SMART VISION INSTITUTE

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PATHOLOGY

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C.V.S

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Structure of blood vessels

♣ Vessel walls are formed of three concentric layers: intima, media and adventitia.

- Arteries:

Arteries are divided into three types:

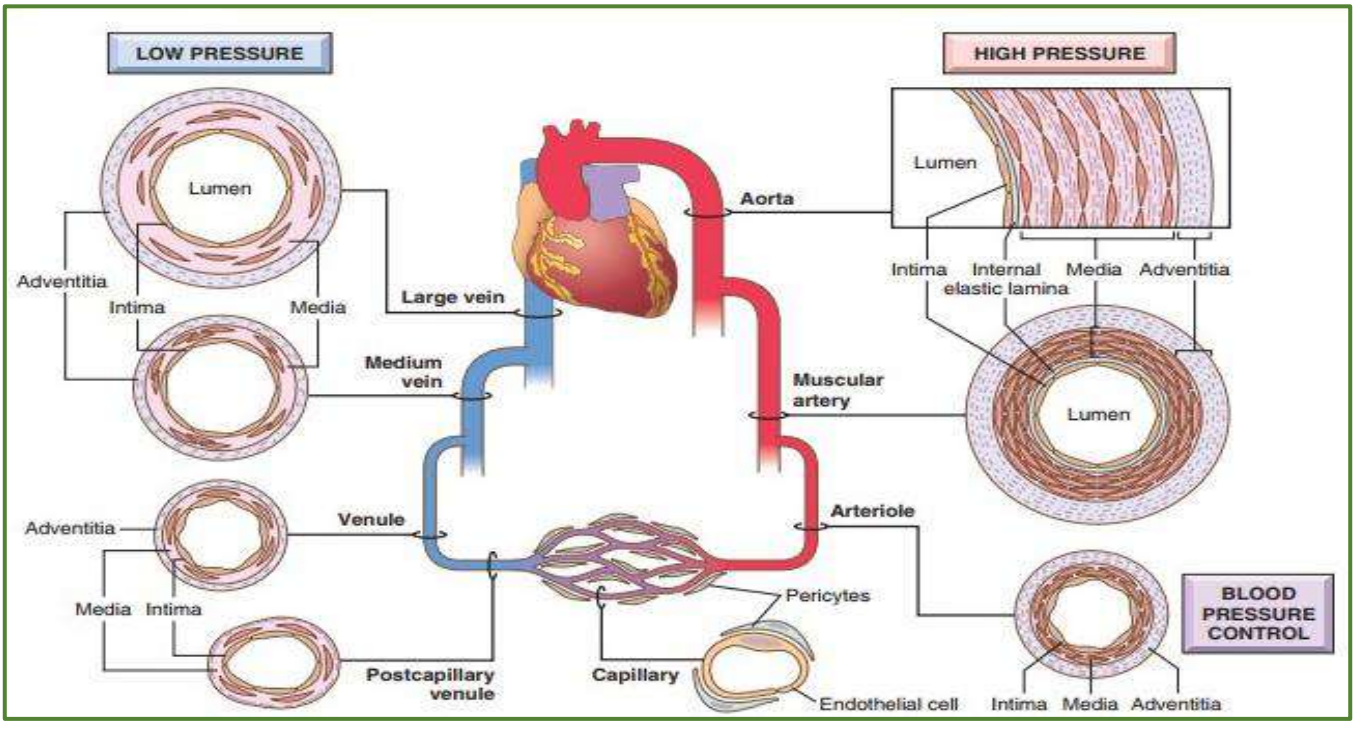
Large elastic arteries	Medium-sized muscular arteries	Small arteries & arterioles
aorta, aortic arch vessels, Iliac and pulmonary arteries	(coronary, renal)	within the connective tissue of organs. (2mm in diameter),
In the media: elastic fibers alternate with smooth muscle cells . in the adventitia: Small arterioles (vasa vasorum) supply outer2/3 of the media. Inner 1/3 receives O_2 and nutrients by diffusion from the vessel lumen.	Elastic fibers are arranged in an internal and external elastic lamina.	Their media is mostly formed of smooth muscles. Arterioles regulate the blood flow resistance.

- Capillaries:

Lined by endothelial cells surrounded by pericytes (smooth muscle like)

- Veins:

have larger diameter, wider lumen, thinner wall, with less distinct layers.



HYPERTENSION

♣ Definition: It is the persistent elevation of resting blood pressure above 140/90

♣ Types of hypertension:

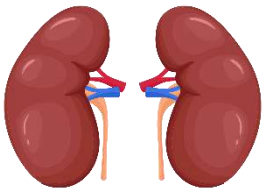
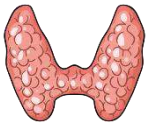
Etiological classification	Clinico-pathological classification
✓ Primary (essential HTN) 95% Without obvious cause. ✓ Secondary HTN	✓ Benign HTN: blood pressure rises gradual& moderate. ✓ Malignant HTN (5%): The blood pressure rises rapidly. The BP usually exceed 200 / 120 It may start de novo or It may appear suddenly in a person with benign hypertension (accelerated HTN)

♣ Etiology and pathogenesis of essential HTN:

➢ The blood pressure is a factor of total peripheral resistance and cardiac output.

- ✓ ↓↓↓ renal sodium excretion → ↑↑↑ blood volume with ↑↑↑ cardiac output.
- ✓ ↑↑↑ vascular resistance due to hypersensitivity of blood vessels to circulating catecholamines.
- ✓ Genetic factors There is familial clustering of hypertension, may be linked to specific variation of angiotensinogen and angiotensin II receptors.
- ✓ Environmental factors as
  - stress,
  - obesity, and
  - excess salt intake.

♣ Causes of secondary HTN: (REV)

Renal	»Renal artery stenosis » Polycystic kidney »Chronic pyelonephritis » Acute and chronic glomerulonephritis 
Endocrine	» Hyperthyroidism. » Exogenous corticosteroids  » Adrenocortical hyper function: 1st hyperaldosteronism, Cushing $ » Pheochromocytoma (adrenal medulla tumor ↑↑ catechol amines).
Vascular	Coarctation of aorta (upper half of the body)

♣ The mechanism of renal hypertension:

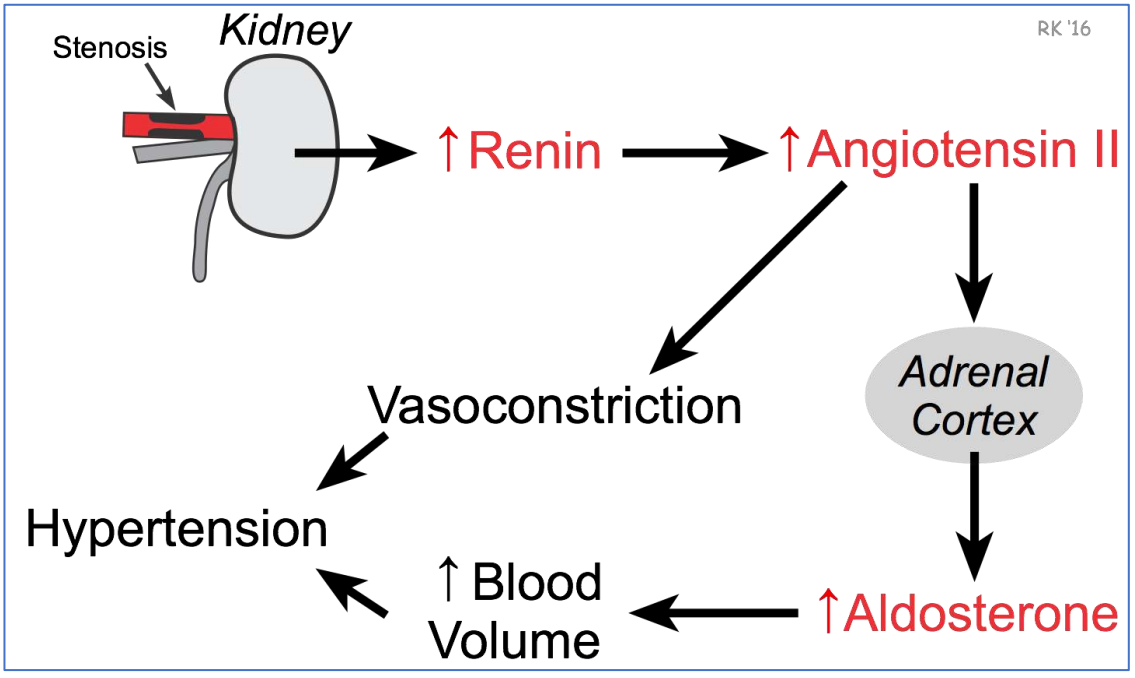
✓ ↑↑↑ renin secretion by the juxtaglomerular cells of the kidney→→

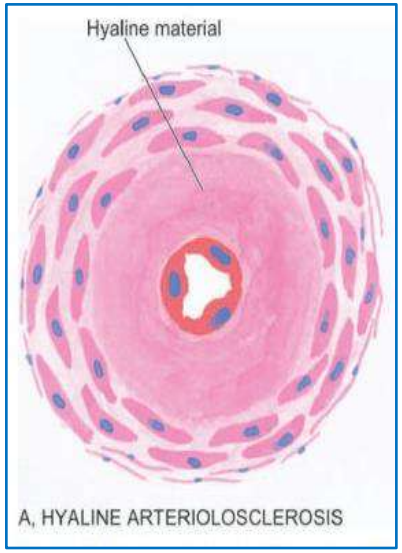
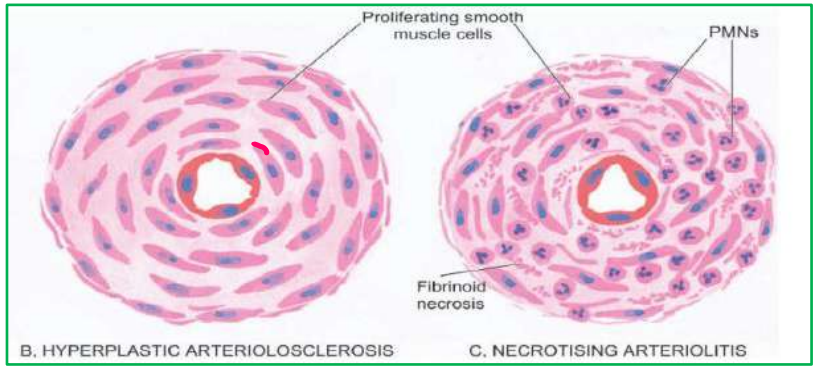
Convert plasma angiotensinogen →→ angiotensin →→ angiotensinII

(By angiotensin-converting enzyme.) ACE

✓ AngiotensinII →→ ↑↑↑ blood pressure by increasing both:-

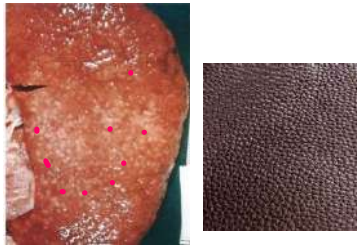
peripheral resistance(vasoconstrictor) & blood volume →→ ↑↑↑aldosterone secretion→→ ↑↑↑distal tubular reabsorption of sodium & ↑↑↑blood volume.



	BENIGN Hypertension	MALIGNANT Hypertension
Blood vessels	♣ Hyaline arteriolosclerosis: ✓ Affect: small arteries and arterioles. ✓ It is due to: endothelial injury by : hemodynamic stress & leakage of plasma components into the intima ✓ Micro: Homogeneous pink hyaline thickening of the intima & media narrowing of the lumen.  A, HYALINE ARTERIOLOSCLEROSIS ♣ Fibroblastic hyperplasia (elastosis):- ✓ Affects : larger arteries ✓ Micro : duplication of internal elastic lamina & thickened media.	♣ Fibrinoid necrosis: ✓ Affect: small arteries and arterioles . ✓ micro: (granular eosinophilic material in vessel wall) ♣ Hyperplastic arteriolosclerosis: ✓ Micro: onion-skin appearance of vessels due to hyperplasia of smooth muscle cells and connective tissue.  B. HYPERPLASTIC ARTERIOLOSCLEROSIS C. NECROTISING ARTERIOLITIS
Heart	♣ Concentric hypertrophy of the left ventricle ♣ ischemic heart disease and angina pectoris: due to Acceleration of atherosclerosis & increase O_2 demand due to muscle hypertrophy	♣ The heart is not markedly hypertrophied : due to short duration.

<table>
<tr>
<td>Kidney</td>
<td>
♣ Benign nephrosclerosis (primary contracted kidney):

✓ Gross:
➢ kidneys are symmetrically atrophic.
➢ surface shows diffuse fine granularity (resembling leather grain).



✓ Microscopic:
➢ Hyaline arteriolosclerosis of afferent arterioles [sclerosis of glomeruli ,diffuse tubular atrophy & fibrosis.]
➢ fibroblastic hyperplasia of large arteries.

✓ Effect:
decrease in GFR mild degree of proteinuria may be present.
</td>
<td>
♣ Malignant nephrosclerosis:

✓ Gross:
➢ The kidney is usually normal in size, or shrunken (in case of accelerated HTN).
➢ The outer surface shows pin-point hemorrhages (Flea- bitten kidney)

✓ Microscopic:
➢ Arterioles show fibrinoid necrosis.
➢ Larger arteries show hyperplastic arteriolosclerosis
➢ Necrosis may also involve the glomeruli with microthrombi.
</td>
</tr>
<tr>
<td>Retina</td>
<td colspan="2">Retinal hemorrhages and exudates, and papilledoema (edema of optic disc, leading to blurring of vision)</td>
</tr>
<tr>
<td>C/P</td>
<td>
1) Heart failure
2) Cerebral hemorrhage
3) Chronic renal failure (rare)
</td>
<td>
1) Acute renal failure
2) Cerebral hemorrhage
3) Heart failure (rarely)
</td>
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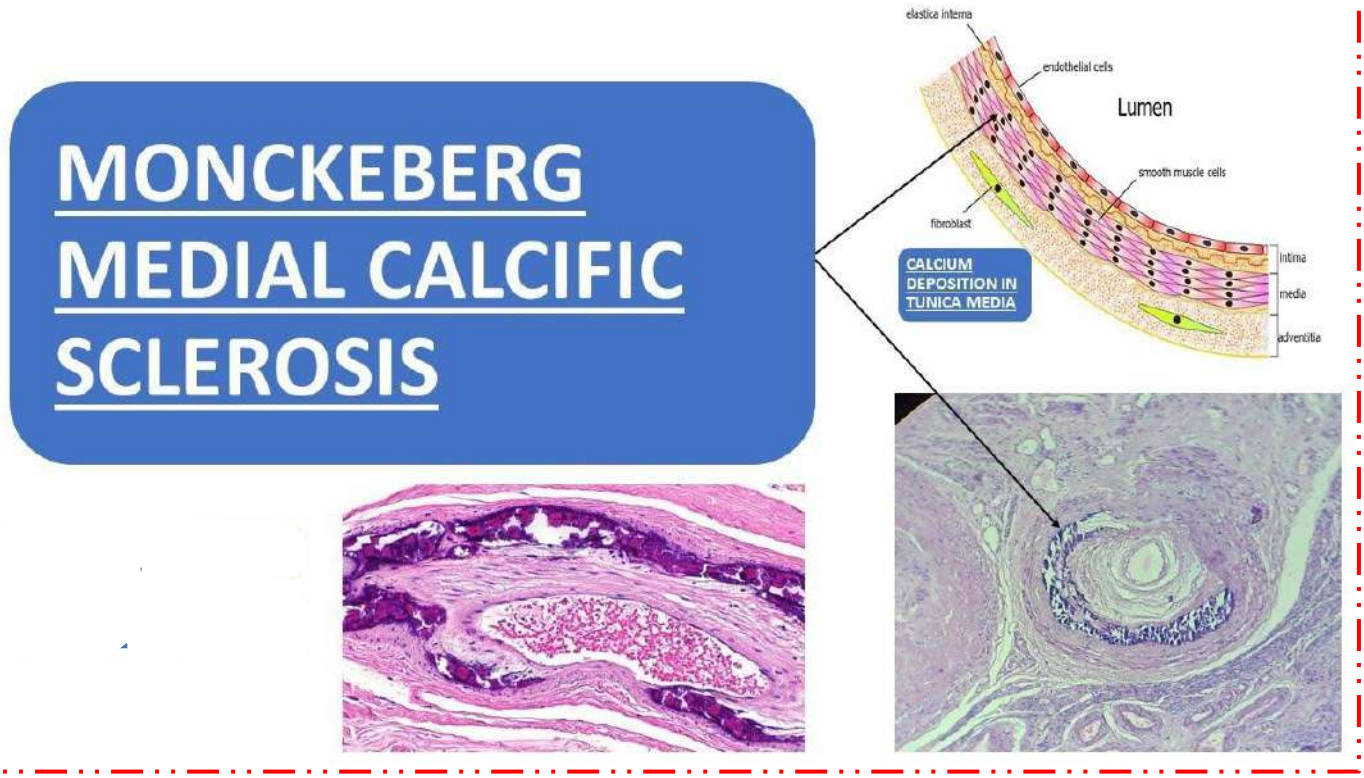
</table>

ARTERIOSCLEROSIS

♣ Definition: Arteriosclerosis means hardening of the arteries.

♣ Types: Three distinct types are recognized:

Arteriolosclerosis	Monckeberg calcific medial sclerosis	Atherosclerosis
✓ Affects: small arteries + arterioles in Hypertension. ✓ Include : a) Hyaline arteriolosclerosis b) Hyperplastic arteriolosclerosis	✓ Calcific deposits in muscular arteries in patients > 50 years. ✓ The lesion do not encroach on vascular lumen SO no clinical significance.	✓ DEF: Thickening and hardening of the vessel ✓ Cause: lipid deposition in the subintimal connective tissue.



ATHEROSCLEROSIS

♣ Definition: is a disease of blood vessels characterized by :

patchy intimal thickening as a result of lipid deposition, covered by fibrous cap.

♣ Risk factors:

Major risk factors (4H)	Constitutional risk factors
1. Hyperlipidemia(hypercholesterolemia): ✓ ↑↑↑levels of low density lipoproteins (LDL) (bad cholesterol) which distributes cholesterol to peripheral tissues. ✓ ↓↓↓ levels of high density lipoproteins (HDL) (good cholesterol) which mobilizes cholesterol from developing plaques and transport to the liver to be excreted in bile. N.B: *↑↑ HDL levels. Exercise and omega 3 fatty acids in fish oil . *↑↑ LDL may be hereditary or related to diet [↑↑intake of animal fat or artificial oils]. 2. Hypertension Both systolic and diastolic BP are important. The high pressure causes endothelial injury. 3. Diabetes mellitus --Hyperglycemia hypercholesterolemia. 4. Heavy Smoking.	1) Advanced age: ➢ becomes clinically apparent at 40-60 years . 2) Gender: ➢ Women in child bearing age are relatively protected (by estrogen) but the incidence increases after the menopause. 3) Genetics: ➢ Family history is an important independent risk factor ✓ Hereditary hypercholesterolemia accounts for a small percentage of cases. ✓ Familial risk is due to the fact that the major risk factor (D.M & HTN) are polygenic in nature controlled by multiple genes 4) Lack of exercise. 5) Stressful life style.

♣ Pathogenesis:

Response to injury hypothesis

(interplay of endothelial injury & inflammation):

1- endothelial injury:

- ✓ leads to: ↑↑↑permeability to LDL & accumulates in the intima.
- ✓ Cause: Hyperlipidemia, HTN , smoking and hemodynamic forces.

(atheromatous plaques tend to occur at ostia of vessels, at branching points of vessels where turbulence is high).

2- Platelets & monocytes:

adhere to the endothelium and migrate into the intima and transform to macrophages.

3- release oxygen free radicals:

- ✓ From: endothelial cells as well as monocytes
- ✓ cause: oxidation of soluble LDL to insoluble oxidized LDL.

Insoluble LDL & cholesterol crystals are taken up by macrophages which change to foam cells.

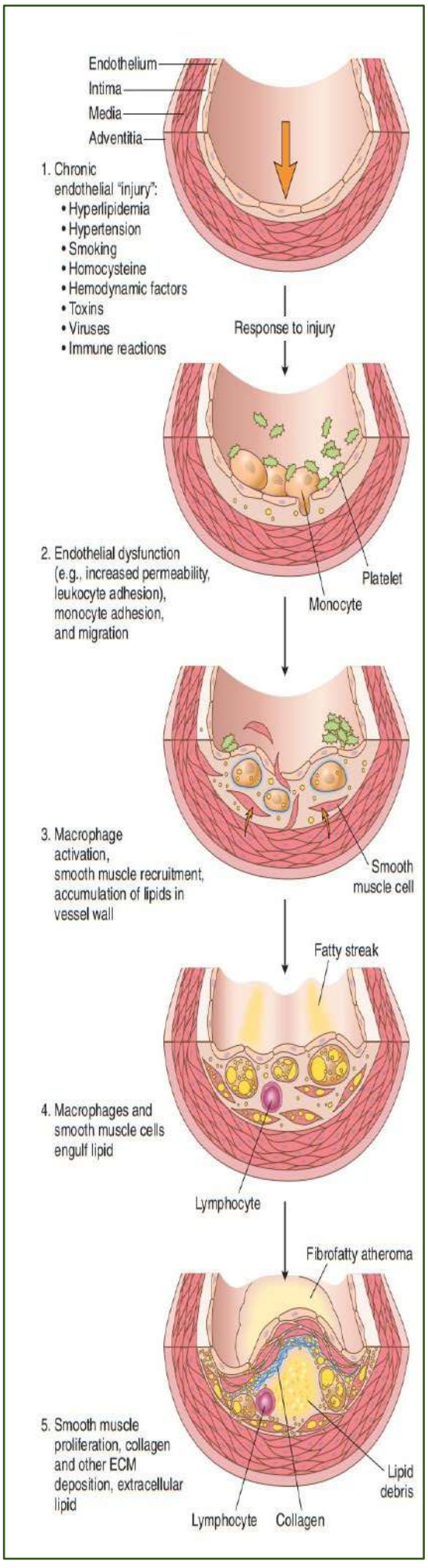
4- Migration & Proliferation of smooth muscle cells:

- ✓ From the media to the intima
- ✓ By : platelet derived growth factors
- ✓ Secrete extracellular matrix (ECM) namely collagen.

5- Recruiting of activated T-cells to intima:

- ✓ Produce: inflammatory cytokines
- ✓ Stimulates: macrophages, endothelial cells and smooth muscle cells

(exact antigen and cause of T cell activation is not clear).



♣ Morphology:

	Fatty Streaks	Atheromatous Plaque
Form of	lipid-filled foamy macrophages	intimal thickening & lipid accumulation
Macro	✓ elongated streaks 1 cm or more in length. N.B: earliest lesions in atherosclerosis as early as age of 10.	✓ White to yellow raised lesions 0.3-1.5 cm in diameter, [but can coalesce to form larger masses.] ✓ Superadded thrombosis →→ red-brown color.
Effect	✓ they are not significantly raised & do not cause any flow disturbance.	✓ affect only part of the circumference of vessels so on cross section the lesion appears eccentric.

♣ Microscopic picture:

✓ Atheromatous plaques consist of:

a central lipid core covered by a fibrous cap of smooth muscle cells and collagen.

✓ The central core:

➢ contains lipids [cholesterol, necrotic debris,

lipid laden macrophages (foam cells, fibrin and other plasma proteins)].

➢ The cholesterol appears as needle shaped clefts

(because the cholesterol dissolves during slide preparation).

✓ The shoulder region:

➢ where the fibrous cap meets the vessel wall is more cellular and contains macrophages, T cells and smooth muscle cells.

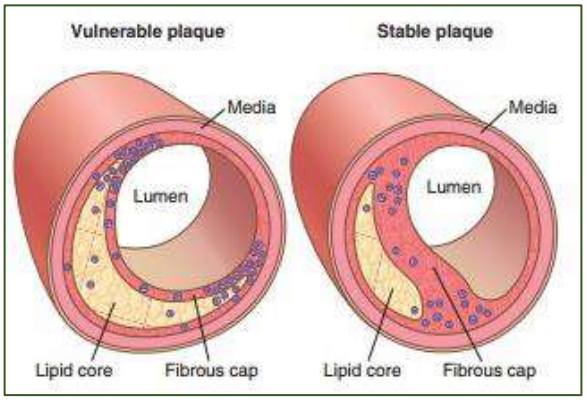
➢ Later it shows neovascularization (proliferated small blood vessels).

✓ Secondary changes:

include ulceration, thrombosis and calcification.

✓ The media opposite the plaques:

are thin due to smooth muscle atrophy and loss.



♣ Sites:

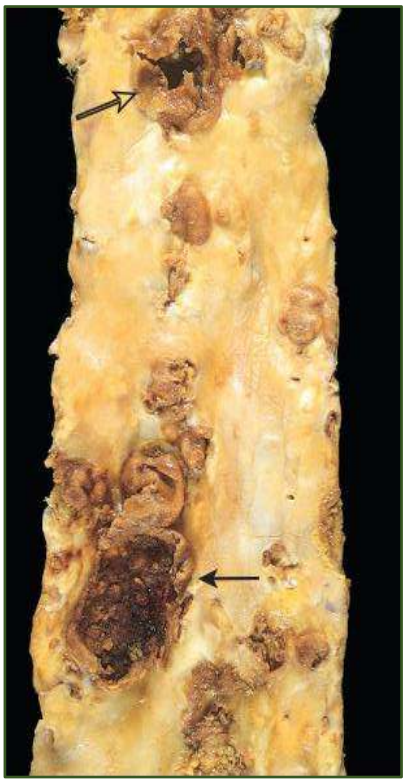
✓ In descending order, the most extensively involved vessels are:

the infrarenal abdominal aorta, the coronary arteries, the popliteal arteries, the internal carotid arteries, and the vessels of the circle of Willis.

✓ Vessels of the upper extremities are usually spared:

as are the mesenteric and renal arteries, except at their ostia .

✓ Severity of atherosclerosis in one artery does not predict its severity in another.



♣ Effect and Complications:

A) In medium sized arteries:

1. Artery stenosis:

➢ leading to diminished tissue perfusion and ischemia e.g:

✓ Cardiac ischemia and angina pectoris.

✓ Lower limb ischemia and intermittent claudication in femoral and popliteal artery lesions.

2. Complete arterial occlusion:

➢ due to rupture of plaque or hemorrhage into the plaque from neo-vascularization, or superadded thrombosis.

B) In Large arteries as aorta:

1. Atheroembolism:

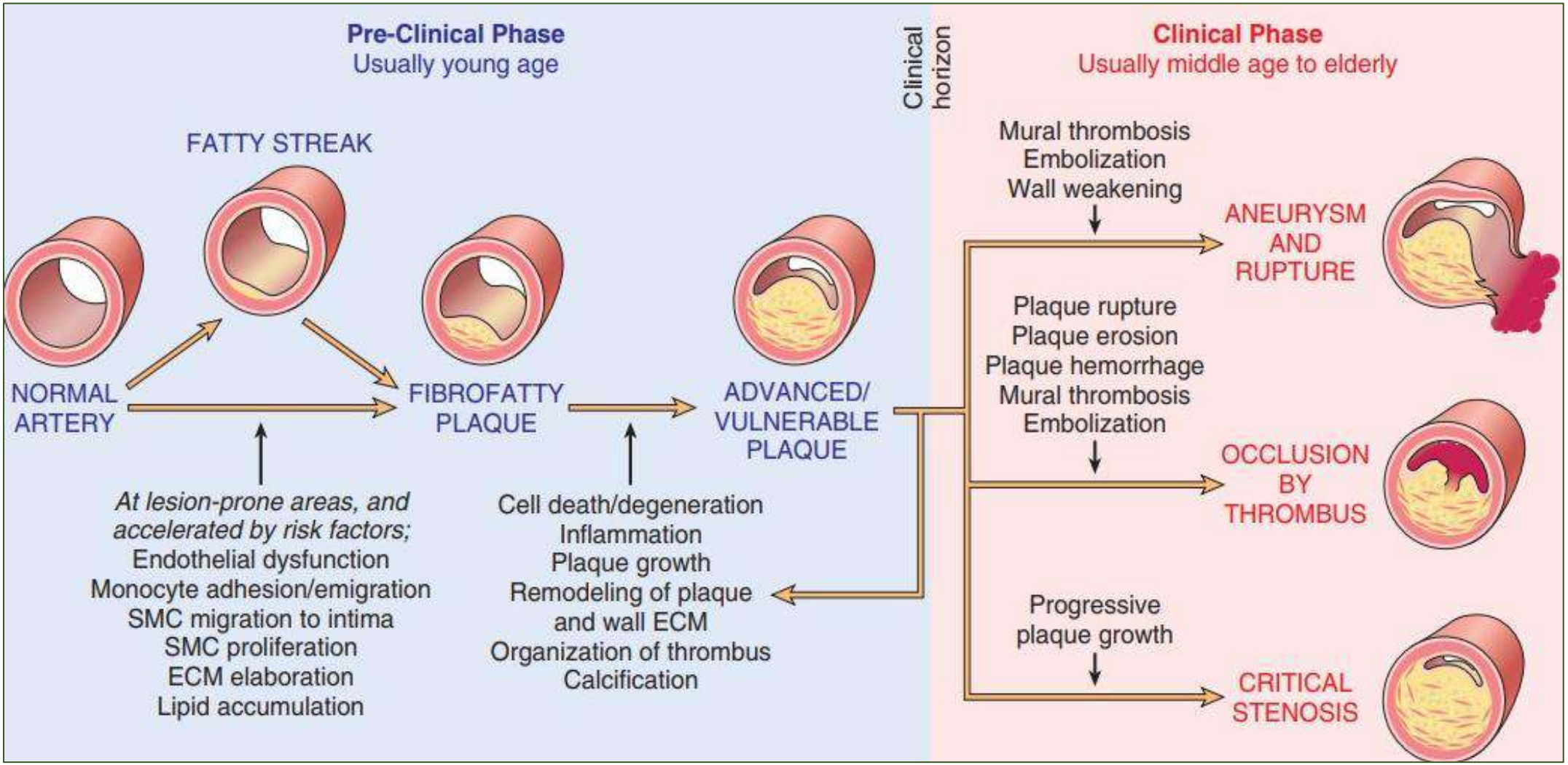
Plaque rupture can discharge atherosclerotic debris into the bloodstream, producing microemboli.

2. Thromboembolism:

Thrombosis on top of atheromatous plaques with embolization.

3. Aneurysm formation:

Pressure atrophy of the media→→ in aneurysmal dilation and potential rupture.


Pre-Clinical Phase
Usually young age
Clinical horizon
Clinical Phase
Usually middle age to elderly
FATTY STREAK
NORMAL ARTERY
FIBROFATTY PLAQUE
ADVANCED/ VULNERABLE PLAQUE
At lesion-prone areas, and accelerated by risk factors;
Endothelial dysfunction
Monocyte adhesion/emigration
SMC migration to intima
SMC proliferation
ECM elaboration
Lipid accumulation
Cell death/degeneration
Inflammation
Plaque growth
Remodeling of plaque and wall ECM
Organization of thrombus
Calcification
Mural thrombosis
Embolization
Wall weakening
ANEURYSM AND RUPTURE
Plaque rupture
Plaque erosion
Plaque hemorrhage
Mural thrombosis
Embolization
OCCLUSION BY THROMBUS
Progressive plaque growth
CRITICAL STENOSIS

VASCULITIS

- ♣ Definition: Vessel wall inflammation.
- ♣ Types & causes:

1. Infectious vasculitis:

-Bacterial or fungal infections of arterial wall (may lead to mycotic aneurysm).

-Syphilitic aortitis

2. Non-infectious vasculitis:

➢ Hypersensitivity vasculitis:

- ✓ These are immune mediated diseases, commonly by immune complex deposition in vessel wall.
- ✓ These are multisystem disorders but affecting mainly highly vascular tissues as the skin, glomerulus, lung and GIT. E.g.
  - polyarteritis nodosa,
  - systemic lupus erythematosus,
  - Henoch-schoenlein purpura.

➢ Buerger disease:

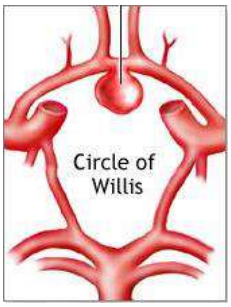
- ✓ (thrombangiitis obliterans) related to smoking

	POLYARTERITIS NODOSA (PAN)	THROMBANGITIS OBLITERANS (BUERGER'S DISEASE)
DEF	✓ An immune mediated systemic disease ✓ 1/3 of patients have chronic hepatitis B infection, which lead to formation of immune complex deposits containing B antigens in the vessel wall. ✓ Sex: males more than female. ✓ Site: small & medium sized arteries.	✓ A segmental, inflammatory disease ✓ It occurs almost exclusively in heavy smokers. ✓ Site: the tibial and radial arteries, adjacent veins, and nerves (neurovascular bundle).
Morphology	✓ Segmental transmural fibrinoid necrosis of arterial well, surrounded by an acute inflammatory reaction. ✓ Lesions usually affect only part of the vessel circumference.	✓ Sharply segmental acute transmural inflammation with microabscess formation. ✓ Inflammation often extends to adjacent veins and nerves. ✓ Thrombosis, organization and recanalization often occurs.
Complications: 1) Thrombosis with vascular occlusion or emboli resulting in infarctions. 2) Aneurysm due to segmental inflammatory weakening of the arterial wall. Occurs in coronaries, cerebral or mesenteric arteries. 3) Rupture resulting in tissue hemorrhages e.g. melena.		Clinical picture: 1) It affects mainly the legs, maybe the hands, with severe pain. 2) progressive ischemic changes which may end in gangrene.

ANEURYSMS

♣ Definition: Localized permanent arterial wall dilatation.

♣ Pathogenesis: Weakness of the vessel wall due to:



1) Congenital:
   - ➢ Absence of muscle wall and
   - ➢ Replacement by fibrous tissue.

2) Loss of smooth muscle cells as in:

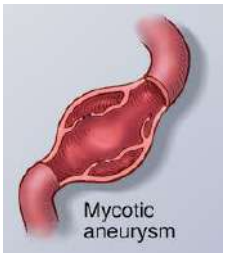
✓ Atherosclerosis:

- ➢ Thickened intima may cause ischemia of the media due to decrease diffusion of nutrients.
- ➢ Thickened vasa vasorum by hypertension may cause ischemia of the outer media.
- ➢ Common site of atherosclerotic aneurysm is abdominal aorta.

✓ Syphlitic aortitis:

- ➢ Inflammation of aorta in tertiary syphilis with endarteritis obliterans of vasa vasorum
- ➢ Causing: ischemia of the media, with loss of muscle cells.
- ➢ Syphilitic aneurysm occurs in ascending aorta.

3) Infection:



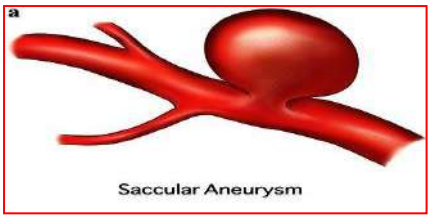
weakening of the arterial wall secondary to resulting in the so-called mycotic aneurysm , which results from:

- ➢ Embolization of infected embolus as a complication of infective endocarditis.
- ➢ Extension of an adjacent suppurative process.
- ➢ Commonest site is in the cerebral arteries.

4) Vasculitis:

- ➢ Immune mediated inflammation of arteries weaken the vessel wall
- ➢ leading to small arterial dilatation as in polyarteritis nodosa.

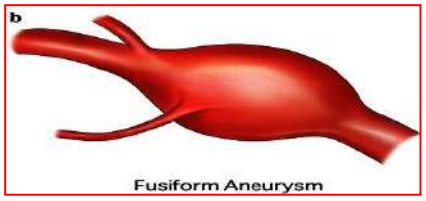
♣ Classification of aneurysms:



✓ According to shape into:

➢ Saccular aneurysm:

- are outpouchings from 5 to 20 cm, from one side of arterial wall, often with a contained thrombus.



➢ Fusiform aneurysms:

are circumferential Dilatations up to 20 cm in diameter.

These commonly involve aortic arch, the abdominal aorta and iliac arteries.

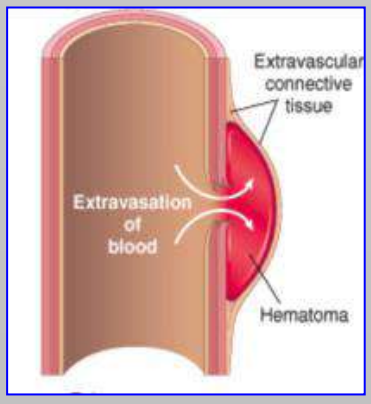
N.B: False aneurysms (pseudoaneurysm):

Aetiology:

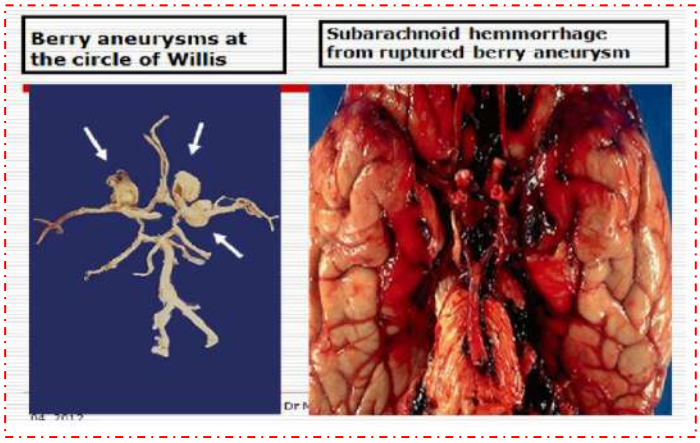
- arise due to a penetrating injury in arterial wall leading to the formation of an extravascular hematoma which becomes surrounded by adventitial fibrosis, forming a sac communicating with the vascular lumen
- (pulsating hematoma).

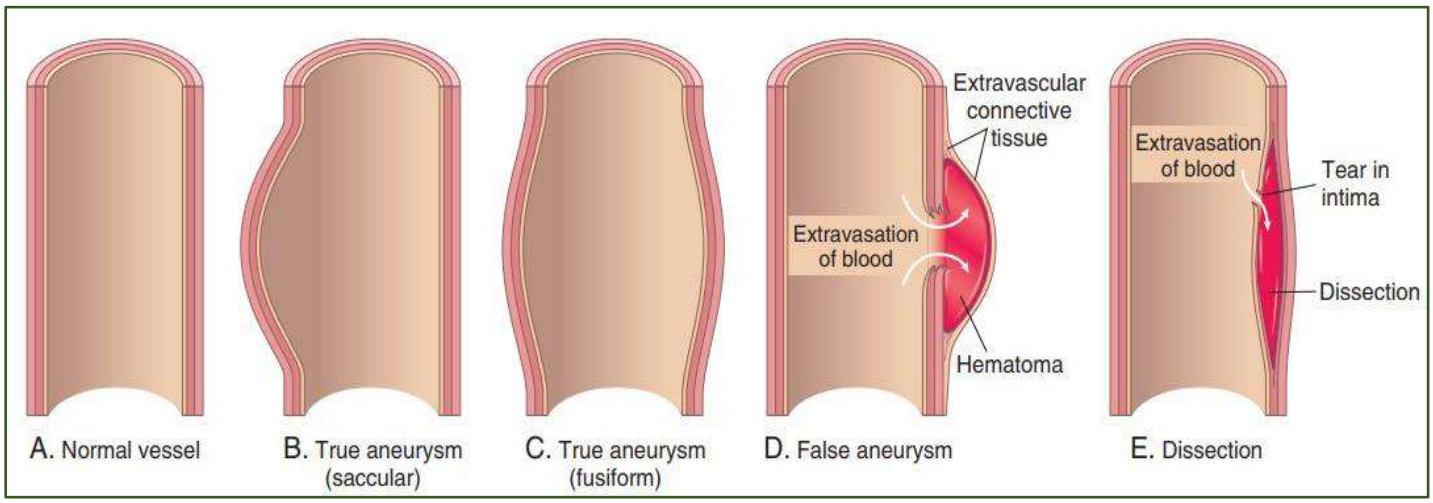
Examples of penetrating injuries:

- bullets or shrapnels,
- femoral artery puncture during arteriography or percutaneous angioplasty.



✓ According to etiology:

1) Congenital aneurysm:	Small berry aneurysm of cerebral arteries at the branching of the circle of Willis. Rupture leads to subarachnoid hemorrhage. 
2) Atherosclerotic aneurysm:	in abdominal aorta, iliac arteries. They are saccular or fusiform in shape.
3) Syphilitic aneurysm:	Saccular in appearance affects ascending part or arch of aorta.
4) Mycotic aneurysm:	cerebral, coronary, mesenteric.
5) Polyarteritis nodosa.	



♣ Complications of aneurysms

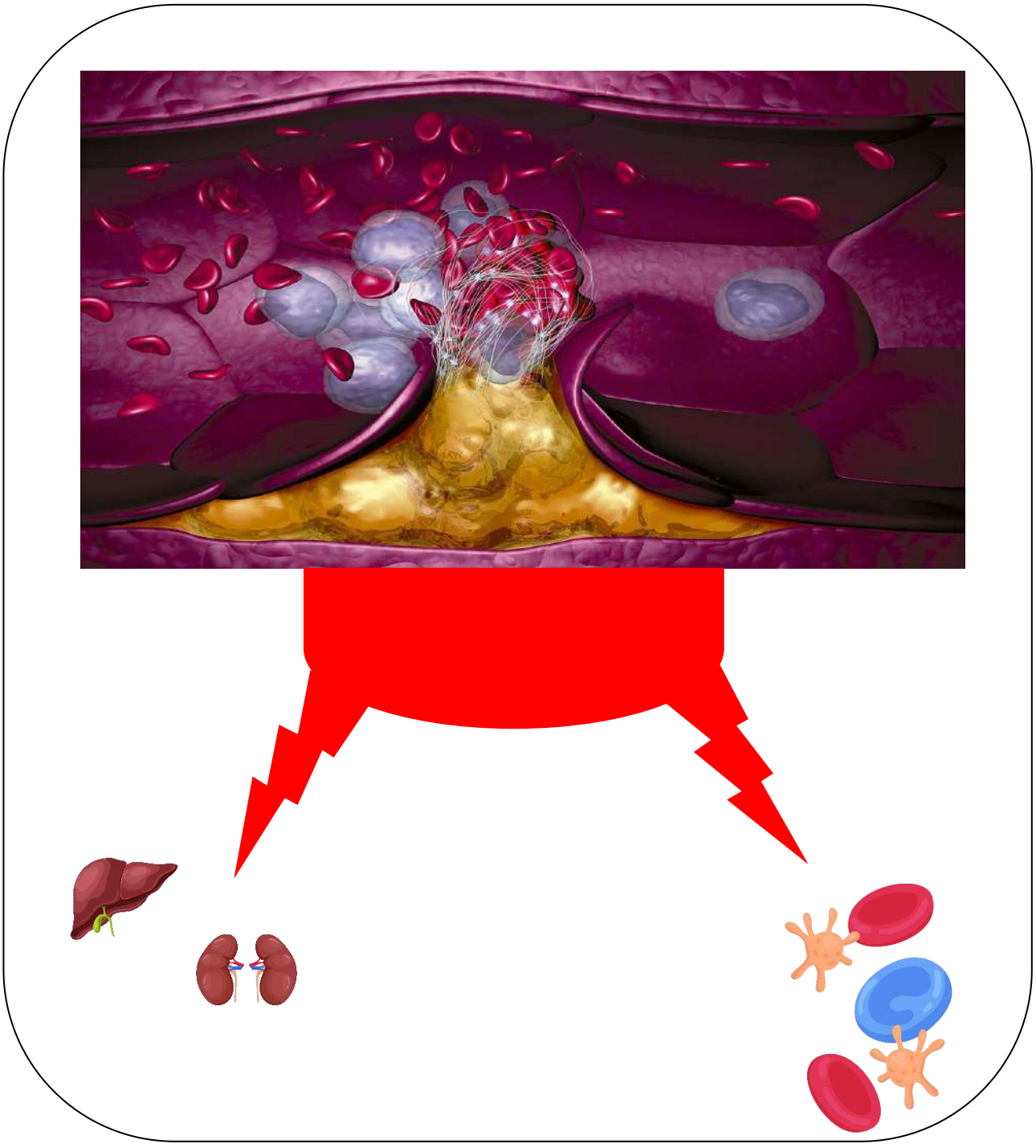
1) Rupture causing hemorrhage.
2) Mural thrombus formation

   with possible occlusion of ostia of vessels branching off the aorta

   e.g. renal or mesenteric artery.

   Embolism may occur.
3) Pressure on surrounding structures

   e.g. vertebrae in cases of aortic aneurysm.



Acute aortic dissection (dissecting aortic aneurysm)

♣ Pathogenesis:

Blood is forced through a tear in the intima of the aorta and tracks down the aorta splitting the media in two, forming a blood-filled channel within the aortic wall.

♣ Causes:

- ✓ Hypertension: is the main cause, with cases presenting at age 40-60.
- ✓ Marfan syndrome:
  congenital absence of fibrillin, a glycoprotein closely associated with elastic fibers. This occurs in younger patients.

♣ Morphology:

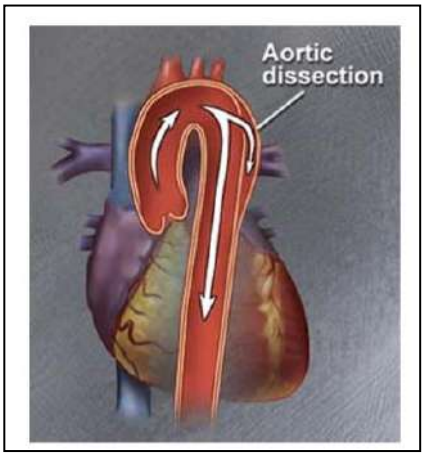
1) The tear commonly start in the aorta within 10 cm of aortic valve.
   The dissection spreads distally or towards the heart.

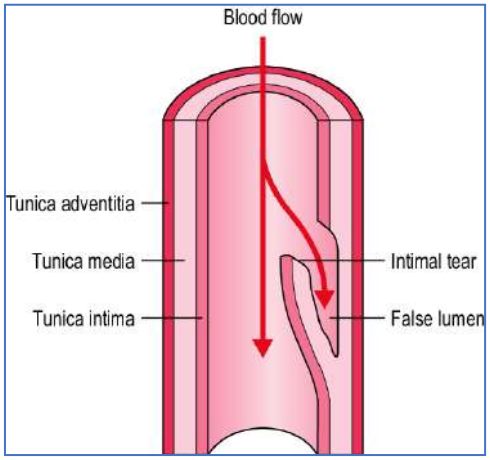
2) Microscopically:
   - ✓ cystic medial necrosis may be seen especially in cases associated with Marfan syndrome.
   - ✓ Medial necrosis is characterized by mucoid degeneration and elastic fiber fragmentation.

3) External rupture causes massive hemorrhage or
   cardiac temponade if it occurs in the pericardial cavity.

♣ Clinical picture:

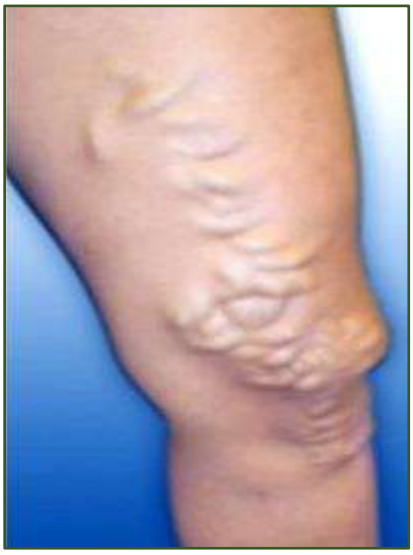
1) Sudden onset of severe pain starting in the chest, radiating to the back between the scapulae as the dissection progresses.
2) It is associated with high mortality and treatment is surgical.





VARICOSE VEINS

♣ Definition: abnormally dilated, tortuous veins,
produced by: prolonged, increased intraluminal pressure
leading to: vessel dilation and incompetence of the venous valves.



♣ Etiology:

✓ Familial predisposition.
✓ Risk factors include :

occupations with prolonged standing,

obesity and pregnancy.

♣ Complications:

1) Pain, congestion, edema and stasis dermatitis.

2) Chronic varicose ulcer (poor wound healing and superadded infection).

➢ Varicose veins of the esophagus (esophageal varices):

✓ Esophageal varices complicate:

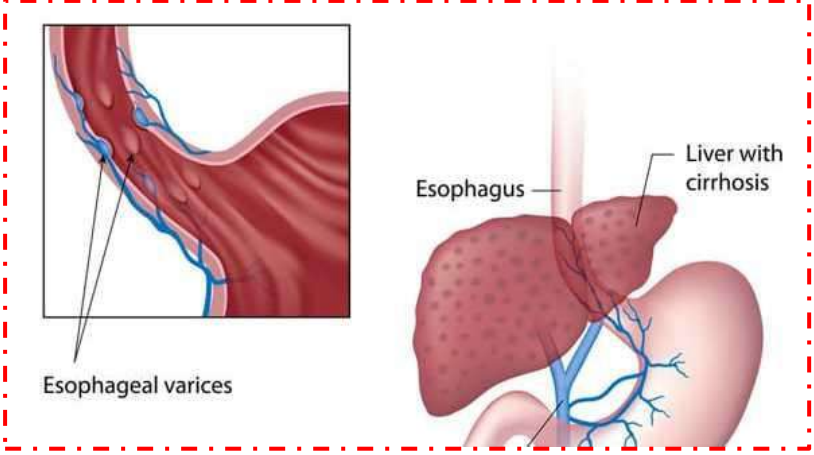
portal hypertension of any cause, .

cirrhosis and

bilharzial periportal fibrosis.

✓ This leads to :

- the opening of porto systemic shunts
- increased blood flow to the veins in lower esophagus.



➢ Hemorrhoids:

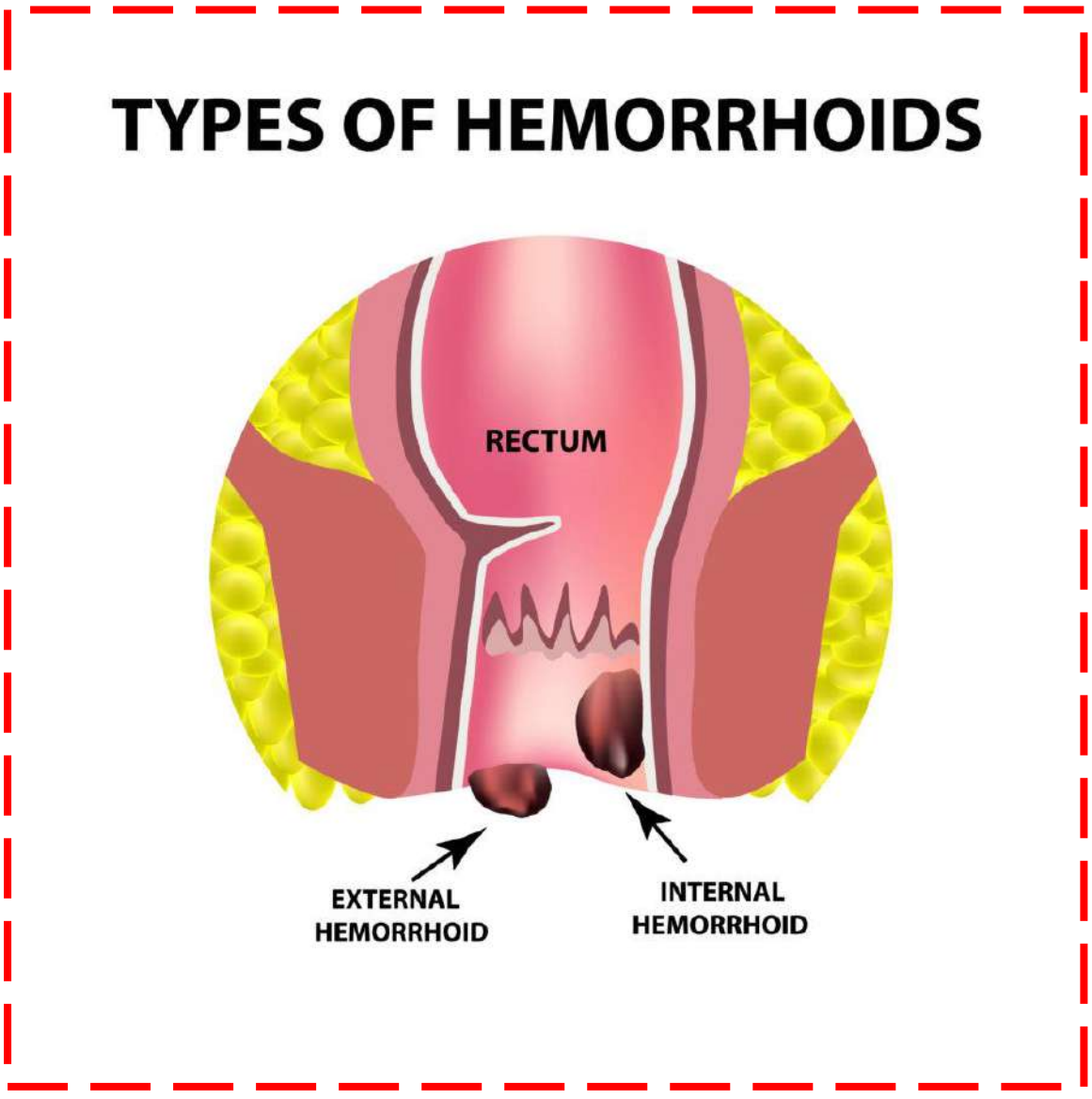
DEFINITION:

- Varicose dilatation of veins at the anorectal junction
- They are a source of bleeding per rectum, liable to thrombosis and painful ulceration.

Aetiology:

✓ from prolonged pelvic venous congestion associated with

- pregnancy
- and straining at defecation.



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PATHOLOGY

2023
2

C.V.S

DR AHMED ELNEMR

CONGENITAL HEART DISEASE

- They are due to faulty Embryogenesis during weeks 3-8 of gestation.
- The most severe anomalies are incompatible with intrauterine survival.
- Significant malformations are common among premature infants & stillborn.

Etiology:

1. Sporadic genetic abnormalities :

- ✓ major known causes of congenital heart disease.
- ✓ Forms: single gene mutations, small chromosomal losses, and additions.
- ✓ Also in association with Trisomy 21 known as Down syndrome.
- ✓ Rarely, they are hereditary.

2. Environmental factors: (alone or in combination with genetic factors).

- ✓ congenital rubella infection:
  (Infection of the mother with German measles during pregnancy)
- ✓ gestational diabetes.
- ✓ Teratogen exposure:
  Thalidomide, alcohol, unknown teratogens & exposure to radiation during pregnancy
- ✓ Nutritional factors :
  may also influence risk. for instance, folate supplementation during early pregnancy may reduce congenital heart disease rink.

Types:

➢ Most of the structural anomalies in congenital heart disease can be organized into three major categories:

- ✓ Malformations causing a left-to-right shunt ⇐
- ✓ Malformations causing a right-to-left shunt ⇒
- ✓ Malformations causing an obstruction e.g.:
  Aortic or pulmonary valve stenosis.

CONGENITAL HEART DISEASE WITH LEFT TO RIGHT SHUNT NON-CYANOTIC

Ventricular septal defect VSD

Types:

✓ **Membranous VSD**

Usually large & present in the membranous part of the septum.

✓ **Muscular VSD**

Small [< 0.5 cm] (Roger's disease)

Effect &complications:

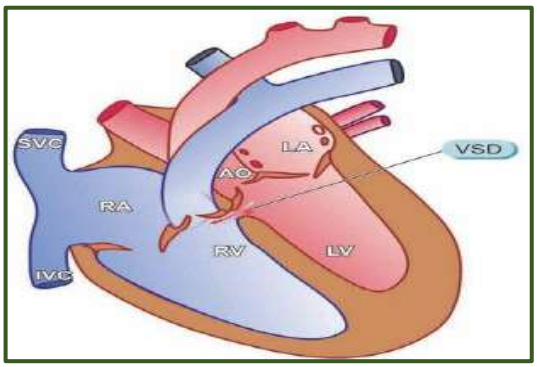
✓ May be isolated or associated with Fallot tetralogy

✓ Smaller lesions are generally well tolerated for years and may not be recognized until much later in life .

✓ **Large VSD:**

➢ **Causes:** left-to-right shunting,
➢ **leads to:** early right ventricular hypertrophy & pulmonary hypertension.
➢ **Result:** shunt reversal, cyanosis, & death .

✓ Infective endocarditis opposite the shunt.



Atrial septal defect ASD

Effect &complications:

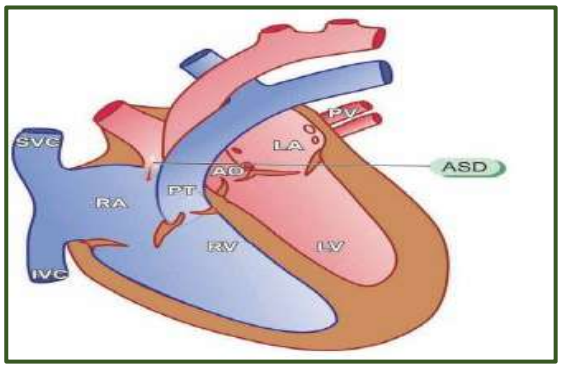
✓ ASDs result in a left-to-right shunt (higher pressure in left atrium).

✓ murmur is often present as a result of excessive flow through the pulmonary valve or through the ASD.

✓ ASDs are generally well tolerated and usually do not become symptomatic before age 30, irreversible pulmonary hypertension is unusual.

✓ **ASD closure :**

➢ (surgical or catheter-based) reverses the hemodynamic abnormalities
➢ prevents the complications, including :
➢ heart failure, paradoxical embolization, and irreversible pulmonary hypertension.



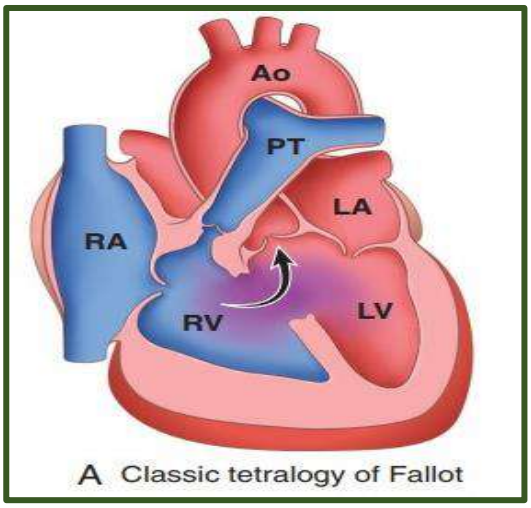
CONGENITAL HEART DISEASE WITH RIGHT-TO-LEFT SHUNTS CYANOTIC

Fallot's Tetralogy [PROVE]

- ✓ Pulmonary stenosis
- ✓ RV hypertrophy.
- ✓ Overriding of aorta

(Aorta opening in both Right & left ventricles(

- ✓ VSD.



A Classic tetralogy of Fallot

♣ Effects & complications of Right to Left Shunt with: [C]

- ✓ Cyanosis.
- ✓ Secondary polyCythemia.
- ✓ Clubbing of fingers.
- ✓ Infective endocarditis.

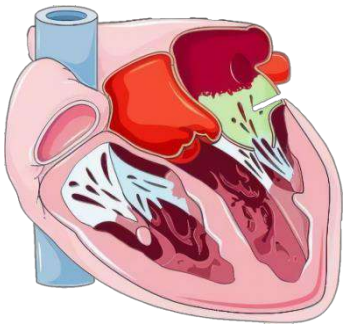
ENDOCARDITIS

♣ Definition:

- ✓ Inflammation of valvular or mural endocardium .
- ✓ It may be infective or non-infective .
- ✓ Valvular endocarditis is characterized by :

vegetations, which are small platelet (pale) thrombi on valve cusps.

♣ Etiology & types:



A. Non infective endocarditis:

1) Rheumatic endocarditis. [Immune mediated]

2) Endocarditis of systemic lupus erythematosus (SLE). (Libmann Sack's endocarditis.)

3) Non-bacterial thrombotic endocarditis:

- It occurs in prolonged debilitating diseases as chronic sepsis, uremia & malignancy.
- It represents a hypercoagulable state. They can be the source of systemic emboli.

B. Infective endocarditis (see later).

1. RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

♣ Definition:

✓ It is an inflammatory, immune mediated multisystem disease
✓ affecting most importantly the heart in the form of a pan carditis
✓ It occurs as a complication of streptococcal pharyngitis & tonsillitis.

♣ Etiology:

Predisposing factors:

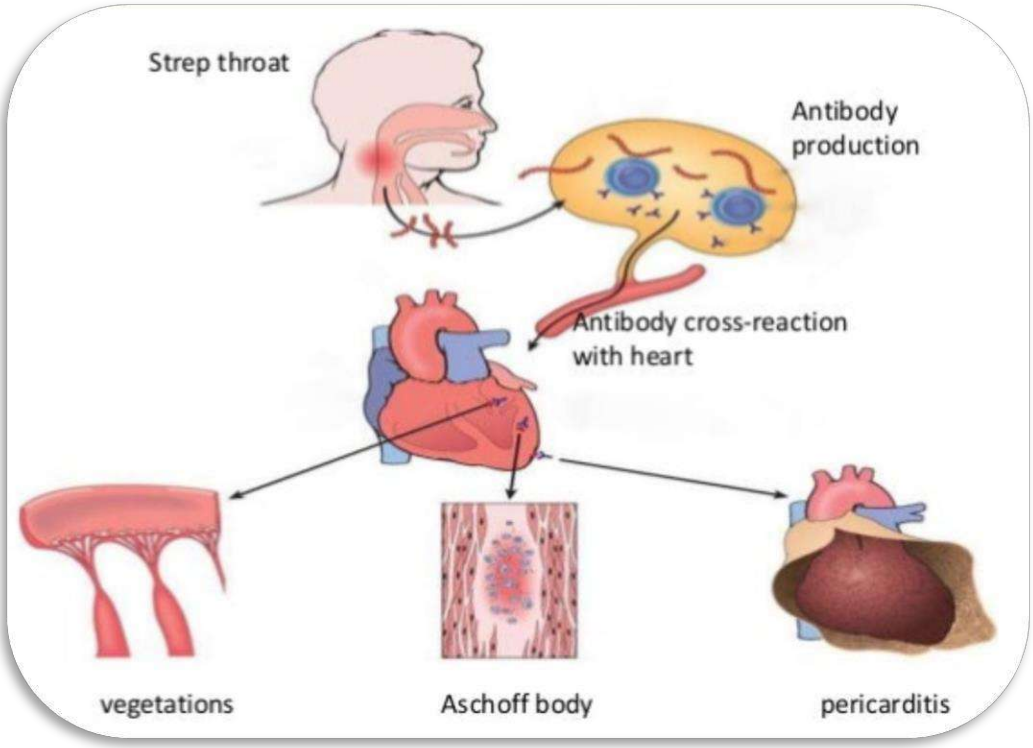
- It is endemic in Egypt and low socio-economic developing countries due to:

[overcrowding, low resistance & increasing of airborne respiratory tract infections]

- It usually starts in children & adolescents 5-15 years
- It starts 2-3 weeks after streptococcal sore throat or streptococcal upper respiratory tract infection by group A, beta hemolytic streptococcus. [GAβS]
- Acute rheumatic carditis is a common manifestation of active R.F & may progress over time to chronic rheumatic heart disease (RHD), mainly manifesting as valvular abnormalities.
- Valvular manifestations may begin to appear years after the first attack of R.F or after repeated attacks produce significant valve deformity.

Pathogenesis:

- Acute R.F results from abnormal host immune responses to [GAβS] antigens that cross-react with host proteins. Such abnormal response may be genetically determined.
- Antibodies and CD4+ T cells directed against streptococcal M-proteins recognize cardiac self-antigens.
- Antibody binding can activate complement & recruit Fc receptor bearing cells (neutrophils and macrophages).
- Cytokine production by the stimulated T cells leads to macrophage activation (e.g., within Aschoff bodies).
- Damage to heart tissue may thus be caused by a combination of antibody- and T cell-mediated reactions.



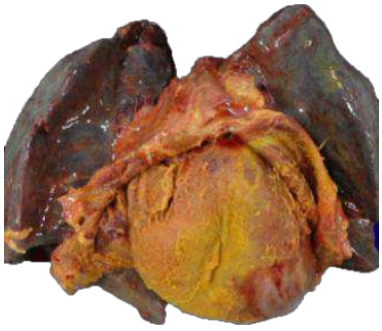
♣ Morphology:

A. ACUTE RHEUMATIC FEVER

1. Cardiac manifestations (Rheumatic pan carditis):

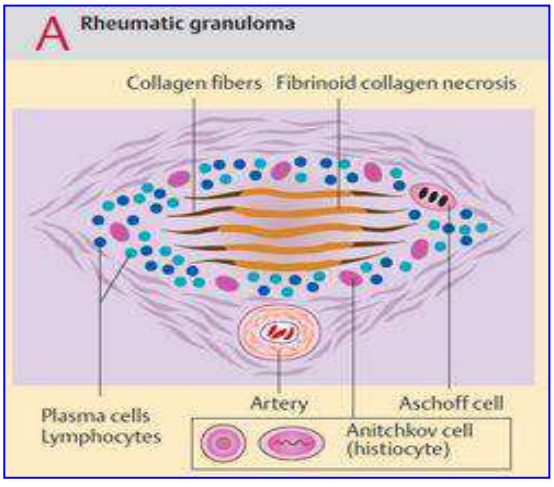
1) Pericardium: Fibrinous pericarditis

(Fibrin deposited between the visceral & parietal layers give the appearance of bread & butter pericarditis)

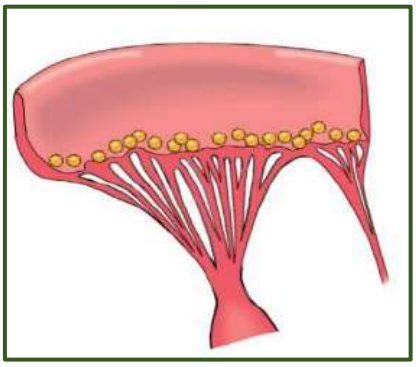


2) Myocardium:

- Distinctive lesions of R.F is called Aschoff bodies.
- Gross: 1-2 mm grey nodules in myocardium
- Micro: They are seen in the myocardial interstitial tissue, in a Para vascular location.
  They consist of foci of fibrinoid degeneration surrounded by T lymphocytes, occasional plasma cells, and plump activated macrophages called Anitschkow cells or caterpillar cells, which may be multinucleated.



3) Endocardium (endocarditis):



➢ Valvular endocardium:

Site: LT site of heart

mitral valve is the most commonly affected,

This is followed by Aortic & tricuspid,

rarely pulmonary valve.

(mitral > aortic >>tricuspid)

Gross:

- Swollen cusps carrying small 1-2 mm pale brown vegetations.

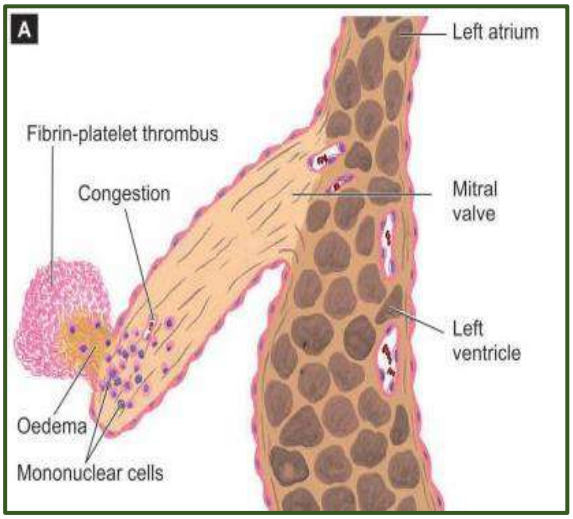
- Vegetations:

✓ are arranged at the line of closure of the cusps in a regular linear pattern

(Site of most friction due to closure of valve and trauma of blood passage).

✓ are adherent (firmly fixed) to the valve.

✓ They are present in face of the direction of blood flow (atrial surface of mitral valve & ventricular surface of the aortic valve).

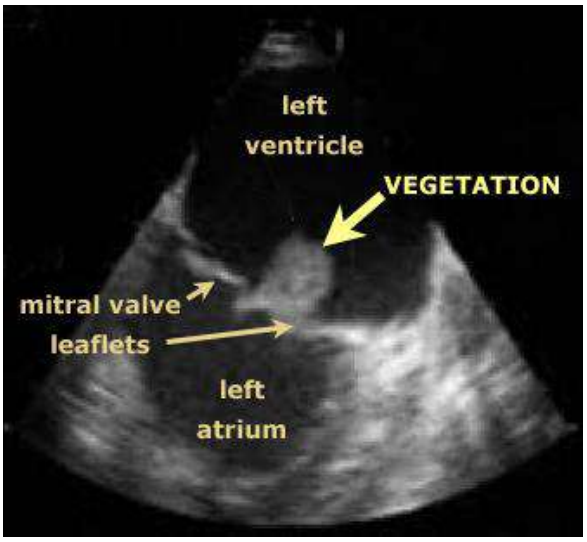


Micro:

Fibrinoid necrosis of cusps and

chordae tendinea with inflammation

and overlying vegetations (platelet thrombi).



➢ Mural endocardium:

of the left atrium may show

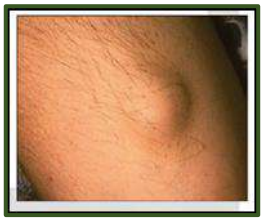
Aschoff bodies (AB) on the posterior wall.

2. Extra cardiac manifestations:

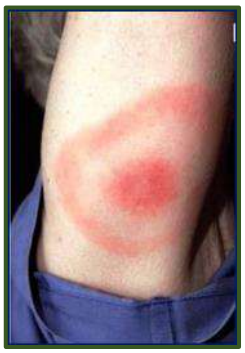
A. Joints :

Fleeting arthritis Large joint (knee or elbow) is red, painful and swollen.
As soon as the inflammation undergoes resolution another joint becomes affected. The arthritis resolves completely with no residual effect on joints.

B. Skin:



✓ Skin rash (erythema annulare or marginatum)
Reddish macules with a pale center.
Seen particularly on the trunk.



✓ Aschoff's nodules:
Subcutaneous nodules (common over bony prominence as knuckles of hand, in the form of 2-20 mm nodules [with a micro similar to AB].

C. Sydenham's chorea: Rheumatic chorea:

spontaneous uncoordinated purposeless movements of an involuntary nature due to: basal ganglia inflammation.

D. Fibrinous pleurisy or peritonitis.

E. Rheumatic pneumonitis may occur.

F. Rheumatic arteritis or hypersensitivity angiitis:

a type III hypersensitivity reaction.

♣ Clinical picture of rheumatic fever:

✓ Rheumatic fever is characterized by:
fever, fleeting arthritis of the large joints, pan carditis (tachycardia, murmurs, pericardial rub), subcutaneous nodules, erythema marginatum of the skin, and Sydenham chorea.

✓ Investigations:
◆ Blood tests include:
elevated sedimentation rate, elevated C reactive protein and high serum level of ASO titre.

♣ Fate of acute rheumatic fever:

✓ Most cases of rheumatic fever show recovery.
✓ Only 1% die from fulminant myocarditis
✓ Recurrent acute attacks:
cause fibrosis and deformity of cardiac valves.

B.CHRONIC RHEUMATIC HEART DISEASE

♣Represents the healing phase of rheumatic fever and is characterized by fibrosis.

a) Pericardium: Commonly heal without sequelae.

Occasionally fibrosis may cause whitish patches on visceral pericardium (Milk spots), adhesions between visceral and parietal pericardium, or rarely adhesive or constrictive pericarditis.

b) Myocardium:

Healing of AB by fibrosis producing fine white streaks of fibrous tissue appear within the muscle.

c) Endocardium:

1. Mural endocardium:

Fibrosis of the AB of the posterior wall of left atrium resulting in:

a whitish patch of fibrous tissue called MacCallum patch.

2. Valvular endocardium:

Site: Mitral valve is commonly affected, followed in frequency by Aortic& tricuspid, rarely pulmonary volve. (One or more valves may be diseased)

Gross: The diseased valves undergo fibrosis becoming thick, opaque whitish and deformed. Leading to:

Mitral stenosis	Mitral incompetence	Double valve lesions
✓ Valve: ➢ Narrow due to Fibrosis & Fusion [has a button hole or fish mouth appearance]. ➢ appears as: a funnel when looked at from the atrium. ✓ chordae tendinea: thick & short. ✓ papillary muscles: hypertrophy.	✓ valve cusps: shrinkage due to fibrosis ➢ Leading to: improper closure with blood leaking despite closure of the valve.	double mitral or double aortic). This is a lesion where the valve suffers a deformity, which produces both stenosis & incompetence. The fibrosis affects both the commissures & the leaflets producing a narrow valve, which doesn't close properly.

♣ Complications of chronic rheumatic heart disease :

1) Subacute infective endocarditis on top of deformed fibrotic valves.
2) Heart failure secondary to valvular lesions.

2.INFECTIVE ENDOCARDITIS

	ACUTE INFECTIVE ENDOCARDITIS	SUBACUTE INFECTIVE ENDOCARDITIS
Etiology +Prognosis	♣Caused by infection of a previously normal heart valve by high virulent organism (e.g. Staphylococcus aureus) ♣rapidly produces necrotizing and destructive lesions.	♣Occurs in bactremic states ♣Organism is of low virulence& non-pyogenic (Such as viridians streptococci)
	♣Organisms reach the blood from: - cutaneous infections - lung infections - genitourinary tract infections.	♣These organisms reach the blood from: - minor surgeries as tooth extraction and tonsillectomies. ♣Such weak organisms do not attack healthy valves: only abnormal endocardium e.g. chronic rheumatic valvular disease, congenital heart disease or prosthetic valves
	♣It is rapidly fatal owing to : - Septicemia & Perforation of cusps [which produces acute heart failure.]	♣Usually treatable. ♣but healing by fibrous tissue produces: more valve deformity such as stenosis or incompetence
Valves	Mitral, aortic, mural endocardium, Valves on right side are affected in intravenous drug users	Mitral & aortic commonly & mural Endocardium
Gross	♣Vegetations: Large, (bulky), yellow (purulent), friable ♣Valve: perforated ♣Cusps : thin (normal) & do not show fibrosis ♣Chordae tendinea: May be ruptured	♣Vegetations: Large (bulky), brownish, friable ♣Valve: NO perforation ♣Cusps: thick (abnormal) & show fibrosis due to previous RHD. ♣No rupture of chordae (already thickened fibrosed)

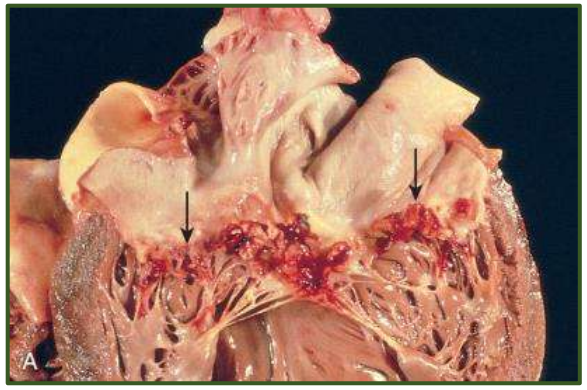
Micro	Platelets, fibrin, bacteria,neutrophils, pus cells & Macrophages	Platelets, fibrin, bacteria, neutrophils, lymphocytes, plasma cells à macrophages (No pus cells

Clinical Pic+ Complications

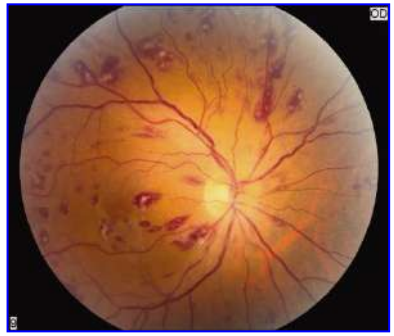
♣Septicemia: Fever, chills, weakness.

♣Embolic manifestations:

- Septic infarctions.
- Pyaemic abscesses.

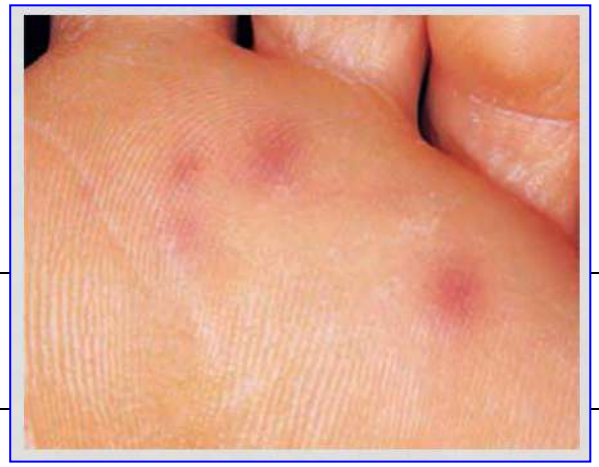


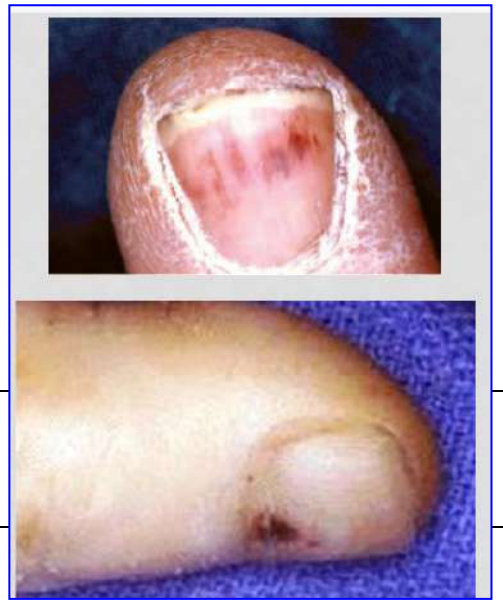
♣Toxemia: Fever, clubbing of fingers.



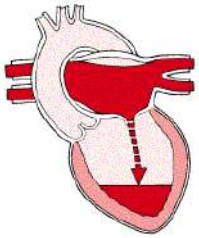
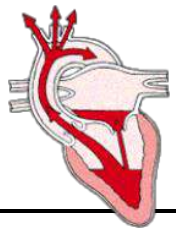
♣Embolic manifestations:

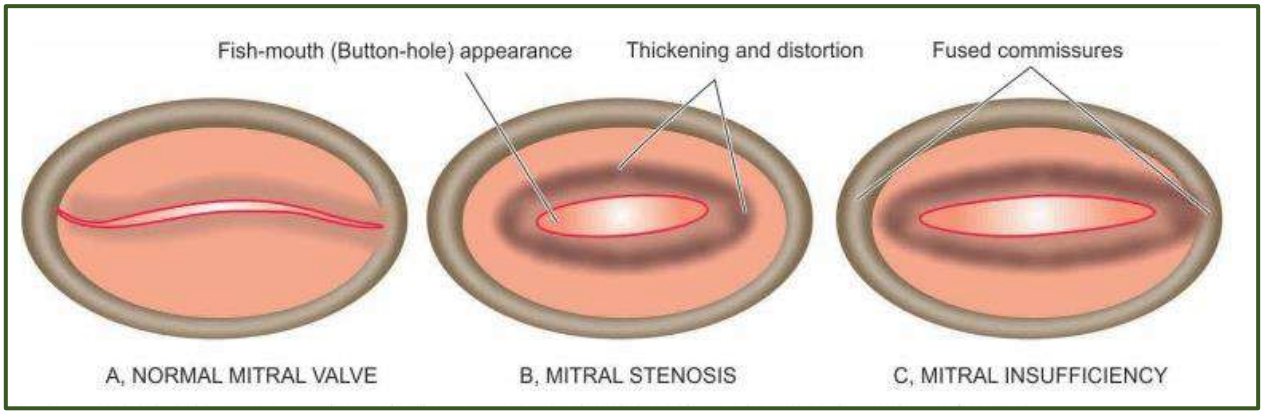
1) Cerebral infarction.
2) Renal infarctions.
3) Splenic infarctions & splenomegaly.
4) Retinal vessel emboli & Hge (Roth spots.)
5) Multiple petechiae in skin & splinter Hge in nail bed (Osler's nodules):
6) painful fingertip nodules.
7) Mycotic aneurysms.
8) Focal glomerulonephritis due to immune complex de position.



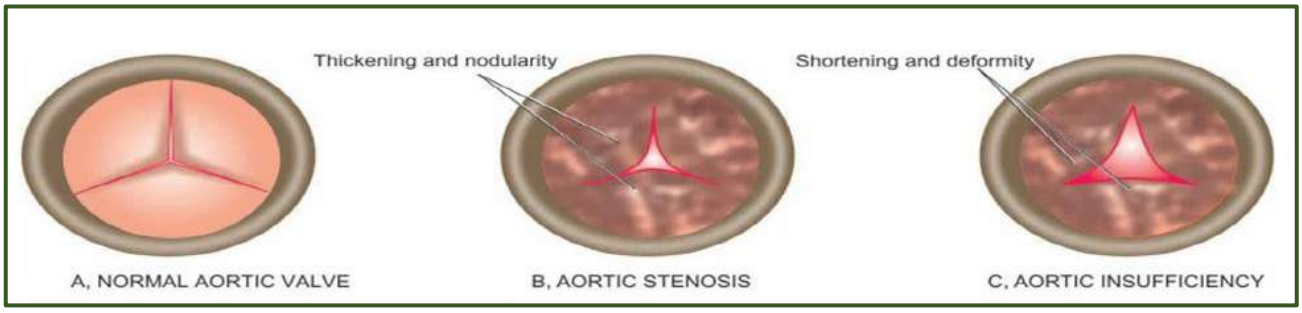


VALVULAR DISEASES

	MITRAL STENOSIS	MITRAL INCOMPETENCE
Causes	♣Chronic rheumatic heart disease common cause in Egypt ♣Healed subacute infective endocarditis - Pathology (see Rheumatic heart disease) 	1) Chronic rheumatic heart disease 2) Healing of subacute infective endocarditis 3) Dilatation of mitral ring 2ry to Lt. ventricular dilatation 4) Rupture of papillary muscle in recent infarction 5) Mitral valve prolapse (myxomatous degeneration of mitral valve).
Effects	1. Progressive dilatation & hypertrophy of left atrium occurs. 2. Left atrial thrombosis can embolize 3. Arrythmias, particularly atrial fibrillation 4. Chronic venous congestion pulmonary hypertension of the lungs, over time leads to 5. Pulmonary hypertension : - right ventricular hypertrophy - Inter dilatation and failure 6. The left ventricle is unaffected in isolated mitral stenosis.	1) Progressive dilatation & hypertrophy of Lt. Atrium & Lt. Ventricle 2) Chronic venous congestion of lung leads to pulmonary hypertension, 3) pulmonary hypertension: reflected on right side of the heart hypertrophy and dilatation 4) Heart failure. 



PULMONARY STENOSIS	TRICUSPID STENOSIS
♣Causes: - Congenital - Healed subacute infective endocarditis - Chronic rheumatic heart disease - Carcinoid syndrome ♣Effects: - Hypertrophy of right ventricle - Right ventricular failure.	♣Causes: - Chronic rheumatic valvulitis - Healed subacute infective endocarditis - Carcinoid syndrome: serotonine released promotes valve fibrosis on the right side of the heart

	AORTIC STENOSIS	AORTIC INCOMPETENCE
Causes	1. Rheumatic heart disease 2. Healed subacute infective endocarditis 3. Congenital aortic stenosis 4. Senile calcific aortic stenosis 5. Calcification of congenital bicuspid valve	1) Rheumatic heart disease. 2) Infective endocarditis. 3) Syphilitic aortitis. 4) Marfan syndrome.
Effects	1. Concentric hypertrophy of left ventricle due to pressure overload. 2. The hypertrophied myocardium tends to be ischemic. - as a result of : diminished microcirculatory profusion - complicated by: coronary atherosclerosis and angina pectoris may occur 3. Eventually cardiac decompensation occurs and congestive heart failure develops. 4. Symptoms include angina, syncope and symptoms of heart failure.	1) Dilatation of all chambers of the heart. 2) The heart is more than 1 Kg (Cor-Bovinum) 3) Heart failure.
		

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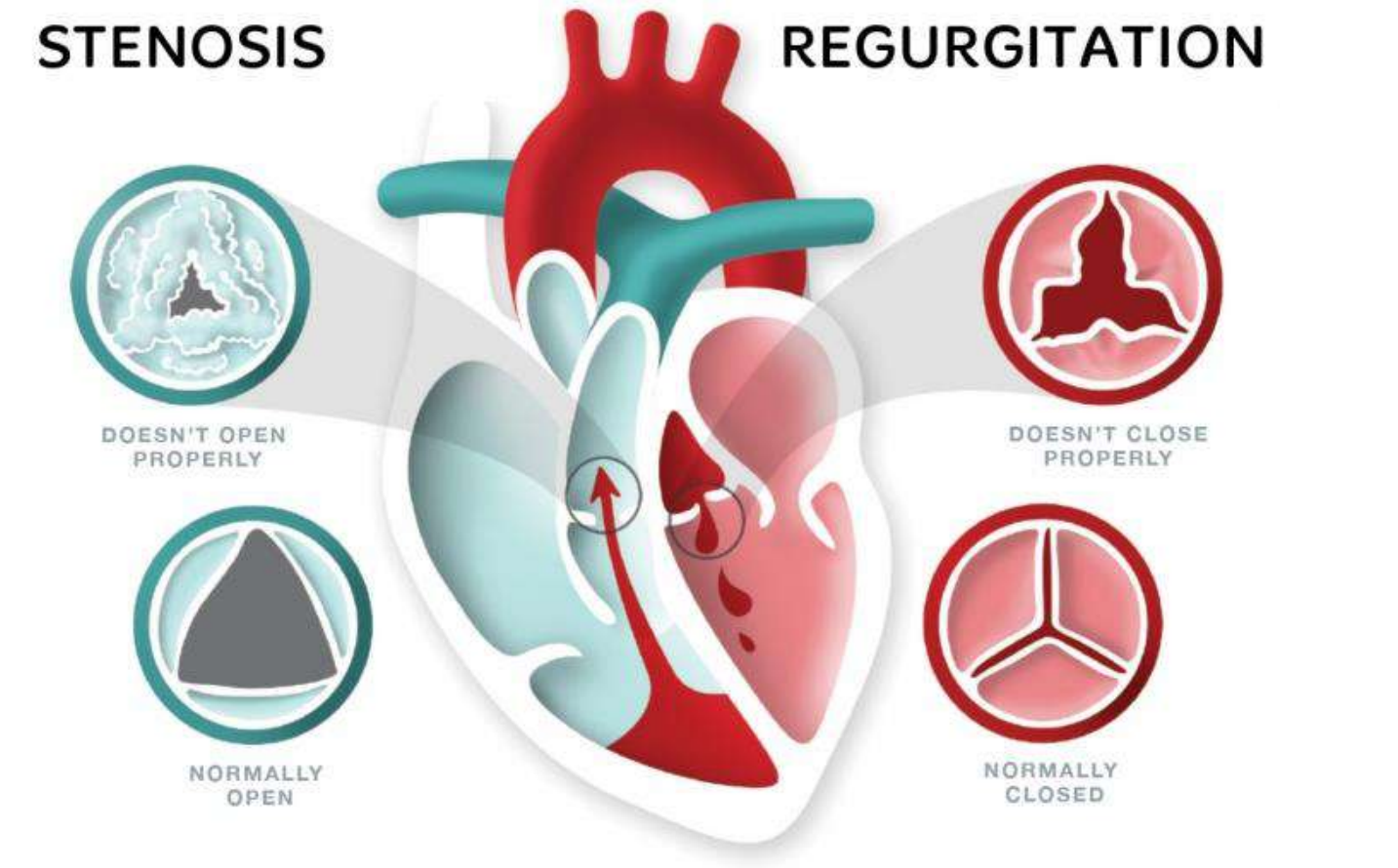
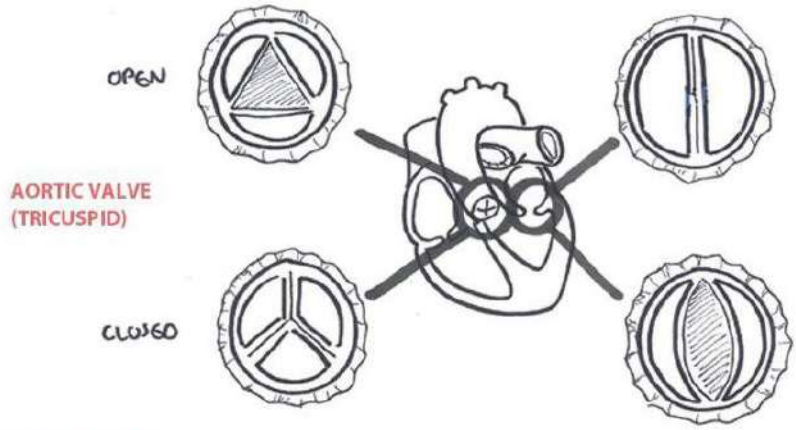
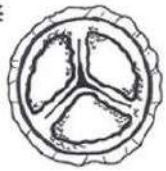




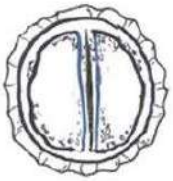
AORTIC STENOSIS

THINK OF AS IN THE ELDERLY WITH EXERTIONAL ANGINA, DYSPNEA, SYNCOPE



MITRAL STENOSIS (THINK AF)

CAUSES: RHEUMATIC, CONGENITAL, CALCIFICATION



AORTIC INCOMPETENCE

ACUTE (INFECTIVE ENDOCARDITIS) OR CHRONIC (MANY CAUSES)

MITRAL INCOMPETENCE (THINK AF)

CAUSES: LV DILATATION, IE, RF, MITRAL VALVE PROLAPSE, POST MI

ISCHEMIC (CORONARY) HEART DISEASE (IHD)

♣ Definition

♣It represents a group of syndromes resulting from: myocardial ischemia

[an imbalance between myocardial supply (perfusion) & cardiac demand for oxygenated blood].

♣ Causes:

1. In more than 90% of cases, it is due to atherosclerosis of the coronary arteries
2. Other causes of ischemia include:
   - Coronary emboli
   - Coronary vessel inflammation
   - Vascular spasm
   - Narrowing of coronary ostia due to syphilitic aortitis
   - Increased oxygen demand as in myocardial hypertrophy, or tachycardia
   - Diminished perfusion as in shock.

♣ presentation:

♣ IHD can present as one or more of the following clinical syndromes:

1. . Myocardial infarction (MI): where ischemia causes frank myocardial necrosis
2. Angina pectoris : chest pain precipitated by exercise
3. Chronic IHD with heart failure Sudden cardiac death (SCD).
4. IHD is the leading cause of death in the developed nations.

♣ Epidemiology: Ischemic heart disease include:

A. CHRONIC ISCHEMIC HEART DISEASE

♣ Causes:

1. Atherosclerosis: more than 90% of patients with IHD have atherosclerosis involving one or more of the coronary arteries
2. Fixed lesion obstructing > 75% of vascular cross-sectional area defines significant coronary artery disease. Patients present with angina pectoris.
3. Clinically significant plaques can be located anywhere along the course of the coronary vessels, although they tend to predominate within the first several centimeters of the left anterior descending (LAD) and left circumflex (LCX).

♣ Pathological changes of chronic ischemic heart disease

1. The heart is normal in size, small in size (atrophy) or increased in size due to associated hypertension
2. The myocardium shows grey fibrous streaks
3. The left ventricle shows subendocardial fibrosis
4. Mitral and aortic valve thickening and calcification, with normal chordae tendinea.

B. MYOCARDIAL INEARCTION

♣ Definition:

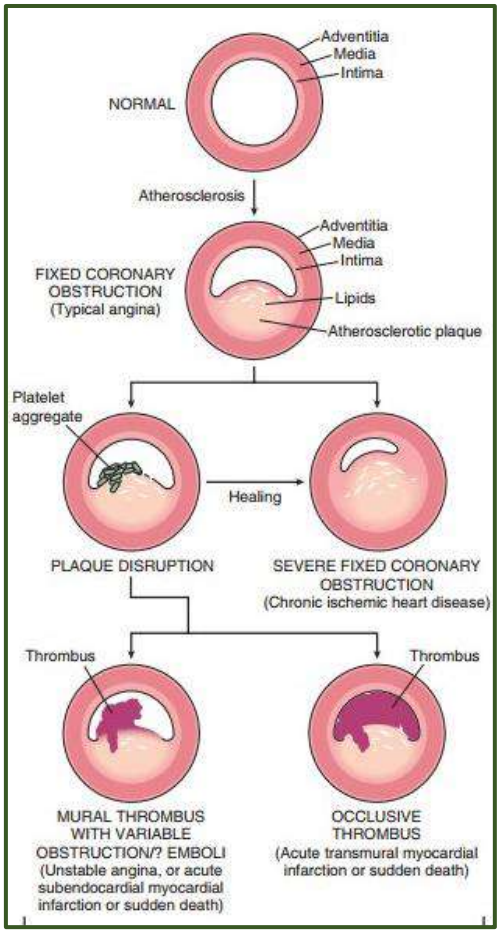
It is also commonly referred to as [heart attack] is the death of cardiac muscle due to prolonged severe ischemia.

♣ Pathogenesis:

1. Coronary Arterial Occlusion 90% of cases :

♣The following sequence of events likely underlies most Myocardial infarctions:

A. A coronary artery atheromatous plaque:
undergoes an acute change consisting of intra plaque hemorrhage, erosion, ulceration, rupture or fissuring



B. Microthrombi :
When exposed to subendothelial collagen & necrotic plaque contents, platelets adhere, become activated, release their granule contents and aggregate to form microthrombi.

C. Vasospasm:
is stimulated by mediators released from platelets.

D. Coagulation pathway:
is activated by Tissue factor, adding to the bulk of the thrombus within minutes, the thrombus can expand to completely occlude the vessel lumen.

2. Other causes 10% of cases:

A. Vasospasm with or without coronary atherosclerosis, perhaps in association with platelet aggregation or due to drug ingestion (e.g.. cocaine or ephedrine)
B. Emboli from the left atrium in association with atrial fibrillation e.g. a left-sided mural thrombus, vegetations of infective endocarditis
C. Dissecting aortic aneurysm involving the coronary ostia.
D. Coronary vasculitis, shock etc……

N.B:
Sudden coronary occlusion may:
- Be asymptomatic, if good collaterals are present,
- Present by sudden death due to fatal arrhythmias e.g. ventricular fibrillation.
- Lead to myocardial infarction.

♣ Pathological features of myocardial infarction:

- Due to the myocardial perfusion pattern from epicardium to endocardium.
- Ischemia is most pronounced in the sub endocardium; thus, irreversible injury.
- Necrosis of ischemic myocytes occurs first in the subendocardial zone, then moves through the myocardium to encompass progressively more of the transmural thickness of the myocardium.

♣ Sites of myocardial infarction:

1) Anterior infarction:

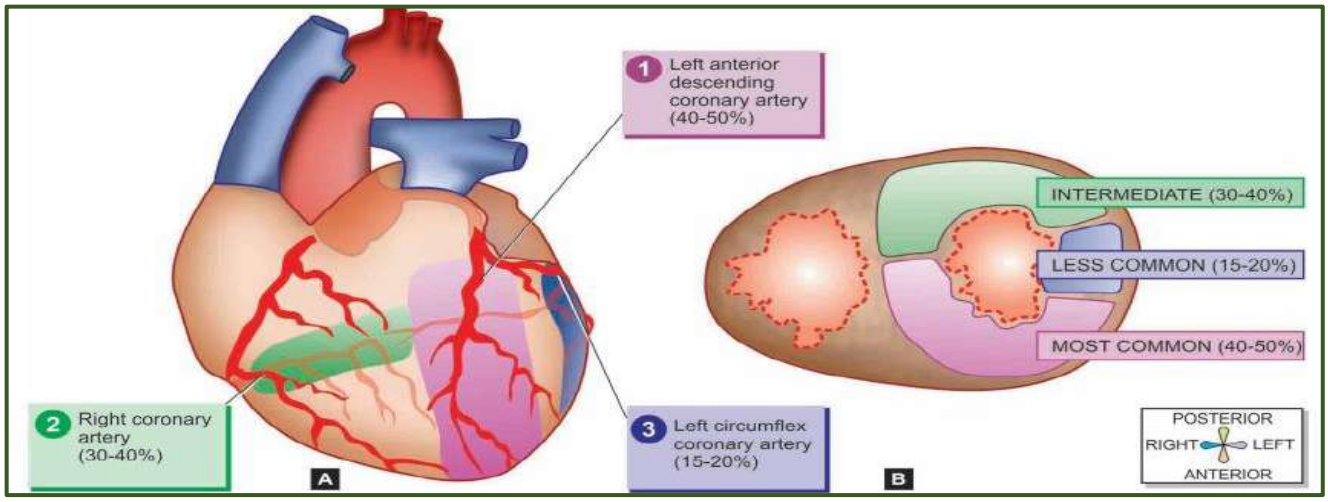
The most common site (40-50%) It is due to occlusion of the left anterior descending branch (LAD) producing an infarction of the anterior ventricular wall, the anterior part of the interventricular septum & the apex of the heart

2) Lateral infarction:

due to occlusion of the left circumflex coronary artery (LCX) resulting in infarction of the lateral ventricular wall.

3) Posterior infarction:

due to occlusion of the right coronary artery with infarction of the posterior wall & the posterior part of the septum.



♣ Patterns of myocardial infarction:

i. Transmural infarction:

due to epicardial vessel occlusion (atherosclerosis, plaque rupture and thrombosis), without any therapeutic intervention.

ii. Subendocardial infarction:

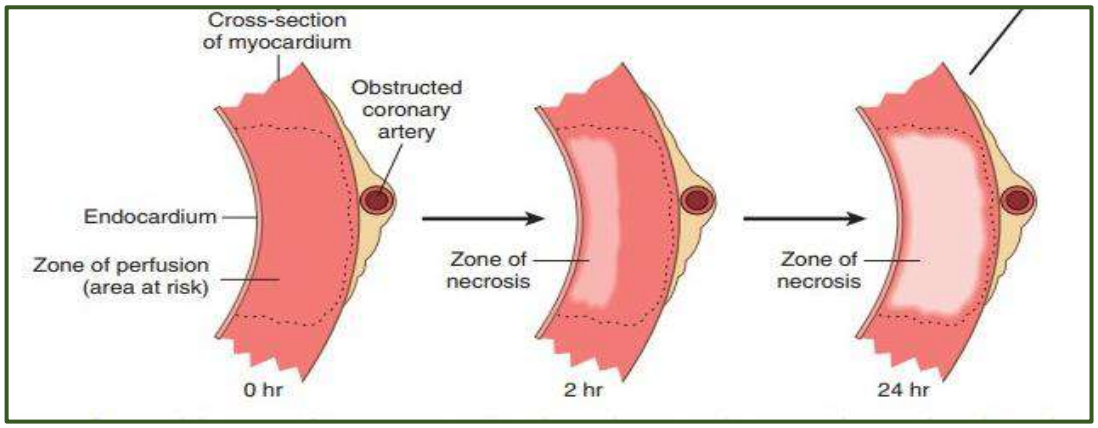
- It is typically involving the inner third of the ventricular wall-can occur due to epicardial vessel occlusion, where thrombus becomes lysed (therapeutically or spontaneously) before necrosis extends to full thickness of myocardium.

- As the subendocardial region is the least perfused region, subendocardial infarcts can also result from prolonged, severe reduction of blood pressure, such as in shock, superimposed on noncritical coronary stenosis. In such case, myocardial damage is usually circumferential, rather than being limited to the distribution of a single major coronary artery.

♣ Morphology of myocardial infarction:

Gross:

- **Around 15 hours:**
  The earliest naked eye changes occur, when the affected muscle appears pale and swollen.
- **By 24-48 hours:**
  there is softening and the color changes from dark red to brown to yellow.
- **After 3-4 days:**
  the infarct is more sharply defined by the development of a peripheral red zone of granulation tissue.
- **At 3 weeks:**
  Removal of dead myocytes by phagocytosis and organization proceeds with variable thinning of the infarcted area and some fibrosis.
- **By 3 months:**
  the scar tissue appears white.
- A fibrinous or hemorrhagic pericarditis is common in transmural infarction.
- **Under the endocardium:**
  a thin surviving subendocardial band of myocytes is almost invariable, these cells being nourished by blood in the ventricular lumen.
- **mural thrombus :**
  often forms on the endocardial surface overlying the infarct after several days.



Microscopic:

- **After 8-12 hours:**
  The infarcted tissue shows the changes of coagulative necrosis
- **By 24 hours:**
  invasion by polymorphs has begun and after a few days' digestion by macrophages and organization with granulation tissue formation occurs at the periphery
- **Over the next 2-3 months:**
  the infarcted myocardium is eventually replaced by a fibrous scar.
- **ventricular remodeling:**
  Hypertrophy of the non-infarcted myocardium with chamber enlargement then compensate for loss of contractility caused by the healed infarct.

♣ Complications of myocardial infarction:

1. Contractile dysfunction and some degree of heart failure with pulmonary congestion & edema. Large infarcts can cause cardiogenic shock.

2. Arrythmias:
   due to myocardial irritability and affection of the conduction system in the atrioventricular septum (tachycardia, heart block, ventricular fibrillation).

3. Cardiac rupture:
   This occurs most frequently 2 to 4 days after MI, when coagulative necrosis, neutrophilic infiltration, and lysis of the myocardial connective tissue have weakened the infarcted myocardium.

4. Pericarditis:
   fibrinous or fibrin hemorrhagic pericarditis usually develops about the 2nd or 3rd day following a transmural infarct as a result of underlying myocardial inflammation

5. Mural thrombosis:
   due to stasis and endocardial damage.

6. Papillary muscle dysfunction:
   early dysfunction, leading to acute mitral incompetence, or late ischemic dysfunction, leading to post infarct regurgitation.

7. Ventricular aneurysm:
   It is a late complication due to weak scar that bulges during the systole. Complications of ventricular aneurysms include mural thrombus, arrhythmias, and heart failure, and occasionally rupture.

8. Progressive late heart failure (CIHD).

♣ Clinical picture:

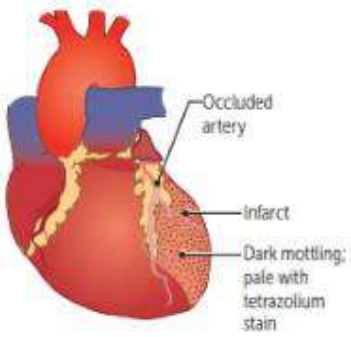
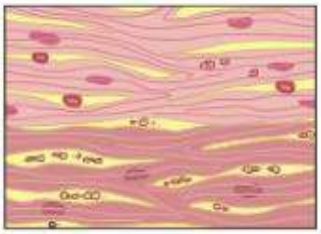
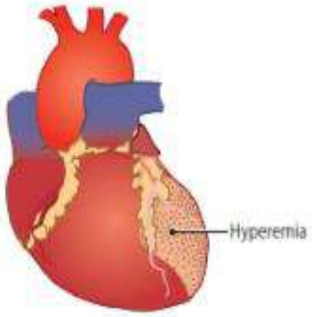
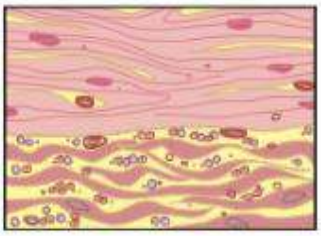
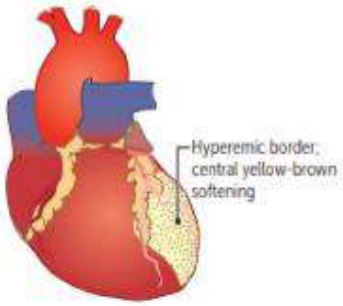
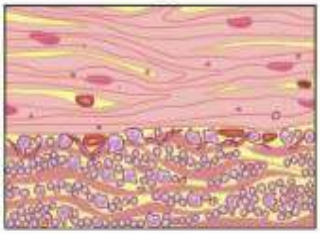
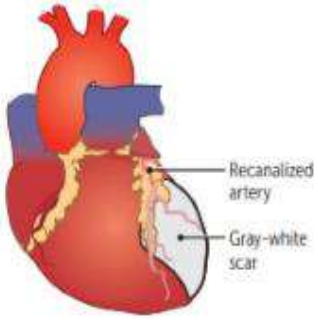
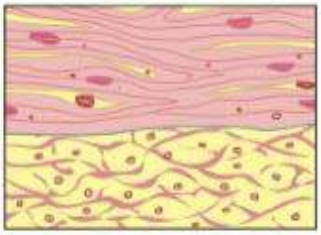
♣ MI is diagnosed by:

clinical symptoms, laboratory tests & characteristic electrocardiographic changes.

➢ Chest pain:
   Prolonged (more than 30 minutes) described as crushing, stabbing, or squeezing, associated with a rapid, weak pulse; profuse sweating (diaphoresis), and nausea and vomiting are common.

➢ Dyspnea:
   ✓ due to impaired contractility of the ischemic myocardium and the resultant pulmonary congestion and edema is a frequent symptom.
   ✓ Pain may be absent in patients with diabetic neuropathy.

➢ The laboratory texts:
   elevation of Cardiac specific troponins and cardiac fraction of creatine kinase.

Time	Gross	LM	Complications
0 – 1 d. (0-24 hr.)	None 	Early coagulative necrosis, release of necrotic cell contents into blood; edema, hemorrhage, wavy fibers. Neutrophils appear. Reperfusion injury, associated with generation of free radicals, leads to hypercontraction of myofibrils through ↑ free calcium influx. 	Ventricular arrhythmia, HF, cardiogenic shock.
1 – 3 d.		Extensive coagulative necrosis. Tissue surrounding infarct shows acute inflammation with neutrophils. 	Postinfarction fibrinous pericarditis.
3 d. – 2 w.		Macrophages, then granulation tissue at margins. 	Free wall rupture → tamponade; papillary muscle rupture → mitral regurgitation; interventricular septal rupture due to macrophage-mediated structural degradation. LV pseudoaneurysm (risk of rupture).
2w.to several		Contracted scar complete. 	Dressler syndrome, HF, arrhythmias, true ventricular aneurysm (risk of mural thrombus).

GROSS:

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The earliest naked eye changes occur, when the affected muscle appears pale and swollen.

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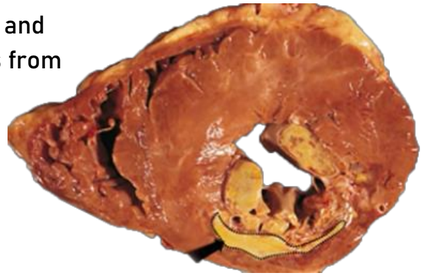
- there is softening and
- the color changes from dark red to brown to yellow.



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the infarct is more sharply defined by the development of a peripheral red zone of granulation tissue.

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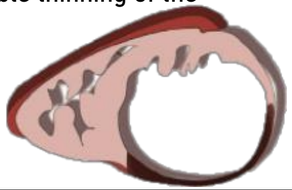
Removal of dead myocytes by phagocytosis and organization proceeds with variable thinning of the infarcted area and some fibrosis.



MICROSCOPIC:

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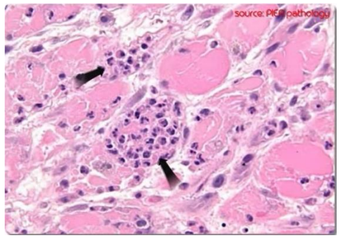
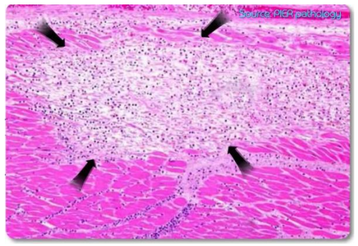
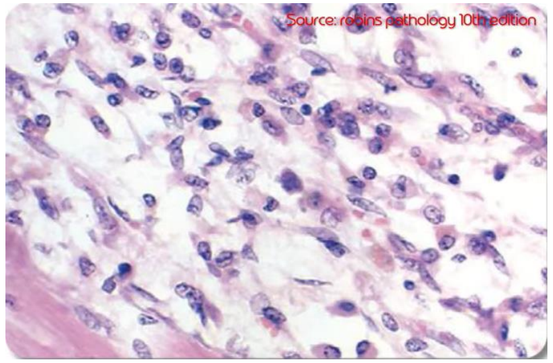




after a few days' digestion by macrophages and organization with granulation tissue formation occurs at the periphery



COMPLICATIONS

1. Contractile dysfunction and some degree of heart failure with pulmonary congestion & edema.
   Large infarcts can cause cardiogenic shock.

2. Arrythmias:
   due to myocardial irritability and affection of the conduction system in the atrioventricular septum
   - Tachycardia,
   - Heart Block,
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   fibrinous or fibrin hemorrhagic pericarditis usually develops about the 2nd or 3rd day following a transmural infarct as a result of underlying myocardial inflammation

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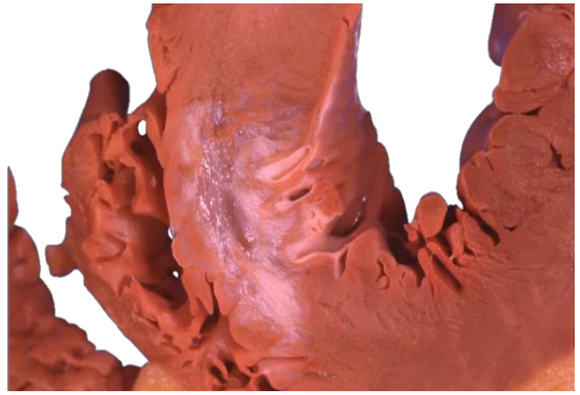
a thin surviving subendocardial band of myocytes is almost invariable, these cells being nourished by blood in the ventricular lumen.

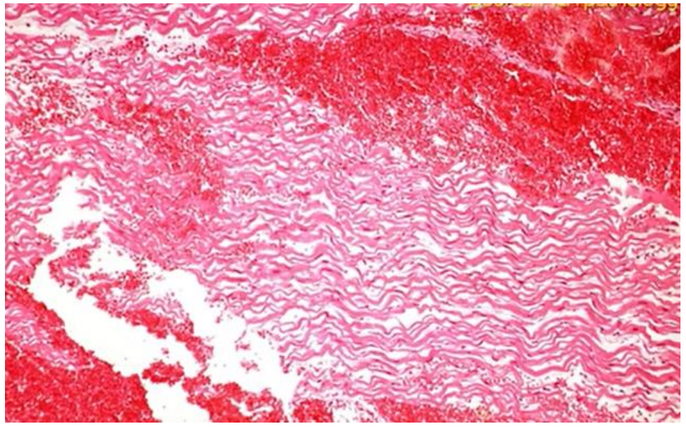


➢ mural thrombus:

often forms on the endocardial surface overlying the infarct after several days.

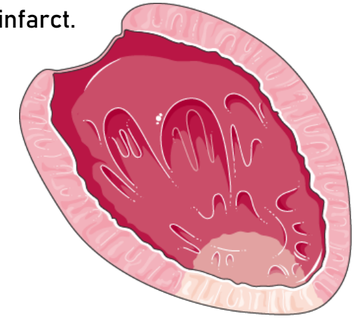
➢ Over the next 2-3 months:

the infarcted myocardium is eventually replaced by a fibrous scar.

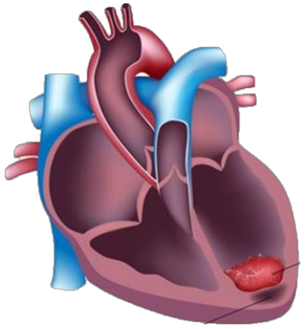


➢ ventricular remodeling:

Hypertrophy of the non-infarcted myocardium with chamber enlargement then compensate for loss of contractility caused by the healed infarct.



6. Mural thrombosis: Due to stasis and endocardial damage.

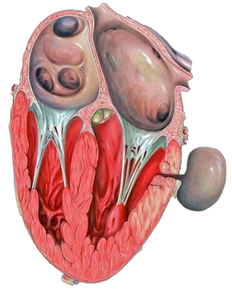


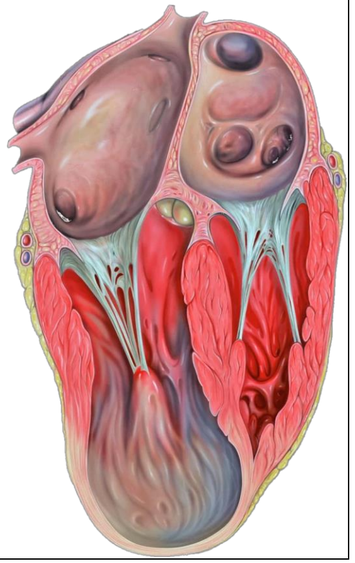
7. Ventricular aneurysm:

It is a late complication due to weak scar that bulges during the systole.

Complications of ventricular aneurysms include

- mural thrombus,
- arrhythmias,
- heart failure, &
- occasionally rupture.





8. Progressive late heart failure (CIHD).

MYOCARDIAL DISEASES

MYOCARDITIS

♣ Definition: Inflammation of the cardiac muscle.

♣ Causes:

1) Acute rheumatic carditis.
2) Viral infections:

   especially Coxsackie virus, but also adenovirus, influenza and HIV.
3) Bacterial infections:

   e.g., diphtheria, clostridia
4) Parasitic infections:

   particularly Chagas' disease Trypanosoma cruzi
5) ionizing radiation.
6) Drugs:

   especially doxorubicin. (Chemotherapeutic agent)
7) Myocarditis can complicate infective endocarditis

   and in some cases, myocardial abscesses form around valve rings

♣ Clinical features:

- In most patients, myocarditis is a self-limiting condition with only mild chest pain and fatigue.
- Cardiac magnetic resonance imaging is a valuable new diagnostic. technique.
- Fatalities are relatively uncommon. In some patients, cardiac failure develops and coronary angiography may be performed to exclude coronary artery disease.
- An endomyocardial biopsy is sometimes performed and may show lymphocytic infiltration and myocyte necrosis.

CARDIOMYOPATHY

♣ Definition:

Cardiomyopathies are a heterogeneous group of myocardial diseases associated with mechanical and/or electrical dysfunction of the heart.

♣ Aetiology:

- ✓ There are a large number of underlying causes, many of which are genetic, and inheritable.
- ✓ In most cardiomyopathies the disease process is confined to the heart but some are part of generalized systemic diseases.

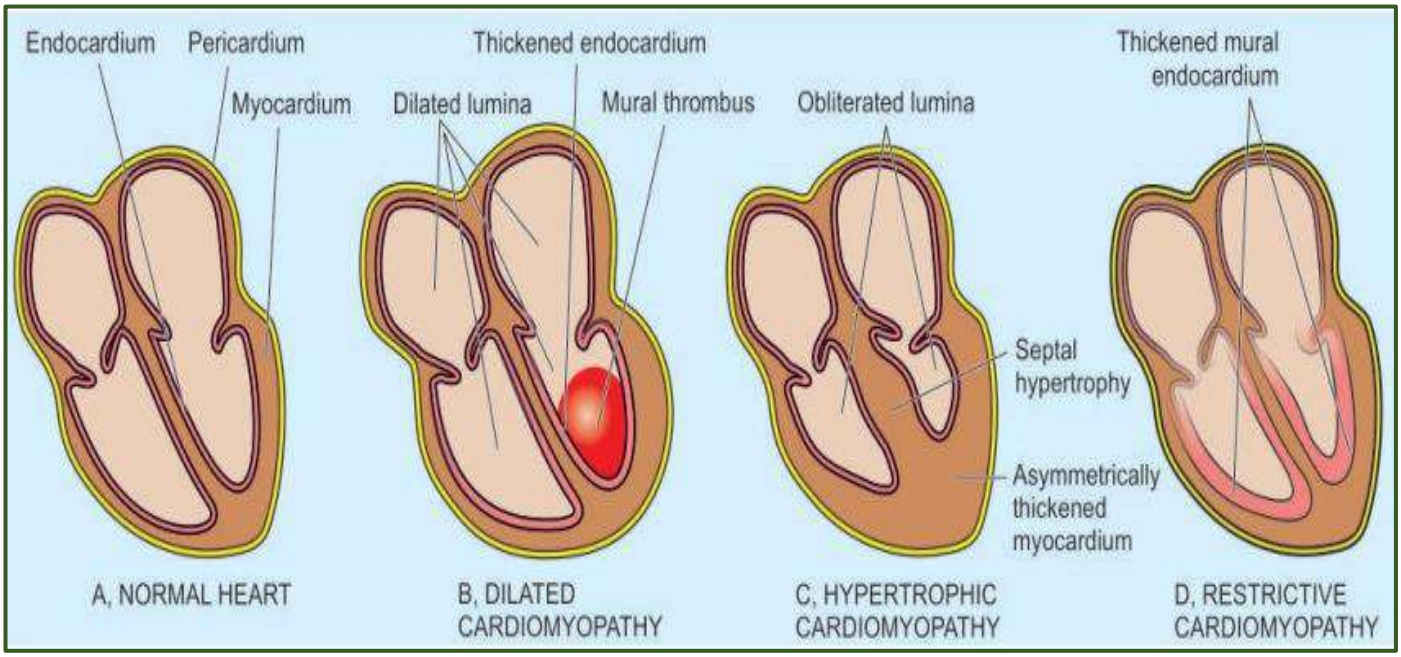
♣ Types:

1) Dilated cardiomyopathy (DCM) [Most common type.]
2) Hypertrophic cardiomyopathy
3) Restrictive cardiomyopathy
   (In systemic diseases as amyloidosis, sarcoidosis, scleroderma)

♣ Diagnosis:

♣When cardiomyopathy is suspected:

it is essential that coronary artery disease, valvular disease, hypertension and congenital heart disease are first excluded.



PERICARDIAL DISEASE

PERICARDITIS

♣ Definition:

An inflammatory reaction involving the visceral and/or parietal pericardial layers.

♣ Types and causes:

1. Fibrinous pericarditis:
   - ✓ Caused by:

   a) Rheumatic fever b) Viral Uremia c)Overlying myocardial infarction

   - ✓ heals by:

   resolution or adhesions between visceral and parietal layer.

2. Suppurative pericarditis:
   - ✓ caused by:

   staphylococci and streptococci, due to spread from intrathoracic focus.

   - ✓ heals by adhesions.

3. Hemorrhagic pericarditis & hemopericardium:

Inflammation associated with a bloody exudate in the pericardial cavity

- ✓ caused by:

  a) Surgery b) Trauma c) Tumor metastases

N.B: Cardiac tamponade:
blood collecting suddenly & in large quantities in the pericardial sac,
resulting in sudden death (acute heart failure).
It occurs in rupture myocardial infarction, ventricular aneurysm or rupture aortic aneurysm.

4. Tuberculous pericarditis:
   - ✓ due to:

- post primary spread or as part of secondary tuberculosis.
- The pericardial sac is filled with caseous material and fibrous adhesions.
- Extensive fibrosis and calcification results in constrictive pericarditis.

5. Constrictive pericarditis:

- The pericardium is transformed into a thick layer of fibrous tissue with calcification which restricts diastolic filling and causes systemic venous congestion.
- The heart is small in size.
- This is the end result of caseous and severe suppurative pericarditis.

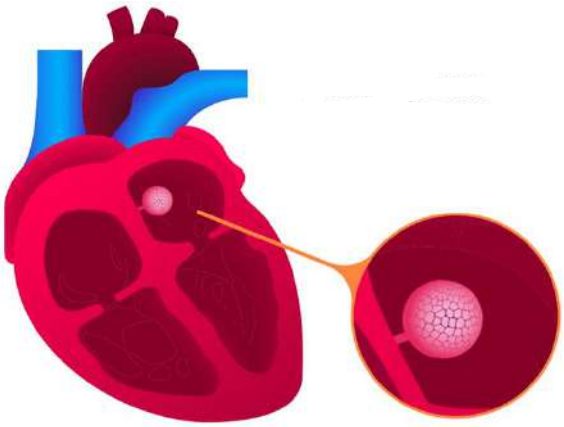
6. Adherent mediastino-pericarditis:

- severe fibrosis between the 2 layers of pericardium and between outer pericardial layer and mediastinum.
- Each ventricular contraction, the heart pulls on the mediastinal structures
- which causes overload on the heart leading to ventricular hypertrophy, dilatation and failure.

TUMORS OF THE HEART

1) Left atrial myxoma:

- Benign tumor although some consider it a myxomatous change in a ball thrombus.



2) Rhabdomyoma:

- benign tumor of striated muscle.

3) Rhabdomyosarcoma :

- The malignant counterpart of rhabdomyoma.

4) Metastasis:

Rare, from lung or melanoma.

Route; direct or lymphatic spread to pericardium,

as in bronchogenic carcinoma or

blood borne to the muscle, as

in melanoma metastasis.

HEART FAILURE

♣ Definition:

A condition where the cardiac output is insufficient to meet the metabolic demands of the tissues.

♣ Pathophysiology: (see physiology book)

Acute and chronic failure

➢ Acute heart failure:
can occur within minutes of a myocardial infarction, with sudden onset of shortness of breath and marked pulmonary edema.

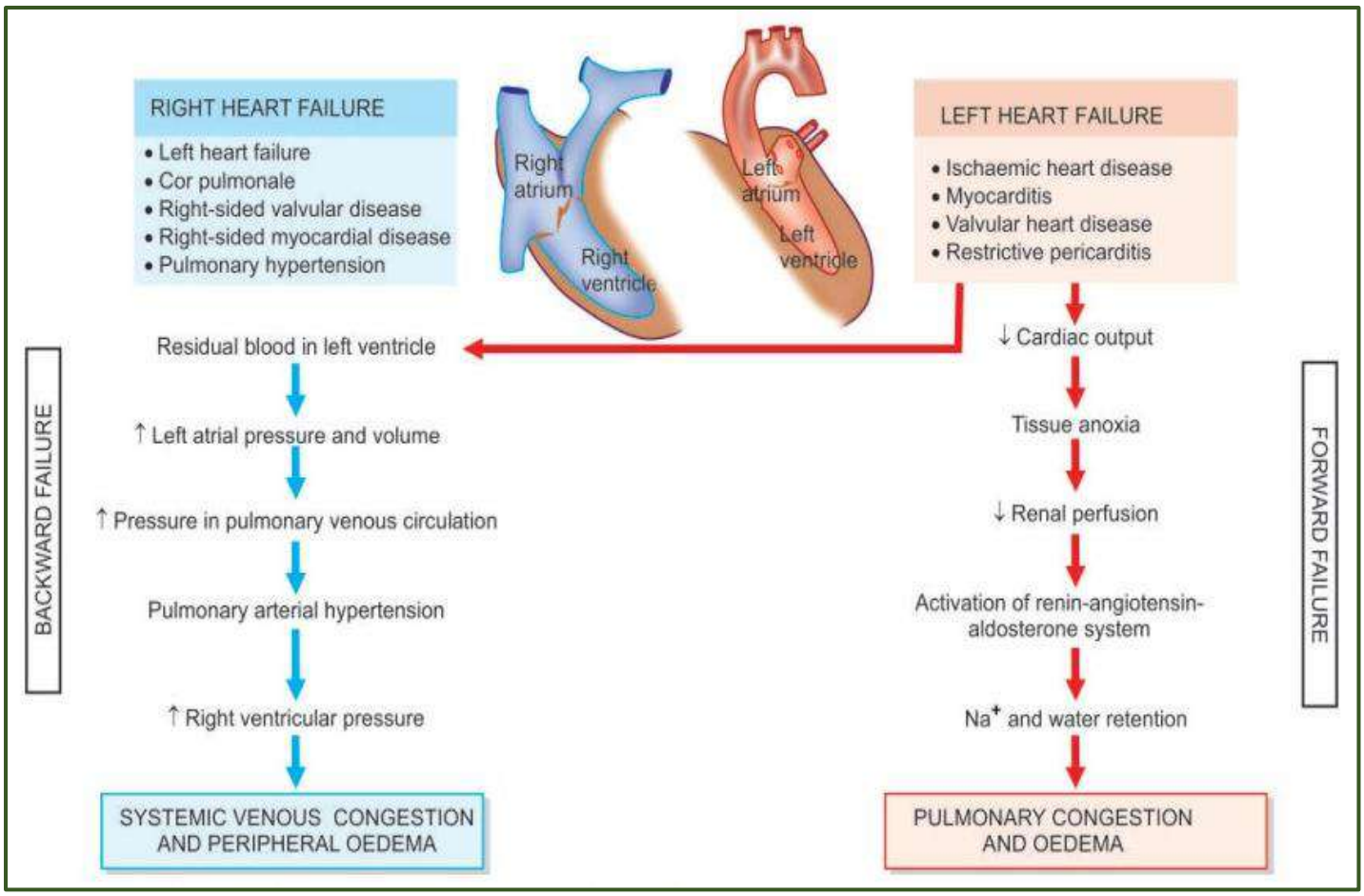
➢ Chronic heart failure:
develops over many years in valvular defects such as mitral stenosis or incompetence. symptoms develop over many years with gradual worsening of symptoms.

Right and left heart failure:

♣The right and left ventricles function together in a closed circuit.

♣In early stages of heart failure, symptoms appear "one-sided".

	left sided heart failure	right sided heart failure
Causes	✓ Consequence of left side failure ✓ Mitral stenosis ✓ Cor pulmonale (explain)	✓ Ischemic heart disease ✓ Systemic hypertension ✓ Valvular heart disease [mitral regurge, aortic stenosis or regurge]
Effect	systemic venous congestion, enlargement of the liver.	pulmonary congestion and edema.



N.B:

♣ Cor pulmonale:

right side heart failure secondary to lung disease as chronic obstructive lung disease (chronic bronchitis and emphysema) and lung fibrosis.

♣ In congestive cardiac failure there is both right and left ventricular failure and a full combination of systemic and pulmonary signs.

♣ Clinicopathological Features of heart failure:

1. The major symptoms and signs of heart failure:-

1) Dyspnea:

due to pulmonary congestion and edema. Orthopnea is dyspnea on lying flat due to increased venous return

Paroxysmal nocturnal dyspnea (sudden and urgent shortness of breath during sleep: probably due to increased pulmonary venous congestion.

2) Systemic venous congestion and edema :

- ✓ Fluid retention, a compensatory mechanism in heart failure, results in volume overload on the ventricles.
- ✓ Veins are the reservoir for the increased blood volume, and there is wide spread congestion of systemic veins. There is distension of superficial jugular vein.
- ✓ The liver is enlarged with engorgement of central veins and hepatic sinusoids.
- ✓ Accumulation of fluids in subcutaneous tissues (edema) and in the pleural, pericardial and peritoneal cavity, due to increased transudation of fluids as a result of high venous pressure.

2. Other changes:-

1) Proteinuria.
2) Nausea, vomiting.
3) Lethargy, headache, and difficulty sleeping due to decreased cerebral blood flow.