MARFAN SYNDROME WITH SEVERE AORTIC INSUFICIENCE ASSOCIATED WITH A RARE DORMOTROPIC INTRAVENTRICULAR CONDUCTION DEFECT

A SÍNDROME de MARFAN COM INSUFICIENCIA AÓRTICA SEVERA ASSOCIADA A TRANSTORNO DE CONDUÇÃO INTRAVENTRICULAR RARO

Andrés Ricardo Pérez Riera MD
Chief of Electro-Vectorcardiographic Sector - Cardiology Discipline-
ABC Faculty – ABC Foundation

Case Report

Interrogatory

Masculine, Caucasian, 37 years old, complete second degree level study, catholic, electrician, natural and resident in Manaus (capital of Amazonas. It is located in the middle of the Amazon forest.).

Guiding by de regional physician to specialized great center because "hereditary cardiac murmur that needs correction". He knew about his cardiac condition since 15 years ago when it was diagnosed the presence of cardiac murmur. He never gave importance because "I am totally asymptomatic".

Unknown family history (he is adopted).

Physical:

Inspection: Tall stature (192m/ 70Kg weight) A long, thin face; deep-set eyes; receding chin; and down-slanting eyes, myopia and ectopia lentis. The palate (roof) is highly arched and the teeth are very crowded.

Long extremities: Arm span greater than height. His arms are stretched to the side, the distance from finger tip to finger tip is greater than his height. Positive wrist sign. The thumb and little finger overlap when he grasp the other wrist. Positive thumb sign. He put the thumb on his hand and it extends beyond the palm. Arachnodactyly.

Reduced upper to lower segment ratio. (The length of his torso (from shoulders to legs) is shorter than the length of his legs.) Flat feet.

Protrusion acetabulae: Extra-deep hip sockets (the thigh bone meets the hip).

Very flexible joints.

Inspection:
The head bobs with each heart beat (positive de Musset sign[1]). The condition was named after the French poet Alfred Louis Charles de Musset-Pathay.
Negative Muller sign(absence systolic pulsation of the uvula.).
Thorax: Pectus carinatum and marked scoliosis.
Palpation: The apical impulse is diffuse and hyperdynamic and it is displaced laterally and inferiorly on 6th intercostals space on left axillary line.
Auscultation:
•Soft S1 and a single S2.
•A systolic ejection sound is best heard on aortic focus.
•A louder aortic diastolic murmur better heard at the third and fourth right sterna borders that on the left.
•A mid-diastolic or presystolic murmur best heard at the apex (Austin Flint's murmur[2]). An Austin low-pitched rumbling murmur.
Booming systolic and diastolic sounds are heard over the femoral arteries. (Traube sign or "pistol shot sounds"[3]).
Pulses: "water-hammer" or collapsing type with abrupt distension and quick collapse (Corrigan pulse). Quick rise of the radial pulse better detected by having the patient raise his arms over his head. Double intermittent murmur over the femoral arteries[4](Duroziez's Sign)
Blood pressure: 145/ 0 mm Hg(Korotkoff sounds persist to zero).

1. **Sapira JD. Quincke, de Musset, Duroziez, and Hill: some aortic regurgitations. South Med J. 1981 Apr; 74:459-467.**
2. **Landzberg JS, Pflugfelder PW, Cassidy MM, Schiller NB, Higgins CB, Cheitlin MD. Etiology of the Austin Flint murmur. J Am Coll Cardiol. 1992 Aug;20:408-413.**
3. **Boudoulas H, Triposkiadis F, Dervenagas S, VanFossen DB, Wooley CF. Mechanisms of pistol shot sounds in aortic regurgitation. Acta Cardiol. 1991;46:139-45.**
4. **Durozie PL. Du double souffle intermittent crural, comme signe de l'insuffissance aortique. Arch. Gen. de Med. Paris. 1861; 107: 417-443, 500-605.**

Clinical commentaries

In patients with severe chronic aortic regurgitation the LV gradually enlarges while the patient remains asymptomatic until the fourth or fifth decade of his life.

The Austin Flint murmur is named after the 19th century American physician Austin (American physician, born October 20, 1812, Petersham, Massachusets; died March 13, 1886, Brooklyn, New York.). It is a presystolic or late diastolic (mitral) heart murmur best heard at the apex of the heart. It is present in some cases of aortic insufficiency and is thought to be due to the vibration of the mitral valve caused by regurgitation of blood from the aorta into the heart before contraction of the ventricles[1;2].

Mechanism

Echocardiography, conventional and color flow Doppler ultrasound, and cine nuclear magnetic resonance (cine NMR) imaging suggest the murmur is caused by the aortic regurgitation jet abutting the left ventricular endocardium, resulting in the generation of a low-pitched diastolic rumbling[3].

1. **A. Flint: *On cardiac murmurs.* American Journal of the Medical Sciences, Philadelphia, 1862, 44: 29-54.Reprinted in Medical Classics, 1940, 4: 864-900.**
2. ***The mitral cardiac murmurs.*American Journal of the Medical Sciences, Philadelphia, 1886, 91: 27-44.**
3. **Landzberg JS, Pflugfelder PW, Cassidy MM, Schiller NB, Higgins CB, Cheitlin MD. Etiology of the Austin Flint murmur. J Am Coll Cardiol. 1992 Aug; 20: 408-413.**

Duroziez's Sign
Dr. Paul Louis Duroziez (1826-1897)

Pathophysiology

The double intermittent murmur over the femoral arteries as a sign of aortic insufficiency
Antegrade followed by retrograde vessel flow

Technique

Stethoscope bell used to compress femoral artery
Start off listening, and gradually increase pressure

Interpretation

Positive sign: 2 murmurs, 1 following another
Results in to-and-fro sound

Possible Etiologies

1. Aortic Regurgitation
2. Other conditions with high Pulse Pressure
3. Essential Hypertension
4. Thyrotoxicosis
5. Anemia

1. **Durozie PL. Du double souffle intermittent crural, comme signe de l'insuffissance aortique. Arch. Gen. de Med. Paris. 1861; 107: 417-443, 500-605.**

Dr. Paul Louis Duroziez (1826-1897)

Classical manuscript content wrote 149 years ago (1861) by Dr Paul L Duroziez

Former chief of the Clinic of the Faculty at the Charite Hospital (Service of Professor Bouillaud)

The femoral arteries, which are subjected to auscultation less frequently than the carotids, offer very valuable information; they are especially unique in their behavior, they are readily compressed and in this respect offer the same advantages as the radial arteries: they are larger than the carotid arteries and have the advantage of being more distant from the heart.

The femoral artery merits careful study. On compression of the femoral artery, a shock or thrill is felt and auscultation reveals a sound, similar to the sound of toc or a sound of unique blowing character, a simple intermittent blowing murmur. The entire femoral artery is capable of giving rise to this blowing murmur. The character of the murmur varies with changes in the blood, the size of the artery, the condition of the vessel wall, and the contractile force of the heart. After compressing the artery for some time and gradually releasing the pressure in a subject with chlorosis (anemia in young women), a continuous humming murmur will appear; at times a continuous, humming sound is audible, at other times a double murmur is audible.

The so-called intermittent double murmur which occurs in certain cases, is a different murmur and our study will concern it. The intermittent double murmur over the femoral arteries was described in aortic insufficiency; but no one, I believe, has given it the significance that it deserves. Everyone has mentioned the murmur occurring in arterial diastole (souffle de la diastole arterielle) which quite frequently occurs without compression of the artery; but very few authors mentioned the murmur occurring during systole. Very frequently it does not appear of its own accord, but must be produced and sought for. The first murmur results from the powerful contraction of the ventricle, but as the second murmur is produced by the systole of the arteries in the legs, a less powerful force, its production must be facilitated by compression of the artery.

In cases of uncomplicated aortic insufficiency, wherein the heart beats vigorously and the arteries pulsate and react forcefully, the double murmur is audible; when, contrarily, aortic insufficiency is complicated by a considerable degree of aortic or mitral stenosis, a not uncommon occurrence, the arteries are moderately distended with blood and thus the second murmur is difficult to hear. It must be carefully sought and even then it will not appear regularly; it will not be detected when weak pulsations are present. It appears or disappears in relationship to increased or decreased action of the heart. At times it can be heard over both femorals, at other times only over one; briefly, distention and recoil, adequate systole of the arteries, are required for its presence; a careful examination is indispensable.

The double murmur can be produced in two ways, by means of the stethoscope or by means of the hand. With the stethoscope pressure is exerted to completely compress the artery; at a certain moment the double murmur will appear; only when the second murmur can be readily produced is it possible to place the stethoscope on the artery without pressure and then gradually slight pressure can be exerted with the hand above and below the stethoscope. Pressure above will produce the first murmur, while pressure below will produce the second murmur; it is evident that the second murmur is produced by the arteries of the legs, which propel the blood backwards and in some manner empty the capillaries.

The double intermittent murmur is of interest not only from the standpoint of diagnosis. The reflux of blood explains some of the symptoms occurring in aortic insufficiency and explains the sudden death which is occasionally observed.

A great disturbance occurs in the circulation; the blood no longer circulates evenly, so to speak, but comes and goes into the arterial system and stagnates in the veins, which continuously try to empty themselves. In the presence of aortic insufficiency, the heart during its powerful diastole, aspirates the blood from the lungs through the pulmonary veins at the same time that it receives the blood from the capillaries; the right ventricle and the lungs are emptied of blood. The blood supply is poor; the patients are pale, die from anemia and syncope. They do not tolerate venesection well.

What a difference occurs with mitral stenosis! Here, on the contrary, the blood is stagnant, forced into the veins, into the right side of the heart and the lungs; the patients die from apoplexy and suffocation; venesection gives relief.

These are two conditions, in opposition to each other, and one may be considered as being beneficial to the other.

Auscultation is an important issue in this connection. We have distinguished by our observations the auscultatory phenomena and particularly those concerning the femoral arteries.

In all heart cases wherein the double intermittent murmur was audible over the femoral arteries, aortic insufficiency was found at autopsy.

CONCLUSIONS

1. The double intermittent murmur audible over the femoral arteries, described by many authors in aortic insufficiency, has to my knowledge never been given as a constant sign of this lesion.
2. Most commonly it is not present and it is necessary to produce it by compression.
3. In aortic insufficiency blood is first propelled from the left ventricle into the extremities, and, being repulsed by the peripheral arteries and drawn back by the left ventricle, flows from the extremities towards the heart.
4. The finger, compressing the artery about two centimeters above the stethoscope, produces the first murmur; two centimeters below, the second murmur.
5. The secondary murmurs which can be produced by lesions of the pericardium, by mitral stenosis, tricuspid stenosis, by pulmonary insufficiency, can be differentiated from the murmur of aortic insufficiency with the help of the double murmur over the femoral arteries, which exists only in the latter condition.

6. If aortic insufficiency is complicated by one or more of the lesions mentioned, and if the diagnosis is rendered difficult by these complications, the phenomena in the femoral arteries will help or even establish the diagnosis.

7. The femoral phenomena less clearly differentiate aortic valvular lesions and lesions of the aorta. The double murmur may appear in certain aneurysms without insufficiency being demonstrable after death.

8. The temporary insufficiency can be demonstrated by the evanescent intermittent double murmur.

9. A continuous murmur can originate in the arteries; this, however, is never audible in aortic insufficiency with its constant intermittent double murmur over the femoral arteries.

10. The double intermittent femoral murmur occurs in typhoid fever, chlorosis, lead intoxication, but only temporarily; it is soon replaced by continuous murmurs.

Babu et al[1] purpose to compare current textbook content with the peer-reviewed literature on the eponymous signs of aortic regurgitation and to assess the role of these signs in clinical practice.
DATA SOURCES: 11 textbooks, MEDLINE (1966 through October 2002), and bibliographies of textbooks and relevant papers.
STUDY SELECTION: English-language reports that were related to the properties of a sign on physical examination, incorporated more than 10 adults, and did not involve prosthetic heart valves or acute aortic regurgitation.
DATA EXTRACTION: Three investigators independently analyzed relevant textbook extracts and 27 reports, using predetermined qualitative review criteria. Data relating to diagnostic accuracy and properties of the index test were also extracted.
12 eponymous signs were described as having varying degrees of importance by textbook authors. Only the Austin Flint murmur, the Corrigan pulse, the Duroziez sign, and the Hill sign* had sufficient original literature for detailed review. Most reports were low quality, with varying sensitivities for all signs. Except for the Hill sign, specificity tended to be poor. Evidence for the Hill sign also suggested a correlation between the popliteal-brachial gradient and aortic regurgitation severity.
The authors concluded that prominent textbook support of the eponymous signs of aortic regurgitation is not matched by the literature. Clinicians and educators should update and improve the evidence for these signs to ensure their relevance in current medical practice.
***Hill sign:** An indication of aortic insufficiency in which systolic blood pressure is higher in the legs than in the arms.

1. **Babu AN, Kymes SM, Carpenter Fryer SM. Eponyms and the diagnosis of aortic regurgitation: what says the evidence?Ann Intern Med. 2003 May 6;138:736-742.**

Austin Flint Biography

Austin Flint, a pioneer of heart research in the U.S., was one of the most eminent of 19th century physicians. Flint made several important contributions to the knowledge of diseases of the heart and the respiratorial system. He is also said to have coined the term broncho-vesicular breathing.

Austin Flint studied at the Harvard University, where he was influenced by the teacher, James Jackson (1777-1868), who was a follower of René Théophile Hyacinthe Laënnec (1781-1826). He graduated in 1832. He first practiced in Boston, but moved to Buffalo, New York as a practitioner. He achieved a great reputation and was one of the founders of the Buffalo Medical College, to which he was appointed professor. In 1861 he became professor of theoretical and practical medicine at the school of medicine at the Bellevue Hospital, New York, later at the medical school of the Long Island Hospital in Brooklyn, where he died in 1886 as one of the most distinguished physicians of North America.

From 1861 to 1886 Austin Flint was professor of medicine at the Bellevue Hospital Medical College, New York City, and president of the American Medical Association from 1883 to 1883. In these capacities, as well as a researcher and physicians he had a great influence on the early course of medicine in the Unites States. A proponent of improved European diagnostic methods, he popularized the binaural stethoscope in the U.S. His Treatise on the Principles and Practice of Medicine (1866) is recognized as a medical classic.

His son Austin (1836-1915), a prominent physiologist, made important studies of liver function and wrote the popular A textbook of Human Physiology (1876). He was professor of physiology at the Medical College in New York.

Austin Flint was a prolific writer, and a collaborator in the American Cyclopedia, and 1872 president of the Academy of Medicine in New York.

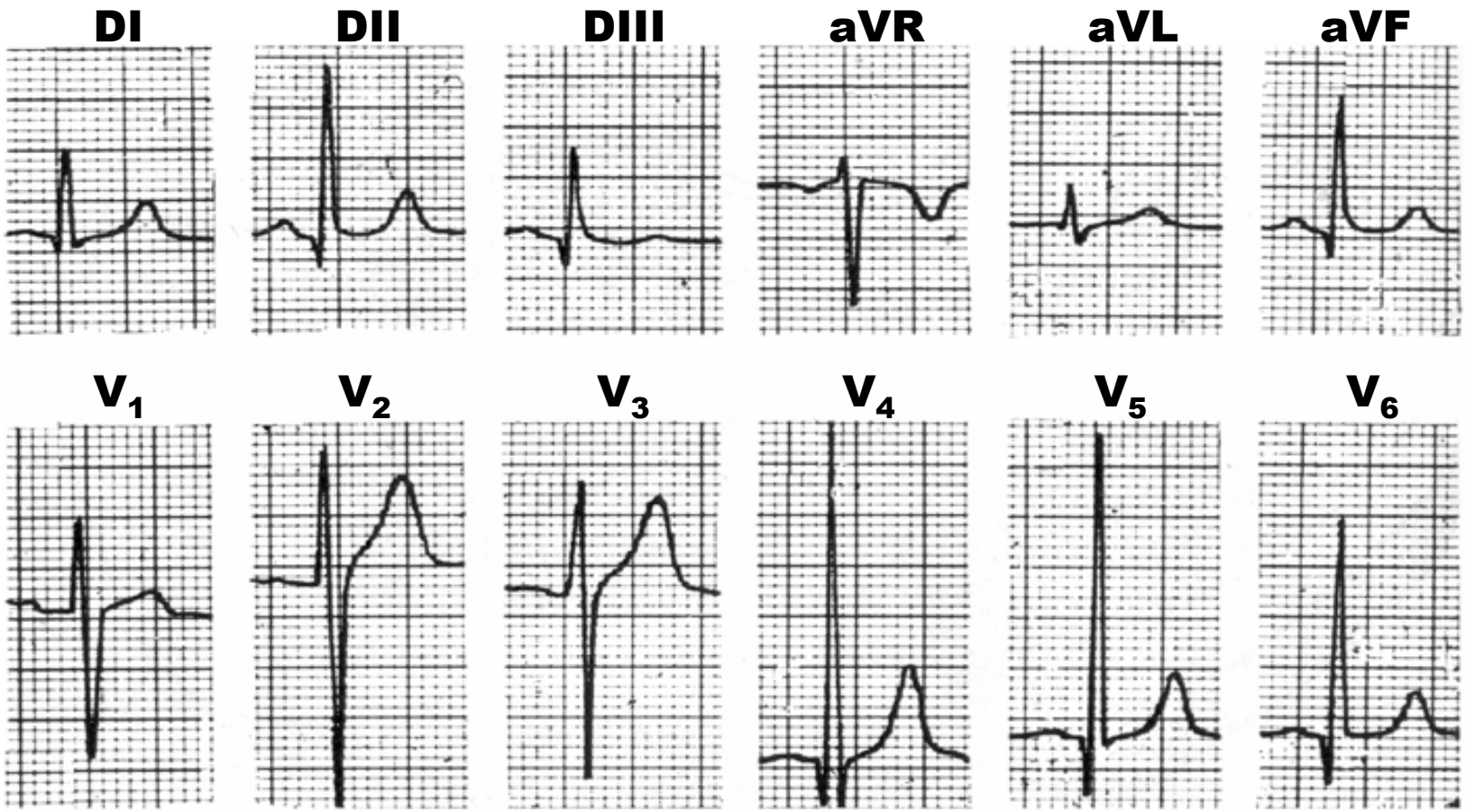
About the Corrigan pulse discovery

The valves of the heart and their diseases have generated a great deal of interest since ancient times. The early observations regarding valvular deformities were confined to the morbid changes with no reference to the hemodynamic significance.
The classic clinical signs of aortic valve insufficiency indicating hemodynamic disturbances were described later[1].
The classic bounding pulse of aortic regurgitation was described by several authors before Corrigan made his comprehensive study of the disorder in 1832[2;3].
The case report of Thomas Cuming[4], was published 10 years earlier Corrigan. This Dublin practitioner not only presented signs and symptoms of aortic regurgitation proved at autopsy, but also attempted with considerable success to relate the signs to circulatory dynamics.

1. **Mehta NJ, Khan IA. Original descriptions of the classic signs of aortic valve insufficiency. J Emerg Med. 2003 Jan; 24: 69-72.**
2. **Kligfield P. An annotation on the pulse in aortic regurgitation: Thomas Cuming, 1822. Am J Cardiol. 1979 Aug; 44: 370-371.**
3. **Vaslef SN, Roberts WC. Early descriptions of aortic regurgitation. Am Heart J. 1993 May;125:1475-1483.**
4. **Adams CW. Aortic insufficiency; "original" descriptions. Am J Cardiol. 1969 Nov;24:731-733.**

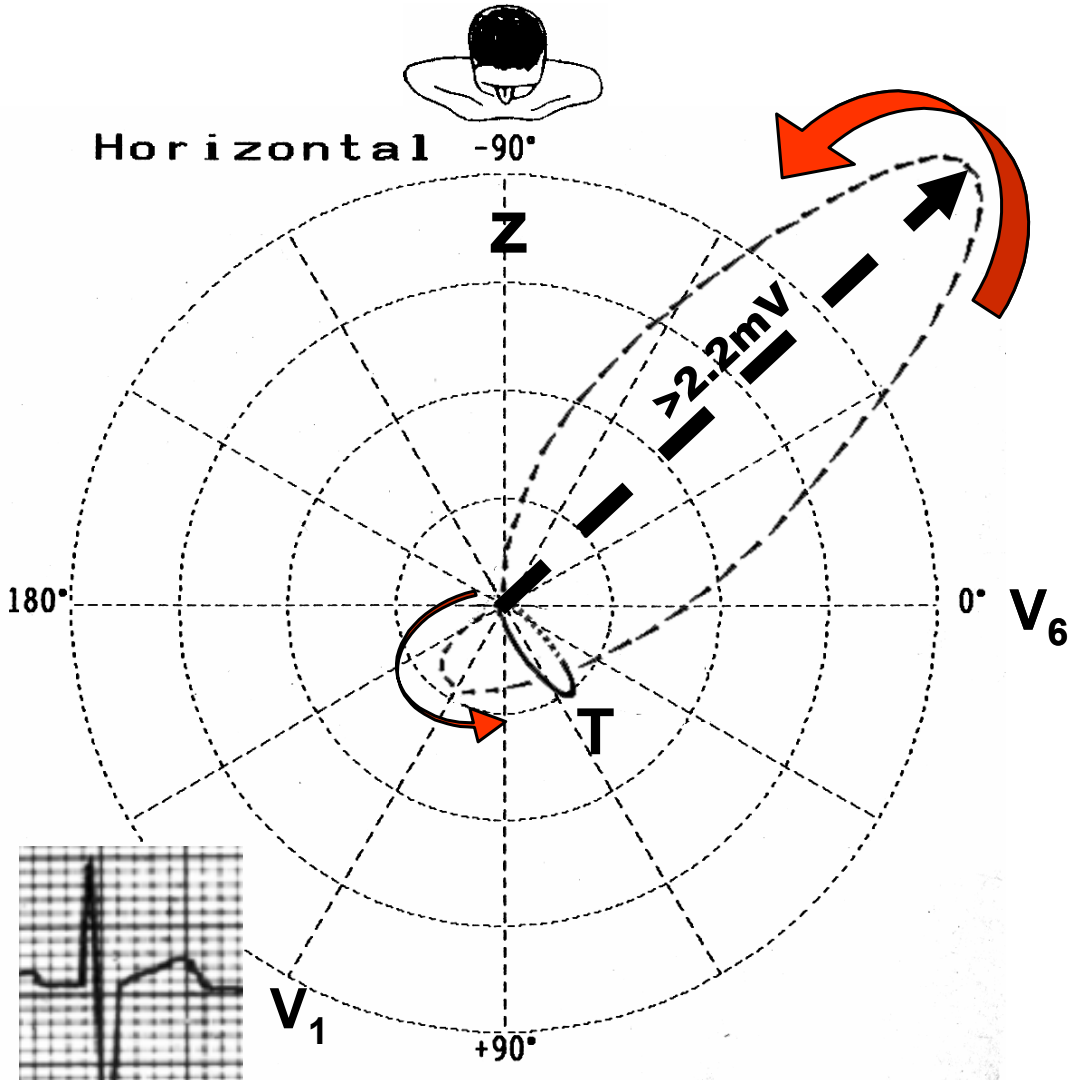
Name: ASP **Gender:** Male **Age:** 27 y.o. **Ethnic Group:** Caucasian
Weight: 70 Kg **Height:** 1,92 m **Biotype:** Ectomorph **Date:** 02/07/2000.

ECG performed approximately ten years ago



ECG diagnosis: LVH with typical LV volume overload or diastolic pattern characterized by increased initial forces because magnified first septal vector. Prominent R wave in V_1 and V_2, profound q waves in inferior leads and from V_4 to V_6.
The ST concave to the top and minimally elevated in V_5 and V_6. T waves matching QRS
Clinical Diagnosis: Aortic insufficiency secondary to Marfan Syndrome.

LV volume overload, Diastolic Pattern LV Enlargement or Eccentric LV overload



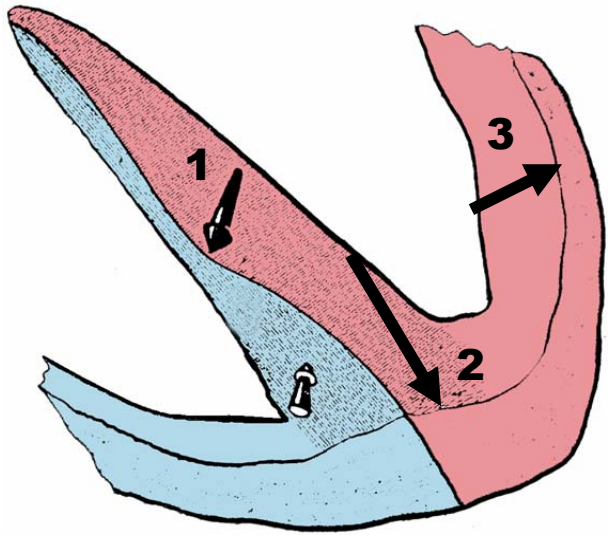
DISCRETE INCREASE OF QRS COMPLEX DURATION

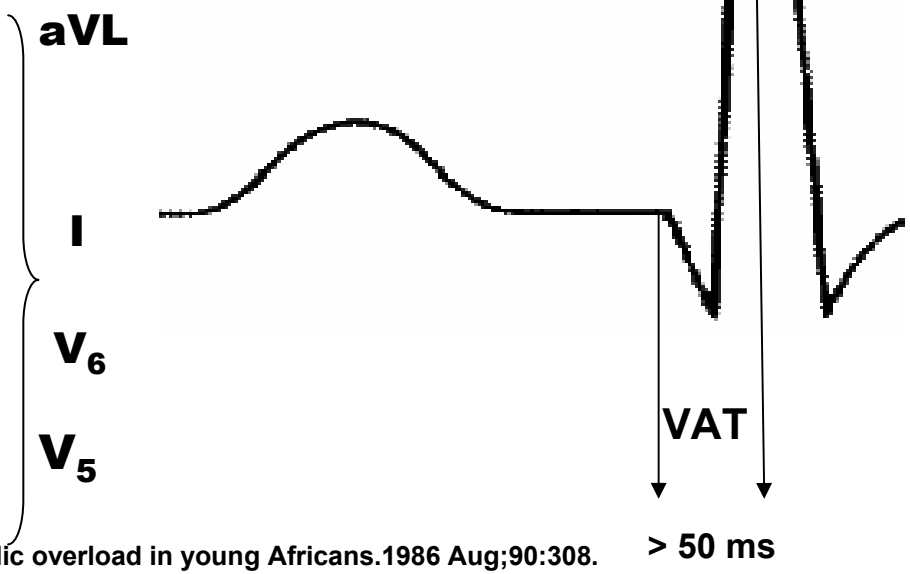
Left Ventricular Diastolic Overload, Eccentric or Volumetric Hypertrohy

Causes:

- **Aortic insufficiency (Ao. I.) The present case report.**
- **Mitral Valve Insufficiency (MVI)**
- **Patent Ductus Arteriosus (PDA)**
- **Ventricular Septal Defect (VSD), hemodynamic group I**
- **Anemia.**

Criteria based on the discrete increase in QRS complex duration at the expense of a delay in the time of appearance of R wave apex: “R peak time” in the leads that are opposite to the LV, initial time of intrinsic deflection or ventricular activation time (VAT).





1. Spodick DH. Chest. Left ventricular diastolic overload in young Africans.1986 Aug;90:308.

R Peak Time, intrinsicoid deflection or Ventricular Activation Time (VAT) in leads close to the LV: I, aVl, V_5-V_6

LV DIASTOLIC, ECCENTRIC OR VOLUMETRIC HYPERTROPHY

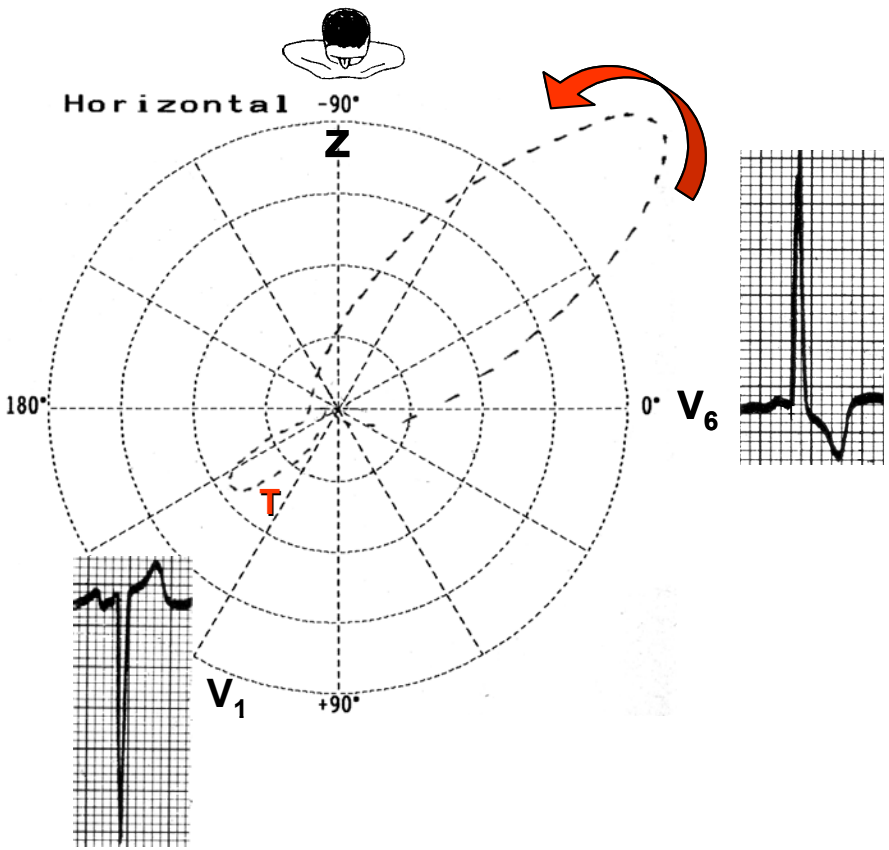
R waves of increased voltage in the leads that face the LV: DI, aVL, V_5 and V_6 and deep S waves in the leads from which vector 3 moves away: V1 and V2. Instantaneous maximal vector > 2.2 mV with pear-shaped narrowing of QRS loop in the HP.

Vector 3 represents the activation from the endo to epicardium of the free walls of both ventricles, and its direction is heading to the predominant LV, i.e. backward, to the left and below. It stretches between 40 ms and 60 ms. ST upwardly concave and elevated in V_5 and V_6.

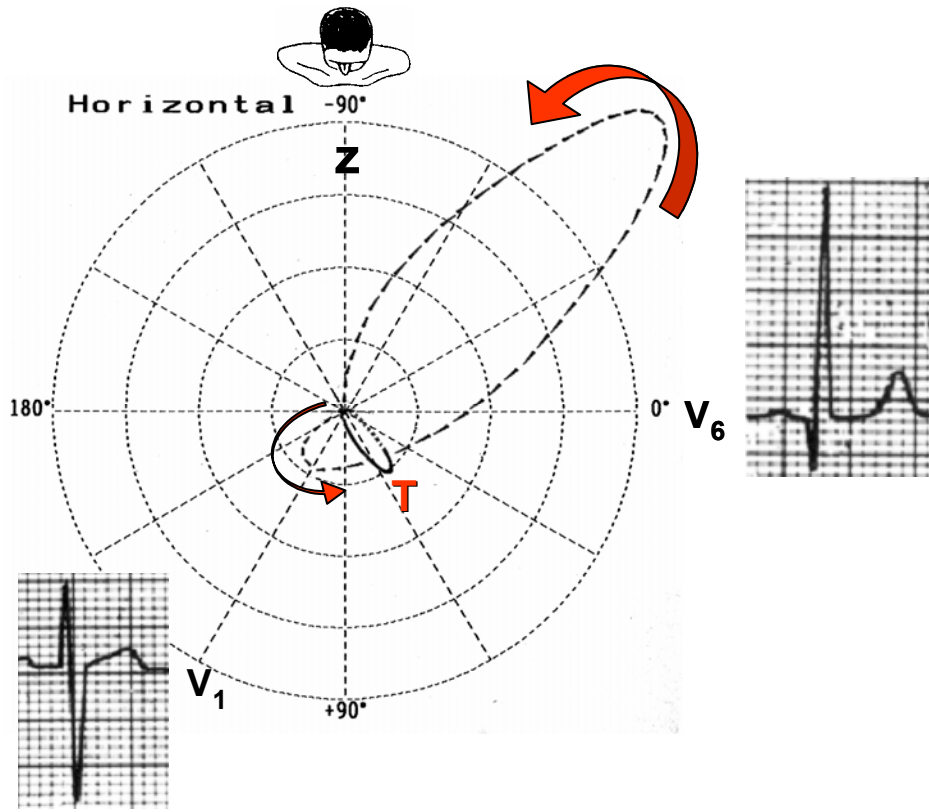
The T waves matching with QRS.

THE TWO CLASSICAL ECG/VCG MODALITIES OF LVH

LV CONCENTRIC OR SYSTOLIC HYPERTROPHY



LV DIASTOLIC ECCENTRIC OR VOLUMETRIC HYPERTROPHY

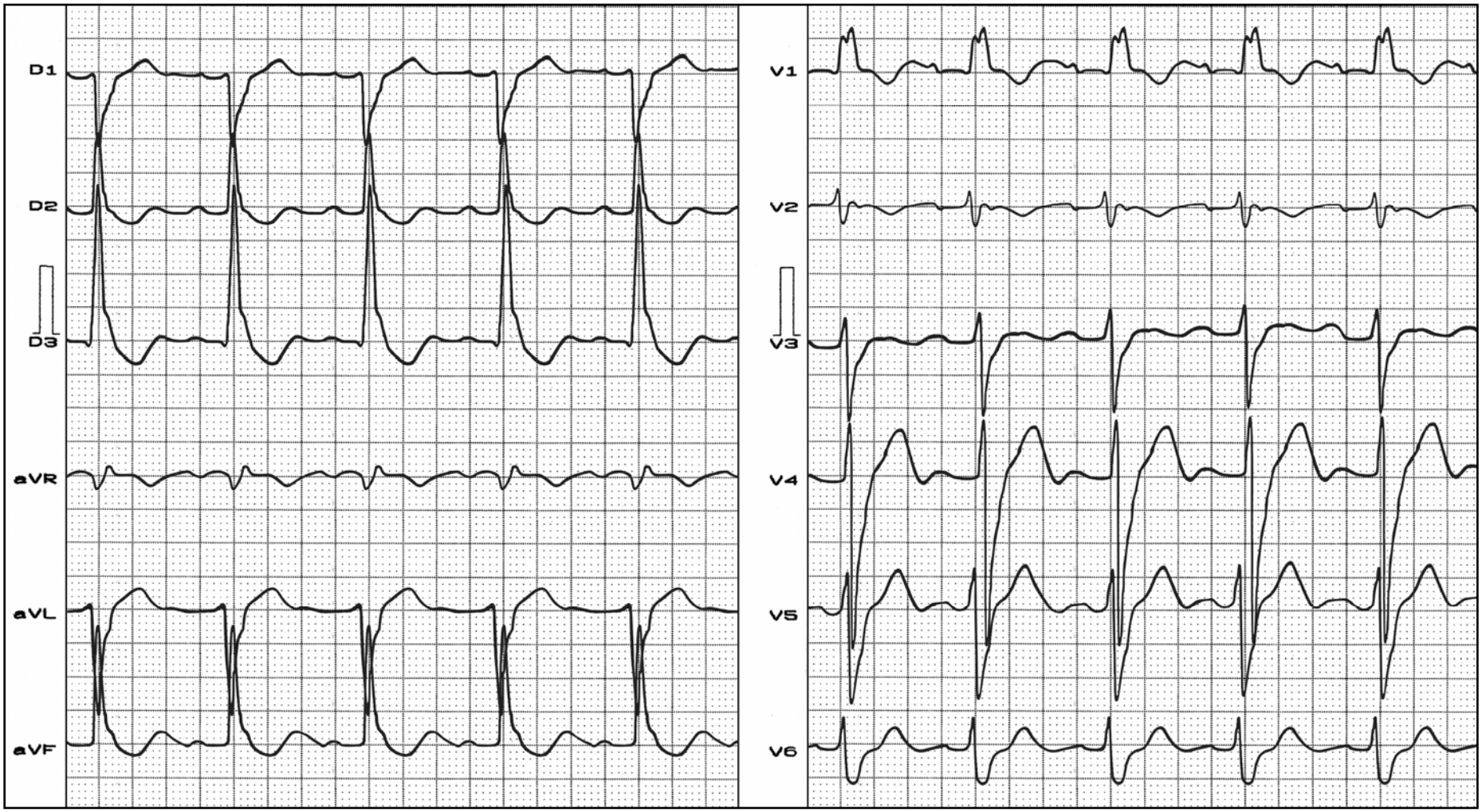


1. Cabrera E, Monroy JR. Am Heart J. Systolic and diastolic loading of the heart. I. Physiologic and clinical data.1952 May;43:661-668.
2. Cabrera E, Gaxiola A. A critical re-evaluation of systolic and diastolic overloading patterns.Prog Cardiovasc Dis. 1959 Nov; 2:219-236.
3. SedziwyL, ShillingfordJ. Cardiographic patterns in systolic and diastolic overload of the left ventricle. Br Heart J. 1961 Sep;23:533-538.
4. Traywick JP, Maron BJ, Schuberth K, Krovetz LJ. The electrocardiographic diagnosis of left ventricular hypertrophy in apparently normal children. J Pediatr. 1973 Aug;83:201-205.
5. Evans W. The electrocardiogram in the diagnosis of systemic hypertension. Br Heart J. 1962 Jul;24:469-482.

Name: ASP **Gender:** Male **Age:** 37 yo. **Ethnic Group:** Caucasian

Weight: 75 Kg.; **Height:** 1,92 m.; **Biotype:** Ectomorph.; Date: 01/31/2010

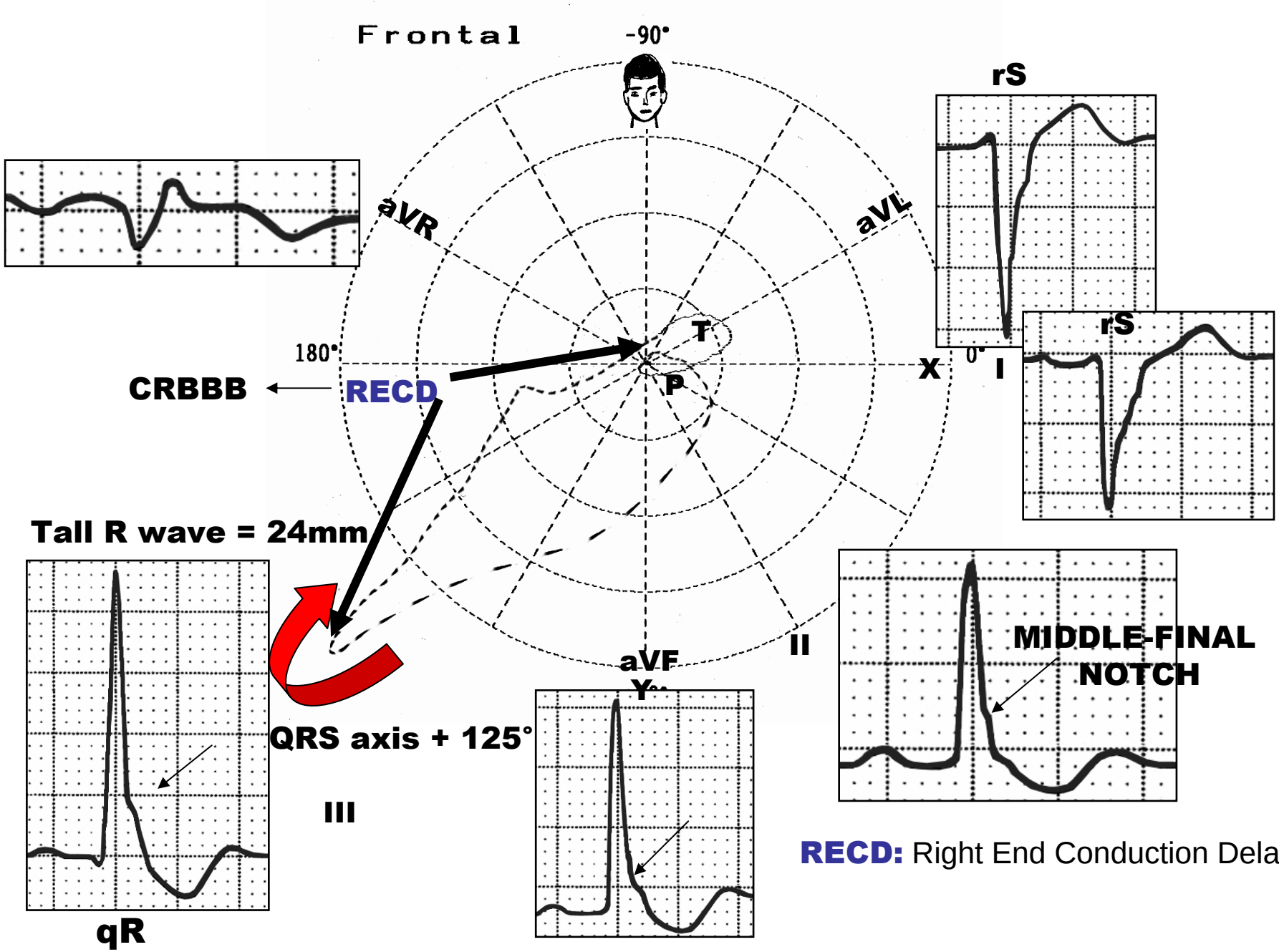
ECG preformed 10 years latter



First degree AV block (PR interval 240ms) Complete RBBB and LPFB.

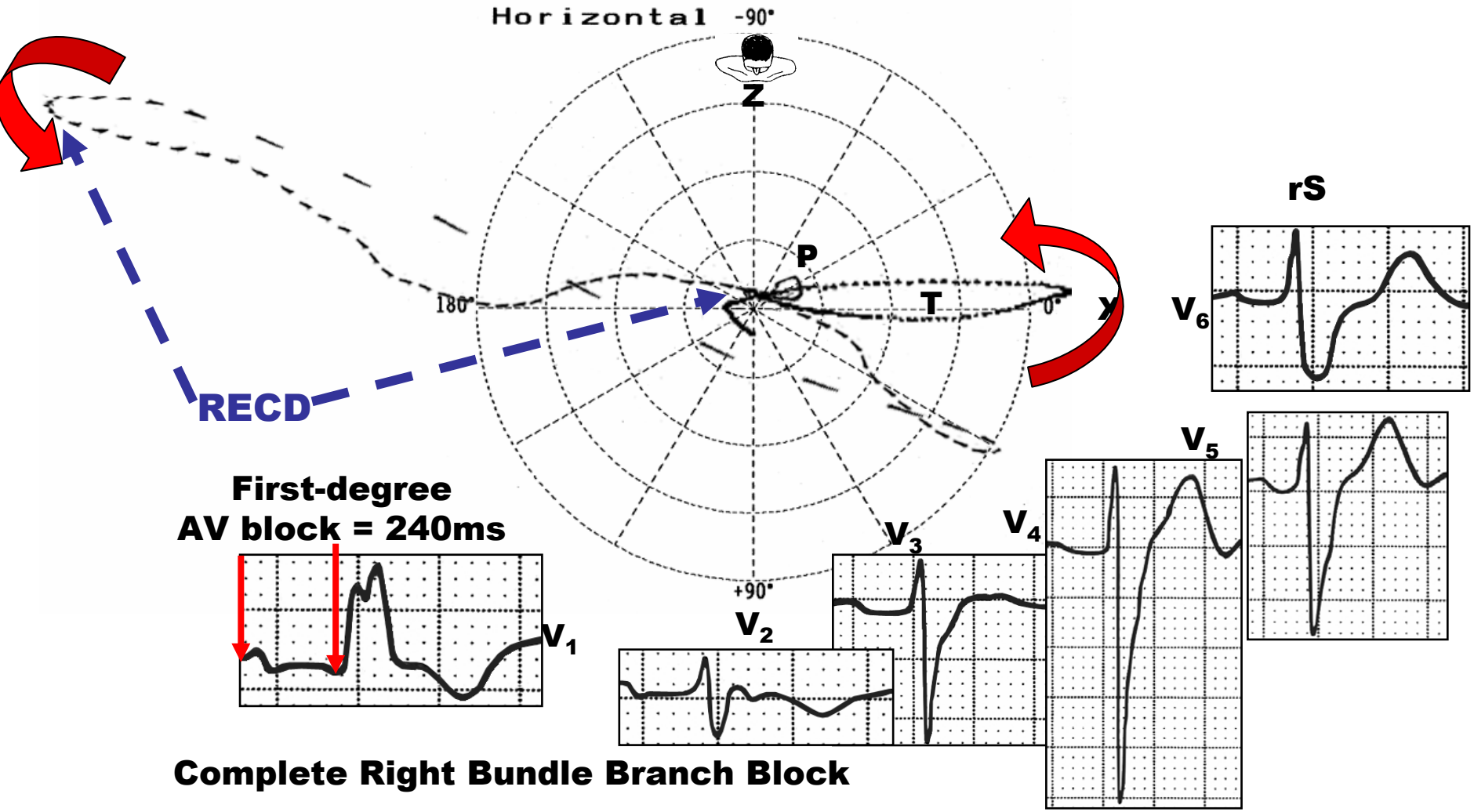
Conclusion: probable trifascicular block.

ECG/VCG CORRELATION FRONTAL PLANE



ECG/VCG CORRELATION HORIZONTAL PLANE

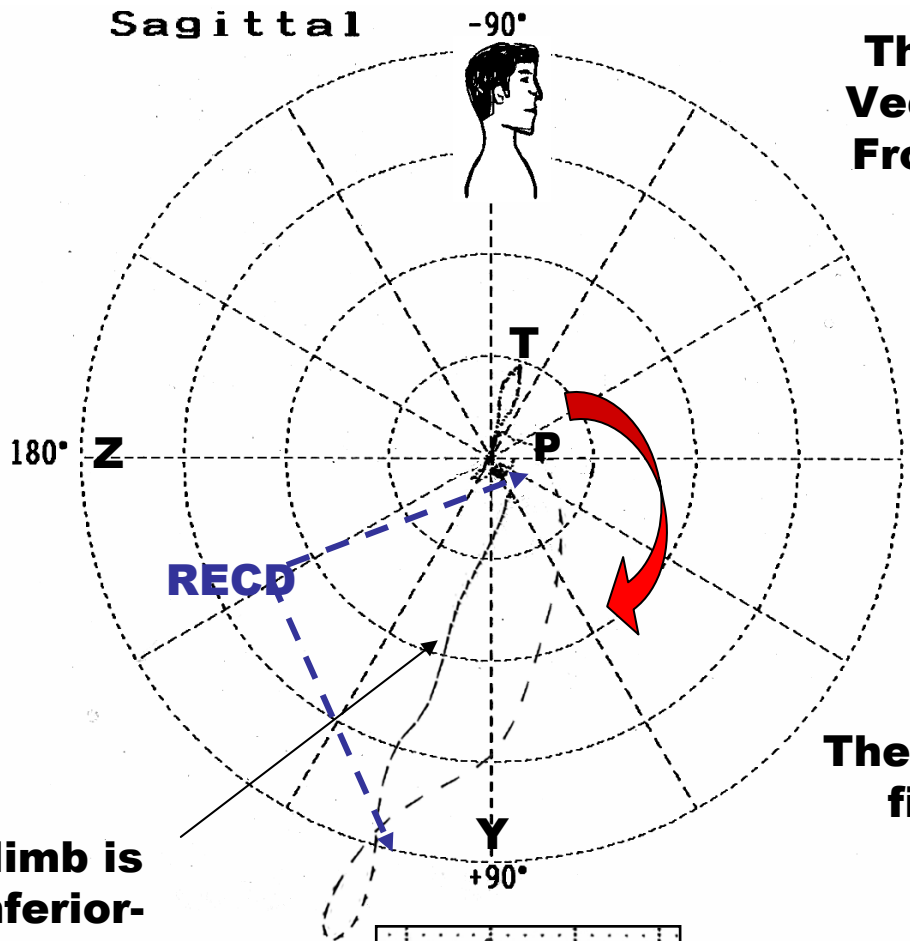
Right Bundle Branch Block
Cabrera VCG type or Kennedy type II



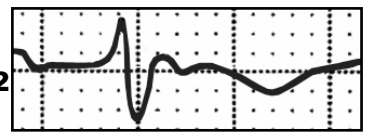
Complete Right Bundle Branch Block

1. Cabrera E, Guzman C, Cardenas M. Diagnostic and physiopathological aspects of the right bundle branch block. Arch Inst Cardiol Mex. 1955 Sep-Oct;25:593-612.
2. Varriale P, Kennedy RJ. Right bundle branch block and right axis deviation in patients with coronary artery disease.Am Heart J. 1971 Feb;81:291-292.

ECG/VCG CORRELATION RIGHT SAGITTAL PLANE



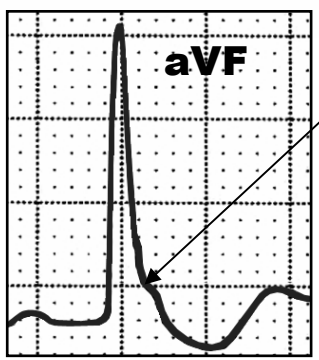
The initial 10-20ms Vector is directed to Front and superiorly



Most of the QRS loop is located on inferoposterior quadrant

The QRS rotation is figure in eight

The afferent limb is Located on inferior-posterior Quadrant



Middle-final Notch

The mean maximal vector is located in +110°

LEFT POSTERIOR FASCICULAR BLOCK(LPFB) POSSIBLE ETIOLOGIES

It is the most rare block of all intraventricular blocks

I) Chronic Chagasic cardiomyopathy the most frequent one in Latin America

II) Coronary insufficiency: it constitutes the main cause in the first world, associated or not to infarction, especially inferior or inferodorsal myocardial infarction or in combination [1;2;3;4;5].

(IIa) During the acute phase of ischemia[5].

(IIb) During a variant of Prinzmetal's anginal syndrome6

(IIc) During the acute phase of infarction: 0.2% to 0.4%: inferior or rarely anterolateral. Histologically, acute changes involving mainly the posterior septal and midseptal fibres are observed in 6 of the 8 cases studied in acute myocardial infarction. The ocurence is an ominous sign, since hospital mortality rate was 87%[7].

(IID) Transient induced by exercise test[8].

1. **Ogawa S, Kimura M, Okada M, Ogino T, Katayama K.A case of acute anterolateral infarction complicated with "left posterior hemiblock".Jpn Heart J. 1976 Jan;17:123-32.**
2. **Rizzon P, Rossi L, Baissus C, Demoulin JC, Di Biase MLeft posterior hemiblock in acute myocardial infarction. Br Heart J. 1975 Jul; 37:711-720.**
3. **Kanemoto N, Chino M, Nakamura Y. Left posterior hemiblock in ischemic heart disease Kokyu To Junkan. 1974 Sep;22:663-669.**
4. **Castellanos A, Portillo B, Vinsant MO, Lemberg L, Myerburg RJ. Difficulties in the diagnosis of "pure" left posterior hemiblock (without right bundle branch block) in acute myocardial infarction.Heart Lung. 1974 Jul-Aug;3:626-633.**
5. **de Kock J, Schamroth L. Right bundle-branch block associated with transient left posterior hemiblock in a case of acute myocardial infarction. S Afr Med J. 1974 Jun 15;48:1237-1240.**
6. **Gorfinkel HJ, Inglesby TV, Lansing AM, Goodin RR. ST-segment elevation, transient left-posterior hemiblock, and recurrent ventricular arrhythmias unassociated with pain. A variant of Prinzmetal's anginal syndrome.Ann Intern Med. 1973 Dec;79:795-799.**
7. **Medrano GA, Brenes CP, Sodi-Pallares D. Necrosis of the posterior aspect of the free left ventricular wall associated with block of the posterior subdivision of the left bundle branch of His. Experimental electro- and vectorcardiographic study. J Electrocardiol. 1971;4:44-49.**
8. **Bobba P, Salerno JA, Casari Transient left posterior hemiblock. Report of four cases induced by exercise test. A.Circulation. 1972;Nov;46:931-938.**

III) Aortic insufficiency: attributed to the mechanical effect of jet regurgitation on the posterior portion of the left septum, the site that the thick left posterior fascicles goes through (LV inflow tract). This is probably the mechanism of the present case.

IV) Aortic stenosis

V) Aortic stenosis associated with aortic insufficiency

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

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

VIII) Supravalvar aortic stenosis

IX) Coarctation of the aorta

X) Dissecting aortic aneurysm

XI) Massive calcification of the "cardiac skeleton"

XII) Cardiomyopathies

XIII) Myocarditis[1]

XIV) Infiltrative myocardial diseases

XV) Systemic hypertension

XVI) Hyperpotassemia

XVII) Transitorily, during contrast injection in the right coronary artery and in acute pulmonary embolism Acute cor pulmonale? (transient LPFB?)[3].

XVIII) Interventricular septum tumor[2].

1. **Forfang K, Lippestad CT. Transient left posterior hemiblock in acute myocarditis. J Electrocardiol. 1974 Feb;7:83-85**
2. **Cola H, Hoffman R, Borrega NG, Lazzar i JO. Left posterior hemiblock related to an interventricular septum tumor. First case in the literature. Eur Heart J. 1992 Apr;13:574-575.**
3. **Scott RC. The S1Q3 (McGinn-White) pattern in acute cor pulmonale: a form of transient left posterior hemiblock?Am Heart J. 1971 Jul;82:135-137.**

Isolated LPFB is a very rare finding; its prognostic significance is unknown and is commonly associated with RBBB. The prognosis is much more serious with a great propensity to develop complete AV block and Adams-Stoke eposides[1]. In presence of RBBB intermittent right axis deviation is a indicative of intermittent LPFB[2]. Exercise treadmill test (ETT) with effort induced LPFB could be indicative of severe reversible myocardial ischemia secondary to a left main coronary arterial stenosis or obstruction; tree or double vessel coronary artery disease(CAD[3;6].

Although isolated LPFB is a very rare conduction disturbance it is clinically important. Its appearance is reliably connected with Inferior MI and generally reflects severe three-vessel CAD or left main requiring invasive investigation[4]. Isolated LPFB in the absence of associated RBBB is an exceptional ECG finding. In view of its anatomy and the fact that it receives a dual blood supply, the posterior fascicle of the left bundle branch appears to be less vulnerable than the anterior fascicle or the right bundle. Additionally mechanical disruption of the left posterior fascicle can produce isolated LPFB. This has been demonstrated in animal models. However, such occurrence has not been noted in humans. The development of isolated LPFB complicating IMI may, therefore, serve to alert to the possible underlying inferior septal rupture[5]. Relation prevalence $^{\text{isolated LPFB}}/_{\text{LPFB +CRBBB}} = 1{:}10^{6}$.

When LBBB shows intermittent right axis deviation could be an evidence of LPFB associated with predivisional LBBB[7].

1. **Elizari MV, Acunzo RS, Ferreiro M.Hemiblocks revisited. Circulation. 2007 Mar 6;115:1154-1163.**
2. **Cheng TO. Intermittent right axis deviation in the presence of complete left bundle branch block. Int J Cardiol. 2006 Nov 18;113:406-407.**
3. **Madias JE, Knez P.Transient left posterior hemiblock during myocardial ischemia-eliciting exercise treadmill testing: a report of a case and a critical analysis of the literature. J Electrocardiol. 1999 Jan; 32:57-64.**
4. **Godat FJ, Gertsch M. Isolated left posterior fascicular block: a reliable marker for inferior myocardial infarction and associated severe coronary artery disease. Clin Cardiol. 1993;16:220-226.**
5. **Rokey R, Chahine RA.Isolated left posterior fascicular block associated with acquired ventricular septal defect. Clin Cardiol. 1984 Jun;7:364-369.**
6. **Demoulin JC, Kulbertus HE.Histopathologic correlates of left posterior fascicular block. Am J Cardiol. 1979 Nov;44:1083-1088.**
7. **Vera Z, Ertem G, Cheng TO. Left bundle branch block with intermittent right axis deviation. Evidence for left posterior hemiblock accompanying predivisional left bundle branch block. Am J Cardiol. 1972 Dec;30:896-901.**

CAUSES OF GREATER VULNERABILITY OF THE LEFT ANTERIOR FASCICLE (LAF) IN COMPARISON TO THE LEFT POSTERIOR FASCICLE (LPF)

1) ANATOMICAL:

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

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

2) ELECTROPHYSIOLOGICAL:

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

3) VASCULAR.

LPF always irrigated by the two systems of the ADA and RCA. LPFB during acute MI is an ominous sign with near 90% of mortality. LAF is irrigated by branches of the LAD in 40 % of cases, branches of the RCA in 10% of cases and finally double irrigation (ADA & RCA) in 50% of cases.

4) TOPOGRAPHIC.

The LPF runs through a more protected area, with less pressure mechanic impact.

NOMENCLATURES USED IN THE LITERATURE SEMANTIC DISCUSION: THE HEXAFASCICULAR NATURE OF THE HIS SYSTEM

1. **Left Posterior Fascicular Block (LPFB)**
2. **Left Posterior Hemiblock (LPHB)**
3. **Block Of The Posterior Subdivision Of The Left Bundle-branch**
4. **Block Of The Posterior Subdivision Of The Left Bundle Branch Of His.**
5. **Left Inferior Intraventricular Block**
6. **Posterior Hemi-block**

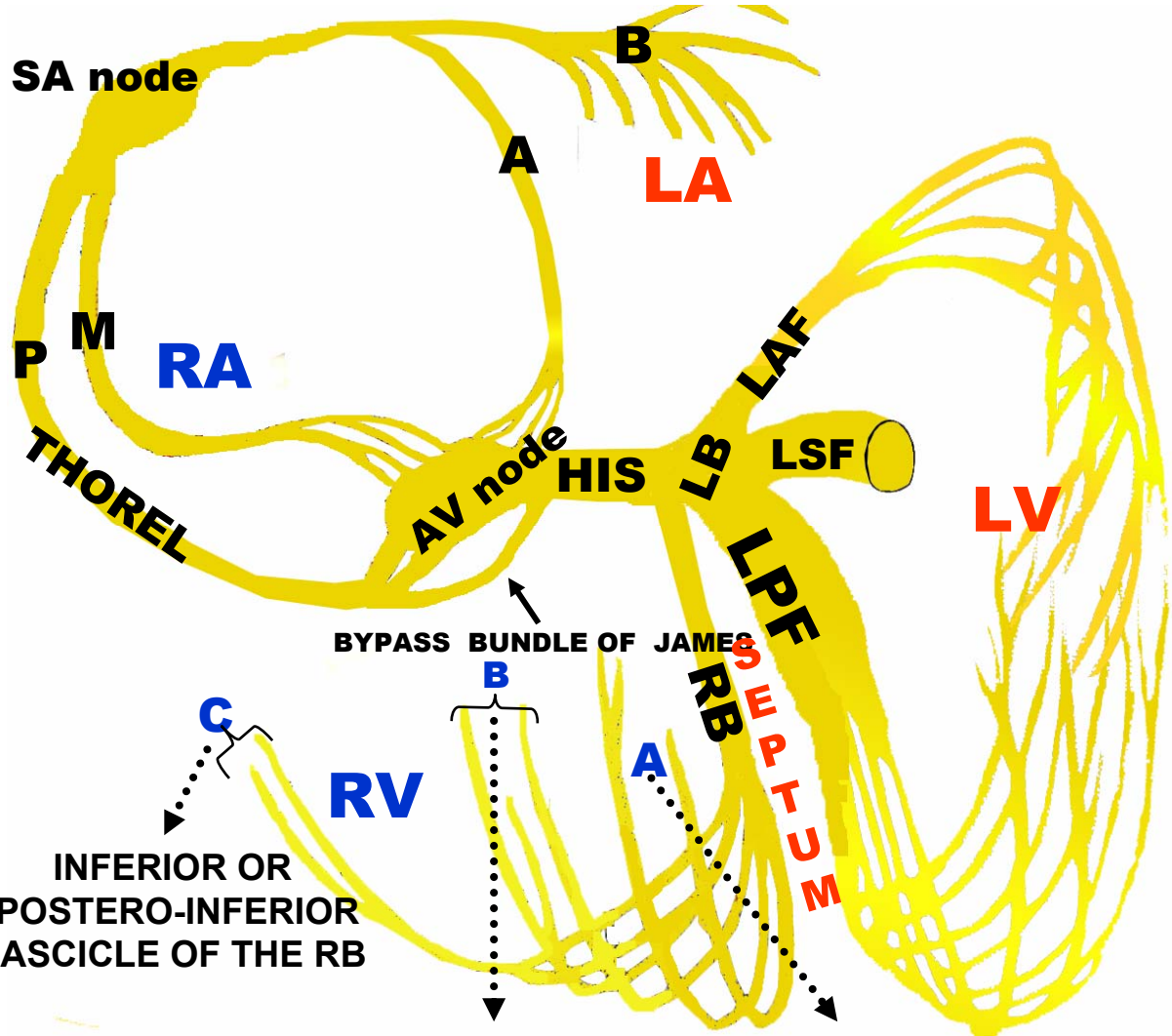
Mauricio Rosenbaum and his school "established" in the world of electrocardiography, the trifascicular concept of the intraventricular His system (RB + LAF + LPF)[1;2;3;4].

Uhley proposed[5] the tetrafascicular concept: RB + LAF + LPF + LSF. We believe in the existence of six fascicles with electrovectocardiographic expression: LAF + LPF + LSF for the Left intraventricular system and three fascicles for the right bundle on the free wall of right ventricle:

1. Superior or subpulmonary fascicle of the right bundle affected in Brugada syndrome and ARVD/C
2. Middle fascicle of the RB, and
3. Inferior or posteroinferior fascicle of the RB.

1) Rosenbaum MB. J Electrocardiol 1969;2:197-206.
2) Rosembaum MB, et al. Am Heart J 1969; 78: 306-312
3) Rosembaum MB, et al. Mod Concepts Cardiovasc Dis 1970 39:141-7
4) Rosembaum MB, et al. The Hemiblocks. Tampa tracing, Florida 1970; p1
5) Uhley HN.: The Quadrifascicular Nature of the Peripheral Conduction System, in Dreifus LS, and Likoff W,.(eds.):Cardiac Arrhythmias (New York): Grune & Stratton. Inc. 1973

SINOATRIOVENTRICULAR AND INTRAVENTRICULAR CONDUCTION



INTERNODAL BUNDLES

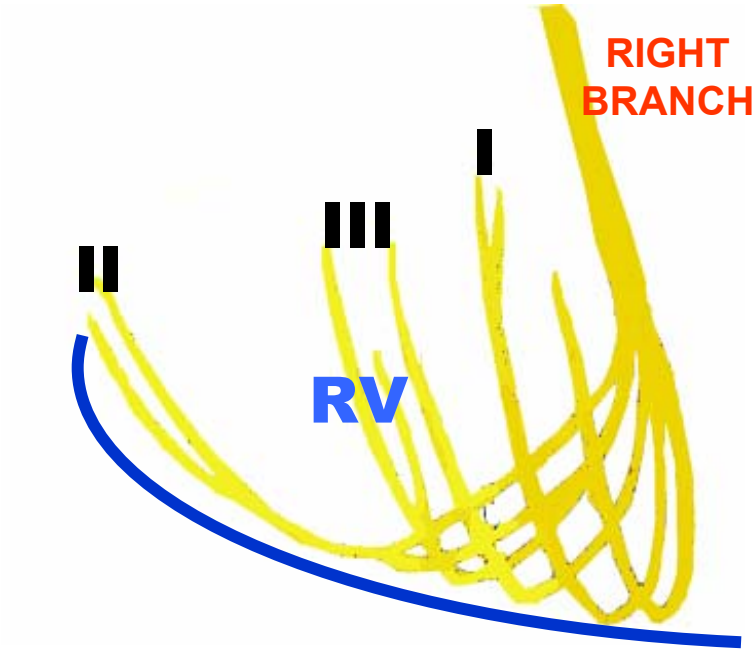
A ANTERIOR

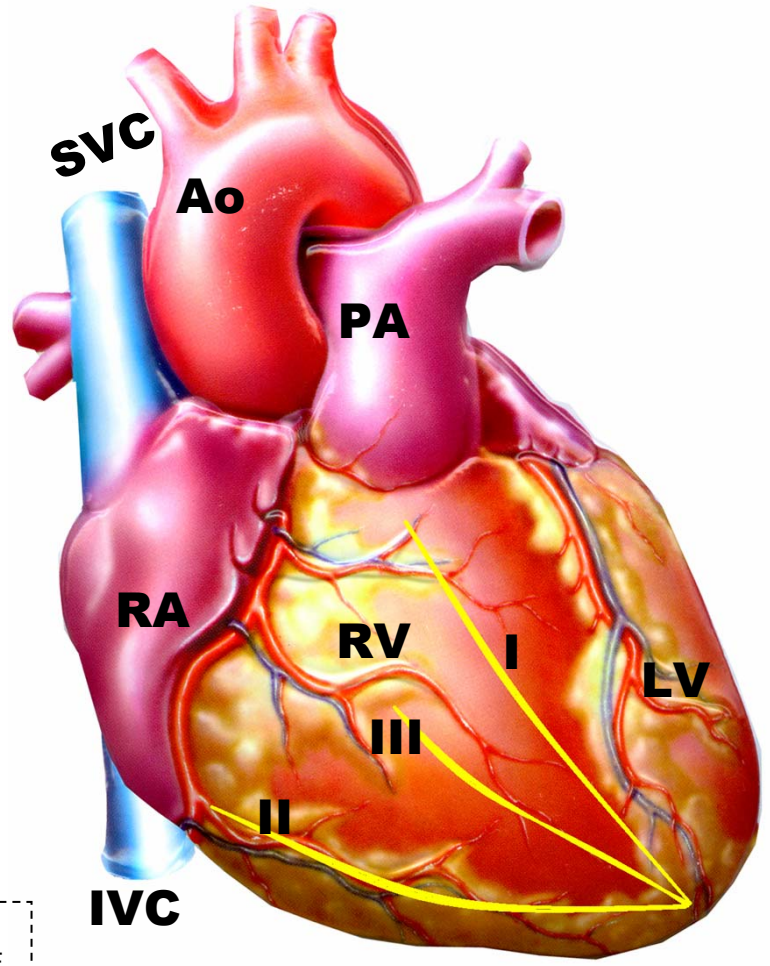
M MIDDLE OR OF WENCKBACH

P POSTERIOR OR OF THOREL

B BACHMAN'S FASCICLE (IT ACTIVATES THE LA)

DISTRIBUTION OF THE THREE FASCICLES OF THE RIGHT BRANCH OF THE HIS BUNDLE IN THE RV FREE WALL





I - TERRITORY OF THE SUPERIOR OR SUBPULMONARY FASCICLE
II – TERRITORY OF THE INFERIOR OR POSTERO-INFERIOR FASCICLE
III – TERRITORY OF THE MIDDLE FASCICLE

The LPFB can be used as the endpoint of ablation to treat verapamil-sensitive **idiopathic left ventricular tachycardia (ILVT) with RBBB configuration and left-axis deviation**. It makes the ablation procedure simple, safe and effective. It is especially important for patients whose VT can not be induced or the inducible condition is unstable. It also implies that perhaps the LPF is a critical part of the re-entry circuit[1].

1. **MA Fu-sheng, MA Jian, TANG Kai, HAN Hao, JIA Yu-he, FANG Pi-hua, CHU Jian-min, PU Jie-lin, ZHANG Shu Left posterior fascicular block: a new endpoint of ablation for verapamil-sensitive idiopathic ventricular tachycardia Chinese Medical Journal, 2006, Vol. 119 No. 5 : 367-372**

ISOLATED LPFB (MONOFASCICULAR BLOCK) ECG CRITERIA

Isolated LSFB is a very rare condition. It is more commonly associated with RBBB, inferior MI or both.

QRS duration <120ms. From 90ms to 110ms. QRS duration augmentation not exceeds 20ms.

Frontal Plane

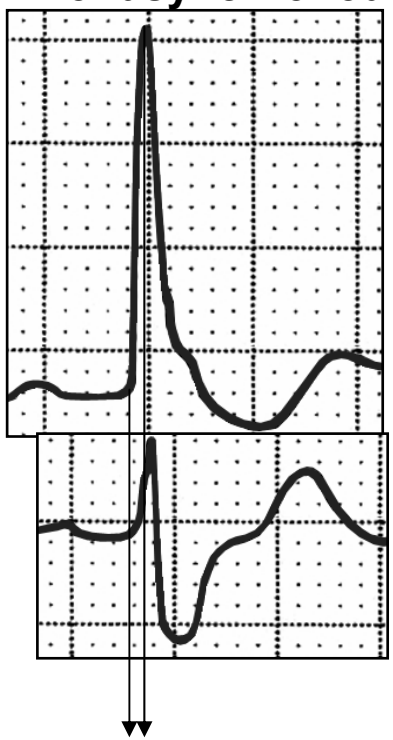
1. **Right axis deviation in the frontal plane (≥90°). Usually close +120° and it is not a pathognomonic sign. The diagnosis is always clinical-electrocardiography. It is necessary no evidence of right ventricular hypertrophy such as cor pulmonale, pulmonary heart disease, pulmonary hypertension, lateral myocardial infarction, and vertical heart (asthenic biotype), because these conditions can result in the identical ECG features.**
2. **rS pattern in lead I and aVL. Since the posteroinferior fascicle is interrupted, the activation of the LV precedes superiorly and to the left. This is due to the fact that anterior superior portion of the LV is initially activated (20ms initial vector). Therefore a small 20ms r wave in lead aVL and I and concomitantly a small q 20ms wave in III, aVF and II.**
3. **qR pattern in leads II, III, aVF. If inferior MI is associated abnormal initial Q wave (≥40 ms) is registered in these leads[1]. initial q wave is always present in III and may be small or absent in II or aVF.**

1. Castellanos A Jr. Bull N Y Acad Med. Diagnosis of left anterior hemiblock and left posterior hemiblock in the presence of inferior wall myocardial infarction.1971 Aug;47:923-30.
2. Rosenbaum MB, Elizari MV. Left anterior and left posterior hemiblocks. Electrocardiographic manifestations.Postgrad Med. 1973 Apr;53:61-66.
3. Medrano GA, Brenes C, De Micheli A, Sodi-Pallares D. Posterior subdivision block of the left branch of the bundle of His isolated and combined with the block of the right branch. Experimental study Arch Inst Cardiol Mex. 1970 Jul-Aug;40:423-436.
4. Medrano GA, Brenes C, Sodi-Pallares D. Isolated posterior-inferior necrosis of the left ventricle, and necrosis associated with block of the posterior subdivision of the bundle of His' left branch. Experimental study Arch Inst Cardiol Mex. 1970 Sep-Oct;40:645-654.

4. R of increased voltage in lead III ≥ R in lead II: QRS axis closer to +120° (III) than +60° (II). When closer to the latter, it would indicate an incomplete form of LPFB.

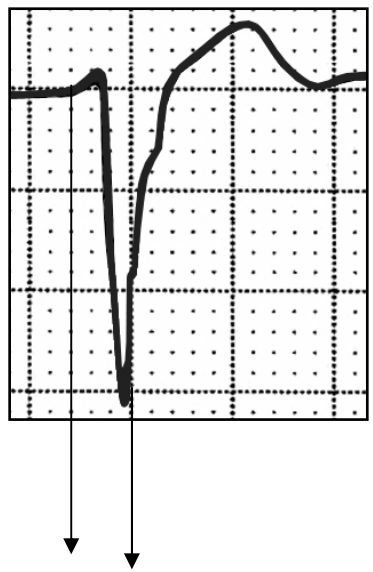
5. Notch in the descending limb of the R wave in III, (middle-final notch) aVF and II
The ST/T are usually normal in isolated LPFB without BRBB, infarction or ventricular enlargement.

6. The inscription of Ventricular Activation Time (VAT) or "R-peak time" in aVF or V6 no exceeds 35ms[1]. The VAT is always mayor in aVL. The phenomenon is consequence of asynchronous LV activation between inferior (aVF, V6) and superior layers of LV



VAT ≤ 35ms

TIME OF APPEARANCE OF R WAVE APEX: "R-PEAK TIME"



VAT ≥ 50ms

1. 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.

Precordial leads

1. **The QRS complexes are predominantly positive in lower V leads and predominantly negative in the higher V leads due to upward displacement of the electrical center of ventricular despolarization.**

2. **S waves in V_2-V_3 very deep because electro-vectorcardiographic features in HP are very similar with type C right ventricular hypertrophy(dislocated to back and rightward).**

3. **Transition zone dislocated to the left (in V_5 or V_6)**

4. **Initial q waves frequently are absent in V_5-V_6 , but small q waves may appear in theses leads recorded at lower level.**

5. **Left precordial leads with RS pattern similar to right ventricular hypertrophy**

1. Rosenbaum MB. The hemiblocks: diagnostic criteria and clinical significance. Mod Concepts Cardiovasc Dis. 1970 Dec;39:141-146

Medrano GA, Brenes CP, De Micheli A, Sodi-Pallares D. Block of the posterior subdivision of the left bundle branch of His. J Electrocardiol. 1970;3:309-315.

Glasser SP, Flannery EP, Czarnecki SW. Intermittent isolated left posterior fascicular block. Am J Med Sci. 1971 Mar;261:155-160.

ISOLATED LPFB: VCG CRITERIA QRS FP

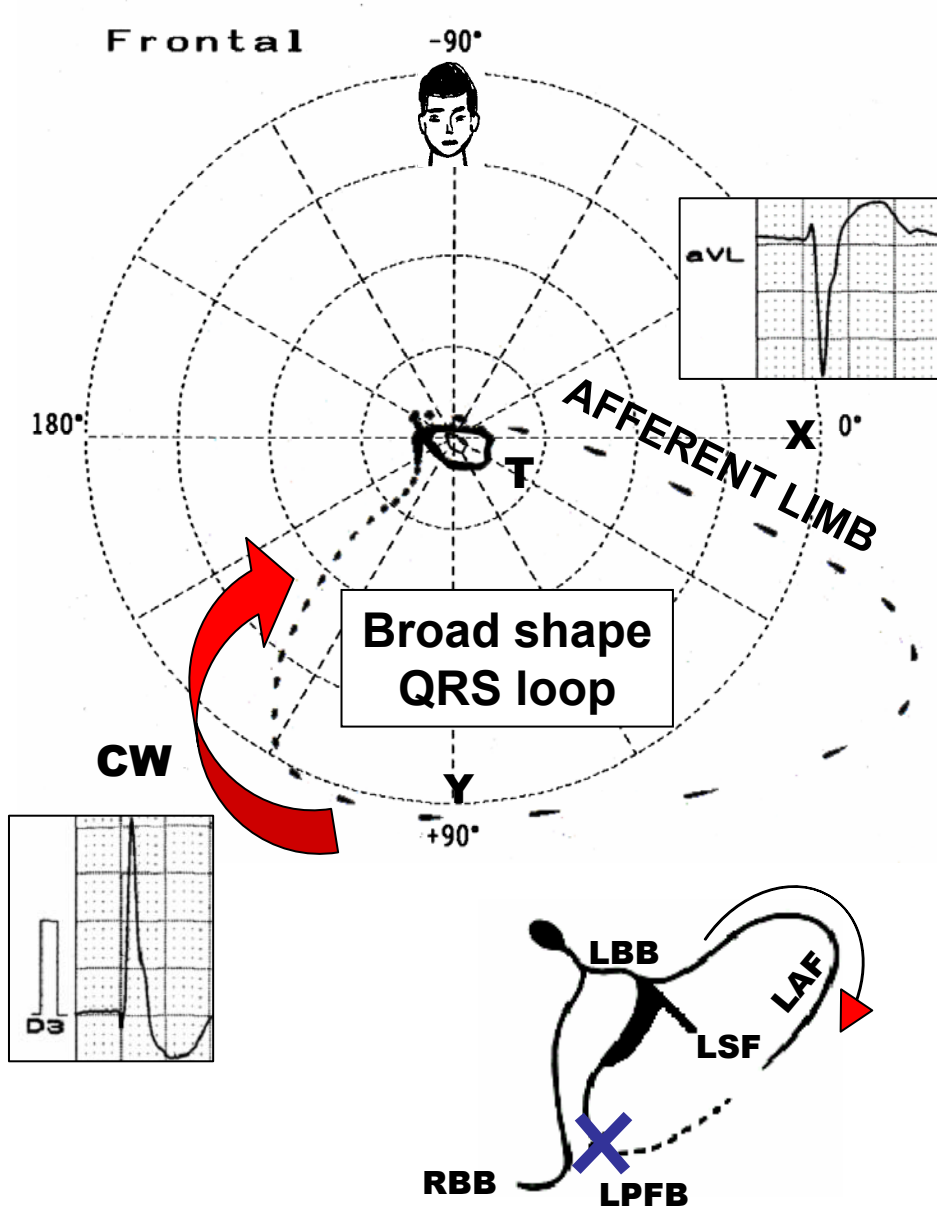
1.The initial QRS 10-20ms vector is directed superiorly and to left. (near -45º) with possible delay (initial 10 to 25 ms). If associated to inferior myocardial infarction, superior initial forces of 25 ms or more (more than 12.5 dashes above the orthogonal X lead. 1 dash = 2 ms).

2. Broad shape QRS loop, with clockwise rotation. Cooksey, Dunn and Massie said that occasionally, it may be in “eight” with a counterclockwise terminal portion (10%).

3.≈20% of QRS loop area is located on right inferior quadrant. If there is association to Complete RBBB, 40% or more

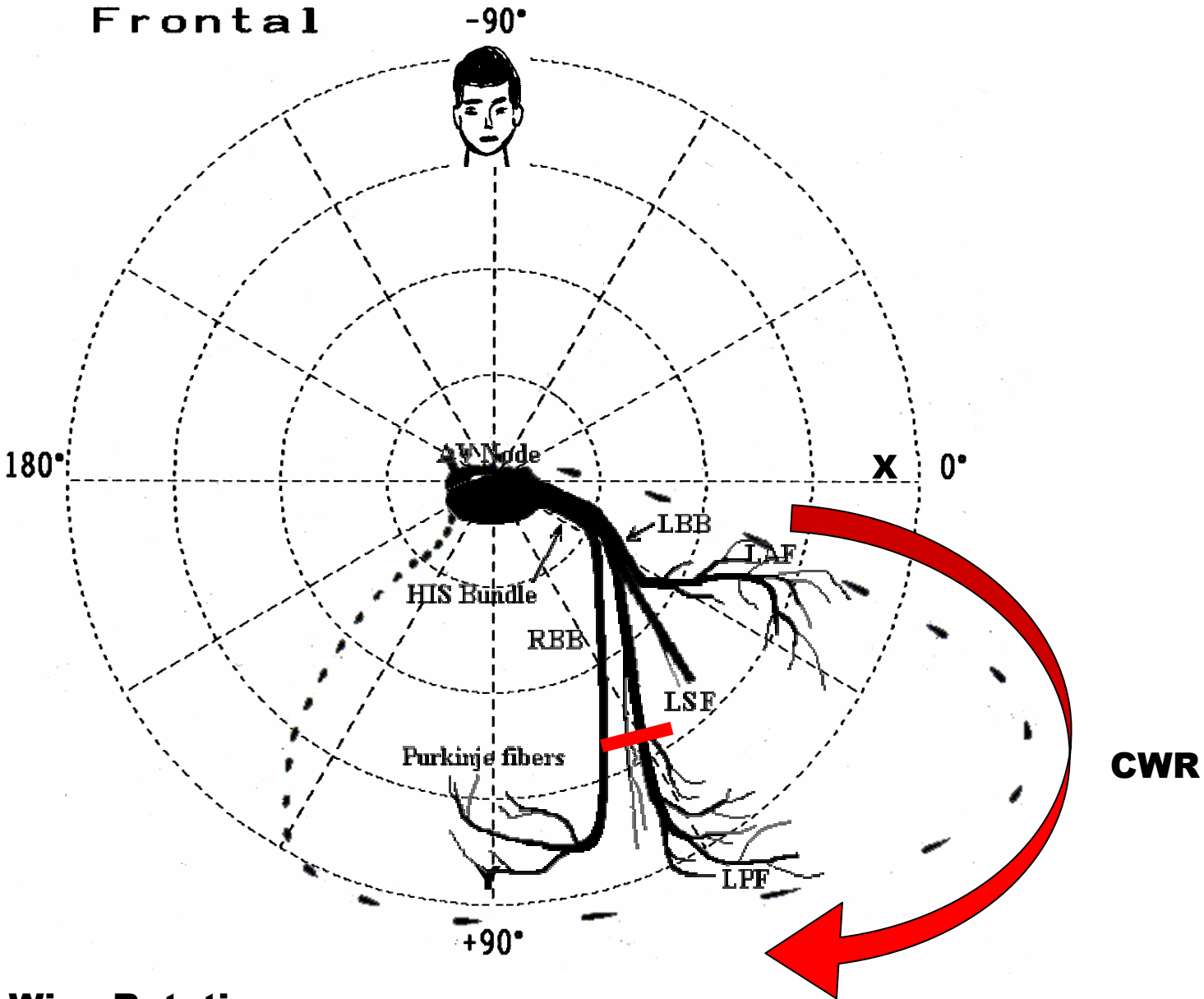
4.The afferent limb is located in right inferior quadrant and the terminal portion of the QRS loop may proceed to the right superior quadrant before returning to E point

5.The mean maximal vector is usually between +80° and +140°.



1. Watt TB Jr, Pruitt RD. Left posterior fascicular block in canine and primate hearts. An electrocardiographic study.Circulation. 1969 Nov;40:677-685.

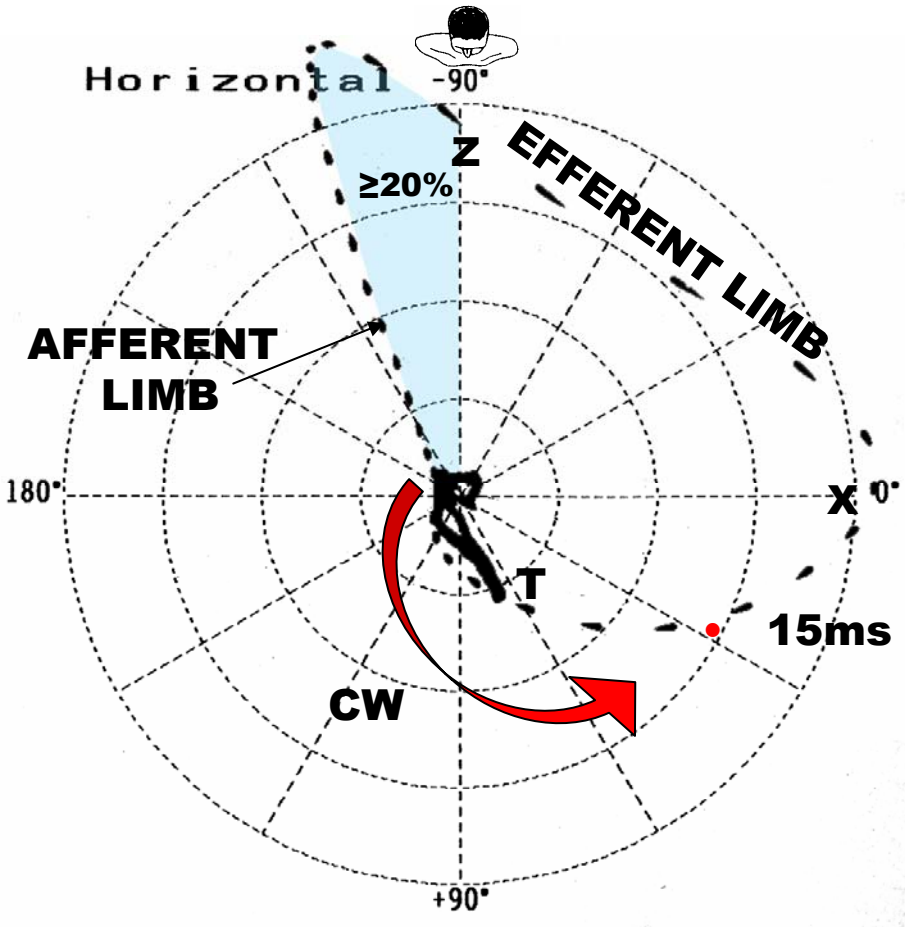
ISOLATED LPFB FRONTAL PLANE



CWR = Clock Wise Rotation

HORIZONTAL PLANE

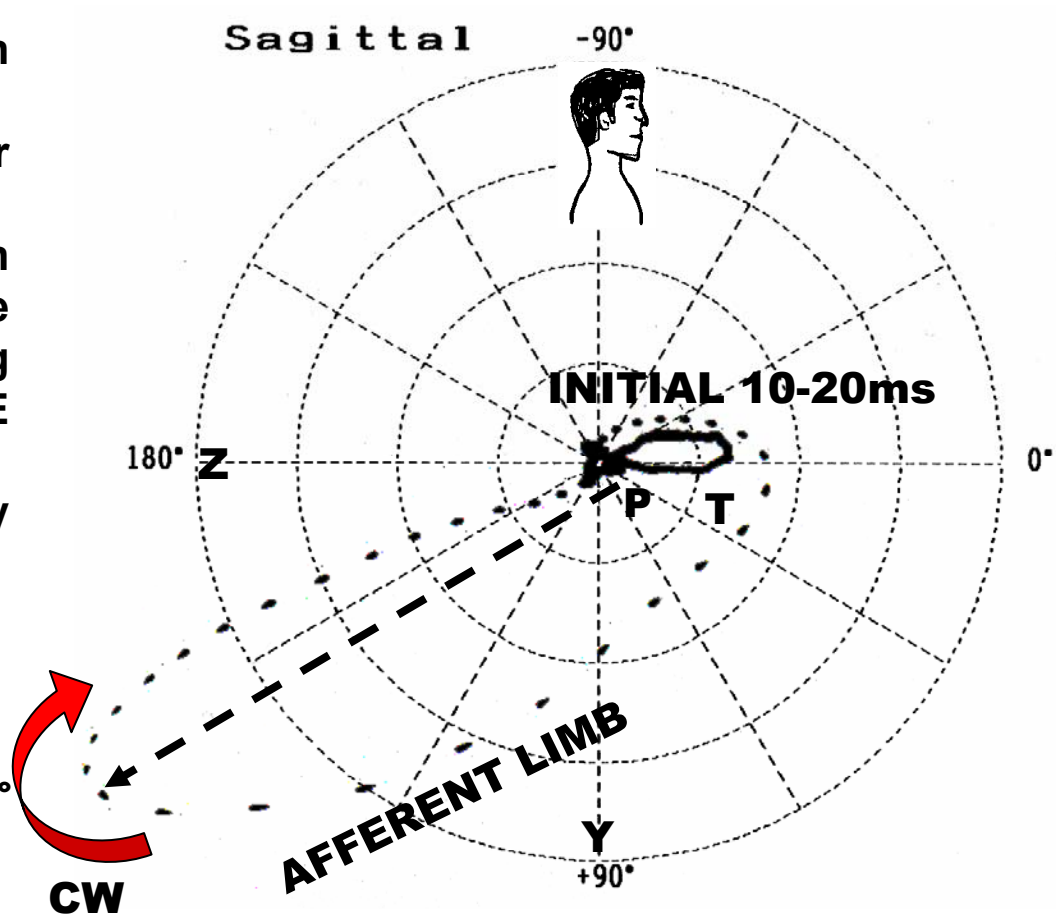
1) Initial 10-20ms vector directed to front and rightward or leftward.
2) The efferent limb located on left posterior quadrant
3) The afferent limb is located on right posterior quadrant and proceeded to the right as it return to the E point
4) The maximal QRS vector is directed posteriorly and to the right.
5) QRS loop with clock wise rotation
6) The mean maxima QRS vector is at -150°
7) ≥ 20% of QRS loop area located on right posterior quadrant
8) T loop to the front and the left (+60º) and clockwise rotation.



1. Lopes VM, Miguel JM, dos Reis DD, da Costa BC, de Pádua F.J Left-posterior hemiblock. Clinical and vectorcardiographic study of twenty cases. J. Electrocardiol. 1974;7:197-214.
2. Tomov I. Vutr Boles. Vectorcardiographic analysis of left posterior hemiblock (lead by the Frank system)] 1973;12:20-25.

RIGHT SAGITTAL PLANE

1. The initial 10-20ms vector is directed to front and superiorly
2. Most of the QRS loop is located on inferoposterior quadrant
3. The QRS rotation is clockwise or figure in eight
4. The afferent limb is located on inferorposterior quadrant, but the terminal part of the limb may swing superiorly before returning to the E point.
5. The mean maximal vector is usually between +80° and +140°



MEAN MAXIMAL VECTOR +140°

LPFB VCG BY FRANK METHOD

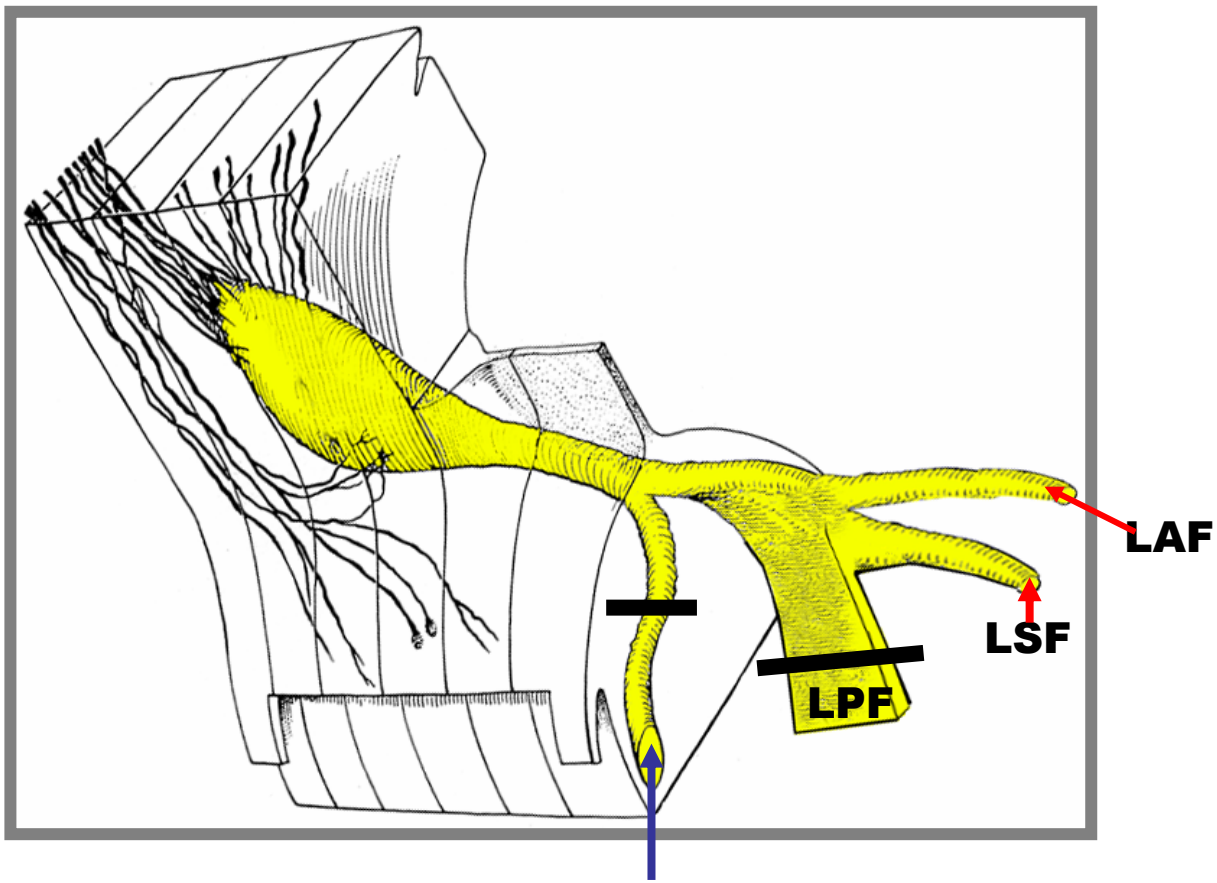
1. The most important diagnostic feature Is the opposite direction of the initial forces (left anterosuperior) and the maximal vector (right posteroinferior): the angle between these two vectors averaged 152°[1].

Other criteria, such as the direction of rotation or the axis of the frontal QRS loop, the vertical direction of the spatial loop, the presence of a q wave in leads II, III, and aVF of the ECG, are not mandatory for the diagnosis of LPFB.

1. **Brohet CR, Arnaud P.Spatial Frank vectorcardiogram in left posterior fascicular block. Criteria and correlation with clinical and electrocardiographic data. Br Heart J. 1977 Feb;39: 126-138.**
2. **Palmieri M, Ruggeri G, Zappalà A, Nava A. Study of left posterior hemiblock associated with right branch block. Clinical, electrocardiographic and vectorcardiographic study G Ital Cardiol. 1974;4:463-469.**
3. **Medrano GA, Brenes C, De Micheli A, Sodi-Pallares D. Clinical electrocardiographic and vectorcardiographic diagnosis of left posterior subdivision block, isolated or associated with RBBB. Am Heart J. 1972 Dec;84:727-737.**
4. **Castellanos A Jr, Chapunoff E, Castillo CA, Arcebal AG, Lemberg L. The vectorcardiogram in left posterior hemiblock associated with inferior wall myocardial infarction. Chest. 1972 Mar;61:221-227.**
5. **Brenes C, Medrano GA, Sodi-Pallares D. Block of the posterior subdivision of the left branch of the bundle of His. Clinical, electro- and vectorcardiographic study Arch Inst Cardiol Mex. 1970 Sep-Oct;40:621-634.**

LPFB associated with CRBBB

The ECG/VCG shows the characteristic features of LPFB in the frontal plane (FP) and the findings of CRBBB in precordial leads or horizontal plane. In FP from zero to 60ms are determinate to the LPFB and from 60ms to 120m or more by the CRBBB.



LAF: Left Anterior Fascicle
LSF: Left Septal Fascicle
LPF: Left Posterior Fascicle; LPFB: Left Posterior Fascicular Block
RBB: Right Bundle Branch. CRBBB: Complete Right Bundle Branch Block

FRONTAL PLANE(FP)

Combined dromotropic disorder (LPFB+CRBBB) have four main directions of the QRS forces in the frontal plane:

10 to 20ms initial forces directed superiorly and to left. (near -45º) with possible delay (initial 10 to 25 ms) resulting in small q waves in inferior leads and small initial r wave in aVL and I. If associated to inferior myocardial infarction, superior initial forces of 25 ms or more (more than 12.5 dashes above the orthogonal X lead. 1 dash = 2 ms).

During 20 to 40 ms(efferent limb) the forces are located on inferior left quadrant and determine descending ramp of S wave in I and aVL and ascending ramp or R in III, aVF and II.

During 40 to 60ms the forces are located on inferior right quadrant point to +120°.

The terminal 60ms(60ms-120ms or more) are produced by CRBBB and directed rightward between +150° to +180°.

1. **Palmieri M, Ruggeri G, Zappalà A, Nava A. Study of left posterior hemiblock associated with right branch block. Clinical, electrocardiographic and vectorcardiographic study G Ital Cardiol. 1974;4:463-469.**
2. **Varriale P, Kennedy RJ. Right bundle branch block and left posterior fascicular block. Vectorcardiographic and clinical features. Am J Cardiol. 1972 Apr;29:459-465.**

HORIZONTAL PLANE(FP)/ PRECORDIAL LEADS

1. The normal q waves found in leads V4 to V6 are generally absent
2. Small q wave may be present in leas V4 to V6 recorder at a lower level[1]
3. A initial q wave is commonly recorded in lead V_1 even in the absence of anteroseptal myocardial infarction
4. There is a tendency for large R/S ratios to occur in the left precordial leads
5. The QRS complexes are predominantly negative in high V leads and largely positive in low V leads[2].

The evolution of bifascicular block CRBBB + LPFB to high degree block or complete AV block varies in different series from 55% (Rosembaum series with Chagasic patients[3]) to only 10% (Dhingra et al series[4]). These authors conclude that the clinical course of most of the patients was benign and the incidence of sudden death was relatively small. Symptomatic conduction disease occurred but could be definitely related to trifascicular disease in only one patient. The authors data suggest that prophylactic pacemaker insertion is not routinely indicated in patients with chronic RBBB and LPFB. Unfortunately this series had a short follow-up period ranged from 91 to 1,231 days (mean 671 +/-68).

1. **Tricot R, Valere PE, Guerot C. Combination of right bundle-branch block and left posterior intraventricular hemiblock (apropos of 6 cases) Arch Mal Coeur Vaiss. 1971; Feb;64:169-189.**
2. **Scanlon PJ, Pryor R, Blount SG Jr. Right bundle-branch block associated with left superior or inferior intraventricular block. Clinical setting, prognosis, and relation to complete heart block.Circulation. 1970 Dec;42:1123-1133.**
3. **Rosenbaum MB. The hemiblocks: diagnostic criteria and clinical significance. Mod Concepts Cardiovasc Dis. 1970 Dec;39:141-146.**
4. **Dhingra RC, Denes P, Wu D, Chuquimia R, Amat-Y-Leon F, Wyndham C, Rosen KM. Chronic right bundle branch block and left posterior hemiblock. Clinical, electrophysiologic and prognostic observations.Am J Cardiol. 1975 Dec;36:867-879.**

TRIFASCICULAR BLOCK

We must to suspect the presence of trifascicular block in the following circumstances[1;2;3]:

1) Presence of CRBBB + LPFB + first degree AV block
2) Presence of CRBBB + LAFB + first degree AV block
3) Presence of CRBBB + LPFB + second degree AV block
4) Presence of CRBBB + LAFB + second degree AV block
5) Chronic CRBBB + LAFB and LPFB on different occasions

The prolonged PR interval with bifascicular bundle branch block means, in the majority of cases, diffuse damage of the conduction system involving the AV node and the infranodal region; starting from 280ms, the delayed PR interval suggests involvement predominantly below the A-V node: to a higher PR interval corresponds a higher HV interval and also, to a relatively shortest AH interval[4].

Prophylactic pacing in perioperative is not necessary in asymptomatic patients with bifascicular block even in the presence of a long PR interval[5].

Simultaneous block of the LAF and LPF is considered a biphasic block and its association with the RBBB a triphasicular block[6].

1. **Seipel L, Gleichmann U, Loogen F.Z Kreislaufforsch. [Electrocardiographic picture of the right-bundle branch block with intermittent left-anterior and left-posterior hemiblock (intraventricular trifascicular block 1972 Mar;61:234-239.**
2. **Rosenbaum MB, Elizari MV, Lazzari JO, Nau GJ, Levi RJ, Halpern MS. Intraventricular trifascicular blocks. The syndrome of right bundle branch block with intermittent left anterior and posterior hemiblock. Am Heart J. 1969 Sep; 78:306-317.**
3. **Nohara Y, Konishi Y, Sakai A, Tomita T, Takegami T.Naika. Electrocardiography of heart block--with special reference to bilateral bundle branch block, trifascicular block, left hemiblock and peri-infarction block 1971;28:849-860**
4. **Barbosa EC, Ginefra P, da Rocha PJ, Musse NS, Boghossian SH, Albanesi Filho FM, Gomes Filho JB. Significance of prolonged PR interval in patients with His bundle branch block, bifascicular type.Arq Bras Cardiol. 1991 May;56:355-358.**
5. **Mikell FL, Weir EK, Chesler E. Perioperative risk of complete heart block in patients with bifascicular block and prolonged PR interval. Thorax. 1981 Jan;36:14-17.**
6. **Medrano GA, Brenes C, De Micheli A, Sodi-Pallares D. 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;40:752-770.**

COLLEAGES COMMENTARIES

Dear friends my intention is to analyze the rare ECG from a young male with full Marfan syndrome.
The first ECG shows an asymmetric hypertrophy remodeling. Is not easy to differentiate in a young male physiological remodeling from pathological remodeling.
Why?
Because the androgen hormone induces physiological hypertrophy remodeling as well as the high take off and the tall peck T wave and the sudden and fast decline of T2 I think that the maximal voltage is 25mm in the young male, over this measure is hypertrophy
The pattern of the phenotype of remodeling is determined by the fetal genes in an 8 month baby.
Why is this? Because the distribution of the tension receptors in the endocardium are heterogenic. In the experimental laboratory was found that higher concentration of tension receptors are in the septum and left lateral area of LV.
The genetic phenotype morphology of the remodelind as is expressed in the ECG will be the basis of the different phenotype in the pathologic hypertrophy remodeling
In according to Laplace law the systolic or diastolic overloading must increase the radius of the cavity to reduce the wall tension
The ECG can express only asymmetric hypertrophy, but how we can explain patients suffering from malignant hypertension with CVA, renal failure and hypertensive retinopathy or severe aortic stenos is of aortic or mitral failure with normal ECG?!!!

There are two situations of systolic overloading and normal ECG:
1.Normal ECG with ECHO hypertrophies this case of symmetric hypertrophy. It is the best scenario, the ECG is normal because the symmetric concealed electrocardiograph vectors, this is the best scenario because symmetric hypertrophy reduces the wall tension, and doesn't evolve to dilatation and isn't arrhytmogenic.
2.ECG normal and ECHO normal this is because annulations in the molecular cascade in the hypertrophy remodeling, but these patients suffered high wall tension which can be the cause of Atrial fibrillation and normal QRSi.

The ECG of the young male shows a deep S wave in V2 , expressing the hypertrophy area in the posterior paraseptal area, SV2 more than 25mm,and a very high R wave in V4 indicating the hypertrophy of the low septum and apex ,R wave more than 35mm

The second ECG shows a rare combination of CRBBB + LPFB with amazing VCG perform by the staff of Prof Andres Riera. Probably this delay in intraventricular conduction is impairment in the supported collagen a contact disease induced by trypanosome Cruzi and Chagasic cardiomyopathy. Amazing case.

Congratulation Prof. Andres

Samuel Sclarovsky

Queridos amigos

Este tema de las hipertrofias me ha tenido ocupado varias horas al dia los últimos 10 años

En referencia al primer ECG del joven varón con enfermedad de Marfan siempre es dificultoso diferenciar la hipertrofia fisiologica de la patológica.

Los andrógenos son hipertrofiantes, ademas de producir high take off , por el efecto de las hormonas masculinas en el metabolismo de calcio y en el ápice de la onda T, debido al control que ejercen sobre el canal de salida rectificador de potasio "slow delayed potasium channel" , y al descenso brusco de la T2, controlando el Ikr " rapid rectifier delayed channel".

Según mi experiencia el voltaje de la onda S en V2 no debe pasar de 25mm en este ECG que esta cortado para esta medida. y la R en V4 no debe pasar de 35 mm. Sin ninguna duda este paciente tiene critério de hipertrofia, muy probablemente consecuencia de insuficiencia aortica.

Me pregunto: porque la hipertrofia se expresa con un fenotipico tan raro, con profunda S en V2 y con una onda R tan alta en V4 mientra que las otras derivaciones no expresan este critérios de voltage?.

Quisiera expresar mi concepción sobre los diferentes fenotipos de la hipertrofia, basandome en información masiva de los laboratorios de ciencias básicas y de mi propia experiencia después de haber analizado personalmente mas de 200.000 ECGs desde recién nacidos hasta mas de 100 años.

Las hipertofias del ventriculo izquierdo tanto fisiológicas cuanto patológicas pueden ser divididas en simétricas y asimétricas. La distribución de los receptores endocárdicos de tensión mecánica es heterogénea, con mayor concentración - según los investigadores de ciencias básicas - en el septum y en la pared apical-lateral (esto en ratones), por lo tanto, el fenotipo de remodelación será asimétrico, manifestandose preferencialmente en el area del ventrículo izquierdo con concentracion mayor de receptores de tensión mecánica.

Pero los investigadores en ECG descubren que existe la remodelacion fisiologica simétrica y asimétrica. En el caso de sobrecargas sistolicas el ECG puede ser normal. Ejemplos de estas los vemos en pacientes con estenosis aórtica o hipertensión arterial severos con complicación renal, cerebral y retinopatia hipertensiva el ECG permanece completamente normal. De acuerdo con la ley de Laplace al reducir el radio de la cavidad disminuye la tensión de la pared. Pero esta ley no es aplicable al corazón humano porque el corazón no tiene forma de un globo ni de un tubo ,donde Laplace investigó su tesis (el corazón es un elipsóide en revolución).El ECG manifiesta unicamente las hipertrofias asimétricas.

Como explicar ECG normal ante severas sobrecargas sistólicas y diastólicas?

ECG normal con ECO que muestra hipertrofias simétricas (el ECG no muestra la hipertrofia porque los potenciales se concelan entre si. Este es el escenario de un paciente con sobrecarga sistólica. La hipertrofia reduce la tensión de la pared y no evoluciona hacia dilatación y no es aritmogénico.

En caso de ECG normal y eco normal el miocardio no pone en marcha la cascada biológica que intervienen el el proceso hipertrófico.

El ECG de este joven muestra dos áreas de hipertrofia asimétrica: En V2 la S profunda expresa la hipetrofia de la cara paseptal posterior y la onda R de gran voltage de V4 indica hipertrofia de la parte inferior del septum y de la pared libre antero-lateral y apical

El segundo ECG con el fantástico VCG muestra un BCRD asociado a BFPI. Será que esta combinación - rara en el Marfan – es resultado de mutacion que afectó el colágeno produciendo el transtorno de condución intraventricular? O será una patologia agregada como ser la tripanozomiasis Americana induciendo a cardiomiopatia Chagásica?

Profesor Andrés: el Brasil es un reservório de patologias variadas como las variaciones biológicas en la fauna y flora de vuestro maravilloso Pais.

Un fraternal abrazo todos los electrocardiófilos

Samuel Sclarovsky

Amigos e Caro Prof. Andrés ou El Potro:
Análise do ECG aos 17 anos:
Ritmo Sinusal (P precedente a todo o QRS, embora só tenhamos um ciclo completo em cada registro)
FC = não é factível.
Duração de P = 0,10; SÂP = 50º
PR = 0,16
QRS = 0,06
QT = 0,38
SÂQRS = +60º
SÂT = 40°
Conclusão: Hipertrofia Ventricular Esquerda Tipo IA

Friends and Dear Prof. Andrés or Mr. Mustang:
Seventeen's ECG analysis:
Sinus Rhythm (P before and ever each QRS, indeed only single traces)
HR = Not calculable.
Length: P = 0,10“.; P axis (SÂP) = 50º
PR = 0,16"
QRS = 0,06" SÂQRS = +60º
QT = 0,38"
SÂT = 40º

Diagnosis:
LVH Type IA
Dr. Adail Paixão - Bahia - Brazil