


ANGINA PECTORIS

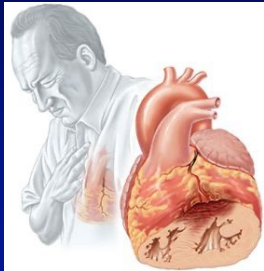
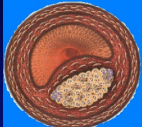
MYOCARDIAL INFARCTION

UNIVERSITY OF DEBRECEN
FACULTY OF MEDICINE
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PATHOPHYSIOLOGY OF MYOCARDIAL ISCHAEMIA

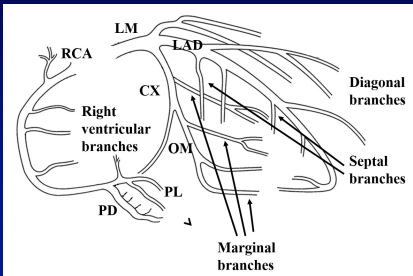
Atherosclerotic plaque



Myocardial necrosis (infarction)

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THE CORONARY SYSTEM



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ANGINA PECTORIS:

Constricting, oppressing chest pain

Typical ischaemic ECG alterations but NO necroenzymes in plasma

Explanation: atherosclerotic plaque + increased O₂ demand

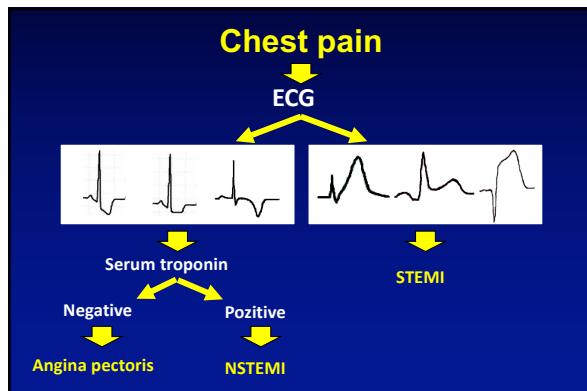
MYOCARDIAL INFARCTION:

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

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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.

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HYPOXIA INDUCED REVERSIBLE ALTERATIONS

ISCHAEMIA

SUBENDOCARDIAL

SUBEPICARDIAL

LAESION

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

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TYPICAL T WAVE ALTERATIONS

NORMAL	ISOELECTRIC	BIPHASIC	NEGATIVE
TALL, PEAKY („HYPERACUT“) T WAVE			DEEP, PEAKY, SYMMETRIC („CORONARY“) T WAVE
THEY MAY INDICATE ISCHAEMIA!			
Differential-diagnosis: change in autonomic tone (norm.) change in plasma K ⁺ level peri- et myocarditis secunder alterations (e.g. BBB)			

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TYPICAL ST ALTERATIONS

ASCENDING	HORIZONTAL	ST-SCOOPING	DESCENDING	
CONCAVE	SADDLE SHAPED	EARLY REPOL.	CONVEX	
Differential-diagnosis: Normal variant, ion disturbance, digitalis effect peri- et myocraditis, secunder (e.g. BBB)				

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Q WAVE

NORMAL FEATURE:

q
QS
positional Q

I, aVL, V5-V6 / II, III, aVF
aVR, V1
III

MYOCARDIAL INFARCTION, PATHOLOGICAL Q WAVE:—

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.

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THE DYNAMICS OF MYOCARDIAL INFARCTION (STEMI)

HYPERACUT PHASE (during the first minutes):
Tall, peaky, „tentet“ (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

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ECG patterns of myocardial infarction (STEMI)

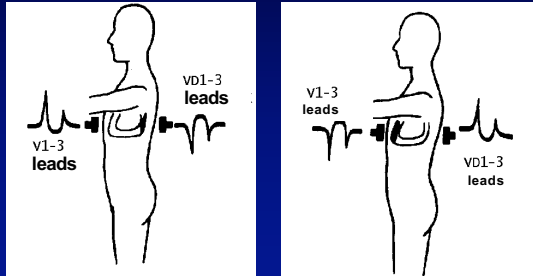
Zone of myocardial ischemia

Zone of myocardial infarction

Zone of myocardial injury

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INTERPRETATION OF ECG MIRROR SIGNS



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LOCALIZATION OF THE MYOCARDIAL INFARCTION

ANTERIOR (ANTEROSEPTAL): V1-V4
ANTEROLATERAL: I, aVL, V5-V6
EXTENSIVE ANTERIOR: I, aVL, V1-V6
HIGH LATERAL: I, aVL

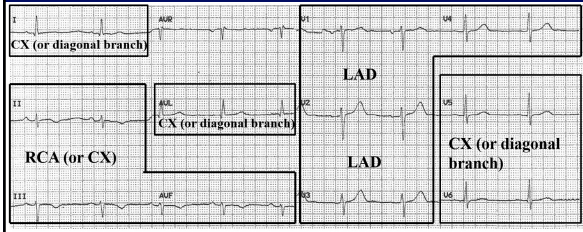
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LOCALIZATION OF THE MYOCARDIAL INFARCTION

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)

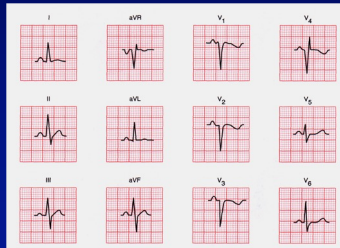
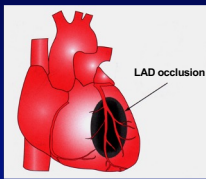
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LOCALIZATION OF THE MYOCARDIAL INFARCTION



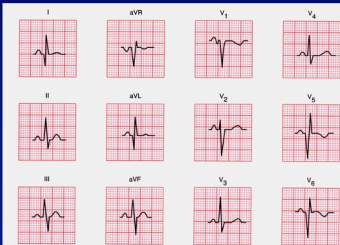
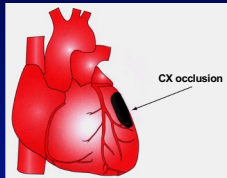
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Anterior myocardial infarction



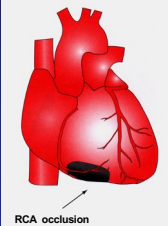
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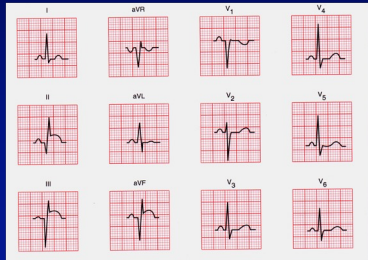
Lateral myocardial infarction



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Inferior myocardial infarction






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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 acute phase with necroenzyme level elevations and disturbances in the wall movement (echo!).
- It can be considered as an unfinished event!

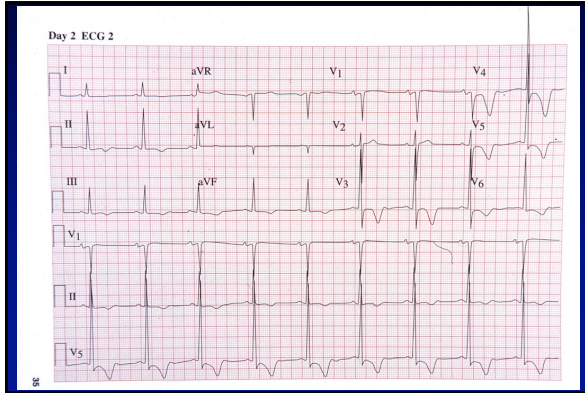


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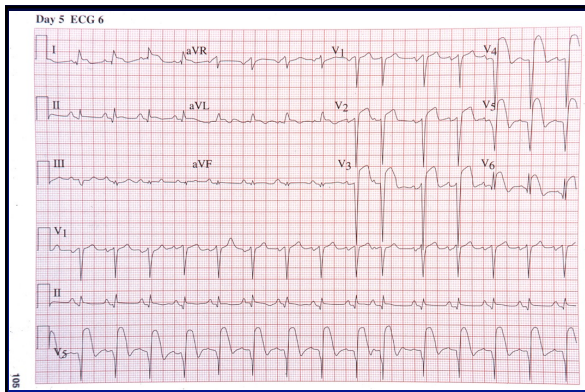
ST ELEVATION CAN BE ALSO INDUCED BY:

1. PRINZMETAL ANGINA (transient ST elevation)
2. PERICARDITIS (in all leads except aVR, saddle shaped)
3. LEVIT 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' duiring the beginning of ST non-pathologic)

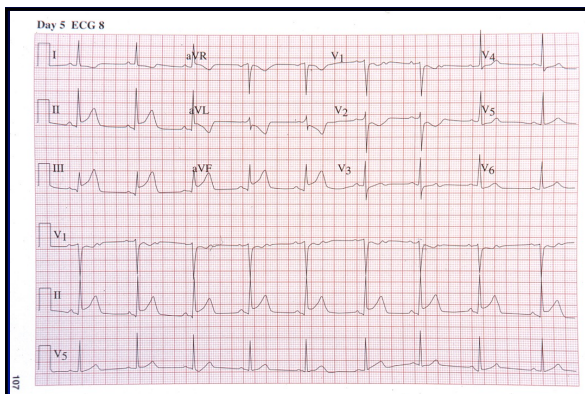
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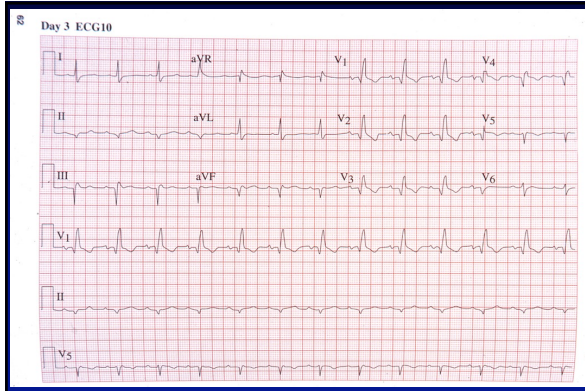
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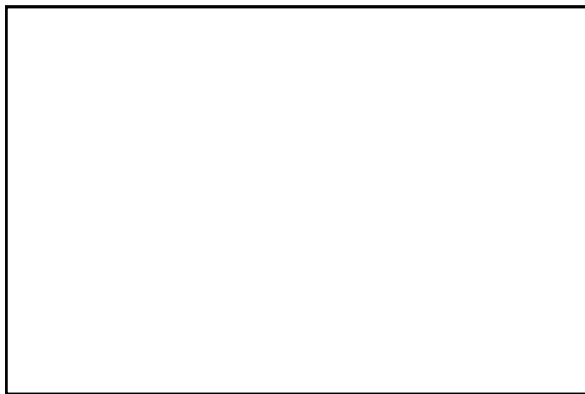
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