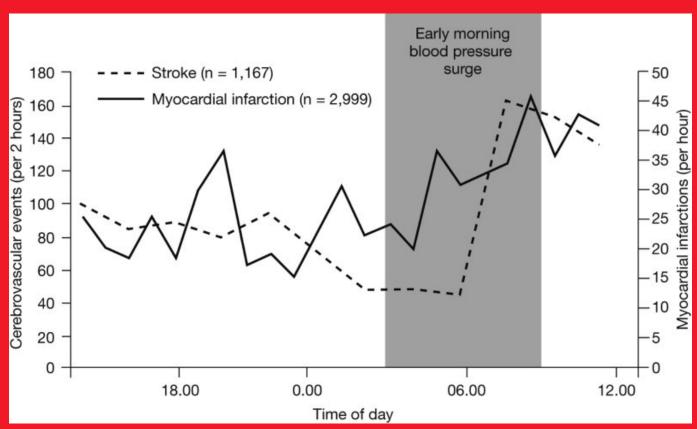


Acute myocardial infarction (AMI)

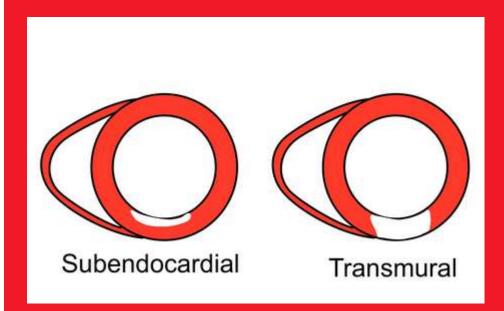
- Generally, the term "infarction" can be used for any local acute ischemia with necrosis, irrespectively of affected organ
- However, myocardial and cerebral infarction most usually lead to death or invalidity
- Myocardial infarction is the most common life threatening complication of coronary atherosclerosis
- 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 Ca²⁺, 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...)

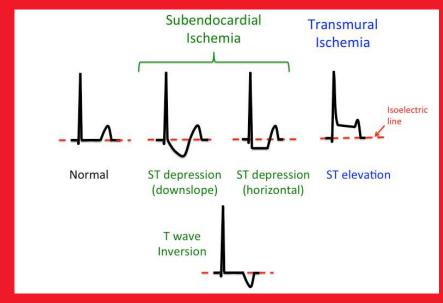
AMIs and strokes during the day



- 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

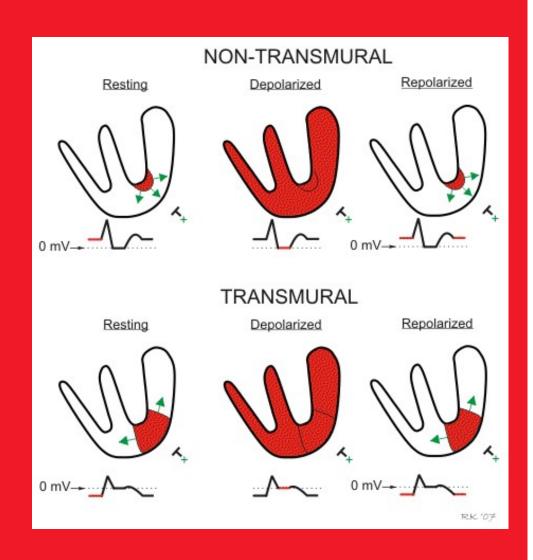
Changes of ST segment during myocardial infarction





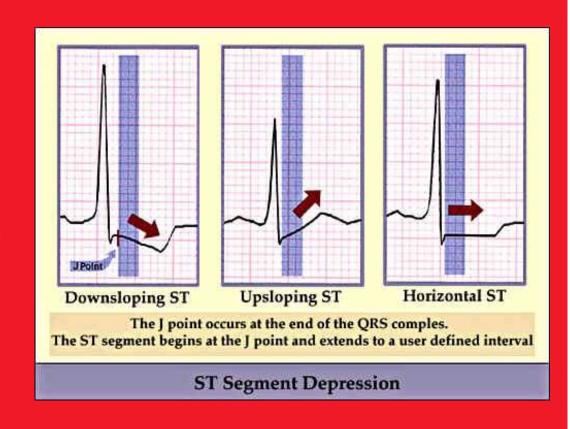
Changes of ST segment 2

- 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

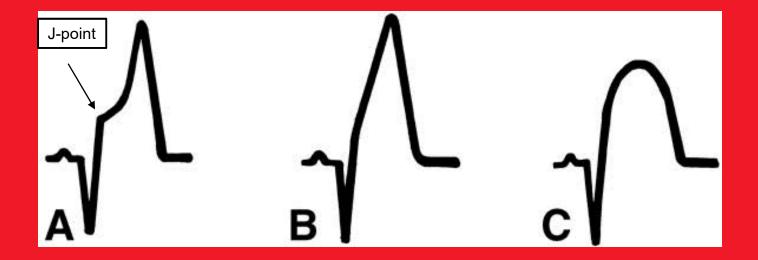


Upsloping, horizontal and downsloping ST segment depressions

- 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



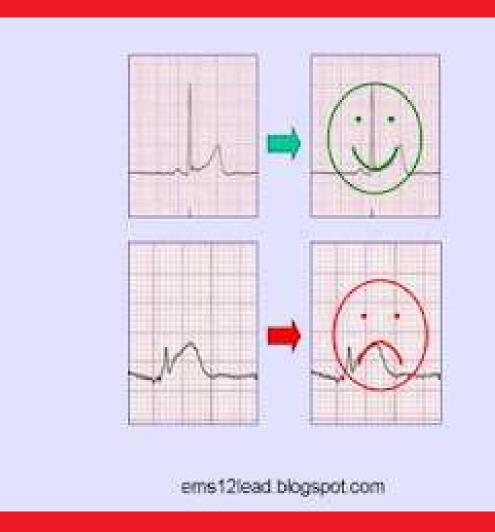
A – concave (often in the hypertrophy of LV)

B – straight

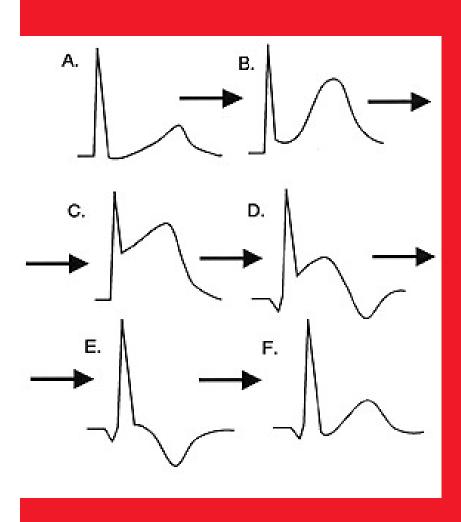
acute transmural myocardial

C – convex

infarction

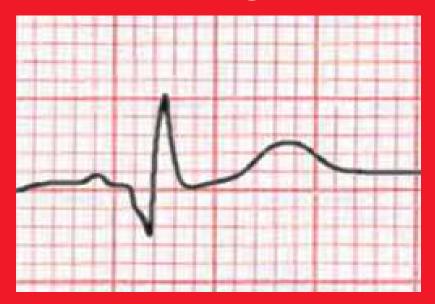


ECG changes during Q-MI

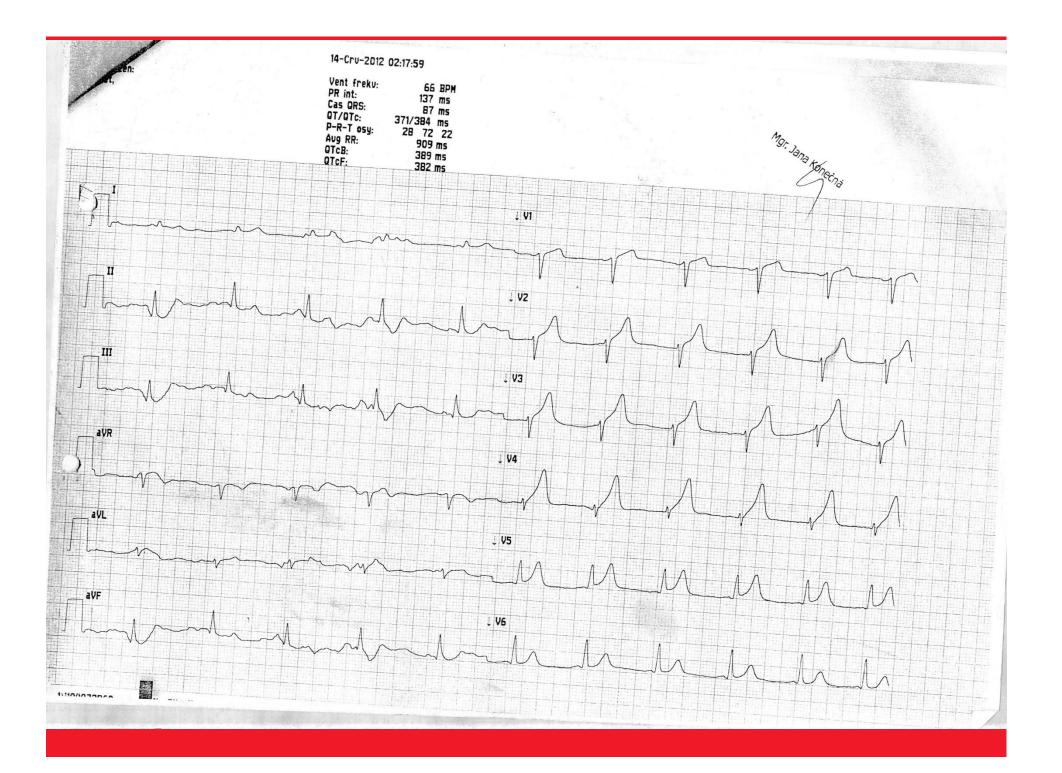


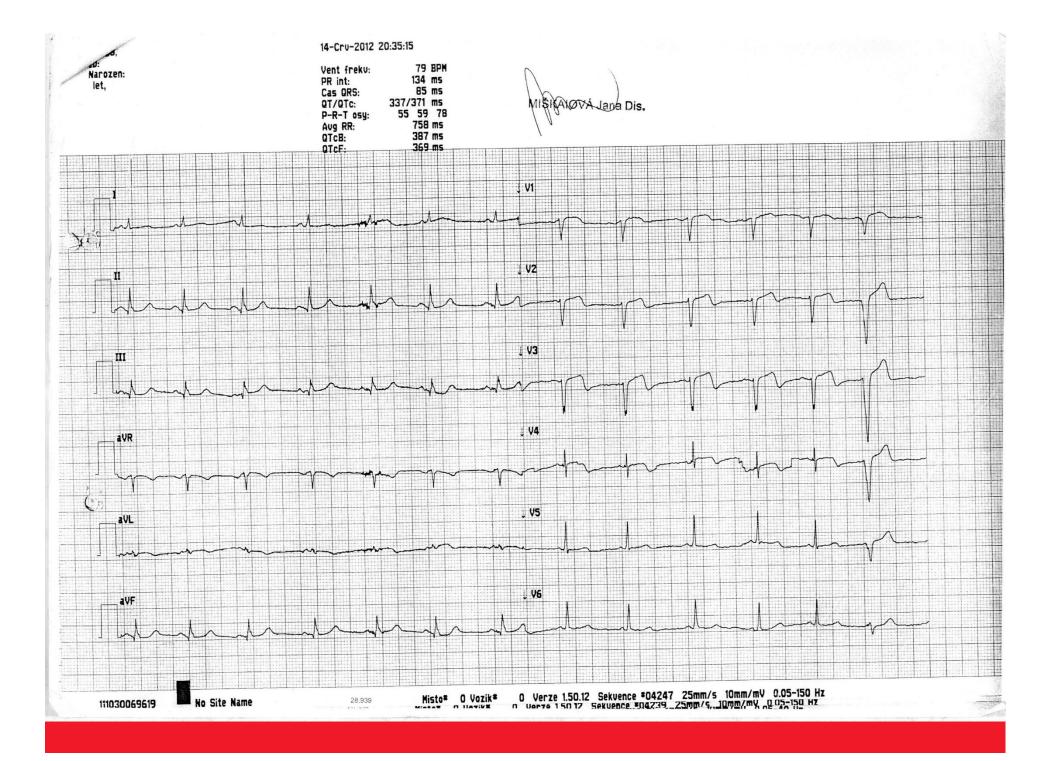
- A. initial physiological state
- B. superacute phase
 - Tall positive T waves (minutes)
- C. acute phase
 - ST elevation = Pardee's waves (tens of minutes to hours) - STEMI
- D. subacute phase
 - Normalization of ST segment
- E. Q-wave devolopment (hours to days), event. T – inversion (persists weeks)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

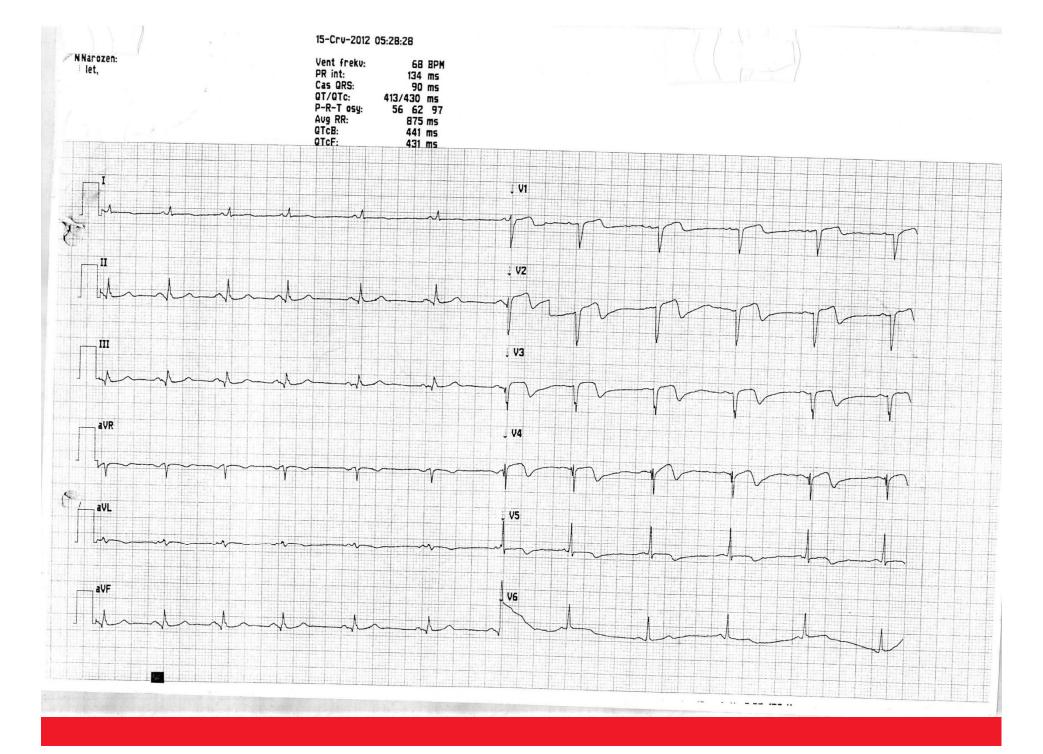
Pathologic Q

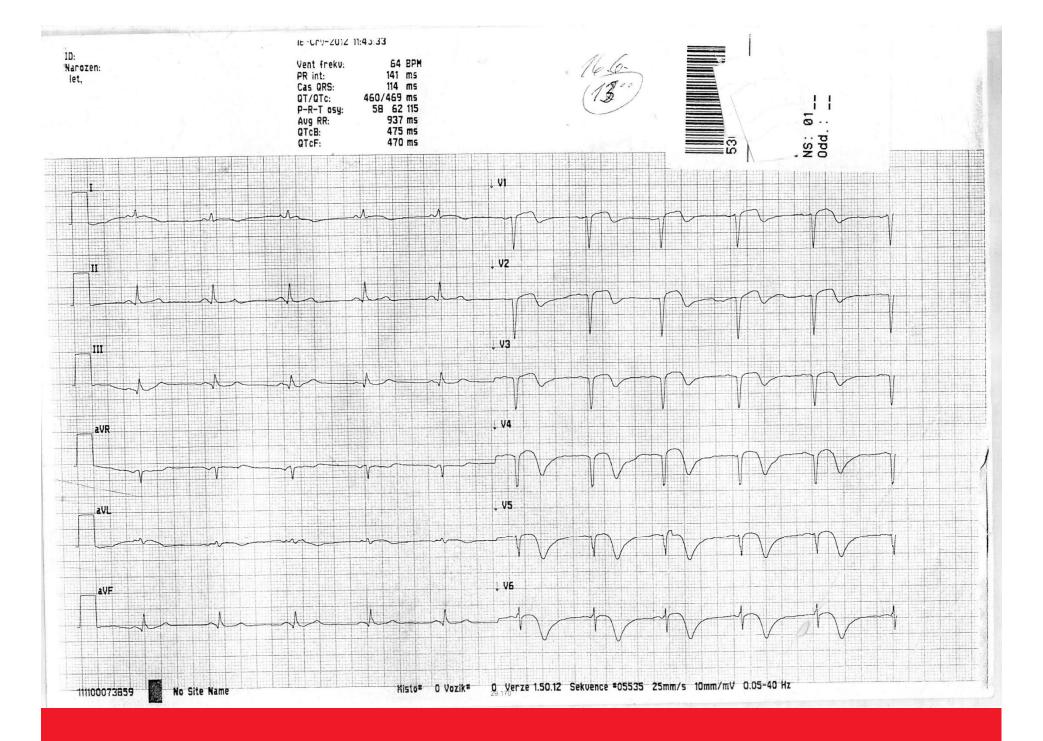


- During several hours after transmural MI, pathologic Q develops
- Pathologic Q corresponds to depolarization of opposing cardiac wall, observed through electrically dead tissue – a scar
- Its depth is > ¼ R (or R is not present at all QS wave) and its duration is at least 40 ms)
- It usually persists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









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