Episode of syncope in hypertensive elderly patient with sudden ECG pattern change

Caucasian hypertensive patient, 75 years old, 1.85m high, 90kg, regular use of medications (olmesartan 40 mg / day + hydrochlorothiazide 12.5 mg / day + amlodipine besylate 5mg / day). No other risk factor. He walks daily one hour.

Reason for consultation: fleeting sudden loss (≈ 20 seconds) and temporary consciousness and postural tone with complete recovery, unrelated to swallowing, urination, defecation, coughing or positional changes (orthostatic). Absence of auras, premonitory symptoms, mental confusion after the event, or focal neurological signs.

Physical examination: symmetrical chest without bulging, jugular venous pulse of normal characteristics, apical impulse located on the sixth left intercostals space 1 cm to the left of the midclavicular line is covered with half past one digitalis pulp.

Regular heart rate two times, HR 60 bpm, leaflets rhythmic phonetically normal, without murmurs. BP:135/80 mmHg, permeable peripheral pulses.

Lungs: Nomal vesicular breath sounds (murmur) without early or late crackles(Early inspiratory and expiratory crackles are the hallmark of chronic bronchitis. Late inspiratory crackles may mean pneumonia, CHF, or atelectasis.)

Abdomen: nothing of note

Absence of edema in the lower limbs.

Questions:

Which electrocardiographic diagnosis obtained in Holter-24 record in the first and second morphology?

What is the underlying evolutionary dromotropic disorder?

Which is the appropriate approach?

Português

Episodio sincopal em idoso hipertenso com mudança súbita do padrão ECG

Paciente hipertenso, branco, 75 anos, 1,85m, 90kg, em uso regular de medicamentos (olmesartana 40mg/dia + hidroclorotiazida 12,5mg/dia + besilato de anlodipino 5mg/dia), ausência de outro fator de risco, prática regular de caminhadas diárias de 1h.

Motivo da consulta: perda súbita fugaz (≈ 20 segundos) e temporária da consciência e tono postural com recuperação completa, não relacionada a deglutição, micção, defecação, tosse ou mudanças posicionais (ortostatismo). Ausência de auras, sintomas premonitórios, confusão mental após o evento, ou sinais neurológicos focais.

Exame físico: tórax simétrico sem abaulamentos, pulso venoso jugular de características normais, ictus cordis localizado no sexto espaço intercostal esquerdo 1cm a esquerda da linha hemiclavicular que é coberto com uma e meia polpa digitálica, ritmo cardíaco regular de dois tempos, FC 60bpm, bulas rítmicas normofonéticas, sem sopros. Pressão arterial 135/80 mmHg, pulsos periféricos permeáveis.

Pulmões: murmúrio vesicular presente.

Abdômen NDN.

Ausência de edema nos membros inferiores.

Perguntas:

Qual o diagnóstico eletrocardiográfico obtido no registro de Holter-24h na primeira e segunda morfologia?

Qual o distúrbio dromótropo evolutivo subjacente?

Qual a abordagem adequada?

Raimundo Barbosa-Barros, MD.
Chief of the Coronary Center of the Hospital de Messejana
Dr. Carlos Alberto Studart Gomes. Fortaleza - Brazil

Colleagues opinions

He has a baseline LBBB pattern which changes to Left IVCD with past fascicular block pattern. Wonder about infra nodal disease and would recommend an exercise study and EP study if necessary.

Melvin M Scheinman
Department of Cardiac Electrophysiology, University of California San Francisco, San Francisco, California, USA.
scheinman@medicine.ucsf.edu

Spanish: Querido Potro: Los complejos QRS de ambos ECGs tienen una duración similar que esta en alrededor de 170 mseg; y en el plano precordial el voltaje de la onda S en ambos es máxima en V2 y muy profunda. En un BRI que tiene una onda S tan profunda en V2 el eje de la misma está en casi los -90º sobre el eje de Y, dicho desplazamiento se debe a: Hipertrofia septal, agrandamiento del VD, necrosis anterior. En este caso en donde teóricamente me están descartando los dos últimos diagnósticos, lo tendríamos que atribuir a hipertrofia septal. Qué pasa con los bloqueos fasciculares derechos + un BRI. En pacientes con aberrancias sin cardiopatía se modifican las fuerzas iniciales del complejo QRS, porque se activa el VD de una manera distinta. Después la activación del VI se realiza como la que uno observa en un BRI. Vamos a suponer que uno de los trazados es un BRI un tanto atípico por su comienzo y las fuerzas máximas en el plano precordial. El otro ECG que es? Tiene la misma duración pero aparentemente hay fuerzas del VD que le ganan a las del VI: desapareció el BRI y se pusieron de manifiestos trastornos de conducción miocárdicos muy importante del tracto de salida del VD que tienen la misma duración? Esto es un estudio Holter: cambio la posición del paciente el cual adoptó el decúbito lateral derecho por lo cual tengo esta imagen? Lo que yo hago con estos pacientes y tratar de reproducir el hallazgo. Espero tu opinión del caso.

Afectuosamente Isabel Konopka MD

English: Dear Andrés, the QRS complexes of both ECGs have a similar duration as this in around 170 ms; and in the precordial plane, S wave voltage in both is maximal in V2 and very deep. In an LBBB with an S wave as deep in V2, the axis of it is at almost -90° on the Y axis, and such shift is due to: septal hypertrophy, RVH, anterior necrosis. In this case in which in theory, the last two diagnoses have been ruled out, we should attribute it to septal hypertrophy. What happens with right fascicular block + LBBB? In patients with aberrations without heart disease, QRS complex initial forces are modified because the RV activates differently. Later the activation of the LV happens as those observed in an LBBB. Let's assume that one of the tracings is a somewhat atypical LBBB considering its start and the maximal forces in the precordial plane. What is the other ECG? They have the same duration but apparently there are RV forces that win over the LV ones: did the LBBB disappear and are myocardial conduction disorders manifest, very important in the RVOT that has the same duration? This is a Holter study: did the position of the patient change, who adopted a right lateral decubitus so this image is produced? What I do with these patients is trying to reproduce the finding. I remain waiting for your opinion on the case.

Warm regards, Isabel Konopka

Dear Andrés: my knowledge of vectorcardiography being very rudimentary, I will give you my impression based on the clinical symptomatology and ECG tracings.
The clinical story is very suggestive of a "true syncope" related to either severe brady or tachy episode.
The ECG's highly suggests paroxysmal syncopal complete AV block with the following conduction disturbances:
CLBBB on the left 12-lead Holter ECG tracing
MASKED RBBB + left posterior AV block + slightly shorter PR (i.e. shorter HV) on the right Holter ECG tracing.
In summary: severe "trifascicular" or "bilateral" bundle branch block
Treatment: DDD pacemaker
Warmest regards and Shabbat shalom from Israel

Castellano/Spanish

Queridos Andrés: mi conocimiento de Vectorcardiografía es muy rudimentario, por lo tanto te daré mi impresión basado en la sintomatología y trazados ECG clínicos.
La historia clínica es muy sugestiva de un "verdadero síncope" relacionado con cualquiera severa brady o episodio taqui. El ECG es muy evocador de bloqueo AV completo paroxístico sincopal con los trastornos de la conducción siguientes: BRI a la izquierda de 12 derivaciones Holter ECG trazado, Bloqueo de rama ENMASCARADO + BRD tarde dejó bloqueo AV + PR ligeramente más corto (es decir, HV más corto) a la derecha trazado Holter ECG.
En resumen: bloqueo "trifascicular" grave o bloqueo de rama "bilateral"
Tratamiento: marcapasos DDD
Saludos cordiales y Shabat shalom desde Israel

Bernard Belhassen
Cardiac Electrophysiology Laboratory, Department of Cardiology, Tel-Aviv Medical Center, Tel Aviv, Israel.

Spanish Querido Andrés: La variación de la morfología y eje eléctrico de la grabación del Holter intento comprenderla de acuerdo a lo referido de que el paciente no padece miocardiopatía demostrada.

En el primer registro del Holter los intervalos RR son de 1023 y 1015 milisegundos, presenta un retraso de la conducción intrauricular y un intervalo PR prolongado de 240 mseg. BCRI con eje eléctrico en + 45°.

En el segundo registro una leve variación de los intervalos RR de 1062 y 1063 mseg. Este aumento de los intervalos RR, es decir levemente más bradicardico modifica la conducción intrauricular levemente y disminuye el intervalo PR en 20 mseg y la duración del QRS en 10 mseg (por lo que llego a medir), y modifica esto al mejorar levemente la conducción a nivel Hisiano que la activación eléctrica alcance el sistema de conducción ventricular con mayor tiempo de recuperación de la conducción del haz de His y causa la variación de la morfología del mismo. Desviándose el eje eléctrico a 180° y presentando en V5 y V6 una onda S terminal.

No presenta criterios de bloqueo del fascículo posterior izquierdo. No me impresiona que sea posicional ya que no varia el eje de la onda P.

Mi impresión es que disminuye levemente el grado de BCRI y se evidencia un bloqueo de la rama derecha por la mejoría en la conducción.

Pensaría en enfermedad del sistema de conducción, esto evidenciado por los cambios en la capacidad de conducir el impulso eléctrico con pequeñas variaciones de los intervalos RR.

Un cordial saludo

Martín Ibarrola MD Buenos Aires Argentina

Area de “puerto Madero” donde se come la mejor carne del mundo

Madero port area where you can taste the best barbecue around the word!

English

Dear Andrés,

I am trying to understand the variation in morphology and the electrical axis of the Holter recording according to the patient stating he does not have a proven cardiomyopathy.

In the first Holter recording, the RR intervals are of 1023 and 1015 ms; he presents a delay of intra-atrial conduction and a prolonged PR interval of 240 ms. CLBBB with electrical axis in +45°.

In the second recording a mild variation of the RR intervals of 1062 and 1063 ms. This increase in RR intervals, i.e. slightly more bradycardic, mildly changes intra-atrial conduction and decreases the PR interval in 20 ms and the QRS duration in 10 ms (as I can measure it), and this changes when conduction at His level improves slightly, as electrical activation reaches the ventricular conduction system with a longer recovery time of His bundle conduction, causing the variation in morphology. The electrical axis shifts at 180° and presents terminal S wave in V5 and V6.

It does not present LPFB criteria. It does not seem to be positional, as the P wave axis does not vary.

My impression is that the degree of CLBBB slightly decreases, and RBBB is manifest by the improvement in conduction.I think this is a conduction system disease, shown by the changes in the capacity to conduct the electrical impulse with small variations of RR intervals.

Kind regards,

Martín Ibarrola MD Buenos Aires Argentina

Portuguese:

Prezado Andrés algumas considerações:

1 - Paciente idoso em uso de três anti-hipertensivos ocasiona comumente hipotensão particularmente se ingeridos pela manhã, juntos. A associação de amlodipina e BRA é potencialmente hipotensora em idosos. Eu inicialmente consideraria a suspensão de um deles.

2 - Síncope em idoso com BRE é motivo avaliar o intervalo HV. Se > 100 ms sem dúvida implante de MP. O extremo desvio do eixo do QRS fala a favor de agravamento intermitente da condução atrioventricular, o que sugere, associado ao quadro de perda súbita da consciência, bloqueio AV de alto grau como causa da síncope.

3 - A modificação da ingestão de anti-hipertensivos deve ser feita independente do distúrbio da condução AV e deve ser considerada mesmo após o implante de MP. Saliento que o bloqueio da condução pode ser sugerido pelo BRE e o desvio intermitente do eixo para a direita. O implante de MP deve ser considerado independentemente da correção do uso da medicação anti-hipertensiva. As duas condições causam síncope, entretanto, pelo fato de ser súbita a perda da consciência, é mais provável ser causada pelo bloqueio cardíaco.

Caso interessante. Um abraço

Dalmo Ribeiro MD PhD Sao Paulo Brazil

English

Dear Andrés some considerations:

1 – Senior patient in use of three antihypertensive drugs frequently causes hypotension particularly if taken in the morning together. The combination of amlodipine and angiotensin II receptor antagonist is potentially hypotensive in the elderly.

2 - Syncope in elderly patients with LBBB is reason to evaluate the HV interval. If> 100 ms undoubtedly pacemaker implant is mandatory.

The extreme deviation of the QRS axis speaks in favor intermittently increase the atrioventricular conduction, which suggests, together with the sudden loss of consciousness above, high-grade AV block as the cause of syncope.

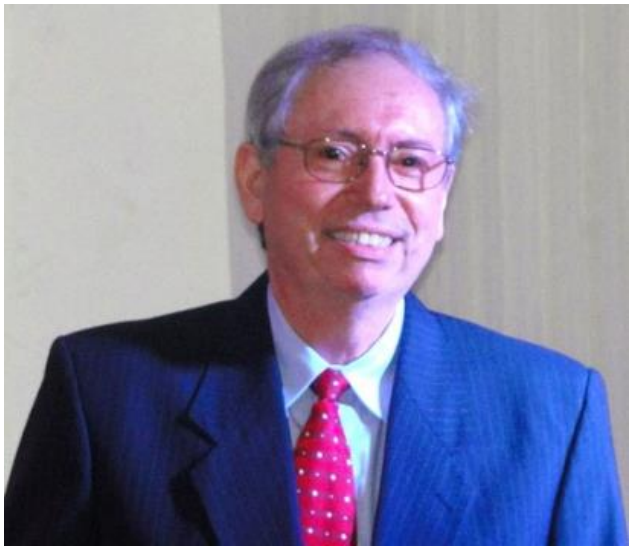
3 - Modification of antihypertensive intake should be made independent of AV conduction disorder and should be considered even after implantation of MP. Emphasize that the conduction block may be suggested by LBBB and intermittent axis deviation to the right. The MP implant should be considered regardless of the correctness of the use of antihypertensive medication. Both conditions cause syncope, however, because it is sudden loss of consciousness, it is more likely to be caused by heart block.

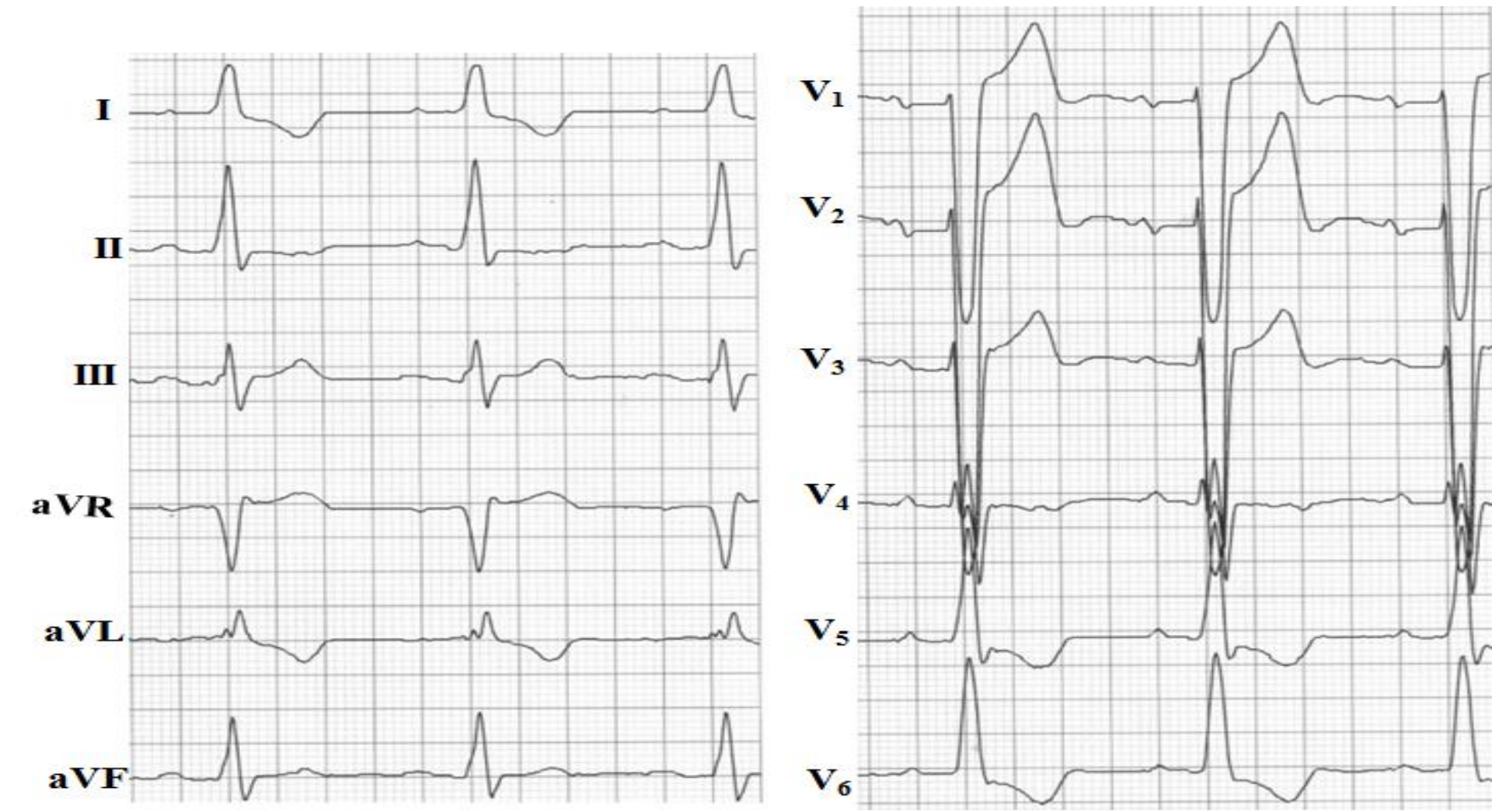
Interesting case

A hug

Dalmo Antoni Ribeiro Moreira MD PhD
Head of the Medical Section of Electrophysiology
and Cardiac Arrhythmias of Cardiology
Dante Pazzanese Institute. São Paulo Brazil

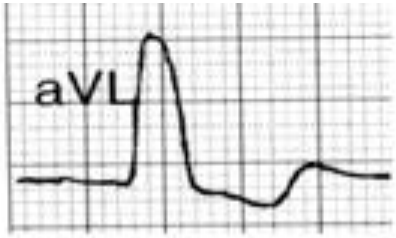
Final comments by
Andrés Ricardo Pérez-Riera
&
Raimundo Barbosa-Barros



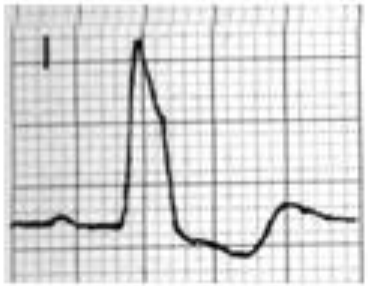


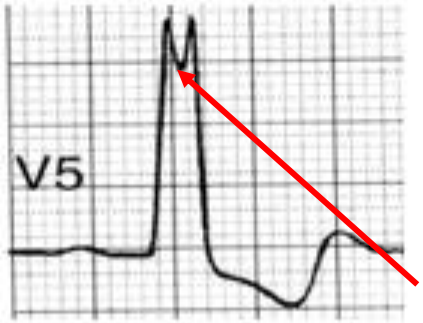
ECG diagnosis: sinus rhythm, HR 60bpm, P axis +60° to back, P duration 130ms (prolonged), negative final slow component, PR interval 280ms, QRS axis +30°, QRS duration 160ms. This tracing does not meet the stricter criteria of truly LBBB following Strauss concept: QRS duration ≥ 140 ms for men and ≥ 130 ms for women, along with mid-QRS notching or slurring in ≥ 2 contiguous leads (absent in the present case). This new criteria are used mainly for Cardiac Resynchronization Therapy (CRT) (**Strauss 2011**). Clearly, this is a pseudo LBBB consequence of severe LVH. **Conclusion:** Left atrial enlargement + left ventricular enlargement/hypertrophy with strain pattern of repolarization + first degree AV block.

Truly Left Bundle Branch Block

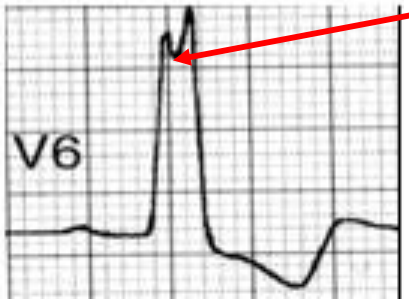


QRS duration
New criteria: ≥ 140 ms for men and ≥ 130 ms for women
Classical criteria: ≥ 120 ms



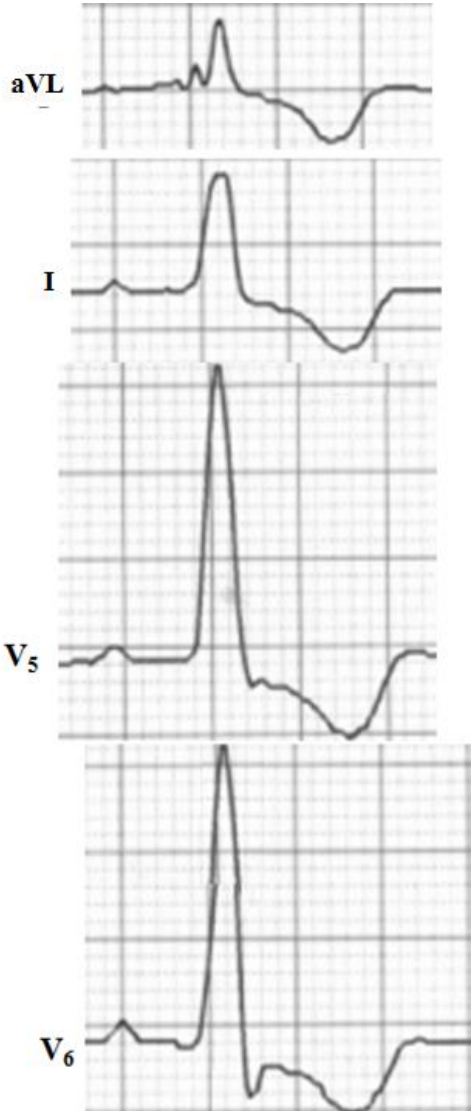


The ST- segment and T-wave opposite to a greater deflection of QRS: positive from V_1 to V_3 and negative in left leads I, aVL, V_5 and V_6: Secondary Repolarization Abnormalities with wide QRS-ST-T angle and normal ventricular gradient. The classic ventricular gradient concept introduced by Wilson et al (**Wilson 1931**).



Mid-QRS notching or slurring in ≥ 2 contiguous leads.

The present case: left leads. This is not a LBBB because there is not mid-QRS notching or slurring in at least 2 contiguous leads (**Strauss 2011**). So what is it? Answer: **N**onspecific or Unspecified **I**ntraventricular **C**onduction **D**elay/Disturbance (NICD).



Nonspecific Intraventricular Conduction Disturbance/Delay (NICD)

Definition: NICD are Intraventricular Conduction Disturbance/Delay characterized by QRS duration ≥ 110 ms in adults, greater than 90 ms in children 8 to 16 years of age, and greater than 80 ms in children less than 8 years of age without ECG criteria for complete or incomplete RBBB, LBBB, Wolff-Parkinson White or masquerading bundle branch blocks (standard, precordial or both) (**Surawitcz 2009**).

Prolonged QRS duration in a standard 12-lead ECG is associated with increased mortality in a general population, with NICD being most strongly associated with an increased risk of arrhythmic death (**Aro 2011**).

QRS duration up to 110ms or even 120ms may be seen in healthy individuals (**Surawicz 2008**).

Notching of QRS complex with normal QRS duration should not be diagnosed as NICD. In most such cases, the notched can be explained by a nealy perpendicular projection of depolarization vector inscribed at that time.

Types of NICD

Eschalier et al (**Eschalier 2015**) divide the NICD in 3 subtypes important for cardiac resynchronization therapy (CRT), but for another purposes we added three more:

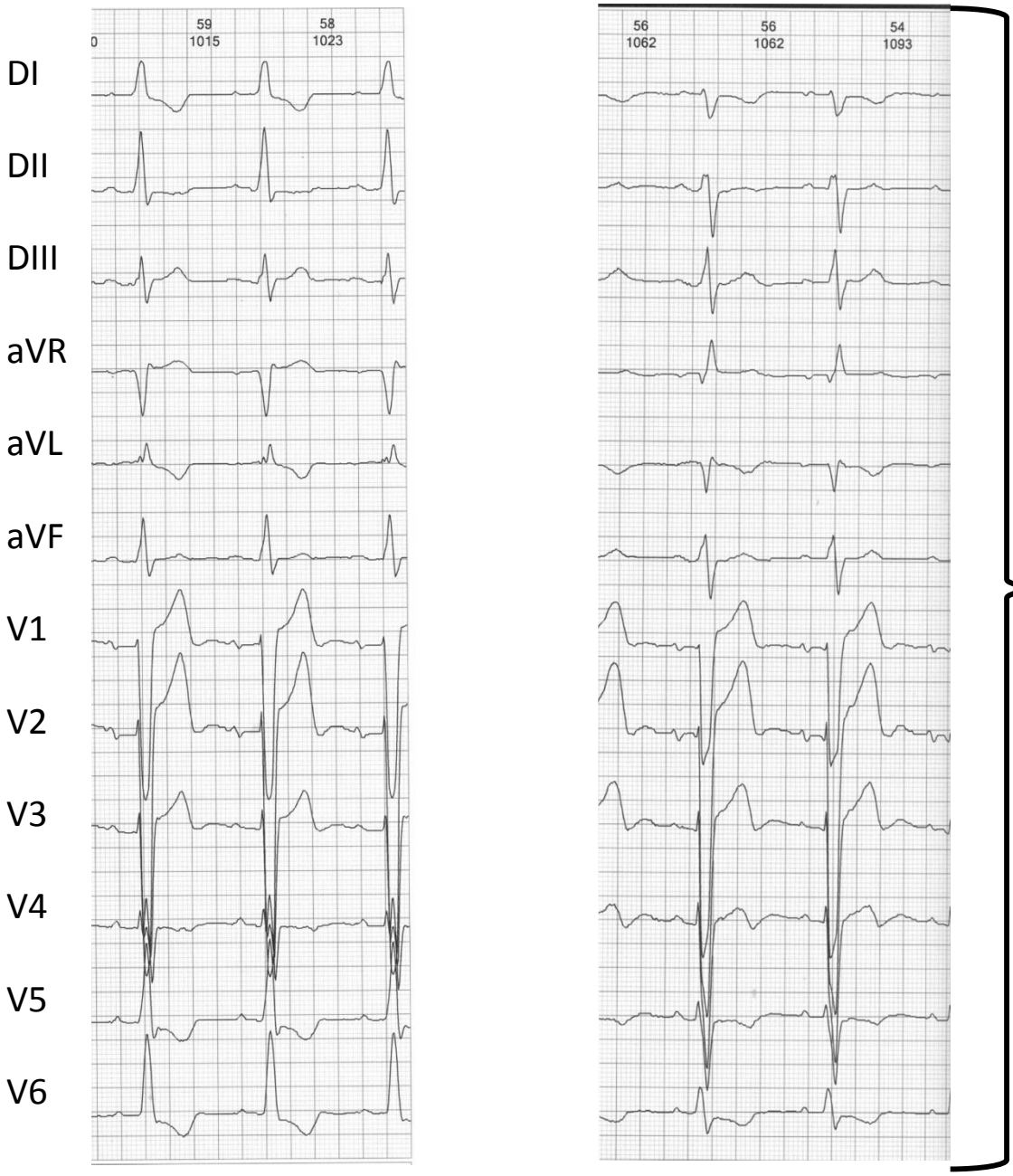
1. **Atpical LBBB:** Consequence of true LBBB associated with post- myocardial infarction The ECG shows broad and deep Q waves in multiple leads, QS complex pattern in anterior wall and QR in V5-V6, wide fragmented QRS related to scar. Ventricular activation is similar to truly LBBB;
2. **Intraventricular parietal block:** it is observed when the affected area is located beyond divisional fascicles (located in Purkinje-muscle) with preserved His branches and fascicles. Significant LVH is observed and QRS duration is consequence of increase of LV mass and intramyocardial conduction disturbance. The ECG pattern of this variant cannot be ascribed to block in a specific portion of bundle branches or fascicles of the specialized conduction system;
3. **Peri-infarct block:** Characterized by broad QRS, Q wave followed by R wave in which the R peak time is very prolonged with delayed epicardium layers activation, wide-fragmented QRS, notched appearance, stuttering, without the classical LBBB, RBBB, LAFB or LPFB pattern.

Comments: These first three variants are important for indication of cardiac resynchronization therapy (CRT). ≈ 30% of patients treated with CRT do not respond. Patients with truly Complete LBBB are best responder than ipatients with RBBB and NICD (**Eschalier 2015**). CRT consist in restoration of the normal coordinated pumping action of the ventricles by overcoming the delay in electrical conduction caused by bundle branch block Most pacemakers typically have 2 electrodes (or leads), one in the right atrium (RA) and one in the right ventricle (RV) , which permit the pacemaker to maintain the normal coordinated pumping relationship between top and bottom of the heart. These leads are connected to a battery pack (pulse generator) placed under the skin in the upper chest. In addition to the 2 leads (RA and RV) used by a common pacemaker, CRT pacemakers have a third lead that is positioned in a vein on the outer surface of the left ventricle (LV). This allows the CRT pacemaker to simultaneously stimulate the LV and RV and restore a coordinated, or synchronous, pumping action. This is sometimes referred to as biventricular pacing because both ventricles are electrically stimulated at the same time. This reduces the electrical delay and results in a narrower and more normal QRS complex on the ECG, when a prolonged QRS duration ≥ 120ms exists, but does not satisfy the criteria for either LBBB or RBBB or pre-excitation pattern, the diagnosis of NICD or conduction delay is referred. Tang et al (**Tang 2010**) randomized 1798 patients with LVEF ≤ 30%, QRSd ≥ 120 ms (or paced QRS ≥ 200 ms), and NYHA class II or III HF to receive an ICD or an ICD-CRT. After a mean follow-up of 40 months, there was a 25% relative reduction both in the primary outcome of death or HF hospitalization as well as in the secondary outcome of total mortality. This trial adds evidence to the efficacy of CRT in selected patients with mild-to-moderate HF, at the time of implementation of new guidelines. If NYHA Class II-IV HF is present, and LVEF ≤35%, ECG QRSd ≥ 120 ms in the presence of LBBB, CRT is indicated. Reevaluation of the data of CRT trials and EP findings in LBBB provided evidence that "true" LBBB requires a QRS width of ≥130 ms (in woman) and ≥140 ms (in man). In "true" LBBB, after the 40th ms of the QRS notched/slurred R waves are characteristic in minimum two of I, aVL, V1, V2, V5 and V6 leads, in addition to a ≥40 ms increase of the QRS complex, as compared to the original QRS complex. In patients with conventional wider LBBB morphology, the presence of mid-QRS notching or slurring is a strong predictor of better response to CRT (**Tian 2013**). In contrast, slowly and continuously widened “LBBB- like" QRS patterns are mostly occur in LVH or in a metabolic/infiltrative disease (intraventricular conduction disturbance) (**Préda 2013**).

Other NICDs causes:

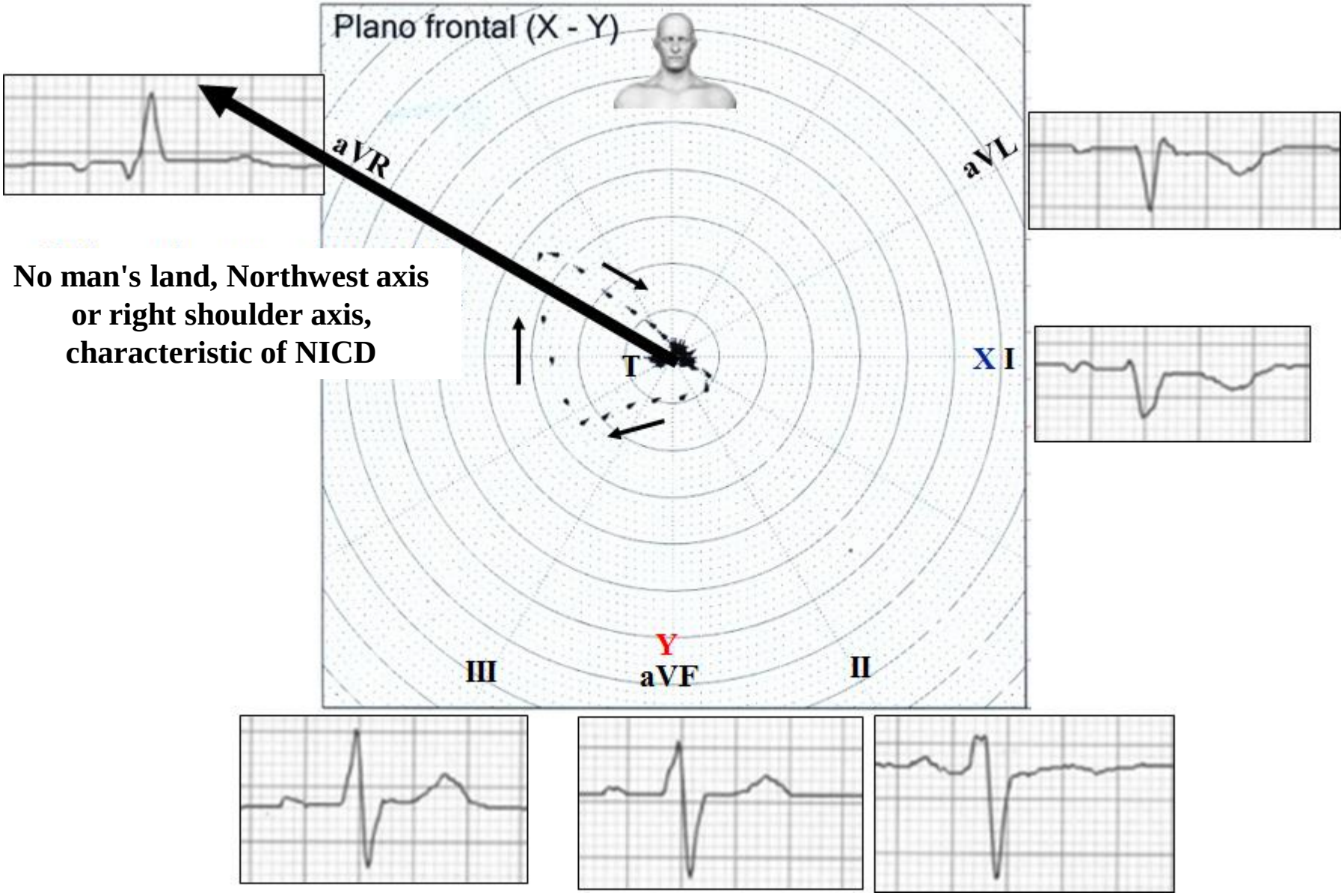
4. Complex delays in the intraventricular conduction systems associated to regional conduction slowing in the myocardium and severe His system disease and severe LVH with fibrosis (**Surawicz 2008**). Probably this is the variety of the present case;
5. Electrolyte disturbance such as severe (7.0-7.5mEq/L) and extreme (>7.6mEq/L) hyperkalemia that causes slower depolarization with widening and distortion of QRS complexes, prolonged QT interval, and frequent premature ventricular beats. When extreme hyperkalemia is present, the ECG shows absent P waves, frequent escape beats. Sinoventricular rhythm The combination of an irregular rhythm The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded). Absent P wave (may simulate atrial fibrillation or atrioventricular block), very broad and bizarre QRS complexes with "Sine QRS wave" appearance, Brugada phenocopy (**Nguyen 2011; Riera 2010**), lead I suggesting RBBB (rS pattern) and precordial leads LBBB, VT/VF or ventricular asystole with potassium concentration above 12 to 14 mEq/L;
6. Drug toxicity that induces block of sodium channels, such as tricyclic antidepressant (TCA) poisoning with characteristic delay near aVR (-150°) between +130 and -270° and consequent prominent final R wave in this lead (**Williamson 2006**), frequently followed by ST-T changes more prevalent in symptomatic patients (**Gheshlaghi 2013**). R-wave amplitude ≥3 mm and R wave/S wave ratio ≥0.7 are the most sensitive predictors of seizures or dysrhythmias in a series of patients who overdosed on TCAs (sensitivity 81% and 75%, respectively) (**Sanaei-Zadeh 2011**). In children with mean age 4.6 +/- 3.0 years with TCA poisoning, absence of an R wave in aVR ≥ 3 mm predicts seizures with a high negative predictive value, and a QRS duration ≥ 100ms predicts coma with a high positive predictive value (**Olgun 2009**). Others important drugs that cause NICD are type I antiarrhythmic drugs of Vaughan Williams mainly type IC (flecainide, propafenone, moricine) with markedly depress phase 0 and minimal effect on repolarization

Holter-24h monitoring obtained in two different recording times



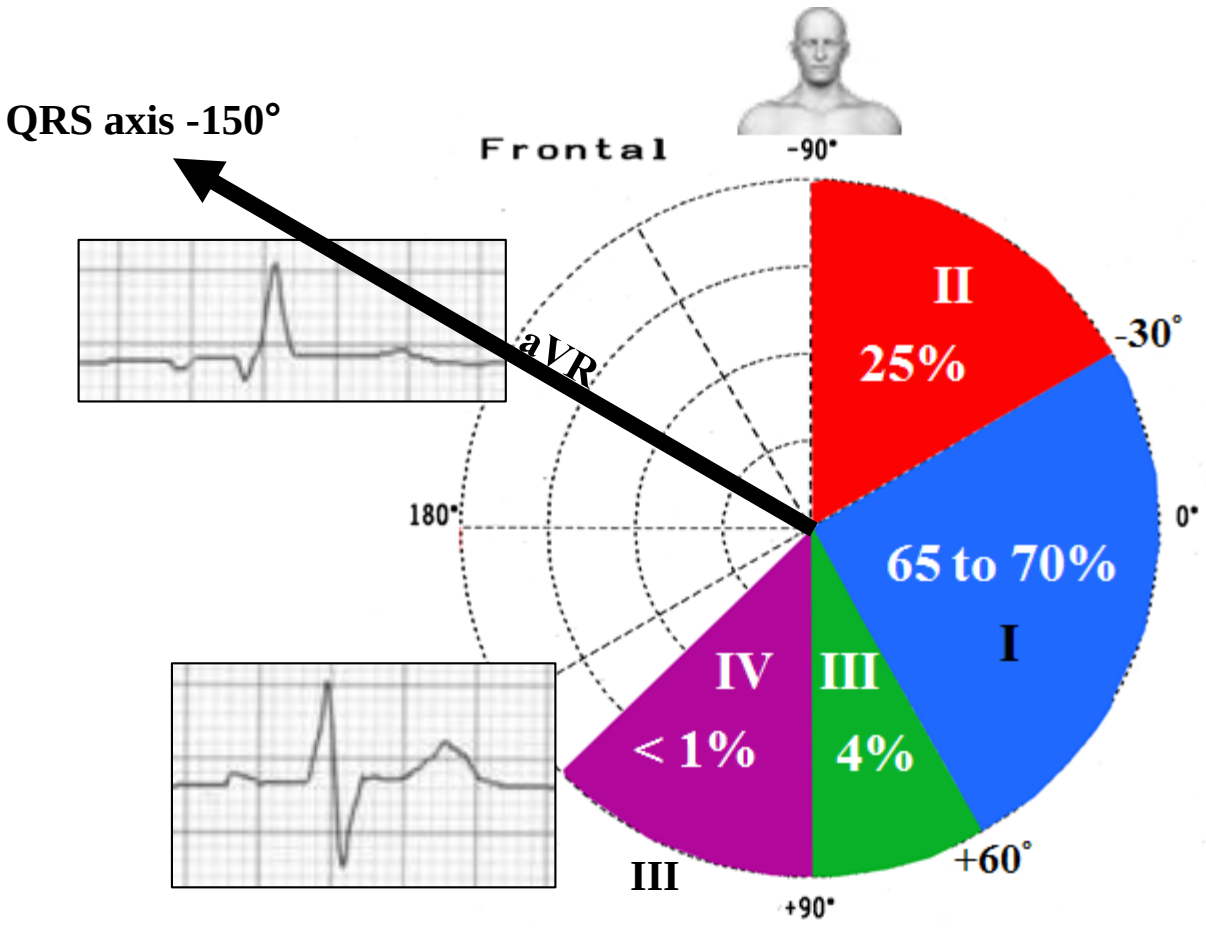
An ECG / VCG correlation shown in the next three slides.

Frontal Plane

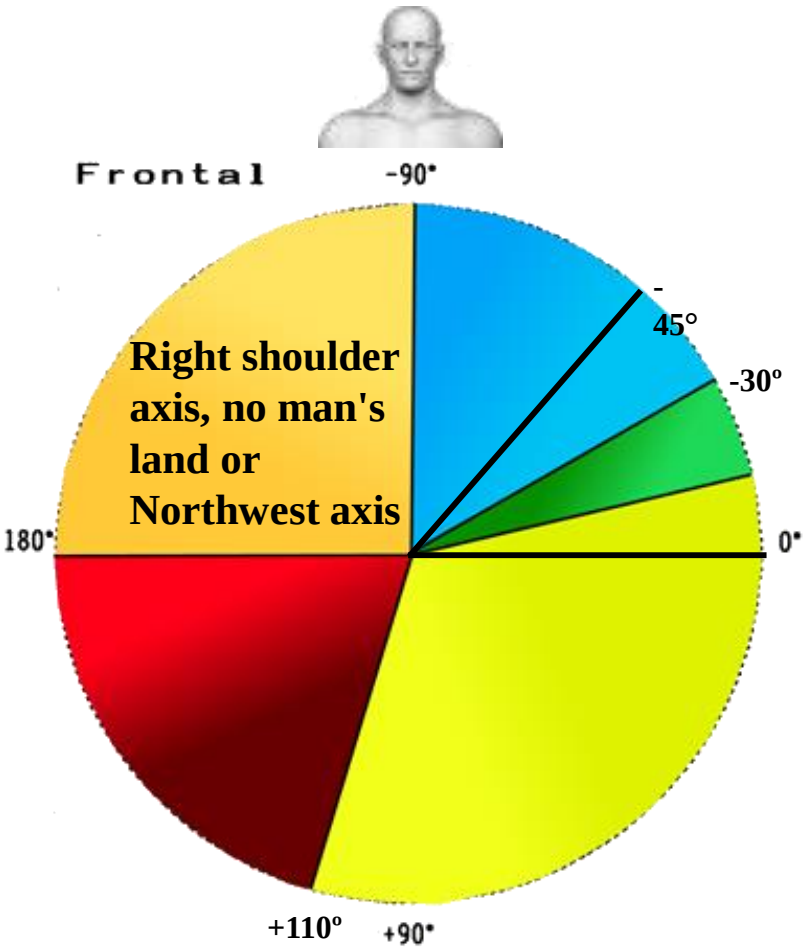


No man's land, Northwest axis or right shoulder axis, characteristic of NICD

QRS axis in the present case and percentage in truly LBBB



QRS axis (SÂQRS) in the Frontal Plane without Bundle Branch Block



Normal QRS axis between -30º and +110º

Minimal left axis QRS deviation or normal*

Extreme left axis QRS deviation between -30º and -90º. It is observed in LAFB, LVH + LAFB, inferior MI. Between -45º and -90º is criteria of LAFB.

Right axis QRS deviation. QRS axis between -+110º and ±180º. It is observed in RVH, LPFB, lateral MI asthenic biotype.

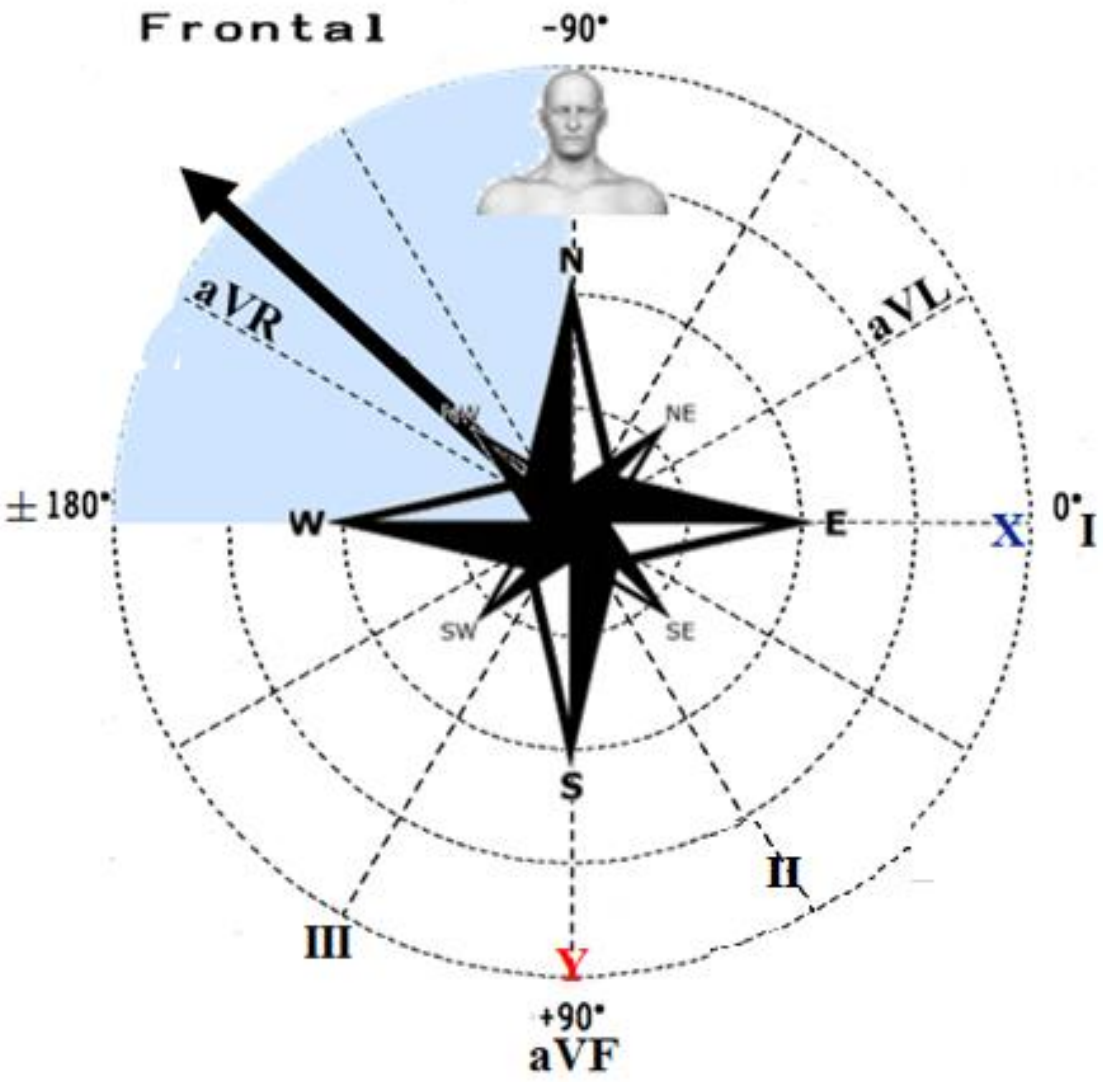
Extreme right axis QRS deviation or right shoulder axis, no man's land or Northwest axis. Negative QRS complexes in lead I and negative QRS complexes in lead aVF. See next slide.

*Note: Axes between 0^0 and -30^0 may be observed in endomorphs and pregnant women. When the axis is between 0 and -30 degrees, it is sometimes referred to as a physiological (as opposed to pathological) left axis deviation.

Axis of QRS in LVH in the frontal plane is considered with left axis deviation when ≥30º. Romhilt-Estes Score (**Romhilt 1968**).

Possible causes of QRS axis on top right quadrant between -90° and ±180°

No man's land, Northwest axis or right shoulder axis



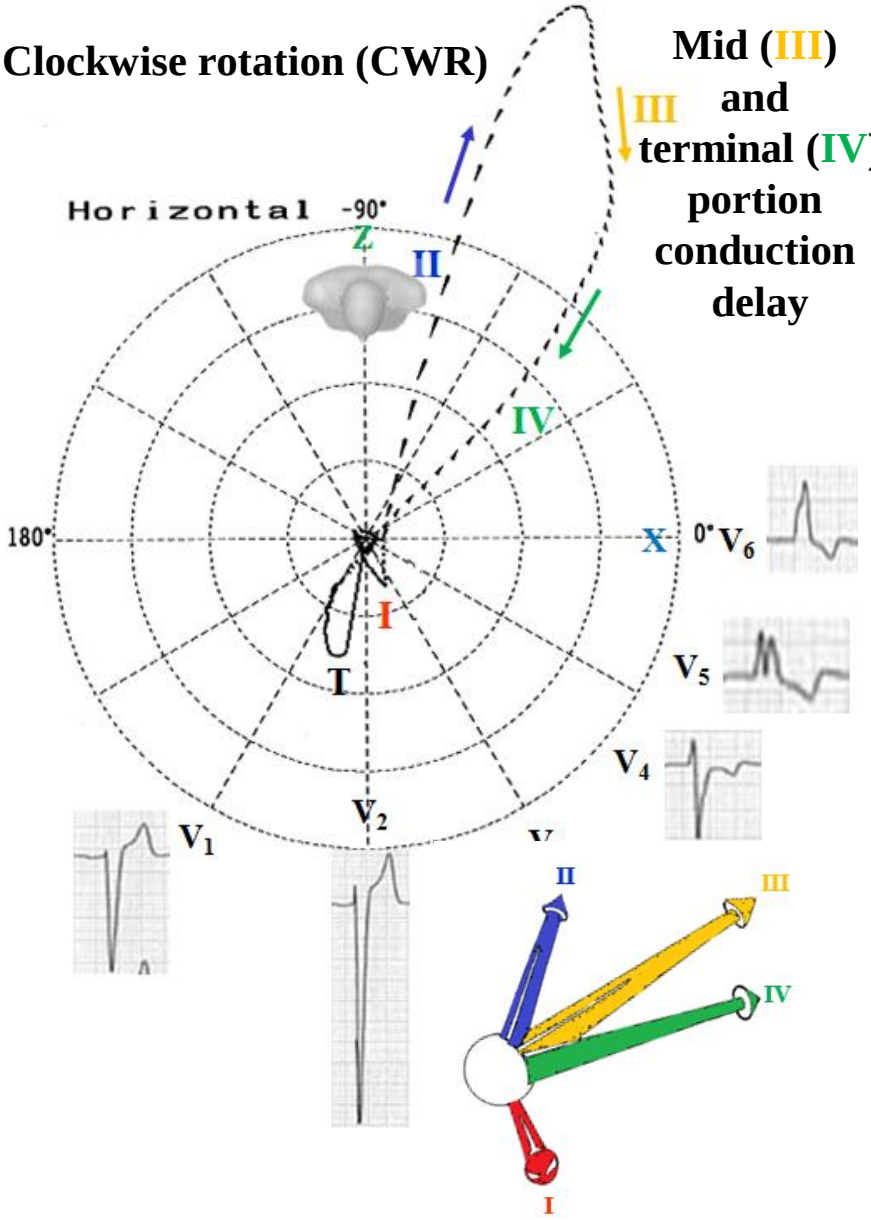
Causes of extreme deviation from the axis in the upper right quadrant on FP

- Emphysema
- Hyperkalemia
- Tricyclic antidepressant poisoning
- Other NICDs: Complex delays in the intraventricular conduction systems associated to regional conduction slowing in the myocardium by consequence of severe His system disease and severe LVH (the present case)
- Accidental exchange of electrodes
- Artificial pacemaker
- Divisional right bundle branch block
- Premature ventricular beats
- Ostium atrioventricularis comunis
- Ventricular Tachycardia

Truly VCG complete LBBB

Clockwise rotation (CWR)

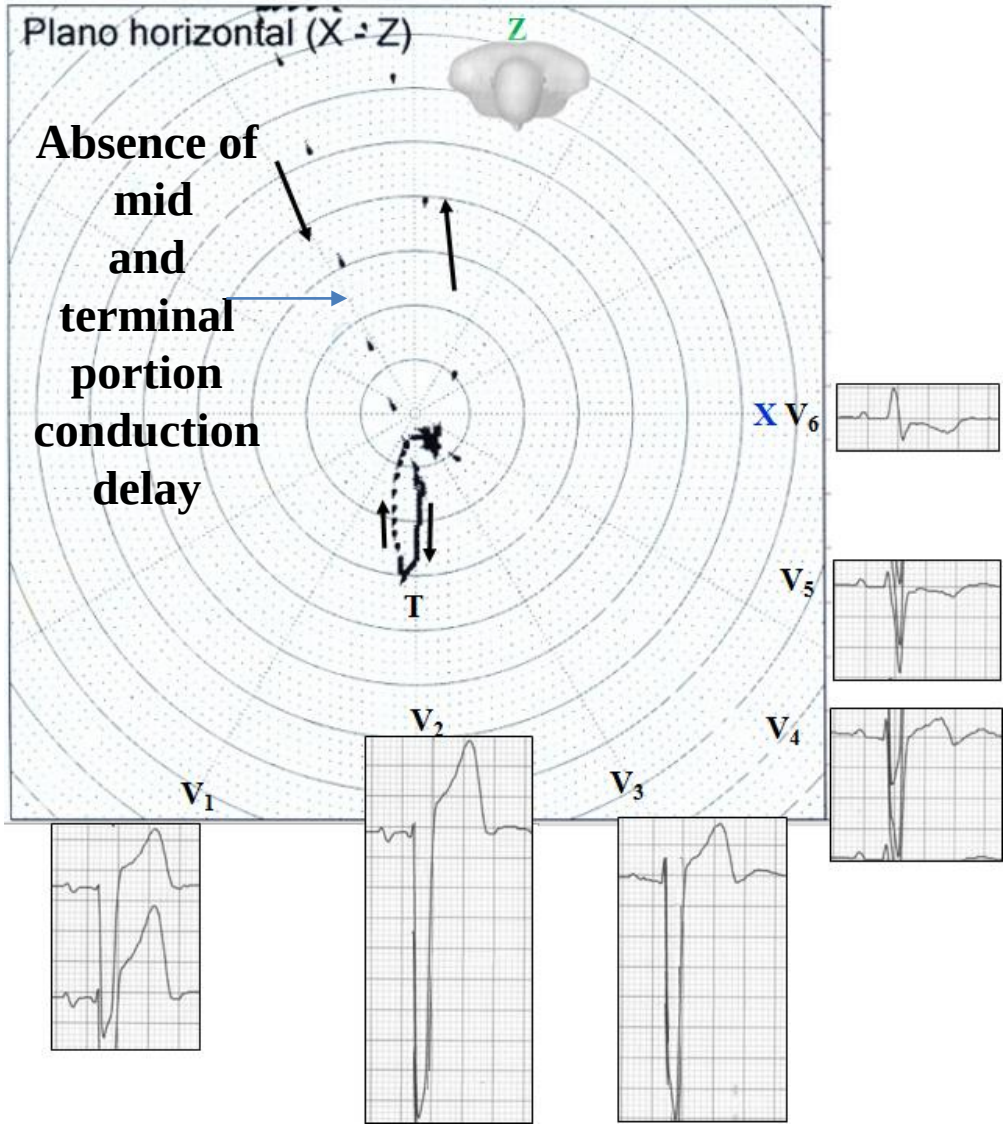
Mid (III) and terminal (IV) portion conduction delay



The present case: pseudo LBBB: NICD

Counterclockwise rotation (CCWR)

Absence of mid and terminal portion conduction delay



VCD differential diagnosis between truly complete LBBB and Nonspecific Intraventricular Conduction Delay/Disturbance (NICD)

	Truly complete LBBB	NICD
QRS loop rotation of the main body in the HP	Always clockwise in uncomplicated genuine LBBB. CCW rotation may indicate parietal CLBBB or LBBB complicated with lateral infarction or severe LVH.	Counterclockwise. Severe LVH.
Presence and location of conduction delay on QRS loop	Always in the mid and terminal portion	Absent
Efferent and afferent limb relationship in the HP	The efferent limb located to right related afferent limb.	The efferent limb located to left related afferent limb.
QRS loop shape in the HP	Narrow, long, and usually in 8.	Frequently bizarre.
Magnitude of the max QRS vector	Always > 2mV	Sometimes > 2mV

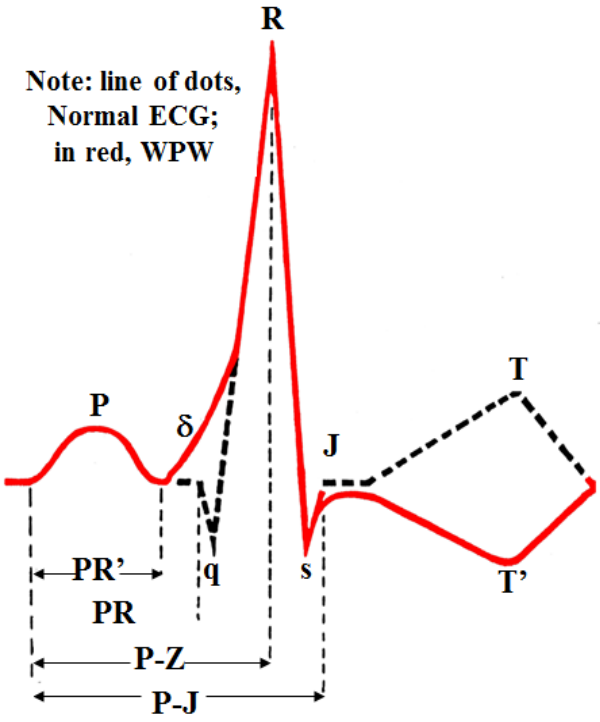
Conduction delay location in VCG

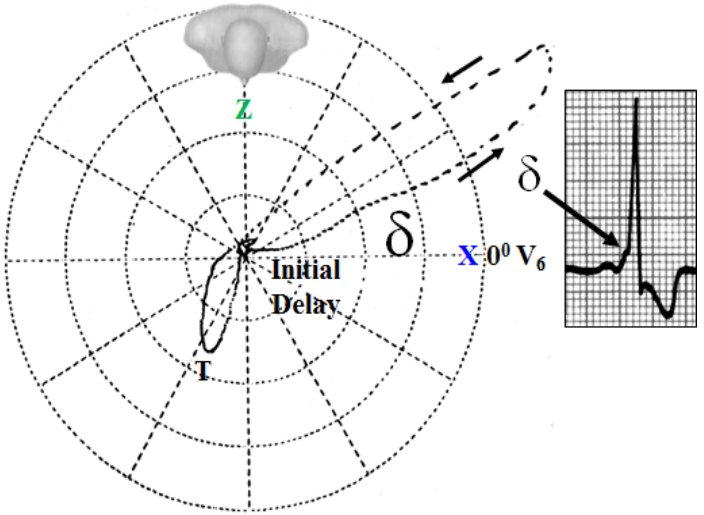
The greater or the lesser distance between dashes indicates the greater or the lesser conduction velocity. Thus, when the dashes are very close to each other, it indicates the presence of conduction delay. To consider the phenomenon as true, it is necessary for it to be evident in at least 2 planes!!!!.

Separate dashes = more dromotropism major velocity

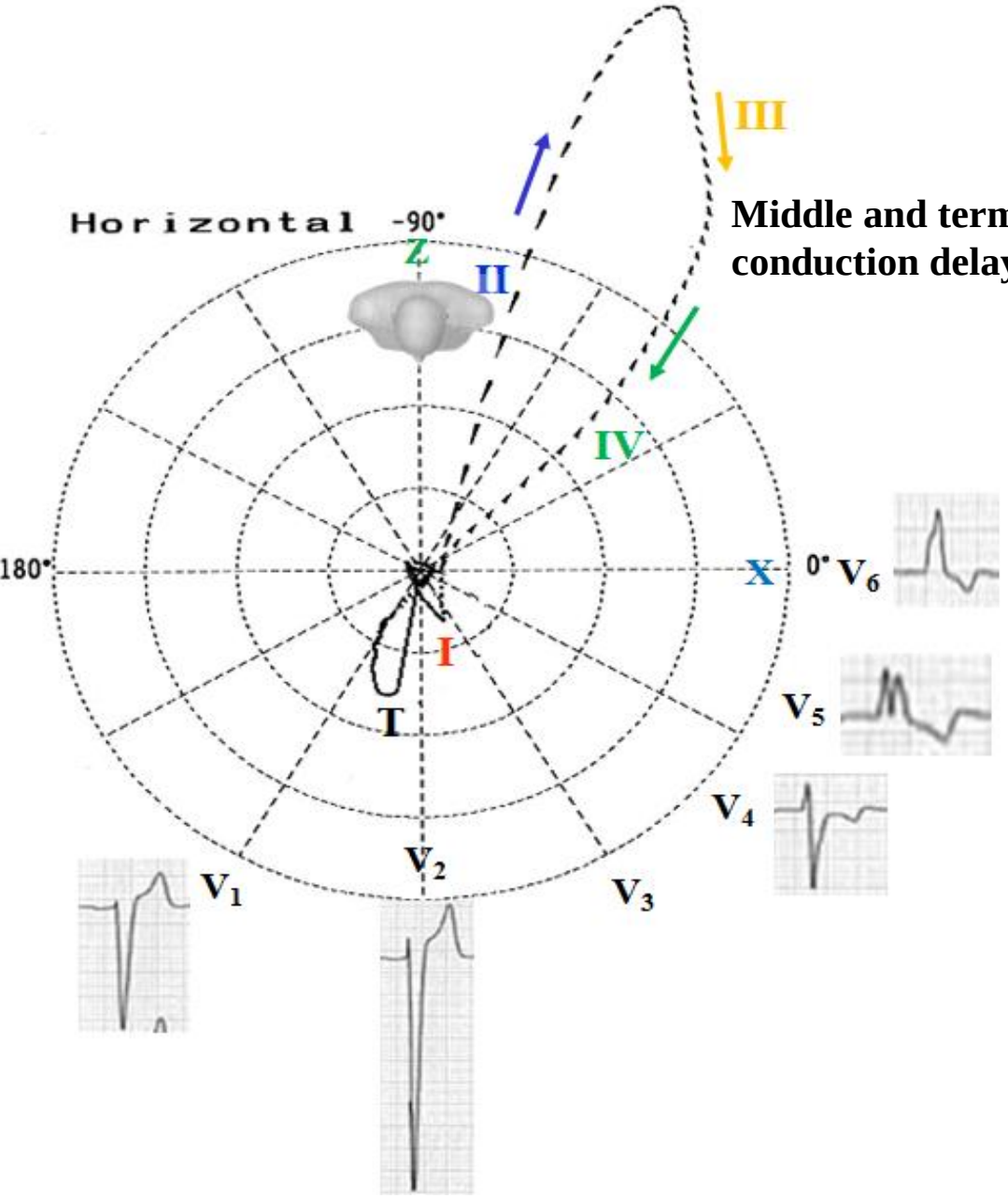
Very close dashes = less dromotropism or conduction delay

If the QRS loop has conduction delay (very close dashes) at the initial portion, ventricular pre-excitation is present (WPW pattern).

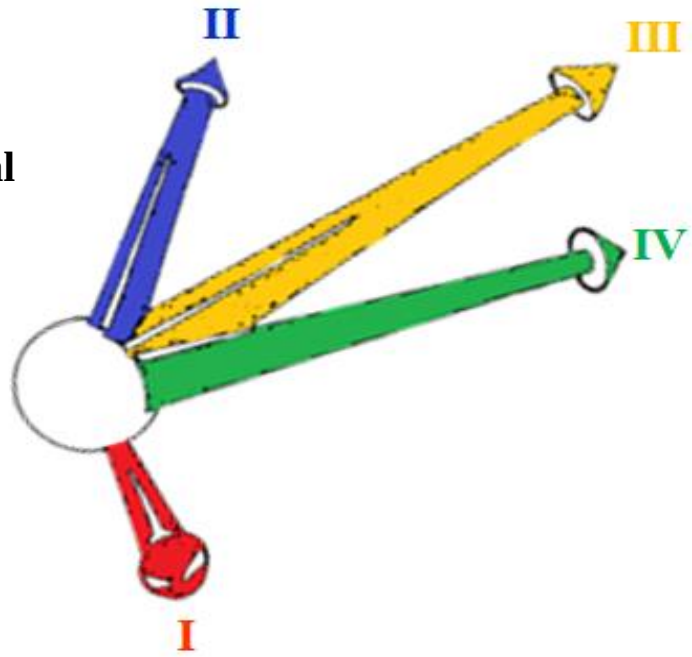




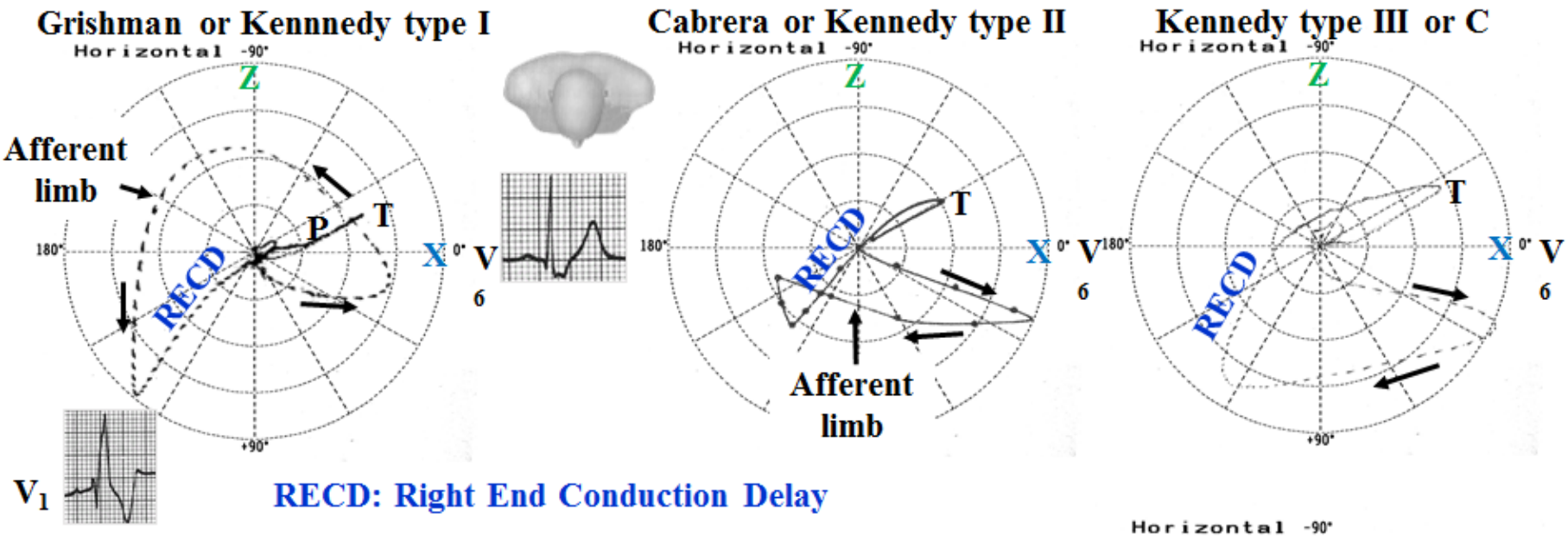
If the QRS loop has conduction delay (very close dashes) at the middle (III) and terminal portion (IV), truly LBBB is present.



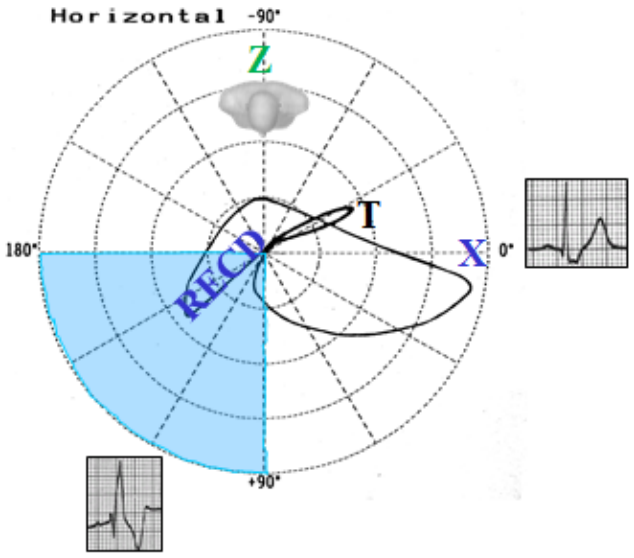
Middle and terminal conduction delay



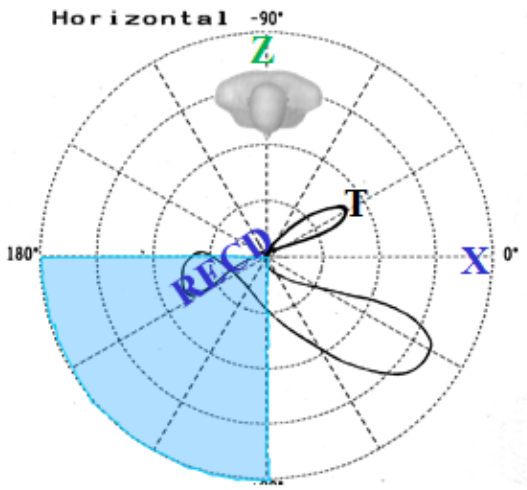
If the QRS loop has conduction delay (very close dashes) at the terminal portion, truly RBBB is present.



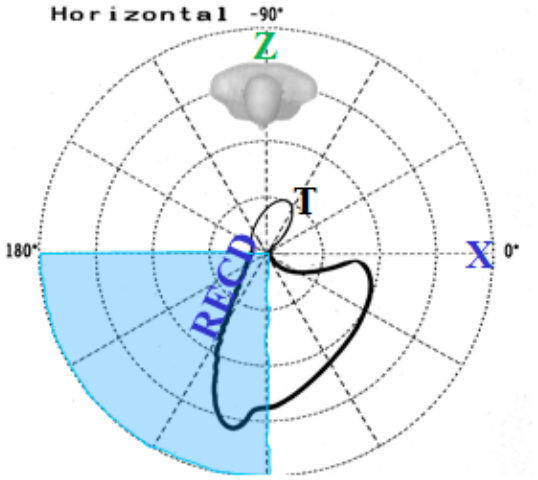
RECD: Right End Conduction Delay



Right Anterior Quadrant

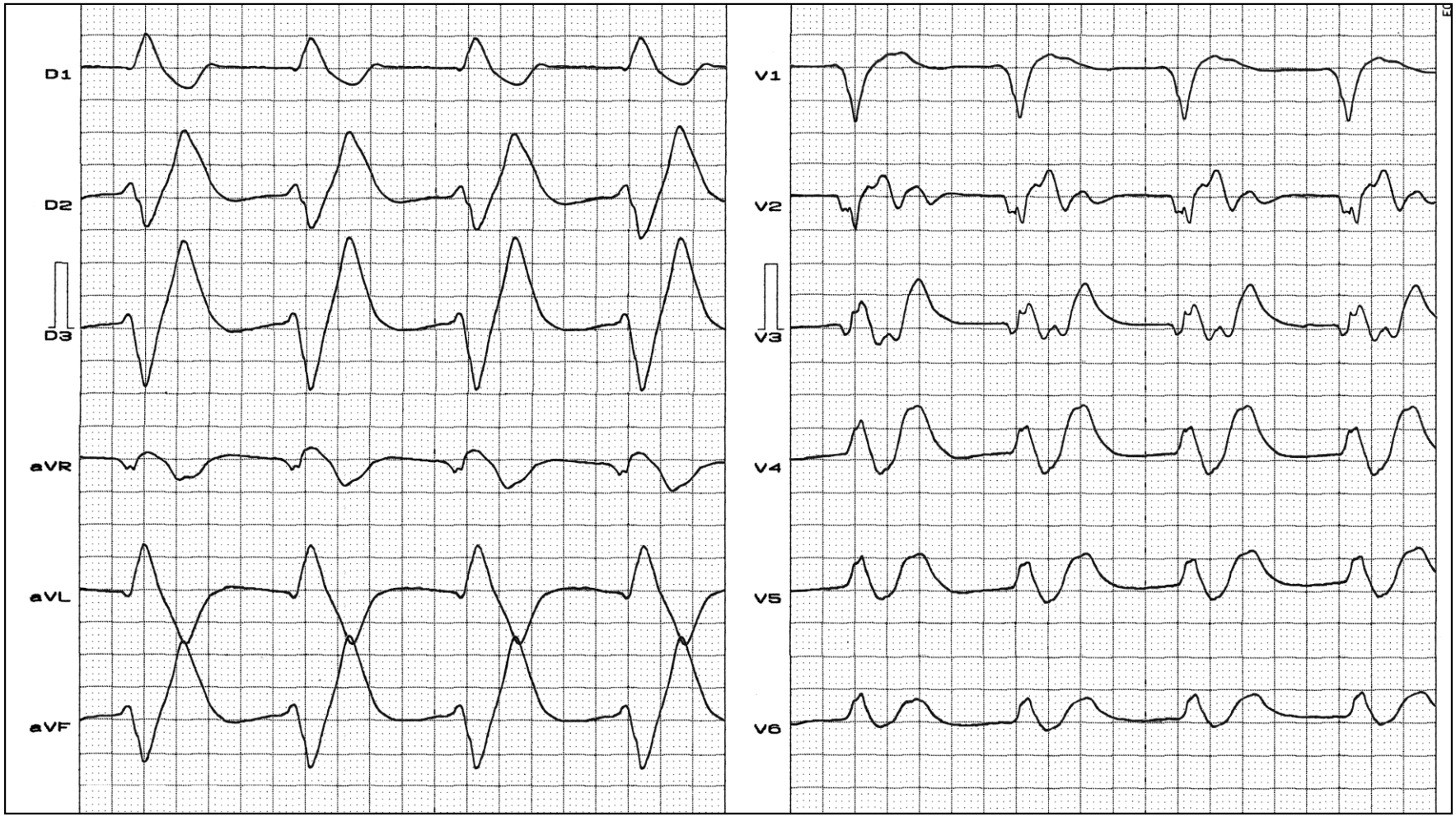


We find type II in ASD, PS, in COPD and more rarely in chronic Chagasic myocardits.



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

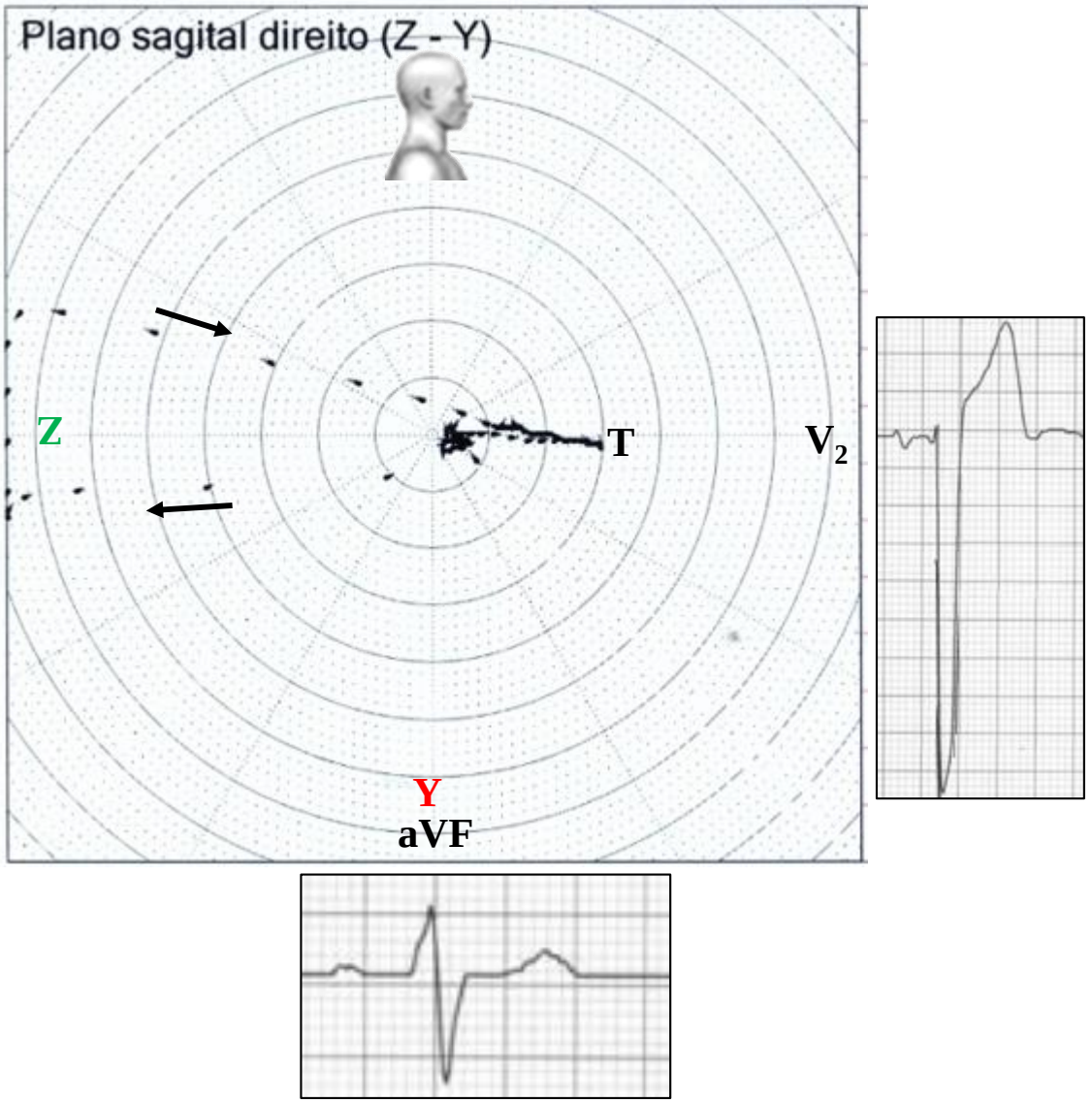
If the QRS loop has conduction delay (very close dashes) at the initial, middle and terminal portion, NICD is present.



Clinical diagnosis: chronic renal insufficiency in dialysis. The patient delayed 72 hours the dialysis session. Severe hyperpotasemia of 9 mEq/L.

ECG diagnosis: absence of P wave, sinoventricular rhythm, HR 57 bpm, morphology of bizarre intraventricular severe disorder (QRSd: 240 ms) that is similar to complete left bundle branch block. T waves with polarity matching with QRS from V3 to V6. Convergence of QRS with T wave that outlines smooth diphasic wave or sine curve.

Right Sagittal Plane



Absence of mid and terminal conduction delay in the QRS loop. This is not compatible with truly LBBB.
Very deep S V2 wave (48mm!!!) indicating severe LVH.

Electrocardiographic classification criteria for Left Bundle Branch Block

I- According to the degree:

1. Criteria (most used in the literature):

a) *Incomplete LBBB:* Incomplete Left Bundle Branch Block (QRS duration from 90 to 110 ms)

b) *Complete LBBB:* Complete Left Bundle Branch Block (QRS ≥ 120 ms) in adults.

c) Stricter criteria for complete LBBB: QRS duration ≥ 140 ms for men and ≥ 130 ms for women, along with mid-QRS notching or slurring in ≥ 2 contiguous leads. This new values are used for Cardiac Resynchronization Therapy (CRT) (**Strauss 2011**)

2. Criteria from the Mexican School (Sodi 1964):

a) 1st degree left bundle branch block;

b) 2nd degree left bundle branch block: a & b correspond to incomplete LBBB;

c) 3rd degree left bundle branch block or complete LBBB.

- Complete LBBB by classical criteria: QRS duration ≥ 120ms
- Stricter criteria QRS duration ≥140 ms (men) or 130 ms (women), QR or rS in leads V1 and V2, and mid-QRS notching or slurring in ≥2 of leads V1, V2, V5, V6, I and aVL.

3. Criteria from the Spanish School (Bayés de Luna 2007). Global left ventricular blocks:

a) Advanced left bundle branch block (ALBBB) or third degree (equivalent to CLBBB; QRS duration ≥ 120 ms),

b) Non-advanced global left ventricular blocks:

- First degree LBBB (partial) corresponds to types I and II of Mexican school: isolated R in V6 with more or fewer slurring but QRS duration < 120 ms.
- Intermittent or second degree LBBB: corresponds to special type of ventricular aberrancy.

Electrocardiographic classification criteria for Left Bundle Branch Block

II- According to topography:

a) Pre-divisional (90%) QRSD = 120 to 160 ms

- Of the left His bundle;
- Of the main stem of the left bundle branch;

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

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

c) Parietal, global Purkinjian, diffuse intraventricular, intramyocardial or intramural (in the Purkinje-muscle union). Characterized by: wider QRS, clockwise rotation of the QRS loop in the HP. In general, they point out greater myocardial involvement. Nowadays, this one is considered NICD (intraventricular parietal block) (**Eschalier 2015**).

III- According to steadiness:

a) Permanent or definite: most of them.

b) Intermittent or of second degree that could be:

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

  - Tachycardia-dependent or in “phase 3”;
  - Bradycardia-dependent or in “phase 4”.

- **Independent from heart rate:**

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

Electrocardiographic classification criteria for Left Bundle Branch Block

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

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

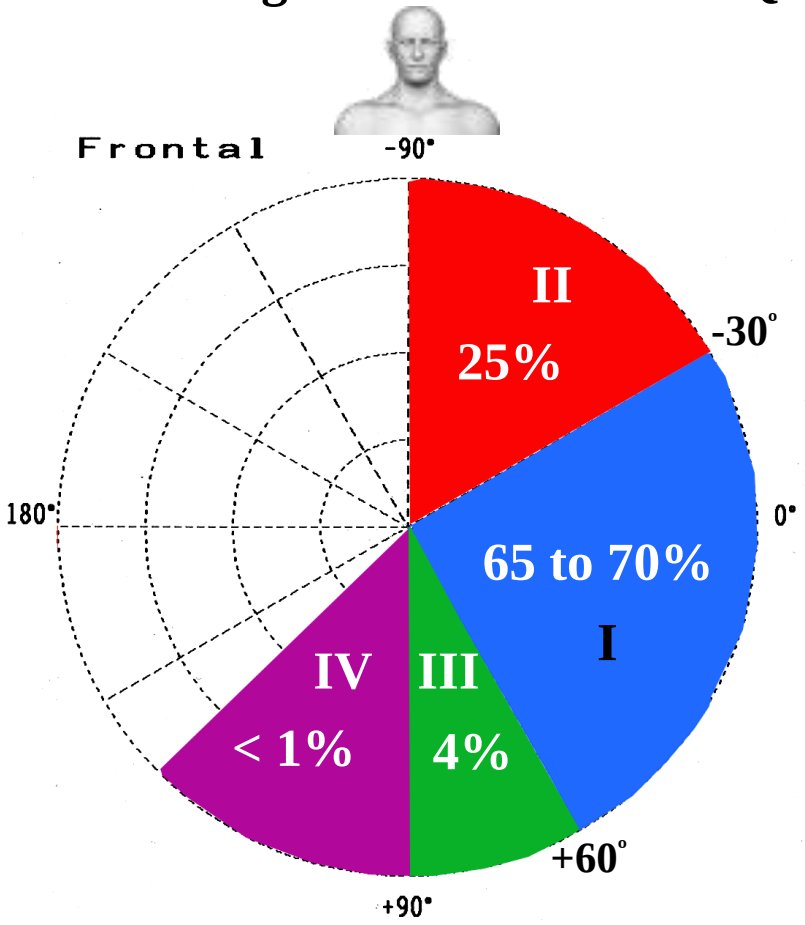
b) With QRS axis with extreme left axis deviation (LAD): beyond –30°: between -30° and -90° (**Parharidis 1997**) (≈25% of cases). The presence of LAD had a 41.9% sensitivity and a 91.6% specificity for the presence of organic heart disease. Aortic valve disease in LBBB pts seems to be frequently accompanied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

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

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

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

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



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

Right axis deviation (RAD) with LBBB is a rare combination. Review of patients from the literature since 1950 indicates that the uncommon combination of LBBB and RAD is a marker of severe myocardial disease, especially congestive cardiomyopathy . The mechanism of production of this ECG pattern appears to be diffuse conduction system involvement in advanced myocardial disease (**Nikolik 1985**). From a database of 636,000 ECGs Childers et al (**Childers 2000**) reported a series of 36 patients with this association. The majority of subjects had dilated cardiomyopathy with biventricular enlargement. LBBB was fixed in 21 of 36 cases. It was freshly acquired, episodic, intermittent, or physiologic in 15 of 36. The RAD was episodic in 30 of 36; it was fixed and concurrent with LBBB in only 2 cases, and never episodically concurrent. The combination of LBBB and RAD was elicited with atrial premature impulses as a rare form of QRS aberration. In one case where the combination was intermittent, a clear relationship with freshly acquired intermittent LPFB. Complete LBBB with episodes of intermittent marked right axis deviation is consequence of the coexistence of pre-divisional LBBB and LPFB. For this diagnosis is necessary to exclude the following causes:

I. Electrolyte imbalances
II. Lateral wall myocardial infarct
III. Intermittent Wolff-Parkinson-White
IV. Conduction right bundle branch block
V. Right ventricular hypertrophy
VI. Pulmonary embolism and
VII. Nonspecific or Unspecified Intraventricular Conduction Delay/Disturbance (NICD)

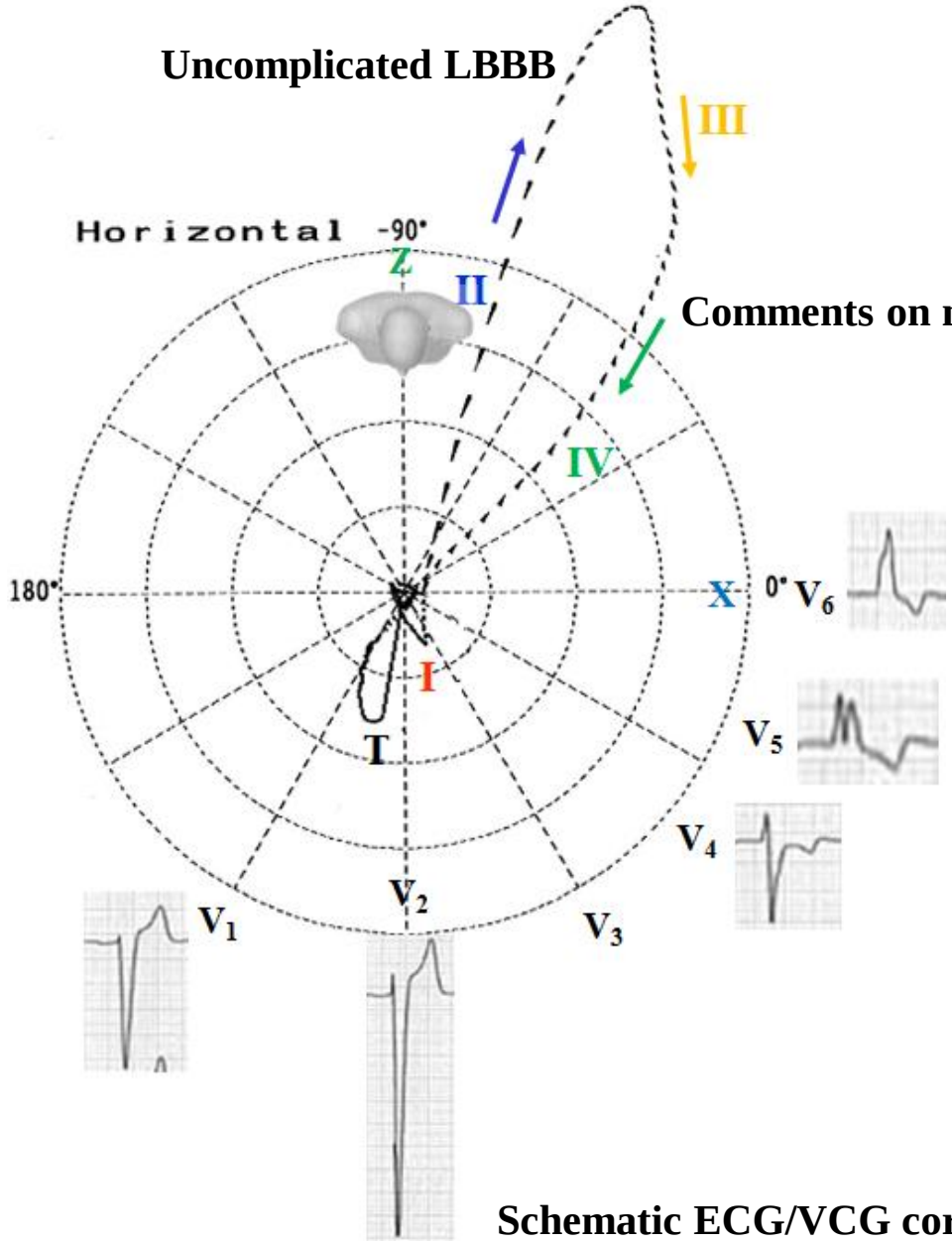
With the purpose to clarify this problem Ravieli et al (**Raviele 1981**) analyzed 23 cases in whom ECG-VCG patterns of "complete" or "incomplete" LBBB were induced by the premature right atrial stimulation. The analysis of these cases has demonstrated that:

1. The same LBBB pattern can be caused by a slowed conduction or block at different sites of left intraventricular conduction system i.e. not only in the main stem of the LBB but also within intra-His LBBB or in all the three fascicles of the LBB distally to its subdivision (fascicular LBBB);
2. ECG-VCG are not able to distinguish the anatomical or functional site of slowed conduction or block;
3. The right or left axis deviation in the LBBB represents or a block at two different sites, the main stem or intra His plus a block in the LAF or LPF of the LBB, or a block at only one side i.e. a troncular, intra-His or divisional LBBB but with prevalent involvement of one fascicle of the LBB;
4. The ECG-VCG Incomplete LBBB pattern is similar to that of the LVH;
5. Atypical LBBB pattern are not specific of myocardial necrosis;
6. The criteria for the diagnosis of "complete" LBBB are not reliable.

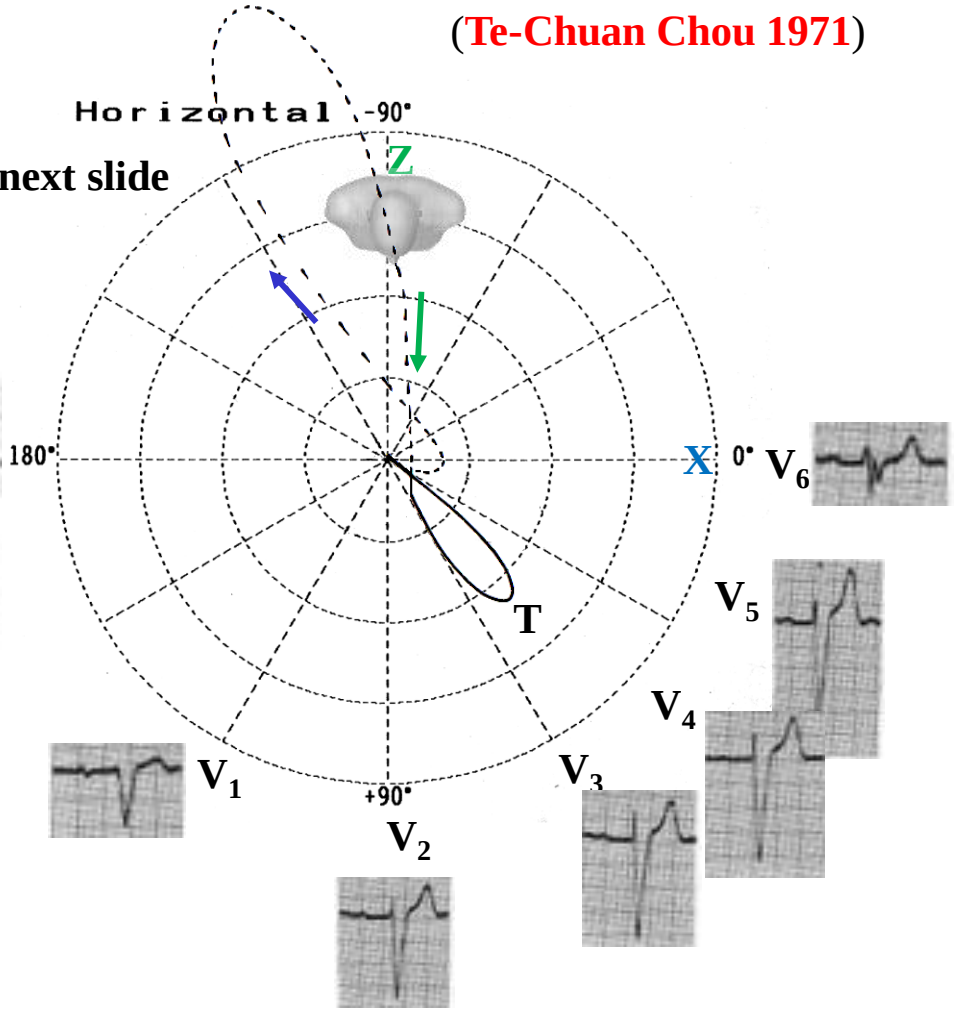
The authors conclude that, since LBBB pattern does not always correspond to a slowed conduction or block in the main stem of the LBB, the current terminology of LBBB is inappropriate and could be changed with another which considers the site of delayed activation and not the site of slowed conduction. Therefore they propose the following terminology:

1. Generalized left ventricular activation delay instead of LBBB without axis deviation;
2. Generalized left ventricular activation delay superiorly predominant instead of LBBB with left axis deviation;
3. Generalized left ventricular activation delay inferiorly predominant instead of LBBB with right axis deviation.

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



Comments on next slide



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

VCG characterization of right ventricular hypertrophy in the presence of LBBB

The VCG characteristics are:

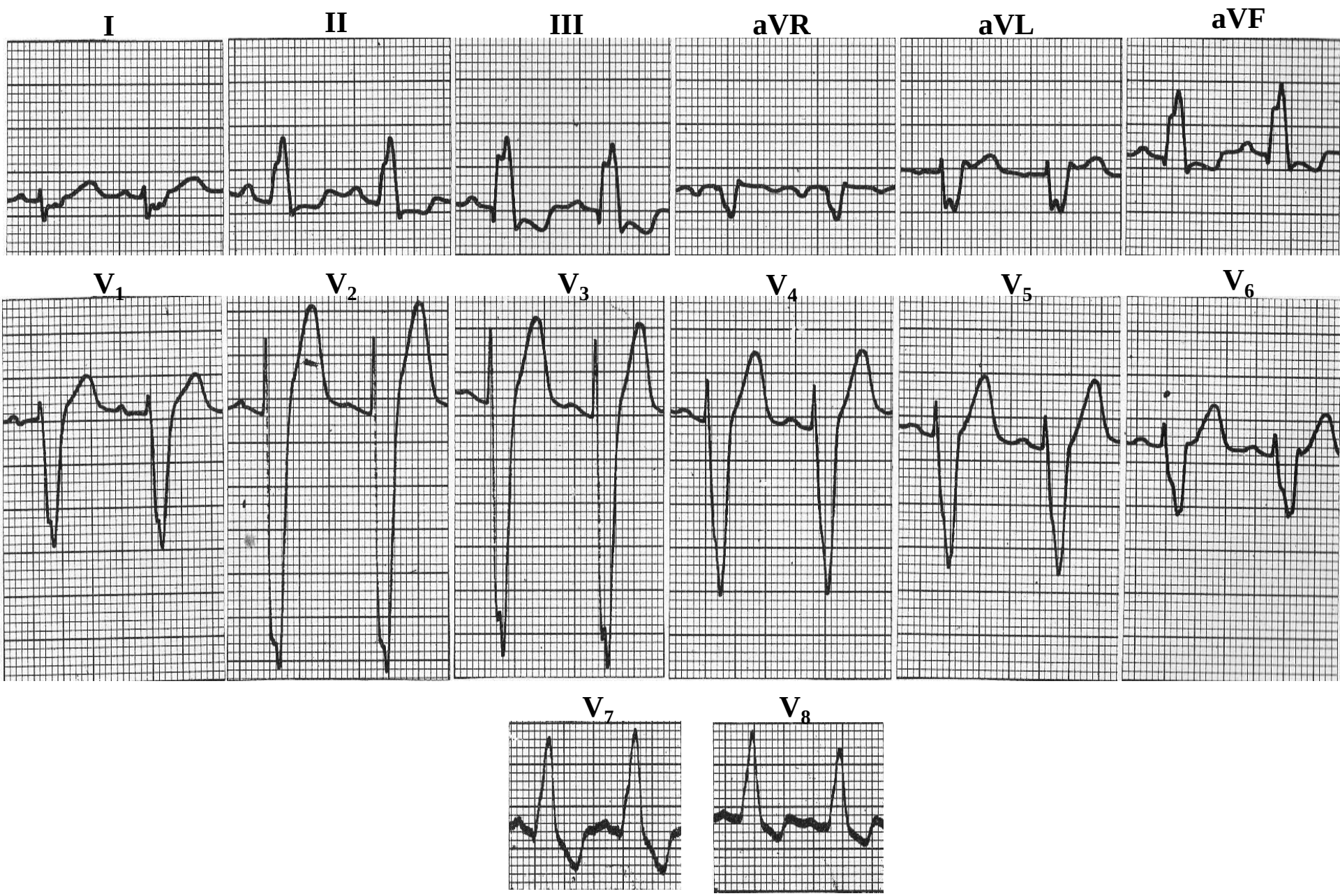
1. QRS loop duration with prolongation;
2. Slow inscription of the mid and late portion of the QRS loop;
3. Leftward and inferior orientation of the initial QRS vectors;
4. Posterior and rightward displacement of the maximum QRS vector;
5. Clock-wise inscription of the major portion of the QRS loop in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

Final comments:

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

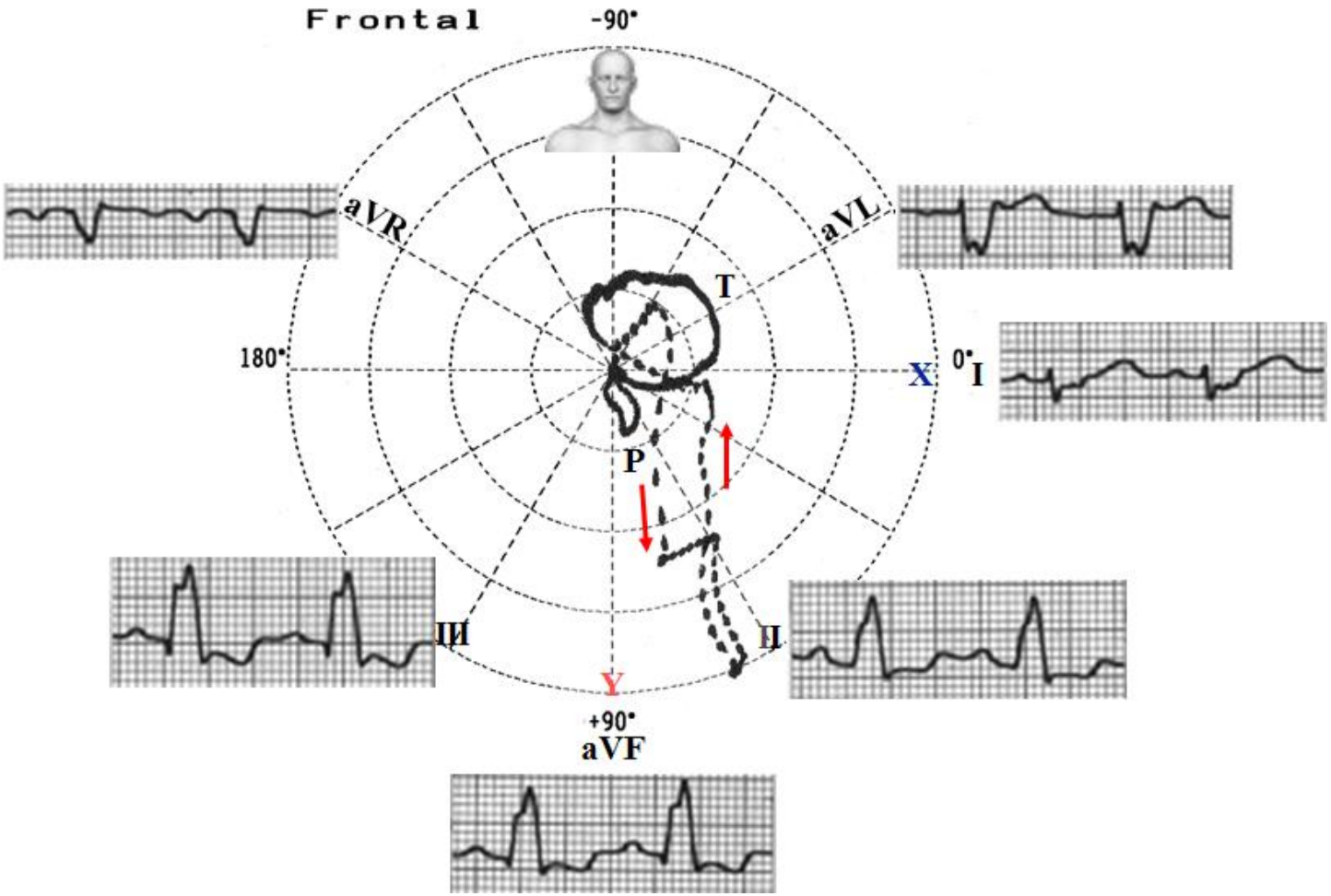
	Isolated LBBB	LBBB + RVH
HP QRS loop	Leftward displacement	Rightward displacement
ST vector and T-loop	Righward orientation	Leftward orientation
ECG lead I	Monophasic R wave	Presence of S wave
QRS axis	From -30º to +60º (≈ 65% to 70% of cases) From –30º to -90º (≈25% of cases)	Beyond +90º (< 1% of cases)

Examples of LBBB with right axis deviation



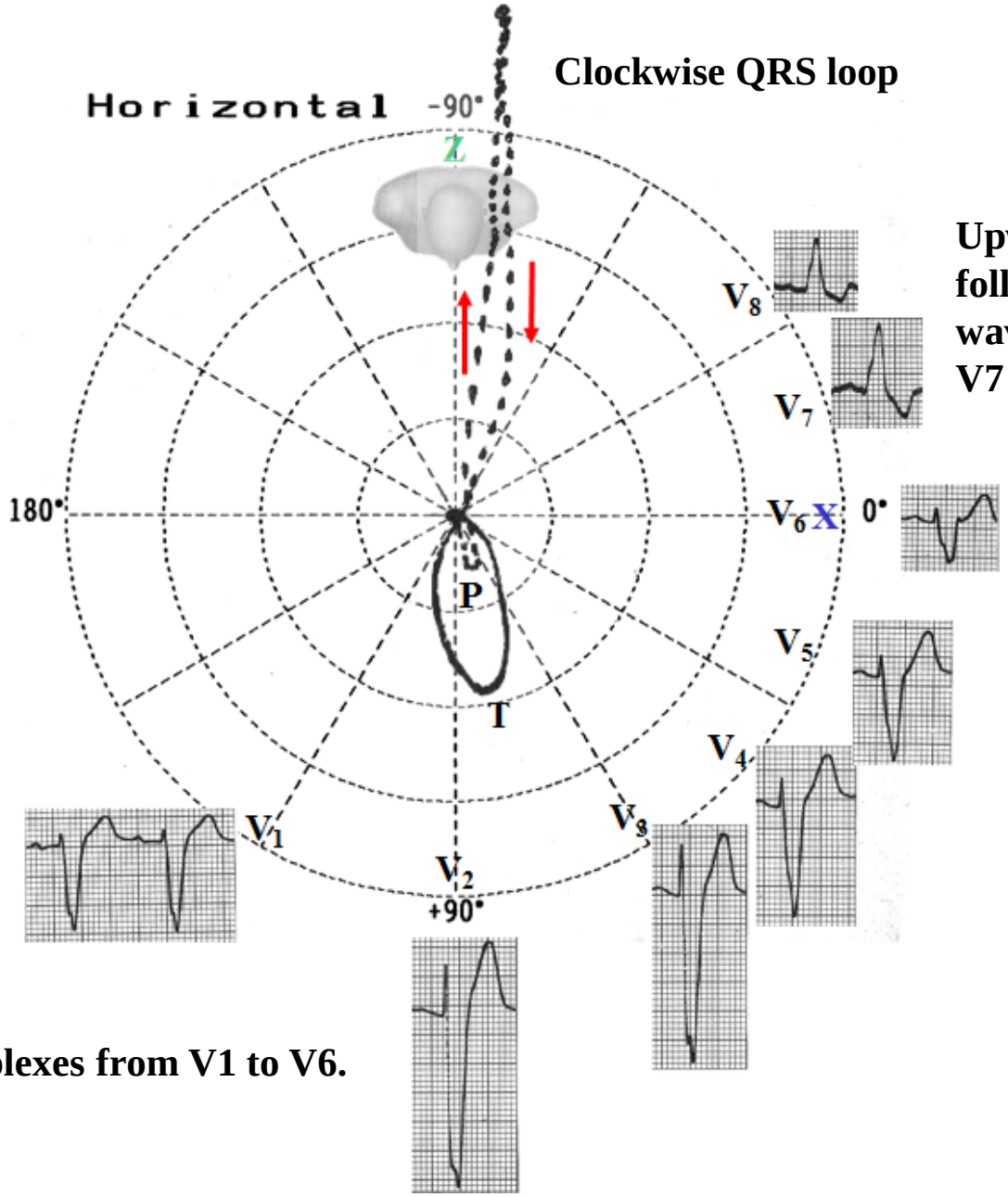
Atypical LBBB because rs in I and rS in aVL and rS from lead V1 through V6. The typical LBBB upward QRS is observed only in inferior and posterior leads (V7-V8)

ECG/VCG correlation on FP



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

ECG/VCG correlation on HP

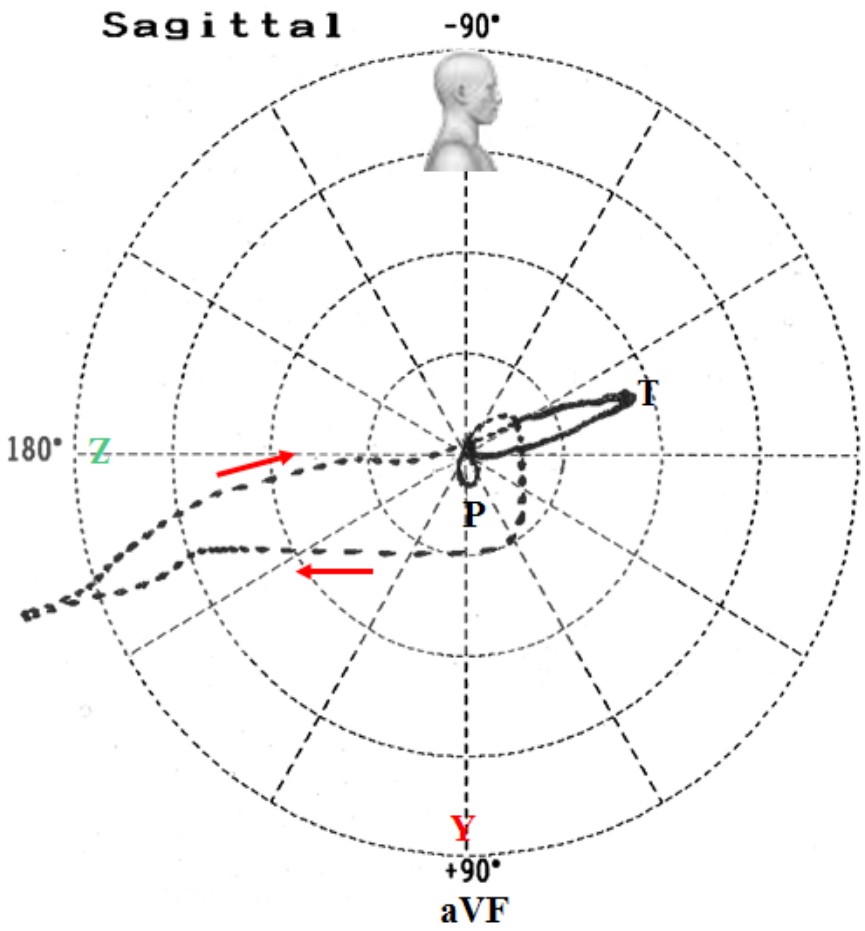


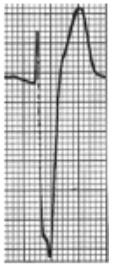
Clockwise QRS loop

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

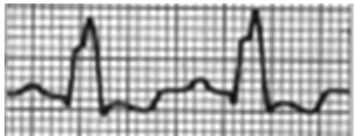
Negative QRS complexes from V1 to V6.

ECG/VCG correlation on RSP





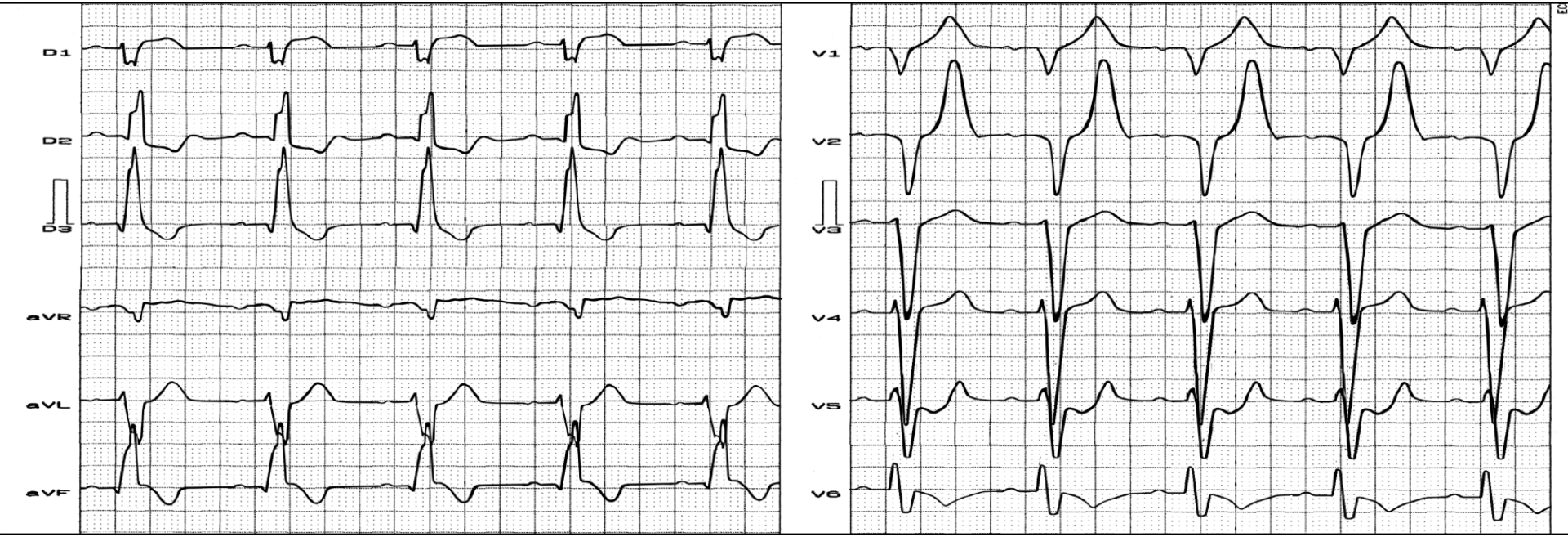
Negative QRS complex in V2 followed by positive T wave



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

Name: ASC; **Sex:** Male; **Age:** 54 yo.; **Race:** White; **Weight:** 86Kg; **Height:** 1.68 m; **Biotype:** Endomorph;

Date: 04/03/2003; **Medication in use:** Enalapril 10 mg 2X + Atenolol 50 mg + Chlortalidone 12.5 mg.



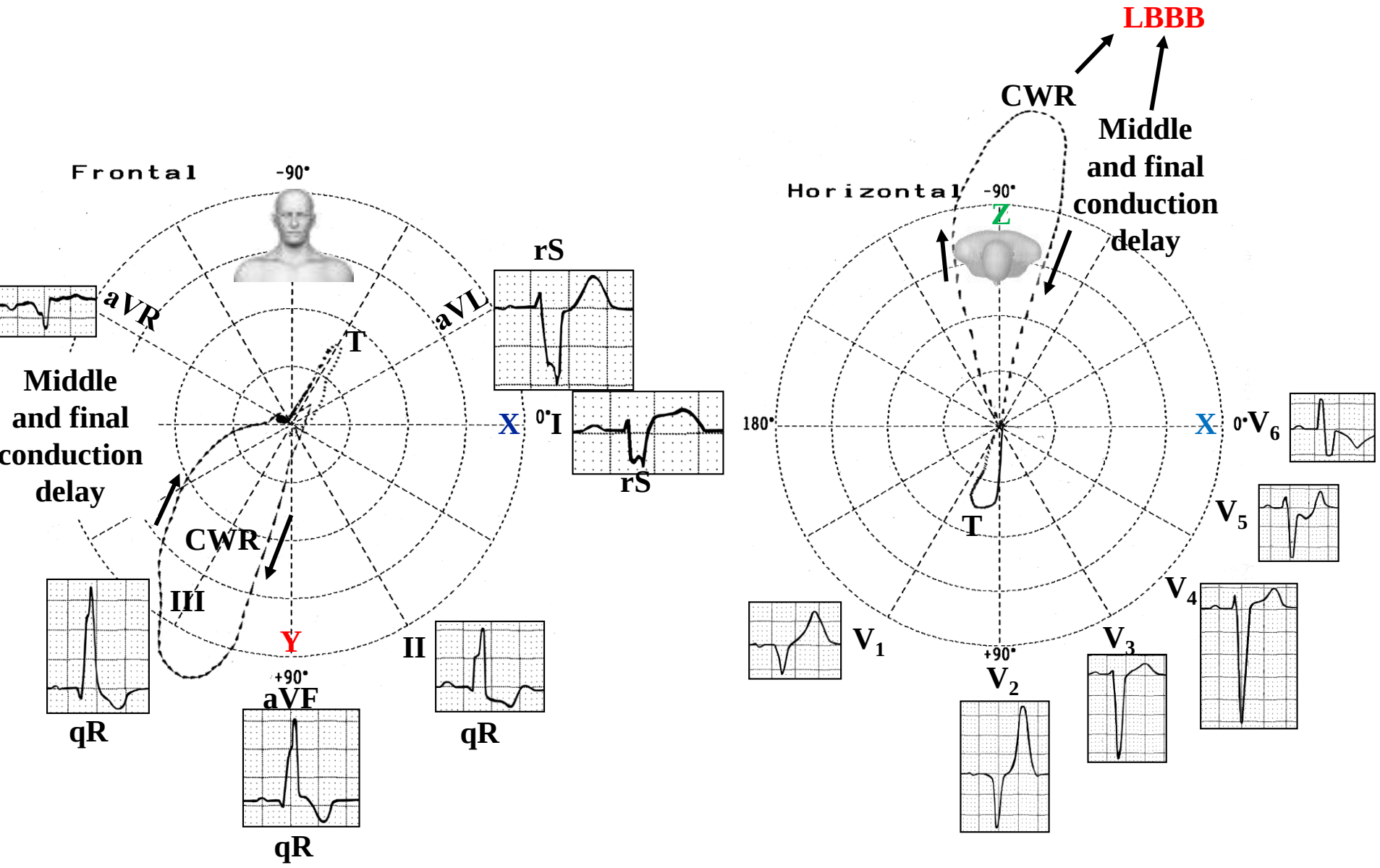
Clinical diagnosis: Hypertensive heart disease + aortic insufficiency by aortic cause.

Echo diagnosis: Moderate concentric hypertrophy: septum 13 mm and posterior wall 14 mm. Moderate aortic insufficiency.

ECG diagnosis: SR; HR: 72 bpm; SAP: $+60^0$; SAQRS: +110°; QRSD: 165 ms; I and aVL = rS; III = qR; RIII > RII. Which is the electrocardiographic foundation for LPFB diagnosis? SÂQRS deviated to the right in clinical absence of RVH, vertical heart or lateral infarction; QRS complexes of the rS type in I and aVL; complexes of the qR type in inferior leads with R wave of III > than R wave of II. There are references in literature to aortic insufficiency by regurgitant jet, which thrown on the posteroinferior wall may cause LPFB. On the other hand, the CLBBB has as its most frequent cause hypertension. An accurate diagnosis of LPFB must obligatorily be clinical and electrocardiographic, as in this case, in which in an obese, endomorph, hypertensive patient, the SAQRS is in $+115^{\circ}$.

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

ECG/VCG correlation on Frontal and Horizontal Plane



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

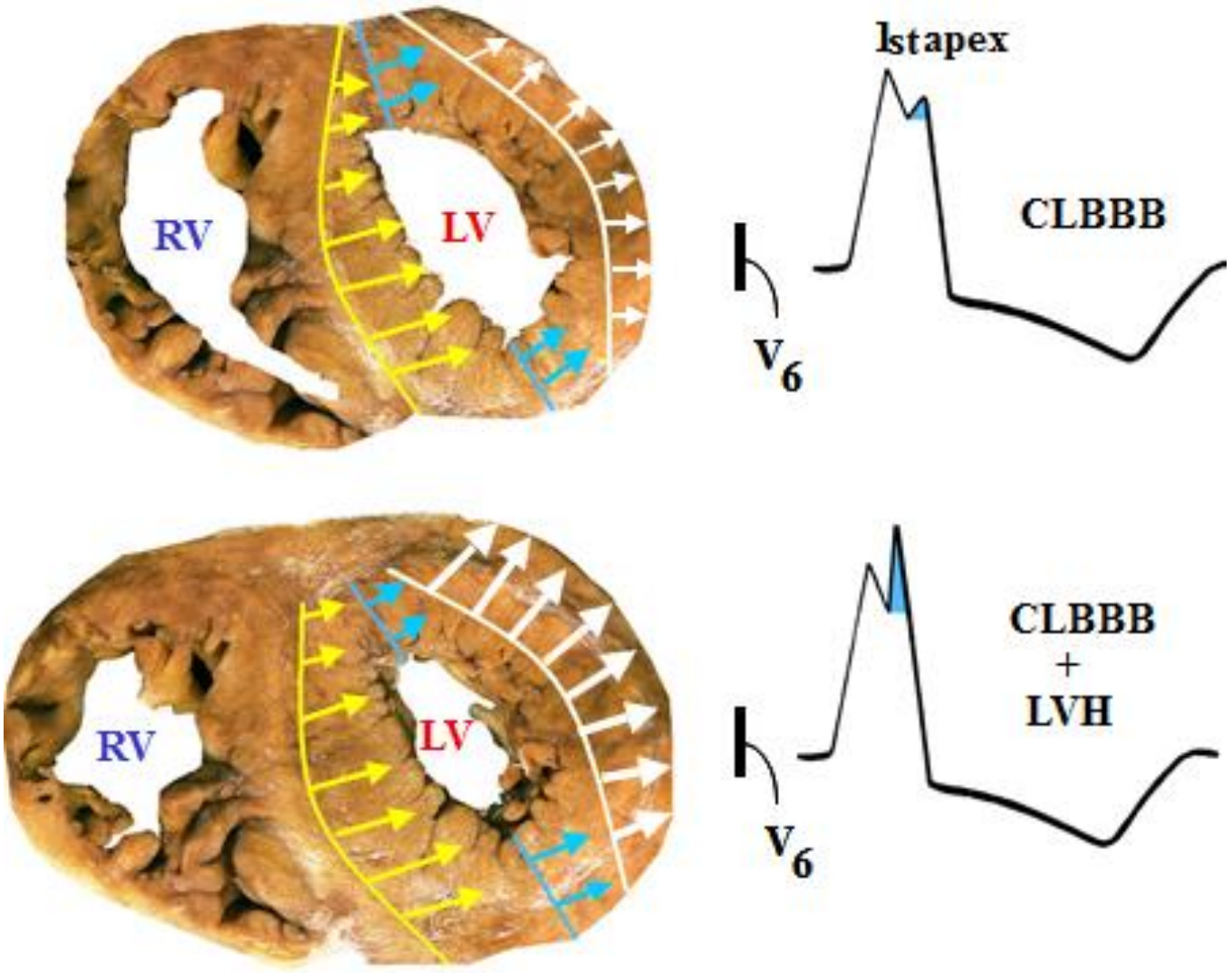
Electrocardiographic diagnosis of LVH in the presence of LBBB

The presence of LBBB on 12-lead ECG may obscure the diagnosis of LVH.
The criterion of SV2 + RV6 greater than 4.5 mV demonstrated a sensitivity of 86% and a specificity of 100% for LVH diagnosis in the presence of LBBB.
QRS duration greater than 160 ms plus left atrial enlargement strongly supports the diagnosis of LVH in the presence of LBBB.(**Klein 1984**)
There are no differences in limb lead voltage, R peak time, or mean frontal plane QRS axis.
The following criteria can be helpful in left bundle branch block: QRS voltage increase, left atrial enlargement, QRS duration >155 ms.(**Oreto 2007**) LVH can be diagnosed in the presence of LBBB with an accuracy at least similar to that observed in patients without this conduction defect. Computer-assisted interpretation of the ECG may be useful in the diagnosis of LVH as it enables the implementation of more accurate algorithms. Diagnostic algorithms, voltage-duration products, and certain compound criteria had the best sensitivities.(**Rodríguez-Padial 2012**) LA abnormality is significantly diagnostic of LVH in the presence of LBBB. Age, body mass index, body surface area, frontal axis, and QRS duration are also significant predictors of LV mass.(**Metha 2000**)

Kafka parameters for the diagnosis of LVH in presence of LBBB

Kafka et al (**Kafka 1985**) selected and used 5 ECG parameters in cumulative fashion for the diagnosis of LVH in the presence of LBBB: 1. R aVL ≥11 mm; 2. QRS axis ≤40º or SII greater than RII; 3. SV1 + RV5 to RV6 ≥40 mm; 4.SV2 ≥30 mm; 5. SV3 ≥25 mm.
This cumulative approach was superior to using single conventional criterion such as SV1 + RV5 or RV6. When LVH was defined as an M-mode index of at least 115 g/m^2, the sensitivity was 75% and specificity 90%. Using M-mode, a mass of at least 215 g was the standard, the sensitivity was 73% and the specificity 66%.
LVH can be diagnosed by ECG criteria in the presence of LBBB at least as reliably as in normal conduction.

LVH criteria in the presence of Complete Left Bundle Branch Block



According to the apex of R wave in V_6, of greater amplitude than the first apex. As the LV free wall has more mass to be depolarized, the last apex is of greater voltage than the first.

Conclusion and management

Clinical diagnosis considerations: The two main causes of syncope are cardiac arrhythmias and neurocardiogenic syncope, also known as "neurally mediated hypotension", "fainting reflex", "vasodepressor syncope", "vasovagal syncope", or "autonomic dysfunction". In this condition blood vessels tend to expand, which leads to pooling of blood in the lower parts of the body. As a result, less blood reaches the brain and this causes fainting. The usual stimulus for this action resides in the nerves of the heart-hence the term neurocardiogenic. Neurocardiogenic syncope occurs in predisposed individuals after prolonged periods of quiet upright posture (such as standing in line), after being in a warm environment (such as in hot summer weather, a hot crowded room, a hot shower or bath), immediately after exercise, after emotionally stressful events (having blood drawn, being scared or anxious), after eating, when blood flow has shifted to the intestinal circulation during the process of digestion. In the present case the patient had a typical syncope episode, very suggestive to be a sudden AV block because unrelated to swallowing, urination, defecation, coughing or positional changes (orthostatic). Additionally, we observe on ECG prolonged PR interval, wide atypical QRS pattern with sudden transient QRS extreme right axis deviation on right shoulder axis, no man's land or Northwest axis. Neurological origin is ruled out because absence of auras, premonitory symptoms, mental confusion after the event, or focal neurological signs.

ECG/VCG pattern: This pattern is compatible with NICD Complex delays in the intraventricular conduction systems associated to regional conduction slowing in the myocardium by consequence of severe His system disease and severe LVH with fibrosis. Probably this is the variety of NICD in the present case.

Approach: The decision to treat an arrhythmia with pacemaker implantation (or any other treatment) depends in part upon whether the person has symptoms or not, as well as the severity of the symptoms.

We think that EPS is mandatory, and probably severe intraventricular conduction disease is present. If confirmed bifascicular/trifascicular block, a dual-chamber pacemaker is indicated (2 pacing leads are implanted, 1 in the RV and 1 in the RA); this is the most common type of implanted pacemaker.

Death in patients with untreated AV block prolonged asystole or bradycardia-triggered VT/VF. Although formal randomized controlled trials (RCTs) of pacing in AV block have not been performed, it is clear from several observational studies that pacing prevents recurrence of syncope and improves survival.

References

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