ANGINA PECTORIS

MYOCARDIAL INFARCTION

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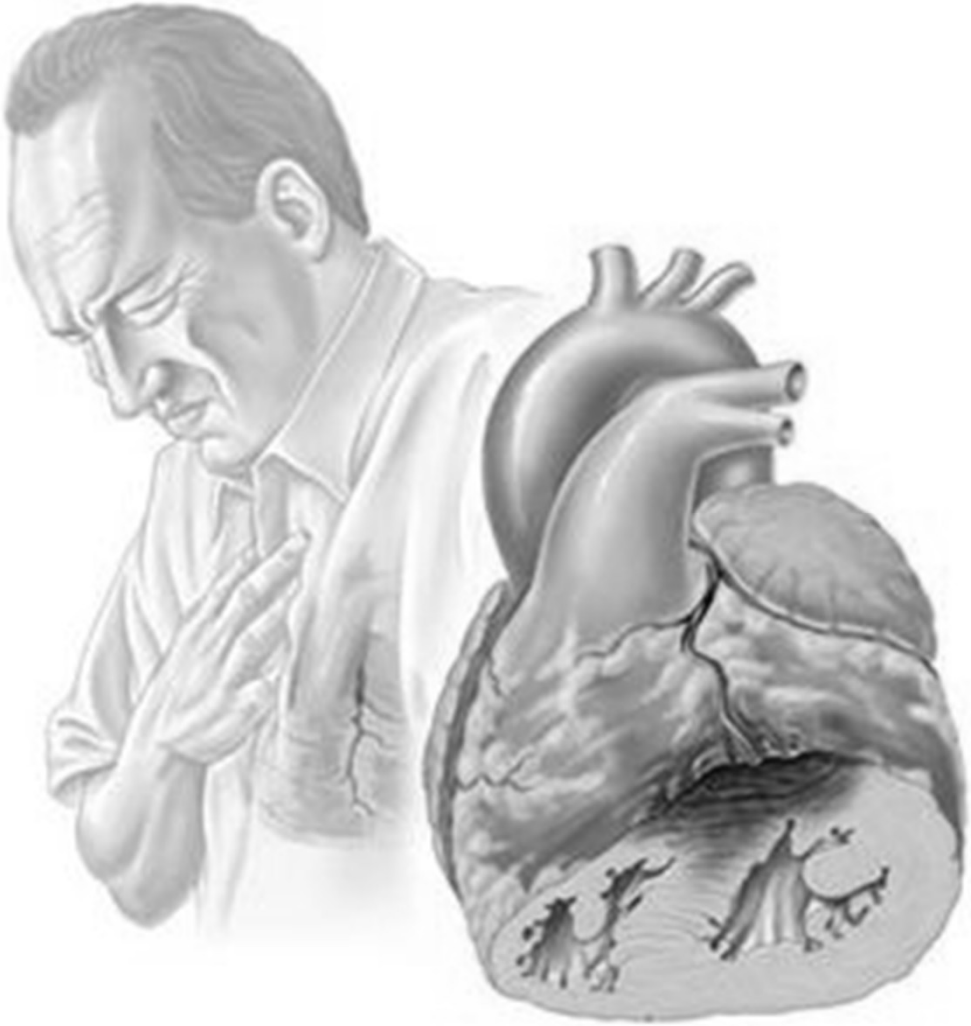
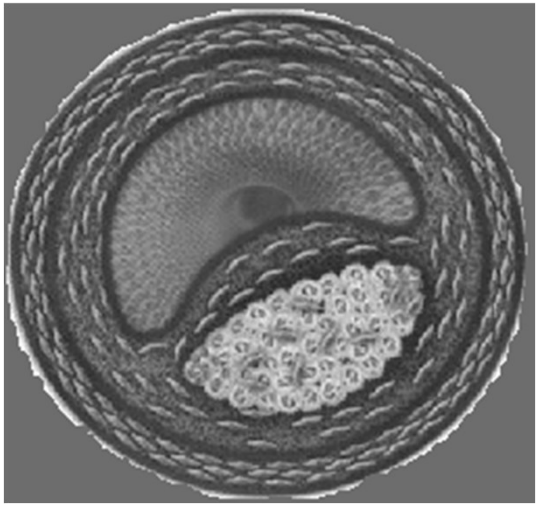
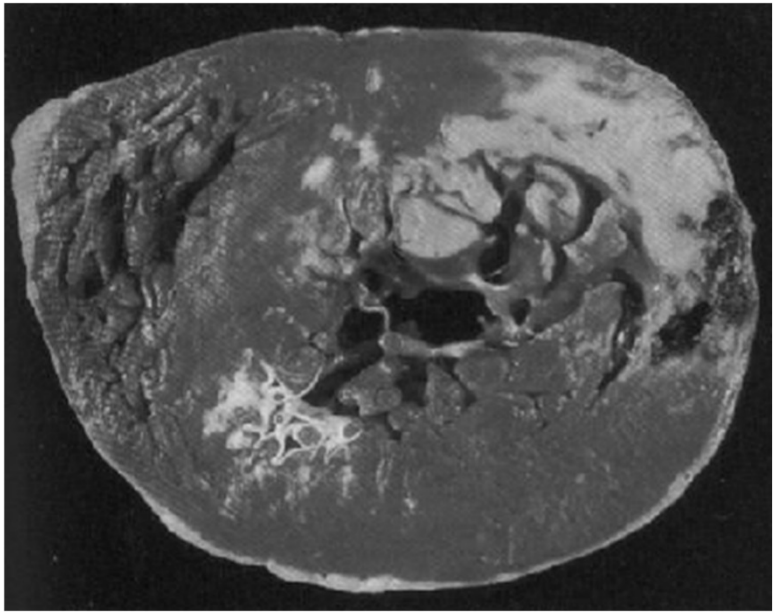
FACULTY OF MEDICINE
DIVISION OF CLINICAL PHYSIOLOGY

PATHOPHYSIOLOGY OF MYOCARDIAL ISCHAEMIA



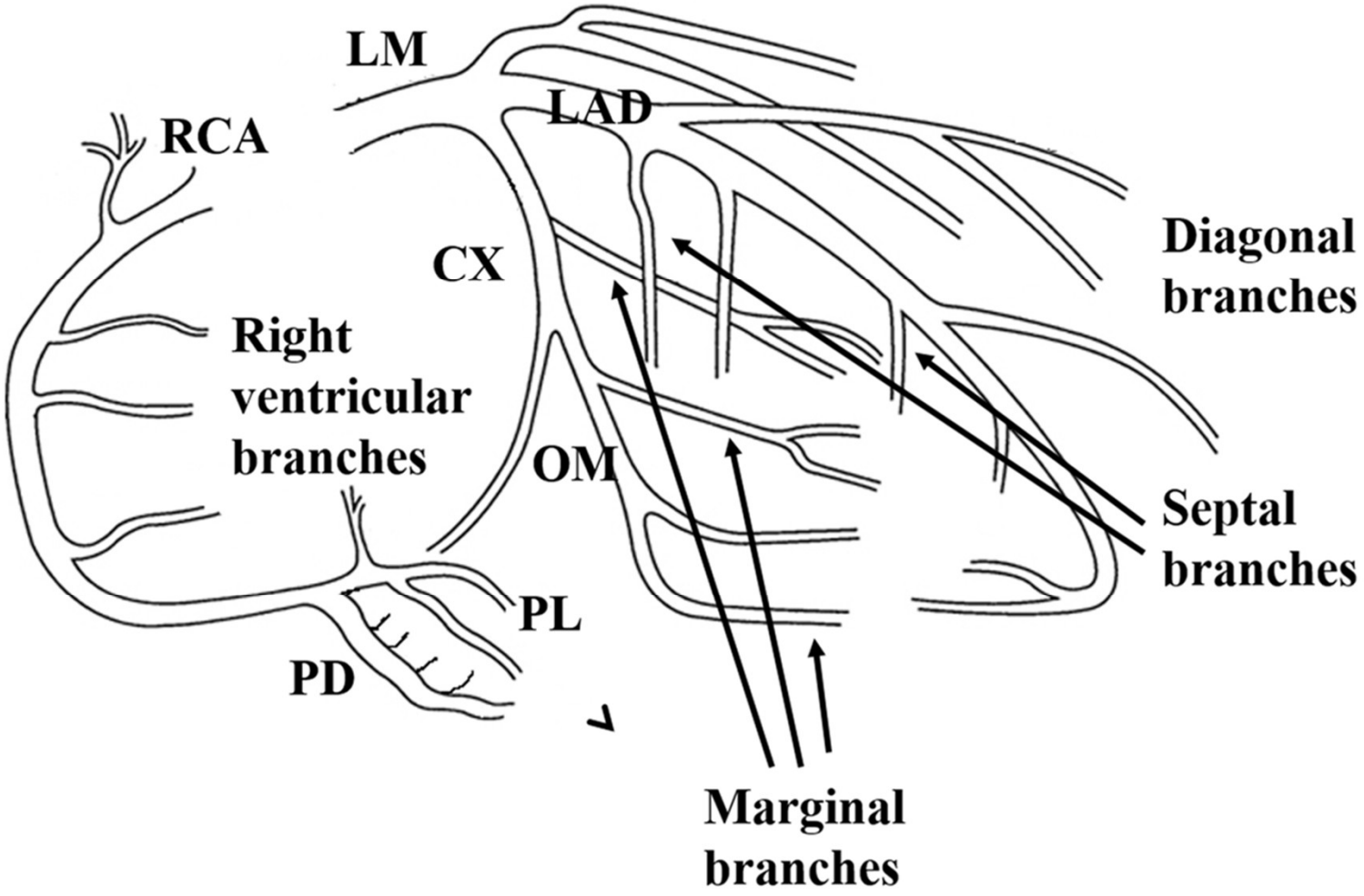


Atherosclerotic plaque



Myocardial necrosis (infarction)

THE CORONARY SYSTEM



ANGINA PECTORIS:

Constricting, opressing chest pain

Typical ischaemic ECG alterations but NO necroenzymes in plasma

Explanation: atherosclerotic plaque + increased O_2 demand

MYOCARDIAL INFARCTION:

Typical (constricting/opressing) intensive chest pain

Typical ECG alterations and elevated necroenzyme levels in plasma

Explanation: rupture of atherosclerotic plaque and thrombocyte aggregation with coronary occlusion

Chest pain

ECG

Serum troponin

STEMI

Negative

Pozitive

Angina pectoris

NSTEMI

ECG patterns of myocardial ischaemia

Coronary stenosis/occlusion may induce myocardial damages with different degrees of severity:

REVERSIBLE alterations (ECG signs of ventricular repolarization)

1. degree: ISCHAEMIA – T wave abnormalities

2. degree: LAESION (injury) – ST segment deviation

IRREVERSIBLE alterations (ECG signs of ventricular depolarization)

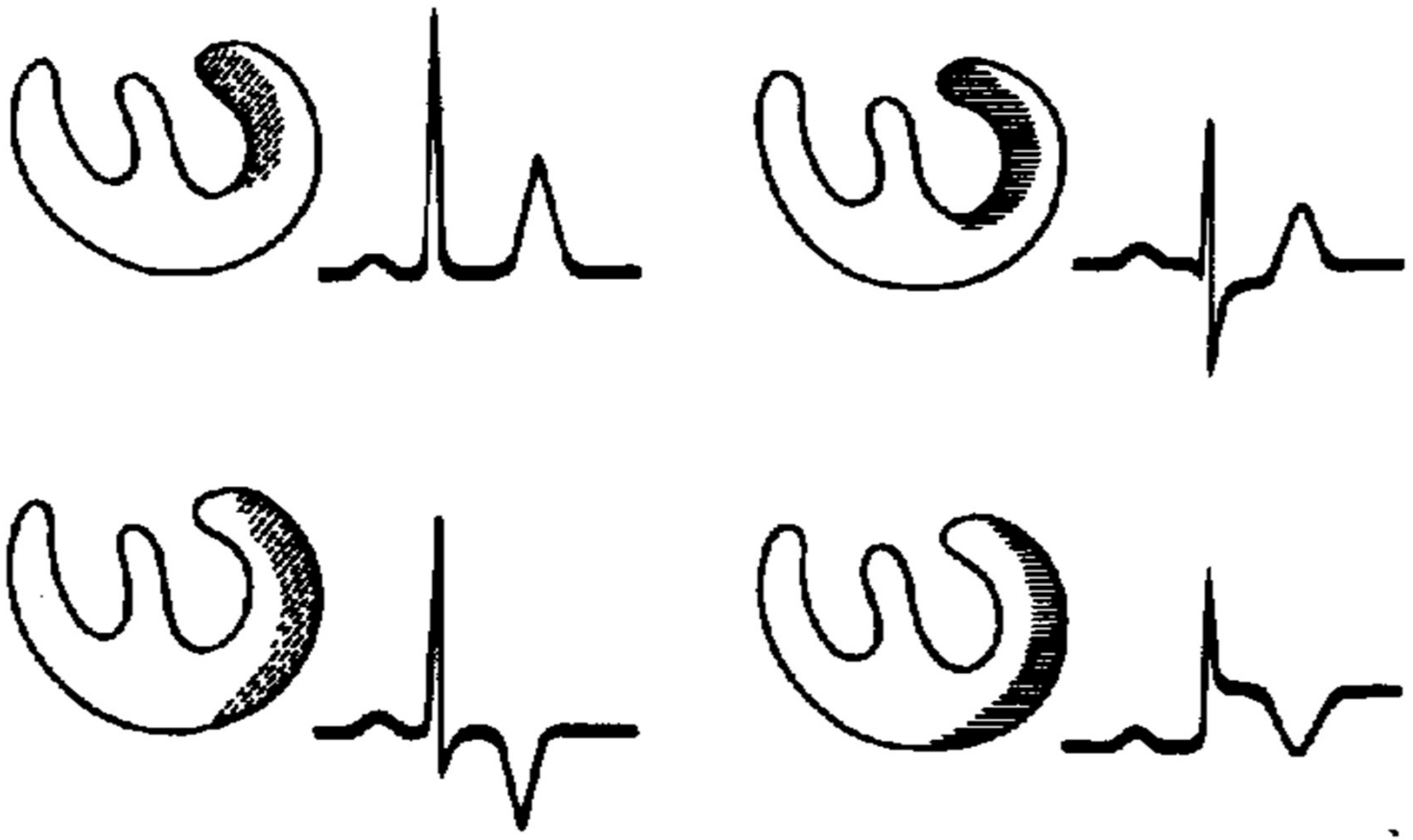
3. degree: NECROSIS – pathologic Q wave

ECG alterations may develop in a successive order with the progression of the disease, but may may also combine with each other.

HYPOXIA INDUCED REVERSIBLE ALTERATIONS

SUBENDOCARDIAL

ISCHAEMIA



LAESION

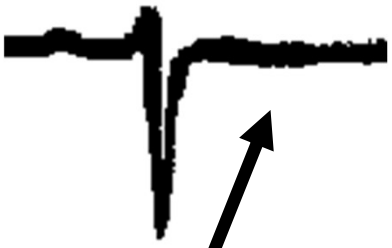
SUBEPICARDIAL

Hypoxia induced ECG alterations are regularly mixed.
E.g.: ST depression + negative T (term: ischaemic ECG signs)

TYPICAL T WAVE ALTERATIONS

NORMAL

ISOELECTRIC



BIPHASIC

NEGATIVE

TALL, PEAKY („HYPERACUT") T WAVE

THEY MAY INDICATE ISCHAEMIA!

DEEP, PEAKY, SYMMETRIC („CORONARY") T WAVE

Differential-diagnosis:

- change in autonomic tone (norm.)
- change in plasma K^+ level
- peri- et myocarditis
- secunder alterations (e.g. BBB)

TYPICAL ST ALTERATIONS

ASCENDING

HORIZONTAL

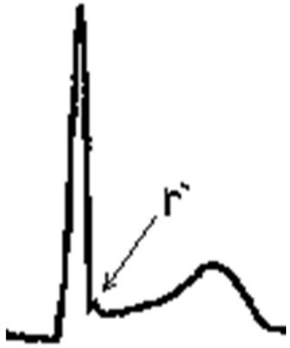
ST-SCOOPING

DESCENDING

CONCAVE

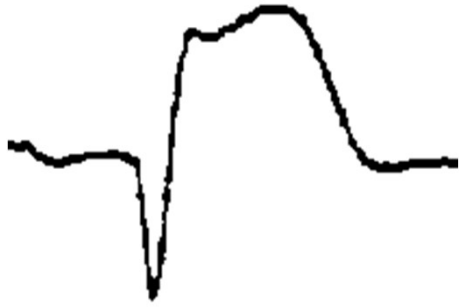
SADDLE SHAPED

EARLY REPOL.



CONVEX

DOME-LIKE



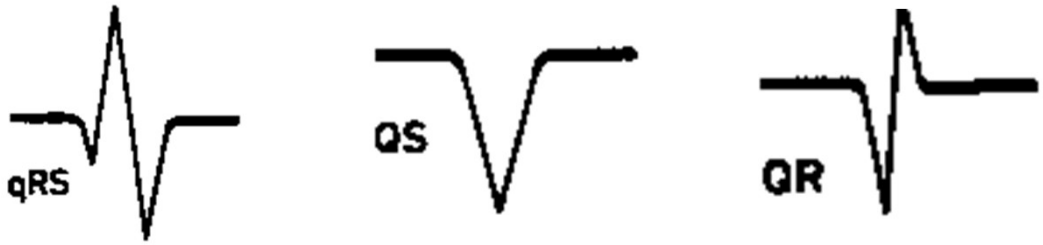
Differential-diagnosis: Normal variant, ion disturbance, digitalis effect

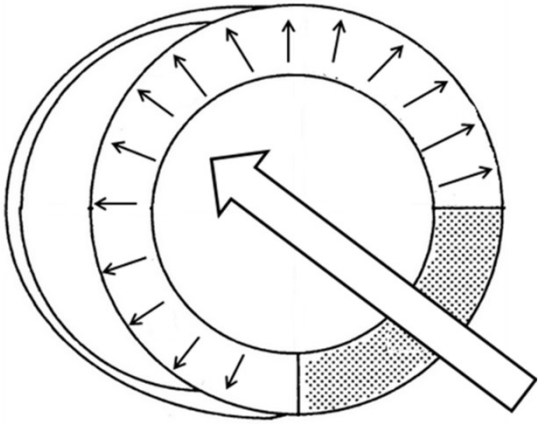
peri - et myocraditis, secunder (e.g. BBB)

Q WAVE

NORMAL FEATURE:

q	**I, aVL, V5-V6 / II, III, aVF**
QS	**aVR, V1**
positional Q	**III**

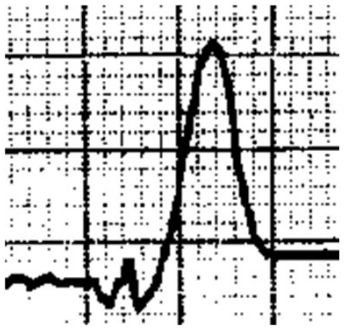




<u>MYOCARDIAL INFARCTION, PATHOLOGICAL Q WAVE:</u>

1. **Deep (>4 mm, or larger than 25% of the subsequent R wave)!**
2. **Wide (≥ 0.04 sec, 1 „small square”)!**
3. **Appears in leads where it is not expected under normal conditions.**
4. **Appears in several leads that are typical for infarction.**
5. **Does not disappear/change for deep inspiration.**

THE DYNAMICS OF MYOCARDIAL INFARCTION (STEMI)



HYPERACUT PHASE (during the first minutes):

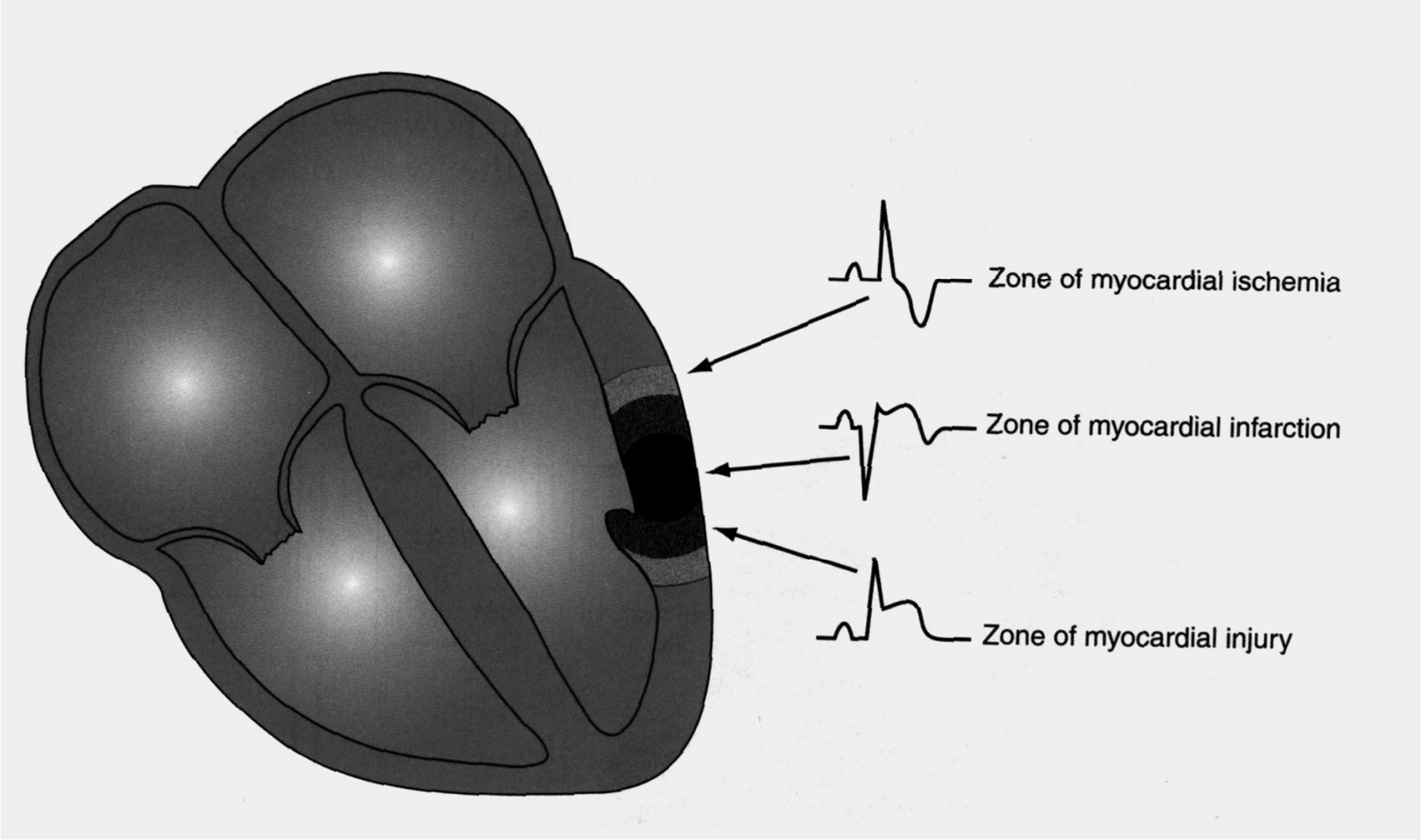
Tall, peaky, „tented" (hyperacut) T waves

Dome-like ST elevation (during the first hours)
non pathologic q wave

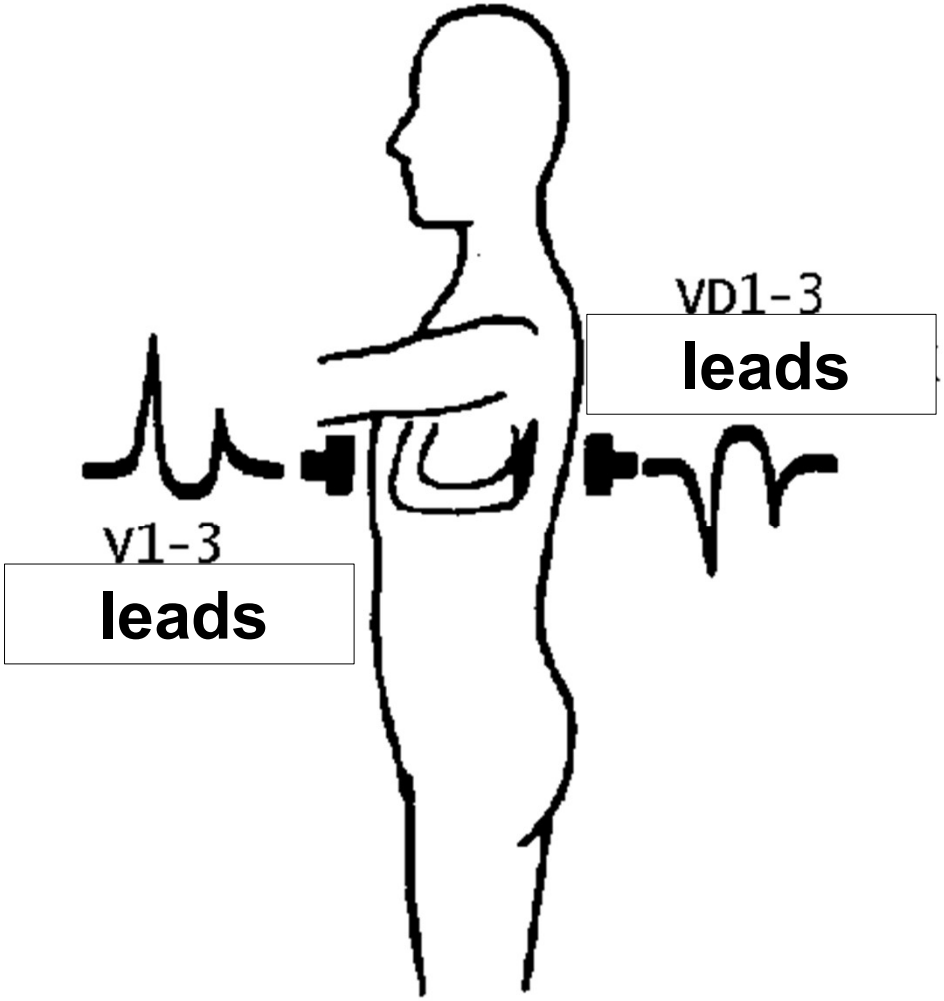
ACUT PHASE (during the first day) :
Pathologic Q wave
Convex ST elevation
Negative T wave
SUBACUT PHASE (during the first days):
Signs of acut phase are more pronounced.

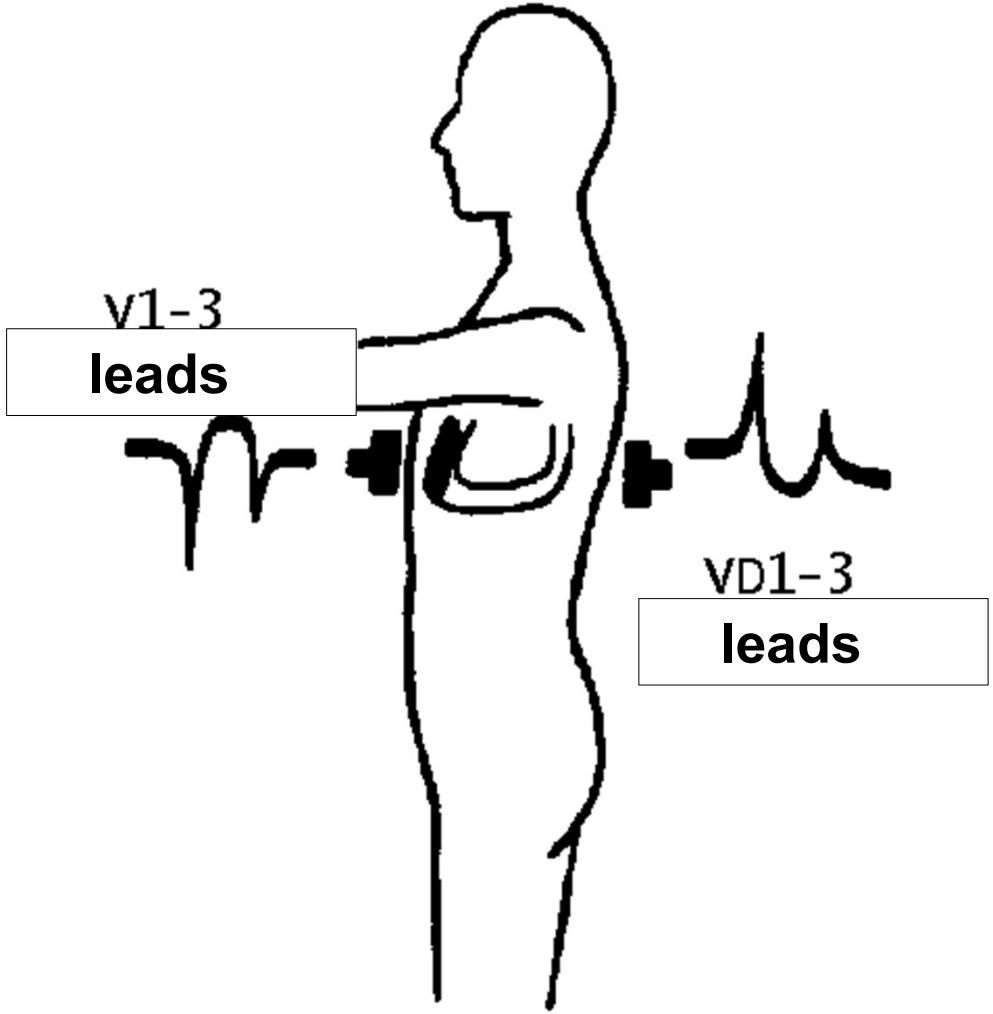
SCAR TISSUE (chronic phase):
Pathologic Q or QS, isoelectric ST, negative or positive T

ECG patterns of myocardial infarction (STEMI)

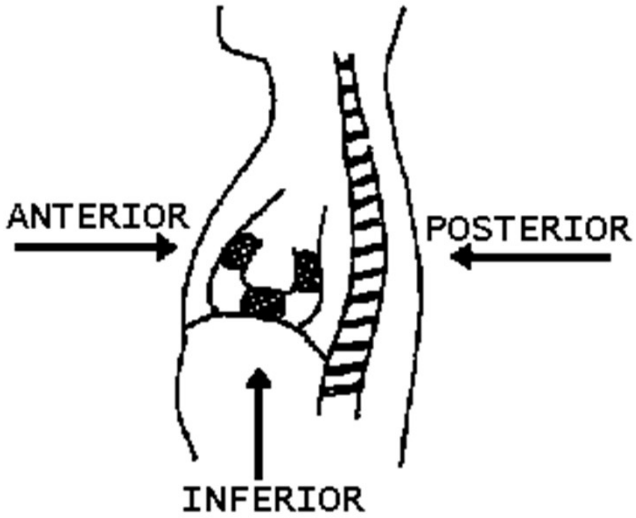


INTERPRETATION OF ECG MIRROR SIGNS





LOCALIZATION OF THE MYOCARDIAL INFARCTION

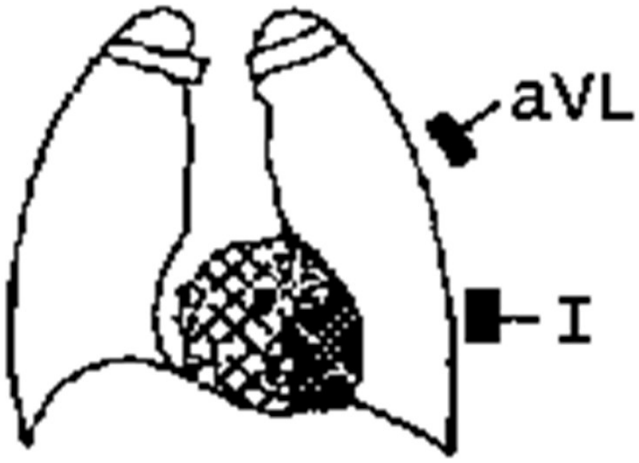


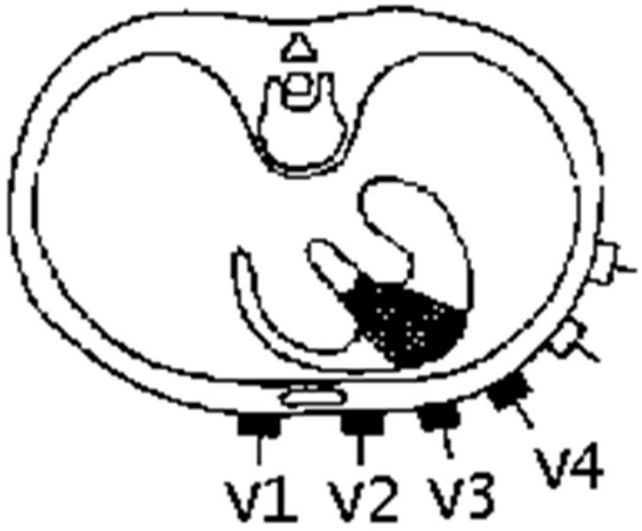
ANTERIOR (ANTEROSEPTAL): V1-V4

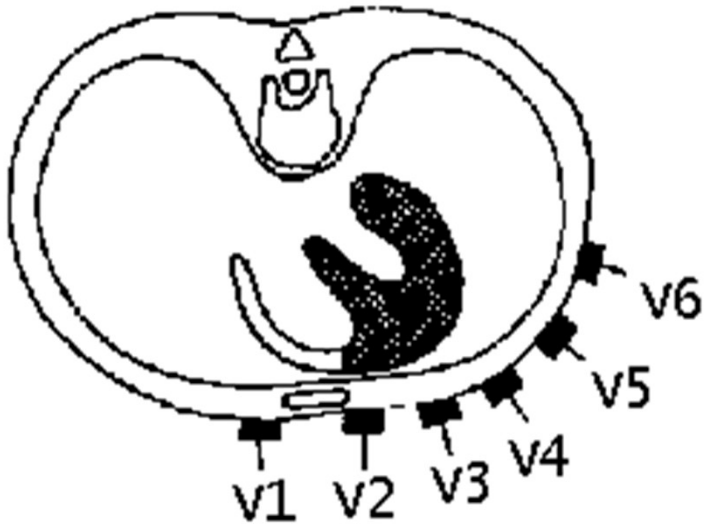
ANTEROLATERAL: I, aVL, V5-V6

EXTENSIVE ANTERIOR: I, aVL, V1-V6

HIGH LATERAL: I, aVL







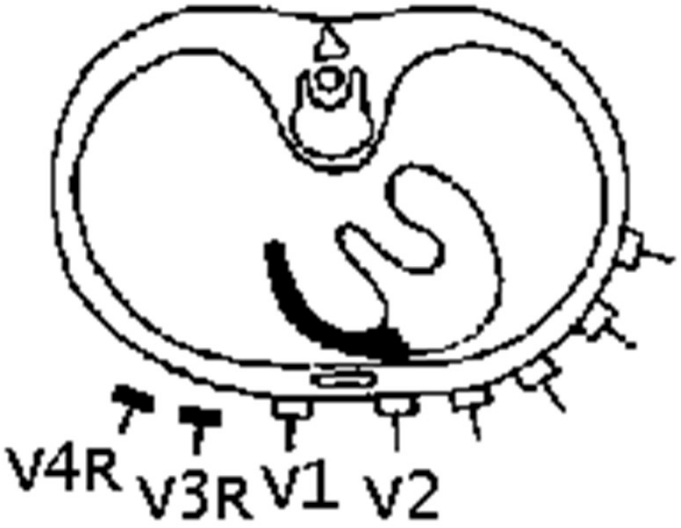
LOCALIZATION OF THE MYOCARDIAL INFARCTION

INFERIOR: II, III, aVF

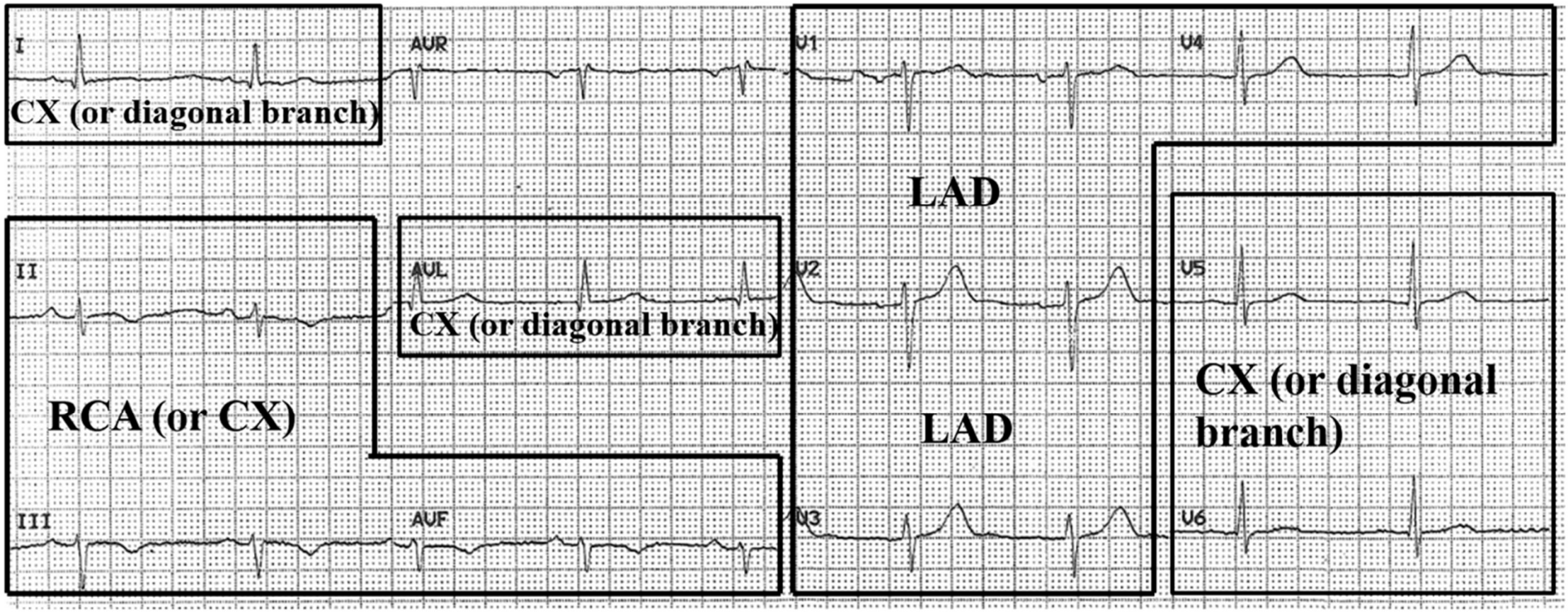
POSTERIOR: VD1-VD3 (V1-V2: mirror im.)

EXTENSIVE INFERIOR: II, III, aVF, VD1-VD3

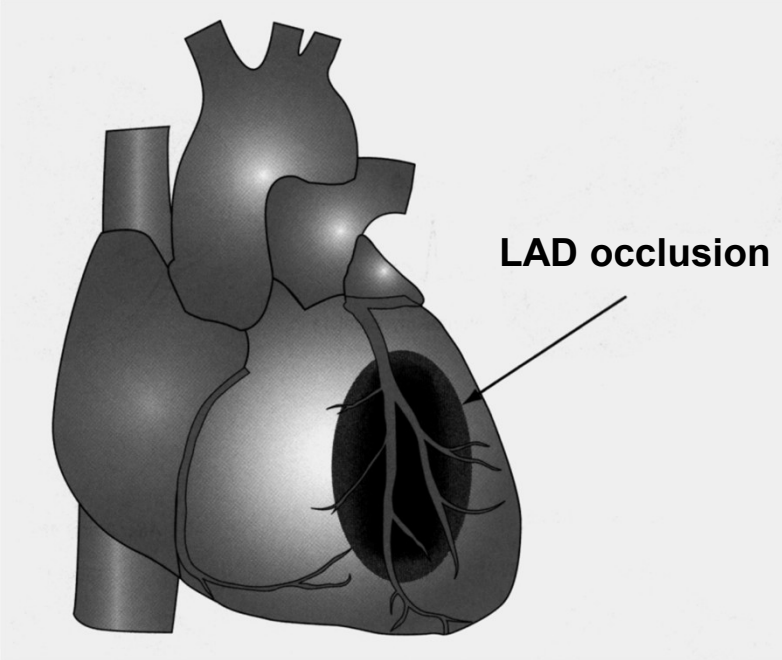
RIGHT VENTRICULAR: V1, V3R, V4R (regularly combines with an inferior myocardial infarction)

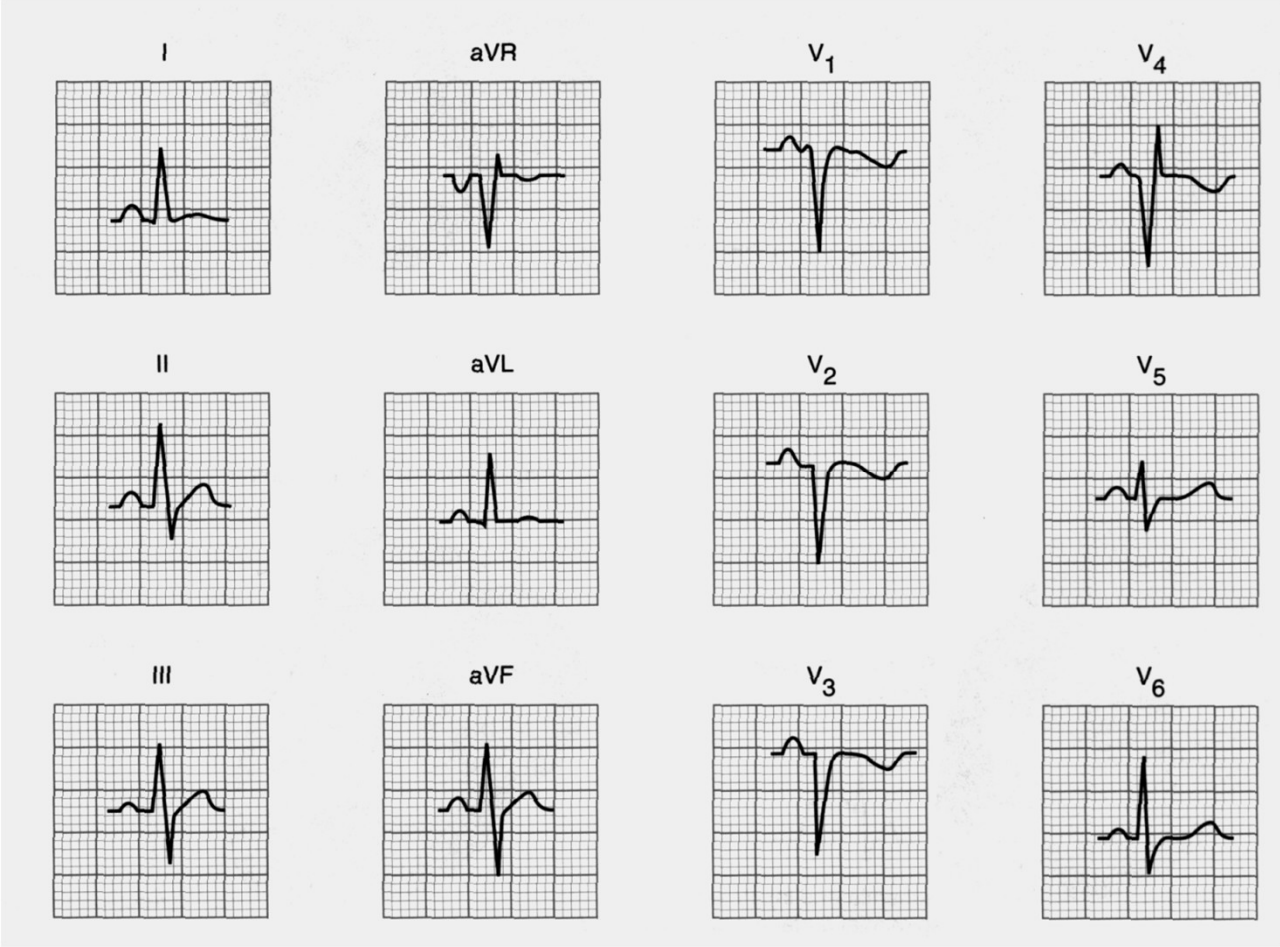


LOCALIZATION OF THE MYOCARDIAL INFARCTION

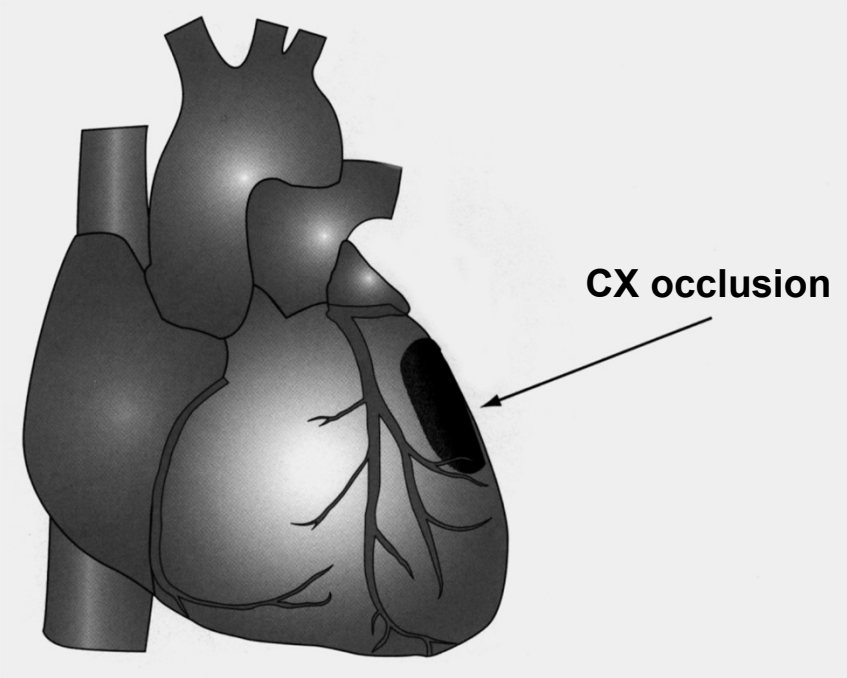


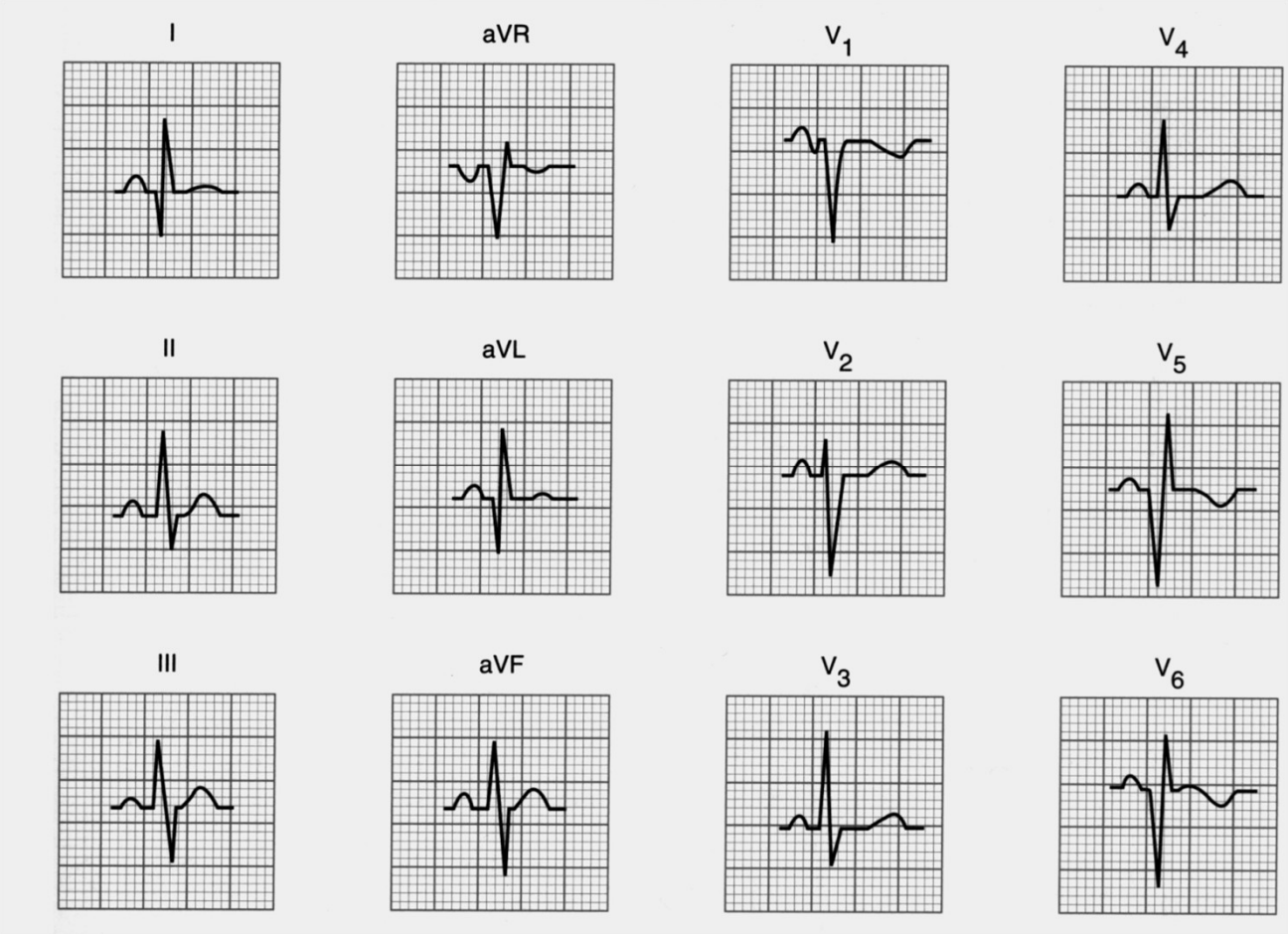
Anterior myocardial infarction



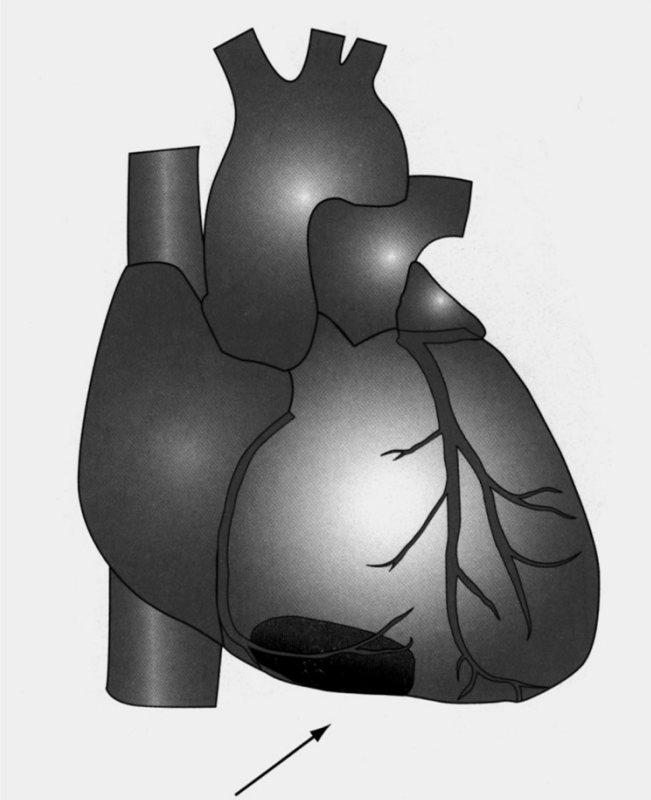


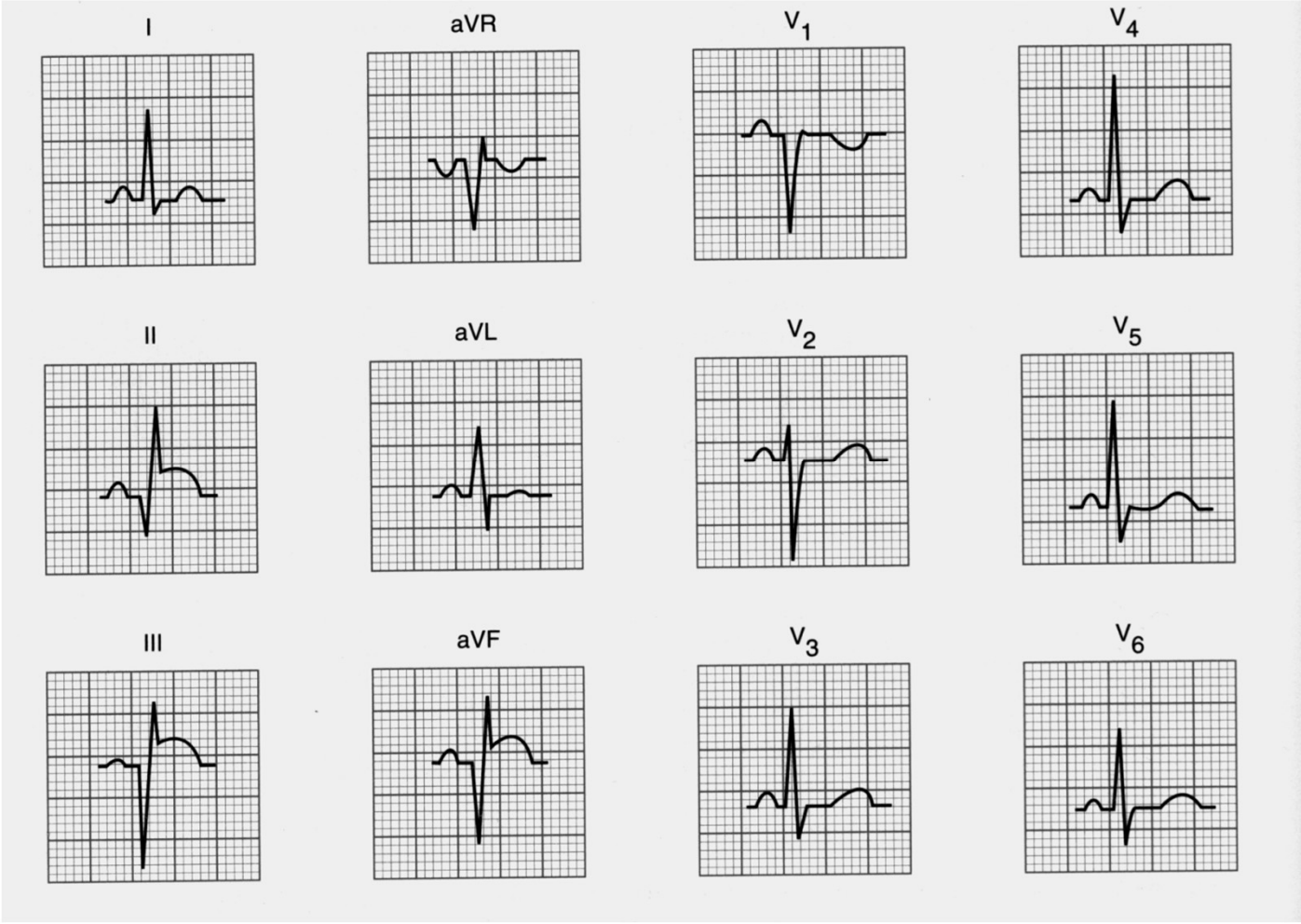
Lateral myocardial infarction





Inferior myocardial infarction





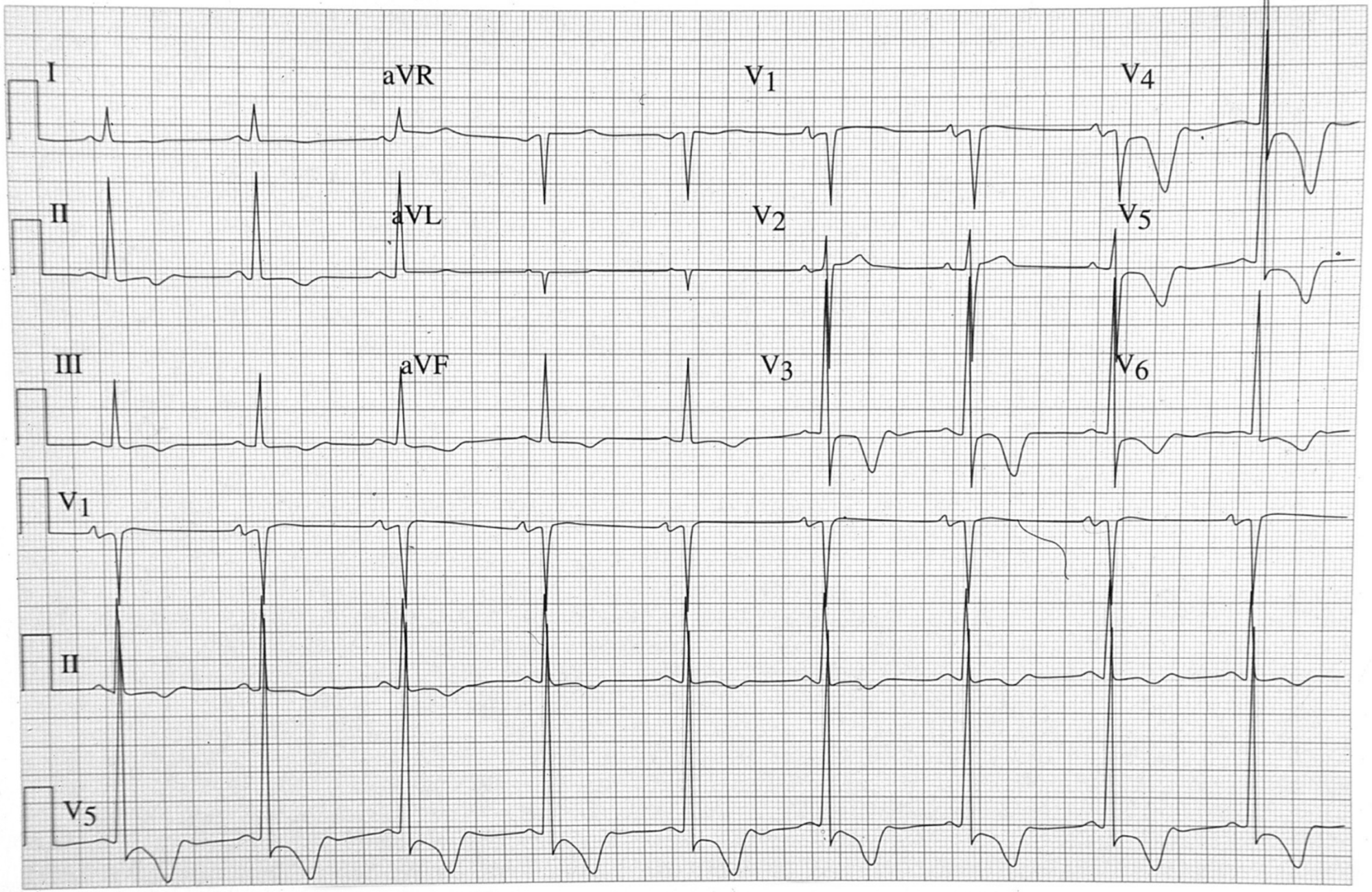
A NON-Q MYOCARDIAL INFARCTION

- There are no pathological Q waves.
- 10% of all myocardial infarction cases are non-Q infarctions.
- Can be accompanied by ST elevations and ST depressions!
- Subendocardial (not transmural!) necrosis.
- Typical physical signs during the acut phase with necroensime level elevations and disturbances in the wall movement (echo!).
- It can be considered as an unfinished event!

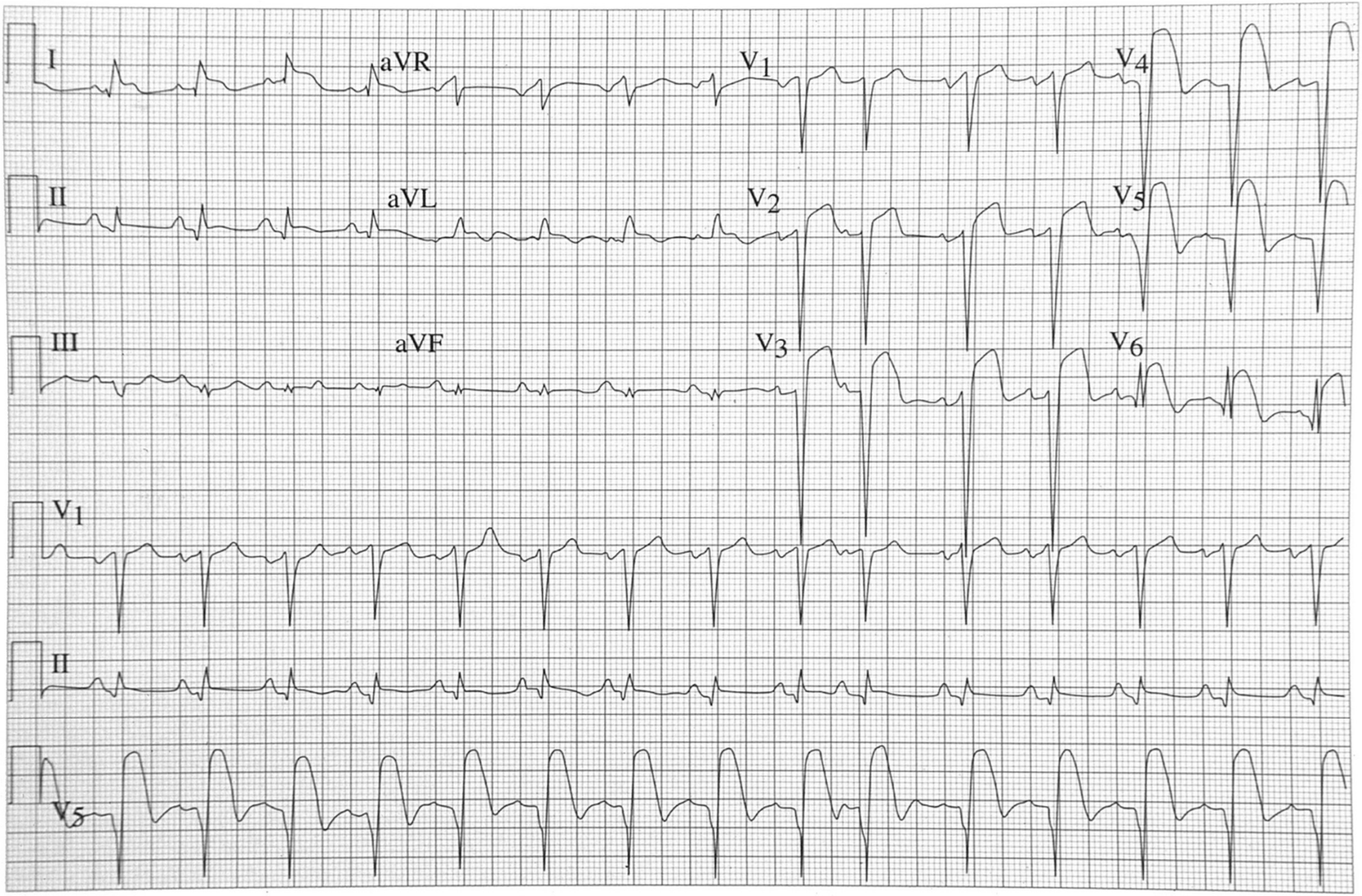
ST ELEVATION CAN BE ALSO INDUCED BY:

1. PRINZMETAL ANGINA (transient ST elevation)
2. PERICARDITIS (in all leads except aVR, saddel shaped)
3. LEVT VENTRICULAR ANEURISM (6 months after AMI ST elevation)
4. BUNDE BRANCH BLOCK (secunder ST alteration)
5. WPW-SYNDROME (secunder ST alteration)
6. TACHYCARDIA (physical exercise: mostly ascending)
7. EARLY REPOLARISATION (r` duirng the beginning of ST non-pathologic)

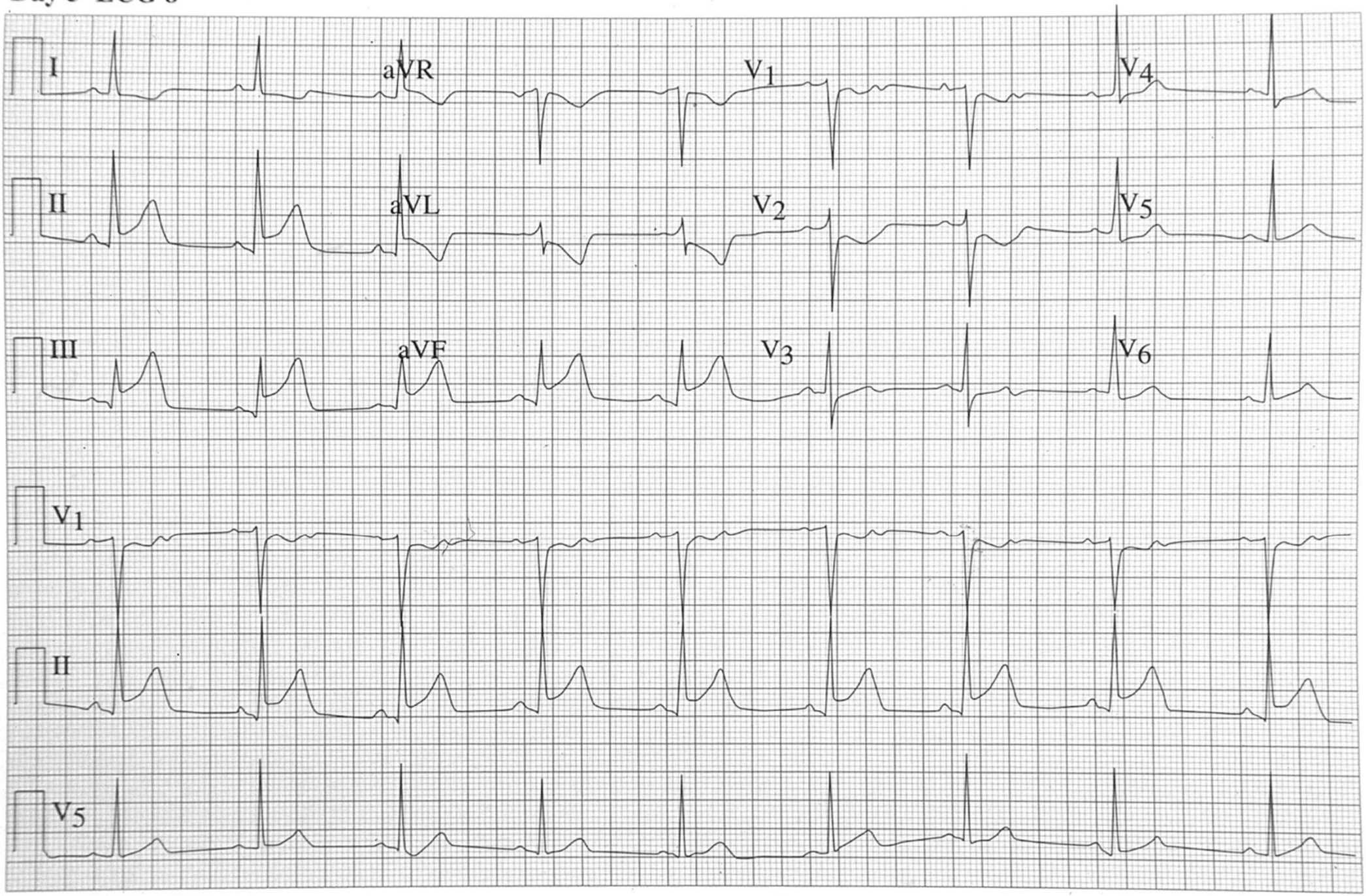
Day 2 ECG 2



Day 5 ECG 6



Day 5 ECG 8



Day 3 ECG10