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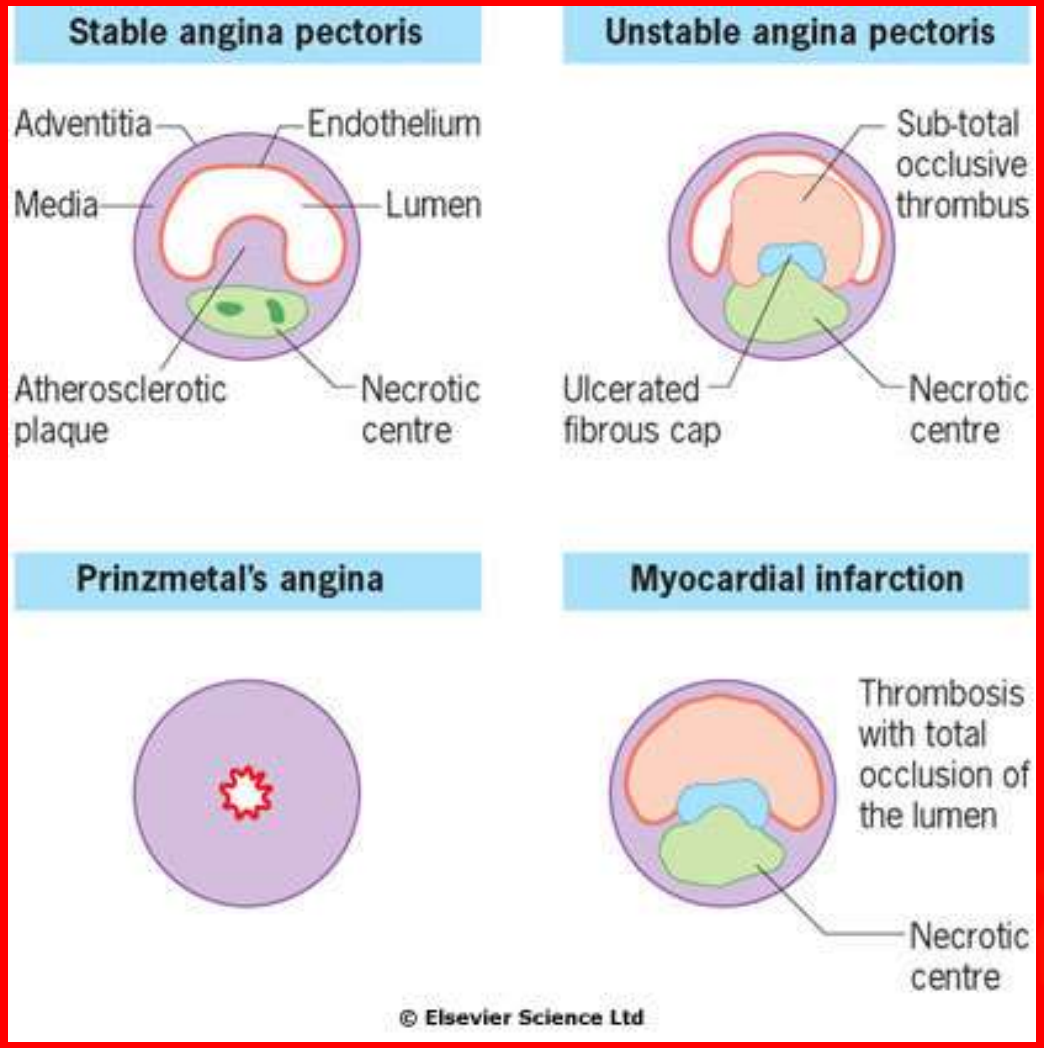
ECG in myocardial infarction and ischemia

Forms of atherosclerosis in coronary vessels:



Thrombosis

- Pathological activation of hemostasis in vascular lumen or in heart chambers
- In arteries, it is usually a consequence of vessel wall damage
- Ulceration or rupture of the fibrous cap

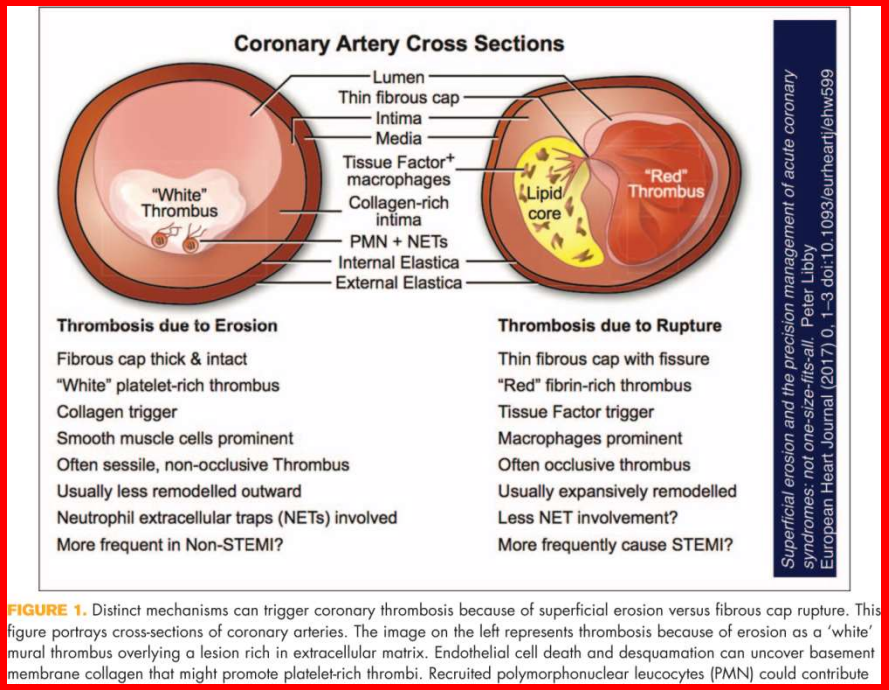
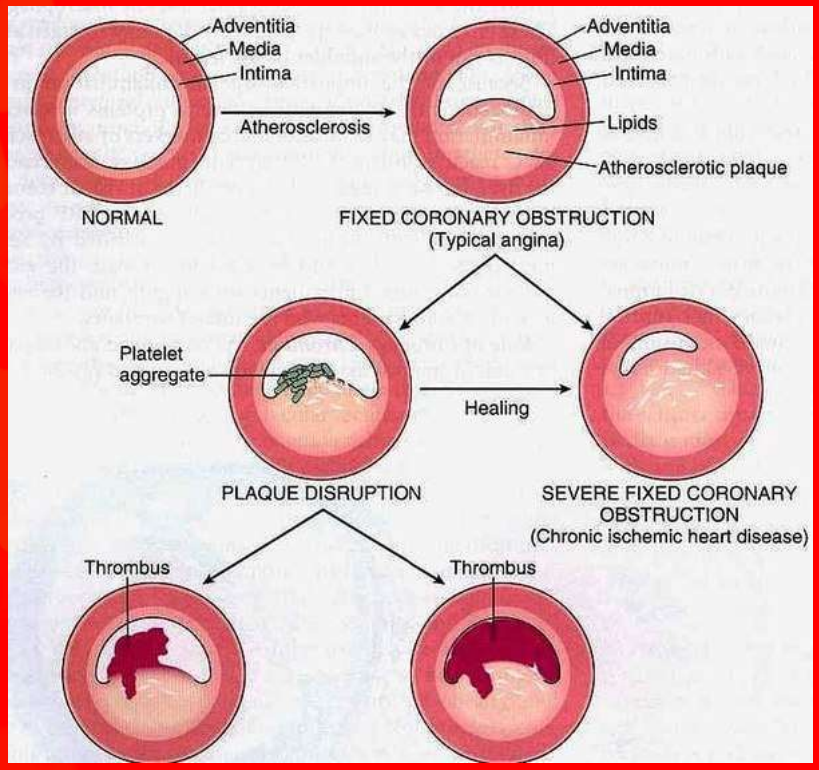


FIGURE 1. Distinct mechanisms can trigger coronary thrombosis because of superficial erosion versus fibrous cap rupture. This figure portrays cross-sections of coronary arteries. The image on the left represents thrombosis because of erosion as a 'white' mural thrombus overlying a lesion rich in extracellular matrix. Endothelial cell death and desquamation can uncover basement membrane collagen that might promote platelet-rich thrombi. Recruited polymorphonuclear leucocytes (PMN) could contribute

Acute myocardial infarction (AMI)

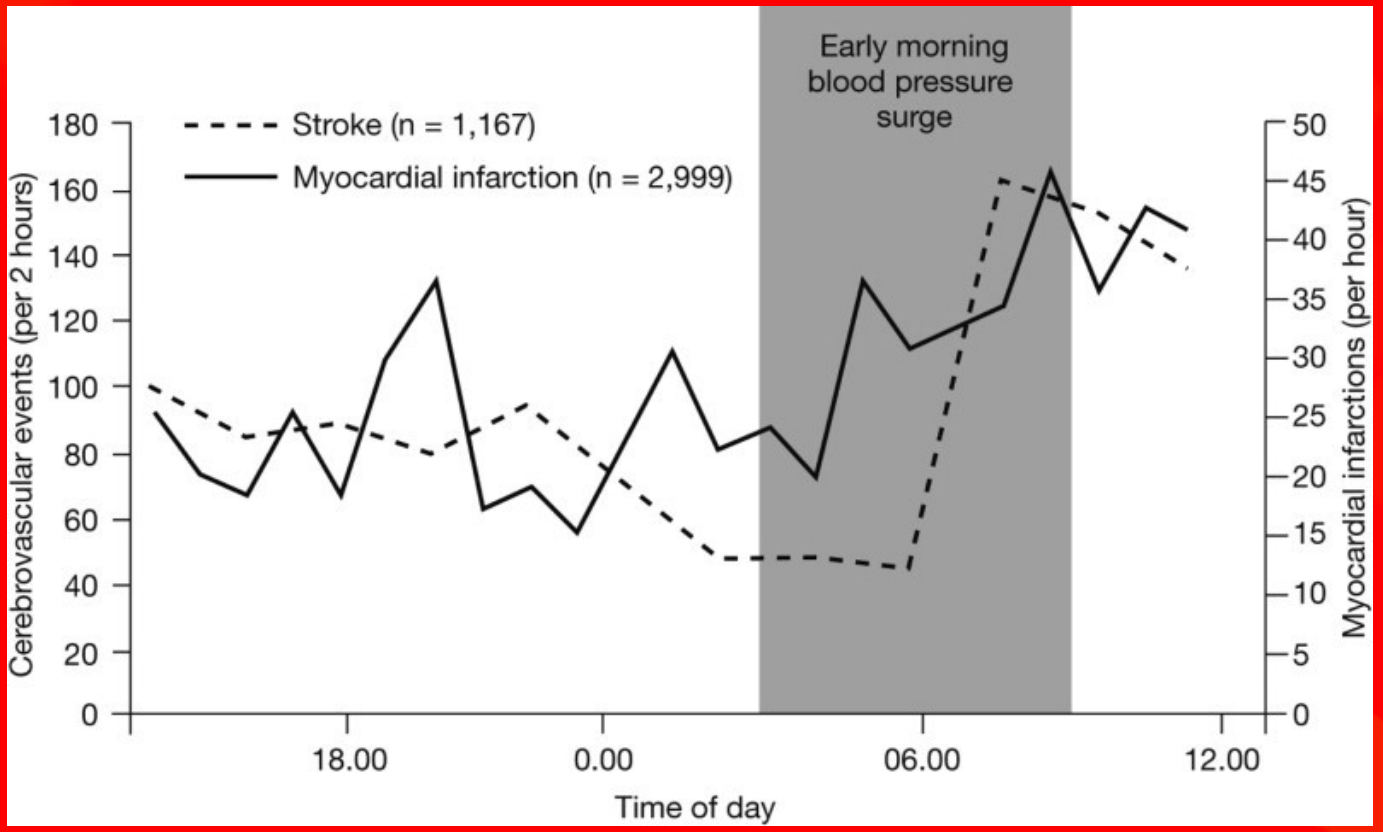
- Generally, the term „infarction“ can be used for any local acute ischemia with necrosis, irrespectively of affected organ
- However, myocardial and cerebral infarction most usually lead to death or invalidity
- Myocardial infarction is the most common life threatening complication of coronary atherosclerosis
- In most cases, its cause is a rupture of unstable atherosclerotic plaque with subsequent thrombosis
- Other main causes: plaque ulceration, calcified nodule
- Rarer causes independent of atherosclerosis: in-stent thrombosis, 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…)

Stable and unstable plaque in IHD



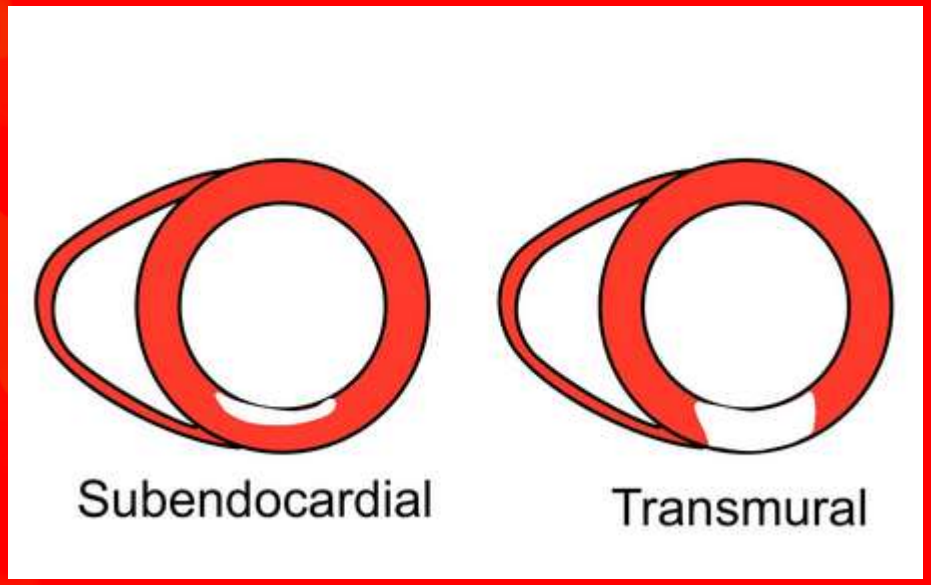
- Stable angina pectoris
  - Chest pain during effort
- Acute coronary syndrome
  - Unstable angina pectoris
    - accelerated AP, or pain at rest, diminished reaction to vasodilatants
    - Form of acute coronary syndrome
  - Minimal myocardial damage
    - „grey zone" - chest pain + laboratory markers of MI
    - No ECG finding or impaired contractility in imaging methods
  - Non-STEMI
    - theoretically ~ non-QIM ~ subendocardial IM)
  - STEMI
    - theoretically ~ QIM ~ transmural IM)

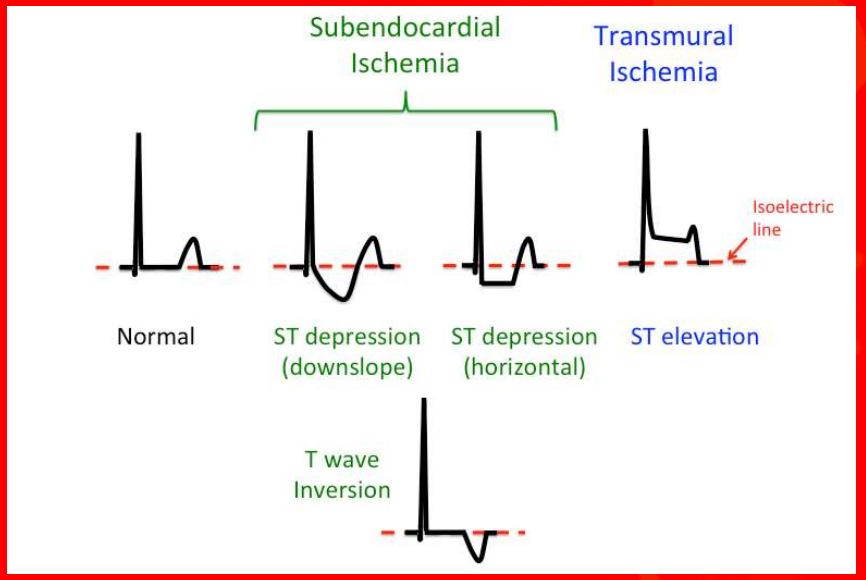
AMIs and strokes during the day



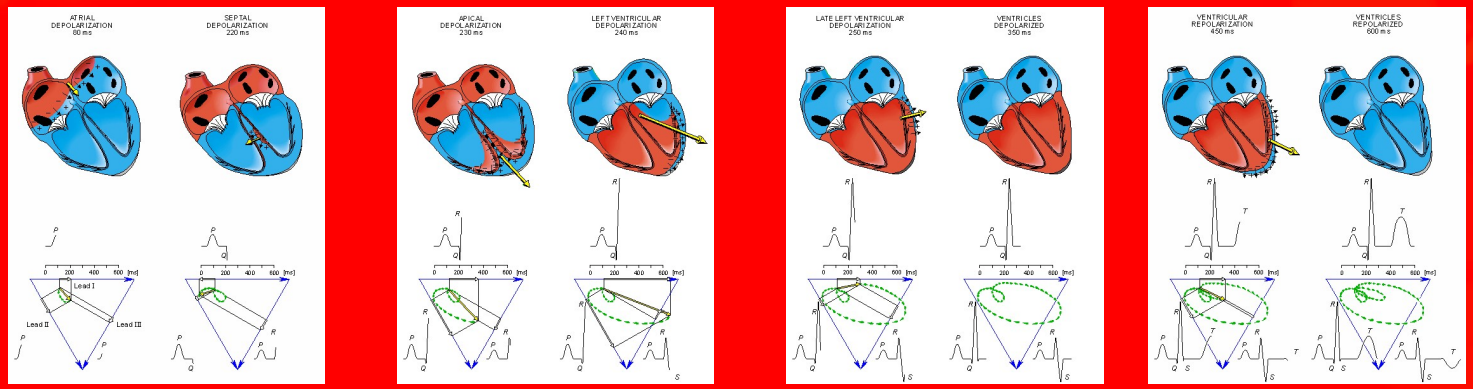
- Higher incidence of cerebral and myocardial infarctions in the morning is caused with higher activity of sympathetic nervous system and higher blood pressure in morning hours

Changes of ST segment during myocardial infarction





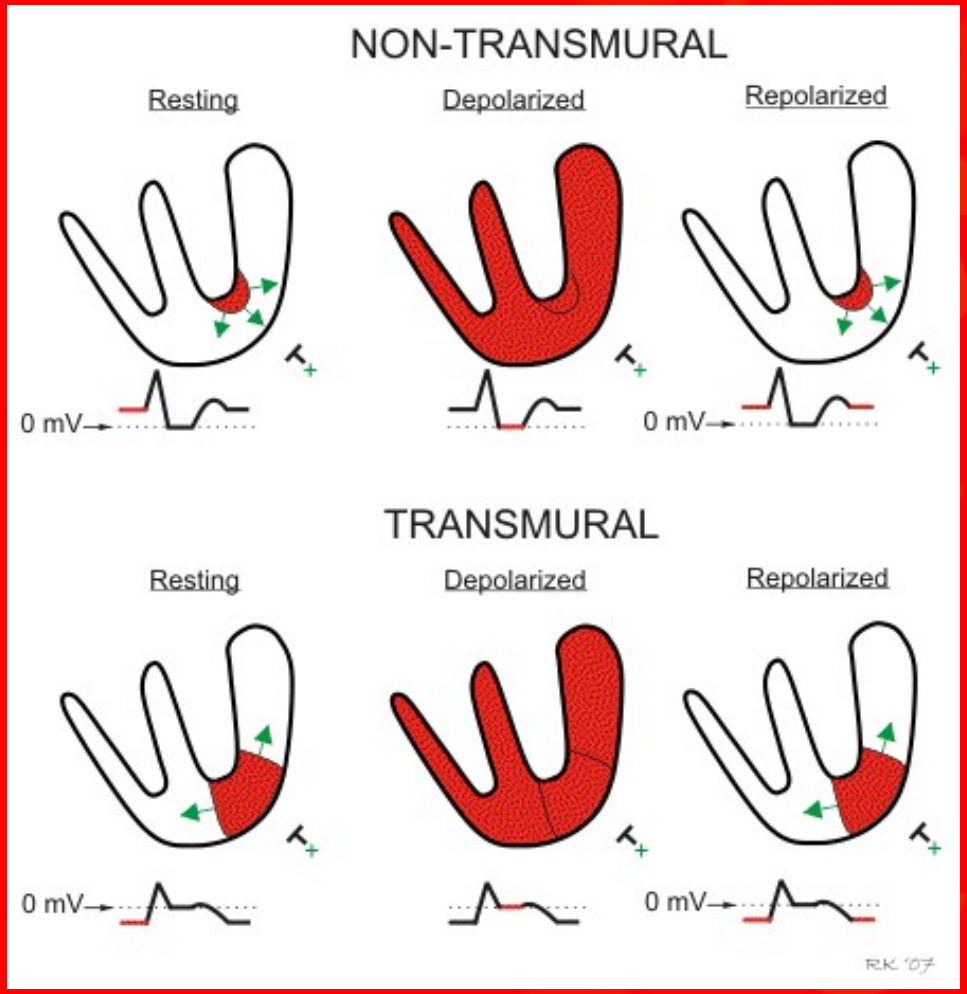
Subendocardial vs. subepicardial myocardium



- 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)

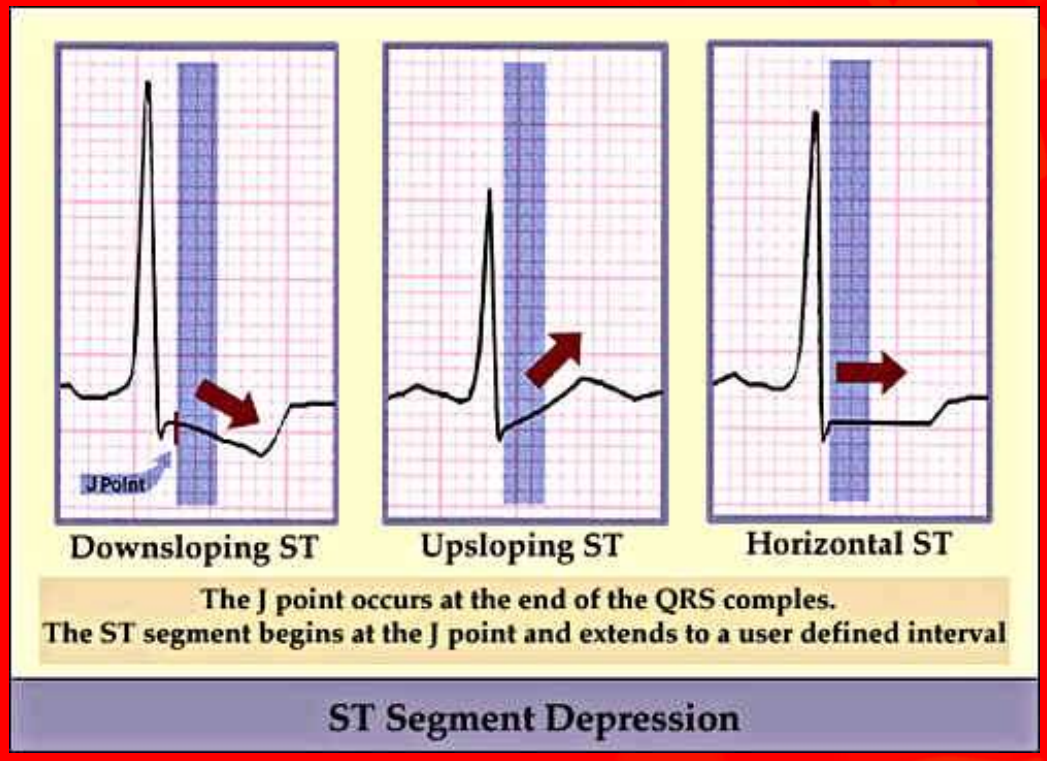
Changes of ST segment 2

- ST segment position changes relatively to isoelectric line (T-P segment)
- During diastole, an ischemic focus generates depolarizing diastolic injury 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 T-P segment in opposite direction
- The differences in the plateau phase and repolarization lead into different shape of ST segment (systolic injury currents) and contribute to ST shifts.

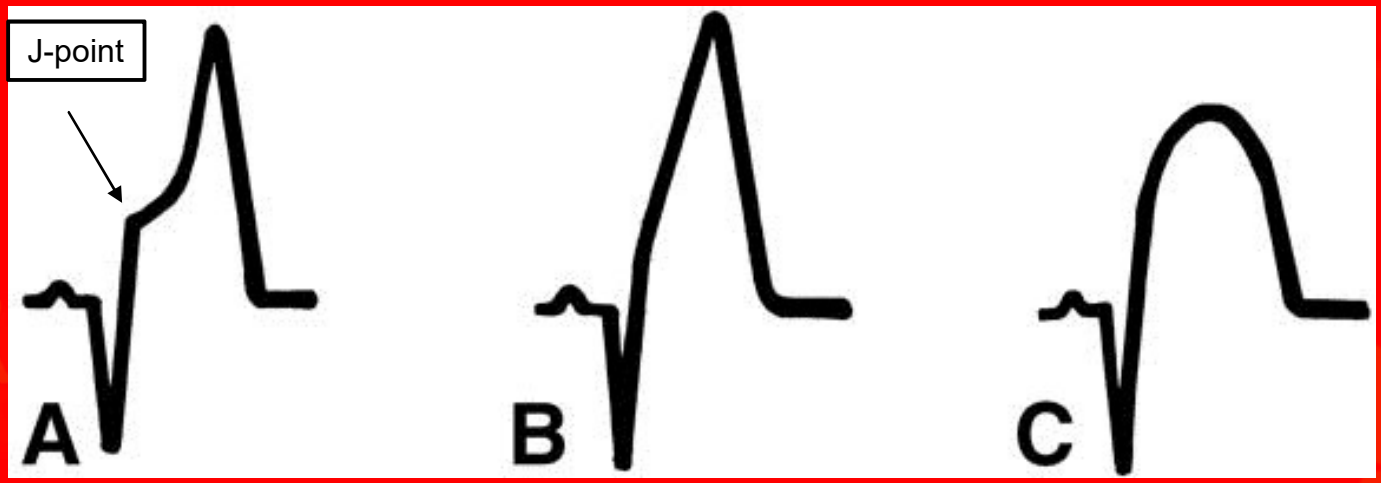


Upsloping, horizontal and downsloping ST segment depressions

- 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

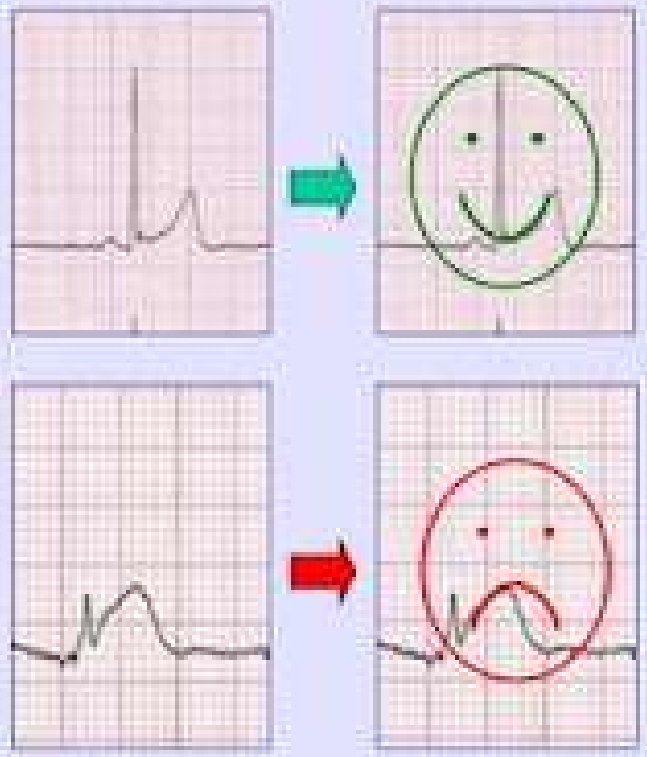


ST elevations



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

acute transmural myocardial infarction



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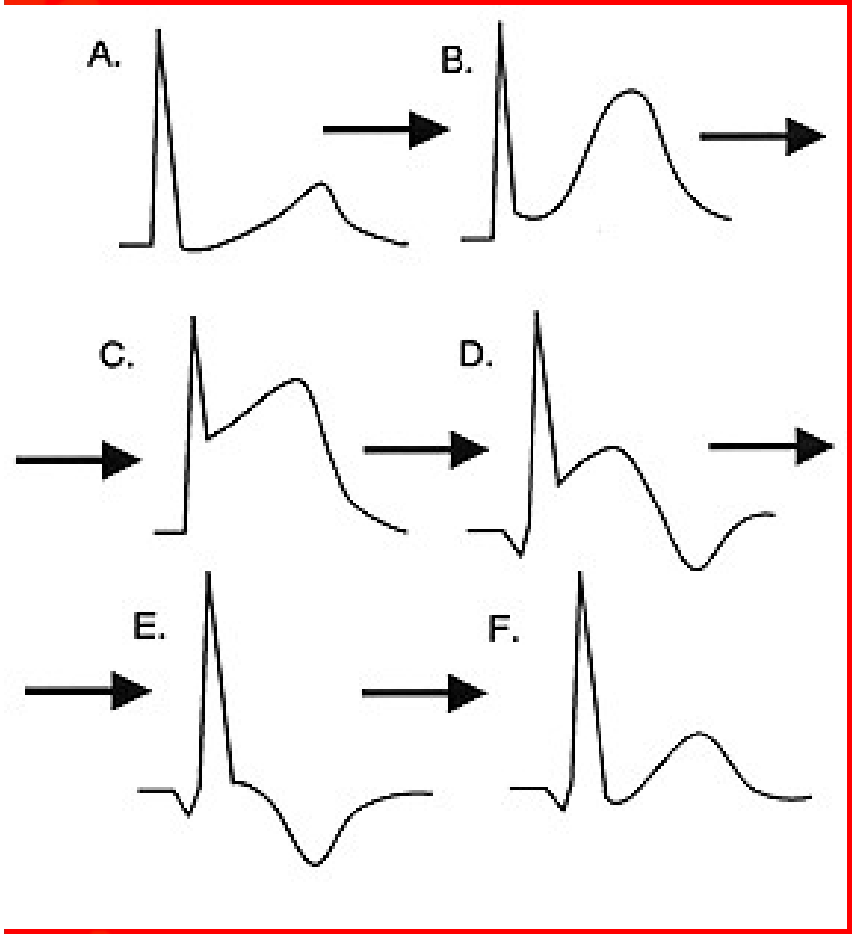
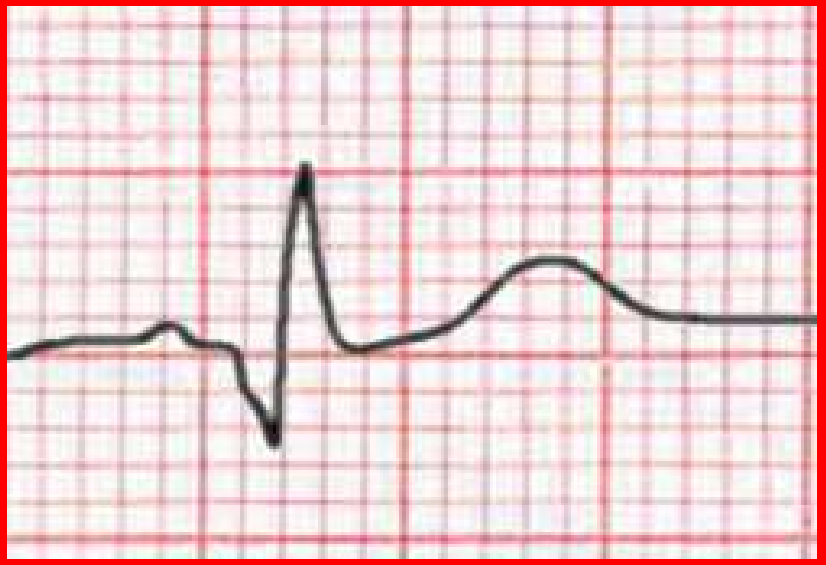
ECG changes during Q-MI

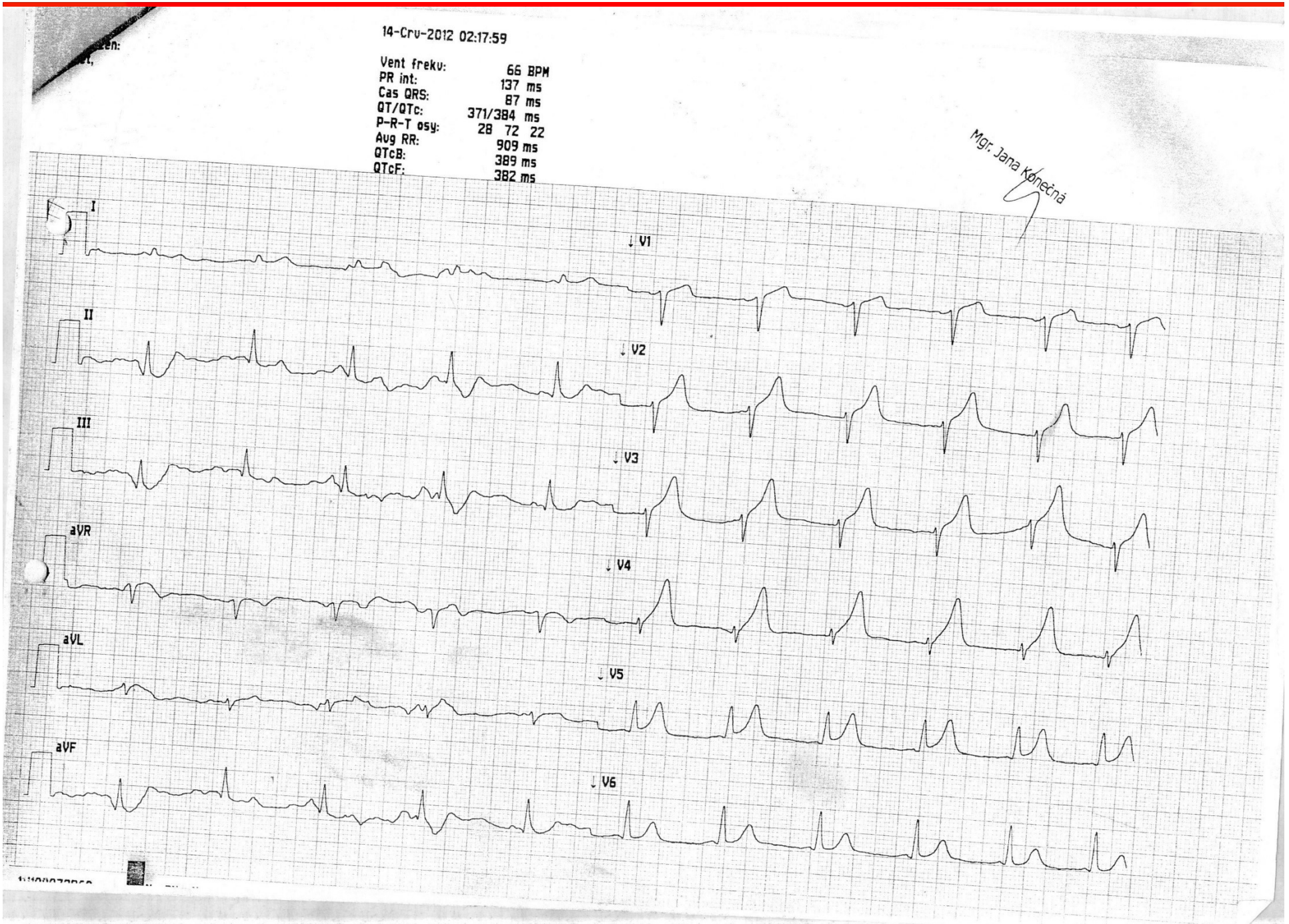


- A. initial physiological state
- B. superacute phase
  - Tall positive T waves (minutes)
- C. acute phase
  - ST elevation = Pardee's waves (tens of minutes to hours) - STEMI
- D. subacute phase
  - Normalization of ST segment
- E. Q-wave devolopment (hours to days), event. T – inversion (persists 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)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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
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

14-Cru-2012 20:35:15

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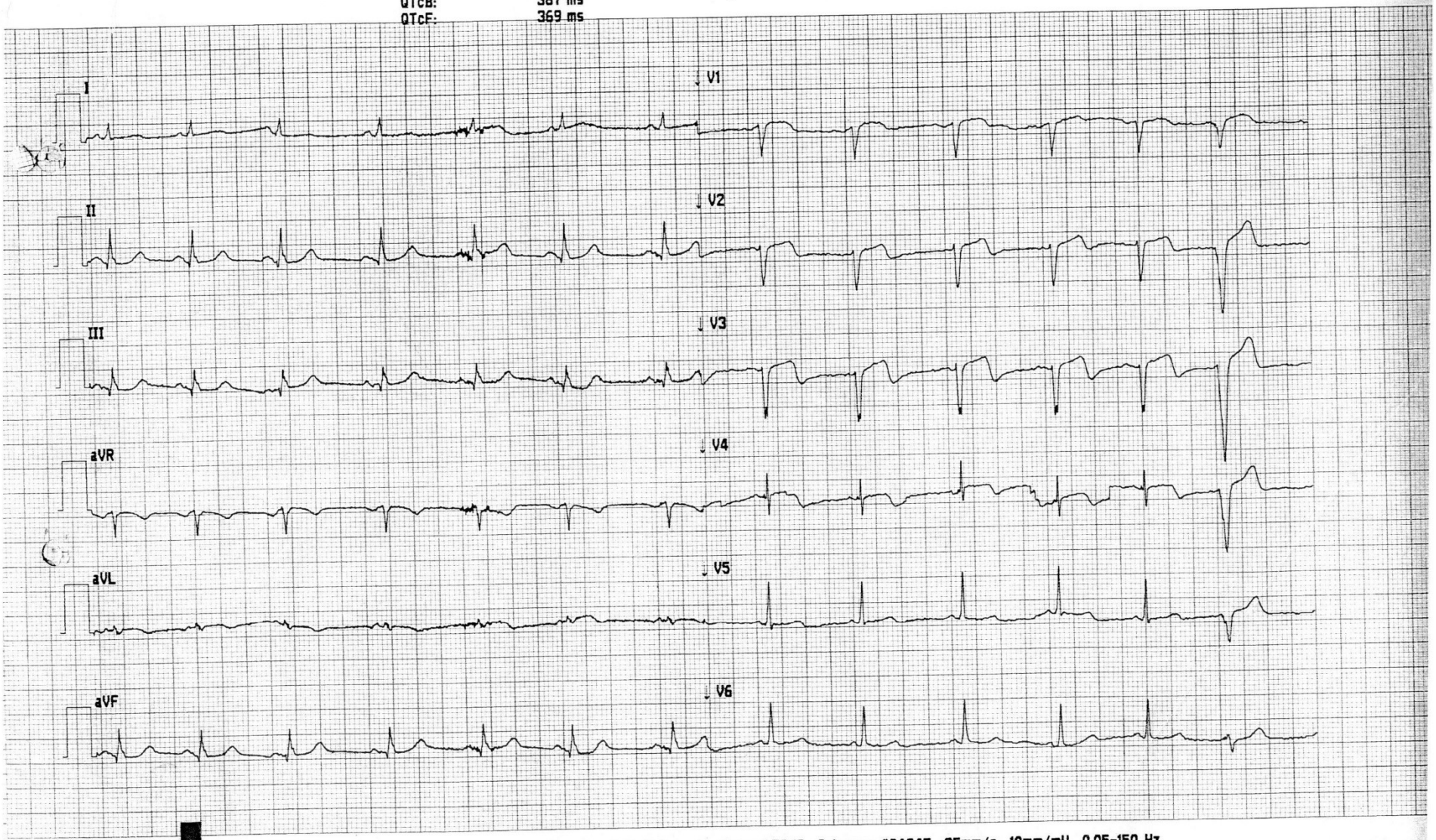
Vent freku:	79 BPM
PR int:	134 ms
Cas QRS:	85 ms
QT/QTc:	337/371 ms
P-R-T osy:	55 59 78
Avg RR:	758 ms
QTcB:	387 ms
QTcF:	369 ms

MIŠKAIOVÁ Jana Dis.



111030069619 [redacted] No Site Name 28.939 Misto* 0 Vozik* 0 Verze 1.50.12 Sekvence *04247 25mm/s 10mm/mV 0.05-150 Hz

15-Cru-2012 05:28:28

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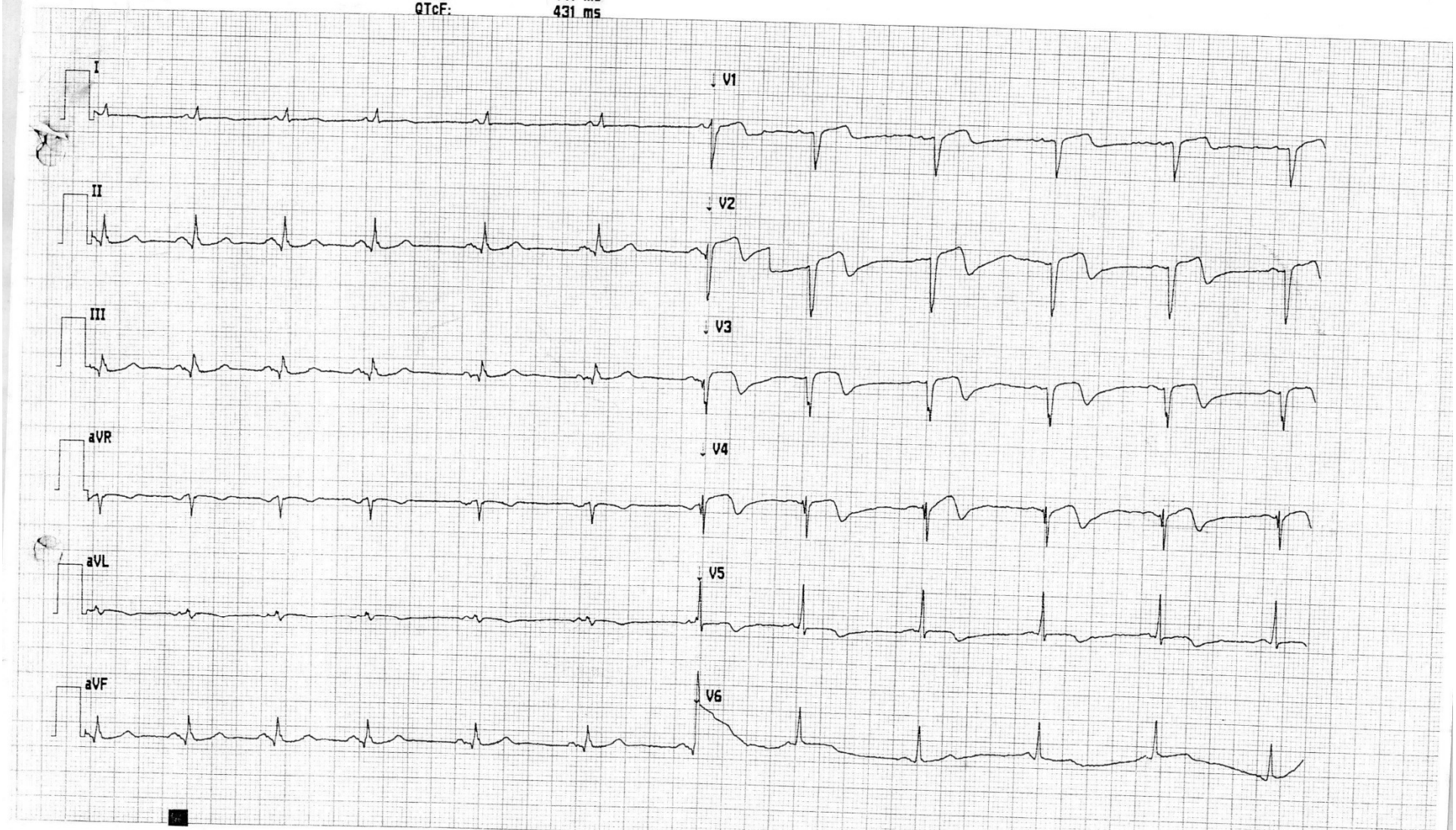
Vent freku:	68 BPM
PR int:	134 ms
Cas QRS:	90 ms
QT/QTc:	413/430 ms
P-R-T osy:	56 62 97
Avg RR:	875 ms
QTcB:	441 ms
QTcF:	431 ms



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16-Dec-2012 11:45:33

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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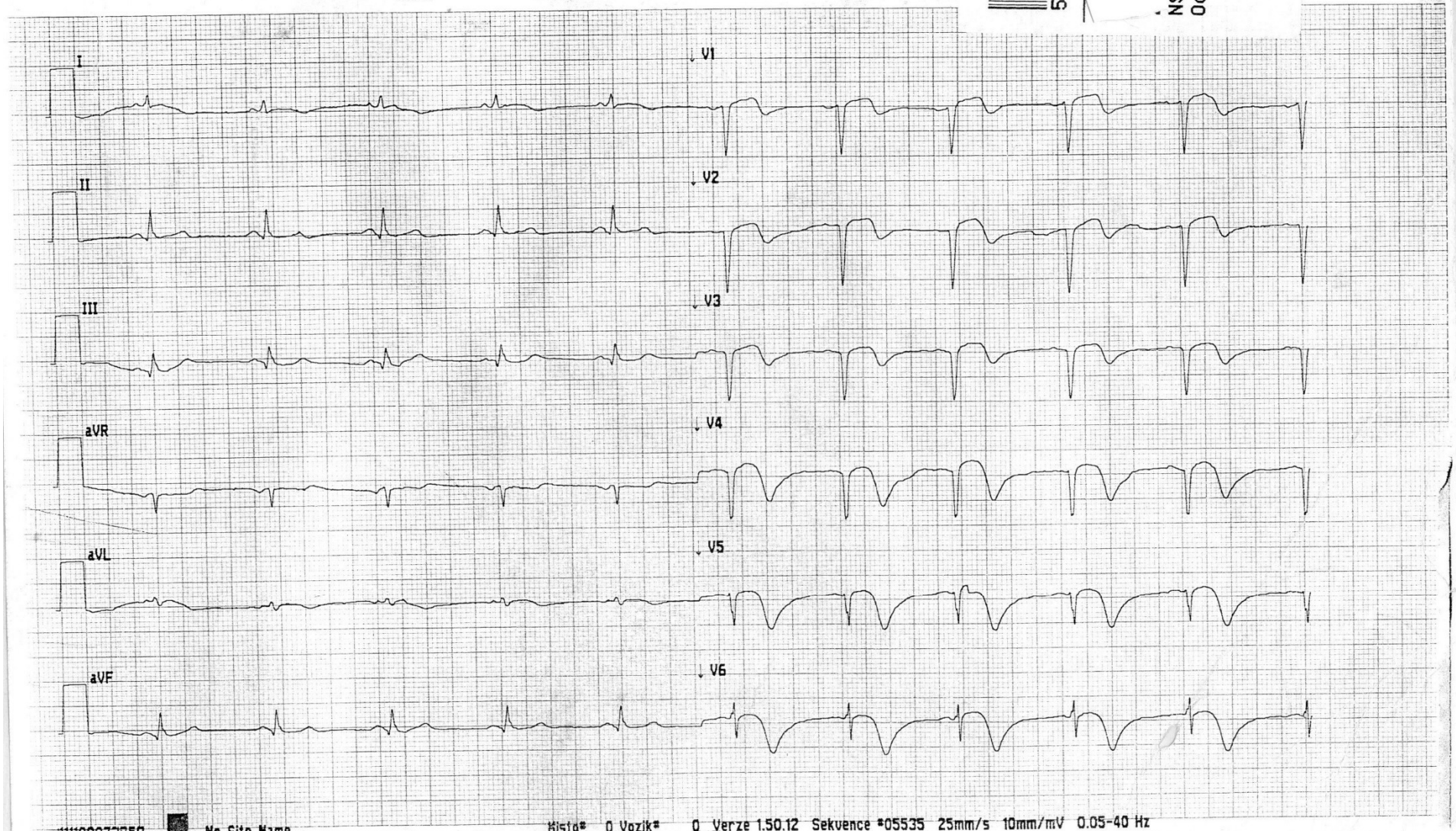

111100073859 No Site Name Kisto* 0 Vozik* 0 Verze 1.50.12 Sekvence *05535 25mm/s 10mm/mV 0.05-40 Hz
29.170

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