Physiology Seminar

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ACUTE MYOCARDIAL INFARCTION

PowerPoint® Seminar Slide Presentation prepared by

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Definition

- **Acute myocardial infarction (AMI),** commonly known as a heart attack, is the irreversible necrosis of heart muscle secondary to prolonged ischemia.
- Results from an imbalance in oxygen supply and demand, caused by plaque rupture with thrombus formation in a coronary vessel, resulting in an acute reduction of blood supply to a portion of the myocardium.

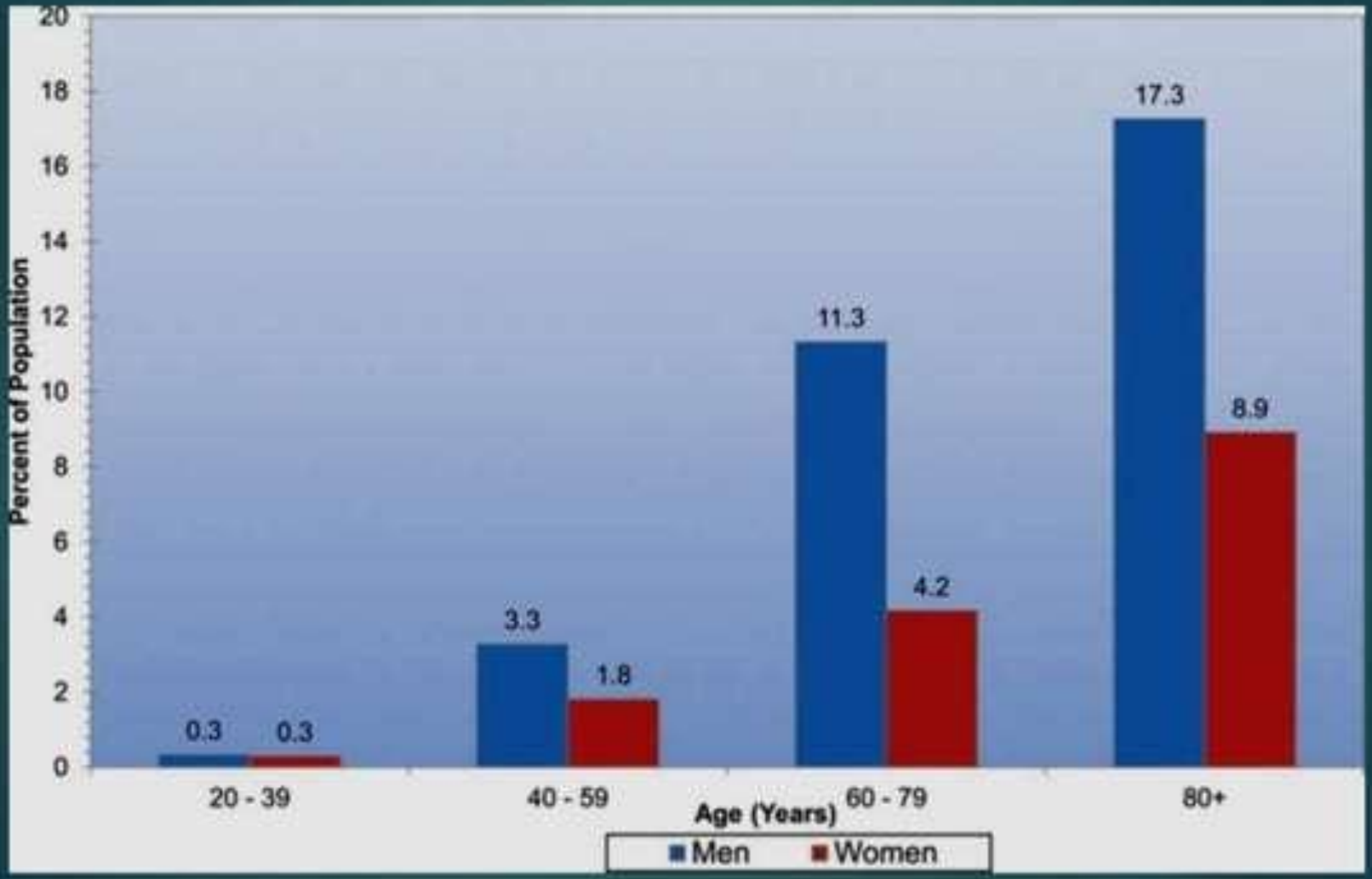
Epidemiology

- Every year about **735,000 Americans** have a heart attack. Of these, 525,000 are a first heart attack and 210,000 happen in people who have already had a heart attack.
- In 2010, approximately 1 in 6 people in the United States died of Acute Myocardial Infarction.
- Approximately every 34 seconds, 1 American has a coronary event, and approximately every 1 minute 23 seconds, an American will die of one.

The incidence of MI in India is 64.37/1000 people in men aged 29-69 years

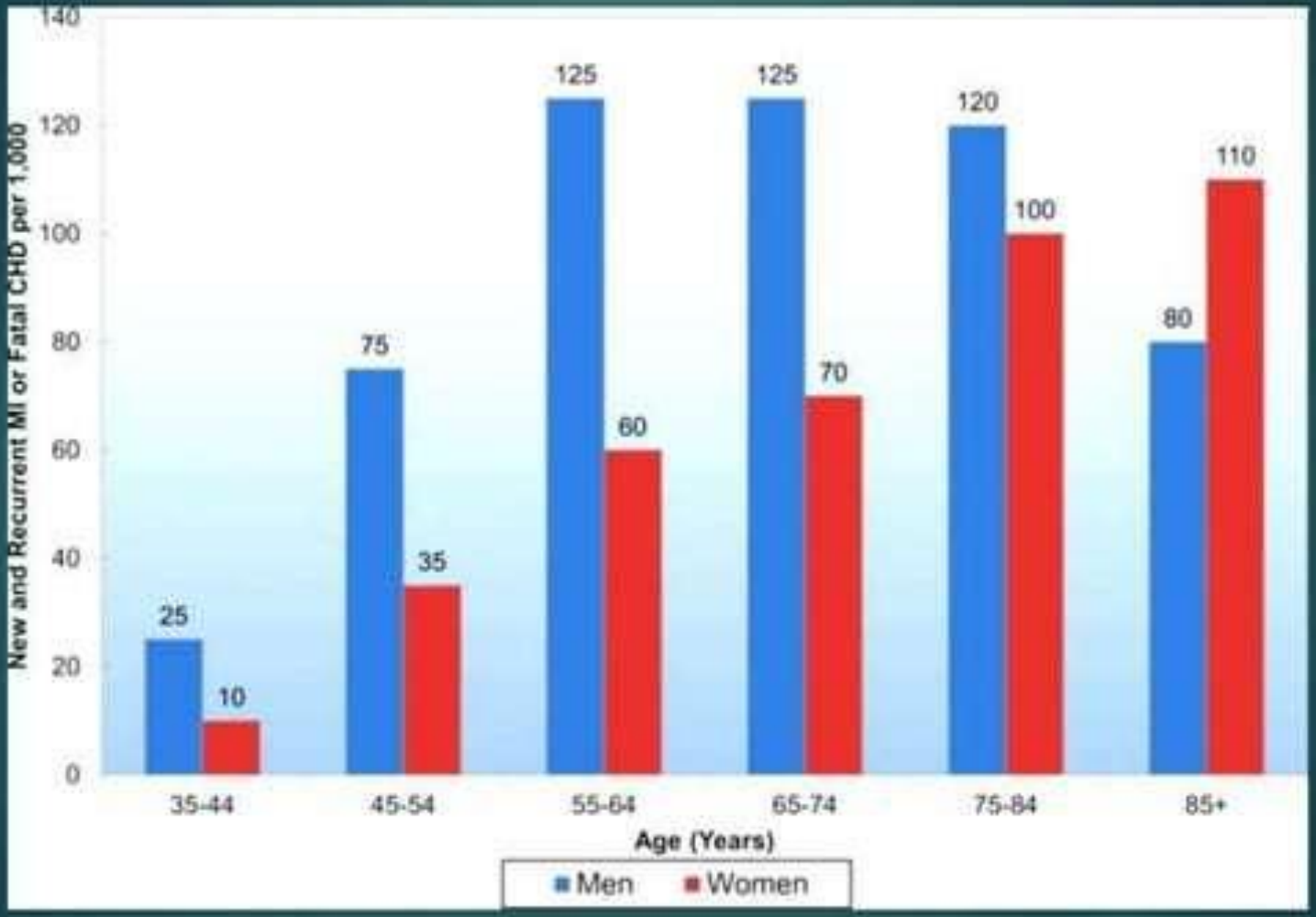
Epidemiology

Prevalence of myocardial infarction by age and sex



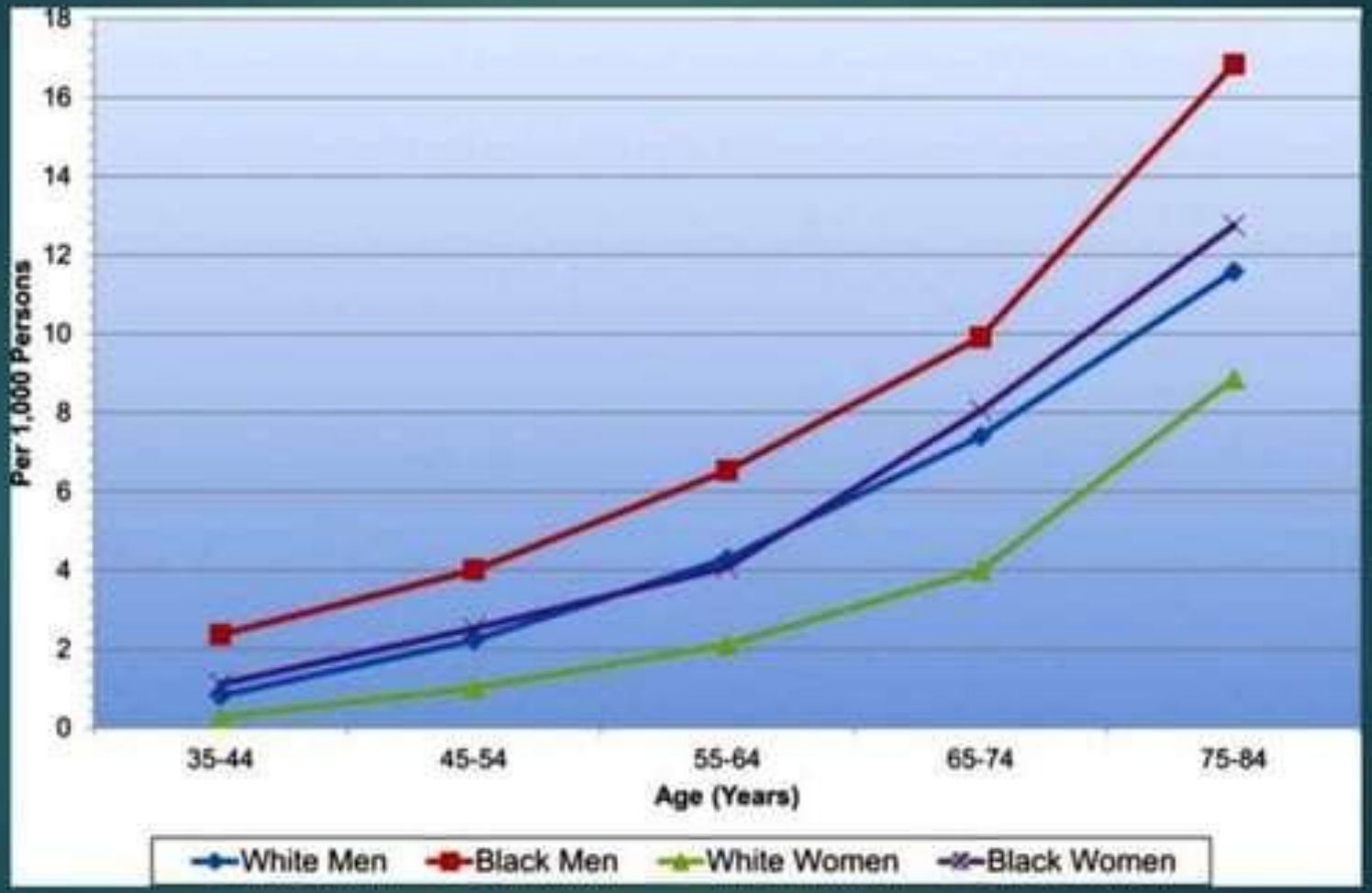
Epidemiology

Annual number of adults per 1000 having diagnosed heart attack or fatal coronary heart disease (CHD) by age and sex



Epidemiology

Incidence of heart attack or fatal coronary heart disease by age, sex, and race



Risk Factor

Lifestyle	Biochemical or physiological characteristics (modifiable)	Personal characteristics (nonmodifiable)
Diet high in saturated fat, cholesterol, and calories	Elevated blood pressure	Older age
Tobacco smoking	Elevated plasma total cholesterol (LDL cholesterol)	Male gender
Excess alcohol consumption	Low plasma HDL cholesterol	Family history of CHD or other atherosclerotic vascular disease at early age (men <55 years, women <65 years)
Physical inactivity	Elevated plasma triglycerides	Personal history of CHD or other atherosclerotic vascular disease
	Hyperglycemia/diabetes	
	Obesity	
	Thrombogenic factors	

Risk Factor

- *Increasing age and male sex*. Individuals aged older than 45 years have an eight times greater risk for AMI.
  Even after menopause, when women's death rate from heart disease increases, it's not as great as men's

- *Hypertension, dyslipidemia,and diabetes*

"A case-control study of AMI in 52 countries, comprising 15,152 cases and 14,820 controls, was conducted. Among the important risk factors for AMI in both men and women were were raised ApoB/ApoA1 ratio (OR = 3.25), history of hypertension (OR = 1.91), and diabetes (OR = 2.37)"

Risk Factor

- Smoking increases a person's risk for heart disease to about 4 times greater than non-smokers.

- Obesity and physical inactivity People who have excess body fat — especially at the waist — are more likely to develop heart disease even if they have no other risk factors.
- Lack of exercise has been linked to 7–12% of cases

- Acute and prolonged intake of high quantities of alcoholic drinks (3-4 or more) increase the risk of a heart attack

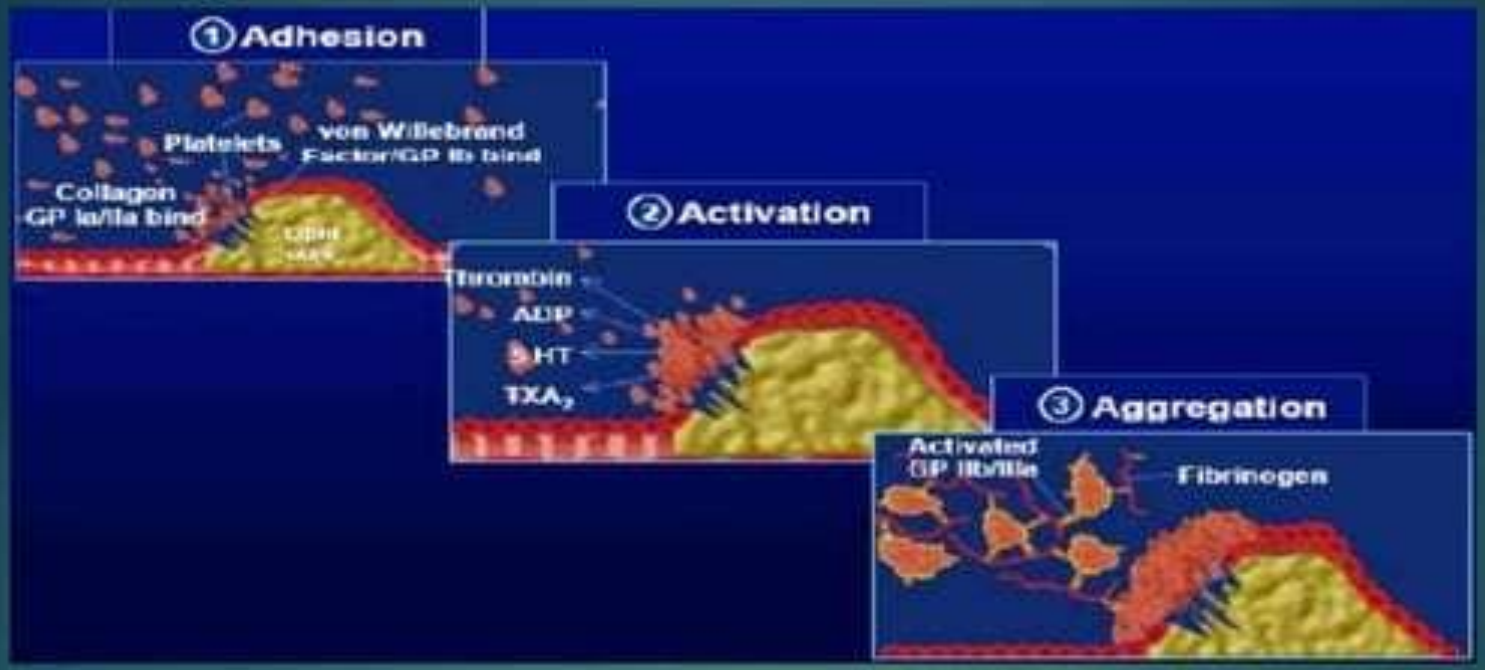
Pathophysiology

Coronary Arterial Occlusion.

- Rupture of high-risk atheromatous plaque in the coronary arteries is a primary causative factor in the development of AMI.

- When exposed to subendothelial collagen and necrotic plaque contents, platelets adhere, become activated, release their granule contents, and aggregate to form microthrombi.

Pathophysiology



- Vasospasm is stimulated by mediators released from platelets.
- Tissue factor activates the coagulation pathway, adding to the bulk of the thrombus.
- Within minutes, the thrombus expands to completely occlude the vessel lumen.

Pathophysiology

- In approximately 10% of cases, AMI occurs in the absence of the typical coronary atherothrombosis.

Vasospasm	Emboli	Others
• Intravascular Platelet aggregation • drug ingestion (e.g., cocaine or ephedrine)	• Vegetations of infective endocarditis, • Intracardiac prosthetic material	• Vasculitis, • Hematologic abnormalities (e.g., sickle cell disease), • Amyloid deposition, • Vascular dissection, • Aortic stenosis, • Lowered systemic blood pressure (e.g., shock)

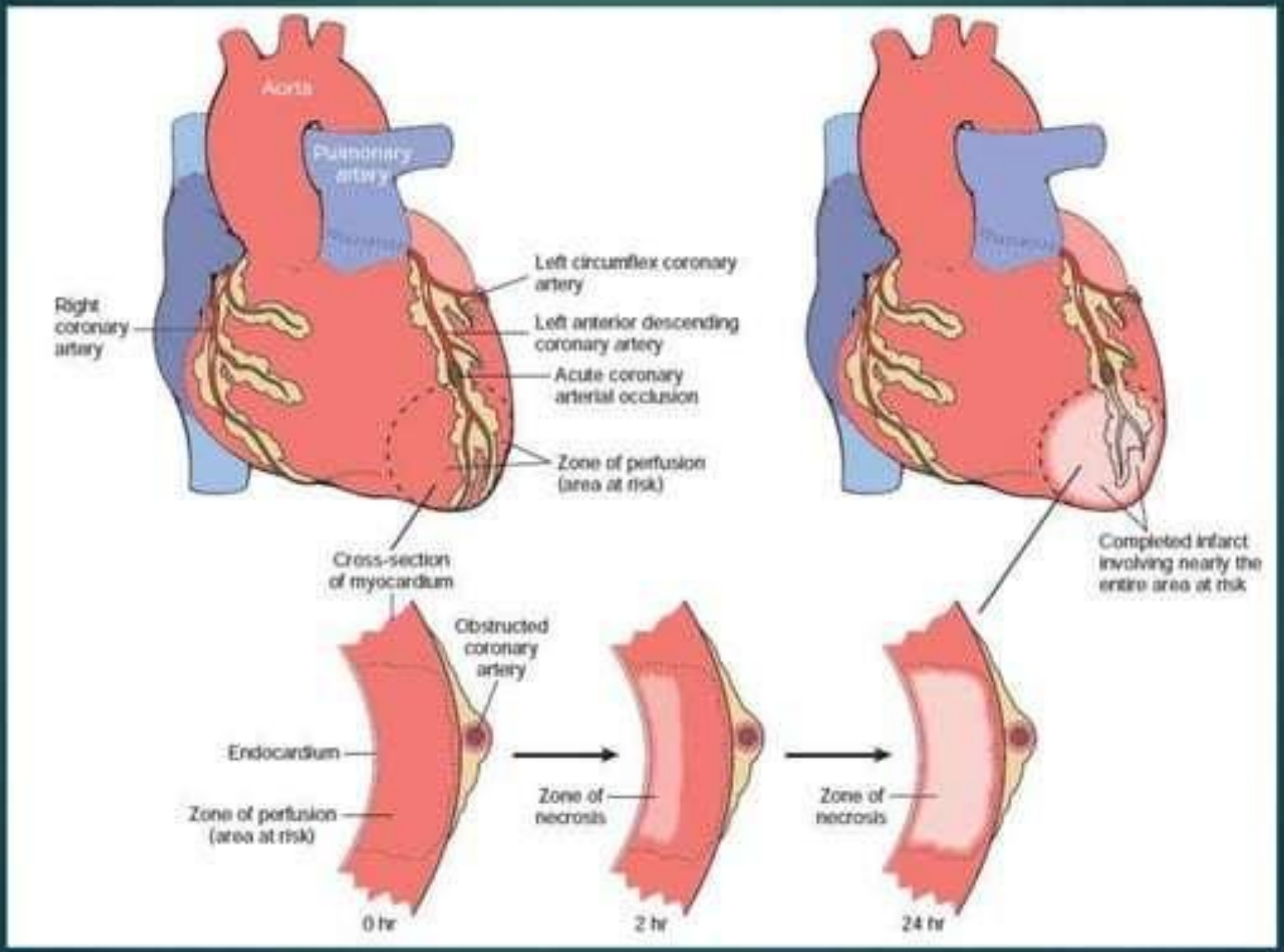
Pathophysiology

Myocardial Response.

- Coronary arterial obstruction diminishes blood flow to a region of myocardium causing ischemia, rapid myocardial dysfunction, and eventually—with prolonged vascular compromise — myocyte death.

- The anatomic region supplied by that artery is referred to as the *area at risk*.

Pathophysiology



Pathophysiology

- Experimental and clinical evidence shows that only severe ischemia lasting 20 to 30 minutes or longer leads to irreversible damage (necrosis) of cardiac myocytes.

- This delay in the onset of permanent myocardial injury provides the rationale for rapid diagnosis in acute MI—to permit early coronary intervention to establish reperfusion and salvage as much "at risk" myocardium as possible.

Pathophysiology

Feature	Time
Onset of ATP depletion	Seconds
Loss of contractility	<2 min
ATP reduced	
to 50% of normal	10 min
to 10% of normal	40 min
Irreversible cell injury	20-40 min
Microvascular injury	>1 hr

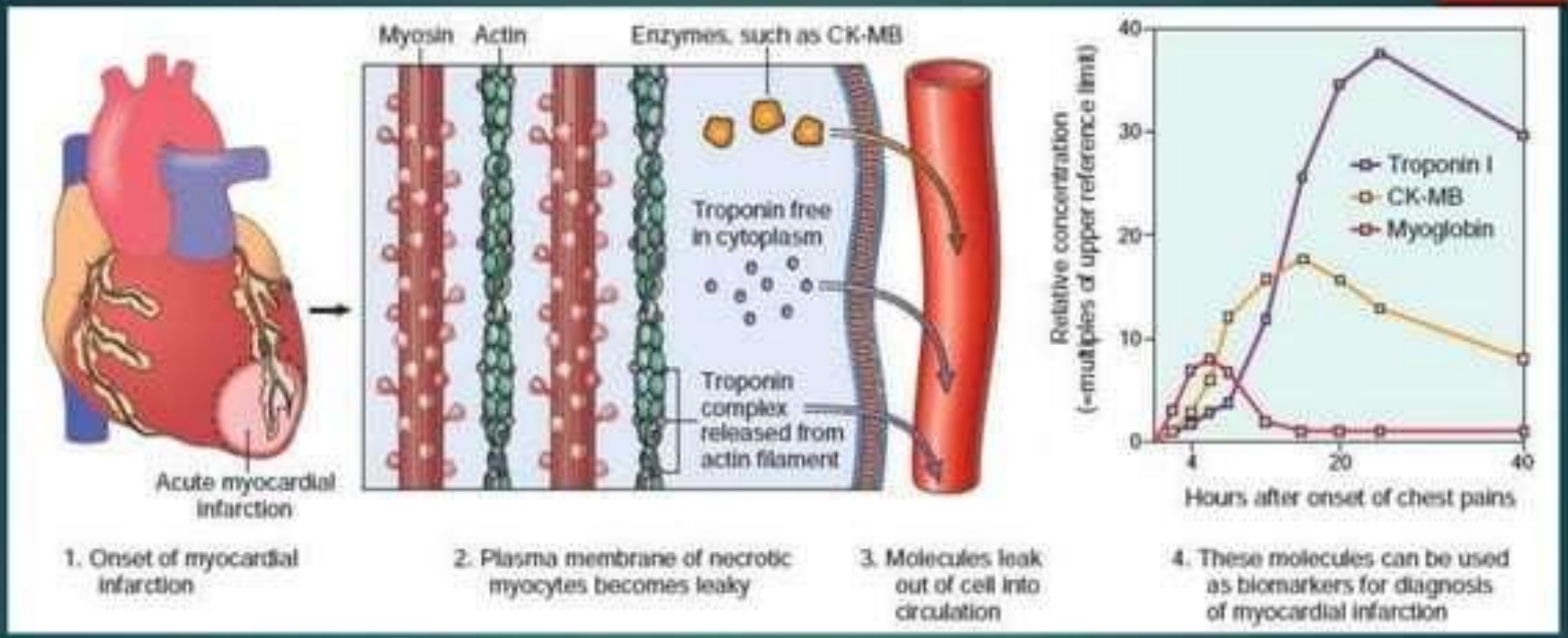
ATP, Adenosine triphosphate.

Pathophysiology

- The earliest detectable feature of myocyte necrosis is the disruption of the integrity of the sarcolemmal membrane, allowing intracellular macromolecules to leak out of necrotic cells into the cardiac interstitium and ultimately into the microvasculature and lymphatics.

- Intracellular myocardial proteins into the circulation forms the basis for blood tests that can sensitively detect irreversible myocyte damage, and are important for managing AMI.

Pathophysiology



- Time to elevation of CKMB, cTnT and cTnI is 3 to 12 hrs
- CK-MB and cTnI peak at 24 hours
- CK-MB returns to normal in 48-72 hrs, cTnI in 5-10 days, and cTnT in 5 to 14 days

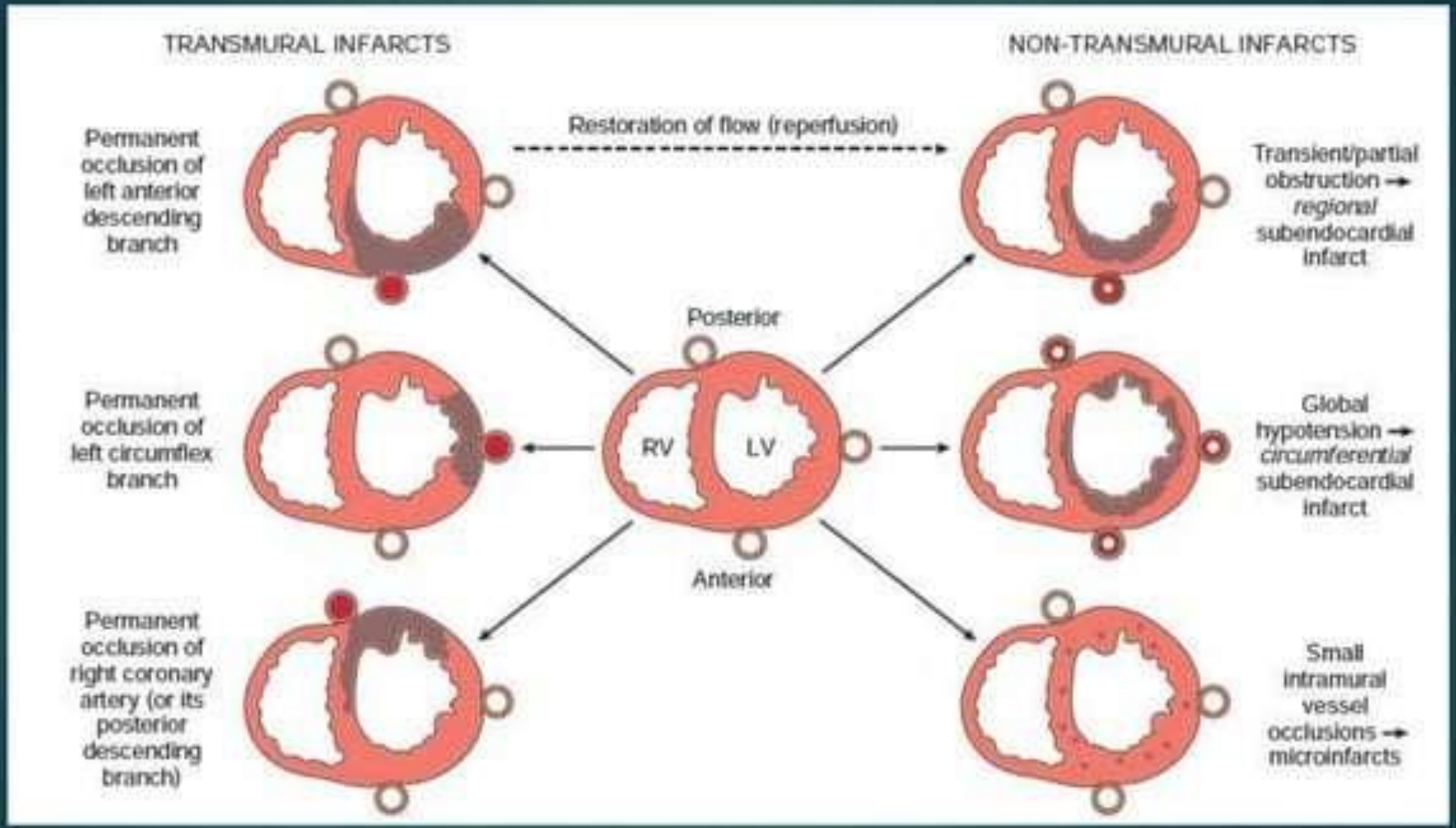
Classification

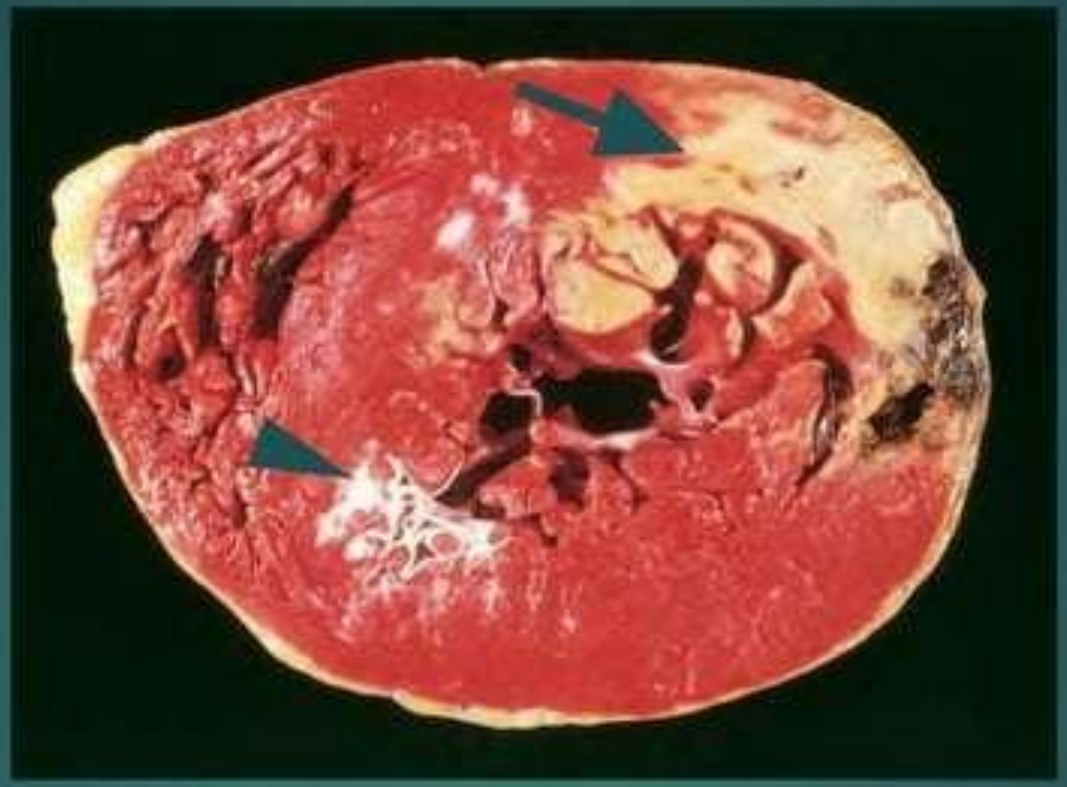
The two main types of acute myocardial infarction, based on pathology, are:

- *Transmural infarction*- Transmural infarcts extend through the whole thickness of the heart muscle and are usually a result of complete occlusion of the area's blood supply.
- *Subendocardial (nontransmural) infarction* - involves a small area in the subendocardial wall of the left ventricle, ventricular septum, or papillary muscles.

A transmural infarct is sometimes referred to as an "ST elevation myocardial infarct" (STEMI) and a subendocardial infarct as a "non–ST elevation infarct" (NSTEMI).

Classification





A transmural acute myocardial infarct, predominantly of the posterolateral left ventricle (arrow). Note the myocardial hemorrhage at one edge of the infarct that was associated with cardiac rupture, and the anterior scar *(arrowhead)*, indicative of old infarct.

Sign and symptoms

- Chest pain
  - most common symptom
  - described as a sensation of tightness, pressure, or squeezing.
  - not relieved by rest, position change or nitrate administration.
  - Pain radiates most often to the left arm, but may also radiate to the lower jaw, neck, right arm, back, and upper abdomen, where it may mimic heartburn.
  - Levine's sign, in which a person localizes the chest pain by clenching their fists over their sternum.

SILENT AMI - 20-30% subjects don't have chest pain, common in patients with diabetes mellitus, hypertension, & in elderly patients.

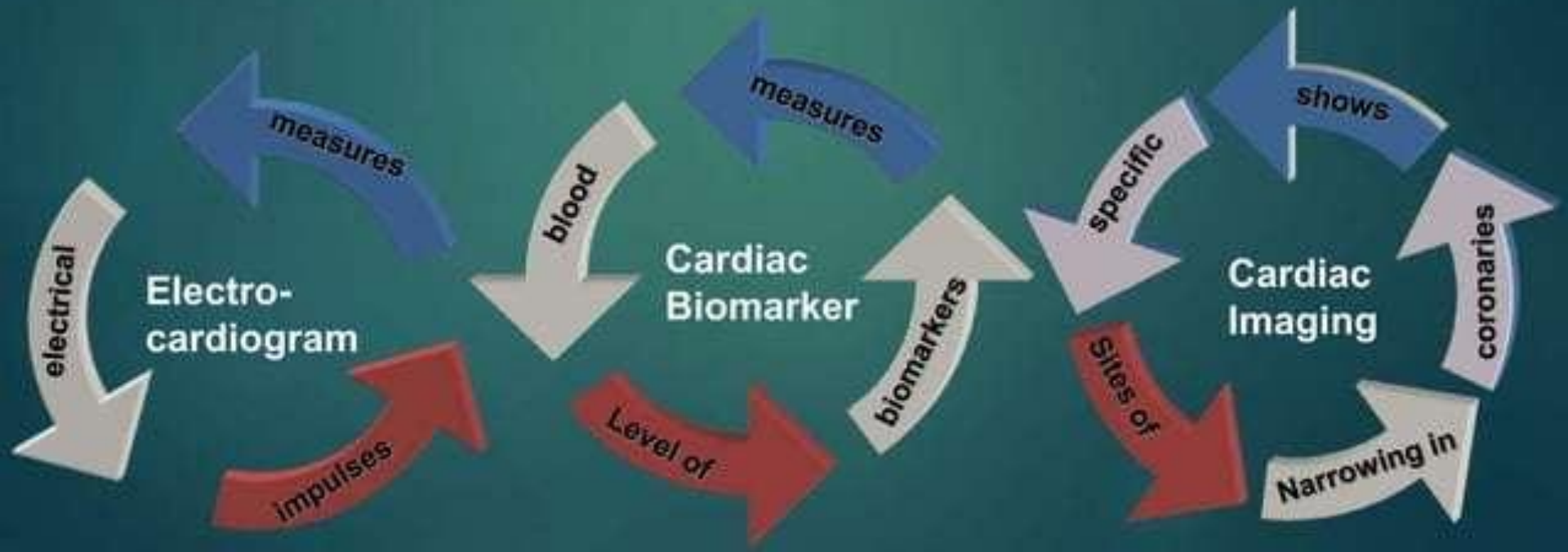
Sign and symptoms

- Nausea and Vomiting
  - Vomiting results as a reflex from severe pain.
  - Vasovagal reflexes initiated from area of ischemia.
- Shortness of breath (dyspnea)
  - the damage to the heart limits the output of the left ventricle, causing left ventricular failure and consequent pulmonary edema.
- Diaphoresis (an excessive form of sweating),
- Light-headedness, and
- Palpitations
- Loss of consciousness
  - inadequate blood flow to the brain and cardiogenic shock.
- Sudden death
  - due to the development of ventricular fibrillation

(massive surge of catecholamines from the sympathetic nervous system)

Diagnosis

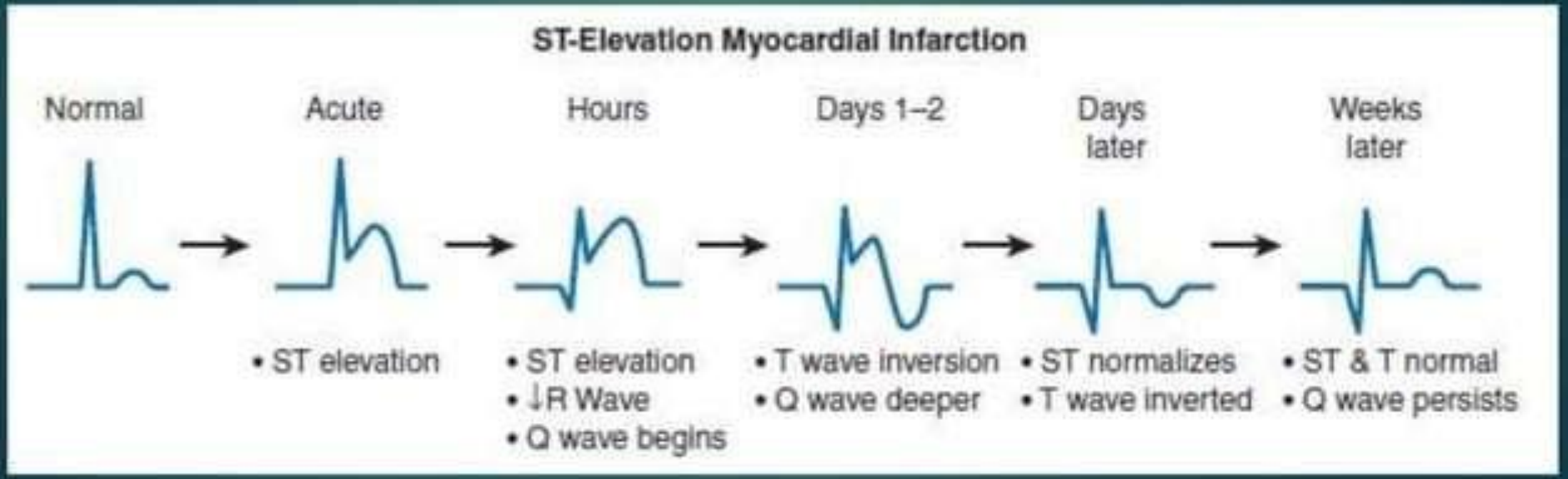
According to the WHO criteria as revised in 2000, a cardiac troponin rise accompanied by either typical symptoms, pathological Q waves, ST elevation or depression or coronary intervention are diagnostic of MI.



Diagnosis

ECG changes

- ST segment elevation, followed by T wave inversion and Q waves, are associated with transmural infarction.
- ST segment depression and T wave inversion are associated with subendocardial infarction.



Diagnosis

- **Serum Cardiac Biomarkers**

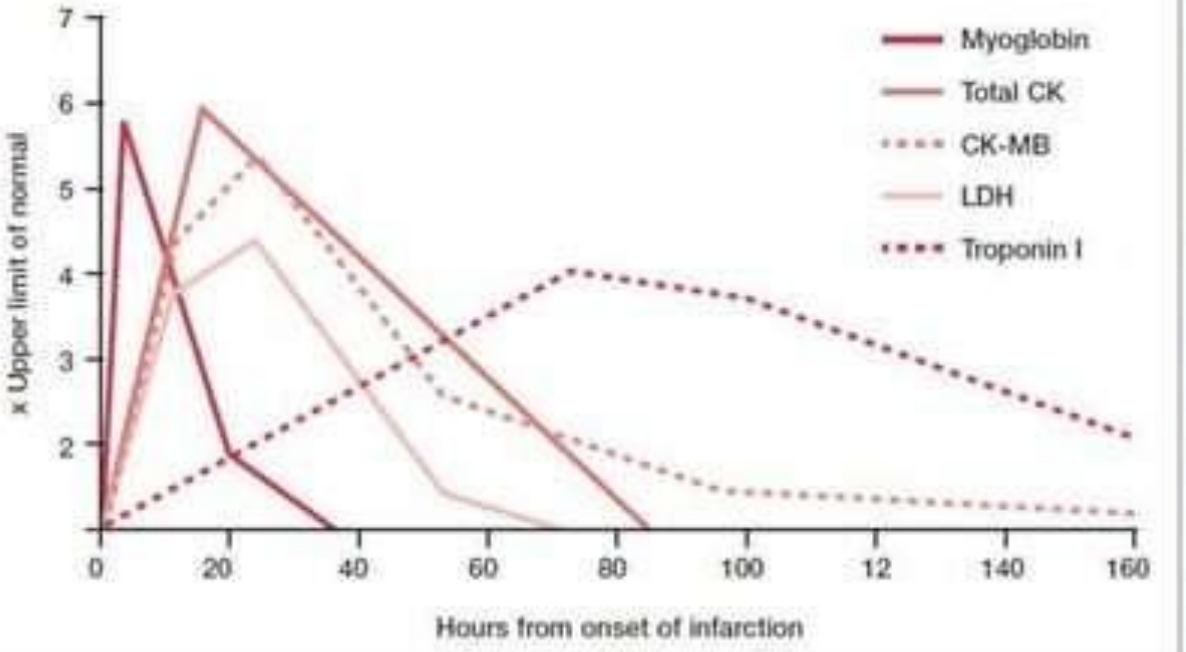


Figure 10. Appearance of cardiac markers in the blood after onset of symptoms. CK: creatine kinase; CK-MB: creatine kinase myocardial band fraction; LDH: lactate dehydrogenase.

The combination of CPK MB and troponin testing have higher sensitivity and is used for the purpose of "ruling out" myocardial infarction.

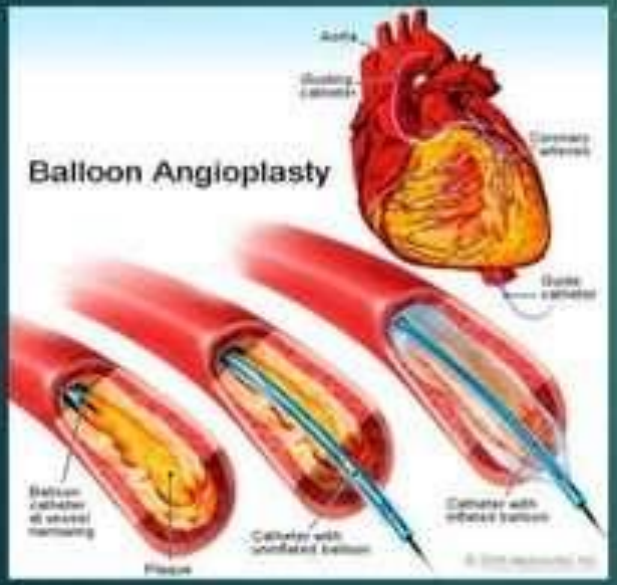
Management overview

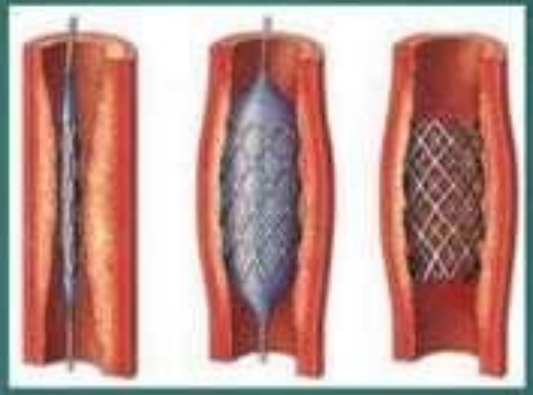
- MONA- B
  - Morphine
  - Oxygen
  - Nitroglycerin
  - Aspirin / Clopidogrel
  - Beta-Blockers

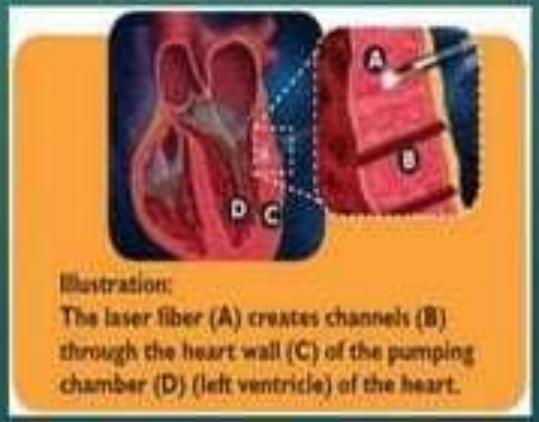
- Other Early Hospital Therapies
  - ACE Inhibitors
  - Non- Dihydropyridine Calcium Channel Blockers.
  - Fibrinolytics
  - ~Targeted temperature management

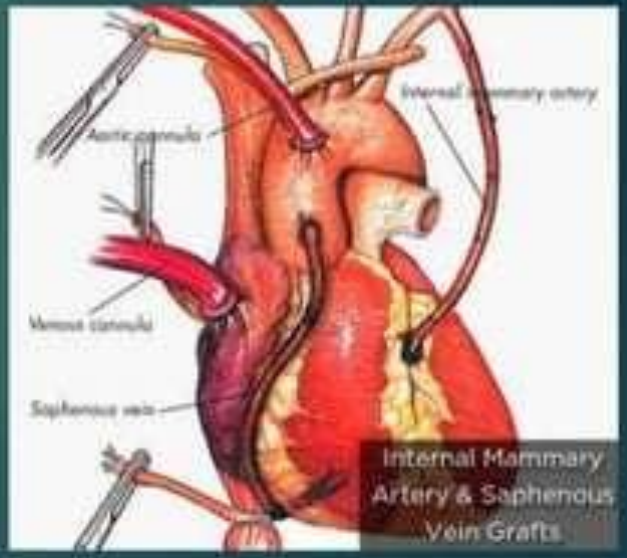
Management overview

- **Revascularization procedures**
  - Percutneous Coronary Intervention / Angioplasty.
  - Coronary Artery By –Pass (CABG).
  - Transmyocardial Laser Revascularization









Preventive Measures

Lifestyle modifications:

- Smoking cessation: Two years after cessation, the risk of AMI drops by 50%
- Alcohol moderation and prevention of illicit drug use.
- Physical activity and exercise:
  - Exercise 30 minutes per day 7 days a week.
  - Physical activity can help control blood cholesterol, diabetes and obesity, as well as help lower blood pressure.
  - losing even 10% from current weight, can lower your heart disease risk.

Preventive Measures

Diet modification

- Diets rich in soluble fiber, vegetables, fruits, and whole grains, and low in saturated fat/trans fat and cholesterol should be encouraged.
- Lipid management:
  - Saturated fat (<7% of total calories),
  - cholesterol and trans fatty acids (<200 mg/day),
  - plant stanols/sterols (2 g/day),
  - viscous fiber (10 g/day),
  - Olive oil, rapeseed oil and related products are to be used instead of saturated fat
  - use of omega-3 fatty acids (fish)

Preventive Measures

Management and control of comorbid diseases

- Hypertension should be managed.
  - Patients with CAD should have their blood pressure maintained at less than 130/80 mm Hg.
  - This may be achieved using a multimodal approach, which includes diet modification, lifestyle changes, exercise, and medications.
- Diabetes control should be appropriate
  - According to the 2007 AHA guideline for management of patients with STEMI, the goal for HbA1c in diabetic patients should be less than 7%

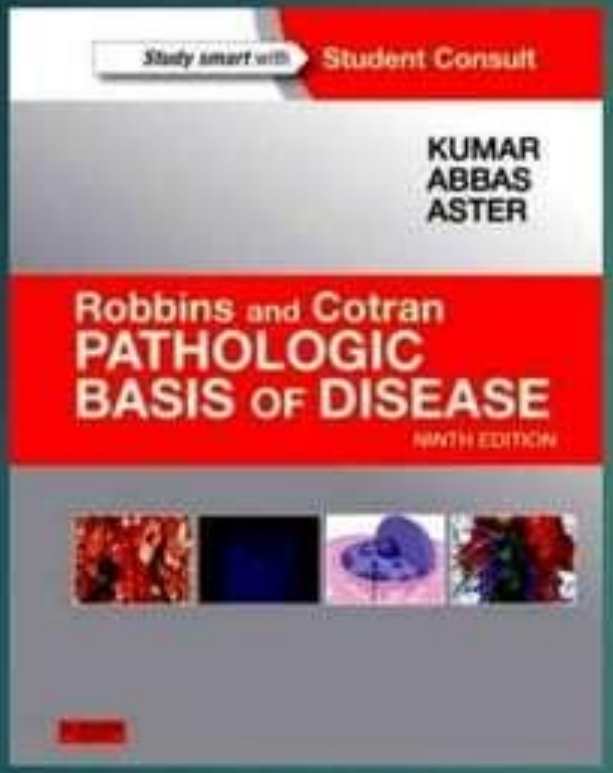
Patient education:

- Patients, their family members, and the community should be educated properly, especially on how to detect and respond to an episode of AMI

Chance of Not Having a Heart Attack If You…..

- Exercise
- Eat Healthy
- Don't Smoke
- Don't Drink

References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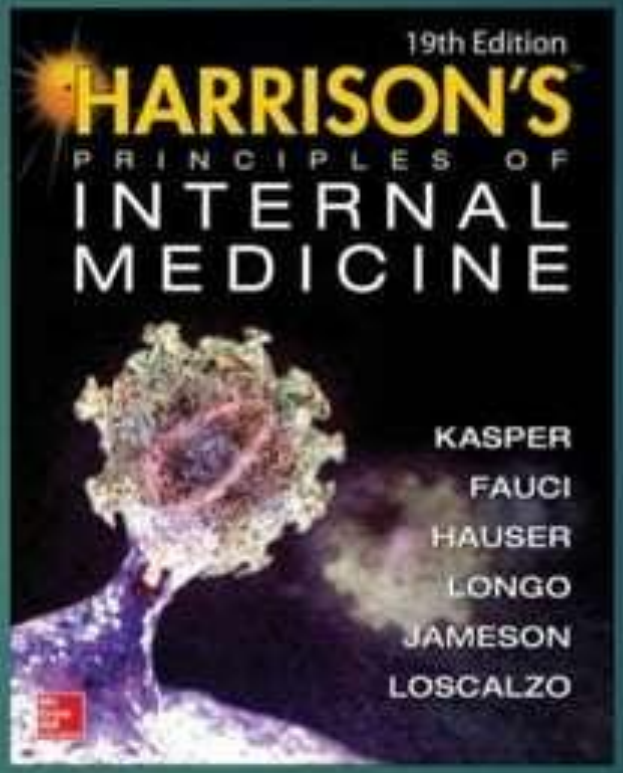
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