The Electrocardiogram Part II.

In jeopardy more than a century after its introduction by Willem Einthoven?

Time for a revival.

by

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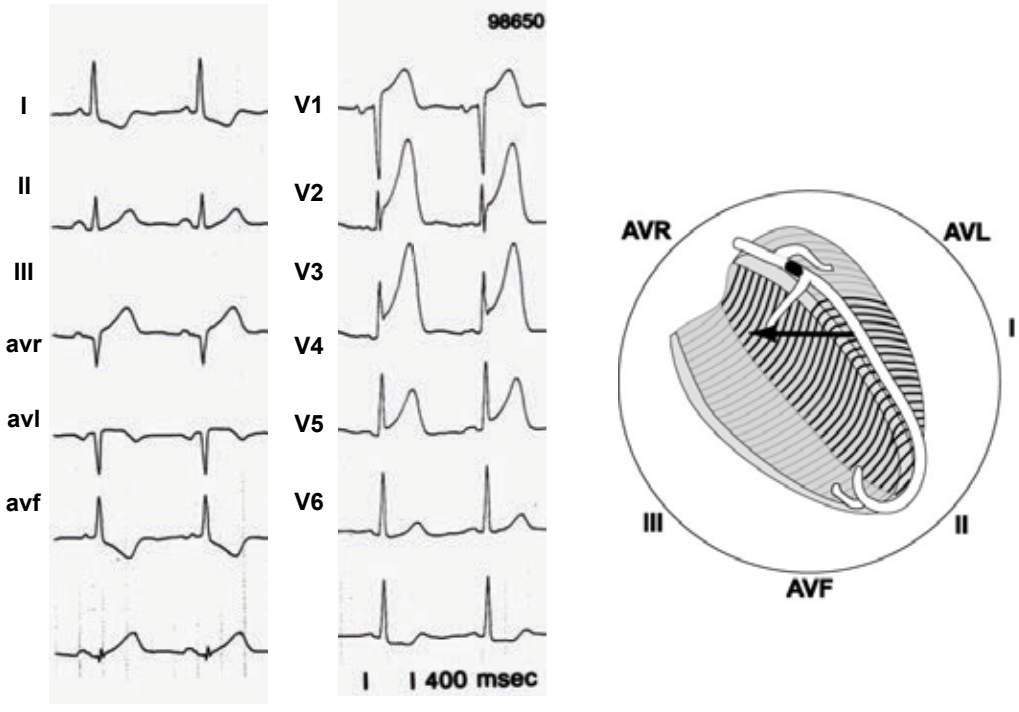

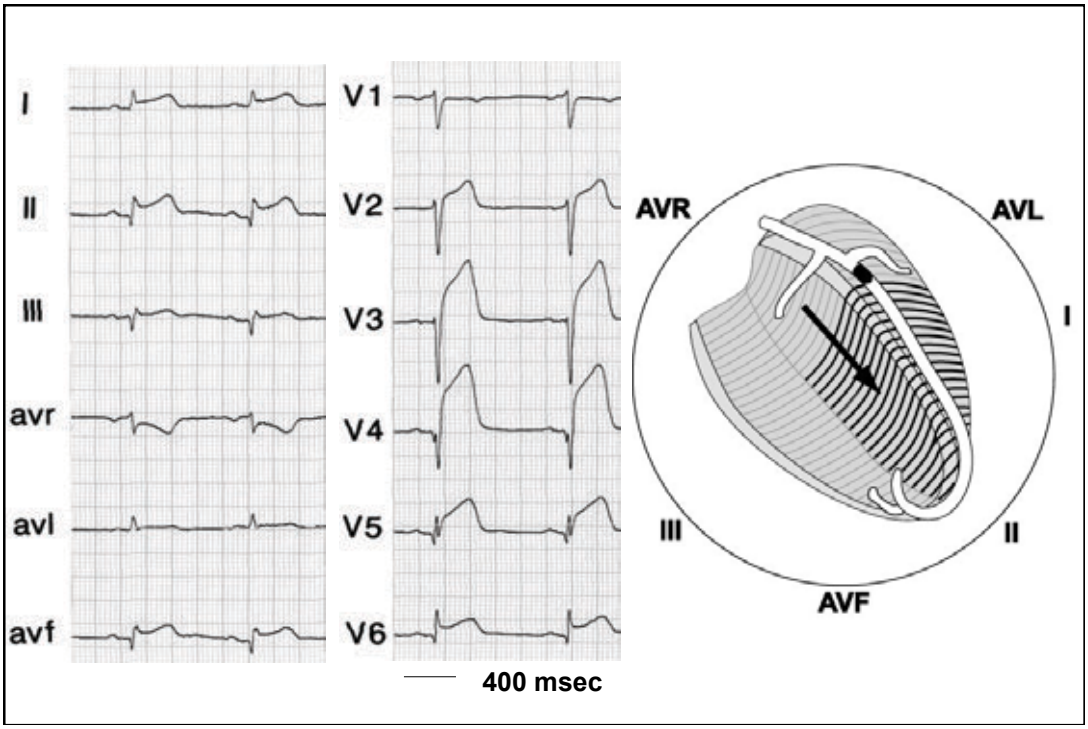
I
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AVR
AVL
I
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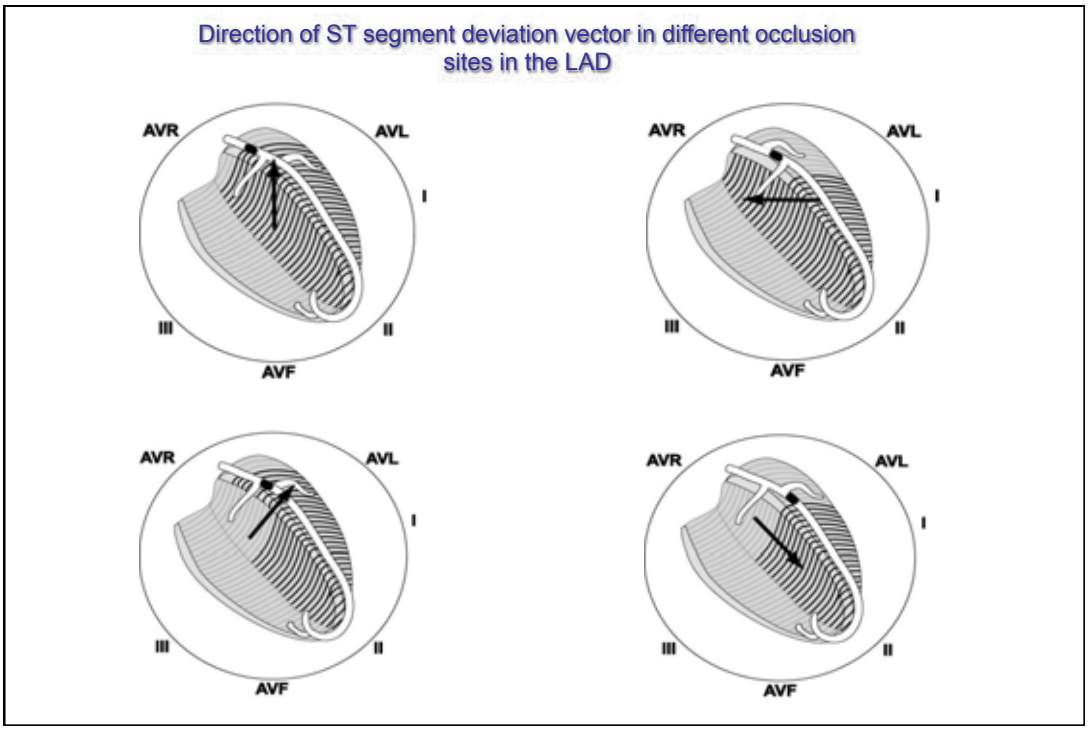
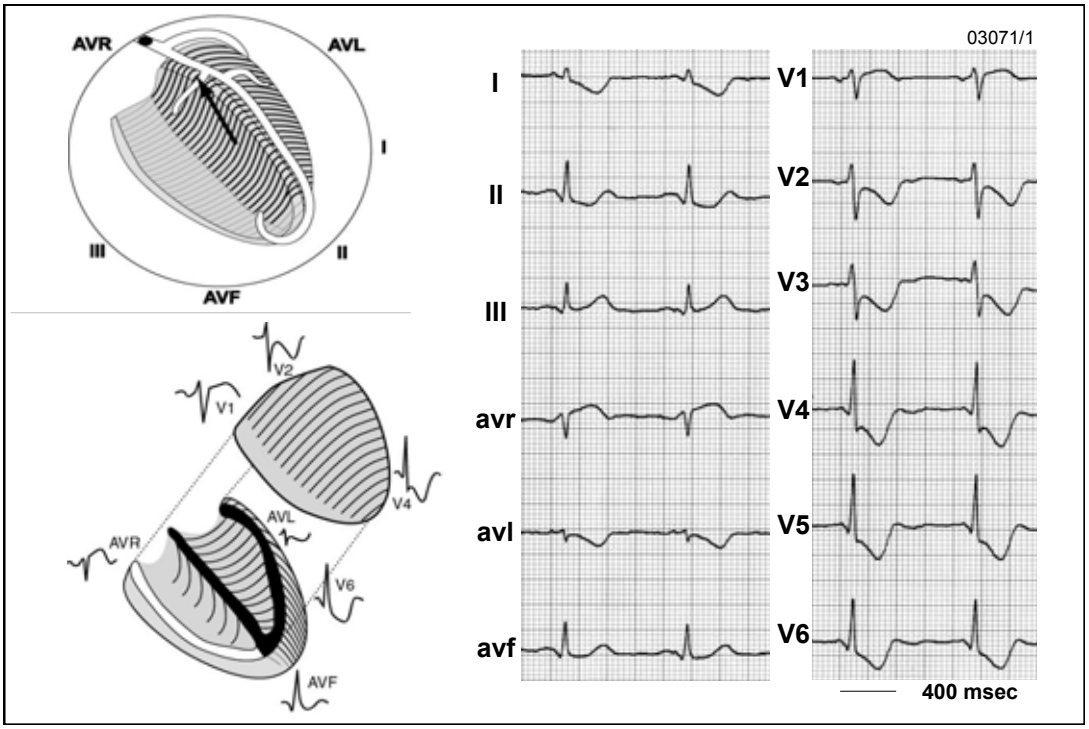
AVF


Direction of ST segment deviation vector in different occlusion sites in the LAD
AVR
AVL
I
III
II
AVF

Approach in anterior wall MI

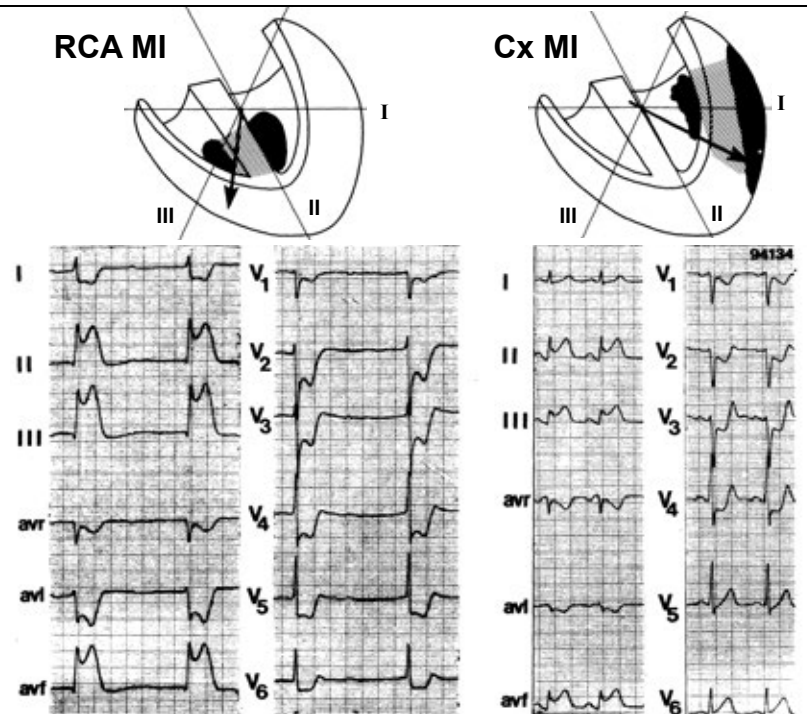
Site of LAD occlusion?	Diagnostic leads
1. Proximal to 1st septal and 1st diagonal branch	II, III. AVR, AVL, V_1
2. Distal to 1st septal , proximal to 1st diagonal branch	II, III, AVL
3. Distal to 1st diagonal, proximal to 1st septal	II, III. AVR, AVL, V_1
4. Distal LAD	II, III, V_4-V_6

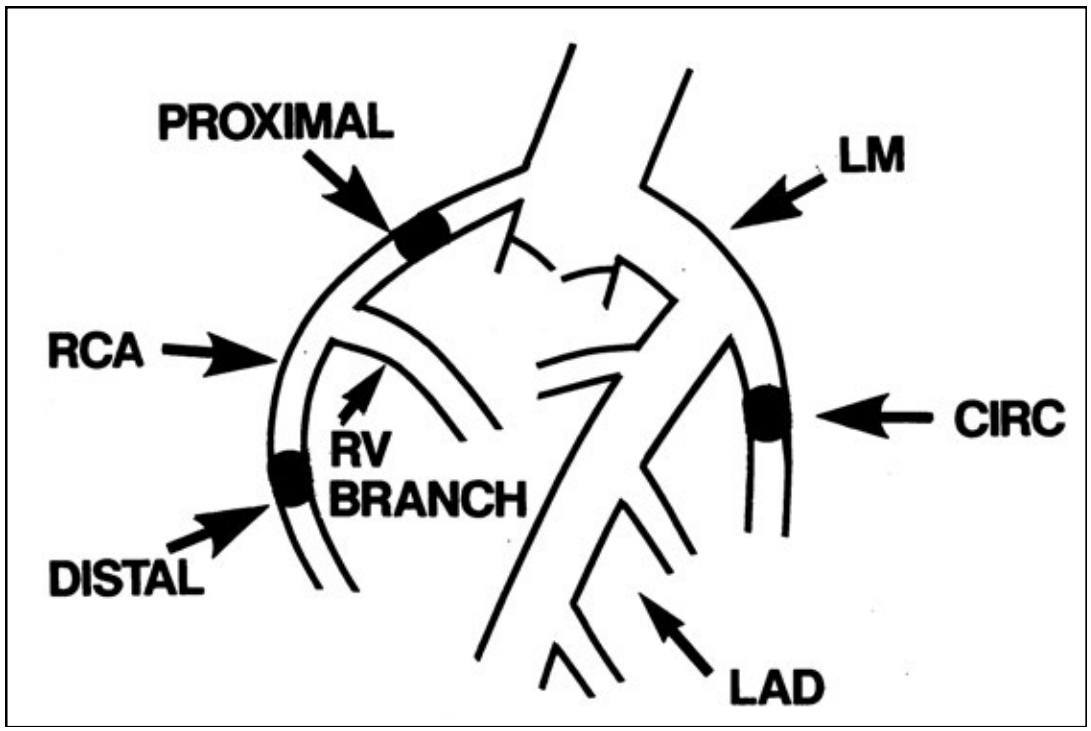


Diagnosis of left main occlusion

ST deviation vector in the frontal plane points to AVR and in the horizontal plane to V_1.

Often wrongly diagnosed as non ST elevation MI.





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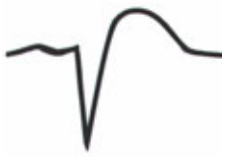
Value of ST-T segment changes in lead V_4R in acute infero-posterior myocardial infarction

ST ↑ ≥ 1mm
Pos T- wave

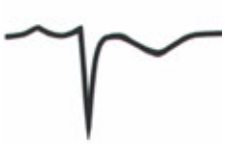


Proximal occlusion RCA

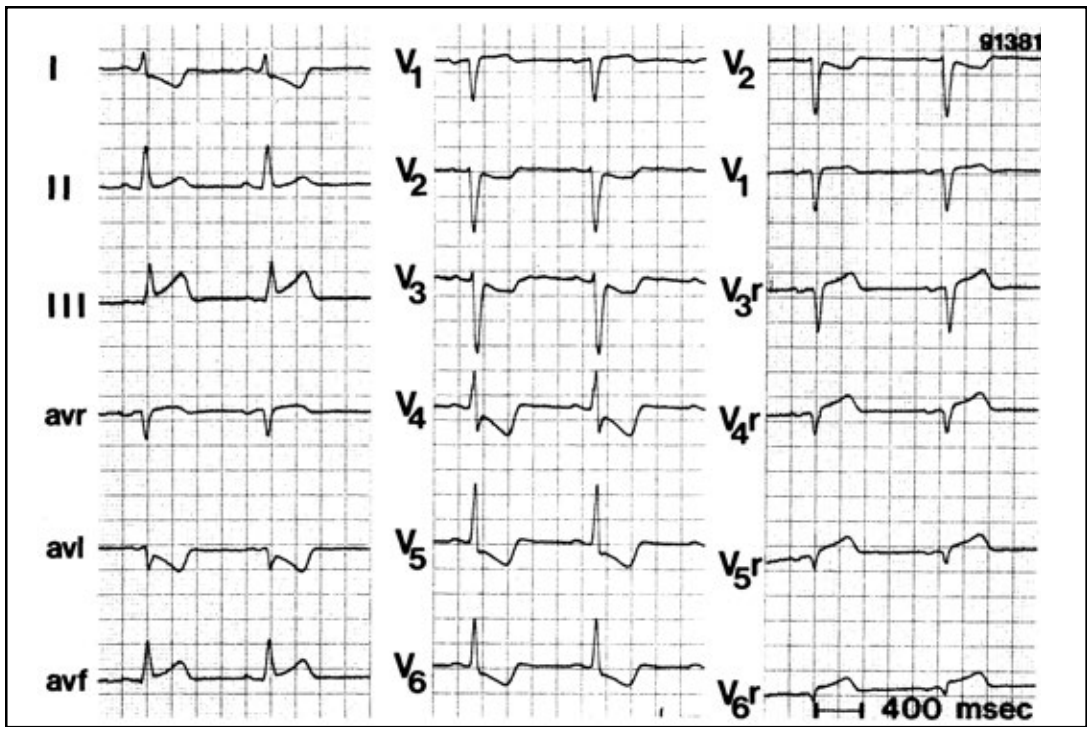
No ST↑:
Pos T-wave

Distal occlusion RCA

Neg T-wave



Occlusion CX


I
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III
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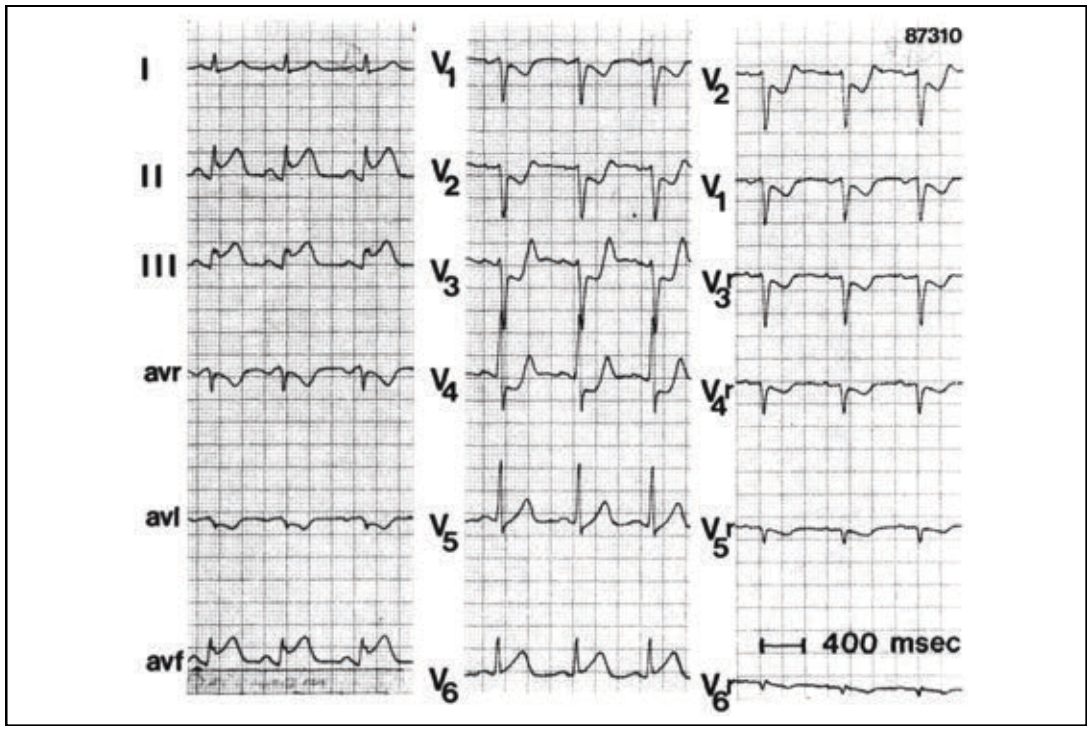
V4r
V5r
V6r
400 msec


I
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V2
V1
V3r
V4r
V5r
V6r
400 msec

Acute infero-posterior MI

Value of V_4R

- Site coronary occlusion
- Right ventricular infarction?
- Risk of AV nodal block

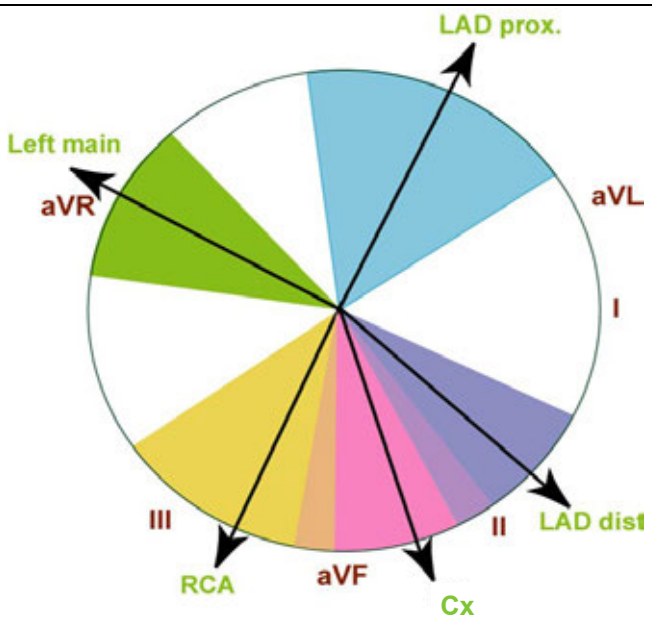
Approach in infero-posterior MI (ST ↑ in leads II, III)

Questions	Diagnostic leads
1. RCA or Cx	II, and III
2. Proximal or distal RCA	V_4R
3. RV involvement	V_4R
4. Posterior wall involvement	Precordial leads
5. Lateral wall involvement	I, avL, V_5 and V_6
6. Atrial infarction	II

Value of the ECG in localizing the site of occlusion in the coronary artery and the size of the area at risk

- Excellent in single vessel disease
- Of less value in:
  - Pre-existent ST changes
  - Following previous CABG or PCI
  - Multi vessel disease
  - Scar in another area than that of the culprit coronary artery
  - LBBB, pre-excitation, V-pacing

However: 12 lead ST deviation ≥ 5 mm should be present!

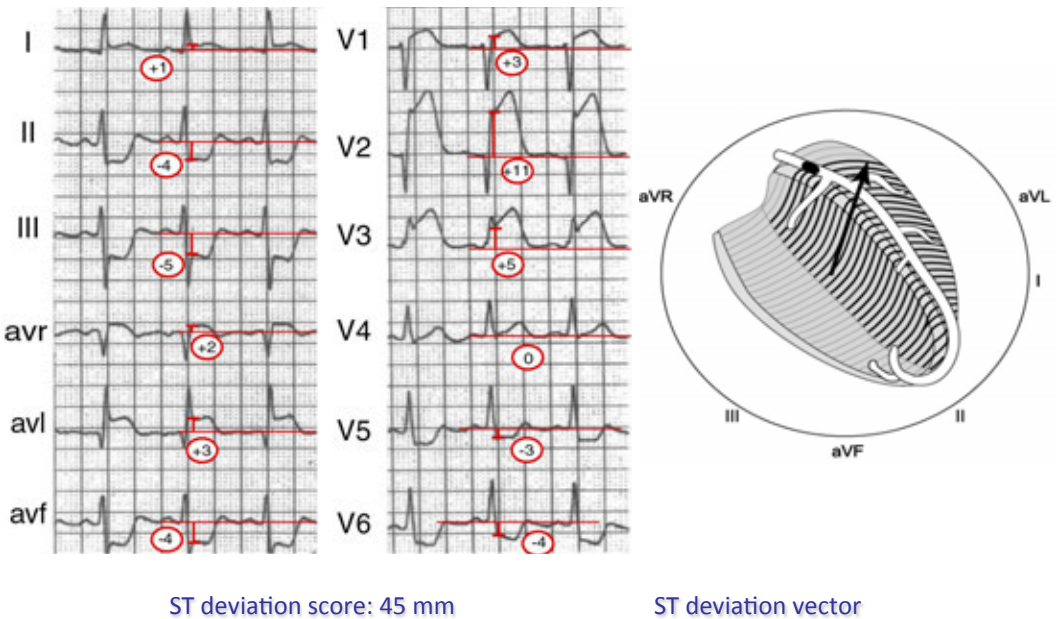


Direction of ST segment deviation in relation to coronary occlusion site in acute MI.

"Can the 12 lead ECG locate the coronary culprit lesion and thereby the size of the area at risk?"

To answer this question an algorithm was used with computer-assisted ST-deviation measurements at 60 m after the J point, measuring both ST elevation and ST depression.

This allowed the determination of the 12 lead ST segment deviation score and the direction of the ST deviation vector.



ST deviation score: 45 mm

ST deviation vector

The culprit lesion sites suggested by the algorithm

- Left main
- LAD proximal (proximal to S_1, D_1 or both)
- LAD distal (to S_1 and D_1)
- RCA proximal
- RCA distal
- Cx
- Multivessel disease
- No localization possible

To differentiate the occlusion site in the proximal and distal RCA and Cx, lead V_4R was recorded instead of lead V_4 after demonstrating that ST deviation in lead V_4 is the mean of ST deviation in lead V_3 and V_5.

386 acute chest pain patients had an artefact free 12 lead ECG made out-of-hospital by ambulance personnel.

Within one hour a coronary angiogram was performed in the Marien hospital (Ruhr-Universität Bochum) in Herne.

In those patients the ECG algorithm diagnosis was compared with the findings on coronary angiography.

Data on the 386 patients studied

Men: 280 (mean age 61,8 ± 13,2 years)

Women: 106 (mean age 69,3 ± 13,4 years)

Previous MI, CABG or PCI: 44

QRS ≥ 120 ms: 53

SR without known CAD and QRS<120 ms: 289

ST dev. score
≥ 5 mm: 185
≤ 4 mm: 104

Predictive value of the ECG algorithm

Sinus rhythm without known CAD or QRS < 120 ms

ST deviation score ≥ 5 mm: 185 pts

ST deviation score ≤ 4 mm: 104 pts

Required information to make a risk profile in "early repolarization" (1)

ECG: J-wave pattern II,III,aVF ?
- Height J-wave?
- ST segment: elevation, positive T wave?
- Length QT, rate relation?

Provocation: Effect of spontaneous or induced (Valsalva, Carotid sinus massage) rate changes on J-wave pattern and ST-T segment?

Required information to make a risk profile in "early repolarization" (2)

Ventricular ectopy: Present?
Coupling interval?
Width?
Site of origin?

Age,Gender,Ethnic background?

Correlation between coronary angiography and ECG algorithm findings in 185 patients with ST deviation score of 5 mm or more

Site of coronary occlusion according to ECG algorithm

Coronary Angiogram Diagnosis	Left Main	LAD Prox.	LAD Dist.	RCA Prox.	RCA Dist.	CX	Multi vessel	No location possible	Correct diagnosis	%
Left Main	13						1		13/14	93
Prox. LAD	1	54	1						54/56	96
Dist. LAD		1	22		1				22/24	92
Prox. RCA				36					36/36	100
Dist. RCA			1	1	10				10/12	83
CX		1		1	1	12	1		12/16	75
Multi vessel	2		1		1		9		9/13	69
Coronary narrowing			3	1	2			1	0/7	0
No occlusion		2	3	1				1	0/7	0

Correctly predicted over all: 156/185 (84%)
Proximal (high risk!) occlusion predicted: 103/106 (97%)

Coronary angiographic and ECG findings in 104 patients with ST segment deviation score of 4 mm or less

ECG findings	Coronary angiographic findings		
	No CAD	Coronary occlusion	Coronary narrowing > 70%
Q-wave MI		18	20
Negative T-waves		6	11
Only ST segment deviation	25	7	17
Total	25	31	48

Note that in this cohort several patients were in a later stage of cardiac ischemia (Q waves, T wave inversion)

QRS ≥ 120 ms (13 % of the cohort)

LBBB	25	44/49 severe CAD
RBBB	24	
VT	3	
V pacing	1	

- ST deviation vector affected by abnormal repolarization in case of a widened QRS.
- In LBBB and acute chest pain no reliable localization coronary culprit lesion.
- In RBBB correct localization "culprit" lesion in 11/13 with an ST deviation score > 5 mm.

In the 44 patients with a previous MI, CABG or PCI (11% of the cohort) the ST deviation score/ vector approach gave no reliable localization of the culprit lesion. Most of these patients had extensive multi vessel disease on coronary angiography.

Conclusions

1) In patients with sinus rhythm, no previous CAD and a QRS < 120 ms (75% of the total cohort) the combined ST deviation score/vector approach allows recognition of the high risk group because of a proximal coronary occlusion, which will maximally profit from rapid reperfusion.
2) In patients with acute chest pain with a previous MI, CABG or PCI and in patients with bundle branch block (25 % of the total cohort) the combined ST deviation score/vector approach usually does not indicate the culprit coronary artery. However the extent of CAD found in those pts indicate that referral to a site allowing rapid PCI is required to determine appropriate treatment.
3) The 12 lead ECG at first medical contact is indispensable for risk stratification, and rapid decision making as to appropriate treatment.
4) The above mentioned findings were integrated in an automated ECG algorithm and published : A.Meissner et al. Netherlands Heart Journal 2010;18:301-306.

Increasing super-specialization in cardiology threatens the implementation of new ECG knowledge in daily cardiology practice.

Both old and recent knowledge of the ECG should be in the core curriculum of every cardiologist, not only during the training phase but also during postgraduate education!

Monogenic disease with increased risk for sudden arrhythmic death

Primary electrical	Structural
LQT	HCM
Short QT	ARVD/C
Brugada	Dilated CM
Cath Poly VT	Lamin A/C CM
Idiop VF	Myotonic dyst 1
Early repolarization	
Early coupled VPB's	

Specific ECG findings helpful in directing genetic analysis in monogenic disease with increased risk of sudden arrhythmic death

	ECG	Nb genes	Relation Phenotype/Genotype	
			rest	provocation/tachy
Primary electrical	LQT	13	60%	70%
	Short QT	3	?	?
	Brugada?	7	15-30%	30%
	Cath Poly VT	2	?	70%
	Idiop VF	1	0	?
	Early repolarization	1	?	?
	Early coupled VPB's	0	?	?
Structural	HCM	15	0	?
	ARVD/C	6	?	?
	Dilated CM	3	?	?
	Lamin A/C CM	1	± 30%	?
	Myotonic dyst 1	1	?	?

The ECG in monogenic arrhythmology

- High specificity but low sensitivity because of marked differences in phenotypic expression.
- Large data bases required.
- ECG helpful in decision making about management (Long QT, Brugada).

Areas where our ECG knowledge should improve

- Risk stratification for sudden death
- Geno-phenotype in genetic arrhythmology
- Better information about location and electrophysiologic – structural relations of the arrhythmia substrate
- Value in selecting and evaluation of regenerative therapy: cell transplantation, gene therapy

Increasing super-specialization in cardiology threatens the implementation of new ECG knowledge in daily cardiology practice.

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Limitations of the 12 lead ECG (1)

- More global than local information
- Dynamic nature of cardiac disease
  - QRS
  - ST-T segment
- Cardiac areas partially or completely hidden during normal ventricular activation
  - Example: Cx territory

Limitations of the 12 lead ECG (2)

Cardiac areas partially or completely hidden during abnormal ventricular activation

- Left bundle branch block
- Ventricular pre-excitation
- Ventricular pacing
- Scar tissue
- Ventricular escape rhythm
- Ventricular tachycardia
- Hyperkalemia

What needs to be done?

- More ECG leads?
- Combining ECG findings for risk stratification?
- Frequency spectra?
- Non-invasive epicardial activation-repolarization imaging?
- Non-invasive three dimensional information about the relation between electrical behavior, structure and mechanical function?

Questions about the J-point elevation pattern (1)

1. The name : - J-point elevation
   - J-wave syndromes
   - "Early" repolarization

2. The mechanism: - "Early" repolarization
   - Delayed depolarization

3. Morphology: - J-point elevation
   - ST segment

Questions about the J-point elevation pattern (2)

4. Clinical significance: - Race
   - Gender
   - Age
   - ECG lead location
   - Pattern
5. Arrhythmia origin and mechanism
6. Provocative tests
7. Genetic background
8. Registries
9. Management

Decline in knowledge on how to obtain relevant information from the ECG, not only during training to become a cardiologist, but also by the practising cardiologist

Reasons:

- No mandatory structural ECG education during training and post graduate
- Automated ECG interpretation
- Diminished interest in prospective studies evaluating value of new ECG findings
- Difficulty to obtain financial support for ECG studies
- ECG reimbursement

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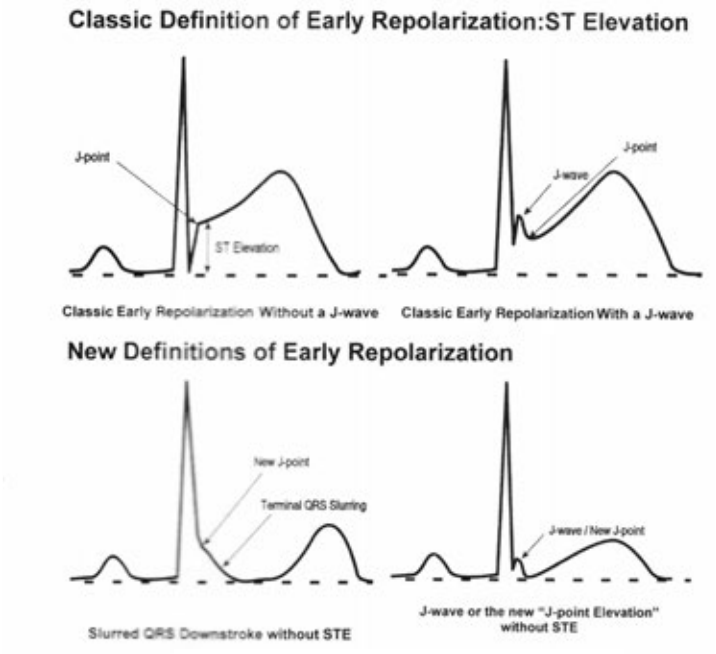

Perez MV et al. Am J Med 2012; 125:843

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The ECG is at a crossroads as to its future integration into modern medical practice. Those most interested in electrocardiography remain the old guard, whose careers evolved with this technology. They remain as enamored by the experiential mythology as by the experimental science of the ECG. Electrophysiologists, who rightly should be carrying on the torch of further ECG development, are too busy with their therapeutic invasive procedures and devices to invest much time in diagnostic decision support. Young physicians in training are too busy learning the plethora of new diagnostic modalities and treatment procedures to even become competent in ECG interpretation. Many of them only have goals to recognize an ST elevation myocardial infarction and atrial fibrillation, and to pass their board examinations. Their understanding of ST elevation myocardial infarction criteria could be easily exposed by asking them to name the contiguous pairs of standard ECG leads. A disappointing number would refer to pairs of leads that are contiguous on the ECG display such as II and III or V_1 and V_4, rather than the leads separated by 30° going around the surface of the heart as specified in the guidelines.[1] Reimbursement provides a further counterincentive: to paraphrase George Bernard Shaw (*The Doctor's Dilemma*, 1926), "the doctor orders the test that pays the most" and that is no longer the ECG, but a panoply of imaging procedures.

"The Electrocardiogram at a Crossroads"
Yong CM et al. Circulation 2013; 128: 89