Apparently healthy elderly male with LBBB extreme right QRS axis deviation beyond +90º: type IV LBBB or paradoxical type of Lepeschkin

Case report

English

Reason for the consultation: periodic biannual evaluation by the club's requirement.

Caucasian patient, 67 years old, athletic, physical trainer, asymptomatic, denies addictions, hypertension, dyslipidemia or diabetes. Daily physical training, excellent health. Family history nothing worthy of note. He says that he has known for more than 30 years that he has a complete LBBB.

Physical examination: unremarkable.

See the ECG in the next slide.

We will present the VCG after the ECG analysis.

Transthoracic echocardiogram: A very dynamic abnormal posterior motion of the interventricular septum occurring within 0.04 seconds of the onset of the QRS complex and preceding the anterior motion of the posterior left ventricular wall during ventricular ejection was observed, typical of complete LBBB.

Portuguese

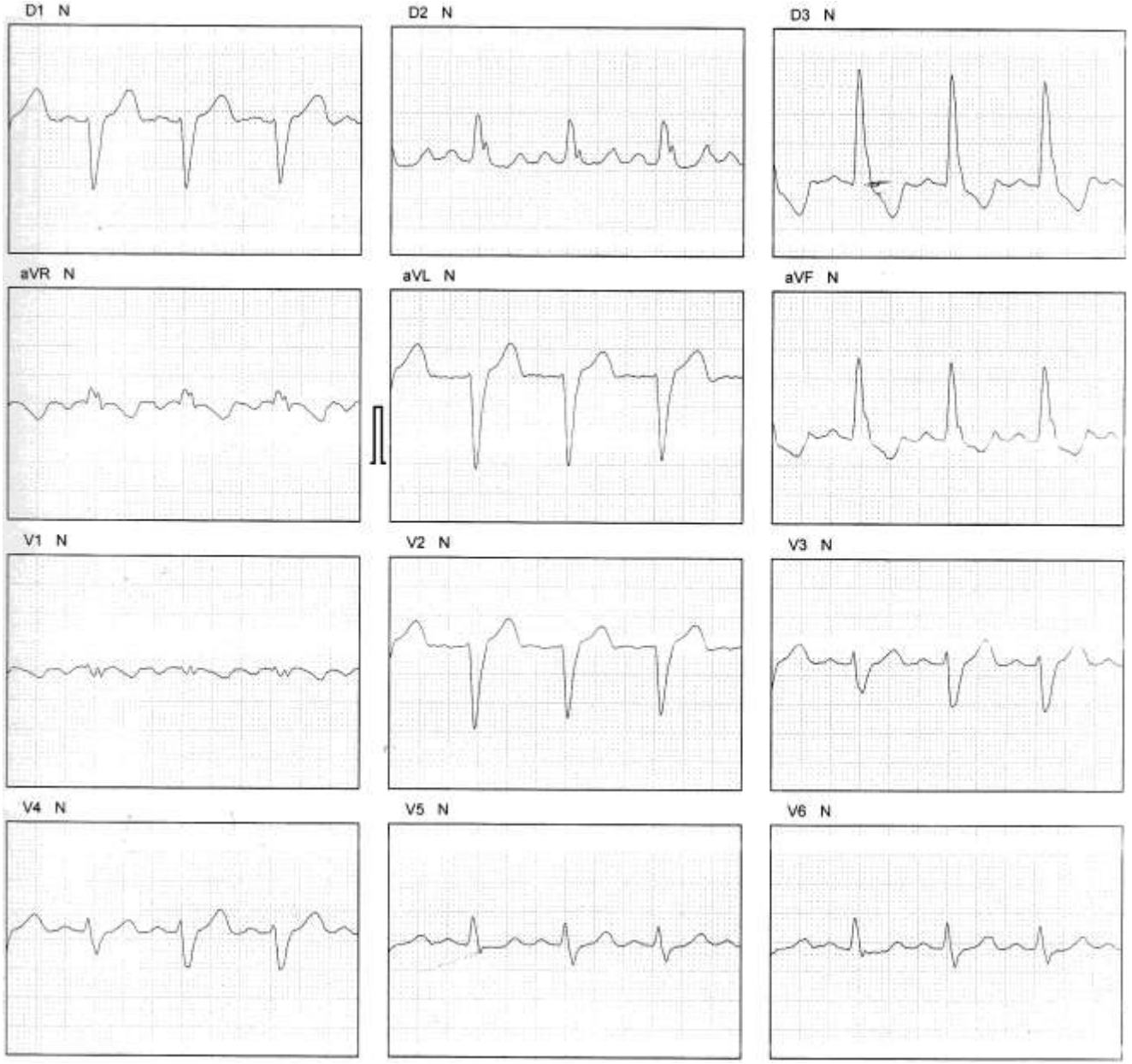
Motivo da consulta: avaliação periódica semestral por exigência do clube.

Paciente branco, de 67 anos, atlético, preparador físico, assintomático, nega vícios, hipertensão, dislipidemia ou diabetes. Treinamento físico diário, ótimo estado de saúde aparente. Antecedentes familiares nada digno de nota. Refere que há mais de 30 anos sabe que é portador de BCRE.

Exame físico: nada digno de nota.

Ecocardiograma transtorácico: movimento anormal muito dinâmico posterior do septo interventricular ocorrendo dentro de 0,04 segundos do início do complexo QRS e precedendo o movimento anterior da parede posterior do ventrículo esquerdo durante a ejeção ventricular foi observado, típico do BRE completo.

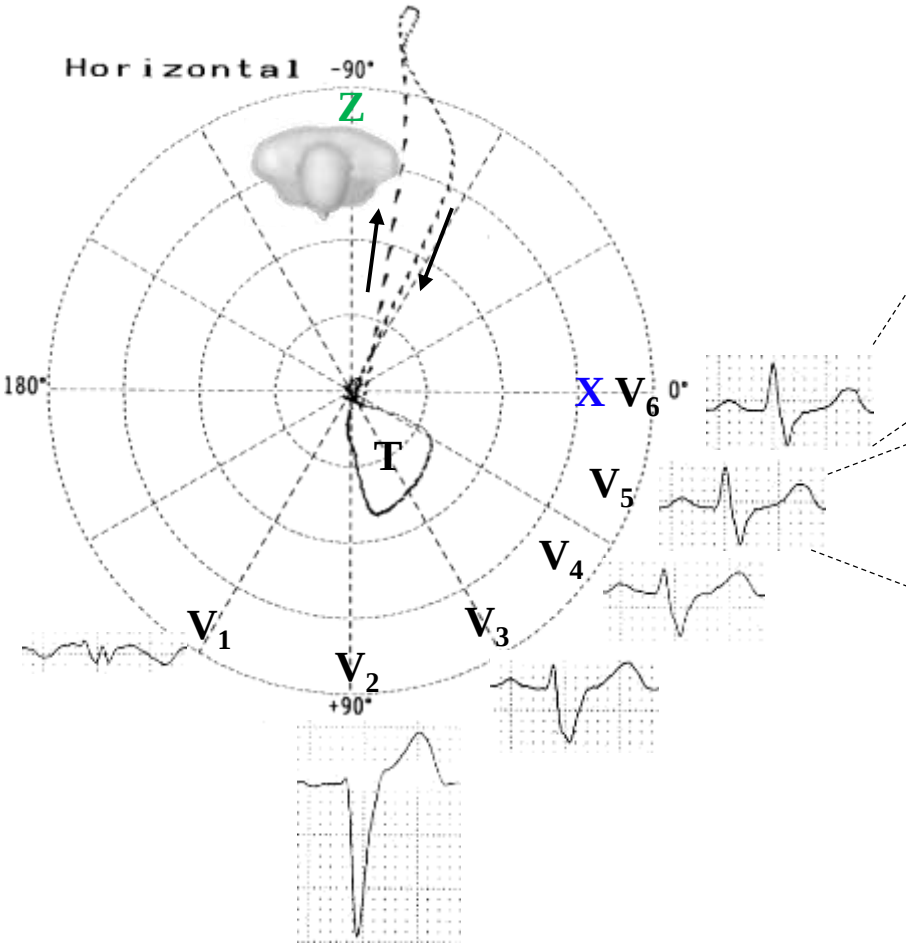
Name: MV; Age: 67 y/o; Sex: Male; Height: 1.82 m; Weight: 83 kg; Race: Caucasian; Date: February 14, 2019; Medication in use: none.

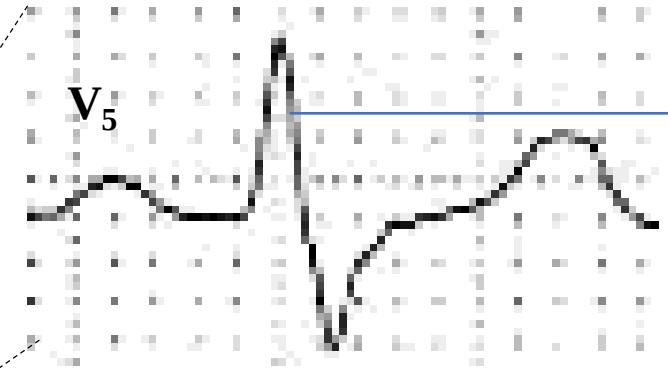


Diagnosis: SR, HR 94 bpm, Pd 110 ms, P voltage 1.5 mm, P axis +50°, PR interval 170 ms, QRSd 160 ms, QRS axis +130°. Conclusion: LBBB with extreme right axis deviation consequence of a higher degree of blockage in the left posterior fascicle compared to the left anterior fascicle.

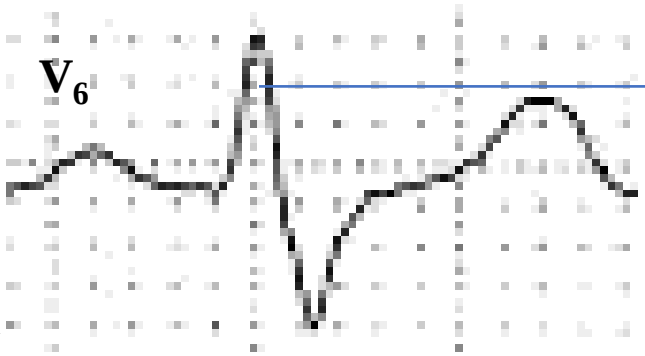
V5 y V6 es la misma figura, eso esta mal cortado...chequea y corregilo!
V5 and V6 is the same figure, that is badly cut ... check and correct it!
AB
Querido amigo me parece que estás equivocado. Te mando el plano horizontal en la correlación ECG/VCG
Dear friend, I think you are mistaken. I send you the horizontal plane in the ECG / VCG correlation.
Andrés.

V_5 and V_6 are very similar but not equal





R-wave voltage = 4mm



R-wave voltage= 3.3 mm

Querido Potro:
Presenta como refiere un BCRI. No presenta BAV de primer grado.
El eje eléctrico del QRS localizado a la derecha (+130°) causado por bloqueo de ramo derecho (en aVR y V1 presenta patrón rSr que evidencia el BRD enmascarado). Concomitante y sobrecarga ventricular derecha probablemente por su entrenamiento físico.
Conclusión: bloqueo bifascicular.
Un abrazo
Dr, Martín Ibarrola
Instituto Cardiovascular Dr. Martín Ibarrola Gran Buenos Aires, Buenos Aires, Argentina
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English
Dear Potro (Andrés´s nick name):
It presents as referred a Complete LBBB. No first-degree AVB.
The electric axis of the QRS located to the right (≈+ 130 °) caused by right bundle branch block (aVR and V1 shows rSr pattern evidenced by masked RBBB). Concomitant right ventricular overload probably due to their physical training.
Conclusion: bifascicular block.
A hug

Martín Ibarrola MD

Instituto Cardiovascular Dr. Martín Ibarrola Gran Buenos Aires, Buenos Aires, Argentina

Andrés : in your comments please discuss the slight but obvious QRS differences between the 3 QRS beats shown. I would have loved seeing high precordial leads.
Finally you will have to explain the prominent (and carrying) S wave in V4-V5-V6
In any case I am not anxious about the patient. The reason is that LBBB with normal or right axis usually have an excellent long term prognosis.
Warmest regards
Bernard Belhassen Tel Aviv Israel

BB: In other words...the ECG looks sicker than the patient.@@@
Andrés: Yes dear BB, the patient is healthy and the ECG is scary.

Dear BB: Right axis deviation with LBBB is an unusual combination. From a database of 636,000 ECGs Childers et al (1) reported a series of 36 patients with this association. The majority of subjects had dilated cardiomyopathy with biventricular enlargement. LBBB was fixed in 21 of 36 cases. It was freshly acquired, episodic, intermittent, or physiologic in 15 of 36. The right axis deviation was episodic in 30 of 36; it was fixed and concurrent with LBBB in only 2 cases, and never episodically concurrent. Reported for the first time here were 4 of 36 cases in which the combination of LBBB and right axis deviation was elicited with atrial premature impulses as a rare form of QRS aberration. In one case where the combination was intermittent, a clear relationship with freshly acquired intermittent left posterior fascicular block was demonstrated. The possible relationship of the deviation with variable degrees of right ventricular overload is possible
Nikolic et al. presented three patients with primary congestive cardiomyopathy (COCM), complete LBBB and right axis deviation. They reviewed 50 additional patients from the literature since 1950 indicates that the rare combination of LBBB and right axis deviation is a marker of severe myocardial disease, especially COCM. The mechanism of production of this electrocardiographic pattern appears to be diffuse conduction system involvement in advanced myocardial disease(2).

1. Childers R, Lupovich S, Sochanski M, Konarzewska H.Left bundle branch block and right axis deviation: a report of 36 cases.J Electrocardiol. 2000;33 Suppl:93-102
2. 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.

Final Comments
Andrés Ricardo Pérez-Riera

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). The presence of LBBB in a structurally normal heart is usually accompanied by a frontal plane QRS axis that ranges between +60° and −30°. This is because the mean QRS vector moves leftward and superior as the left ventricle gets activated transseptally; the same holds true during supraventricular tachycardia (**Wellens HJ. 2001**.)

- With QRS axis with extreme left axis deviation(LAD): beyond –30° (≈25% of cases). LBBB with LAD is associated with a higher incidence of myocardial dysfunction, more advanced disease of the conduction system, and an earlier mortality rate than is LBBB with a normal axis (**Dhingra RC, 1978**)

- With QRS axis right axis deviation(RAD): between +60° and +90° (≈ 3.5 a 5% of cases)

- With QRS axis with extreme RAD: beyond +90° (< than 1% of cases). It is called "paradoxical type of Lepeschkin“ (**Lepeschkin 1951**).

The Occurrence of RAD is therefore extremely unusual when there is LBBB. It is usually associated with a normal axis or LAD. When it is seen in association with RAD it is felt to be a marker of diffuse dilated myocardial disease(**Nikolic G, 1985**). Khurana et al described a case of Wegener granulomatosis with LBBB and RAD in which predivisional LBBB with predominant left posterior fascicular block secondary to mechanic calcification(Lev disease), genetic (Lenegre disease) ischemic, or inflammatory involvement of the conduction system as a possible explanation. (**Khurana C, 2000**). In the present case the mechanism for the ECG pattern of LBBB with extreme RAD is unknown. In these cases the authors speculated to result from association with right ventricular hypertrophy (RVH), cor pulmonale, or left ventricular free wall

Myocardial infarction (**Doucet P, 1966**). Some investigators (**Rosenbaum MB, E 1970; Vera Z 1972**) have ascribed the extreme RAD in LBBB to altered intraventricular conduction using the fascicular bock model. Atrial pacing techniques, while confirming the fascicular block concept, have not to date evoked LBBB aberration with an extreme RAD (defined as an axis ≥90° reflecting the rarity of this combination). This patient did not have RVH, cor pulmonale, or extensive left ventricular free wall infarction. Predivisional LBBB with predominant left posterior fascicular block secondary to several possibilities:

1) **Lev's disease** or progressive idiopathic sclerosis of the "cardiac skeleton". It has a clinical behavior similar to Lenègre disease, however, it occurs in elderly patients**:** Fibrosis and sclerosis of the conduction system is the most common cause of acquired conduction system disease, accounting for about half of cases of AV block, and can be induced by several different conditions, which often cannot be distinguished clinically. Lev's disease is a result of proximal bundle branch calcification or fibrosis. It is postulated as a hastening of the aging process by hypertension and arteriosclerosis of the blood vessels supplying the conduction system.

2) **Calcification of the aortic or (less commonly) mitral valve annulus** can extend to the nearby conduction system and produce AV block. As noted, the HB penetrates the central fibrous body adjacent to the fibrous continuity between the aortic and mitral valves that is the usual site of dystrophic calcification, and extension of calcification can directly involve the HB or the origin of the LBBB, or both. Coronary atherosclerosis severity index (CASI) depended on aortic stenosis (AS) severity. In subgroups without AS and diabetes mellitus CASI was associated with combined presence of AVC and mitralannulus calcification, glomerular filtration rate , and besides with age and cholesterol level in man.(**Ivanov VP 2018**) .

3. **Lenègre's disease, progressive cardiac conduction defect (PCCD) or "idiopathic" sclerosis of the intraventricular His system:** by mutation in the SCN5A gene, (the same one affecting Brugada Syndrome) or others.it is a sclerodegenerative process that occurs in a younger population and involves the more distal portions of the bundle branches. Calcification of the aortic or (less commonly) mitral valve annulus can extend to the nearby conduction system and produce AV block. in heritable progressive cardiac conduction disease (referred to as hereditary Lenègre disease, progressive cardiac conduction disease, and familial AV block), conduction slowing may be attributed to loss-of-function mutations in *SCN5A*. Whether age-dependent fibrosis of the conduction system is a primary degenerative process in progressive cardiac conduction disease or a physiological process that is accelerated by Na^+ current (I_{Na}) reduction is still unknown. Lenegre disease is secondary to genetic nutations of the heart electrical conduction system and may cause syncope and sudden death. Schott et al reported the first mutation in the *SCN5A* gene that segregated with progressive conduction defect (PCCD) in an autosomal-dominant manner in a large French family and a second *SCN5A* mutation that co-segregated in a smaller Dutch family with familial nonprogressive conduction defect (**Schott JJ, 1999**). Fifteen patients from the French family were clinically and electrocardiographically affected (the mean QRS duration was 135 ± 7 ms). RBBB was present in five patients, LBBB in two, LAFB or LPFB in three, and long PR interval (>210ms) in eight. None of the patients had structural heart disease. Of significance, four patients received a pacemaker implantation because of syncope or complete AV block, and in a number of affected patients, the conduction defect increased in severity with age. On the other hand, in the Dutch family, the proband presented after birth with an asymptomatic first-degree AV block associated with RBBB. Three brothers were asymptomatic, one of whom had RBBB, and the asymptomatic mother had a nonspecific conduction defect with a QRS duration of 120 ms. By use of markers flanking *SCN5A* in the French family, these investigators demonstrated segregation of the disease with marker D3S1260 in every affected individual, and analyses with flanking markers of the region confirmed a linkage to the 3p21 locus. Sequencing the entire

3. Continuation….. *SCN5A* coding region in this family identified a T→C substitution in the highly conserved +2 donor-splicing site of intron 22. This abnormal transcript predicts an in-frame skipping of exon 22 and an impaired gene product lacking the voltage-sensitive domain III S4 segment. Importantly, this mutation was found in all affected members, but not in 100 control chromosomes. In the Dutch family, sequence analysis of the *SNC5A* gene These findings also indicated that with aging there is a progressive increase in cardiac fibrosis, which, in association with the *SNC5A*gene mutation, can slow the impulse along the electrical conduction system. In the Dutch family, the mutation conferring a premature stop codon and the presentation of PCCD at birth suggests that as a consequence of the sodium channel mutation a congenital phenotype can arise that may be either progressive or immediate. It is worth noting that none of the affected individuals had LQTS or BrS, although heterozygous mutations in the cardiac *SCN5A* gene have been associated with LQTS, BrS, and progressive conduction system disease. The same mutation in *SCN5A* can lead either to BrS or to an isolated cardiac conduction defect (**Kyndt F, 2001**). In a large family with both BrS and isolated cardiac conduction defects, a G-to-T mutation at position 4372 was found in 13 affected mem bers and was predicted to change a glycine for an arginine (G1406R) between the S5 and S6 segments of domain III of the Na^+ channel protein. Four individuals showed typical BrS phenotypes, including ST-segment elevation in the right precordial leads and RBBB, and seven individuals had isolated cardiac conduction defects but no BrS phenotype; one patient with an isolated cardiac conduction defect (CDD) had an episode of syncope and required pacemaker implantation. These findings suggest that modifier gene(s) may influence the phenotypic consequences of a *SCN5A* mutation. Often a mutant cardiac sodium channel may be associated with multiple biophysical defects and concomitant clinical features of BrS and CCD. For example, LQT3, which is caused by mutations in the human cardiac *SCN5A* gene, may present, in addition to LQT, with bradycardia and sinus pauses. Veldkamp and associates reported the effect of the 1795insD Na^+ channel mutation (previously characterized by the presence of a persistent inward current (I_{pst}) at −20 mV and a negative shift in voltage dependence of inactivation) on

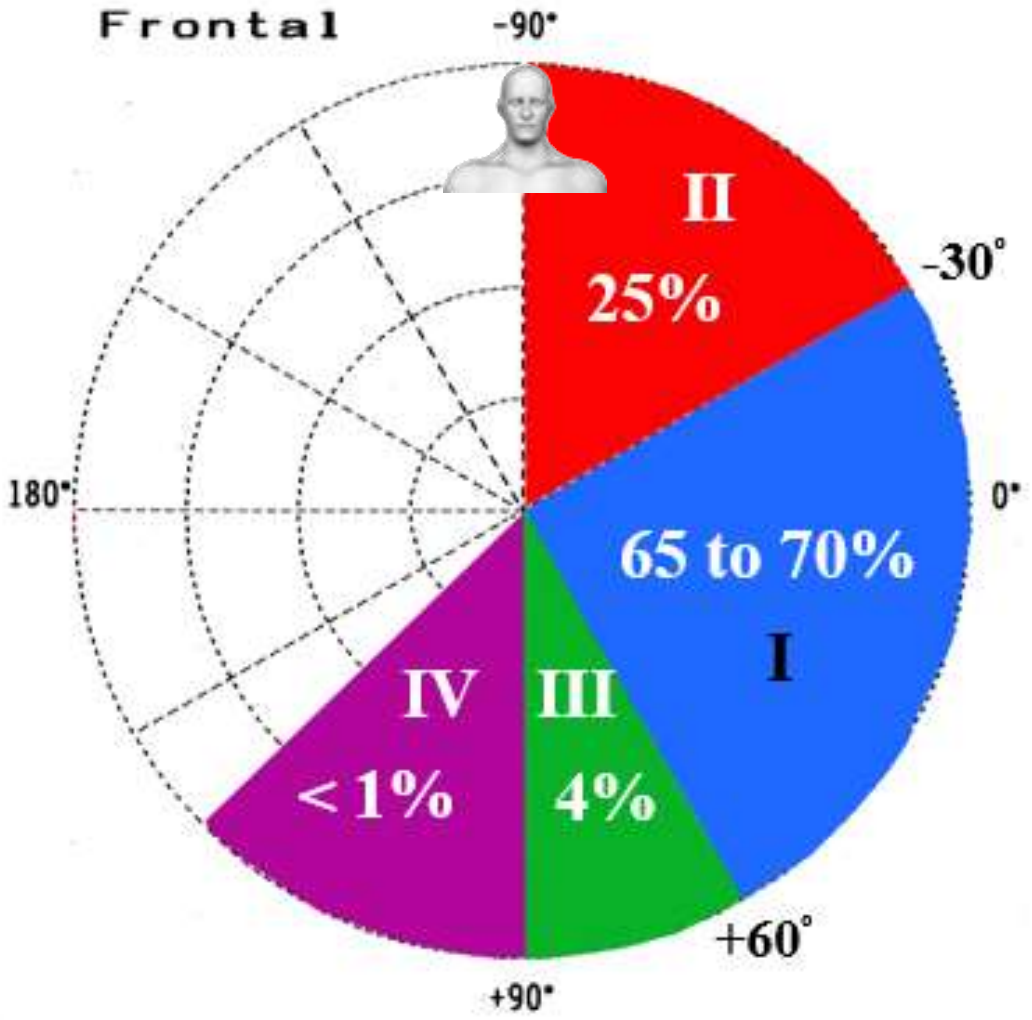
3. sinoatrial (SA) pacemaking.(Veldkamp MW, Wilders R, Baartscheer A, Zegers JG, Bezzina CR, Wilde AA.Contribution of sodium channel mutations to bradycardia and sinus node dysfunction in LQT3 families. Circ Res. 2003 May 16;92(9):976-83) By use of functional studies, I_{pst} was characterized over the complete voltage range of the SA node AP by measuring whole-cell Na^+ currents (I_{Na}) in HEK-293 cells expressing either wild-type or 1795insD channels. I for 1795insD channels varied between 0.8 ± 0.2% and 1.9 ± 0.8% of peak I_{Na}, and the activity of 1795insD channels during SA node pacemaking was confirmed by AP clamp experiments. When implemented into SA node AP models, the negative shift decreased sinus rate by decreasing diastolic depolarization rate, whereas I_{pst}decreased sinus rate by AP prolongation, despite a concomitant increase in diastolic depolarization rate. Furthermore, moderate I_{pst}together with the shift reduced sinus rate by approximately 10%. Further increase in I could result in plateau oscillations and failure to repolarize completely. The authors concluded that Na^+ channel mutations displaying an I or a negative shift in inactivation may account for the bradycardia seen in LQTS3, whereas SA node pauses or arrest may result from failure of SA node cells to repolarize under conditions of extra net inward current. On the other hand, a CCD such as complete atrial-ventricular block (AV block) or sick sinus syndrome (SSS) can be the only electrical rhythm disorder associated with *SCN5A* mutations. Wang and associates have reported the clinical, genetic, and biophysical features of two new *SCN5A* mutations that resulted in AV conduction block (**Wang DW, 2002**) Molecular analysis demonstrated two G to A transition mutations that resulted in the substitution of serine for glycine (G298S) in the domain I S5-S6 loop and asparagine for aspartic acid (D1595N) within the S3 segment of domain IV. Both mutations impair fast inactivation but do not exhibit sustained non-inactivating currents. The mutations also reduce Na^+ current density and enhance slower inactivation components. AP simulations predicted that this combination of biophysical abnormalities could significantly slow myocardial conduction velocity. In addition, Benson and associates have screened the α-subunit of *SCN5A* as a candidate gene in 10 pediatric patients from 7 families with congenital SSS(**Benson DW, 2003**.). a

3. molecular basis for some forms of congenital SSS and define a recessive disorder of a human heart voltage-gated sodium channel. Compound heterozygosity for six distinct *SCN5A* alleles, including two mutations previously associated with dominant disorders of cardiac excitability, was identified in probands from three kindreds. Among 27 heterozygotes, no individual exhibited ECG evidence of BrS. With heterologously expressed recombinant human heart sodium channels, biophysical characterization of mutant channels demonstrated either loss of function or significant impairment(s) in channel gating (inactivation) consistent with reduced myocardial excitability. These findings contribute to establishing a molecular basis for some forms of congenital SSS and in explaining a recessive disorder of a cardiac voltage-gated Na^+ channel. A novel Na^+ channel mutation in *SCN5A*, E161K, has been identified in individuals of two nonrelated families with symptoms of bradycardia, sinus node dysfunction, generalized conduction disease, and BrS, or combinations thereof. Mutation suggests that a loss of Na^+ channel function is not only associated with Brugada syndrome and conduction disease, but may also cause sinus node dysfunction in carriers of this mutation.(**Smits JP, 2005**) Functional studies of mutant Na^+ channels were performed with wild-type or E161K Na^+ channel α-subunit and β-subunit co-transfected into tsA201 cells. Whole-cell sodium current (I_{Na}) gauged using whole cell patch-clamp technique from cells containing the E161K mutation exhibited an almost threefold reduction in current density and an 11.9-mV positive shift of the voltage-dependence of activation, whereas the inactivation properties of wild-type and mutant Na^+ channels were similar. These data suggested an overall reduction of I_{Na} in E161K mutants. Computational models demonstrated a marked atrial and ventricular conduction slowing, as well as a reduction in sinus rate stemming from slowing of the diastolic depolarization rate and upstroke velocity of the sinus node AP. This reduction in sinus rate was further aggravated by application of acetylcholine, simulating the dominant vagal tone during the night. Rarely the ECG shows an LBBB with changing QRS morphology and changing axis deviation.

4. Ischemic
5. Minimal inflammatory involvement of the conduction system which rapidly reversed spontaneously is a possible explanation in the present case.

Percent distribution of the QRS electrical axis in cases of LBBB

- **I: Without deviation 65 to 70%;**
- **II With extreme deviation to the left: 25%;**
- **III With deviation to the right: 4%;**
- **IV With extreme deviation to the right <1%**

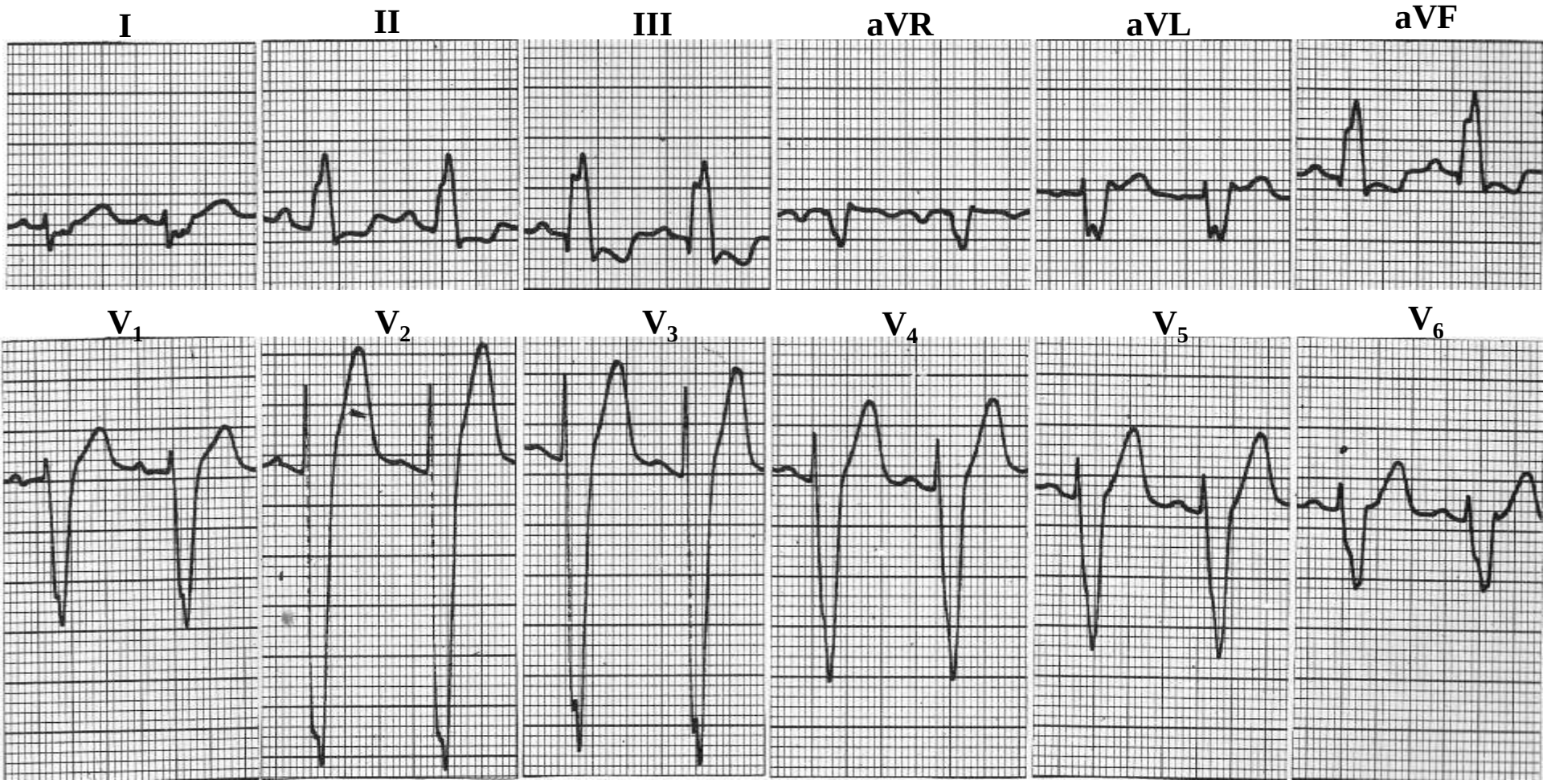


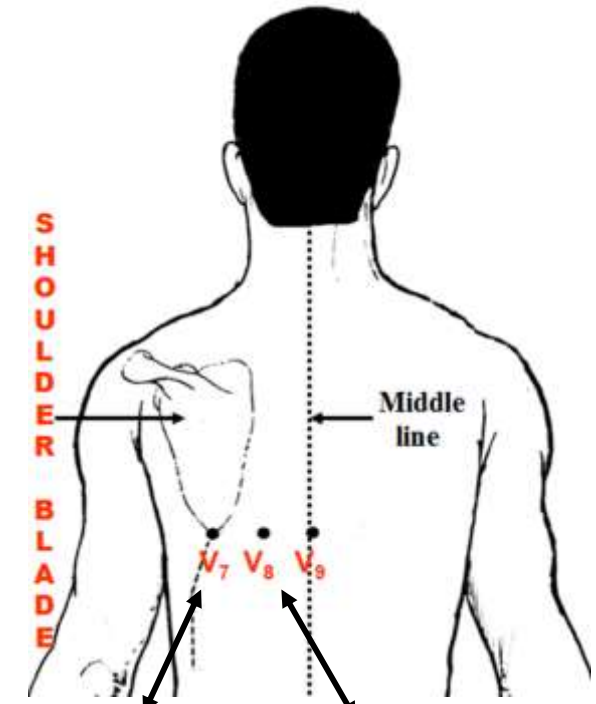
ECG VCG examples of LBBB with right axis deviation or extreme right axis deviation

Possible clinical scenarios

1) *LBBB associated with right ventricular hypertrophy/enlargement: Severe dilated cardiomyopathy, congestive heart failure, Chronic Obstructive Pulmonary Disease (COPD): Omious prognosis*
2) *Divisional LBBB with predominant left posterior fascicular block: frequent absence of structural heart disease.* The intermittent positive aspect of the neglected lead aVR indicates an intermittent right axis deviation in the presence of complete LBBB. Patanè et al describe the case of a 78-year-old woman admitted to the Cardiology Unit with acute myocardial infarction and permanent AF. The ECG showed AF and LBBB with intermittent left axis deviation or AF and LBBB with intermittent right axis deviation. An additional LPFB accompanying divisional LBBB is the possible explanation. (**Pahlm US 1996; Patanè S2008; Ranginani A 2000; Vera Z 1972.**
3) *Left bundle-branch block with technical right-axis deviation. Technical Problems/ Lead switches*
4) *LBBB associated with left ventricular free wall myocardial infarction:*

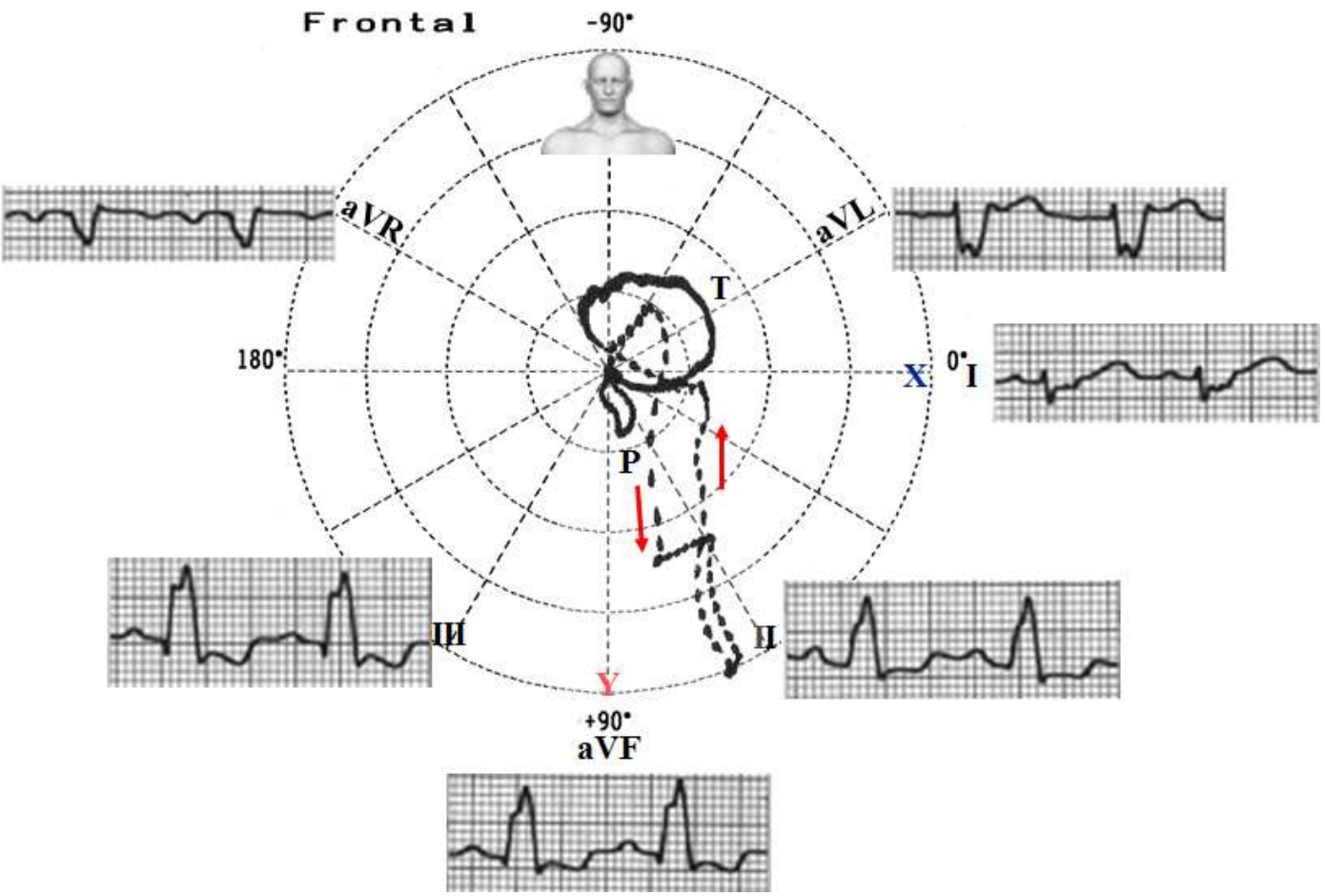
Examples of LBBB associated with right ventricular hypertrophy/enlargement





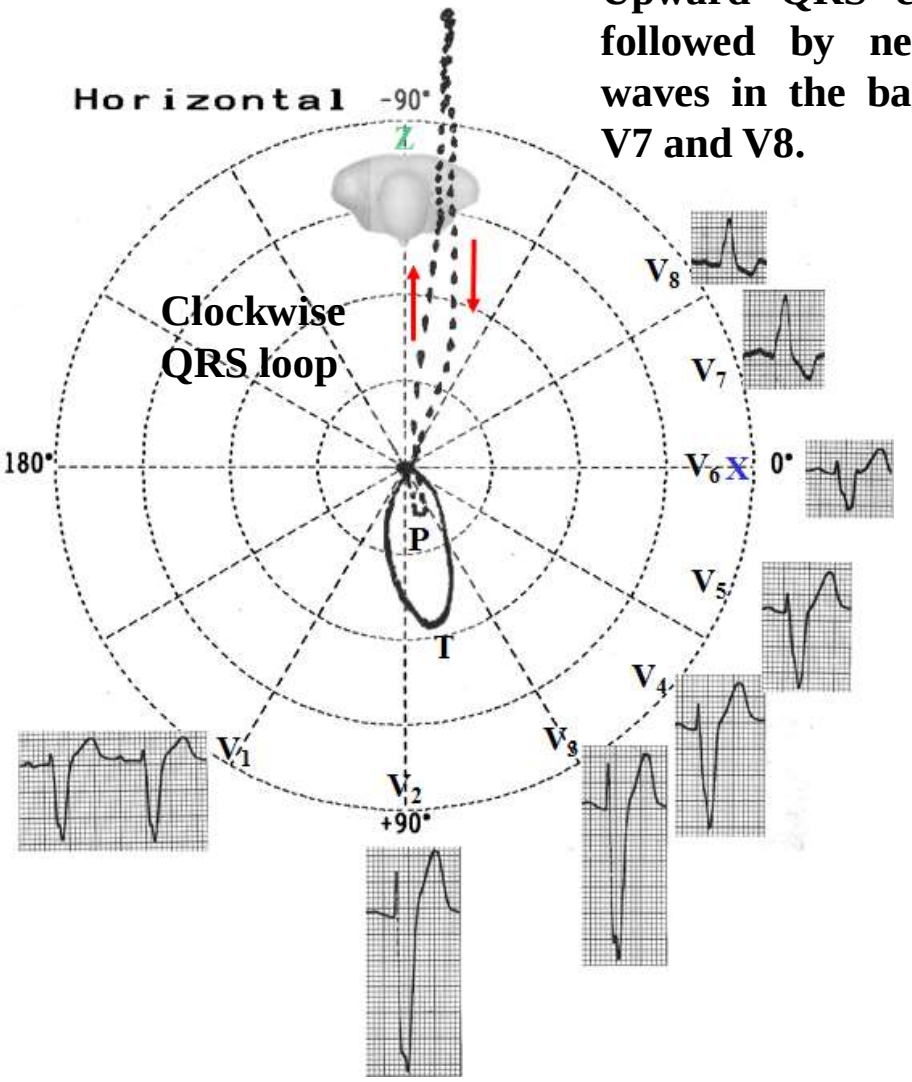
Atypical LBBB because rs in I and rS in aVL and rS from lead V1 through V6. The typical LBBB upward QRS is observed only in inferior and posterior leads (V7-V8)

ECG/VCG correlation in the frontal and horizontal planes



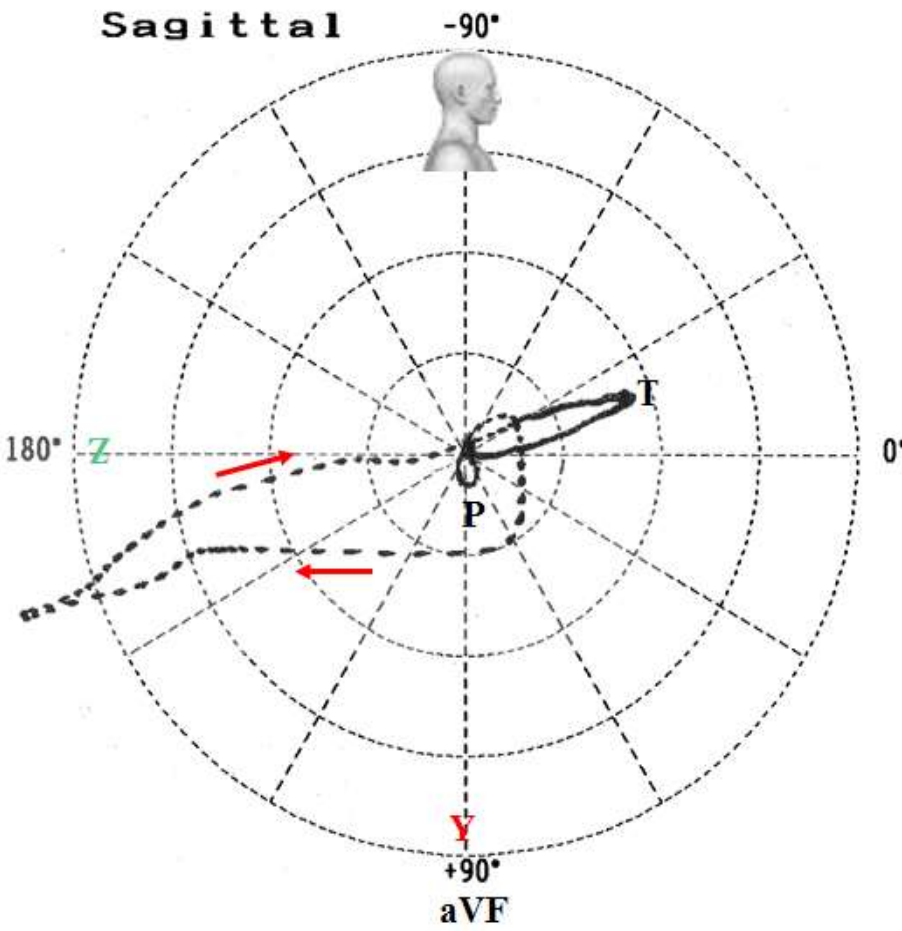
Right axis deviation. SÂQRS at +110º.
QRS loop with predominant CCW rotation with maximal QRS vector +74º.

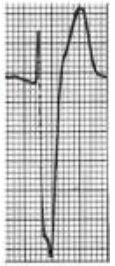
Upward QRS complexes followed by negative T waves in the back (leads V7 and V8.



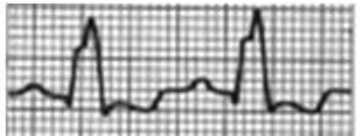
Negative QRS complexes from V1 to V6.

ECG/VCG correlation in the RSP



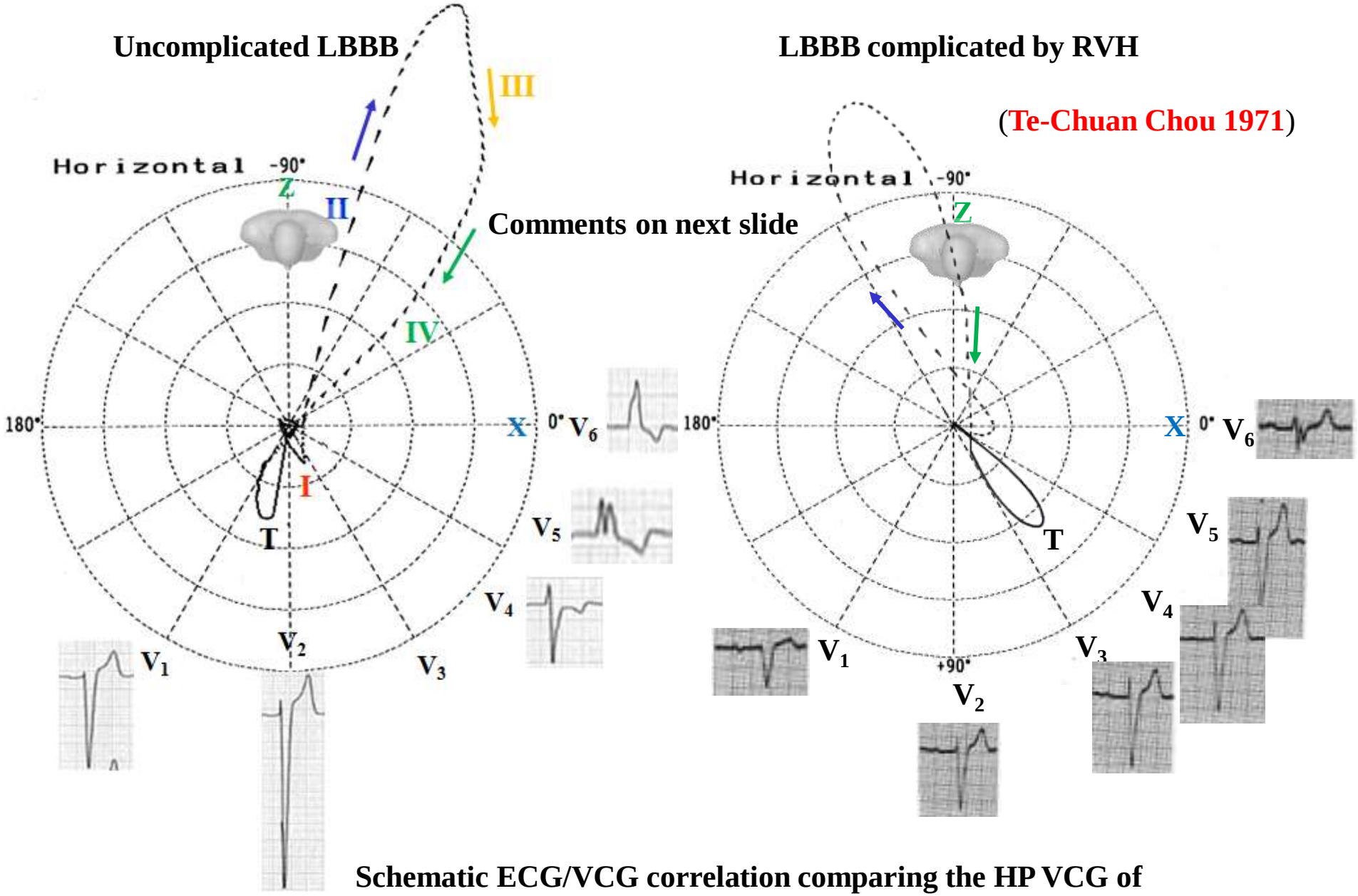


Negative QRS complex in V2 followed by positive T wave



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

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



Schematic ECG/VCG correlation comparing the HP 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. Clock-wise inscription of the major portion of the QRS loop in the HP;
6. Anterior and leftward orientation of the ST vector and T-loop.

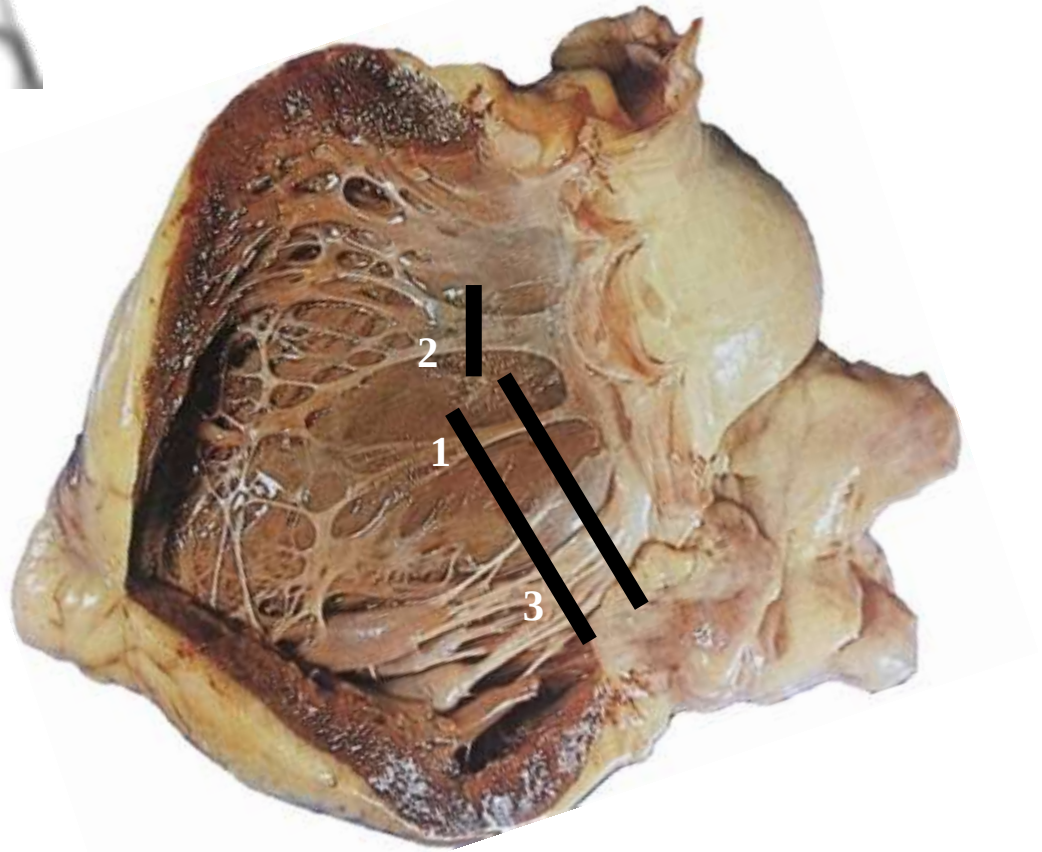
Final comments:

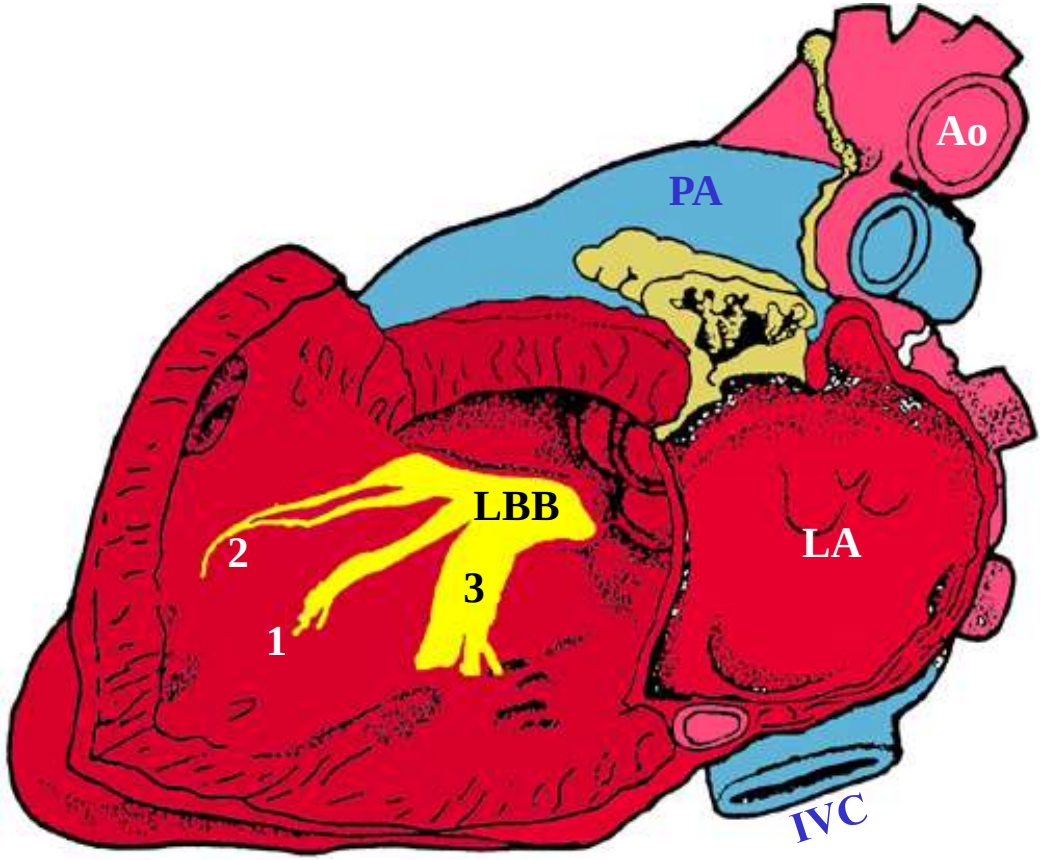
The changes in the HP VCG differed from the typical LBBB pattern only in the rightward displacement of the QRS loop and leftward orientation of the ST vector and T-loop.

	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)

2) Divisional LBBB with predominant left posterior fascicular block

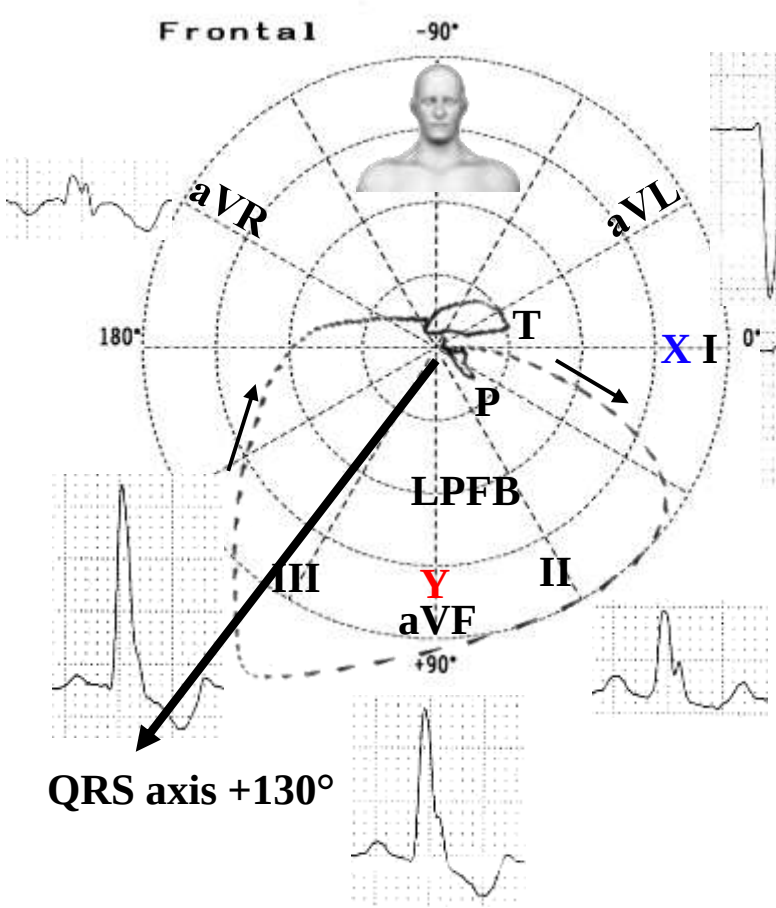
Left sagittal projection: divisional LBBB of a higher degree of blockage in the left posterior fascicle



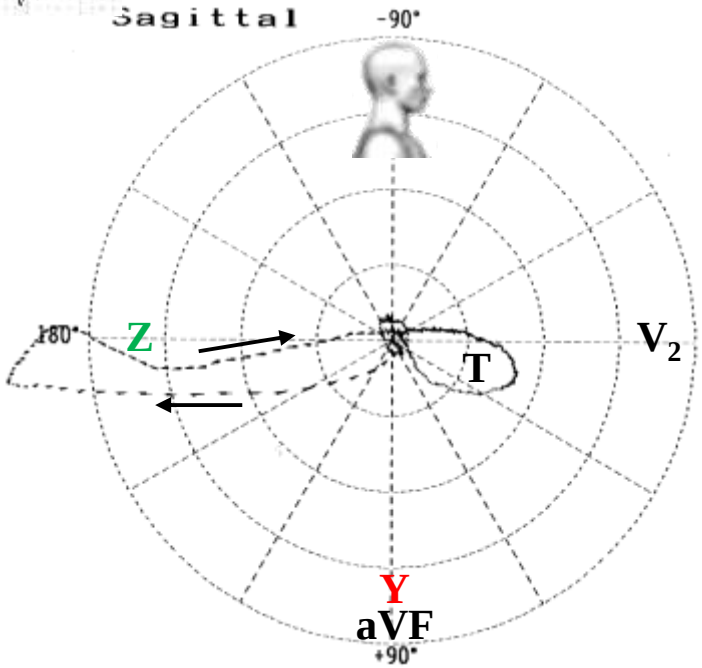


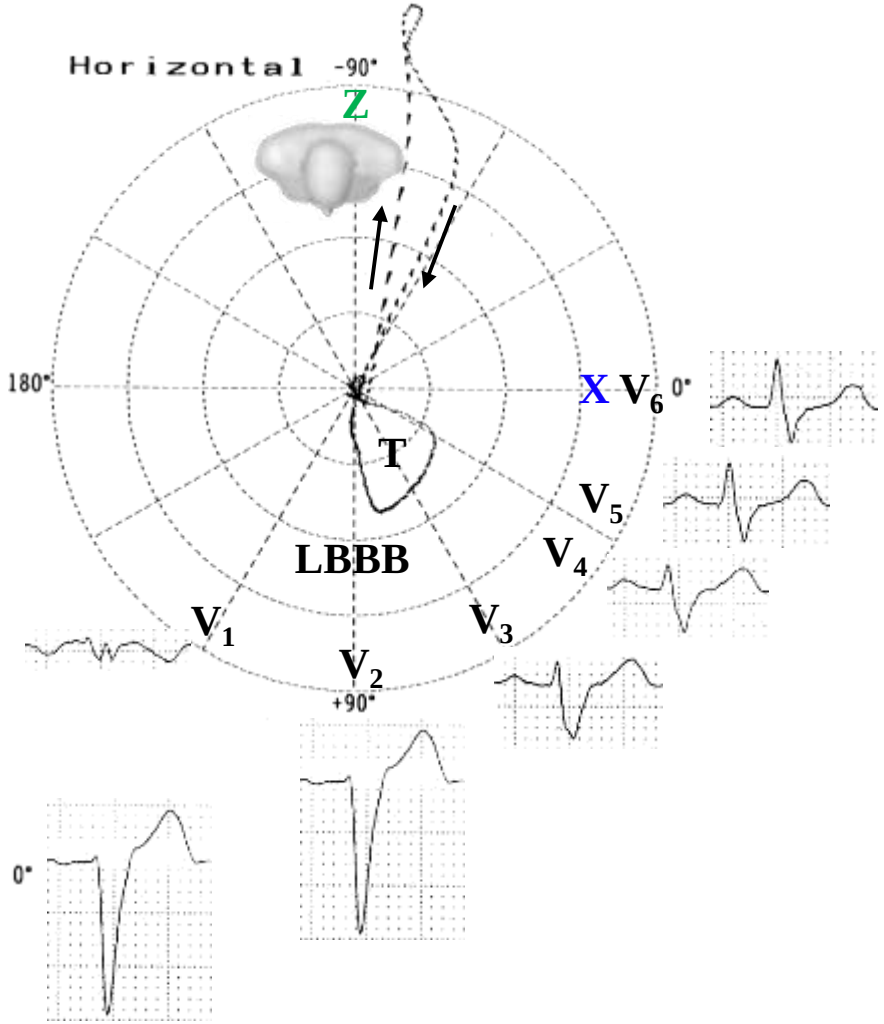
1) Left Septal Fascicle (LSF)
2) Left Anterior Fascicle (LAF)
3) Left Posterior Fascicle (LPF)

ECG/VCG correlation



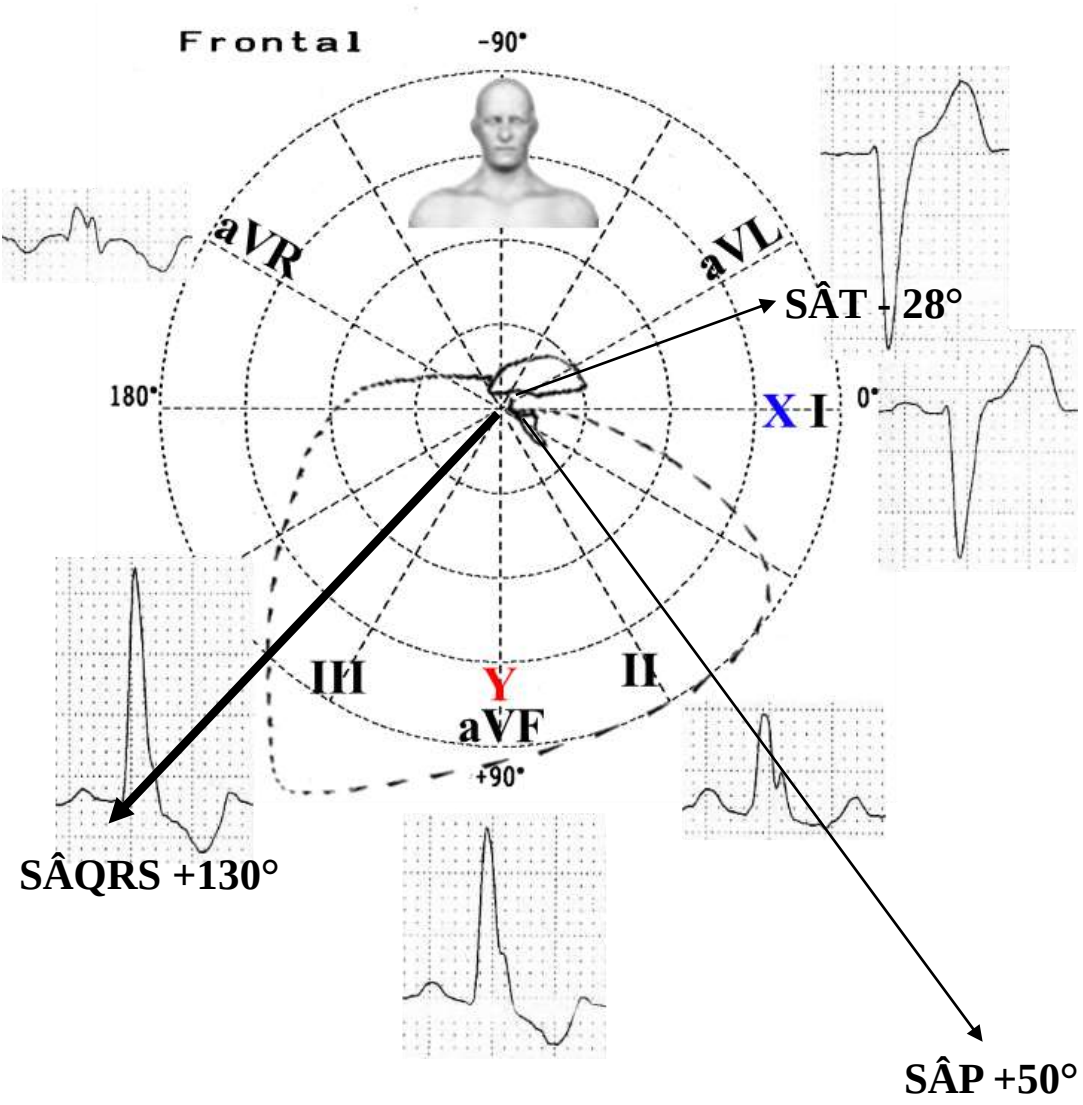
QRS axis +130°





FP: typical LPFB pattern.
HP and RSP: typical complete LBBB.

ECG/VCG correlation on Frontal Plane



ECG criteria for LPFB

1. SÂQRS on Frontal plane axis between +90° and ±180° in adults;
2. rS pattern in leads I and aVL
3. qR pattern in III, aVF and II: Q wave is always present in III and may be small or absent in II or aVF.
4. Notch in the descending limb of the R wave in III (middle-final notch);
5. RIII > RII: SÂQRS closer to +120º (III) than +60º (II), when closer to the latter, it would indicate an incomplete form of LPFB.
6. The q wave in III is always greater than the q wave in II and aVF. If there is association with inferior infarction, the Q wave > 40 ms.
7. When isolated the QRS duration <120 ms. When associate with RBBB or LBBB > 120ms.
8. Ventricular activation time, or R-wave peak time or intrinsicoid deflection (ID) in aVF≥35 ms.

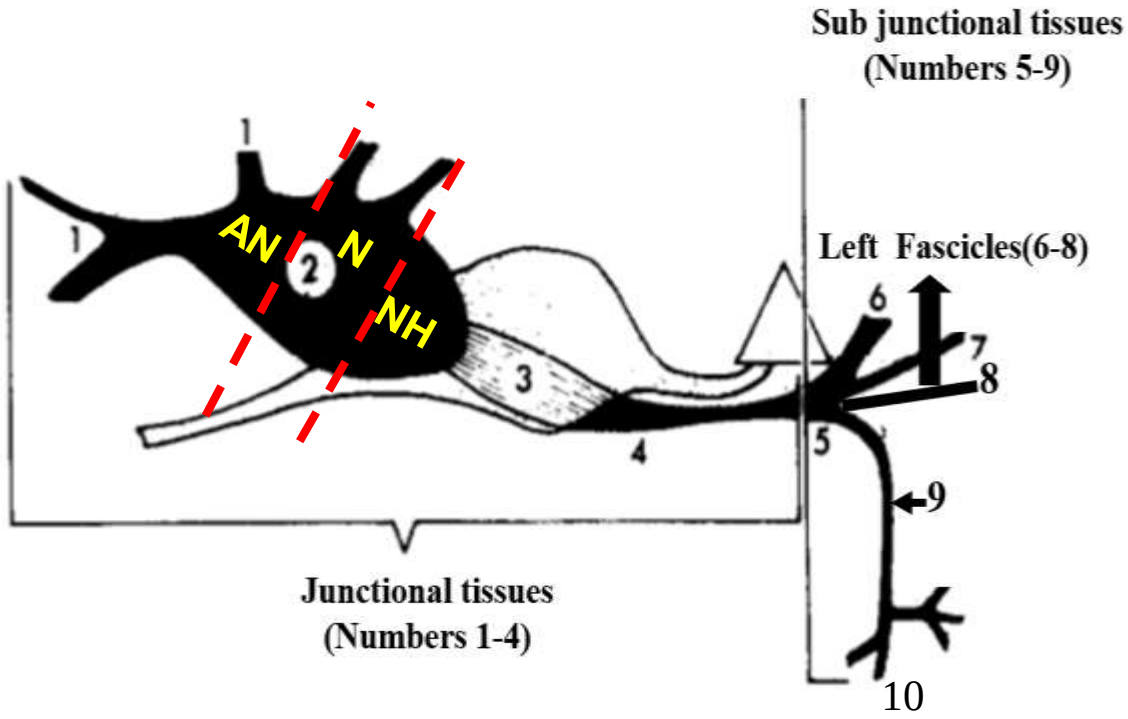
VCG criteria for LPFB

1. Vector of initial 10 to 20 ms heading above and to the left (near -45º) with possible delay (initial 10 to 25 ms).
2. Broad QRS loop, with clockwise rotation. Occasionally, it may be in “eight” with a counterclockwise terminal portion (10%).
3. Maximal vector near +110º (+80º to +140º).
4. Almost all the loop is located below the X line (0 to ±180°) in the inferior quadrants.
5. Afferent limb heading below and slightly to the left, and the efferent one to the right.
6. Middle-terminal portion of the QRS loop (vector of 90 ms to >120 ms) with delay. It may possibly reach the right superior quadrant.
7. QRS loop duration >120 ms consequence of complete LBBB.
8. Normal ST-T vectors in isolated LPFB: T loop with clockwise rotation, heading below and to the left. Complete LBBB: secondary alteration to ventricular repolarization.

Atrioventricular Conduction System

I) Junctional tissue

- The approaches fibers to the AV node 1
  - Superior AN
  - Middle N
  - Inferior or lower NH
- Penetrating portion of His bundle 3
- Nonpenetrating portion of His bundle 4



II Sub junctional tissue

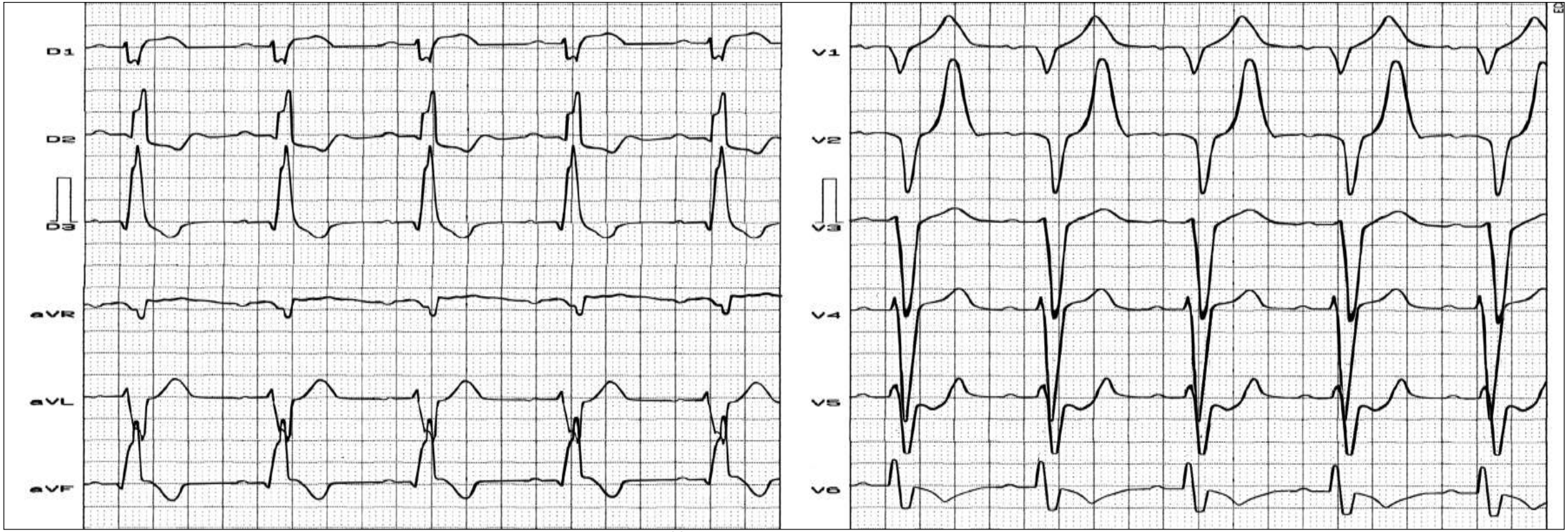
- Branching portion of His bundle 5

Bundle branches

- Main or stem Left Bundle Branch 5
  - Left Posterior Fascicle (LPF) 6
  - Left Anterior Fascicle (LAF) 7
  - Left Septal Fascicle (LSF) 8
- Right Bundle Branch (RBB) (septal) 9
- Right free wall RBB or divisional RBB 10

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.



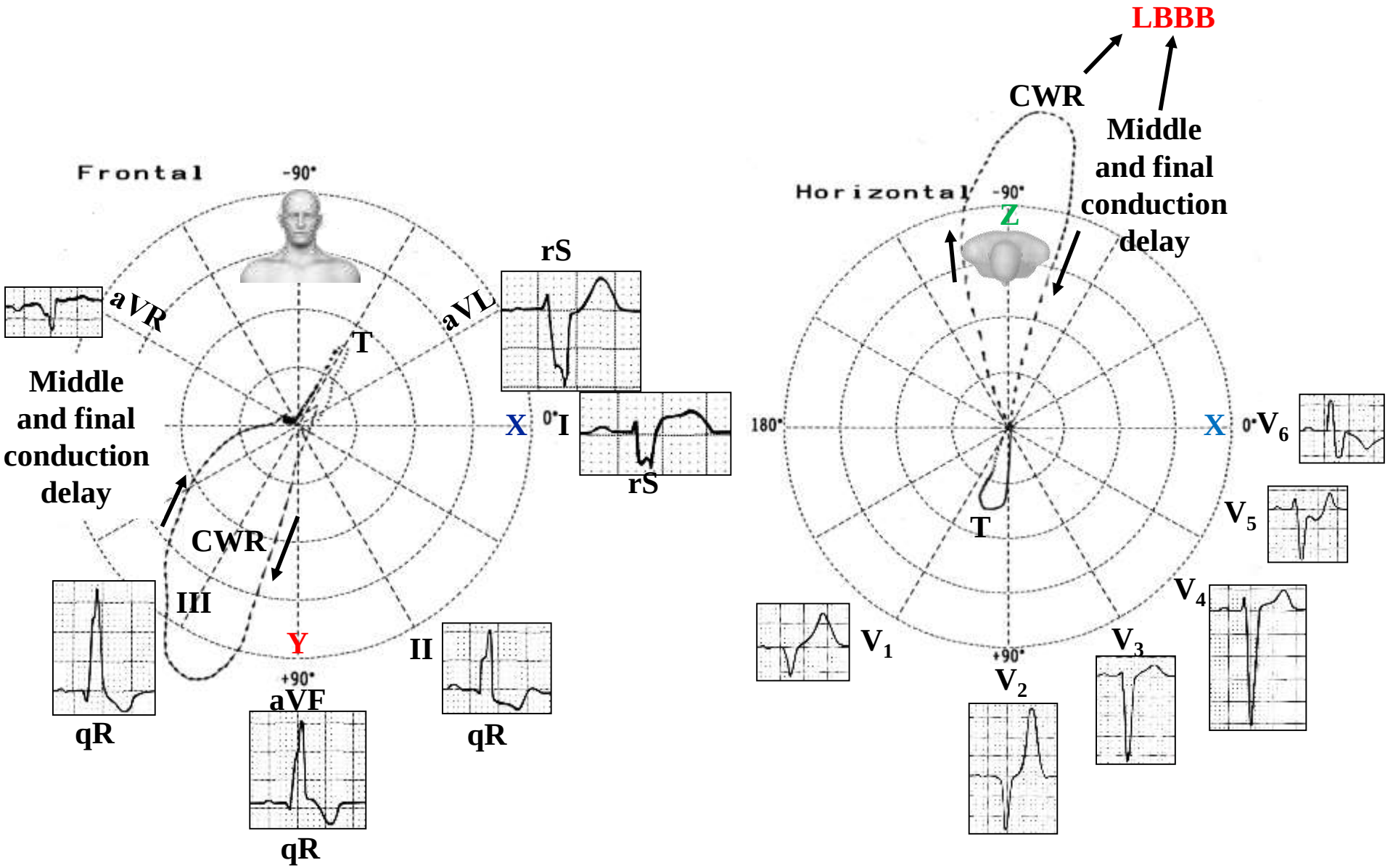
Clinical diagnosis: Hypertensive heart disease + aortic insufficiency by aortic cause.

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 $+115^{\circ}$.

Conclusion: 1) CLBBB; 2) LPFB (Left Posterior Fascicular Block).

ECG/VCG correlation on Frontal and Horizontal Plane



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

3) Left bundle-branch block with technical right-axis deviation. Technical problems/ Lead switches

The following ECG shows complete LBBB with marked right-axis deviation. The tracing was obtained during a routine clinic visit from an asymptomatic 64-year-old man with systemic hypertension and primary conducting system disease. All previous tracings had consistently shown LBBB with slight left-axis deviation. Because of the latter and the fact that the person obtaining the ECG was an insufficiently trained pulmonary technician, electrode misplacement was suspected. Fortunately, the electrodes were still attached to the patient. Surprisingly, the authors found that the technician had placed the left arm electrode on the chest between the V_2 and V_3 electrodes, so that 7 electrodes were on the precordium and none were on the left arm. Because of this arrangement, aVL displayed the rS morphology expected to be recorded by a unipolar precordial electrode reflecting the electrical activity of the previously mentioned anatomic site. This new aVL resulted in the mandatory change in morphology of aVR and aVF (and of leads I and III) to conform to the equation of the central terminal (aVR + aVL + aVF=0), thus explaining the right-axis deviation. Proper positioning of the electrodes resulted in the ECG shown in the next figure, which was similar to the previous ones.

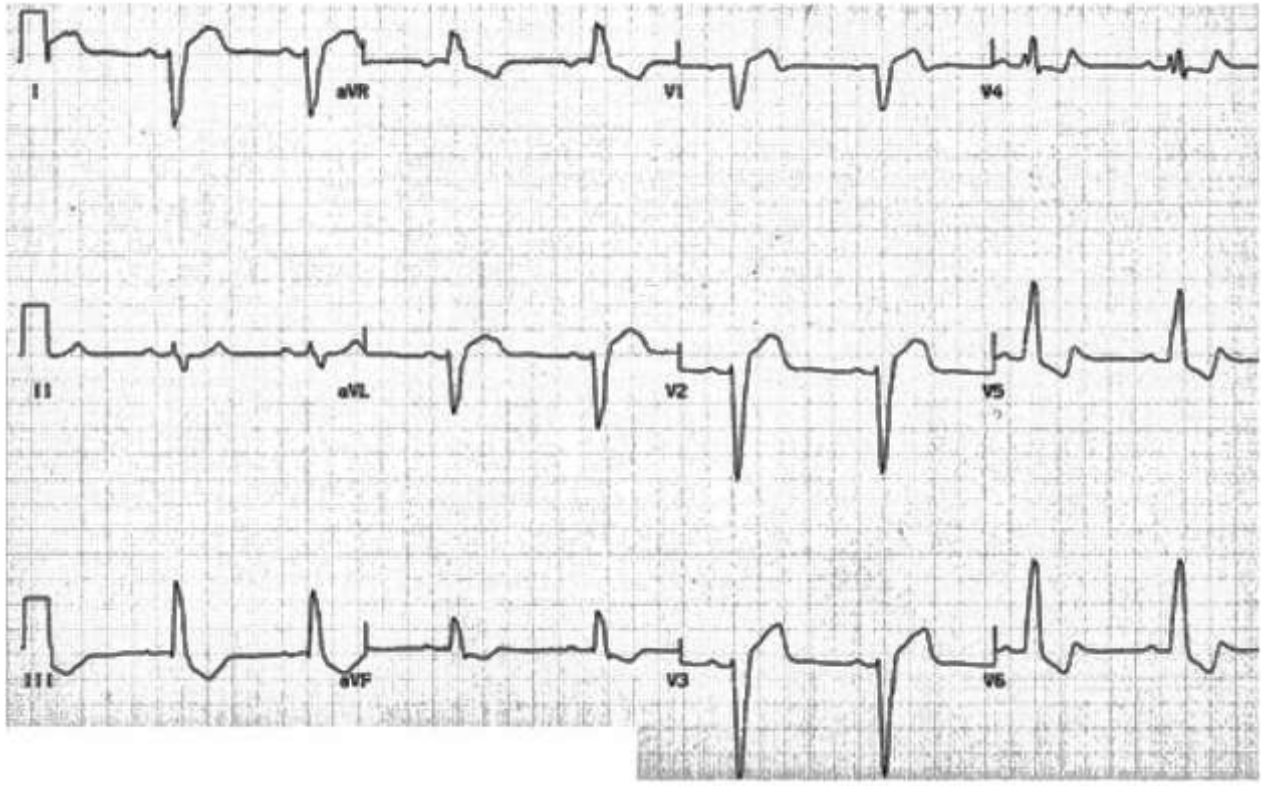


Figure 1. ECG from a patient with complete left bundle-branch block. This pattern resulted from (mis)placement of the left arm electrode in between V_2 and V_3, which resulted in 7 electrodes on the chest and none on the left arm(**Castellanos A 2002**).

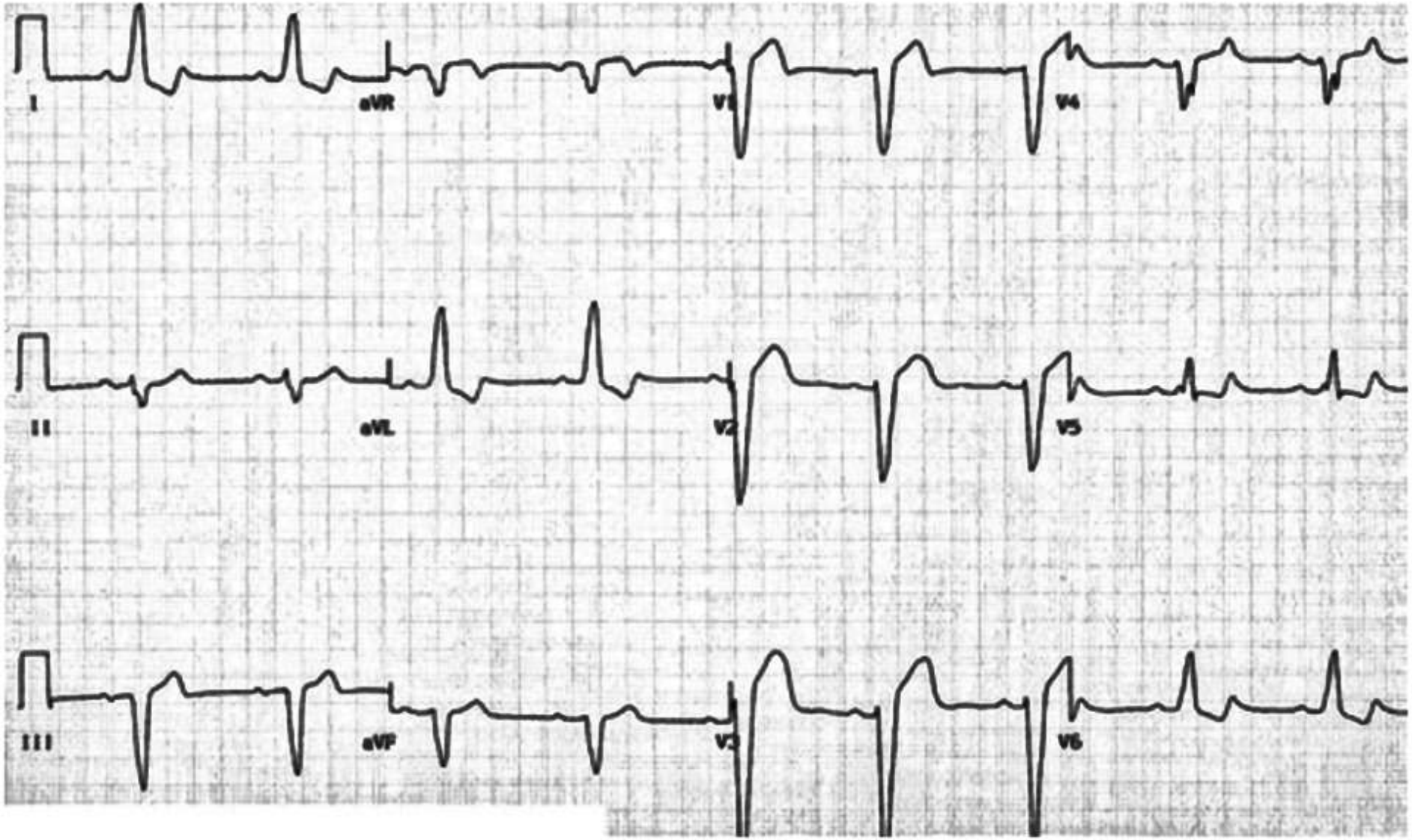


Figure 2. ECG from the same patient with proper electrode placemen In this case, it was not difficult to suspect electrode misplacement. The problem was determining which electrodes were involved. The attending and assistant physicians initially thought that the left arm and, possibly, the V_2 electrodes had been switched. When this happens, however, the morphology of the normal aVL (large R wave) should appear in the electrocardiographic position corresponding to V_2 and that of V_2 in aVL. This was subsequently corroborated by intentionally interchanging the latter electrodes (Figure 3).

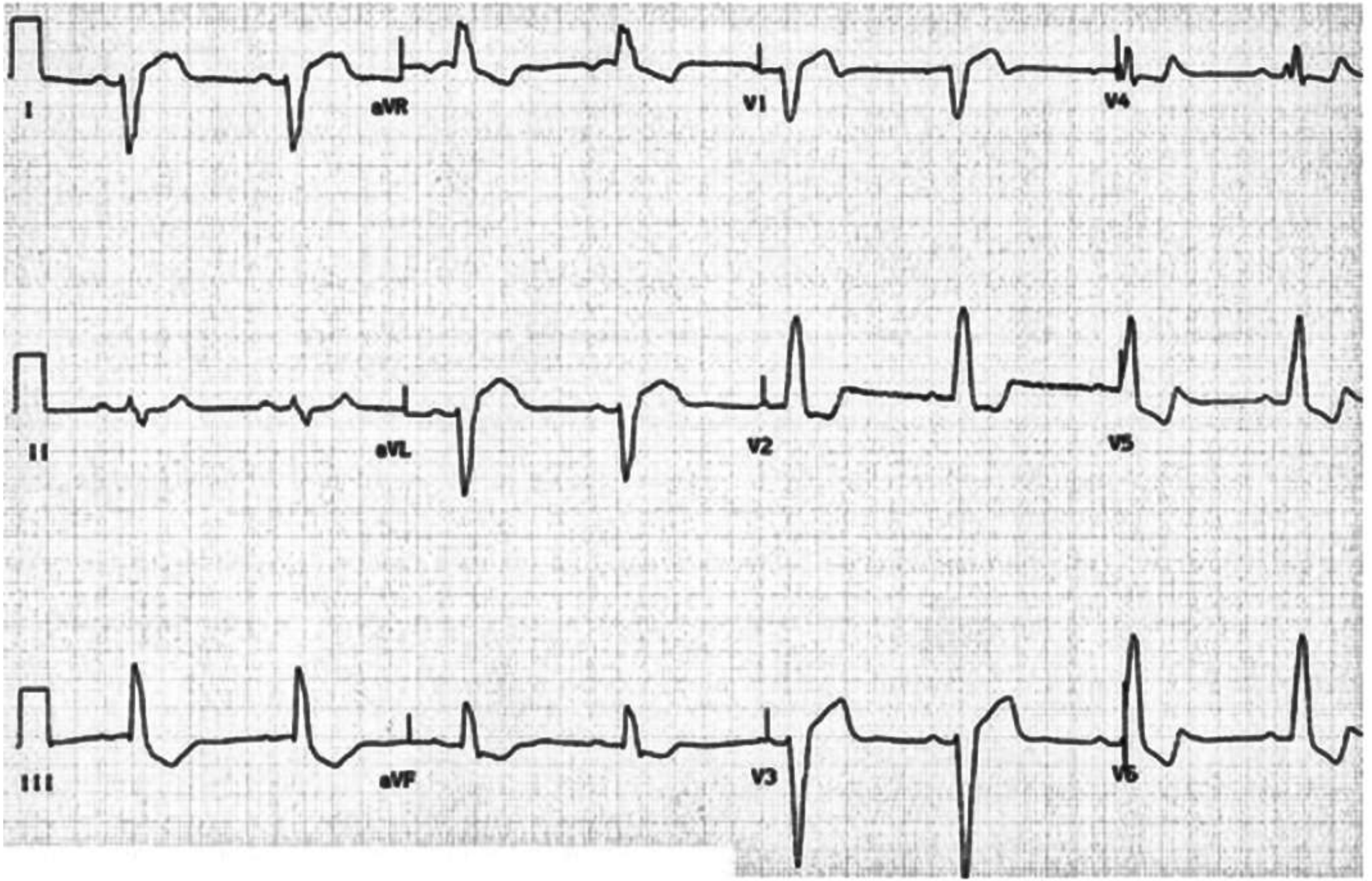
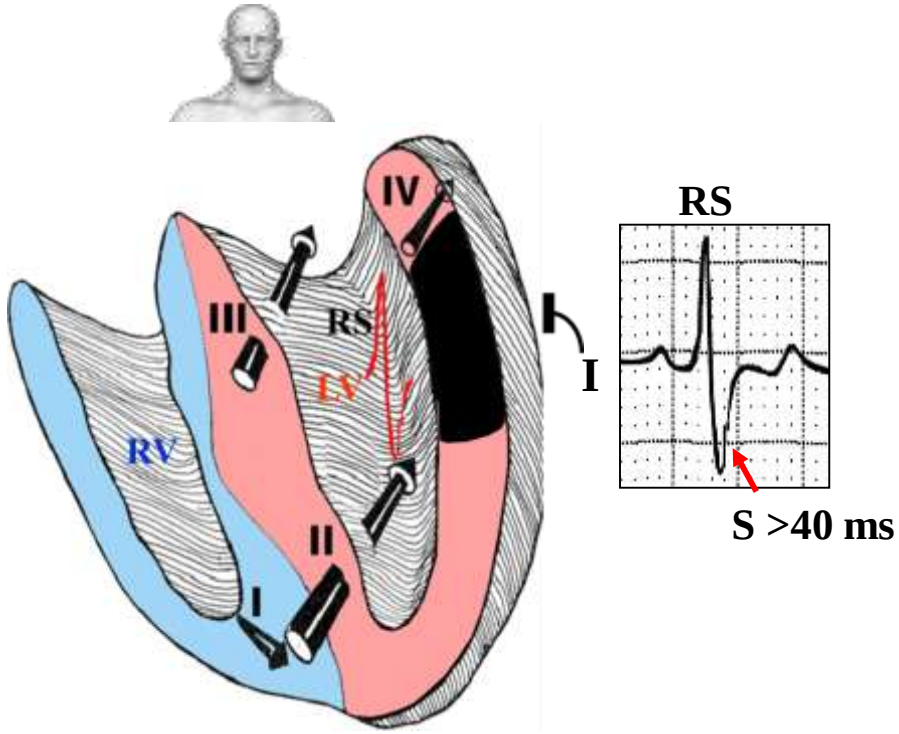
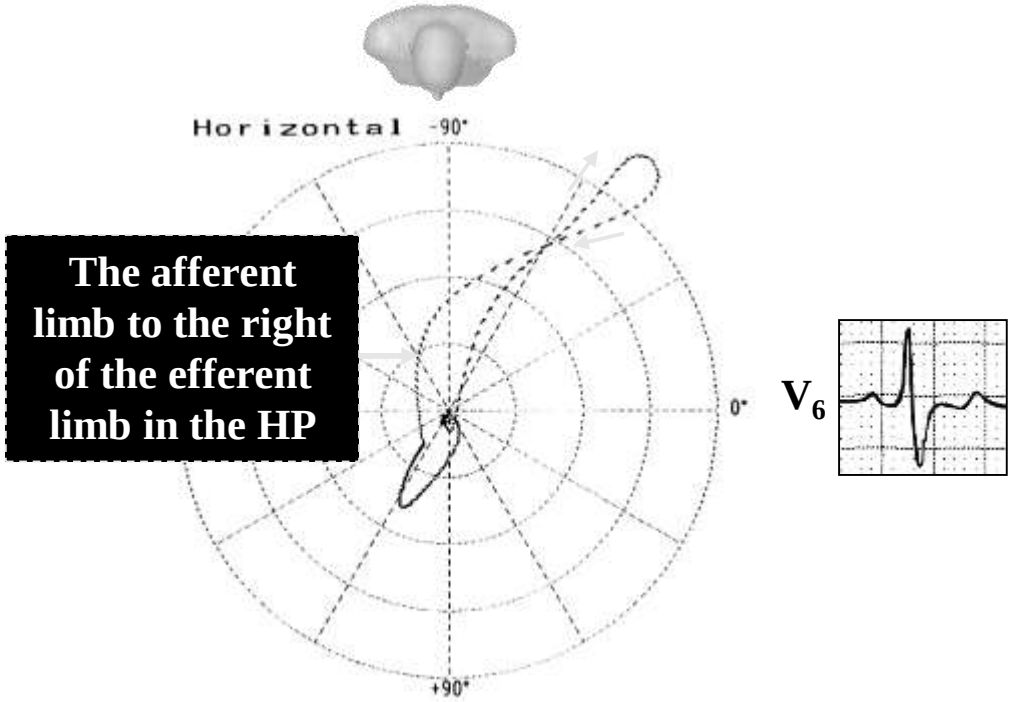


Figure 3. ECG obtained with the left arm and V_2 electrode interchanged.

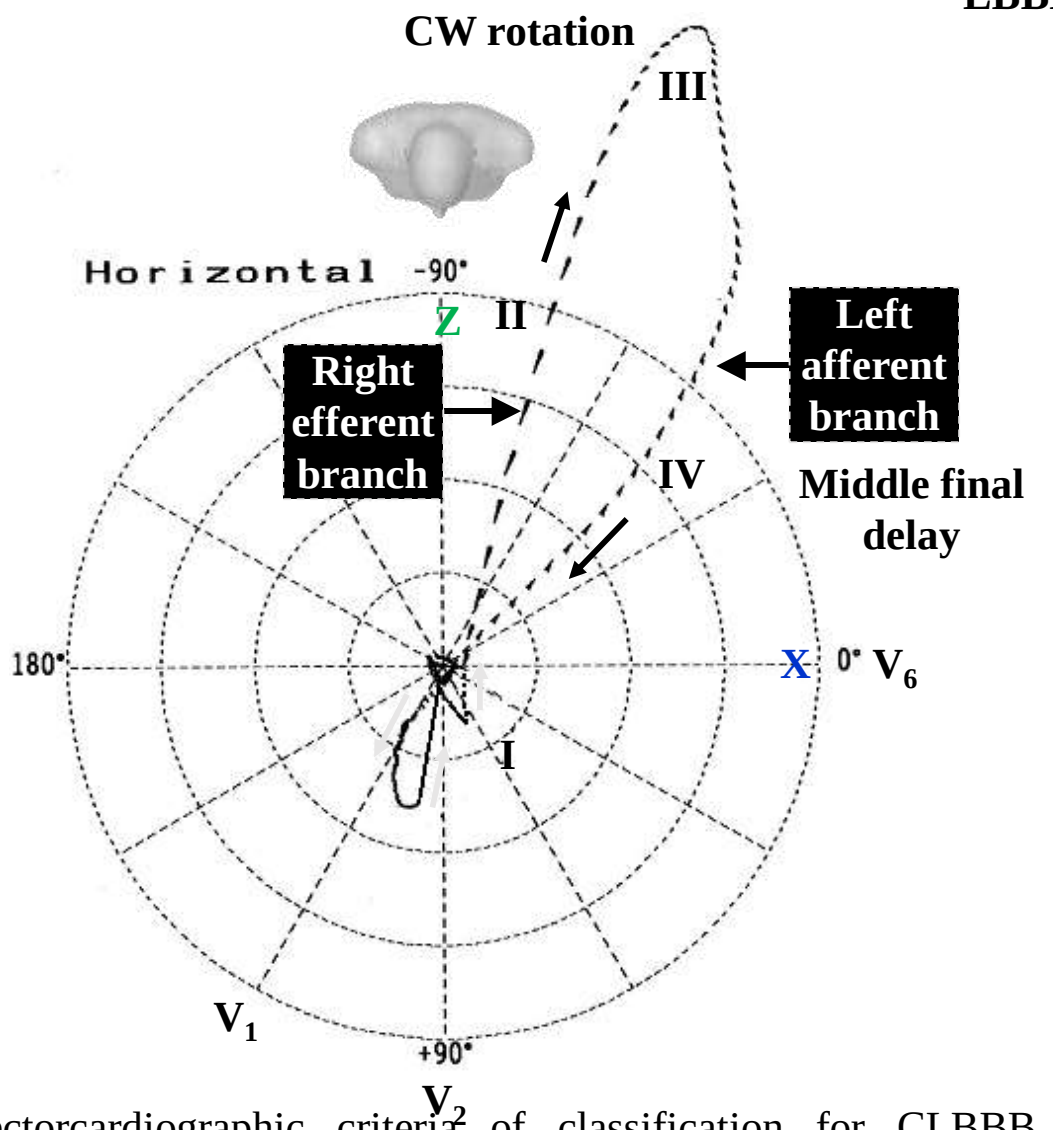
4) LBBB associated with left ventricular free wall myocardial infarction



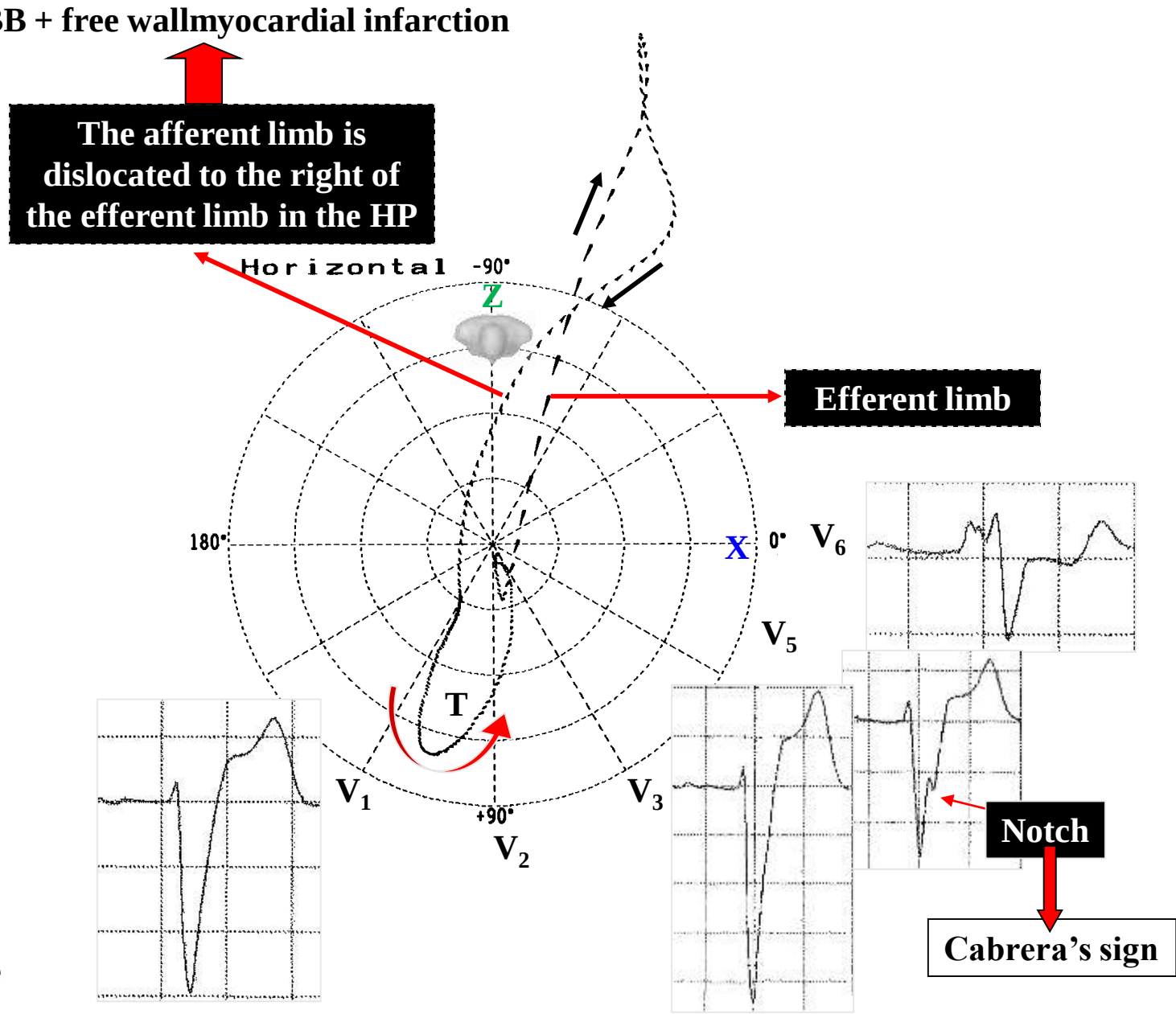


When electrocardiography was starting, Wilson postulated that the S wave of V_6 in the LBBB associated to lateral infarction was due to the sensing by the exploring electrode of V_6 of intracavitary potential of the LV (RS): it is called the “electric window” of Wilson.
Today we know that the afferent limb is dislocated to right of the efferent limb.

QRS loop in uncomplicated LBBB in the HP



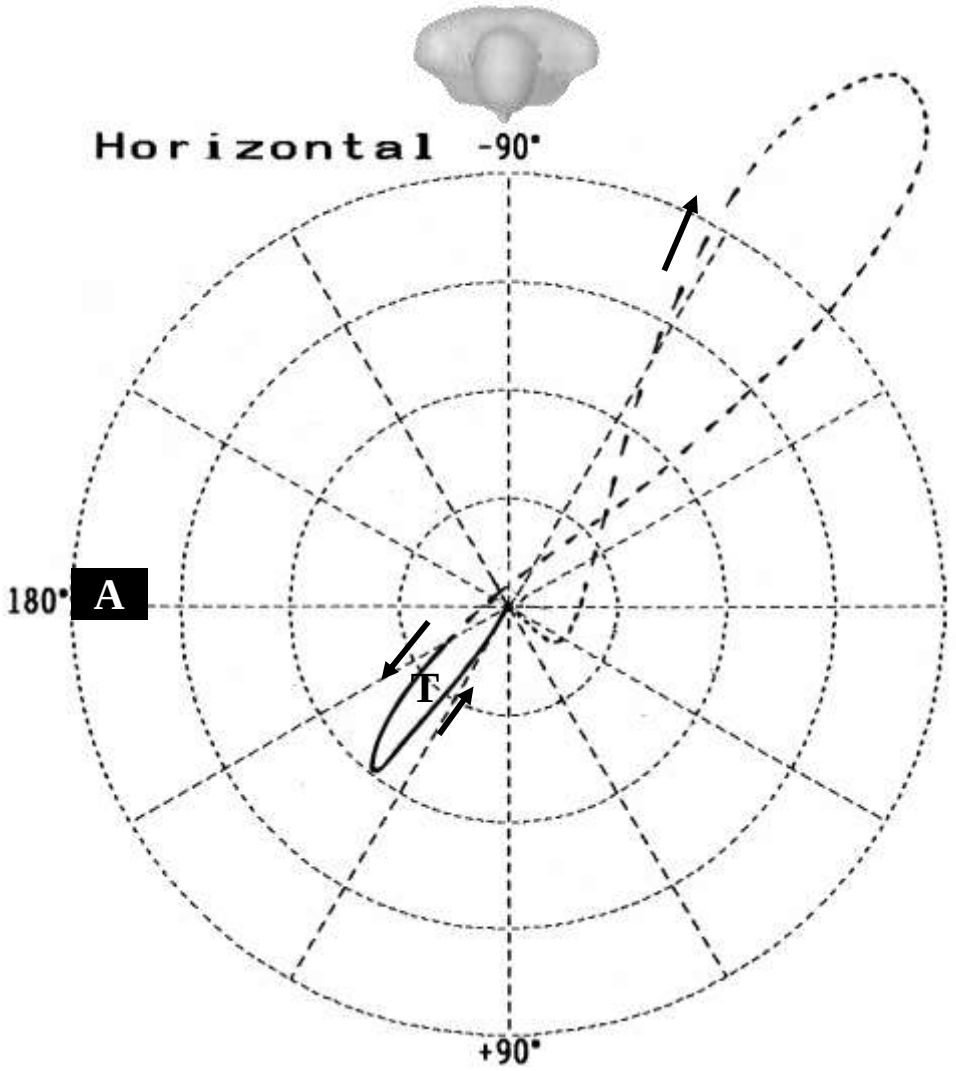
LBBB associated with LV free wall MI



Vectorcardiographic criteria of classification for CLBBB, highlighting clockwise rotation of QRS loop in the HP. The middle final delay is in the opposite location of repolarization (ST-T loop).

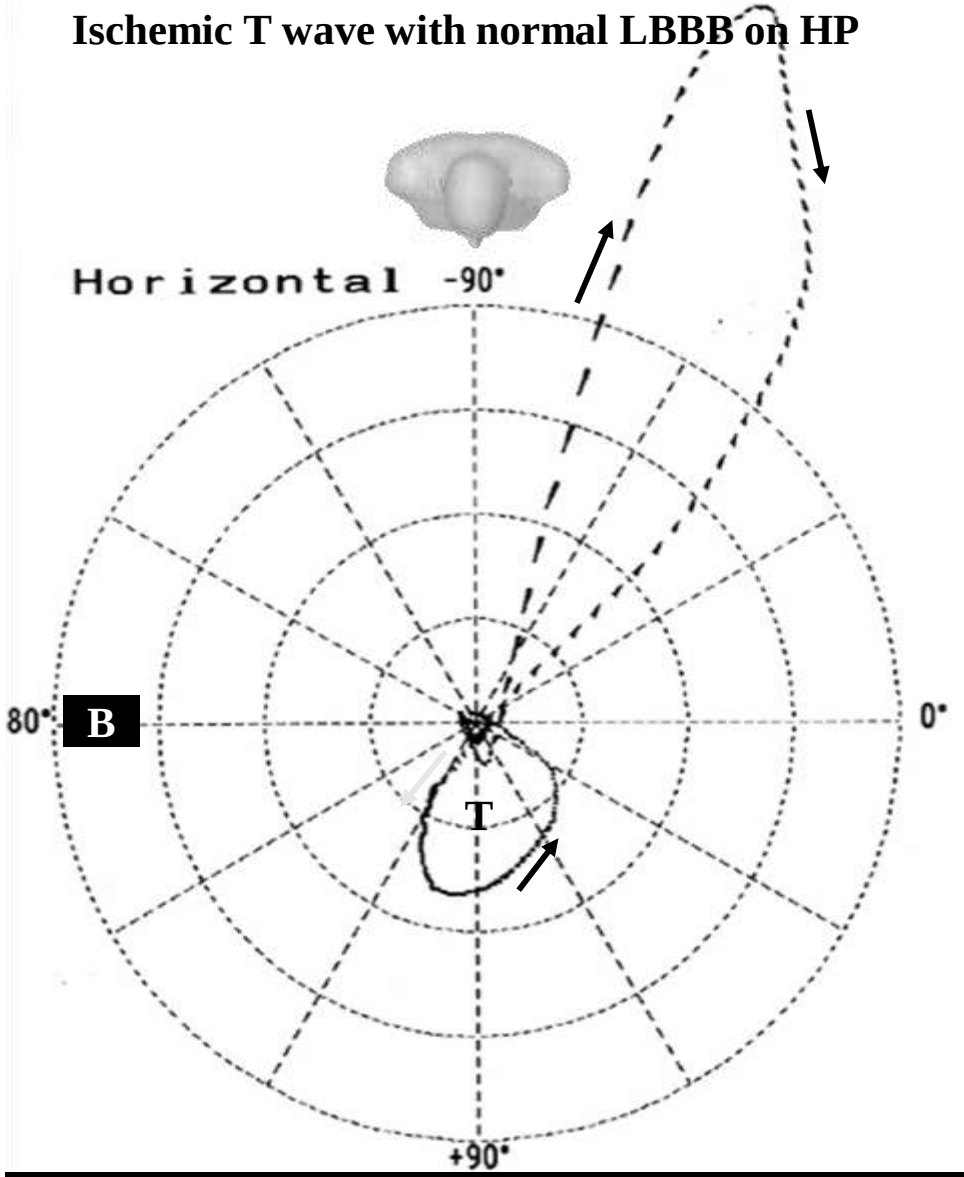
Normal (A) and ischemic (B) T loop in the HP (Kors 1998)

Normal VCG of LBBB in the HP



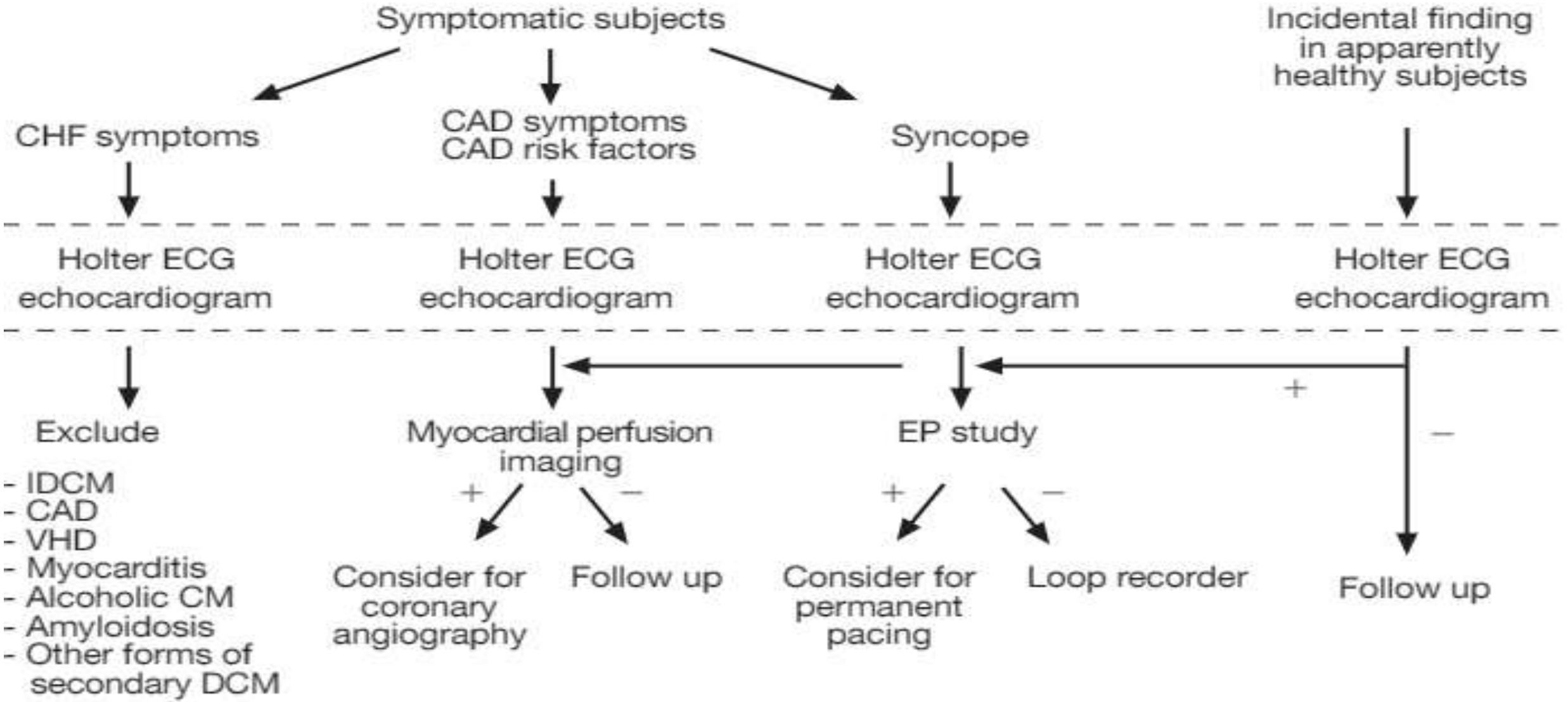
Secondary T loop is elongated or elliptical.

Ischemic T wave with normal LBBB on HP



The T loop is symmetrical in afferent and efferent limbs. Abnormal length/width (l/w) ratios of the T-loop. T waves with circular and bulgy morphology.

Left bundle branch block management



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 = electrophysiologic, IDCM = idiopathic dilated cardiomyopathy, VHD = valvular heart disease, CM = cardiomyopathy, DCM = dilated cardiomyopathy.

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Malbec: “the Argentinian adopted wine”
Commemoration by Adrian Baranchuk QRS malbec wine launch

Adrian Baranchuk´wine qRs Malbec

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Antiarrhythmic mechanisms of Malbec wine and resveratrol in isolated rat heart

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Bacchus God of wine