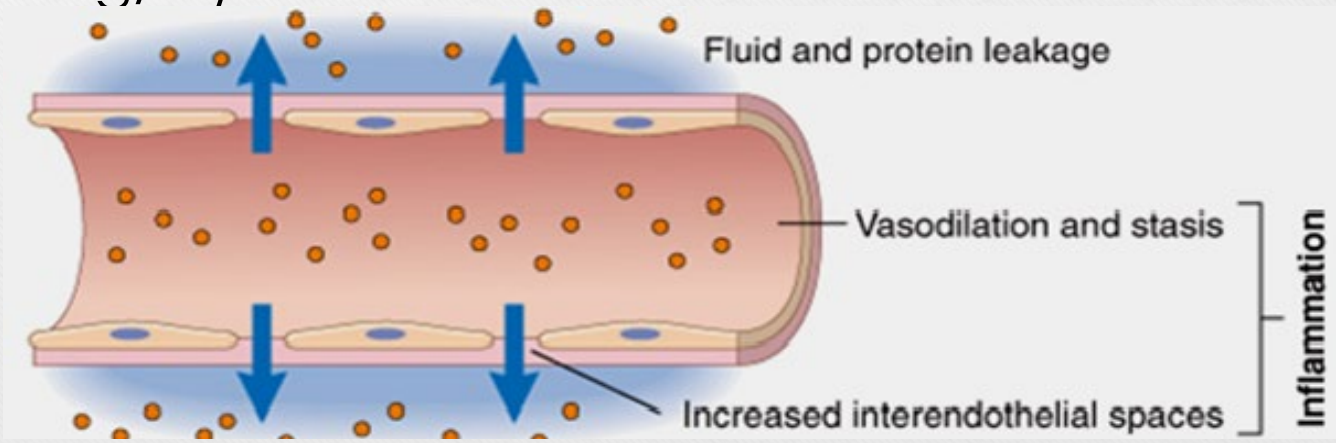
a. Transudate :

- Thin protein-poor fluid
- It has a specific gravity below 1.020 (protein concentration less than 2.5 g/dl).
- It is usually related to increased hydrostatic pressure within the intravascular compartment.
- e.g. in heart failure or nephrotic syndrome



b. Exudate :

- Thick protein-rich.
- Associated with inflammation.
- Resulting from increased vascular permeability that leads to escape of intravascular proteins principally albumin.
- Having a specific gravity > 1.020 (protein concentration more than 2.5 g/dl).



Pathophysiology of Edema

1. Increased Hydrostatic Pressure

1- Impaired venous return;

a. Congestive heart failure, Constrictive pericarditis.
b. Liver cirrhosis.
c. Venous obstruction or compression;
- Thrombosis
- External pressure (e.g., mass)
- Lower extremity inactivity with prolonged dependency

2- Arteriolar dilation;

a. Heat.

b. Neurohumoral dysregulation.

2. Reduced Plasma Osmotic Pressure (Hypoproteinemia);

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

3. Lymphatic Obstruction;

- Inflammatory.
- Neoplastic
- Postsurgical
- Post irradiation

4. Sodium Retention

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

5. Inflammation

- Acute inflammation, Chronic inflammation, Angiogenesis

A. Increased Hydrostatic Pressure
- Impaired Venous Return
1- Congestive heart failure
2- Constrictive pericarditis
3- Ascites (liver cirrhosis)
4- Venous obstruction or compression; thrombosis, external pressure (e.g., mass), lower extremity inactivity with prolonged dependency
- Arteriolar Dilation; heat, neurohumoral dysregulation
B. Reduced Plasma Osmotic Pressure (Hypoproteinemia)
1- Protein-losing glomerulopathies (nephrotic syndrome)
2- Liver cirrhosis (ascites)
3- Malnutrition, protein-losing gastroenteropathy
C. Lymphatic Obstruction
1- Inflammatory
2- Neoplastic
3- Postsurgical & Postirradiation
D. Sodium Retention
Excessive salt intake with renal insufficiency
Increased tubular reabsorption of sodium; renal hypoperfusion, increased renin-angiotensin-aldosterone secretion
E. Inflammation;
Acute inflammation, chronic inflammation, angiogenesis

1. Increased capillary hydrostatic pressure:

A. Generalized or B. Localized

A. Generalized:

Right ventricular failure leading to;

a. Generalized chronic venous congestion (that is why the heart failure (Rt) is called congestive cardiac failure). This type of edema known as ***'cardiac edema'***.

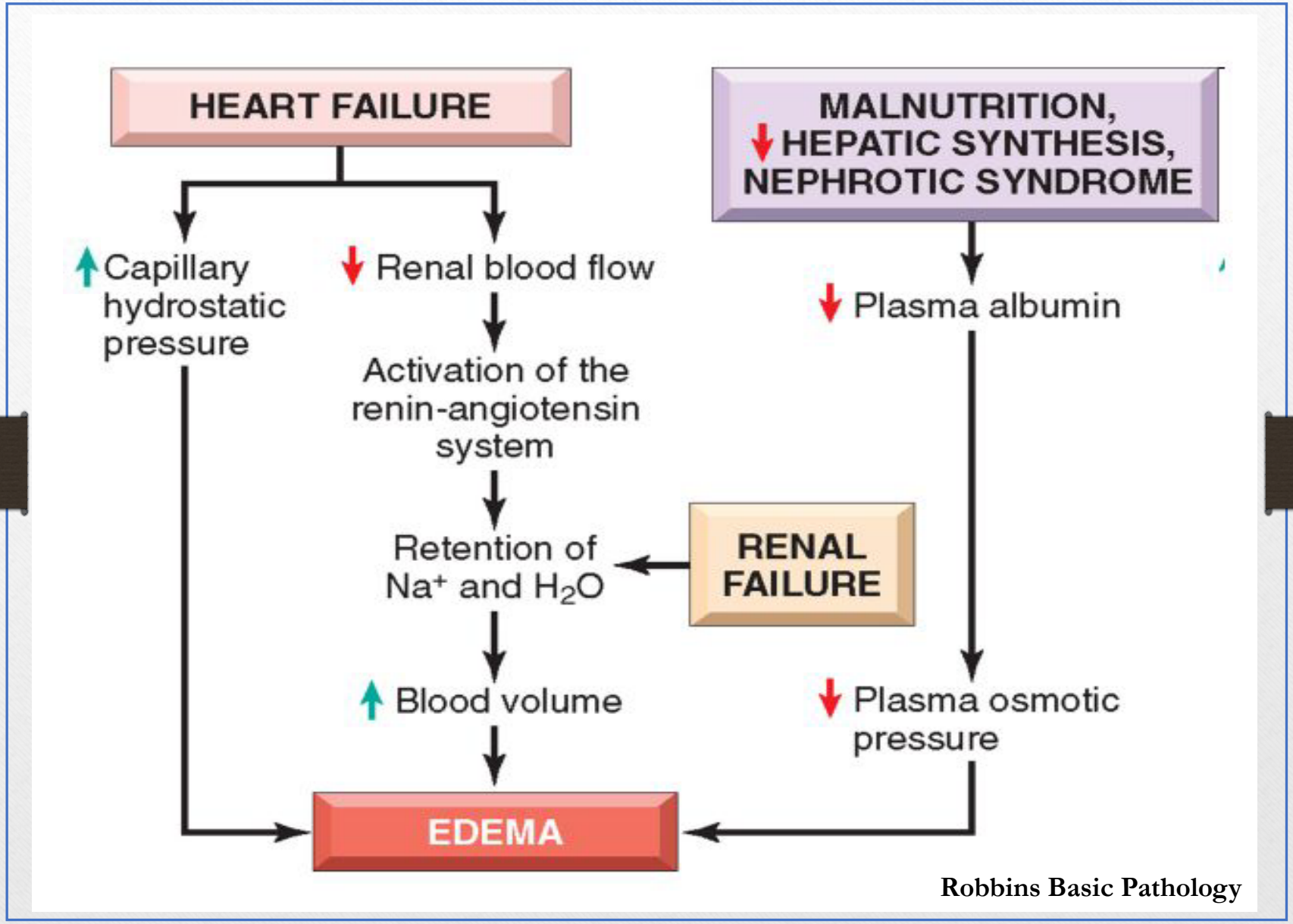
b. Reduction in the blood flow to kidneys;

- Result in excessive tubular reabsorption of water.
- Also stimulate the juxta-glomerular apparatus triggering the renin-angiotensin-aldosterone system that results in the excessive secretion of renin.

- This in turn enhances the secretion of aldosterone by the adrenal cortex with consequent reabsorption of sodium by the renal tubules.

c. Sodium retention stimulates secretion of antidiuretic hormone (ADH) by the neurohypophysis, and so more water is reabsorbed through the renal collecting tubules.

d. Accumulation in the tissues of waste products, which by their osmotic (oncotic) action, will tend to attract more water from the blood.



Robbins Basic Pathology

B. Localized:

The combination of the two following factors lead to the localized type of edema.

1- Obstruction :

Partial or complete obstruction of a large venous trunk lead to venous congestion and therefore venous pressure which is transmitted to the capillaries.

2- Inflammation:

Capillary injury: impairment of the oxygenation and nutrition of the capillary endothelial cells, which become more permeable.

Examples of Localized Edema are:

1. Portal hypertension caused by liver cirrhosis. This produces a transudate in the peritoneal cavity (ascites).

2. Pressure of **gravid uterus** on the iliac veins produces congestion and edema of the lower limbs.

3. Acute left ventricular failure causes **acute pulmonary edema**. This is due to damming of blood and increase in the Lt atrial pressure which is transmitted back through the pulmonary veins.

4. Thrombosis or occlusion of major veins.

2. Decrease in colloid osmotic pressure of plasma (Hypoproteinemia):

Normal protein is (6.4- 8.6 g/dl), When it drops below 2.5 g/dl or when the albumin fraction drops below 1.5 g/dl (normal 4.0 - 5.5 g/dl), the osmotic (oncotic) pressure of the blood diminishes.

Examples:

1. **Nephrotic syndrome** resulting from , e.g. amyloidosis, diabetes and rapidly progressive glomerulonephritis.
2. **Diffuse liver disease** e.g. cirrhosis (decreased synthesis of plasma proteins by the diseased liver).
3. **Malnutrition** e.g. famine edema, severe protein-calorie malnutrition (Kwashiorkor)
4. **Protein losing gastro-enteropathy.**

3. Obstruction of lymphatic vessels:

It is a non pitting edema, which also seen in **myxedema** and **lipedema**.

Causes:

a. Congenital absence of lymphatic vessels.

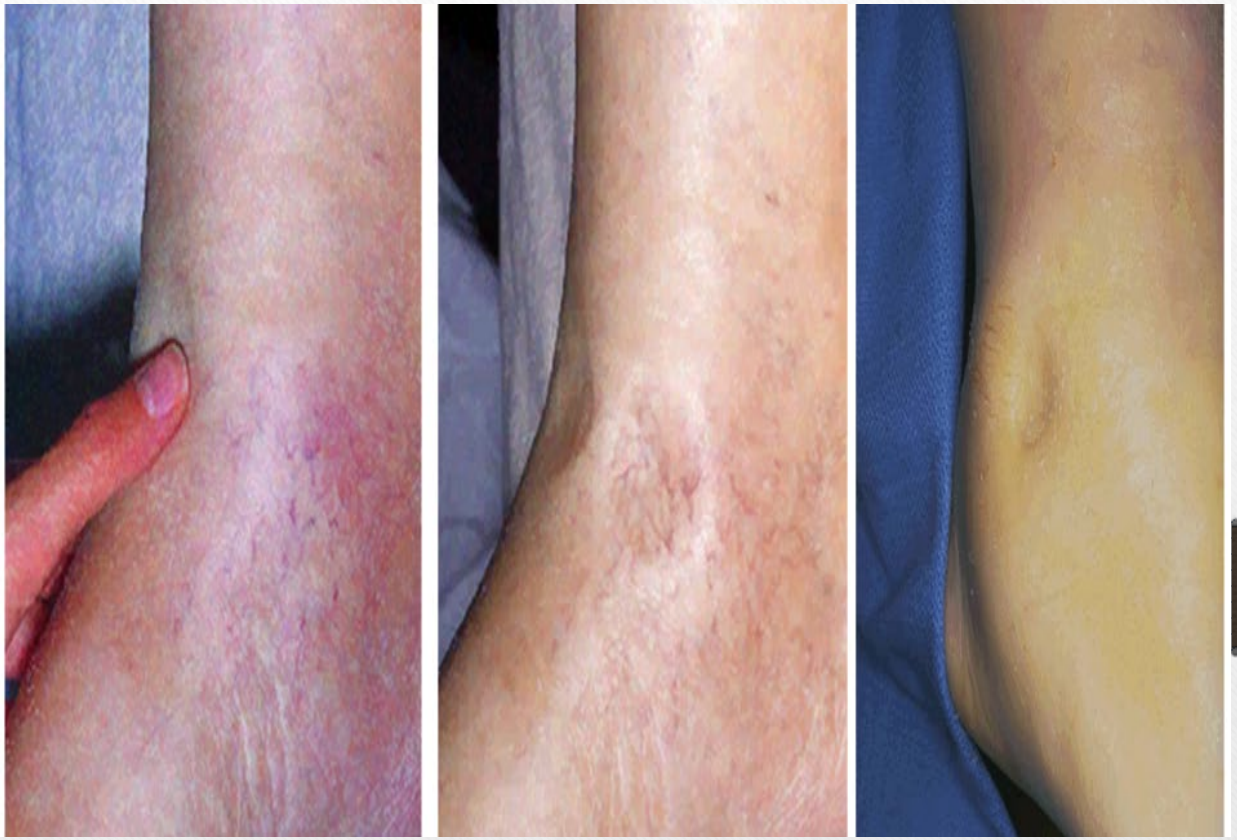
b. Parasitic infestation; e.g.; chronic infection with the filarial worm (Filariasis); *Wuchereria bancrofti* induces lymphatic blockage, most often in the inguinal regions producing epidermal thickening and massive edema of the external genitalia and legs. This has been likened to that of elephants (elephantiasis).

c. Tumor invasion; Such as breast cancer with superficial lymph. vessels leading to breast skin edema (peau d'orange "orange peel").

d. Post-radiation.

e. Post-surgical.

Pitting Edema



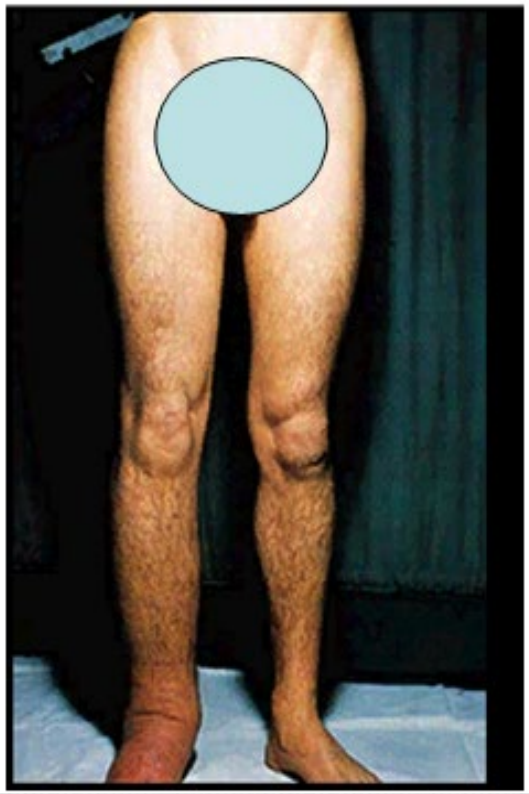
1. Lung Diseases.
2. Obesity and Pregnancy.
3. Medications.
4. Low Levels of Protein.
5. Heart Failure.

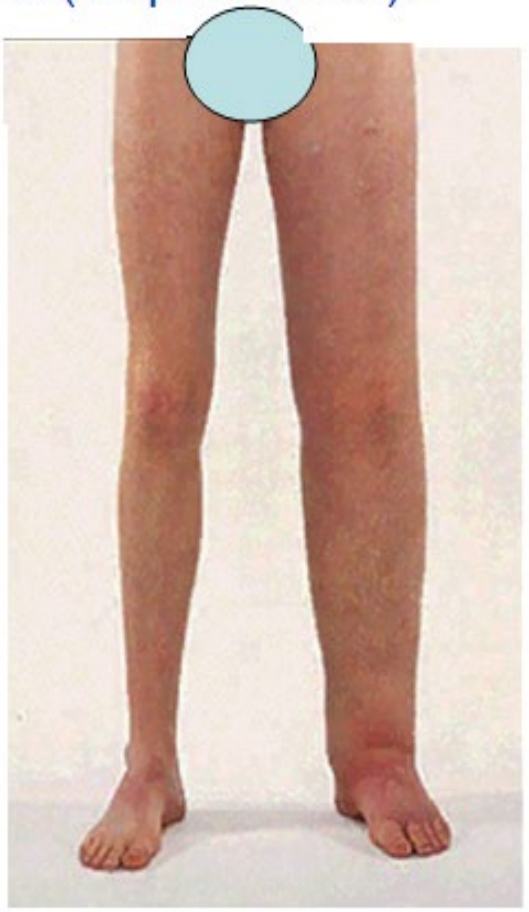
Lymphedema

non-pitting edema

As the disease become more chronic, the limb becomes hard and woody and the skin thickened and wrinkled (elephantiasis).

1- Myxedema
2- Lipedema





4. Sodium and water retention:

- **Contributory;** aggravating factors in several forms of edema (may also be a primary cause of edema);

Increased salt retention (which is associated with obligate water retention), causes both increased HP (due to expansion of intravascular fluid volume) and diminished vascular colloid osmotic pressure (due to dilution).

Salt retention may occur with any acute reduction of renal function, including post-streptococcal glomerulonephritis and acute renal failure.

5. Increased capillary permeability:

A. Localized: in inflammatory lesions a high osmotic (oncotic) pressure occur , which draws more water from the blood into the tissue spaces. The lymph flow is increased, but still, the exudate accumulates and causes a localized swelling or inflammatory edema.
Edema is one of cardinal gross appearance of acute inflammation.

B. Generalized: increased capillary permeability is responsible for generalized edematous lesions, which accompany some allergic condition such as urticaria or angioneurotic edema (allergic edema).

Anasarca; a severe, generalized edema, including profound subcutaneous tissue fluid accumulations.

Ascites (hydroperitoneum); collection of fluid (edema) in the peritoneal cavity.

Pleural effusion (hydrothorax); collection of fluid in the pleural cavity.

Pericardial effusion (hydropericardium); collection of fluid within the pericardial cavity.

Hydropic change (cellular edem); accumulation of fluid inside cells.

Causes of Edema

1. Increased hydrostatic pressure (e.g., heart failure)
2. Increased vascular permeability (e.g., inflammation)
3. Decreased colloid osmotic pressure, due to reduced plasma albumin;
   - Decreased synthesis (e.g., liver disease, protein malnutrition)
   - Increased loss (e.g., nephrotic syndrome)
4. Lymphatic obstruction (e.g., inflammation or neoplasia).
5. Sodium retention (e.g., renal failure)

Clinical manifestations of edema

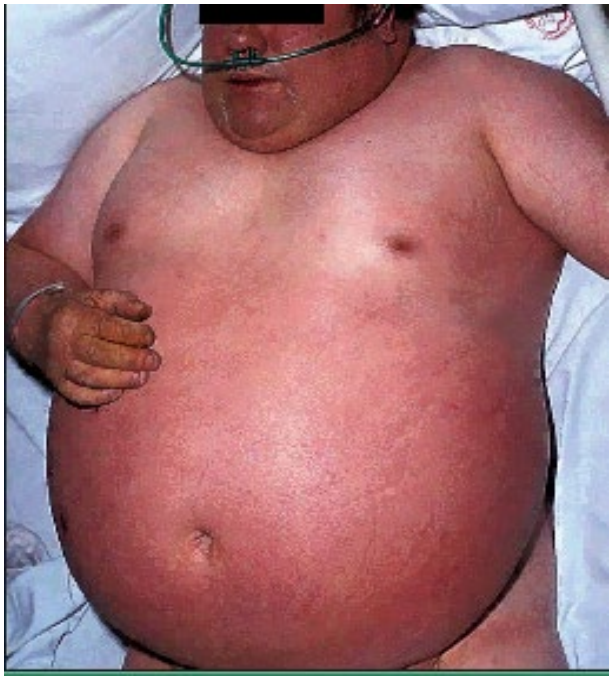
- **Subcutaneous edema;** pitting or non-pitting swelling of the legs or sacral area (depending edema), or periorbital. Usually impaired wound healing or the clearance of infection.

- **Edema of the lung** can cause very severe dyspnea and anoxia. It can cause death by interfering with normal ventilation.

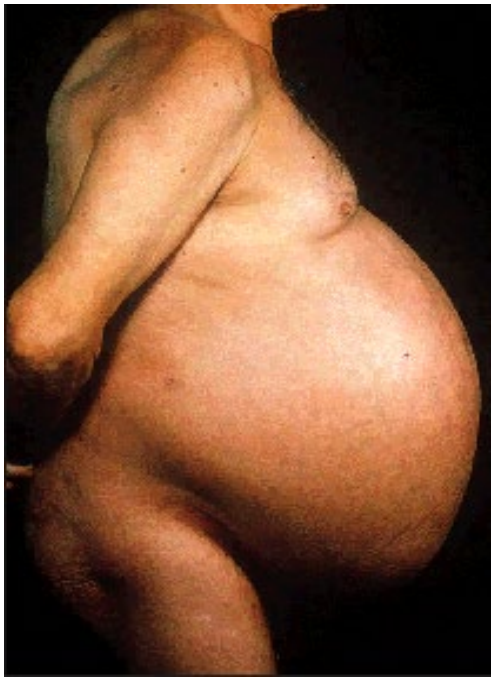
- **Edema of the brain** is serious and can be rapidly fatal. If severe, brain substance may be herniated (pushed out) through e.g. foramen magnum, resulting in direct compression of brain stem or its vascular supply. Either of these can injure the vital centers within the medulla and cause death.

- **Ascites;** abdominal distension and discomfort with dyspnea

Ascites (severe form)

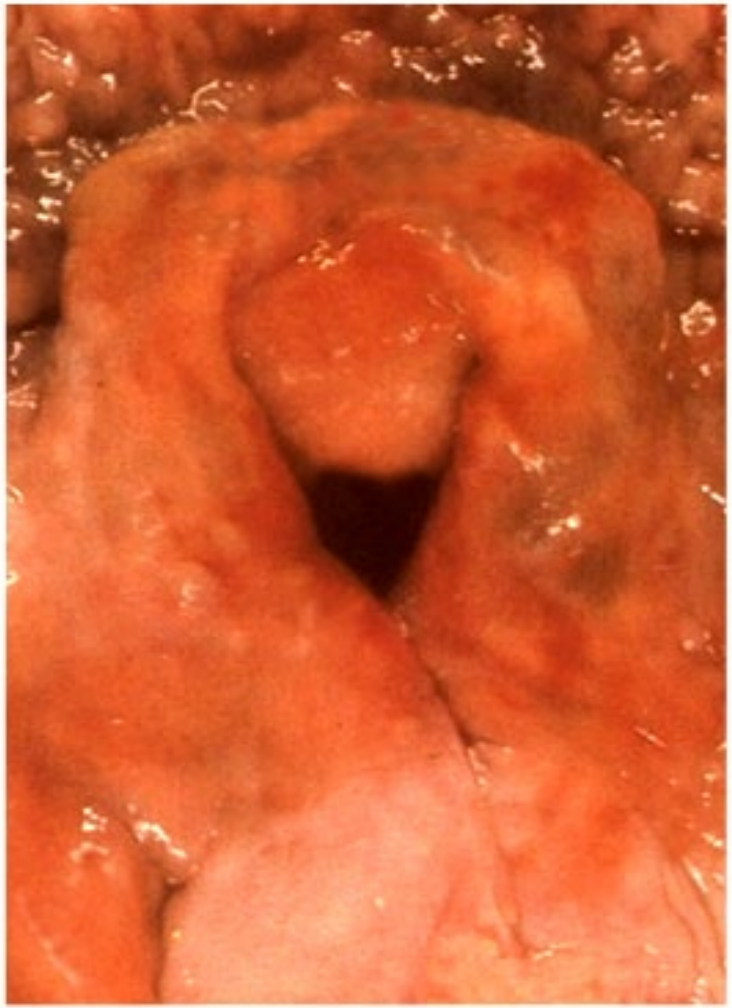


Rt sided heart failure secondary to lung disease (note cyanosis)



A case of liver cirrhosis

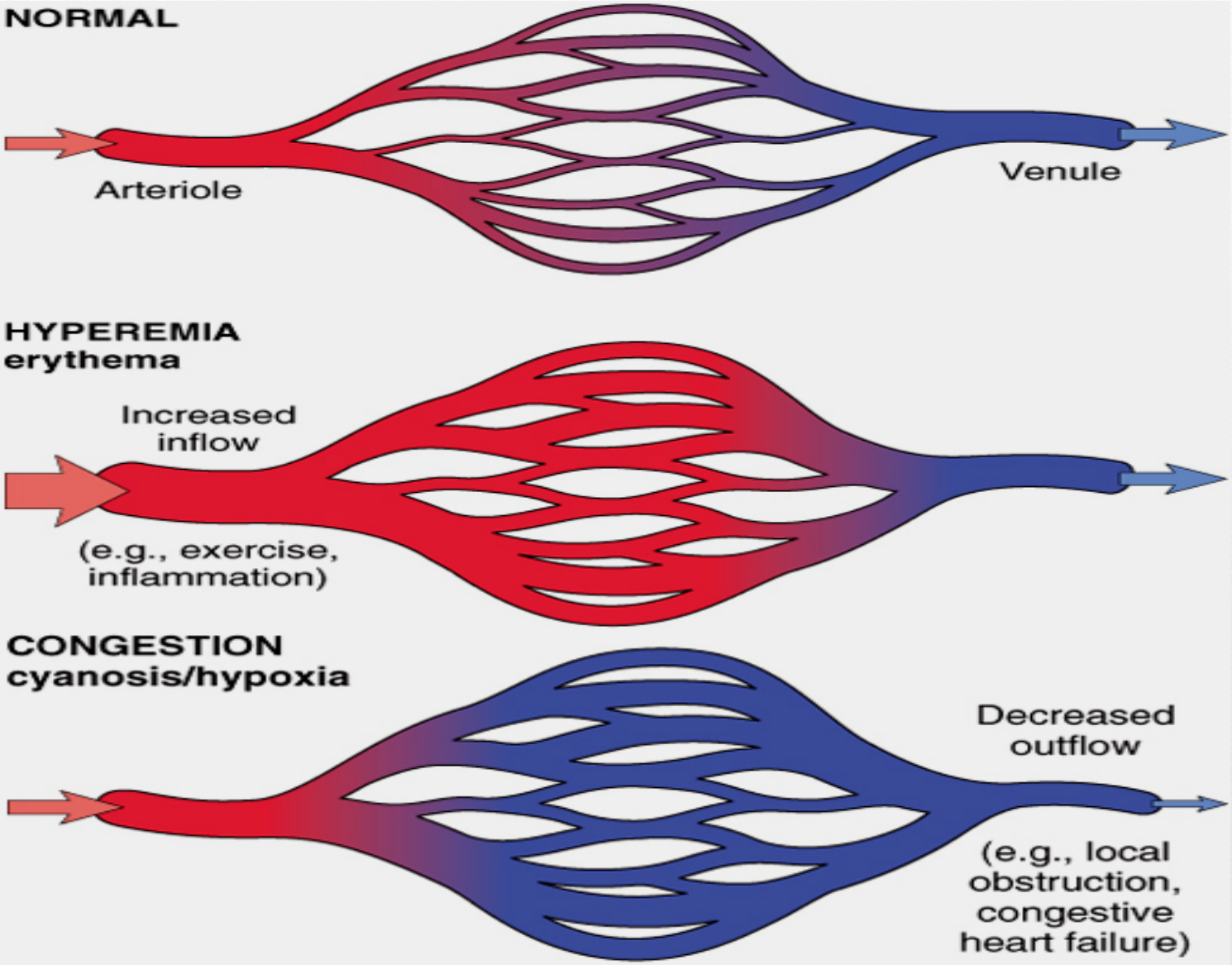
- **Edema of the larynx** may cause suffocation (asphyxia), which is the most urgent.



Congestion and marked narrowing of laryngeal orifice.

Objectives

1. Edema (oedema).
2. **Congestion and Hyperemia.**
3. Hemorrhage.
4. Thrombosis.
5. Embolism.
6. Infarction.
7. Shock.


NORMAL
Arteriole
Venule
HYPEREMIA
erythema
Increased
inflow
(e.g., exercise,
inflammation)
CONGESTION
cyanosis/hypoxia
Decreased
outflow
(e.g., local
obstruction,
congestive
heart failure)

Congestion & Hyperemia

In both conditions there is increase in the volume of blood in an affected tissue or part of the body. Usually hyperemia is active while congestion is passive.

- **Active Hyperemia (arterial) congestion:**

It is an active process resulting from increased arterial blood inflow because of arteriolar dilatation.

The affected tissue is reddened because of engorgement of tissues with **oxygenated** blood & therefore the tissue appears bright red.

This occurs for e.g. in exercise, blushing, febrile states and locally in inflammation.

The arterial and arteriolar dilatation is brought about by:

1- Sympathetic neurogenic mechanisms.

2- Release of vasoactive substances.

➢ Passive (venous) Congestion

It is a passive process resulting from impaired venous outflow from a tissue, due to an obstruction or a back pressure of the venous flow.

- The affected tissue has a red-blue color due to accumulation of **deoxygenated** blood & appears blue.

Hyperemic tissue is warm, while congested blood is cold & clammy.

Either; Localized or generalized.

Venous Congestion

A- Localized venous congestion.

- pulmonary venous congestion; mostly due to Left sided heart failure.

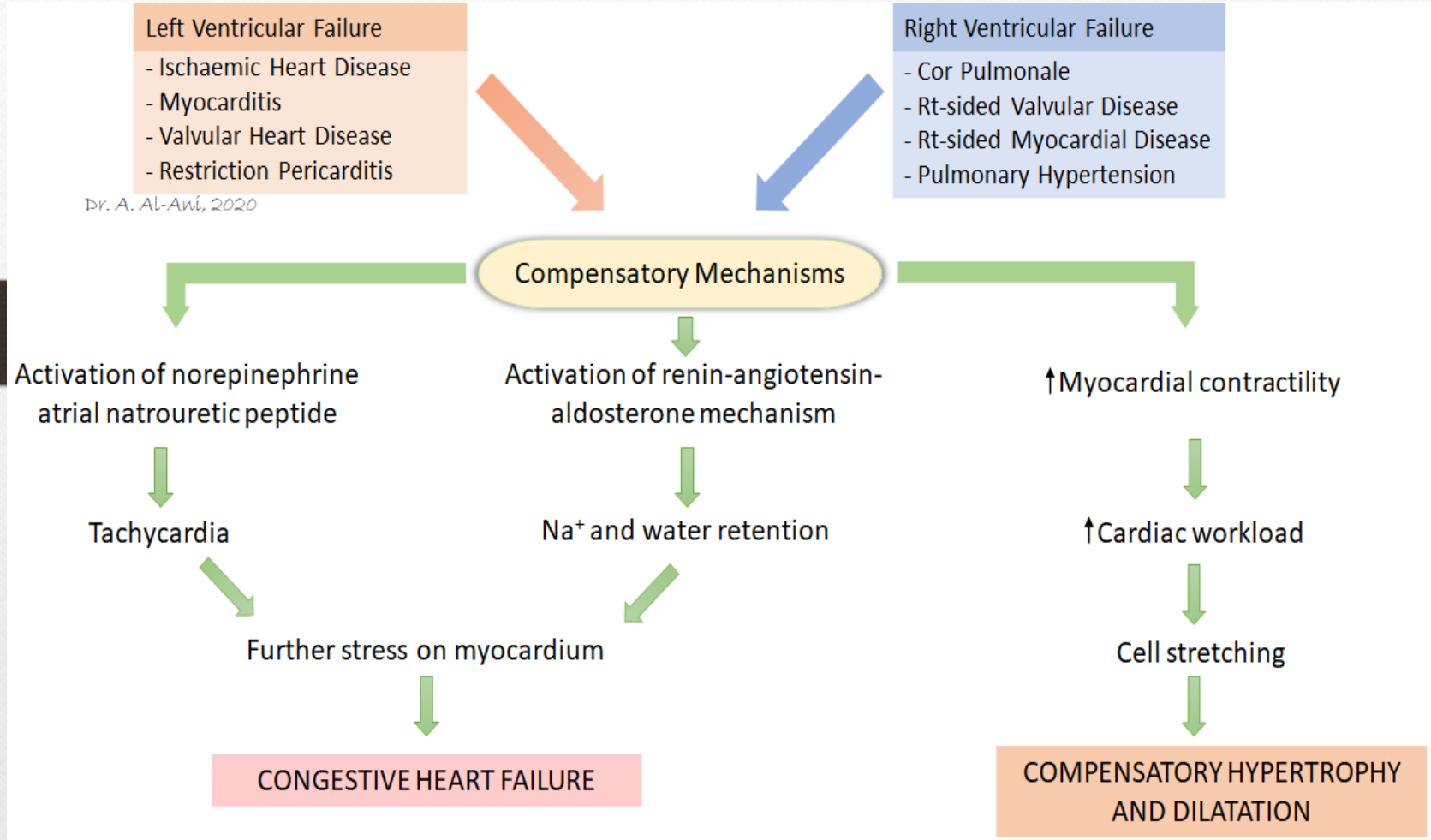
- localized venous congestion; due to a local cause.

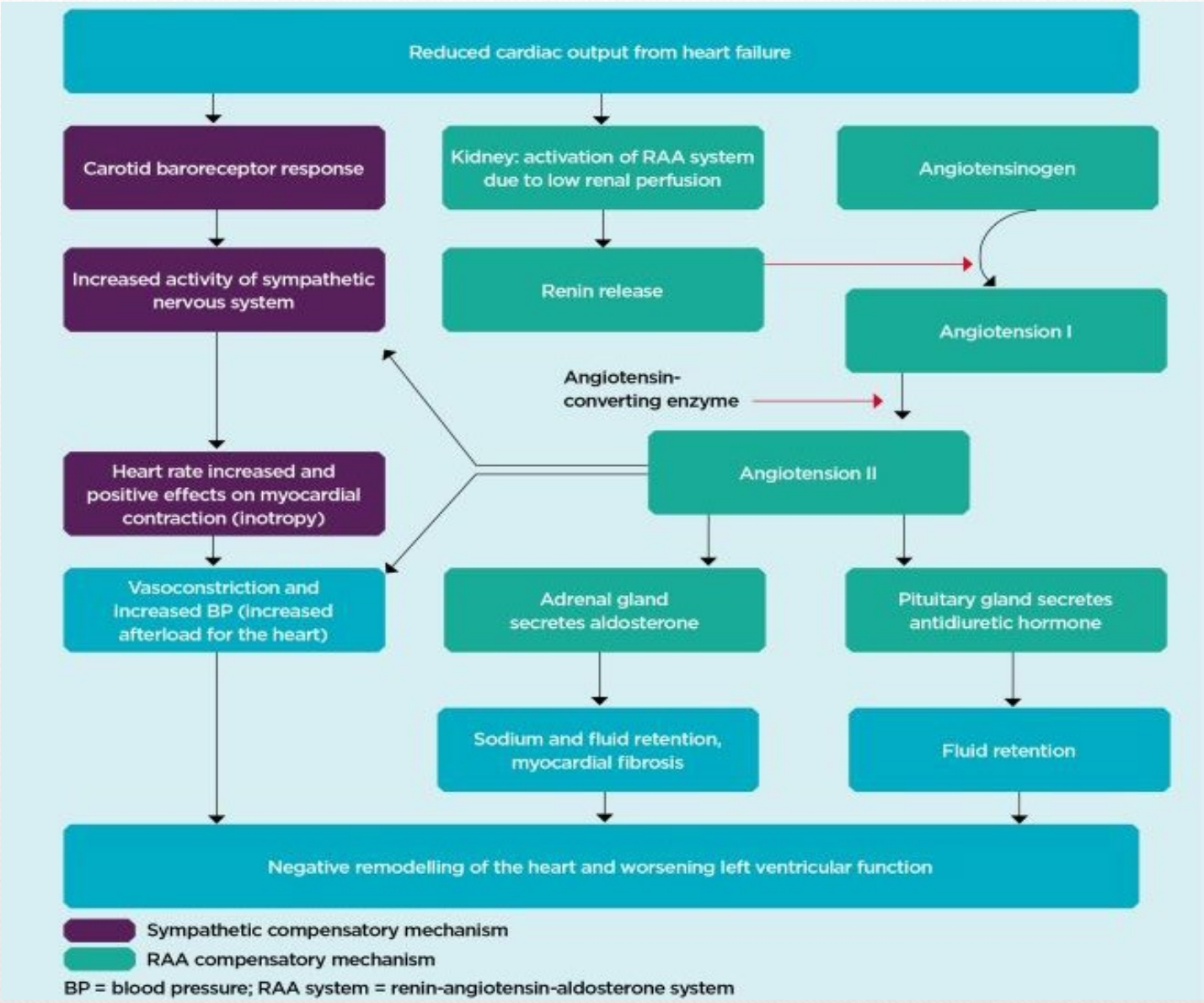
B- Generalized Systemic venous congestion.

Mostly seen in right sided heart failure(chronic); leads to generalized edema, mostly liver, kidney and spleen. Peripheral edema also manifested, mainly of the lower limbs.

❖ Pathophysiology:

In case of impaired cardiac output, there will be a reduced venous return with consequence of edema.




Reduced cardiac output from heart failure
Carotid baroreceptor response
Kidney: activation of RAA system due to low renal perfusion
Angiotensinogen
Increased activity of sympathetic nervous system
Renin release
Angiotension I
Angiotensin-converting enzyme
Heart rate increased and positive effects on myocardial contraction (inotropy)
Angiotension II
Vasoconstriction and increased BP (increased afterload for the heart)
Adrenal gland secretes aldosterone
Pituitary gland secretes antidiuretic hormone
Sodium and fluid retention, myocardial fibrosis
Fluid retention
Negative remodelling of the heart and worsening left ventricular function
Sympathetic compensatory mechanism
RAA compensatory mechanism
BP = blood pressure; RAA system = renin-angiotensin-aldosterone system

A- Localized causes of venous congestion:

1. Venous thrombosis of a major vein; e.g. femoral or popliteal that leads to congestion and swelling of the lower limb distal to the obstruction.

2. Cirrhosis of the liver;

- Congestive splenomegaly.
- A transudate collects in the peritoneal sac **(ascites).**
- The collateral venous radicles between the portal and systemic circulation open up. They become dilated, tortuous and prominent. e.g. at the cardio-esophageal junction **(esophageal varices)**, and lower end of the rectum **(hemorrhoids).**

3. Mechanical compression of veins; e.g., by a tumor, strangulated hernias, volvulus of the intestine, torsion of a vascular pedicle (e.g. of the ovary, testis).

B- Systemic venous congestion:

- **Right sided heart failure** (chronic), leads to increased arteriolar tone which is caused by sympathetic stimulation.

The inferior vena cava becomes congested, and the continued effect leads to congestion of the organs.

The main organs effected are;

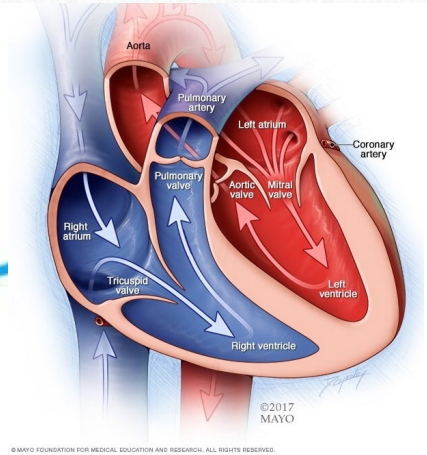
1- Liver.

2- Spleen.

3- Kidneys.

4- Body, mainly lower limbs.

- **Left sided heart failure**, leads to its main effect on the pulmonary circulation and consequence lung congestion, which may leads to congestive heart failure.

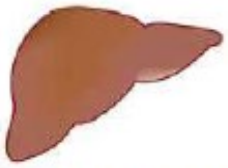


Right-sided failure

Pressure transmitted upstream of right heart

SYSTEMIC VENOUS CONGESTION

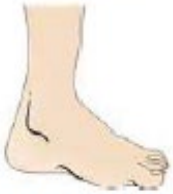
CVC LIVER



CVC SPLEEN

CVC KIDNEY

CONGESTED LEG VEINS

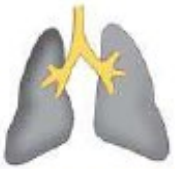


Left-sided failure

Pressure transmitted upstream of left heart

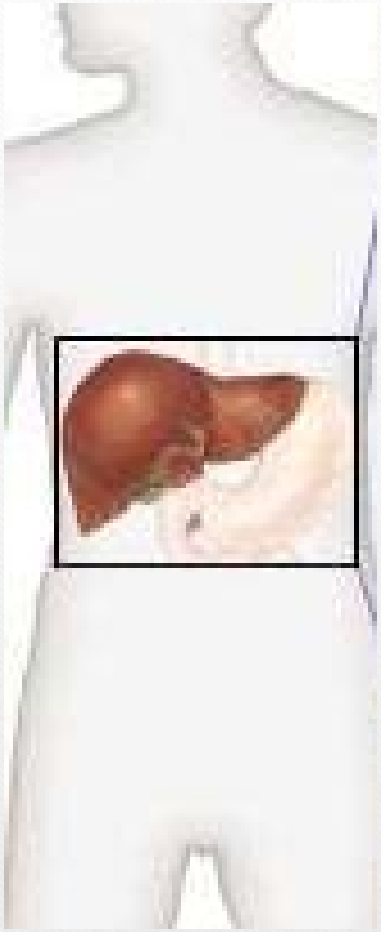
PULMONARY CONGESTION

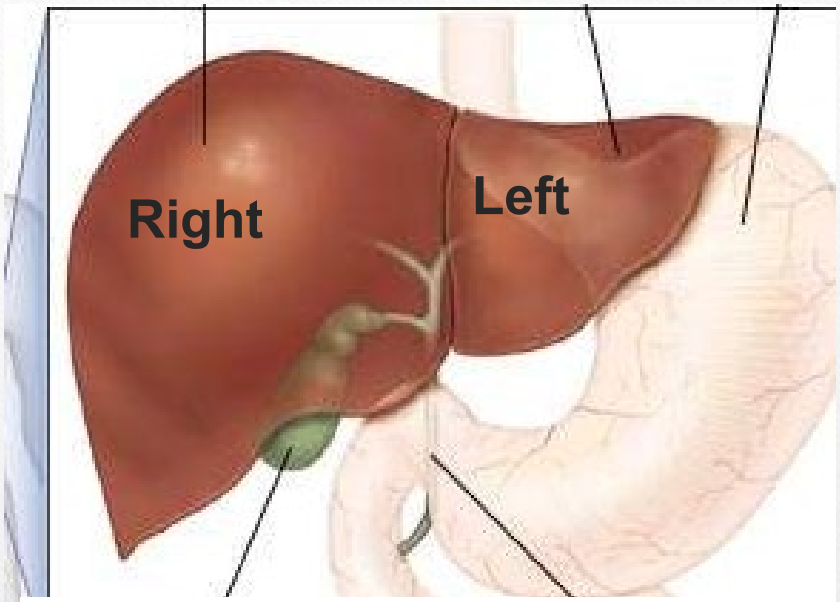
CVC LUNGS

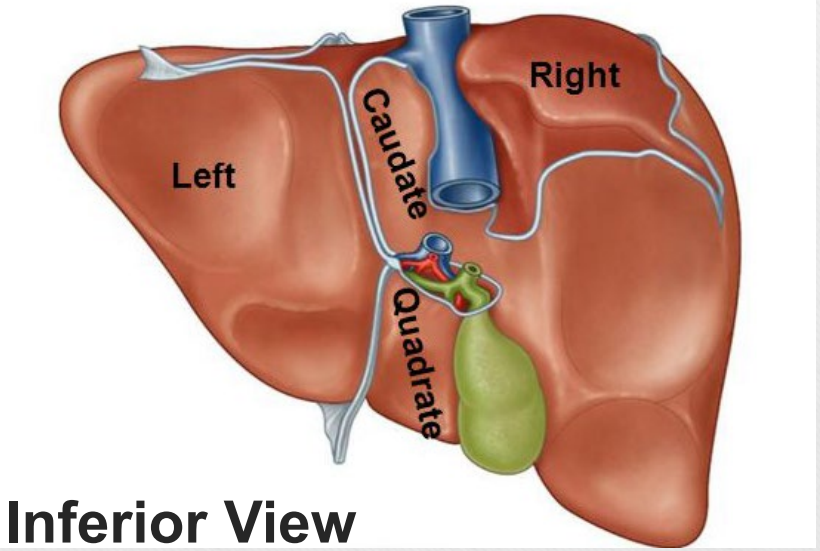


Morphological Changes in Chronic Venous Congestion

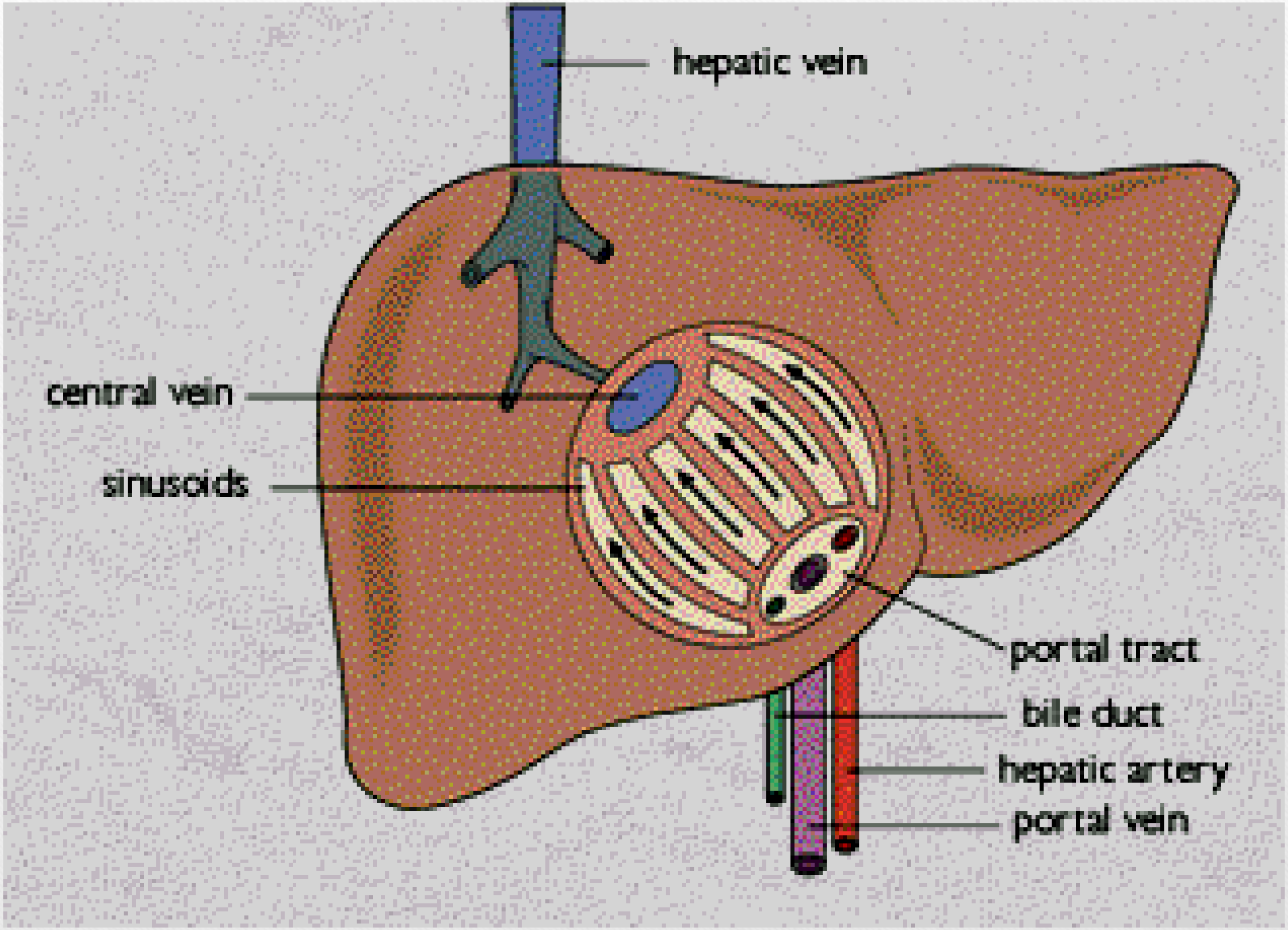
1. Liver.
2. Lung.
3. Kidney.
4. Spleen.





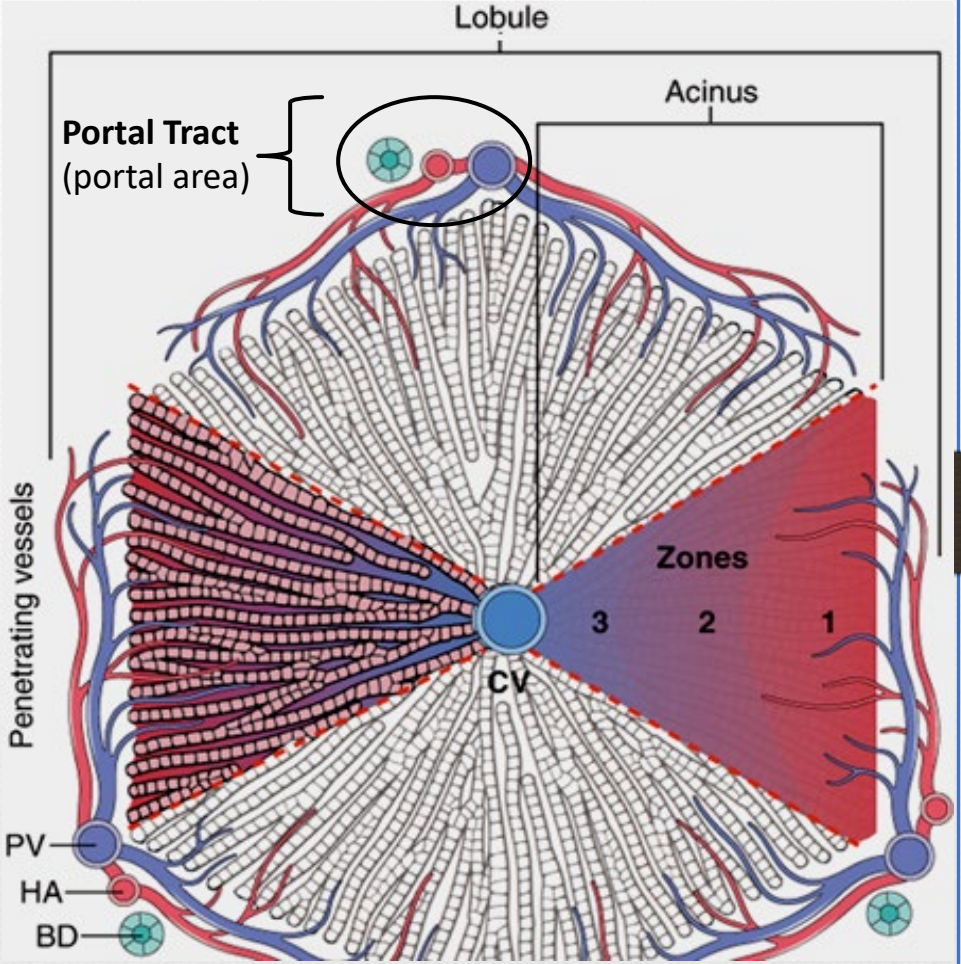


Inferior View

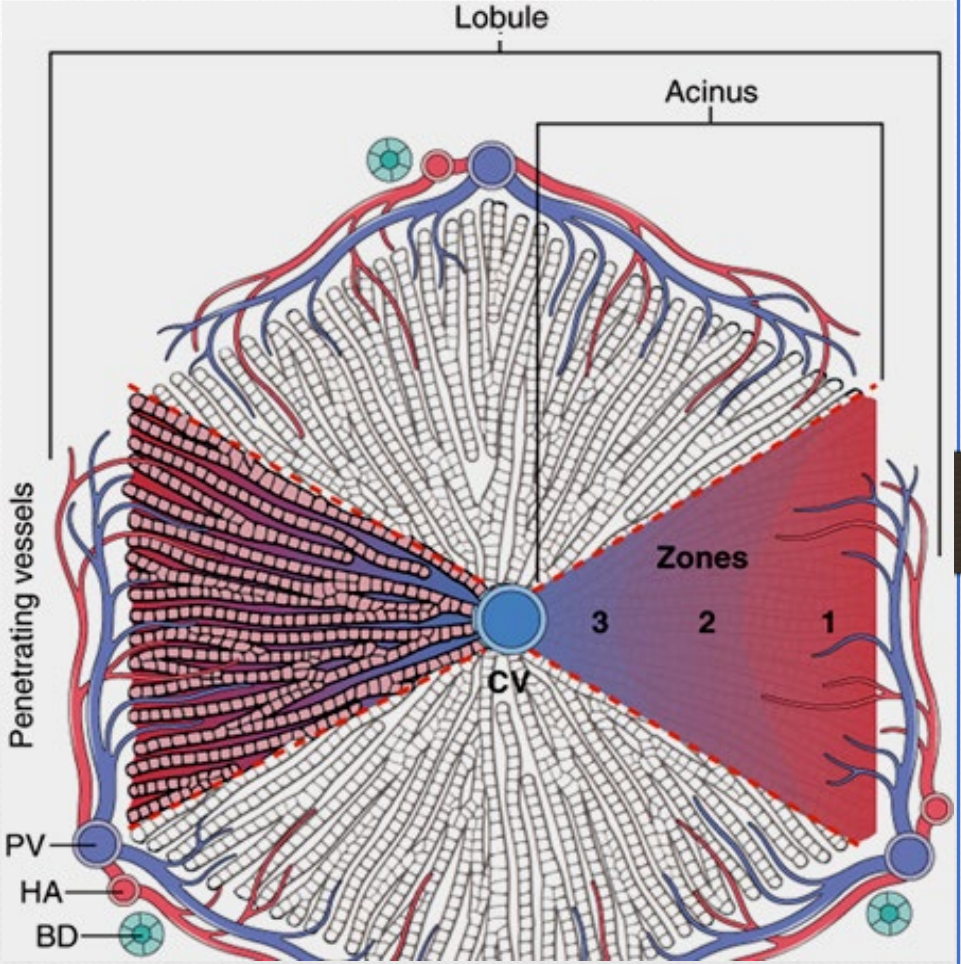

hepatic vein
central vein
sinusoids
portal tract
bile duct
hepatic artery
portal vein

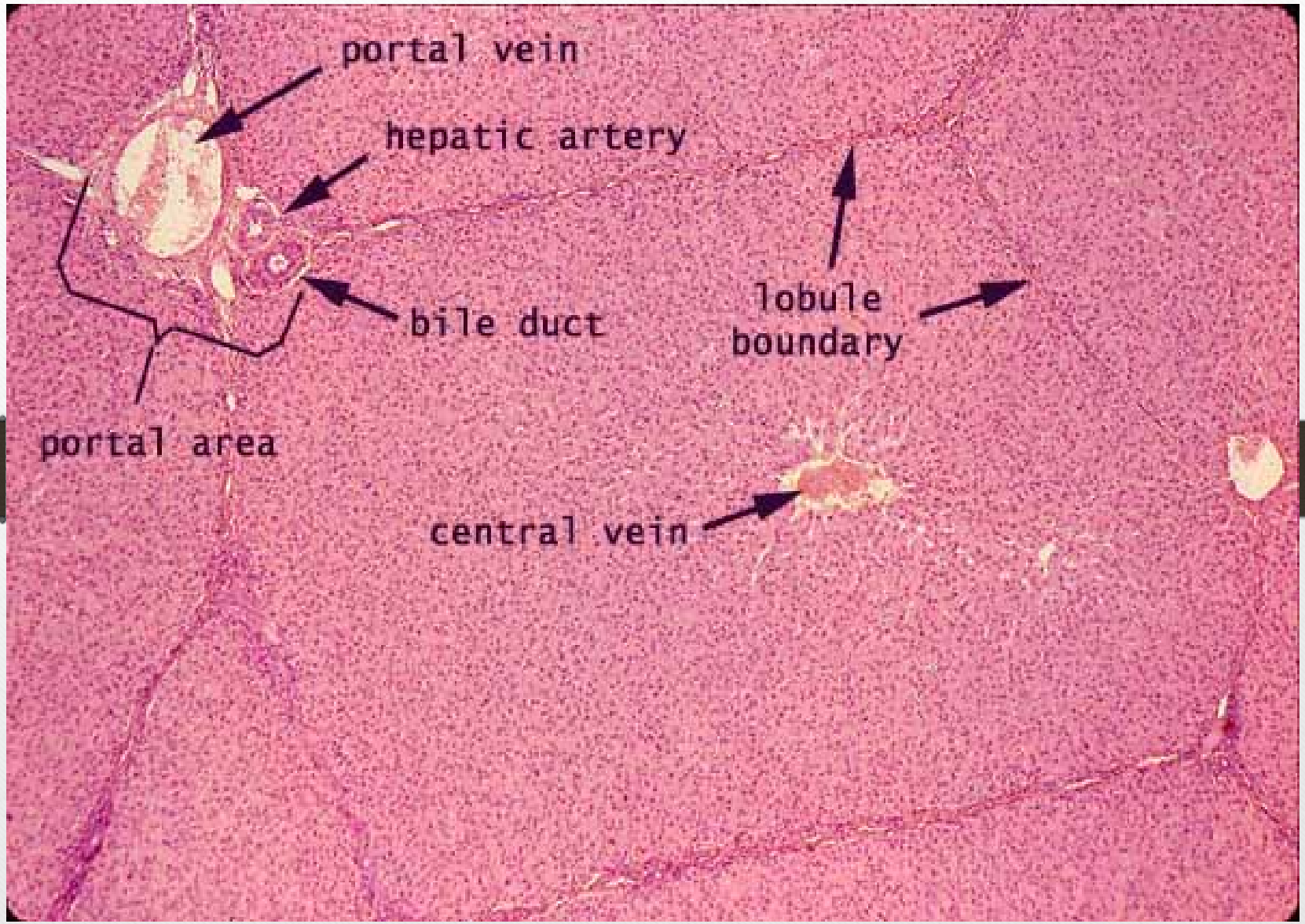
Microscopic architecture of the liver parenchyma

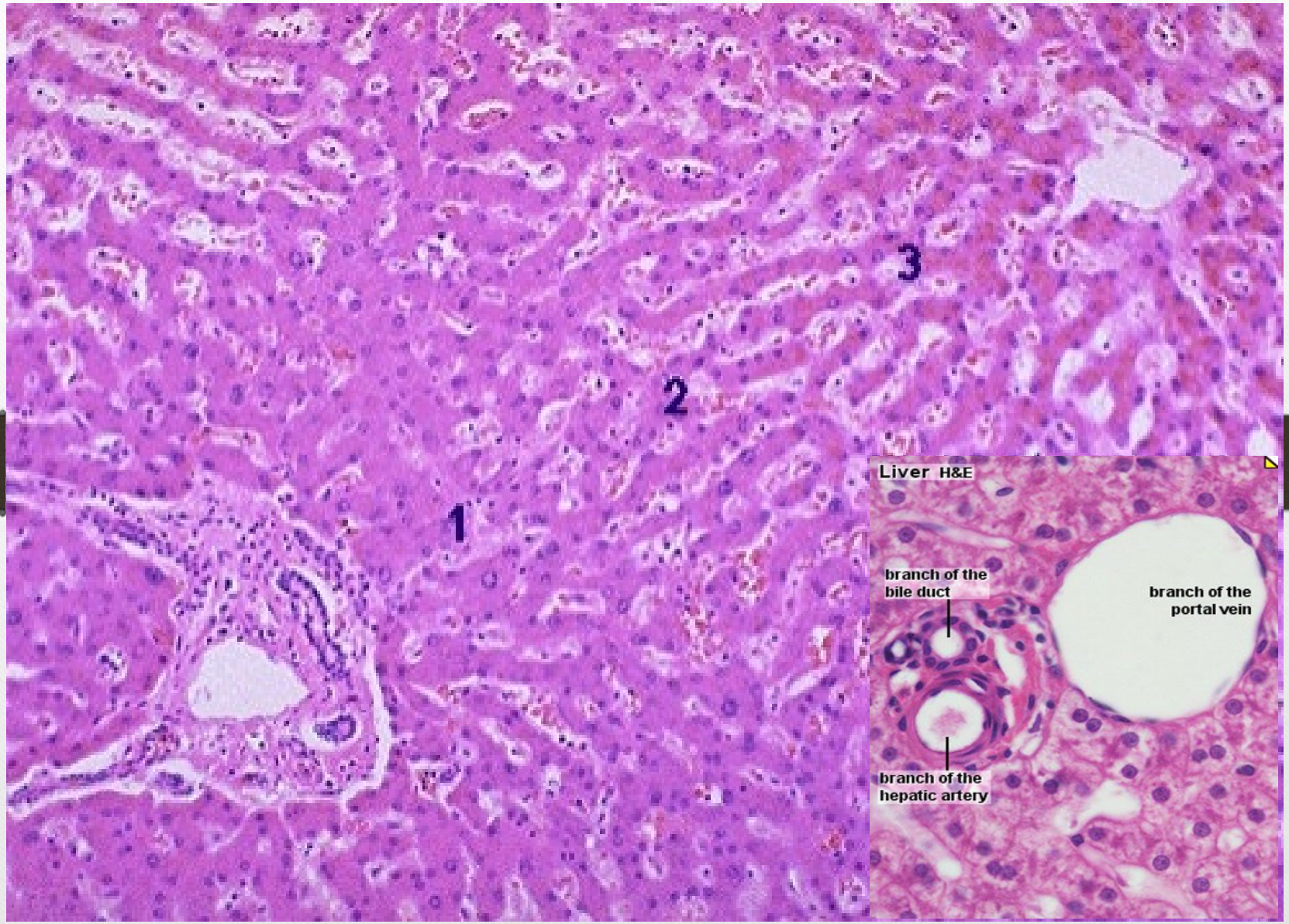
The classic hexagonal **lobule** is centered around a **central vein** (CV), known as terminal hepatic venule, and has **portal tracts** (portal area) at three of its apices. The portal tracts contain branches of the portal vein **(PV)**, hepatic artery **(HA)**, and the bile duct **(BD)**. Regions of the lobule are generally referred as "periportal", "midzonal" and "centrilobular" according to their proximity to portal spaces and central vein.



Blood supply as a source of reference. Using this approach, **triangular acini** can be recognized. Acini have at their base branches of portal vessels penetrate parenchyma "penetrating vessels". On the basis of the distance from the blood supply, the acinus is divided into zones: 1 (closest to blood source), 2, and 3 (farthest from blood source).




portal vein
hepatic artery
bile duct
lobule boundary
portal area
central vein


1
2
3
Liver H&E
branch of the bile duct
branch of the portal vein
branch of the hepatic artery

Liver Changes in CVC

In chronic heart failure, the right side of the heart fails to maintain its output, the inferior vena cava and hepatic veins become congested, and the continued effects of compression by dilated sinusoids and hypoxia lead to its effect on liver cells causing its death.

When venous congestion is prolonged, perivenular loss of hepatocytes may be extensive, and perivenular fibrosis may occur.

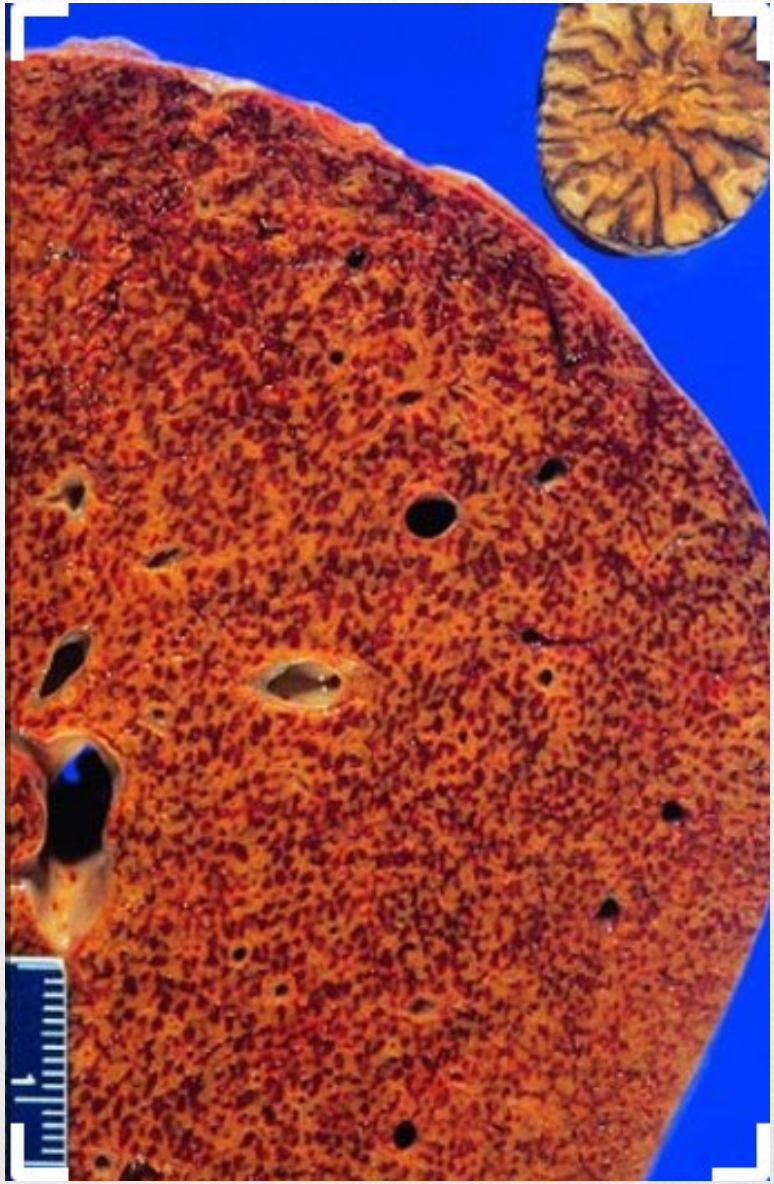
❖ Gross appearance;

(naked eye appearance, macroscopic appearance)

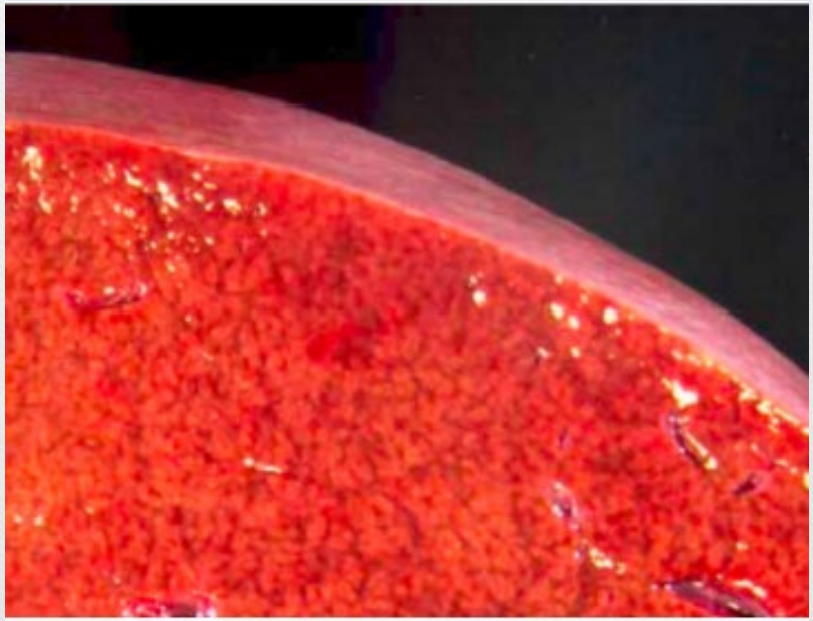
The liver is enlarged and firm in consistency.

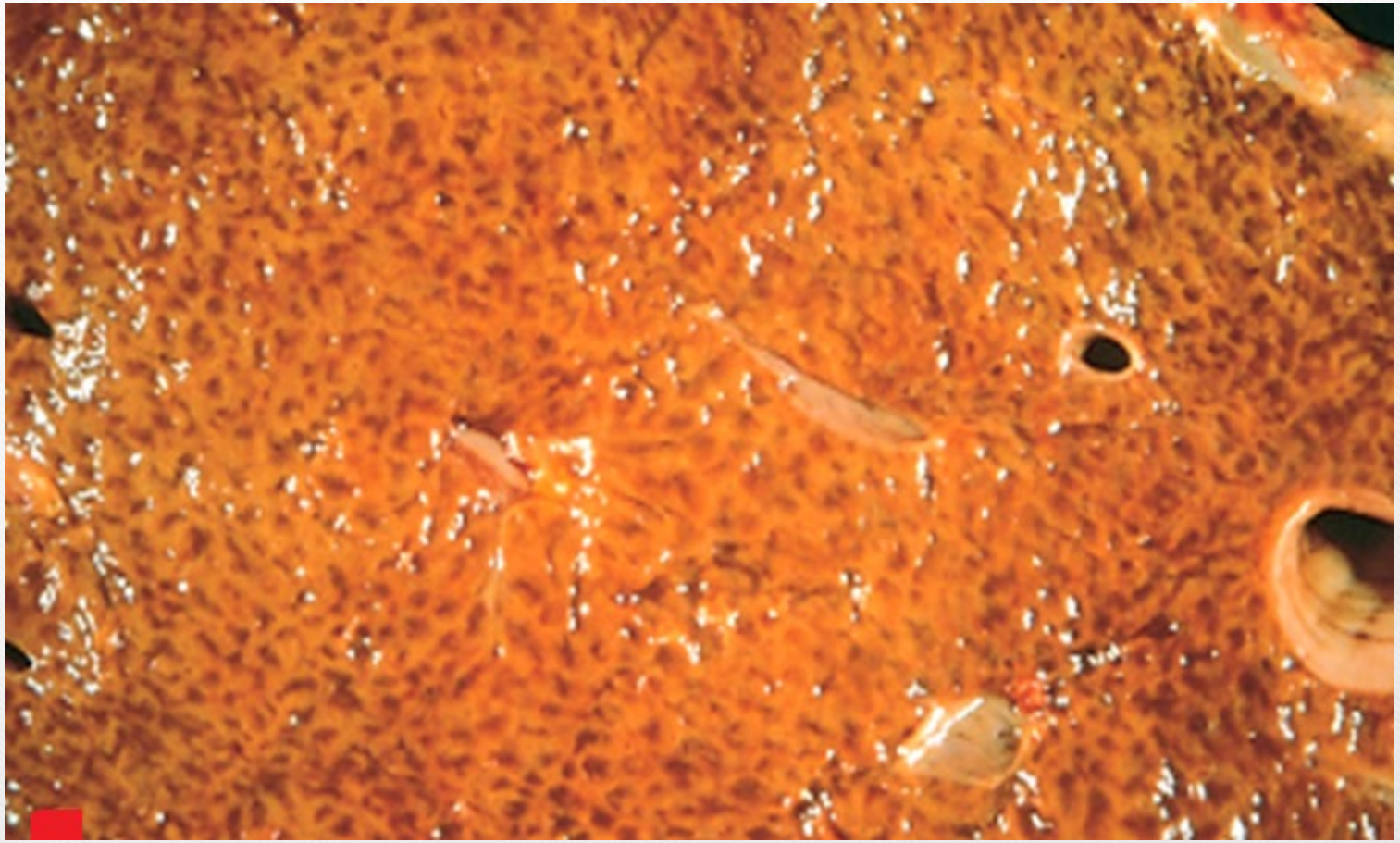
Cut surface shows; a mottled appearance of dark areas (where centrilobular zones are congested by blood), contrasting with pale peripheral (periportal) areas. Therefore, each lobule presents a dark brown center (congestion), and a light yellow periphery (fatty degeneration). This appearance is similar to that of the cut surface of a nutmeg seeds, hence the term **"nutmeg liver".**

If the congestion is unrelieved, there will be necrosis of the anoxic central zones with replacement by fibrosis **(cardiac cirrhosis).** The livers were usually smaller and firmer than the average normal organ.



Nutmeg





Central areas are red and slightly depressed compared with the surrounding tan viable parenchyma, forming a **"nutmeg liver"** pattern (so called because it resembles the alternating pattern of light and dark areas seen when a whole nutmeg is cut).

Liver changes in CVC (cont'd);

❖ Microscopically :

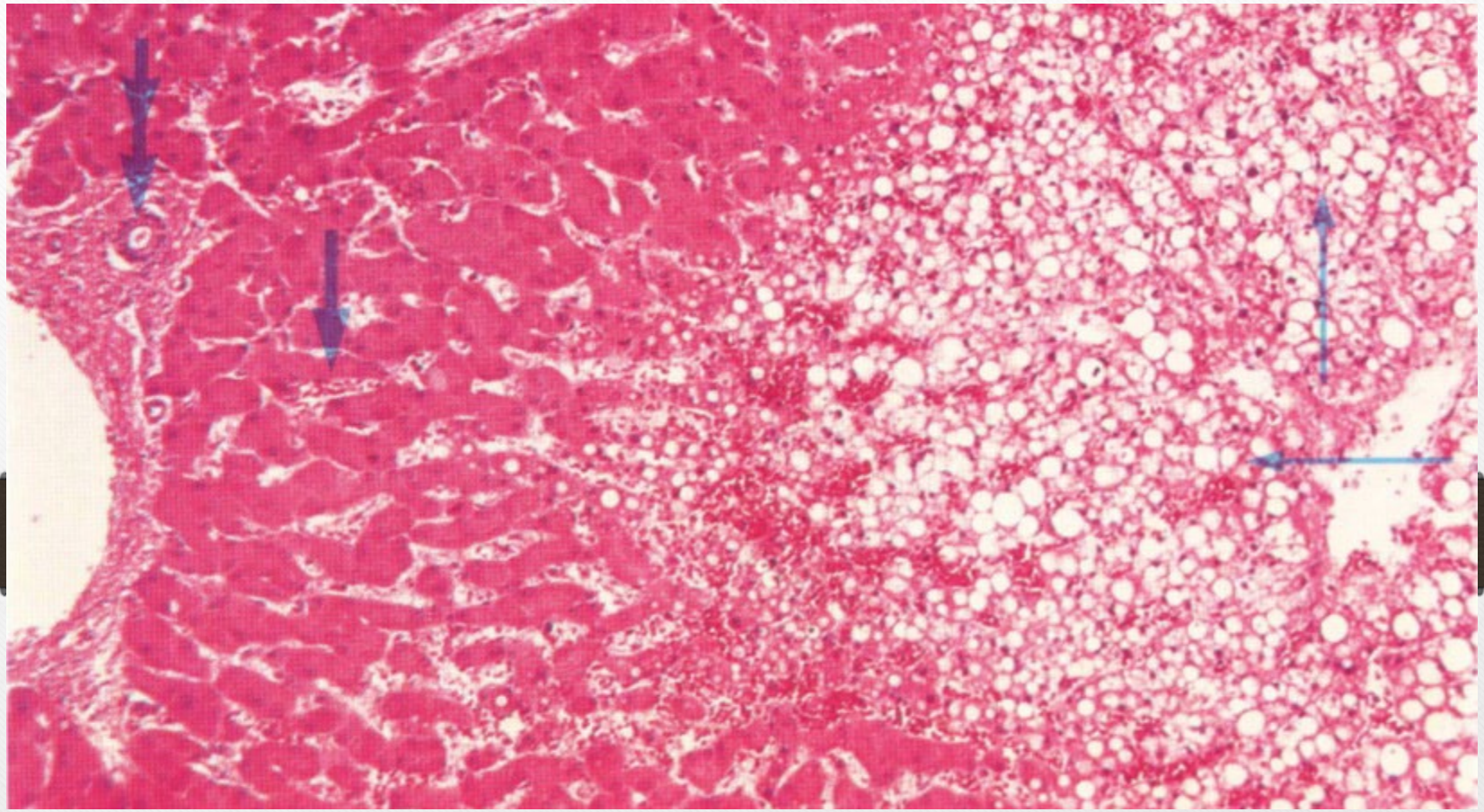
The main changes are noted around the central veins of the hepatic lobules (centrilobular regions).

- The central vein and the central ends of the sinusoids appear **distended** and packed with **red cells**.

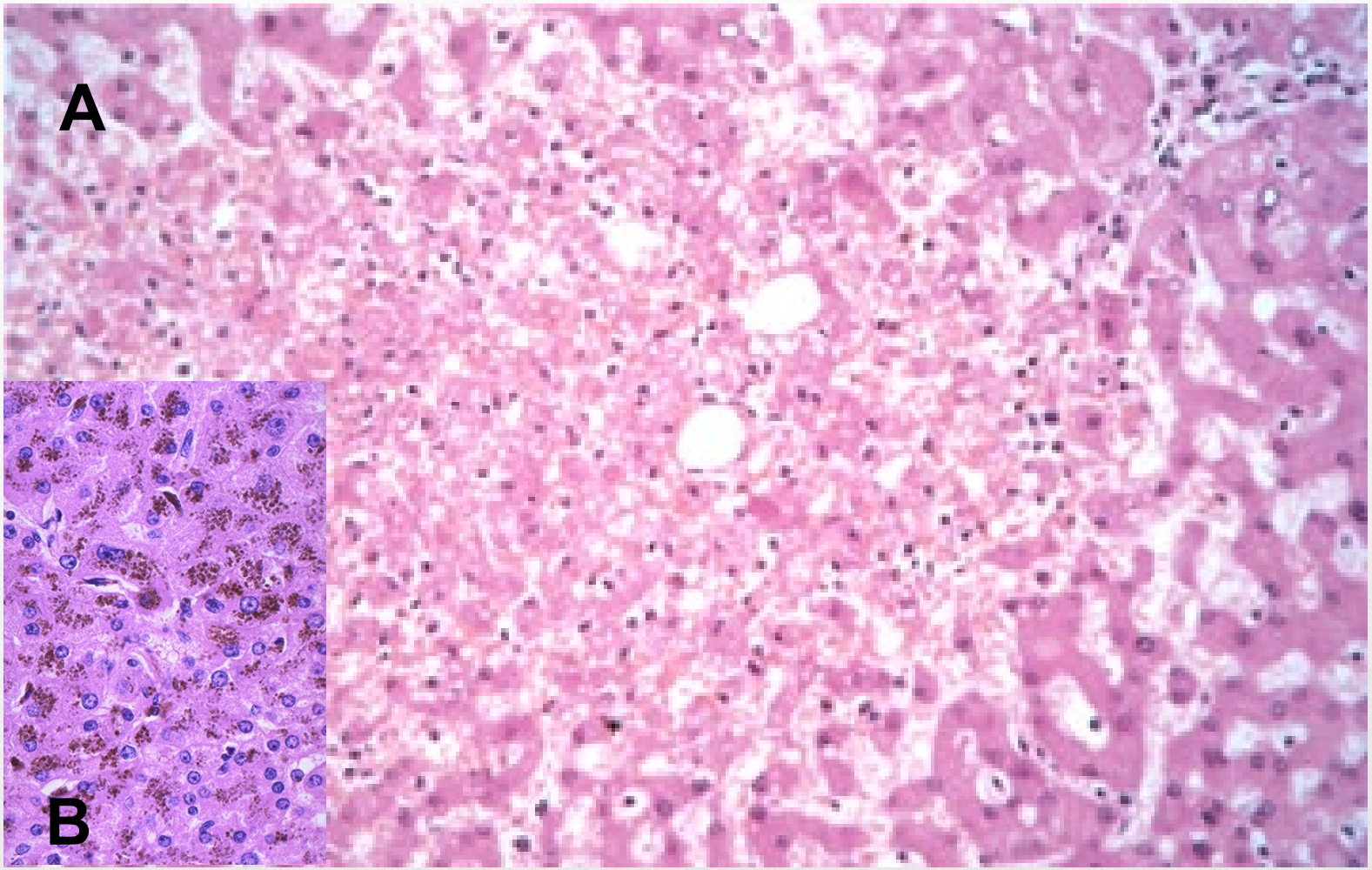
- The hepatic cells in the center of the lobule undergo **degeneration** and sometimes **necrosis** as a result of the anoxia and the pressure effects of congested (dilated) sinusoids.

- The hepatic cells at the periphery of the lobule are either normal or show mild form of injury such as fatty change.

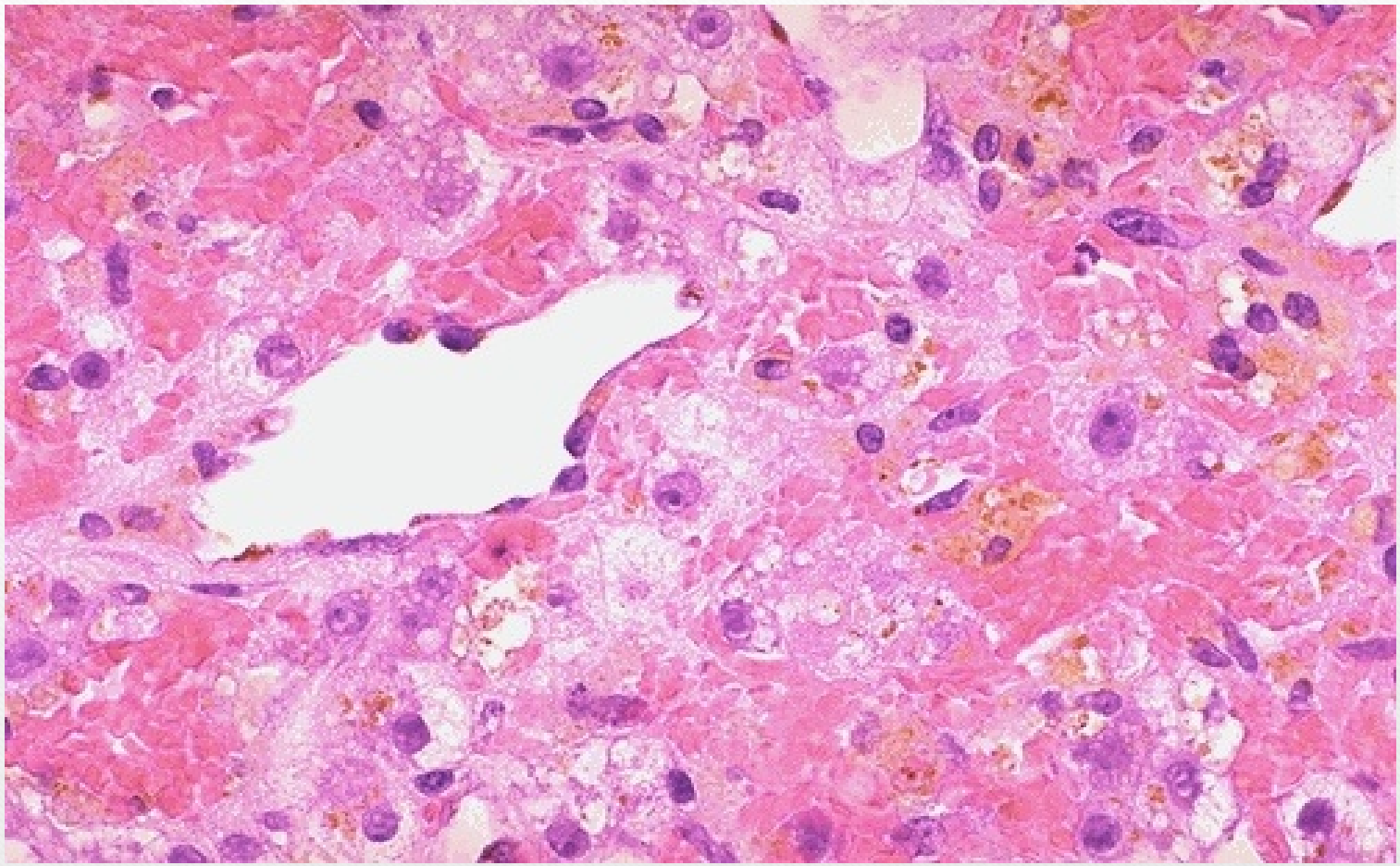
- The hepatocytes and Kupffer cells here are full of granular brown deposits of hemosiderin from accumulation of excess iron in the liver (**hemosiderin-laden macrophages**), may leads to "pigment" cirrhosis.



On the left the hepatocytes are relatively large and normal in appearance, and adjoining them the portal tract shows a dilated branch of the portal vein (double arrow). The sinusoids in the mid-zonal and centrilobular regions are very congested (thick arrow). The centrilobular zone shows severe fatty change (thin arrows) and many hepatocytes are atrophic.



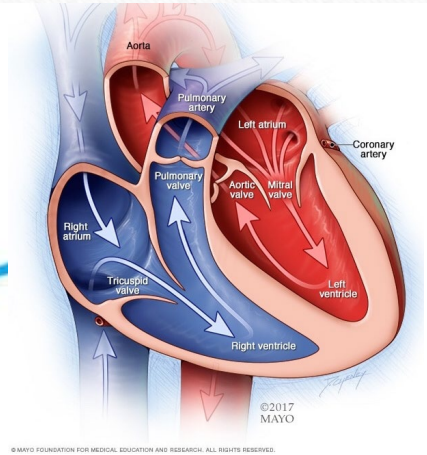
A- Centrilobular necrosis with degenerating hepatocytes and hemorrhage.
B- Hemosiderin-laden macrophages



The light brown pigments seen in the necrotic hepatocytes around the central vein (zone 3) is lipochrome (lipofuscin). Breakdown products within cells from oxidation of lipids and lipoproteins also called “wear and tear” pigments

Morphological Changes in Chronic Venous Congestion

1. Liver.
2. **Lung.**
3. Kidney.
4. Spleen.

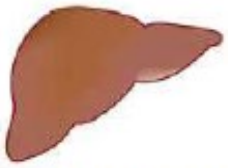


Right-sided failure

Pressure transmitted upstream of right heart

SYSTEMIC VENOUS CONGESTION

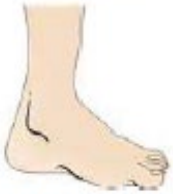
CVC LIVER



CVC SPLEEN

CVC KIDNEY

CONGESTED LEG VEINS

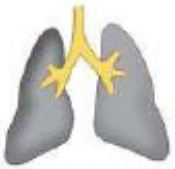


Left-sided failure

Pressure transmitted upstream of left heart

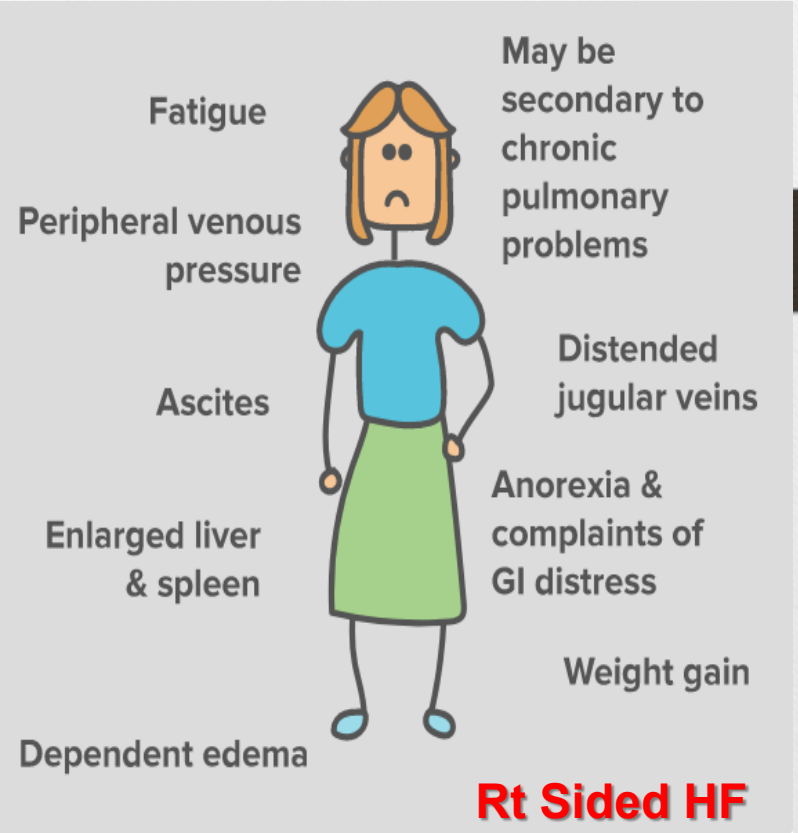
PULMONARY CONGESTION

CVC LUNGS



In **Rt-sided** heart failure, there is an impairment of blood flow to the organs and lungs, either due to **reduced ejection fraction (systolic failure**), The Rt ventricle loses its ability to contract normally. It may occur due to loses of Rt ventricle ability to **relax normally (diastolic failure)**.

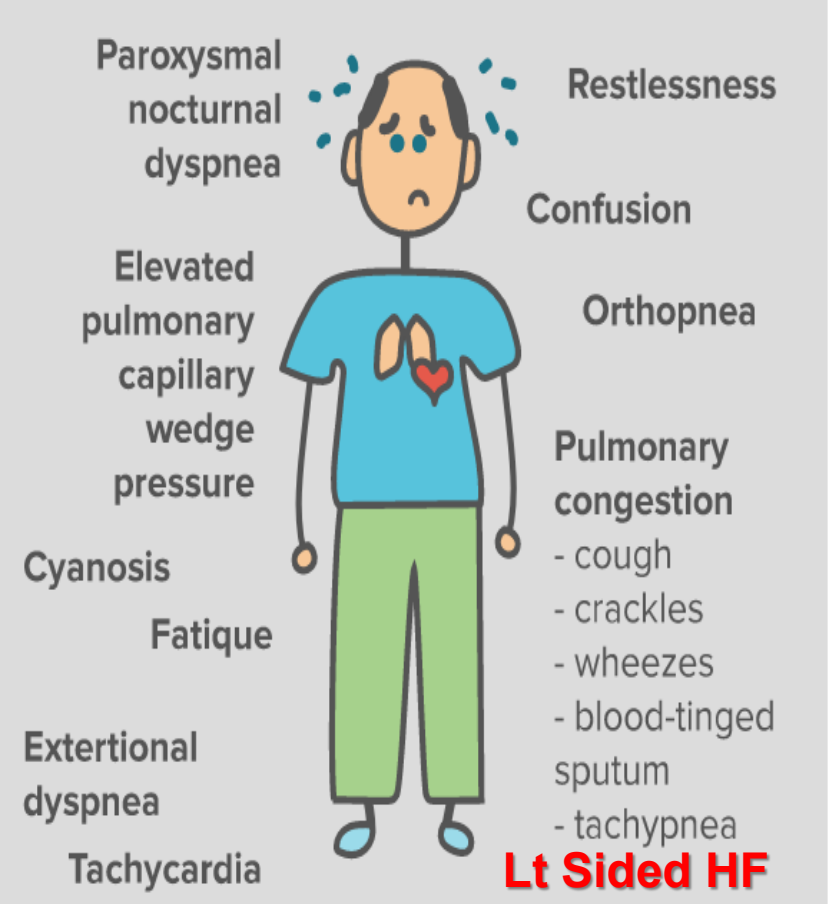
Mostly due to coronary heart diseases or hypertension. The symptoms include; congestion in the body's tissues, often swelling (edema) results. Most often in the legs and ankles, but it can happen in other parts of the body, including lungs, liver and spleen.



In **Lt-sided** heart failure, symptoms are often caused by fluid buildup and impaired gas exchange in the lungs.

In more severe cases, pulmonary hypertension develops and leads to Rt-sided heart failure **(Cor Pumonale).**

Typical symptoms include shortness of breath, rapid breathing, orthopnea (shortness of breath when lying flat) and paroxysmal nocturnal dyspnea (attacks of severe shortness of breath and coughing that generally occur at night)



Chronic Venous Congestion: Lung

❖ Pathophysiology:

Left-sided heart failure, which causes blood to back up into the lungs because the left ventricle is not pumping the blood out.

In consequence of the venous congestion, a **transudate** collects in the alveolar spaces (edema fluid).

The raised blood pressure has caused the alveolar capillaries and venules to become **dilated, tortuous and packed with red cells**.

The distended capillaries are usually ruptured with **intra-alveolar hemorrhage**

A large population of macrophages has escaped into the alveoli and ingested the erythrocytes, with the release of hemosiderin (iron-containing salts) within their cytoplasm (hemosiderin laden macrophages).

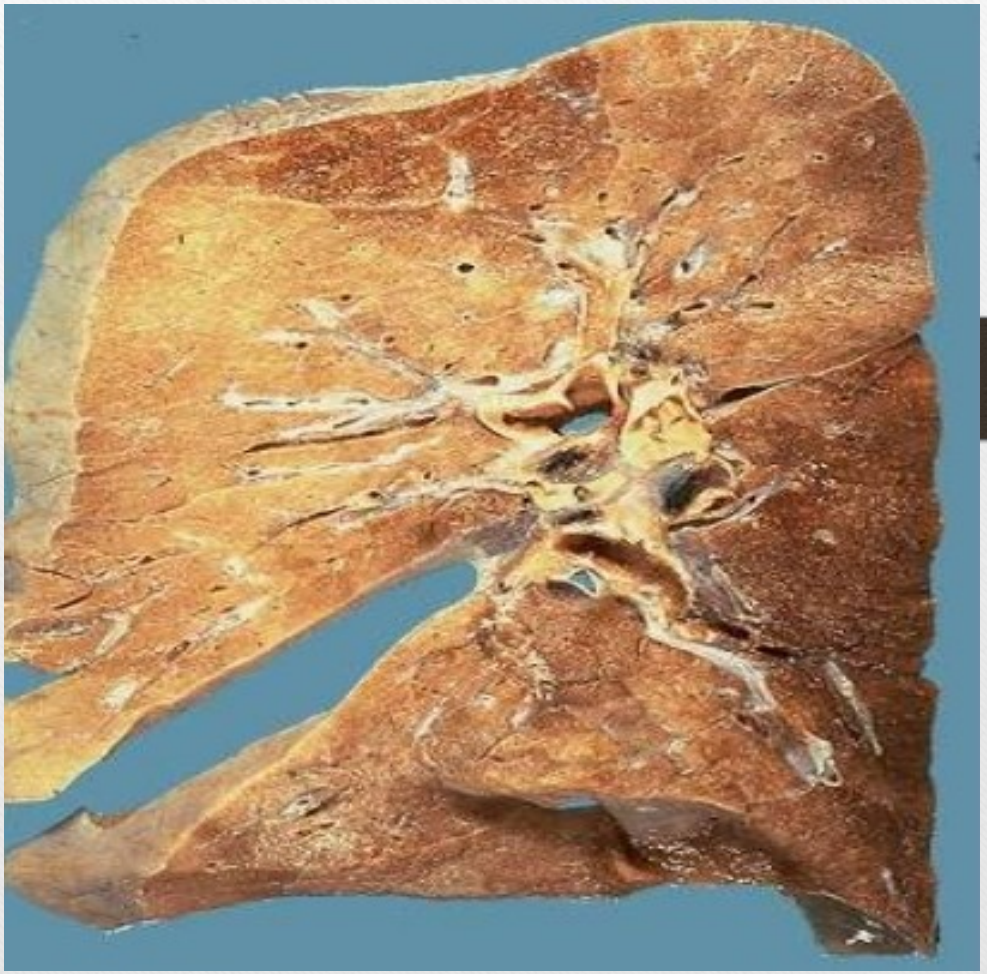
These cells also tend to congregate around the respiratory bronchioles, and they may release the hemosiderin which is deposited in the connective tissue fibres of the lung.

This deposition with its fibrogenic effects leads to increases the amount of connective tissue in the lungs, reflected by a **brown induration**.

Pathological Changes;

❖ Gross :

- The lungs are heavy and firm in consistency.
- Appear reddish-blue, brown, darkly pigmented, **brown induration of lungs**, due to red cell stasis and oxyhemoglobin.
- The section of lung is from a person with mitral stenosis and long-standing pulmonary venous hypertension.



❖ Microscopically:

A **transudate** edema fluid, in the alveolar spaces which appears pale red homogeneous material.

Fine strands of **fibrin** seen within the edematous fluid.

The **alveolar capillaries** and venules are dilated, tortuous and packed with red cells.

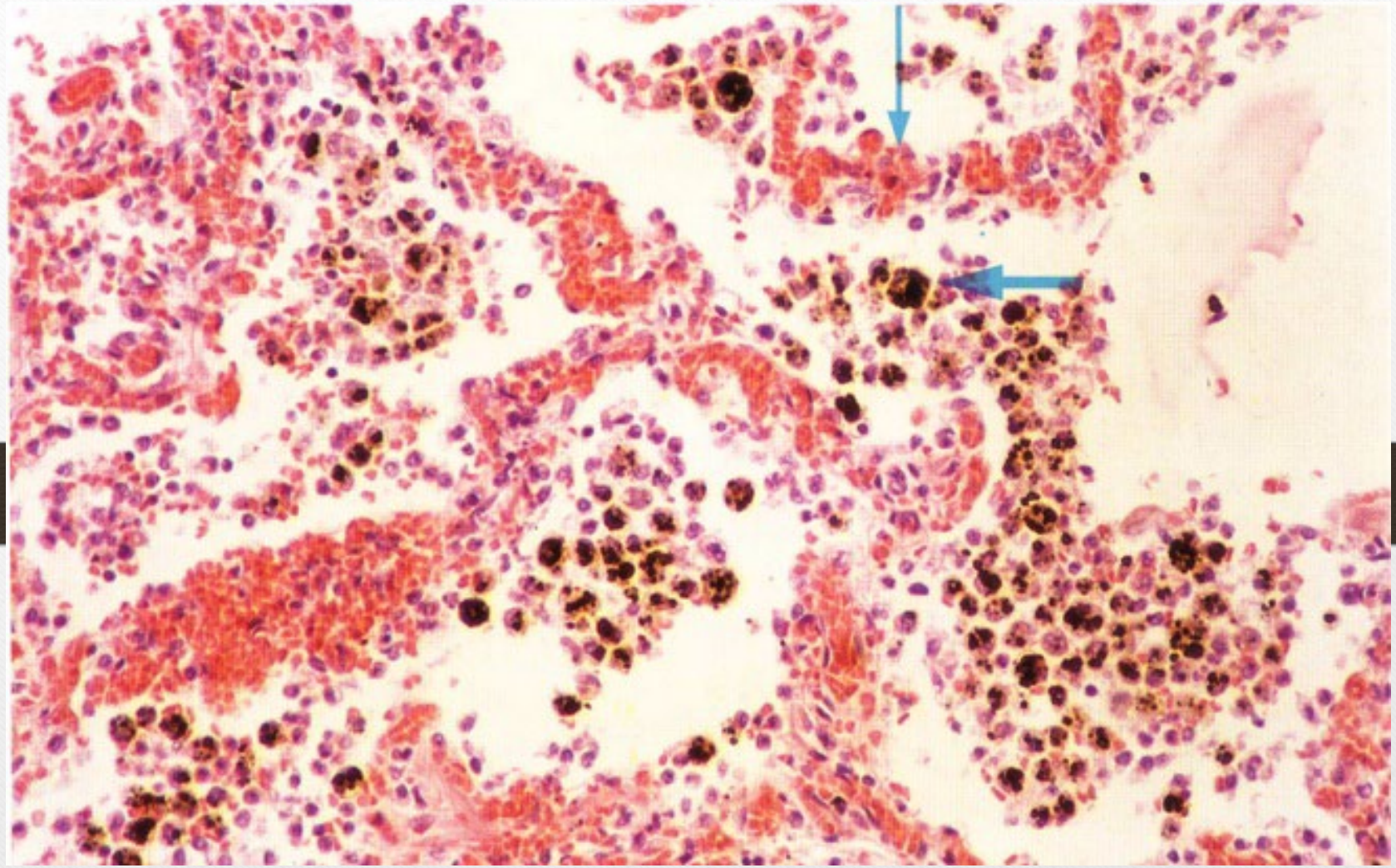
Presence of red blood cells, due to the **intra-alveolar hemorrhage**.

Hemosiderin-laden macrophages, largest cells which contain hemosiderin pigment in their cytoplasm are noticed in the alveolar spaces and within the alveolar wall. Also called **heart failure cells** because of the association of their presence and heart failure.

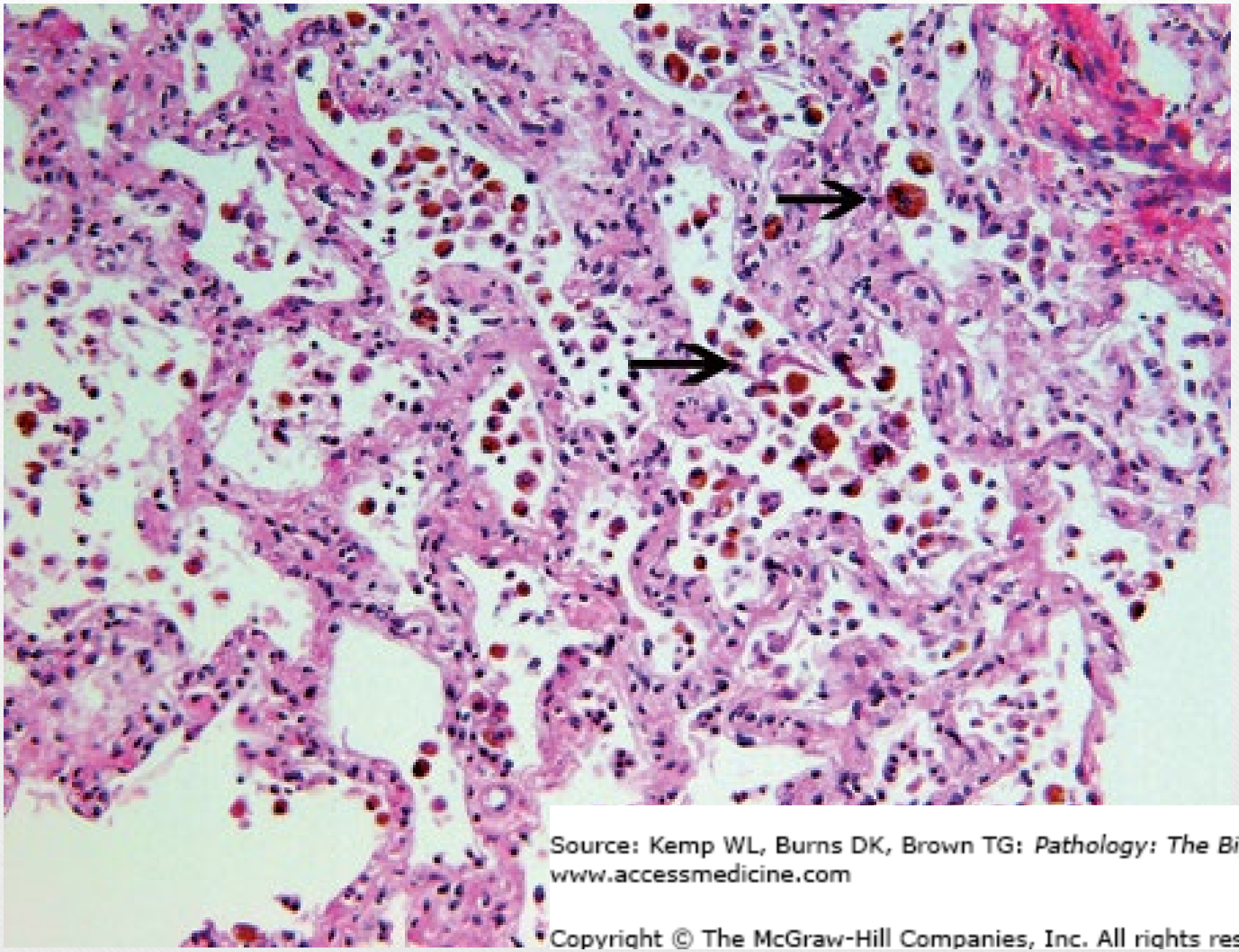
Free granules of hemosiderin may be present.

A **fibrotic** element within alveolar walls may also present.

- Rupture of congested vessel results in edema & hemorrhage.
- The alveolar septa appears thickened and fibrotic. The alveoli are dilated and contain edema fluid and RBC's and macrophages.
- Lysis of RBC's releases hemosiderin pigment which is taken up by macrophages in the alveoli forming the hemosiderin laden macrophages; **heart failure cells.**



Dilated alveolar bd vessels, packed with red cells (thin arrow), escaped RBCs, and Hemosiderin-laden macrophages (thick arrow).



Source: Kemp WL, Burns DK, Brown TG: *Pathology: The Big Picture*: www.accessmedicine.com

Hemosiderin-laden macrophages (arrows) with fibroblast cells proliferation and fibrosis of the alveolar wall.

B- localized venous congestion:

- **Pulmonary venous congestion**
- **localized venous congestion**

- Pulmonary venous congestion

. Left sided heart (ventricular) failure

. Mitral valve stenosis

Chronic pulmonary venous congestion is complicated by the development of pulmonary arterial hypertension, which often leads in time to right ventricular failure and hence to systemic venous congestion **(Cor Pumonale).**

Congestion and edema: lung

Can be acute or chronic forms.

In **acute** condition, which due to the failure of the left ventricle, causes the blood pressure to rise abruptly in the pulmonary veins, and passive congestion in these vessels.

The lungs becomes heavy and edematous.

In more severe cases, pulmonary hypertension develops and leads to failure of the right side of the heart.

The patient died of **acute left ventricular failure.**

The lungs were edematous and there was also frothy fluid in the air passages.

❖ Microscopically:

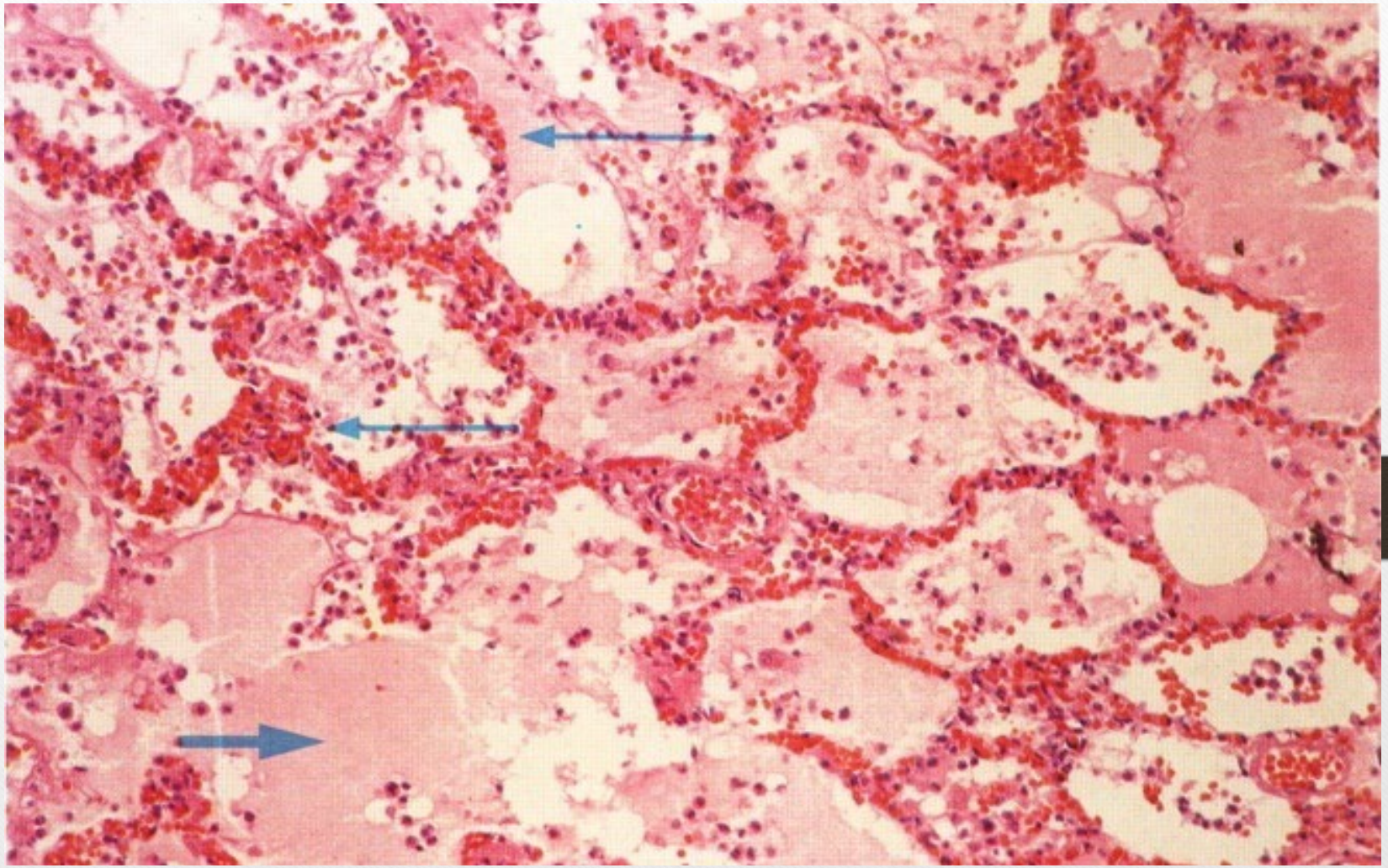
The small veins in the lung are swollen, and the capillaries in the alveolar septa are markedly distended and tortuous.

The pulmonary alveoli are full of amorphous eosinophilic fluid which has leaked from the congested capillaries.

A eosinophilic strands (probably fibrin) are also present in the lumen of some of the alveoli.

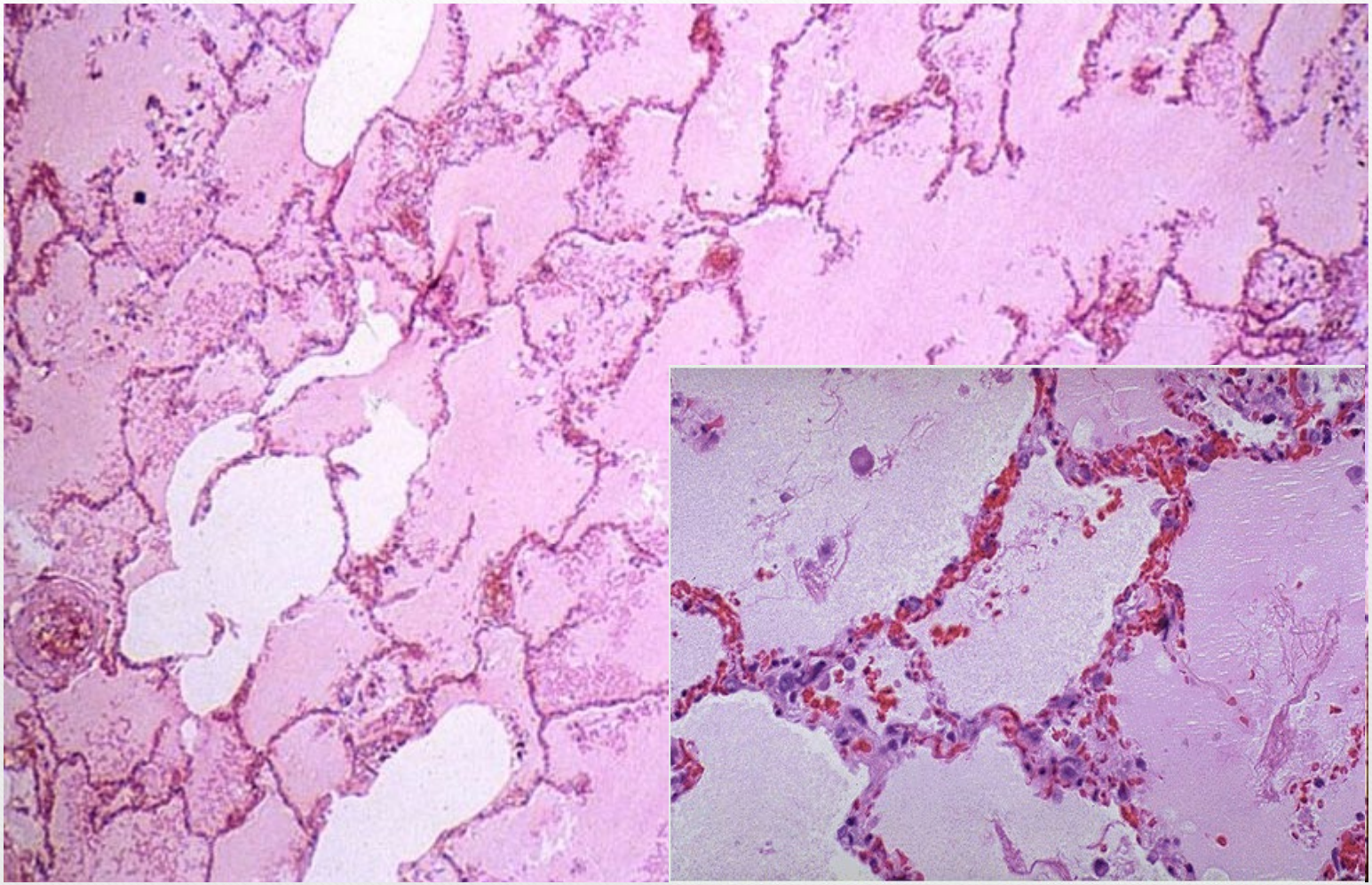
There are also fairly numerous leukocytes (predominantly polymorphs) within the alveoli and in the alveolar septa. The fluid in the lungs is a **transudate**, which is **usually cell-free.**

The polymorph leukocytes in the fluid within the alveoli suggest therefore that **broncho-pneumonia** was starting to develop.



The pulmonary alveoli are full of amorphous eosinophilic fluid (thick arrow) with polymorphs and few eosinophilic strands (fibrin?). Distended alveolar vessels (thin arrows).

Pulmonary edema (Acute left heart failure)



The alveoli are filled by amorphous pinkish edema fluid with congestion of capillaries within the alveolar septa.


شكراً جزيلاً
ngiyabonga
рахмат
danke
謝謝
spas
teşekkür ederim
Баярлалаа
mersi
kia ora
barka
welalin
tack
mahalo
tapadh leat
спасибо
faafetai lava
vinaka
спасибі
blagodaram
dank je
misaotra
paldies
grazzi
matondo
kiitos
dankie
nanni
nandri
dhanyavad
thank you
gracias
хвала
asante
manana
obrigada
tenki
bedankt
bayarlalaa
gracie
hvala
mauruuru
köszönöm
dankon
aciū
akun
djiere dieuf
tau
mochchakkeram
murakoze
enkosi
dziękuję
дякую
mamnun
chokrane
chnorakaloutioun
grafias ago
gràcies
sulpáy
taiku
go raibh maith agat
sobodi
dėkuji
sagolun
sukriya
kop khun krap
arigatô
takk
dakujem
trugarez
obrigado
mèsi
didi madloba
ありがとう
tanemirt
grazie
najis tuke
rahmet
terima kasih
kam sah hamnida
rahmat
diolch
dhanyavadagalu
shukriya
merce
мерси
তোমাকে ধন্যবাদ
감사합니다
xièxie
ευχαριστώ
merci