Acute Myocardial Infarction

Snejana Vetrila PhD
Associate professor

Definition

- Acute myocardial infarction (MI) is a clinical syndrome that results from occlusion of a coronary artery, with resultant death of cardiac myocytes in the region supplied by that artery

General Considerations

- The risk of having an acute MI increases with age, male gender, smoking, dyslipidemia, diabetes, hypertension, abdominal obesity, a lack of physical activity, low daily fruit and vegetable consumption, alcohol overconsumption, and psychosocial index
- As much as 90% of the risk of acute MI has been attributed to the modifiable risk factors

THIRD Universal Definition
Acute Miocardial Infarction

V*ersion 2012*

(STEMI)

5 tipes

Type I – spontaneous AMI

Typical rise of biochemical markers of myocardial necrosis (troponin CK-MB) with at least one of the following:

- 1. Ischemic clinical symptoms
- 2. ECG new changes indicative of ischemia (ST segment elevation or depression or LBBB
- 3. Development of pathologic Q waves on the ECG
- 4. Imaging data of new disturbances of regional contractility
- 5.Identification of intracoronarian trombus on angiografy or autopsy

Type II AMI - Sudden death

symptoms of ischemia and suggesting the ECG ischemic changes or new bundle block, but death was before biomarkers raised

Type III AMI -related with percutaneous coronary intervention

- elevation of troponin cTn (> 5 X99 th, from normal value or raising of cTn >20% if troponin was elevated before procedure

Type IV- AMI related with thrombosis of implanted stent

- diagnosed by angiography or autopsy and elevation of cardiac biomarkers at least one time from normal

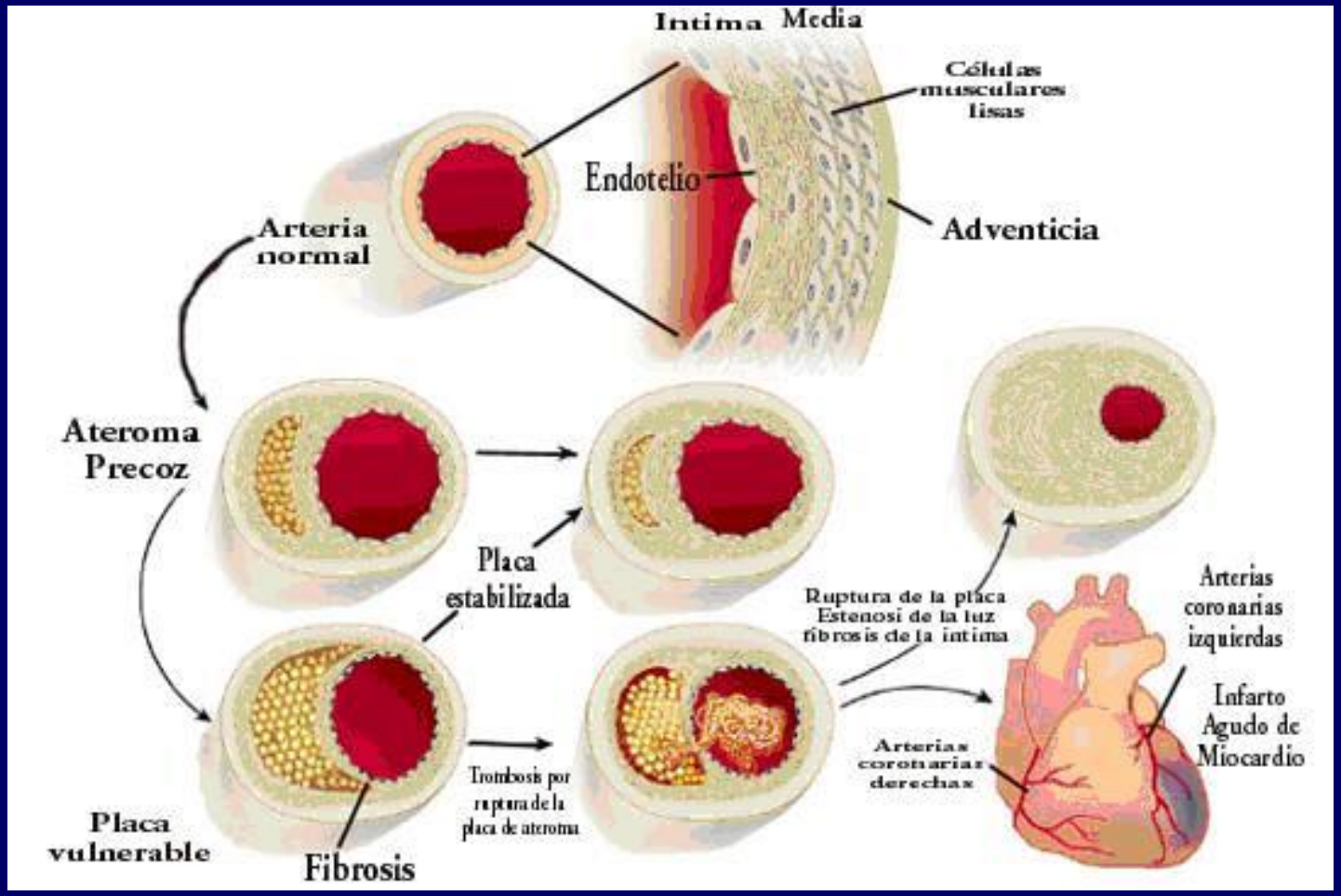
Type V- AMI related with by-pass

- Elevation of cardiac biomarkers (>10* in patients with normal cTn
- Associated with Q pathologic or LBBB, or new occlusion of coronary artery or imagistic data of disturbances of contractility

Pathophysiology & Etiology

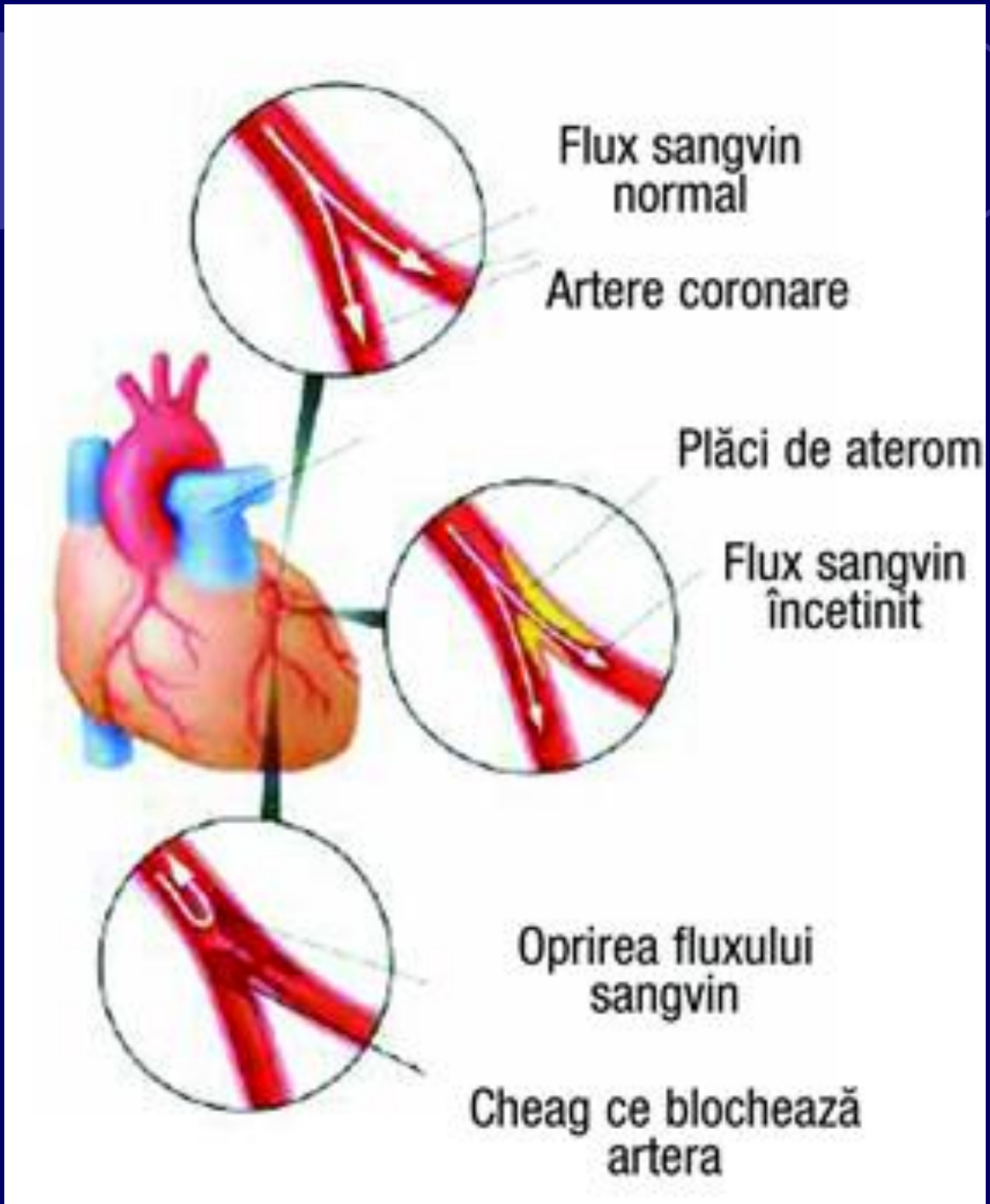
- A prolonged imbalance between myocardial oxygen supply and demand leads to the death of myocardial tissue
- Coronary atherosclerosis is an essential part of the process in most patients
- At any stage in this process, the atherosclerotic lesion may erode, ulcerate, fissure, or rupture

Acute MI occurs when this thrombosis propagates and occludes flow within the artery, resulting in necrosis of cardiomyocytes distal to the obstruction



Procoagulant factors

- **tissue factor** within the endothelial cells of the coronary artery, can cause thrombosis
- This potent procoagulant stimulus results in thrombus development in this region


Flux sangvin normal
Artere coronare
Plăci de aterom
Flux sangvin încetinit
Oprirea fluxului sangvin
Cheag ce blochează artera

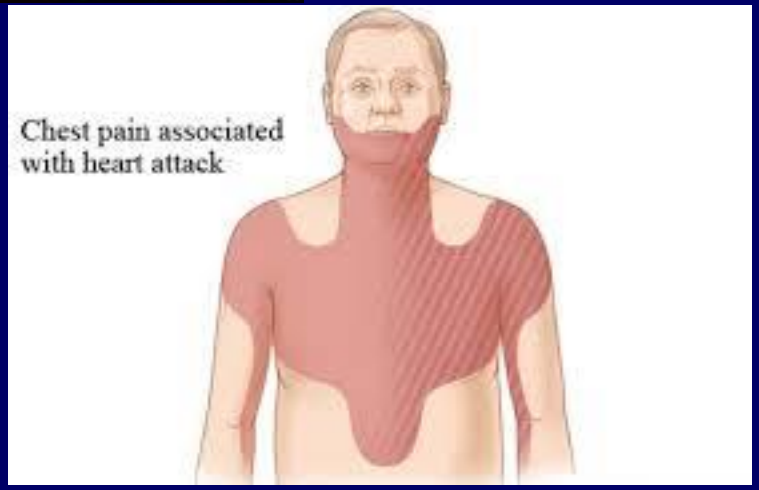
Symptoms and Signs

- Chest pain- most common symptom
- it is usually described as “pressure,” “dull,” “squeezing,” or “aching,”
- The discomfort is usually in the center of the chest and commonly radiates to the left arm or the neck

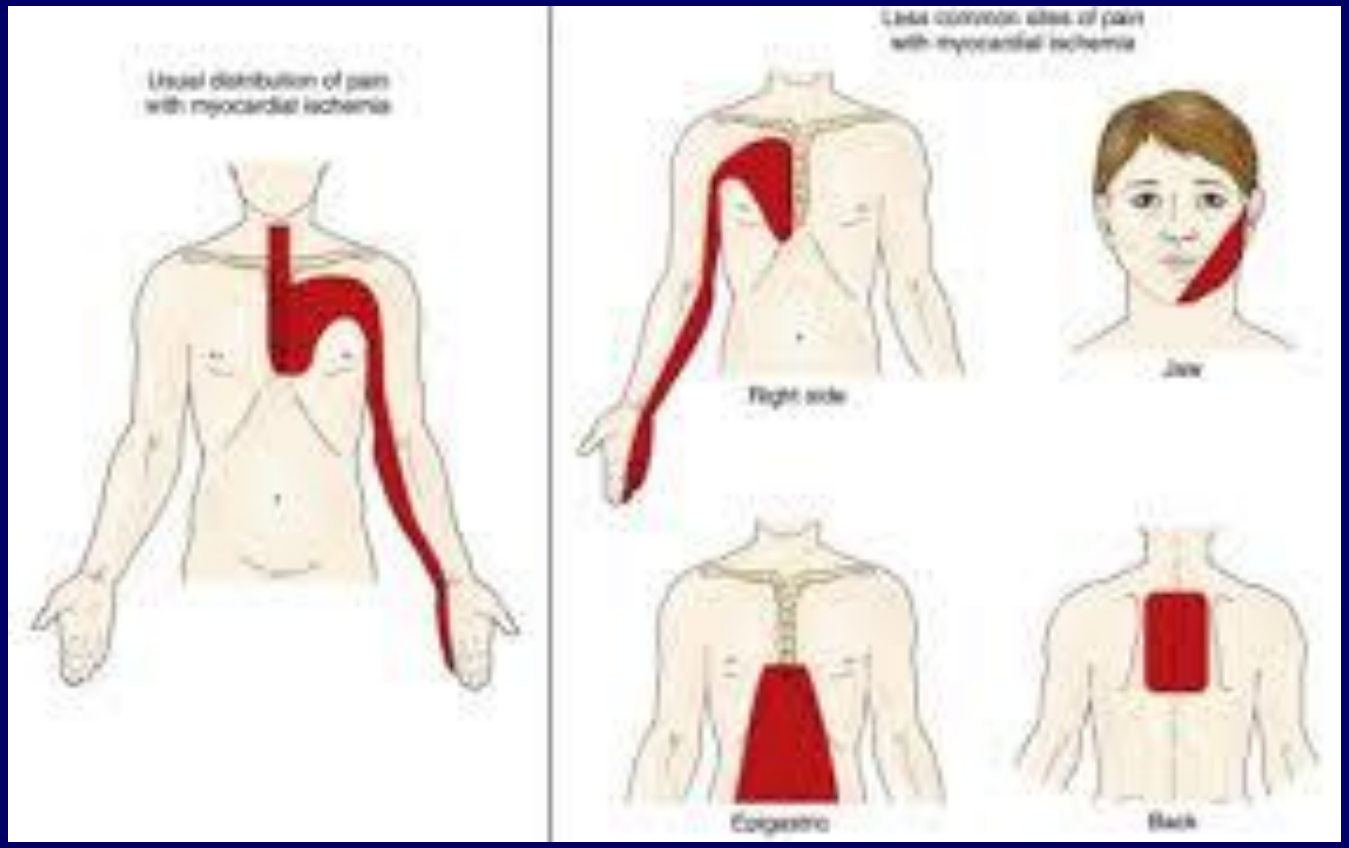
Chest pain

- May also radiate to the right arm, epigastrium, jaw, teeth, or the back
- The nature of the pain may lead patients to place a hand or fist over the sternum (**Levine sign**)
- These clinical signs and symptoms were originally defined in groups of males It is now clear that women often have more atypical symptoms

Simptoms

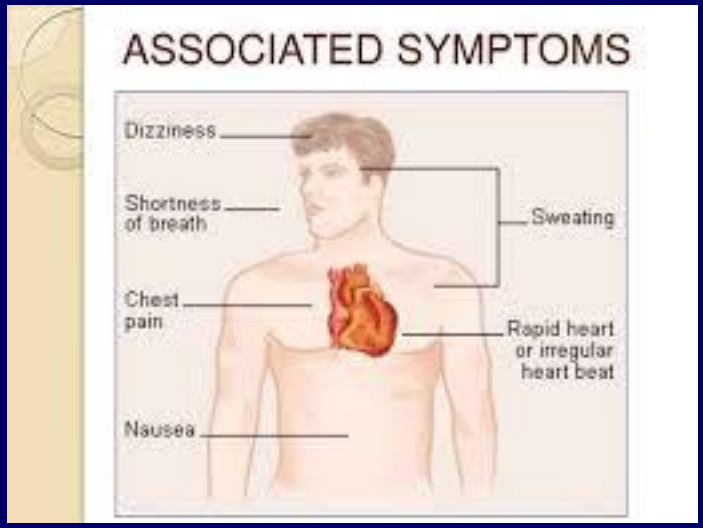


Location of pain in AMI



Associated symptoms

- Dyspnea
- nausea (particularly in inferior infarction)
- palpitations
- a sense of impending doom



Atypical presentations

- diabetic person may have abdominal pain that mimics the discomfort commonly associated with gallstones
- In elderly patients, heart failure is often the presenting symptom.

Physical Examination

- Findings may vary tremendously, from markedly abnormal, with signs of severe congestive heart failure, to totally normal
- Most patients with a large MI appear pale or sweaty and may be agitated or restless

Auscultation of heart

- Heart rate should be measured for arrhythmia, heart block, or sinus tachycardia
- This is crucial before administration of β-blockers

Assessment of blood pressure

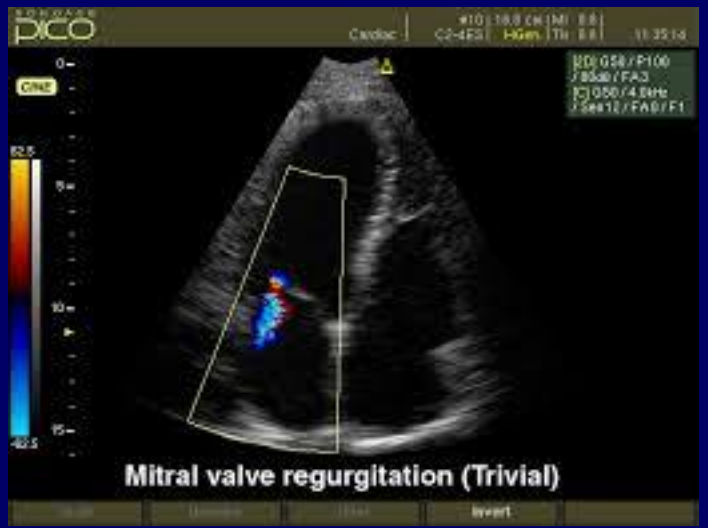
- Hypertension (which may be due to the pain) is a contraindication to fibrinolytic treatment and must be treated emergently
- Hypotension in the setting of acute MI may be due to cardiogenic shock, which alters treatment strategy

The jugular venous pulse

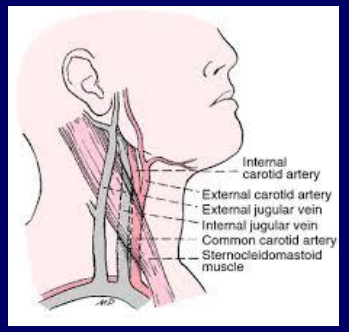
- Its elevation in the setting of inferior MI without left heart failure suggests right ventricular MI
- Detection of right ventricular MI is vital because it portends a much worse prognosis than isolated inferior MI, and the management strategy is different than isolated inferior MI

Cardiac auscultation

- Acute MI may result in ischemic mitral regurgitation with a soft S1 and a pansystolic murmur
- Acquired ventricular septal defect (VSD) may also result in a pansystolic murmur



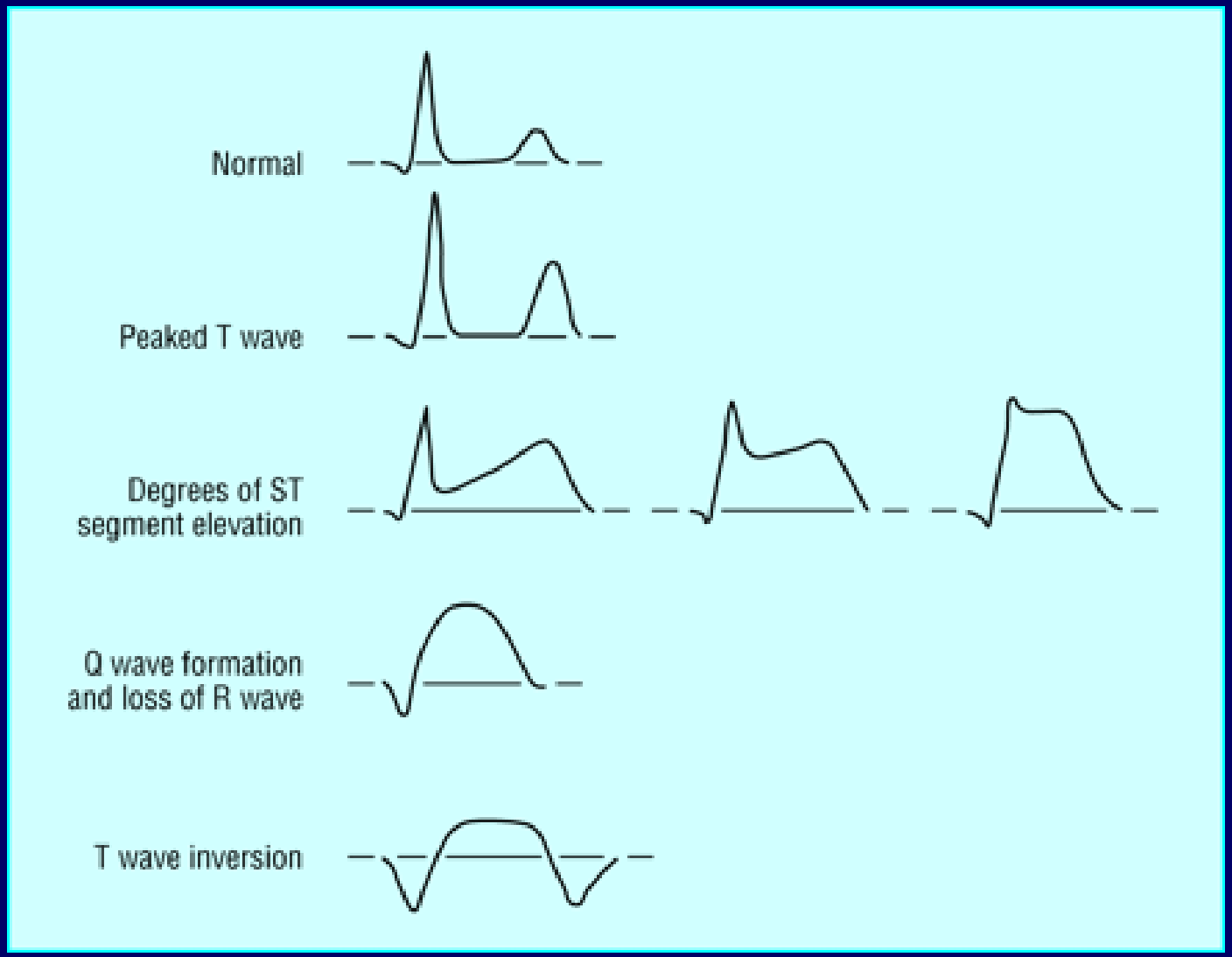
- The presence of a pericardial friction rub may indicate established infarction which has happened days earlier
- Heart failure due to large infarctions may result in a third heart sound. Signs of left heart failure, such as rales and pulmonary hypertension, should also be sought



Diagnostic Studies

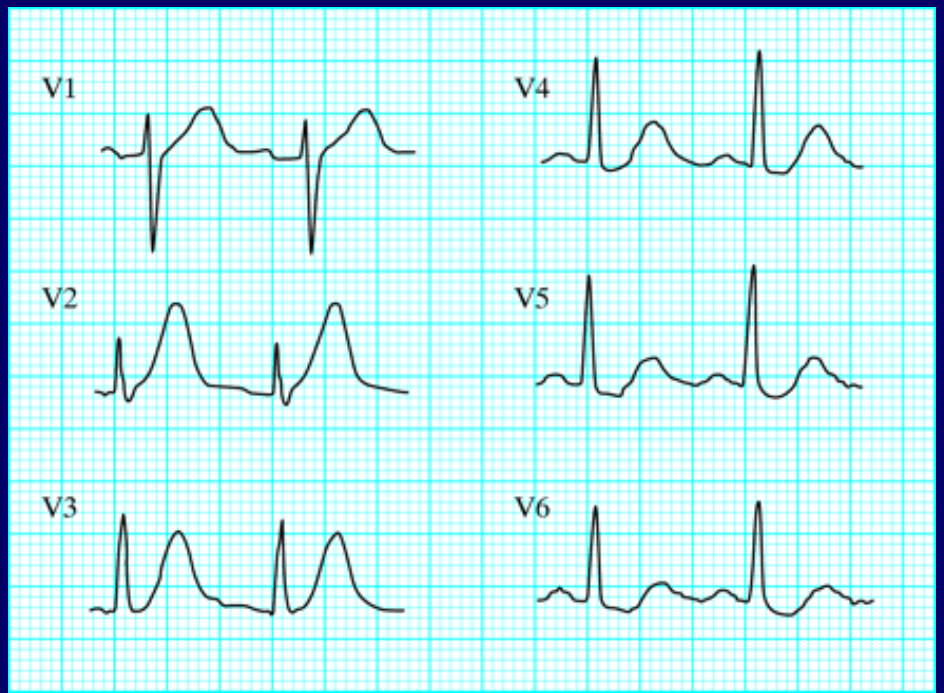
- 12-lead ECG
- It should be performed as soon as possible, preferably within 10 minutes, after the patient's arrival in the emergency department For a diagnosis of ST elevation MI (STEMI), ST elevation must be present in at least two contiguous leads

Sequence of changes seen during evolution of myocardial infarction



Phase I - Hyperacute T waves

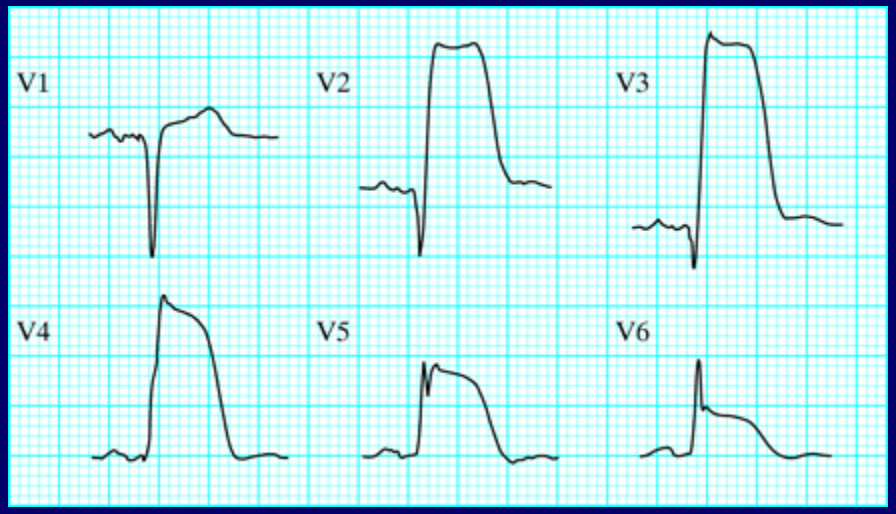
- These changes in T waves are usually present for only 5 to 30 minutes after the onset of the infarction and are followed by ST segment changes



Phase II-ST segment changes

- Is usually evident within hours of the onset of symptoms
- Initially the ST segment may straighten, with loss of the ST-T wave angle
- Then the T wave becomes broad and the ST segment elevates, losing its normal concavity

ST segment elevation (showing “tombstone” R waves)



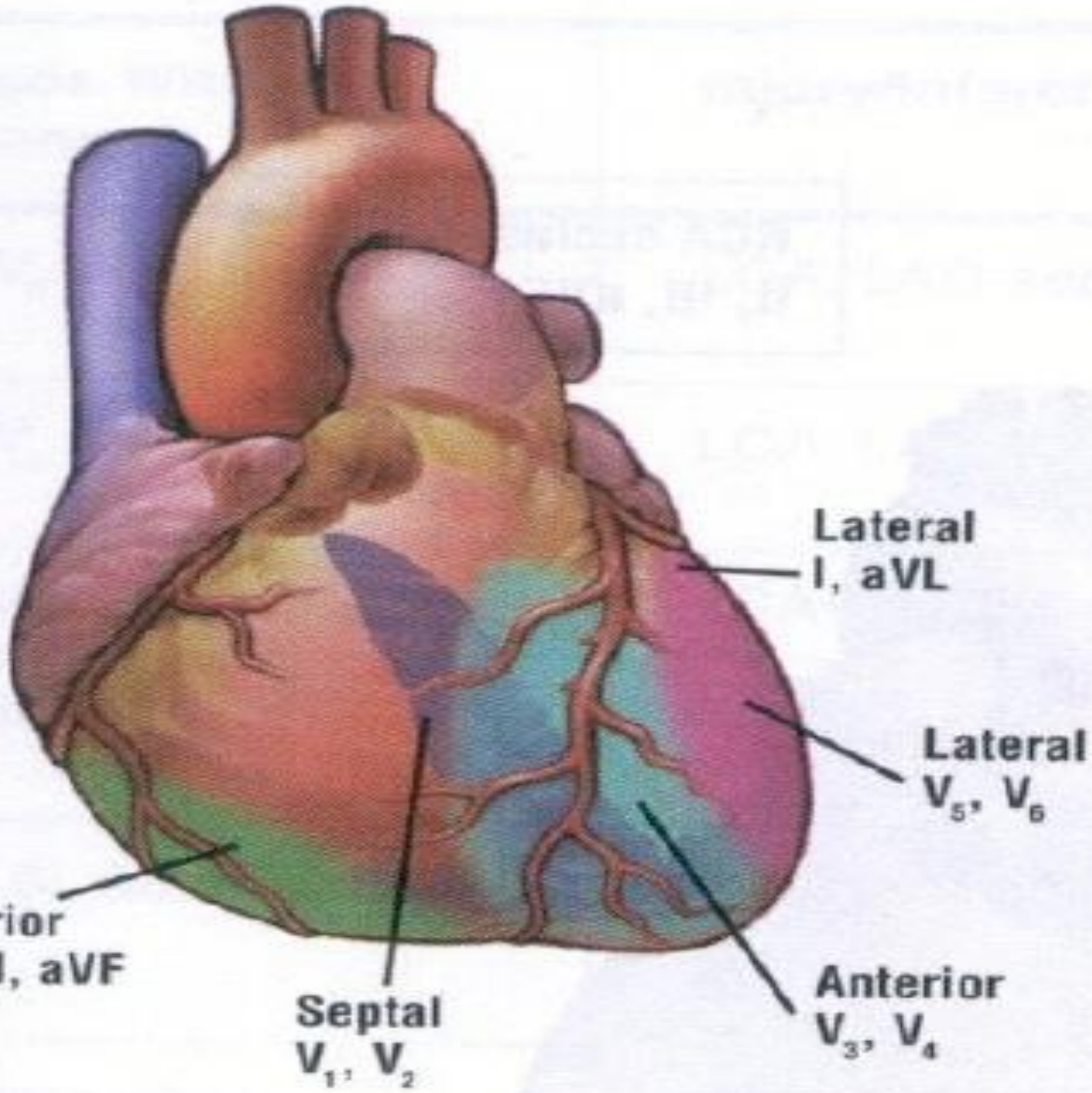
Phase III-pathological Q waves

- As the acute myocardial infarction evolves, changes to the QRS complex include loss of R wave height and the development of pathological Q waves

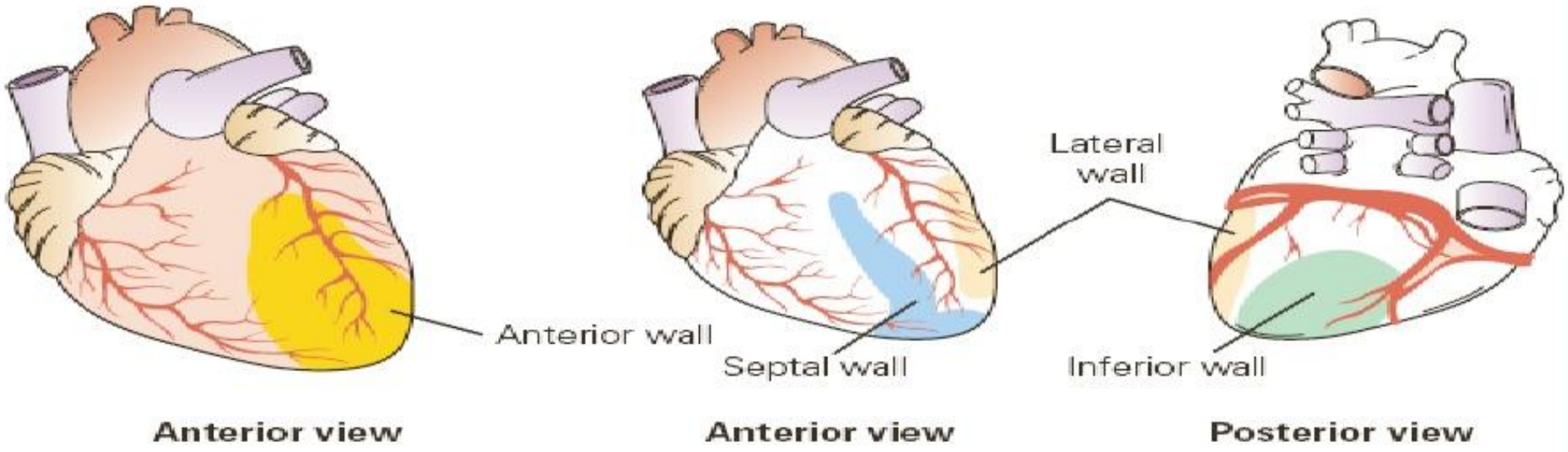
Localisation of site of infarction

- *Inferior wall*—Leads II, III, and aVF
- *Anterior wall*—Leads V1 to V4
- *Lateral wall*—Leads I, aVL, V5, and V6

A


Lateral
I, aVL
Lateral
V_5, V_6
Inferior
II, III, aVF
Septal
V_1, V_2
Anterior
V_3, V_4

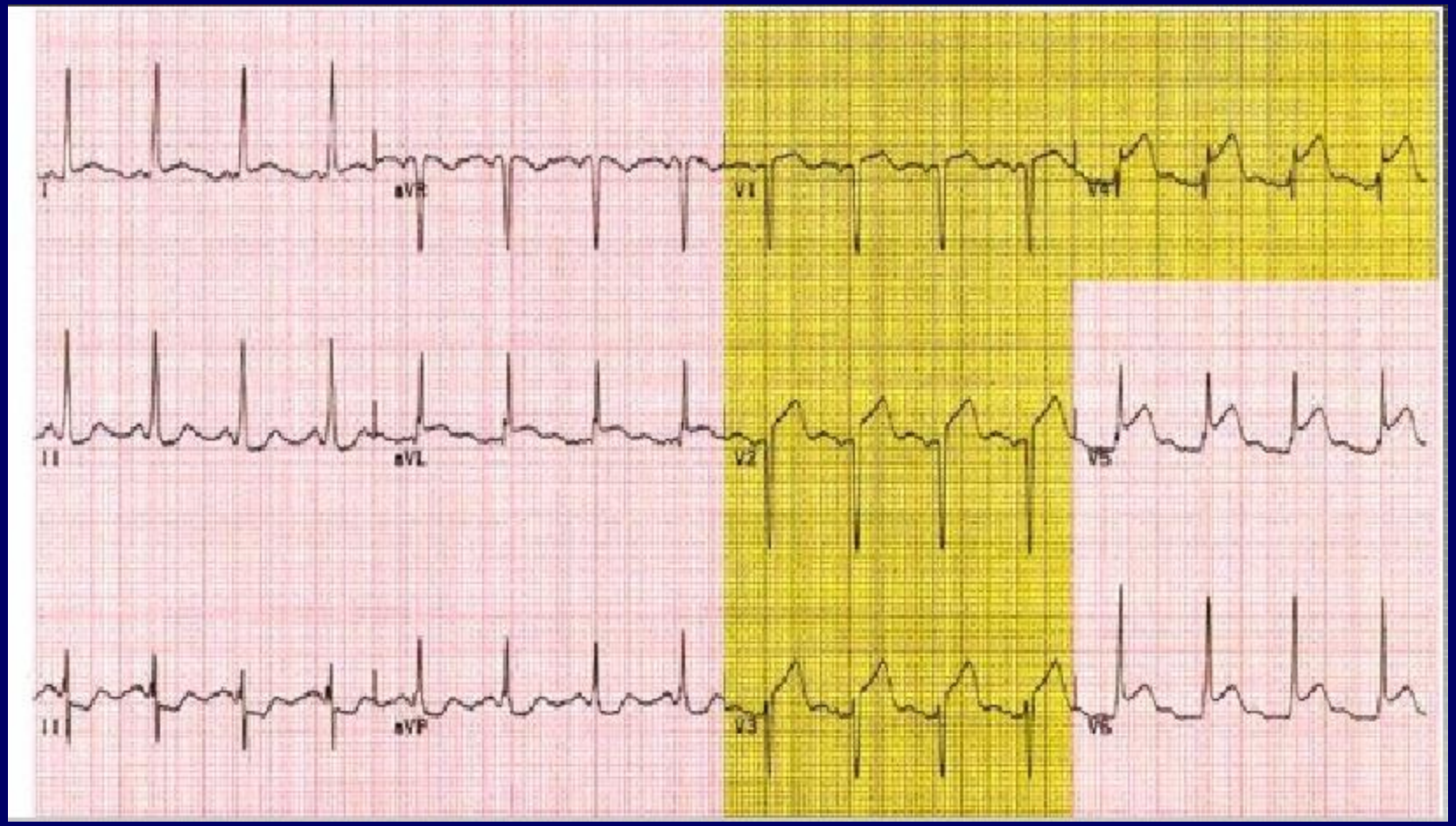
Ischemia, Injury, or Infarction in Relation to the Heart



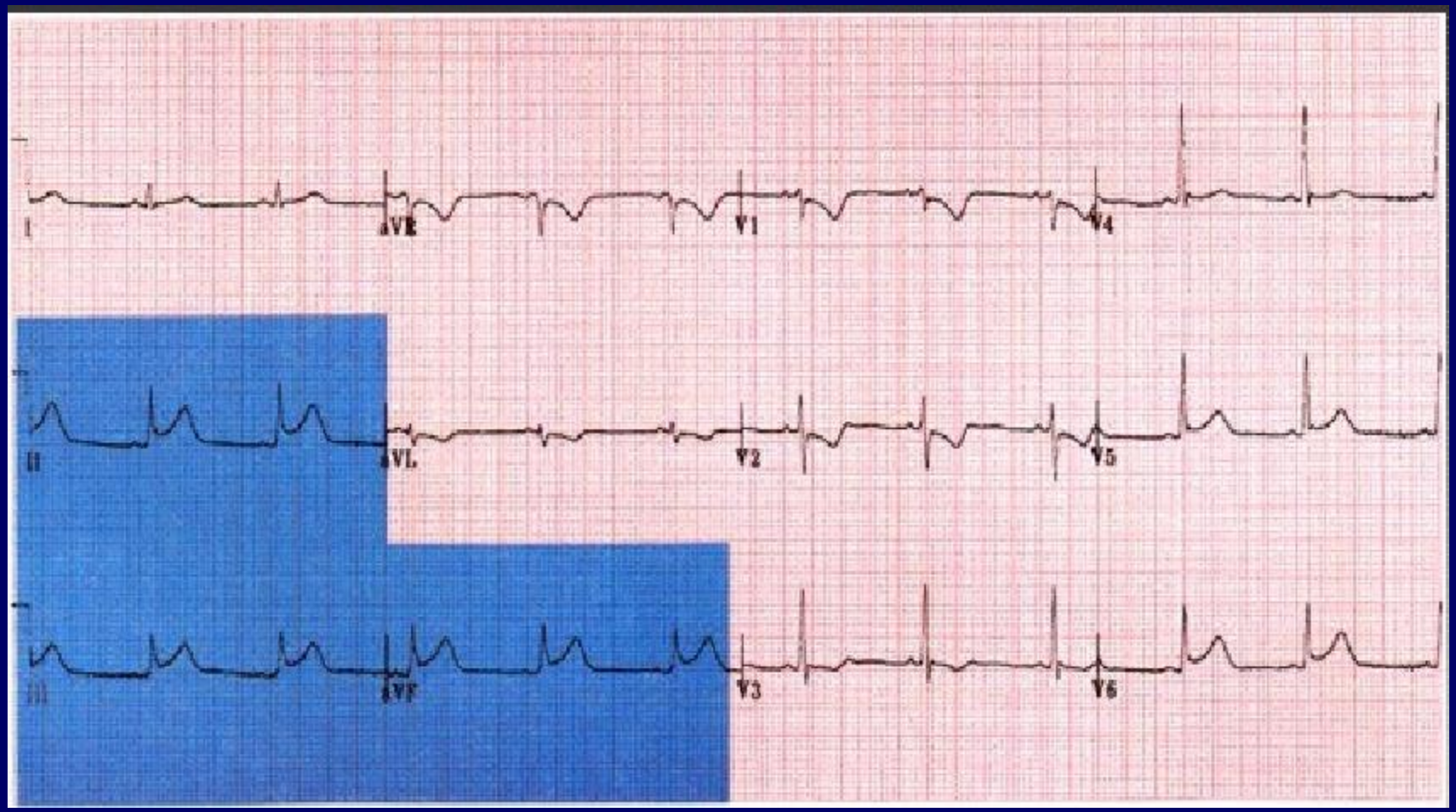
Location of MI by ECG Leads

I lateral	aVR	V_1 septal	V_4 anterior
II inferior	aVL lateral	V_2 septal	V_5 lateral
III inferior	aVF inferior	V_3 anterior	V_6 lateral

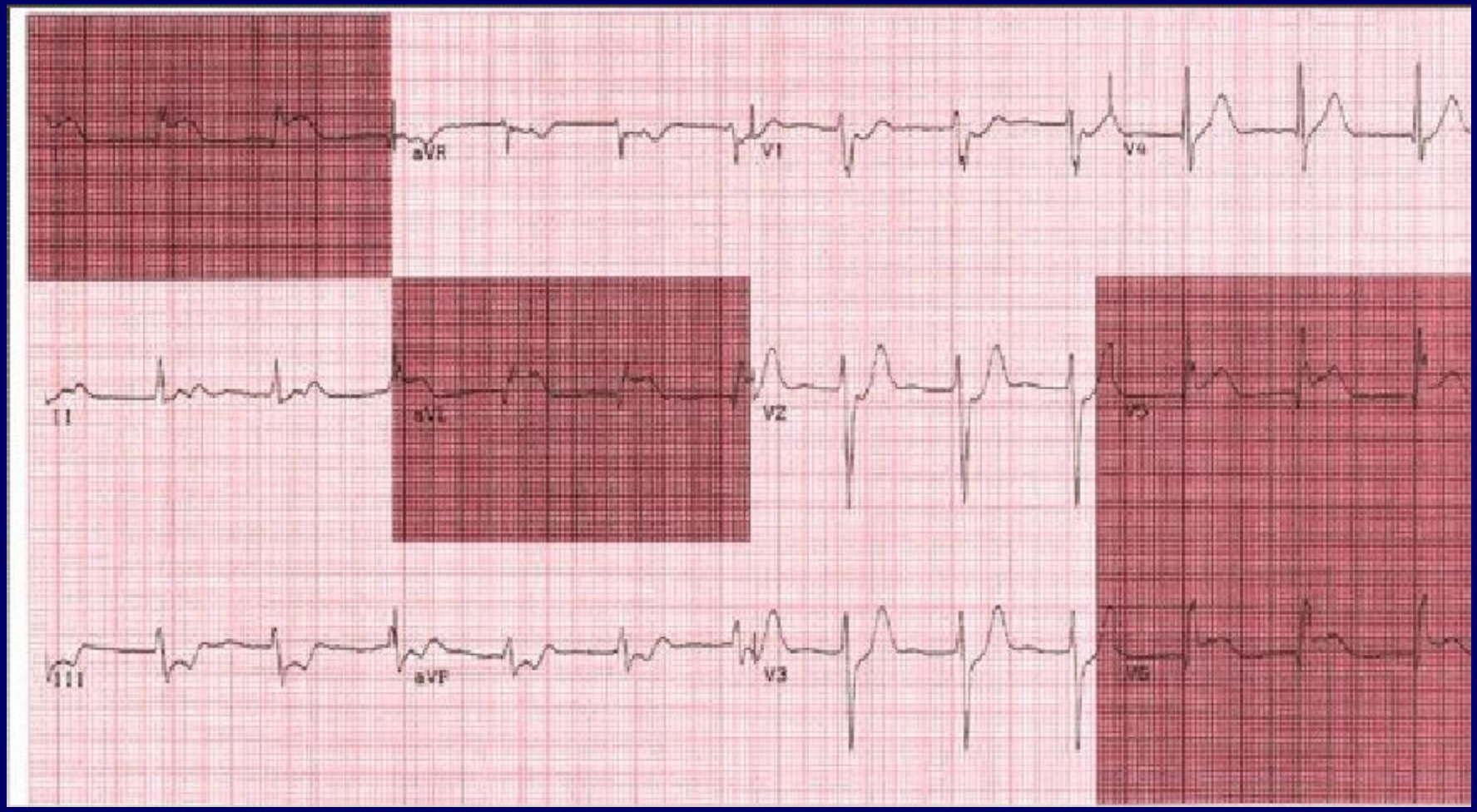
IMA ANTERIOR



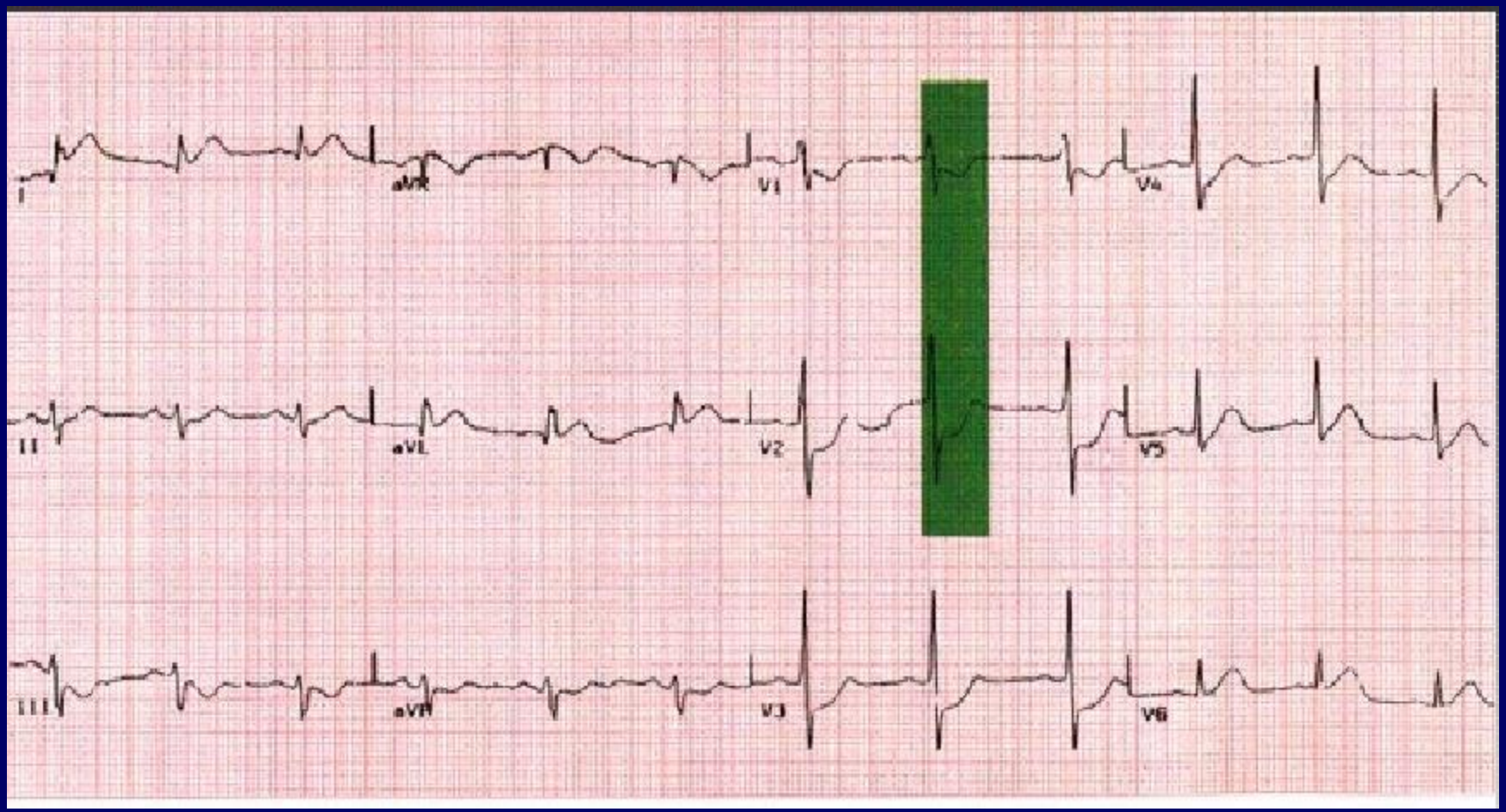
IMA INFERIOR

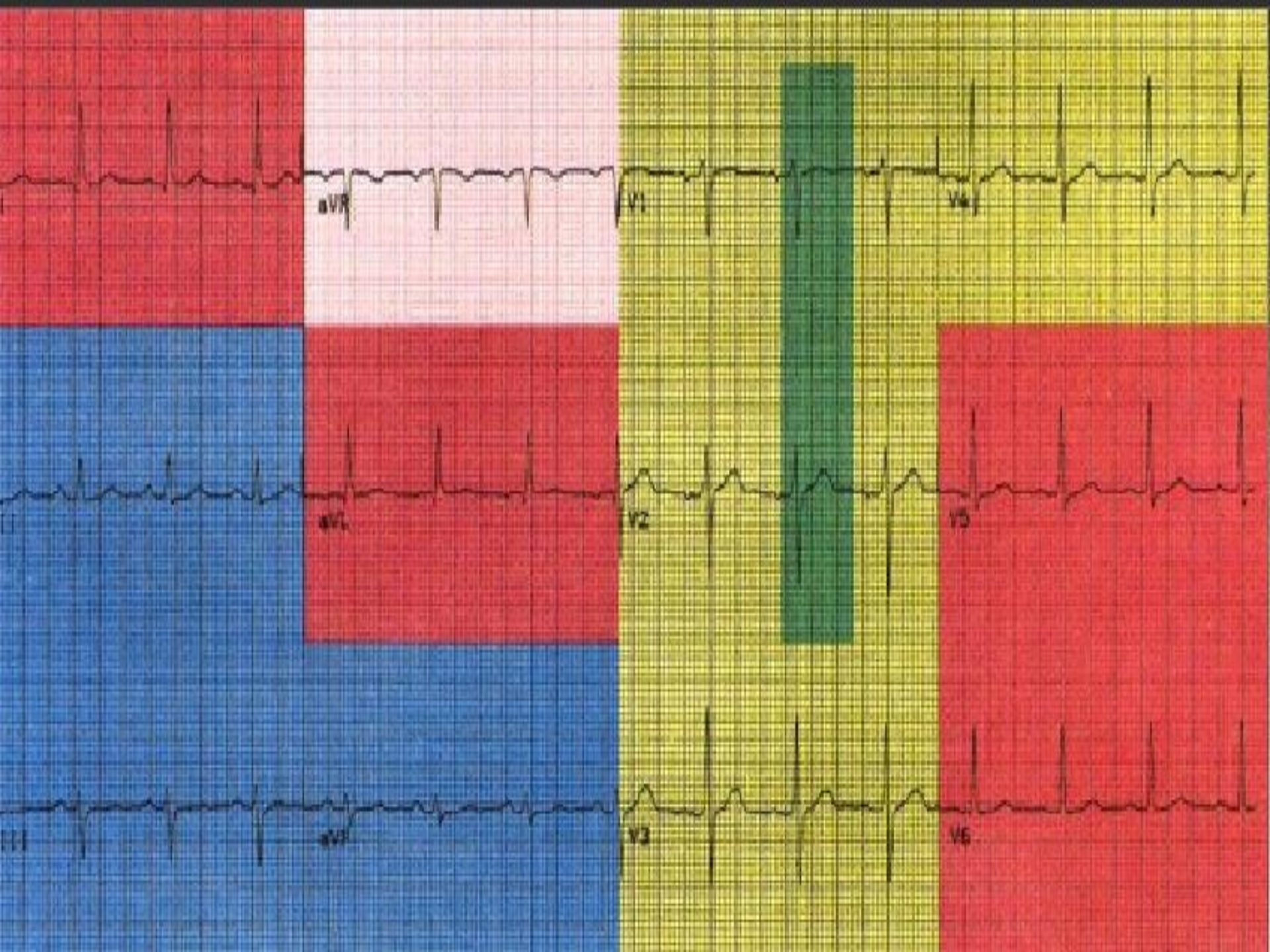


IMA LATERAL



IMA POSTERIOR

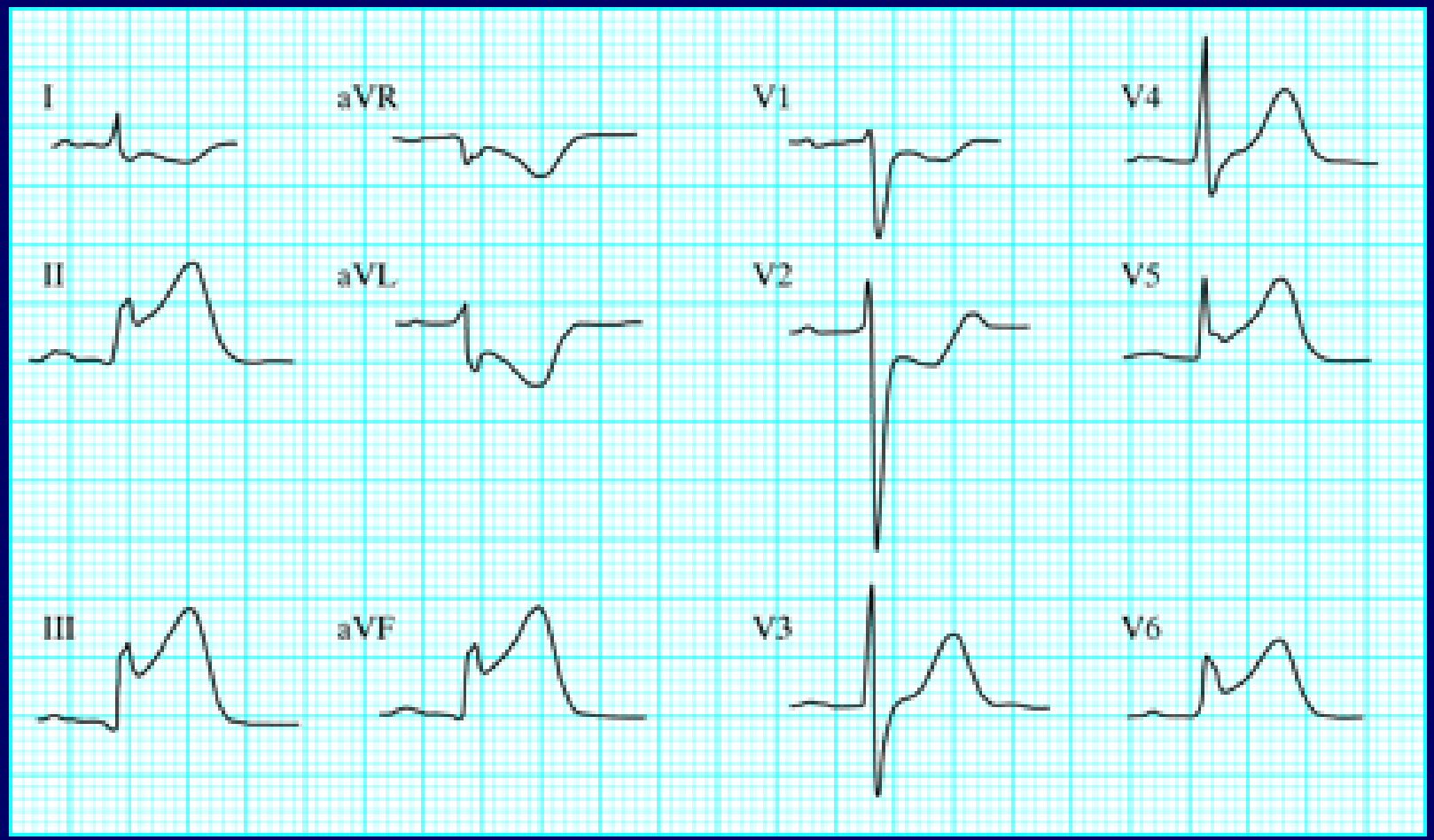



aVR
V1
V4
aVL
V2
V5
aVF
V3
V6

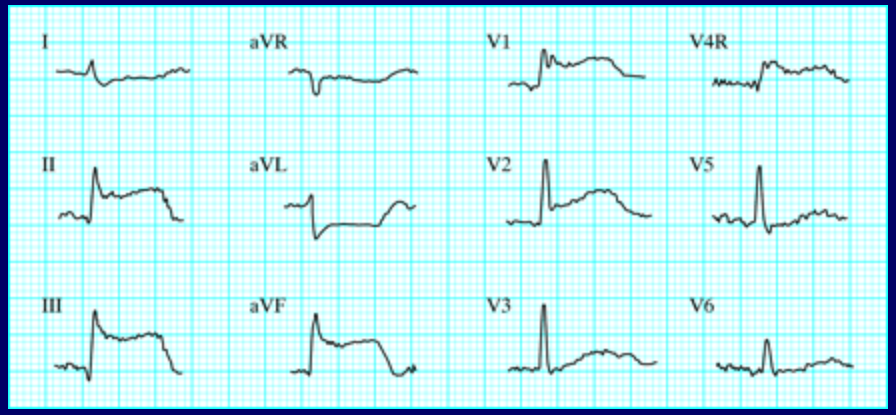
Reciprocal ST segment depression

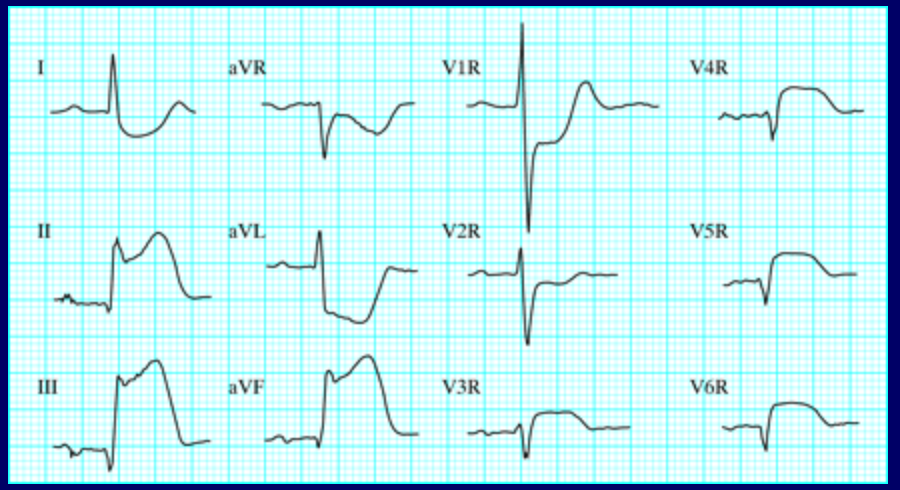
- Reciprocal changes are seen in up to 70% of inferior and 30% of anterior infarctions.
- Typically, the depressed ST segments tend to be horizontal or downsloping

An inferior -lateral myocardial infarction with reciprocal changes in leads I, aVL, V1, and V2



Acute inferior myocardial infarction with right ventricular involvement





Non-ST elevation MI (NSTEMI)

- There may be no ECG changes
- Patients may have ST depression, T wave flattening, or T wave inversion
- Serial ECGs are necessary to diagnose dynamic changes
- In patients with symptoms suggestive of MI and no evidence of ST elevation on ECG, the diagnosis of acute coronary syndrome is made
- This encompasses unstable angina pectoris and NSTEMI. The distinction between these two entities is made on the presence or absence of elevated biomarkers of MI

Cardiac troponins I and T

- Troponin levels (either I or T) are significantly more sensitive and specific for myocardial damage than CK
- Troponin becomes detectable in serum between 4 hours and 6 hours after onset of an acute MI, peaks and then falls to lower levels, and remains elevated at these low levels for 5–7 days

Imaging : Echocardiography in STEMI.

- In most cases is not recommended because it delays reperfusion therapy
- Is helpful in diagnosing complications of MI, such as VSD, papillary muscle rupture or free wall rupture, and tamponade
- Echocardiography may be considered if the diagnosis of MI remains in doubt (eg, equivocal history, uninterpretable ECG)

Imaging : Echocardiography in NSTEMI

- In NSTEMI that is diagnosed on elevated plasma levels of cardiac biomarkers, nuclear scintigraphy or echocardiography may help determine the region of the heart affected by the MI, but these are not standard diagnostic tools

The goals of treatment

- stabilization of the patient
- salvage of as much myocardium as possible

Pre-Hospital Management

- Continuous cardiac monitoring
- Aspirin, 162–325 mg - immediately
- Oxygen
- Sublingual nitroglycerin

Emergency Department Therapy

- 12-lead ECG performed immediately
- If aspirin has not been given, 162–325 mg of aspirin should be administered immediately
- Continuous cardiac ECG monitoring
- Intravenous access (two separate intravenous lines)
- Sublingual nitroglycerin
- Intravenous morphine if patients have active chest pain
- Oxygen saturations monitoring noninvasively rather than by arterial blood gas measurement.

Oral β-blockers

- has been shown to improve outcomes and limit the size of infarction
- Intravenous β-blockers could be considered when there is hypertension or tachyarrhythmia
- However, they should be avoided in patients with signs of heart failure, in those with contraindications to β-blockers, and in those at high risk for cardiogenic shock (age > 70 years, heart rate > 110/min or < 60/min, systolic blood pressure < 120 mm Hg, or prolonged time since the onset of symptoms

ANTICOAGULANTS

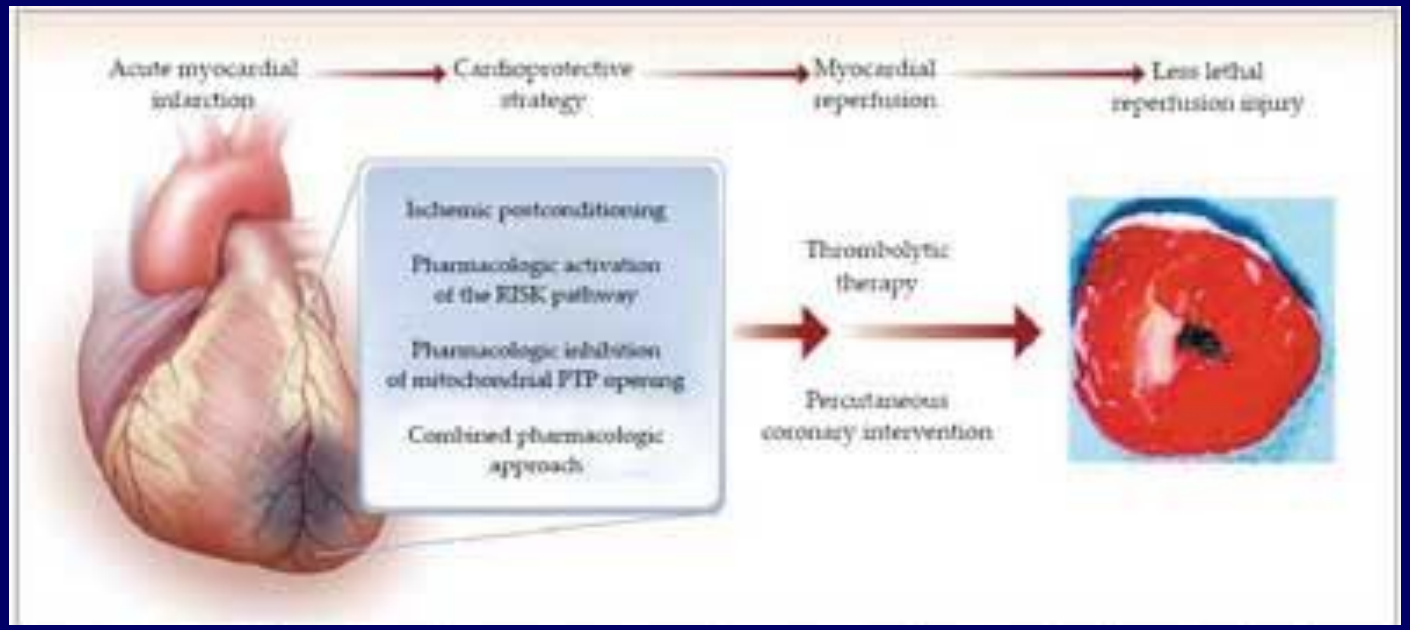
- Heparin should be administered to all patients with acute MI, unless a contraindication exists
- Unfractionated heparin is preferred in most institutions for invasive therapy (primary PCI) because it has a short half-life, it can be turned off rapidly
- In contrast, LMWHs have a long half-life, and there is no bedside test of their anticoagulant efficiency

Aspirin and Heparin

- Regardless of the fibrinolytic agent used, all patients should receive aspirin and heparin (either UFH or LMWH) to counteract the procoagulant effect of the fibrinolytic agent
- The standard dose of UFH is usually a bolus of 5000 units, followed by a 1000-unit-per-hour infusion until the partial thromboplastin time can be used to titrate a dose between 1.5 and 2 times the normal range

Reperfusion Therapy

- Fibrinolysis- thrombolytic treatment
- Percutaneous coronary intervention



Reperfusion Therapy: general indication

- STEMI within the first 12 hours after symptom onset should be considered for urgent reperfusion of the infarct-related artery, but the earlier therapy is begun, the greater the benefit
- particularly if chest pain is ongoing or heart failure or shock has developed, but the benefit of reperfusion therapies after more than 12 hours is less well established
- improve patency of the infarct-related artery, reduce infarct size, and lower mortality rates

Indications for thrombolytic treatment

- ST elevation >1 mm in two contiguous limb leads or >2 mm in two contiguous chest leads
- Posterior myocardial infarction
- Left bundle branch block

Contraindications for Fibrinolysis

Absolute contraindications

- **Any prior ICH**
- **Known structural cerebral vascular lesion (eg, AVM)**
- **Known malignant intracranial neoplasm (primary or metastatic)**
- **Ischemic stroke within previous 3 months**
- **Suspected aortic dissection**
- **Active bleeding or bleeding diathesis (excluding menses)**
- **Significant closed head or facial trauma within 3 months**
- **Severe uncontrolled hypertension (SBP > 180 mm Hg and/or DBP > 110 mm Hg)**

Streptokinase

- Streptokinase is derived from streptococcal bacteria and activates plasminogen indirectly, forming an activator complex with a slightly longer half-life than streptokinase alone (23 minutes versus 18 minutes after a bolus)
- Because it activates both circulating plasminogen and plasminogen bound to fibrin, both local and systemic effects occur; that is, circulating fibrinogen degrades substantially (fibrinogenolysis as well as fibrinolysis occurs).

Urokinase

- Urokinase is a direct activator of plasminogen. It has a shorter half-life than streptokinase (14 ± 6 minutes) and is not antigenic
- Its effects on both circulating and bound-to-fibrin plasminogen are similar to those from streptokinase
- It is therefore difficult to understand why intravenous doses of urokinase (2.0 million units as bolus or 3 million over 90 minutes) seem to induce coronary artery patency more rapidly than does streptokinase
- There is substantial synergism between urokinase and t-PA.

Tissue Plasminogen Activator

- Despite the short half-life, lytic activity persisted for many hours after clearance of the activator
- Although t-PAs are considered “fibrin-specific,” no activator is totally fibrin-specific, and fibrin specificity is lost at higher doses
- At clinical doses, however, less fibrinogen degradation took place than with nonspecific activators
- Tissue plasminogen activator clearly opened coronary arteries more rapidly than nonspecific activators and this is likely why its use improved mortality rates
- Bleeding was not less and there was a slight increase in the number of intracranial bleeds, which was in part due to the need for dosage adjustment for lighter-weight patients

Reteplase

- **Reteplase, a mutant form of t-PA, lacks several of the structural areas of the parent molecule (the finger domain, kringle 1, and the epidermal growth factor domain)**
- **It is less fibrin-specific (causes more systemic degradation of fibrinogen) than the parent molecule and has a longer half-life**
- **Accordingly, it is used as a double bolus of 10 units initially followed by a second bolus 30 minutes later, and this requires no adjustment for patient weight. Although not shown to be superior to t-PA, many clinicians have elected to use reteplase because of the convenience of the double bolus administration.**

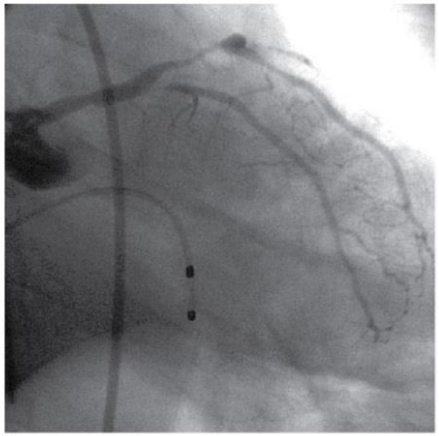
Tenecteplase

- Tenecteplase is also a mutant form of t-PA
- It has substitutions in the kringle 1 and protease domains to increase its half-life, increase its fibrin specificity, and reduce its sensitivity to its native inhibitor (PAI-1)
- Although not shown to be superior to t-PA, tenecteplase is generally being used in preference to the parent molecule because of the convenience of a single bolus dose.

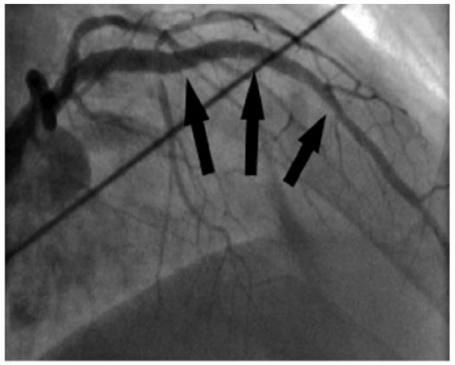
Adverse Effects of Fibrinolytic Therapy

- Intracranial bleeding is by far the most dangerous bleeding complication because it is often fatal
- For most plasminogen activators, the incidence of intracranial hemorrhage is less than 1%, but it may be as high as 2–3% in elderly patients
- Risk factors for intracranial bleeding include a history of cerebrovascular disease, hypertension, and age
- These factors must be taken into account when determining whether a thrombolytic agent has an appropriate benefit-to-risk relationship
- Changes in mental status require an immediate evaluation—clinical and computed tomography or magnetic resonance imaging
- If bleeding is strongly suspected, heparin should be discontinued or neutralized with protamine

Primary PCI for acute MI: 6-12 hours



A



B

Complications of Myocardial Infarction

- Cardiogenic shock
- Congestive heart failure (CHF)
- Ischemic mitral regurgitation
- Ventricular septal defect (VSD)
- Free wall rupture
- Recurrent ischemia
- Pericarditis
- Conduction disturbances Arrhythmias
- Mural thrombus
- Aneurysm or pseudoaneurysm of the left ventricle
- Right ventricular infarction

Cardiogenic Shock

- Cardiogenic shock is characterized by peripheral hypoperfusion and hypotension refractory to volume repletion. This occurs secondary to inadequate cardiac output resulting from severe left ventricular dysfunction.
- Goals of therapy for cardiogenic shock include hemodynamic stabilization to ensure adequate oxygenation of perfused tissue and prompt assessment for reversible causes of the cardiogenic shock. If reversible causes are not found, immediate reperfusion, especially with PCI, is indicated

Congestive Heart Failure

- Echocardiography has been the technique of choice in evaluating such patients from a perspective of both valvular and myocardial function
- Nitroglycerin is often the best agent to use for ischemia in patients with CHF. In some instances, the hemodynamic profile provided by nitroprusside may be desirable
- However, nitroprusside may exacerbate ischemia by inducing a coronary steal phenomenon; in this setting, nitroprusside would be a second-line therapy
-

Acute Mitral Valve Regurgitation

- The development of acute severe mitral valve regurgitation occurs in approximately 1% of patients with acute MI and contributes to 5% of deaths.