Transient Prominent Anterior QRS Forces (PAF) in the setting of Acute Myocardial infarction: which is the mechanism?

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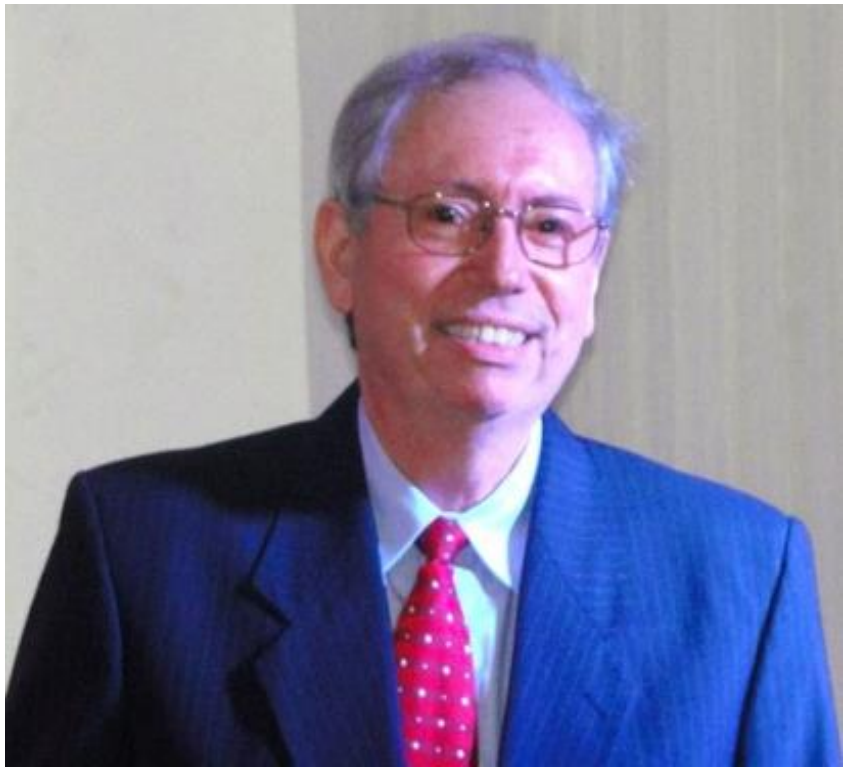
https://ekgvcg.wordpress.com



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Name: AS; **Age:** 51 y/o (**Date of birth:** 09/15/1961); **Sex:** M; **Ethnic group:** Caucasian: **Weight:** 82 kg; **Height:** 1.76 m; **Profession:** journalist
History of present illness: The patient is a 51 year-old gentleman with a potential history of coronary artery disease (CAD) in the past, who had undergone a stress test approximately 3 years ago while living in Brazil. The patient has been visiting the United States and after a few days began developing chest discomfort with shortness of breath on exertion as well as chest pressure. The symptoms got worse and he proceeded to the Emergency Department. The patient's ECG was unremarkable at the time. His symptoms were relieved with medications, but troponins were significantly elevated and he ruled in for AMI. He underwent a cardiac catheterization on June 27th revealing **Multi-vessel coronary disease**. Cardiac surgery department was consulted. The doctor spoke with the patient and his family to discuss indication, risks and alternatives of the proposed bypass surgery. After discussion, the patient did wish to proceed. He underwent the coronary artery bypass graft x5 on July 2nd without any complication and has improved postoperatively that he is ready for discharge at this time.
Date of admission: 06/25/2013
Date of discharge: 07/07/2013
Admitting diagnosis: Acute Myocardial infarction (AMI)
Primary discharge diagnosis: Status post urgent coronary artery bypass graft times 5 on July 2, 2013.
Pathological personal background:

- CAD: actual Acute myocardial infarction on June 25, 2013
- Hypertension
- Non-insulin-dependent diabetes mellitus (type 2)
- Anemia
- Dyslipidemia
- Mild gastroesophageal reflux disease (GERD).

Procedures performed: coronary artery bypass grafting (CABG) x 5 on July 2, 2013
Hospital course by systems
Neurologic: The patient has been doing from a neurologic standpoint. He has not had any neurologic deficits postoperatively. He has been seen by the physical therapist to assist in increasing the ambulation and activity and has done stairs multiple times. His pain has been well managed Tylenol, oxycodone p . r. n. He will need to observe sternal precautions, which have been discussed with him multiple times.

Cardiac: The patient has a normal Left ventricular ejection fraction(LVEF) of approximately 60%.
Postoperatively he has remained in a sinus rhythm. No postoperative dysrhythmias. Low-dose of β-blockers were initiated and titrated up as blood pressure and heart rate as tolerated. His epicardial pacing wires removed in a timely fashion without any incident. To note, this patient did have episodes of 2:1 heart block preoperatively and did have a transvenous pacing wire placed preoperatively. Postoperatively he has not had any block. β-blockers were able to be increased and he has not had any rhythm problems.
Pulmonary: The patient was extubated very soon after arrival into the intensive care unit. He has been doing his incentive spirometer, deep breathing and cough exercises. Chest tubes placed in the operating room were removed postoperatively in a timely fashion. He has been receiving nebulizer treatments p . r. n.
Renal: The patient's baseline creatinine is 0.8. He did not have any postoperative renal dysfunction. The patient had diuretics given to him to assist with bringing his weight down and bringing his back towards a euvolemic state. The patient will be discharged to home with several days of diuretics. Foley catheter placed in the operating zoom removed and he has been voiding without difficulty.
GI: The patient's diet was increased as tolerated. He has been tolerating a consistent carbohydrate diet. He is eating well and moving his bowels postoperatively.
Endocrine: The patient is a known diabetic, who is on metformin 850 b. i. d. at home. Initially the patient was on an insulin infusion and he was subsequently transitioned back to his Glucophage. The dose wag increased back up to his preoperative dose of 850 mg b. i. d. A Glucometer was provided for the patient and he will need to check his fingersticks twice a day, fasting and before supper, to insure adequate glucose control. This procedure was reviewed with both the patient and his family by the nursing staff. He was seen by nutrition tor diabetic diet reinforcement.
Hematologic: The patient did not require transfusions of any blood bank products. He does not require anticoagulation. He was started on an enteric-coated aspirin which he will need to continue.
Infectious disease: The patient has been afebrile. He received standard perioperative antibiotics, His incisions are clean, dry, and intact. He has had no postoperative fevers or cultures and has no cultures pending at this time.
Condition at discharge: The patient's physical exam at discharge includes no acute distress. His vital signs are stable. He is afebrile with temp of 36.4°C, heart rate sinus rhythm at 80 beats per minute, blood pressure 102/71, respirations 16, 0_2 sat are 97% on room air. Preoperative weight 100.5 kg, discharge weight 102.1 kg. HEENT: NC/AT, PERL, EOMI, nonicteric, OP negative. Neck: Supple. No JVD or bruits. Lungs: Clear to auscultation, very minimal decrease at both bases . Cardiac: SI, S2 regular without murmur or rub. Chest: Incision on the sternum and donor leg are clean dry and intact, sternum is stable . Abdomen: Softly distended, nontender, positive bowel sounds. Extremities: Warm x4. 1+ pitting edema in his lower extremities. Neurologic: Grossly intact. No focal sensory, motor or cognitive deficits.

Discharge data: Includes laboratory study WBC of 9.6, hemoglobin 8.5, hematocrit 25.5, platelet count 195, sodium 130, potassium 4.5, and chloride 98, $C0_2$ 26, BUN 17, creatinine 0.7, glucose 118.

ECG Via telemetry shows the patient to be in a sinus rhythm without any acute ST charges.
Two view PA and lateral chest x-ray performed July 5, 2013 showed a slightly poor inspiration, tiny pleural effusion with possible minimal left basilar atelectasis.
Allergies: Patient has no known drug allergies or adverse drug reactions.

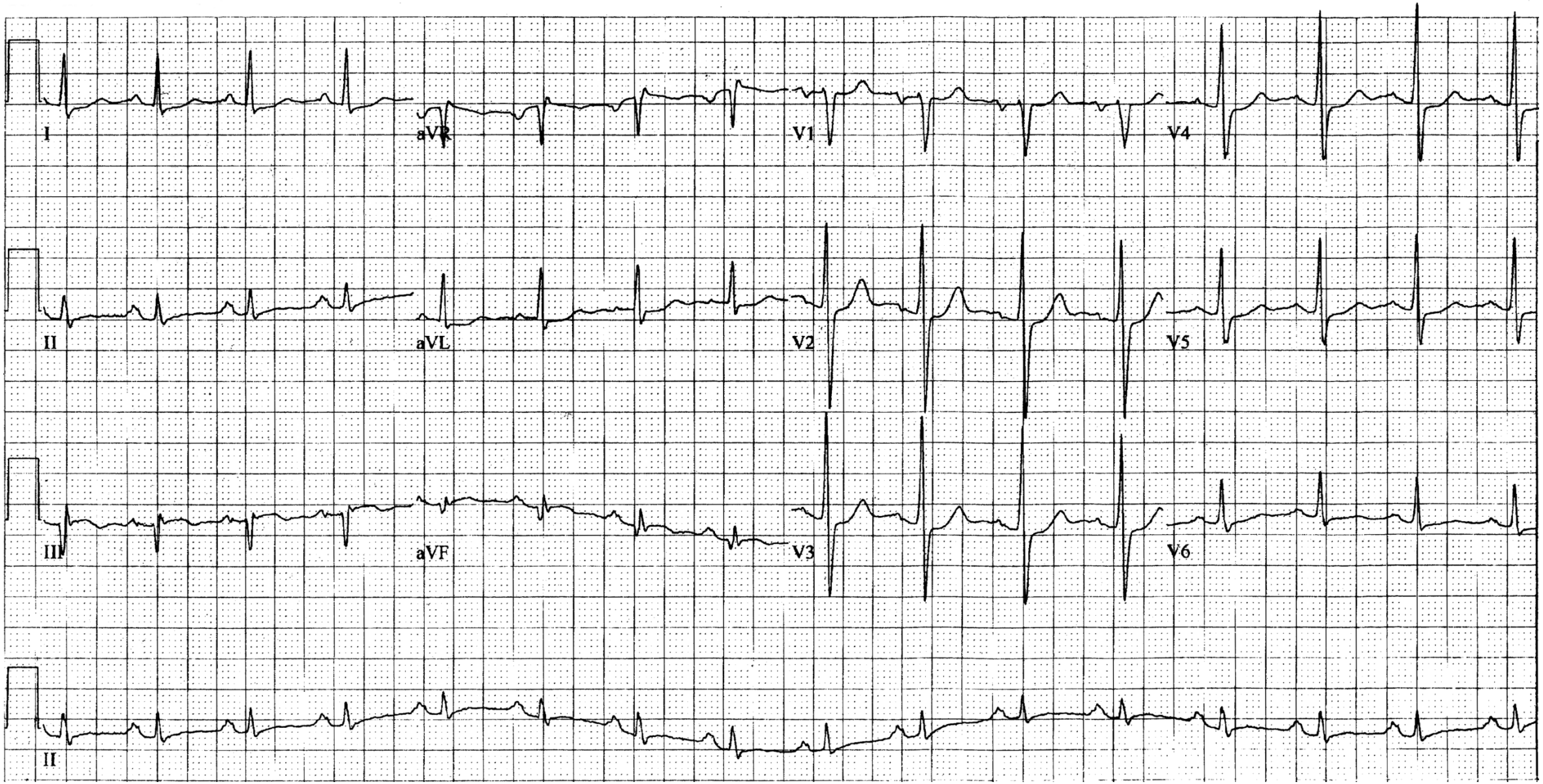
Medications on discharge:

- Tylenol® (acetaminophen) 650 mg p.o. q. 6 hours p.r.n.
- Enteric-coated aspirin 325 mg p.o. daily
- Atenolol 50 mg p.o. daily
- Colace® (docusate) 100 mg p.o. b.i.d., hold for loose stool
- Furosemide 20 mg p.o. daily x5 days
- Metformin 850 mg p.o. b.i.d.
- Oxicodone-hydrochloride 5 mg p.o. b.i.d.(it is a semisynthetic opioid synthesized from thebaine, an opioid alkaloid found in the Persian poppy, and one of the many alkaloids found in the opium poppy.)
- Potassium 10 mEq p.o. daily x5 days
- Pravastatin 20 mg p.o. at bedtime
- Senokot® (Sennosides) 17.2 mg p.o. b.i.d., hold for loose stool (it is a treating for constipation, a stimulant laxative. It works by irritating bowel tissues, resulting in bowel movements).

Discontinued medications: Enalapril 10 mg p.o. daily. To note, all of the above discharge medications are new for the patient with the exception of metformin 850 mg p.o. b.i.d., which was an admission medication.

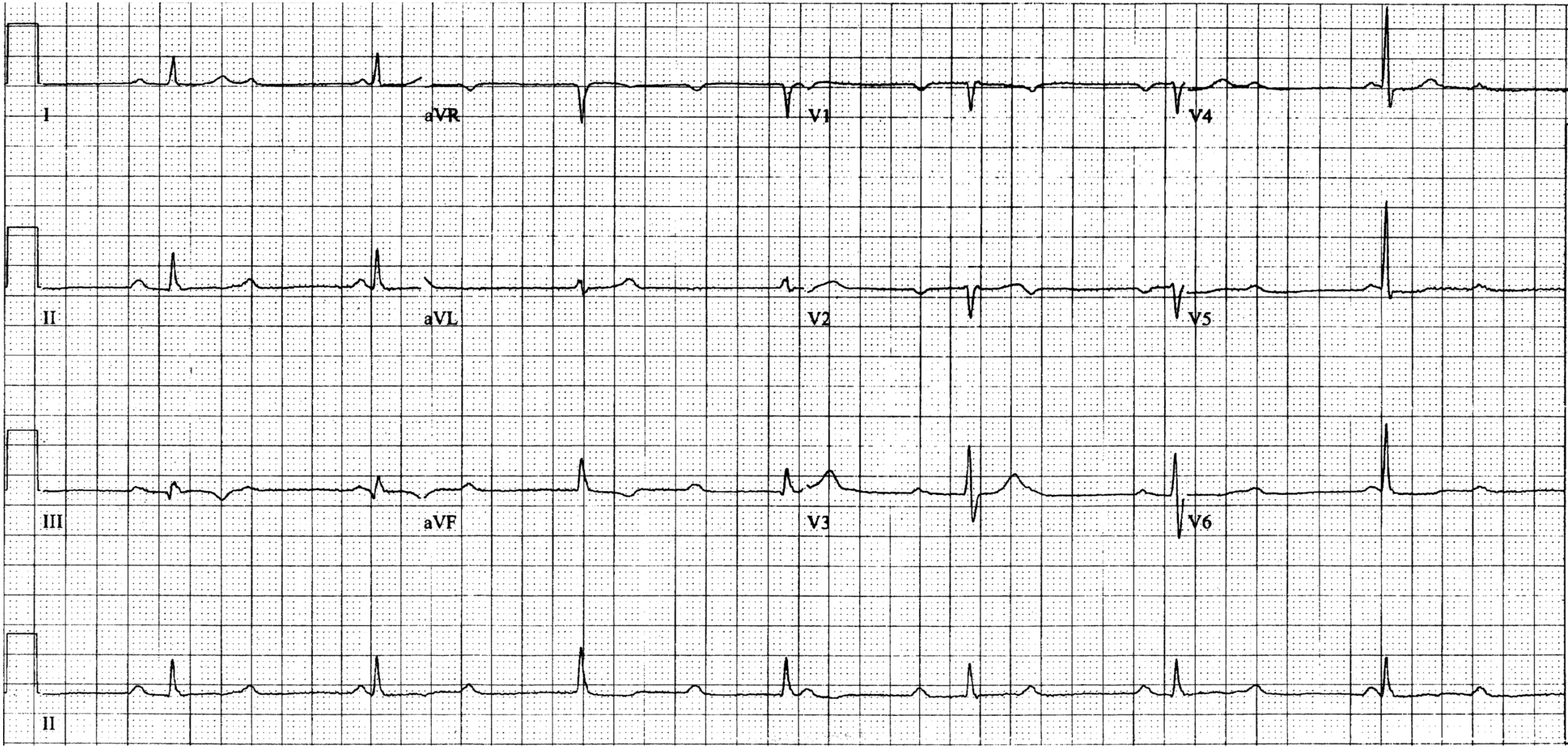
Question
Which is the explanation for transient Prominent QRS Anterior Forces (PAF) observed in **ECG-3 June 29, 2013**?

ECG-1 June 25, 2013



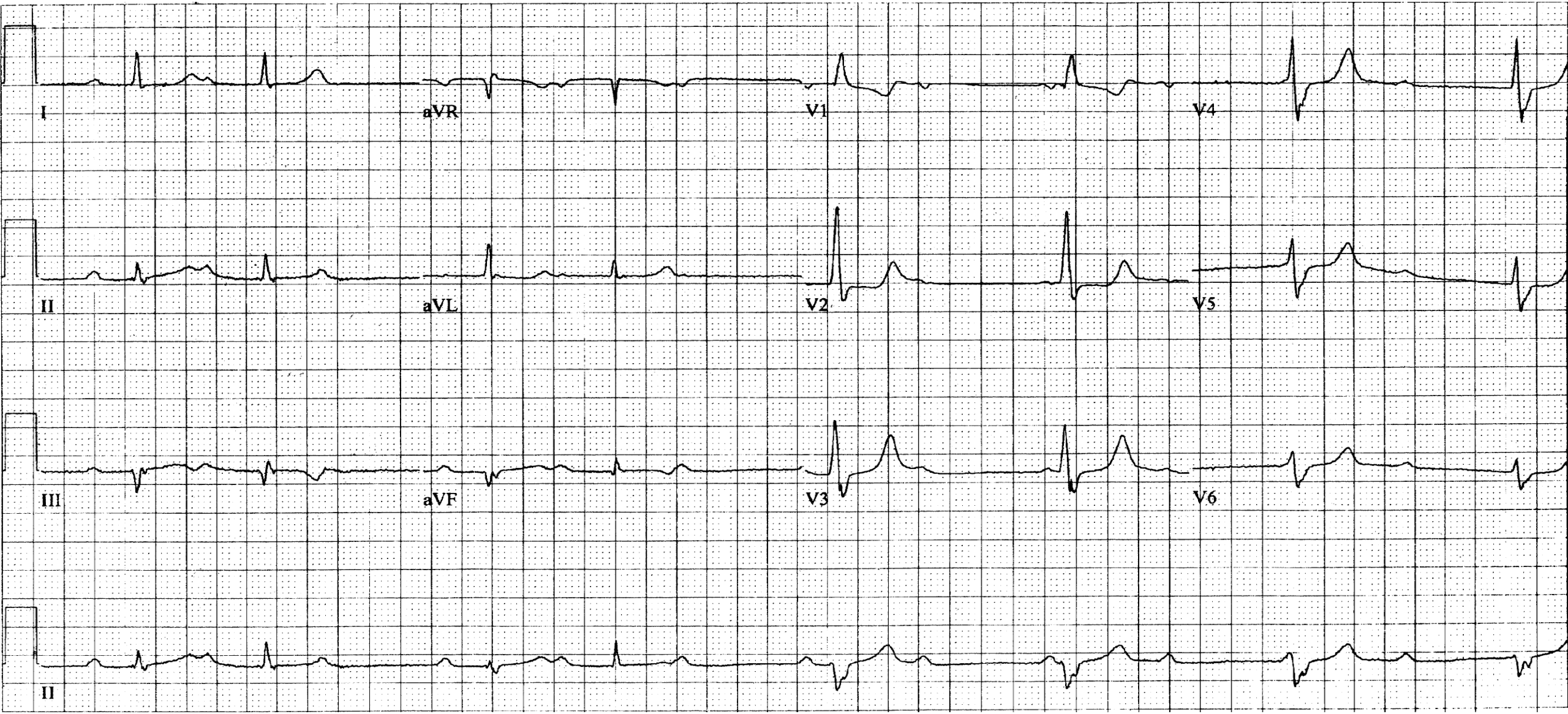
ECG diagnosis:

ECG-2 June 28, 2013



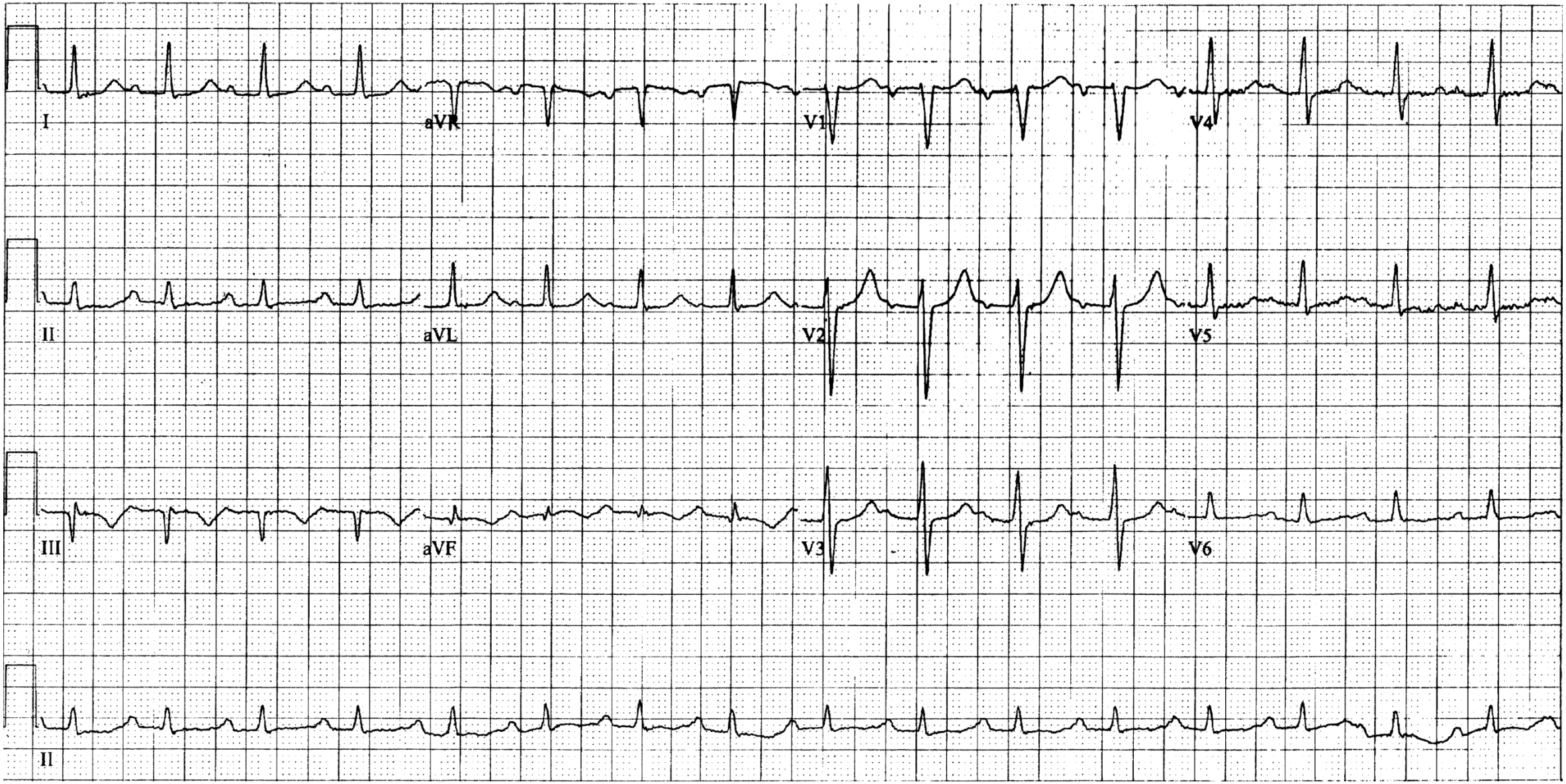
ECG diagnosis:

ECG-3 June 29, 2013



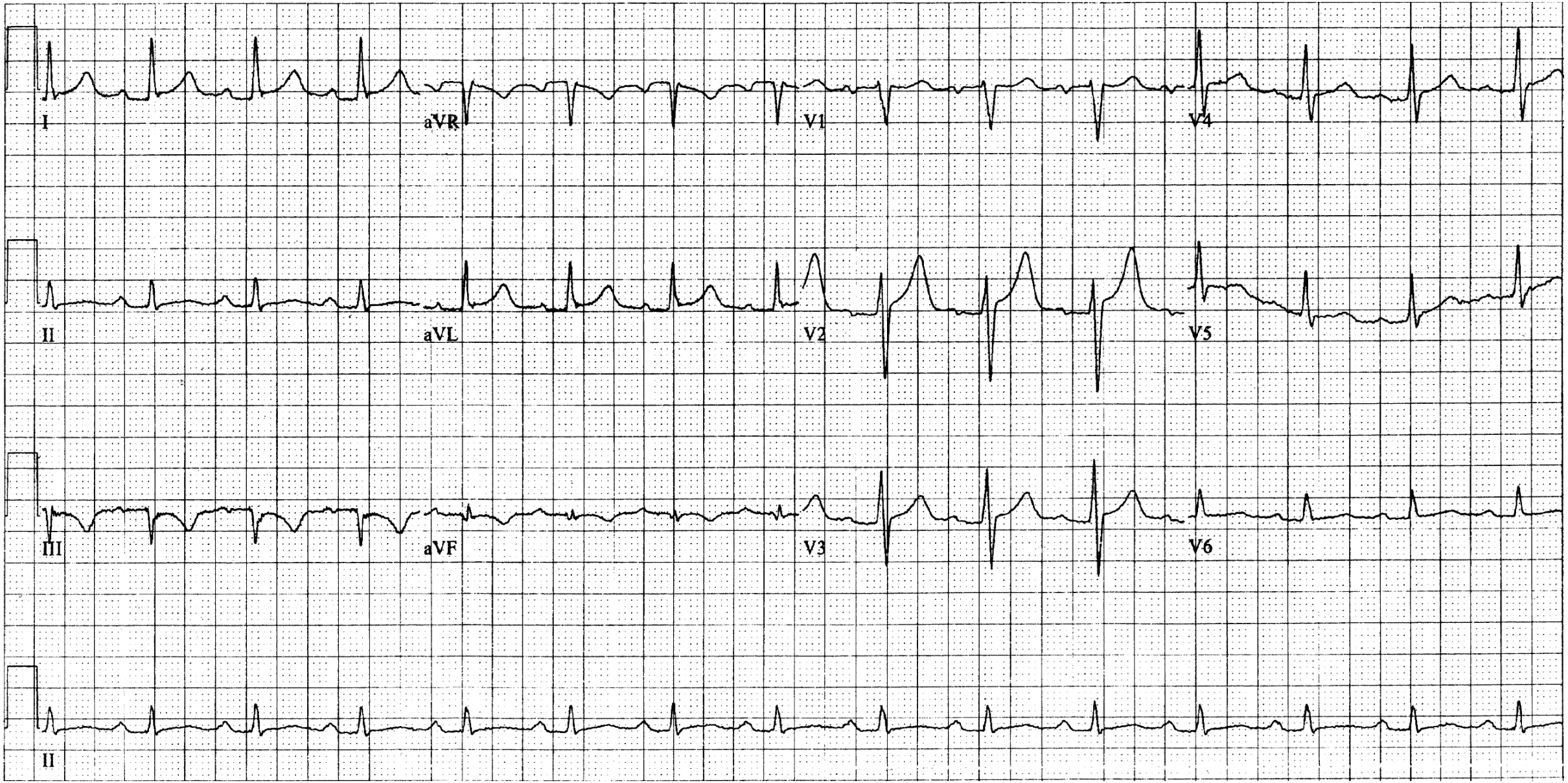
ECG diagnosis:

ECG-4 July 02, 2013



ECG diagnosis:

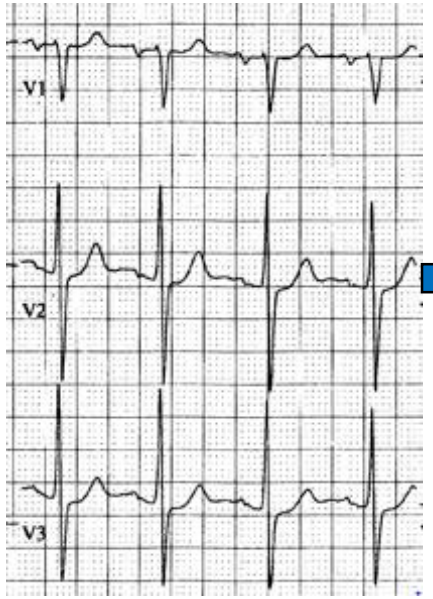
ECG-5 July 03, 2013



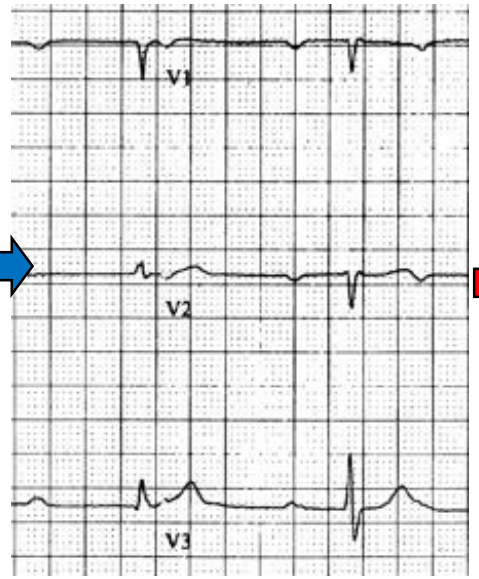
ECG diagnosis:

Right precordial leads patterns

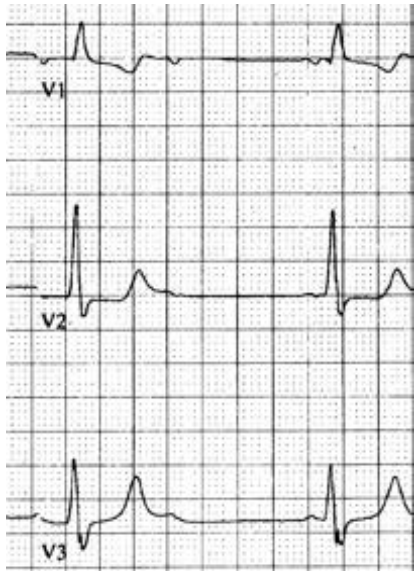
ECG-1 June 25, 2013



ECG-2 June 28, 2013

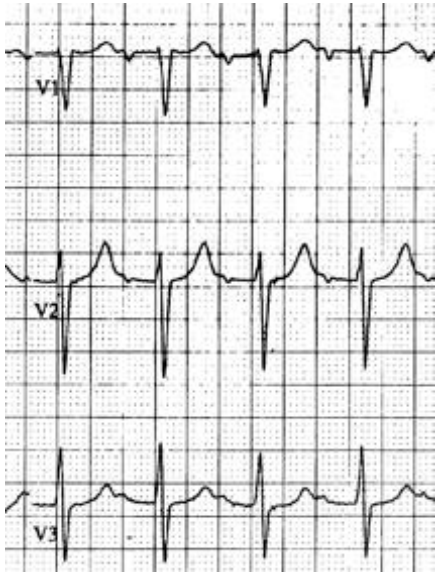


ECG-3 June 29, 2013

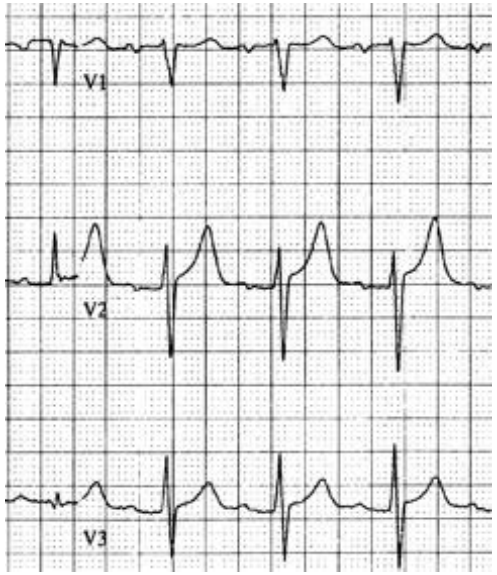


Transient Prominent Anterior QRS Forces (PAF)

ECG-4 July 02, 2013



ECG-5 July 03, 2013



Colleagues opinions

ECG June 25: Sinus, left atrial enlargement. Recent inferolateral STEMI (Q waves, mild ST elevation with terminal T wave inversion in III and aVF, tall R waves in V2-V3 with mild ST depression and positive T waves (reciprocal change of ST elevation with negative T waves)

June 28: Complete AV block. Junctional rhythm. ST elevation inferior leads resolved. Q waves only in lead III. ST depression anterior leads resolved. Tall R waves disappeared. Might be that leads v1-V2 were placed too high.

June 29: complete AV block with wide QRS rhythm with RBBB configuration. Is it AIVR? Is it new RBBB in a setting of post-STEMI?

July 02: Sinus with first degree AV block. Q waves with t wave inversion in III and aVF. No ST depression anterior leads, T waves are tall (reciprocal change of inverted T waves)

Serial changes of inferolateral STEMI complicated by complete AV block and possible AIVR

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Dear Potro and Raimundo – thank you for this challenging case with dynamic ECG changes.
My analysis:
ECG 1: Bordeline ST elevations in III and aVF, ST depressions in V2-V5 without ST depression in V1 – this is probably a proximal acute occlusion of the RCA with involvement of the right ventricle. P terminal force (diastolic dysfunction with elevated LV filling pressure).
ECG 2: III degree AV block. T inversions in III, aVF, V1-V2 are post-ischemic "finger-print" changes as a sign of a problem in the RCA + right ventricle in the acute phase. Electrode placement in V1-V2 is now higher on the chest (negative P in V1-V2).
ECG 3: III degree AV block and slow idioventricular rhythm causing high R waves with RBBB-type QRS in the precordial leads.
ECG 4: Sinus tachycardia, I degree AV block. P terminal force.
ECG 5: Pericardial post-operative inflammatory reaction with wide-spread ST elevations and PR depressions (PR elevation in aVR).

But I may be wrong also this time!

Thank you

Kjell **Nikus**

Tampere

Finland

Dear Andres and Raimundo,
Thank you for sharing this interesting series of ECG's. My thoughts are as follows:
ECG#1 (6/25): Evolving inferior wall myocardial infarction; the prominent R wave in V2 suggests early transition and not really prominent anterior forces. There is probable left atrial enlargement.6/29).
ECG#2 (6/28): Incomplete AV dissociation due to high grade 2nd degree AV block (the 5th QRS comes slightly early and is a sinus capture); the other beats are junctional escapes (45 bpm).
ECG#3 (6/29): Now it becomes more interesting. High grade 2nd degree AV block continues. The sinus rate is ~70 bpm; the first 4 QRS beats are 3:2 second degree AV block (Wenckebach or Type 1); the last 4 wide QRS beats with PAF and AV dissociation are left ventricular escape beats (~30 bpm). Beats 1 and 3 have slightly different morphology than the sinus (beats 2 and 4) and are fusion beats as they look somewhat in-between the morphology of the ventricular escapes and the sinus beats.
ECG #4 and #5: show 1st degree AV block with continued evolution of the inferior myocardial infarction.

As always I look forward to the interpretation of our colleagues and you final excellent summary.
Regards,
Frank

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Spanish Querido Andrés,
ECG 3
En I, II, III se inicia un Wenckenbach y en V1 los dos complejos QRS son de escape de la unión con aberrancia por la rama derecha y Ps no conducidas (la 2º porque se le adelanta el escape).
Esta es mi opinión. Gracias por el bello caso.
Antonio
English
Dear Andrés
ECG3: In I, II, III a Wenckenbach starts and in V1 the two QRS complexes are escape beats from the AV junction with aberrancy by the right bundle branch and non-conduced P waves (the 2nd beat because the escape go on before).
This is my opinion.
Thank you for the beautiful case.
Antonio

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I will only answer the question asked by our friends about the interpretation of ECG 3 (29/6/2013).
.This tracing begins with Wenckebach periods and narrow QRS complexes to suddenly switch to high degree or complete AV block with WIDE QRS complexes of RBBB type and prominent R waves in V1-V3. These wide QRS complexes are escape ventricular beats and not conducted beats so that we should be very careful before doing speculation on their possible mechanism.
Any way, these prominent QRS complexes in V1-V3 suggest a ventricular ectopic rhythm originating from the LV-posterobasal wall, consistent with the infarcted area.
Another theoretical explanation would be an AV junctional rhythm associated with bradycardia-dependent (phase 4) RBBB but the slow rate highly suggests a VENTRICULAR rather than an AV JUNCTIONAL ESCAPE.
Finally, despite the presence of severe AV conduction disturbances (m/p AV nodal located), I do not think the patient had significant RV infarction.(no obvious ECG signs during both conducted beats or even during ventricular escape).
More interestingly will be to understand the possible reason of the PROMINENT R in V1-V3 on the first ECG trace as compared to the last 2 late ECG tracings. But this question was not asked by our dear South American colleagues and I will not elaborate on it !!!
It is always a pleasure to learn from you my dear friend.

Shalom from Jerusalem, Israel @@@

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http://www.ncbi.nlm.nih.gov/pubmed?term=belhassen%20b

ECG1 Acute inferior MI due to RCA occlusion
ECG2 High grade but complete AV block
ECG 3 AV Wenkebach conduction with accelerated idiofascular rhythm from post fascicle (PR too close to allow for conduction of wide complexes).
ECG 4/5 return of 1:1conduction with evolving IMI

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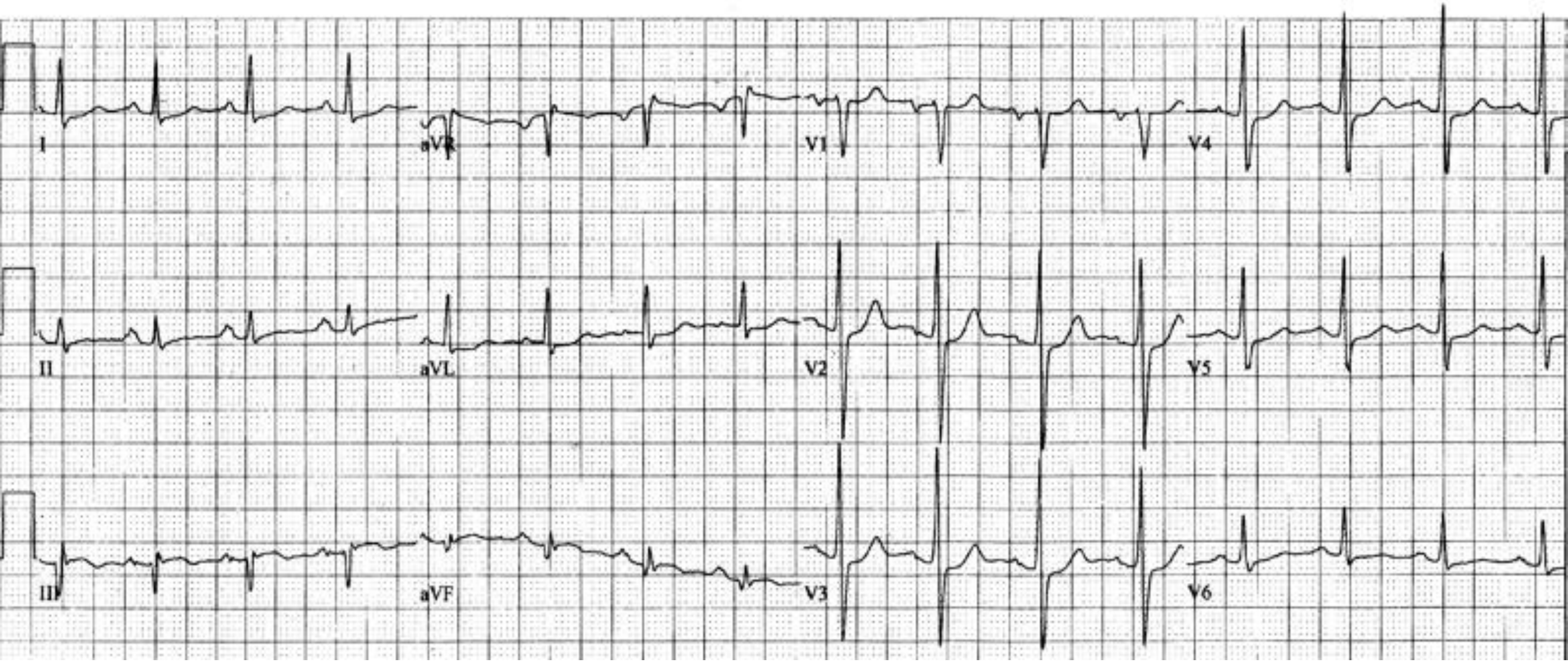
Final comments
by Andrés Ricardo Pérez-Riera & Raimundo Barbosa-Barros

ECG-1 June 25, 2013

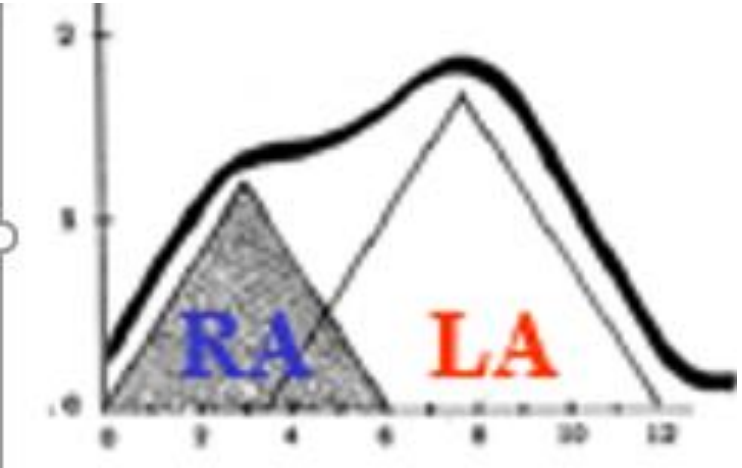


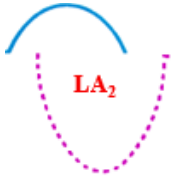
ECG diagnosis: Sinus rhythm, HR 96bpm, P axis +58°, P duration: 121ms, P voltage 2mm, final deep and slow component of P wave in V1: left atrial enlargement (LAE), PR interval duration: 180ms; Q waves in III and aVF and mild ST segment elevation followed by terminal negative T, relative tall R waves in V2-V3, mild ST segment depression in anterolateral leads I, aVL, V2-6 followed by positive T waves in as expression of mirror image. In patients with MI of the inferior and/or lateral wall, a prominent T wave in V2 with respect to V6 reflects greater infarct extent in the lateral wall (**Roval 2015**): Acute inferolateral STEMI. See ludic explanation in the next slides……..

ECG-1 June 25, 2013

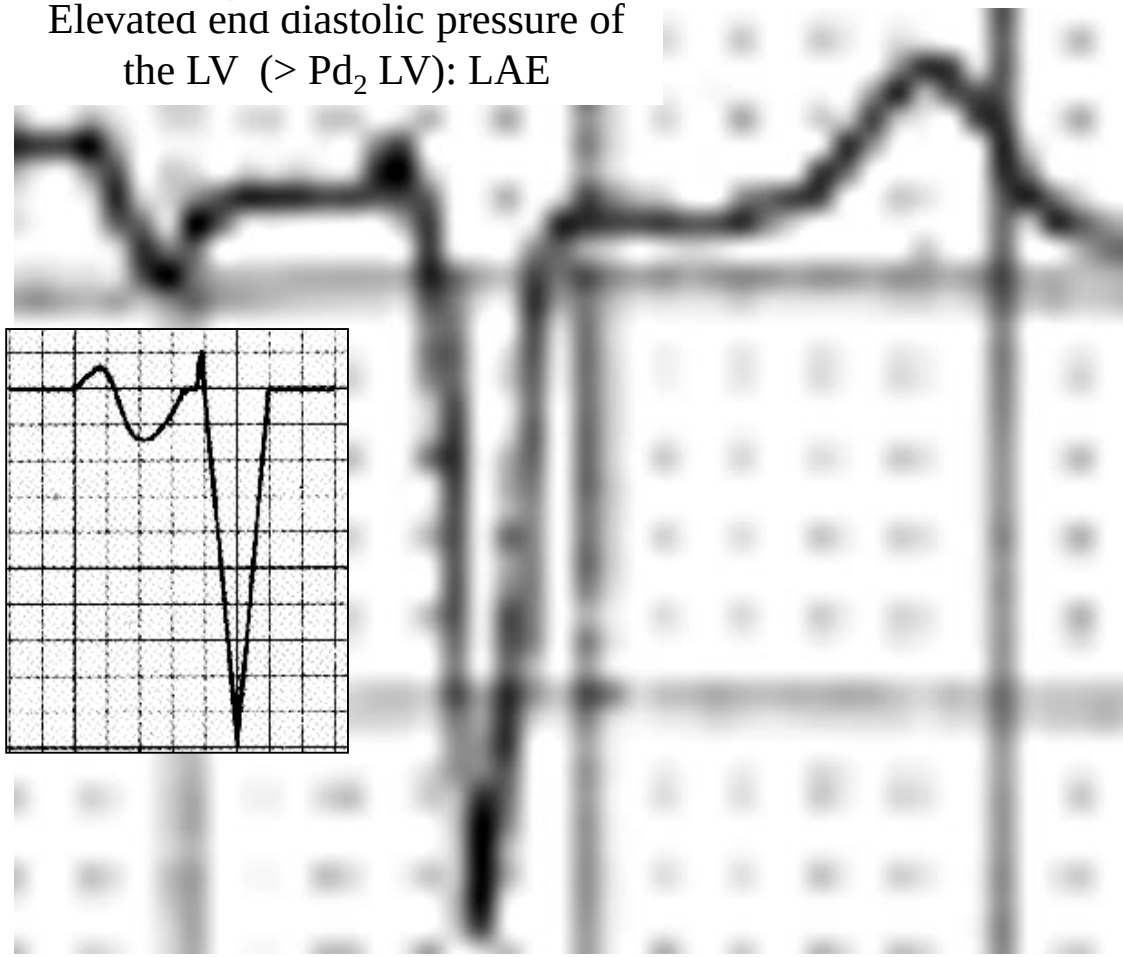
P duration: 121ms: LAE

AGE RANGE	NORMAL MAXIMAL VALUE OF P WAVE DURATION
0 to 12 months:	80ms (two little squares).
1 to 12 years:	90 ms.
> 12 years:	100 ms. (2.5 little squares).
Seniors	110ms



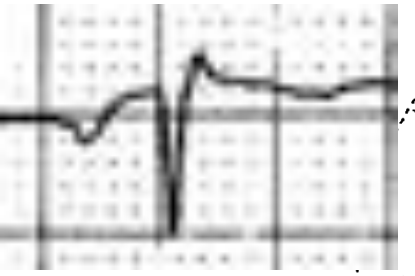


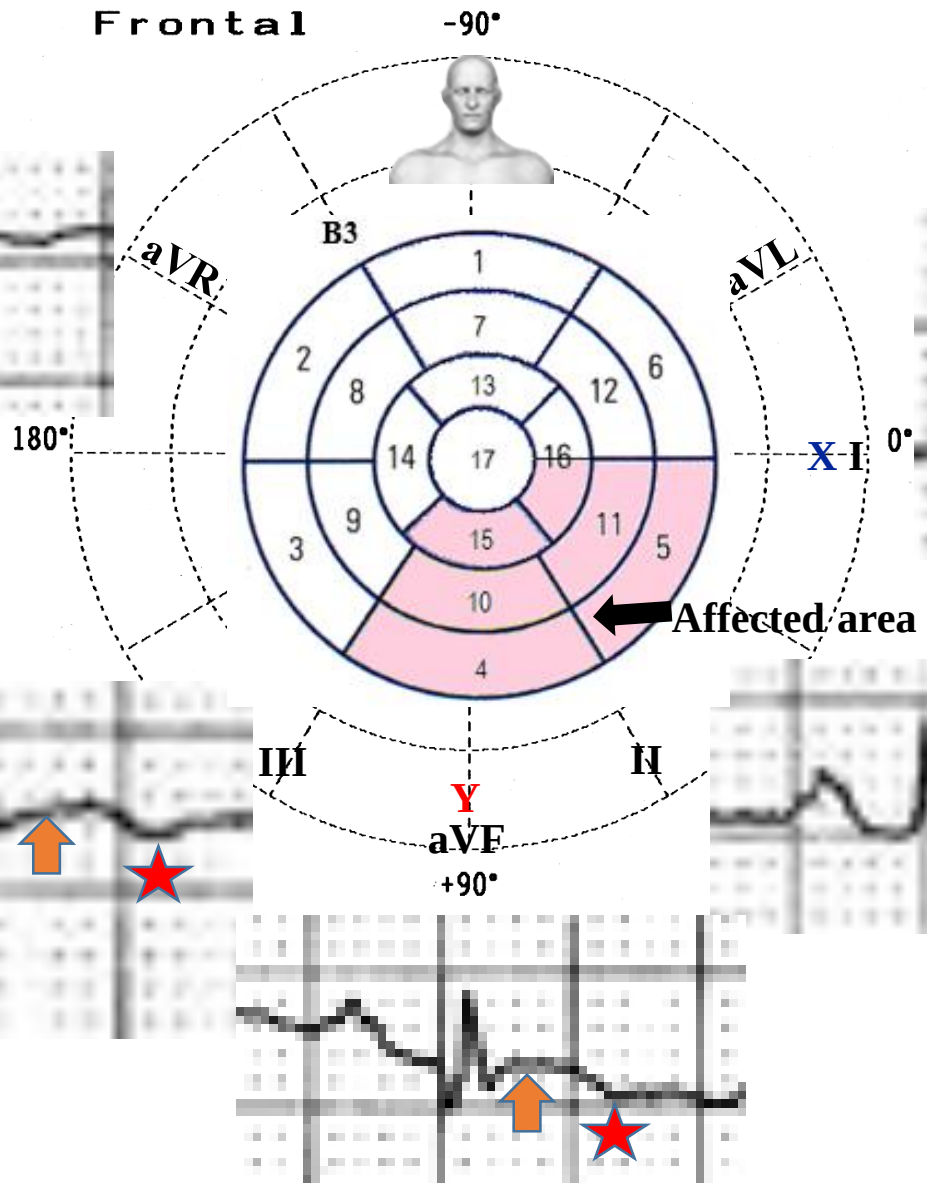
Elevated end diastolic pressure of the LV (> Pd_2 LV): LAE

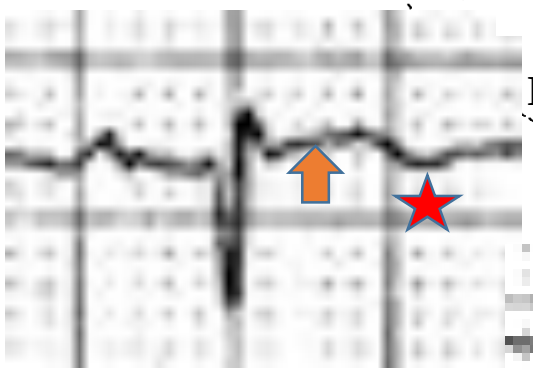


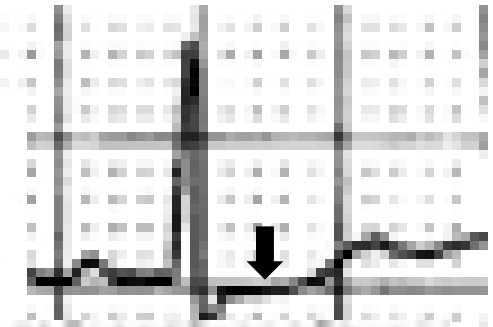
Abnormal negative final components of the P wave in V1. When the value calculated using the width in seconds and the height in millimetres of the negative mode exceeds 40 mm + ms, it is considered abnormal. $LA_{2/}$: final deep and slow component: LAE ≥ the area of one small square; the final minus portion indicates left atrial enlargement, abnormality or advanced interatrial block.

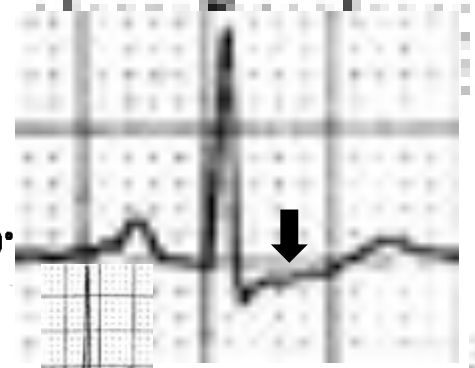
ECG-1 June 25, 2013

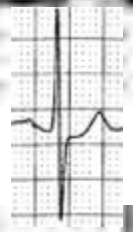


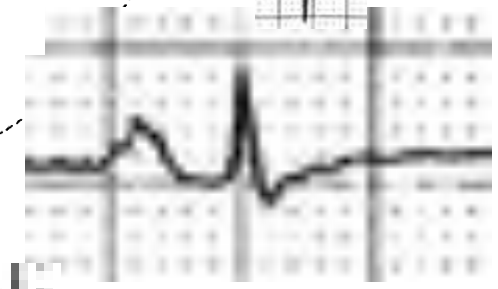


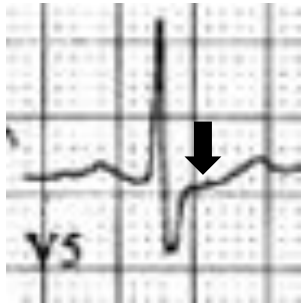


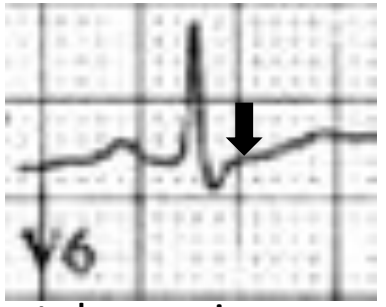






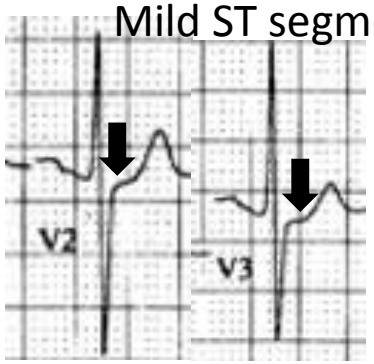






Mild ST segment depression in anteroateral leads **I, aVL, V2-6** followed by positive T waves in as expression of mirror image or reciprocal changes. ST segment depression in leads I and aVL indicates RCA-associated inferior wall AMI with a sensitivity of 70% and 100%, and a specificity of 63% and 38%, respectively, whereas the lack of ST segment depression in these leads indicates proximal LCX with a sensitivity of 71% and 86%, and a specificity of 65% and 100%, respectively (**Hasdai 1995**).

Mild ST segment depression

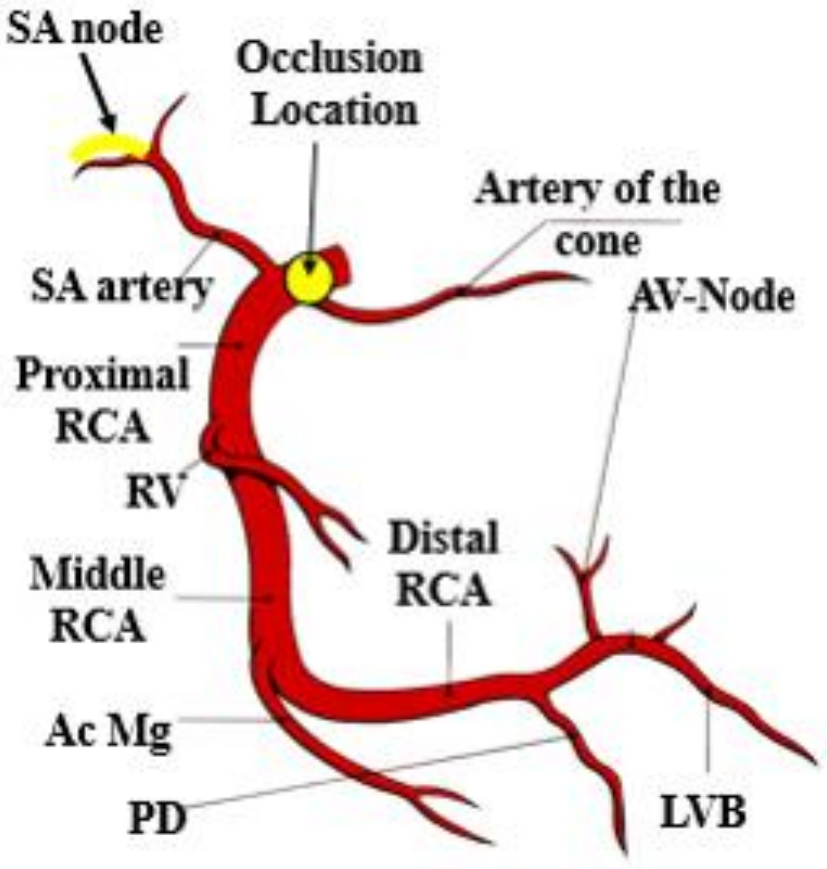


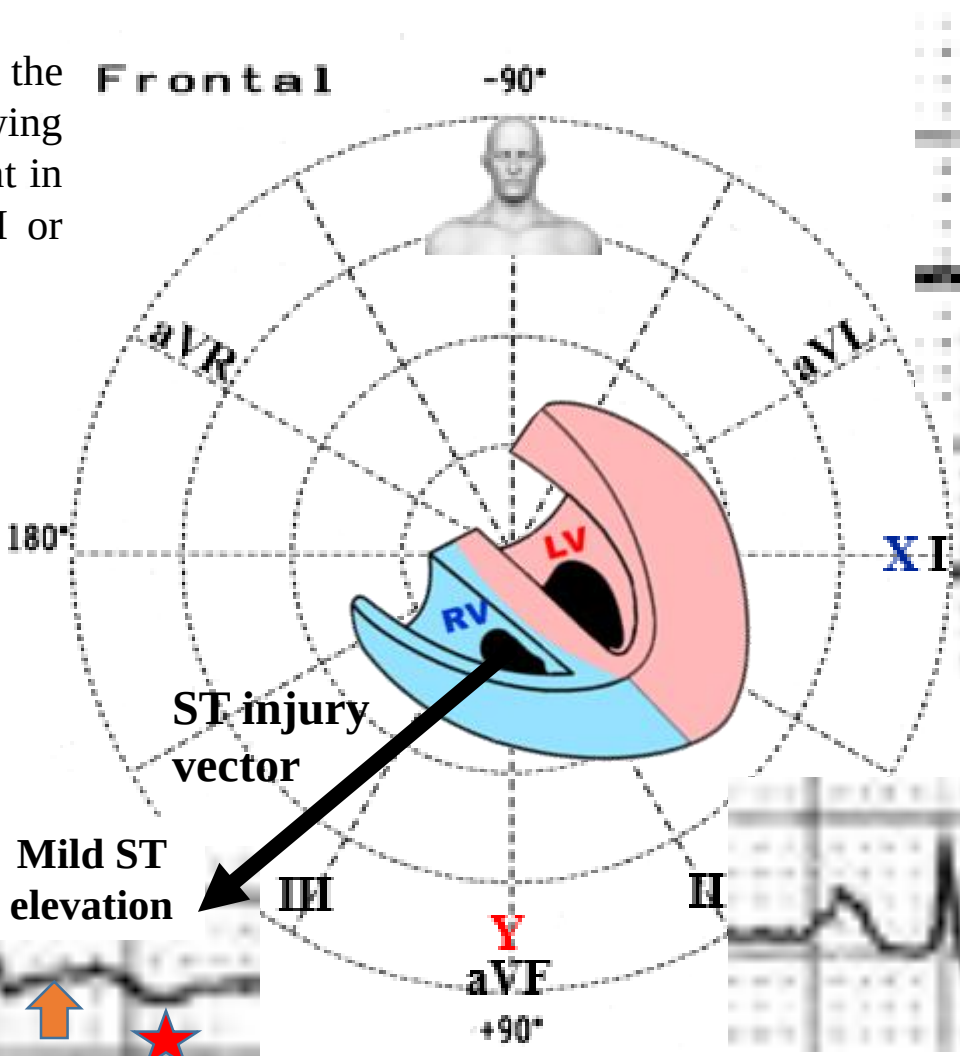
B3 – Q in III, aVF and mild ST segment elevation ⬆ followed by terminal negative T wave i in III and aVF ★

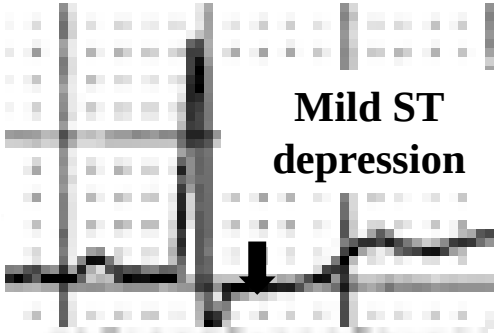
Injury vector pointed to down and rightward: proximal RCA occlusion

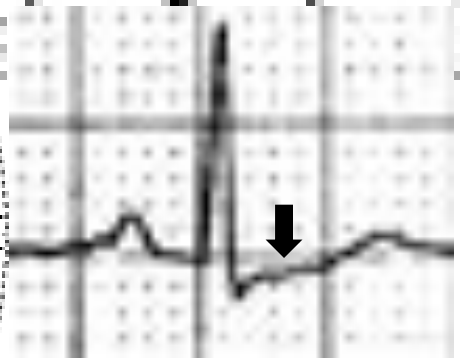
ECG-1 June 25, 2013

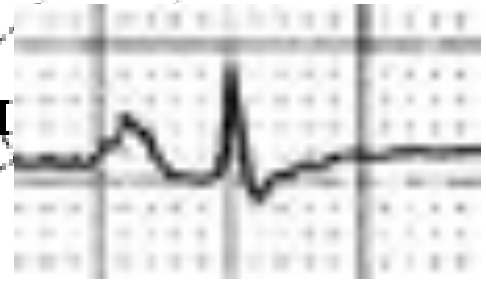
The conventional ECG algorithm for identification of the RCA as the infarct-related artery (IRA) in inferior STEMI includes the following criteria: (a) ST-segment elevation (STE) in lead III exceeding that in lead II, and (b) ST-segment depression (STD)≥1 mm in lead I or aVL.

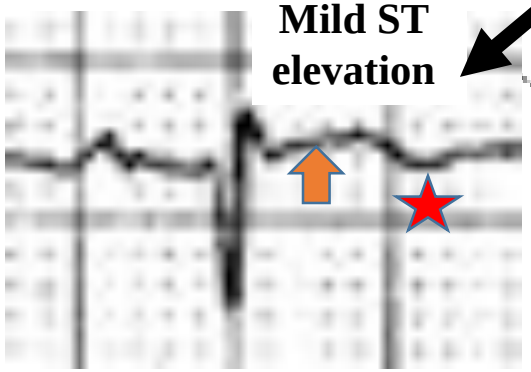










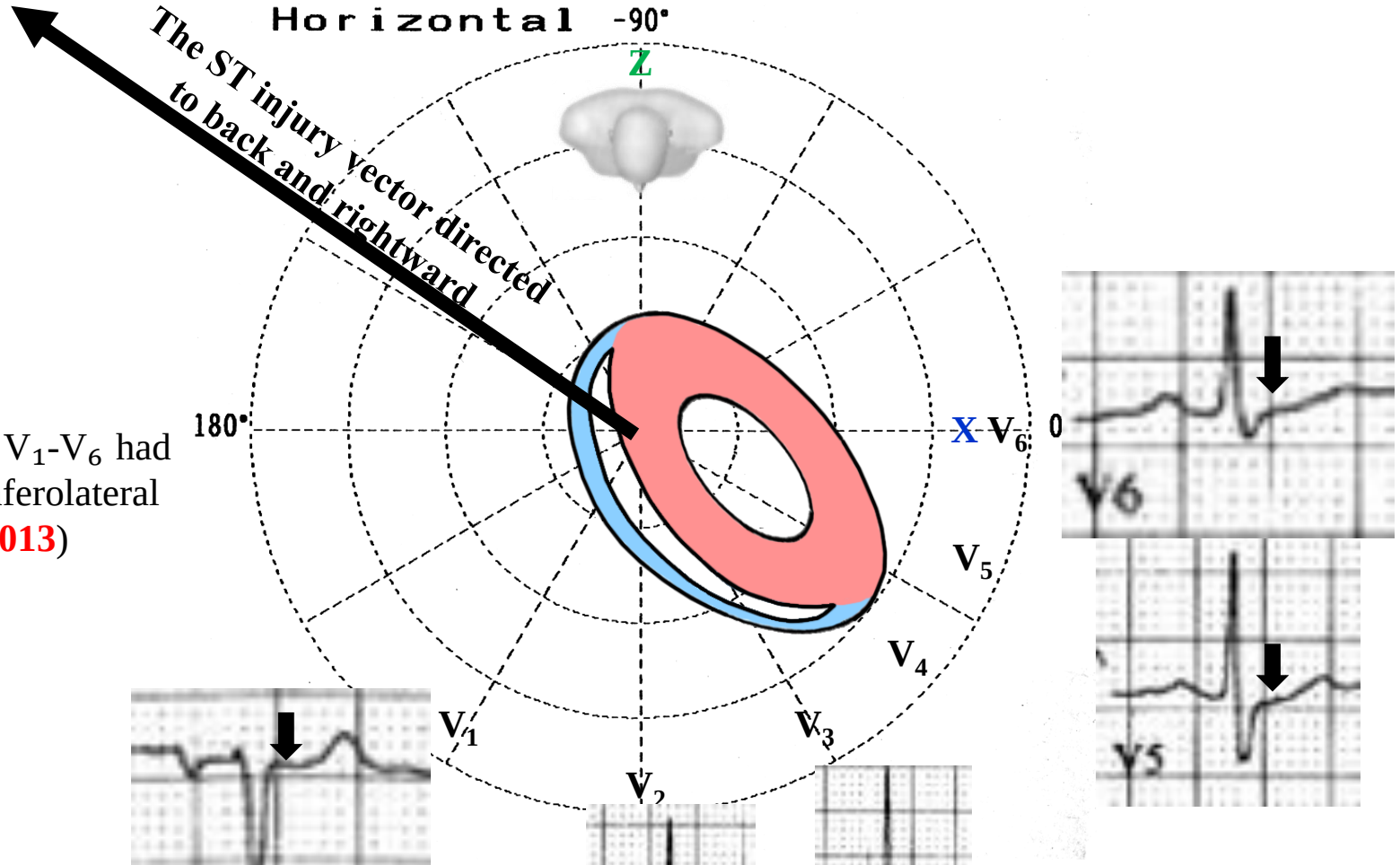


The vector of ST-segment deviations(ST injury vector) on the 12-lead ECG during acute transmural ischaemia is an indicator of the site of coronary artery occlusion (**Wagner 2009**).

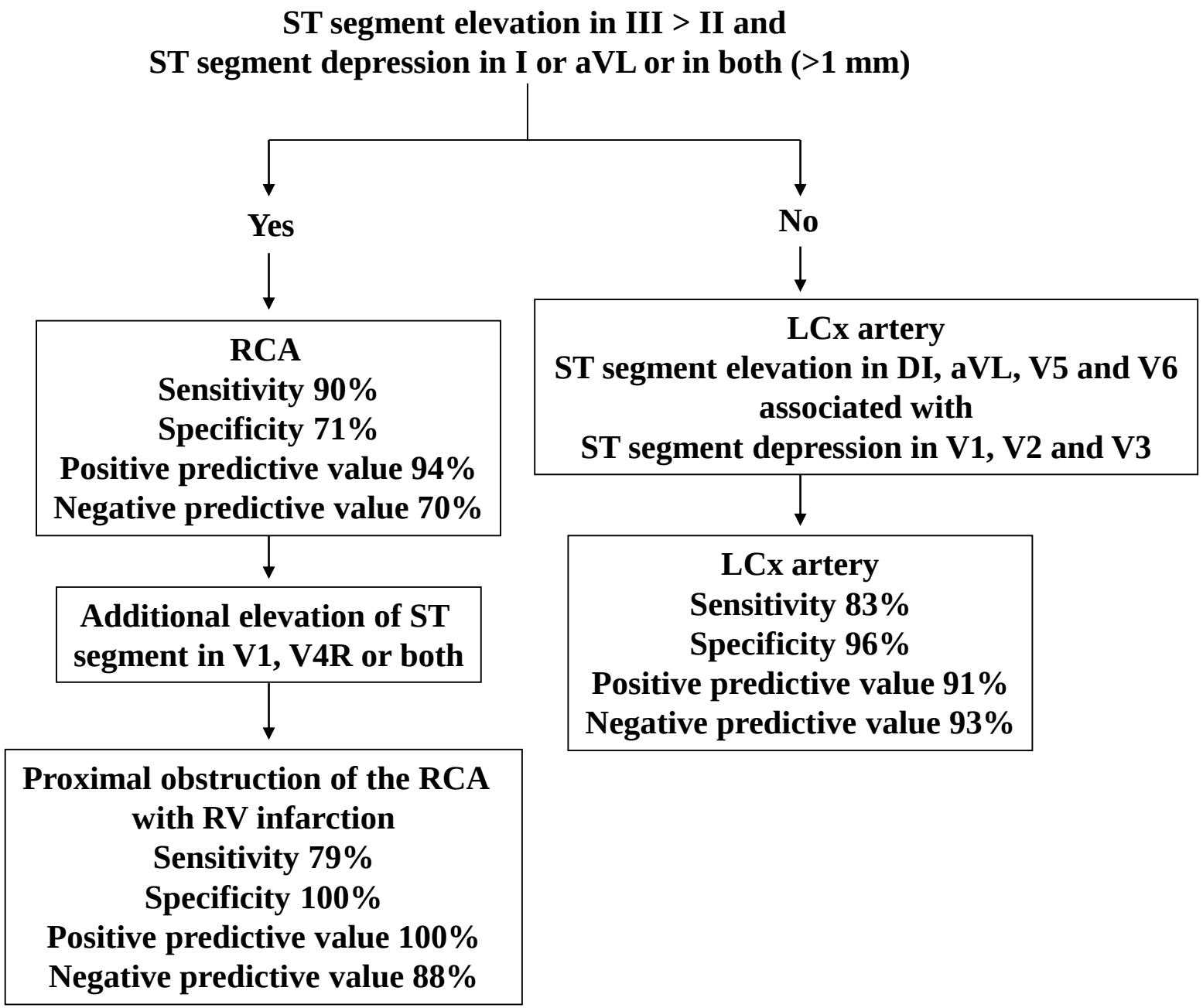
In HP proximal RCA occlusion has the ST injury vector directed to back and rightward: ST segment depression across precordial leads

ECG-1 June 25, 2013

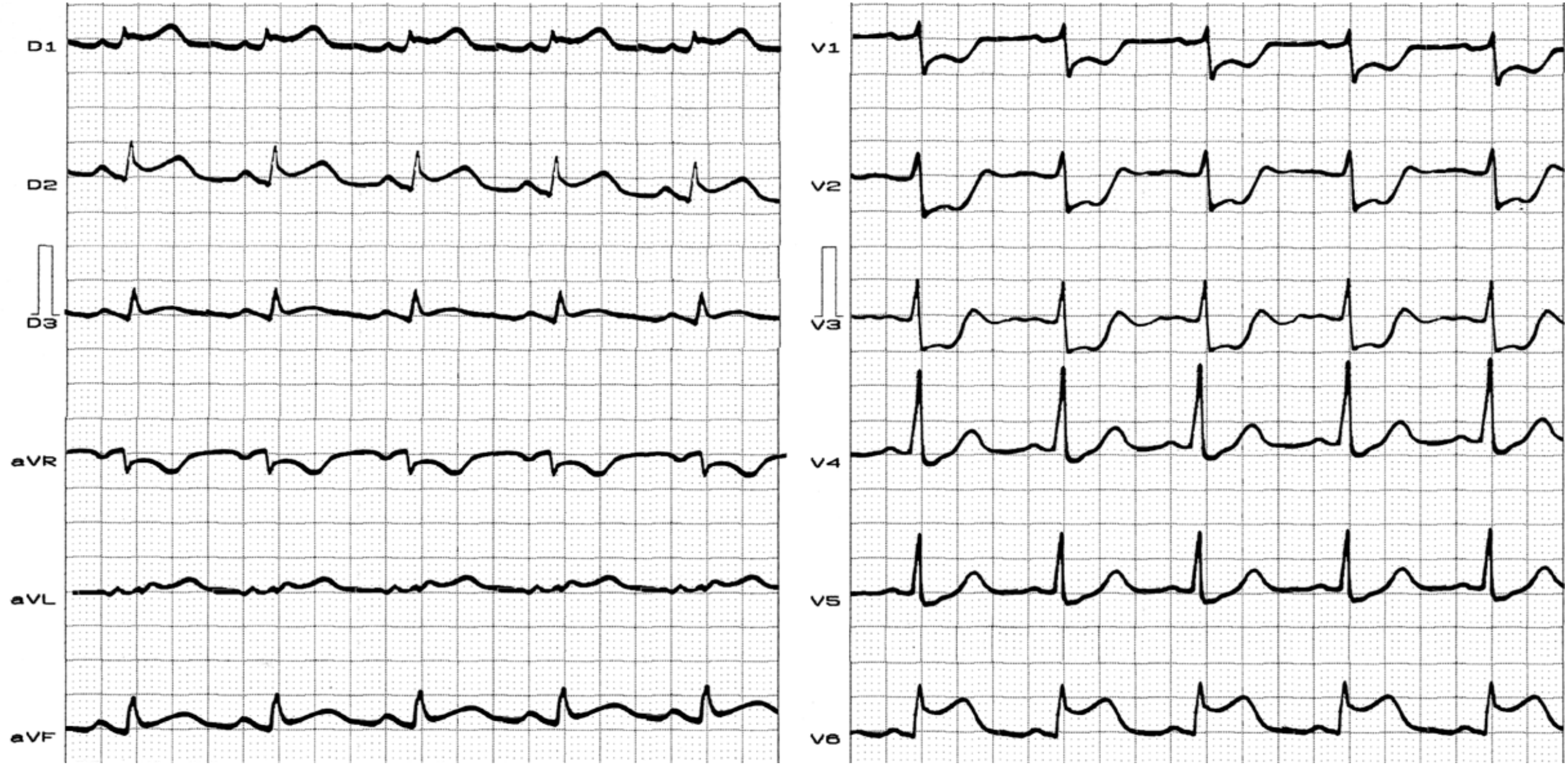
A large number of patients with STD in V_1-V_6 had angiographic evidence compatible with inferolateral (old posterior) STEMI equivalent (**Wei 2013**)



Algorithm to identify the artery involved with inferior acute myocardial infarction by ECG (Zimetbaum 2003)

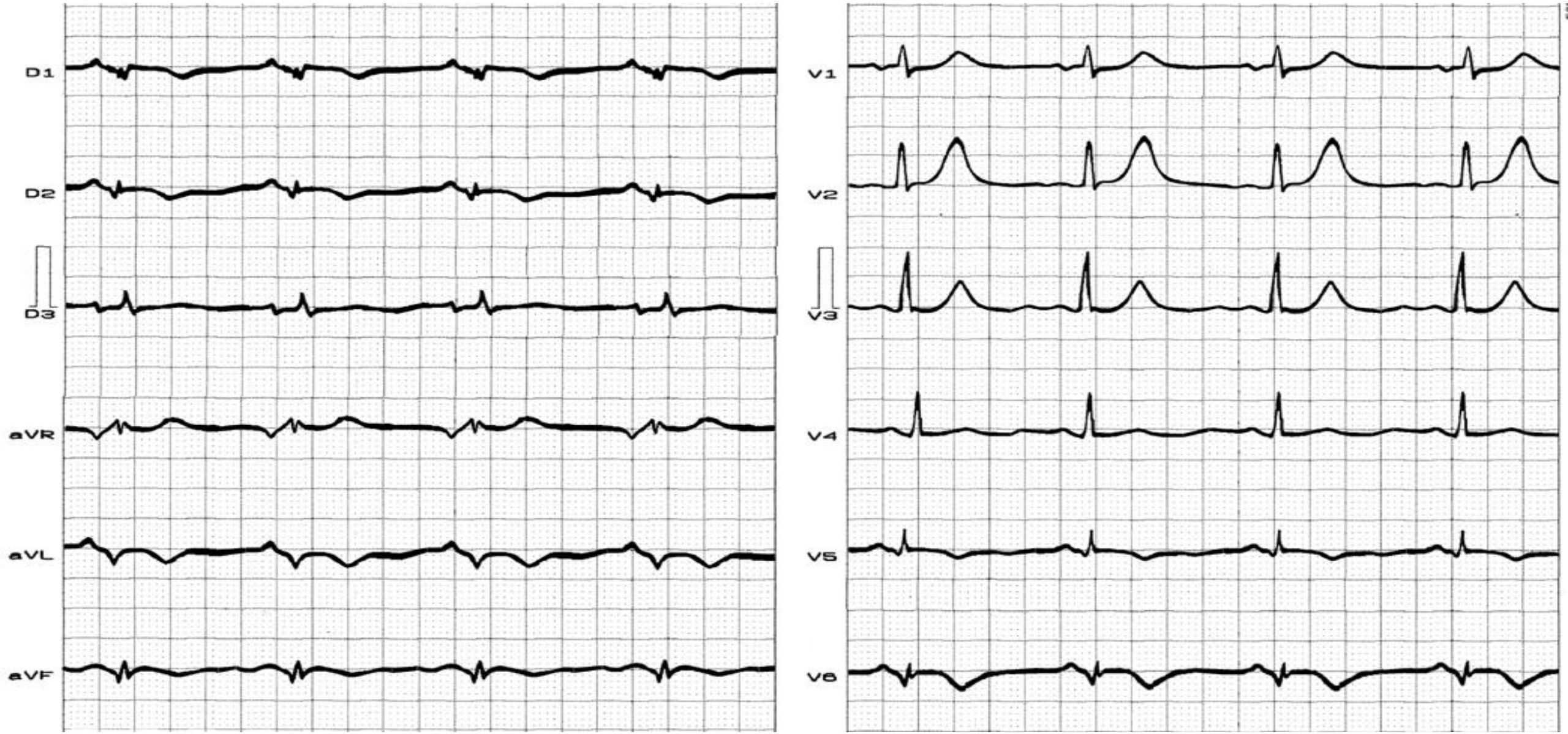


Typical example of hyperacute phase inferolateral STSE myocardial infarction



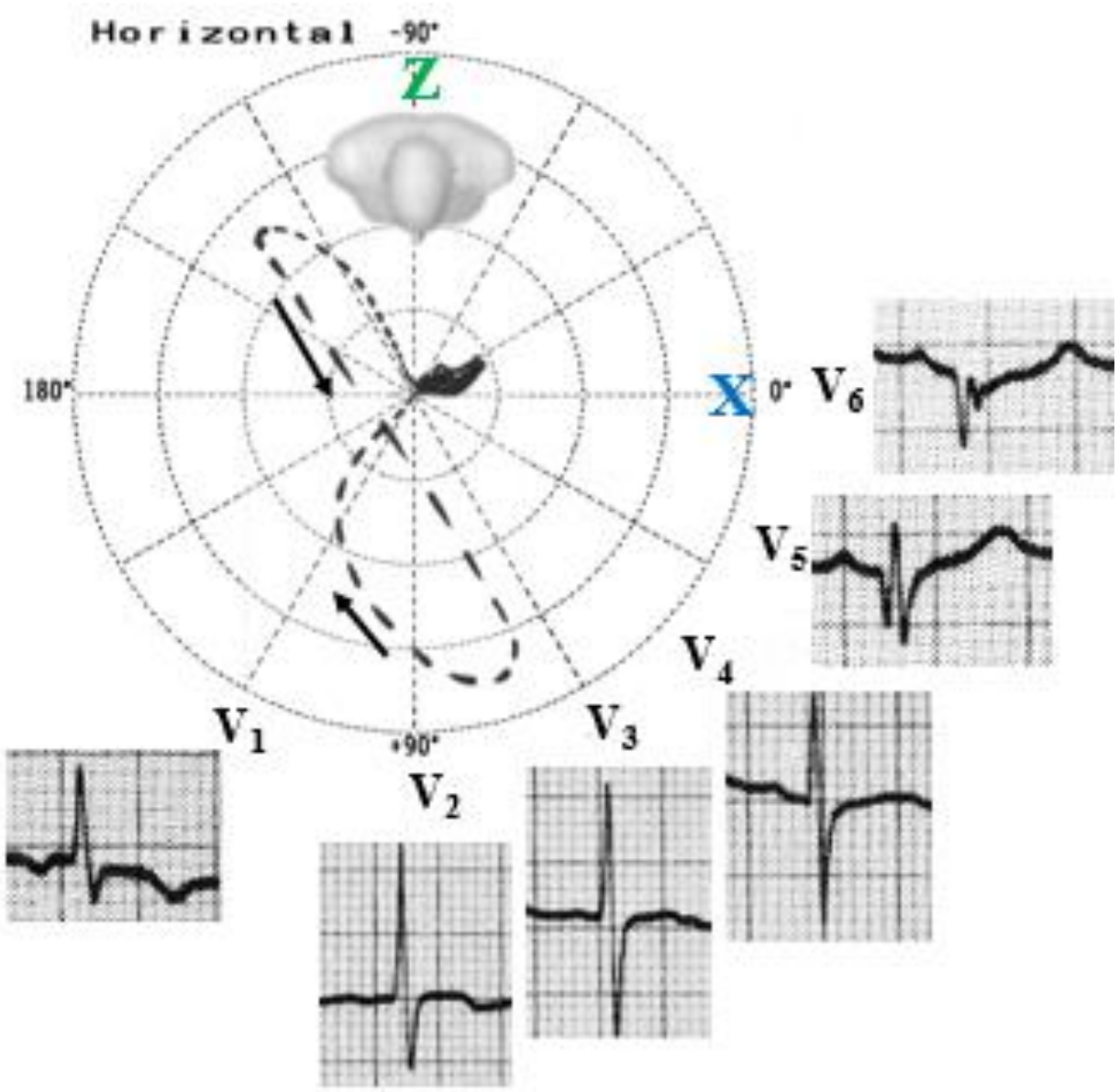
ECG diagnosis: Significant ST segment elevation is observed in the inferolateral wall leads and significant ST segment depression from V1 to V3. Infarction in the hyperacute phase with ST segment elevation in the inferolateral region (STEMI-ACS) required an immediate intervention.

The same patient in late phase of inferolateral myocardial infarction



This ECG is from the same patient, made 20 days after the event. Q waves appeared in the inferior wall, prominent R waves from V1 to V3 with positive and symmetrical T waves, low voltage of R in V5-V6 and QS in I and aVL. These changes represent inferolateral myocardial infarction in the late phase.

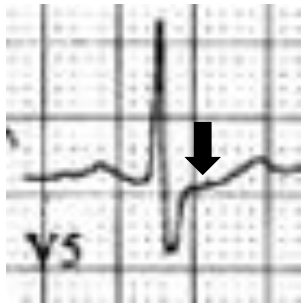
Lateral Myocardial Infarction ECG/VCG correlation on HP



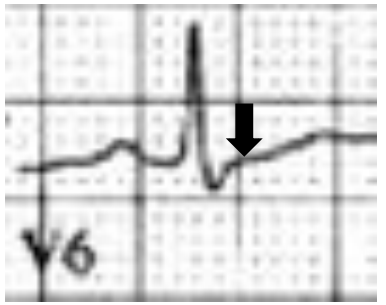
R > S in V1-V2

The present case lateral precordial leads

ECG-1 June 25, 2013
Acute phase



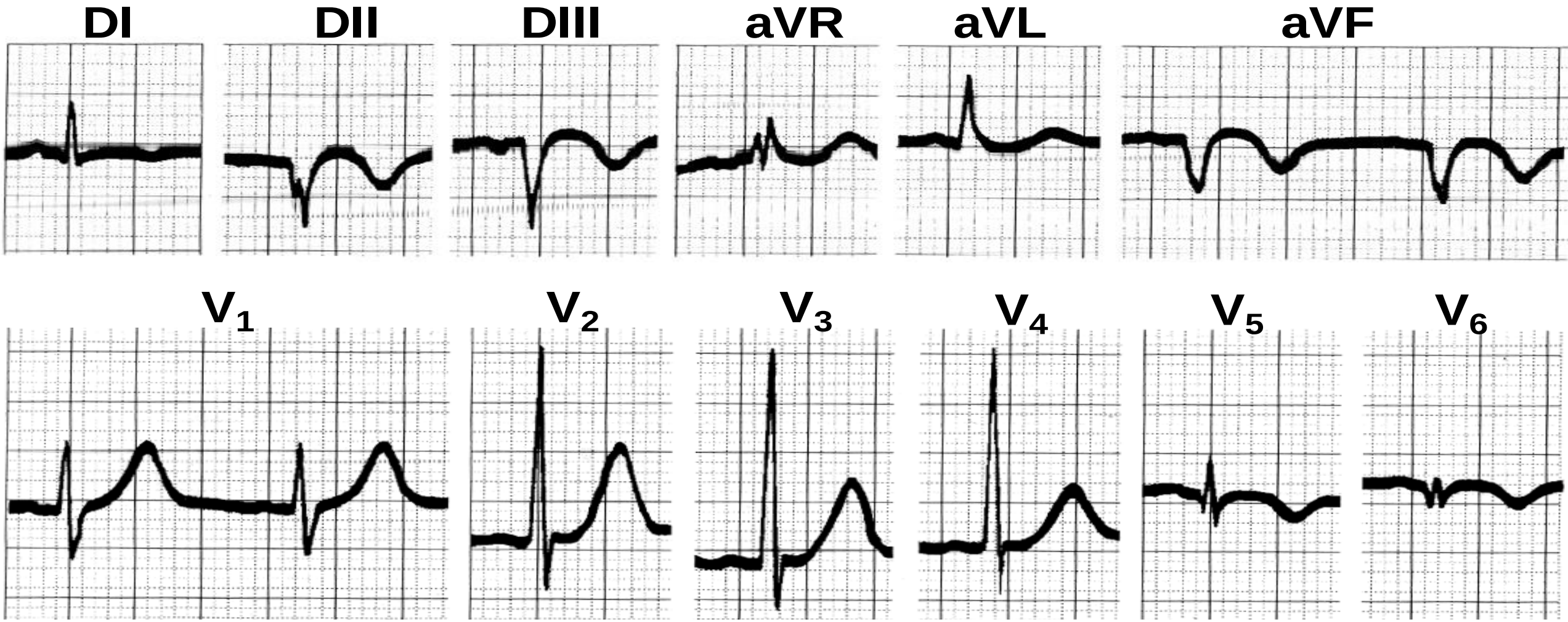
R wave 7 mm



ECG-5 June 25, 2013

R wave 4 mm
After bypass graft on July 2nd

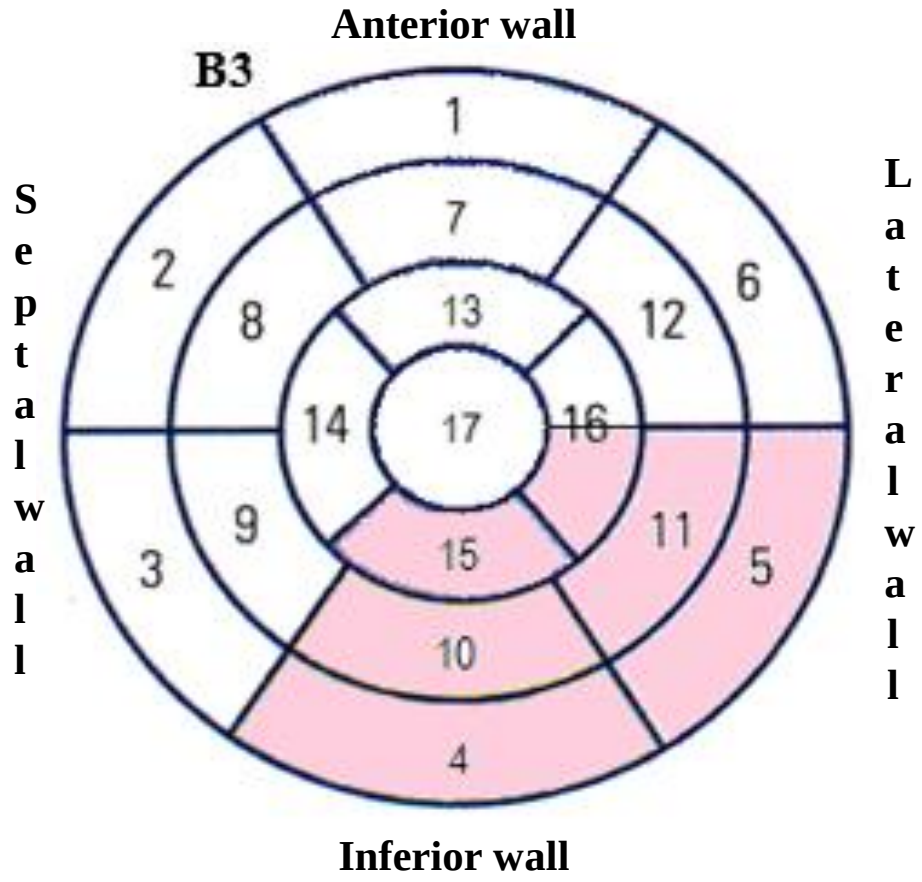
A case of inferolateral wall late infarction of the B3 type



ECG diagnosis: Inferolateral electrically inactive area corresponding to B3 infarction using the new ECG/cardiac NMR correlation classification: QS in II, III and aVF with prominent R in V1-V2 and low voltage r/R in V5 and V6. Inferior infarction is extensive, affecting the entire inferior wall, which explains the absence of final r or R wave in II, III and aVF. Involvement of the lateral wall represented by small r waves in leads V5 and V6 indicates that the necrosis also extends into this wall.

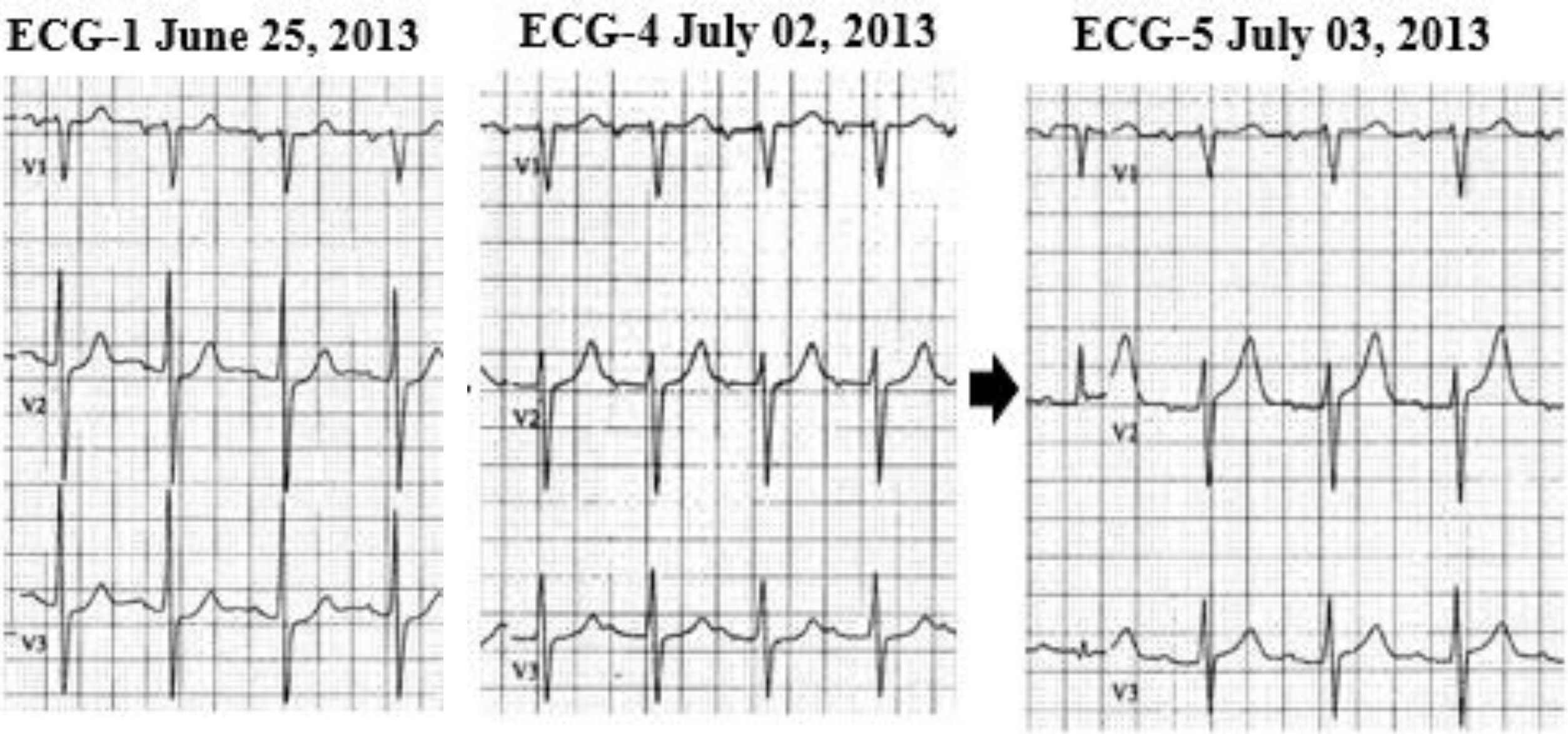
Prominent R waves in V1-V2 are no longer thought to be caused by true posterior or dorsal infarction but to lateral left ventricular wall infarction. The coronary angiogram in this case revealed total obstruction of the posterior descending branch of a dominant left circumflex artery.

Inferolateral myocardial infarction type B-3 (MRI) “bull’s eyes”



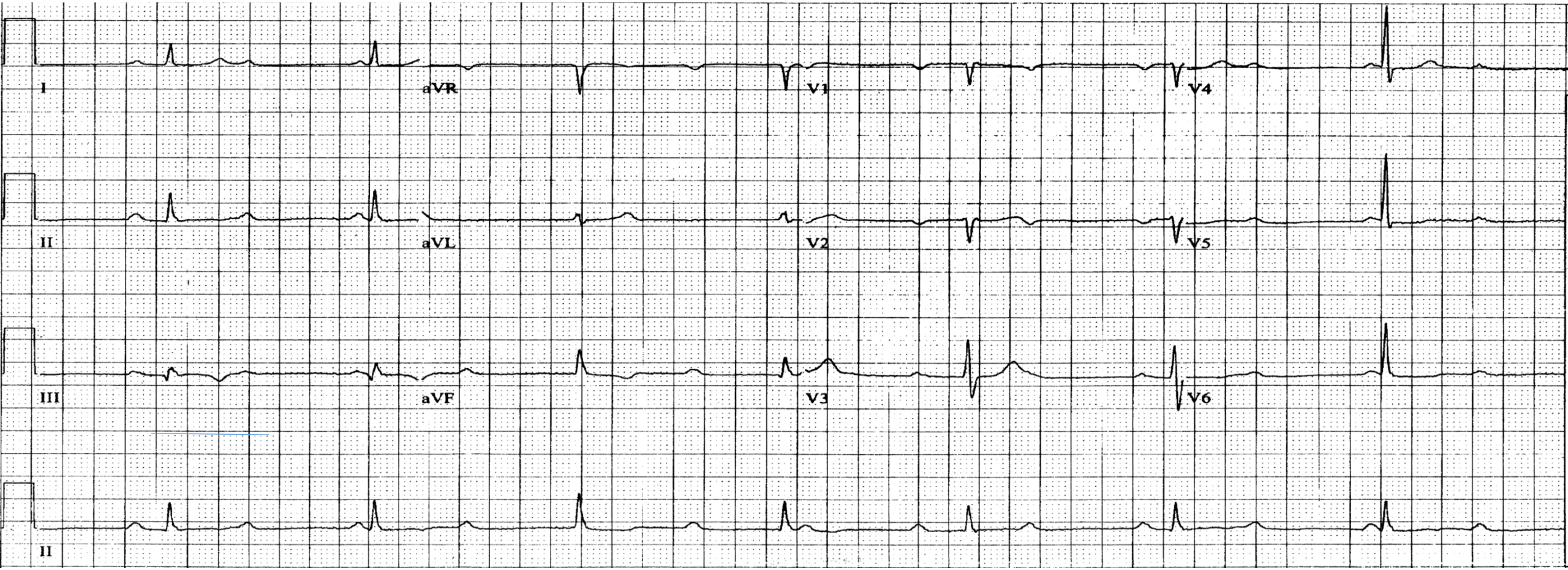
- **Type:** B-3
- **Most likely site of occlusion:** proximal RCA.
- **ECG pattern:** QS in II, III and aVF and Q in I, aVL, V5-V6.

1. **Segments affected by infarction in cardiac NMR:** 15, 10 and 4/5, 6, 12, 11 and 16 (**Bayés de Luna 2006a,b; 2007 a;b;c;d; 2015; Cino 2006; Ponds Ladó 2006 b; Goldwasser 2015**)



The R waves of V2-V3 on June 1 are higher than the R waves of V2 and V3 on June 3 and 4: The pattern is expression of lateral extension of Acute Myocardial Infarction.

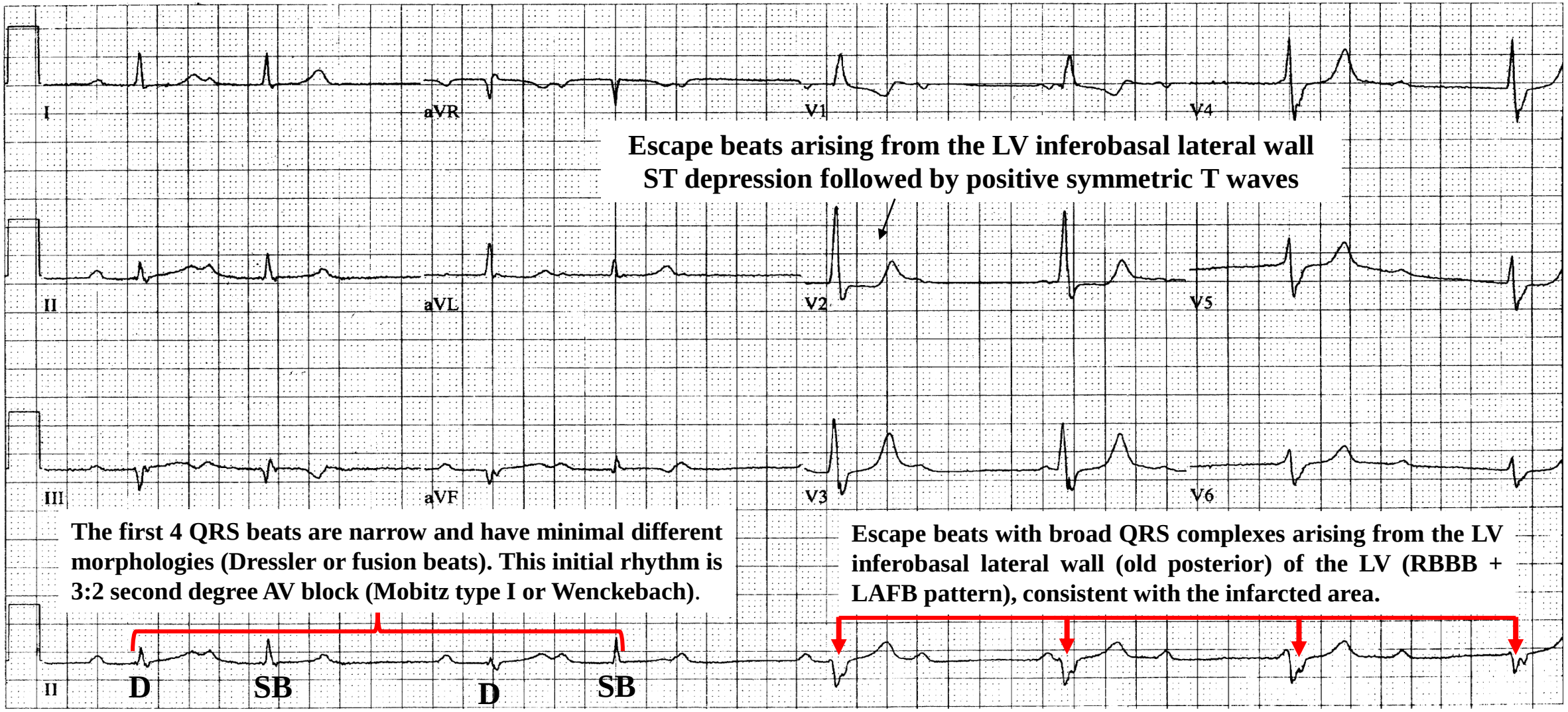
ECG-2 June 28, 2013



ECG diagnosis: Complete or third degree AV block, atrial HR with constant 83 bpm (without ventriculophasic sinus arrhythmia). It is a non-respiratory sinus arrhythmia frequently seen in complete AV block. When the arrhythmia is present, the P-P interval enclosing a QRS complex is shorter than a PP interval not enclosing a QRS. Ventricular command with HR of 40bpm, narrow QRS complexes. Note that the focus is located in AV node or in penetrating portion of His bundle (it penetrates the central fibrous body) (junctional rhythm).

Negative post ischemic T waves in III, aVF, V1-V2 (the negative T wave of ischemic origin may be seen in the clinical setting of post myocardial infarction as a consequence of reperfusion in case of aborted MI when the artery has opened spontaneously or after fibrinolysis, percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG) or coronary spasm (**Bayés de Luna 2014**)). Relatively tall T waves disappeared because V1-V2 were place too high.

ECG-3 June 29, 2013

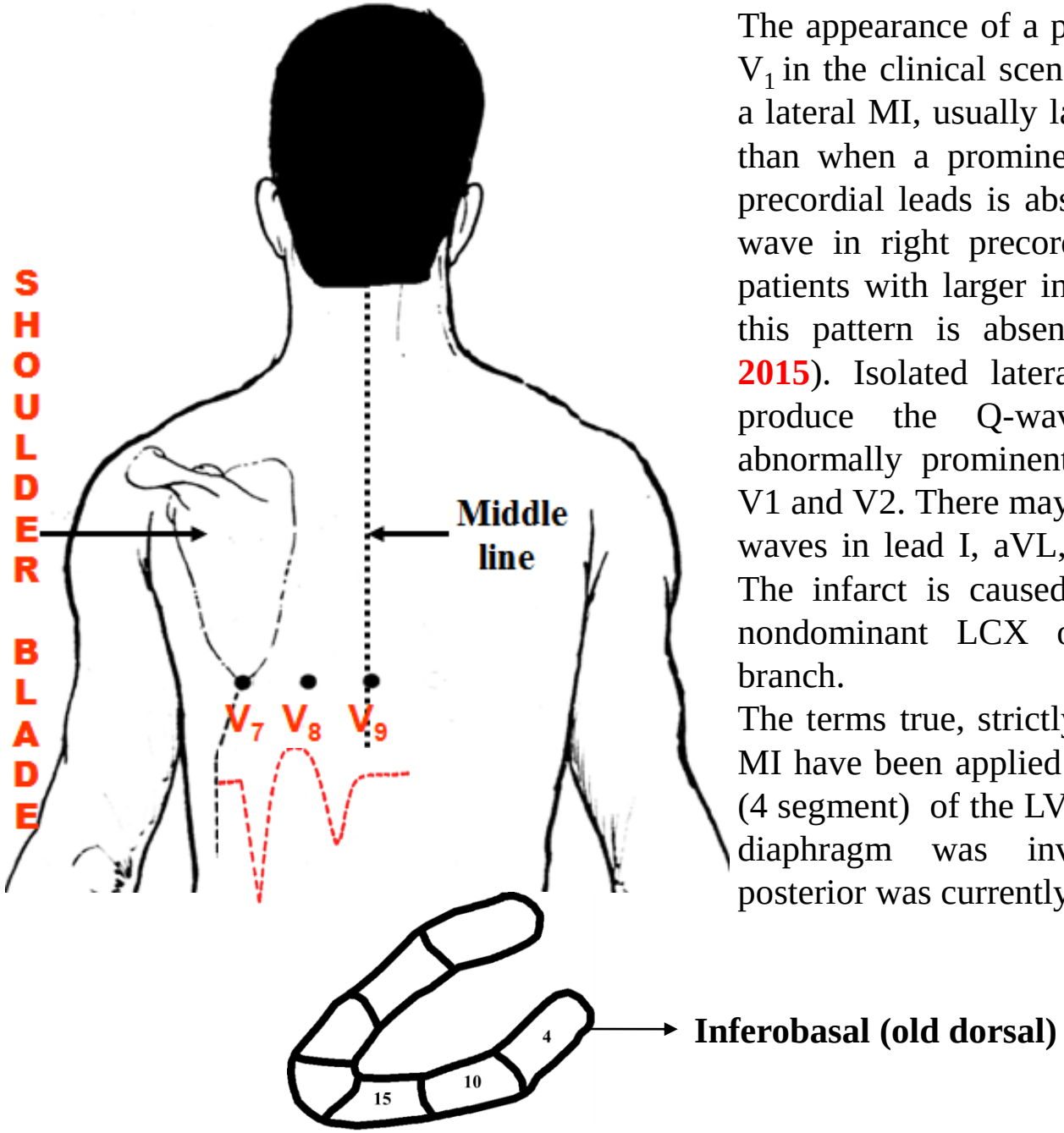


ECG diagnosis: High second degree AV block. Ventricular command with slow idioventricular rhythm and variable focus or conduction. QRS with RBBB pattern sometimes associated with QRS axis directed superiorly and to left; suggesting that the last fourth beats in long II trip are arising from the posterior wall of the LV (RBBB + LAFB pattern) or from Purkinje network of the posterior division or within the inferobasal left ventricular wall (**Rosembaum 1969**). The first fourth QRS beats show 3:2 second degree AV block Mobitz type I (Wenckebach).

SB: Sinus Beat; **D:** Dressler or fusion beats (these are hybrid beats with intermediate morphology between **SBs** and ventricular escapes).

For decades, a tall and broad R wave in right precordial leads has been considered the sign of a dorsal or strictly posterior MI. This conclusion has been accepted as paradigm/dogma/conviction.

Today we know that in the presence of normal variant with marked counterclockwise rotation of the heart around the longitudinal axis of the heart resulting in a shifting of the transition area (R=S) early, i.e. to the right of the precordial lead V_2 **(Yanagisawa 1981; Mori 1992; Paparella 1987),** misplaced precordial leads **(MacKenzie 2004; Mattu 2001),** RVH, complete RBBB, Left septal fascicular block **(Uchida 2006; Riera 2008 a,b c; Pérez-Riera 2011a;b, 2015 a,b; 2016;),** LSFB and degree of RBBB in association, WPW with posterior anomalous pathway, Duchenne's cardiomyopathy **(Secchi 1982; Yotsukura 1999),** endomyocardial fibrosis **(Tobias 1992),** dextroposition (example: left pneumonectomy) **(Pérez Riera 2011).**

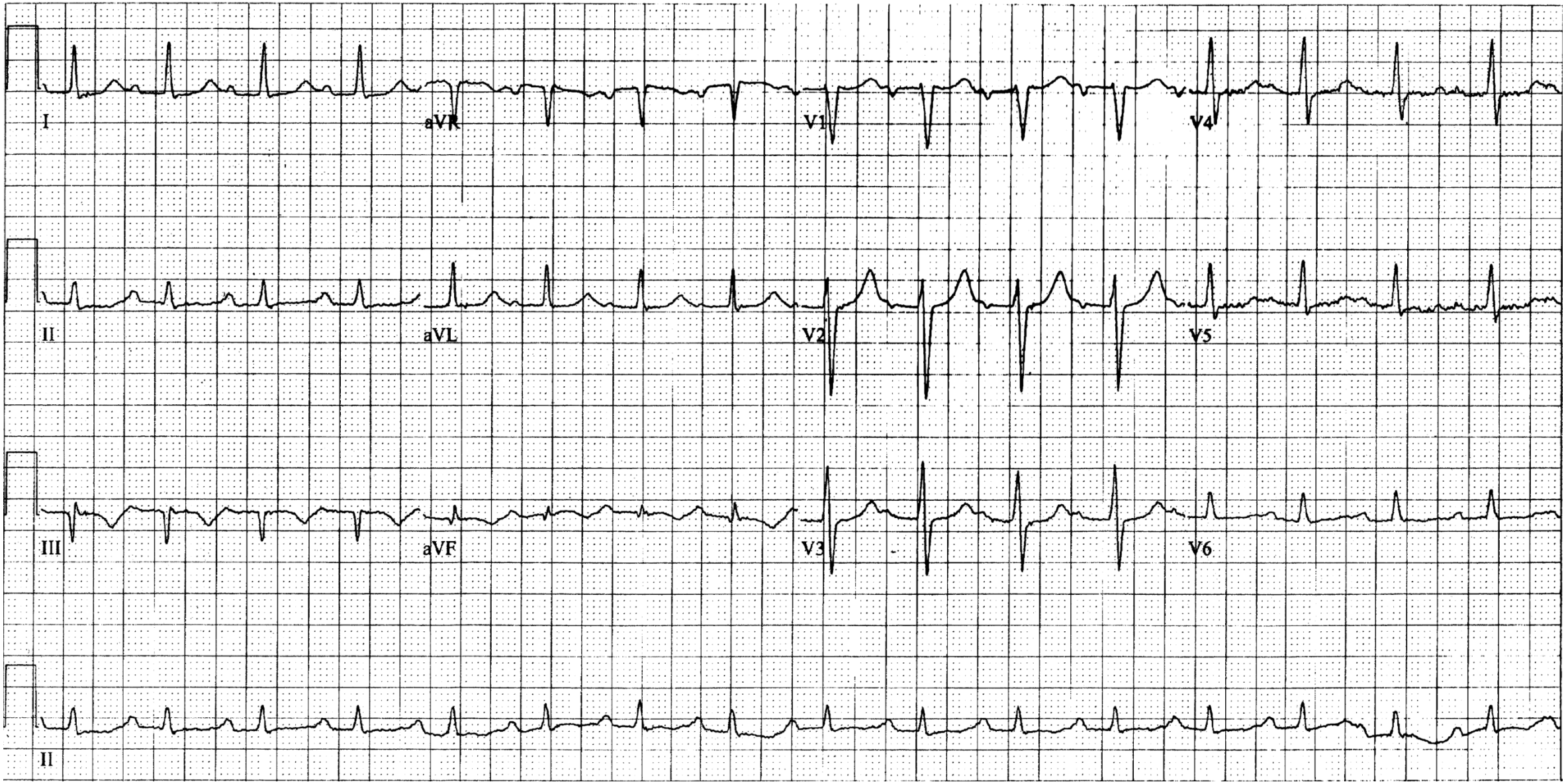


The appearance of a prominent R wave in V_1 in the clinical scenario of ACS reflects a lateral MI, usually larger and transmural than when a prominent R wave in right precordial leads is absent. A prominent R wave in right precordial leads identifies patients with larger infarctions than when this pattern is absent (**Bayés de Luna 2015**). Isolated lateral MI infarcts may produce the Q-wave equivalents of abnormally prominent R waves in leads V1 and V2. There may also be abnormal Q waves in lead I, aVL, and/or V5 and V6. The infarct is caused by occlusion of a nondominant LCX or of its marginal branch.

The terms true, strictly posterior or dorsal MI have been applied when the basal part (4 segment) of the LV wall that lies on the diaphragm was involved. The term posterior was currently abandoned.

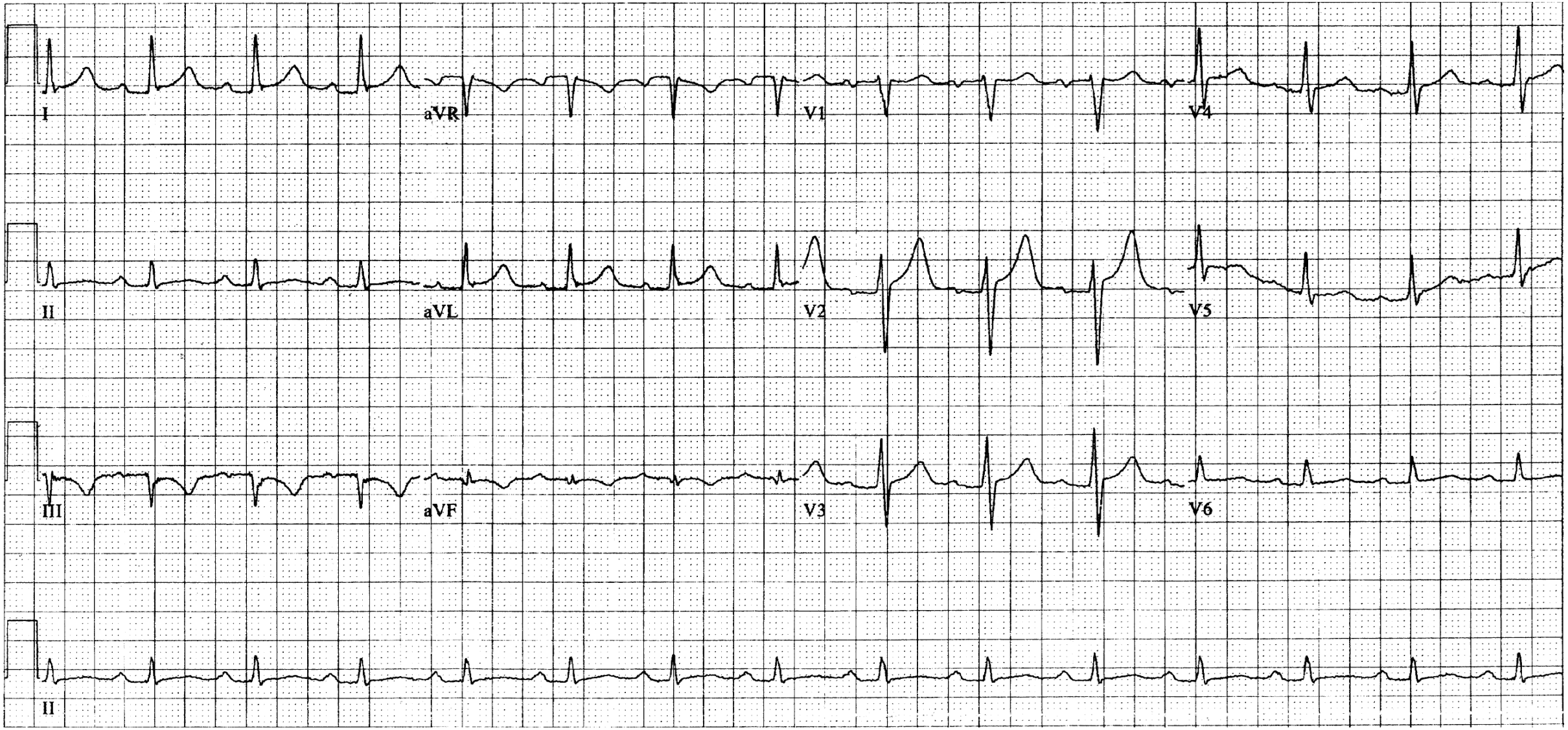
The figure shows the location of the posterior accessory leads V7, V8 and V9 correspondent to inferobasal segment (old posterior).

ECG-4 July 02, 2013



ECG diagnosis: Sinus rhythm, left atrial enlargement, prolonged PR interval, inferior necrosis and ischemia observed in III and aVF

ECG-5 July 03, 2013



ECG diagnosis: Sinus rhythm, HR 88bpm, left atrial enlargement, prolonged PR interval, inferior necrosis and negative T waves observed in III and aVF. The negative T wave of ischemic origin may be seen in the clinical setting of post MI as a consequence of reperfusion in case of aborted MI when the artery has opened spontaneously or after fibrinolysis, percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG) or coronary spasm (**Bayés de Luna 2014**)).

Multi-vessel coronary disease: Percutaneous Coronary Intervention (PCI) versus Coronary Artery Bypass Grafting (CABG)?

Patients with multi-vessel disease comprise the majority of patients undergoing PCI today and will likely remain so. With improved techniques, stents, and adjunctive drugs, outcomes have improved significantly. It is anticipated that if the early experience with drug eluting stents is replicated in multi-vessel disease then the outcomes of PCI will be equivalent to CABG. PCI would therefore become a preferred strategy for the majority of patients needing revascularization. Initial estimates suggest that the number of PCI procedures will grow by 10% while surgical cases will fall. The decline in restenosis will be equally offset by increased percutaneous revascularization. The future is clearly bright for angioplasty and the advances over the past 25 years have been truly remarkable.

Comparative effectiveness of CABG surgery and PCI in patients with diabetes(the present case)

Currently, the appropriateness of PCI using drug-eluting stents (DES) versus CABG for patients with diabetes and multi-vessel disease (MVD) is uncertain due to limited evidence from few randomized controlled trials (RCTs). using an evidence-based approach.

Ariyaratne et al (**Ariyaratne 2014**) conducted systematic review and meta-analyses to compare the risk of all-cause mortality, myocardial infarction (MI), repeat revascularization, cerebrovascular events (CVE), and major adverse cardiac or cerebrovascular events (MACCE).

A total of 1,837 and 3,052 DM-MVD patients were pooled from 4 RCTs (FREEDOM, SYNTAX, VA CARDS, and CARDia) and five non-randomized studies. At mean follow-up of 3 years, CABG compared with PCI-DES was associated with a lower risk of all-cause mortality and MI in RCTs. By contrast, no significant differences were observed in the mean 3.5-year risk of all-cause mortality and MI in non-randomized trials. However, the risk of repeat revascularizations following PCI-DES compared with CABG was 2.3 folds higher in RCTs and non-randomized trials, respectively. Accordingly, the risk of MACCE at 3 years following CABG compared with PCI-DES was lower in both RCTs and non-randomized trials respectively.

Based on these pooled results, the authors recommend CABG compared with PCI-DES for patients with DM-MVD. Although non-randomized trials suggest no additional survival-, MI-, and CVE- benefit from CABG over PCI-DES, these results should be interpreted with care.

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