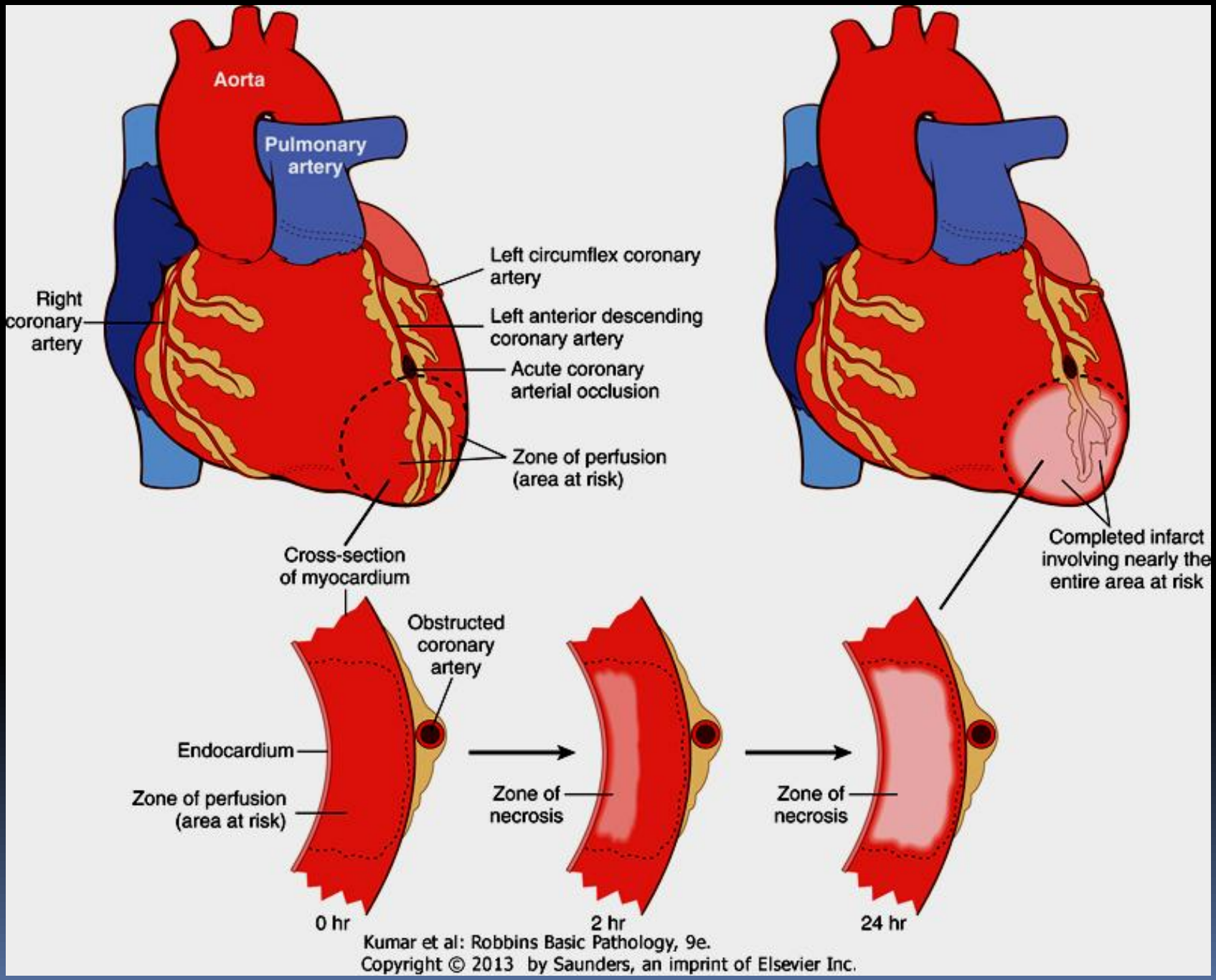
Myocardial Infarction

- 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.

- *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.**

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


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:***

  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.**

- CK-MB is the **second best** marker after the cardiac-specific troponins.
- Since various forms of 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.**

1- (<24 hr)➔ coagulative necrosis and wavy fibers. Necrotic cells are separated by edema fluid.

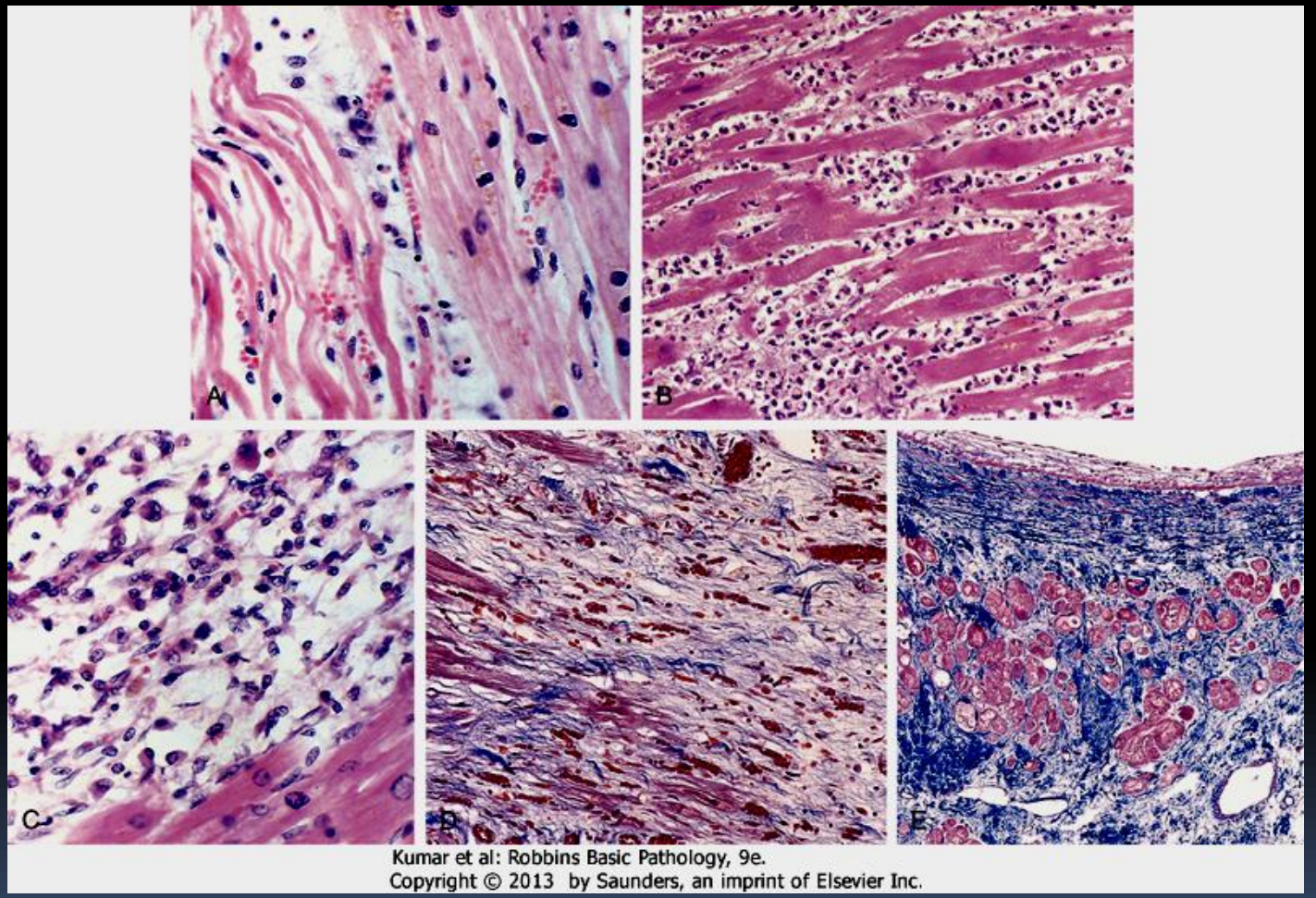
2- (2- to 3-day) - infarct➔ Dense neutrophilic infiltrate

3- (7 to 10 days)➔ complete removal of necrotic myocytes by phagocytic macrophages

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

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

Microscopic features of myocardial infarction and its repair.



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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.
- Since the 1960s 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 lt ventricle).
- 70% mortality rate and (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

- ***4-Pericarditis.***

  - fibrinous or hemorrhagic pericarditis

  - usually within 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) = ***mural thrombosis → 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

Complications of ventricular aneurysms include :

- 1-mural thrombus
- 2-arrhythmias
- 3-heart failure

- ***8-Papillary muscle dysfunction (***postinfarct 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

- progressive heart failure as a consequence of ischemic myocardial damage; sometimes punctuated by episodes of angina or MI.
- Arrhythmias are common along with CHF

- In most instances there is a history of MI.
- Chronic IHD usually results from postinfarction cardiac decompensation that follows exhaustion of the hypertrophic viable myocardium.

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.