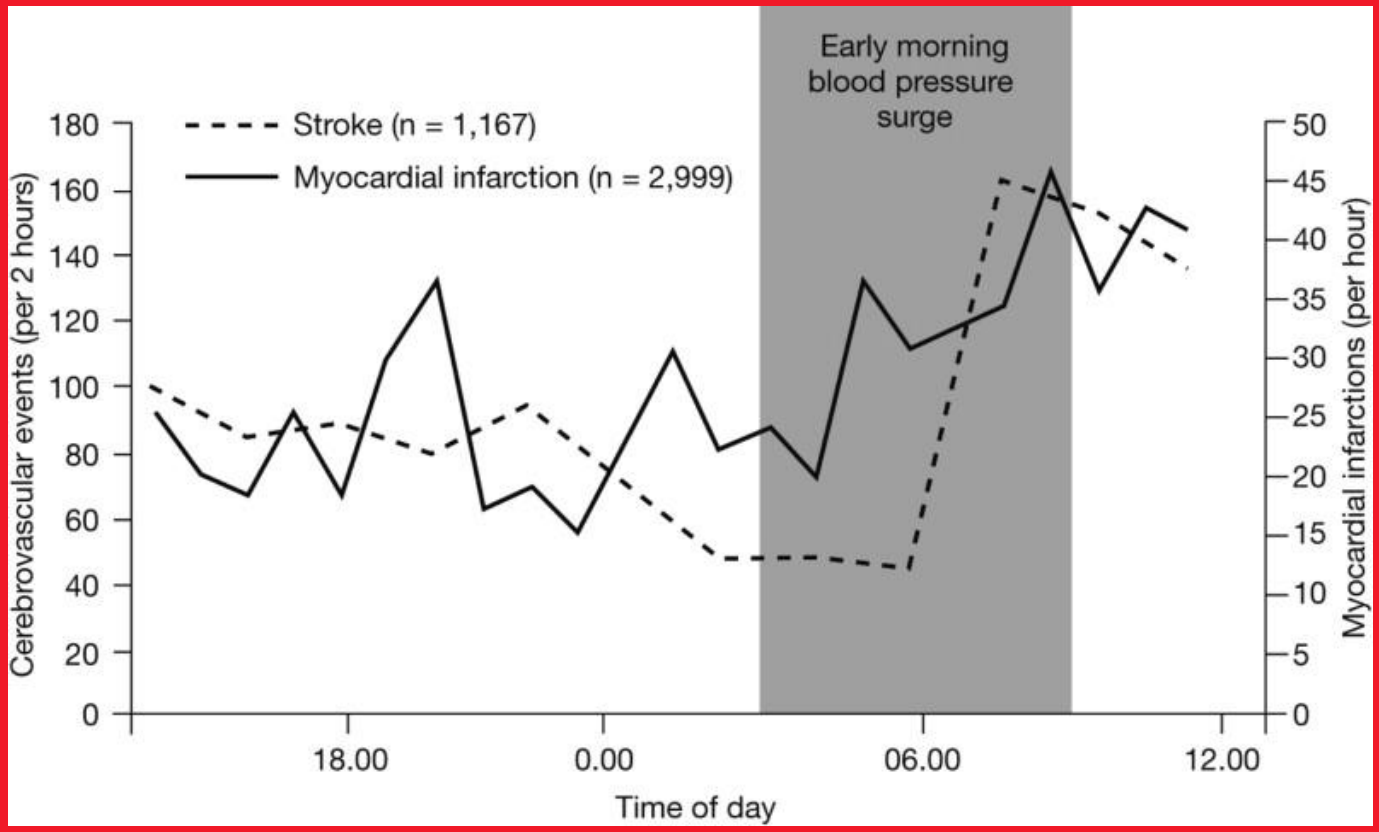
ECG in myocardial infarction and ischemia

Acute myocardial infarction (AMI)

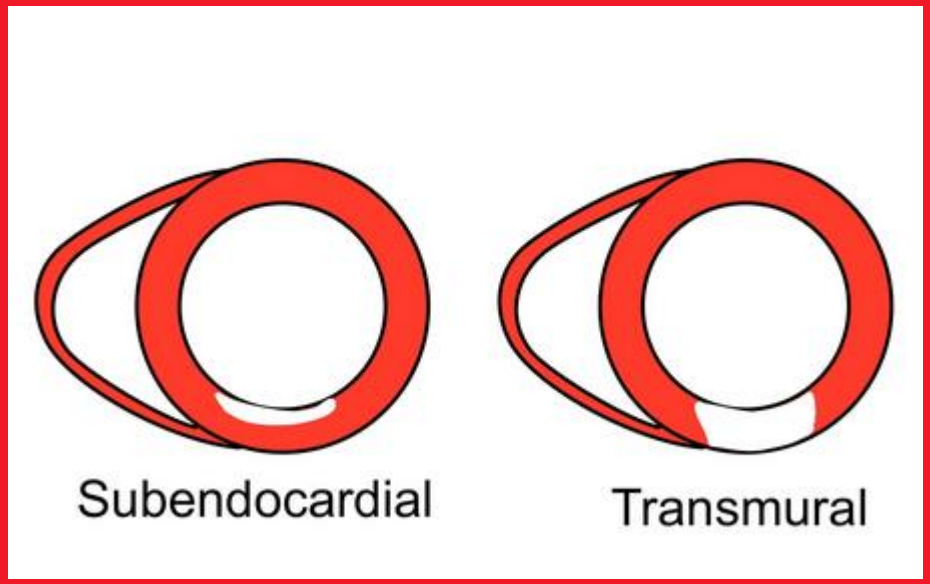
- Generally, the term „infarction“ can be used for any local acute ischemia with necrosis, irrespectively of affected organ
- However, myocardial and cerebral infarction most usually lead to death or invalidity
- Myocardial infarction is the most common life threatening complication of coronary atherosclerosis
- 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…)

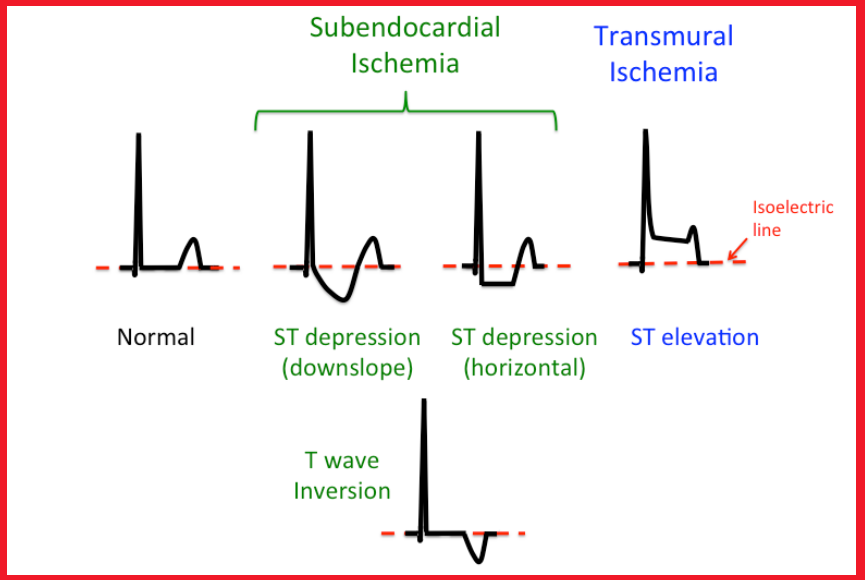
AMIs and strokes during the day



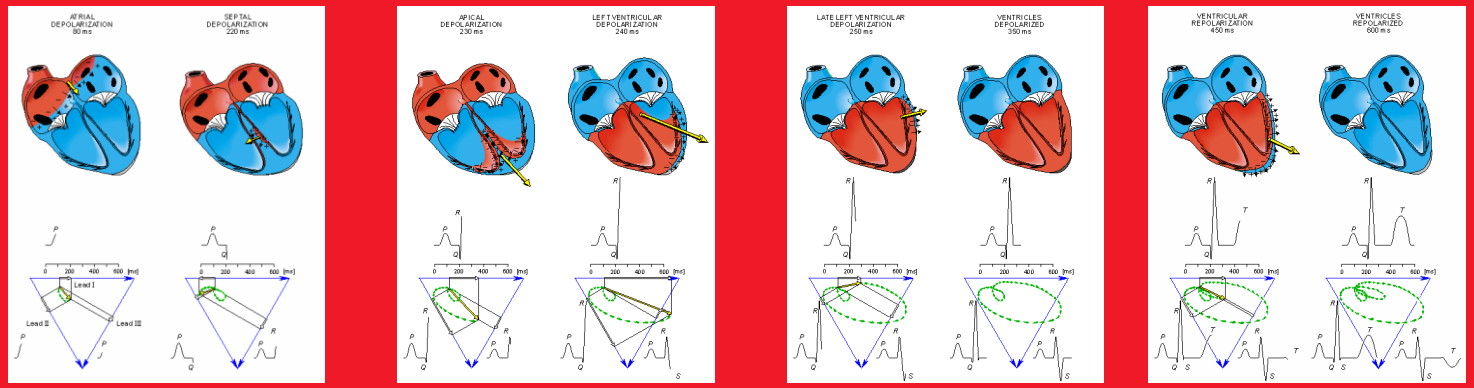
- Higher incidence of cerebral and myocardial infarctions in the morning is caused with higher activity of sympathetic nervous system and higher blood pressure in morning hours
- An important exception are the patients with sleep apnea syndrome

Changes of ST segment during myocardial infarction





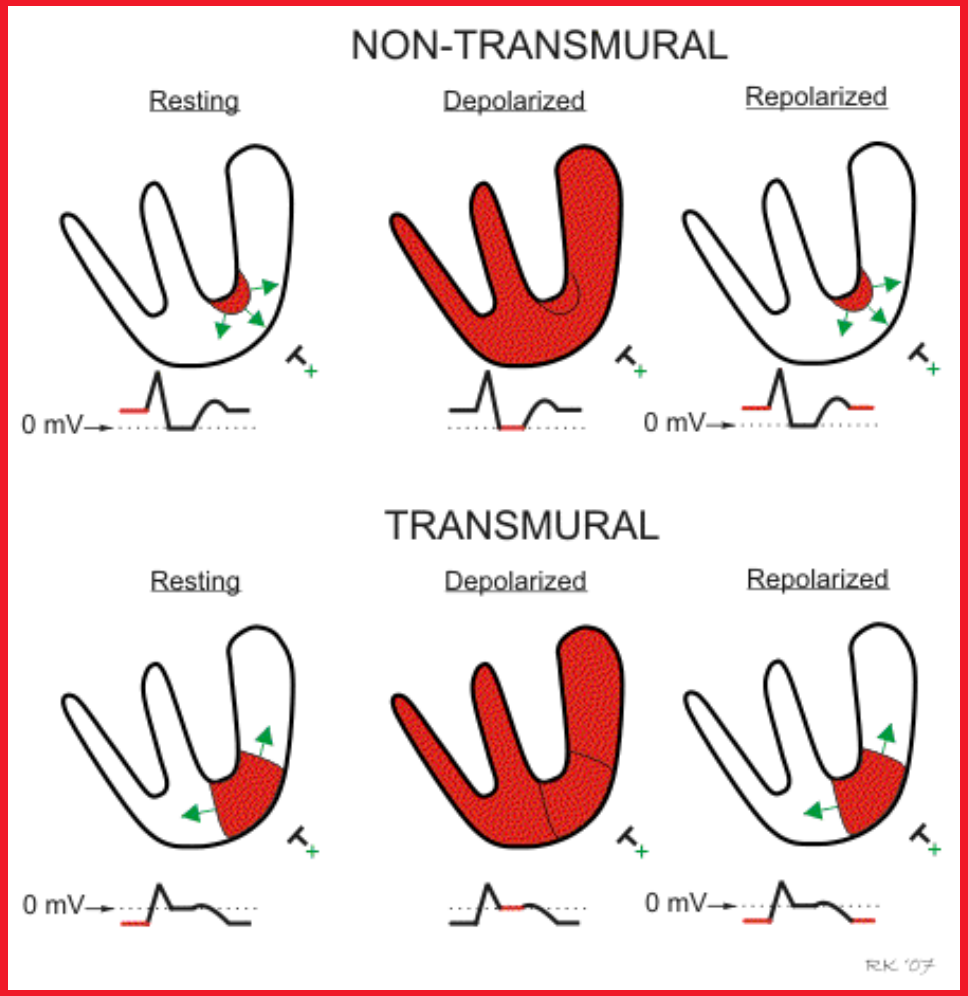
Subendocardial vs. subepicardial myocardium



- 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 wal l(transmural IM)

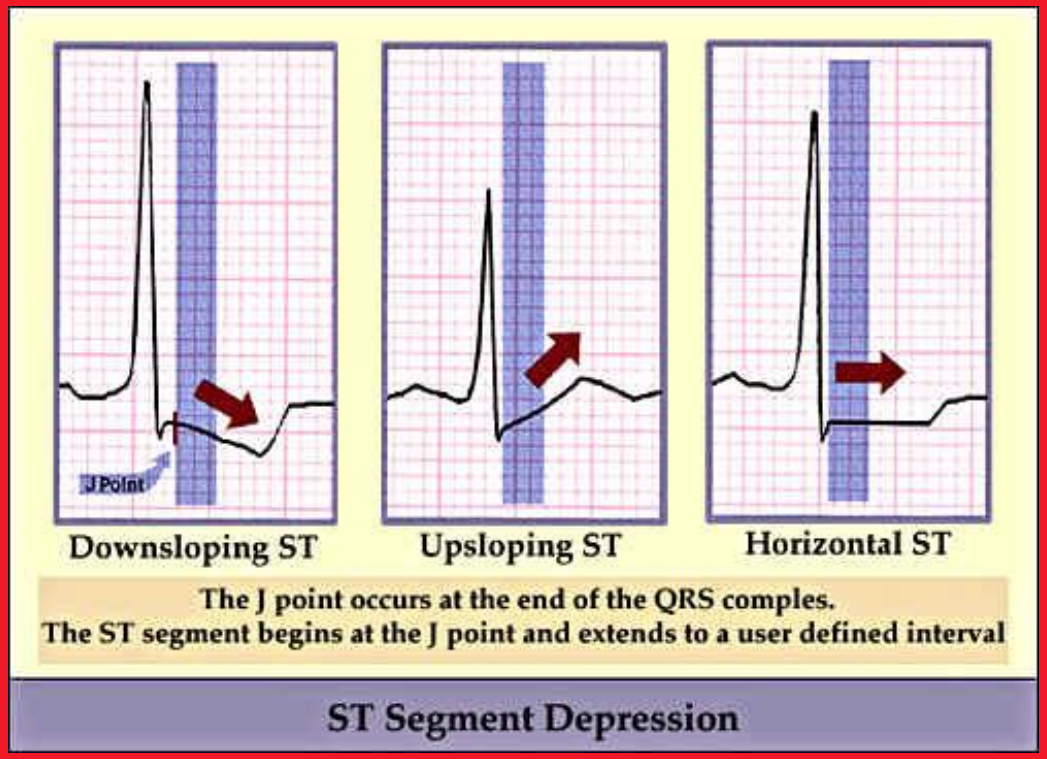
Changes of ST segment 2

- 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

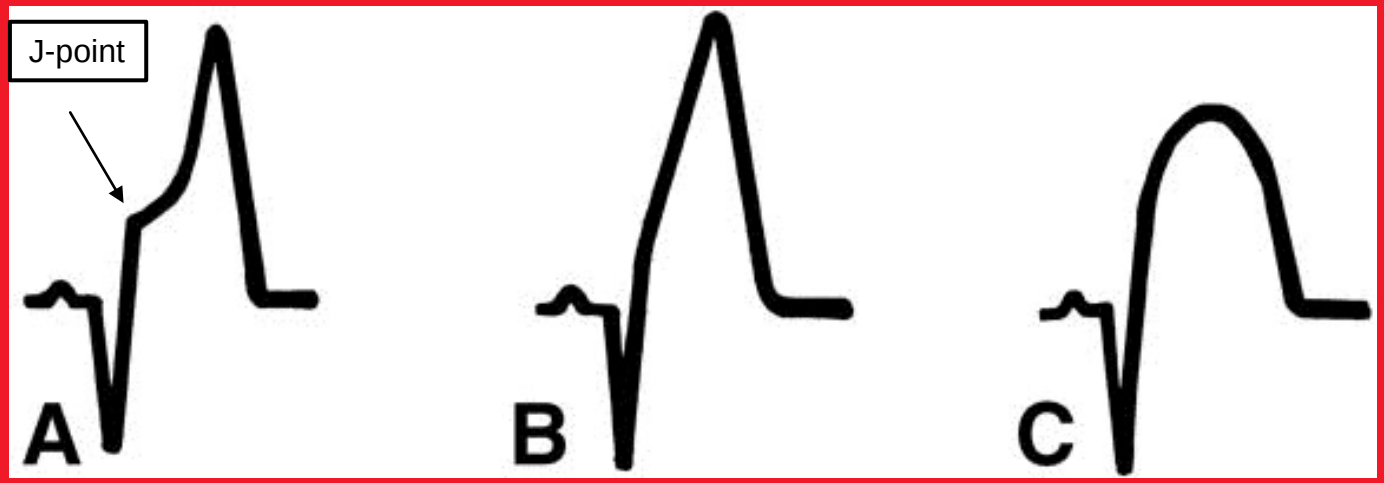


Upsloping, horizontal and downsloping ST segment depressions

- 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

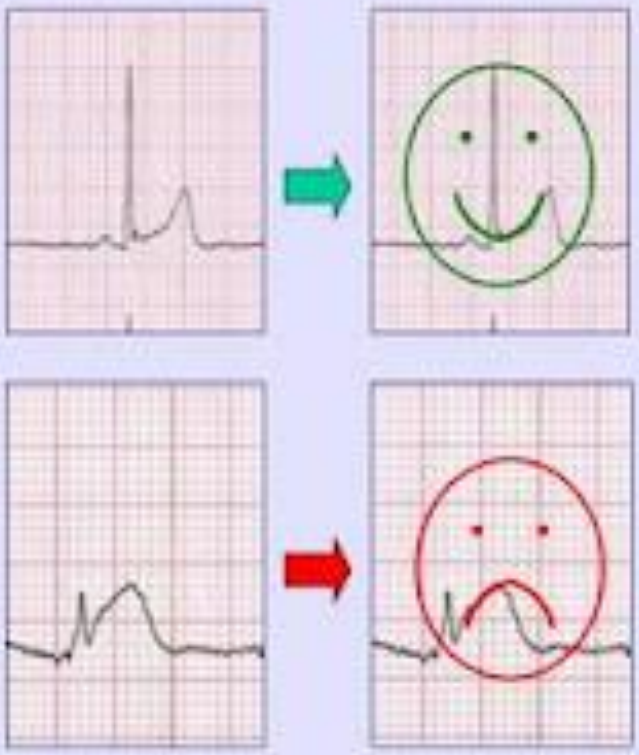


ST elevations



A – concave (often in the hypertrophy of LV)
B – straight
C – convex

acute transmural myocardial infarction



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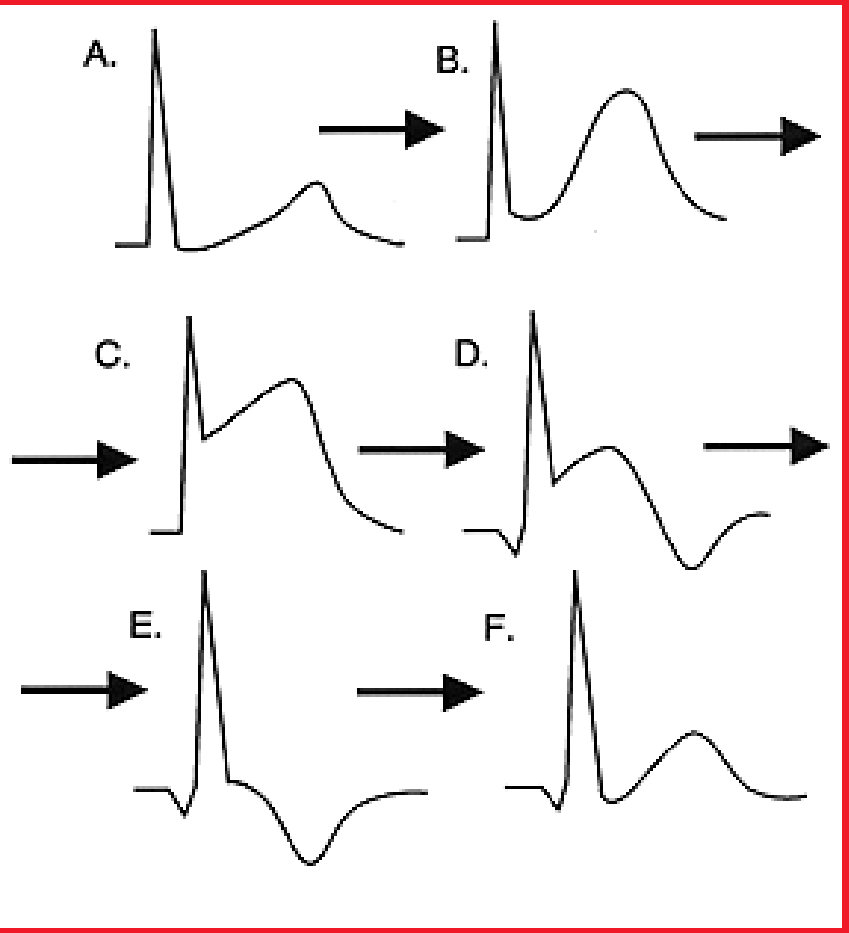
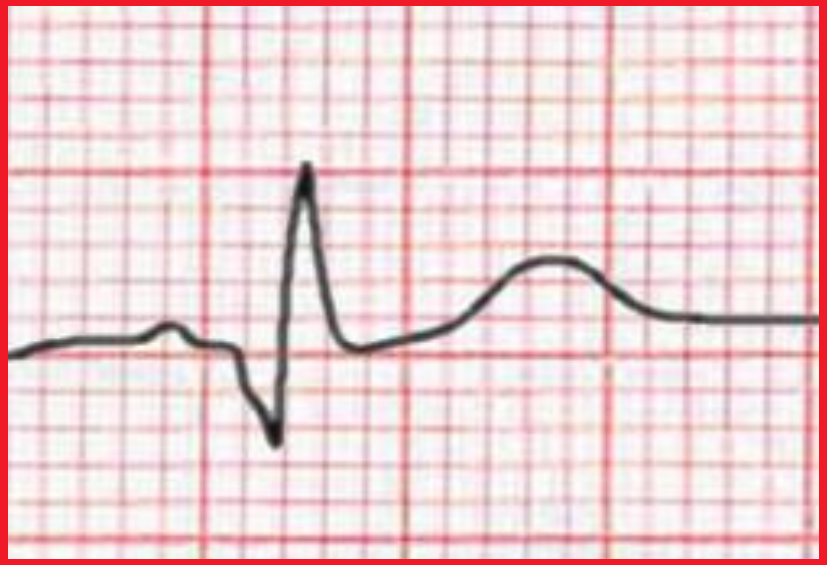
ECG changes during Q-MI

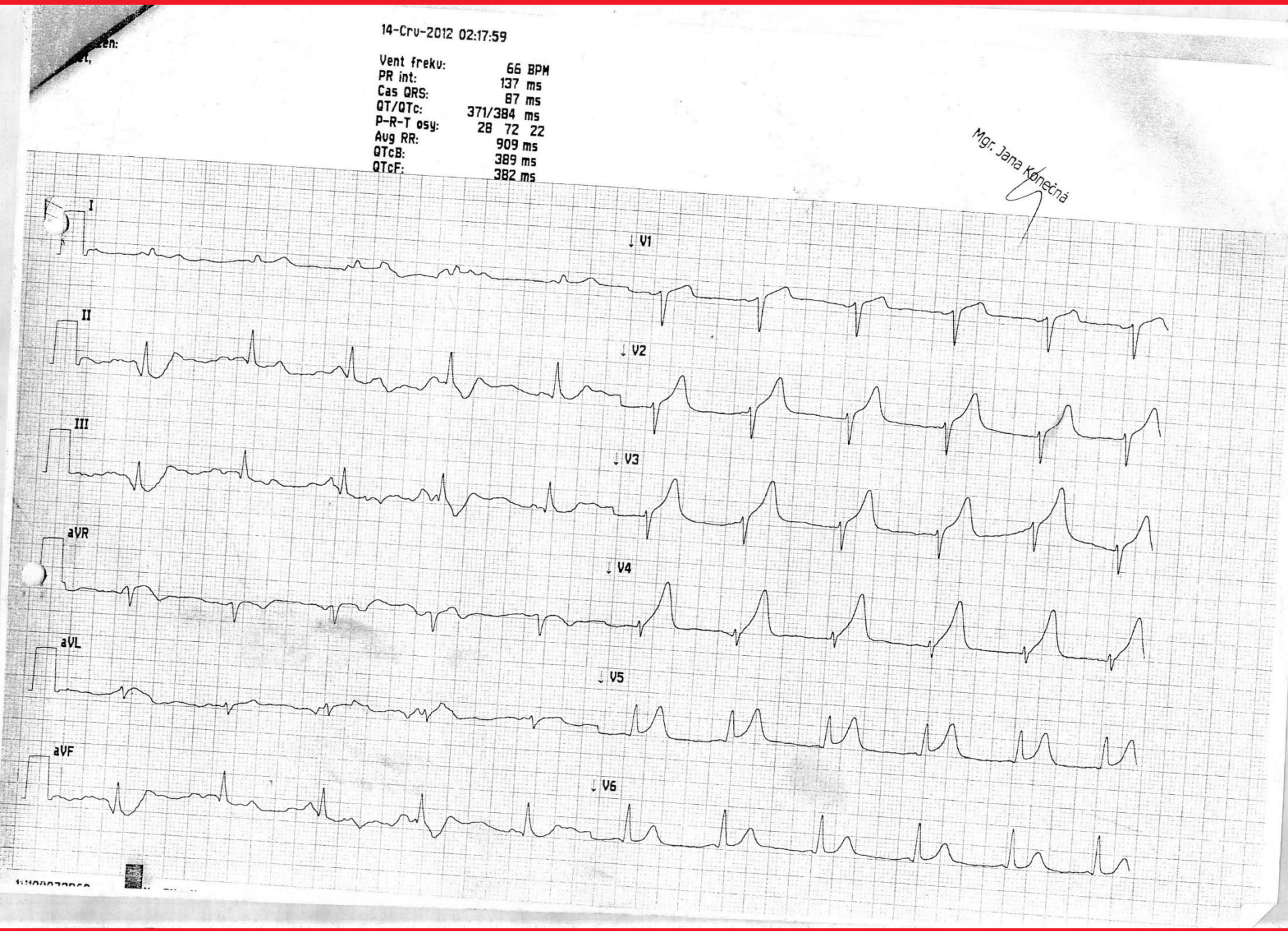


- A. initial physiological state
- B. superacute phase
  - Tall positive T waves (minutes)
- C. acute phase
  - ST elevation = Pardee's waves (tens of minutes to hours) - STEMI
- D. subacute phase
  - Normalization of ST segment
- E. Q-wave devolopment (hours to days), event. T – inversion (persists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


14-Cru-2012 02:17:59
Vent frekv: 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
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

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Vent frekv:	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

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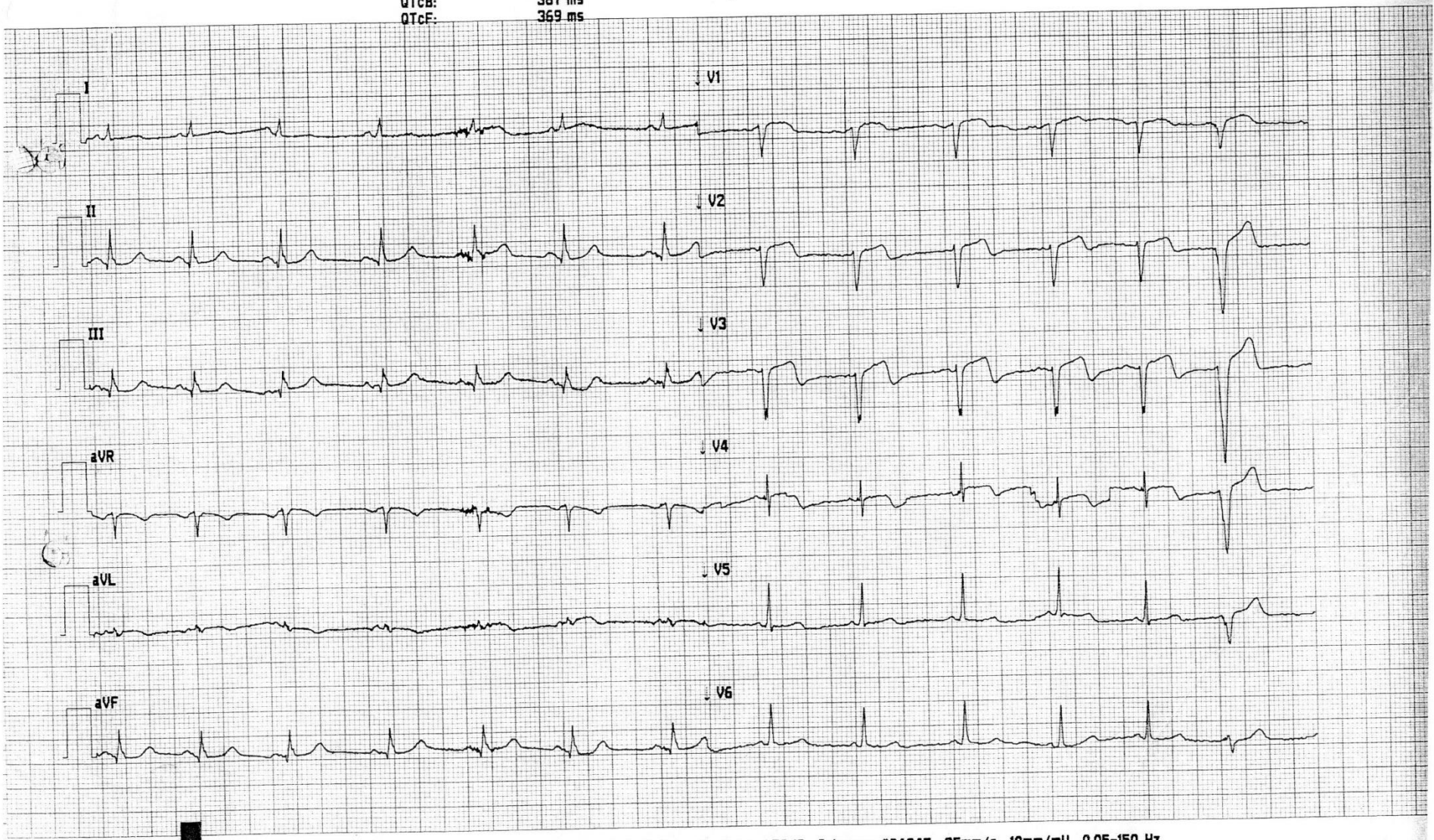

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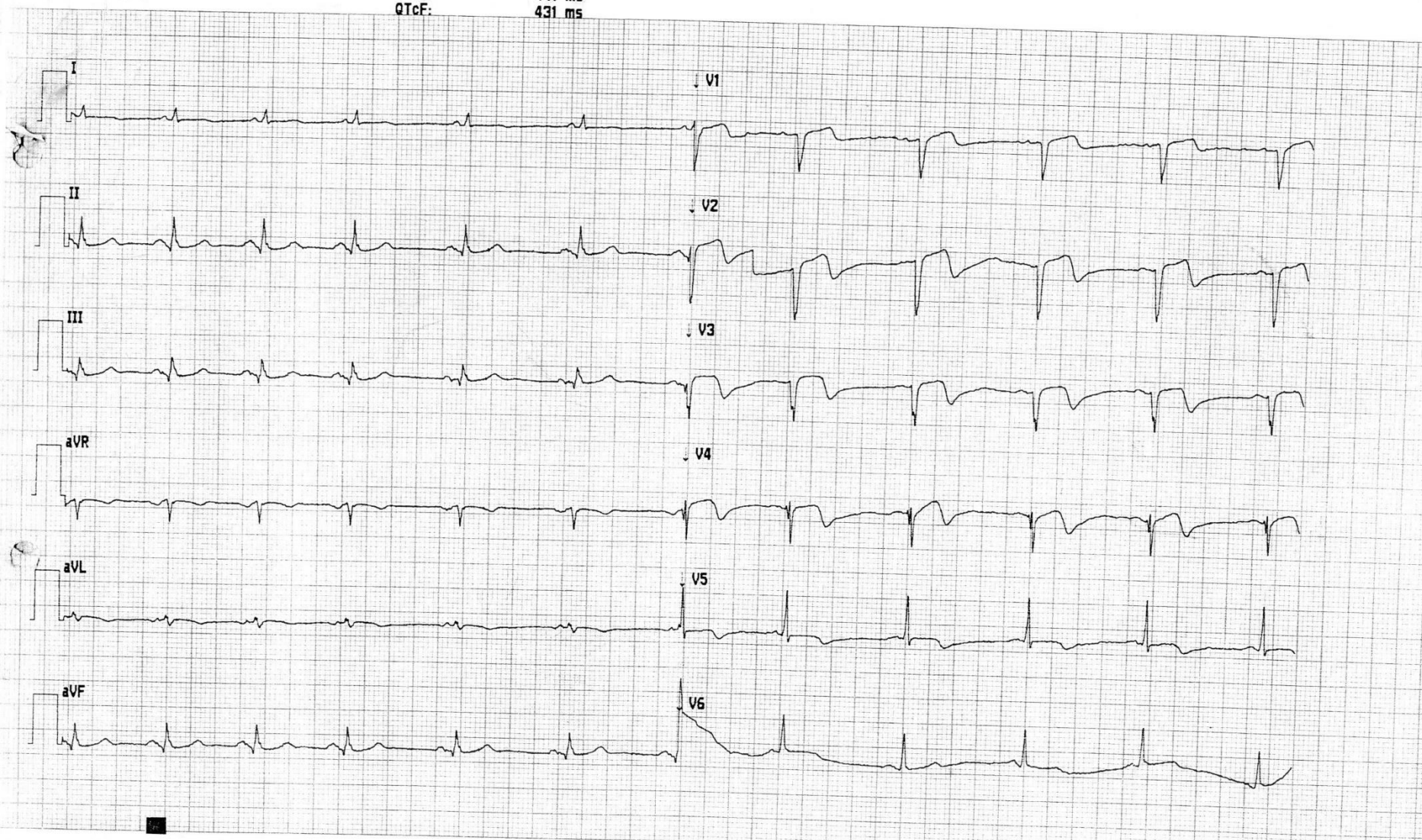
15-Cru-2012 05:28:28

Vent freku:	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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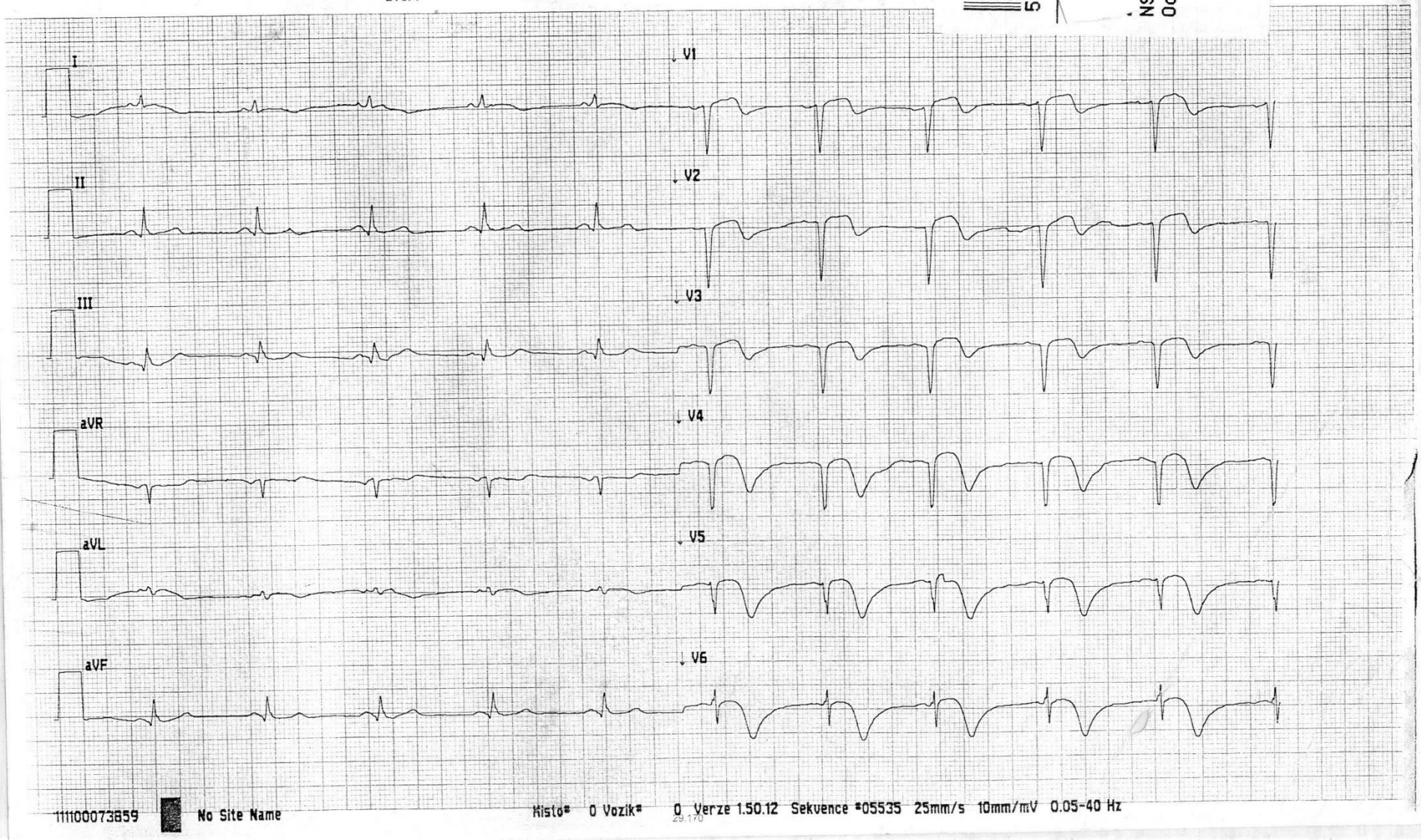
Vent frekv: 64 BPM
PR int: 141 ms
Cas QRS: 114 ms
QT/QTc: 460/469 ms
P-R-T osy: 58 62 115
Avg RR: 937 ms
QTcB: 475 ms
QTcF: 470 ms



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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