ECG & VCG in Left Bundle Branch Block

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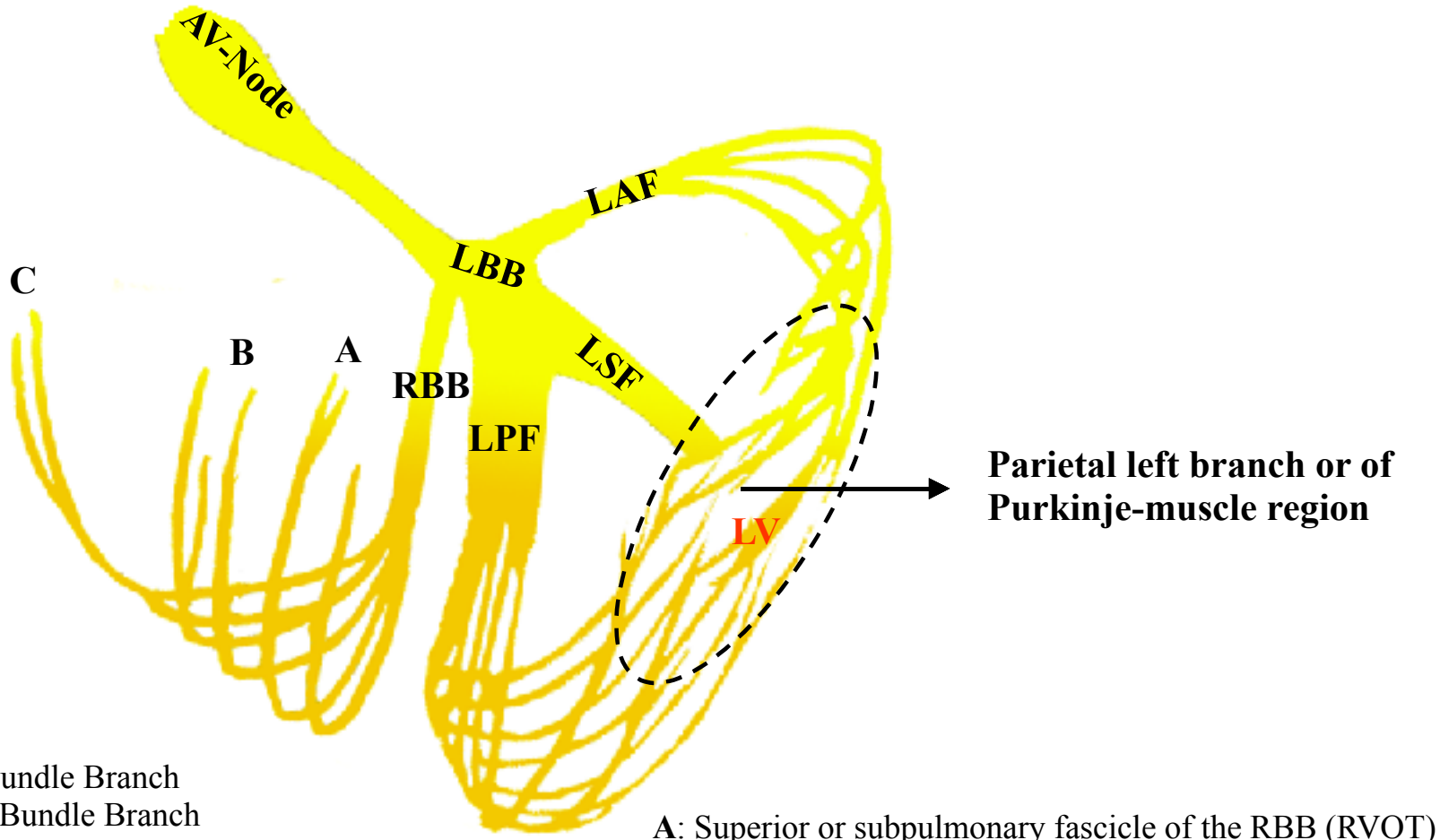
Outline of the Hexafascicular Nature of the Human Intraventricular His System



LBB: Left Bundle Branch
RBB: Right Bundle Branch
LAF: Left Anterior Fascicle
LSF: Left Septal Fascicle or Middle Septal Fascicle
LPF: Left Posterior Fascicle

A: Superior or subpulmonary fascicle of the RBB (RVOT)
B: Middle fascicle of the RBB
C: Inferior or posteroinferior fascicle of the RBB

The intraventricular His system has three left fascicles: **LAF**, **LPF** and **LSF** and the **RBB** has three right branches or false tendons located on right ventricle free wall (**A, B, C**).

Left Bundle Branch Block (LBBB)

Concept: LBBB, left His system global block or left ventricular global block, is any delay in left ventricular (LV) activation as a consequence of a dromotropic disorder located in one or more of the following sites:

I) Proximal, predivisional or membranous

1. **Left His bundle**
2. **Stem (truncus) of Left Bundle Branch of His bundle (LBB):** 1 and 2 are known as pre-divisional, troncular or membranous LBBB.

II) Divisional or fascicular

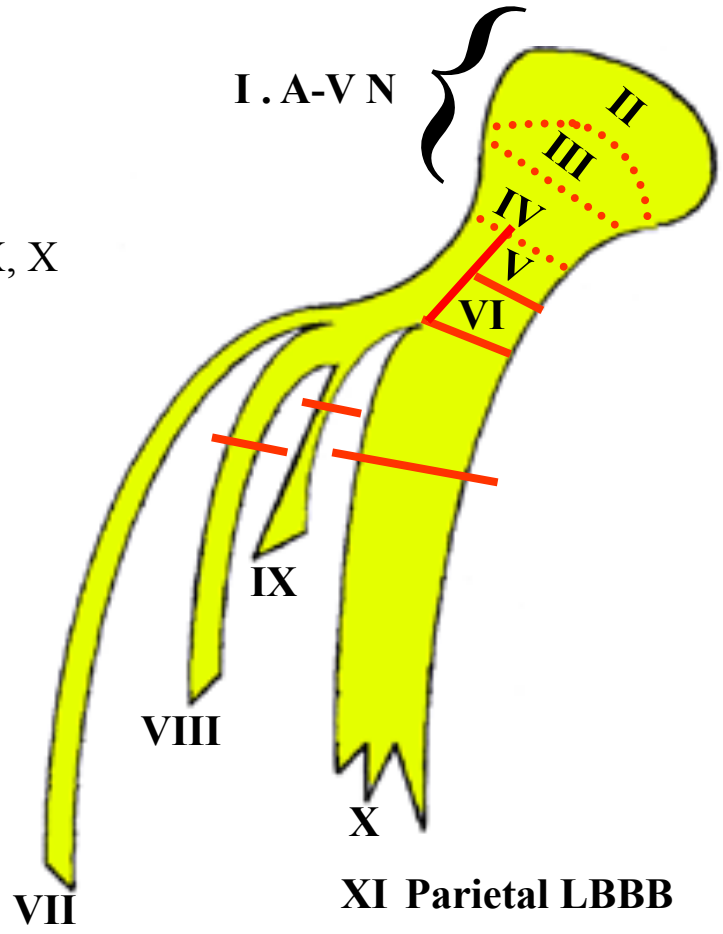
3. **Fascicles or divisions of LBB of the His bundle concomitantly:** left anterior fascicle (LAF), left posterior fascicle (LPF), and left septal or middle-septal fascicle (LSF); this type is known as fascicular or divisional LBBB.

III) Intramyocardial or Purkinje muscle

4. **Left Purkinje globally approached:** This is known as parietal, Purkinje, or intramural

Outline of the three portions in the LBBB and nomenclature of the intraventricular His System

1. Troncular or predivisional LBBB: V-VI
2. Divisional or fascicular LBBB: VIII, IX, X
3. Purkinje-muscle LBBB: XI



I. **A-V N:** Atrio-Ventricular Node
II. **A-N:** Atrio-Nodal region of the A-V node
III. **N:** Nodal region of A-V Node
IV. **N-H:** Node-His region of the A-V Node
V. **Left His contingent**
VI. **LBB:** Left Bundle Branch
VII. **RBB:** Right Bundle Branch of the His Bundle

Blocks in this portion are called predivisional or membranous LBBB

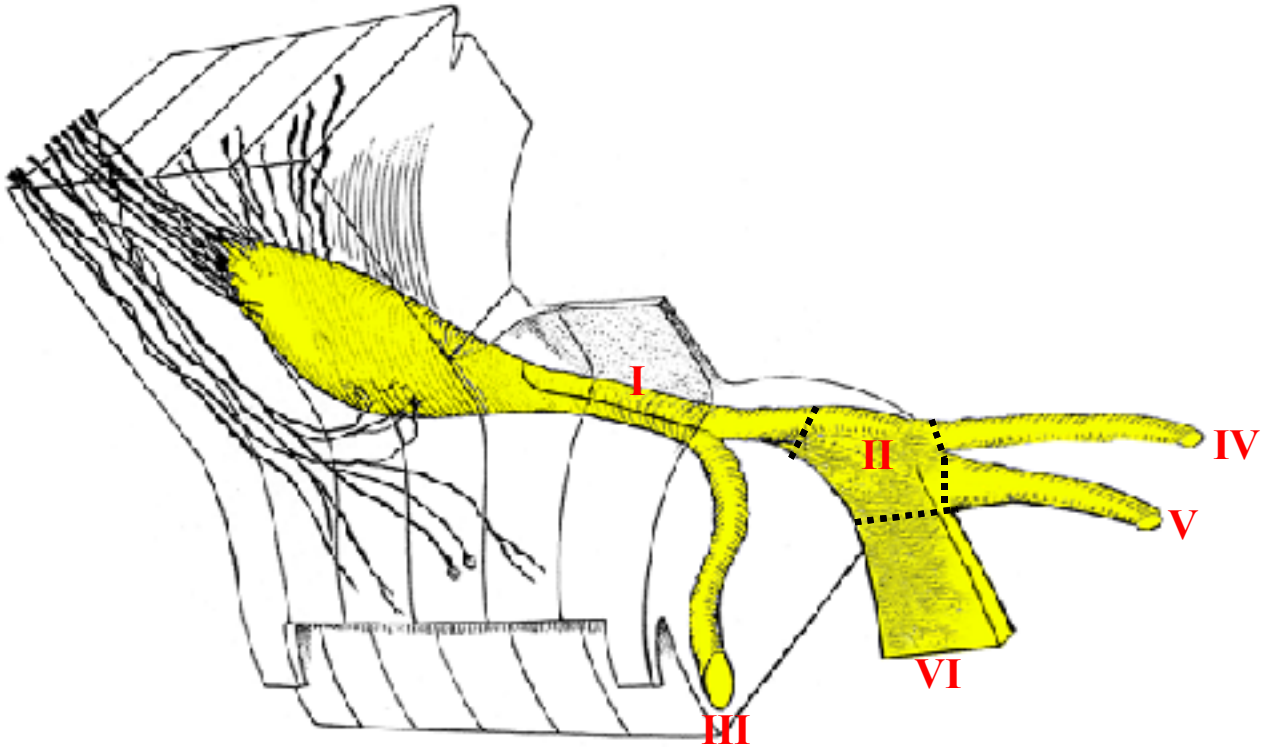
VIII. **LAF:** Left Anterior Fascicle
IX. **LSF:** Left Septal Fascicle
X. **LPF:** Left Posterior Fascicle

Blocks in this portion are called fascicular or divisional LBBB

XI. **Parietal**

Blocks in Purkinje-muscle or intramyocardial are called parietal LBBB

Outline that shows the CLBBB according to topography



I. Penetrating portion of left His bundle
II. Stem or truncus of LBB

Blocks in I and II are called predivisional, troncular or membranous LBBBs

III. Right Bundle Branch (RBB)
IV. Left Anterior Fascicle (LAF)
V. Left Posterior Fascicle (LPF)
VI. Left Septal Fascicle (LSF)

Blocks in IV+V+VI are called divisional or fascicular LBBB

Characteristics of the structural components of predivisional CLBBB

I. Penetrating portion of left His bundle

- **Systematization:** longitudinal
- **Length:** 75 mm (50 to 100 mm)
- **Neighboring structures:** fibrous trigone, mitral-aortic rings and membranous septum.

II. Truncus of Left Bundle Branch (LBB)

- **Length:** 10 mm (five times shorter than the **RBB**).
- **Diameter:** in its onset 5 mm and at the end 9 mm (four to eight times longer than the **RBB**).
- **Color:** white.
- **Cell type:** Purkinje. These are large cells of 15 to 30 mm of diameter and 20 to 100 mm of length.
- **Conduction velocity:** 5 m/sec (fast fibers)
- **Characteristics of Action Potential (AP):** fast fiber type, Na^+ dependent, phase 4 with automatism (diastolic depolarization) and with a refractory period shorter than the right bundle branch (RBB): faster depolarization and repolarization.
- **Neighboring structures:** very close to the following structures: Noncoronary and right coronary aortic valves, aortic ring (the reason why is frequent in aortic valve disease), membranous septum, subaortic septal endocardium, apex of muscular septum, right bundle branch.
- **Irrigation:** assured by two arterial systems:

  1) Branches of the posterior descending artery (90% of the right coronary branch):

  - ✓ AV node artery: ramus septi fibrosi.
  - ✓ Ramus septi ventriculorum superior.
  - ✓ Ramus cristae.

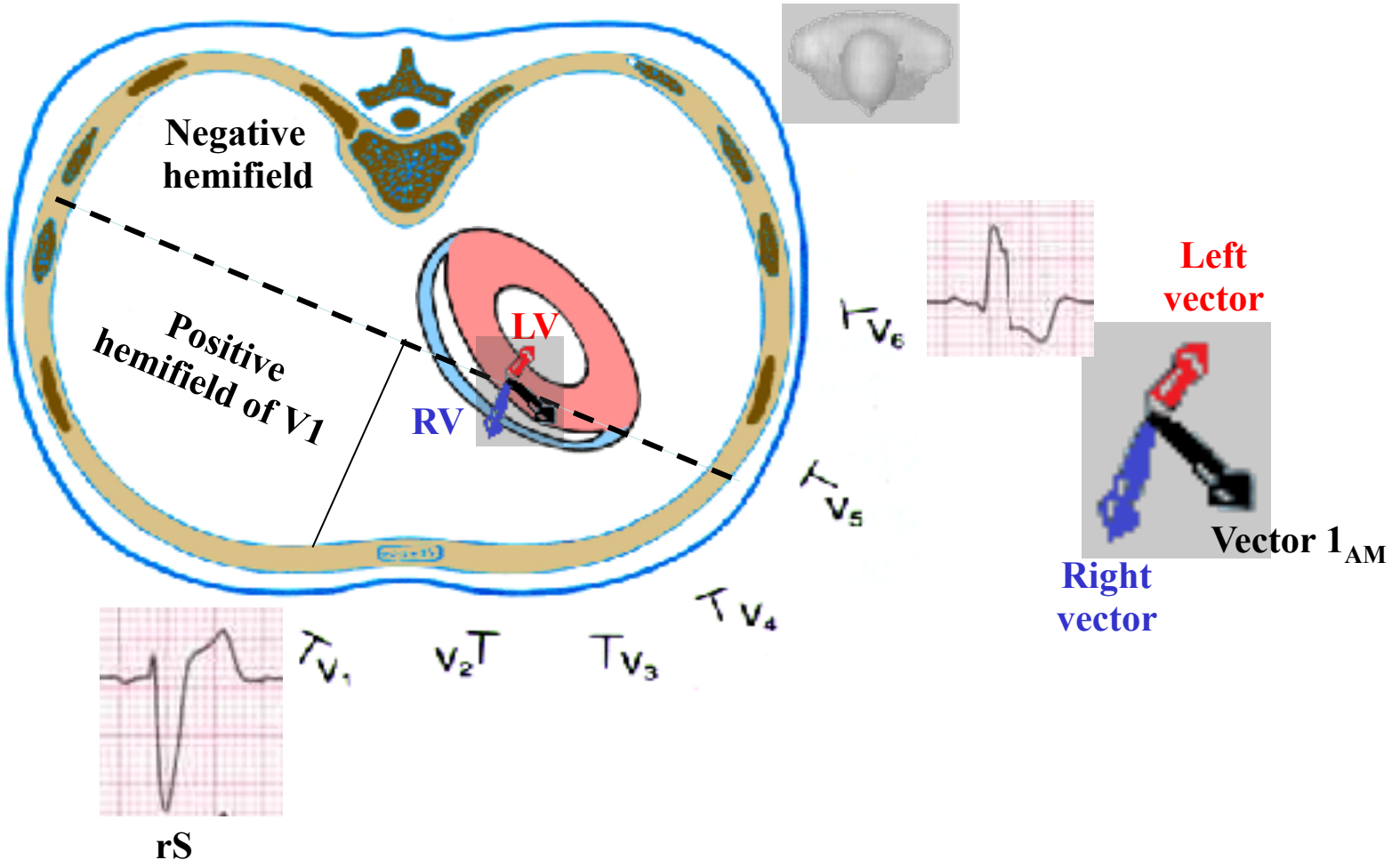
  2) Branches of left anterior descending artery:

  - ✓ Ramus limbi sinistri (equivalent to ramus limbi dextri of the left anterior descending coronary artery).

Possible etiologies of complete LBBB

1. Systemic hypertension (SH) (**Rodríguez-Padial L 2012**);
2. Coronary Heart Disease (CHD) **(Liakopoulos 2013)**;
3. Association of SH and CHD;
4. Cardiomyopathies (**Chan 2014**) /diffuse myocardial disease;
5. Idiopathic Dilated Cardiomyopathy (IDCM): LBBB is observed in ~25% of cases. (**Brembilla-Perrot 2008**)
6. Aortic valve disease (**Poels 2014**);
7. Mitral valve disease **(Silva 1996)**;
8. Myocarditis
9. Sclerosis of the left side of the cardiac skeleton: Lev disease (**Bharati 1975**);
10. "Idiopathic" sclerosis of the His conduction system: Lenègre disease. This is a genetic disease that affects the SCN5A gene (**Kyndt 2001**);
11. Cardiomyopathies of collagenopathies(**Mavrogeni 2014**);
12. Miscellaneous:
    - ✓ Congenital heart diseases. E.g.: late phase of aortic stenosis;
    - ✓ Blood or crystalloid cardioplegia;
    - ✓ Use of taxol, cytotoxic antineoplastic drugs **(Rowinsky 1991)**;
    - ✓ Primary amyloidosis (**Bellavia 2009**);
    - ✓ Sarcoidosis (**Strauss 2011**);
    - ✓ Hyperpotassemia (**Manohar 2003**);
    - ✓ Post-operative segmentectomy in hypertrophic obstructive cardiomyopathy (**Riera 2002**);
    - ✓ After coronary angiography (**Al-Hadi H**).
13. Without apparent cause (idiopathic, cryptogenetic, primary or essential).

Outline that shows the ECG/VCG correlation in the HP of the initial 10 ms to 20 ms vector (phase 1) in CLBBB



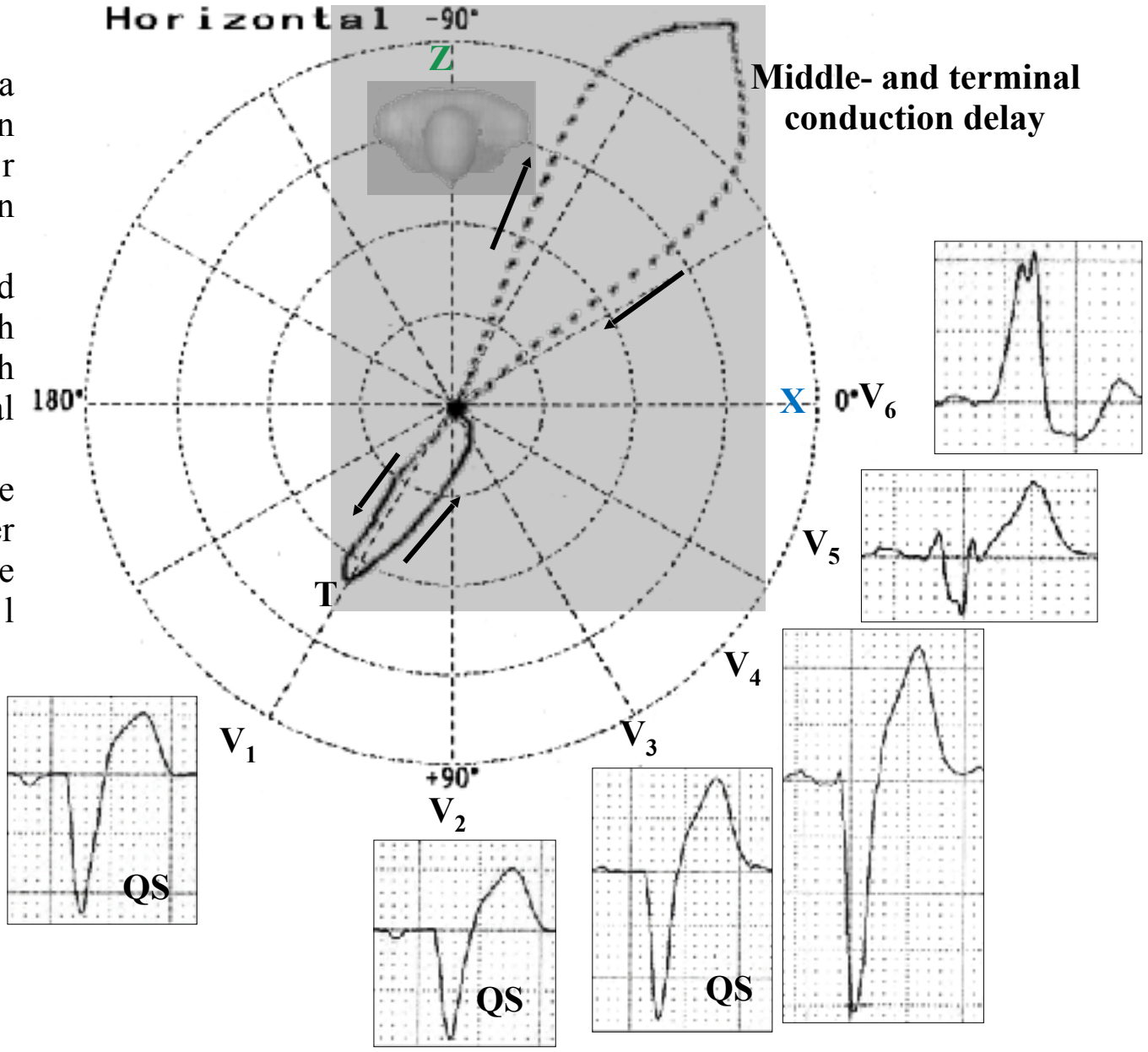
First phase: vector 1or I_{AM} of initial 10 ms, oriented almost always to the front and leftward (80% of cases). More rarely, the vector of the initial 10 ms is pointing backward. In this case, it may be parietal CLBBB; however, in most cases it indicates CLBBB complicated with septal inactive area.

Outline that shows the ECG/VCG correlation in the HP in CLBBB, in the cases where the vectors of the initial 10 ms are pointing backward and to the left

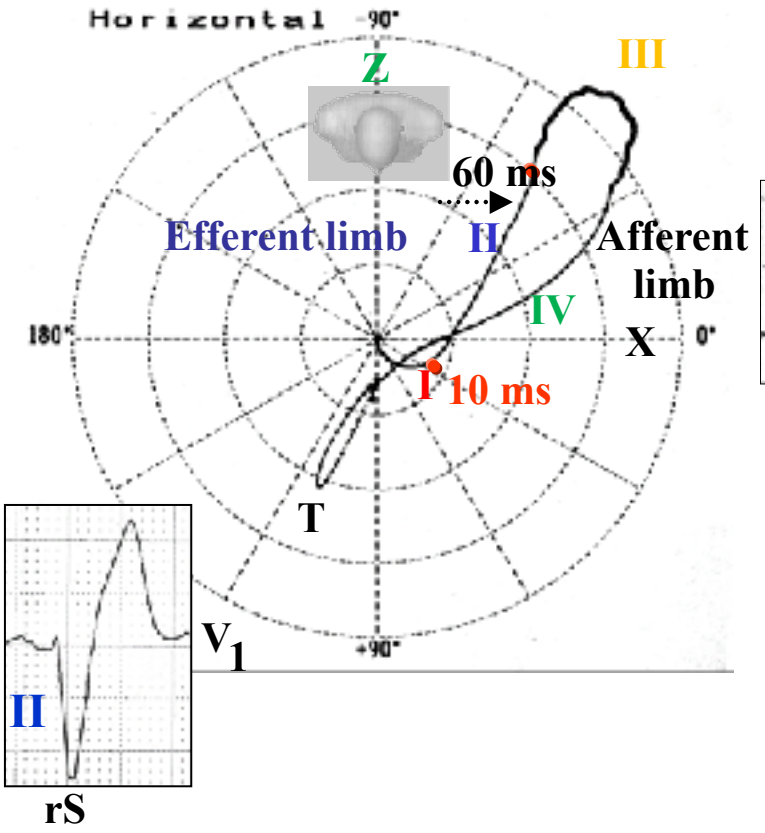
The QRS loop has a clockwise rotation (CWR) and clear middle-final conduction delay.

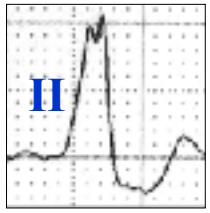
QS pattern is recorded from V1 to V3, which may be confused with anterior or anteroseptal MI.

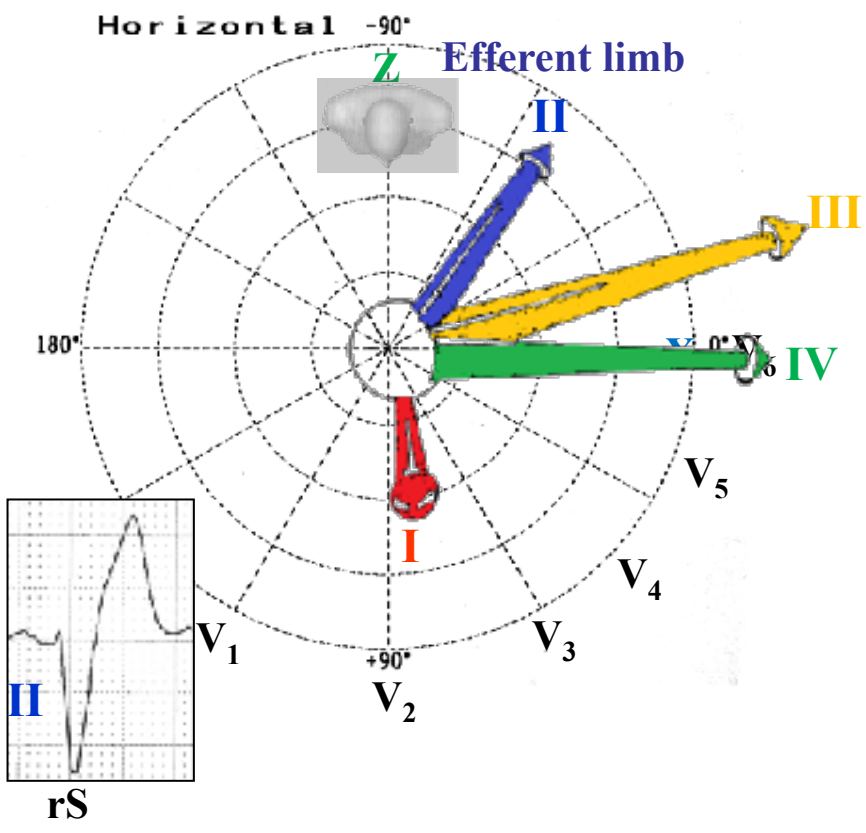
Only V6 shows the typical pattern in tower of monophasic R wave with middle-final conduction delay.



Ventricular activation sequence in CLBBB on HP: Phase II – 10 to 60 ms vector (duration 50 ms). It is responsible in the HP for the fast centrifugal efferent limb of the QRS loop

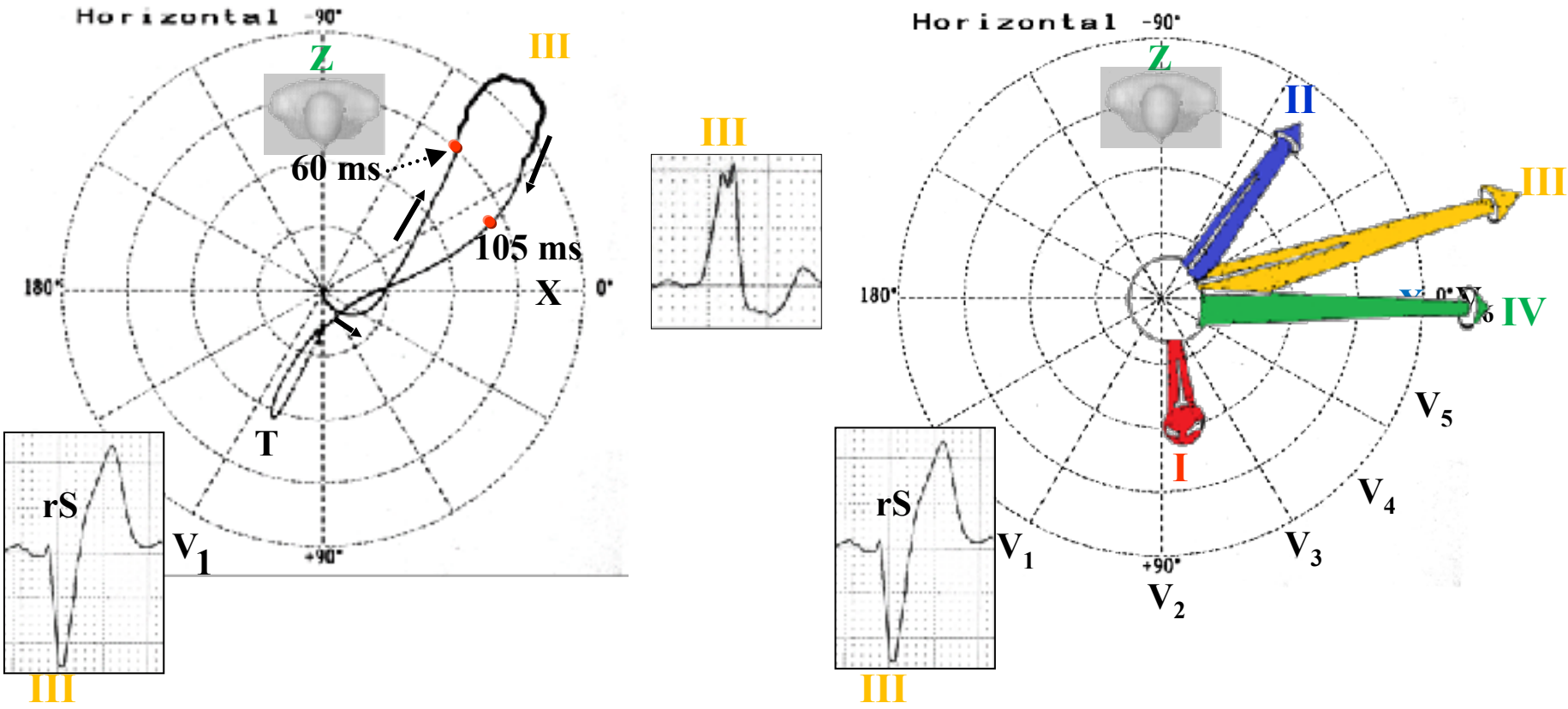






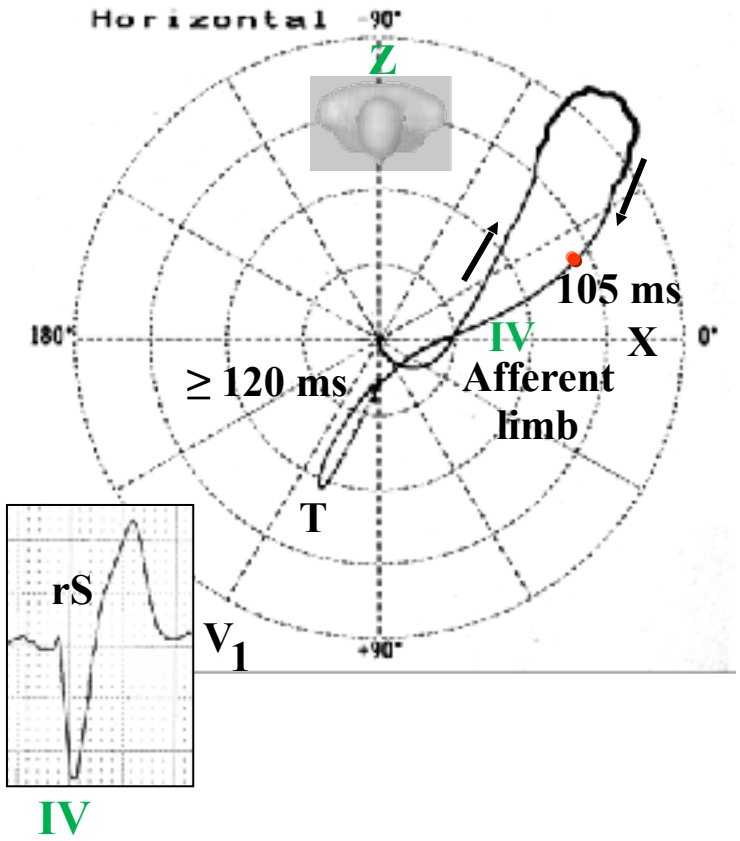
Outline that shows the second phase or phase 2 of depolarization in CLBBB (blue color).
In the VCG QRS-loop, phase 2 corresponds to the efferent limb of fast recording and it is located to the right in relation to the afferent limb.
Vector II corresponds to the ascending ramp of R wave of left leads (V_5 & V_6) and the descending ramp of S wave of right precordial leads (V_1 & V_2)

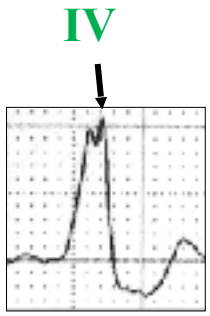
Ventricular activation sequence in CLBBB in the HP: Phase III – 60 to 105 ms vector (duration of 45 ms)

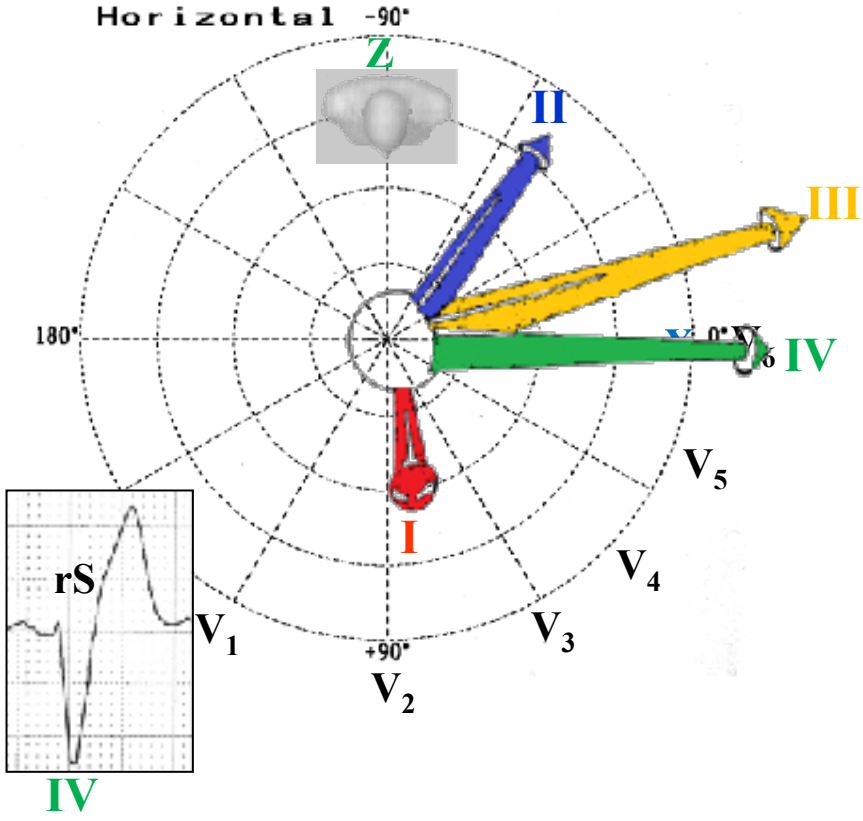


Outline that shows the third phase or phase 3 of depolarization in CLBBB. In the VCG loop, phase 3 corresponds to high septal and posterobasal activation of the LV free wall, responsible for the initial apex of R wave of left leads and nadir of S wave in V1. Vector III is processed slowly (dashes very close to each other). It represents high septal and posterobasal activation of the LV free wall. It is responsible for initial apex of R wave of left leads and by the nadir of S wave of right precordial leads.

Ventricular activation sequence in CLBBB in the HP: Phase IV –105 to ≥ 120 ms vector (duration from 15 to 70 ms)

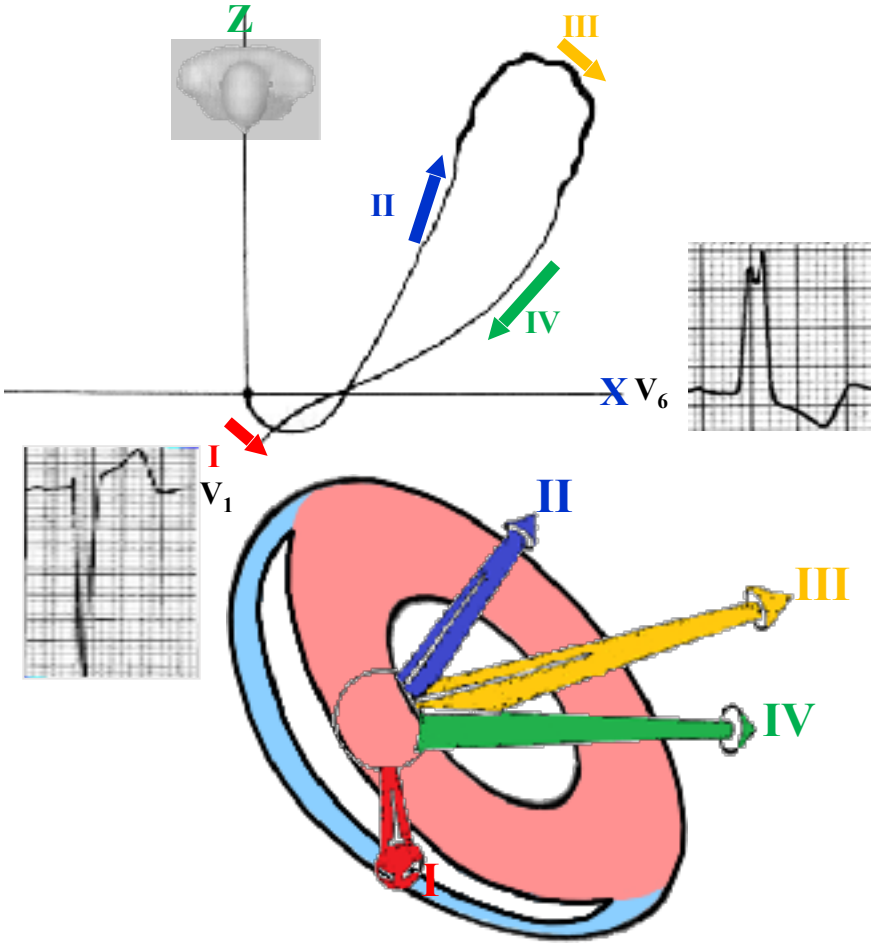


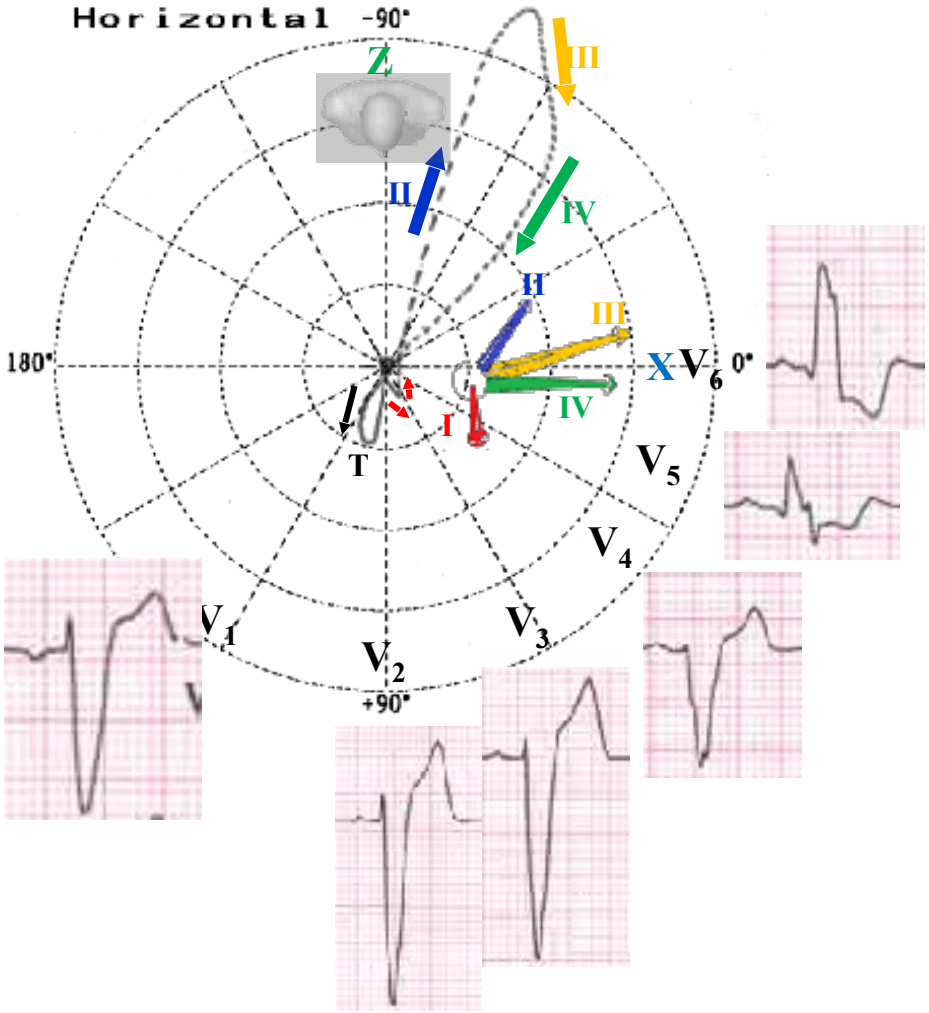




Outline that shows the fourth phase or phase 4 of depolarization in CLBBB. In the VCG QRS-loop, phase 4 corresponds to the afferent limb (**IV**) of slow recording and located to the left of the efferent limb (**II**). Vector **IV** is also responsible for the second apex of R wave in left leads and by the second apex in the nadir of S wave of V1 and V2.

Outline that shows the four depolarization-activation vectors in CLBBB in the HP. There is an ECG/VCG correlation of the QRS loop and the leads V1 and V6





- ✓ The QRS loop duration is ≥120 ms
- ✓ The QRS loop shape is elongated and narrow
- ✓ The main body of the QRS loop is inscribed posteriorly and to the left within the range - 90 to - 40°.
- ✓ Conduction delay noted in the mid and terminal portion
- ✓ The main body of QRS loop is inscribed clockwise (CW)
- ✓ The magnitude of the max QRS vector is increased above normal exceeding 2 mV.
- ✓ ST segment and T wave vector are directed rightward and anteriorly (opposite to QRS-loop)

Summary of QRS VCG loop in the HP in uncomplicated and complicated LBBB (Sodi-Pallares 1970; Goldman 1969; Cabrera 1959)

A) Uncomplicated Left Bundle Branch Block

1) The initial 10 to 20 ms vector is directed to the left and anteriorly;
2) The main body of the QRS loop is inscribed clockwise (CW)
3) The magnitude of the max QRS vector is increased above normal exceeding 2 mV.

B) Complicated Left Bundle Branch Block

(B-1) Anteroseptal myocardial infarction (Medrano 1963)

1) The initial forces (20 ms) are directed to the left and posteriorly.
2) The 20 ms vector is displaced posteriorly and usually inferiorly in the LSP
3) The main body of the QRS loop is inscribed clockwise (CW).

(B-2) Localized anterior myocardial infarction

1) The 10 ms vector is directed rightward and anteriorly, which indicates that initial forces are present
2) In the LSP the 10 ms vector is directed anteriorly and superiorly, but the 20 ms vector is displaced posteriorly and inferiorly.

(B-3) Anterolateral, lateral or free wall myocardial infarction (Doucet 1966)

1) The main body of the QRS loop has a counterclockwise inscription with the afferent limb to the right of the efferent limb in the HP.

(B-4) Massive septal myocardial infarction

1) The HP shows a large initial deflection in a CCW direction with the rest of the QRS loop displaying the usual features of the LBBB.

(B-5) Inferior myocardial infarction associated with left bundle-branch block

The diagnosis of inferior myocardial infarction with LBBB was made by VCG

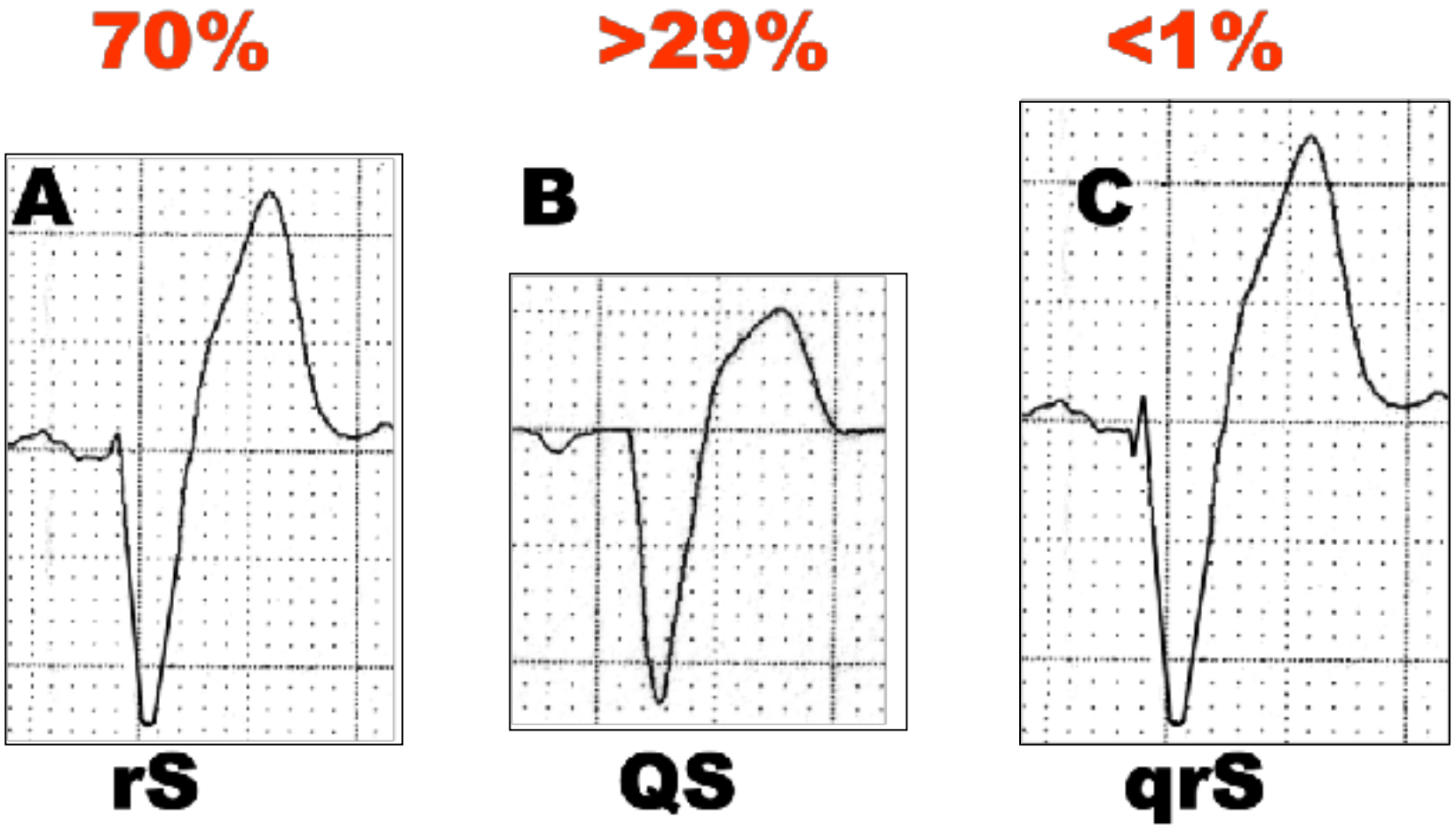
1) An upwards displacement of the QRS loop with preservation of the superior orientation of the initial forces.
2) Atypical appearances of LBBB with a posterior and right-sided shift of the efferent loop following the anterior and left-sided orientations of the initial forces.

The sensitivity of the VCG is low in inferior myocardial infarction with LBBB because the block may mask the electrical signs of inferior infarction (**Bruntz 1985**).

Value of VCG for the diagnosis of acute myocardial infarction in the presence of LBBB (Eriksson 1997)

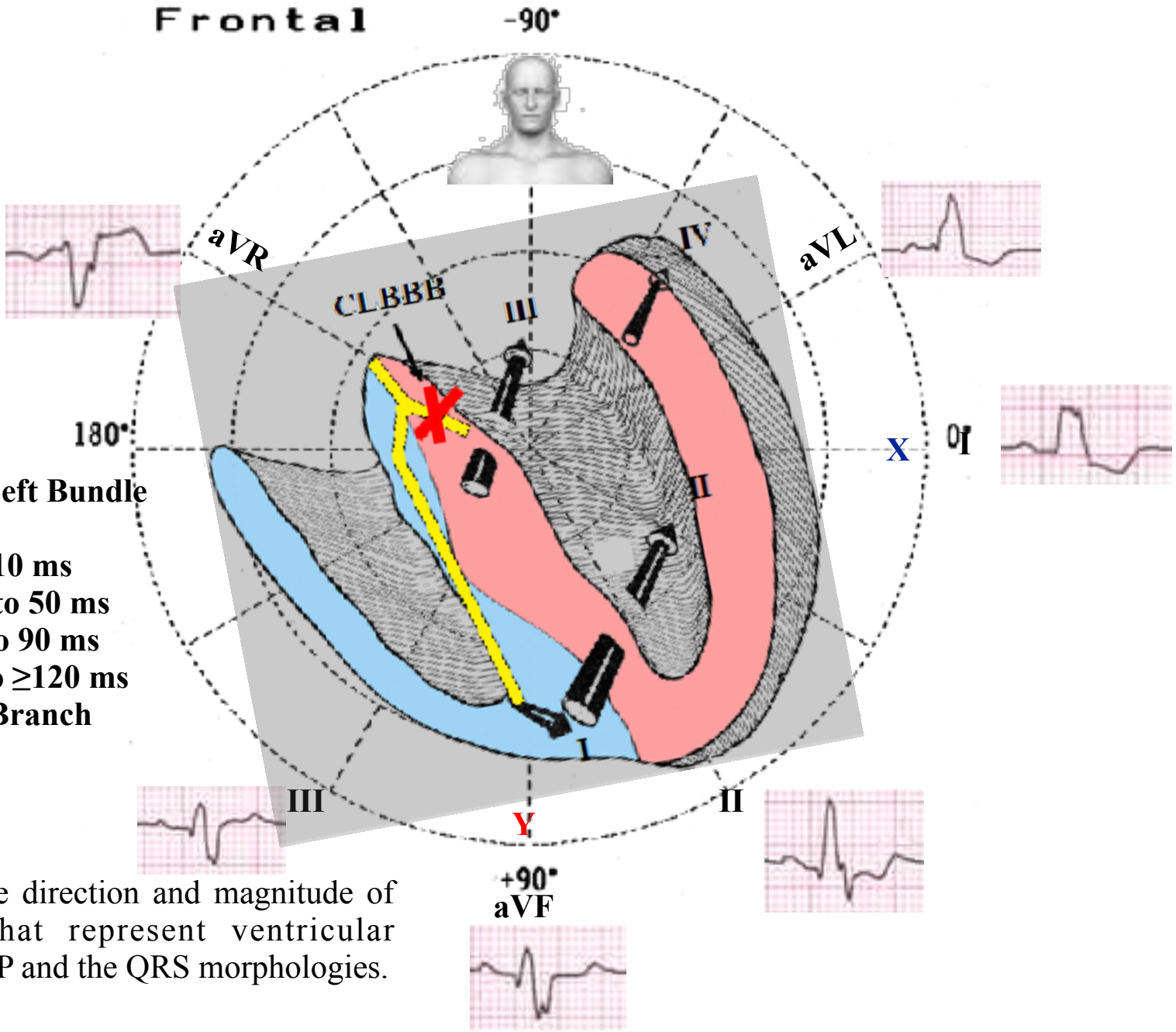
Eriksson et al studied 65 patients admitted to the coronary care unit with bundle-branch block and suspected acute MI monitored by dynamic VCG. In 28 patients, a clinical diagnosis of acute MI was made. In patients with LBBB and acute MI, the pattern of QRS vector-difference evolution was similar to that in patients with the narrow QRS complex, while ST vector-magnitude changes increased over time. Using a cut-off value for QRS vector-difference at 12 h of more than 20 microVs and a specific trend curve pattern, acute MI in the presence of LBBB could be diagnosed with an accuracy of 71%. Dynamic VCG is a valuable tool in diagnosing and monitoring acute MI in patients with bundle branch block.

Morphologies from V1 to V2 in non-complicated CLBBB



The three possible morphologies in V1 in non-complicated CLBBB: rS (70%), QS (>29%) and qrS (<1%).

Sequence of ventricular activation in CLBBB in the FP

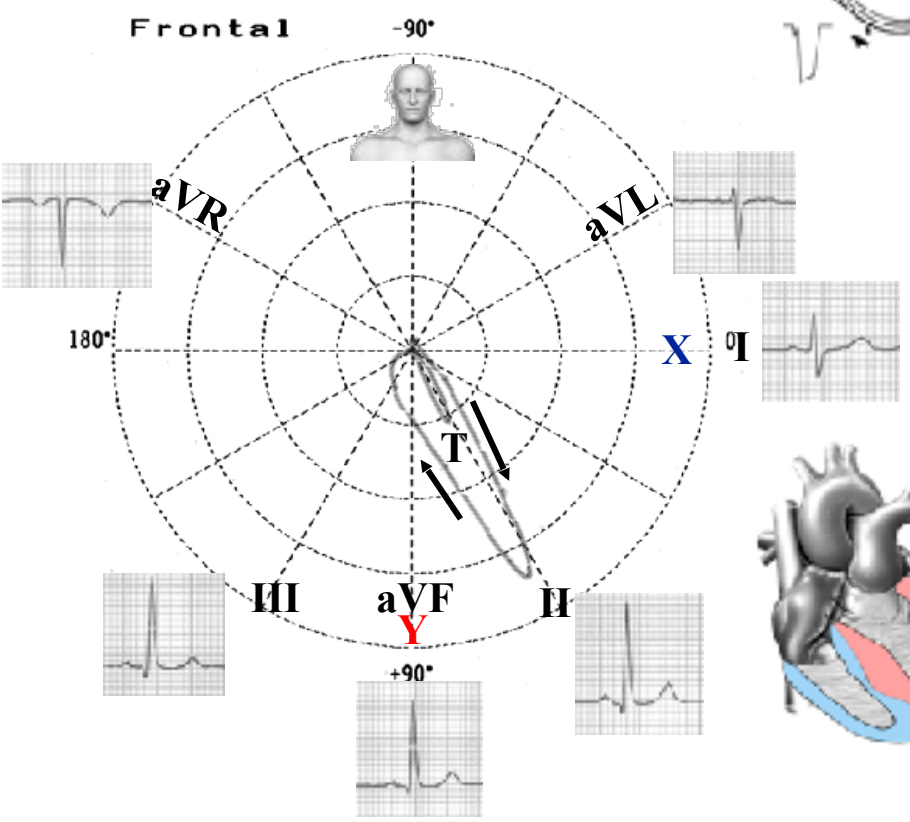


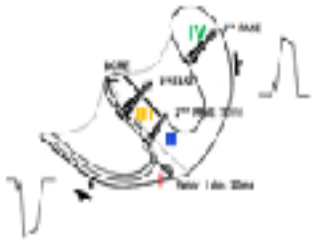
CLBBB: Complete Left Bundle Branch Block
1st phase: from 0 to 10 ms
2nd phase: from 10 to 50 ms
3rd Phase: from 50 to 90 ms
4th phase: from 90 to ≥120 ms
RBB: Right Bundle Branch

Outline that shows the direction and magnitude of the four vectors that represent ventricular depolarization in the FP and the QRS morphologies.

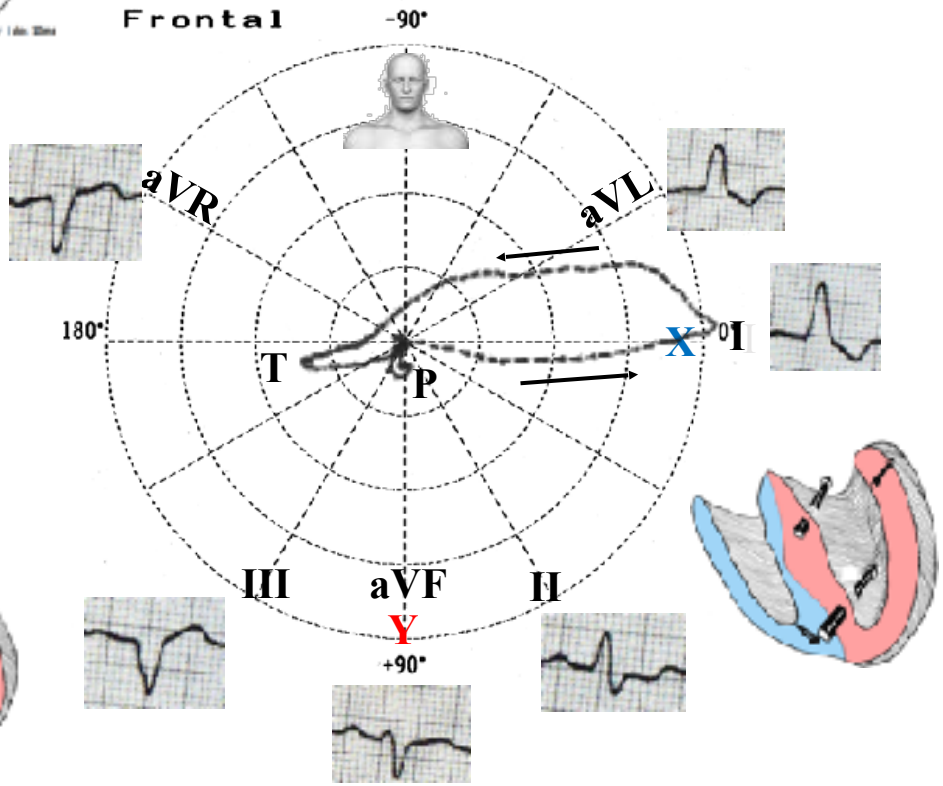
ECG/VCG correlation in the FP in normal conduction and CLBBB

Normal QRS and T loops



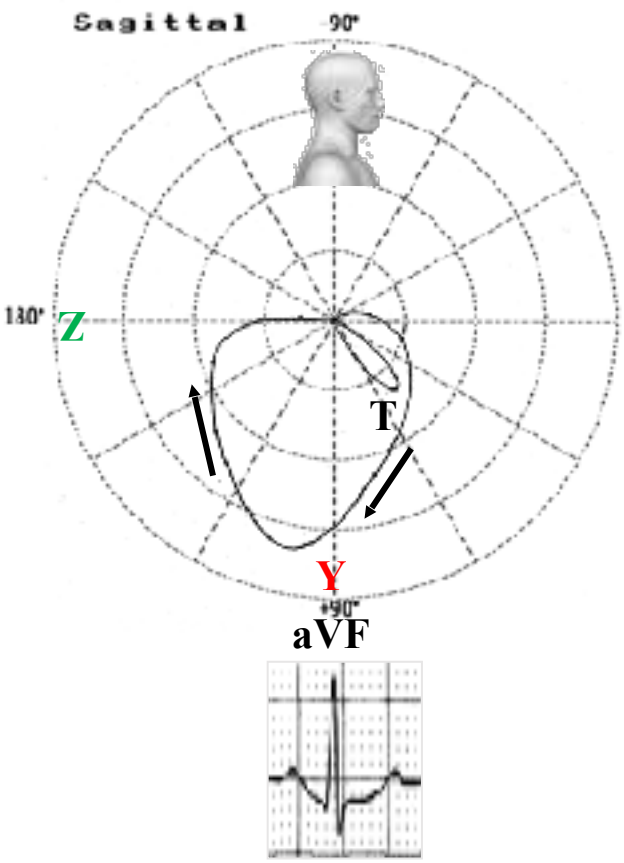


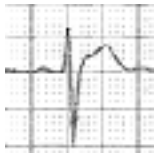
QRS and T loops in the LBBB

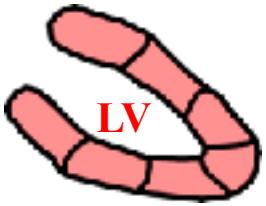


ECG/VCG correlation in the RSP in normal conduction and CLBBB

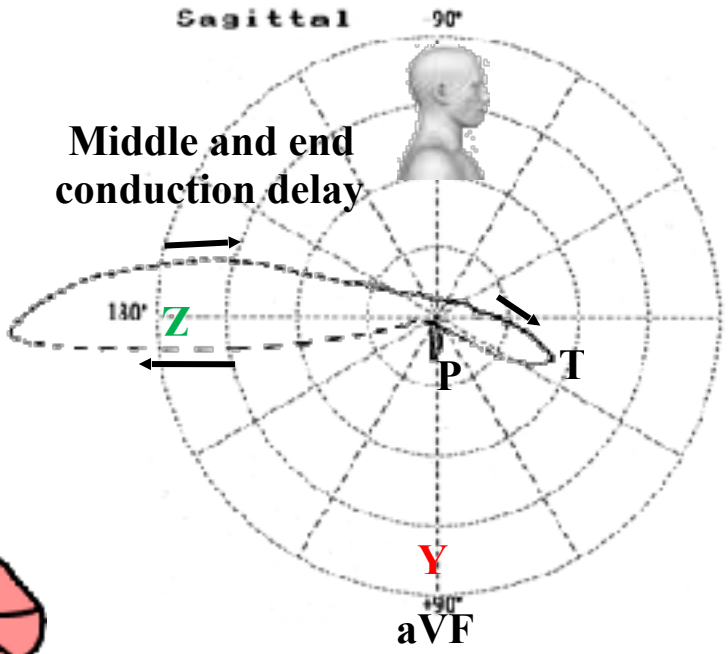
Normal QRS and T loops

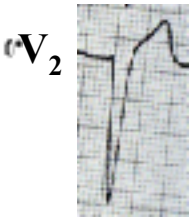






QRS and T loops in the LBBB





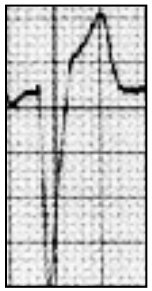
ECG criteria in non-complicated Complete LBBB and correlation with VCG in the HP

1. Supraventricular command: If the rhythm is sinus, the PR interval is ≥ than 120 ms.
2. **QRS duration:** ≥120 ms in adults, ≥100 ms between 4 to 16 years of age and ≥90 ms in children less than 4 years of age. If New York Heart Association Class II-IV heart failure is present, and LVEF≤35%, ECG QRS width ≥120 ms in the presence of LBBB, cardiac resynchronization therapy is indicated. Reevaluation of the data of cardiac resynchronization trials and electrophysiological findings in LBBB provided evidence that "true" LBBB requires a QRS width of ≥130 ms (in woman) and ≥140 ms (in man). In "true" LBBB, after the 40th ms of QRS, notched/slurred R waves are characteristic in at least two of I, aVL, V1, V2, V5 and V6 leads, in addition to a ≥40 ms increase of the QRS complex, as compared to the original QRS complex. In contrast, slowly and continuously widened "LBBB like" QRS patterns mostly occur in LVH or in a metabolic/infiltrative disease (**Préda 2013**). Cardiac resynchronization therapy (CRT) has emerged as an attractive intervention to improve left ventricular mechanical function by changing the sequence of electrical activation. Unfortunately, ≈30% of patients receiving CRT do not benefit (non-responders) but are subjected to device complications and costs. Thus, there is a clear need for better selection criteria. Three key studies have suggested that $^1/_3$ of patients diagnosed with LBBB by conventional ECG criteria may not have true complete LBBB, but likely have a combination of LVH and LAFB. **Note**: Current criteria for CRT eligibility include a QRS duration ≥120 ms. However, studies have suggested that only patients with LBBB benefit from CRT, and not patients with RBBB or nonspecific intraventricular conduction delay. Strauss et al (**Strauss 2011**) reviewed the pathophysiologic and clinical evidence supporting why only patients with complete LBBB benefit from Cardiac Resynchronization Therapy (CRT). Additionally, they review how the threshold of 120 ms to define LBBB was derived subjectively at a time when criteria for LBBB and RBBB were mistakenly reversed. These authors propose stricter criteria for complete LBBB that include a QRS duration ≥140 ms for men and ≥130 ms for women, along with mid-QRS notching or slurring in ≥2 contiguous leads. Further studies are needed to reinvestigate the electrocardiographic criteria for complete LBBB and the implications of these criteria for selecting patients for CRT

For this entity, the term latent cardiomyopathy had been suggested previously (**Breithardt G 2012**). New strict LBBB criteria increase the specificity of complete LBBB diagnosis in the presence of LV hypertrophy/dilatation and incomplete LBBB, which is critical for selecting CRT patients (**Galiotti 2013**). In patients with guideline-defined LBBB, the absence of ECG markers of residual left bundle conduction was predictive of a greater improvement in LV function with CRT. An r wave ≥1 mm in lead V1 (r-V1) and/or a q wave ≥1 mm in lead aVL (q-aVL) is used to identify patients with residual LB conduction (**Perrin 2012**). In patients with conventional wider LBBB morphology, the presence of mid-QRS notching or slurring is a strong predictor of better response to CRT (**Tian 2013**). The typical surface ECG feature of LBBB is a prolongation of QRS above 110 ms in combination with a delay of the ventricular activation time, intrinsicoid deflection or "R peak time" in left leads V5 and V6 of more than 60 ms and no septal q waves in leads I, V5, and V6 due to the abnormal septal activation from right to left. LBBB may induce abnormalities in left ventricular performance due to abnormal asynchronous contraction patterns which can be compensated by biventricular pacing (resynchronization therapy). Asynchronous electrical activation of the ventricles causes regional differences in workload which may lead to asymmetric hypertrophy and left ventricular dilatation, especially due to increased wall mass in late-activated regions, which may aggravate preexisting left ventricular pumping performance or even induce it. Of special interest are patients with LBBB and normal left ventricular dimensions and normal LVEF at rest but who may present with an abnormal increase in pulmonary artery pressure during exercise, production of lactate during high-rate pacing, signs of ischemia on myocardial scintigrams (but no coronary artery narrowing), and abnormal ultrastructural findings on myocardial biopsy.

3. QRS complexes in the right precordial leads (V1 and V2) total or predominantly negative: rS, QS or qrS.

70%

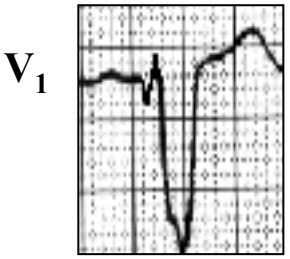


rS

29%

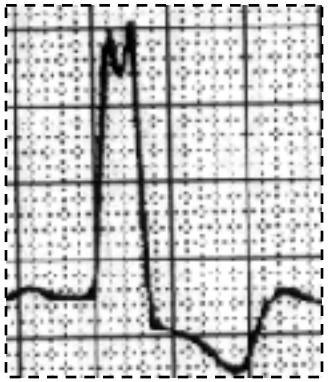
QS

<1%

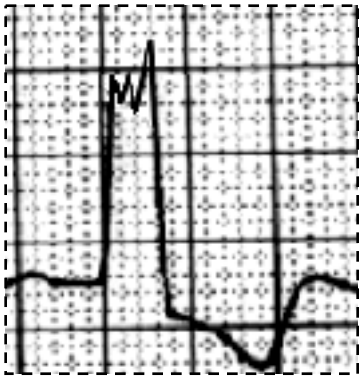


qrS

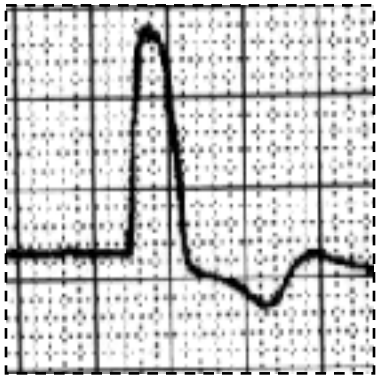
4. Monophasic, broad notched or slurred R wave, recorded slowly in the left leads: I, aVL, V5 and V6.



"Tower" with one notch



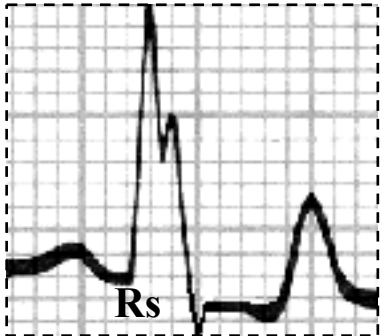
"Tower" with double notch



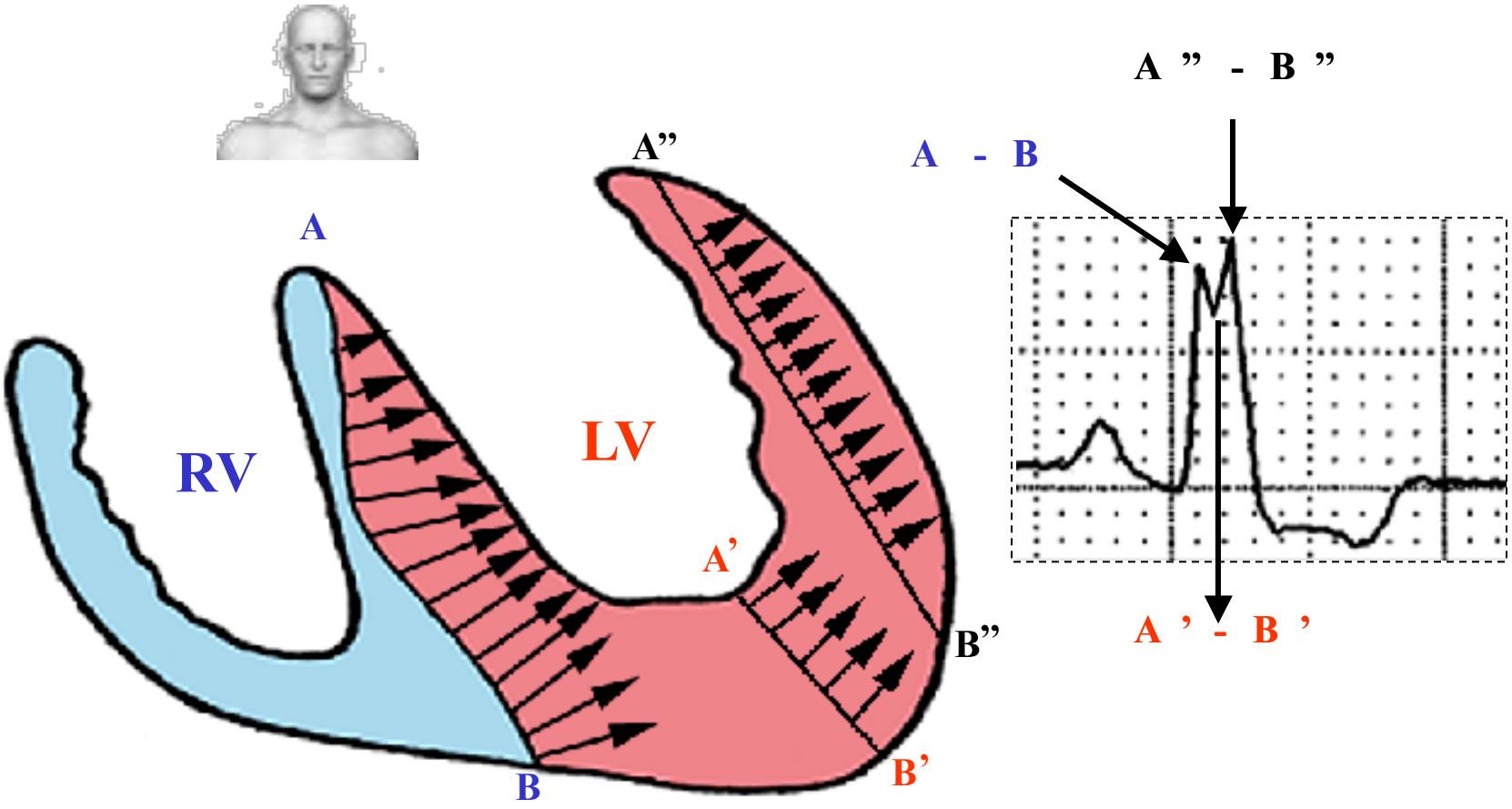
Plateau

There may be initial narrow q in aVL and exceptionally in I, however, never in V_5 and V_6.
Occasional Rs or RS pattern in V_5 and V_6. In this case , it may indicate:

1) Displaced transition of QRS complex to left;
2) Associated right ventricular hypertrophy or RVE;
3) Associated left anterior fascicular block (LAFB);
4) Electrically inactive area (MI) of free wall associated to complete LBBB;



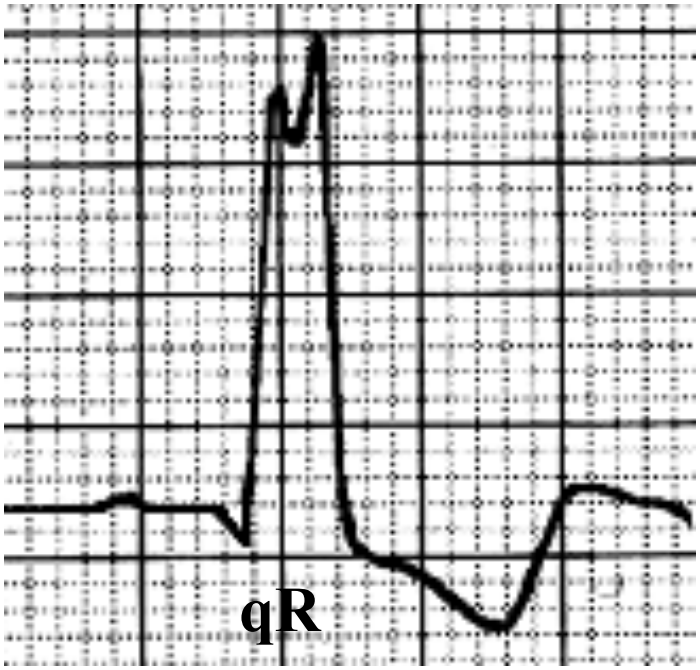
Monophasic R wave of slow recording in left leads I, aVL, V5 and V6 and electrophysiological explanation



Septal depolarization from right to left makes a wide A-B wave front; however, when the stimulus reaches the central portion of the LV (cavity), it suffers a marked decrease in wavefront width (A’-B’) responsible for the notch in the apex of R wave. Next, the wavefront reaches the LV free wall increasing again the width of the wavefront (**A”-B”**), responsible for the second apex of the R wave. In the severe hypertrophies of the free wall, this second apex presents a higher voltage related to the first one.

Initial q wave left leads

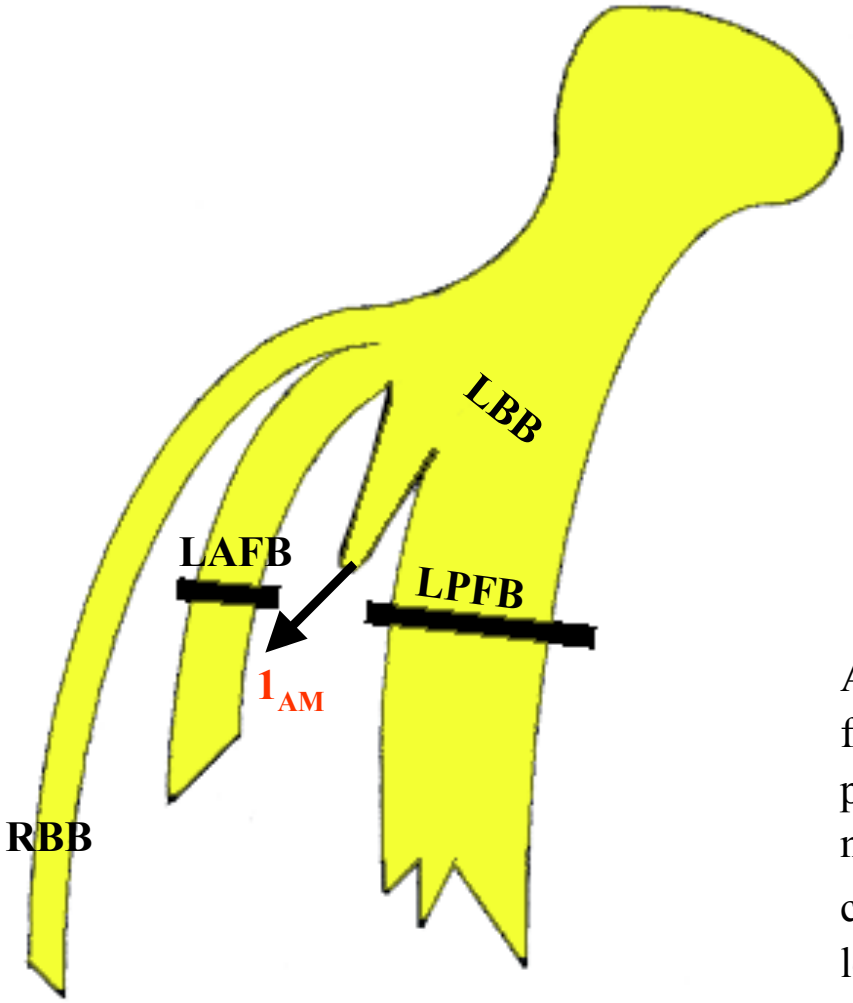
aVL

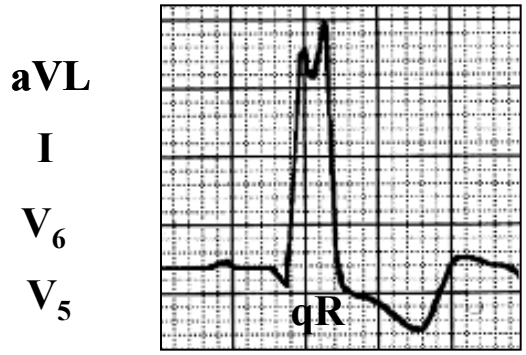


In Complete LBBB the absence of initial q waves in leads I, V5 and V6 is characteristic, but in the lead aVL a narrow q wave may be present in the absence of myocardial pathology.

The pure monophasic R wave is characteristic in the left leads (I, aVL, V5 and V6). Since the aVL lead is higher, it can rarely show qR pattern in absence of complicated LBBB. When the left septal division emerges before the block area, it preserves the septal vector I as normal, heading to the right and the front: qR in left leads (atypical CLBBB). See next slide.

Divisional CLBBB with initial q wave in left leads

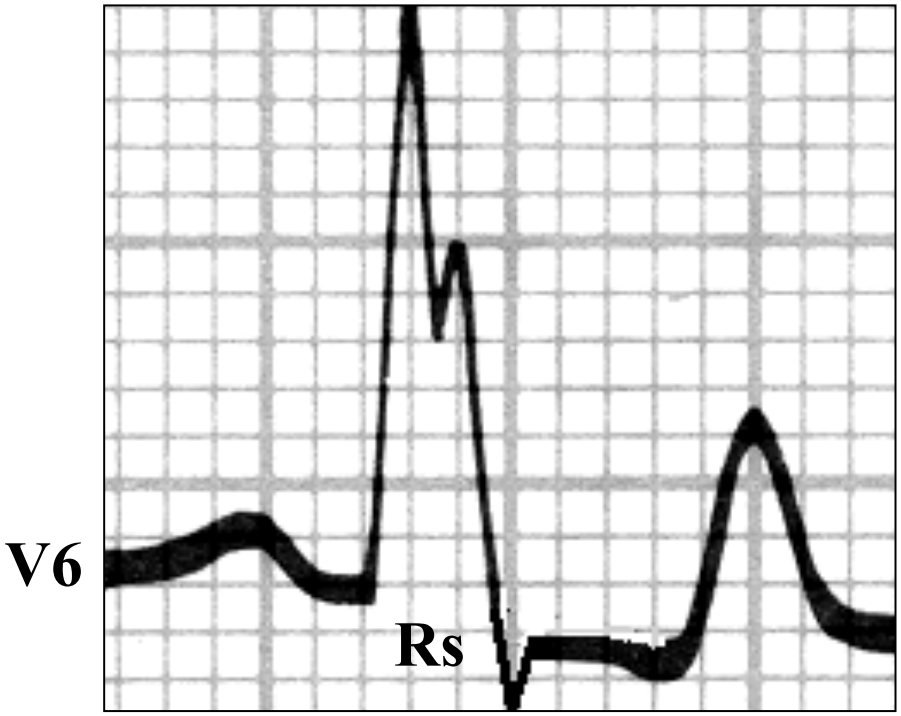




According to Medrano (**Medrano 1970**), if the fibers of the septal fascicle (SF) originate proximally to the block of the LAF and LPF, the middle-septal activation is preserved (1_{AM}), causing atypical CLBBB with q waves in left leads.

Outline of CLBBB with q wave in left leads. The left septal division emerges before the block area, preserving septal vector I as normal, heading to the right and the front: qR in left leads (atypical CLBBB).

Occasional Rs or RS pattern in V_5 & V_6

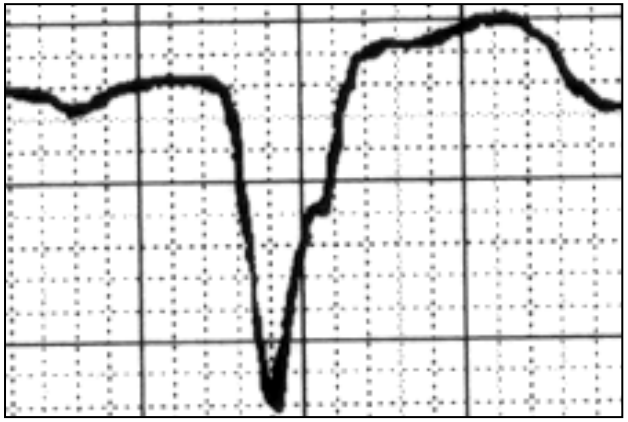


The left leads may show Rs pattern in absence of another associated problem. The presence of Rs pattern in the left precordial leads may indicate:

1. Transitional recording due to LVH: Displaced transitional zone of QRS complex to the left;
2. Association with Left Anterior Fascicular Block;
3. Association with Right Ventricular Hypertrophy;
4. Association with myocardial infarction on LV free wall.

QRS pattern in aVR

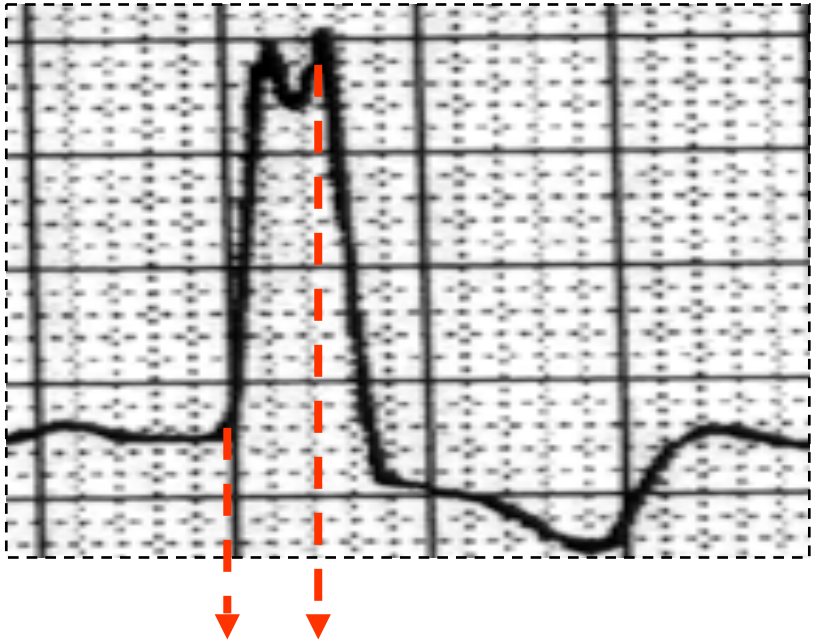
aVR



QS

QRS complex of the QS type almost constant in aVR.

Prolonged Ventricular Activation Time (VAT) in left leads

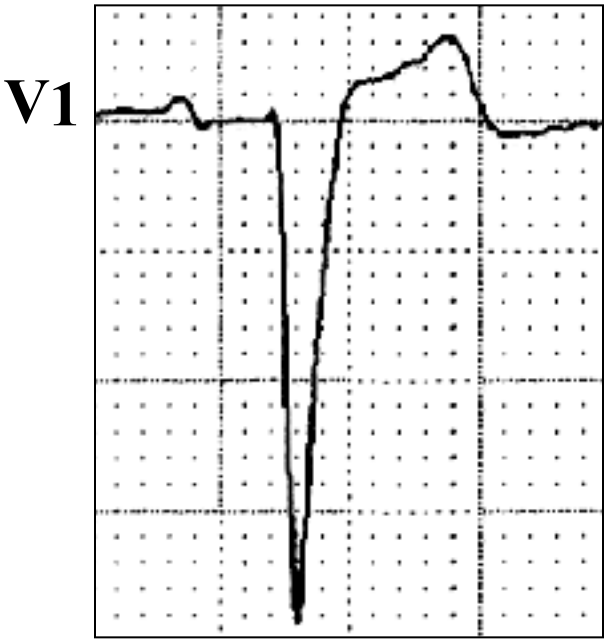


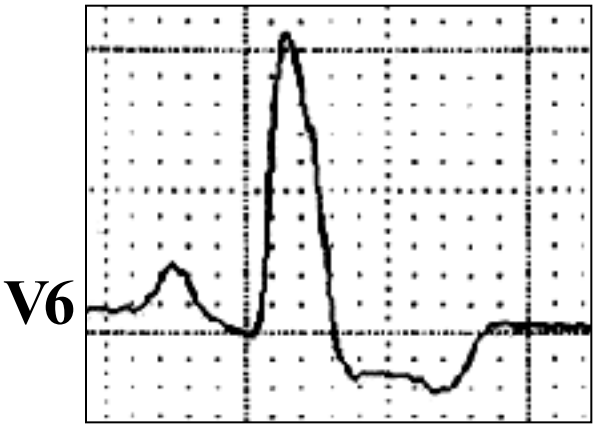
VAT ≥ 50 ms

VAT, intrinsicoid deflection or R-peak time ≤60 ms in I and V5-V6 but normal in V1-V2 and V3, when small initial r waves can be discerned in the above leads.

Repolarization in V_1 and V_6 in uncomplicated complete LBBB in secondary repolarization abnormalities

ST and T waves usually opposite in direction to QRS



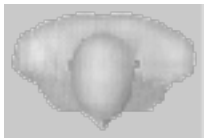


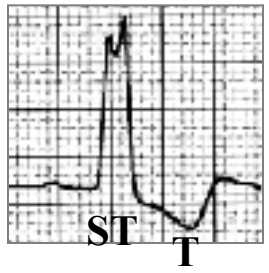
Depressed ST segment and/or negative T wave in leads with negative QRS (negative concordance) are abnormal (**Sgarbossa 1996; Gunnarsson 2001**).

Positive T waves in leads with upright QRS may be normal (positive concordance)

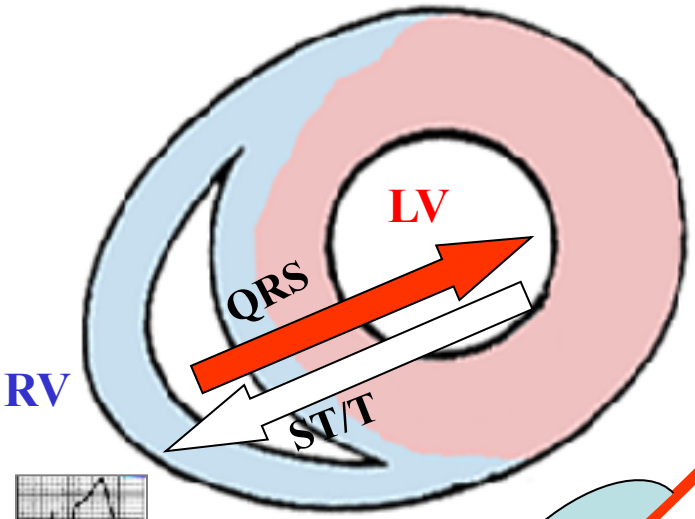
Abnormalities in the ST segment and T wave that occur as the direct result of changes in the sequence and/or duration of ventricular depolarization, manifested electrocardiographically as changes in QRS shape and/or duration, are referred to as secondary repolarization abnormalities. Recognition of secondary repolarization abnormalities is usually not difficult. In left bundle-branch block, the ST- segment and T-wave vectors are generally directed opposite to the mean QRS vector.

Ventricular repolarization in complete LBBB

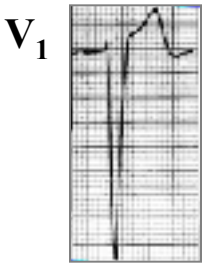


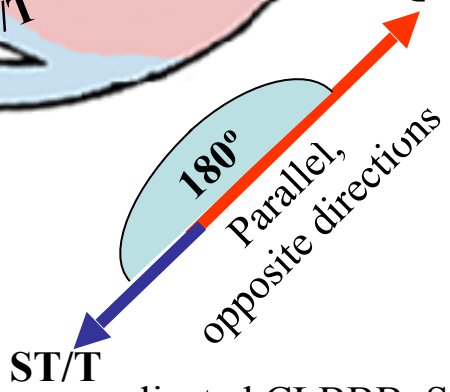


Depression upwardly convex of ST segment in left leads (I, aVL, V_5 and V_6). It is called Secondary Repolarization Abnormalities



ST segment elevation downwardly convex of ST segment in V_1 and V_2

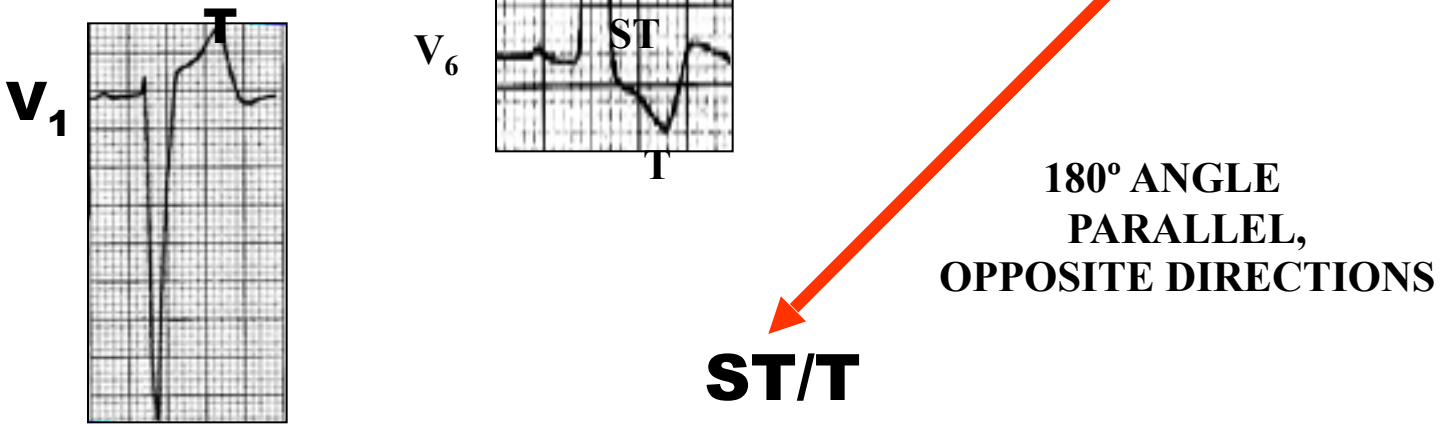




Outline representing ventricular repolarization in non-complicated CLBBB. Secondary alteration of ventricular repolarization is observed with QRS/ST-T angle near 180º. The ST-segment and T-wave vectors are generally directed opposite to the mean QRS vector. The distinction between primary and secondary repolarization abnormalities is clinically relevant because primary abnormalities indicate changes in the repolarization characteristics of ventricular myocytes, whereas secondary changes do not. The designation of the ST- and T-wave abnormalities as primary or secondary is appropriate, and it is recommended that automated interpretative algorithms be programmed to identify them.

Ventricular repolarization in Uncomplicated Complete LBBB

The ST- segment and T-wave vectors opposite to a greater deflection of QRS: positive from V_1 to V_3 and negative in left leads I, aVL, V_5 and V_6. These are Secondary Repolarization Abnormalities with wide QRS-ST-T angle and normal ventricular gradient. The classic ventricular gradient concept introduced by Wilson et al(1) in 1931 is of some theoretical interest concerning primary versus secondary repolarization abnormalities. Ventricular gradient in a single ECG lead is the net time integral of the ECG voltage from the beginning of the P wave to the end of the U wave. Its spatial counterpart is the ventricular gradient vector determined from the orthogonal XYZ leads. The practical usefulness of the ventricular gradient in differentiating primary from secondary repolarization abnormalities has not been demonstrated(2). When the direction of the QRS axis is normal, an abnormal direction of the T-wave axis is generally an indication of primary repolarization abnormalities.



Outline representing ventricular repolarization in non-complicated CLBBB. Secondary alteration of ventricular repolarization is observed with QRS/ST-T angle near the 180º.

1. **Wilson FN, Macleod AG., Barker PS. The T deflection of the electrocardiogramTrans Assoc Am Physicians, 46 (1931), pp. 29–38.**
2. **Surawicz, B. ST-T abnormalitiesMacFarlane PW, Lawrie TDV. (Eds.), Comprehensive Electrocardiology, Pergamon Books, Ltd, New York, NY (1988), pp. 511–563**

VCG and ECG criteria to distinguish new from old LBBB

There are no established criteria to differentiate new from old CLBBB. Differentiating these LBBB patterns is very important for the management of patients with LBBB in acute coronary syndrome scenarios (Shvilkin 2010). In a significant proportion of patients with LBBB in acute MI scenario with a culprit lesion and positive biomarkers, immediate catheterization with the intent of primary percutaneous coronary intervention is indicated when presenting with suspected ST-segment elevation myocardial infarction, ischemic symptoms, and presumed new LBBB, particularly if concordant ST-segment elevation is present. The table below shows the main ECG differences between new and old LBBB:

	New LBBB	Old LBBB
T-vector magnitude	**Larger: 1.20 +/- 0.07 mV**	**Smaller: 0.71 +/- 0.01 mV**
QRS/T vector magnitude ratio	**Smaller: 1.79 +/- 0.03**	**Larger: 3.92 +/- 0.04**
The ratio of deepest S to largest T wave in precordial leads (Max S/T)	**Smaller: 1.66 +/- 0.05**	**Larger: 3.54 +/- 0.08**

A rule using QRS/T <2.25 and max S/T <2.5 had 100% sensitivity and 96%-68% specificity in diagnosing new LBBB, including subsets of patients with tachycardia and ischemia.

Electrocardiographic classification criteria for Left Bundle Branch Block

I- According to the degree:

1. **Criteria (most used in literature):**
   - *a) Incomplete LBBB:* Incomplete Left Bundle Branch Block (QRS duration from 90 to 110 ms)
   - *b) Complete LBBB:* Complete Left Bundle Branch Block (QRS ≥120 ms) in adults.
   - c) Stricter criteria for complete LBBB: QRS duration ≥140 ms for men and ≥130 ms for women, along with mid-QRS notching or slurring in ≥ 2 contiguous leads. This new values are used for Cardiac Resynchronization Therapy (CRT) (**Strauss 2011**)

2. **Criteria from the Mexican School (Sodi 1964):**
   - a) 1st degree left bundle branch block;
   - b) 2nd degree left bundle branch block: a & b correspond to incomplete LBBB;
   - c) 3rd degree left bundle branch block or complete LBBB.
     - Complete LBBB by classical criteria: QRS duration ≥120 ms
     - Stricter criteria: QRS duration ≥140 ms (men) or 130 ms (women), QR or rS in leads V1 and V2, and mid-QRS notching or slurring in ≥2 of leads V1, V2, V5, V6, I and aVL.

3. **Criteria from the Spanish School (Bayés de Luna 2007). Global left ventricular blocks:**
   - a) Advanced left bundle branch block (ALBBB) or third degree (equivalent to CLBBB; QRS duration ≥120 ms),
   - b) Non-advanced global left ventricular blocks:
     - First degree LBBB (partial) corresponds to types I and II of Mexican school: isolated R in V6 with more or fewer slurring but QRS duration <120 ms.
     - Intermittent or second degree LBBB: corresponds to the special type of ventricular aberrancy.

Electrocardiographic classification criteria for Left Bundle Branch Block

II- According to topography:

a) Predivisional (90%) QRSD = 120 to 160 ms

- Of the left His bundle;
- Of the truncus of the left bundle branch;

Note: The intermittent forms are nearly always pre-divisional.

b) Fascicular or divisional: by unequal dromotropic involvement of divisions or fascicles of the left bundle branch: LAF, LPF and LSF.

c) Parietal, global Purkinjian, diffuse intraventricular, intramyocardial or intramural (in the Purkinje-muscle union). Characterized by: wider QRS, clockwise rotation of the QRS loop in the HP. In general, they point out greater myocardial involvement.

III- According to steadiness:

a) Permanent or definite: most of them.

b) Intermittent or of second degree that could be:

- **Rate-dependent intermittent LBBB (Arias 2006):**

  - Tachycardia-dependent or in "phase 3";
  - Bradycardia-dependent or in "phase 4".

- **Independent from heart rate:**

  - Mobitz type I;
  - Mobitz type II by Wenckebach phenomenon;
  - By significant hypopolarization.

Electrocardiographic classification criteria for Left Bundle Branch Block

IV- According to electrical axis of QRS complex in the Frontal Plane. See figure next slide.

a) With QRS axis not deviated: between -29° and +60° (≈ 65% to 70% of cases)

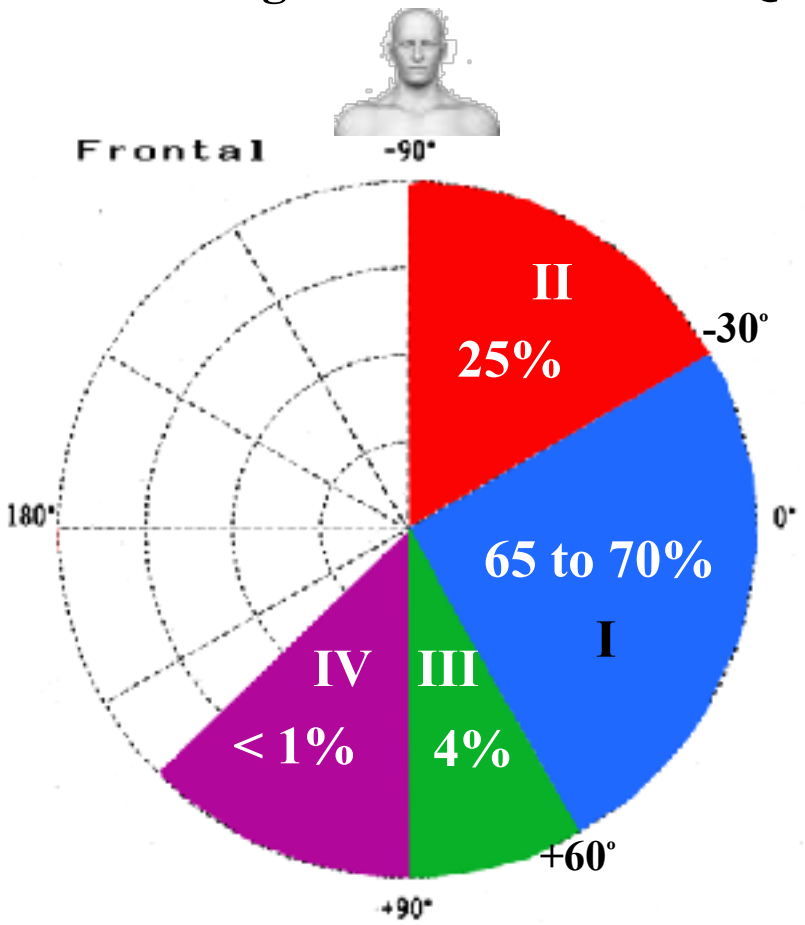
b) With QRS axis with extreme deviation to the left: beyond –30°: between -30° and -90° (**Parharidis 1997**) (≈25% of cases). The presence of left axis deviation had a 41.9% sensitivity and a 91.6% specificity for the presence of organic heart disease. Aortic valve disease in LBBB pts seems to be frequently accompanied by left axis deviation. In LBBB patients, those without left axis deviation seem to benefit more from cardiac resynchronization therapy with defibrillator (CRT-D) than those with left axis deviation **(Brenyo 2013**).

c) With QRS axis deviated to the right: between +60° and +90° (≈ 3.5 to 5% of cases)

d) With QRS axis with extreme deviation to the right: beyond +90° (< than 1% of cases). It is named "paradoxical type of Lepeschkin“ (**Lepeschkin 1951**). The majority of subjects had dilated cardiomyopathy with biventricular enlargement (**Childers 2000**). The uncommon combination of LBBB and right axis deviation is a marker of severe myocardial disease, specially primary congestive cardiomyopathy. The mechanism of production of this ECG pattern appears to be diffuse conduction system involvement in advanced myocardial disease (**Nikolic 1985**). Causes that determine paradoxical complete LBBB:

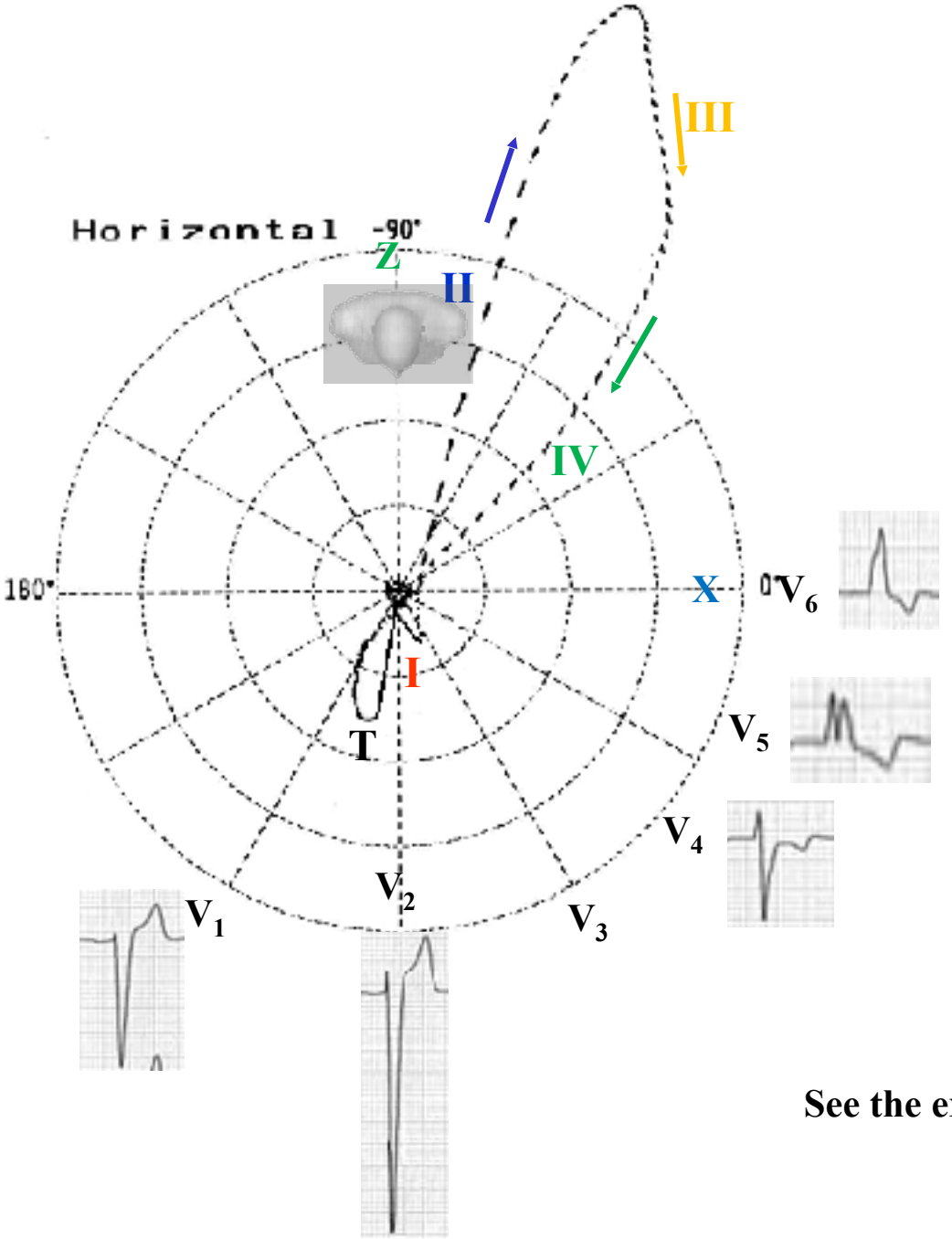
- Complete LBBB associated to right ventricular hypertrophy/enlargement or severe cardiomyopathy with biventricular enlargement or diffuse advanced myocardial disease.(3) >98% of cases.
- Fascicular Complete LBBB (LAFB + LPFB) with a higher degree of block in the postero-inferior division. In the presence of AF, LBBB with intermittent right axis deviation is explained by an additional LPFB accompanying predivisional LBBB (**Patenè 2008; 2012**)
- LBBB in Wegener granulomatosis (**Khurana 2000**)
- Complete LBBB associated to lateral infarction (free wall of left ventricle)
- Complete LBBB with accidental exchange of limb electrodes
- Complete LBBB associated with true dextrocardia (**Salazar 1978**)

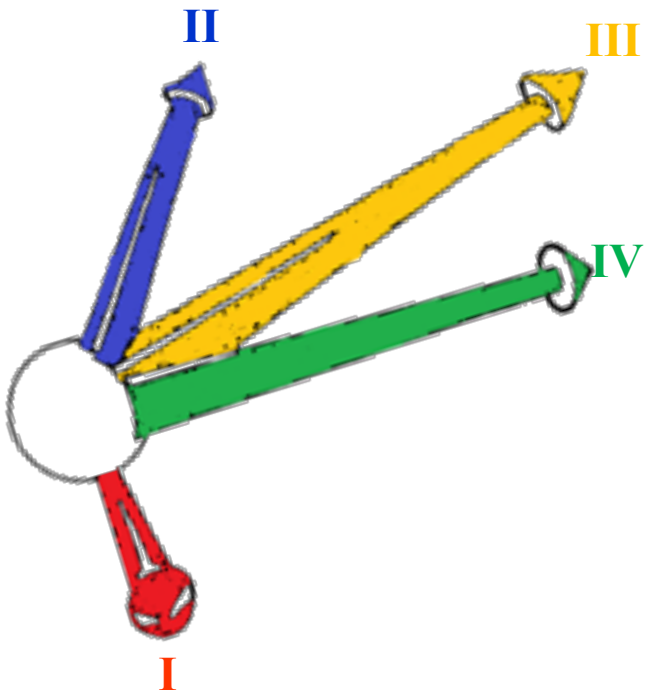
Types of CLBBB according to electrical axis of QRS complex in the FP



- With QRS axis not deviated: between -30° and +60° (≈ 65% to 70% of cases)
- With QRS axis with extreme deviation to the left: beyond –30⁰ (≈25% of cases)
- With QRS axis deviated to the right: between +60° and +90° (≈ 3.5 to 5% of cases)
- With QRS axis with extreme deviation to the right: beyond +90° (< than 1% of cases). It is named "paradoxical type of Lepeschkin“ (**Lepeschkin 1951**).

ECG/VCG correlation of CLBBB in the HP





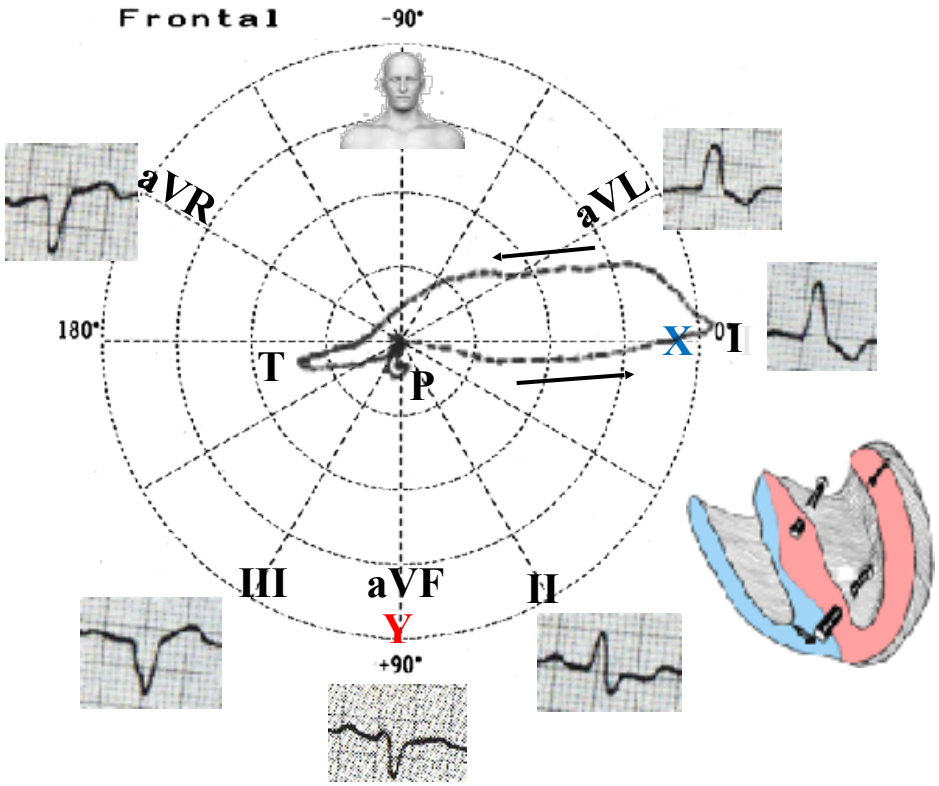
See the explanation in the next slide.

Vectorcardiographic criteria of non-complicated CLBBB in the HP

- Narrow, long QRS loop, and with morphology usually in 8.
- The QRS loop duration is ≥120 ms
- The QRS loop shape is elongated and narrow
- The main body of the QRS loop is inscribed posteriorly and to the left within the range -90 to -40°.
- Maximal vector of QRS located in the left posterior quadrant (between –40º to -80º) and of increased magnitude (>2 mV).
- Main portions of QRS loop of clockwise rotation. CCW rotation may indicate parietal CLBBB or complicated with lateral infarction or severe LVH.
- The efferent limb (**II**) located to right related afferent limb (**III** and **IV**).
- Conduction delay observed in the mid and terminal portion
- The main body of QRS loop is inscribed clockwise (CW)
- The magnitude of the max QRS vector is increased above normal exceeding 2 mV.
- ST segment and T wave vector are directed rightward and anteriorly.
- T loop of counterclockwise recording. The clockwise rotation of T wave in this plane suggests CLBBB complicated with infarction or LVH.

Vectorcardiographic criteria of non-complicated CLBBB in the FP

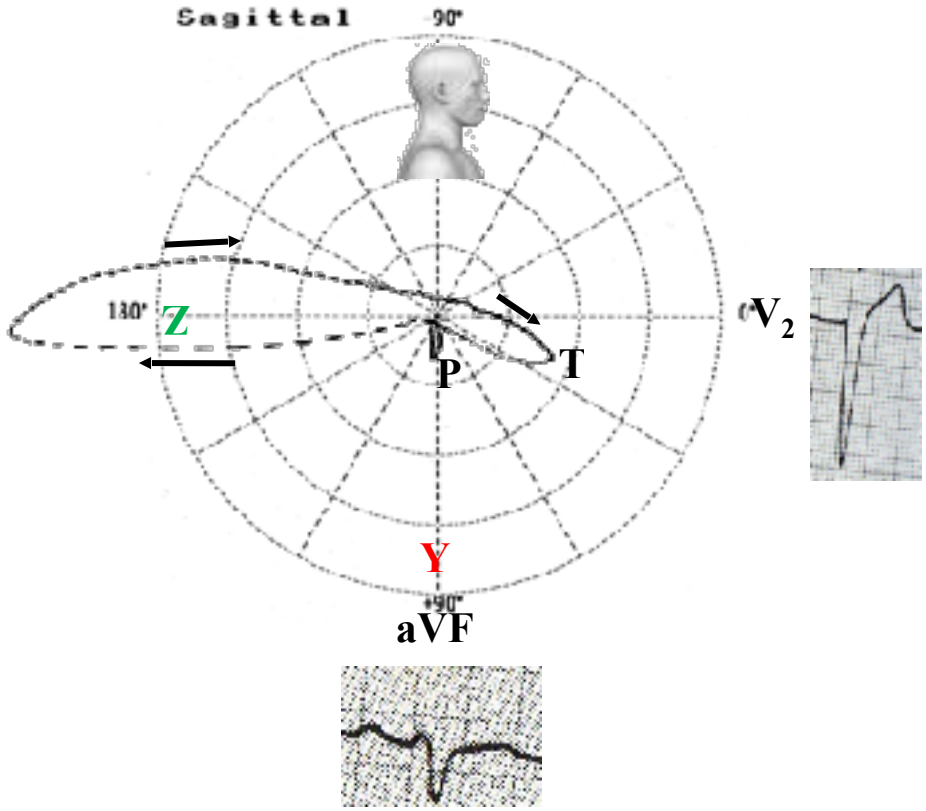
QRS and T loops in the LBBB



- Vector of initial 10 ms, to the left and inferior; rarely to the left and superior;
- QRS loop of counterclockwise rotation or in eight;
- QRS loop with characteristic middle final delay;
- Direction of maximal vector usually between +30º and –30º;
- Vectors of ST and T opposite to QRS (angle around 180º) and of counterclockwise rotation.

Vectorcardiographic criteria of non-complicated CLBBB in the RSP

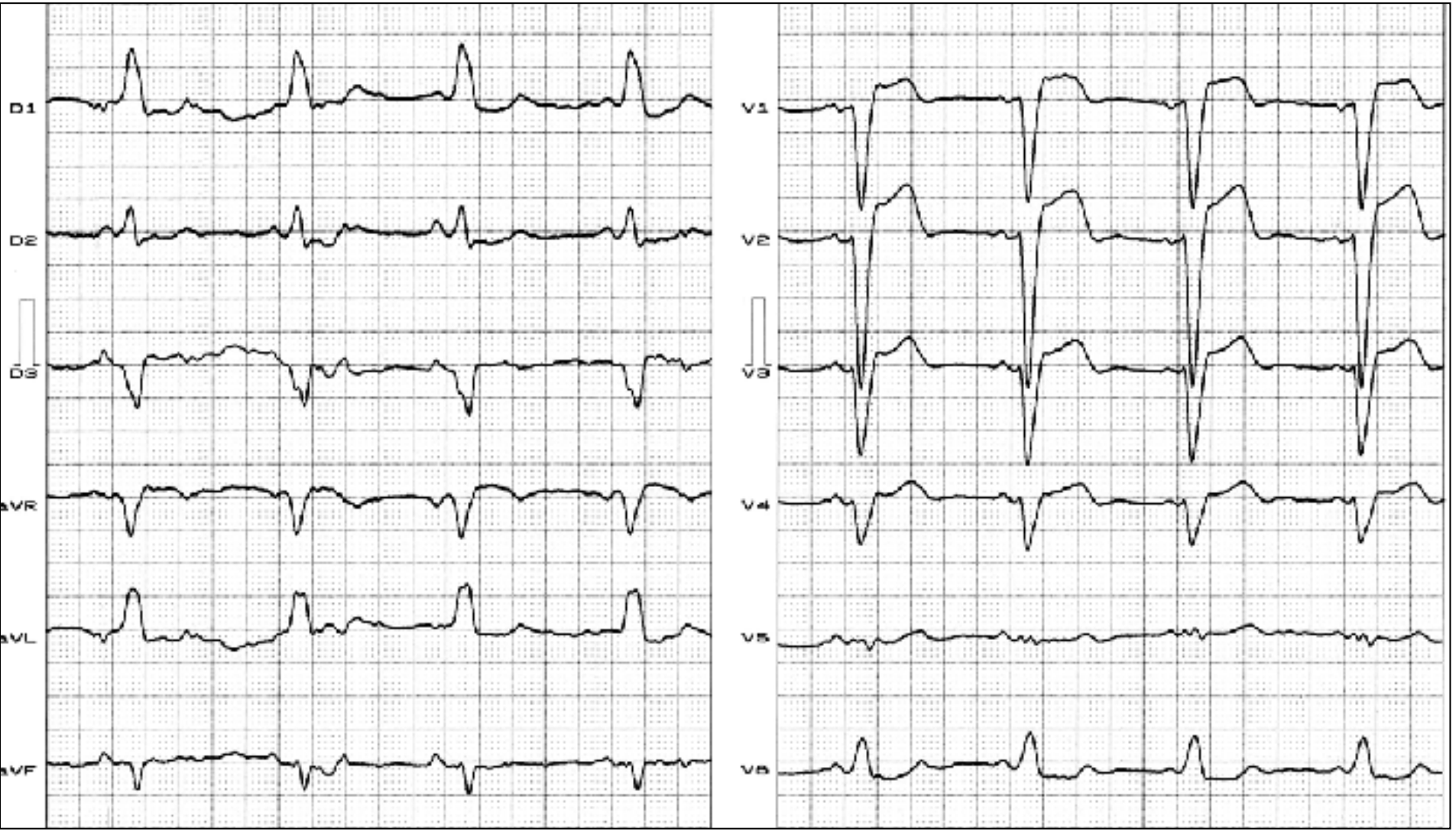
QRS and T loops in the LBBB



- Vector of initial 10 ms to the front and below (or to the back);
- QRS loop of clockwise rotation (RSP) or counterclockwise (LSP) rarely in 8;
- QRS loop with characteristic middle final delay;
- Direction of maximal vector of posterior orientation (between +150° and -175°);
- T loop of location opposite to the QRS loop (anterior) and of clockwise (RSP) or counterclockwise (LSP) rotation.

LBBB ECG / VCG examples

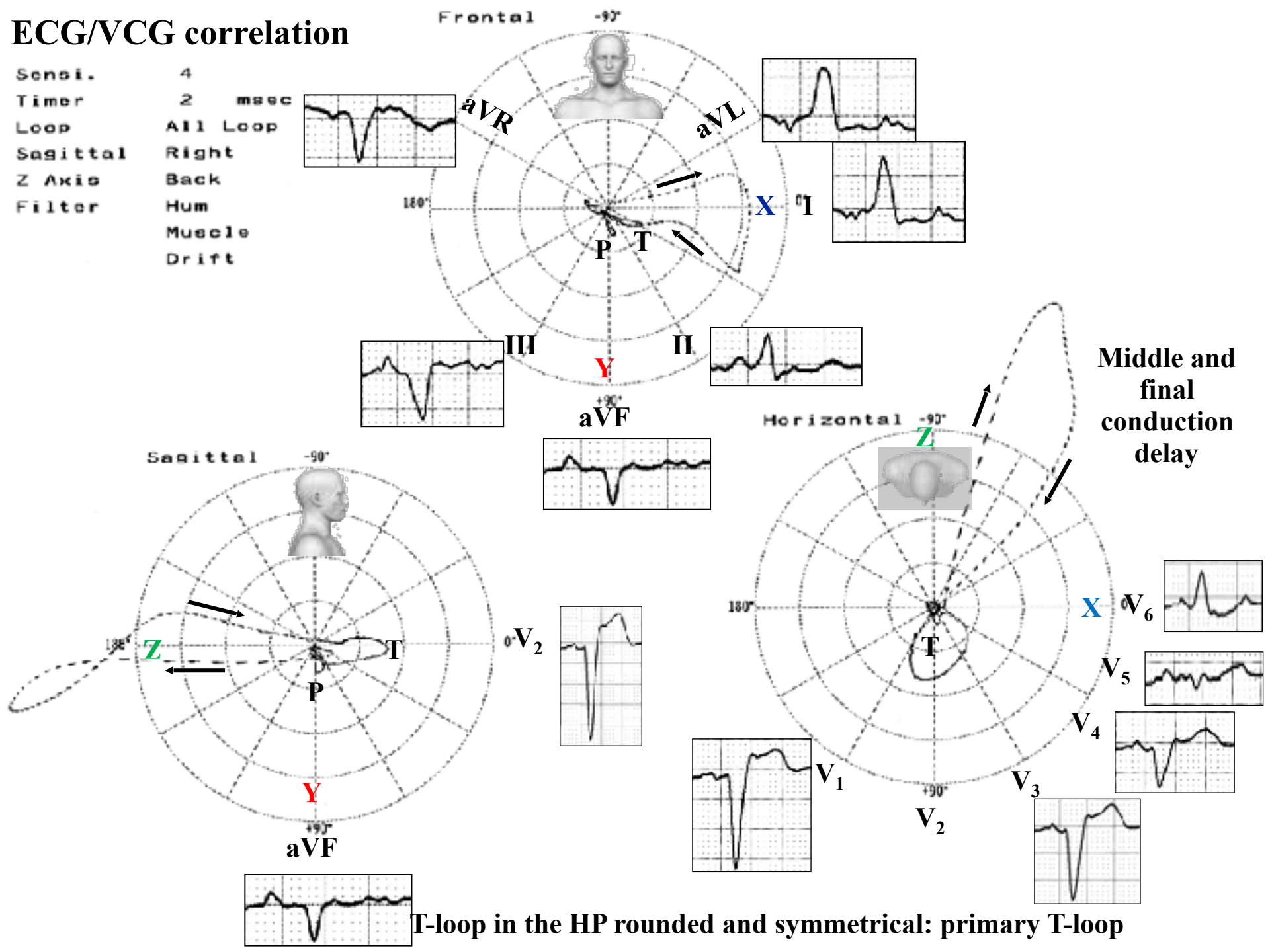
Name: MBP; **Age:** 78 y.o.; **Sex:** F; **Ethnic group:** White; **Weight:** 46 Kg; **Height:** 1.50 m; **Biotype:** Athletic
Med. in use: Enalapril 20 mg 2X, Atenol 50 mg 1X, Hydrochlorothiazide



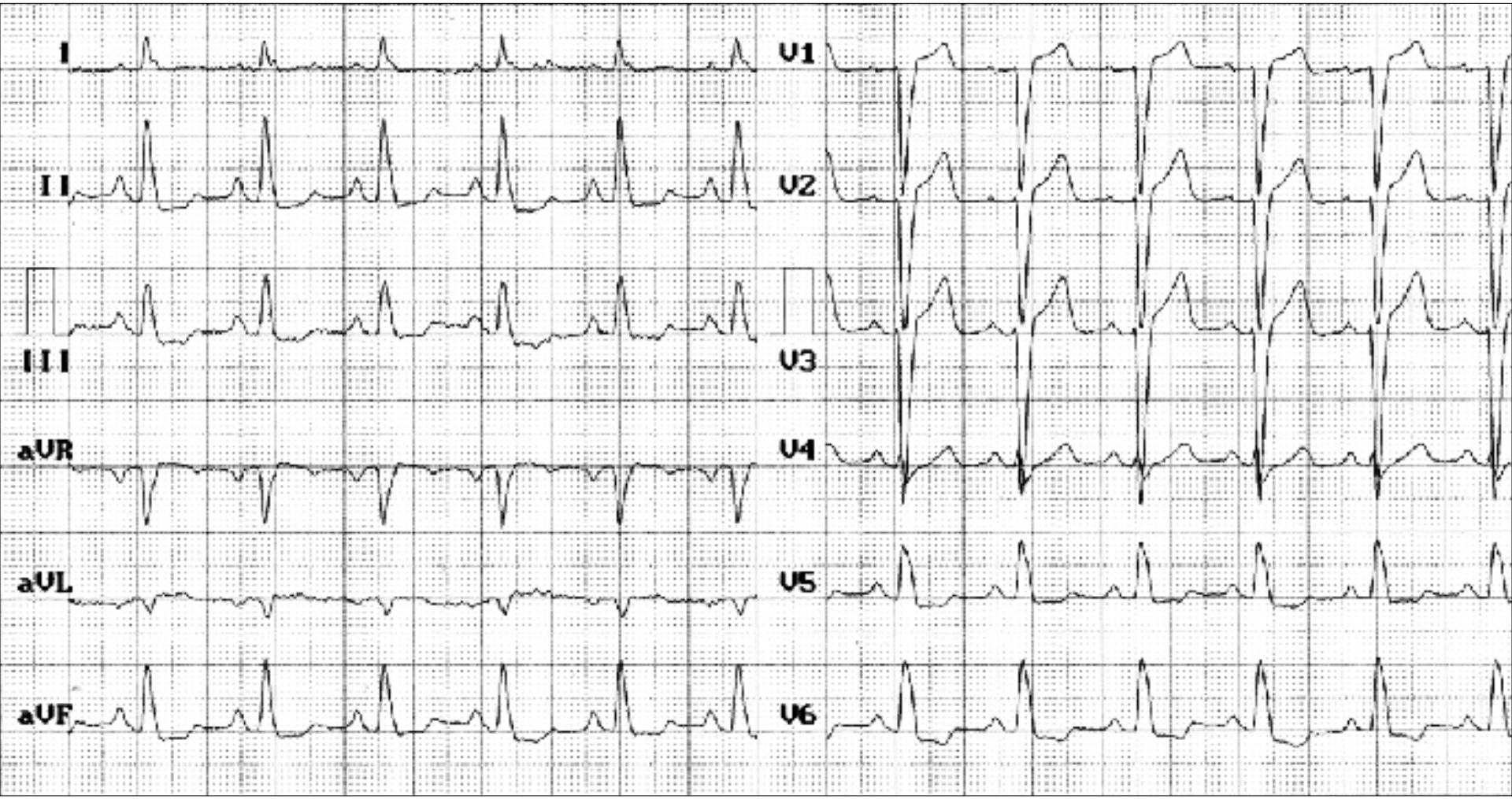
Clinical diagnosis: Hypertensive cardiomyopathy.

ECG diagnosis: Sinus rhythm; HR 59 bpm; PR interval: 120 ms; SÂQRS: -10°; QRS duration: 165 ms; R wave in tower in I, aVL and V6, CLBBB.

ECG/VCG correlation

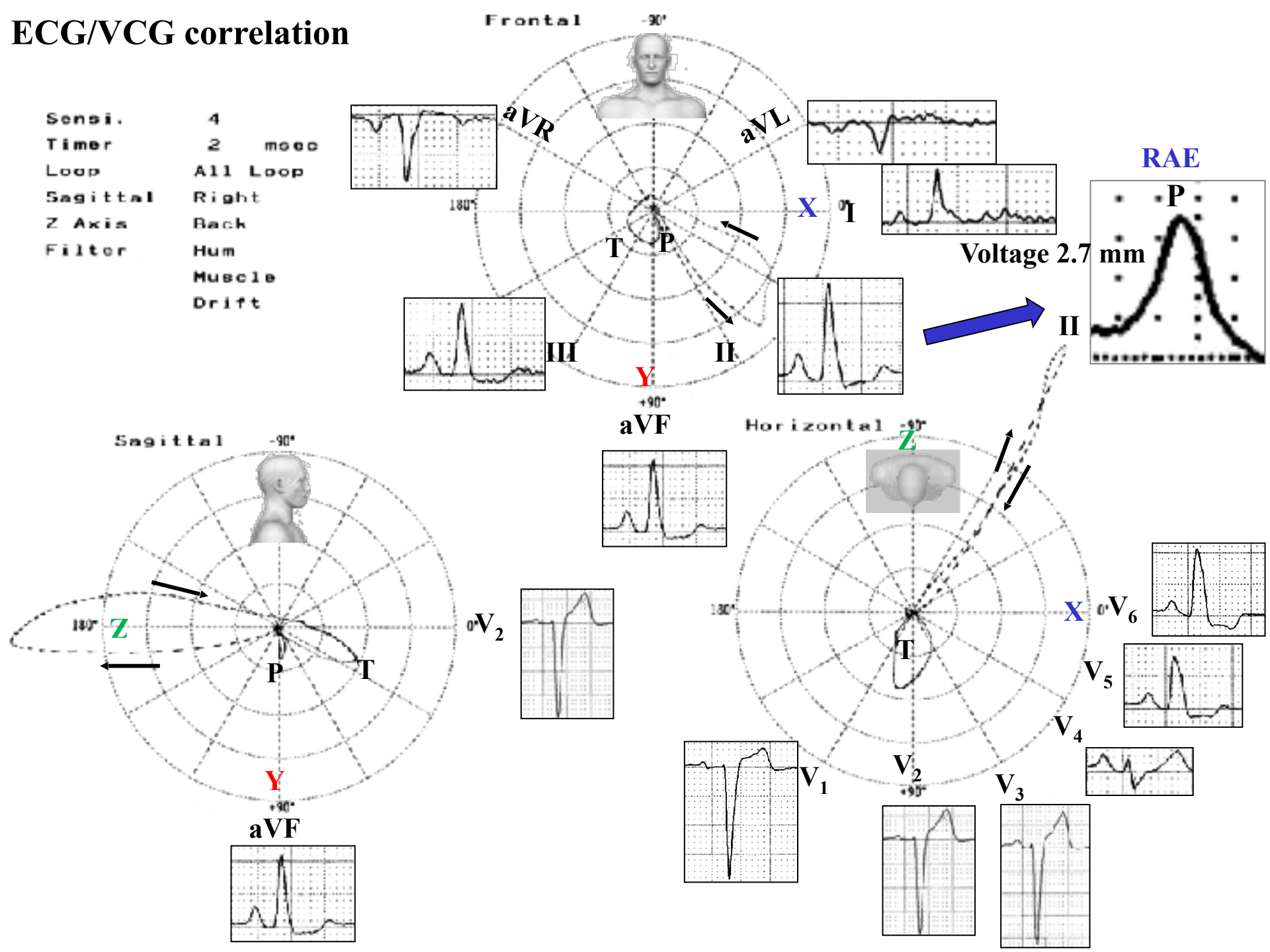


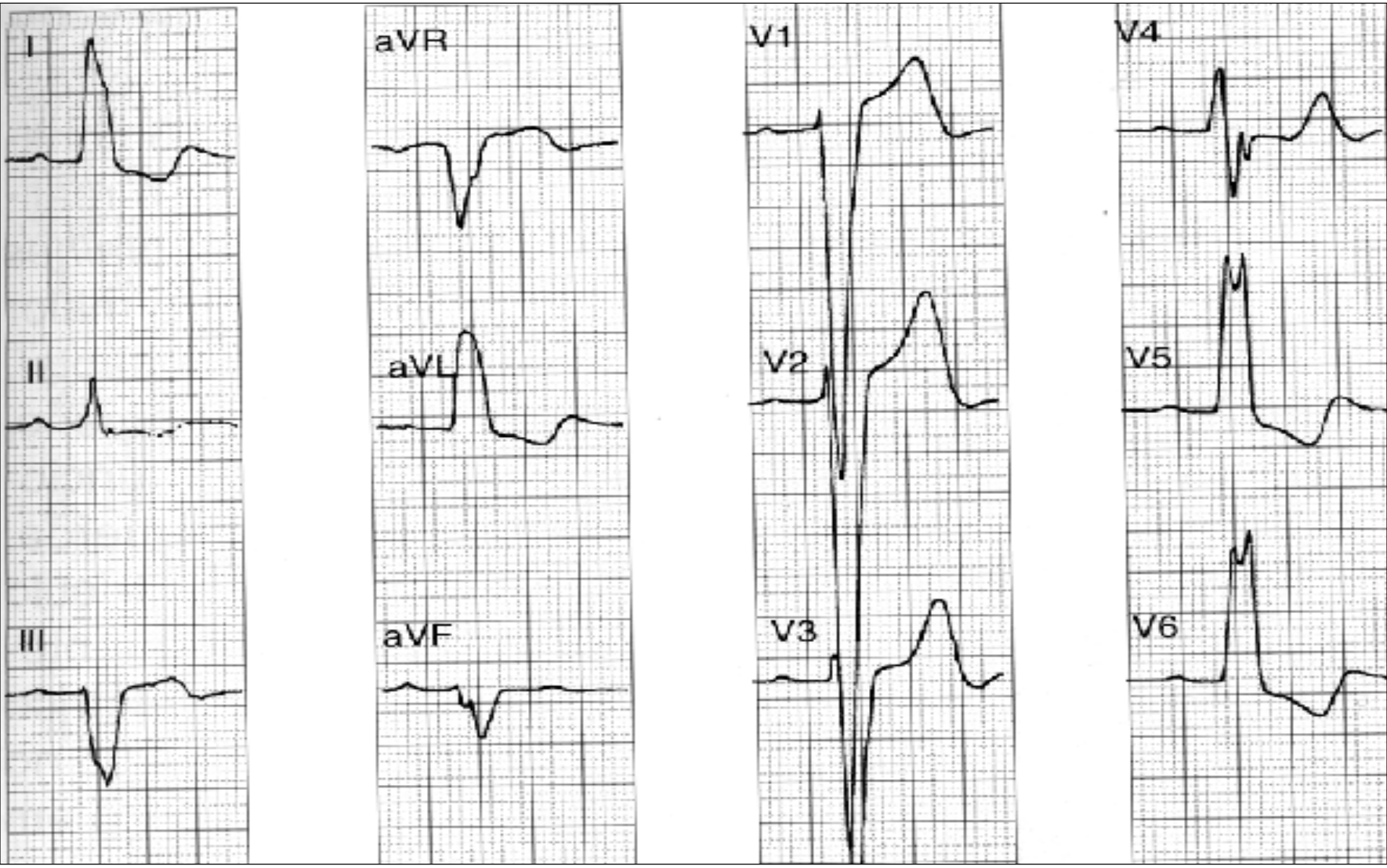
Name: MMNO; **Age: 72 y.o.; Race:** Black; **Sex:** F.; **Date:** 14/03/1997; **Height:** 1.58 m.; **Weight:** 78 Kg.;
Medication in use: Adalat Oros 30 mg, Hygroton 12.5 mg



Clinical diagnosis: Hypertensive heart disease + tricuspid insufficiency.
ECG diagnosis: SR; HR: 87 bpm; P wave: SÂP: + 70°; P wave voltage: 2.7 mm: RAE; duration of 110 ms; PR interval: 180 ms; SÂQRS: + 70 °; QRSD: 130 ms; QRS of V1 and V2 totally or predominantly negative: rS; monophasic R wave of slow registration in left leads V5 and V6. ST-T opposite to greater deflection of QRS: CLBBB.

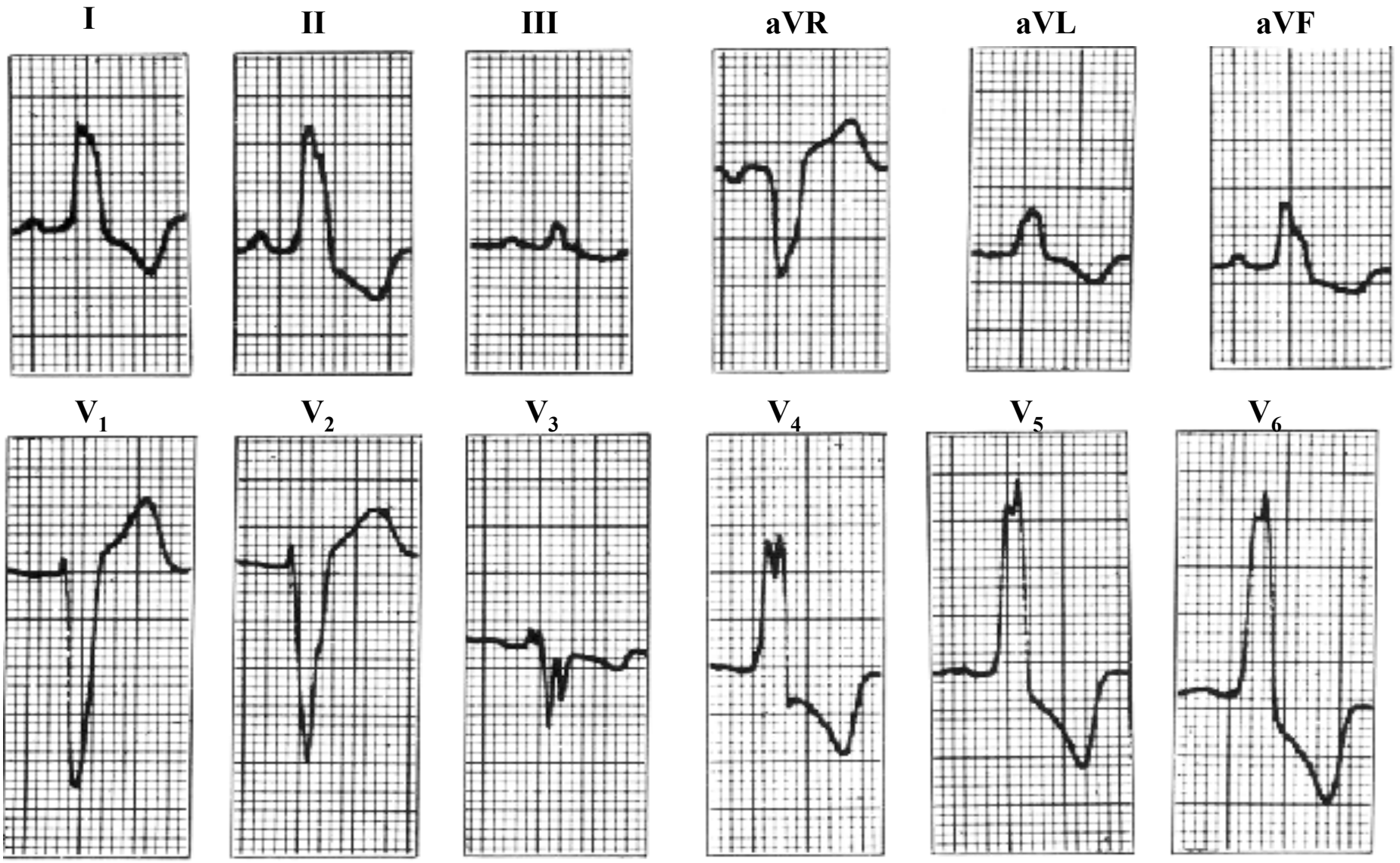
ECG/VCG correlation





Clinical diagnosis: man, 60 years, afro-descendent, with hypertension and coronary heart disease.

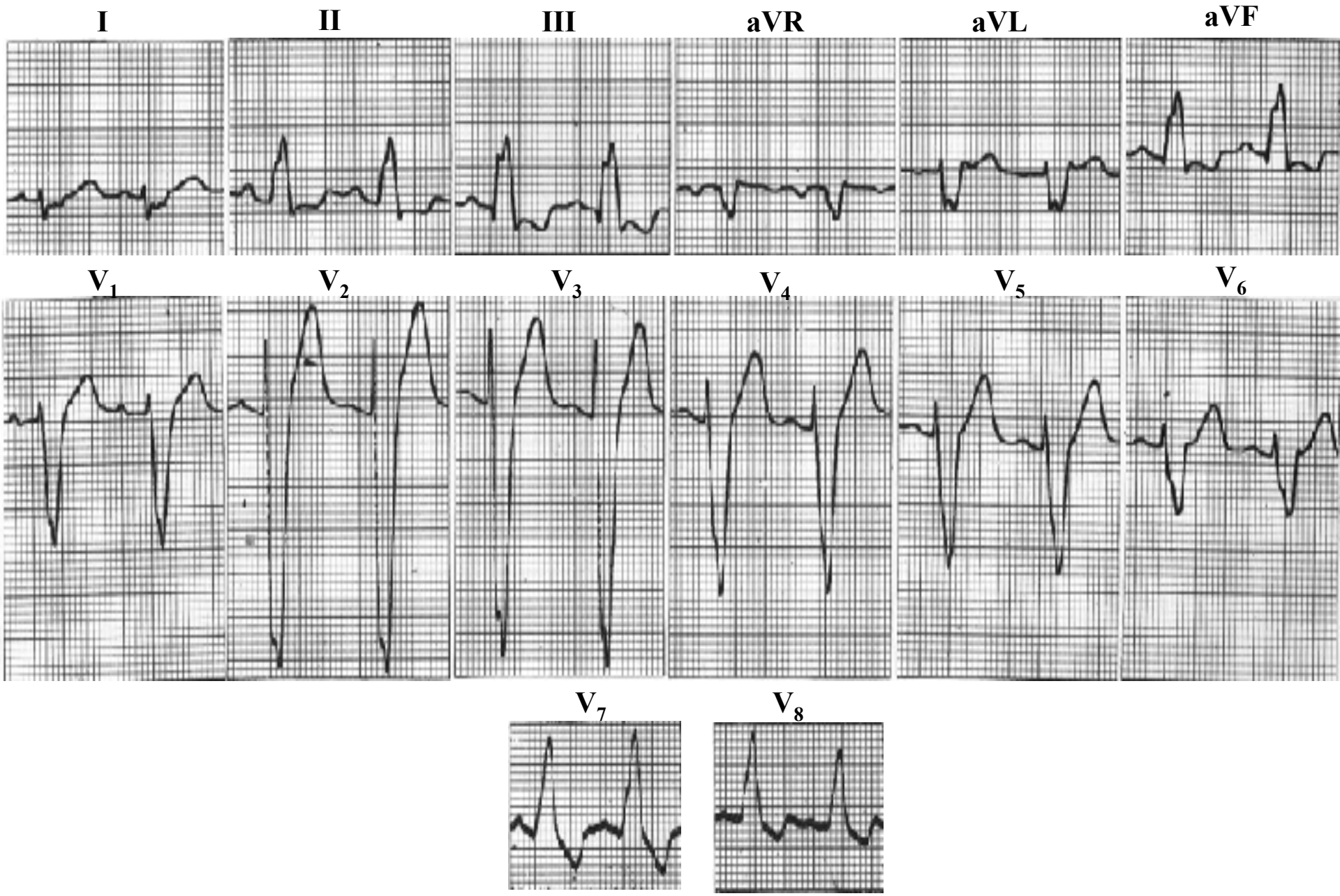
ECG diagnosis: Classical CLBBB, wider QRSD (QRSD = 170 ms) in the presence of supraventricular command; SAQRS –10°. Monophasic R wave in left leads DI, aVL, V_5 and V_6. QRS complexes predominantly negative in V_1 and V_2 type rS, ST/T opposite to QRS.



Clinical diagnosis: 45 y.o. woman with systemic hypertension a long time ago.

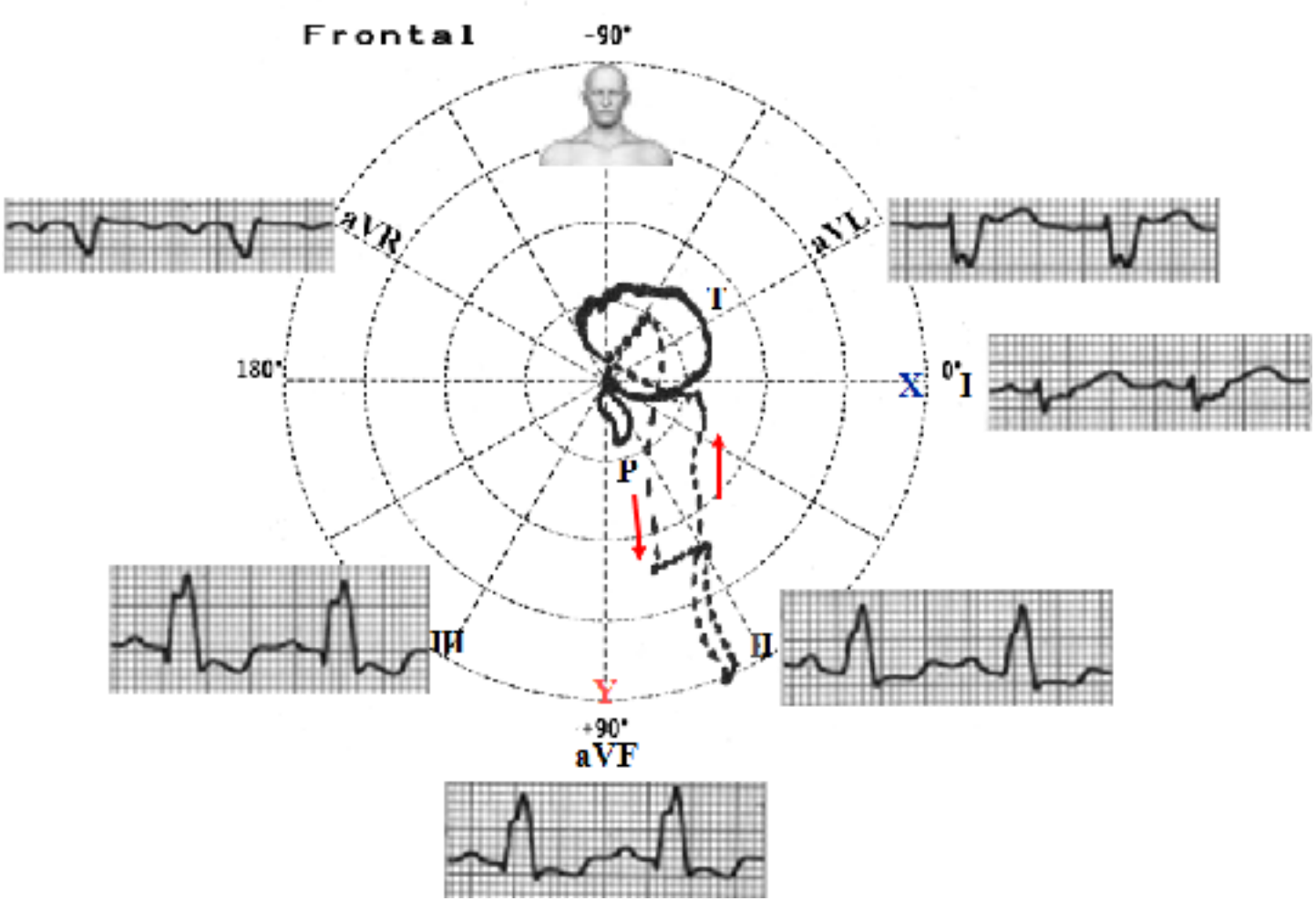
ECG diagnosis: Classical CLBBB, wider QRS (QRSD = 160 ms) in the presence of supraventricular command, SÂQRS +40º, pure R wave in left leads I, aVL, V_5 and V_6, QRS complexes predominantly negative in V_1 and V_2 type rS, ST/T opposite to QRS.

Examples of LBBB with right axis deviation



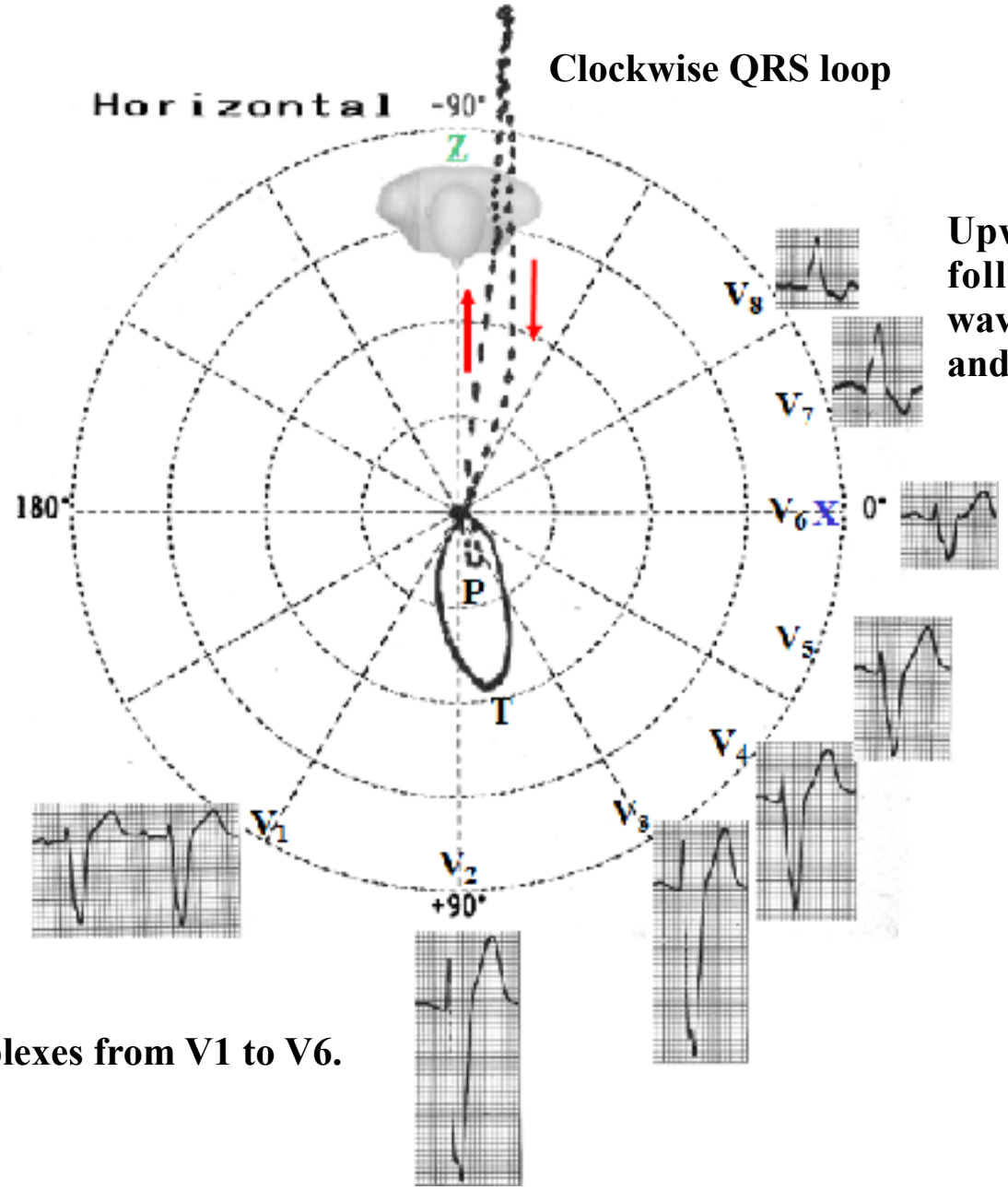
Atypical LBBB because rs in I and rS in aVL and rS from lead V1 through V6. Typical LBBB; upward QRS is observed only in inferior and posterior leads (V7-V8)

ECG/VCG correlation in the FP



Right axis deviation. SÂQRS at +110º.
QRS loop with predominant CCW rotation
with maximal QRS vector +74º.

ECG/VCG correlation in the HP

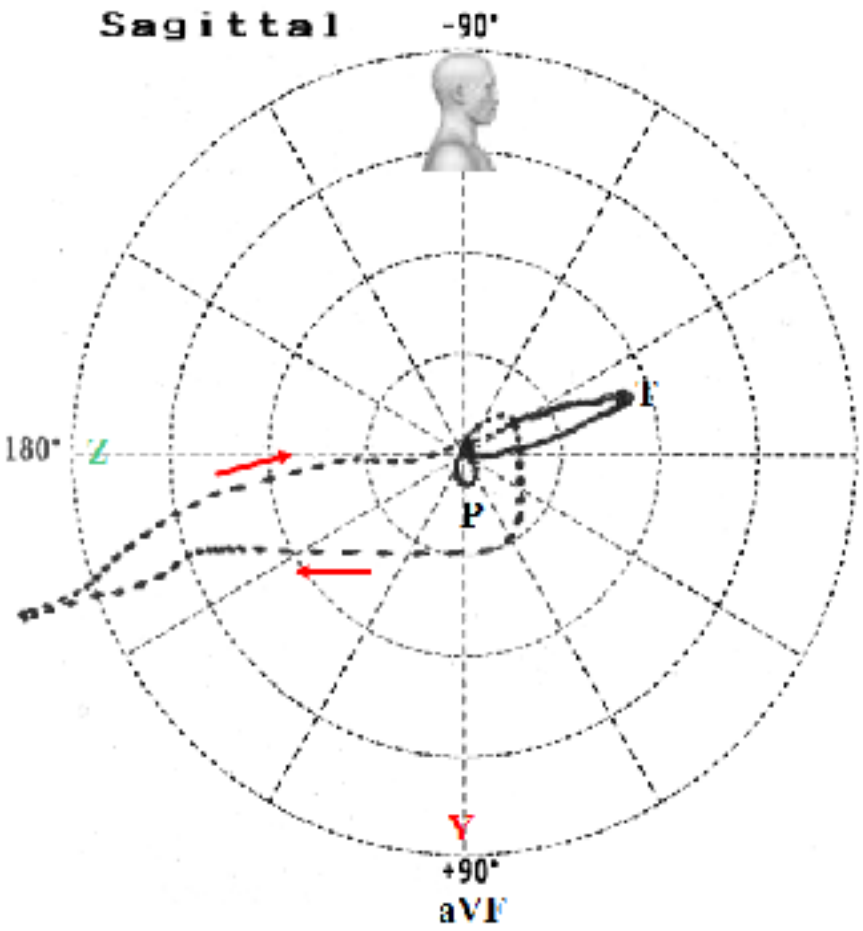


Clockwise QRS loop

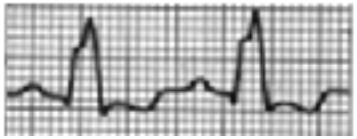
Upward QRS complexes followed by negative T waves in the back (leads V7 and V8).

Negative QRS complexes from V1 to V6.

ECG/VCG correlation in the RSP

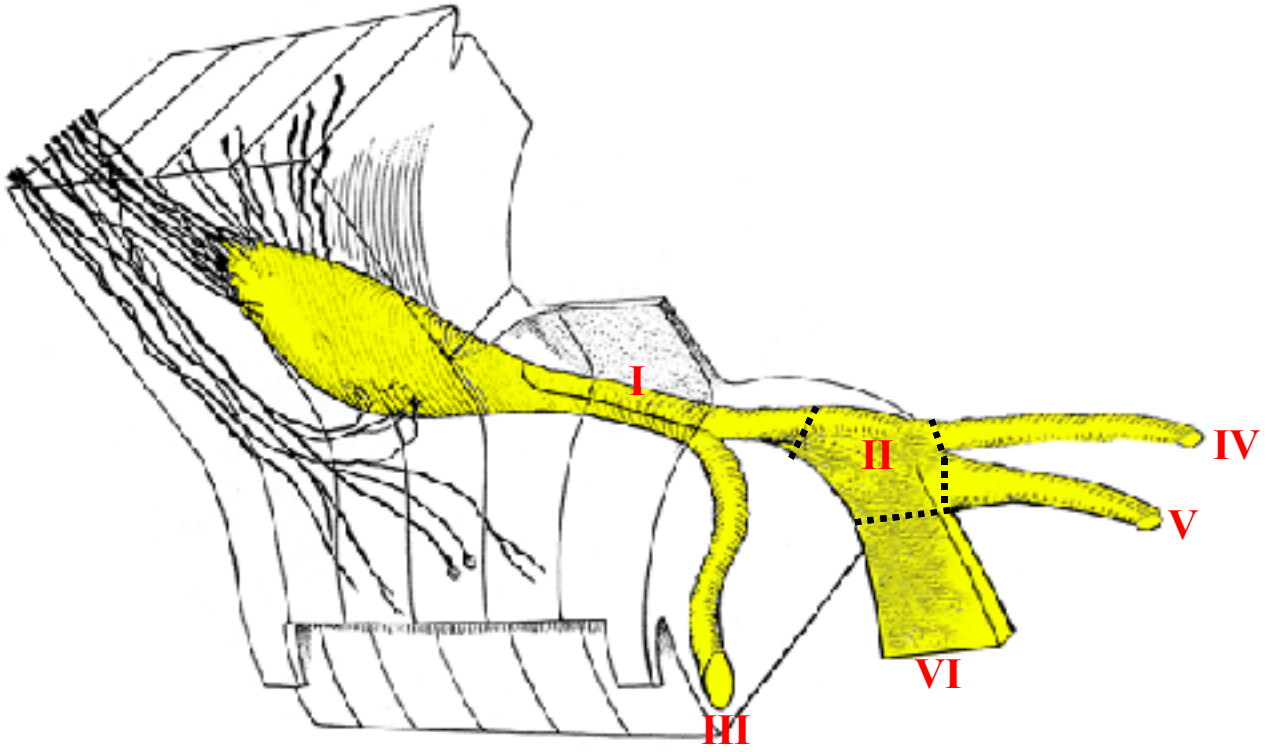


Negative QRS complex in V2 followed by positive T wave



Upward QRS complex in inferior lead aVF: right QRS axis and right P axis

Divisional CLBBB type IV (LAFB + LPFB) by degree of block in LPF greater than the block in LAF



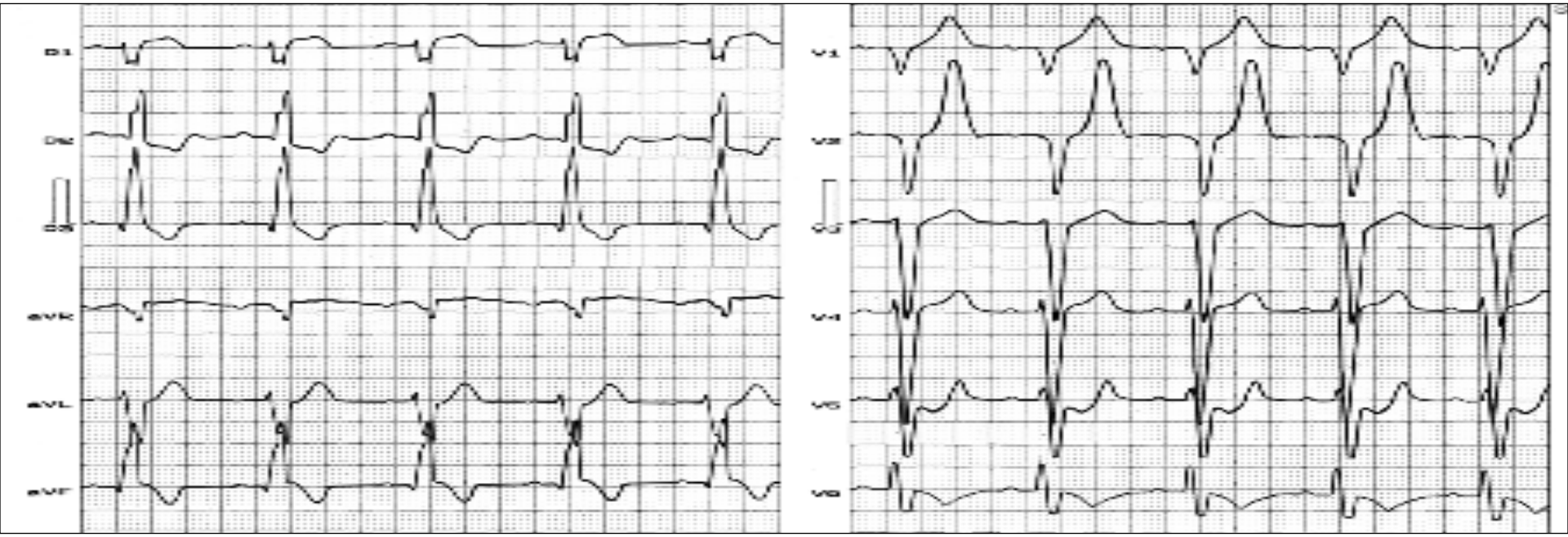
I. Penetrating portion of left His bundle
II. Stem or truncus of LBB

} Blocks in I and II are called predivisional, troncular or membranous LBBBs

III. Right Bundle Branch (RBB)
IV. Left Anterior Fascicle (LAF)
V. Left Posterior Fascicle (LPF)
VI. Left Septal Fascicle (LSF)

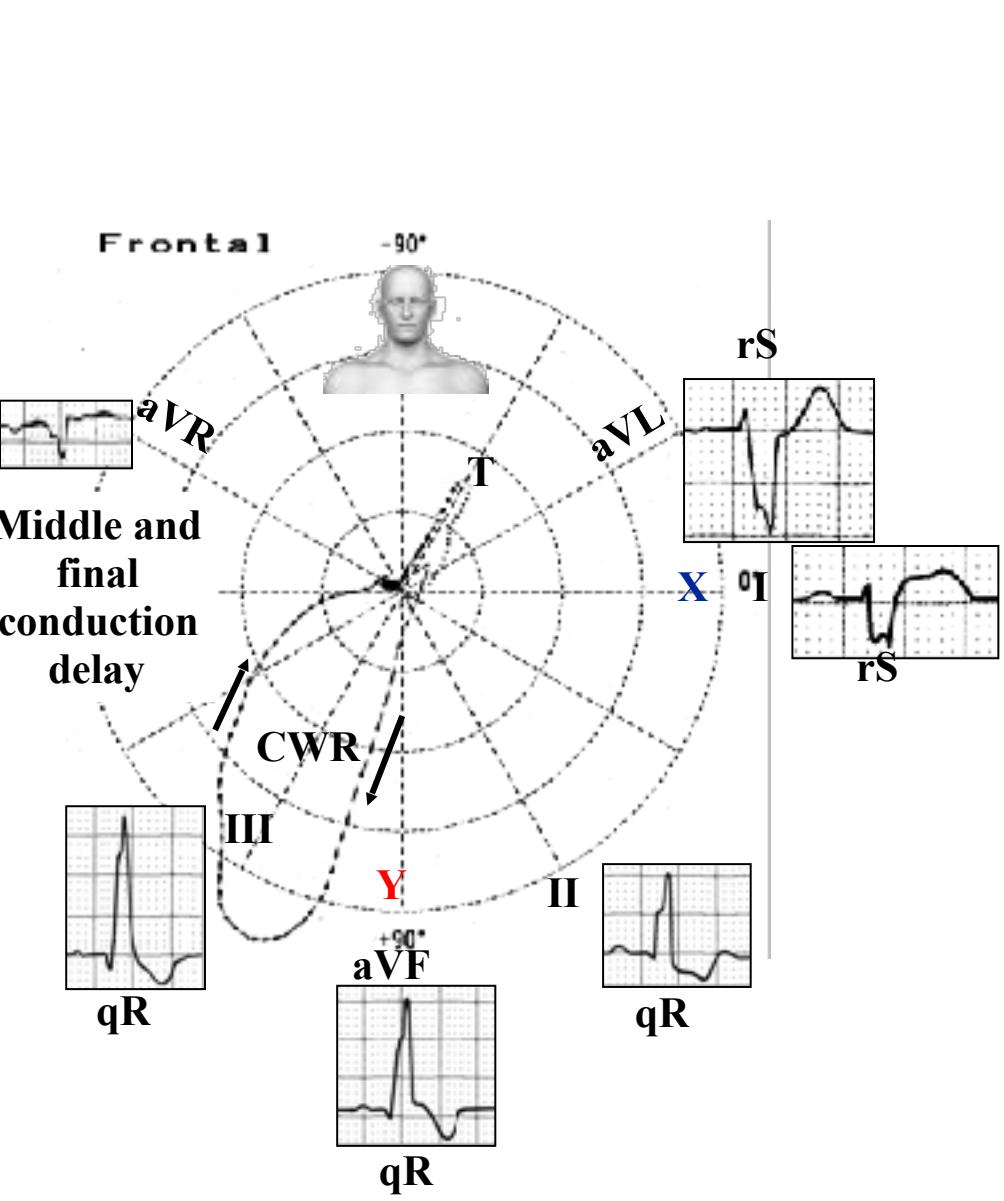
} Blocks in IV+V+VI are called divisional or fascicular Left Bundle Branch Block

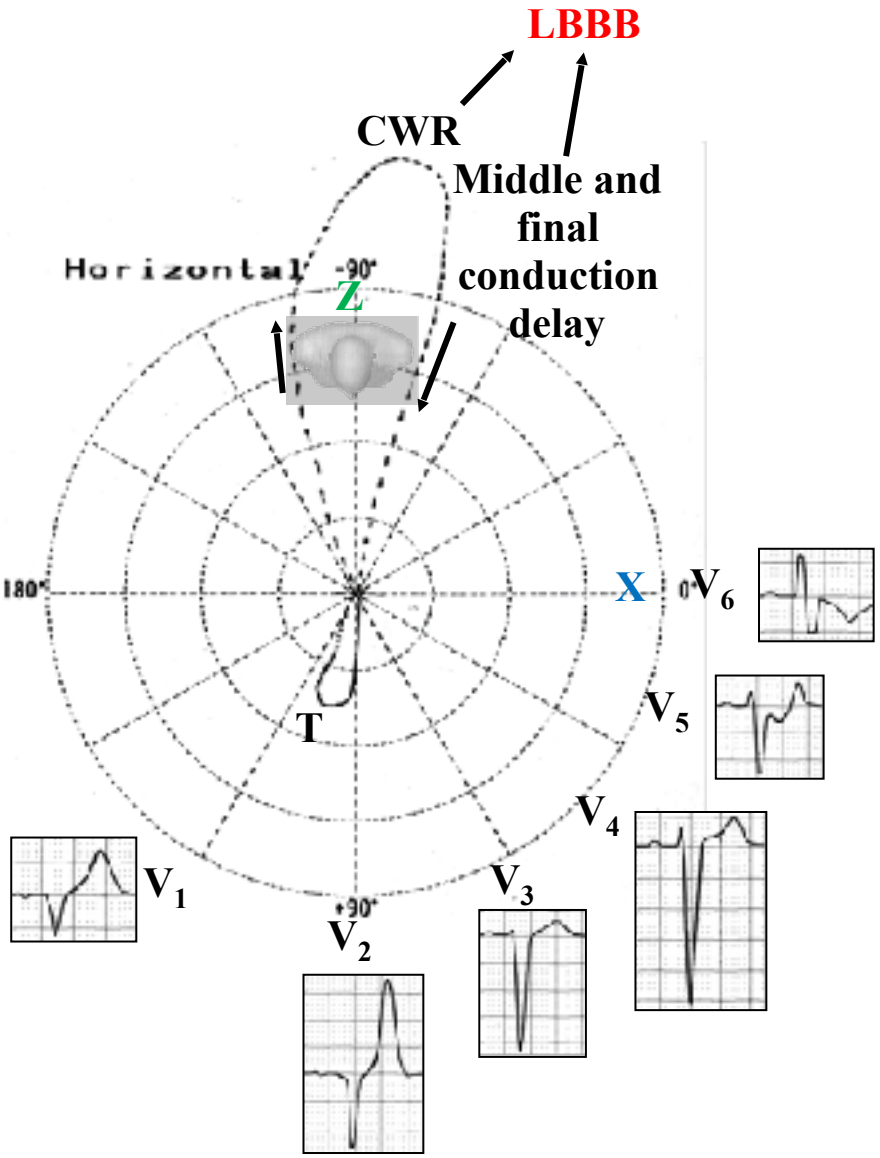
Name: ASC; **Sex:** Male; **Age:** 54 yo.; **Race:** White; **Weight:** 86Kg; **Height:** 1.68 m; **Biotype:** Endomorph;
Date: 04/03/2003; **Medication in use:** Enalapril 10 mg 2X + Atenolol 50 mg + Chlortalidone 12.5 mg.



Clinical diagnosis: Hypertensive heart disease + aortic insufficiency by aortic cause.
Echo diagnosis: Moderate concentric hypertrophy: septum 13 mm and posterior wall 14 mm. Moderate aortic insufficiency.
ECG diagnosis: SR; HR: 72 bpm; SAP: $+60^0$; SAQRS: $+110^0$; QRSD: 165 ms; I and aVL = rS; DIII = qR; RIII > RII. Which is the electrocardiographic foundation for LPFB diagnosis? SÂQRS deviated to the right in clinical absence of RVH, vertical heart or lateral infarction; QRS complexes of the rS type in I and aVL; complexes of the qR type in inferior leads with R wave of III > than R wave of II. There are references in literature to aortic insufficiency by regurgitant jet, which thrown on the posteroinferior wall may cause LPFB. On the other hand, the CLBBB has as its most frequent cause hypertension. An accurate diagnosis of LPFB must obligatorily be clinical and electrocardiographic, as in this case, in which in an obese, endomorph, hypertensive patient, the SAQRS is in $+115^o$.
Conclusion: 1) CLBBB; 2) LPFB (Left Posterior Fascicular Block).

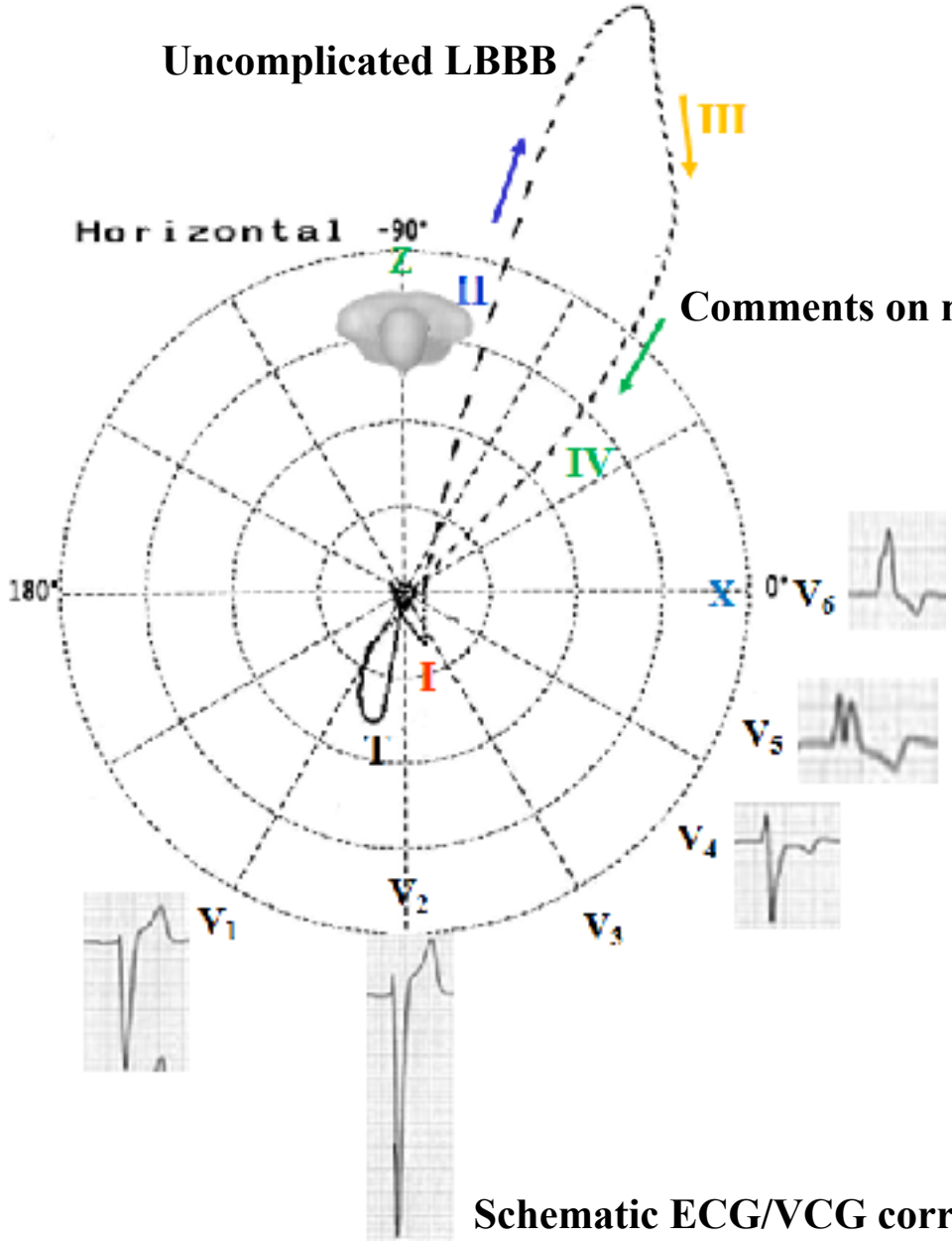
ECG/VCG correlation in the Frontal and Horizontal Planes





SÂQRS +110° + RIII > RII + rS I and aVL = LPFB

ECG / VCG difference between LBBB and LBBB associated to RVH in the HP



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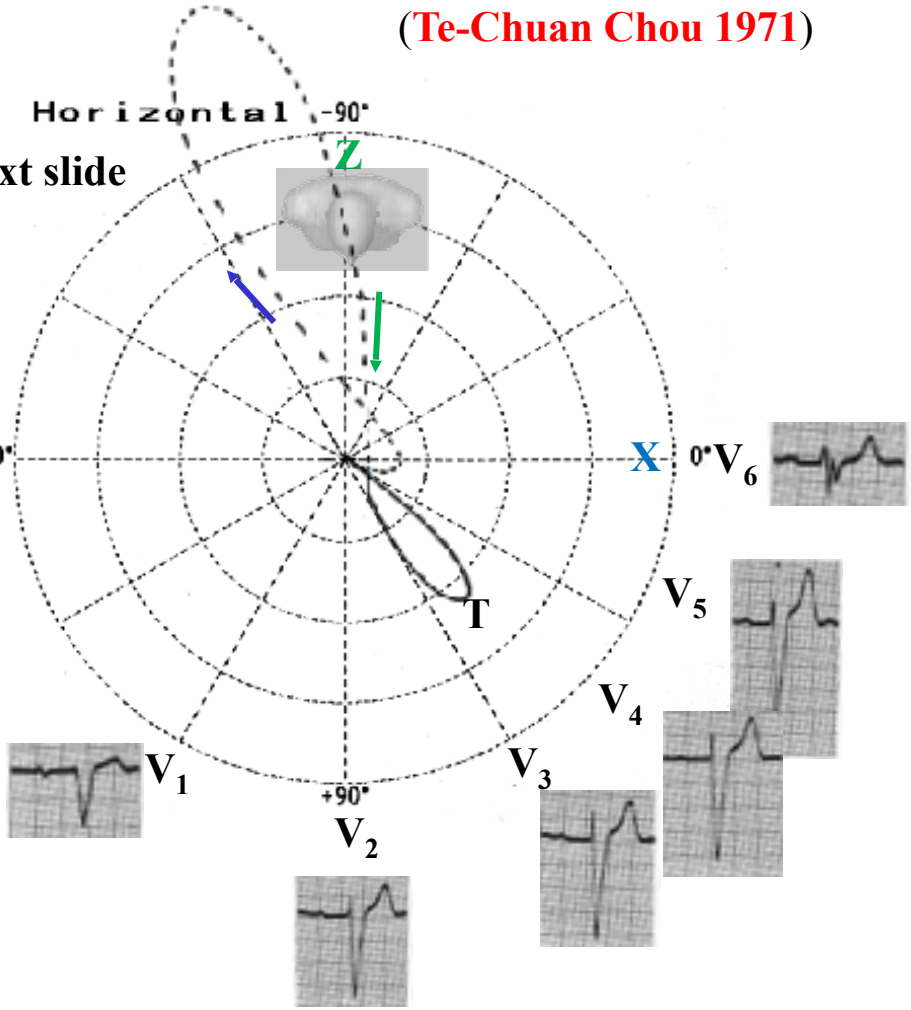

Schematic ECG/VCG correlation comparing the HP of VCG of typical LBBB with LBBB complicated by RVH

VCG characterization of right ventricular hypertrophy in the presence of LBBB

The VCG characteristics are:

1. QRS loop duration with prolongation;
2. Slow inscription of the mid and late portion of the QRS loop;
3. Leftward and inferior orientation of the initial QRS vectors;
4. Posterior and rightward displacement of the maximum QRS vector;
5. Clockwise inscription of the major portion of the QRS loop in the HP;
6. Anterior and leftward orientation of the ST vector and T-loop.

Final comments:

The changes in the HP VCG differed from the typical LBBB pattern only in the rightward displacement of the QRS loop and leftward orientation of the ST vector and T-loop.

	Isolated LBBB	LBBB + RVH
HP QRS loop	Leftward displacement	Rightward displacement
ST vector and T-loop	Righward orientation	Leftward orientation
ECG lead I	Monophasic R wave	Presence of S wave
QRS axis	From -30º to +60º (≈ 65% to 70% of cases) From –30º to -90º (≈25% of cases)	Beyond +90º (< than 1% of cases)

References:

1. Al-Hadi H, Sallam M. Asymptomatic Permanent Left Bundle Branch Block (LBBB) complicating Diagnostic Left Heart Catheterisation. Sultan Qaboos Univ Med J. 2010 Apr;10(1):114-9
2. Arias MA, Sánchez AM, López JM. Repetitive intermittent left bundle branch block. Pacing Clin Electrophysiol. 2006 Nov;29(11):1306-9.
3. Bayés de Luna. Basic Electrocardiography. Normal and abnormal ECG Patterns. Blackwell Publishing. Ventricular Blocks pp 57 2007.
4. Bellavia D, Pellikka PA, Abraham TP, et al. 'Hypersynchronisation' by tissue velocity imaging in patients with cardiac amyloidosis. Heart. 2009 Mar;95(3):234-40.
5. Bharati S, Lev M, Dhingra RC, et al. Electrophysiologic and pathologic correlations in two cases of chronic second degree atrioventricular block with left bundle branch block. Circulation. 1975 Aug;52(2): 221-9.
6. Breithardt G, Breithardt OA. Left bundle branch block, an old-new entity. J Cardiovasc Transl Res. 2012 Apr;5(2):107-16.
7. Brenyo A, Rao M, Barsheshet A, et al.QRS axis and the benefit of cardiac resynchronization therapy in patients with mildly symptomatic heart failure enrolled in MADIT-CRT. J Cardiovasc Electrophysiol. 2013 Apr;24(4):442-8.
8. Brembilla-Perrot B, Alla F, Suty-Selton C, et al. Nonischemic dilated cardiomyopathy: results of noninvasive and invasive evaluation in 310 patients and clinical significance of bundle branch block. Pacing Clin Electrophysiol. 2008 Nov;31(11):1383-90.
9. Bruntz JF, Perrot B, Medeiros C, et al. [Vectorcardiography in inferior infarction associated with left bundle-branch block]. Arch Mal Coeur Vaiss. 1985 Feb;78(2):233-9.
10. Cabrera E, Costa Rocha J, Flores G. [The vectorcardiogram of myocardial infarcts with disorder of intraventricular conduction]. Arch Inst Cardiol Mex. 1959 Nov-Dec;29:625-46.
11. Chan DD, Wu KC, Loring Z, et al. Comparison of the relation between left ventricular anatomy and QRS duration in patients with cardiomyopathy with versus without left bundle branch block. Am J Cardiol. 2014 May 15;113(10):1717-22.

12. Childers R, Lupovich S, Sochanski M, et al. Left bundle branch block and right axis deviation: a report of 36 cases. J Electrocardiol. 2000;33 Suppl:93-102.
13. Doucet, P, Walsh, TJ, Massie, E: Vectorcardiographic and electrocardiographic study of left bundle branch block with myocardial infarction. Am J Cardiol 17: 171, 1966.
14. Eriksson P, Andersen K, Swedberg K, et al. Vectorcardiographic monitoring of patients with acute myocardial infarction and chronic bundle branch block. Eur Heart J. 1997 Aug;18(8):1288-95.
15. Galeotti L, van Dam PM, Loring Z, et al. Evaluating strict and conventional left bundle branch block criteria using electrocardiographic simulations. Europace. 2013 Dec;15(12):1816-21.
16. Goldman MJ, Pipberger HV. Analysis of the orthogonal electorocardiogram and vectorcardiogram in ventricular conduction defects with and without myocardial infarction. Circulation. 1969 Feb;39(2): 243-50.
17. Gunnarsson G, Eriksson P, Dellborg M. ECG criteria in diagnosis of acute myocardial infarction in the presence of left bundle branch block. Int J Cardiol. 2001 Apr;78(2):167-74.
18. Khurana C, Mazzone P, Mandell B.New onset left bundle branch block with right axis deviation in a patient with Wegener's granulomatosis. J Electrocardiol. 2000 Apr;33:199-201.
19. Kyndt F, Probst V, Potet F, et al. Novel SCN5A mutation leading either to isolated cardiac conduction defect or Brugada syndrome in a large French family. Circulation. 2001 Dec 18;104(25):3081-6.
20. Lepeschkin E. Modern Electrocardiopgraphy: vol. 1. The P-Q-R-S-T-U complex (Williams & Wilkins, Baltimore, 1951).
21. Liakopoulos V, Kellerth T, Christensen K. Left bundle branch block and suspected myocardial infarction: does chronicity of the branch block matter? Eur Heart J Acute Cardiovasc Care. 2013 Jun;2(2):182-9.
22. Manohar N, Young ML. Rate dependent bundle branch block induced by hyperkalemia. Pacing Clin Electrophysiol. 2003 Sep;26(9):1909-10.
23. Medrano GA, De Micheli A, Bisteni A, et al. [Experimental study of the electric manifestations of the septal infarct associated with a right bundle-branch block in the light of the process of ventricular activation]. Arch Inst Cardiol Mex. 1964 Mar-Apr;34:151-73.

24. Medrano GA, Brenes C, De Micheli A, et al. [Simultaneous block of the anterior and posterior subdivisions of the left branch of the bundle of His (biphasic block), and its association with the right branch block (triphasic block). Experimental and clinical electrocardiographic study]. Arch Inst Cardiol Mex. 1970 Nov-Dec;40(6):752-70.
25. Nikolic G, Marriott HJ. Left bundle branch block with right axis deviation: a marker of congestive cardiomyopathy. J Electrocardiol. 1985 Oct;18(4):395-404.
26. Parharidis G, Nouskas J, Efthimiadis G, et al. Complete left bundle branch block with left QRS axis deviation: defining its clinical importance. Acta Cardiol. 1997;52(3):295-303.
27. Patanè S, Marte F, Di Bella G. Atrial fibrillation with left bundle branch block and intermittent right axis deviation during acute myocardial infarction. Int J Cardiol. 2008 Jun 23;127(1):e1-2. Epub 2007 Apr 5.
28. Patanè S, Marte F, Dattilo G, et al. Acute myocardial infarction and left bundle branch block with changing axis deviation. Int J Cardiol. 2012 Feb 9;154(3):e47-9.
29. Perrin MJ, Green MS, Redpath CJ, et al. Greater response to cardiac resynchronization therapy in patients with true complete left bundle branch block: a PREDICT substudy. Europace. 2012 May;14(5):690-5.
30. Poels TT, Houthuizen P, Van Garsse LA, Maessen JG, de Jaegere P, Prinzen FW. Transcatheter aortic valve implantation-induced left bundle branch block: causes and consequences. J Cardiovasc Transl Res. 2014 Jun;7(4):395-405.
31. Préda I. Results of randomized studies on cardiac resynchronization therapy and the reevaluation of cardiac ventricular activation in left bundle branch block. Orv Hetil. 2013 May 5;154(18):688-93.
32. Riera AR, de Cano SJ, Cano MN, Gimenez VM, de Padua Fleury Neto LA, Sousa JE. Vector electrocardiographic alterations after percutaneous septal ablation in obstructive hypertrophic cardiomyopathy. Possible anatomic causes. Arq Bras Cardiol. 2002 Nov;79(5):466-75.
33. Rodríguez-Padial L, Rodríguez-Picón B, Jerez-Valero M, et al. Diagnostic accuracy of computer-assisted electrocardiography in the diagnosis of left ventricular hypertrophy inleft bundle branch block. Rev Esp Cardiol (Engl Ed). 2012 Jan;65(1):38-46.

34. Rowinsky EK, McGuire WP, Guarnieri T, et al. Cardiac disturbances during the administration of taxol.J Clin Oncol. 1991 Sep;9(9):1704-12.
35. Salazar J, Lej FA.Electrocardiographic changes following surgical repair of ostium primum defect.Acta Cardiol. 1978; 33: 55-61.
36. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med.1996 Feb 22;334(8):481-7.
37. Shvilkin A, Bojovic B, Vajdic B, et al. . Vectorcardiographic and electrocardiographic criteria to distinguish new and old left bundle branch block. Heart Rhythm 2010 Aug;7(8):1085-92
38. Silva JA, Khuri B, Barbee W, et al. Systolic excursion of the mitral annulus to assess septal function in paradoxic septal motion. Am Heart J. 1996 Jan;131(1):138-45
39. Sodi D, Bisteni A, Medrano G. Electrocardiografia y vectorcardiografia deductivas. Vol 1. Mexico, DF: La Prensa Médica Mexicana, 1964.
40. Sodi-Pallares, D; Medrano, GA; Bisteni, A; et al. Deductive and polyparametric electrocardiography (México: Instituto Nacional de Cardiología de México, 1970)
41. Strauss DG, Olson CW, Wu KC, et al. Vectorcardiogram synthesized from the 12-lead electrocardiogram to image ischemia. J Electrocardiol. 2009 Mar-Apr;42(2):190-7.
42. Strauss DG, Selvester RH, Wagner GS. Defining left bundle branch block in the era of cardiac resynchronization therapy. Am J Cardiol. 2011 Mar 15;107:927-934.
43. Te-Chuan Chou, Helm RA. The diagnosis of Right Ventricular Hypertrophy in the presence of Left Bundle Branch Block in Proc. Xith International Vectorcardiography Symposium – North Holland Publishing Company 1971. Pp. 289-296.
44. Tian Y, Zhang P, Li X, et al. True complete left bundle branch block morphology strongly predicts good response to cardiac resynchronization therapy. Europace. 2013 Oct;15(10):1499-506.

ECG & VCG in Right Bundle Branch Block

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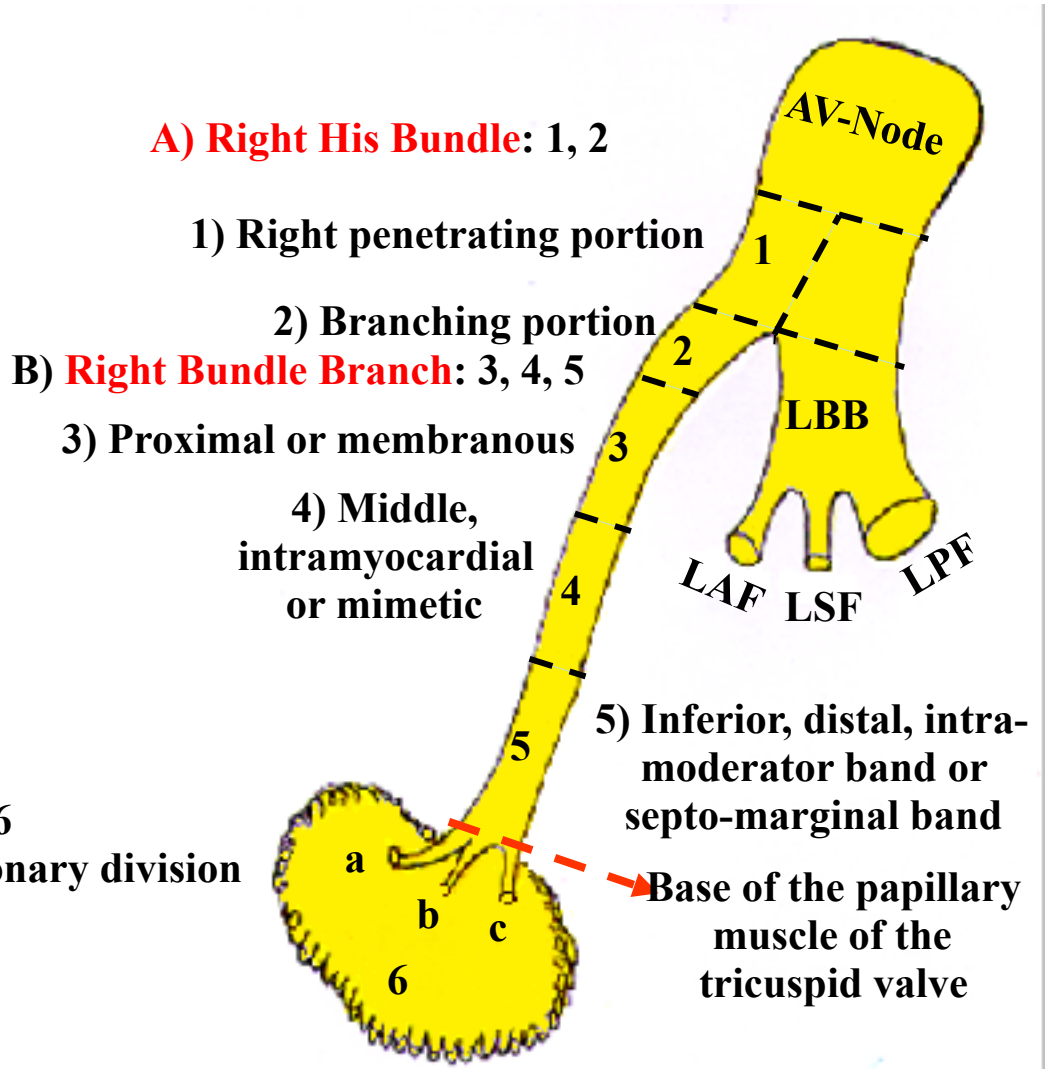
The author does not report any conflict of interest regarding this presentation

Dromotropic disorders of the Right His System or Right Bundle Branch Block (RBBB)

Other denominations: Right His System Block (RHSB); Global Right Ventricular Blocks (GRVB).

Concept: RBBB, RHSB or RGVB is any delay in activation of the Right Ventricle (RV) as a consequence of a dromotropic disorder located in any point of the Right His System (RHS), which causes the biventricular chamber to depolarize sequentially and no longer simultaneously; a fact that necessarily extends ventricular depolarization time (QRS duration prolongation). The anomalous sequence in depolarization is responsible for the alteration secondary to the ventricular repolarization that conditions ST/T to be opposite to the last slow deflection of ventricular depolarization.

The Right His System (RHS) is constituted by three portions:



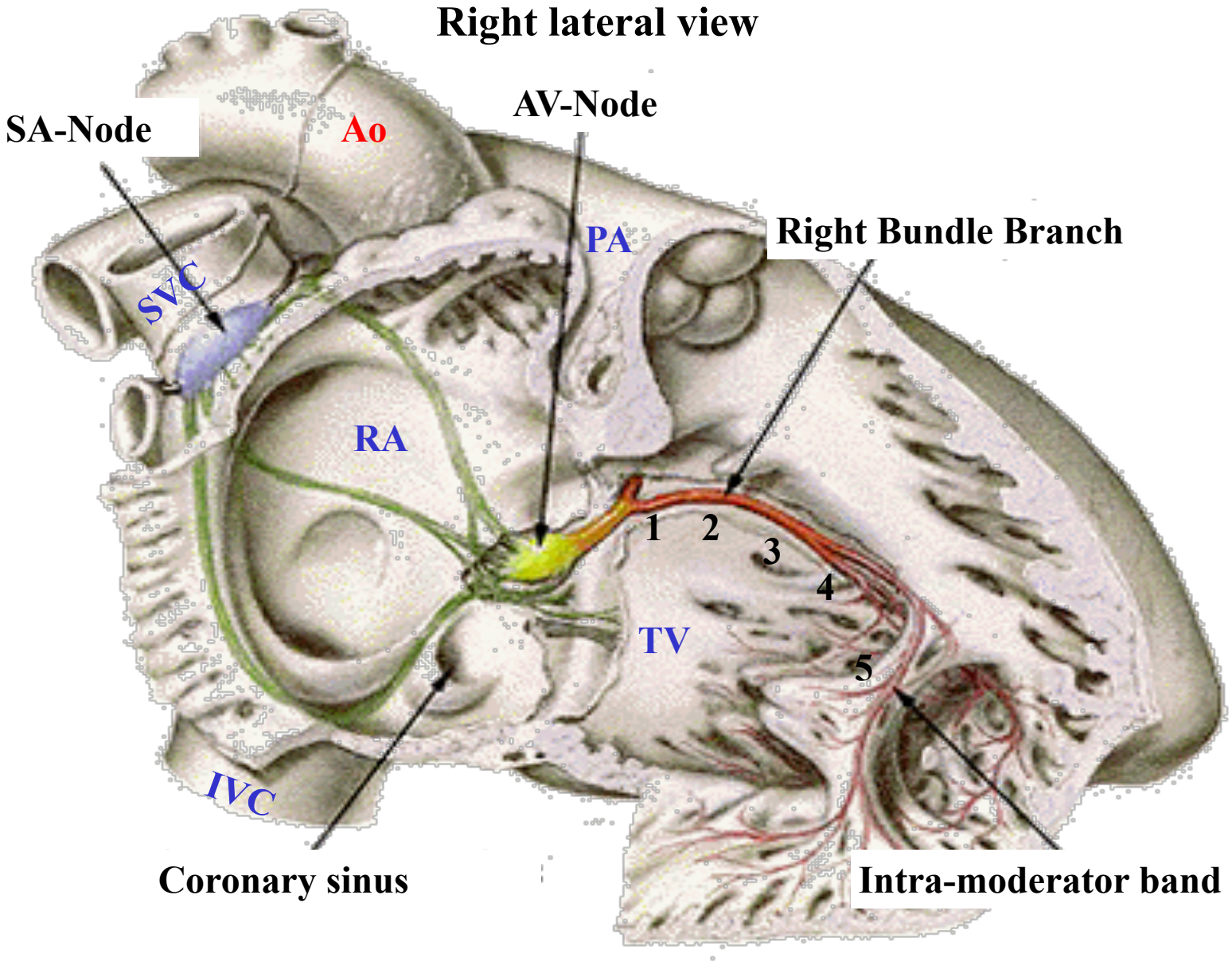
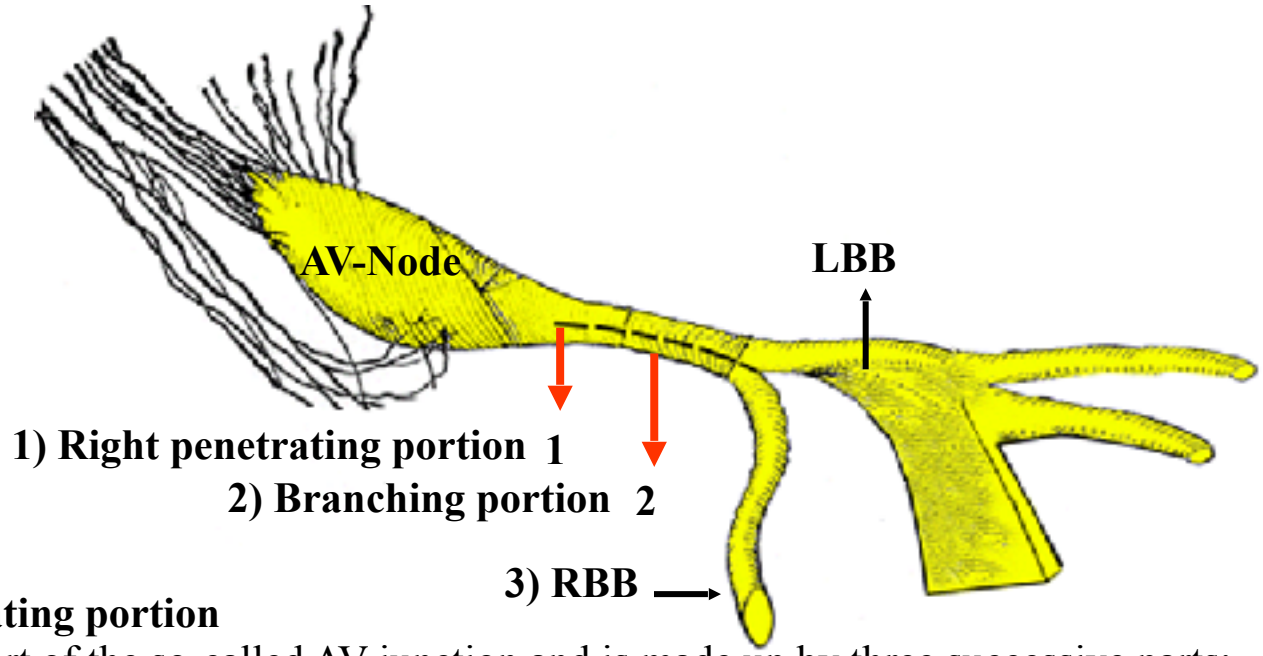


Image from An Atlas and Practical Guide to Histology, by Dr. Kenneth Chan with technical assistance from Dr. Simon Cool & Mr. Duncan McCardle. The University of Queensland, Department of Anatomical Sciences © 1998

Analysis of the two portions of the Right His System (RHS)



1) Right penetrating portion

This portion is part of the so-called AV junction and is made up by three successive parts:

A) Area of transition cells: this area is the site where the RA gets in touch with the compact portion of the AV node through slow and fast bundles;

B) Compact portion of AV node proper: This is found in an anterior and superior position regarding the coronary sinus, above the insertion of the septal fascicle of the tricuspid valve. This area is located in the apex of the so-called triangle of Koch, formed by the tricuspid ring, Todaro's tendon and the ostium of the coronary sinus. Blood supply of the compact portion of the AV node is ensured in 85% of the cases, by branches of the right coronary artery, and in the remaining 15% by the circumflex artery branches.

C) Right initial penetrating portion of the His bundle: The name of the penetrating portion is due to the fact of its going through the fibrous trigone. It stretches from the inferior limit of the AV node, up to the origin of the first fibers that make up the Posterior Fascicle of the Left Branch (LPF). Its extension is from 50 mm to 100 mm. Histological studies have revealed that the right half carries the group of fibers to the homolateral ventricle (right).

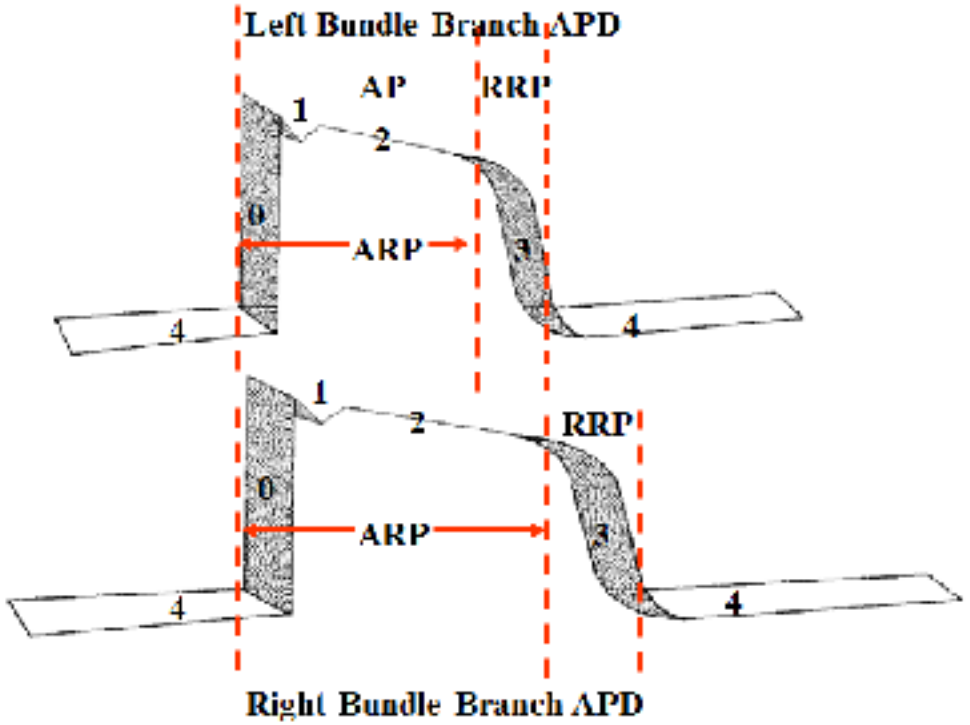
2) Right branching portion of the His bundle

It stretches from the origin of the LPF of the LBB until the origin of the right branch (RBB) and the left anterior fascicle (LAF) of the LBB. This portion is closely related with the interventricular membranous septum and with the non-coronary and right coronary cuspids of the aortic valve. Its lesion causes Complete Right Bundle Branch Block (CRBBB) almost constantly, in association with Left Anterior Fascicular Block (LAFB) by the close neighboring relationship between the right branch and the Left Anterior Fascicle (LAF).

3) Right Bundle Branch

I. Anatomical characteristics: Length: 45 to 60 mm; Diameter: 1.5 to 2 mm; Color: whitish.
II. Cellular type: Purkinje cells;
III. Conduction velocity: 5m/sec (fast fibers);
IV. Characteristics of Action Potential (AP): Fast response type, Na^+ dependent, phase 4 with automatism (diastolic depolarization) and refractory period longer than the left branch: slower depolarization and repolarization.

RRP: Relative Refractory Period
ARP: Absolute Refractory Period
APD: Action Potential Duration



V. Portions of the Right Bundle Branch:

1) **Proximal or membranous**: It is 15 to 20 mm long and it is related to the following structures: septal fascicle of the tricuspid valve, right coronary valve and non-coronary valve of the aortic valve, anterior fascicle of the left branch, fibrous trigone and membranous septum.

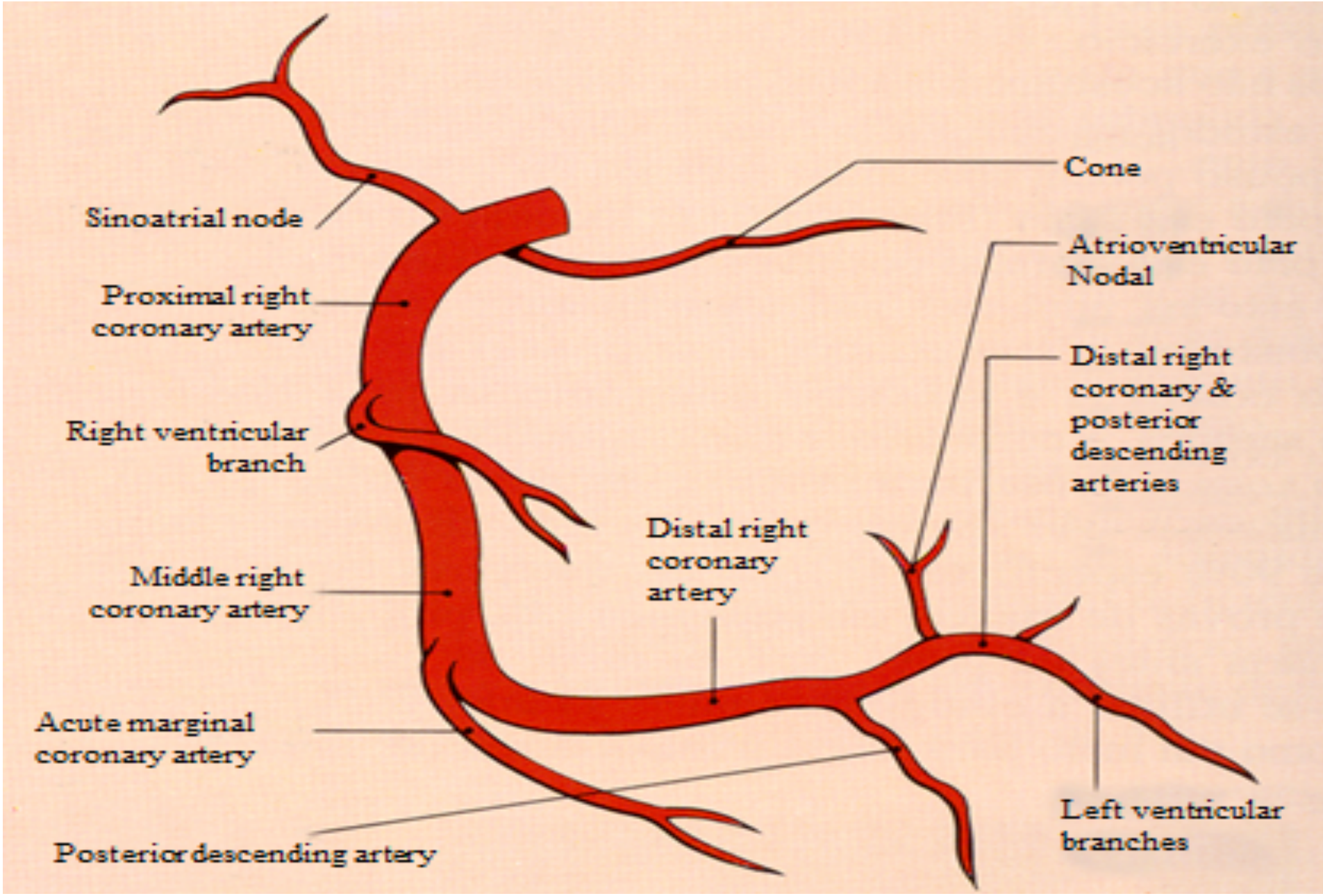
2) **Middle, intramyocardial or mimetic**;

3**) Inferior, distal or intra-moderator band**: From 30 mm to 40 mm long, with an initial portion of 20 mm to 25 mm called, middle, intramyocardial or mimetic, which begins in the apex of the muscular septum and ends where the moderator band begins, and it continues with the distal or subendocardial portion from 10 to 15 mm, which runs inside the moderator band, ending in the base of the papillary muscle of the tricuspid valve, where it splits in three into its superior, middle and inferior divisions.

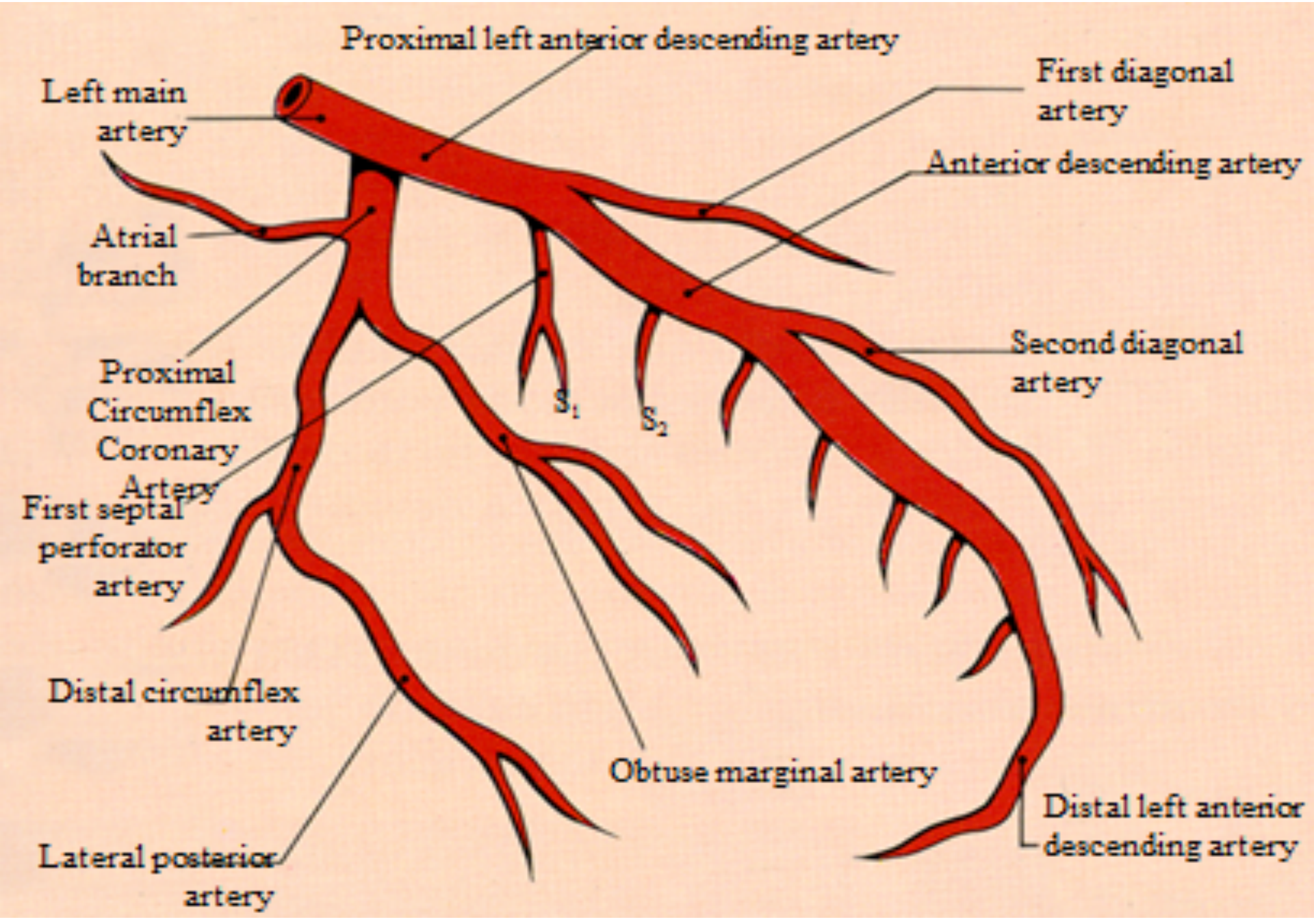
VI. Irrigation of the Right His System (RHS): The proximal portion of the right bundle branch and the His bundle is irrigated by the AV node artery of the right coronary artery (RCA) and the first septal perforator artery of the left anterior descending artery (LAD). Possibly, the right branch in its middle portion is irrigated by: septal branches of the posterior descending artery (PDA), of the second septal perforator artery of the LAD, and Kugel's artery, branch of the left circumflex artery (LCA). The middle and distal portions of the right branch are irrigated by the "ramus limbi dextri," branch of the second septal perforator of the LAD.

VI. Irrigation of the Right His System (RHS)

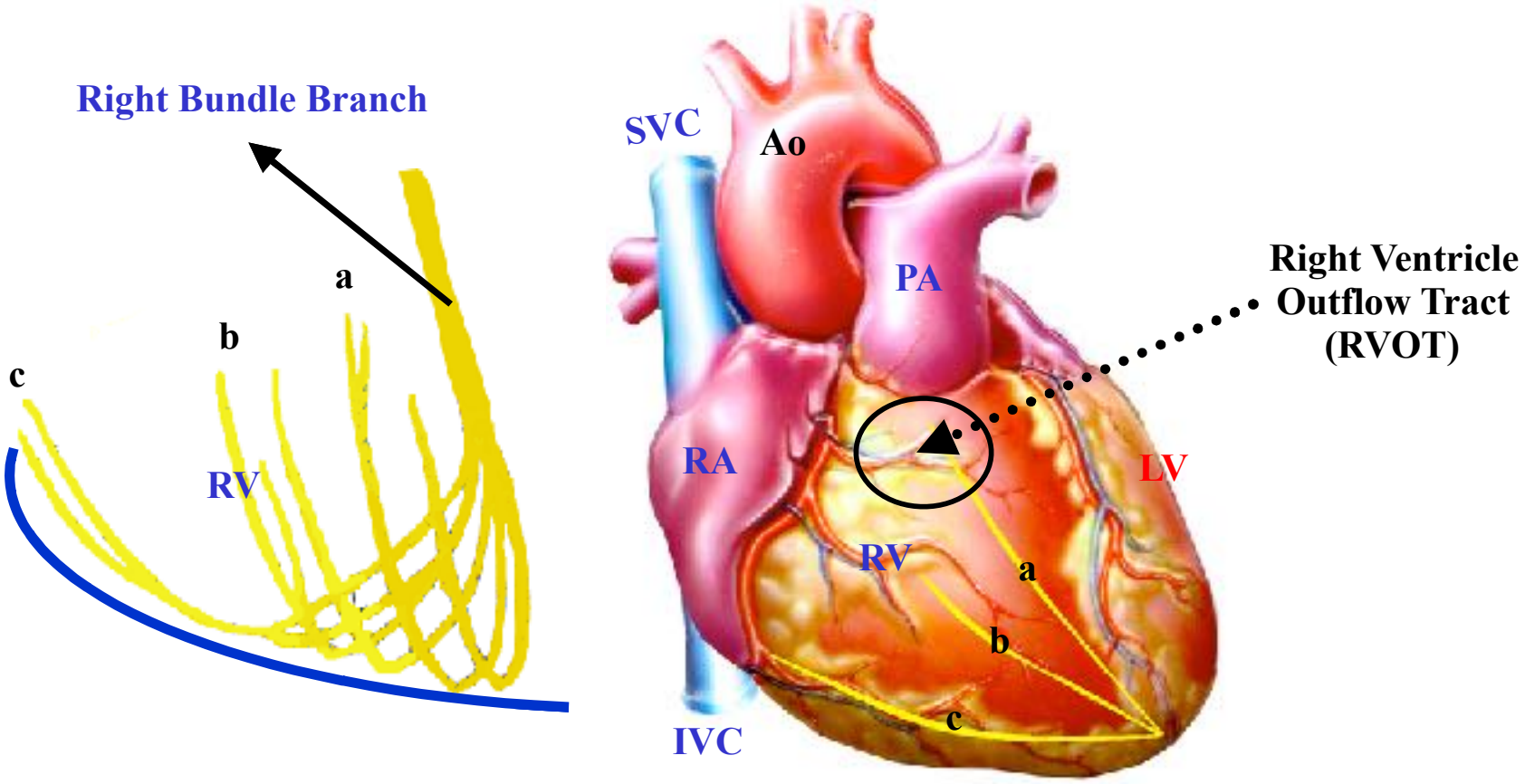
Right coronary artery



Left coronary artery



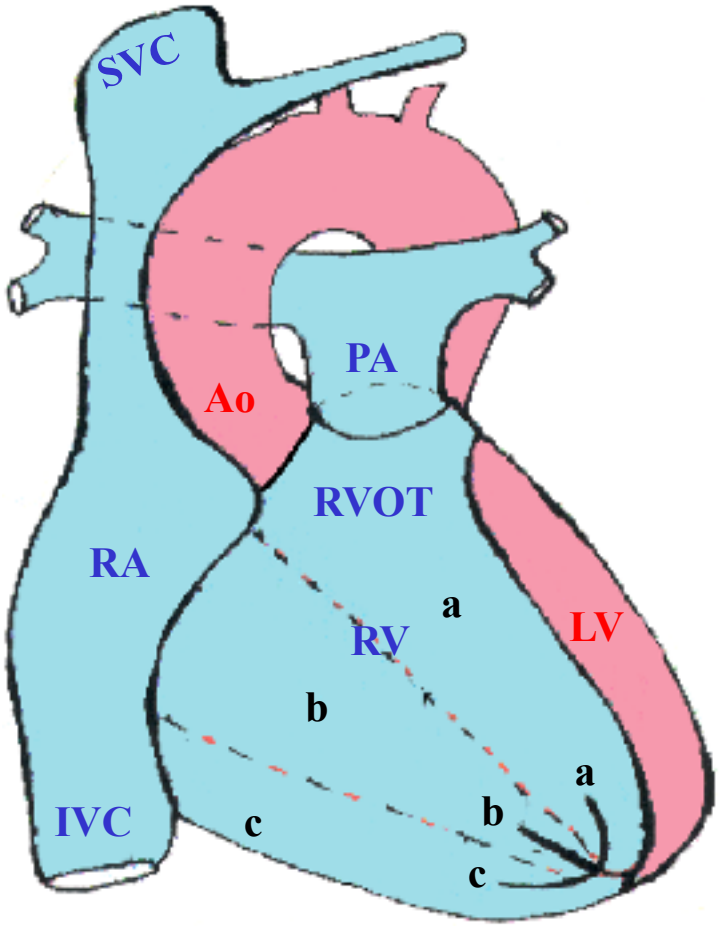
Right Branch Fascicles on free wall of RV: Divisional portion of the Right His System



The blocks at this level (level 6), occur in the free wall and the three divisions are involved at the same time, and thus they are called Right Global Fascicular Blocks (RGFB). When they are in isolation, they are called focal, Purkinje, zonal or divisional blocks.

a) Superior, anterosuperior or subpulmonary division of Right Bundle Branch
b) Middle, septal or anteroinferior division of Right Bundle Branch
c) Inferior, posterior or posteroinferior division of Right Bundle Branch

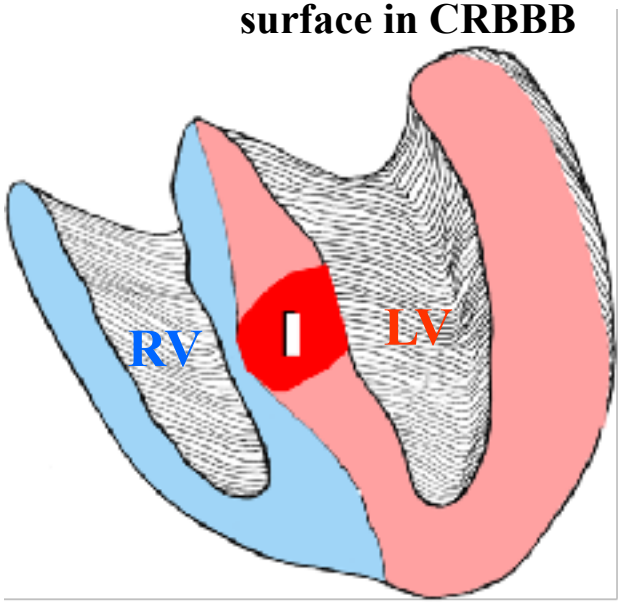
Distribution of the three divisions of the right branch in the RV free wall



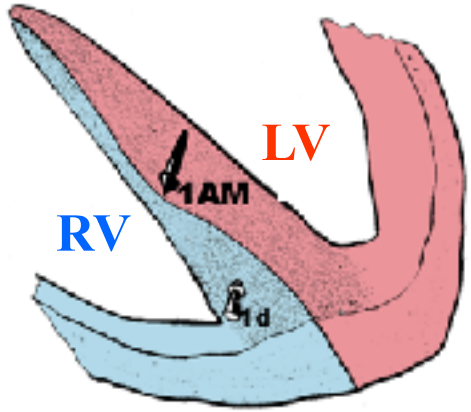
Divisions of the Right Bundle Branch:

a) Territory of the superior, anterosuperior or subpulmonary division;
b) Territory of the middle, septal or anteroinferior division;
c) Territory of the inferior or posteroinferior division.

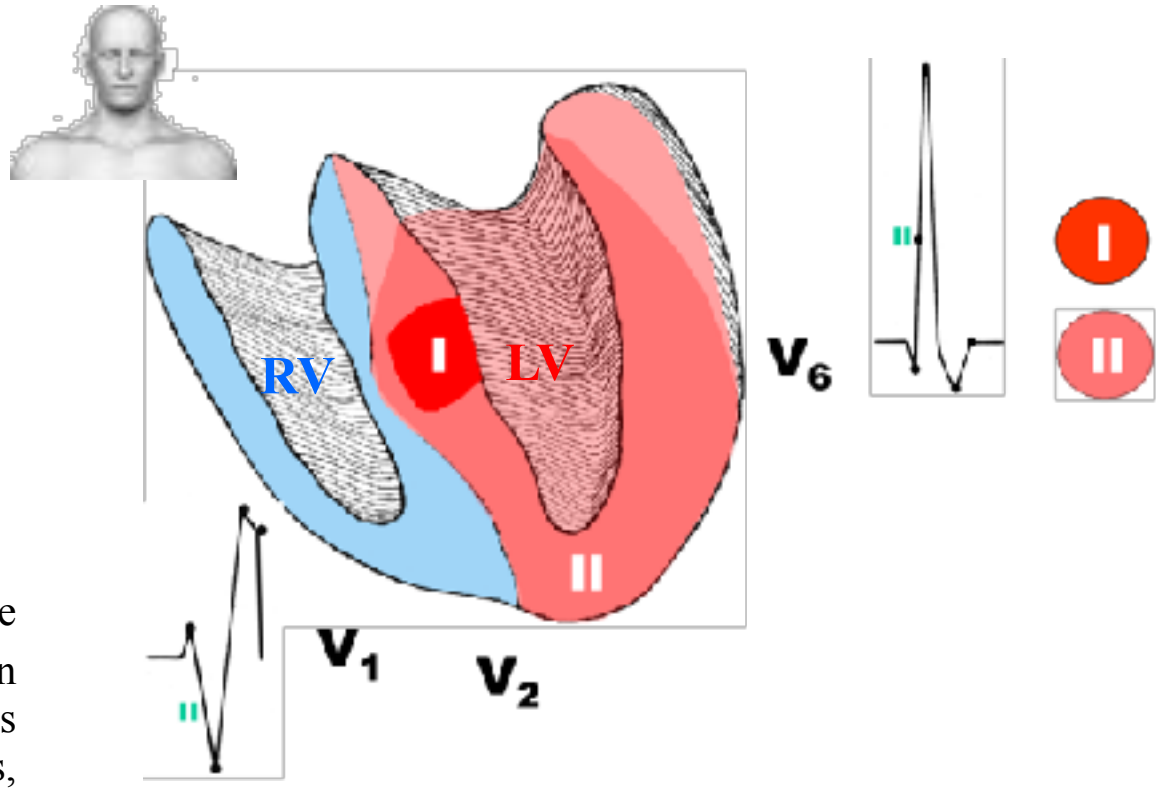
Location of initial activation (10 ms) of vector I_{AM} in the middle third of left septal surface in CRBBB



Note: vector 1 or vector 1_{AM} depends on the left system, does not present the opposition to vector 1d of blocked right branch. This fact does not modify the initial 10 to 20 ms, because left forces are stronger.



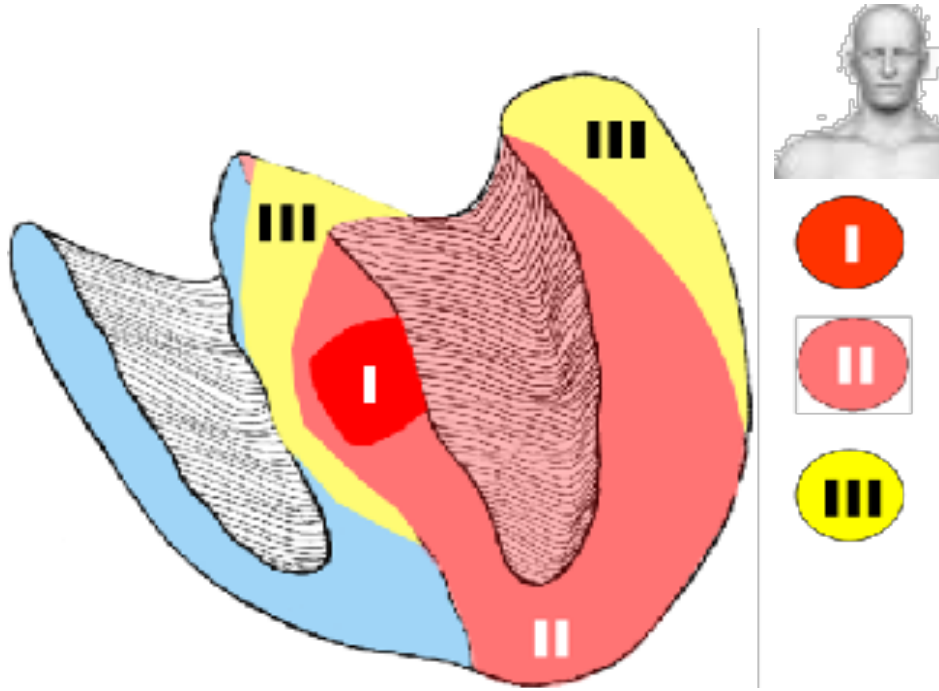
Representation of activation of vector II from 20 ms to 40 ms in CRBBB



Vector II of 20 to 40 ms. Over this time, the activation of the left side of the septum and the apical and anterior region of the left ventricle occurs. The low septum is facing the intermediary leads V_3 and V_4 while the apical region by the leads V_5 and V_6.

A) Descending ramp of r wave of V_1;
B) End of ascent of initial r wave of V_2;
C) Onset of ascent of R wave of left leads V_5, V_6, I and aVL.

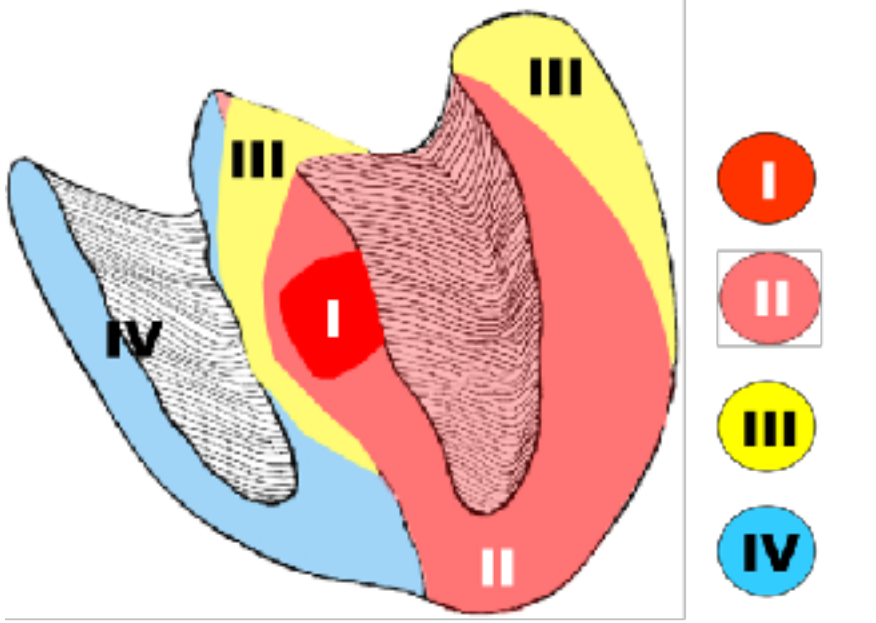
Representation of activation of vector III from 40 to 80 ms in CRBBB



In this phase the LV activation is completed, initially in the apical region of the free wall (V_5, V_6) and finally in the high lateral one (I and aVL) from the endocardium to the epicardium (vector III). Concomitantly the stimulus goes through the septal barrier from left to right, where it suffers a noticeable dromotropic delay, activating a left part of the right septum.

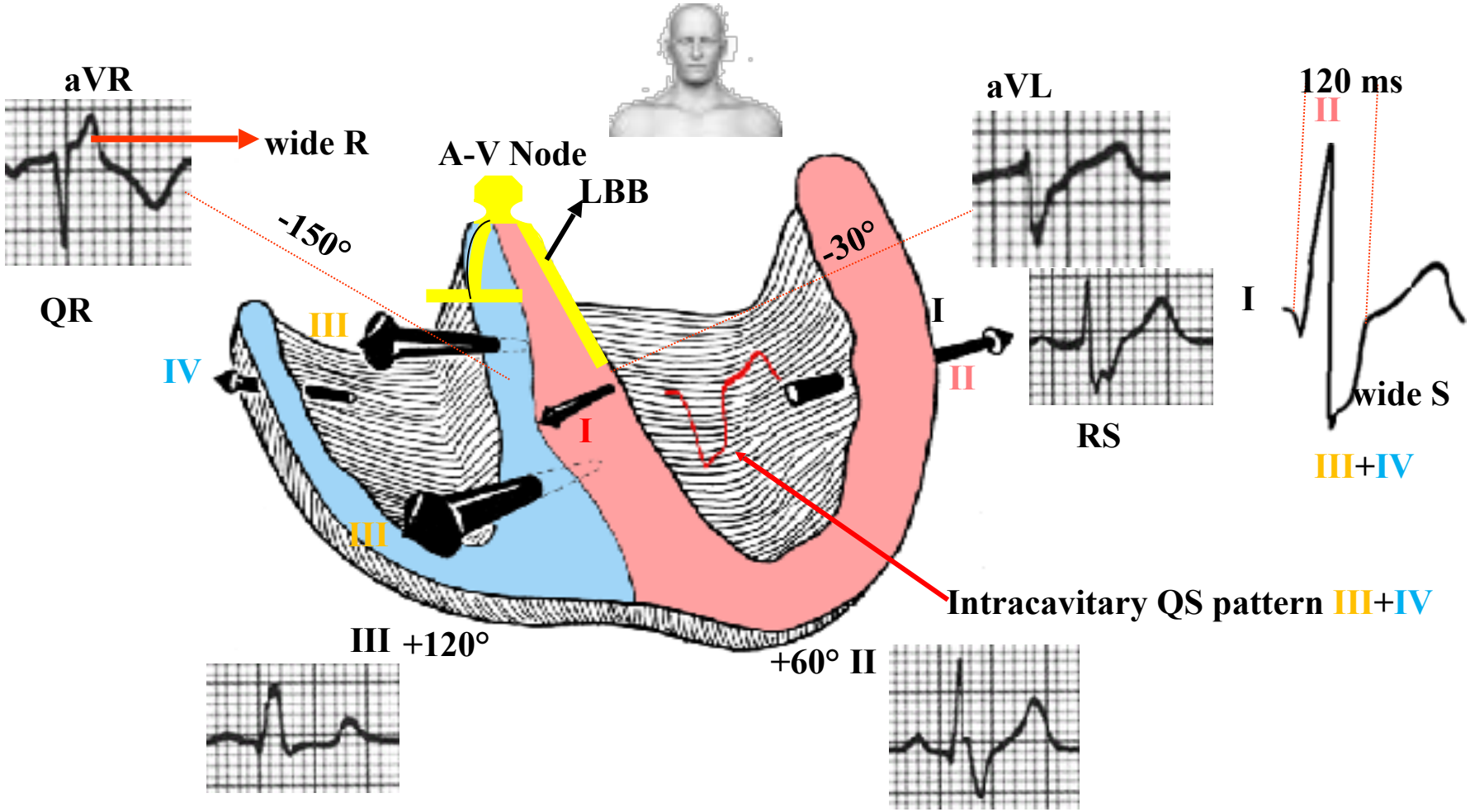
Vector III is responsible for the nadir of S wave from V_1 and V_2 and the apex of R wave of left leads V_5, V_6, DI and aVL.

Representation and explanation of activation of vector IV from 80 to 120 ms or more in CRBBB



In phase IV the septal and RV free wall activation is completed in a tangential way, as in the atria, and the wall is reached first by two wavefronts (anterior and posterior) emerging from the apex, and constituting a front of activation with a V shape in the direction of the RVOT, heading to the front and the right. The activation of this last structure is then processed by two fronts: one coming from the free wall and the other from the high septal portion through the crista supraventricularis. This explains the QRS loop of VCG, which in its final part is located to the front and the right, forming an appendage that resembles a finger pointing to the direction of the RVOT: to the front and right. Phase IV is responsible for wide R' wave of right precordial leads V_3R, V_1 and V_2 and the wide S wave in left leads V_5, V_6, I and aVL.

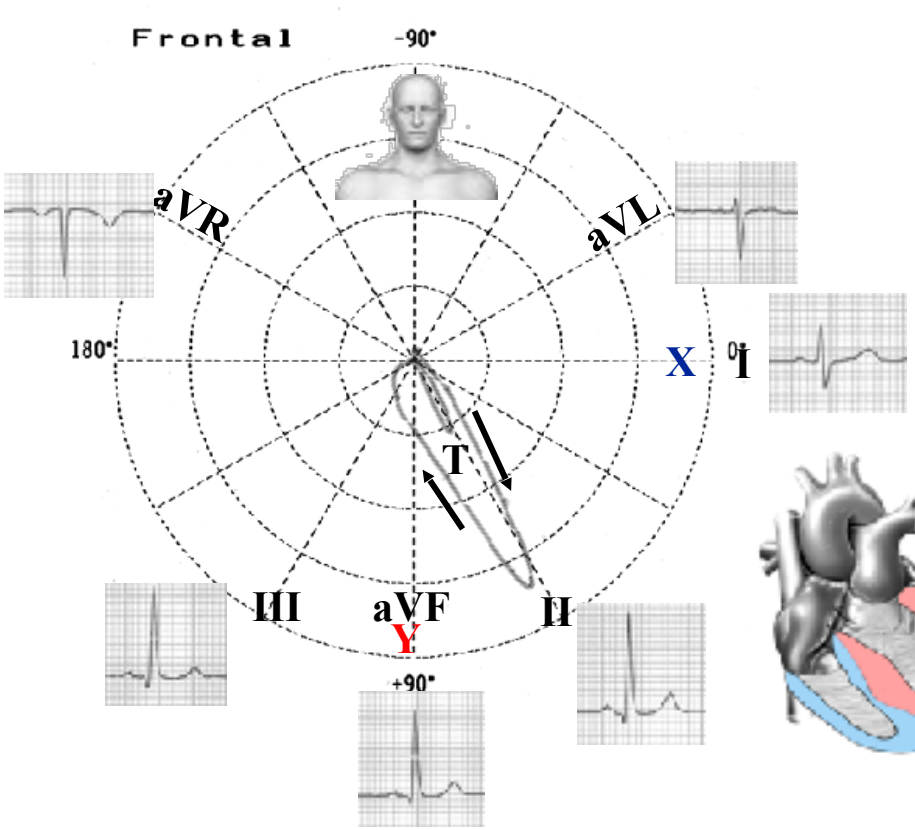
The four activation vectors of the biventricular chamber in CRBBB



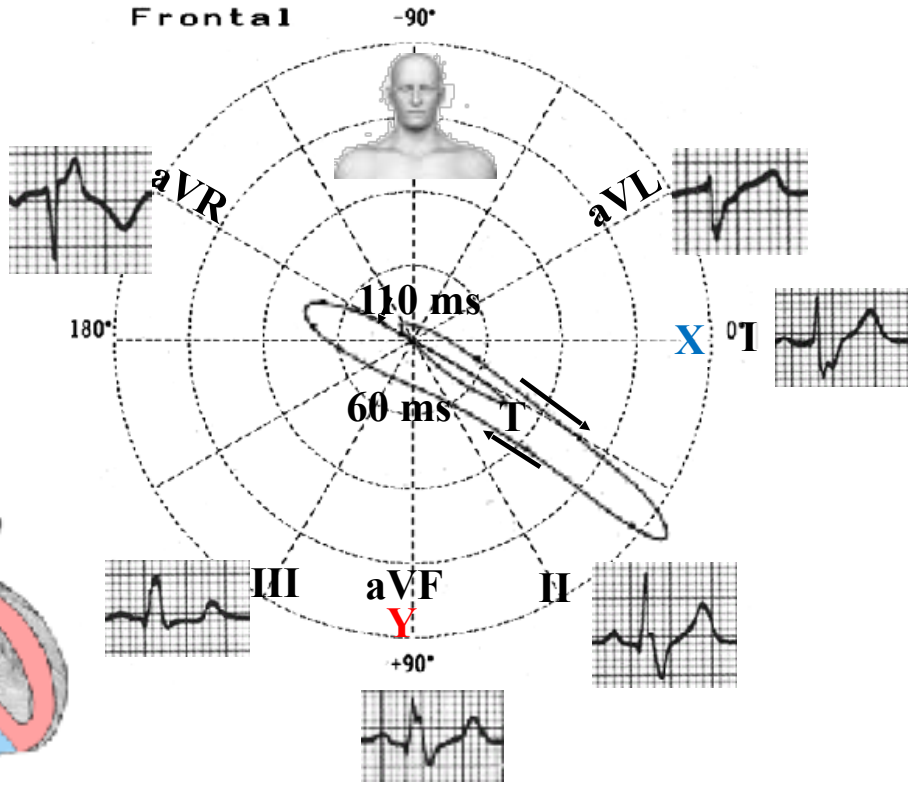
I: Middle third of left septal surface;
II: LV free wall from endo to epicardium;
III: Slow trans-septal vectors;
IV: RV outflow tract (RVOT) .

Comparison between normal QRS and T and RBBB loops in the FP

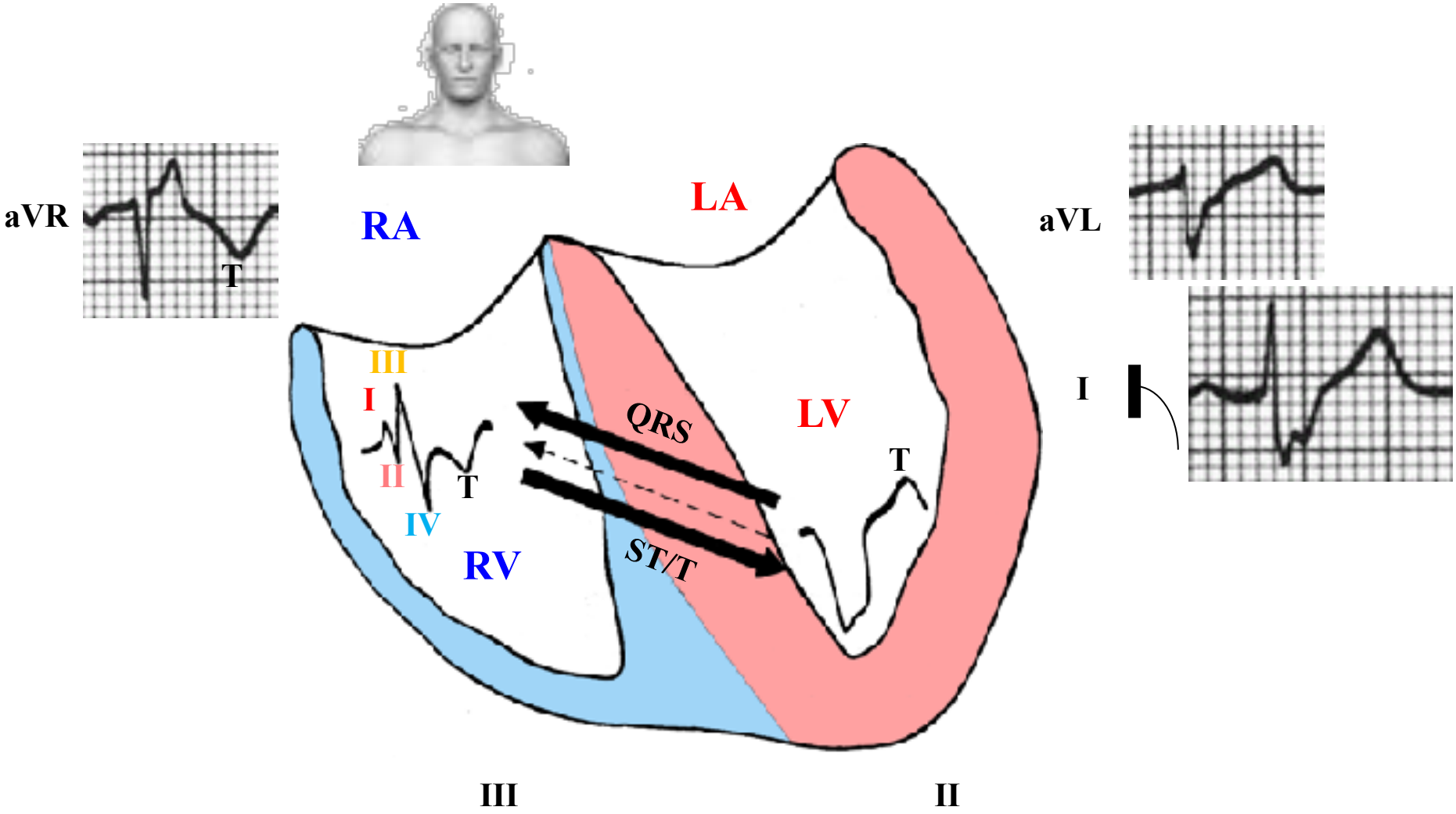
Normal QRS and T loops



QRS and T loops in RBBB



Representation of ventricular repolarization in CRBBB



QRS-T angle near 180º. Secondary alteration of ventricular repolarization: asymmetrical T wave with initial branch of slow and finally fast recording. Only intra RV cavitary morphology has the 4 depolarization vectors. T loop of VCG with efferent limb that shows dashes closer than the dashes of the afferent limb.

Possible etiologies of Complete RBBB

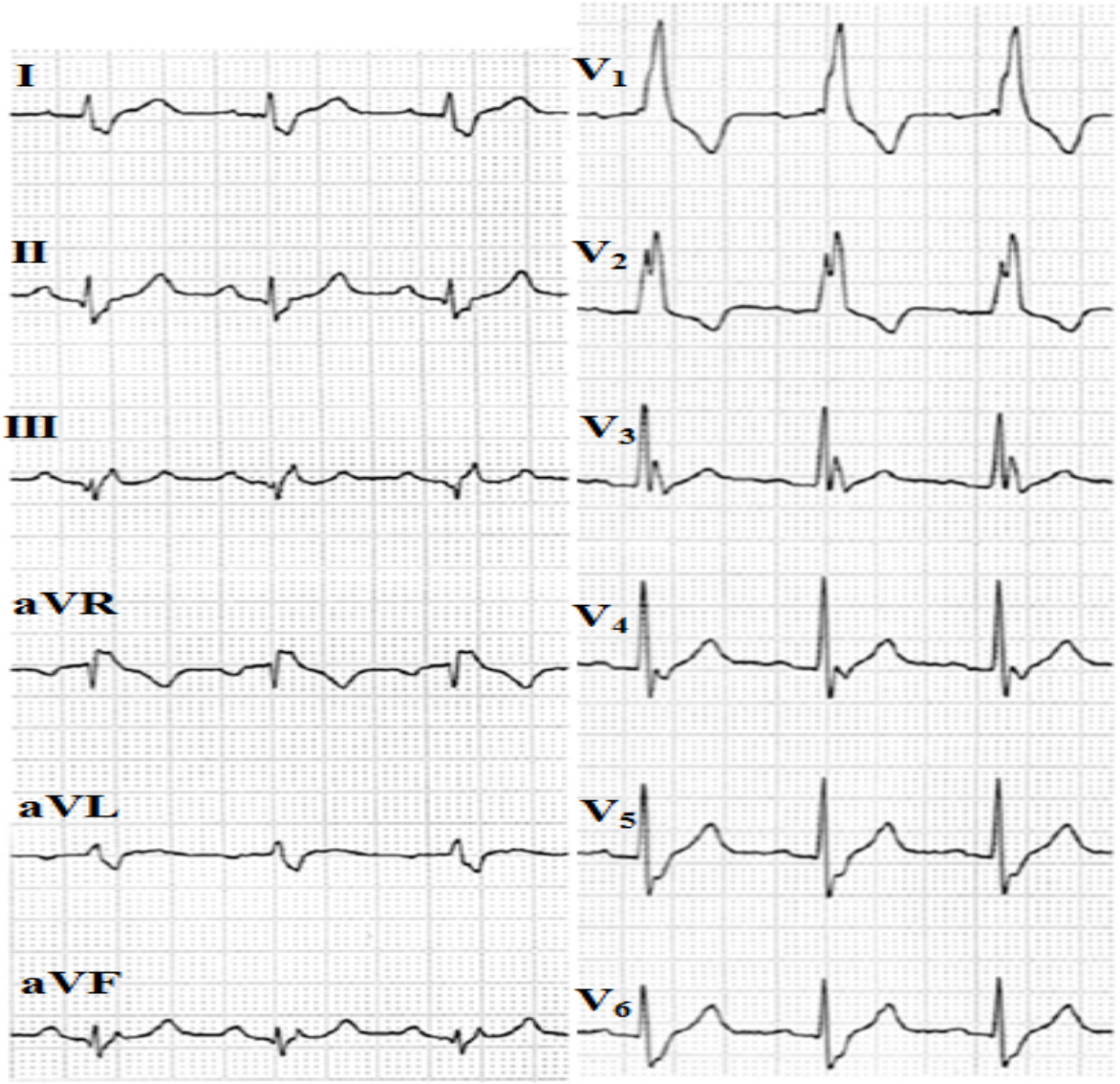
1) Normal variant: rare: the incidence is 1.8 per 1,000. Below age 30 the incidence is 1.3 per 1,000 and between ages 30 and 44 is ranged from 2.0 to 2.9 per 1,000 (**Hiss 1962**). r' wave <0.6 mV than the initial R wave that comes last < 0.8 mV. RBBB and IRBBB were two to three times more common among men than women. RBBB was associated with increased cardiovascular risk and all-cause mortality, whereas IRBBB was not. Contrary to common perception, RBBB in asymptomatic individuals should alert clinicians to cardiovascular risk (**Bussink 2013**).

2) Congenital Heart Diseases:

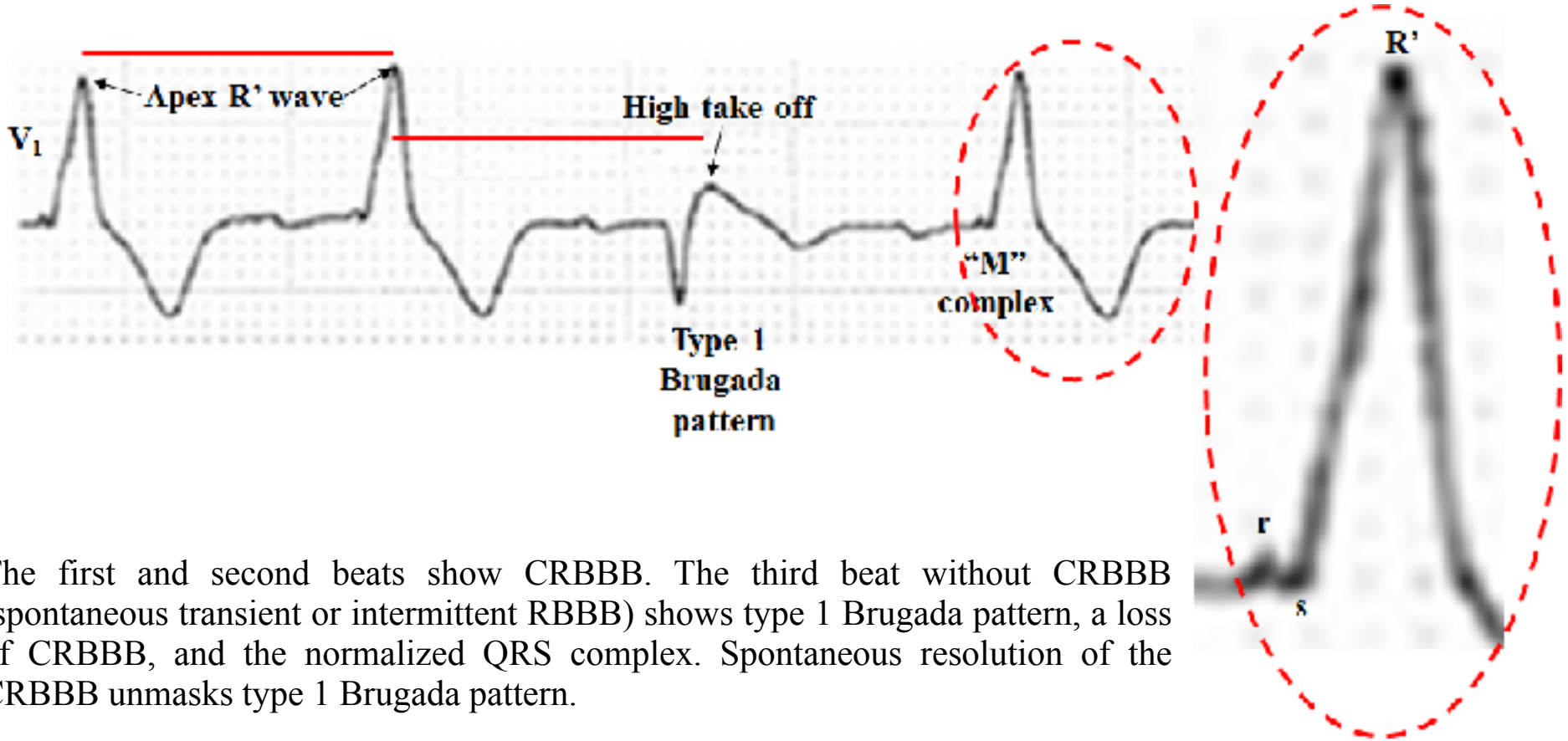
- Atrial Septal Defects (ASD): present in more than 90% of the cases, whether in the ostium secundum or in the ostium primum (**de Micheli 1978**).
- Partial or total anomalous pulmonary vein drainage in the right atrium;
- Ebstein's anomaly: bizarre RBBB, of low voltage and with initial q wave (**Tabatabaei 2009**);
- Uhl's anomaly (parchment RV) (**Hébert 2010**);
- Ventricular Septal Defects (VSD) in the presence of Biventricular Hypertrophy (BVH);
- Pulmonary Stenosis (PS), especially in the moderate form and particularly for PS, TOF or larged VSD (**Gelband 1971**);
- Tetralogy of Fallot (TOF) (pre & post-surgery) (**Krongrad 1974**);
- Aortic Stenosis (Ao.S.): congenital, bivalvular, calcification (**Koos 2011**);
- After injection of absolute alcohol in the first septal perforator artery of the Left Anterior Descending artery (LAD), in the treatment of non responsive severe forms to pharmacological treatment in hypertrophic obstructive cardiomyopathy (HCM-O) (**Riera 2002**).

3) Genetic-familial causes:

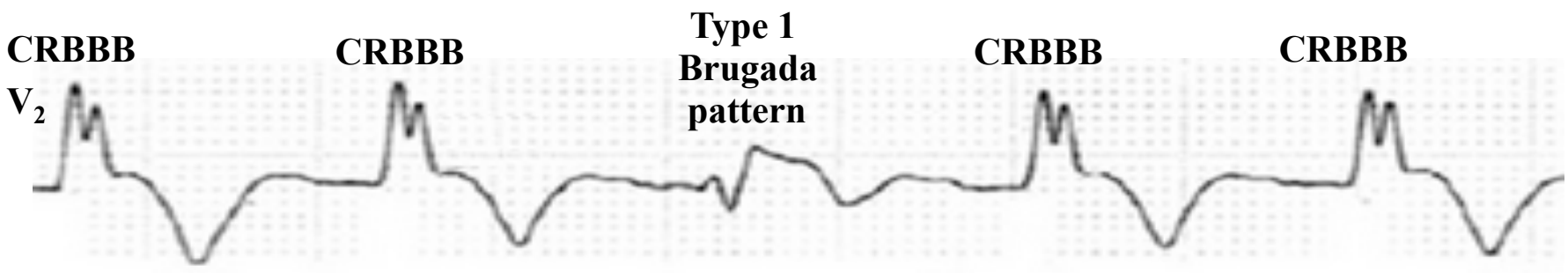
- Brugada syndrome: atypical CRBBB, frequent absence of wide S wave in left leads and ST segment elevation from V_1 to V_3 (SCN5A gene). Additionally, Brugada syndrome can be masked by CRBBB (**Tomita 2012**).



The 12-lead ECG showed CRBBB pattern: QRSD 140 ms, late R in V_1, final broad R wave in aVR, and wide terminal S in left leads. QRS duration = 140 ms.



The first and second beats show CRBBB. The third beat without CRBBB (spontaneous transient or intermittent RBBB) shows type 1 Brugada pattern, a loss of CRBBB, and the normalized QRS complex. Spontaneous resolution of the CRBBB unmasks type 1 Brugada pattern.



- Sclerosis, degeneration and idiopathic fibrosis of the specific His conduction system or Lenègre disease (SCN5A gene).
- Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy (ARVD/C)

4) Associated to acquired heart diseases:

- Chronic Chagasic Cardiomyopathy (**Elizari 1999**): classical extreme shift of SÂQRS to the left by association with LAFB.
- Complications of Acute Myocardial Infarction (AMI): high risk of evolving into complete AV block (**Melgarejo-Moreno 1997**).
- Mitral Stenosis (MS): RBBB existence correlates with the severity of the disease and the grade of valve calcification in moderate and severe pure mitral stenosis (**Ocal 2006**);
- Chronic Obstructive Pulmonary Disease (COPD): Right Atrial Enlargement (RAE), RVH, RBBB, marked clockwise rotation of the heart, a QS pattern in leads III and aVF, LAD, PACs, and SVTs were significantly more prevalent in patients with severe COPD than in patients with mild or moderate COPD (**Holtzman 2011; Miguel 1958; Caird 1962**);
- Acute Pulmonary Embolism (APE): it appears suddenly and briefly (**Zhong-Qun 2014**);
- Hypertension: It was significantly more common; observed in 60% of cases from the Framingham study during follow-up period of 18 years. Although the initial appearance of RBBB was usually unaccompanied by overt clinical events, the subsequent incidence of CHD and CHF was two and one-half and four times greater, respectively, than that in matched control subjects without RBBB. The incidence of cardiovascular disease mortality was almost three times greater in people who developed RBBB than in an age-matched sample of the population-at-large. This excess of cardiovascular disease mortality was related primarily to the high prevalence of associated cardiovascular abnormalities. (**Schneider 1980**);
- Sclerosis of the left side of the cardiac skeleton or Lèv disease (**Lev 1975; Lev 1964**) (do not mistake with Lenègre);
- Sclerosis of the fibrous trigon, membranous septum, aortic valve ring, and apex of the muscular septum.

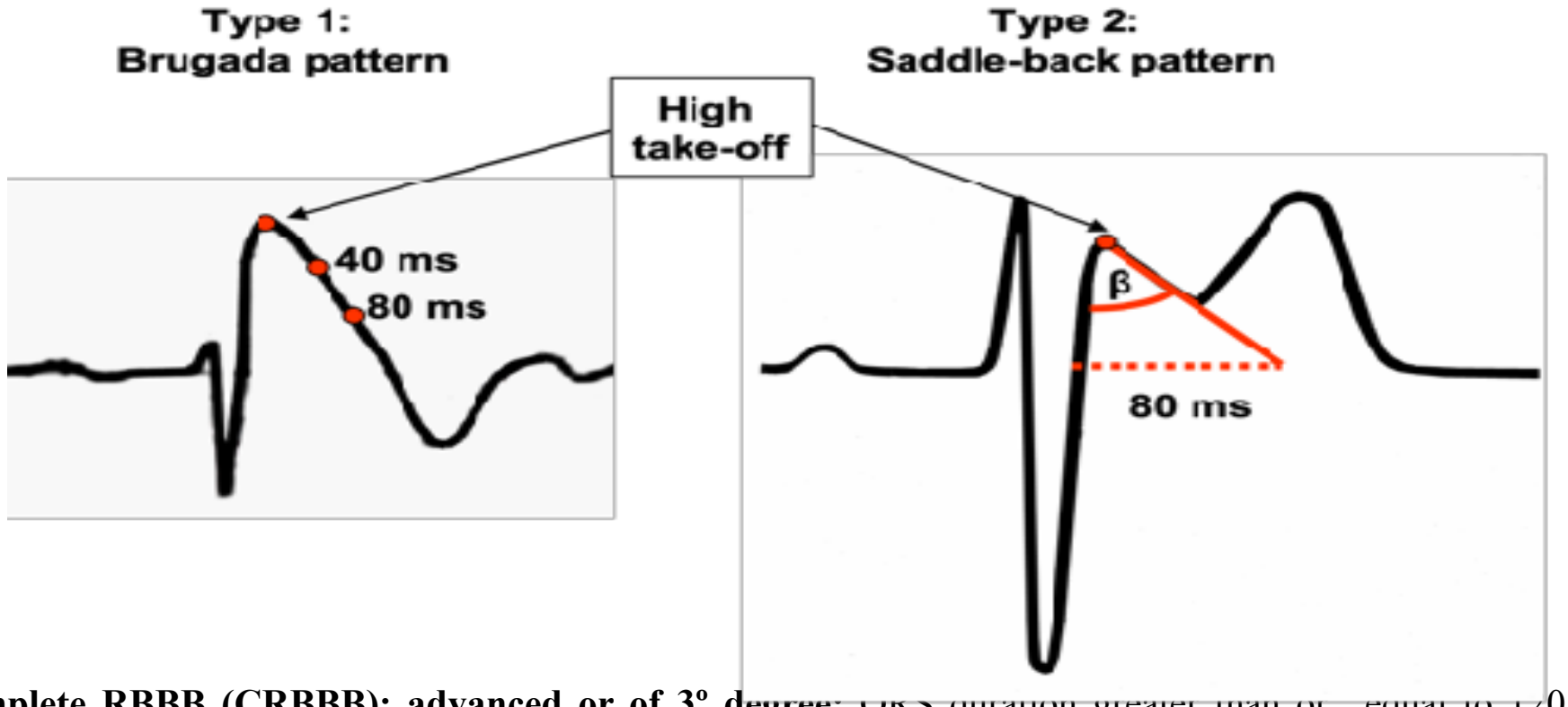
- Acute congestive heart failure (ACHF): RBBB is a powerful predictor of mortality in patients with ACHF. Early identification of this high-risk group may help to offer tailored treatment in order to improve outcome (**Mueller 2006**).
- Systemic sclerosis: ECG abnormalities are common in patients with early systemic sclerosis and are associated with the severity of lung and heart involvement. RBBB is an independent predictor of mortality, and should be considered a marker of disease severity in systemic sclerosis.(**Draeger 2013**)
- Acquired complete right bundle branch block without overt cardiac disease (**Lancaster 1972**).
- Following non-penetrating chest trauma without evidence of cardiac contusion (**Kumpuris 1979**).

Criteria for RBBB classification

A) According to QRS duration (**Surawicz 2009; Willems 1985**)

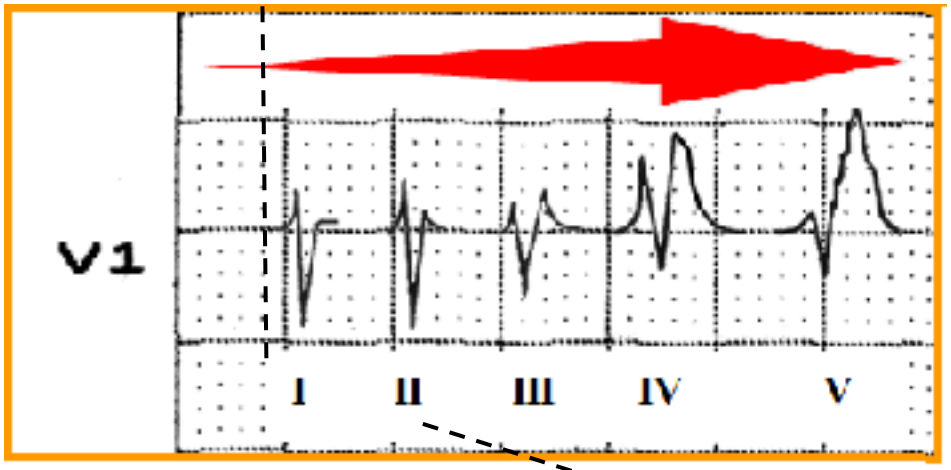
Incomplete RBBB (IRBBB): QRS duration between 100 and 120 ms in adults, between 90 and 100 ms in children between 4 and 16 years of age, and between 86 and 90 ms in children less than 8 years of age.
The evidence presented by Moore and his colleagues that the incomplete RBBB pattern may be due to a heritable focal hypertrophy of the RV, and not because of delayed conduction within the right bundle branch, should stimulate us to reappraise the validity of our electrocardiographic vocabulary. Continued anatomic and electrophysiologic research is vital to verify or disprove our current electrocardiographic concepts, so that the words accurately reflect the best known facts about electrical activity of the heart (**Massing 1972; Barker 1949**).
In patients with suspected Brugada syndrome, simple ECG criteria can enable discrimination between incomplete RBBB and types 2 and 3 Brugada patterns. The angle between a vertical line and the downslope of the r‘ wave, and β, the angle between the upslope of the S-wave and the downslope of the r - wave in benign or “innocent” IRBBB is narrower related to type 2 Brugada ECG pattern (**Chevallier2011**). Truly Type 2: The r´ wave is rounded, wide and usually of relatively low voltage. Angle between the upslope of the S-wave and the downslope of the r‘ wave (β angle) > 58°.

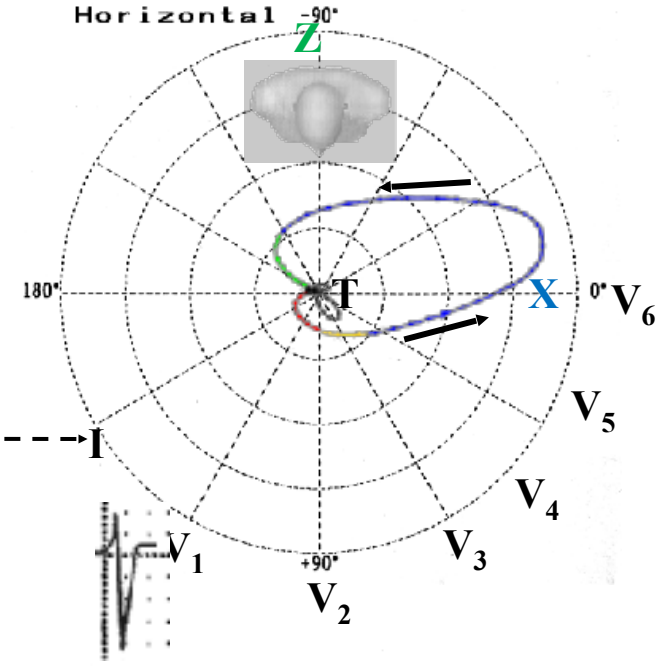
The descending arm of r´ coincides with the beginning of the ST segment (J point). The duration of the base of the triangle of r´ at 5 mm from high take-off >3.5 mm. QRS duration is longer in Brugada pattern type 2 than in other cases with r´ in V1 and there is a mismatch between V1 and V6. In Brugada pattern the QRS complex end is earlier in V6 than in V1-V2.(**Bayés de Luna 2012**).

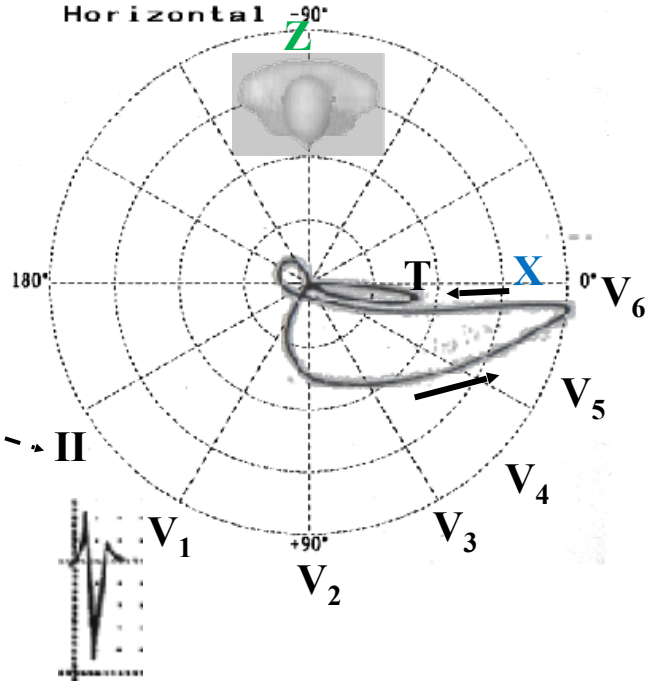


Complete RBBB (CRBBB): advanced or of 3º degree: QRS duration greater than or equal to 120 ms; greater than 100 ms in children ages 4 to 16 years; and greater than 90 ms in children with less than 4 years of age. It is considered complete RBBB when the QRS duration is ≥120 ms or ≥60 dashes. The figure in the next slide shows the correlation between the QRS pattern in V1 and the QRS loop in progressive increase degree of RBBB.(**Peñaloza 1961**).

I – Normal conduction
II, III, IV – Incomplete RBBB
V – Complete RBBB







Criteria for Right Bundle Branch Block classification

B) According to the Mexican school criteria (Sodi 1964; Rodriguez 1952)

1) First degree RBBB
2) Second degree RBBB
3) Third degree or complete RBBB

C) According to the Spanish school criteria (Bayés de Luna 1999):

1) Global Right Ventricular Block (GRVB)

- According to topography: Proximal
- Peripheral.
- According to the degree:
  - ✓ Advanced, complete or of the third degree;
  - ✓ Not advanced (it corresponds to the first and second degree or incomplete);

2) Partial blocks: it corresponds to divisional, terminal, focal, divisional, or Purkinje blocks (**Tobias 1986; Pastore 1983; Luna Filho 1989**):

- Superoanterior;
- Inferoposterior;
- Middle.

D) According to Van Dam, RTh. and Janse, MJ (Janse 1971).

Complete or Proximal Right Bundle Branch Block (CRBBB);

- Incomplete Right Bundle Branch Block (IRBBB):
  - ✓ Distal or in the moderator band (predivisional).
  - ✓ Terminal, focal, divisional or Purkinje.

Our proposal of classification for RBBB: Classification of CRBBB by topography: pre and post-divisional

1) By topography:

1a) Predivisional

a) Proximal: Of right His or of the trunks of the right bundle branch;

b) Peripheral: intramyocardial or middle and of the moderator band or inferior;

1b) Post divisional

- Parietal or Divisional: Global end conduction delays (ECD): divisional, terminal, focal.
- Purkinje or partial.

2) By duration of QRS:

- **Complete or Advanced Right Bundle Branch Block (CRBBB or ARBBB):** QRS duration ≥ 120 ms in adults, greater than 100 ms in children from 4 to 16 years of age, and greater than 90 ms in children less than 4 years of age. Always in the truncus!!!!

- **Incomplete Right Bundle Branch Block (morphology of IRBBB):** with QRS duration between 100 ms and 110 ms in adults, between 90 and 100 ms in children between 4 and 16 years of age, and between 80 and 90 ms in children less than 8 years, which, by the morphology in V1 may be:

  2a) Typical: rsr' rSr' in V1 or V2.

  2b) Atypical: rS in V1 with S presenting notch in ascending slope.(**Schamroth 1985**)

Incomplete RBBB may be present in the absence of heart disease, particularly when the V1 lead is recorded higher than or to the right of normal position and r´ is less than 20 ms. In children, incomplete RBBB may be diagnosed when the terminal rightward deflection is less than 40 ms but greater than or equal to 20 ms.

3) By steadiness:

(3a) Permanent;

(3b) Temporary, transient or transitory (see next slide);

(3c) Intermittent (Okajima 1980):

(3c1) Dependent on heart rate:

- Tachycardia-dependent or in phase 3 (**Izumi 1996**);
- Bradycardia-dependent (**Kinoshita 2003**) or in phase 4: by mild or
- Moderate hypopolarization.

(3c2) Independent from hear rate:

By severe hypopolarization.

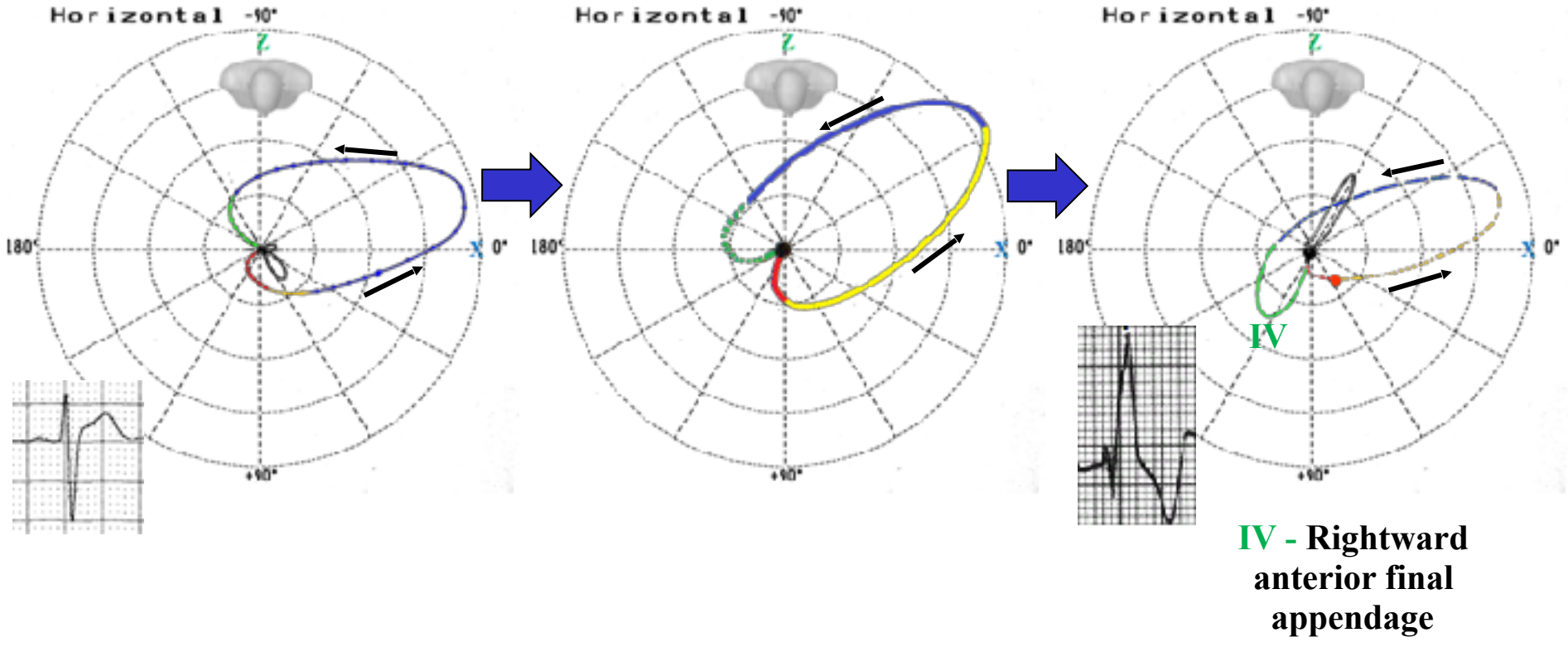
4) By form of onset:

(4a) Sudden: after acute pulmonary embolism Mobitz type II (**Nielsen 1989**).

(4b) Progressive: Mobitz type I (rare).

The figure below shows the effect on the configuration of the horizontal QRS loop produced by development of RBBB during cardiac catheterization. With onset of the block, a rightward anterior final appendage was produced, which gradually went away as the transient BBB cleared. A slight increase in the leftward forces, which were partially uncancelled because of the delay in activation of the RV, occurred with the block and gradually cleared.

Normal **Transition** **CRBBB**



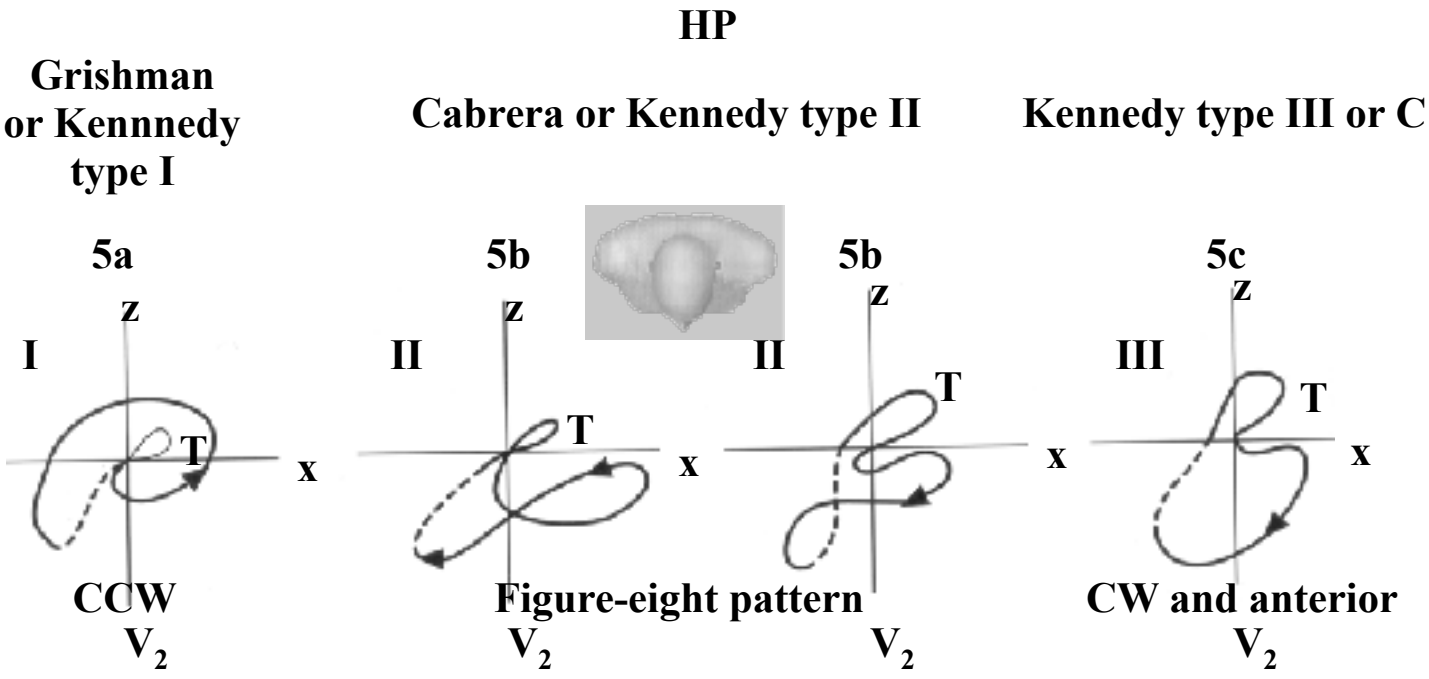
IV - Rightward anterior final appendage

5) By vectorcardiographic criteria: (it takes into account only the QRS loop in the horizontal plane)

(5a) "Grishman" or Kennedy type I. In turn it may be a or b;

(5b) "Cabrera" or Kennedy type II. In turn it may be a or b;

(5c) Kennedy type III or C.

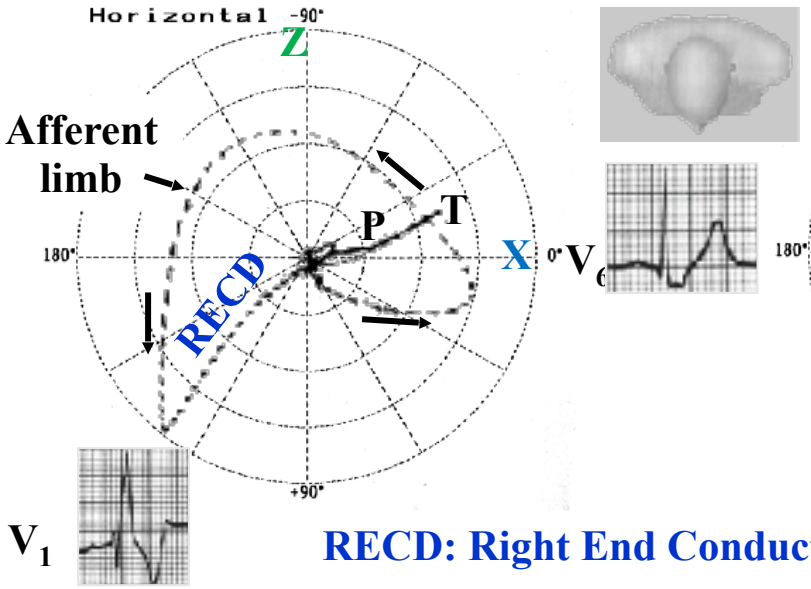


Classification criteria by vectorcardiographic criteria in the horizontal plane: type I; Grishman or Kennedy type II; Cabrera or Kennedy II and type III or C. In patients with RBBB, the position of the afferent limb in the horizontal plane can be used to predict cardiac failure or severe pulmonary disease (**Fedor 1976**).

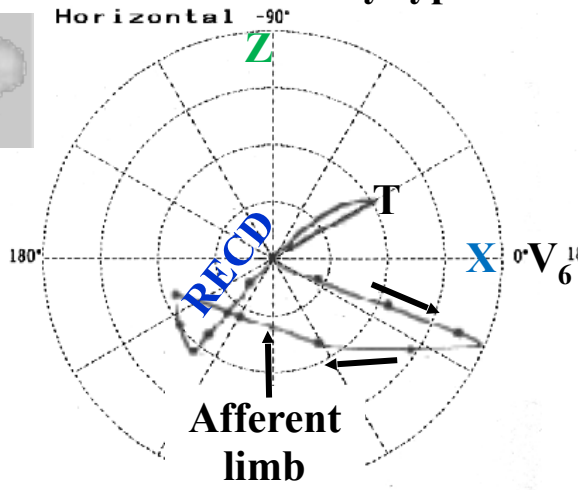
The QRS loop in CRBBB rotates in CCW or figure-eight pattern in most patients with benign RBBB, but in patients with RBBB associated with RVH, the QRS loop is more likely to be clockwise and completely anterior (**Baydar 1965**). The terminal dots are close together, reflecting end conduction delay. Since the RV is depolarized later than normal, the LV depolarizes without some of the normal cancelation from RV forces, and thus the maximum spatial voltage to the left (LMSV) may be increased slightly, simply from the conduction abnormality.

VCG classification of Complete Right Bundle Branch Block in the HP

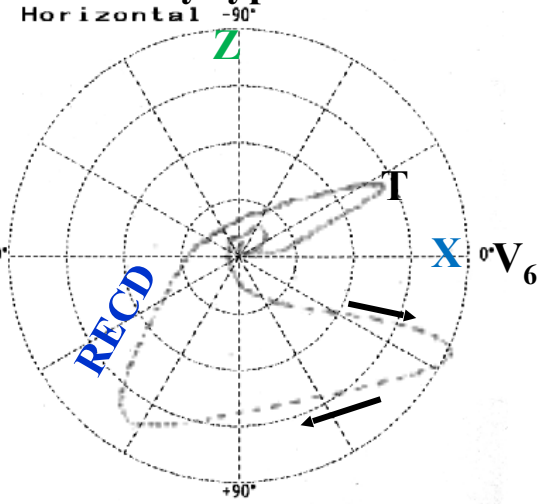
Grishman or Kennnedy type I



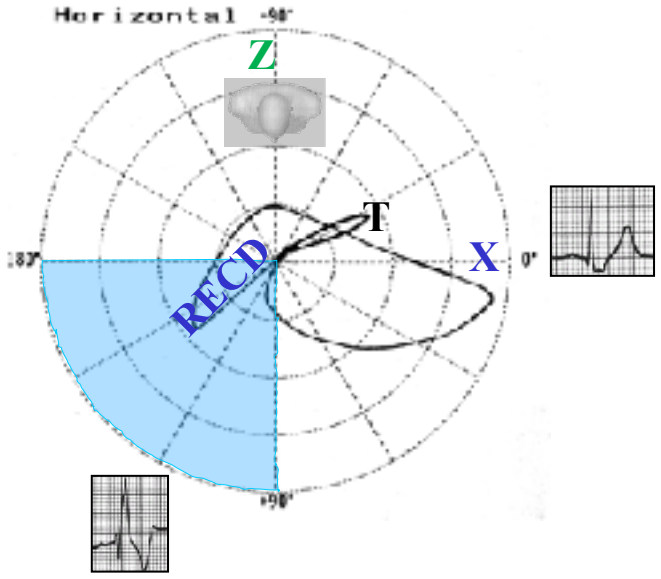
Cabrera or Kennedy type II

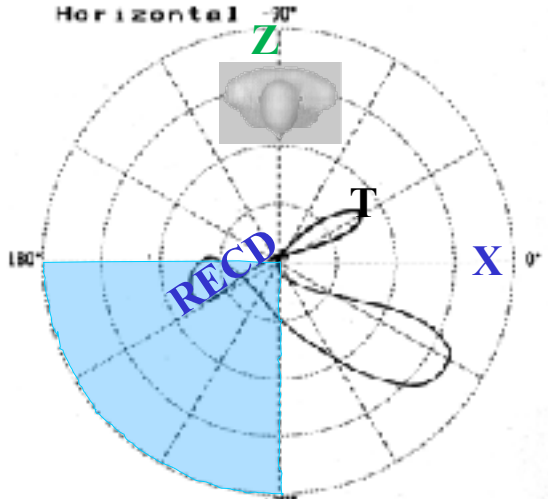


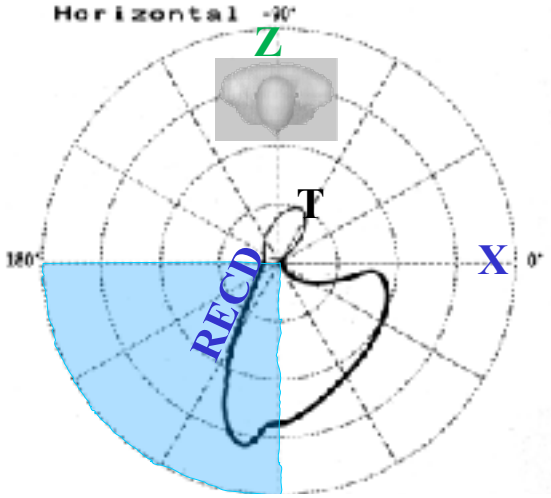
Kennedy type III or C



RECD: Right End Conduction Delay







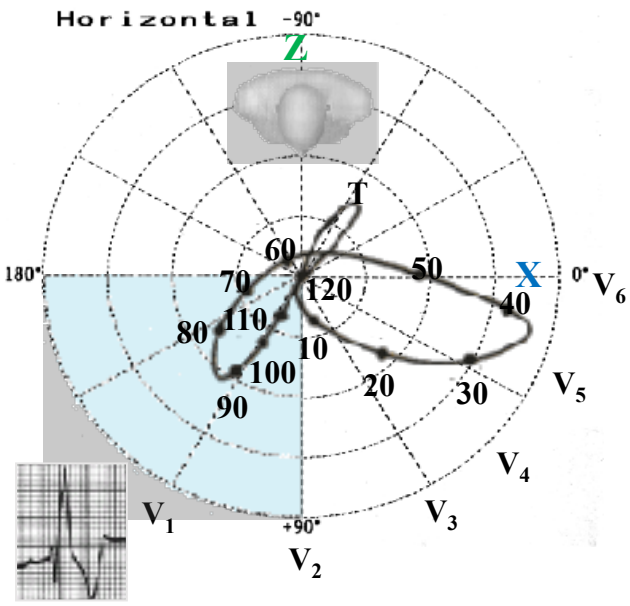
Right Anterior Quadrant

We find type II in ASD, PS, in COPD and more rarely in chronic Chagasic myocarditis.

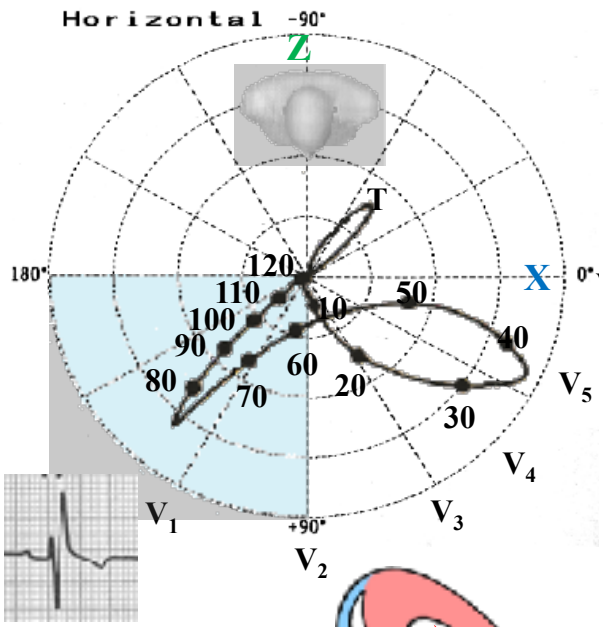
Initial vector to the front, QRS loop of CW rotation and main body located in anterior quadrants.

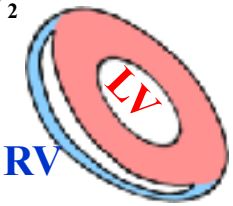
In three patterns the terminal vector of 60 ≥120 ms in "glove finger" (finger-like terminal appendix) located in the right anterior quadrant

Grishman or Kennnedy type I

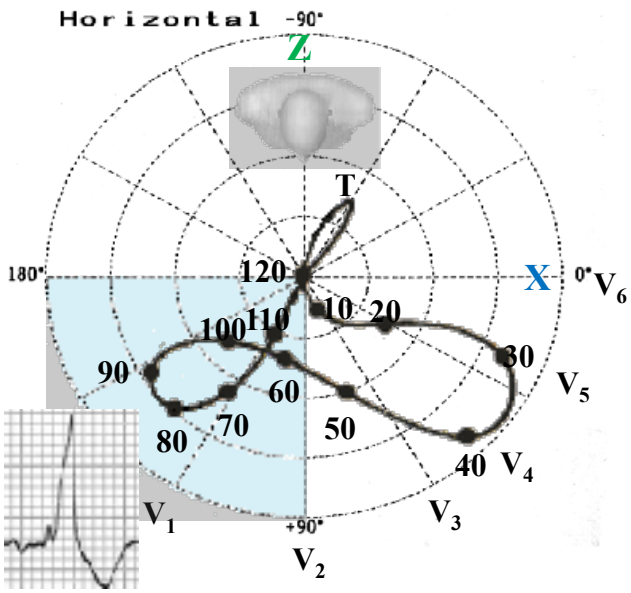


Cabrera or Kennedy type II





Kennedy type III or C



Right anterior quadrant

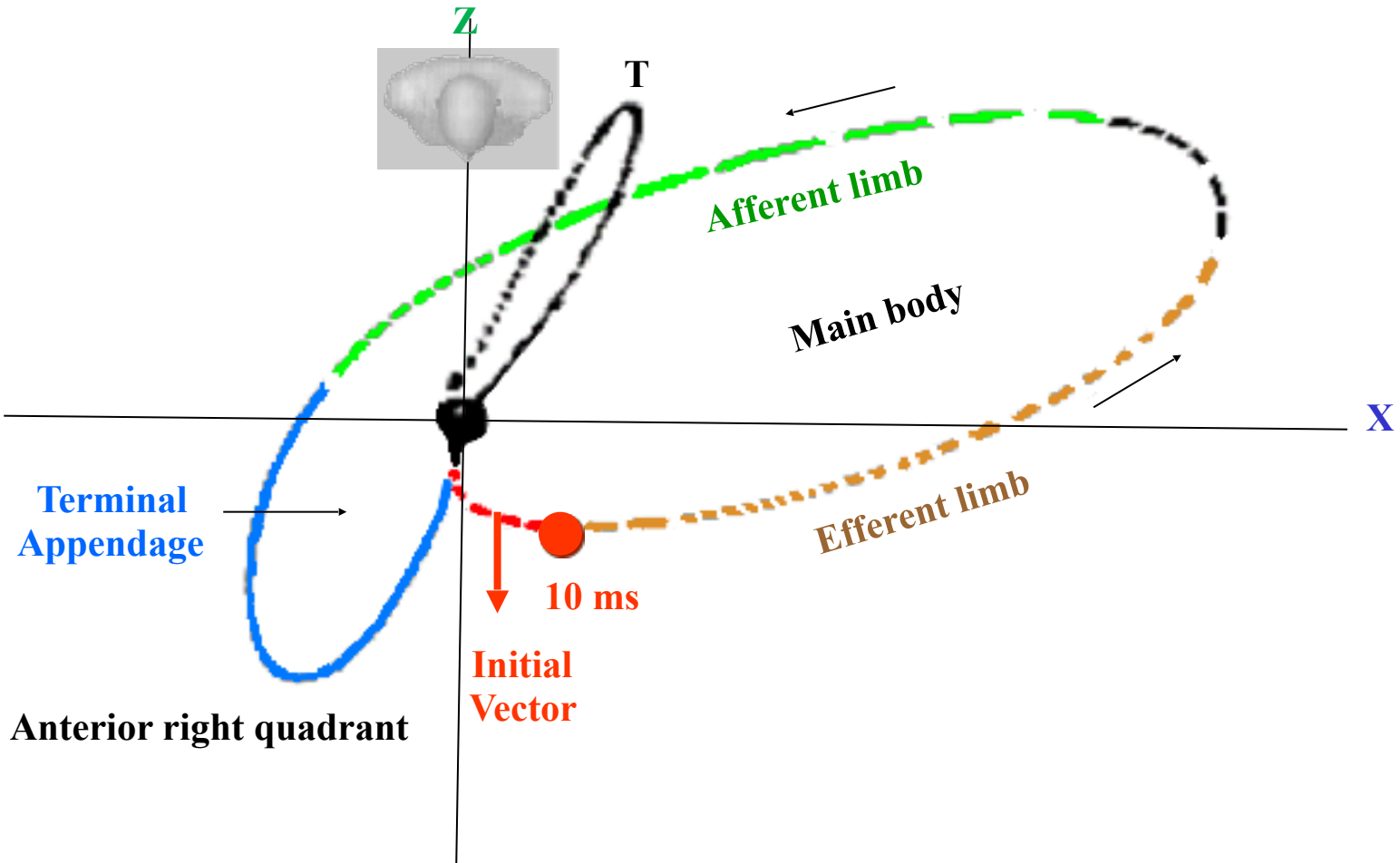
Note: The numbers are expressed in miliseconds

Electrocardiographic criteria of isolated Complete Right Bundle Branch Block (Surawicz 2009)

1) Supraventricular cardiac command
2) QRS duration ≥120 ms (or 0.12 s) in adults, greater than 100 ms in children 4 to 16 years of age, and greater than 90 ms in children younger than 4 years of age.
3) If the rhythm is sinus, PR ≥120 ms (0.12 s);
4) SAQRS in the frontal plane is variable; however, frequently shifted to the right and below;
5) Right precordial leads (V3R, V1 or V1 and V2) with rsr´, rSR' or rsR' pattern. The R´or r´ deflection is usually wider than the initial R wave. The final R' wave wide and sometimes notched: triphasic QRS complex called "M complex". In a minority of cases, a wide an often notched pure R wave pattern may be seen in lead V1 and/or V2. When a pure dominant R wave with or without a notch is present in V1, R-peak time ≥50 ms in lead V1 is present.
6) QR or Qr pattern in aVR lead with wide final R wave followed by negative T wave.
7) S wave of greater duration than R wave or grater than 40 ms in leads I and V6 in adults.
8) A delay in the appearance of the ventricular activation time or "R-peak time intrinsicoid deflection >50 to 80 ms (**Lerecouvreux 2005**) may also be observed in the right precordial leads and normal in V5-V6.
9) Ventricular repolarization (ST/T) with opposite direction to the terminal deflection of the QRS complex: T wave polarity opposite to the polarity of the last deflection of QRS complex.

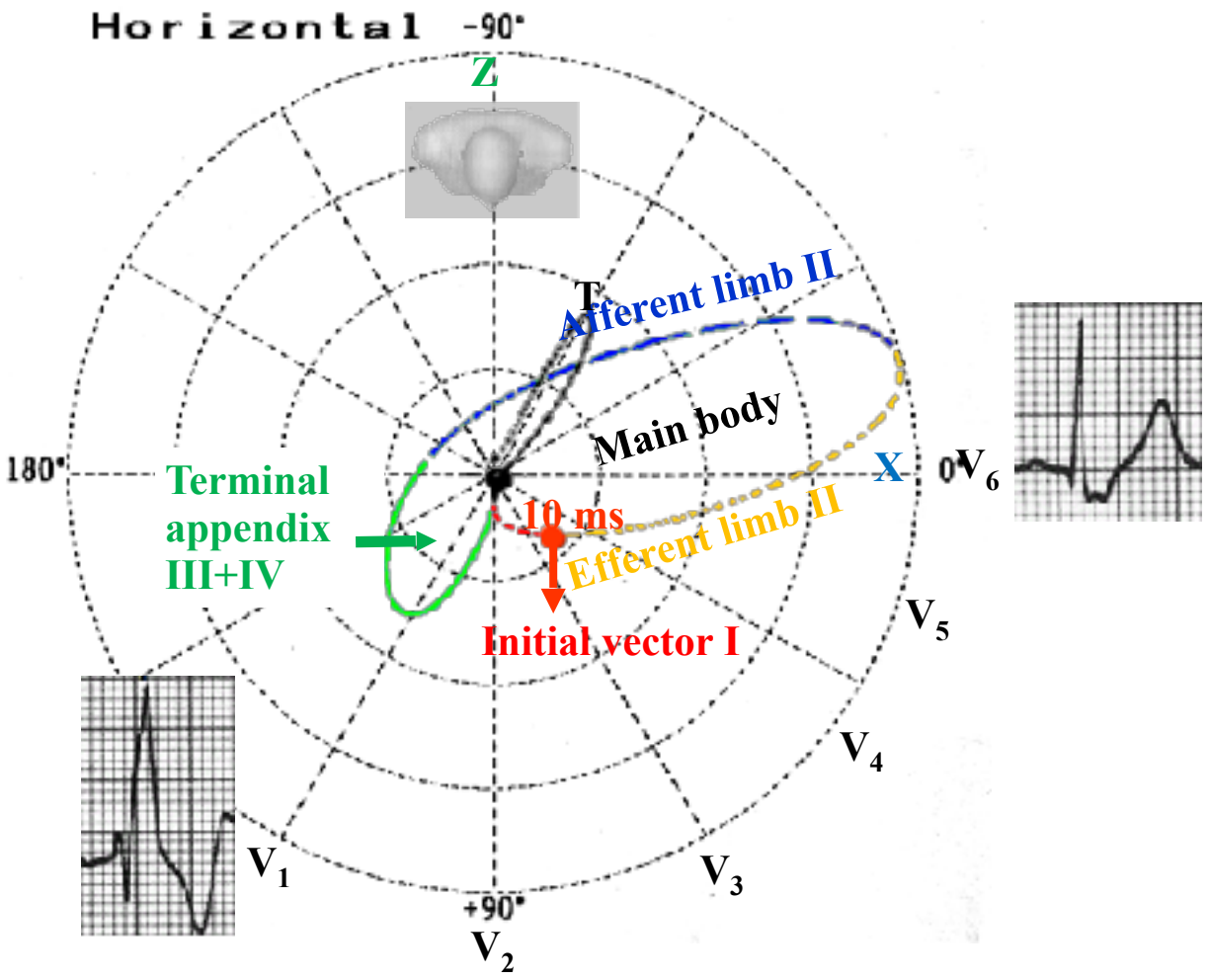
Vectorcardiographic criteria of uncomplicated Complete Right Bundle Branch Block in the Horizontal Plane

1. QRS loop of duration ≥120 ms. This corresponds to 60 or more dashes, in the cases in which 1 dash = 2 ms;
2. Maximal vector discretely decreased, pointing to the left and with a variable degree of anterior dislocation;
3. QRS loop in the HP formed by four components of depolarization: initial 10 to 20 ms vector, efferent limb, afferent limb, main body and terminal appendage after 60 ms with delay (slow recording) always located in the anterior right quadrant. These 4 components are followed by a repolarization component: the T wave heading to the back and the left (**Baydar 1965; Massing 1972; Rodriguez 1952**).



	Grishman VCG pattern or I	Cabrera or II	Kennedy III or C
Initial vector	To front and rightward or to the left	To front and rightward or to the left	To front and rightward or to the left
QRS duration	≥120 ms (≥60 dashes)	≥120 ms (≥60 dashes)	≥120 ms (≥60 dashes)
QRS-loop rotation	CCW	In 8	CW
Efferent limb	To front orthogonal X lead	To front orthogonal X lead	To front orthogonal X lead
Afferent limb	Behind orthogonal X	To front orthogonal X lead	To front orthogonal X lead
Terminal appendix	Right Anterior Quadrant and slow inscription	Right Anterior Quadrant and slow inscription	Right Anterior Quadrant and slow inscription
Maximal vector	Decreased and with mild anterior displacement	Decreased and with moderate anterior displacement	Decreased and with significant anterior displacement
Clinical significance	Absence of RVH: normal, hypertension, AoS or PS	Eventually RVH: ASD, OS, cor pulmonale, BVH (VSD, cardiomyopathy, rheumatic heart disease)	Severe RVH
T loop in the HP	CW rotation and directed to back and leftward	CW rotation and directed to back and leftward	CW rotation and directed to back and leftward

The four components of the QRS loop in right bundle branch block



Initial vector I

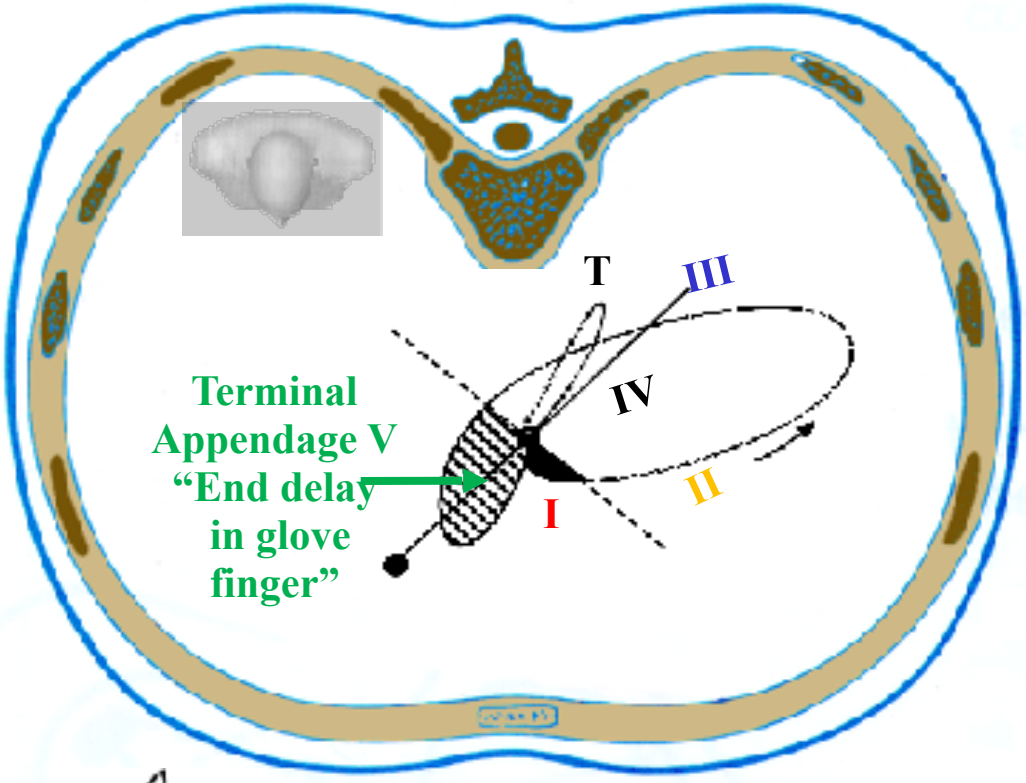
Efferent limb II

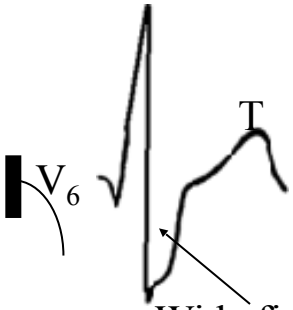
Afferent limb II

Terminal appendix III+IV

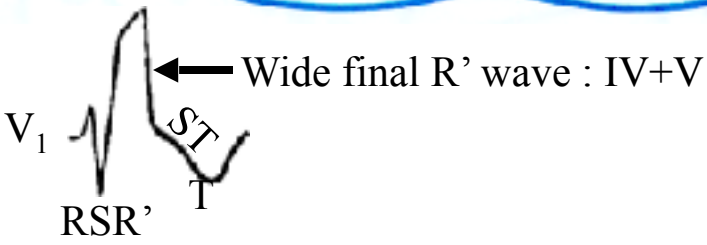
Right precordial leads (V_3R,V_1 or V_1 and V_2) rSR' type or rsR' or with broad R' wave and eventually with notch: triphasic QRS complex called "M" complex.
Left precordial leads with final wide S wave.

ECG/ VCG correlation in the HP with ECG in CRBBB





Wide final S wave IV+V



I) Vector of initial 10 ms;
II) Efferent limb;
III) Afferent limb;
IV) Main body;
V) Final appendage.

QRS/T loops in the horizontal plane showing the 4 components of depolarization: initial vector, efferent limb, afferent limb and terminal appendage and T wave heading to the back and the left, and its correlation with the QRS/ST-T complex in V1 and V6.

Prognosis of RBBB

The experience with bundle branch block at the USAF School of Aerospace Medicine was reviewed by Rotman et al (**Rotman 1975**). The clinical and follow-up status was evaluated in 394 subjects with RBBB and 125 subjects with LBBB. The majority of subjects were asymptomatic at the time of bundle branch block diagnosis. The subjects were divided into subgroups based on ECG findings to determine if any one subgroup was at higher risk for initial or follow-up morbidity of cardiovascular disease or follow-up mortality. At initial diagnosis and clinical evaluation, 94% of RBBB and 89% of LBBB subjects had no evidence of cardiovascular disease. In the RBBB group, 3 and 2% had CHD and hypertension, respectively; in LBBB subjects, 9 and 7% had CHD and hypertension, respectively. No one ECG subgroup in either the RBBB or LBBB group had a higher incidence of cardiovascular disease. Complete follow-up information was available in 94% of the RBBB subjects and 91% of the LBBB subjects. The mean follow-up period was 10.8 ± 4.7 years in the RBBB group and 8.8 4.8 in the LBBB group. In the follow-up period, new cases of CHD and hypertension occurred in 6% of the RBBB group and 5 and 8%, respectively, in the LBBB group. Fourteen (4%) RBBB and nine (8%) LBBB subjects died during the follow-up period. No differences for follow-up morbidity of cardiovascular disease or mortality were observed in contrasting the individual ECG subgroups. Progressive electrical dysfunction in the form of complete heart block occurred in one subject each in the RBBB and LBBB groups. Thus the prognosis of bundle branch block is determined by the presence or absence, and degree of associated cardiovascular disease.

Kusomoto et al (Kusomoto 2014) investigated the clinical course of complete RBBB or RBBB with axis deviation in terms of subsequent pacemaker implantation for high-degree AV block or sick sinus syndrome. Among the 16,170 atomic-bomb survivors in biennial health examination between July 1967 and December 2010, the authors detected 520 newly-acquired RBBB subjects with no organic heart disease, and selected 1038 age-and sex-matched subjects without RBBB to serve as comparison subjects. Multivariate Cox regression analysis was used to estimate the hazard ratios (HRs) for the risk of pacemaker implantation due to all causes, AV block or sick sinus syndrome between RBBB and comparison subjects and between RBBB subjects with and without axis deviation.

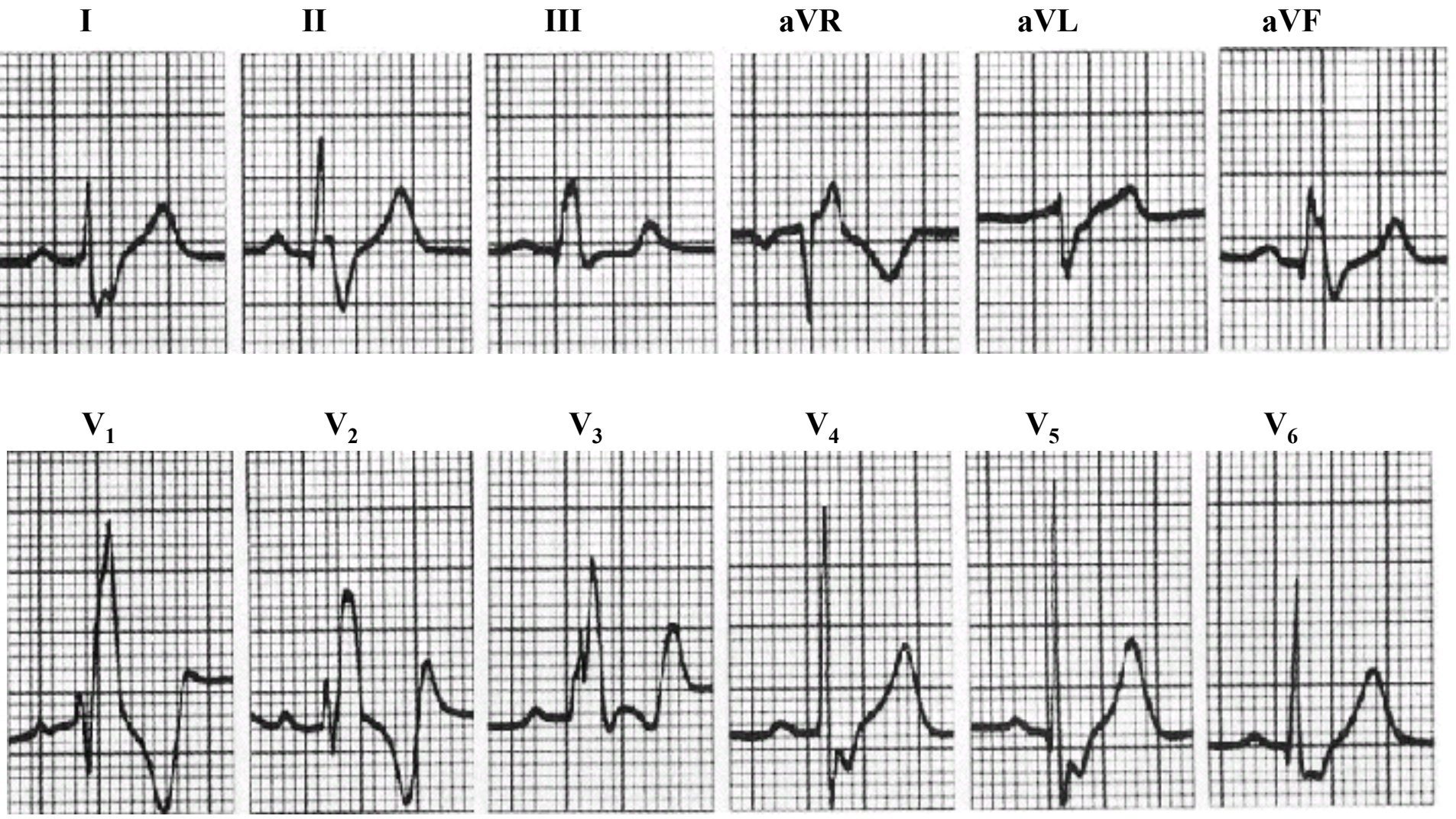
The risk of pacemaker implantation for RBBB was 4.79 (95% confidence interval [CI] 1.89-12.58; P=0.001), 3.77 (95% CI, 1.09-13.07; P=0.036), and 6.28 (95% CI, 1.24-31.73, P=0.026) when implantation was for all causes, AV block and sick sinus syndrome, respectively. RBBB subjects with axis deviation had a higher risk for all-cause pacemaker implantation than subjects without axis deviation. RBBB subjects with axis deviation were younger than subjects without axis deviation at the time of RBBB diagnosis, and their progression from diagnosis to pacemaker implantation took longer. RBBB, especially with axis deviation, progresses to AV block and sick sinus syndrome that requires pacemaker implantation; the mechanisms by which the conduction defect progresses differ among patients with and without axis deviation.

Bussink et al (**Bussink 2013**) followed 18,441 participants included in the Copenhagen City Heart Study examined in 1976-2003, free from previous myocardial infarction, chronic heart failure, and LBBB through registry linkage until 2009 for all-cause mortality and cardiovascular outcomes. In this cohort study, RBBB and IRBBB were two to three times more common among men than women and CRBBB was associated with increased cardiovascular risk and all-cause mortality, whereas IRBBB was not. Contrary to common perception, RBBB in asymptomatic individuals should alert clinicians to cardiovascular risk.

Patients with AMI complicated with bundle branch block (BBB) have a poor prognosis, but distinction between LBBB and RBBB is seldom made in epidemiological studies. Lewinter et al (Lewinter 2011) studied long-term mortality associated with RBBB and LBBB in the Trandolapril Cardiac Evaluation (TRACE) trial. The authors studied consecutive patients presenting with MI and recorded clinical, electro- and echo-cardiographic variables. Subsequently, deaths were recorded during a minimum follow-up of 15 years. In total, 6,676 consecutive patients with MI were hospitalized at 27 centres in Denmark. Of these 4% had RBBB and 4% had LBBB. Overall, 5,196 (78%) patients died, 256 (94%) with LBBB and 235 (90%) with RBBB compared with 4,705 (77%) of those without BBB. In multivariable analyses, hazard ratios of RBBB and LBBB were 1.23 and 1.05 respectively. There was interaction between each type of BBB and LV systolic function. RBBB was associated with a worse prognosis in patients with reduced LV systolic function while LBBB had a poor prognosis in patients with preserved LV systolic function. RBBB was a predictor of increased mortality only in patients with reduced LV systolic function, whereas LBBB was a marker of increased mortality in patients with preserved LV systolic function.

Kleemann et al **(Klemannn 2008)** evaluated the incidence and clinical impact of RBBB in patients with NSTEMI compared to patients with STEMI. Both LBBB and RBBB have been associated with increased inhospital and long-term mortality in patients with STEMI. However, the prognostic role of RBBB in NSTEMI is not well known. From the German prospective multicenter registry "Maximal Individual Therapy of Acute Myocardial Infarction" (MITRA PLUS), 6,403 consecutive patients with NSTEMI and 20,233 patients with STEMI were analyzed. Patients with LBBB were excluded. The median follow-up time for NSTEMI was 378 days and for STEMI 479 days. A total of 455 (7.1%) patients with NSTEMI and 894 (4.4%) patients with STEMI presented with RBBB on admission. In general, RBBB patients were older, more often had comorbidities, and less often received short-term inhospital treatment according to guidelines. In STEMI, RBBB patients had higher peak enzyme levels and lower LVEF than patients without BBB. RBBB in STEMI was associated with an increased inhospital and long-term mortality. In NSTEMI, however, peak enzyme levels and LVEF were similar in both groups with and without RBBB. RBBB in NSTEMI was not independently associated with a worse outcome. Unlike RBBB in STEMI, RBBB in NSTEMI is not an independent predictor of inhospital and long-term mortality.

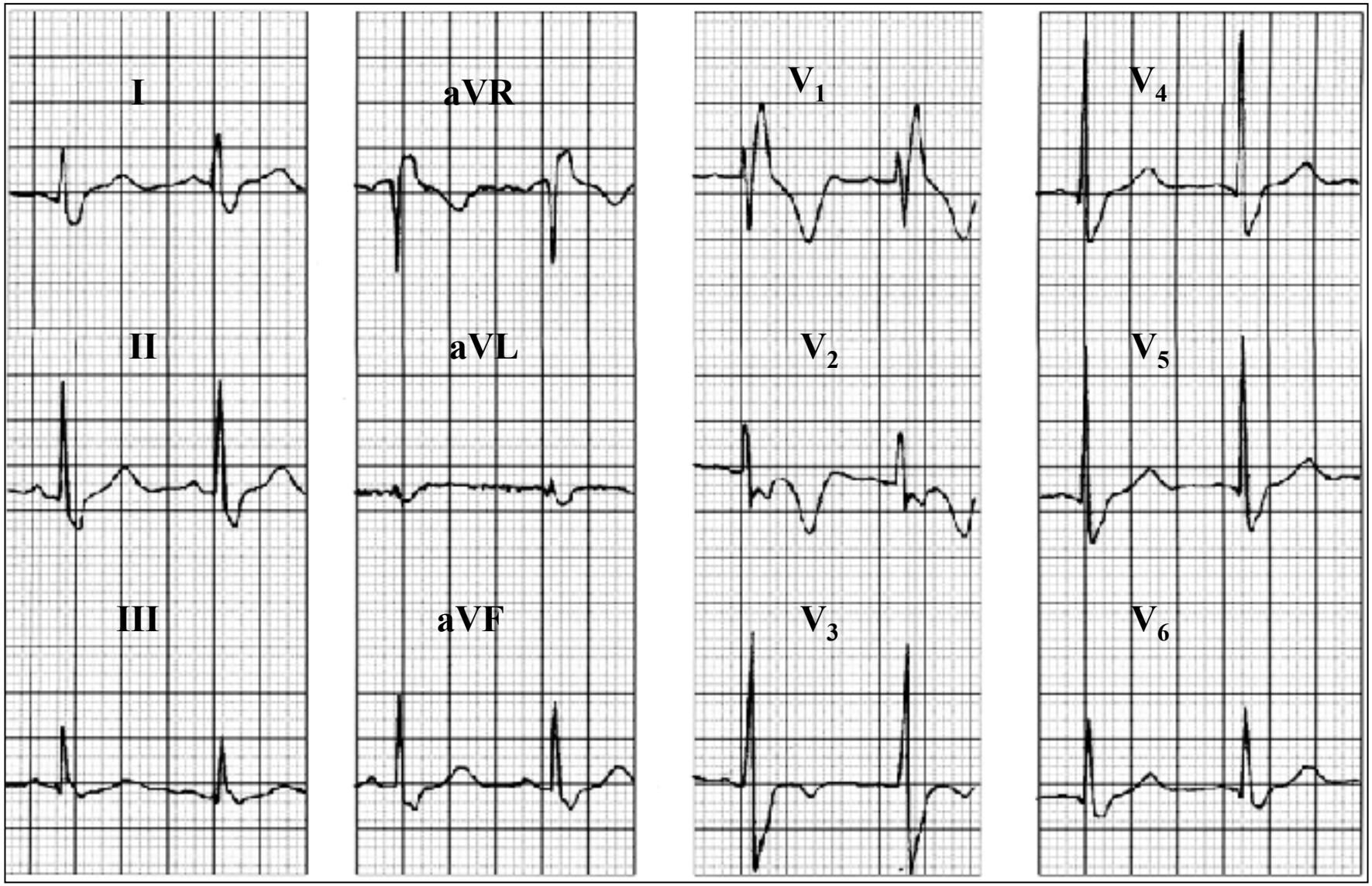
Examples of RBBB



Clinical diagnosis: 36 years, female, high blood pressure.

ECG diagnosis: Sinus rhythm SAP + 60º and to the front; PR interval 160 ms; SÂQRS + 100º, QRS duration: 160 ms, wide S and with notch in I, aVL, V_5 and V_6, Complex in "M" with wide R' in V_1 and V_2. Ventricular repolarization (ST/T) with direction opposite to the terminal deflection of the QRS complex.

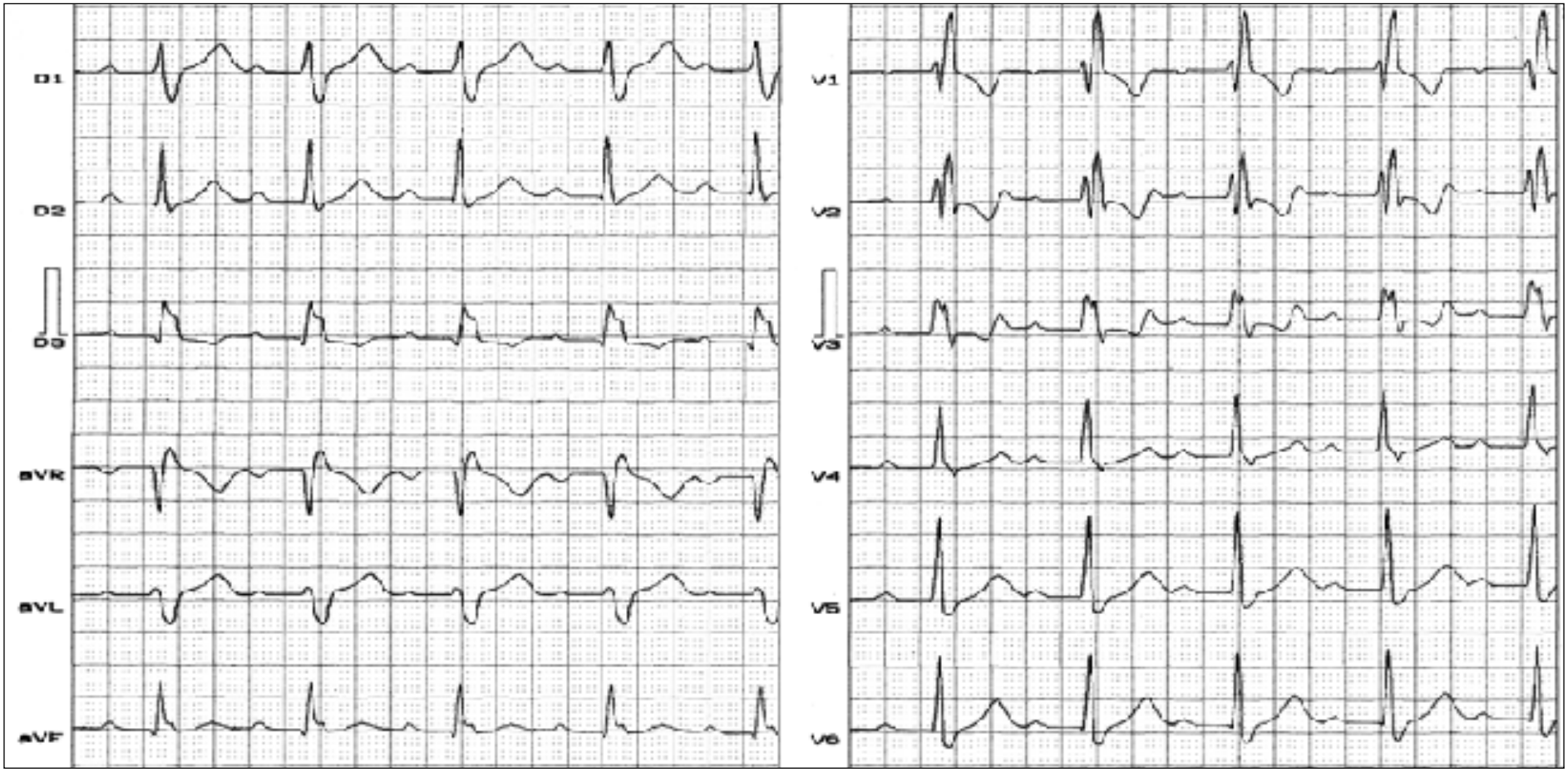
Conclusion: CRBBB



ECG diagnosis: Sinus rhythm; SÂP + 60º and to the front, PR 160 ms, SÂQRS + 80º QRS duration: 160 ms, wide S in I, aVL, V_5 and V_6 Complex in "M" with wide R' in V_1 and V_2. Ventricular repolarization (ST/T) with a direction opposite to the terminal deflection of the QRS complex.

Conclusion: CRBBB

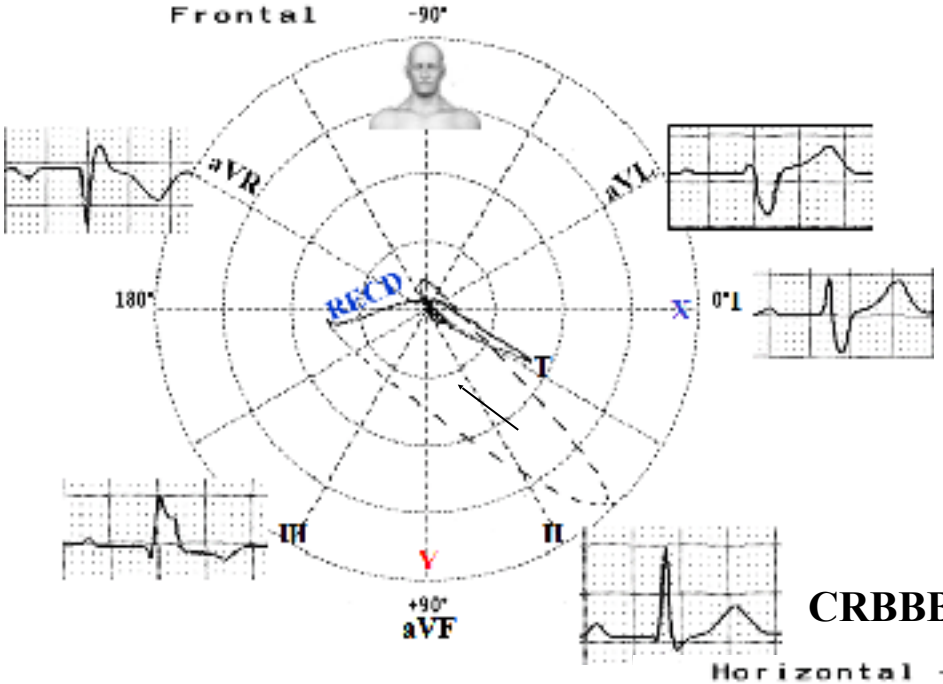
Name: RFC; **Date:** 14/05/1996; **Age:** 32 y.o.; **Gender:** F.; **Race:** W.; **Weight:** 56 Kg; **Height:** 1.65 m; **Biotype:** Athletic



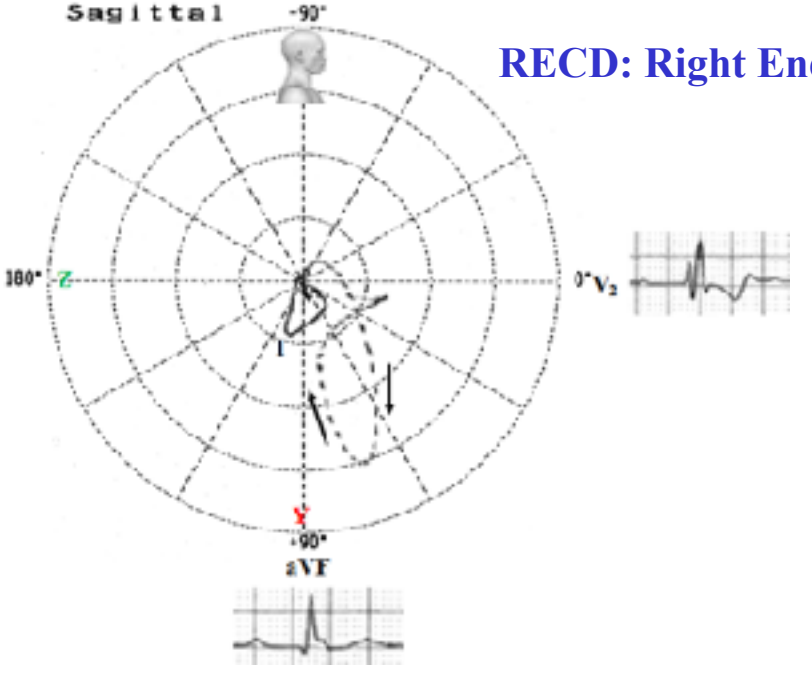
Clinical diagnosis: Steinert's Myotonic Dystrophy.

ECG diagnosis: First degree AV block (PR 28 ms) + CRBBB. This situation does not enable to make the diagnosis for trifascicular block. Only the electrophysiology study revealed CRBBB and dromotropic disorder equivalent in the two divisions of the LB (trifascicular). There were normal PA and AH intervals (40 and 70 ms respectively) with prolonged HV (75 ms), indicating that the dromotropic disorder was infra-His. The CRBBB morphology associated to long PR, indicates the dromotropic difficulty in the left divisions (infra-His long PR).

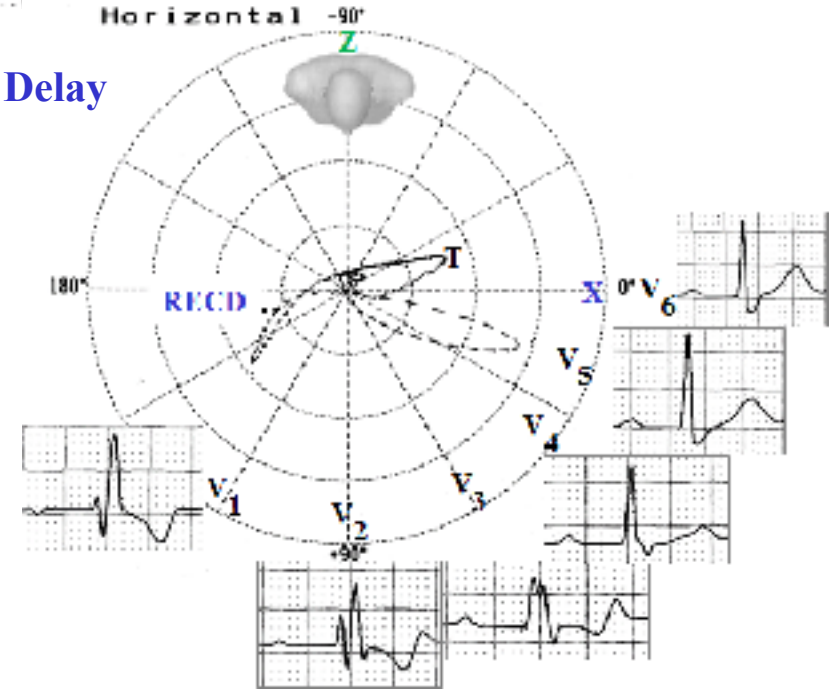
ECG/VCG correlation



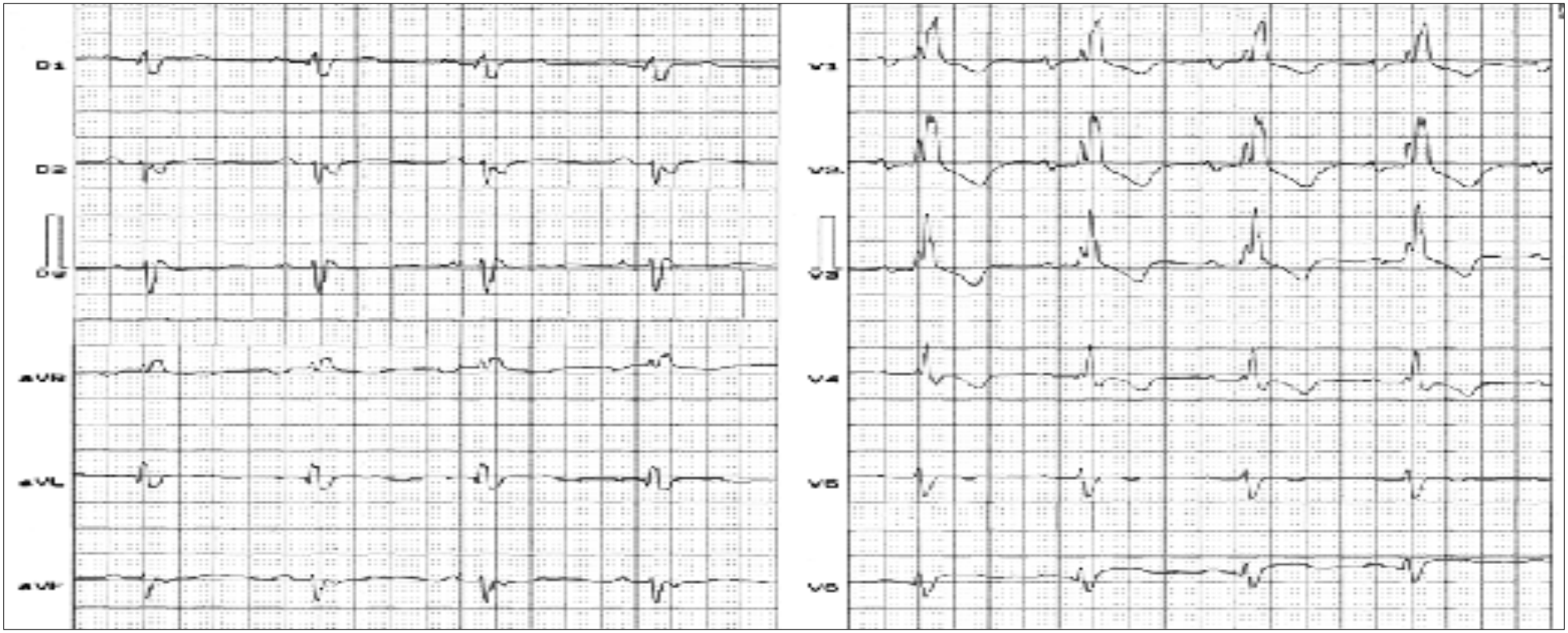
CRBBB Grishman type



RECD: Right End Conduction Delay

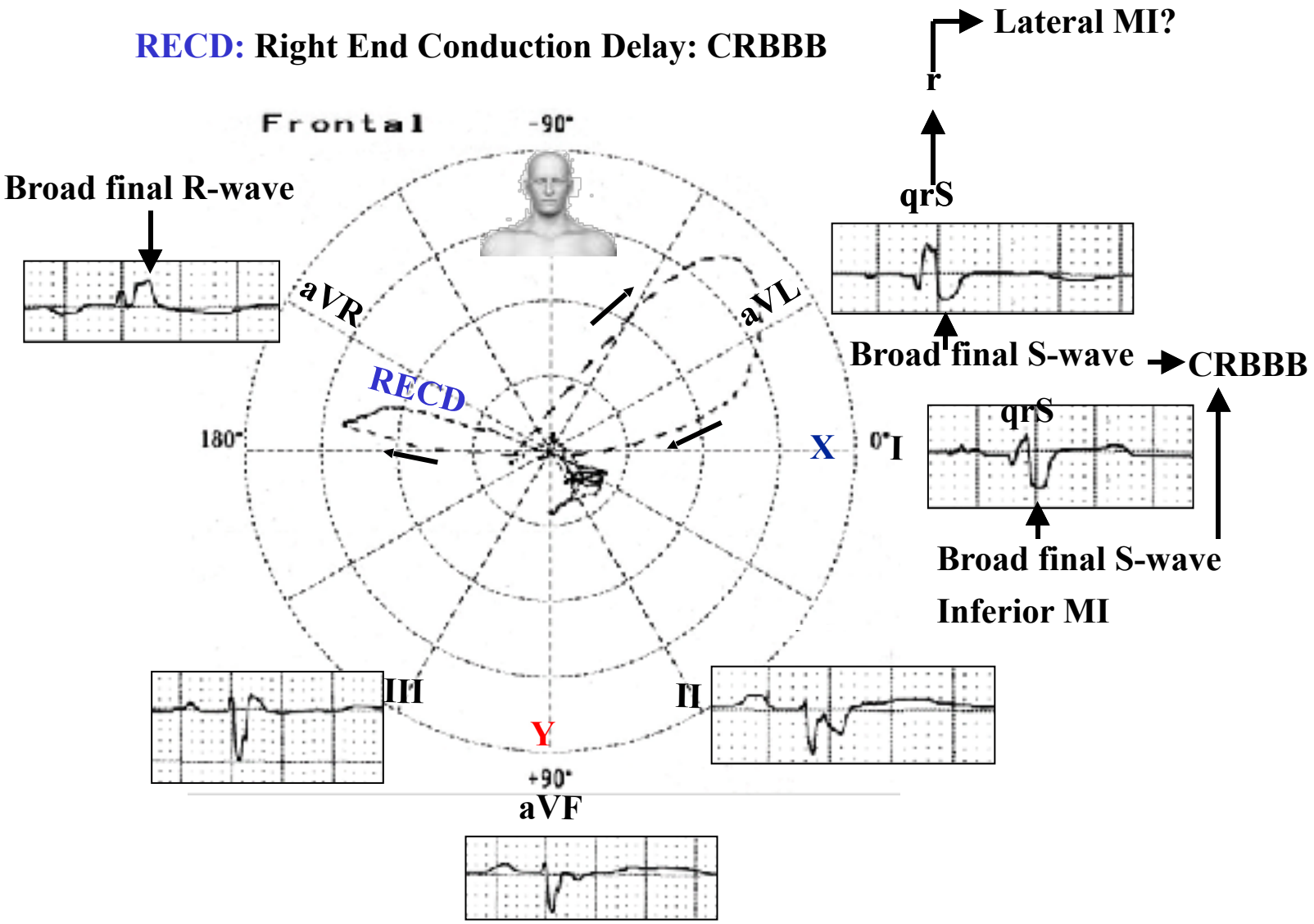


Name: CFS; **Gender:** male.; **Age:** 45 y.o.; **Race:** white; **Weight:** 72 Kg; **Height:** 1.70 m; **Biotype:** athletic; **Date:** 12/11/2003; **Medication in use:** Digoxin 0.25 mg, Furosemide 40 mg, Spironolactone 25 mg, Enalapril 20 mg.

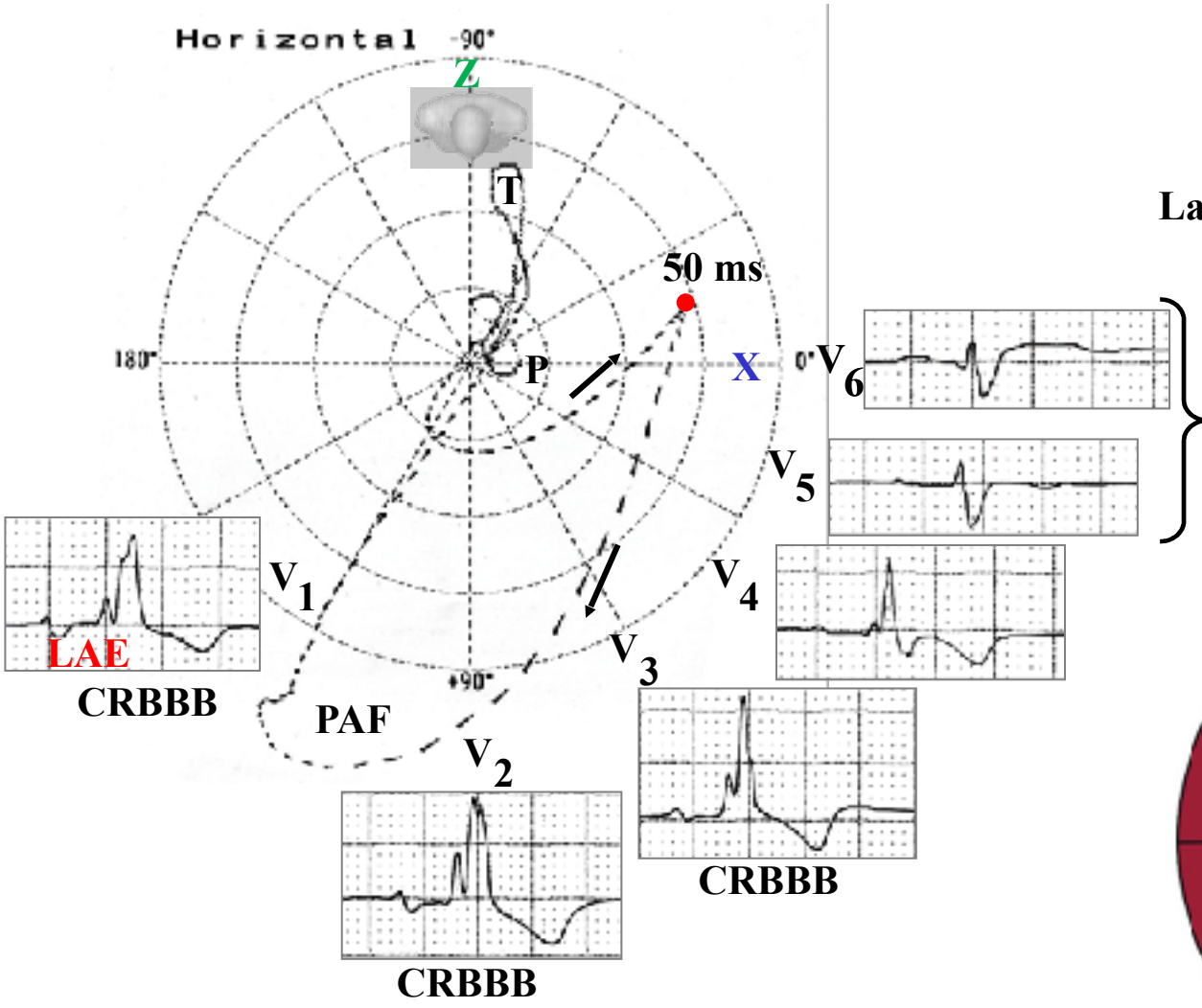


Clinical diagnosis: Chronic Chagas cardiomyopathy, mixed form, dilated and dromotropic.
Echocardiogram: Dilated Cardiomyopathy of moderate repercussion. Diffuse hypokinesis with infero-laterobasal predominance (ancient dorsal). Normal RV.
ECG diagnosis: Sinus rhythm; HR: 65 bpm; P wave duration: 110 ms; SÂP +70º bimodal notched aspect in I; slow final negative component in V1: LAE. The presence of LAE criteria of 1st degree AV block; QRS–SÂQRS with extreme deviation in the right superior quadrant; QRS duration: 160 ms; wide S wave in left leads; morphology in "M" from V1 to V4: CRBBB; PAF; R wave of low voltage in V5 and V6.
Conclusion: LAE or BAE? 1st degree AV block? CRBBB, PAF, infero-latero-dorsal electrically inactive area? RVE?

ECG/VCG correlation in the FP showing inferolateral MI and RECD of CRBBB

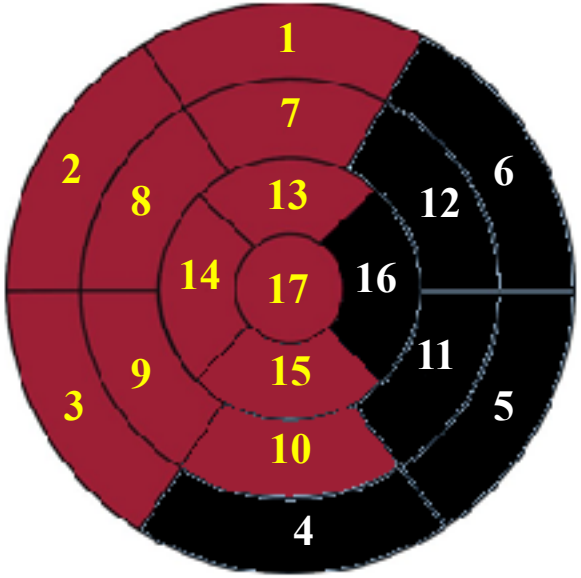


ECG/VCG correlation in the HP showing laterobasal MI and RECD of CRBBB



Lateral MI: 5, 6, 11, 12, 16

Low r-wave voltage

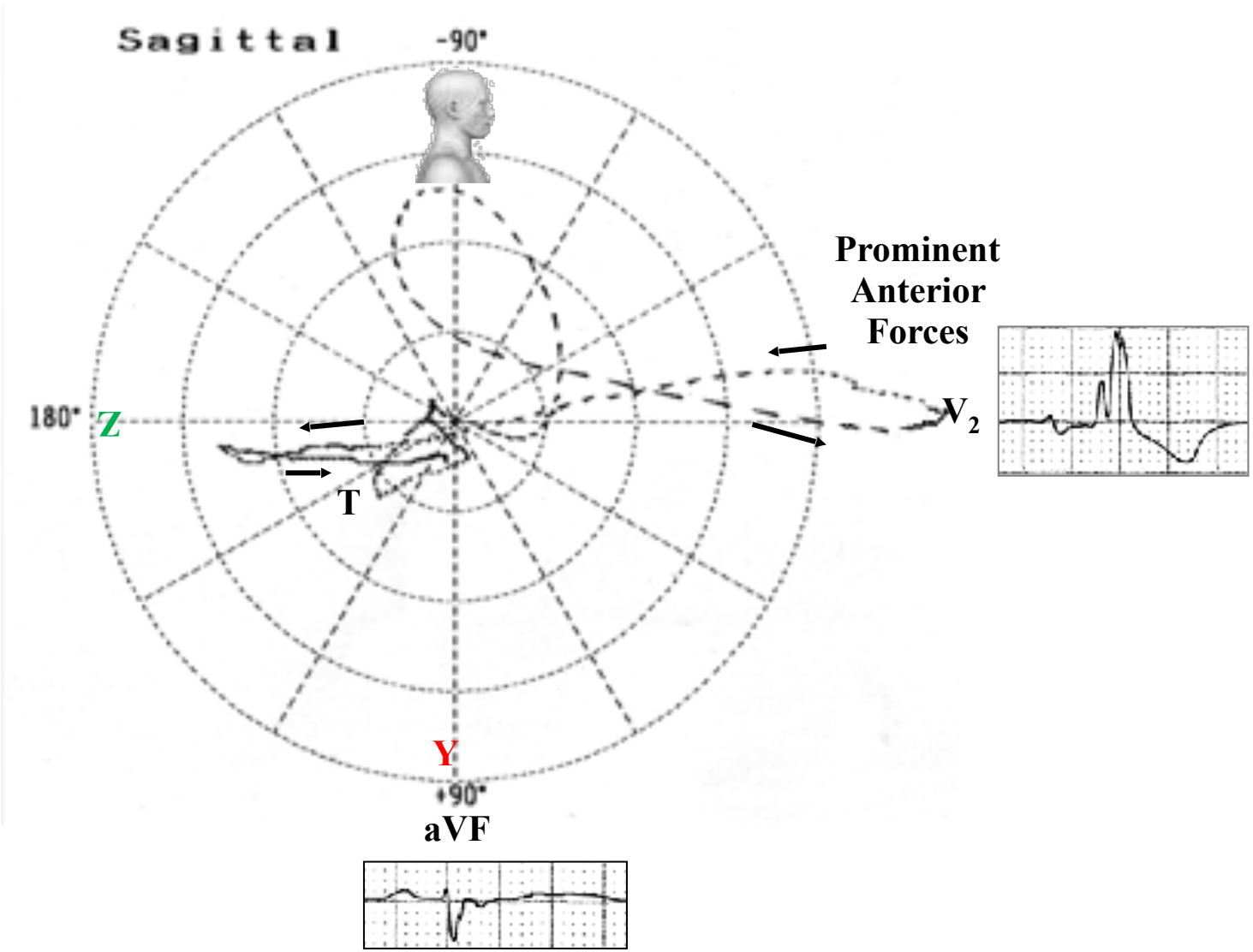


Black: Infarction area
4: Basal inferior (ancient dorsal)

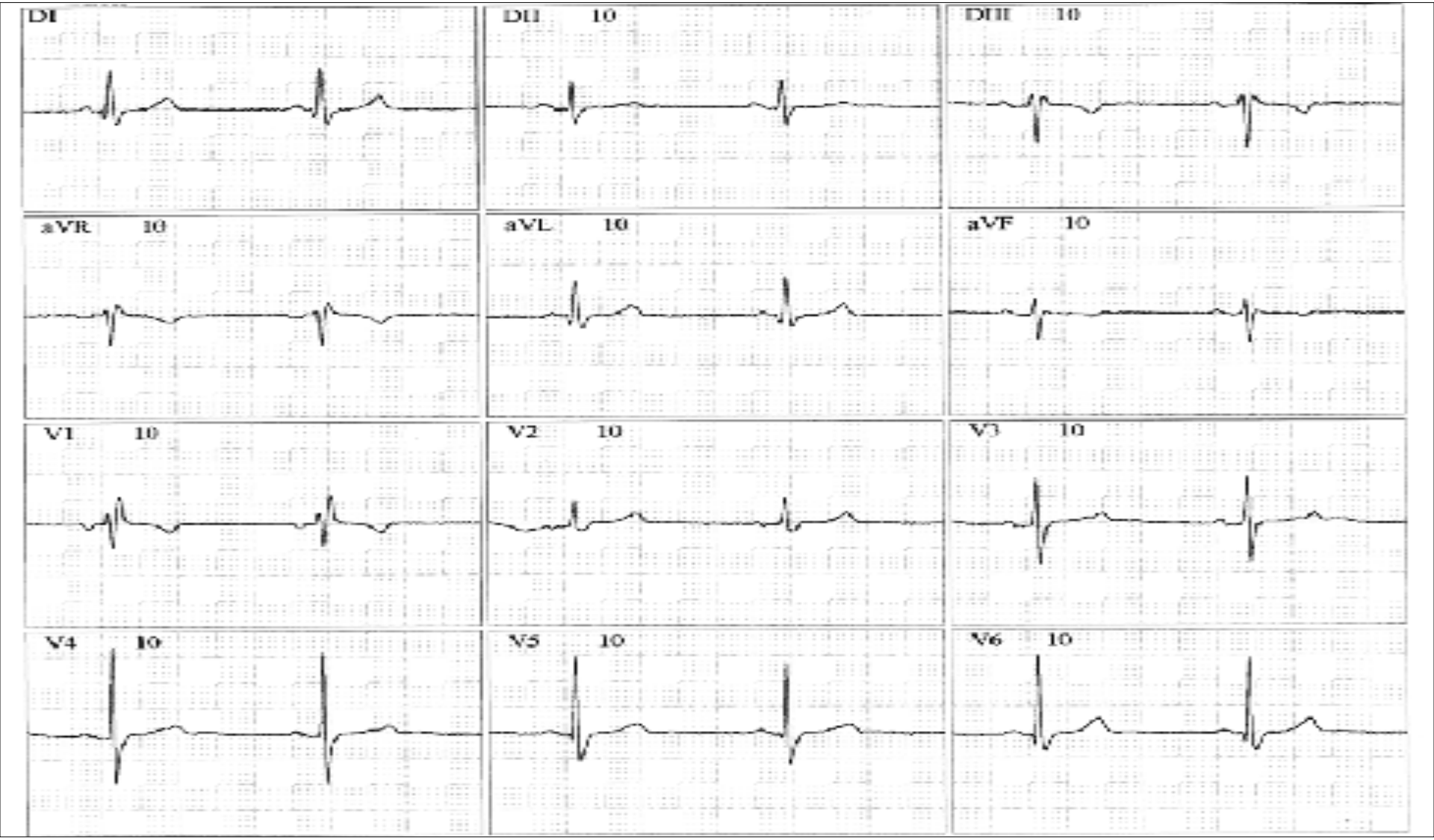
LAE: Left Atrial Enlargement

PAF: Prominent Anterior Forces: CRBBB + laterobasal MI (ancient dorsal MI nomenclature)

ECG/VCG correlation in the RSP

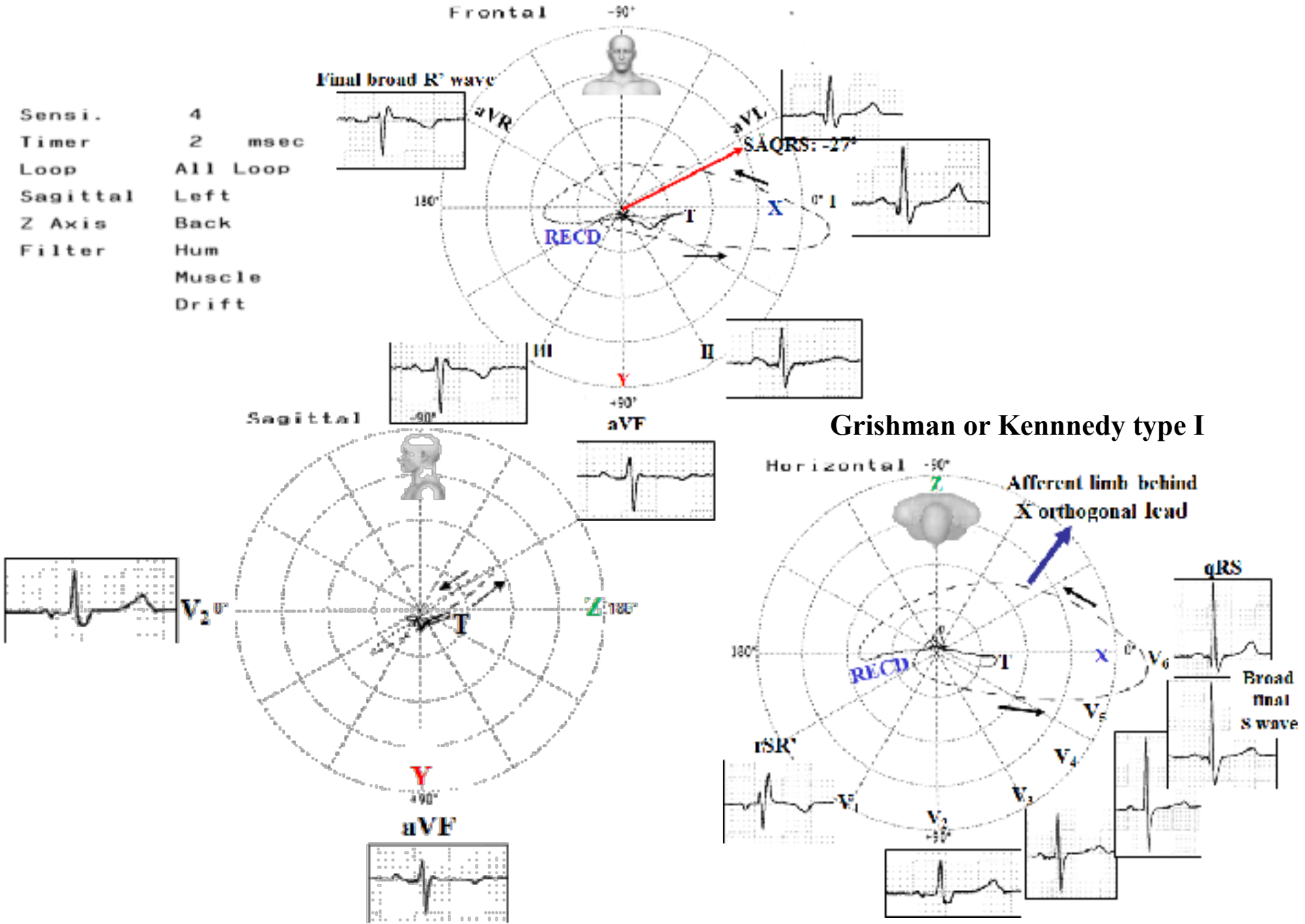


Name: JC; **Gender:** male; **Age:** 68 y.o.; **Race:** White; **Weight:** 66 Kg; **Height:** 1.70 m; **Biotype:** Athletic; **Date:** 14/09/2004**; Medication in use:** Isocord 40 mg 3X; ASA 2X; Enalapril 20 mg 2X; Chlortalidone 2.5 mg 1X; Atenolol 50 mg 1X;

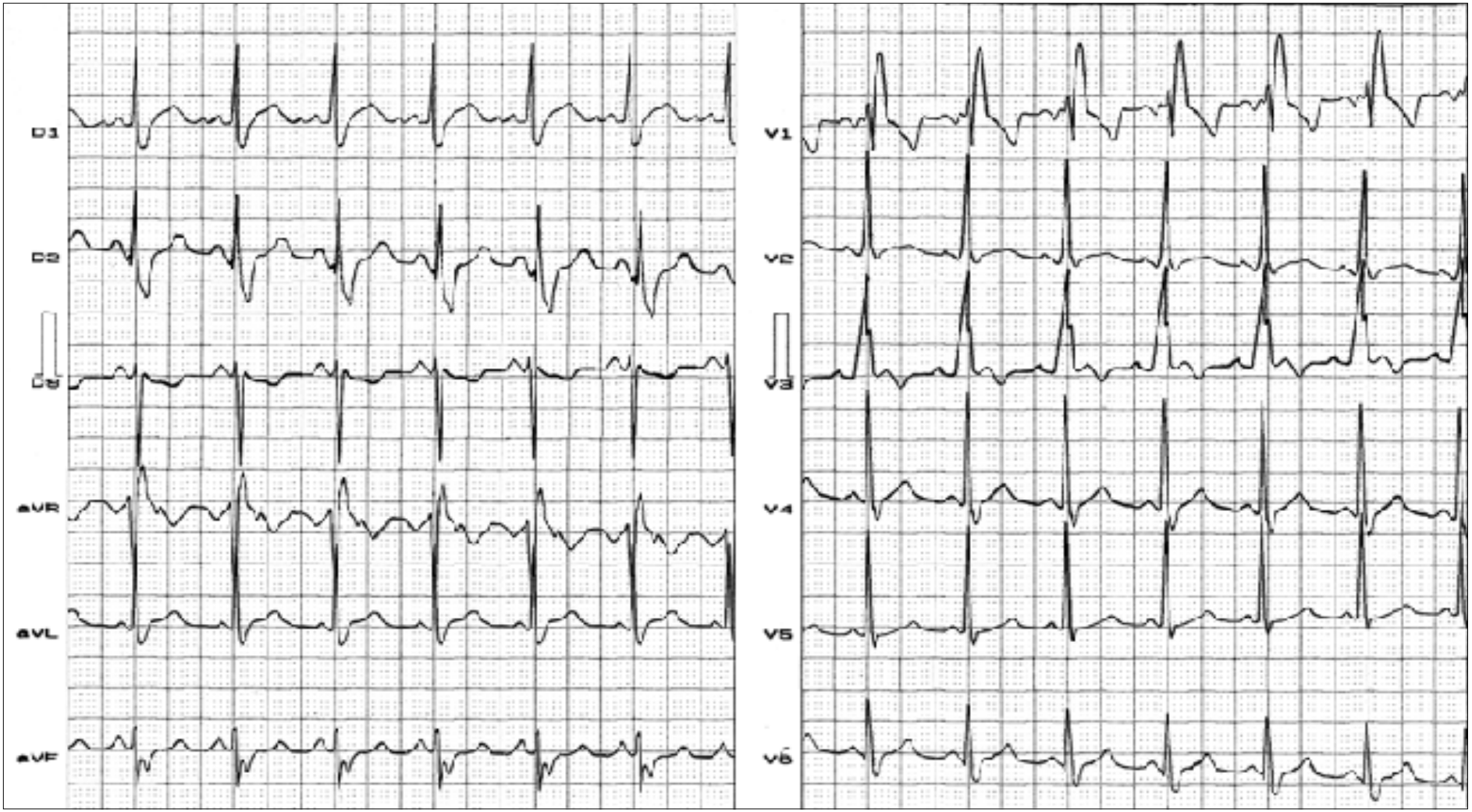


Clinical diagnosis: Systemic hypertension.
ECG diagnosis: SR; HR: 53 bpm. Sinus bradycardia. CRBBB. QRSD: 121 ms.

ECG/VCG correlation



Name: MLPA; **Age:** 40 y.o.; **Gender:** F.; **Race:** White**; Weight:** 87 Kg; **Height:** 1.63 m; **Biotype:** Normal;
Med. in use: Lisinopril 20 mg; Hydrochlorothiazide 25 mg; Amlodipine 5 mg

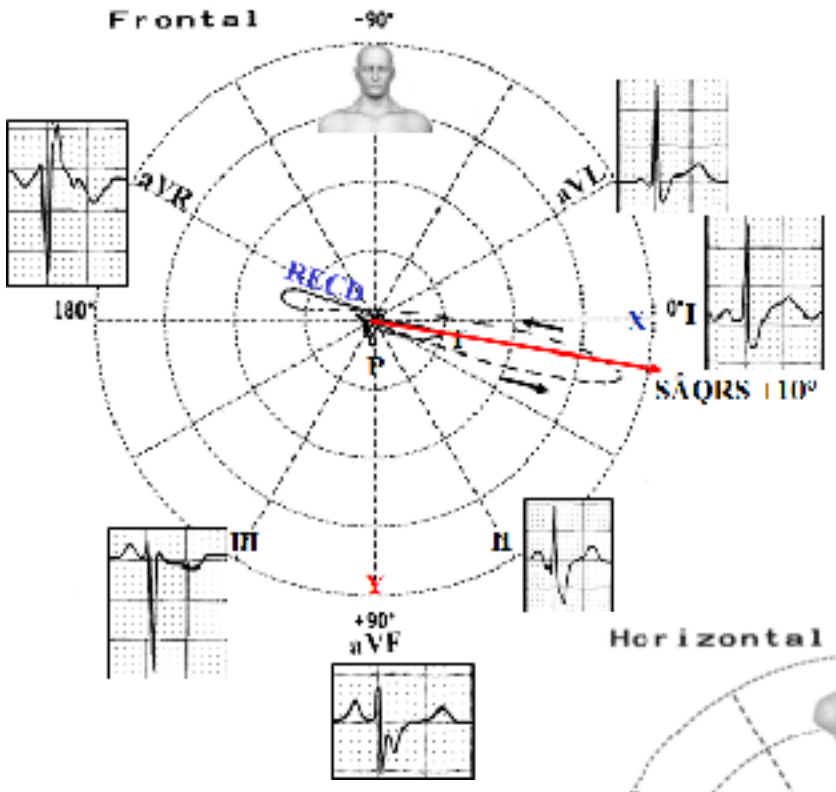


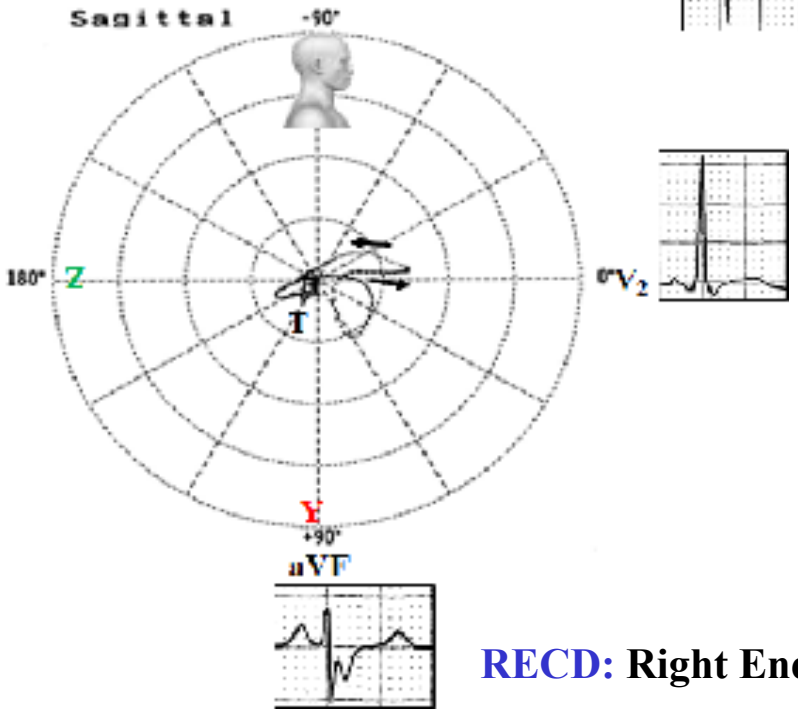
Clinical diagnosis: obesity and significant systemic hypertension.
ECG diagnosis: CRBBB and prominent anterior forces (PAF). R of great voltage in V2 and V3; it suggests counterclockwise rotation of the heart on its longitudinal axis. The fact may be observed in LV enlargement.
ECHO: septum and posterior wall of 16 mm: LV hypertrophy

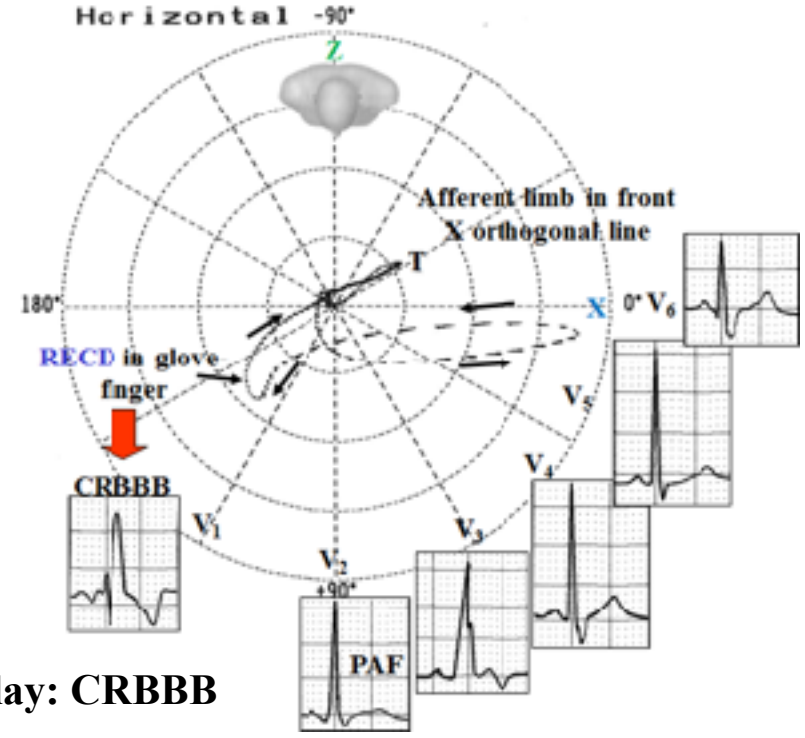
ECG of CRBBB in a hypertensive patient. Prominent anterior forces that suggest counterclockwise rotation in the longitudinal axis.

ECG/VCG correlation

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

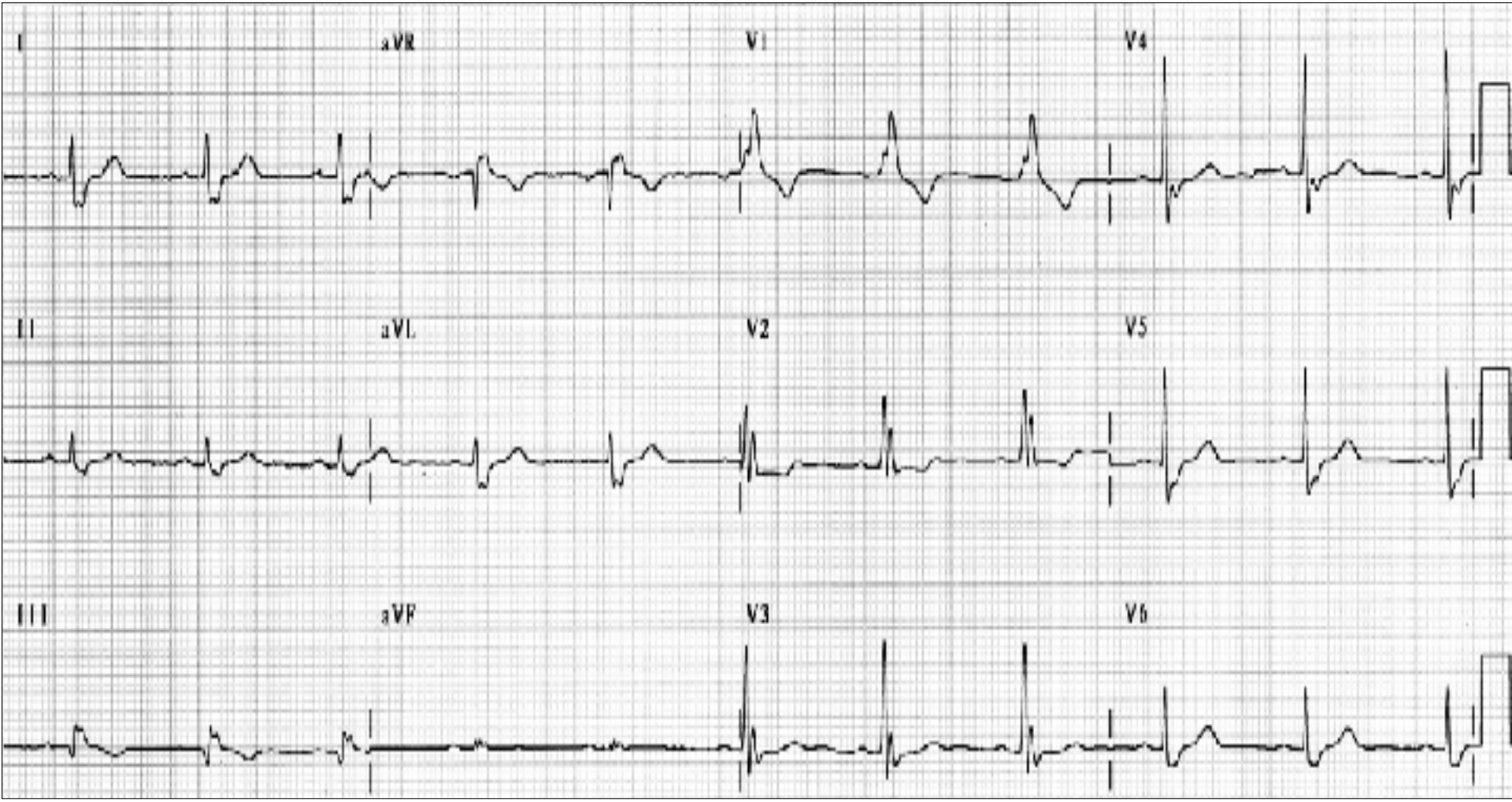






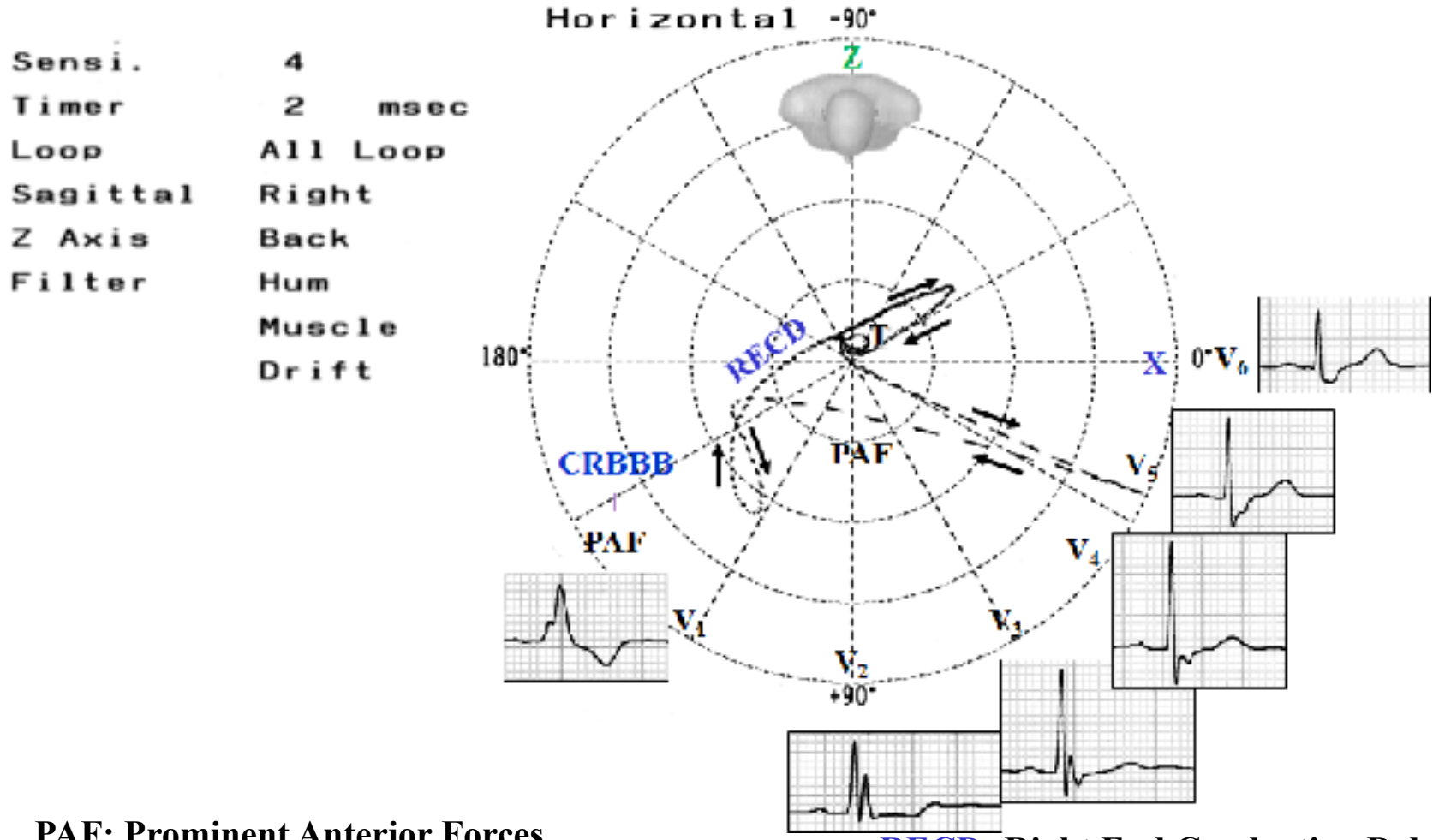
RECD: Right End Conduction Delay: CRBBB

Name: ARH; **Date:** 8/07/2003; **Age:** 73 yo.; **Number:** 507; **Gender:** M.; **Race:** W.; **Weight:** 68 Kg; **Height:** 1.65 m.; **Biotype:** Athletic



Clinical diagnosis: healthy patient. ECG performed in check up. Normal echocardiogram.
ECG diagnosis: sinus rhythm, HR: 63 bpm, P wave: SÂP +50º to the front, PR: 186 ms, SÂQRS between +91º to 269º and to the front (near +170º). QRS duration: 138 ms, morphology: broad S wave in I, aVL, V5 & V6, pure R wave with notch in ascending slope in V1, R wave with notch in V2 and Rsr's' in V3.
Conclusion: Complete Right Bundle Branch Block + Prominent Anterior Forces.

ECG/VCG correlation in the HP of CRBBB of VCG Kennedy type III or C

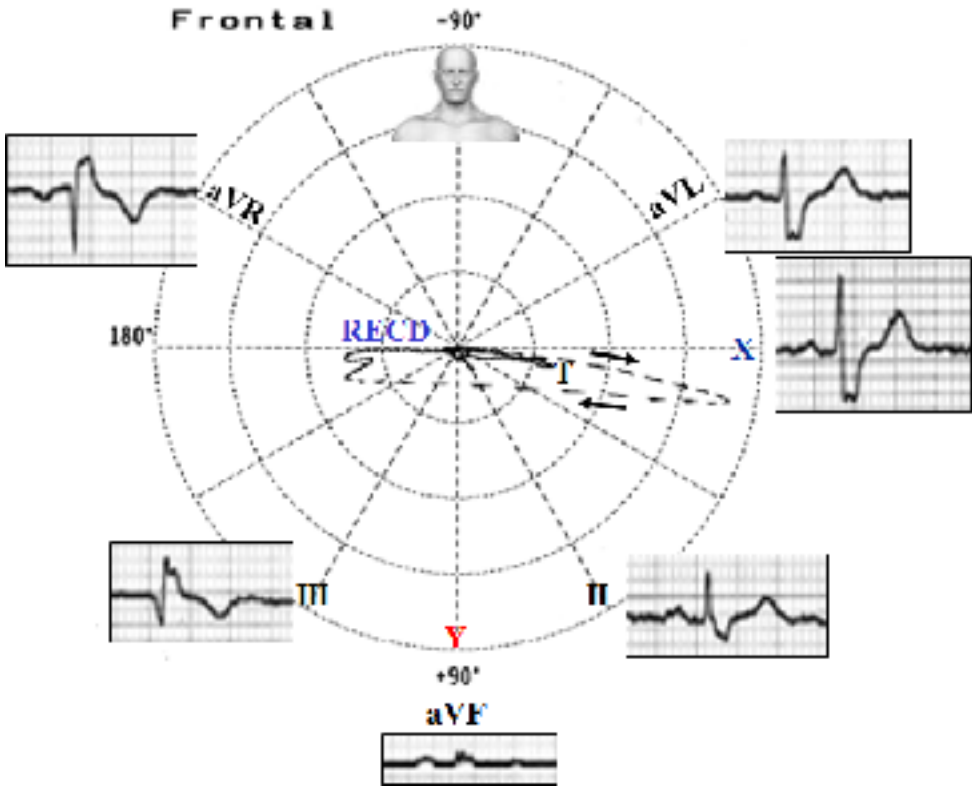


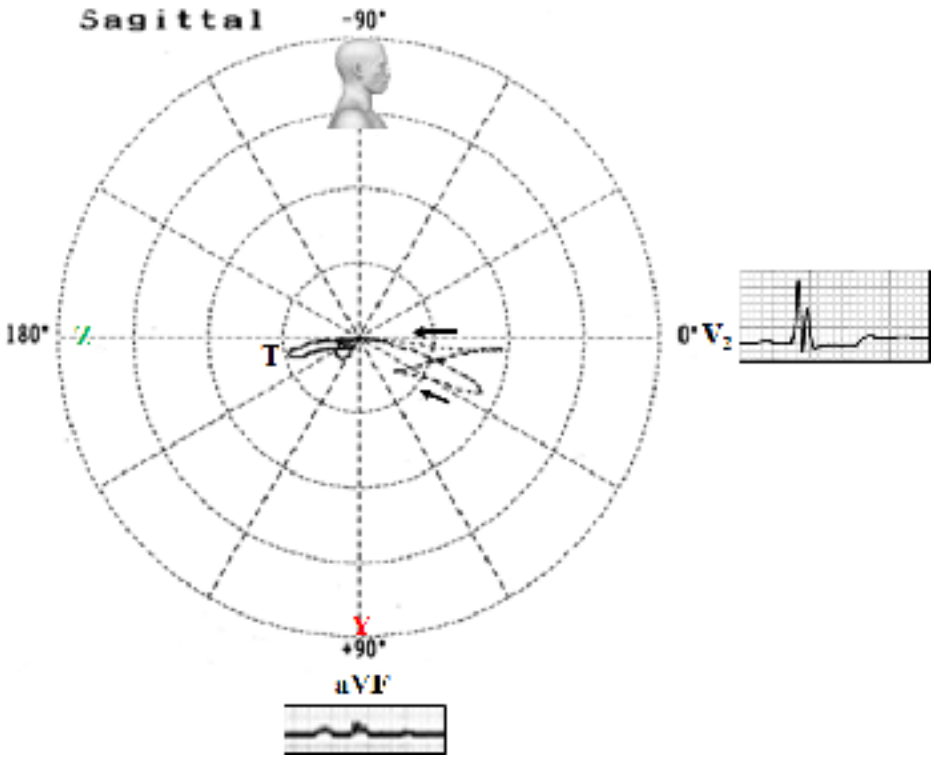
PAF: Prominent Anterior Forces

RECD: Right End Conduction Delay: CRBBB

ECG/VCG correlation in the frontal plane Kennedy type III. QRS loop totally dislocated in anterior quadrants and of clockwise rotation. In general, this type of loop usually means significant RVH, but it may correspond to normal cases like this one. Initial vector to the front, QRS loop of clockwise rotation, except for a minimal part (*) of end delay or VCG Kennedy type III or C, is more frequent in the presence of associated RVH; however it may be normal. Main body of the QRS loop located in anterior quadrants (in front of the x line)

ECG/VCG correlation in the FP and RSP





RECD: Right End Conduction Delay: CRBBB

Complete Right Bundle Branch Block associated to Right Ventricular Hypertrophy

The diagnosis of RVH in the presence of CRBBB by ECG criteria

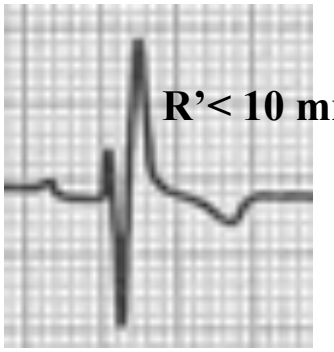
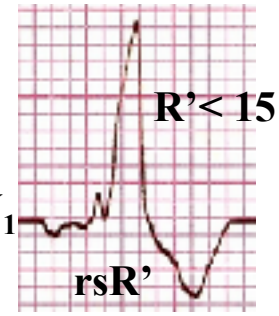
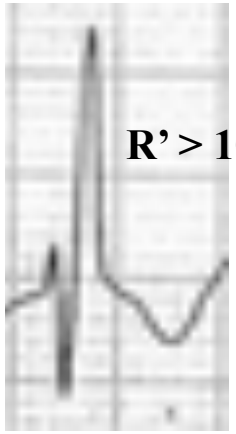
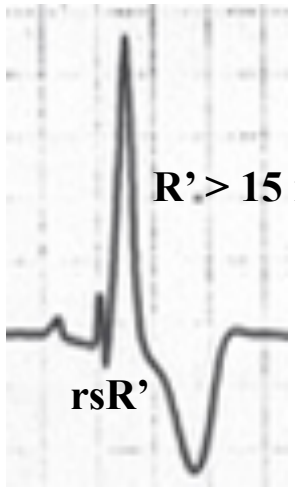
In the Frontal Plane

	Isolated CRBBB	CRBBB associated to RVH
I and aVL	qRS	rS
II- III- aVF	Variable.	QR; R or qR

In the precordial leads

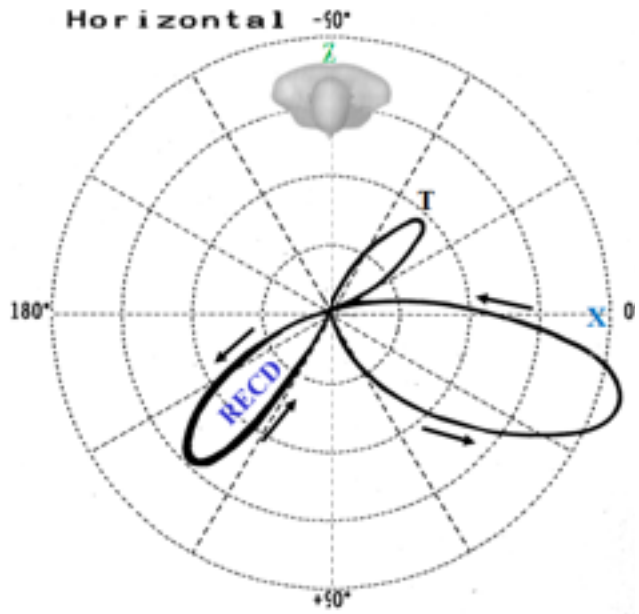
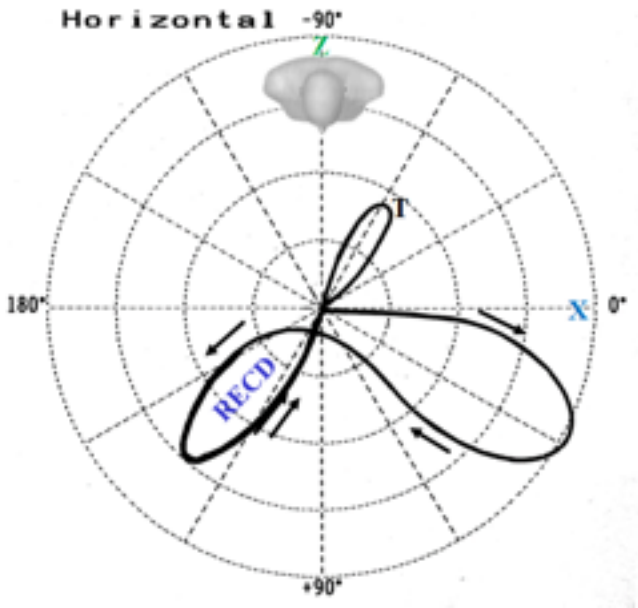
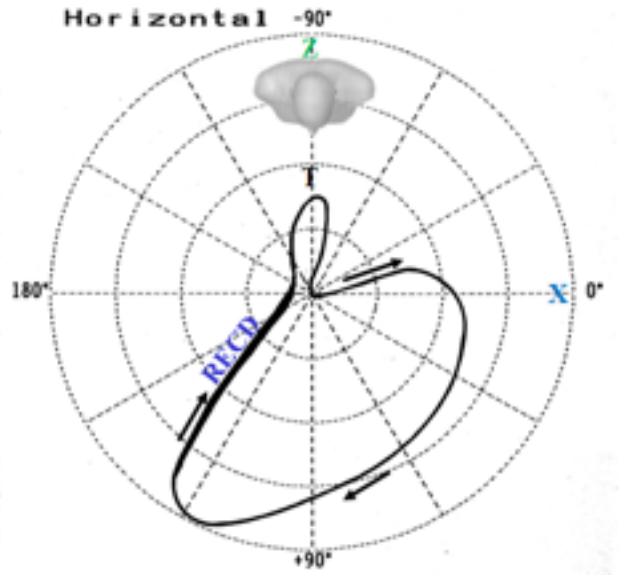
- Voltage of R' wave of V_1 (rsR') of 15 mm of height or greater in the presence of CRBBB;
- Voltage of R' wave of V_1 (rSR') of 10 mm of height or greater in the presence of IRBBB;
- R' wave of great voltage is more likely to correspond to RVH in children than in adults;
- Persistence of triphasic morphology (rSR') in intermediary precordial leads (V_3 and V_4). This sign suggests hypertrophy of RV free wall;
- qR pattern in V_1 may be an indirect sign of RAE and this of RVH;
- Tetraphasic pattern (rsr's') in V_2, V_3 and up to V_4 suggests hypertrophy of trabecular region of the RV;
- Complex of the R/S type with negative T waves, beyond V_4, suggests hypertrophy of the low right paraseptal region of the RV;
- Initial q wave disappears, decrease of R voltage and increase of S depth in V_5 and V_6 are observed in Complete RBBB associated to great RVH;
- Pattern of Incomplete RBBB or Complete RBBB of sudden onset, suggests acute RVH by pulmonary embolism;
- Presence of P wave criteria of RAE associated to Complete RBBB suggests RVH, except for Ebstein's anomaly and tricuspid atresia.

Elements that suggest RVH in V_1 in the presence of IRBBB and CRBBB

IRBBB QRS duration < 120 ms	CRBBB QRS duration ≥ 120 ms
Isolated IRBBB V_1 	Isolated CRBBB 
IRBBB + RVH RsR' 	CRBBB + RVH R' > 15 mm CRBBB + RVH 

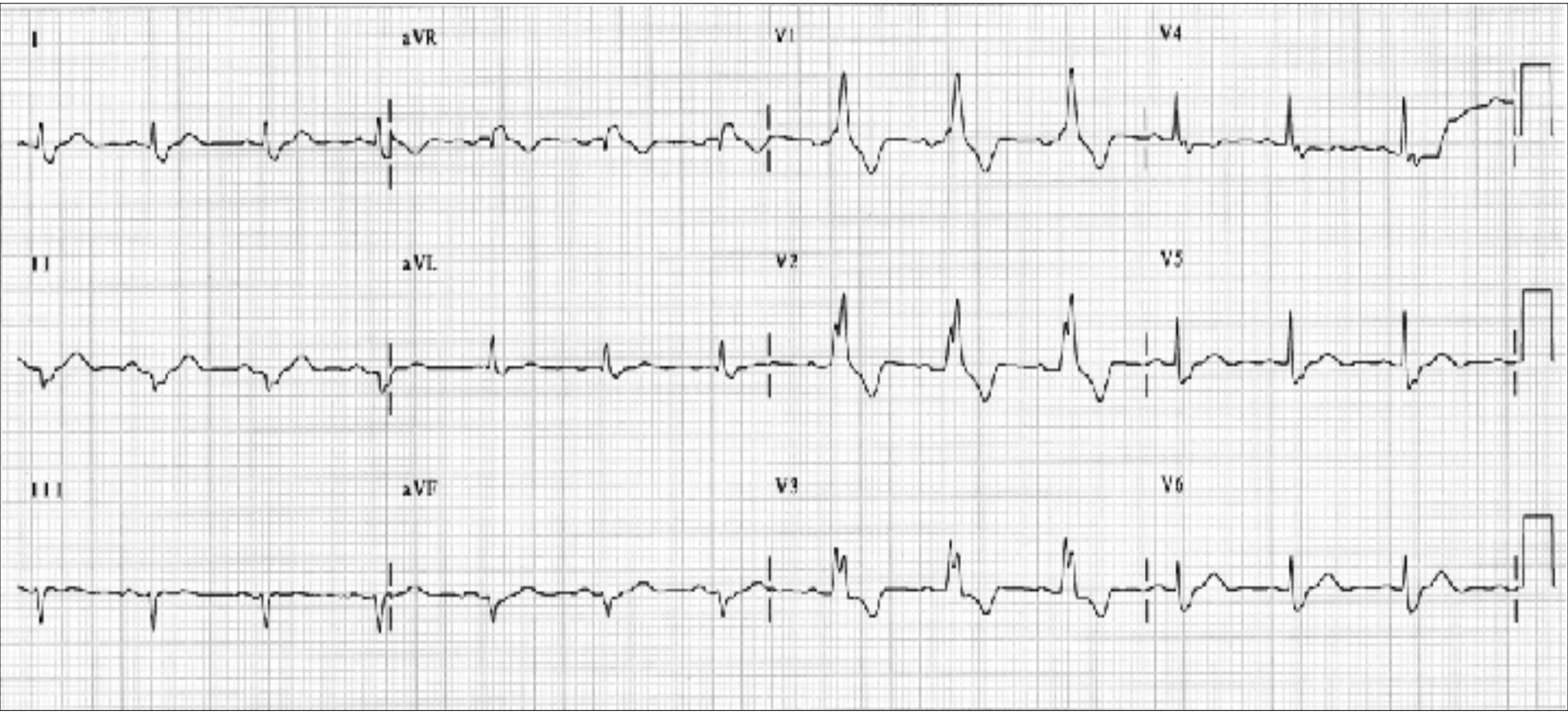
Voltage criteria of R' in V1 > 10 mm for IRBBB and > 15 mm for CRBBB that indicates associated RVH.

VCG criteria of CRBBB associated to RVH on HP (Miquel 1958)

CRBBB + Mild RVH	CRBBB + Moderate RVH	CRBBB + Severe RVH
		
CRBBB VCG Grishman type or Kennedy type I	**CRBBB VCG Cabrera type or Kennedy type II**	**CRBBB VCG Kennedy type III or C**
CCW rotation	**In 8 rotation**	**CW rotation**

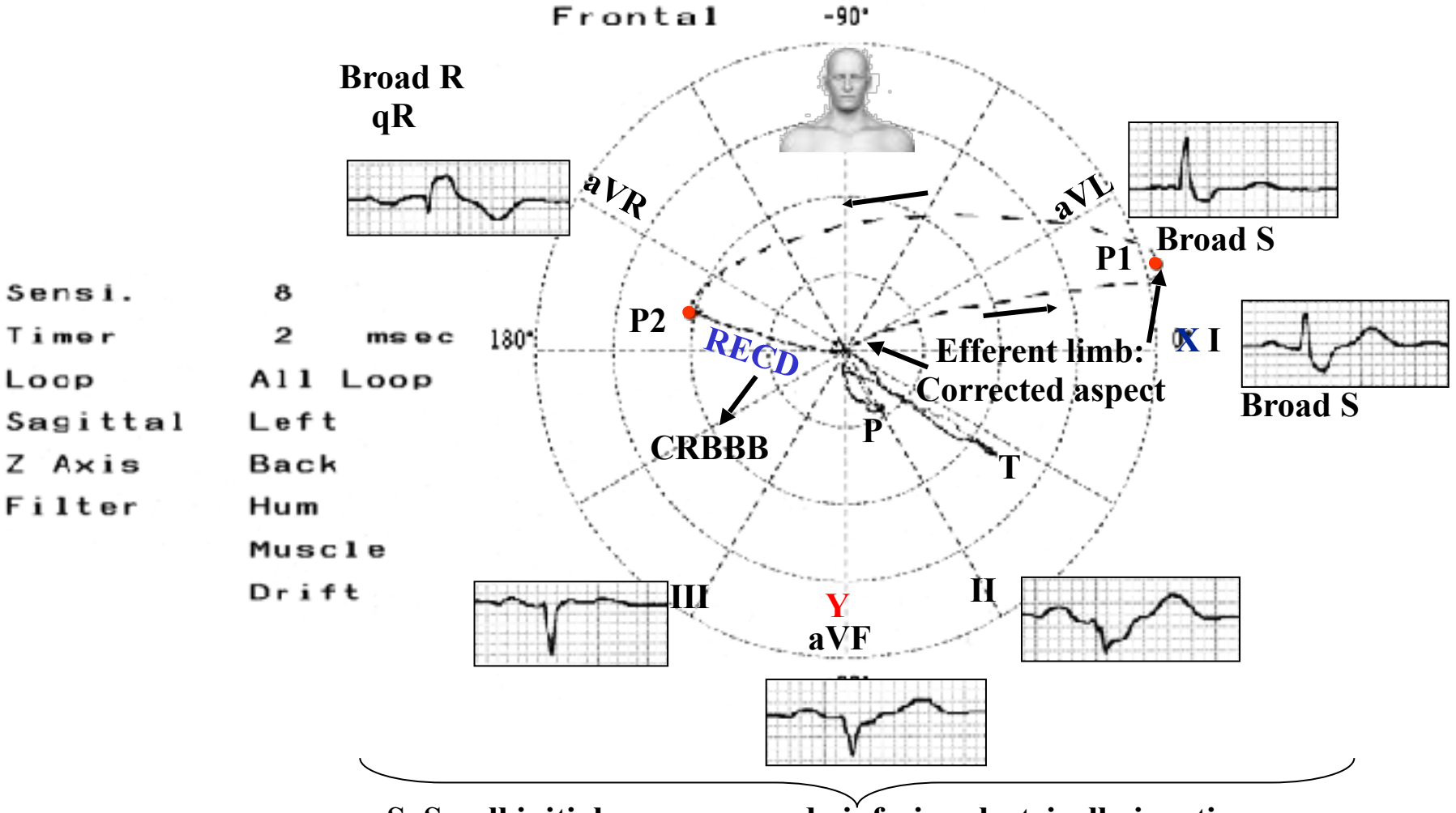
(1) a CW rotation of the QRS loop in the HP, (2) a ratio of the magnitude of the R wave to that of the S wave (R/S ratio) in lead X at less than 2.0, (3) a mean QRS vector in lead X more negative than--10 mv.msec, or (4) a maximal QRS vector located between 90º and -90º in the HP. In contrast, an R/S ratio in lead X that was ≥ 2.0 or an azimuth angle of the mean spatial QRS vector that was not between 90º and ± 180º would indicate that the right ventricular conduction defect is probably uncomplicated (**Brohet 1978**).

Name: PAG; **Gender:** male; **Age:** 75 y.o.; **Race:** white; **Weight:** 80 Kg; **Height:** 1.70 m; **Date:** 16/12/2003
Medication in use: Enalapril 20 mg; Prednisteroids 20 mg per day; Salbutamol 2 per day.



Clinical diagnosis:. Emphysema and systemic hypertension
Echocardiogram: mild concentric hypertrophy. Mitral ring calcification. Mild RV dilatation.
ECG diagnosis: SR, HR: 78 bpm; P wave: SÂP: +63°; duration: 80 ms; Voltage: 1 mm. PR: 172 ms. QRS: SÂQRS: with extreme deviation in the right superior quadrant; -120°; QRSD: 140 ms; SAT: +50° and to the back; QT: 430 ms; QTc: 490 ms.
Conclusion: Complete Right Bundle Branch Block + PAF (Prominent Anterior Forces). Cause? RVH? SFB? Extreme deviation of SÂQRS in the right superior quadrant: LAFB? Electrically inactive inferior area? Association of both?

ECG/VCG correlation in the FP



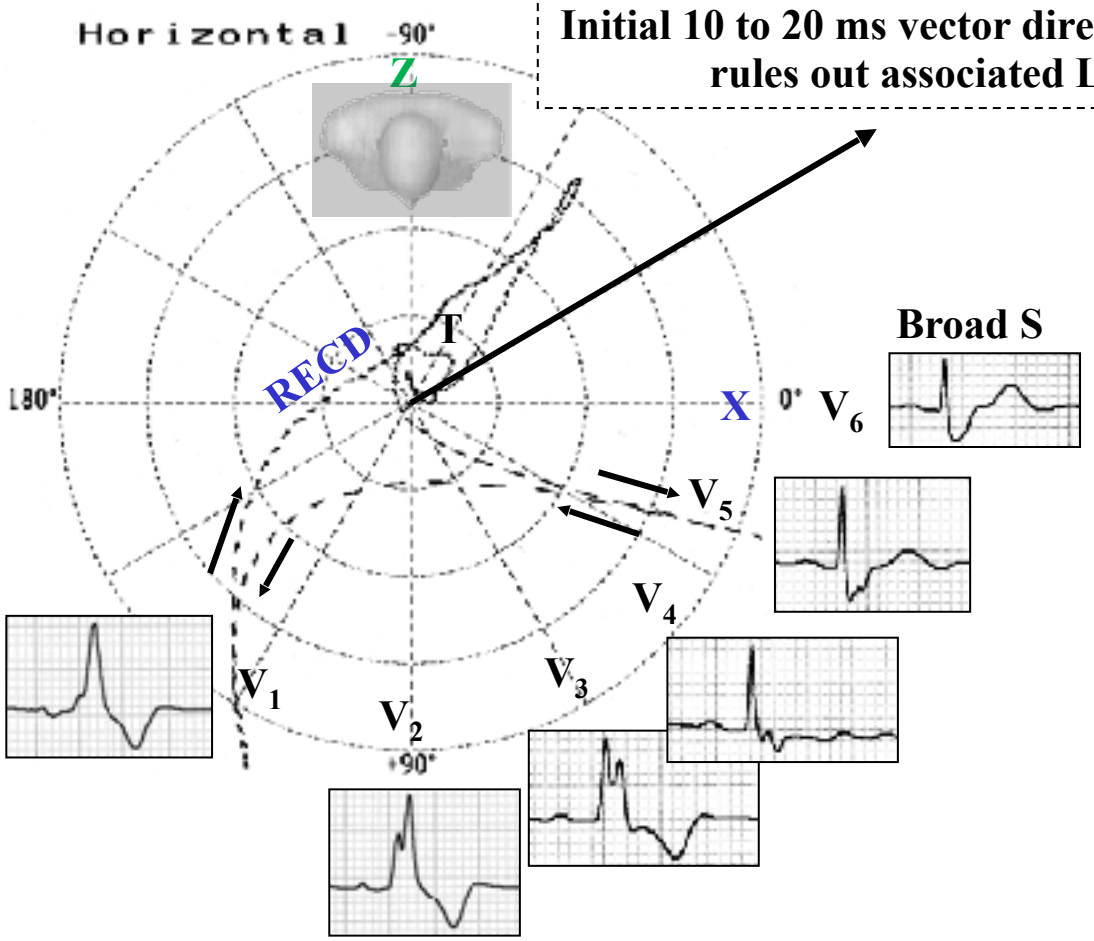
rS. Small initial r wave: pseudo inferior electrically inactive area

Note: The diagnosis of LAFB and/or inferior electrically inactive area is not configured. The initial forces are directed to the left and upward. The greatest part of the QRS loop located in the right superior quadrant rules out LAFB (in spite of its CCW rotation). The fast recording of QRS loop onset in the FP and the corrected aspect of the efferent limb rule out the diagnosis of inferior Myocardial Infarction. In spite of the extreme deviation of the QRS axis in the superior quadrants, associated LAFB is not configured, even with a CCW rotation. RECD is indicative of CRBBB.

ECG/VCG correlation in the HP

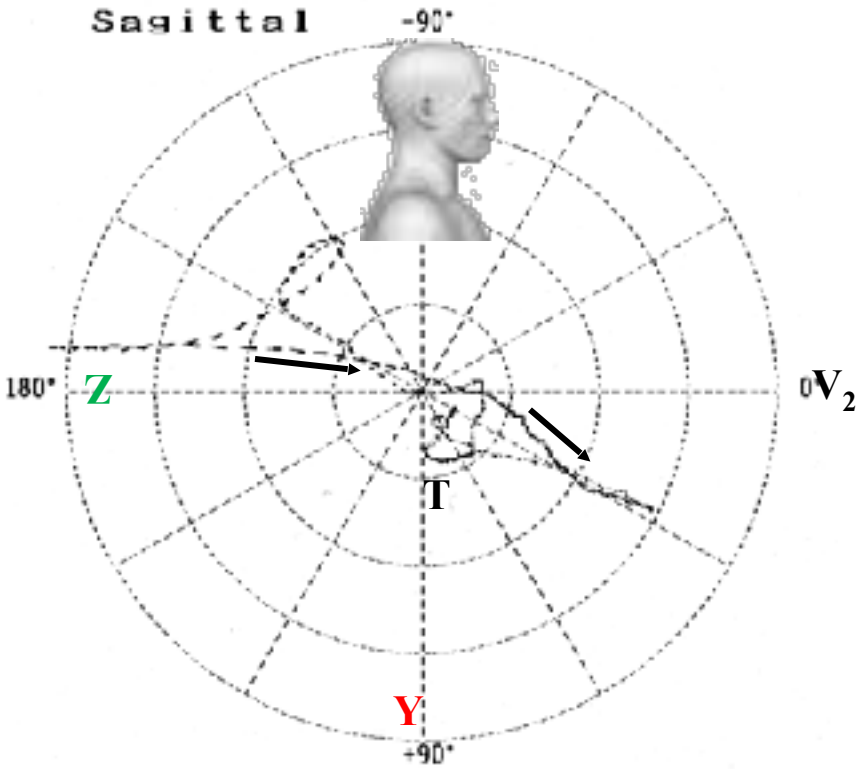
CRBBB
Kennedy
Type III of VCG: anterior dislocation of QRS loop with CW rotation
+
PAF

RVH

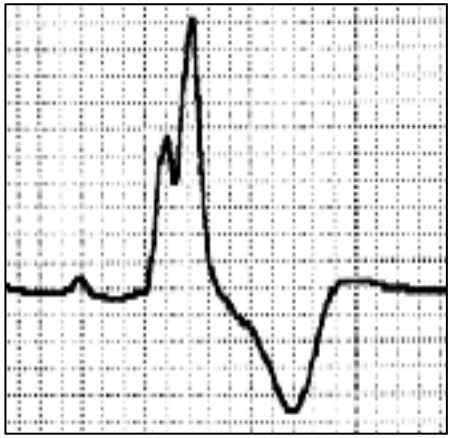


Monophasic R waves with notch from V1 to V3: CRBBB + PAF (Prominent Anterior Forces).

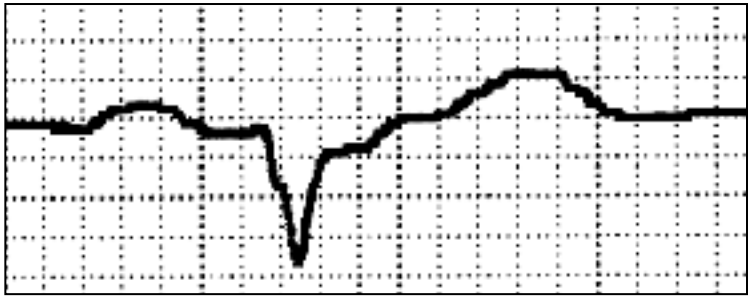
ECG/VCG correlation in the RSP

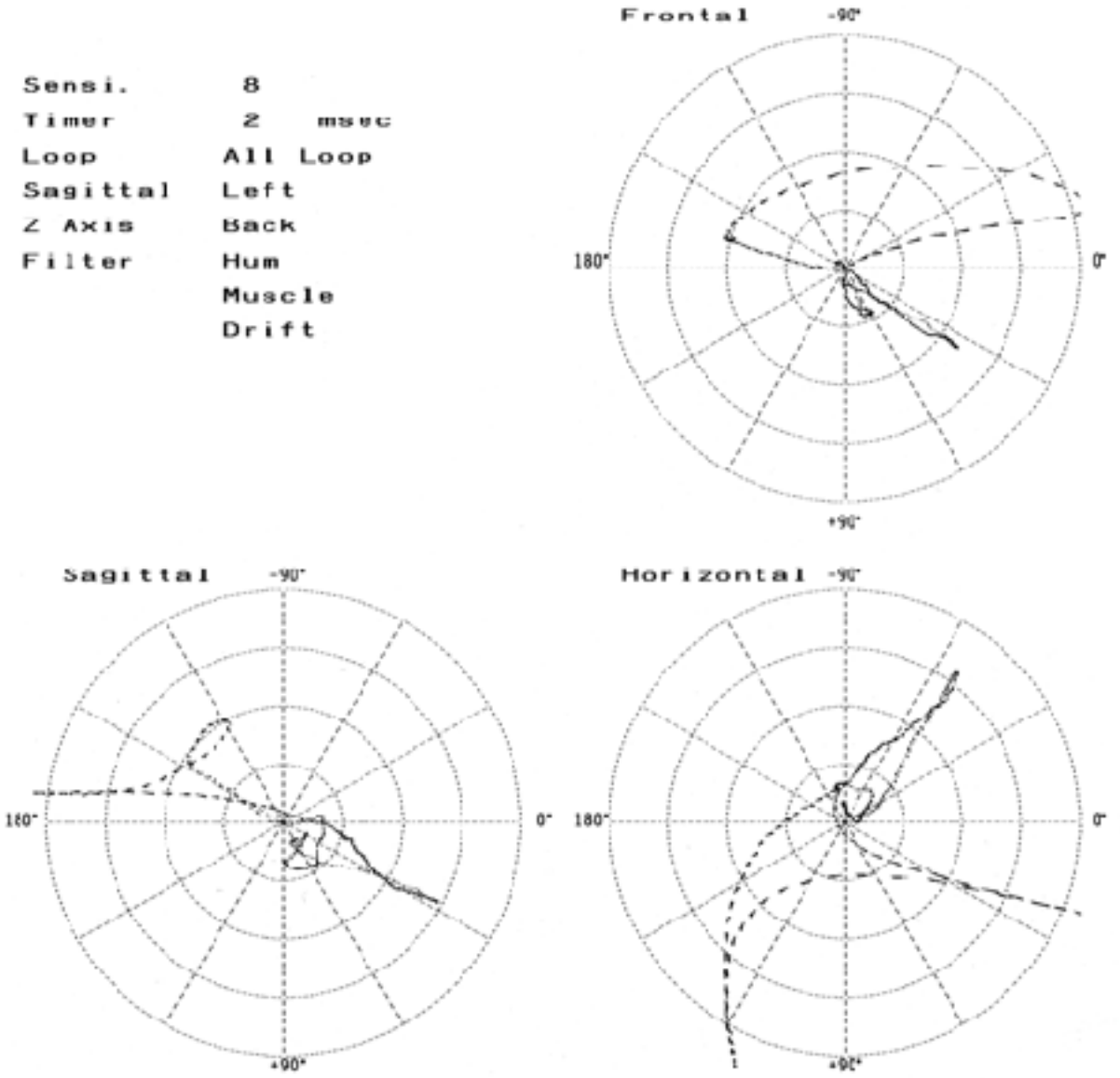


V_2



aVF





VCG of the same patient in three planes.

Diagnostic conclusion: ECG/VCG

1) CRBBB VCG Kennedy type III;
2) RVH;
3) Prominent Anterior Forces (PAF).

Comment: by VCG in the HP, CRBBB may be:

1) Kennedy type I or Grishman (afferent limb behind the X line);
2) Kennedy type II or Cabrera (afferent limb in front of the X line with loop in 8);
3) Kennedy type III (QRS loop of clockwise rotation and completely located in the anterior quadrants in the HP).

Right Bundle Branch Block associated to Left Ventricular Hypertrophy

ECG criteria of CRBBB associated to LVH

1) Presence of Morris criteria for left atrial enlargement in absence of mitral valve stenosis. Specificity: 90%, sensitivity: 32%. The correlation of LA abnormality by ECG and LVH by echocardiography conclude that LA abnormality by ECG is significantly diagnostic of LVH in the presence of RBBB (**Metha 1998**);
2) SÂQRS deviation to the left beyond -30° to -90° (sensitivity 59%, specificity 71%) (**Vandenberg 1989**).
3) Voltage of R wave of I > than 10 mm. Specificity: 90%, sensitivity: 39%;
4) Voltage of R wave of aVL > than 7 mm. Specificity: 74%, sensitivity: 50%;
5) In I and aVL, qRs pattern, with q and R wave of greater voltage and s wave of reduced voltage;
6) In the leads of the inferior wall: II, III and aVF; rSr' pattern;
7) R unipolar morphology of right precordial leads observed in intermediary precordial leads V_3 and V_4;
8) Increase in S wave depth in V_1: rSr'. The S corresponds to vector III of the hypertrophic LV free wall that gets away from V_1;
9) Voltage of R wave of $V_5 \geq$ than 20 mm. Specificity: 90%, sensitivity: 20%;
10) S wave of V_1 + R of V_5 or V_6 > 35 mm. Specificity: 100%, sensitivity: 4%;

11) S wave in lead III + the maximal R+S in a precordial lead > or = 35 mm. (sensitivity 68%) (**Vandenberg 1989**).(**Oreto 2007**)

12) Combination of left axis deviation and SIII + (R + S) maximal precordial lead greater than or equal to 30 mm (sensitivity 52%, specificity 84%). (**Vandenberg 1989**). Prolonged ventricular activation time (VAT) "R peak time", or intrinsicoid deflection of V_5 and $V_6 \geq$ than 50 ms;

13) In patients with RBBB none of the criteria have an acceptable performance (sensitivities ranged from 17% to 41% and specificities ranged from 54% to 85%). (**Fragola 1992**).

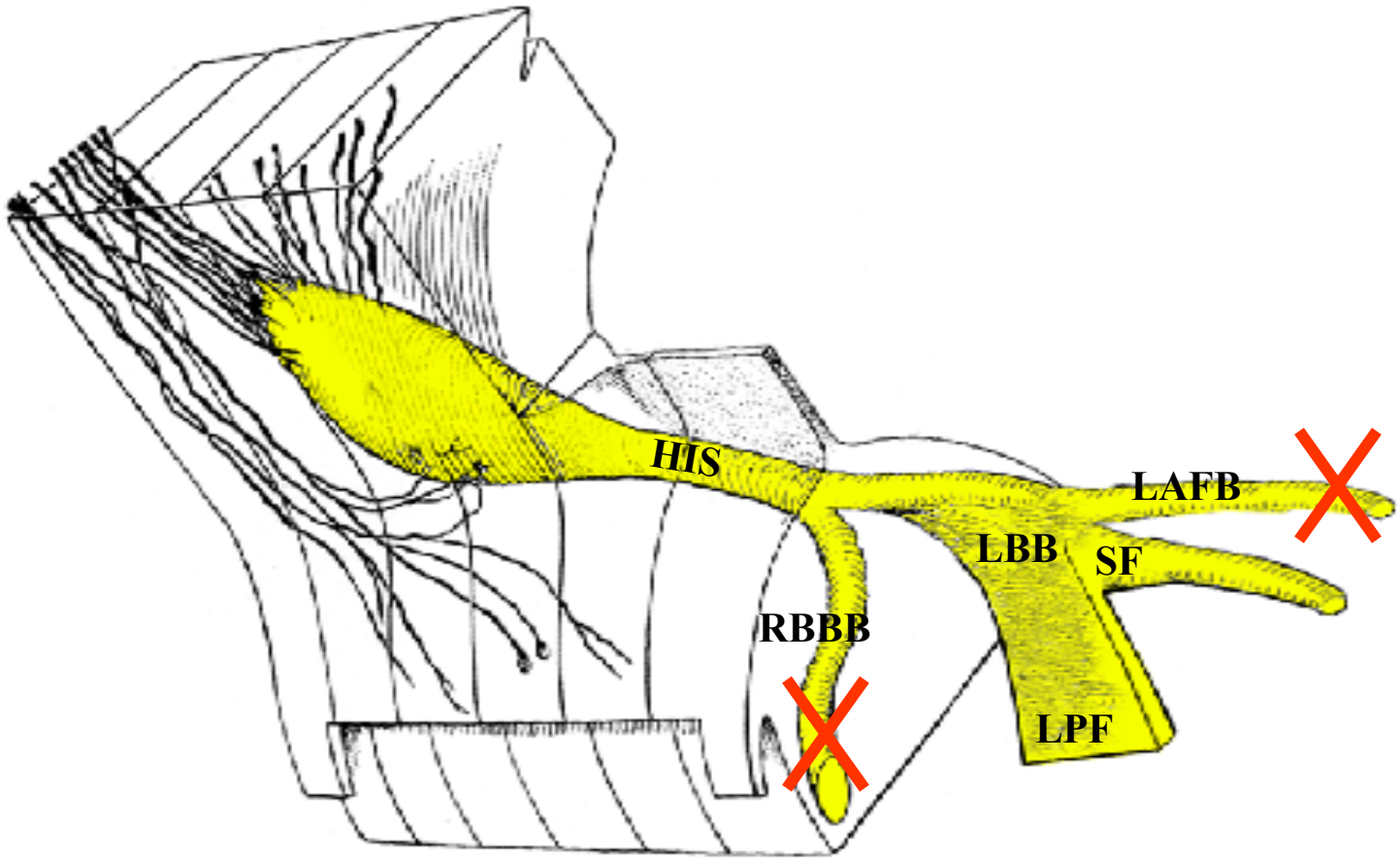
14) The electrocardiographic criteria with the highest sensitivity and specificity greater than 90% were left axis deviation of -30 degrees to -90 degrees and SV1 greater than 2 mm (sensitivity 34%), point-score system

15) RaVL greater than 12 mm and RI + SIII greater than 25 mm (each with a sensitivity of 27%). In general, limb lead voltage criteria such as RaVL greater than 11 mm (sensitivity 29%, specificity 86%) had higher sensitivities than using right precordial lead S-wave voltage criteria such as SV1 + RV5, V6 greater than 35 mm (sensitivity 2%, specificity 100%)

BBB is associated with an increase in initial QRS forces (RV1, RV2, and QV6) but significant decreases in mean mid-QRS amplitudes that reflect left ventricular depolarization (RaVL, SV1, SV3, RV5, and RV6).

All late QRS forces are increased (R' V1, SV5, SI). As a result, voltages criteria classically used for LVH are significantly reduced such as Sokolow-Lyon voltage (SV1+RV5 or RV6), and Cornell voltage (SV3+RaVL). RBBB is associated with significant reduction in "left ventricular" QRS amplitudes of the standard ECG, consistent with cancellation, rather than unmasking, of left ventricular mid-QRS forces by altered septal and delayed right ventricular depolarization. Because QRS voltages that are routinely combined for the detection of LVH are reduced in RBBB, standard LVH criteria will perform with lower sensitivity in patients with RBBB. (**Chan 2006**)

CRBBB associated to LAFB



CRBBB associated to LAFB

It constitutes the most frequent type of bifascicular block. In the first world it was estimated in 1.4% of all ECGs. In patients that developed complete AV block and had to have a pacemaker implanted, this association was found previously in 35% of the cases.

Etiologies

1. **Chronic chagasic myocarditis**: it constitutes the most frequent association in Latin America, where chronic chagasic myocarditis exists from the Argentinean Patagonia up to the frontier with USA. In the CRBBB of chronic chagasic myocarditis, the high association with LAFB stands out: 70% of the cases. In patients younger than 40 years old, from the endemic area, with ECG that shows association of CRBBB and LAFB, there is a high suspicion of chronic chagasic myocarditis, and even more with the additional presence of polymorphic ventricular extrasystole and primary alterations of ventricular repolarization. The association considered typical and most frequent, is complete right bundle branch block (CRBBB) of the His bundle and left anterior fascicular block (LAFB). A longitudinal study of 5,710 infected patients, showed that the presence of CRBBB associated to primary alterations of repolarization and electrically inactive areas, indicates high risk of death. Autopsy studies conducted by Andrade, revealed that most of the patients with chronic chagasic heart disease present a significant involvement of the excito-conductor system at the level of the N-H portion of the AV node, right penetrating and branching portion of the His bundle, proximal portion of the right branch and the anterosuperior division of the left branch. We conclude that CRBBB of chronic chagasic heart disease is of the proximal type.(**Shabelman 1961**)(**Rosembaum 1964**) (**Dubner 2008**). The most common ECG changes are the following: complete RBBB (35%) and LAFB (35%). (**Marques 2006**). CRBBB with LAFB is strongly related to Chagas disease in older patients. (**Ribeiro 2014**) **(Garzon 1995**)

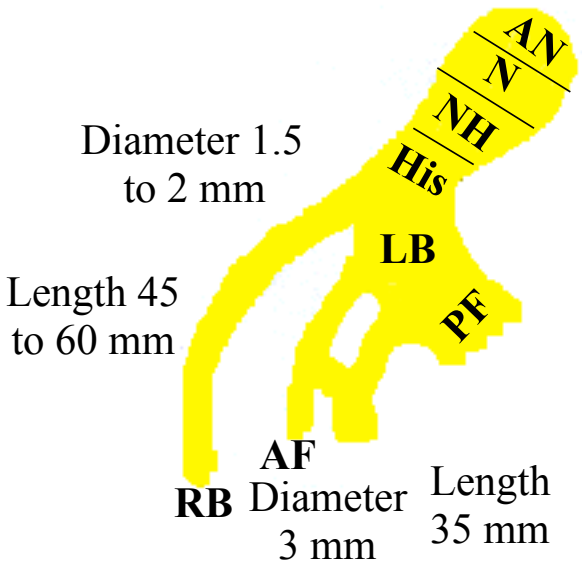
Characteristics and frequency of CRBBB associated to LAFB (bifascicular block)

2. **Coronary artery disease:** it constitutes the main cause in the first world, where Chagas disease does not exist. It is estimated in 1% of the hospital population. It constitutes a complication of acute myocardial infarction of approximately 6%, almost always by obstruction of the LAD artery, since the RBB and the left anterior fascicle are irrigated by the perforator branches of this artery.
3. **Hypertensive heart disease**: 20 to 25% of the cases;
4. Sclerodegenerative disease of the His system, genetic Lenègre disease with or without high blood pressure.
5. Lev disease or sclerosis of the left side of the cardiac skeleton;
6. Chest trauma. The closed trauma of the chest without a cut is frequently accompanied by CRBBB. In this case, the CRBBB frequently disappears after some hours.
7. Familial with syncope or sudden death;
8. **Other myocarditis;**
9. **Sarcoidosis;**
10. **Granulomatosis;**
11. **Aortic valve disease;**
12. **Hyperkalemia or hyperpotassemia**
13. **Congenital isolated;**
14. **Associated to progressive external ophthalmoplegia;**
15. **Post-surgical trauma:** 1) After corrective surgery of Fallot, we may find CRBBB associated to LAFB (7 to 25% of the cases). It is of the truncus and it indicates that the LAF has been injured concomitantly when the surgeon sutured the "patch", placed to increase the RVOT. The patients that remained with bifascicular block after corrective surgery do not present a greater index of late mortality. 2) In 4% of the cases after corrective surgery of VSD. 3) After surgery of substitution of tricuspid valve: it has been described as a complication in the exchange of tricuspid valve. 4) After surgery of bypass of internal thoracic and/or saphenous artery, it has been observed in 4% of the cases. The CRBBB in isolation was observed in 6% and LAFB in 6%;

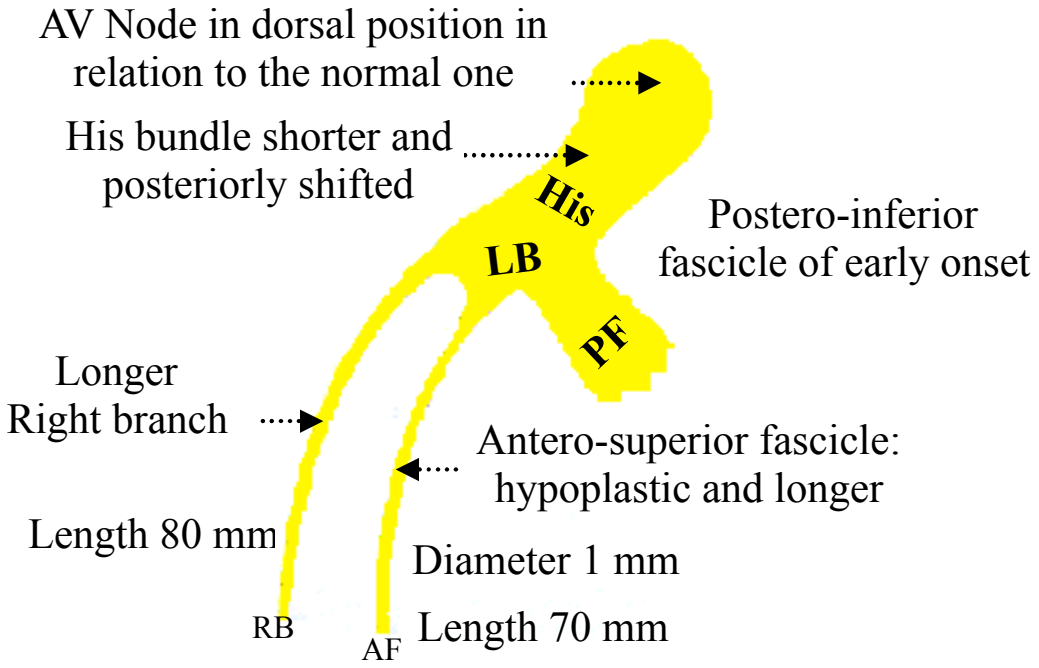
16. **Post-orthotopic heart transplantation:** The most frequent case is ARBBB or IRBBB in isolation (present in 45% to 80%), which may be associated to LAFB in approximately 20%, presenting in most of the cases a permanent nature.
17. **Atrial septal defect of the ostium primum type or endocardial cushion defects**: In the horizontal plane we frequently find incomplete RBBB or complete RBBB pattern with signs that suggest RVE and/ or BVE. Thus, in the right precordial leads V3R and V1 we can see triphasic patterns of the rsR' or rSR' type with opposite ST and T indicating not very high pressure in the pulmonary artery. In endocardial cushion defects, the right branch is congenitally longer; a fact responsible for the incomplete RBBB or advanced RBBB pattern. In fact, it is a false branch block, since the pattern is due to a delay in RV activation, because the stimulus must go through a longer trajectory. In more than 98% of the cases, extreme deviation of SAQRS is verified concomitantly in the left or right superior quadrants. The latter is more frequent in the total form and showing a pattern of counterclockwise rotation of QRS loop in the frontal plane of the LAFB type. Indeed, there is no true LAFB, but there is early activation of the LV postero-inferior wall by early onset too, of the postero-inferior fascicle of the His bundle, associated to a hypoplasia and extension greater than the antero-superior fascicle, which delays even more the activation of the antero-superior wall of the LV.

CRBBB associated to LAFB: Etiology

Normal intraventricular His system



Intraventricular his system in endocardial cushion defects



It explains RBBB and early postero-inferior activation of the AFB.
Delayed antero-superior region activation

Etiology of CRBBB associated to LAFB by endocardial cushion defects.

CRBBB associated to LAFB: Electrocardiographic criteria

The criteria for CRBBB are observed in the horizontal plane, while those of LAFB in the frontal plane; on the other hand, LAFB modifies the middle portions of ventricular activation while CRBBB modifies the final ones.

1) QRS complex of duration ≥ than 120 ms in the presence of supraventricular command.
2) QRS of duration >130 ms and SAQRS from -30° to –90°, which individualize the patients with a greater probability of cardiovascular disease;
3) Extreme deviation of SAQRS in the superior quadrants: beyond -30^0, which may go beyond -90^0. The axis between -30^0 and -45^0 suggests minor degree of LAFB. If SAQRS is located in the right superior quadrant, it indicates important participation of CRBBB and/or association with RVE;
4) qR pattern in aVL. qRS or RS morphology may be found. The small initial q wave indicates the first vector heading downward and to the right, produced by the non blocked septal and posterior fascicles. The R wave indicates superior shift of the loop by the LAFB when activating the blocked antero-superior region;
5) Right precordial leads V3R, V1 and V2: triphasic pattern of the rSR' or rsR' type, with R' greater than r. There may be R with notch, ("M shaped"), rR' or qR;
6) Broad S wave with a duration ≥40 ms in adults in left leads V5, V6 and I by end delay to the right and the front of CRBBB.
7) J point, ST and T opposite to the final deflection of QRS: in the left leads with the J point and the ST segment discretely elevated with positive T and asymmetrical limbs (slow ascending and fast descending).

In an 11-year period 209 cases of bilateral BBB (RBBB + LAFB or LPFB). The majority of patients had evidence of CAD or hypertension. A significant number had no clinical evidence of heart disease. The majority of patients had follow-up for about 2 years. The incidence of complete heart block was 14.4% (30 of 209). Complete heart block developed more than 10 years after the discovery of bilateral BBB in several patients. It is anticipated that with more complete and longer follow-up the incidence of complete heart block will be even higher.(**Scanlon 1970**)

CRBBB associated to LAFB: Vectorcardiographic criteria (Benchimol A 1971)

The initial portion of the loop behaves as a LAFB and the final part as a Complete RBBB. The duration of QRS loop ≥ than 120 ms.

Frontal plane:

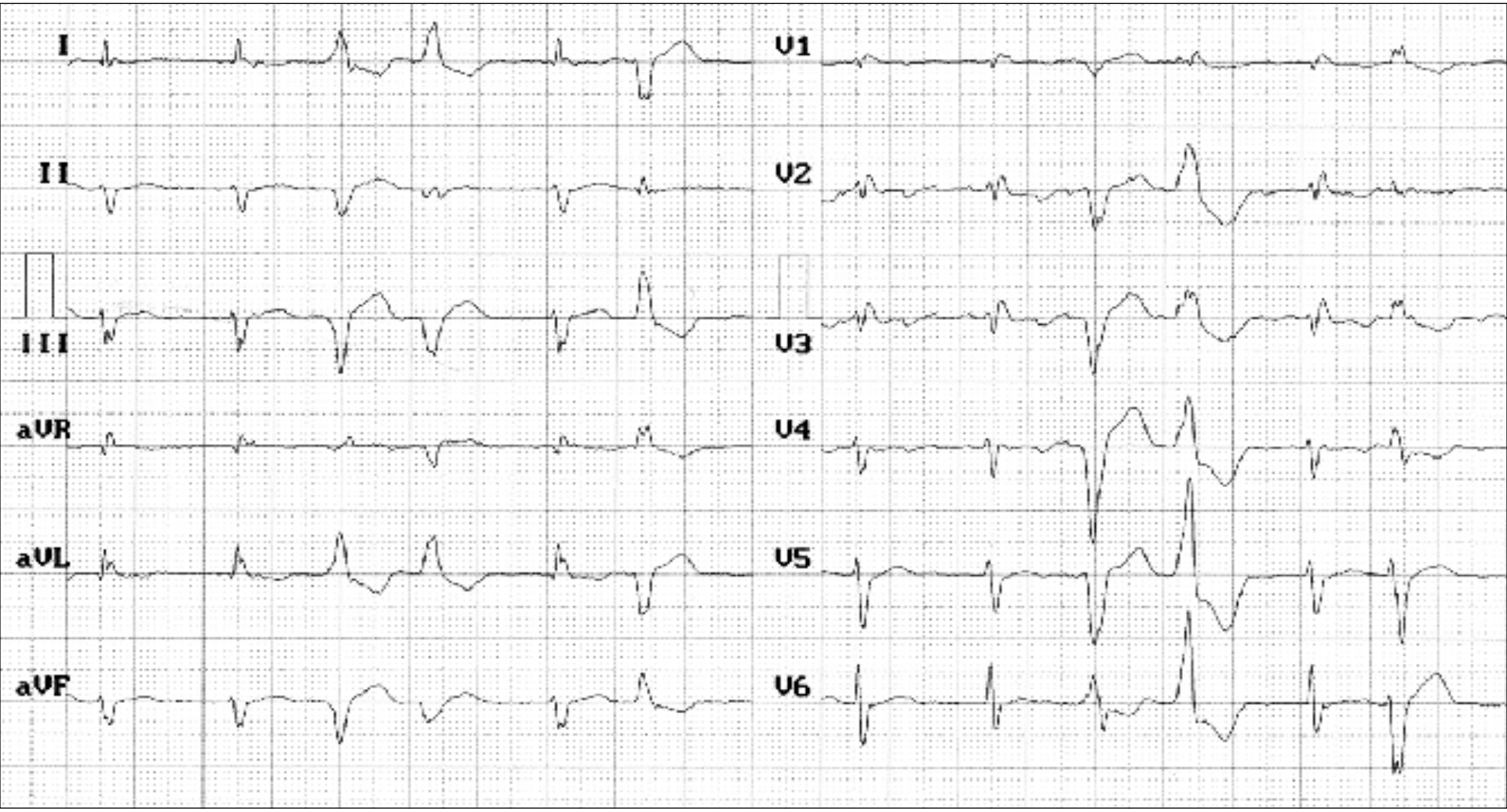
Very similar to isolated LAFB loop:

1) Initial vectors from 10 to 20 ms heading downward and to the right (type I) or downward and to the left (type II);
2) QRS loop of counterclockwise rotation;
3) SAQRS with extreme deviation to the left beyond -30^0;
4) Efferent limb of QRS loop heading to the left and finally to the left and upward;
5) Afferent limb that begins above and slightly to the left, to finally end in a final appendage of slow recording and located to the right and above.

Horizontal plane:

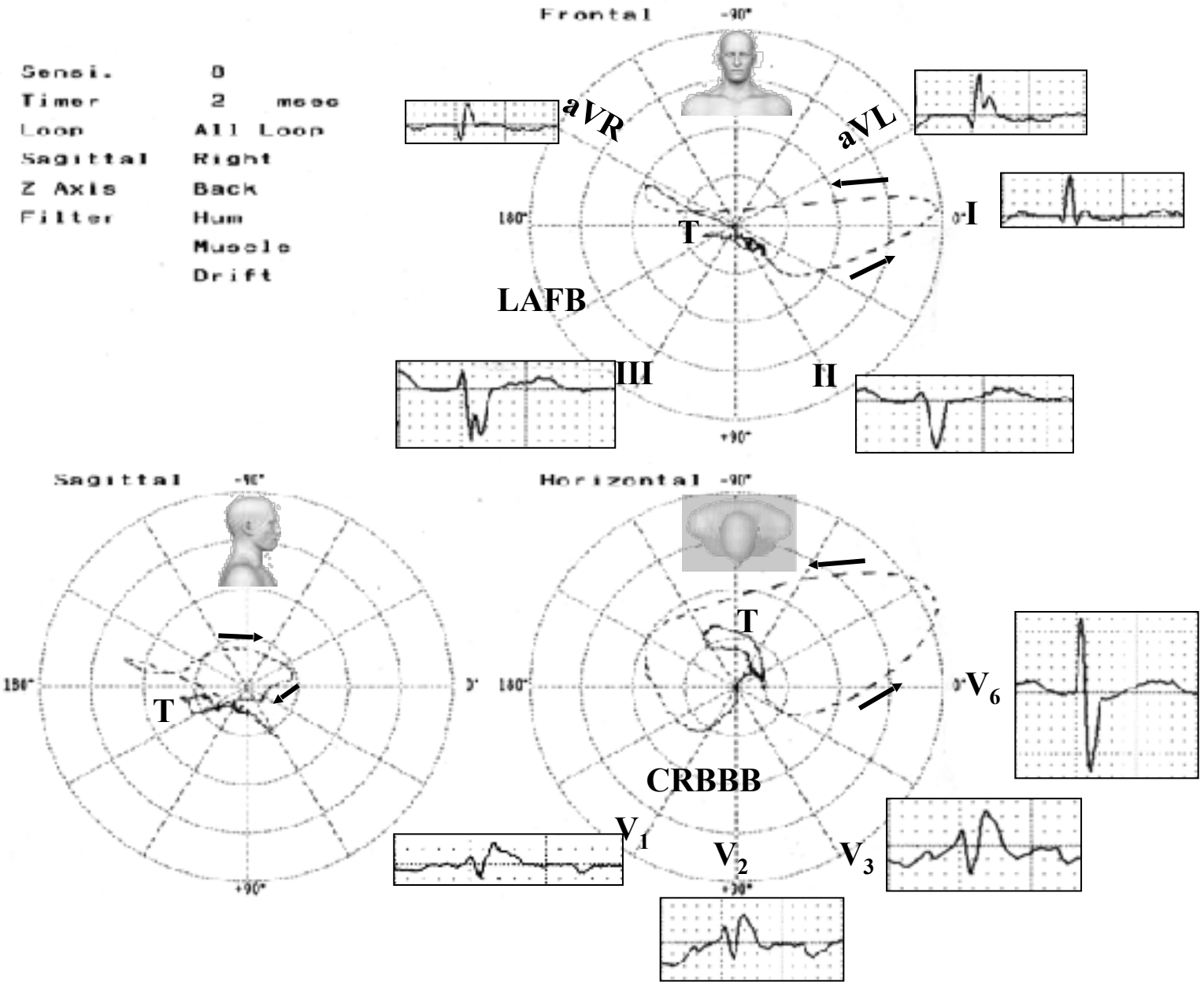
Typical of QRS loop of isolated Complete RBBB.(**Zamfirescu 1978**)

1. Vectors from initial 10 to 20 ms heading to the front and the right or left (**Retamal 1972**)
2. Efferent limb of QRS loop from right to left and with variable degrees of anteriorization;
3. Main body of QRS loop with counterclockwise (type I), eight or clockwise rotation (type II). The type of rotation seems to lack clinical significance; however, type II appears in a greater number of patients in CHF;
4. Afferent limb of QRS loop in front of the X line from left to right;
5. Efferent limb of QRS loop behind or in front of the X line;
6. End delay located in the right anterior quadrant.(**Medrano 1969**)(**Cergueira-Gomes 1972**)
7. Ventricular repolarization with T loop opposite to the final portion of the QRS loop to the left, behind and below. (**Kukbertus 1970**)(**Lichstein 1973**)



Clinical diagnosis: Chronic chagasic myocarditis.
ECG diagnosis: P wave of difficult visualization, indicating intense fibrosis of atrial tissue.
LAFB: extreme deviation of AQRS in the left superior quadrant, around -75°, qR in I and aVL, rS in inferior leads with S in V_5 and V_6
CRBBB: triphasic complex of the rsr' type from V_1 to V_3, wide r of aVR and S in V_5 and V_6
Coupled polymorphic premature ventricular contractions.
Classical triad: CRBBB + LAFB + Polymorphic ventricular contractions

ECG/VCG correlation



VCG in the three planes of the same patient and its correlation with ECG. See the diagnosis of LAFB in the frontal plane and CRBBB in the horizontal plane.

The classical electrocardiographic hallmark of chronic chagasic myocarditis includes the association of complete right bundle branch block with left anterior fascicular block.

We know today that the ECG complex called since Richman “masquerading bundle-branch block”, is essentially a complete right bundle branch block and left anterior fascicular block, with further modifications of the initial and final QRS vectors, so that standard leads, and at times the left precordial leads, resemble left bundle branch block.

We present a case of standard masquerading bundle branch block associated with concomitant hidden right bundle branch block on right precordial leads consequence of low anteroseptal and lateral extensive electrically inactive area.

We think that this is a new type of masquerading bundle branch block.

Chronic Chagasic Myocarditis (CCM) is an important cause of heart failure in Latin America, but is rare in the United States. Also, due to migratory currents between countries and far-distant regions, CCM is likely to become ubiquitous 7. A reflection of this tendency is exemplified by the recent growing awareness regarding the occurrence of CCM in the United States. Based on a prevalence of 4.5% of *T. cruzi* serologically detected infection in 205 Latin American immigrants to the USA, and on estimates of the number of such immigrants, approximately half a million infected people are believed to exist now in that country 8. Conditions for vectorial transmission range between latitudes 42°N and 40°S of the American Continent, from Mexico to Argentina. It is estimated that as many as 8 to 11 million people in Mexico, Central America, and South America have Chagas disease, most of whom do not know they are infected. On the basis of limited serological surveys, 4% to 7% of more than 200 million Latin Americans are estimated to be chagasic in extensive areas of 21 countries, and 65-90 million are at risk of becoming infected (**Schmunis 1996**).

Cross-sectional epidemiological studies in Brazil and Venezuela assessed the prevalence of clinical manifestations and mortality due to CCM. However, no clear-cut epidemiological picture of CCM is yet available, due to the lack of appropriately designed large-scale studies to address this serious public health problem in extensive areas of Latin America. In addition, case reporting is not reliable even in areas of high endemicity. Probably because of marked variations in the genetic background, parasite strain, climate, socio-economic and related hygienic-alimentary conditions, and health care policies, the morbidity and mortality rates ascribed to Chagas' disease are extremely variable even among endemic areas of each country (**Wanderley 1995**). Although the true prevalence of CCM is unknown, these rough estimates clearly indicate that CCM is undoubtedly the most common form of cardiomyopathy in Latin-American countries (**Marin-Neto 1998**). Moreover, rural-urban migration from endemic areas in Brazil is believed to have brought to large cities half a million infected people in the last three decades (**Wanderley 1995**).

Transmission

In Chagas-endemic areas, the main mode of transmission is through an insect vector called a triatomine bug (1). A triatomine becomes infected with *T. cruzi* by feeding on the blood of an infected person or animal. During the day, triatomines hide in crevices in the walls and roofs. The bugs emerge at night, when the inhabitants are sleeping. Because they tend to feed on people's faces, triatomine bugs are also known as "kissing bugs". After they bite and ingest blood, they defecate on the person. Triatomines pass *T. cruzi* parasites (called trypomastigotes) in feces left near the site of the bite wound. Scratching the site of the bite causes the trypomastigotes to enter the host through the wound, or through intact mucous membranes, such as the conjunctiva. Once inside the host, the trypomastigotes invade cells, where they differentiate into intracellular amastigotes. The amastigotes multiply by binary fission and differentiate into trypomastigotes, which are then released into the bloodstream. This cycle is repeated in each newly infected cell. Replication resumes only when the parasites enter another cell or are ingested by another vector. The tropical rainforests and urban habitats are not ideal for the establishment of the human transmission cycle.

In regions where the sylvatic habitat and its fauna are thinned by economic exploitation and human habitation, such as in newly deforested areas, piassava palm culture areas, and some parts of the Amazon region, a human transmission cycle may develop as the insects search for new food sources. *T. cruzi* can also be transmitted through **blood transfusions**. With the exception of blood derivatives (such as fractionated antibodies), all blood components are infective. The parasite remains viable at 4°C for at least 18 days or up to 250 days when kept at room temperature. It is unclear whether *T. cruzi* can be transmitted through frozen-thawed blood components. Other modes of transmission include organ transplantation, through breast milk, and by accidental laboratory exposure. Chagas disease can also be spread congenitally (from a pregnant woman to her baby) through the placenta, and accounts for approximately 13% of stillborn deaths in parts of Brazil

1. **"DPDx – Trypanosomiasis, American. Fact Sheet". Centers for Disease Control (CDC). http://www.dpd.cdc.gov/dpdx/HTML/TrypanosomiasisAmerican.htm. Retrieved 12 May 2010**
2. **Teixeira AR, Monteiro PS, Rebelo JM (2001). "Emerging Chagas disease: trophic network and cycle of transmission of Trypanosoma cruzi from palm trees in the Amazon". *Emerging Infect Dis* 7: 100–112.**

Oral transmission is an unusual route of infection, but has been described. In 1991, farm workers in the state of Paraíba, Brazil, were infected by eating contaminated food; transmission has also occurred via contaminated açaí palm fruit juice and sugar cane juice. A 2007 outbreak in 103 Venezuelan school children was attributed to contaminated guava juice.Chagas Disease is a growing problem in Europe, because the majority of cases with chronic infection are asymptomatic and because of migration from Latin America.[6

The juice of the "açaí" fruit, when handmade, could be one of the main culprits for outbreaks of Chagas disease in Brazil. Between June 2006 and June 2007, 116 people were contaminated after ingesting the drink in the states of Amapá, Amazonas and Pará. The contamination of the juice happens when the insect carrier of the protozoan parasite that causes the disease, is grinded jointly with the fruit. According to the parasitologist Aldo Valente, from the Evandro Chagas Institute, an institution linked to the Department of Surveillance in Health of the Ministry of Health, outbreaks of Chagas disease transmitted orally have been occurring since 1968, but it was under-reported, mostly because of the lack of information. Now, as health care agencies have paid more attention to this issue, the number of recorded cases has increased. Besides, Valente reminds us that the ecological imbalance caused by deforestation pushes insects away from their natural habitat and sources of food, contributing in a decisive manner to the occurrence of outbreaks. The main problem brought about by the oral transmission of Chagas disease is that the ingestion implies a large amount of the protozoan parasite that causes the disease, the Trypanosoma cruzi, released in to the blood flow. This fact entails the reduction of the incubation period of the disease: as to conventional transmission, the first symptoms appear between the fourth and eighth week after contagion; in oral transmission, this period reduces to nearly 10 days and the disease can quickly evolve into its more severe forms. So, the violent evolution of this patient could be due to the mentioned mechanism.

Açai fruit →

The ECG

The classical electrocardiographic (ECG) hallmark of chronic chagasic cardiomyopathy includes the association of complete right bundle branch block (CRBBB) and left anterior fascicular block (LAFB). Today we know that the ECG complex coined by the first time in 1954 by Richman et al (**Richman 1954**) as "masquerading bundle-branch block", is essentially CRBBB with LAFB, with further modifications of the initial and final QRS vectors, so that standard leads, and at times the left precordial leads, resemble left bundle branch block (LBBB).(**Schamroth 1975; Unger 1958**)

Since the pioneer Rosenbaum´s et al studies **(Rosenbaum 1968; Rosenbaum 1973**) we know two ECG types: The "standard type" (*"standard masquerading right bundle-branch block")* and the "precordial type". (*"precordial masquerading right bundle-branch block")*

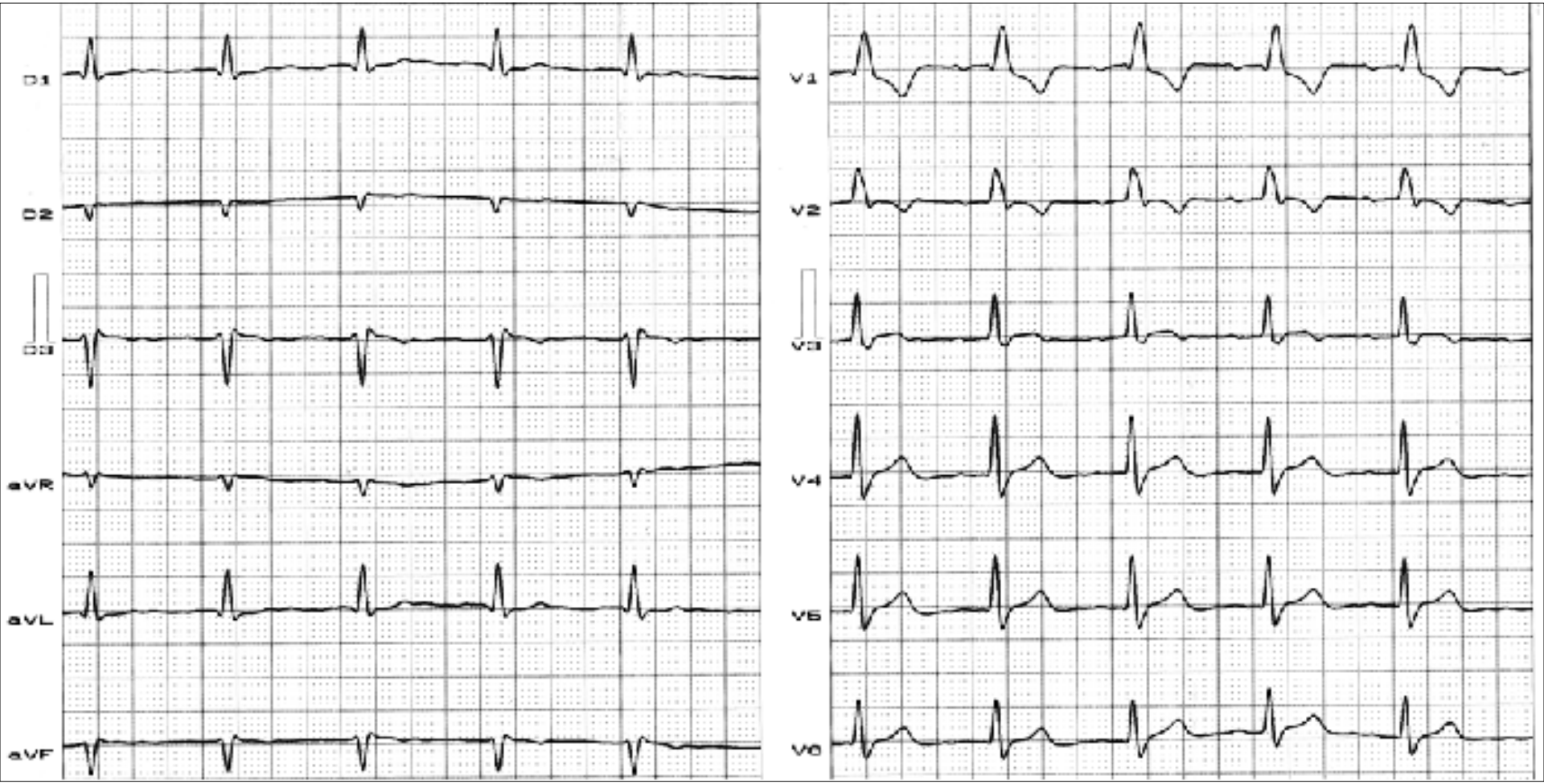
In the "standard type" the LAFB obscured totally or partially the diagnosis of CRBBB only in the frontal plane leads by abolishing (or it becomes very small) the final broad S wave in the left leads I and aVL (**Ortega-Carnicer**) and the precordial leads remain with the typical CRBBB pattern.

In the standard type, in the frontal plane, there are four main developmental phases that do not necessarily occur in a chronological sequence. Table 1 shows the four main developmental ECG patterns of standard type.

The precordial type shows the pattern of CRBBB in the right precordial leads and complete left branch block pattern (CLBBB) in the left-side precordial leads. This results from CRBBB associated with severe left ventricular hypertrophy (LVH), a localized block in the anterolateral wall of the left ventricle often due to myocardial infarction, and usually LAFB. Presumably, the intramural left ventricular block, together with LVH or LAFB, or both, produce predominant leftward forces which tend to cancel out the late rightward forces of the RBBB in the left precordial leads. Finally, masquerading bundle branch block can be associated with severe and diffuse conduction system disease, and patients with this finding may require permanent pacemaker implantation, especially if they are symptomatic (**Kowey 1989**).

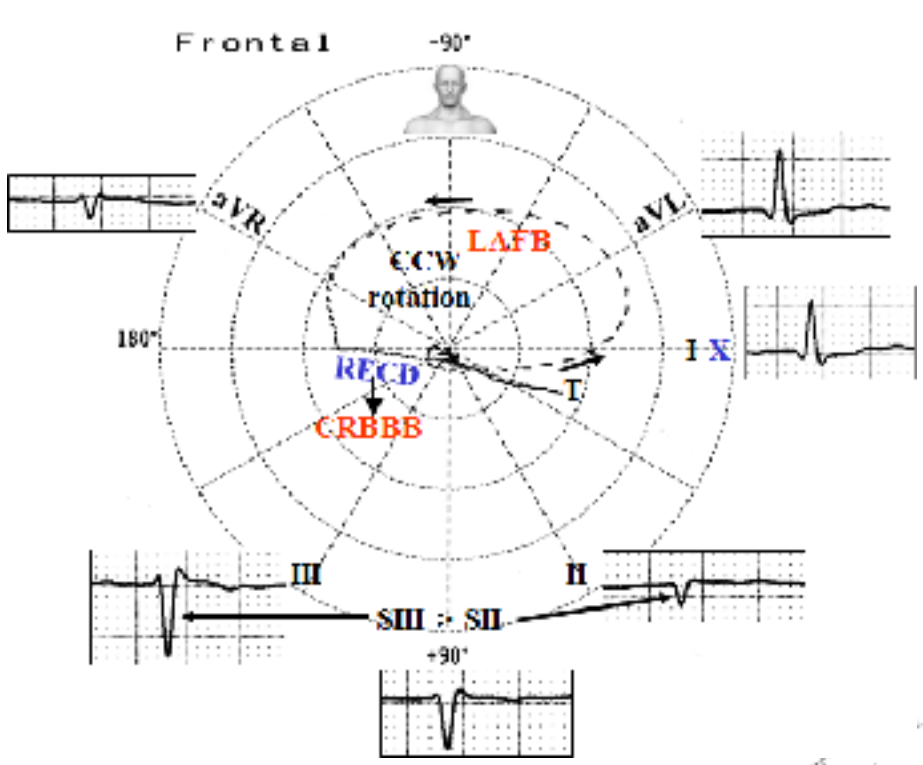
In the present case we report a possible new masquerading variant in a young woman with severe *chronic* fibrotic form *of chagasic cardiomyopathy* where there is c*oncomitant standard and precordial masquerade CRBBB* present. The standard left limbs I and aVL show qR pattern with low r voltage and wide QRS duration (pseudo-atypical complicated LBBB pattern) and in the anterior wall from V1 to V4, QS or Qr pattern consequence of severe anterior fibrosis that masks the existent CRBBB. Only the left leads V5-V6 show RBBB. Progression to high degree atrioventricular block is quite common in the presence of masquerading bundle branch block. It is frequently associated to advanced heart failure, so the prognosis is usually poor (**Bayés de Luna 1988; Gómez Barrado 1997**).

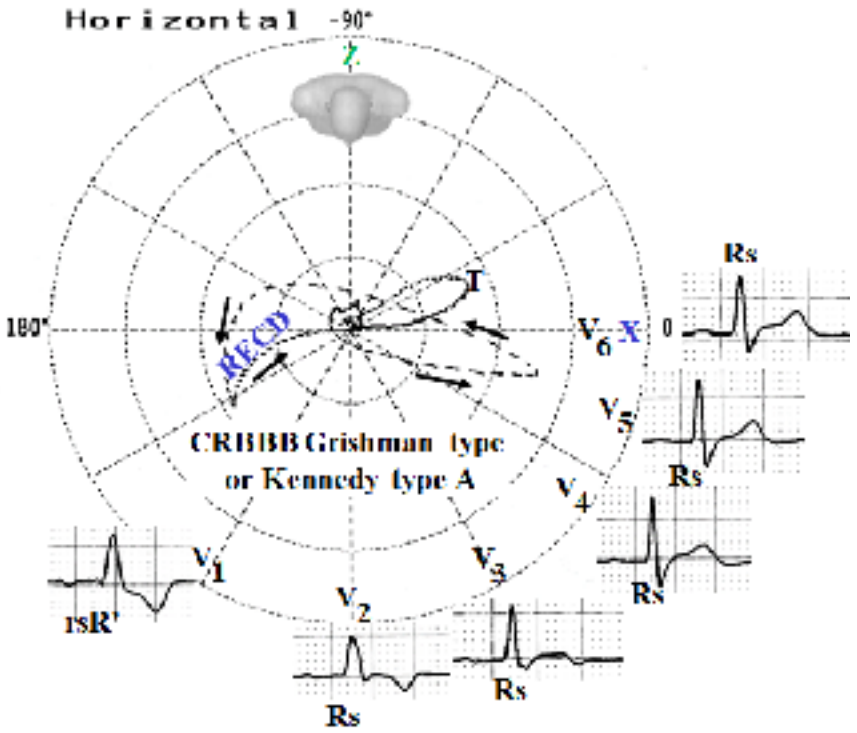
Name OO; **Sex:** Male; **Age:** 51 y; **Race:** Asian; **Weight:** 71 Kg; **Height:** 1.69 m**; Date:**19/04/2000

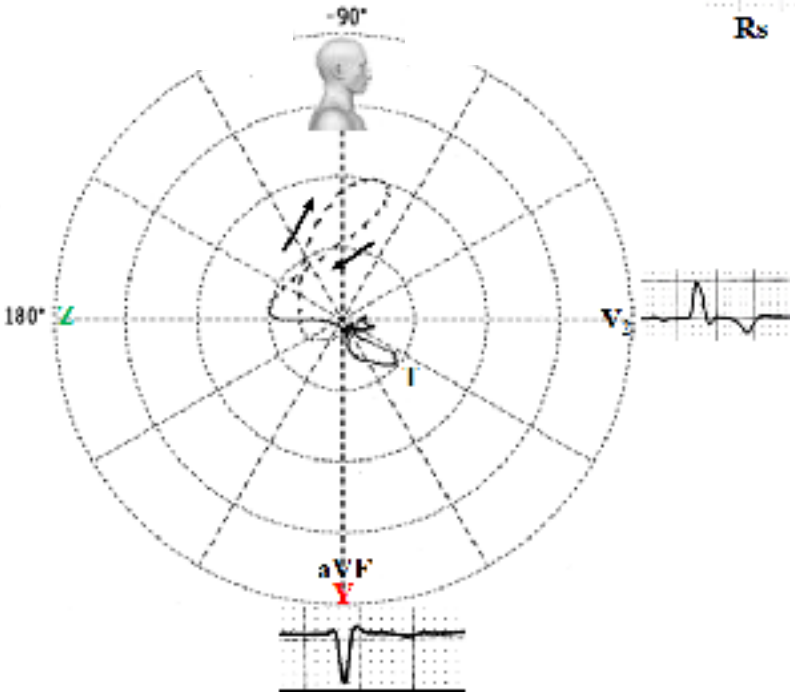


Clinical diagnosis: Mitral valve prolapse with CRBBB + LAFB and PAF.
Echocardiographic diagnosis: telesystolic prolapse with mild escape. EF: 73%.
ECG diagnosis: HR: 77 bpm, P wave of difficult visualization in the frontal plane; PR: 200 ms; SÂQRS –70°; QRSD: 150 ms; Rs from V_2 to V_6.
Conclusion: CRBBB + LAFB + prominent anterior forces (PAF). The difficult visualization of P wave in the FP may point out a certain degree of fibrosis in the atrial wall (sinoventricular conduction).

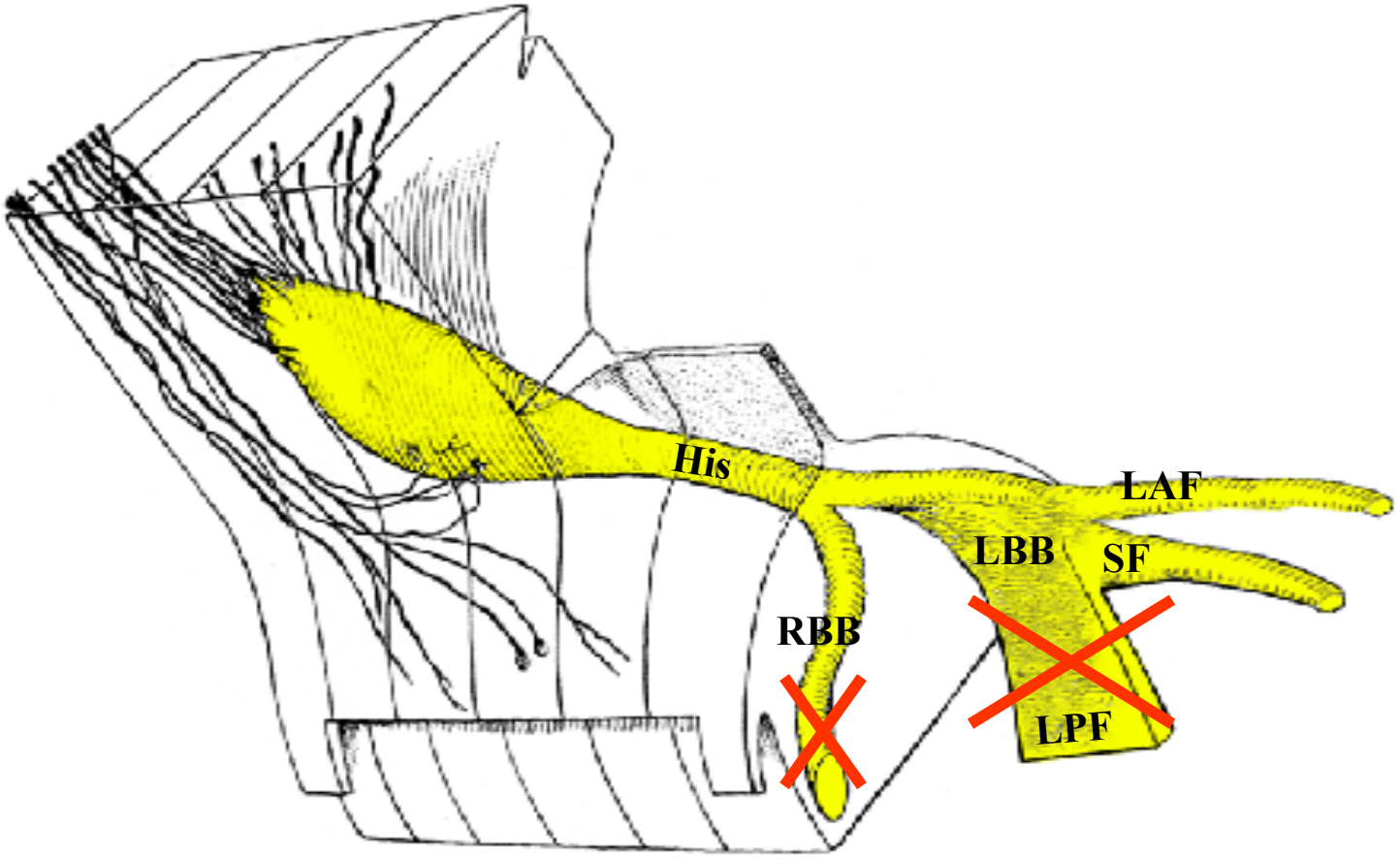
ECG/VCG correlation







Complete Right Bundle Branch Block associated to Left Posterior Fascicular Block: CRBBB + LPFB



RBBB associated to LPFB - Etiologies

This association is very unusual and its diagnosis can only be clinico-electrocardiographic. Absence of vertical heart of asthenic biotype, and clinical situations that condition RVE, such as pulmonary hypertension either primary or secondary, and lateral wall myocardial infarction.

A) Lenègre disease: in elderly patients, without apparent heart disease, maybe it should be attributed to this entity (only if possible later diagnosis shows it). The patients older than 60 years, present more frequent association with complete RBBB.

B) Aortic Valve Sclerosis: it is interesting to point out as etiologic factor, the aortic insufficiency attributed to the mechanical effect of regurgitation in jet, on the posterior portion of the left septum in the LV inflow tract; a site where the large-sized fascicle of the left branch occurs (LPF);

C) Coronary Insufficiency: frequently complicating inferior infarction. In the first world, this is the most frequent cause.

Acute post-infarction is found in 0.8% of the cases, and always involving a great extension of the septum, RB and LPF injury, and three-artery or RCA involvement;

D) Cardiomyopathy: it is found with relative frequency in our area, in chronic chagasic heart disease, particularly in patients younger than 40 years.

Etiological considerations of CRBBB associated to LPFB. Its rareness is highlighted, and the need for a clinico-electrocardiographic diagnosis.

RBBB associated to LPFB: Etiologies – ECG criteria

It is characterized by the typical pattern of RBBB in precordial leads and SÂQRS deviation around +120° in the frontal plane (between +90° and ± 180°).

1) PRi interval: frequently prolonged (>200 ms). If PRi is associated to RBBB (60 to 70% of the cases) and LAFB, it indicates trifascicular block: ARBBB + LAFB + incomplete LPFB;
2) QRS duration of 120 ms or more;
3) Pattern of RBBB with SÂQRS shifted to the right near +120° in absence of vertical heart, any clinical condition that may cause RVE and lateral infarction;
4) I and aVL, rS and inferior leads of the qR type; q is obligatory in III and aVF;
5) R wave of III greater than R wave of II;
6) Notch in descending ramp of R wave in inferior leads;
7) Right precordial leads with rsr', rsR pattern; R with notch similar to M;
8) Broad S wave in left leads with a duration greater than 40 ms;
9) S wave of broad left leads with a duration in adults greater than 40 ms;
10) Intrinsicoid deflection in V_1 ("R peak time") when there is complex in M equal or greater than 50 ms and normal in V_5 and V_6 (**Rusconi 1980**);
11) Initial forces heading to the left, above and the front, and final forces with the maximal vector heading to the back and below with a middle angle of 152º between both, are considered the most characteristic ones;

RBBB associated to LPFB: Etiologies – VCG criteria

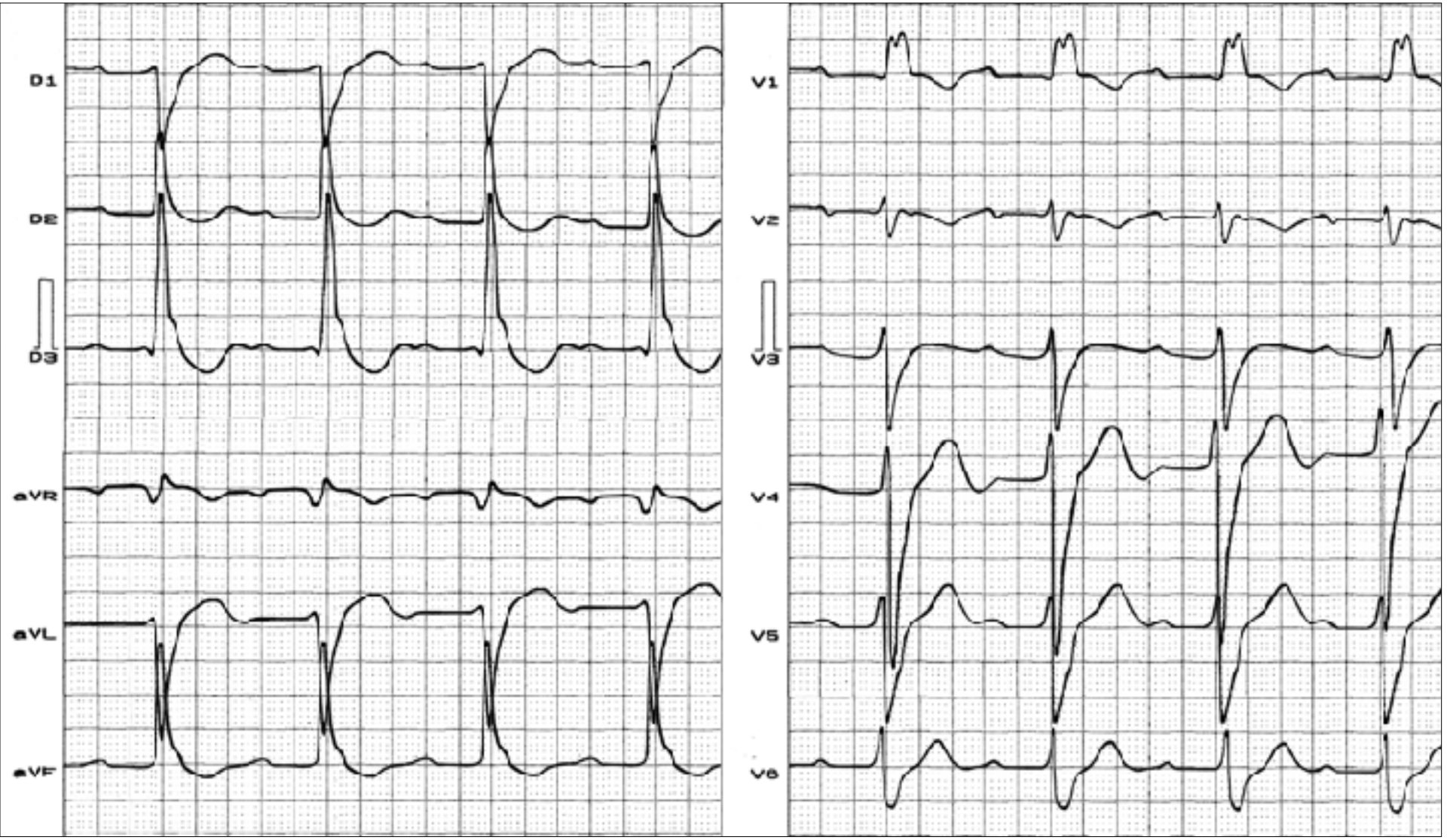
Frontal Plane:

1) Initial vectors with delay, pointing upward and to the left;
2) Clockwise rotation;
3) More than 40% of the QRS loop, to the right of the Y line (**Varriale 1972**);
4) SÂQRS to the right of +90°;
5) Final vectors with delay, always to the right.

Horizontal Plane:

1) Initial vectors to the front and the left; clockwise rotation;
2) Rotation in eight (Cabrera type or Kennedy type II) or clockwise rotation (type III of Kennedy);
3) Maximal vector heading to the right;
4) Terminal appendage with delay to the right and to the front.

Name: VFD; **Date:** 08/02/1995; **Age:** 45 y.o.; **Number:** 538; **Gender:** F; **Race:** W.; **Weight:** 65 Kg; **Height:** 1.65 m.; **Biotype:** athletic; **Medication in use:** digoxin 0.25 mg, furosemide 40 mg, aldactone 25 mg, enalapril 10 mg 2x



Clinical diagnosis: severe aortic failure.
ECG diagnosis: first degree AV block + CRBBB + LPFB.

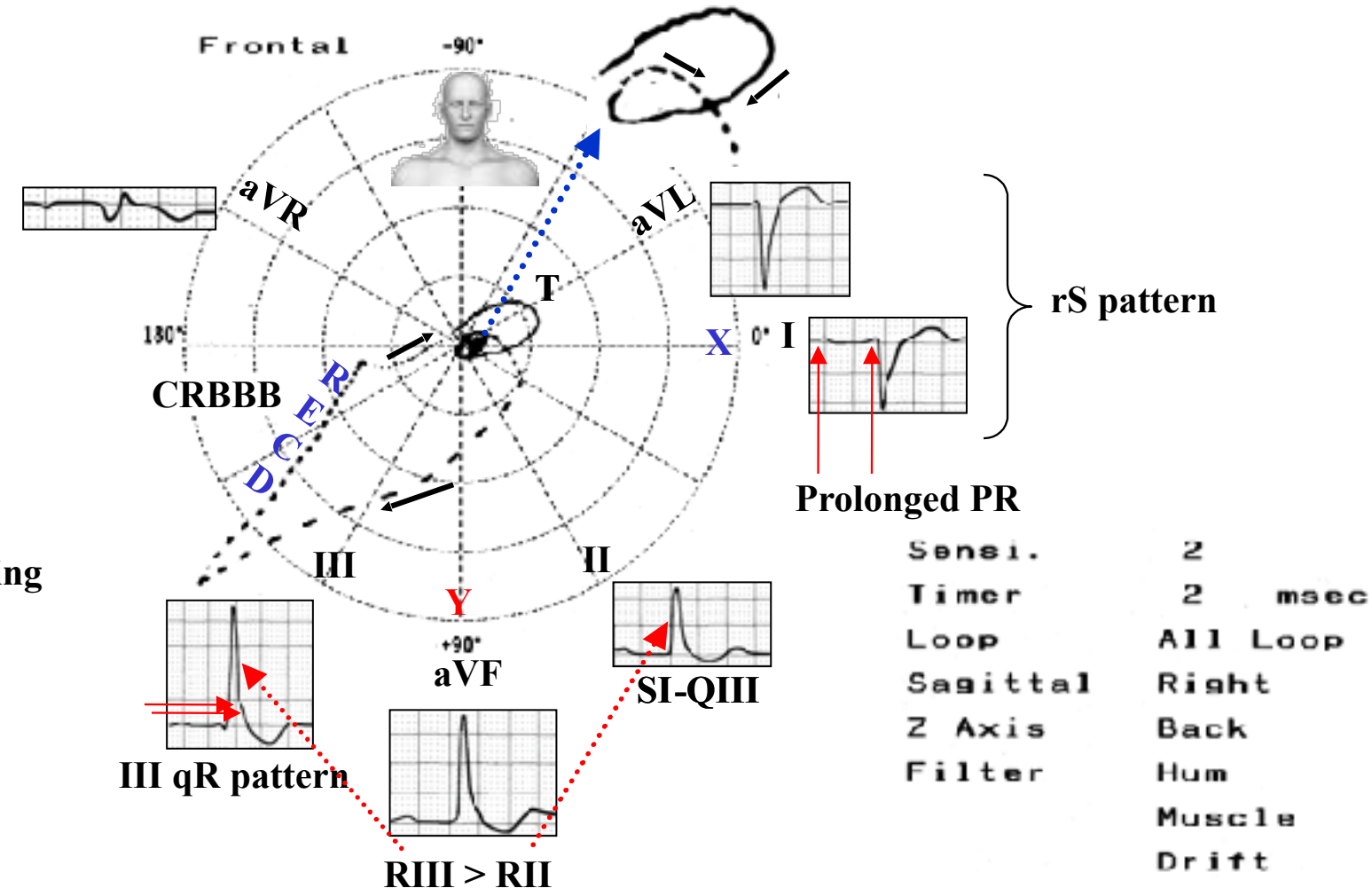
Name: VFD; **Date:** 08/02/1995; **Age:** 45 y.o.; **Number:** 538; **Sex:** F.; **Race:** W.; **Weight:** 65 Kg; **Height:** 1.65 m; **Biotype:** athletic; **Medication in use:** Digoxin 0.25 mg, Furosemide 40 mg, Aldactone 25 mg, Enalapril 10 mg 2X

ECG/VCG correlation in the FP

40% or more of QRS loop below and to the right: LPFB + CRBBB

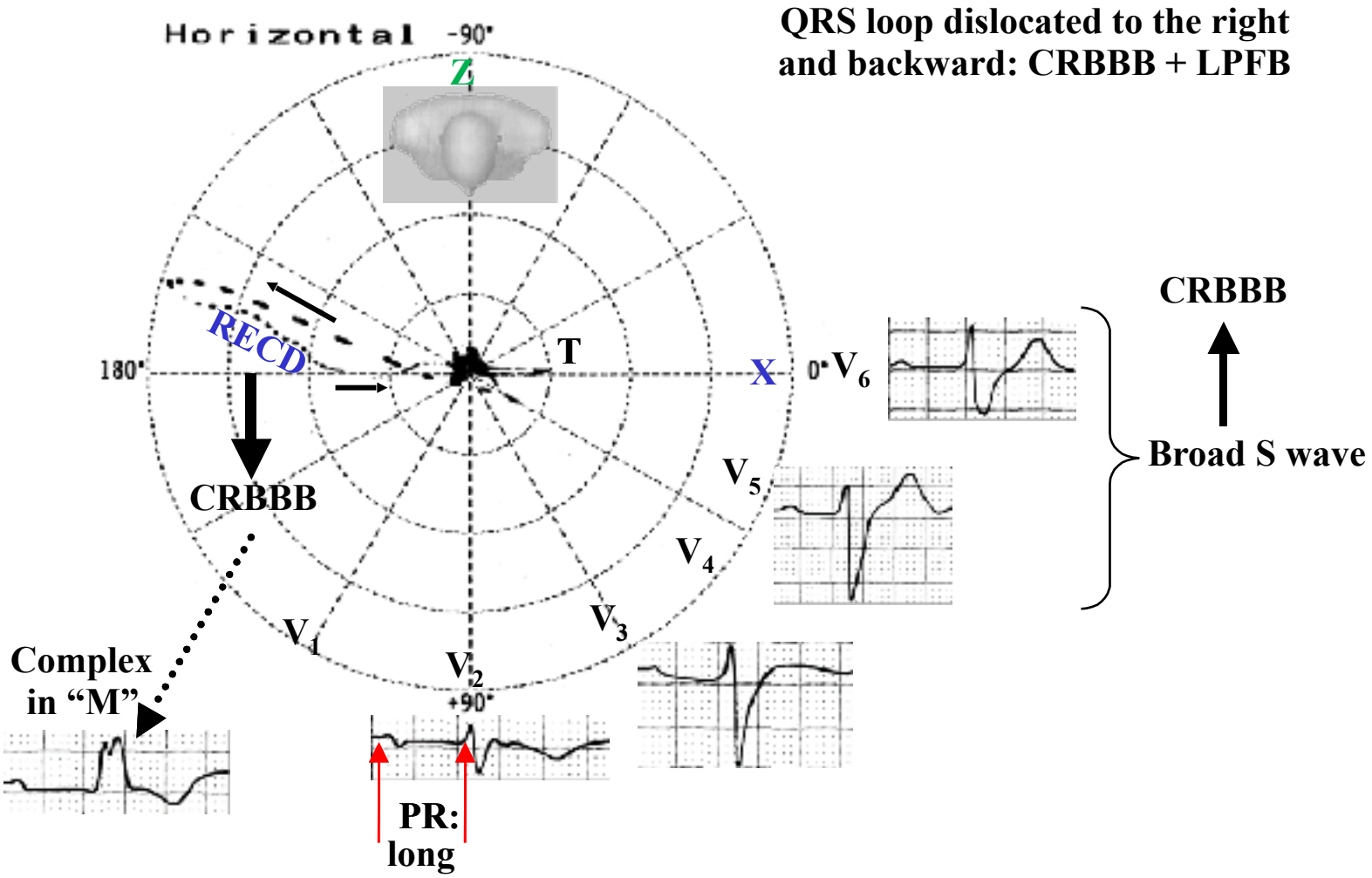
SÂQRS with deviation to the right around +100º

Notch in descending limb of R (middle final)

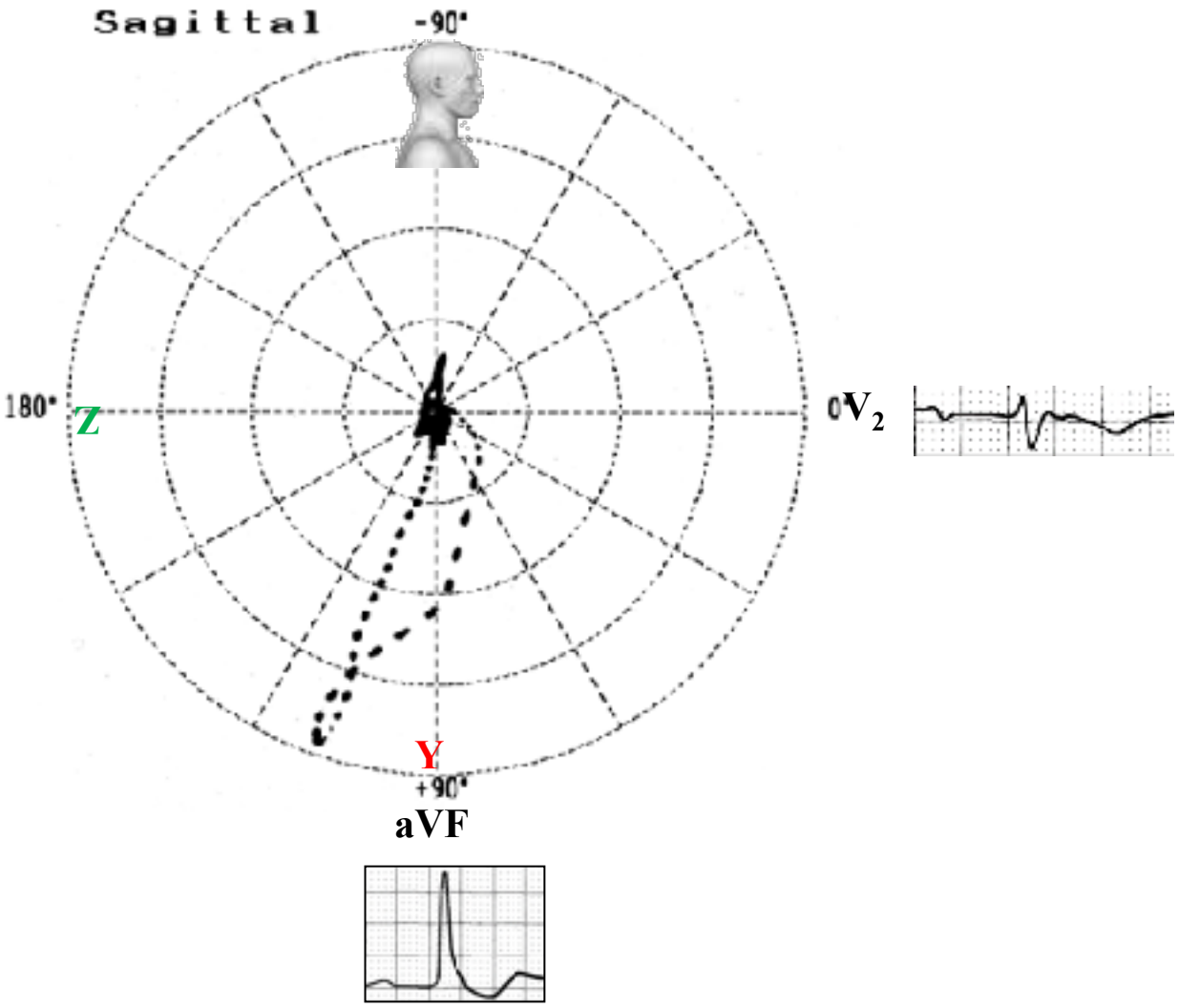


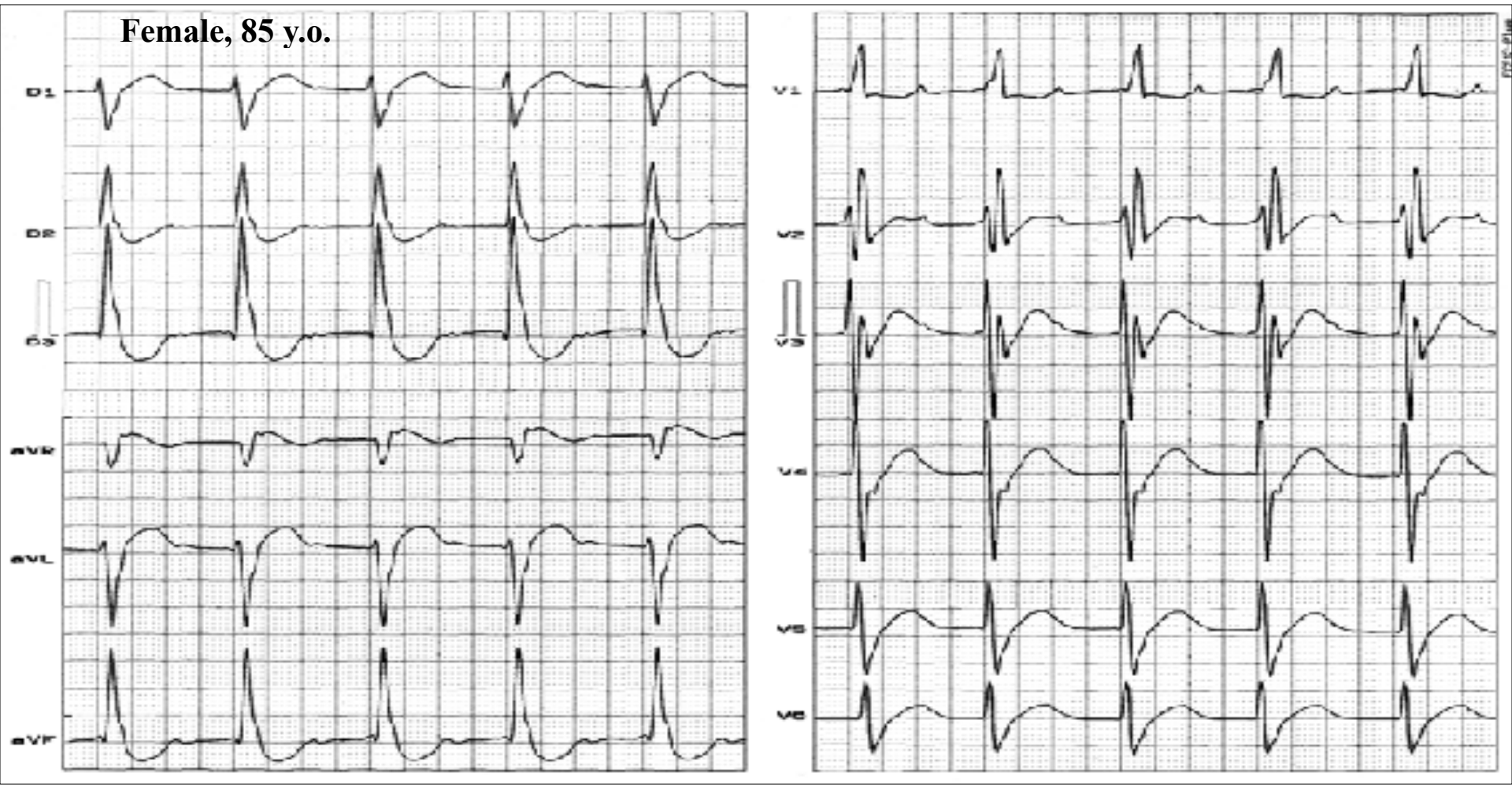
ECG/VCG correlation in the frontal plane of the same case, which shows the characteristics of LPFB associated to CRBBB. The initial vectors heading upward and to the left are highlighted, as well as shift of the axis to the right, RIII > RII and ECD of CRBBB.

ECG/VCG correlation in the Horizontal Plane



ECG/VCG correlation in the horizontal plane, where we see dislocation backward and to the right of the loop, ECD proper of CRBBB, and long PR interval. The loop dislocated to the right and backward is characteristic of CRBBB and LPFB.


Sagittal
-90°
180°
Z
0°
V2
Y
+90°
aVF



Autopsy diagnosis: Lèv disease, sclerosis of the left side of the "cardiac skeleton". This entity along with Lenègre disease is called progressive cardiac conduction defect (Lev-Lènegre).

ECG diagnosis: first degree AV block (PR 35 ms) + LPFB + CRBBB: possible trifascicular block. Digitalis effect. Surface ECG cannot provide accuracy for the topography of the block. To consider it trifascicular, the block should be found below the His bundle. An electrophysiology study is necessary for this.

ECG of an elderly lady, 85 years old, carrier of Lèv disease with 1st degree AV block, LPFB and CRBBB. Repolarization is observed with the shape of a "spoon" suggesting digitalis effect.

Masquerading Right Bundle Branch Block concept

We know today that the ECG complex coined since Richman as "masquerading bundle-branch block"(**Richman 1954**) is essentially a complete RBBB and high degree LAFB, with further modifications of the initial and final QRS vectors, so that standard leads, and at times the left precordial leads, resemble left bundle branch block (**Schamroth 1975**).

Masquerading BBB is not a specific entity but is an electrocardiographic complex; the result of RBBB with varying combinations of LAFB, intramural left ventricular block, left ventricular enlargement/hypertrophy and anterior myocardial infarction or fibrosis.

Since the pioneer Rosenbaum´s et al studies (**Rosenbaum 1968; Rosenbaum 1973**) we know two ECG types of "Masquerading" Bundle-Branch Block. There is a third type that is the association of both:

I. ***The "Standard Type" or Standard Masquerading Bundle-Branch Block:*** *consisting of the pattern of left bundle-branch block (LBBB) in the limb leads and right bundle-branch block (RBBB) in the unipolar precordial leads.*
II. ***The "Precordial Type" or Precordial Masquerading Bundle-Branch Block***
III. ***The Standard and Precordial Masquerading Bundle-Branch Block in Association.***

I. The "standard type" (*standard masquerading right bundle-branch block)*

In *standard masquerading right bundle-branch block* the presence of a high degree left anterior fascicular block (LAFB) obscured totally or partially the diagnosis of right bundle branch block (RBBB) only in the frontal plane by abolishing (or it becomes very small) the final broad S wave in the leads I and aVL (**Ortega-Carnicer 1986**). Consequently, the limb leads may resemble left bundle branch-block (LBBB) although the precordial ECG remains typical for CRBBB. The precordial leads reflect the feature of RBBB. Figure 2

Conditions necessary for the presence of standard masquerading right bundle branch block

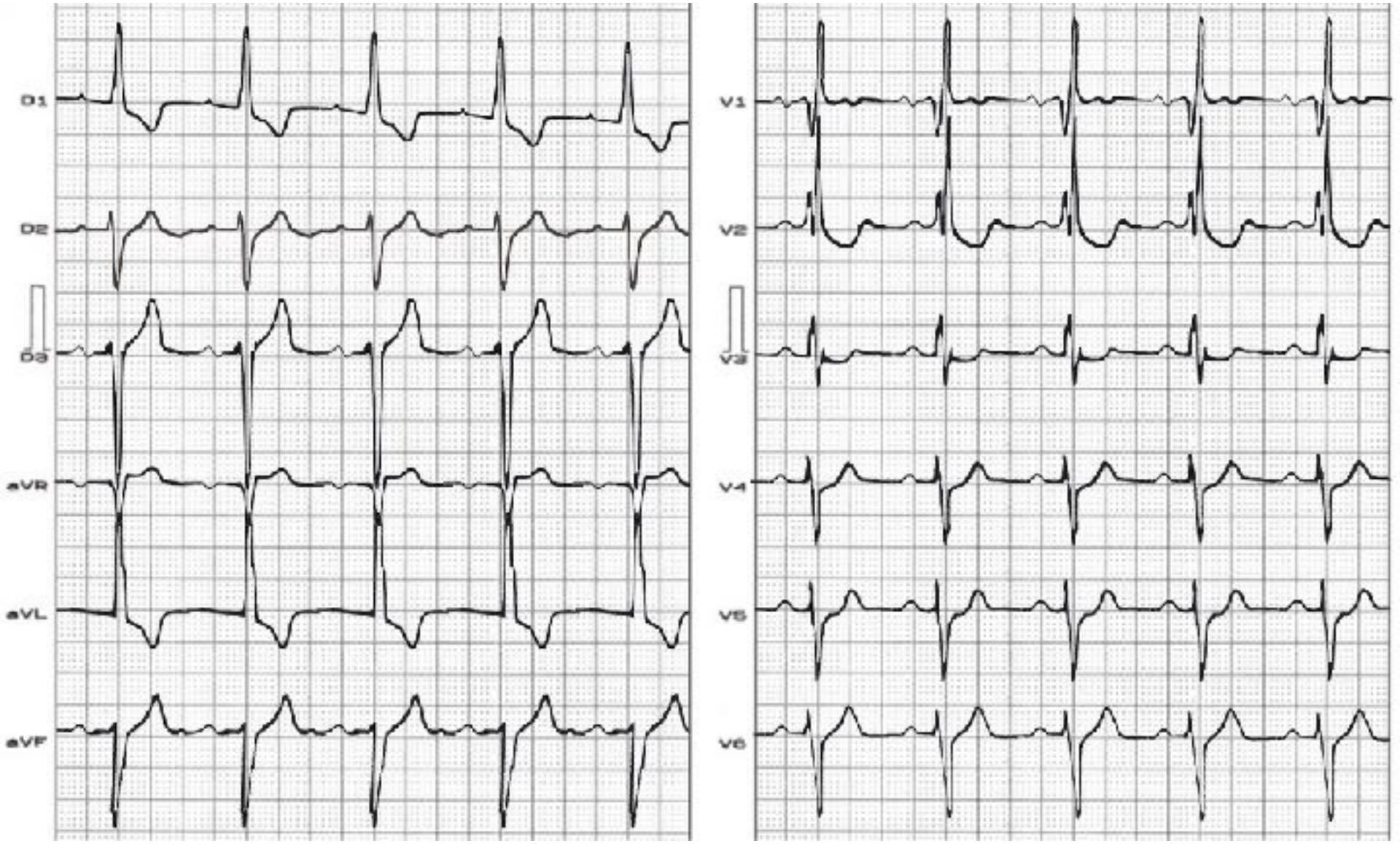
1. High degree of left anterior fascicular block
2. Right Bundle-Branch Block
3. Bilateral bundle-branch lesions of considerable intensity, which do not completely disrupt the continuity of the branches (**Unger 1958**)
4. Left Ventricular Enlargement or Hypertrophy (LVE/LVH) and marked biventricular hypertrophy
5. Localized block in the left ventricle.
6. Frequent severe fibrosis, or truly massive myocardial infarction mainly in anterior wall.

Etiologies

1) Coronary heart disease
2) Long standing systemic hypertension
3) Cardiomyopathy. E.g. Chronic Chagasic myocarditis
4) Lev´s disease
5) Association of the previous ones.

Prognosis: always poor.

Example of *Standard Masquerading Right Bundle-branch Block*



Extreme QRS left axis deviation (SÂQRS -50°), SIII>SII: LAFB. The limb leads show LBBB-like pattern, but the precordial leads show RBBB. SIII >15 mm: Type IV Rosenbaum LAFB: association of LAFB + LVE or LVH.

Symptomatic elderly man (syncope) with critical coronary obstruction on LAD treated 6 months ago with stent implantation and current dynamic dromotropic disorder on his successive ECGs

Patient of 67 years carrying Coronary Artery Disease (CAD), with the antecedent of stent implantation on the LAD artery 6 months ago.

The first ECG was performed on admission at 02,10.14, 08:21' AM.

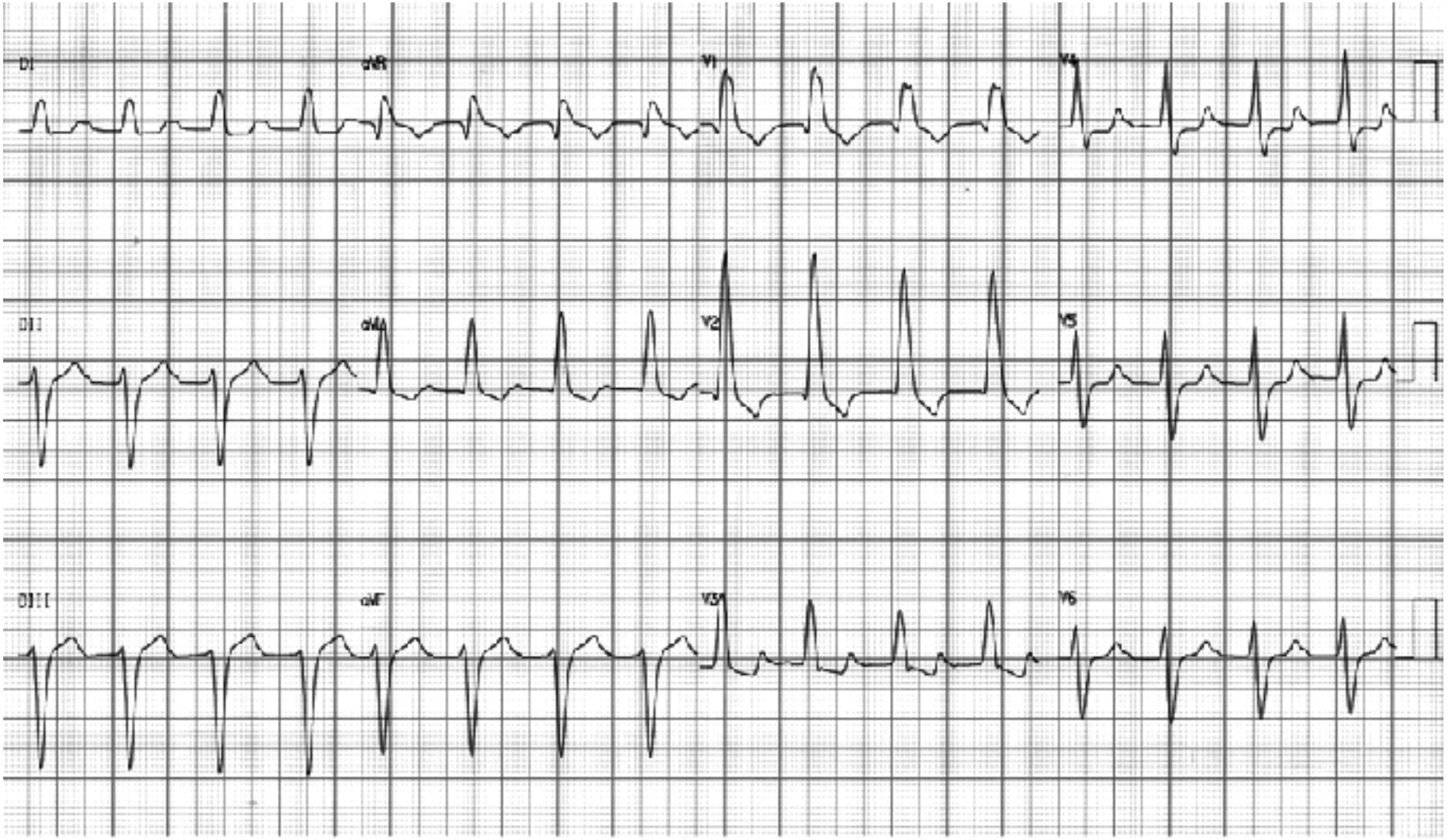
The second one was recorded shortly after the syncopal episode occurred approximately two hours after admission (02.10.14, 10:38').

The 24 h Holter monitoring revealed intermittent LBBB pattern.

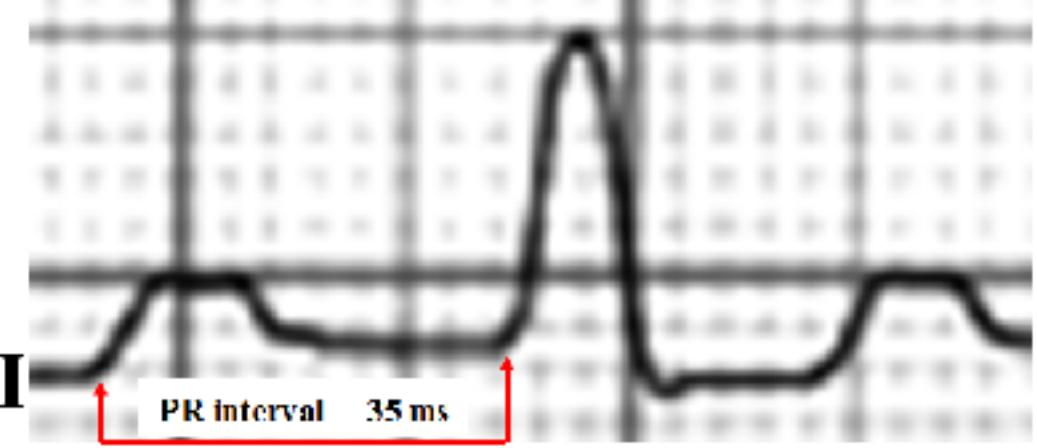
The electrophysiology study (EPS) revealed HV interval prolongation = 84 ms (normal 55 ms). Concomitantly, the AH interval also was prolonged.

Raimundo & Andrés

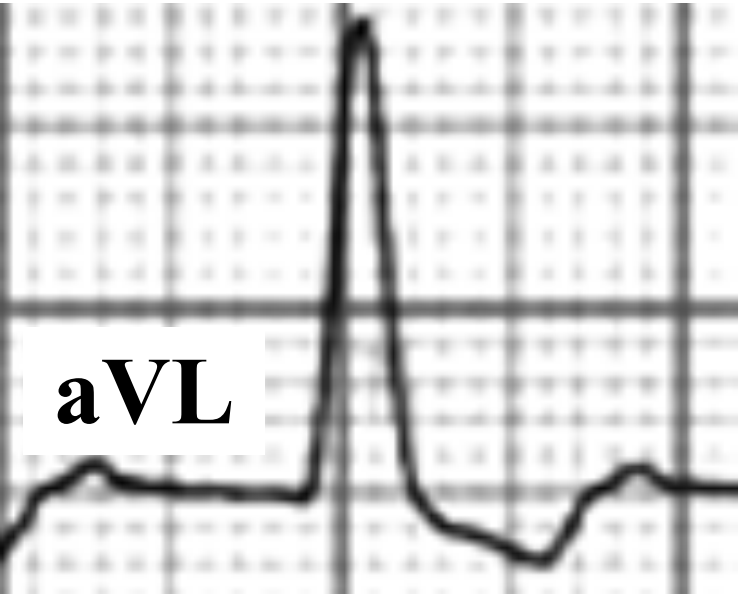
ECG1=02-10-14 08:21´ Admission

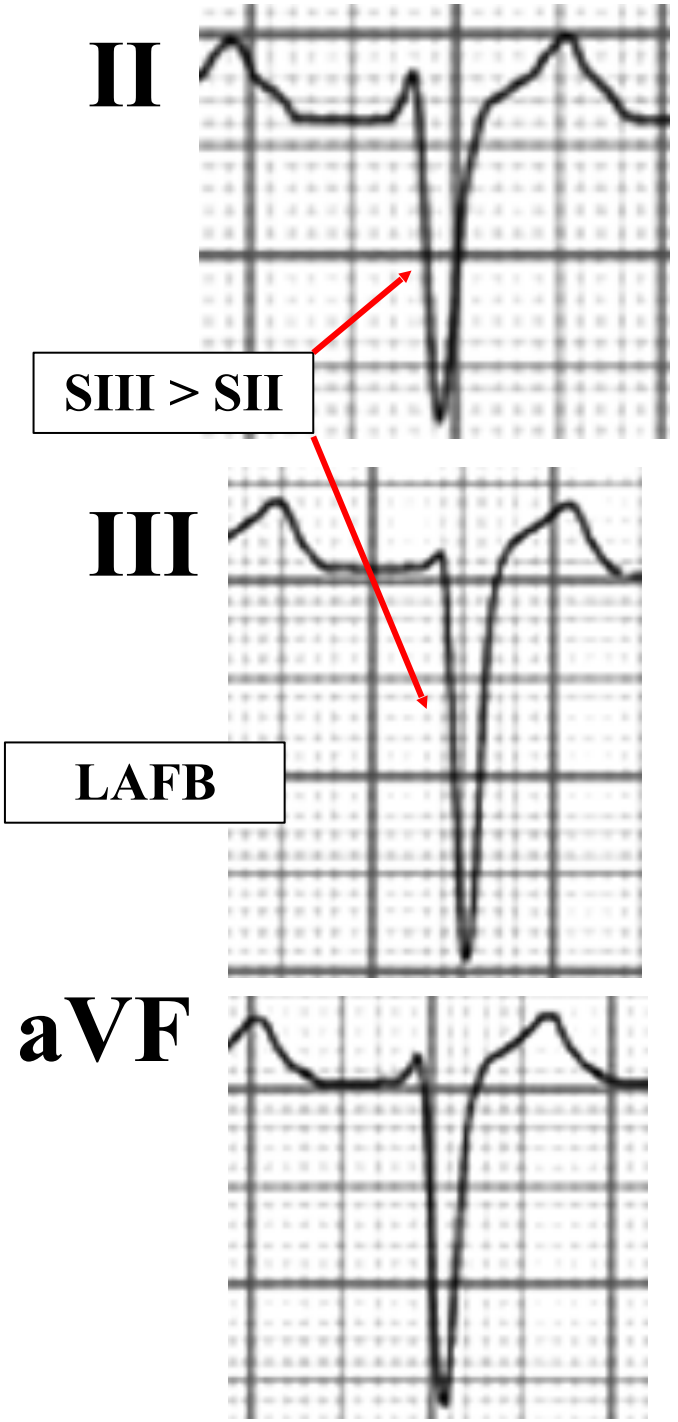


ECG diagnosis: Standard Masquerading Right Bundle-Branch Block + LAFB + LSFB + prolonged PR interval: probable claudication of posteroinferior fascicle suggesting tretrafascicular block. Why? See next slide.

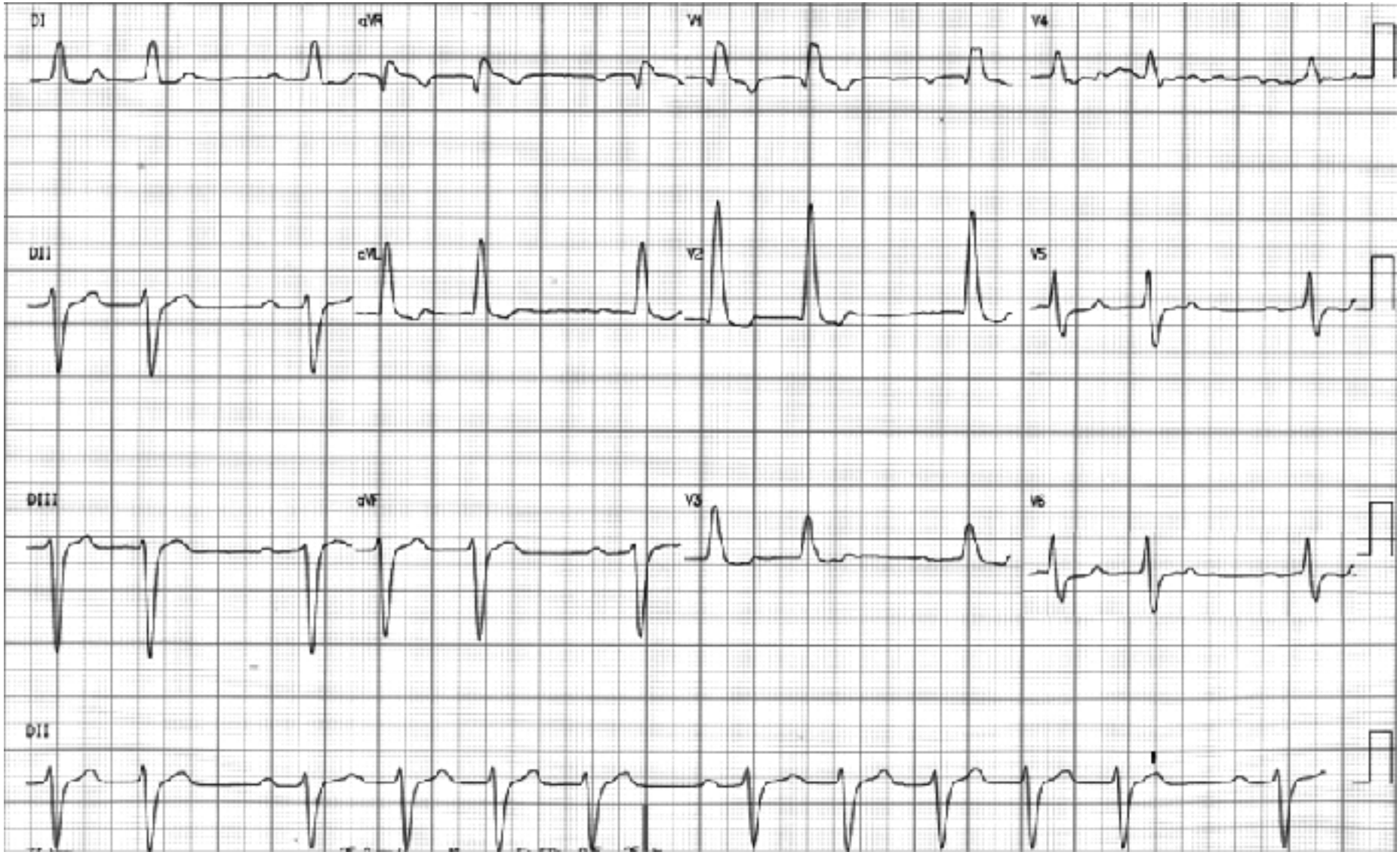


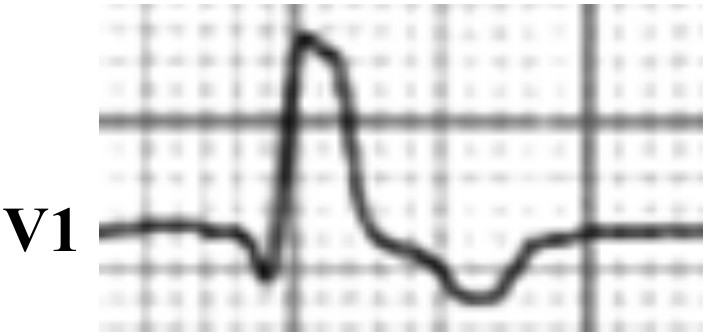
In *standard masquerading right bundle-branch block* the presence of a high degree LAFB obscured totally or partially the diagnosis of RBBB only in the FP by abolishing (or it becomes very small) the final broad S wave in the leads I and aVL. Consequently, these limb leads may resemble LBBB although the precordial ECG remains typical for CRBBB.

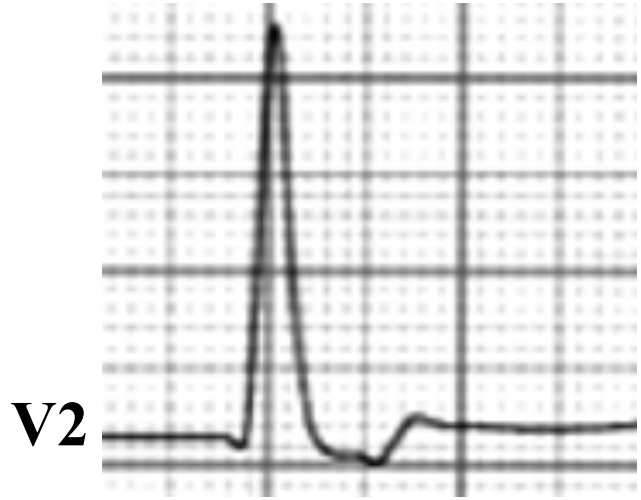


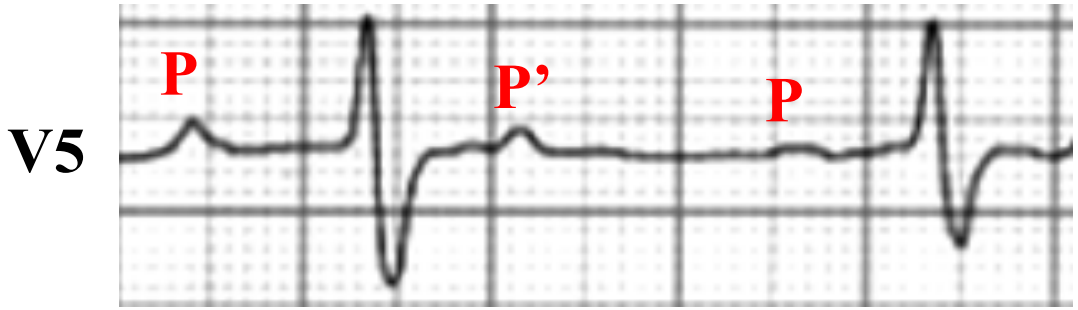


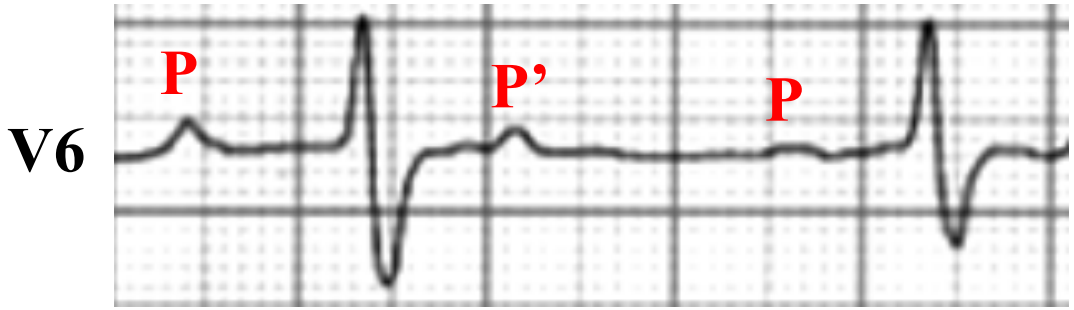
ECG2= 02-10-14 10:38´







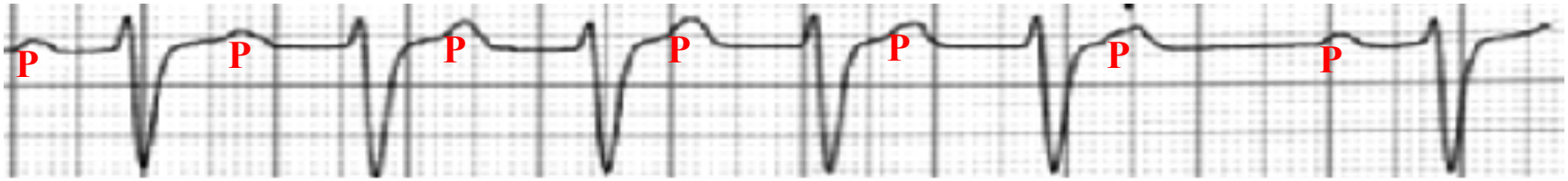




2:1 2nd degree AV block or pseudo 2:1 2nd degree AV block, the second (non conducted) and third P waves (P') have a different morphology from the conducted P wave, being ectopic, premature and not conducted: sinus rhythm with blocked atrial extrasystoles. P' waves are atrial extrasystoles.

V1: qR pattern. RBBB with septal myocardial infarction.
V1-V2: qR pattern. Increased intrinsicoid deflection of V_1 and V_2, R wave "in crescendo", R wave voltage of $V_1 \geq 5$ mm, small q wave in V2 or V1 and V2, R wave of V2 > 15 mm, absence of q wave in V5, V6 and I (by absence of the first septal vector 1_{AM}): Left Septal Fascicular Block.

ECG 2: Long II suggests type I AV block (Wenkebach): progressive PR prolongation



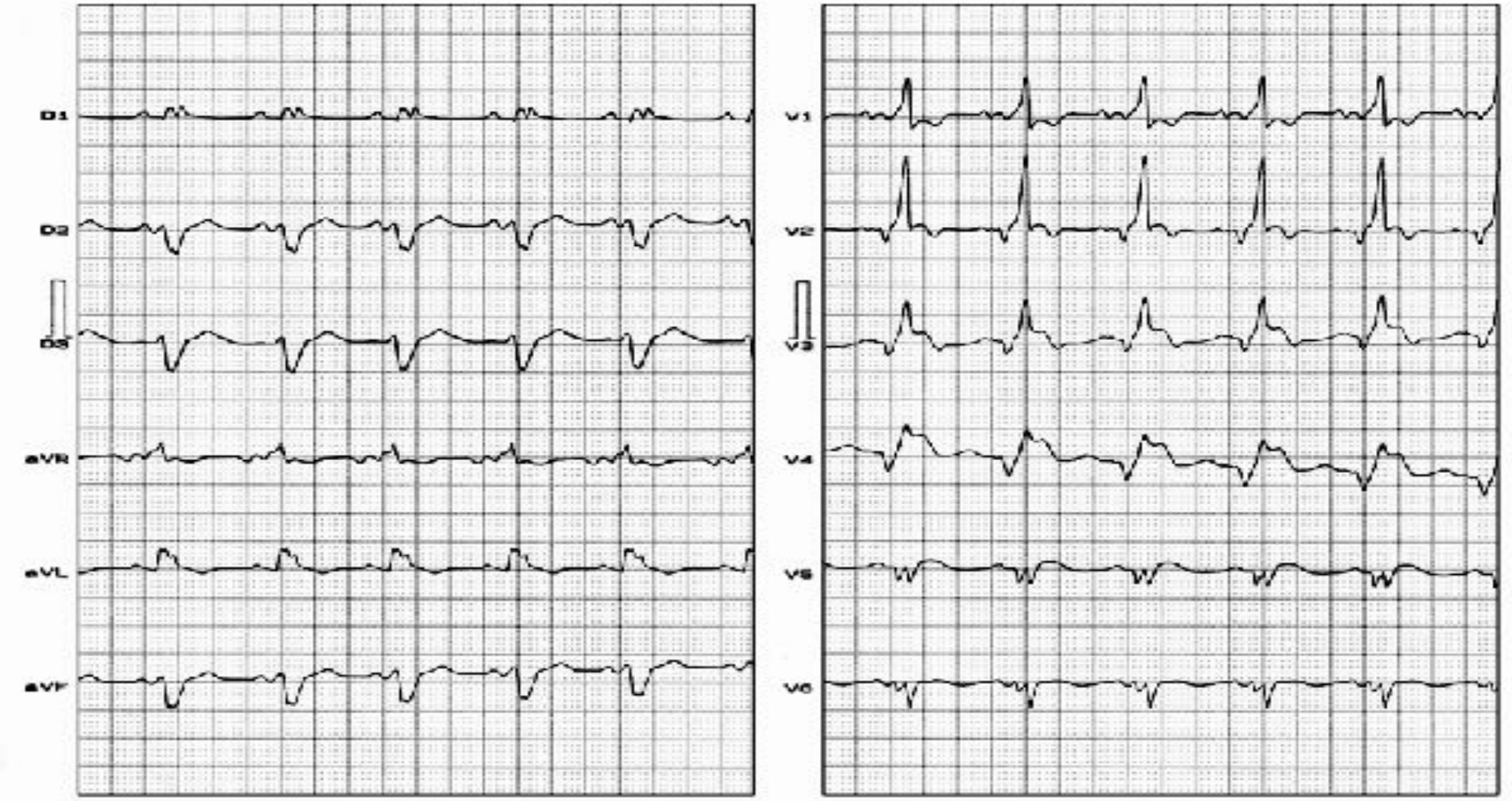
Conclusions:

1. First degree AV block;
2. Second degree AV block Mobitz type I (Wenkebach) in the second ECG and pseudo 2:1 2nd degree AV block?
3. LAFB: extreme left axis deviation SIII > SII;
4. LSFB: qR pattern. Increased intrinsicoid deflection of V_1 and V_2, R wave "in crescendo", R wave voltage of $V_1 \geq 5$ mm, small q wave in V2 or V1 and V2, R wave of V2 > 15 mm, absence of q wave in V5, V6 and I (by absence of the first septal vector 1_{AM}).
5. Standard masquerading RBBB
6. RBBB with septal MI: qR pattern in V1
7. First degree AV block + bifascicular left fascicular block + RBBB = tetrafascicular block (this is a new nomenclature used by us.)
8. LBBB on Holter monitoring is indicative of alternanting BBB, such as the following next three slides.

EPS study shows HV and AH prolongation and transient LBBB pattern in Holter monitoring.

The four main developmental ECG patterns of standard masqueranding type

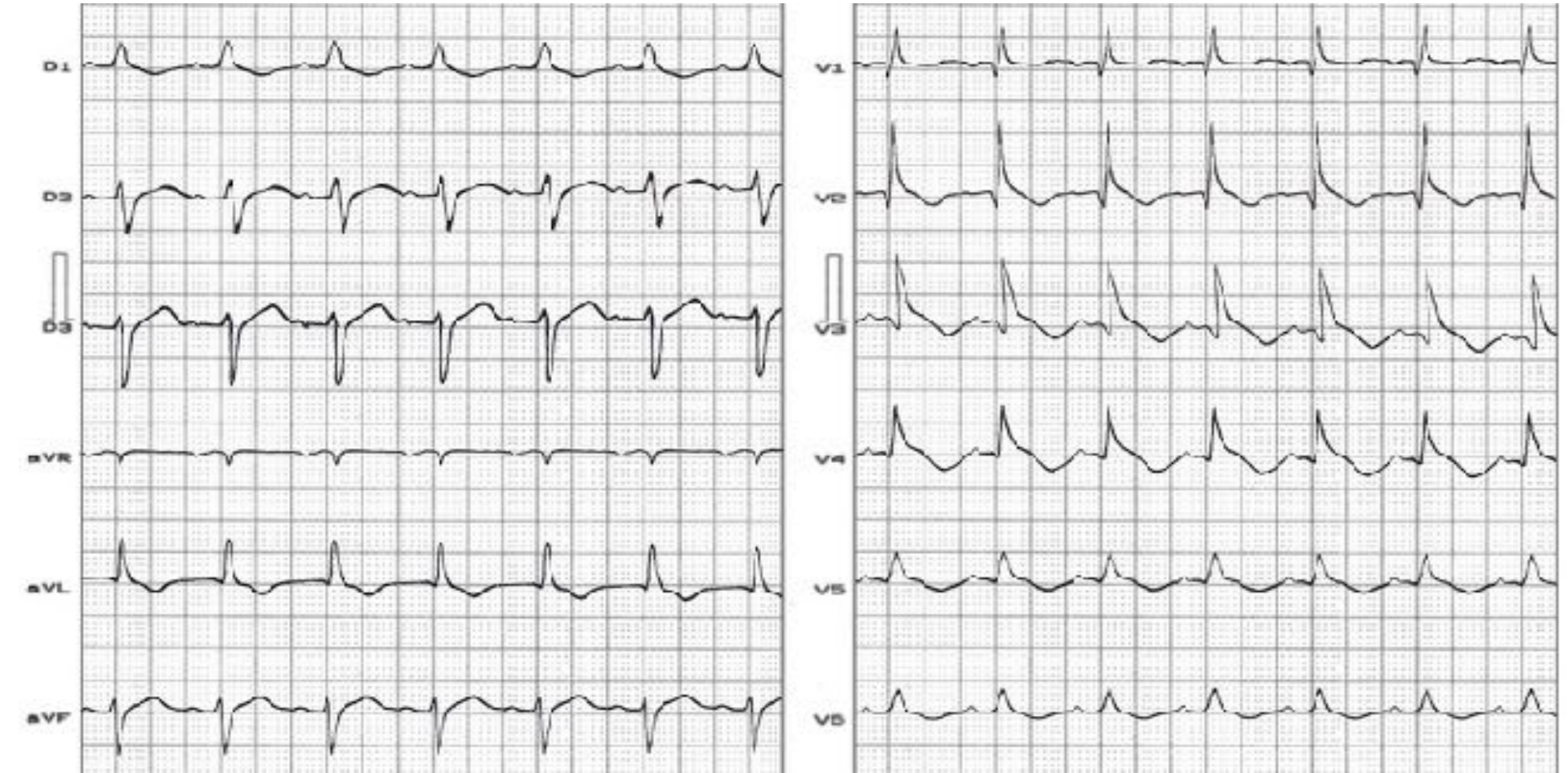
	aVL	I	II	III
1. Uncomplicated LAFB: QRS duration <120 ms	qR	qR	rS	Rs (SIII>SII)
2. LAFB with CRBBB: QRS duration ≥120 ms	qRS	qRS	rS with notch on ascending ramp of S	Rs with notch on ascending ramp of S
3. LAFB with CRBBB and diminution of the final QRS vector. QRS duration≥120 ms	qR	qR	rS	rS
4. LAFB with CRBBB and diminution of the final QRS vector and diminution of the initial QRS vector.	R	R	QS	QS



Acute extensive anterior myocardial infarction associated with standard masquerading bundle branch block: LAFB associated with RBBB. Extreme QRS left axis deviation (SÂQRS -70°), SIII>SII: LAFB. The limb leads show an atypical LBBB-like pattern (isolated r wave in I and aVL), but the right precordial leads show a RBBB.

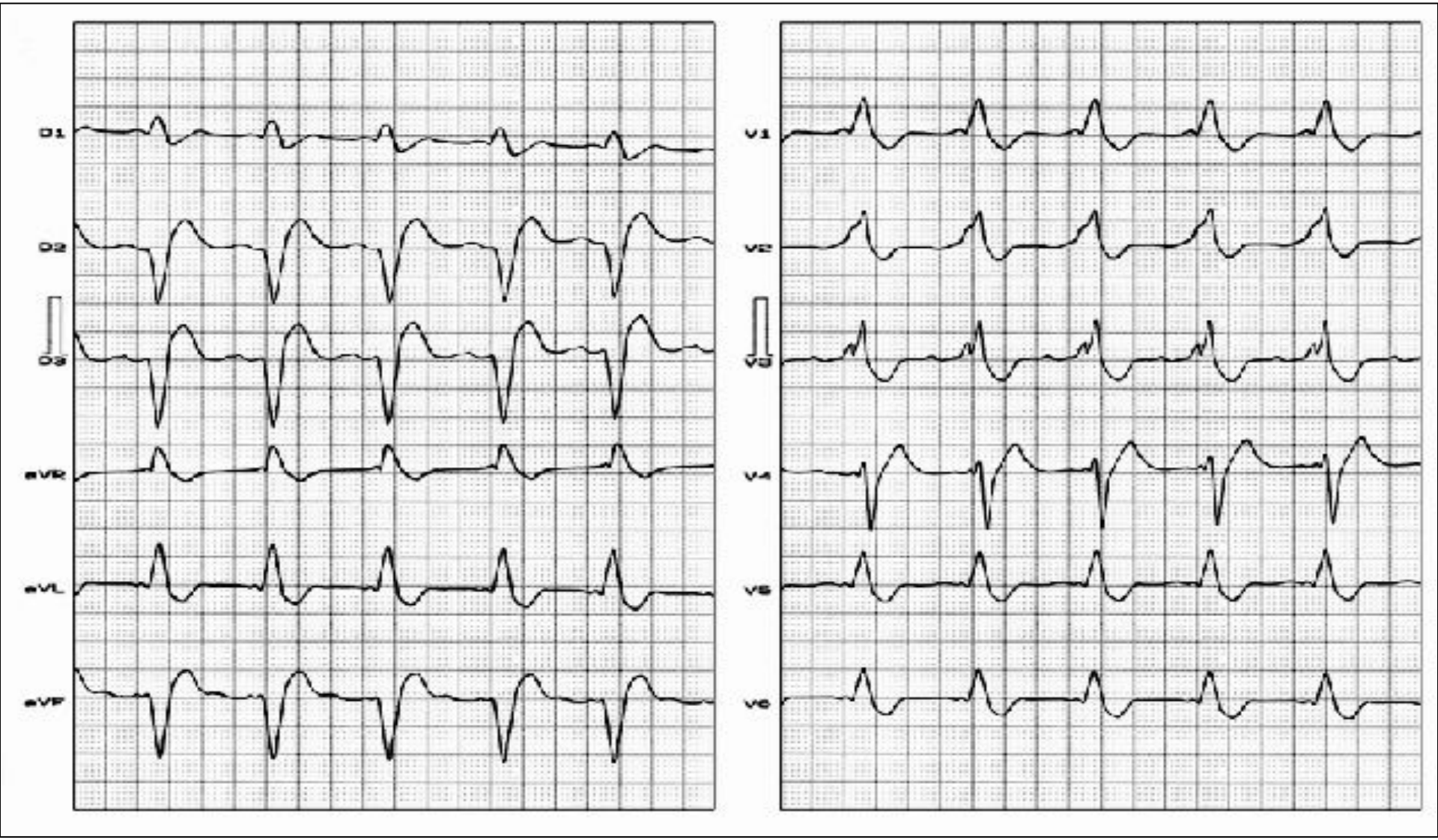
II. The precordial type (*precordial masquerading right bundle-branch block)*

This type shows the pattern of CRBBB in the right precordial leads and complete left branch block pattern (CLBBB) in the left-side precordial leads. This results from CRBBB associated with severe left ventricular hypertrophy/enlargement (LVH/LVE), a localized block in the anterolateral wall of the left ventricle often due to myocardial infarction, and usually LAFB. Presumably, the intramural left ventricular block, together with the LVH or the LAFB, or both, produce predominant leftward forces which tend to cancel out the late rightward forces of the RBBB in the left precordial leads. Finally, masquerading bundle-branch block can be associated with severe and diffuse conduction system disease, and patients with this finding may require permanent pacemaker implantation, especially if they are symptomatic (**Kowey 1989**).



III. The Standard and Precordial masquerading bundle-branch block in association

In this case the limb leads show an apparent Left bundle-branch block pattern with extreme left axis deviation (LAFB) and the precordial leads exhibit the pattern of CRBBB in the right precordial leads and LBBB pattern in left precordial leads V5-V6. Additionally, an abnormal Q waves are frequently present in the right precordial leads



Case report

RMC female patient, 36 years old, Caucasian, married, housewife, basic education, born and raised in Cametá, Pará (PA) Brazil.

Main complaint: tiredness on mild exertion and leg swelling for four months.
For the past four months she began displaying symptoms of chest discomfort not related to exertion, epigastric pain and fatigue with great efforts initially, progressing rapidly to moderate and slight, followed by the appearance of swelling in the legs and abdomen. One day before the consultation she had been notified by the municipality sanitary authorities as a carrier of Chagas disease and due to complaints was referred to the local tertiary referral Hospital for cardiological evaluation.
At the time when seeking medical attention she was diagnosed with congestive heart failure and treated with association of furosemide 80mg/daily, spironolactone 25 mg, enalapril maleate 20 mg 2 x daily, and carvedilol 25 mg 2 x day. Enalapril maleate was suspended in a few days due to presenting very low blood pressure. At the time of consultation the patient was in New York Heart Association (NYHA) functional class III even on optimized medication. (Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20–100 m). Comfortable only at rest.)

Personal history: denied hypertension, diabetes mellitus, dylipidemia, smoking, or any other addiction.

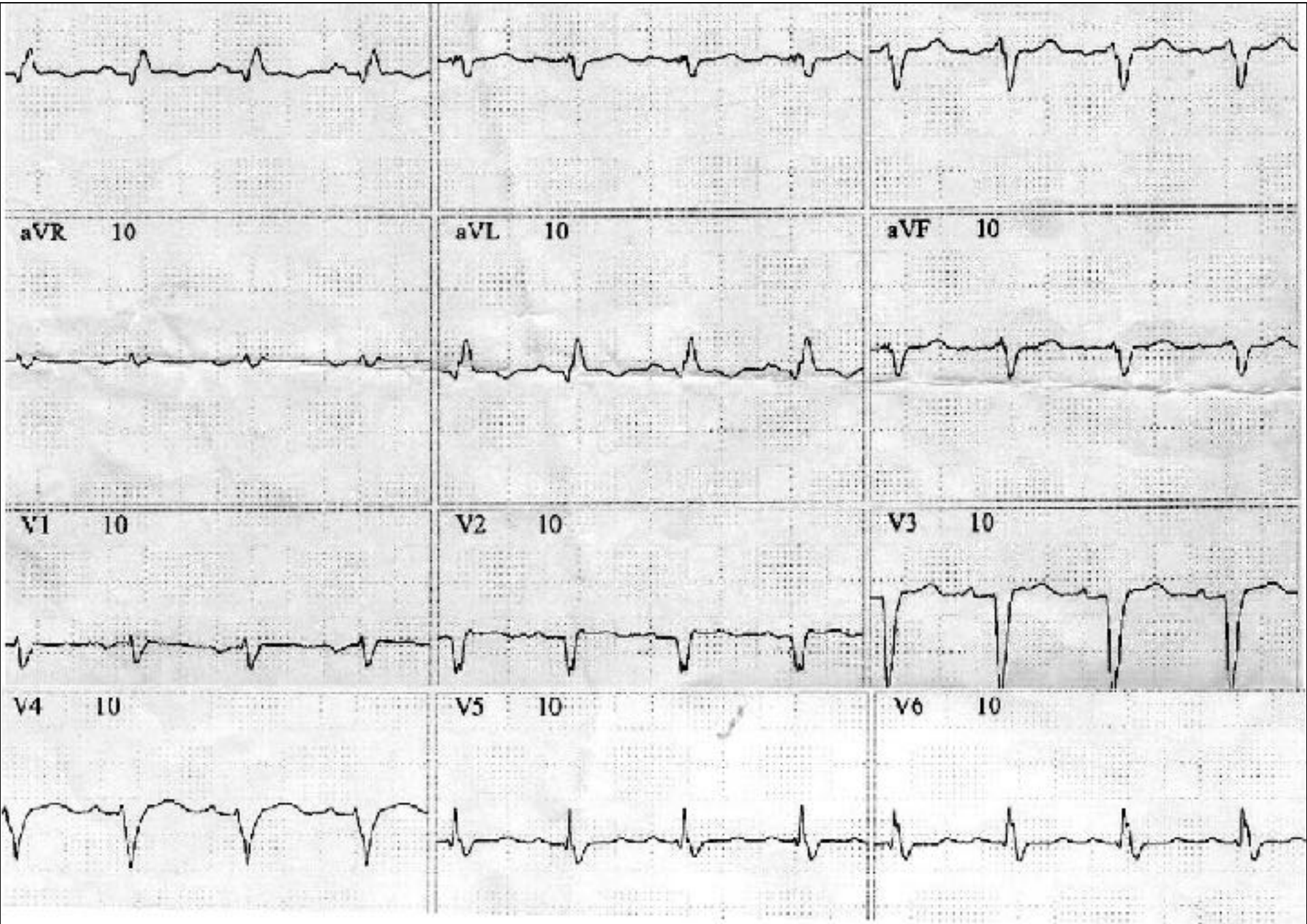
Family history: Nothing of note.

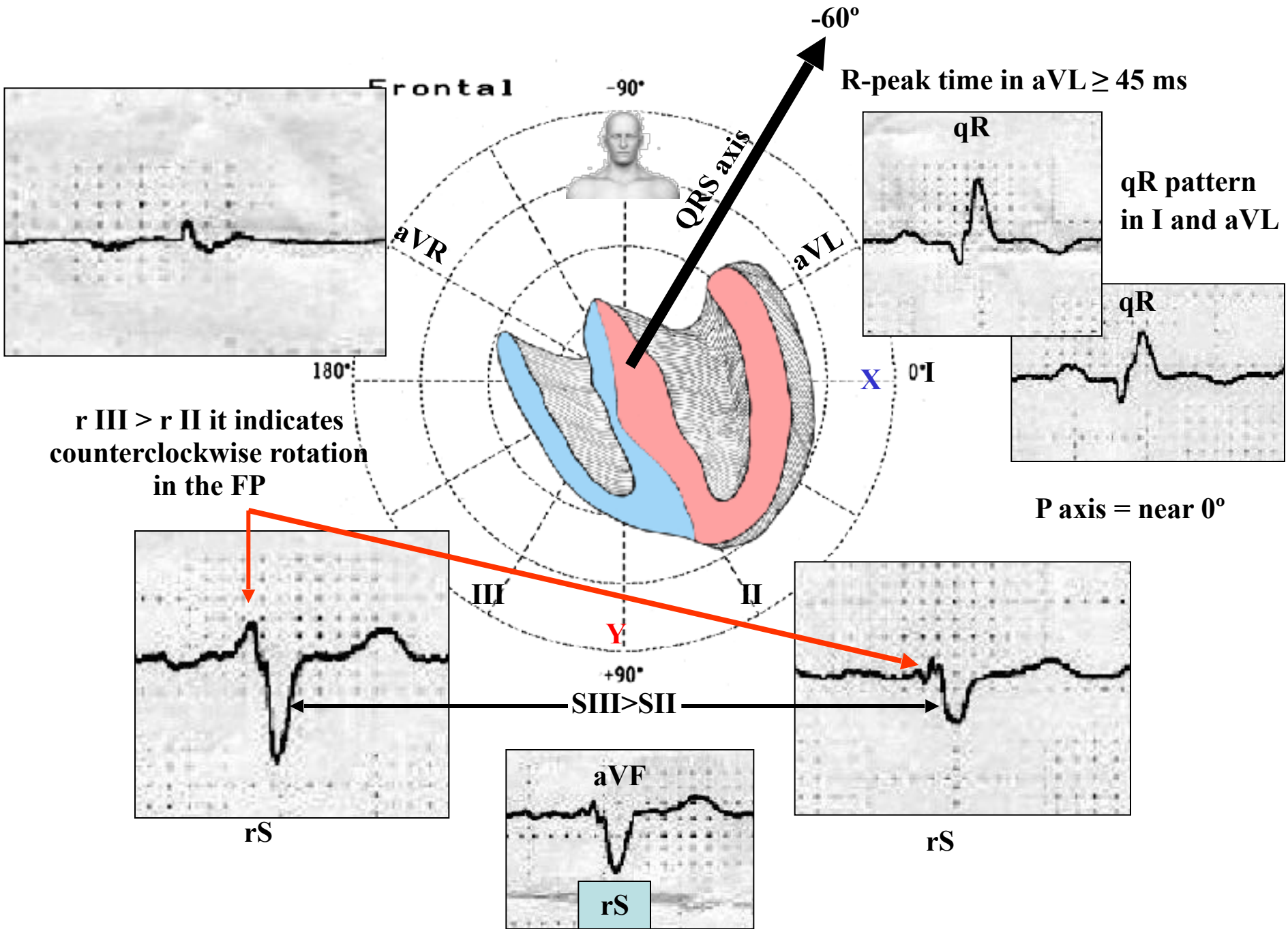
Epidemiological background: In spite of residing in an urban area, her house was made of wood and mud roofed and with no backyard. Food habits: red meat, poultry and fish. She frequently ate the “açai” fruit (***Euterpe oleracea***) bought in street booths.

Physical Examination

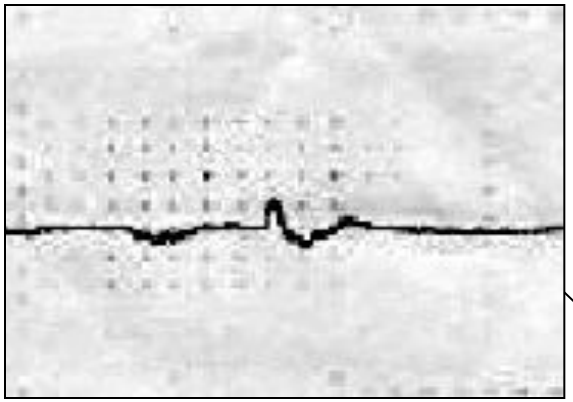
Vital signs: blood pressure 84/56 mmHg, heart rate 96 bpm, dyspnea at rest +++/4, no fever, pale skin and mucous membranes ++/4, acyanotic.
Neck: Distended neck veins, JVD to 12 cm. Carotid arteries without bruits.
Lungs: pulmonary auscultation: bilateral vesicular murmur. Absence of adventitial sounds.
Heart: visible and palpable ictus cordis in the sixth intercostal space on anterior axillary line, not covered with two fingertips. Arrhythmic heart sounds, holosystolic murmur Grade 3/6 in mitral focus, radiated to the armpit; protodiastolic murmur audible in the aortic and accessory aortic focus. Systolic murmur of tricuspid regurgitation and third heart sound with gallop cadence.
Abdomen: Liver palpable five centimeters below right costal margin and slightly tender. HJR+. Non-tender to palpation, +Bowel sounds 4 quadrants.
Limbs: 4+ pitting edema of lower limbs up to the knees. Nail beds minimally cyanotic, no clubbing. Pulses present, arrhythmic and filiform.
Positive Chagas antibody tests
ECG
Figure 1
Echo: significant increase in diameter of the left ventricle and left atrium, severe diffuse hypokinesis, significant degree of mitral regurgitation, mild aortic reflux, minimal pericardial effusion and pulmonary artery systolic pressure estimated at 56 mmHg
Holter Monitoring: 676 polymorphic PVCs, short non-sustained VT, 359 supraventricular premature contractions and wide permanent QRS complexes duration.
Management: Optimization of medication and repetition of Doppler echocardiogram, which revealed a thrombus in the apex of the left ventricle without change in other parameters. The anticoagulant warfarin 5mg/day was added.
Evolution: Refractory Cardiac heart failure with anasarca, jaundice, low output and death from ventricular arrhythmias within a few days.

Figure 1. Which is the ECG diagnosis?




-60°
R-peak time in aVL ≥ 45 ms
Frontal
-90°
QRS axis
aVR
aVL
180°
0°I
X
Y
+90°
III
II
qR
qR pattern
in I and aVL
qR
r III > r II it indicates
counterclockwise rotation
in the FP
P axis = near 0°
SIII>SII
rS
aVF
rS
rS

Hypothetical ventricular activation in FP



-90º

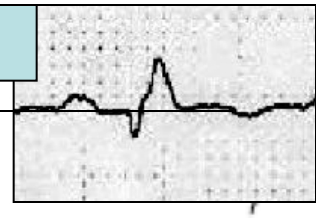
200 ms

90 ms

RV

LV

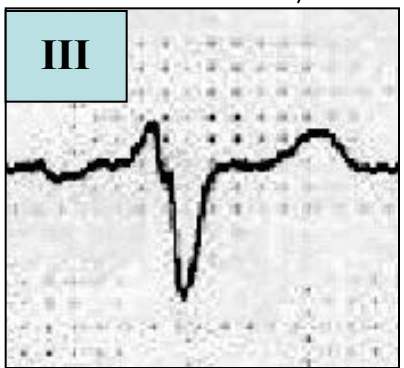
I



40 ms

+120º

III

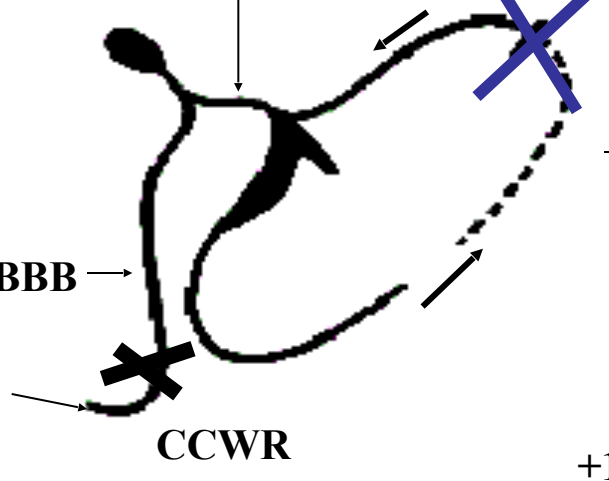


LBB

LAFB

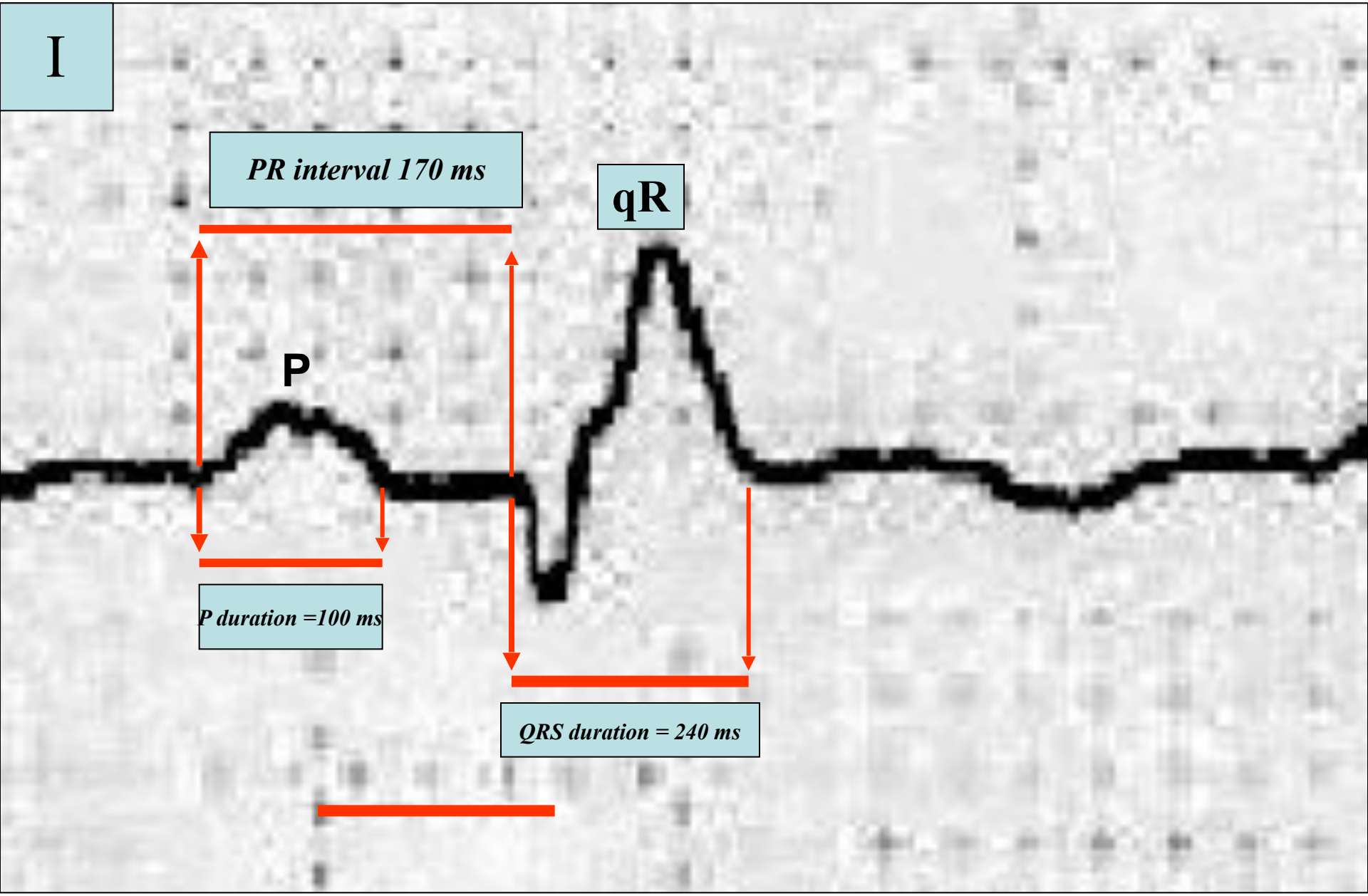
RBBB

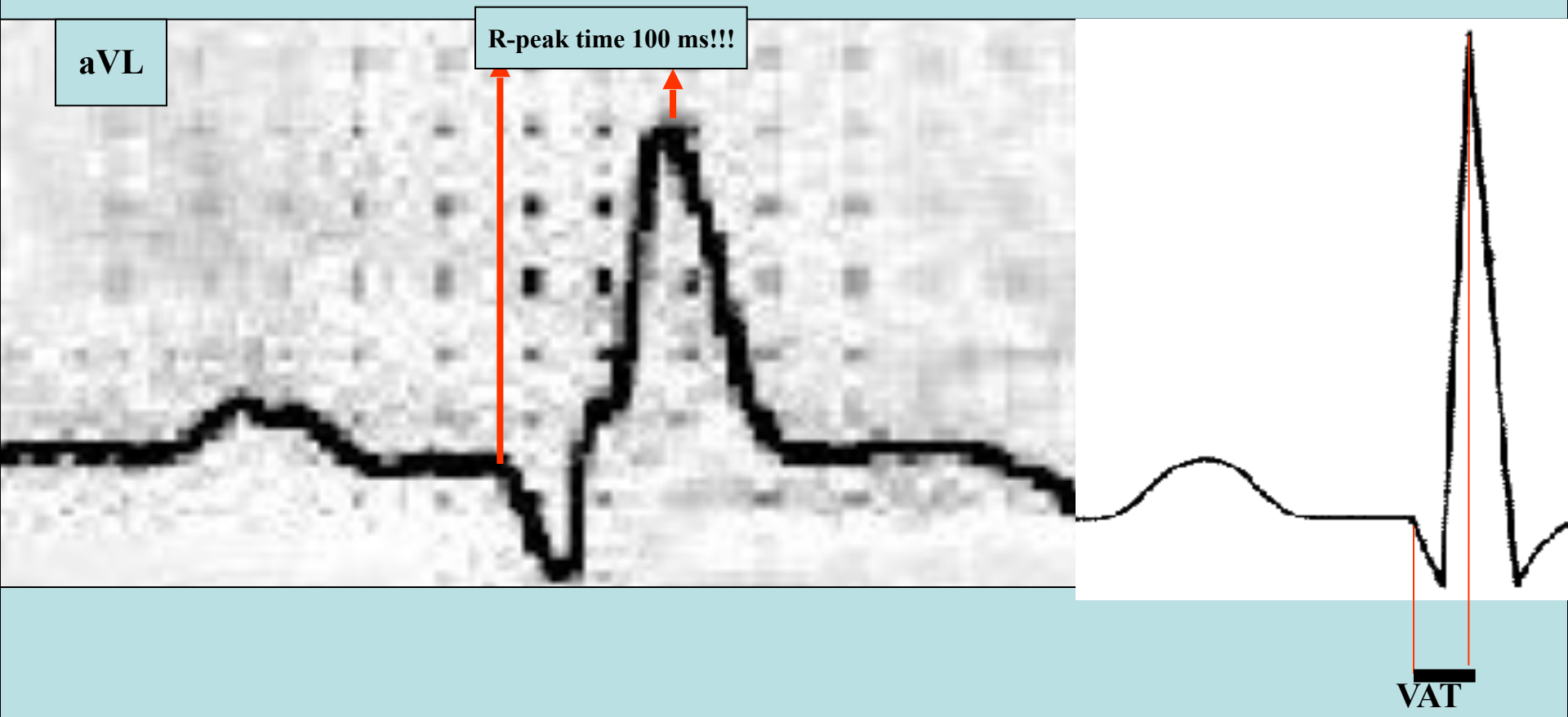
CCWR



Outline where the CCW activation in the frontal plane is shown. CCWR = counter clockwise rotation

P wave, PR interval and QRS complex duration

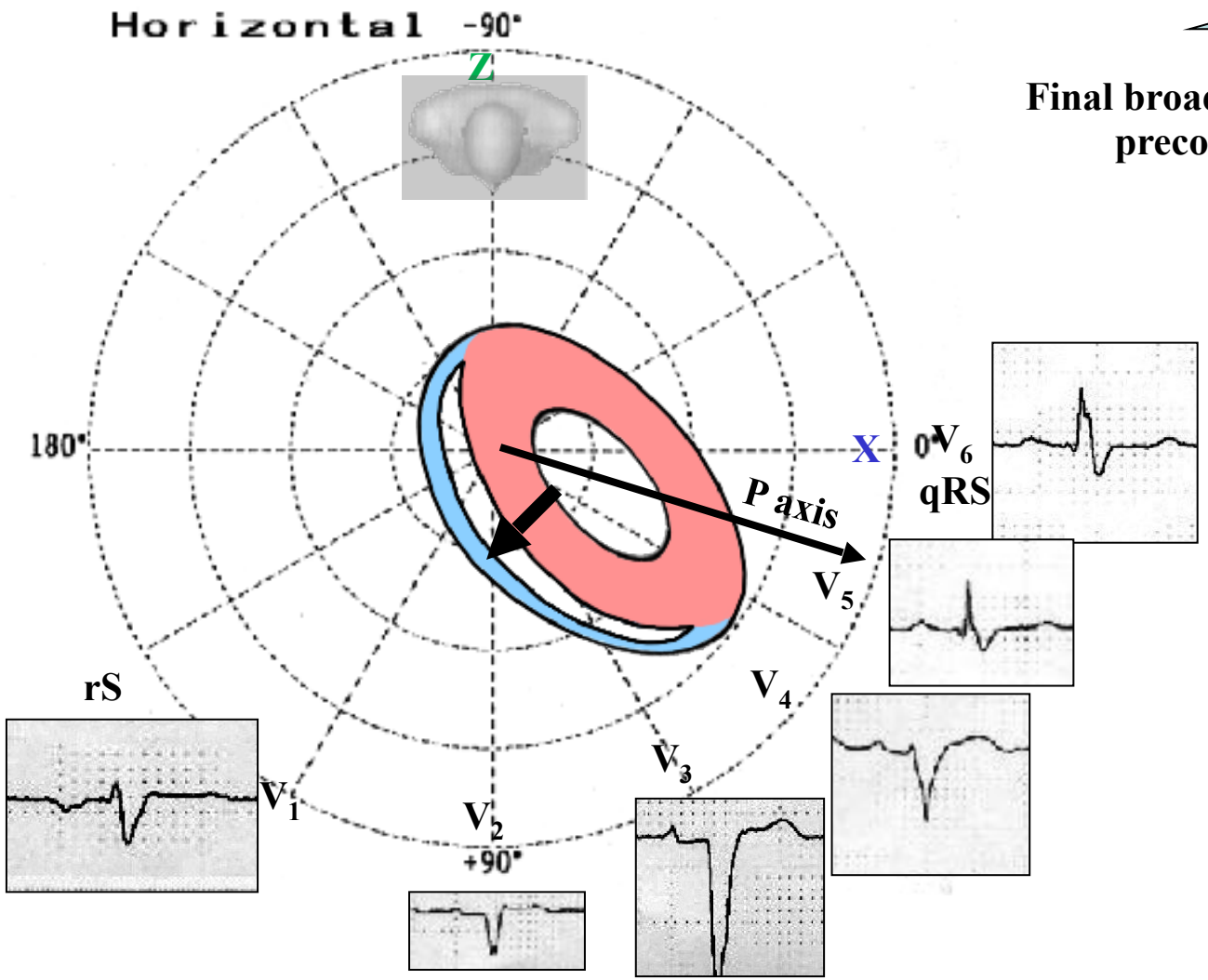




R-peak time, ventricular activation time (VAT) or Intrinsicoid deflection in aVL ≥ 45 ms

RBBB

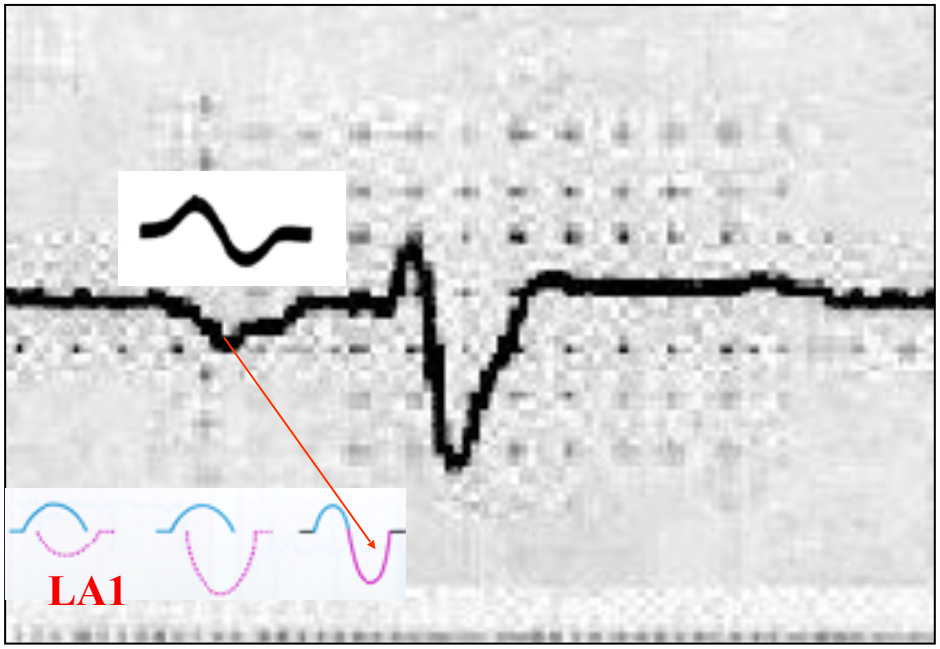
Final broad S waves in left precordial leads

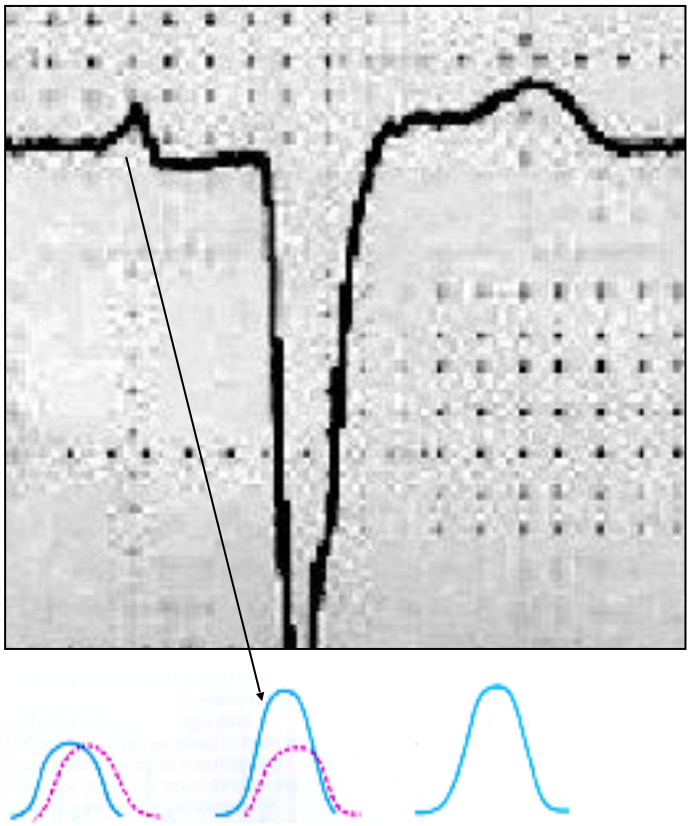


Impossible CRBBB diagnosis in the right precordial leads.

Preserved first vector. Low septal fibrosis and in free lateral wall

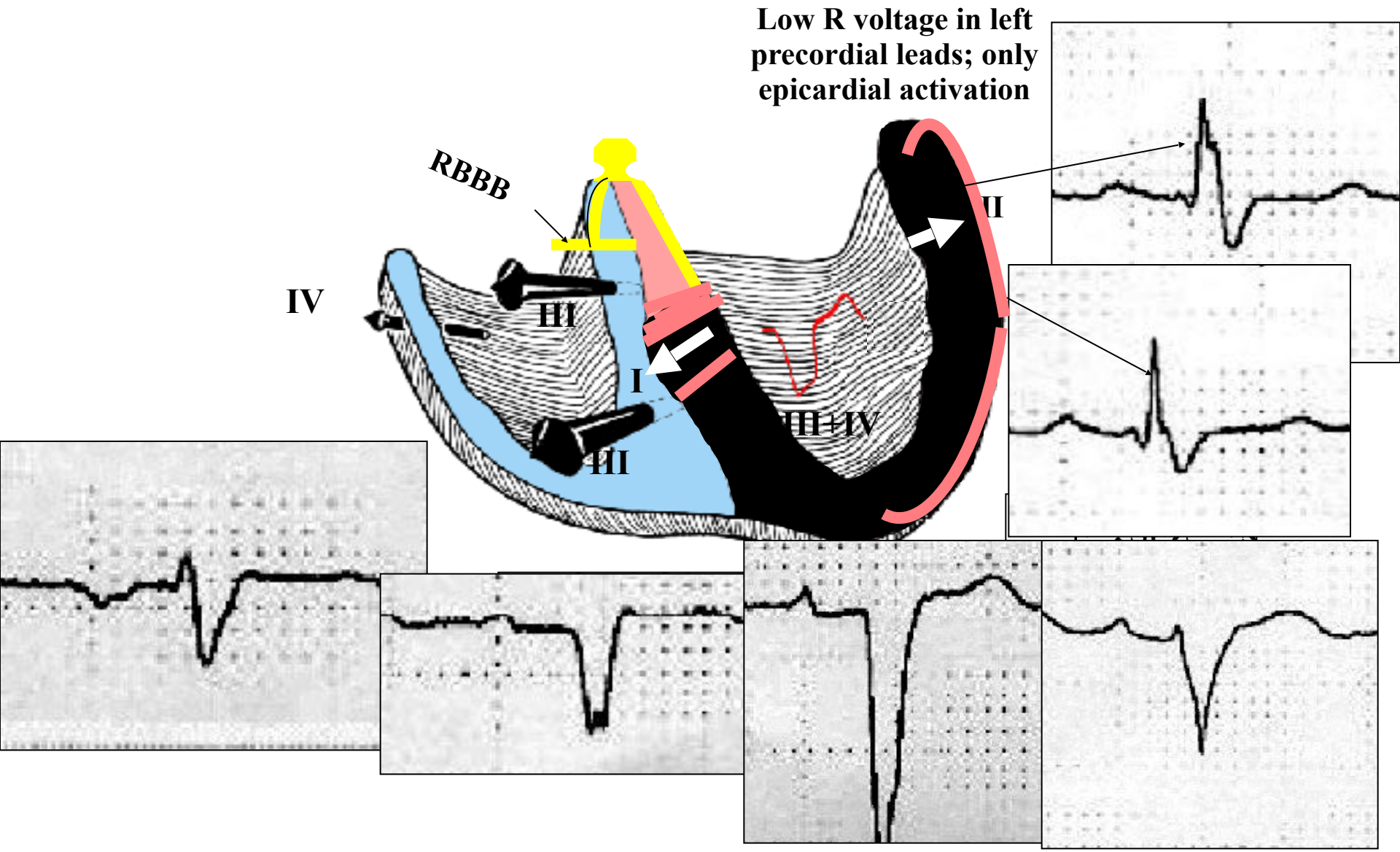
1. Association of P axis in 0º in the FP with P axis to the front (left anterior quadrant) in HP = biatrial enlargement.
2. Significant negative final P wave component in V1+ positive 1 mm; P wave in V3 is suggestive of biatrial enlargement

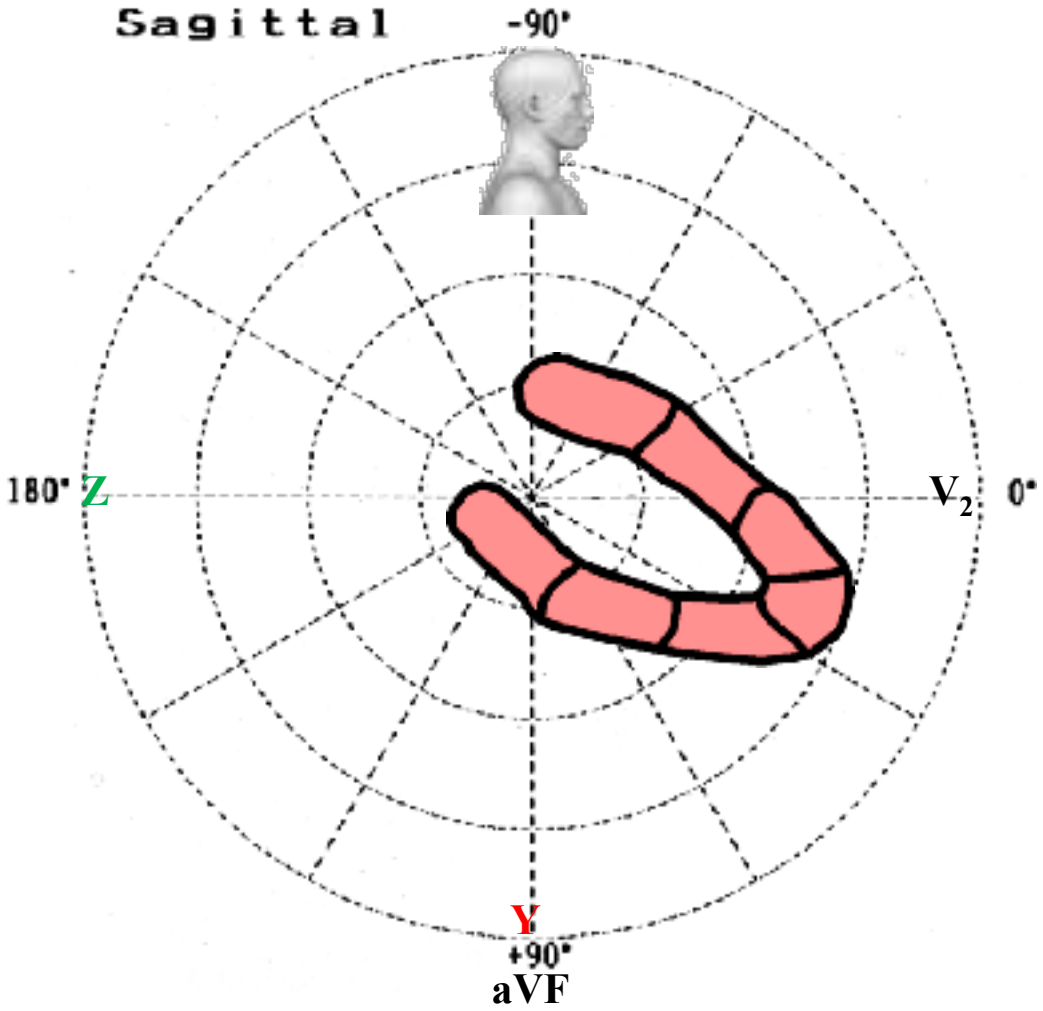


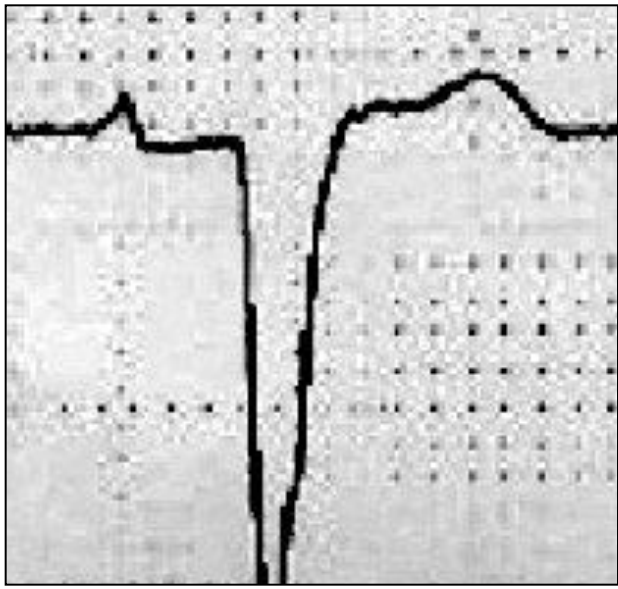


LA2/: final deep and slow component: LAE
≥ the area of one small square; the final minus portion indicates left atrial enlargement, abnormality, or advanced interatrial block

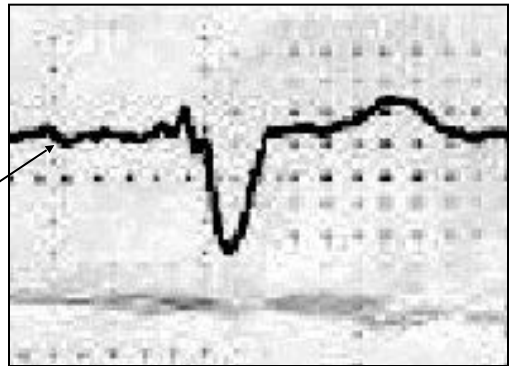
Complete RBBB complicated with low septum and free wall extensive fibrosis







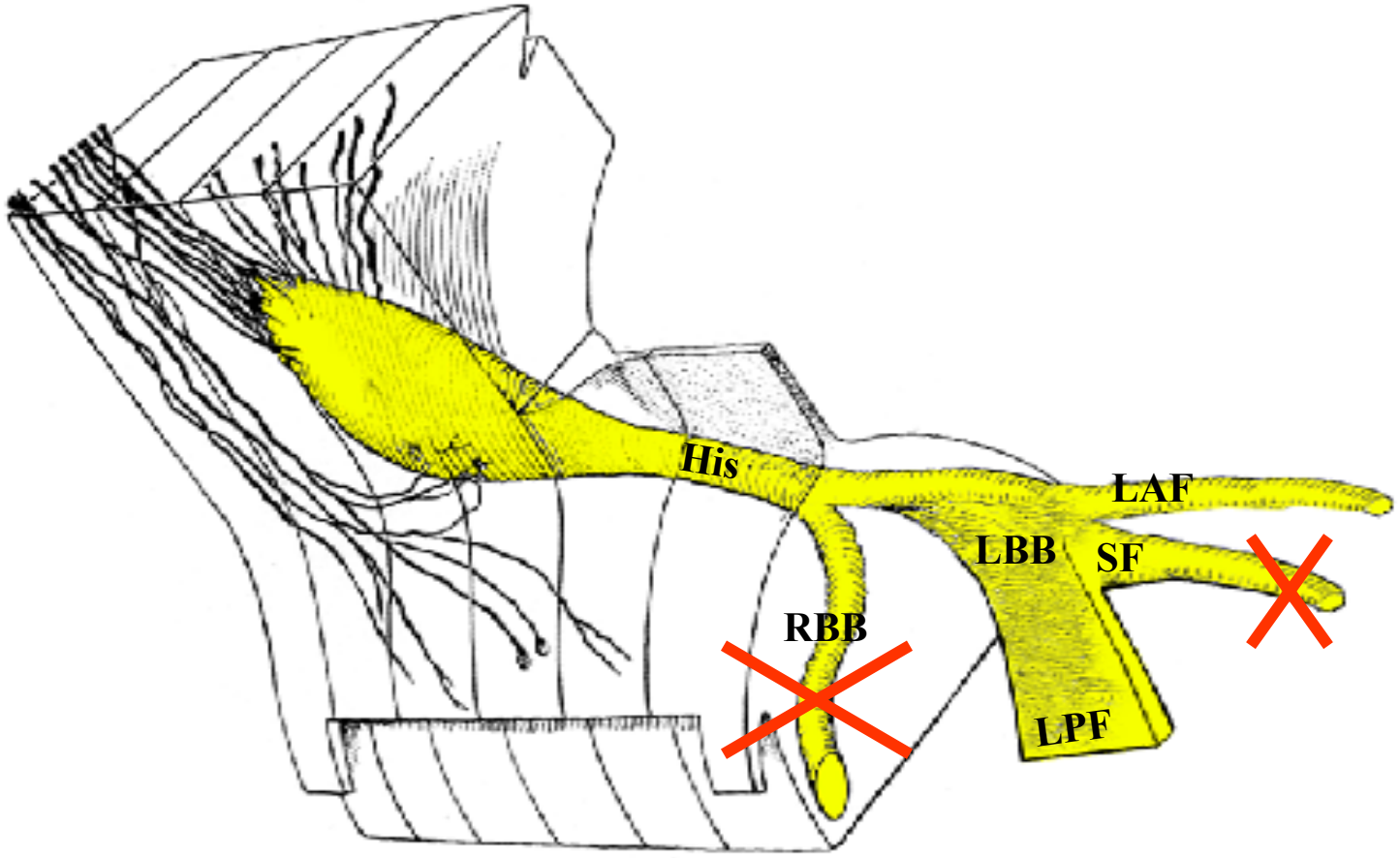
P wave axis
perpendicular to +90º



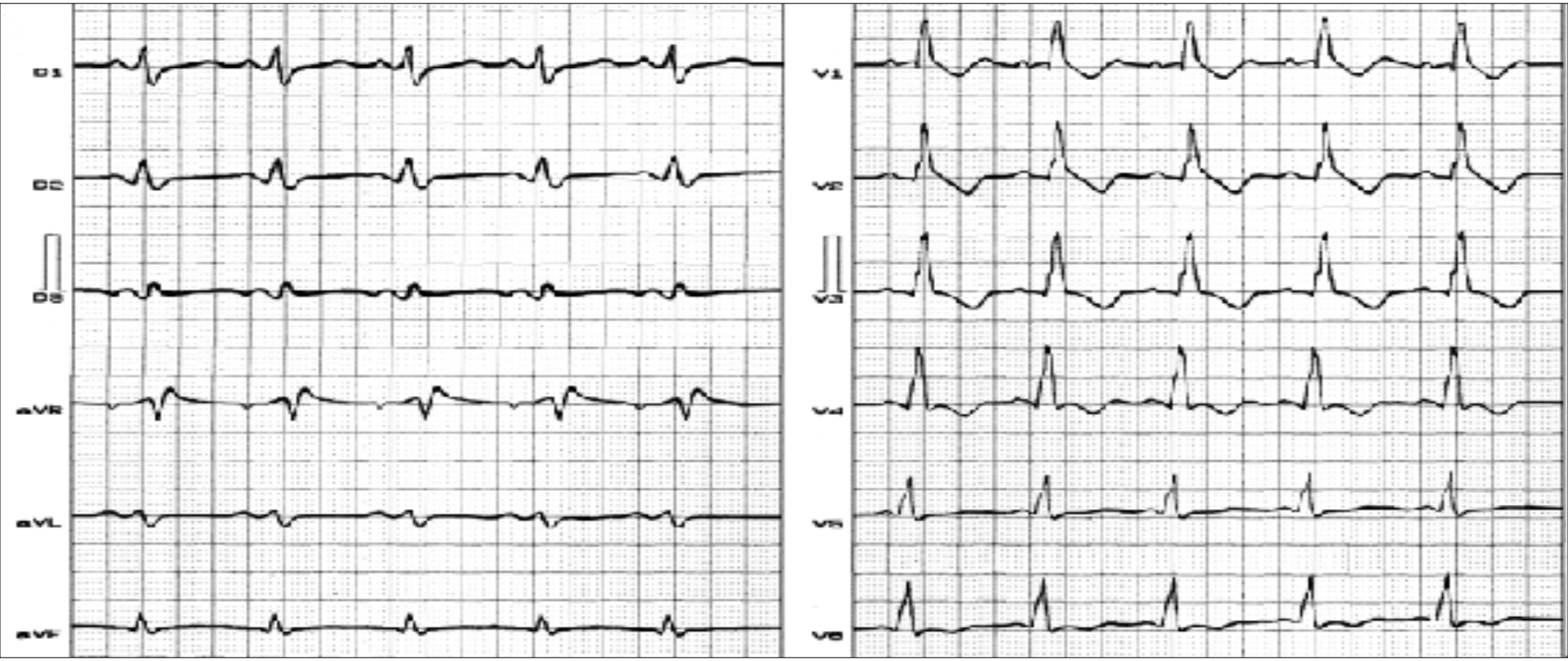
ECG analysis. Figure 1

Rhythm: Sinus rhythm
Heart rate: 82 bpm.
P wave: P axis near 0º (LAE), P duration = 100 ms, notched P wave in II and peaked P wave in V3 (RAE) = Suggestive of biatrial enlargement?
PR interval: 170 ms. Normal.
QRS axis: axis -60º Extreme left axis deviation. QRS duration (QRSd) = 240 ms very broad.
QRS voltage: Low QRS voltage complexes in the frontal plane (FP); no wave exceeds 5 mm (one large square or 5 small squares, vertically). In the FP it is considered low voltage. In the horizontal plane no wave exceeds 10 mm: QRS low voltage in both planes. Why? Consequence of severe universal fibrosis.
Left Anterior Fascicular Block (LAFB): QRS axis -60º, isodiphasic QRS complexes in aVR (QRS perpendicular to aVR), negative QRS complexes in inferior leads with rS pattern, r III > r II, SIII > SII, and qR pattern in I and aVL, prolonged R peak time.
Electrically inactive low and inferior septum with lateral wall extension (Low R voltage waves V5-V6).
Standard Masquerading Right Bundle Branch Block. This is an atypical form of RBBB with LAFB where S wave in I and aVL becomes very small or disappears, the limb leads may resemble LBBB.
In other words, LAFB obscured totally the diagnosis of CRBBB only in the FP by abolishing the broad S final wave in left standard leads I and aVL.
In the unipolar anterior precordial wall leads from V1 to V4, as a consequence of low septum anterior electrically inactive area, the Complete RBBB is almost totally hidden and is reflected by a Rs, QS or Qr pattern in these leads. The presence of Complete RBBB could be recorded with the high right-sided chest leads (**Sclarovsky 1979**). The final wide S waves in left precordial leads show the presence of Complete RBBB.

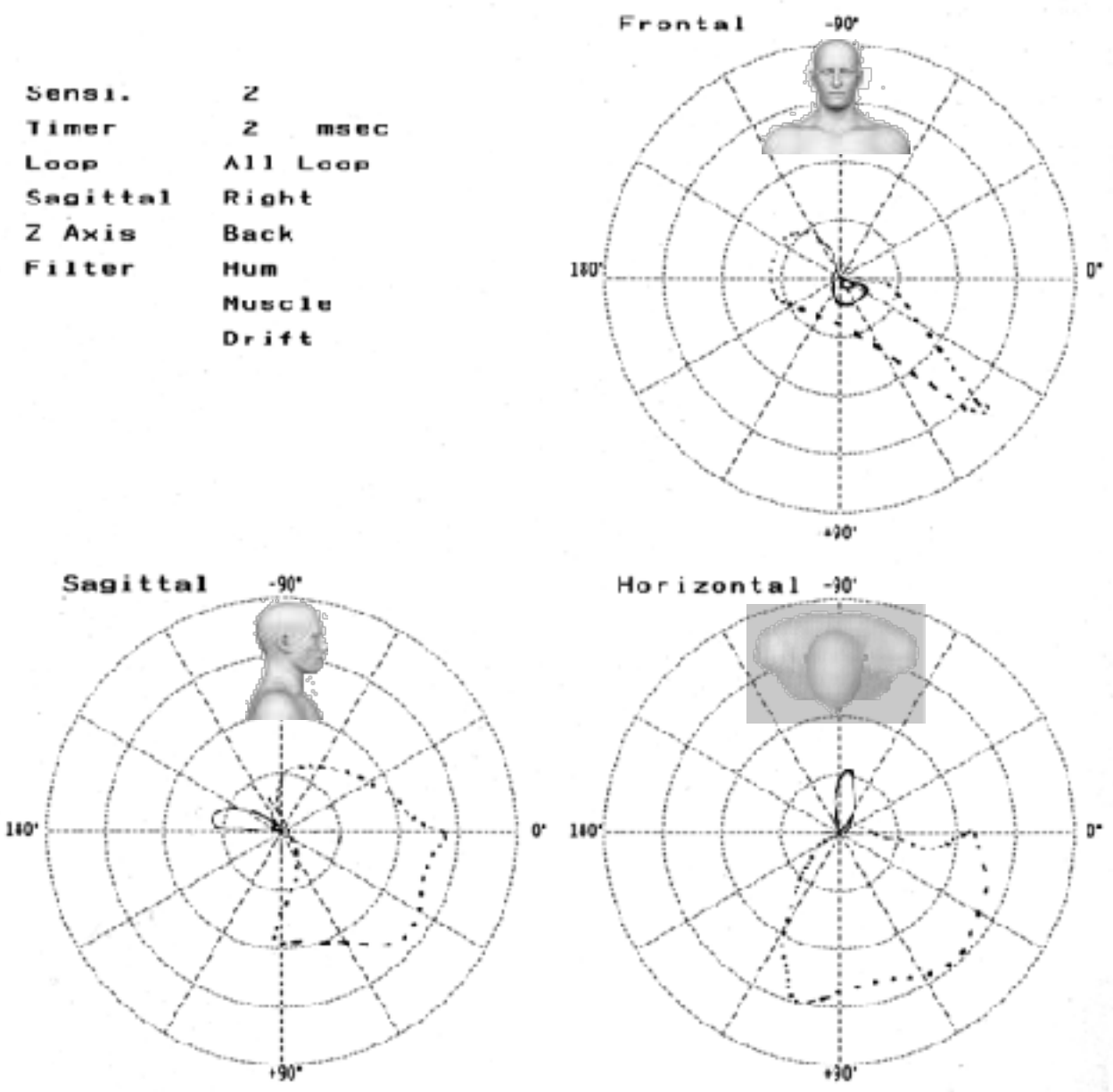
CRBBB associated to SFB



Name: AB; **Date:** 07/10/1988; **Age:** 45 y.o.; **Gender:** M.; **Race:** W; **Weight:** 70 Kg; **Height:** 1.70 m.;
Biotype: athletic; **Medication in use:** nothing stated.

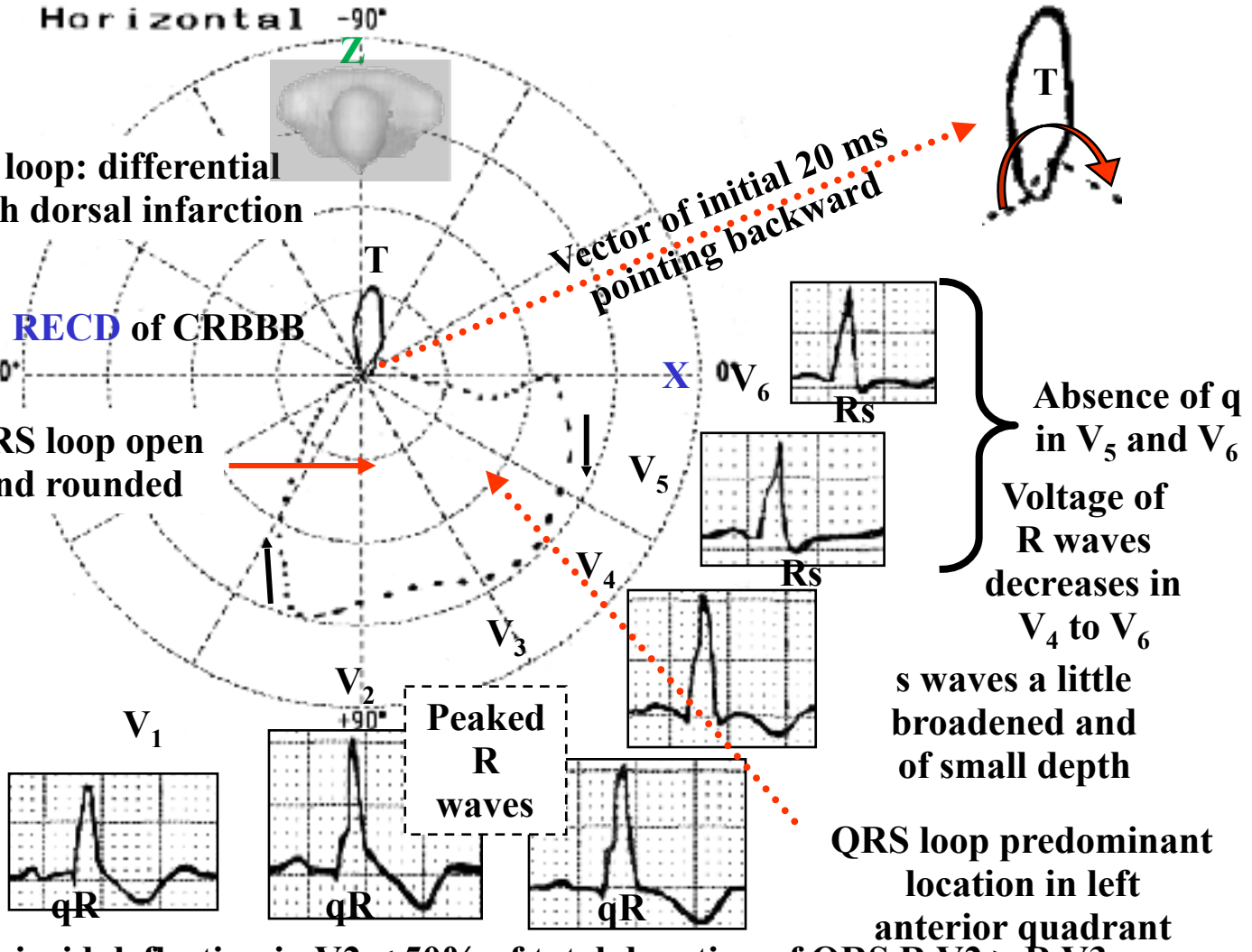


Clinical diagnosis: chronic chagasic cardiomyopathy, dromotropic form.
ECG diagnosis: CRBBB + SFB = bifascicular block. Sinus Rhythm; HR: 79 bpm; P WAVE: SÂP close to 0° and to the front; PR interval: 170 ms; SÂQRS: perpendicular to the frontal plane, duration: 220 ms, morphology: broad S from I and aVL, qR from V_1 to V_3 with peaked R waves and without the plateau proper of CRBBB. Broad descending limb of V_2 and V_3. Intrinsicoid deflection in V_2 <50% of total duration of QRS. Rs waves from V_4 to V_6, voltage of R grows from V_1 to V_2 and V_3 and decreases from V_4 to V_6, absence of q in V_5 and V_6 and s wave a little broadened and with small depth in these leads, as it would be in CRBBB in isolation.



VCG in the three planes, where in the horizontal plane we observe typical CRBBB associated to SFB. Vectors of initial 10 ms pointing backward; loop almost completely located in the left anterior quadrant. The frontal plane shows ECD in the right superior quadrant.

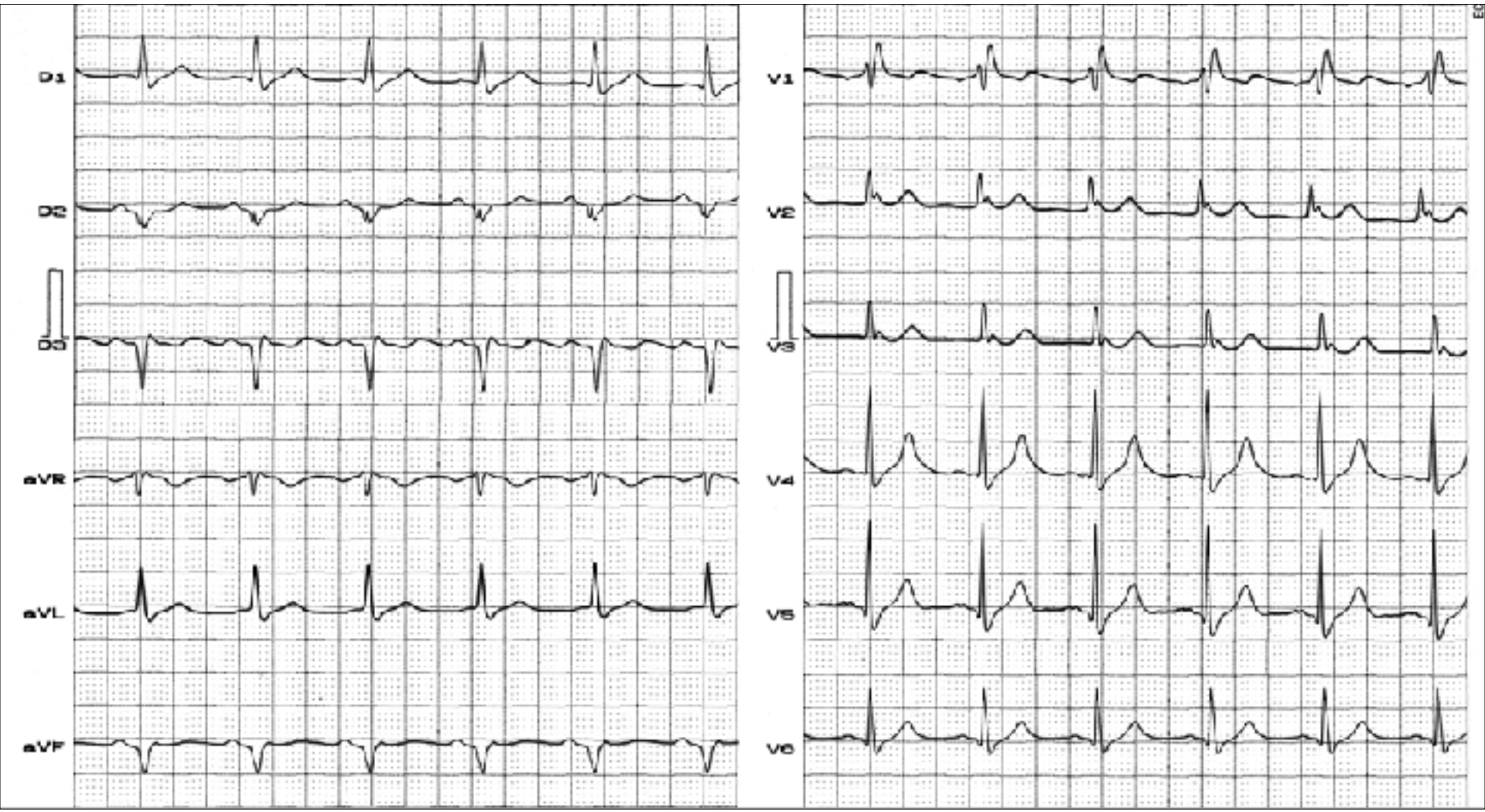
ECG/VCG correlation in the HP (SFB + CRBBB)



ECG/VCG correlation in the horizontal plane that shows association of CRBBB and SFB: vectors of initial 20 ms heading backward, QRS loop open and rounded of clockwise rotation and predominantly located in the left anterior quadrant. Leads V1, V2 and V3, qR pattern with peaked R waves. Absence of q wave in V5 and V6, voltage of R waves decreasing from V4 to V6 and S waves a little broadened.

CRBBB associated to inferior electrically inactive area and LAFB

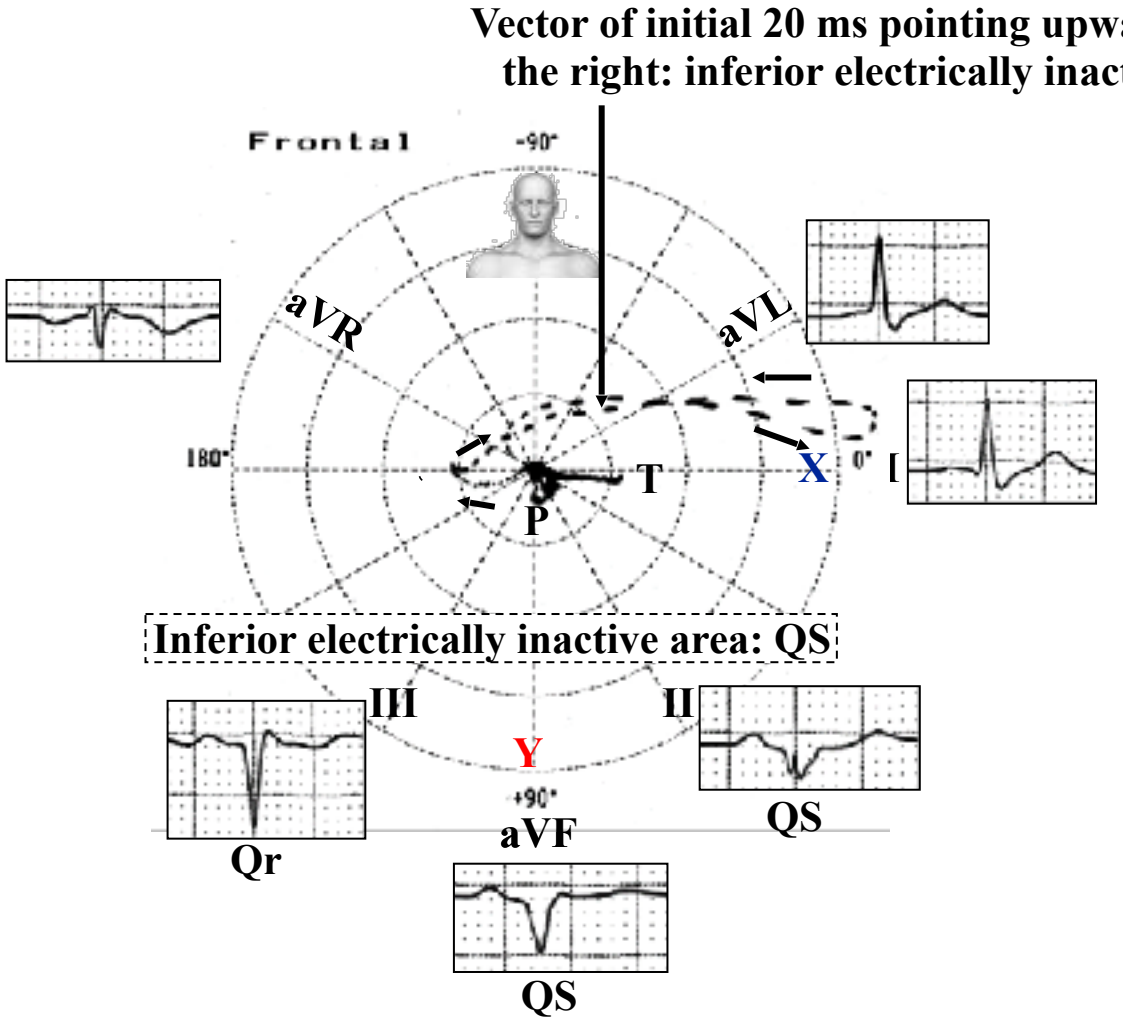
Name: DS; **Sex:** Male; **Age:** 65 yo; **Race:** White; **Weight:** 80 Kg; **Height:** 1.72m; **Date:** 19/09/1994



Clinical diagnosis: Coronary artery insufficiency; myocardial infarction two years ago.
ECG diagnosis: electrically inactive area in inferior wall: abnormal Q wave (II, III and aVF) associated to Complete RBBB. rsr' in V1 with broad final S wave in left leads.

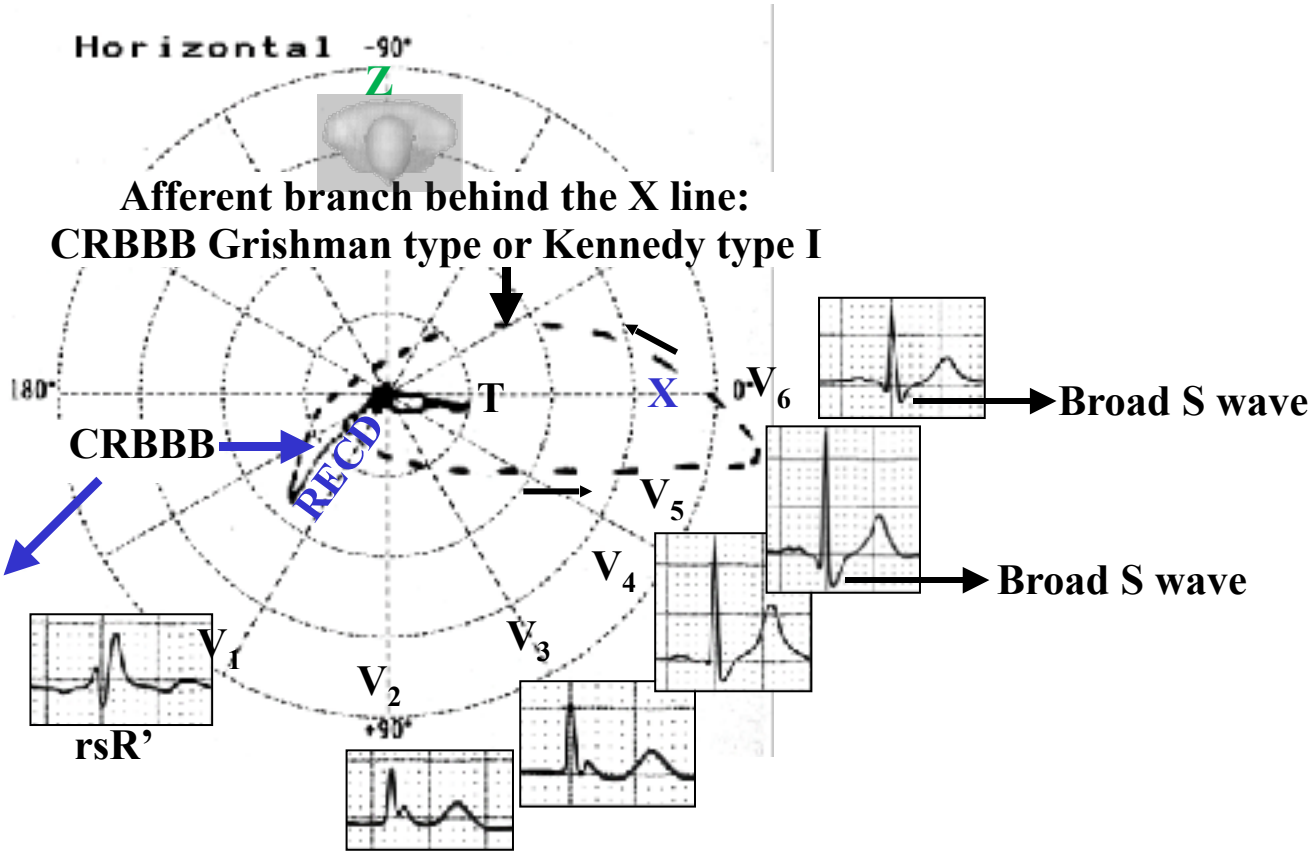
ECG of patient with coronary artery disease that shows CRBBB and inferior electrically inactive area with extreme deviation of the axis in superior quadrants, indicating the possibility of associated LAFB.

ECG/VCG correlation in the Frontal Plane



ECG/VCG correlation in the frontal plane. The QRS loop shows clockwise initial rotation and final counterclockwise rotation proper of LAFB associated to inferior electrically inactive area and ECD to the right, configuring the triple association of CRBBB + LAFB + inferior electrically inactive area.

ECG/VCG correlation in the Horizontal Plane



References:

1. Barker JM, Valencia F. The precordial electrocardiogram in incomplete right bundle branch block. Am Heart J. 1949 Sep;38(3):376-406.
2. Baydar ID, Walsh TJ, Massie E. A vectorcardiographic study of right bundle branch block with the Frank lead system. clinical correlation in ventricular hypertrophy and chronic pulmonary disese. Am J Cardiol. 1965 Feb;15:185-94.
3. Bayés de Luna A, Torner P, Oter R, Oca F, Guindo J, Rivera I et al. Study of the evolution of masked bifascicular block. PACE 1988; 11: 1.517-1.521.
4. Bayés de Luna A. Clinical Electrocardiography: A Text Books. II Edition. New York: Futura, 1999.
5. Bayés de Luna A, Brugada J, Baranchuk A, Borggrefe M, Breithardt G, Goldwasser D, Lambiase P, Pérez-Riera AR, Garcia Niebla J, Pastore CA, Oreto Guiuseppe, McKenna William. Zareba W, Brugada R, Brugada P. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. Journal of Electrocardiol. 2012; 45:433-
6. Brohet CR, Styns M, Arnaud P, et al. Vectorcardiographic diagnosis of right ventricular hypertrophy in the presence of right bundle branch block in young subjects. Am J Cardiol. 1978 Oct;42(4):602-12.
7. Bussink BE, Holst AG, Jespersen L, et al. Right bundle branch block: prevalence, risk factors, and outcome in the general population: results from the Copenhagen City Heart Study. Eur Heart J. 2013 Jan; 34(2):138-46.
8. Bussink BE, Holst AG, Jespersen L, et al. Right bundle branch block: prevalence, risk factors, and outcome in the general population: results from the Copenhagen City Heart Study. Eur Heart J. 2013 Jan; 34(2):138-46.
9. Caird FI, Wilcken DE. The electrocardiogram in chronic bronchitis with generalized obstructive lung disease. Its relation to ventilatory function. Am J Cardiol. 1962 Jul;10:5-13.
10. Caird FI, Wilcken DE, Williams RS. The elecrocardiogram in diffuse interstitial disease of the lung and its relation to pulmonary function. Am J Cardiol. 1962 Jul;10:14-9.

11. Chevallier S, Forclaz A, Tenkorang J, et al. New electrocardiographic criteria for discriminating between Brugada types 2 and 3 patterns and incomplete right bundle branch block. J Am Coll Cardiol.2011 Nov 22;58: 2290-2298.
12. de Micheli A, Medrano GA, Martínez Rios MA. [Right blocks in interauricular communication]. Arch Inst Cardiol Mex. 1978 Nov-Dec;48(6):1091-113.
13. Draeger HT, Assassi S, Sharif R,et al. Right bundle branch block: a predictor of mortality in early systemic sclerosis. PLoS One. 2013 Oct 31;8(10):e78808.
14. Dubner S, Schapachnik E, Riera AR, Valero E. Chagas disease: state-of-the-art of diagnosis and management. Cardiol J. 2008;15:493-504.
15. Elizari MV. [Chagasic myocardiopathy: historical perspective]. Medicina (B Aires). 1999;59 Suppl 2:25-40.
16. Fedor JM, Walston A 2nd, Wagner GS, Starr J. The vectorcardiogram in right bundle branch block: correlation with cardiac failure and pulmonary disease. Circulation. 1976 Jun;53(6):926-30.
17. Gómez Barrado JJ, Turégano Albarrán S, et al. Clinical and electrocardiographic characteristics of masquerading bifascicular block.Rev Esp Cardiol. 1997 Feb;50:92-97. Massing GK, James TN. Conduction and block in the right bundle branch, real and imagined. Circulation. 1972 Jan;45(1):1-3.
18. Gelband H, Waldo AL, Kaiser GA, et al. Etiology of right bundle-branch block in patients undergoing total correction of tetralogy of Fallot. Circulation. 1971 Dec;44(6):1022-33.
19. Hébert JL, Duthoit G, Hidden-Lucet F, et al. Images in cardiovascular medicine. Fortuitous discovery of partial Uhl anomaly in a male adult. Circulation. 2010 Jun 8;121(22):e426-9.
20. Hiss RG, Lamb LE. Electrocardiographic findings in 122,043 individuals. Circulation. 1962 Jun; 25:947-61.
21. Holtzman D, Aronow WS, Mellana WM, et al. Electrocardiographic abnormalities in patients with severe versus mild or moderate chronic obstructive pulmonary disease followed in an academic outpatient pulmonary clinic. Ann Noninvasive Electrocardiol. 2011 Jan;16(1):30-2.

22. Izumi K. Optimal control of intermittent normal conduction in a tachycardia-dependent right bundle branch block. Mater Med Pol. 1996 Oct-Dec;28(4):141-8.
23. Janse MJ, van Dam RT. Effect of sudden changes in heart rate on refractory periods of ventricular myocardium and specialized conducting system in dog's heart. Br Heart J. 1971 Jan;33(1):148.
24. Kinoshita S, Katoh T, Tsujimura Y, et al. Apparent bradycardia-dependent right bundle branch block associated with atypical atrioventricular Wenckebach periodicity as a possible mechanism. J Electrocardiol. 2003 Oct;36(4):355-61.
25. Kleemann T, Juenger C, Gitt AK, et al.Incidence and clinical impact of right bundle branch block in patients with acute myocardial infarction: ST elevation myocardial infarction versus non-ST elevation myocardial infarction. Am Heart J. 2008 Aug;156(2):256-61.
26. Koos R, Mahnken AH, Aktug O, et al. Electrocardiographic and imaging predictors for permanent pacemaker requirement after transcatheter aortic valve implantation. J Heart Valve Dis. 2011 Jan;20(1): 83-90.
27. Kowey PR, Koslow M, Marinchak RA Masquerading Bundel-branch block – Electrophysiological correlation J electrophysiol. 1989; 3:156-159.
28. Krongrad E, Hefler SE, Bowman FO Jr, et al. Further observations on the etiology of the right bundle branch block pattern following right ventriculotomy. Circulation. 1974 Dec;50(6):1105-13.
29. Kusumoto S, Kawano H, Makita N, et al. Right bundle branch block without overt heart disease predicts higher risk of pacemaker implantation: the study of atomic-bomb survivors. Int J Cardiol. 2014 Jun 1;174(1):77-82.
30. Kumpuris AG, Casale TB, Mokotoff DM, et al. Right bundle-branch block. Occurrence following nonpenetrating chest trauma without evidence of cardiac contusion. JAMA. 1979 Jul 13;242(2):172-3.
31. Lancaster MC, Schechter E, Massing GK. Acquired complete right bundle branch block without overt cardiac disease. Clinical and hemodynamic study of 37 patients. Am J Cardiol. 1972 Jul 11;30(1):32-6.
32. Lerecouvreux M, Perrier E, Leduc PA,et al. Right bundle branch block: electrocardiographic and prognostic features]. Arch Mal Coeur Vaiss. 2005 Dec;98(12):1232-8.

33. Lev M. Anatomic basis for atrioventricular block. Am J Med. 1964 Nov;37:742-8.
34. Lev M, Bharati S. Atrioventricular and intraventricular conduction disease. Arch Intern Med. 1975 Mar; 135(3):405-10.
35. Lewinter C, Torp-Pedersen C, Cleland JG, Køber L. Right and left bundle branch block as predictors of long-term mortality following myocardial infarction. Eur J Heart Fail. 2011 Dec;13(12):1349-54.
36. Luna Filho B, Bocanegra JA, Pfeferman A, Andrade JL, Martinez Filho EE. [Fascicular block of the His bundle: critical approach for its identification]. Arq Bras Cardiol. 1989 Nov;53(5):261-5.
37. Marin-Neto JA, Simões MV, Maciel BC. Specific diseases: cardiomyopathies and pericardial diseases. Other cardiomyopathies. In: Yusuf S, Cairns J, Camm J, Fallen E, Gersh BJ, eds. - Evidence Based Cardiology. London, GB: BMJ Books, Brit Med Association, 1998: 744-61.
38. Massing GK, James TN. Conduction and block in the right bundle branch, real and imagined. Circulation. 1972 Jan;45(1):1-3.
39. Mavrogeni S, Sfikakis PP, Karabela G, et alCardiovascular magnetic resonance imaging in asymptomatic patients with connective tissue disease and recent onset left bundle branch block. Int J Cardiol.2014 Jan 15;171(1):82-7.
40. Melgarejo-Moreno A, Galcerá-Tomás J, Garciá-Alberola A, et al. Incidence, clinical characteristics, and prognostic significance of right bundle-branch block in acute myocardial infarction: a study in the thrombolytic era. Circulation. 1997 Aug 19;96(4):1139-44.
41. Miquel C, Sodi-Pallares D, Cisneros F, et al. Right bundle branch block and right ventricular hypertrophy; electrocardiographic and vectorcardiographic diagnosis. Am J Cardiol. 1958 Jan;1(1):57-67.
42. Mueller C, Laule-Kilian K, Klima T, et al. Right bundle branch block and long-term mortality in patients with acute congestive heart failure. J Intern Med. 2006 Nov;260(5):421-8. Rodriguez MI, Sodi-Pallares D. The mechanism of complete and incomplete bundle branch block. Am Heart J. 1952 Nov;44(5):715-46.
43. Nielsen TT, Lund O, Rønne K, et al. Changing electrocardiographic findings in pulmonary embolism in relation to vascular obstruction. Cardiology. 1989;76(4):274-84.

44. Okajima S, Okumura M, Sotabata I. Comparison of Frank-vectorcardiograms of normal conduction and right bundle branch block in patients with intermittent or transient right bundle branch block. Jpn Heart J. 1980 Mar;21(2):257-71.
45. Ortega-Carnicer J, Malillos M, Muñoz L, Rodriguez-Garcia J. Left anterior hemiblock masking the diagnosis of right bundle branch block. J Electrocardiol. 1986 Jan; 19: 97-98. Penaloza D, Gamboa R, Sime F. Experimental right bundle branch block in the normal human heart. Electrocardiographic, vectorcardiographic and hemodynamic observations. Am J Cardiol. 1961 Dec;8:767-79.
46. Pastore CA, Moffa PJ, Spiritus MO, et al. [Fascicular blocks of the right branch. Standardization of vectorelectrocardiographic findings]. Arq Bras Cardiol. 1983 Sep;41(3):161-6.
47. Richman JL, Wolff L. Left bundle branch block masquerading as right bundle branch block. Am Heart J. 1954 Mar; 47: 383-393.
48. Riera AR, de Cano SJ, Cano MN, et al. Vector electrocardiographic alterations after percutaneous septal ablation in obstructive hypertrophic cardiomyopathy. Possible anatomic causes. Arq Bras Cardiol. 2002 Nov;79(5):466-75.
49. Rodriguez MI, Sodi-Pallares D. The mechanism of complete and incomplete bundle branch block. Am Heart J. 1952 Nov;44(5):715-46.
50. Rosembaum MB,, Elizari MV, Lazzari JO. Los hemibloqueos. Buenos Aires; Paidos 1968.
51. Rosenbaum MB, Yesuron J, Lazzari JO, Elizari MV. Left anterior hemiblock obscuring the diagnosis of right bundle branch block.Circulation. 1973 Aug; 48: 298-303.
52. Rotman M, Triebwasser JH. A clinical and follow-up study of right and left bundle branch block. Circulation. 1975 Mar;51(3):477-84.
53. Rusconi L, Nava A, Sermasi S, Antonioli GE. The left posterior fascicular block: is the diagnosis possible only by ECG? G Ital Cardiol. 1980;10:1129-1134.
54. Schamroth L, Dekock J. The concept of 'masquerading' bundle-branch block. S Afr Med J. 1975 Mar 15; 49: 399-400.

55. Schamroth L, Myburgh DP, Schamroth CL. The early signs of right bundle branch block. Chest. 1985; 87:180-5. Ocal A, Yildirim N, Ozbakir C, et al. Right bundle branch block: a new parameter revealing the progression rate of mitral stenosis. Cardiology. 2006;105(4):219-22.
56. Schmunis GA, Zicker F, Moncayo A. Interruption of Chagas' disease transmission through vector elimination. Lancet 1996; 348: 117.
57. Schneider JF, Thomas HE, Kreger BE,et al. Newly acquired right bundle-branch block: The Framingham Study. Ann Intern Med. 1980 Jan;92(1):37-44.
58. Sclarovsky S, Lewin RF, Strasberg B, Agmon J. Left anterior hemiblock obscuring the diagnosis of right bundle branch block in acute myocardial infarction. Circulation. 1979 Jul; 60: 26-32.
59. Sodi D, Bisteni A, Medrano G. Electrocardiografia y vetorcardiografia deductivas. Vol. 1 Mexico, DF: La Prensa Médica Mexicana, 1964.
60. Surawicz B, Childers R, Deal BJ, et al. American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009 Mar 17;53(11): 976-81.
61. Tabatabaei N, Katanyuwong P, Breen JF, et al. Images in cardiovascular medicine. Uncommon variant of Ebstein anomaly with tricuspid stenosis. Circulation. 2009 Jul 7;120(1):e1-2.
62. Tobias NM, Pastore CA, Moffa PJ, et al. [Divisional blocks of the right branch in Chagas' cardiomyopathy]. Arq Bras Cardiol. 1986 Dec;47(6):387-91.
63. Tomita M, Kitazawa H, Sato M, et al. A complete right bundle-branch block masking Brugada syndrome. J Electrocardiol. 2012 Nov-Dec;45(6):780-2.
64. Unger PN, Lesser ME, Kugel VH, Lev M, The Concept of "Masquerading" Bundle-Branch Block An Electrocardiographic-Pathologic Correlation Circulation. 1958;17:397-409.

65. Varriale P, Kennedy RJ. Right bundle branch block and left posterior fascicular block. Vectorcardiographic and clinical features. Am J Cardiol. 1972 Apr;29(4):459-65.
66. Wanderley DMV, Corrêa FMA. Epidemiology of Chagas' heart disease. S,,o Paulo Med J 1995; 113: 742-9.
67. Willems JL, Robles de Medina EO, Bernard R, et al. Criteria for intraventricular conduction disturbances and pre-excitation. World Health Organizational/International Society and Federation for Cardiology Task Force Ad Hoc. J Am Coll Cardiol. 1985 Jun;5(6):1261-75.
68. Zhong-Qun Z, Bo Y, Nikus KC, et al. Correlation between ST-segment elevation and negative T waves in the precordial leads in acute pulmonary embolism: insights into serial electrocardiogram changes. Ann Noninvasive Electrocardiol. 2014 Jul;19(4):398-405.