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Complications of acute myocardial infarction

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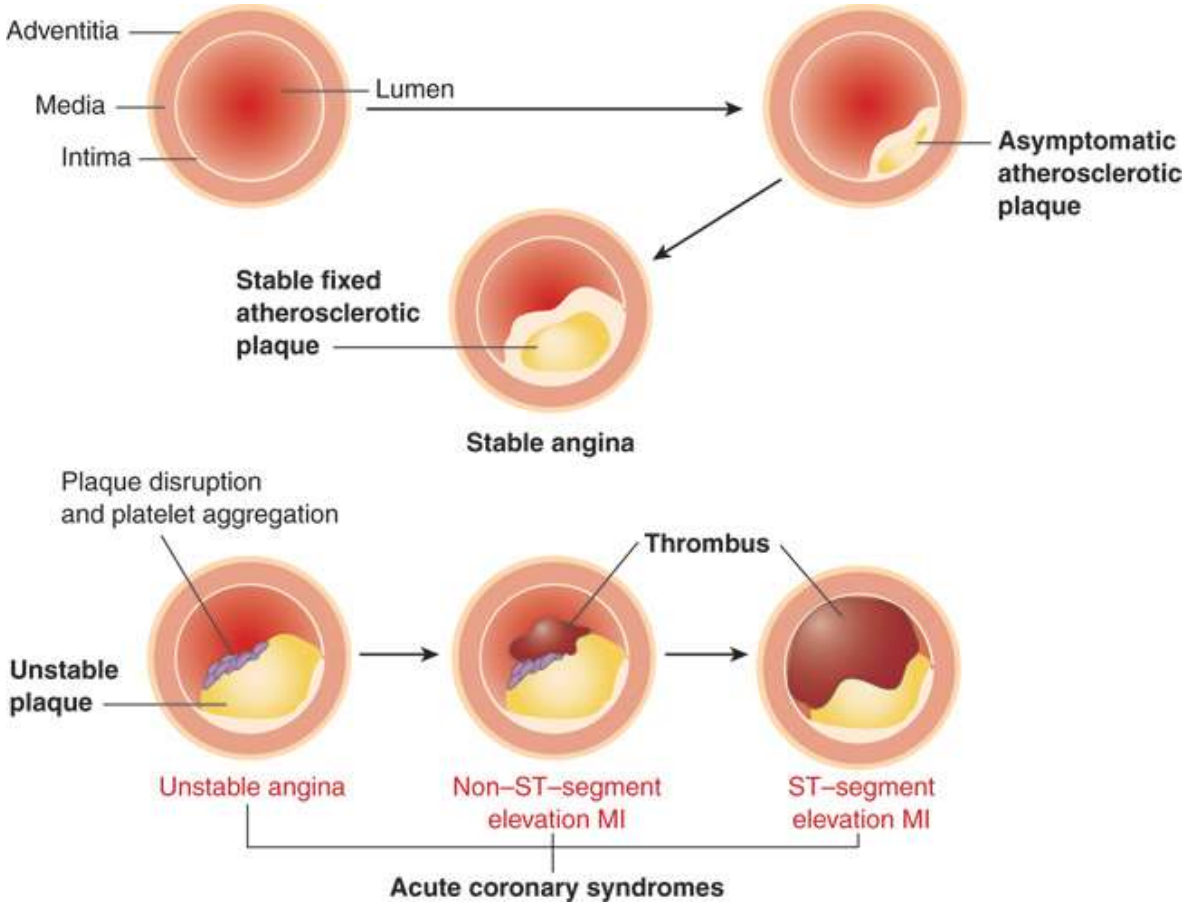
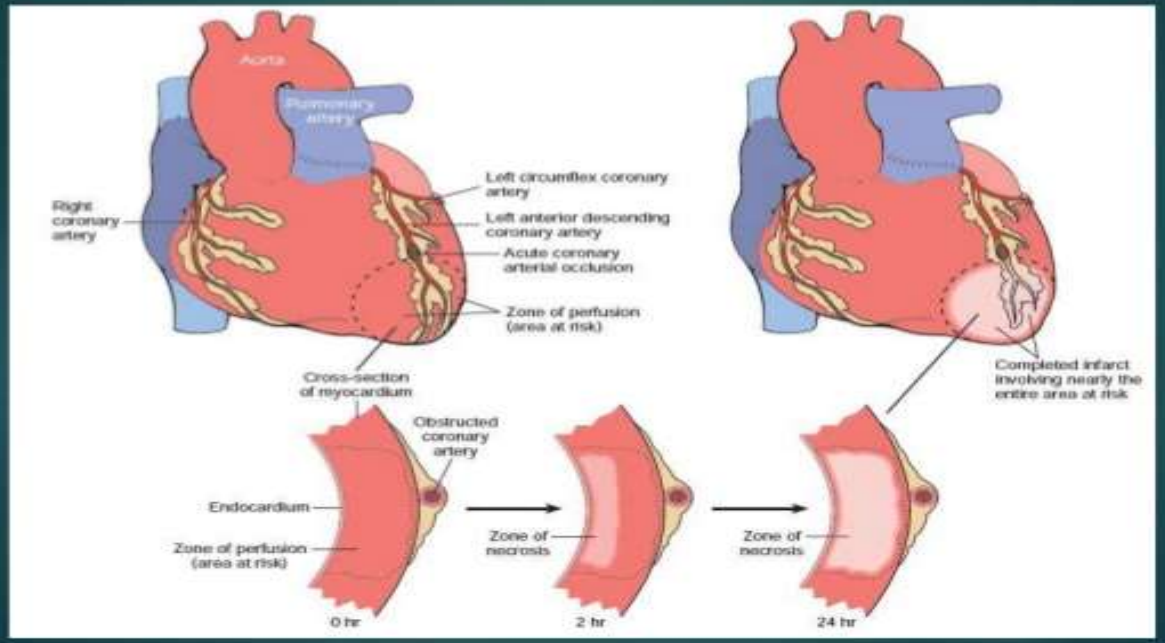
The term myocardial infarction should be used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischaemia.

Stable Angina

Unstable Angina | NSTEMI | STEMI



Pathophysiology



After a coronary occlusion, there is a **lack in perfusion and in oxygen supply** that cause **functional, morphological, and biochemical alterations**.

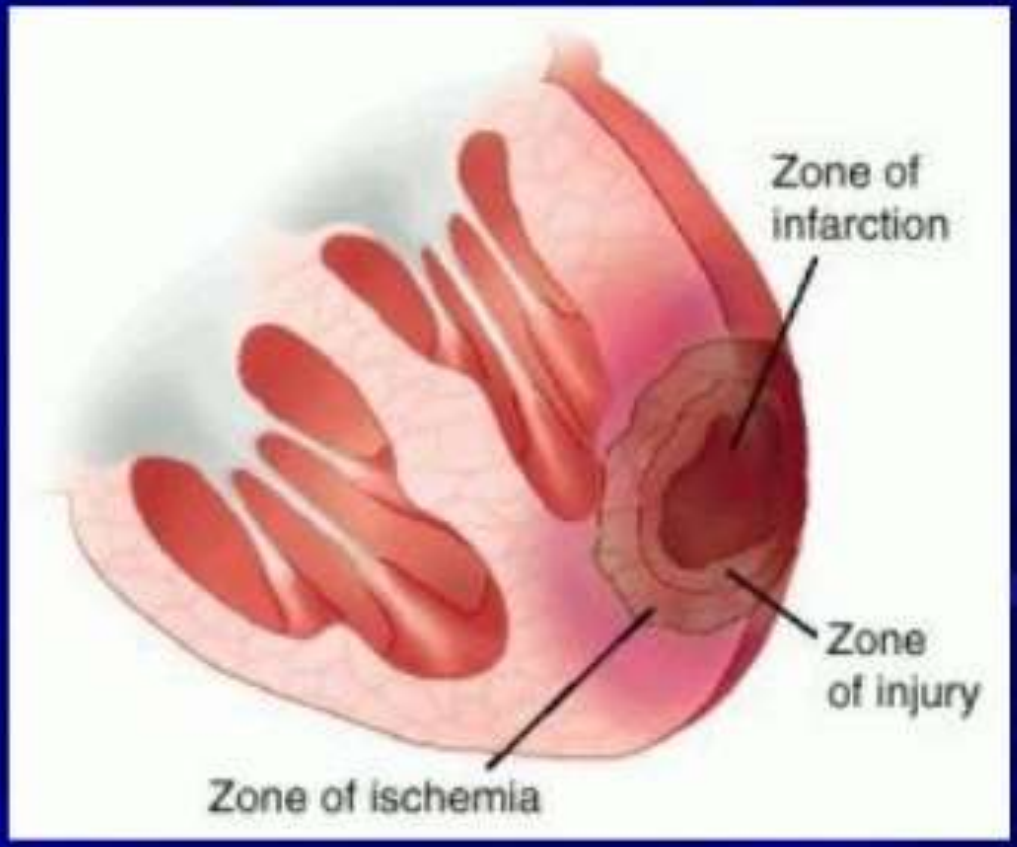
In the first 30 minutes from the occlusion, reversible changes happen

After half an hour, ischaemic necrosis begins, and the irreversible damage occurs.

Complete necrosis of myocardial cells requires at least 2–4 hours

The principal mechanism is coagulative necrosis, with neutrophil infiltration, oedema, and loss of myofibrils. After 6–12 hours, loss of vitality is complete. In one week, macrophagic phagocytosis and collagen disruption begin, and tissue becomes weaker; that is the most dangerous step in which heart ruptures are more frequent.

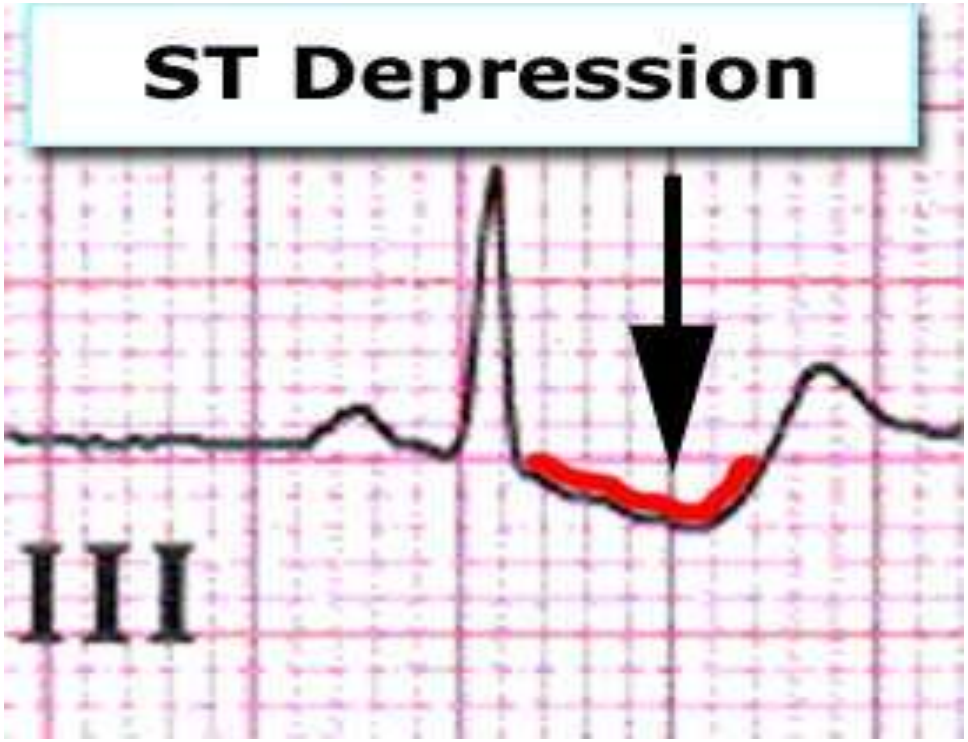
Myocardial Infarction



The Three I's

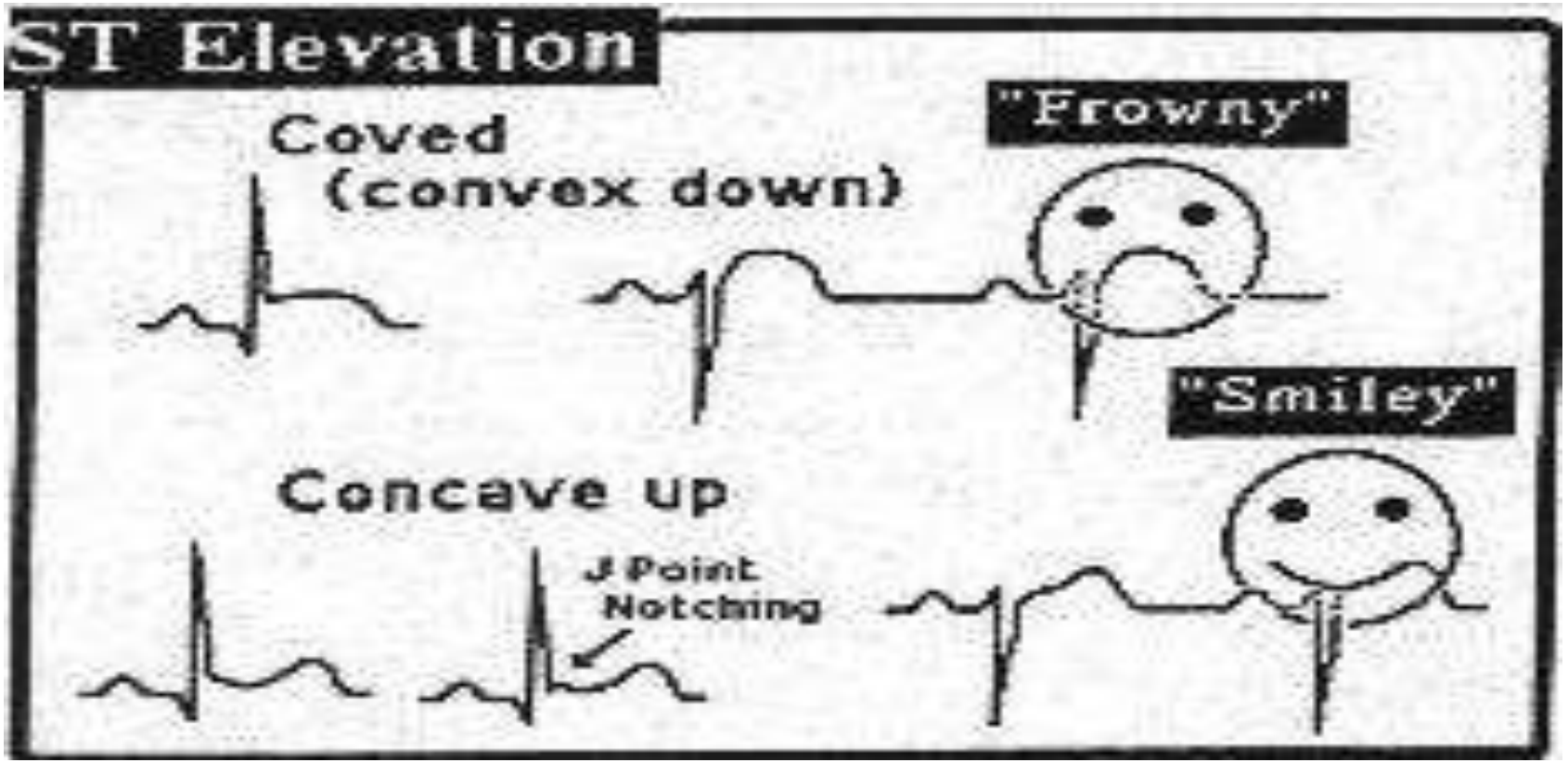
Ischemia= ST depression or T-wave inversion

Represents lack of oxygen to myocardial tissue



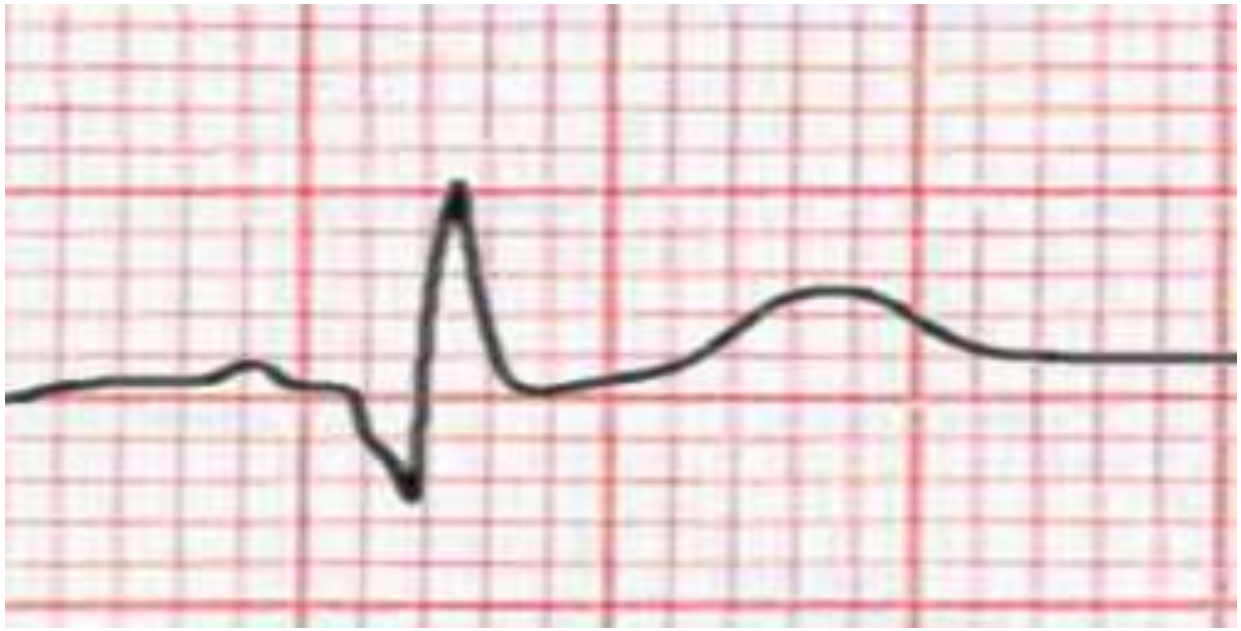
The Three I's

Injury = ST elevation -- represents prolonged ischemia; significant when > 1 mm above the baseline of the segment in two or more leads



The Three I's

Infarct = Q wave – represented by first negative deflection after P wave; must be pathological to indicate MI



• Complications

(1)ischaemic complications, which include infarct extension, recurrent infarction, and post infarction angina;

(2) arrhythmic complications, in terms of atrial or ventricular arrhythmias, and sinus or atrioventricular node dysfunction;

(3) embolic complications towards central nervous system or peripheral embolization;

(4)inflammatory disturbances, such as pericarditis;

(5)mechanical complications, as myocardial rupture, mitral valve dysfunction, ventricular aneurysms and cardiogenic shock up to heart failure.

Ischaemic complications:

-**Post - infarction angina**

- Angina with ischemic ECG changes and/or CHF

Recurrent and persistent anginal symptoms may occur in the early post-infarction period at >24 hours but <2 weeks after a documented MI

and have been associated with an unfavourable prognosis.

- infarct extension

- recurrent infarction

Electrical Complications

arrhythmic complications

In acute M.I , Arrythmias are mainly due to reentry caused by **inhomogenicity of ischaemic myocardium** .

Reperfusion Arrythmias (after Reperfusion by thrombolysis and angioplasty) are due to washout of the accumulated ions and metabolites .

Traditionally dysrhythmias are classified as early or late in relation to the cardiac event.

Early dysrhythmias, occurring within the first 24-48 hours are due to myocardial ischaemia

Late dysrhythmias occur after 48 hours and are a reflection of the size of ventricular damage.

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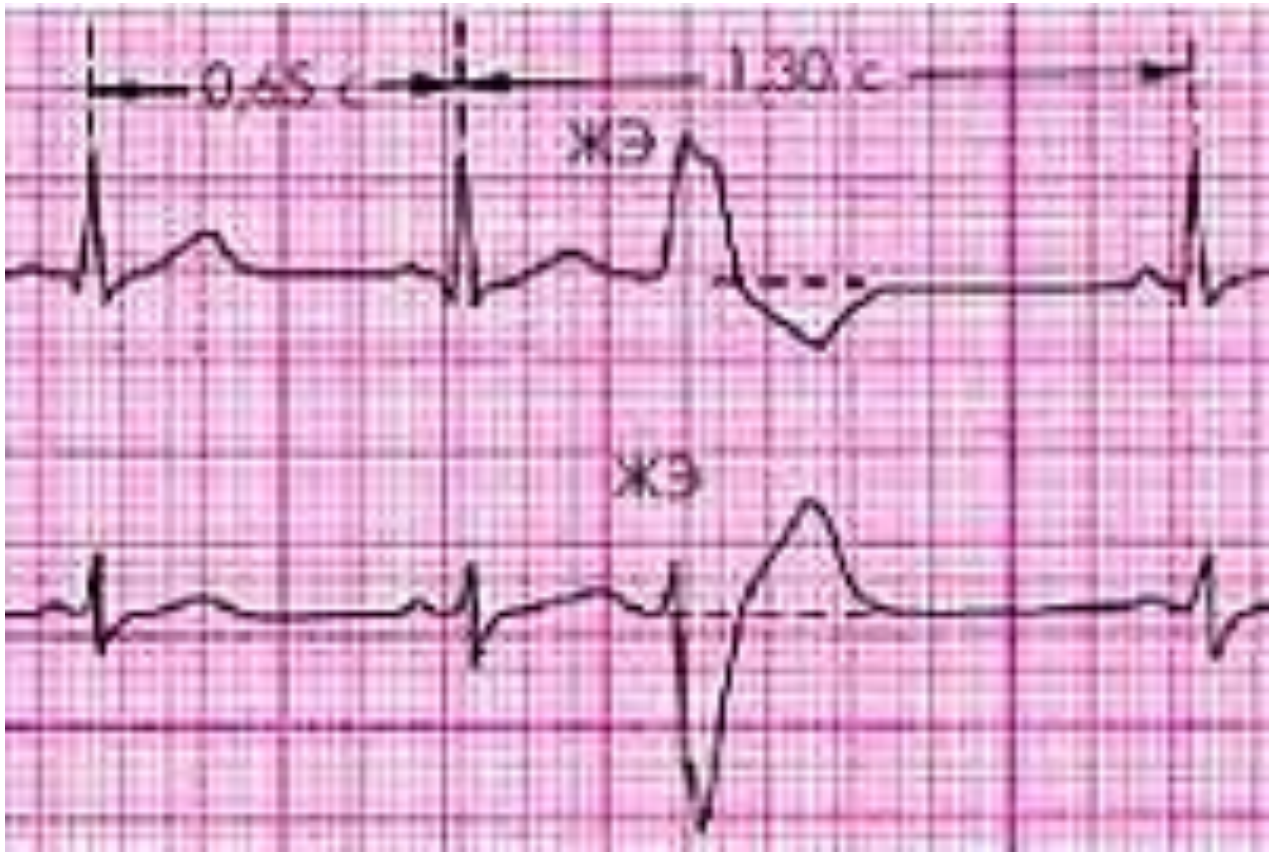
Ventricular Arrythmias

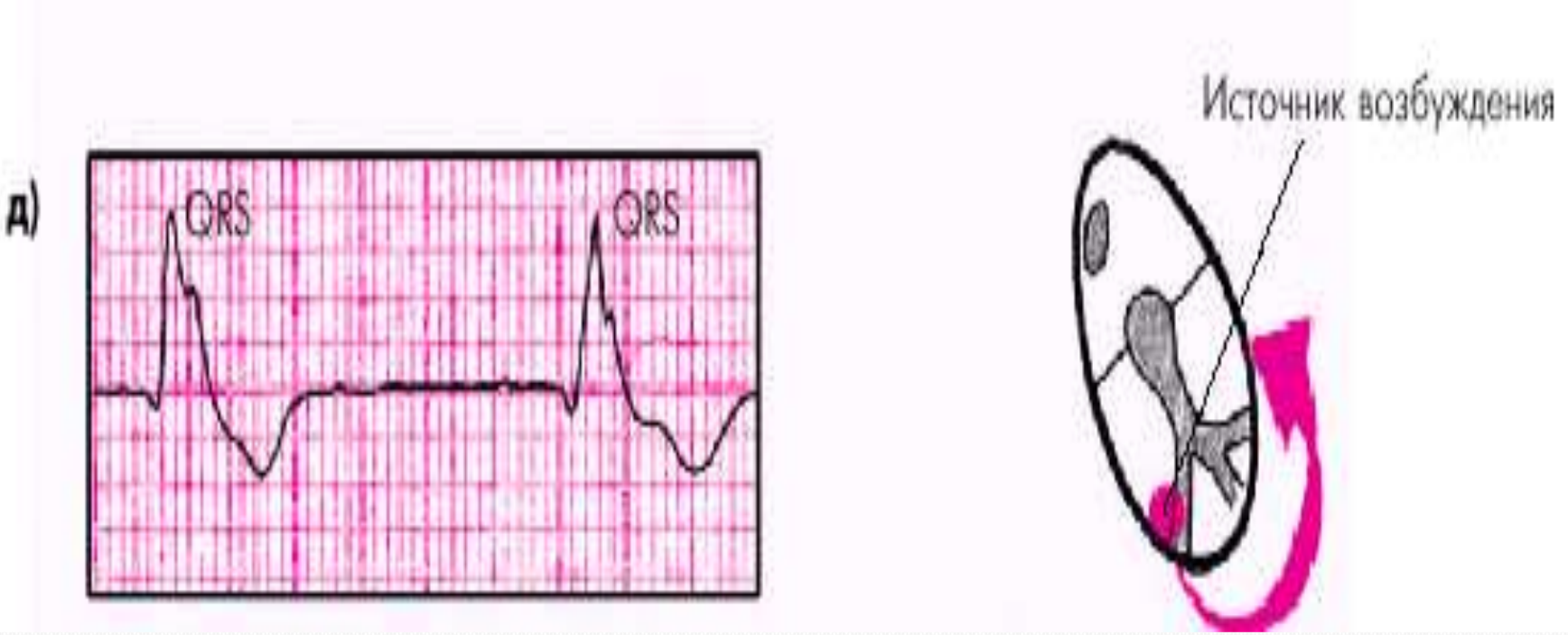
1. **Ventricular premature beats (extrasystoles)**

Can be origin of primary ventricular Fibrillation .



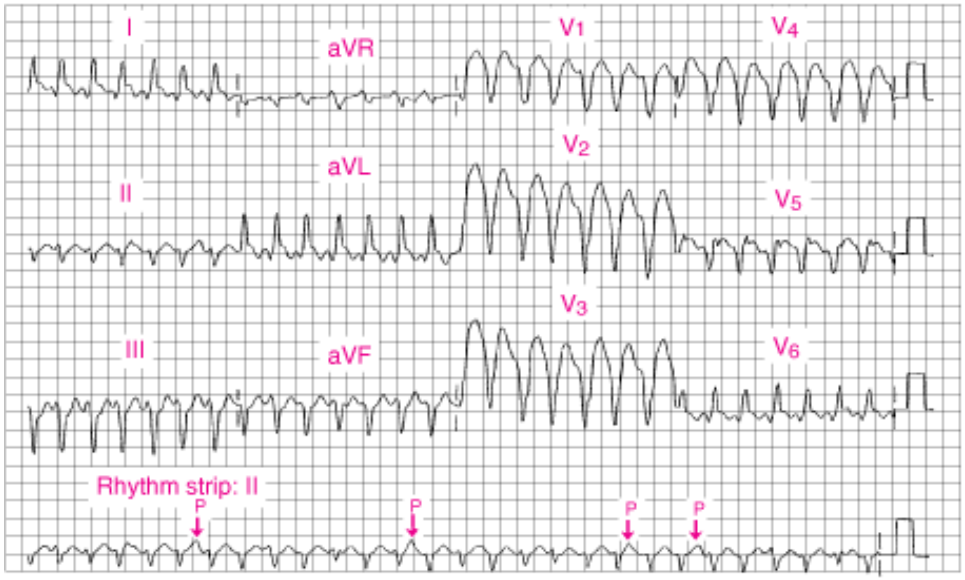
2. **Accelerated idioventricular rhythm (rhythm from His-Purkinje System):**

occurs in 20% of patients with STEMI , most often after successful reperfusion .



3. Ventricular Tachycardia :

Ventricular Tachycardia with haemodynamic instability is the sign of progressive left ventricular heart felure



Brief episodes of ventricular tachycardia may not cause any symptoms in some people.
Or the patient can have:

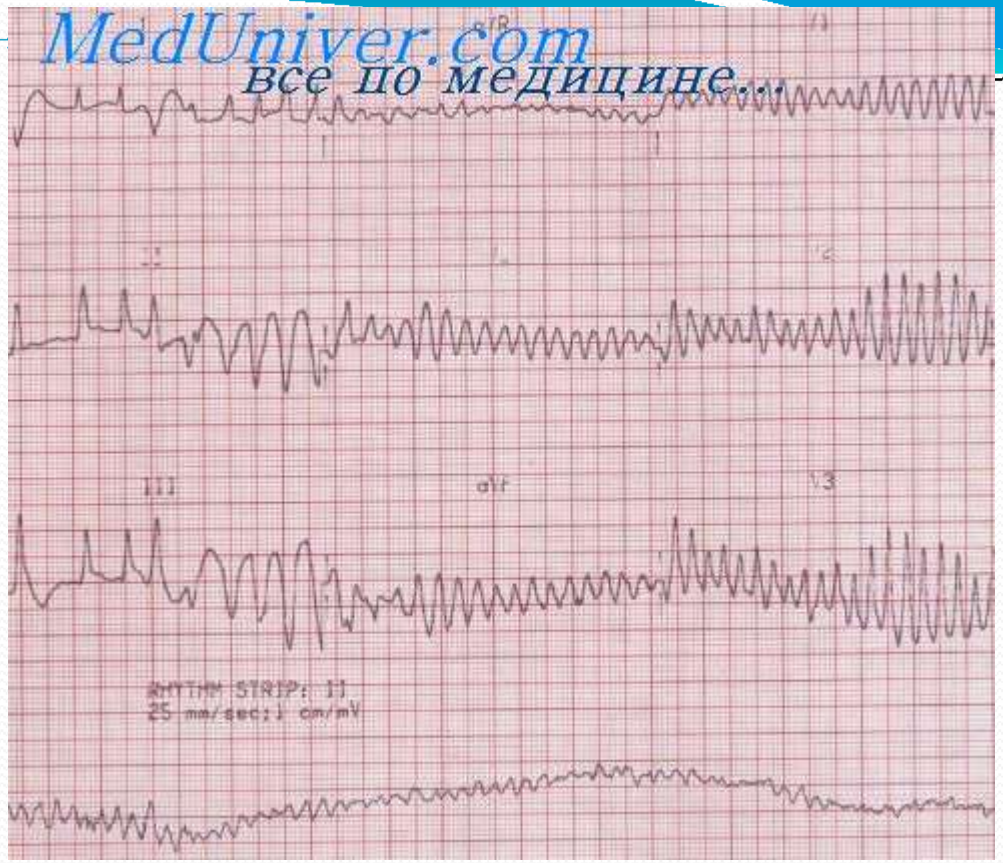
-Dizziness

-Shortness of breath

- Palpitation

- Chest pain (angina)

4. Ventricular fibrillation :



- Fainting or losing consciousness are the most common symptoms of VF, earlier symptoms include:

- chest pain
- dizziness
- nausea
- rapid, fluttering heartbeat
- shortness of breath

Supraventricular Arrythmias

1. **Sinus Tachycardia :**

very common in patients with acute M.I esp. Anterior M.I , it may be caused by other post-infarction causes as persistant pain , LV dysfunction , fever , hypotension , hypovolaemia , pericarditis , pulmonary embolism , etc

2. **Premature Atrial beats :**

may be due to atrial ischaemia or pericarditis .

3. **Paroxysmal SVT :**

usually transient.

4. **Atrial Flutter and Fibrillation** :

both are usually transient and may occur with patients with LV dysfunction , extension of ischaemia or pulmonary embolism .

Bradyarrythmias

1 **Sinus Bradycardia :**

it's common especially in **Inferior and posterior Infarctions** . If heart rate is below 40-50 .

2. First Degree A-V block :

it may be caused by the use of Beta blockers or Calcium channel blockers

3. **Second degree AV block :**

Type I: Does not require treatment except if ventricular rate is below 50 bpm , then atropine is indicated.

Type II: has a potential to progress to complete heart block so temporary pacing is indicated and set at about 60 bpm.

4. **Complete AV block :**

- If associated with Inferior infarctions , it's usually transient and resolves within 72 hrs .
- If associated with anterior infarction , it usually occurs suddenly , after 12-24 hrs , they have unstable escape rhythm and a wide complexes at a rate ≤ 40 bpm . They are associated with an extensive septal necrosis and may need permenant pacing .

5. **Intraventricular block :**

Right and Left bundle branch block

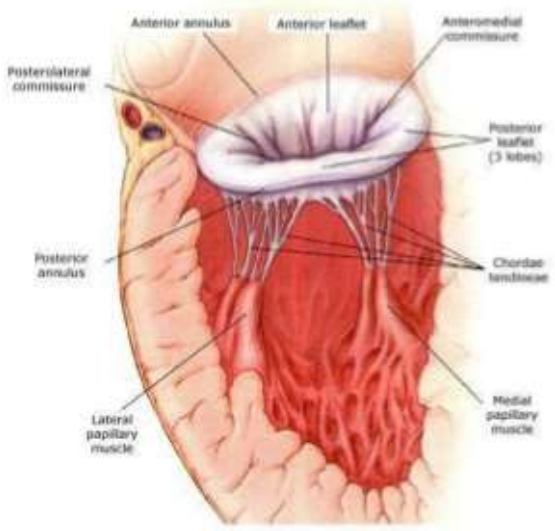
Sudden death

Fatality from MI remains formidably high, with 50 per cent of patients who die after an acute coronary occlusion doing so within the first hour after the onset of symptoms (Rawles, 1997). Death is commonly due to the dysrhythmia, ventricular fibrillation.

The risk of sudden death is highest at the onset of symptoms and declines progressively over a number of hours (Resuscitation Council UK, 2000). This is why individuals need early access to defibrillation in the event of cardiac arrest (Department of Health, 2000).

Mechanical complications

Anatomy: Mitral valve



Pic source: www.heart-valve-surgery.com

- The mitral valve connects the left atrium (LA) and the left ventricle (LV).
- The mitral valve opens during diastole to allow the blood flow from the LA to the LV.
- During ventricular systole, the mitral valve closes and prevents backflow to the LA.

The mitral apparatus is composed of

1. the left atrial wall
2. the annulus
3. the leaflets
4. the chordae tendineae
5. the papillary muscles
6. the left ventricular wall

1. **Acute Mitral Regurgetation**

Papillary muscle dysfunction:

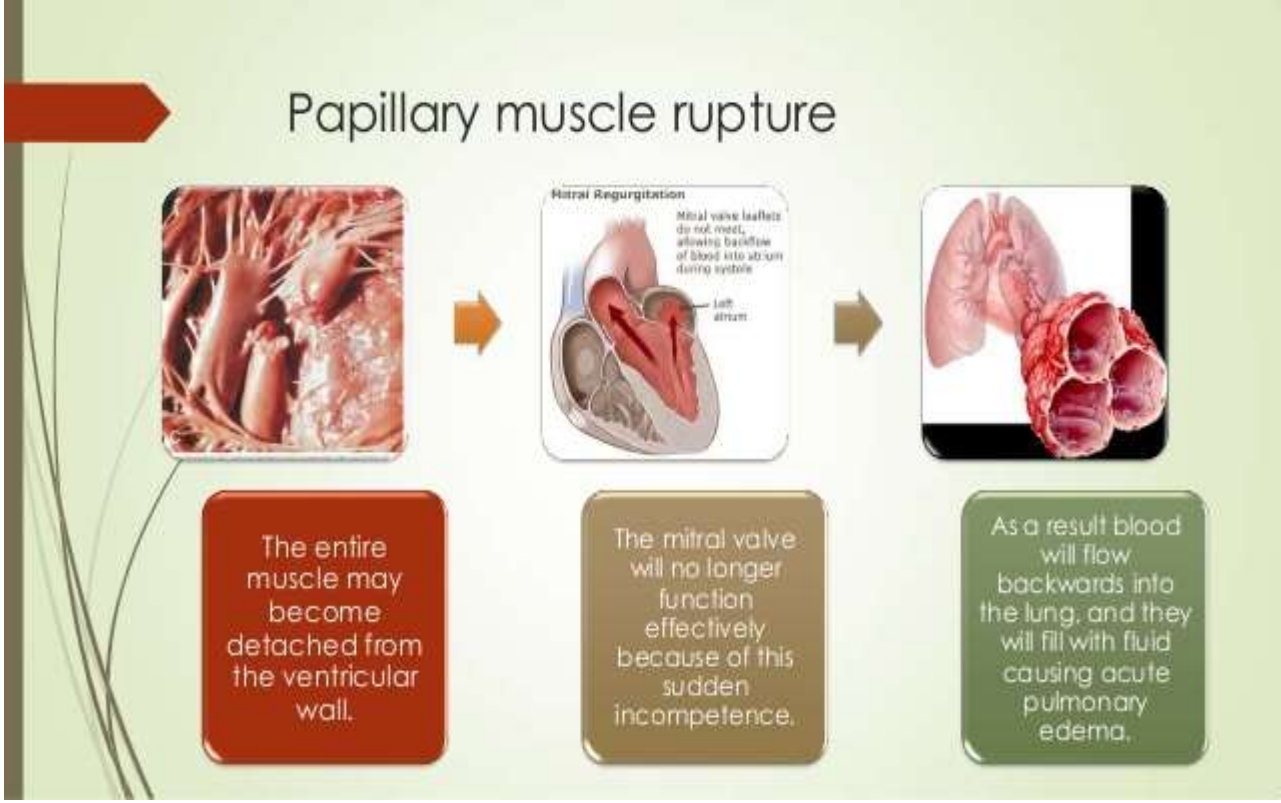
- Caused by posteromedial papillary muscle dysfunction , It's usually transient during ischaemia .

- It usually presents with an apical systolic murmur , confirmed by echocardiography .

Papillary muscle Rupture

-It occurs with **Inferior Infarction** 6-12 times more than Anterior infarctions .

-**PMR** may be complete, with a massive mitral regurgitation and rapid onset of symptoms up to hemodynamic collapse and death, or partial, with a moderate to severe mitral regurgitation.



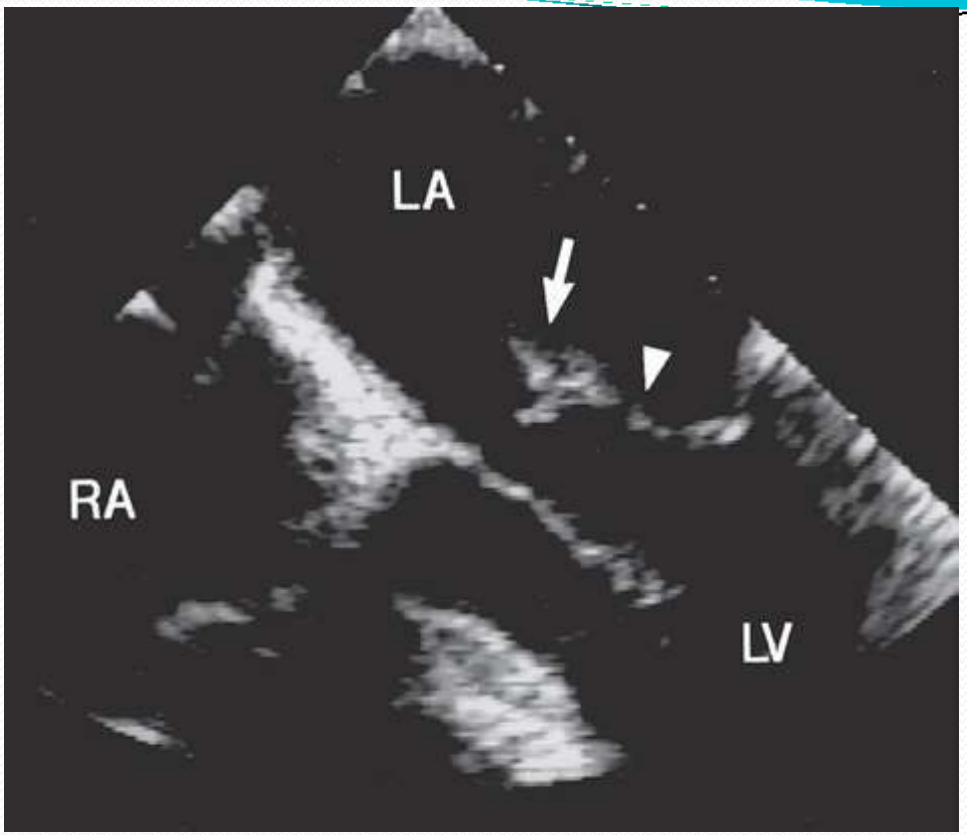
Clinical presentation

-usually presents dyspnoea, hypotension, acute pulmonary oedema cardiogenic shock.

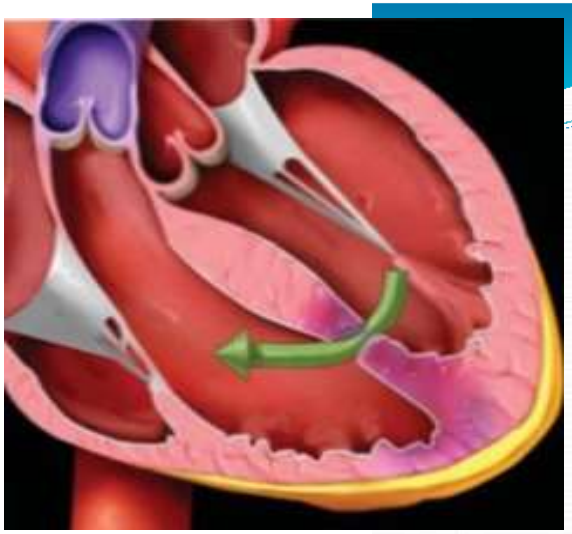
At the physical examination, a soft murmur without thrill may be present, even if the absence of new heart murmur does not exclude the diagnosis.

The gold standard in diagnosis is Doppler ultrasound (is a noninvasive test that can be used to estimate the blood flow through blood vessels), transthoracic and transesophageal echocardiography, with the evidence of a tear in papillary tissue and the flail of mitral leaflet leading to severe mitral regurgitation

Echocardiography



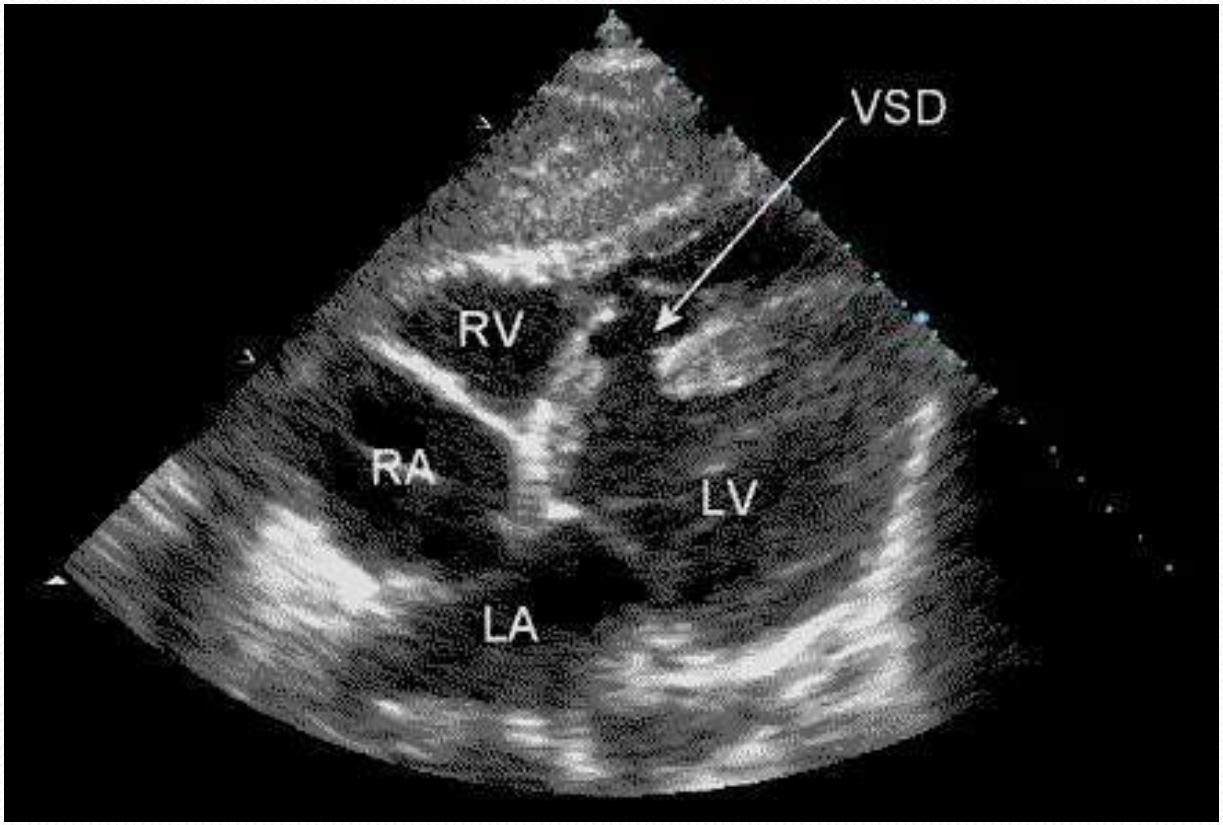
Papillary muscle rupture complicating acute inferior myocardial infarction; magnified four-chamber view. The ruptured head of the posteromedial papillary muscle (*arrow) prolapses freely into the* left atrium; the posterior mitral valve leaflet (*arrowhead)* is flail.



2. Ventricular Septal Rupture

- It occurs equally with Inferior and Anterior infarctions.
- Rupture with Anterior Infarctions tend to be apical , while it's basal and has a worse prognosis with inferior infarctions
- Signs and symptoms may include recurrent chest pain and dyspnoea, hypotension and biventricular failure (often predominantly right-sided failure), up to cardiogenic shock is possible. At physical examination, a harsh and loud pansystolic murmur at the left lower sternal border is present in over 90% of cases. A palpable thrill can be detected in up to 50% of patients

Echocardiography



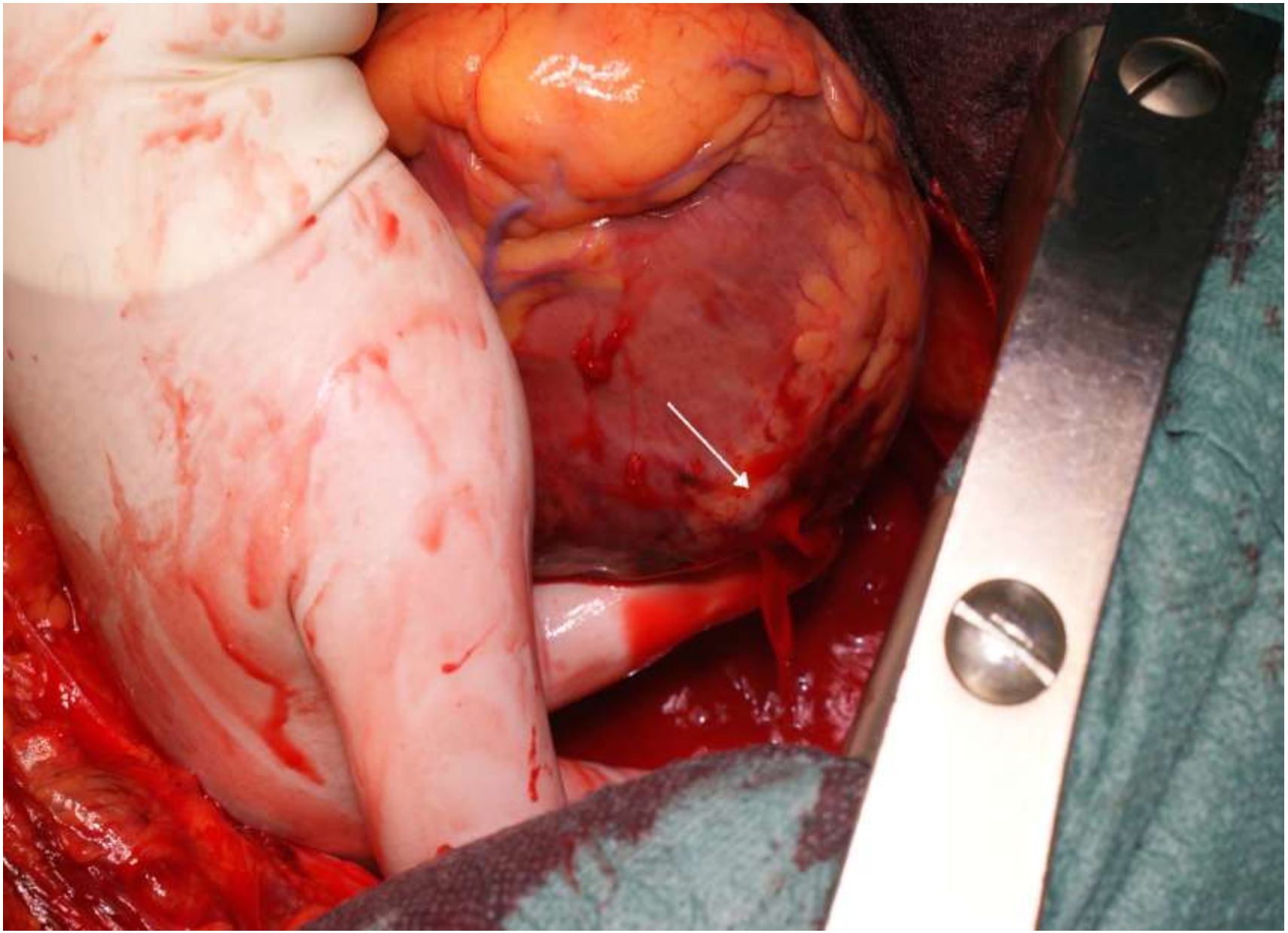
Transthoracic sub-costal 4-chamber echocardiographic findings showing a **large ventricular septal rupture (VSR)**

3. Free wall Rupture :

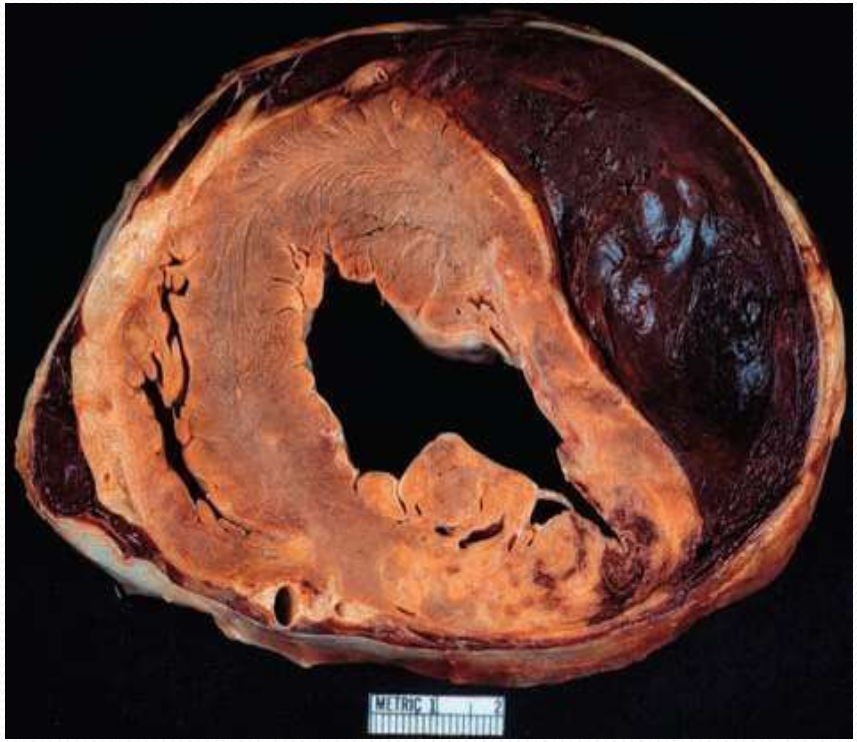
- Occurs within 2 weeks , and most common cause of death after cardiogenic shock and arrythmias .
- Early use of thrombolytic therapy appears to reduce the incidence of cardiac rupture , while late use appears to increase the incidence .
- Cardiac rupture manifests as chest pain, hypotension and dyspnoea. Death is rapid due to haemopericardium and the resultant cardiac tamponade leading to a pulseless electrical activity cardiac arrest.

- A severe jugular venous destention and cyanosis may be present. Death occurs within a few minutes.

- Few cases can be salvaged by immediate pericardiocentesis , emergency Thoracotomy and surgical repair .



Cardiac free wall rupture post myocardial
infarction.



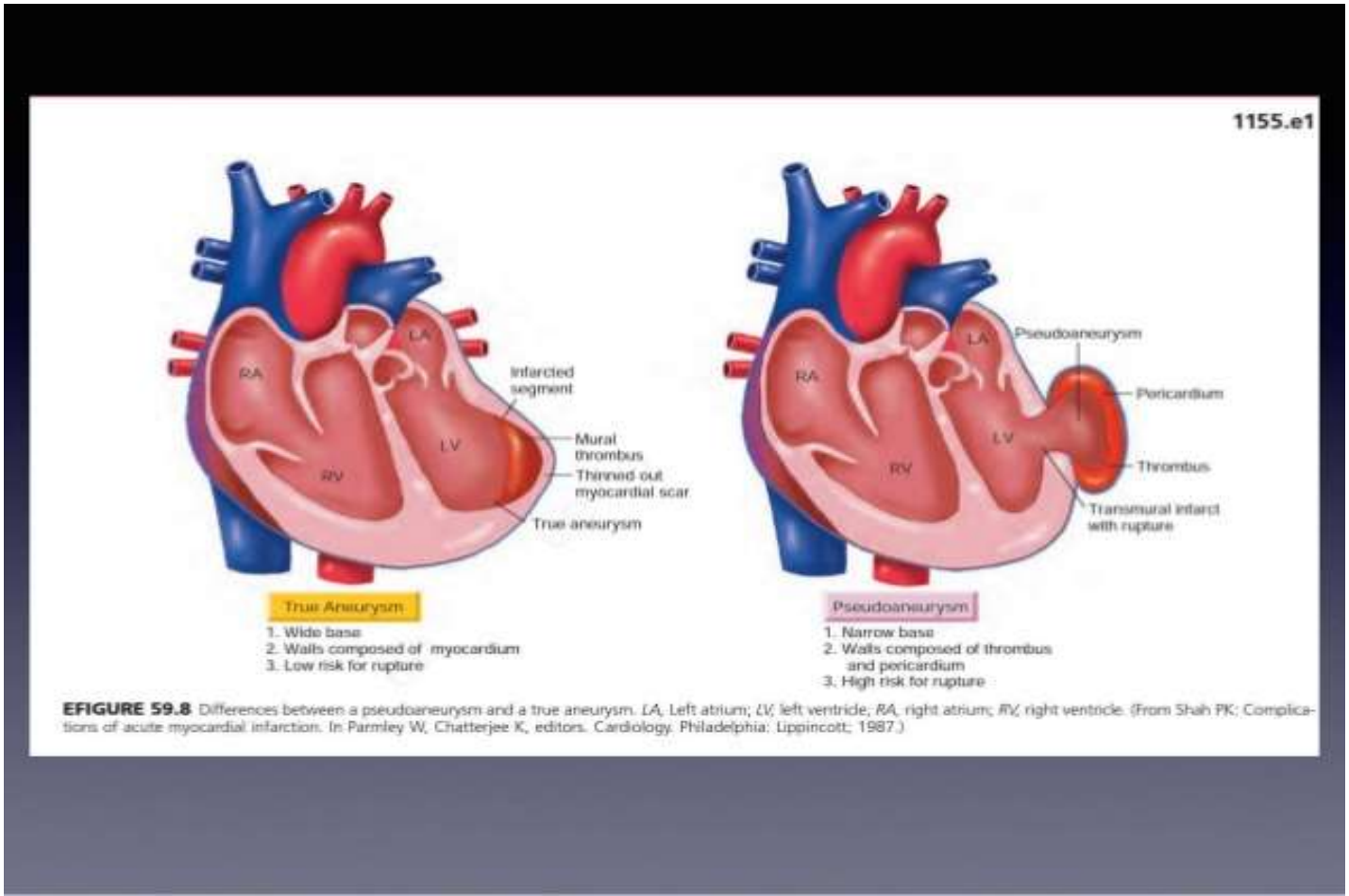
Pericardial tamponade from left ventricular free
wall rupture and hemopericardium.

4. Ventricular Aneurysm :

An **aneurysm** is a section of defective wall that bulges outward.

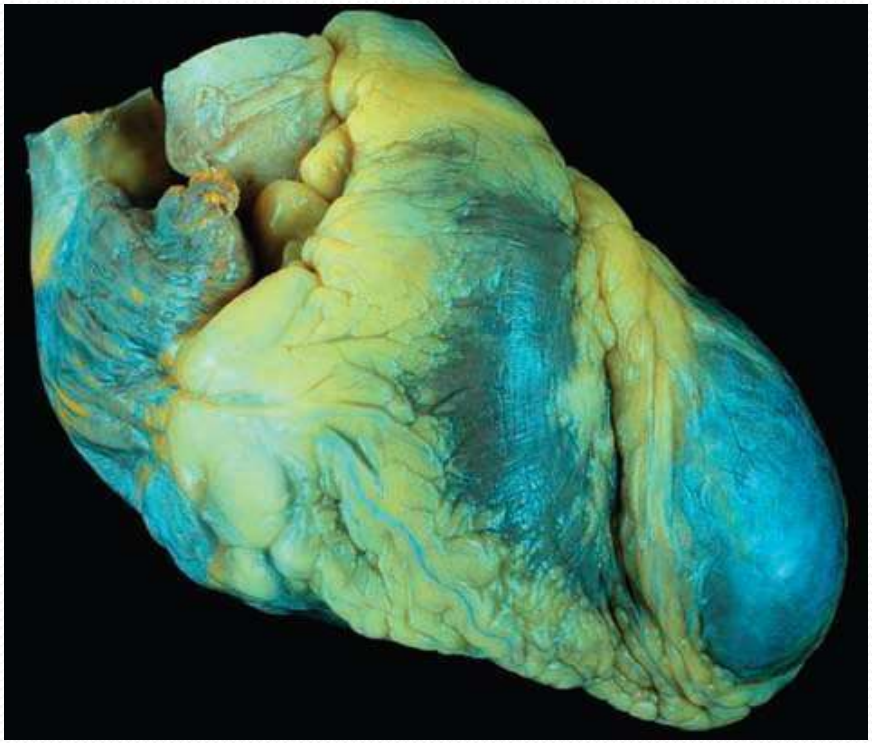
True aneurysm is the result of the gradual thinning and the expansion of the scarred left ventricular wall after transmural infarction.

Pseudoaneurysm - does not contain all the three layers of the myocardium and is frequently lined by pericardium and mural thrombus



4. **Ventricular Aneurysm :**

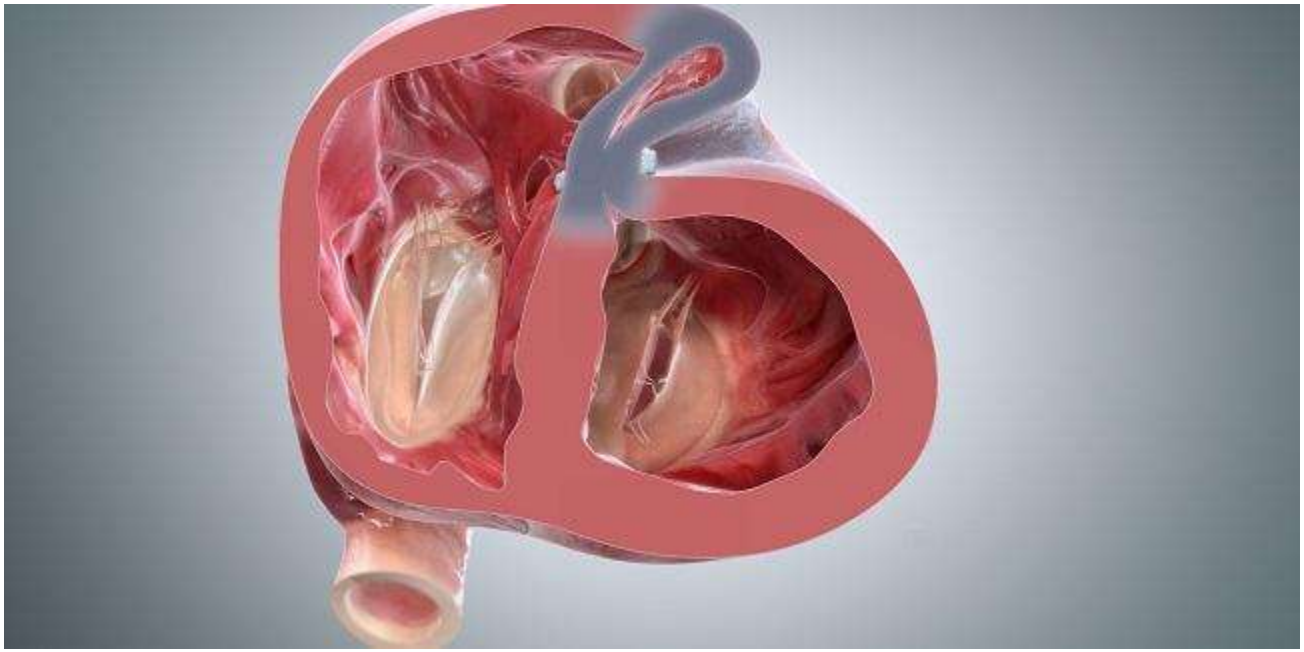
Diff. between True and Pseudo-aneurysm .



True Aneurysm

Pseudo-Aneurysm

The 85% of true aneurysm is located anterolaterally, near the apex of the heart.
Clinical presentation often involves:
angina (in more than 60% of patients, three-vessels coronary disease is present), **dyspnoea**, and **symptoms of congestive heart failure**, **arrhythmias**
Atrial and ventricular arrhythmias may occur in the scar tissue, producing palpitations, syncope, and even sudden death.
A **mural thrombus** is often found (50%)
abnormal precordial pulsation in the 3rd left intercostal space



Identifying aneurysm

- ECG

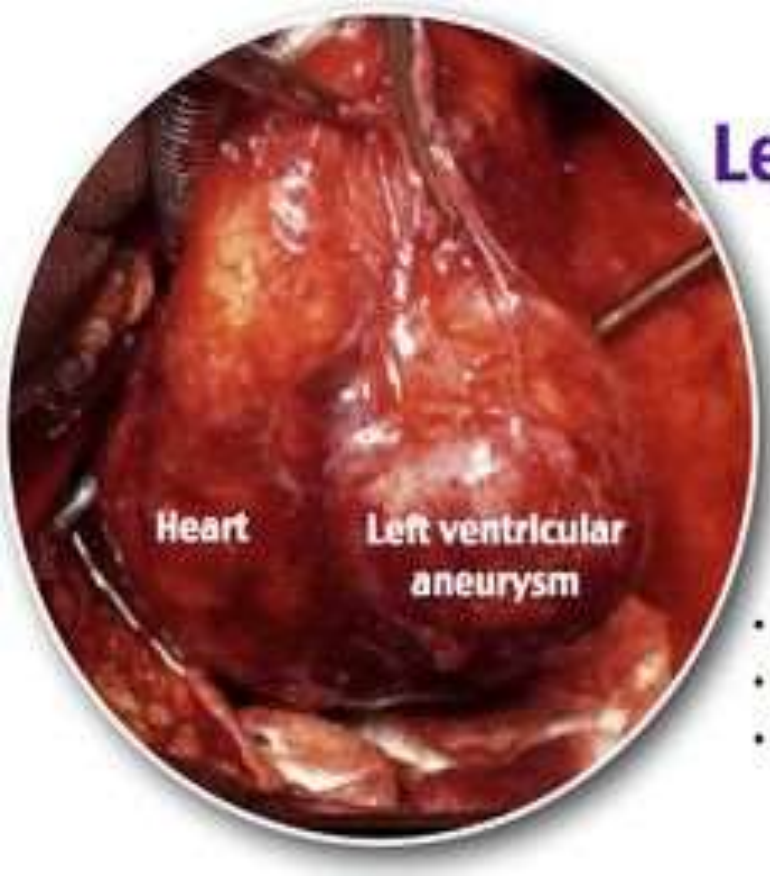
-Echocardiography

- Angiography and **left ventriculography** is the gold standard, estimating the size of aneurysm and evaluating cardiac function, coronary status.

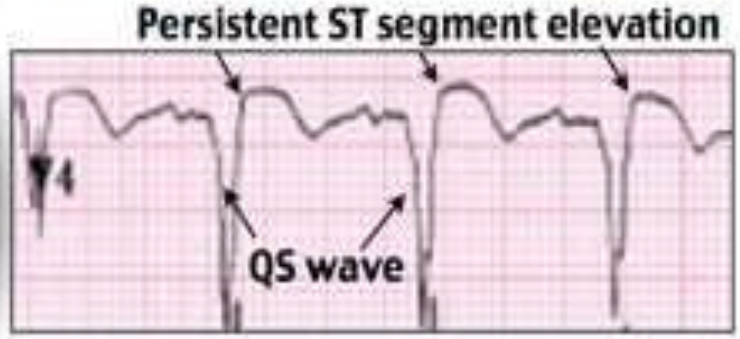
- Tomographic three-dimensional echocardiography

- Magnetic resonance imaging are the most reliable means of evaluating left ventricular volume.

- **Positron emission tomography (PET)** can be helpful in an early phase to differentiate true aneurysm from hibernating myocardium with reversible dysfunction.

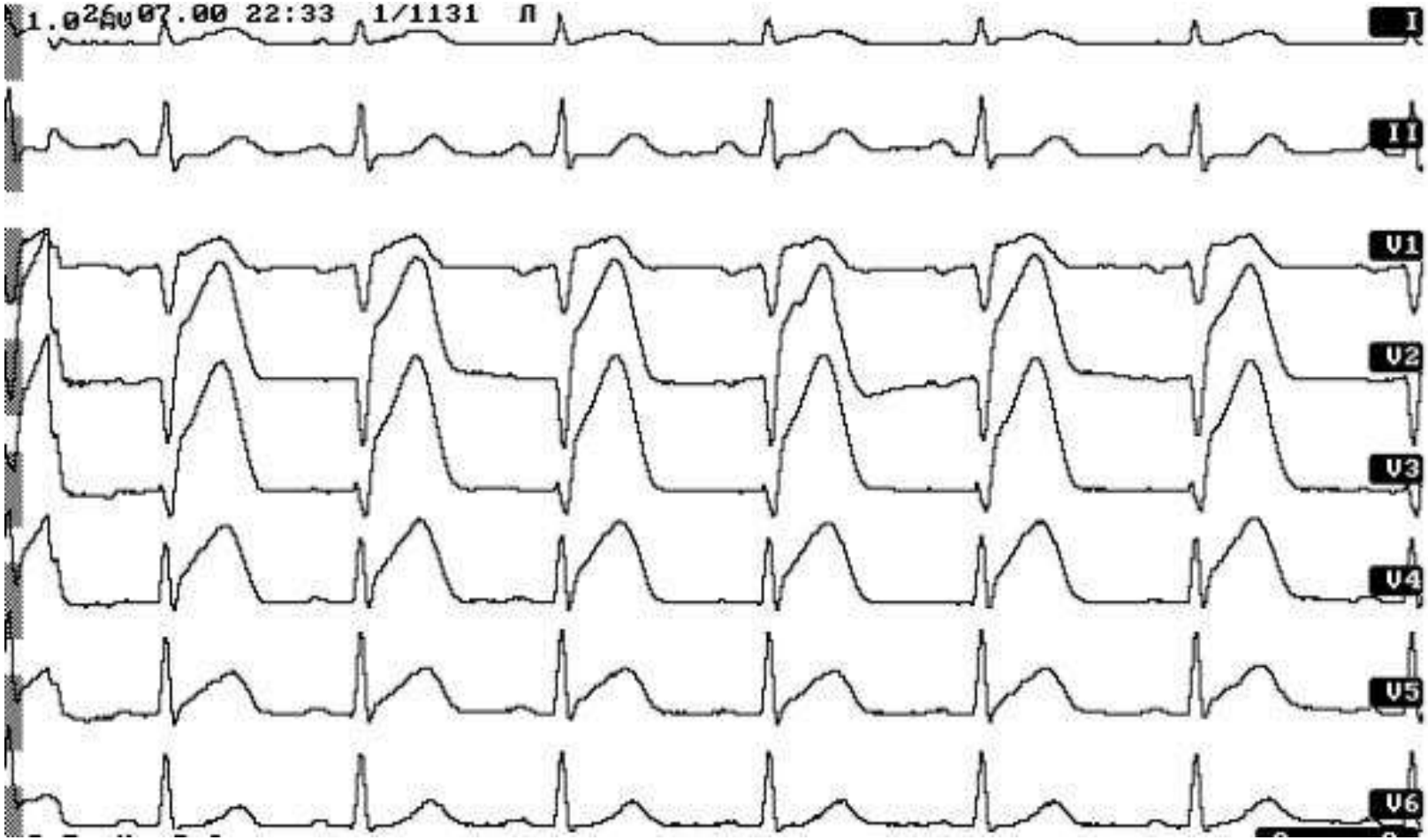


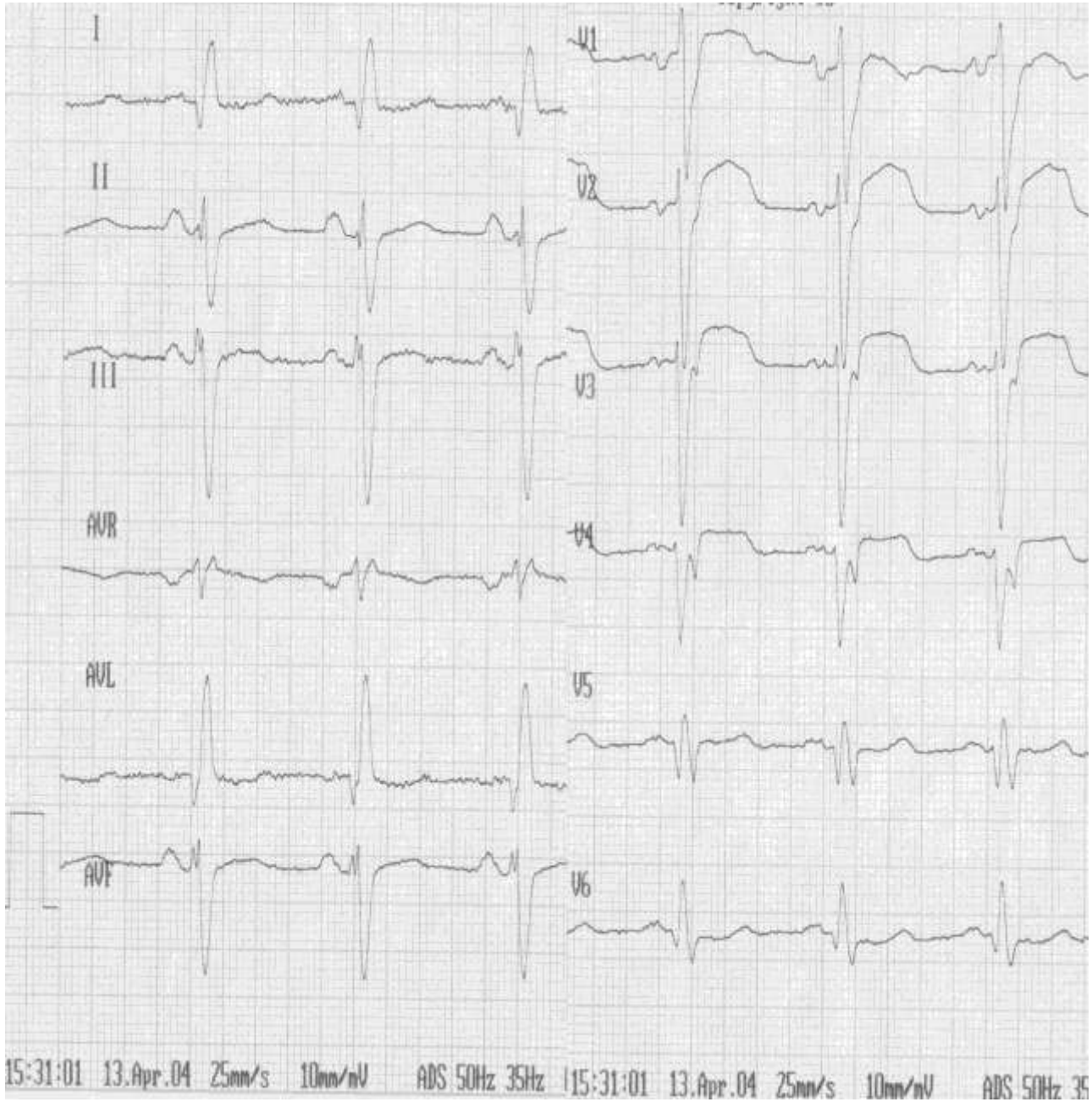
Left Ventricular Aneurysm



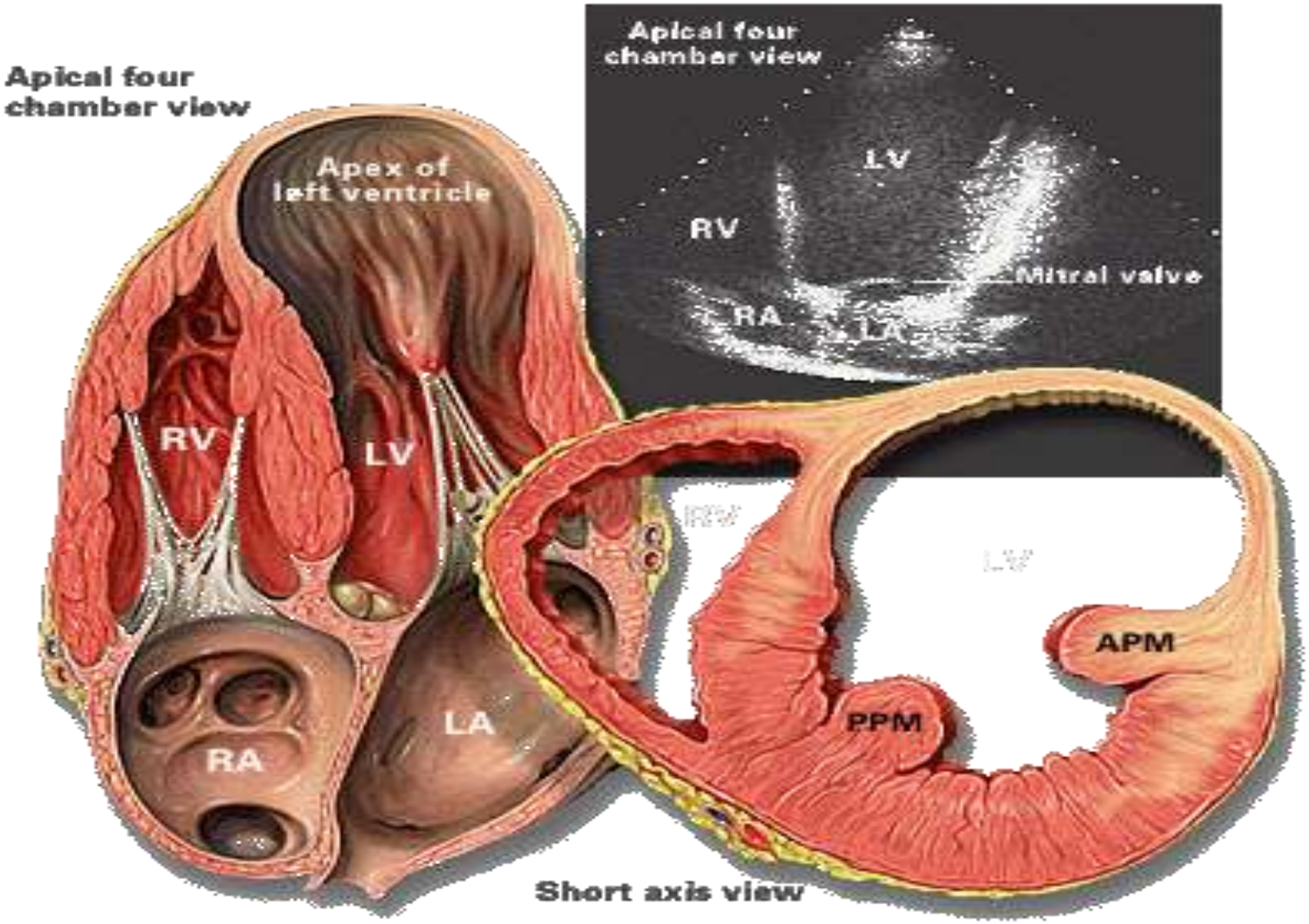
- **Persistent ST elevation** present > 2 weeks **following an acute MI**
- Most commonly present in **precordial leads**
- Associated with **Q or QS waves**

Unchanged ECG, without evolution

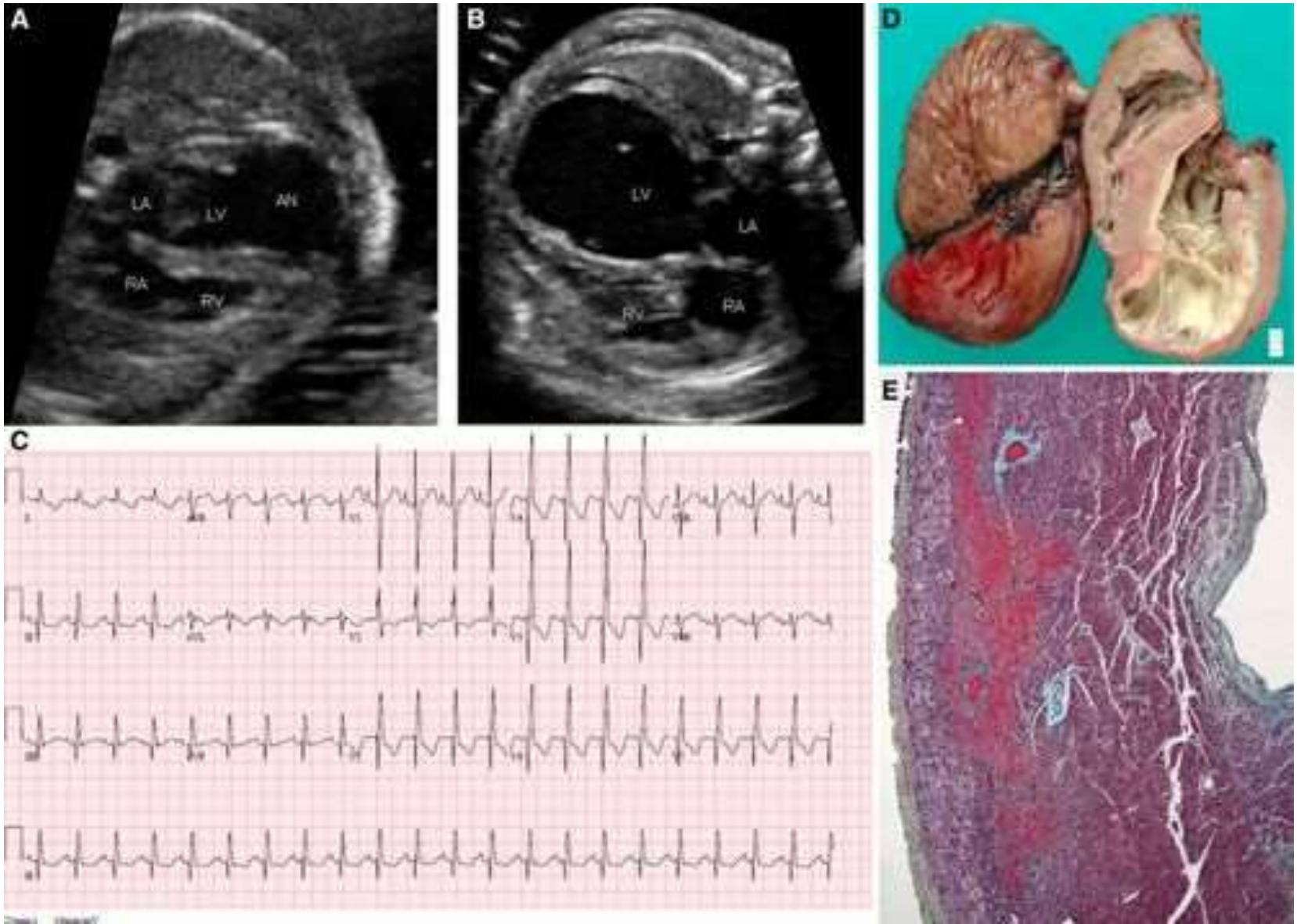

1.0 25 mm/s 07.00 22:33 1/1131 Л
I
II
V1
V2
V3
V4
V5
V6


I
II
III
AVR
AVL
AVF
V1
V2
V3
V4
V5
V6
15:31:01 13.Apr.04 25mm/s 10mm/mV ADS 50Hz 35Hz
15:31:01 13.Apr.04 25mm/s 10mm/mV ADS 50Hz 35

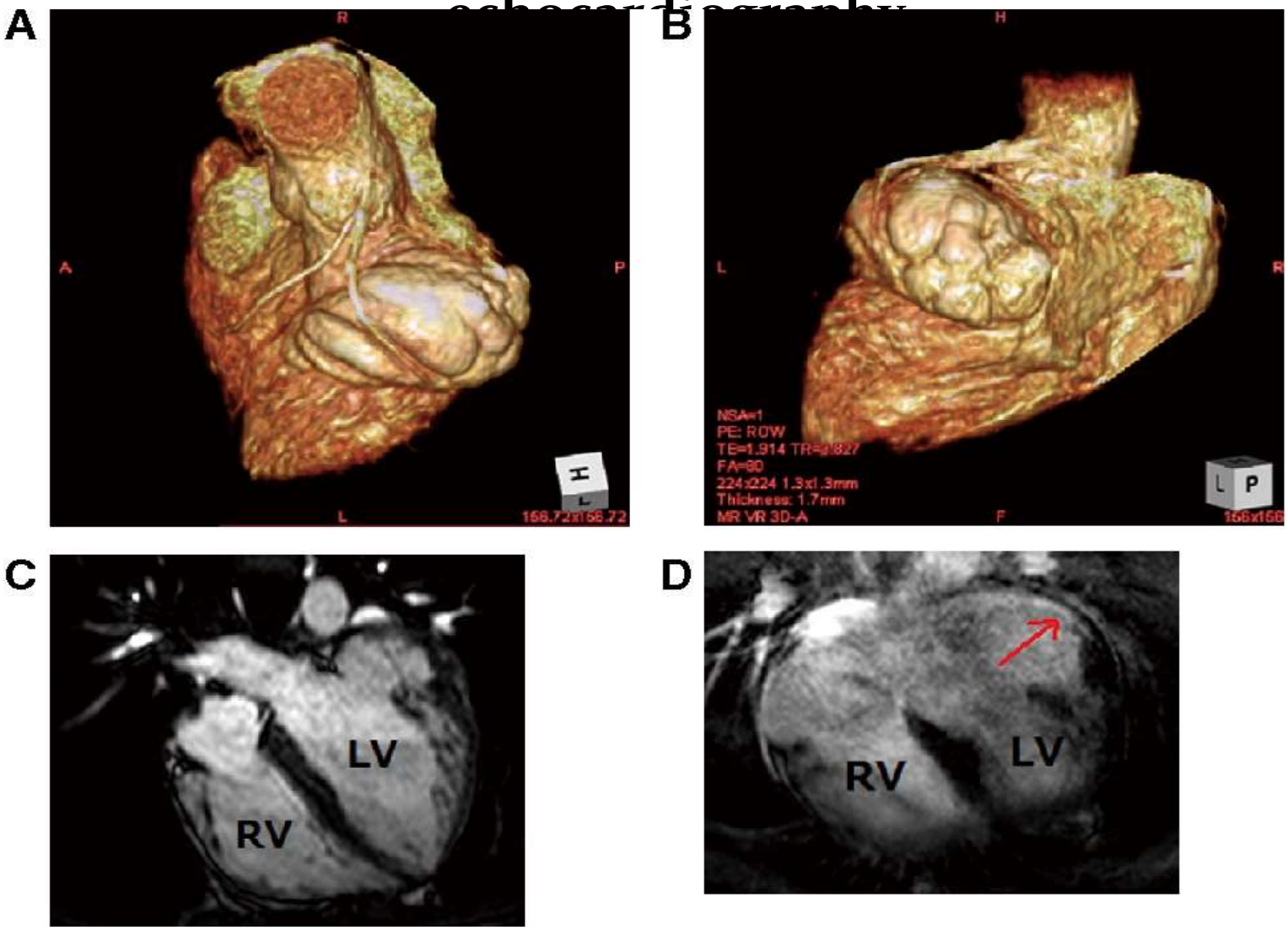
Echocardiography



Echocardiography



Tomographic three-dimensional echocardiography

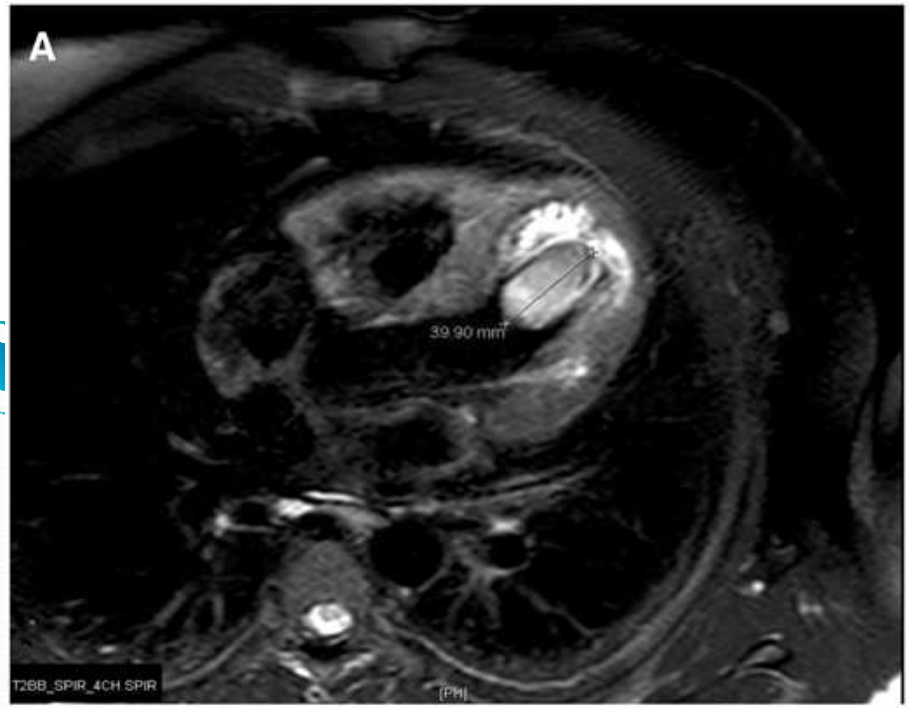


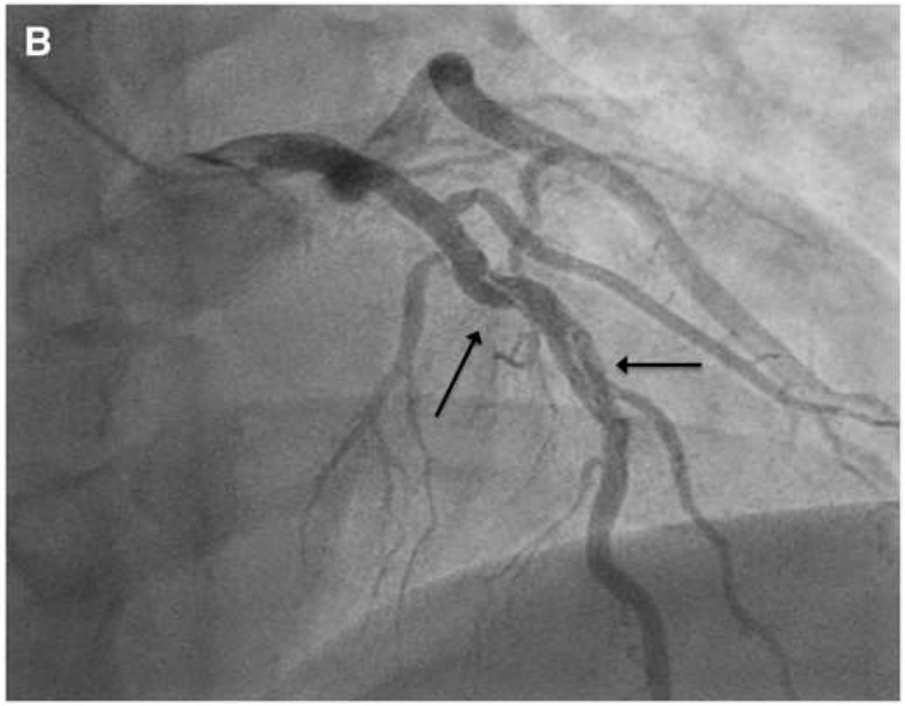
Three-dimensional cardiac magnetic resonance imaging showing left ventricular (LV) aneurysm (6.7 cm×3.9 cm) in the anterolateral wall. (C) Cine magnetic resonance imaging showing hypokinetic apical LV wall motion and mild thinning of the aneurysm wall. These findings suggested congenital LV aneurysm. (D) Late gadolinium enhancement is apparent on the endocardial side of the aneurysm. RV, right ventricle. Less

7. LV thrombi

- LV thrombi occurs in 20-40 % of **anterior Infarction** Patients
- LV thrombus is clearly demonstrated in Echocardiography

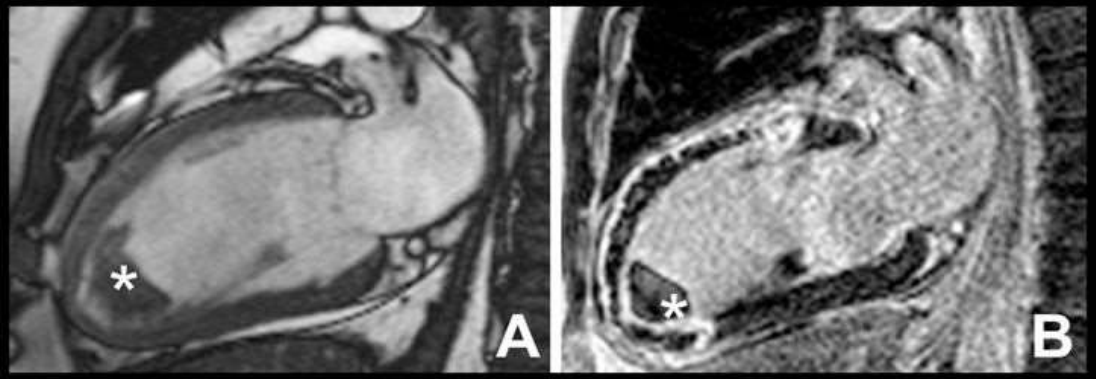
- Weakness
- sweating
- prolonged low-grade fever
- persistent tachycardia
- progressive heart failure





Magnetic resonance imaging

Fig. 1



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Embolic Complications

Prevalence

The incidence of clinically evident systemic embolism after MI is lower than 2%. The incidence increases in patients with anterior wall MI. The overall incidence of mural thrombus after MI is approximately 20%. Large anterior MI may be associated with mural thrombus in as many as 60% of patients.[47,48]

Pathophysiology

Most emboli arise from the left ventricle as a result of wall motion abnormalities or aneurysms. Atrial fibrillation in the setting of ischemia may also contribute to systemic embolization.

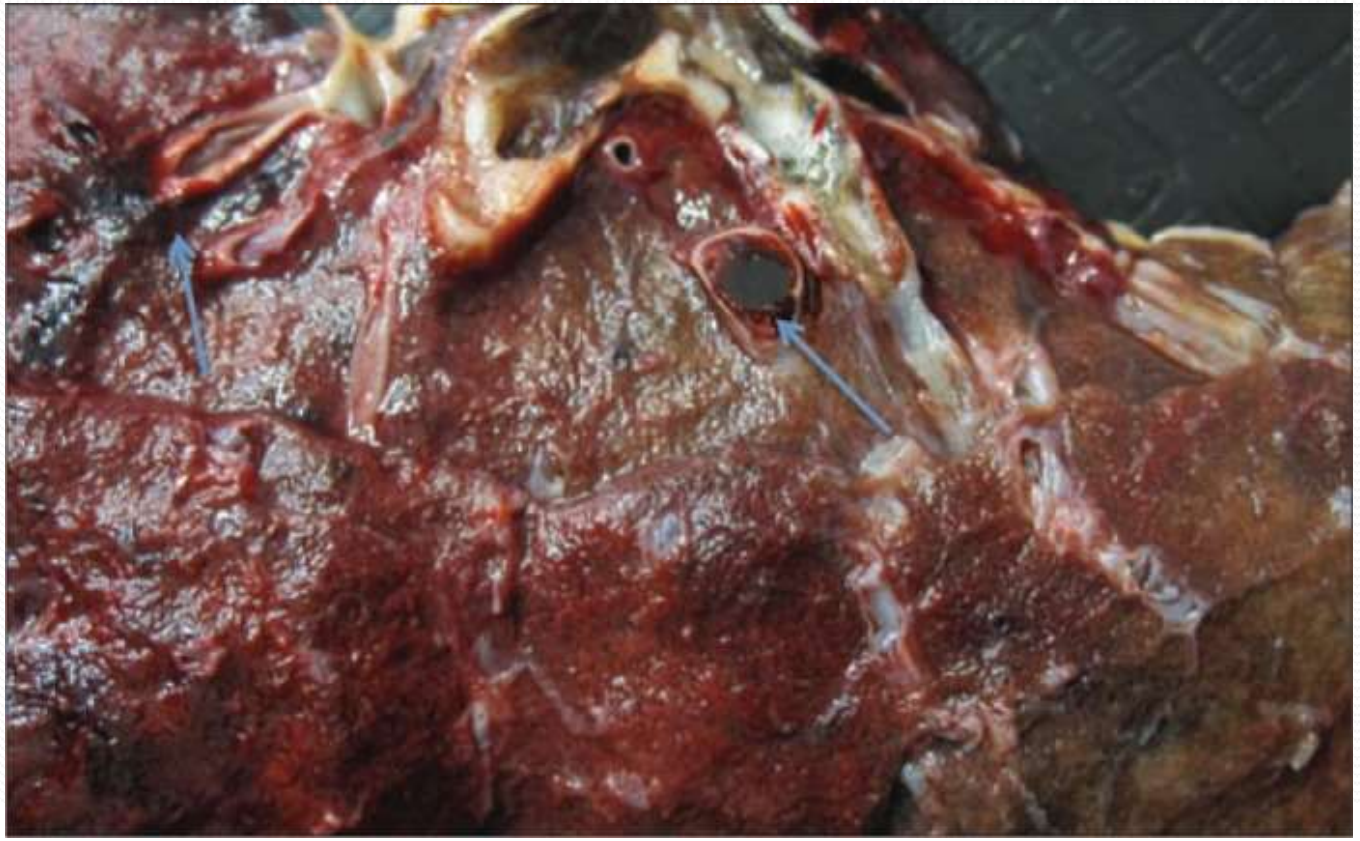
Signs and Symptoms

The most common clinical manifestation of embolic complications is stroke, although patients may have limb ischemia, renal infarction, or intestinal ischemia. Most episodes of systemic emboli occur in the first 10 days after acute MI. Physical findings vary with the site of the embolism. Focal neurologic deficits occur in patients with central nervous system emboli. Limb ischemia manifests with limb pain in a cold pulseless extremity. Renal infarction manifests with flank pain and hematuria. Mesenteric ischemia manifests with abdominal pain out of proportion to physical findings and bloody diarrhea.

Other Mechanical Complications

5. Systemic embolism Pulmonary Embolism:

- Post-infarction patients have greater tendancies for Pulmonary embolism (P.E) due to decreased cardiac output and immoblization ,
-



Heart Failure

- Heart Failure occurs when LV function decrease by 30% of normal function .
- **The Killip Classification is frequently used during acute myocardial infarction**.

- **Killip Class**

Class I: No evidence of heart failure (mortality 6%)

Class II: Findings of mild to moderate heart failure (S3 gallop, rales < half-way up lung fields or elevated jugular venous pressure (mortality 17%)

Class III: Pulmonary edema (mortality 38%)

Class IV: Cardiogenic shock defined as systolic blood pressure < 90 and signs of hypoperfusion such as oliguria, cyanosis, and sweating (mortality 67%)

Cardiogenic shock

- **Cardiogenic shock** is persistent hypotension with a systolic pressure <80 mm Hg for more than 30 minutes in the absence of hypovolemia. It occurs when ≥ 40 % of myocardium is affected .
- **The most common causes of cardiogenic shock include**

1) **large left ventricular infarct** (usually >40% of left ventricle) seen in about 80% of shock patients

2)right ventricular infarct in 10% of shock patients

3) **mechanical complications of myocardial infarction** (ventricular septal defect, acute mitral regurgitation, tamponade) in 10% of shock patients.

SYMPTOMS:

- Clammy skin.
- Decreased systolic blood pressure
- Tachycardia
- Rapid respirations
- Oliguria.
- Cyanosis.
- Mental confusion

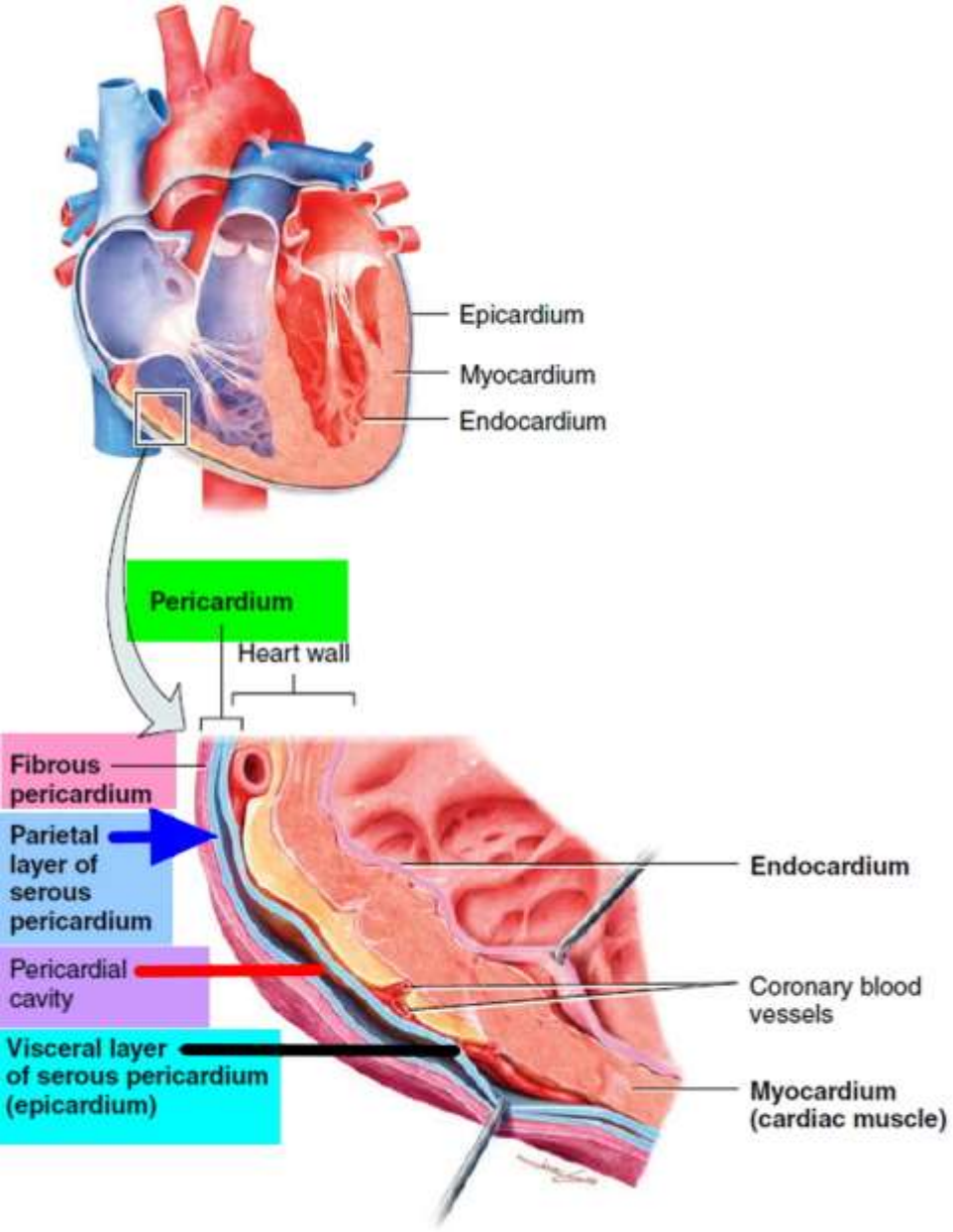
- **Pericarditis :**

1. **Early post-infarction Pericarditis (episthenocarditis)**

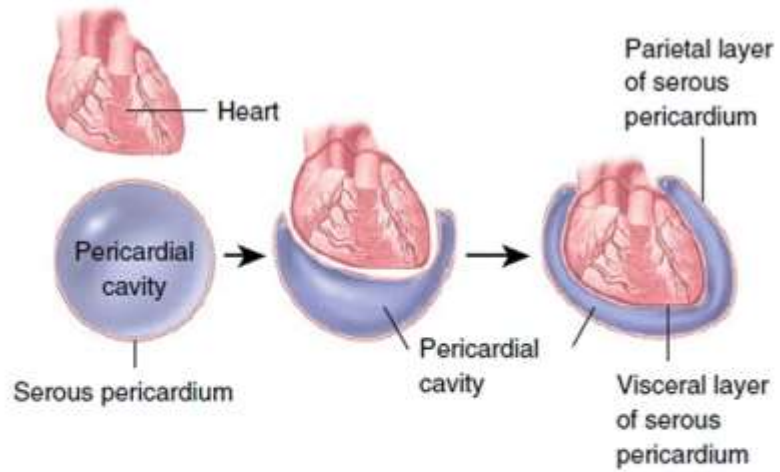
- Occurs 2-4 days following acute infarction .
- Patients who develop this condition usually have larger infarcts , lower EF and higher incidence of CHF .
- Presented by Fever , Chest pain and friction rub .
- Pain is aggrevated by movement and inspiration , Radiated to the trapezius .

2. Post MI autoimmune syndrome (Dressler's Syndrome)

- Dressler syndrome is a multiple organ autoimmune inflammatory process which manifests with secondary pericarditis, pleurisy, damage of the synovial membranes of the joints.
- develops **weeks to months after a myocardial infarction** characterized by pleuritic chest pain, worse on inspiration, low-grade fever, pericarditis, joints pain
- **Blood tests.** The results of certain tests can indicate inflammatory activity that's consistent with Dressler's syndrome.
- Leukocytosis
- Elevated erythrocyte sedimentation rate

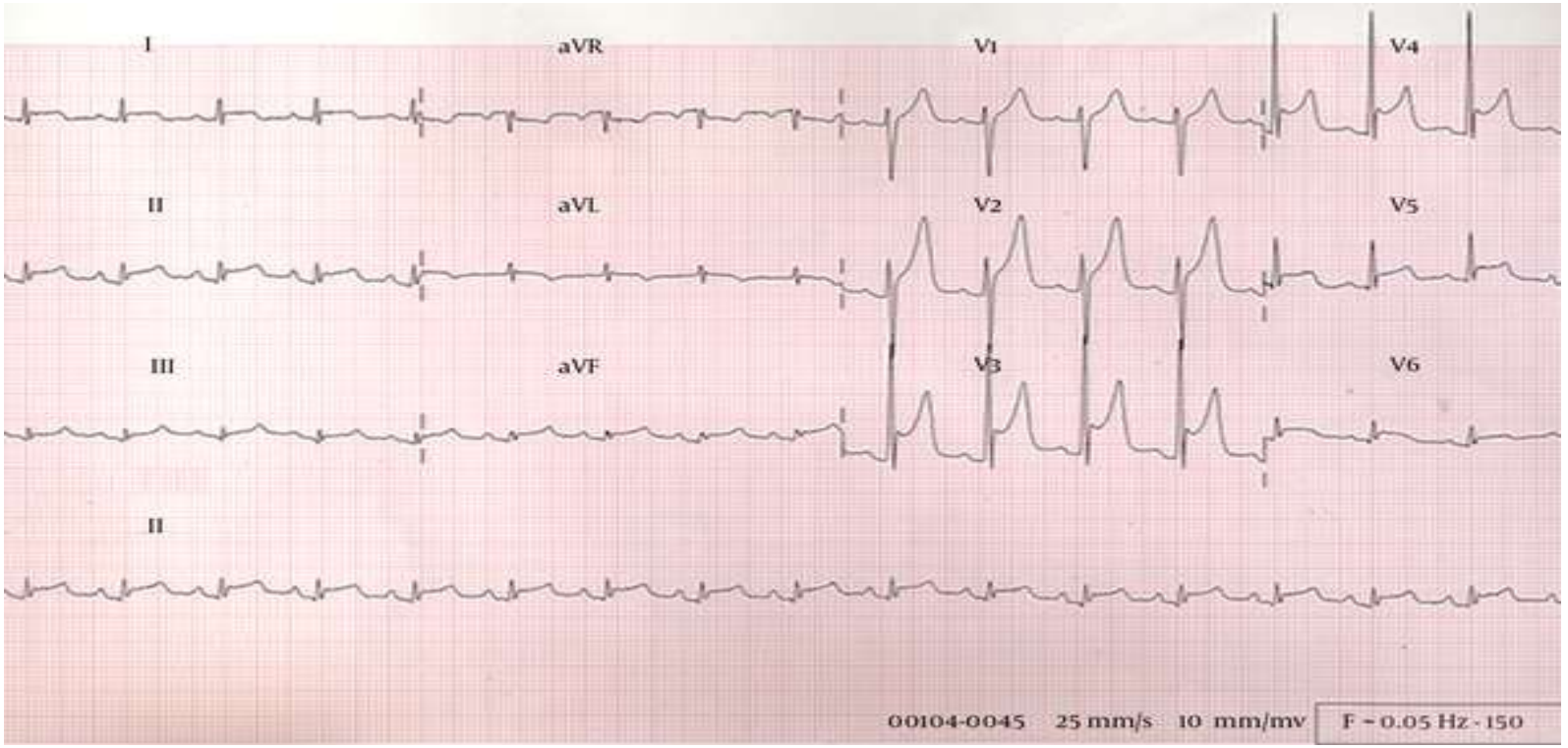


(a) Portion of pericardium and right ventricular heart wall showing the divisions of the pericardium and layers of the heart wall



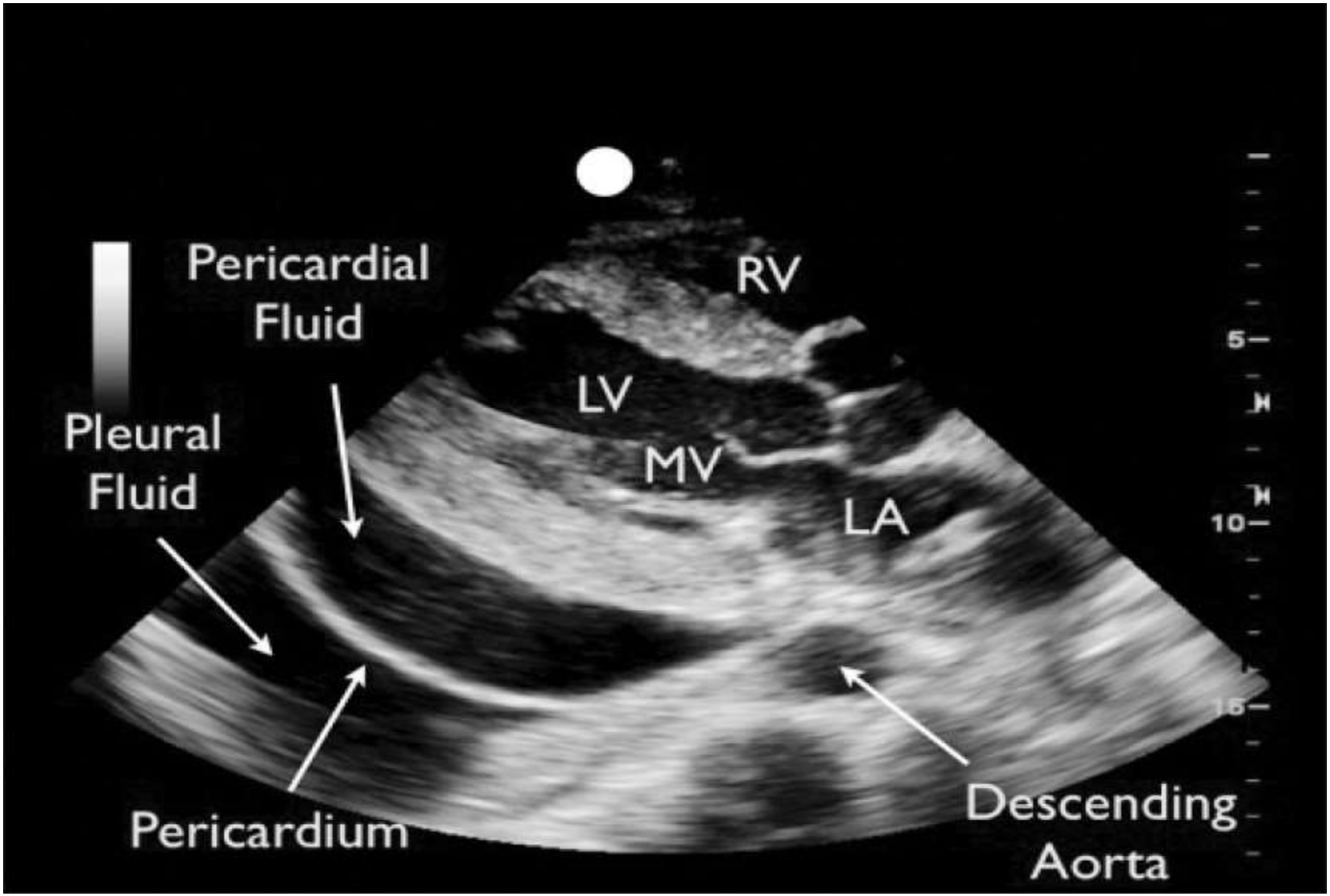
(b) Simplified relationship of the serous pericardium to the heart

Electrocardiogram.

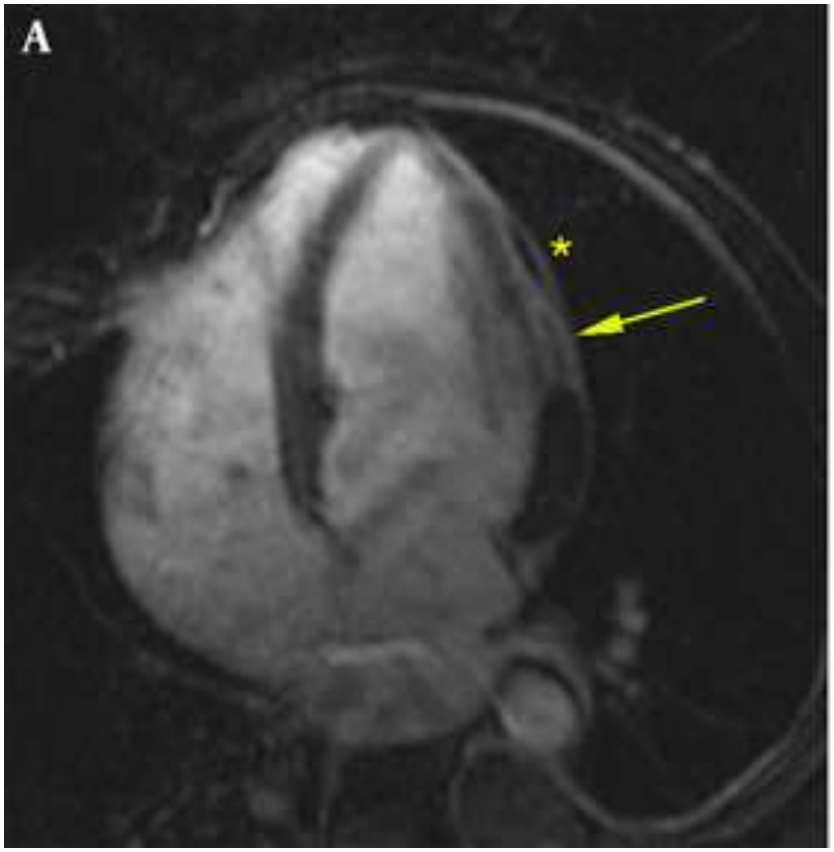


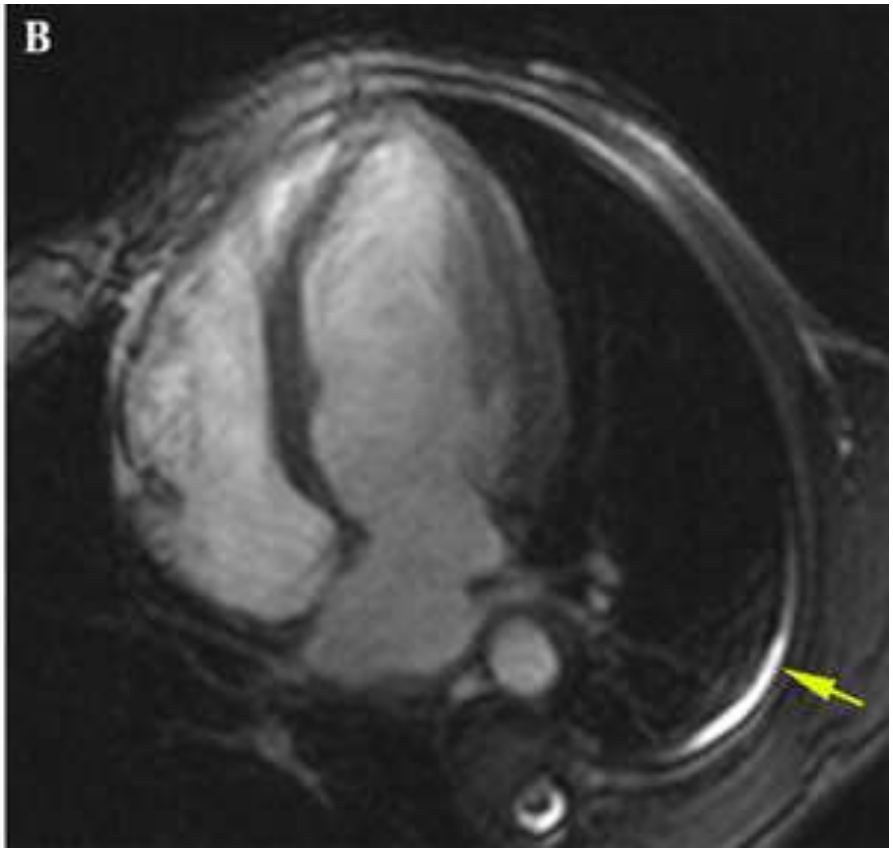
Electrocardiogram changes Showing Diffuse **ST Segment Elevation** with
Depression of the PR Segment, mainly in the II standart lead

Echocardiogram



CMR





CMR images demonstrating: A, **left ventricle inferolateral transmural fibrosis** in white (arrow) and **pericardial damage***; and B, late gadolinium enhancement sequences (four chamber) with a **small pleural effusion** (arrow) (cine SSFP four chamber image); CMR: cardiac magnetic resonance imaging, SSFP: steady state free precession.

- Cardiogenic shock and heart failure are the most common causes of death in patients hospitalized with acute myocardial infarction.

- At least 75% of patients with acute myocardial infarction (MI) have an arrhythmia during the periinfarct period.

Thanks for your attention