# ECG in myocardial infarction and ischemia
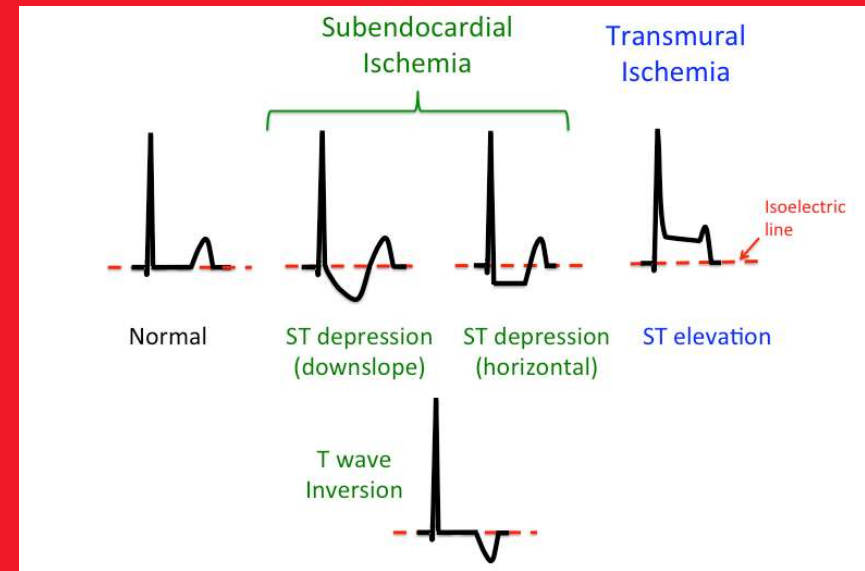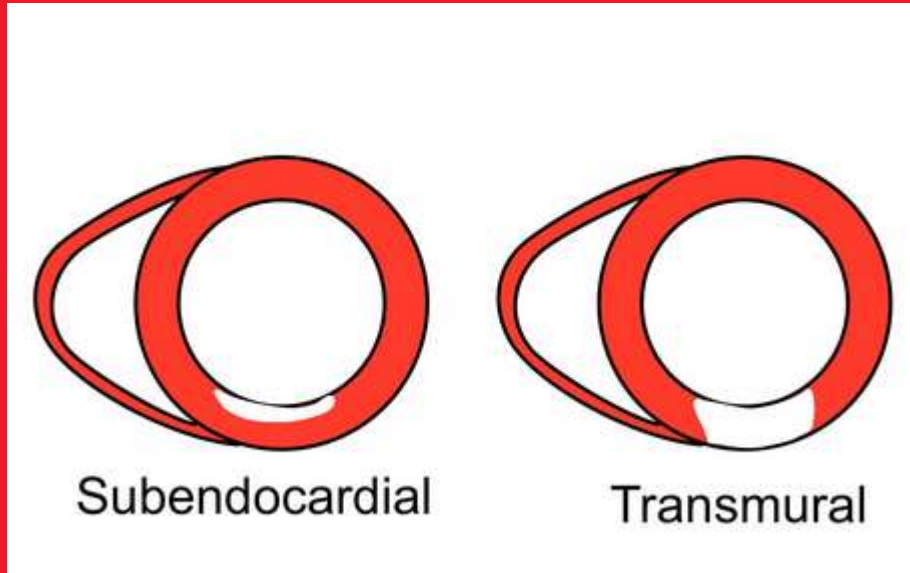
Acute myocardial infarction (AMI)

- Generally, the term „infarction" can be used for any local acute ischemia with necrosis, irrespectively of affected organ

- However, myocardial and cerebral infarction most usually lead to death or invalidity

- Myocardial infarction is the most common life threatening complication of coronary atherosclerosis

- In most cases, its cause is a rupture of unstable atherosclerotic plaque with subsequent thrombosis

- Rare causes: thrombembolism, coronary artery dissection, acute overload of ischemized myocardium

- The ischemia leads to decrease of ATP and subsequent overload of cardiomyocyte by Ca^{2+}, local lactacidosis, permanent depolarization

- Cell death: myocardial necrosis, apoptosis in prolonged ischemia, autophagy is rather protective

- Compared to AMI, causes of cerebral stroke are much more heterogenous, atherosclerosis is often not required (thrombosis, thrombembolism, hemorrhage…)
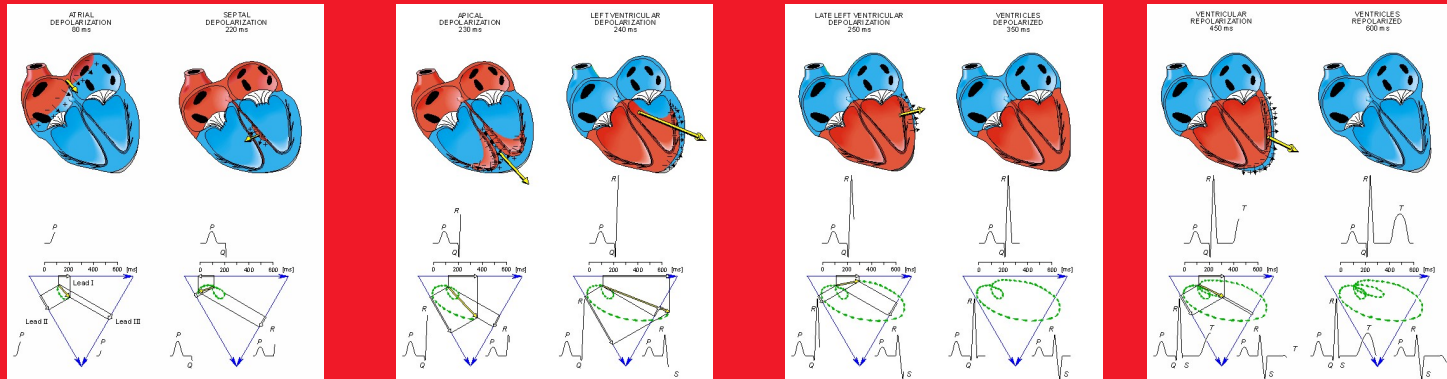
AMIs and strokes during the day
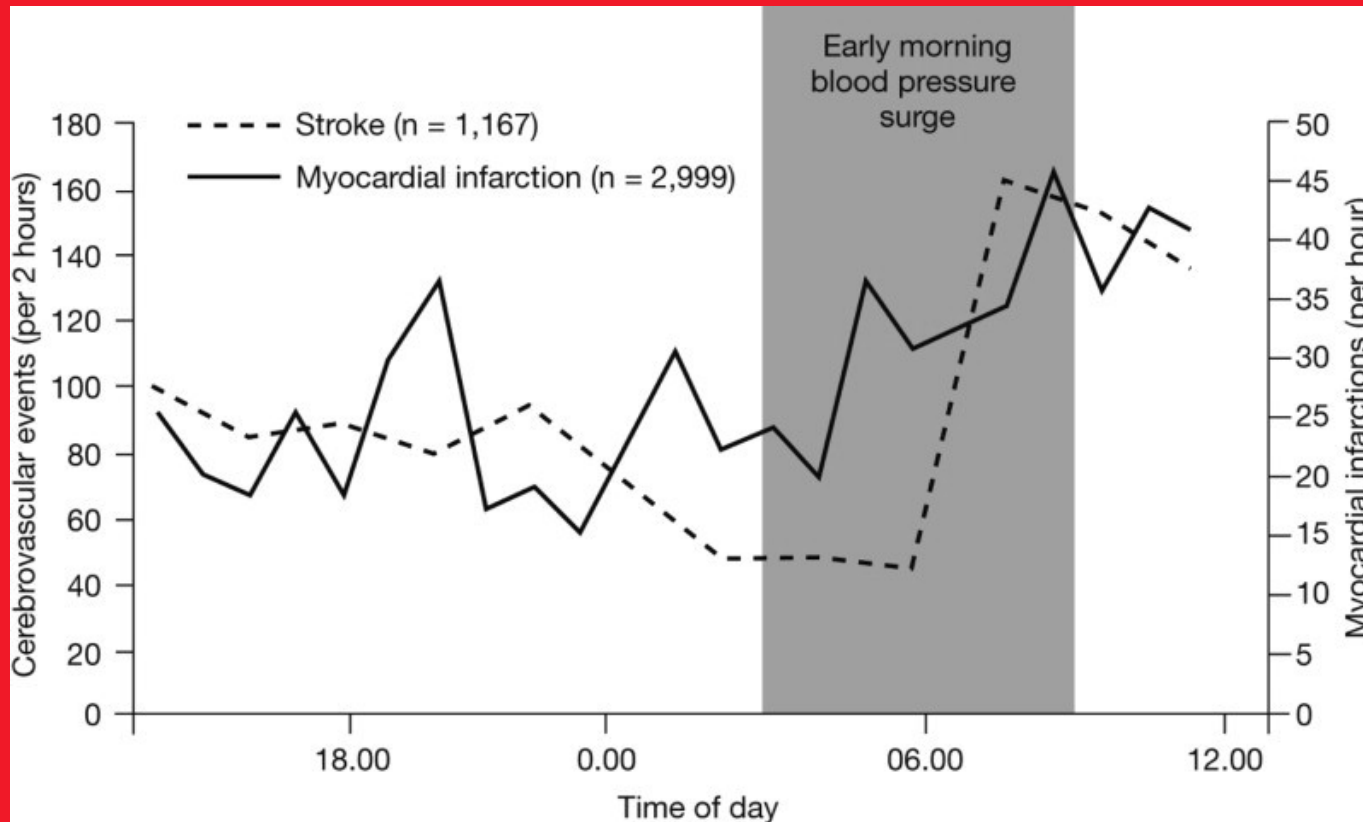
- Higher incidence of cerebral and myocardial infarctions in the morning is caused with higher activity of sympathetic nervous system and higher blood pressure in morning hours

- An important exception are the patients with sleep apnea syndrome

Changes of ST segment during myocardial infarction

Subendocardial   Transmural

Subendocardial Ischemia   Transmural Ischemia

Normal   ST depression (downslope)   ST depression (horizontal)   ST elevation

Isoelectric line

T wave Inversion

Subendocardial vs. subepicardial myocardium

- QT interval and contraction are longer, and therefore the metabolical needs are higher in subendocardial cells

- On contrary, blood supply from the coronary aa. is better in subepicardial myocardium

- Transient, incomplete or limited coronary obstruction therefore always affects the subendocardial myocardium rather than subepicardial

- Severe coronary obstruction affects the whole cardiac wall(transmural IM)
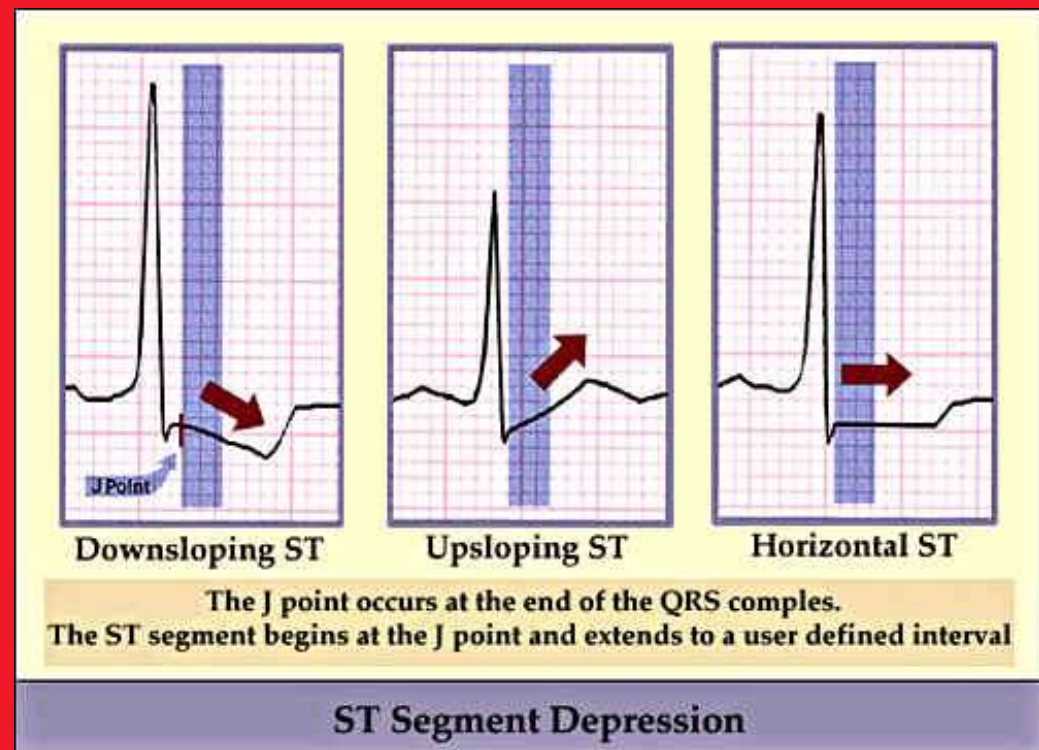
Changes of ST segment 2

- ST elevations or depressions during AMI are caused mainly by a shift of isoelectric line, not ST segment

- During diastole, an ischemic focus generates electric currents

- Depending on its prevailing direction, we can observe elevations (transmural AMI) or depressions (non-transmural AMI) of ST segment – in fact, there is a shift of isoelectric line in opposite direction

- The differences in the plateau phase and repolarization lead into different shape of ST segment
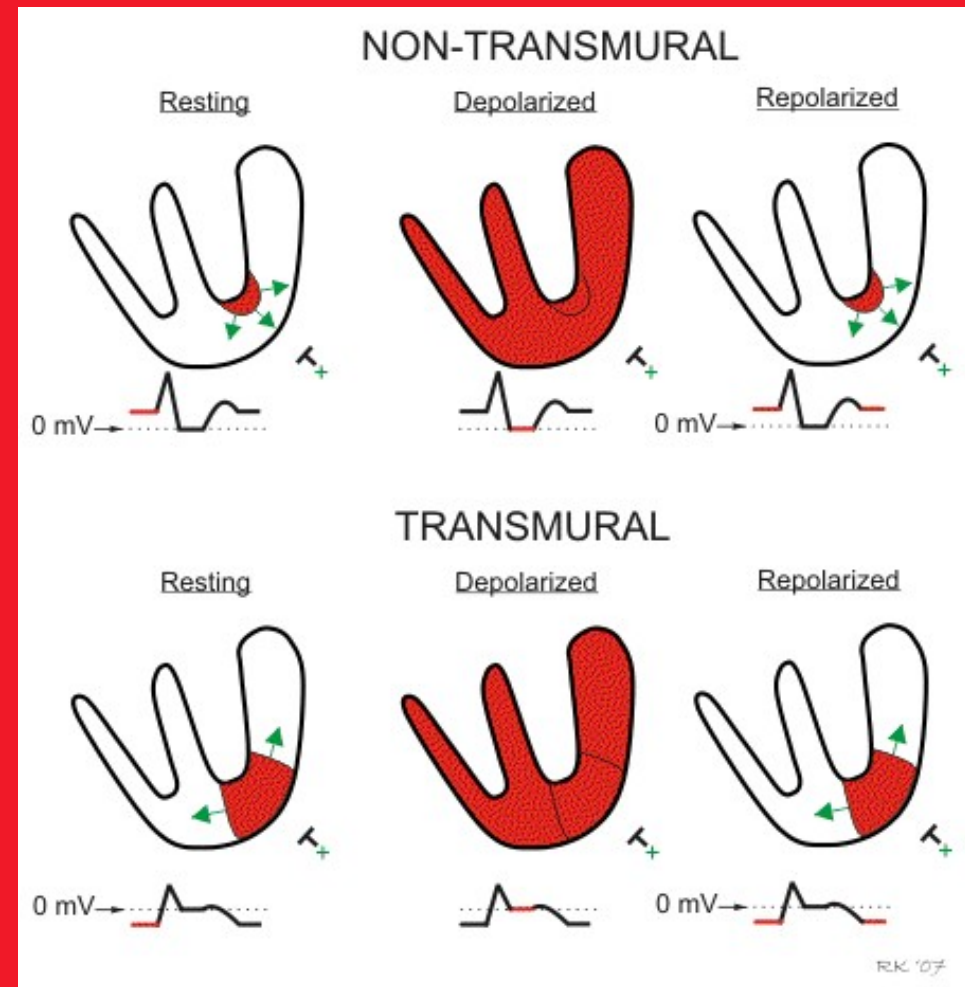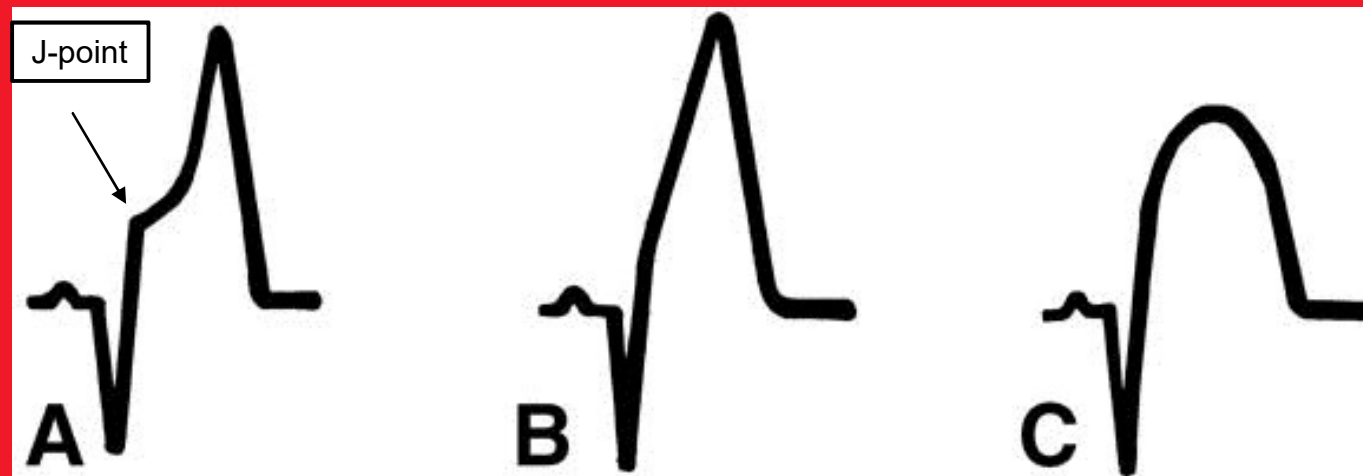
Upsloping, horizontal and downsloping ST segment depressions

- Subendocardial ischemia - horizontal or downsloping depressions of ST segment

- Downsloping depressions occur also e.g. in bundle branch blocks (phase of plateau is different for each part of the ventricle) or digoxin intoxication

- On the other hand, mild (0.1 – 0.2 mV) upsloping ST depressions occur frequently in healthy heart during exercise

Downsloping ST   Upsloping ST   Horizontal ST

The J point occurs at the end of the QRS comples.
The ST segment begins at the J point and extends to a user defined interval

ST Segment Depression

ST elevations

J-point

A
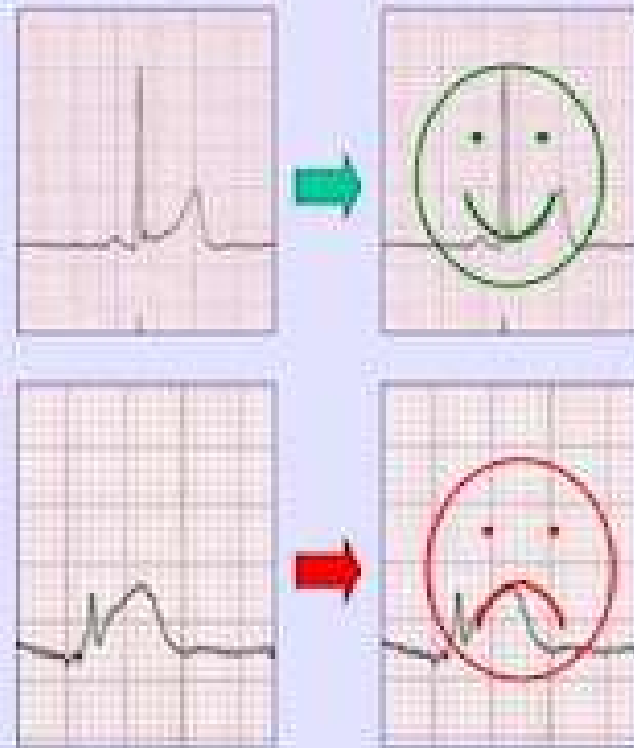
B

C

A – concave (often in the hypertrophy of LV)
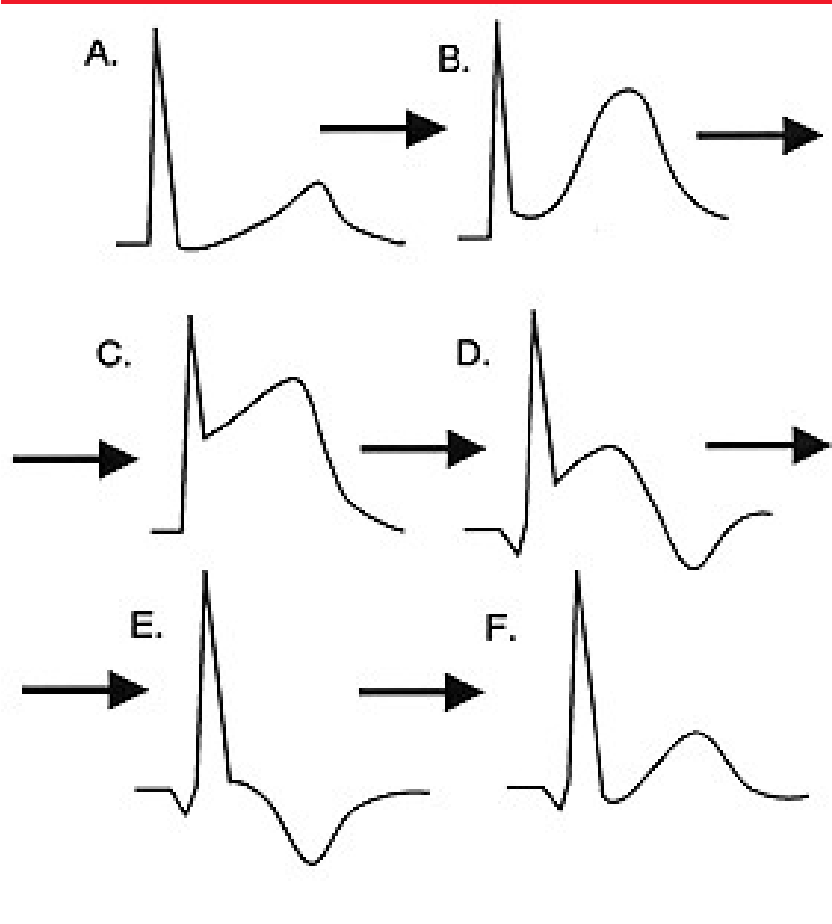B – straight
C – convex   acute transmural myocardial
                  infarction

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ECG changes during Q-MI

- A. initial physiological state
- B. superacute phase
  - Tall positive T waves (minutes)
- C. acute phase
  - ST elevation = Pardee's waves (tens of minutes to hours) - STEMI
- D. subacute phase
  - Normalization of ST segment
- E. Q-wave devolopment (hours to days), event. T – inversion (persists weeks)
- F. ECG after Q-MI
  - persistence of Q

Pathologic Q

- During several hours after transmural MI, pathologic Q develops
- Pathologic Q corresponds to depolarization of opposing cardiac wall, observed through electrically dead tissue – a scar
- Its depth is > ¼ R (or R is not present at all – QS wave) and its duration is at least 40 ms)
- It usually persists lifelong (except certain cases of stunned myocardium)

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Vent freku:	66 BPM
PR int:	137 ms
Cas QRS:	87 ms
QT/QTc:	371/384 ms
P-R-T osy:	28 72 22
Avg RR:	909 ms
QTcB:	389 ms
QTcF:	382 ms

Mgr. Jana Konečná
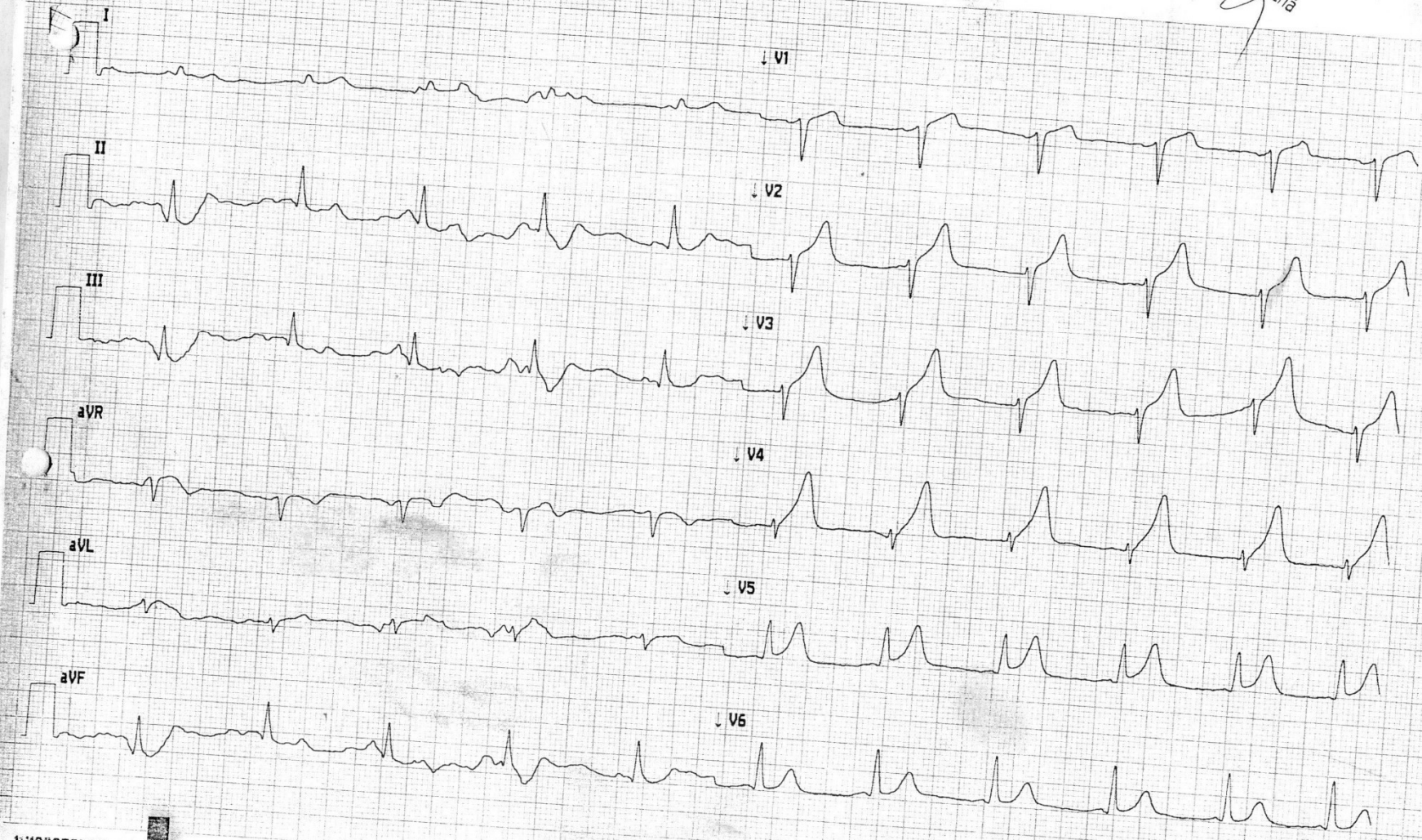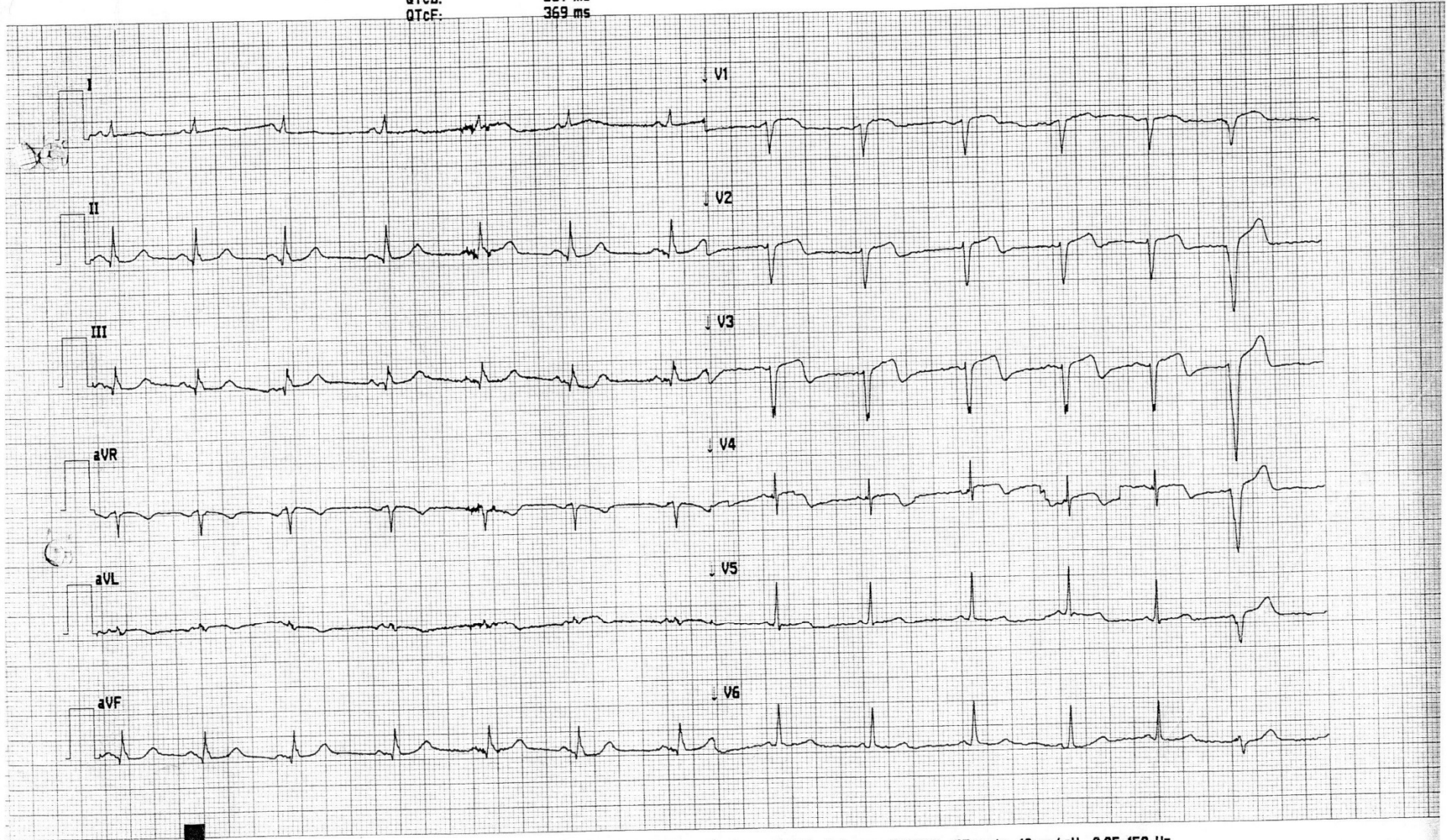
Vent freku: 79 BPM
PR int: 134 ms
Cas QRS: 85 ms
QT/QTc: 337/371 ms
P-R-T osy: 55 59 78
Avg RR: 758 ms
QTcB: 387 ms
QTcF: 369 ms

MIŠKAIOVÁ Jana Dis.

I
II
III
aVR
aVL
aVF

V1
V2
V3
V4
V5
V6

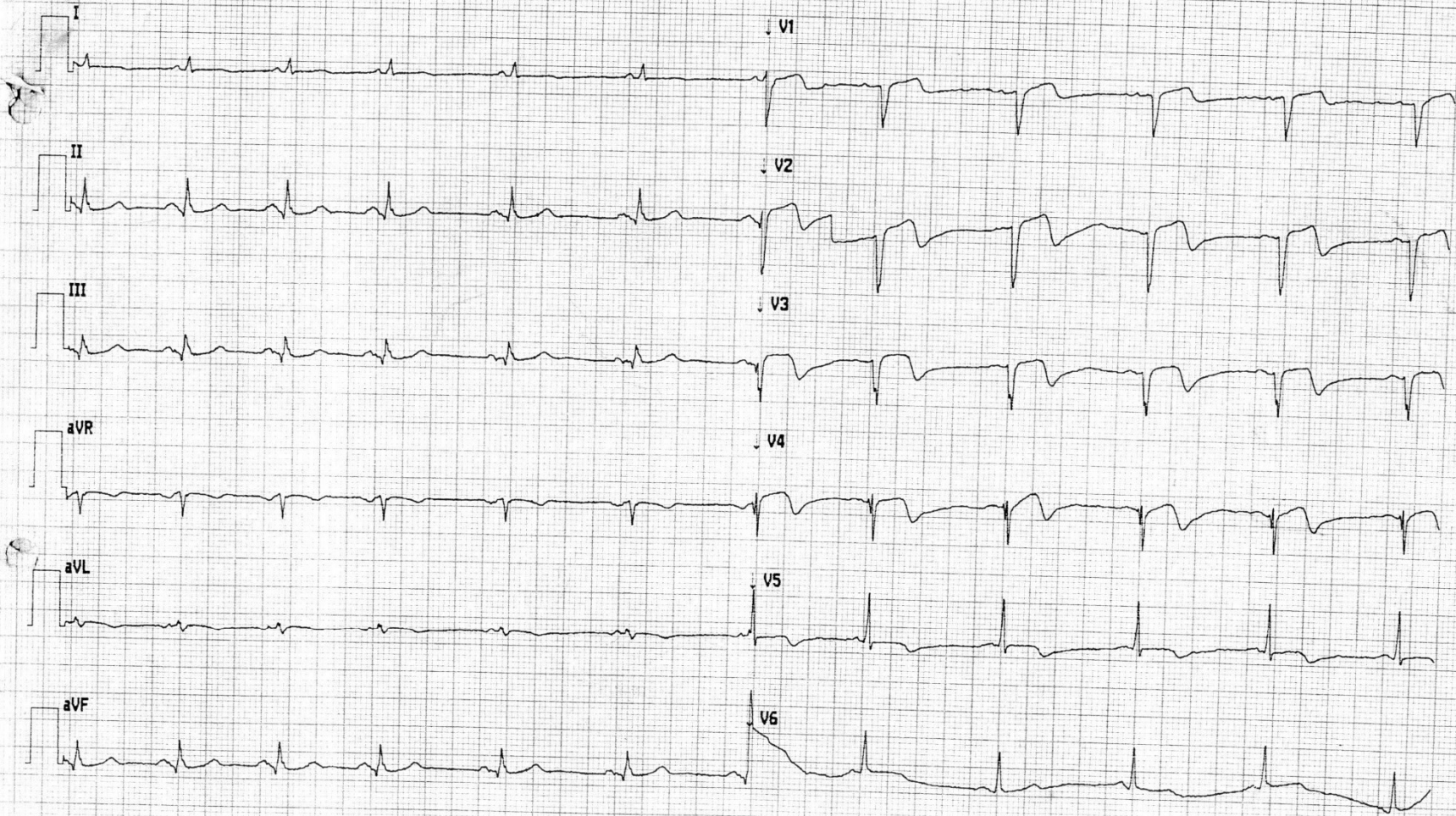111030069619   No Site Name   28.939   Misto# 0 Vozik#   0 Verze 1.50.12 Sekvence #04247 25mm/s 10mm/mV 0.05-150 Hz
                                           Misto# 0 Vozik#   0 Verze 1.50.12 Sekvence #04239 25mm/s 10mm/mV 0.05-40 Hz

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Vent frekv: 68 BPM
PR int: 134 ms
Cas QRS: 90 ms
QT/QTc: 413/430 ms
P-R-T osy: 56 62 97
Avg RR: 875 ms
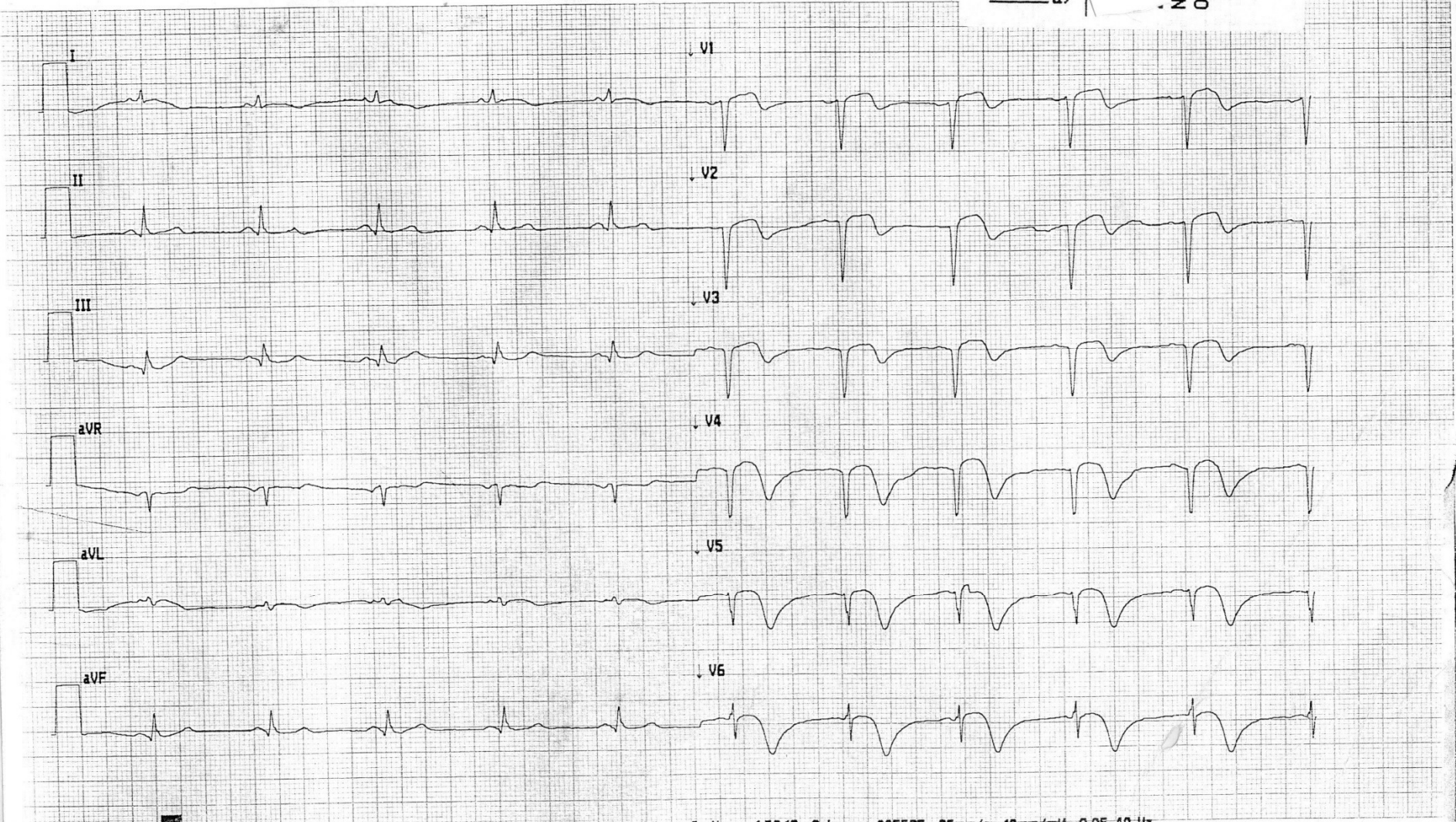QTcB: 441 ms
QTcF: 431 ms

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I
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III
aVR
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V1
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Clinical case

- 59 years old man with acute chest pain, because of ST elevations, coronary arteriography was performed within 1 hour after onset

- LAD occlusion was detected and recanalization was performed

- The finding at coronary arteriography well corresponds with the diagnosis of anterior wall STEMI, based on ECG findings