Circulatory Disturbances

Reset Revision

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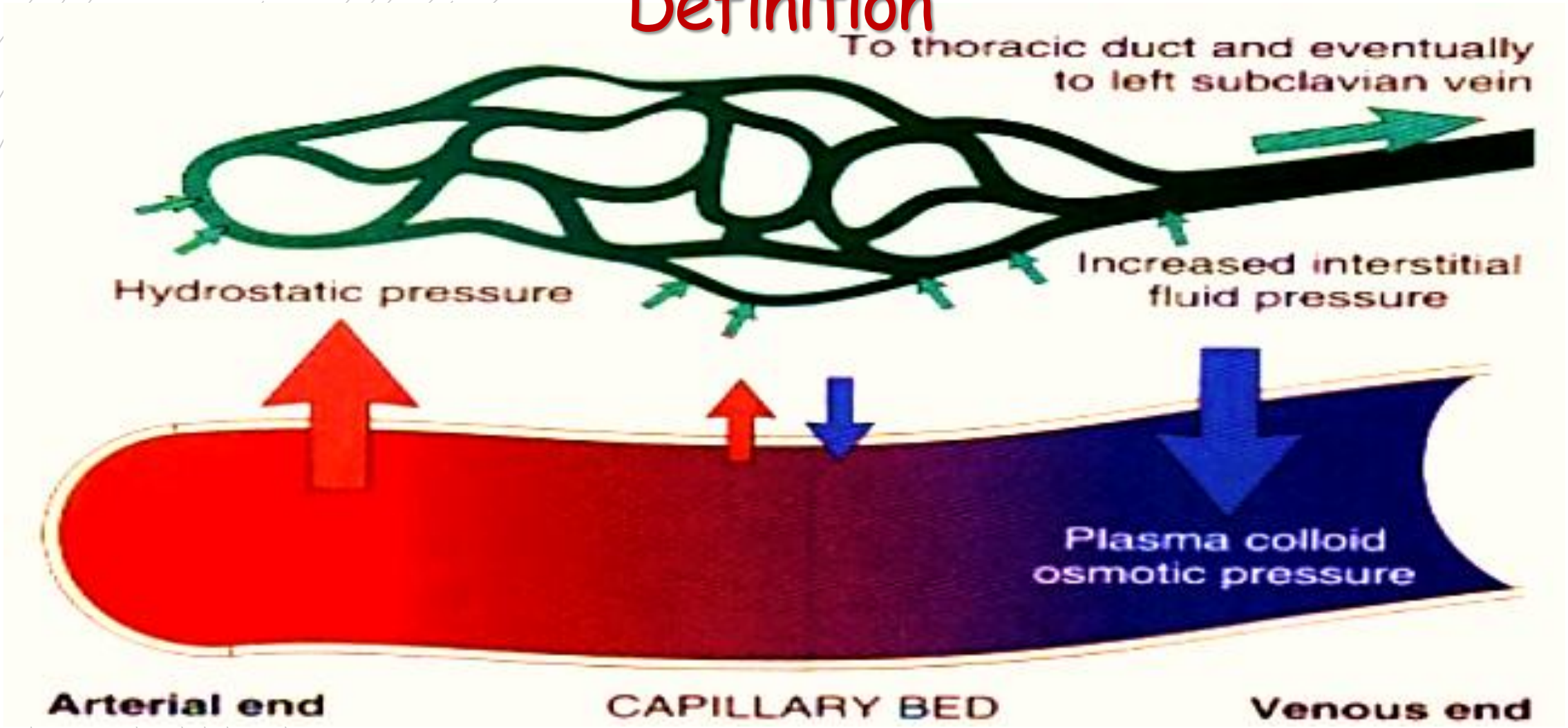
Edema
Definition



- Edema is abnormal accumulation of fluid in the interstitial tissue or body cavities.

Edema
Pathogenesis

- 1- Increased vascular hydrostatic pressure in:
  - Heart failure… cardiac edema, or impaired venous return (local or generalized)
- 2- Decreased blood osmotic pressure due to decreased albumin in:
  - Nutritional edema
  - Decreased protein synthesis as in liver disease
  - Increased Protein loss as in renal disease
- 3- Sodium retention as in impaired renal function

In 1, 2, 3 lead to Pitting, mostly Generalized edema

Edema
Pathogenesis

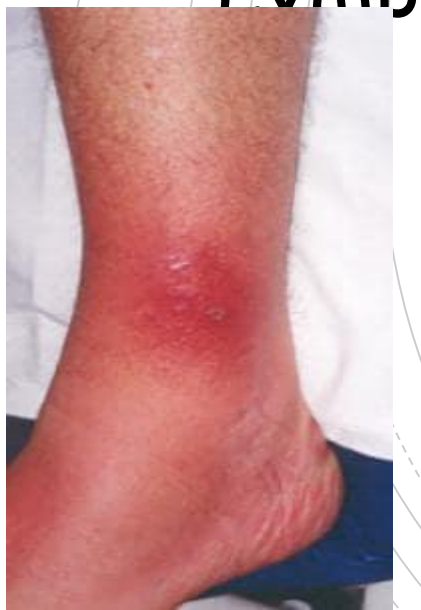
- 4. Increased vascular permeability as in acute inflammation
- 5. Lymphatic obstruction as
  - Surgical removal of LNs.
  - Inflammation (lymphangitis)
  - Infection: Filariasis.
  - Irradiation.
  - Tumor infiltration.

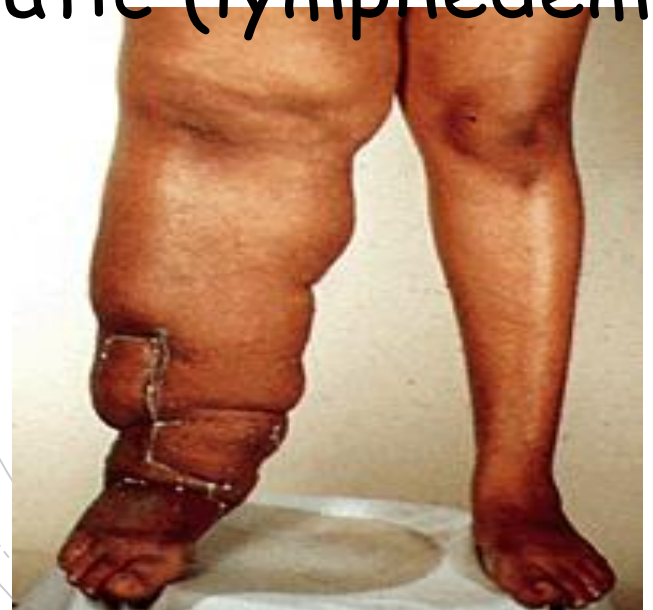
In 4 & 5 lead to Localized, non pitting edema

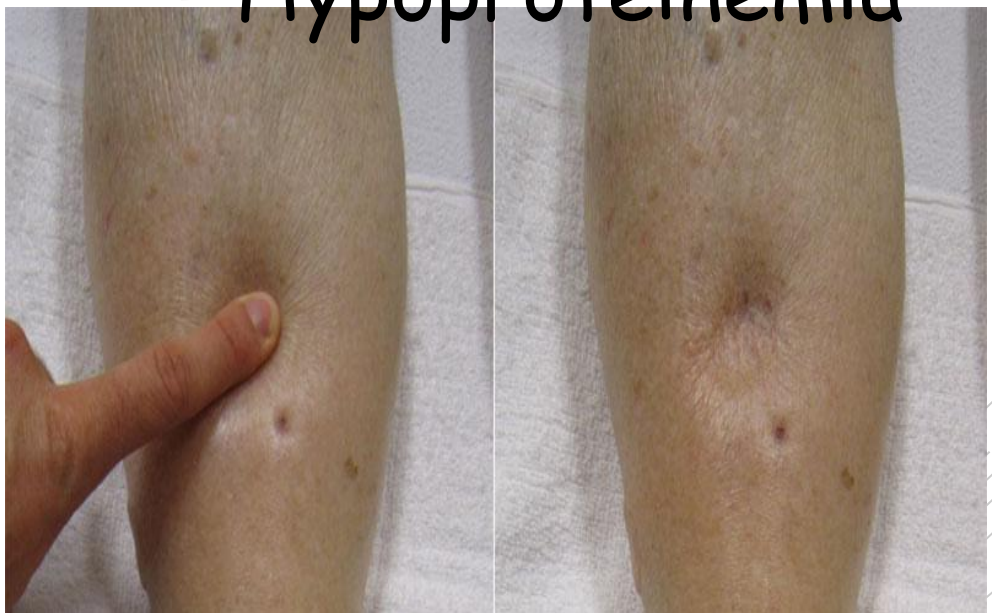
Edema
Classification according to distribution

Localized
- Inflammatory (exudate)
- Localized venous obstruction
- Lymphatic (lymphedema)

Generalized
- Cardiac
- Renal
- Hypoproteinemia

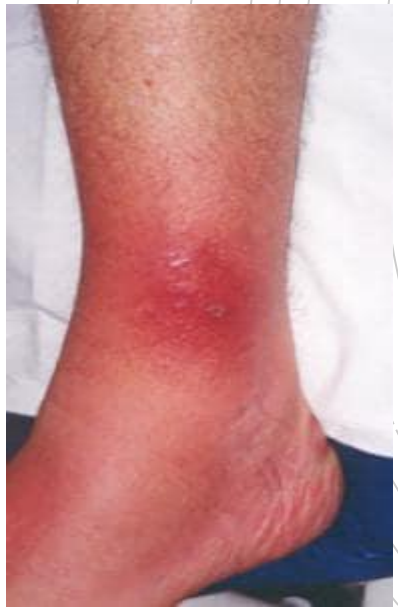


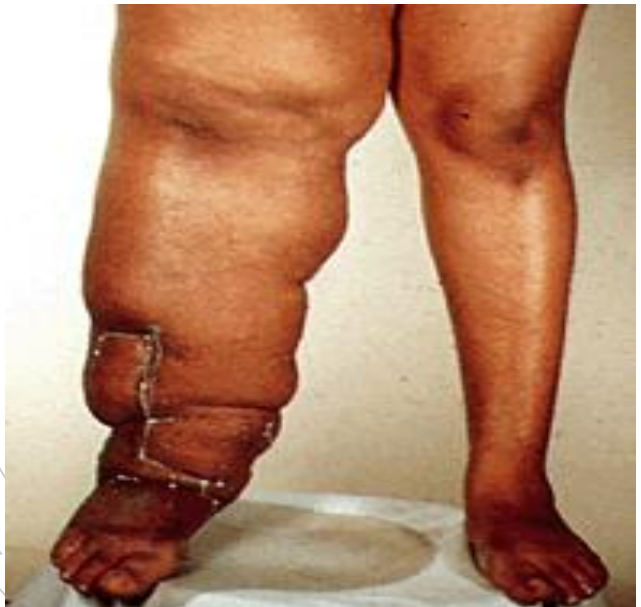




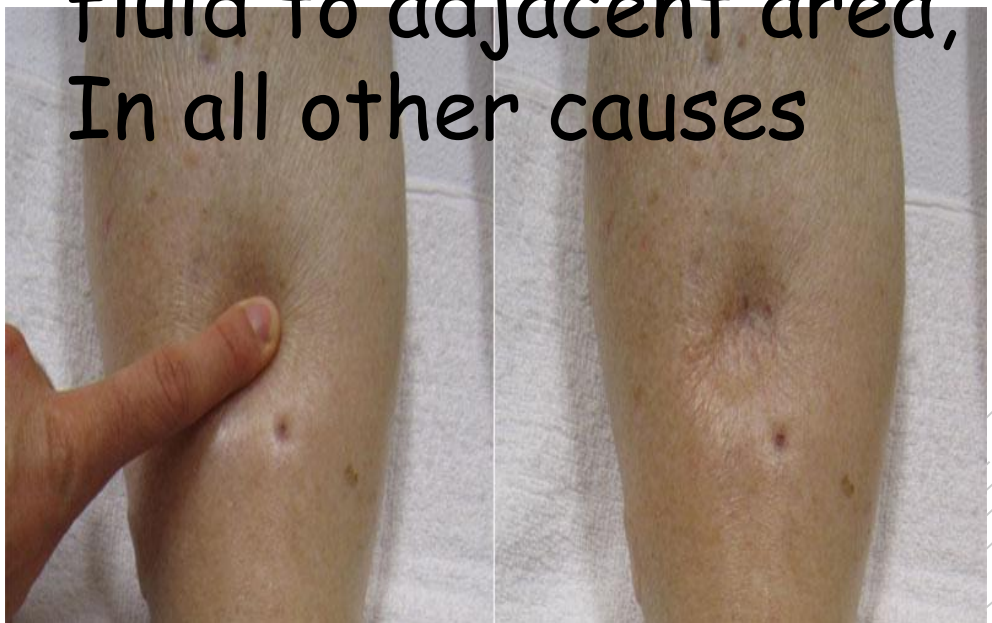
Edema
Classification according to distribution

Non-Pitting
- Inflammatory (exudate)
- Lymphatic (lymphedema)





Pitting
On pressure, a pit is formed due to forcing fluid to adjacent area, In all other causes

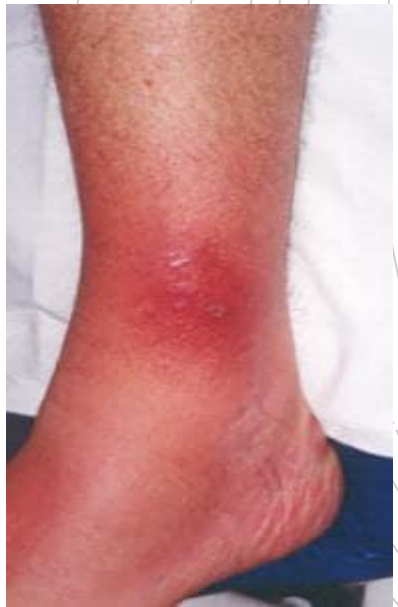


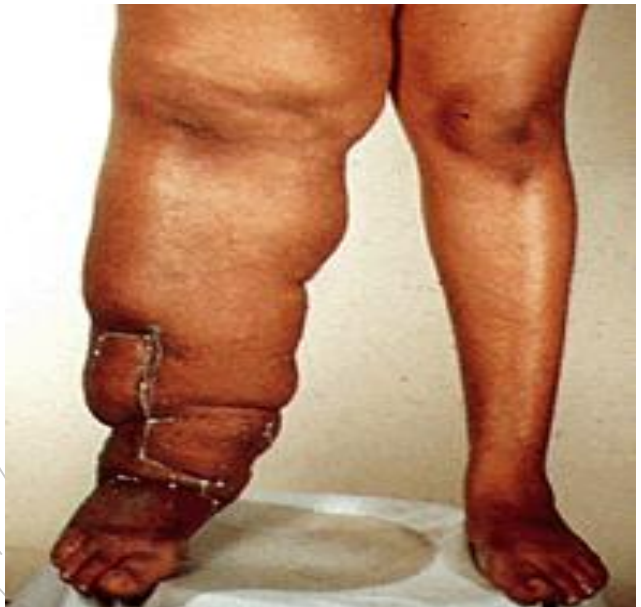
Edema
Classification according to fluid nature

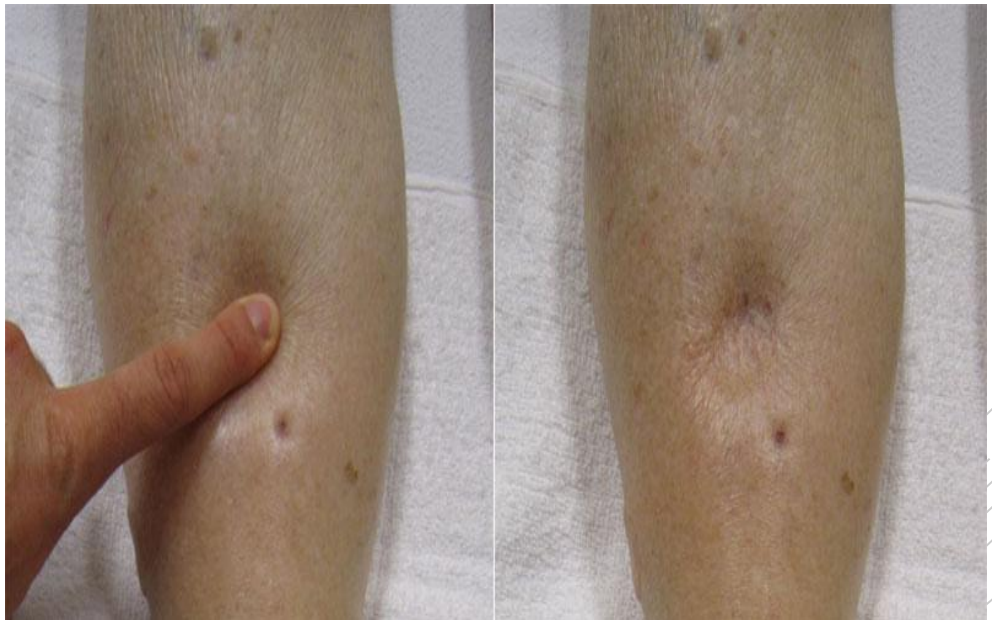
Exudate
-Inflammatory

Lymph
-Lymphedema

Transudate
-Generalized edema
-Localized venous edema



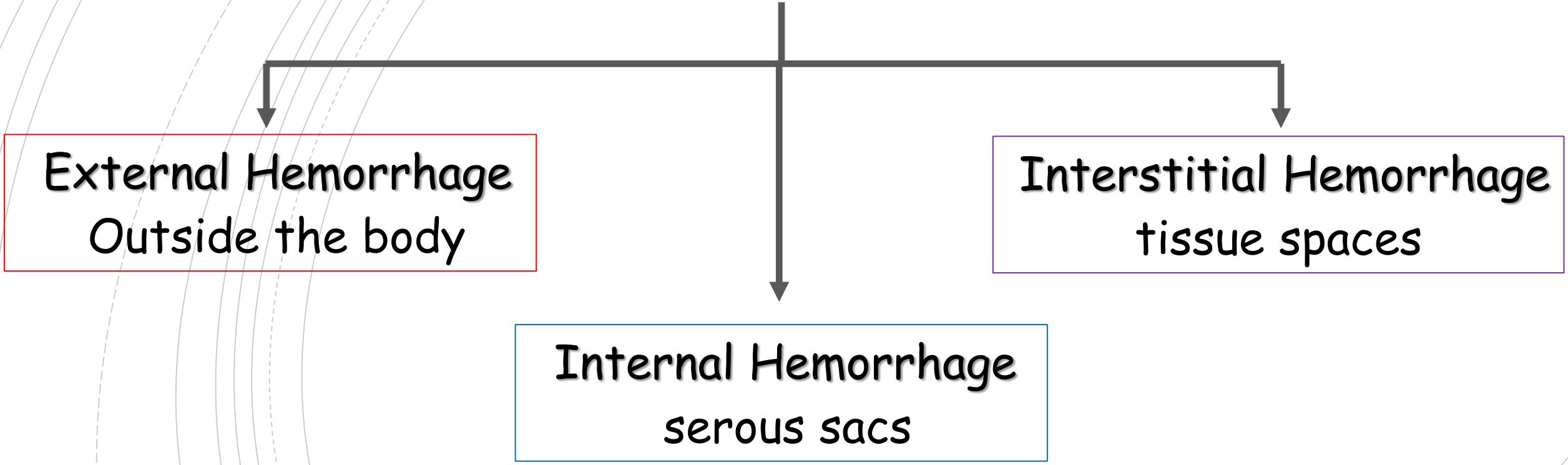




Edema Terminology

- Hydrothorax (pleural effusion) is….
- Hydro pericardium (pericardial effusion) is….
- Hydroperitonium (ascitis) is….
- Hydroarthrosis is….
- Hydrocele is….
- Anasarca is….
- Edema due to lymphatic obstruction is called….

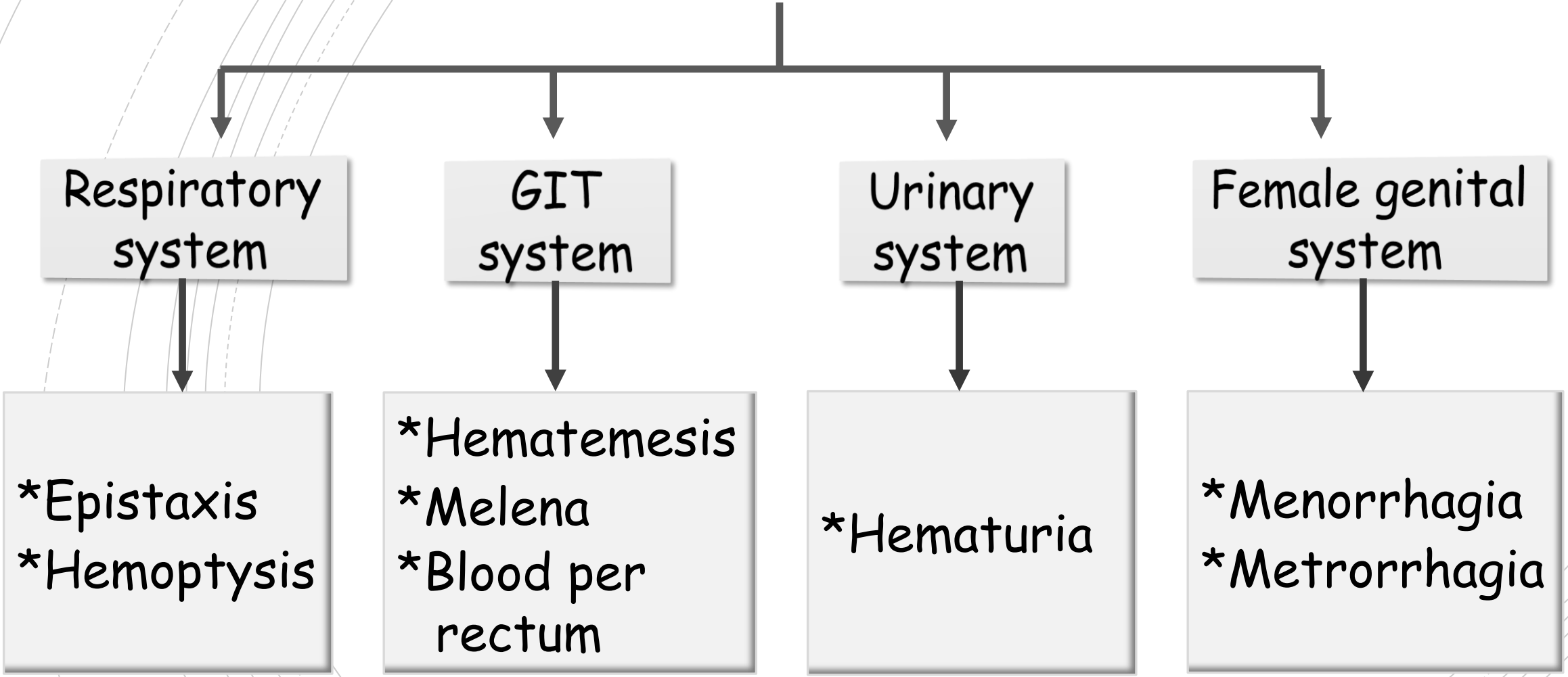
Hemorrhage Classification



Hemorrhage

External

It is hemorrhage through body orifices.

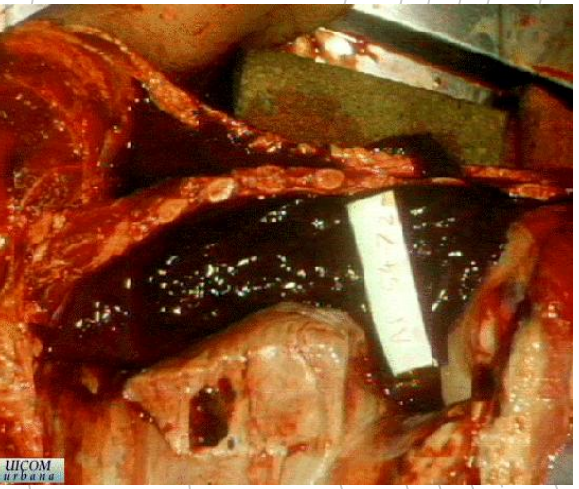


Hemorrhage

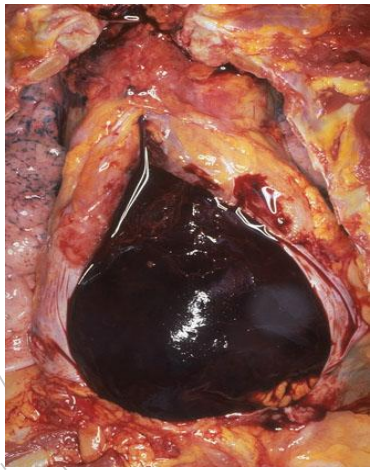
Internal

It is hemorrhage inside body cavities.

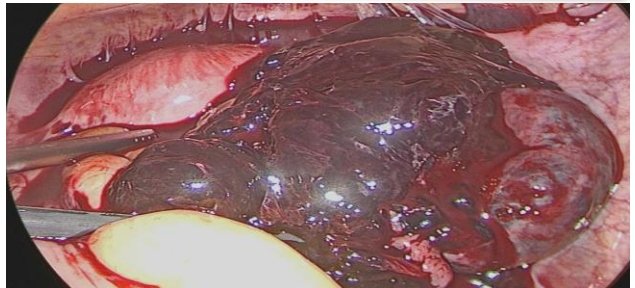
- Hemothorax



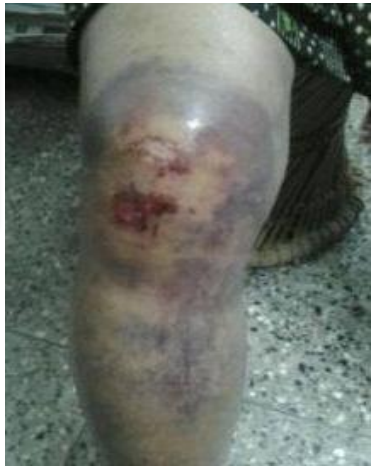
- Hemopericardium



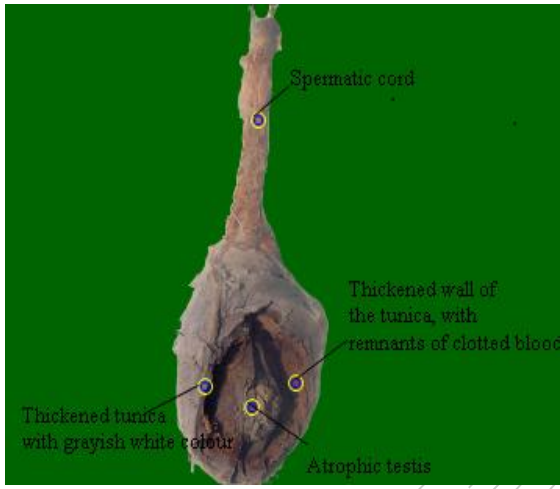
- Hemoperitonium



- Hemarthrosis



- Hematocele

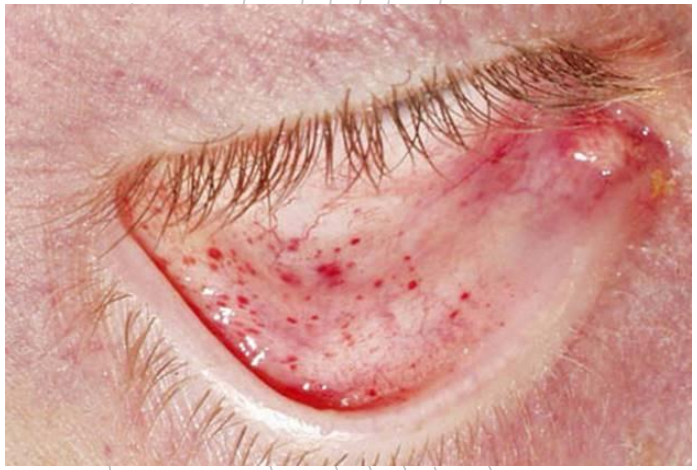


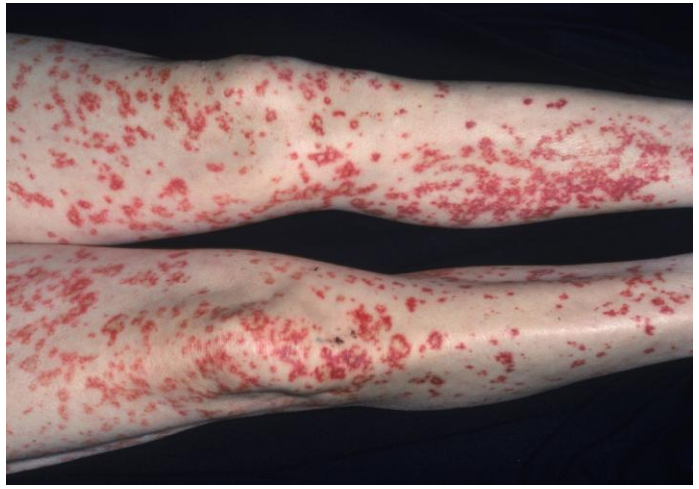
Hemorrhage

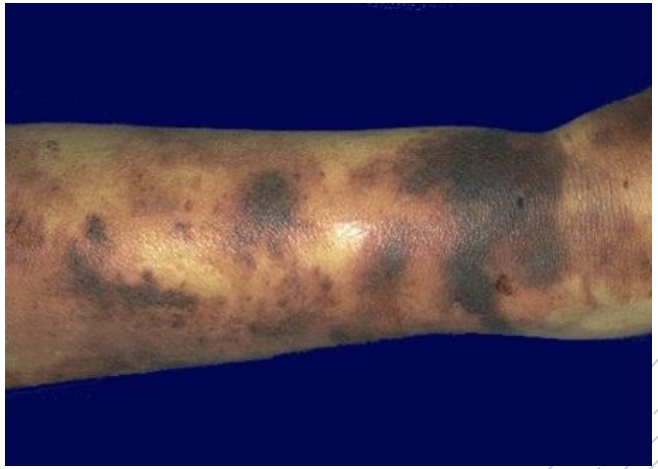
Interstitial

Accumulation of blood within tissues (cutaneous, mucus membranes, under serosal surfaces or organs).

Petechiae	Purpura	Ecchymosis
1-2 mm	>3 mm	1-2 cm







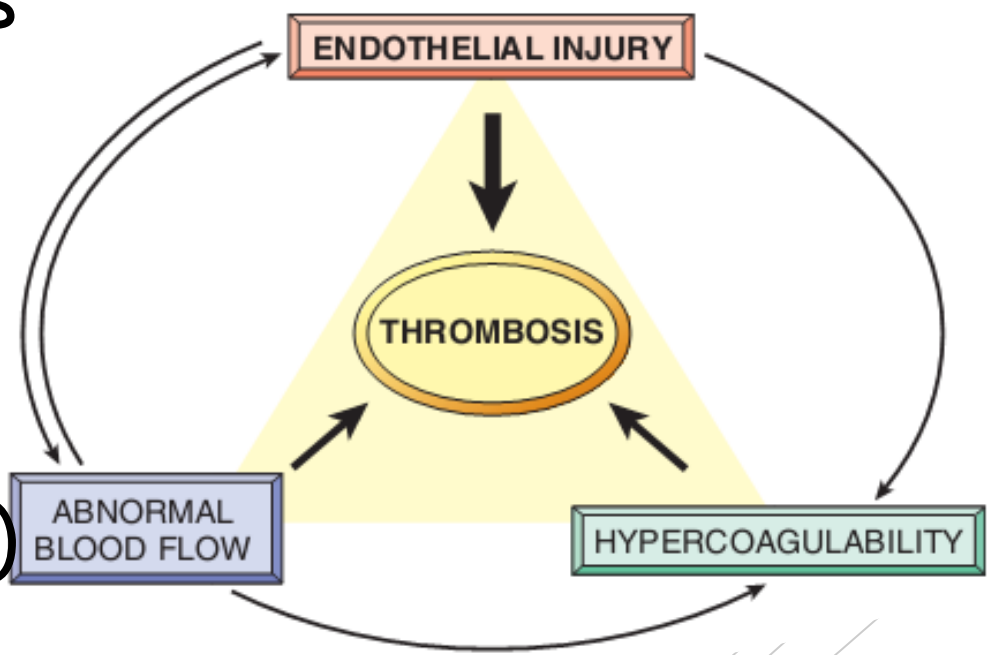
Hematoma: Interstitial hemorrhage with mass effect

Thrombosis
Definition

Formation of a compact mass composed of the circulating blood elements inside a vessel or a heart cavity during life.

Causes & pathogenesis: (Virchow's triad)

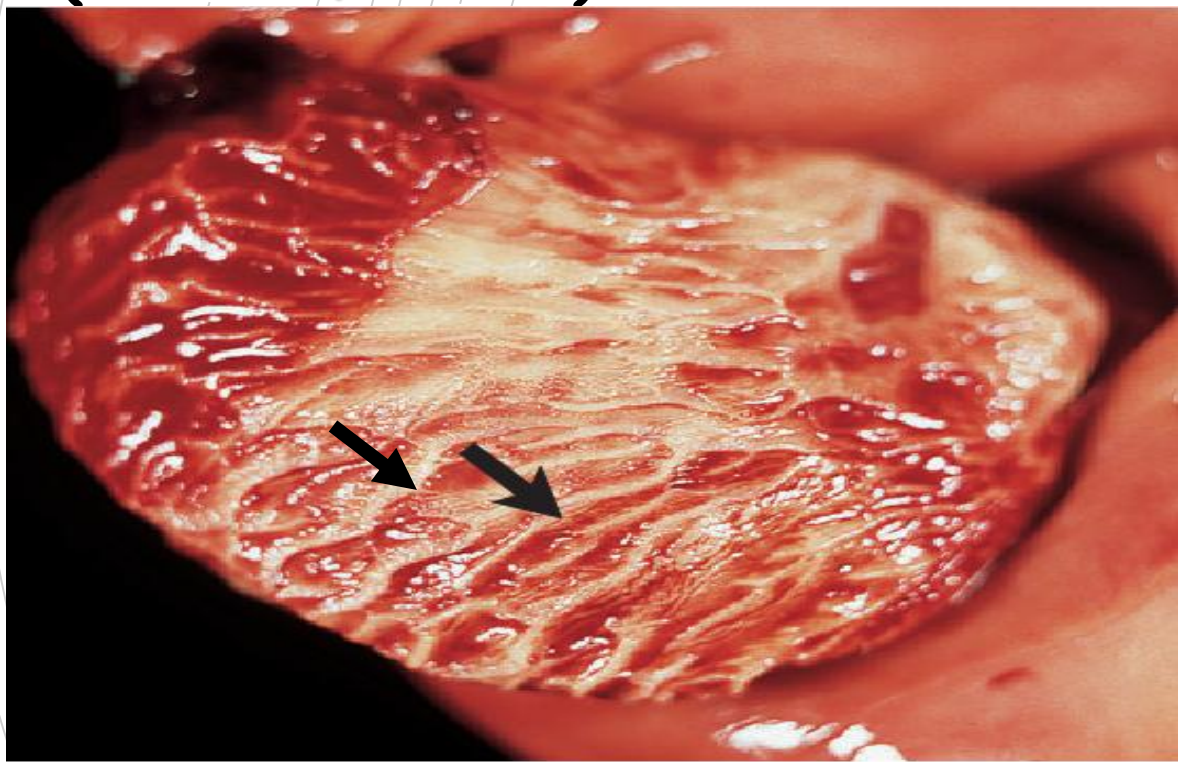
1. Roughness of the intima (Endothelial injury) as in: trauma, inflammation, atherosclerosis
2. Slowing of blood flow (stasis) as in: heart failure, prolonged bed rest
3. Changes of composition of blood (hypercoagulability) as in: leukemia, polycythemia, dehydration (↓ plasma)



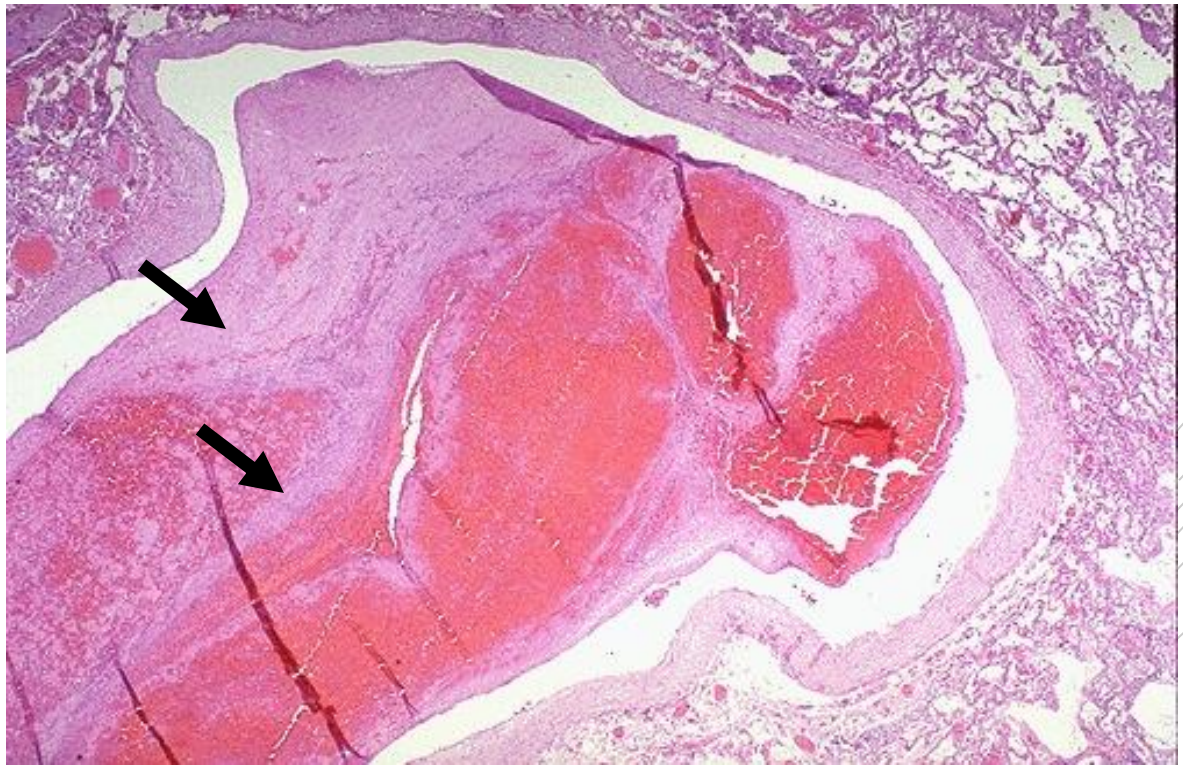
Thrombosis Morphology

N/E: Adherent to vessel wall

Firm, friable and rough with apparent laminations (lines of Zahn)



M/E: lines of Zahn are pale (platelet and fibrin) alternating with darker red cell-rich layers.



Thrombosis
Pathological Effects

Venous

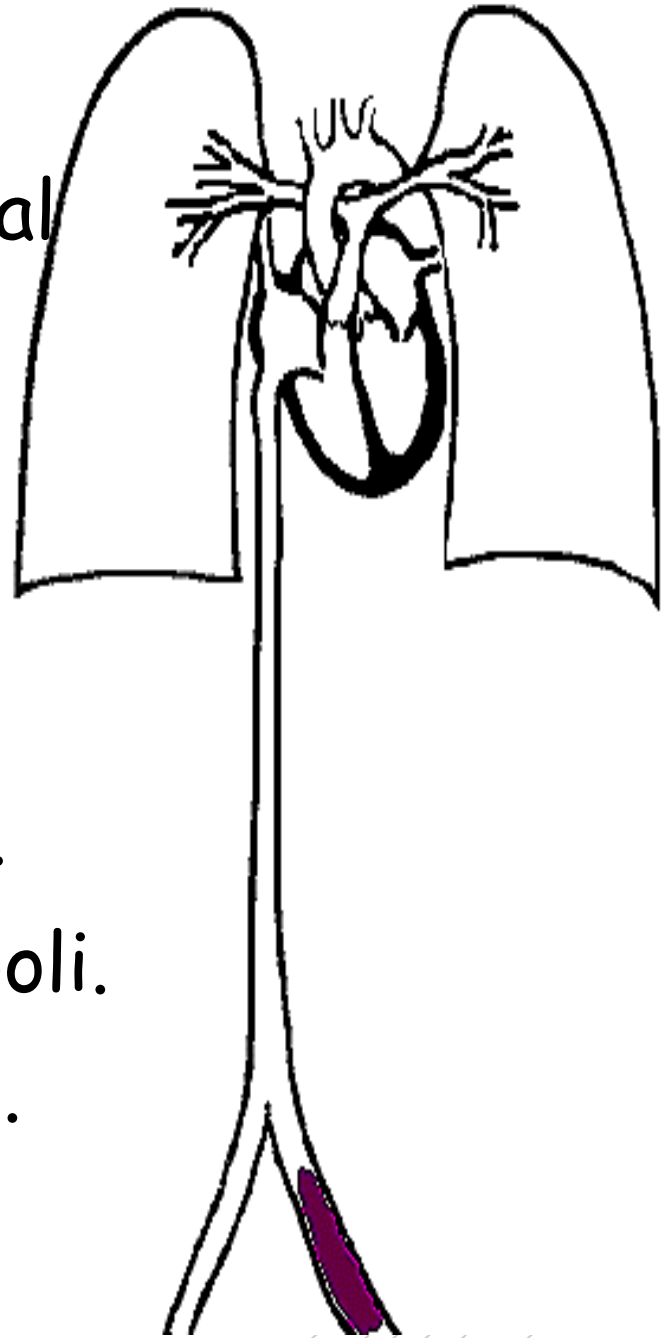
- Congestion
- Edema
- Embolization:
  - Aseptic(infarction)
  - Septic (pyemic abscess)

Arterial

- ischemia
- Infarction
- Gangrene

Embolism
Definition

Embolism: The circulation of an **insoluble** material (solid, liquid, or *gaseous*) **in the blood** and its sudden **impaction** in a narrow vessel. This material is called an **embolus**.



Types of emboli

1- Thrombo-embolism.

2- Fat embolism.

3- Air embolism.

4- Parasitic emboli.

5-Amniotic fluid embolism.

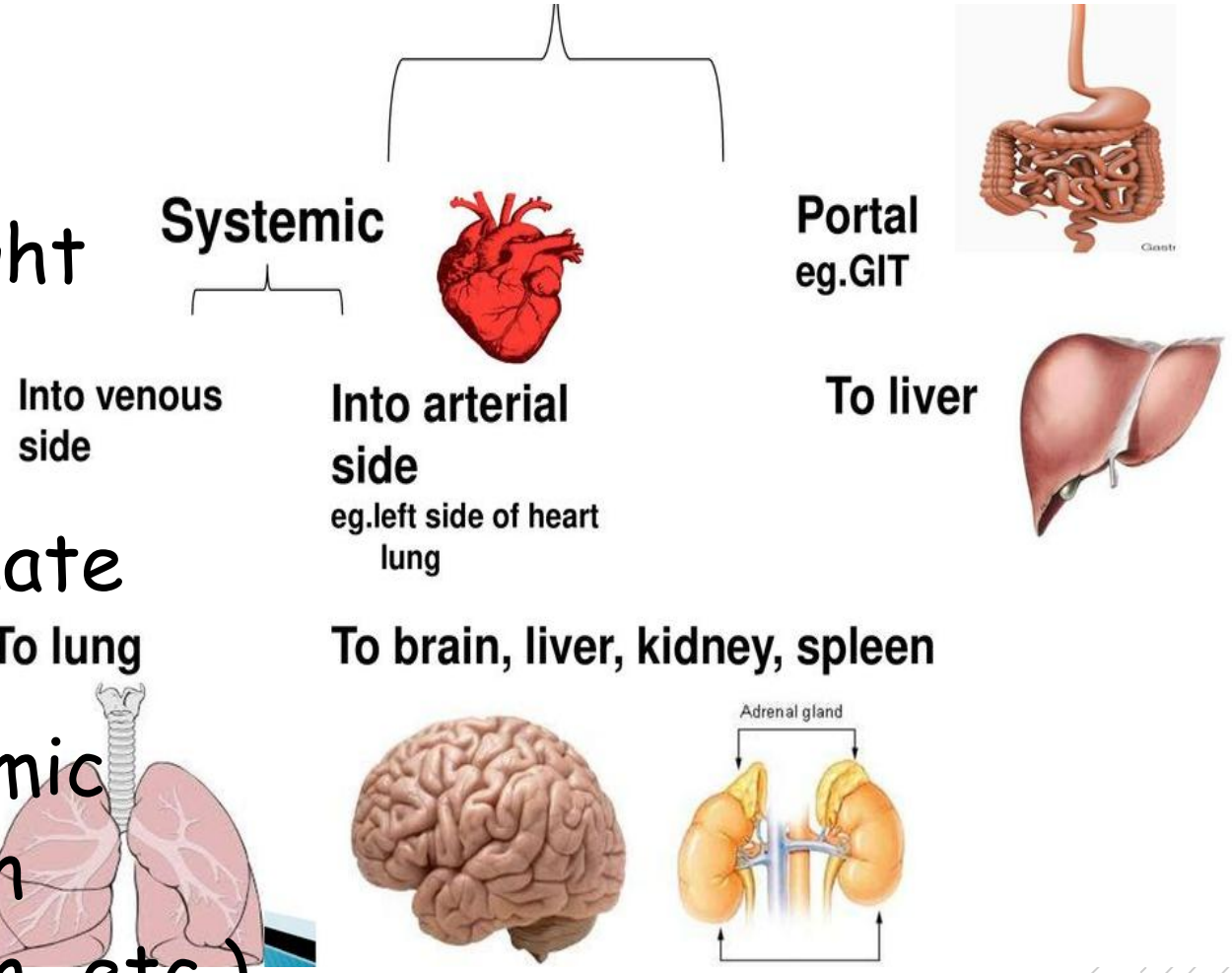
6- Tumor emboli.

Embolism Types

1- Thrombo-embolism 99%

Origin:

- Systemic veins reaches the right side of heart, then lungs → pulmonary embolism
- Cardiac thrombi: Usually originate in the left side of the heart. They are carried by the systemic arterial circulation to impact in any organ (spleen, kidney, brain…etc.)
- Portal vein or its branches passes to the liver (portal embolism).

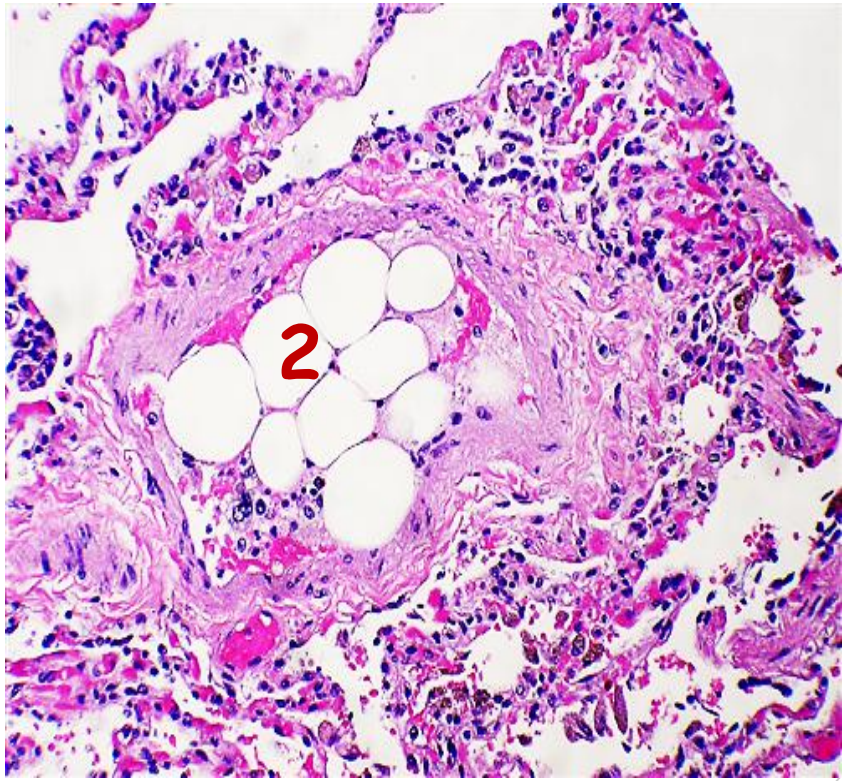


Embolism
Types

2-Fat embolism.

Bone fractures
Severe burn
Acute pancreatitis
Atheroma (atherosclerosis)

Fat globules enter through the ruptured veins



Embolism Types

3- Air emboli.

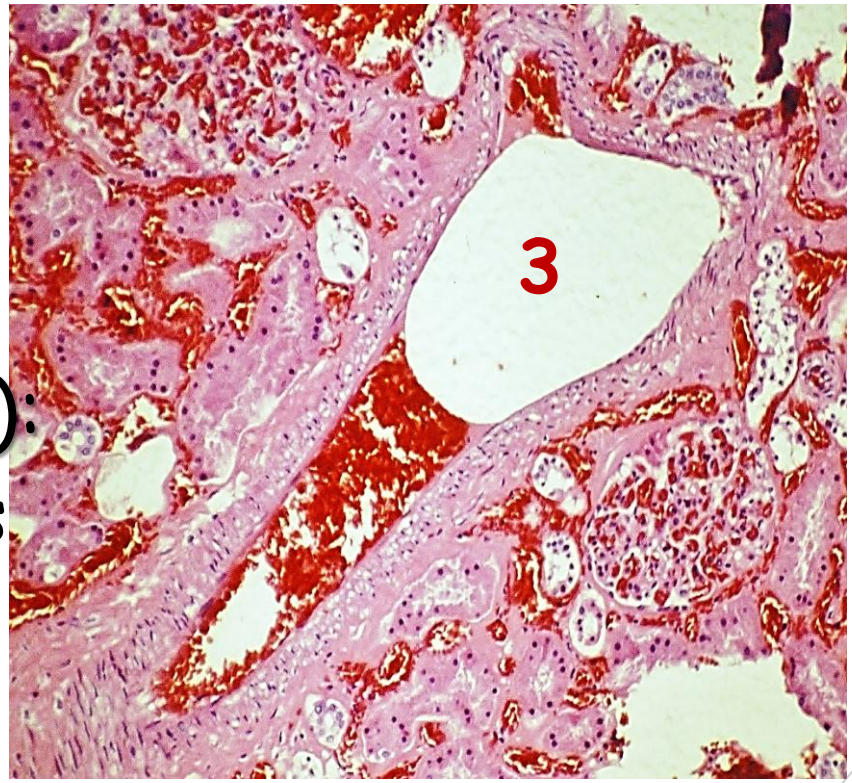
100 mL or more can lead to sudden death.

Air enter the venous circulation through

*Injury of neck veins

*Faulty injection technique

*Decompression sickness(Caisson's disease):



-A type of air embolism occurs when deep divers work under high atmospheric pressure, where their nitrogen gas is dissolved in the tissues and blood.

-Sudden decompression (sudden ascent) produces nitrogen bubbles which act as gas emboli.

-Spinal cord is mainly affected.

Embolism Types

4- Parasitic emboli.

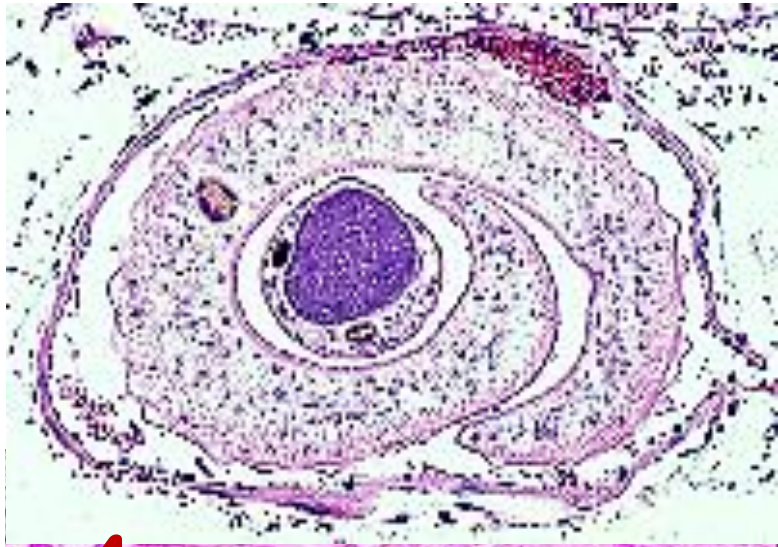
Bilharzial ova and worms

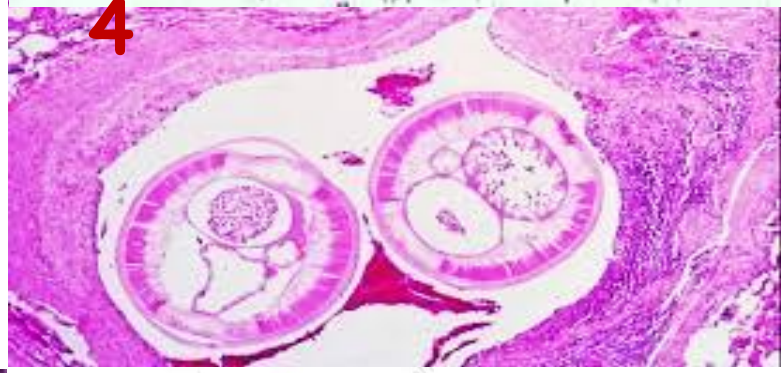
5- Amniotic fluid embolism.

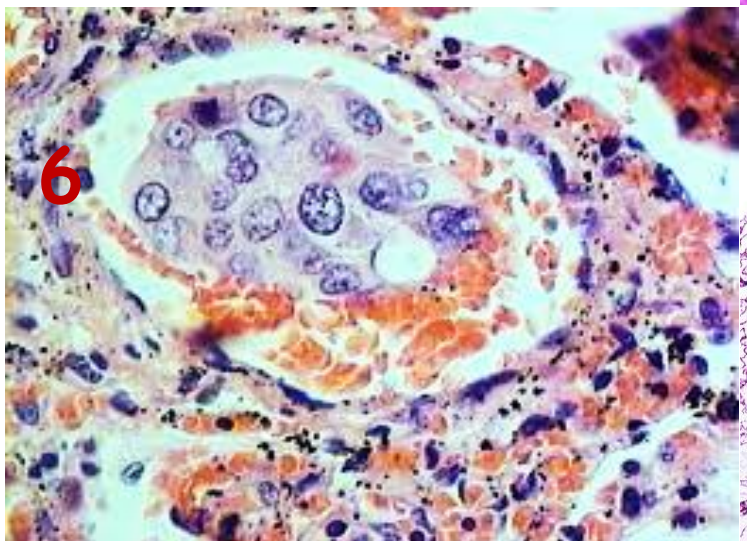
Rare, occurs during delivery→ fatal pulmonary embolism.

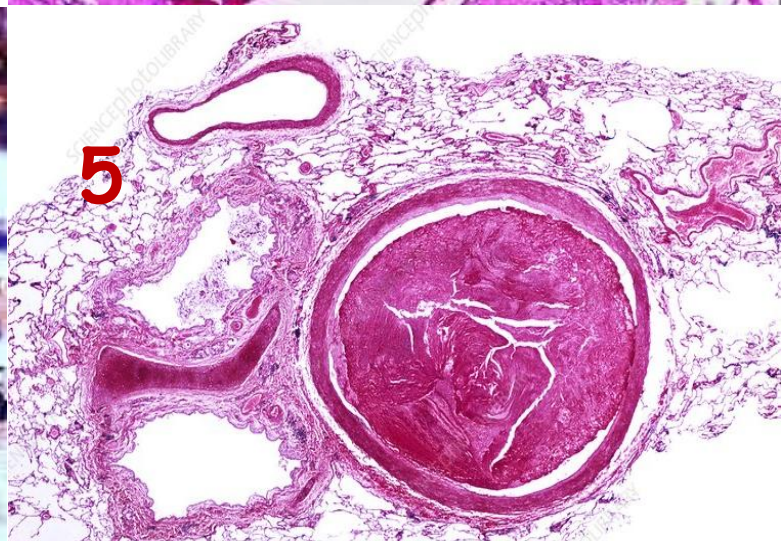
6- Tumor emboli:

Causes distant hematogenous spread





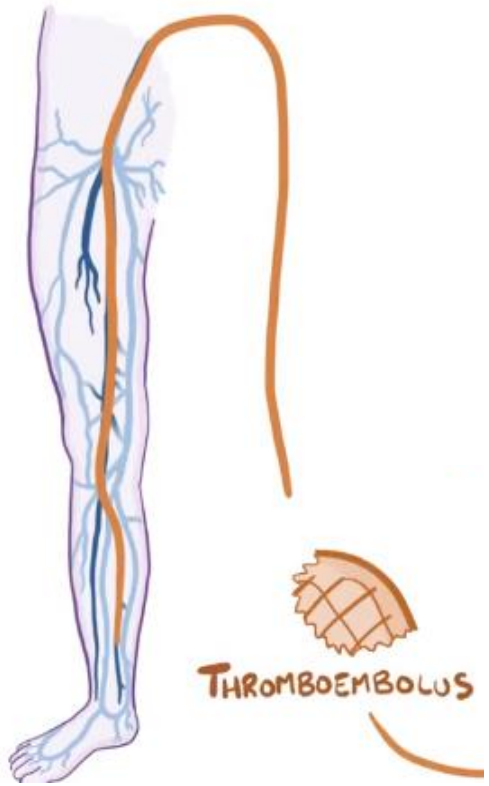


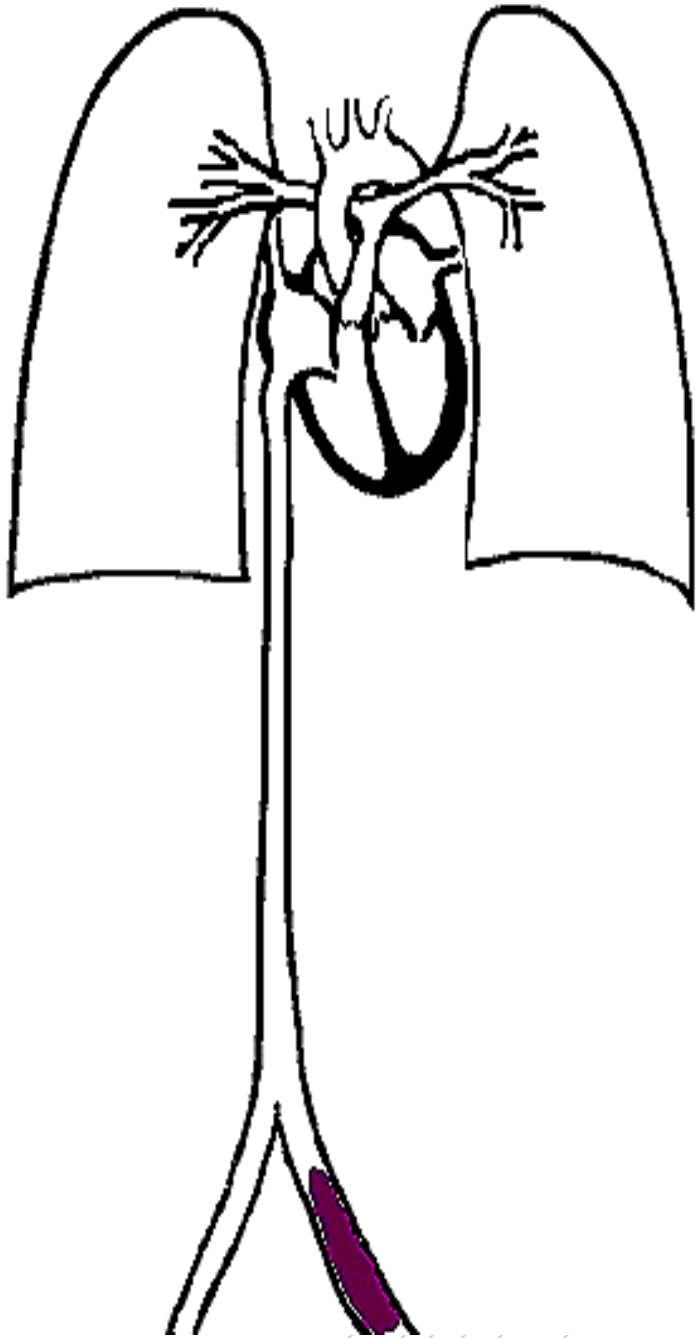


Pulmonary embolism
Causes

Pulmonary embolism Could be

- Thrombo-embolism from systemic veins; the most common is deep vein thrombosis
- Others as fat embolism
  air embolism





Ischemia
Definition

Decrease of blood supply to a part of tissue due to occlusion of its artery.

Types of ischemia:

- Sudden(acute)ischemia:
  - *Thrombosis
  - *Embolism
  - *Arterial spasm
  - *Surgical ligature
  - *Twisting of the organ's pedicle
- Gradual(chronic)ischemia:
  - *Pressure on the artery by tumor or enlarged L.N.
  - *In the wall of the artery as atherosclerosis

Ischemia
Effects

*No effect * Minimal effect(degenerative or atrophic)

*Death of tissue *Death of person

Effects of embolus depends on:

- Nature of the vascular supply (end artery or dual blood supply)
- Rate of vascular occlusion
- Vulnerability of tissue to hypoxia: Neurons (3 to 4 minutes), Myocardial cells (20 to 30 minutes)
- Oxygen content of the blood

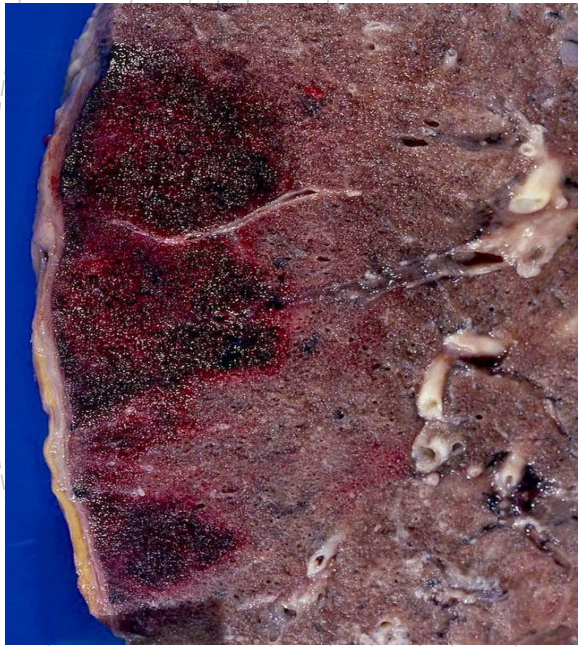
Infarction

Definition

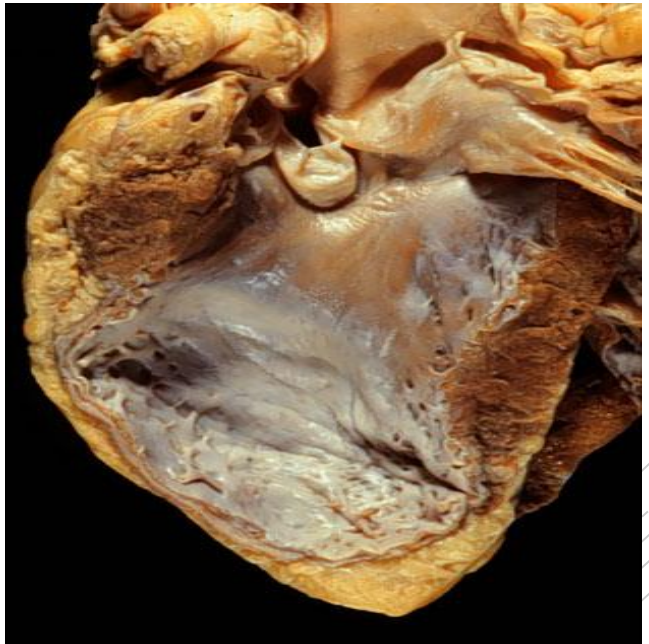
It is an area of ischemic necrosis (coagulative or liquefactive) caused by occlusion either of arterial supply or rarely venous drainage of a particular tissue.

Types

Red(hemorrhagic)infarct: in soft and vascular organs as intestine and lung.



Pale(anemic)infarct: in firm and less vascular organs as Kidneys and heart.

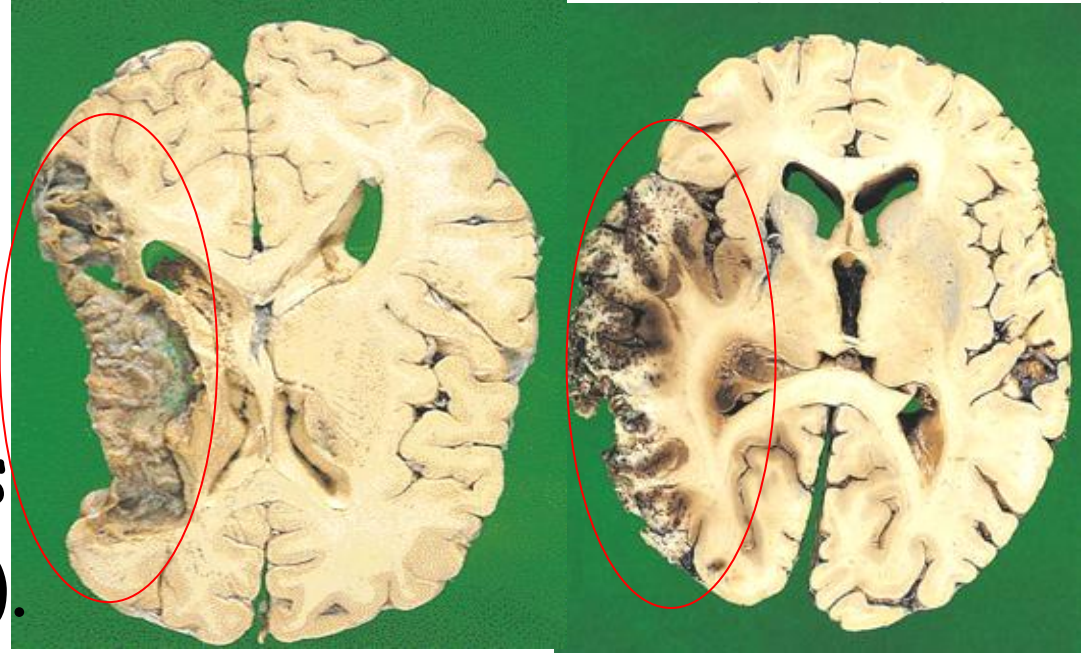


Infarction of brain and spleen may be pale or red.

Infarction Fate

Small infarct:

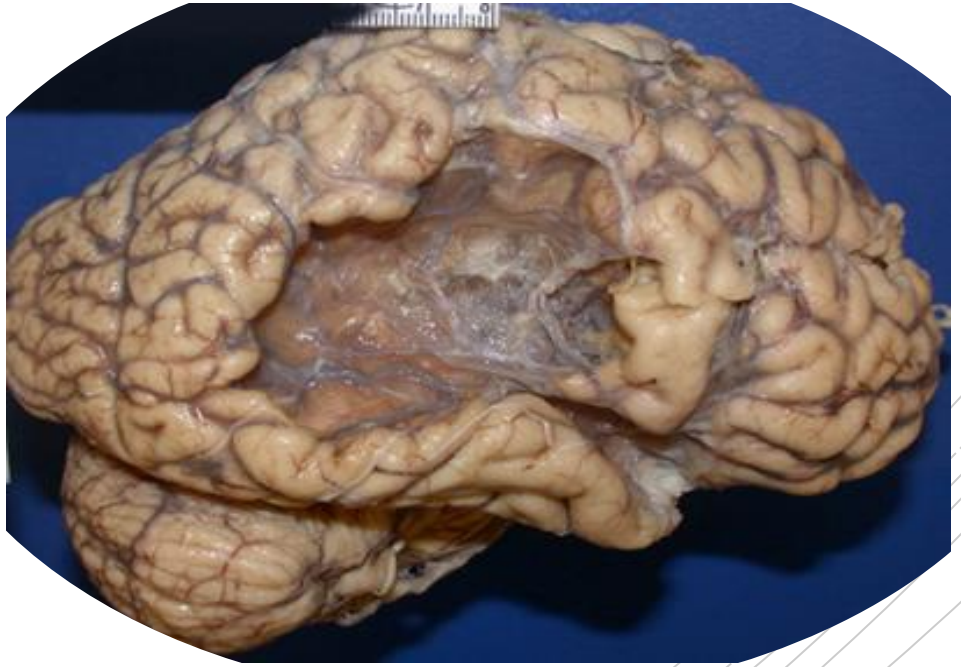
Necrotic tissue is removed by macrophages→ granulation tissue fills the defect→ fibrosis (gliosis in brain).

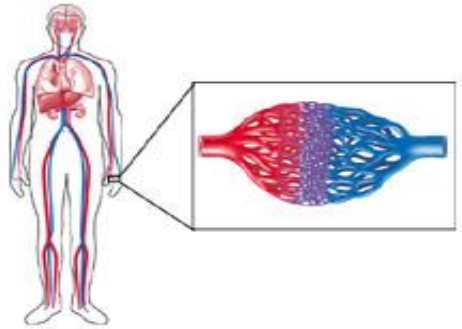


Large infarct:

Surrounded by fibrous capsule, may show dystrophic calcification.

In the brain(due to high lipid content): it leaves a cyst surrounded by glial tissue.





Vascular Disorders

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Atherosclerosis, Def.& Incidence

Def.:It is a chronic, progressive, multifocal disease of the vessel wall characterized by formation of fibro-fatty plaques.

Incidence:

*Males are more affected than premenopausal females. After menopause, males and females are equally affected

* More in developed countries

Atherosclerosis, Risk Factors

Major

Non-modifiable

Increasing age

Male gender

Family history

Genetic abnormalities

Modifiable

Hyperlipidemia

Hypertension

Cigarette smoking

Diabetes

Minor

Obesity

Physical inactivity

Stress ("type A" personality)

Postmenopausal

High carbohydrate intake

Alcohol

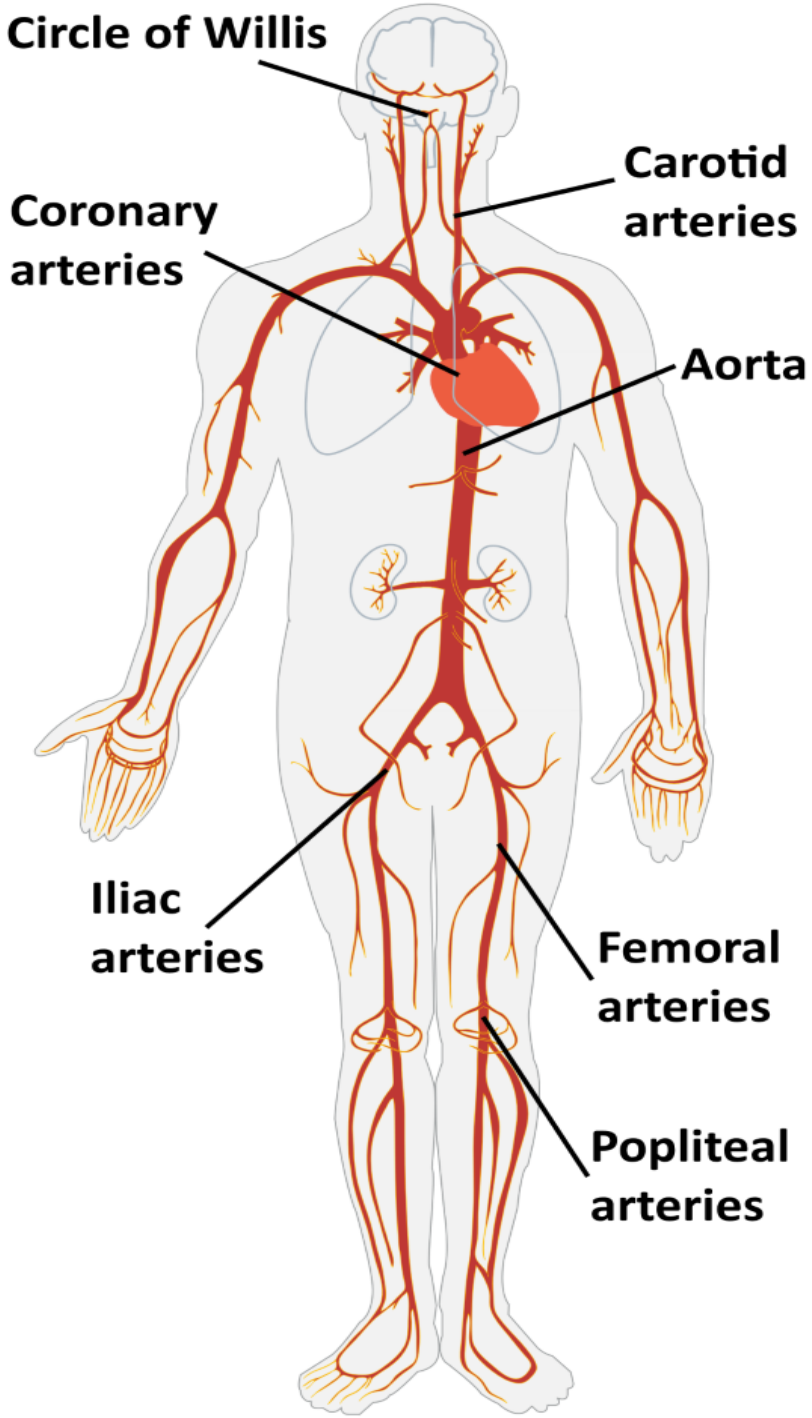
Lipoprotein Lp(a)

unsaturated fat intake

Atherosclerosis, Sites

In order of frequency:

1. Aorta, especially descending
2. Coronaries
3. Cerebral & internal carotids.
4. Femoral , renal, superior mesenter



Atherosclerosis, Pathogenesis

Response to injury theory

- Development of focal regions of chronic endothelial cell injury caused by endotoxins, hypoxia, cigarettes, virus, stress
- Insudation of lipoproteins into the intima of the vessel? LDL,VLDL
- Migration of the monocytes into the intima and transformed into macrophages and foam cells due to engulfing lipid causing lipoproteins oxidation
- Adhesion of platelets and secrete platelet derived growth factor (PDGF)

Atherosclerosis, Pathogenesis

- Smooth-muscle migration: mediated by mitogenic factors released from activated platelets, macrophages, endothelial cells .
- Proliferation of smooth muscle cells in the intima and deposition of Extracellular matrix and collagen and proteoglycans
- Lipid accumulation extra-and intra-cellular in macrophages and smooth muscle cells that become foam cells.
- Formation of fibrous cap that separate the lesion from the lumen

Atherosclerosis, Morphology, Gross

- **Fatty streak:**

✓ Multiple, smooth, yellowish fatty dots.

✓ Not raised above endothelial surface (flat)

✓ • Coalesce to form larger ones

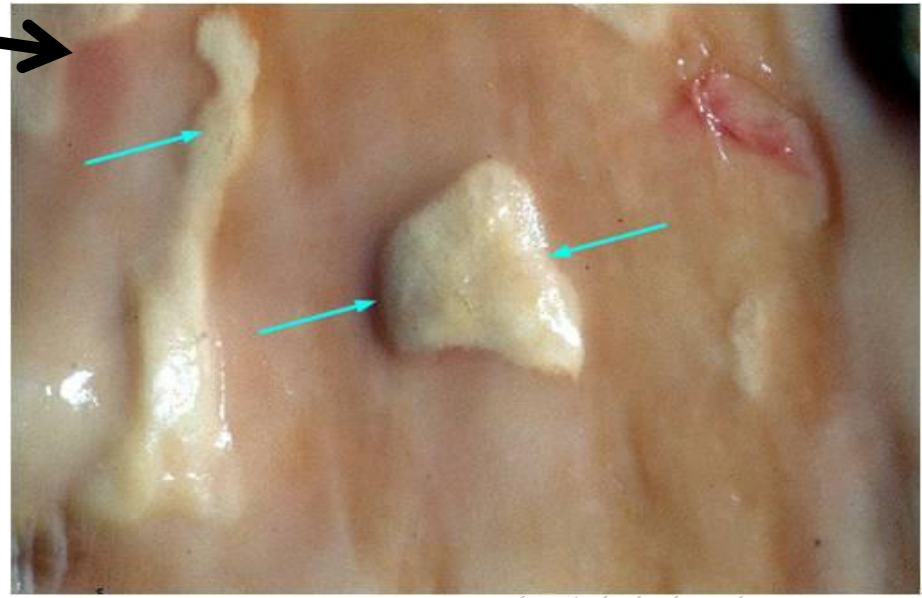
- **Uncomplicated atheroma:**

✓ disc- like patches.

✓ Color ranges from yellow to white according to relative amount of fat and fibrous tissue

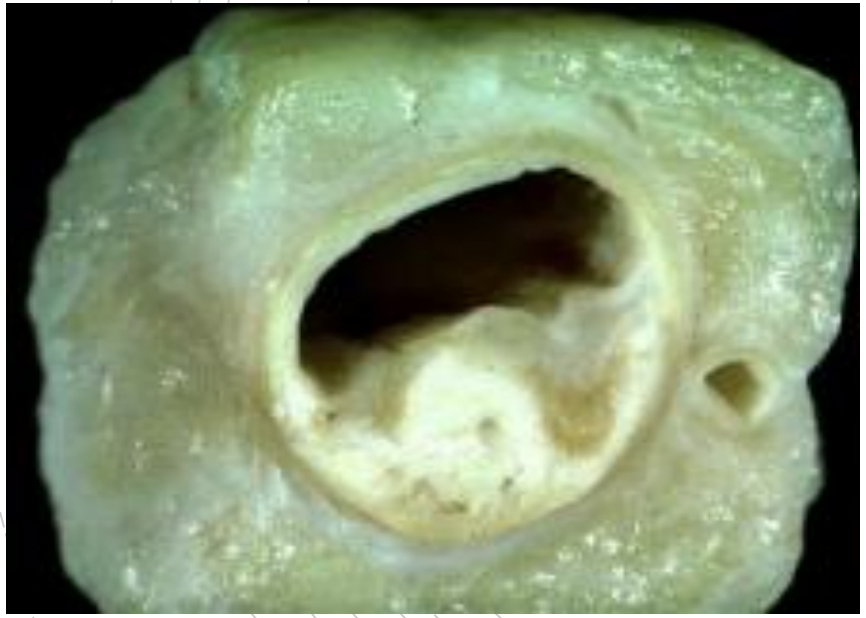
✓ covered by glistening intima.

✓ more around the mouths of the branches.

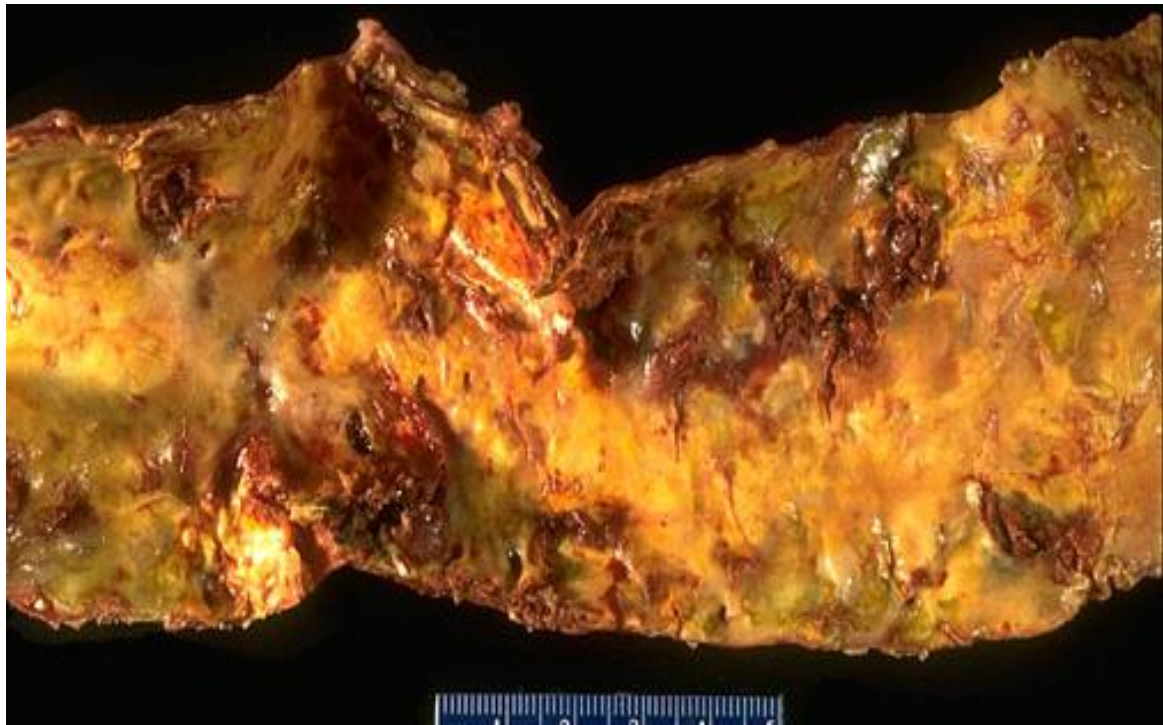


Atherosclerosis, Morphology, Gross

- **Cut surface**
  - ✓ grayish white, firm
  - ✓ fibrous coat and a yellow soft center



- **Complicated atheroma:**
  - ✓ calcification, ulceration, thrombosis



Atherosclerosis, Morphology, Microscopic

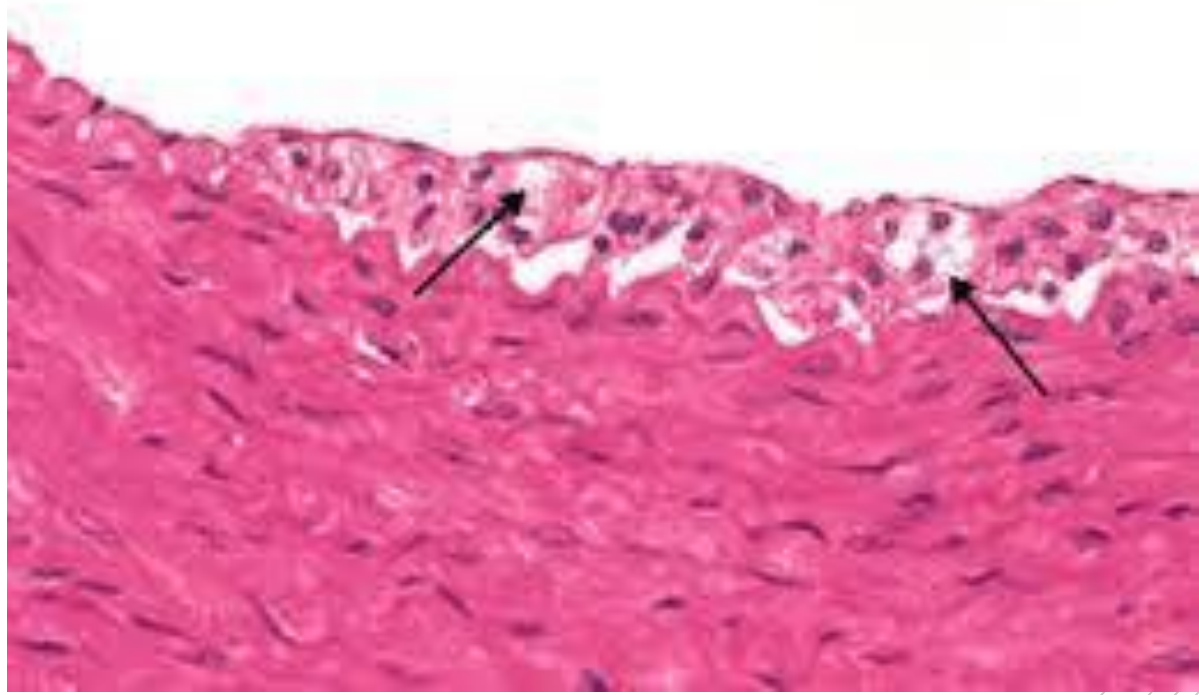
- **Fatty streak:**

✓ Cholesterol clefts.

✓ Lipid-laden foam cells

✓ Extracellular lipid

✓ Lymphoplasmacytic infiltrate

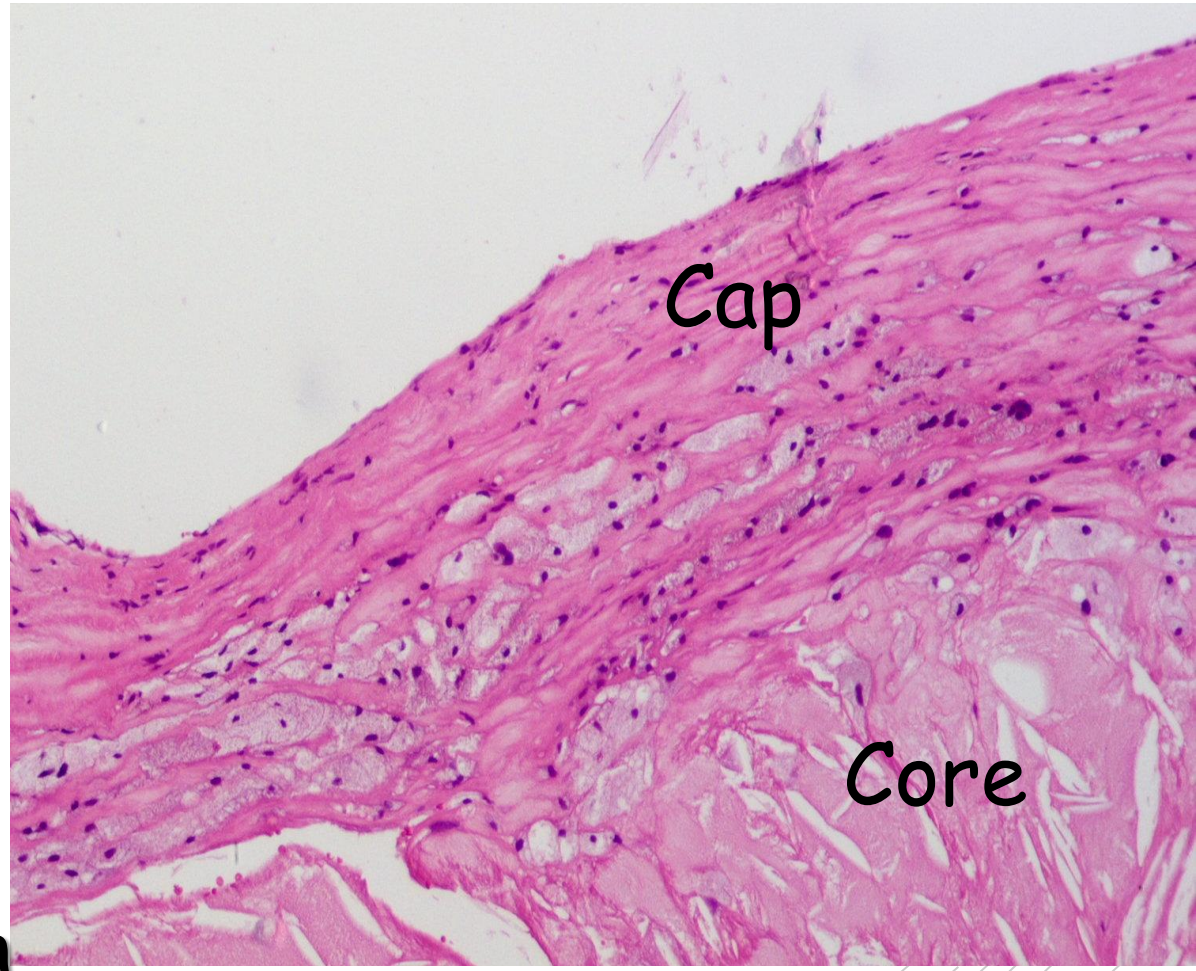


Atherosclerosis, Morphology, Microscopic

- **Uncomplicated atheroma plaque**

1- Subendothelial fibrous cap formed of proliferated smooth muscle cells, foam cells and extra cellular matrix

2- Central core of cholesterol and cholesterol esters, lipid laden macrophages (foam cells), necrotic debris and calcification.



Atherosclerosis, Morphology, Microscopic

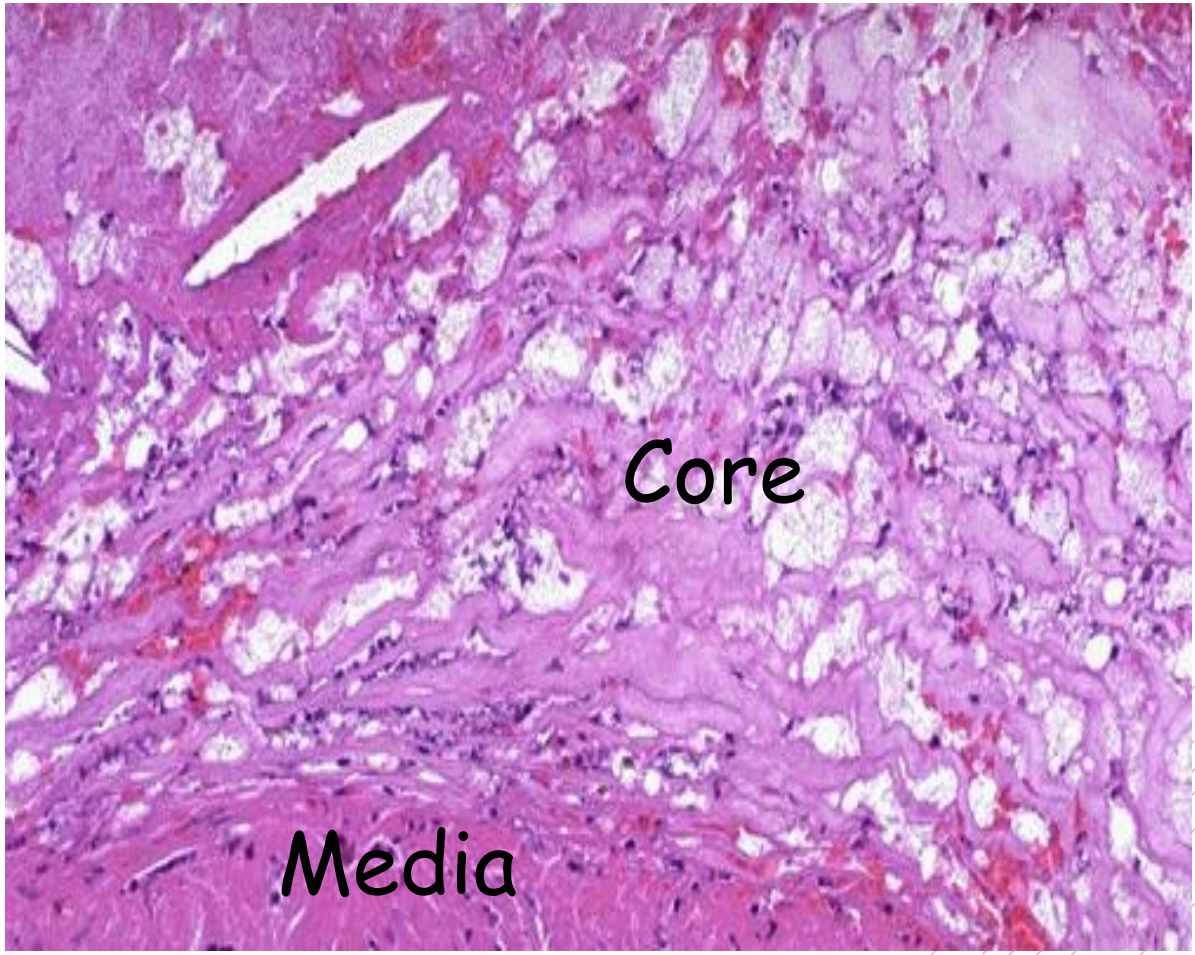
- **Uncomplicated atheroma plaque (cont.)**

3- Neovascularization around the periphery.

4- Media:

Disrupted elastic lamina.

Atrophy of the media deep to the plaque



Atherosclerosis, Complications

1- Narrowing of vascular lumen…chronic ischemia.

2- Superimposed thrombosis…acute ischemia… Infarction

3- Ulceration with liberation of fatty core …fat emboli… acute ischemia… Infarction

4- Dystrophic calcification.

5- Pressure atrophy of the media with fibrosis….weakening of the wall …. Aneurysm.

Now,
can you answer the following?

Which one is a modifiable major risk factor of atherosclerosis?

a. Genetics.
b. Age.
c. Gender.
d. Hypertension.
e. Genetic abnormalities.

Aneurysm, Def., Classification

Def.: An aneurysm is a localized abnormal dilation of a blood vessel or the heart.

Classifications:

1-True or false aneurysm.

2-According to size and shape aneurysms are classified into :

- Saccular aneurysms -Fusiform aneurysms

3-According to etiology:

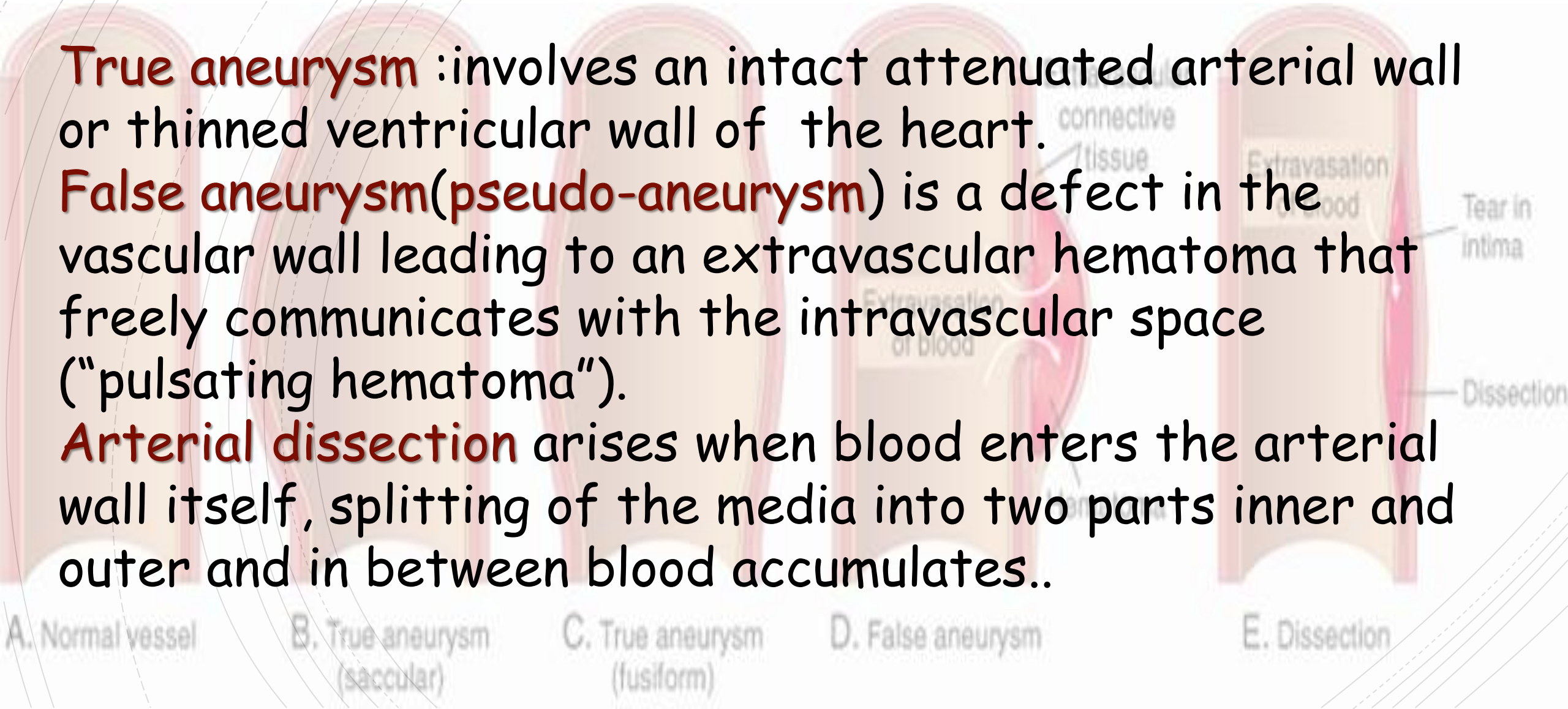
- Congenital -Acquired

Aneurysm, Def., Classification

True aneurysm :involves an intact attenuated arterial wall or thinned ventricular wall of the heart.

False aneurysm(pseudo-aneurysm) is a defect in the vascular wall leading to an extravascular hematoma that freely communicates with the intravascular space ("pulsating hematoma").

Arterial dissection arises when blood enters the arterial wall itself, splitting of the media into two parts inner and outer and in between blood accumulates..



Aneurysm, Causes

*Weakening of arterial wall:

-Congenital: As Berry aneurysms of brain (cerebral arteries).

-Traumatic: As arterio-venous fistula.

•Infectious : Mycotic aneurysms, T.B. syphilis.

•Immunological injury.. vasculitis as Polyarteritis Nodosa.

-Degenerative: As atherosclerosis (atheroma)

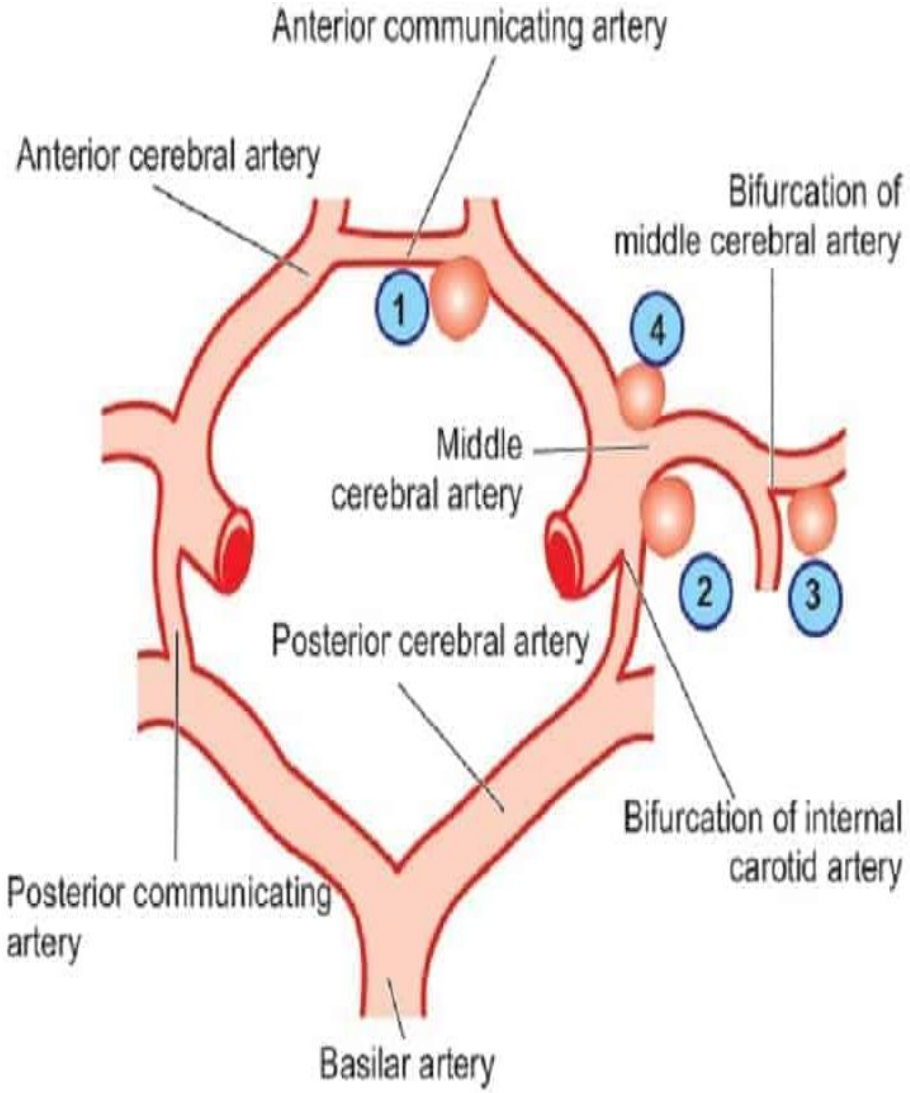
*Stretching of the weakened wall by increased pressure (hypertension), complicating healed cardiac infarction

Aneurysm, Causes

Congenital: Most commonly affects circle of Willis in the brain (berry aneurysm)

Causes: They are caused by congenital defects of the media. Their rupture causes subarachenoid or cerebral hemorrhage.

Morphology: single or multiple saccular small aneurysms, up to 2 cm in diameter at the points of bifurcation of the cerebral arteries.



Aneurysm, Causes

Mycotic aneurysm: It occurs mainly in coronary, cerebral or mesenteric arteries

Causes: Originate at the site of septic embolus(as from infective endocarditis) within a vessel or bacteria directly infecting the arterial wall.

Morphology: Mild inflammation occurs in the arterial wall followed by fibrosis, and small aneurysmal dilatation. ischemia is common.

Aneurysm, Causes

Atherosclerotic

Site: Aortic mainly abdominal aorta, located usually below the renal arteries.

Morphology: Saccular (balloon like), up to 15 cm in greatest diameter and of variable length (up to 25cm).

- Mural thrombus frequently found inside.

Complications: The most serious complication is rupture into peritoneal cavity or retroperitoneal tissue causing massive or fatal hemorrhage.

Aneurysm, Causes of aotic & cerebral

Aortic aneurysms

- Atheromatous

-It is the commonest type.
-In descending thoracic aorta, below renal arteries.

- Syphilitic aneurysm

-In the tertiary syphilis. Mainly in arch of aorta.
-It is fusiform or saccular

Cerebral aneurysms

- Congenital: Occur mainly in circle of Wills. They are multiple and small
- Intracranial microaneurysms of benign hypertension.
- Mycotic aneurysms Emboli of subacute bacterial endocarditis.
- Atheromatous

Aneurysm, Complications

*Occlusion: Direct pressure or mural thrombus formation, particularly of the iliac, renal, mesenteric or vertebral branches that supply the spinal cord.
*Embolism from the atheroma or mural thrombus.
*Compression over adjacent structures Presentation as an abdominal mass (often palpably pulsating) that simulate a tumor such as compression of a ureter, erosion of vertebra.
*Rupture of the aneurysm
*Dissection

Aneurysm, Dissecting aneurysm

Occurs in the aorta with splitting of the media into inner and outer and in between blood accumulates (producing 2 telescoped tubes).

Causes:

➢Hypertension is the major risk factor: men 40-60 years of age. Intimal tear often precedes the dissection. When intimal tear is present, hypertension can enhance progression.

➢Younger age groups have inherited abnormality of the connective tissue that affects aorta as Marfan syndrome.

➢Iatrogenic: Aortic dissection can be caused by cardiac surgery, coronary artery surgery, cardiac catheterization

Aneurysm,
Dissecting aneurysm

Fate:

Healing may occur either by clotting and organization of the blood in the media or rupture into the lumen of Aorta.

Rupture of the aneurysm with fatal hemorrhage.

Extension into the aortic branches and may narrow their opening leading to ischemia.

Arteritis, Def., Classification

Def.: Inflammation of arteries.

Classifications:

Infective

As (staph aureus).

*Acute as in arteries passing in area of acute inflammation

*Chronic as in (endarteritis obliterans).

Non-infective

Inflammatory immune mediated.

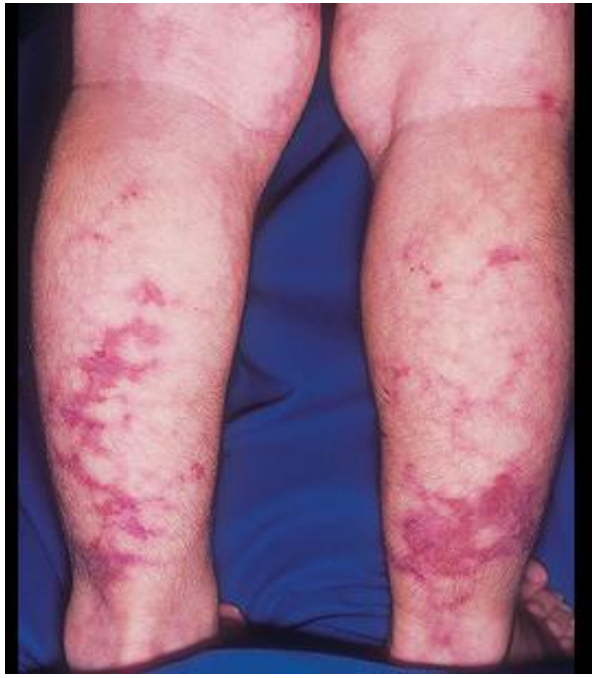
*Polyarteritis nodosa.

*Systemic lupus erythematosus.

*Thrombangitis obliterans.

*Giant cell arteritis.

Arteritis, Polyarteritis nodosa



Def: It is a necrotizing inflammation of small and medium sized arteries caused by type III hypersensitivity reaction.

N/E: Nodular segmental affection ,mostly affects kidney, GIT, CNS and musculoskeletal vessels.

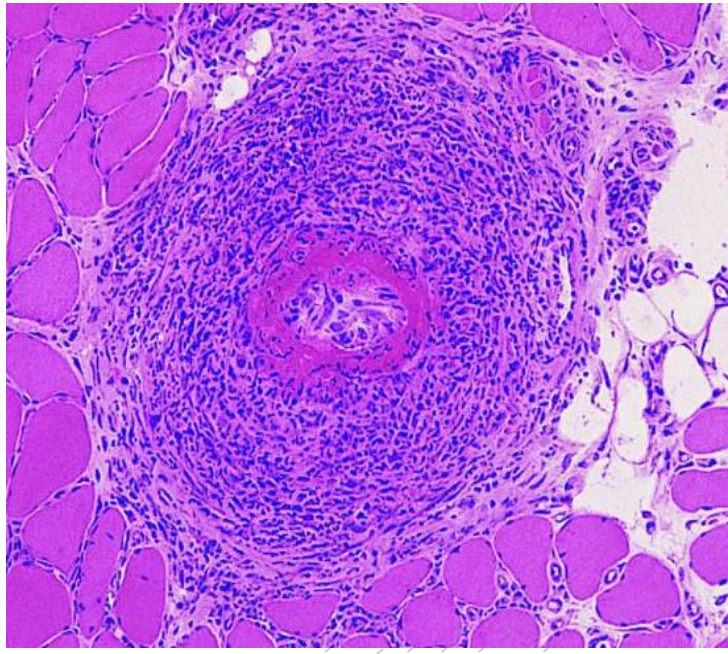
M/E: Panarteritis - fibrinoid necrosis – inflammatory reaction & fibrosis - thrombosis

Complications:

Ischemia: It may be chronic due to fibrosis or acute due to thrombosis.

Rupture due to fibrinoid necrosis leading to hemorrhage.

Aneurysms due to weak fibrosed media.



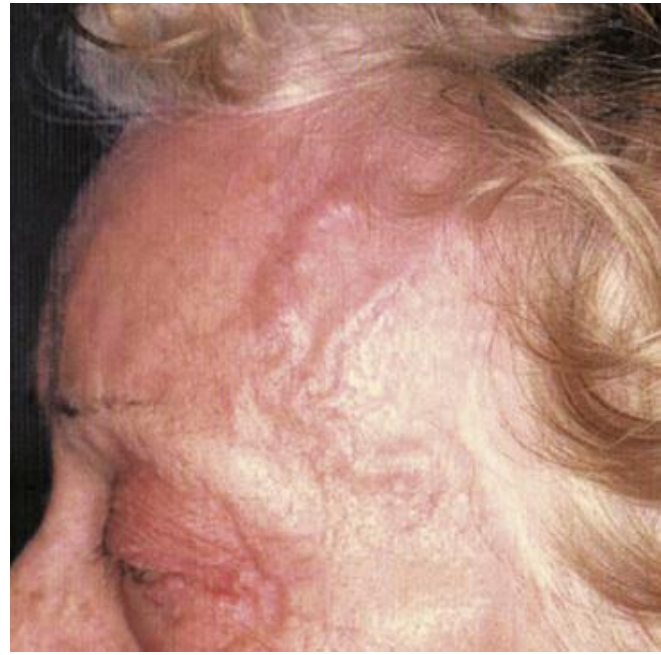
Arteritis,
Systemic lupus erythematosus

Def: Multi-system disease, type III hypersensitivity.

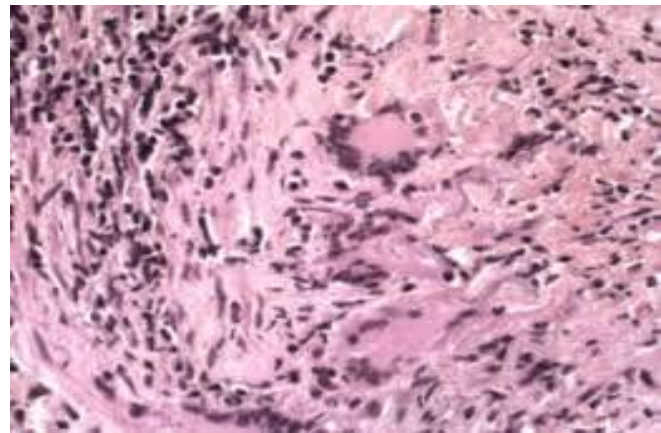
Blood vessels: Small arteries, arterioles or even venules are affected nearly all-over the body.

Death is usually due to:

- Hypertension
- Renal failure



Giant cell arteries (temporal arteritis)



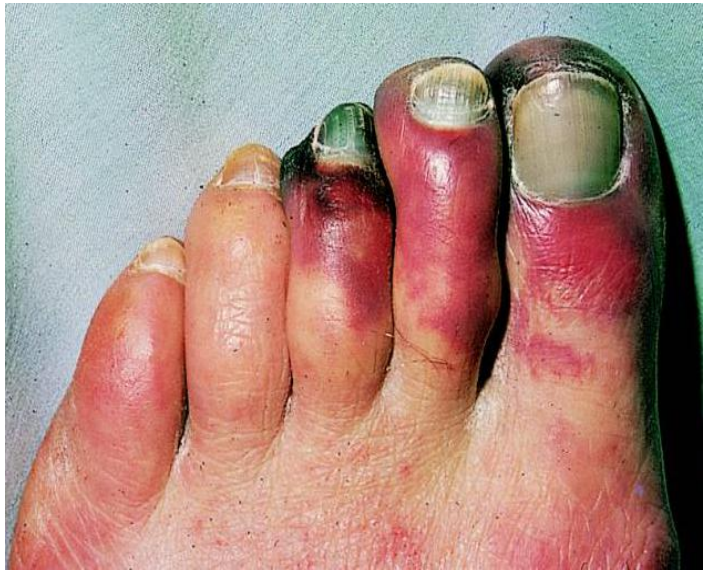
It seen in medium to large sized arteries with granuloma formation.

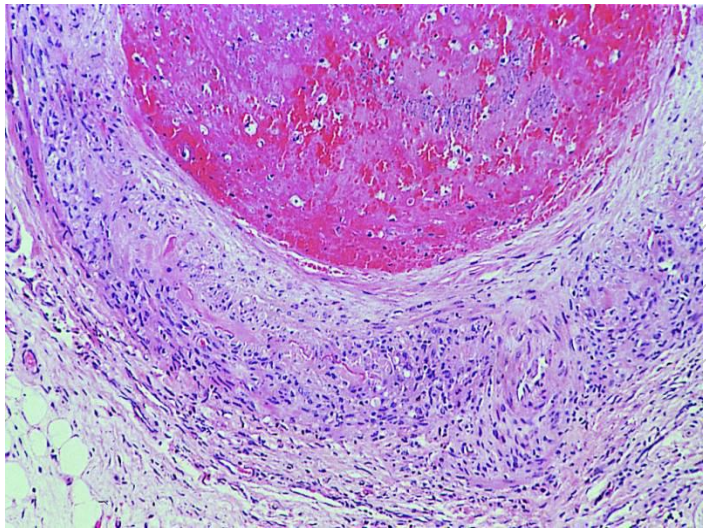
Arteritis, Thromboangitis obliterans

Def: called Burger's disease, is an acute inflammation (followed by chronic) involving small to medium-sized arteries of the extremities extending to adjacent vein and nerve.

Cause: Hypersensitivity to tobacco products with hereditary predisposition.

Complications: Ischemia which may lead to intermittent claudication (chronic ischemia) and gangrene (acute ischemia).





Hypertension, Def., Classification

Def.: Abnormally high blood pressure that consistently above 140/90 on 3 separate occasions.

Classifications according to severity

Benign:

90% of cases

*pressure rises gradually to 180/100

*prolonged for many years

2-Malignant :

5 - 10 %.

*pressure rises suddenly to 200/110

*course is short for months or few years

Hypertension, Classification

Classifications according to cause

I-Essential hypertension (primary)

95% of cases

Risk Factors:

Hereditary/ race/ gender/ age / obesity/ diet/ diabetes/ stress/ sedentary life

2-Secondary hypertension

10 %.

*Renal ischemia as – Renal ischemia(chronic)

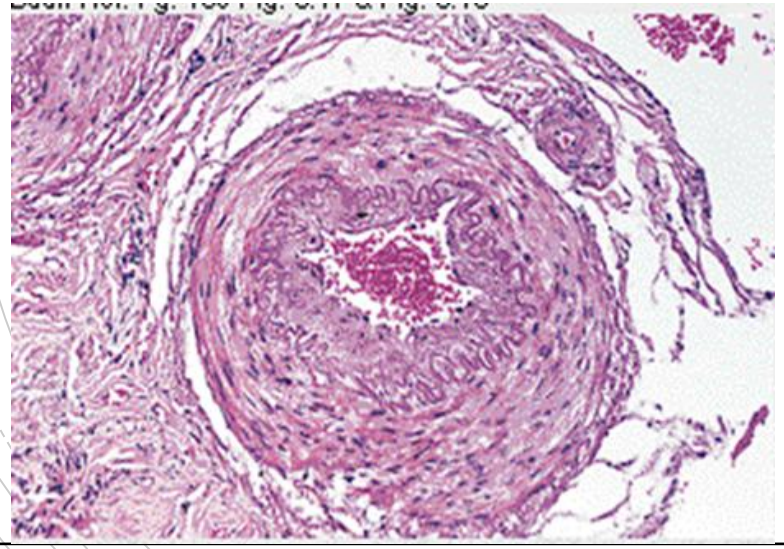
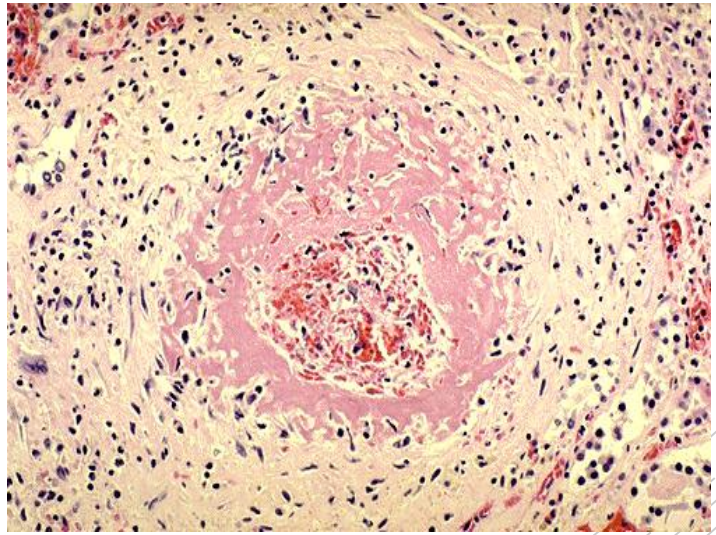
*Endocrine as Thyrotoxicosis.

*Vascular as Coarctation of aorta

Hypertension, Morphology

Character	Benign hypertension	Malignant hypertension
Heart lesions	Concentric left ventricular hypertrophy with bulge of the interventricular septum. Followed by decompensated stage (Left ventricular failure) 	These cardiac changes are minimal (no time to occur).

Hypertension, Morphology

Character	Benign hypertension	Malignant hypertension
Blood vessels lesions	Benign arteriolosclerosis: This is manifested as - Elastosis. 	Malignant arteriolosclerosis: This is manifested as Necrotizing arteriolitis (fibrinoid necrosis) 

Hypertension, Morphology

Character	Benign hypertension	Malignant hypertension
Renal lesions	Benign nephrosclerosis: (contracted kidney). -Progressive chronic renal failure.	Malignant nephrosclerosis The kidneys are normal in size. -Acute renal failure.
Brain lesions	Benign hypertensive encephalopathy: diffuse cerebral dysfunction due to brain edema.	Malignant Hypertensive encephalopathy: The same with liquefactive necrosis due to cerebral hemorrhage.

Hypertension, Morphology

Character	Benign hypertension	Malignant hypertension
Retinal lesions	Benign hypertensive retinopathy: gradual retinal exudate	Malignant hypertensive retinopathy: hemorrhage, necrosis may lead to blindness.

Complications & cause of death

Left ventricular failure
Renal failure
Cerebral hemorrhage

Now,
can you answer the following?

Which of the following is a character malignant hypertension

a. Arterial elastosis
b. Gradual rise in blood pressure.
c. left ventricular hypertrophy.
d. Necrotizing arteriolitis .
e. Acute infective arteritis.

Varicose Veins,
Def., Etiology

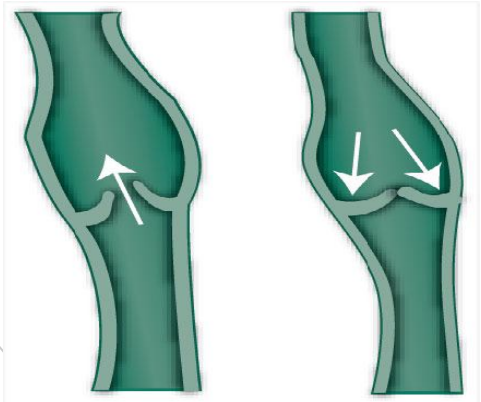
Def.: Dilatation, elongation, thickening and tortuosity of veins.

Etiology:

Predisposing factors

*Familial tendency

*Congenital weakening of veins or valves.



Exciting factors

↑venous pressure → stretching of the wall. Due to:

*Prolonged standing .

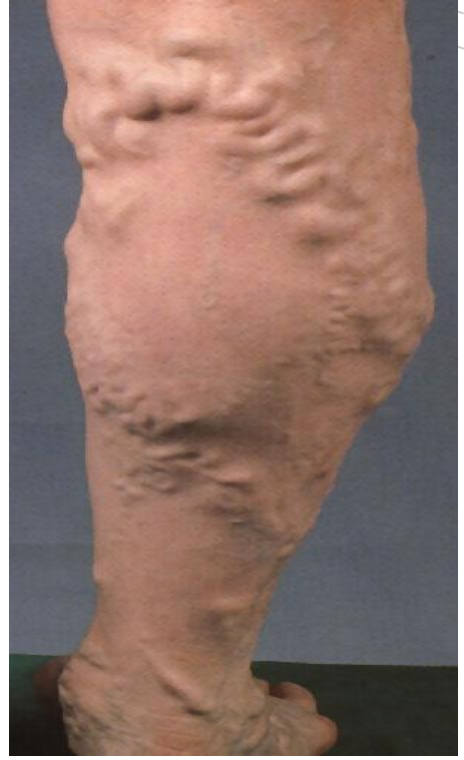
*Chronic constipation and straining .

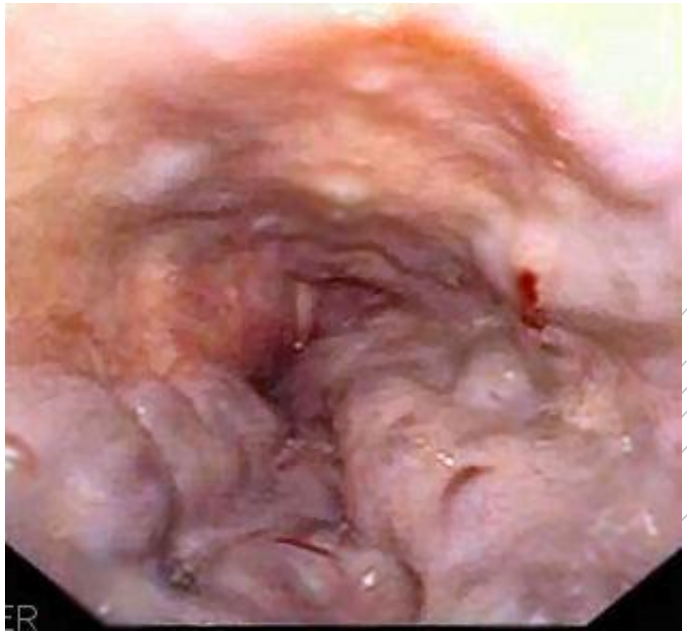
*Obstruction of venous return.

Varicose Veins,
Sites

1- Superficial veins of the lower limbs (Long saphenous vein) ….in persons who spend much of their time standing.

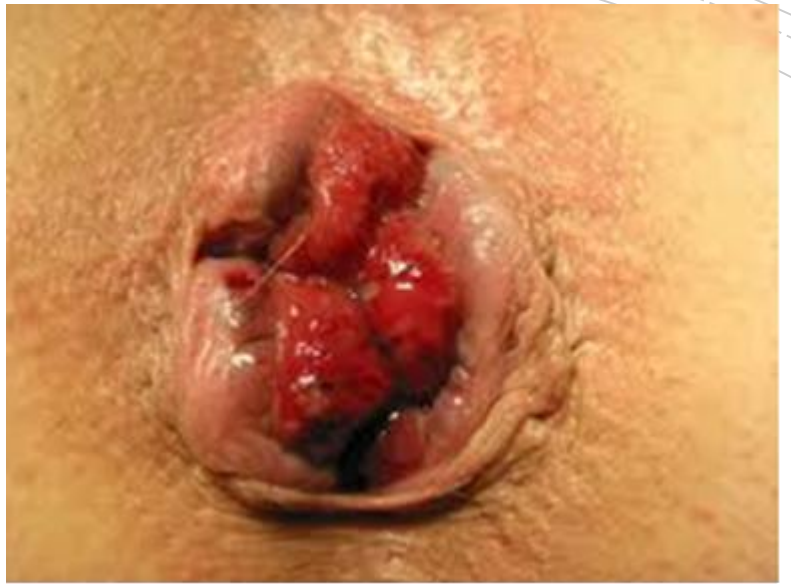
2- Varices of the esophagus Veins of lower 1/3 esophagus and cardia of the stomach. …found in portal hypertension as in liver cirrhosis or bilharzial fibrosis.





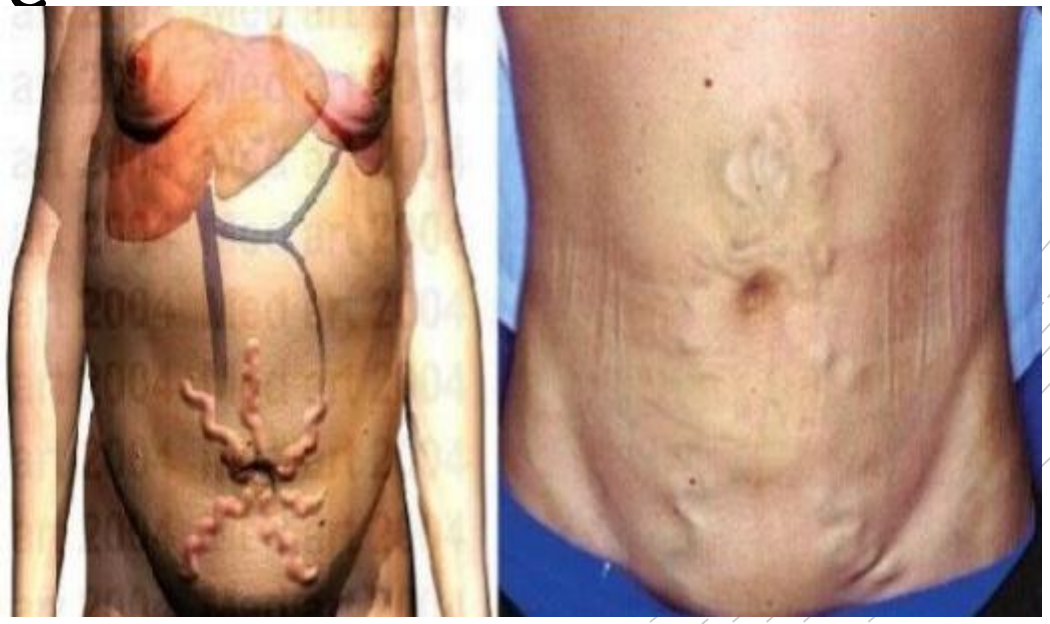
Varicose Veins,
Sites

3- Hemorrhoids …varices of the internal or external hemorrhoidal plexus of the rectum (piles).



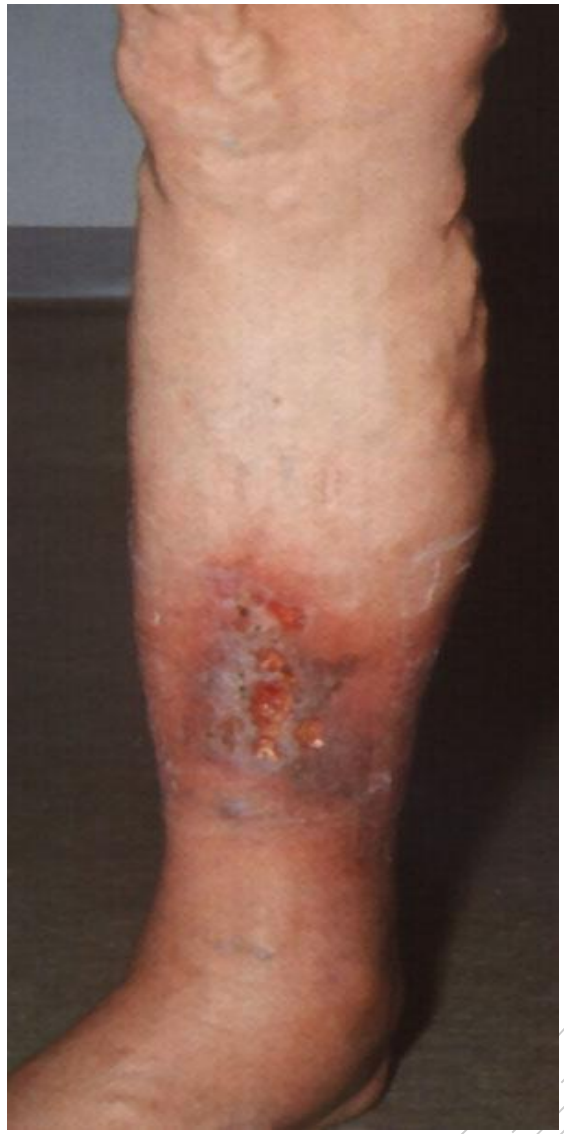
4- Varicocele …….varices of the pampiniform plexus of the spermatic cord.

5- Caput Medusa --- around the umbilicus.



Varicose Veins,
Complications

1- Local chronic venous congestion and persistent edema in limbs and secondary ischemic skin changes, including **stasis dermatitis** and **ulcerations**.



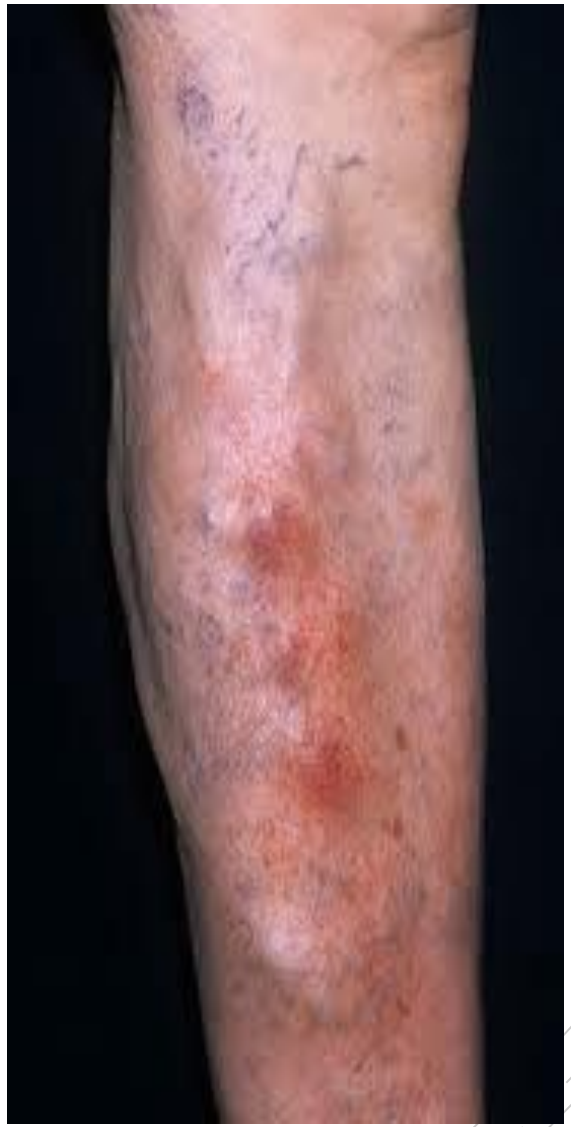
2- Varicose ulcers which are premalignant (gives rise to **squamous cell carcinoma**). It occurs in the lower inner part of the leg above medial malleolus.

Varicose Veins,
Complications

3. Thrombosis and embolism

4. Hemorrhage: Hematemesis and melena from esophageal varices.

5. Inflammation leading to septic thrombophlebitis, septic emboli and pyemia.



Ischemic heart disease (IHD), Def., Etiology

Def.: Is a group of closely related syndromes resulting from myocardial ischemia (coronary heart diseases)

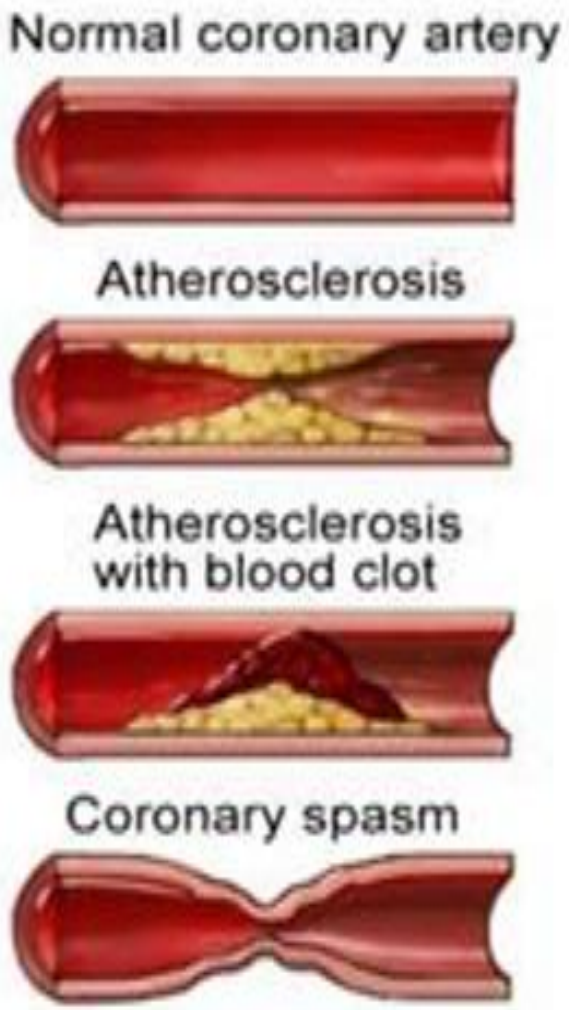
Imbalance between the supply (perfusion) and demand of the heart for oxygenated blood.

Etiology:

*The most common is complicated coronary artery **atherosclerosis**, as **thrombosis**.

***Vasospasm**: constriction of the vessels.

* Vasculitis



Ischemic heart disease (IHD), Clinical Varients

Angina pectoris

Myocardial infarction

Chronic IHD with heart failure

Sudden cardiac death

Ischemic heart disease (IHD),

Angina pectoris

Def. Transient Chest Discomfort (Pain) (15 seconds to 15 minutes), recurrent can radiate down the left arm or to the left jaw.
different qualities (constricting, squeezing, choking, or knifelike)

Cause:

Transit myocardial ischemia from seconds to minutes due to inadequate perfusion then perfusion is restored.
*In angina pectoris there is no death of heart tissue.

Ischemic heart disease (IHD),

Myocardial infarction

Definition:

Massive area of coagulative necrosis of myocardium due to complete cut of arterial blood supply.

Causes It occurs when the severity or duration of ischemia is enough to cause cardiac muscle death.

Risk factors include the risk factors for atherosclerosis:

-Hypertension
-Diabetes mellitus
-Cigarette smoking,
-Genetic hypercholesterolemia

Ischemic heart disease (IHD),

Myocardial infarction

Morphology

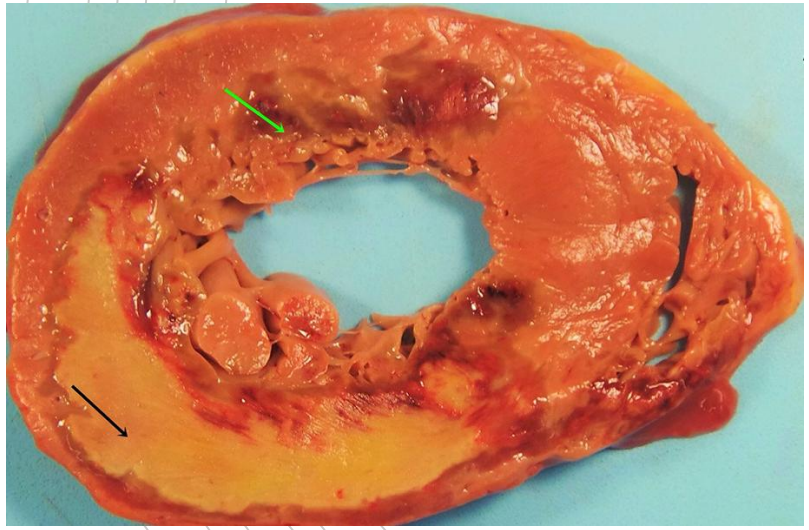
NE

Immediately:

No apparent changes

By 12-72 hours:

pale to yellow



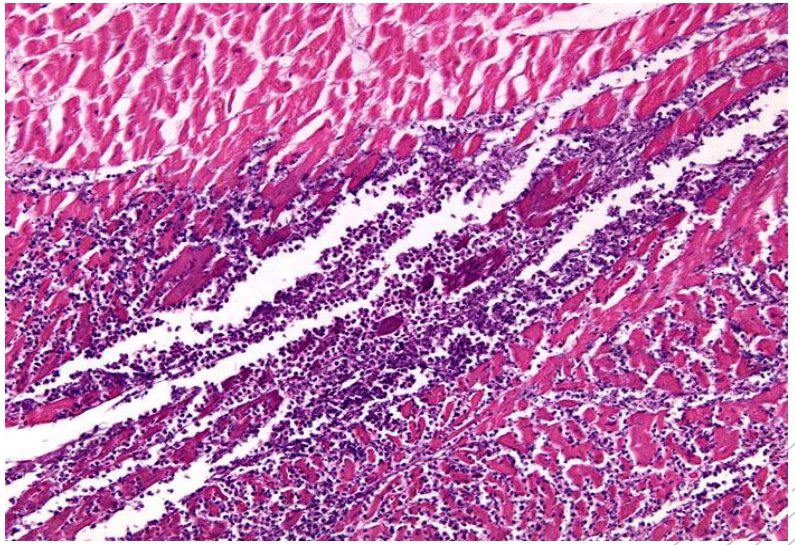
ME

Immediately:

No apparent changes

By 12-72 hours:

coagulative necrosis, inflammatory cells.



Ischemic heart disease (IHD),

Myocardial infarction

Morphology

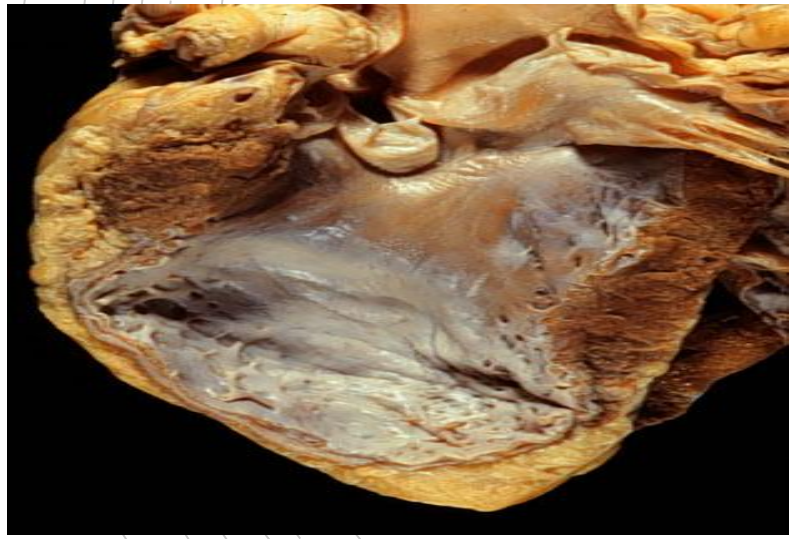
NE

By 3-7 days:

Hyperemic

Late:

White scar



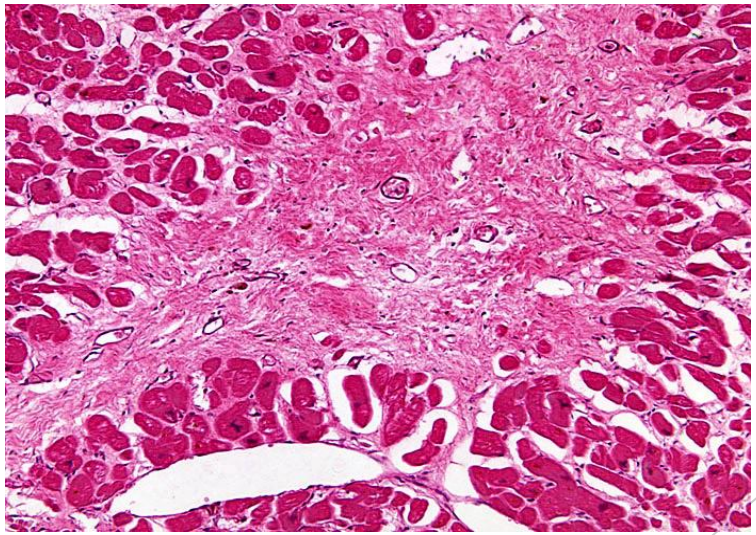
ME

By 3-7 days:

Granulation tissue.

Late:

Fibrosis



Ischemic heart disease (IHD),

Myocardial infarction

Complications:

1.Arrhythmia…..most common cause of death in the first few hours following infarction

2. Myocardial (pump) failure

3. Myocardial Rupture : Occurs within 4-7 days after Infarction (due to neutrophilic liquifactive enzymes).

4. Myocardial aneurysm with thrombosis inside.

5. leg thrombosis &pulmonary embolism due to Prolonged confinement to bed

6. Chronic heart failure

Ischemic heart disease (IHD),

Chronic IHD with heart failure

- Progressive heart failure as a result of ischemic myocardial damage.
- Called ischemic cardiomyopathy
- In most cases, there has been prior MI and sometimes previous coronary arterial bypass graft surgery or other interventions.

ENDOCARDITIS

Definition: Inflammation of the endocardium.

It may be:

a- Non-infective: as in

(a) Rheumatic fever

(b) Systemic lupus erythematosus

(c) Marantic (Terminal) endocarditis: seen in mitral and aortic valves of patients died from wasting diseases.

b- Infective: due to a wide variety of organisms e.g. T.B, syphilis, typhoid, viruses, fungus and histoplasma capsulatum especially in drug addicts.

The most common clinically seen form is infective bacterial endocarditis.

ENDOCARDITIS

Infective Bacterial Endocarditis

It is characterized by colonization or invasion of the heart valves or the mural endocardium by a microbe, leading to the formation of bulky, friable vegetations, often associated with destruction of the underlying cardiac tissues. It may be acute or subacute

	Acute bacterial endocarditis	Subacute bacterial endocarditis.
Nature	Suppurative inflammation caused by virulent organisms	Subacute inflammation caused by less virulent organisms.
Type of organism	Mostly staphylococcus. Aureus and streptococcal pyogens	Mostly streptococcus viridans (commensals in the body).
State of valve and predisposing factor	Normal valves a- Septic focus e.g.septicemia and pyemias. b- Intravenous infections in drug addicts. c- Impaired defense mechanisms.	Abnormal valves a- Abnormal valves due to rheumatic fever, syphilis and congenital abnormalities b- Bacteremia following tooth extraction, tonsillectomy, instrumentation of the GIT as during endoscopy. c- Impaired defense mechanisms.

ENDOCARDITIS

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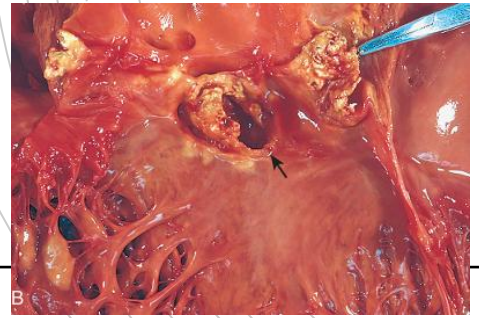
ENDOCARDITIS
Infective Bacterial Endocarditis

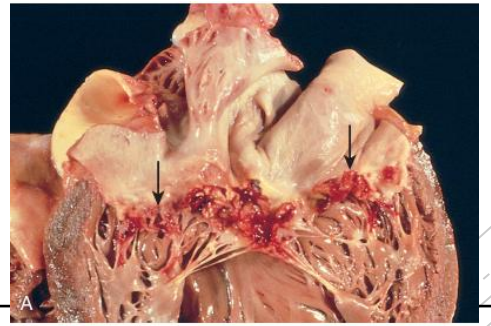
	Acute bacterial endocarditis	Subacute bacterial endocarditis.
NE	a- Mitral and aortic **valves** commonly affected and tricuspid in drug addicts only. b- The valve **cusps** show acute suppurative inflammation with progressive valve destruction that may lead to valve perforation c- **Vegetations** cover the valve cusps and are large, polypoid, irregular yellow and friable. d- **Myocardium** shows abscesses	a- Mitral and aortic **valves** together or separately b- The valve **cusps** shows edema and hyperemia c- **Vegetations** cover the cusps and are irregular, polypoid and **smaller** than those of acute. The color vary from yellowish grey to brown they are friable d- **Myocardium** shows cloudy swelling and fatty changes.



Vegetations

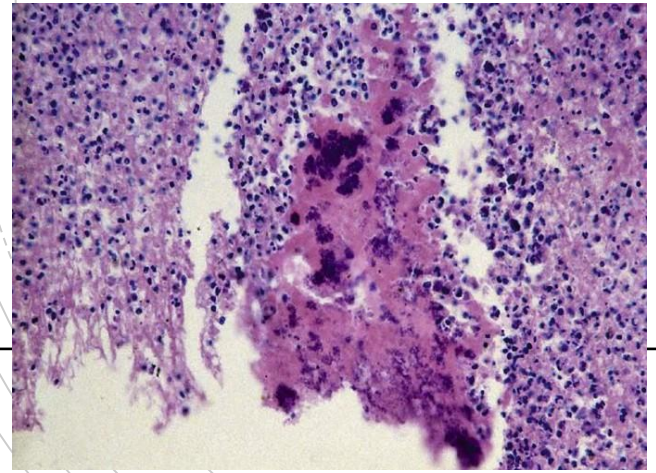
Acute

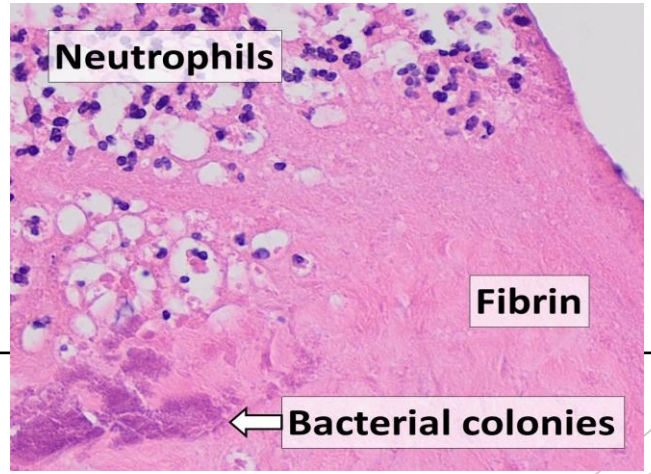
Subacute endocarditis



ENDOCARDITIS
Infective Bacterial Endocarditis

	Acute bacterial endocarditis	Subacute bacterial endocarditis.
ME	a- The **cusps** show picture of acute suppurative inflammation with excess neutrophils and necrosis. b- **Vegetations** are formed of platelets, fibrin, microorganisms and neutrophils	a- The **cusps** are vascularized and show edema, and inflammatory cells. b- **Vegetations** are formed of fibrin, platelets and microorganism with no or few neutrophils.





ENDOCARDITIS

Infective Bacterial Endocarditis

	Acute bacterial endocarditis	Subacute bacterial endocarditis
Effects and associated lesion	a- Septic emboli are formed by breakdown of vegetations causing pyemia. b- Toxemia leading to bone marrow depression fatty change and necrosis of parencymatous organs.	a- Emboli from break down of vegetations produce systemic infarctions but not pyemia. b- Toxemia: leading to clubbing of fingers, fatty change, bone marrow depression and sub acute combined degeneration of spinal cord. c- capillary lesions leads to: (1) Petechial heamorrhage in skin, mucous membranes and serous sacs. (2) Focal or diffuse glomerulonephritis.
Course and prognosis	Untreated cases died within 8 weeks from toxic myocarditis and acute heart failure.	Untreated cases died within 6 months to 2 years from cardiac failure, renal failure or embolic effects.

CARDIOMYOPATHY

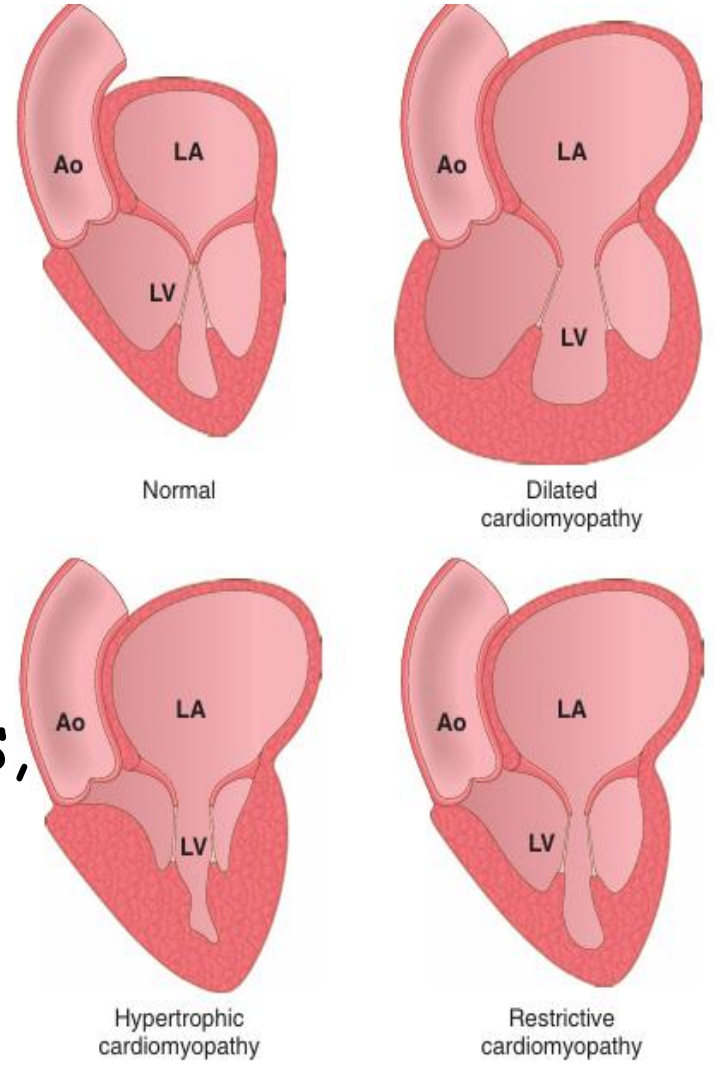
Definition: Is a principal cardiac dysfunction which may be:
primary, of unknown causes or may be secondary.

A) Primary

1) Hypertrophic cardiomyopathy
2) Dilated cardiomyopathy
3) Restrictive cardiomyopathy

B) Secondary to:

Diabetes Mellitus , amyloidosis, hemochromatosis, muscle dystrophy, drugs, thyroid disease and mitochondrial cytopathy.



CARDIOMYOPATHY

Hypertrophic Cardiomyopathy(HCM)

Definition: Genetic heart disease characterized by hypertrophy and rigidity that occur in all cardiac chambers especially in left ventricle, which interferes with diastolic filling

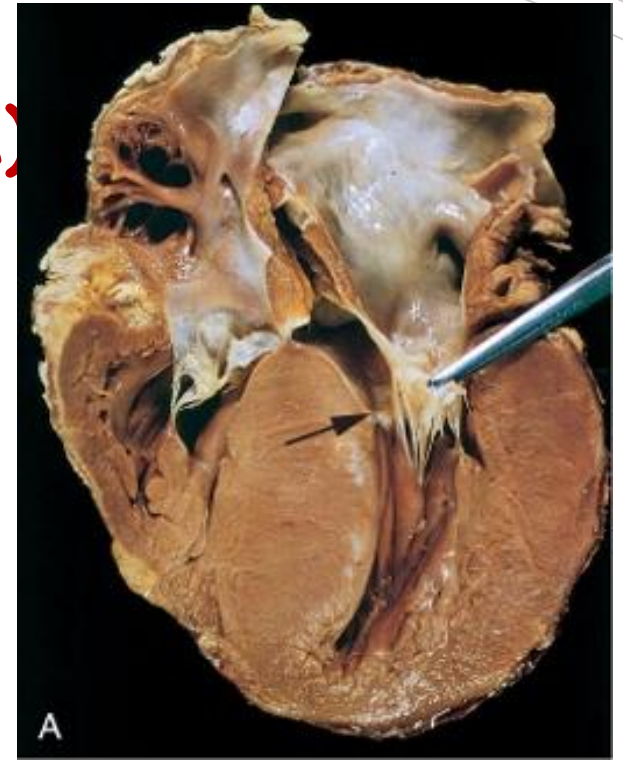
Aetiology: mutations in sarcomeric proteins, most common is B-myosin heavy chain mutation.

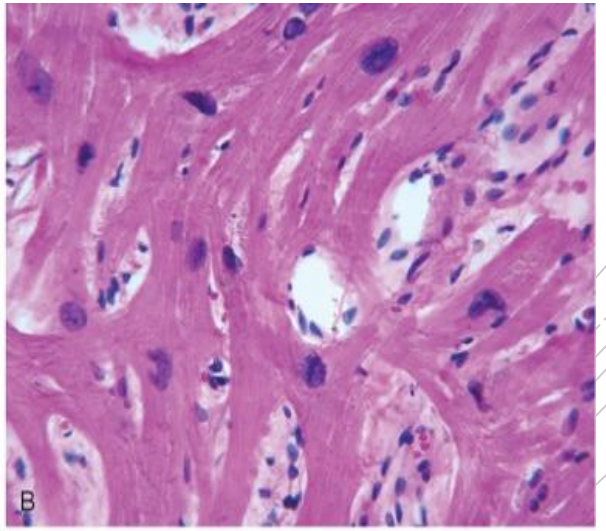
NE: Concentric hypertrophy and asymmetric septal hypertrophy, with narrow banana like left ventricle cavity

ME: marked myocyte hypertrophy with disorganized myofilament arrangement within the myocyte.

Complications: May lead to CHF, SCD.

HCM is one of the most common causes of unexplained sudden death in young athletes.





CARDIOMYOPATHY

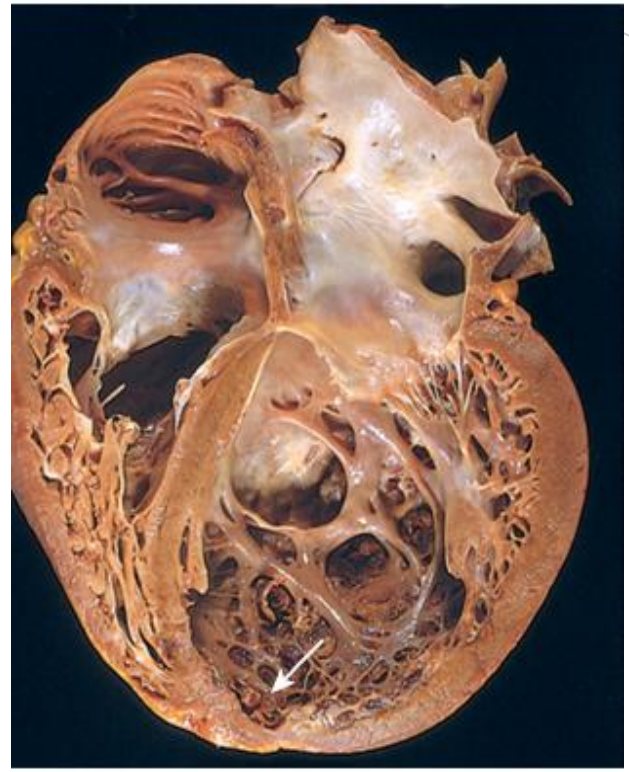
Dilated Cardiomyopathy

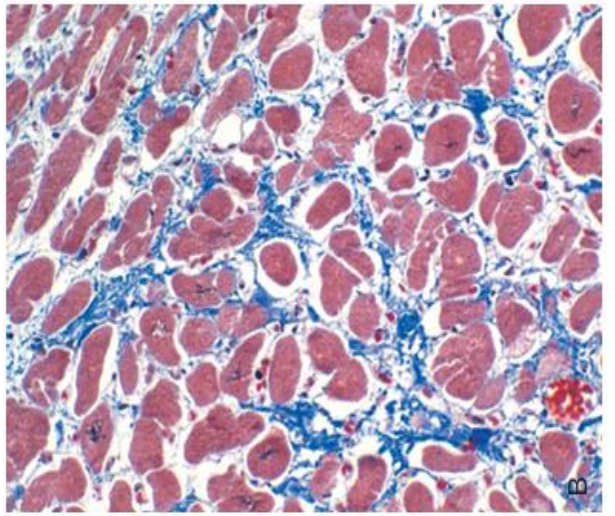
Definition :Gradual four chamber hypertrophy and dilation result in Systolic dysfunction with hypocontraction and progressive CHF,

Causes include:

1. Genetic abnormalities involving cytoskeletal proteins e.g. dystrophin in X-linked cardiomyopathy
2. Myocarditis
3. Alcohol and other toxins
4. Superphysiologic stress as persistent tachycardia or hyperthyroidism
5. Peripartum: months around delivery
6. Iron over load as in heamochromatosis

ME: diffuse myocyte hypertrophy and interstitial fibrosis





CARDIOMYOPATHY

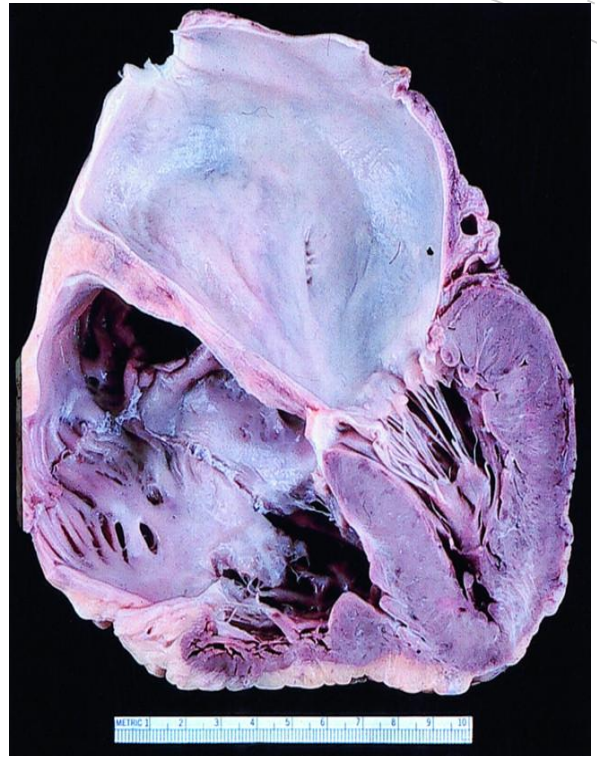
Restrictive Cardiomyopathy

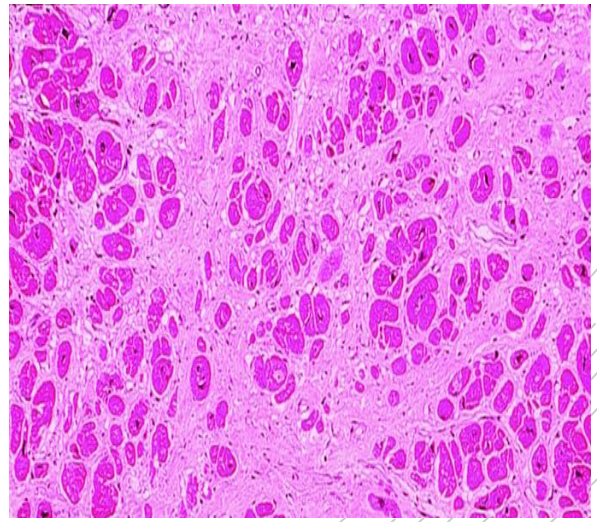
Definition: Characterized by restriction of ventricular filling leading to reduced cardiac output and heart failure
Contractility normal and ventricle size is normal with dilation of both atria
Most common in patients ≤ 2 years
ME: one of the following may be seen

A-Endomyocardial fibrosis:
ventricular sub-endocardial fibrosis

B-Loeffler endomyocarditis: associated with peripheral eosinophilia and eosinophilic infiltrate in many organs specially the heart

C-Endocardial fibroelastosis (in children): focal or diffuse fibroelastic thickening of endocardium.





PERICARDITIS

Definition: Inflammation of pericardium

Etiology

Infective

a) Bacteria: pyogenic cocci from distant focus, T.B. and syphilis.
b) Viruses as Coxackie B.
c) Actinomycosis.
d) Parasitic as amoeba.

Non-infective

As in trauma,
Rheumatic fever,
SLE,
Myocardial infarct,
Uremia,
Tumors or even unknown.

PERICARDITIS

Pathology: pathologically the pericarditis may be:

1)Sero-fibrinous: In all types of pericarditis except in cases of pyogenic infection and T.B.

Complications: adherent pericarditis or mediastino-pericarditis.

2) Suppurative: In cases of pyogenic infection.

N/E: pericardium filled with pus.

M/E: suppurative inflammation.

Complications: a) Adherent pericarditis or mediastino-pericarditis.
b) Constrictive pericarditis.

3) Tuberculous: as in T.B

Complications: a) Adherent pericarditis or mediastino-pericarditis.
b) Constrictive pericarditis.

4) Constrictive: either idiopathic or as a complication of other types especially suppurative and T.B.

Complications: -)congestive heart failure

Rheumatic fever, Def., Etiology

Def.: Diffuse collagen disease affecting heart, joints and subcutaneous tissue.

- 90% of heart diseases in children and about 40% in adults.

Predisposing factors:

- Hereditary predisposition
- Cold climate, low socio-economic standards, overcrowding and malnutrition ⟶ recurrent streptococcal pyogens infections as tonsillitis

Exciting cause: abnormal immune reaction.

Rheumatic fever, Pathogenesis

- Infection by Group A β hemolytic streptococci ⟶ stimulate immune system with production of antibodies.
- Streptococcal antigen is similar to autoantigen in heart and other tissues (antigenic mimicry).
- The antibodies + complement against strept. **cross react** with the similar autoantigens.
- The immune reaction causes **damage of autoantigens** with formation of **allergic granuloma** called "Aschoff's body".

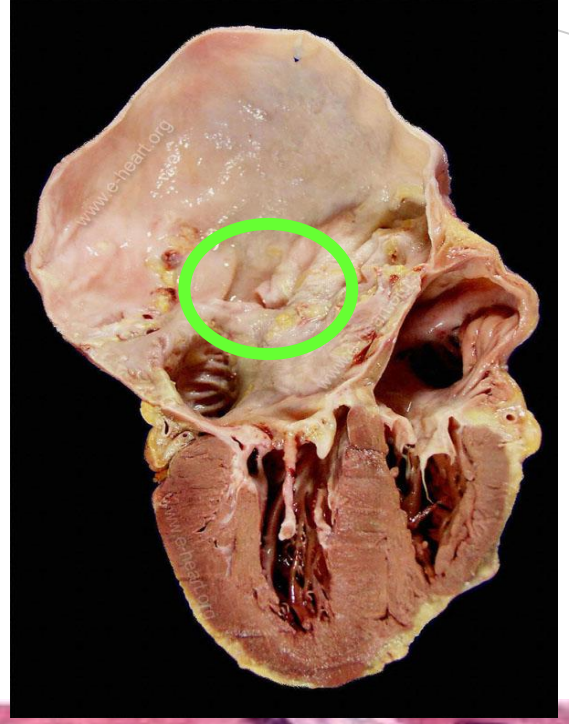
Rheumatic fever, Tissue reaction

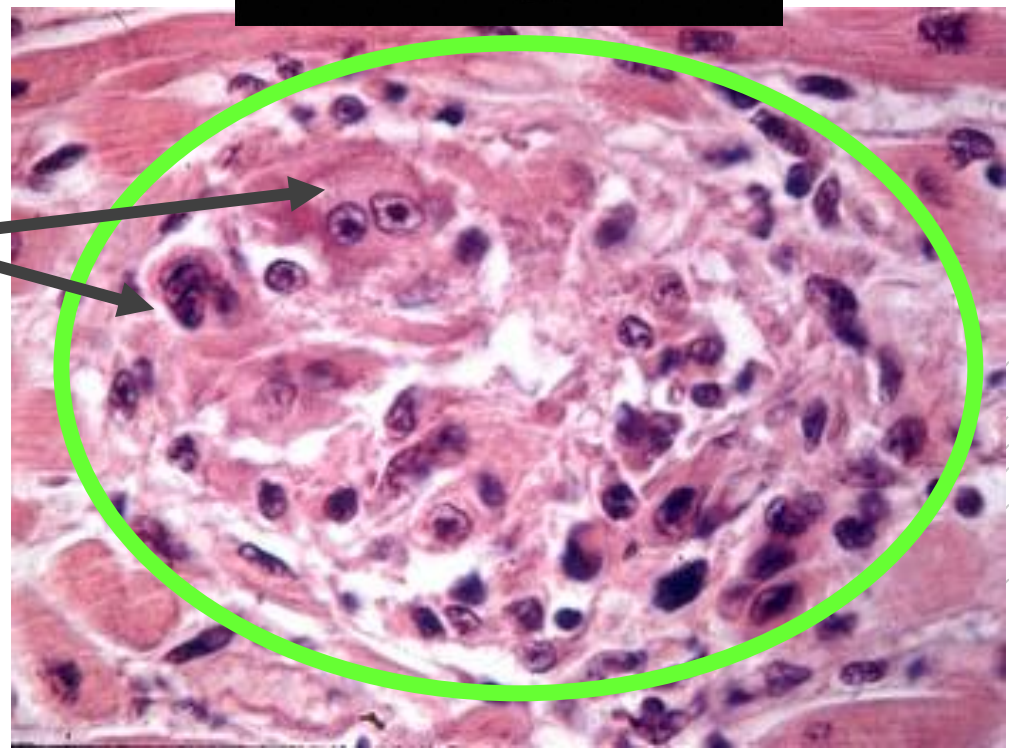
N/E: Small pale nodules seen in heart, joints, S.C. and sometimes C.N.S.

M/E:

- Central area of fibrinoid necrosis.
- Collection of chronic inflammatory cells (lymphocytes &macrophages) with Aschoff giant cells.
- Thick-walled blood vessels

Fate: Fibrosis and calcification.



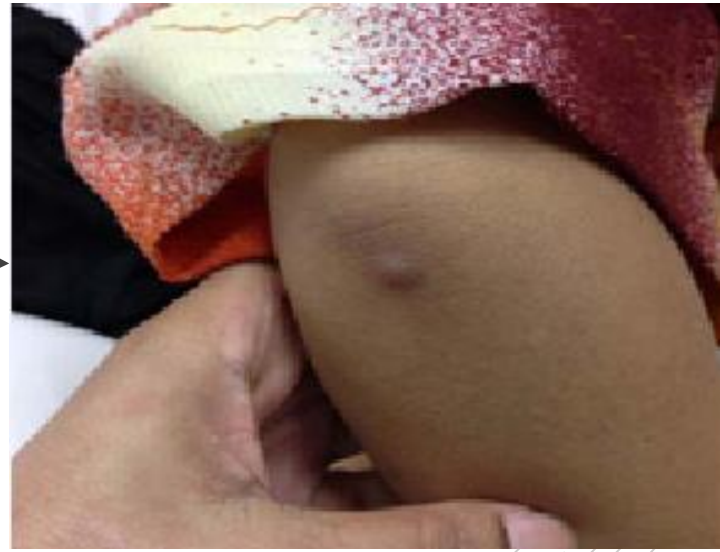


Rheumatic fever, Pathology

I- Rheumatic Pancarditis
  1- Rheumatic Endocarditis
  2- Rheumatic Myocarditis
  3- Rheumatic pericarditis.

II- Rheumatic polyarthritis

III- Rheumatic subcutaneous nodules



IV- Rheumatic chorea (CNS affection)

Rheumatic fever, Endocarditis

1. Valvular endocarditis:

Early

*Valve cusps are edematous, red, and opaque.

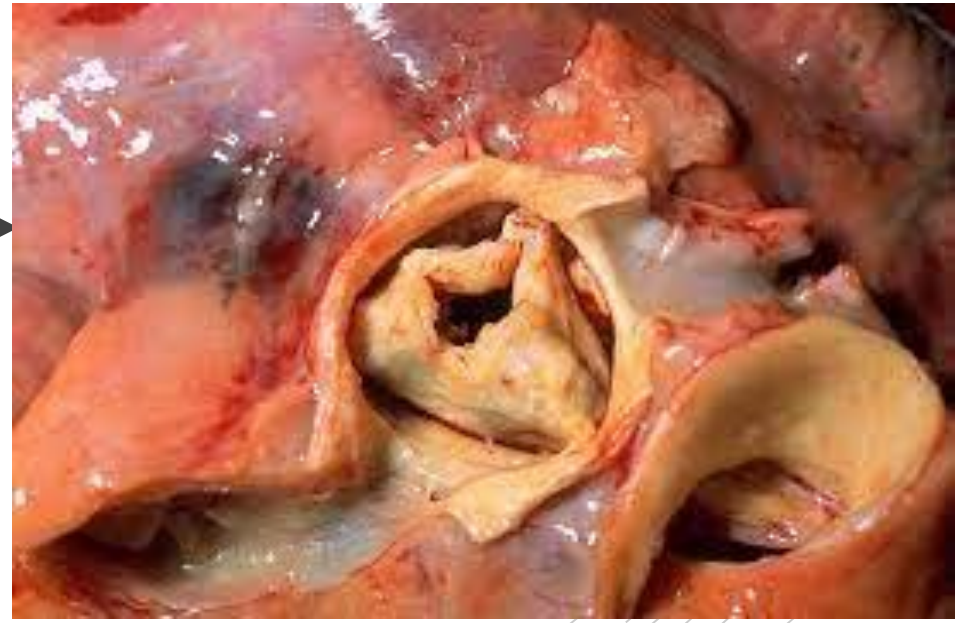
*Vegetations, free margin of cusps on the atrial surface, small, beaded, pale and adherent

Late

Fibrosed, calcified, thick cusps.

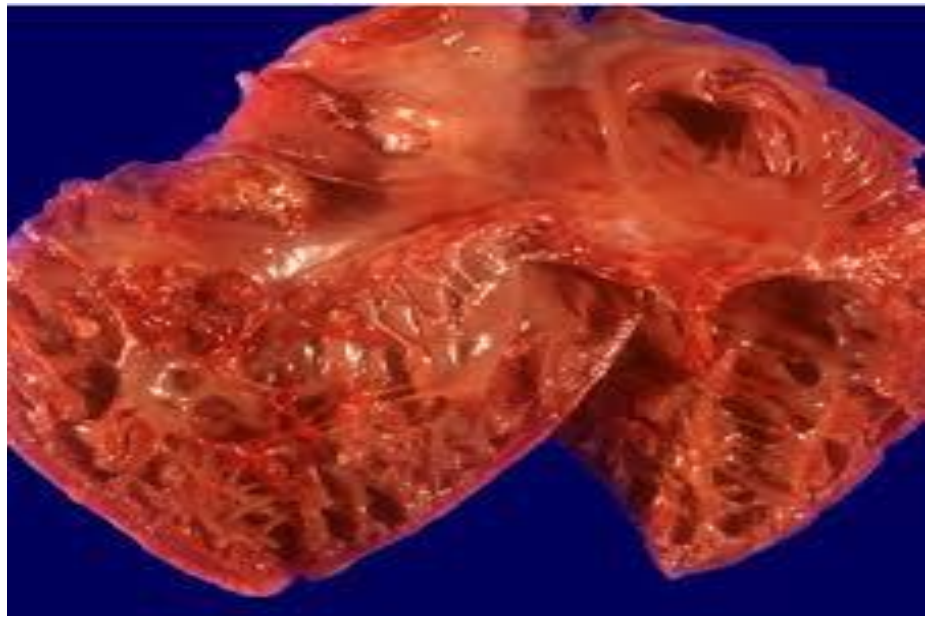
a) Stenosis b) Incompetence

Mitral stenosis is the commonest



Rheumatic fever, Endocarditis

2. Mural endocarditis: It affects mainly the posterior wall of left atrium above the mitral valve called Mac Callum's patch .

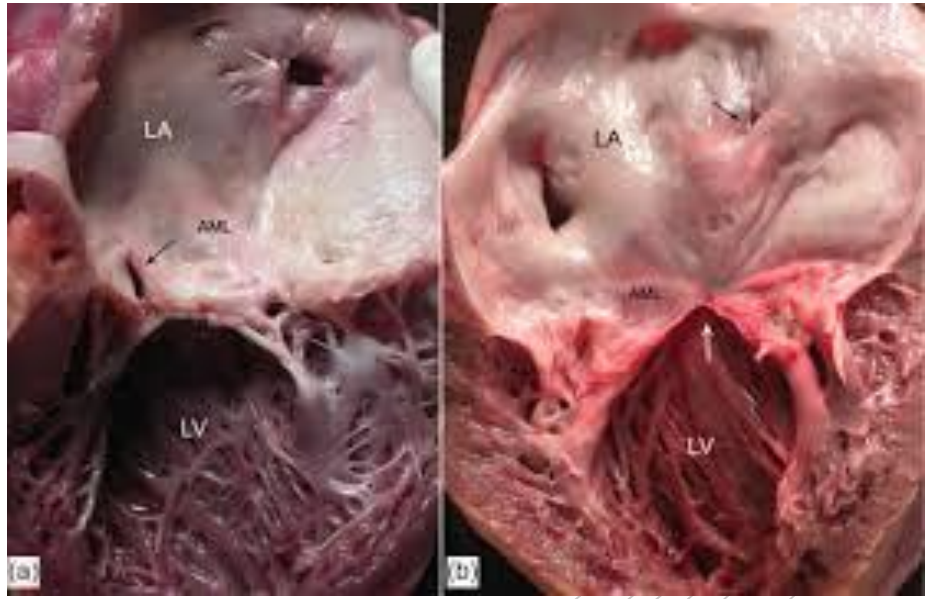


Early

The characteristic rheumatic reaction is seen with Aschoff's bodies.

Late

Later on fibrosed (white, roughened patch commonly complicated by thrombosis).

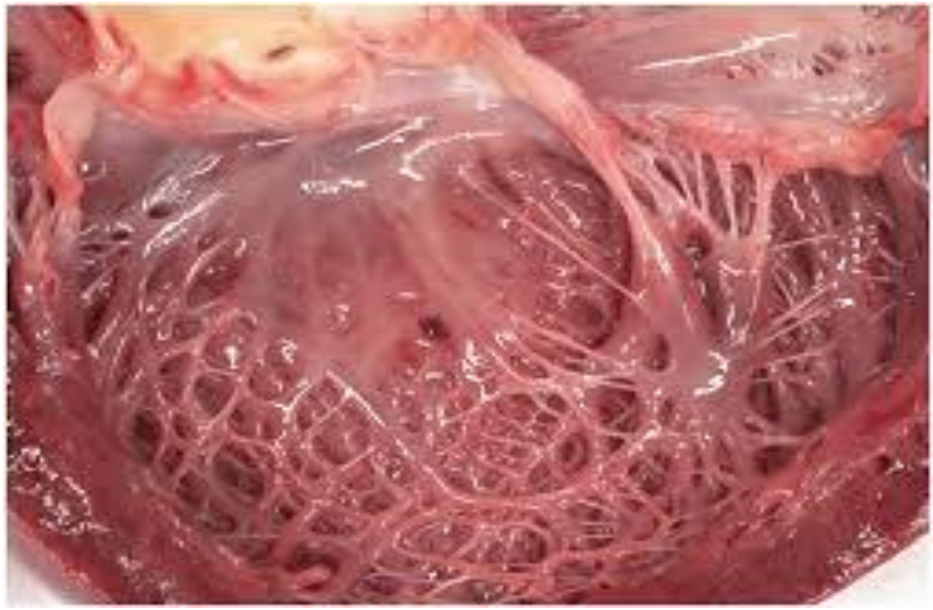


Rheumatic fever, Myocarditis

left atrium is the most affected.

N/E:

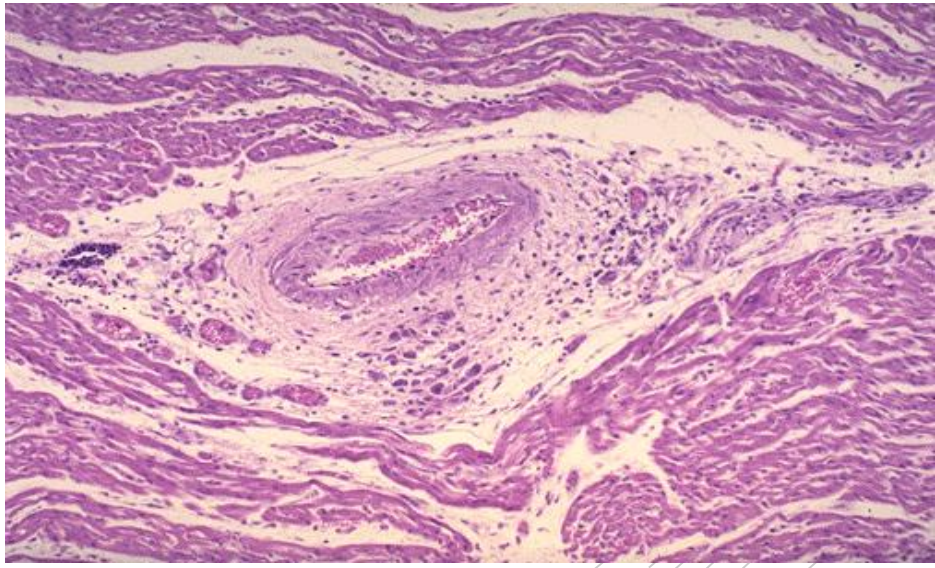
The muscle is swollen, flabby and the chambers are dilated. Aschoff's bodies may be seen as scattered pale foci.



M/E:

Cloudy swelling or necrosis of myocardium & Aschoff's bodies are seen commonly on the endocardial side.

The lesions heal by fibrosis.

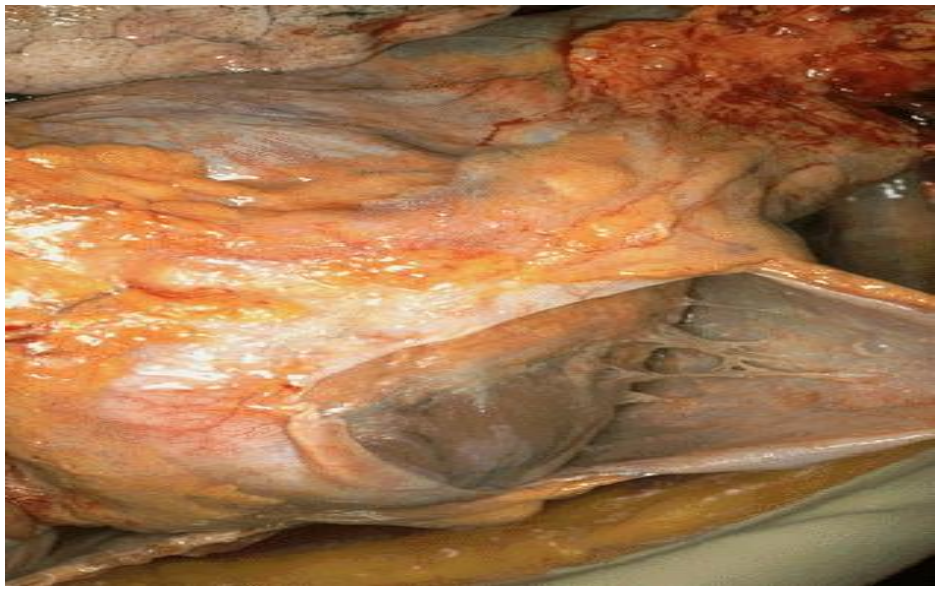


Rheumatic fever, Pericarditis

Sero-fibrinous pericarditis mainly at the heart base.

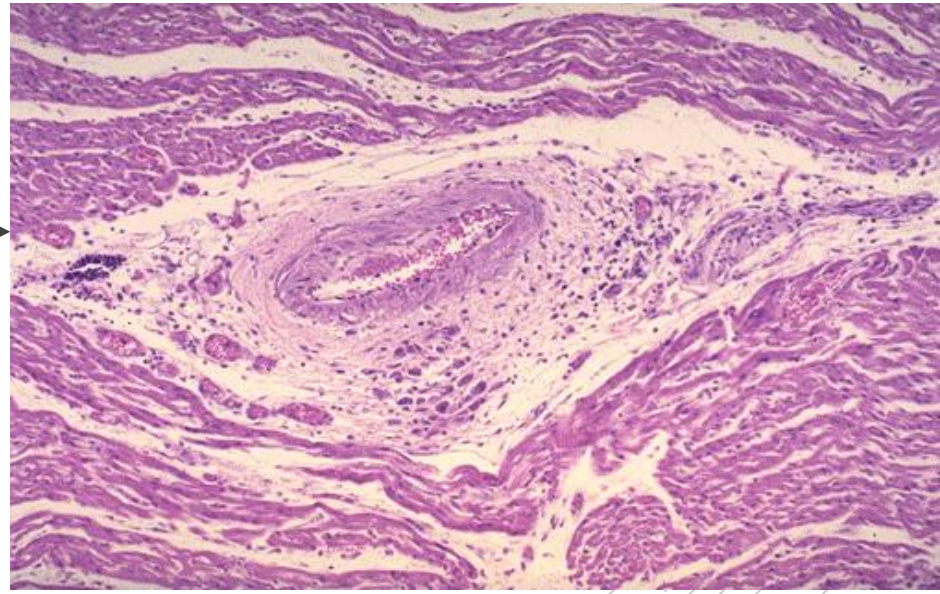
N/E:

The pericardial sac is filled with serous fluid and fibrin is deposited on both visceral and parietal pericardium. Separation of which produces bread and butter appearance.



M/E:

Sero-fibrinous inflammation + Aschoffs' bodies




Thank you