MYOCARDIAL INFARCTION

- Myocardial infarction is an irreversible injury to a part of the heart or myocardial tissue that results from ischemia and hypoxia finally necrosis of particular cells.
- Usually this is because one of the coronary arteries that supplies blood to the heart develops a blockage due to an unstable buildup of atherosclerotic plaques and other blood cells.
- Myocardial infarction is one of the leading killer in the United States.
- This is called Acute MI if it is sudden and serious.
- 64% of cases does not have chest pain or other symptoms.
- This is called Silent myocardial infarction.

ETIOPATHOGENESIS

The etiologic role of severe coronary atherosclerosis (more than 75% compromise of lumen) of one or more of the three major coronary arterial trunks in the pathogenesis of about 90% cases of acute MI is well documented by autopsy studies as well as by coronary angiographic studies.

1. Myocardial ischaemia

Myocardial ischaemia is brought about by one or more of the following mechanisms:

i) Diminished coronary blood flow e.g. in coronary artery disease, shock.

ii) Increased myocardial demand e.g. in exercise, emotions.

iii) Hypertrophy of the heart without simultaneous increase of coronary blood flow e.g. in hypertension, valvular heart disease.

2. Role of platelets

Rupture of an atherosclerotic plaque exposes the subendothelial collagen to platelets which undergo aggregation, activation and release reaction.

3. Acute plaque rupture

But acute complications in coronary atherosclerotic plaques in the form of superimposed coronary thrombosis due to plaque rupture and plaque haemorrhage is frequently encountered in cases of acute MI:

i) Superimposed coronary thrombosis due to disruption of plaque is seen in about half the cases of acute MI. Infusion of intracoronary fibrinolysins in the first half an hour of development of acute MI in such cases restores blood flow in the blocked vessel in majority of cases.

ii) Intramural haemorrhage is found in about one-third cases of acute MI.

4. **Non-atherosclerotic** causes About 10% cases of acute MI are caused by non-atherosclerotic factors such as coronary vasospasm, arteritis, coronary ostial stenosis, embolism, thrombotic diseases, trauma and outside compression as already described.
5. **Transmural versus subendocardial infarcts** There are some differences in the pathogenesis of the transmural infarcts involving the full thickness of ventricular wall and the subendocardial (laminar) infarcts affecting the inner subendocardial one-third to half. These are as under:

i) Transmural (full thickness) infarcts are the most common type seen in 95% cases. Critical coronary narrowing (more than 75% compromised lumen) is of great significance in the causation of such infarcts.

- ii) Subendocardial (laminar) infarcts have their genesis in reduced coronary perfusion due to coronary atherosclerosis but without critical stenosis (not necessarily 75% compromised lumen), aortic stenosis or haemorrhagic shock.

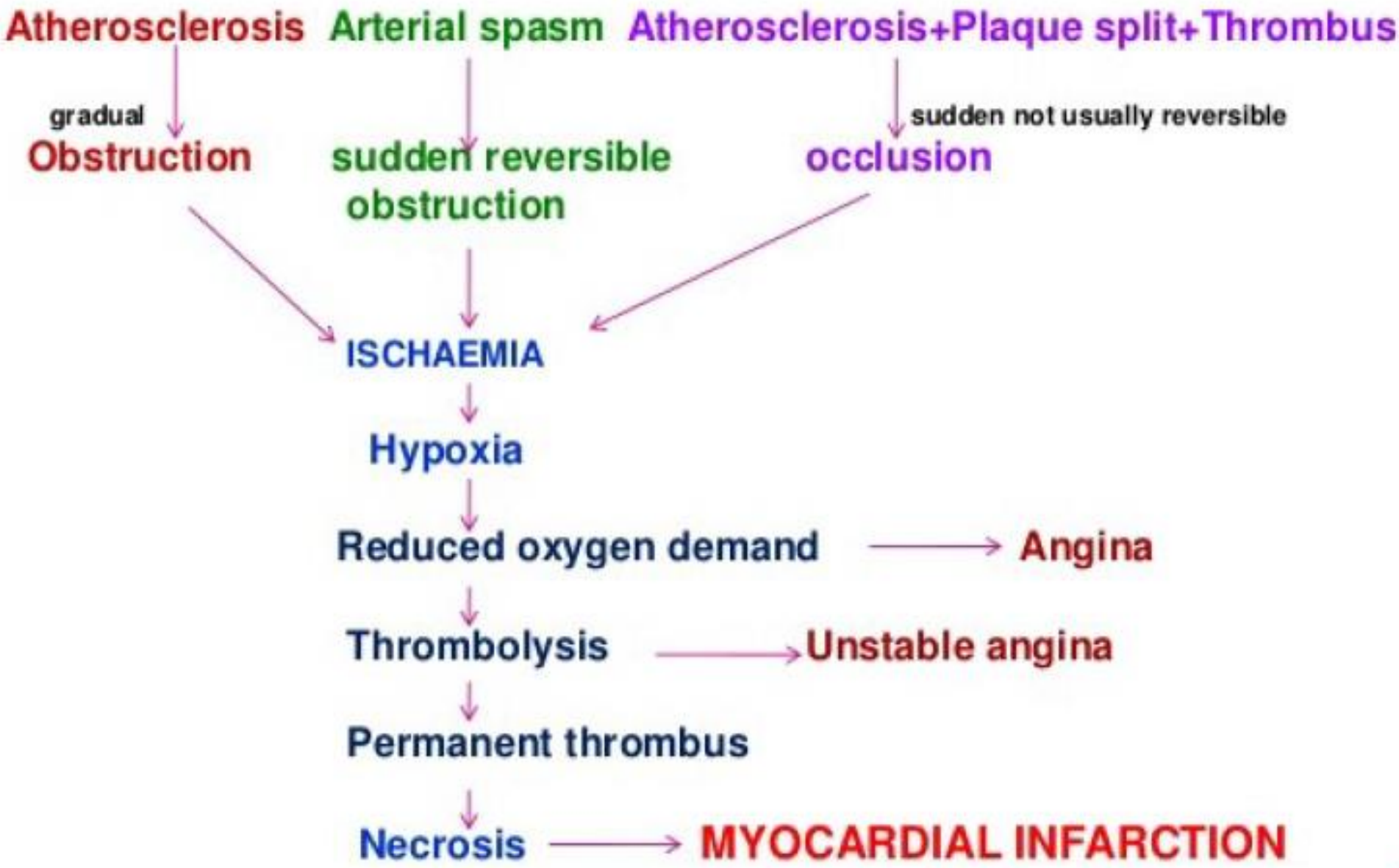
Risk Factors

- The presence of any risk factor is associated with doubling the risk of an MI.
- **Non Modifiable**
- Age
- Gender
- Family history
- **Modifiable**
- Smoking
- Diabetes Control
- Hypertension
- Hyperlipidemia
- Obesity
- Physical Inactivity

SYMPTOMS

- Sudden chest pain that is felt behind the sternum and sometimes travels to the left arm or the left side of the neck.
- Shortness of breath,
- Sweating,
- Nausea,
- Vomiting,
- Abnormal heartbeats,
- Anxiety
- Weakness,
- Feeling of indigestion,
- Fatigue.

PATHOPHYSIOLOGY



Pathophysiology

- Ischemia develops when there is an increased demand for oxygen or a decreased supply of oxygen.
- Ischemia can develop within 10 seconds and if it lasts longer than 20 minutes, irreversible cell and tissue death occurs.
- Myocardial cell death begins at the endocardium. The area most distal to the arterial blood supply.
- As vessel occlusion continues cell death spreads to the myocardium and eventually to the epicardium.
- Severity of the MI depends on three factors.
- Level of occlusion
- Length of time of occlusion
- Presence or absence of collateral circulation

Chest Pain

- The most common initial manifestation is chest pain or discomfort.
- This is not relieved by rest, position change or nitrate administration.
- Pain is described by heaviness, pressure, fullness and crushing sensation.

CHANGES IN EARLY INFARCTS

These are as under:

1. Electron microscopic changes

i) Disappearance of perinuclear glycogen granules within 5 minutes of ischaemia.

ii) Swelling of mitochondria in 20 to 30 minutes.

iii) Disruption of sarcolemma.

iv) Nuclear alterations like peripheral clumping of nuclear chromatin.

2. Chemical and histochemical changes

i) Glycogen depletion in myocardial fibres within 30 to 60 minutes of infarction.

ii) Increase in lactic acid in the myocardial fibres.

iii) Loss of K+ from the ischaemic fibres.

iv) Increase of Na+ in the ischaemic cells.

v) Influx of Ca++ into the cells causing irreversible cell injury.

TYPES OF INFARCTS

Infarcts have been classified in a number of ways by the physicians and the pathologists:

1. According to the anatomic region of the left ventricle involved, they are called anterior, posterior (inferior), lateral, septal and circumferential, and their combinations like anterolateral, posterolateral (or inferolateral) and anteroseptal.

2. According to the degree of thickness of the ventricular wall involved, infarcts are of two types:

i) Full-thickness or transmural, when they involve the entire thickness of the ventricular wall.

ii) Subendocardial or laminar, when they occupy the inner subendocardial half of the myocardium.

3. According to the age of infarcts, they are of two types:

i) Newly-formed infarcts called as acute, recent or fresh.

ii) Advanced infarcts called as old, healed or organised.

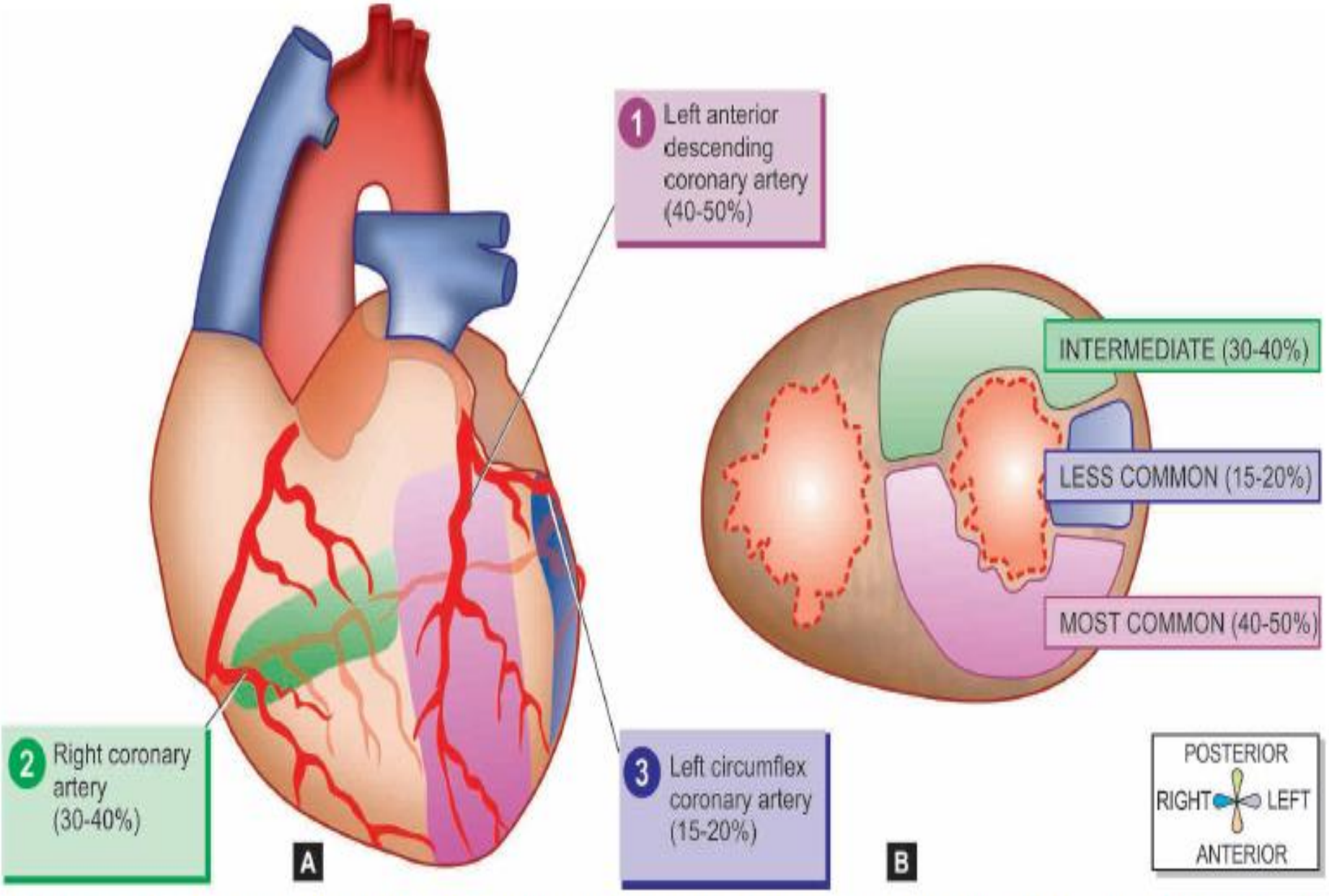


Figure 14.16 Common locations and the regions of involvement in myocardial infarction. The figure shows region of myocardium affected by stenosis of three respective coronary trunks in descending order shown as: 1) left anterior descending coronary, 2) right coronary and 3) left circumflex coronary artery. A, As viewed from anterior surface. B, As viewed on transverse section at the apex of the heart.

Table 14.4 Sequential pathologic changes in myocardial infarction.

TIME	GROSS CHANGES	LIGHT MICROSCOPY
FIRST WEEK		
0-6 hours	No change or pale; TTC/ NBT test negative in infarcted area	No change; (?) stretching and waviness of fibres
6-12 hours	-do-	Coagulative necrosis begins; neutrophilic infiltration begins; oedema and haemorrhages present
24 hours	Cyanotic red-purple area of haemorrhage	Coagulative necrosis progresses; marginal neutrophilic infiltrate
48-72 hours	Pale, hyperaemic	Coagulative necrosis complete, neutrophilic infiltrate well developed
3rd -7th day	Hyperaemic border, centre yellow and soft	Neutrophils are necrosed and gradually disappear, beginning of resorption of necrosed fibres by macrophages, onset of fibrovascular response
SECOND WEEK		
10th day	Red-purple periphery	Most of the necrosed muscle in a small infarct removed; fibrovascular reaction more prominent; pigmented macrophages, eosinophils, lymphocytes, plasma cells present
14th day	—	Necrosed muscle mostly removed; neutrophils disappear; fibrocollagenic tissue at the periphery
THIRD WEEK	—	Necrosed muscle fibres from larger infarcts removed; more ingrowth of fibrocollagenic tissue
FOURTH TO SIXTH WEEK	Thin, grey-white, hard, shrunken fibrous scar	Increased fibrocollagenic tissue, decreased vascularity; fewer pigmented macrophages, lymphocytes and plasma cells

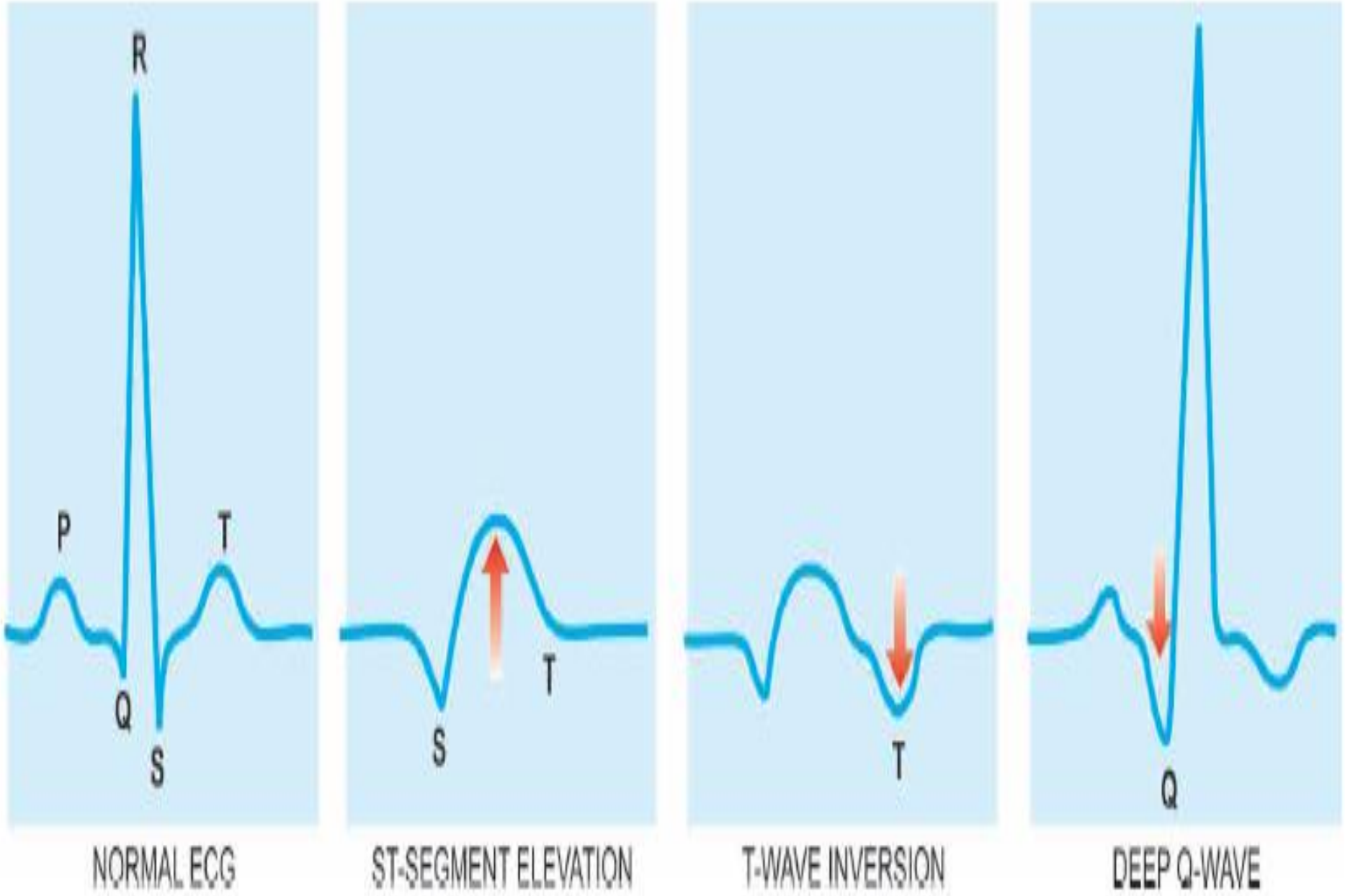


Figure 14.20 Some common ECG changes in acute myocardial infarction.

COMPLICATIONS

Following an attack of acute MI, only 10-20% cases do not develop major complications and recover.

1. Arrhythmias
2. Congestive heart failure
3. Cardiogenic shock
4. Mural thrombosis and thromboembolism
5. Rupture
6. Cardiac aneurysm
7. Pericarditis
8. Postmyocardial infarction syndrome

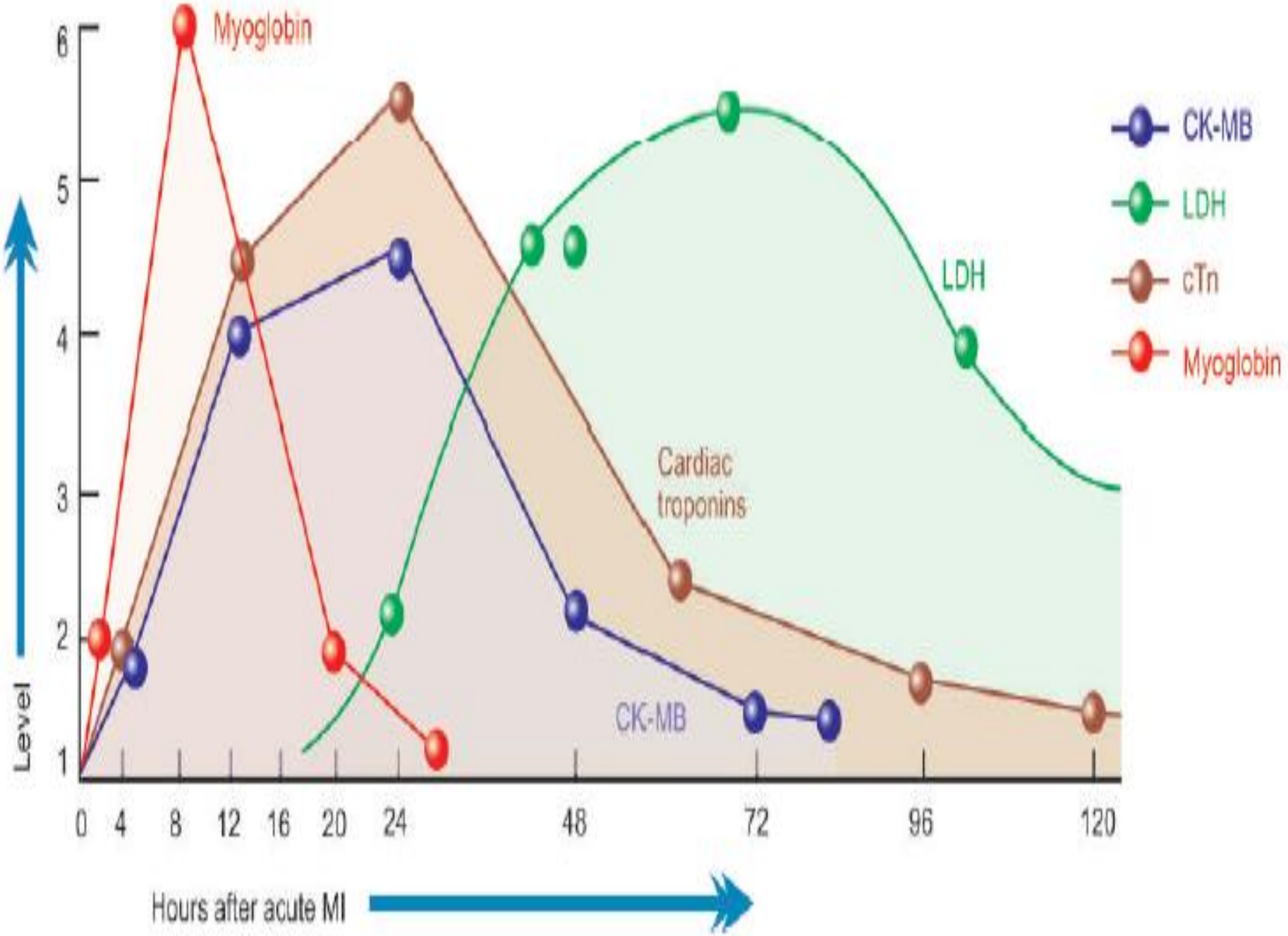


Figure 14.21 Time course of serum cardiac markers for the diagnosis of acute MI.

DIAGNOSIS

The diagnosis of acute MI is made on the observations of 3 types of features:

1. Clinical features Typically, acute MI has a sudden onset.

i) Pain

ii) Indigestion

iii) Apprehension

iv) Shock

v) Oliguria

vi) Low grade fever

vii) Acute pulmonary oedema.

2. ECG changes

The ECG changes are one of the most important parameters. Most characteristic ECG change is ST segment elevation in acute MI (termed as STEMI); other changes inlcude T wave inversion and appearance of wide deep Q waves.

3. Serum cardiac markers

Certain proteins and enzymes are released into the blood from necrotic heart muscle after acute MI. Measurement of their levels in serum is helpful in making a diagnosis and plan management.

i) *Creatine phosphokinase (CK) and CK-MB CK has three forms—*

a) CK-MM derived from skeletal muscle;

b) CK-BB derived from brain and lungs; and

c) CK-MB, mainly from cardiac muscles and insignificant amount from extracardiac tissue.

ii) Lactate dehydrogenase (LDH) Total LDH estimation also lacks specificity since this enzyme is present in various tissues besides myocardium such as in skeletal muscle, kidneys, liver, lungs and red blood cells.

- iii) Cardiac-specific troponins (cTn) Troponins are contractile muscle proteins present in human cardiac and skeletal muscle but cardiac troponins are specific for myocardium. There are two types of cTn:
- a) cardiac troponin T (cTnT); and
- b) cardiac troponin I (cTnI).

- iv) Myoglobin Though myoglobin is the first cardiac marker to become elevated after myocardial infarction, it lacks cardiac specificity and is excreted in the urine rapidly. Its levels, thus, return to normal within 24 hours of attack of acute MI.

TREATMENT

- Aspirin - which prevents further blood from clotting, chest pain
- Nitroglycerin – Vasodilator.
- STEMI is treated by reperfusion therapy, angioplasty (arteries are pushed open) Thrombolytics.

THANK YOU