Ischemic Heart Diseases IHD

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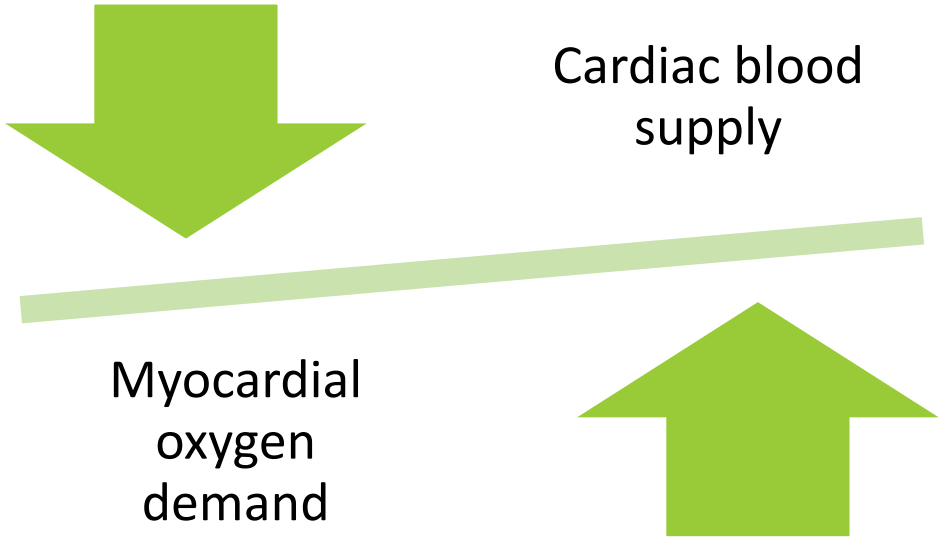
Ischemic Heart Diseases

- IHD is a group of related diseases resulting from **myocardial ischemia**
- It happens due to an imbalance between **cardiac blood supply (perfusion)** & **myocardial oxygen demand**



Ischemic Heart Diseases

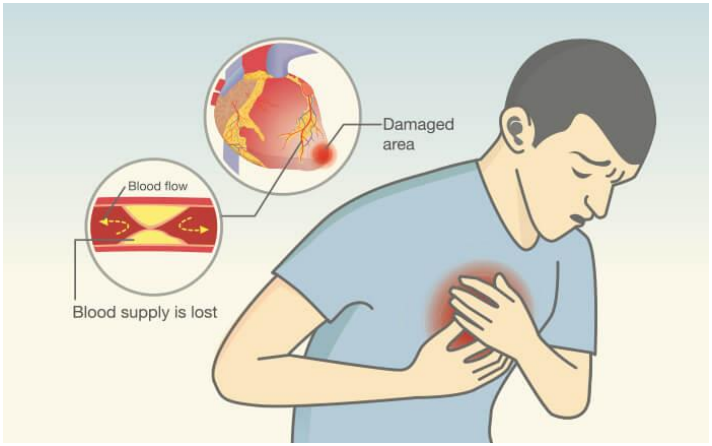
- IHD is synonymous with coronary artery disease (CAD).
- More than 90% of cases due to **obstructive atherosclerotic vascular disease**

Others:

- Increased demand (e.g., with increased heart rate or hypertension)
- Diminished blood volume (e.g., with hypotension or shock)
- Diminished oxygenation (e.g., due to pneumonia or congestive heart failure)
- Diminished oxygen-carrying capacity (e.g., due to anemia or carbon monoxide poisoning).

IHD: Clinical presentation

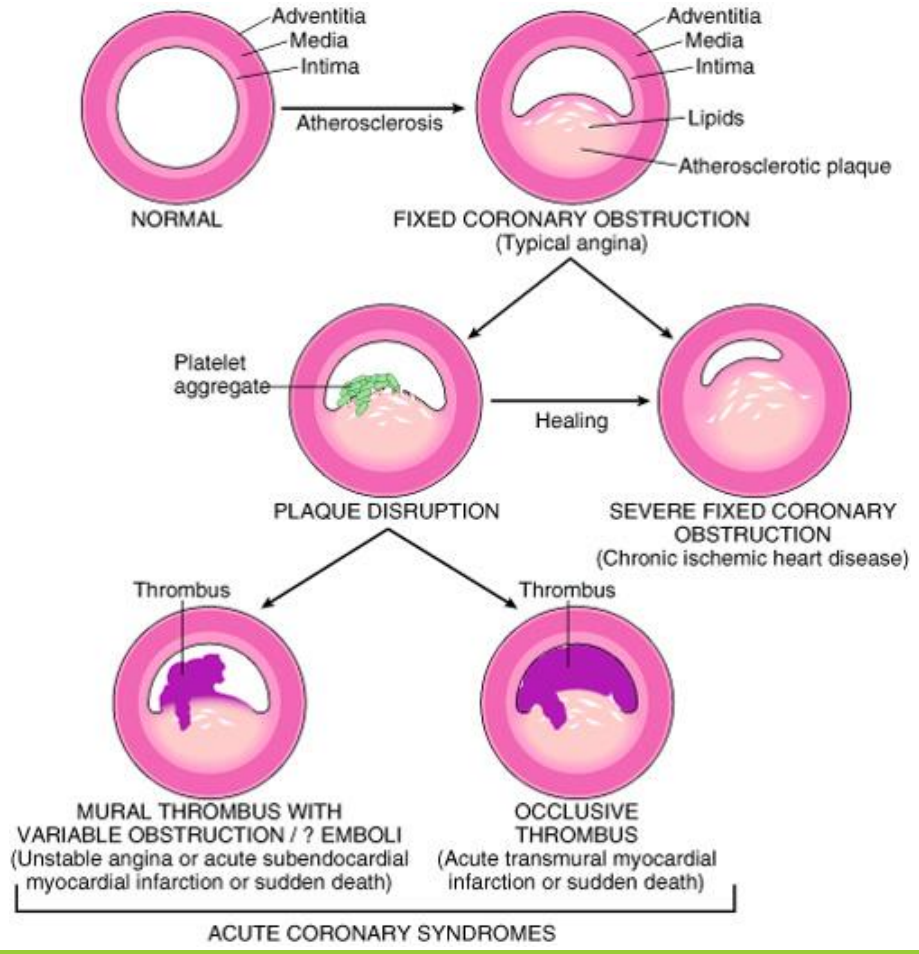
- **Angina pectoris**: Ischemia induces pain but is **insufficient** to cause cardiomyocyte death.
- **Acute myocardial infarction (MI):** Ischemia **sufficient** to cause cardiomyocyte death.
- **Chronic IHD**: Progressive cardiac heart failure following MI.
- **Sudden cardiac death (SCD):** can occur due to lethal arrhythmia after MI



Acute coronary syndrome is a term is applied to the three catastrophic manifestations of IHD: **unstable angina, acute MI, and SCD**

Pathogenesis

- Inadequate coronary perfusion relative to myocardial demand.
- Critical stenosis occurs when the lesion is obstructing **70% to 75% or more** of a single vessel lumen, generally causing stable angina (inadequate perfusion in setting of increased demand).
- **A fixed 90% stenosis can lead to inadequate coronary blood flow even at rest**.

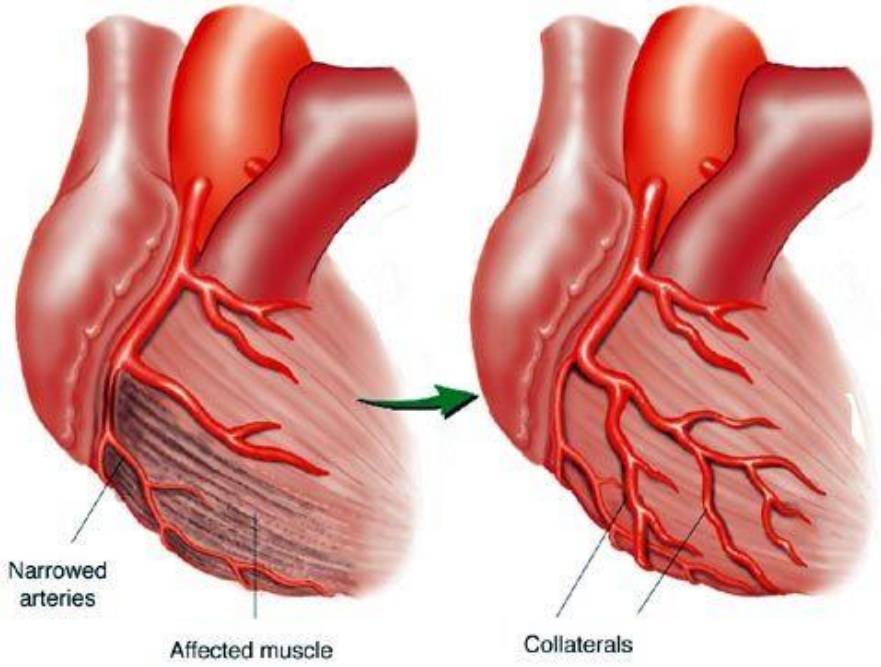


Pathogenesis

Note:

- Slow rate atherosclerosis, over the years, can lead to **collateral perfusion** that can subsequently protect against MI even if the vessel eventually becomes completely occluded.

- Acute coronary blockage: no time for the collateral flow to develop and infarction results.



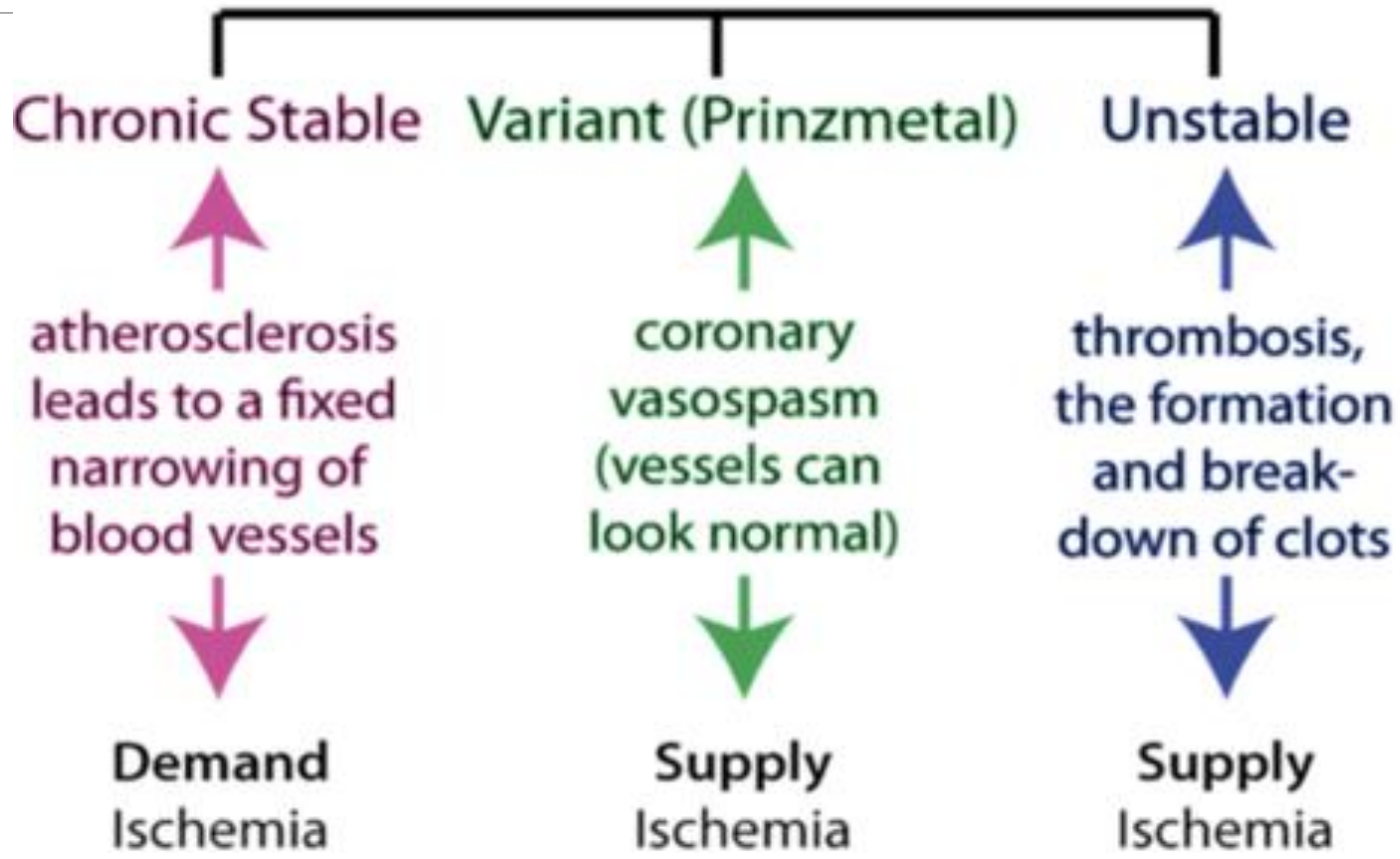
Angina Pectoris

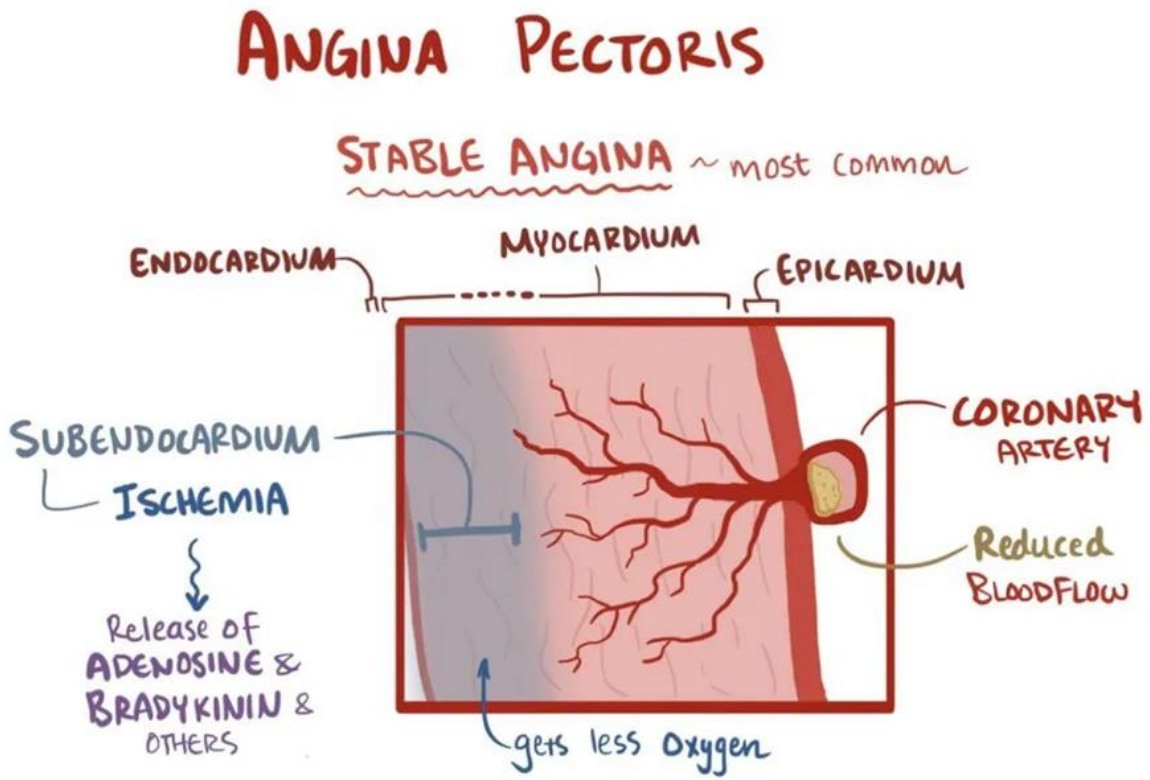
Is **intermittent chest pain** caused by **transient, reversible myocardial ischemia**.

There are 3 variants:

- **Typical or stable angina**
- **Unstable angina (crescendo angina)**
- **Prinzmetal or variant angina**

Angina


Chronic Stable
Variant (Prinzmetal)
Unstable
atherosclerosis leads to a fixed narrowing of blood vessels
coronary vasospasm (vessels can look normal)
thrombosis, the formation and break-down of clots
Demand Ischemia
Supply Ischemia
Supply Ischemia



Angina Pectoris

- Intermittent chest pain
- Ischemia-induced release of adenosine, bradykinin, and other molecules that stimulate the autonomic afferents and causes chest pain.

Typical or stable angina

- Episodic chest pain associated with particular levels of **exertion** (e.g., tachycardia or hypertension due to fever, anxiety, fear)
- Crushing or squeezing substernal sensation that can radiate down the left arm or to the left jaw (referred pain)
- Pain relieved by rest (reducing demand) or by drugs such as nitroglycerin (vasodilator)

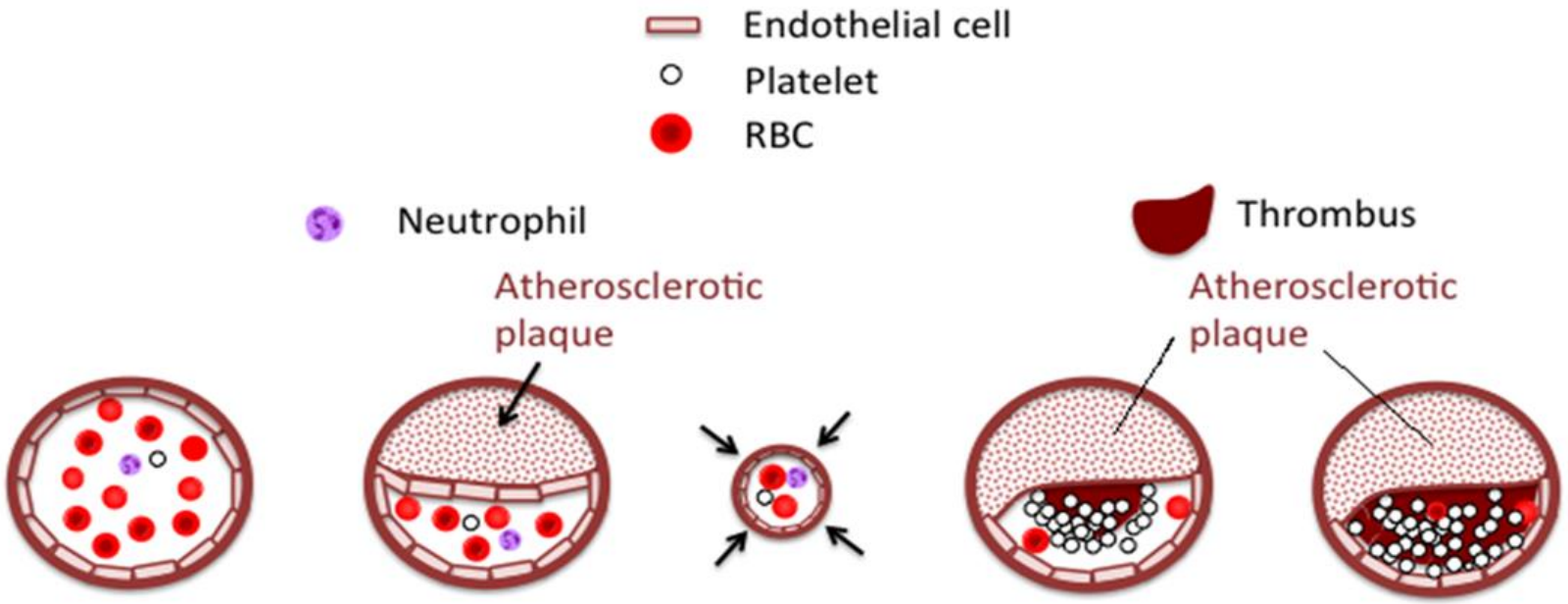
Unstable angina (crescendo angina)

- Increasingly frequent pain, precipitated by progressively less exertion or even occurring at rest.
- Associated with plaque disruption and superimposed thrombosis, and/or vasospasm.
- It is a serious condition that carries a risk for potentially irreversible ischemia (due to complete luminal occlusion by thrombus) & is therefore sometimes called **pre-infarction angina**

Prinzmetal or variant angina

- Occurs at rest
- Caused by coronary artery **spasm.**
- Completely normal vessel can be affected.
- The etiology is not clear
- Responds to vasodilators such as nitroglycerin and calcium channel blockers.

Blood Flow to the Heart



Normal

Chronic Stable Exertional Angina
(ischemia - cell injury)
Temporary

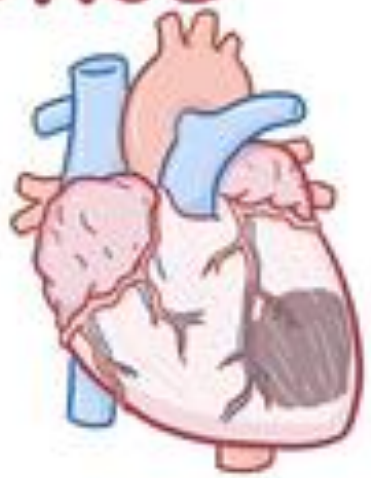
Printzmetal's Vairiant Vasospastic Angina
(ischemia - cell injury)
Temporary

Unstable Angina
Acute Coronary Syndrome
(ischemia - cell injury)
Temporary

Myocardial Infarction
(**heart attack** - cell death)
Permanent

CORONARY ARTERY DISEASE

* CAN LEAD to MYOCARDIAL ISCHEMIA
  - MYOCARDIUM isn't getting a SUFFICIENT BLOOD SUPPLY
* CHARACTERIZED by ANGINA PECTORIS



VASOSPASTIC DISEASE

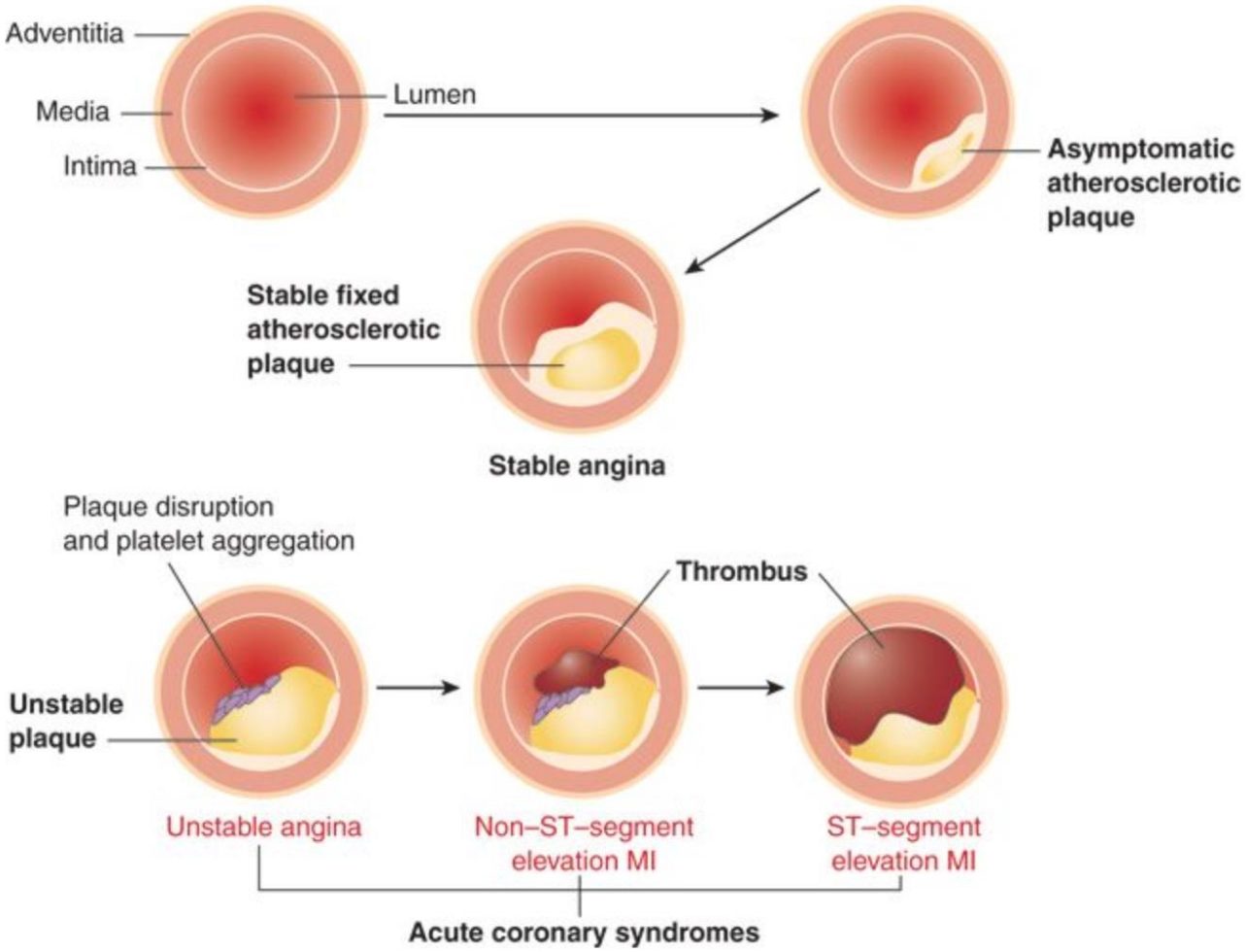
(PRINZMETAL ANGINA)

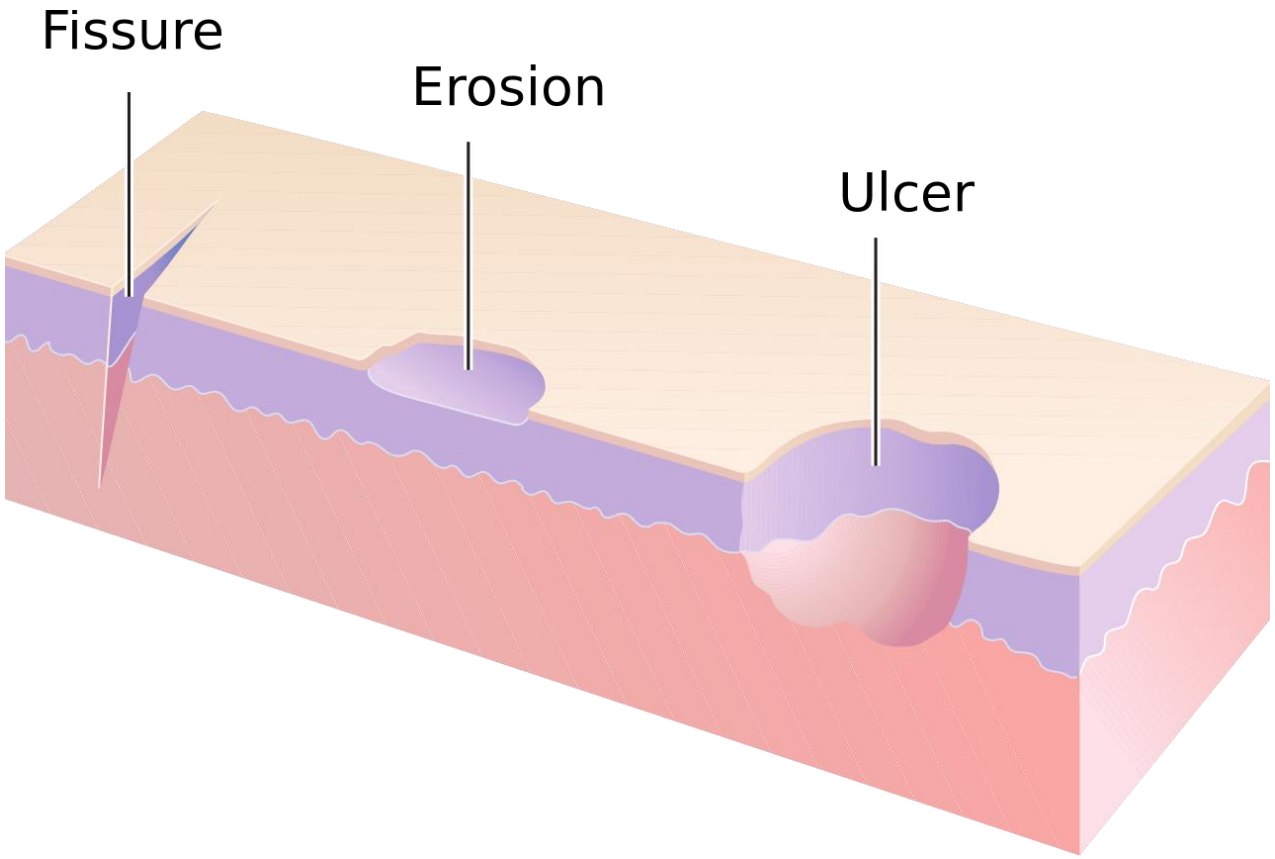
- TRANSIENT VASOCONSTRICTION of a CORONARY ARTERY
  - → TRANSIENT ISCHEMIA
- ATTACKS OCCUR at REST
- ATTACKS OCCUR in CLUSTERS

ATHEROSCLEROTIC DISEASE

- CORONARY ARTERY NARROWS due to ATHEROSCLEROTIC PLAQUE

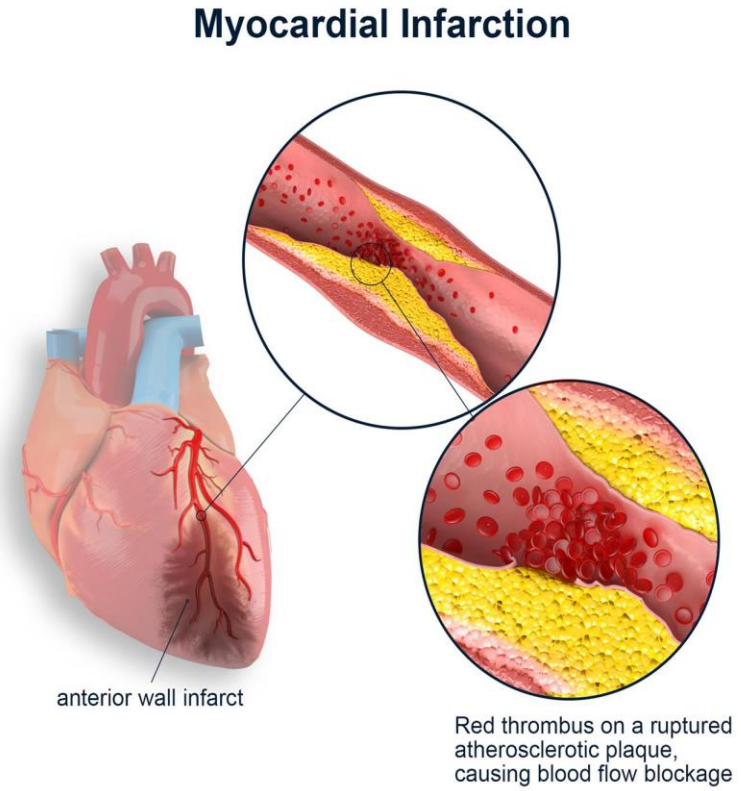
- STABLE ANGINA
- UNSTABLE ANGINA
- MYOCARDIAL INFARCTION
  - ACUTE CORONARY SYNDROME


Adventitia
Media
Intima
Lumen
Asymptomatic atherosclerotic plaque
Stable fixed atherosclerotic plaque
Stable angina
Plaque disruption and platelet aggregation
Thrombus
Unstable plaque
Unstable angina
Non–ST–segment elevation MI
ST–segment elevation MI
Acute coronary syndromes


Fissure
Erosion
Ulcer

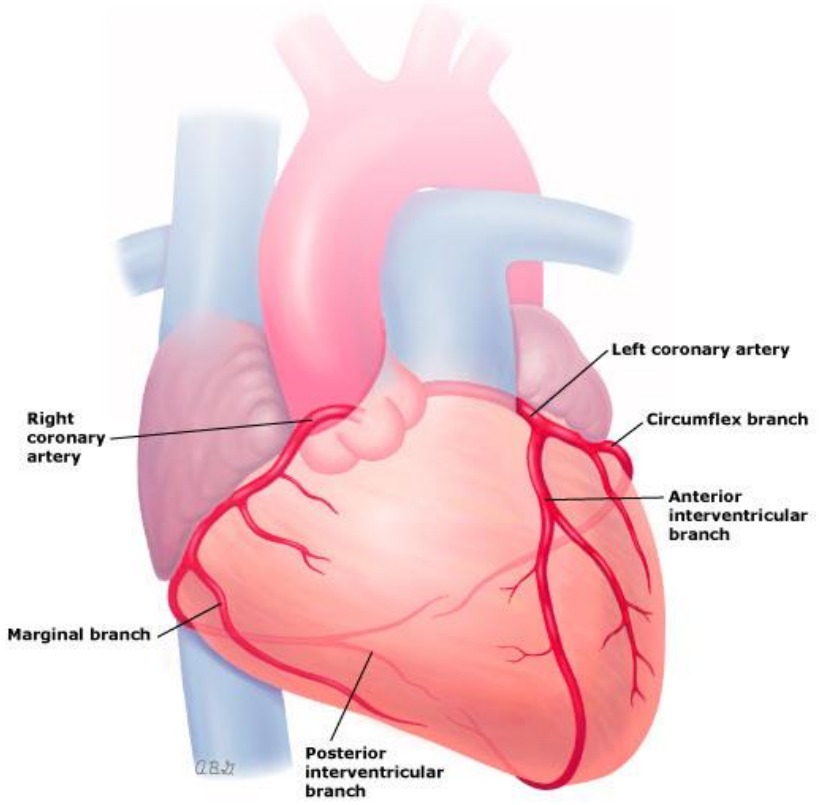
Myocardial Infarction (MI)

- Also called “Heart attack”
- **Necrosis** of heart muscle due to ischemia.
- Any age, frequency rises with increasing age and with increasing atherosclerotic risk factors
- **Vast majority of MIs are caused by acute coronary artery thrombosis**
- 10% of MIs occurs in the absence of occlusive atherosclerosis ➡ embolization from mural thrombi



Vessels involved in MI

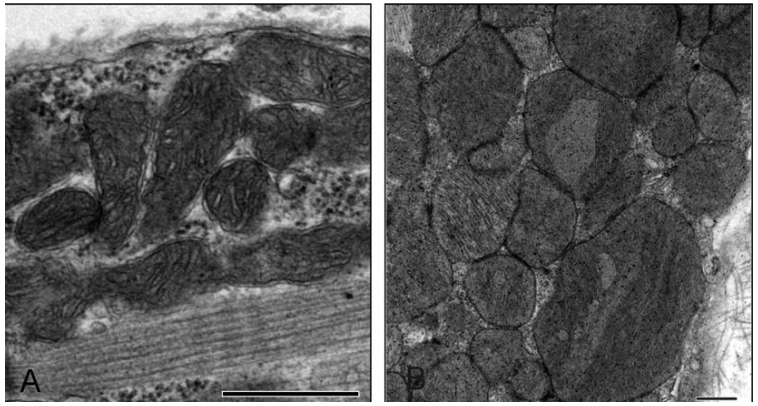
- **Left anterior descending artery (40% to 50%):** MI involves anterior LV, anterior IVS, & apex circumferentially.
- **Right coronary artery (30% to 40%):** MI involves posterior LV, posterior IVS, & RV free wall in some cases.
- **Left circumflex artery (15% to 20%):** MI involves lateral LV, except the apex.



Myocardial Response to Ischemia

- **Within seconds of vascular obstruction:** aerobic glycolysis ceases ⟹ drop in ATP ⟹ accumulation of potentially noxious metabolites (e.g., lactic acid) in the cardiac myocytes.
- **Within a minute of the onset of ischemia:** **Rapid loss of contractility**
- **Within few minutes:** **Ultrastructural changes**: myofibrillar relaxation, glycogen depletion, cell and mitochondrial swelling

These early changes are potentially **reversible**



Myocardial Response to Ischemia

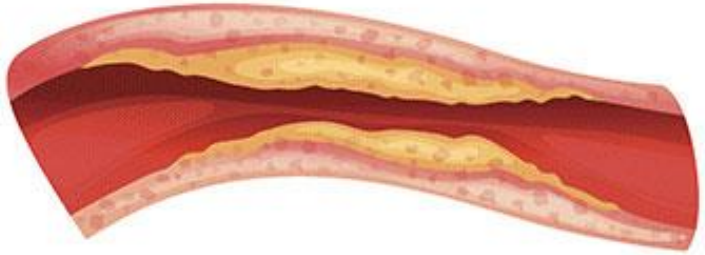
- **Severe ischemia lasting 20 to 40 minutes:** **irreversible** damage and coagulative necrosis.

NOTE:

- If myocardial blood flow is restored before irreversible injury occurs, cell viability can be preserved
- This is the rationale for early diagnosis and prompt intervention by **thrombolysis** or **angioplasty** to salvage myocardium at risk.

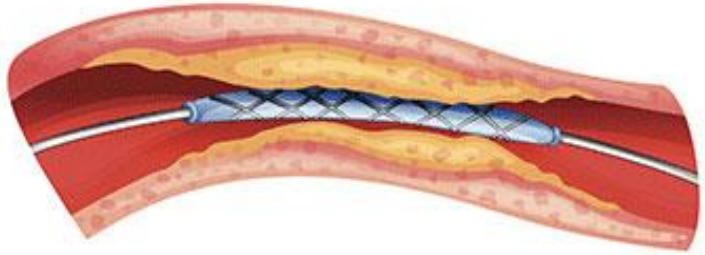
Stent with Balloon Angioplasty

1



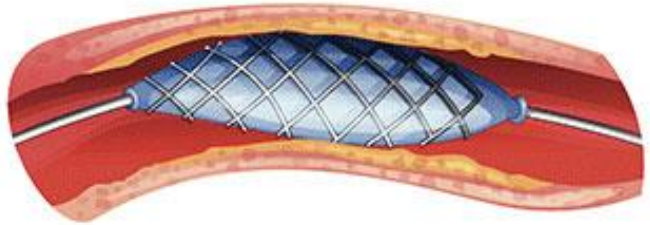
Build up of cholesterol partially blocking blood flow through the artery.

2



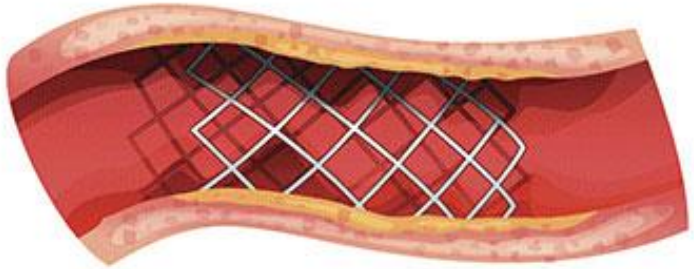
Stent with balloon inserted into partially blocked artery.

3



Balloon inflated to expand stent.

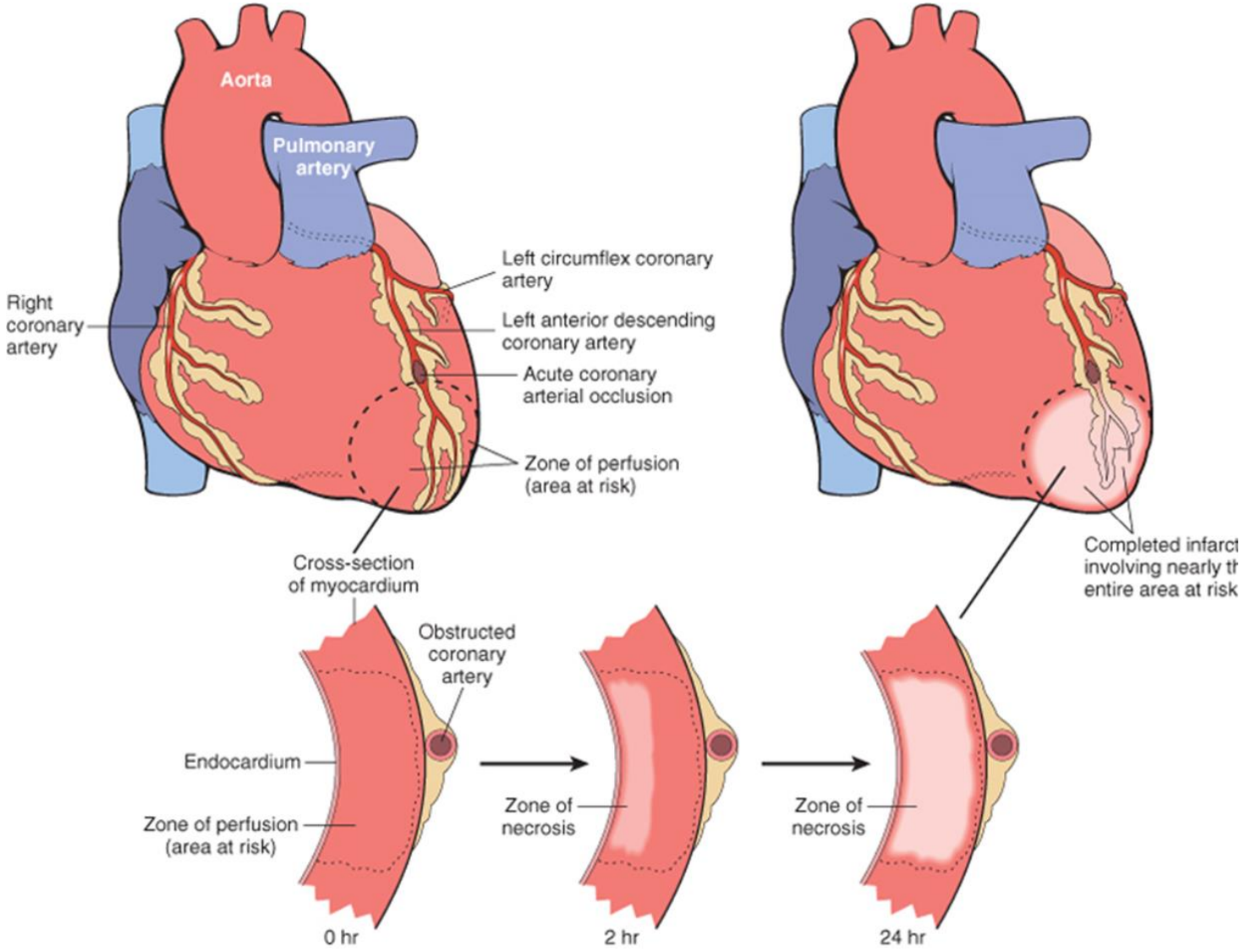
4



Balloon removed from expanded stent.

Myocardial Response to Ischemia

- Irreversible injury of ischemic myocytes first occurs in the subendocardial zone (because sub endocardium is the last area to receive blood delivered by the epicardial vessels)
- Also, sub endocardium is exposed to relatively high intramural pressures, which act to impede the inflow of blood.
- **With more prolonged ischemia,** a wave front of cell death moves, with the infarct usually achieving its full extent within 3 to 6 hours

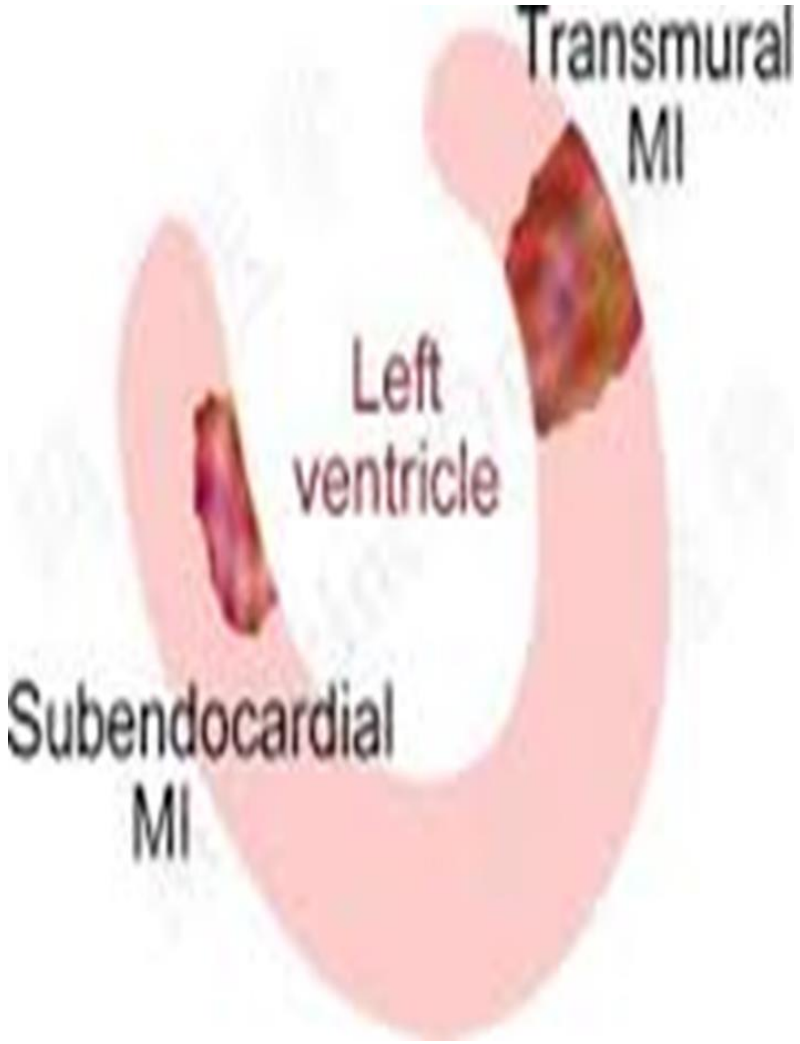


Progression of myocardial necrosis after coronary artery occlusion

Patterns of Infarction

The location, size, and morphologic features of an acute MI depend:

- ✓The size and distribution of the involved vessel
- ✓The rate of development and the duration of the occlusion
- ✓Metabolic demands of the myocardium
- ✓Extent of collateral supply



Patterns of Infarction

Transmural infarctions:

- Involve the full thickness of the ventricle
- ST segment elevations on ECG: ST elevated MIs (STEMIs).

Subendocardial infarctions:

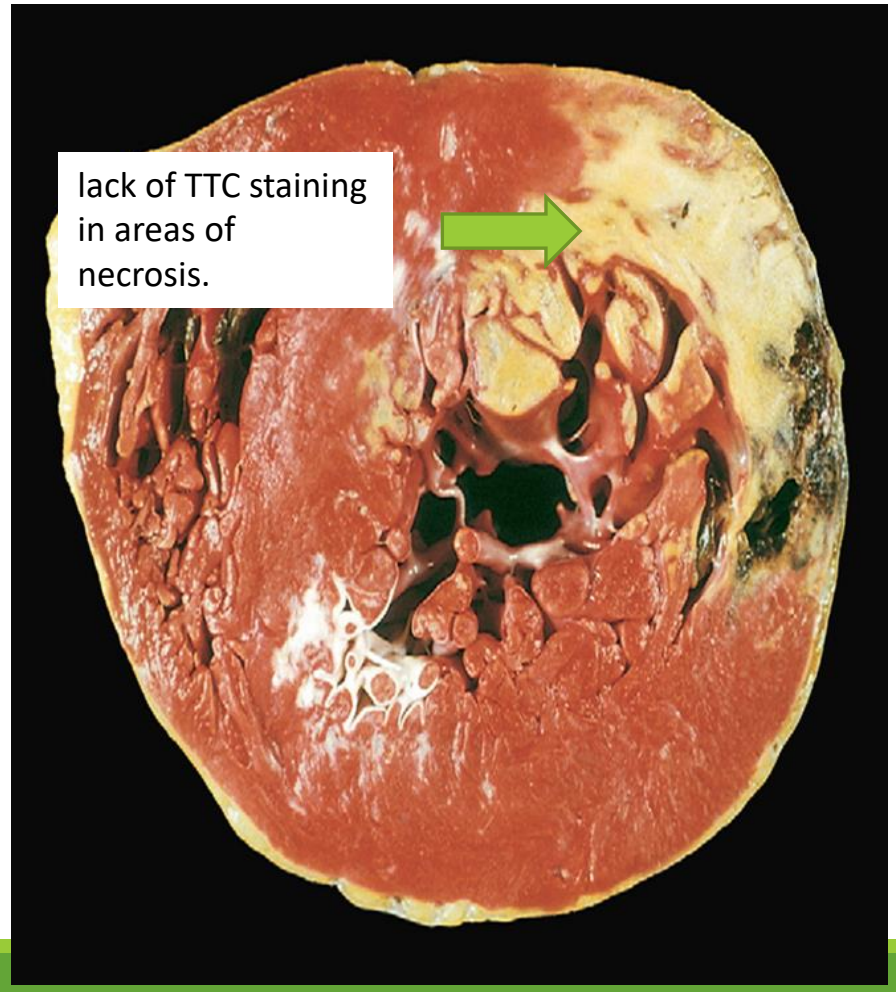
- Limited to the inner third of the myocardium
- Non-ST elevation infarcts

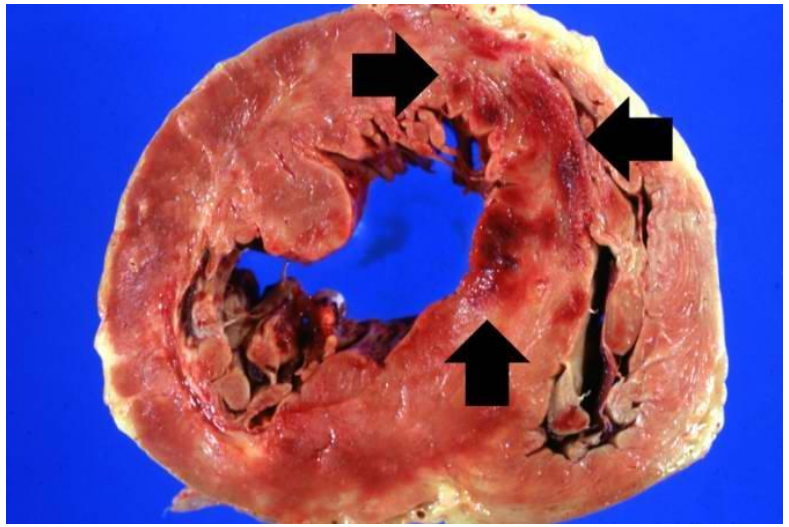
Myocardial Infarction (Gross morphology)

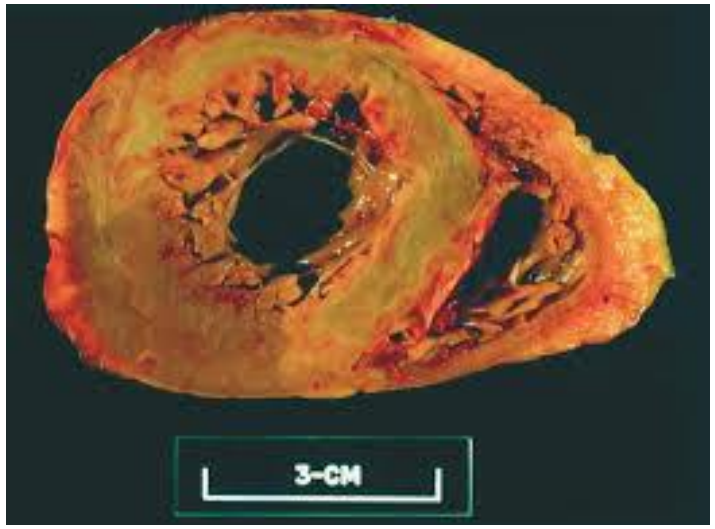
The gross and microscopic appearance depends on the age of the injury

Gross:

- Infarcts more than 3 hours old can be visualized by exposing myocardium to **triphenyltetrazolium chloride**, a substrate for lactate dehydrogenase.
- This enzyme is depleted in the area of ischemic necrosis (it leaks out of the damaged cells), and the **infarcted area is unstained (pale)**







Myocardial Infarction (Gross morphology)

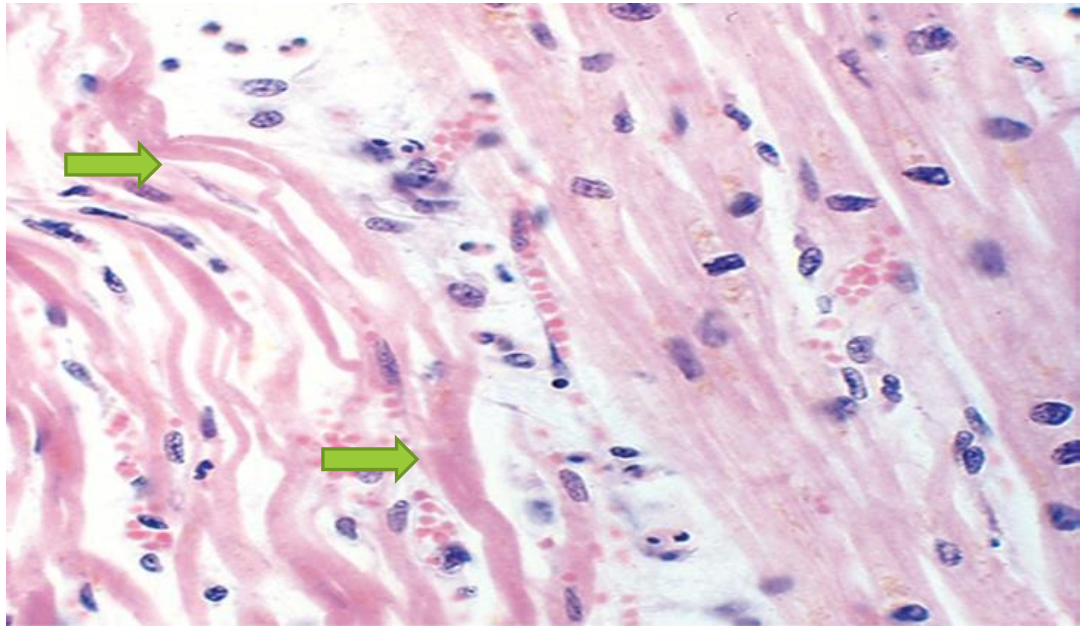
- **12 - 24 hours after MI: red-blue discoloration** caused by stagnated, trapped blood.
- Then, infarcts become soft, yellow-tan areas
- **10 - 14 days**: infarcts are rimmed by hyperemic (highly vascularized) **granulation tissue.**
- Over the succeeding weeks: **fibrous scar**.

0-12 hours	There are no morphological changes yet.
12-18 hours	**Coagulation** necrosis begins, the cytoplasm of the necrotic myocytes becomes eosinophilic, loss of cross striations, pyknosis and karyorrhexis. **Wavy fiber** change at the periphery of the infarct.
18-72 hours	The area shows a slight pallor. **Neutrophils** begin to show up and **peak about 3 days** and subsequently diminish. **contraction bands** at the periphery of the infarct produced by hypercontraction of myofibrils in dying cells.
4-7 days	The infarct will appear pale firm with a hyperemic boarder. **Macrophages,** fibroblasts and capillaries first appear at the margins then begin to migrate into center. Macrophages begin to phagocytoze the necrotic myocytes.
10 days	The necrotic area is yellow, soft; the **granulation tissue** is visible grossly at the edge of the infarct as a red-purple zone. Collagen fibers are seen and many macrophages with remnants of myocytes.
4-8 weeks	Vascularity diminishes and most infarcts have been replaced by **dense scar** tissue. The ventricular wall is thinned, firm, and gray at the site of the healed infarct

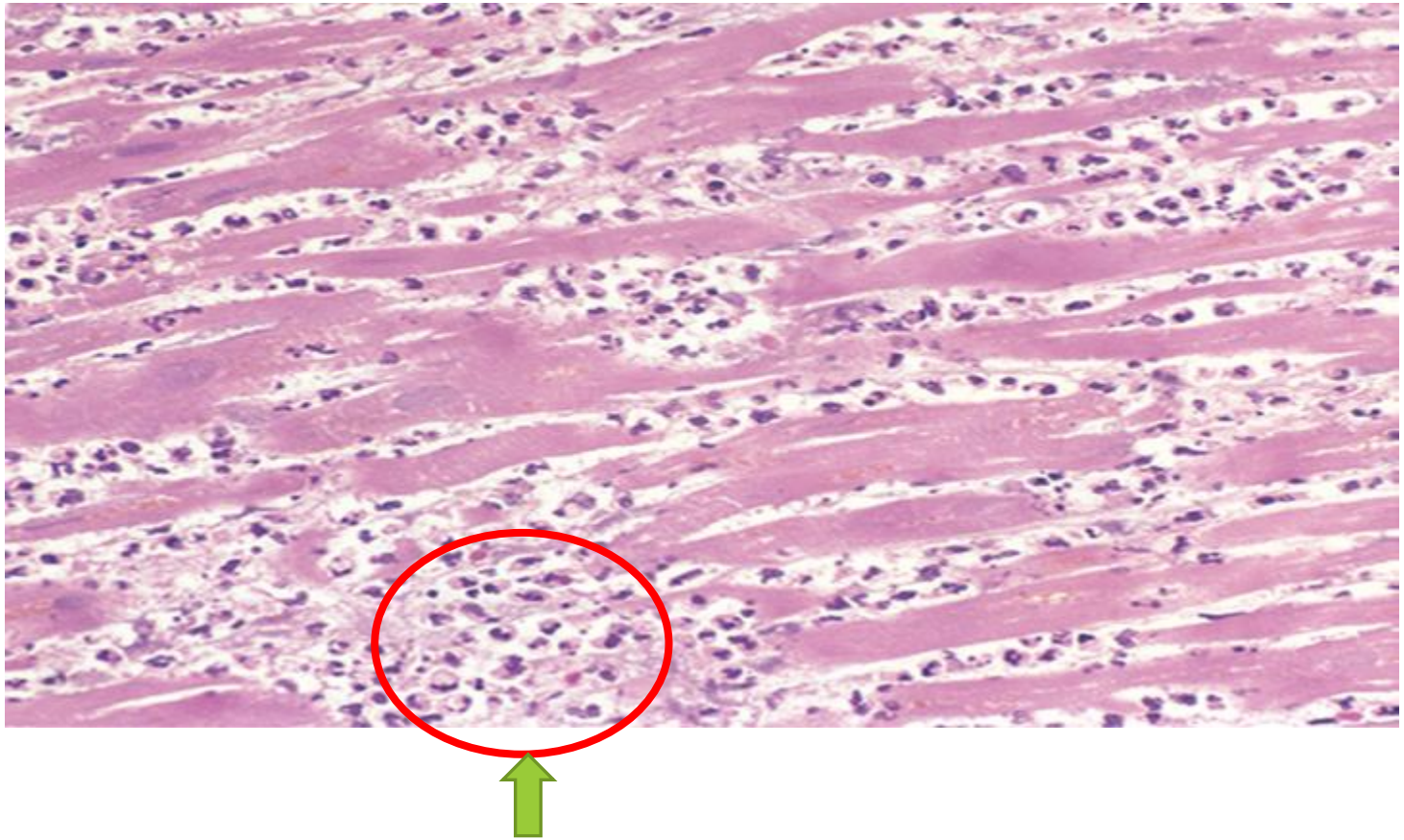
Myocardial Infarction (histology)

Coagulative necrosis:

4 to 12 hours of infarction.
“Wavy fibers” can be present



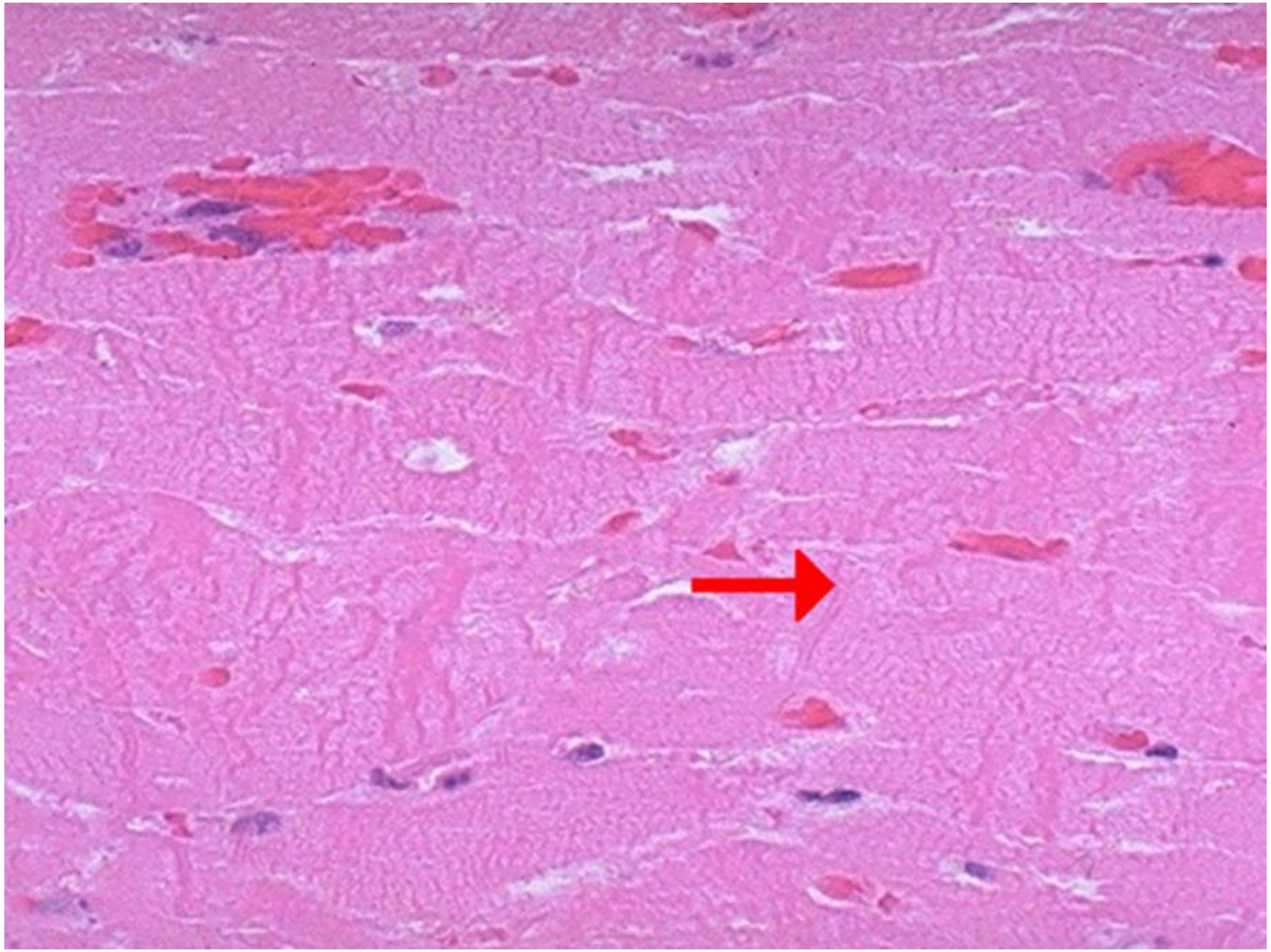
Myocardial Infarction (histology)



Collection of acute inflammation neutrophils

Acute inflammation:

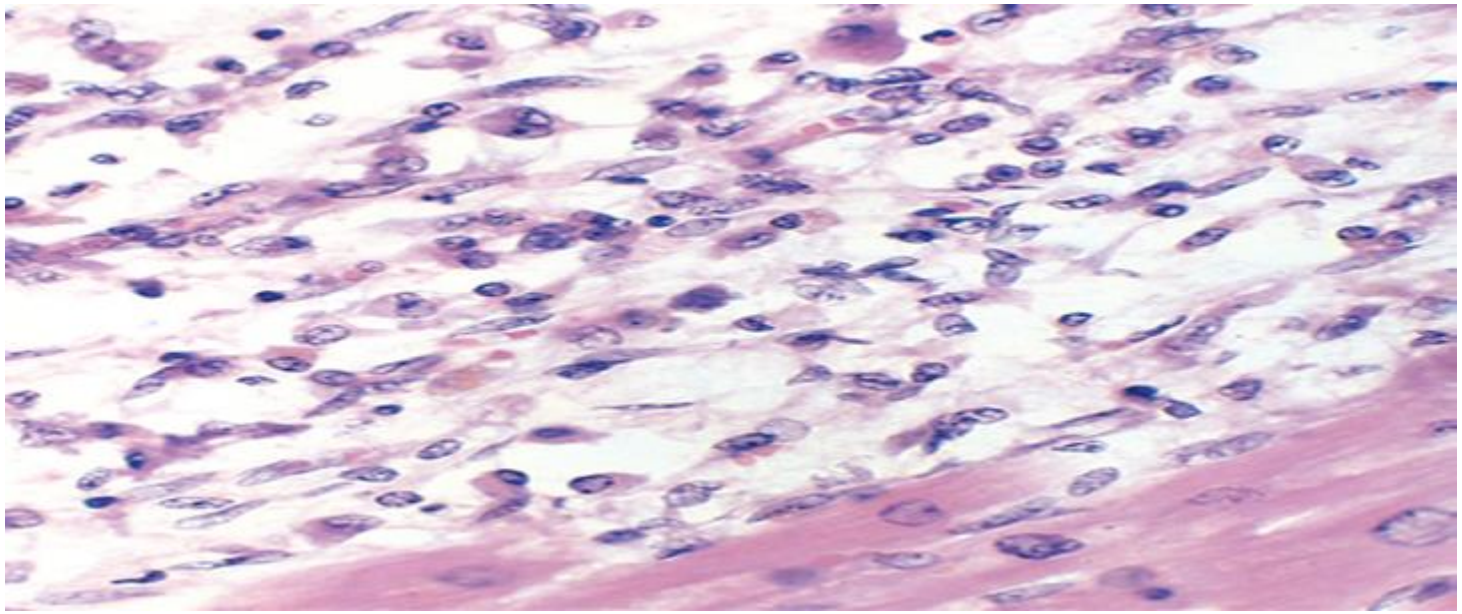
1 to 3 days after MI



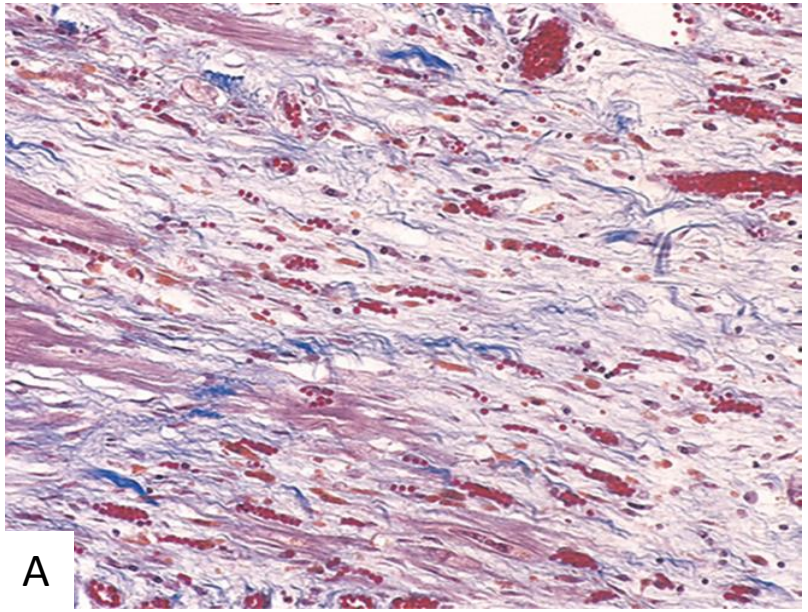
Myocardial Infarction (histology)

Contraction fibers

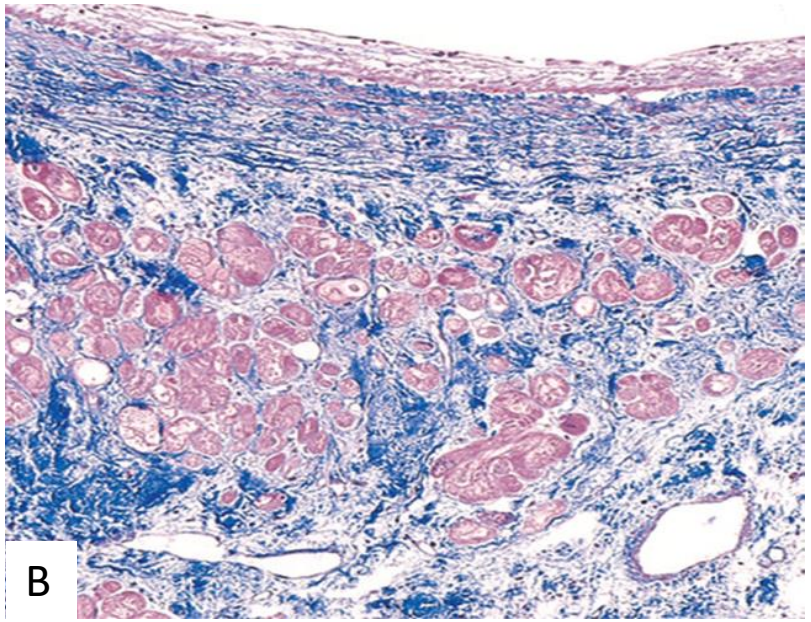
Myocardial Infarction (histology)



Macrophages: 4 to 7 days after MI



A



B

Myocardial Infarction (histology)

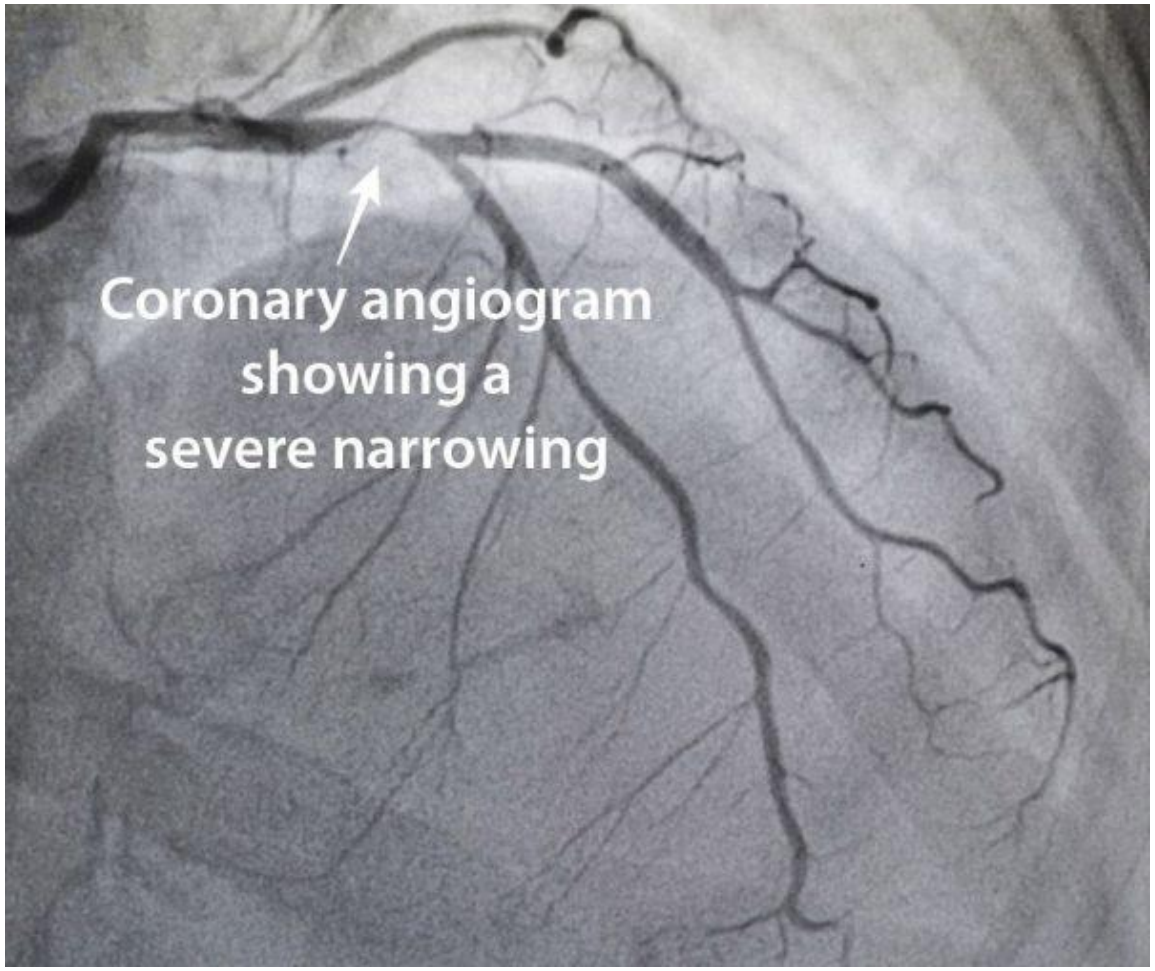
A. Infarcted zone replaced by granulation tissue: 1 to 2 weeks after MI

B. Scarring is well advanced by the end of the sixth week.

Note: once an MI is completely healed, it is impossible to distinguish its age: Whether present for 8 weeks or 10 years, fibrous scars look the same.

Clinical Features of MI

- Severe, crushing substernal chest pain (or pressure) that can radiate to the neck, jaw, epigastrium, or left arm.
- Pain typically **lasts several minutes to hours and is not relieved by nitroglycerin or rest.**
- Silent infarcts : 10% to 15% of MIs
- Common in diabetes mellitus and elderly.
- The pulse generally is rapid and weak, and patients are often diaphoretic (sweaty) and nauseous
- With massive MIs (involving more than 40% of the left ventricle): cardiogenic shock develops



Clinical Features of MI

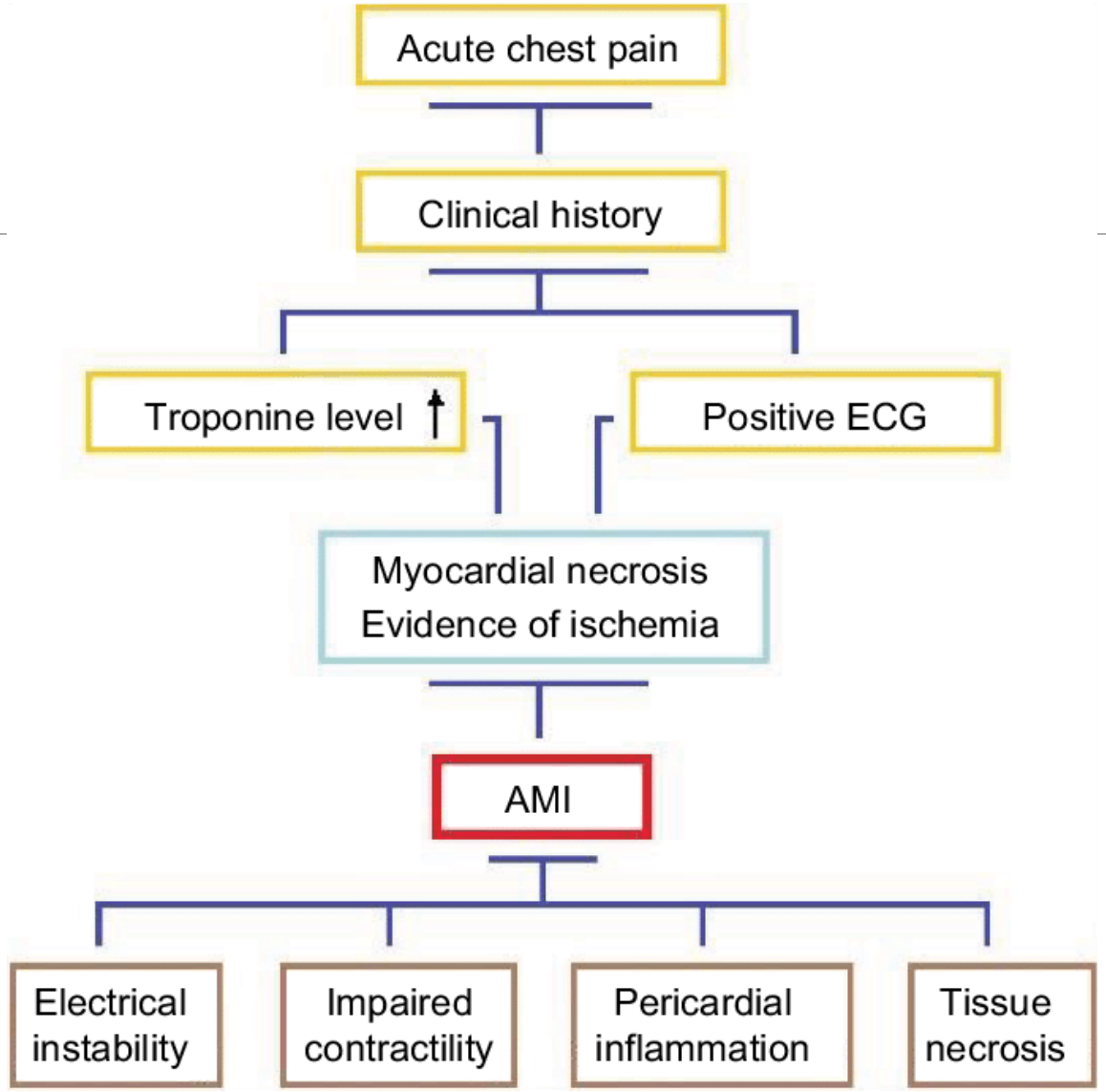
How to diagnose MI:

- Symptoms (history)
- Electrocardiographic findings (ECG)
- Biochemical markers (troponin, serum creatine kinase, creatine kinase-MB)
- Coronary angiogram: allows visualization of narrowing or obstructions on the heart vessels, and therapeutic measures can follow immediately

Complications of MI

- Overall, in-hospital death rate for MI is approximately 7%.
- Out-of-hospital mortality is substantially worse:
  - A third of persons with (STEMIs) will die, usually of an **arrhythmia (mainly VF)** within an hour of symptom onset, before they reach the hospital

✓Advanced age, female gender, diabetes mellitus, & previous MI are associated with poor prognosis

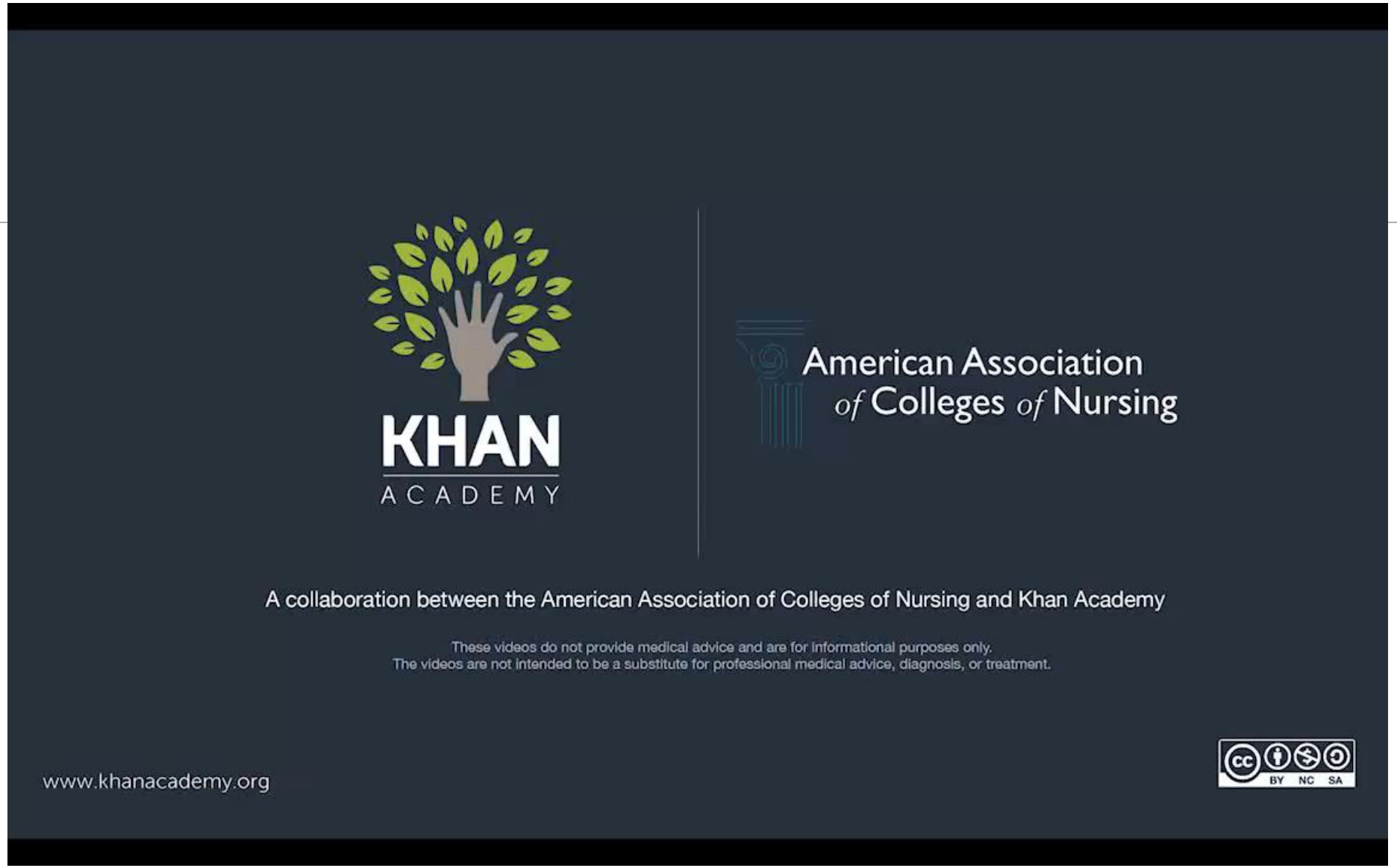

Acute chest pain
Clinical history
Troponine level ↑
Positive ECG
Myocardial necrosis
Evidence of ischemia
AMI
Electrical instability
Impaired contractility
Pericardial inflammation
Tissue necrosis

Complications of MI

- Nearly three-fourths (3/4) of patients have one or more complications after acute MI

MAIN COMPLICATIONS OF MI:

Contractile dysfunction (LVF)
Cardiogenic shock
Papillary muscle dysfunction
Myocardial rupture
Pericarditis
Mural thrombus
Ventricular aneurysm
Congestive heart failure
Arrhythmias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Contractile dysfunction (LVF)

- An MI affects left ventricular pump function approximately proportional to its size.

- Typically, there is some degree of LV failure, with hypotension.

Cardiogenetic shock

Cardiogenic shock means severe "pump failure"

- Occurs in 10% to 15% of patients after acute MI
- Generally, with a massive large infarct (often > 40% of the LV)
- Accounts for nearly 70% mortality rate

Arrhythmias

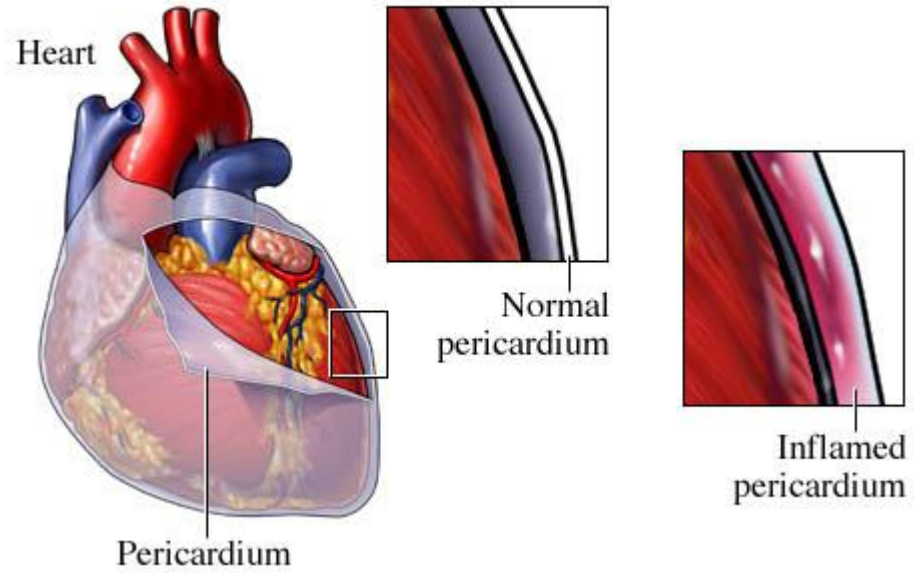
- Arrhythmias: are the most common (80% to 90%) cause of sudden deaths following an MI.
- It include the most serious ventricular fibrillation (VF) & heart block.
- In addition, other arrhythmias, such as sinus bradycardia, tachycardia, ventricular premature contractions or ventricular tachycardia may occurs.

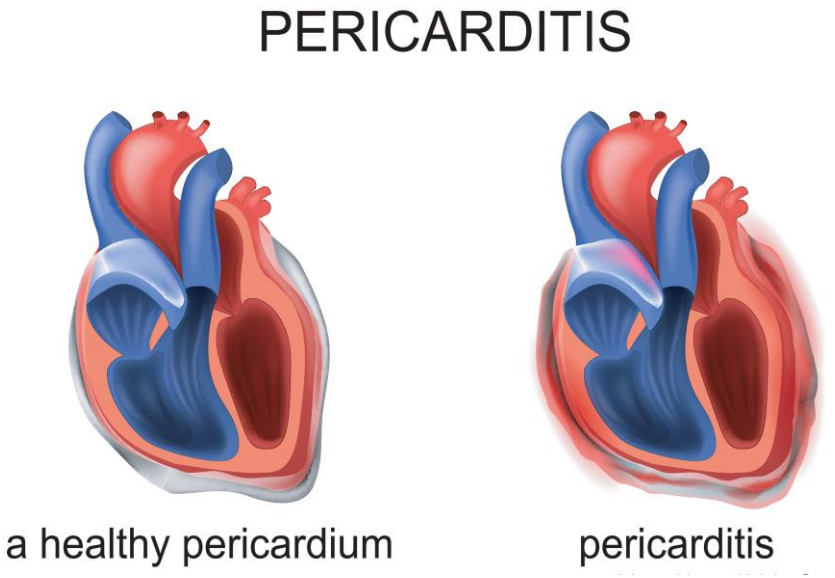
Pericarditis

- **Pericarditis:** a fibrinous or hemorrhagic pericarditis usually develops within 2 to 3 days of a transmural MI & typically spontaneously resolves with time. Inflammatory response to the area of myocardial infarct.

- **Dressler syndrome** is a secondary form of pericarditis. The symptoms tend to occur 2–3 weeks after myocardial infarction. It is believed to result from an autoimmune inflammatory reaction to myocardial neo-antigens formed as a result of the MI.

Pericarditis



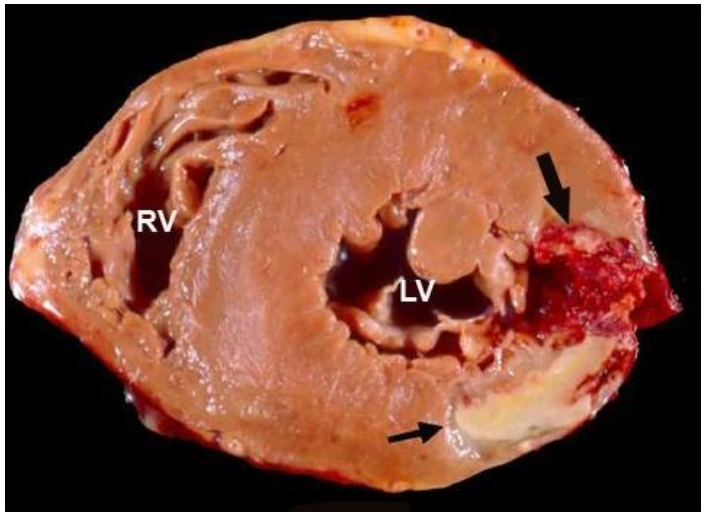


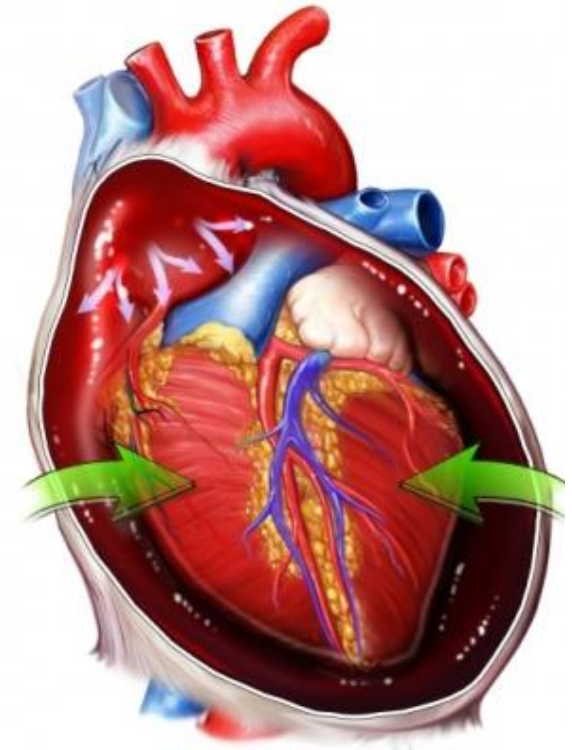
Myocardial Rupture

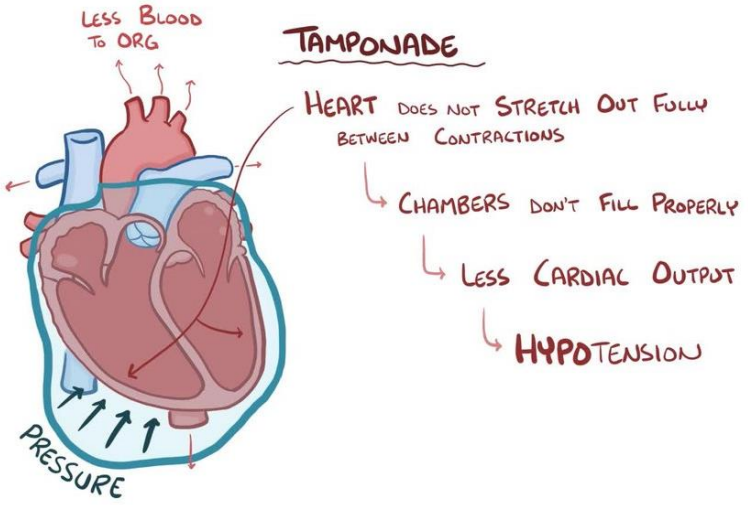
Myocardial rupture complicates 1% to 5% of MI and includes:

1. rupture of the **ventricular free wall**, with fatal hemopericardium & cardiac tamponade
2. rupture of the **infarcted IVS**, leading to a new VSD & left-to-right shunt
3. rupture of **infarcted papillary muscle**, resulting in severe mitral regurgitation

NOTE: Rupture can occur at almost any time after MI but is most common 3 to 7 days after infarction; when granulation tissue has not deposited sufficient collagenous matrix to repair the wall.







rupture of the **ventricular free wall**

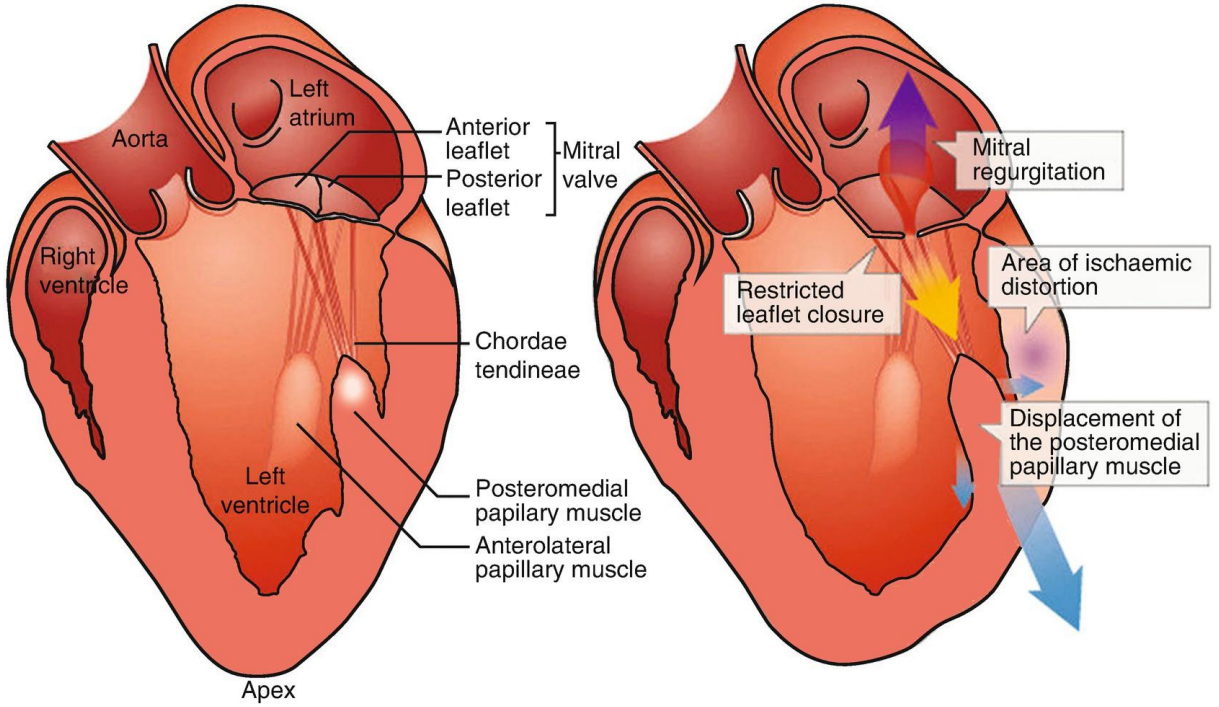
rupture of infarcted papillary muscle

Dysfunction of a papillary muscle after MI causes **mitral regurgitation**

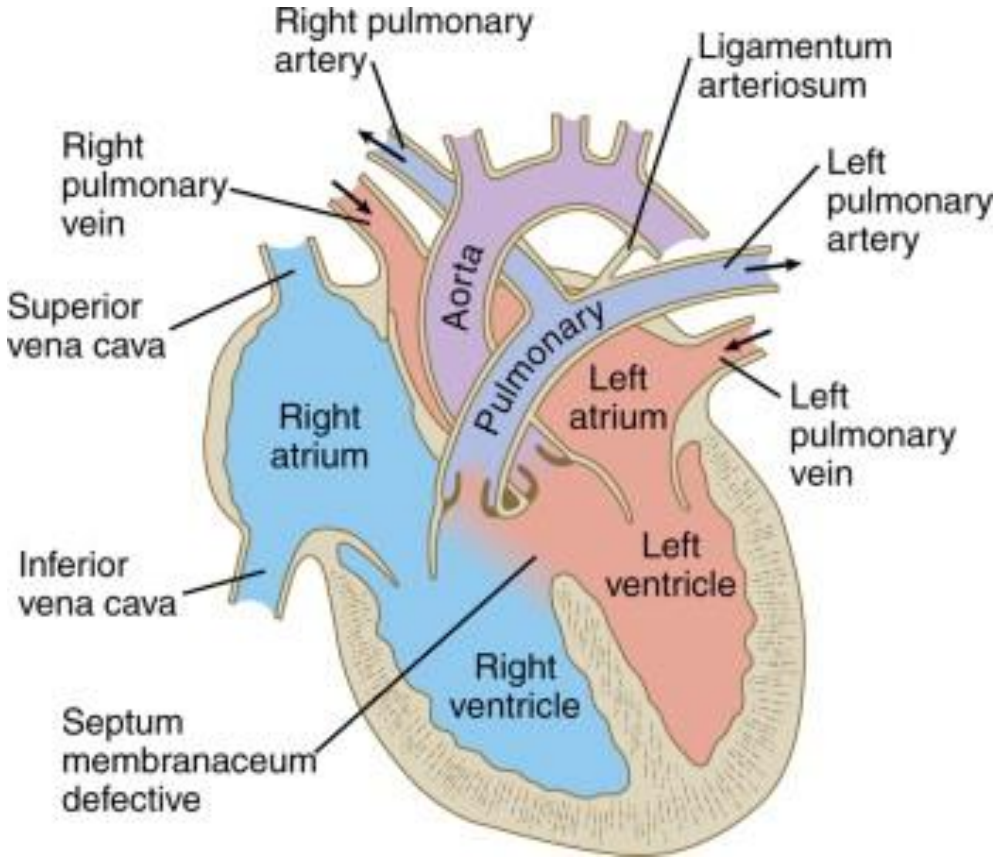
Papillary muscle dysfunction can result from

I. **Ischemia** of papillary muscle & the underlying myocardium

II. Or rarely **rupture** of the infarcted papillary muscle



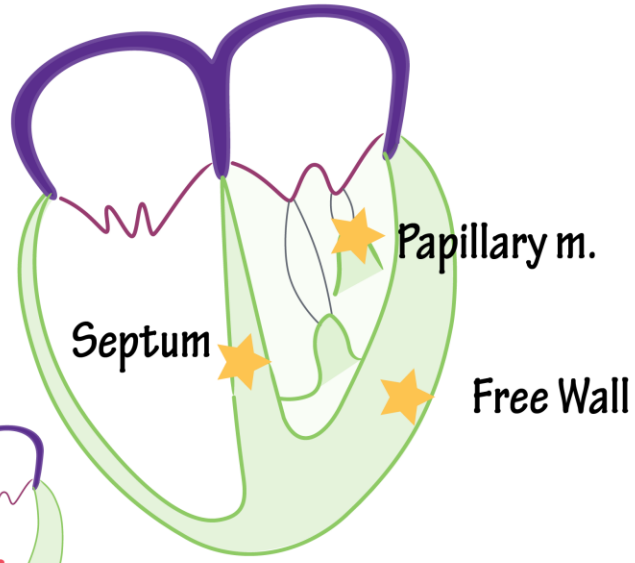
rupture of the infarcted IVS



Myocardial Infarctions: Complications

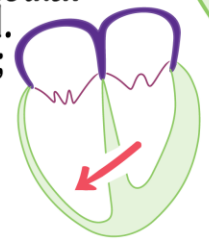
Myocardial Rupture

Rupture (tear) occurs because necrotic tissue weakens the myocardium.



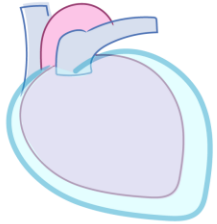
Septal rupture:

Leads to left-to-right shunting, which produces right ventricular volume & pressure overload. Listen for systolic murmur; requires surgical closure.



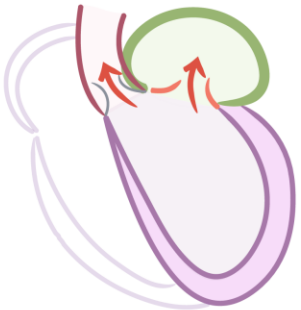
Free wall rupture:

Can lead to cardiac tamponade (fluid in pericardial sac) & sudden death. Acute is sudden, often fatal w/no time for intervention; Subacute tears may be surgically repaired.



Papillary muscle rupture:

Leads to mitral valve regurgitation, cardiogenic shock, heart failure. Rupture may be partial or complete; Surgical repair/replacement required.

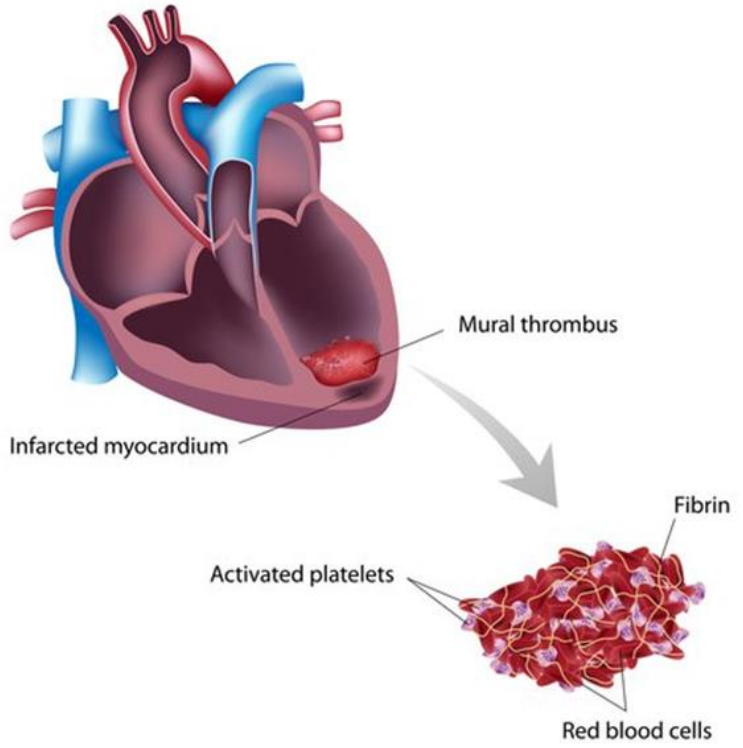


Mural thrombus

Mural thrombus:

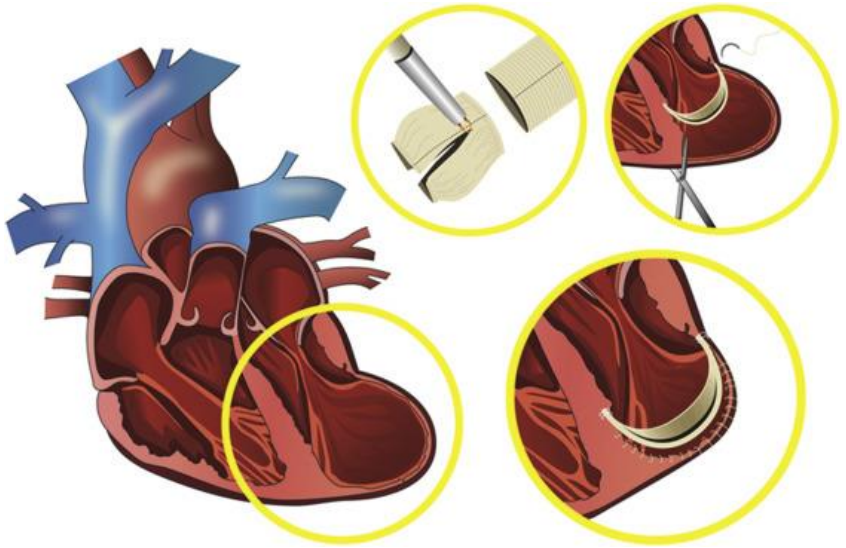
- Infarct expansion causes weakening of necrotic muscle leading to thinning, & dilation of the infarcted area. Also, local loss of contractility (causing stasis)

- This results in mural thrombosis &, potentially, systemic thromboembolism

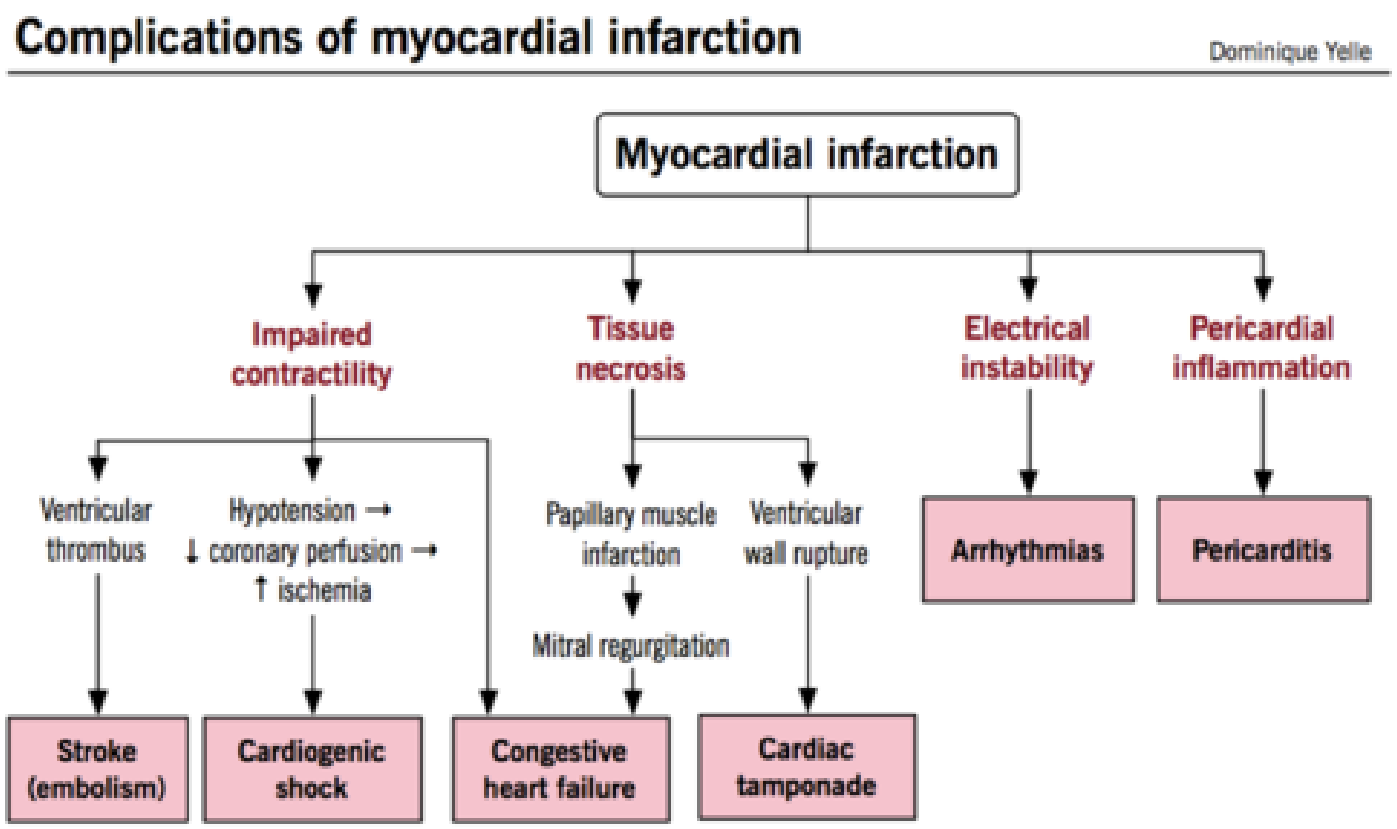


Ventricular aneurysm

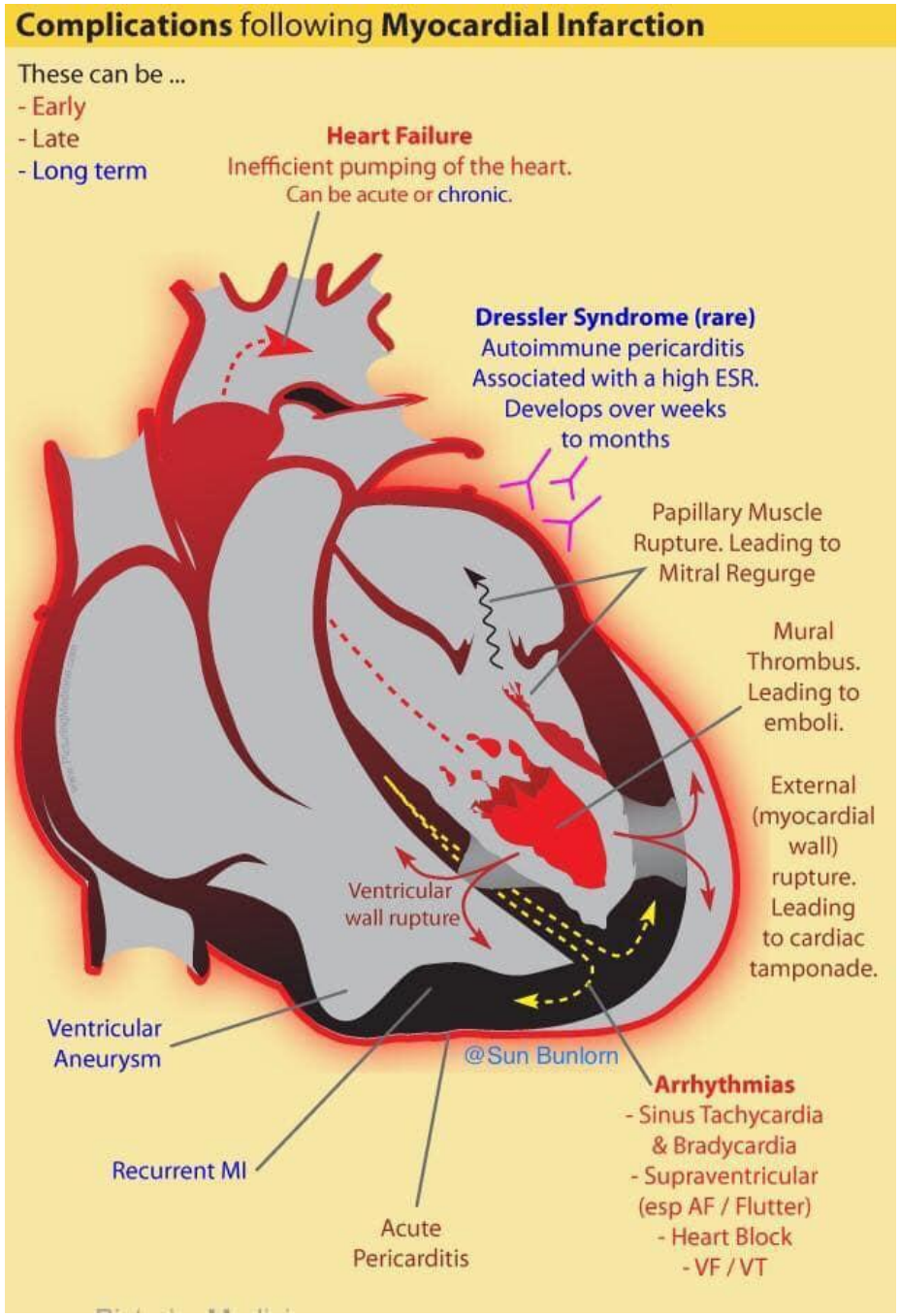
- ✓Ventricular aneurysm is a late complication
- ✓ aneurysms of the ventricular wall most commonly result from a large transmural MI that heals with the formation of thin scar tissue
- ✓Complications of ventricular aneurysms include mural thrombus, arrhythmias & heart failure
- ✓Rupture of the fibrotic aneurysmal wall usually does not occur.



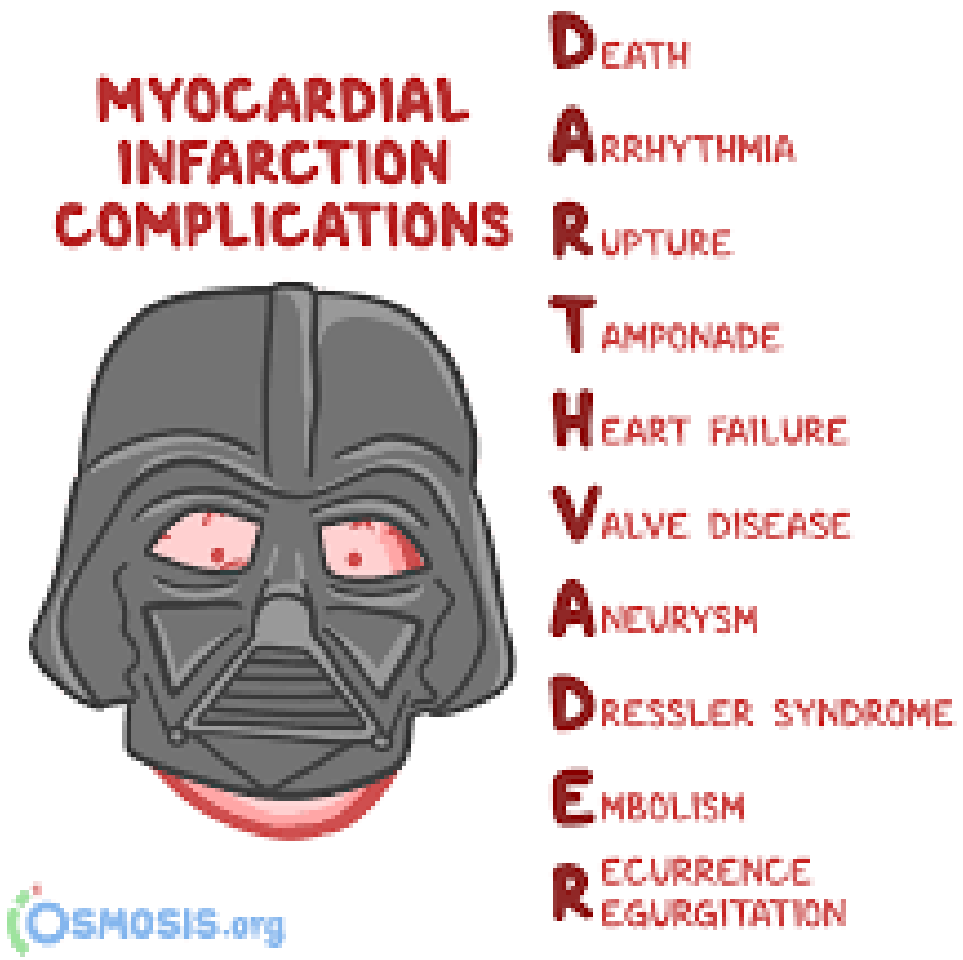
Summary of MI complications



Summary of MI complications



DARTH VADER


MYOCARDIAL INFARCTION COMPLICATIONS
DEATH
ARRHYTHMIA
RUPTURE
TAMPONADE
HEART FAILURE
VALVE DISEASE
ANEURYSM
DRESSLER SYNDROME
EMBOLISM
RECURRENCE
REGURGITATION
OSMOSIS.org

Chronic Ischemic Heart Disease

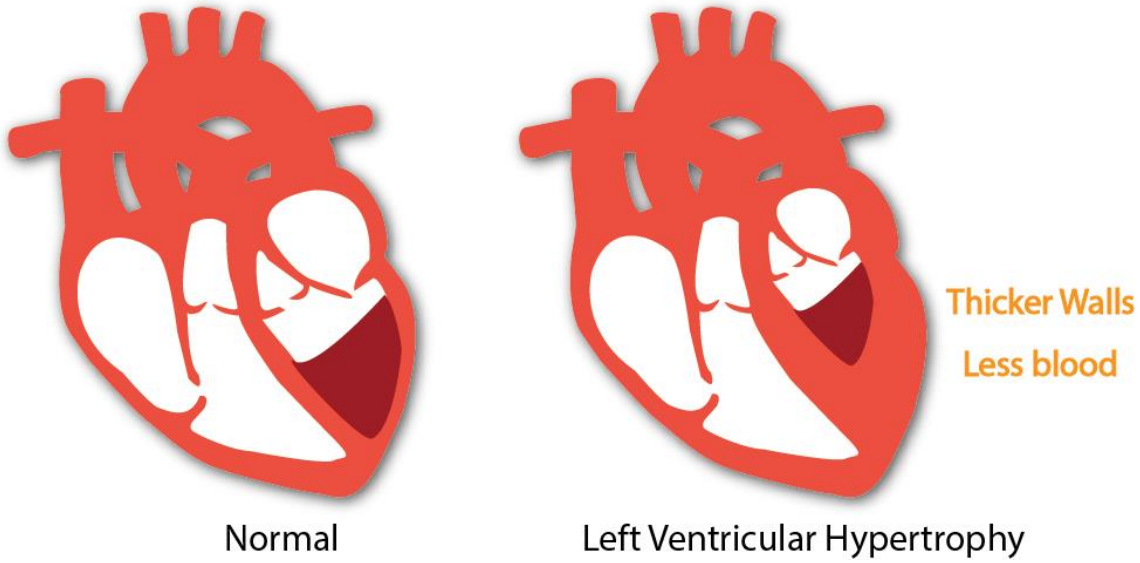
- Also called **ischemic cardiomyopathy**
- **Progressive heart failure secondary to ischemic myocardial damage.**
- History of previous MI
- Appears when the compensatory mechanisms (e.g., hypertrophy) of residual viable myocardium begin to fail.
- **Left ventricular dilation and hypertrophy,** often with discrete areas of gray-white **scarring** from previous healed infarcts.

Hypertensive Heart Diseases

Hypertensive Heart Disease

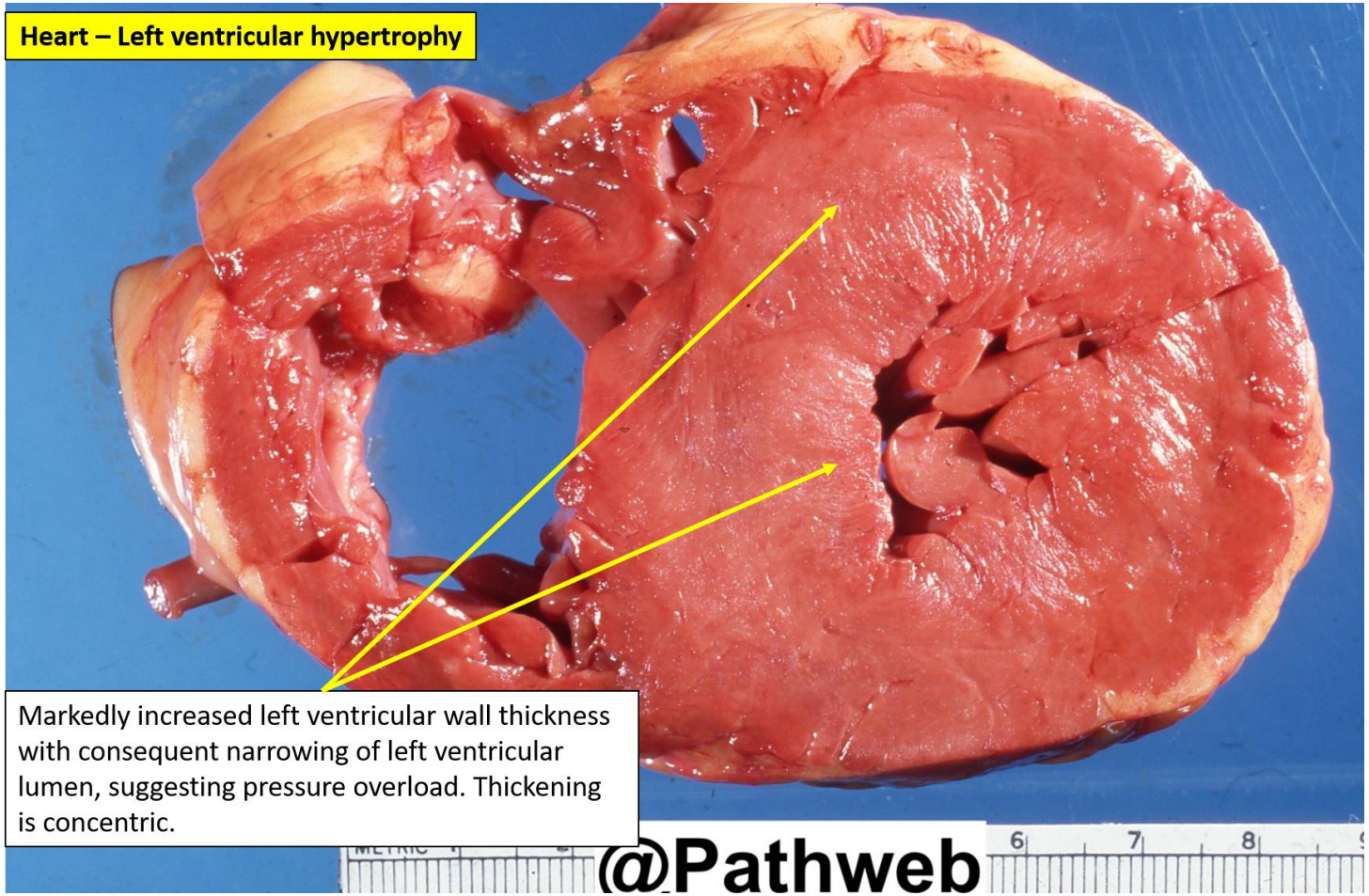
- Major cardiac complications of hypertension, result from **pressure overload**
- Myocyte hypertrophy is an adaptive response, but there are limits
- Persistent hypertension eventually can culminate in dysfunction, cardiac dilation, CHF, and even sudden death.
- Systemic hypertension: affects the left side of the heart
- Pulmonary hypertension: can cause right-sided hypertensive changes called **cor pulmonale.**

Systemic (Left-Sided) Hypertensive Heart Disease



(1) left ventricular hypertrophy in the absence of other cardiovascular pathology (e.g., valvular stenosis)

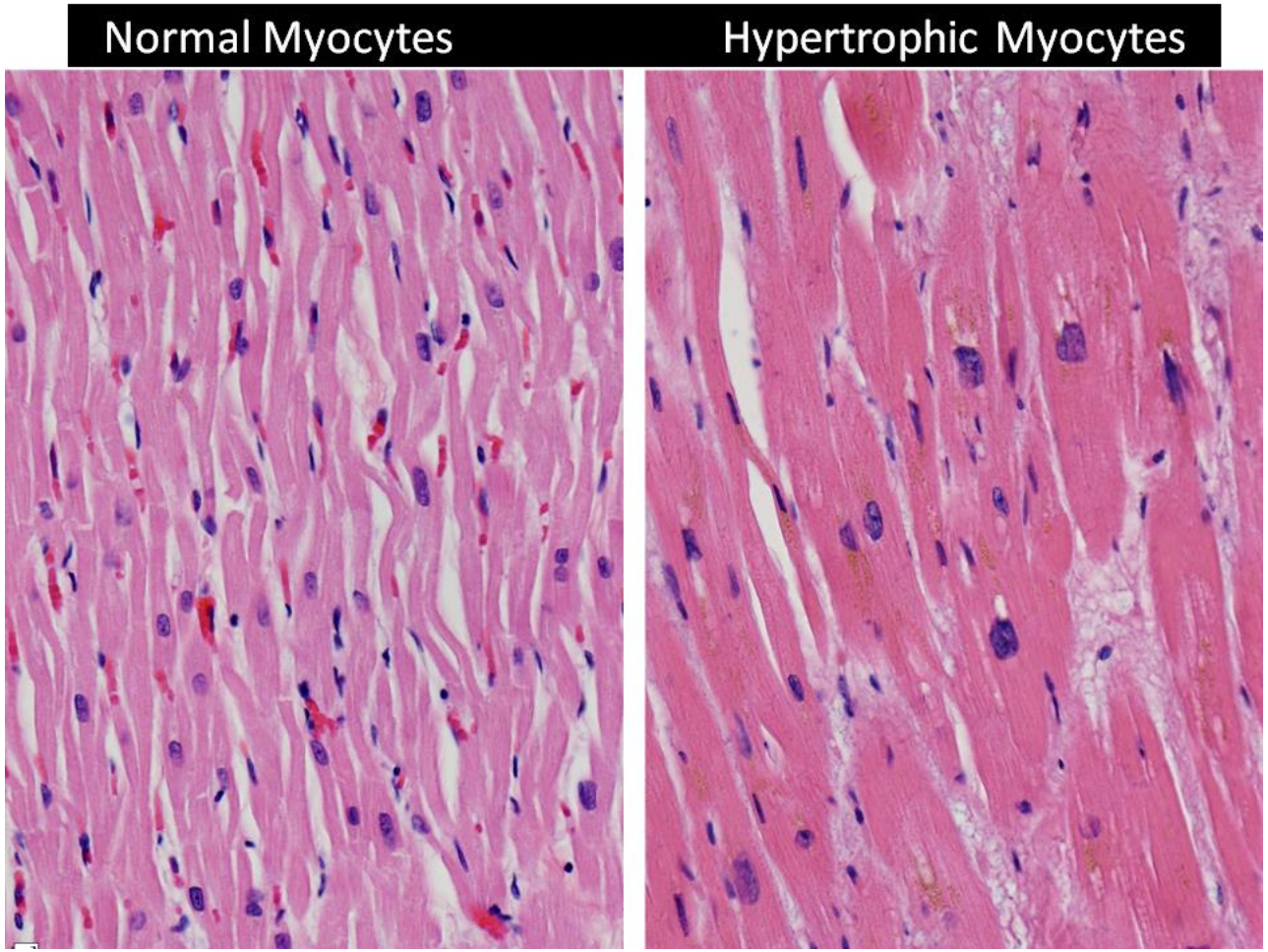
(2) a history or pathologic evidence of hypertension.



Morphology Gross

Left ventricular hypertrophy:

- No ventricular dilation until very late in the process
- Heart weight can exceed 500 g (normal, 320 - 360 g)
- Left ventricular wall thickness can exceed 2.0 cm (normal, 1.2 - 1.4 cm).



Morphology
Histology

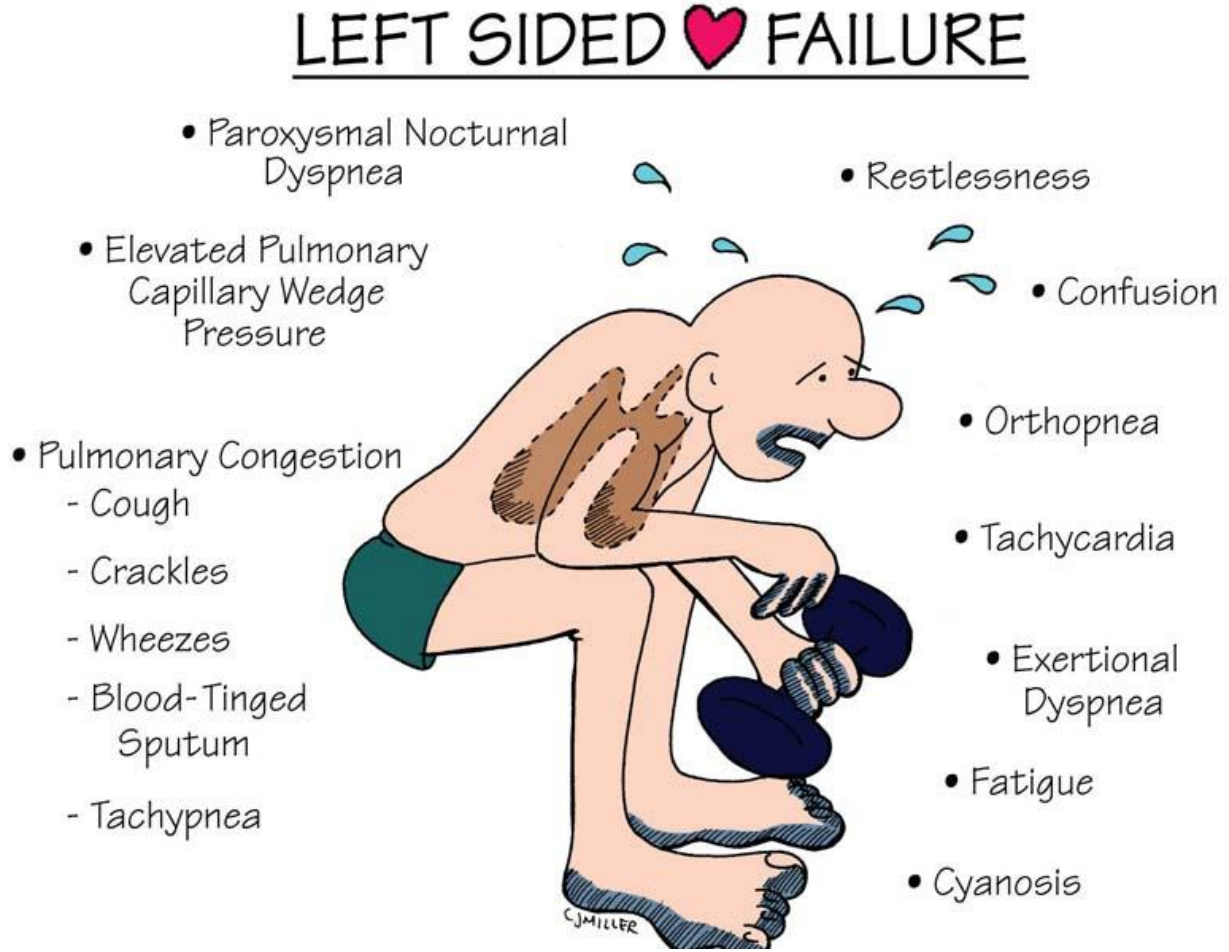
Microscopically:

- Transverse diameter of myocytes is increased
- Prominent nuclear enlargement and hyperchromasia
- Intercellular fibrosis.

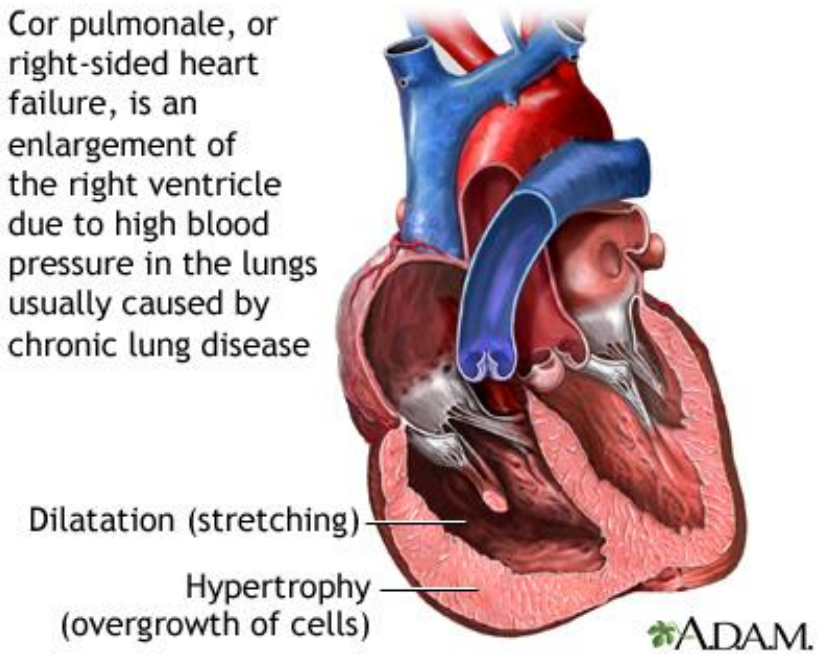
Clinical Features:

- Compensated hypertensive heart disease typically is asymptomatic
- The disease can comes to attention with the onset of atrial fibrillation and/or CHF.

Left-sided heart failure symptoms



Pulmonary Hypertensive Heart Disease: Cor Pulmonale

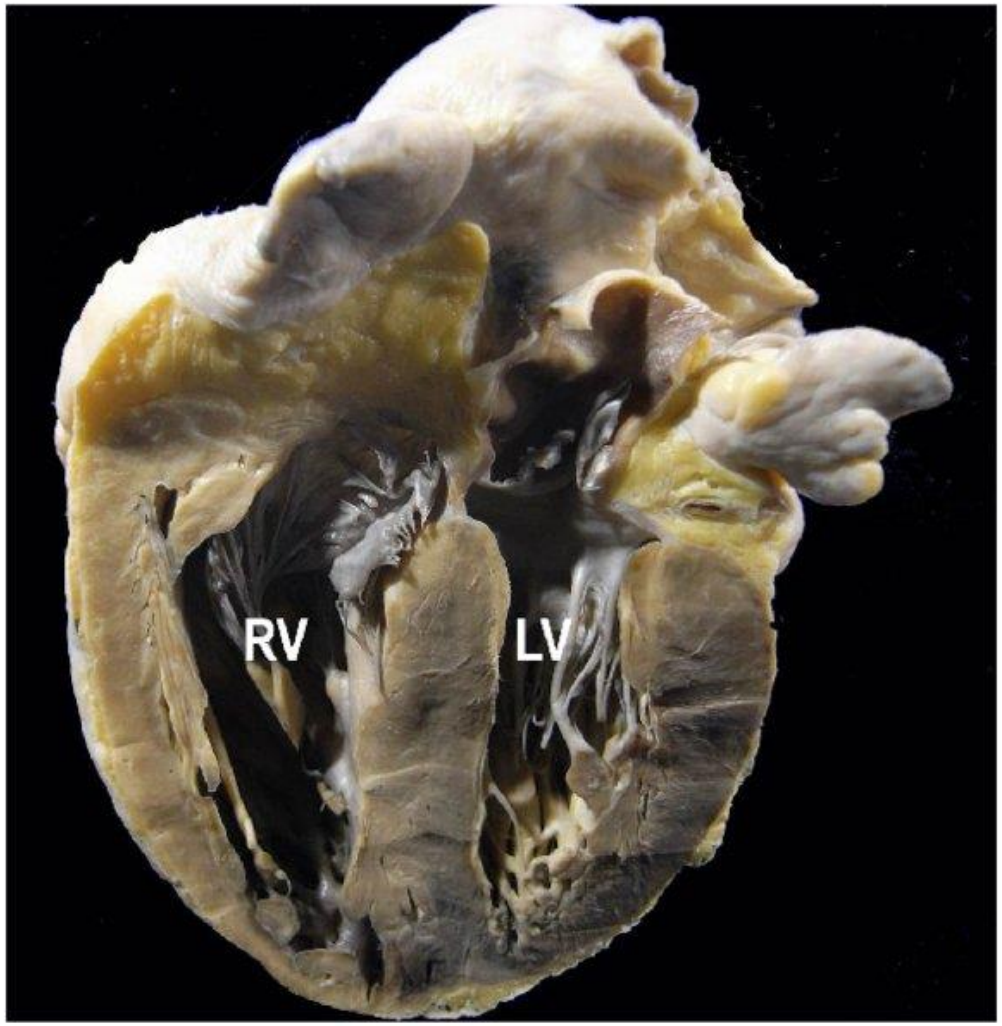


Cor Pulmonale is right ventricular hypertrophy and dilation, frequently accompanied by right heart failure.

Cause of chronic cor pulmonale:

- Primary disorders of the lung parenchyma
- Disorder of pulmonary vasculature

Acute: pulmonary embolism



Chronic cor pulmonale: Right ventricular hypertrophy

MORPHOLOGY

Acute cor pulmonale:

- Right ventricle usually shows only dilation
- If an embolism causes sudden death, the heart may even be of normal size.

Chronic cor pulmonale:

- Right ventricular hypertrophy
- When ventricular failure develops, the right ventricle and atrium are dilated.

Right-sided heart failure symptoms