Definiton

The clinical presentations of CAD include:

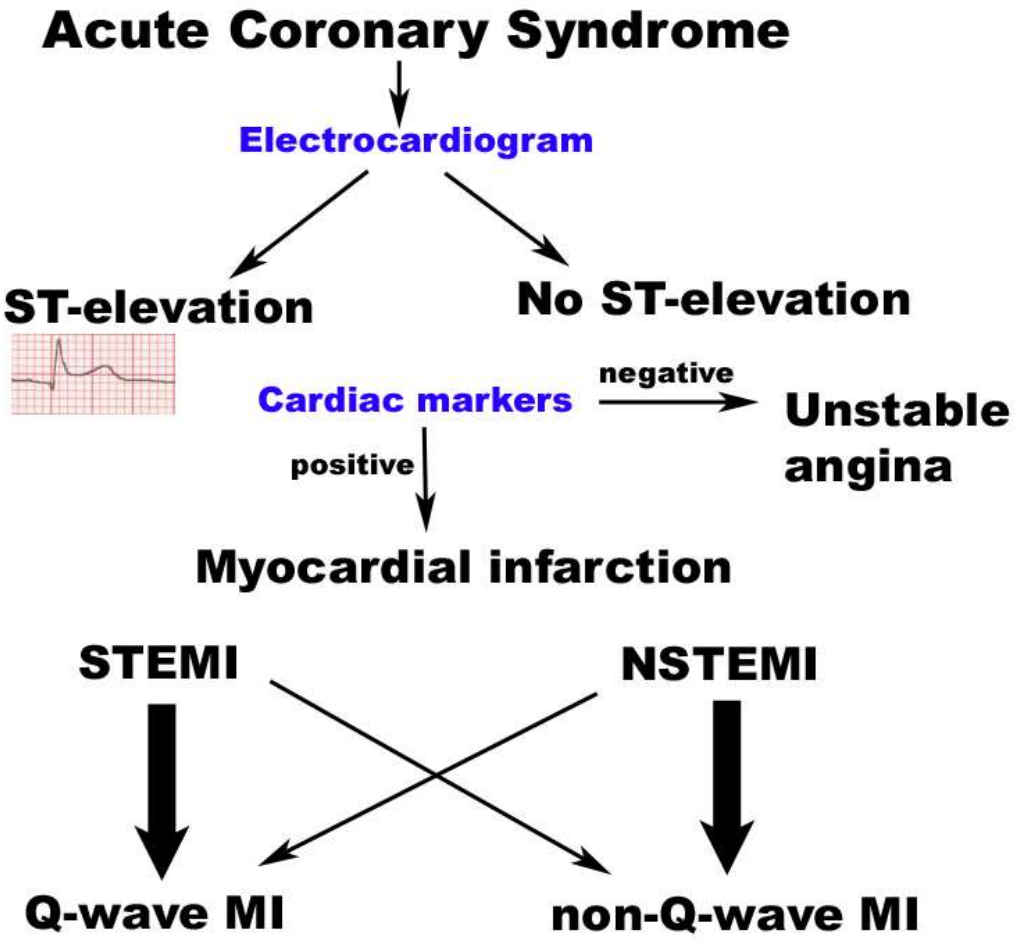
- silent ischaemia
- stable angina pectoris
- heart failure
- **unstable angina**
- **myocardial infarction (MI)**
- **sudden death**

Acute coronary syndromes

ACS are usually divided into:

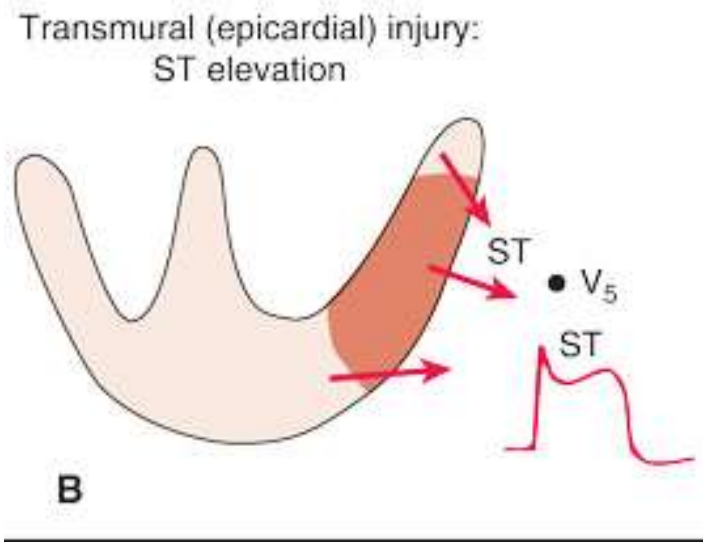
- Unstable angina - only ischemia, lack of necrosis
- STEMI - ST - elevation MI
- NSTEMI - non-ST elevation MI
- Sudden death - due to cardiac arrhythmias

Acute coronary syndromes

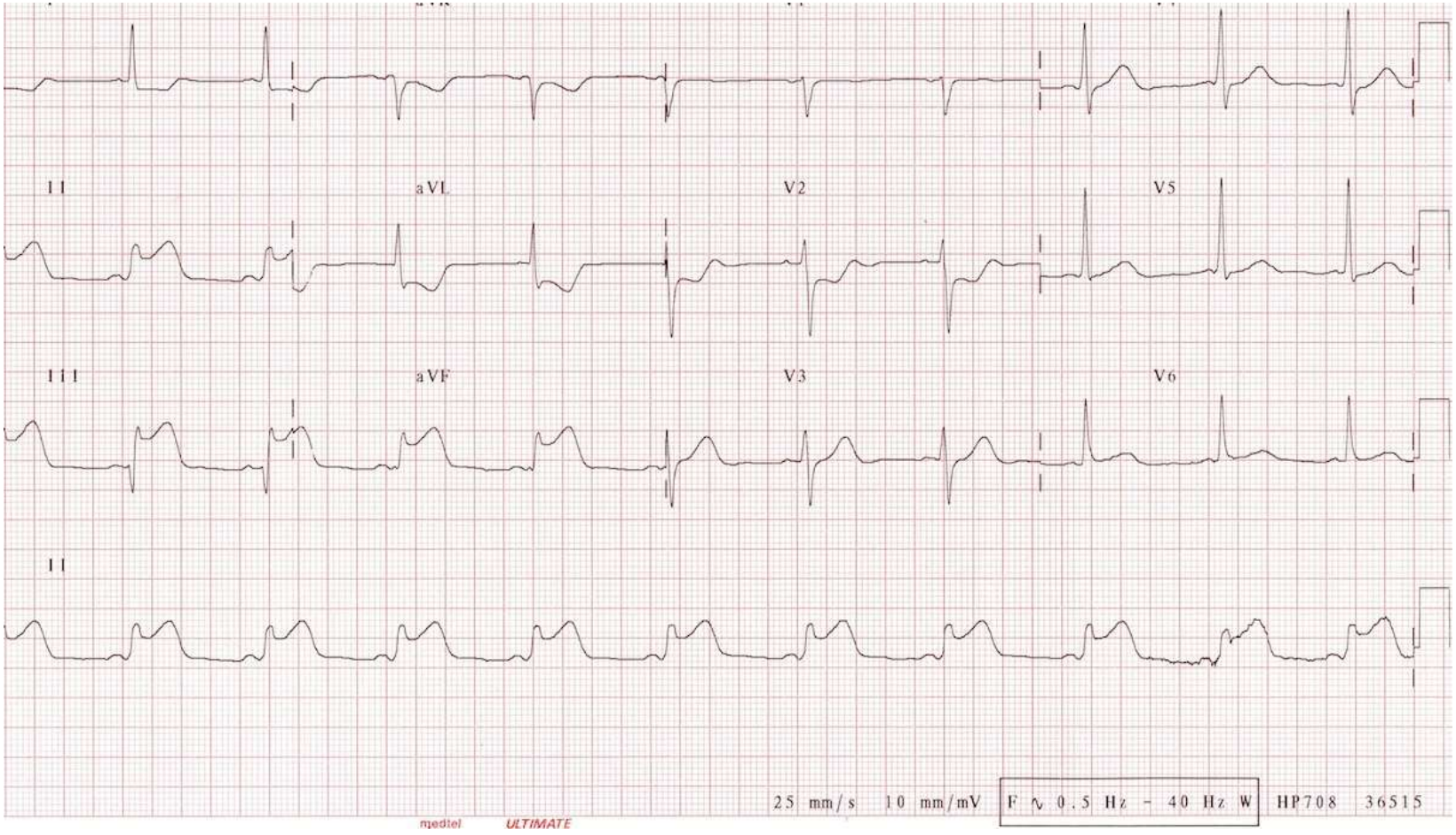


Definition

- **ST-elevation ACS (STE-ACS):**
- typical acute chest pain and persistent (>20 min)
- ST-segment elevation
- generally reflects an acute total coronary occlusion
- most will ultimately develop an ST-elevation MI (STEMI).

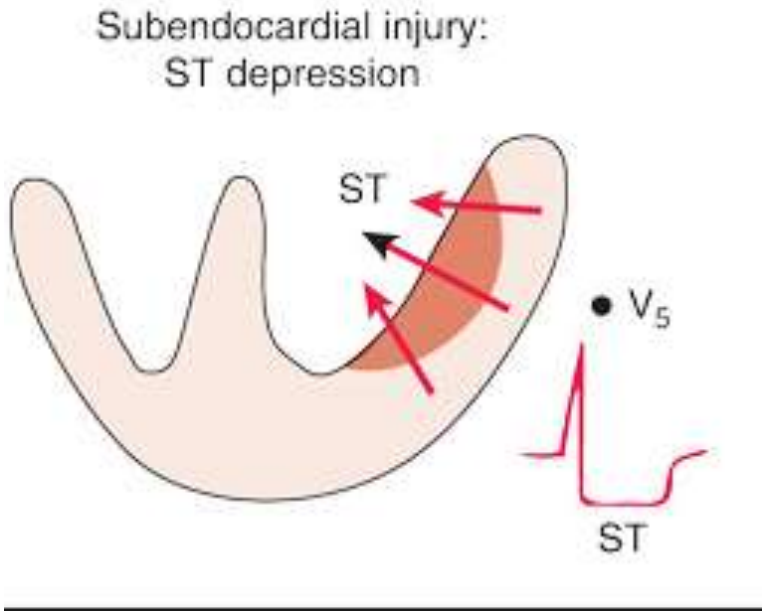


ST elevation on the ECG

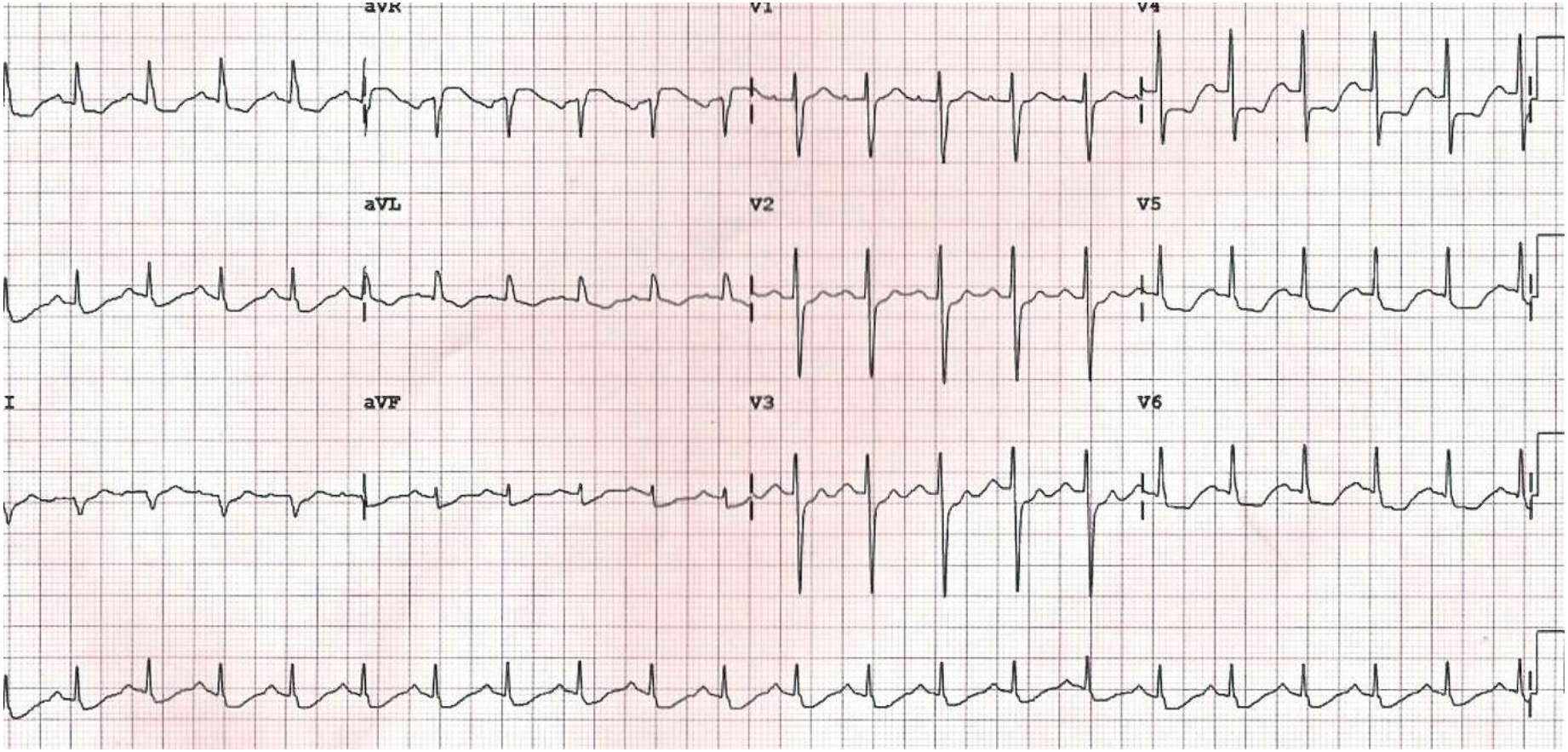


Definition

- **non-STE-ACS (NSTE-ACS):**
- acute chest pain
- without persistent ST-segment elevation
- persistent or transient ST segment depression or T-wave inversion
- further qualified into non-ST elevation MI (NSTEMI) or unstable angina.

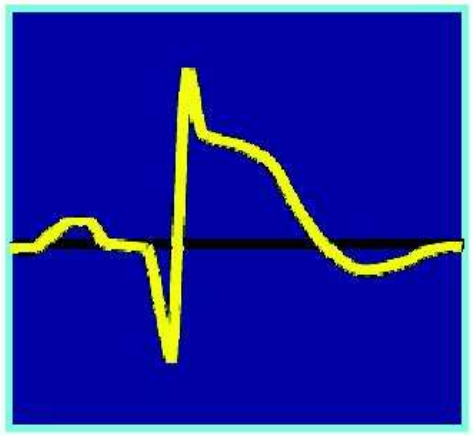


ST depresion on the ECG



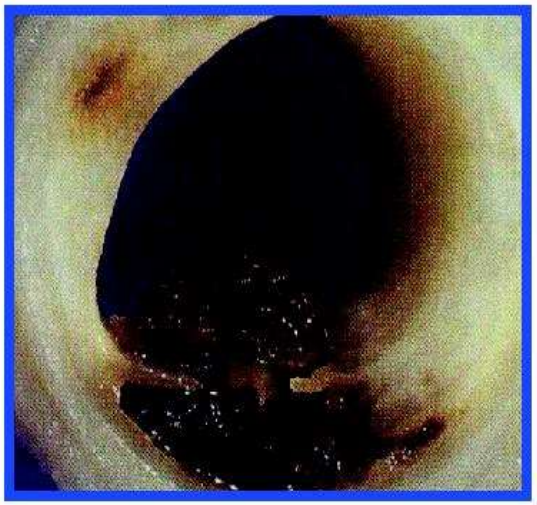
ACS with persistent ST-segment elevation

Adapted from Michael Davies

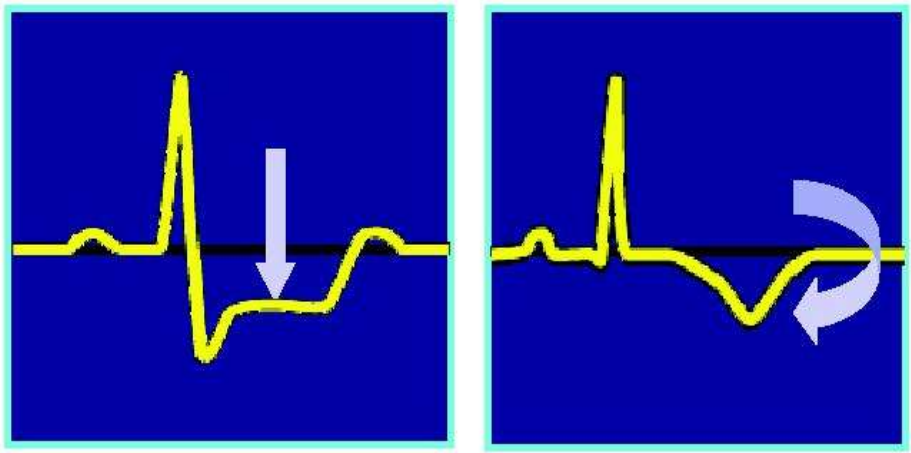


Troponin elevated

ACS without persistent ST-segment elevation



Adapted from Michael Davies



Troponins elevated or not

Pathophysiology of ACS

Atherothrombosis

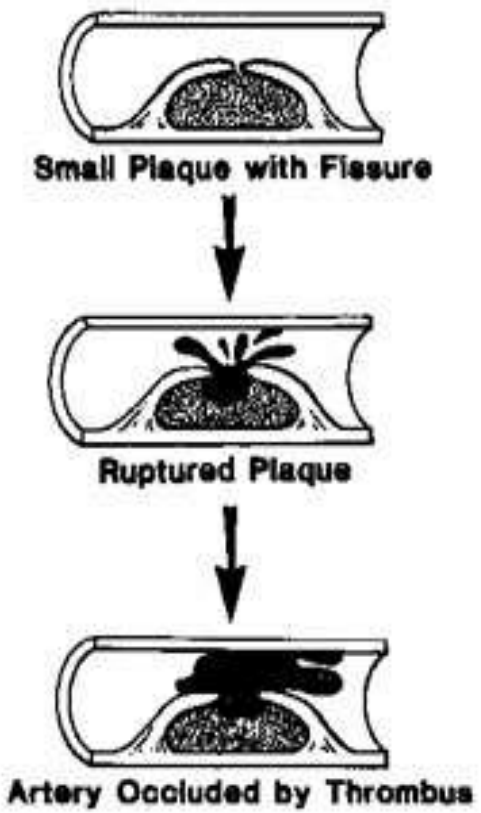
- Atherosclerosis - a fixed and barely reversible process of gradual luminal narrowing

  (slowly over decades)

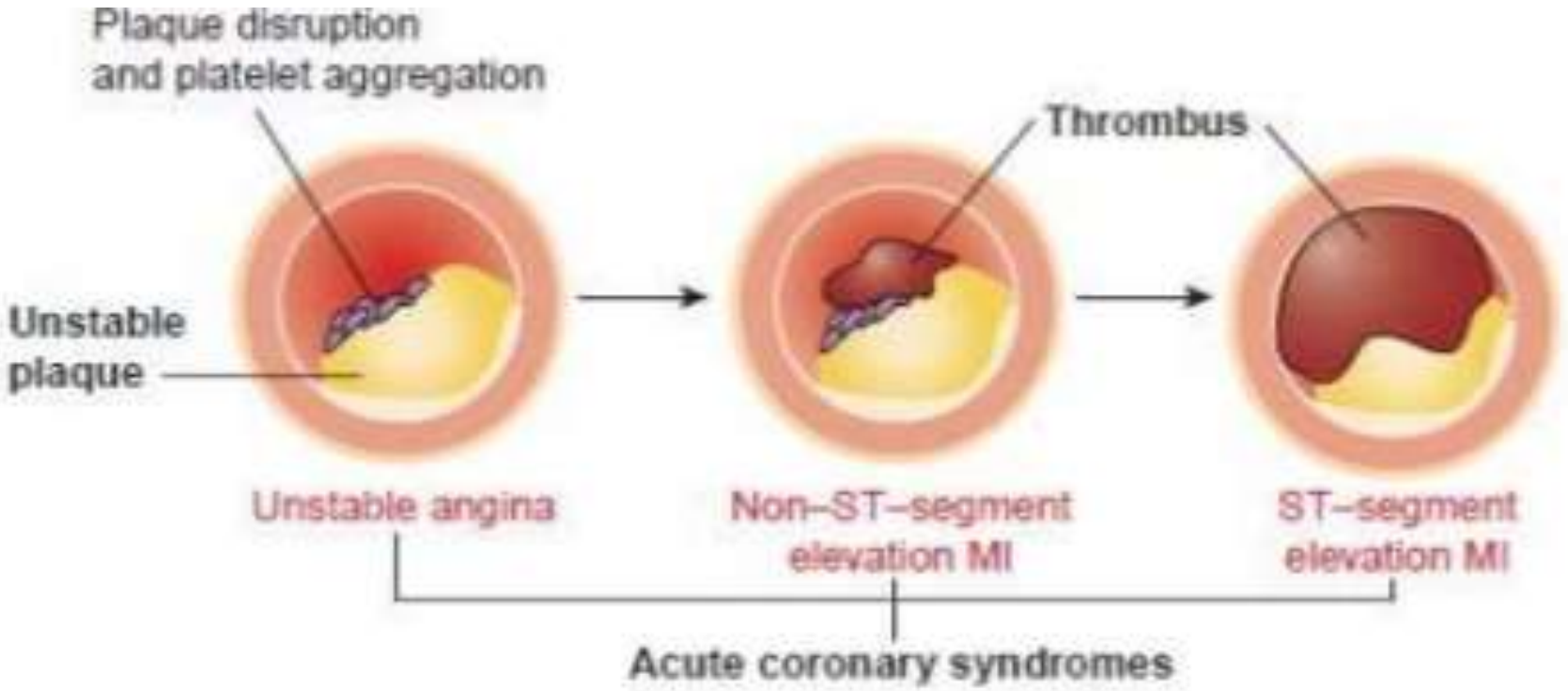
- Thrombosis - a dynamic and potentially reversible process causing rapid complete or partial occlusion of the coronary artery

Vulnerable plaque

- a large lipid core
- a low density of smooth muscle cells
- a high concentration of inflammatory cells
- a thin fibrous cap covering the lipid core
- acute thrombosis induced by a plaque rupture



Vulnerable plaque



Prognosis of STE vs. NSTE-ACS

Hospital mortality

- higher in patients with STEMI than among those with NSTE-ACS (7 vs. 5%)

6 months mortality

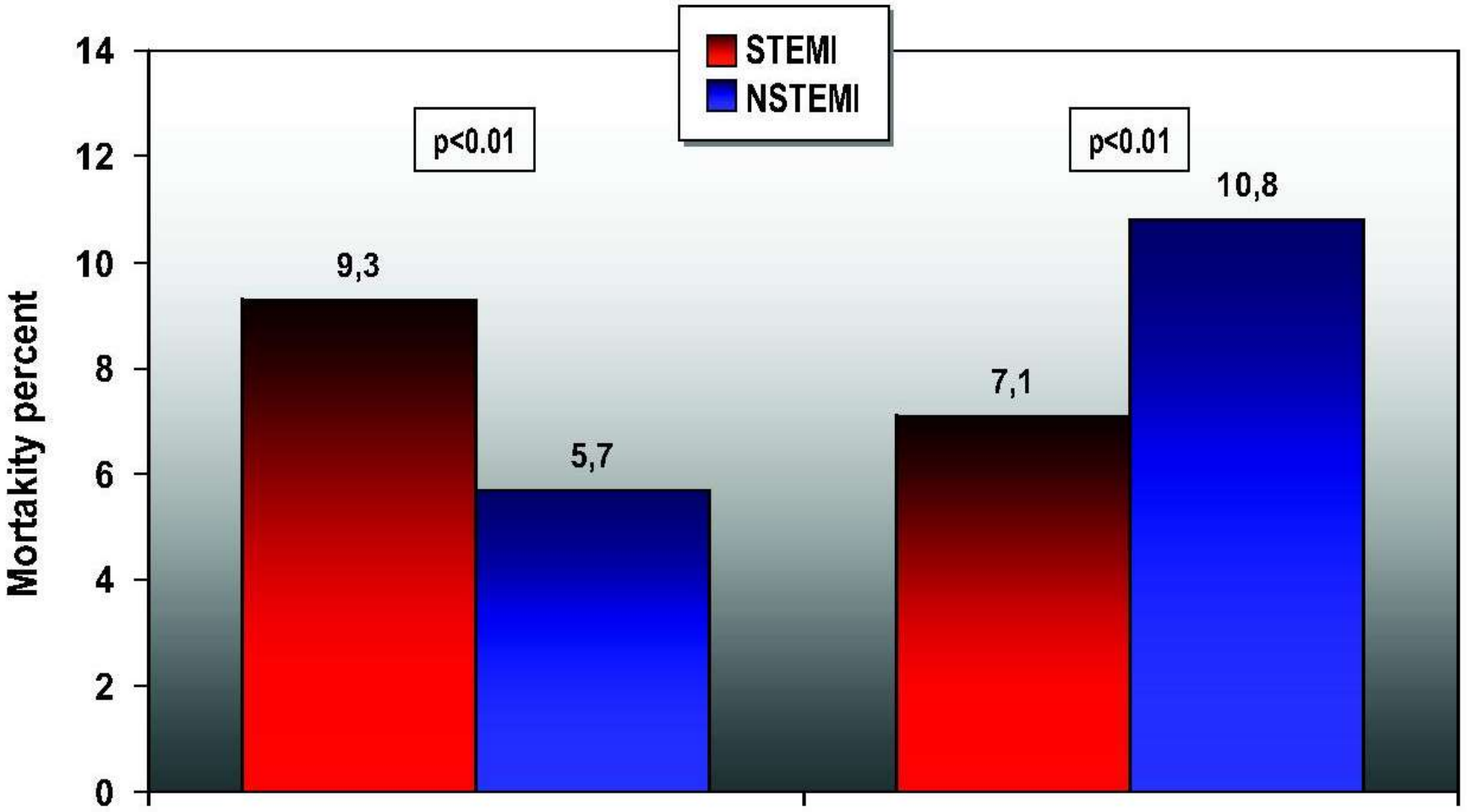
- the mortality rates are very similar in both conditions (12 vs. 13%)

Long-term follow-up

- death rates higher among those with NSTE-ACS than with STE-ACS

STEMI versus NSTEMI

Hospital vs 1-Year-Mortality



Myocardial infarction

1. **Atherosclerotic aetiology** (type 1)
2. **Non-atherosclerotic aetiology:** (type 2-5)

- arteritis
- trauma
- dissection
- congenital anomalies
- cocaine abuse
- complications of cardiac catheterization, CABG

Diagnosis of acute MI

2 from 3 criteraia must be fulfilled :

- **Clinical symtoms**
  - Chest pain
- **ECG changes**
  - ST elevation or depression
  - negative T wave
- **Elevated cardiac biomarkers**
  - Troponin I or T
  - CK-MB
  - myoglobin

Diagnosis of ACS

- Clinical presentation
- History of patient
- Physical examination
- Electrocardiogram
- Biochemical markers - troponin
- Non-invasive imaging - Echo
- Imaging of coronary arteries - coronary angiography

Clinical presentation

STE/NSTE-ACS:

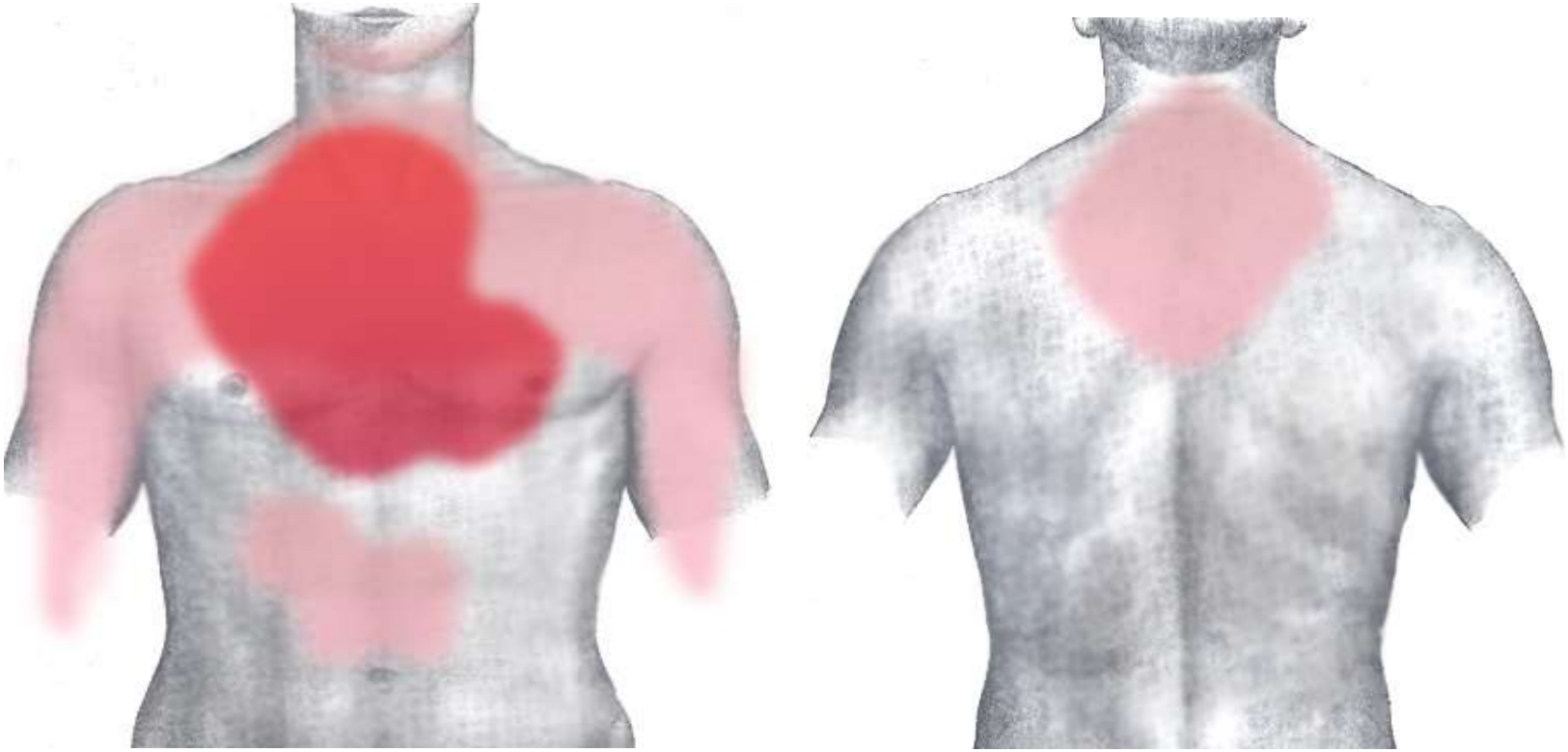
- Intense prolonged (20 min) pain at rest - retrosternal pressure or heaviness ('angina') radiating up to the neck, shoulder and jaw and down to the ulnar aspekt of the left arm
- May be accompanied by other symptoms such as diaphoresis, nausea, abdominal pain, dyspnoea,…

Unstable angina:

- New onset severe angina (class III of CCS)
- Recent destabilization of previously stable angina with at least CCS III angina characteristics (crescendo angina)
- Post-MI angina.

Clinical presentation

1) Typical chest pain



2) Nauzea
3) Sweating

Clinical presentation

Atypical presentations are not uncommon

- epigastric pain
- recent-onset indigestion
- stabbing chest pain
- chest pain with some pleuritic features
- increasing dyspnoea

- often can be observed in younger (25-40y.), older (75y.), in women, in pts. with diabetes, chronic renal failure, or dementia.

Clinical presentation

- The presence of tachycardia, hypotension, or heart failure needs rapid diagnosis and management, often indicating a poor prognosis of this patient with ACS

- It is important to identify the clinical circumstances such as anaemia, infection, inflammation, fever, and metabolic or endocrine (in particular thyroid) disorders (may exacerbate or precipitate ACS)

Physical examination

- Frequently normal
- Signs of heart failure or haemodynamic instability
- **Dif. dg.:**

– nonischaemic cardiac disorders: pulmonary embolism, aortic dissection, pericarditis, valvular heart disease)

- extra-cardiac causes: pulmonary diseases - pneumothorax, pneumonia, pleural effusion)

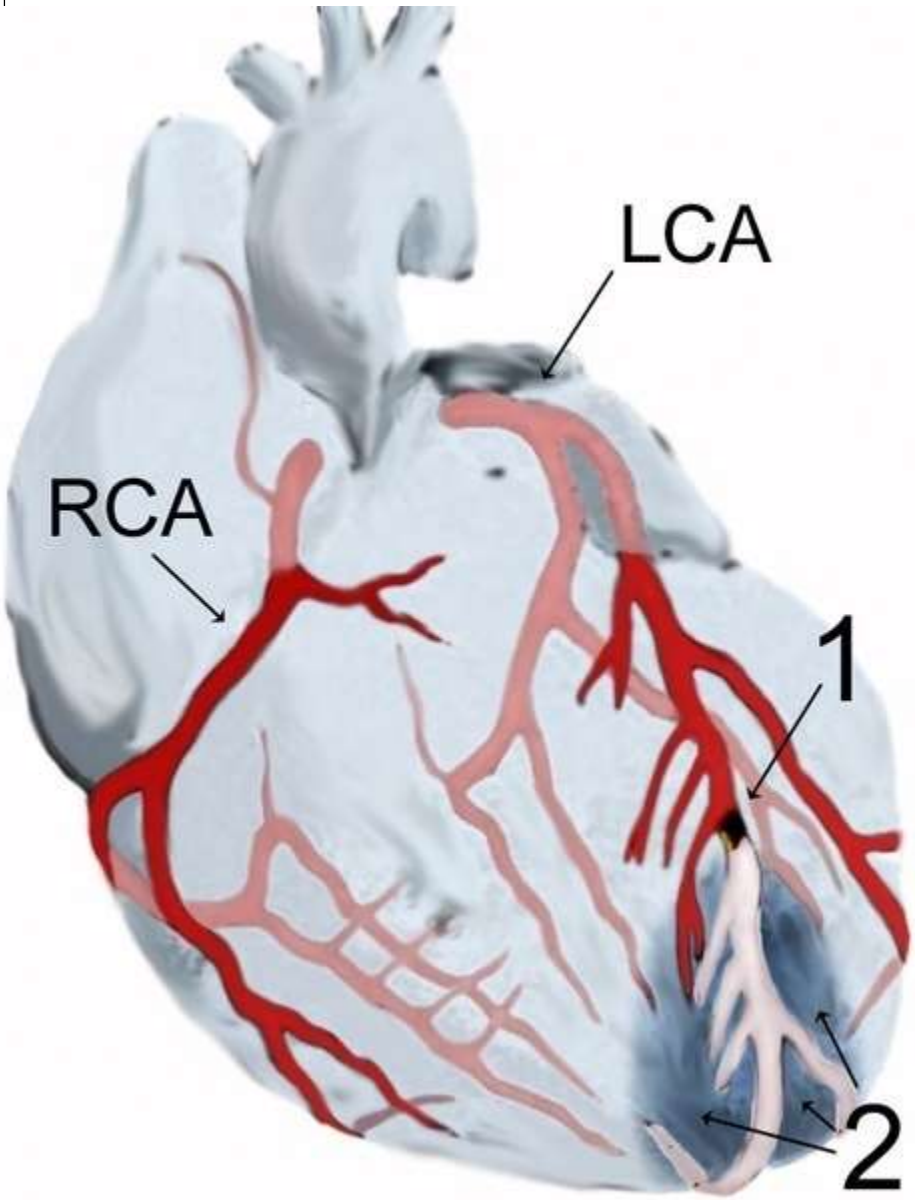
Physical examination

- Heart failure
  - Tachycardia, tachypnoe
  - Pulmonary rales (pulmonary congestion)
  - RV failure - ↑ jugular congestion, hepatomegaly
- Hypotension ↓ 100/60 mmhg
  - cardiac shock (tachycardia)
  - ↑ vagal nerve activity (bradycardia - inferior IM)
- Bradycardia
  - AV block
    - Inferior IM - non-serious, frequent
    - Anterior IM - serious, rare

Electrocardiogram

- The resting 12-lead ECG is the first-line diagnostic tool in the assessment of patients with suspected ACS.
- STE-ACS… ST-elevation
- NSTE-ACS…ST-segment shifts and T-wave changes
- A completely normal ECG does not exclude the possibility of ACS.

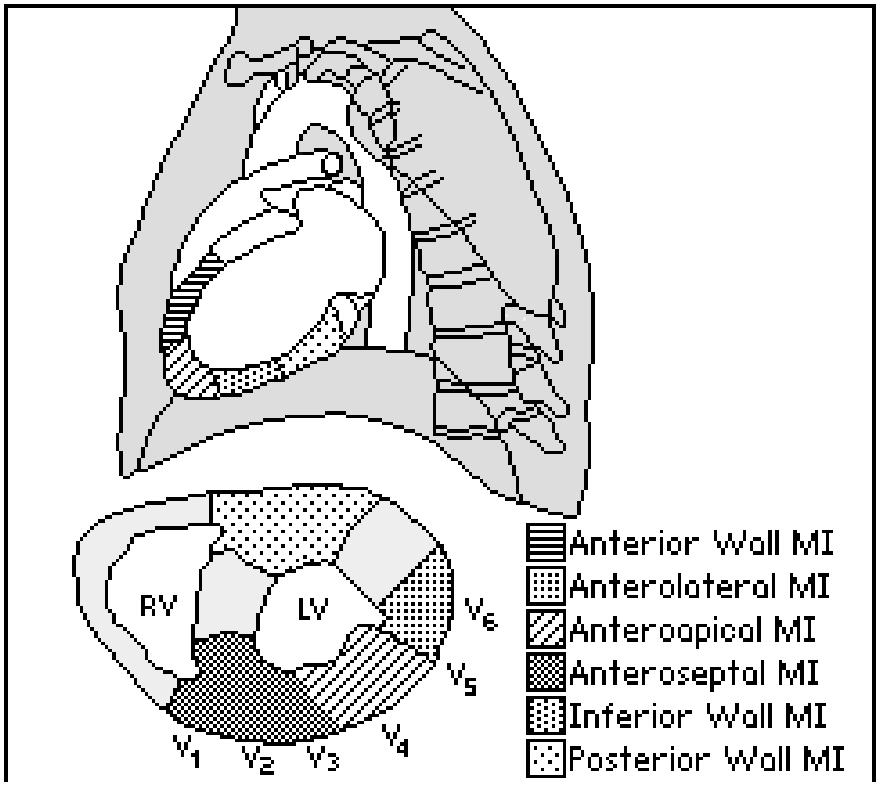
Location of MI



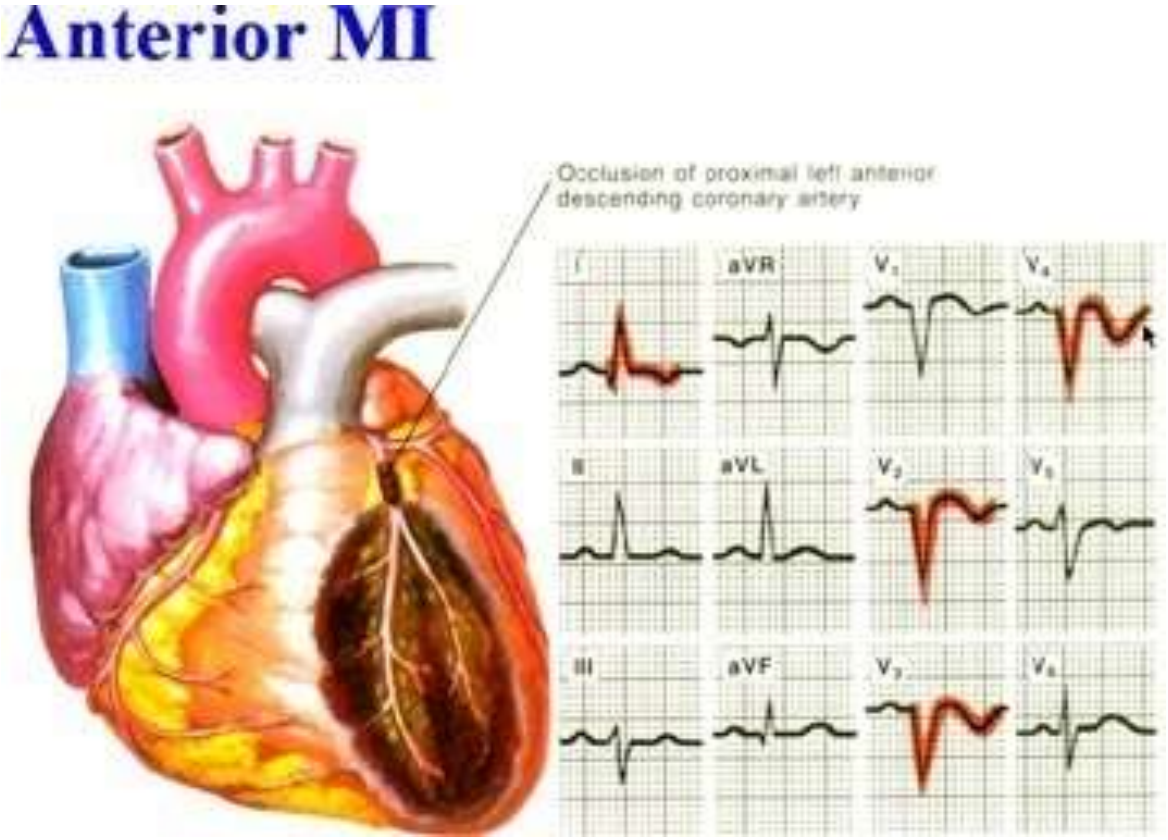
Location of MI

ST elevation only:

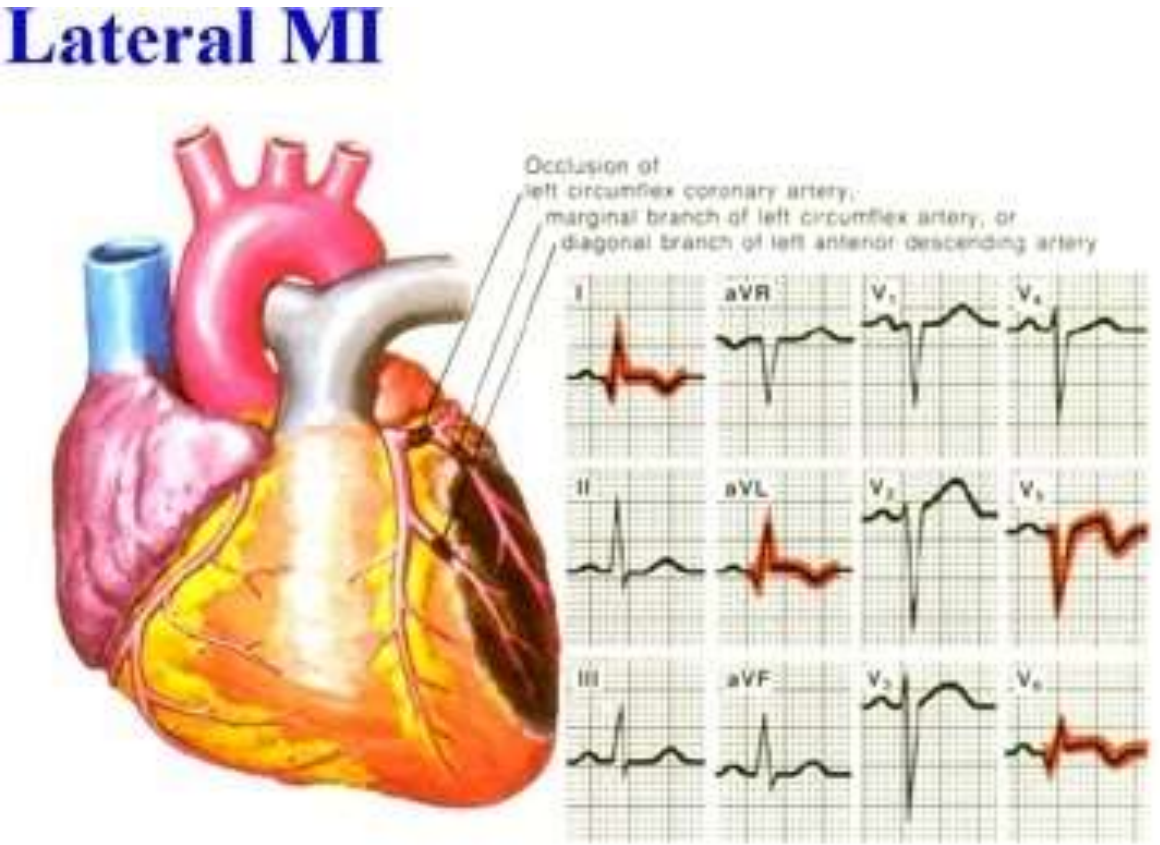
- Anteroseptal - V1-V3
- Anterolateral - V1-V6
- Inferior wall - II, III, aVF
- Lateral wall - I, aVL, V4-V6
- Right ventricular - RV4, RV5
- Posterior- R/S ratio >1 in V1 and T wave inversion



Location of MI

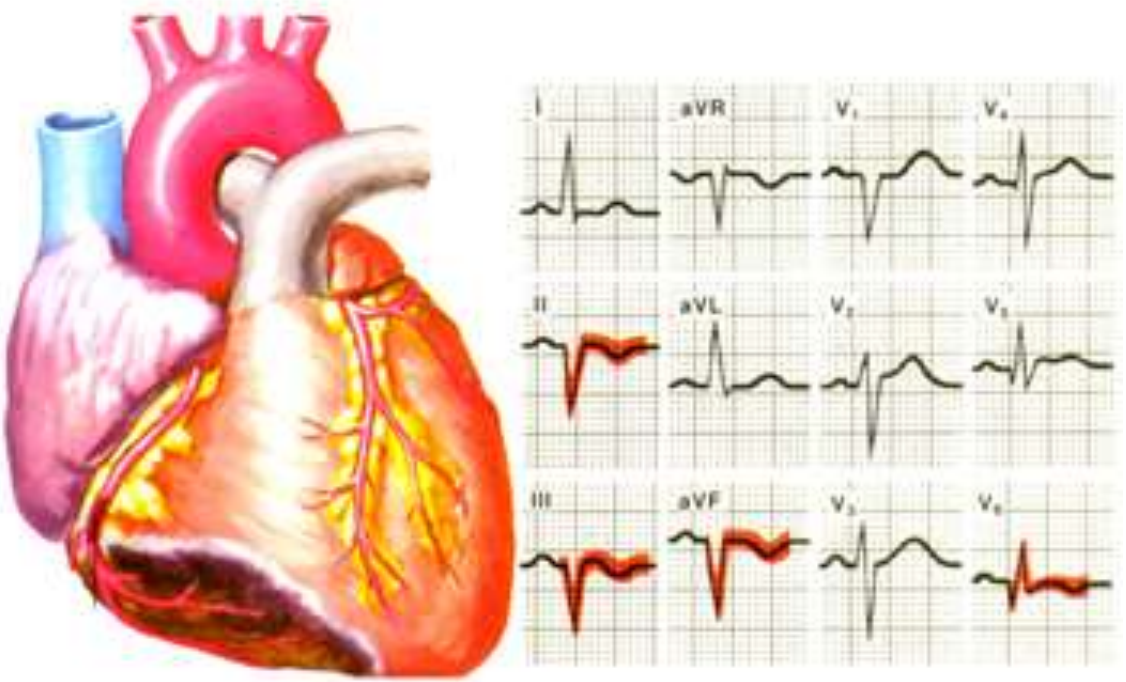


Location of MI



Location of MI

Inferior MI



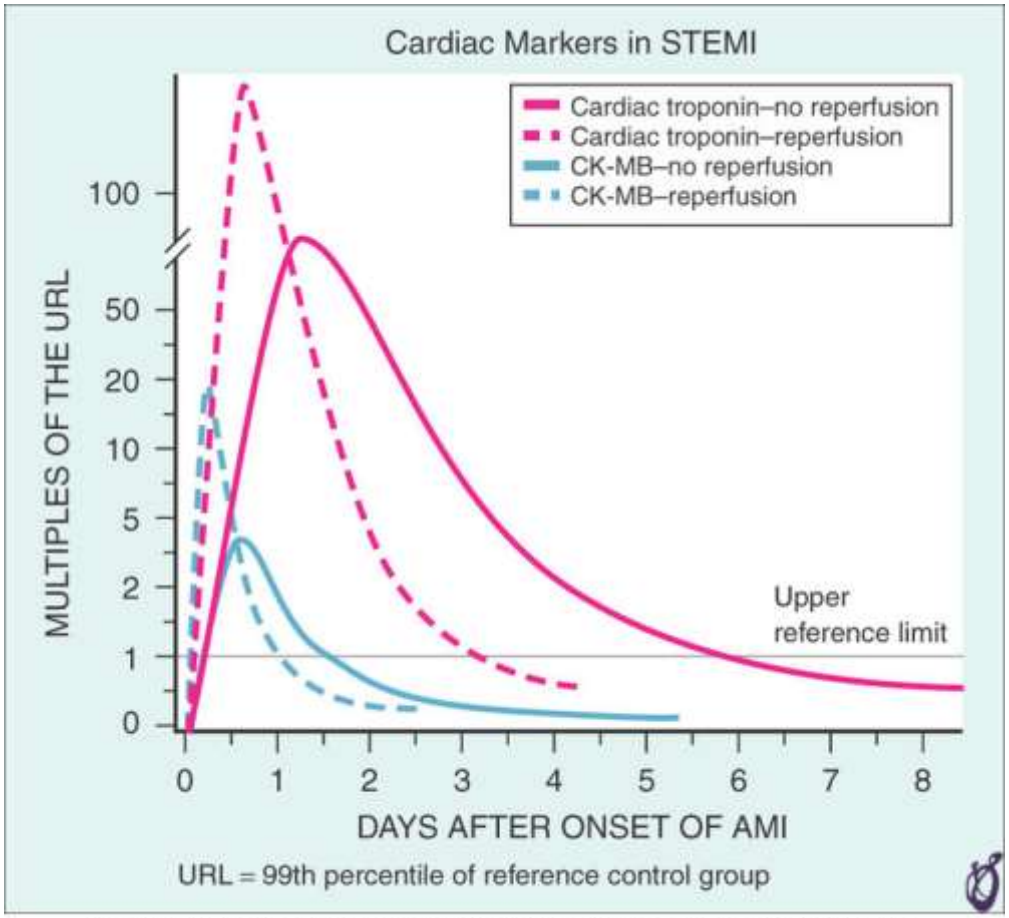
Biochemical markers

Markers of myocardial injury:

- cardiac troponins (I and T)
- creatinine kinase (CK)
- CK isoenzyme MB (CK-MB)
- Myoglobin

- repeated blood sampling and measurements are required 6–12 h after admission and after any further episodes of severe chest pain

Biochemical markers in ACS



(Modified from Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA Guidelines for the Management of Patients with ST-Elevation Myocardial Infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines [Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction]. American College of Cardiology Web site, 2004. [www.acc.org/clinical/guidelines/stemi/index.pdf]. Accessed 9/21/06.)

Biochemical markers

Non-coronary condition with Troponin elevation

- Severe congestive heart failure
- Aortic dissection, valve disease
- Myocarditis
- Hypertrophic CMP, Stress CMP
- Hypertesive crisis
- Acute and chronic renal failure
- Acute neurological disease
- …

Other biomarkers

- C-reactive protein - inflamation
  - long-term prognosis
- Natriuretic peptides - heart failure
  - shor-term prognosis
- Serum creatinine - renal function
  - Short and long-term prognosis

No role for the diagnosis of ACS, but effect on short- or long-term prognosis and dif. Dg.

Non-invasive myocardial imaging

- **Echocardiography**

  - to evaluate LV systolic function, aortic stenosis, aortic dissection, pulmonary embolism, or hypertrophic cardiomyopathy

  - should be routinely used in emergency units for the risc stratification

- **Stress echocardiography, stress scintigraphy** - evidence of ischaemia or myocardial viability (in stabilized patients)

Imaging of the coronary anatomy

- The imaging of the coronary anatomy is the most importat diagnostics method in evaluation of acute coronary syndrome

- The gold standard of patients with ACS is conventional invasive coronary angiography

Treatment of MI

- while STEMI is an urgent situation with turbulent symptomatology, NSTEMI may have symptoms much milder and above its immediate prognosis is better
- Pts. should stay on coronary care unit - 2-3 days, than standard cardiology department
- the total length of hospitalization is around 1 week
- even after leaving the CCU patients are able to move around the room and in the following days rehabilitate and before discharge they are able to walk up the stairs
- return to job possible approximately one month after the onset of the symptoms

Treatment of STEMI

- Open the occluded artery **as soon as possible** to restore blood flow for the heart

"Time is muscle"

- Check for complication of myocardial infarction and treat them:
  - arrhythmia
  - heart failure
  - bleeding

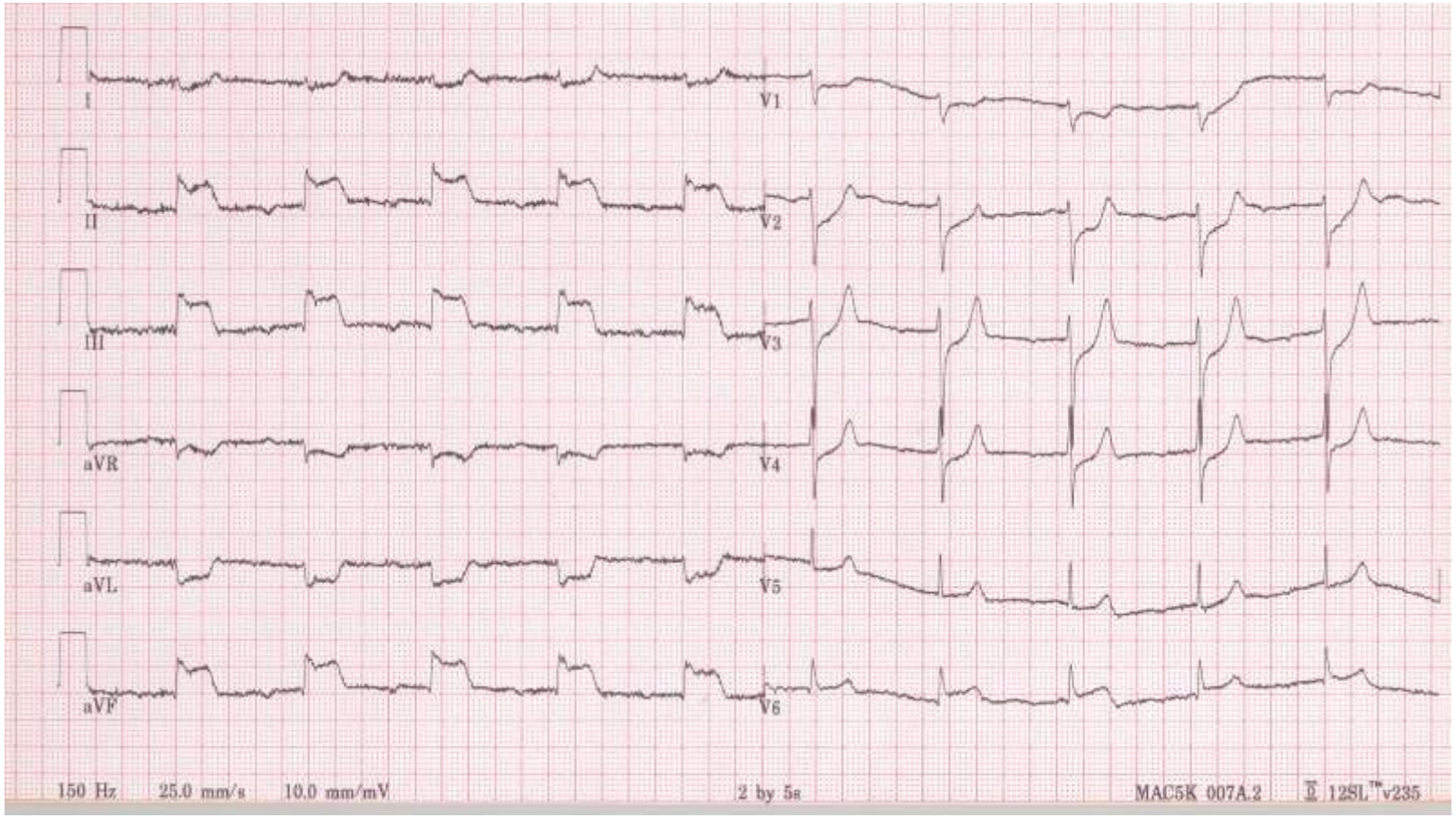
Pre-hospital management

- Betablockers - tachycardia, hypertension
  - Metoprolol - dose 25-50mg oral or 2 mg i.v.
- ACE inhibitors - hypertension
  - Perindopril - dose 5 mg oral
- Diuretic - heart failure
  - Furosemide 20 - 40mg i.v.
- Anti-arrhythmic drugs -no prophylaxis
  - Mesocain 1% 10 mL i.v.
  - Amiodarone 150 mg i.v. bolus

Hospital and discharge therapy

- Antiplatelet therapy
  - Acetylosalicid acid - dose 100 mg p.o.
  - Clopidogrel 75mg or ticagrelor 90mg twice a day or prasugrel 10mg
- Statins - benefit for all patients with IM
  - Atorvastatin 40 - 80mg, rosuvastatin 20 - 40mg
- ACE inhibitors - benefit for all patient with IM, more expressed in left ventricular dysfunction
  - perindopril - dose 5-10 mg oral
- Betablockers - 1 - 3 years after MI, longer for pts. With left ventricular dysfunction, tachyarrhythmia

Case report - 1



57-old female smoker, family history of CAD, pain 6 hours, nausea

Complications of MI

- **Early complications**
  - Heart failure, cardiogenic shock
  - Mechanical complications :

    - rupture of free wall of left ventricle

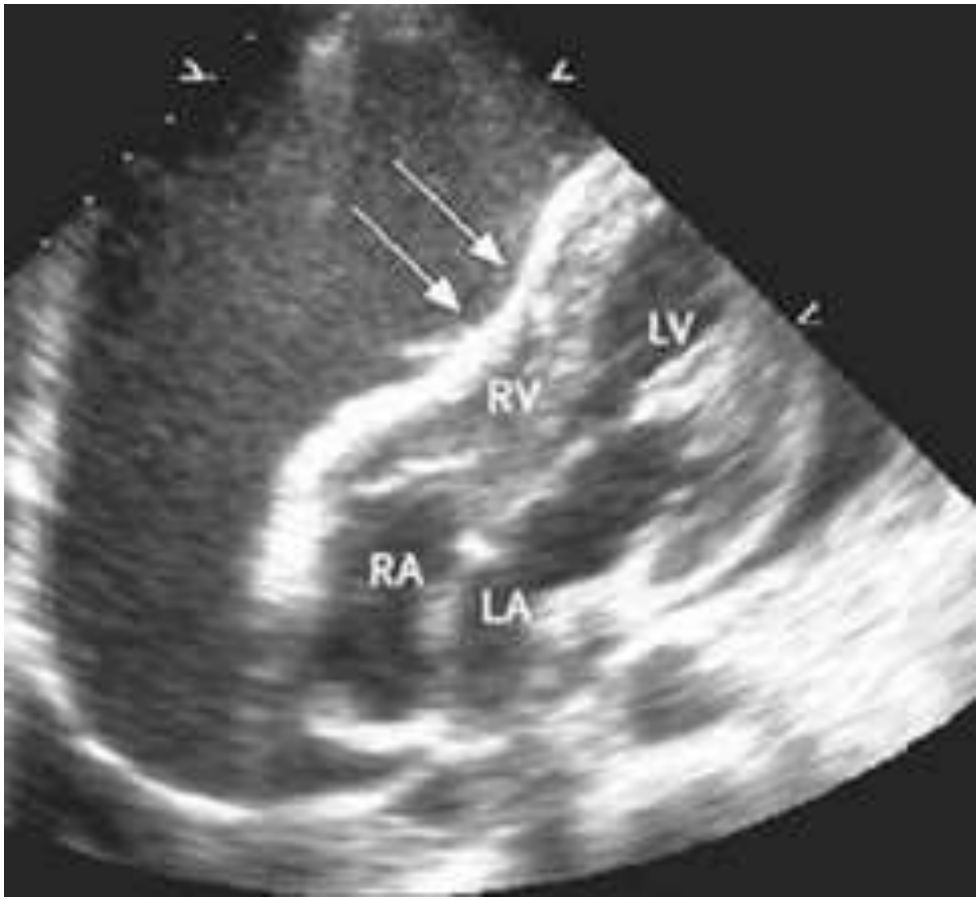
    - ventricular septal defect

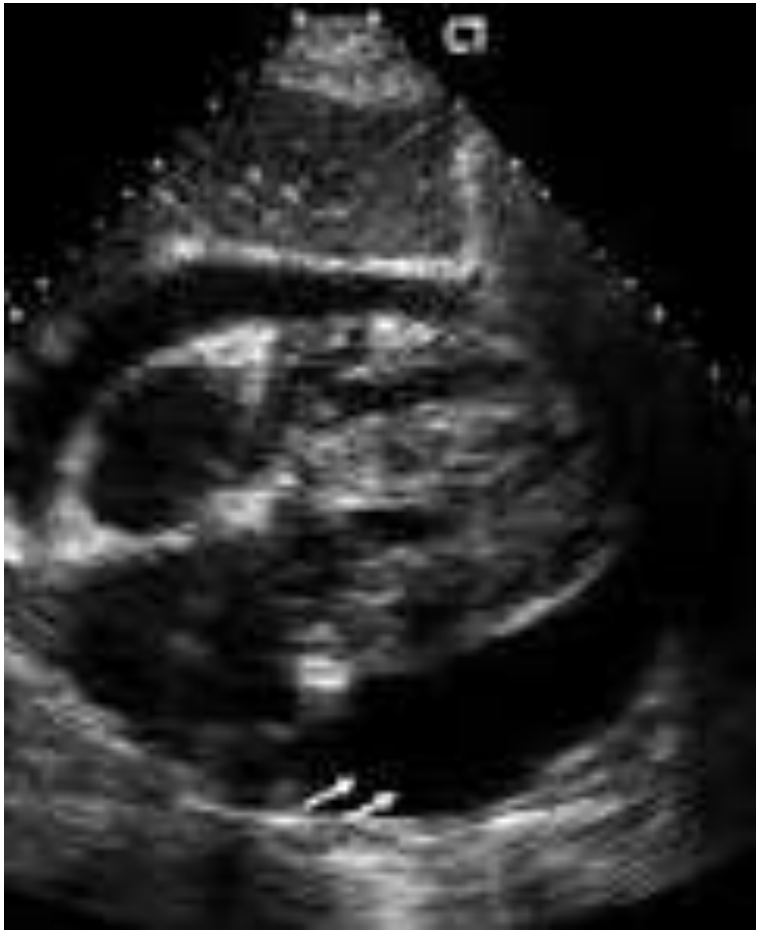
    - acute mitral regurgitation
  - Arrhythmia

    - ventricular (up to 48 h)

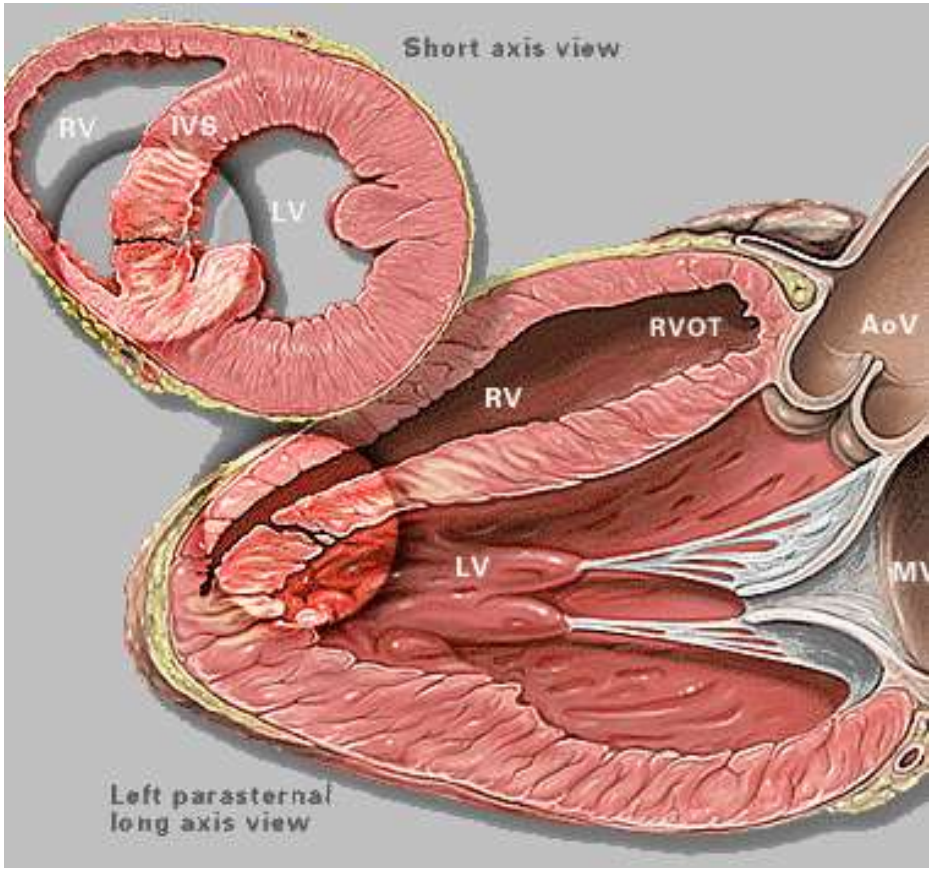
    - bradycardia (9-25% of pts)
- **Late complications**
  - pericarditis
  - Aneurysm of left or right ventricle

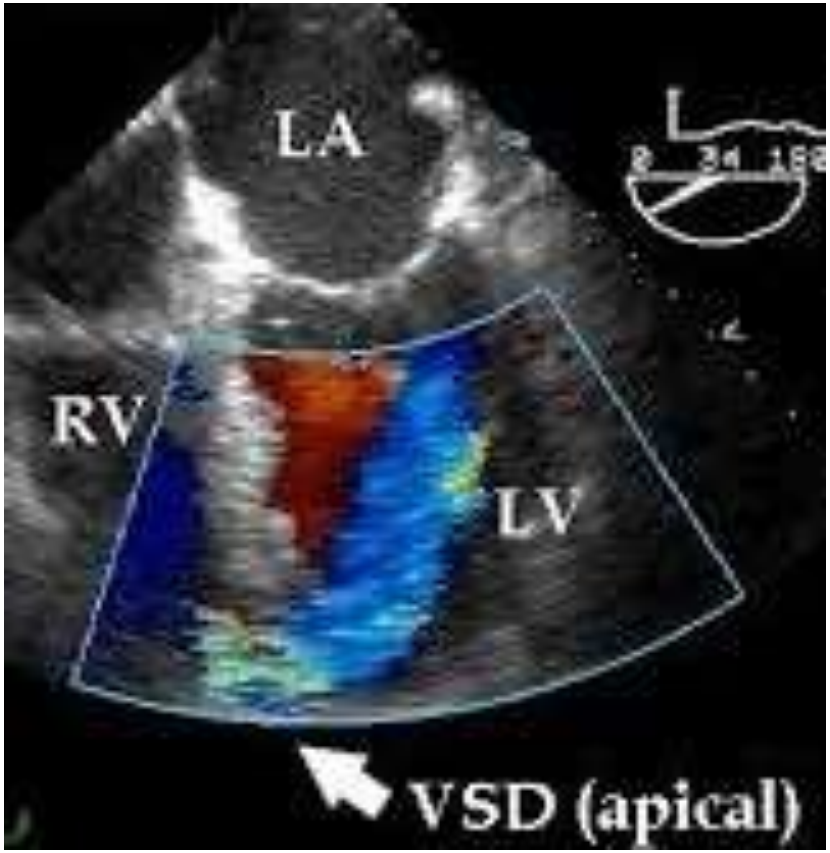
Tamponade



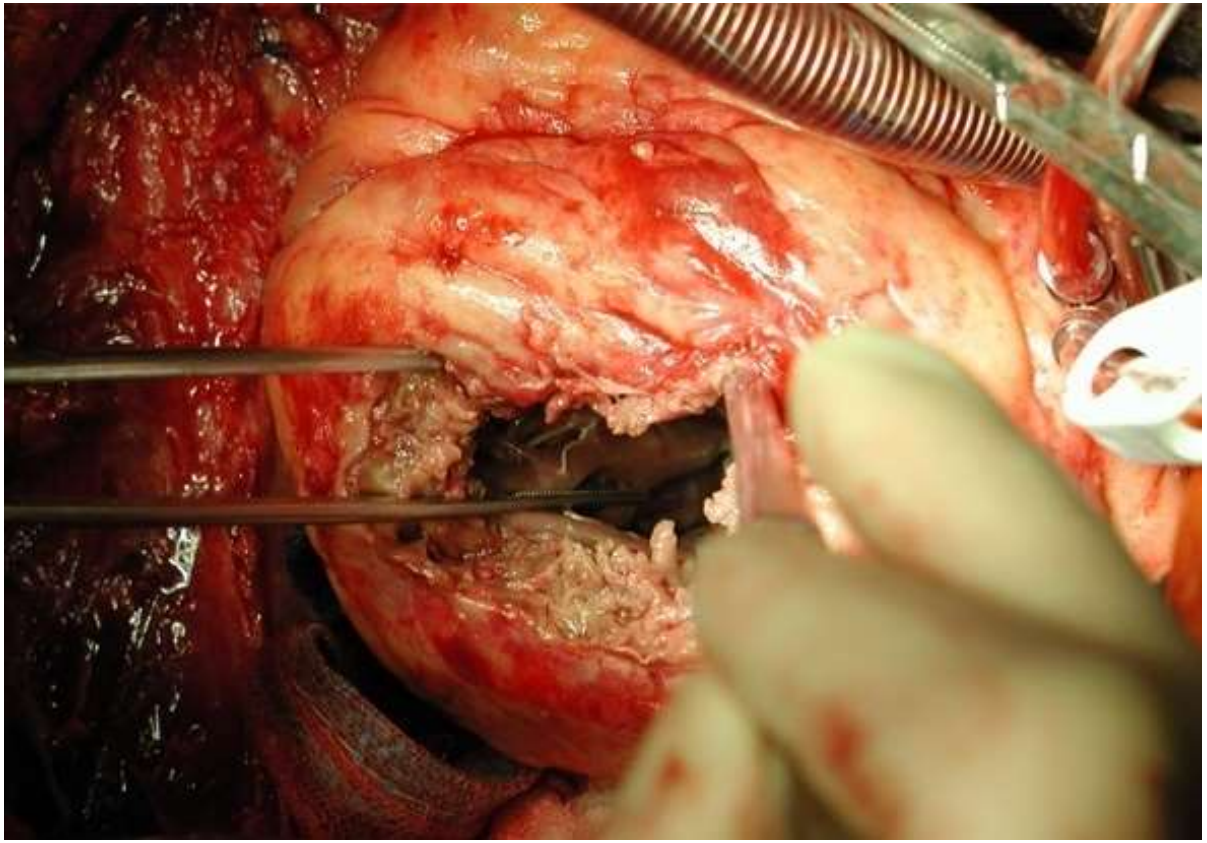


VSD


Short axis view
RV
IVS
LV
RVOT
AoV
RV
LV
Left parasternal
long axis view


LA
RV
LV
VSD (apical)

VSD



Aneurysm