

# Prominent QRS anterior forces during Acute Coronary Syndrome with ST Segment Elevation Myocardial Infarction



Raimundo **Barbosa-Barros**, M.D. (nickname “Raymond, the Fox”)  
Chief of Coronary Center of the Hospital de Messejana Dr. Carlos Alberto Studart Gomes, Fortaleza, Ceará CE, Brazil

## English: Case report

Black, 56-year-old, female patient. She was admitted into the ER of the *Hospital Geral*, where because of sensory decrease, she was initially diagnosed with stroke, not confirmed by brain CT (normal result). She presented cardiogenic shock by low cardiac output. There was a considerable delay to refer her to our specialized tertiary hospital (3 h 45 min). Taking her hemodynamic instability and the ECG pattern in admission (ECG1) as basis, there was a change in diagnosis to ST segment elevation acute coronary syndrome, and there was a subsequent therapeutic strategy. ECG2 corresponds to the tracing made 3 h 45 min after ECG1, being immediately sent to the hemodynamic lab to perform primary percutaneous coronary intervention, the coronary angiography of which revealed proximal LADA and LCx artery occlusion and total RCA occlusion due to a thrombi. Probably, RCA was supplying collateral circulation to the ADA territory. In the hemodynamic room, the patient presented sudden electrical activity with no pulse, followed by asystole, which did not respond to reanimation maneuvers. The patient presented cardiorespiratory arrest at the end of the examination.

PCI (thromboaspiration + stent in the RCA). She recovered sinus rhythm 30 minutes after reanimation maneuvers (post-cath ECG). There was an attempt of intervention in the ADA, that was unsuccessful. The patient was sent back to the ER with low cardiac output syndrome, and he died soon after.

Questions:

1. Which is/are the underlying mechanism(s) of very tall R wave (“Giant R waves“) from V1 to V3 of ECG-1?
2. Which are the diagnoses of ECG2?

## Portuguese: Relato de caso

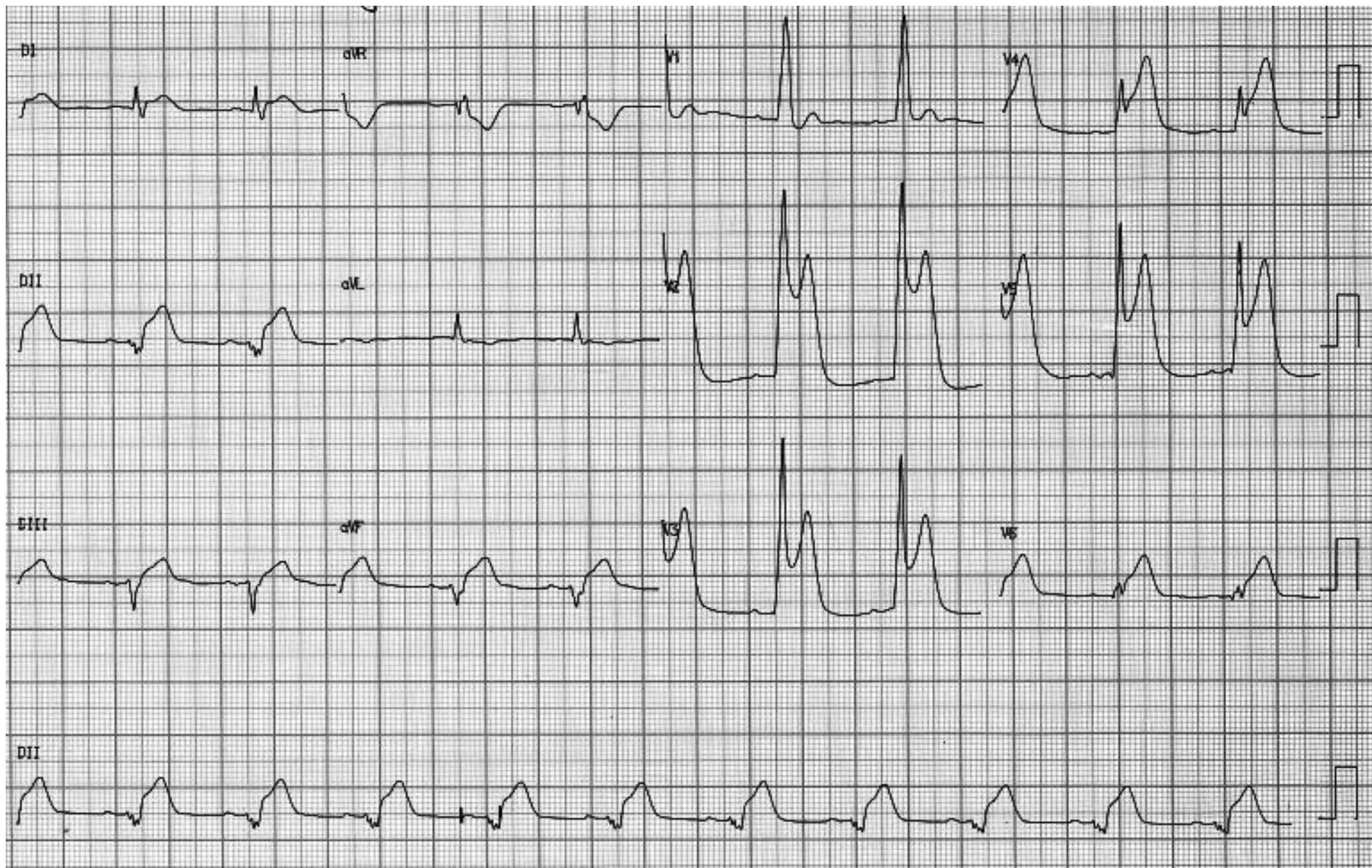
Paciente feminina, negra, 56 anos, deu entrada na emergência de um Hospital Geral, onde, em virtude do rebaixamento do sensório, teve diagnóstico inicial de acidente vascular cerebral (AVC), não confirmado na tomografia cerebral computadorizada (resultado normal). Apresentava choque cardiogênico por baixo débito. Houve retardo considerável para o encaminhamento ao nosso hospital terciário especializado (3h45). Tendo como base a instabilidade hemodinâmica e o padrão eletrocardiográfico admissional (ECG-1), houve mudança de diagnóstico para síndrome coronariana aguda com elevação do segmento ST, e conseqüentemente mudança de estratégia terapêutica.

O ECG-2 corresponde ao traçado realizado 3h45 depois do ECG-1, sendo imediatamente encaminhada para o laboratório de hemodinâmica para a realização de intervenção coronária percutânea primária, cuja coronariografia revelou oclusão proximal da DA e Cx e oclusão total da coronária direita, por trombo nesta última. Provavelmente, a coronária direita supria de circulação colateral o território da DA. A paciente apresentou PCR ao término do exame PCI (tromboaspiração + stent na CD). Recuperou o ritmo sinusal 30' após manobras de reanimação (ECG pós cate). Houve uma tentativa de intervenção na DA sem sucesso. Foi encaminhado de volta para sala de emergência com síndrome de baixo débito vindo à falecer logo em seguida.

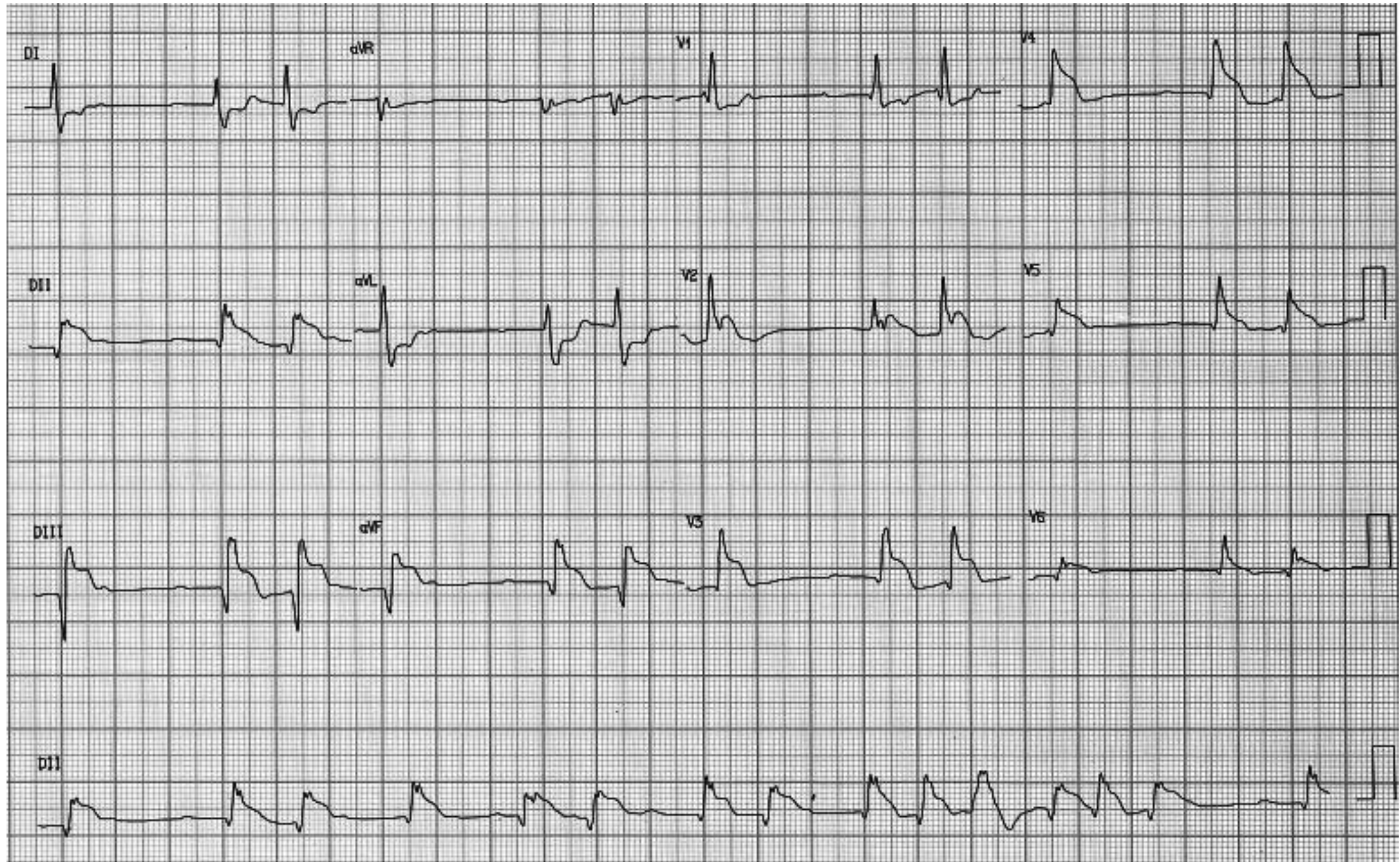
Perguntas:

1. Qual o mecanismo das forças anteriores proeminentes do ECG-1?
2. Quais são os diagnósticos do ECG-2?

# ECG1 - At admission 17h



# ECG2 - 20h45



Colleagues opinions

Dear Andres,

Here are my thoughts on this:

ECG 1 represents an acute anterolateral myocardial infarction. The tall R waves may represent posterior myocardial infarction. That fits with the patient presentation with three vessel disease.

ECG 2 is AV nodal Wenckebach. Decreasing the amplitude of the QRS. The location of the myocardial infarction remains the same. Lowering the amplitude presents the question that if the patient had tamponade, which could also cause her cardiogenic shock.

Please let me know your final diagnosis.

Very best,

Mohammad **Shenasa MD, FACC, FESC, FAHA, FHRS,**

Heart & Rhythm Medical Group

105 N. Bascom Ave Suite 204

San Jose, CA 95128. USA

408-930-9400 (Mobile)

408-286-2922 (Fax)

[mohammad.shenasa@gmail.com](mailto:mohammad.shenasa@gmail.com)



Portuguese

Caro Andres,

Aqui estão os meus pensamentos sobre isso:

ECG 1 representa um infarto do miocárdio agudo ântero-lateral. As ondas R pode representar infarto do miocárdio posterior. Que se encaixa com a apresentação de paciente com doença dos três vasos.

ECG 2 é um bloqueio AV tipo Wenckebach nodal. Diminuindo a amplitude do QRS. A localização do enfarte do miocárdio permanece a mesma.

Diminuindo a amplitude apresenta a questão que se o paciente teve tamponamento, que também poderia causar o choque cardiogênico.

Por favor, deixe-me saber o seu diagnóstico final.

O melhor,

Mohammad

Dear Andrés and Raimundo – again a rare case!

Due to the described severely disturbed coronary artery circulation (left main equivalent + RCA occlusion), there is very wide-spread ST elevation including the anterior and inferior regions and even to some extent the lateral region. There is no ST elevation in V1, probably due to the involvement of the LCx. There is severe grade III ischemia in the anterior wall. There seems to be a left axis deviation, probably due to LAFB. There are also extreme prominent anterior forces. Comparing with the second ECG, it does not seem to represent an old mirror-image Q wave (of old lateral MI). It could be LSFB, but one would have expected small initial q waves in V1-V3 and absence of q waves in V5-V6 in a typical case. In the second ECG the ST elevations have decreased and there seems to be RBBB. Q waves have developed in the inferior leads and there is a slight change in the frontal QRS axis – lead aVL is now predominantly negative, the axis is in the normal range.

A very severe case in many ways. Thank you for sending these challenging cases to us!

Thank you

Kjell Nikus (nick name: “The English Lord”), Tampere, Finland

Andrés’s comment: Why this nickname? Because he has English Lord life philosophy:

If you wish to let the world know you are in love with a particular man, treat him with formality, and everyone else with ease and freedom.

If you are disposed to be pettish or insolent, it is better to exercise your all humors on your dog, your cat, or your servant, than your friends.

If you would preserve beauty, rise early.

If you would preserve esteem, be gentle.

If you would live happy, endeavor to promote the happiness of others.





Great case!

One thing for sure. The transient tall R waves in V1-V3 are not Q wave equivalent of inferolateral infarction, as they decreased in size in the second ECG. Both ECGs show RBBB, the first one with left axis deviation. Severe ischemia of the LAD can cause transient increase in the R wave amplitude (QRS distortion) that resolves over time (collaterals? Or even without reperfusion).

The initial ECG showed ST elevation in I, II, III and aVF and V2-V6 with ST depression in aVR. Despite having LM equivalent occlusion, supporting the idea that lead aVR is a calculated lead (I and II) and not an independent lead.

The decrease in ST elevation in the precordial leads in the second ECG may indicate either decrease in the severity of ischemia (collaterals) or that the inferolateral ischemia attenuates the ST elevation in the anterior leads.

Yochai **Birnbaum, M.D., FAHA, FACC**

Thank a lot for your input dear Yochai, and happy Jewish new year for you and family  
All the best Andres.



Dear Edgardo, Raimundo and Andrés,

This ECG suggests that the morphological base of the heart presents septal anterior hypertrophy in the right, left, superior, middle and inferior septum (V1 to V4). There is a technical problem: the left precordial electrodes were exchanged in their positions: V4 is V5 and vice versa.

I) Elevated ST-T in I and not in aVL, suggesting that ischemia affects the middle lateral wall and not the high basal lateral, which expresses in aVL.

II) It presents the same morphology as V6, suggesting that the ischemic border is in the inferior area of the posterior lateral wall.

III) ST-T smaller than DII, suggesting the edge of the inferior wall is expressing the inferior edge of ischemia.

aVR) Depressed ST-T means mirroring morphology to V5

aVL) It constitutes the reciprocal remodeling edge of DIII

aVF) ST-T greater than DIII, suggesting that the posterior wall is more ischemic than the inferior one

V1) It expresses the right septum, which constitutes the lateral edge of the ischemic process, suggesting that the septum has double circulation from the marginal or right conal arteries and the left septal arteries. If it was only from the left septal arteries, he would also be presenting very elevated ST-T. QRS is high because of right septal hypertrophy.

V2) Degree 3 of ischemia: ST-T segment more elevated, expressing that the ischemic focus is in the superior septum. ST-T elevation of more than 20 mm is due to septal hypertrophy.

V3, V4 express the potentials of the inferior septal areas, but decreasing.

V5) It is in the position of V4, expressing the ischemic edge.

All of the above suggests sudden obstruction of ADA, dominant distal to the first diagonal artery (if it had been proximal, it would be expressed with elevated ST-T in aVL).

ECG2 is pre-mortem, suggesting the first stage of electrical-contraction dissociation. Non-arrhythmogenic sudden cardiac death.

As my friend, Kjell C. Nikus would say, according to the ECG first the LADA should be opened. Nikus is the pioneer of ECG reading in the hemodynamic lab.

Best regards and the discussion is open, and to all the Jewish members of the forum, Happy New Year! (we celebrate New Year twice per year).

Samuel Sclarovsky MD Israel

Estimando Edgardo, Raimundo y Andrés; Este ECG sugiere que la base morfológica del corazón tiene hipertrofia anterior septal en septo derecho, izquierdo, superior, medio e inferior (V1 a V4) Hay un problema técnico: se cambio de posición de los electrodos precordiales izquierdos: V4 es V5 y viceversa

DI) ST-T elevado en DI y no en aVL sugiriendo que la isquemia afecta la pared lateral media y no la lateral basal alta, que se expresa aVL

DII) Tiene la misma morfología que V6, sugiriendo que el borde isquémico está en el área inferior de la pared lateral posterior.

DIII) El ST-T menor que DII sugiriendo el borde de la pared inferior expresa el borde inferior de la isquemia

aVR) ST-T deprimido significa morfología en espejo de V5

aVL) forma el borde de remodelación recíproca de DIII

aVF) ST-T mayor que DIII, sugiriendo que la pared posterior está más isquémica que la inferior

V1) expresa el septo derecho que forma el borde lateral del proceso isquémico, sugiriendo que el septo tiene doble circulación de la marginal o conal derecha y de las septales izquierdas. Si fuese únicamente de las septales izquierdas presentaría también ST-T muy elevado. El QRS alto es debido a una hipertrofia septal derecha

V2) isquemia grado 3: el segmento ST-T es más elevado expresando que el foco isquémico está en el septo superior. La altura del ST-T más de 20mm es debido a la hipertrofia septal V3, V4 que expresan los potenciales de las áreas septales inferiores, pero van "in decrescendo"

V5) que está en posición V4 expresa el borde isquémico

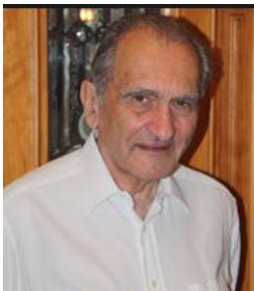
Todo lo dicho sugiere una obstrucción súbita de la arteria coronaria descendente anterior, dominante distal a la diagonal primera (si fuese proximal se expresaría con un ST-T elevado en aVL)

El ECG-2 es de premortem, sugiriendo la etapa primera de la disociación eléctrica-contracción: Non arritmogenic sudden death

Como diría mi amigo Kjell C Nikus, según el ECG hay que abrir primero a la LADA. Nikus es el pionero de la lectura de ECG en el laboratorio hemodinámico

Un fraternal abrazo y la discusión está abierta y a todos los foristas judíos feliz año Nuevo (nosotros festejamos 2 veces al año nuevo)

Samuel Sclarovsky



Portuguese: Caro Andrés Envio minha humilde opinião:

ECG 1: Ritmo sinusal , elevação do segmento ST envolvendo as paredes infero-lateral e anterior.

Não existe depressão do segmento ST como imagem recíproca dos segmentos ST nas 12 derivações. Teria sido interessante poder analisar as derivações V7-V9.

As forças anteriores proeminentes provavelmente se devem ao envolvimento da parede dorsal do ventrículo esquerdo secundário à oclusão aguda proximal de uma coronária direita dominante, acrescido de um bloqueio médio septal esquerdo.

Acho também que existe um BDASE associado (um vetorcardiograma esclareceria sua existencia).

Quanto ao ECG 2 esses agrupamentos dos QRS seguidos de pausa sugerem bloqueio AV 2o grau tipo I a nível nodal pois os intervalos PR observados são longos, fato comum no infarto agudo envolvendo a parede inferior.

Aguardo as opiniões dos grandes mestres.

Grande abraço

Marcelo Leal, MD

Médico Assistente da Divisão de Cardiologia do Hospital das Clinicas da Faculdade de Medicina de Ribeirão Preto- USP. SP, Brasil.



English Dear Andrés

I send my humble opinion:

ECG 1: sinus rhythm, ST segment elevation involving the inferiolateral and anterior walls.

There is no ST segment depression as reciprocal image of the ST segment on the 12 leads. It would have been interesting to analyze the V7-V9 leads.

The prominent anterior forces are probably due to the involvement of the dorsal wall of the left ventricle secondary to acute proximal occlusion of a right coronary dominant, plus a left septal middle block.

I also think there is an associated LAFB (a vectorcardiogram clarify its existence).

Regarding ECG 2, these two groups of QRS followed by pause suggest second degree AV block type I at nodal level as the PR intervals observed are long, which is common in acute myocardial infarction involving the inferior wall.

I await the opinions of the great masters.

Big hug

Looks like left main equivalent with generalized ST elevation, prominent anterior forces may be due to post inferior MI with reciprocal changes or perhaps associated left midseptal fascicular block.

ECG2 shows MI in evolution and likely AV node Wenkebach conduction.

**Melvin M. Scheinman, MD, FHRS**

Department of Cardiac Electrophysiology, University of California San Francisco, San Francisco, California, USA.

[scheinman@medicine.ucsf.edu](mailto:scheinman@medicine.ucsf.edu)

Professor of Medicine

Address:

UCSF

Electrophysiology Service

500 Parnassus Avenue

San Francisco, CA 94143-1354. USA.

Telephone/FAX/E-mail:

Phone: (415) 476-5706



Good case presented by our friends!!

ECG 1. I think that the ECG pattern can be explained by: the ST elevated in II, III, aVF is due to acute occlusion of the RCA. The ST elevated in V2 to V6 is due to ischemia in the territory of the LAD who was receiving collateral circulation from the RCA. The ST is not elevated in V1 because the LCX is chronically occluded. The deviation of the axis to the left seems LAFB. High voltage R wave is due to blockade of the Middle fibers (transient).

ECG 2. The deviation of the axis to the left is gone. There is a pattern of RBBB. Sinus rhythm with second degree AV block and variable AV conduction.

Best Regards

*Miquel Fiol Sala, MD, PhD*

*Director Científico del Instituto de Investigación*

*Sanitaria de Palma (IdISPa)*

*IP del CIBERobn*

*Hospital Son Espases*

*Ctra de Valldemossa, 79*

*07010 Palma de Mallorca*

*Tfno: 871205987/871205050, ext 75987*

*e.mail: [miguel.fiol@ssib.es](mailto:miguel.fiol@ssib.es)*



# Final comments

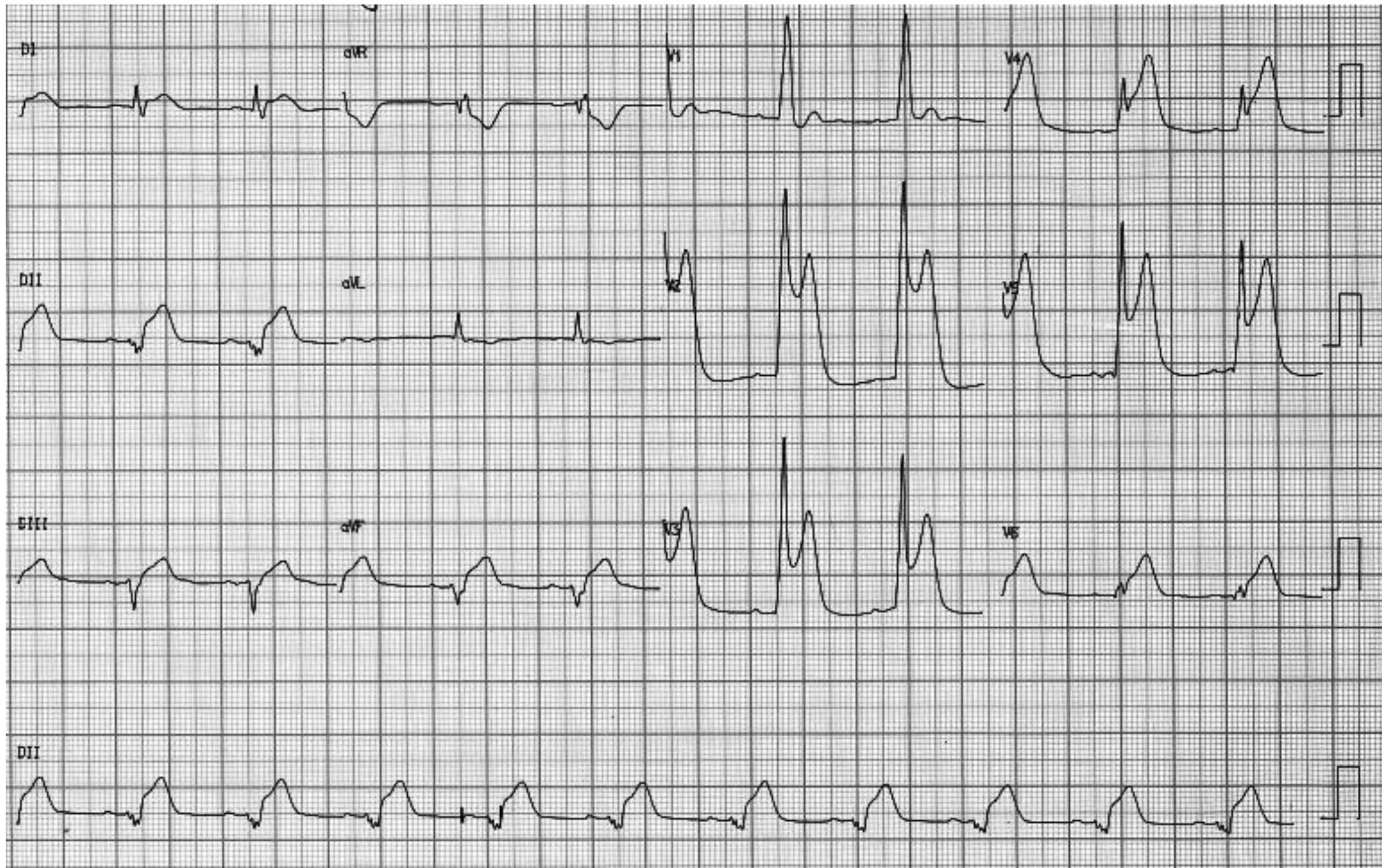


Design of Studies and Scientific Writing Laboratory at the ABC School of Medicine, Santo André, São Paulo, Brazil

**ECG / VCG Pérez-Riera | my cardiology site of scientific interests:**

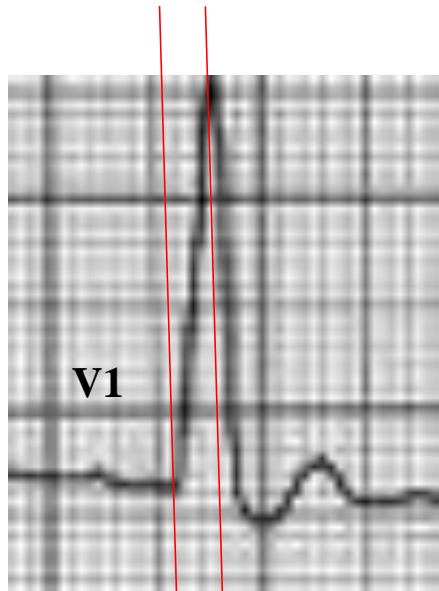
<https://ekgvcg.wordpress.com>

# ECG1 - At admission 17h



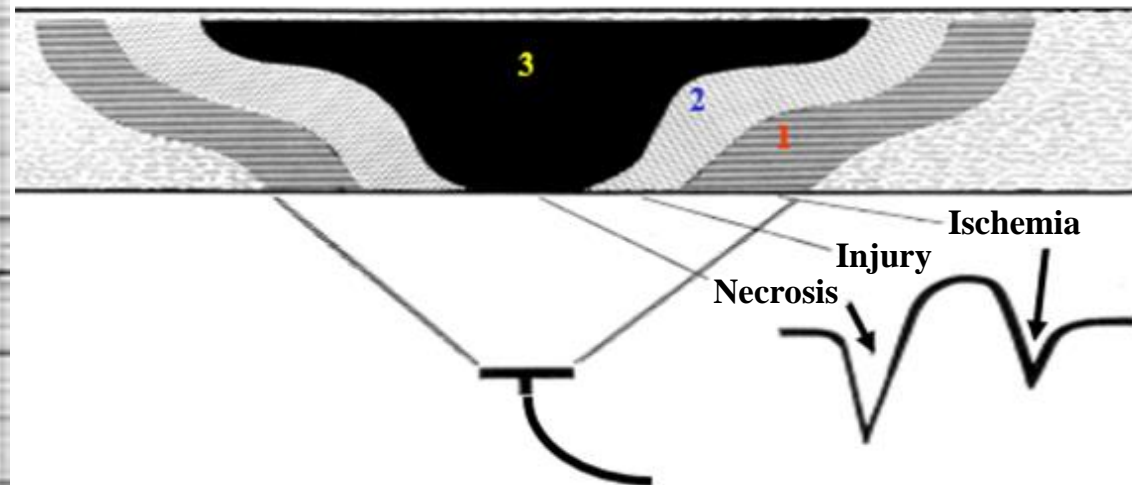
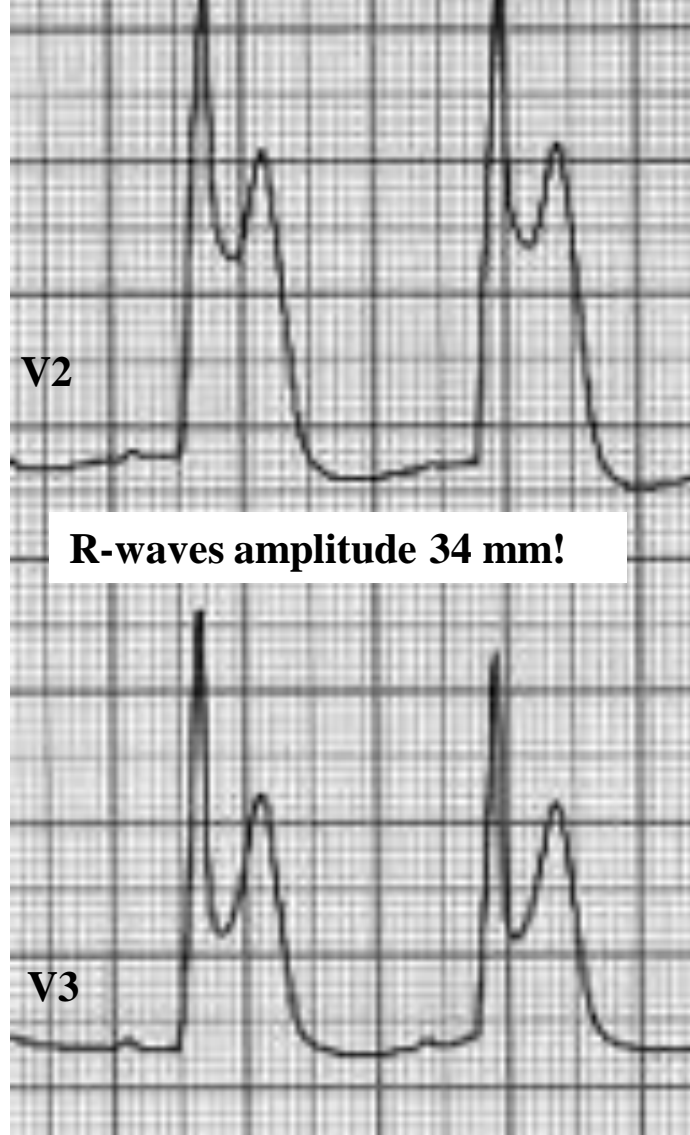


# ECG-1 17h

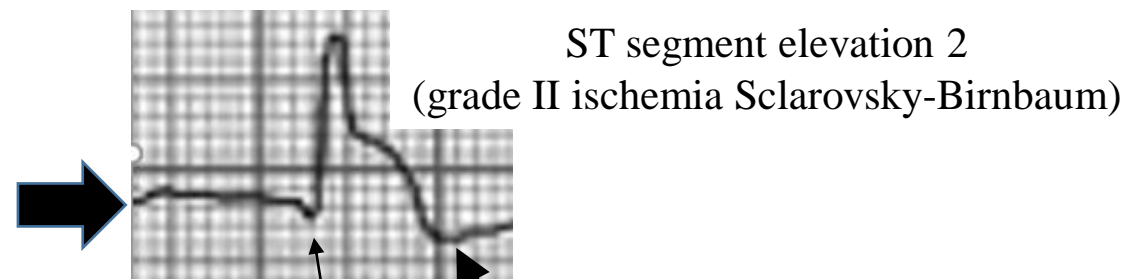


Prolonged R-wave peak time (RWPT) = 85ms.

The normal value of RWPT in V1-V2 is < 35 to 50 ms (Pérez-Riera 2016 a)



# ECG-2 20h45. Three hours and 45 minutes later



Tall, symmetrical, and peaked T-waves (grade I ischemia Sclarovsky-Birnbaum);  
 No S waves in leads V1-V3, J-point/R wave ratio  $\geq 0.5$  ST segment elevation (grade III ischemia)  
 Transient development of very tall R wave ("Giant R waves") or Prominent Anterior QRS Forces(PAF) Increase in the amplitude of the R-waves  
 Disappearance of the S waves in leads V1-V3(grade III ischemia Sclarovsky-Birnbaum)

Necrosis Insinuation 3

Changes in the terminal portion of the QRS complex (grade III ischemia)

# The Grades of Ischemia on the Presenting ECG of Patients With ST Elevation Acute Myocardial Infarction

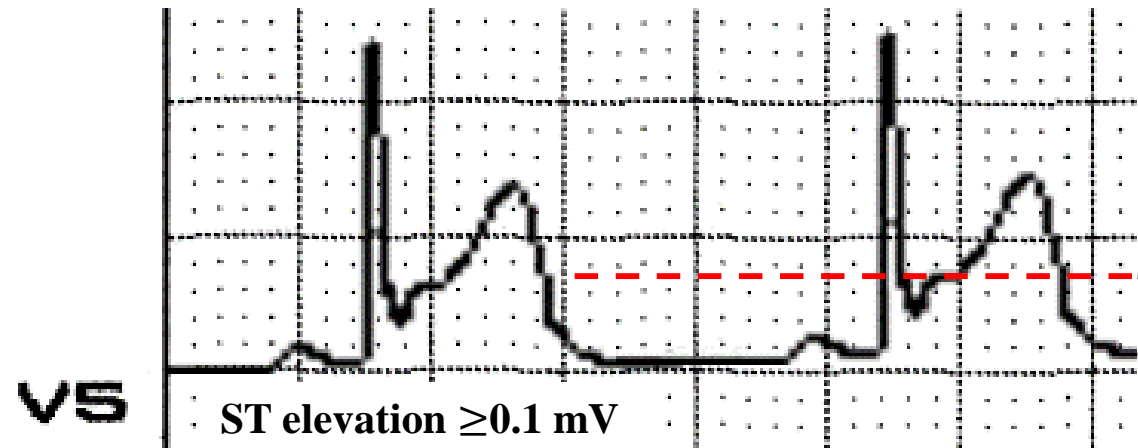
(Birnbaum-Sclarovsky 2001)

## Sclarovsky-Birnbaum Ischemia Grading System

- I. **Grade I (GI1):** first, the T waves become tall, symmetrical, with broad base, peaked not tented or not pointed but without ST elevation.



- II. **Grade II (GI2):** ST elevation  $\geq 0.1$  mV without distortion of the terminal portion of the QRS. The S waves in leads V1–V3 are preserved and the J-point/ R-wave ratio is  $< 0.5$ . This might be related to less protection by “ischemic preconditioning” or presence of collateral circulation.

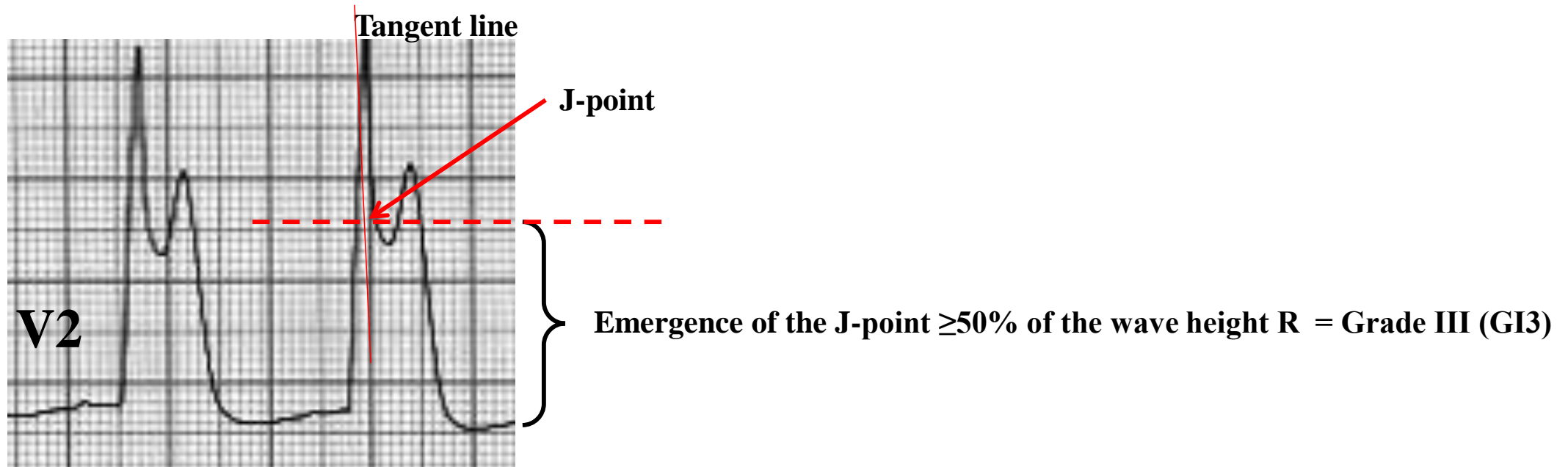


J-point/ R-wave ratio is  $< 0.5$

**III. Grade III(GI3):** Patients with acute myocardial infarction who have increase in the amplitude of the R waves, with ST-segment elevation, and terminal QRS distortion in  $\geq 2$  adjacent leads: No S waves in leads V1–V3, J-point/R wave ratio  $\geq 0.5$ . In other words 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. This grade is indicative of more severe ischemia, higher mortality, larger final infarct size, and faster progression of necrosis. GI3 is an independent predictor of mortality among patients with STEMI. Mortality increased significantly with symptom duration in both GI2 and GI3. Primary percutaneous coronary intervention may be especially beneficial for patients with GI3 presenting early, whereas patients with GI3 presenting late and treated with thrombolytic treatment are at particular risk of reinfarction (**Sejersten 2006**). Additionally, GI3 patient are older (**Birnbaum 2001;2003**).

The present case

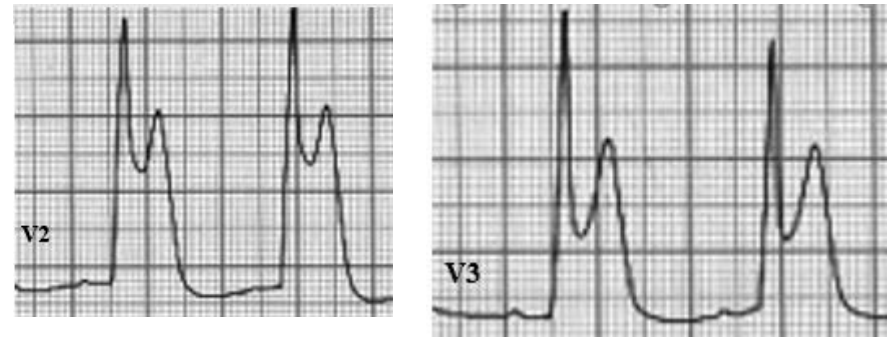
(**Sclarovsky 1989; 1990**)



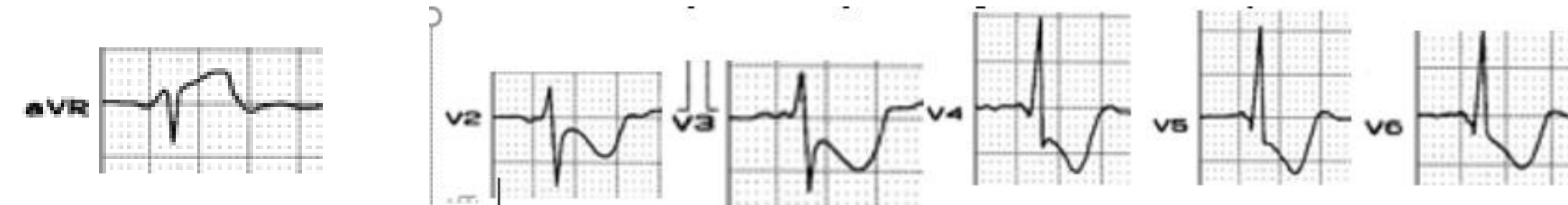
No S waves in leads V1–V3, J-point/R wave ratio  $\geq 0.5$

Initially, Schakowsky et al in 1989 (**Sclarovsky 1989**) classify patients with ischemia but without tachycardia according to ECG patterns recorded during chest pain by precordial leads as follows:

**I. Group 1:** those with ST segment elevation followed by a positive T waves. These patients have anterior wall ischemia and the LAD artery is usually involved. They are candidates for primary percutaneous coronary intervention (PPCI) ( the present case).



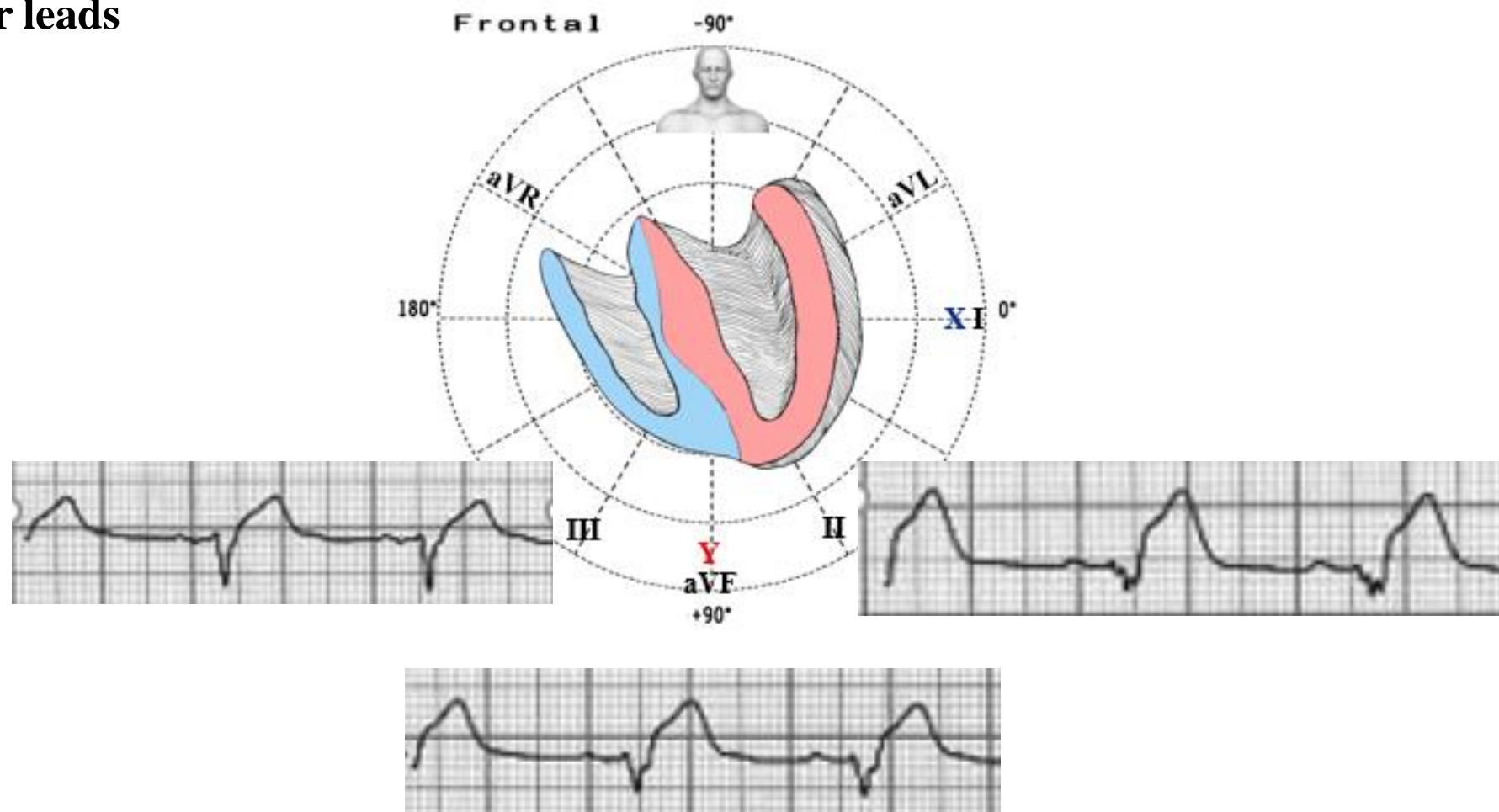
**II. Group 2:** ST segment depression followed by negative T waves: this pattern reflect extensive subendocardial myocardial ischemia: LMCA or three vessel equivalent (LMEQ). Frequent cardiogenic shock. Immediately PPCI is indicate. Circumferential ischemia  $\geq 7$  surface leads combined with ST elevation in aVR > V1. (absent in this case).



This ECG pattern is not always caused by LMCA/LMEQ disease; therefore, the term "suspect circumferential subendocardial ischemia" may be preferred because other medical conditions may also be associated with a similar ECG pattern. Specificity for LMCA/LMEQ disease is low: 26% (**Knotts 2013**).

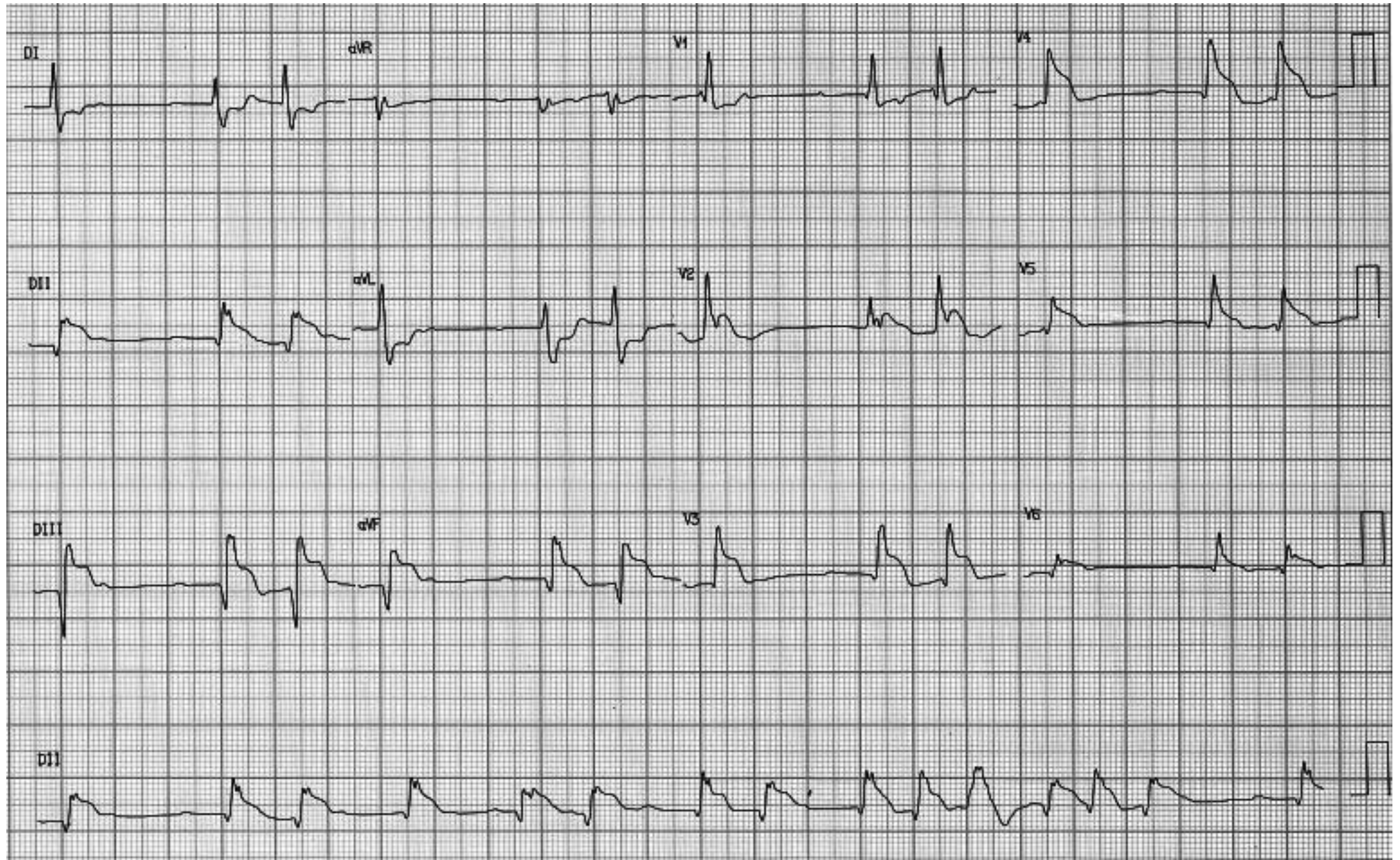
**III. Group 3:** ST depression followed by positive T waves. **Observation:** Patients with ischemic ST depression maximal in V2-V3 should not be included in this group since these changes are reciprocal with posterolateral wall ischemia. The presence of reciprocal ST segment depression during an acute inferior myocardial infarction correlates with the presence of multivessel coronary artery disease and may not be only an electrical phenomenon (**Zoghi 2003**).

# ECG-1- inferior leads

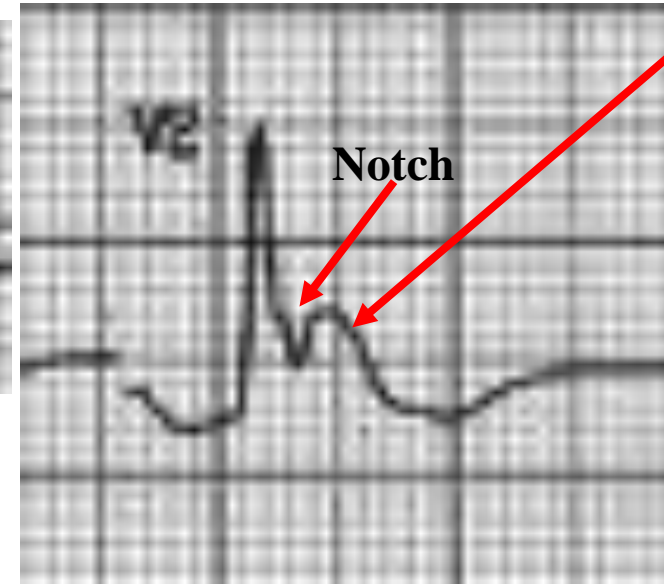
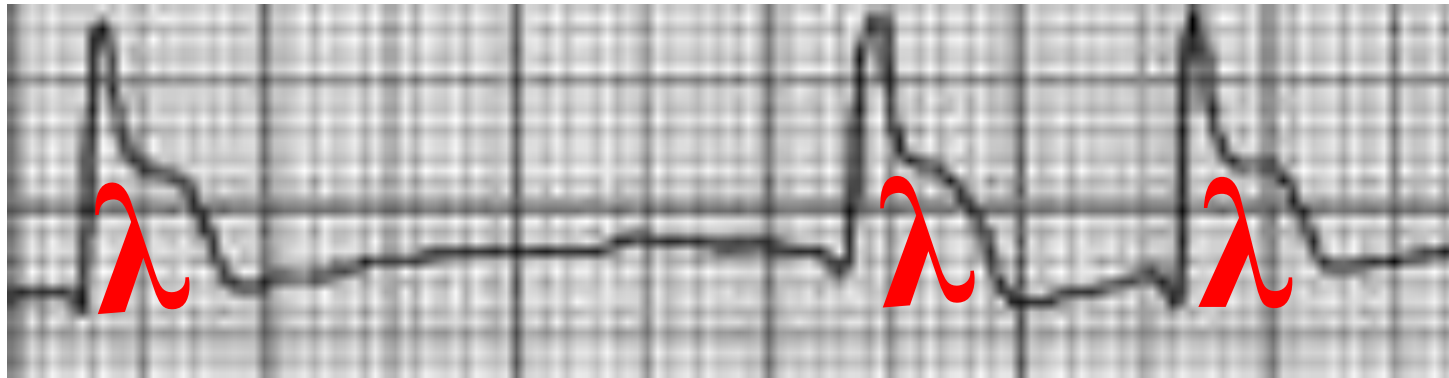


“Giant” R-waves in association with an acute inferior myocardial infarction (MI). Such ECG pattern has been associated heretofore with the hyperacute phase of an anterior MI, and unstable, and Prinzmetal angina, although it is found in conjunction with inferior MI. The “giant” R-waves, along with ST-SEs, were noted transiently such as the present case during very , early in the clinical course of MI. Subsequent evolution of the ECG revealed classic appearances for an inferior MI. Cardiac enzymes, and thallium-201 myocardial perfusion scintigraphy reveal evidence for inferiorly-located myocardial necrosis. Coronary arteriography show total obstruction stenosis of the RCA, associated with proximal LAD for which the patient underwent an uneventful angioplasty and "stenting" of the culprit vessel (**Madias 2001**).

# ECG2 - 20h45



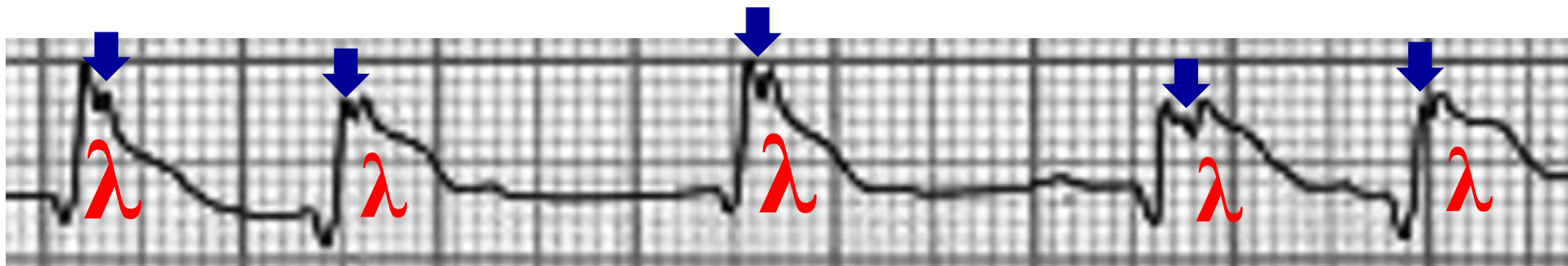
**ECG-2 pre-mortem: Acquired Early Repolarization Syndrome (ERS) with Lambda wave (A) and QRS notching with non-hypothermic acquired J-wave (B) without ST segment elevation**



Ischemia-mediated J-wave, J deflection, "the camel's hump"/ camel-hump sign, "late delta wave", elevated J-point, hathook junction, Non-hypothermic J-wave, prominent J wave, K wave, H wave, current of injury or Osborn wave.

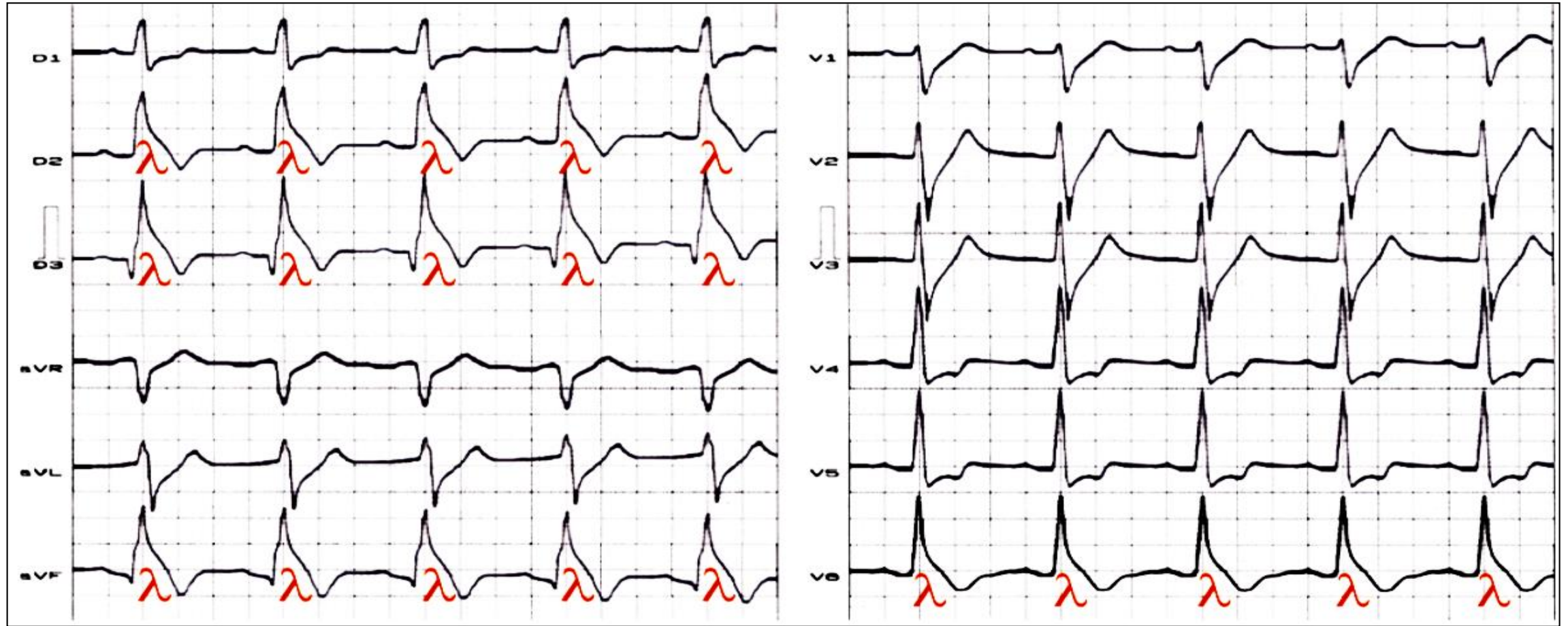
**Lambda (λ)-wave or Gussak wave (Gussak 2004). This is a marker of fatal arrhythmias**

A short period of ischemia during balloon inflation of primary percutaneous coronary intervention may induce visible electrical alternans and alternating QRS complexes with a characteristic ST-segment elevation with lambda morphology. A higher ST segment is associated with hypotension and vice versa (Oguro 2015).



Furthermore, near the apex of each QRS complex we observe the presence of clear QRS fragmentation (f-QRS), indicated by blue arrows, which multiplies the possibility of fatal arrhythmias (f-QRS + λ). The regular irregularity is consequence of Mobitz type 1 AV block (Wenckebach AV block).

Convex upward J waves, with horizontal/descending ST segments or "lambda-wave" ST shape are suggestive of IVF with early repolarization abnormalities. Premature ventricular contractions with very short coupling and "R on T" phenomenon are characteristics with two pattern: When originate from right ventricular outflow tract left bundle branch block morphology and from peripheral Purkinje network, left bundle (**Pérez-Riera 2012**). The following ECG below belongs to a young Asian man with typical lambda wave in the context of J-wave syndromes type II.



The patient was a young symptomatic (repetitive syncope episodes) Thai man, with positive familial background of SCD in young first degree relatives. He died 24h after performing this ECG. The ECG shows persistent ST segment elevation in the inferior and low lateral precordial apical leads (V5-V6), associated with concomitant reciprocal or mirror image in the anteroseptal wall that was not modified with the use of sublingual nitrate in absence of hypothermia, electrolyte imbalance or ischemia (**Riera 2004**).



## TYPICAL PVT IN ATYPICAL BRUGADA SYNDROME

Name: Y. A. S.

Gender: Male

Age: 26 years old.

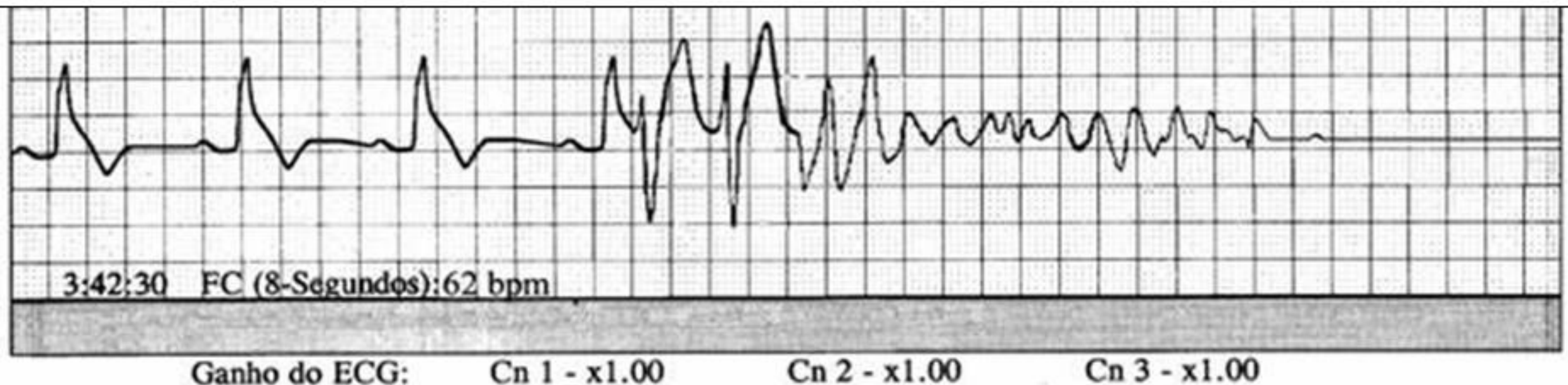
Ethnic Group: Asian Weight: 64 Kg.

Height: 1,68 m

Date: 03/05/2002

Time: 3:42:30 AM

Patient Sleeping.



Impresso: WED MARCH 06 18 19 17 2002

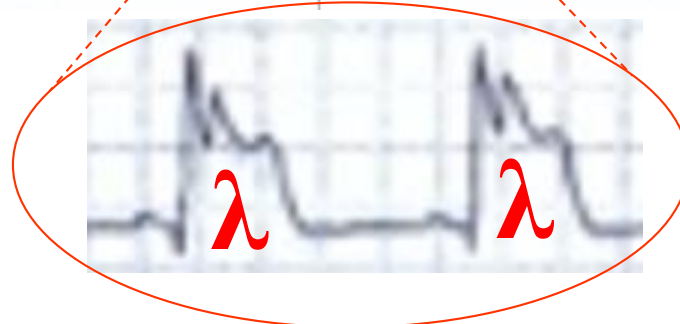
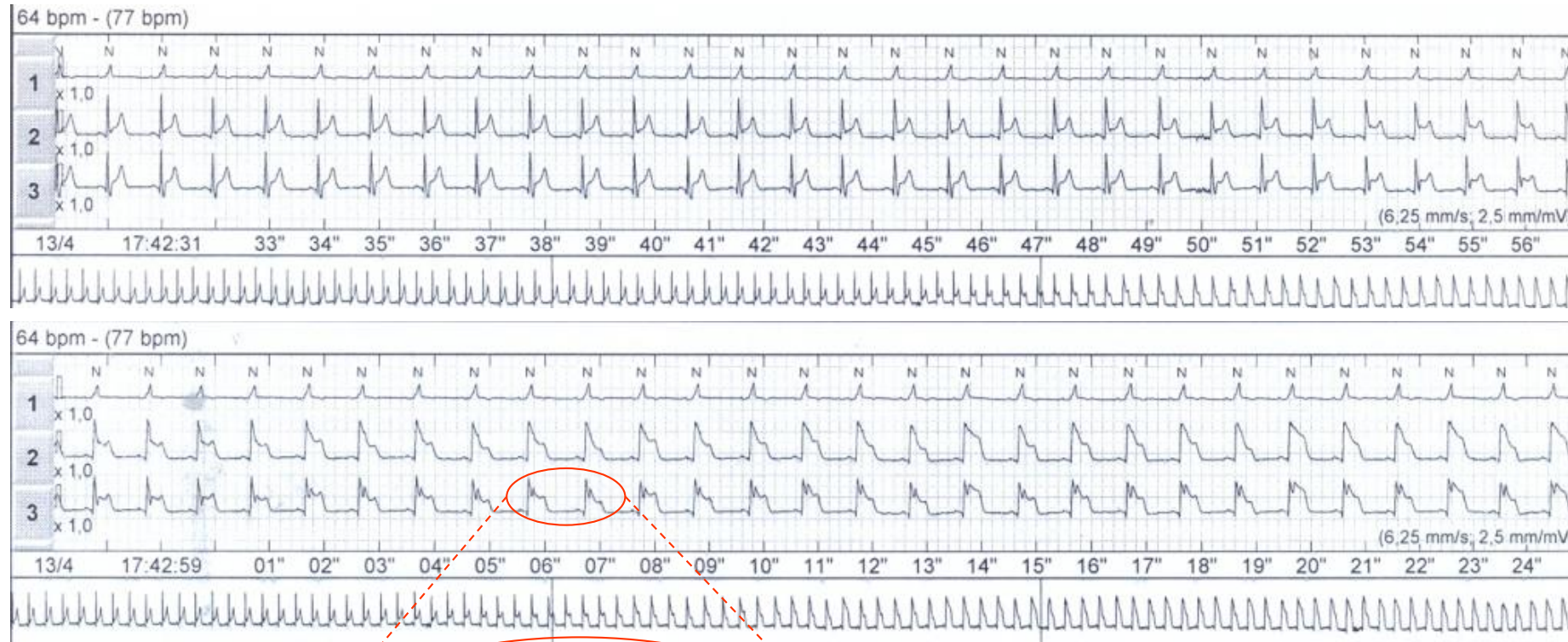
Produzido Por DMS DO BRASIL. (c) Copyright 1995. All Rights Reserved. Tradução Pacife & Pacife DMS 4.00

Os Dados Devem Ser Revisados Pelo Médico

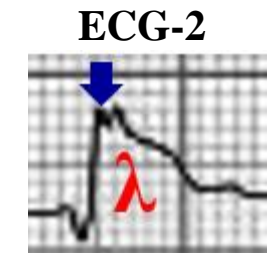
**LONG-TERM (HOLTER) ELETROCARDIOGRAPHIC RECORDING**  
**Sudden Cardiac Death by IPVT/IVF with short coupling ending in cardiac arrest.**

During myocardial ischemia in patients with Prinzmetal vasospastic angina. J-wave augmentations caused by myocardial ischemia during coronary spasms has lambda wave morphology. The presence and augmentation of J waves, especially prominent J waves with the characteristic ST-elevation patterns, were associated with VF (**Sato 2012**).

We show a continuous Holter monitoring below belonging to a man who had coronary revascularization a time ago, during an episode of angina and concomitant ST segment elevation and ischemic giant J-wave "lambda-like type" associated with Premature Ventricular Contractions with Bigeminy sequence and very short coupling. The PVCs disappear immediately after cessation of vasospastic ischemia with administration of sublingual nitrate.



**Observation:** the pattern is very similar with ECG-2 from the present case because we have f-QRS + lambda wave.

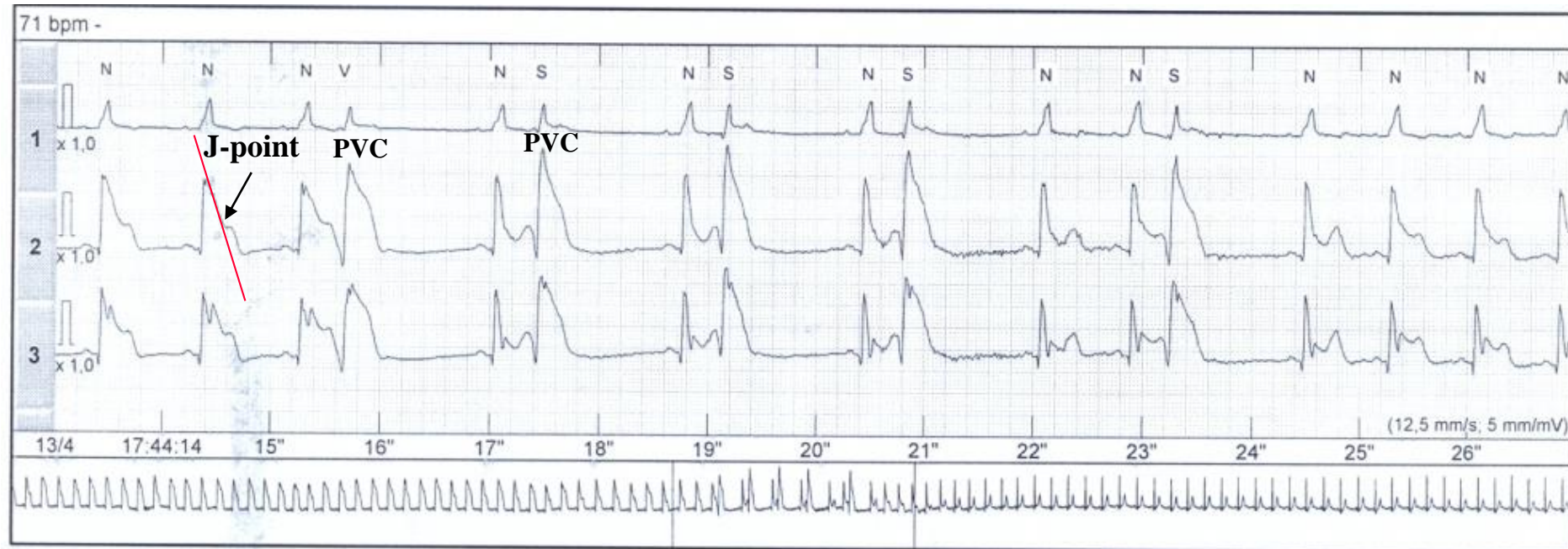




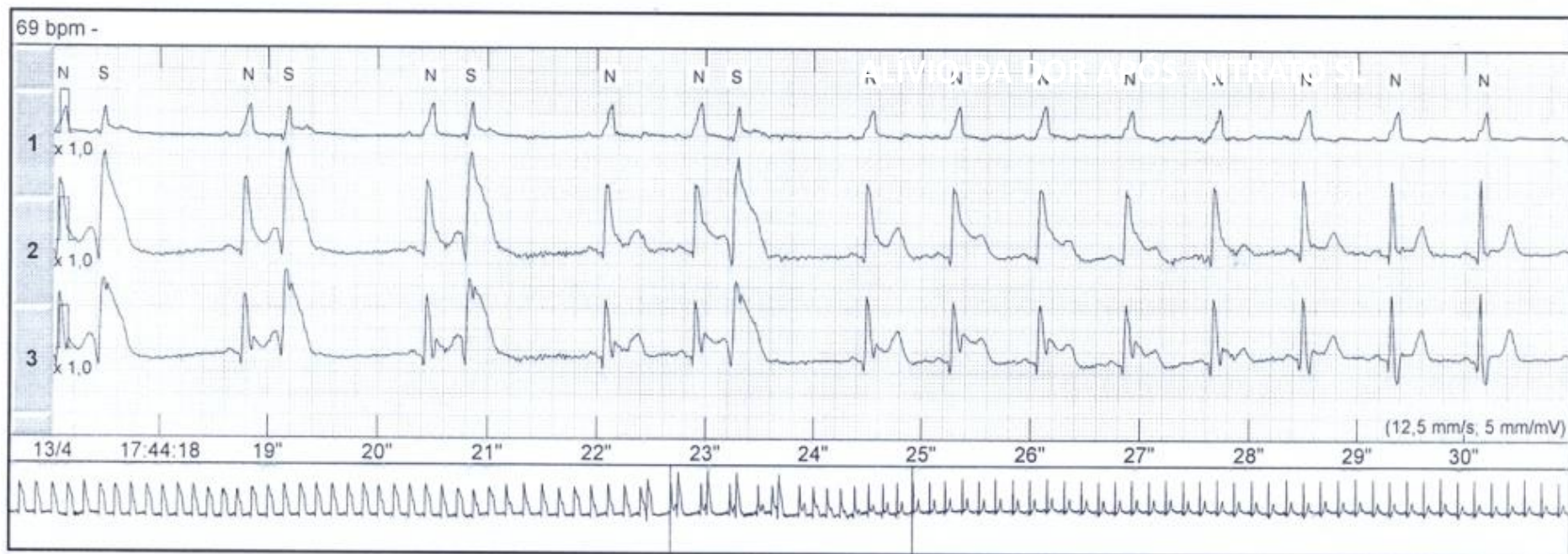
Holter monitoring shows STSE with lambda wave pattern during Prinzmetal angina. The last 4 group of beats are formed by sinus beat followed by premature ventricular contraction.



The first beat is sinus rhythm with minimal STSE. The second one has drastically augmentation of STSE followed by short coupled PVC. From third to eighth beat there are minimal STSE. The remaining trace has normal ST segment level.

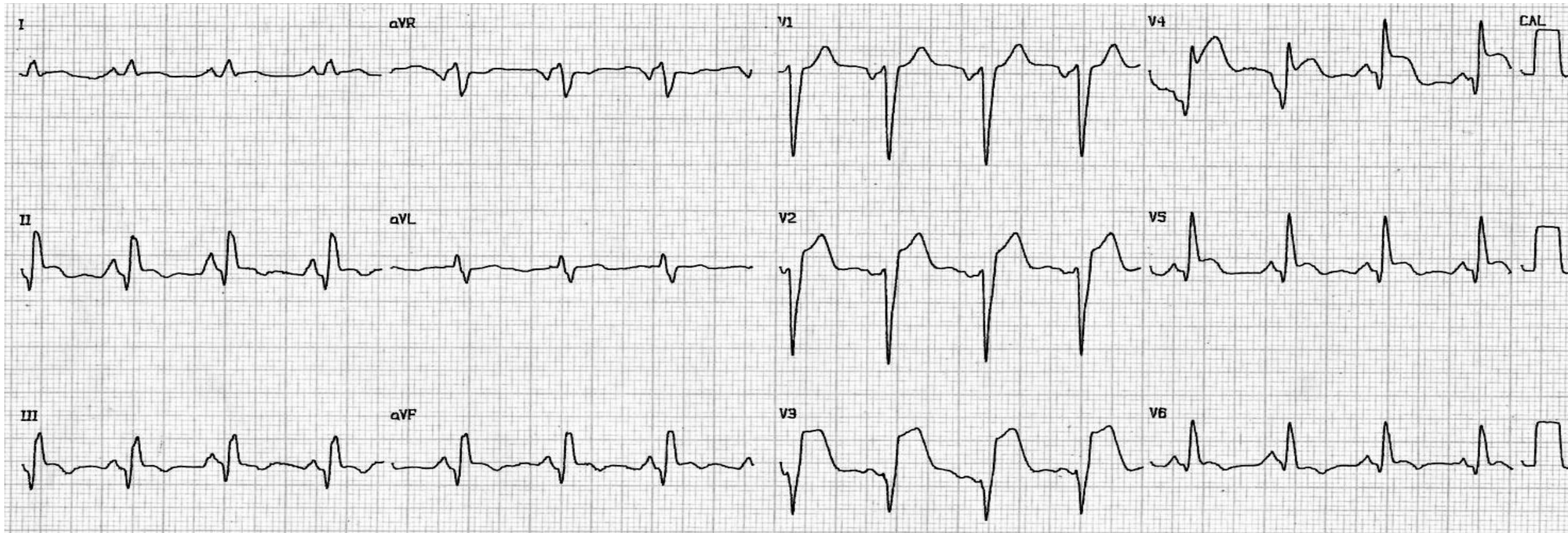


Premature ventricular contractions (PVCs) occurring after each sinus beat (bigeminy) with lambda wave shape.



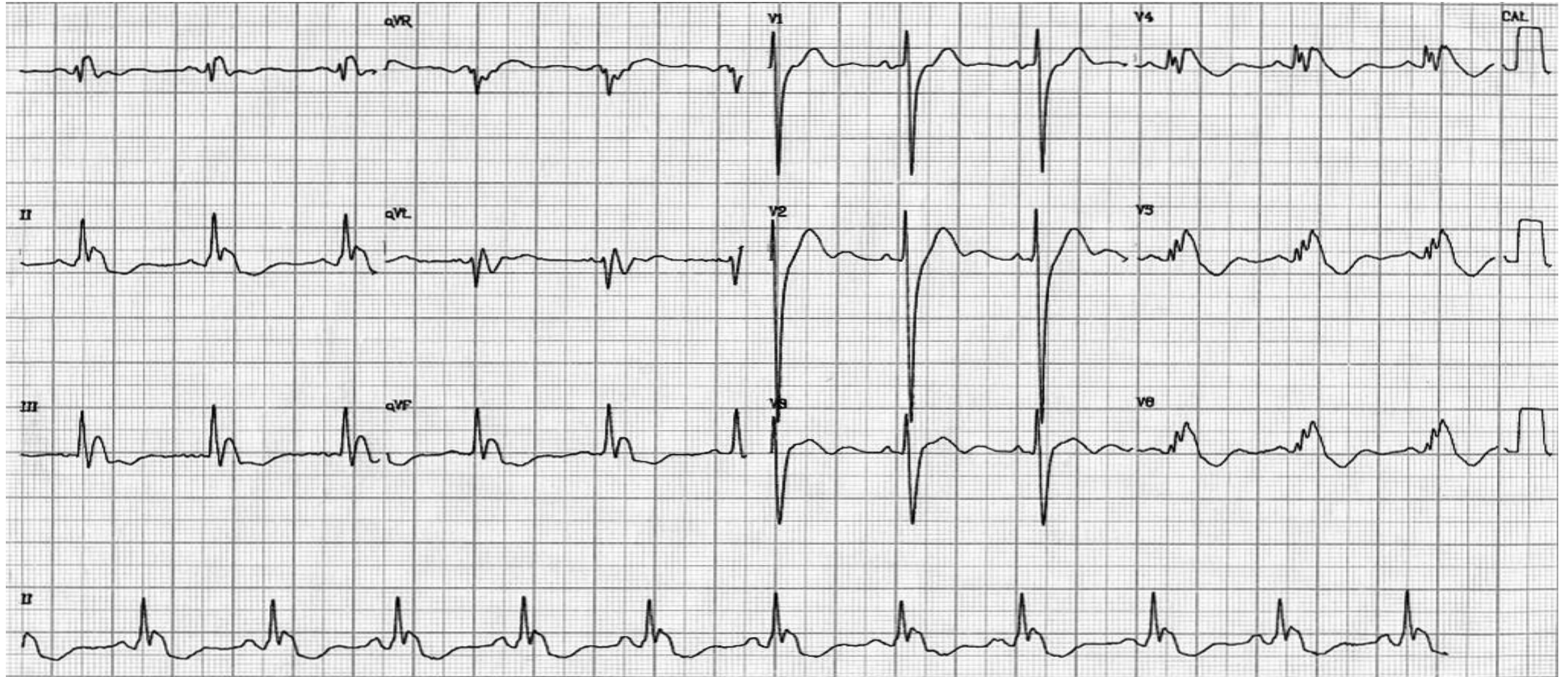
Female, 53-year-old patient, admitted in the Messejana Hospital on 29-04-2004, with STSEMI. She was immediately sent to hemodynamic laboratory. Coronarography showed severe lesion in the left main coronary artery (LMCA) (95%) + occlusion in RCA + LCx in 90%. She was sent to emergency surgery with placement of saphenous graft (LADA and LCx marginal). Echo showed: LV=70/54; LA=44; EF=45% + moderate mitral valve insufficiency.

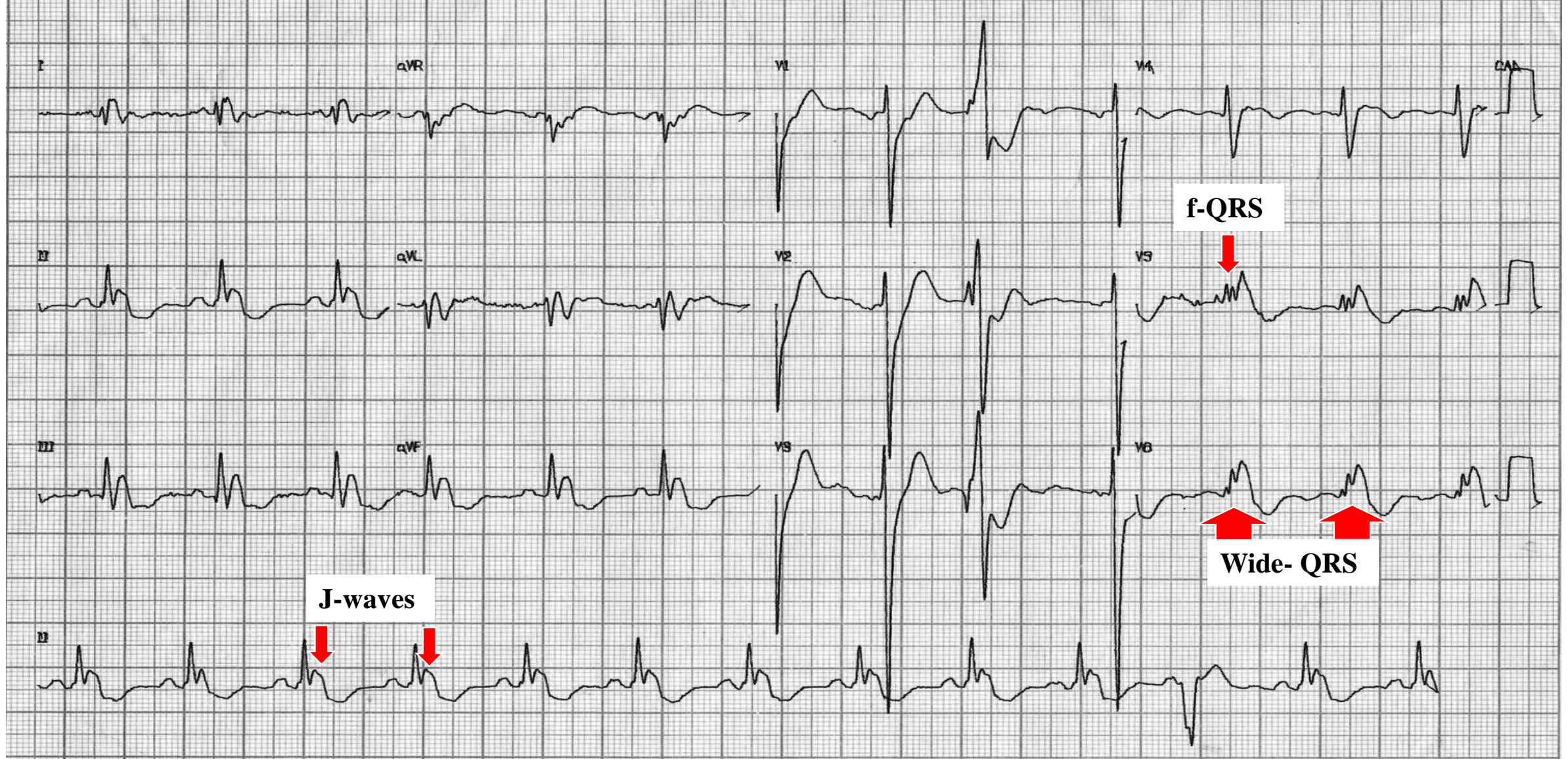
### ECG-1 performed on 2004



She was admitted in 2012 with ischemic cardiomyopathy, CHF functional class IV  
Left heart catheterization: aorto-coronary by-pass, all occluded. Severe systolic ventricular dysfunction (LVEF=30%).  
This patient was in functional class III in the infirmary, waiting for a heart transplant.  
She died suddenly.

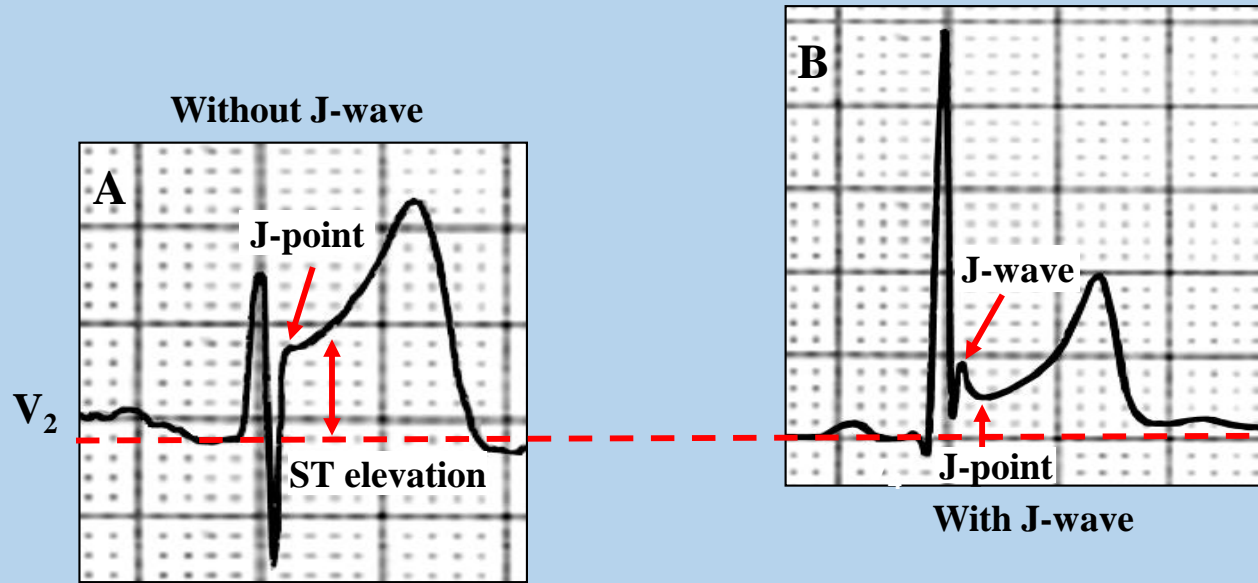
### ECG-1 performed on 2012



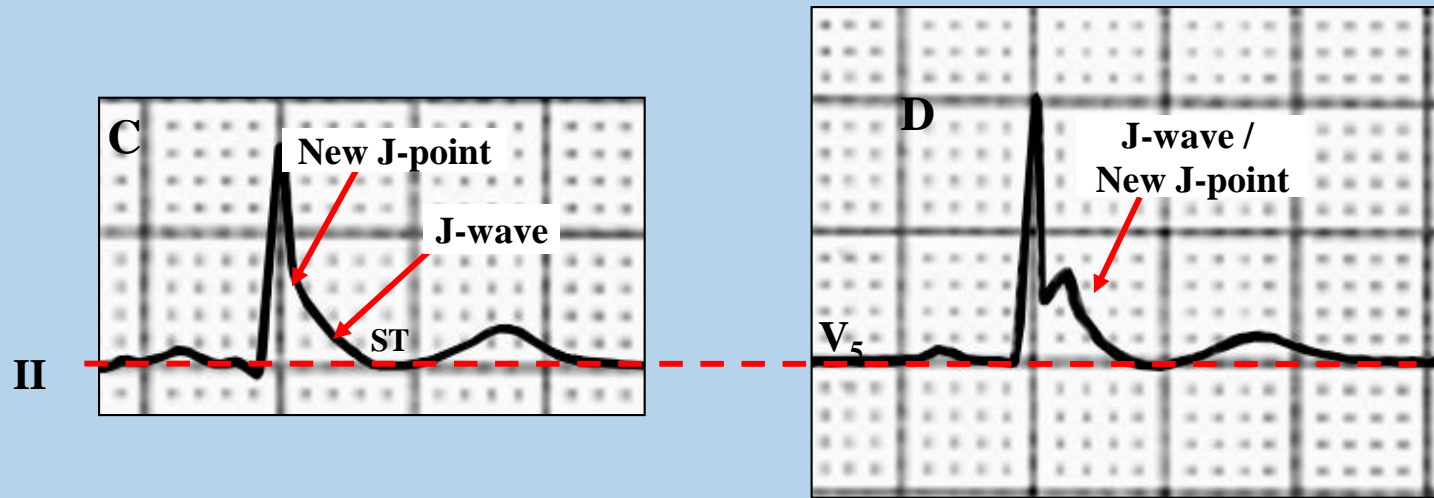


Association of f-QRS in at least two contiguous leads on the 12-lead ECG + Wide QRS complexes + J-waves  $\geq 0.1$  mV combined with a descending/horizontal ST segment constitute a malignant ER pattern (**Misuzawa 2014**). Identifying patients with higher risk of fatal arrhythmias after CABG surgery. All are components of multifactorial risk for increased morbidity and mortality, sudden cardiac death and recurrent cardiovascular events.

### Classical definition of early repolarization pattern: ST segment elevation



### New definition of early repolarization pattern: without ST segment elevation



Slurred QRS downstroke without STE

J-wave or the new "J-point elevation" without STE

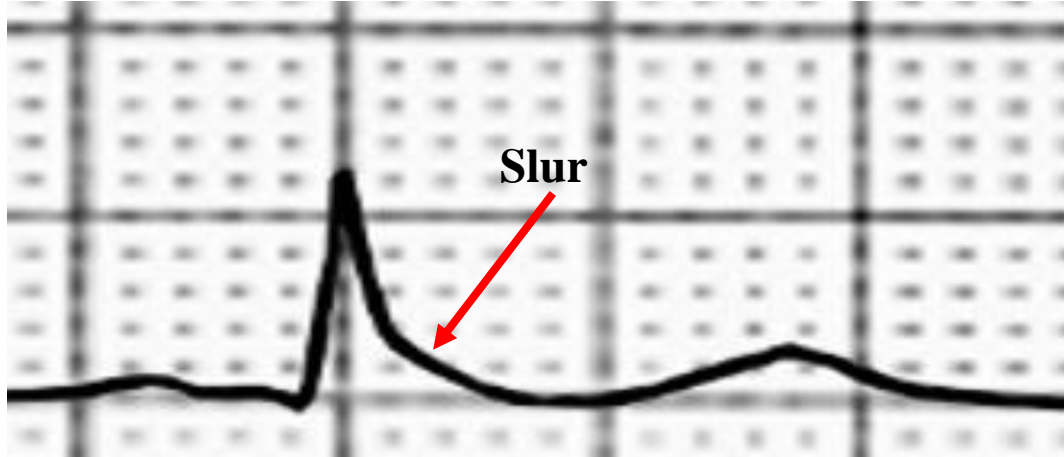
QRS slurring without STSE

QRS notching without STSE

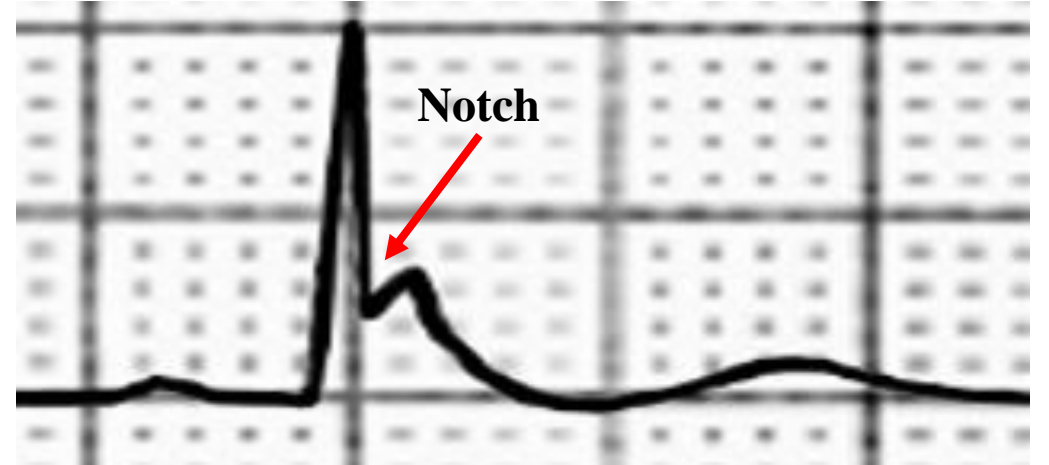


# New definition of Early Repolarization with terminal QRS slur/notch and without STSE

QRS slurring without STSE



QRS notching without STSE



## Malignant versus benign early repolarization pattern

	Malignant ERP	Benign ERP
Resuscitation from cardiac arrest or documented VF	Yes, very suggestive	Asymptomatic
Positive Family history for SCD in Young relative	Possible	Absent
Sinus bradycardia	No	It is the rule
Axes of QRS, ST segment and T wave	Frequently discordant	Oriented to the same direction
Mirror or reciprocal image	Frequently in several leads	Only aVR
Transient augmentation of J waves	Characteristic	Absent
Short coupled PVCs	Frequently	Absent
Co-existing channelopathies such as BsS, SQTS, idiopathic VF	Frequently	No
ST segment elevation	Frequently > 2 mm	Usually < 2 mm
Widespread J waves in inferior and lateral leads and/or globally across leads	Strong signal	No
J waves convex upward or lambda wave pattern	It is the rule	ST concave upward followed by T waves of great voltage and polarity matching QRS
J waves in the inferior leads	Also present	Possible
J waves in lateral leads, tall R waves, rapidly ascending ST segments	No	Characteristic

# Typical ECG of “benign” early repolarization pattern in an athlete with bradycardia

Name: DAS

Age: 24y

Sex: Male

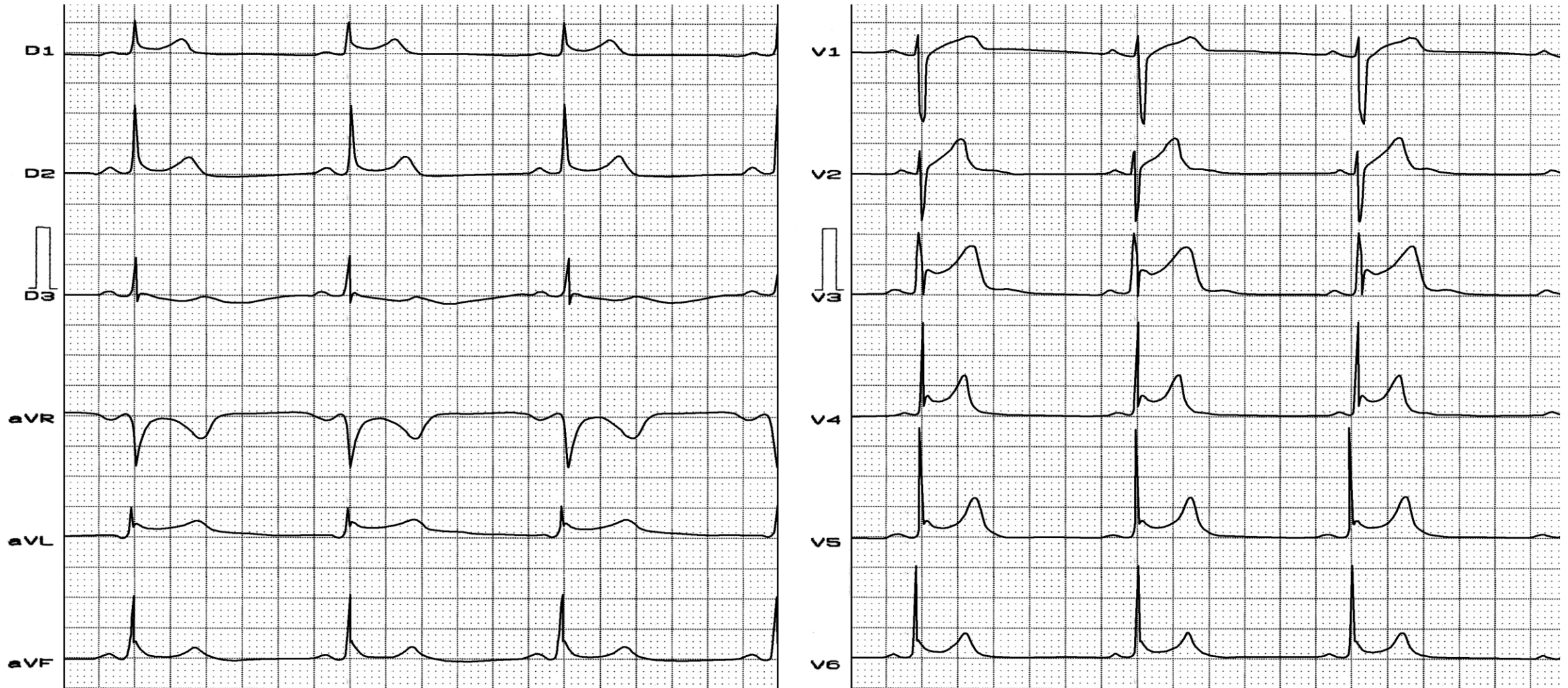
Race: Black

Weight: 82 kg

Height: 1.91 m

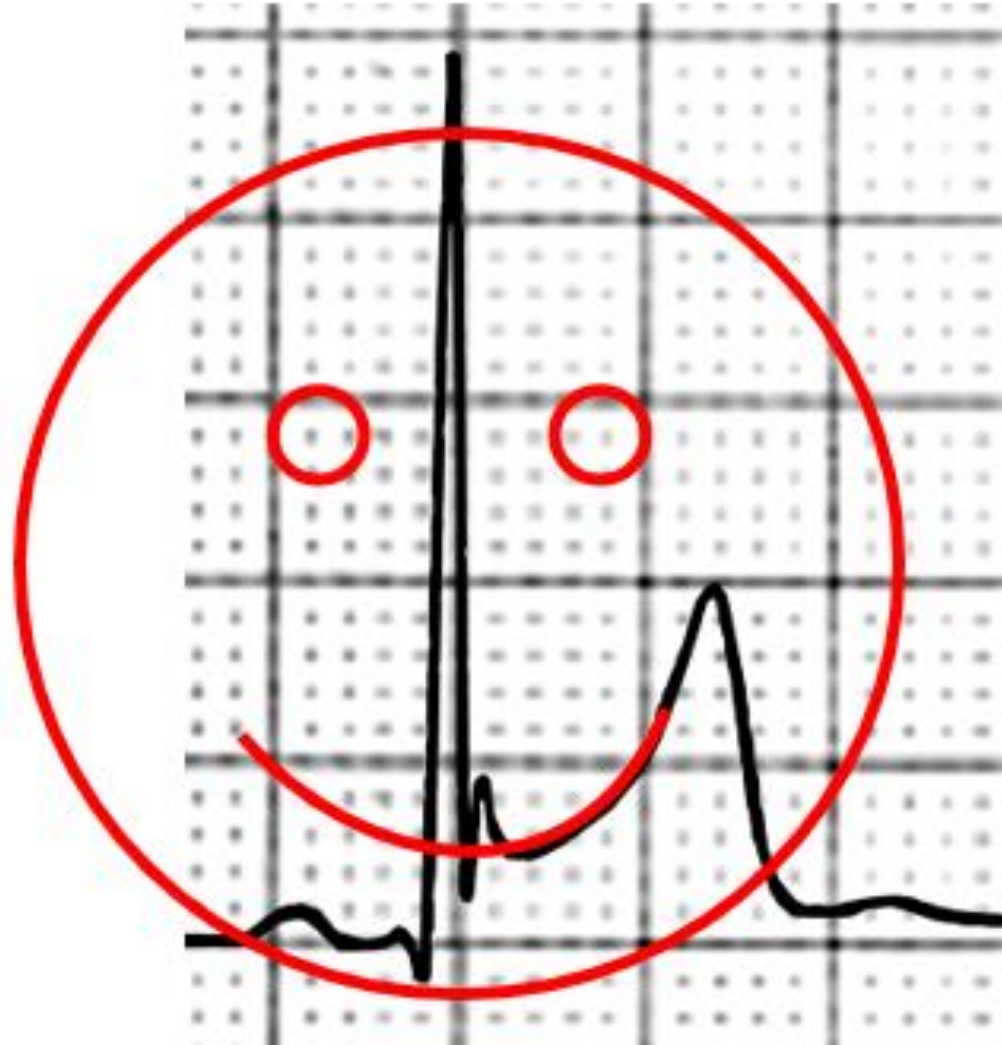
Biotype: Athletic

Profession: professional basketball player



**ECG diagnosis:** sinus bradycardia, (HR 50 bpm). J point and ST segment with elevation  $> 4$  mm in precordial leads from  $V_3$ - $V_5$  of superior concavity. Notch or slurring of terminal portion of the QRS complex (J point). ST segment elevation  $> 4$  mm in precordial leads  $V_3$ ,  $V_4$  and  $V_5$ .

**Conclusion:** sinus bradycardia, early repolarization syndrome.



The figure shows in lead V4 ST segment elevation with "superior concavity" followed by wide positive T wave that resembles a "smiling face".

# ECG characteristics of “benign” Early Repolarization Pattern

## I. Heart rate

Predominant sinus bradycardia frequently associated with phasic sinus arrhythmia. Resting sinus bradycardia is a very common feature among athletes (89%) and only in 36% of cases of sedentary age-matched healthy controls (**Bianco 2001**).

## II. PR interval

First degree AV block is observed in 5% to 39% among professional athletes. In the nonathlete population it is observed in 0.65% of cases. Shorter and depressed P-R interval is reported.

## III. QRS axis

Vertical electrical axis is the rule (**Mehta 1999**). The frontal plane QRS axis and ST segment axis and T wave axis are all in the same direction.

## IV. QRS duration

This parameter is higher in ERP ( $90 \pm 10$  ms) subjects than in age-matched healthy controls ( $80 \pm 10$  ms) (**Dilaveris 2004**). Contrarily, the mean QRS duration in BrS is  $110 \pm 2$  ms. It is higher than in individuals with ERP who present a mean QRS duration of  $90 \pm 10$  ms (up to 100 ms).

## V. QRS morphology

Notching or slurring of the terminal QRS complex (**Brady 1999**). A slur on the R wave. Prominent, relatively deep but narrow q waves may appear in the left precordial leads.

## VI. QRS voltage

Associated R waves are usually tall. Eventually voltage criteria of left ventricular hypertrophy are observed in male competitive athletes:  $SV1 + RV5 > 35$  mm (Sokolow index). High QRS voltage is more frequent in male athletes, but its correlation with left ventricular hypertrophy is low. Voltage decreases after deconditioning is slow. The distinction between the physiological athlete's heart and pathological conditions has critical implications for professional athletes. Criteria and guidelines for screening of athletes in competitive sports are recommended (**Chee 2005**). An abrupt transition may occur from right-oriented complexes to left-oriented complexes in the precordial leads, secondary to counterclockwise rotation on precordial leads. About two-thirds of clockwise rotation and counterclockwise rotation could be attributed to the septal angle by anatomical rotation of the heart in one plane around the long axis, but other factors appear to be responsible for such ECG findings in the remaining one-third of cases. Relatively higher positions of the precordial ECG leads, as observed in the vertical heart, appeared to be responsible for clockwise rotation in some patients, and left septal fascicular block is suspected to be responsible for counterclockwise rotation in others patients (**Tahara 1991**).

## VII. J-point elevation

Notching, irregular or slurring contour of the terminal QRS complex (J point). Variant of Osborn wave is seen in ERP. The Osborn wave arises from the R down stroke, frequently in V2 to V6.

## VIII. ST segment

Widespread ST segment elevation (precordial greater than limb leads). The characteristic ST segment is elevated, upward, concave, confined more frequently in precordial leads, with reciprocal depression only in aVR. The concavity is observed at the initial up-sloping portion of ST segment or upwardly concave ST segment morphology. Unfortunately, concave ST morphology cannot be used to rule out ST elevation from acute myocardial infarction (AMI) with left anterior descending coronary occlusion because it is common in these circumstances (**Smith 2006**). The ST elevation is most frequently evident in ECG lead V4. There is a distinct J wave and ST segment in the left precordial leads V4 through V6. The ST elevation in ERP is usually  $< 2$  mm (but can rarely be  $> 5$  mm) in the precordial leads and the greatest ST elevation is usually seen in the mid-to-left precordial leads. The ST segment elevation is usually  $< 0.5$  mm in the limb leads. T wave characteristics. Concordant T waves of large amplitude (prominent, matching T waves), typical pseudo-asymmetrical ("symmetroid") or slightly asymmetrical, matching T waves often of large amplitude, upright, tall and peaked, most conspicuously from V2 to V4 or V5, sometimes seen in leads II, III and aVF as a rule. T waves may appear as of large amplitude, "peaked" or pointed, symmetric and matching. Vagotonic or high T wave voltages followed by U waves are frequent when sinus bradycardia is present. Tall, positive and symmetric or symmetroid T waves are not only seen occasionally in the very early stages of MI, but also in hyperkalemia and in ERP sinus bradycardia.

## IX. QT intervals

- **QT maximum:** The maximum Q-onset-T-end interval. This parameter is higher in ERP subjects than in normal controls (**Dilaveris 2004**).
- **QTp maximum:** Maximum Q-onset-T-peak interval. This parameter is higher in ERP subjects than in age-matched healthy controls (11.3).
- **Rate-corrected QTc maximum:** This parameter is lower in ERP subjects than in age-matched healthy controls.
- **QTpc maximum:** This parameter is lower in ERP subjects than in age-matched healthy controls.
- **U wave:** Because bradycardia U waves are frequent, in ERP they are best observed in the V3 lead. U waves are frequent when sinus bradycardia is present.

## **Other ECG characteristics of ERP**

Relative temporal stability of the ST segment and T wave pattern is observed. Reciprocal changes are not seen in ERP. There are no evolutionary short-term changes in the ST segment and T waves; and Q waves do not appear.

## **Treadmill stress ECG**

Exercise and isoproterenol tend to normalize the ST segment elevation. A progressive decrease in ST segment elevation and normalization of the ECG with mild exercise was a predictable response in patients who had ERP with otherwise normal ECGs and no history of cardiovascular disease. This phenomenon may be clinically useful as a bedside diagnostic tool in evaluating patients who present with ERP, otherwise normal ECGs, and negative cardiac history, in which the diagnosis of acute myocardial infarction/injury or pericarditis is being considered. These findings warrant formal evaluation in a prospective clinical trial (**Spratt 1995**). The presence of ERP does not preclude diagnosis of exercise-induced myocardial ischemia by treadmill testing, and coronary vaso-occlusive lesions may be demonstrated in some patients with the ERP pattern on ECG.

## **Vectorcardiographic features in ERP**

- VCG markers of spatial T amplitude: This parameter is higher in ERP subjects than in age-matched healthy controls.
- Spatial QRS amplitude: This parameter is higher in ERP subjects than in age-matched healthy controls.
- Spatial QRS-T angle: This parameter is higher in ERP subjects than in age-matched healthy controls.
- A stable ECG pattern is a very important feature for differential diagnosis with AMI and pericarditis (both have dynamic ECG changes) (**Turnipseed 2006**).

# “Malign” early repolarization pattern





Shortly after occlusion of a coronary artery, serial ECG changes are detected by leads facing the ischemic zone (**Birnbaum2003**):

- I) First: Tall, symmetrical and peaked T-waves the T waves (grade I ischemia Sclarovsky-Birnbaum);
- II) Second: ST segment elevation (grade II ischemia), without distortion of the terminal portion of the QRS;
- III) Third, changes in the QRS complex:
  1. Transient development of very tall R waves: the “giant R waves” or Prominent Anterior QRS Forces (PAF) and concomitant reduction or complete disappearance of S waves. (**Holland 1976; David1982; Madias 1993**) observed very early in the clinical course in the hyperacute phase of myocardial ischemia or myocardial infarction (MI). Additionally, have been described during treadmill stress testing (**Grosse-Heitmeyer 1993**), episodes of Prinzmetal angina (**Feldman 1986**) and during brief episodes of transmural ischemia induced by first balloon occlusion (**Sinno 2008**). The regional or intramyocardial conduction delay conduction (**Holland 1976; David 1982; Spekhorst 1990**) decreases the degree of cancellation, resulting in an increase in R wave amplitude in leads with terminal R wave and decrease in the S wave amplitude in leads with terminal S wave on the surface ECG (**Wagner 1988; Barnhill 1989**). We think that Prominent QRS Anterior Forces (PAF) are sometimes consequence of transient left septal fascicular block (LSFB). This is the electro-vectorcardiographic expression of proximal obstruction of Left Anterior Descending Artery (**Pérez-Riera 2016b**) See discussion in the next slide.
  2. Changes in the terminal portion of the QRS complex (grade III ischemia) (**Sclarovsky 1989; Sclarovsky 1990; Sclarovsky 1999; Birnbaum 1999**) explained by prolongation of the electrical conduction in the Purkinje fibers in the ischemic region (**Holland 1976; David1982**). The Purkinje fibers are less sensitive to ischemia than the contracting myocytes (**DeHann1961; Schiebler 1956**). Hence, for an alteration in the terminal portion of the QRS to occur, there should probably be a severe and prolonged ischemia that would affect the Purkinje fibers (**Feldman 1986; Holland1976**). In the presence of collateral circulation no changes are detected in the QRS complex during balloon angioplasty (**Spekhorst1990**). Thus, absence of distortion of the terminal portion of the QRS, despite prolonged ischemia, is a sign for myocardial protection (probably by persistent myocardial flow due to subtotal occlusion or collateral circulation, or due to myocardial preconditioning). Disappearance of the S waves in leads with terminal S (Rs configuration) (mainly leads V1–V3) is and hallmark of this situation.

## Discussion

The ECG/VCG phenomena of PAF. In this case increase in the amplitude of the R wave in right precordial leads from V1 to V3. The presence of PAF in ECG occurs when the voltage of the R wave in any precordial lead of the anterior or anteroseptal wall from V<sub>1</sub> (+115°) through V<sub>4</sub> (+47°)

is greater than the normal maximal limit for gender and age. ECG/VCG criteria of PAF should be age and gender-related. PAF should be distinguished from other conditions that also produce anterior QRS shift in the HP as: normal variants (athlete heart and CCW rotation around longitudinal axis), RVH (types B and A VCG), misplaced precordial leads, lateral MI (old dorsal), RBBB, type A Wolff-Parkinson-White preexcitation (type A has a positive delta wave and QRS complexes are predominantly upright in the precordial leads with  $R/S > 1$  in  $V_1$ ). The dominant R wave in lead V1 may be misinterpreted as RBBB, obstructive and non-obstructive forms of HCM, diastolic LVH, combined or biventricular hypertrophy, endomyocardial fibrosis, Duchenne muscular dystrophy, dextroposition or pseudo dextrocardia, and **left septal fascicular block (LSFB)** or a combination of the above. The two highly frequent etiologies of LSFB are: **coronary artery disease (CAD)** with critical proximal obstruction of the LAD before the first septal perforating branch( $S_1$ ), and in Latin America, chronic Chagasic myocarditis. Literature data and our own experience have identified the following etiological causes for LSFB: Chronic Chagas Cardiomyopathy. Predominant in Latin America countries (**Moffa 1982; Vichi 1982; Moffa 2001**); Coronary Artery Disease (CAD): critical lesion of LAD and/or its septal perforating branches before the first septal ( $S_1$ ) (**Hilsenrath 1971; Nakaya 1978; Tranchesi 1979; Hassett 1980; Bateman 1982; Feldman 1986; Selvester 1988; Moffa 1997**); coronary artery disease with Wellens' syndrome (**Riera 2008a**); Non-Obstructive Hypertrophic Cardiomyopathy (NO-HCM) (**Chen 1979**); Obstructive Hypertrophic Cardiomyopathy (O-HCM); Aortic valve disease (**Pérez-Riera 2015**); Diabetes Mellitus (**Magnacca 1988**). Magnacca M. et al described the presence of LSFB pattern in ECGs of patients with diabetic mellitus. The authors attribute the dromotropic disorder to the involvement of the IVS microcirculation; Papillary Muscle Dysfunction (**Nakaya 1978**): In a case of papillary muscle dysfunction, where the patient died suddenly, the septal fascicle of the left bundle was found to be markedly fibrotic by serial histological examination of the intraventricular conduction system. A case of angina pectoris with PAF later developed anteroseptal myocardial infarction. Ischemia of the anterior wall of the left ventricle might cause prominence or absence of the anterior QRS force in VCG. The prominence may be explained by LSFB, and the absence may be caused by the loss of the electromotive forces of the heart due to myocardial necrosis. Intermittent PAF was also observed, which was an important finding to support the existence of this new type of fascicular block; Kearns-Sayre syndrome (**Riera 2008b**). The ECG/VCG hallmark of the LSFB is prominent anterior QRS forces (PAF) on the horizontal plane. Proof of transient LSFB, as part of the requisites to recognize a new ECG dromotropic disturbance, is considered mandatory. There were few cases of transient ischemic LSFB in literature (associated with proximal obstruction of the LAD coronary artery before the  $S_1$  or ischemia triggered during an exercise stress test), additionally we described a phase-4 rate-dependent or bradycardia-dependent mechanism of LSFB. Rate dependent blocks are the best models to study any new conduction disturbances in the conducting tissue, as they are free from possible "contaminants" associated with transient injuries to the surrounding tissue, as it happens in ischemia (**Ibarrola 2014**).

**In the next slides we will show four undeniable examples of LSFb in the context of CAD and other miscellaneous entity**



**Andrés Ricardo Pérez-Riera**



**Raimundo Barbosa-Barros**

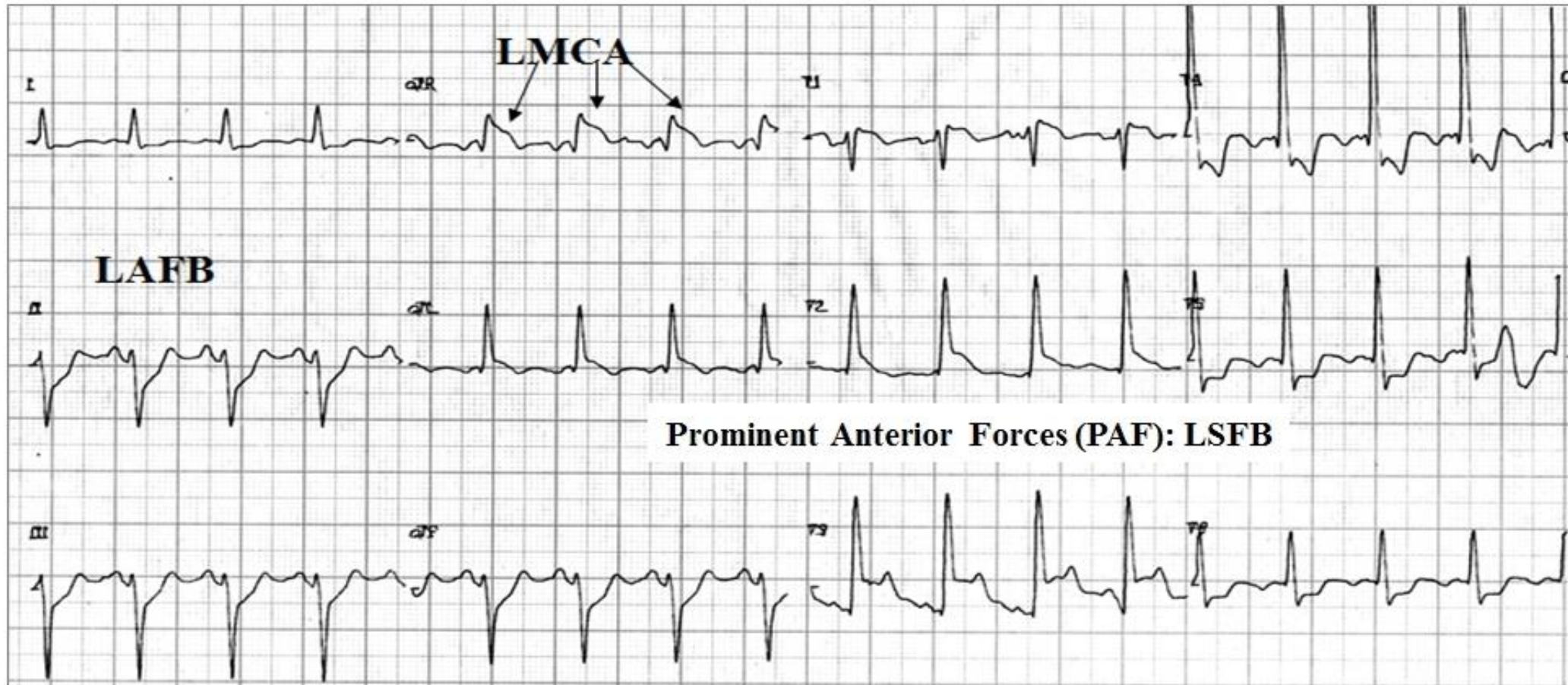


**Adrian Baranchuk**

## I. ECG of LMCA obstruction associated with LAFB and LSFB

Clinical features: ACS: 72-year-old male patient, admitted in the emergency room with typical precordial pain that yielded after the administration of IV nitroglycerin. Coronary angiography revealed LMCA spasm + proximal critical lesion of the LAD. Management: the patient was urgently revascularized, successfully (Coronary Artery Bypass Graft).

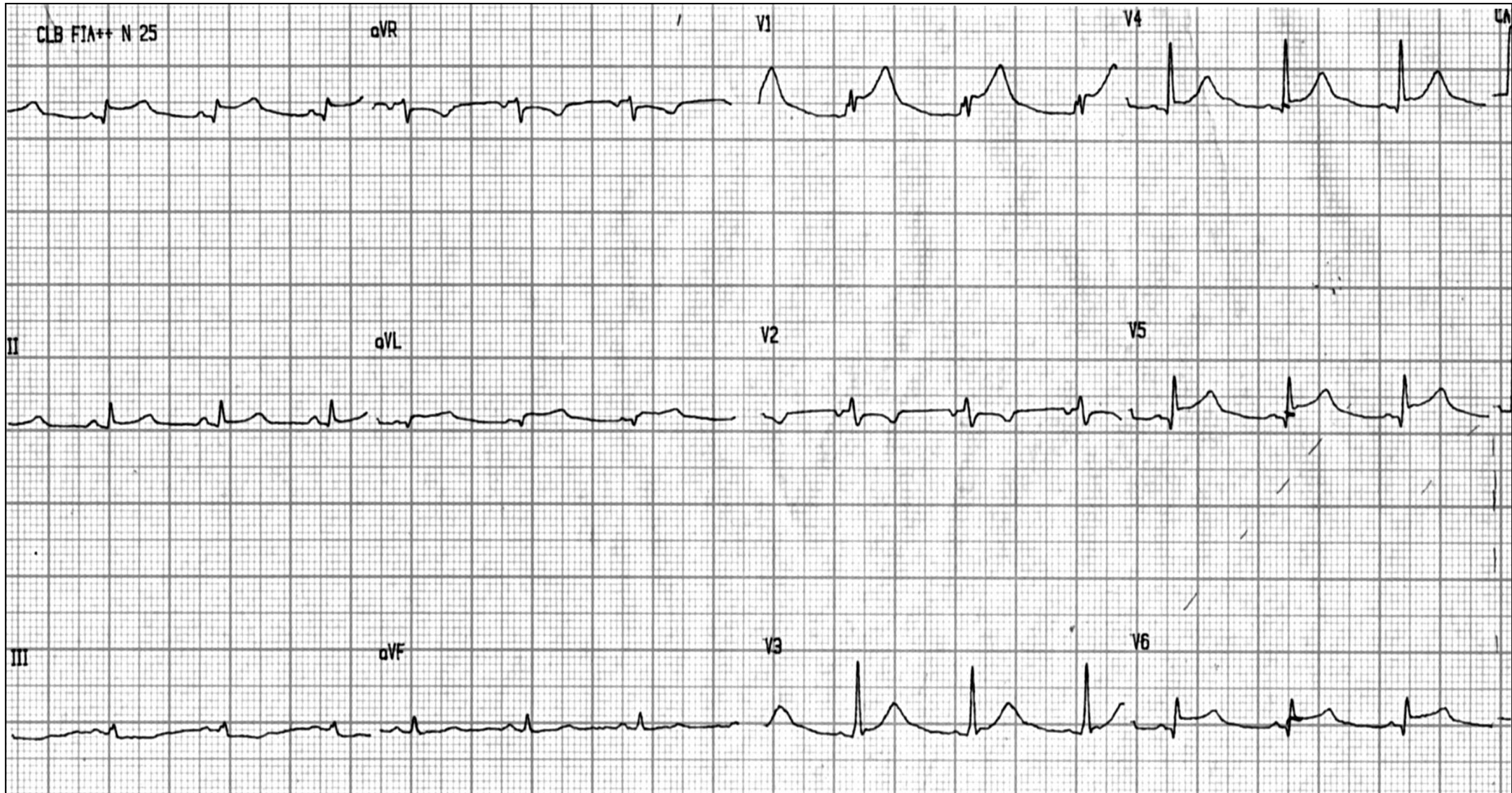
**ECG at admission - Name:** AR.; **Date:** 02/01/2009.; **Age:** 72 y/o; **Gender:** Male; **Ethnic Group:** Caucasian; **Weight:** 72 Kg.; **Height:** 1.74 m; **Biotype:** Mesomorphic



**ECG diagnosis:** 1) LAFB + 2) LSFB: PAF + Injury block + aVR lead with ST segment elevation suggestive of obstruction in the LMCA. **Laboratory:** There was no increase of necrosis markers (CK-MB/troponin).

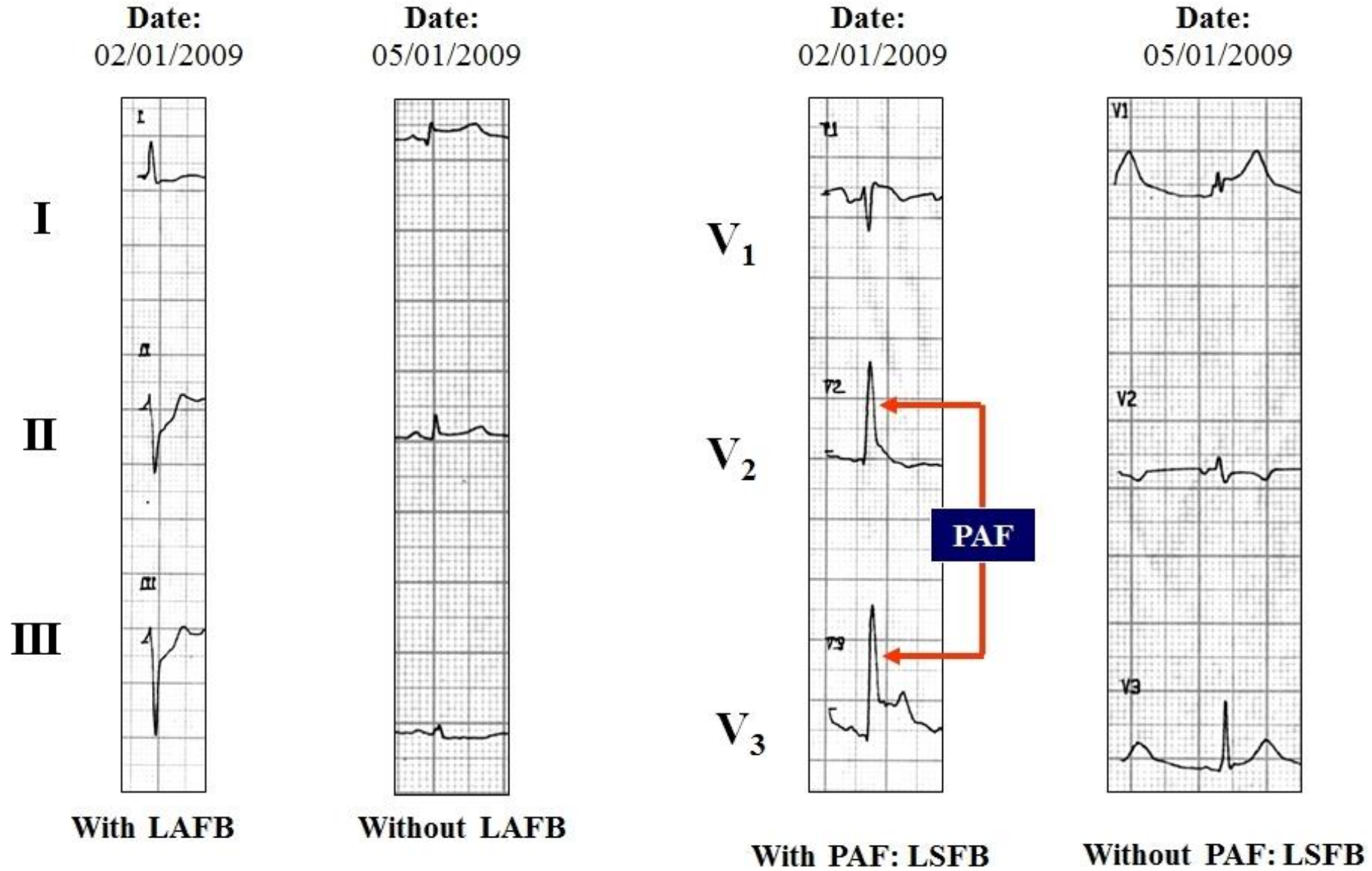
# ECG performed on the third day after successful Coronary Artery Bypass Graft

**Name:** AR; **Date:** 05/01/2009; **Age:** 72 y/o; **Gender:** Male; **Ethnic:** Caucasian; **Weight:** 72 Kg; **Height:** 1.74 m; **Biotype:** Mesomorphic; **Management:** Coronary Artery Bypass Graft (CABG) 72 hours before.



**ECG diagnosis:** Both fascicular and divisional blocks have disappeared; the extreme shift of QRS electric axis to the left in the frontal plane (LAFB) is not seen, and prominent anterior forces (LSFB) disappeared.

# Comparison among inferior and right precordial leads with/without left bifascicular block (LAFB + LSFB)



PAF: Prominent Anterior Forces

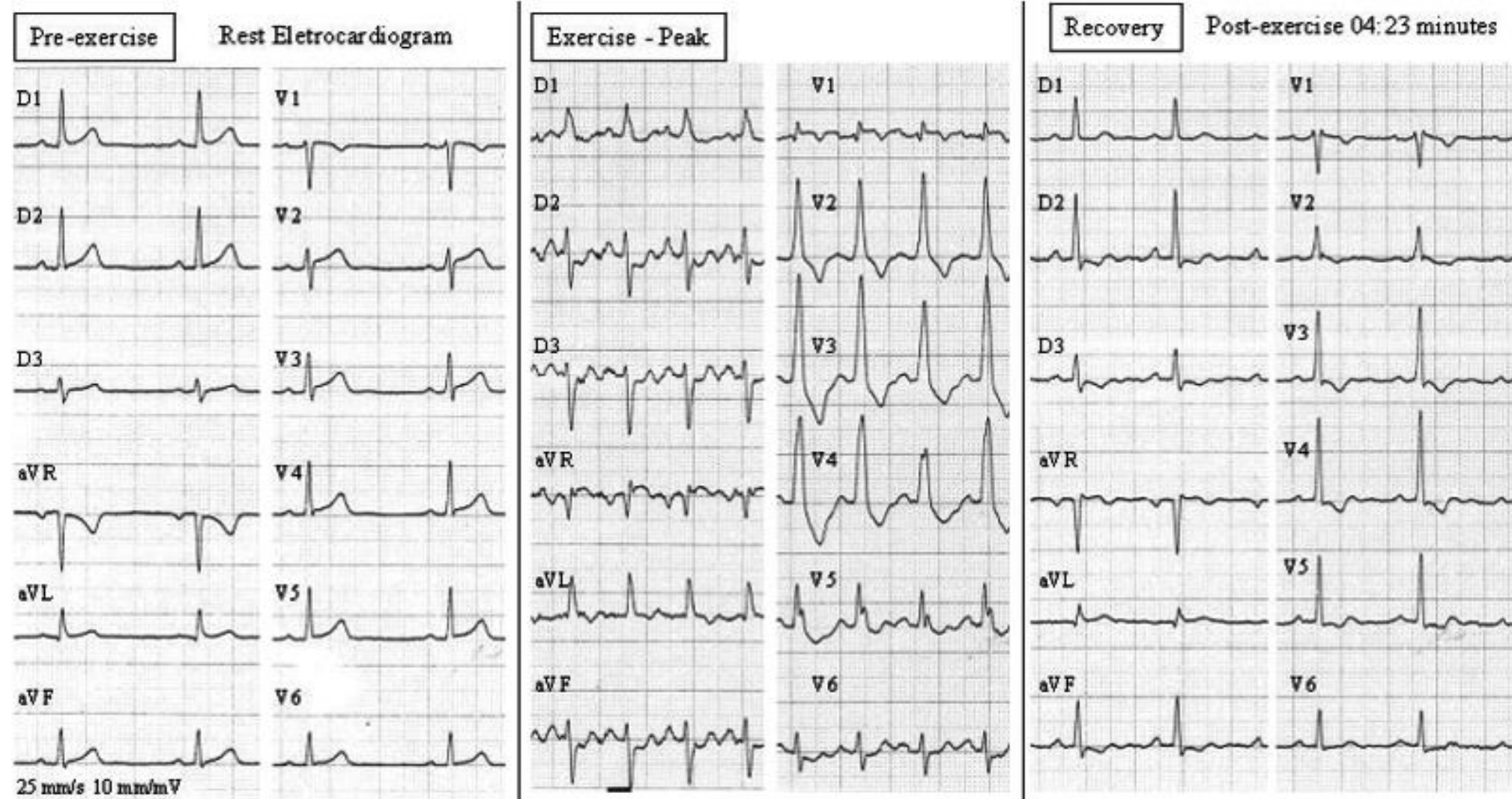
## Comments about evidence that reinforces the trifascicular nature of the Left His System

The following evidence and arguments reinforce the trifascicular theory of the left intraventricular His system, unlike the classical bifascicular concept, coined in a conclusive way from the work by Rosenbaum and his associates ([Rosenbaum 1968; 1969; 1969; 1970; 1972; 1973; Elizari 1972; 2007; 2012; Aravindakshan 1970; Halpern 1971; Cola 1992](#)).

The electrophysiological demonstration of the activation of the middle third of the left septal surface 5 ms before the antero-superior and postero-inferior regions, was made in 1970 by Durrer and his colleagues ([Durrer 1970](#)). In Dr. Durrer's studies, the activation of the middle-septal region occurs in most cases from the anterior "false tendons" that originate from the LSF. According to the authors, the LSF in its final portion opens as a fan, and the anterior pseudo-tendons are those resulting in activation of the middle-septal region.

We know that one of the anatomical variations of the LSF (type 3) is precisely the one that depends on the LPF (2.4% of cases). Another piece of evidence is determined by the electrical or electrovectorcardiographic recording of the anterior ventricular depolarization, sometimes intermittent or transitory, translated in ECG by R waves with great voltage in intermediary precordial leads V3 and V4. This is represented on the VCG by an anterior and leftward shift of a QRS loop in the HP. This may occur in association with a critical proximal lesion of the LAD before S1. It also requires the absence of other factors capable of causing prominent anterior forces (PAF), such as dorsal or posterior wall infarction, RVH, cardiomyopathies, RBBB, WPW, to name a few. In some patients studied angiographically, coronary disease was isolated to the LAD and ventricular dysfunction confined to the LV anterior wall. PAF was observed intermittently with LAFB. These observations, in addition to serial studies following surgery, strongly suggest that the mechanism for PAF in these cases is conduction delay in the LSF ([Moffa 1997; Uchida 2006](#)). We presented a 54-year-old male, with a history of systemic hypertension, hyperlipidemia and typical chest pain during exercise. During the exercise stress test, ECG demonstrated abrupt prominent anterior forces, an increase in R wave amplitude from V1 to V4, extreme left axis deviation, and minor ST segment depression in II, III and aVF. The post-exercise period showed progressive return of the QRS axis in both the frontal and horizontal planes, and the ST depression worsened by 1 mm. Coronary angiograms showed a critical proximal LAD critical occlusion.

## II. Demonstration of transient bifascicular block (LAFB + LSFB) during exercise testing



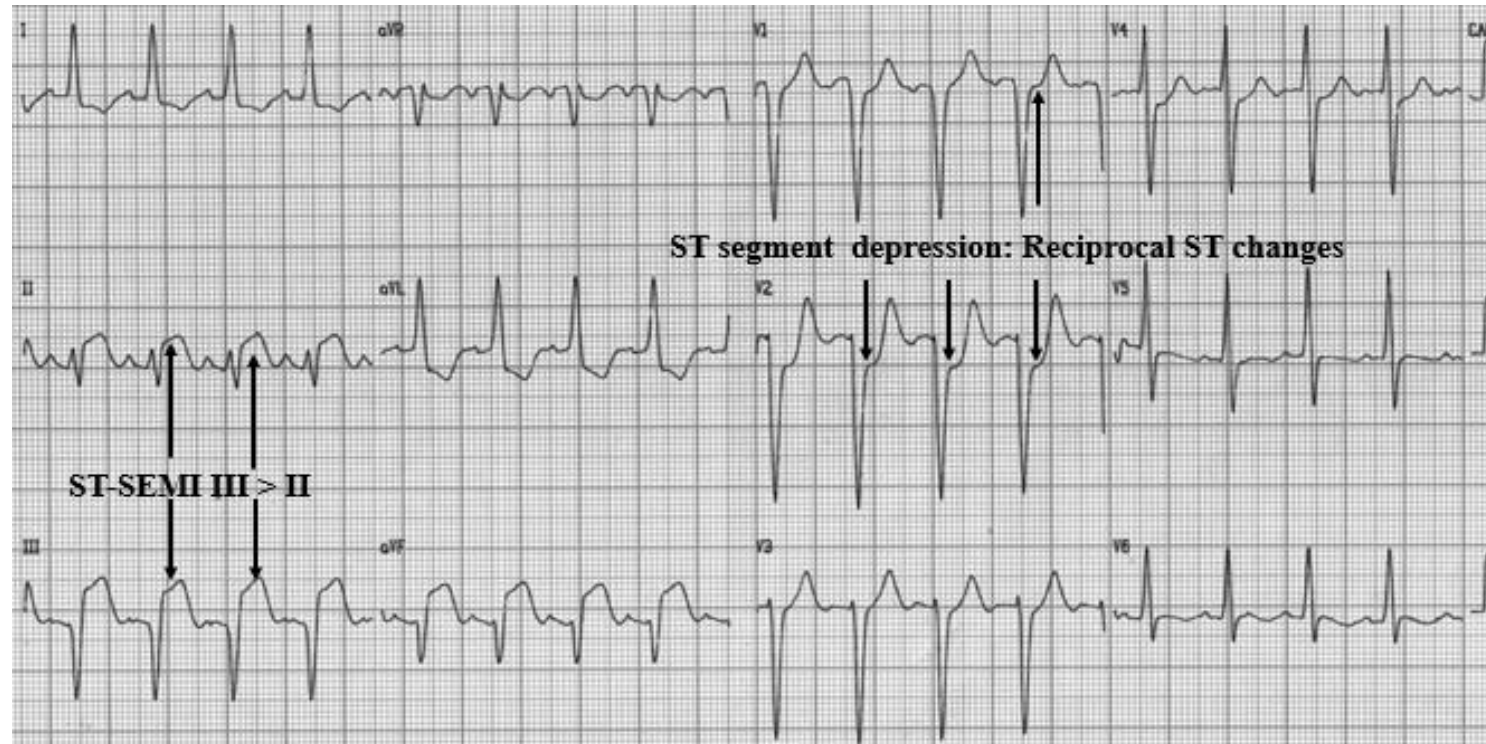
During peak of exercise we observe tall R waves in V1-V4. It is a hallmark ECG finding in LSFB. The proposed ECG criteria for LSFB are: prominent R waves in V1-V3, minimal QRS prolongation (QRS < 120 ms), T wave morphologic alteration (flat or inversion), frequent initial embryonic q wave in right and/or middle precordial leads, absence of initial q wave in left leads consequence of absence of  $I_{AM}$  vector, and clinical absence of other causes of prominent anterior forces. During exercise peak we also observe LAFB, demonstrating left bifascicular block (LAFB + LSFB).



### III. Transient Left Septal Fascicular Block: A hallmark expression of critical proximal obstruction of LAD

**Case Report** Female, 78 years old, Caucasian patient, who was admitted to the emergency room with typical clinical-electrocardiographic image of ST-segment elevation acute coronary syndrome (STE-ACS). A standard 12-lead ECG was performed at admission within the first 12 hours of chest pain having started. **ECG1**. Immediately, the patient was submitted to primary Percutaneous Transluminal Coronary Angioplasty (PTCA) with successful stent placement in proximal RCA. Additionally, the angiocoronariography shows critical proximal obstruction of the LAD before the first septal perforator ( $S_1$ ). The strategy was to carry out an elective angioplasty in a second time. A few days later, the patient returned with a typical episode of effort-induced precordial pain. **ECG2**. An ECG was conducted on the third day after the successful PTCA of the LAD. **ECG3**.

**ECG1**. Standard 12-lead ECG performed at admission within the first 12 hours of chest precordial pain onset.



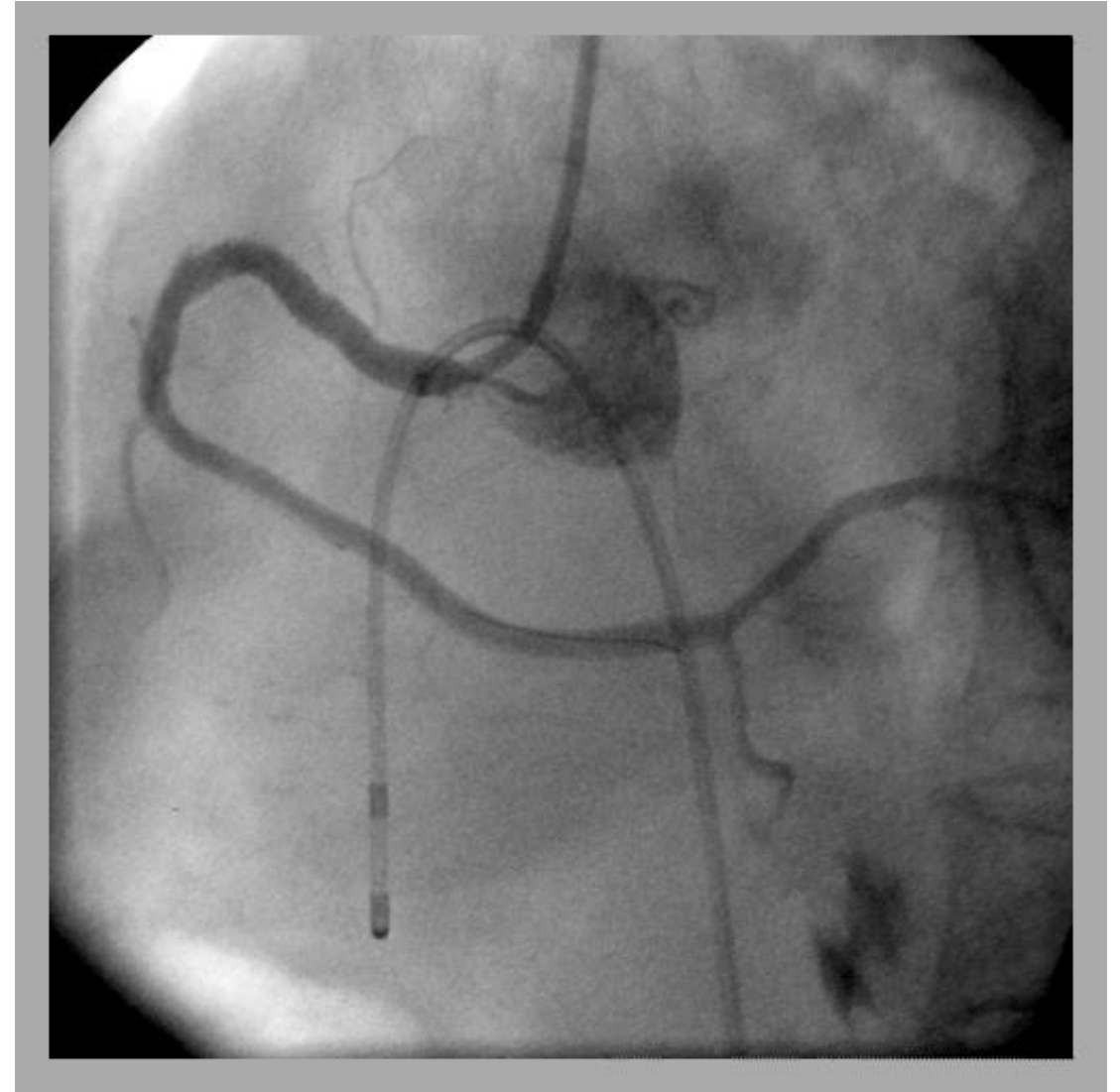
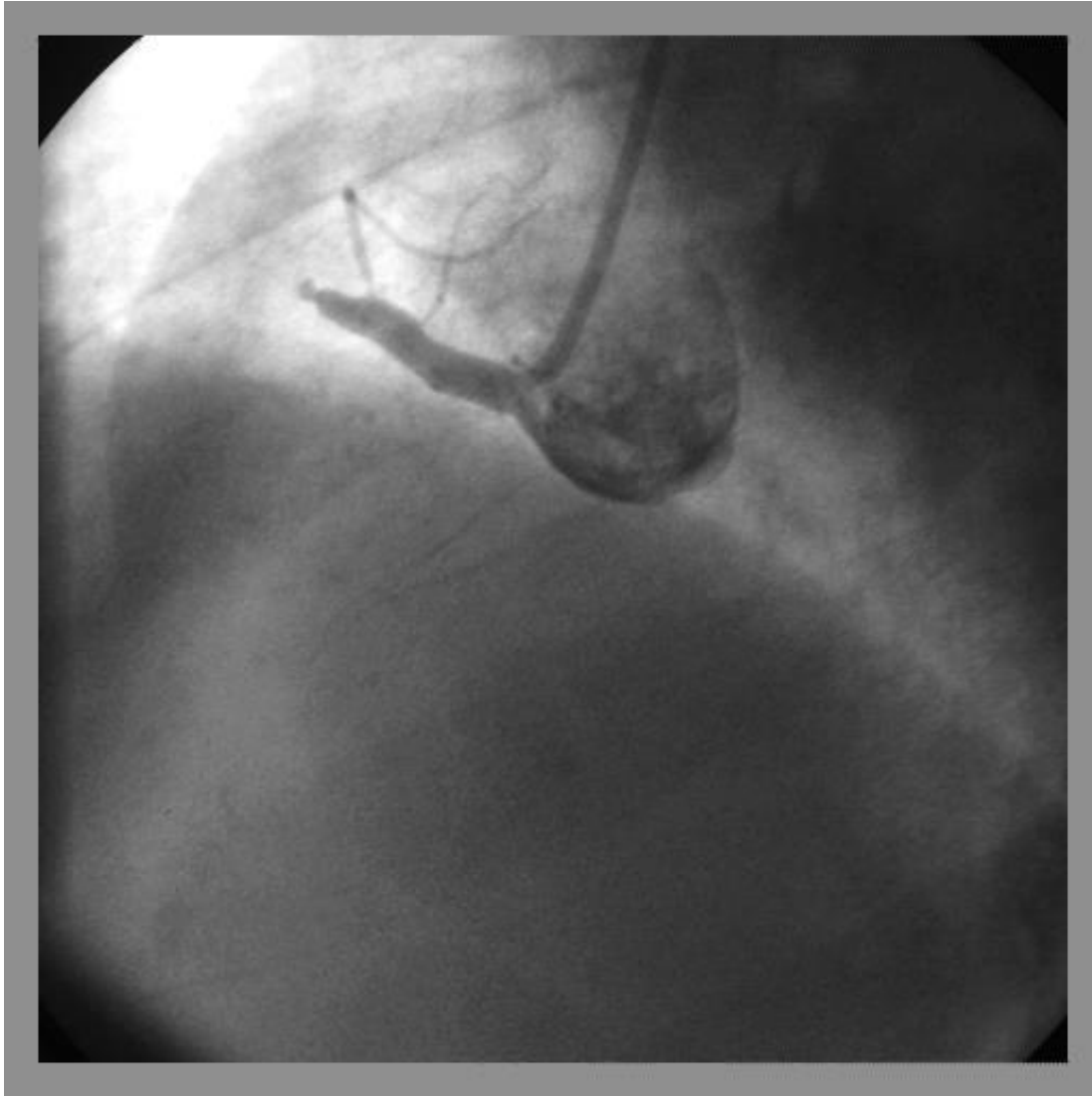
**ECG diagnosis:** sinus tachycardia (HR125 bpm), QRS axis  $-30^\circ$ : minimal degree of LAFB?), pathological QS waves in III and aVF followed by upwardly convex ST segment elevation  $\geq 1$  mm (inferior injury). ST segment elevation in lead III $>$ II is suggestive of RCA occlusion. Additionally, ST segment depression is observed in V1-V2. These represent reciprocal ST changes (mirror image) in leads remote

**Conclusion:** sinus tachycardia and inferior acute myocardial infarction consequence of RCA occlusion.

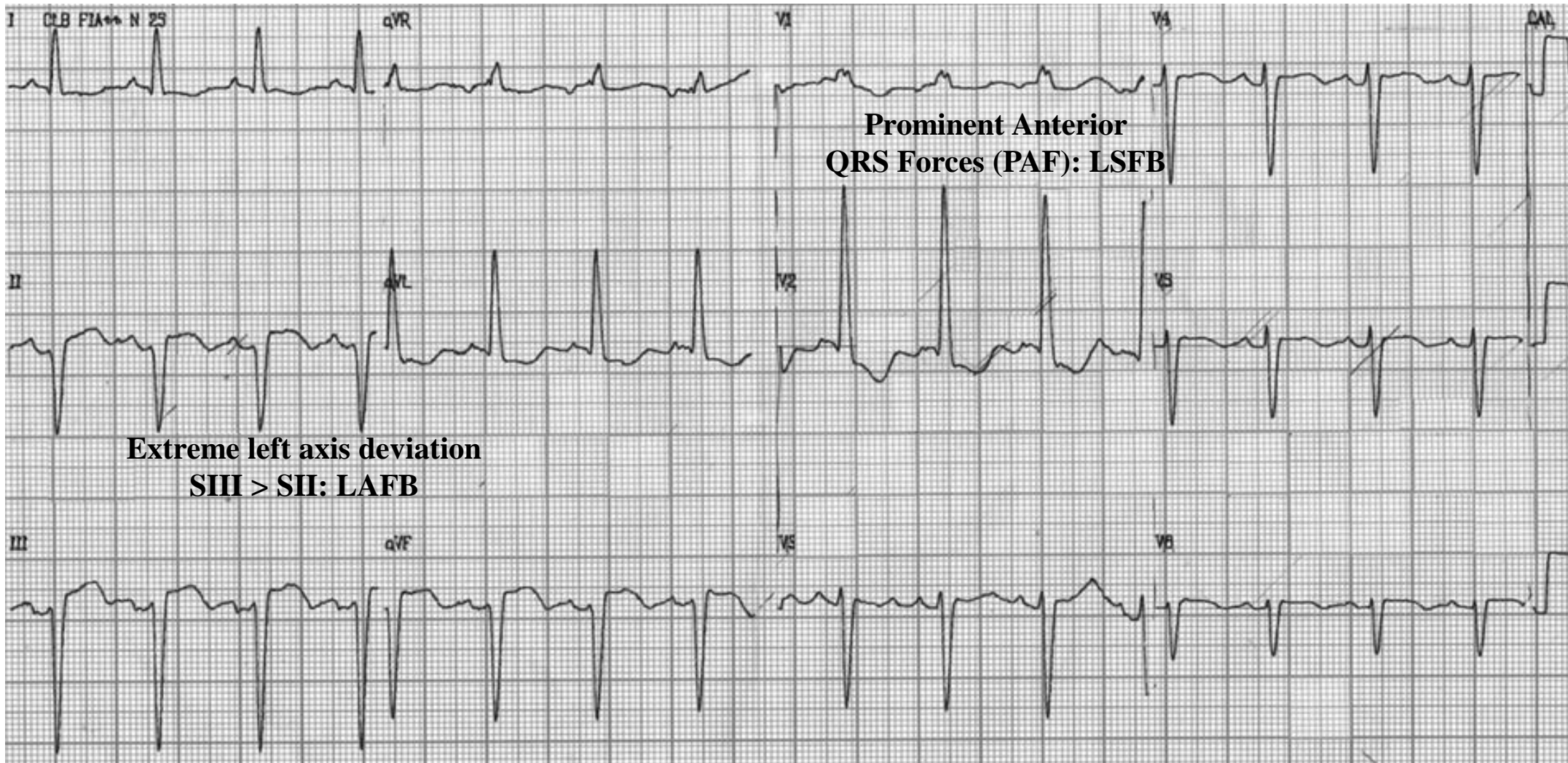
# Before and after Primary Percutaneous Transluminal Coronary Angioplasty (PTCA) in RCA

**Before**

**After**

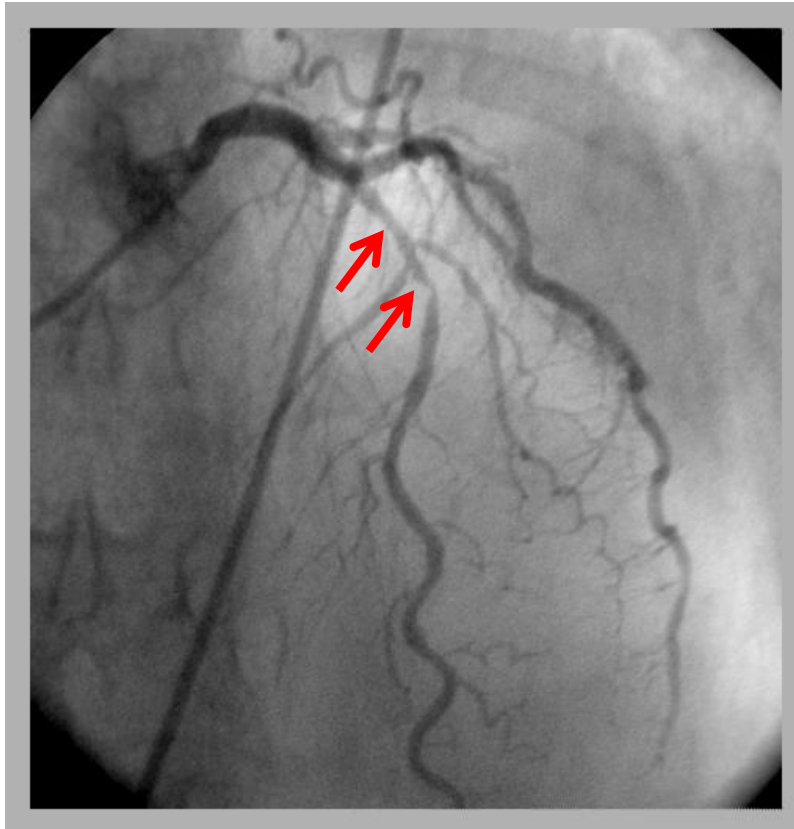


## ECG2. Performed immediately after effort-induced episode of typical angor pectoris

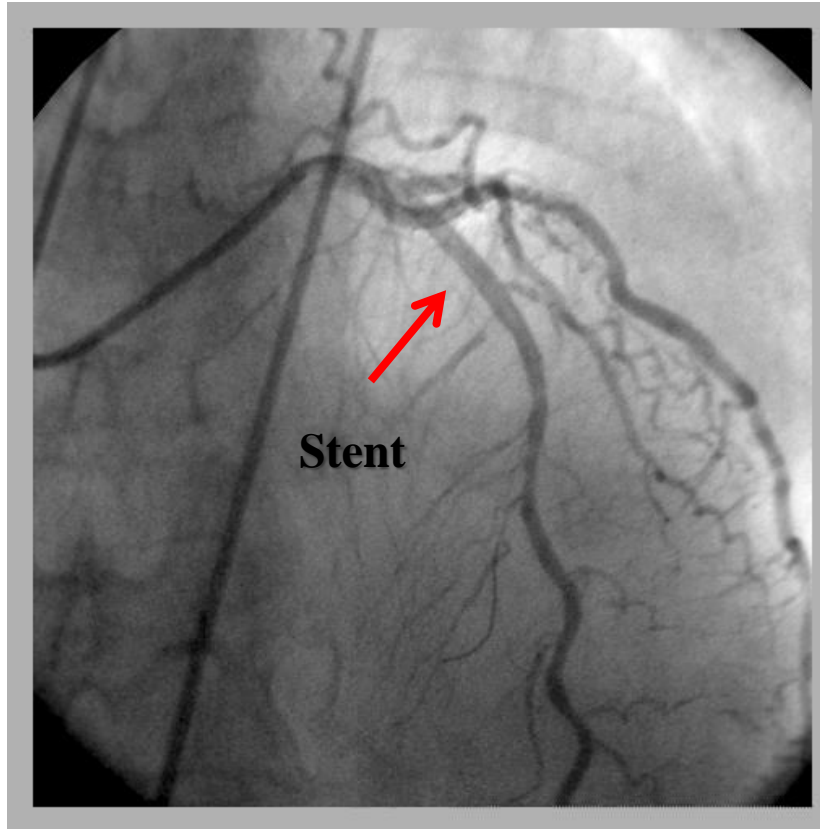


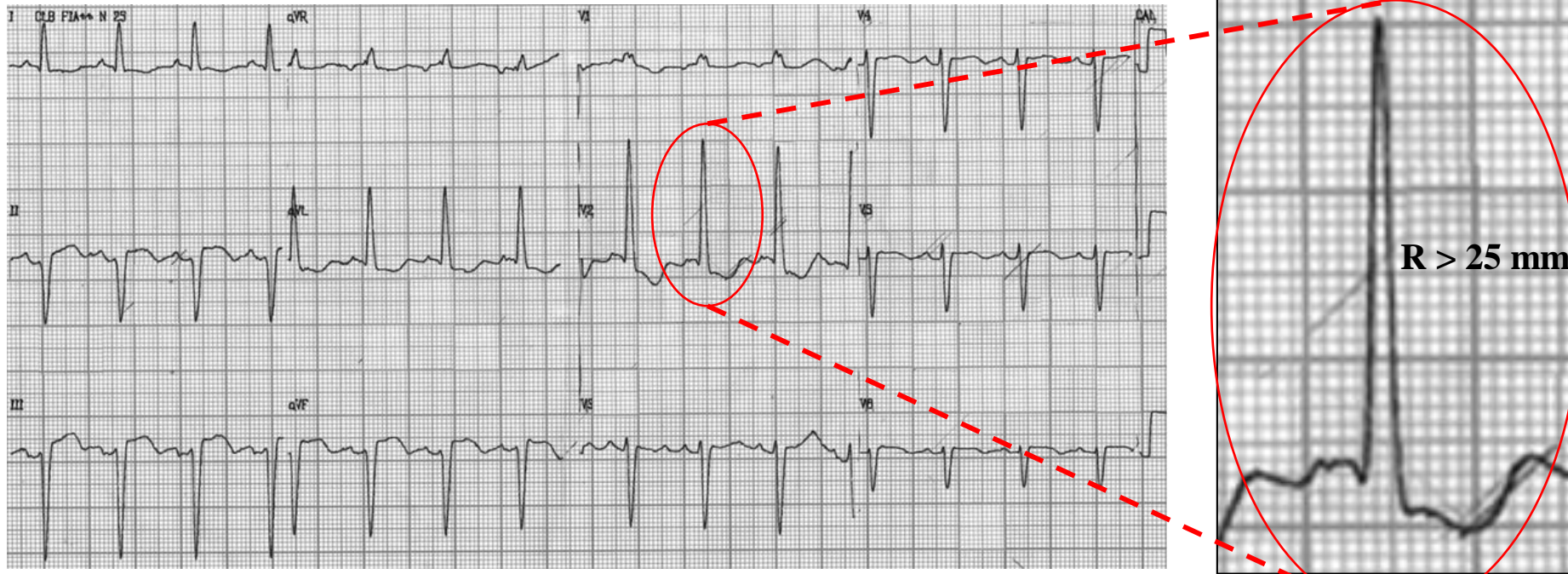
**ECG2 diagnosis:** sinus rhythm, heart rate 94 bpm, extreme QRS left axis deviation in the frontal plane (QRS axis  $-70^\circ$ ), QRS duration 115 ms, qR pattern on I and aVL, rS pattern on inferior leads,  $S_{III} > S_{II}$ ,  $S_{III} > 15$  mm and rS pattern in left precordial leads V5-V6: Rosenbaum's type IV Left Anterior Fascicular Block (LAFB). Additionally, inverted T wave on left leads I, aVL, V<sub>5</sub> and V<sub>6</sub> are observed; ischemic T waves? QRS duration  $< 120$  ms (close to 115 ms). qR pattern in V<sub>2</sub> lead, embryonic initial q wave in V<sub>2</sub> followed by very tall R wave in V<sub>2</sub> (R wave  $> 15$  mm): Prominent Anterior QRS Forces (PAF), absence of initial q wave on left precordial leads: Left Septal Fascicular Block (LSFB).  
**Conclusion:** bifascicular left fascicular block: LAFB associated with LSFB.

**Before**



**After**

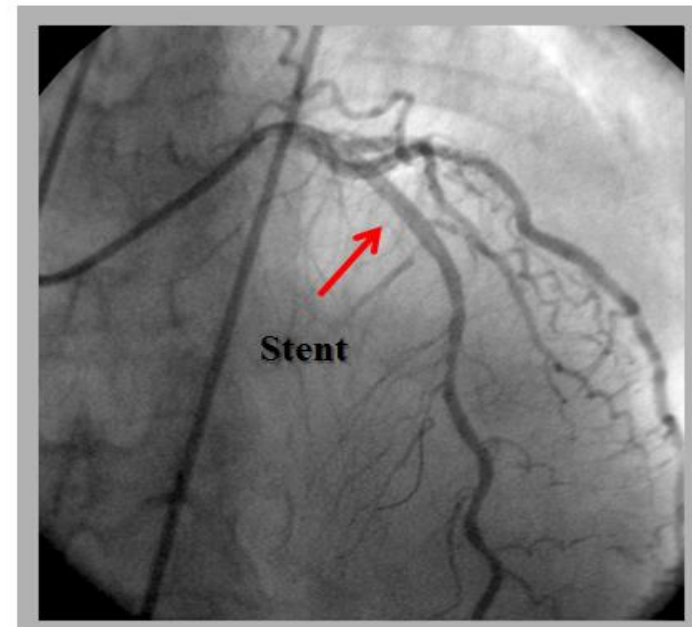
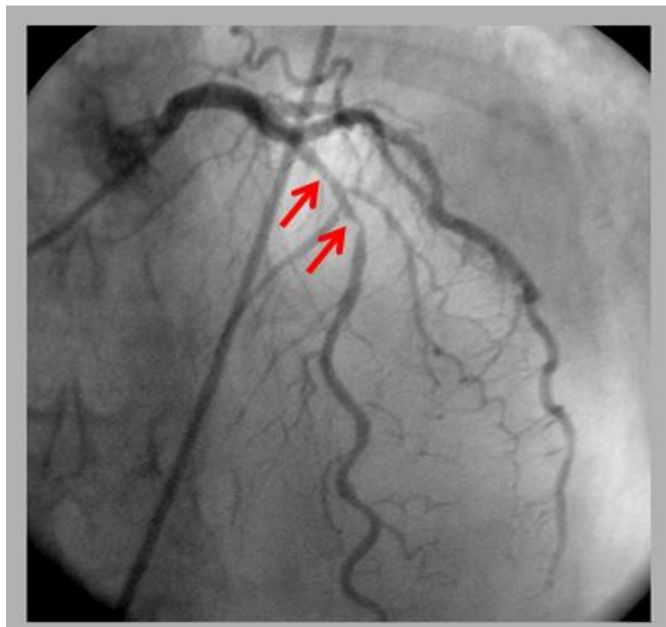




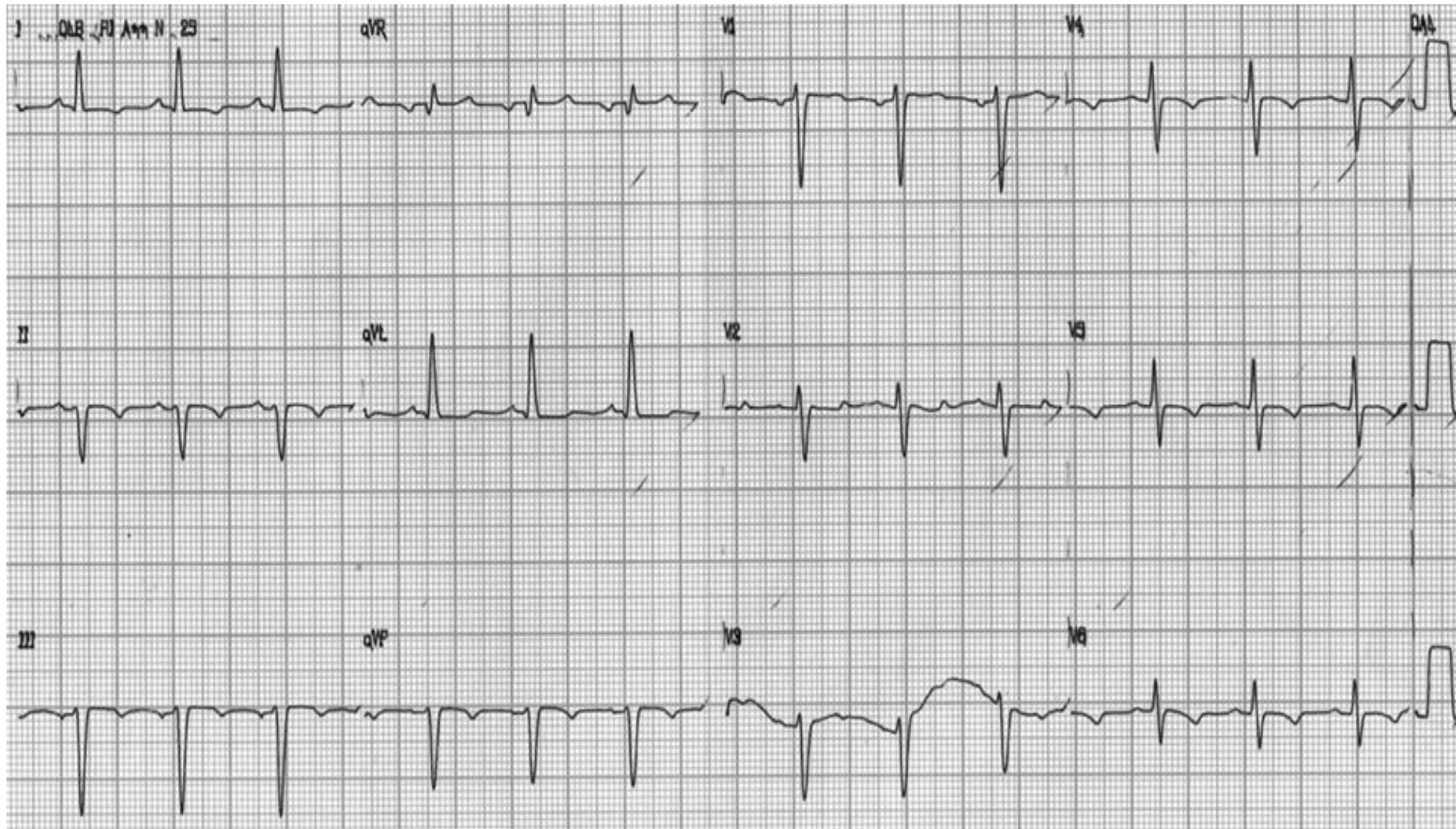
### Stent placement

Before

After



### ECG3. Performed on the third day after successful stent placement in the proximal LAD



**ECG diagnosis:** extreme left axis QRS deviation in the frontal plane, QRS axis  $-65^{\circ}$ , qR pattern in I and aVL, rS pattern in inferior leads,  $S_{III} > S_{II}$ , RS in left precordial leads: LAFB. In  $V_2$  the prominent QRS anterior forces (LSFB) disappeared. Negative T wave observed in anterolateral wall, suggestive of anterolateral ischemia.

**Conclusion:** LAFB associated to anterolateral ischemia. LSFB disappeared.

## Successful stent placement in the proximal LAD performed three days later



Discussion: this case report irrefutably is a typical transient or intermittent LSFb consequence of severe proximal obstruction of LAD associated with LAFB: bifascicular left bundle block.

The sudden onset and intermittent prominent anterior forces (PAF) in the right and/or middle precordial leads is indicative of critical proximal obstruction of the LAD secondary to left septal block (LSFB).

The LSF is irrigated exclusively by the septal perforating branches of the LAD. Consequently, critical lesions of the LAD before the first perforator septal branch ( $S_1$ ) constitute the main cause of LSFb in developed countries, and it is a major determinant of high R wave amplitude in right or intermediate precordial leads during acute myocardial ischemia (**Riera 2008-2008; Pérez-Riera 2011**). The LSFb eventually is exercise-induced, transient or intermittent, and sometimes it originates giant R waves.

Intermittent LSFb secondary to critical lesion of LAD was described recently during exercise testing (**Uchida 2006**). The appearance of LSFb in critical LAD lesions, speaks in favor of the proximal lesion of LAD and therefore, of a worse prognosis.

## **IV. Mitochondrial disease: Kearns-Sayre syndrome. Electro-vectorcardiographic evolution for the left septal fascicular block of the His bundle**

### **Background**

The Kearns-Sayre syndrome is a neuromyopathic disorder associated with mitochondrial abnormalities and characterized by the triad of chronic external ophthalmoplegia, atypical pigmentary retinopathy and progressive conduction system disorders. The observation of ragged red muscle fibers showed that they seem to contain an excess of altered mitochondria. The disease affects both sexes alike, beginning during the first or the second decade of life.

The following manifestations are observed: central bilateral deafness, pyramidal signs, ataxia, asymmetrical ptosis, external ophthalmoplegia and progressive muscular weakness associated with a significant increase of proteins of cerebrospinal fluid. A variety of endocrinopathies may occur.

### **Case report**

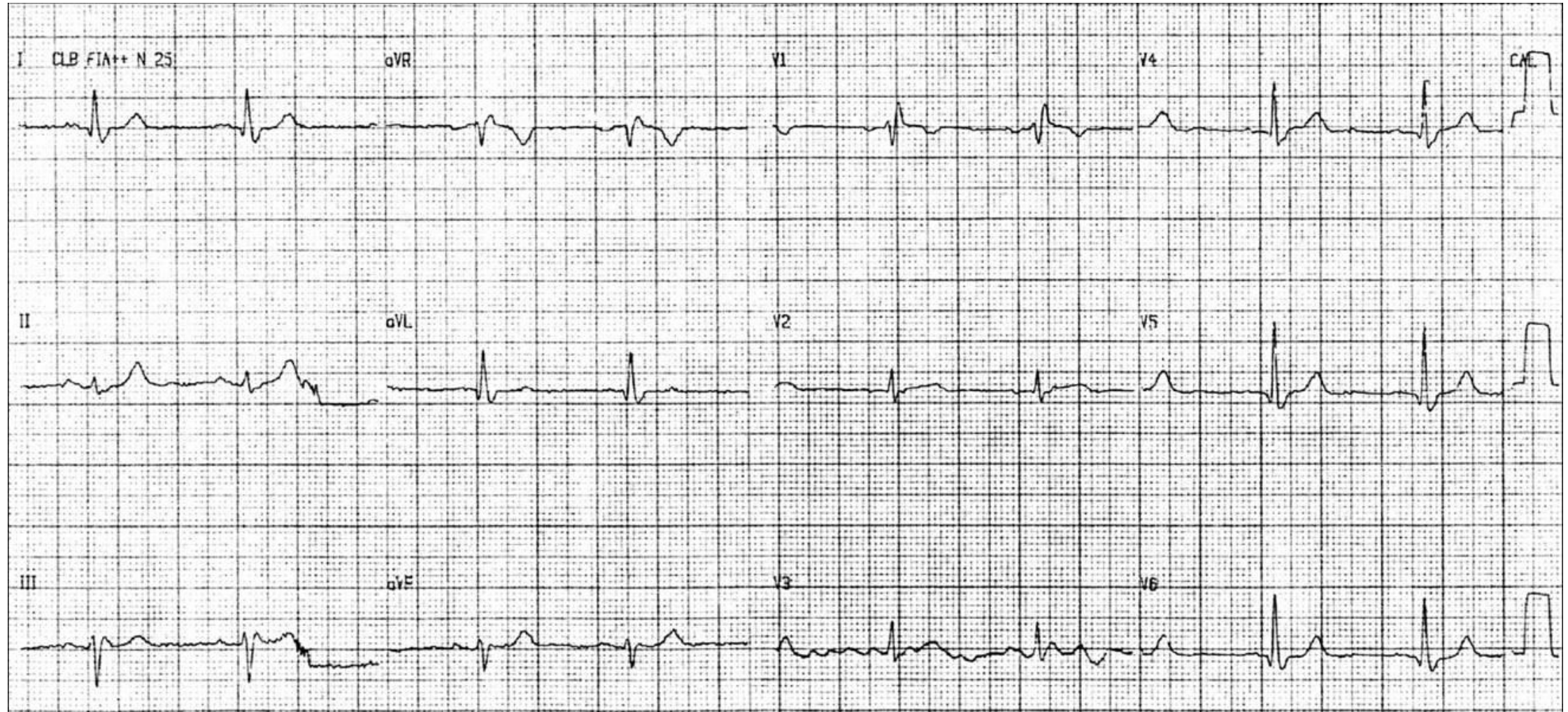
A 23-year-old man of African descent was referred to a cardiologist for evaluations based on alterations in retinal exam that are commonly associated with cardiac disease.

He reported progressive muscular weakness over the previous two years requiring interruption of regular practice of soccer three months prior to this presentation. Sudden and intermittent double vision developed 20 days before and ptosis of his left eye occurred 10 days previously, resulting in referral for a detailed ophthalmologic evaluation. On examination of his visual fields he was shown to have tubular vision while the presence of external ophthalmoplegia explained the double vision. He was also found to have an early nuclear cataract in the right eye. Fluorescein angiography of the retina showed atypical retinal pigmentary degeneration. The presence of the retinal pigmentation and external ophthalmoplegia raised concerns about Kearns Sayre Syndrome and he was referred for a cardiac evaluation.

In 1986, at the age of 6 years old, he had a pre-operative electrocardiogram that was interpreted as normal. At the age of 9 years old, an ECG obtained prior to an appendectomy showed incomplete right bundle branch block (IRBBB).



## Caucasian, 9-year-old child, with ECG performed before appendectomy in 1989

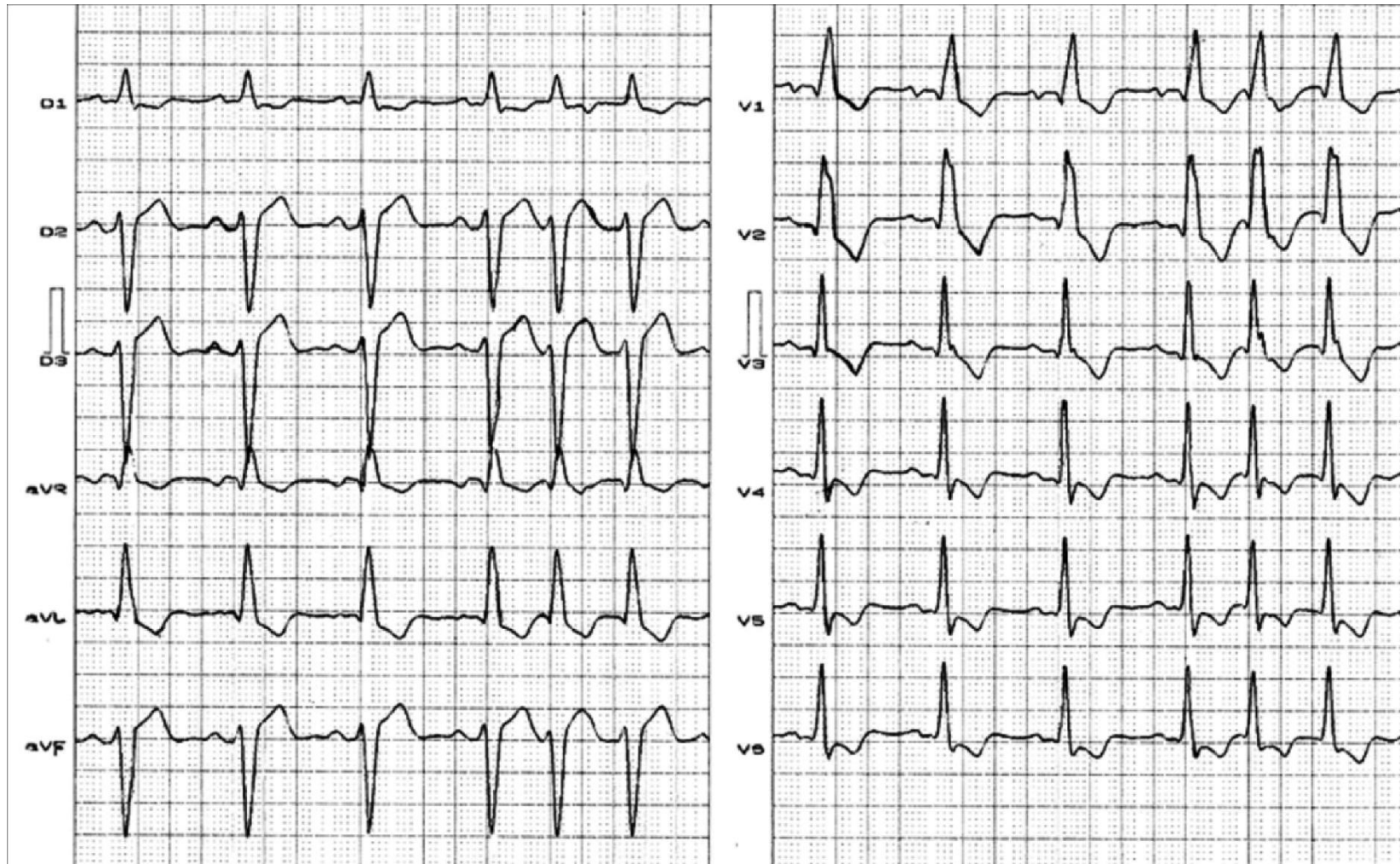


### **ECG diagnosis:** Incomplete RBBB.

Because of the abnormal ECG, an echocardiogram was obtained and reported as being normal. Three serological evaluations for Chagas disease were all negative. These included the ELISA, indirect immunofluorescence and indirect hemagglutination tests.

The patient appeared to be in good health, had an ectomorphic build with a height of 1.56 m, weight of 64 Kg, and BP 120/70 mmHg. He had a normal precordial examination, left ptosis and slight deafness (bilateral). Deep hyperreflexia was present bilaterally and a Babinsky sign was positive in the right foot.

**23 years old, 54 kg, 1.56 m, April 04, 2003**



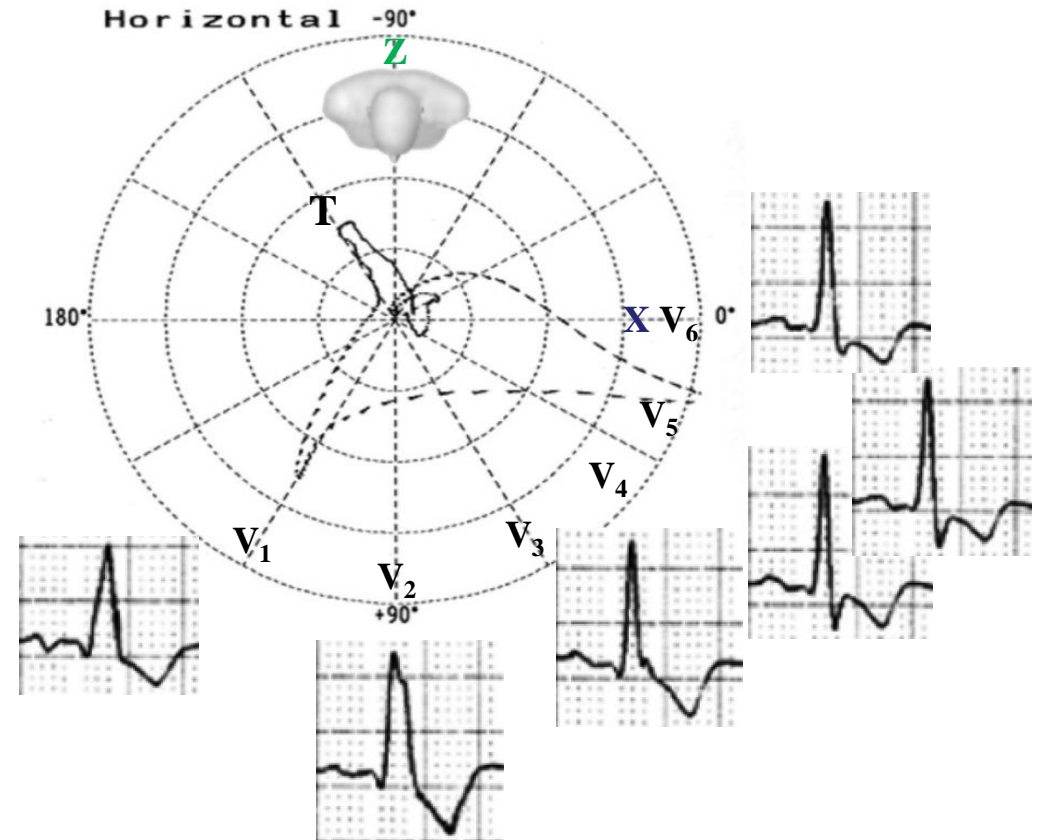
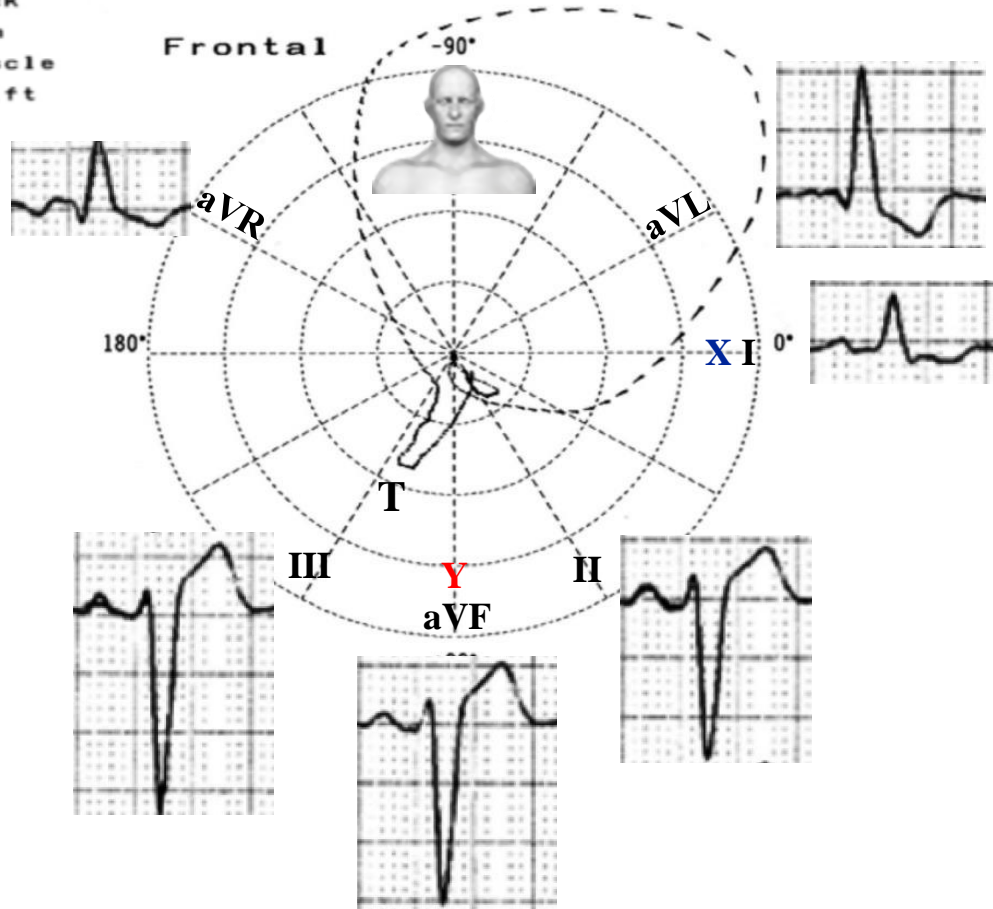
**ECG diagnosis.** Extreme left axis deviation in the frontal plane, QRS duration  $> 120$  ms, S in V5-V6 and final broad R in aVR, SIII  $>$  SII, rS pattern in inferior leads and qR pattern in aVL. Presence of PAF not explained by right ventricular hypertrophy or old posterior myocardial infarction (actual lateral myocardial infarction).

**Conclusion:** Complete RBBB, LAFB and LSFB: Trifascicular block.

**Note:** Sudden increase of heart rate from the fifth beat onward. Surely, this is a supraventricular tachycardia with a rate close to 150 beat/min, which maintains a morphology similar to the baseline complex.

# Vectorcardiogram

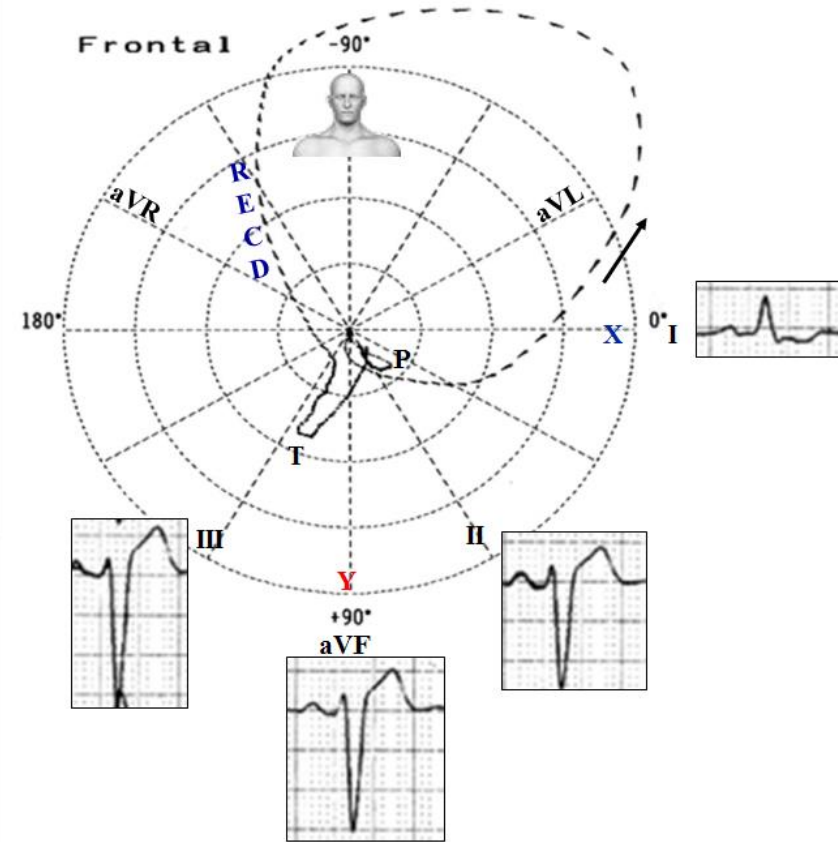
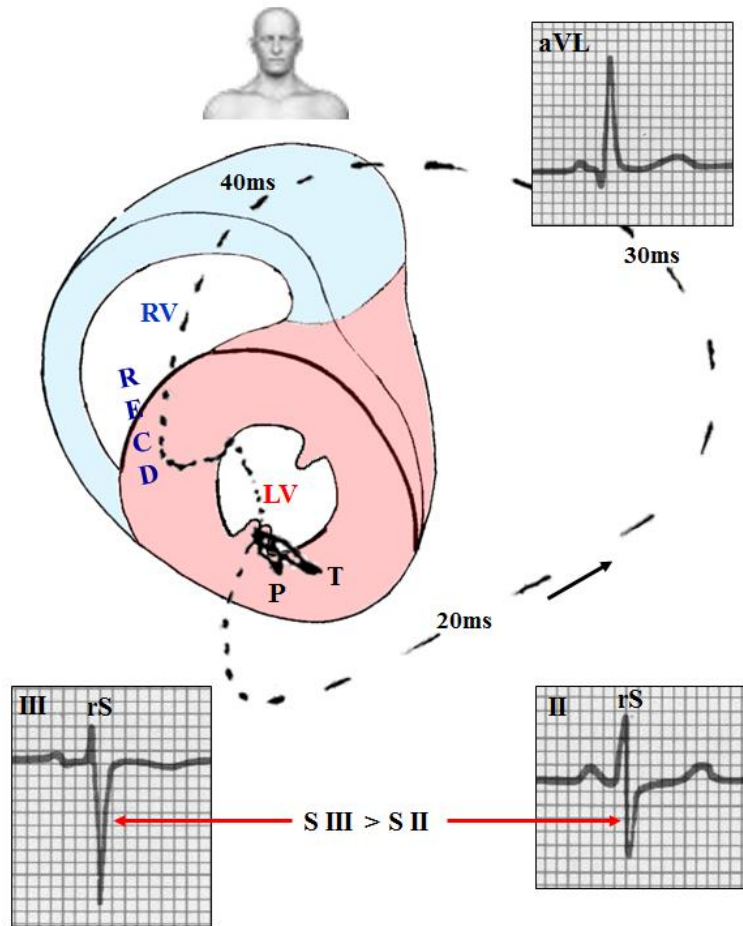
Sensi. 4  
Timer 2 msec  
Loop All Loop  
Sagittal Right  
Z Axis Back  
Filter Hum  
Muscle  
Drift



**Frontal plane:** QRS loop with counterclockwise rotation and extreme left deviation of the electrical axis in the left superior quadrant. The final portions of the QRS loop are located in the right superior quadrant, which indicates association with CRBBB.

**Horizontal plane:** the QRS loop is located predominantly in the anterior left quadrant. The QRS loop has clockwise rotation and right end conduction delay located in the right anterior quadrant. Unlike isolated CRBBB, the initial vectors from 10 to 20 milliseconds head backward and to the left by predominance of initial activation of the posteroinferior region in charge of the posteroinferior fascicle of the left branch; the only branch not blocked, thus configuring the typical element of LSFBB. This diagnosis must necessarily be clinical and electrocardiographic. The clinical absence of the causes of right ventricular hypertrophy or lateral myocardial infarction (previously called dorsal) is necessary.

# VCG comparison in the FP between LAFB + CRBBB and the present case (LAFB + LSFB + CRBBB)



## LAFB + CRBBB

The first 10 ms QRS loop is directed to the right and down in the frontal plane ( $\approx +120^\circ$ ) responsible for the presence of a small Q wave in leads I and aVL and a small and sharp R wave in II, III and aVF (Elizari 2007).

## LAFB + LSFB + CRBBB

The first 10 to 20 ms QRS loop is directed to the left and down. Consequently, the initial small Q wave in lead I disappears. This phenomenon is due to the absence of the  $1_{AM}$  vector (LSFB).

An echocardiogram showed indirect signs of decrease in ventricular compliance consistent with diastolic dysfunction.

Otorhinolaryngologic assessment: audiometry showed a moderate degree of bilateral sensorineural central hearing loss.

The neurological evaluation showed discrete signs of pyramidal tract involvement, decreased movement of his eye (external ophthalmoplegia) and abnormalities of the proximal muscles of his limbs and trunk consistent with probable myopathic (rhizomelic predominance) involvement.

A suboccipital puncture was performed for the analysis of cephalarachidian fluid. This showed 110 mg/dL proteins (the normal level being up to 30 mg/dL), glucose 50 mg/dL (normal), urea 30 mg/dL (normal), Pandy reaction +. Negative immunology for syphilis and cysticercosis.

His fasting blood sugar was 80 mg/dL

Venous plasma lactic acid was 40 mg/dL (normal level up to 22 mg/dL).

Serum aldolase was normal, as was a test for serum myoglobin using the nephelometric method.

His urine was screened for myoglobin, which was negative.

Levels of free carnitine and total carnitine were both elevated at 122 nmol/mL (reference value: up to 70 nmol/mL) and 140 nmol/mL (reference value: up to 90 nmol/mL) respectively.

Growth Hormone with glucagon stimulation demonstrated an increase of 2.5 mcg/L (reference value: increase of 5 mcg/L or more).

Growth Hormone with insulin stimulation did not show any significant increase (reference value: increase of 5 mcg/L or more).

Level of DPK: normal.

A muscle biopsy obtained from the deltoid muscle showed ragged-red fibers using standard microscopy. Electron microscopy showed mitochondrial aggregation with paracrystalline inclusions.

## **Discussion**

Cardiac involvement in the Kearns-Sayre syndrome may manifest by different degrees of AV block through the His-Purkinje system, giving rise to bundle branch blocks (right or left), frequently associated to left fascicular blocks. The symptoms have an evolutionary character, with the potential of reaching complete or third degree atrioventricular block requiring implantation of a permanent pacemaker.

In this report, we present the electrocardiographic evolution of the carrier of the syndrome, who having a normal ECG tracing initially, evolved into incomplete RBBB and over the course of 17 years into a rare trifascicular block: complete right bundle branch block (CRBBB), left anterior fascicular block (LAFB), and left septal fascicular block (LSFB) (Figures 02 and 03).

Since the pioneer publication of Durrer et al (Durrer 1970), the left branch of the His bundle is considered to divide into three anatomical-functional divisions. These serve to simultaneously activate the endocardium of the left septal surface at three points: the central region of the left septum, the antero-superior region at the base of the papillary muscle of the mitral valve, and the postero-inferior in the base of the postero-inferior papillary muscle of the same valve.

The existence of a third division that originates from the main trunk of the left bundle has been labeled by a multiplicity of names including left septal fascicle, middle or antero-medial division, and centro-septal fasciculus of the left branch. Its presence has been demonstrated by both anatomical dissections in humans (Sakaguchi 1988), and in dogs (Gallagher 1974), and careful analysis of the ventricular activation displayed on a 12 lead electrocardiogram (Madias 1993) or vectorcardiogram. Conduction disorders involving the left septal fascicle have been shown as a progressive or intermittent abnormality (Uchida 2006; Pérez-Riera 1999); and by electrophysiology study and surface endocardial mapping of both right and left ventricles (Sanchez 2001). Using invasive methodologies, Dahla et al (Dahla 1996), showed that the left septal fascicular delay/block manifests in ECG after transcatheter RBB ablation. The multifascicular nature of left intraventricular conduction is more apparent when diseased and unmasked by concomitant block in the RBB.

The left septal fascicle has, in most cases, fibers that originate in the angle formed by the emergence of other subdivisions of the left branch, as a separate tract. Less frequently, it originates as a ramification of the left anterior fascicle or the left posterior fascicle as a fan that cannot be individualized or rarely (15%), it may not exist (Kullbertus 1976).

The left septal fascicle activates the middle-septal region of the left septal surface, being responsible for the vector from the initial 10 to 20 ms, which is directed to the front and the right (85% of the cases) or to the front and the left (remaining 15%) depending on ventricular orientation. When there is concomitant conduction delay in the left septal fascicle and the left anterior fascicle of the left branch, and by the RBB (trifascicular block), initial septal activation depends exclusively on the left posterior fascicle which is directed posteriorly, manifested by an initial small q wave that is usually seen in the right precordial leads from V1 to V2, or from V1 to V3, because in these circumstances such activation heads toward the anterior chest leads.

All electrovectorcardiographic criteria of LSFB has to be validated, - similarly to LPFB – by first verifying the clinical absence of right ventricular hypertrophy, lateral myocardial infarction (previously called dorsal MI), type-A ventricular pre-excitation, septal hypertrophy, and other miscellaneous causes responsible for prominent anterior QRS forces. The diagnosis of isolated *Left Septal Fascicular Block* (LSFB) is based on the presence of:

1. Prominent Anterior QRS Forces (PAF) not explained by right ventricular hypertrophy or lateral myocardial infarction (previously called posterior MI) (MacAlpin 2003; Bayes d Luna 2007);
2. *QRS duration* <120 ms
3. The presence of qR pattern from V<sub>1</sub> to V<sub>3</sub> in absence of clinical causes of right ventricular hypertrophy, misplacement of electrocardiographic precordial leads (Marafioti 2004), RBBB associated with anteroseptal myocardial infarction;
4. R wave of V<sub>2</sub>-V<sub>3</sub> ≥ 15 mm or from V<sub>1</sub>;
5. Progressive decrease of R wave voltage from V<sub>4</sub> to V<sub>6</sub>;
6. Loss of septal initial q waves in left precordial leads V<sub>5</sub>-V<sub>6</sub> by initial activation from right to left in the posteroinferior region of the left ventricle (MacAlpin 2003).
7. Prolonged ventricular activation time >35 ms in V<sub>1</sub>- V<sub>2</sub>.
8. Frequently negative T waves in the right precordial leads

The presence of bifascicular block, usually manifested as RBBB and left anterior fascicular block in the Kearns-Sayre syndrome, is a class I indication for pacemaker implantation according to the ACC/AHA guidelines (Vardas 2007). This is the first electro-vectorcardiographically documented report of the presence of LSFb in the Kearns-Sayre syndrome, as part of a variant of a very rare trifascicular block.

# Left Septal Fascicular Block

Characterization,  
Differential Diagnosis and  
Clinical Significance

Andrés R. Pérez-Riera  
Raimundo Barbosa-Barros  
Adrian Baranchuk

 Springer



## References

1. Aravindakshan V, Elizari MV, Rosenbaum MB. Right bundle-branch block and left anterior fascicular block (left anterior hemiblock) following tricuspid valve replacement. *Circulation*. 1970;42(5):895-902.
2. Barnhill JE, Tendera M, Cade H, et al. Depolarization changes early in the course of myocardial infarction: significance of changes in the terminal portion of the QRS complex. *J Am Coll Cardiol* 1989;14(1):143–9.
3. Bateman T, Gray R, Maddahi J, Rozanski A, Raymond M, Berman D. Transient appearance of Q waves in coronary disease during exercise electrocardiography: consideration of mechanisms and clinical importance. *Am Heart J*. 1982;104(1):182-4.
4. Bayes de Luna A. Location of Q-wave myocardial infarction in the era of cardiac magnetic resonance imaging techniques: an update. *J Electrocardiol*. 2007;40(1):69-71.
5. Bianco M, Bria S, Gianfelici A, Sanna N, Palmieri V, Zeppilli P. Does early repolarization in the athlete have analogies with the Brugada syndrome? *Eur Heart J*. 2001;22(6):504–10.
6. Birnbaum Y, Kloner RA, Sclarovsky S, et al. Distortion of the terminal portion of the QRS on the admission electrocardiogram in acute myocardial infarction and correlation with infarct size and long-term prognosis (Thrombolysis in Myocardial Infarction 4 Trial). *Am J Cardiol*. 1996;78(4):396-403.
7. Birnbaum Y, Wagner GS. The Initial electrocardiographic pattern in acute myocardial infarction: correlation with infarct size. *J Electrocardiol*. 1999;32(suppl):122–8.
8. Birnbaum Y, Sclarovsky S. The grades of ischemia on the presenting electrocardiogram of patients with ST elevation acute myocardial infarction. *J Electrocardiol*. 2001;34 Suppl:17-26.
9. Birnbaum Y, Drew BJ. The electrocardiogram in ST elevation acute myocardial infarction: correlation with coronary anatomy and prognosis. *Postgrad Med J*. 2003;79(935):490-504.
10. Brady WJ, Chan TC. Electrocardiographic manifestations: benign early repolarization. *J Emerg Med*. 1999;17(3):473–8.
11. Chee CE, Anastassiades CP, Antonopoulos AG. Cardiac hypertrophy and how it may break an athlete's heart — the Cypriot case. *Eur J Echocardiogr*. 2005;6(4):301–7.
12. Chen CH, Nobuyoshi M, Kawai C. ECG pattern of left ventricular hypertrophy in non obstructive hypertrophic cardiomyopathy: The significance of the mid-precordial changes. *Am Heart J*. 1979;97(6):687-95.
13. Cola H, Hoffman R, Borrega NG, et al. Left posterior hemiblock related to an interventricular septum tumour. First case in the literature. *Eur Heart J*. 1992;13(4):574-5.

14. Dahla A, Zuelgaray JG, Deshpande S, Blanck Z, Biehl M, et al. Unmasking the trifascicular left intraventricular conduction system by ablation of the right bundle branch. *Am J Cardiol* 1996;77(9):706-12.
15. David D, Naito M, Michelson E, et al. Intramyocardial conduction: a major determinant of R wave amplitude during acute myocardial ischemia. *Circulation* 1982;65(1):161-7.
16. DeHaan R. Differentiation of the atrioventricular conducting system of the heart. *Circulation*. 1961;24:458-70.
17. Dilaveris P, Pantazis A, Gialafos E. Assessment of ventricular repolarization alterations in subjects with early repolarization. *Int J Cardiol*. 2004;96(2):273-9.
18. Elizari MV, Lazzari JO, Rosenbaum MB. Phase-3 and phase-4 intermittent left anterior hemiblock. Report of first case in the literature. *Chest*. 1972;62(6):673-7.
19. Elizari MV, Acunzo RS, Ferreiro M. Hemiblocks revisited. *Circulation*. 2007;115(9):1154-63.
20. Elizari MV, Chiale PA. The electrocardiographic features of complete and partial left anterior and left posterior hemiblock. *J Electrocardiol*. 2012;45(5):528-35. Feldman T, Chua KG, Childers RW. R wave of the surface and intracoronary electrogram during acute coronary artery occlusion. *Am J Cardiol*. 1986;58(10):885-90.
21. Grosse-Heitmeyer W. Is there a specific response of the ECG R-wave amplitude to exercise-induced myocardial ischemia? Exercise test and dipyridamole test. *Schweiz Med Wochenschr*. 1993;123(10):414-21
22. Gussak I, Bjerregaard P, Kostis J. Electrocardiographic "lambda" wave and primary idiopathic cardiac asystole: a new clinical syndrome? *J Electrocardiol* 2004;37(2):105-7.
23. Hassett MA, Williams RR, Wagner GS. Transient QRS changes simulating acute myocardial infarction. *Circulation* 1980;62(5):975-9.
24. Hilsenrath J, Glassman E, Roberts G, Hamby RI, Hoffman I. Pitfalls in the prediction of coronary artery obstruction from ECG-VCG anterior infarct patterns. In: Hoffman I, Hamby RI, Glassman E, editors. *Vectorcardiography 2. Proceedings of the XIth International Symposium on Vectorcardiography*. New York. Amsterdam: North Holland Publishing Co. 1971. P. 362-7.
25. Holland R, Brooks H. The QRS complex during myocardial ischemia: an experimental analysis in the porcine heart. *J Clin Invest*. 1976;57:541-50.
26. Ibarrola M, Chiale PA, Pérez-Riera AR, Baranchuk A. Phase 4 left septal fascicular block. *Heart Rhythm*. 2014;11(9):1655-7.
27. Knotts RJ, Wilson JM, Kim E, et al. Diffuse ST depression with ST elevation in aVR: Is this pattern specific for global ischemia due to left main coronary artery disease? *J Electrocardiol*. 2013;46(3):240-8.

28. Kulbertus HE, Demoulin J. Pathological basis of concept of left hemiblock. In: The Conduction System of the Heart. Wellens HJJ, Lie KI, Janse MJ, editors. Lea &Febiger, Philadelphia; 1976. P. 287-95.
29. MacAlpin RN. Left Septal Fascicular Block: Myth Or Reality? Indian Pacing Electrophysiol. J. 2003;3(3):157-77.
30. Madias JE.: The "giant R waves" ECG pattern of hyperacute phase of myocardial infarction. J Electrocardiol. 1993;26(1):77-82.
31. Madias JE, Attari M, Bravidis D.Giant R-waves in a patient with an acute inferior myocardial infarction. J Electrocardiol. 2001;34(2):173-7.
32. Magnacca M, Valesano G, Rizzo G, Trotti F, Pagetto A, Boverio R. Diagnostic value of electrocardiogram in septal fascicular conduction disorders of the left branch in diabetics. Minerva Cardioangiologica. 1988;36(7-8):361-3.
33. Marafioti V, Variola A. Pseudoinfarction pattern by misplacement of electrocardiographic precordial leads. Am J Emerg Med. 2004;22(1):62-3.
34. Mehta M, Jain AC, Mehta A. Early repolarization. Clin Cardiol. 1999;22(2):59-65.
35. Mizusawa Y, Bezzina CREarly repolarization pattern: its ECG characteristics, arrhythmogeneity and heritability.J Interv Card Electrophysiol. 2014 Apr;39(3):185-92.
36. Moffa PJ, Pastore CA, Sanches PCR et al. The left-middle (septal) fascicular block and coronary heart disease. In: Liebman J, editor. Electrocardiology' 96 – From the cell to body surface. Cleveland, Ohio, Word Scientific; 1996. P. 547-50.
37. Moffa PJ, Ferreira BM, Sanches PC, Tobias NM, Pastore CA, Bellotti G. Intermittent antero-medial divisional block in patients with coronary disease. Arq Bras Cardiol. 1997;68(4):293-6.
38. Moffa PJ, Sanches PCR. Tranchesí Electrocardiograma Normal e Patológico. Editora Roca Ltda; 2001. Capítulo 19, p. 413-61.
39. Nakaya Y, Hiasa Y, Murayama Y, et al. Prominent anterior QRS force as a manifestation of left septal fascicular block. J Electrocardiol. 1978;11(1):39-46.
40. Oguro T, Fujii M, Fuse K, et al. Electrical alternans induced by a brief period of myocardial ischemia during percutaneous coronary intervention: The characteristic ECG morphology and relationship to mechanical alternans. Heart Rhythm. 2015;12(11):2272-7.
41. Pérez Riera AR. Electrocardiographic and Vectorcardiographic Sequential Demonstration of Unknown Left Trifascicular Intraventricular Conduction Block: Right Bundle Branch Block, Left Posterior Fascicular Block and Left Septal Fascicular Block: Anterior Conduction Delay. Paper presented at the 1rst Virtual Congress of Cardiology. October, 1999 to March, 2000 on Internet (<http://www.fac.org.ar/cvirtual/cvirteng/cienteng/areng/arm3601i/irierape/irierape.htm>).
42. Pérez-Riera AR, Abreu LC, Yanowitz F, et al. "Benign" early repolarization versus malignant early abnormalities: clinical-electrocardiographic distinction and genetic basis. Cardiol J. 2012;19(4):337-46.

43. Pérez-Riera AR, Nadeau-Routhier C, Barbosa-Barros R, Baranchuk A. Transient Left Septal Fascicular Block: An Electrocardiographic Expression of Proximal Obstruction of Left Anterior Descending Artery? *Ann Noninvasive Electrocardiol.* 2016;21(2):206-9. a
44. Pérez-Riera AR, de Abreu LC, Barbosa-Barros R, et al. R-Peak Time: An Electrocardiographic Parameter with Multiple Clinical Applications. *Ann Noninvasive Electrocardiol.* 2016;21(1):10-9. b
45. Riera AR, Ferreira C, Schapachnik E, Sanches PC, Moffa PJ. Brugada syndrome with atypical ECG: downsloping ST-segment elevation in inferior leads. *J Electrocardiol.* 2004;37(2):101-4.
46. Riera AR, Ferreira C, Ferreira Filho C, et al. Wellens syndrome associated with prominent anterior QRS forces: an expression of left septal fascicular block? *J Electrocardiol.* 2008;41(6):671-4 a.
47. Riera AR, Kaiser E, Levine P, et al. Kearns-Sayre syndrome: electro-vectorcardiographic evolution for left septal fascicular block of the His bundle. *J Electrocardiol.* 2008;41(6):675-8 b.
48. Rosenbaum MB, Elizari MV, Lazzari JO. *Los hemibloqueos.* Editora Paidós Buenos Aires; 1968.
49. Rosenbaum MB, Elizari MV, Levi RJ, Nau GJ, Pisani N, Lazzari JO, Halpern MS. Five cases of intermittent left anterior hemiblock. *Am J Cardiol.* 1969;24(1):1-7.
50. Rosenbaum MB, Nau GJ, Levi RJ, Halpern MS, Elizari MV, Lazzari JO. Wenckebach periods in the bundle branches. *Circulation.* 1969;40(1):79-86.
51. Rosenbaum MB. The hemiblocks: diagnostic criteria and clinical significance. *Mod Concepts Cardiovasc Dis.* 1970;39(12):141-6.
52. Rosenbaum MB, Elizari MV, Lazzari JO. Differential diagnosis of hemiblocks. *Medicina (B Aires).* 1972;32(3):315-31.
53. Rosenbaum MB, Yesuron J, Lazzari JO, Elizari MV. Left anterior hemiblock obscuring the diagnosis of right bundle branch block. *Circulation.* 1973;48(2):298-303.
54. Sakaguchi Y, Konishi N, Enoki N, et al. Morphological study of the left bundle branch in the normal human heart. *Acta Pathol Jpn* 1988;38(4):417-24.
55. Sanches PCR, Moffa PJ, Sosa E, et al. Electrical Endocardial Mapping of Five Patients With Typical ECG of Left-Middle (Septal) Fascicular Block. In: *Proceeding of The XXVIII International Congress on Electrocardiology, Guarujá SP Brazil; 2001.* P. 89-95.
56. Sato A, Tanabe Y, Chinushi M, et al. Analysis of J waves during myocardial ischaemia. *Europace.* 2012;14(5):715-23.
57. Sclarovsky S, Mager A, Davidson E, et al. Classification of acute myocardial ischemia by electrocardiograph. *Harefuah.* 1989;116(1):35-9.
58. Sclarovsky S, Mager A, Kusniec J, et al. Electrocardiographic classification of acute myocardial ischemia. *Isr J Med Sci.* 1990;26(9):525–33.
59. Sclarovsky S. *Electrocardiography of acute myocardial ischaemic syndromes.* London: Martin Dunitz, 1999.

60. Schiebler T, Stark M, Caesar R. Die stoffwechselsituation des reizleitungssystems. *Klin Wochenschr*1956;34:181–3.
61. Sejersten M, Birnbaum Y, Ripa RS, et al. Influences of electrocardiographic ischaemia grades and symptom duration on outcomes in patients with acute myocardial infarction treated with thrombolysis versus primary percutaneous coronary intervention: results from the DANAMI-2 trial. *Heart*. 2006;92(11):1577-82.
62. Selvester RH, Wagner NB, Wagner GS. False-positive posterior myocardial infarct criteria during left anterior descending coronary angioplasty. *J Electrocardiol*. 1988;21 Suppl:S105-11.
63. Smith SW. Upwardly concave ST segment morphology is common in acute left anterior descending coronary occlusion. *J Emerg Med*. 2006;31(1):69–77.
64. Sinno MC, Kowalski M, Kenigsberg DN, et al. R-wave amplitude changes measured by electrocardiography during early transmural ischemia. *J Electrocardiol*. 2008;41(5):425-30.
65. Spekhorst H, Sippens Groenewegen A, David G, *et al*. Body surface mapping during percutaneous transluminal coronary angioplasty. QRS changes indicating regional myocardial conduction delay. *Circulation*, 1990;81:840–9.
66. Spratt KA, Borans SM, Michelson EL. Early repolarization: normalization of the electrocardiogram with exercise as a clinically useful diagnostic feature. *J Invasive Cardiol*. 1995;7(8):238–42.
67. Tahara Y, Mizuno H, Ono A, Ishikawa K. Evaluation of the electrocardiographic transitional zone by cardiac computed tomography. *J Electrocardiol*. 1991;24(3):239–45.
68. Tranchesi J, Moffa PJ, Pastore CA, et al. Block of the antero-medial division of the left bundle branch of His in coronary diseases. Vectrocardiographic characterization. *Arq Bras Cardiol*. 1979;32(6):355-60.
69. Turnipseed SD, Bair AE, Kirk JD, Diercks DB, Tabar P, Amsterdam EA. Electrocardiogram differentiation of benign early repolarization versus acute myocardial infarction by emergency physicians and cardiologists. *Acad Emerg Med*. 2006;13(9):961–6.
70. Uchida AH, Moffa PJ, Riera AR, Ferreira BM. Exercise-induced left septal fascicular block: an expression of severe myocardial ischemia. *Indian Pacing Electrophysiol J*. 2006;6(2):135-8.
71. Vardas PE, Auricchio A, Blanc JJ, et al. Guidelines for cardiac pacing and cardiac resynchronization therapy. The Task Force for Cardiac Pacing and Cardiac Resynchronization Therapy of the European Society of Cardiology. Developed in collaboration with the European Heart Rhythm Association.. *Europace*. 2007;9(10):959-98.
72. Zoghi M, Gürgün C, Yavuzgil O, et al. The angiographic correlation between ST segment depression in noninfarcted leads and the extent of coronary artery disease in patients with acute inferior myocardial infarction: a clue for multivessel disease. *Can J Cardiol*. 2003 Jan;19(1):67-71.