

**Mulher de 59 anos portadora de miocardiopatia chagásica crônica forma mista:
com arritmia e distúrbios dromótopos intraventriculares e comprometimento
moderado do desempenho ventricular.**

**Woman of 59 years suffering from chronic chagasic myocarditis mixed form:
dromotropic intraventricular disturbances, premature ventricular contractions and
moderate impaired of left ventricular function.**

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História clínica

Feminina, 59 anos, branca, do lar, nascida e criada na zona rural pobre do sertão Baiano, Educação primária incompleta, baixa condição socioeconômica.

MC: viajou para se consultar a São Paulo porque no mês passado, ao tentar doar sangue para um familiar não foi possível por apresentar sorologia positiva para doença de Chagas.

Queixas: refere que há aproximadamente dois anos vem sentido dificuldade na deglutições (“engasgo”) Nos últimos três meses apresentou quatro episódios de tontura forte sem perda da consciência e palpitações com pulso lento.

Tem 11 irmãos sendo que quatro faleceram jovens (< de 40 anos) devido à doença de chagas. Dois deles por morte súbita.

Refere que a moradia é de adobe e taipa e que na área rural onde reside com frequência passa “o pessoal da SUCAM” (Superintendência de Campanhas de Saúde Pública) desinfetando as residências.

Ao exame físico: afebril, corada, acianótica, eutrófica.

RCI frequentes extrasístoles. Ictus cordis no sexto espaço 1cm fora da linha hemiclavicular, tercer ruido na protodiástole com cadencia de galope e de baixa frecuencia e sendo assim assim melhor ouvido com a campana do estetoscopio . Pressão arterial 115/70mm, Ausência de estase jugular

Abdome sem hepatomegalia. Fígado palpável na borda costal direita e sem edema de membros inferiores.

ECOCARDIOGRAMA ventrículo esquerdo com disfunção sistólica de grau moderado, hipocinesia difusa moderada, com predomínio íferobasal.

Diâmetro diastólico do ventrículo esquerdo moderadamente aumentado(60mm), espessura do septo e parede posterior 70mm, FE or Teichholz 45%, VD normal.

Qual o diagnóstico eletrocardiográfico? E porque?

Case report

Female, 59 years old, white, housewife, born and raised in a poor rural backlands of Bahia, north-east Brazil incomplete primary education, lower socioeconomic status.

MC: he traveled to refer to São Paulo city because last month trying to donate blood for a family was unable to present positive serology for Chagas disease.

Complaint: states that approximately two years ago has difficulty in swallow. Over the past three months had four strong episodes of dizziness without loss of consciousness and palpitations with slow pulse.

He has 11 siblings and four died young (<40 years) due to Chagas disease. Two of them from sudden death. States that the house is of adobe and rammed earth and in the rural area where he lives is often "staff SUCAM" (Superintendent of Public Health Campaigns) disinfecting their homes.

Physical examination no fever, acyanotic, ashamed, eutrophic.

RCI frequent premature contractions. Apical impulse in the sixth space 1cm outside the left midclavicular line. A triple rhythm in the early diastole with gallop cadence results from the presence of a third heart sound S3 of low frequency and thus best heard with the bell of the stethoscope.

Blood pressure 115/70mm,

No jugular venous distension

Abdomen without hepatomegaly. Liver palpable in the right costal margin

Extremities: absence of lower limb edema.

ECHOCARDIOGRAPHY: moderate left ventricular systolic dysfunction, moderate, diffuse hypokinesis, predominantly basal inferior. LV end-systolic dimension and diastolic diameter moderately enlarged (60 mm), Diastolic thickness of septum and posterior wall normal 7mm, FE 45% (Teichholz)

Normal right ventricle.

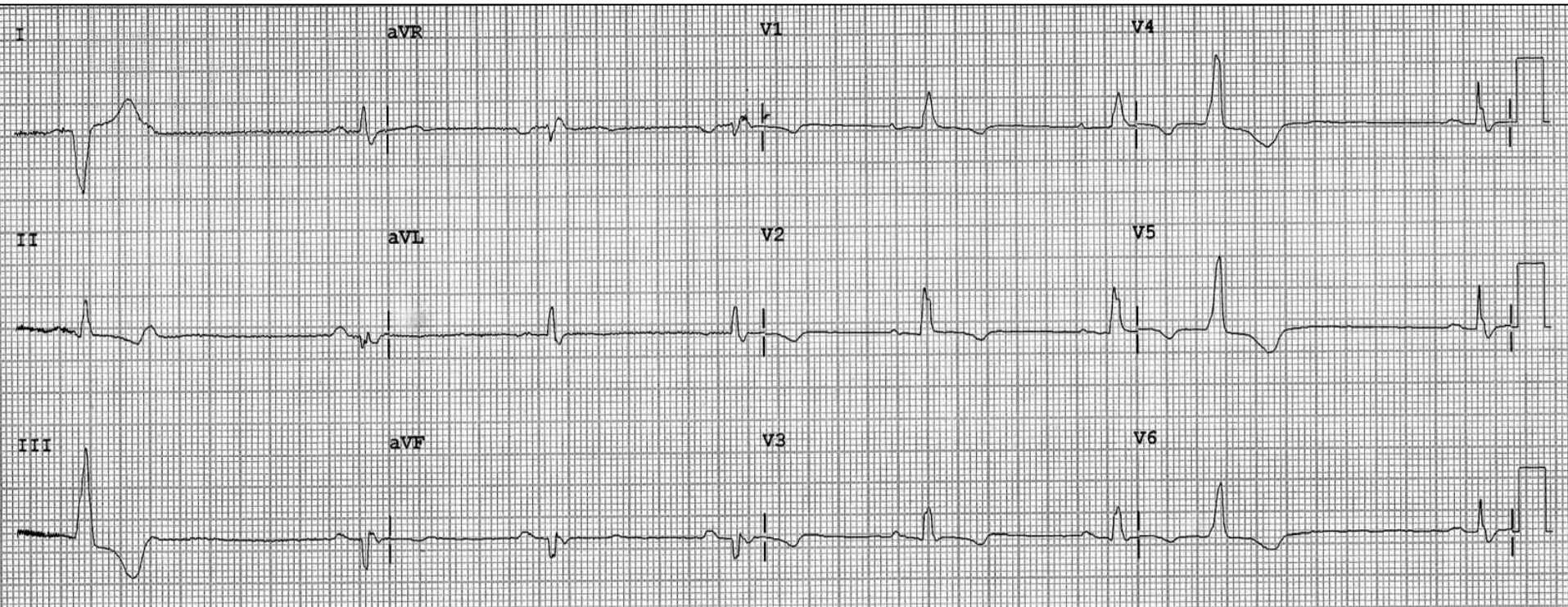
What is the electrocardiographic diagnosis? And why?

Name: MBF;

Age: 59 yo;

Weight: 75 Kg;

Height: 1,67m



Colleagues opinions

Queridos amigos del forum , mujer de 59 años con trastorno dromotrópico severo.

Distancia P-P 1240, frecuencia cardiaca 42 lpm

En DI, DII, DIII se registra una P bloqueada, que me parece que es un bloqueo funcional o bloqueo falso, debido a que la P cae en el período refractario de la conducción retrógrada de la extrasístole ventricular dado que no tiene P retrógrada

Presenta una onda P muy ancha .sugiriendo un disturbio de conducción intra-atrial probablemente por lesión fibrótica en la auricular izquierda

Deberíamos plantearnos cual es el mecanismo de la bradicardia: 1)es de origen sinusal? o 2) es debido a una severa afectación en el dromotropismo intratrial? Para responder estas preguntas hay que realizar un Holter o una Ergometria. Las características clinicas electrofisiológicas arritmológicas y terapéuticas son muy diferentes entre estas dos situaciones

El bloqueo de rama derecha y la extrasístole del ventrículo derecho pueden hacer sospechar una afección de esta cámara, a pesar que se demostró por el ECO apenas una alteración de contracción izquierda

Es casi imposible que esta mujer no padezca de Chagas, pero los maestros Brasileños, deben "guardar el cuchillo bajo el poncho" en este caso

Un fraternal abrazo

Samuel Sclarovsky Israel.

**Dear friends of the forum, 59 year old woman with severe dromotrópico disorder.
Distance P-P 1240, heart rate: 42 bpm.**

In DI, DII, DIII recorded a P blocked, I think it is a functional block or pseudo-block, because the P falls in the refractory period of retrograde conduction of ventricular premature since no retrograde P. It has a very wide P wave suggesting a disturbance of intra-atrial conduction probably fibrotic lesion in the left atrium.

We should ask what is the mechanism of bradycardia: 1) is of sinus origin? or 2) is due to a severe impairment in intratrial dromotropism? To answer these questions have to do a Holter or exercise testing. Clinical features and therapeutic electrophysiological arritmiológico are very different between these two situations.

The right bundle branch block and right ventricular extrasystole suspect right ventricle afectation, despite being proved by the ECO just an exclusively alteration of left ventricle contraction.

It is almost impossible that this woman does not suffer from Chagas disease, but Brazilian teachers should "keep the knife under the poncho" in this case.

A fraternal embrace

Samuel Sclarovsky MD Israel.

En la cardiopatía chagásica los signos y síntomas suelen ser idénticos a los de otras miocardiopatías dilatadas. Adicionalmente, se puede presentar disfunción autonómica, que produce bradicardia e hipotensión arterial.

En el ecocardiograma, se observan trastornos segmentarios de la motilidad, particularmente en la región inferoposterior y en el ápex, además de aneurismas apicales, habitualmente asociados con la presencia de trombos murales.

La RMN puede ser de utilidad para detectar fibrosis miocárdica, en especial en pacientes a los que no se les observan alteraciones en el ecocardiograma. De hecho, el hallazgo de fibrosis demostró ser un factor de mal pronóstico de esta enfermedad.

Por otro lado, se sugiere efectuar un Holter o prueba ergométrica en los individuos con antecedentes de síncope o mareos sin causas claras, a fin de estratificar el riesgo de muerte súbita.

Saludos Edgardo Schapachnick M.D. Buenos Aires Argentina

In Chagas' heart disease signs and symptoms are identical to those of other dilated. In addition, autonomic dysfunction may occur, resulting in bradycardia and hypotension.

On echocardiography, there are segmental motility disorders, particularly in the inferoposterior region and at the apex, as well as apical aneurysms, usually associated with the presence of mural thrombi. MRI can be useful for detecting myocardial fibrosis, especially in patients who were not observed alterations in the echocardiogram. In fact, the finding of fibrosis proved to be an indicator of poor prognosis of this disease.

On the other hand, suggests making a Holter or exercise testing in individuals with a history of syncope or dizziness without clear reasons, in order to stratify the risk of sudden death.

Greetings

Edgardo Schapachnick M.D. Buenos Aires Argentina

Estimado Maestro: Ritmo sinusal con 52 latidos por min, crecimiento auricular izquierdo, intervalo PR prolongado (0,22 seg) eje del QRS desviado a la izquierda y BCRD. La imagen de QSr en cara inferior la atribuyo a fibrosis de la misma por su miocardiopatía chagásica.No impresiona de etiología isquémica. Presencia de EV sin pausa compensadora completa sin una tira de ritmo podría sospechar latido de escape ventricular. No solo tiene afectación del dromotropismo como ud describe también del inotropismo y cronotropismo. Comparto con el Master Edgardo la necesidad de solicitar ergometría y holter para evidenciar la afectación cronotrópica y carga arritmica. Como establecer el riesgo de MS en la miocarditis chagásica crónica? el solo hecho de padecerla ya la situa en esta población, con deterioro de la FEY y los distúrbios comentados estaria tentado de pensar que tiene alto riesgo. Desconozco si hay evidencia de esto, los estudios prospectivos con CDI no incluyen a la población chagásica. Tal vez Edgardo y Ud nos iluminen en este aspecto. Como estratifico el riesgo de MS en una paciente con Chagas y afectacion de la FEY?

Marcapasos de eso estoy seguro. pero CDI? espero Uds respondan a este punto.

Un abrazo Martin Ibarrola M.D.

Dear Teacher: Sinus rhythm with 52 beats per min, left atrial crecimiento, prolonged PR interval (0.22 sec) QRS axis deviated to the left and BCRD. QSr image in the inferior wall attributed to fibrosis of the same for her chagasic cardiomyopathy I don't hinkt in ischemic etiology..

Presence of EV without compensatory pause is complete without a rhythm strip may suspect ventricular escape beat. Not only has dromotropismo involvement as you described but also inotropism and chronotropismo afectation. I share with the Master Edgardo must solicit ergometry and Holter monitoring for evidence of involvement arritmica chronotropic and cargo.

How to set the risk of MS in chronic chagasic myocarditis? the mere fact of suffering and the situation in this population, with impaired commented FEY and riots would be tempted to think that is high risk. Know if there is evidence of this, prospective studies do not include ICD chagasic population. Edgardo and you might enlighten us on this. As stratified the risk of MS in a patient with Chagas and affectation of the Fey? Pacemakers that's for sure. but CDI? I hope you respond to this point. A hug Martin Ibarrola M.D. Argentina

Dear Andrés,

An interesting ECG to consider: the rhythm is sinus bradycardia (~48 bpm) with two PVC's (perhaps multifocal); the PR interval is 200 ms; the QRS duration is 140 ms; the QT interval ~450 ms; the QRS axis is left ~-50 degrees mainly due to the prominent Q-waves in II, III, aVF indicative of an inferior left ventricular wall electrically silent area (also seen on the echocardiogram with basal inferior hypokinesis). There is a right bundle branch block and prominent anterior forces likely related to the basal inferior hypokinesis. In the U.S. this would probably be indicative of myocardial infarction, but this is also compatible with her known diagnosis of Chagasic myocarditis.

As always, I look forward to your interesting comments and teaching on the case.

Regards,

Frank G. Yanowitz, MD

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440 D Street, Suite 206 Salt Lake City, UT 84103. 801-408-5302**

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Estimado Andrés,

Un interesante ECG para tener en cuenta: el ritmo es sinusal bradicárdico (bradicardia sinusal ≈48 ppm) con dos tipos de extrasístoles ventriculares (tal vez multifocal), el intervalo PR es de 200 ms y la duración del QRS es de 140 ms, el intervalo QT ≈450 ms, y el eje del QRS en ≈ 50°, debido principalmente a las destacadas ondas Q en II, III, aVF indicativo de una área eléctricamente silente en la pared inferior del ventrículo izquierdo (también detectada en el ecocardiograma traducida por hipocinesia infero-basal). Hay un bloqueo de rama derecha y las fuerzas anteriores prominentes probablemente consecuencia de la hipocinesia basal inferior. En los EE.UU. esto sería probablemente indicativo de infarto de miocardio, pero también es compatible con su diagnóstico conocido de miocarditis chagásica.

Como siempre, espero sus comentarios interesantes y didácticos sobre el caso.

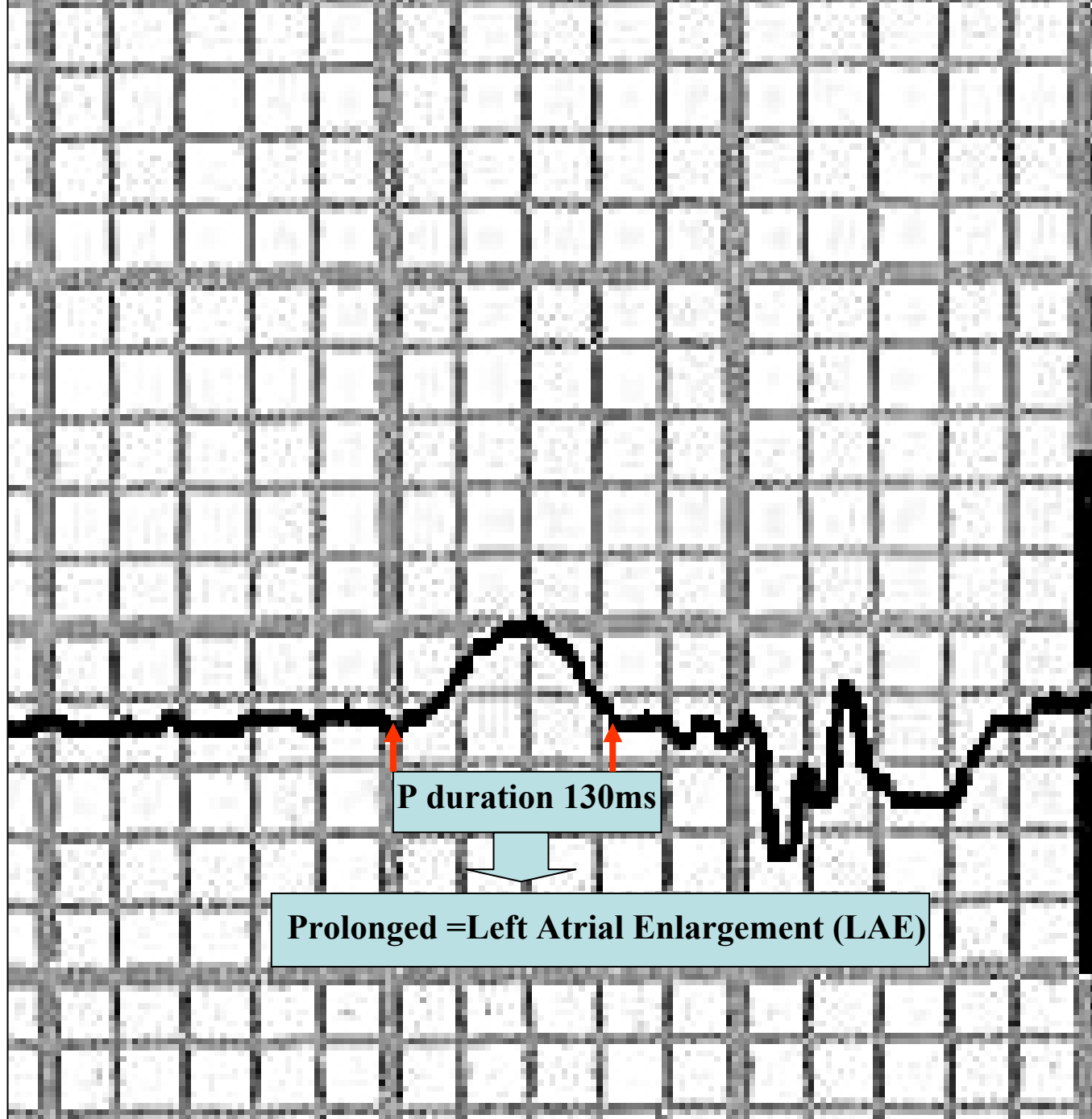
Un cordial saludo

Frank

Final Comments

By Andrés Ricardo Pérez-Riera M.D. Ph.D.

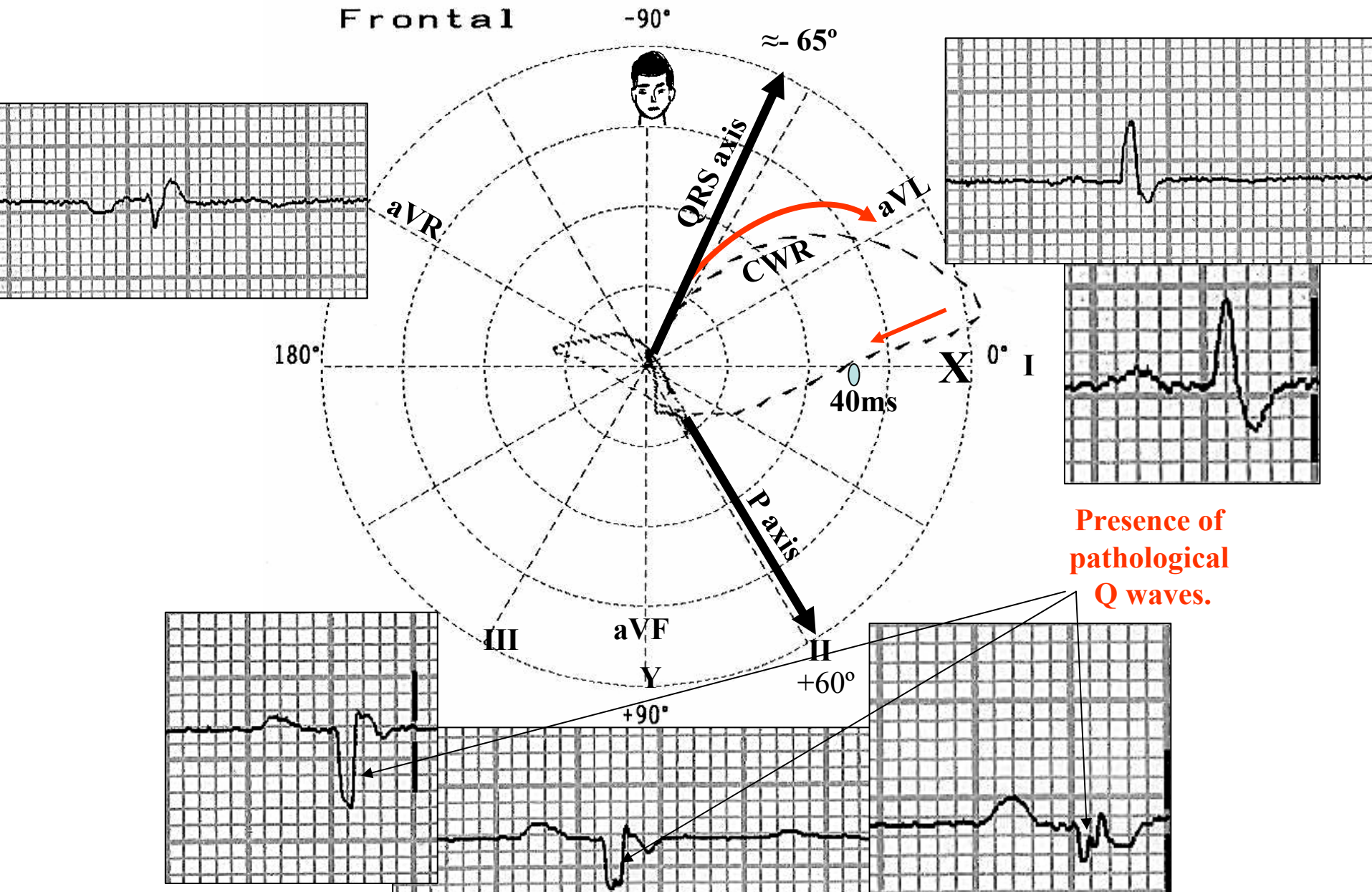
II



P duration 130ms

Prolonged = Left Atrial Enlargement (LAE)

ECG/VCG correlation on frontal plane

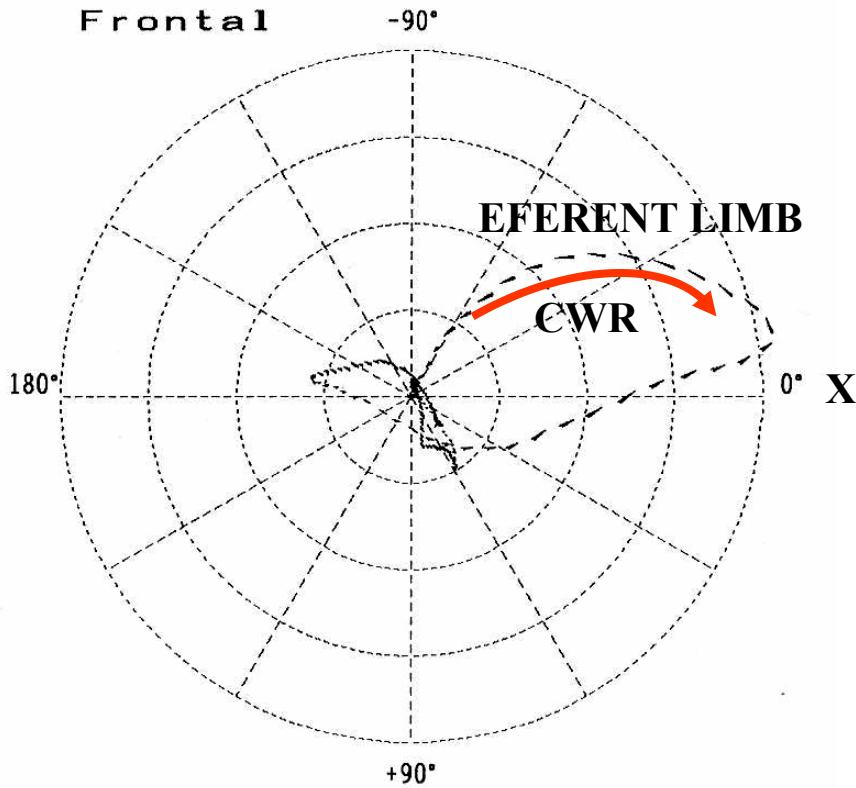


Presence of pathological Q waves.

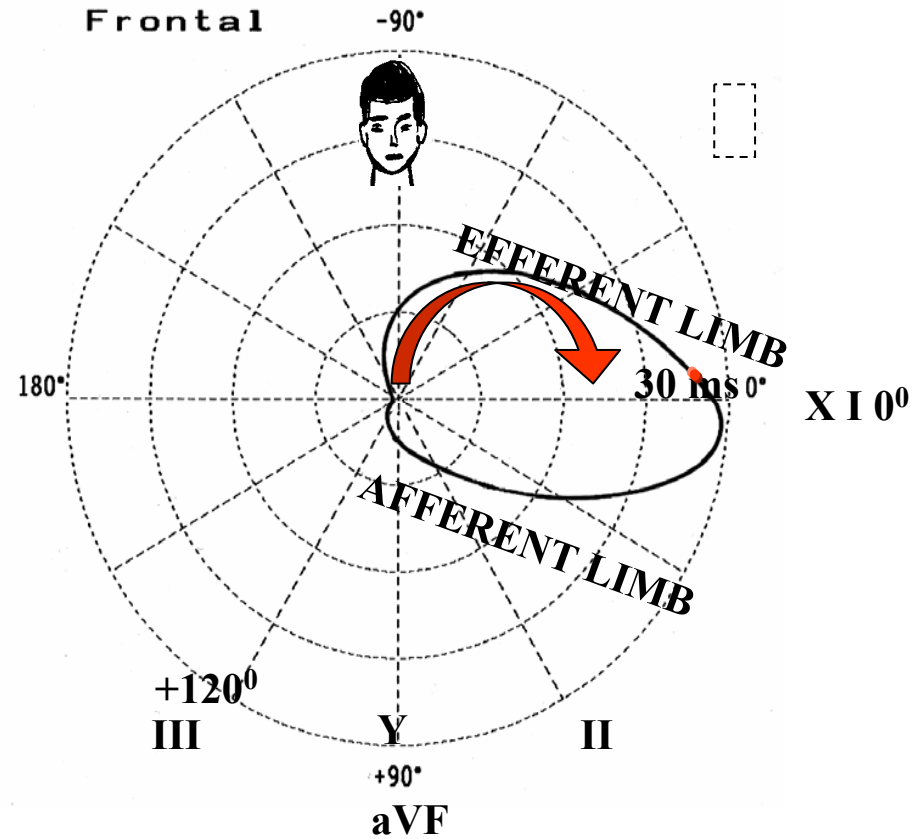
Broad ($\geq 40\text{ms}$) initial pathological Q wave in inferior leads: Electrically inactive area in diaphragmatic wall. Initial QRS VCG loop of clockwise rotation (CWR) from right to left with $>40\text{ms}$ above the X line.

VCG LOOP IN NON-EXTENSIVE DIAPHRAGMATIC ELECTRICALLY INACTIVE AREA

The preset case



QRS loop of clockwise rotation in electrically inactive inferior wall



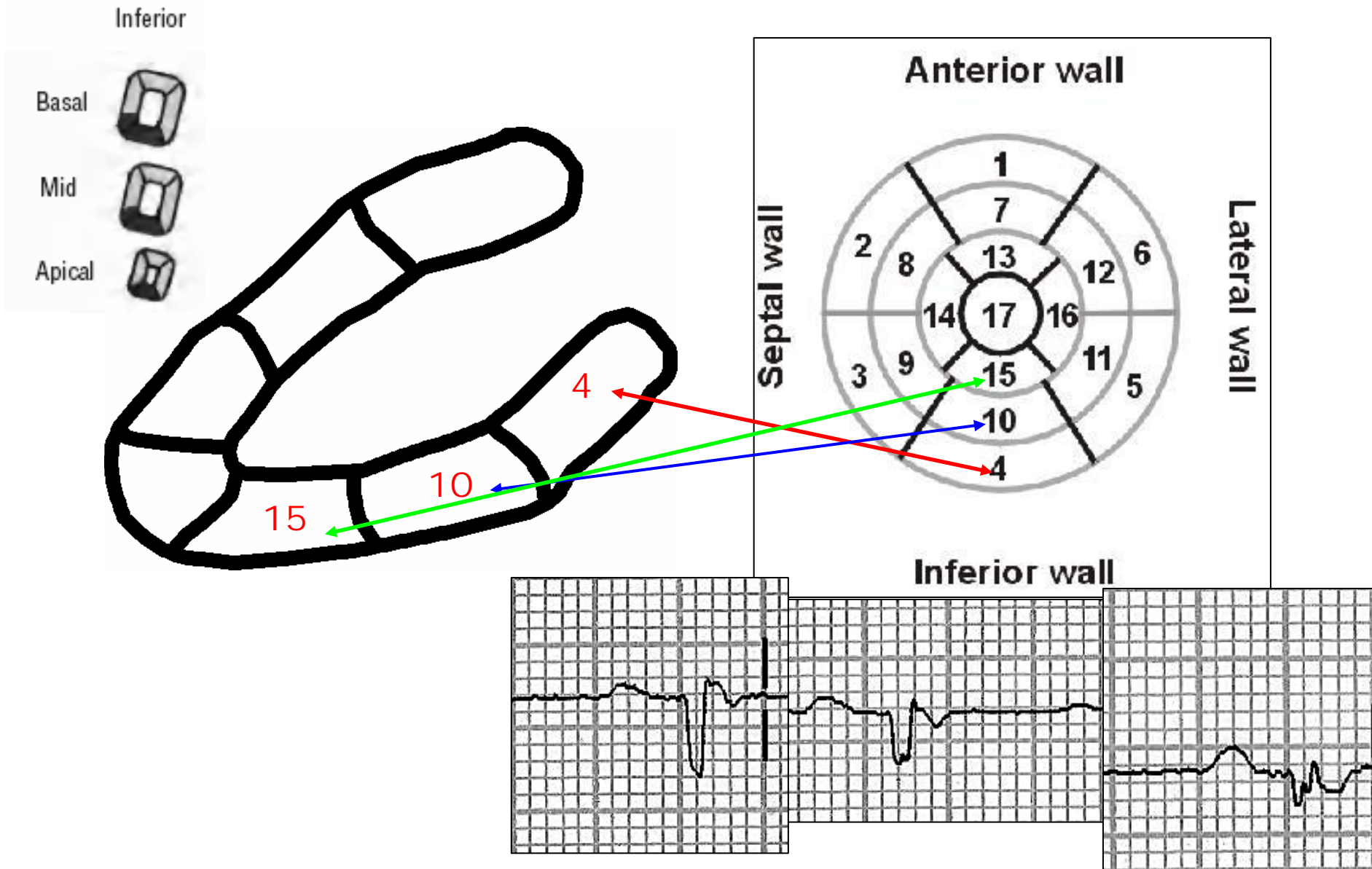
QRS loop with efferent limb of clockwise rotation (CWR), heading from right to left and located above the orthogonal X lead (at least 80% of the QRS loop presents CWR).

VECTORCARDIOGRAPHIC CHARACTERISTIC OF FRONTAL PLANE ON DIAPHRAGMATIC ELECTRICALLY INACTIVE AREA(1)

- 1) QRS loop with efferent limb of clockwise rotation, heading from right to left and located above the orthogonal X lead (at least 80% of the QRS loop presents clockwise rotation);
- 2) Abnormal superior dislocation of the initial 20 to 40 ms vectors (at least 25 ms located above the orthogonal X lead);
- 3) The time from the zero point up to the intersection with the orthogonal X lead will be at least 25 ms. If each dash has a 2 ms duration, there must be obligatorily, more than 12 dashes above the orthogonal X lead;
- 4) Efferent limb always totally above the orthogonal X lead (with the possible exception of the initial 10 ms vector);
- 5) Location of the maximal vector between -40° and $+30^{\circ}$. Always less than $+15^{\circ}$;
- 6) Efferent limb of superior convexity in initial 20 to 40 ms;
- 7) The initial 10 ms vector may have a superior orientation (group I of Young and Williams) or inferior (group II). This variety is more rare;
- 8) Possible recording of an alteration in the middle-final portion of the QRS loop (afferent limb), called by Young et al¹ as type A, B, C and D deformities, present in quadrant I of the FP. These deformities would be useful in the cases in which the above criteria are doubtful or absent.

1. Young E, Levine HD, Vokonas PS, et al. The frontal plane vectorcardiogram in old inferior myocardial infarction. II. Mid-to-late QRS changes. Circulation. 1970; 42:1143-1162.

Electrically inactive area in inferior wall: segments 15, 10 and 4



LV Segment affected are numbers 15, 10 and 4. Electrically inactive area in inferior wall

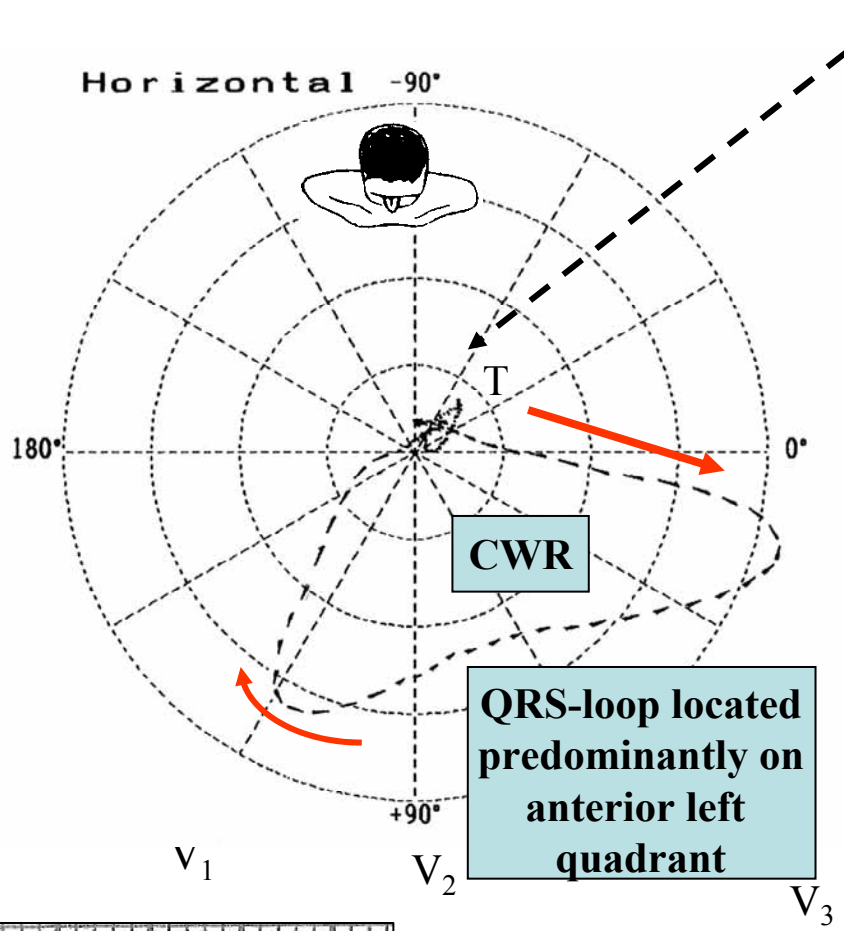
Gil Salles et al.(1) studied 738 outpatients in the chronic phase of Chagas' disease were enrolled in a long-term follow-up study. The patients were evaluated at least 2 times a year Maximal heart rate-corrected QT (QTc) and T-wave peak-to-end (TpTe) intervals and QRS, QT, JT, QTapex, and TpTe dispersions and variation coefficients were measured manually and calculated from 12-lead ECGs obtained on admission. Clinical, radiological, and 2-dimensional echocardiographic data were also recorded.

Primary end points were all-cause, Chagas' disease-related, and SCDs defined as that occurring in spontaneously or within 1 hour after the onset of symptoms in patients known to be previously stable. During a follow-up of 58±39 months, 62 patients died, 54 of Chagas' disease-related causes and 40 suddenly.

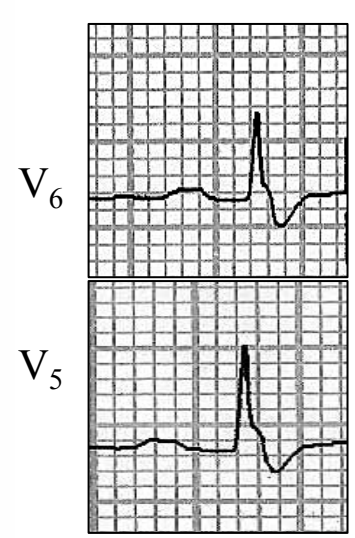
Multivariate Cox survival analysis revealed that the QT-interval dispersion (QTd) and echocardiographic LV end-systolic dimension were the most important mortality predictors in patients with Chagas' disease. Heart rate, **the presence on ECG of pathological Q waves**, frequent PVCs, and isolated LAFB refined the mortality risk stratification.

- 1. Salles G, Xavier S, Sousa A, Hasslocher-Moreno A, Cardoso C. Prognostic value of QT interval parameters for mortality risk stratification in Chagas' disease: results of a long-term follow-up study. *Circulation*. 2003 Jul 22;108:305-312.**

ECG/VCG correlation horizontal plane

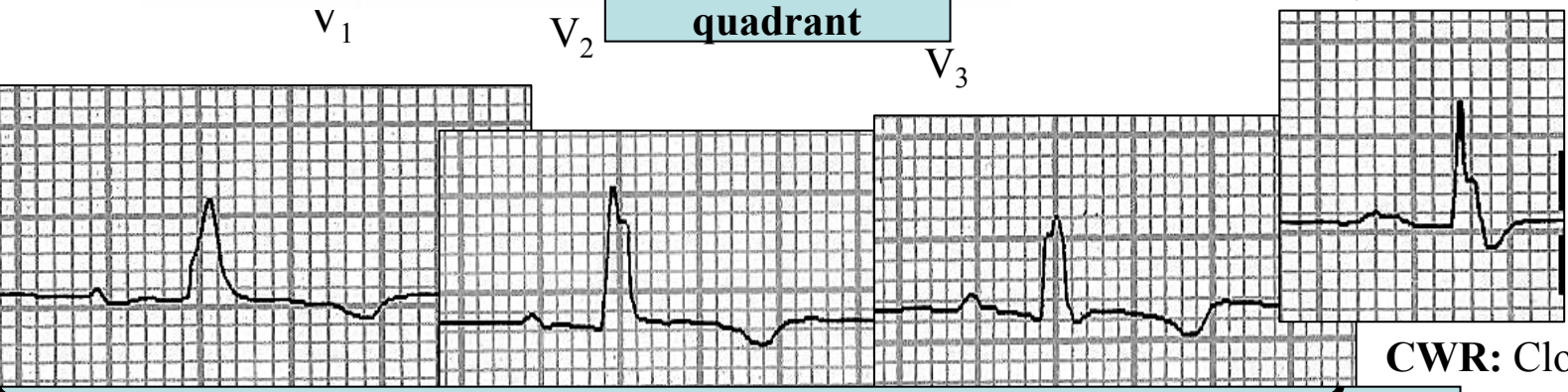


Initial 10 to 20 ms QRS vector directed to the left. Absence of normal convexity to the right of the initial 10 to 20 ms of the QRS loop: Absence of first middle septal vector or anteromedial (1_{AM})



Absence of initial q wave in V5, V6 and I (by absence of the vector 1_{AM}). Final S wave is not Indicative of End Conduction Delay (ECD).

It is consequence of final QRS loop is located in negative hemifield of V5-V6.



CWR: Clock Wise Rotation

Prominent Anterior QRS Forces: PAF

THE POSSIBLE CAUSES OF PROMINENT ANTERIOR QRS FORCES (PAF) (1-4)

- 1) Normal variant: counterclockwise rotation (CCWR) of the heart around the longitudinal axis and rarely in athlete's heart..
- 2) Misplaced precordial leads
- 3) Right Ventricular Enlargement (RVE)/ Right Ventricular Hypertrophy (RVH) Vectorcardiographic types A and B.
- 4) Diastolic, volumetric or eccentric Left Ventricular Enlargement (LVE) or Left Ventricular Hypertrophy (LVH) secondary to septal hypertrophy (magnitude of increase of the first vector or 1_{AM} vector) and/or CCW heart rotation of the heart around its longitudinal axis
- 5) **Strictly posterior or dorsal infarction (Old nomenclature).???**
- 6) Postero-lateral or Posterolateroinferior infarction, and lateral MI.

1. *Macfarlane PW, Lawrie TDV, eds. The normal electrocardiogram and vectorcardiogram. In: Comprehensive Electrocardiology: Theory and Practice in Health and Disease. Vol 1-3. New York, NY: Pergamon Press, 1989.*
2. *Yang TF, Macfarlane PW. Normal limits of the derived vectorcardiogram in Caucasians. Clin Physiol. 1994; 14: 633-646.*
3. *Yang TF, Chen CY, Chiang BN, Macfarlane PW. Normal limits of derived vectorcardiogram in Chinese. Electrocardiol. 1993; 26: 97-106.*
4. *Mattu A, Brady WJ, Perron AD, et al. Prominent R wave in lead V1: electrocardiographic differential diagnosis. Am J Emerg Med. 2001; 19: 504-513.*

1. **Complete Right Bundle Branch Block. (CRBBB)???**
2. Wolff-Parkinson-White syndrome with anomalous pathway of posterior location: Type A WPW
3. Hypertrophic cardiomyopathy: obstructive and non-obstructive forms.
4. Duchenne-Erb disease, pseudo hypertrophic muscular dystrophy linked to sex or infantile malignant (DMD).
5. Endomyocardial fibrosis
6. **Left Septal Fascicular Block (LSFB).**2;3;4
7. Associations of the previous ones: E.g.: RVE/RVH + CRBBB

1. *Cheng CH, Nobuyoshi M, Kawai C, et al. ECG pattern of left ventricular hypertrophy in non obstructive hypertrophic cardiomyopathy: The significance of the mid-precordial changes. Am Heart J 1979; 97:687-695.*
2. *Dhala A, Gonzalez Zuelgaray J, Deshapande S, et. al.: Unmasking the trifascicular left intraventricular conduction system by ablation of the right bundle block. Am J Cardiol 1996; 77: 706-712*
3. *Moffa PJ, Ferreira BM, Sanches PC, Tobias NM, Pastore CA, Bellotti G. Intermittent antero-medial divisional block in patients with coronary disease Arq Bras Cardiol 1997; 68:293-296*
4. *Uchida AH, Moffa PJ, Riera AR, Ferreira BM.Exercise-induced left septal fascicular block: an expression of severe myocardial ischemia. Indian Pacing Electrophysiol J. 2006 Apr 1;6:135-138.*

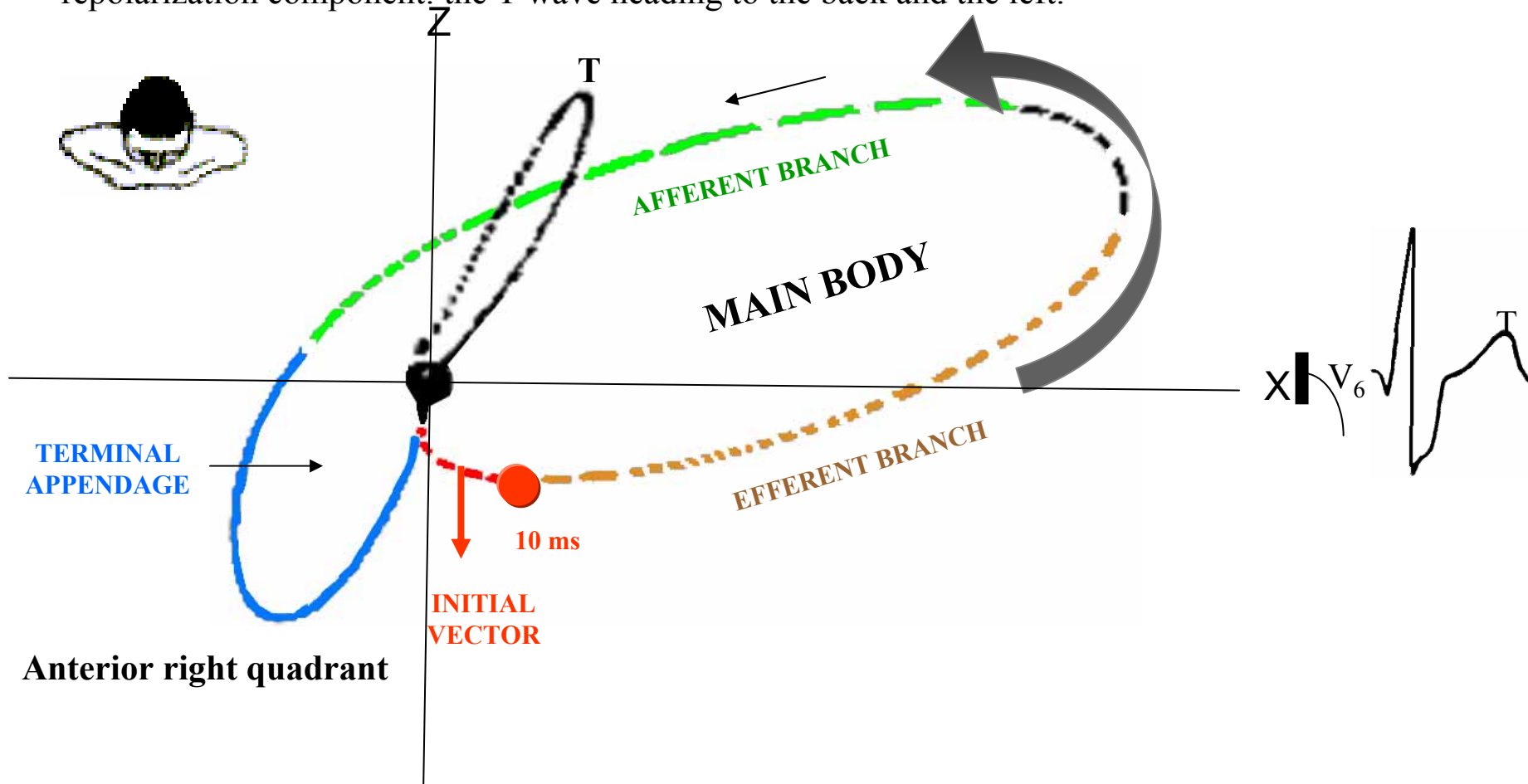
First question:

***Why these Prominent QRS
Anterior Forces(PAF) observed in
horizontal plane are not due to a
Complete Right Bundle Branch
Block?(CRBBB)***

The answer in next slides....

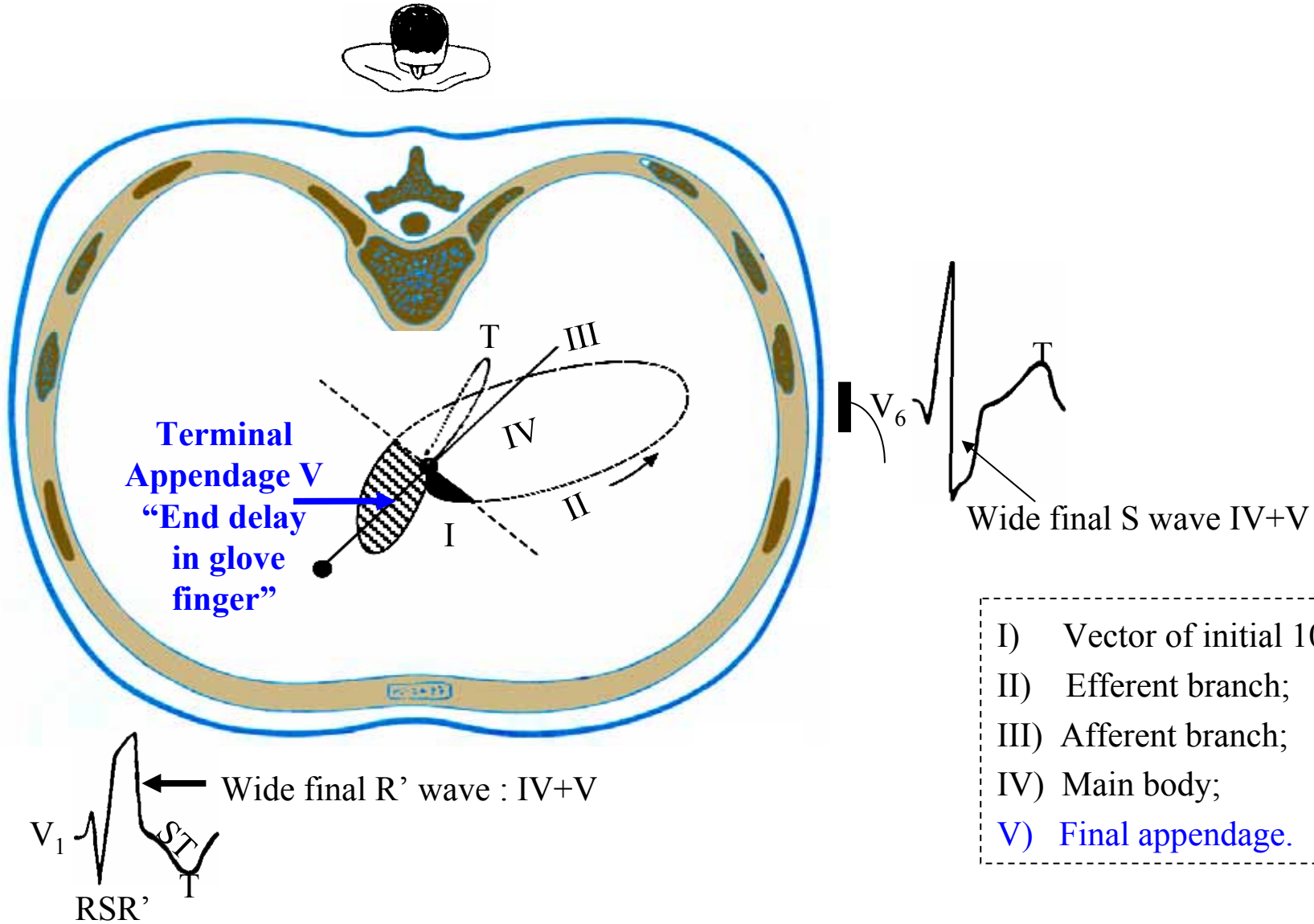
Vectocardiographic criteria of uncomplicated Complete Right Bundle Branch Block on Horizontal Plane

1. QRS loop of duration ≥ 120 ms. This corresponds to 60 or more dashes, in the cases in which 1 dash = 2 ms;
2. Maximal vector discretely decreased, pointing to the left and with a variable degree of anterior dislocation;
3. QRS loop in the HP formed by fourth components of depolarization: **initial 10 to 20ms vector**, **effere[n]t branch**, **afferent branch**, main body and terminal appendage after 60 ms with delay (slow recording) always located on anterior right quadrant. These 4 components are following by a repolarization component: the T wave heading to the back and the left.



ECG/ VCG CORRELATION IN THE HP WITH ECG IN CRBBB

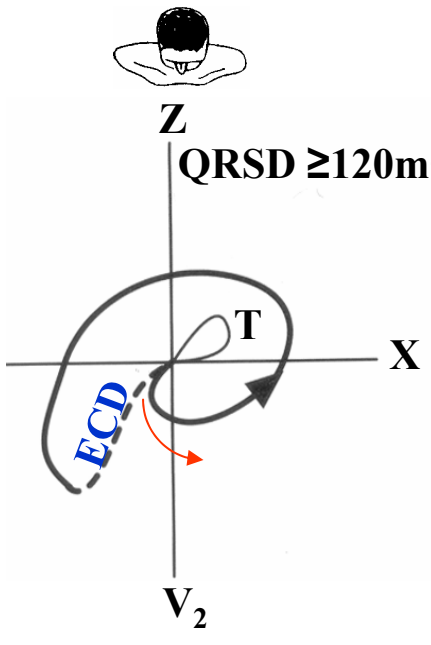
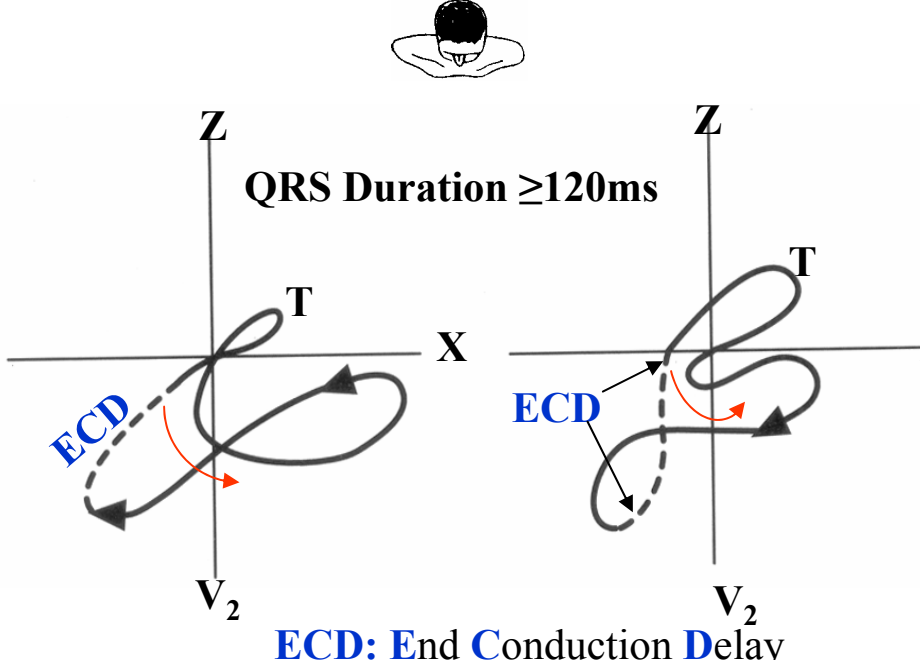
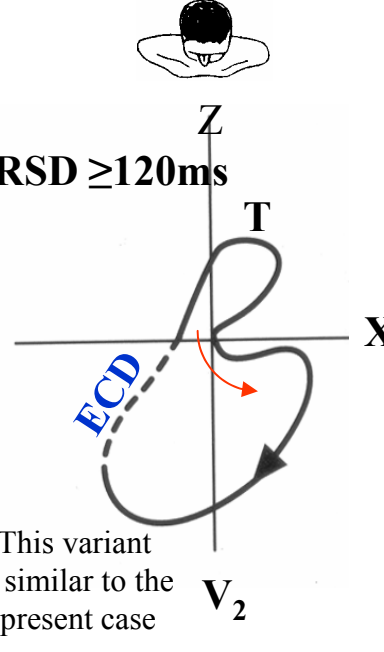
HP



QRS/T loops in the horizontal plane showing the 4 components of depolarization: initial vector, efferent branch, afferent branch and terminal appendage and T wave heading to the back and the left, and its correlation with the QRS/ST-T complex in V1 and V6.

Vectorcardiographic types of complete right bundle branch block: All characteristic in HP

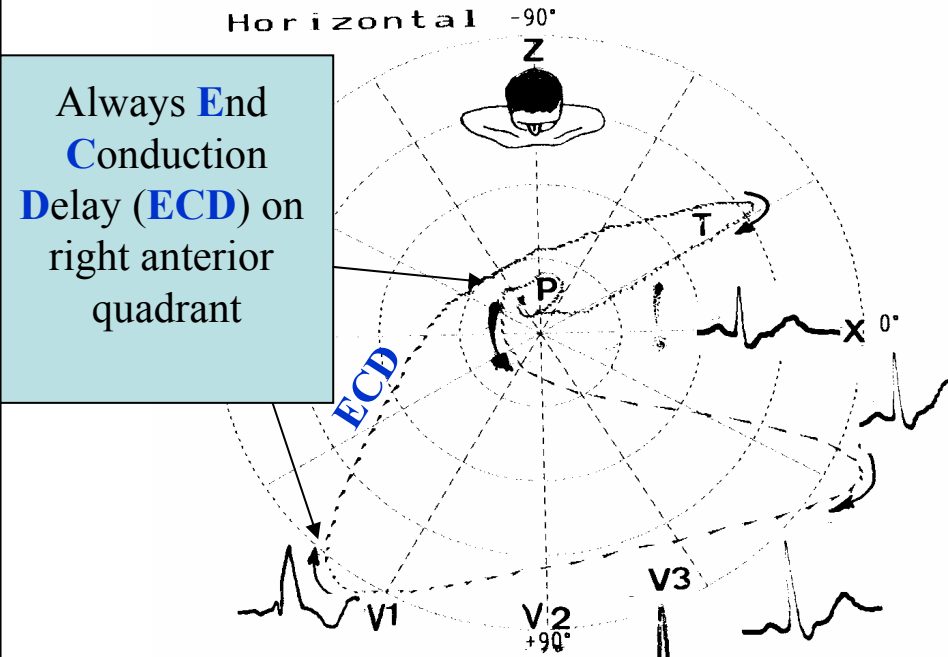
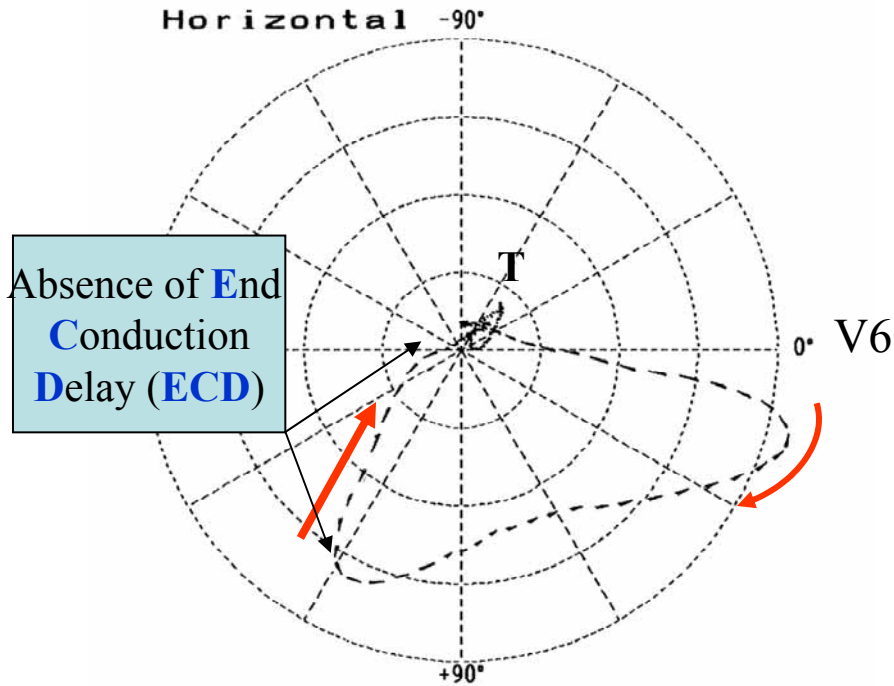
It takes into account only the QRS loop pattern in the horizontal plane we have 3 types of CRBBB

<p>“Grishman” Type or Kennedy type I.</p>	<p>“Cabrera” type or Kennedy type II.</p>	<p>CRRRB Kennedy type III or C.</p>
 <p>QRSD ≥ 120ms</p>	 <p>QRS Duration ≥ 120ms</p> <p>ECD: End Conduction Delay</p>	 <p>QRSD ≥ 120ms</p> <p>This variant is similar to the present case</p>
<p>Initial 10 to 20ms vector to the front</p>	<p>Initial 10 to 20 ms vector to the front,</p>	<p>Initial vector to the front,</p>
<p>Afferent branch behind the X line</p>	<p>Afferent branch facing the X line</p>	<p>Afferent branch facing the X line</p>
	<p>QRS loop in eight rotation and main body located in anterior quadrants</p>	<p>QRS loop of clockwise rotation and main body located in anterior quadrants.</p>
<p>Always ECD on right anterior quadrant</p>	<p>Always ECD on right anterior quadrant</p>	<p>Always ECD on right anterior quadrant</p>

Differential diagnose between Left Septal Fascicular block and Complete Right Bundle Branch Block

The present case (typical LSFB)

CRBBB Kennedy VCG type III or C



Initial 10 to 20 ms vector directed to the left and eventually to back Absence of normal convexity to the right of the initial 20 ms of the QRS loop of HP.

Initial 10 to 20 ms vector always to the front.

QRS loop of clockwise rotation (complete LSFB) and main body located predominantly in left anterior quadrant

QRS loop of clockwise rotation and main body located predominantly in right anterior quadrant

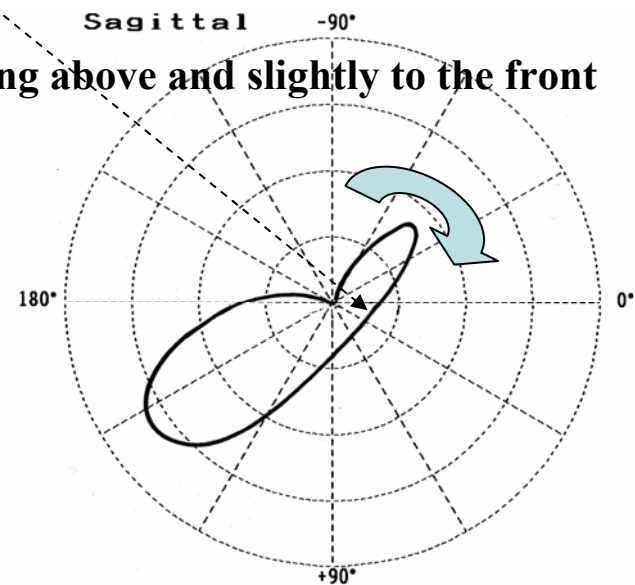
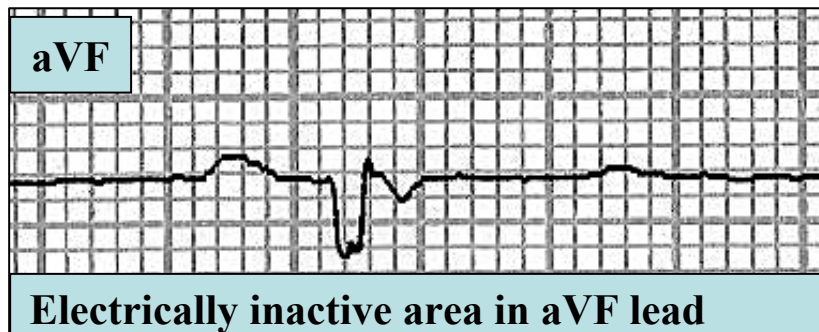
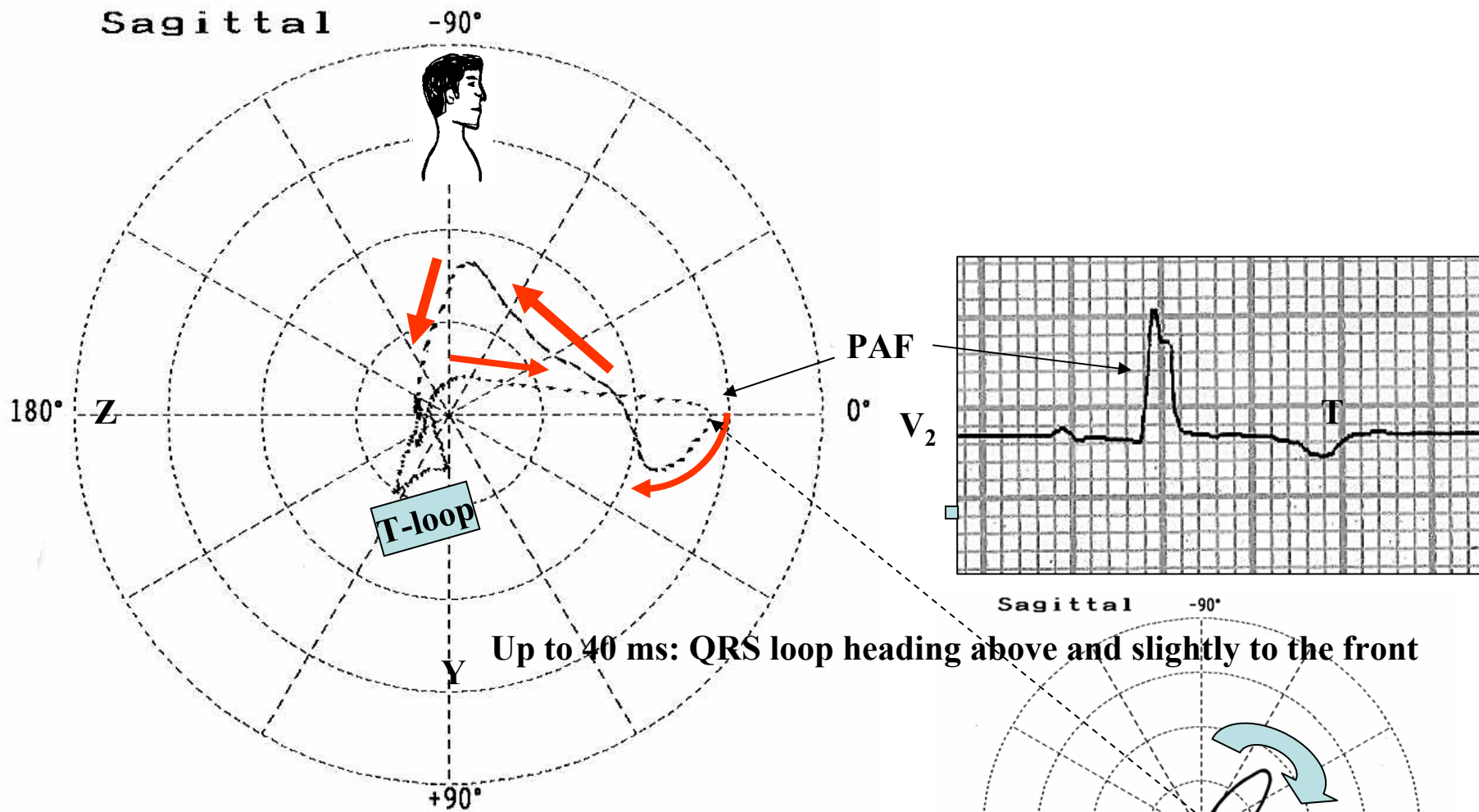
QRS duration usually <120ms. Rarely ≥120ms

QRSD ≥120ms

Final portion of QRS loop without ECD

Terminal appendage after 60 ms with delay (slow recording) always located on anterior right quadrant.

ECG/VCG correlation right sagittal plane



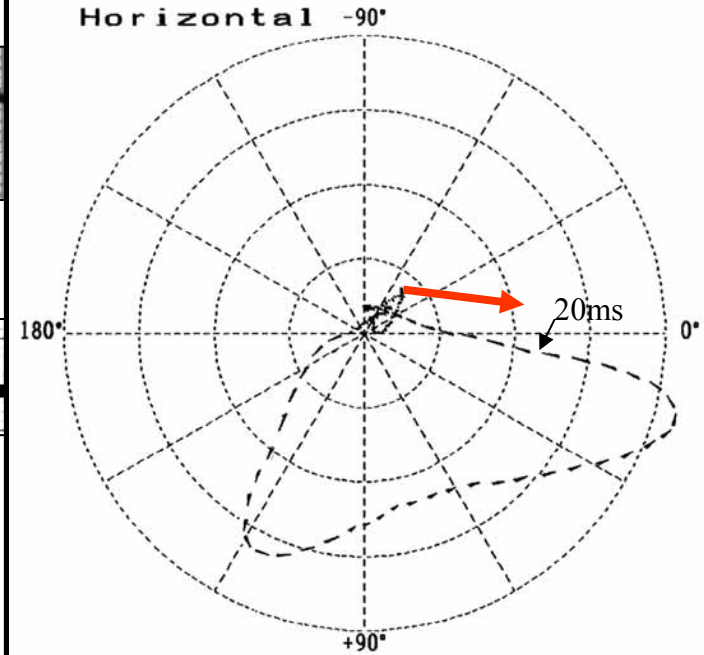
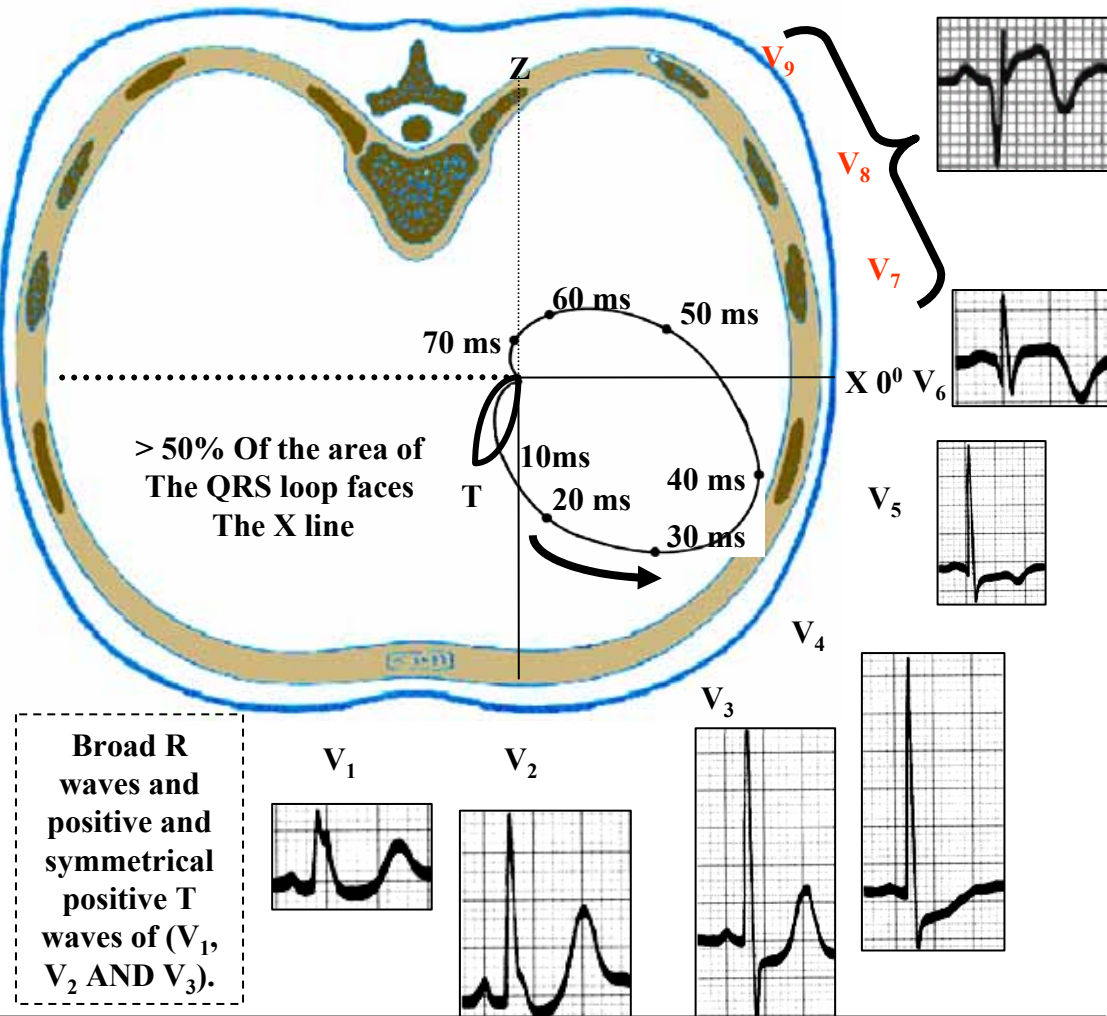
Second question:

Why these Prominent QRS Anterior Forces(PAF) observed in horizontal plane are not due to a inferobasal myocardial infarction?(Strictly posterior MI in ancien nomenclature)

The answer in next slide....

INFEROBASAL MYOCARDIAL INFARCTION ANCIEN STRICTLY POSTERIOR OR DORSAL MI

The present case



In the present case the first 10 to 20ms vector of the QRS loop is affected and directed from right to left and frequently to back: absence of q wave in V5, V6 and I (by absence of the vector 1_{AM})

The 10 to 20ms initial vectors have essentially normal characteristics, since these vectors represent depolarization potentials arising in uninvolved regions of the septum that are activated early in the QRS interval.

DIFFERENTIAL DIAGNOSIS BETWEEN DORSAL INFARCTION AND LSFb

	DORSAL INFARCTION	LSFB
Artery territory involved when both are of coronary etiology	RCA or Cx or PD branch	In 100% before the first septal perforating artery.
Association with RBBB or LAFB	Rare. Pseudo IRBBB in 40%.	Frequent.
R wave of V₁- V₂ with a duration ≥40 ms and R/S ratio in these leads greater than 1	Yes	No
Terminal R', R with notch or rsR', RSR', rSR' rsR'' in V_{1y} V₃R:	Frequent: 40% called pattern of pseudo IRBBB	No
Association with inferior infarction (postero-inferior): Q of II y aVF ≥ 40 ms and Q wave ≥ 25% of R width associated to R of V₁- V₂ with a duration ≥ 40 ms and R/S ratio in these leads greater than 1:	Frequent observation: in inferior infarction the presence of ST with depression > 1 mm in right precordial leads, which indicates dorsal extension, pointing out Cx or distal RCA occlusion.	Rare Such this case.
Association with inferolateral or lateral infarction:	Frequent: Q in aVL, I, II, III and aVF	Rare. Such this case
Association with RV + inferior + lateral infarction:	Possible. The extension to the RV is translated by QS in V ₄ R and V ₅ R, ST elevation in III > II and ST elevation in V ₂ /elevation in aVF ratio < 0.5	No
Positive epidemiology for Chagas:	Negative	Frequent in Latin America countries
Q waves >40ms in V₆- V₇ -V₈ and V₉:	Present	No
Possible low voltage of QRS complexes in the FP (< 5 mm)	Yes	No
Intermittence of the phenomenon:	No	Possible and diagnostic.
T wave	Anterior, symmetrical and wide-based.	Heading backward.

	DORSAL INFARCTION	LSFB
In the acute phase of the event:	R wave in crescendo from V ₁ to V ₃ and negative T wave in V ₁ .	From V ₁ to V ₃ ST segment with depression of superior concavity followed by positive and prominent T wave in V ₃ R
QRS loop of the HP located in the anterior quadrants at least up to 40 ms	Always	Variable
Absence of convexity to the right of the initial 10-20 ms of the QRS loop in the hp:	Absent. Vector of initial 20 ms normal. Not affected. Always to the front and the right.	Characteristic. It may go backward and to the left by predominance of the 1PI vector.
Direction of the rotation of the QRS loop in the hp:	Always counterclockwise or in eight.	It may be counterclockwise or clockwise.
Anterior dislocation only of the afferent limb in the horizontal and sagittal planes:	Typical	No
Hypokinesis in infero-dorsal wall by echo or by ventriculography	Frequent	Rare. Such this case.
Better diagnosed by VCG accuracy, predictive values :	Yes, when strictly posterior	Yes

A) ELECTROCARDIOGRAPHIC:

- 1) Normal QRS duration or with a discrete increase (up to 110 ms).
- 2) Frontal Plane leads with no modifications: normal QRS if isolated.
- 3) Increased intrinsicoid deflection of V_1 and V_2 .
- 4) R wave voltage of $V_1 \geq$ than 5 mm;
- 5) R/S ratio in $V_1 > 2$, if Rs pattern present;
- 6) R/S ratio in $V_2 > 2$ if Rs pattern present;
- 7) S wave depth in $V_2 < 5$ mm.
- 8) Possible small initial q wave in V_2 or V_1 and V_2 ;
- 9) R wave of $V_2 \geq 15$ mm height (low sensitivity);
- 10) RS or Rs in V_2 and V_3 (frequent rS in V_1) with R wave "in crescendo" from V_1 to V_3 and decreasing from V_5 to V_6 ;
- 11) Absence of q wave in V_5 , V_6 and I (by absence of the vector 1_{AM})
- 12) Prominent QRS Anterior Forces (PAF): permanent, intermittent(2;3;) or transient (4)

1. *Athanassopoulos CB. Transient focal septal block. Chest 1979 Jun;75:728-730.*
2. *Hassapoyannes CA, Nelson WP. Myocardial ischemia-induced transient anterior conduction delay. Am Heart J 1991; 67:659-660.*
3. *Mori H, Kobayashi S, Mohri S. Electrocardiographic criteria for the diagnosis of the left septal fascicular block and its frequency among primarily elderly hospitalized patients. Nippon Ronen Igakkai Zasshi 1992;29:293-7*
4. *Riera AR, Ferreira C, Filho CF, Dubner S, et al. Wellens syndrome associated with prominent anterior QRS forces: an expression of left septal fascicular block? J Electrocardiol. 2008 Nov-Dec; 41(6):671-4*
5. *Pérez Riera AR, Ferreira C, Ferreira Filho C, Dubner S, Baranchuk A. Electrovectorcardiographic diagnosis of left septal fascicular block: anatomic and clinical considerations. Ann Noninvasive Electrocardiol. 2011 Apr;16(2):196-207.*

B) VECTOCARDIOGRAPHIC: (all in the Horizontal Plane) (1-4)

1. QRS loop in the HP with an area predominantly located in the left anterior quadrant ($\geq 2/3$ of the loop area facing the orthogonal X line);
2. Absence of normal convexity to the right of the initial 10 to 20 ms of the QRS loop;
3. Discrete dextro-orientation with moderate delay of the vector from 20 ms to 30 ms;
4. Anterior location of the vector from 40 to 50 ms;
5. Posterior location with a reduced magnitude of the vector from 60 to 70 ms;
6. Maximal vector of the QRS loop located to the right of $+30^\circ$;
7. T loop of posterior orientation (useful for the differential diagnosis with inferobasal old dorsal infarction);
8. The QRS loop rotation may be:
 - (8a) Counterclockwise: incomplete LSFB.
 - (8b) Clockwise: advanced or complete LSFB or in association with CRBBB, LAFB or LPFB.

1. *Alboni P, Malacarne C, De Lorenzi E, Pirani R, Baldassarri F, Masoni A. Right precordial q waves due to anterior fascicular block. Clinical and vectorcardiographic study. J Electrocardiol. 1979 Jan;12(1):41-48.*
2. *Tranchesi J, Moffa PJ, Pastore CA, et al. Block of the antero-medial division of the left bundle branch of His in coronary diseases. Vectrocardiographic characterization. Arq Bras Cardiol 1979;32:355-360.*
3. *Inoue H, Nakaya Y, Niki T, Mori H, Hiasa Y. Vectorcardiographic and epicardial activation studies on experimentally –induced subdivision block of the left bundle branch. Jpn Circ J 1983;47:1179-1189.*
4. *Riera AR, Kaiser E, Levine P, Schapachnik E, Dubner S, Ferreira C, Filho CF, Bayés de Luna A, Zhang L. Kearns-Sayre syndrome: electro-vectorcardiographic evolution for left septal fascicular block of the his bundle J J Electrocardiol. 2008 Nov-Dec; 41(6): 675-8.*

ELECTRO-VECTORCARDIOGRAPHIC FINAL DIAGNOSIS

- 1) Sinus Rhythm**
- 2) Heart Rate 47bpm: Sinus bradycardia**
- 3) P axis +60°, P voltage 1,5mm, P duration 130ms(prolonged): Left Atrial Enlargement**
- 4) PR interval 190 ms : normal**
- 5) QRS axis -65° Extreme left axis deviation consequence of loss of inferior forces**
- 6) Electrically inactive area in inferior wall**
- 7) Prominent Anterior QRS Forces (PAF): Left Septal Fascicular Block (LSFB). Why this woman has not Complete RBBB? Because absence of first vector directed to front and absence of right End Conduction Delay (ECD) on right anterior quadrant ALWAYS present in a CRBBB.**
- 8) Why this woman has not dorsal infarction? Because dorsal infarction does not affect initial 10 to 20 ms vector of QRS loop.**
- 9) Polymorphic Premature Ventricular Contractions (PVCs).**

What was the reason for the particular case to be chosen?

Answer:

The choice was aimed at proving the significance of the vectorcardiogram, a method currently relegated to a few teaching and researching centers. Without the VCG, the diagnosis of this dromotropic disorder would not have been possible.

What was the learning experience for you from the case?

The experience and the learning that the present case left us, is the clear idea that we should not accept what literature teaches us as certain and passively. We should always try to find the truth at all costs, and if possible break a paradigm. The Left Septal Fascicular Block (LPFB) is a reality and the present case markedly contributed to reinforce the idea that the human His system is tetrafascicular and not trifascicular (the right branch plus three fascicles in the left branch).

How did the case change the way you cared and treated future patients?

The present case shows us that when there are prominent QRS anterior forces in the absence of right overload, dorsal infarction may not correspond to right bundle branch block, even recording final S waves in the left leads.

Therapeutic options:

First it is necessary and imperative to stratify risk. The stratification is controversial, but studies conducted by Rassi et al(1) in Brazil, showed us that risk stratification should assess four elements: NYHA III/IV functional class, cardiomegaly in chest X-ray, involvement of LV function evaluated by echo and catheterization, and 24 h Holter looking for the presence of nonsustained VT.

Prognostic factors and risk stratification

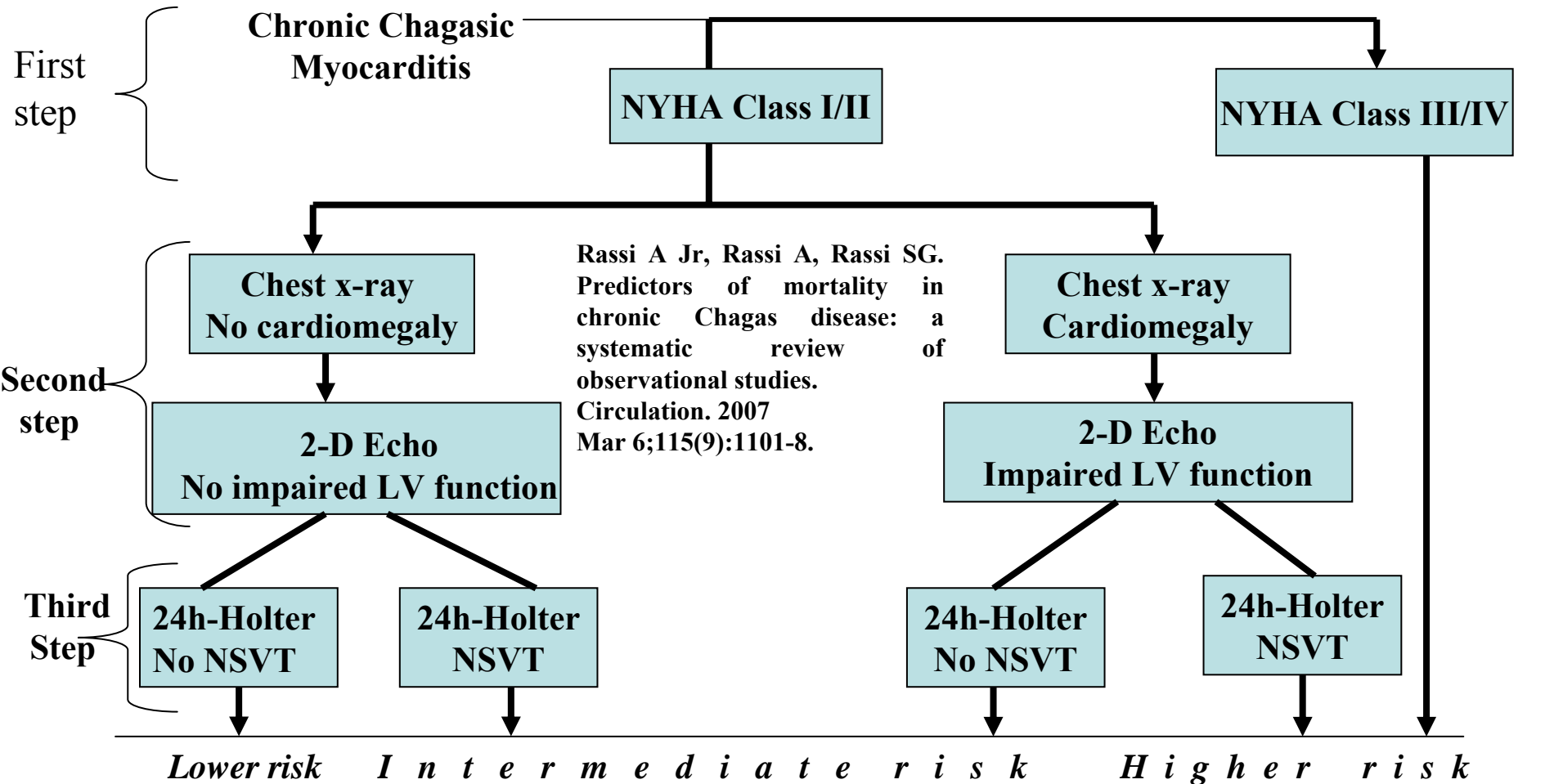
CHD is a heterogeneous condition with a wide variation in clinical course and prognosis. Many patients remain asymptomatic throughout life; some have only conduction defects and mild segmental wall motion abnormalities; some develop severe symptoms of HF, thromboembolic phenomena and multiple disturbances of rhythm; and others die suddenly, often in the absence of previous significant symptoms. In general, SCD (usually due to VF) is the most common cause of death (55-65% of patients), followed by CHF (25-30% of patients) and cerebral or pulmonary embolism (10-15% patients). Although SD predominates in patients with less extensive myocardial involvement and in those with significant pre-existing ventricular arrhythmias, death from pump failure is slightly more common in patients. Knowledge of predictors of unfavourable outcome can help to identify patients at different degrees of risk, facilitate choice among treatment alternatives. Despite differences in the sample populations between studies (e.g., some studies included only patients with CHD while others included patients with and without manifested cardiomyopathy) and in the set of prognostic variables investigated in each study, four independent markers of increased risk of death were identified:

- 1. NYHA functional class III/IV**
- 2. Cardiomegaly on chest radiography**
- 3. Impaired LV function by echo or cineventriculography**
- 4. NS-VT on 24 h Holter monitoring.**

1. Rassi Jr A, Rassi A, Marin-Neto JA. Chagas heart disease: pathophysiologic mechanisms, prognostic factors and risk stratification. Mem Inst Oswaldo Cruz. 2009 Jul;104 Suppl 1:152-8.)

Patients in NYHA class III/IV (because all of them invariably manifest associated cardiomegaly on chest radiography, global systolic dysfunction on echocardiogram and NSVT on Holter monitoring) and patients who are in NYHA class I/II and also have LV dysfunction on echocardiogram and NSVT on Holter monitoring are at the highest risk of death and should be regarded as candidates for aggressive therapeutic management. Conversely, patients with an abnormal ECG but who are in NYHA class I/II with neither LV dysfunction on echocardiography nor NSVT on Holter are at low risk of death. These patients should be followed up annually or biannually. Between these two extremes are patients with either LV dysfunction or NSVT. Such patients are at intermediate risk and their treatment strategies should be individualized.

Proposed algorithm to guide mortality risk assessment and therapeutic decision making in Chagas myocarditis



Annual reevaluation. Antiparasitic drugs?	Antiparasitic drugs? Amiodarone?	ACE inhibitors Amiodarone, Diuretic B-blockler. ICD?	ACE inhibitors Diuretic β-blockers Amiodarone ICD?	Digitalis Diuretic ACE inhibitors Spironolactone β-blockers Amiodarone ICD Cardiac Transplant
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