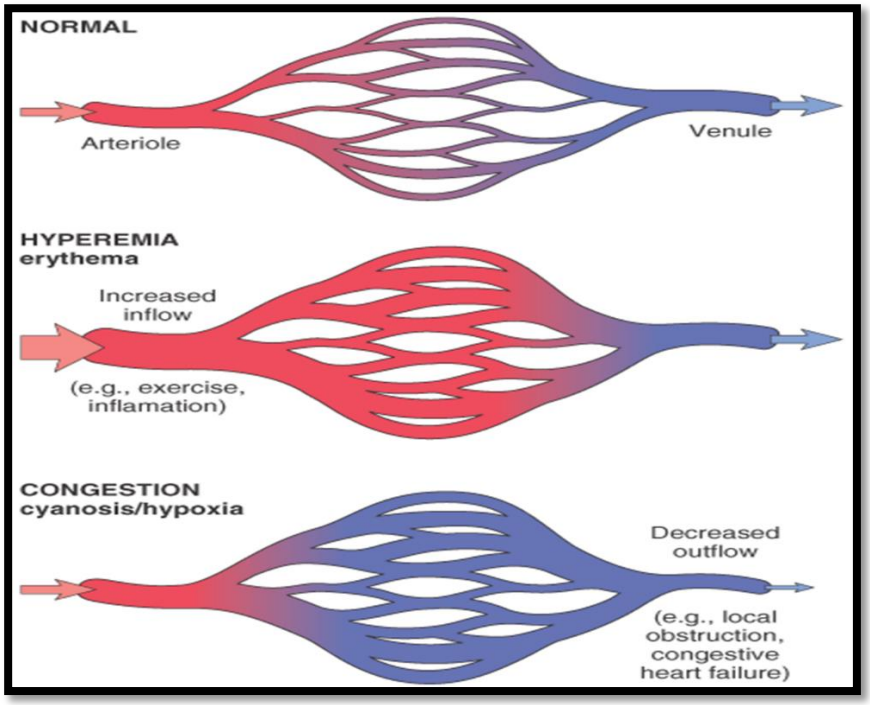
Hemodynamic Disorders

Hyperemia & Congestion both indicate a locally increased blood volumes in particular tissue.

Hyperemia: Is an active process resulting from arteriolar dilation & increased blood flow (e.g at sites of inflammation or in skeletal muscle during exercise).

The hyperemic tissues are redder than normal because of engorgement with oxygenated blood.



Congestion: is a passive process resulting from reduced outflow of venous blood from a tissue. It can be systemic (cardiac failure)or local (venous obstruction).The congested tissues have abnormal blue- red color (cyanosis) due to accumulation of deoxygenated hemoglobin.

Morphology: **Ex**: **Liver** in **acute hepatic congestion**→ the central vein and sinusoids are distended; centrilobular hepatocytes can be frankly ischemic while the periportal hepatocytes better oxygenated may only develop fatty change while In **chronic hepatic congestion** the centrilobular regions are grossly red-brown and slightly depressed (because of cell death) and are surrounding zones of uncongested tan liver (**nutmeg appearance**)

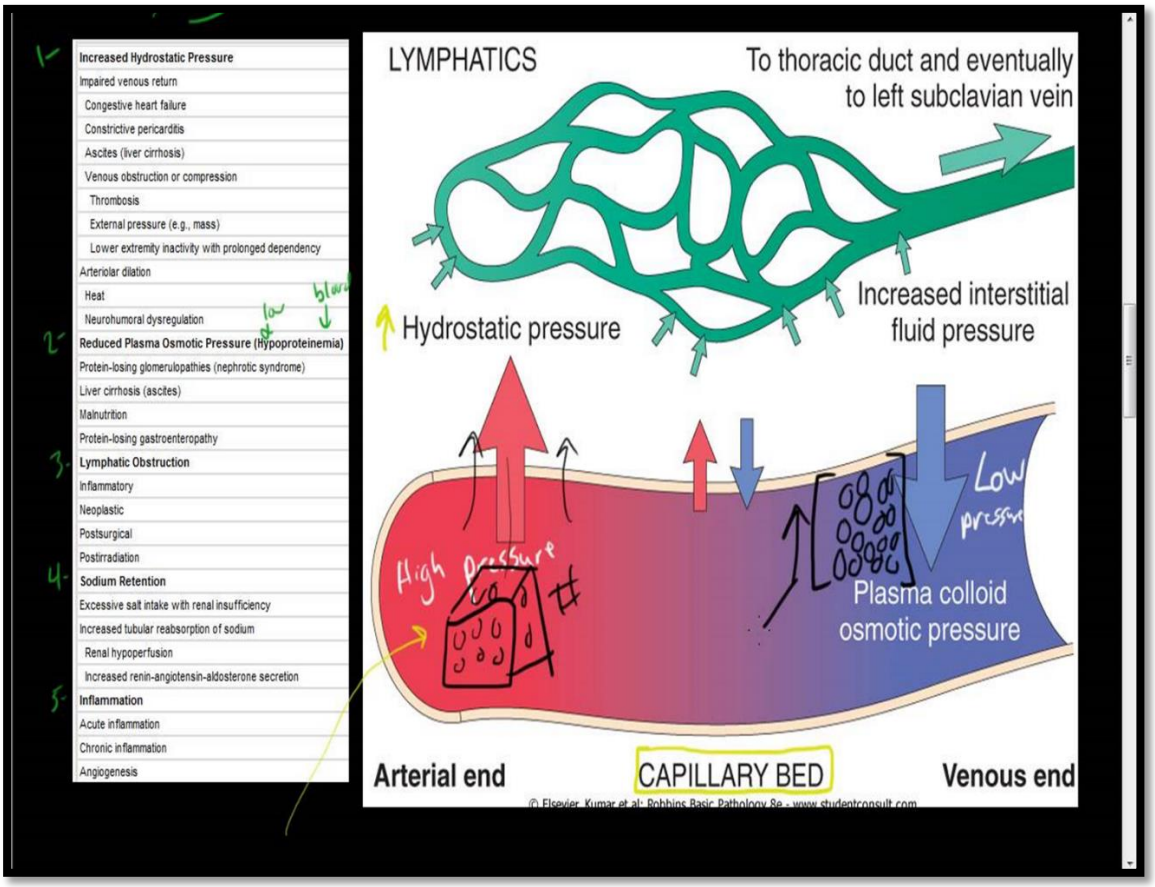
Microscopically there is centrilobular necrosis, with hemorrhage and in long standing severe hepatic congestion (in heart failure) lead to hepatic fibrosis (cirrhosis) can develop

Edema

An abnormal increase in interstitial fluid within tissues is called **edema**

while fluid collections in the different body cavities are variously designated hydrothorax (pleural cavity), hydropericardium (pericardial cavity), and hydroperitoneum or ascites (peritoneal cavity)

Anasarca is a severe and generalized edema



Causes of edema

1. Increased Hydrostatic Pressure:

Regional increases in hydrostatic pressure can result from a focal impairment in Venous return (deep venous thrombosis in a lower extremity), or generalized increases in venous pressure (in congestive heart failure).

2. Reduced Plasma Osmotic Pressure:

Occurs when albumin, is not synthesized in adequate amounts (ex. In chronic liver diseases or in protein malnutrition) **OR** is lost from the circulation (ex: nephrotic syndrome)

3. Sodium and Water Retention:

Increased salt retention–with water–causes both increased hydrostatic pressure (due to intravascular fluid volume expansion) and diminished vascular colloid osmotic pressure (due to dilution) Ex: Renal failure.

4. Lymphatic Obstruction.

Impaired lymphatic drainage results in lymphedema (ex: chronic inflammation & invasive malignant tumors).

Hemorrhage

Is defined as the extravasation of blood into the extravascular space.

Tissue hemorrhage can be manifested by different appearances:

1- **Hematoma**: accumulated hemorrhage that can be confined within a tissue

2–Petechiae:tiny l– to 2–mm hemorrhages in to skin & mucous membranes (mainly due to platelets defect).

3–Purpura: slightly larger (3–5 mm) hemorrhages can be secondary to trauma, vascular inflammation (vasculitis)

4–Ecchymoses larger (1 to 2 cm) subcutaneous hematomas (bruises) .

5–Depending on the location a large accumulation of blood in a body cavity is denoted as a hemothorax, hemopericardium,hemoperitoneum or hemarthrosis (in joints).

Hemostaisis

Normal hemostasis is a consequence of tightly regulated processes that maintain blood in a fluid state in normal vessels & also permit the rapid formation of a hemostatic clot at the site of a vascular injury. **The pathologic counterpart of hemostasis is thrombosis**

The general sequence of events in hemostasis at a site of vascular injury:

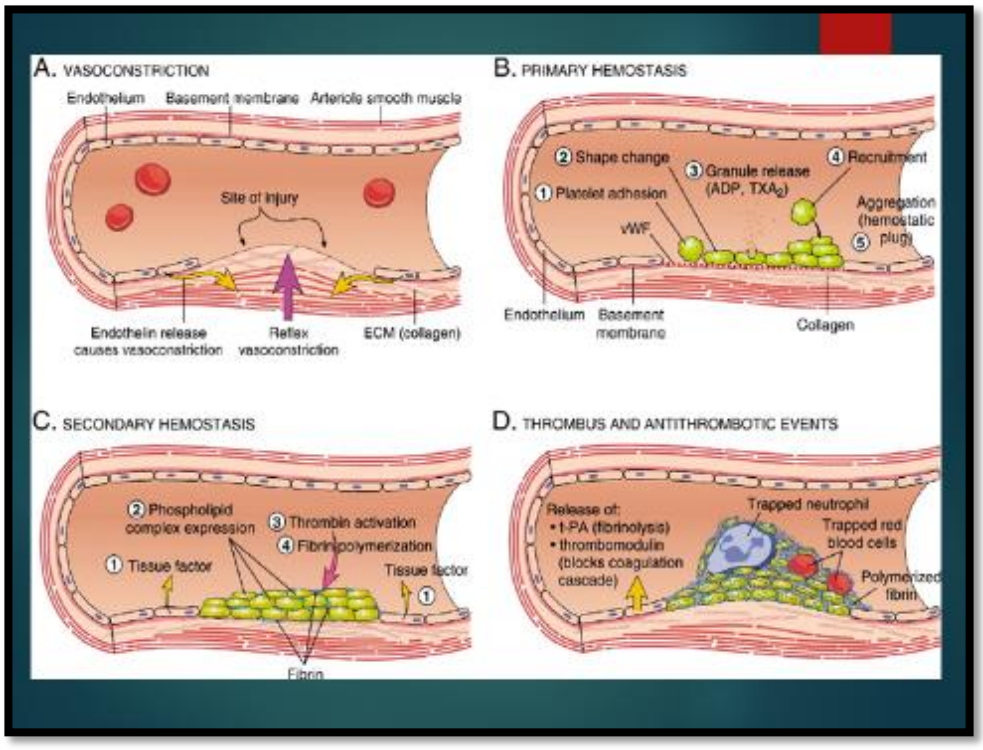
1. After initial injury there is a brief period of arteriolar vasoconstriction. The effect is transient, however, and bleeding would resume if the platelet and coagulation systems not activated.

2. Endothelial injury exposes highly thrombogenic subendothelial extracellular matrix (ECM), facilitating platelet adherence , activation and within minutes the additional platelets aggregate (aggregation) to form a hemostatic plug, this process is referred to as **primary hemostasis**.

3. Tissue factor is also exposed at the site of injury synthesized by endothelial cells.It acts with factor VII, is the major in vivo initiator of the coagulation cascades of reaction that culiminates in **thrombin** generation.Thrombin cleaves circulating fibrinogen into insoluble fibrin, creating a fibrin mesh work, and also is a potent activator of platelets, leading to additional platelet aggregation at the site of injury. This sequence, referred to as **secondary hemostasis**, consolidates

the initial platelet plug

4-Clot stabilization and resorption. Polymerized fibrin and platelet aggregates undergo contraction to form a solid,permanent plug that prevents further hemorrhage. At this stage, counter regulatory mechanisms are set into motion that limit clotting to the site of injury.



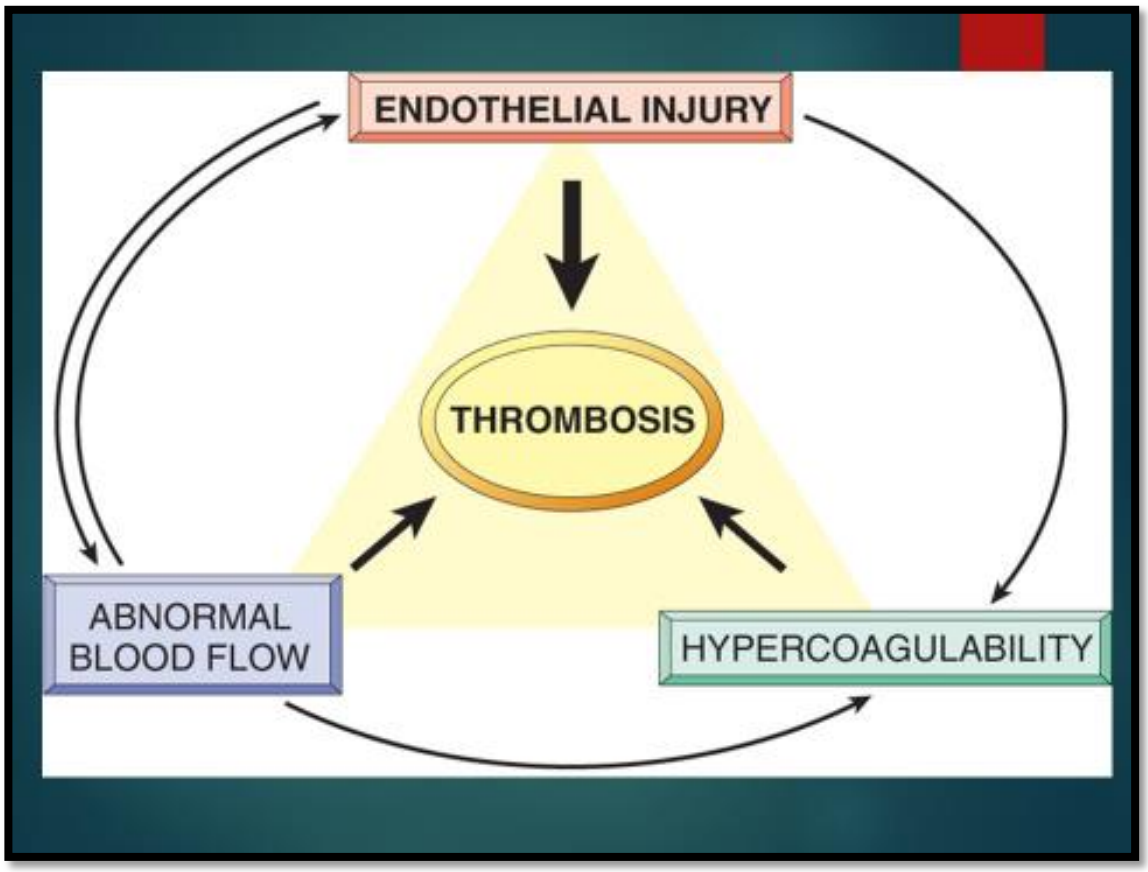
THROMBOSIS

Mean formation of blood clots within intact vessels.there are three primary abnormalities that lead to thrombus formation (called Virchow's triad)

1– endothelial injury.

2– stasis or turbulent blood flow .

3– blood hypercoagulability



Morphological types

1. **mural thrombi** :Thrombi occurring in heart chambers or in the aortic lumen. ex (dilated cardiomyopathy, or myocardial infarction in the heart) while (ulcerated atherosclerotic plaque and aneurysmal dilation) in aorta.

2. **Arterial thrombi**: are the coronary, cerebral, and femoral arteries. these are usually superimposed on a ruptured atherosclerotic plaque, vasculitis & trauma.

3. **Venous thrombosis** (phlebothrombosis) : The veins of the lower extremities are most commonly involved (90% of cases).

4. Vegetations : Thrombi on heart valves .blood-borne bacteria or fungi can adhere to damaged valves & can induce the formation of large thrombotic masses (infective endocarditis) **sterile vegetation** an also develop on non-infected valves in persons with hypercoagulable states (non- bacterial thrombotic endocarditis)

Fate of the Thrombus:

1. **Propagation**. Thrombi accumulate additional platelets and fibrin.

2. **Embolization**. Thrombi dislodge and travel to other sites in the vasculature.

3. **Dissolution** is the result of fibrinolysis, which can lead to the rapid shrinkage and total disappearance of recent thrombi.

4.**Organization and recanalization** older thrombi become organized by the ingrowth of endothelial cells, smooth muscle cells, and fibroblasts

Embolism

Embolus is an intravascular solid, Liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin , emboli can lodge anywhere in the vascular tree,99% of all emboli represent a dislodged thrombus so called thromboembolism.

Morphological types:

1. **Pulmonary embolism** (**PE**): In more than 95%of cases originate from leg deep vein thromboses (DVTs), depending on the size can occluded the main pulmonary artery, in bifurcation or the pass to the smaller arteries and branches.

2. **Systemic thromboembolism**: Most (80%) arise from intracardiac mural thrombi, two thirds of which are associated with left ventricular wall infarcts & left atrial fibrillation.The remainder originate from aortic aneurysms, or fragmentation of a valvular vegetation.

3.**Fat embolism**: fat globules-with or without associated hematopoietic marrow elements-can be found in the circulation and impacted in the pulmonary vasculature after fractures of long bone or, rarely, in the setting of soft tissue trauma and burns.

4. **Air embolism**: Gas bubbles within the circulation can coalesce form frothy masses that obstruct vascular flow (and cause distal ischemic injury). Ex; air trapped in a coronary

artery during bypass surgery, or in the cerebral circulation by neurosurgery in the "sitting position"

5. Amniotic fluid embolism: is an ominous complication of labor,the underlying cause is the infusion of amniotic fluid or fetal tissue into the maternal circulation via a tear in the placental membranes or rupture of uterine veins.

Infarction

An infarct is an area of ischemic necrosis caused by occlusion of the vascular supply of the affected tissue, nearly all infarcts result from thrombotic or embolic artery.

Morphology. Infarcts are classified a cording to color and the presence or absence of infection: they are either red (hemorrhagic) or white (anemic) and may be septic or non septic (bland)

a-Red Infarction occur:

1-With venous occlusions (ex. ovarian torsion),

2-In loose tissues (e.g., lung)

3-in tissues with dual circulations (e.g., lung and Small intestine).

4-In tissues previously congested by sluggish venous outflow.

b-White infarcts: occur with arterial occlusions in solid organs with end-arterial circulation (ex. heart, spleen, and kidney).

-Infarcts tend to be wedge-shaped with the occluded vessel at the apex and the periphery of the organ forming the base. The dominant histologic characteristic of infarction is ischemic coagulative necrosis.Acute inflammation is present along the margins of infarcts within a few hours.The brain is an exception to these generalizations, as central nervous system infarction results in liquefactive necrosis.

c-Septic infarctions: occur when infected cardiac valve vegetation embolized or when microbes seed necrotic tissue.In these cases the infarct is converted into an abscess, with a correspondingly greater inflammatory response.

Shock

Is the final common pathway for several potentially lethal clinical events, including severe hemorrhage, extensive trauma or burns, large myocardial infarction, massive pulmonary embolism & microbial sepsis.

The causes of shock fall into three

1. **Cardiogenic shock** results from low cardiac output due to myocardial pump failure. This can be due to intrinsic myocardial damage (infarction), ventricular arrhythmias, or outflow obstruction (e.g., pulmonary embolism).

2. **Hypovolemic shock**: results from low cardiac output due to the loss of blood or plasma volume, such as can occur with massive hemorrhage or fluid loss from severe burns.

3. **Septic shock**: results from vasodilation and peripheral pooling of blood as Part of systemic immune reaction to bacterial or fungal infection.