Clinical Features of MI

1) Severe, crushing substernal chest pain
2) radiate to the neck, jaw, epigastrium, or left arm.
3- rapid and weak pulse
4- nausea (posterior MI).
5- cardiogenic shock (massive MIs >40% of the left ventricle)
6- dyspnea (pulmonary congestion and edema)

- 10% to 15% of MIs are asymptomatic (**silent infarcts**)➔ particularly in patients with:

  1- DM (peripheral neuropathies)

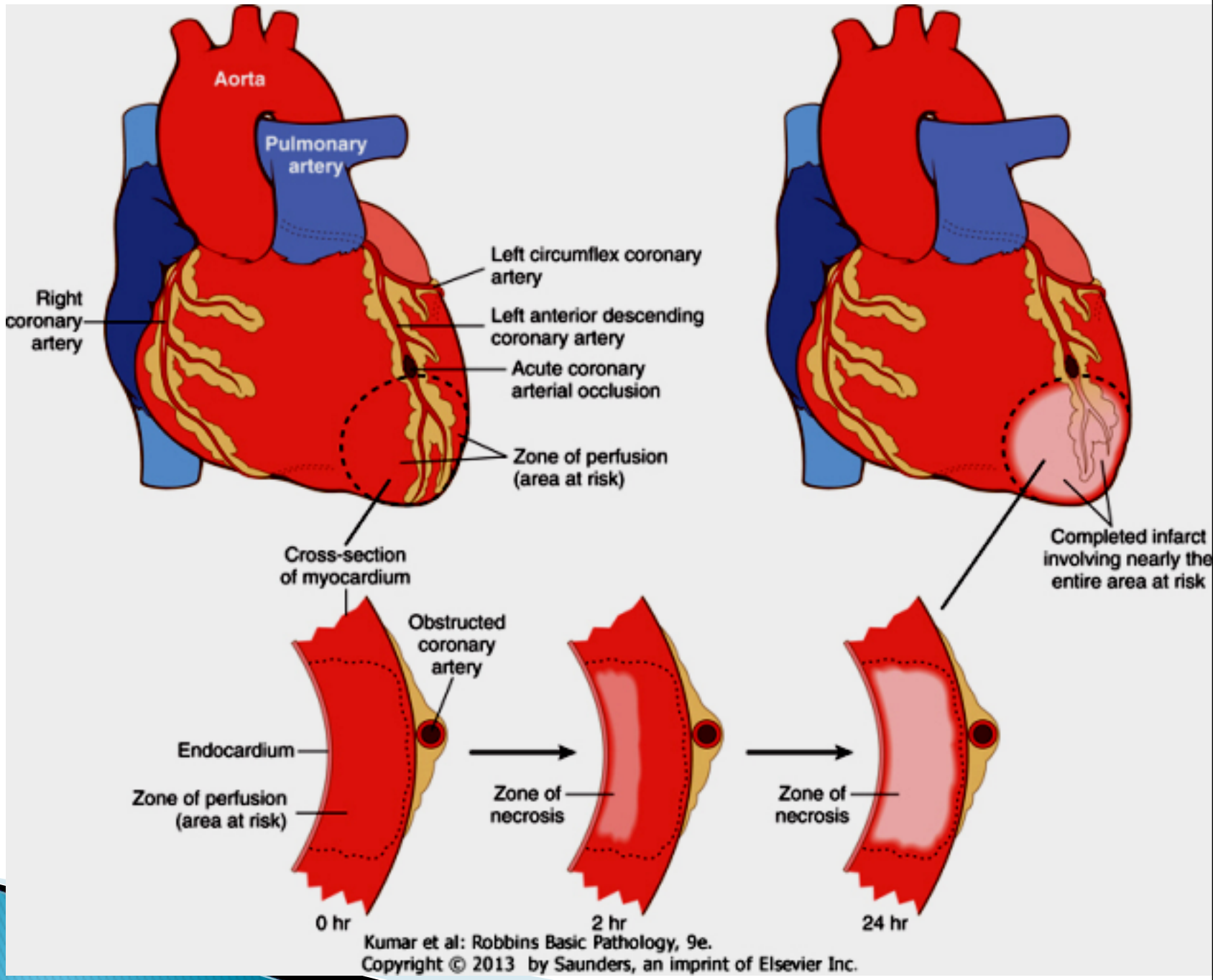
  2- the elderly

Myocardial Infarction

- MI = *heart attack*
- *necrosis of heart muscle due to ischemia.*
- A very significant cause of death worldwide.
- 33% -50% die before 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*

- *frequency* of MIs *rises with increasing age* and presence of risk factors such as hypertension, smoking, and diabetes


Aorta
Pulmonary artery
Right coronary artery
Left circumflex coronary artery
Left anterior descending coronary artery
Acute coronary arterial occlusion
Zone of perfusion (area at risk)
Cross-section of myocardium
Completed infarct involving nearly the entire area at risk
Obstructed coronary artery
Endocardium
Zone of perfusion (area at risk)
Zone of necrosis
Zone of necrosis
0 hr
2 hr
24 hr
Kumar et al: Robbins Basic Pathology, 9e.
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Evaluation of MI

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

  blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes.

Cardiac enzymes in MI

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 troponins T and I (TnT, TnI), are** the **best** markers for acute MI.
- **creatine kinase** CK-**MB** is the **second best** marker after the cardiac-specific troponins.

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

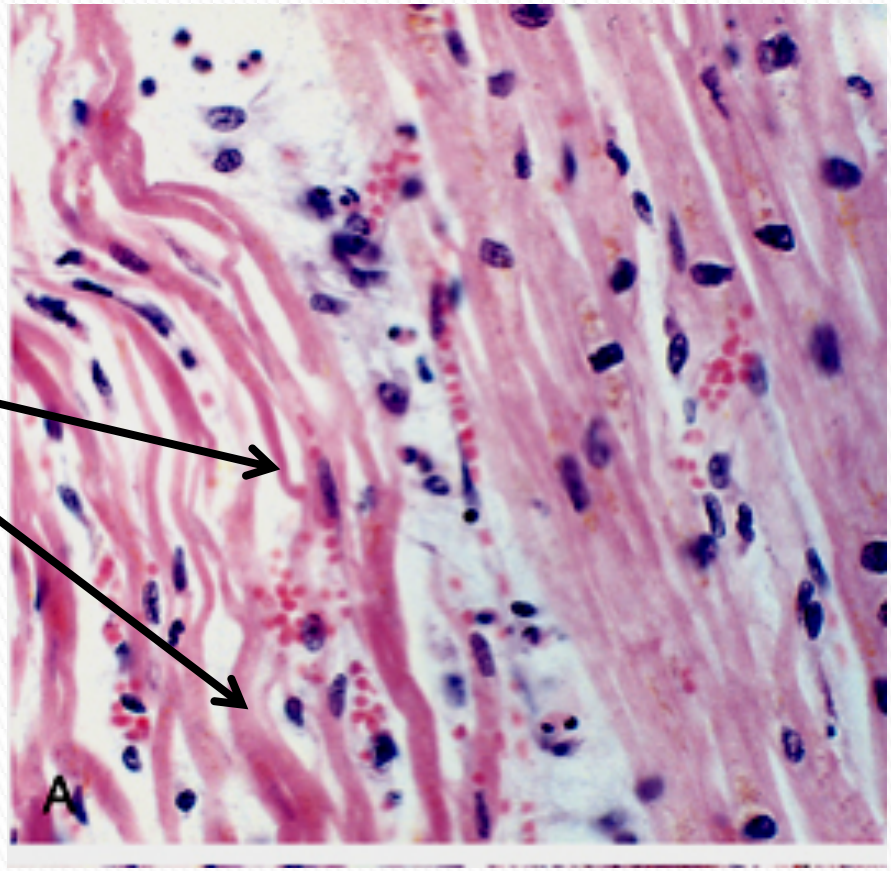
(7 to 10 days)➔ complete removal of necrotic myocytes by phagocytic **macrophages**

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.

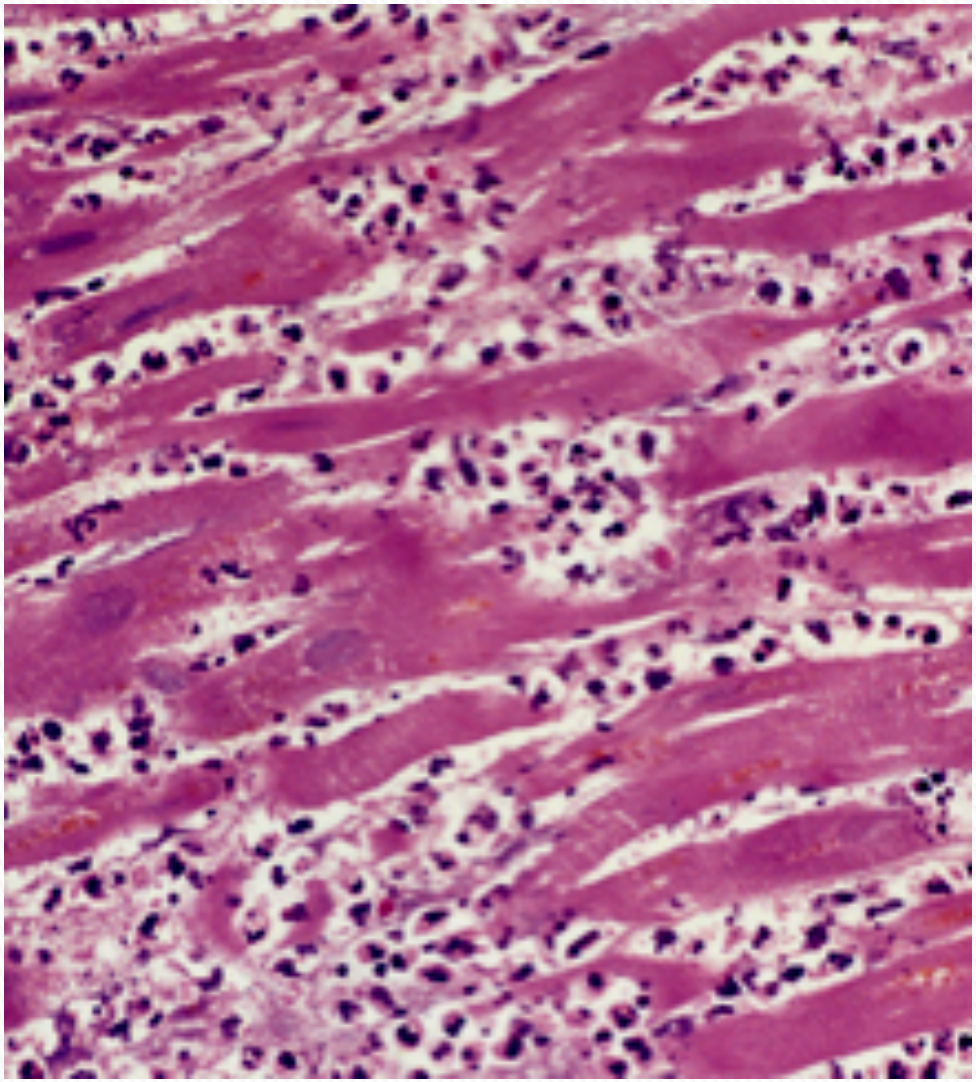
(<24 hr)➔
coagulative **necrosis** and **wavy fibers** Necrotic cells are separated by edema fluid



Microscopic features of myocardial infarction and its repair.

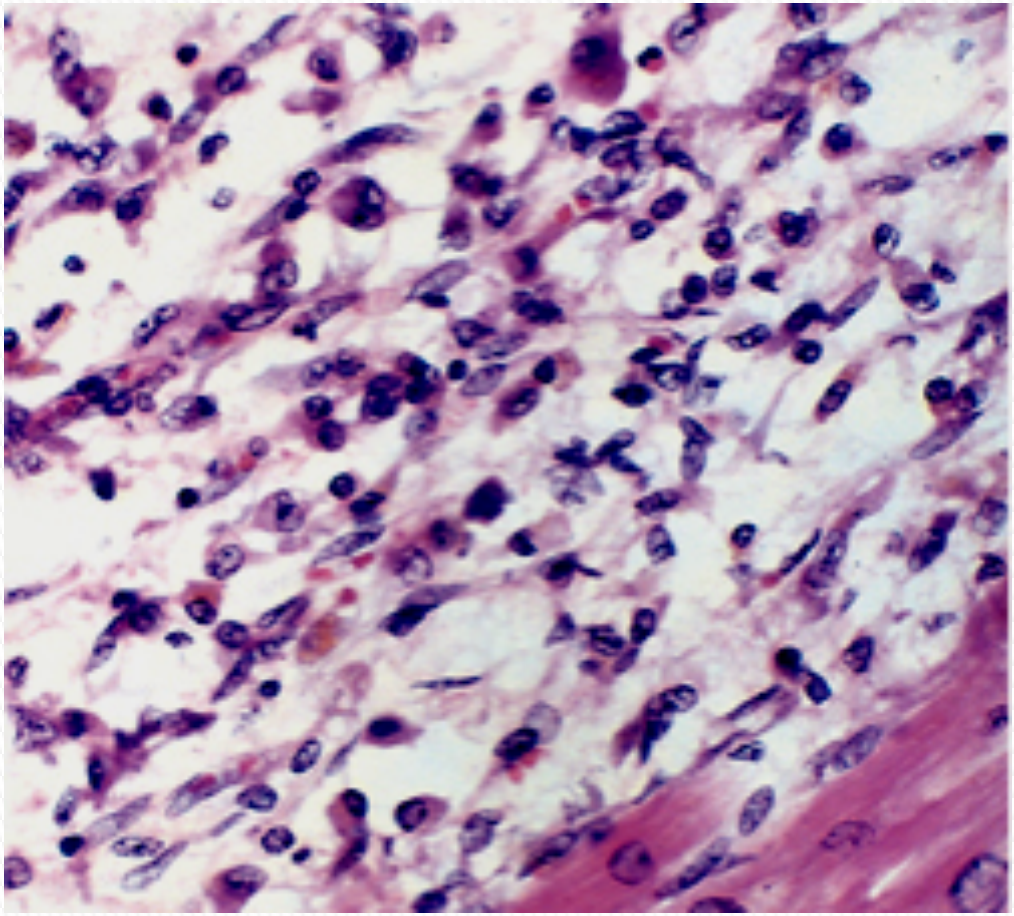
2- to 3-day old - infarct→ Dense **neutrophil** infiltrate

- in case of reperfusion→ contraction bands



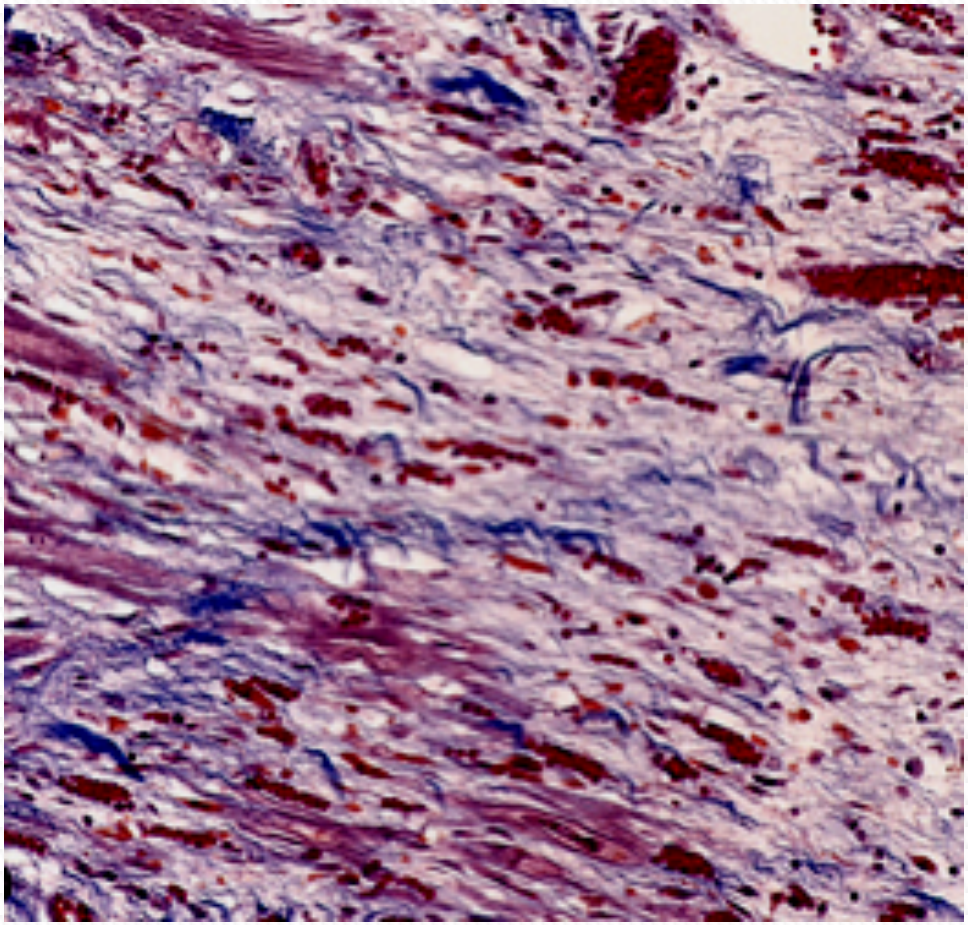
Microscopic features of myocardial infarction and its repair.

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



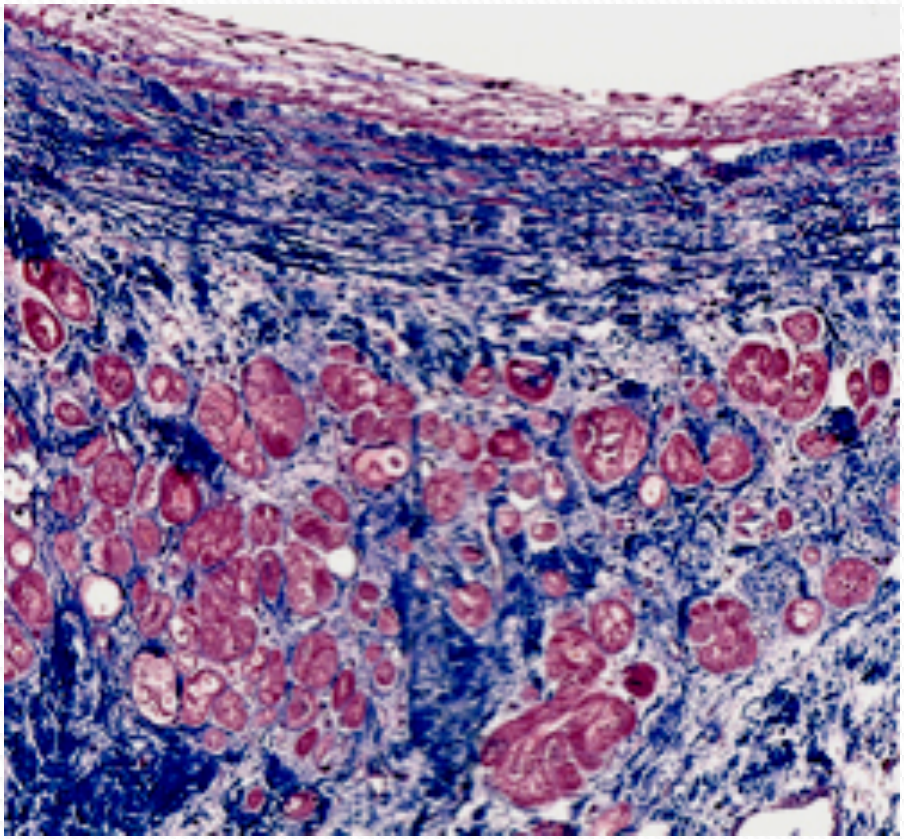
Microscopic features of myocardial infarction and its repair.

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



Microscopic features of myocardial infarction and its repair.

several weeks➔ Healed infarct consisting of a dense collagenous **scar (blue)**



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***

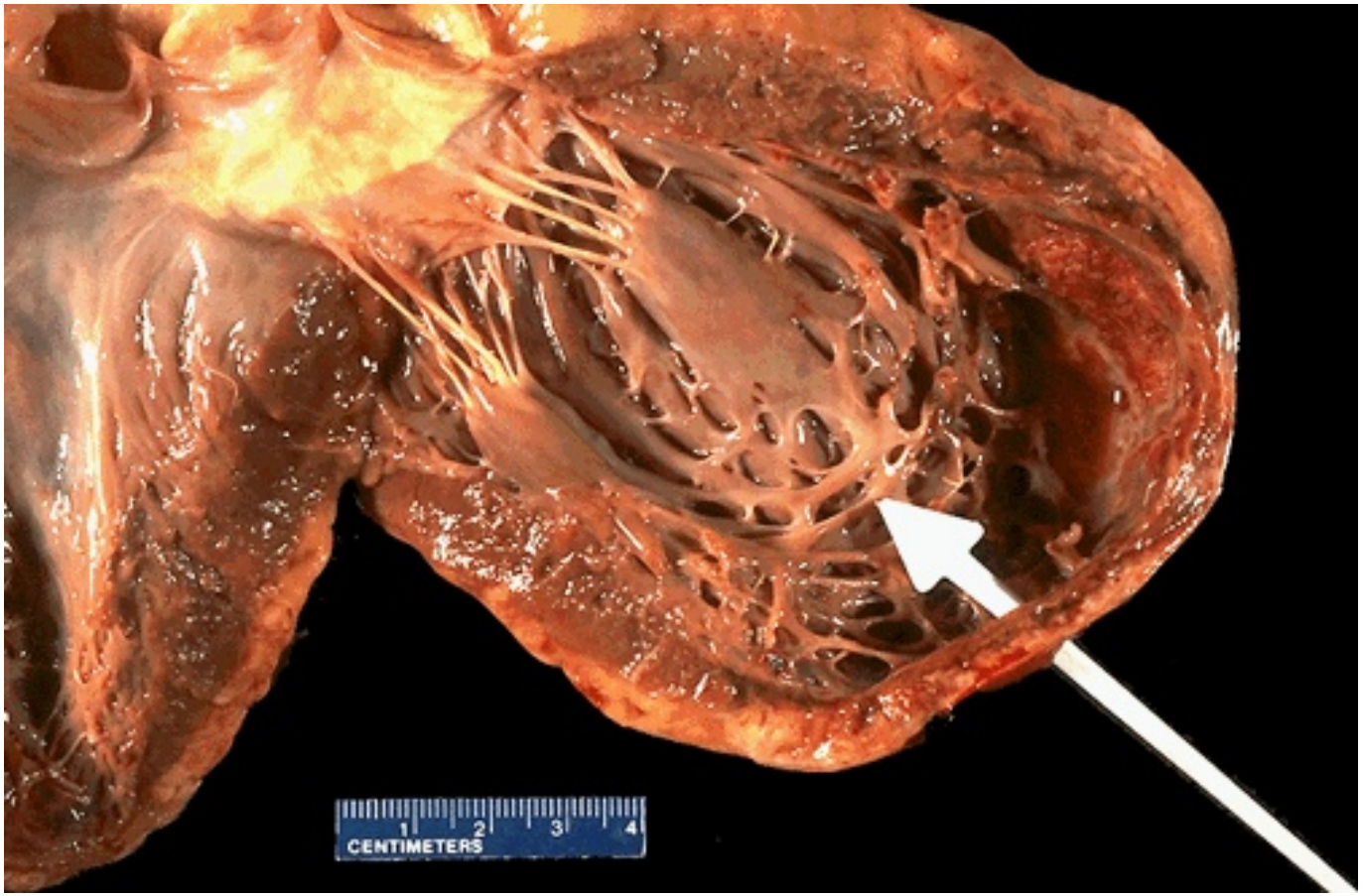
Complications of myocardial rupture include:

(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)

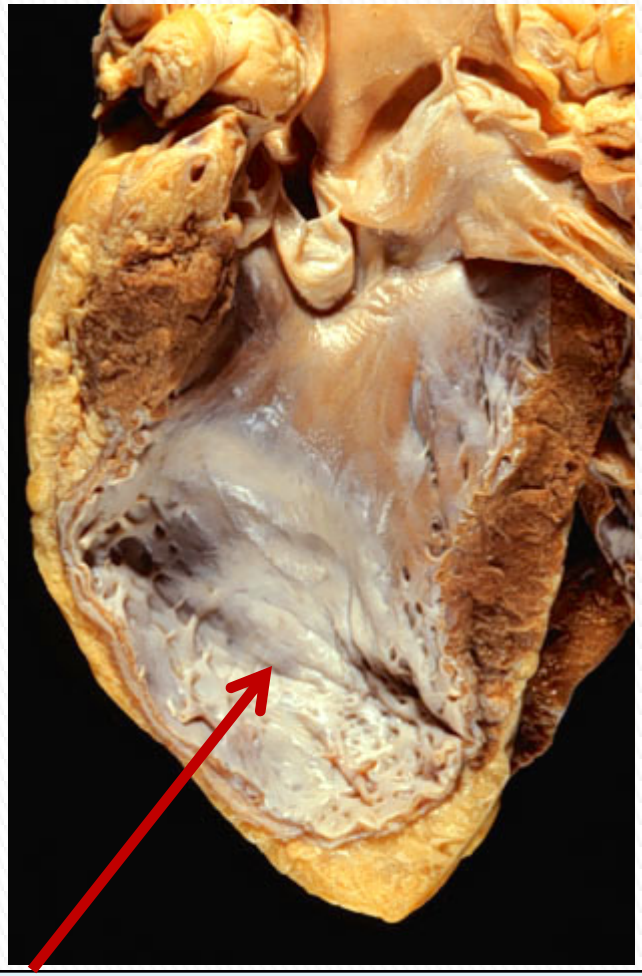
- ***6-Mural thrombus.***

  -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**
- **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.