1. HYPERTENSION
2. ISCHAEMIC HEART DISEASE
3. VARICOSE VEIN AND ANEURYSM

ILOs

- After the lecture, students should be able to:

➢Define **systemic hypertension**, enumerate its **causes** and mention its **types.**

➢Describe **the effects** of systemic hypertension, particularly on the vessels, heart, kidney and brain and list

➢**causes of death** in patients affected with systemic hypertension.

➢-Define **secondary hypertension** and list **its causes**.

SYSTEMIC HYPERTENSION

❑*Definition:*

➢Persistent/sustained **elevation** of the systolic and diastolic blood pressure above 140/90 mm of mercury in adult person due to ↑ peripheral resistance in the arterioles.

❑ *Clinical classification of hypertension:*

CATEGORY	SYSTOLIC (mmHg)	DIASTOLIC (mmHg)
Normal	< 120	< 80
Prehypertension	120-139	80-89
Hypertension Stage 1 Stage 2	 140-159 >160	 90-99 >100
Isolated systolic hypertension	>140	< 90
Malignant hypertension	> 200 (sudden onset)	> 140 (sudden onset)

SYSTEMIC HYPERTENSION

❑Types of systemic hypertension

I. Primary or essential hypertension:

- **Common (95%)** and is of two types:
  A. Benign essential hypertension 90%
  B. Malignant essential hypertension 10%

II. Secondary hypertension:

- **Uncommon (5%).** It is also classified as:
  A. Benign 80%
  B. Malignant 20%

ESSENTIAL HYPERTENSION

❑Etiology:

• ***Genetic factors:***

Essential hypertension often run in families.

• ***Sodium intake:***

The disease is more common in populations with high sodium intake.

• ***Neurogenic factors:***

Stress and emotional stimuli increase the sympathetic tone.

• ***Humoral vasoconstrictor factors:***

In renal hypertension the ischemic kidney releases renin by the Juxtaglomerular cells.

A. Benign essential hypertension

- A common disease which occurs above the age of **40** years.
- It is characterized by a slowly **progressive rise** in the blood pressure and a long course for **30** years or more.
- **Lifestyle factors** that increase the risk include excess salt and body weight, smoking and alcohol.

❑ ***Pathological Lesions:***

I. Vascular Lesions (benign arteriolosclerosis):

- It affects **small arteries** and **arterioles** less than 100 micron in diameter.
- The changes occur in the vessels of the kidney, brain and retina.
- The lesion takes a **patchy distribution**

a. ***Hyalinosis:***

- Hyaline change in the intima and media of the small arterioles leading to thickening of the wall and narrowing of the lumen.

b. ***Elastosis:***

- Hyperplasia and splitting of the internal elastic lamina into several layers affects the larger artetioles and small arteries.

II. Kidney Lesions (Benign nephrosclerosis):

❑ ***Gross picture:***

- In long standing cases of hypertension both kidneys are small, contracted and firm **(primary contracted kidney)**.
- **The capsule** is adherent.
- The outer surface is finely granular and shows small retention cysts.
- **The cut surface** shows irregular atrophy and fibrosis of the cortex with loss of demarcation between the cortex and medulla.
- The arteriole are thick walled and prominent.

❑ ***Microscopic picture:***

1. The **afferent and efferent arterioles** show **hyalinosis** and **elastosis.**
2. **The glomeruli** show thickening of the glomerular basement membrane due to ischemia followed by atrophy, fibrosis and hyalinosis of the whole glomerulus.
3. **The tubules** attached to the fibrosed glomeruli undergo **atrophy** and **fibrosis**, while the tubule, attached to the functioning glomeruli show compensatory dilatation and may form small retention cysts.
4. **The interstitial tissue** is increased and shows lymphocytic infiltration.

III. Cardiac Lesions (Hypertensive Cardiomyopathy):

a. **Concentric hypertrophy** of the left ventricle due to the high resistance caused by the elevated blood pressure.

- The wall becomes thickened, 2 cm or more.

- **The papillary muscles** show hypertrophy and **the septum** is thickened and bulges into the right ventricular cavity.

- Next the left atrium undergoes hypertrophy.

- The hypertrophy enables the heart to remain in a state of compensation for a long time.

b. Eventually decompensation occurs.

The myocardium starts to **fail** and the cavities of the left side dilate.

IV. Retinal Lesions:

a. Arteriolosclerosis of the retinal arterioles with compression of the veins.

b. Retinal hemorrhages.

V. Cerebral Lesions:

- **Micro-aneurysms** occur in the small cerebral arteries which may rupture causing massive cerebral hemorrhage.

Causes of Death:

1. Left sided heart failure
2. Cerebral hemorrhage
3. Chronic renal failure (uremia)

B. MALIGNANT Essential HYPERTENSION

- A rare disease affecting mainly the **young adults**.
- It is characterized by a **rapid** progressive rise in the blood pressure and a short fatal course.

❑Pathological Lesions:

I. Vascular lesions (Malignant Arterioloscierosis):

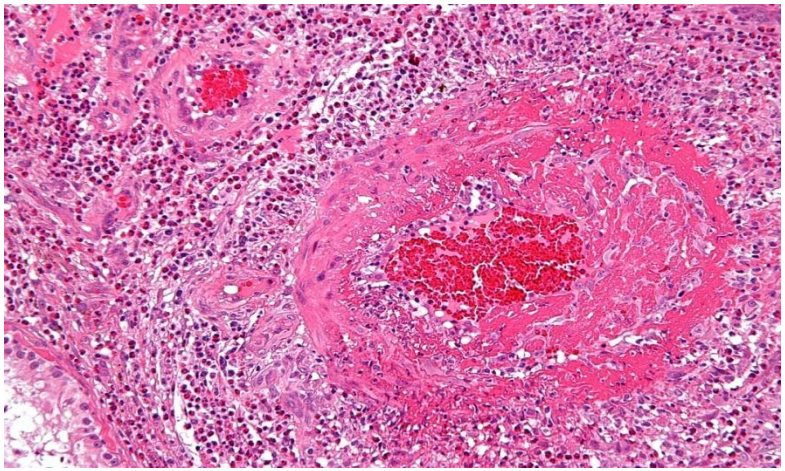
a. ***Arteriolar hyalinosis and elastosis:*** Are less marked than in benign hypertension due to the short course of the disease.

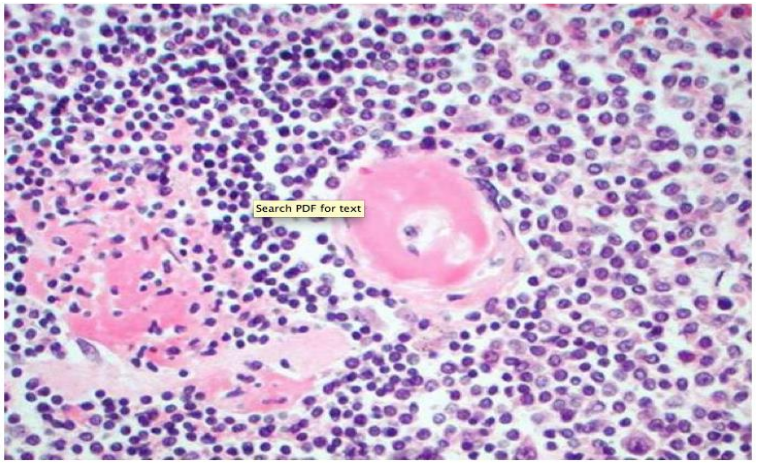
b. Cellular hyperplasia:

- **Concentric hyperplasia** of the subendothelial connective tissue and smooth muscles of the media of small arteries and large arterioles.
- The vessel becomes narrowed and its wall thickened and shows the ***onion-skin*** appearance.

c. Acute arteriolar necrosis:

- ***Fibrinoid necrosis*** in the wall of the arterioles and small arteries mainly in the kidney, brain and retina (**hallmark of malignant hypertension).**
- **The vessel wall** appears thickened, necrotic and infiltrated by fibrin, RBCs and PMLs.
- **The lumen** is reduced and often show thrombosis.





Picture 11: Fibrinoid necrosis ที่ผนังหลอดเลือด

II. Kidney Lesions (Malignant Nephrosclerosis):

❑ ***Gross picture:***

- The kidneys are of **normal size**.
- **The capsule** strips easily.
- **The outer surface** is smooth and shows focal hemorrhages.
- **The vessels** are prominent, thick and narrow.
- If malignant hypertension occurs on top of pre-existing benign hypertension, the kidney is **reduced in size**, granular and shows focal hemorrhages on the surface.

❑ ***Microscopic picture:***

a. **Necrosis** of the afferent arterioles and glomerular capillaries.

b. **Hemorrhage** in the Bowman's capsules.

c. **Cellular hyperplasia** in the small arteries.

d. Glomerular hyalinosis and tubular atrophy or dilatation may be present.

III. **Cardiac Lesions:**

- Slight hypertrophy of the left ventricle.

IV. **Retinal Lesions:**

- Retinal hemorrhages.

V. **Cerebral lesions:**

- Arteriolar necrosis causing massive cerebral hemorrhage may complicate malignant hypertension.

Causes of Death:

1. Acute renal failure in 95 % of cases.
2. Cerebral hemorrhage.
3. Left sided heart failure.

Secondary hypertension

- **causes of secondary hypertension:**

1. **Renal causes (the commonest):**
   A. Acute glomerulonephritis
   B. Chronic glomerulonephritis
   C. Chronic pyelonephritis
   D. Renal artery stenosis
   E. Renin-producing tumors

2. **Endocrine causes:**
   A. Cushing's syndrome
   B. Myxedema
   C. Thyrotoxicosis
3. **Vascular causes:**
   A. Coarctation of aorta
   B. Aortic insufficiency
4. **Neurogenic causes:**
   A. Psychogenic
   B. Increase Intracranial Tension
   C. Polyneuritis, poliomyelitis

ISCHAEMIC HEART DISEASE

❖ **ILOs:**

- **After the lecture, students should be able to:**

1. List the risk factors of **ischemic heart disease** and enumerate cause and effects of coronary occlusion.
2. Discuss sites, macroscopic changes and relate these to histological changes of **myocardial infarction**.
3. List the possible **complications** of myocardial infarction.

ISCHAEMIC HEART DISEASE

- ***Definition:*** acute or chronic form of cardiac disability arising from imbalance between the myocardial supply and demand for oxygenated blood.

Ischemic heart diseases (IHD): are the leading cause of death in most industrialized countries.

- Death rates from IHD ↑ with age, which affects men earlier and more common than women.
- The incidence in women ↑ after menopause.
- As atherosclerosis of coronary artery is the main cause of IHD, so, **predisposing (risk) factors of IHD** are those of atherosclerosis.

- **Myocardial ischemia** results from imbalance between myocardial oxygen supply and demand.

- **This imbalance results from:**

A. **Decreased coronary blood flow (90%)**

B. **Increased myocardial demand exceeding vascular supply**

C. **Decreased oxygen carrying capacity of the blood**

A. Decreased coronary blood flow:

➢Causes of Coronary Occlusion:

1. Coronary atherosclerosis: Is the main cause of sudden coronary occlusion (90% or more). **The mechanisms of occlusion are:**

- (a) *Thrombosis:* Due to roughness of the intima and narrowing of the lumen.
- (b) *Intimal haemorrhage:* The area of atheroma shows formation of new wide capillaries. They are derived from the intimal endothelium. The pressure in the coronaries is reflected to these capillaries which rupture specially in cases with hypertension. **The rupture causes:**

- A haematoma in the subendothelial connective tissue causing enlargement of the atheromatous nodule and occlusion of the vessel.
- Release of thromboplastic substances from the necrotic intima and the blood cells causing thrombosis.

2. Coronary embolism: A rare cause for coronary occlusion. The embolus is derived from vegetations of subacute bacterial endocarditis, mural thrombosis in the left ventricle or a thrombus over an atherosclerotic lesion at the root of the aorta.

3. Syphilitic aortitis: Causing narrowing of the coronary orifice followed by thrombosis.

4. Dissecting aneurysm: When it involves the coronary ostium.

5. Impaired coronary flow: As in shock and severe aortic stenosis produces the effects on coronary occlusion

- **Results of Coronary Occlusion:**

(I) Sudden death due to ventricular fibrillation.

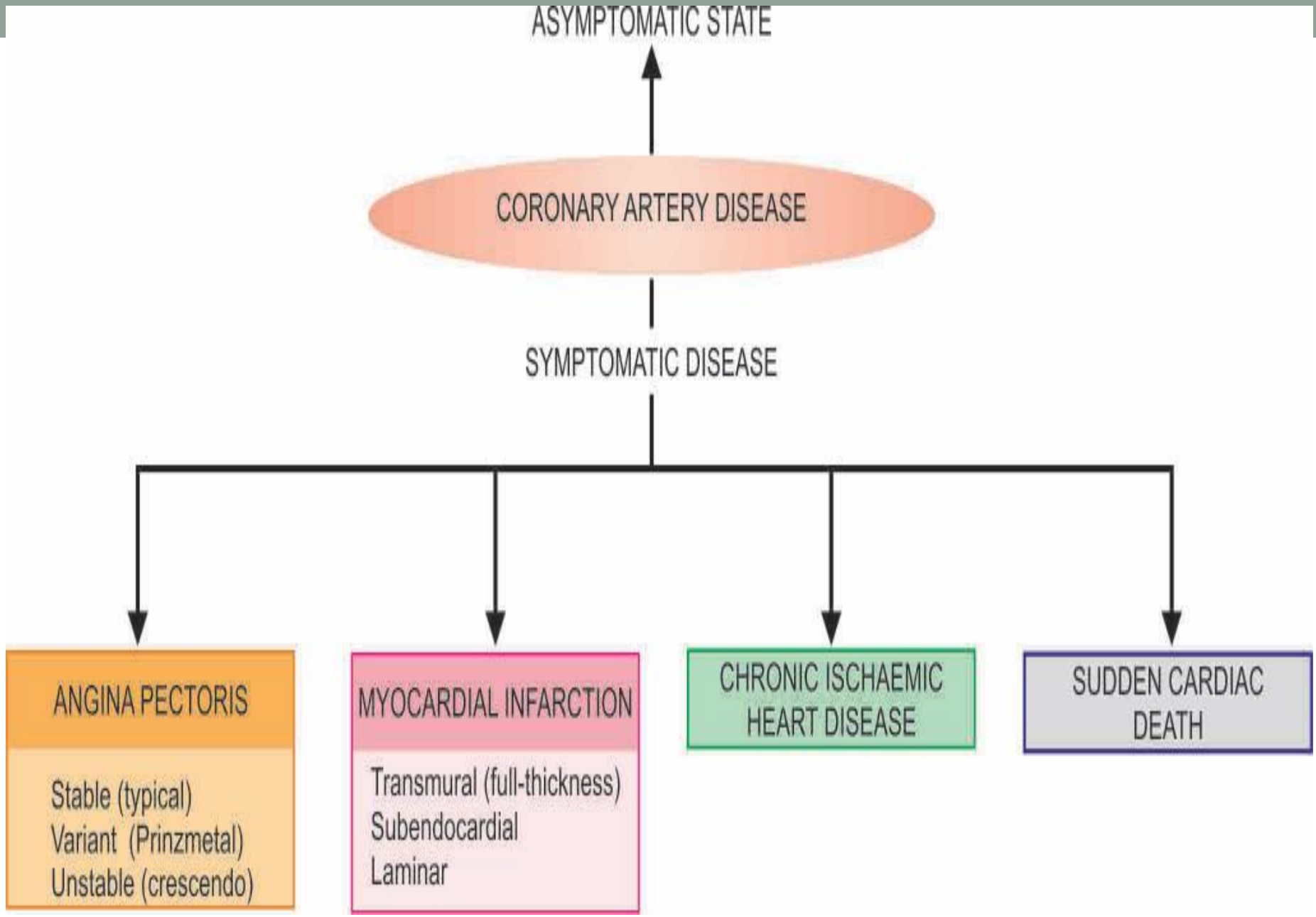
(2) Myocardial infarction.

B. **Increased myocardial demand exceeding vascular supply in case of e.g.**

1. Severe exercise and tachycardia
2. Hypertension and cardiac hypertrophy
3. LV hypertrophy

C. **Decreased oxygen carrying capacity of the blood e.g.**

1. Severe anemia
2. Advanced lung diseases, as in severe pneumonias
3. Congenital cyanotic heart disease


ASYMPTOMATIC STATE
CORONARY ARTERY DISEASE
SYMPTOMATIC DISEASE
ANGINA PECTORIS
Stable (typical)
Variant (Prinzmetal)
Unstable (crescendo)
MYOCARDIAL INFARCTION
Transmural (full-thickness)
Subendocardial
Laminar
CHRONIC ISCHAEMIC HEART DISEASE
SUDDEN CARDIAC DEATH

Angina Pectoris

- Angina pectoris is a clinical syndrome of IHD resulting from transient myocardial ischaemia.
- Angina is an **episodic chest pain** of variable severity often gripping, compressing or crushing.
- It is due to **transient reversible myocardial ischemia**.
- The pain is usually retro-sternal and may radiate to the neck and jaw or to the upper and inner aspect of either or both arms and hands.

❑ *Variants of Angina*

I. Typical Stable Angina:

- This is **the commonest** form of angina.
- It is due to progressive stenosis of the coronary artery by atheroma.
- It is **precipitated by exertion, emotional stress**, and is relieved by rest or sublingual nitroglycerin.
- **Pain** is classically crushing or squeezing substernal and may radiate to the left arm or to the left jaw.

II. Variant Angina "Prinzmetal's Angina":

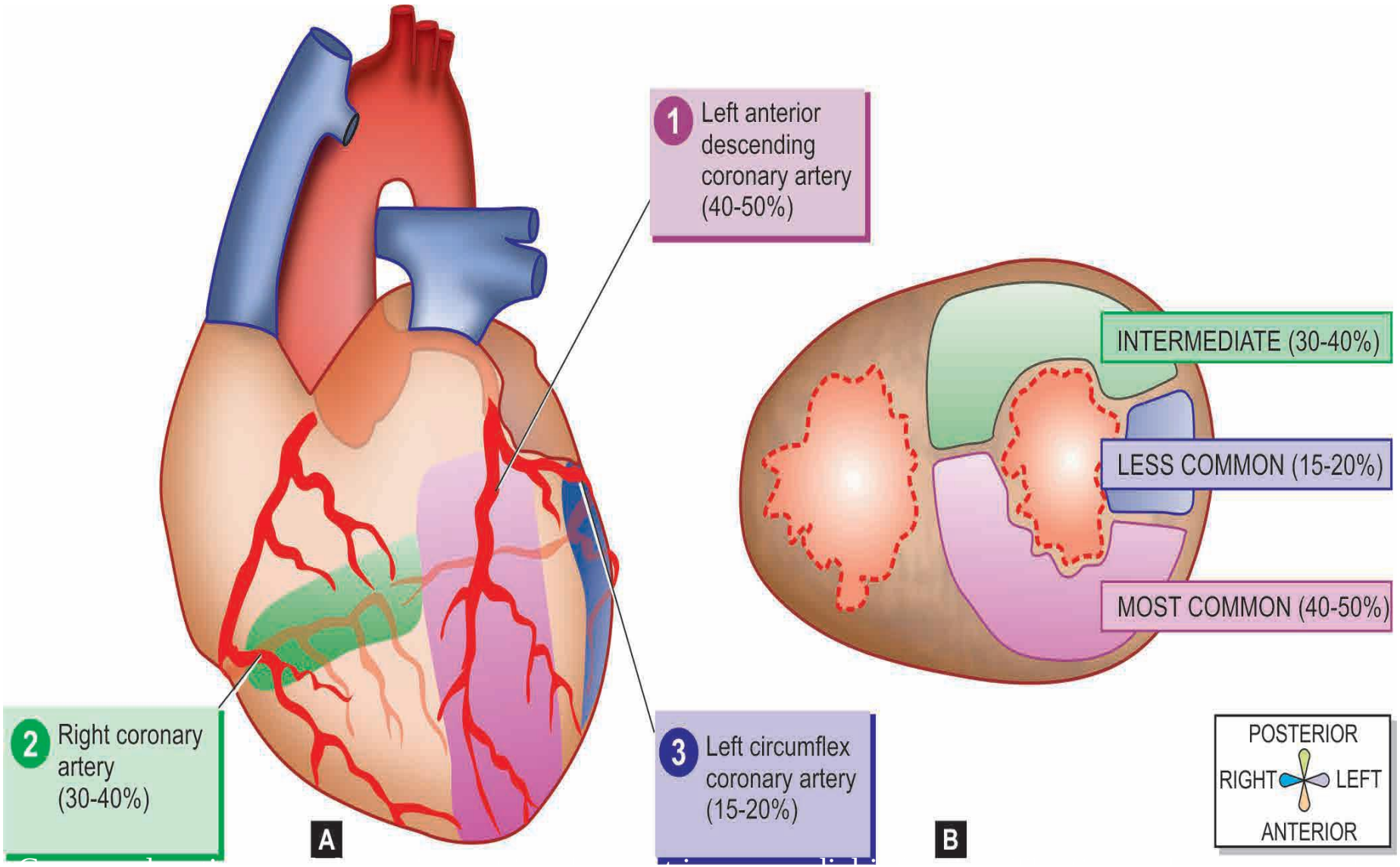
- The attacks of pain are **not** related to exercise and can occur at rest.
- It is due to spasm of large or medium-sized coronary arteries, often at or near the site of atheromatous narrowing.
- In 15% of cases, however, the coronary arteries appear normal.
- The mechanisms of vasospasm are not known.
- Prinzmetal angina typically respond to vasodilators as nitroglycerin and calcium channel blockers.

Myocardial Infarction (MI)

- It is commonly referred to as a “heart attack”.
- **Acute myocardial infarction (MI)** is **the most important consequence** of coronary artery disease. Many patients may die within the first few hours of the onset, while remainder suffer from effects of impaired cardiac function.
- **Risk factors** are the same as those of atherosclerosis.

Sites of the Infarct:

1. Occlusion of the anterior descending branch of the left coronary causes infarction in **the anterior wall of the left ventricle** towards the apex and the anterior portion or the interventricular septum. This is the **commonest site.**
2. Occlusion of the right coronary causes infarction in the **posterior wall of the left ventricle** near the base and the posterior part of the septum. Affection of the right ventricle is minimal or absent.
3. Occlusion of the left circumflex artery causes infarction in **the lateral wall of the left ventricle.**


1 Left anterior descending coronary artery (40-50%)
2 Right coronary artery (30-40%)
3 Left circumflex coronary artery (15-20%)
A
B
INTERMEDIATE (30-40%)
LESS COMMON (15-20%)
MOST COMMON (40-50%)
POSTERIOR
RIGHT
LEFT
ANTERIOR

Gross Picture:

- ***Recent infarct:*** Appears swollen, dark red and friable. Later the color changes to yellowish brown with remains of hemorrhage at the edges. The infarct is covered on the endocardial surface by a mural thrombus which may fragment forming aseptic emboli, and on the pericardial surface by a patch of fibrinous pericarditis.

- ***Healed infarct:*** Appears as a thin greyish white fibrous patch which is weak and inelastic. The rest of the myocardium shows compensatory hypertrophy. The pericardium covering the infarct shows fibrosis and adhesions.

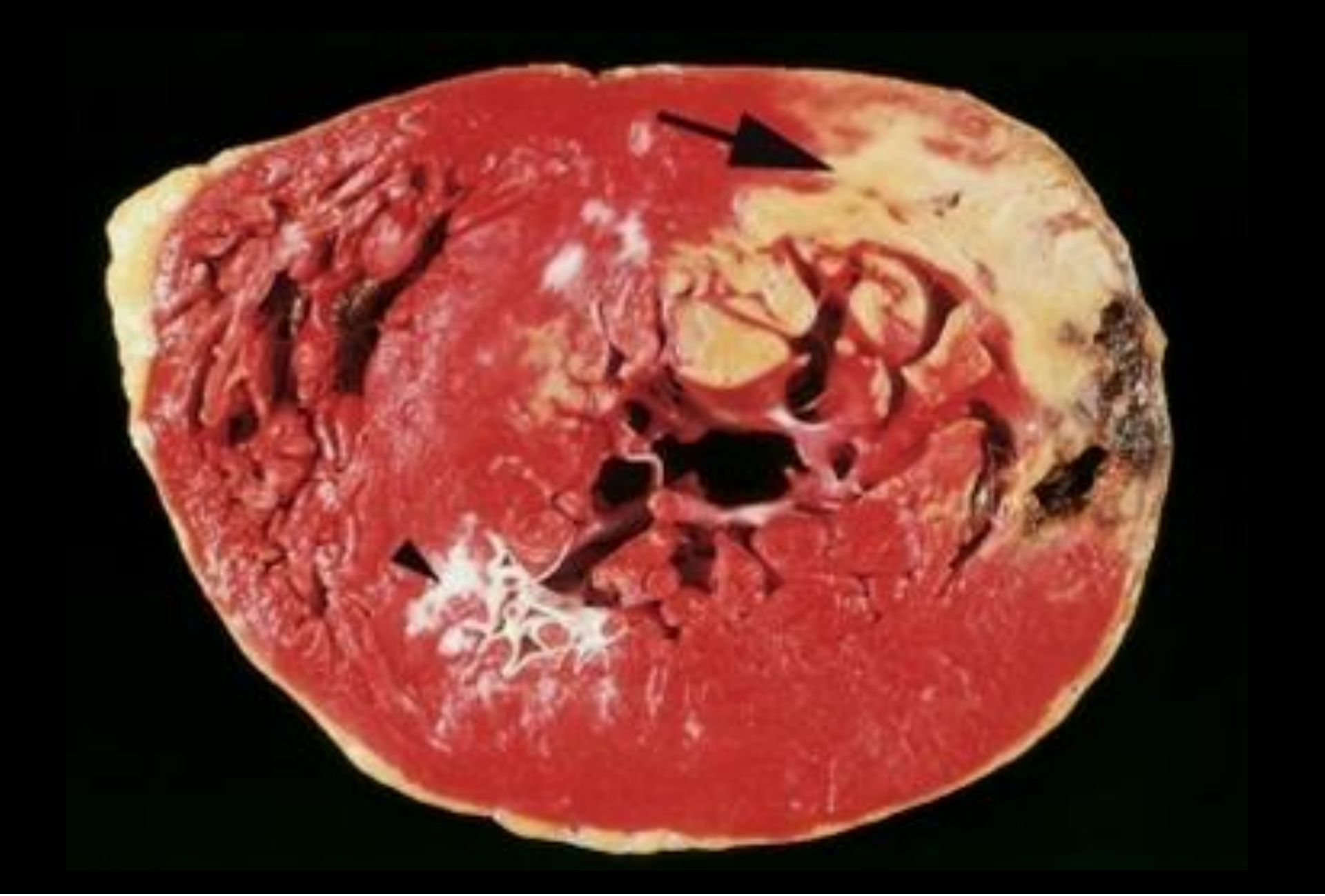
- **Size and rate of development of MI depend on:**
  1. The size and distribution of the affected vessel.
  2. The rate of development and duration of occlusion.
  3. Metabolic demands of the myocardium (affected by blood pressure and heart rate).
  4. Extent of collateral circulation.

❑ Microscopic Picture:

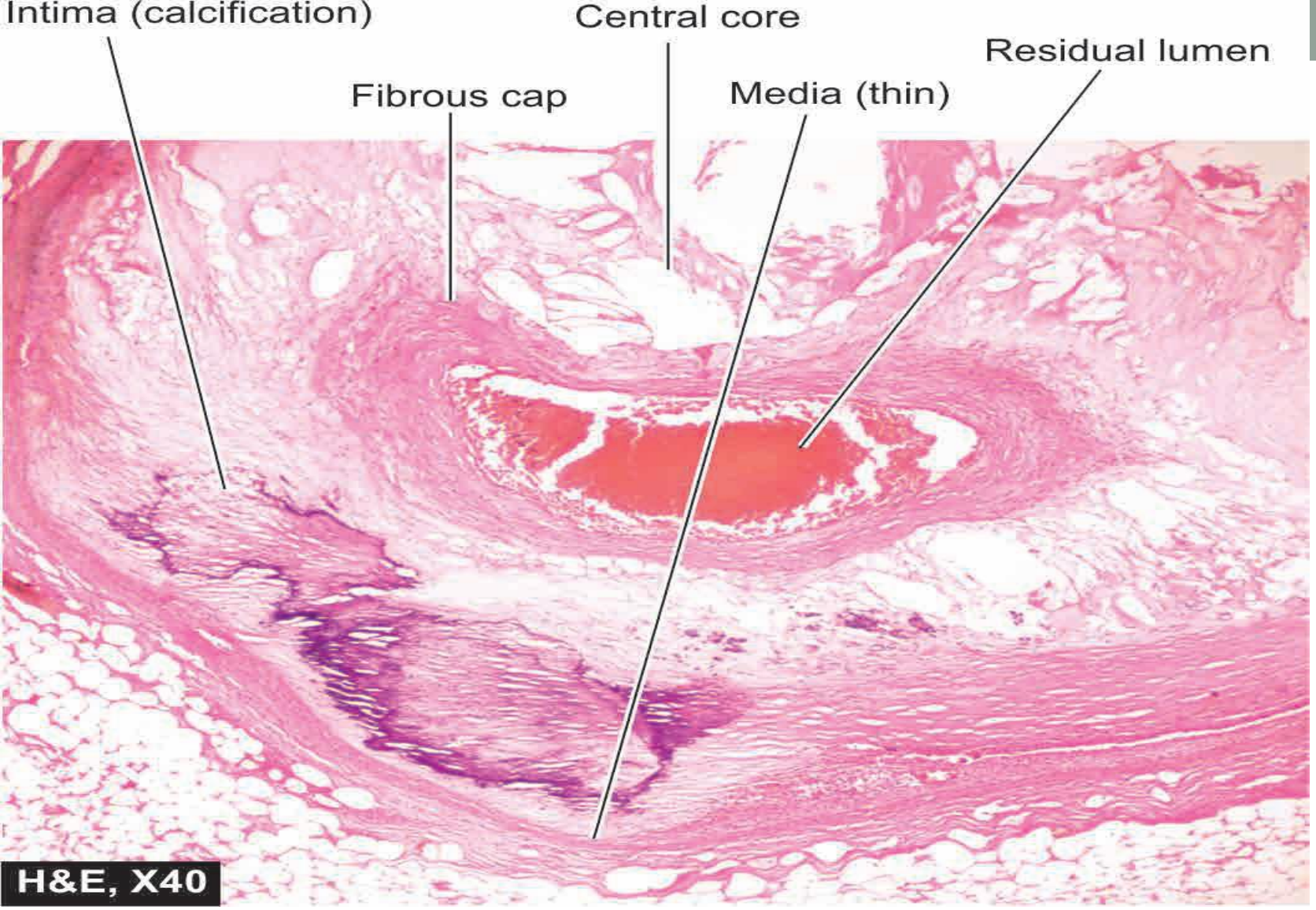
- Microscopic evidence of **coagulative necrosis** appears by the end of 6 hours.
- The cross striations of the muscles and the nuclei disappear.
- The necrotic muscles stain homogenous pale pink with eosin.
- The area shows at first **leucocytic infiltration** followed by the appearance of **macrophages** which start to remove the necrotic tissue.

❑ Fate and Complications:

1. Acute heart failure due lo ventricular fibrillation.
2. Mural thrombosis may give rise to systemic arterial embolism.
3. **The recent infarct** may rupture during exertion causing haemopericardium and sudden death.



Acute Myocardial Infarction


Intima (calcification)
Central core
Residual lumen
Fibrous cap
Media (thin)
H&E, X40

Varicose veins, Aneurysm

ILOs

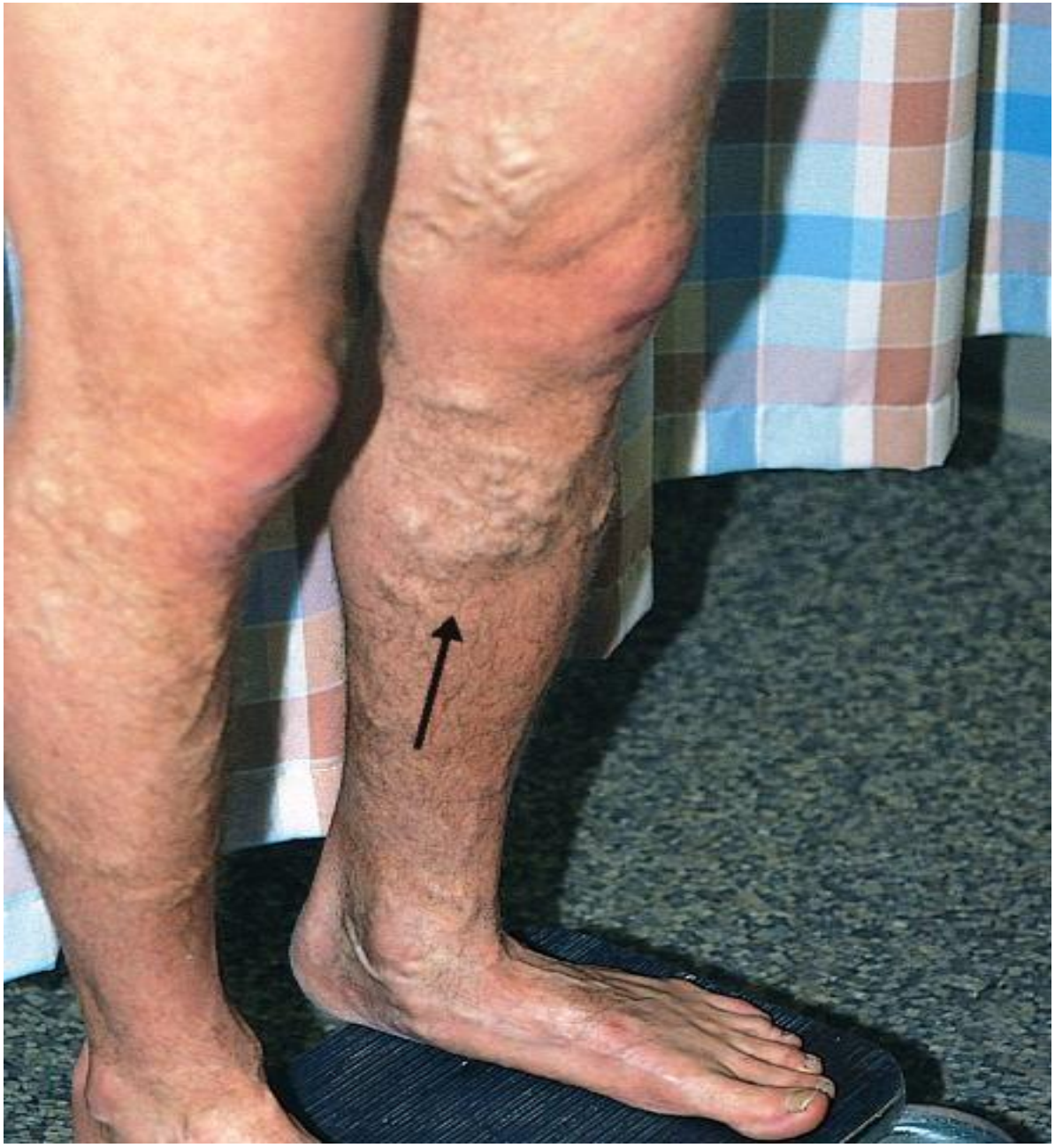
After the lecture, students should be able to:

1. Define **aneurysm** and outline simple classification.
2. Define **varicose veins** and enumerates its common sites.

Varicose veins

- **Definition**: Dilatation , elongation and tortuousity of the veins.
- **Sites:**

1) Superficial veins in lower limbs specially the saphenous
2) Veins at the lower end of the esophagus (esophageal varices)
3) Veins at the lower end of the rectum (piles or hemorrhoids)
4) Pampiniform plexus of the spermatic cord (varicocele)



Causes:

1)**Deficient support** to venous wall either congenital or caused by obesity and senility

2)**Congenital defect** in venous muscular coat

3)**Incompetence** or rupture of the valves in the veins

4)**Increased pressure i**nside the vein due to:

a)Prolonged standing

b)Venous compression in pregnancy and by uterine ,ovarian, prostatic and rectal tumours.

c)Venous obstruction by thrombosis.

d)Right sided heart failure causing increase in systemic venous pressure.

e)Liver cirrhosis and bilharzial hepatic fibrosis causing portal hypertension.

Complications:

1)Phlebothrombosis causing venous congestion and edema

2)Thrombophlebitis causing pyemia

3)Rupture causing hemorrhage and pigmentation in the surrounding tissue

4)Compression and devitalization of the surrounding tissues causes **a varicose ulcer** as in the lower limbs.

Aneurysm

- ***Definition***
  - Localized dilatation of the wall of an artery
- ***Etiology:***

➢ **weakness of the vascular wall due to connective tissue or smooth muscle abnormality**

  - Atherosclerosis: cause aneurysm in abdominal aorta
  - Hypertension: ascending aortic aneurysm
  - Congenital weakness of the arterial wall: congenital cerebral aneurysm
  - Medionecrosis: causing dissecting aortic aneurysm
  - Trauma: cause false aneurysm
  - Syphilis : cause aneurysm in the thoracic aorta

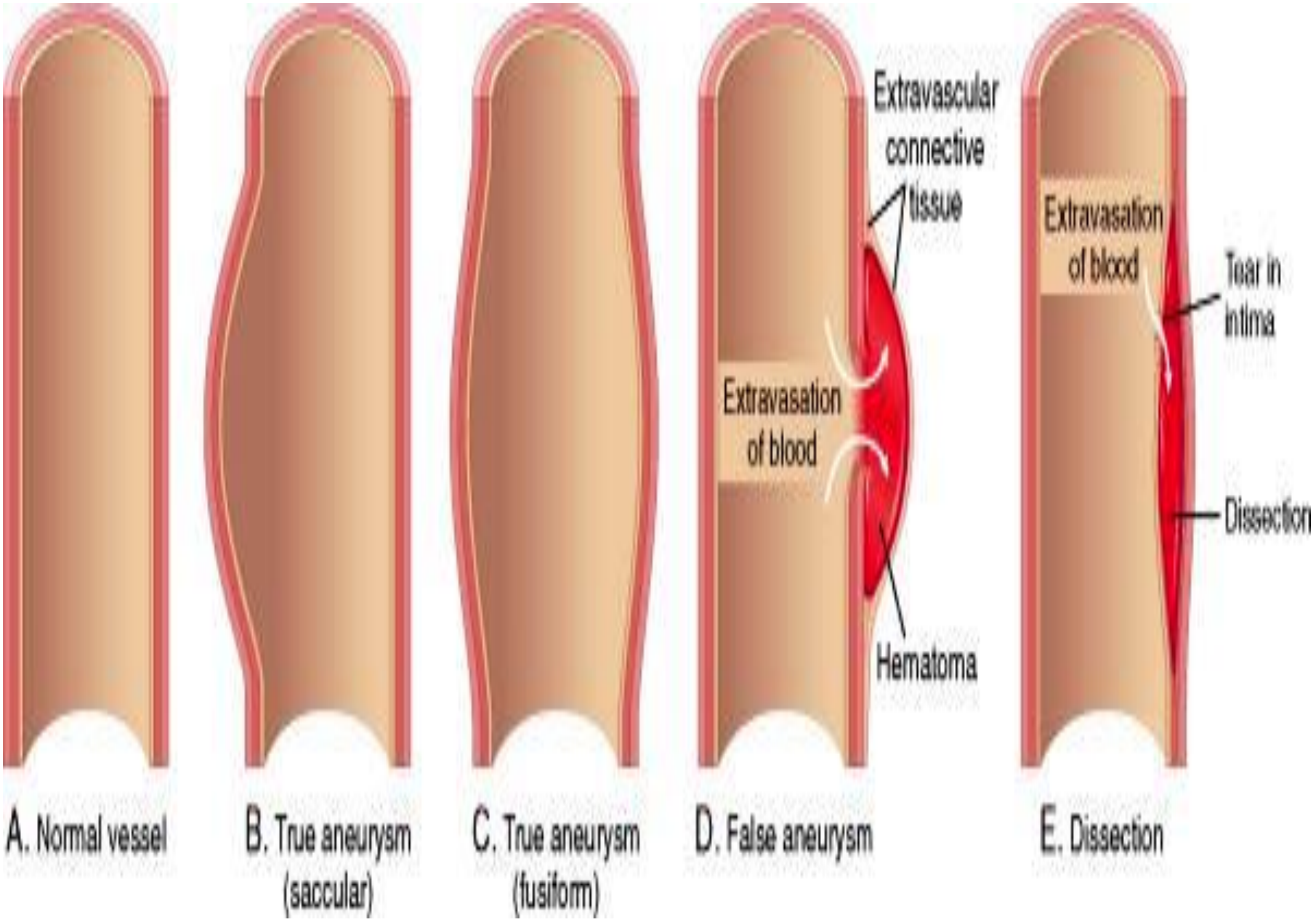
Types

- **True aneurysm:** involve all layers of the vascular wall

According to shape

- Fusiform
- Sacular
- Dissection

- **False aneurysm:** wall of the aneurysm is not part of the arterial wall


Extravascular connective tissue
Extravasation of blood
Hematoma
Extravasation of blood
Tear in intima
Dissection
A. Normal vessel
B. True aneurysm (saccular)
C. True aneurysm (fusiform)
D. False aneurysm
E. Dissection

True aneurysm

- The wall of aneurysm is part of the arterial wall
- Types:

1. Atherosclerotic aneurysm: affect aorta mainly the abdominal part and the main branches in old age. The dilatation affect the whole circumference so the aneurysm **appear fusiform** in shaped. Its rupture causes fatal hemorrhage

2.Syphilitic aneurysm: Sacular type, affecting the ascending aorta

3. Dissecting aneurysm: rare and affect the aorta in old age

4. Mycotic aneurysm: occurs in cerebral , coronary or mesenteric arteries as a result of : Emboli containing low virulent organisms as in subacute bacterial endocarditis.

5. Congenital aneurysm : single or multiple saccular small aneurysms at the points of bifurcation of the cerebral arteries. They are caused by congenital defects of the media.

Their rupture causes subarachnoid or cerebral haemorrhage

- ***False aneurysm***

The wall of the aneurysm is not a part of the arterial wall

The wall consists of **fibrous tisssue**

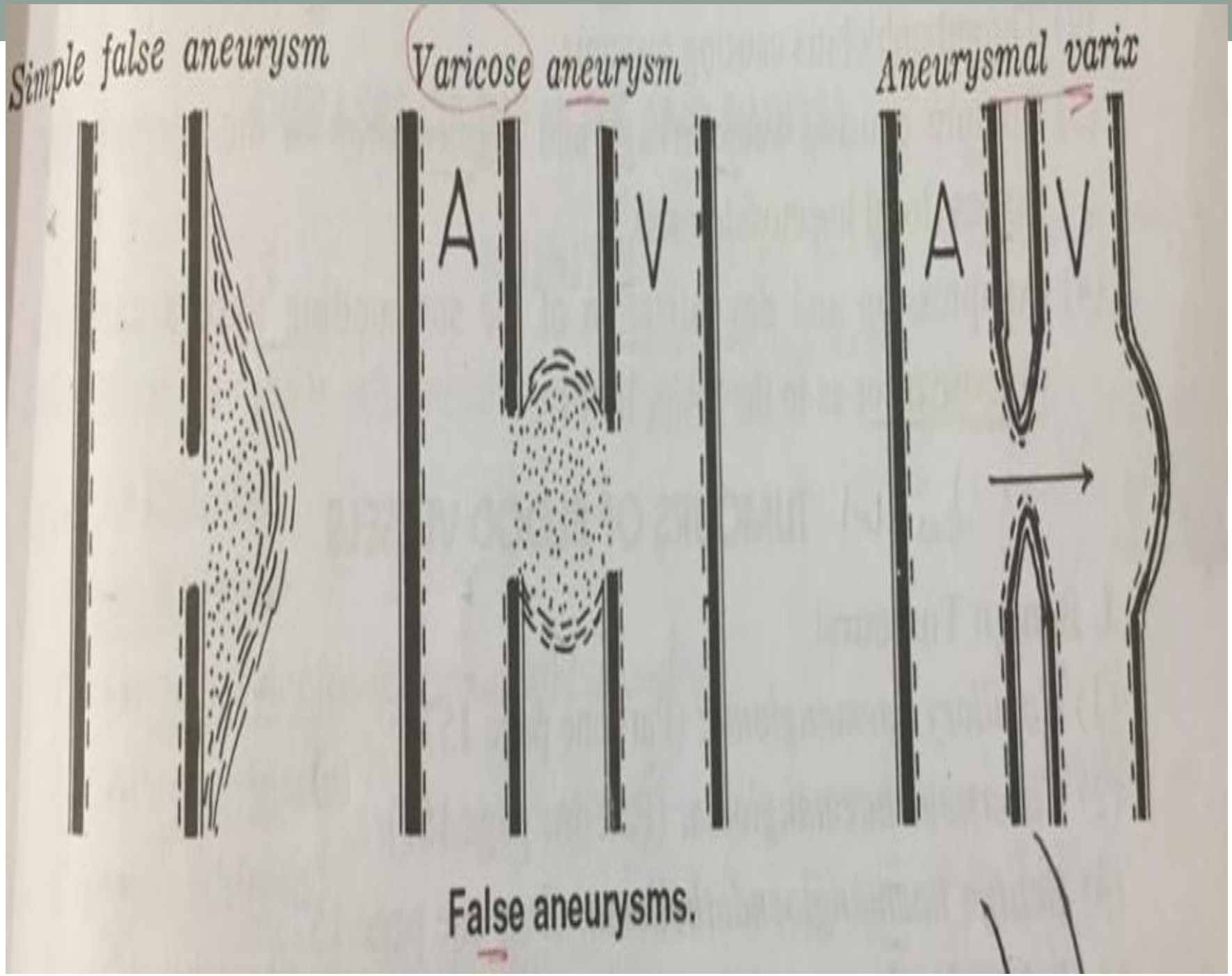
Types

- **Pulsating hematoma** (simple false haematoma): resulting from trauma to an artery causing a small damage .The escaped blood forms a hematoma in the periarterial tissue. The periphery of the hematoma undergoes organization and forms a fibrous sac which communicates with the artery through a small opening.

- **Arterio-venous fistula**: caused by traumatic injury to an artery and the adjacent vein as that caused by bullet or car accident. The injury result in either:

a) Aneurysmal varix: direct communication between the artery and the vein without the intervention of a sac .

B)Varicose aneurysm: A false sac around a hematoma intervenes between and communicates the two vessels.



False aneurysms.

- ***Complications***
  1. Rupture and hemorrhage
  2. Thrombus formation with ischemic effects
  3. Pressure on the surrounding structures


Thanks for your attendance