# Pericardial effusion with emphasis on the electrocardiographic aspects

By Andrés Ricardo Pérez-Riera MDPhD

# ECG In pericarditis Pericardial effusion

## Pericardium

The pericardium is a double sheet made up by two layers of not so distensible fibrous tissue that wraps the heart.
The internal or visceral layer is adhered to the heart.
The external or parietal layer is wrapped by the visceral one. Between both there is a space with a small amount of serofibrinous liquid (≈20 to 50 ml).
The parietal layer fixes the heart in its place within the chest and prevents direct contact between the organ and neighboring structures.

## Functions of the pericardium

The pericardium has three main functions: mechanical, membranous and ligamentous.
**Mechanical function:** it restricts cardiac dilatation increasing the efficiency of the heart, maintaining ventricular compliance and distributing hydrostatic forces. Additionally, it creates a closed chamber with subatmospheric pressure, aiding atrial filling and reducing parietal transmural pressures.

**Membranous function:** it protects the heart, reducing its external friction and acting as a barrier against propagation of infections and neoplasia's.

**Ligamentous function:** it anatomically fixes the heart, preventing the latter from balancing.

**Other functions**: Barrier against infections; barrier against dissemination of neoplasias; preventing excessive movements of the organ; preventing direct contact of the heart with neighboring structures; conditioning less friction between the heart and other organs; allowing diastolic distention of the chambers due to negative atmospheric pressure.

# Pericarditis

**Concept of pericarditis:** syndrome caused by inflammation of the pericardium, a sack made up by two sheets (parietal and visceral) that wrap the heart and the great vessels.

## Etiological classification of pericarditis

- **Idiopathic (unknown):** 26-86% of cases. Difficult to differentiate from virous.
- **Infectious**
  - **Virous**: (1-10% of cases). Etiologies: Coxsackie, enterovirus, influenza A and B, HIV, Echo, varicella/zoster, parotiditis, measles, hepatitis, Epstein-Barr, herpes simplex type 1, parainfluenza 2 and sintitial respiratory virus.
  - Bacterian: Streptococcus pneumoniae, Staphylococcus, Proteus, Escherichia coli, Pseudomonas, Klebsiella, Salmonella, Shigella, Neisseria meningitidis, and Haemophilus influenzae. Anaerobic: present in almost 40% of children with AP.
  - Tuberculosis: 4% of cases.
  - Mycotic: histoplasmosis, blastomycetes, coccidioides, aspergillosis and candida.
  - By Rickesia.
  - Entamoeba.
  - Echinococcus.
  - Toxoplasma.
- **Collagenotic**
  - Lupic: ≈25% of patients with SLE develop AP. They rarely evolve into tamponade and constrictive pericarditis.
  - Scleroderma;
  - Rheumatoid arthritis: 11-50% of patients with rheumatoid arthritis develop silent AP. The diagnosis is made in only 2-6% of cases.
  - Dermatomyositis;
  - Other collagenopathies.

- **Acute post-infarction -** ≈24 h after transmural AMI, fibrinous pericardial exudate arises. Before the thrombolytic era, nearly 1/5 of patients with AMI developed AP. After adopting the thrombolytic therapy and angioplasty, this incidence dropped 5-8%. Effusions may occur, but they rarely evolve into tamponade. Pericardial involvement does not contraindicate thrombolytic or anticoagulant therapy 3 to 5 days after transmural infarction.
- **Dressler's syndrome:** it appears at the 2º or 3º week after AMI Association of AP + Dressler < 4%.
- **Neoplastic** - Primary or metastatic tumor: they are responsible for 5-17% of cases. Dyspnea is the most common symptom
- **Metabolic**
  - Uremic;
  - Gout;
  - Hypothyroidism: ≈ 4% of cases of AP. Great effusions may develop. Evolution into tamponade is rare.
- **Actinic:** by radiation: pericarditis is the most frequent manifestation of post-radiotherapy cardiac toxicity. It may evolve with pericardial effusion or chronic constrictive pericarditis.
- **Chylopericardium**
- **Traumatic:** Penetrating; closed; postpericardiotomy syndrome: similar to Dressler's syndrome, however with incidence of 10-40% ≈1% develops tamponade; esophageal rupture; pancreatico-pericardial fistula; epicardial ablation; epicardial pacemaker.
- **By hypersensitivity:** Serum sickness; reaction to drugs: penicillin, doxorubicin, cyclophosphamide, procainamide, hydralazine, methyldopa, isoniazid, mesalazine and reserpine; and immunopathies.

## Evolutionary classification of pericarditis

- **Acute:** <6 weeks (fibrinous with pericardial effusion).
- **Sub-acute:** 6 weeks to 6 months (constrictive and pericardial effusion).
- **Chronic:** >6 months (it divides into constrictive, pericardial effusion and adhesive without constriction).

## Conditioning factors of ECG manifestations in pericarditis

Three causes are acknowledged as responsible for ECG modifications:

- Accumulation of liquid between the two sheets: responsible for QRS complexes low voltage and electrical alternance.
- Lesion of epicardial region of myocardium by liquid or fibrin pressure: responsible for ST segment elevation, depression in $V_1$ and aVR and PR segment depression (STa) and elevation in aVR.
- Superficial myocarditis (epicarditis): changes in T wave.

## ECG in pericardial diseases (acute pericarditis)

Altered in 90% of cases. There are 4 stages or phases described on ECG, present in only 50% of cases:

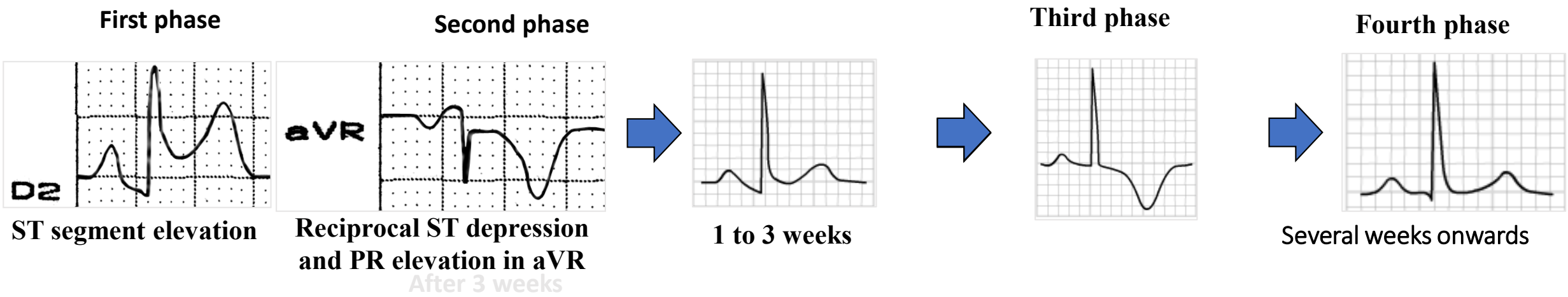
**First phase:** Frequent sinus tachycardia in acute pericarditis due to pain and/or pericardial effusion.

Widespread concave ST segment elevation (<5 mm) of superior concavity. It is observed only two hours before chest pain and it lasts for several days. ST segment changes are *extensive and not too intense,* normally noticeable in several leads simultaneously, excluding $V_1$. Occasionally, reciprocal alterations are observed in aVR. PR depression throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6).

**Second phase:** ST segment returns to baseline and generalized flat T wave in 7 to 21 days (pseudonormalization: transition).

**Third phase:** inversion of T wave, with no formation of Q wave after 3 weeks.

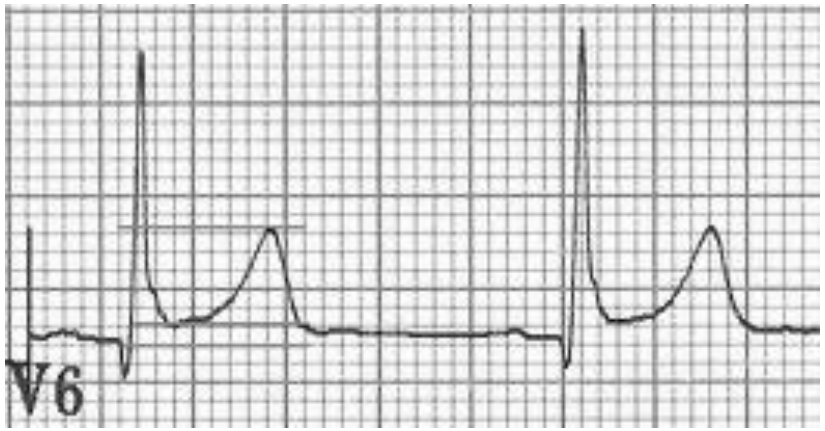
**Fourth phase:** ECG normalization with gradual reversion of T wave inversion.



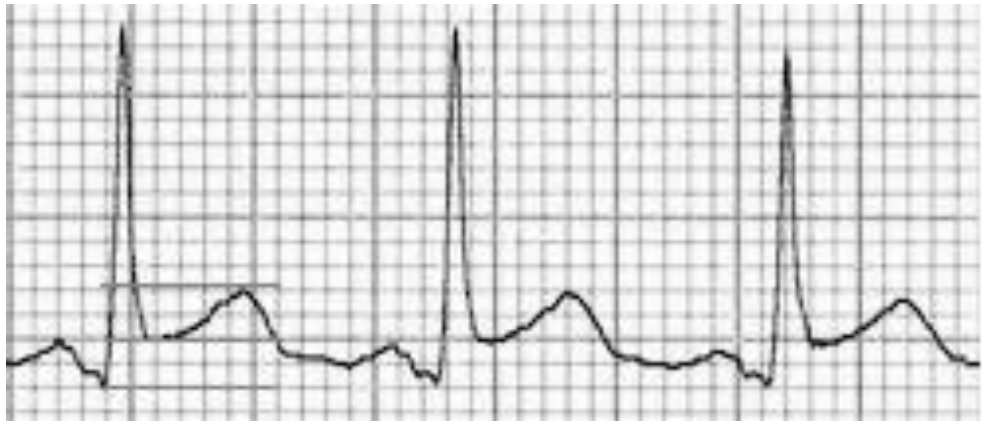

Observation: Less than 50% of patients progress through all 4 classical stages and evolution of changes may not follow this typical pattern.

# Differential diagnosis between pericarditis and benign early repolarization

Pericarditis can be difficult to differentiate from BER as both conditions are associated with concave ST elevation. One useful trick to distinguish between these two entities is to look at the **ST segment / T wave ratio:** The vertical height of the ST segment elevation (from the end of the PR segment to the J point) is measured and compared to the amplitude of the T wave in V6; a ratio of >0.25 suggests pericarditis; a ratio of <0.25 suggests BER



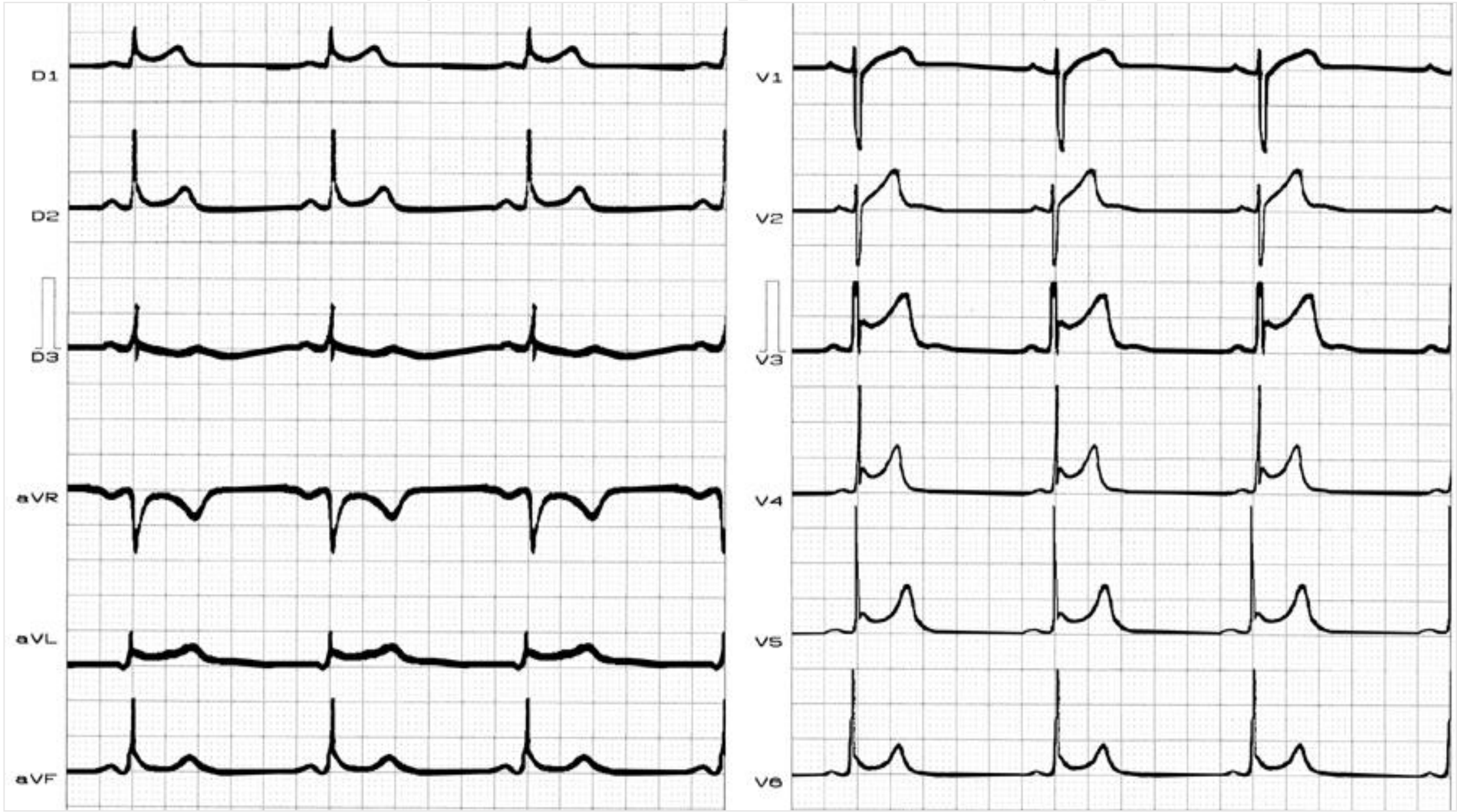

**Benign Early Repolarization**




**Pericarditis**

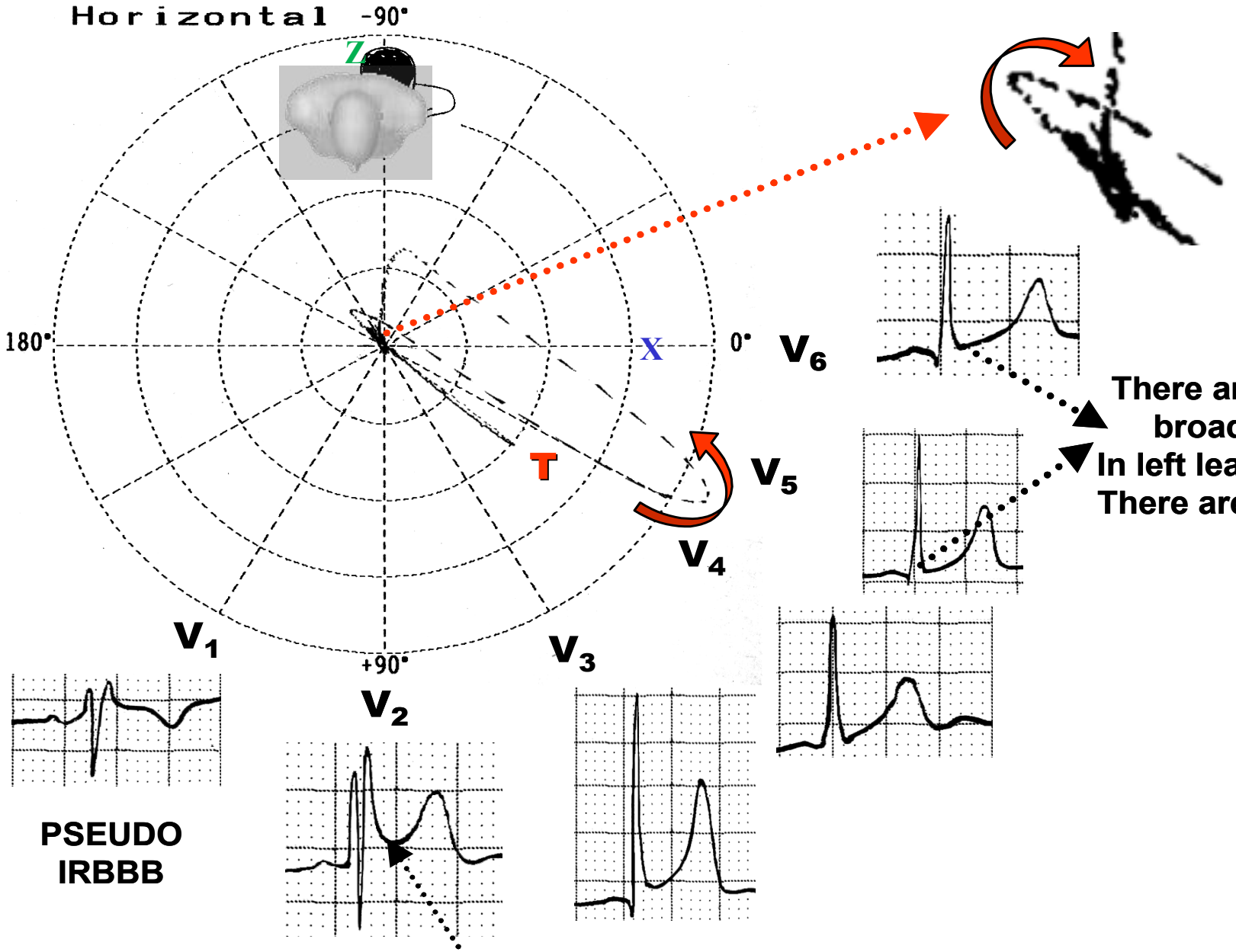
| Benign Early Repolarization | Pericarditis |
|---|---|
| ST segment height = 1 mm | Generalized ST segment elevation. Height = 2 mm |
| T wave height = 6 mm | T wave height = 4 mm |
| ST / T wave ratio = 0.16 | ST / T wave ratio = 0.5 |
| The ST / T wave ratio < 0.25 followed by prominent T waves | Universal elevation (all of them). There may be reciprocal depression in opposite leads. |
| The presence of a notched or irregular J point: the so-called “fish hook” pattern. This is often best seen in lead V4. | Absence of “fish hook” appearance in V4 |
| ST elevation limited to the precordial leads. Reciprocal depression only in aVR. | Normal T wave amplitud. It decreases amplitude in hours. Lower voltage. Only increased in early phase. |
| Absence of PR depression | Presence of PR depression |
| ECG changes usually stable over time (i.e. non-progressive) | ECG changes evolve slowly over time |
| **Response to strain**: Frequent return of ST to baseline. T wave may normalize. | ST segment elevation is not modified. |
| **Hyperventilation:** T polarity may be modified. | T polarity is not modified. |
| Frequent bradycardia. | Frequent tachycardia. |
| **Presentation**: Stable. | Transitory. |
| **Clinic**: Asymptomatic. | Marked alteration. |
| **Age range**: 20 to 40 years old. | 40 or more. |

# Differential diagnosis between acute pericarditis and early repolarization




Sinus bradycardia, upwardly concave ST segment elevation in middle and left precordial leads and notch in terminal portion of QRS complex (J point). **Conclusion:** early repolarization

# Differential diagnosis of acute pericarditis early repolarization



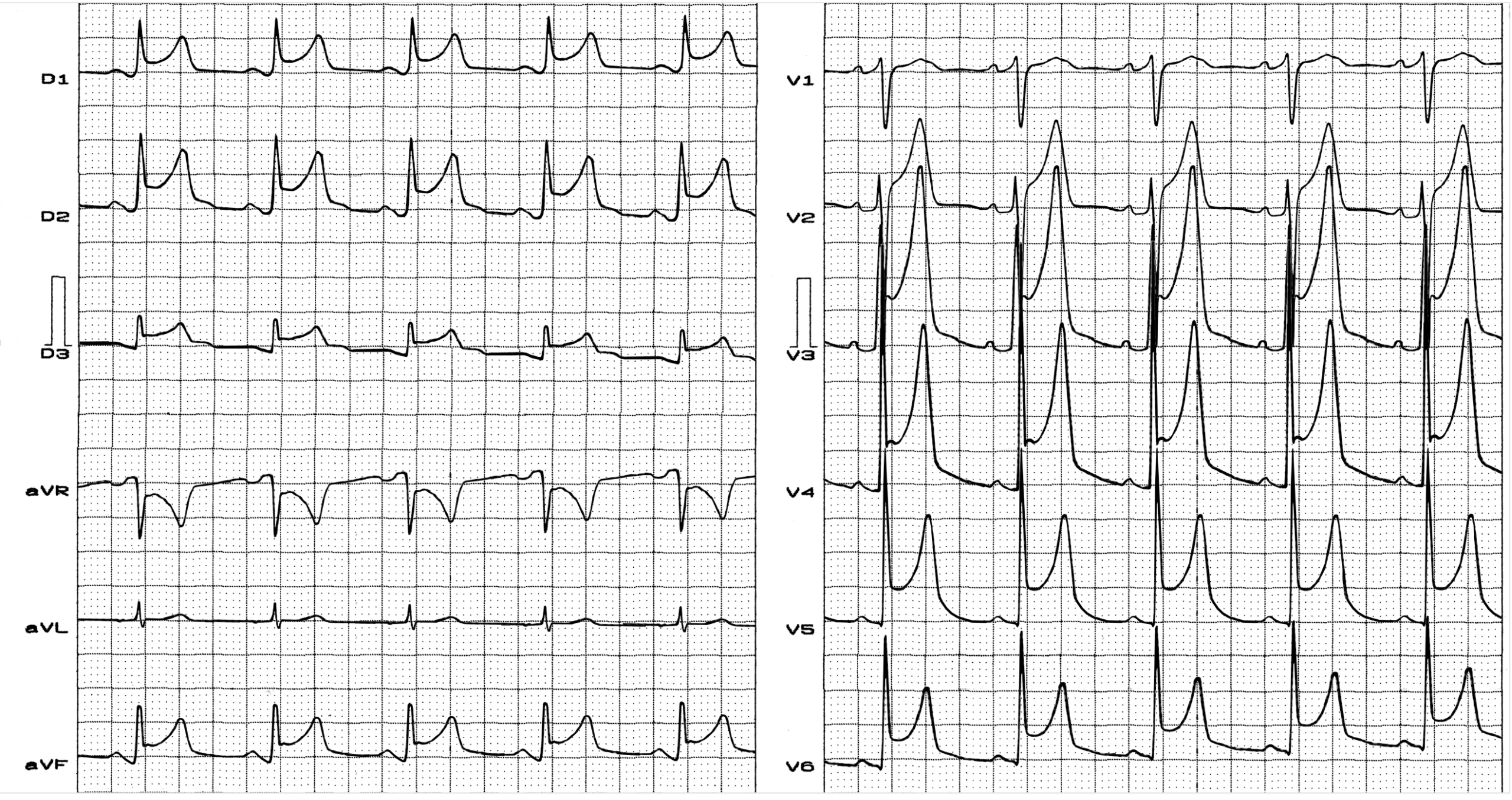
# Differential diagnosis between coronary artery disease with ST segment elevation and pericarditis

| | Coronary artery disease (STEMI) | Pericarditis |
|---|---|---|
| Number of involved leads | Less (segmentary) | Major (diffuse) and extensive |
| Intensity of the phenomena | Major | Lesser |
| Reciprocal effect or mirror image | Frequently present | Absent, except aVR |
| ST segment elevation convex upward or horizontal | Frequently. In the initial phases is possibly concave upward. | Absent, always concave to the top |
| PR segment depression | Possible if atrial infarction | Only in viral pericarditis |
| STSE III > II | Characteristic when present | Absent |
| Pleuritic positional pain | Rare, but possible | Characteristic |

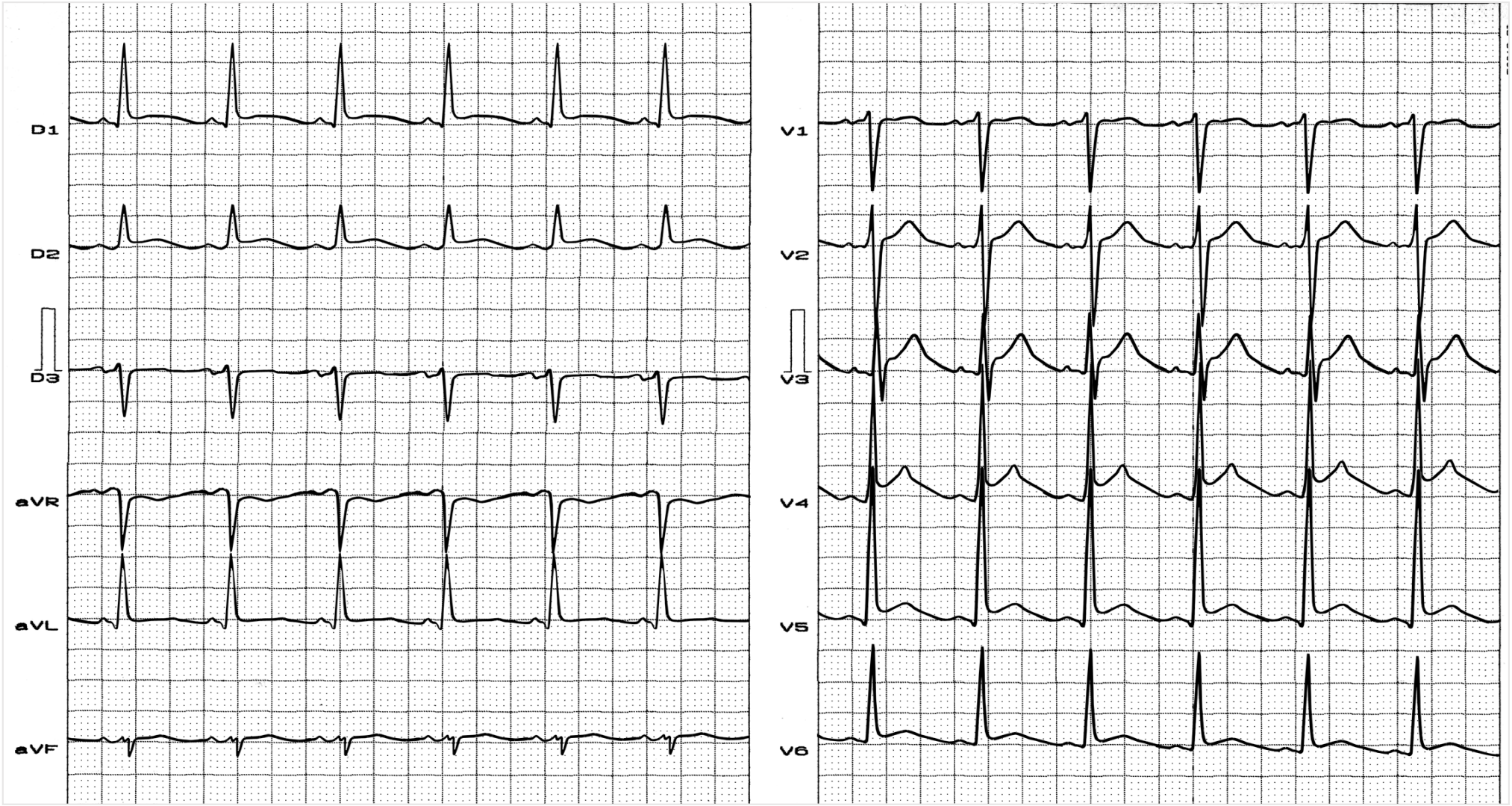
Steps to distinguish pericarditis from ST segment elevation Myocardial Infarction (STEMI):

1. Is there ST depression in a lead other than AVR or V1? *This is STEMI*
2. Is there convex up or horizontal ST elevation? *This is STEMI*
3. Is there ST elevation greater in III than II? *This is STEMI*
4. Now look for PR depression in multiple leads… this suggests pericarditis (especially if there is a friction rub!)

**Typical ECG of acute pericarditis in the first phase with diffuse elevation of upwardly concave STSE followed**



Male patient, 59 years old



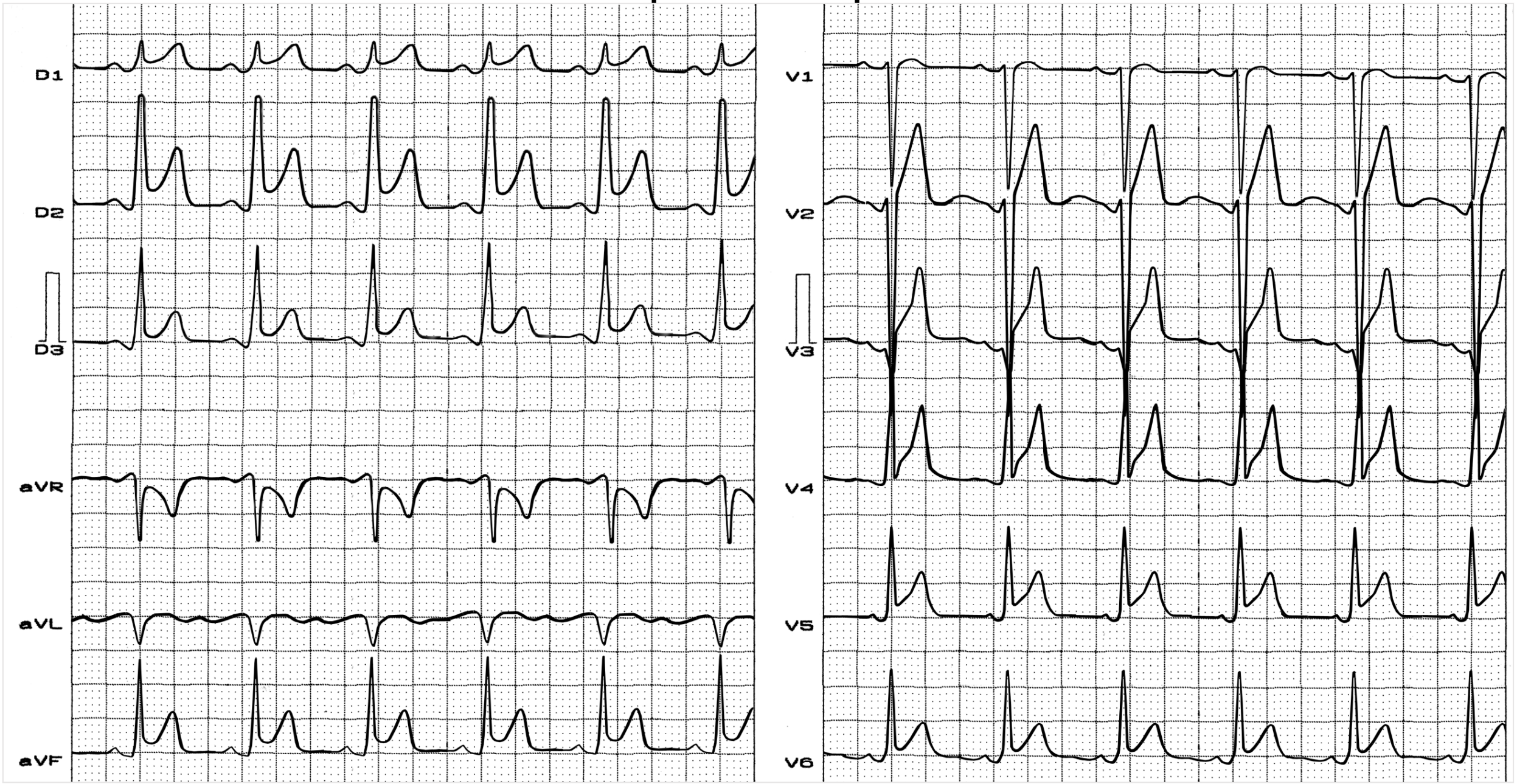

**Complaint:** chest pain, odontalgia for 1 week and fever

**Physical examination:** inflammatory signs of right submandibular region, fever (39º), negative auscultation. Absence of pericardial friction.

**Diagnosis:** Ludwig's angina (cellulitis of the mouth floor and periodontal abscess) and descending necrotizing mediastinitis. Death by septicemia in 48 hours.

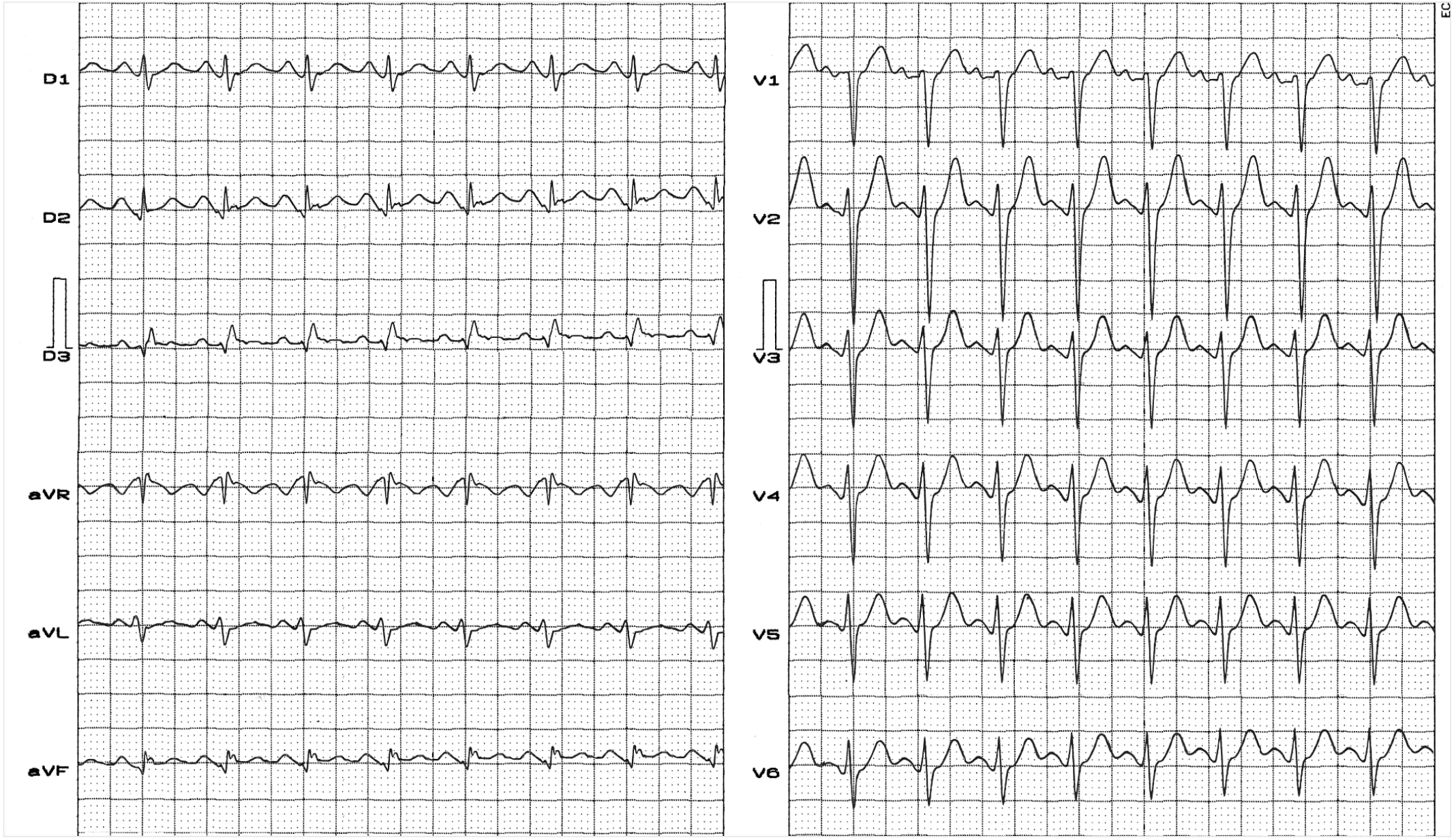
**ECG diagnosis**: ST segment elevation with superior concavity from V3 to V6 / I, II and aVL of 0.5 mV.

# Acute pericarditis in phase 1



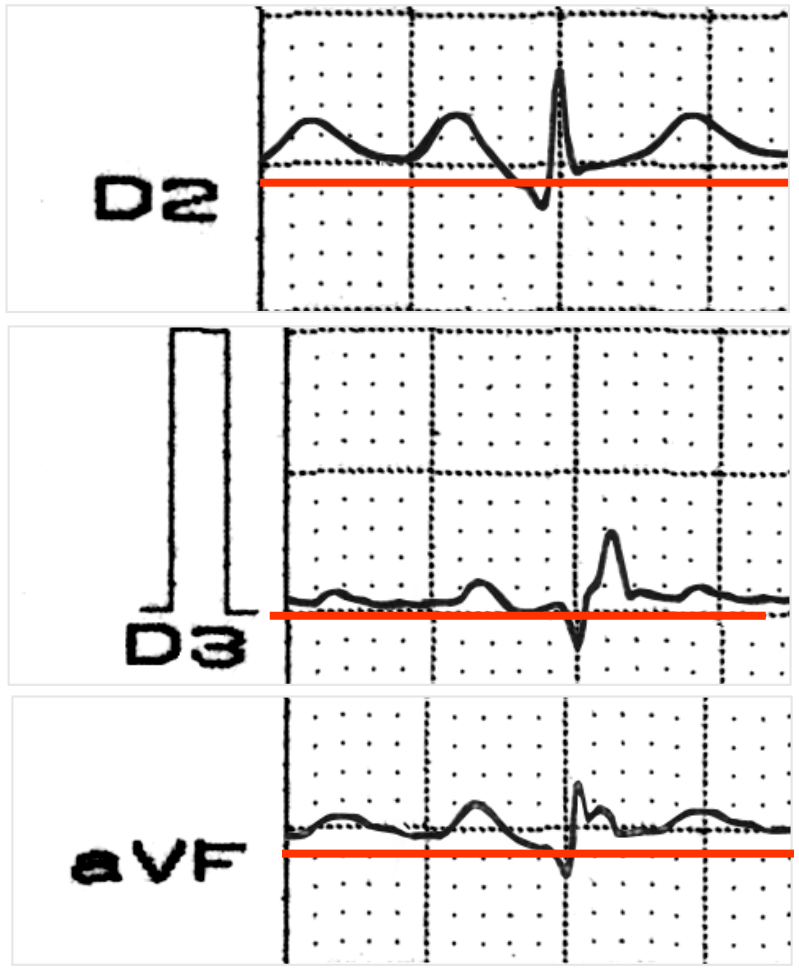

**ST segment elevation with superior concavity from V3 to V6 / I, II and aVL of 0.5 mV. ST segment depression in aVR.**

**Name:** RBS; **Sex:** Male; **Age:** 30 y/o; **Ehnic group:** Caucasian; **Weight:** 73 Kg; **Height:** 1.71 m; **Date:** 09/06/2005.



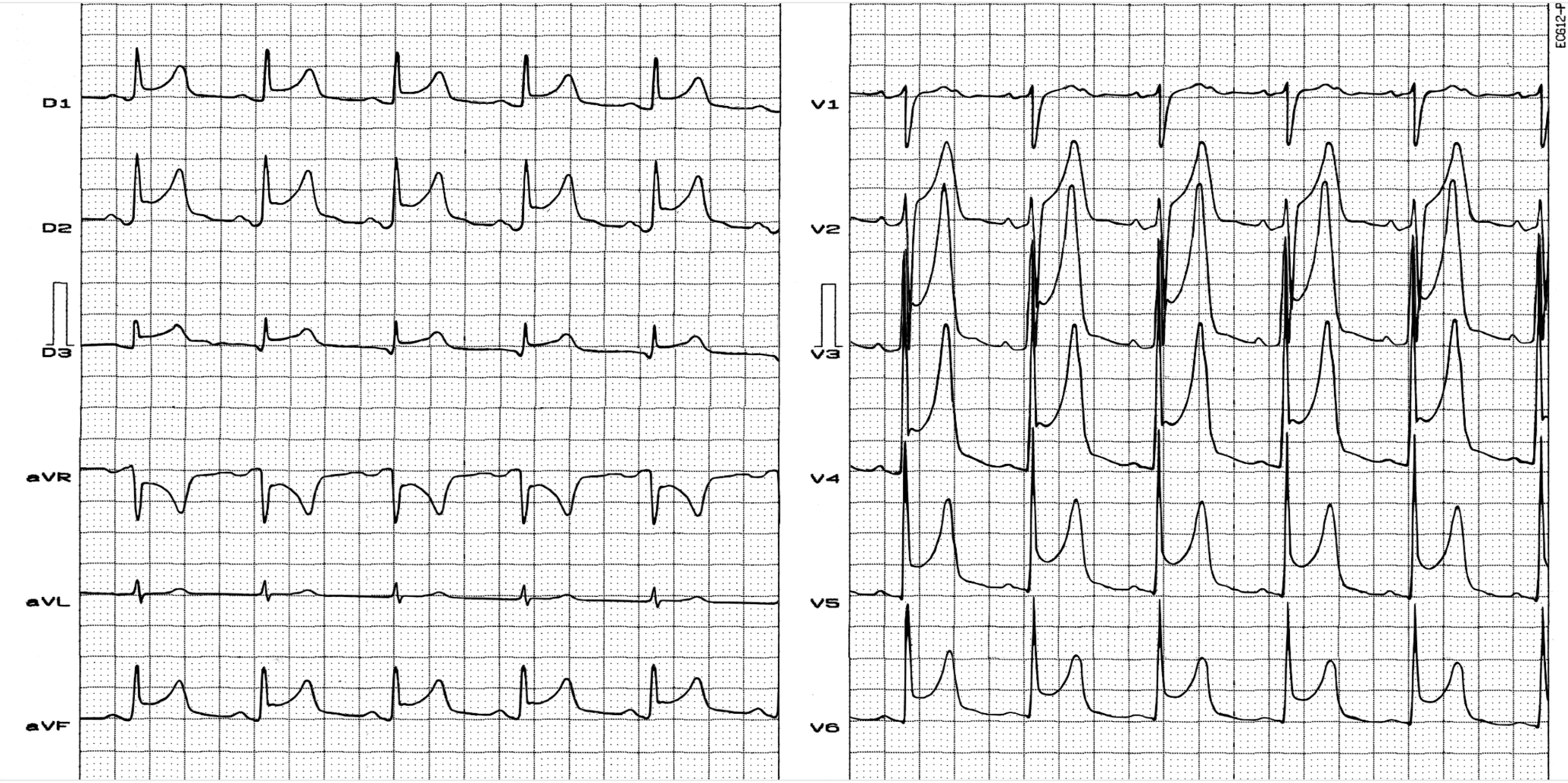

ECG that displays typical modifications in acute phase of pericarditis. Conducted 6 hours after the pain started. PR segment depression in II, III and aVF and from V1 through V6.

# Inferior leads of the previous tracing




**PR segment depression compared to ST segment in the same beat, observed in inferior wall. The red lines show PR segment depression compared to ST segment in the same beat.**

# Acute pericarditis




**Acute pericarditis with significant and diffuse ST segment elevation.**

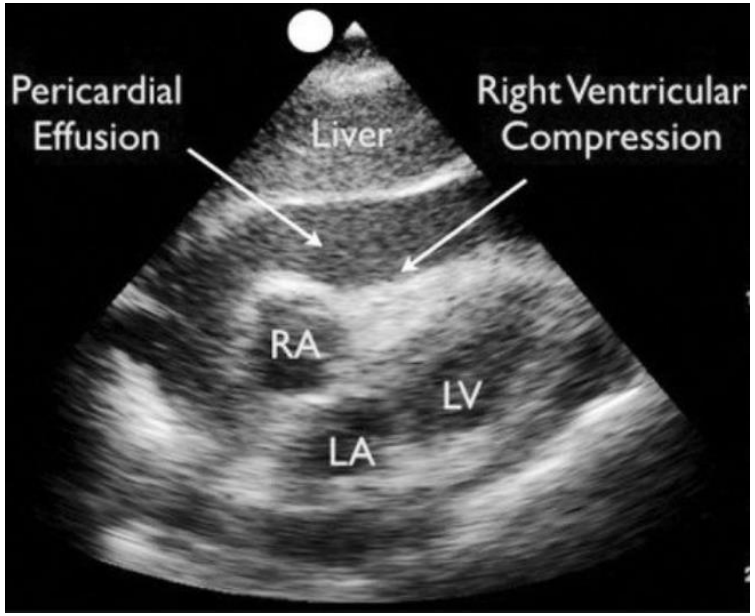
# ECG in cardiac tamponade Massive pericardial Efussion

Cardiac tamponade occurs when fluid accumulation in the finite serous pericardial space causes an increase in pressure, with subsequent cardiac compression and hemodynamic compromise. It is characterized by the electocardiographic triad: Low QRS voltage, Tachycardia and Electrical alternansLow voltage, ST segment elevation and frequent electrical alternance, considered pathognomonic of cardiac tamponade, and is characterized by changes in P wave, QRS complex and T wave voltages from beat to beat. The most frequent type of alternance is the one that affects only QRS. When it includes the three waves is called total alternance, observed in cases of cardiac tamponade, characterized by Beck's triad Jugular venous distension, Hypotension and Muffled noises.

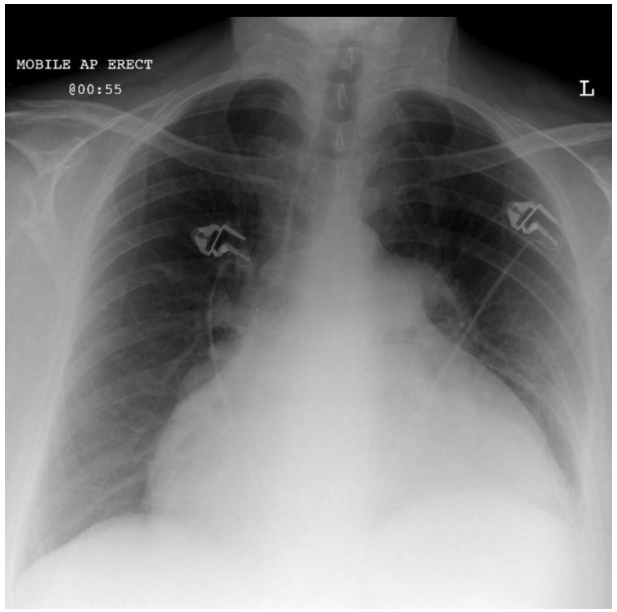
Electrical alternance is the result from a heart that wavers in an extensive effusion. Electrical alternans occurs when consecutive, normally-conducted QRS complexes alternate in height.

The heart swings backwards and forwards within a large fluid-filled pericardium.

Patients with this ECG pattern need to be immediately assessed for clinical and echocardiographic evidence of tamponade.

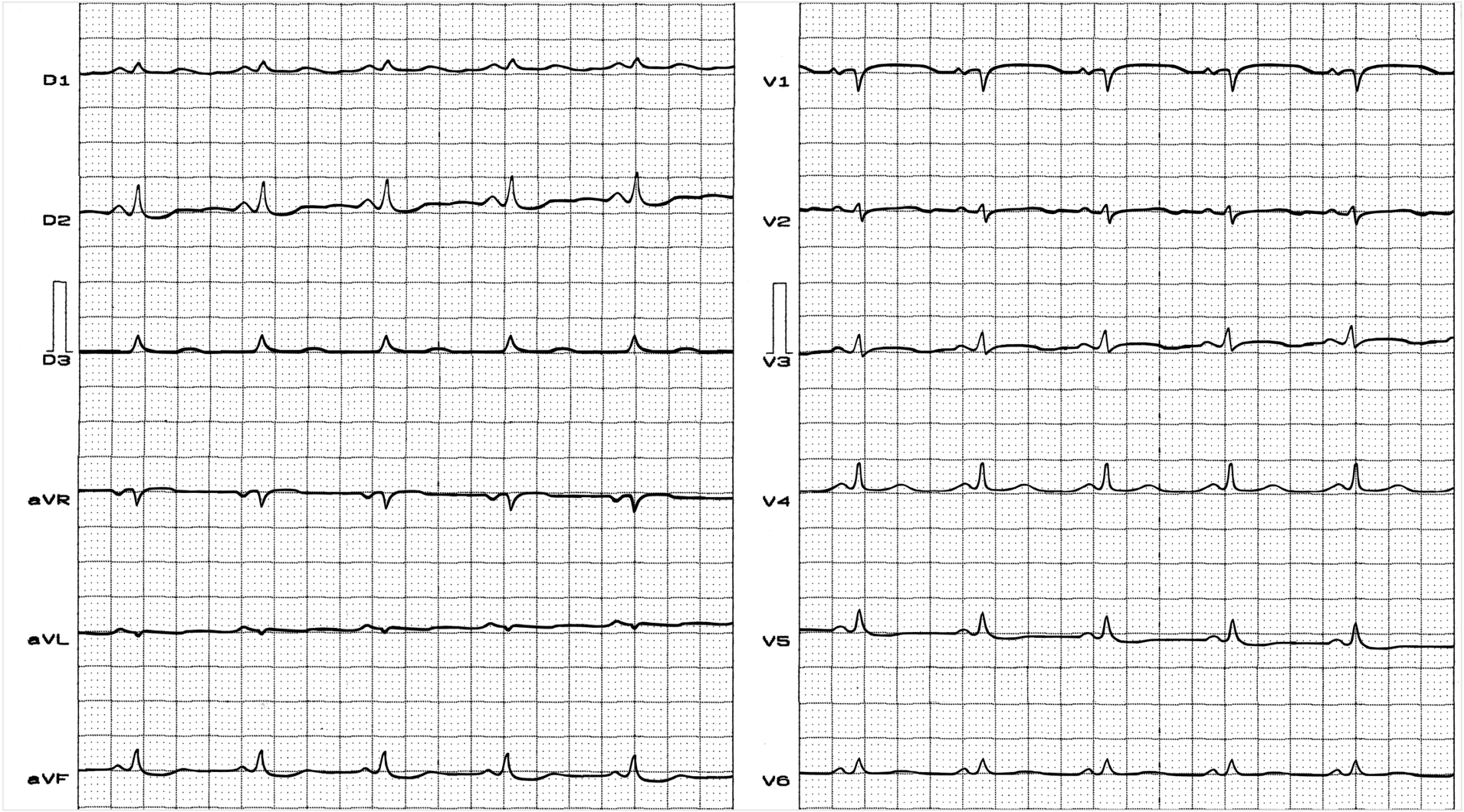



In this image, the effusion is larger, and has completely squashed the right ventricle so that it cannot fill and therefore can not pump blood through the lungs to the left ventricle and from there around the body.




Front chest x-ray demonstrates marked enlargement of the cardiac silhuete. This was due to pericardial effusion and is a good example of the water bottle sign. This refers to the shape of the cardiac silhouette on erect frontal chest x-rays in patients who have a very large pericardial effusion.

# ECG Constrictive pericarditis




**Constrictive pericarditis, low voltage of QRS complexes in all leads (this sign is present in more than 50% of cases). Diffuse alterations of ventricular repolarization: flat T waves.**

# Constrictive pericarditis or Pericardial Constriction: Electrocardiographic characterization

**Definition:**

Constrictive pericarditis refers to an abnormal scarring and loss of elasticity of the pericardium, resulting in impaired ventricular filling and decreased cardiac output.

**Etiology:**

The frequency of different causes of constrictive pericarditis depends on the population and geography in question. In developed countries, cardiac surgery and idiopathic constriction are the leading cause, while in certain developing countries tuberculous remains the number one etiology.

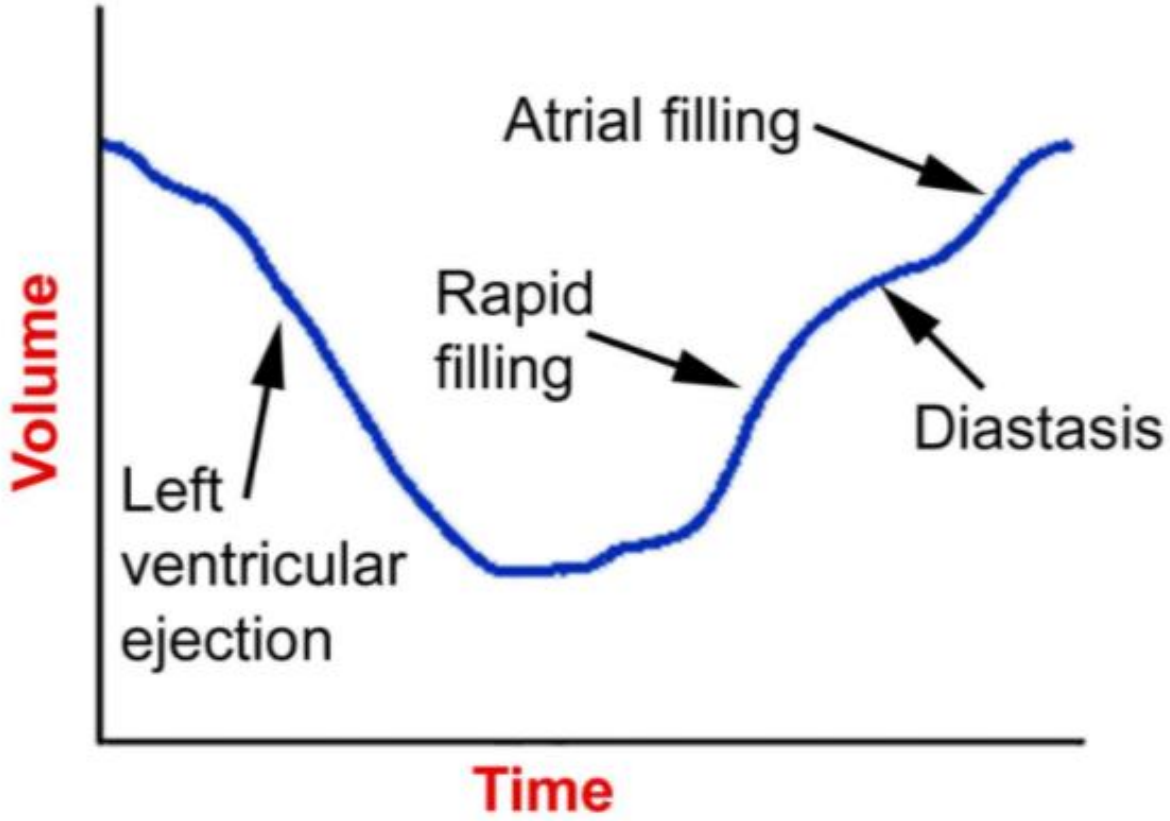
**Pathophysiology**

The normal pericardium is composed of 2 layers: the tough fibrous parietal pericardium and the smooth visceral pericardium. Usually, approximately 50 mL of fluid (plasma ultrafiltrate) is present in the intrapericardial space to minimize friction during cardiac motion. Acute and subacute forms of pericarditis (which may or may not be symptomatic) may deposit fibrin, which, in turn, can evoke a pericardial effusion. This often leads to pericardial organization, chronic fibrotic scarring, and calcification, most often involving the parietal pericardium. In constrictive pericarditis, the easily distensible, thin parietal and visceral pericardial linings become inflamed, thickened, and fused. Because of these changes, the potential space between the linings is obliterated, and the ventricle loses distensibility. Venous return to the heart becomes limited, and ventricular filling is reduced, with associated inability to maintain adequate preload. Filling pressures of the heart tend to become equal in both the ventricles and the atria.

Since the myocardium is unaffected, early ventricular filling during the first third of diastole is unimpeded. After early diastole, the stiff pericardium affects flow and hemodynamics. Accordingly, the ventricular pressure initially decreases rapidly (producing a steep *y* descent on right atrial pressure waveform tracings) and then increases abruptly to a level that is sustained until systole (the “dip-and-plateau waveform” or “square root sign” seen on right or left ventricular pressure waveform tracings).

The preservation of myocardial function in early diastole aids in distinguishing constrictive pericarditis from restrictive cardiomyopathy. Systolic function is rarely affected until late in the course of the disease, presumably secondary to infiltrative processes that affect the myocardium, atrophy, or scarring or fibrosis of the myocardium from the overlying adjacent pericardial disease.

Experimental models indicated that a change in volume-elasticity curves (see the image below) was the fundamental pathophysiologic change associated with the disease. During development of the constriction, right and left ventricular diastolic pressure increased, and stroke volume decreased. A small increase in volume resulted in a considerable increase in end-diastolic pressure.




**Left ventricular volume curve obtained with acoustic quantification. Arrows indicate phases of cardiac cycle**

## Electrocardiographic features

**Rhythm:** frequent sinus rhythm loss. Presence of AF in 25% of cases. Presence of atrial flutter in 8% of the cases.
**P wave:** notches that suggest intraatrial block, low voltage and signs of atrial enlargement.
**SÂQRS:** not modified by positional changes of the body.
**QRS complex:** low voltage in more than 55% of cases.
**Q wave:** possible pseudo necrosis.
**T wave:** inverted or flat, or with possible notch in all cases. The more negative they are, a greater adherence of the pericardium to the underlying myocardium is suggested.