

# **Idosa com quadro de síndrome coronariana aguda e síncope de longa data**

## **Old woman with acute coronary syndrome and history of repetitive syncope**

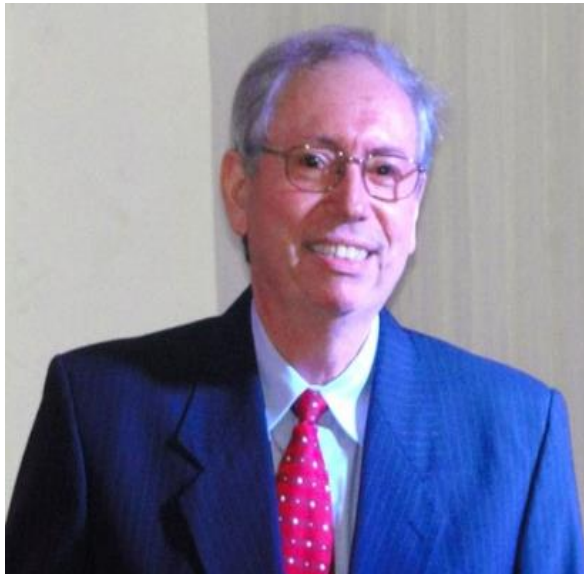
Raimundo **Barbosa-Barros**, M.D. (nickname “Raymond, the Fox”)

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<https://ekgvcg.wordpress.com>



## Portuguese Relato de caso

Queixa principal: Dor precordial e desmaio

Paciente idosa, (83 anos) diabética, dislipidêmica, e hipotiroidea. Admitida na emergência por quadro de síncope o que refere ocorrer periodicamente desde os 38 anos que ela associa a contrariedades. Após a recuperação da consciência refere dor precordial opressiva e dispnéia. O ECG realizado nesse momento mostrava probable bloqueio completo do ramo do ramo direito, bloqueio divisional ântero-superior e alterações de repolarização ventricular (ARV), sobrecarga do ventrículo esquerdo (SVE) e aumento dos biomarcadores de necrose miocárdica (troponina T; 0.3/ml). Feita a hipótese diagnóstica de síndrome coronariana aguda sem elevação do segmento ST, se solicita cinecoronariografia diagnóstica a qual não foi realizada em consequência de novo rebaixamento da consciência, pico hipertensivo e disfunção renal.

Refere dor precordial associado a náuseas e dispneia há 3 meses sem investigação prévia.. Referida a UTI para continuidade dos cuidados. Da entrada alerta, cooperativa, Glasgow 15 sem déficit com bom padrão respiratório e hipertensão assintomática. PA 203/105 mmHg, FC 80bpm RCR me 2 tempos , bulhas rítmicas e normofonéticas sem sopros.

EC0: 16/10 discreto aumento do VVE para o biótipo ( 33mm/m<sup>2</sup>) hipertrofia concêntrica discreta do VE e contratilidade global e segmentar do VE preservada

Raimundo e Andres

Perguntas

1. Qual o diagnostico eletrovetorcardiografico ?
2. Qual o quais as causas das forcas anteriores proeminentes ? E por que ?

## Case Report

Main complaint: precordial pain and fainting.

Elderly, female patient (83 years old): diabetic, dyslipidemic, and with hypothyroidism. She was admitted in the ER due to symptoms of syncope, which she mentions has been happening to her since she was 38 years old, associated to different troubles. After recovering consciousness, she reported oppressive precordial pain and dyspnea. The ECG made at the time revealed complete right bundle branch block, left anterior fascicular block, and ventricular repolarization alterations (VRA), left ventricular enlargement (LVE) and positive myocardial necrosis biomarkers (troponin T; 0.3/ml). A diagnostic hypothesis of non-ST segment elevation acute coronary syndrome was made, and diagnostic coronary angiography was requested, which could not be done as her consciousness got depressed again, presented hypertensive peak and renal dysfunction. She mentioned precordial pain associated to nausea and dyspnea 3 months before, with no previous investigation. She was sent to the ICU to continue with her care. She was admitted alert, cooperative, in Glasgow 15 with no deficit, and good respiratory pattern and asymptomatic hypertension. BP 203/105 mmHg, HR 80 bpm, split regular heart rhythm, rhythmic and normal sounds with no murmurs.

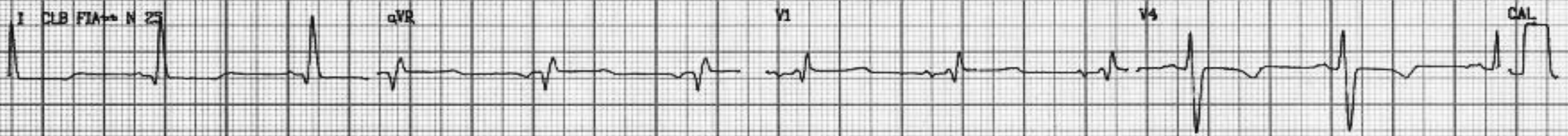
Echo: 16/10, discrete increase of left ventricular volume for the biotype (33 mm/m<sup>2</sup>), discrete concentric LV hypertrophy and preserved overall and segmentary LV contractility.

Raimundo and Andres.

Questions

1. What is the electro vectorcardiographic diagnosis?
2. What is / are the causes of the prominent anterior QRS forces? And Why?

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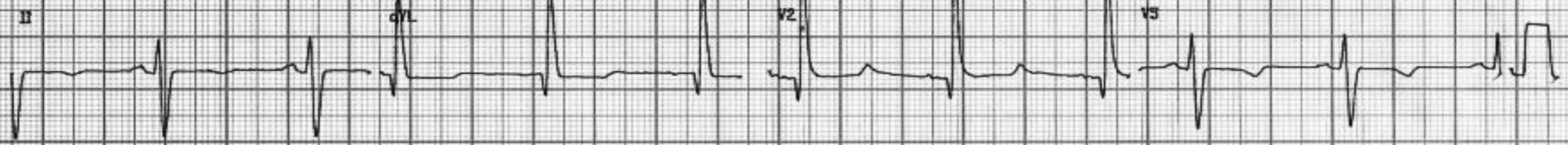
aVR

V1

V4

CAL

II



aVL

V2

V5

III



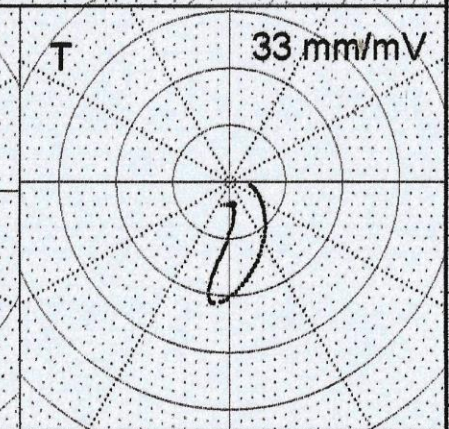
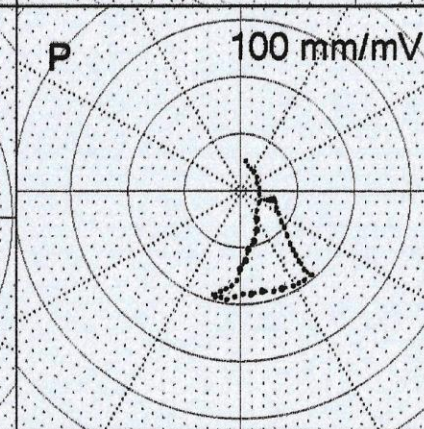
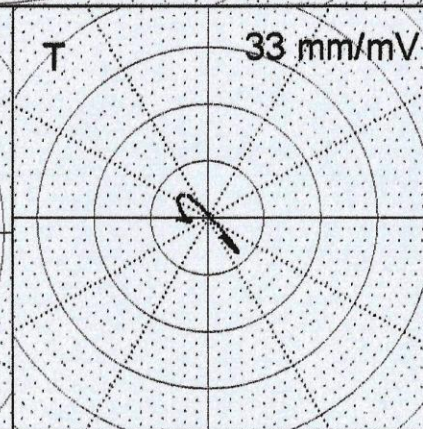
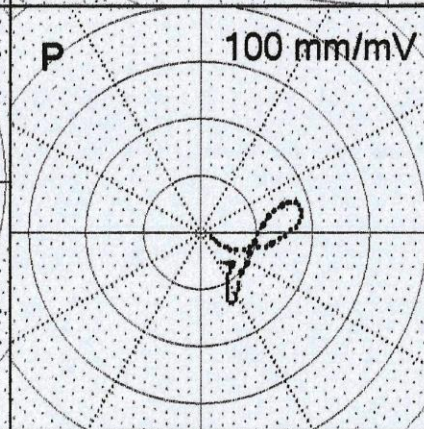
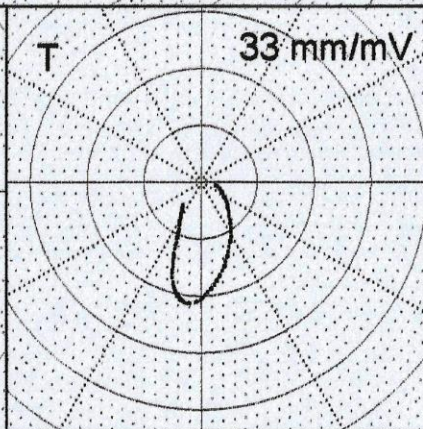
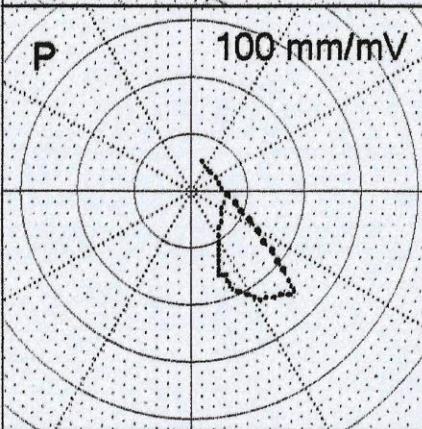
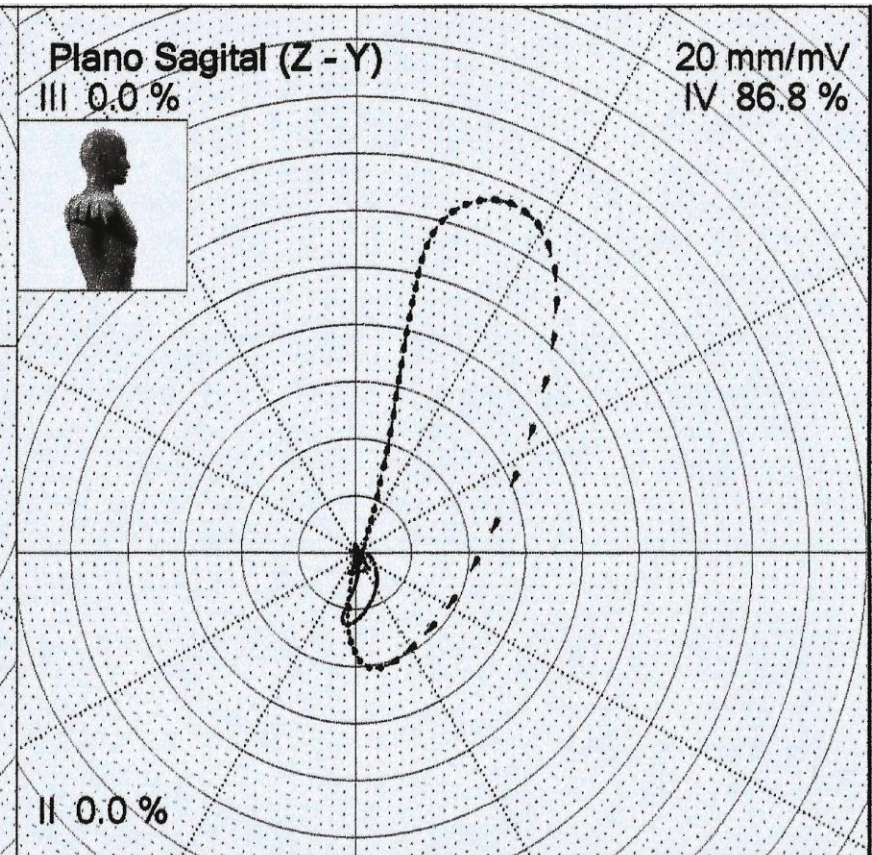
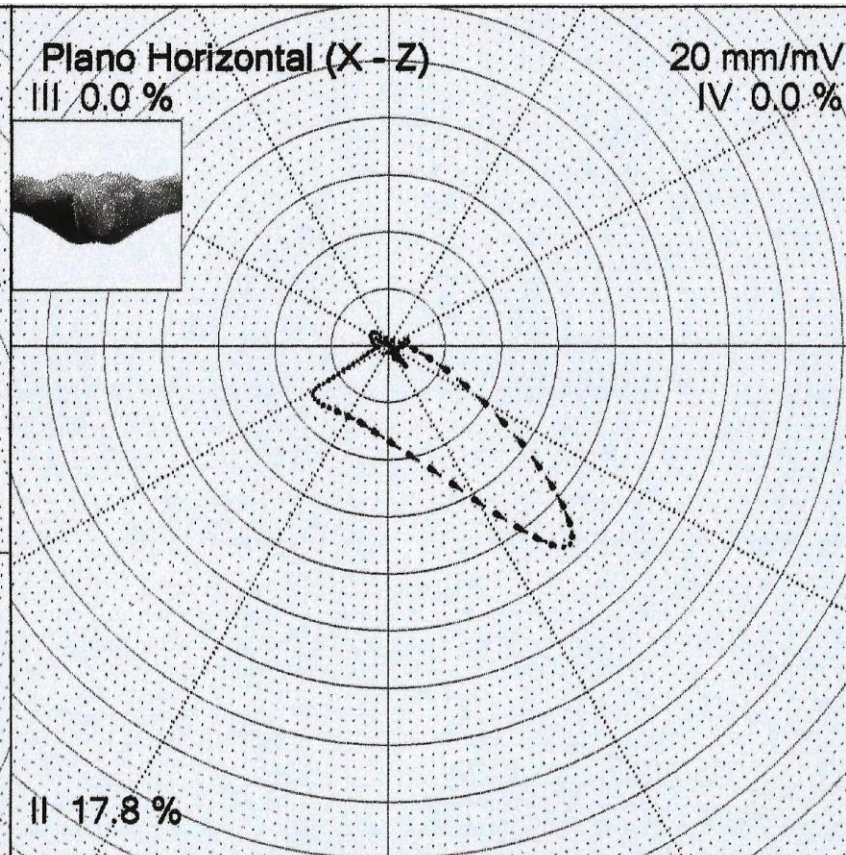
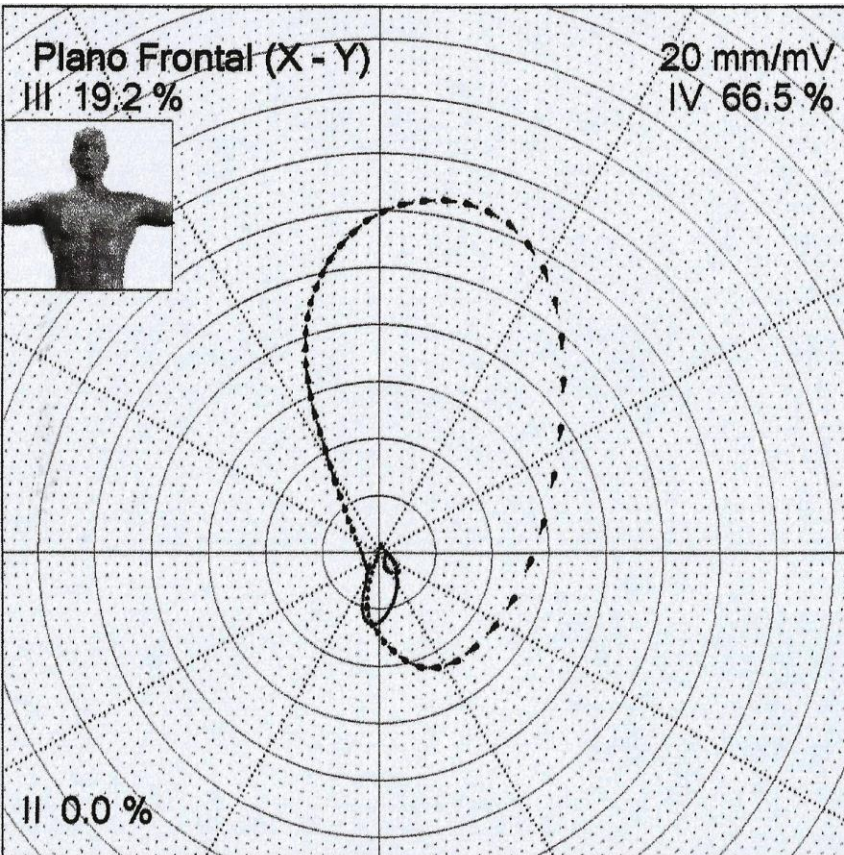
aVF

V3

V6

II





# **Colleagues opinions**

Dear Andrés,

Concentric left ventricular hypertrophy, complete right bundle branch block and a PR too short for a 83 year-old woman: Fabry disease should be ruled out. Syncope may come from neurological manifestations of this disease. The QRS axis can be ascribed to the patient thorax morphology (brief and large)? The acute coronary syndrome is expected in old patient with a metabolic syndrome. Hypothyroidism may also play a role in eCG features?

I look forward to your comments,

Best regards,

**Philippe Chevalier**

[philippe.chevalier@chu-lyon.fr](mailto:philippe.chevalier@chu-lyon.fr)

Chef du service de Rythmologie GHE, CHU Lyon  
Claude Bernard University Lyon, France



**Portuguese:**

**Um caso muito interessante professores.**

**De maneira resumida, vemos um registro em ritmo sinusal, com associação de BDAS + BDAM + AFC pelo ramo direito. e alterações de repolarização em parede anterolateral.**

**Marcador de doença avançada e importante no sistema de condução, o que deve justificar as síncope**

**Obrigado por partilhar**

**Horácio Gomes Pereira Filho. University of São Paulo, São Paulo Brazil. Expertise in Cardiology and electrocardiology.**

[hgpfilho@gmail.com](mailto:hgpfilho@gmail.com)

**English**

**A very interesting case teachers.**

**Briefly, we see a sinus rhythm associated with Left Anterior Fascicular Block(LAFB) + Left Septal Fascicular Block (LSFB) + Right End Conduction Delay (**RECD**) in the Right Bundle Branch and repolarization changes in the anterolateral wall.**

**Marker of advanced and important disease in the intraventricular conduction system, which should justify the syncope's episodes.**

**Thanks for sharing**

**Horácio Gomes Pereira Filho M.D.**

[hgpfilho@gmail.com](mailto:hgpfilho@gmail.com)





Spanish: Hola Andrés: La paciente presenta ritmo sinusal, sobrecarga atrial izquierda (duración de onda P 120 mseg), PR 160 mseg, eje eléctrico del QRS muy desviado a la izquierda por bloqueo del fascículo anterior izquierdo (Ondas R predominantes en I y aVL con una pequeña onda q inicial; onda r en II, III y aVF seguidas de una onda S profunda).

Fuerzas del QRS anteriores prominentes por bloqueo del fascículo medio-septal izquierdo o secuela lateral. Observo además supra desnivel del segmento ST en V1 y V2 y DIII y aVF, con supradesnivel del ST también en aVR. ST rectificado y onda T negativas simétricas de V4 a V6 sugestivo de hipertrofia ventricular izquierda.

Mi interpretación diagnóstica es el de un síndrome coronario agudo por enfermedad de múltiples vasos en una paciente portadora de feocromocitoma benigno o incidentaloma por la historia de síncope, disnea y angina. Las crisis de insuficiencia renal son descriptos asociados a esta entidad.

Un cordial saludo

**Martín Ibarrola MD** Provincia de Buenos Aires Argentina

English Hi Andrés: The patient has sinus rhythm, left atrial enlargement (P-wave duration=120 ms), PR interval 160 ms, QRS axis with extreme lefts deviation consequence of left anterior fascicular block (qR in I and aVL, initial r wave in II, III and aVF followed by a deep S wave).

Precordial leads shows Prominent QRS anterior(PAF) consequence of LSFB or lateral MI sequel. Additionally, ST segment elevation in V1-V2 and aVR, III and aVF,. Rectified an negative ST segment followed by symmetrical negative T wave in precordial lateral leads V4 to V6 suggestive of LVH.

My diagnostic interpretation is: acute coronary syndrome by multivessels disease in a patient with benign pheochromocytoma or incidentaloma by the history of syncope, dyspnea and angina. The crises of kidney failure are described associated with this entity.

A cordial greeting

Martin Ibarrola MD



Dear Andrés. Short PQ-interval, maybe tendency to reentry SVT. No delta wave. LVH + “strain” pattern in lateral leads. LAFB+RBBB. Possibly left septal fascicular block.

Coronary artery disease can be treated conservatively with medication.

Holter indicated. Pheochromocytoma to be ruled out, although rare at old age.

Best regards

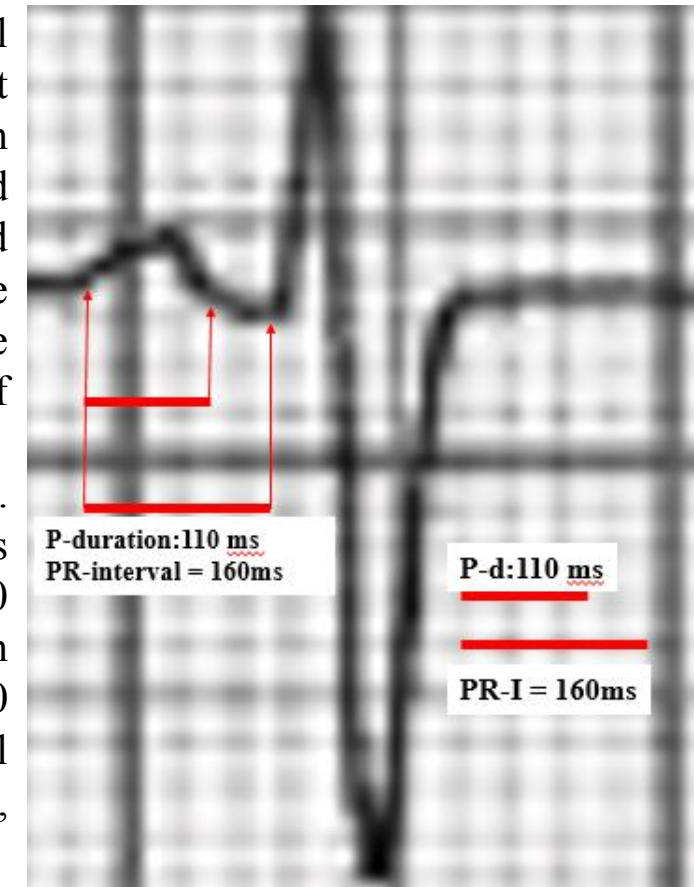
**Kjell Nikus M.D.Ph.D.**

Tampere, Finland



**Answer to Philippe, Martin and Kjell from Andrés:** Fabry disease (FD) is an inherited X-chromosomal lysosomal storage disease resulting in intracellular storage of globotriaosylceramide. Cardiac involvement is most frequently manifested as LVH. However, patients with FD may also have various conduction abnormalities before LVH develops. P-wave duration, PR-interval and QRS width are shorter and repolarization dispersion more pronounced in patients with FD compared with heart rate and age-matched controls. The earliest ECG manifestation are **shortened PR interval** and **resting bradycardia**. The significant shortening of the PR-interval in FD occurs because of a marked shortening of the P-wave duration, which in itself demonstrated a high sensitivity and specificity for early detection and treatment of this disease.

**PR or PQ interval:** The PR interval duration increases directly with age and inversely with HR increases. The normal duration of the neonatal (birth to 30 days) PR interval is between 70 and 140 ms and averages 100 ms. In infants and children from 30 days to 1.5 years the maximal PR interval is 160 ms for HR <70 bpm, 150 ms for HR 71-110 bpm, 140 ms for HR 111-130 bpm, and 130 ms for HR >130 bpm. In children between 1.5 and 6 years of age the maximal PR interval is 170 ms for HR <90 bpm, 160 ms for HR 91-110 bpm, 150 ms for HR 111-130 bpm, and 140 ms for HR >130 bpm. From age 7 to 13 years the normal maximal PR interval is 180 ms for HR <70 bpm, 170 ms for HR 70-90 bpm, 160 ms for HR 91-110 bpm, 150 ms for HR 111-130 bpm, and 140 ms for HR >130 bpm.



**Table below** shows normal values for the PR interval in different age ranges for HRs between 70 and 90 bpm.

**Normal values of the PR interval in the different age ranges (HR values between 70 bpm and 90 bpm)**

<b>Newborn babies</b>	<b>70 to 140 ms</b>
<b>Infants</b>	<b>70 to 160 ms</b>
<b>Children</b>	<b>100 to 160 ms</b>
<b>Teenagers</b>	<b>100 to 180 ms</b>
<b>Adults</b>	<b>120 to 200 ms</b>
<b>Elderly people</b>	<b>120 to 210 ms</b>

RBBB and LAFB, old ASMI, prominent anterior forces due to old posterior lateral MI.

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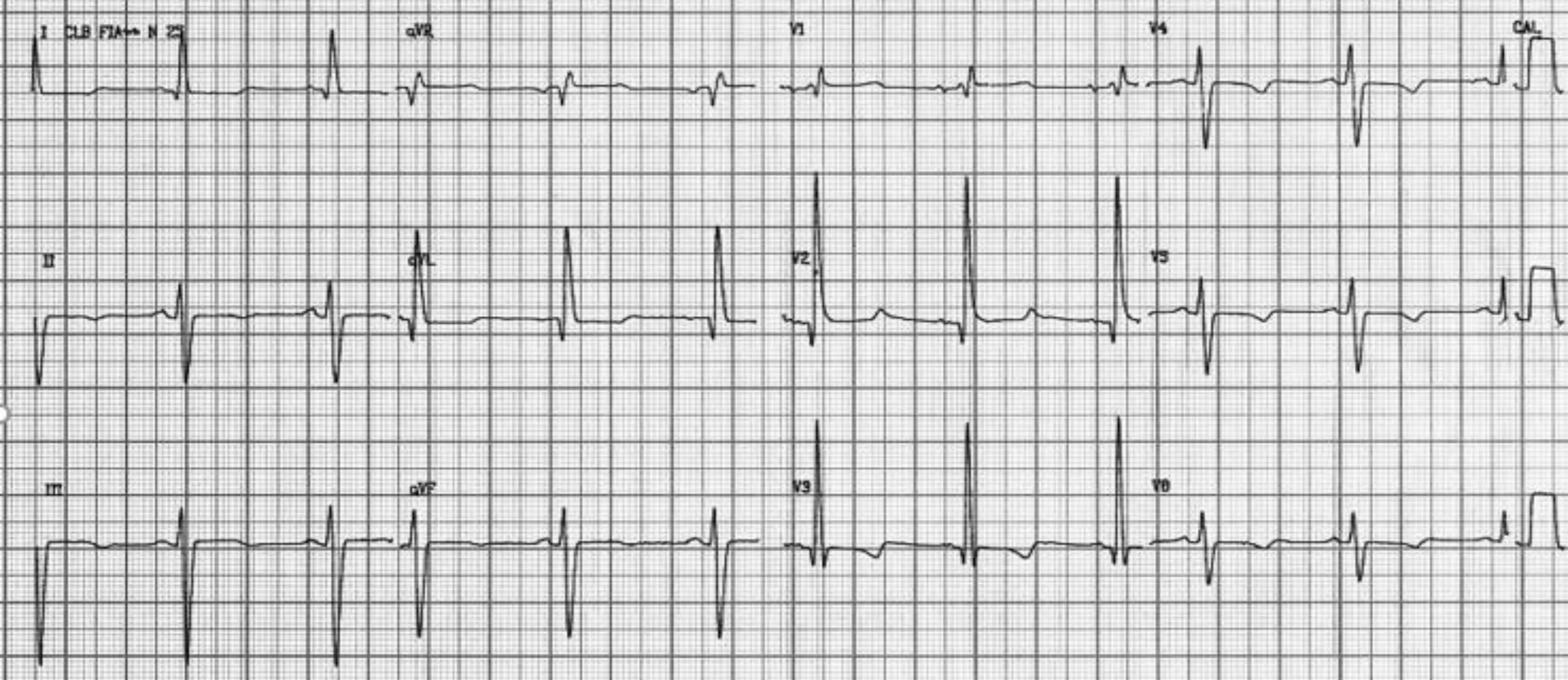
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# Final Comments



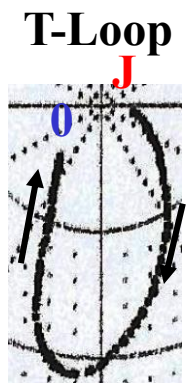
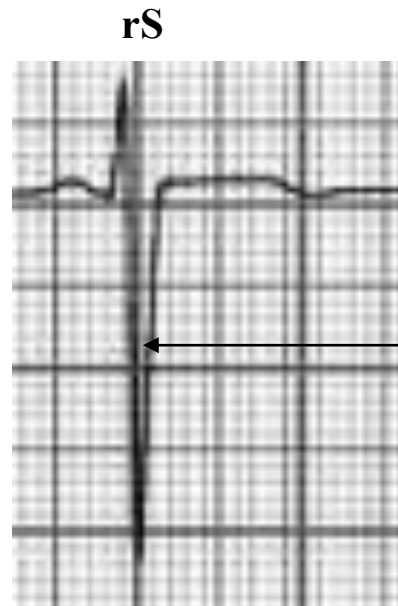
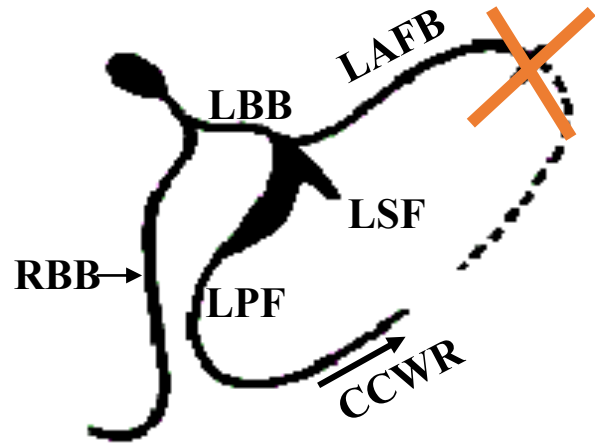
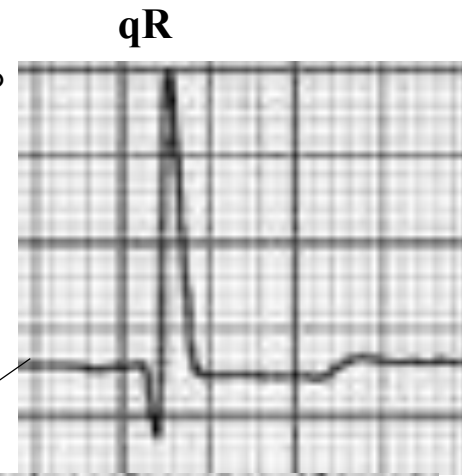
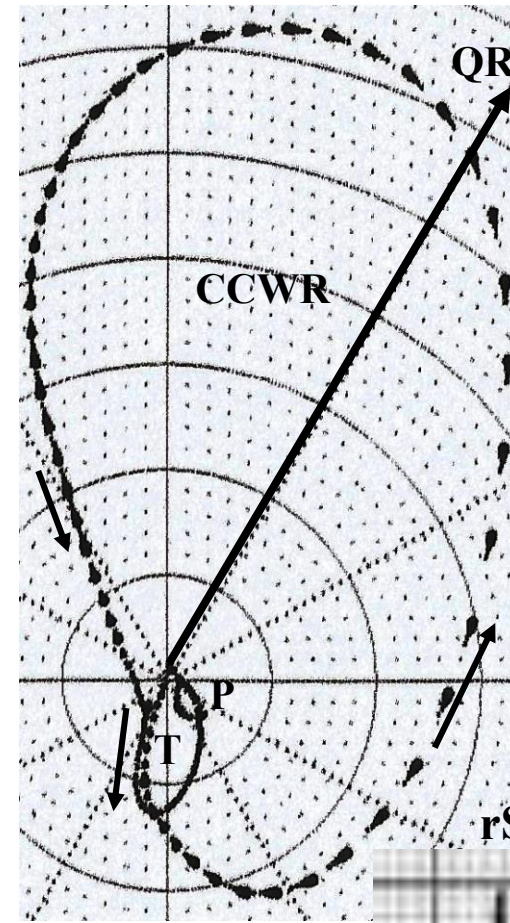
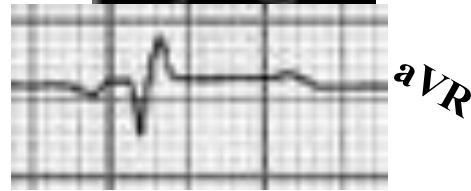
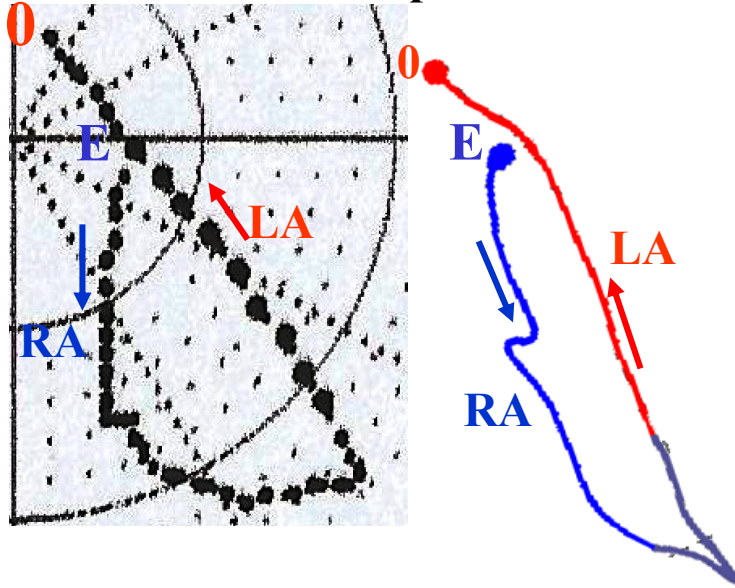


**ECG diagnosis** Extreme shift of S<sup>∧</sup>QRS in the left superior quadrant ( $-60^\circ$ ), rS in II, III and aVF,  $r_{III} > r_{II}$  (it indicates CCW rotation in the FP);  $S_{III} > S_{II}$  (this criterion differentiates it from Right End Conduction Delay of the right bundle branch and SI-SII-SIII syndrome, where  $S_{II} > S_{III}$ ); qR pattern in I and aVL; prolonged R-peak time in aVL ( $\geq 45$  ms); aVR with QR pattern Left Anterior Fascicular Block. Additionally, QRS duration  $\geq 120$  ms; prominent QRS anterior forces (PAF), R wave “in crescendo” from V1 to V2, R wave of V2, is preceded by a small q wave (V2 R wave voltage  $> 15$  mm, and an apiculate shape of R’V2 wave, rS in V5-V6, initial QRS vectors directed to the back(Q in V2)} LAFB.

**Conclusion** LAFB and LSFB atypical degree of RBBB in association and LVI.

# ECG/VCG correlation on the Frontal Plane

## Normal P-Loop



**RBB:** Right Bundle Branch; **LBB:** Left Bundle Branch; **LAF:** Left Anterior Fascicle; **LAFB:** Left Anterior Fascicular Block; **LPF:** Left Posterior Fascicle; **LSF:** Left Septal Fascicle; **CCWR:** Counter-Clockwise Rotation activation

Extreme shift of  $\hat{A}QRS$  (beyond  $-30^\circ$  up to  $-90^\circ$ ).  
Some authors accept  $-45^\circ$ : LAFB

**aVL**



**R- Wave Peak Time (RWPT) in aVL  $\geq$  45 ms**

**QRS duration 120ms consequence of LAFB(+2ms) and LPFB(+2ms) + degree of RBBB**



# Frontal Plane

60ms

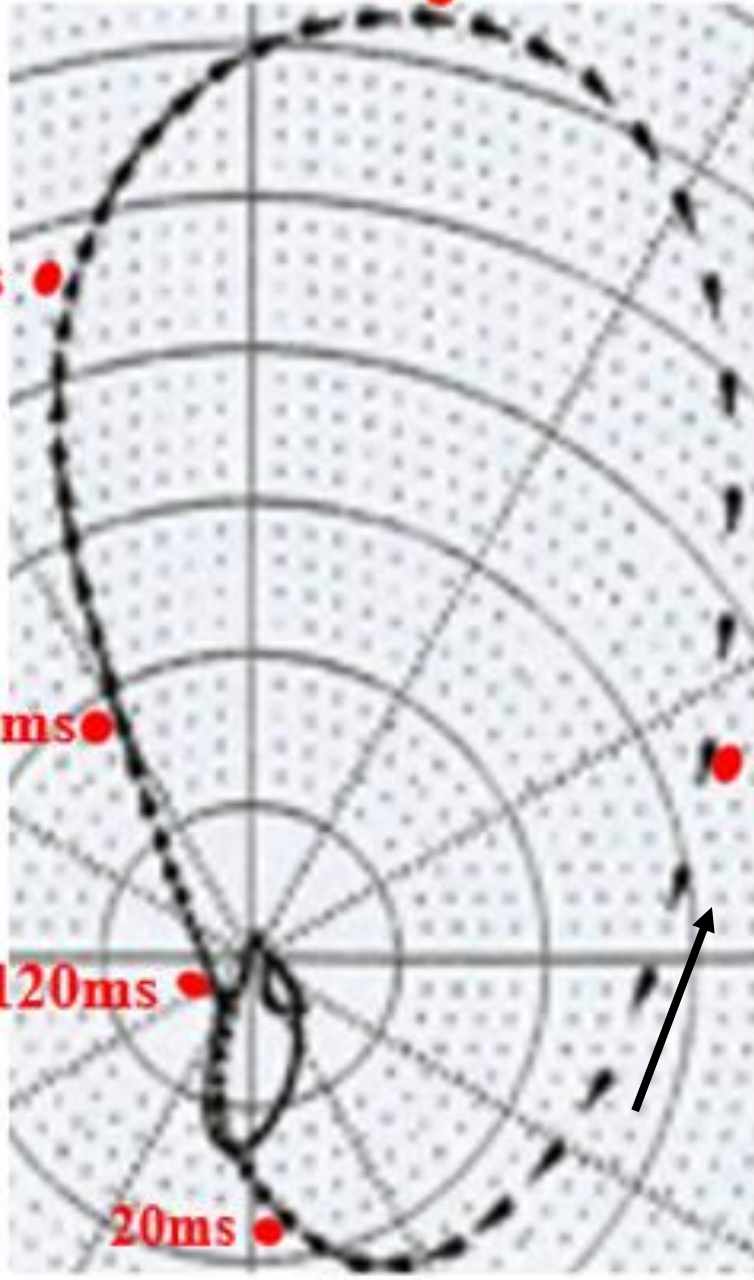
80ms

100ms

120ms

20ms

40ms



Each dash or comet represents a time of 2ms, with the present calibration of the device. In the present case we have 69 dashes or 120ms

Each dash stretches for 2ms (0.0002 s)



The greater or the lesser distance between dashes indicates the greater or the lesser conduction velocity in the area. Thus, when they are very close to each other, it indicates the presence of conduction delay. To consider the phenomenon as true, it is necessary for it to be evident in at least 2 planes.

Separate dashes = more dromotropism



Very close dashes = less dromotropism



The measurement of QRS duration should be made from the onset of the QRS wave to the J point.

<b>Fetus and post-birth days of life and adolescence</b>	<b>QRS duration measured in V5 in milliseconds (ms)</b>
<b>Between 18 and 22 weeks of pregnancy</b>	<b>47 ms</b>
<b>40 weeks</b>	<b>53 ms</b>
<b>0–1 days</b>	<b>20 to 80 (average 50)</b>
<b>1–3 days</b>	<b>20 to 70 (average 50)</b>
<b>3–7 days</b>	<b>20 to 70 (average 50)</b>
<b>Adolescence</b>	<b>70 ms to 90ms</b>
<b>Adults</b>	<b>Until 100ms</b>
<b>Elderly</b>	<b>Until 110ms</b>

## ECG diagnostic criteria of LAFB in the Frontal Plane (**Surawicz 2009; Willems 1985; Ioan 1988**)

### Frontal plane:

- 1) Extreme shift of SÂQRS in the left superior quadrant (beyond  $30^\circ$  up to  $-90^\circ$ ). Some authors accept between  $-45^\circ$  and  $-90^\circ$  (**Elizari 2012**);
- 2) rS in II, III and aVF. If SÂQRS was in  $-30^\circ$  II would be R = S;
- 3)  $r_{III} > r_{II}$  (it indicates CCW rotation in the FP). The voltage of the r waves is 3 to 5 mm in average;
- 4)  $S_{III} > S_{II}$ : this criterion differentiates it from ECD of the right branch and SI-SII-SIII syndrome, where  $S_{II} > S_{III}$ ;
- 5) qR pattern in I and aVL;
- 6) Frequent notch of the descending limb of R wave in I and aVL (this sign would be present in 80% of the cases);
- 7) R-peak time or intrinsicoid deflection in aVL  $\geq 45$  ms;
- 8) aVR always begins with q or Q wave: qR, QR or Qr. QS is rare;
- 9) Possible notch in R wave of aVR;
- 10) QRS duration less than 120 ms when isolated.

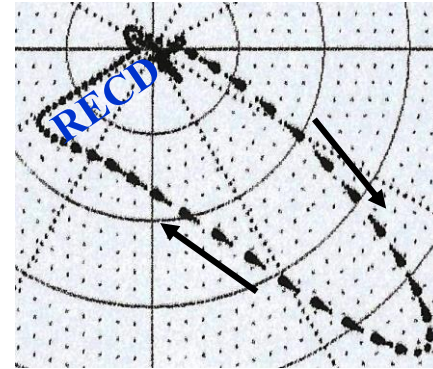
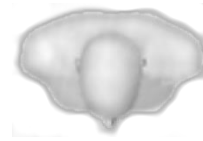
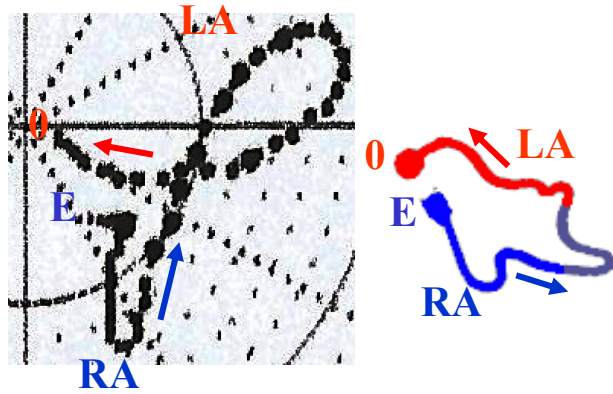
## VCG criteria of LAFB in the Frontal Plane (**Longo 1981**)

### Frontal Plane (**Ramalhão 1976**):

- 1) QRS loop  $\leq 100$  ms( when isolated). When intermittent, the increase does not exceed 20 ms;
- 2) Constant counterclockwise rotation of QRS loop when not associated to other diseases; in the presence of LVE or AMI, the rotation of the initial 20 ms may change;
- 3) Vector of initial 10 to 20 ms heading below and rightward;
- 4) Maximal vector of QRS heading above around  $-20^\circ$ ;
- 5) QRS loop located in the left superior quadrant;
- 6) ST/T loop always normal in non-complicated LAFB.

# ECG/VCG correlation on the Horizontal Plane

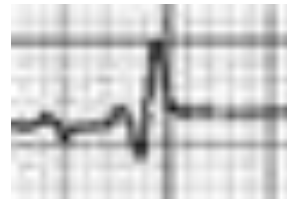
## Normal P-Loop



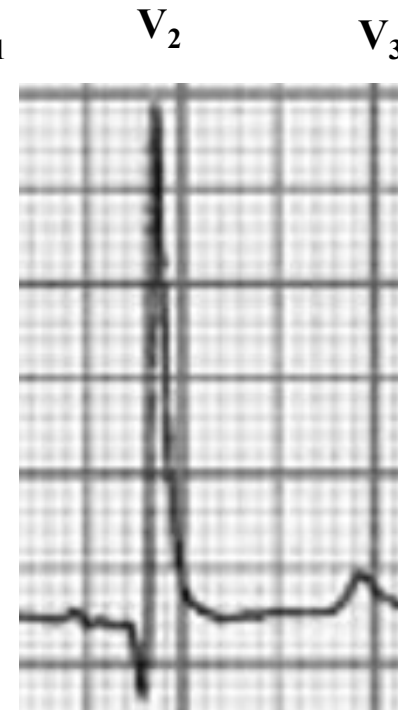
X V<sub>6</sub>

V<sub>5</sub>

V<sub>4</sub>

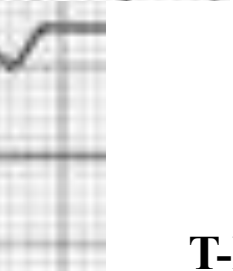
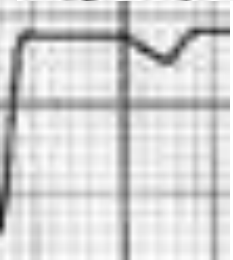
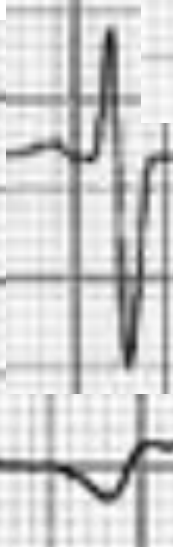
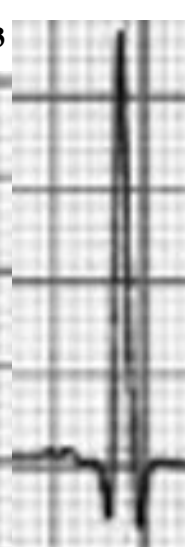


V<sub>1</sub>

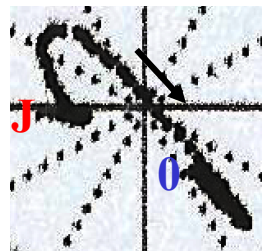


V<sub>2</sub>

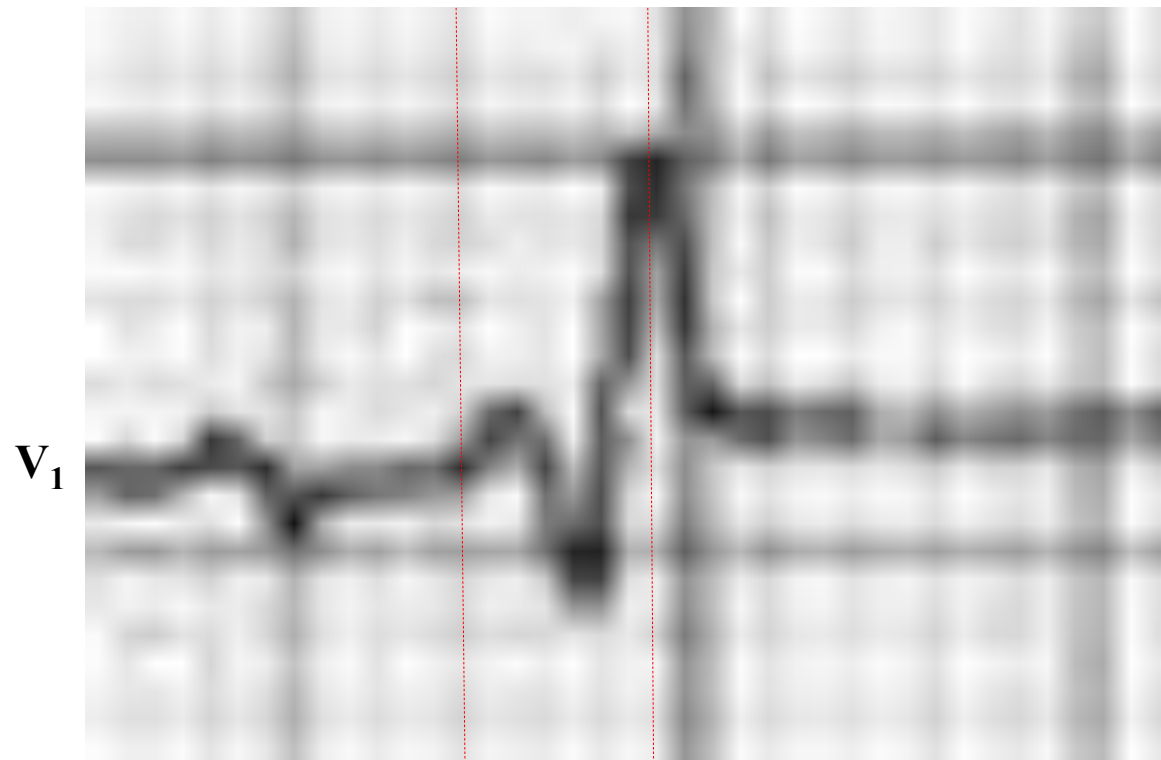
V<sub>3</sub>



## T-Loop



Prominent QRS anterior forces (PAF), initial QRS vectors directed to back (Q in V<sub>2</sub>), R wave “in crescendo” from V<sub>1</sub> to V<sub>2</sub>, R wave of V<sub>2</sub> presents a great voltage and is preceded by a small q wave (V<sub>2</sub> R wave voltage > 15 mm), and has an apiculate shape of R wave, rS in V<sub>5</sub>-V<sub>6</sub>, and QRS loop located predominantly in the anterior left quadrant, absence of initial q wave in V<sub>5</sub> and V<sub>6</sub> (by absence of septal vector 1<sub>AM</sub>) = Left Septal Fascicular Block. Additionally, Right End Conduction Delay (RECD) located on anterior right quadrant, triphasic QRS pattern in V<sub>1</sub>(rsR'), very broad R-Wave Peak Time (RWPT) in V<sub>1</sub>, and QRS duration ≥ 120ms: LSFBS + degree of RBBB.

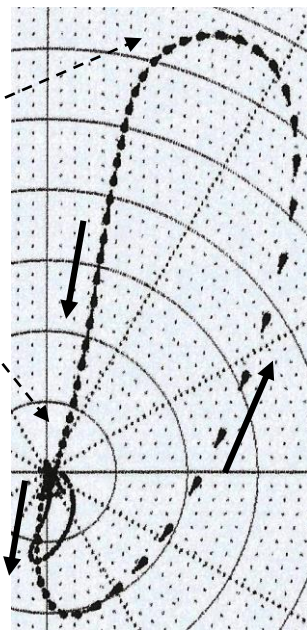


**Very broad R-Wave Peak Time(RWPT) in V<sub>1</sub> > 70 ms (=110ms) suggesting BRBB LSFb in association**

# ECG/VCG correlation on the Right Sagittal Plane (RSP)



End Conduction Delay located on anterior and superior quadrant

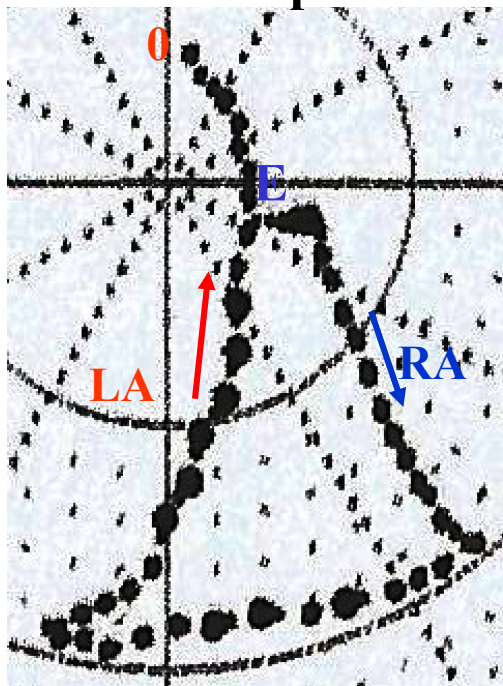


V<sub>2</sub>

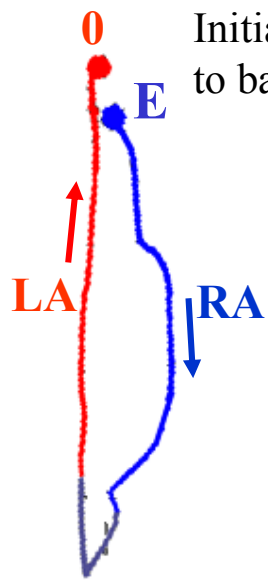


R wave of V<sub>2</sub> presents a great voltage and is preceded by a small q wave (V<sub>2</sub> R wave voltage > 15 mm), and has an apiculate shape of R wave typical of LSFB

**P-loop**



Z



Initial 10 to 20 ms QRS heading to back and inferiorly

**Y**  
aVF



**T-loop**



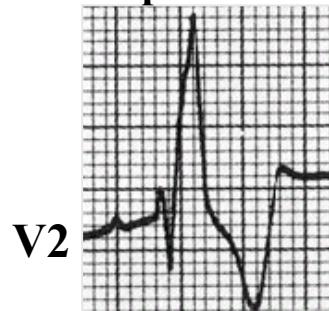
VCG performed with DMS technology

RSP: clockwise

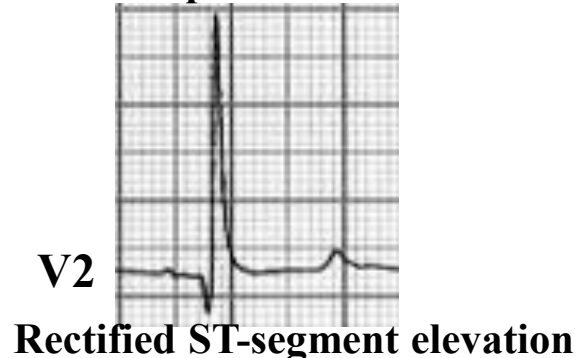
## Final conclusion

- I. LAFB
- II. LSFB
- III. Some degree of RBBB in association
- IV. J-point and rectified ST-segment elevation in leads V1-V2, suggesting ischemic phenomena because in uncomplicated RBBB ST-segment in right precordial leads is depressed.
- V. LVH

**Uncomplicated RBBB**



**The present case**



Why the prominent anterior QRS forces (PAF) could not be caused by dorsal or lateral walls sequela of myocardial infarction?

**Answer:** because in the case of dorsal MI, this wall does not exist as demonstrated by numerous publications of Professor Bayés, endorsed by a consensus of experts published in the Circulation journal(1). Finally, the PAF could not be caused by lateral MI sequela because clearly the echocardiogram does not reveal hypokinesia in any wall.

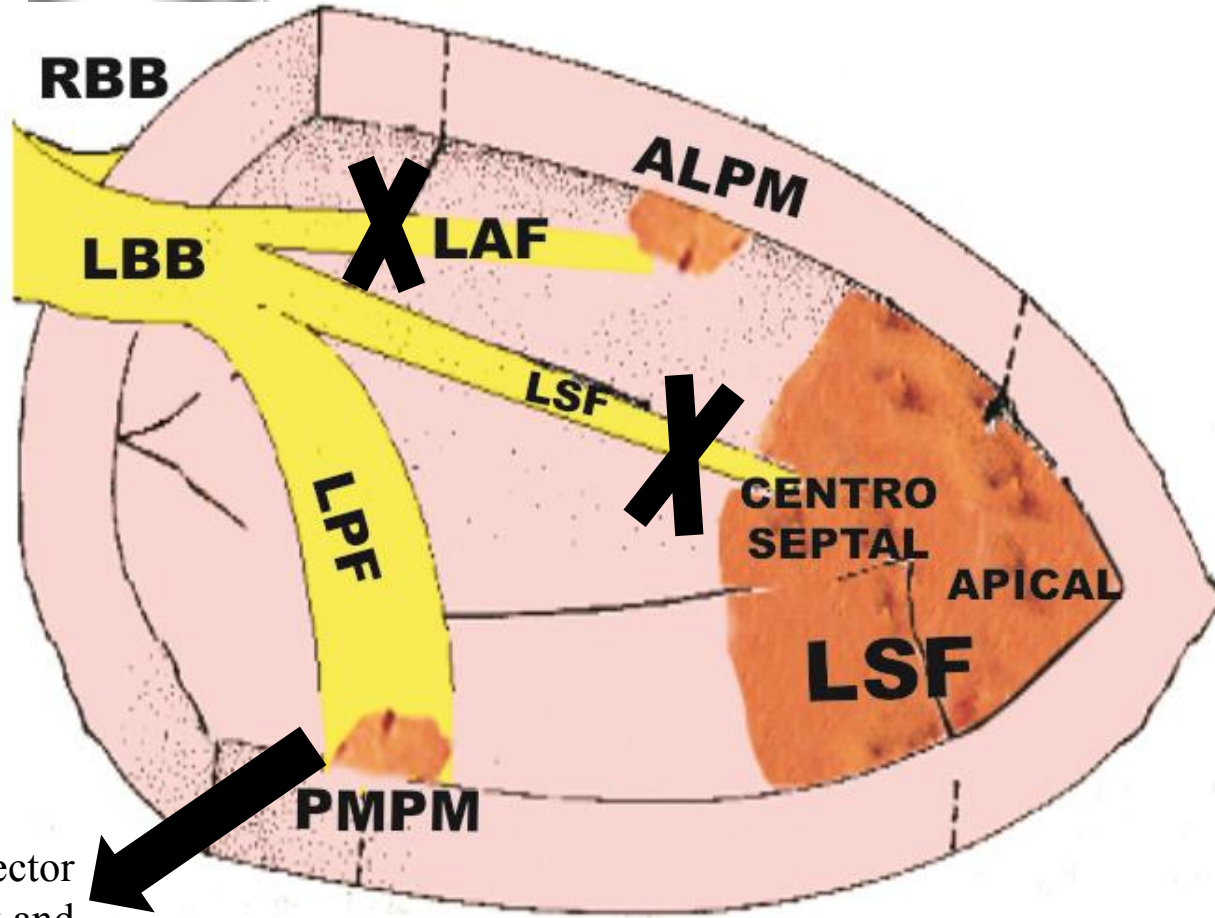
Why not anteroseptal MI?

Answer: because initial q-waves in V2-V3 have duration <40 ms. Additionally, the q-wave is caused by the vector of left posterior fascicle, because the vectors of left anterior fascicle (LAF) and left septal fascicle (LSF) are blocked. The vector-dependent of left posterior fascicle (LPF) is heading backward, below and to the right. See ludic explanation in the next slide.

1. Bayés de Luna A, Wagner G, Birnbaum Y, Nikus K, Fiol M, Gorgels A, Cinca J, Clemmensen PM, Pahlm O, Sclarovsky S, Stern S, Wellens H, Zareba W; International Society for Holter and Noninvasive Electrocardiography. A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. Circulation. 2006 Oct 17;114(16):1755-60.

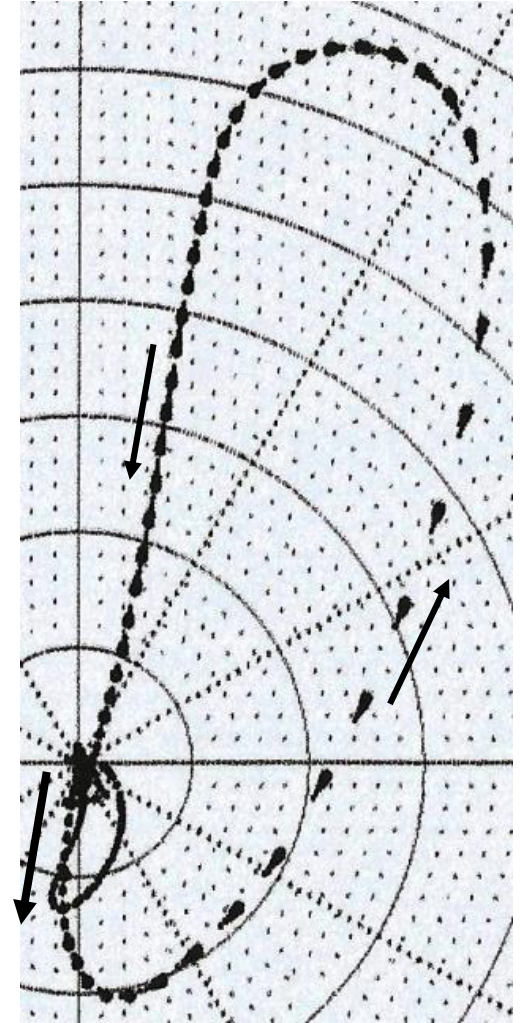


### RIGHT LATERAL VIEW MAJOR AXIS



First LPF vector directed to back and downward

**ALPM:** anterolateral papillary muscle of mitral valve;  
**PMPM:** posteromedial papillary muscle of mitral valve



First LPF vector directed to back and downward

**Y**  
**aVF**

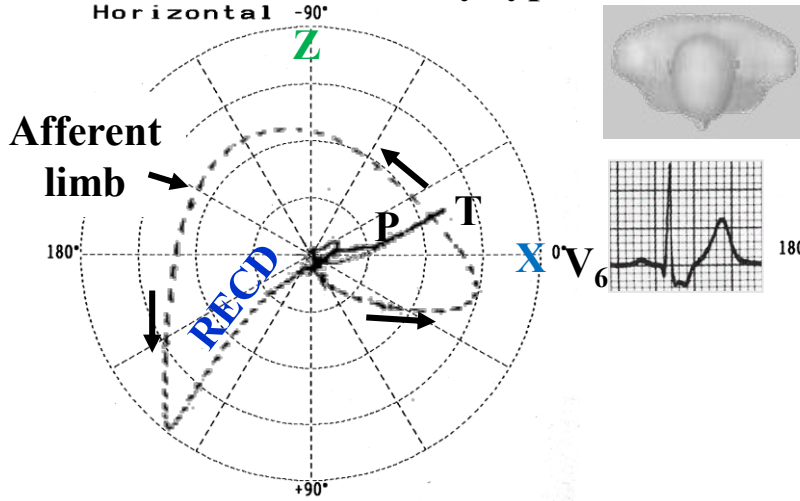


## Possible causes for Prominent QRS Anterior Forces (PAF)

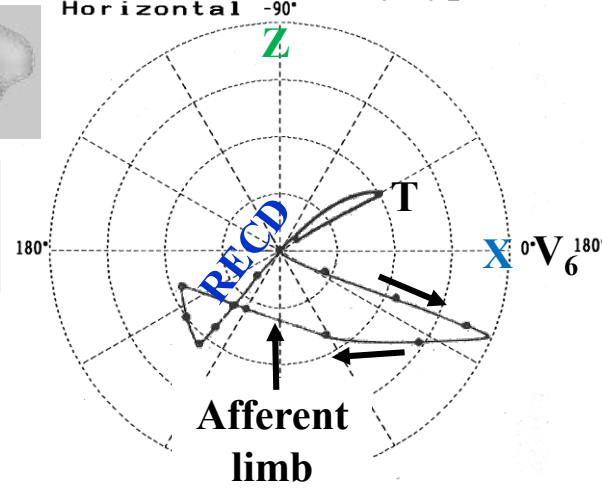
1. **Normal subjects:** PAF are observed in only 1% of normal subjects (**Mattu 2001**).
  1. Normal variant with marked counterclockwise rotation of the heart around the longitudinal axis of the heart resulting in a shifting of the transition area (R=S) early, i.e. to the right of the precordial lead V<sub>2</sub> (**Yanagisawa 1981; Mori 1992; Paparella 1987**).
  2. Athlete's heart (**Ferst 1984; Zema 1990**).
2. **Misplaced precordial leads** (**MacKenzie 2004; Mattu 2001**).
3. **Ancient strictly posterior, dorsal, high posterobasal MI** (**McManus 2014**). Actual lateral MI (**Bayés de Luna 2006**);
4. **Right ventricular hypertrophy (RVH): vectorcardiographic types A** (**Brohet 1990; Suzuki 1978; Hugenholtz 1964**) and B (**Lebowitz 1986; Ellison 1972**);
5. **Diastolic LVH, volumetric or eccentric LVH, secondary to septal hypertrophy** Increase magnitude of  $I_{AM}$  vector) and CCW heart rotation around the longitudinal axis (**Cabrera 1960; Donoso 1955**);
6. **Combined or biventricular hypertrophy** (**Elliott 1963**);
7. **CRBBB** (**Baydar 1965; Chen 1980**);
8. **Pre-excitation variant of Wolff-Parkinson-White type A** (**Chung 1965**);
9. **HCM: both obstructive and non-obstructive forms** (**Pérez-Riera 2013**);
10. **Progressive muscular dystrophy of childhood, Duchenne's cardiomyopathy, Duchenne's muscular dystrophy, X-linked muscular dystrophy, pseudo-hypertrophic muscular dystrophy, childhood muscular dystrophy** (**Secchi 1982; Yotsukura 1999**);
11. **Endomyocardial fibrosis** (**Tobias 1992**);
12. **Dextroposition. Example: left pneumonectomy** (**Pérez Riera 2011**).
13. **LSFB**;
14. **A combination of the above: this is the present case.**

# VCGs patterns of Complete Right Bundle Branch Block in the Horizontal Plane

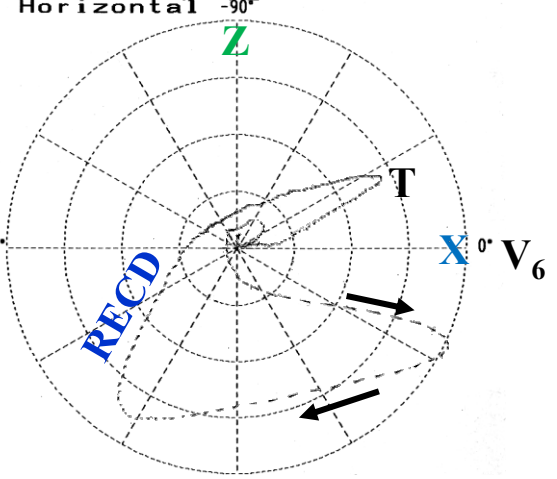
## Grishman or Kennedy type I



## Cabrera or Kennedy type II



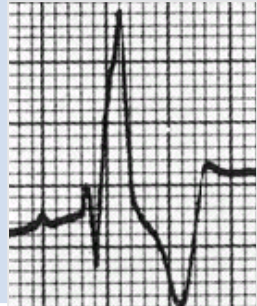
## Kennedy type III or C

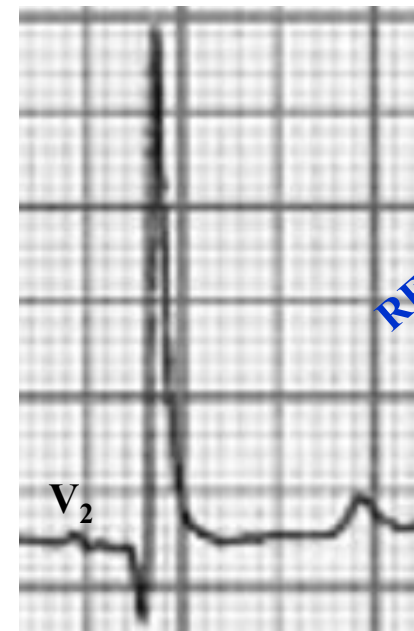


**RECD: Right End Conduction Delay**

Initial vector to the front, QRS loop of CW rotation and main body located in anterior quadrants.

## The present case

	RBBB Kennedy type III	The present case
Initial 10 to 20ms	To front	To back qR in V2-V3
QRS loop location	Predominantly on <b>right</b> anterior quadrant	Predominantly on <b>left</b> anterior quadrant
QRS pattern and shape in V2		R wave of a great voltage preceded by a small q wave. R wave voltage > 15 mm), and has an apiculate shape.



Initial 10 to 20ms directed to back

