ECG & VCG in Right Fascicular Blocks and Incomplete Right Bundle Branch Block concept: electrovectorcardiographic criteria, proposal for classification and clinical significance - New Concepts about old problems

Andrés Ricardo Pérez Riera MD PhD

Components of the cardionector system of sinoatrioventricular & intraventricular conduction system

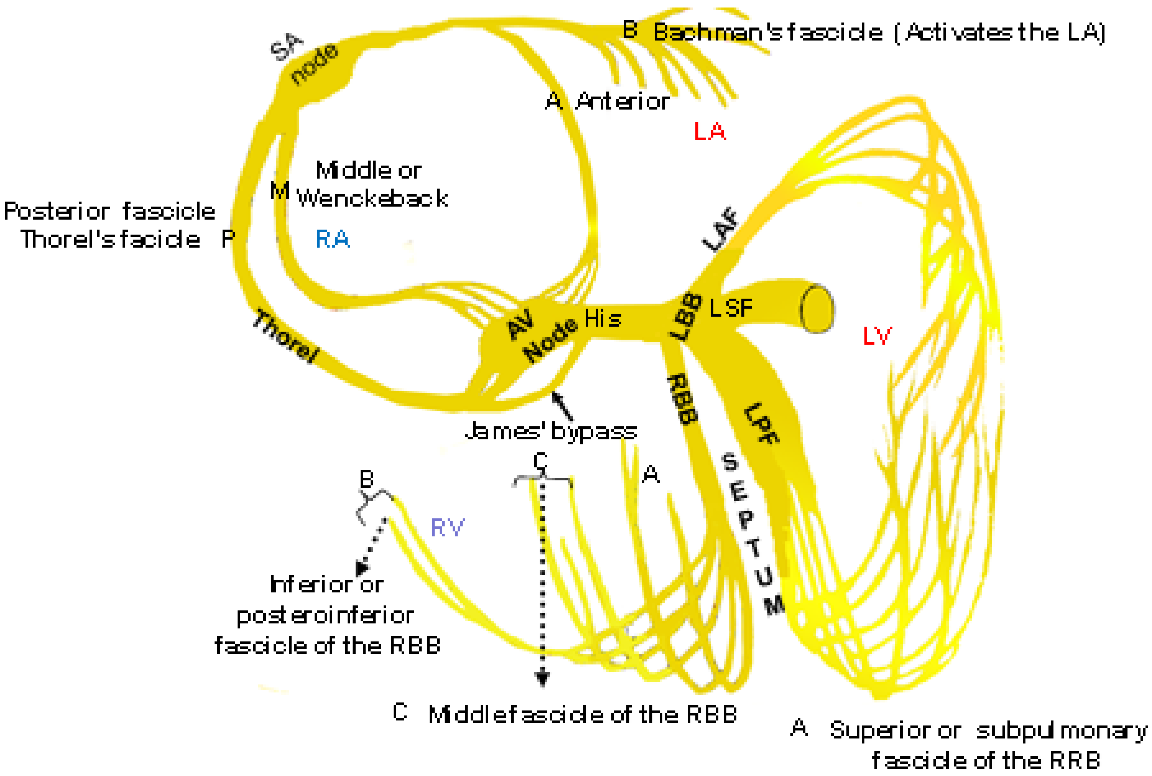

Figure 1. You can see the SA node, atrial internodal bundles (anterior, middle and posterior), AV node, His bundle and its divisions (3 left and 3 right) (Magri and Brusca 1956).

Components of the Right His System (RHS)

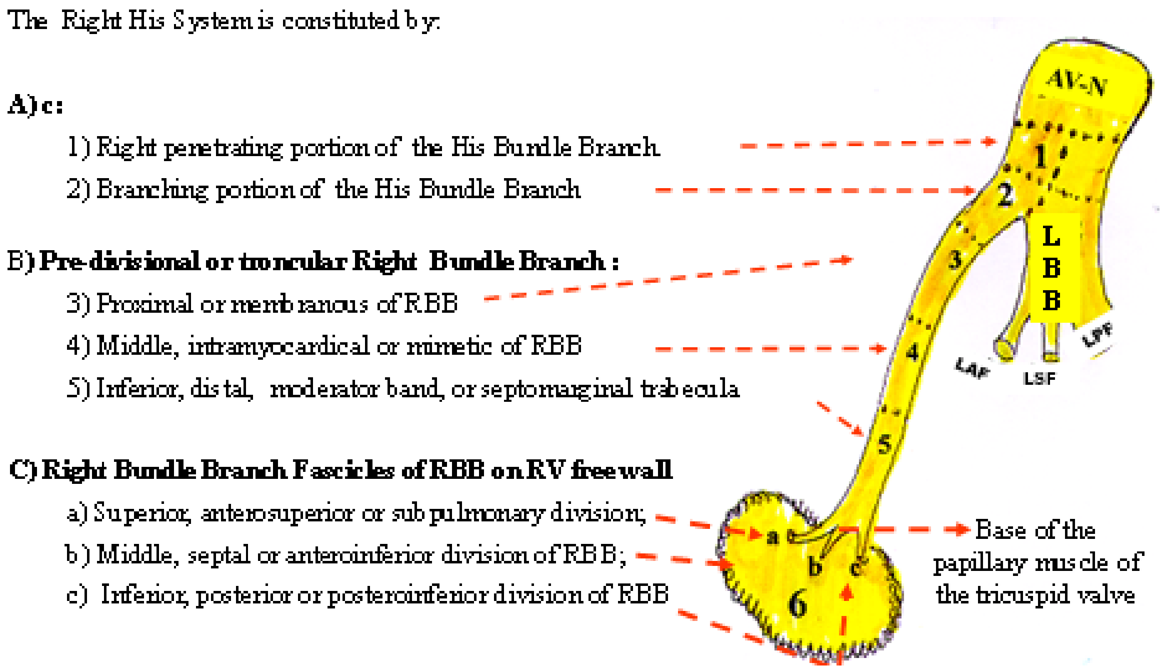

Figure 2. Components of the right his system, divided into three parts: two predivisional, and one division either terminal or divisional in the RV free wall).

Blocks of the Right Branch or of The Right His System (RHS) topographic classification

1) In the high interventricular septum: Right His Bundle (Right penetrating portion of the His Bundle Branch and Branching portion of the His Bundle Branch) and Troncular Right Bundle Branch Block;

2) In the middle and/or low septum: Peripheral Right Bundle Branch Block;

3) In the Right Ventricular free wall:

- Global or Parietal Right Bundle Branch Blocks;
- Right End Conduction Delays (RECDs), End Conduction Delays (ECDs), right bundle branch fascicular blocks, terminal, parietal, zonal, Purkinje blocks or incomplete right bundle-branch block (IRBBB). It usually is thought to be associated with abnormalities of the peripheral

Purkinje system (Liao, Emidy et al. 1986) (Barker and Valencia 1949). IRBBB may be a developmental variation in thickness of the RV free wall rather than an abnormality of the RV conduction system in cases without apparent heart disease. The developmental variant appears to have a genetic basis (Hoffman, Moore et al. 1962, Moore, Hoffman et al. 1964) (Moore, Boineau et al. 1971). If the electrocardiographic pattern of QRS prolongation up to 110 ms (in adults), with a terminal r' in V, and broad S wave in left leads V5 and V6 or standard lead I and aVL, were often the sole consequence of delayed conduction within the right bundle branch, then the term IRBBB to describe this pattern might be appropriate. Conversely, if delay in conduction in the right bundle branch is only inconsistently present in this electrocardiographic constellation, then the diagnosis of IRBBB would be at best imprecise and often incorrect (Massing and James 1972).

Concept of global Parietal His System Blocks in the Right Ventricular Free Wall

Concept: blocks resulting from the involvement of the three right branch fascicles at the same time (false tendons), secondary to parietal diffuse lesion of right Purkinje, in the RV free wall, sparing the right branch in the interventricular septum.

The lesion occurs after the division of the right branch.

Etiology: We find the typical example in the postoperative of total correction of tetralogy of Fallot (T4F) and VSD with an approach through the RV free wall (right ventriculotomy) (Scherlis and Lee 1961). The surgical incision involves globally the parietal right bundle branch in approximately 80% of cases and in 12.3% additional left anterior fascicular block following surgery (Basagoitia, Iturralde et al. 1991). We must distinguish it from CRBBB associated to LAFB, found in 7% to 25% of the cases of total correction of T4F and secondary to truncus lesion (in the septum) of the right branch, which involves concomitantly the neighboring Left Anterior Fascicle at the moment in which the surgeon sutures the patch placed with the aim of decreasing the stenosis in the RV outflow tract in the infundibulum.

1. QRS duration ≥ 120 ms

2. rSR' pattern in V_1

3. A notched S wave in lead V1 and in the right precordial accessory leads associated with a final R' wave suggests the possibility of concealed RBBB (incomplete or complete) (Zhong-qun, Nikus et al. 2014).

4. SÂQRS with extreme deviation in the superior quadrants in the FP, was attributed to lesion predominance in the Purkinje branches dependent on the superior or subpulmonary fascicle of the right branch

5. Deep S wave in DII, DIII and aVF

6. Frequently SII ≥ SIII

7. Middle-final delay of QRS complex/loop), located above and to the right.

His Electrogram in Global Parietal Right Bundle Branch Blocks

a) Normal H-V (35 ms to 55 ms): time elapsed between the His spike (H) and the onset of ventricular activation (V);

b) Normal V-RVA (5 ms to 20 ms): time elapsed between initial ventricular activation (V) and the spike of the Right Ventricular Apex (RVA);

c) Prolonged RVA-HRER (normal value = 37 ms): time elapsed between the activation of the RV Apex (RVA) and the High Right Epicardial Region (HRER).

Electrophysiological differences between RBBB according to topography

Time	Truncus	Peripheral	Fascicular
	TRBBB	PRBBB	FRBBB
H-V	Normal	Normal	Normal
V-RVA	Prolonged	Normal	Normal
RVA-HRER	Normal	Normal	Prolonged

Incomplete Right Bundle Branch Block: Electrocardiographic criteria

Incomplete RBBB is defined by QRS duration between 110 and 120 ms in adults, between 90 and 100 ms in children between 4 and 16 years of age, and between 86 and 90 ms in children less than 8 years of age. Other criteria are the same as for complete RBBB. In children, incomplete RBBB may be diagnosed when the terminal rightward deflection is less than 40 ms but greater than or equal to 20 ms.

The ECG pattern of incomplete RBBB may be present in the absence of heart disease, particularly when the V1 lead is recorded higher than or to the right of normal position and r′ is less than 20 ms. The terms rsr′ and normal rsr′ are not recommended to describe such patterns, because their meaning can be variously interpreted.In children, an rsr′ pattern in V1 and V2 with a normal QRS duration is considered a normal variant (Surawicz, Childers et al. 2009, Surawicz, Childers et al. 2009) (Camerini and Davies 1955).

Other denominations: morphology of IRBBB; right bundle branch conduction disorder; Right End Conduction Delay (RECD). If there is a pattern similar to a complete right bundle branch block but the width of the QRS complexis less than 120 ms, we consider that to be an incomplete bundle branch block pattern. Incomplete right bundle branch block is very commonly seen and can be considered a normal variant the QRS complex. This is frequently seen in teenagers and does not necessarily represent a heart disease or serious heart condition.

1) Duration of QRS: between 90 ms and 110 ms < 120 ms (if < 100 ms), normal variant predominates.
2) Triphasic pattern rSr’ in right precordial leads of the rsr’, rsR’, rSR’ type or complex in M.
3) R’ wave of V1 and V2, usually of greater voltage than initial r when pathological;
4) R’ wave of aVR, prominent and/or broad;
5) Broad S wave in left leads (I, aVL, V5 and V6);

6) Characteristic small Right End Conduction Delay (RECD) located to the right (30 or more dashes very close: 60 ms) (Pastore, Moffa et al. 1983).

Clinical significance: complete RBBB and IRBBB are two to three times more common among men than women. Complete Right bundle branch block is associated with increased cardiovascular risk and all-cause mortality, whereas IRBBB is not. Contrary to common perception, CRBBB in asymptomatic individuals should alert clinicians to cardiovascular risk (Bussink, Holst et al. 2013).

The Mexican school divides incomplete right bundle branch block in the first and second degrees according the characteristics of the final part of QRS loop of VCG in the horizontal plane. Thus, the incomplete right branch block of the first degree has only a slight change in the final portion of the QRS loop characterized by change in it rotation which is clockwise (normally it is has clockwise). This rotation change is reflected in the ECG by the presence of a notch in the final S wave of V1. At the same time, there is an increasing in duration of final S wave in left leads I, aVL, V5-V6 or an S wave appears if not exist before (Figure 3A).

In the incomplete right bundle branch block of second degree the end of the QRS loop in the horizontal plane is located on right anterior quadrant right because there is a "wave jump" and all the low septal region is abnormally activated from left to right. In these cases, the ECG pattern in V1 is manifested by a final R’ wave higher than the initial r and the final S on the left leads is wider. (Figure 3B) (Sodi-Pallares, Demicheli et al. 1964) (Sodi-Pallares and Testelli 1964).

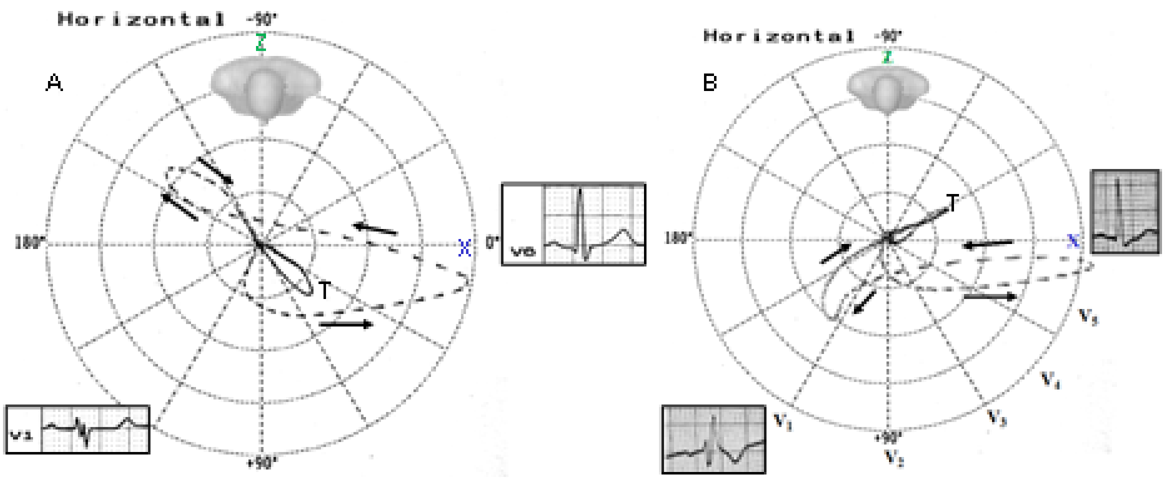

Figure 3. ECG / VCG correlation.

The causes of the triphasic pattern or morphology of IRBB in right precordial leads V1

1) **Normal variant:**

ECG abnormalities can be found in a relatively large proportion of young individuals. Incomplete right bundle branch block, left anterior fascicular block, and first-degree AV block are the most frequent findings in this population (Kobza, Cuculi et al. 2012). 3% in children, 1.5% in young people and 0.65% in adults and more frequent in athlete heart IRBBB (9%) and CRBBB (3%).

Among trained athletes, CRBBB and IRBBB appear to be markers of a structural and physiological cardiac remodeling triad characterized by RV dilation, a relative reduction in the RV systolic function at rest, and interventricular dyssynchrony (Kim, Noseworthy et al. 2011, Kim and Baggish 2015).

Also incomplete right bundle branch block, represent common variants of ECGs of athletes reflecting physiological and training-related cardiac adaptations (Pressler and Halle 2012). Normal variant with triphasic pattern on right precordial leads are characterized by:

a) r’ or R’ wave of < voltage than initial R;

b) r’ or R’ wave < 6 mm;

c) Initial R or r wave < 8 mm (0.8 mV);

d) r’ or R’ smaller than S;

e) r’ wave disappears if the exploring electrode is placed one intercostal space lower;

f) R/S ratio < 1 (Tapia and Proudfit 1960).

2) Wrong placement of exploring electrode: one intercostal space above

We must suspect inadequate placement of the electrodes in a patient with no cardiac disorder who has a negative P wave in V_1, meanelly if this is accompanied by a rSr‘ pattern. Additionally, the V_2 recording of the negative component of the P wave, on many occasions accompanied by a diphasic P wave in V1 with predominance of the negative component, is also a firm indication of the high placement of these electrodes.

3) Chest deformities:

Chest deformities are therefore associated with several electrocardiographic signs simulating most cardiac diseases. A good knowledge of these modifications should help to avoid the numerous diagnostic traps associated with these signs.

- Decrease of chest anteroposterior diameter:
    - Pectus excavatum (sunken chest):

Pectus excavatum is a deformity of the chest occurring in about one in 500 children. The deformity consists of backward displacement of the sternum and costal cartilages giving rise to a depression in the xiphisternal area.

The change in cardiac position caused by the thoracic deformity in patients with pectus excavatum produces more or less constant characteristic electrocardiographic findings, that can be suggestive of this anomaly.

These findings consist of: (a) SI-SIII or SI QIII (Pileggi, Tranchesi et al. 1961); (b) negative P waves in V1 (Martins De Oliveira, Sambhi et al. 1958) (Martins De Oliveira, Sambhi et al. 1958); (c) qr or rsr' in V1; (d) Pseudo anterior myocardial infarction: QR or small embryonic r wave from V1 to V3-V4. These findings are chiefly related to clockwise rotation of the heart on its longitudinal axis, backward orientation of atrial vector, and transmission of intracavitary potentials to the right precordial leads on account of compression of the heart. These ECG changes, can be seen also in ostium secundum atrial septal defect and mitral stenosis with pulmonary hypertension, may lead to erroneous diagnosis if the ECG is analyzed without previous knowledge of the existence of such thoracic deformity. Incomplete RBBB was attributed to pressure.

On the heart resulting form sternal deformity o hypertrophy of the infundibulo-ventricular crest and to changes in the position of the heart ECG follow-ups have been done also after surgical correction of the deformity in the sternum, and regression in R' wave amplitude in V, thereby demonstrated.

Pectus excavatum with spontaneous type 1 ECG Brugada pattern: Phenocopy

Definitions

A phenotype that is not genetically controlled but looks like a genetically controlled phenotype.

An environmentally induced phenotype that resembles the phenotype produced by a mutation.

A phenotypic variation that is caused by unusual environmental conditions and resembles the normal expression of a genotype other than its own.

Case Report: Young, male 19-year-old patient: asymptomatic, who presented at our office of an evaluation prior to the practice of sports. Negative personal and family history for syncope or sudden death in first-degree relatives younger than 45 years old.

Physical examiantion: The visual or ectoscopic test of the chest, reveals very significant Pectus excavatum with the lower third of the sternum more affected than the higher third, which was virtually normal. He mentioned that such deformity was noticed since his birth, with a progressive worsening. No first-degree relative was a carrier of pectus excavatum, Marfan syndrome, or Poland syndrome.

Cardiac auscultation: mild systolic murmur ++ in pulmonary focus. No click in the mitral valve.

Lung sounds appear diminished at both bases.

The ECG revealed spontaneous type 1 Brugada-like pattern, and several of the typical elements of pectus excavatum: completely negative P wave in V1 and V2, qR pattern from V1 to V3, and right bundle branch pattern.

The echocardiogram was normal.

The X-Ray of PA chest showed a pseudo-increase of the cardiac area and lateral projection, significant decrease of the antero-posterior diameter of the chest.

Functional respiratory test: Mild restrictive ventilatory disorder. The pulmonary volumes are reduced and there is reduction of total pulmonary capacity that indicates restrictive disorder (Mild restrictive ventilatory disorder.).

Name: BNE; **Gender:** Male; **Age:** 19 years old; **Ethnic background:** White/Caucasian; **Weight:** 65Kg; **Height:** 1,72m; **Biotype:** Normoline; **Date:** 03/4/2009.

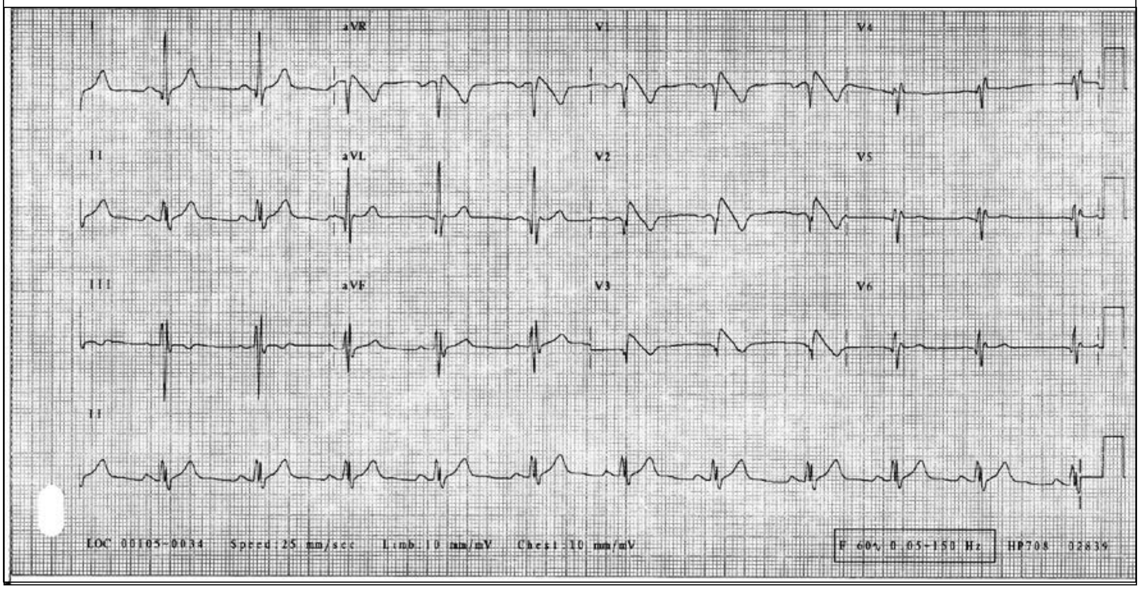

Figure 4. Clinical diagnosis: Pectus excavatum.

Electrocardiographic diagnosis

Rhythm: Normal sinus;

Heart rate: 67bpm;

P wave: P axis + 28^0 on frontal plane, entirely negative in leads V1-V2 and perpendicular to V3;

PR interval duration: 177ms;

QRS: QRSd: 122ms, QRS axis: + 60^0 on frontal plane. QRS complex: QR pattern from V_1 to V3 and absence of the normal increase of R voltage waves on precordial leads.;

ST/T: ST segment elevation coved to the top ≥ 2mm on right precordial leads and aVR lead (aVR_sign).; T axis + 28^0 on frontal plane and with negative T polarity from V1 to V3;

QT/QTc: intervals: 375/390ms.

Conclusions: Entirely negative P wave on right precordial leads. It is frequently observed in pectus excavatum consequence to right displacement of heart and modification of spatial orientation of the mean atrial activation vector. The atrial vector is oriented backwards so producing a negative P wave in right precordial leads or only in V1 leads.

Complete Right Bundle Branch Block (CRBBB): QRSd ≥120ms and QR pattern from V_1 to V_3 and absence of increase R voltage waves on precordial leads was described in pectus excavatum, secondary to rotation of the heart.

Spontaneous Type 1 Brugada ECG pattern Prominent R wave in aVR: aVR sign. A prominent R wave in lead aVR (aVR sign) is an element of risk for development of arrhythmic events in BrS. In the presence of BrS, prominent R wave in lead aVR may reflect more right ventricular conduction delay and subsequently more electrical heterogeneity, which in turn is responsible for a higher risk of arrhythmia (Babai Bigi, Aslani et al. 2007).

ECG/VCG correlation on Horizontal Plane

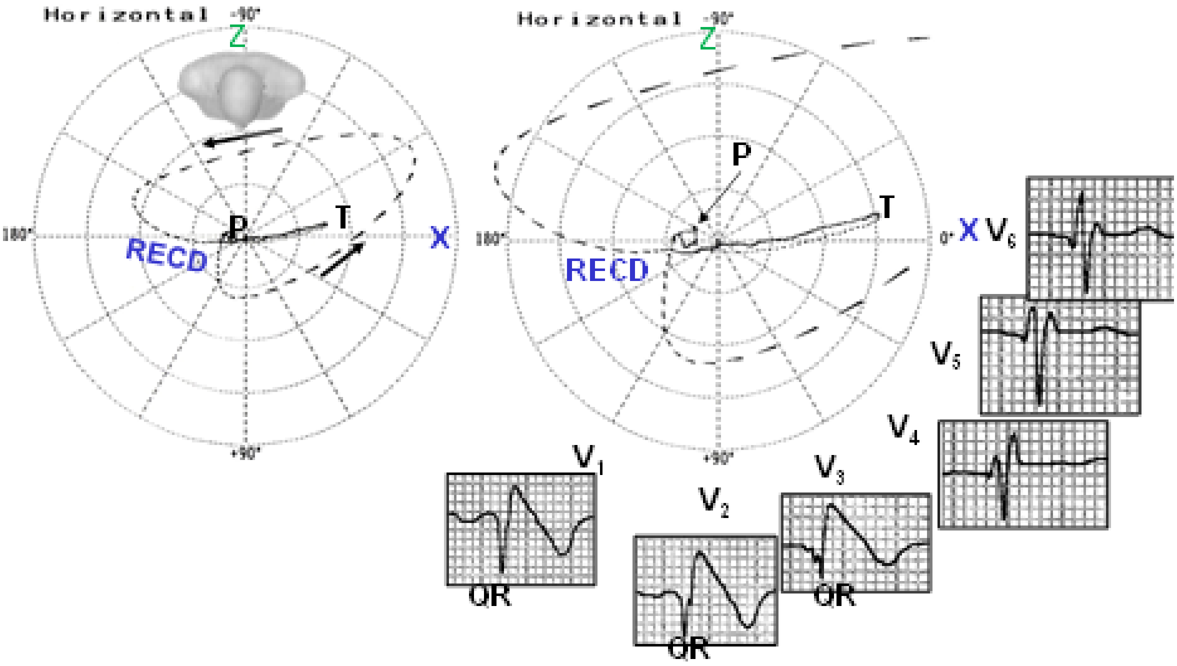

Figure 5. Brugada phenocopy ECG. (RECD: Right End Condution Delay: CRBBB)

ECG/VCG correlation on Frontal Plane

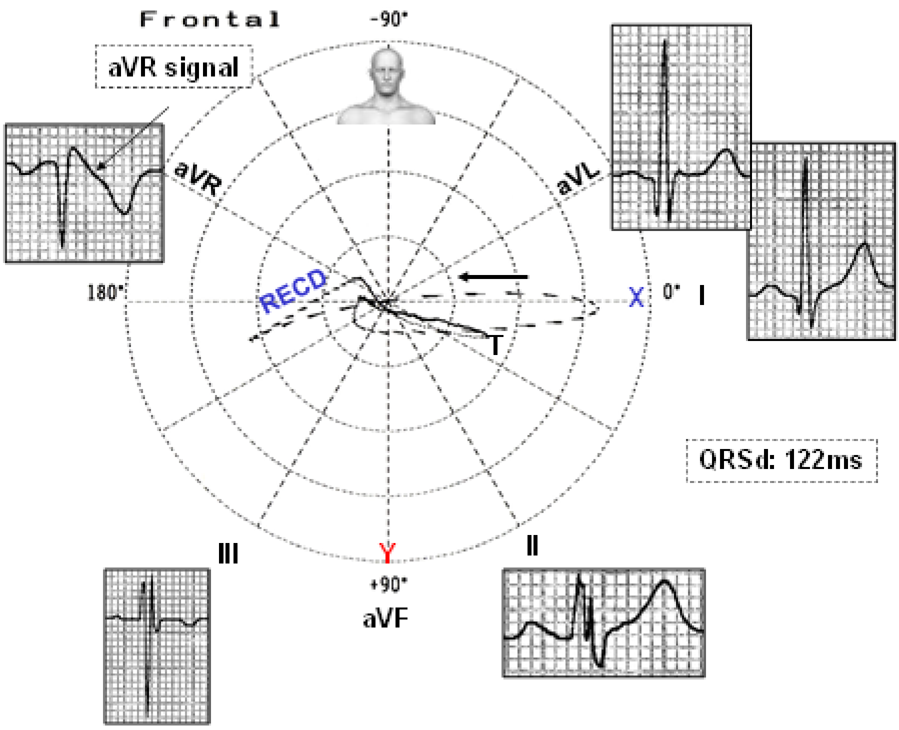

Figure 6. Brugada phenocopy ECG/VCG correlation.

ECG/VCG correlation on Right Sagittal Plane

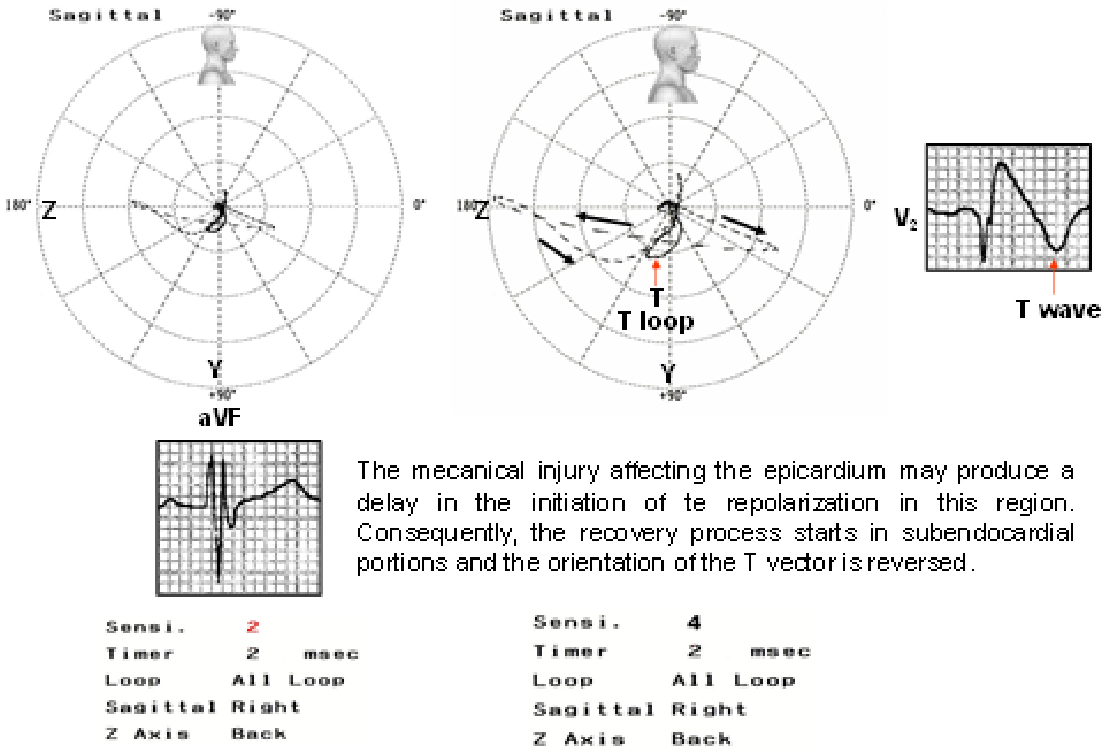

Figure 7. Brugada phenocopy ECG/VCG correlation.

Main ECG features in pectus excavatum and normal hearts

I) Negative P waves on right precordial leads: consequence of modification of spatial orientation of the mean atrial activation vector. The atrial vector is oriented backwards so producing a negative P wave in right precordial leads or only in V1 lead.

II) S_I-S_{III} or S_I-Q_{III} pattern.

III) rsr´ pattern in V1: in cases with minimal cardiac rotation, the presence of a final r´ wave may be explained by the rightward and forward deviation of the mean depolarization vector of the basal ventricular portion. This pattern in V_1 does not mean, at least as a rule, a block in the right branch of the bundle of His itself.

IV) qr or QR pattern in right precordial leads: The right atrial assumes the position directly below the exploring electrode of V_1 as consequence of a greater rotation of the heart. This lead now reflects the atrial intracavitary potentials and a qr or QR pattern appears. QR pattern can be observed in:

- Severe systolic right ventricular hypertrophy (extreme strain pattern) suprasystemic right intraventricular pressure: i.e. severe pumonary stenosis;
- Significative Right atrium dilatation i.e. Ebstein's anomaly with tricuspid insufficiency;
- Right Bundle Branch Block associated wit anterior or anteroseptal myocardial infarction;
- Right Bundle Branch Block with isoelectric initial r wave in V1;
- Situs inversus: ventricular inversion: inverted septal activation.;
- Pectus excavatum.

V) Exceptionally, Brugada type 1 pattern.

VI) Pseudo anterior MI

Chest X-ray

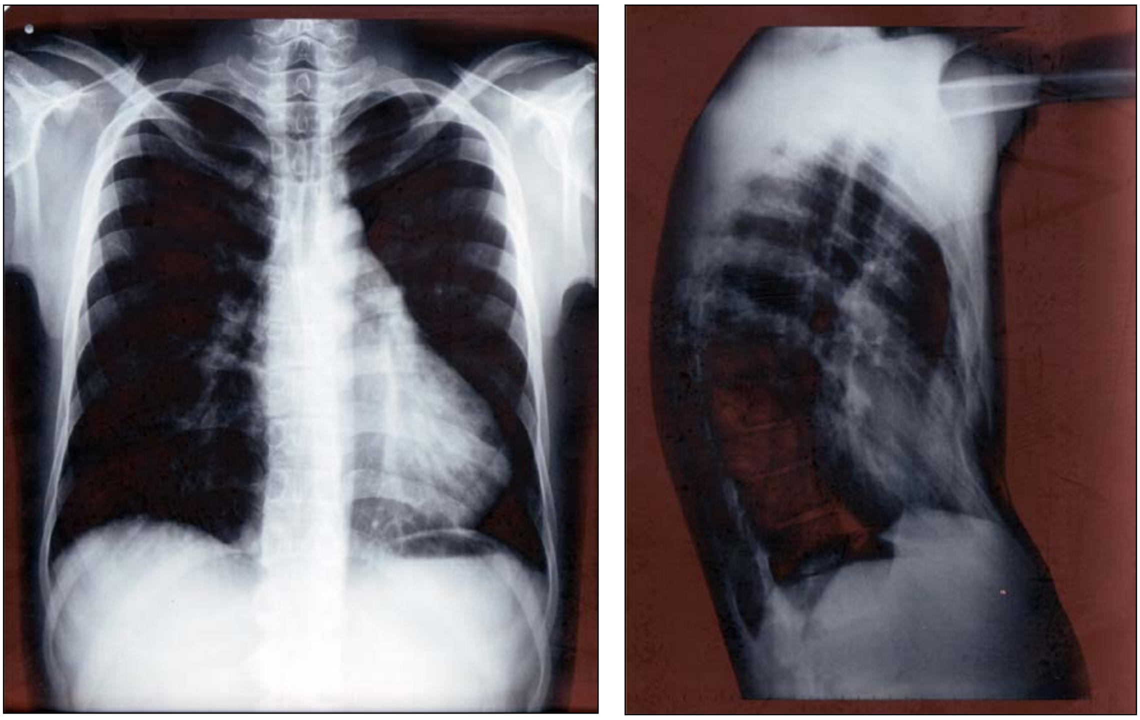

Figure 8. Lateral projection, significant decrease of the antero-posterior diameter of the chest.

Computed tomography (CT) scan of the chest

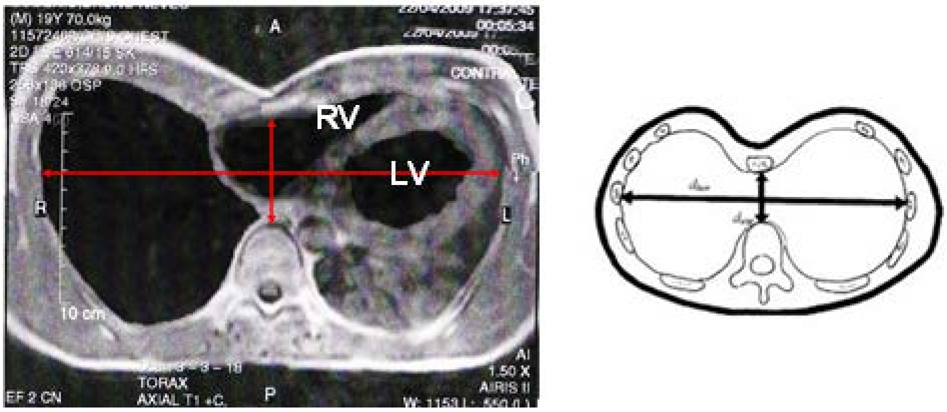

Figure 9. Haller Index > 2.5 The ratio of the transverse diameter (the horizontal distance of the inside of the ribcage) and the anteroposterior diameter (the shortest distance between the vertebrae and sternum. A normal Haller Index should be about 2.5.

b) Straight-Back Syndrome(SBS): It is a 'pseudo-heart disease' that can mimic congenital abnormalities, especially ostium secundum atrial septal defect. It typically occurs in young thin individuals who have a reduced sagittal diameter of the thoracic cage because of the absence of a normal thoracic kyphosis. SBS can cause critical tracheal obstruction and brachiocephalic artery and anterior vertebral displacement (Grillo, Wright et al. 2005). The often prominent murmur is caused by compression of the right ventricular outflow tract by the sternum and therefore is reduced with deep inspiration. Accentuated but physiologic splitting of the second heart sound and incomplete right bundle branch block in the ECG are common associated findings (Esser, Monroe et al. 2009) but ECG is normal in the majority (Ansari 1985) (Deleon, Perloff et al. 1965).

Most patients are asymptomatic; if symptomatic, chest pain and palpitations are most common. On examination, the abnormal clinical findings simulate organic heart disease that needs to be ruled out. The entity is diagnosed by physical and lateral chest radiograph findings. Radiographic computed tomographic (CT) findings are very useful for the diagnosis and the evaluation of its severity. This condition is commonly associated with mitral valve prolapse and bicuspid aortic valve.

Figure 10. X-ray.

In the PA the cardiothoracic ratio is 0.58 (upper limit of normal is 0.5) Cardiomegaly? The heart looked flattened with a prominent main pulmonary artery.

There is a slight scoliosis of the thoracic spine. I n the lateral film the most striking feature is loss of normal dorsal curvature of the thoracic spine and obliteration of the retrosternal and retro-cardiac spaces. The anteroposterior diameter of the thorax is markedly decreased such that the ratio of the anteroposterior to lateral diameter of the thorax is only 0.32.

4) Right Ventricular Hypertrophy: By selective hypertrophy of the crista or by dilatation of the right ventricular chamber: right ventricular volume enlargement and acute dilatation.

Congenital:

- Ostium secundum atrial septal defect (OS-ASD): Incomplete RBBB does not require further evaluation in the presence of a negative family/personal history and physical examination. Because incomplete RBBB is a typical ECG finding in patients with an OS-ASD, symptoms and a wide fixed split of the second heart sound associated with a grade II to II systolic pulmonary ejection murmur on auscultation should be excluded because this is typical of OS-ASD (Rodriguez and Kuzman 1968). OS-ASD is the most frequent heart defect observed in adulthood. Although it is usually non-symptomatic, it may result in heart failure, arrhythmic complications and paradoxical embolism related morbidity or mortality if the diagnosis is late. Presence of RBBB pattern, and crochetage R wave (a notch near the apex of the R wave in inferior limb leads) ECG may contribute to early detection in patients with OS-ASD.
- Ostium primum atrial septal defect OP-ASD by endocardial cushion defect (Boineau 1973),
- Ebstein’s anomaly (Kastor, Goldreyer et al. 1975),
- Partial anomalous drainage
- Mild to moderate Pulmonary Stenosis (PS),
- Tetralogy of Fallot (15%) (Depasquale and Burch 1961),
- Ventricular Septal Defects (VSDs) with pulmonary hypertension (hemodynamic groups III and IV with biventricular enlargement pattern).

Acquired:

1. Mitral stenosis
2. Chronic Cor Pulmonale,
3. Acute Pulmonary Embolism (transitory).

RVH as the cause for triphasic pattern in right precordial leads (V1 and V2) by hypertrophy of crista or congenital or acquired RV dilatation.

5) Hereditary causes

- Brugada syndrome (BrS): Type-2 Brugada ECG Pattern IRBBB or CRBBB frequently atypical, associated to upwardly convex elevation of the J point and ST segment in V1-V2 or V3 (Type I) or in “saddle-back” (Types II and III). Vectorcardiograms in patients with Brugada type 1 ECG pattern have distinctive characteristics compared with healthy individuals with incomplete and CRBBB. These differences relate to the spatial location of the end conduction delay (right superior and posterior quadrant in the BrS group) and the morphology, size, and velocity of inscription of afferent and efferent limbs of the T loop (circular, small, of symmetrical limbs) and with a 1:1 length/width ratio (Perez-Riera, Ferreira Filho et al. 2012). The classic ECG abnormalities constitute the hallmark of BrS. The last consensus report has reduced the Brugada ECG patterns to only two variants: (de Luna, Garcia-Niebla et al. 2014) (Baranchuk, Enriquez et al. 2015)
- Type 1 (coved pattern) characterized by ST-segment elevation ≥ 2 mm in right precordial leads (V1-V2) followed by a symmetric negative T wave. Only type 1 has irrefutable dignostic value.
- Type 2 (saddle-back pattern) that includes “old” patterns 2 and 3 from the previous consensus and is characterized by an r´ in leads V1-V2, followed by concave to the top ST segment elevation 0.5 mm followed by positive T wave in lead V2 and variable polarity in V1. The differential diagnosis of type-2 (Kataoka 2000, Kataoka 2002) Brugada pattern should be performed with “inocent” incomplete RBBB and other benign rSr´ patterns on right precordial leads. The ECG criteria for this purpose are:

(1) The terminal r’ wave is peaked (acute angle) in “inocent” incomplete RBBB. On the other hand type-2 Brugada ECG pattern has broad rounded final r’ wave on right precordial leads.

(2) High takeoff of r’ (that it does not coincide with J point) (2 mm).

(3) Descending arm of r’ coincides with the beginning of ST (sometimes is not well seen).

(4) Minimum ST-segment elevation (0.5 mm).

(5) T wave is usually (but not always) positive in lead V2 (T peak > ST minimum > 0) and of variable morphology in lead V1 (mildly positive, flat or mildly negative).

Angle between both arms of the r´ wave (ß angle) wider than in other cases with r´ in lead V1.

Base of triangle of r’ at 5 mm of high takeoff > 4mm (Bayes de Luna, Brugada et al. 2012) (Serra, Baranchuk et al. 2014, Serra, Baranchuk et al. 2015) (de Luna, Garcia-Niebla et al. 2014)

Mismatch between QRS duration in leads V1 and V6 (longer in lead V1) (de Luna, Garcia-Niebla et al. 2014)

Arrhythmogenic Right Ventricular Cardiomyopathy/Dysplasia (ARVD/C): it causes CRBBB or IRBBB in approximately 40% of the cases. IRBBB may be associated to ST segment elevation in V1 and V2 (Corrado, Nava et al. 1996).

Familial OS-ASD, associated to “web neck”: prolonged PR interval and/or Mobitz type I and II AV block (Kilic, Ucar et al. 2002).

Facioscapulohumeral muscular dystrophy without cardiac symptoms: Baseline ECG demonstrated incomplete right bundle branch block (RBBB) in 33%, complete RBBB in 4%, and other minor abnormalities in 16%. Comparison of incomplete right bundle branch block in facioscapulohumeral muscular dystrophy without cardiac symptoms with the normal population showed a higher prevalence of incomplete RBBB (9.7 times higher) and of complete RBBB (4.8 times higher) (van Dijk, van der Kooi et al. 2014).

6) Left Anterior Fascicular Block (may be wrongly attributed to IRBBB);In these cases, it is useful to map in right precordial leads V3R and V4R and V2h (high V2). In the cases where it is true LAFB or final R’ wave or r’ wave of V2 is greater than the one of V3R and V4R, indicating that the final forces are heading predominantly to the left.

7) By true IRBBB in middle-aged patients that may evolve into CRBBB:

A follow-up of more than 20 years showed a higher rate of cardiovascular mortality;

8) Over the course of right heart catheterization, when applying pressure on the septal surface:

In theses cases, the IRBBB is transitory and septal (not divisional or parietal).

9) Ancient denominate Dorsal, posterobasal or strictly posterior myocardial infarction:

It may show triphasic pattern of the rSr’ type, rSR’ or rsR’ in V3R and V1 simulating IRBBB in 40%!!! of the cases (Raunio, Rissanen et al. 1978). In the acute phase, it manifests by positive T waves, which are symmetrical and wide-based, accompanied by ST segment depression from V1 to V3. The phenomenon reflects the reciprocal or mirror image of the events that occur in the basal infererior wall (V7 to V9) recorded in the opposite one: V1 to V3. On the contrary, the T wave is usually negative in these leads in true IRBBB and in RVH. The appearance of Q waves of more than 40 ms in inferior leads and/or I and aVL may be another datum in favor of infarction, by the frequent association with inferolateral infarction.

]

Triphasic pattern in V_1 in basal inferior myocardial infarction

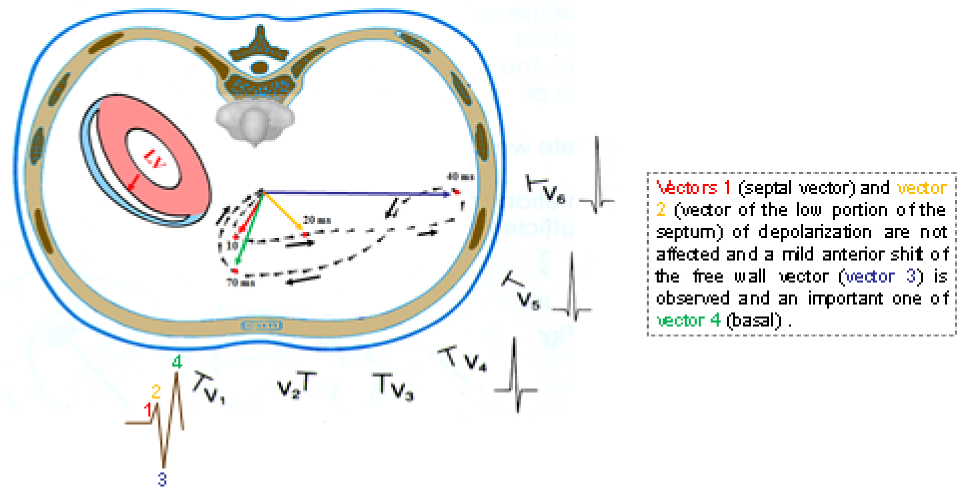

Figure 11. Basal inferior myocardial infarction affects only middle and final portions of QRS between 40 to 100 ms (the second half of the QRS loop). This abnormal anterior shift of QRS loop in the HP has at least 50% of the área in front of orthogonal X line. Triphasic QRS pattern in right precordial leads simulating IRBBB of the rSr', rSR' or rsR' type in V3R and V1 is present in approximately 40% of the cases.

Right End Conduction Delays (RECD) Zonal, Fascicular, Parietal, Peripheral, Distal or Purkinje Right Ventricular Blocks

Concept: Electrovectorcardiographic changes, secondary to physiological delay or to true dromotropic disorders in the territory of one of the three fascicles of the right bundel branch, in isolation in the RV free wall. To speak about blocking it is necessary the presence of dromotropic disorder or slowing of ventricular activation process because in its absence cannot call it so properly. These blocks cause localized or regional delay on basal portion of RV on its free wall. Zonal right ventricular blocks correspond to block of the superoanterior division of the right bundle on RV free wall (on RVOT) or inferoposterior zone (on RVIT) of the right free wall ventricle.

Others denominations Search: Parietal focal blocks (Masini, Testoni et al. 1952) (Rossi 1954) (Alzamora-Castro, Abugattas et al. 1953); Right focal blocks; Peripheral branch block of the right bundle; Peripheral blocks of the right branch; Right peripheral fascicular blocks (Pastore, Moffa et al. 1983); Right peripheral blocks; Distal right bundle block; Divisional blocks of the right branch; Fascicular block of the His bundle; Delayed activation of the wall of the Right Ventricle.

Electrocardiogrfically are characterized by QRS duration is < 120ms, frequent absence of evident final broad r' in V1 (Uhley and Rivkin 1961).

Blocks of the superoanterior division of the right bundle on RV free wall or block of the superoanterior zone produce prolongation of ventricular activation time, R peak time or intrinsecoide deflection on aVR, V3R and eventually in V1 (VAT = 50 ms, maximum normal = 40 ms), additionally, frequently SI-SII-SIII pattern indistinguishable from the positional SI-SII-SII or the one produced by right ventricular enlargement (Bayes de Luna, Carrio et al. 1987), QRS axis on superior quadrants (above and between ± 180º and 0º). Concomitantly, there are slurred of S waves in leads to "face" the opposing regions. This is the block observed constantly in Brugada syndrome (de Micheli, Medrano et al. 2009).

Experimentally, it has been demonstrated that these blocks result from a peripheral delay of the stimulus localized in a certain right ventricular zone on RV free wall.

Distribution of the three fascicles of the right branch of the His bundle in the RV free wall (Lev 1964) (Lev 1968)

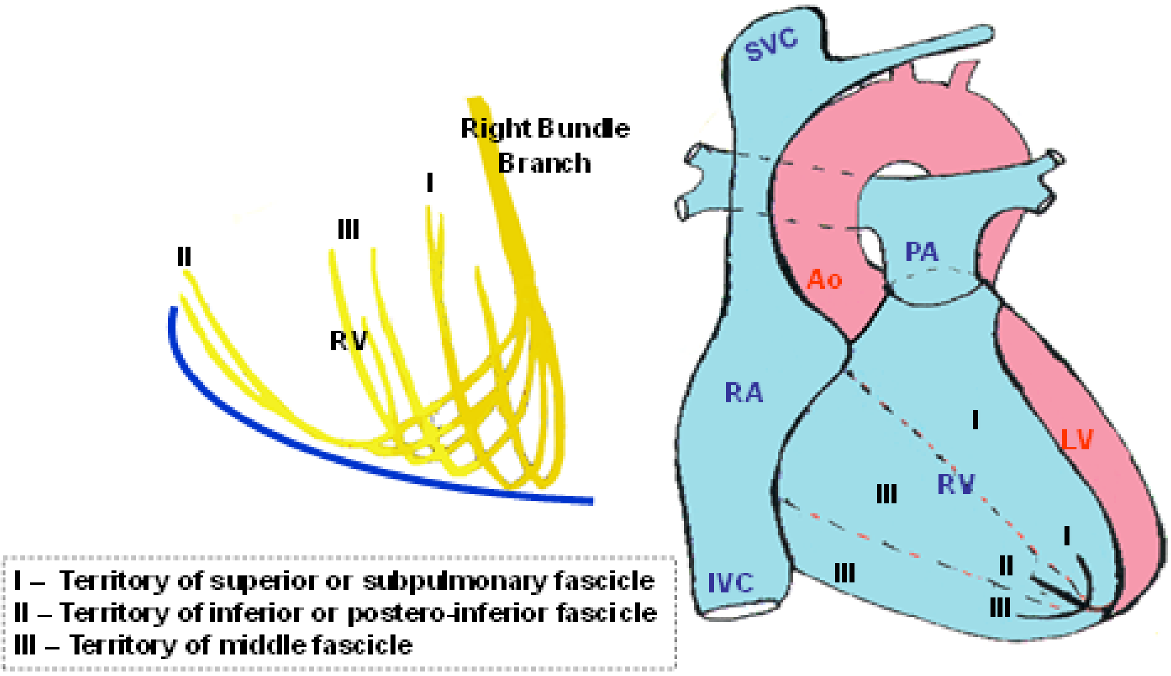

Figure 12. Distribution of three divisions of the right branch in the RV free wall.

Frontal Plane: Location of End Conduction Delay (RECD) in the 3 types

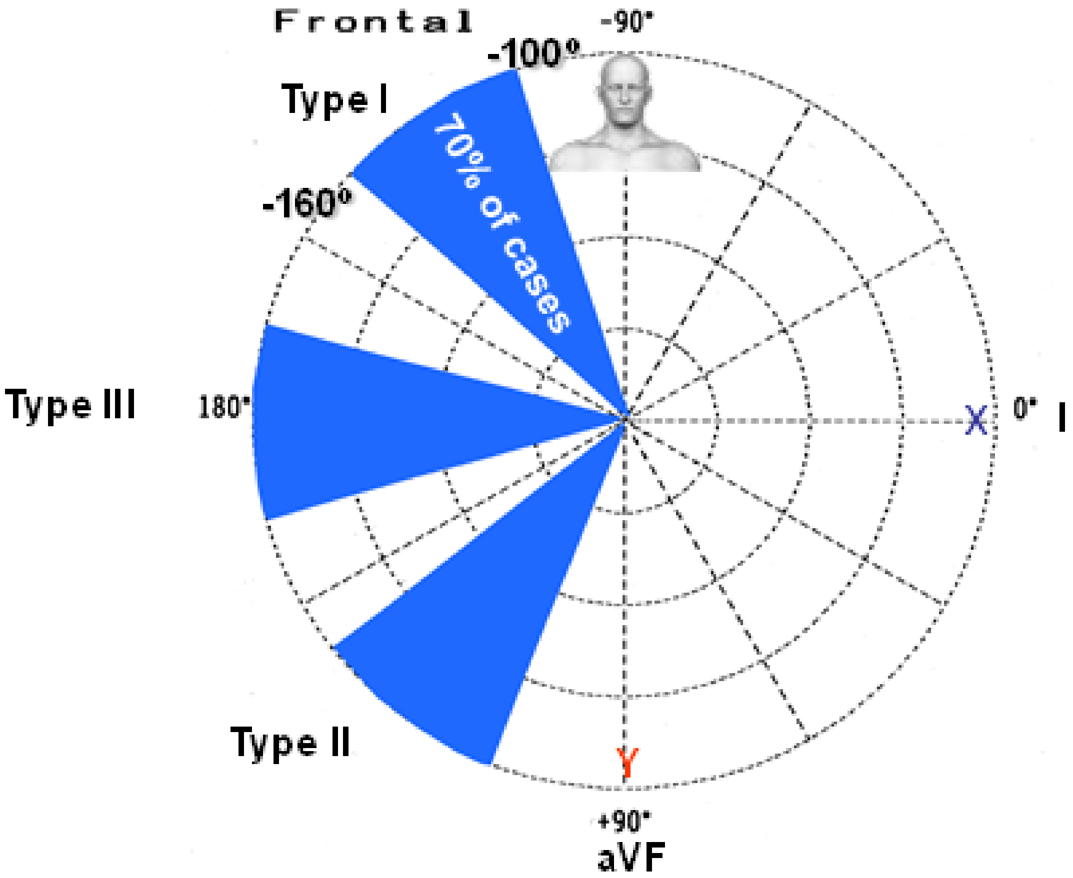

Figure 13. Classification of RECD, taking into account the location of the end delay of the QRS loop in the frontal plane in Type I, II and III. New proposal of VCG classification.

New proposal of VCG classification of RECD according to QRS loop in the FP

1) Type I or right anterior subdivision block (de Micheli and Medrano 1987)

a) Type IA: QRS loop predominantly located in the left superior quadrant, (SÂQRS with extreme deviation to the left), counterclockwise rotation and RECD located in the right superior quadrant. Very similar to LAFB;

b) Type IB: QRS loop pointed, clockwise or in eight, with the initial portion located in the left inferior quadrant and RECD located in the right superior quadrant. SÂQRS difficult to determine or shifted to the right;

c) Type IC: QRS loop of clockwise rotation with SÂQRS with no deviation or with a mild shift to the right. In the three types with RECD located in the right superior quadrant;

VCG Type I RECD variants according to QRS rotation on FP

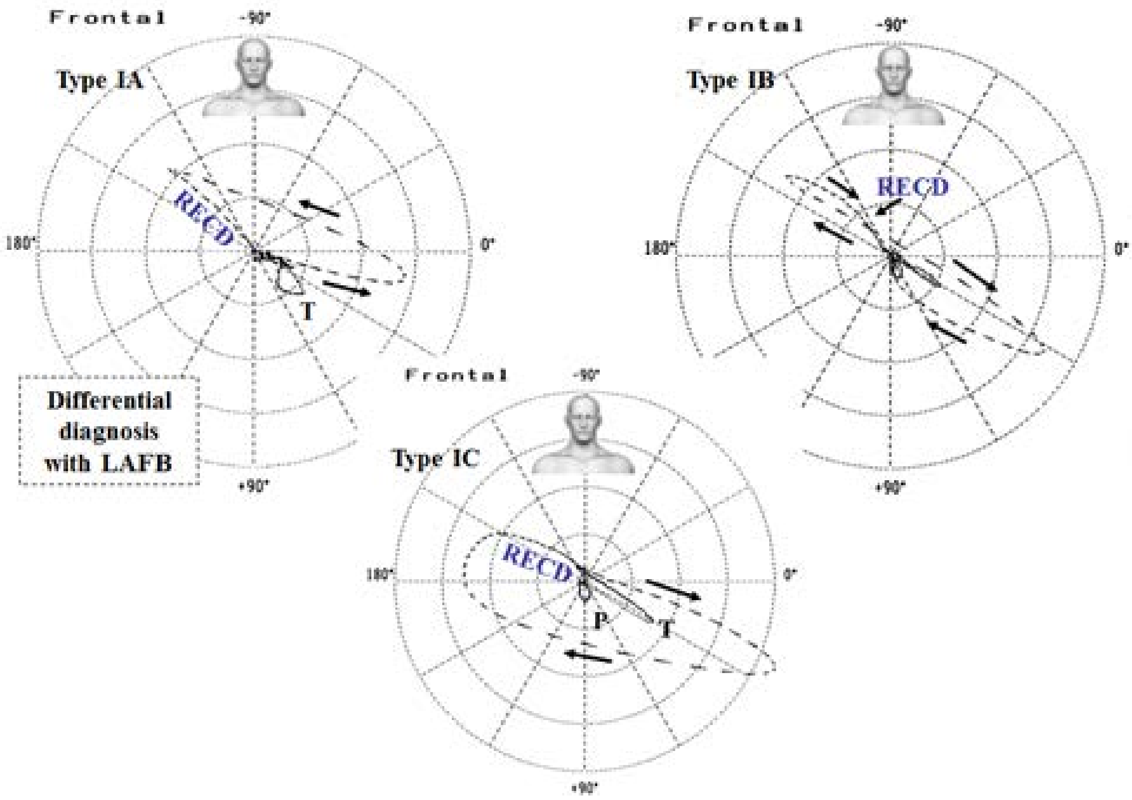

Figure 14. Vectorcardiographic loop in the frontal plane of the three subtypes of Type I. It is clear that only type IA may be confused with LAFB.

Vectocardiographic classification of RECD according to QRS loop in the FP

Other types:

- Type II: RECD located in the right inferior quadrant, in the territory of the inferior fascicle of the right branch;
- Type III: RECD located on the right portion of the 0±180° line, corresponding to the territory of distribution of the middle fascicle of the right branch;
- Type IV: RECD located in the line of – 90° (neither to the right nor the left). Does it exist?

RECD type I

Characterized by presenting end conduction delay (RECD), located in the right superior quadrant of the FP, corresponding to the territory of the superior or subpulmonary fascicle of the right branch, in the site of the RV outflow tract between -100° and 160°.

The location of the delay justifies the recording of prominent R waves with a certain delay in the lead that faces the RV outflow tract: aVR. Additionally, notched in the apex of the R wave, which prolongation of ventricular activation time, R peak time or intrinsicoid deflection (≥ 50 ms) circunscript only in the right unipolar lead that explore the right ventricular outflow tract affected region (aVR). The superior infundibular subpulmonary region of the RV is the last one to activate, generating a final basal vector (basal vector 3d), heading upward and to the right between -100º and -160º in the FP.

This is the most frequent variant of RECD (70% of all of our cases). By the aspect and rotation of the QRS loop in the FP, we propose to divide the RECD Type I into: RECD type IA; RECD type IB and RECD type IC.

RECD type IA

1) SÂQRS with extreme deviation in the left superior quadrant between -30º and -90°;
2) QRS loop of counterclockwise rotation in the frontal plane;
3) Rapid passage from left to right of the QRS loop;
4) Discrete RECD of 30 ms (15 dashes) located in the right superior quadrant between – 100° and – 160°; or greater.
5) QRS complexes predominantly negative in inferior leads: prominent S wave in these leads;

6) SII>SIII: useful for the differential diagnosis with LAFB;

7) R wave of aVR prominent and/or broad. aVR of the qR or QR type, with R being frequently broad.

Typical case of RECD type IA showing ECG/VCG correlation in the Frontal Plane

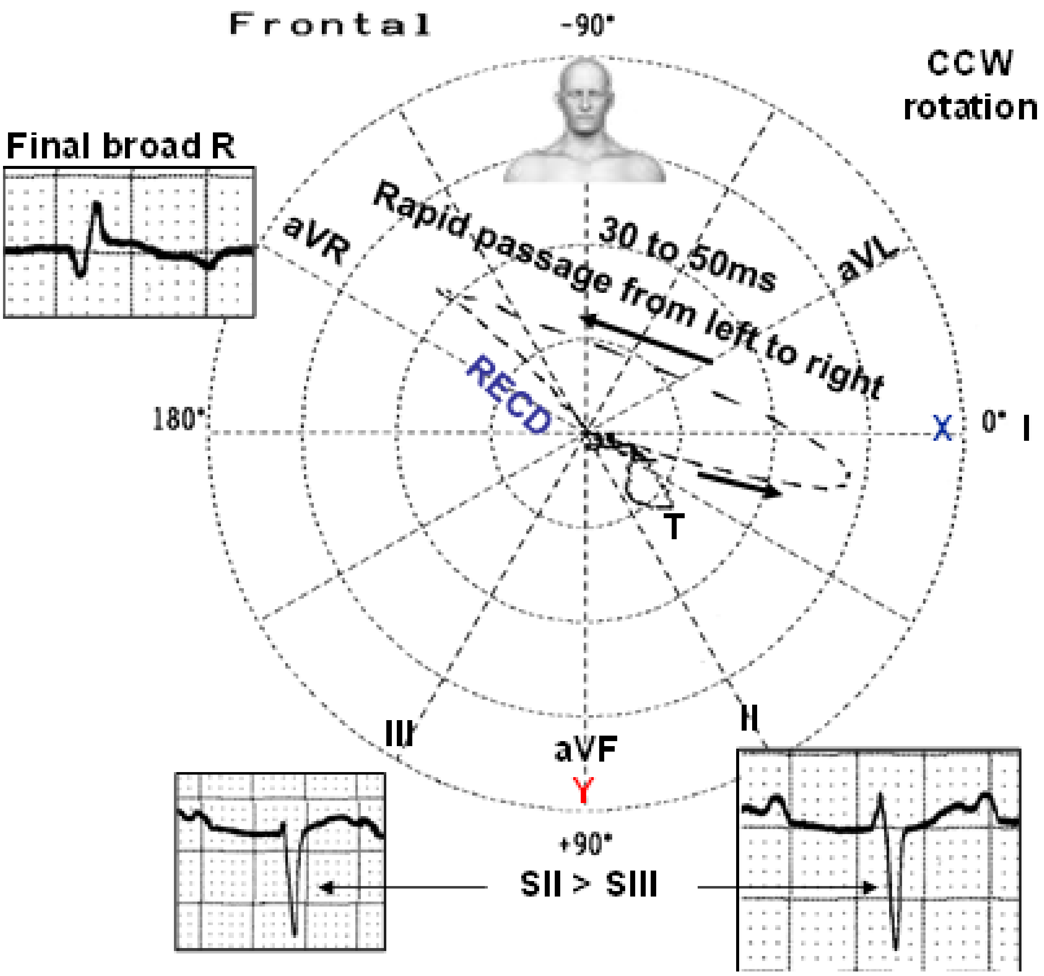

Figure 15. Typical case of RECD type IA showing ECG/VCG correlation in the Frontal Plane.

Differential diagnosis between RECD type IA and LAFB

	RECD type IA	LAFB
Depth of S wave in II and III	SII > SIII (inconstant)	SIII > SII (inconstant)
I and aVL	Rs	Qr
Prominent and broad R wave in aVR	Present and characteristic: QR or qR.	Absent: Qr or QS.
Vector of initial 10 to 20 ms	Downward and to the left (inconstant)	Downward and to the right. (inconstant)
Rapid passage from left to right	Yes	No
RECD	In the right superior quadrant.	With or without delay, above and to the left.
Triphasic pattern in V_1 or V_1 and V_2.	Very frequent.	Possible. Final R' wave or r' wave of V_2 is greater than in V_3R and V_4R, indicating that the final forces are heading predominantly to the left.

Criteria for differential diagnosis between RECD type IA and LAFB.

ECG/VCG correlation between RECD type IA and LAFB

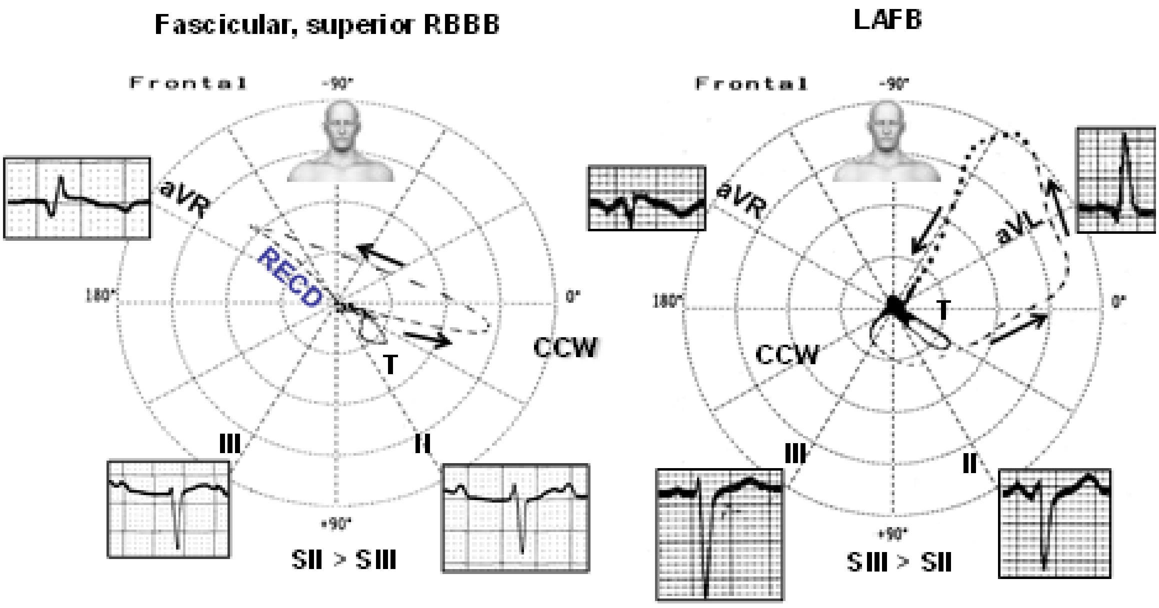

Figure 16. ECG/VCG correlation.

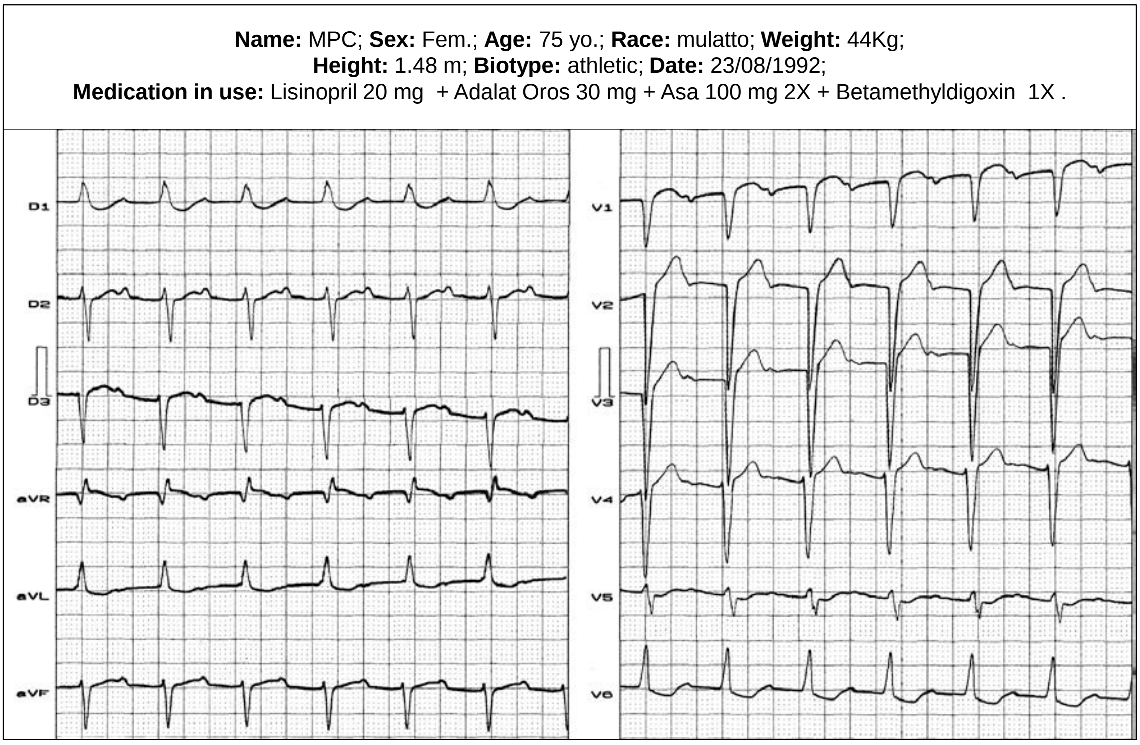

Figure 17. Electro-vectocardiogram of a typical case of RECD type IA.. Clinical diagnosis: Hypertensive heart disease. Echocardiogram: Concentrical myocardial hypertrophy. ECG diagnosis: Extreme deviation of SÂQRS –65º. Cause? + 1st degree AV block, LVE + digitalis action.

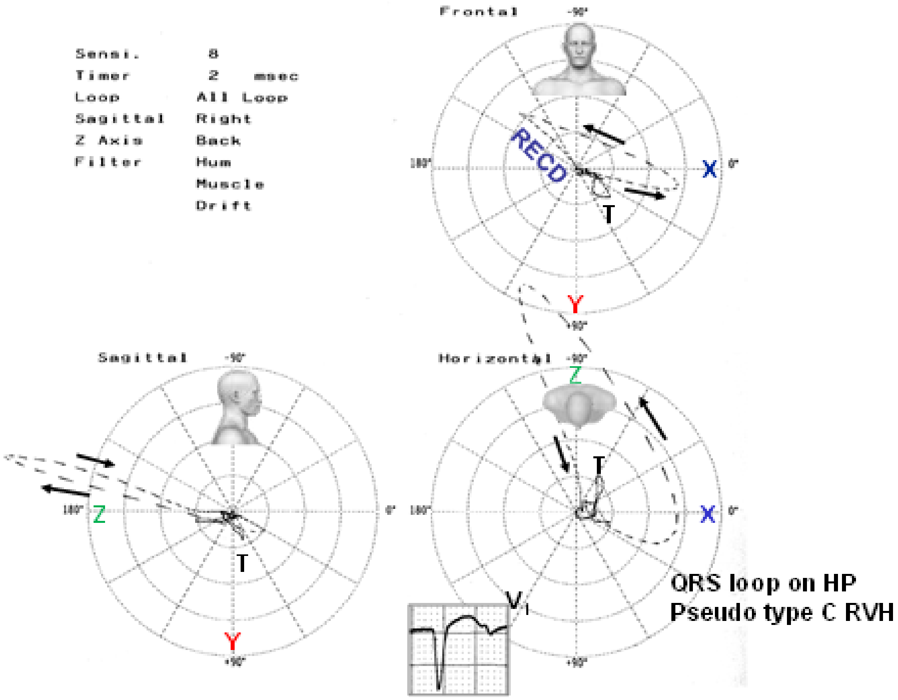

Figure 18. Electro-vectocardiogram of a typical case of RECD type IA.

ECG/VCG correlation on FP - RECD type IA

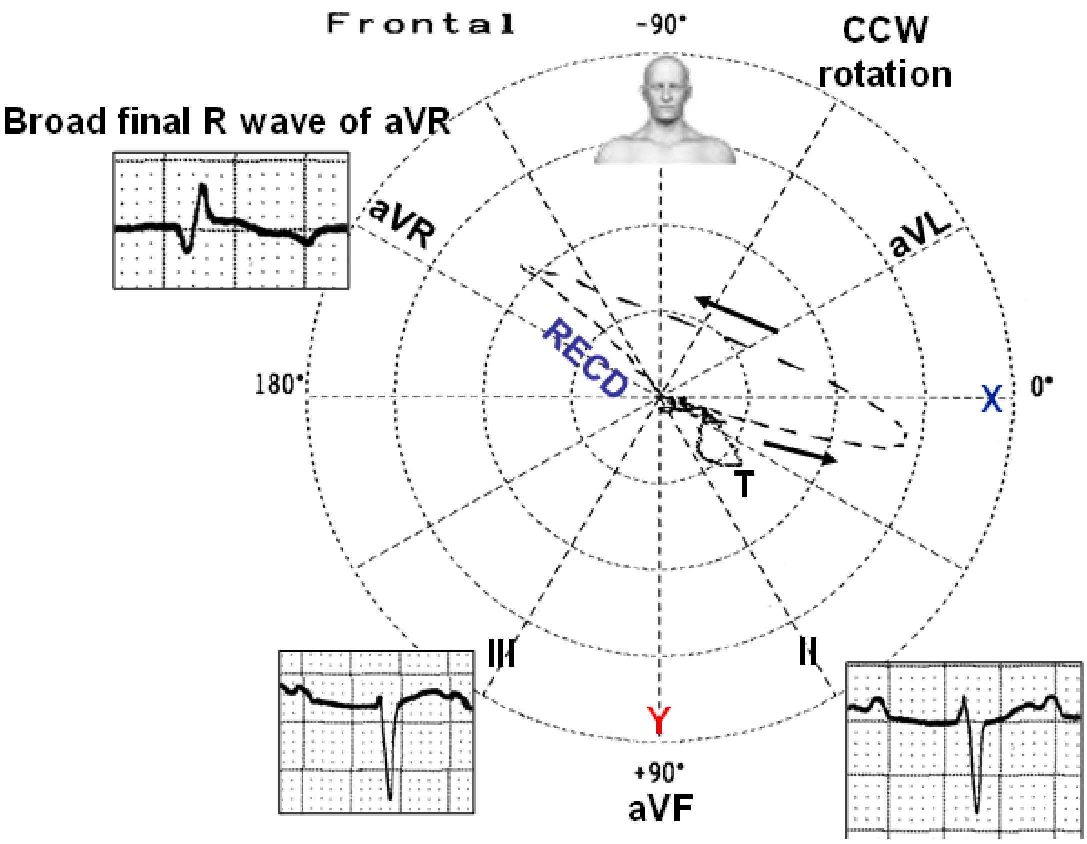

Figure 19. RECD Type IA: QRS loop of predominant location in the left superior quadrant (SÂQRS with extreme deviation to the left), rapid passage from left to right, counterclockwise rotation, RECD located in the right superior quadrant, broad final R wave of aVR and SII > SIII. Very similar to LAFB.

Name: OCA; **Sex:** Fem.; **Age:** 90 yo.; **Race:** White; **Weight:** 47 Kg; **Height:** 1.54 m; **Biotype:** Athletic; **Date:** 20/08/2003; **Medication in use:** Amlodipine Besylate, Hydrochlorothiazide

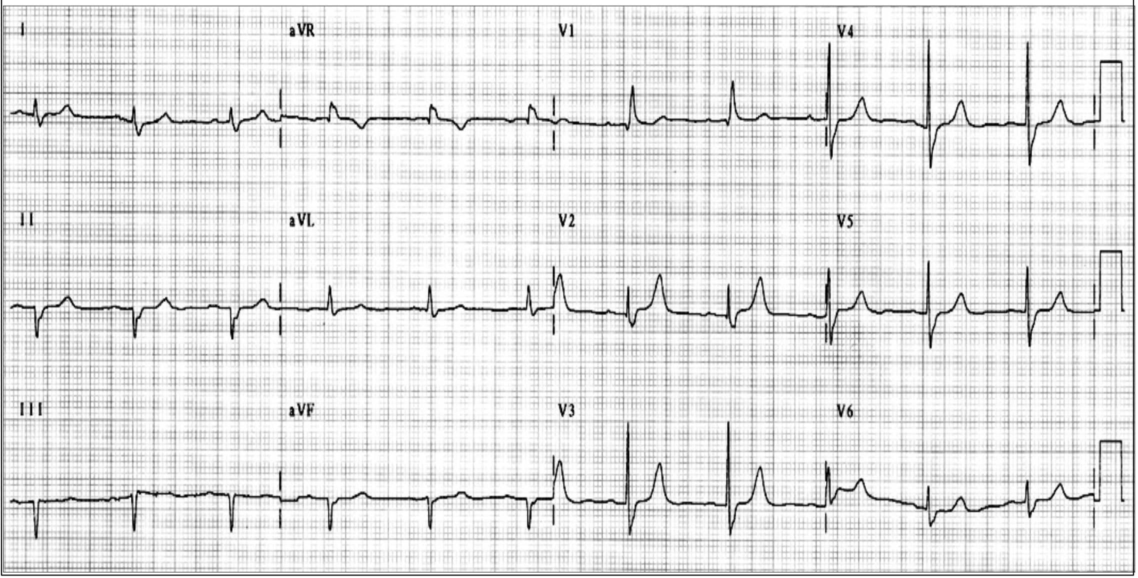

Figure 20. Electro-vectocardiogram of a typical case of RECD type IA with pseudo-inferior infarction and LAFB. Clinical diagnosis: systemic hypertension. Echocardiogram: decrease of LV compliance, thickened aortic valve without stenosis. ECG diagnosis: SR; HR: 65 bpm; P wave SAP + 23º to the front, duration of 80 ms; PR interval: 198 ms; SÂQRS: near -90º; QRS duration: 105 ms. Initial r wave in inferior leads (possible electrically inactive area in inferior wall).

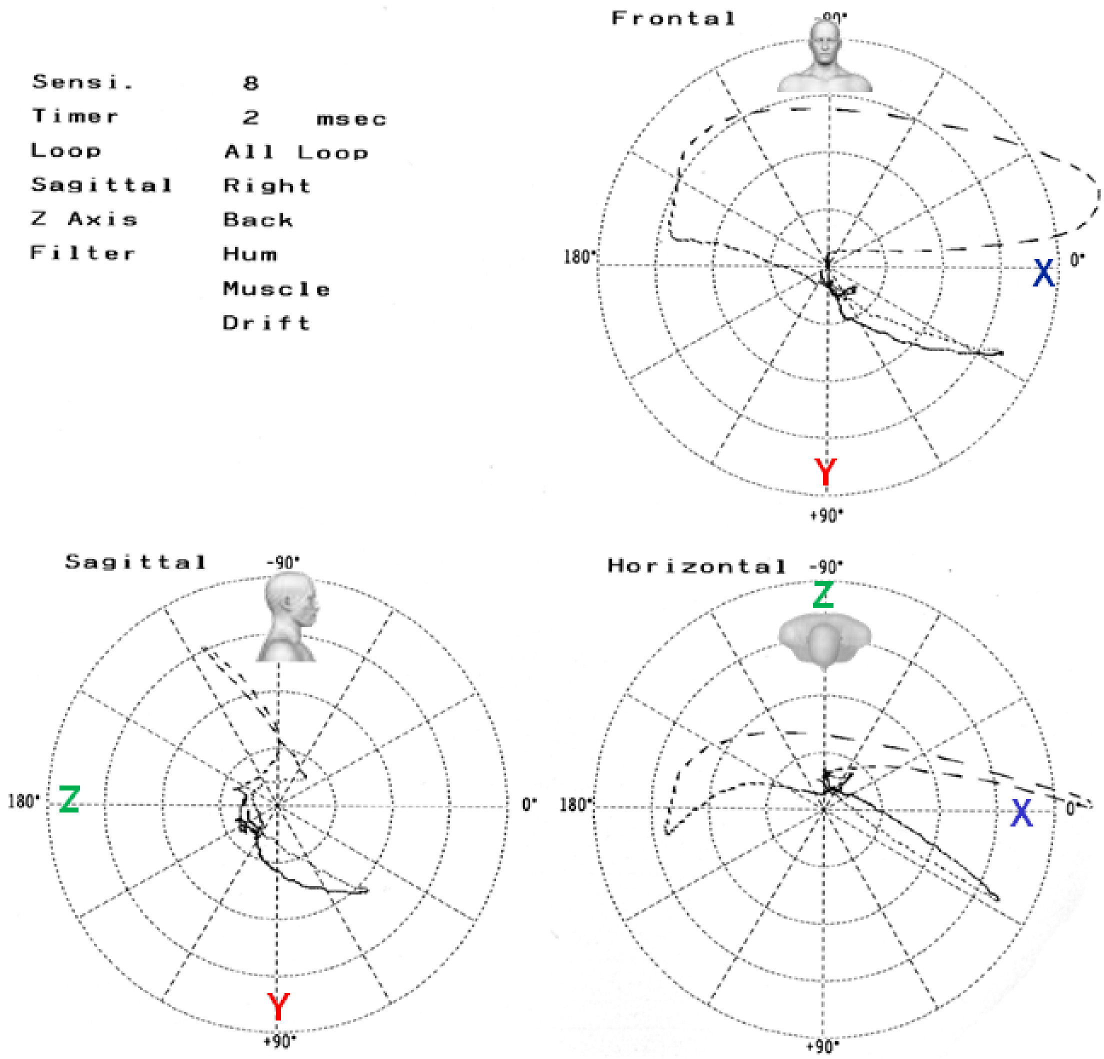

Figure 21. Electro-vectocardiogram of a typical case of RECD type IA with pseudo-inferior infarction and LAFB.

ECG/VCG correlation on FP - RECD type IA with pseudo-inferior infarction and pseudo LAFB

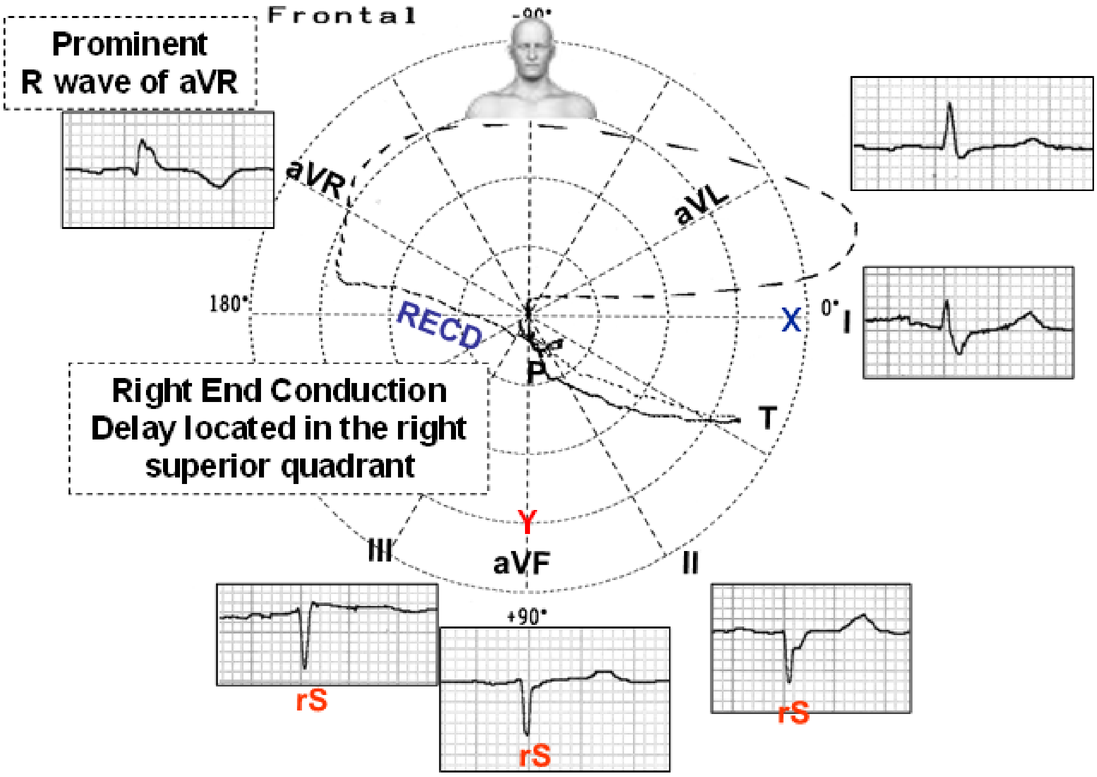

Figure 22. Extreme deviation of SAQRS in the superior quadrants, close to –900. Counterclockwise rotation. Inferior wall: pattern of initial r wave S (rS). There might be a suspicion of old electrically inactive area in inferior wall. Conclusion: RECD type IA, pseudo-inferior infarction associated to LAFB. The echocardiogram ruled out segmentary disease in inferior wall.

ECG/VCG correlation on HP

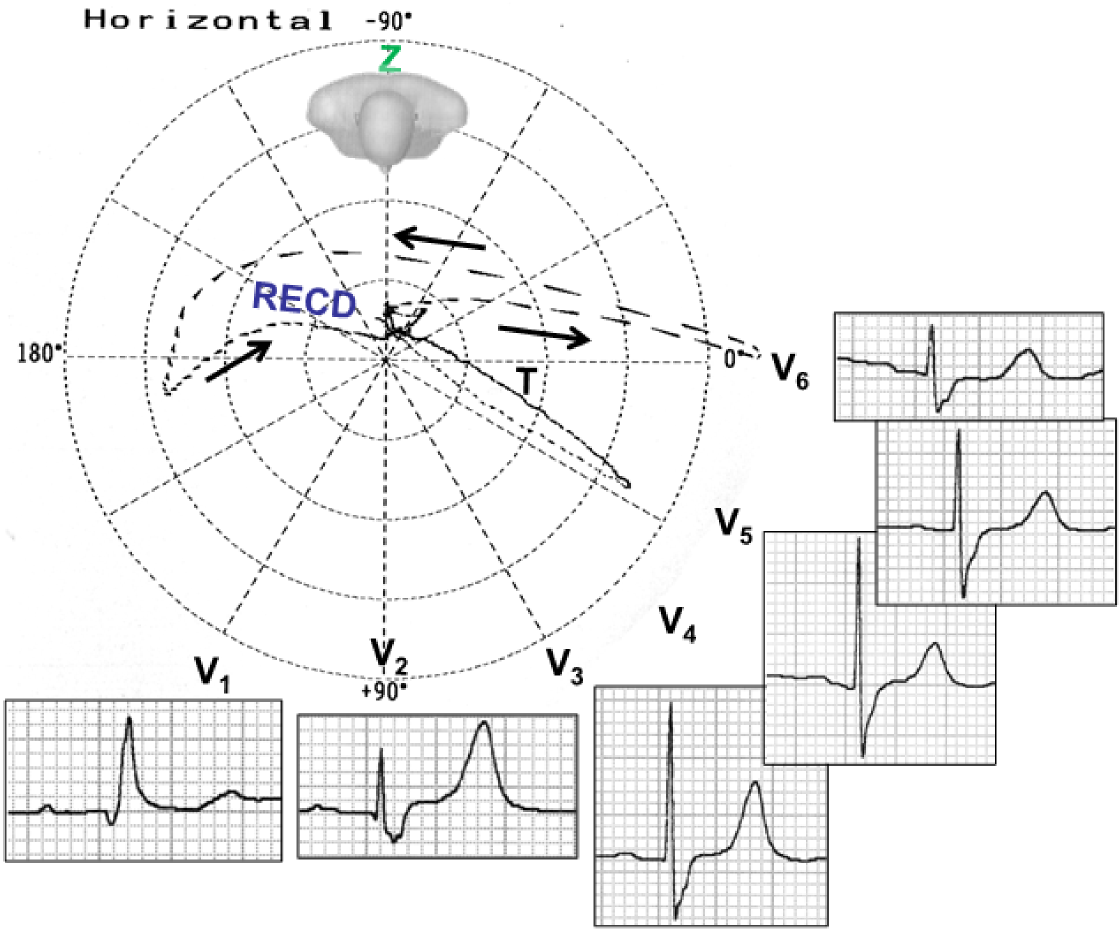

Figure 23. Pattern of qR in V1 may suggest CRBBB associated to septal fibrosis; however, the duration of QRS is only 105 ms. Initial vector heading to the back and the left, which explains initial q wave in V1-V2. Conclusion: RECD by one of the fascicles of the right branch.

RECD type IB

Electrovectocardiographic characterization

1. SÂQRS hard to determine: indeterminate or perpendicular to the frontal plane, suggesting the presence in all the leads of the FP, of isodiphasic complexes that make the determination of SÂQRS difficult;

2. QRS loop pointed or in eight in the FP, with the initial portions located in the left inferior quadrant and the final ones in the right superior quadrant, where you find the RECD;

3. The aspect of QRS loop is similar to the propeller of a plane;

4. Sagittal plane clearly shows QRS loop perpendicular to the Y line;

Name: ANFC **; Sex:** F; **Age:** 10 yo; **Race:** white; **Weight:** 43 Kg;
Height: 1.54 m; **Biotype:** asthenic**; Date:** 17/01/2000

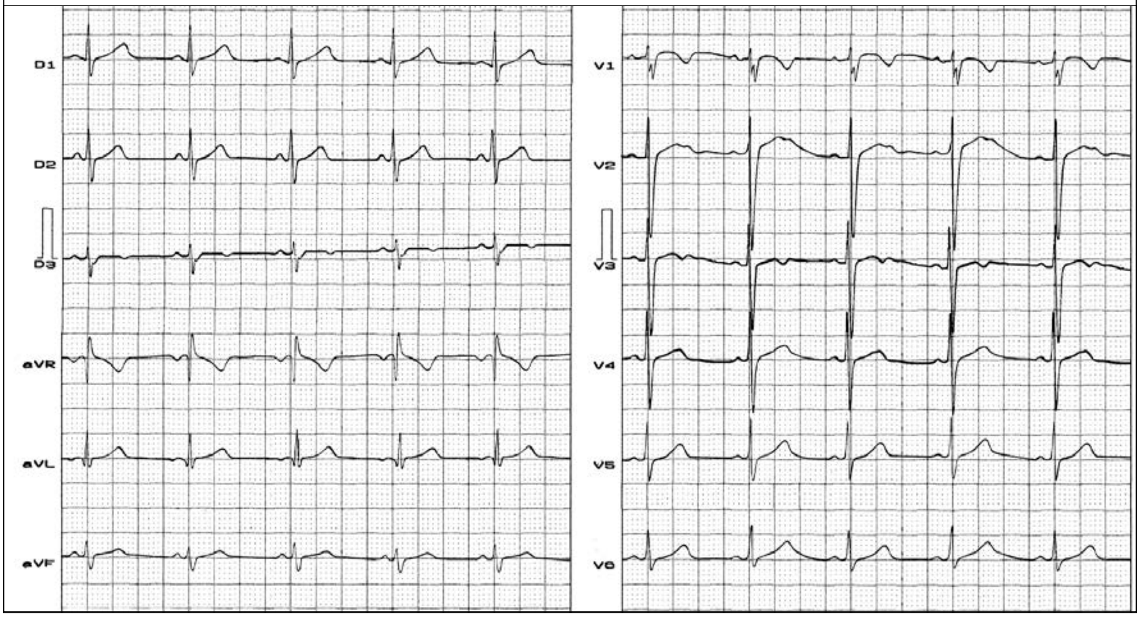

Figure 24. Clinical diagnosis: ASD-OS with minimal hemodynamic repercussion, aneurysm of the interventricular septum, sequel from spontaneous closing of VSD. ECG diagnosis: SÂQRS hard to determine, tendency to be perpendicular to the frontal plane. SI-SII-SIII: prominent and somehow broad R wave of aVR, V1: rS with notch: morphology of atypical IRBBB. RECD. Electro-vectocardiogram of a typical case of RECD type IB.

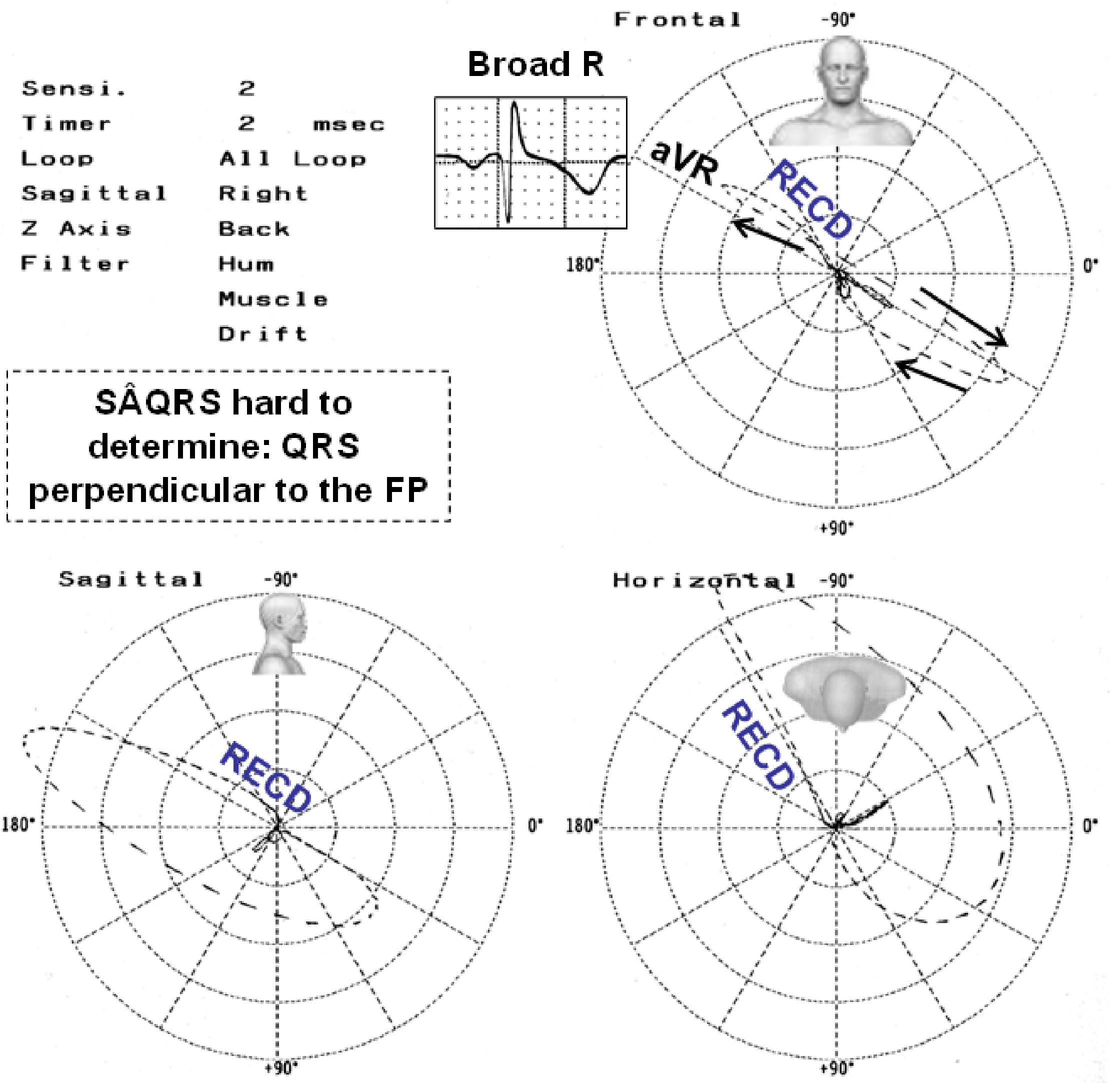

Figure 25. Electro-vectocardiogram of a typical case of RECD type IB. RECD type IB: QRS loop pointed, in eight with clockwise initial portion in the left inferior quadrant and final counterclockwise portion with RECD, in the right superior quadrant. SÂQRS hard to determine.

RECD type IC

Electrovectocardiographic characterization

1) SÂQRS not deviated or deviated to the right;
2) Pointed aspect of QRS loop in the frontal plane
3) QRS loop of clockwise rotation in the frontal plane;
4) RECD in the right superior quadrant in the frontal plane;
5) Frequent SI-SII-SIII;
6) Broad R wave of aVR;
7) Morphology of IRBBB: rSR’ in V_1.

Name: FCS; **Sex:** M; **Age:** 21 yo; **Race:** White; **Weight:** 72 Kg;
Height: 1.70 m**; Biotype:** Athletic**; Date:** 8/04/2003

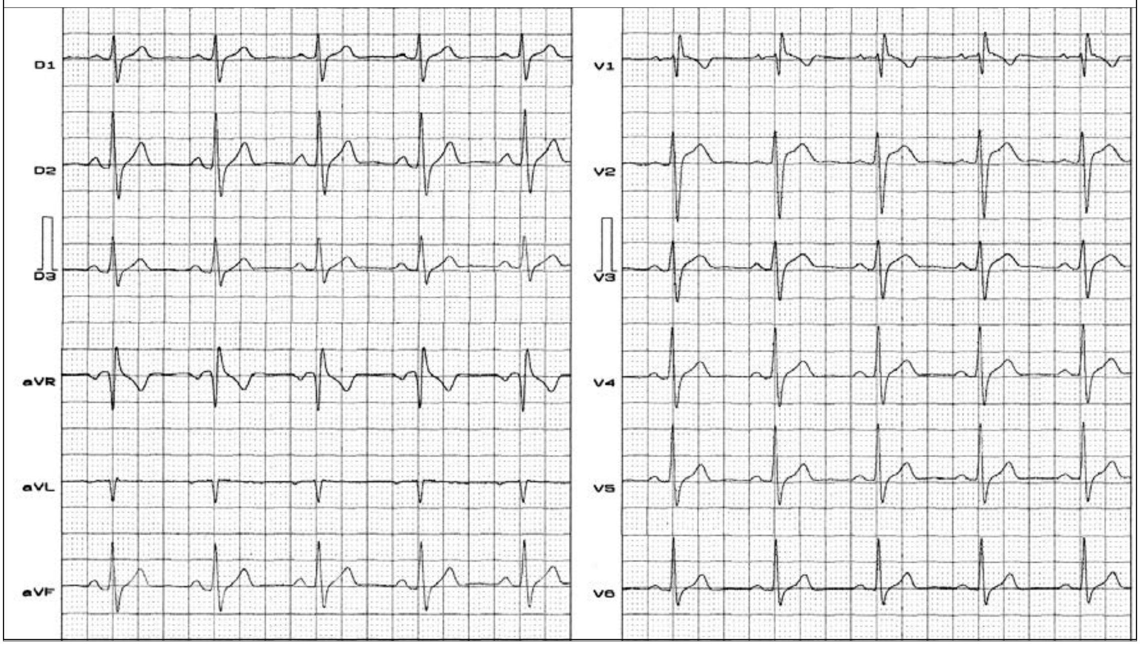

Figure 26. Electrovectocardiogram of two typical cases of RECD type IC. Clinical diagnosis: mitral valve prolapse, competent and minimal. Normal variant. ECG diagnosis: SÂQRS slightly shifted to the right +110°; right bundle branch conduction disorder: RECD, normal variant.

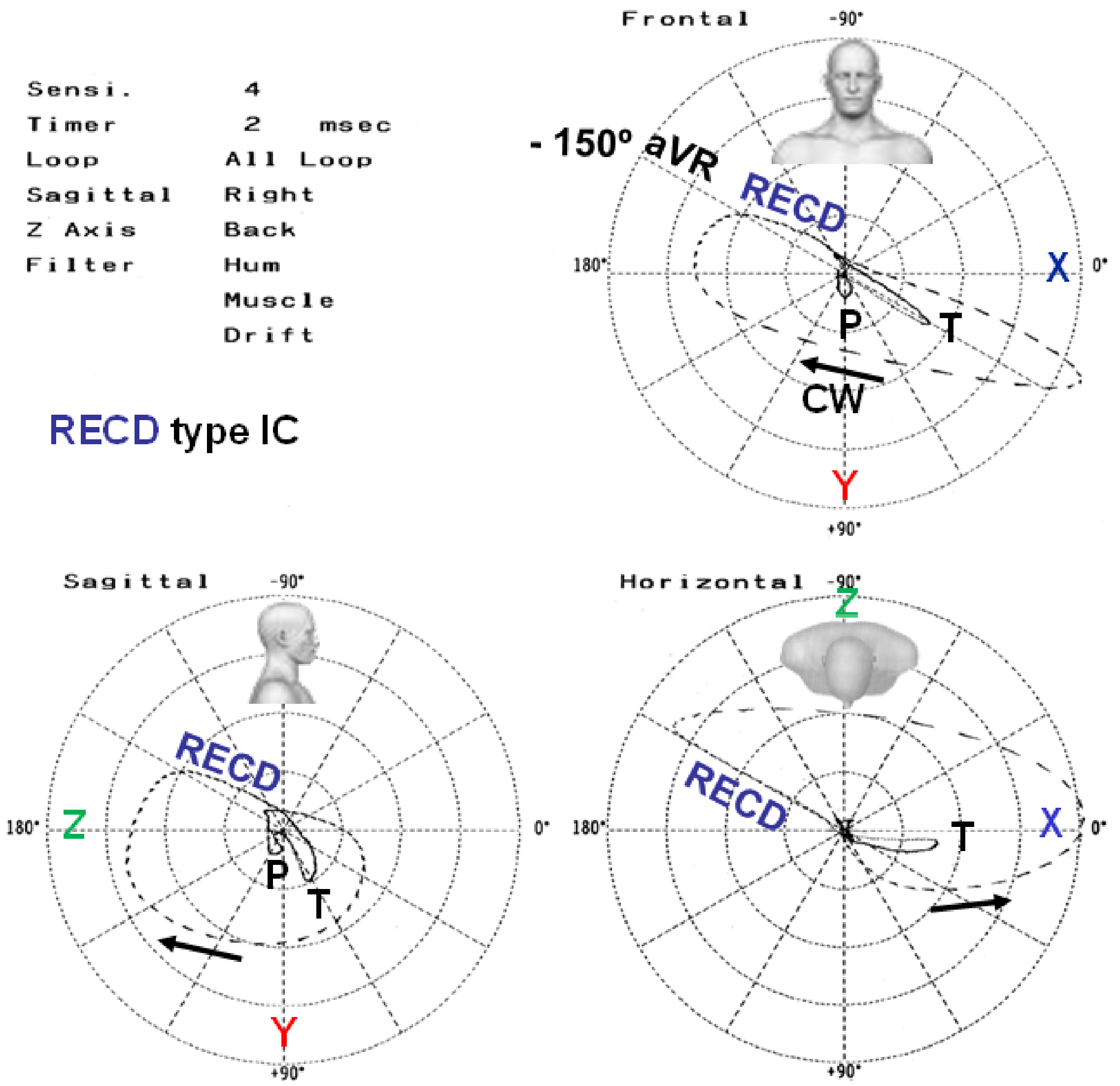

Figure 27. Vectorcardiogram of typical case of RECD type IC: RECD near aVR on FP and QRS loop with CW rotation.

ECG/VCG correlation on Frontal Plane - RECD type IC

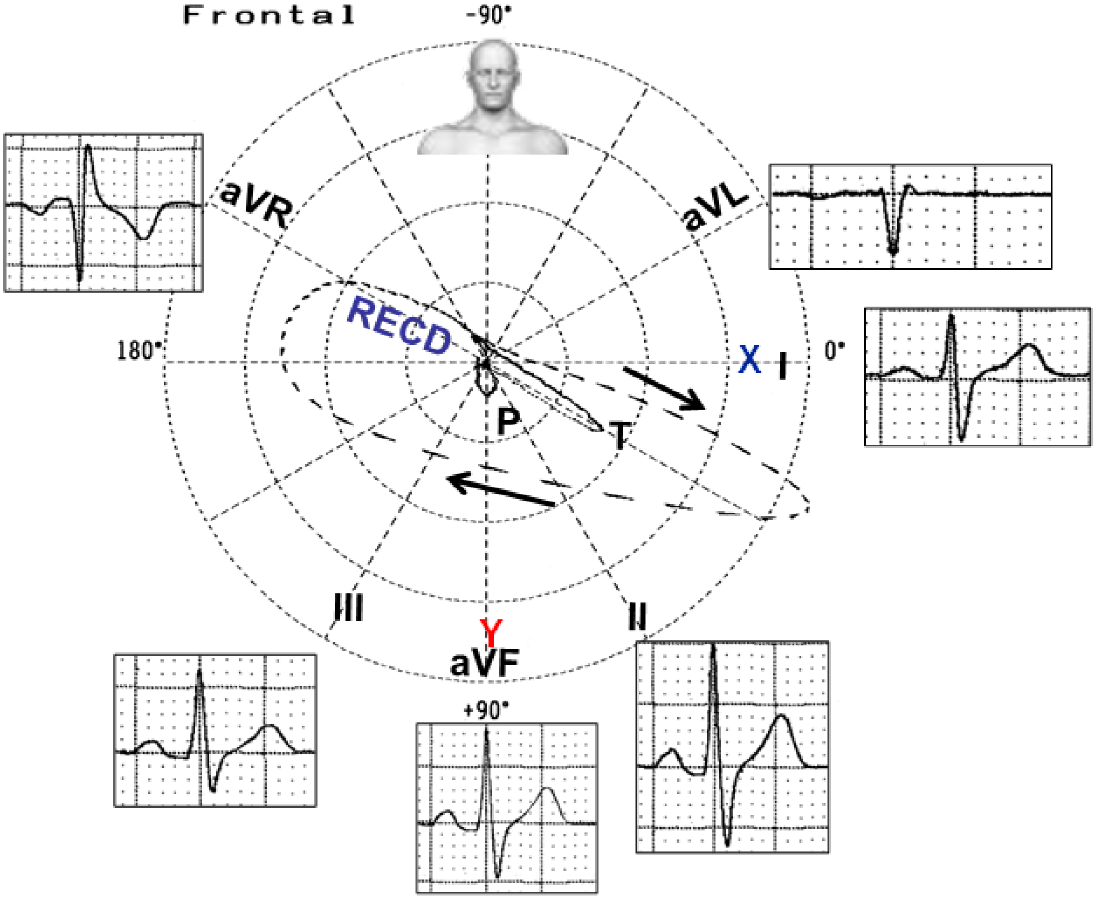

Figure 28. RECD located in the right superior quadrant near aVR lead; broad R wave of aVR: RECD type IC; SI-SII-SIII; QRS loop of clockwise rotation.

ECG/VCG correlation on Horizontal Plane - RECD type IC

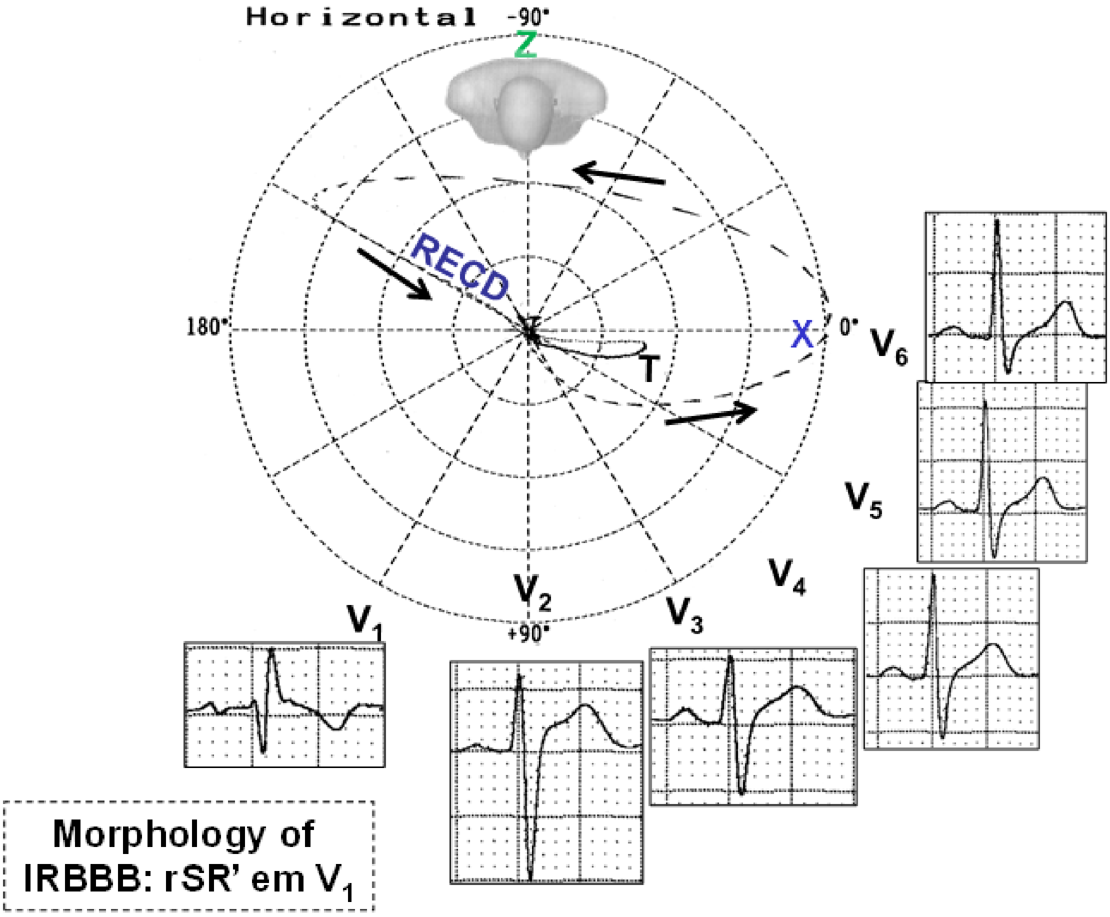

Figure 29. Characteristic QRS loop with type C RVH like morphology in HP (≥ 20% area on right posterior quadrant) (Luna Filho, Bocanegra et al. 1989). Consequently we observed deep S waves in V2-V3.

Name: DAS; **Sex:** F; **Age:** 22 yo; **Race:** white**; Weight:** 55Kg; **Height:** 1.60 m;
Biotype: asthenic;; **Date:** 22/04/2001

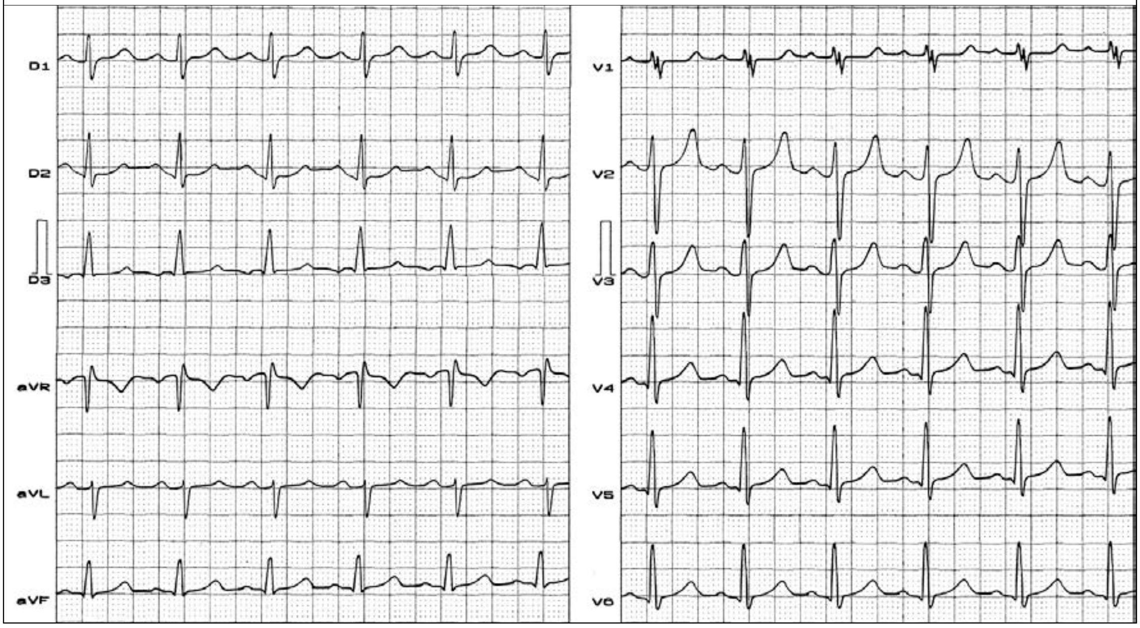

Figure 30. Clinical diagnosis: ASD of the Ostium Secundum type (ASD-OS). ECG diagnosis: SR; HR: 91 bpm; morphology of IRBBB; QRS duration: 110 ms; tetraphasic pattern of low voltage in V1; End Conduction Delay (RECD). Commentary: the most frequent cause of RBBB in congenital heart diseases is ASD. Conclusion: RECD by one of the right bundle branch fascicles.

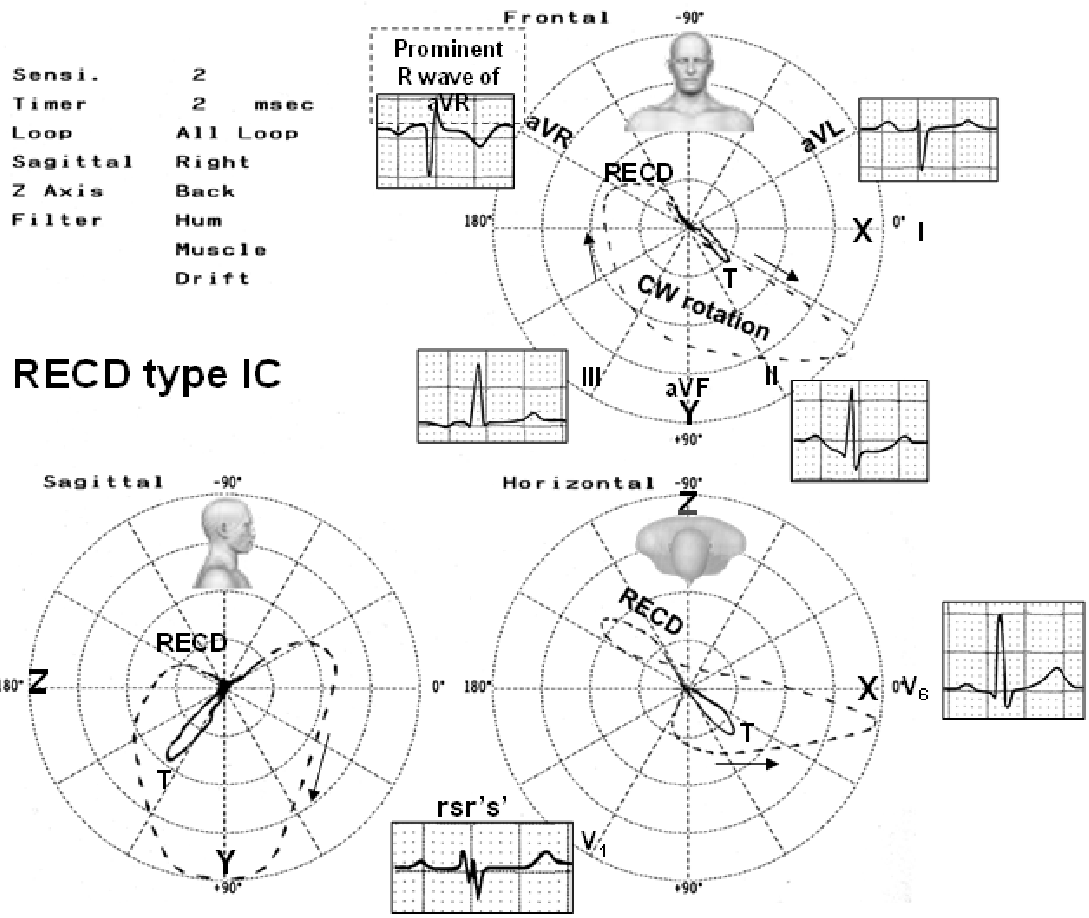

Figure 31. Conclusion: RECD type IC - QRS loop of CW, SÂQRS +90º and RECD located in the right superior quadrant: Right superior or subpulmonary fascicular block. In HP RECD located on right posterior quadrant.

Example of RECD type IC pseudo anterior myocardial infartion

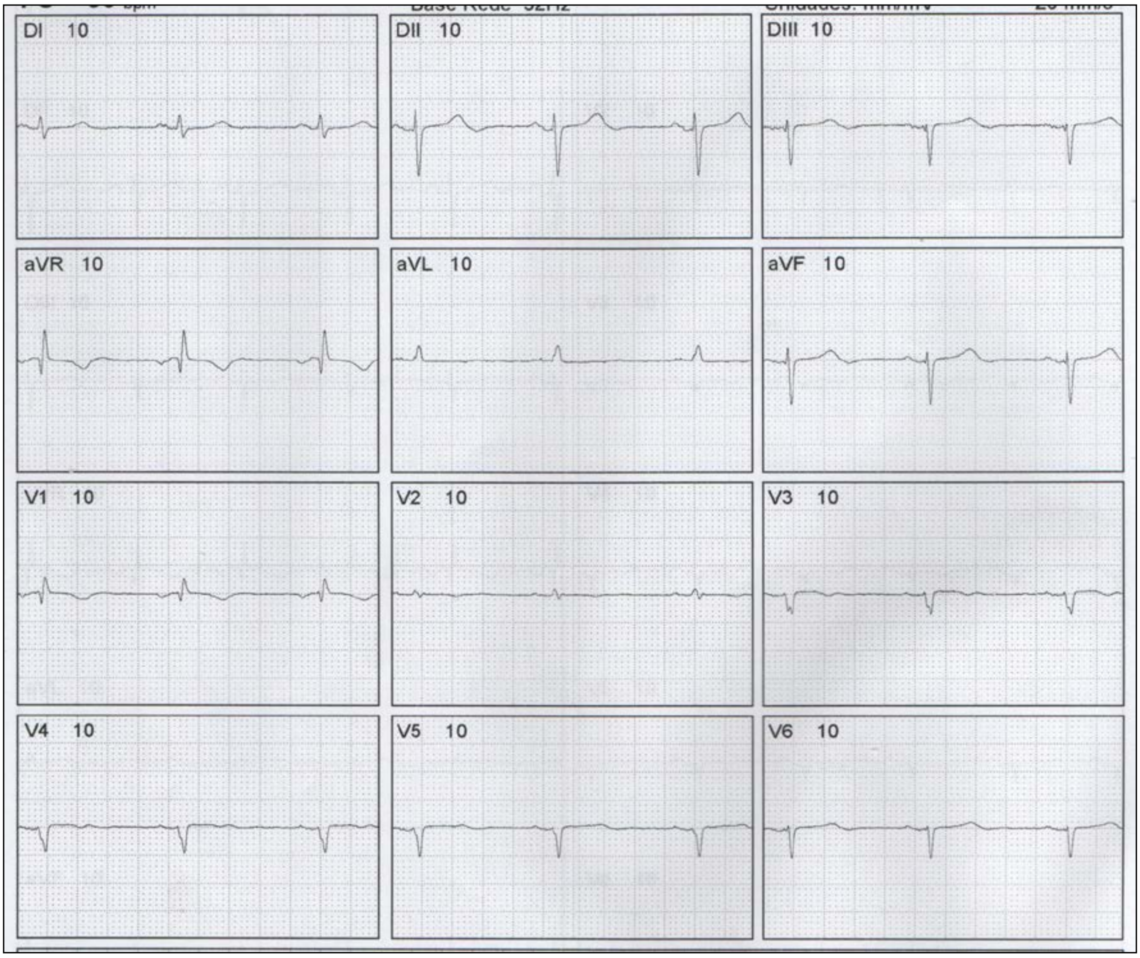

Figure 32. ECG.

ECG/VCG correlation

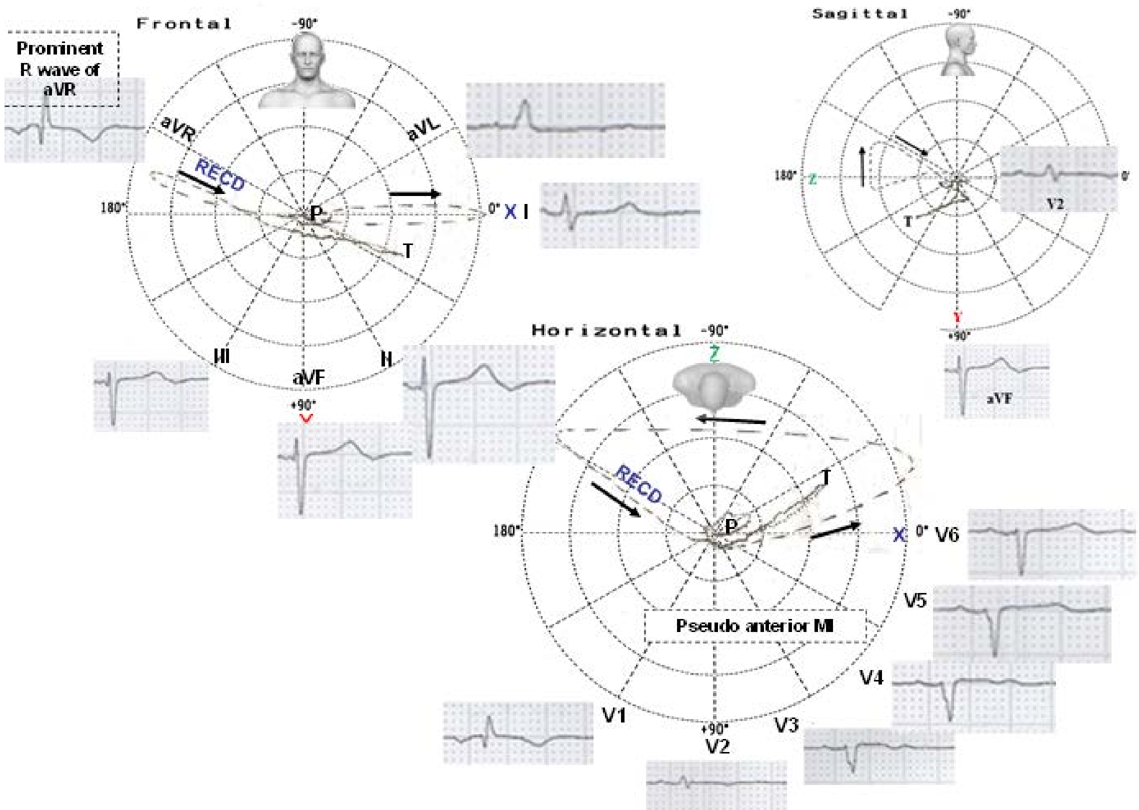

Figure 33. ECG/VCG correlation.

RECD type IC in a patient with Brugada syndrome

Aborted Sudden Death, positive familiar background of sudden death. Family history of premature sudden death (<35years) in first degree relatives, genetic research performed: negative.

Identification : **Name**: AS **Age**: 35; **Gender**: Male;
Ethnic Group: Asian; **Weight**: 72kg; **Height** 1,71m

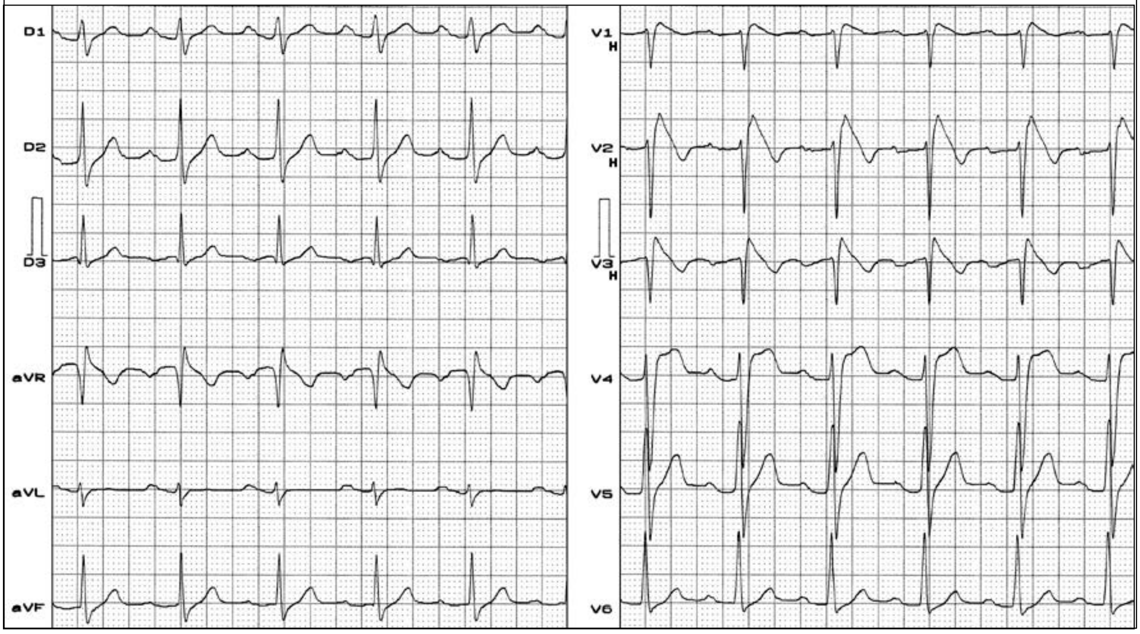

Figure 34. HR: 82bpm P Axis: +33°; PR Interval: 258ms; QRS Axis: +84°; QRS Duration: 112ms; QT/QTc: 360/420ms; T Axis +50°. Conclusion: First degree AV block + type 1 Brugada ECG pattern on right precordial leads.

ECG/VCG correlation on Frontal Plane

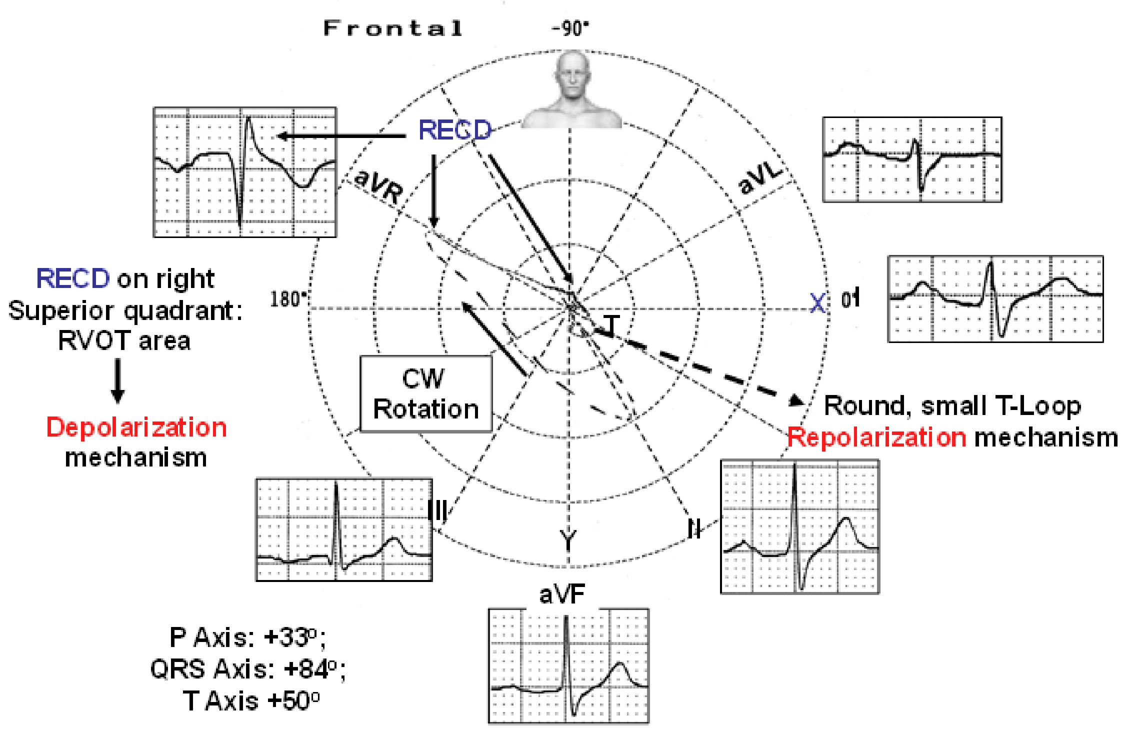

Figure 35. RECD type IC: Right End Conduction Delay located on top right quadrant. QRS loop with CW rotation.

ECG/VCG correlation on Horizontal Plane

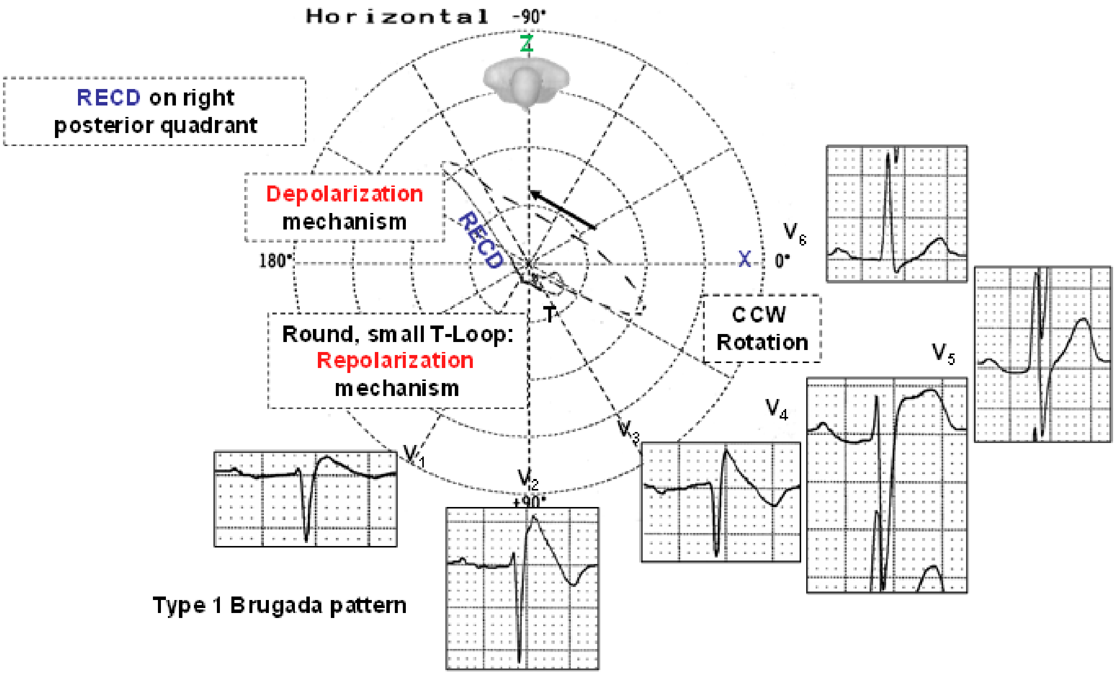

Figure 36. QRS loop with type C RVH like morphology in HP (≥ 20% area on right posterior quadrant). Deep S waves in V2-V3.

RECD type II, Right Posterior Subdivision Block or Right Inferior Fascicular Block

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

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

A) Electrocardiographic criteria:

- ✓ SÂQRS between + 70° and + 110°;
- ✓ QRS duration normal;
- ✓ SI – RII - RIII pattern, with RII and RIII of voltage not increased (usually ≤10 mm), never reaching 15 mm (essential element for the differential diagnosis with LPFB);
- ✓ RII ≥ RIII (in LPFB RIII > RII);
- ✓ Prolonged ventricular activation time on V5R, V3R and V4R (and aVF in horizontal hearts). This is because these leads are located opposite the blocked area;
- ✓ aVR of the QS type;
- ✓ Possible notch in the descending ramp on inferior leads;
- ✓ S wave of V_2 and/or V_3 of increased depth;
- ✓ Persistent with notched S wave in V_5 and/or V_6;
- ✓ V_1: rS, RS or rSR' with S of V_1 and V_2 possibly broadened.

B) VCG criteria: Right End conduction delay in the three planes located to the right and below.

Frontal Plane:

1) Initial vectors always to the left, above and below;
2) Clockwise rotation;
3) Predominant location in the inferior quadrants;
4) Rapid change from left to right between 30ms and 50ms;
5) RECD to the right and below between $+120^{\circ}$ and $+150^{\circ}$.

Horizontal Plane:

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

Right Sagittal Plane:

1) Initial vectors upward or downward;
2) Clockwise rotation;
3) Marked posterior/inferior dislocation;
4) RECD downward and backward.

Differential diagnosis between RECD type II and Left Posterior Fascicular Block (LPFB)

	RECD type II or Right Posterior Subdivision Block	LPFB
PR interval	Normal.	Frequent prolongation.
Association with inferior infarction	No.	Frequent.
Voltage of RII and RIII	≤ 10 mm.	≥ 15 mm.
RII/RIII voltage ratio	RII >RIII.	RIII > RII.
Notch in the descending ramp of R wave of inferior leads	Absent.	Constant middle-final notch.
Ventricular activation time in aVF, V5 and V6	Normal.	Increased: up to 30 ms.
Ventricular activation time in aVL	Normal.	Decreased: up to 15 ms.
QRS loop in the FP	Clockwise rotation and with characteristic rapid passage from left to right between 30 and 50 ms. RECD on inferior right quadrant.	Clockwise, aspect of “fat” loop and maximal vector close to + 120°.

Name: BMB; **Sex:** M; **Age:** 20 yo; **Race:** White; **Weight:** 78 Kg; **Height:** 1.81 m;
Biotype: Athletic; **Date:** 18/09/2004; **Medication in use:** nothing stated

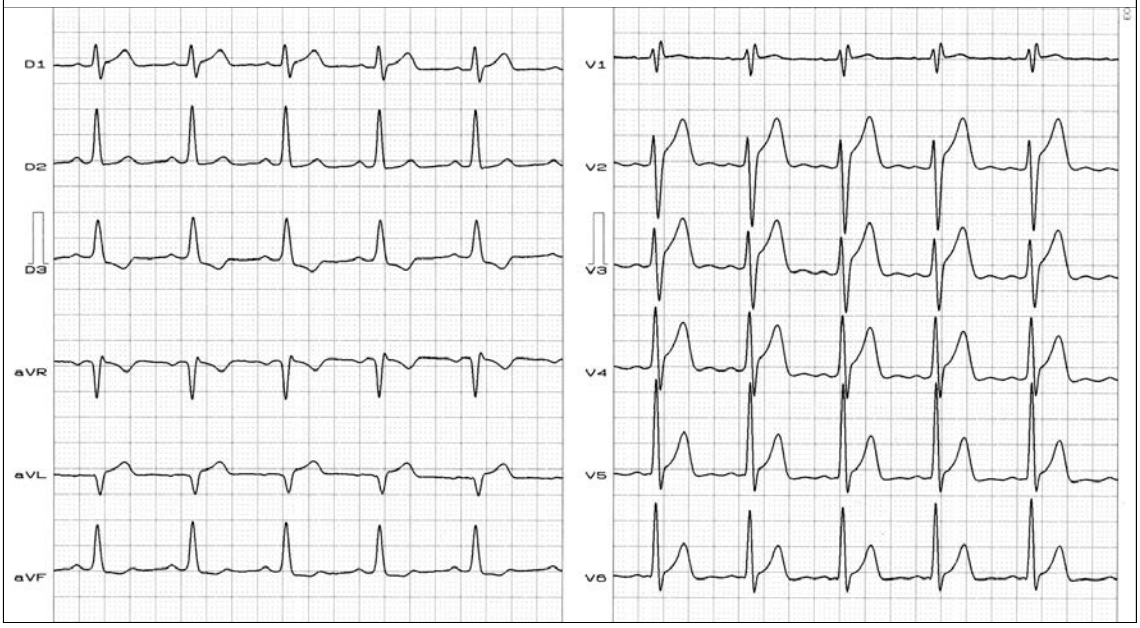

Figure 37. Clinical diagnosis: Healthy patient. He came to the office to have his aptitude for the practice of sports evaluated. ECG diagnosis: SÂQRS: + 85º. RII > RIII. SAT: + 5º to the front and the left. Morphology of IRBBB: rSR’ in V1. Conclusion: ECG of RECD type II.

ECG/VCG correlation on Frontal Plane

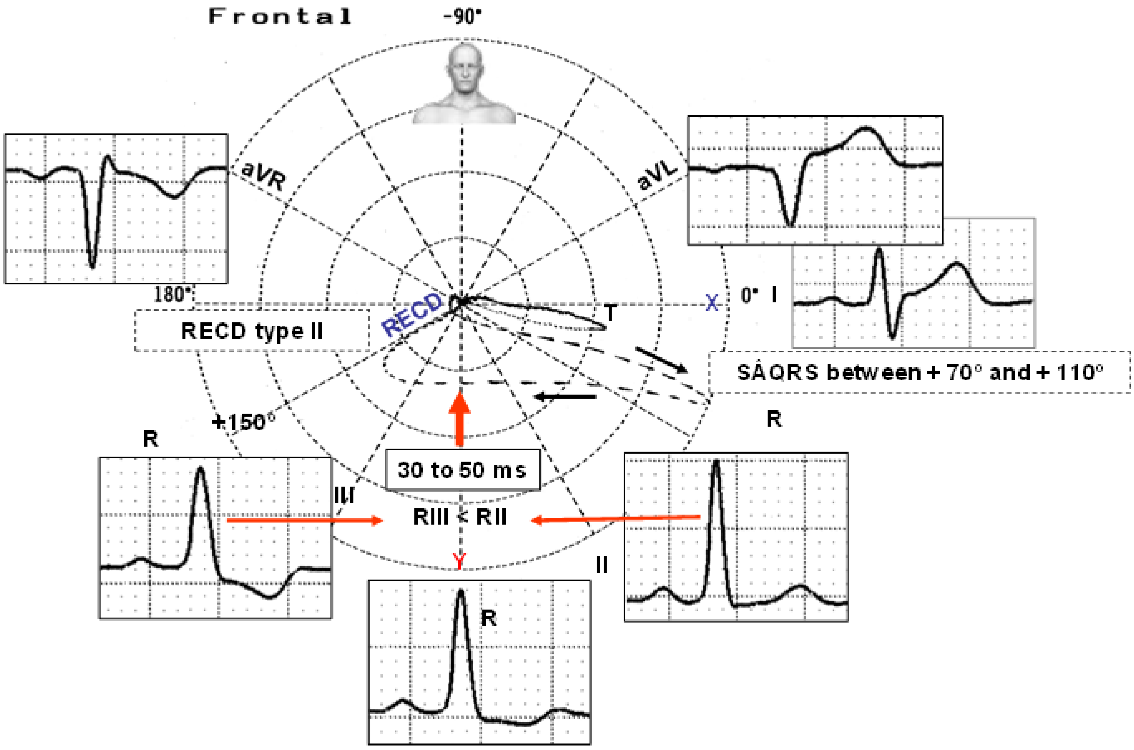

Figure 38. QRS loop located on inferior quadrants with CW rotation, rapid passage from left to right between 30 to 50 ms and RECD located on inferior right quadrant.

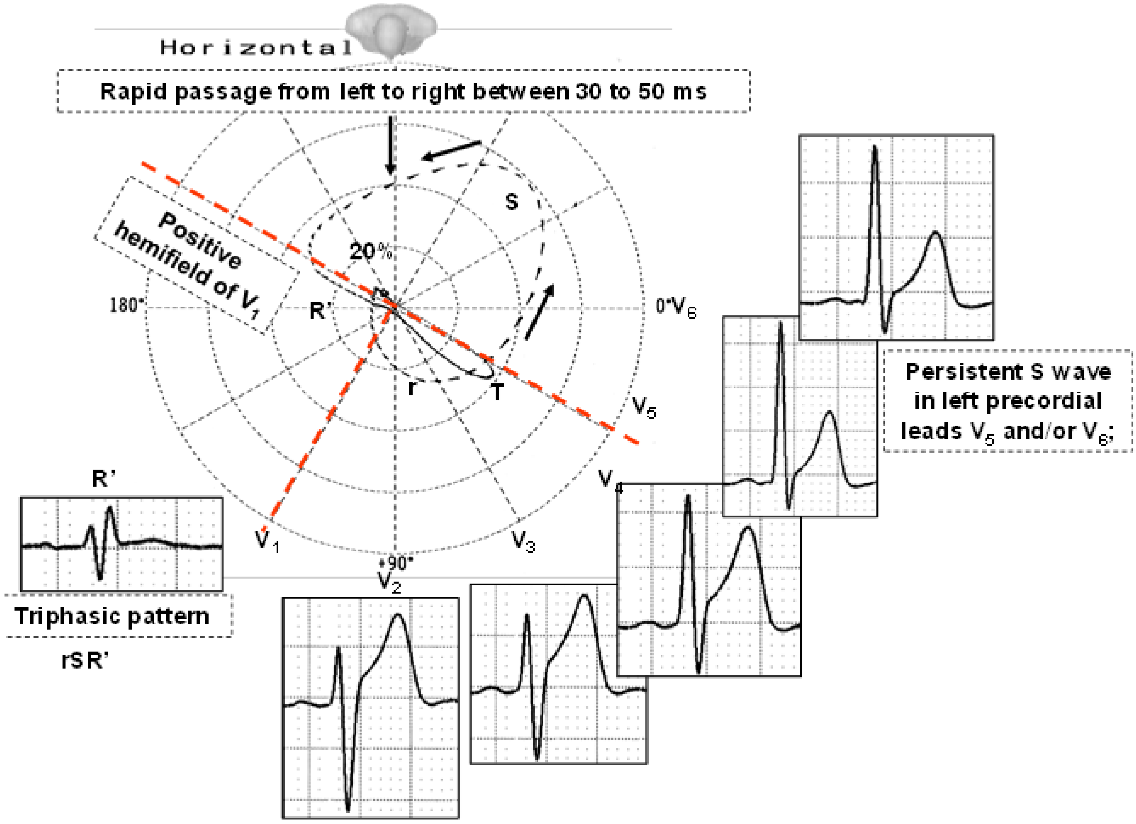

Figure 39. ECG/VCG correlation in horizontal plane of ECD type II.

Name: CJO **Sex:** male. **Age:** 22 Y. **RACE:** White
Weight: 70 Kg **Height:** 1.71 m **Biotype:** Athletic **DATE:** 15/05/2001

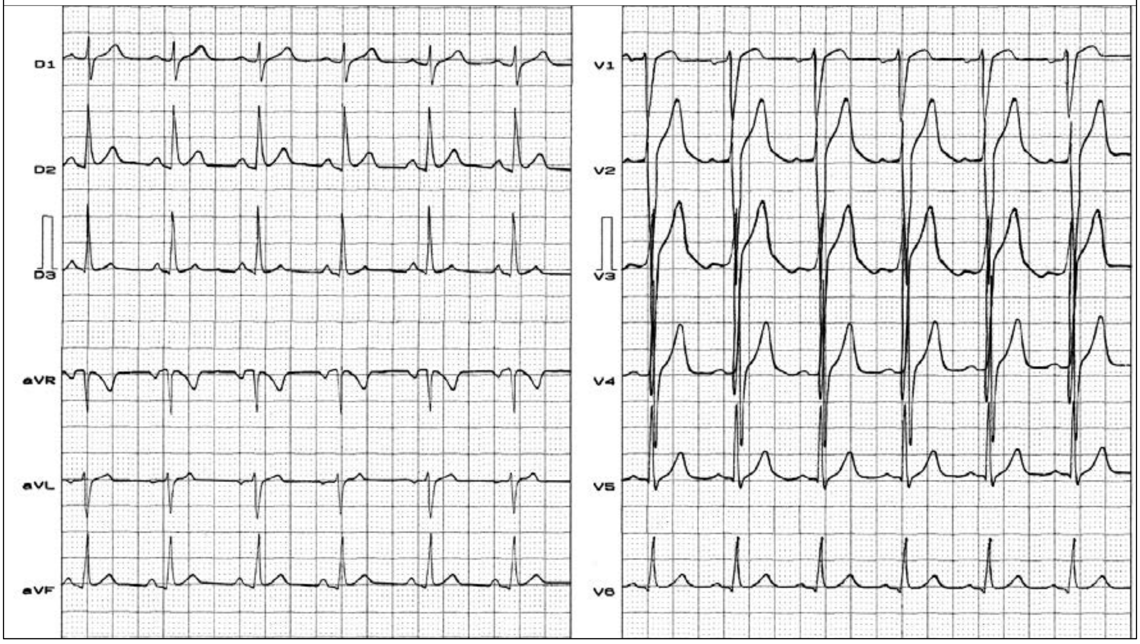

Figure 40. Clinical diagnosis: CM: preoperative evaluation for abdominal surgery, physical examination background: nothing important. ECHO nothing important. Chest X rays: nothing important; ECG diagnosis: SÂQRS:+ 95º. SI-RII-RIII pattern (RIII < 15 mm). I and aVL: rS. II and III: qR. Descending ramp of R wave is slightly slow. It may present diagnostic doubt with LPFB.

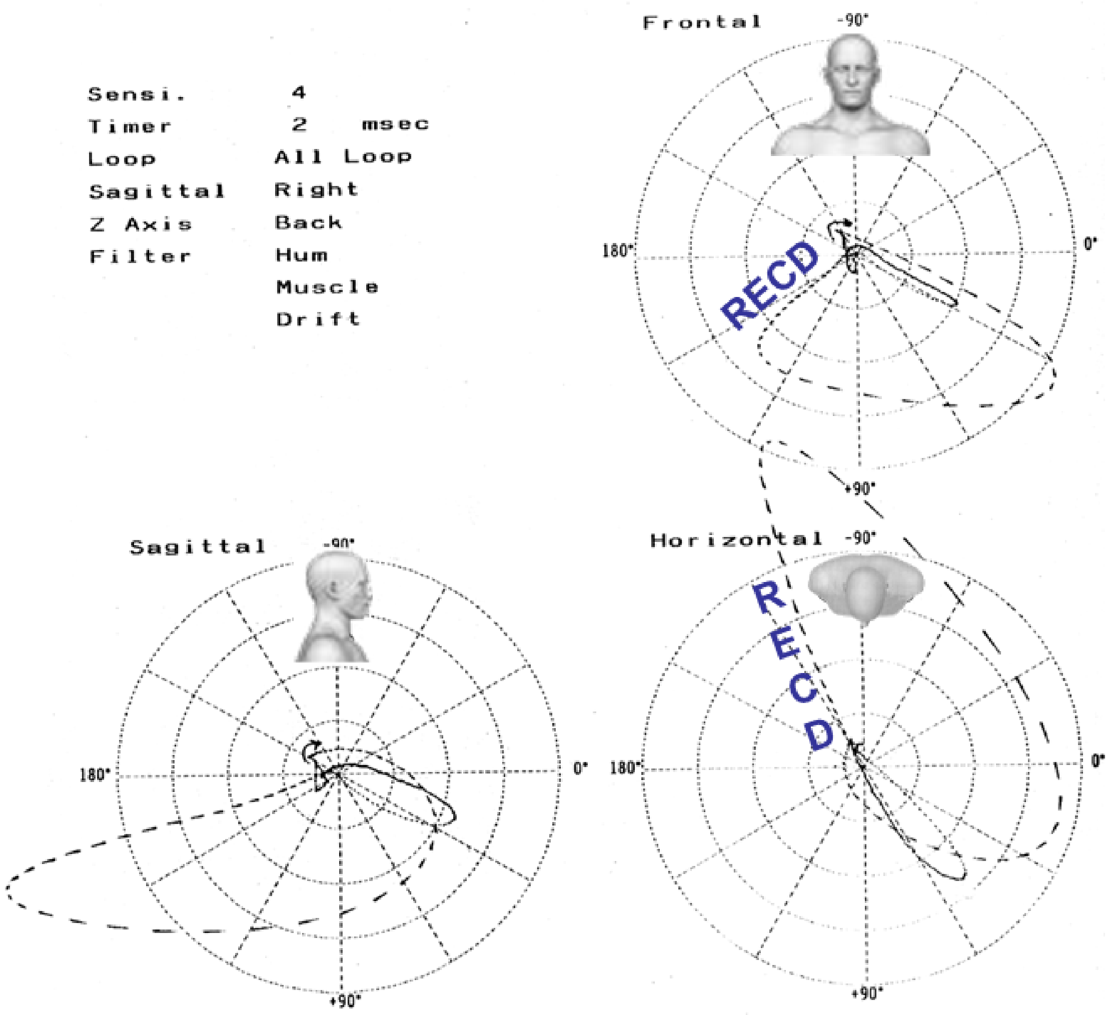

Figure 41. VCG of RECD type II.

ECG/VCG correlation on Frontal Plane

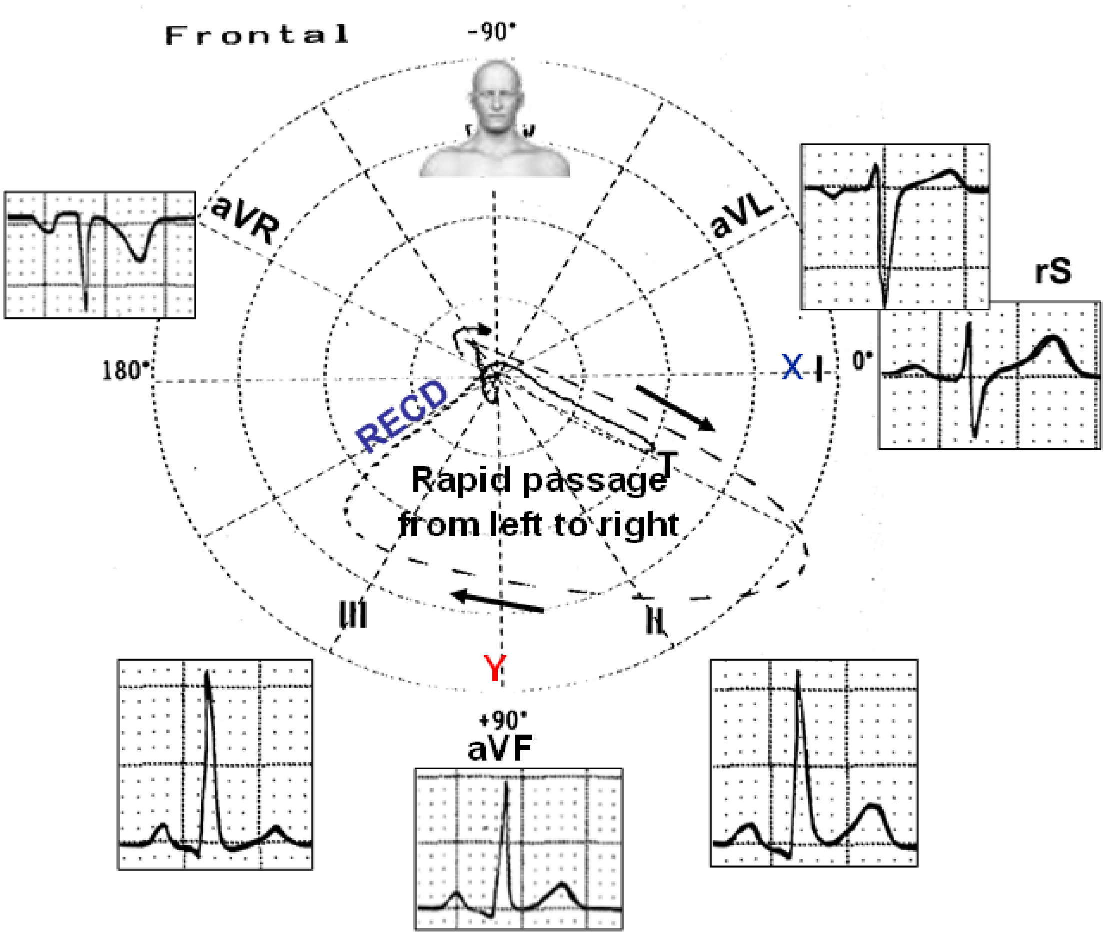

Figure 42. RECD type II: RECD located in the right inferior quadrant in the territory of the inferior fascicle of the right bundle branch. SÂQRS: +95º. SI-RII-RIII pattern (RIII < 15 mm). I and aVL: rS. II and III: qR. The descending ramp of R wave is slightly slow. It may present differential diagnosis with LPFB.

ECG/VCG correlation on Horizontal Plane

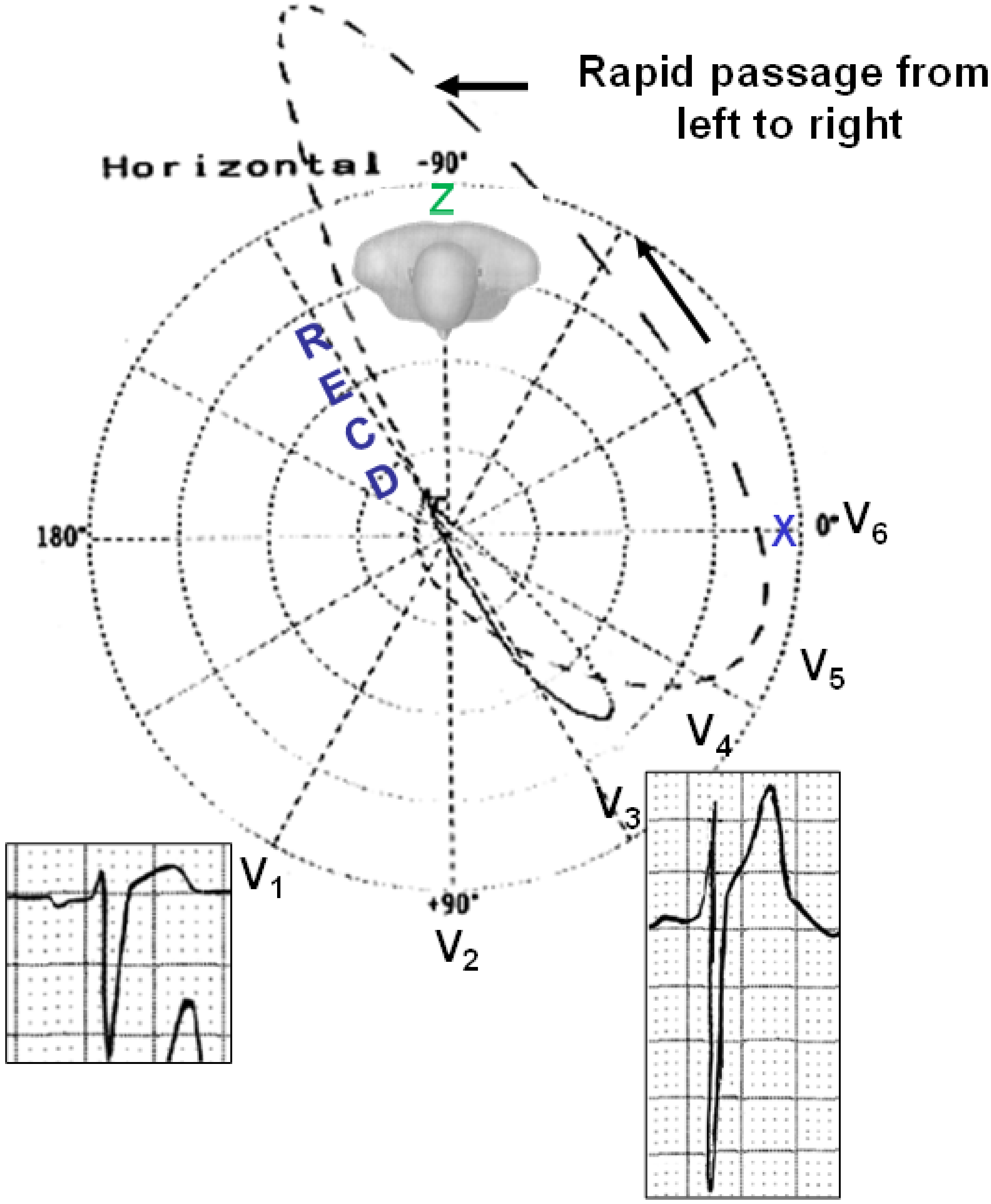

Figure 43. QRS loop with initial portions to the front, counterclockwise rotation, important posterior dislocation, rapid passage from left to right and RECD located to the back and the left.

RECD type III corresponding to Right Middle Fascicle Block (RMFB)

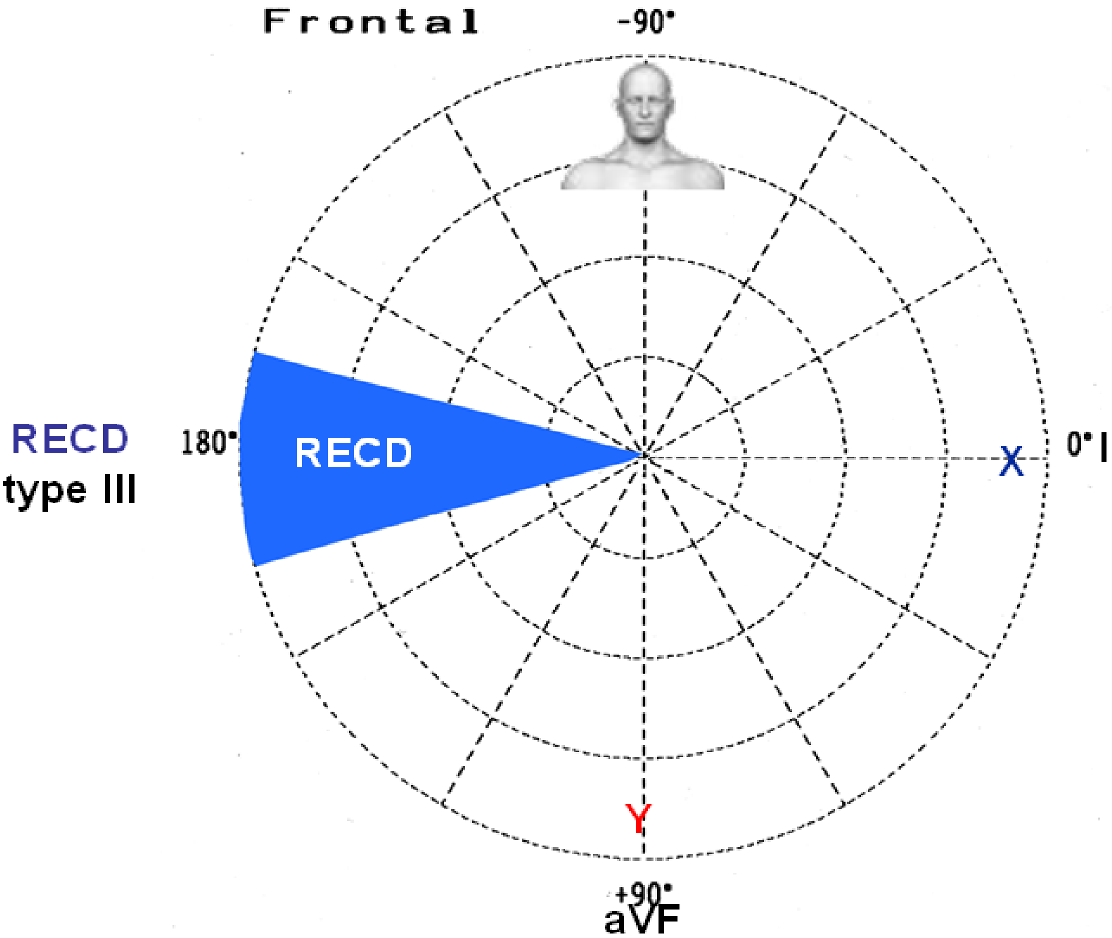

Figure 44. RECD located in the territory of the middle or anterior/superior fascicle of the right bundle branch, i.e. very close or on the orthogonal X line of ±180º.

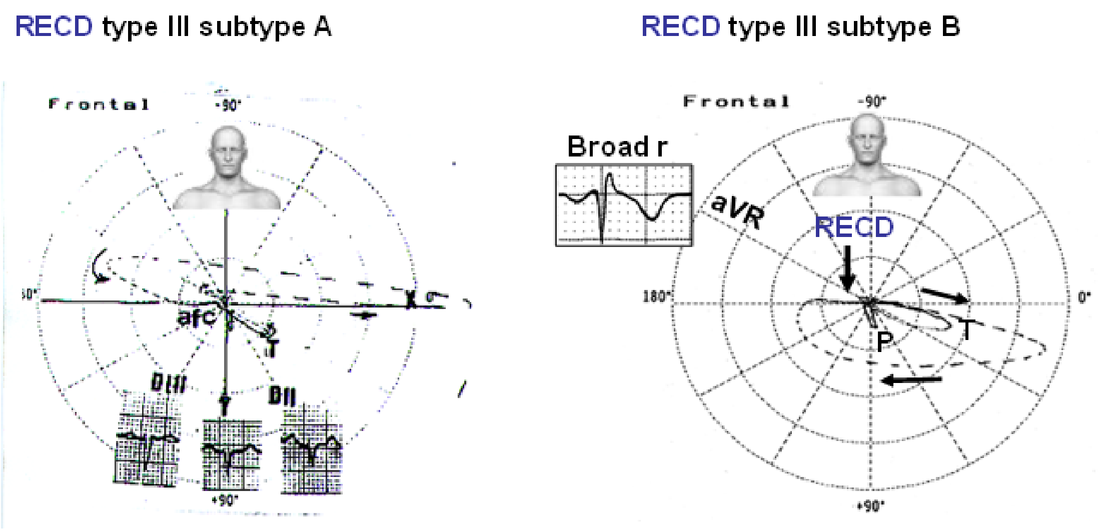

Figure 45. RECD type III subtype A may be confused with RECD type IA.

Its characterization is made by observing the efferent branch of the QRS loop in the FP that accompanies from left to right the X line, of fast recording and ending in an end delay located very near this line and to the right, indicating that the territory with final activation is the middle one of the RV free wall, depending on the fascicle with the same name.

In the frontal plane, the QRS loop may rotate in a counterclockwise way, with SÂQRS presenting extreme deviation in the superior quadrants.

(Subtype A) however presents RECD in the line of ± 180º. Another variant, called Subtype B, presents clockwise rotation, and SÂQRS not deviated.

Two subtypes of RECD type III: type IIIA and IIIB. Type IIIA may be confused with LAFB and RECD type IA.

Name: AB; **Sex:** M; **Age:** 52 yo; **Race:** W; **Weight:** 90 Kg; **Height:** 1.80 m;
iotype: Athletic; **Date:** 27/08/1991

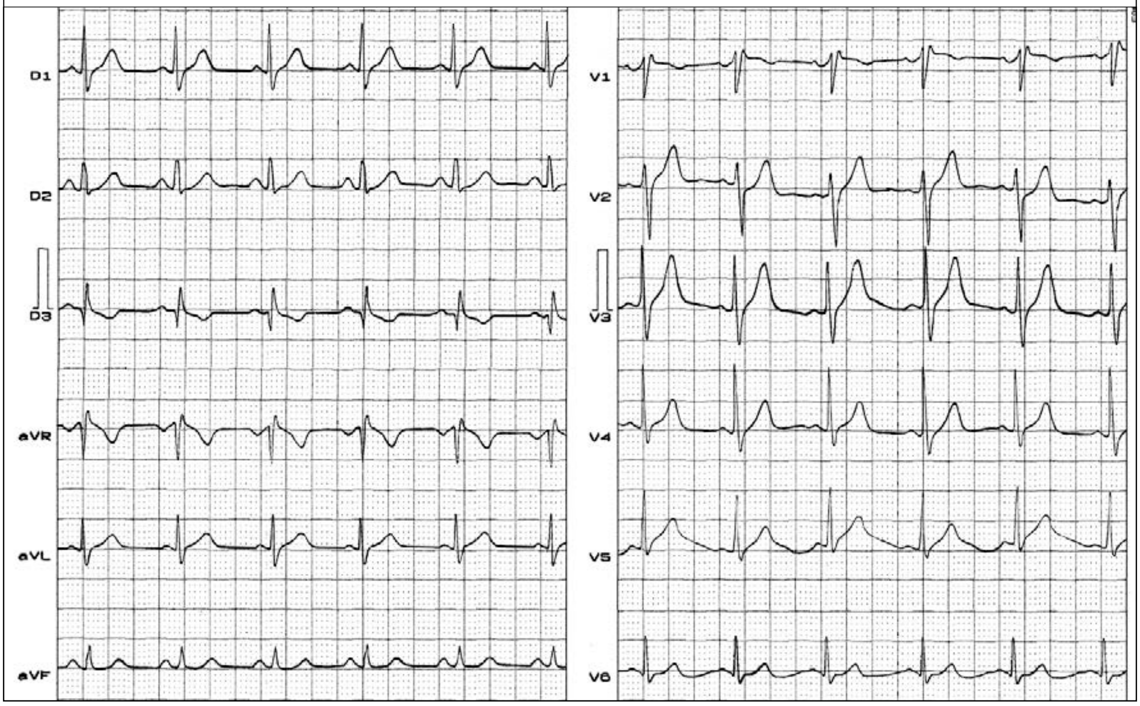

Figure 46. Clinical diagnosis: mild systemic hypertension. Normal echocardiogram. ECG diagnosis: Right end conduction delay. Morphology of IRBBB: rSr' in V1.

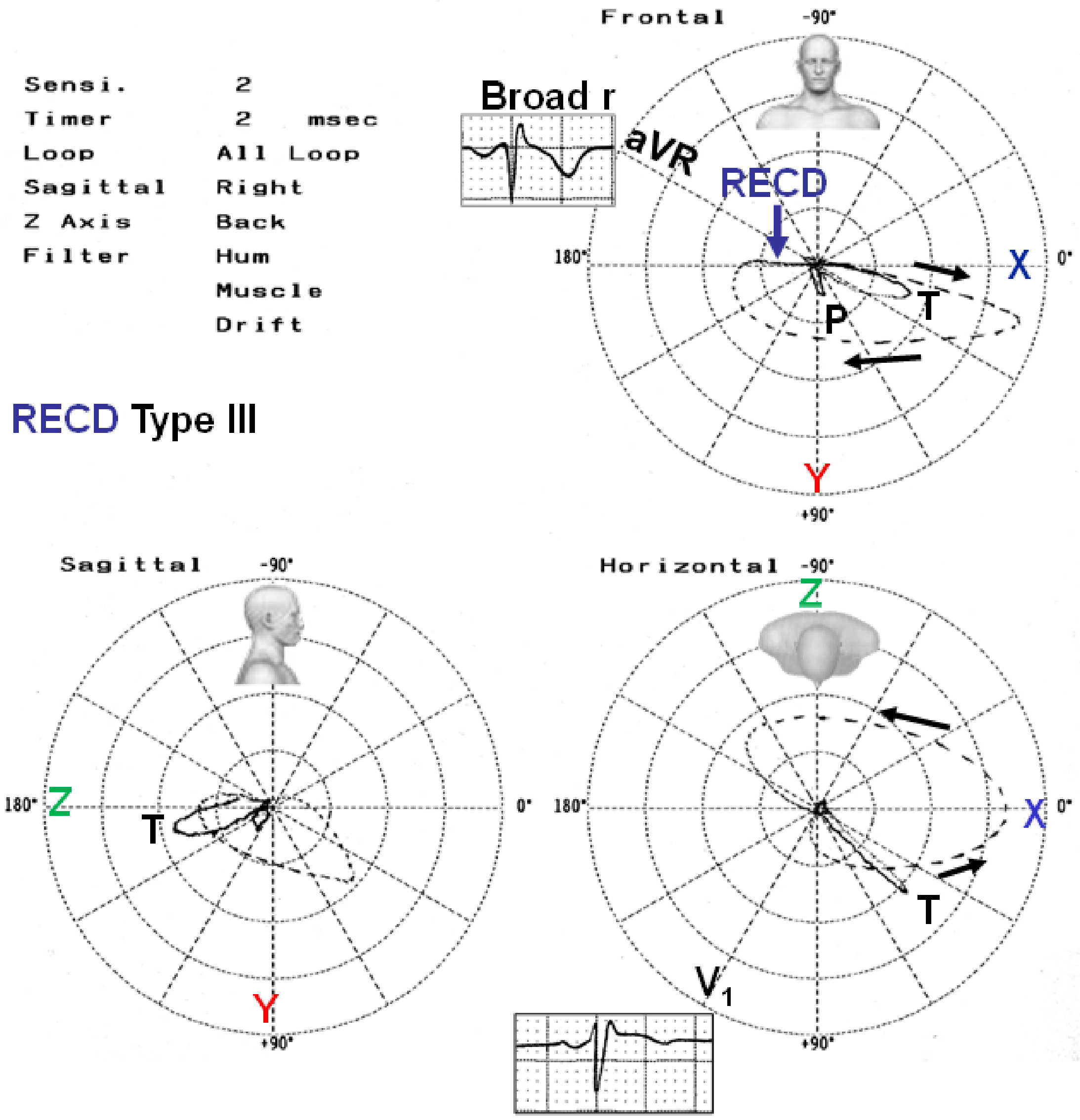

Figure 47. Typical ECG/VCG of a case of RECD type III.

Typical ECG/VCG of a case of RECD type III

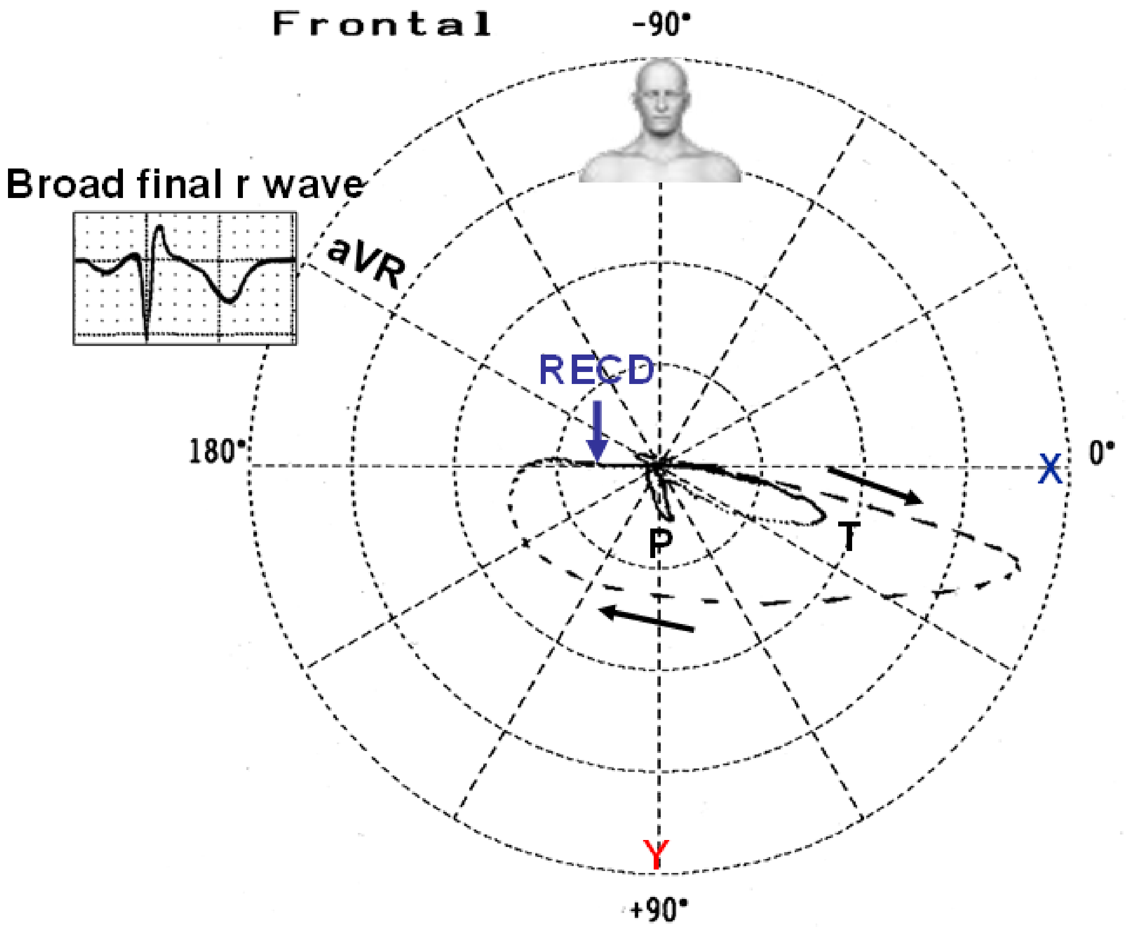

Figure 48. RECD type III: ECD located on the right portion of the orthogonal line/±180º, corresponding to the territory of distribution of the middle fascicle of the right branch.

Clinical significance of RECD

Its clinical significance and interest lies in the fact that:

1) They may be confused with left fascicular blocks: Left Anterior Fascicular Block (LAFB) and Left Posterior Fascicular Block (LPFB);

2) They may be confused with electrically inactive areas (pseudo electrically inactive areas) both in the anterior and the inferior walls.

3) They may represent the electro-vectocardiographic pattern of Brugada syndrome and one subpopulation of Arrhythmogenic Right Ventricular Dysplasia (ARVD/C).

From 100 consecutive cases of our series, 15 presented diagnostic doubt by ECG with electrically inactive area. This caused us to request a VCG to clarify this issue. From these, 12 raised the suspicion of inferior MI electrically inactive area (IEIA) and 3 anterior or septal MI.

From these 12 that presented diagnostic doubt with inferior electrically inactive area, 11 were diagnosed as RECD Type IA and 1 as RECD Type III subtype A.

The criteria for the differential diagnoses are the following:

	RECD	Inferior MI
Initial r wave in inferior leads:	It is the rule.	When present only in III
Q wave in II, III and aVF:	If present < 40 ms.	Present in 65 to 70% of the cases and > 40 ms. In the three leads in 15%, only in III 25%, only in aVF 5 to 10%, in II and aVF 25 to 30%. aVF is the most sensitive lead. III presents many false positives.
QRS loop in the FP:	Variable.	Characteristic rotation from right to left of inferior concavity, clockwise rotation, abnormal superior dislocation of initial forces (minimum 25 ms). +12 dashes above the X line. Maximal vector -40º and +30º (almost always less than +15º). The initial 10 ms may have a superior orientation (group I of Young and Williams) or more rarely inferior (group II).

Name: OSA; **Sex:** F; **Age:** 74 yo; **Race:** White; **Weight:** 60 Kg; **Height:** 1.41 m;
Date: 26/11/2003; **Medication in use:** nothing is stated

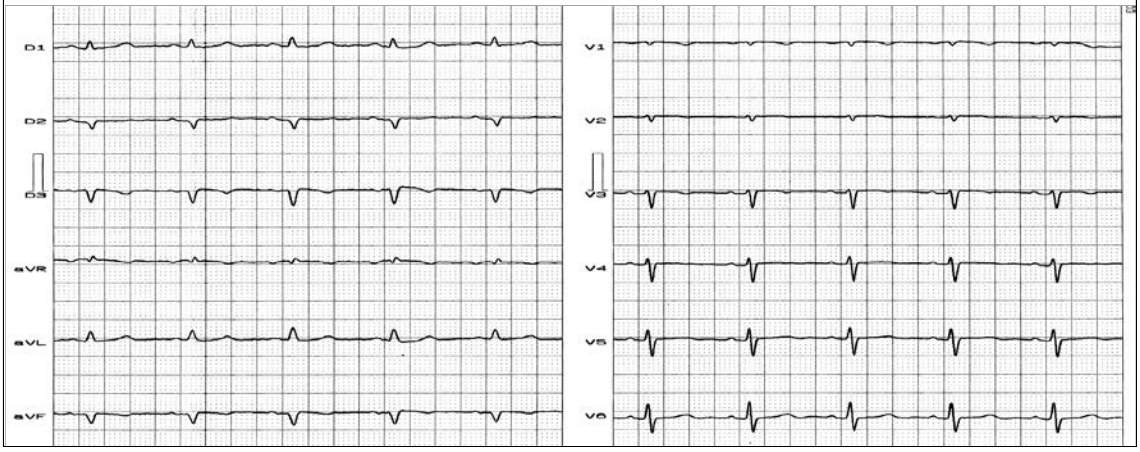

Figure 49. Clinical diagnosis: asymptomatic. Routine consultation due to a check-up. Echocardiogram: Normal. ECG diagnosis: SR, HR: 75 bpm, P wave: SAP: +30º; duration: 75 ms; voltage: 0.3 mV. PR: 165 ms. QRS: extreme shift of SAQRS in the left superior quadrant – 60º; duration: 75 ms. Low voltage in the frontal plane, qs in inferior leads: probable electrically inactive area in inferior wall. Slow growth of r wave from V2 to V6, rS from V3 to V6: RVE type C or special? SAT: - 20º; QT: 360 ms; QTc: 408 ms. Conclusion: doubtful electrically inactive area in inferior wall? RVH Type C or special? Anterior electrically inactive area?: r wave with scant growth in precordial leads. ECG/VCG sequence of a case of RECD type IA, which resembles LAFB, inferior and anterior electrically inactive area and RVH type C.

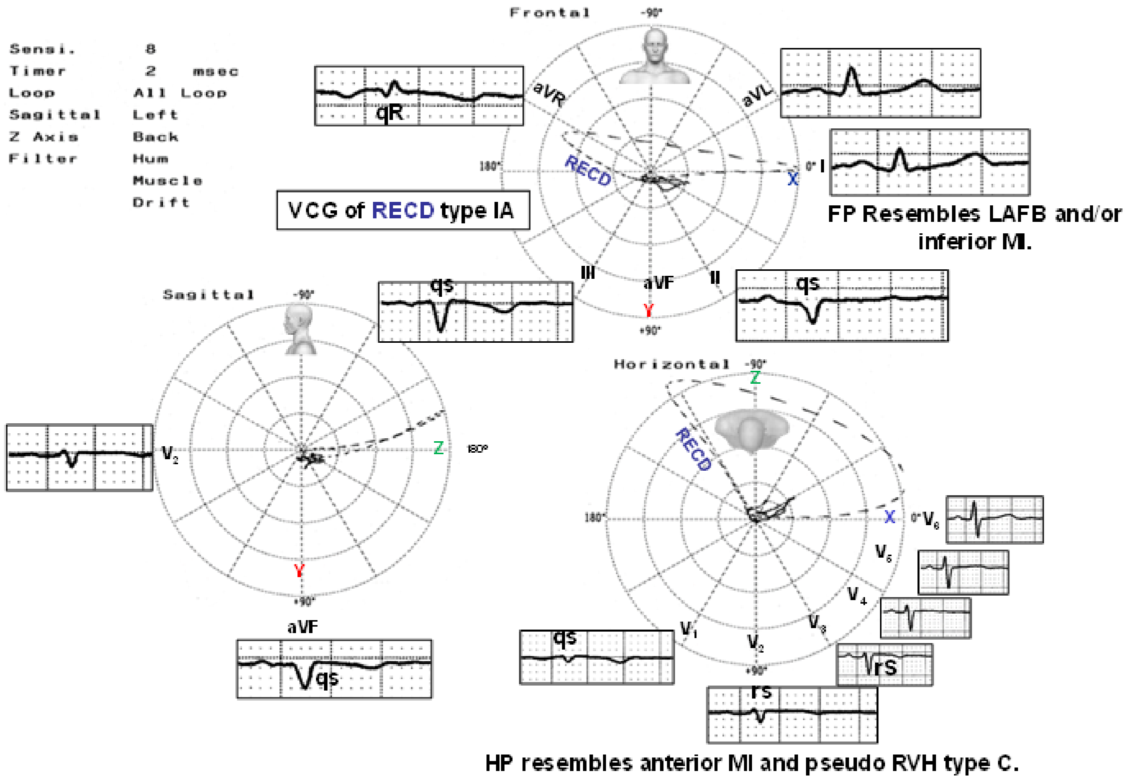

Figure 50. ECG/VCG correlation.

REFERENCES

Alzamora-Castro, V., R. Abugattas, C. Rubio, J. Bouroncle, C. Zapata, E. Santa-Maria, G. Battilana, T. Binder, R. Subiria and D. Paredes (1953). "[Parietal focal block; an experimental and electrocardiographic study]." Circulation **7**(1): 108-115.

Ansari, A. (1985). "The "straight back" syndrome: current perspective more often associated with valvular heart disease than pseudoheart disease: a prospective clinical, electrocardiographic, roentgenographic, and echocardiographic study of 50 patients." Clin Cardiol **8**(5): 290-305.

Babai Bigi, M. A., A. Aslani and S. Shahrzad (2007). "aVR sign as a risk factor for life-threatening arrhythmic events in patients with Brugada syndrome." Heart Rhythm **4**(8): 1009-1012.

Baranchuk, A., A. Enriquez, J. Garcia-Niebla, A. Bayes-Genis, R. Villuendas and A. Bayes de Luna (2015). "Differential diagnosis of rSr' pattern in leads V1 -V2. Comprehensive review and proposed algorithm." Ann Noninvasive Electrocardiol **20**(1): 7-17.

Barker, J. M. and F. Valencia (1949). "The precordial electrocardiogram in incomplete right bundle branch block." Am Heart J **38**(3): 376-406.

Basagoitia, A. M., P. Iturralde, O. Galvan, J. Rosado, C. Vazquez, L. Colin, L. Romero and J. Gonzalez Hermosillo (1991). "[Disorders of the rhythm and conduction in patients operated on for a total correction of tetralogy of Fallot]." Arch Inst Cardiol Mex **61**(1): 27-32.

Bayes de Luna, A., J. Brugada, A. Baranchuk, M. Borggrefe, G. Breithardt, D. Goldwasser, P. Lambiase, A. P. Riera, J. Garcia-Niebla, C. Pastore, G. Oreto, W. McKenna, W. Zareba, R. Brugada and P. Brugada (2012). "Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report." J Electrocardiol **45**(5): 433-442.

Bayes de Luna, A., I. Carrio, M. T. Subirana, P. Torner, J. Cosin, F. Sagues and J. Guindo (1987). "Electrophysiological mechanisms of the SI SII SIII electrocardiographic morphology." J Electrocardiol **20**(1): 38-44.

Bussink, B. E., A. G. Holst, L. Jespersen, J. W. Deckers, G. B. Jensen and E. Prescott (2013). "Right bundle branch block: prevalence, risk factors, and outcome in the general population: results from the Copenhagen City Heart Study." Eur Heart J **34**(2): 138-146.

Camerini, F. and L. G. Davies (1955). "Secondary R waves in right chest leads." Br Heart J **17**(1): 28-32.

Corrado, D., A. Nava, G. Buja, B. Martini, G. Fasoli, L. Oselladore, P. Turrini and G. Thiene (1996). "Familial cardiomyopathy underlies syndrome of right bundle branch block, ST segment elevation and sudden death." J Am Coll Cardiol **27**(2): 443-448.

de Luna, A. B., J. Garcia-Niebla and A. Baranchuk (2014). "New electrocardiographic features in Brugada syndrome." Curr Cardiol Rev **10**(3): 175-180.

de Micheli, A. and G. A. Medrano (1987). "[Disorders of intraventricular conduction]." Arch Inst Cardiol Mex **57**(3): 247-258.

de Micheli, A., G. A. Medrano and P. Iturralde-Torres (2009). "[Uncomplicated and complicated myocardial peripheral blocks]." Arch Cardiol Mex **79 Suppl 2**: 3-12.

Deleon, A. C., Jr., J. K. Perloff, H. Twigg and M. Majd (1965). "The Straight Back Syndrome: Clinical Cardiovascular Manifestations." Circulation **32**: 193-203.

Depasquale, N. P. and G. E. Burch (1961). "The electrocardiogram, vectorcardiogram, and ventricular gradient in the tetralogy of Fallot." Circulation **24**: 94-109.

Esser, S. M., M. H. Monroe and L. Littmann (2009). "Straight back syndrome." Eur Heart J **30**(14): 1752.
Grillo, H. C., C. D. Wright, P. G. Dartevelle, J. C. Wain and S. Murakami (2005). "Tracheal compression caused by straight back syndrome, chest wall deformity, and anterior spinal displacement: techniques for relief." Ann Thorac Surg **80**(6): 2057-2062.
Hoffman, B. F., E. N. Moore and J. H. Stuckey (1962). "Electrocardiographic changes due to delayed activation of the wall of the right ventricle." Surg Forum **13**: 200-201.
Kastor, J. A., B. N. Goldreyer, M. E. Josephson, J. K. Perloff, D. L. Scharf, J. H. Manchester, J. C. Shelburne and J. W. Hirshfeld, Jr. (1975). "Electrophysiologic characteristics of Ebstein's anomaly of the tricuspid valve." Circulation **52**(6): 987-995.
Kataoka, H. (2000). "Electrocardiographic patterns of the Brugada syndrome in right ventricular infarction/ischemia." Am J Cardiol **86**(9): 1056.
Kataoka, H. (2002). "Electrocardiographic patterns of the Brugada syndrome in 2 young patients with pectus excavatum." J Electrocardiol **35**(2): 169-171.
Kilic, Z., B. Ucar, F. Bas, E. C. Dinleyici and E. Sari (2002). "Familial secundum atrial septal defect with dysrhythmia associated with web neck." Turk J Pediatr **44**(1): 69-72.
Kim, J. H. and A. L. Baggish (2015). "Electrocardiographic right and left bundle branch block patterns in athletes: prevalence, pathology, and clinical significance." J Electrocardiol **48**(3): 380-384.
Kim, J. H., P. A. Noseworthy, D. McCarty, K. Yared, R. Weiner, F. Wang, M. J. Wood, A. M. Hutter, M. H. Picard and A. L. Baggish (2011). "Significance of electrocardiographic right bundle branch block in trained athletes." Am J Cardiol **107**(7): 1083-1089.
Kobza, R., F. Cuculi, R. Abacherli, S. Toggweiler, Y. Suter, F. Frey, J. J. Schmid and P. Erne (2012). "Twelve-lead electrocardiography in the young: physiologic and pathologic abnormalities." Heart Rhythm **9**(12): 2018-2022.
Lev, M. (1964). "The Normal Anatomy of the Conduction System in Man and Its Pathology in Atrioventricular Block." Ann N Y Acad Sci **111**: 817-829.
Lev, M. (1968). "Conduction system in congenital heart disease." Am J Cardiol **21**(5): 619-627.
Liao, Y. L., L. A. Emidy, A. Dyer, J. S. Hewitt, R. B. Shekelle, O. Paul, R. Prineas and J. Stamler (1986). "Characteristics and prognosis of incomplete right bundle branch block: an epidemiologic study." J Am Coll Cardiol **7**(3): 492-499.
Luna Filho, B., J. A. Bocanegra, A. Pfeferman, J. L. Andrade and E. E. Martinez Filho (1989). "[Fascicular block of the His bundle: critical approach for its identification]." Arq Bras Cardiol **53**(5): 261-265.
Magri, G. and A. Brusca (1956). "[Morphology of the human epicardial electrocardiogram in right intraventricular blocks]." Atti Soc Ital Cardiol **1**: 137-138.
Martins De Oliveira, J., M. P. Sambhi and H. A. Zimmerman (1958). "The electrocardiogram in pectus excavatum." Br Heart J **20**(4): 495-501.
Masini, V., F. Testoni and A. Farulla (1952). "[Focal blocks of the right intraventricular conduction: morphology, pathogenesis and clinical significance]." Cardiologia **21**(3): 171-187.
Massing, G. K. and T. N. James (1972). "Conduction and block in the right bundle branch, real and imagined." Circulation **45**(1): 1-3.
Moore, E. N., J. P. Boineau and D. F. Patterson (1971). "Incomplete right bundle-branch block. An electrocardiographic enigma and possible misnomer." Circulation **44**(4): 678-687.

Moore, E. N., B. F. Hoffman, D. F. Patterson and J. H. Stuckey (1964). "Electrocardiographic Changes Due to Delayed Activation of the Wall of the Right Ventricle." Am Heart J **68**: 347-361.
Pastore, C. A., P. J. Moffa, M. O. Spiritus, N. M. Tobias, A. P. de Moraes, E. Del Nero Junior, L. V. Decourt and F. Pillegi (1983). "[Fascicular blocks of the right branch. Standardization of vectorelectrocardiographic findings]." Arq Bras Cardiol **41**(3): 161-166.
Perez-Riera, A. R., C. Ferreira Filho, L. C. de Abreu, C. Ferreira, F. G. Yanowitz, F. Femenia, P. Brugada, A. Baranchuk and V. C. G. I. G. International (2012). "Do patients with electrocardiographic Brugada type 1 pattern have associated right bundle branch block? A comparative vectorcardiographic study." Europace **14**(6): 889-897.
Pileggi, F., J. Tranchesi, M. Ebaid, H. Germiniani, B. Grandisky, R. J. de and L. V. Decourt (1961). "[Vectocardiographic analysis of ventricular activation in individuals with S1 S2 S3-type electrocardiograms]." Arq Bras Cardiol **14**: 373-379.
Pressler, A. and M. Halle (2012). "[ECG diagnostics in competitive athletes. Current implications for preparticipation screening]." Herz **37**(5): 474-484.
Raunio, H., V. Rissanen, C. Jokinen and O. Penttila (1978). "Significance of a terminal R wave in lead V1 of the electrocardiogram." Am Heart J **95**(6): 702-706.
Rodriguez, R. and W. J. Kuzman (1968). "Atrial septal defect--ostium secundum variety. A review of 117 cases." Calif Med **109**(2): 105-111.
Rossi, P. (1954). "[Atypical electrocardiographic aspects of focal blocks of the right intraventricular conduction]." Cuore Circ **38**(1): 16-22.
Scherlis, L. and Y. C. Lee (1961). "Right bundle branch block following open heart surgery. Electrocardiographic and vectorcardiographic study." Am J Cardiol **8**: 780-791.
Serra, G., A. Baranchuk, A. Bayes-De-Luna, J. Brugada, D. Goldwasser, L. Capulzini, D. Arazo, A. Boraita, M. E. Heras, J. Garcia-Niebla, R. Elosua, R. Brugada and P. Brugada (2014). "New electrocardiographic criteria to differentiate the Type-2 Brugada pattern from electrocardiogram of healthy athletes with r'-wave in leads V1/V2." Europace **16**(11): 1639-1645.
Serra, G., A. Baranchuk, A. Bayes-De-Luna, J. Brugada, D. Goldwasser, L. Capulzini, D. Arazo, A. Boraita, M. E. Heras, J. Garcia-Niebla, R. Elosua, R. Brugada and P. Brugada (2015). "Base of the triangle to determine a Brugada electrocardiogram pattern." Europace **17**(3): 505.
Sodi-Pallares, D., A. Demicheli, L. Martinesi and G. A. Medrano (1964). "[Effects of Some Electrolyte Solutions on the Electrocardiographical Evolution of Acute Experimental Infarct]." Atti Soc Ital Cardiol **25**: 107-112.
Sodi-Pallares, D. and M. R. Testelli (1964). "Electrocardiography in the Diagnosis of Congenital Heart Disease." Heart Bull **13**: 24-30.
Surawicz, B., R. Childers, B. J. Deal, L. S. Gettes, J. J. Bailey, A. Gorgels, E. W. Hancock, M. Josephson, P. Kligfield, J. A. Kors, P. Macfarlane, J. W. Mason, D. M. Mirvis, P. Okin, O. Pahlm, P. M. Rautaharju, G. van Herpen, G. S. Wagner, H. Wellens, E. American Heart Association, C. o. C. C. Arrhythmias Committee, F. American College of Cardiology and S. Heart Rhythm (2009). "AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology." J Am Coll Cardiol **53**(11): 976-981.

Surawicz, B., R. Childers, B. J. Deal, L. S. Gettes, J. J. Bailey, A. Gorgels, E. W. Hancock, M. Josephson, P. Kligfield, J. A. Kors, P. Macfarlane, J. W. Mason, D. M. Mirvis, P. Okin, O. Pahlm, P. M. Rautaharju, G. van Herpen, G. S. Wagner, H. Wellens, E. American Heart Association, C. o. C. C. Arrhythmias Committee, F. American College of Cardiology and S. Heart Rhythm (2009). "AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: endorsed by the International Society for Computerized Electrocardiology." Circulation **119**(10): e235-240.

Tapia, F. A. and W. L. Proudfit (1960). "Secondary R waves in right precordial leads in normal persons and in patients with cardiac disease." Circulation **21**: 28-37.

Uhley, H. N. and L. Rivkin (1961). "Electrocardiographic patterns following interruption of main and peripheral branches of the canine right bundle of His." Am J Cardiol **7**: 810-816.

van Dijk, G. P., E. van der Kooi, A. Behin, J. Smeets, J. Timmermans, S. van der Maarel, G. Padberg, N. Voermans and B. van Engelen (2014). "High prevalence of incomplete right bundle branch block in facioscapulohumeral muscular dystrophy without cardiac symptoms." Funct Neurol **29**(3): 159-165.

Zhong-qun, Z., K. C. Nikus, A. R. Perez-Riera and W. Chong-quan (2014). "Electrocardiographic findings in accessory right precordial leads in adults and seniors with notched S waves in lead V1-a preliminary study." Ann Noninvasive Electrocardiol **19**(3): 234-240.