

# The importance of good analysis of the ECG to take decisions in a catheterization laboratory

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# Contents

- General aspects
  - The interventionist & the electrocardiologist
  - Anatomic and physiologic information in the ECG
- Risk stratification in acute coronary syndrome
  - ECG-based “risk score”
  - Potential time points in the decision-making process for utilization of the ECG
- Anatomic information in the ECG
  - Basis and case reports (note: ECGs in 50 mm/sec)
- “Preinfarction syndrome” vs. evolving MI
  - A new tool for decision making in ST-elevation MI (STEMI)!

# Background

- The ECG in acute coronary syndrome (ACS) contains critically important *anatomic and physiologic* information
- Lack of collaboration between interventionists and ECG “experts”
- STEMI guidelines:
  - Invasive therapy preferred almost exclusively (“trauma centre”)
  - Decisions about reperfusion strategy within minutes
- Doubt about cost-effectiveness of treating all STEMI patients with primary PCI 24h/7d world-wide
- Alternative: **Individual** risk stratification!

# STEMI treatment strategy – Alternatives to “trauma centers”?

*(Nikus K et al. J Electrocardiol 2005;38:4)*

The interventionist working within the framework of **telemedicine** for instant access to ECG from the patient with comparison to previous ECGs if needed enables individual risk stratification based on:

- Patient history
- Clinical picture
  - Haemodynamic situation
  - Signs of heart failure (cardiogenic shock)
  - Presence of life-threatening arrhythmias
- Anatomical and physiological ECG interpretation

# Potential use for the ECG in decision making of ACS treatment logistics

- When to transport a patient from a long distance acutely for primary PCI
- Need for a doctor in the ambulance
- When to collect an invasive team during night hours
- Case priority in cath labs with heavy workloads
- To define the culprit lesion with the angiography available in multi-vessel disease when the culprit artery is open
- Re-ischemia early post-PCI – re-occlusion or other etiology?

# STEMI – general aspects

- Immediate invasive therapy, usually PCI, is superior to traditional (in-hospital) fibrinolytic therapy (FT), and at least equal to pre-hospital (on-scene) FT
- Every 30 minutes delay to PCI results in an 8% increase in 1-year mortality (*De Luca G et al. Circulation 2004;109:1223*)
- Pre-hospital compared to in-hospital FT saves lives (*Morrison LJ et al. JAMA 2000;283:2686*)
- Rescue PCI (=mechanical therapy when FT does not open the artery) is better than conservative therapy ( $\pm$ another dose of a fibrinolytic) (*Gershlick et al. NEJM 2005;353:275*)

# Benefits from prehospital ECG transmission in ST-elevation ACS

- Increase in usage of reperfusion therapy (=mechanical or pharmacologic) (*Wall T et al. North Carolina Med J 2000;61:104*)
- Reduction in time to reperfusion therapy (*Wall T et al. North Carolina Med J 2000;61:104*)
- Reduction in door-to-needle time (*Morrison LJ et al. Acad Emerg Med 2006;13:84, Brainard AH et al. Am J Emerg Med 2005;23:351; Terkelsen CJ et al. Eur Heart J 2005;26:770*)
- Reduction in door-to-balloon time (*Campbell PT et al. J Electrocardiol. 2005;38:300; Adams GL et al. JACC 2006;47:383A; Canto JG et al. JACC 1997;29:498*)

# STEMI - Telemedicine

- Fibrinolytic therapy may result in intracranial bleeding in 1-2% of patients → false positive diagnosis is dangerous
- ECG interpretation is not easy, some cases are not diagnosed adequately
- Risk of missed diagnoses especially in the real-world scenario of **inexperienced physicians on call**
- ECG transmission to a cardiologist improves diagnostic accuracy without loss of information and speeds up the treatment logistics (*Leibrandt PN et al. Am Heart J. 2000;140:747; Terkelsen CJ et al. Eur Heart J 2005;26:770*)
- Ambulances with ECG transmission stay only 5 minutes longer on scene



# The preinfarction ischemic syndrome

(Sclarovsky S. *Electrocardiography of acute myocardial ischemic syndromes*, 1999)

- The initial ECG manifestation of acute ischemia before the development of an MI
- **Window of opportunity** before irreversible myocardial damage develops
- Prediction of the culprit artery, the level of obstruction in respect to side branches and the dimensions of the culprit artery
- Information about the underlying myocardial milieu
  - Myocardial protection

# “Morphologic ECG risk score” for clinical decision making

- Culprit artery
  - Size of artery
  - Site of occlusion
- Estimated area at risk
- Grade of ischemia → Speed of necrosis
  - “Preinfarction syndrome” or evolving MI → Salvageable myocardium?
-

# ECG markers indicating high priority for immediate invasive evaluation

- Malignant arrhythmia
- New ST-elevation “on top of” old Q-wave MI in other myocardial region
- Grade III ischemia
- Proximal LAD occlusion
- Inferior + lateral injury vector (dominant artery)
- Inferior + right ventricular injury vector with AV-block or hypotension
- Circumferential subendocardial ischemia
- Anterior evolving MI without signs of reperfusion

# STEMI or not?

- Decision-making in patients with ST-elevation and a suspicion of acute coronary syndrome is not always straightforward
- Especially pericarditis, high take off, sport heart and persistent ST-elevation patterns may pose challenges for the physician on duty

# STEMI or not?

- Pericarditis:
  - The PR segment is usually depressed in all lead except aVR and occasionally  $V_1$
  - The ST segment may be elevated in all leads except in aVR and  $V_1$
  - The distribution of ST-elevation does not usually follow anatomically distributed myocardial segments
- Early repolarization (high take-off)
  - Typically concave ST-elevation in  $V_1$ - $V_3$
  - Often associated with high T waves
  - Notched J point if present in lateral precordial leads

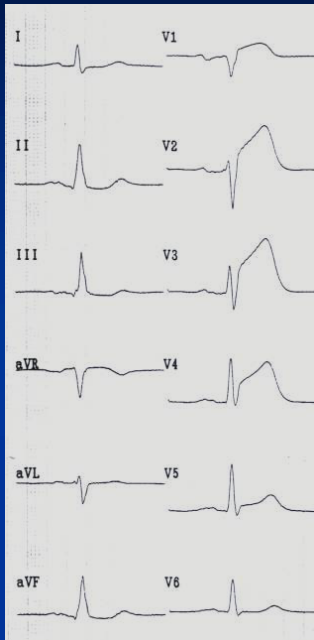
# Grade of ischemia

*(Sclarovsky S et al. Isr J Med Sci 1990;26:525)*

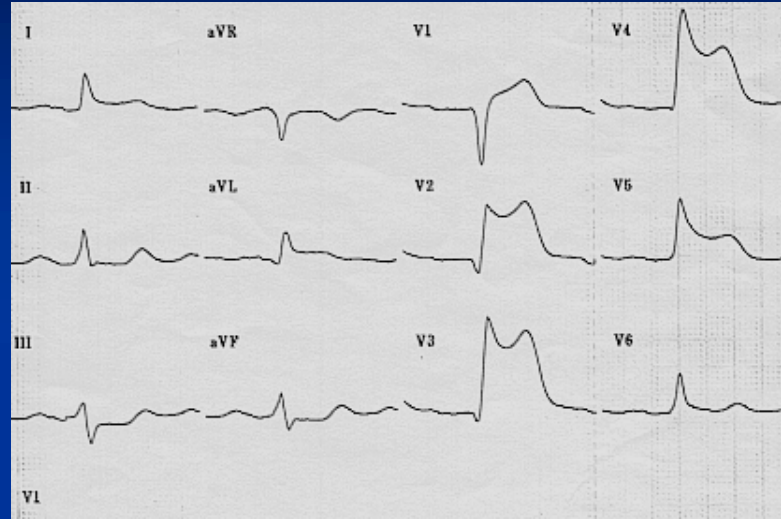
*(Birnbaum Y, Sclarovsky S. J Electrocardiol 2001;34,S17)*

- Grade I ischemia: Tall, symmetrical, and peaked T waves
- Grade II ischemia: ST elevation without distortion of the terminal portion of the QRS
- Grade III ischemia: Changes in the terminal portion of the QRS complex:
  - Emergence of the J point  $\geq 50\%$  of the R wave in leads with qR configuration, or disappearance of the S wave in leads with an Rs configuration

## GRADE II



## GRADE III



### Grade III compared to grade II:

- More severe ischemia and worse short-and long-term prognosis
- Less myocardial protection and faster progression of necrosis
- Less benefit from fibrinolytic therapy or primary angioplasty
- Impact on choice of reperfusion strategy needs to be defined

# Prerequisites and basis for anatomical interpretation of the ECG in STEMI

(Sclarovsky S. *Electrocardiography of acute myocardial ischemic syndromes*, 1999)

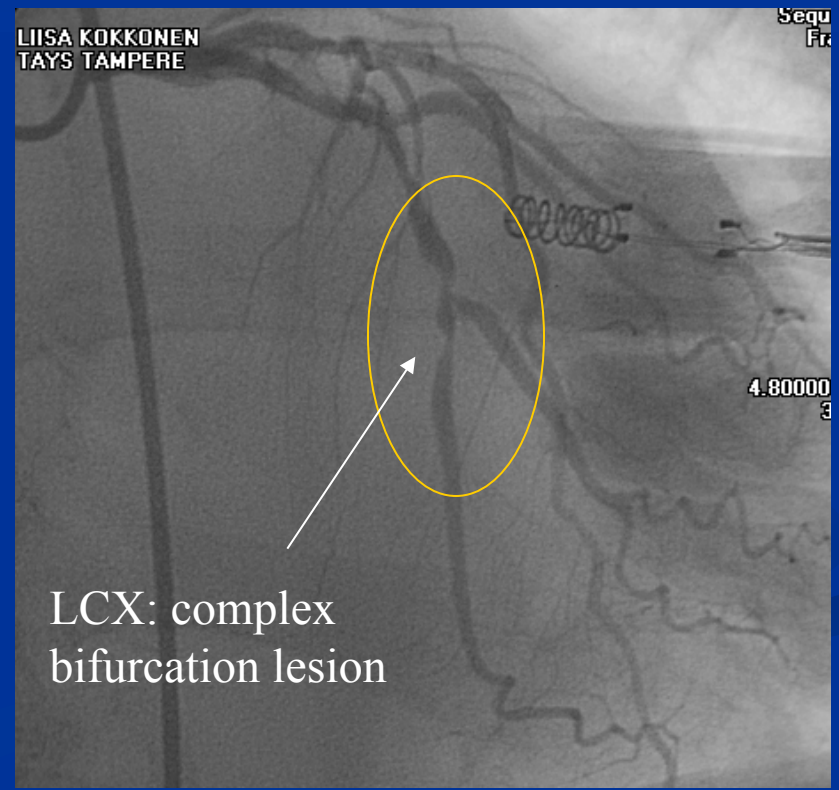
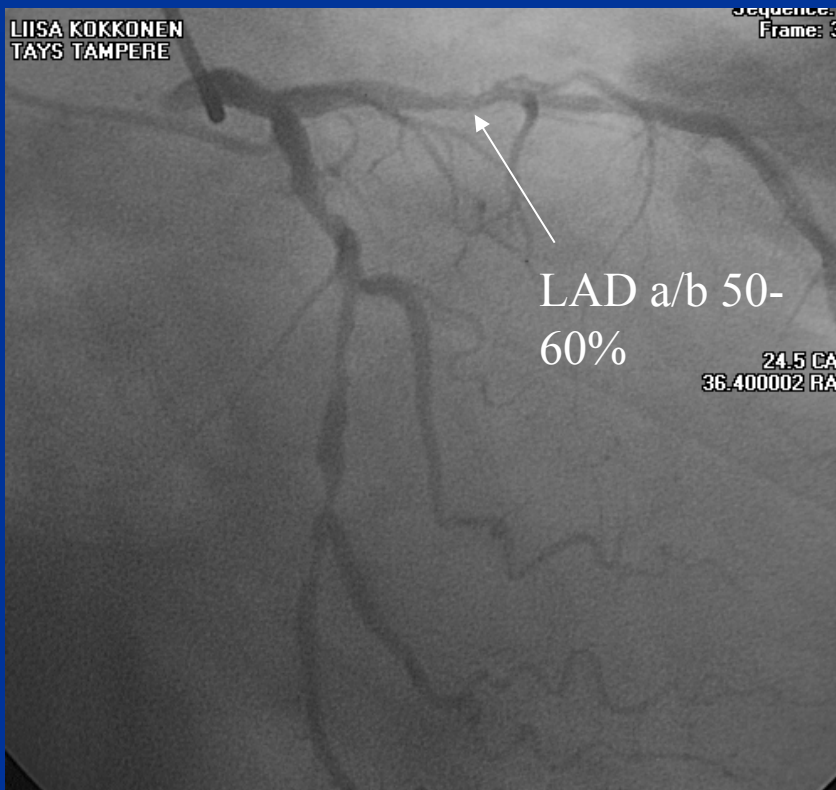
- ECG from the acute phase - the “preinfarction syndrome” - available
- *Absence of major confounding factors*
  - LVH, LBBB, pacemaker, WPW, old Q waves
- Core of ischemia to predict the **culprit artery**
- Localization of ST-elevation and reciprocal ST-depression + signs of attenuation to predict the **level of occlusion and dimension of artery**



# A case to illustrate use of the ECG for decision support after determination of coronary anatomy with coronary angiography

- 67 year old woman
- Hypertension, heart failure, stroke, atrial fibrillation, obesity
- Previous stable CCS 2 angina
- Acute chest pain → Local hospital
- VT → VF, DC x 2
- Urgent coronary angio

- Two-vessel disease
- Left circumflex: tight bifurcation lesion
- Left anterior descending: marginally significant 50-60%

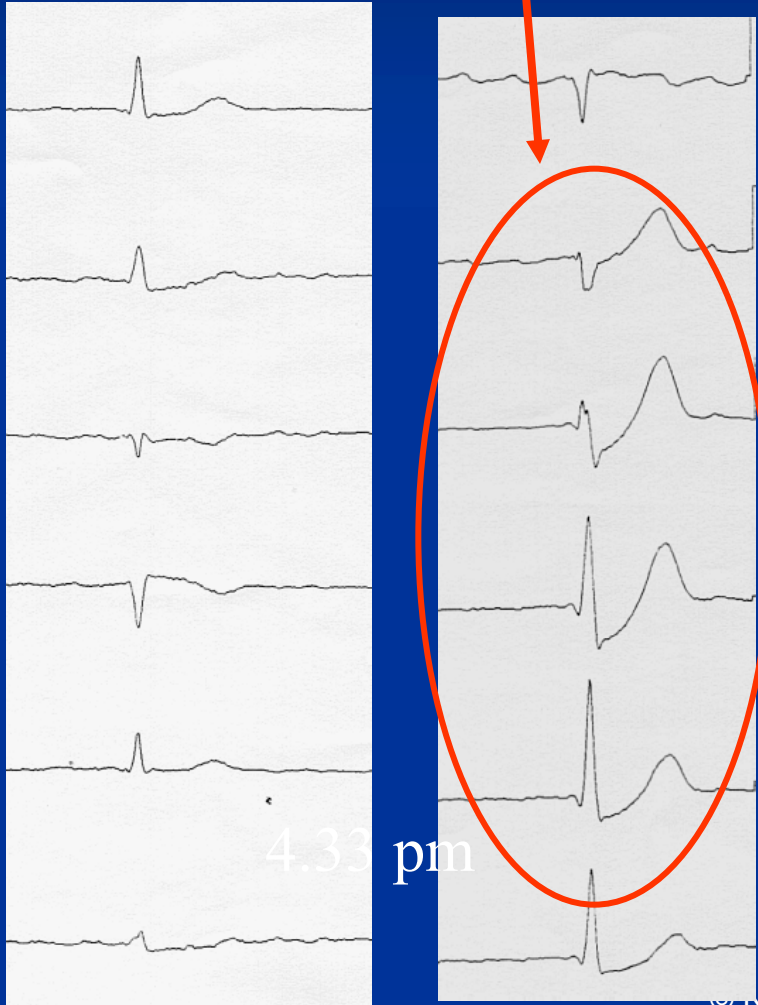


# After the angio- what next?

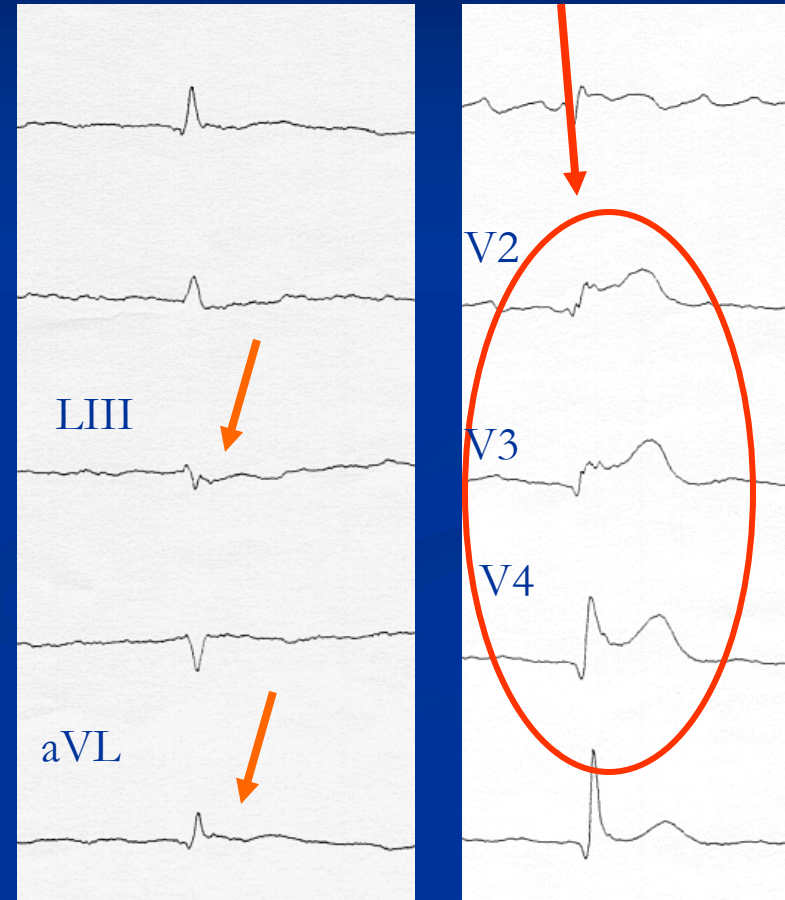
LCX-PCI? PCI of a non-culprit artery may not be without risks - acute closure or no-reflow may be disastrous in non-infarcted regions which should compensate (with hyperkinesis) for the loss of myocardium in the infarcted area

**What anatomic and physiologic information did the ECG from the acute phase contain?**

**ECG n:o 1: Regional subendocardial ischemia/  
Subtotal LAD- or total side-branch occlusion (LD, IM)**



**ECG n:o 2: Grade III ischemia (QRS-distortion)  
Unprotected myocardium**



PROXIMAL LAD OCCLUSION: CORE OF ISCHEMIA: V<sub>2</sub>-V<sub>4</sub>, ST-ELEVATION IN aVL WITH RECIPROCAL ST-DEPRESSION IN LIII

## Therapeutic decision:

- Treat the culprit lesion (LAD) now
- Treat the bifurcation lesion (LCX) later if indicated by persistent symptoms
  - Safer when the patient is hemodynamically stable

# ”Complete” vs. incomplete revascularization - PCI of lesions well controlled by medication?

- Especially in elderly patients, a strategy of incomplete revascularization and symptom-guided strategy may be preferable
- PCI of all significant stenoses → risk for complications and restenosis
- CABG: acute/subacute complications, graft occlusions

**CASES TO ILLUSTRATE RISK  
STRATIFICATION BASED ON  
ECG PATTERNS IN ST-  
ELEVATION ACUTE  
CORONARY SYNDROME**

## STE-ACS ECG patterns with correlation to coronary angiography findings

| ECG finding   | Core of ischemia   | Culprit artery/lesion  |
|---|--------------------|--|
| <p>Anterior STEMI</p> <ul style="list-style-type: none"> <li>•STE: I, aVL</li> <li>•STD: II, III, AVF</li> <li>•STE II, III, aVF</li> <li>•Right bundle branch block</li> </ul> | Leads V2-V4        | <ul style="list-style-type: none"> <li>➤Proximal LAD</li> <li>➤Proximal LAD</li> <li>➤Distal, "wrap-around-the-apex" LAD</li> <li>➤Proximal LAD</li> </ul> |
| <p>Inferior STEMI</p> <ul style="list-style-type: none"> <li>•STE: V5, V6</li> <li>•STD: V4, V5</li> <li>•STE: V1/V4R</li> <li>•STE: I (aVL)</li> </ul>                         | Leads II, III, aVF | <ul style="list-style-type: none"> <li>➤Dominant artery</li> <li>➤Concomitant 3-VD or LAD</li> <li>➤Proximal RCA</li> <li>➤Proximal LCX</li> </ul>         |
| <p>Lateral STEMI</p> <ul style="list-style-type: none"> <li>•STE: I, aVL and V2</li> <li>•STE: I, aVL, STD: V2</li> <li>•STE V5, V6</li> </ul>                                  | I, aVL, V5, V6     | <ul style="list-style-type: none"> <li>➤First diagonal branch</li> <li>➤First marginal branch</li> <li>➤Marginal branch</li> </ul>                         |

Abbreviations: STE=ST-elevation; STD=ST-depression; LAD= left anterior descending; LCX=left circumflex coronary artery; RCA=right coronary artery; VD= vessel disease



# Inferior STEMI – critical ECG data for decision making illustrated by cases

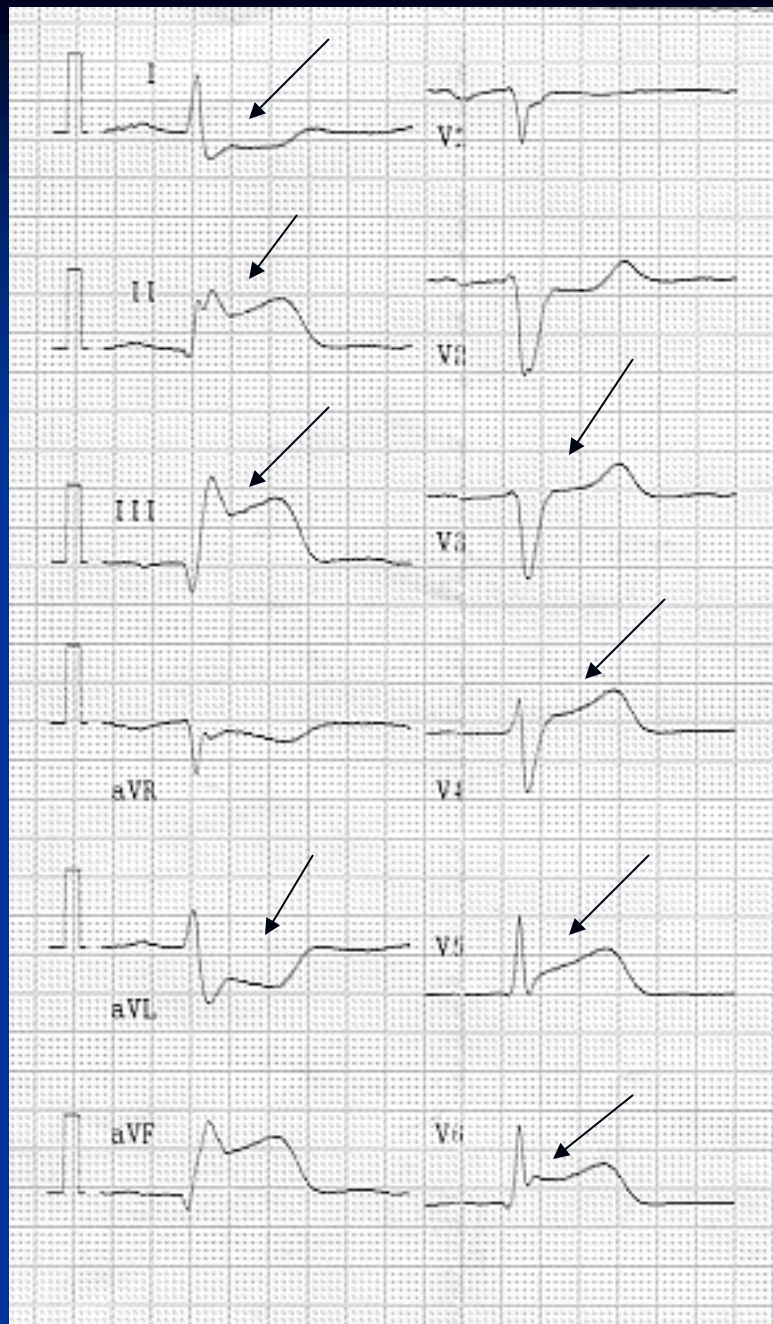
- ST↑ also in  $V_5$ - $V_6$ ?
  - Dominant artery
- Proximal culprit lesion?
  - ST↑ in  $V_4R$  or  $V_1$ ? (proximal RCA)
  - ST↑ in LI? (Proximal LCX)
- Proximal occlusion of small right coronary artery
  - ST↑ in  $V_1$  and III (not II and aVF), ST↓ in  $V_6$
- ST↓ in  $V_4$ - $V_5$ 
  - Signs of two- or three-vessel disease
- Grade of ischemia?

# CASE 1:

## Morphologic ECG analysis:

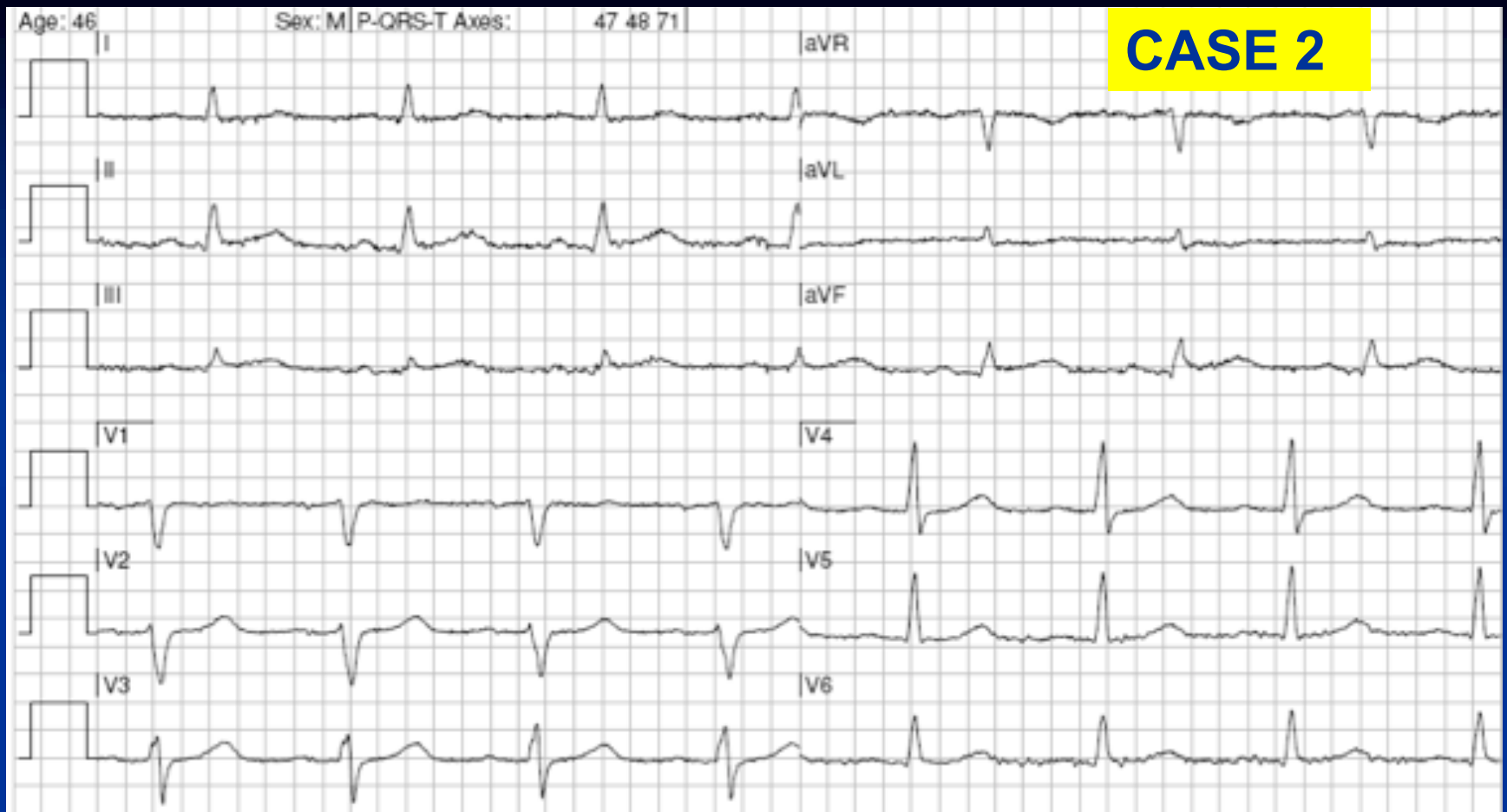
1. Core of ischemia: II, III, aVF → Culprit RCA or LCX
2.  $ST\uparrow$  III > II &  $ST\downarrow$  >1mm I, aVL ⇒ RCA (*Chia B-L et al. Am J Card 2000;86:341*)
3.  $ST\uparrow \geq 2$  mm V<sub>5</sub>-V<sub>6</sub> ⇒ "Mega-artery" (*Assali A et al. Am J Card 1998;81:81*)
4. J-point >50% of R-wave amplitude III, aVF → Grade III ischemia

Invasive priority: HIGH



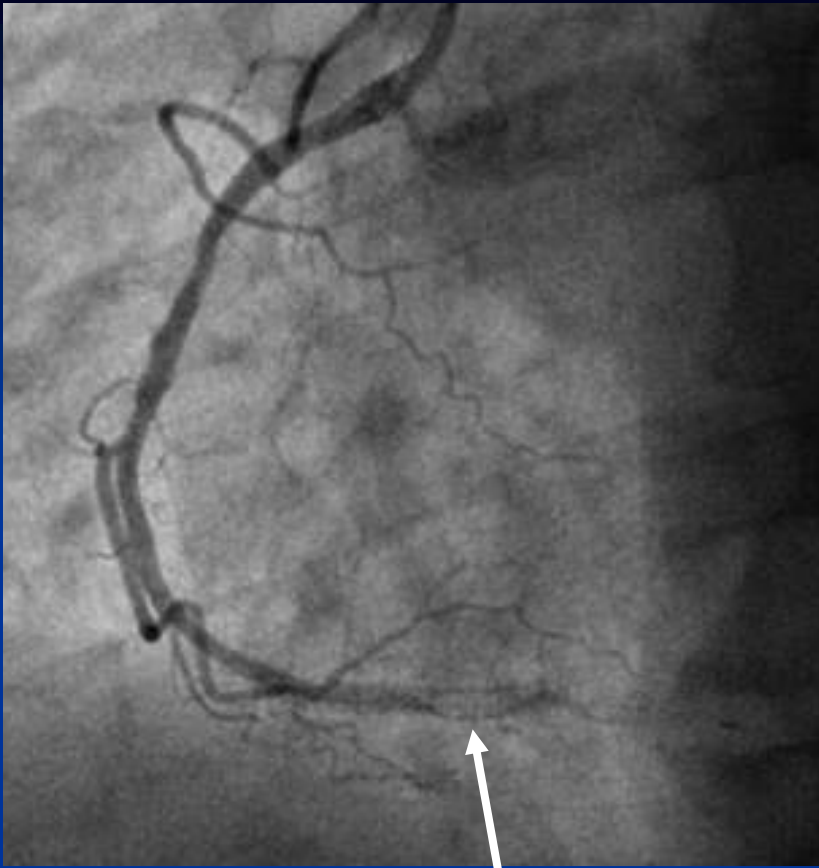
# CASE1: Right coronary artery after stenting



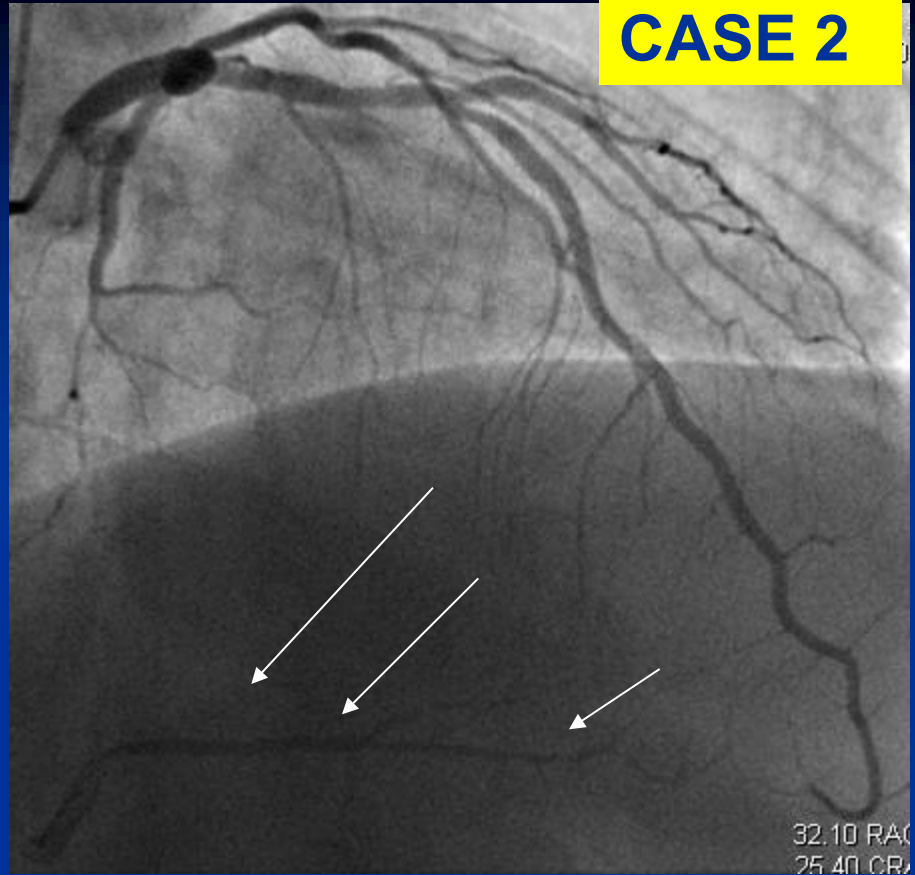


- Minor ST-elevation in II, III, aVF; reciprocal ST-depression in aVL
  - No ST-deviations in the precordial leads
  - Grade II ischemia
- **Distal occlusion and well protected myocardium**

**CASE 2**

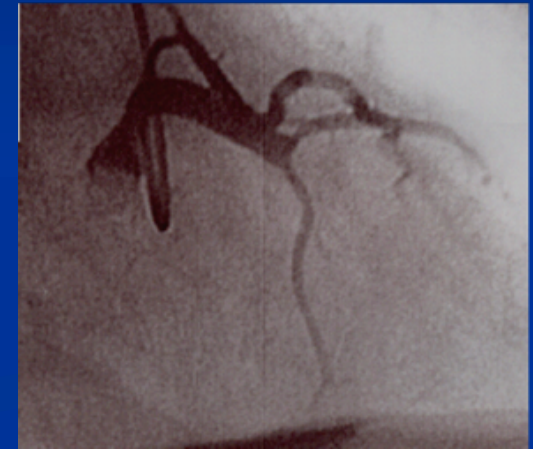
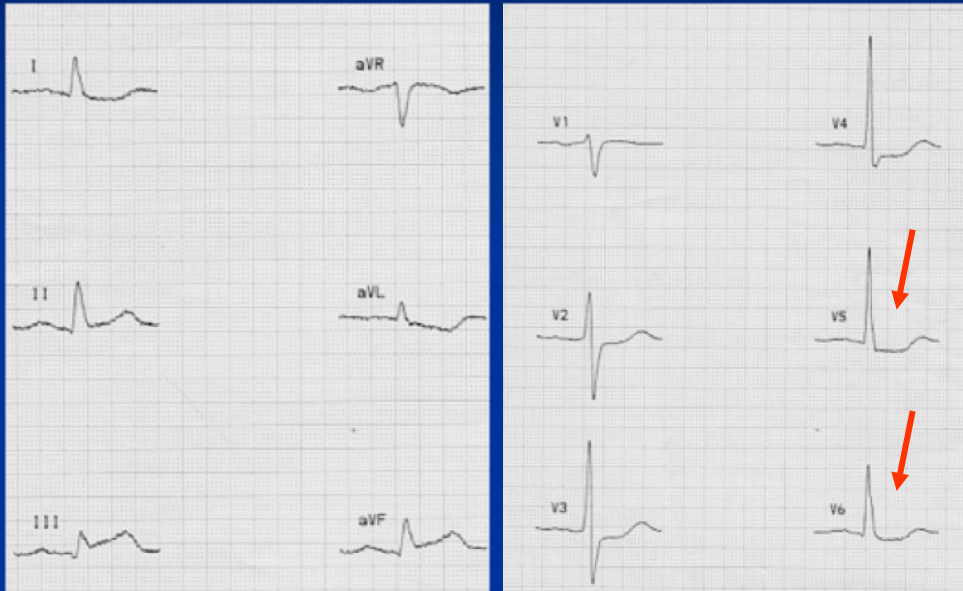


Subacute stent thrombosis is the distal part of the right coronary artery



Good collateral flow from the septal branches of the LAD to the PD branch of the RCA

# ECG SIGNS OF 2- OR 3-VESSEL DISEASE IN INFERIOR STEMI



Inferior STEMI with ST↓ maximally in the lateral precordial leads is a sign of 3-vessel disease or concomitant LAD

disease (*Hasdai D et al. Int J Cardiol*

*1997;58:273; Eskola M et al, J Electrocardiol*

*2004;37:257)*

# Anterior STE-ACS – ECG markers for clinical decision making

- Area at risk larger if proximal culprit lesion:
  - ST $\uparrow$  I, aVL and/or aVR
  - Reciprocal ST $\downarrow$  in II, III, aVF
- Evolving MI without ECG signs of reperfusion (high invasive priority)
- Grade III of ischemia

# Preinfarction syndrome vs. evolving MI

A new tool for decision making in  
ST-elevation MI

(Eskola MJ et al. Eur Heart J; doi:10.1093/eurheartj/ehm428.

Published online ahead of print October 11, 2007)

*(Sclarovsky S. Electrocardiography of acute myocardial ischemic syndromes, 1999)*



# The infarct process

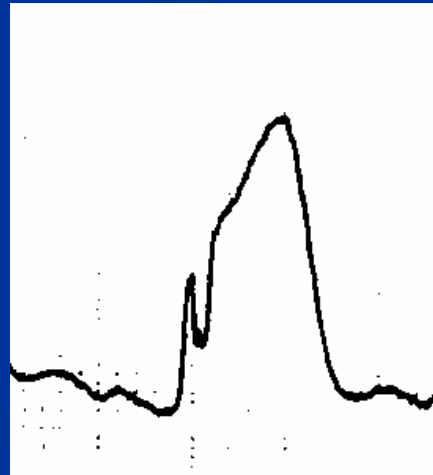
- Sudden occlusion of a coronary artery results within seconds in positive and tall T waves, followed by elevation of the ST-segment
  - In patients with acute chest pain this represents the **pre-infarction syndrome**
- From the pre-infarction stage, the ischemic process usually **evolves towards MI** with or without Q waves

# The infarct process

- Successful reperfusion results in normalization of the ST-segment, usually accompanied by T-wave inversion
- T-wave inversion is a marker of an open infarct-related artery with restored myocardial blood flow

# Definitions

- Pre-infarction syndrome, **PIS**
  - Represents the window of opportunity to treat before irreversible myocardial damage develops
  - **ECG: an elevated ST-segment and a peaked T wave**



# Definitions

- Evolving myocardial infarction, **EMI**
  - **ECG signs of necrosis (pathological Q waves) and/or signs of reperfusion (negative or biphasic T waves)**
  - **There are three types of evolving MI**
    - **EMI without ECG signs of reperfusion**
    - **EMI with incomplete reperfusion**
    - **EMI with complete reperfusion**

# Definitions

- Evolving myocardial infarction, **EMI**
  - **EMI without ECG signs of reperfusion**
    - **A deep Q wave without a notch in the descending limb, an elevated ST-segment, and a positive T wave**



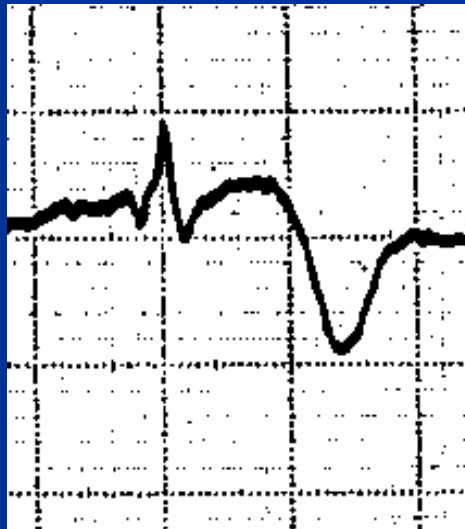
# Definitions

- Evolving myocardial infarction, **EMI**
  - **EMI with incomplete reperfusion**
    - **ST-elevation, a biphasic T wave (negative terminal portion) with or without Q wave**



# Definitions

- Evolving myocardial infarction, **EMI**
  - **EMI with complete reperfusion**
    - **No or minor ST-elevation, inverted T wave with or without Q wave**



# Use of the PIS and EMI ECG patterns in optimizing reperfusion for STEMI

- Results from a post hoc analysis of 1300 patients from the DANAMI-2 trial (Eskola et al. Eur Heart J, 2007)
  - The PIS pattern was more common than the EMI pattern both in patients with anterior (58 vs. 42%, respectively) and inferior STEMI (86 vs. 14%)
  - EMI patients had >1h longer median time delay from symptom onset to randomization
    - This is consistent with the presence of greater myocardial necrosis at presentation



# Use of the PIS and EMI ECG patterns in optimizing reperfusion for STEMI

- The event rate for the composite endpoint of death, reinfarction, and disabling stroke was higher in EMI vs. PIS patients at a median follow-up of 2.7 years
  - 11.4 vs. 6.9 per 100 person-years, respectively, RR=1.6,  $p<0.001$ 
    - The difference was explained with higher mortality in the EMI group than in the PIS group

# Use of the PIS and EMI ECG patterns in optimizing reperfusion for STEMI

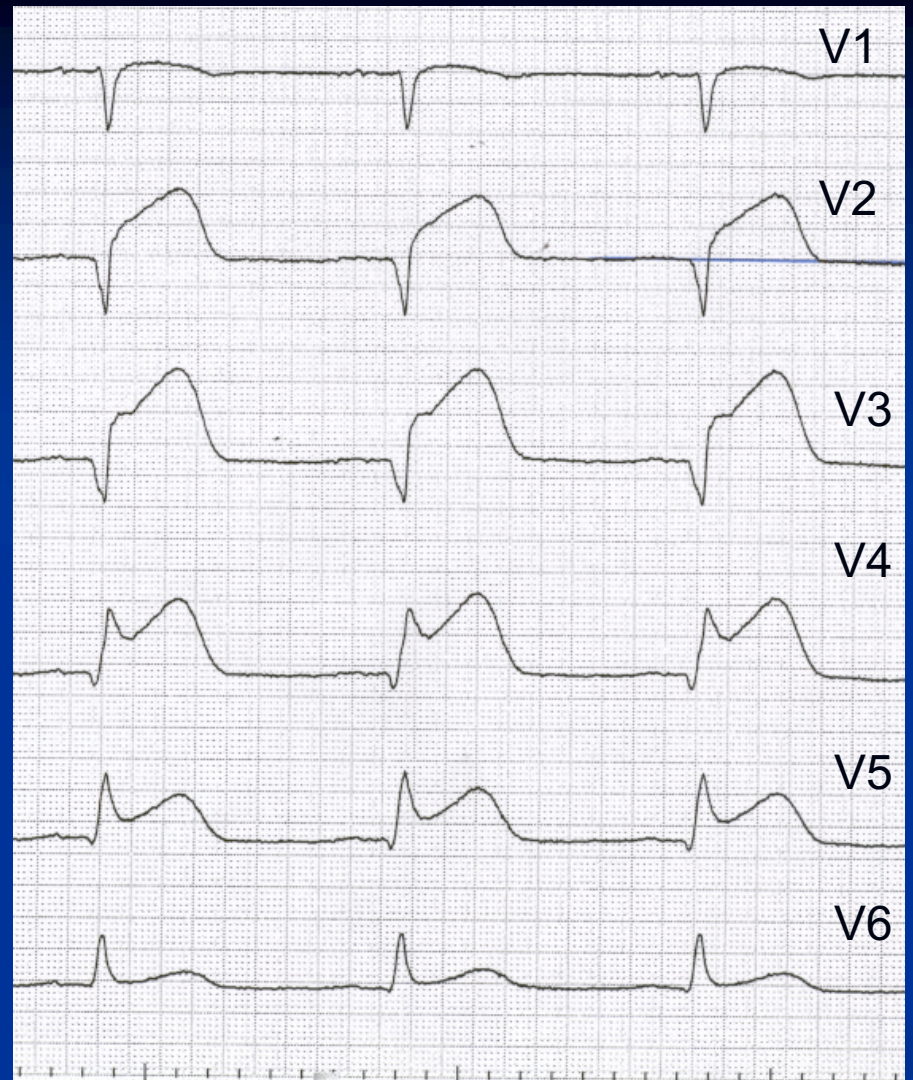
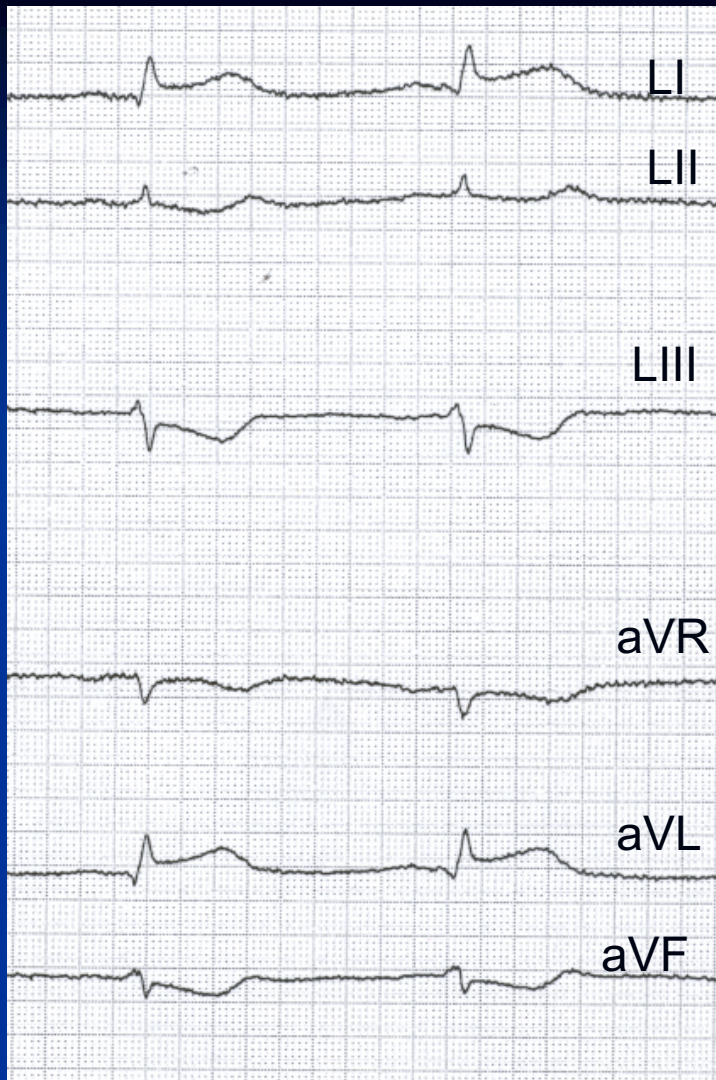
- In the PIS group, the event rate of composite endpoint was lower in patients treated with primary PCI compared with fibrinolytic therapy (FT) at 2.7 years FU
  - 5.5 vs. 8.5 per 100 person years, respectively, RR=0.6,  $p=0.004$ 
    - The difference was explained with lower reinfarction rate in the primary PCI group than in the FT group

# Use of the PIS and EMI ECG patterns in optimizing reperfusion for STEMI

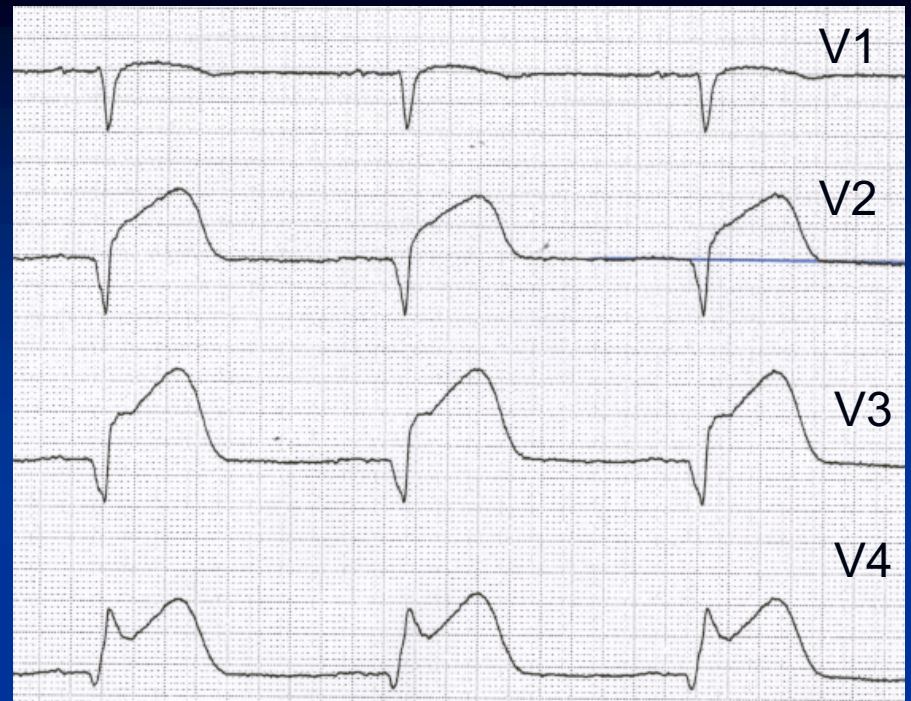
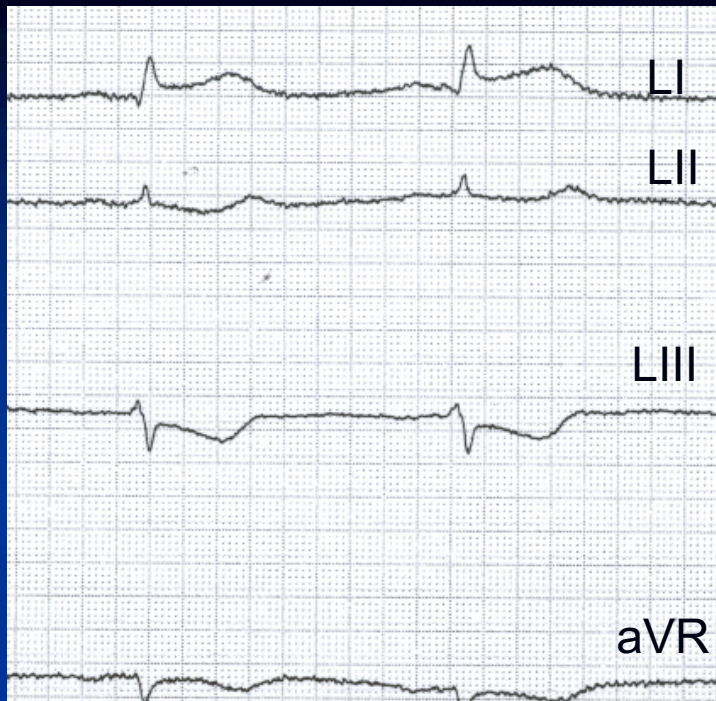
- In the EMI patients, there were no significant differences between reperfusion therapies
  - However, in the anterior EMI group, the patients with no signs of reperfusion on the ECG, treated with primary PCI, had a better prognosis than patients treated with FT
    - The superiority of primary PCI over FT was driven by a 51% reduction in the relative risk of composite endpoint
- In the EMI group, there was no difference in composite endpoint according to presence or absence of Q waves

# Case

- A 60-y old male with acute chest pain since 7 hours
- ECG recorded “on scene” by paramedics
- The ECG was transmitted to the cardiologist on call by telemedicine



**Primary PCI, fibrinolysis or no reperfusion therapy?**



The ECG reveals:

**1) Anterior ST-elevation MI**

**2) Evolving MI with no signs of reperfusion**

- Q waves in right precordial leads
  - Q wave is not a marker of a "lost case" for reperfusion therapy
- ST-elevation in leads I, Avl, V<sub>1-5</sub> with positive T waves

# Treatment based on the ECG information

- Fibrinolysis or primary PCI?
  - Staging of the infarct process based on patient-reported “ischemic time” may be inferior to ECG criteria for PIS and EMI
  - Increased bleeding risk with FT
  - EMI with no signs of reperfusion on ECG
    - The number needed to treat with primary PCI in order to avoid one death, reinfarction, or disabling stroke in a 2.7 years period compared with FT is 5 (Eskola et al, Eur Heart J, 2007)

**Every effort should be made to transfer directly to primary PCI**

# Anatomic information in the ECG post-PCI

Case: ECG interpretation after PCI of the right  
coronary artery



# Blood supply of the right ventricle

- The right coronary artery (RCA) supplies most of the right ventricular (RV) free wall
- The lateral RV free wall is subtended by the RV branches
- The inferoposterior RV free wall is subtended by the right acute marginal branches and partly by the posterior descending branch
- Blood supply to the anterior free wall is provided by the conus branch of the RCA and the RV branches of the left anterior descending coronary artery

# Significance of lead V<sub>4</sub>R

- Recording of right-sided chest leads, usually lead V<sub>4</sub>R, during chest pain
  - Distinguishes patients with occlusion of the RCA proximal to RV branches from those with a distal RCA occlusion
    - 90% sensitivity, 91% specificity (*Chou T-C et al. Am J Med 1981;70:1175; Croft CH et al. Am J Cardiol 1982;50:421*)
  - Is associated with enhanced use of fibrinolytic therapy in acute myocardial infarction (*Harju JA. J Electrocardiol. 2006;39:368.e1*)

# Recording of right ventricular electrical potentials

- ST-elevation may be present both in lead  $V_4R$  and in the right precordial leads
- Recordable potentials in the right precordial leads depend on
  - The degree of clockwise rotation of the heart in the horizontal plane
  - Body geometry
  - The attenuation phenomenon

# Recording of right ventricular electrical potentials

- ECG manifestations of RV injury in the right precordial leads are usually not present as:
  - In cases with proximal occlusion of the RCA, the dominant forces of the accompanying LV inferior wall injury attenuates the anterior wall ST-elevation
    - This opponent injury current is absent in isolated occlusion of the RV and/or right acute marginal branches

# The ECG pattern of isolated RV infarction

- This ECG pattern contains

**ST-elevation in leads  $V_4R$  and  $V_{1-3}$**

**without ST elevation  
in inferior leads II, III and aVF**

# The ECG pattern of isolated RV infarction

- May be misinterpreted as an acute anterior STEMI caused by an occlusion of the left anterior descending coronary artery (LAD)
  - In LAD occlusion the maximal ST segment elevation (the “core of ischemia”) is almost exclusively in leads  $V_{2-4}$  and ST elevation is higher in lead  $V_3$  than in lead  $V_1$

# The ECG pattern of isolated RV infarction

- It is important to recognize the ECG pattern of isolated RV infarction because
  - During stenting of the right coronary artery, the orifice of the RV- and/or right acute marginal branch is frequently covered and there is a possibility for side-branch occlusion
  - Decision-making in the catheterization laboratory and in the CCU also post-PCI should be based on anatomic information in the ECG

# Case

- A 68-year-old female was referred to the hospital with acute coronary syndrome
  - Troponin I was minimally elevated and thus non-ST-elevation myocardial infarction was diagnosed
  - Aspirin, low molecular weight heparin, glycoprotein IIb/IIIa inhibitor, betablocker therapy and nitrate-infusion was initiated



# Case

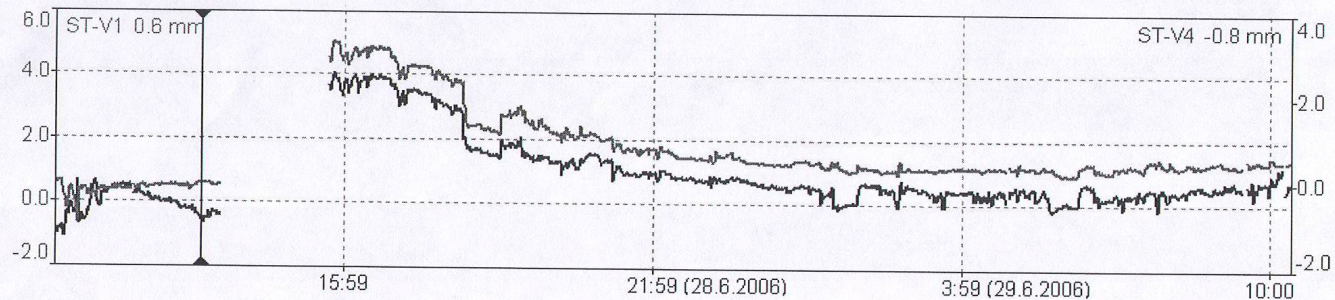
- Coronary angiography performed 9 hours after admission showed
  - 50% stenosis in the mid part of the LAD
  - The culprit lesion was a 95% stenosis starting from the proximal part of the RCA and covering the mid part of the vessel
- Two drug-eluting stents were needed to cover the diseased segment – both the RV- and the acute marginal branch were jailed by the stents

# EASI-ECG recorded in the CCU before the PCI

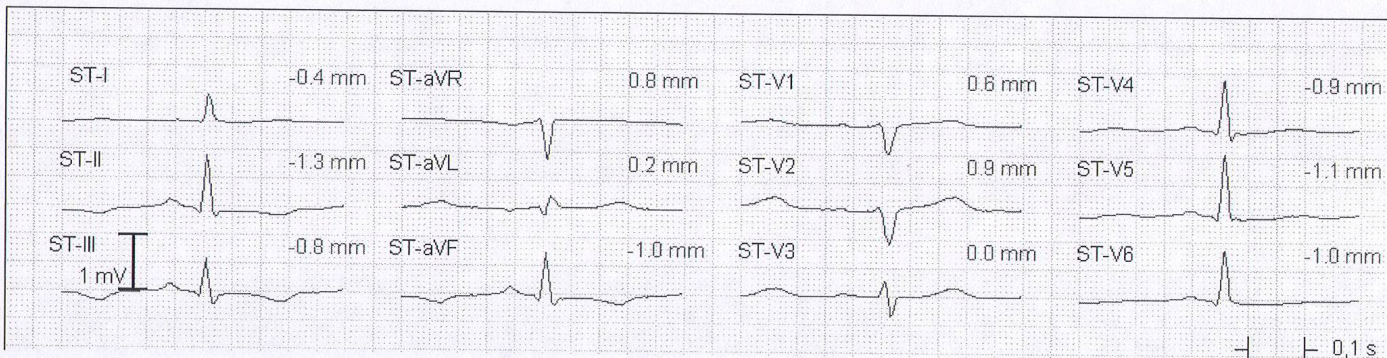
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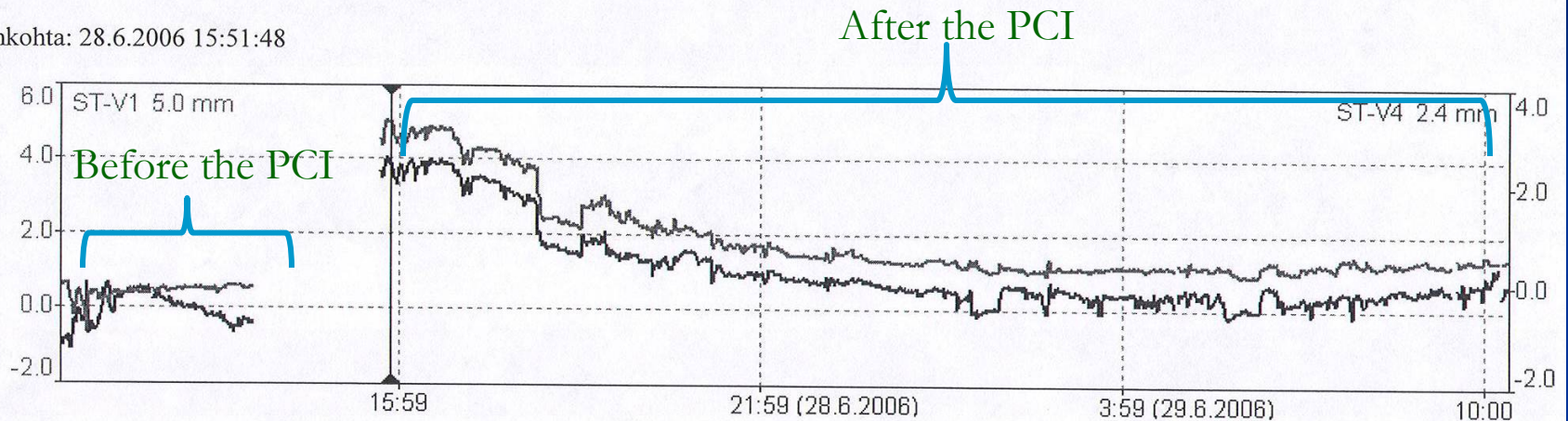


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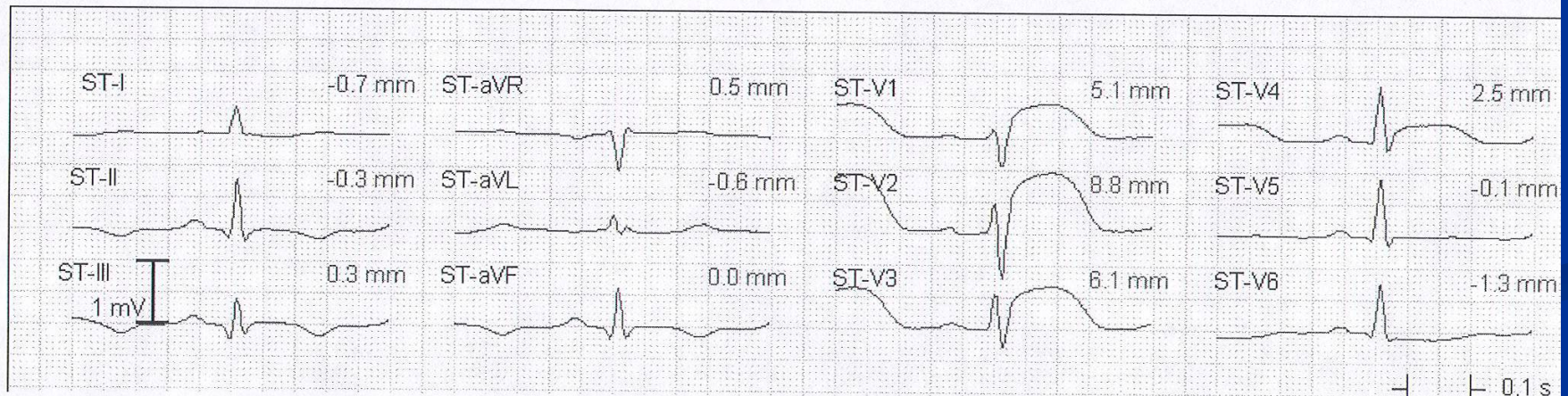
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# EASI-ST-segment trend and EASI-ECG recorded in the CCU immediately after the PCI of the RCA

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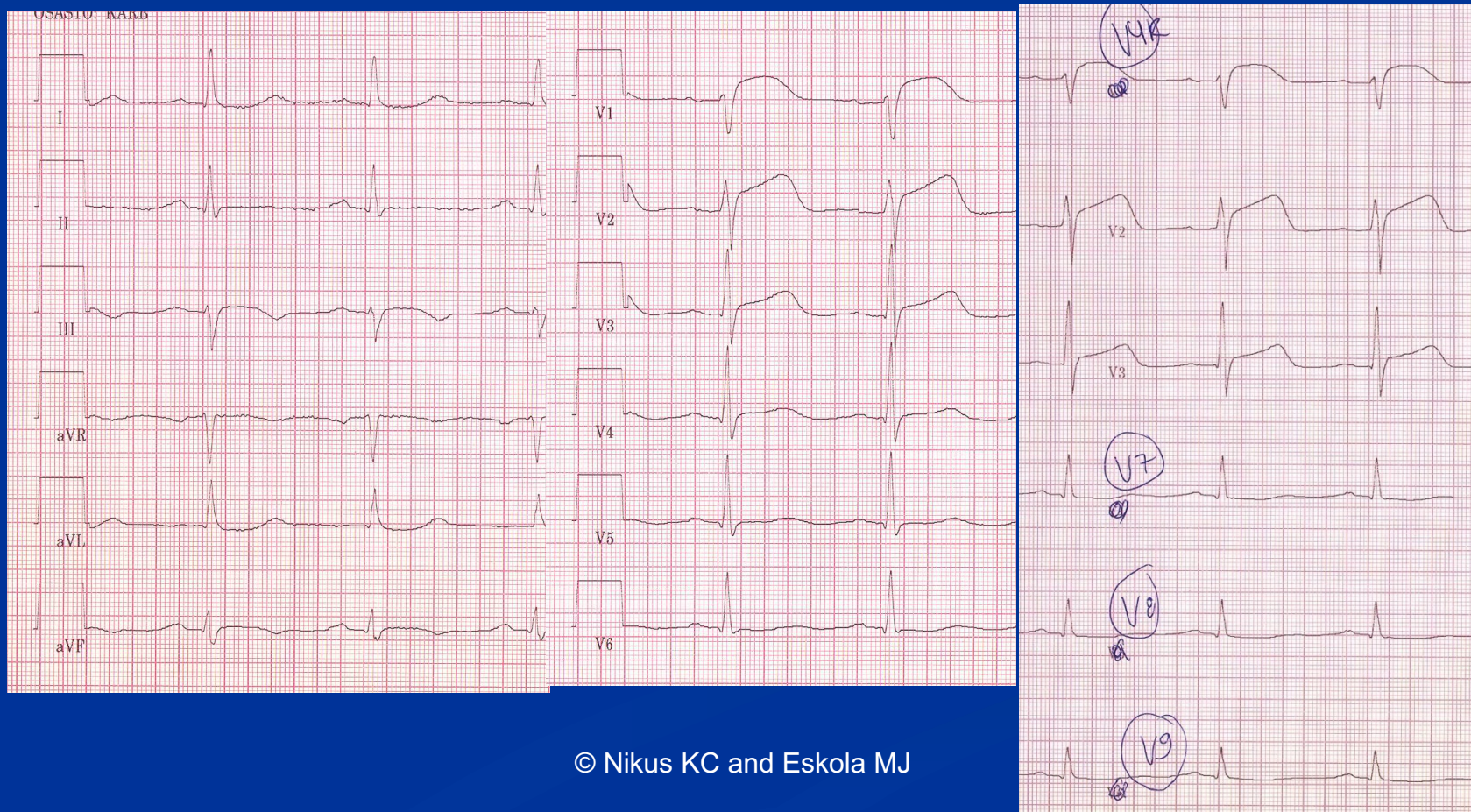
Selaa trendiä: ◀ ▶



15-lead ECG recorded at the time of chest pain in the CCU immediately after the PCI

**Resident consults you, what do you suggest?**

- **Procedure-related thromboembolism to the LAD?**
- **Second active plaque in the LAD?**



# Interpretation of the 15-lead ECG recorded in the CCU

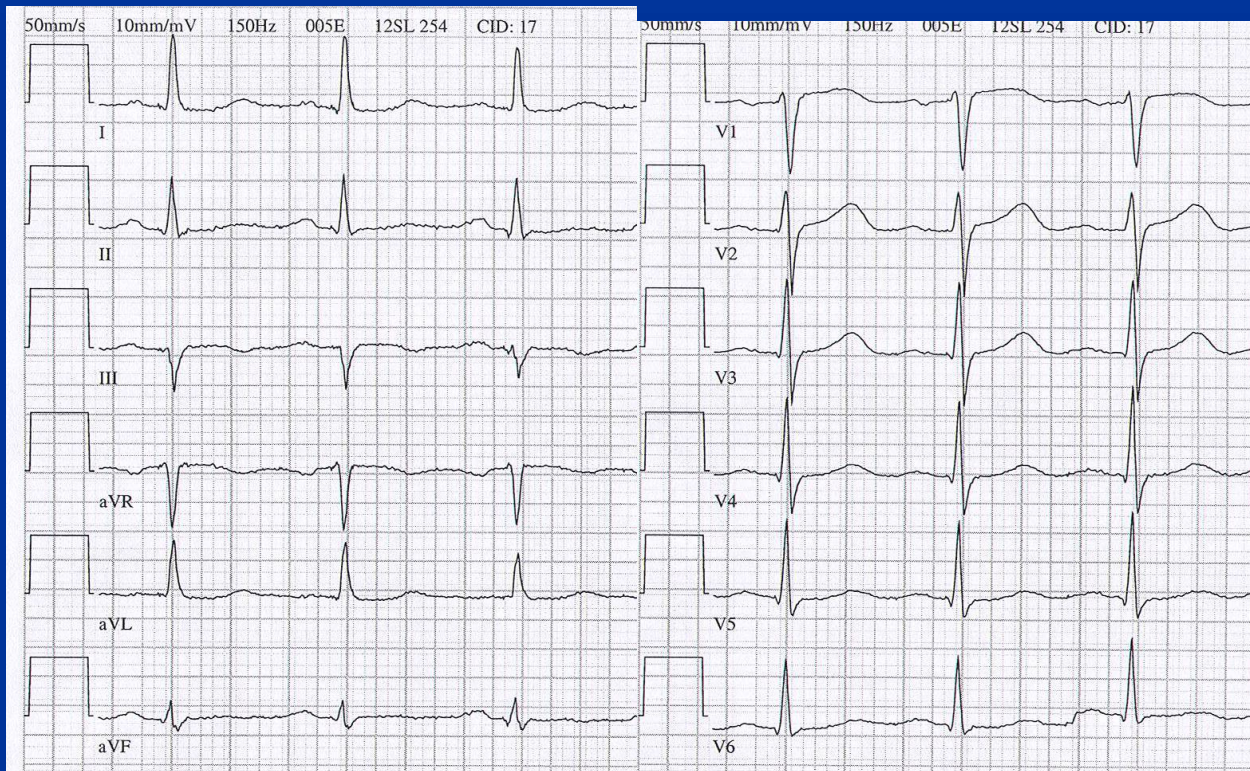
- The ST-segments in the inferior and posterior ( $V_{7-9}$ ) leads are iso-electric
- The ECG reveals the pattern of isolated RV infarction
  - ST-elevation in leads  $V_4R$  and  $V_{1-4}$
  - The sum of the ST segment elevations in leads  $V_{1-2}$  is greater than in leads  $V_{2-3}$

# Therapeutic strategy

- Chest pain post-PCI was related to the RV infarction, not to an acute anterior wall infarction
- The ECG finding is compatible with an RV branch occlusion
- Coronary angiography was not repeated
  - No signs of hemodynamically significant RV-MI were present
- The patient soon become asymptomatic

# 12-lead ECG recorded the next day

ST segments (except in  $V_1$ ) are iso-electric and no Q-waves have developed



# Five different patterns of non-ST elevation acute coronary syndrome representing different pathophysiology and clinical significance

*Nikus K et al. J Electrocardiol 2004;37:247*



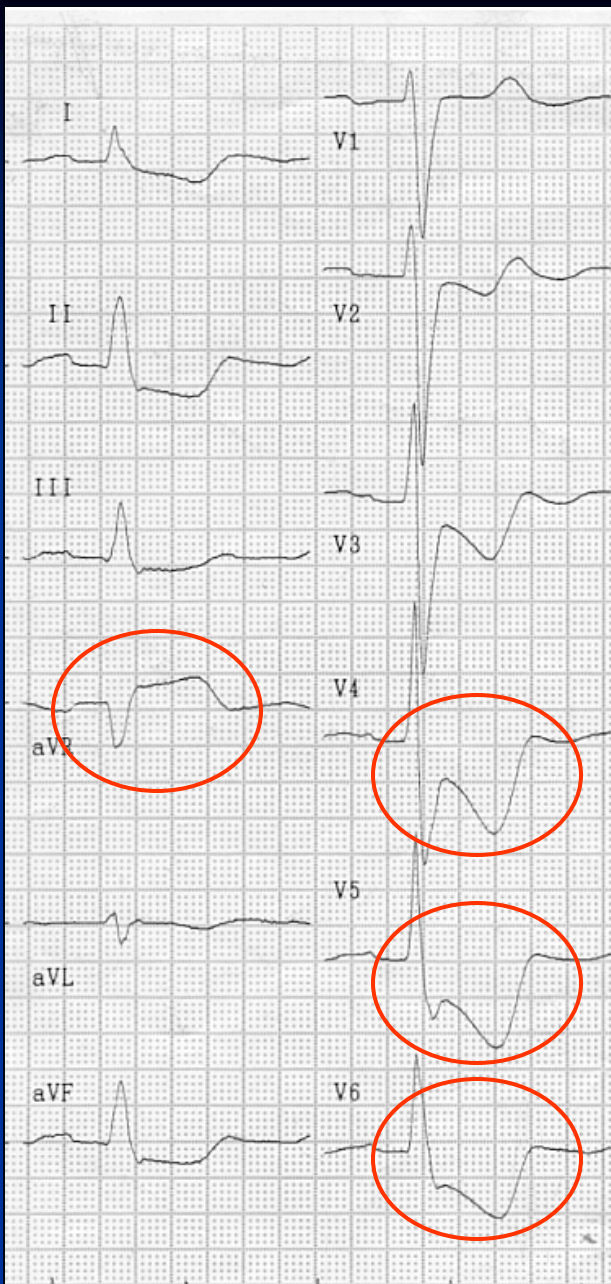
## Acute coronary syndrome presenting with ST-depression during pain.

| ECG pattern   | Note   | Anatomic correlate  |
|---|--|---|
| STD maximally in V4-5 +<br>Inverted T waves +<br>STE in aVR ( $\pm$ V1) | Non-specific finding during<br>tachycardia or if chronic changes             | LMCA or 3-VD  |
| STD + peaked positive T<br>waves maximally in V2-<br>V4                 | Leads V7-V9 recommended for<br>differential diagnosis                        | Subtotal occlusion of the<br>LAD or total occlusion of a<br>diagonal or intermediate side<br>branch |
| Symmetrical inverted T<br>waves in V2-V4                                | Post-reperfusion change<br>Also present in apical ballooning<br>(tako-tsubo) | LAD disease – “Wellens’<br>sign”  |
| STD max in V1-V3 (T $\uparrow$<br>or T $\downarrow$ )                   | Leads V7-V9 recommended for<br>differential diagnosis                        | Non-proximal occlusion of<br>LCX: "mirror image STEMI"  |
| Tall symmetrical T waves  | First sign of abrupt coronary<br>occlusion                                   | Grade I ischemia  |

Abbreviations: STE=ST-elevation; STD=ST-depression; LAD= left anterior descending; LCX=left circumflex artery; LMCA=left main coronary artery

# Pattern 1 – Circumferential subendocardial ischemia

- Transient ST depression recorded during anginal pain with maximal changes in  $V_{4-5}$  with inverted asymmetric T waves and heart rate less than 100 bpm is due to a transient sudden obstruction of left main coronary artery, left main equivalent- or severe three vessel disease
- This pattern is induced by a sudden increase in end-diastolic pressure with a minor increase in end-diastolic volume



**WIDE-SPREAD ST-DEPRESSION, MAXIMALLY IN  
 $V_4-V_5$**

**+ INVERTED T WAVES**

**+ST-ELEVATION IN LEAD aVR =**

**CIRCUMFERENTIAL SUBENDOCARDIAL  
ISCHEMIA**

**IF TRANSIENT AND NOT INDUCED BY  
TACHYCARDIA:**

**HIGH PROBABILITY OF LEFT MAIN OR SEVERE  
3-VESSEL DISEASE**

**URGENT CORONARY ANGIOGRAPHY INDICATED**

**HIGH PROBABILITY FOR BYPASS SURGERY**

*(Sclarovsky S et al. Am Heart J 1988; 116:933)*

# Troponin-positive ACS without tachycardia

## ST-depression during pain

|                 | A = Inverted T | B = Upright T | p-value |
|-----------------|----------------|---------------|---------|
| 0-VD            | 0              | 8             | 0.49    |
| 1-VD            | 0              | 56            | <0.001  |
| 2-VD            | 0              | 8             | 0.49    |
| Non-severe 3-VD | 0              | 20            | 0.05    |
| Severe 3-VD     | 24             | 0             | 0.02    |
| LM- or LME-CAD  | 76             | 8             | <0.001  |

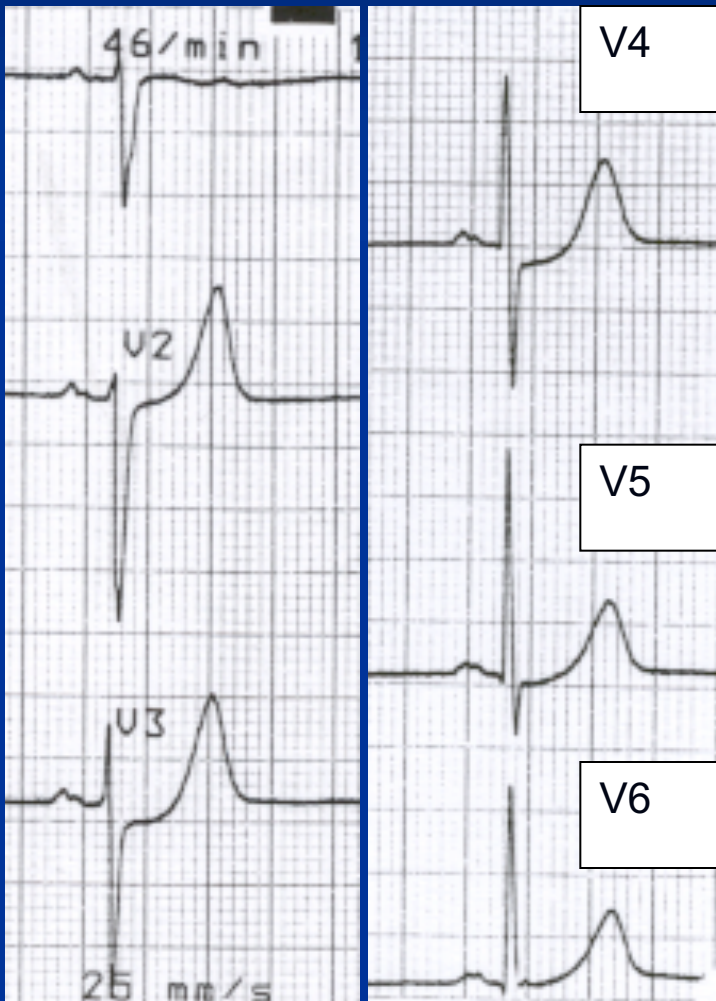
VD= vessel disease; LM= left main; LME= left main equivalent;  
CAD= coronary artery disease

*(Nikus K et al. ANE. 2004; 9: 207)*

## Pattern 2 – Regional subendocardial ischemia

- ST-segment depression and a positive T wave in the same precordial lead has been described as regional subendocardial ischemia
- Transient precordial ST depression recorded during anginal pain with maximal changes in  $V_2$ - $V_4$  with positive T is associated with a subtotal obstruction of the LAD or a total occlusion of a first diagonal or intermediate side branch
- The limited area of ischemia has been proposed as the basis for the discordance between the ST- and T vectors

# Pattern 2 – Regional subendocardial ischemia

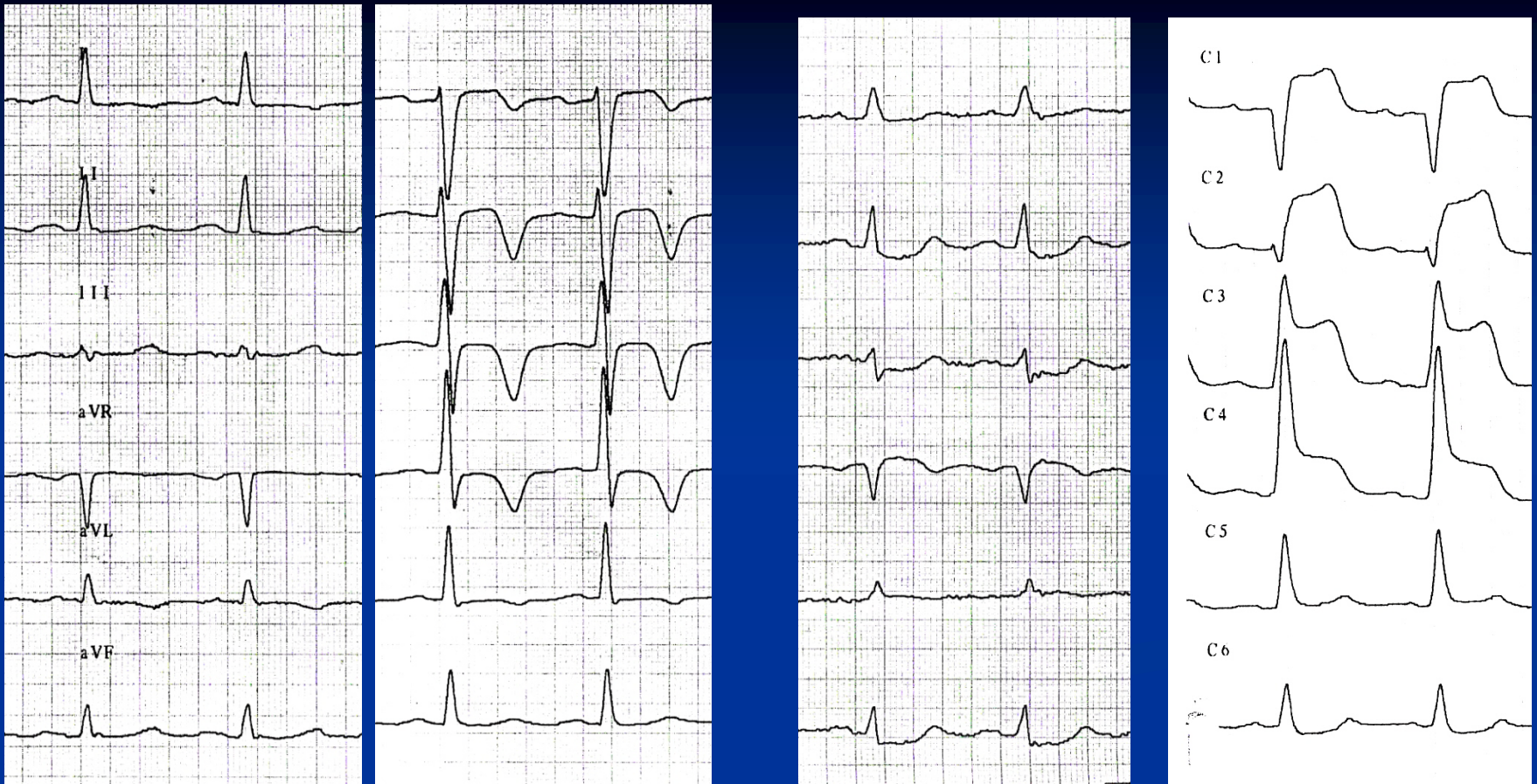


- ST-segment depression and positive T waves in the precordial leads indicate high probability of one-vessel disease, usually significant LAD stenosis or side branch occlusion, treatable with percutaneous coronary intervention
- If the ECG changes are present in V<sub>2</sub>-V<sub>4</sub>, there is high probability of a **sub-total occlusion in the proximal part of the LAD**
- Prompt initiation of anti-thrombotic and anti-ischemic medication followed by invasive evaluation within 24 – 48 hours is recommended

(Sclarovsky S et al. *Am Heart J* 1988;116:933)

# Pattern 3 – The “Wellens’ sign”

- Minimally elevated or isoelectric ST-segments and inverted T waves in the precordial leads without changes in the QRS complex is strongly associated with significant stenosis in the left anterior descending coronary artery
- In the vast majority of cases it represents the post-ischemic reperfusion phase – spontaneous or induced by reperfusion therapy - of an anterior STEMI with a **potential for impending re-occlusion**, especially if not recognized and treated aggressively (*de Zwaan C et al. Am Heart J. 1982; 103: 730; de Zwaan C et al. Am Heart J. 1989; 117: 657*)



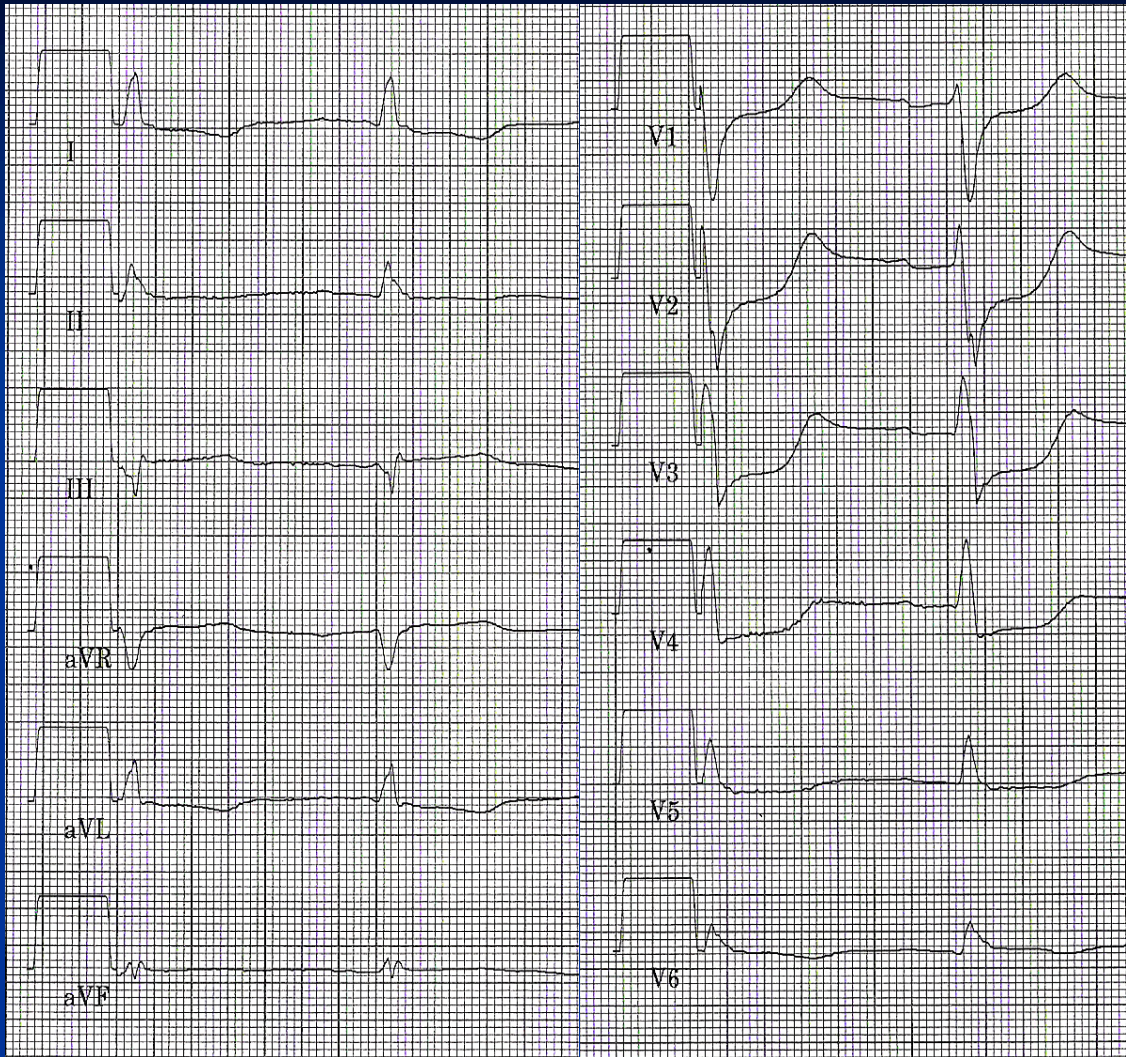
The ECG on the left was recorded at arrival to the emergency department when the patient was pain free. Antithrombotic therapy was initiated and coronary angiography performed within 24 h showed a subtotal occlusion of the left anterior descending coronary artery proximal to the first diagonal branch. Direct stenting was successful. The ECG on the right was recorded less than 15 minutes **earlier** during chest pain, before hospital arrival.



# Pattern 4 – The “mirror-image” STEMI

- ST-depression in leads  $V_1$ - $V_3$  without significant ST-elevations in any of the 12 standard ECG leads represents, with high probability, a “mirror pattern” of transmural injury in the basal inferolateral wall of the left ventricle, and is associated with a culprit lesion in the left circumflex or mid/distal right coronary artery
- In patients with ST-depression in the right precordial leads, especially when accompanied by ST-segment elevation in the posterior chest leads ( $V_7$ - $V_9$ ), reperfusion therapy should be considered, even in the absence of significant ST-segment elevation in any of the traditional 12 ECG leads

*Sclarovsky S et al. Am Heart J 1987;113:1085*  
*Antman EM et al. J Am Coll Cardiol 2004;44:E1*  
*Eskola M et al, J Electrocardiol 2004;37:257*

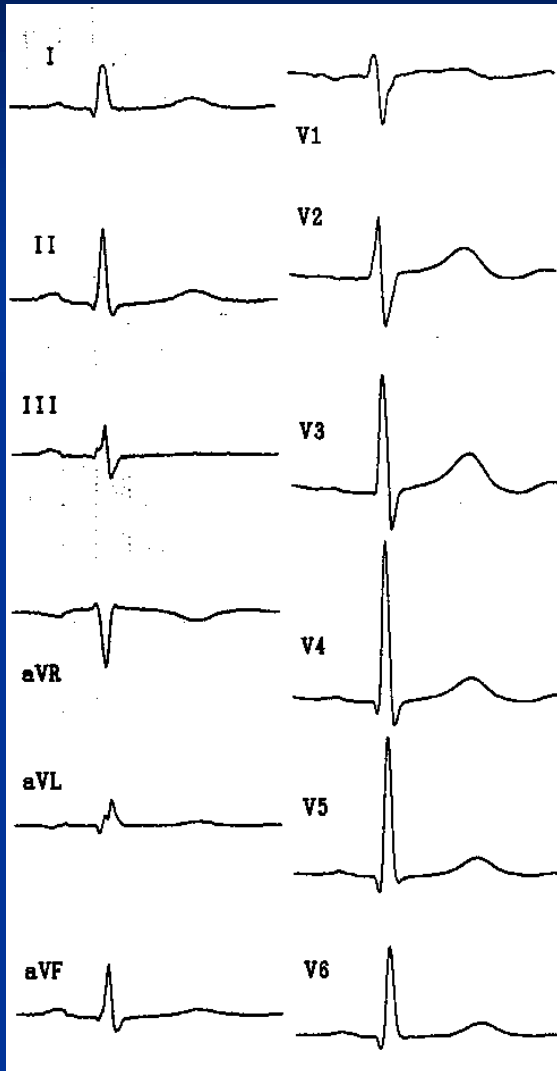


- 12-lead ECG recorded during chest pain shows deep ST-depression in the right precordial leads
- Only minor ST-elevation is present in lead III
- Coronary angiography showed acute occlusion of the left circumflex artery
- Stenting was successful

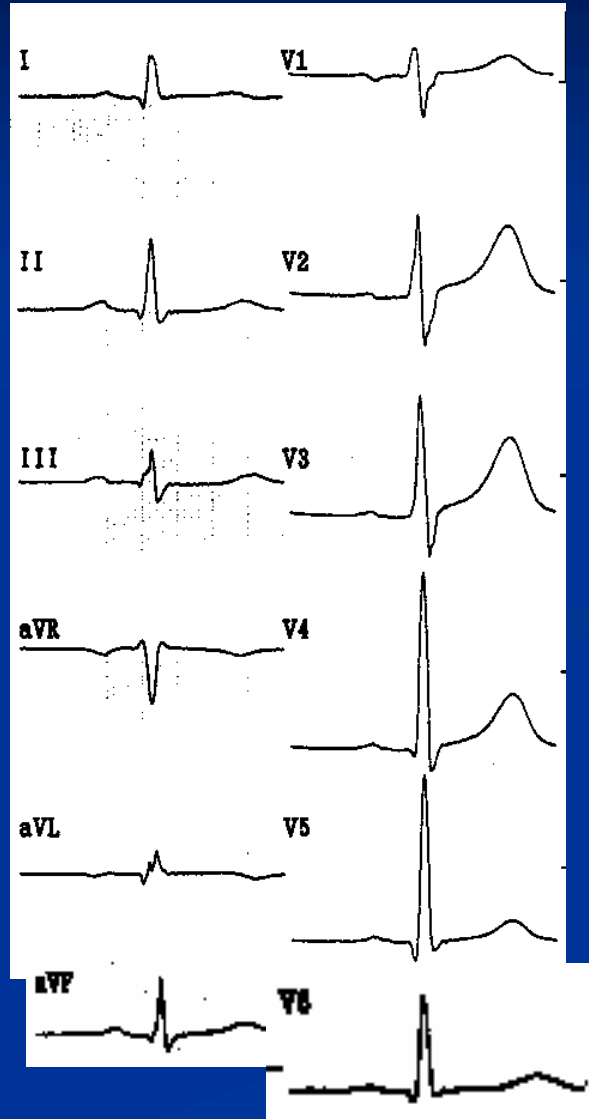
# Pattern 5 – Tall and peaked T waves

- In patients with **transient** peaking of the T waves in the precordial leads recorded during chest pain, the probability of coronary artery disease is very high
- As the state may progress to acute total vessel occlusion, patients should have **close surveillance** with follow-up ECGs or continuous recording

## ECG when asymptomatic



## ECG during chest pain



Repeated ECG recordings or continuous recording are excellent tools to follow the dynamic ischemic process of acute coronary syndromes

Telemedicine-based communication between ambulance- or emergency department personnel and the interventionist enables swift changes in decision-making about need for invasive evaluation or re-evaluation according to ECG-indicated changes in the pathophysiologic process

ECG is a cheap, well-documented, universally available tool for immediate risk stratification of acute coronary syndrome

ECG has been somewhat neglected in the era of high technology

ECG registered during chest pain contains important information about coronary anatomy, myocardial protection and reperfusion

ECG should be used to guide treatment: to find patients who should have emergent or urgent angiography and to decide what lesion to treat in multi-vessel disease