

MYOCARDIAL INFARCTION

Learning objectives

- 1-Pathophysiology
- 2-Size and location
- 3-Clinical assessment
- 4-Diagnostic approach and laboratory tests
- 5-Management
- 6-Complications

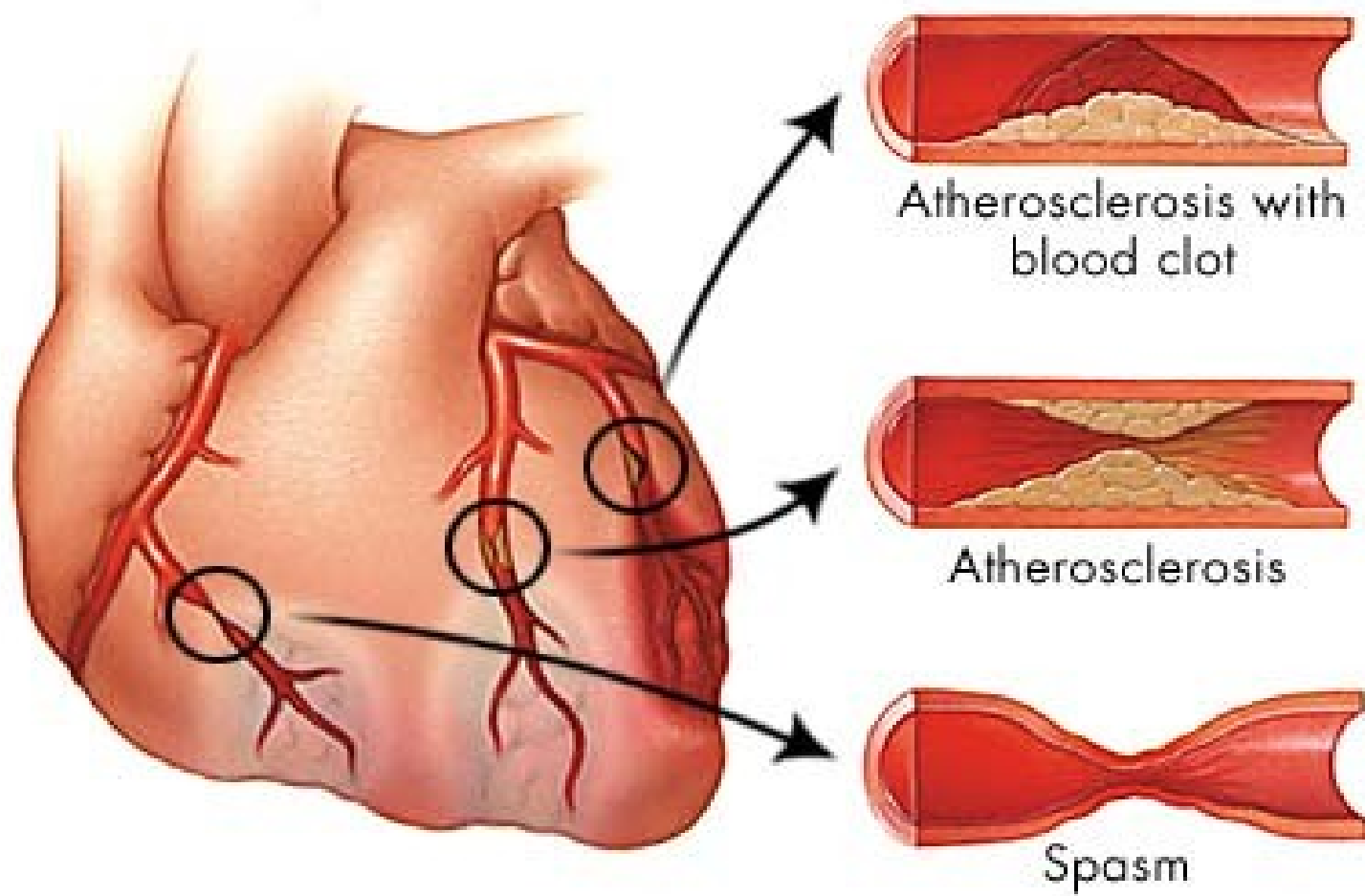
- ▶ **Myocardial infarction (MI) or acute myocardial infarction (AMI)**, commonly known as a **heart attack**, is the interruption of **blood supply** to part of the **heart**, causing some heart cells to die.
- ▶ Either one of the following criteria satisfies the diagnosis for an acute, evolving, or recent myocardial infarction:
 - 1. Typical rise and gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following:**
 - a. Ischemic symptoms**
 - b. Development of pathologic Q waves on the electrocardiogram**
 - c. Electrocardiographic changes indicative of ischemia (ST segment elevation or depression)**
 - d. Coronary artery intervention (e.g., coronary angioplasty).**
 - 2. Pathological findings of an acute myocardial infarction**

Pathophysiological Principles

- ▶ Most patients who sustain an MI have coronary atherosclerosis.
- ▶ The thrombus formation occurs most often at the site of an atherosclerotic lesion, thus obstructing blood flow to the myocardial tissues.
- ▶ Plaque rupture is believed to be the triggering mechanism for the development of the thrombus in most patients with an MI.
- ▶ When the plaques rupture, a thrombus is formed at the site that can occlude blood flow, thus resulting in an MI.

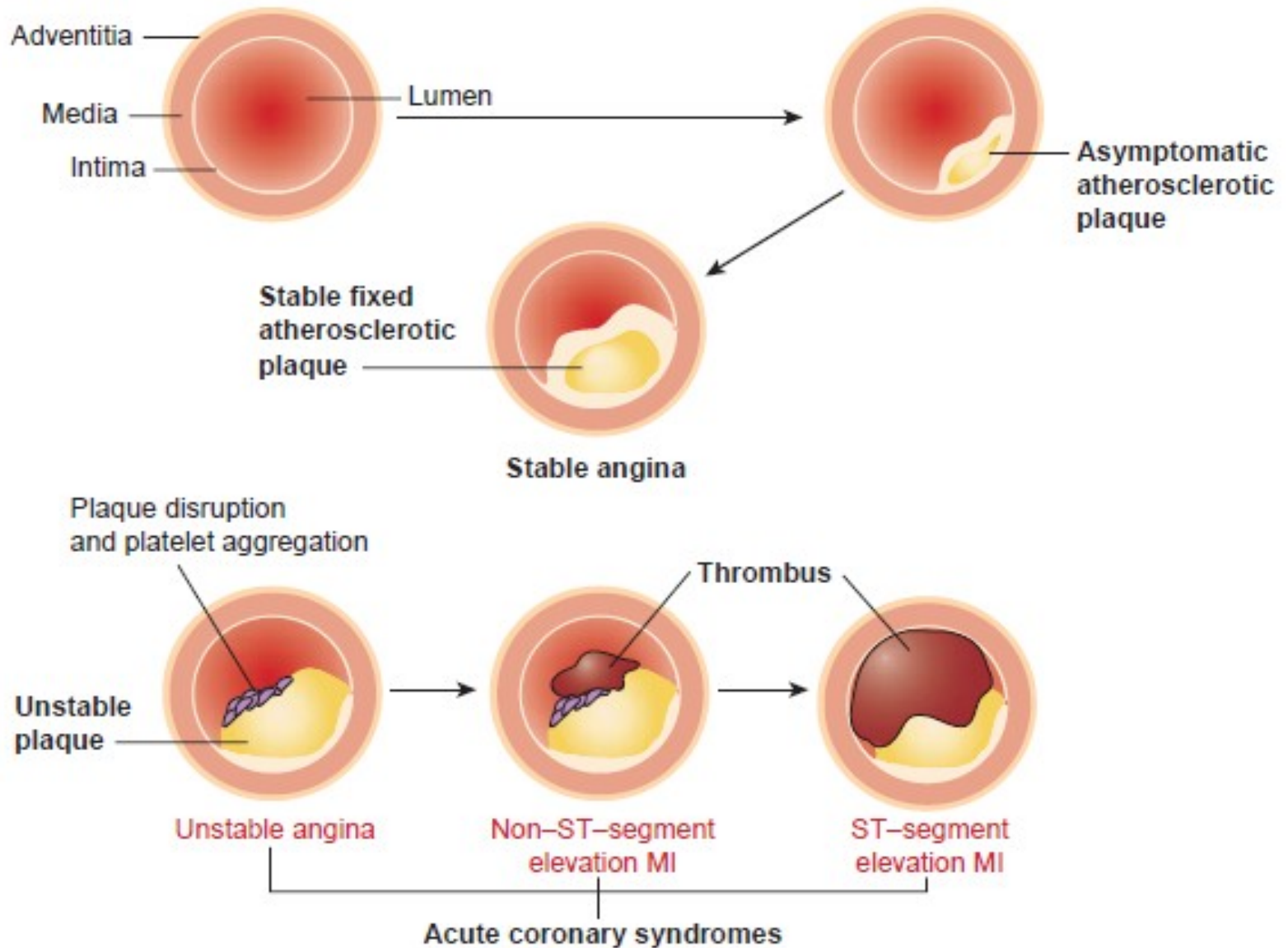
- Irreversible damage to the myocardium can begin as early as 20 to 40 minutes after interruption of blood flow.
- The dynamic process of infarction may not be completed, however, for several hours.
- Necrosis of tissue appears to occur in a sequential fashion.
- cellular death occurs first in the subendocardial layer and spreads like a “wavefront” throughout the thickness of the wall of the heart.

- a substantial amount of myocardial tissue can be salvaged if flow is restored within 6 hours after the onset of coronary occlusion.
- The cellular changes associated with an MI can be followed by:
 1. the development of infarct extension (new myocardial necrosis),
 2. Ventricular remodeling (a disproportionate thinning and dilation of the ventricle).



- " **Ischaemia** " refers to an insufficient amount of blood. The coronary arteries are the only source of blood for the heart muscle. If this coronary arteries are blocked, the blood supply will reduce.





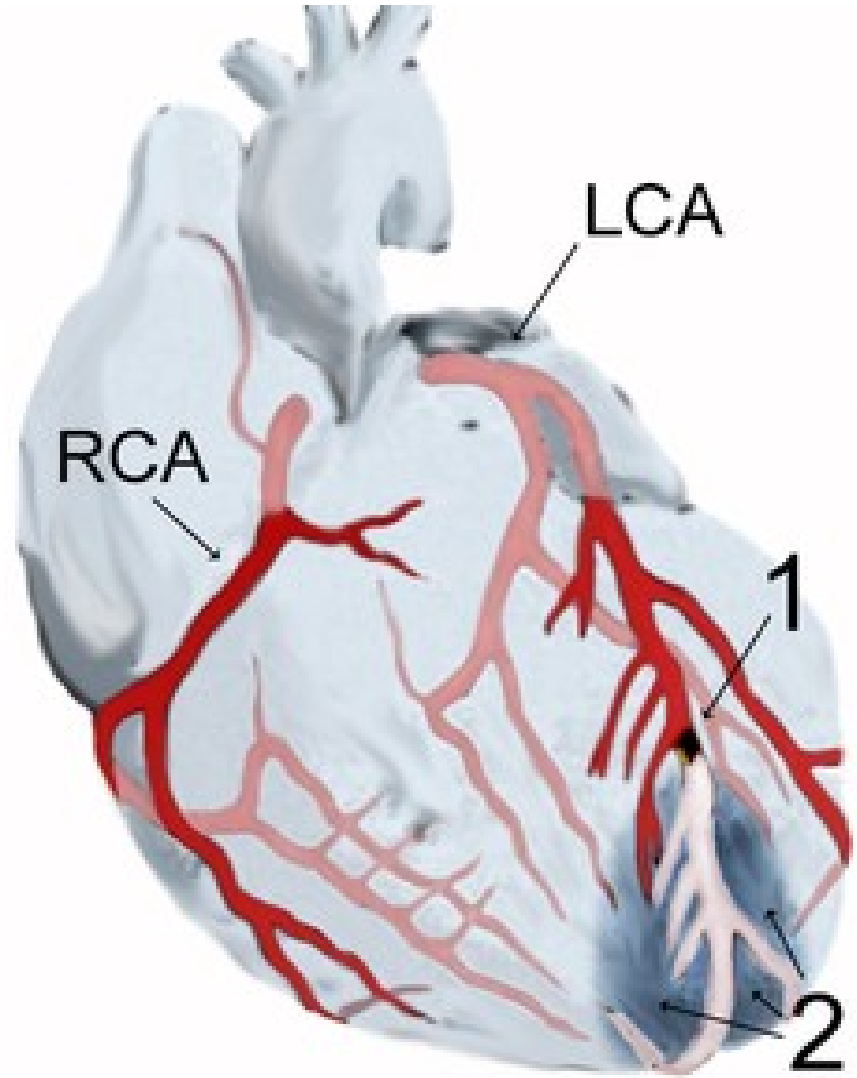
SIZE OF THE INFARCTION

- ▶ Several factors determine the size of the resulting MI.
- ▶ These factors include the extent, severity, and duration of the ischemic episode; the size of the vessel; the amount of collateral circulation; the status of the intrinsic fibrinolytic system; vascular tone; and the metabolic demands of the myocardium at the time of the event.
- ▶ MIs most often result in damage to the left ventricle, leading to an alteration in left ventricular function.

- ▶ The term *transmural infarction* is used to imply an infarction process that has resulted in necrosis of the tissue in all the layers of the myocardium.
- ▶ Because the heart functions as a squeezing pump, systolic and diastolic efforts can be significantly altered when a segment of the heart muscle is necrotic and nonfunctional.
- ▶ If the area of the transmural infarction is small, the necrotic wall may be **dyskinetic**, a term meaning “difficulty in moving.”
- ▶ If the damage to the myocardial tissue is more extensive, the myocardial muscle may become **akinetic**, meaning “without motion.”

LOCATION OF THE INFARCTION

- MIs can be located in the anterior, septal, lateral, posterior, or inferior walls of the left ventricle.



Assessment

HISTORY

- patients with MI describe a heaviness, squeezing, choking, or smothering sensation.
- Patients often describe the sensation as “someone sitting on my chest.”
- The substernal pain can radiate to the neck, left arm, back, or jaw.
- Unlike the pain of angina, the pain of an MI is often more prolonged and unrelieved by rest or sublingual nitroglycerin.
- Associated findings on history include nausea and vomiting, especially for the patient with an inferior wall MI.
- These gastrointestinal complaints are believed to be related to the severity of the pain and the resulting vagal stimulation.

PHYSICAL EXAMINATION

- patients usually appear restless and in distress.
- The skin is warm and moist.
- Breathing may be labored and rapid. Fine crackles, coarse crackles, or rhonchi may be heard when auscultating the lungs.
- an increased blood pressure related to anxiety or a decreased blood pressure caused by heart failure.
- The heart rate may vary from bradycardia to tachycardia.
- On auscultation, the first heart sound may be diminished as a result of decreased contractility.
- A fourth heart sound is heard in almost all patients with MI, whereas a third heart sound is detected in only about 10% to 20% of patients.
- Transient systolic murmurs may be heard

- Patients with right ventricular infarcts may present with jugular vein distension, hypotension, peripheral edema, and an elevated central venous pressure .

DIAGNOSTIC TESTS

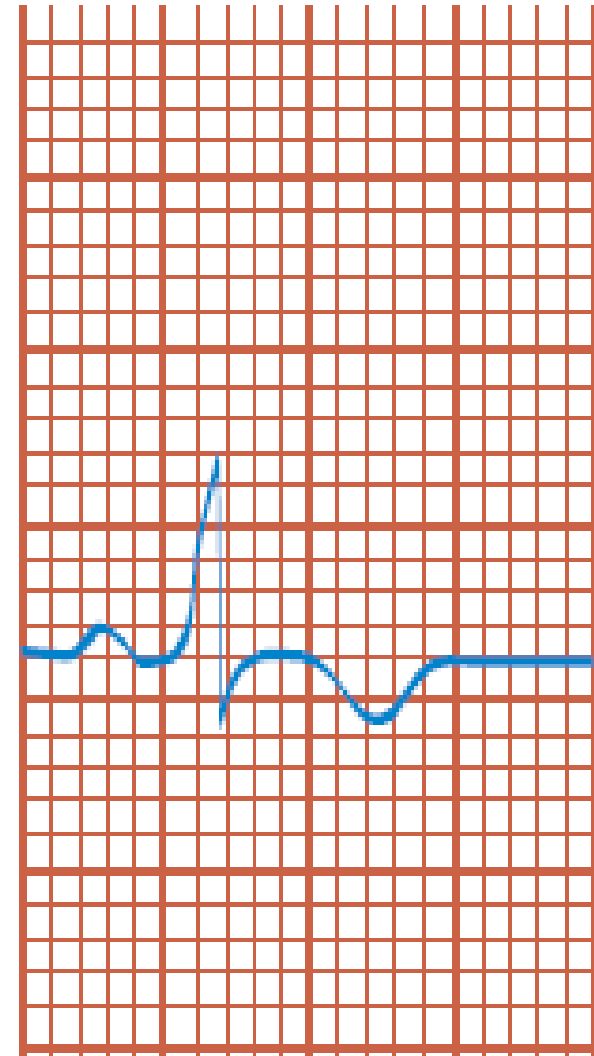
The Electrocardiogram

- An ECG can be used to detect patterns of ischemia, injury, and infarction

Ischemia.

- On the ECG, myocardial ischemia results in T-wave inversion or ST segment depression in the leads facing the ischemic area.
- The inverted T wave representative of ischemia is symmetrical, relatively narrow, and somewhat pointed.

- ST segment depressions of 1 to 2 mm or more for a duration of 0.08 second may indicate myocardial ischemia.
- Ischemia also should be suspected when a flat or depressed ST segment makes a sharp angle when joining an upright T wave rather than merging smoothly and imperceptibly with the T wave



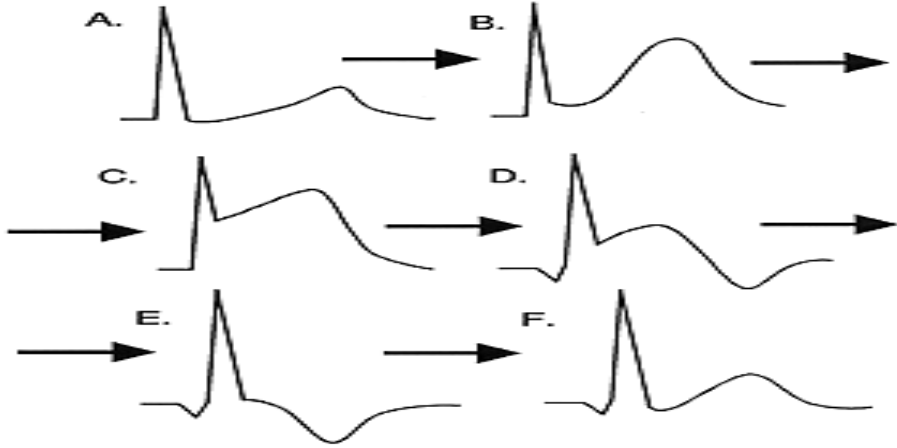
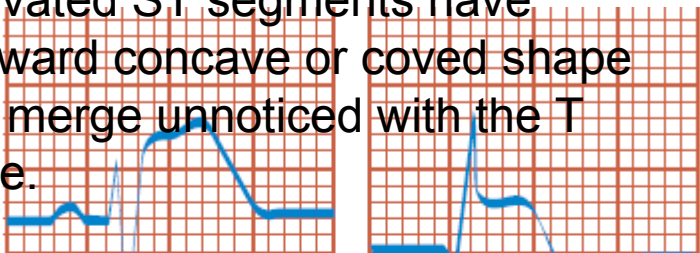
Injury.

- the injury process begins in the subendocardial layer and moves throughout the thickness of the wall of the heart like a wave.
- If the injury process is not interrupted, it eventually results in a transmural MI.
- On ECG, the hallmark of acute myocardial injury is the presence of ST segment elevations.
- In the normal ECG, the ST segment should not be elevated more than 1 mm in the standard leads or more than 2 mm in the precordial leads.
- With an acute injury, the ST segments in the leads facing the injured area are elevated.
- The elevated ST segments also have a downward concave or coved shape and merge unnoticed with the T wave

(A) ST segment elevation without T-wave inversion.

(B) ST segment elevation with T-wave inversion.

The elevated ST segments have a downward concave or coved shape and merge unnoticed with the T wave.



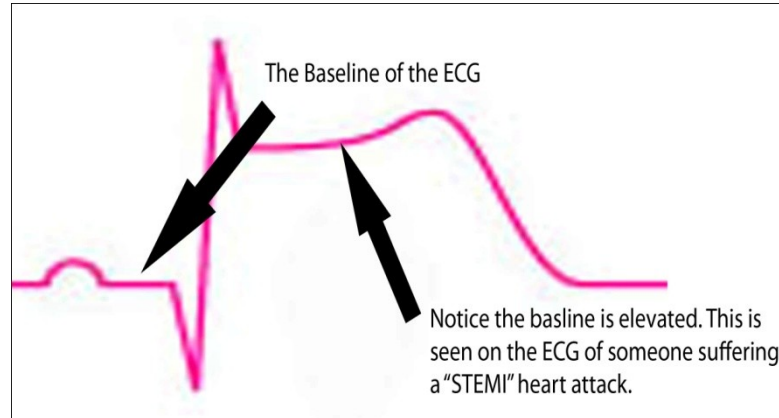
Evolution of Acute MI

- a b

ECG

- STEMI:

- Q waves , ST elevations, hyper acute T waves; followed by T wave inversions.
- Clinically significant ST segment elevations:
 - > than 1 mm (0.1 mV) in at least two anatomical contiguous leads
 - or 2 mm (0.2 mV) in two contiguous precordial leads (V2 and V3)



- Note: LBBB and pacemakers can interfere with diagnosis of MI on ECG

Infarction. When myocardial injury persists, MI is the result.

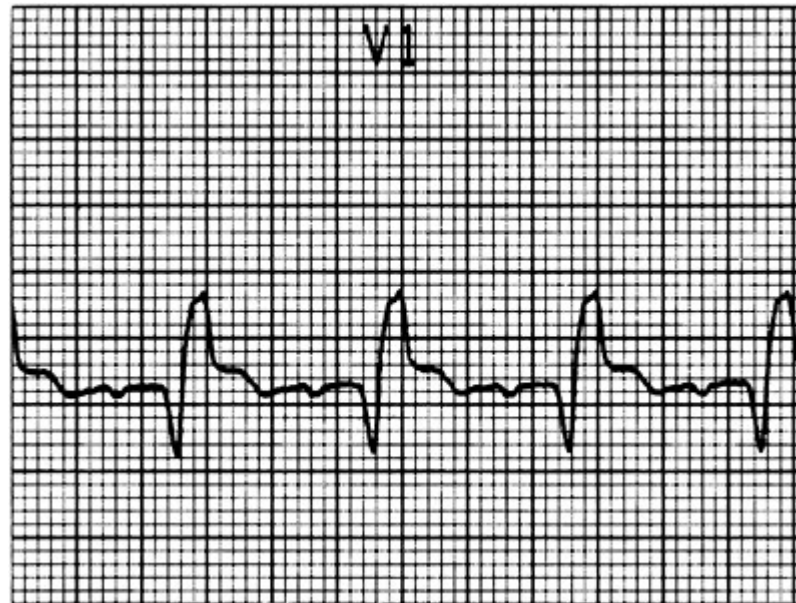
- During the earliest stage of MI, known as the hyperacute phase, **the T waves become tall and narrow**. This configuration is referred to as hyperacute or peaked T waves.
- Within a few hours, these hyperacute T waves invert.
- Next, the ST segments elevate, a pattern that usually lasts from several hours to several days.
- In addition to the ST segment elevations in the leads of the ECG facing the injured heart, the leads facing away from the injured area may show ST segment depression.
- This finding is known as **reciprocal ST segment changes**.
- Reciprocal changes are most likely to be seen at the onset of infarction, but their presence on the ECG does not last long.
- Reciprocal ST segment depressions may simply be a mirror image of the ST segment elevations.

- However, others have suggested that reciprocal changes may reflect ischemia due to narrowing of another coronary artery in other areas of the heart.
- The last stage in the **ECG evolution of an MI is the development of Q waves**, the initial downward deflection of the QRS complex.
- Q waves represent the flow of electrical forces toward the septum. Small, narrow Q waves may be seen in the normal ECG in leads I, II, III.
- Q waves compatible with an MI are usually 0.04 second (one small box) or more in width or one-fourth to one-third the height of the R wave.
- Q waves indicative of infarction usually develop within several hours of the onset of the infarction, but in some patients may not appear until 24 to 48 hours after the infarction.

- Within a few days after the MI, the elevated ST segments return to baseline.
- Persistent elevation of the ST segment may indicate the presence of a ventricular aneurysm.
- The T waves may remain inverted for several weeks, indicating areas of ischemia near the infarct region.
- Eventually, the T waves should return to their upright configuration.
- The Q waves do not disappear and therefore always provide ECG evidence of a previous MI.

Q waves indicate tissue necrosis and are permanent.

A pathologic Q wave is one that is greater than 3 mm in depth or greater than one-third the height of the R wave.



- To attain an accurate view of the right ventricle, right-sided chest leads are recorded by placing the six chest electrodes on the right side of the chest using landmarks analogous to those used on the left side
- To detect posterior wall abnormalities, three of the precordial electrodes are placed posteriorly over the heart, a view known as V7, V8, and V9.
- V7 is positioned at the posterior axillary line; V8 at the posterior scapular line; and V9 at the left border of the spine

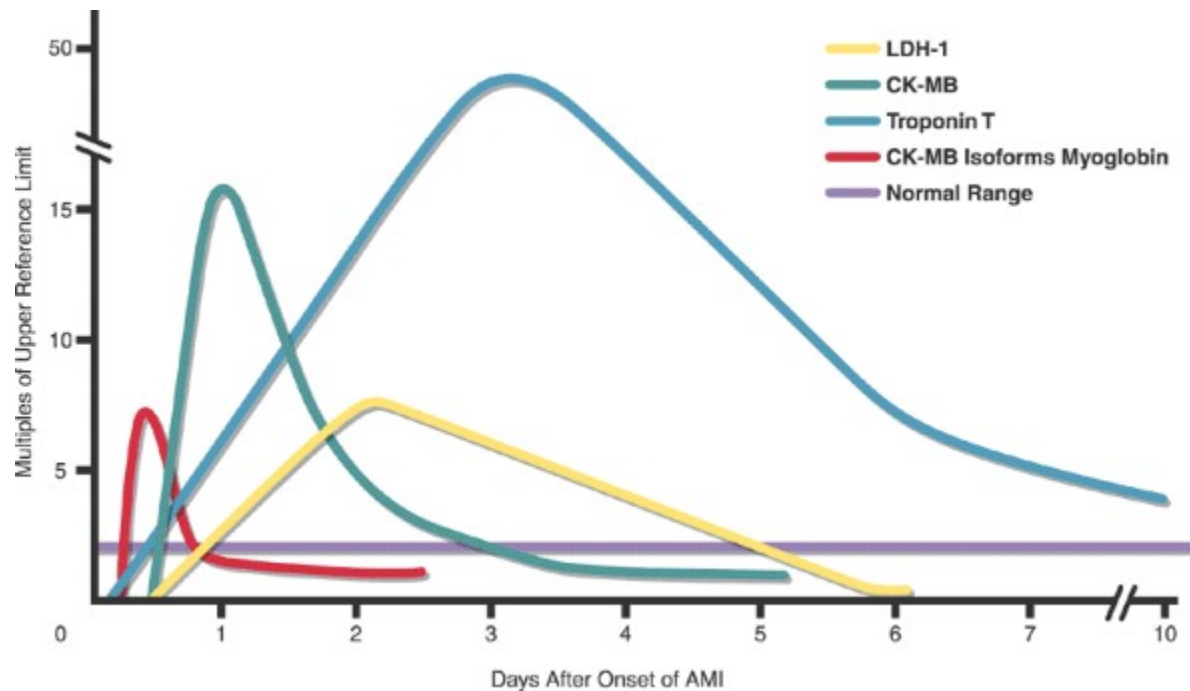
Laboratory Tests

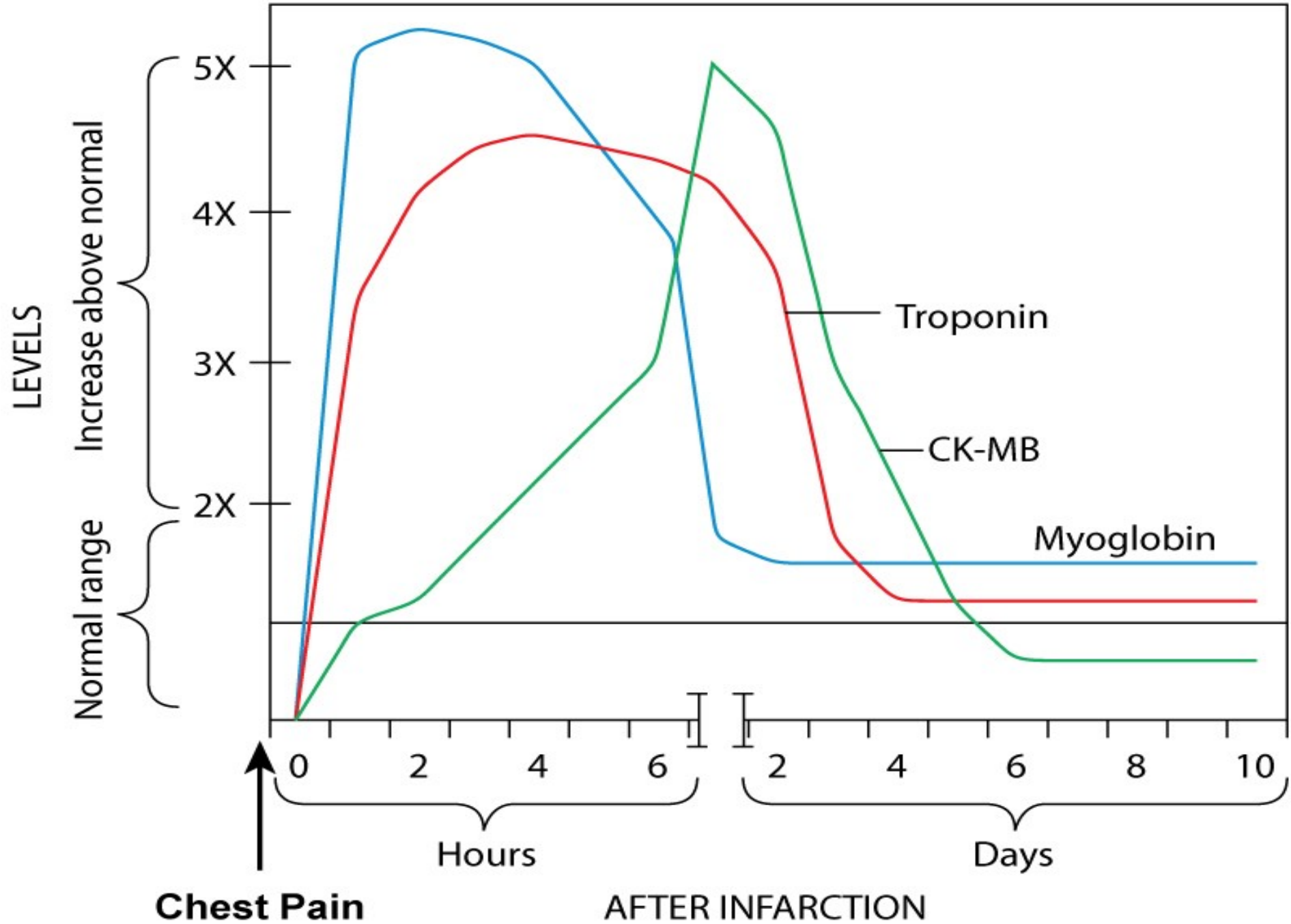
- *Creatine Kinase*
- CK-MB appears in the serum in 6 to 12 hours, peaks between 12 and 28 hours, and returns to normal levels in about 72 to 96 hours.
- Serial samplings are performed every 4 to 6 hours for the first 24 to 48 hours after the onset of symptoms
- *Creatine Kinase Isoforms*: CK-MB1 is the isoform found in the plasma, and CK-MB2 is found in the tissues. In the patient with an MI, the CK-MB2 level rises, resulting in a CK-MB2 to CK-MB1 ratio greater than one

- *Myoglobin*: Myoglobin is an oxygen-binding protein found in skeletal and cardiac muscle. Myoglobin's release from ischemic muscle occurs earlier than the release of CK.
- The myoglobin level can elevate within 1 to 2 hours of acute MI and peaks within 3 to 15 hours.
- Because myoglobin is also present in skeletal muscle, an elevated myoglobin level is not specific for the diagnosis of MI. Consequently, its diagnostic value in detecting an MI is limited

- *Troponin*. (troponin T and troponin I):
- Troponin I levels rise in about 3 hours, peak at 14 to 18 hours, and remain elevated for 5 to 7 days.
- Troponin T levels rise in 3 to 5 hours and remain elevated for 10 to 14 days

Release of cardiac markers after acute myocardial infarction (AMI).





Management

EARLY MANAGEMENT

- The patient's history and 12-lead ECG are the primary methods used to determine initially the diagnosis of MI.
- The ECG is examined for the presence of ST segment elevations of 1 mV or greater in contiguous leads.
- 1. Administer aspirin, 160 to 325 mg chewed.
- 2. After recording the initial 12-lead ECG, place the patient on a cardiac monitor and obtain serial ECGs.
- 3. Give oxygen by nasal cannula.

- 4. Administer sublingual nitroglycerin (unless the systolic blood pressure is less than 90 mm Hg or the heart rate is less than 50 or greater than 100 beats/minute).
- 5. Provide adequate analgesia with morphine sulfate.

Thrombolytic Therapy

- Thrombolytic drugs lyse coronary thrombi by converting plasminogen to plasmin.
- Thrombolytic therapy provides maximal benefit if given within the first 3 hours after the onset of symptoms.
- Significant benefit still occurs if therapy is given up to 12 hours after onset of symptoms.

Absolute Contraindications

- Previous hemorrhagic stroke at any time;
other strokes
or cerebrovascular events within 1 year
- Known intracranial neoplasm
- Active internal bleeding (does not include menses)
- Suspected aortic dissection

Thrombolytic Therapy

Cautions/Relative Contraindications

- Severe uncontrolled hypertension on presentation (blood pressure >180/110 mm Hg)
- History of prior cerebrovascular accident or known intracerebral disease not covered in contraindications
- Current use of anticoagulants in therapeutic doses (international normalized ratio [INR] ≥ 2.3); known bleeding diathesis
- Recent trauma (within 2–4 weeks), including head trauma or traumatic or prolonged (>10 minutes) cardiopulmonary resuscitation (CPR) or major surgery (<3 weeks)

- Noncompressible vascular punctures
- Recent (within 2–4 weeks) internal bleeding
- For streptokinase/anistreplase: prior exposure (especially within 5 days to 2 years) or prior allergic reaction
- Pregnancy
- Active peptic ulcer

Primary Percutaneous Transluminal Coronary Angioplasty (PTCA)

- (PTCA) is an effective alternative to reestablish blood flow to ischemic myocardium.
- Primary PTCA is an invasive procedure in which the infarct-related coronary artery is dilated during the acute phase of an MI without prior administration of thrombolytic agents.
- Primary PTCA may be an excellent reperfusion alternative for patients ineligible for thrombolytic therapy.
- carefully monitor the patient after a primary PTCA for evidence of complications.
- These complications can include retroperitoneal or vascular hemorrhage, other evidence of bleeding, early acute reocclusion, and late restenosis.

INTENSIVE AND INTERMEDIATE CARE MANAGEMENT

- Prophylactic antidysrhythmics during the first 24 hours of hospitalization **are not recommended**.
- Daily aspirin is continued on an indefinite basis.
- Clopidogrel
- IV beta blocker therapy should be administered within the initial hours of the evolving infarction, followed by oral therapy provided there are no contraindications.
- Beta blockers are one of the few pharmacological agents that have been shown to reduce morbidity and mortality in the patient with an MI.
- They reduce oxygen demand by decreasing the heart rate and contractility.
- They also increase coronary artery filling by prolonging
- diastole.

- Calcium channel blockers
- may be given to patients in whom beta blocker therapy is ineffective or contraindicated.
- Angiotensin-converting enzyme (ACE) inhibitors are administered to patients with anterior wall MI and to patients who have an MI with heart failure in the absence of significant hypotension.
- ACE inhibitors help prevent ventricular remodeling (dilation) and preserve ejection fraction.
- Heparin is given to patients undergoing percutaneous or surgical revascularization and for those receiving thrombolytic therapy with alteplase.
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Additional Diagnostic Tests:

- *Radionuclide Imaging*
- *Echocardiogram*
- *Stress Test*
- *Coronary Angiography*

Complications

Vascular Complications

- Recurrent ischemia
- Recurrent infarction

Mechanical Complications

- Left ventricular free wall rupture
- Ventricular septal rupture
- Papillary muscle rupture with acute mitral regurgitation

Myocardial Complications

- Diastolic dysfunction
- Systolic dysfunction
- Congestive heart failure
- Hypotension/cardiogenic shock
- Right ventricular infarction
- Aneurysm formation.

Pericardial Complications

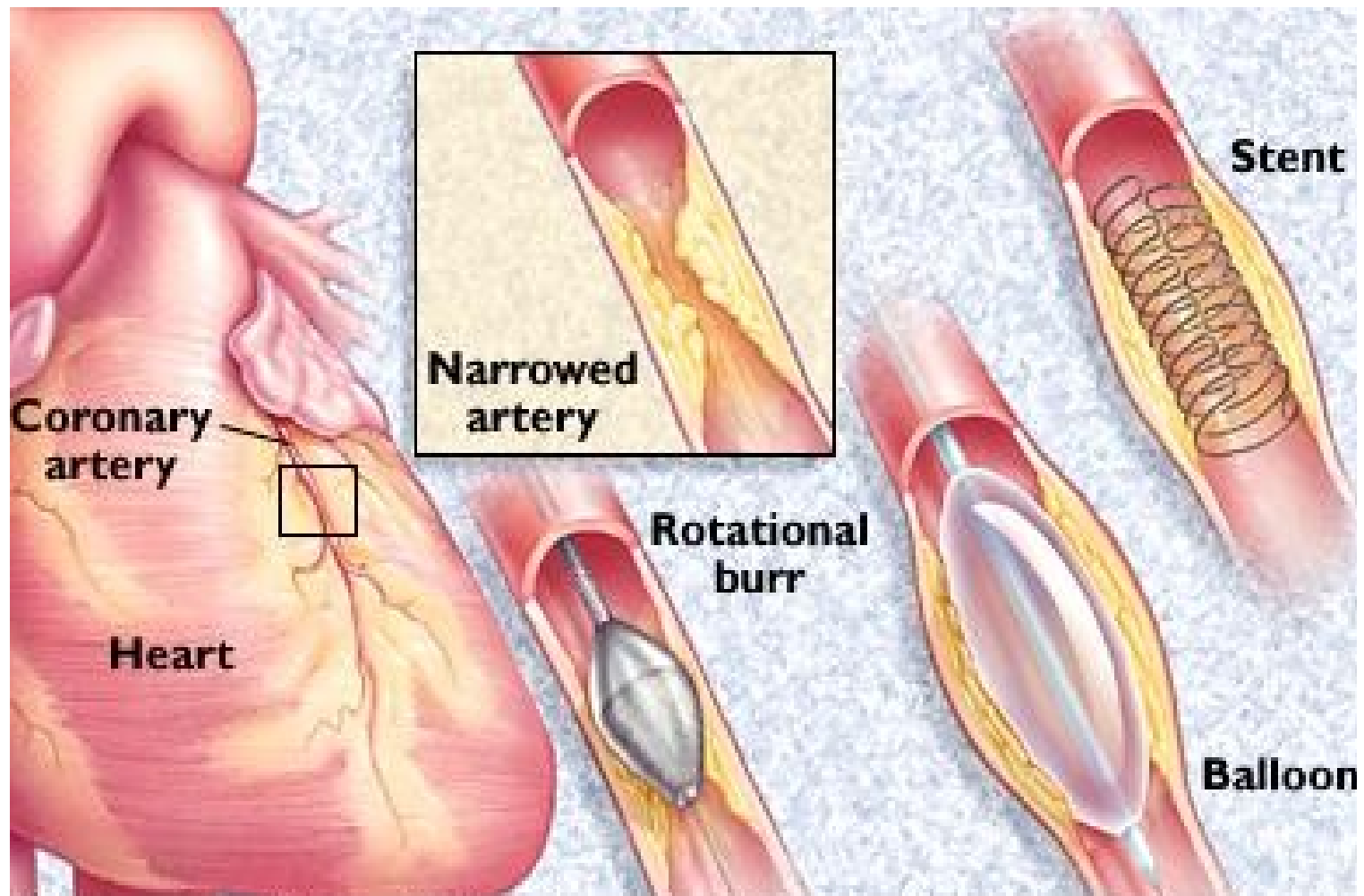
- Pericarditis
- Dressler's syndrome
- Pericardial effusion

Thromboembolic Complications

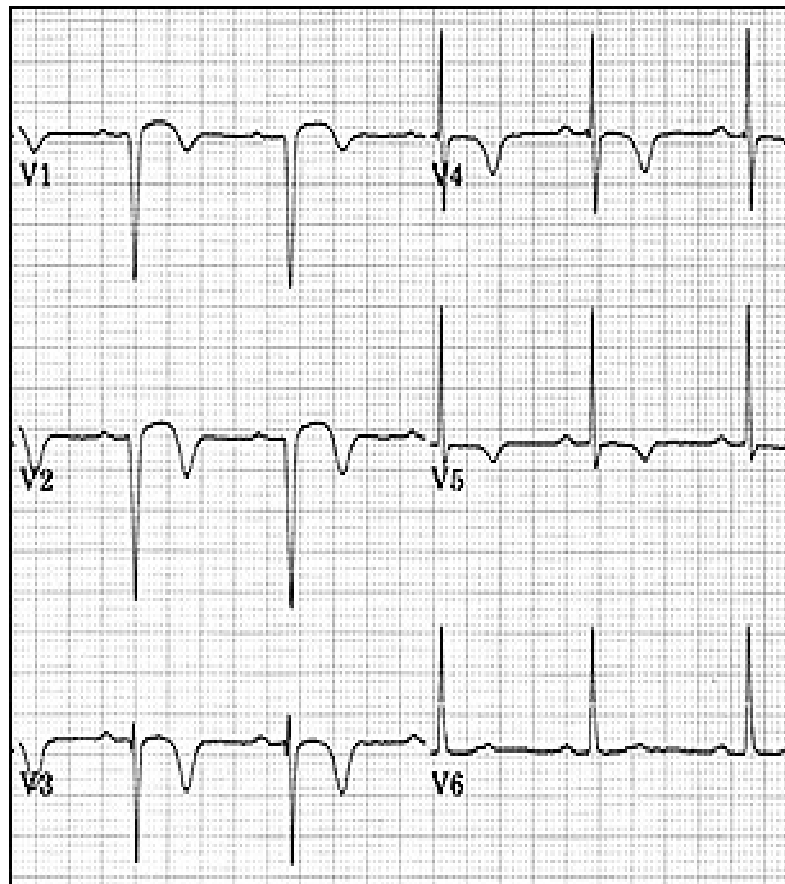
- Mural thrombosis
- Systemic thromboembolism

Electrical Complications

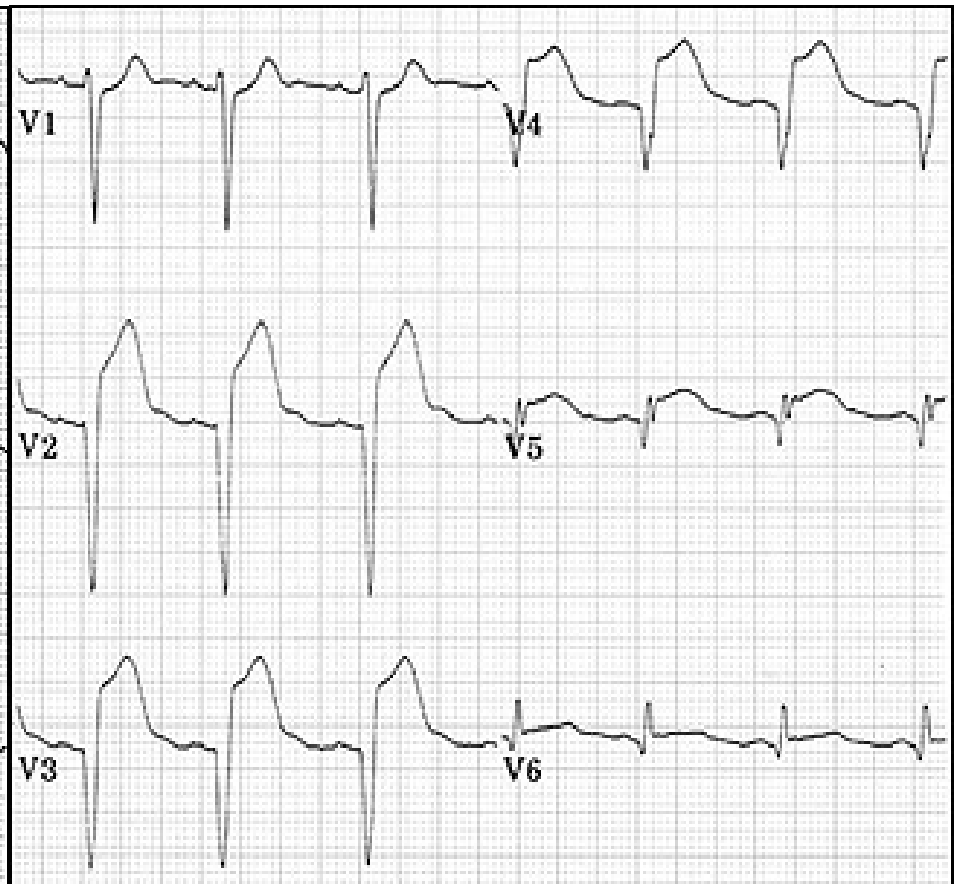
- Ventricular tachycardia
- Ventricular fibrillation
- Supraventricular tachydysrhythmias
- Bradydysrhythmias
- Atrioventricular block (first, second, or third degree)



Anterior MI



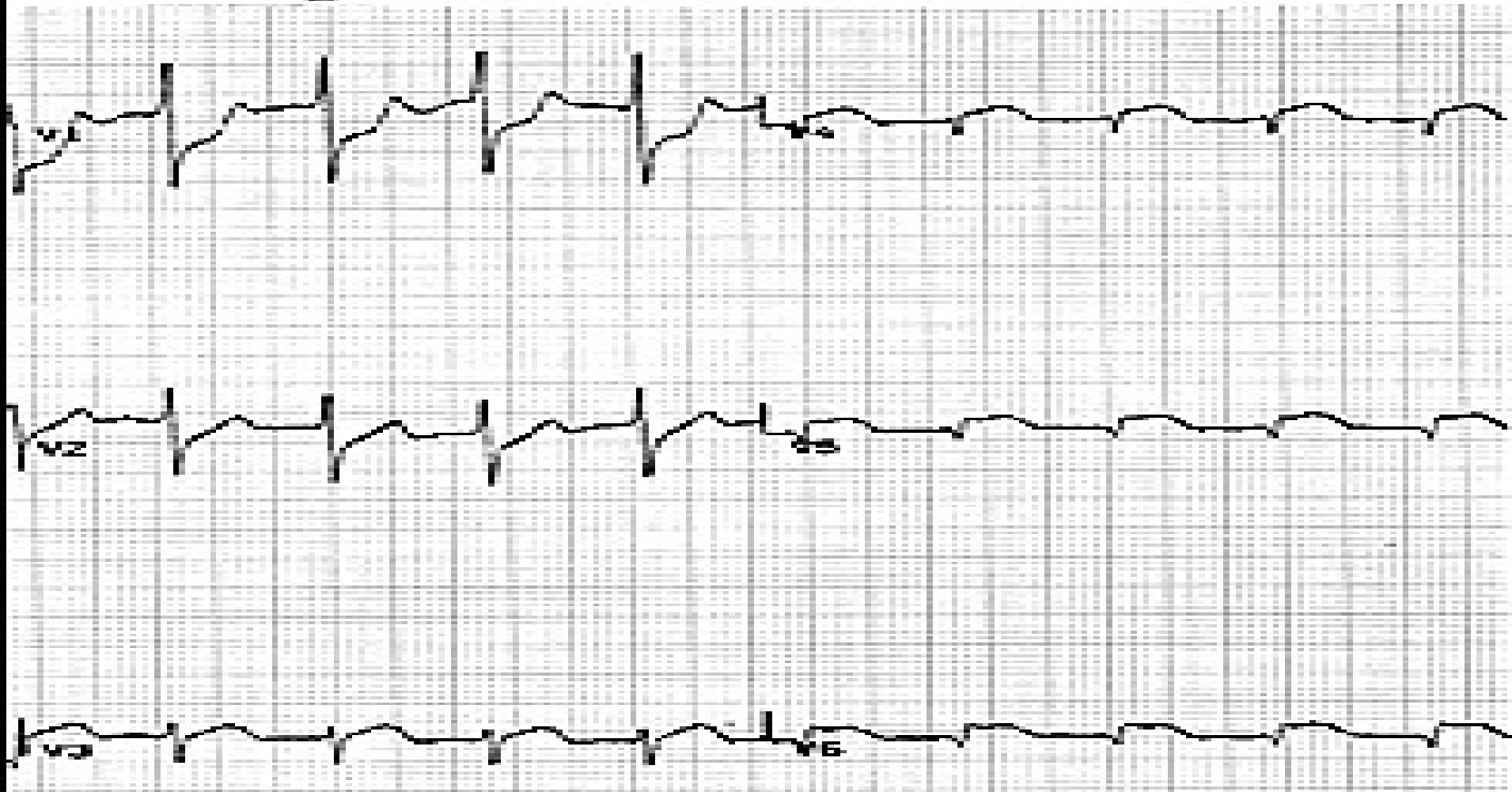
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Right Ventricular MI

Right side chest leads V1R - V6R



Inferoposterior MI

