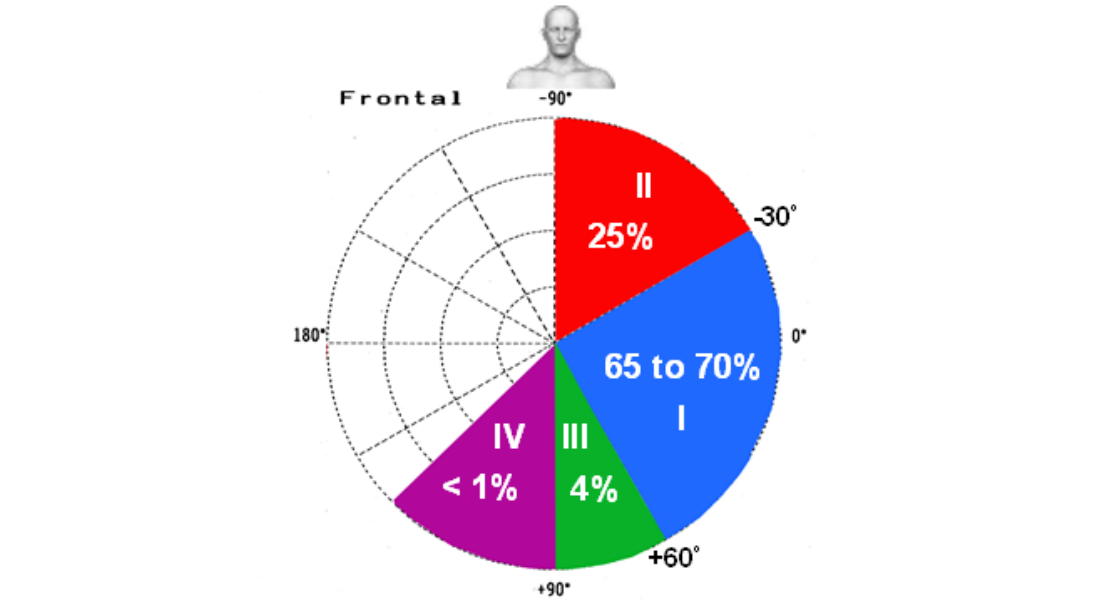
Types of CLBBB according to electrical axis of QRS complex in the FP



With QRS axis not deviated: between -30° and +60° (≈ 65% to 70% of cases)
With QRS axis with extreme deviation to the left: beyond -30^0 (≈25% of cases)
With QRS axis deviated to the right: between +60° and +90° (≈ 3.5 a 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 E, 1951**). Causes that determine paradoxical complete LBBB:

Figure 1. Types of CLBBB according to electrical axis of QRS complex in the FP.

ECG/VCG correlation of CLBBB in the HP

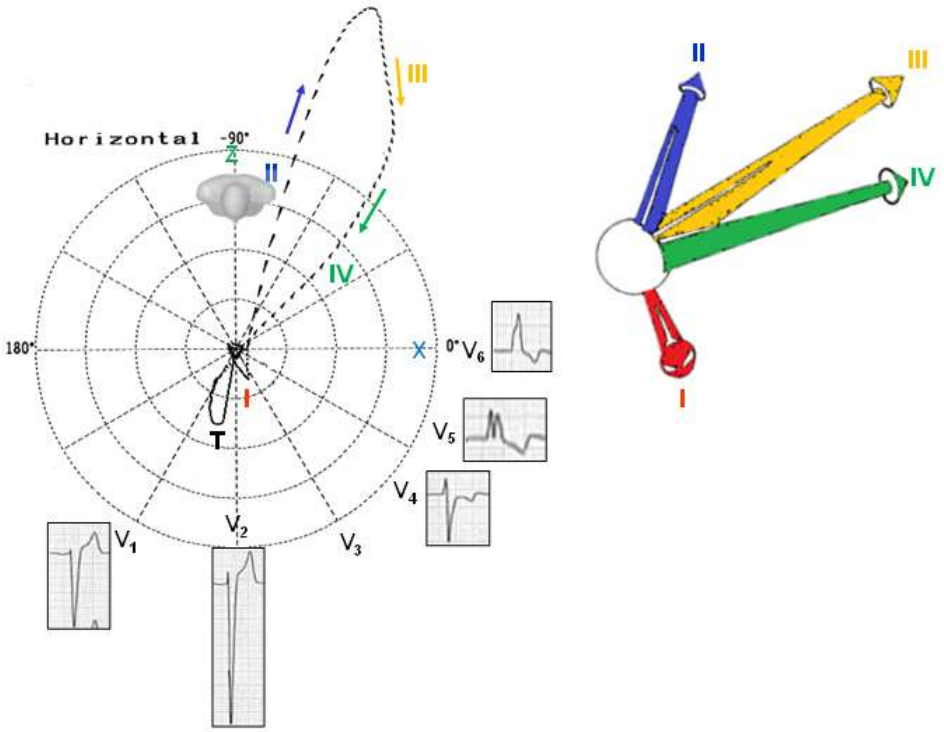


Figure 2. ECG/VCG correlation of CLBBB in the HP.**Vectorcardiographic criteria of CLBBB not complicated in the HP**

- *Narrow, long QRS loop, and with morphology usually in 8.*
- *The QRS loop duration is ≥ 120ms*
- *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 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 2mV.*
- *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 CLBBB not complicated in the Frontal Plane

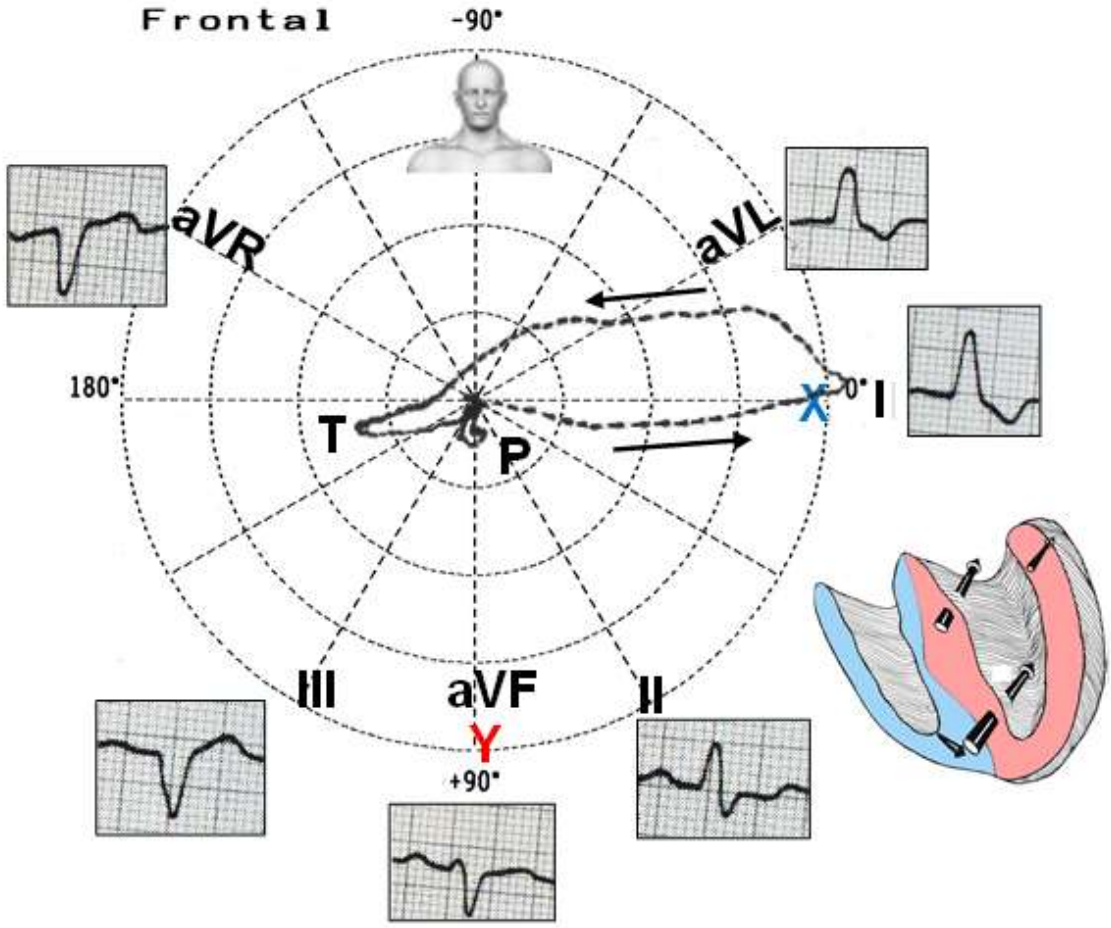


Figure 3. QRS and T loop in the LBBB.

- 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 CLBBB not complicated in the Right Sagittal Plane

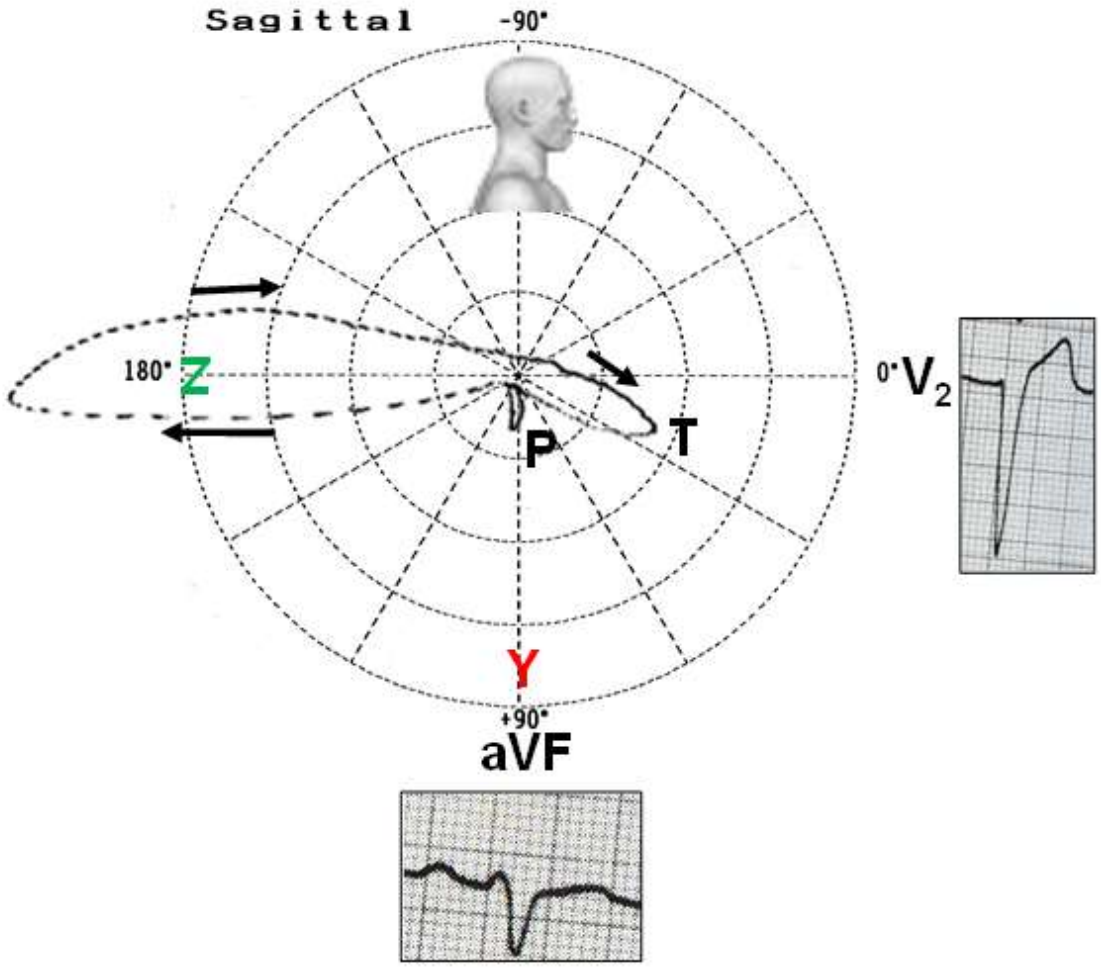


Figure 4. QRS and T loop in the LBBB.

- 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.

Summary of QRS VCG loop on HP in uncomplicated and complicated LBBB (Sodi-Pallares, Ponce de Leon et al. 1970) (Goldman and Pipberger 1969) (Cabrera, Costa Rocha et al. 1959)

A) Uncomplicated Left Bundle Branch Block

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

B) Complicated Left Bundle Branch Block

(B-1) Anteroseptal myocardial infartion (Medrano, Brenes et al. 1970)

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

(B-2) Localized anterior myocardial infarction

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

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

- The main body of QRS loop has a countreclockwise inscription with the afferent limb to the right of the efferent limb on HP.

(B-4) Massive septal myocardial infarction

- The HP show a large initial deflection in a CCW direction with the reminder of the QRS loop displayed 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, Perrot et al. 1985).

Value VCG for the diagnosis of acute myocardial infarction in presence of LBBB (Eriksson, Andersen et al. 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.

LBBB ECG / VCG exemples

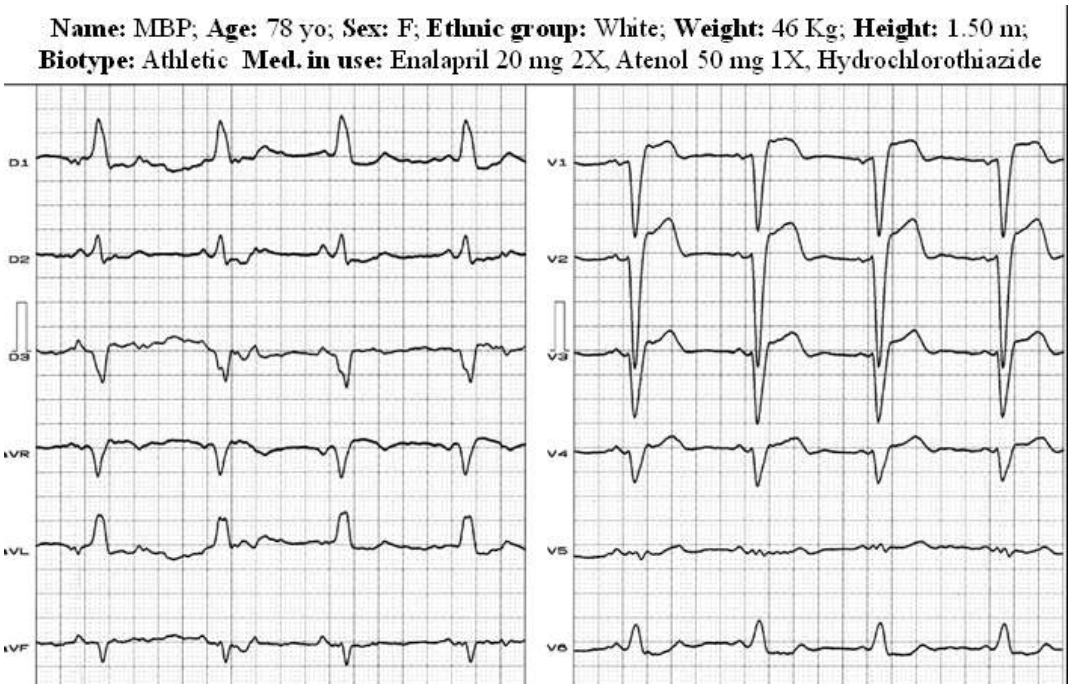


Figure 5. Clinical diagnosis: Hypertensive heart disease. ECG diagnosis: Sinus rhythm; HR 59 bpm; PR interval: 120 ms; SÂQRS: -10°; QRS duration: 165 ms; R wave in tower in I, aVL and V6, CLBBB.

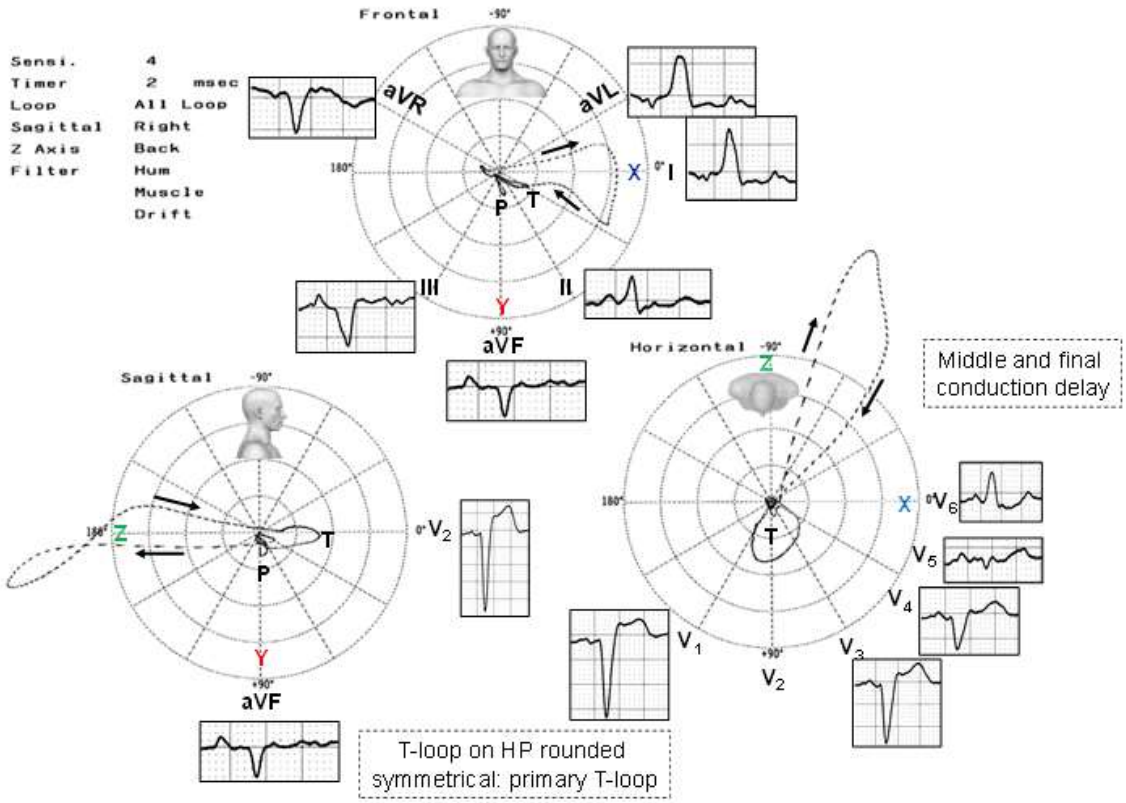


Figure 6. ECG/VCG correlation.

Name: MMNO; **Age: 72yo.; 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

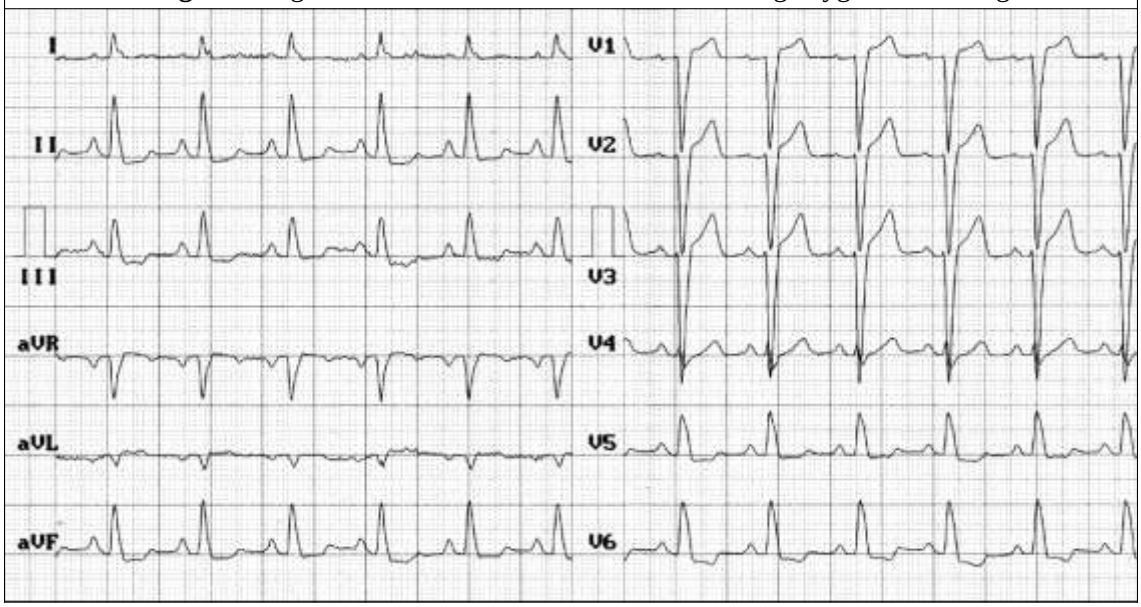


Figure 7. Clinical diagnosis: Hypertensive heart disease + tricuspid insufficiency. ECG diagnosis: SR; HR: 87 bpm; P wave: SÂP: + 70°; P wave voltage: 2.7 mm: RAE; duration of 110 ms; PR interval: 180 ms; SÂQRS: + 70 °; QRSD: 130 ms; QRS of V1 and V2 totally or predominantly negative: rS; monophasic R wave of slow registration in left leads V5 and V6. ST-T opposite to greater deflection of QRS: CLBBB.

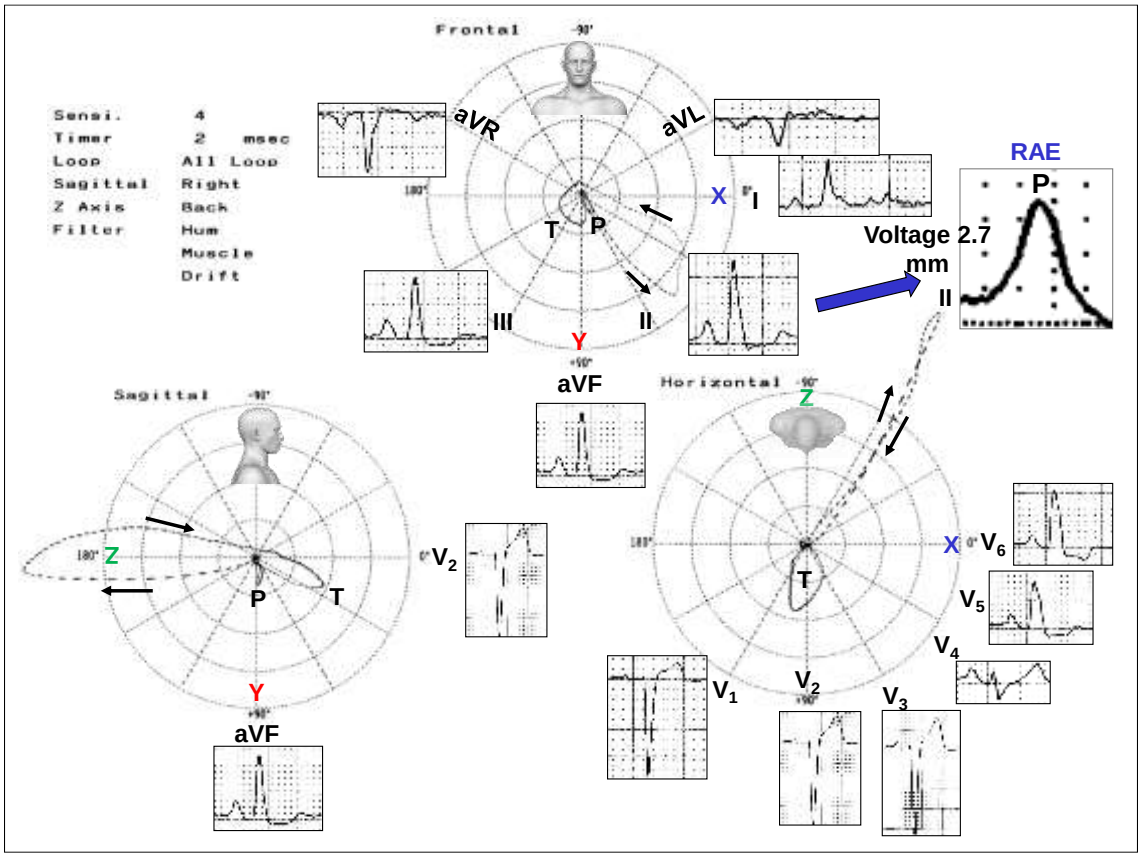


Figure 8. ECG/VCG correlation.

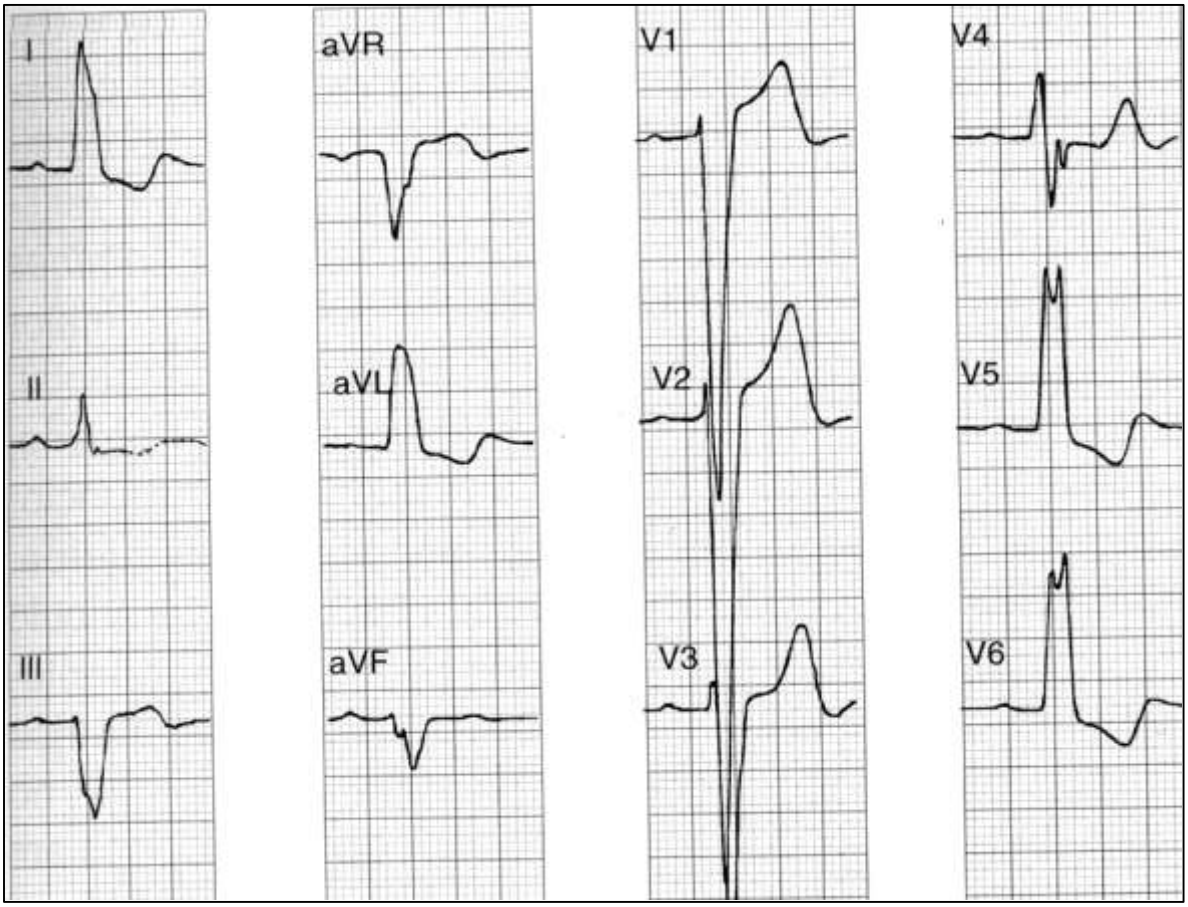


Figure 9. Clinical diagnosis: man, 60 y, afro descendent, with hypertension and coronary heart disease. ECG diagnosis: Classical CLBBB, wider QRSD (QRSD = 170 ms) in the presence of supraventricular command; SAQRS –10º. Monophasic R wave in left leads I, aVL, V5 -V6. QRS complexes predominantly negative in V1 and V2 type rS, ST/T opposite to QRS: discordant appropriate repolarization.

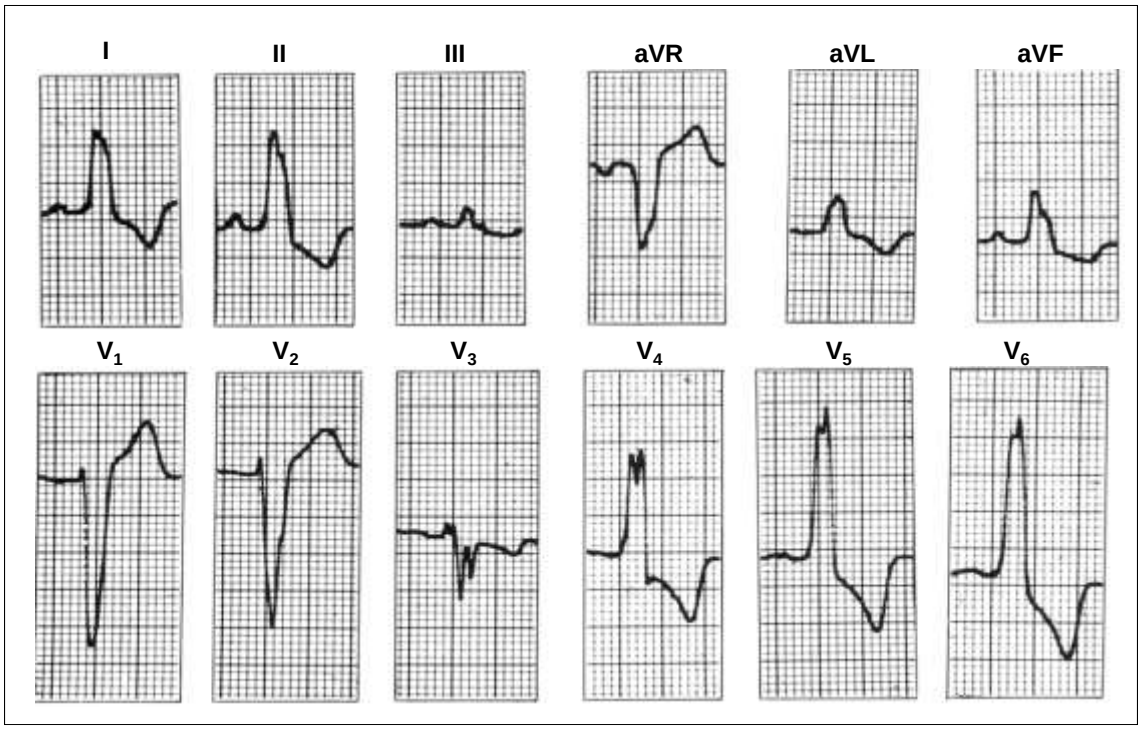


Figure 10. Clinical diagnosis: 45yo. woman with systemic hypertension a long time ago.ECG diagnosis: Classical CLBBB, wider QRS (QRSD = 160 ms) in the presence of supraventricular command, SÂQRS +40º, pure broad R wave in left lateral leads I, aVL, V5 and V6, QRS complexes predominantly negative in V1 and V2 rStype, ST/T opposite to QRS(Dobb).

LBBB with right axis deviation

LBBB Classification According to electrical axis of QRS complex in the Frontal Plane.

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 –30⁰: between -30° and -90° (Parharidis, Nouskas et al. 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, Rao et al. 2013).*

c) *With QRS axis deviated to the right: between +60° and +90° (≈ 3.5 a 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 E et al, 1951). The majority of subjects had dilated cardiomyopathy with biventricular enlargement (Childers, Lupovich et al. 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 and Marriott 1985). Causes that determine paradoxical complete LBBB:*

e) *Complete LBBB associated to right ventricular hypertrophy/enlargement or severe cardiomyopathy with biventricular enlargement. or diffuse advanced myocardial disease >98% of cases.*

f) *Fascicular/ divisional LBBB (LAFB + LPFB) with a higher degree of block in the posteroinferior fascicle/division. In presence of AF LBBB with intermittent right axis deviation is explained by an additional LPFB accompanying prefascicular / divisional LBBB*

g) *LBBB in Wegener granulomatosis (Khurana, Mazzone et al. 2000)*

h) *Complete LBBB associated to lateral infarction (free wall of left ventricle)*

i) *Complete LBBB with accidental exchange of limb electrodes*

j) *Complete LBBB associated with true dextrocardia (Salazar, Garcia et al. 1978)*

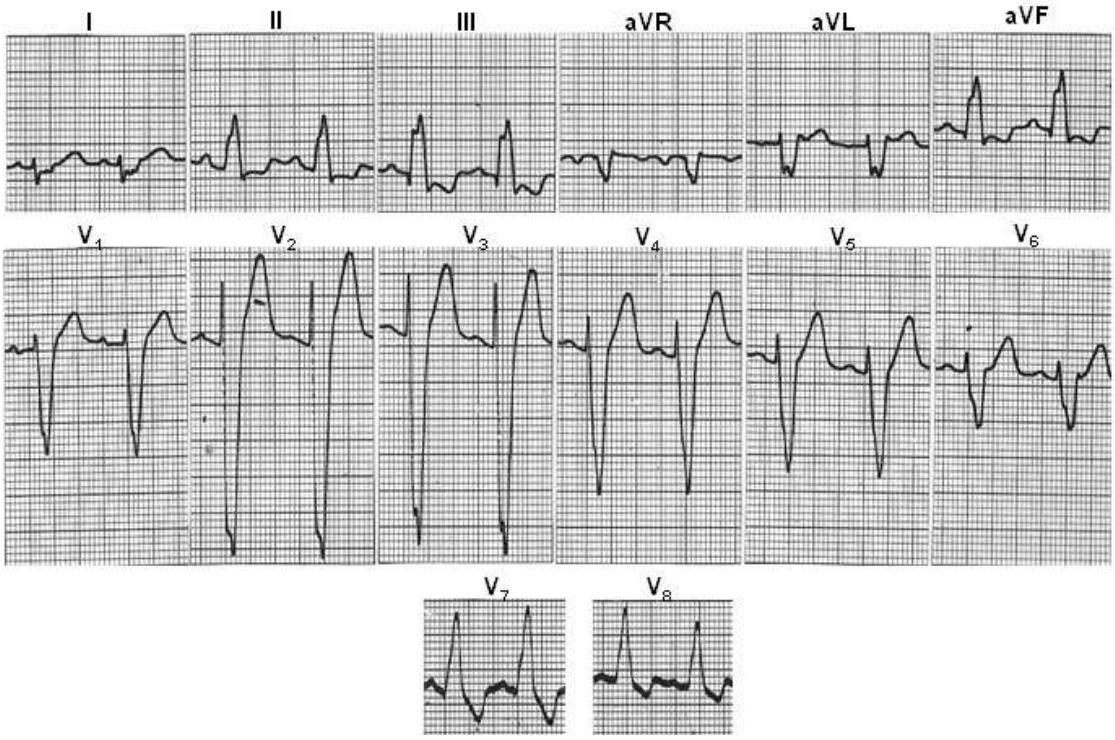
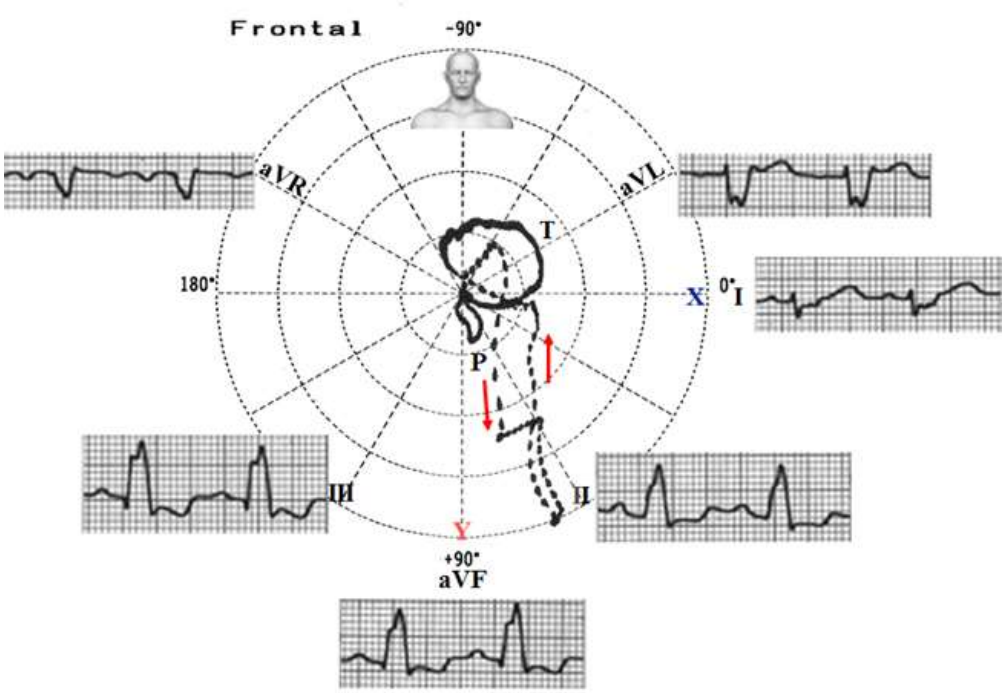


Figure 11. Atypical LBBB because rs in I and rS in aVL and rS from lead V1 through V6. The typical LBBB upward QRS is observed only in inferior and posterior leads (V7-V8).

ECG/VCG correlation on Frontal Plane

Figure 12. Right axis deviation. SÂQRS at +110º. QRS loop with predominant CCW rotation with maximal QRS vector +74º.



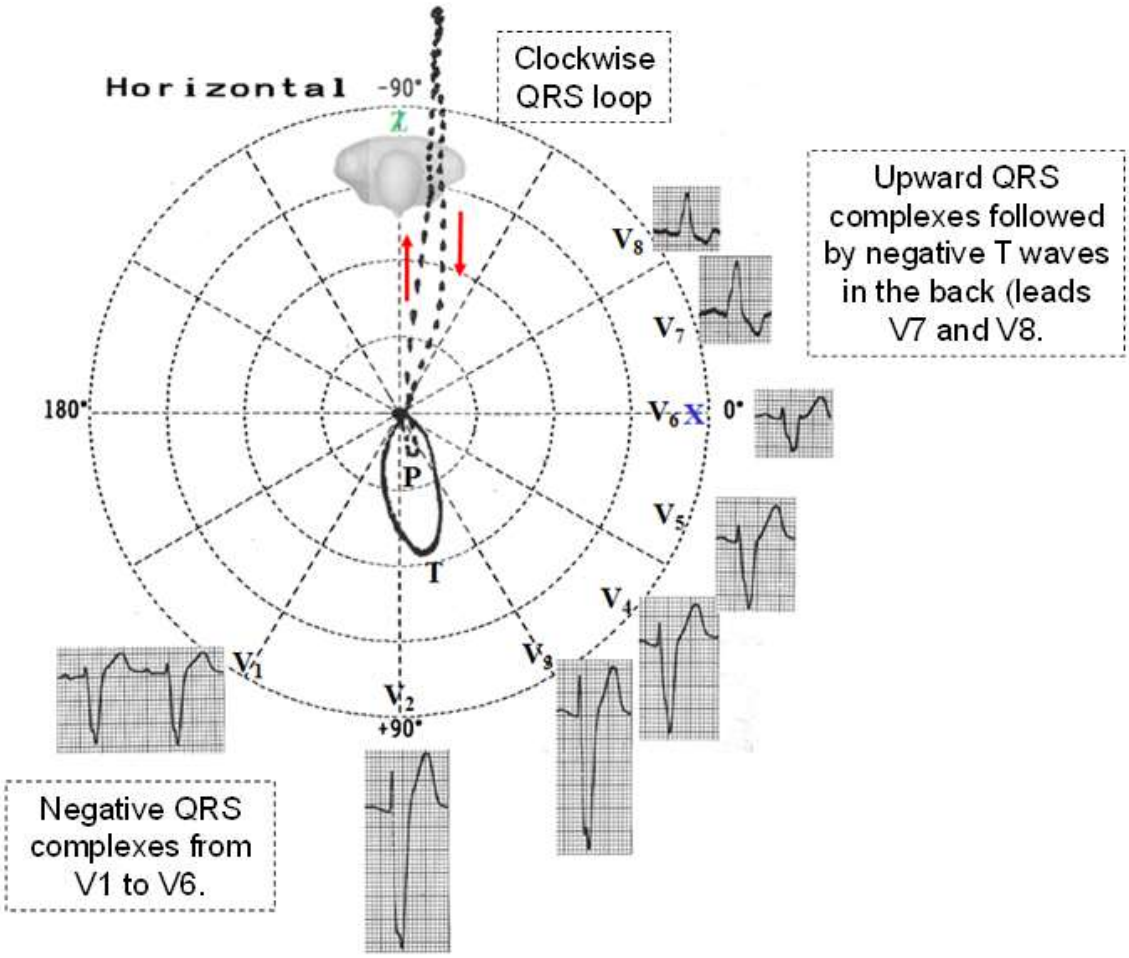


Figure 13. ECG/VCG correlation on Horizontal Plane

ECG/VCG correlation on RSP

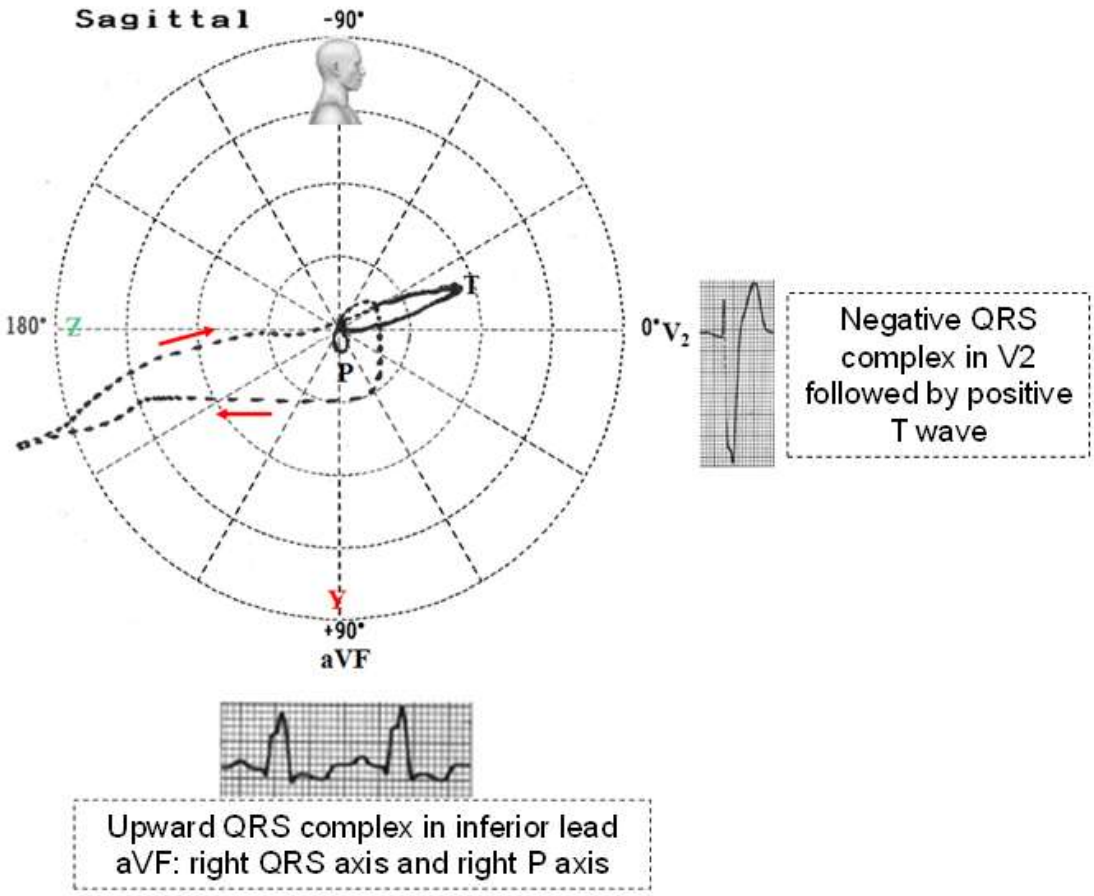


Figure 14. ECG/VCG correlation on RSP.

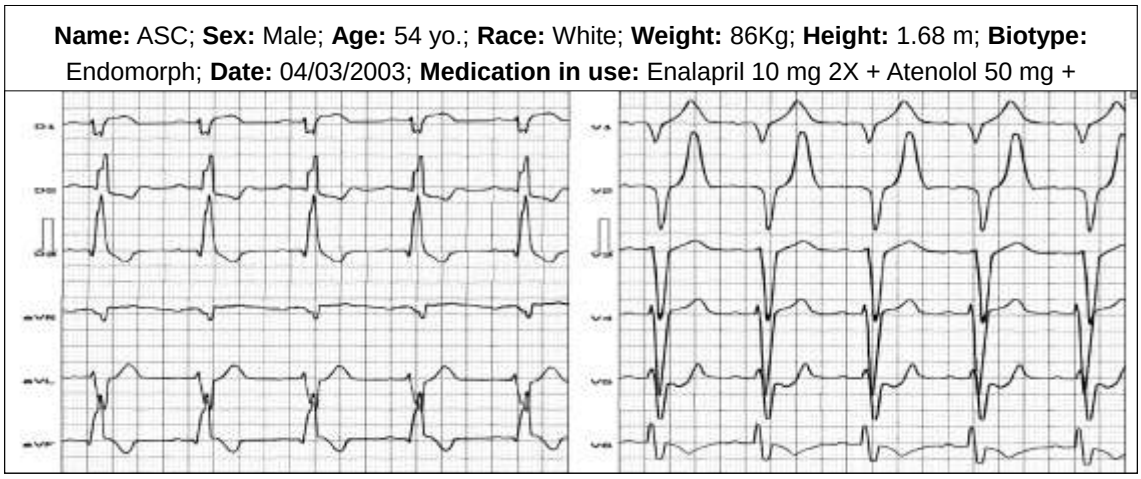


Figure 15. Clinical diagnosis: Hypertensive heart disease + aortic insufficiency by aortic cause. Echo diagnosis: Moderate concentric hypertrophy: septum 13 mm and posterior wall 14 mm. Moderate aortic insufficiency. ECG diagnosis: SR; HR: 72 bpm; SAP: +600; SAQRS: +110d; QRSD: 165 ms; I and aVL = rS; 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 reegreeferences 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 +115º. Conclusion: 1) CLBBB; 2) LPFB (Left Posterior Fascicular Block).

ECG/VCG correlation on Frontal and Horizontal Plane

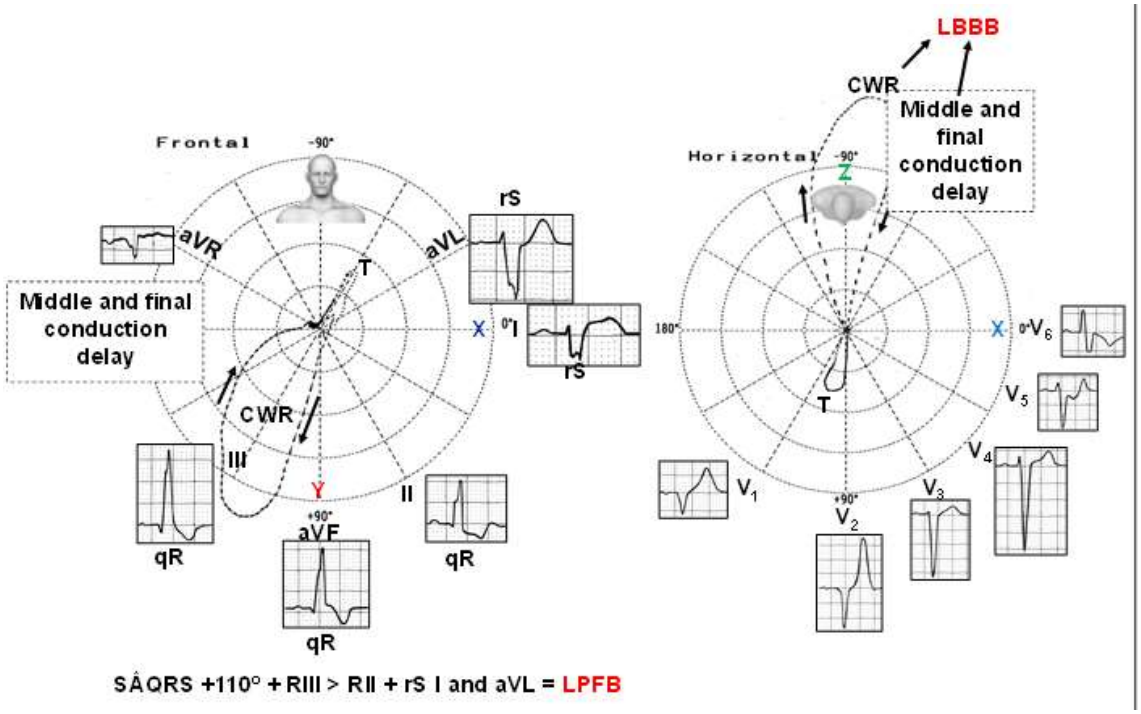


Figure 16. ECG/VCG correlation on Frontal and Horizontal Plane.

ECG / VCG difference between LBBB and LBBB associated to RVH on HP (Te-Chuan Chou, 1971)

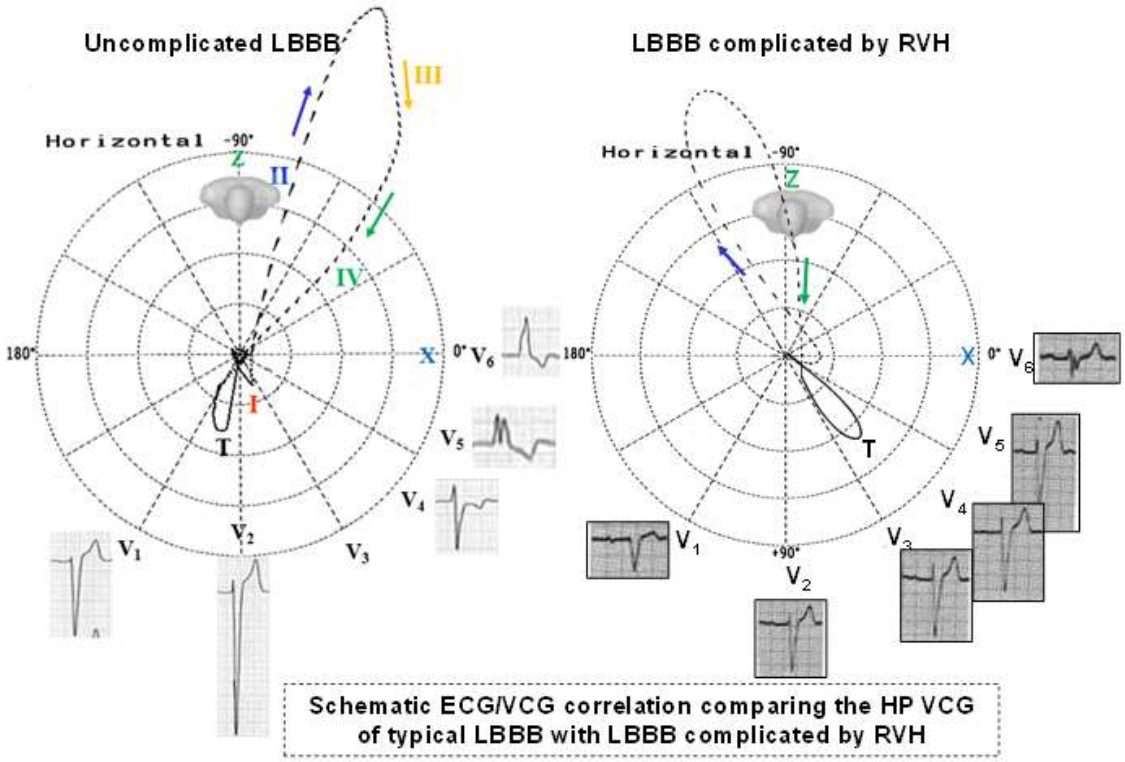


Figure 17. ECG / VCG difference between LBBB and LBBB associated to RVH on HP.

VCG characterization of right ventricular hypertrophy in the presence of LBBB

The VCG characteristics are:

- QRS loop duration with prolongation;
- Slow inscription of the mid and late portion of the QRS loop;
- Leftward and inferior orientation of the initial QRS vectors;
- Posterior and rightward displacement of the maximum QRS vector;
- Clock-wise inscription of the major portion of the QRS loop in the HP;
- 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)

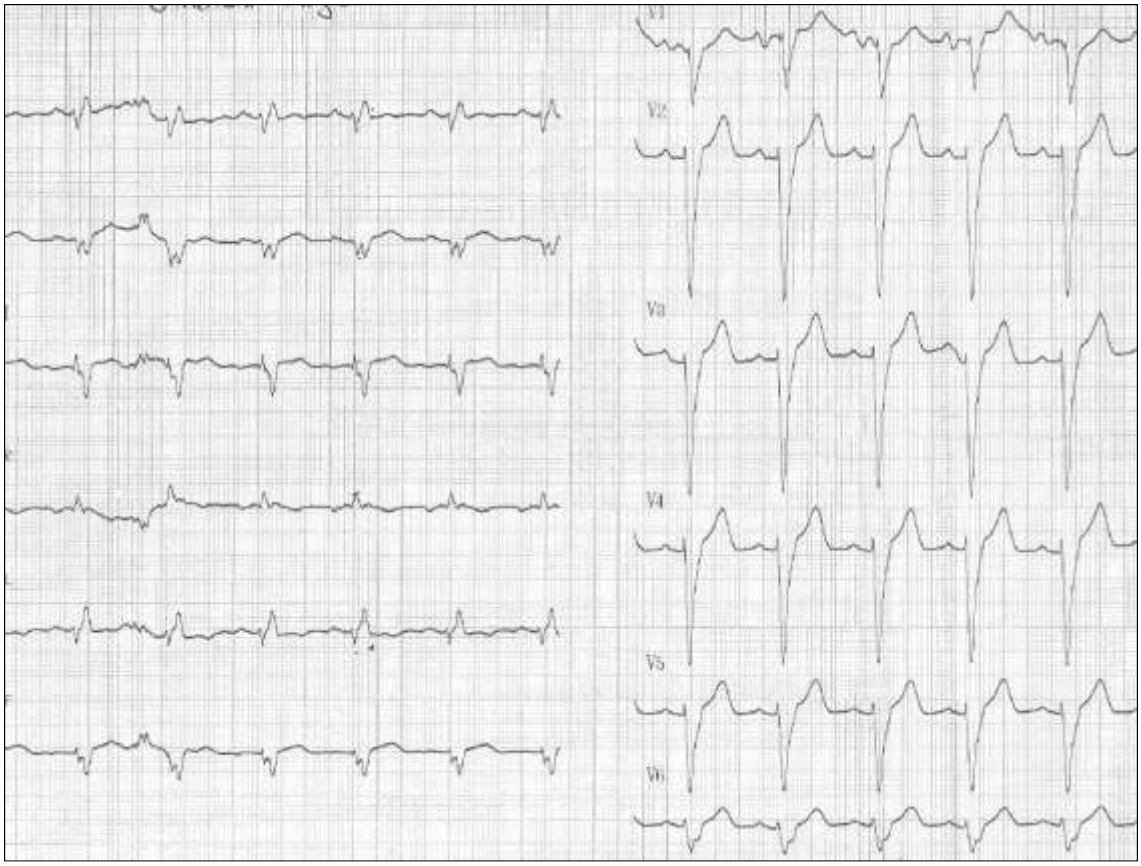


Figure 18. ECG tracing.

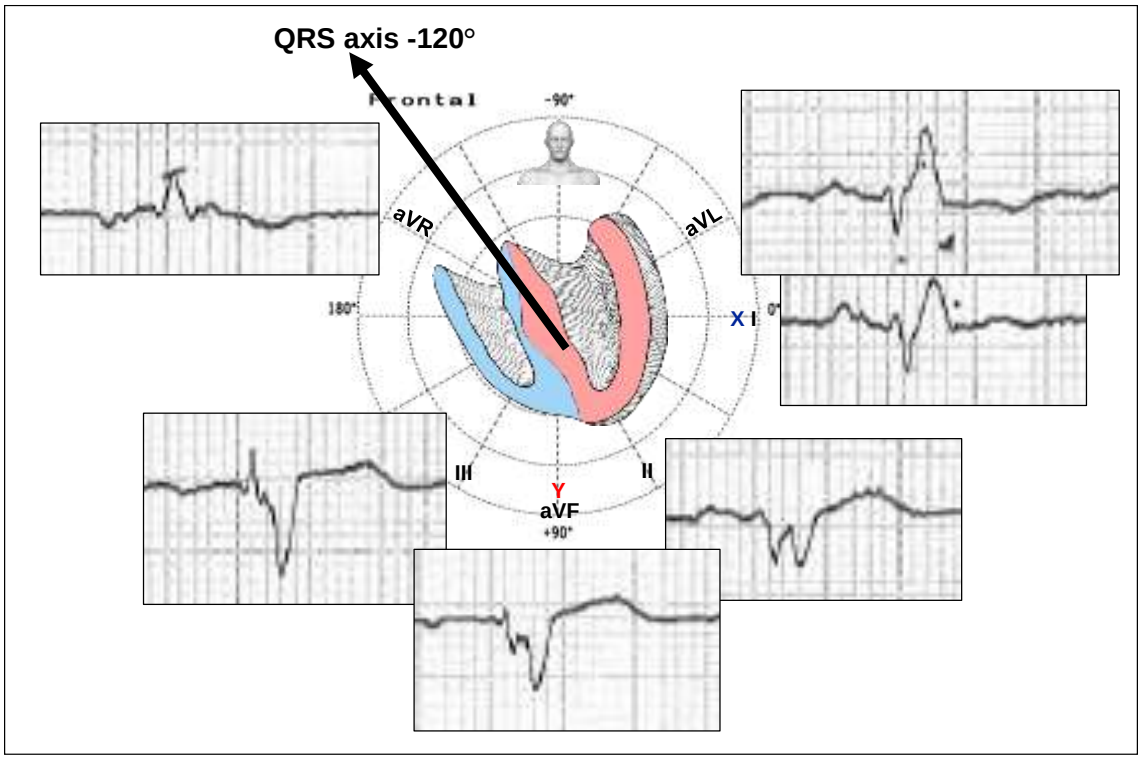


Figure 19. Prolonged P duration (140ms), bimodal shape, P axis +50º PR 170ms, very prolonged QRS duration 170ms, QRS axis -120º: No man's land. Fragmented wide QRS in inferior leads. Conclusion: left atrial enlargement+ Severe right ventricular hypertrophy (AKA "Northwest Axis" Extreme Right axis or No man's land) + LAFB + fragmented wide QRS(fw-QRS) high risk of arrhythmia.

The prognosis factors in Complete Left Bundle Branch Block

LBBB is a common ECG abnormality seen in patients, in whom cardiac conduction along the anterior, mid and posterior left fascicles of the His-Purkinje system is compromised (fascicular or divisional LBBB).

Although LBBB is often associated with significant heart disease and is often the result of myocardial injury or hypertrophy, it can also be seen in patients without LV disease.

An isolated LBBB without cardiac symptoms or abnormalities does not necessarily impair the prognosis of the patient. However, LBBB can have markedly negative prognostic impact, especially in patients presenting with acute chest pain, syncope and in those suffering from heart failure with reduced LV ejection fraction. New onset LBBB should always be considered a sign of pathology and is a marker of acute

myocardial infarction in a small proportion of patients. Although LBBB is no longer considered as an equivalent to ST-segment elevation MI equivalent in patients presenting with chest pain, concordant LBBB (Sgarbossa criteria), especially if new-onset, may indicate acute coronary occlusion. LBBB is associated with poorer prognosis both in comparison to normal intraventricular conduction and RBBB (Baldasseroni, Opasich et al. 2002) (Freedman, Alderman et al. 1987) (Hesse, Diaz et al. 2001) (Schneider, Thomas et al. 1979).

Patients with LBBB have increased rates of cardiovascular mortality, sudden cardiac death and heart failure (Baldasseroni, Opasich et al. 2002) (Hesse, Diaz et al. 2001) (Rotman and Triebwasser 1975) (Schneider, Thomas et al. 1979) (Smith and Hayes 1965).

Chronic BBB and nonfunctional atrioventricular (AV) block induced by incremental atrial pacing and/or infranodal conduction time (HV≥70 ms) had a significantly higher incidence of progression to spontaneous 2nd- or 3rd -degree AV block, with subjects with HV interval ≥100 ms presenting the highest risk (Petrac, Radic et al. 1996) (Peters, Scheinman et al. 1982, Scheinman, Peters et al. 1983). Compared with concordant LBBB, discordant LBBB morphology was associated with more severe CAD (Khalil, Bernard et al. 2016) and HF and worse prognosis, even in patients receiving a CRT with defibrillator capacity (Padeletti, Aimo et al. 2018).

Additionally, there was a trend towards more frequent occurrence of VT/VF/deaths in patients with discordant than in concordant LBBB, but statistical significance was not reached. Isolated LBBB is associated with an increased risk of developing overt cardiovascular disease and increased cardiac mortality.

The study included 110,000 participants in a screening program, 310 subjects with BBB without apparent or suspected heart disease were identified. Their outcome after a mean follow-up of 9.5 years was compared with that of 310 similarly screened age- and sex-matched controls (Fahy, Pinski et al. 1996).

In a study by Eriksson et al. with 28 years of follow up with 7392 men without a history of myocardial infarction or stroke and without angina or dyspnea at baseline, men with LBBB had increased risk of developing AMI, heart failure, high-degree atrioventricular block and increased risk of coronary death, but not all-cause mortality.

Thus, LBBB can be a sign of a progressive degenerative disease that affects not only the conduction system but also the myocardium itself (Eriksson, Wilhelmsen et al. 2005).

It should be realized that one cannot exclude the possibility of undetected cardiovascular disease in patients with LBBB.

While the prognosis of isolated LBBB without associated cardiovascular disease varies from controversial to neutral, in otherwise normal hearts, LBBB leads to mechanical asynchrony with reduction of LV ejection fraction and redistribution of circumferential shortening and myocardial blood flow from the septum to the left lateral wall. It was shown in an animal model study that LBBB leads to asymmetric hypertrophy and dilatation of the left ventricle. Thus, LBBB can solely initiate remodeling in a normal heart (Vernooy, Verbeek et al. 2005).

In several studies on chronic and acute CAD, LBBB was found to be an excellent predictor of mortality and events (Freedman, Alderman et al. 1987); (Hindman, Wagner et al. 1978); (Wong, Stewart et al. 2006); (Col and Weinberg 1972) (Guerrero, Harjai et al. 2005)

In 681 patients with acute myocardial infarction (AMI) enrolled in the Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) (McCullough, Hassan et al. 2005) and Global Utilization of Streptokinase and t-PA for Occluded Arteries (GUSTO) 1 protocols (Newby, Pisano et al. 1996), the incidence of LBBB was found to be 7%. The occurrence of both RBBB and LBBB was closely related to factors indicating more extensive myocardial damage (such as number of diseased vessels, peak creatinine phosphokinase, ejection fraction) and mortality. In patients showing persistent rather than transient BBB, the 30 days-risk of death was six times higher than in those without BBB, patients with LBBB mostly contributing to this outcome.

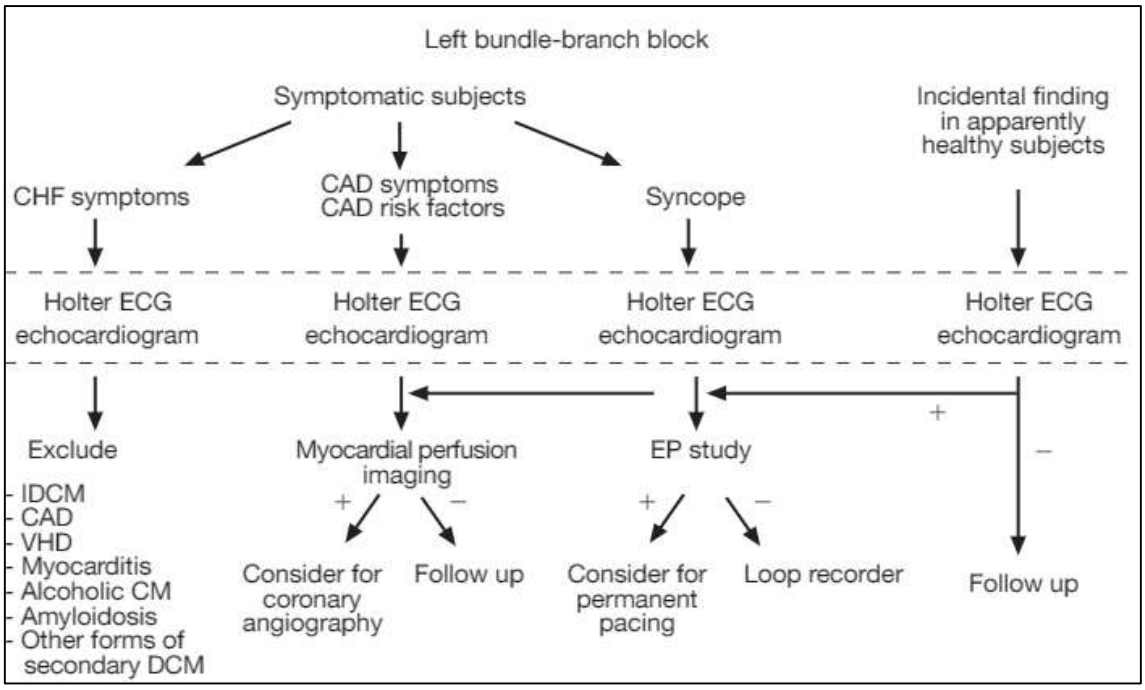


Figure 19. Flow-chart of proposed clinical approach to an individual or patient presenting with left bundle-branch block. CHF = congestive heart failure, CAD = coronary artery disease, EP = electrophysiological, IDCM = idiopathic dilated cardiomyopathy, VHD = valvar heart disease, CM = cardiomyopathy, DCM = dilated cardiomyopathy.