ECG & VCG in Left Posterior Fascicular Block and its differential diagnosis: our point of view

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Left Posterior Fascicular Block (LPFB): possible causes (Elizari 2007; Hecht 1973; Rosenbaum 1973)

It is the most rare block of all intraventricular blocks. Very rare without association with others blocks.

1) Coronary artery disease (**Rizzon 1975**): LPFB is a rare but clinically important intraventricular conduction disturbance. Its appearance is reliably connected with IMI and generally reflects severe three-vessel CAD, requiring invasive investigation (**Godat 1993**).

   (2a) During the acute phase of ischemia (**Patanè 2009**). Or transient during exercise treadmill testing (**Madias 1999**).

   (2b) During the acute phase of infarction: 0.2% to 0.4% (**Demoulin 1979**). A case of transient LPFB and various intraventricular conduction disturbances associated with acute anterolateral infarction was reported by Ogawa et al (**Ogawa 1976**).

   (2c) LPFB and posteroinferior myocardial infarction accounted for Q waves in leads II, III and aVF. However, R amplitude in these same leads is increased after LPFB but decreased after posteroinferior myocardial infarction. The mean QRS axis in the frontal plane was shifted toward the vertical in LPFB but little changed or shifted slightly away from the vertical in posteroinferior myocardial infarction. When LPFB and posteroinferior myocardial infarction coexist, there may be masking, imitation or enhancement of the effects of one lesion by the presence of the other (**Watt 1982**).

3) Lenègre disease, progressive cardiac conduction defect (PCCD) or “idiopathic” sclerosis of the intraventricular His system: by mutation in the SCN5A gene, the same one affecting Brugada Syndrome.

4) Lev disease or progressive idiopathic sclerosis of the “cardiac skeleton”. With a clinical behavior similar to Lenègre disease, however, it occurs in elderly patients;

5) Aortic insufficiency: attributed to the mechanical effect of jet regurgitation on the posterior portion of the left septum, the site that the thick LPF goes through (LV inflow tract);

6) Aortic stenosis;

Left Posterior Fascicular Block (LPFB): possible causes (Elizari 2007; Hecht 1973)

7. Aortic stenosis associated with aortic insufficiency;
8. Supravalvar aortic stenosis;
9. Coarctation of the aorta;
10. Dissecting aortic aneurysm;
11. Massive calcification of the “cardiac skeleton”;
12. Chronic chagasic myocarditis: the most frequent one in Latin America.
13. Cardiomyopathies, myocarditis and infiltrative myocardial diseases;
14. Systemic hypertension;
15. Interventricular septum tumor (**Cola 1992**);
16. Hyperpotassemia;
17. Transitorily, during contrast injection in the right coronary artery and in
18. Acquired ventricular septal defect: in cases of inferior wall myocardial infarction, complicated by rupture of the inferior septum, resulting in isolated LPFB. (**Rokey 1984**)
19. Acute pulmonary embolism?
20. Hereditary: pseudo LPFB? (**Lorber 1988**)

Causes of greater vulnerability of the Left Anterior Fascicle (LAF) in comparison to the Left Posterior Fascicle (LPF)

1) Anatomical: (**Rosenbaum 1970**.)

a) Less diameter (LAF: 3 mm; LPF: 6 mm).

b) Greater extension (LAF: 35 mm; LPF: 30 mm).

2) Electrophysiological:

As a consequence of its greater extension and less diameter, the depolarization and repolarization of LAF is slower than LPF, i.e. the "QT of LAF" is greater than the one of LPF, a fact that makes it more vulnerable.

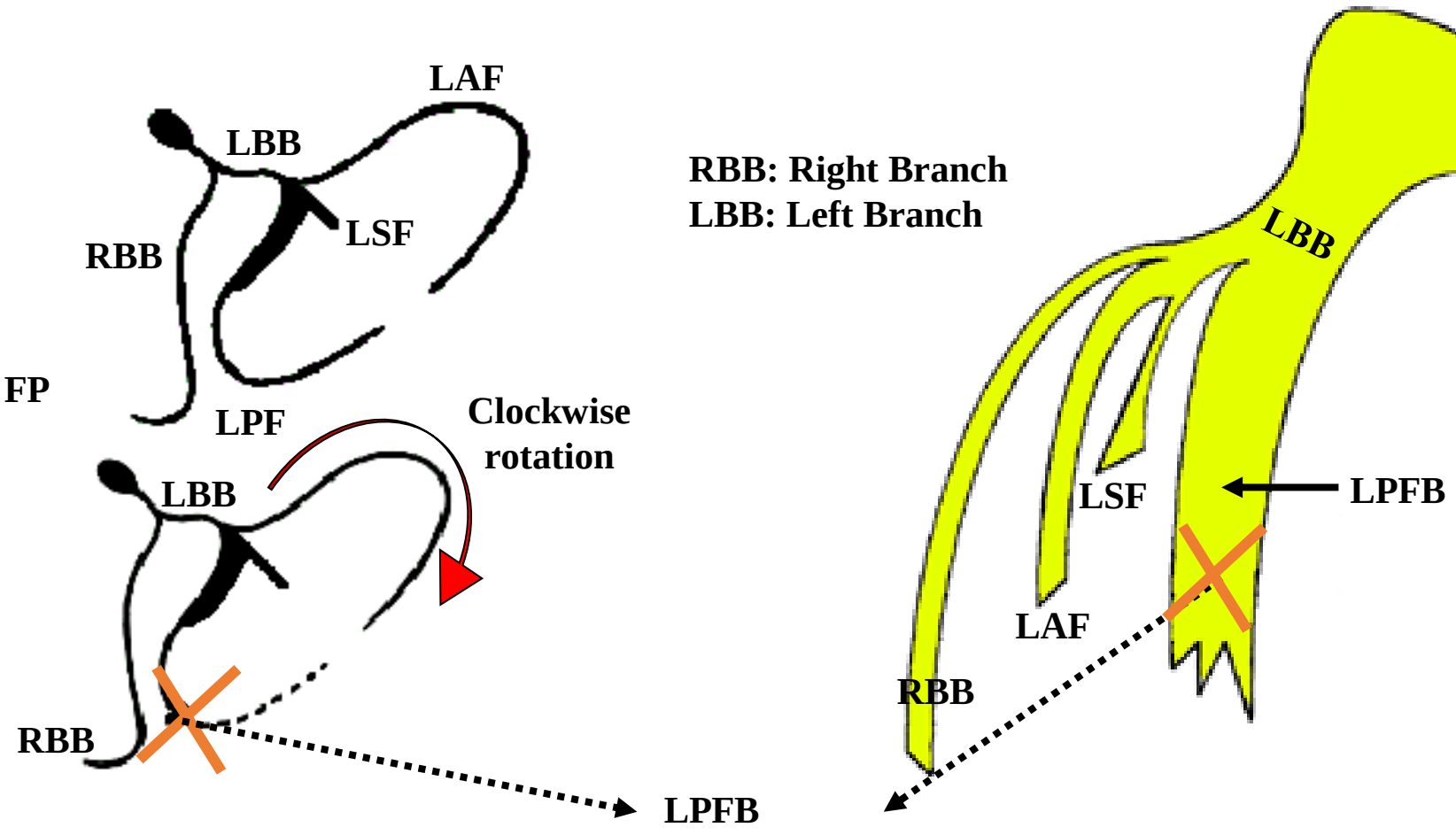
3) Vascular:

Posteroinferior fascicle always irrigated by the two systems of the ADA and RCA.

4) Topographic:

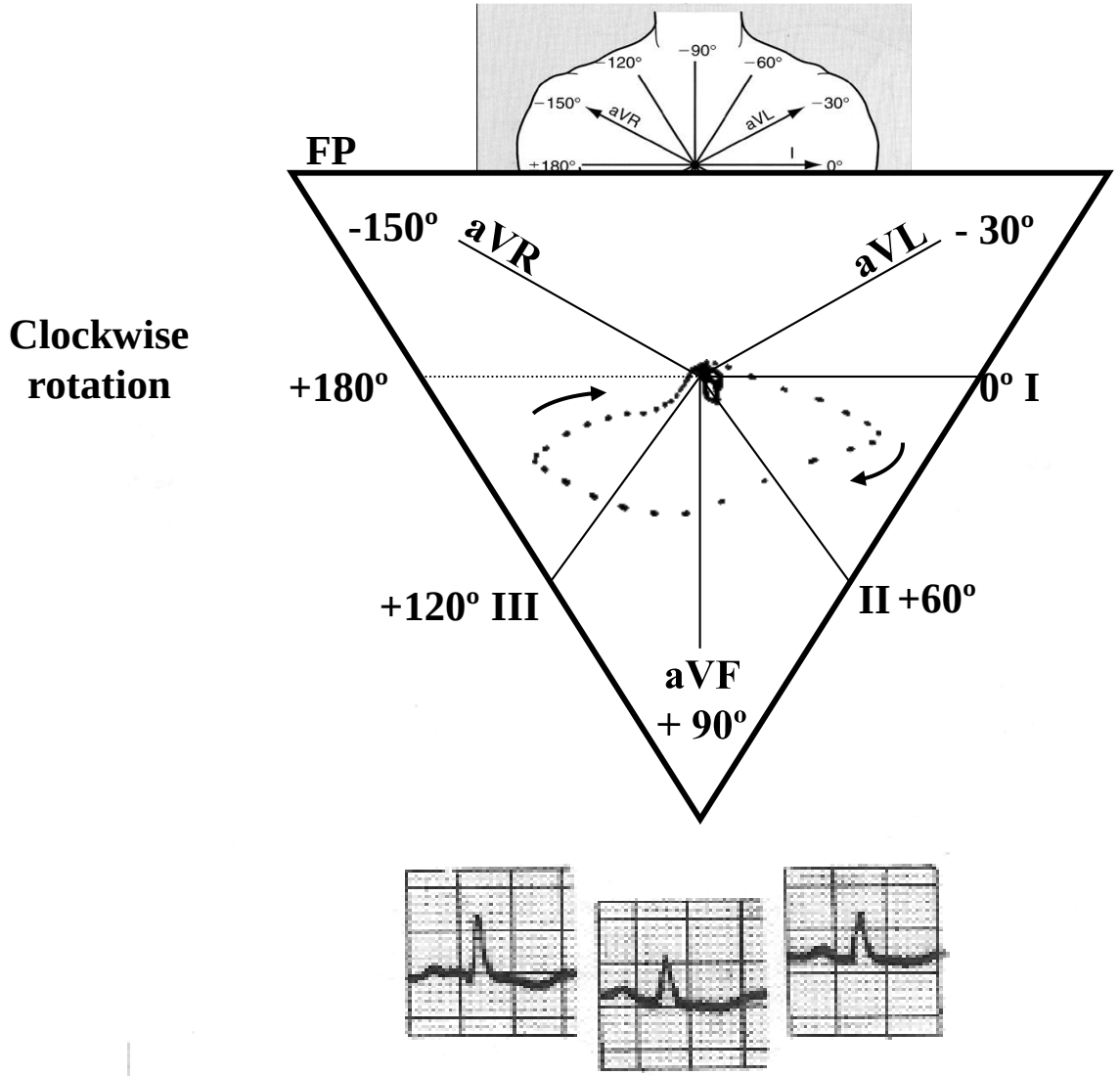
The LPF runs through a more protected area, with less pressure mechanic impact. The LAF runs diagonally through the Left Ventricle Outflow Tract (LVOT) by the subendocardium. This region is subject to a great turbulence and high pressure, which justifies the greater vulnerability of the LAF when compared to the LPF, which runs through an area in the LV Inflow Tract (LVIT), which is much less exposed to turbulence, which explains the rarity of the LPFB.

Left Posterior Fascicular Block (LPFB)



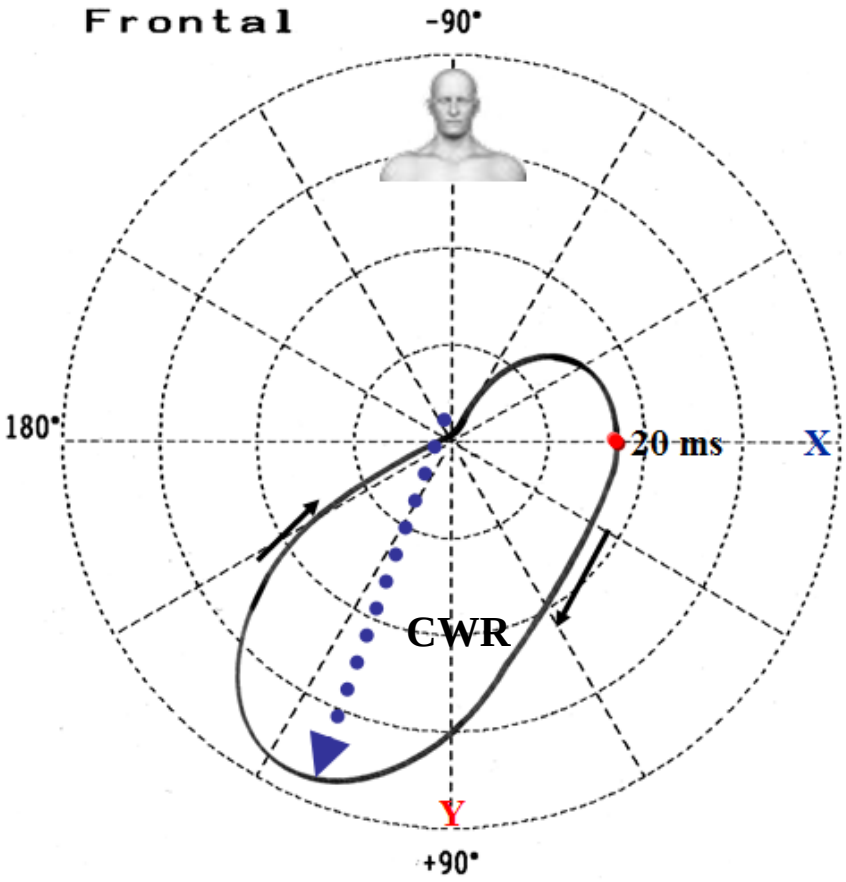
Outline of activation with clockwise rotation in the frontal plane in the LPFB.

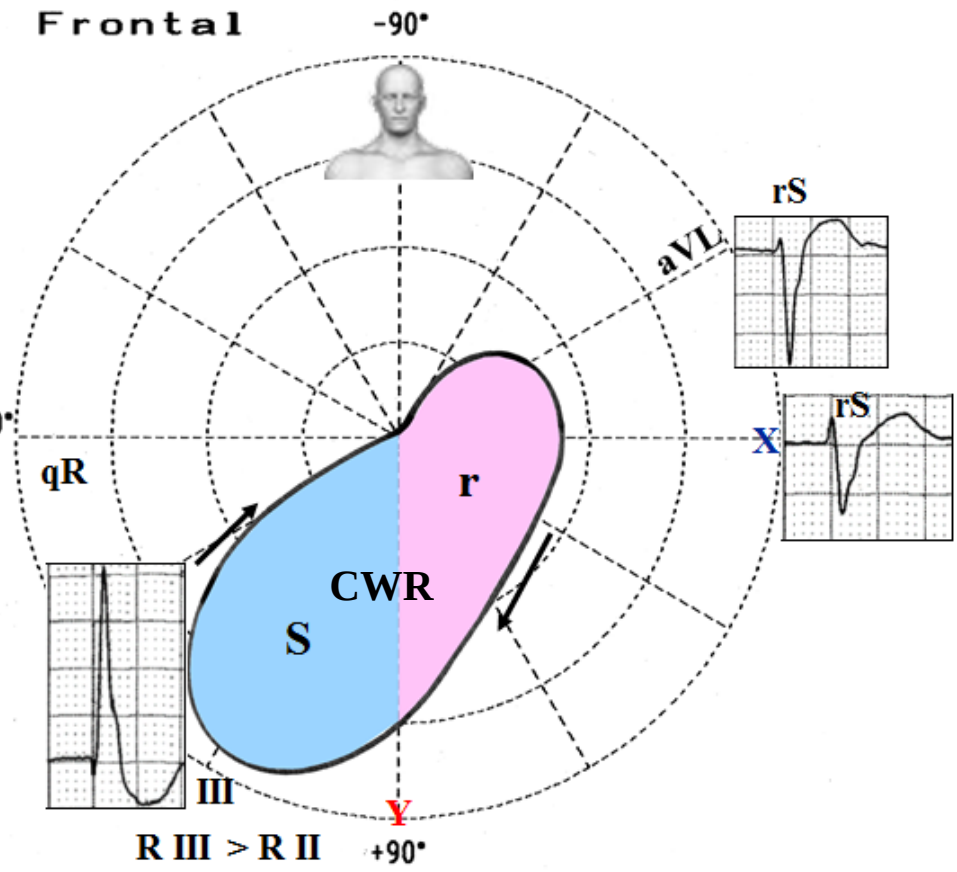
Vectorial representation of ventricular activation in LPFB in the Frontal Plane



Typical QRS loop in the frontal plane in LPFB. See the clockwise rotation, the "broad" aspect of the QRS loop and the discrete shift of the axis to the right.

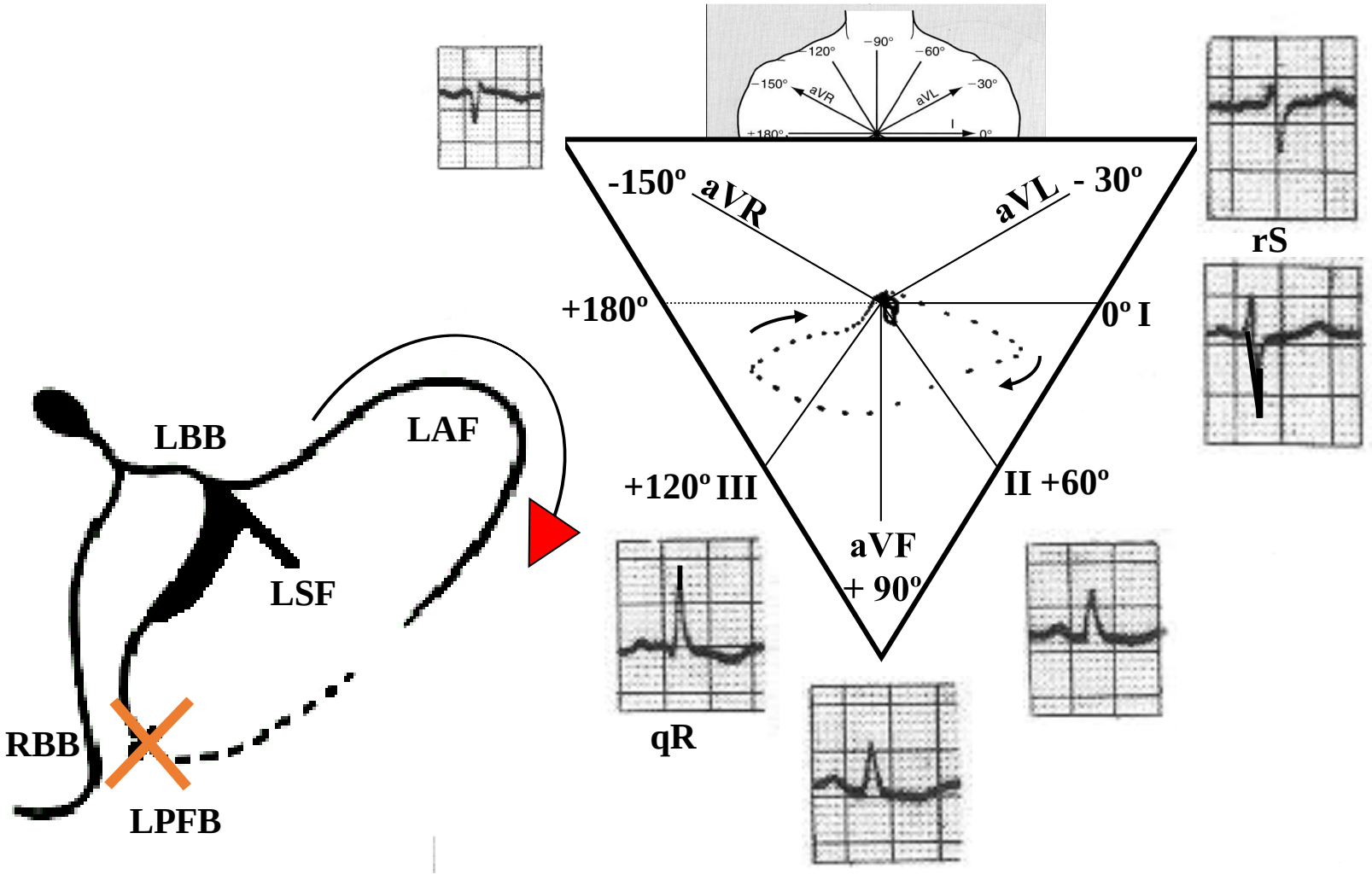
ECG/VCG correlation in LPFB: QRS loop in the FP





Characterization of QRS loop in the frontal plane: Vector of initial 20 ms heading above and to the left; efferent limb to the left; clockwise rotation (CWR); greater area of QRS loop located in the right inferior quadrant; maximal vector heading below and to the right near +110º (from +80º to +140º); QRS loop of "broad" aspect (“fat” loop); afferent limb located in the right inferior quadrant. Typical QRS loop in the frontal plane that explains the rS pattern in I and aVL. Typical QRS in the frontal plane that explains the qR pattern in III with notch in the descending limb of the R wave and R wave in III > R in II. Notch in the descending limb of the R wave in III (middle-final notch).

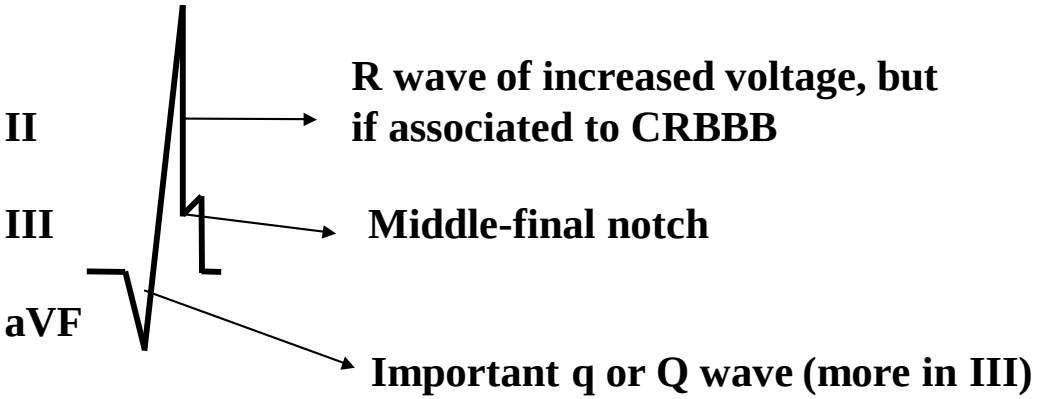
LPFB: ECG-VCG correlation in the FP



ECG/VCG correlation in LPFB in the FP: rS in I and aVL; qR in II, III and aVF; QRS loop of CW rotation with the axis shifted to the right.

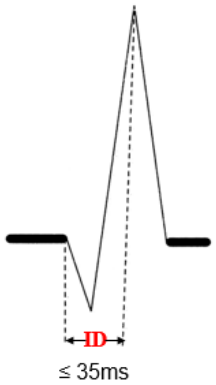
ECG criteria of LPFB in the Frontal Plane (Palmieri 1974; Medrano 1972)

1) Frontal plane axis between +90 and 180 degree in adults;
2) rS pattern in leads I and aVL
3) qR pattern in III, aVF and II: Q wave is always present in III and may be small or absent in II or aVF.
4) Notch in the descending limb of the R wave in III (middle-final notch);
5) RIII > RII: SÂQRS closer to +120º (III) than +60º (II), when closer to the latter, it would indicate an incomplete form of LPFB. RIII > 15mm.
6) The q wave in III is always greater than the q wave in II and aVF. If there is association with inferior infarction, the Q wave > 40 ms.
7) QRS duration less than 120 ms if isolated (without RBBB)



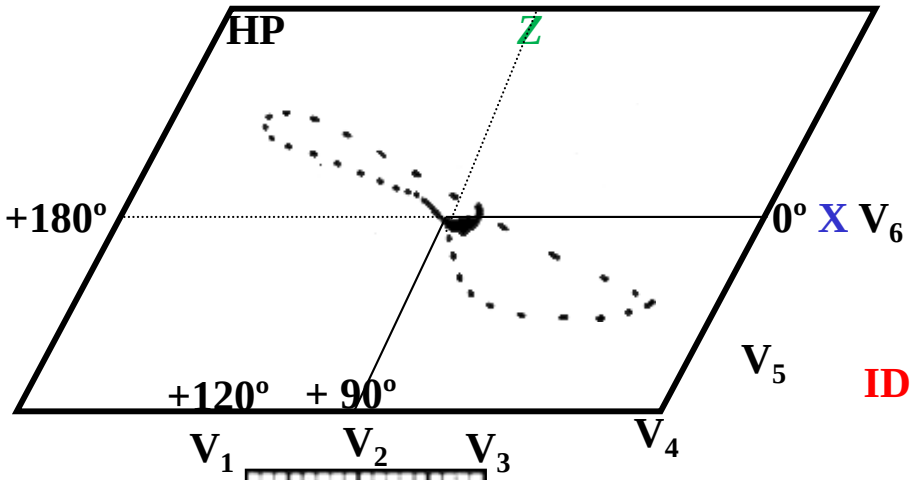
8) Ventricular activation time, R-peak time or intrinsicoid deflection (ID) in aVF :≤ 35 ms.
(Rusconi 1980)

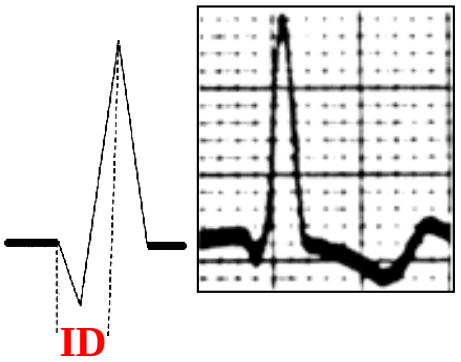
Time of appearance of R wave apex: "R-peak time"



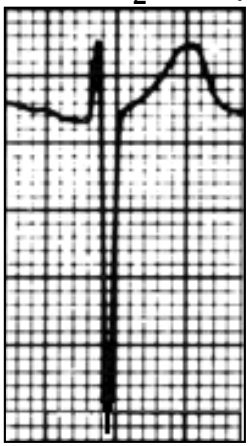
ECG criteria of LPFB in the HP

1) V_1 and V_2: rS pattern, QS rarely.
2) S wave of V_2 -V_3 very deep by posterior dislocation and to the right of the final forces.
3) Scant progression of growth of r wave in precordial leads: dislocation to the left of the transition area.
4) V_5 and V_6: qRs or Rs patterns.
5) Increased intrinsicoid deflection of V_5 and V_6 (> 45 ms to 50 ms)
6) Disappearance of q wave in V5 and V6 when LPFB occurs.





ID of V_5 and V_6 > 45 to 50 ms



S wave of V_2 very deep

ECG/VCG correlation of the QRS loop in the horizontal plane related to the V2 and V6 leads. In V2, deep S wave and in V6 intrinsicoid deflection > 45 ms to 50 ms.

VCG criteria for LPFB (Brohet 1977)

Frontal Plane:

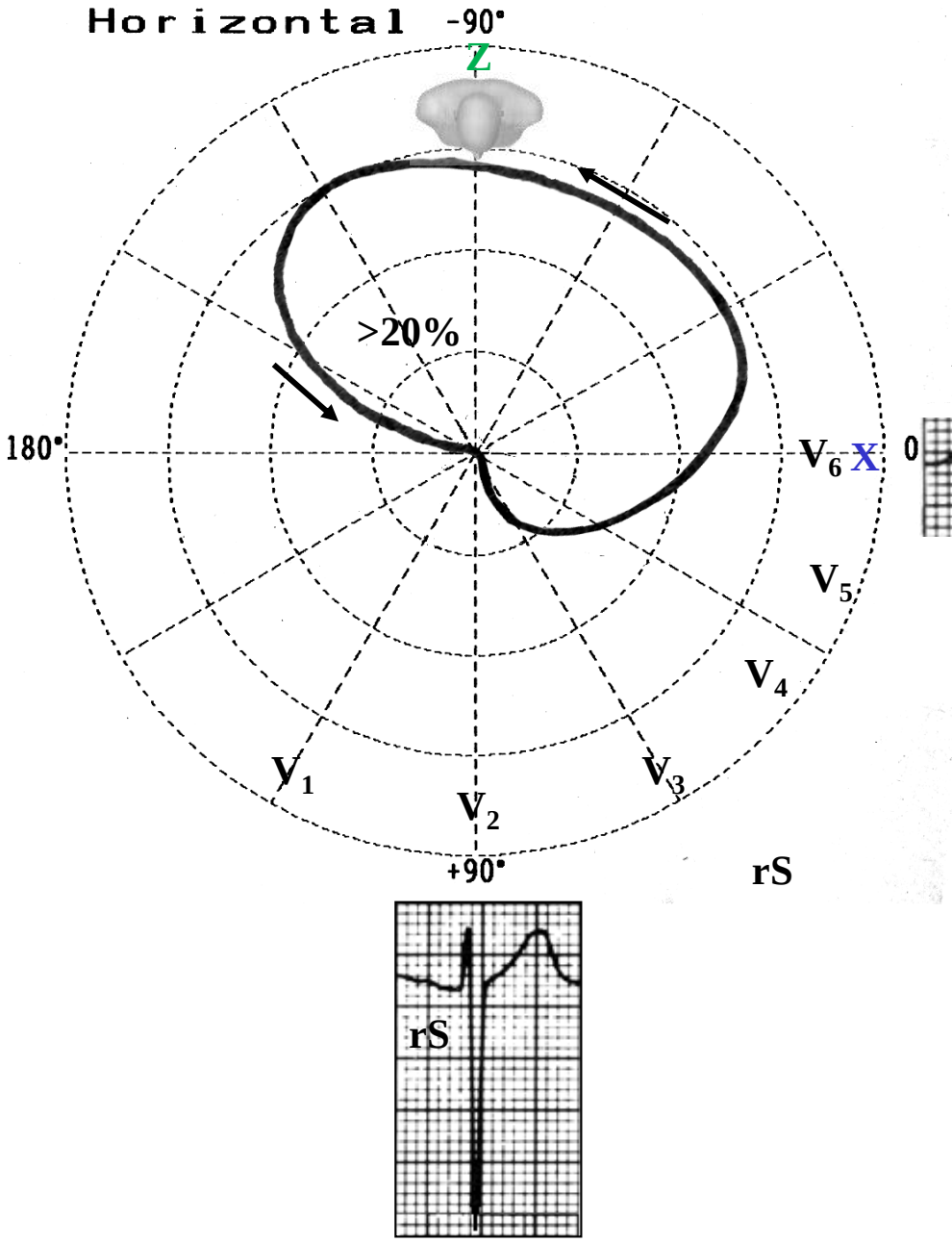
- Vector of initial 10 to 20 ms heading above and to the left (near -45°) with possible delay (initial 10 to 25 ms). If associated to inferior infarction, superior initial forces of 25 ms or more (more than 12.5 dashes above the orthogonal X lead. 1 dash = 2 ms) (Castellanos 1972).
- Broad QRS loop, with clockwise rotation. Cooksey, Dunn and Massie said that occasionally, it may be in "eight" with a counterclockwise terminal portion (10%).
- Maximal vector near +110º (+80º to +140º)
- Almost all the loop is located below the X line (0 to ±1800) in the inferior quadrants
- 20% of the loop located in the right inferior quadrant. If there is association to CRBBB, 40% or more
- Afferent limb heading below and slightly to the left, and the efferent one to the right.
- Middle-terminal portion of the QRS loop (vector of 60 ms to 100 ms) with delay. It may possibly reach the right superior quadrant
- QRS loop duration up to 110 ms if in isolation. In association to Complete RBBB > 120 ms
- Normal ST-T vectors in isolated LPFB: T loop with clockwise rotation, heading below and to the left. If in association to Complete RBBB: alteration secondary to ventricular repolarization.

Horizontal Plane:

- QRS loop very similar to RVH of type C;
- QRS loop of counterclockwise rotation. It is admitted that the rotation could be in "eight";
- Vector of initial 10 to 20 ms heading to the front and the right or left;
- Greater area of QRS loop located in the left posterior quadrant;
- Maximal vector of QRS around -60^0 to -110^0;
- Final portions with delay (60 ms to 100 ms) and located in the right posterior quadrant;
- 20% or more of the area of the QRS loop located in the right posterior quadrant;
- T loop to the front and the left ($+60^0$) and clockwise rotation.

Vectorial representation of QRS loop of ventricular activation in LPFB in the HP

Typical QRS loop in the LPFB in the horizontal plane. The following stand out: vector from the initial 10 to 20 ms heading to the front and the left or right; precordial transition area dislocated to the left; deep S wave in V2 or V2 and V3; frequent RS in left leads V5 and V6; QRS loop similar to RVE type C; QRS loop of CCW rotation; 20% or more of the QRS loop area located in the right posterior quadrant; left precordial leads with RS pattern similar to RVH type C.



VCG criteria of LPFB in the Sagittal Plane

RSP	LSP
1) Vector of initial 10 to 20 ms to the front and above with possible delay	1) Vector of initial 10 to 20 ms to the front and above with possible delay.
2) Most of the QRS loop located in the infero-posterior quadrant.	2) Most of the QRS loop located in the infero-posterior quadrant.
3) QRS loop of clockwise rotation.	3) QRS loop of counterclockwise rotation.
4) Maximal vector around +120º (+140º to +80º).	4) Maximal vector around +120º (+140° to + 80°).
5) Constant end delay and possible initial delay.	5) Constant end delay and possible initial delay.
6) T loop heading to the front and below with clockwise rotation.	6) T loop heading to the front and below with counterclockwise rotation.

LPFB associated with complete RBBB: bifascicular block

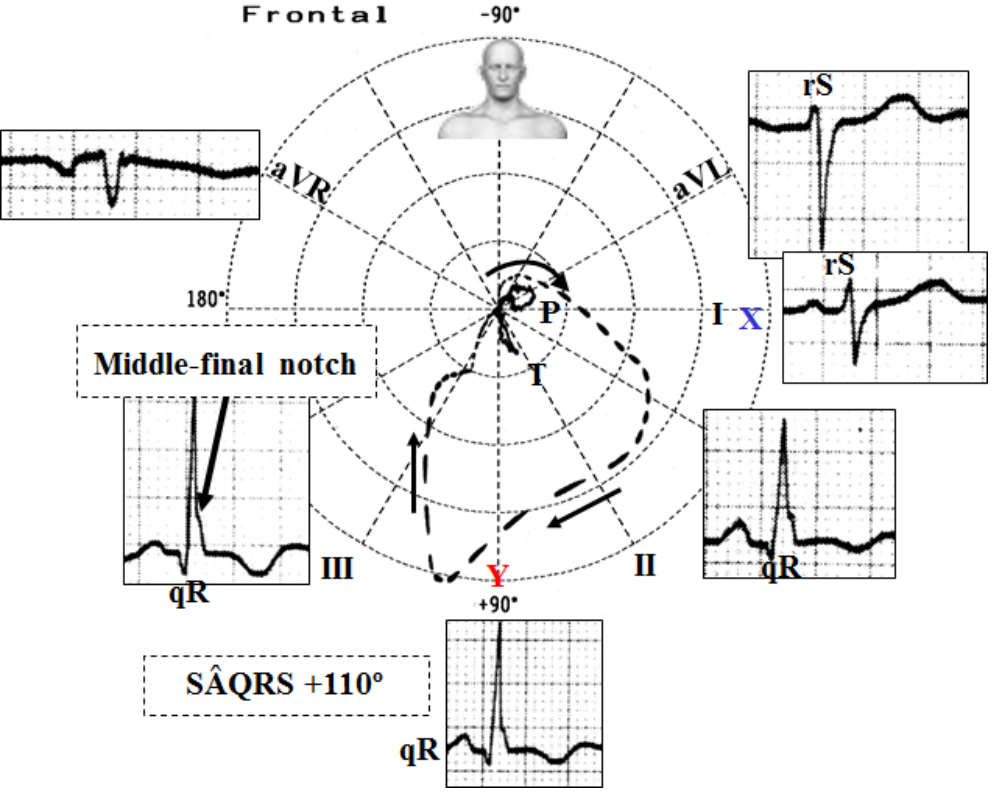
A combination of RBBB and right axis deviation of the first half of the QRS complex is indicative of concomitant LPFB, provided the other criteria for the diagnosis are met and other possible causes of right axis deviation are excluded. When RBBB and LPFB are combined, there are three main direction of the QRS forces in the FP. During the first 20ms of the QRS complex, the forces point superiorly and leftward to about -45°, resulting in small q waves in inferior leads with qIII > qII concomitantly a small r waves in leads I and aVL. During the next 40 to 60ms the forces are directed inferiorly and rightward to about +120°. These initial vectors are due to the LPFB. The terminal 40 to 60 ms QRS vector produced by RBBB, is directed to rightward toward from 150 to 180° axis. The combination of RBBB and LPFB may be diagnosed when RBBB the vector for the first half of the QRS complex is directed to about +120° with an S_IQ_{III} pattern in the standard leads, provided a vertical heart, emphysema, RVH, and lateral wall myocardial infarction can be excluded. The diagnosis is further supported by the presence of tall waves in the leads III (>15mm), II and aVF, and of AV conduction disturbances.

The findings in the precordial leads in RBBB with LPFB are generally as follows:

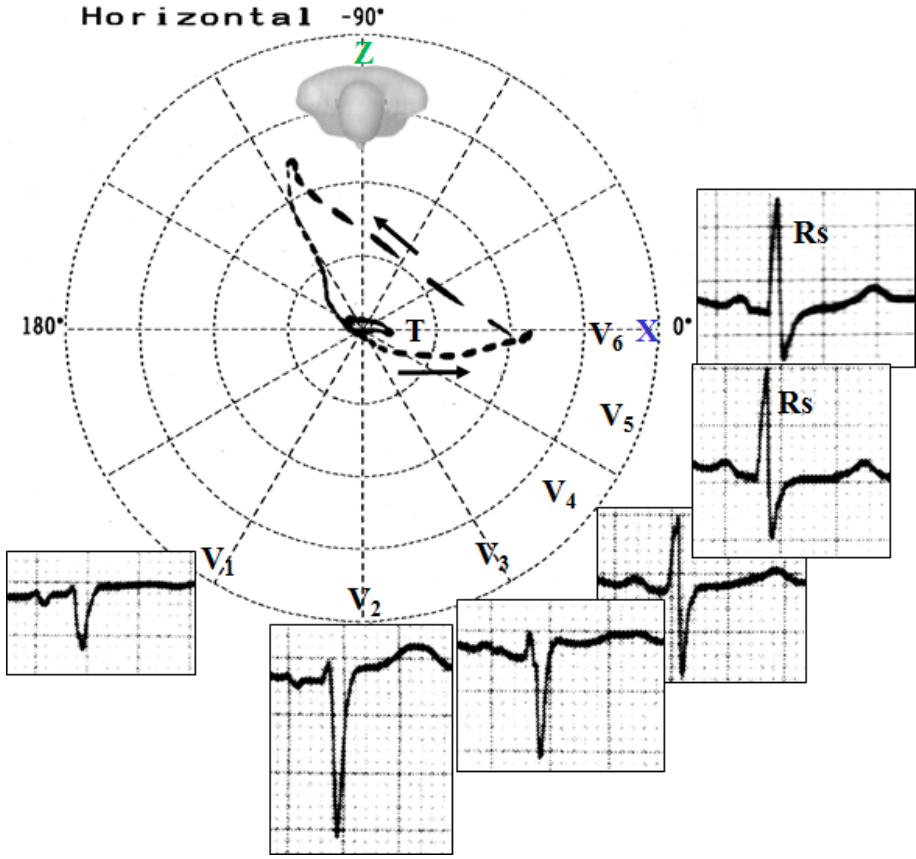
1) The normal q wave found from V4 to V6 is generally absent, however, small q waves may be present in V4 to V6 recorded at a lower level.
2) A q wave is commonly recorded in V1, even in the absence of anteroseptal MI.
3) There is a tendency for large R/S ratios to occur in the left precordial leads.
4) The QRS complexes are predominantly negative in high V leads and largely positive in low V leads.

Examples of Left Posterior Fascicular Block

ECG/VCG correlation of isolated LPFB

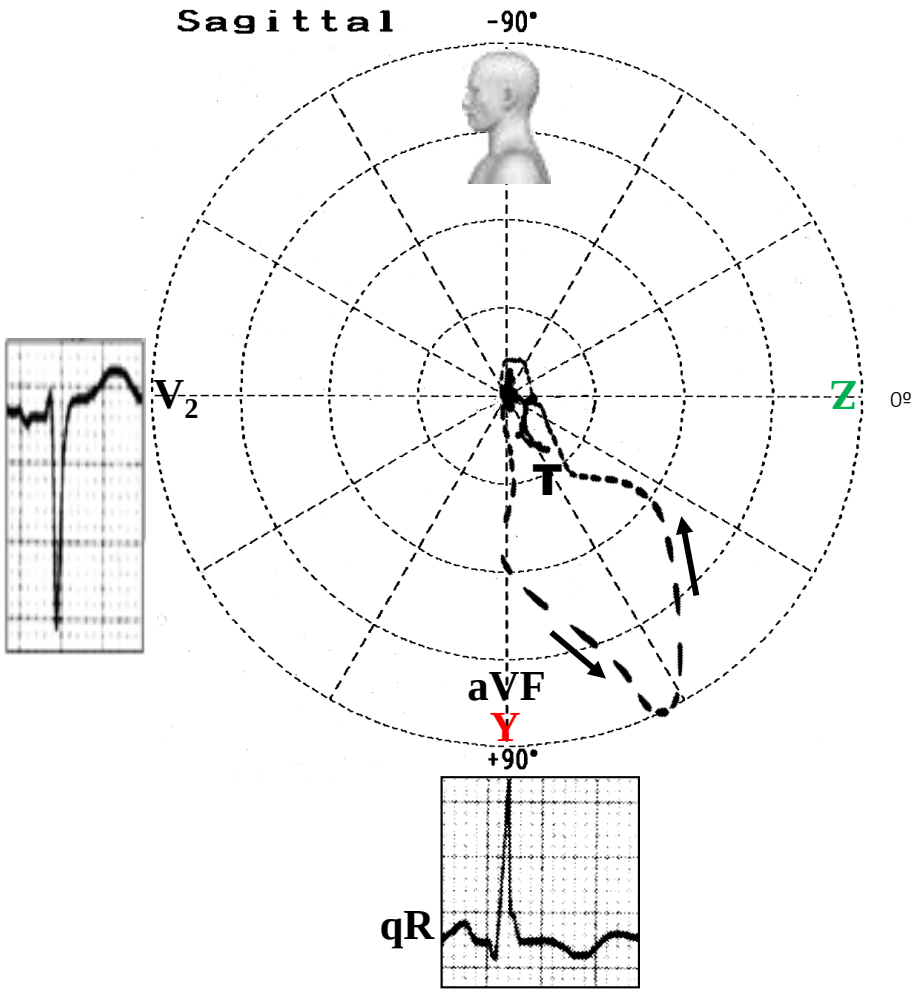


ECG/VCG correlation in the frontal plane of a typical case of LPFB: vector of initial 10 to 20 ms heading upward and to the left; rS pattern in I and aVL; qR in inferior leads; R in III > R in II; middle final notch in ascending limb of R wave of III; QRS loop of clockwise rotation and broadened morphology in clinical absence of RVH, vertical heart or lateral infarction (the diagnosis of LPFB must obligatorily be clinico-electrocardiographic). Only diagnosis if there is clinical absence of RVH, “vertical heart” or lateral infarction.



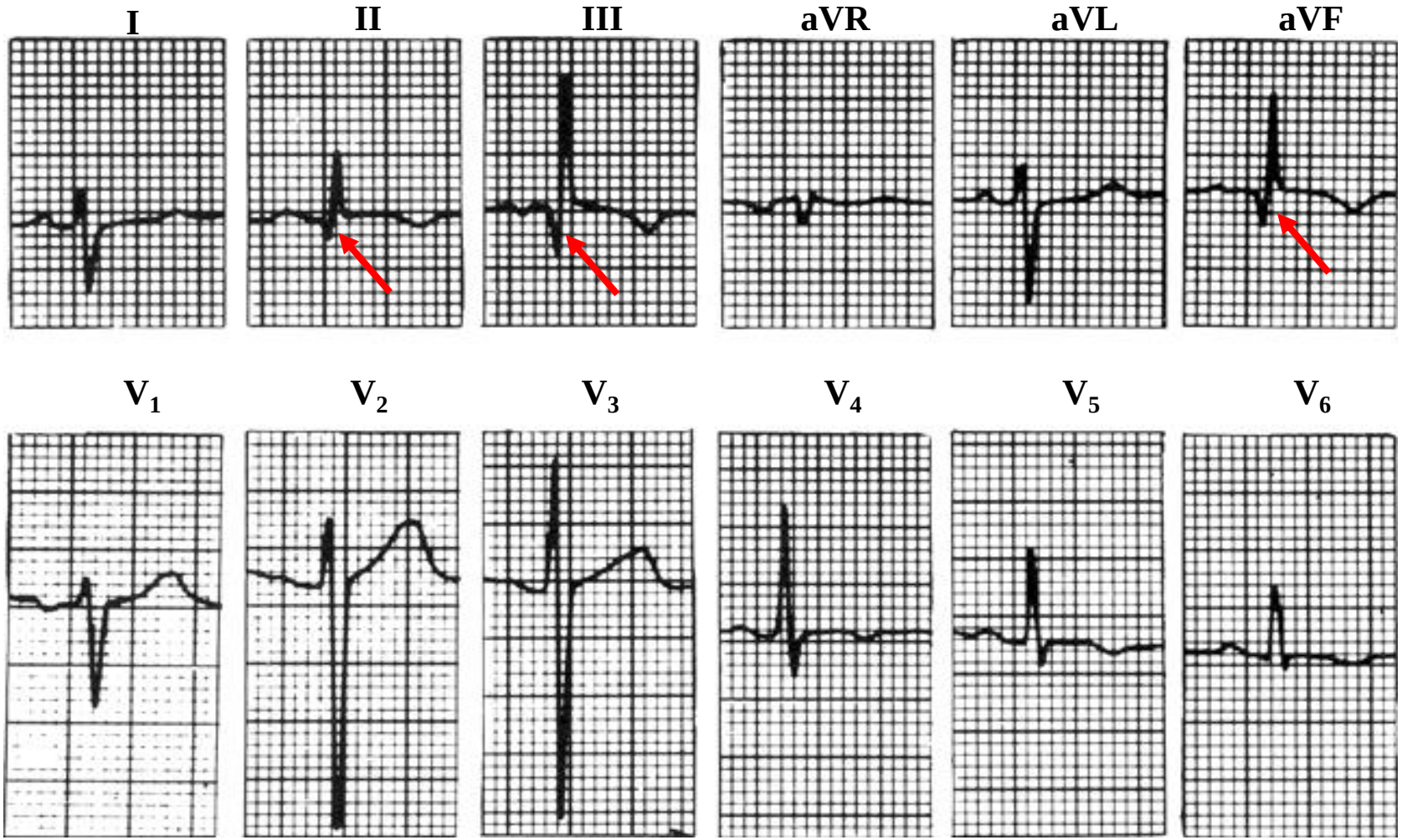
ECG/VCG correlation in the horizontal plane of a typical case of LPFB: vector of initial 10 to 20 ms heading to the front and the left; counterclockwise rotation; > 20% of the area of QRS loop located in the right posterior quadrant; deep S waves in V2 by posterior dislocation of final forces; dislocation to the left of the transition area in precordial leads; RS complexes in V5 and V6.

ECG/VCG correlation in the LSP



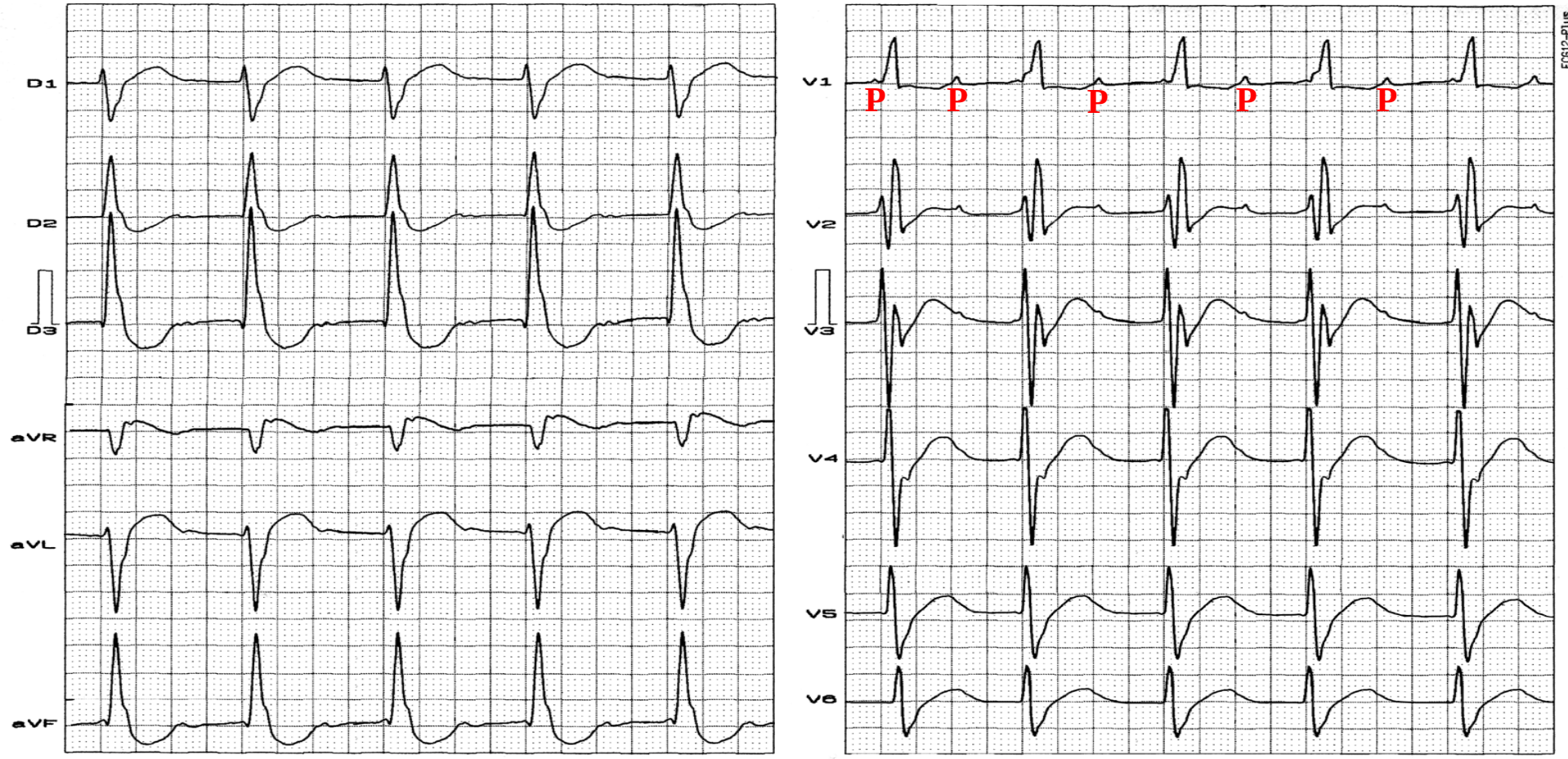
ECG/VCG correlation in the left sagittal plane of a typical case of LPFB: QRS loop of counterclockwise rotation and totally located in the postero-inferior quadrant. In aVR a qR pattern is observed, as well as middle final notch in the ascending limb of the R wave. The presence of the initial q wave points out that the vectors of the initial 20 ms are heading above.

ECG: LPFB + Inferior Myocardial Infarction



QRS axis in +115º, I and aVL rS; III qR; II, III and aVF qR with inferoapical ischemic T wave; V_2-V_3 deep S waves. LPFB and inferior MI accounted for Q waves in leads II, III and aVF (red arrows). However, R amplitude in these same leads is increased after LPFB but decreased after inferior MI. The mean QRS axis in the frontal plane was shifted toward the vertical in LPFB but little changed or shifted slightly away from the vertical in inferior MI.

ECG of a female, elderly patient (85 y.o.), carrier of Lev disease with trifascicular block: 1st degree AV block + LPFB + CRBBB + digitalis effect (ST segment "in spoon").



Autopsy diagnosis: Lev disease, left side sclerosis of the “cardiac skeleton”.
This entity is called Lev disease or progressive cardiac conduction defects.
ECG diagnosis: 1st degree AV block (PR 35 ms) + LPFB + CRBBB: probable trifascicular block. Digitalis effect. Surface ECG cannot provide certainty as to the topography of the block. It should be considered trifascicular only by electrophysiology study.

ECG: Clinical comments

Both Lenègre disease (known as progressive "primary" fibrosis of the His-Purkinje system) (**Lenègre 1964**) and the secondary mechanic injury, left side sclerosis of the "cardiac skeleton" or Lev disease (**Lev 1964**), cause intraventricular dromotropic disorders with QRS broadening into values of 120 ms or more (CLBBB or CRBBB), frequently associated to fascicular blocks.

Occasionally, they progress to more advanced (trifascicular) blocks, which may be translated by PR interval prolongation (1st degree AV block) with potential to cause sudden cardiac death (SCD) by total trifascicular AV block.

Lenègre and Lev diseases are a major cause for pacemaker implantation need in the first world: 0.15 implantations per 1,000 inhabitants per year (in Latin America is Chagas disease).

Both entities, called Progressive Cardiac Conduction Defects (PCCD) are grouped inappropriately as a single disease (Lev-Lenègre disease). However, Lenègre disease is genetic and Lev disease is degenerative. Lev disease is observed in elderly people and is characterized by progressive mechanic fibrosis of the left cardiac "skeleton" and mitral ring, central fibrous body, membranous part of the base of the aorta and muscular septum apex calcification.

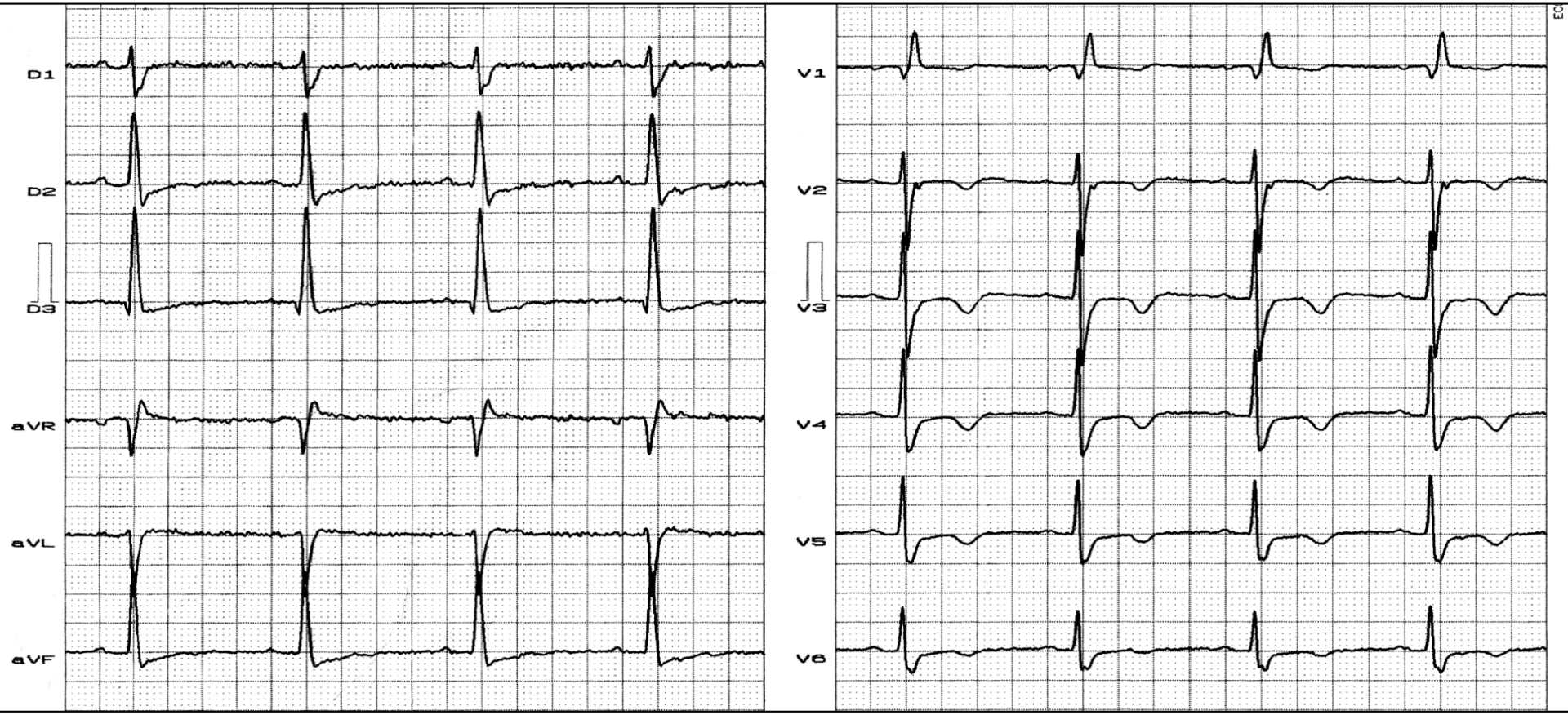
The fibrosis and calcification may involve the intraventricular His system, cause CLBBB or CRBBB associated to fascicular blocks: LAFB or LPFB with no other extra-cardiac manifestation (**Sugiura 1969**).

In the series by Dhingra et al (**Dhingra 1979**) of 452 patients with bifascicular block, 86 (19%) had PCCD as underlying cause.

In fibrosis or progressive "idiopathic" sclerosis of the His-Purkinje conduction system or Lenègre disease, the genetic mutation identified as responsible is in the same gene as in Brugada syndrome: the SCN5A gene, which is associated to atrioventricular block (**Kyndt 2001**).

In Brugada Syndrome, the PR interval of ECG and HV of the electrocardiogram are prolonged in approximately 50% of the cases. HV may reach twice its normal maximal limit.

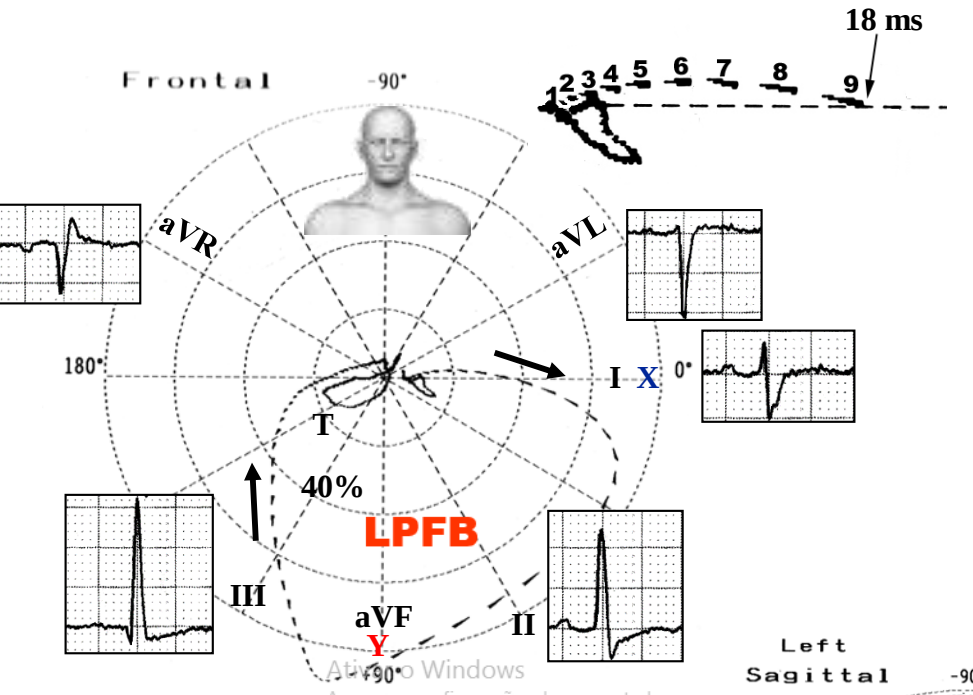
Name: GRT; **Sex:** F; **Age:** 81 y/o; **Race:** Caucasian; **Weight:** 64Kg; **Height:** 1.63 m; **Date:** 04/03/2004;
Medication in use: Isosorbide + Digoxin 0.25 mg + Enalapril 10 mg 2X + Atenolol 50 mg +ASA 200 mg



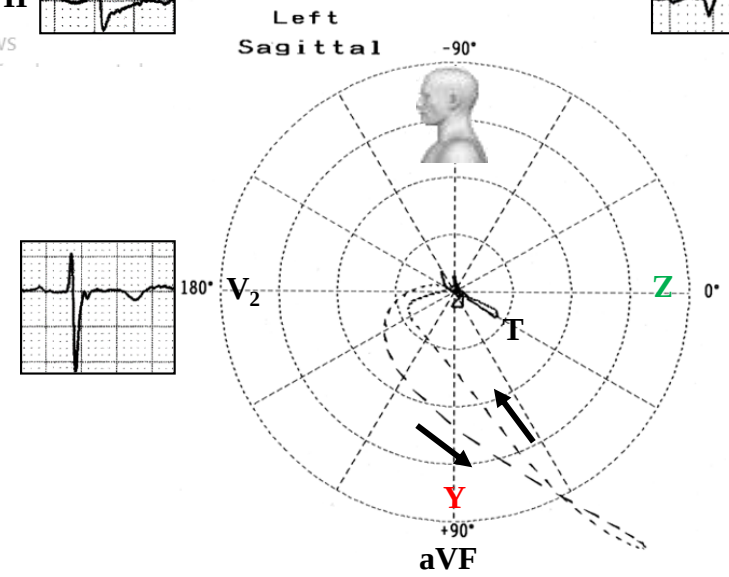
Clinical diagnosis: Hypertensive and ischemic heart disease.
ECG diagnosis: SÂQRS: +115°; QRS duration: 140 ms; I and aVL= rS; III= qR; RIII > RII; qR in V1; final broad S wave in left leads; inverted and symmetrical T wave in precordial leads;
Conclusion: 1) CRBBB; 2) LPFB: Left Bifascicular Block; 3) Anterior ischemia.
Note: ectomorphic vertical heart, RVH and lateral wall infarction were clinically ruled out.

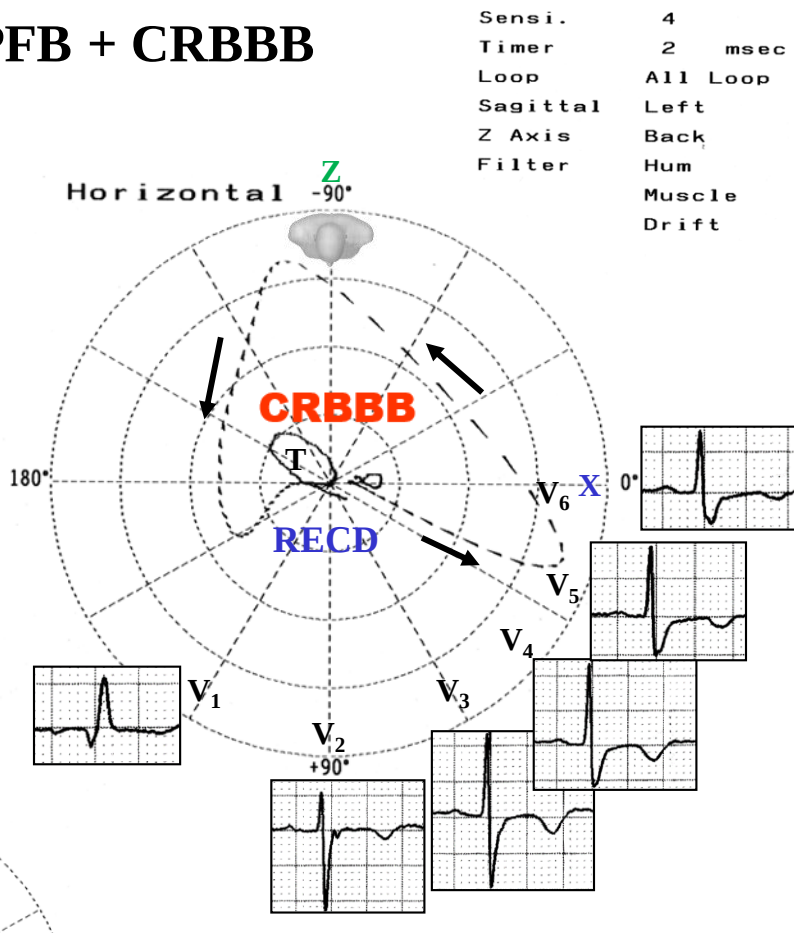
ECG of a female patient, carrier of hypertensive and ischemic cardiomyopathy that shows left bifascicular block formed by: CRBBB + LPFB. Inferior ischemia (symmetrical and inverted T waves from V2 to V6) and qR pattern in V1 are observed.

ECG/VCG correlation of LPFB + CRBBB



ECG/VCG correlation in the frontal plane where the following stands out: rS in I and aVL; qR in III; voltage of R wave of III >15 mm and > R wave of II; vector of initial 18 ms above the X line; QRS loop of CW rotation; aspect of "fat" QRS loop; ≥ 40% of the QRS loop located to the right: LPFB associated to CRBBB.

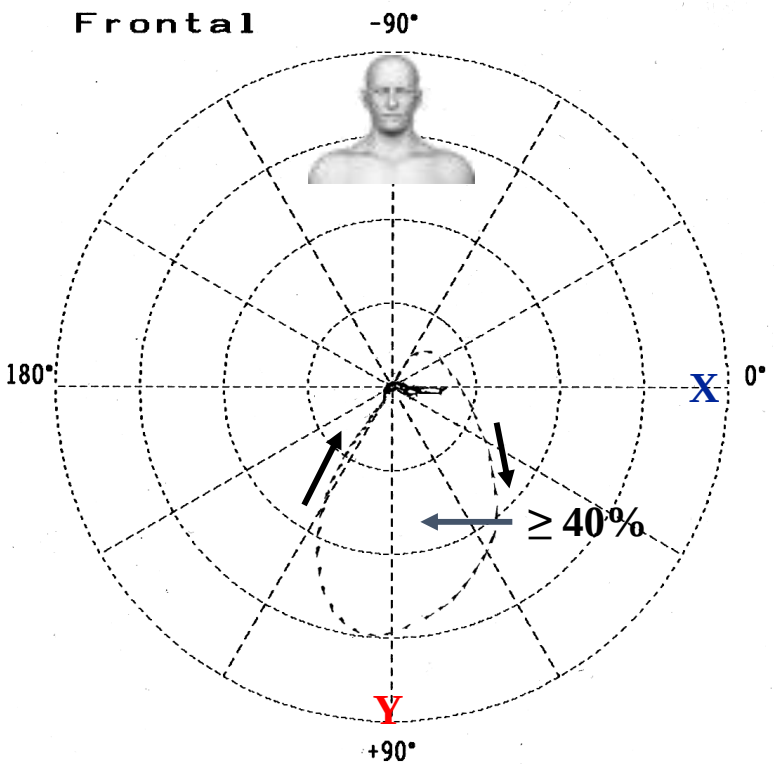




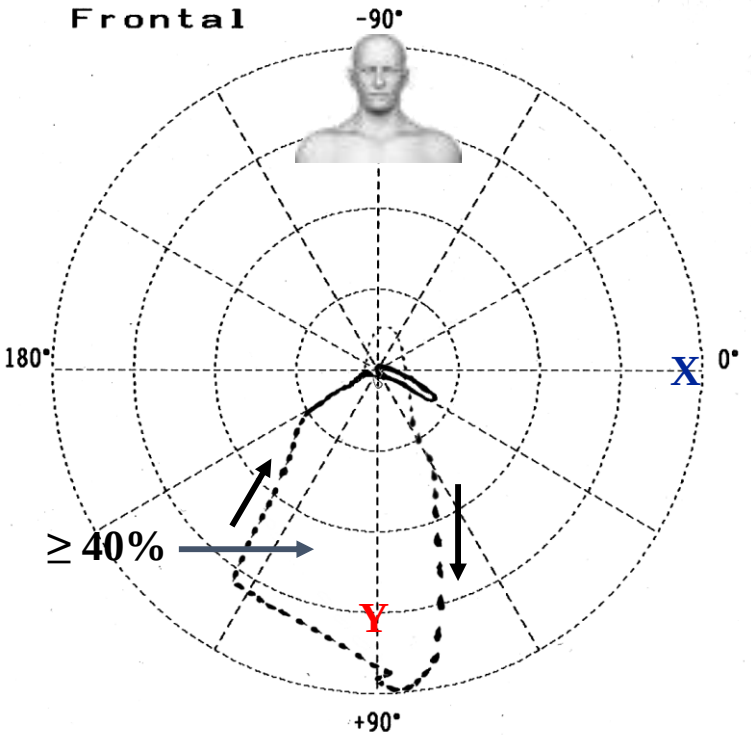
ECG/VCG correlation in the HP where the following stand out: qR pattern in V1 (it may be observed in CRBBB associated to LPFB even in absence of septal infarction); "broad" S wave of left leads: CRBBB; right end conduction delay in "glove finger" located in the right anterior quadrant: CRBBB; afferent limb of the QRS loop located behind the orthogonal X lead: CRBBB of the VCG Grishman type or Kennedy type I.

Differences in the FP between isolated LPFB and in association to CRBBB

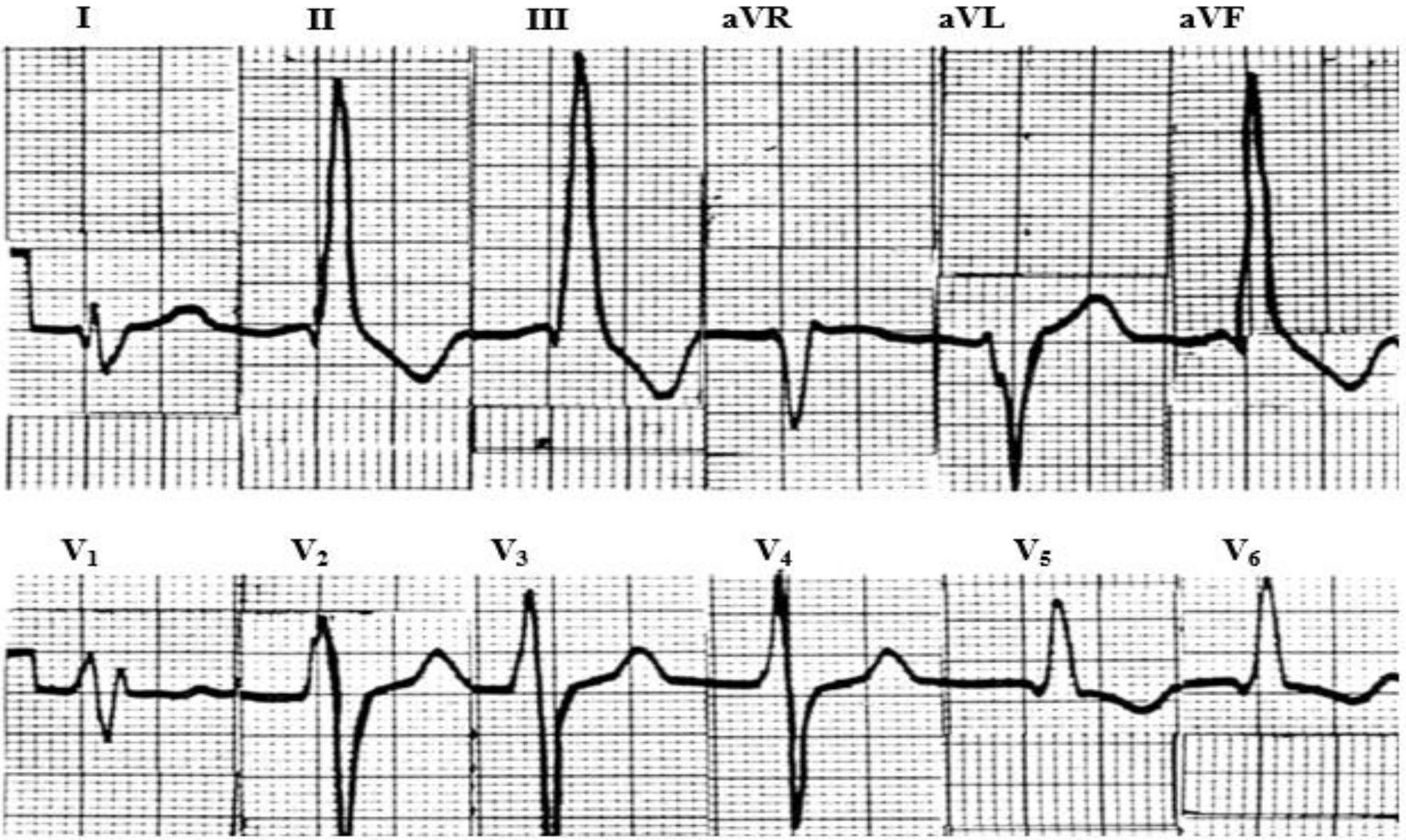
	Isolated LPFB	LPFB + CRBBB
QRS duration:	90 to 110 ms	≥120 ms
Location of QRS loop	≥ 40% left of Y line	≥ 40% to the right of the Y line
Vector of final 20 ms	There might be delay, but discrete.	With important delay to the right.



45 to 50 dashes in QRS loop: 1 dash = 2 ms



≥ 60 dashes in QRS loop: 1 dash = 2 ms



SÂQRS +110º, qR pattern in III, II and aVF, RIII = 30 mm > RII, in V_1 deep rSr' with QRSd > 120 ms, deep S wave in V_2-V_3, and strain pattern of repolarization in V_5 and V_6.

Conclusion: CRBBB + LPFB + LVE

LPFB differential diagnosis

Obligatorily, the diagnosis of LPFB must be clinical-electrovectorcardiographic. The diagnosis is not possible in the presence of:

- A vertical heart in slender subjects (ectomorphic biotype);
- Presence of any cause for right ventricular hypertrophy/RVE, especially COPD/emphysema: frequent right atrial enlargement;
- A large myocardial infarction of lateral wall: QS in I and aVL **(Elizari 2007)**;
- Right End Conduction Delay (RECD) by the inferior fascicle of the right bundle branch or RECD type II of our classification.
- Hereditary right axis deviation with pseudo left posterior fascicular block and incomplete right bundle branch block **(Lorber 1988)**

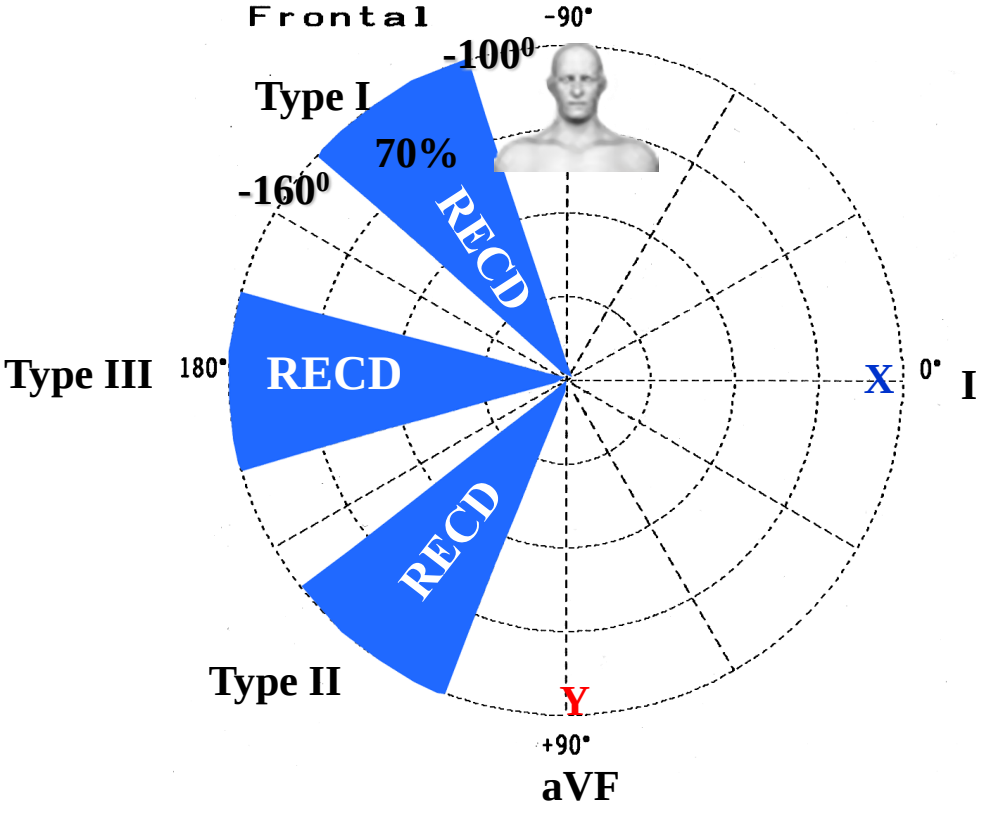
Clinical causes that prevent the electrocardiographic diagnosis of LPFB.

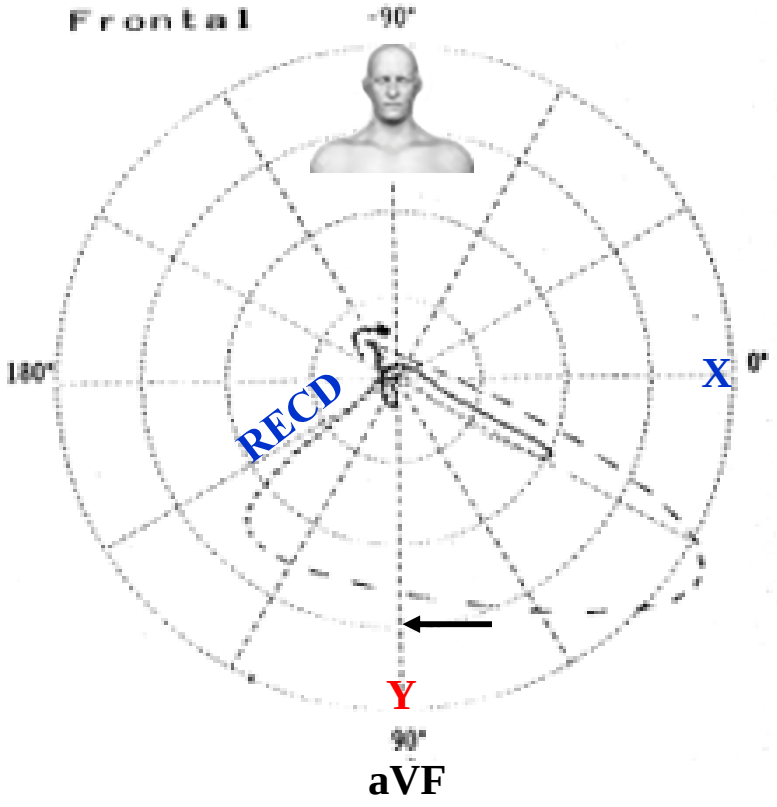
Right End Conduction Delay (RECD) type II or Right Inferior Fascicle Block (RIFB)

Characterized by presenting RECD located in the right inferior quadrant in the territory of the inferior fascicle of the right branch. It corresponds to the territory of the RIFB.

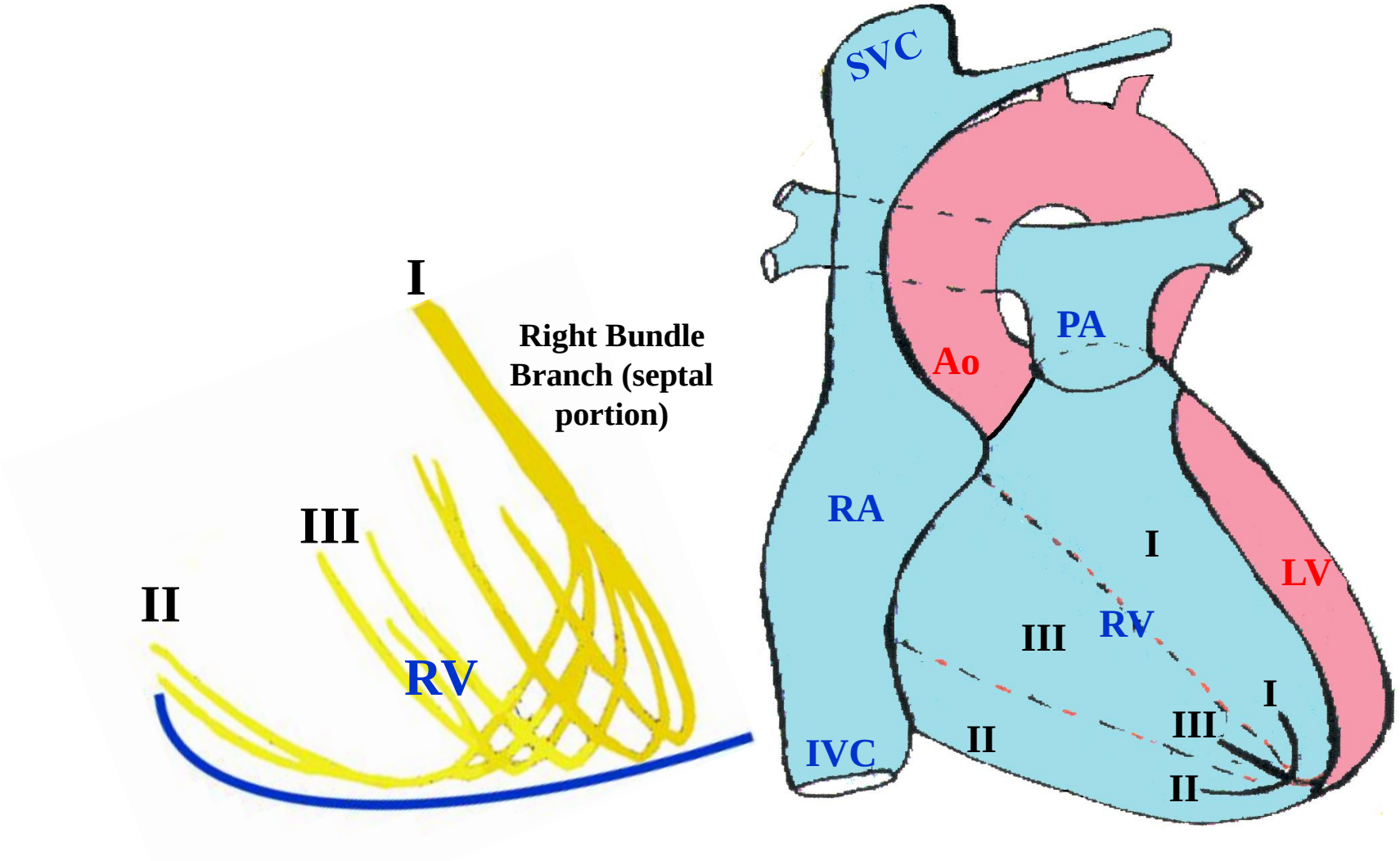
The differential diagnosis occurs with left posterior fascicular block (LPFB). Many of the cases described in literature as LPFB are, the way we see it, RECD Type II, and since their electro-vectocardiographic differences are very subtle, the diagnosis must always be clinico-electrovectocardiographic.

Location of RECD of right bundle on RV free wall and types



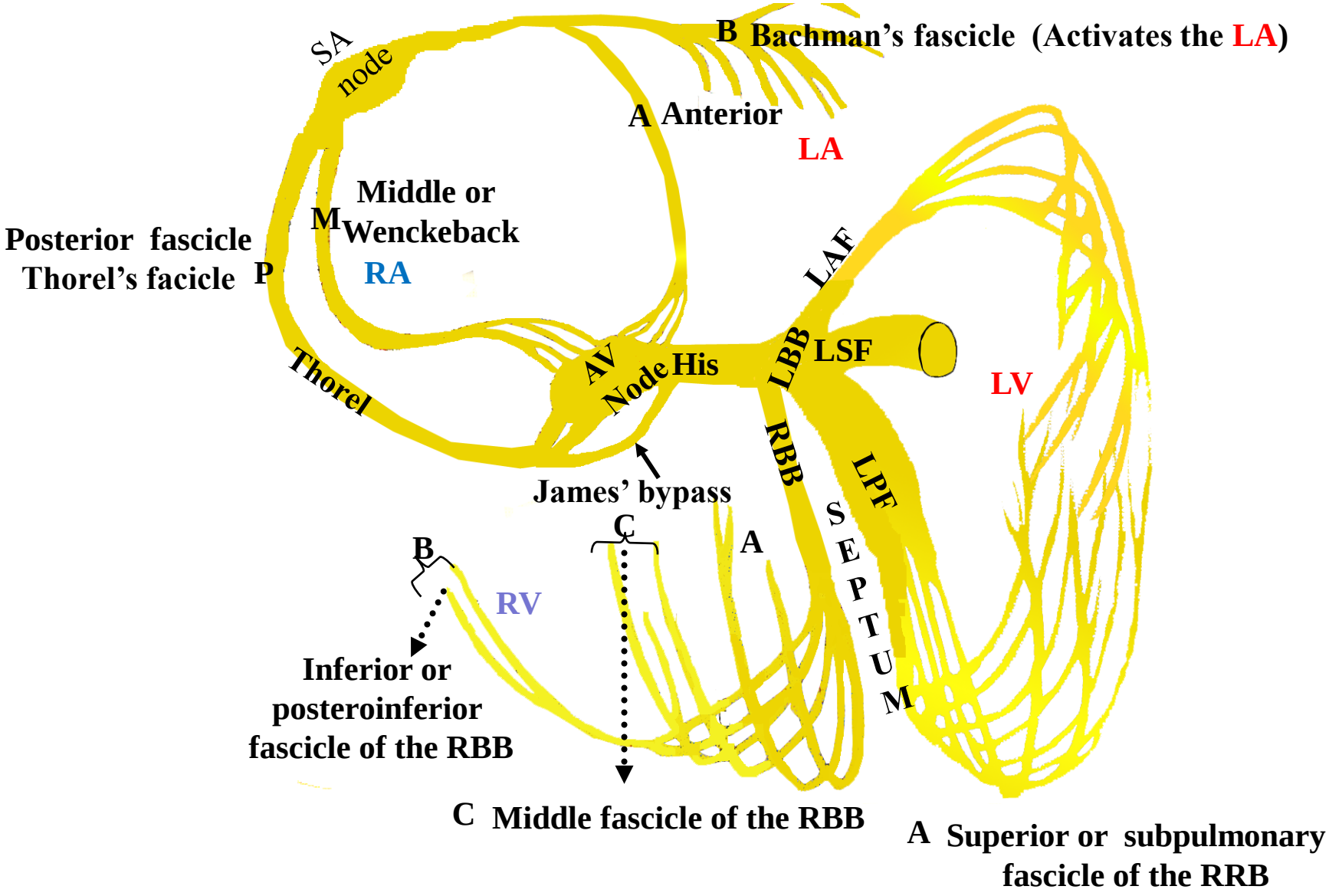


Distribution of the three fascicles or contingents of the Right Bundle Branch of the His in the RV Free Wall



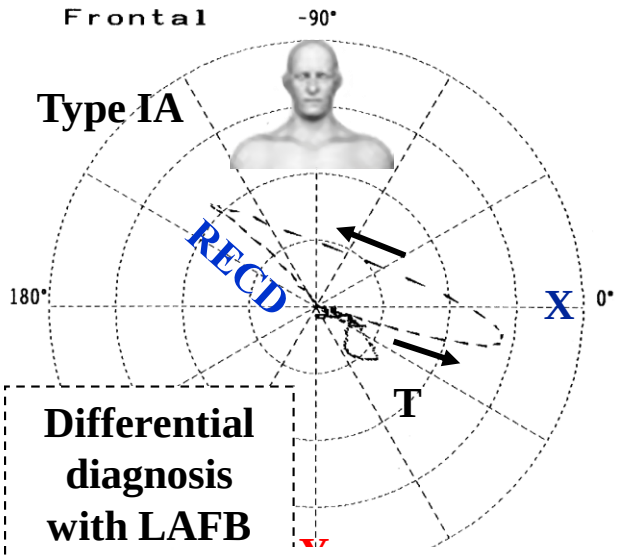
I - Territory of Superior or Subpulmonary Fascicle
II – Territory of Inferior or Posteroinferior Fascicle
III – Territory of Middle Fascicle

Components of the cardionector system of sinoatrioventricular & intraventricular conduction system

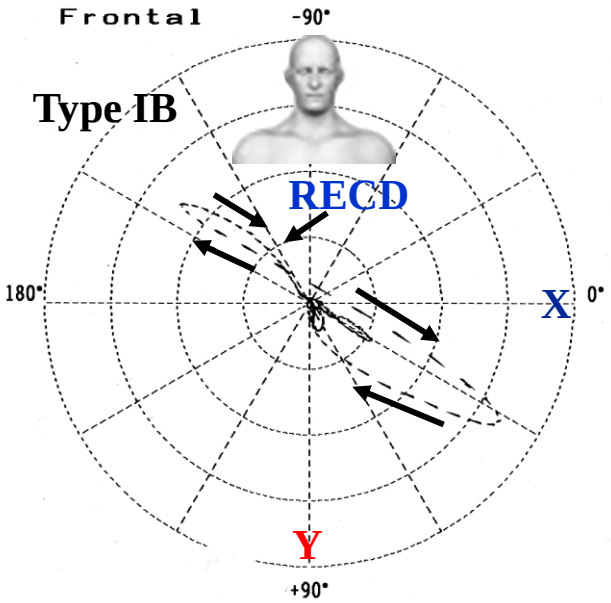


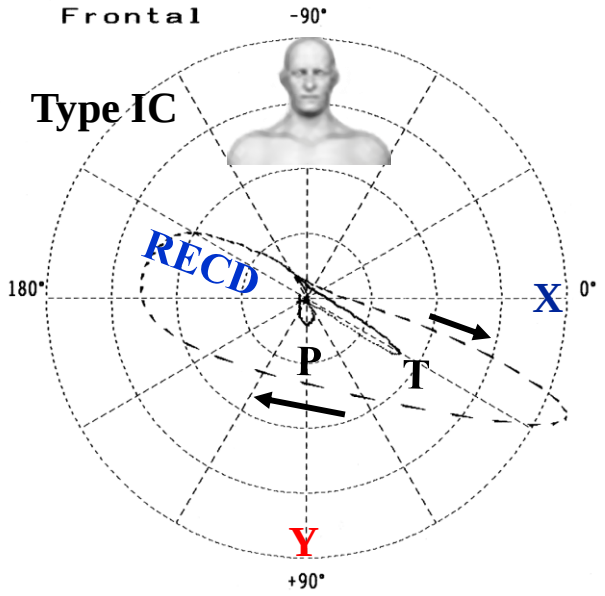
You can see the SA node, atrial internodal bundles (anterior, middle and posterior), AV node, His bundle and its divisions (3 left and 3 right).

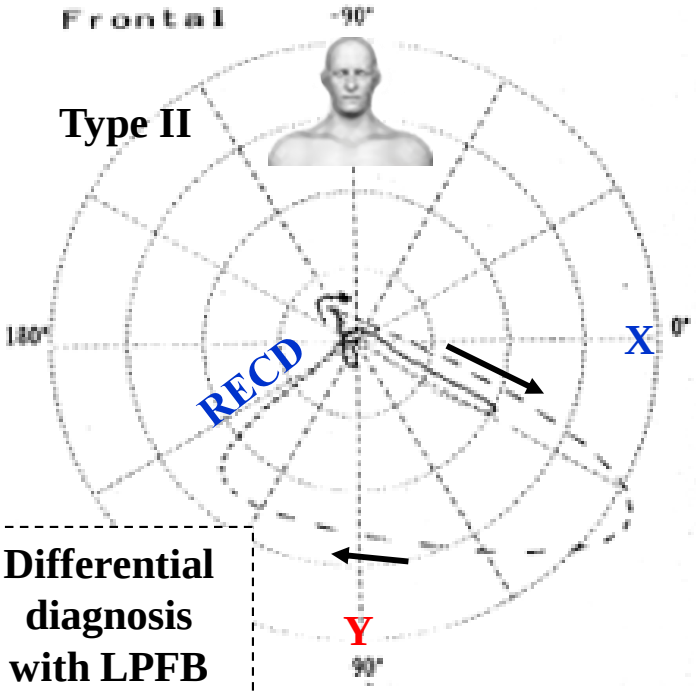
VCG proposal of classification of Right Bundle Branch fascicles on RV free wall



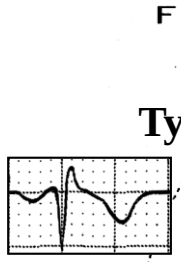
Differential diagnosis with LAFB



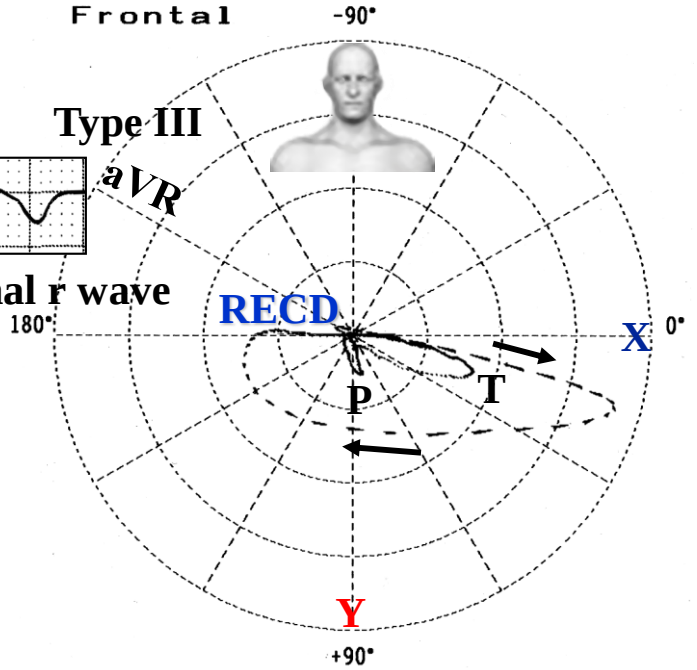




Differential diagnosis with LPFB



Broad final r wave



RECD type II – ECG/VCG characterization

A. Electrocardiographic criteria:

1) QRS axis (SÂQRS) between + 70° and + 110°;
2) QRS duration: normal;
3) SI RII RIII pattern, with RII and RIII of voltage not increased (usually ≤10 mm), never reaching 15 mm (essential element for the differential diagnosis with LPFB);
4) RII ≥ RIII (in LPFB RIII > RII);
5) aVR of the QS type;
6) Possible notch in the descending ramp of inferior leads;
7) S wave of V_2 and/or V_3 of increased depth;
8) Persistent S wave until V_5 and/or V_6;
9) V_1: rS, RS or rSR' with S of V_1 and V_2 possibly broadened.

B. Vectocardiographic criteria:

RECD in the three planes located to the right and below.

Frontal plane:

- Initial vectors always to the left, above and below;
- Clockwise rotation;
- Predominant location in the inferior quadrants;
- Rapid change from left to right between 30ms and 50ms;
- RECD to the right and below between +120º and +150º.

Horizontal plane:

- QRS loop of counterclockwise rotation;
- Marked posterior dislocation;
- Rapid change from left to right between 40 and 50 ms;
- RECD to the right and behind.

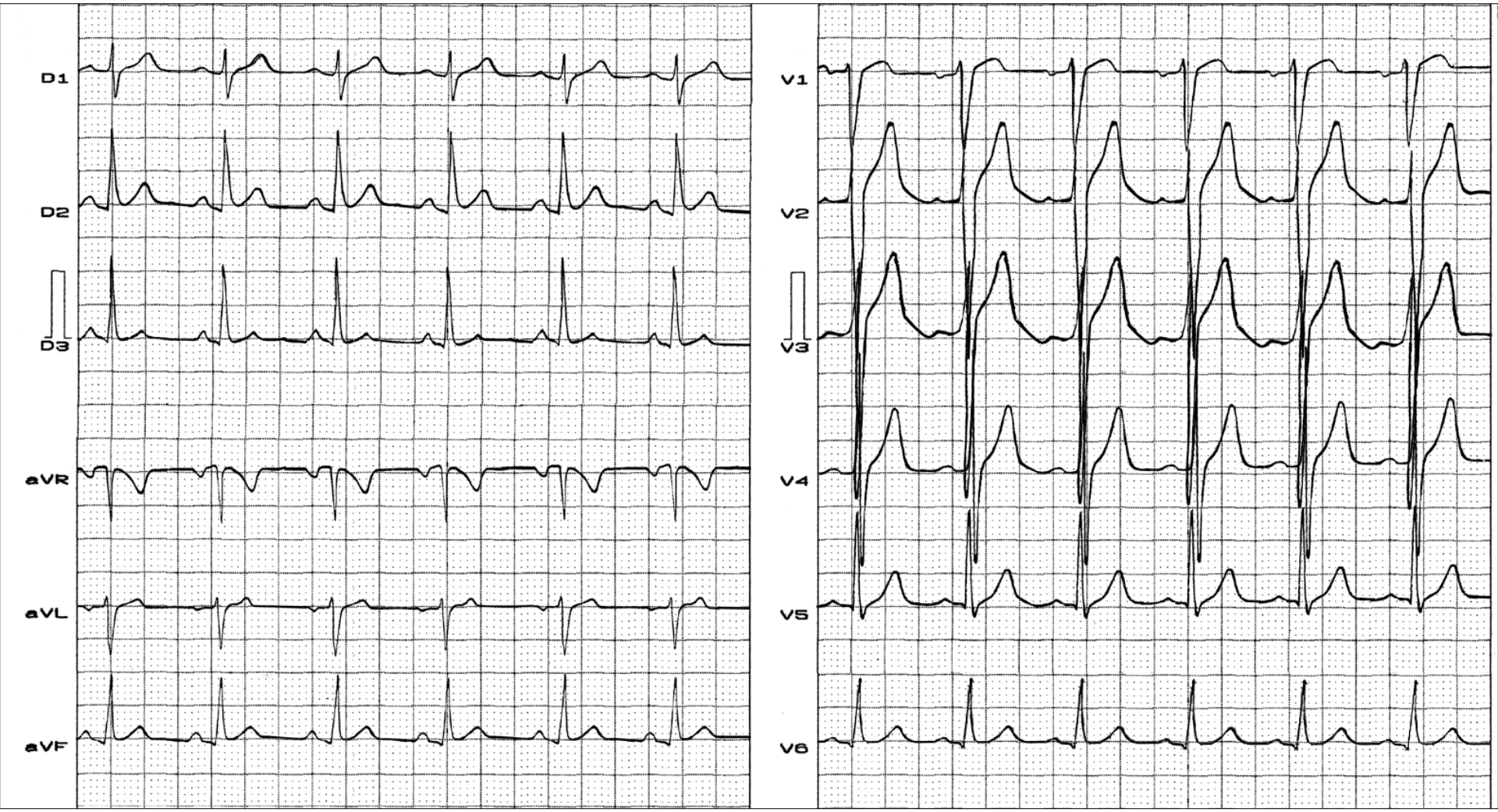
Right sagittal plane:

- Initial vectors upward or downward;
- Clockwise rotation;
- Marked postero-inferior dislocation;
- RECD downward and backward.

Differential diagnosis between RECD type II and LPFB

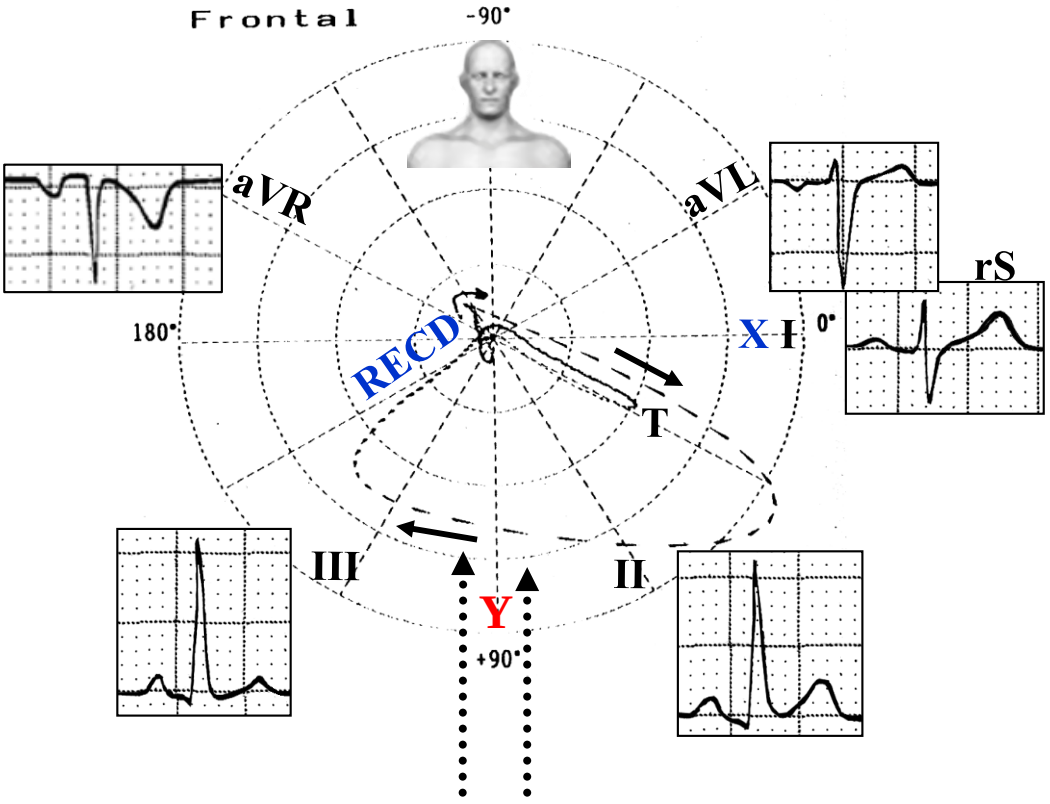
	RECD type II	LPFB
PR interval duration	Normal	Frequent prolongation
Association with inferior myocardial infarction	No	Frequent
Voltage of RII and RIII	≤ than 10 mm	≥ than 15 mm
RII/RIII voltage ratio	RII >RIII	RIII > RII
Notch in the descending ramp of R wave of inferior leads	Absent	Constant middle-final notch
R-peak time, ventricular activation time (old intrinsicoid deflection) in aVF, V5 and V6	Normal	Increased: up to 30 ms
R-peak time in aVL	Normal	Decreased: up to 15 ms
Aspect of QRS loop in the FP	Clockwise and with characteristic rapid passage from left to right between 30 and 50 ms.	Clockwise rotation of “fat” loop and maximal vector close to +120°
Clinical factors that should be excluded	Not stated	Vertical heart, RVH and lateral myocardial infarction.

Name: CJO; **Sex:** M**; Age:** 22 y/o; **Race:** Caucasian; **Weight:** 70 Kg; **Height:** 1.71 m; **Biotype:** Athletic
Date: 15/05/2001

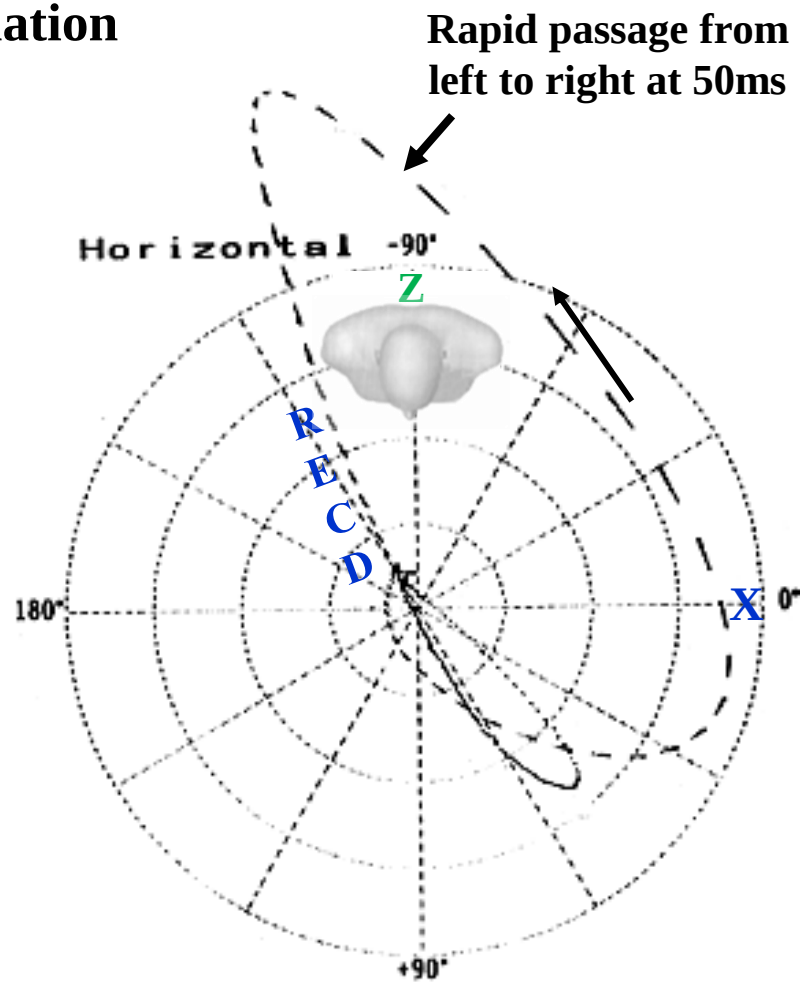


Clinical diagnosis: CM: preoperative evaluation for abdominal surgery, physical examination background: nothing important. ECHO nothing important. Chest X rays: nothing important;
ECG diagnosis: SÂQRS:+ 95°. SI-RII-RIII pattern (RIII < 15 mm). I and aVL: rS and qR in II and III. Descending ramp of R wave is slightly slow. It may present diagnostic doubt with LPFB.
Conclusion: RECD type II: normal variant.

ECG/VCG correlation



Rapid passage from left to right

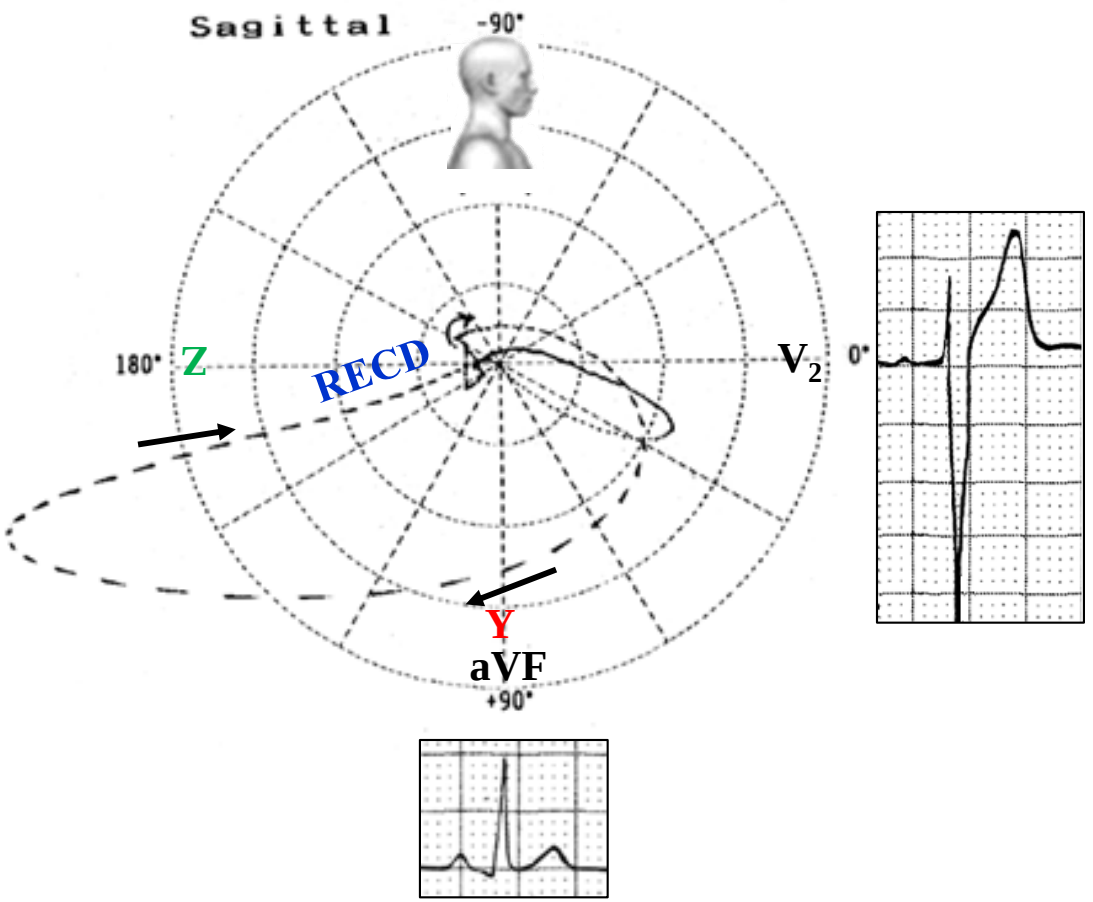


RECD type II: RECD (≥ 15comets) located in the right inferior quadrant in the territory of the inferior fascicle of the right bundle branch.
SÂQRS: +95°. SI-RII-RIII pattern (RIII < 15 mm). I and aVL: rS.
II and III: qR. The descending ramp of R wave is slightly slow. It may present differential diagnosis with LPFB.

QRS loop with initial portions to the front, counterclockwise rotation, important posterior dislocation, rapid passage from left to right and RECD located to the back and the left. QRS loop similar to RVH type C.

Name: CJO; **Sex:** M**;** **Age:** 22 y/o; **Race:** Caucasian; **Weight:** 70 Kg; **Height:** 1.71 m; **Biotype:** Athletic
Date: 15/05/2001

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



Depth S wave of V_2 because QRS loop is dislocated to back, very similar to LPFB and type C VCG RVH.

Which is the clinical importance of right fascicular blocks or RECDs? These are electrovectorcardiographic changes, secondary to physiological delay or to true dromotropic disorders in the territory of one of the three fascicles of the right branch, in isolation in the RV free wall. They were denominated with several nomenclatures: Right End Conduction Delays (RECDs), End Conduction Delays (ECDs), right bundle branch fascicular blocks, terminal, parietal, zonal or Purkinje blocks or incomplete right bundle-branch block (IRBBB). It usually is thought to be associated with abnormalities of the peripheral Purkinje system. IRBBB may be a developmental variation in thickness of the RV free wall rather than an abnormality of the RV conduction system in cases without apparent heart disease. The developmental variant appears to have a genetic basis.(**Moore 1971**). If the electrocardiographic pattern of QRS prolongation up to 110 ms (in adults), with a terminal r' in V, and broad S wave in left leads V5 and V6 or standard lead I and aVL, were often the sole consequence of delayed conduction within the right bundle branch, then the term IRBBB to describe this pattern might be appropriate. Conversely, if delay in conduction in the right bundle branch is only inconsistently present in this electrocardiographic constellation, then the diagnosis of IRBBB would be at best imprecise and often incorrect.(**Massing 1972**)

Its clinical significance and interest lies in the fact that:

1) They may be confused with left fascicular blocks: Left Anterior Fascicular Block (LAFB) and Left Posterior Fascicular Block (LPFB);
2) They may be confused with electrically inactive areas (pseudo electrically inactive areas) both in the anterior and the inferior walls.
3) They may represent the electro-vectocardiographic pattern of Brugada syndrome and one subpopulation of Arrhythmogenic Right Ventricular Dysplasia (ARVD/C) (**Pérez-Riera 2011**).
4) They may be confused with type C RVH

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