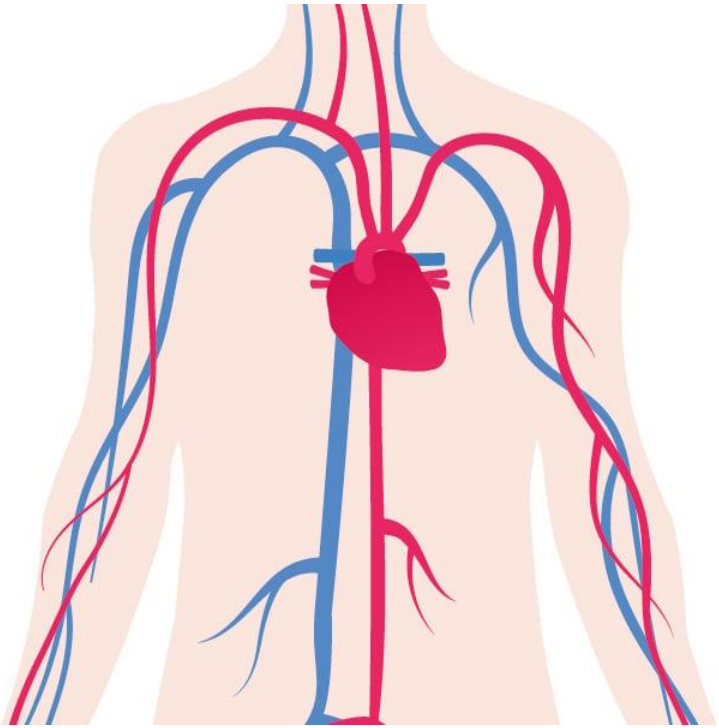
HEMODYNAMIC DISORDERS



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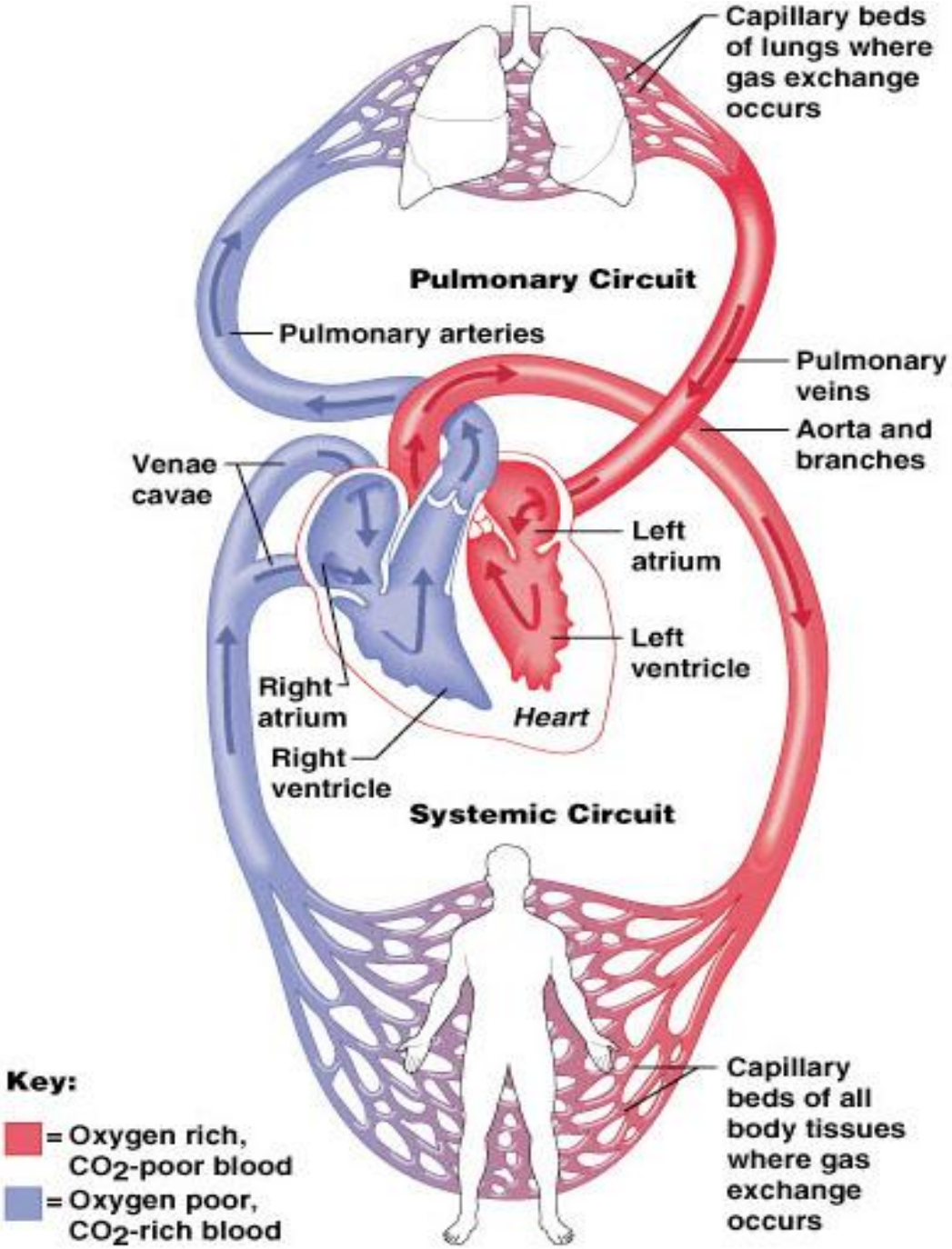
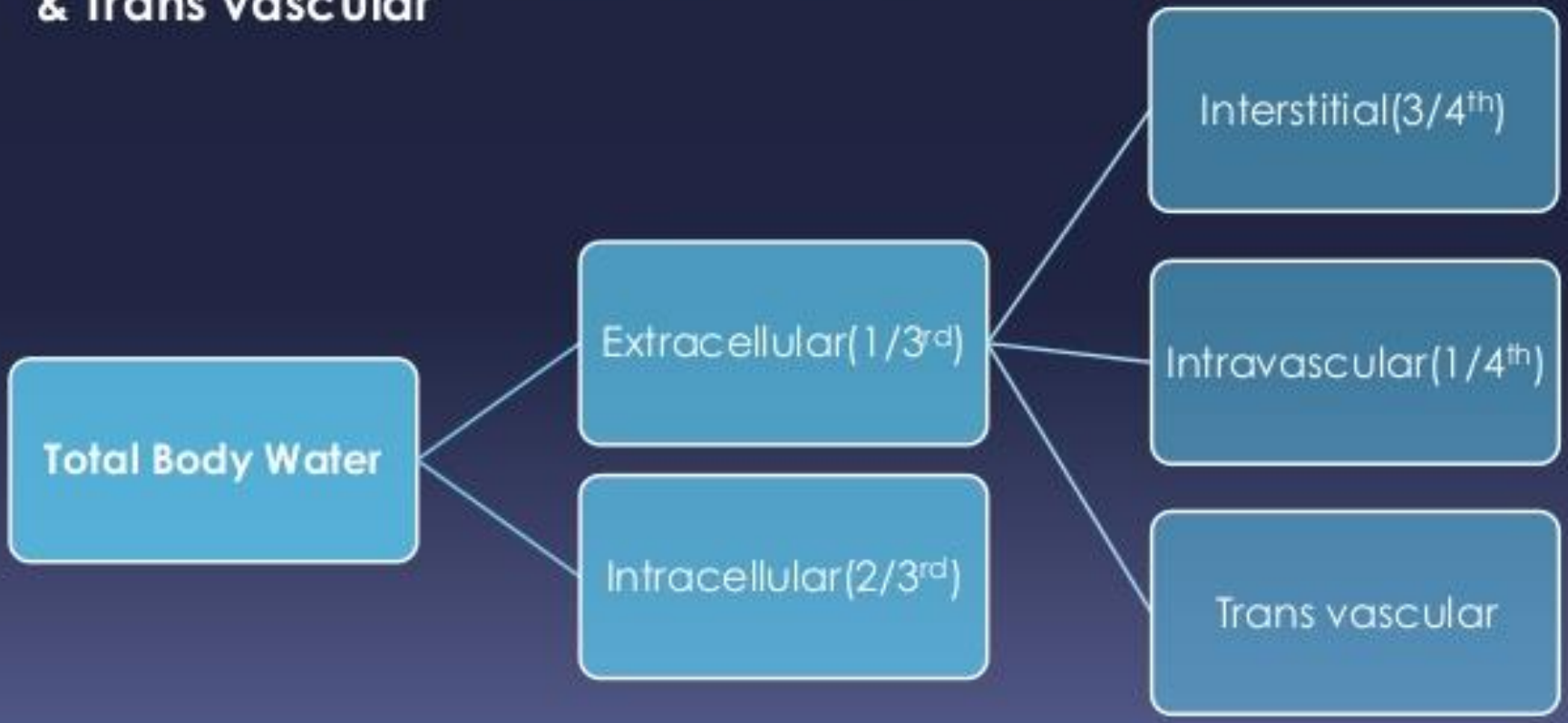

Capillary beds of lungs where gas exchange occurs
Pulmonary Circuit
Pulmonary arteries
Pulmonary veins
Aorta and branches
Venae cavae
Left atrium
Left ventricle
Right atrium
Heart
Right ventricle
Systemic Circuit
Key:
= Oxygen rich, CO_2-poor blood
= Oxygen poor, CO_2-rich blood
Capillary beds of all body tissues where gas exchange occurs

Approximately **60%** of lean body weight is water; **two thirds** of this water is intracellular, and the remainder **one third** is found in the extracellular space (mostly as interstitial fluid) and only about **5%** of total body water **(60%)** is in blood plasma.

Edema: Is a pathological accumulation of excess fluid in the interstitial tissue spaces or it is a fluid accumulation in the body cavities in excessive amount.

GENERAL

- 60% of body's weight is water.
- 2/3rd of it is INTRACELLULAR and 1/3rd is EXTRACELLULAR.
- EXTRACELLULAR is further divided into Intravascular, Interstitial & Trans vascular



- **Edema formation** is determined by the following factors:

1) Hydrostatic pressure
2) Oncotic pressure
3) Vascular permeability
4) Lymphatic channels
5) Sodium and water retention

Physiology of fluid movement

There are **four** primary forces that determine fluid movement across the capillary membrane. These four primary forces are known as **Starling forces** & they are:

1. The capillary hydrostatic pressure. This pressure tends to force fluid outward from the intravascular space through the capillary membrane to the interstitium.
2. The interstitial fluid hydrostatic pressure. This pressure tends to force fluid from the interstitial space to the intravascular space.

3. The capillary (plasma) colloid osmotic (oncotic) pressure. This pressure tends to cause osmosis of fluid inward through the capillary membrane from the interstitium. The plasma oncotic pressure is caused by the presence of plasma proteins.
4. The interstitial fluid colloid osmotic (oncotic) pressure. This pressure tends to cause osmosis of fluid outward through the capillary membrane to the interstitium.

Pathogenesis of edema

i. **Increase hydrostatic pressure**: which could be

a. **generalized** :

(cardiac) right ventricular failure or combined right and left ventricular failure: because of insufficient pumping power of the heart, there is accumulation of blood that is reflected back into the systemic veins as an increase in systemic venous pressure.

(renal) Decrease renal blood flow which stimulate juxta glomerular apparatus to activate the **rennin-angiotensin-aldosterone system** that result in the secretion of excess of rennin this in turn enhances the secretion of **aldosterone by the adrenal cortex** with consequent reabsorption of sodium Na by the renal tubules. The **reabsorption of sodium activate the secretion of antidiuretic hormone** from the hypothalamus and so more water will be absorbed.

b- localized which is seen in :

- left ventricular failure (pulmonary edema),
- thrombosis of major veins,
- pressure of gravid uterus,
- pressure of tumor on the surrounding vessels
- incompetent venous valve (varicose vein)

ii. decrease in colloid oncotic pressure of plasma: seen in

a-Nephrotic syndrome (protein loss)

b- Liver disease (decrease protein synthesis)

c- Malnutrition (decrease protein intake)

d- Protein-losing gastroenteropathy

iii. **Lymphatic obstruction**: could be occurring due to:

- tumor,
- post-surgical,
- post radiation
- parasitic infection (filariasis) that causes lymphatic obstruction.

iv. increased capillary permeability as in

- inflammation,
- sever allergy and anaphylactic shock

Types of oedema

Classification:

1) According to pathophysiological mechanism:
    a) Transudate (low protein content)
    b) Exudate (high protein content)
2) According to location:
    a) Localized
    b) Generalized
3) According to clinical finding:
    a) Pitting
    b) Non-pitting.

According to the composition of the accumulated fluid edema divided to:

Non-inflammatory edema (Transudate):

- Serous protein-poor fluid < 2.5 gm/dl.
- Resulting from increase in plasma hydrostatic pressure (congestion) within intravascular compartment and decrease in plasma oncotic pressure.
- It has specific gravity below 1.012
- This type of edema can be encountered in heart failure and nephrotic syndrome.

Inflammatory edema (Exudate): this differs from transudate in:

- Protein rich: > than 2.5 gm/dl
- Resulting from inflammation-induced increased permeability and leakage of plasma proteins especially albumin.
- it has specific gravity above 1.012
- seen in inflammation and allergic reaction

According to the location:

- Generalized: edema all over the body as in heart failure, renal failure
- Localized: edema occurs in limited area due to impaired venous return

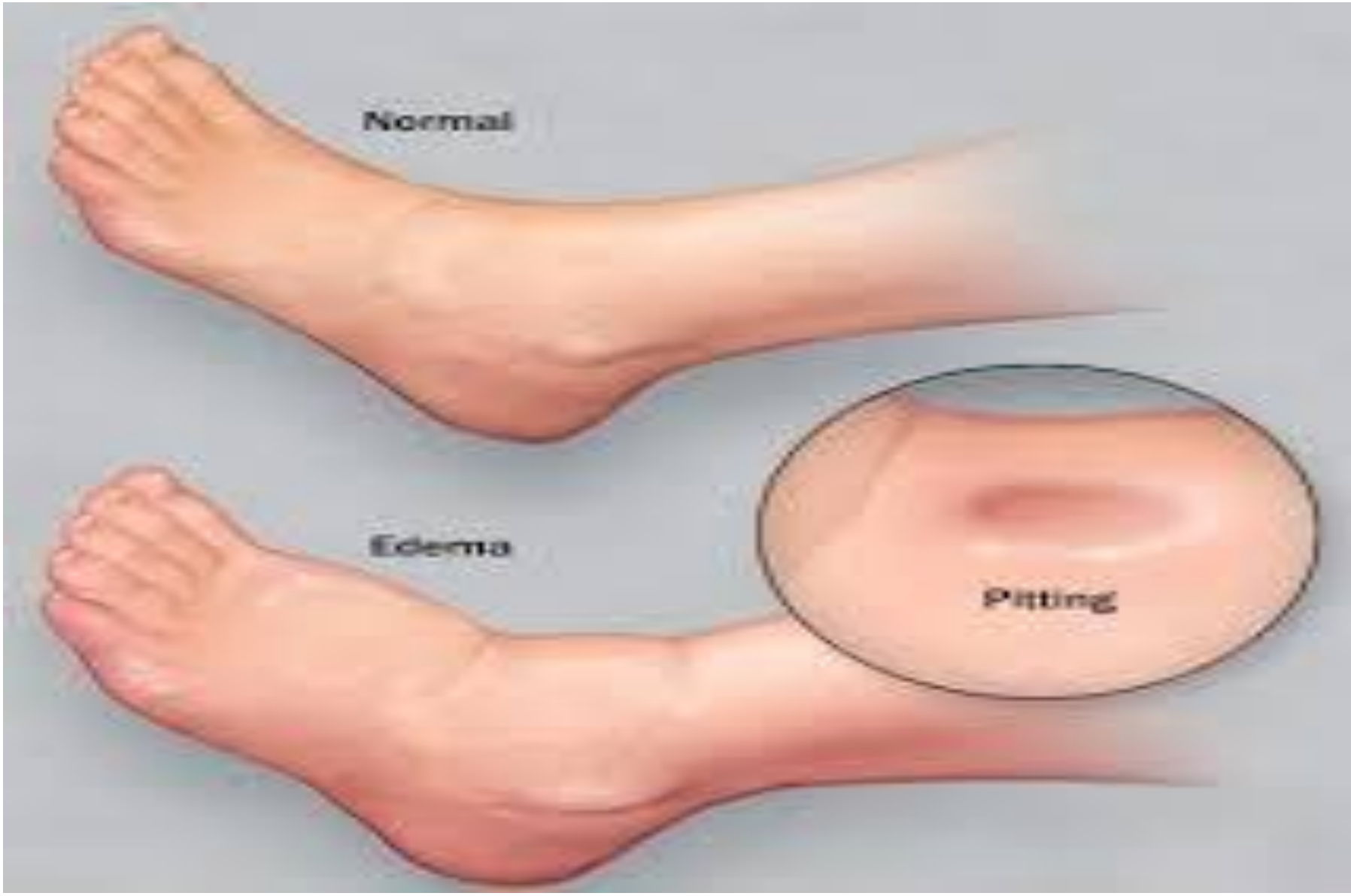
1. Acute left ventricular failure
2. Thrombosis of major veins
3. Valve incompetence
4. Tumor pressure on the adjacent vessels

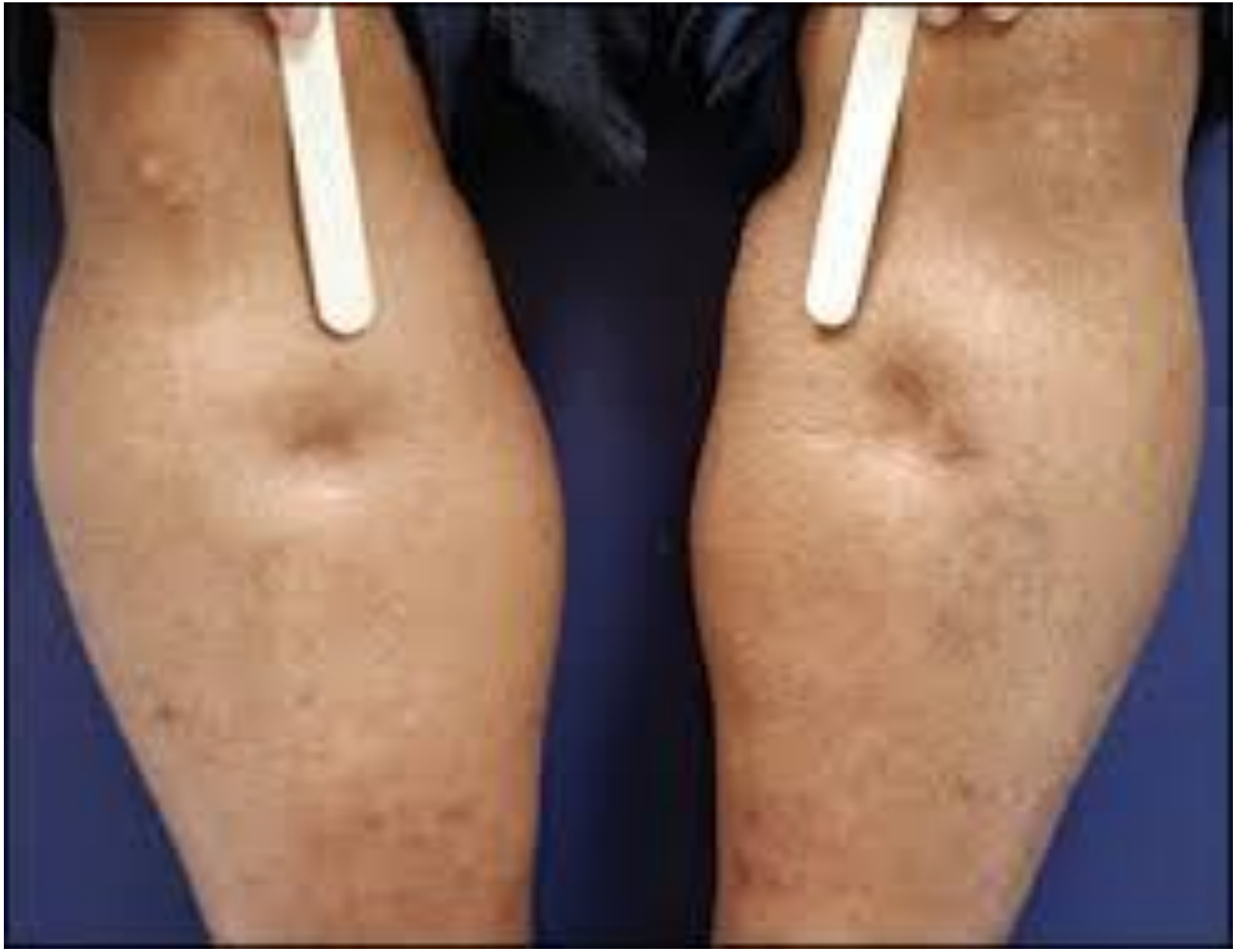
According to clinical finding:

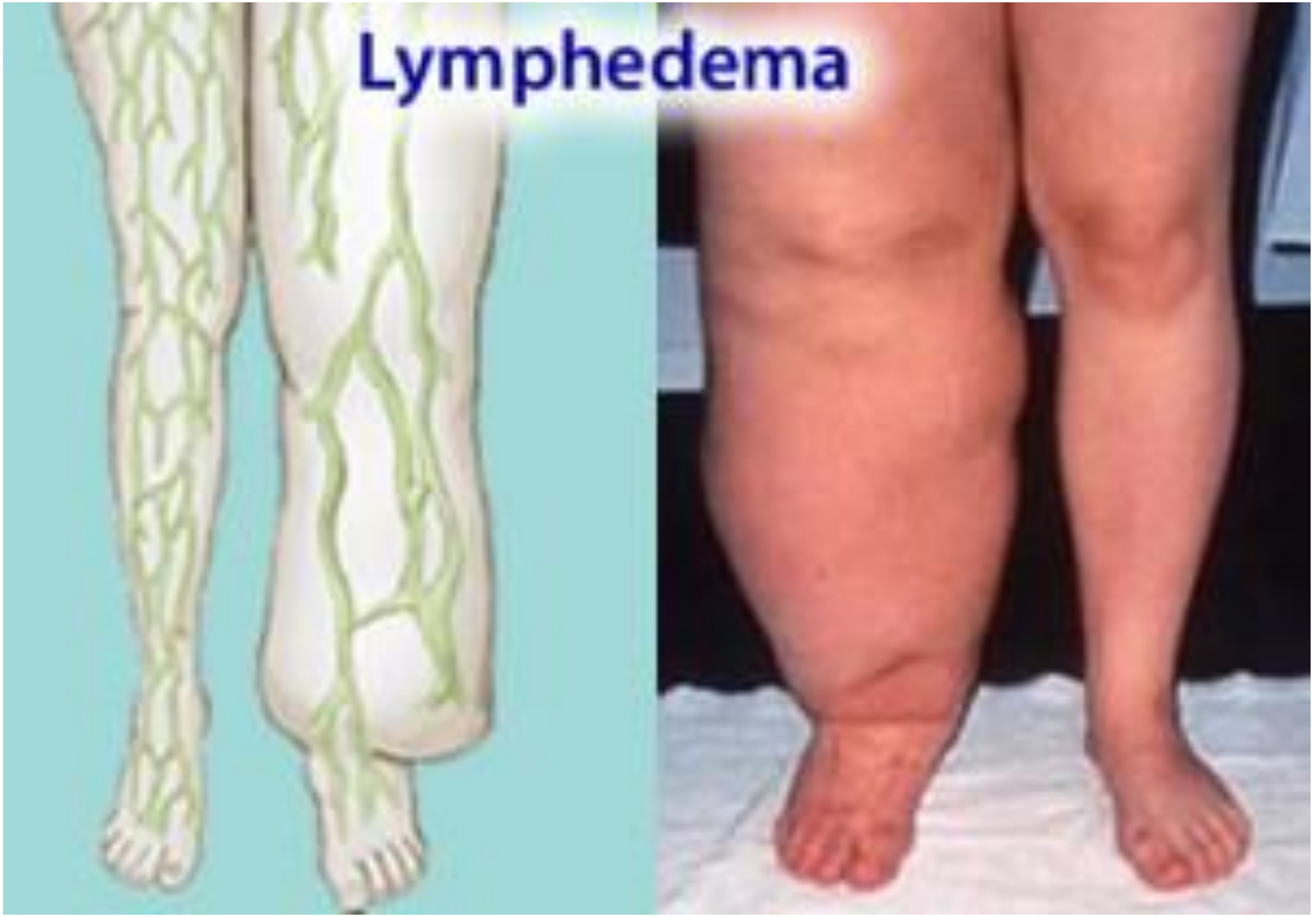
- Pitting edema: fluid can easily move when pressing the affected part (usually occurs in transudate including the causes of generalized and localized edema)

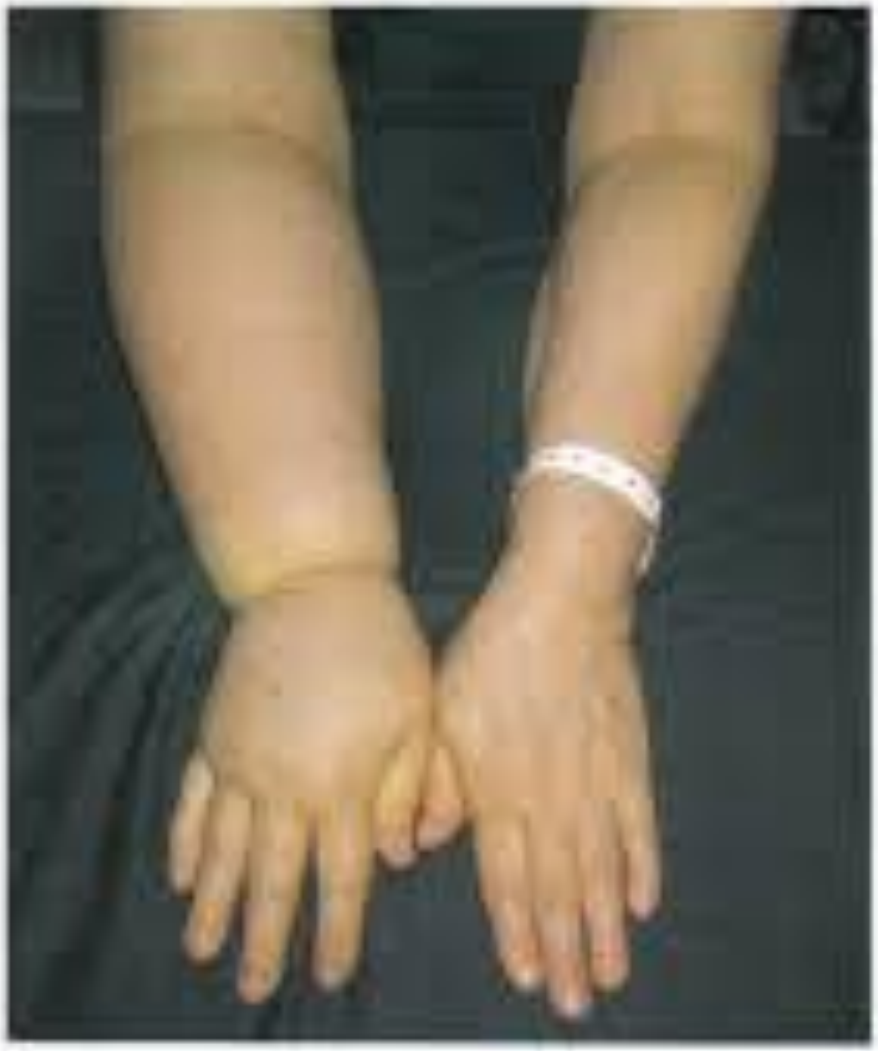
- Non pitting edema: fluid cannot easily move when pressing the affected part (usually occurs in exudate including inflammation, allergic reaction and lymphatic obstruction)


1+
2+
3+
4+
2 mm
4 mm
6 mm
8 mm

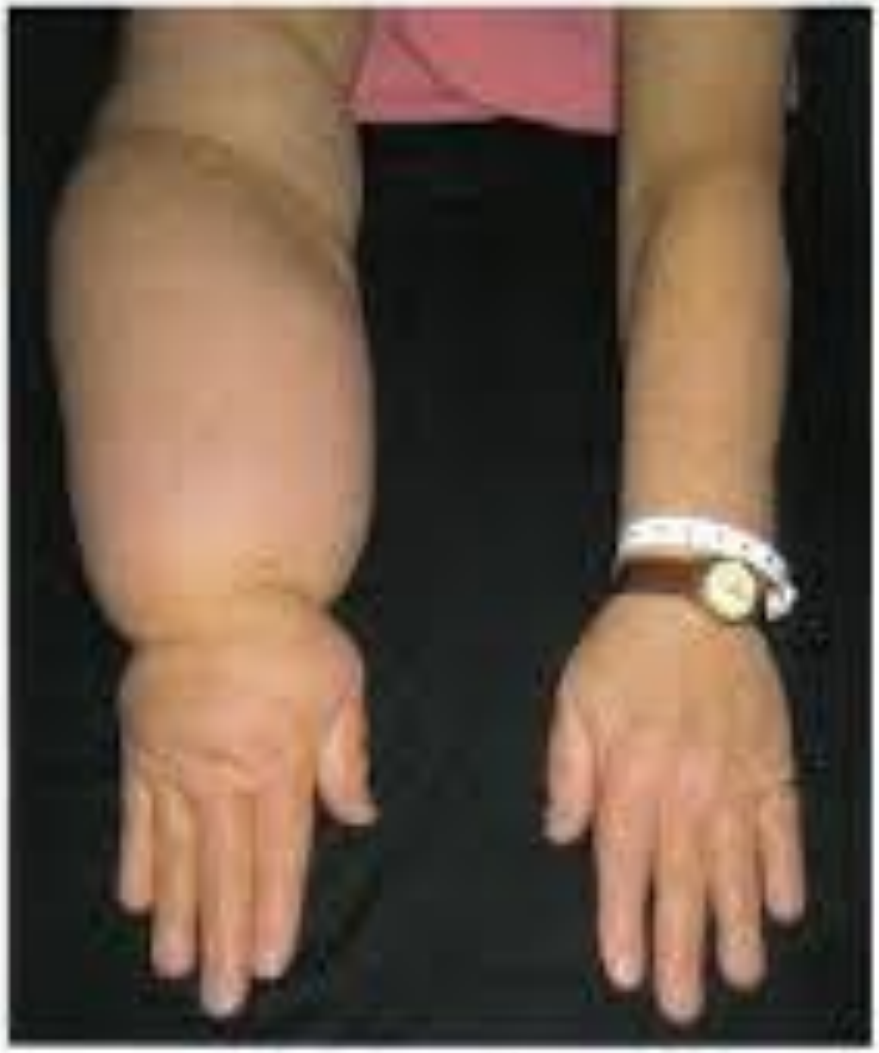

Normal
Edema
Pitting




Lymphedema



A

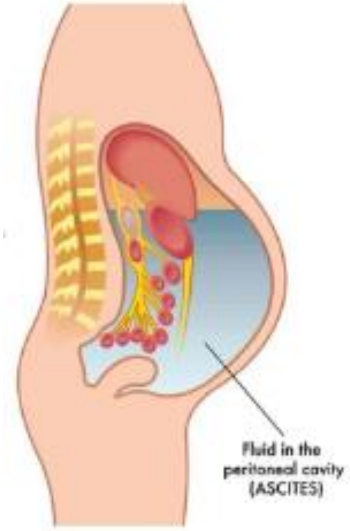


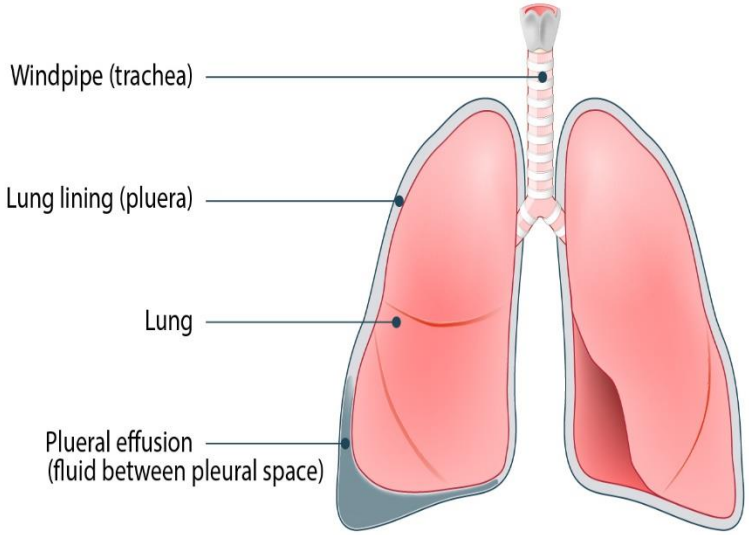
B

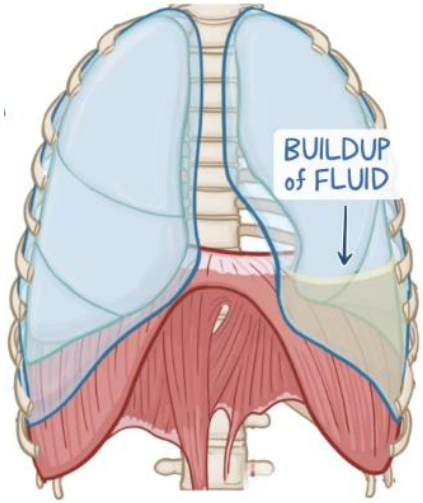
The most important clinical conditions of edema:

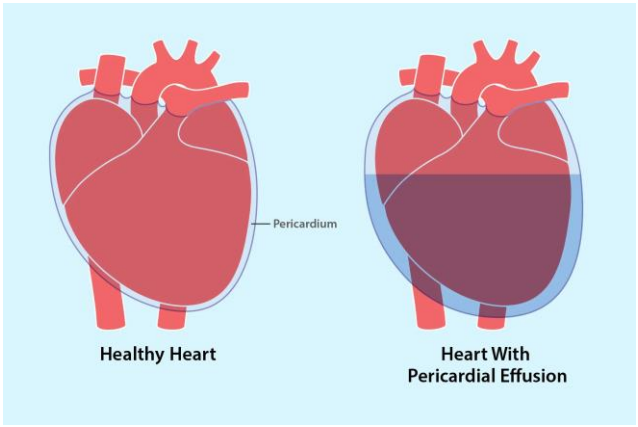
- **pulmonary edema**: due to left ventricular failure associated with sever dyspnea and anoxia it interfere with normal ventilation.
- **Brain edema**: it is serious and rapidly fatal. It leads to increase intracranial pressure . cerebral thromboembolism is one of the major causes.
- **Laryngeal edema**: serious may cause suffocation.

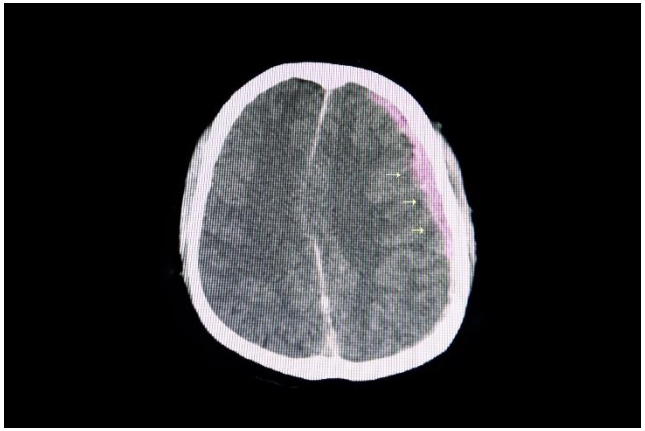
- **Anasarca**: is a severe and generalized edema with profound subcutaneous tissue swelling.
- **Hydrothorax** (pleural effusion): accumulation of fluid in the pleural cavity.
- **Hydropericardium (pericardial effusion):** accumulation of fluid in the pericardium.
- **Hydroperitoneum (ascites)**: accumulation of fluid in the peritoneal cavity.

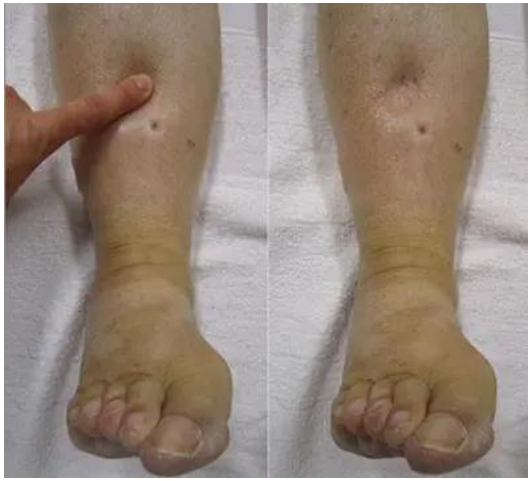

Fluid in the
peritoneal cavity
(ASCITES)


Windpipe (trachea)
Lung lining (pluera)
Lung
Plueral effusion
(fluid between pleural space)


BUILDUP
of FLUID


Pericardium
Healthy Heart
Heart With
Pericardial Effusion





Hyperemia

Hyperemia is an active increase blood volume in arterial and arteriolar blood vessels (arterial and arteriolar dilatation) which can occur in:

1. exercise and heat exposure
2. inflammation and release of vasoactive substances
3. sympathetic neurogenic mechanism

Affected tissue appears red (oxygenated blood) and warm

Congestion

Congestion: is a passive increase in blood volume in venous part of blood vessels (impaired venous drainage). Congestion could be localized venous congestion or generalized venous congestion.

Affected tissue appears blue-purple and swollen due to accumulation of deoxygenated blood

Haemorrhage

Hemorrhage: is an extravasation of blood outside the blood vessels.

Causes:

- Ruptured blood vessels wall: physical trauma – Stabbing - Stick injury - Gunshot - Motor vehicle accident

- Inadequacies in blood clotting which can be due to:

A. qualitative & quantitative defect of platelets

B. Missing or low amount of clotting factors e.g. Low levels of prothrombin, fibrinogen & other precursors.

C. Inadequate vitamin K leads to clotting factor deficiency because this vitamin is important in the synthesis of the clotting factors by the liver.

Terminology:

1) **Petechiae:** Minute 1-2 mm hemorrhages occurring in the skin, mucosal membrane, or serosal surface.

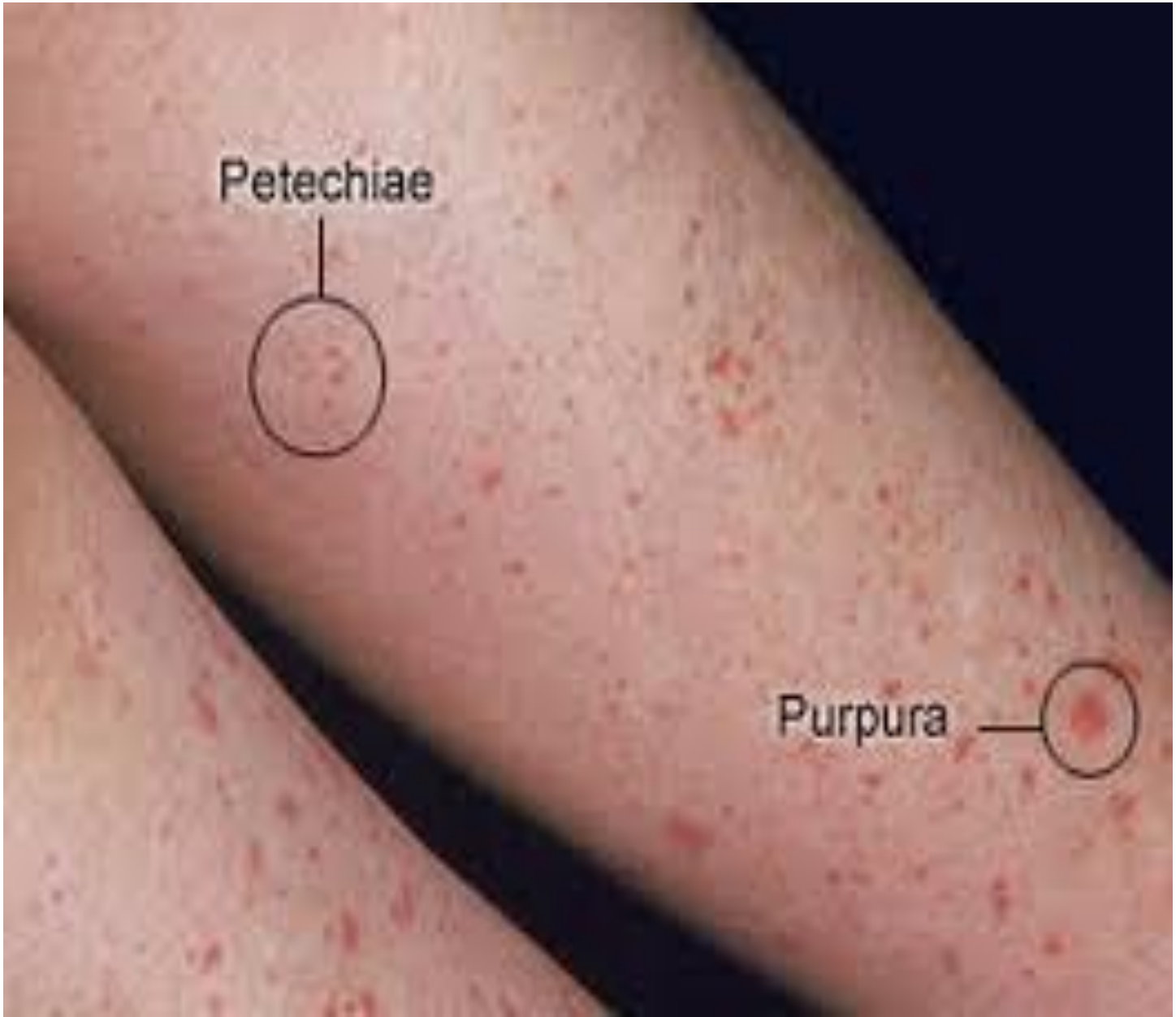
2) **Purpura:** Slightly > 3mm – 1 cm hemorrhage occurring in the skin.

3) **Ecchymosis (bruises):** Larger than 1-2cm subcutaneous hemorrhage. It is typical after trauma.

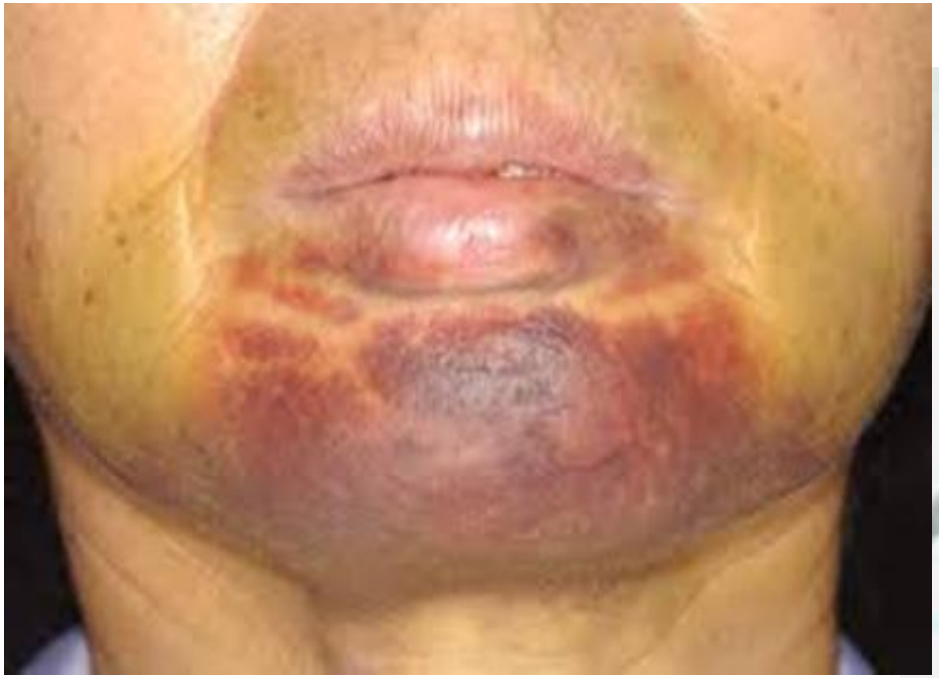
4) **Hematoma:** Haemorrhage enclosed within a tissue or a cavity.

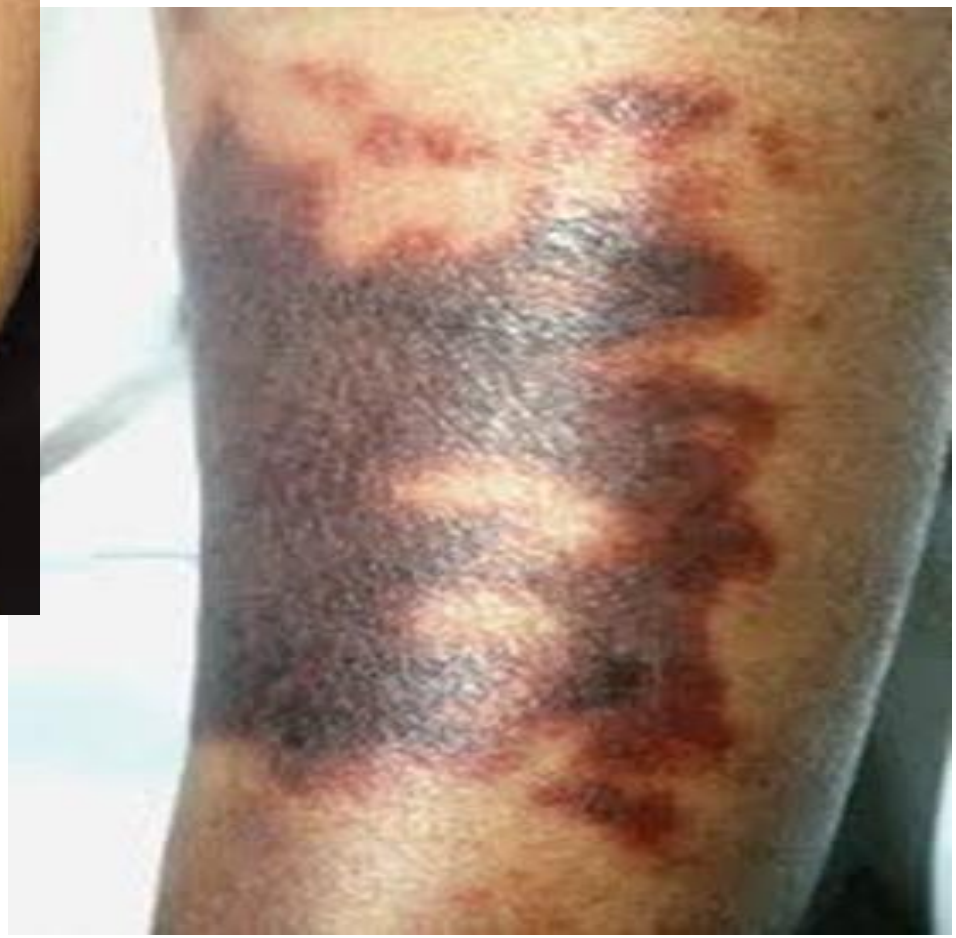
Effects of haemorrhage: depend on the rate and amount of blood loss:

- If > 20% the total blood volume is rapidly lost from the body, it may lead to hypovolumic shock & death.
- Chronic loss of blood leads to anaemia.


Petechiae
Purpura

ecchymosis (bruises)



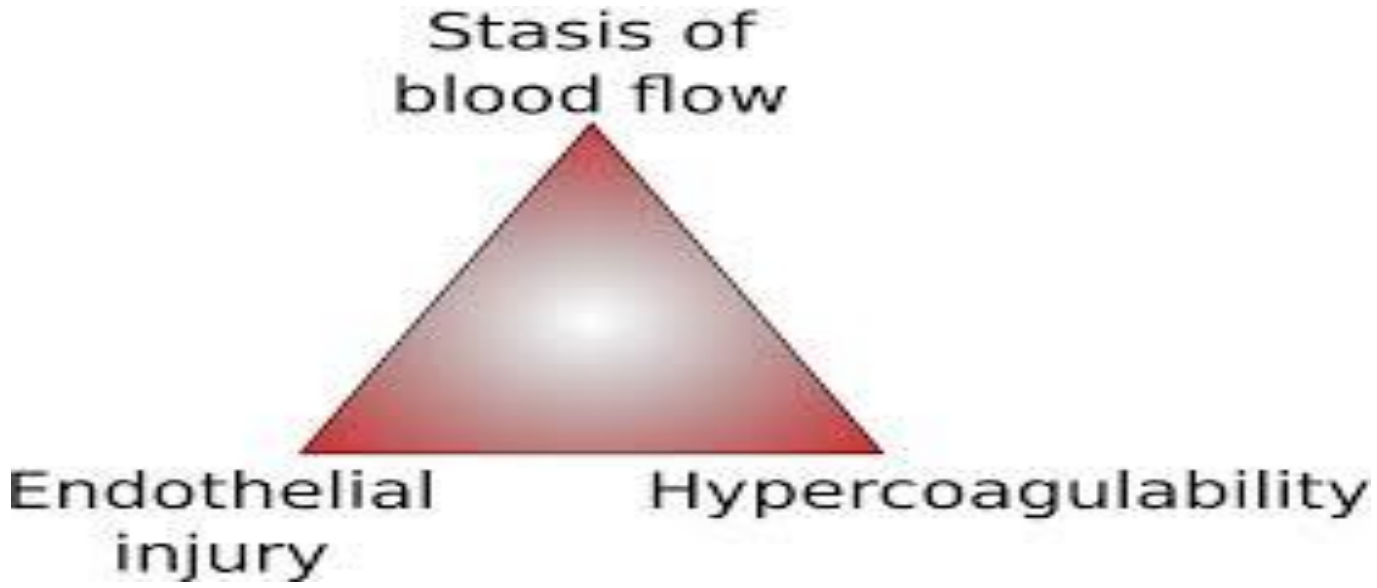


hematoma

Thrombosis

Thrombosis is defined as the formation of a solid or semisolid mass from the constituents of the blood within the vascular system during life.

Pathogenesis: thrombus formed due to the following three factors are called **Virchow's triad**:



A: Endothelial injury

1. Mechanical injury: e.g rupture, torsion
2. Degeneration of endothelial cell as in atherosclerosis and aneurysm
3. Inflammation: phlebitis and arteritis

B: Changes in blood flow: Stasis or turbulence of blood flow

1. Arrhythmia of heart
2. heart failure
3. Prosthetic heart valve
4. Incompetent vascular valves (varicose veins)

C: Changes in composition of blood: Hypercoagulability

1. Quantitative and qualitative changes in the function of platelets: e.g after surgery or radiation
2. Blood disease e.g Polycythemia
3. smoking

Types of thrombi:

i. Pale thrombus: composed mainly of platelets and fibrin strands. This type is seen in arteries

ii. Red thrombus: composed of platelets, fibrin strands and red blood cells. this type seen in venous thrombosis

iii. According to the presence or absence of pyogenic bacteria, thrombi can be classified to septic and aseptic respectively.

Fates (outcome) of a thrombus:

Thrombus can have one of the following fates:

A: Propagation:

The thrombus may accumulate more platelets and fibrin & propagate to cause vessel obstruction.

B: Embolization:

The thrombus may dislodge and travel to other sites in the vasculature. Such a traveling thrombus is called **thromboembolism**. An embolus may obstruct a vessel. The obstruction leads to the death of the tissue supplied by the blood vessel. Death of a tissue due to a decreased blood supply or drainage is called infarction.

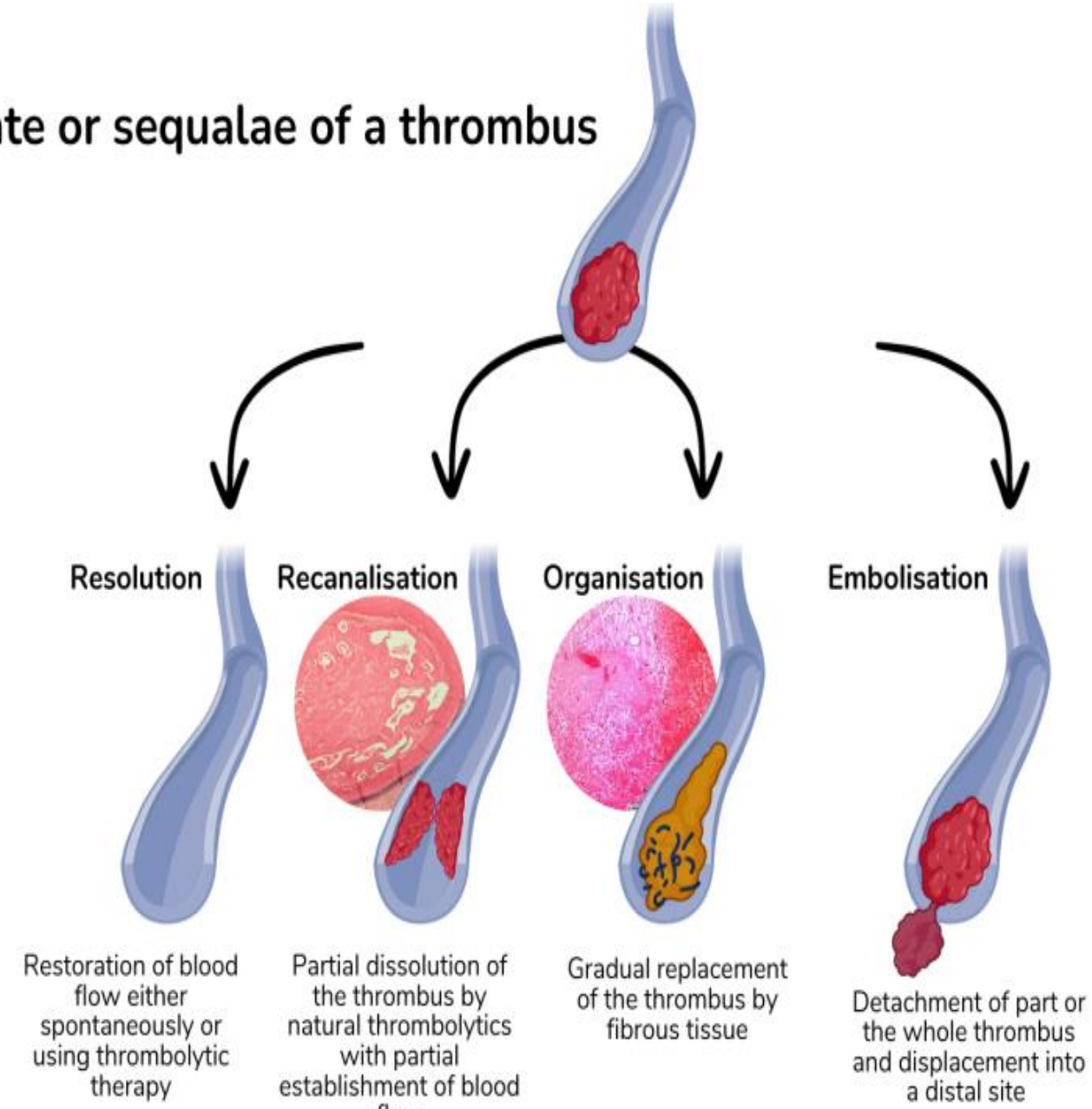
Therefore, an embolus can eventually lead to an infarction of an organ. E.g cerebral infarction can be caused by a thromboembolus from deep venous thrombosis.

C: Dissolution: The thrombus may be removed by fibrinolytic activity.

D: Organization and recanalization

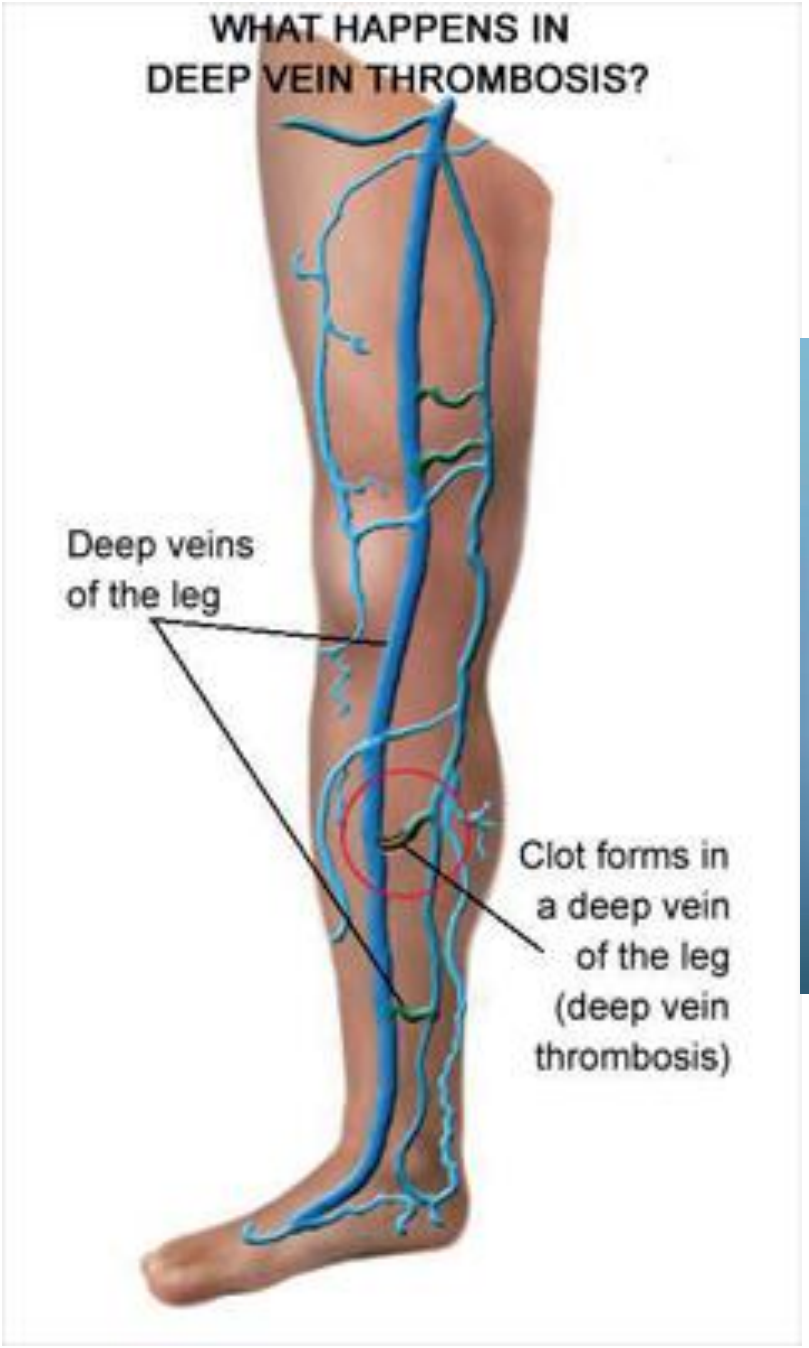
Organization refers to the ingrowth of endothelial cells, smooth muscle cells, and fibroblasts into the fibrin-rich thrombus. Organization is accompanied by the formation of capillary channels across the thrombus, re-establishing lumen continuity to some extent. This is known as recanalization. The recanalization eventually converts the thrombus into a vascularized mass of tissue which is later incorporated as a subendothelial swelling of the vessel wall.

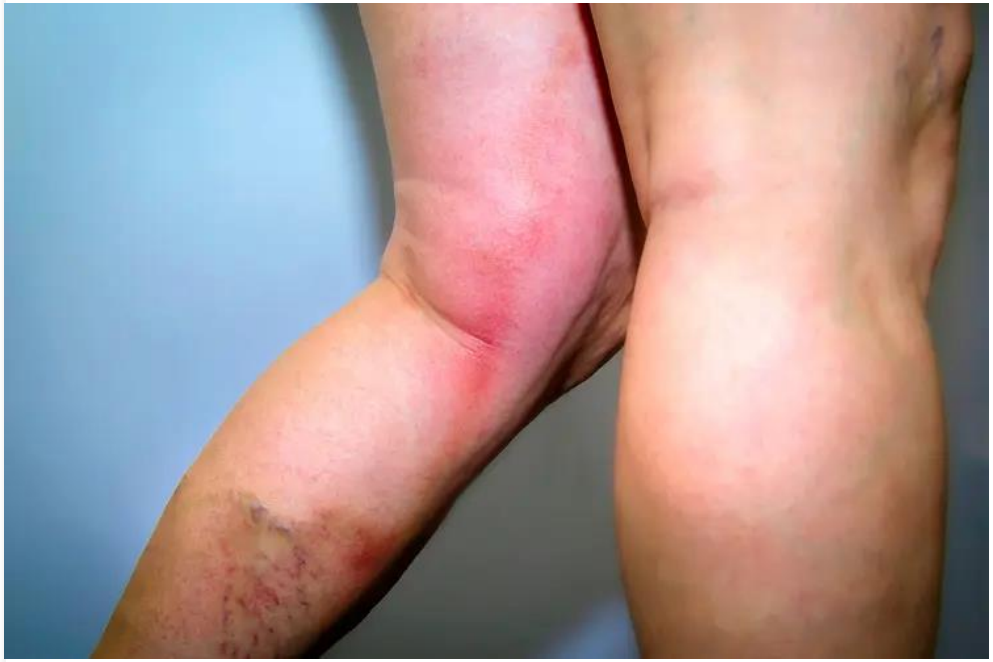
Fate or sequalae of a thrombus



Deep venous thrombosis (DVT):

- Usually starts in deep veins within the calf muscles.
- Patient present with local pain, heat & edema
- Has higher incidence in middle aged & elderly people, after surgery or any patient have predisposing factors for thrombus formation
- May dislodge and cause pulmonary embolism.


WHAT HAPPENS IN
DEEP VEIN THROMBOSIS?
Deep veins
of the leg
Clot forms in
a deep vein
of the leg
(deep vein
thrombosis)



EMBOLISM

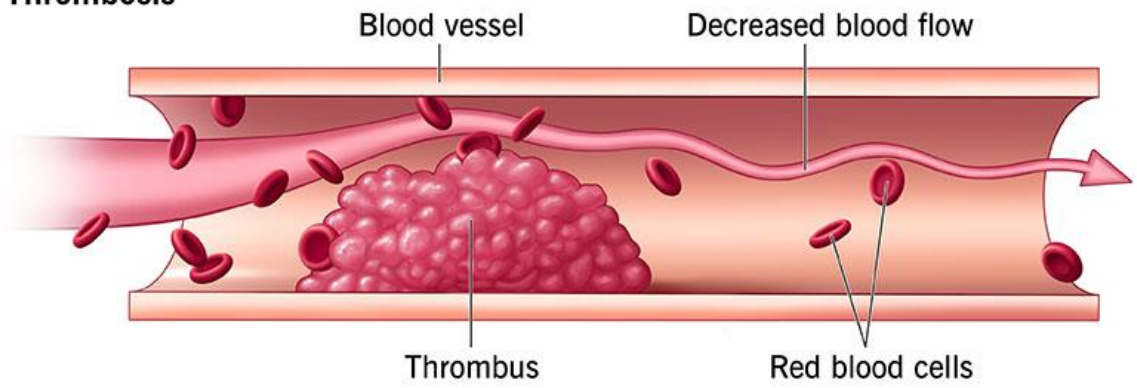
Definition: An embolus is a detached intravascular solid, liquid or gaseous mass that is carried by blood to sites distant from its point of origin. After traveling via the blood, the embolus can obstruct a vessel.

Causes of embolism:

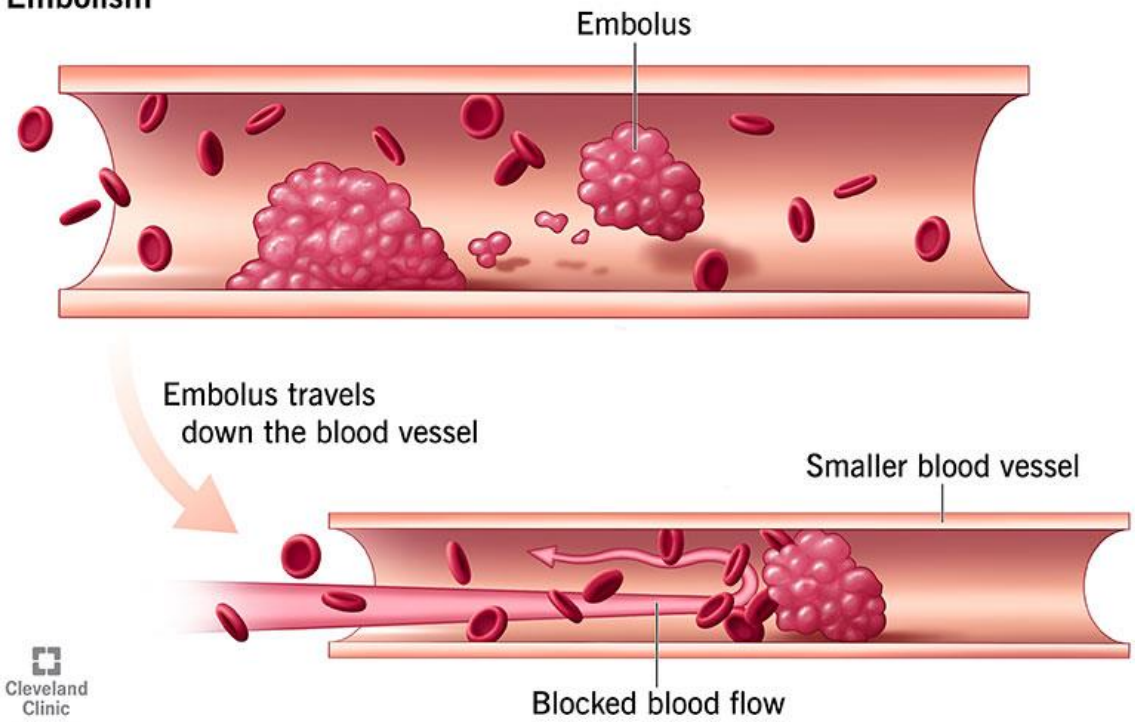
An embolus can arise from:

- o Thrombus → thromboembolism
- o Platelets aggregates
- o Fragment of material from ulcerating atheromatous plaque
- o Fragment of a tumor
- o Fat globules
- o air
- o Amniotic fluid
- o Infected foreign material

Thrombosis



Embolism



Thromboembolism

Based on its sites of origin & impaction, thromboembolism can be divided into:

a) Pulmonary thromboembolism (PTE) : PTE refers to the impaction of an embolus in the pulmonary arteries & their branches. Such an embolus is derived from a thrombus in the systemic veins or the right side of the heart. 95% of PTE arise from thromi in the deep leg veins.

1. If the thrombus is large, it may block the outflow tract of the right ventricle or the bifurcation of the main pulumonary trunk (saddle embolus) or both of its branches, causing sudden death or right side heart failure (cor pulmonale).

2. If the embolus is very small (as in 60-80% of the cases), the pulmonary emboli will be clinically silent.

3. Embolic obstruction of medium sized arteries manifests as pulmonary haemorrhage but usually does not cause infarction because of dual blood inflow to the area from the bronchial circulation.

4. If the cardiorespiratory condition of the patient is poor (i.e., if the patient previously had cardiac or pulmonary disease), then obstruction of a medium sized pulmonary artery by a medium-sized embolus can lead to pulmonary infarction.

5. Recurrent thromboembolism can lead to pulmonary hypertension in the long run.

b) Systemic thromboembolism: Systemic emboli arise from the left side of the heart due to prosthetic heart valve, rheumatic heart valve, arrhythmia. It refers to emboli travelling within arterial circulation & impacting in the systemic arteries.

Systemic thrombi may impact in:

- Lower extremities (75%), brain (10%), with the rest lodging in the intestines, kidney, & spleen.

c) Crossed embolism (Paradoxical embolism):

- This occurs when an embolus is transferred

from the right to the left side of the heart, then into the systemic circulation.