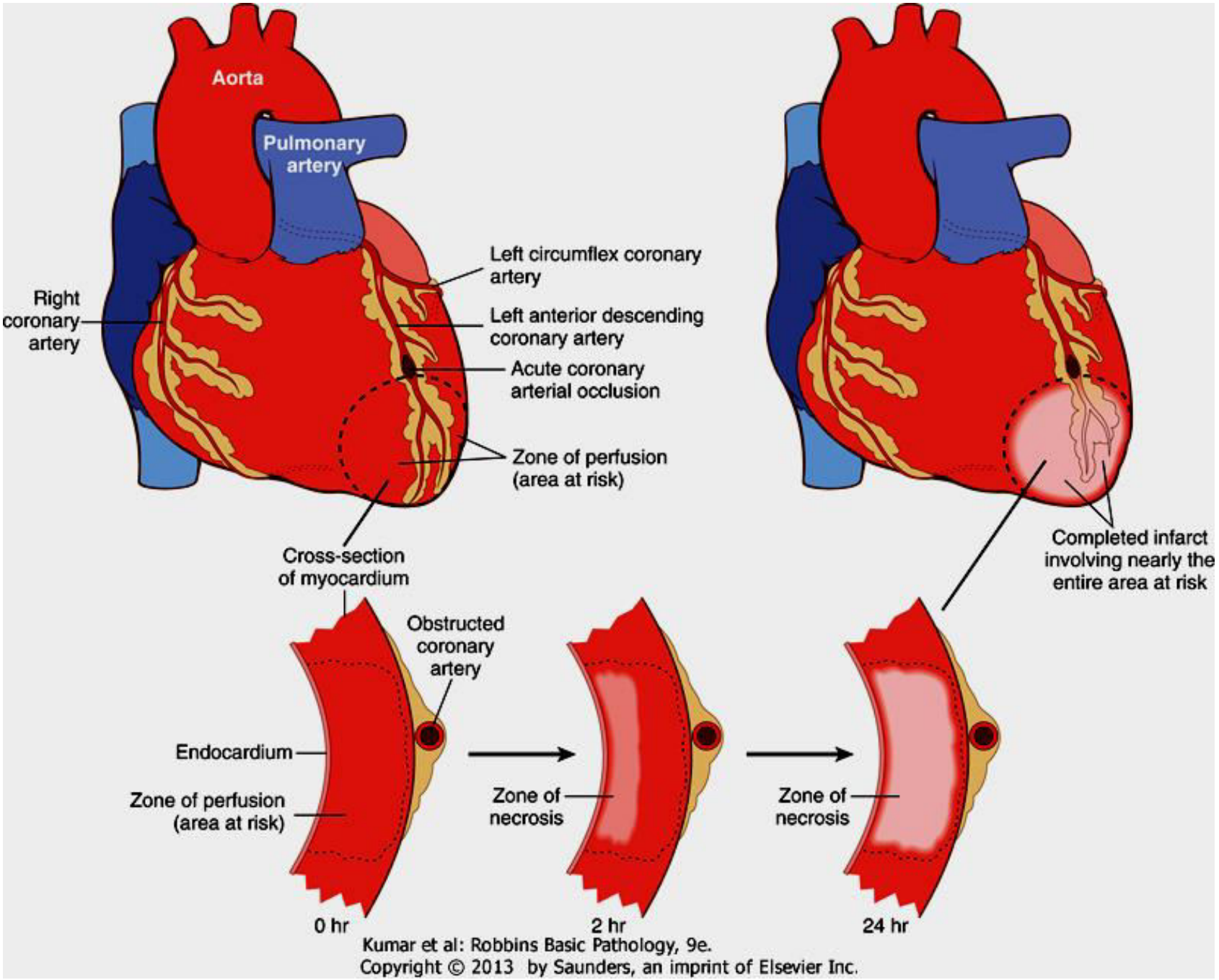
Myocardial Infarction

- MI = *heart attack*
- *Defined as necrosis of heart muscle resulting from ischemia.*
- A very significant cause of death worldwide.
- of these deaths, 33% -50% die before they can reach the hospital➔ lethal arrhythmia ➔ Sudden Cardiac Death
- **Arrhythmias are caused by electrical abnormalities of the ischemic myocardium and conduction system.**

- Acute occlusion of the proximal left anterior descending (**LAD**) artery is the cause of *40% to 50% of all MIs* and typically results in infarction of the anterior wall of the left ventricle, the anterior two thirds of the ventricular septum, and most of the heart apex

- *The frequency* of MIs *rises progressively with increasing age* and presence of other risk factors such as hypertension, smoking, and diabetes
- **Approximately only 10% of MIs occur in people younger than 40 years.**



Kumar et al: Robbins Basic Pathology, 9e.

Evaluation of MI

- *Clinical signs and symptoms*
- *Electrocardiographic(ECG) abnormalities*
- ***Laboratory evaluation:***

  is based on measuring the blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes.
- **these molecules include :**

1-myoglobin.

2-cardiac troponins T and I (TnT, TnI)

3-creatine kinase (CK, and more specifically the myocardial-specific isoform, CK-MB)

4- lactate dehydrogenase

Cardiac enzymes in MI

- **Cardiac troponins T and I (TnT, TnI), are** the **best** markers for acute MI. **persistence of elevated troponin levels for approximately 10 days allows the diagnosis of acute MI long after CK-MB levels have returned to normal.**

- **creatine kinase** CK-**MB** is the **second best** marker after the cardiac-specific troponins.

- Since various forms of **creatine kinase** (CK) are found in brain, myocardium, and skeletal muscle, total CK activity is not a reliable marker of cardiac injury (i.e. it could come from skeletal muscle injury). Thus, the **CK-MB** isoform-principally derived from myocardium is the more specific indicator of heart damage.
- CK-MB activity begins to rise within 2-4 hours of MI, peaks at 24-48 hours, and returns to normal within approximately 72 hours.

Microscopic changes of MI **and its repair.**

(<24 hr)➔ coagulative **necrosis** and **wavy fibers**. Necrotic cells are separated by edema fluid.

2- to 3-day old - infarct➔ Dense **neutrophil** infiltrate

(7 to 10 days)➔ complete removal of necrotic myocytes by phagocytic **macrophages**

up to 14 days➔ **Granulation tissue** characterized by loose connective tissue and abundant capillaries.

several weeks➔ Healed myocardial infarct consisting of a dense collagenous **scar**.

Microscopic features of myocardial infarction and its repair.

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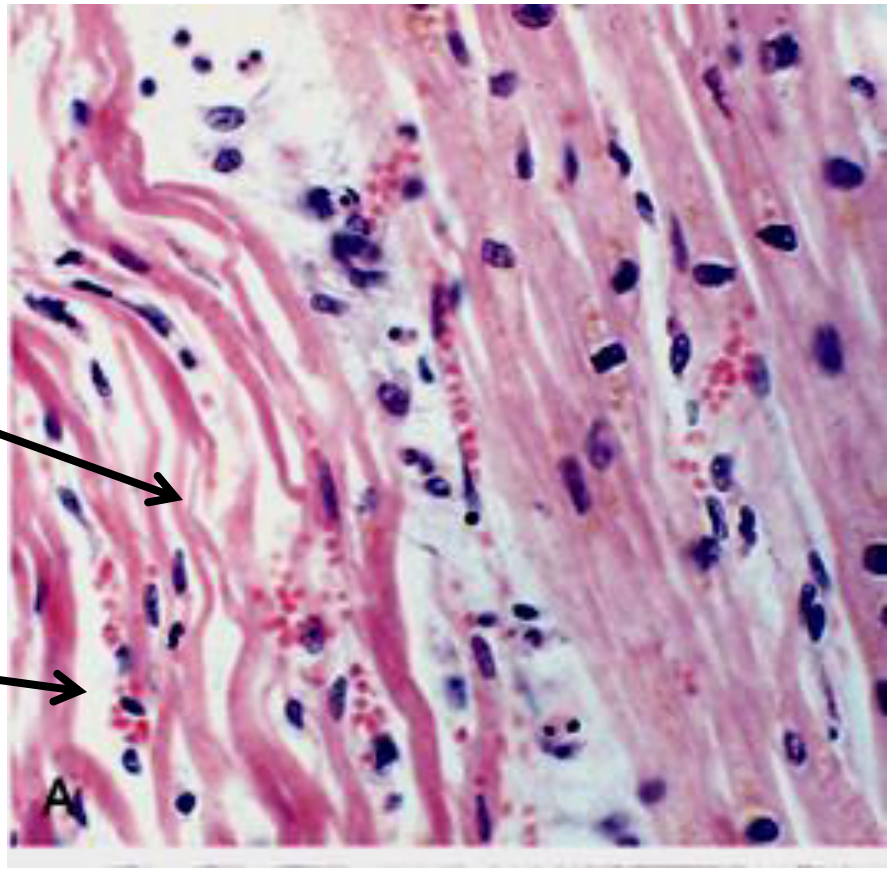
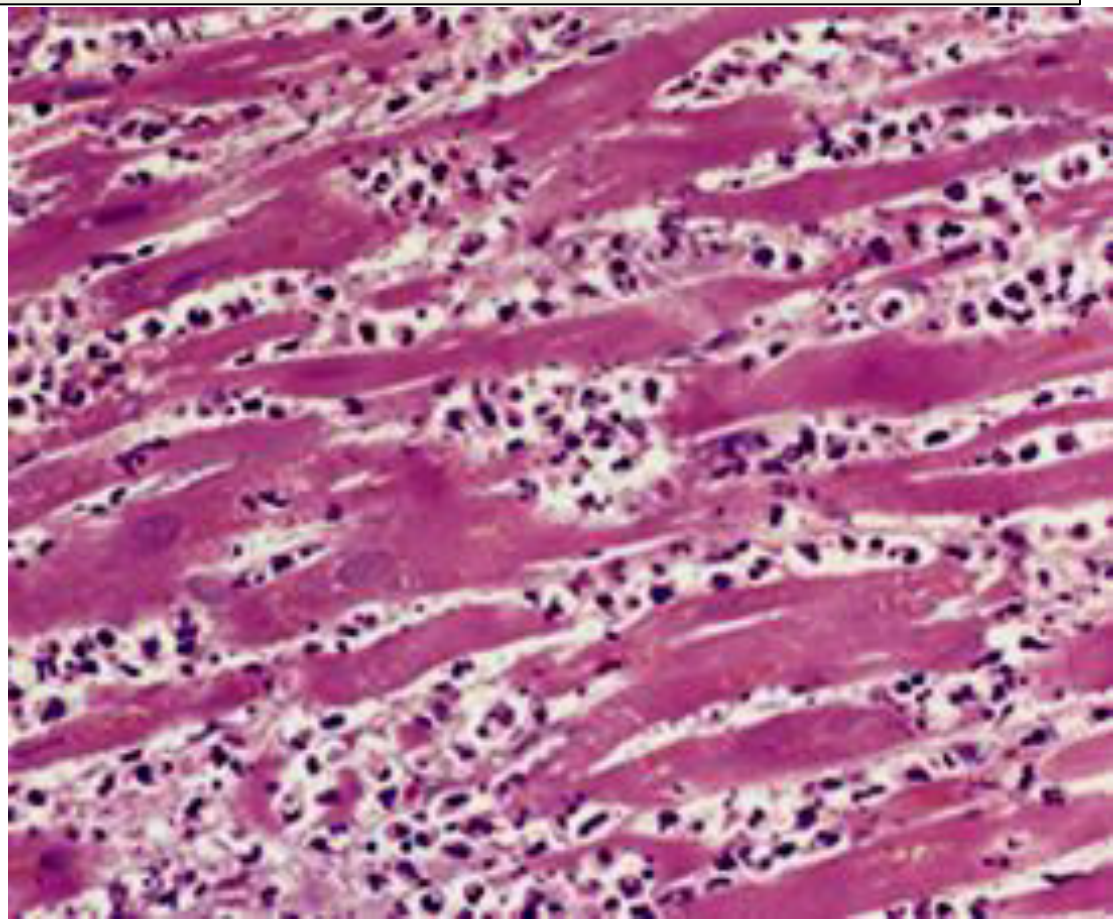


Microscopic features of myocardial infarction and its repair.

2- to 3-day old - infarct → Dense **neutrophil** infiltrate

- in case of reperfusion → contraction bands



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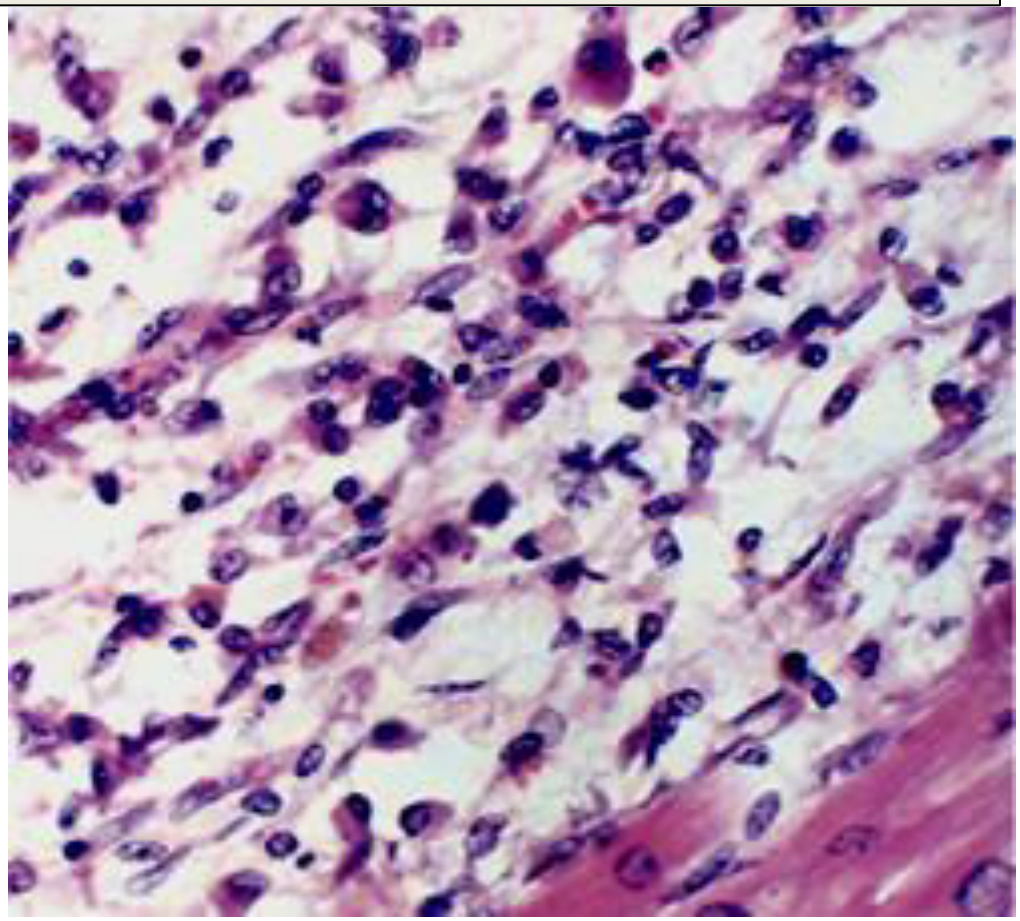
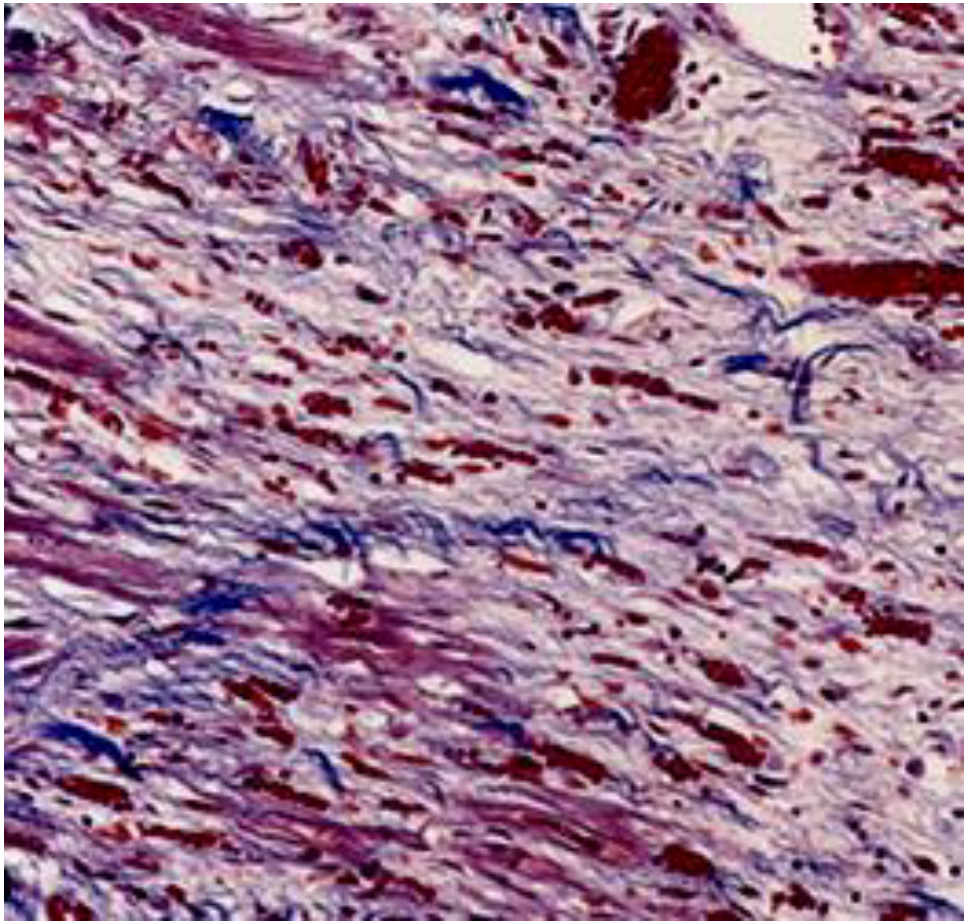


Microscopic features of myocardial infarction and its repair.

up to 14 days➔ Granulation tissue characterized by loose connective tissue (blue) and abundant capillaries (red)



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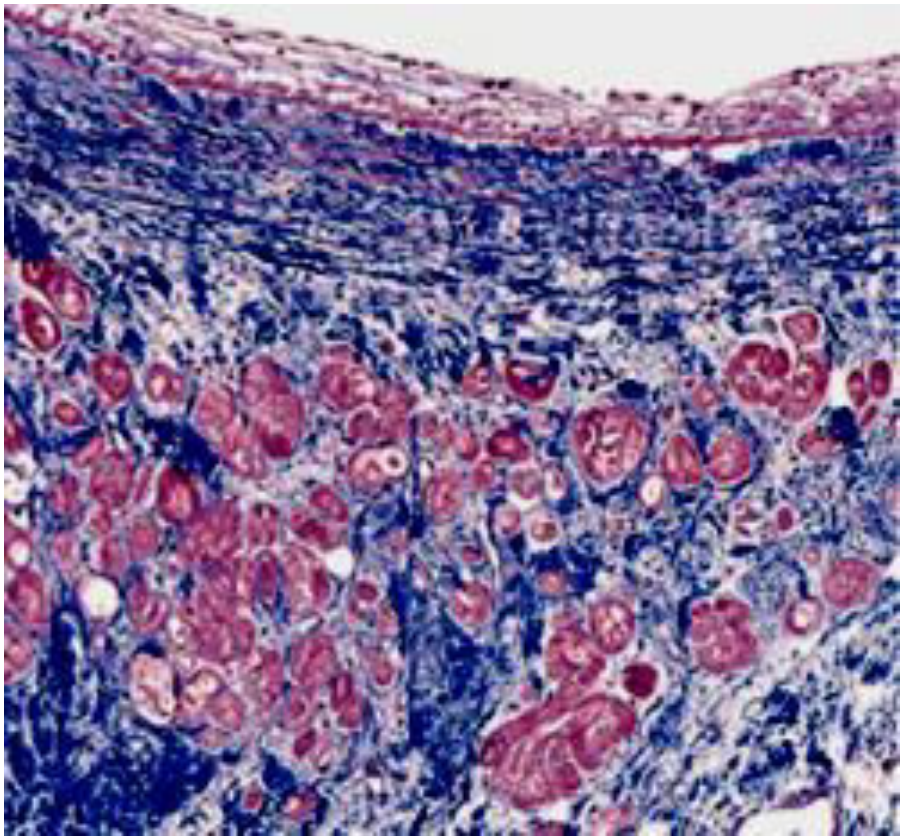


Consequences and Complications of MI

- **1- Death**: Unfortunately, **50% of the deaths** associated with acute MI occur in individuals who never reach the hospital (within 1 hour of symptom onset-usually as a result of arrhythmias)
- Extraordinary progress has been made in patient outcomes subsequent to acute MI (the*in-hospital) death rate* has declined from approximately 30% to an overall rate of between 10% and 13%).

Consequences and Complications of MI

- ***2- cardiogenic shock.***

- (10% to 15%) of patients after acute MI
- with a large infarct (>40% of the Left ventricle).
- 70% mortality rate; 2/3 of in-hospital deaths.

- ***3-Myocardial rupture***
- ***4-Pericarditis.***
- ***5-Infarct expansion***
- ***6-Ventricular aneurysm***
- ***7-Progressive late heart failure***

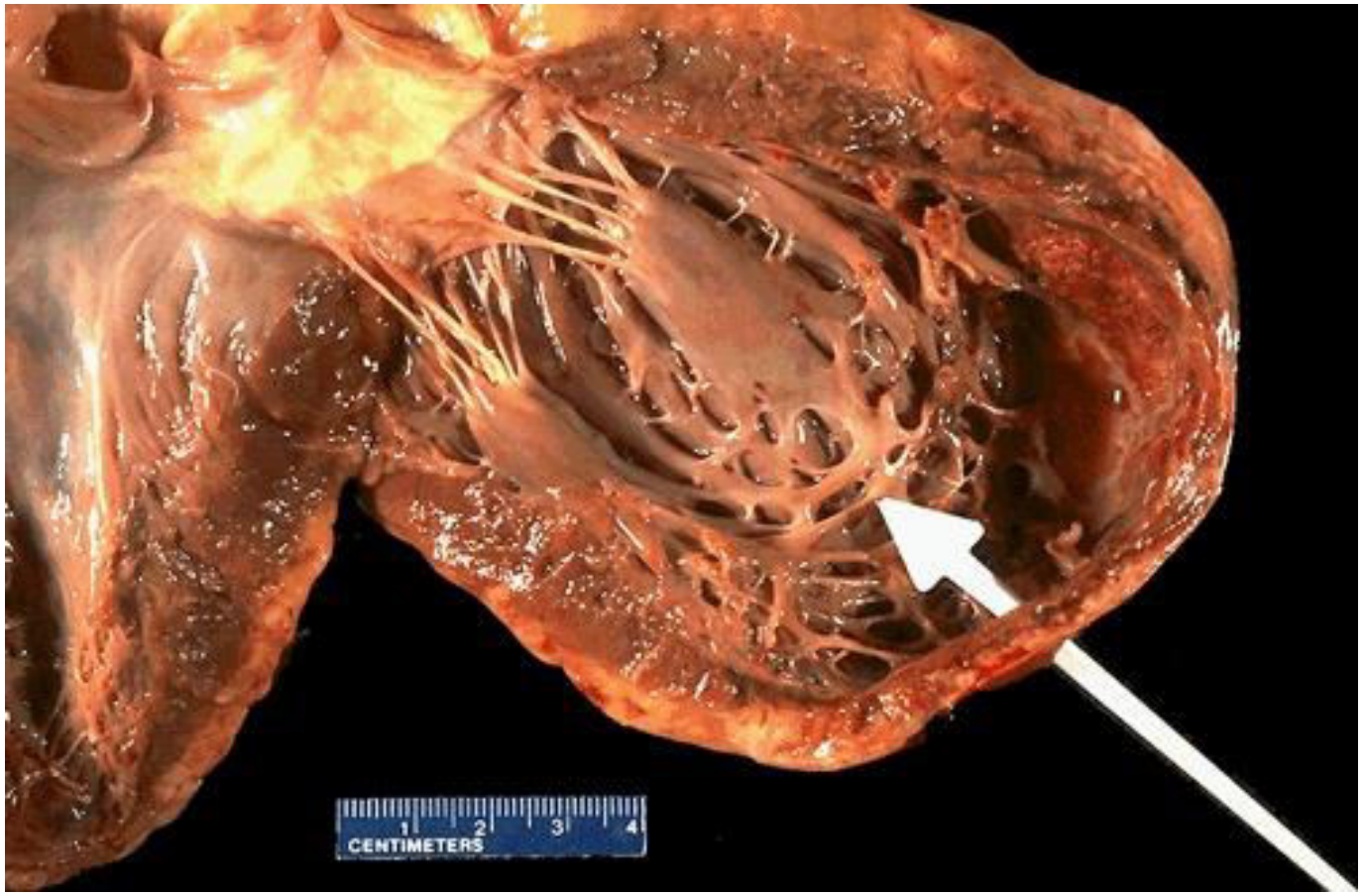
Complications of myocardial rupture include:

(1) rupture of the ventricular free wall→ hemopericardium and cardiac tamponade (usually fatal)

(2) rupture of the ventricular septum→ VSD and left-to-right shunt

(3) papillary muscle rupture→ severe mitral regurgitation

myocardial rupture



- ***4-Pericarditis.***

  - fibrinous or hemorrhagic pericarditis

  - usually 2 to 3 days of a transmural MI

  - typically spontaneously resolves with time (immunologic mechanism).

- ***5-Infarct expansion.***

  Because of the weakening of necrotic muscle, there may be disproportionate stretching, thinning, and dilation of the infarct region (especially with anteroseptal infarcts)

- ***6-Mural thrombus.***

  -the combination of a local loss of contractility (causing stasis) + endocardial damage (causing a thrombogenic surface) → ***thromboembolism***

- ***7-Ventricular aneurysm.***

  - A late complication

  - most commonly result from a large transmural anteroseptal infarct that heals with the formation of thin scar tissue

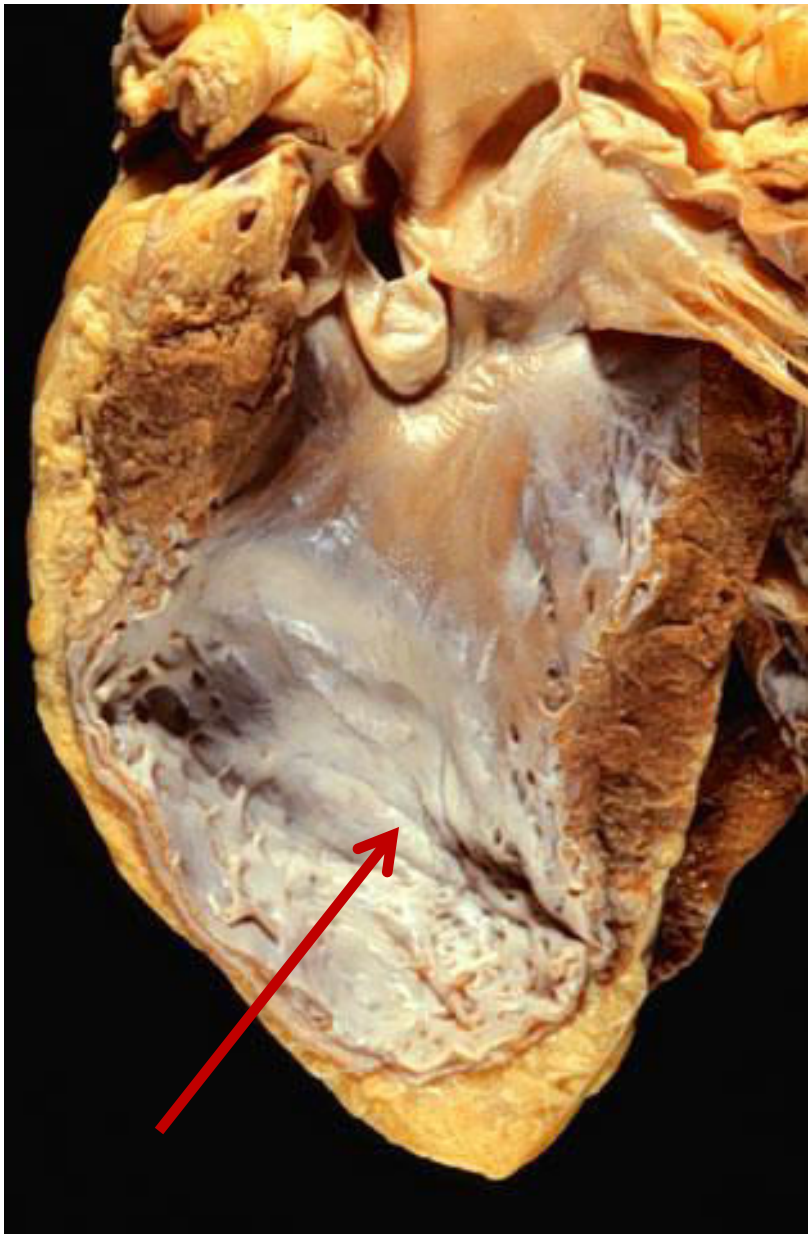
Ventricular aneurysm

Complications of ventricular aneurysms include:

1-mural thrombus

2-arrhythmias

3-heart failure



- ***8-Papillary muscle dysfunction*** (post-infarct mitral regurgitation)
- dysfunction of a papillary muscle after MI occurs due to:

1- rupture.

2- ischemic dysfunction

3- fibrosis and shortening

4- ventricular dilation.

- ***9-Progressive late heart failure***

Long-term prognosis after MI

- depends on many factors, the most important of which are left ventricular function and the severity of atherosclerotic narrowing of vessels perfusing the remaining viable myocardium.
- Mortality rate within the first year =30%
- Thereafter, the annual mortality rate is 3% to 4%.

Chronic Ischemic Heart Disease

- Chronic IHD usually results from **post-infarction** cardiac decompensation that follows exhaustion of the hypertrophic viable myocardium.
- **progressive heart failure** as a consequence of ischemic myocardial damage; sometimes punctuated by episodes of angina or MI.
- Arrhythmias are common along with **CHF**

Sudden Cardiac Death (SCD)

- Affecting some 300,000 to 400,000 individuals annually in the United States
- SCD is most commonly defined as **unexpected death from cardiac causes either without symptoms or within 1 to 24 hours of symptom onset** . The most common mechanism is ventricular fibrillation.
- **Coronary artery disease is the most common underlying cause**
- In many adults SCD is the first clinical manifestation of IHD.
- With **younger** victims, other **non-atherosclerotic** causes are more common:

Other non-atherosclerotic causes of SCD

- Congenital coronary arterial abnormalities
- Aortic valve stenosis
- Mitral valve prolapse
- Myocarditis
- Dilated or hypertrophic cardiomyopathy
- Pulmonary hypertension
- Hereditary or acquired abnormalities of the cardiac conduction system.
- Isolated myocardial hypertrophy.
- unknown causes.