AN ECG/VCG BORDERLINE

UN ECG/VCG LIMÍTROFE

By Andrés Ricardo Pérez-Riera MD PhD

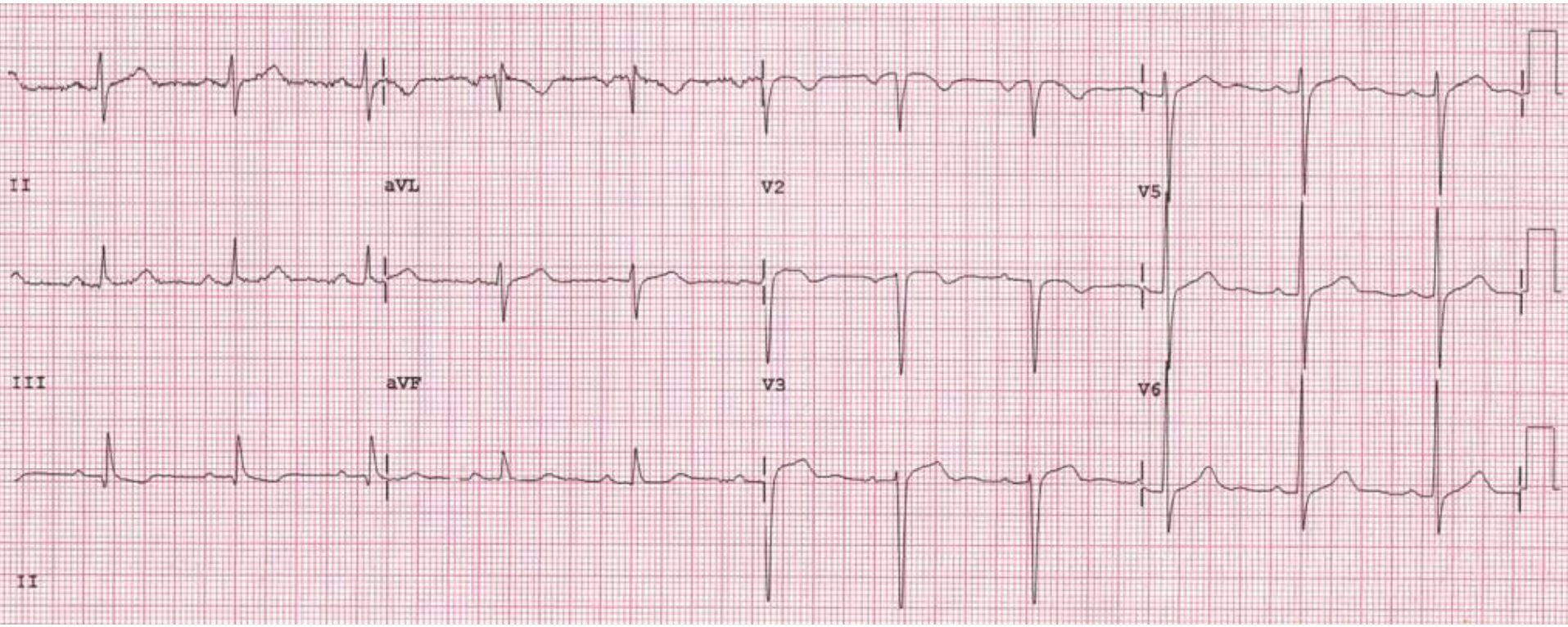
Masculino, asiático, 58anos, nivel superior, sem vícios. Assintomático.
Motivo da consulta: avaliação prévia a iniciação da prática esportiva.
Antecedentes pessoais: refere no passado asma brónquica que desaparera na adolescência. Antecedentes familiares não significativos.
Exame físico: normal con excepção de discreta hipertensão arterial desconhecida até o momento exame PA 145/95. MAPA/24h confirma hipertensão arterial sistémica em 75% das aferições diurnas. Queda normal da PA noturna.
Rx tórax PA: botão aórtico saliente com Indice cardio-torácico normal.
Ecocardiograma transtorácico normal.
Prova de função pulmonar: distúrbio ventilatorio obstrutivo leve prova farmaco-dinâmica não significativa ao broncodilatador aplicado (salbutamol 400mcg)
Pergunta: qual o diagnóstico ECG/VCG e como se explica?

Male Asian 58yo, higher level instruction, no vices. Assymtomatic.
Reason for consultation: evaluation prior to initiation of the sport.
Personal history of bronchial asthma in the past relates to disappear in adolescence.
No significant family history.
Physical examination: normal except for mild hypertension known to date examining (BP 145/95mmHg). Ambulatory bood pressure monitoring 24h confirms systemic hypertension in 75% of daytime measurements. Normal nocturnal BP fall.
PA chest x-ray: prominent aortic knob with normal cardiothoracic index.
Normal transthoracic echocardiogram.
Spirometry: mild obstructive respiratory disease pharmaco-dynamic test does not apply significant bronchodilator (salbutamol 400 mcg)
Question: What is the electrocardiographic/VCG diagnosis and how is it?

Identification:

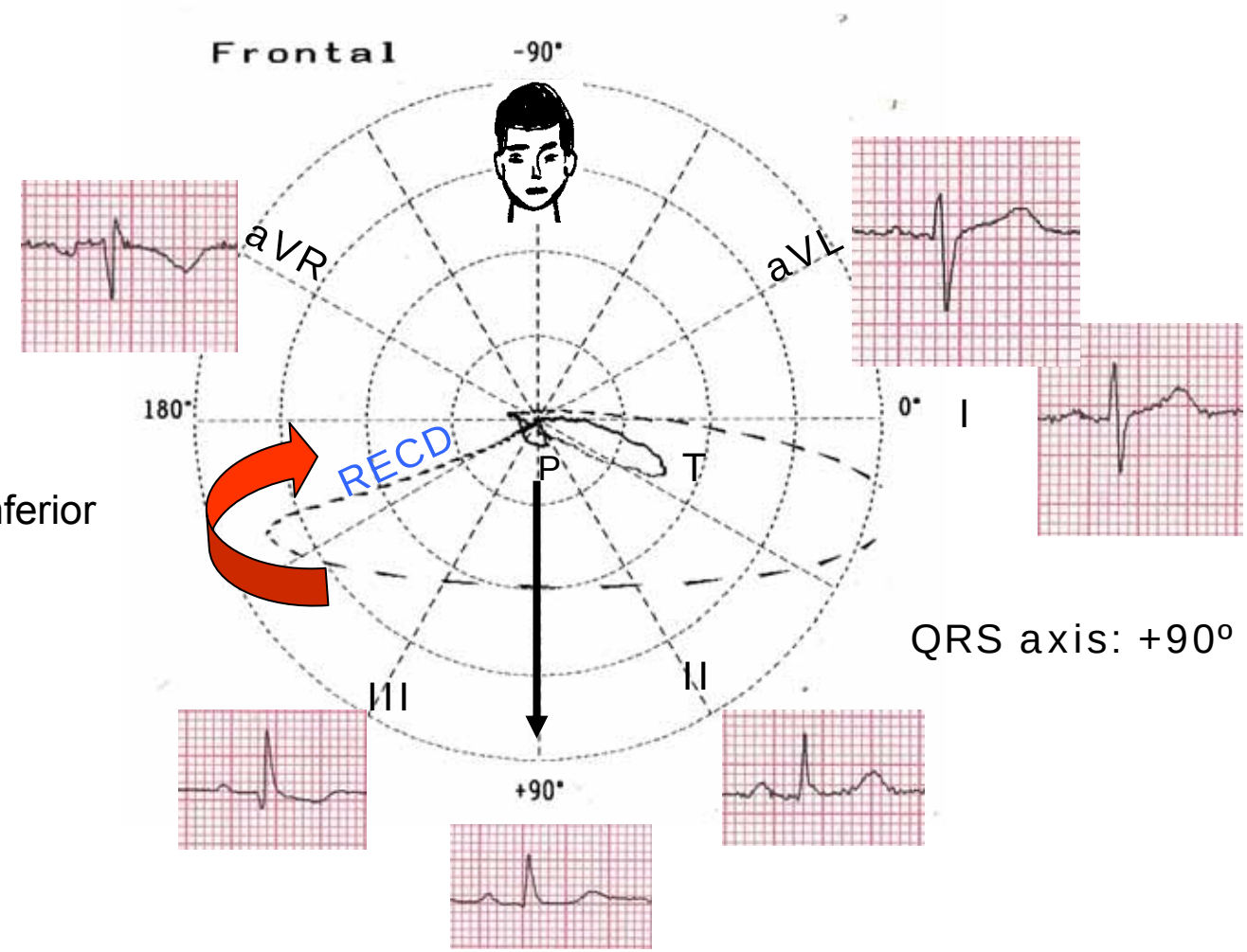
Name: AS**; Age:** 58 yo; **Gender:** Male; **Ethnic Group:** Asian; **Weight:** 82kg; **Height** 1,74m.

Date: June 20 2011



ECG analysis: HR 68bpm P axis: + 43º; PR interval: 192ms; QRSD: 94ms, QRS axis: +90º vertical heart; T axis: + 22º; QT: 429; QTc 451. Vertical heart. Poor R wave progression in precordial leads. Transition zone displaced to leftward. In normal adults the transitional zone usually is located between lead V_2 and V_4. When is displaced leftward and beyond lead V_5 clock wise rotation is present. Leftward displacement of transitional zone is observed in older subject.

ECG/VCG CORRELATION ON FRONTAL PLANE



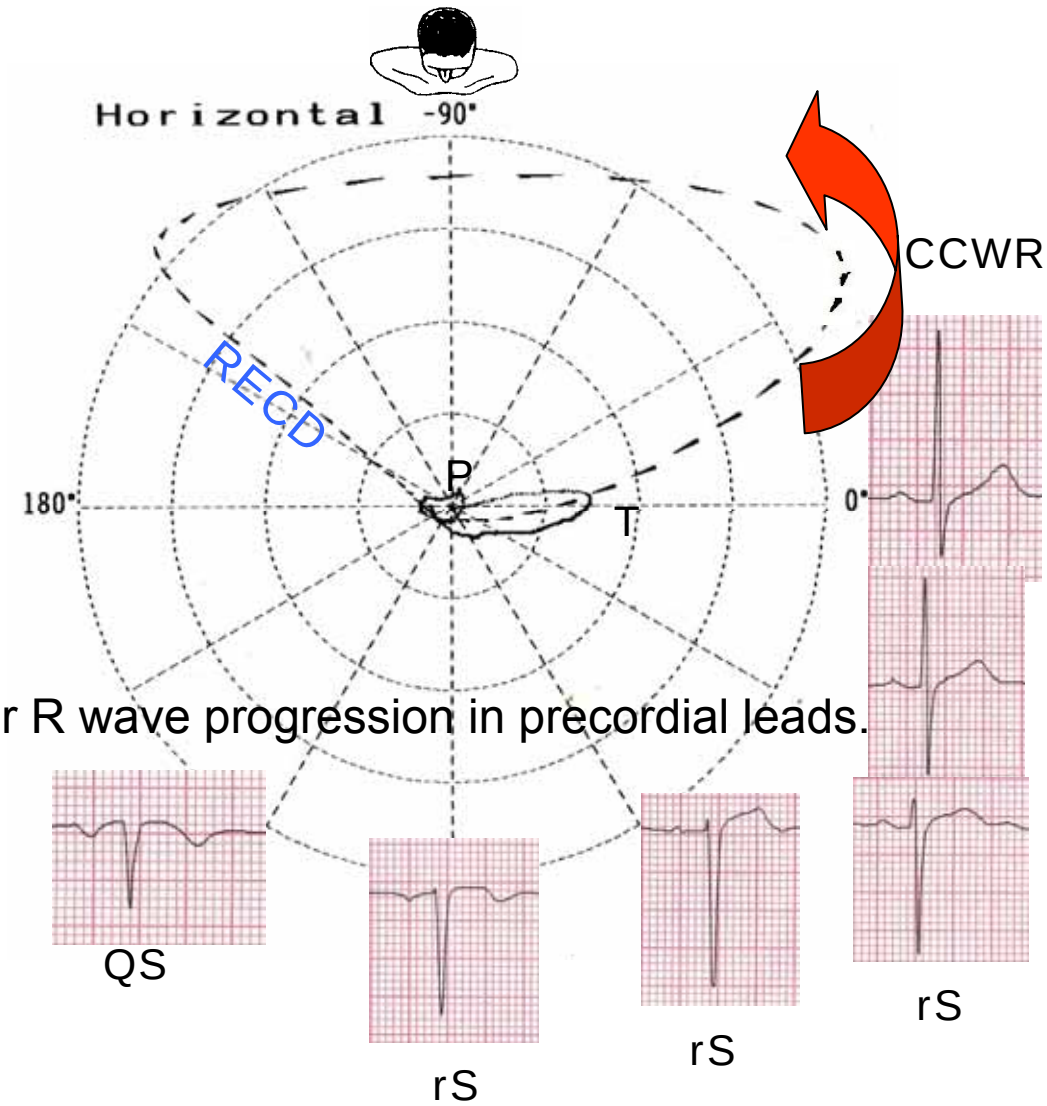
RECD on right inferior quadrant.

QRS axis: +90º

RECD: Right End Conduction Delay

ECG/VCG CORRELATION ON HORIZONTAL PLANE

RECD on right
Posterior quadrant

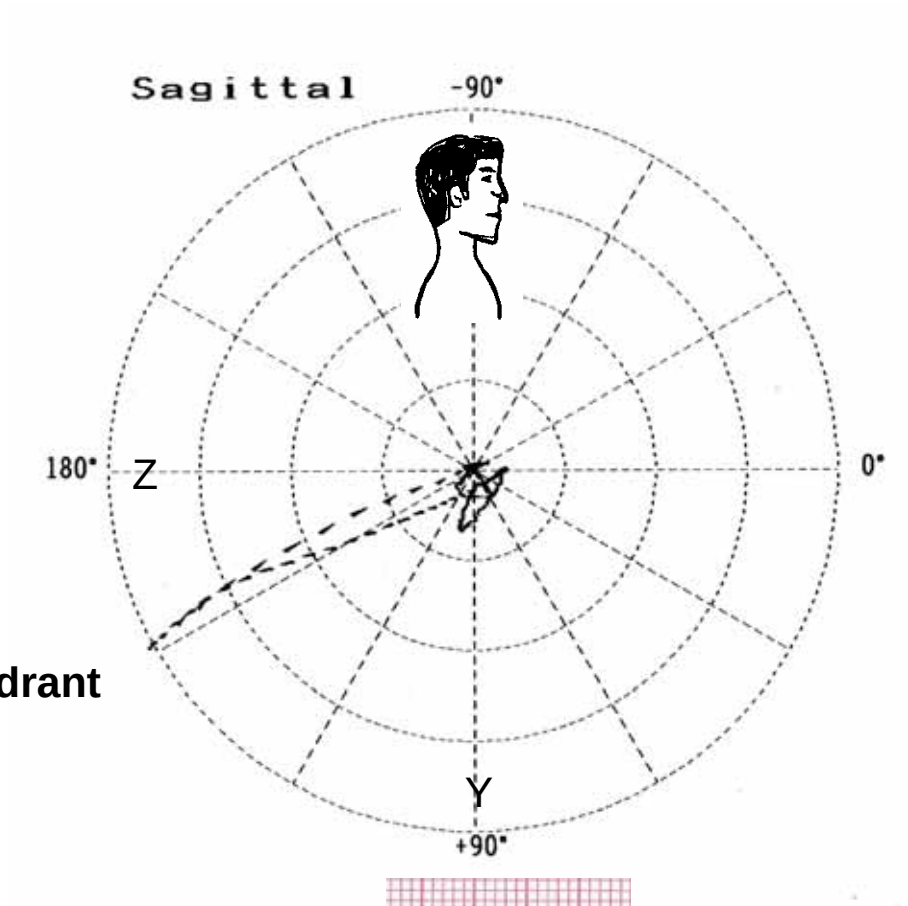


Poor R wave progression in precordial leads.

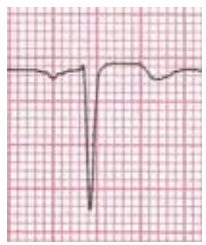
CCWR: Counter Clock Wise Rotation of QRS loop

RECD: Right End Conduction Delay

ECG/VCG CORRELATION ON RIGHT SAGGITAL PLANE

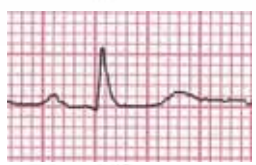


V_2



QRS loop 100% on Posteroinferior quadrant

aVF



Nike propaganda →

COLLEAGUES OPINION

Queridos amigos del forum voy a tratar de analizar el ECG del hombre con hipertension arterial moderada: Corazon horizontal; con SI- QIII. Derivaciones precordiales: P invertidas en V1 .y V2 sugeriendo que que estas derivaciones se registraron en una posicion alta, No hay progresion de las r hasta V_4 (pobre progresión) , generalmente indicando un proceso fibrotico en el septo izquierdo. La falta de progression de las ondas r en las precordiales puede ser debido en en sexo masculino a un proceso fibrotico, a un infarto crónico o a una reacción a sobrecarga sistolica y en casos raros a una miocardiopatia diabética. En mujeres puede ser debido a la menopausia (en ratas oviariectomizadas aparece fibrosis septal). La punta de la onda T en V_2 es negativa , probablemente debido al registro alto de V2 (P Iinvertida). La persistencia de las ondas S, s hasta V_6 que indica? como no existe desviacion del eje frontal a la izquierda , (que llevan el eje anteroposterior hacia atrás), entonces se debe explicar este fenomeno. El eje anteroposterior esta desviado hacia la cara posterior, es decir que las fuerzas electrotonicas posteriores son dominantes. Podria ser que este hombre tuviese una hipertrofia fisiologica que viene arrastrando desde su juventud Estas hipertofias la detecta el ECG ,pero no el ecocardiograma ,ya que son hIpertrofias longitudinales. Esto lo podremos saber si se registró un ECG 10 anos antes. Y queda otra posibilidad No todos los hipertensos aun los severos, hacen hipertrofias cardiacas ,generalmente debido a una mutacion en algunas moleculas que intervienen en el complicada cascada de las hipertrofias. Algunos hacen hipertrofias severas con sobrecargas moderadas , vean el articulo que envie al Circulation 2002 de Esposito, explicando este fenómeno Otro concepto importante es que las sobrecargas sistolicas .comienzan con hipetroficas fisiologica, pero despues aparece hipertrofias transversales, con signos muy evidentes de hipertrofia en el ECO y ECG y al final dilatation del VI. Es muy probable que este está en la primera etapa e hipertrofia que la expresa el ECG y no el ECO.Este último método descubrira en la segunda etapa de la hipertrofia. En fin me parece que este paciente tiene una hipertrofia fisiolologica excéntrica que la arrastra de la juventud o una hipetrofia posterior excéntrica fisiologica como primera etapa de la sobrecarga sistolica.

Un fraternal abrazo , y perdon por la larga discusion ante un ECG inocente no dramático

Samuel Sclarovsky

Dear friends from the forum, I will try to analyze the ECG by the man with moderate high blood pressure: horizontal heart; with SI-QIII. Precordial leads: inverted P in V1 and V2, suggesting that these leads were recorded in a high position. There is no progression of r until V4 (poor progression), generally indicating a fibrotic process in the left septum. The lack of progression of r waves in precordial leads could be due to, in the male sex, a fibrotic process, a chronic infarction, or a reaction to systolic overload, and in rare cases to a diabetic cardiomyopathy. In women, it could be due to menopause (septal fibrosis appears in rats with ovariectomy). The tip of the T wave in V2 is negative, probably due to the high recording of V2 (inverted P). The persistence of S waves, s up to V6, what does it mean? Since there is noshift of the frontal axis to the left (which lead the anteroposterior axisto the back), then this phenomenon should be explained. The anteroposterior axis is shifted to the posterior side, i.e. that the posterior electrotonic forces are dominant. Maybe this man has a physiologic hypertrophy, which he carries since his youth. These hypertrophies are detected in ECG, but not in the echocardiogram, since they are longitudinal hypertrophies. We would know about this, if an ECG was taken 10 years earlier. And there is another possibility. Not all hypertensive patients, even severe ones, develop cardiac hypertrophies, generally due to a mutation in some molecules that intervene in the complex cascade of hypertrophies. Some develop severe hypertrophies with moderate enlargements. Check the article that I have sent to Circulation 2002 by Esposito, explaining this phenomenon. Another important concept is that systolic overloads start with physiologic hypertrophies, but later transversal hypertrophies appear, with very evident signs of hypertrophy in the Echo and ECG, and finally LV dilatation. Very likely, this patient is in the first stage and with hypertrophy expressed in ECG but not in Echo. The last method will uncover it in the second stage of hypertrophy. Anyway, I think this patient has an eccentric physiologic hypetrophy that he carries since his youth, or a physiologic eccentric posterior hypertrophy as a first stage of systolic overload. Warm regards, and I apologize for the lengthy discussion about anon-dramatic innocent ECG. Samuel Sclarowsky.

Dr Perez Riera: QRS forces (but not P!) consistent with mild emphysema--- vertical axis + poor r-wave progression V1-V3 + mild ST –T abnormality III and aVR (frontal R-T angle difference approximately 80 degrees (90 deg.- 10 deg.)

Of course I am not aware of the ethnic norms for Asians. Please advise

With thanks, David H. Spodick, MD, FACC, MACP, FCCP, FAHA

Spanish: Las fuerzas del QRS pero no la P son sugestivas de discreto enfisema: por el eje vertical y la pobre progresión del crescimiento de la r en V1 a V3, discretas alteraciones del ST y T en III y aVR.

Por supuesto que yo no conozco los patrones para asiaticos. Por favor me informe

Gracias

David.

Dr David Spodick biography.

Dr. David H. Spodick attended Bard College and was awarded a Doctorate in Science for his work in the field of noninvasive clinical cardiology and physiology. He interned at St. Francis Hospital in Hartford, and completed his residency training at Beth Israel Hospital and New England Medical Center. He also served in the Air Force, which afforded him the opportunity to travel extensively. Travel later became an integral part of his professional career. Although Dr. Spodick became interested in the emerging subspecialty of cardiology during his residency, his career started when he became David Littmann's first fellow in cardiology in 1956. After participating as a special post-doctoral fellow, sponsored by the National Heart Institute at the West Roxbury Veterans Administration Hospital, he moved to the Lemuel Shattuck Hospital. He then began a 19 year academic career including academic appointments at all three of the Boston medical schools and read all of the Boston Evening Clinic's electrocardiograms for 15 years without remuneration.

David Spodick became Chief of Cardiology at St. Vincent Hospital in 1976, where he joined Chief of Medicine Gilbert Levinson, an established cardiovascular researcher. Dr. Spodick has remained at St. Vincent Hospital, where he is a skilled practitioner and revered educator. Until recently, he also oversaw the Noninvasive Unit. His academic appointment at the University of Massachusetts Medical School has enriched the young careers of countless medical students.

David Spodick's career as a clinician, researcher, educator, and administrator in cardiovascular medicine continues to evolve after 50 years. He has focused on four areas: noninvasive evaluation of the heart, including physical examination; diseases of the atria; diseases of the pericardium; and electrocardiography. His meticulous examination of all available data and his ability to synthesize the information has led him to become a world expert on the latter two of these topics. As such, he has been referred many difficult cases for second, third, and fourth opinions.

His curriculum vitae includes well over 400 articles, as well as numerous books, chapters, and abstracts. He has held many editorial positions and is an esteemed reviewer for many cardiovascular journals. In 1998, he received the Burger Award of the European Society of Noninvasive Cardiovascular Dynamics. In 2003, Dr. Spodick was awarded the Melvin L. Marcus Memorial Award for his distinguished contribution as a gifted teacher in cardiology by the International Academy of Cardiology at the 3rd World Congress of Heart Disease. His cardiovascular fellows have recognized him with teaching awards on an almost yearly basis.

David Spodick continues to be highly productive in the cardiovascular medical community through his work at St. Vincent Hospital, where he is Director Emeritus of the Cardiovascular Medicine Fellowship Program and at the University of Massachusetts Medical School where he is Professor of Medicine Emeritus.

LVH, Cornell criteria* and posterior directed loop on VCG. Possible left atrial enlargement as well from V_1

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Sobrecarga Ventricular izquierda por el criterio de Cornell y bucle QRS dirigido para atrás en el VCG. Posible SAI

Cornell criteria* [1]: Cornell index (CI) or criterion of Casale: CI = R aVL + SV_3 > than 28 mm in men or > 20 mm in women indicates LVH.
QRS voltage-duration product(Cornell Product)[2;3]: RaVL + SV3 with 6 mm added in women x QRS duration. Values ≥ 2440 mm/ms are diagnostic of LVH (Positive criteria of LVH CP≥2440 mm x ms). The Cornell product is a useful ECG marker, reflecting not only left ventricular mass but also LV geometry and diastolic function in Japanese hypertensive patients[4]. Reduction in Cor P ECG LVH during antihypertensive therapy is associated with fewer hospitalizations for HF, independent of blood pressure lowering, treatment method, and other risk factors for HF[5].

1. **Casale PN, et al. Electrocardiographic detection of left ventricular hypertrophy: development and prospective validation of improved criteria. J Am Coll Cardiol. 1985; 6: 572–580.**
2. **Molloy TJ, Okin PM, Devereux RB, et al. Electrocardiographic detection of left ventricular hypertrophy by the simple QRS voltage-duration product. *J Am Coll Cardiol*. 1992;20(5):1180-1186.**
3. **Okin PM, et al. Electrocardiographic identification of increased left ventricular mass by simple voltage-duration products. J Am Coll Cardiol. 1995; 25: 417–423.**
4. **Shirai T, et al.Evaluation of hypertensive cardiac abnormalities using the Cornell product.Circ J. 2007;71:731-735.**
5. **Okin PM, Devereux RB, Harris KE, Jern S, Kjeldsen SE, Julius S, Edelman JM, Dahlöf B; LIFE Study Investigators. Regression of electrocardiographic left ventricular hypertrophy is associated with less hospitalization for heart failure in hypertensive patients. Ann Intern Med. 2007 Sep 4;147(5):311-319.**

Dear Andres,

This ECG tracing looks a bit suspicious to me for the following:

1. The abnormal T wave morphology in V2 for a Asian male at his age.

2. Borderline QT interval 440-460 ms

3. Prolong QU interval 650 ms. The U wave is a bit too wide in V3-5 the reason QU is prolonged.

Did he use asthma inhaler prior to getting the ECG?

Kind regards,

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Querido Andrés:

Este ECG se muestra sospechoso para mi por lo siguiente:

1. **Morfologia anorma de T en V2 para un hombre asiático de esta edad.**
2. **El intervalo QT es limítrofe 440-460ms**
3. **Intervalo QT prolongado de 650ms . La onda U es un poco amplia en V3-V5 razón por la cual está prolongado el QU.**
4. **Usó drogas inhaladas antes de la realización de este ECG?**

Abrazo

Li

FINAL COMMENTARIES

ECG analysis: HR 68bpm; P axis: +43º; PR interval 192ms; QRSD 94ms, QRS axis: +90º; T axis: + 22º; QT: 429; QTc 451. Vertical heart.

Poor R wave progression in precordial leads: Possible causes[1]

1) Normal variant: Ex. Asthenic body build.
2) Technical problem: abnormally high placement of the right precordial electrodes
3) Myocardial infarction in anterior wall
4) LVH
5) Incomplete or complete LBBB
6) LAFB
7) Ventricular pre-excitation
8) Chronic Obstructive Lung Disease
9) Large posterior pericardial effussion
10) Dextrocardia
11) Corrected transposition of the great vessels
12) Right fascicular block.

Transitional zone in V_5: displaced to leftward. In normal adults the transitional zone usually is located between lead V_2 and V_4. When is displaced leftward and beyond lead V_5 clock wise rotation is present and is observed in older subject.

Prolonged QT interval. For men QT in seconds heart rate 67 Mean value: 365ms, lower limit: 324ms. Upper limit: 404ms[2]

- **Surawicz B, Knilans TK. Chou´s ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE. Adult and Pediatric Sixth Edition 2008.Chapter 8 pp 193-194.**
- **Sagie A, Larson MG, Goldberg RJ, et alAn improved method for adjusting the QT interval for heart rate (the Framingham Heart Study) Am J Cardiol. 1992 Sep 15;70(7):797-801.**

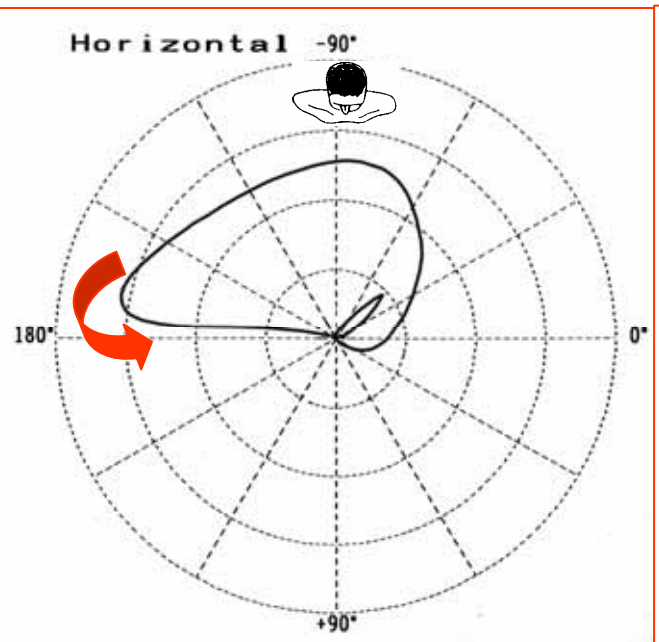
The horizontal plane shows VCG type C III or special right ventricular hypertrophy pattern. This is characterized by a QRS loop with initial vectors heading to the front and the leftward, preservation of counter clockwise rotation or rotation in eight, and ≥ 20% of QRS loop area located in the right posterior quadrant and posterior displacement: more than 70% of the area of the loop in posterior quadrants and ≥ 20% of in the right posterior one. In extreme cases, 100% of the QRS loop is in the right posterior quadrant. There is a marked posterior and right dislocation of the QRS loop.

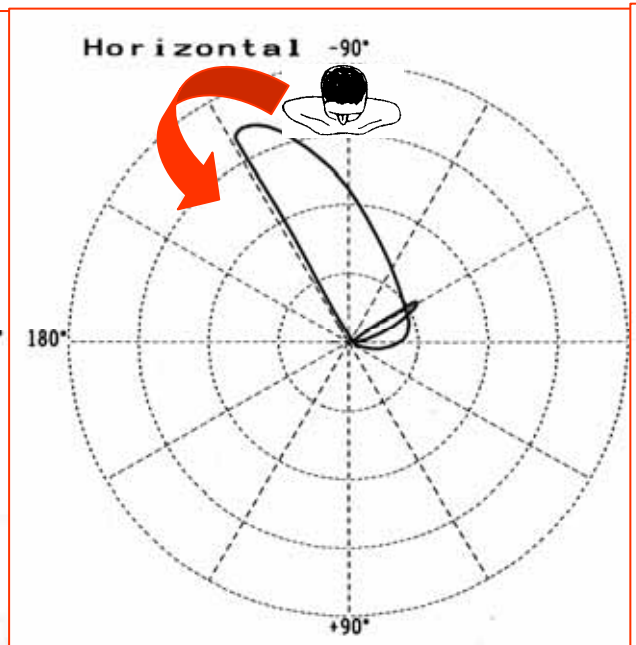
The QRS pattern on precordial leads shows QS or rS from V_1 to V_6. Eventually, QS pattern from V_1 to V_3 (pseudo anteroseptal infarction) by the relatively high position of the precordial electrodes in relation to the height of the heart as a consequence of diaphragm descent pushed by hyper-insufflated lungs.

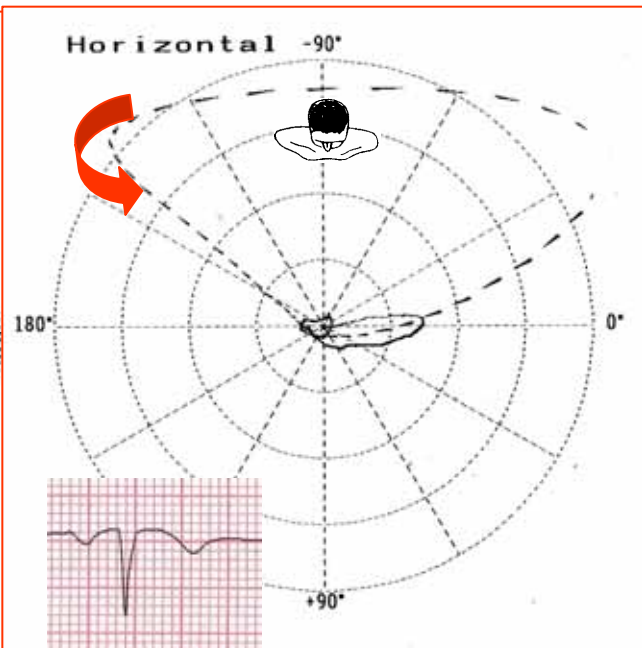
Tendency to low voltage in left precordial leads.

RVH VCG TYPE C, III OR SPECIAL IN HORIZONTAL PLANE

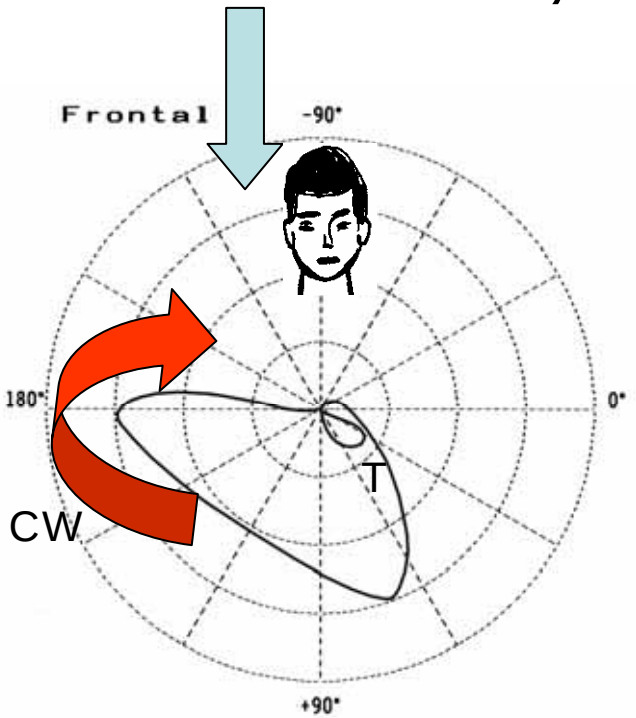
THE PRESENT CASE

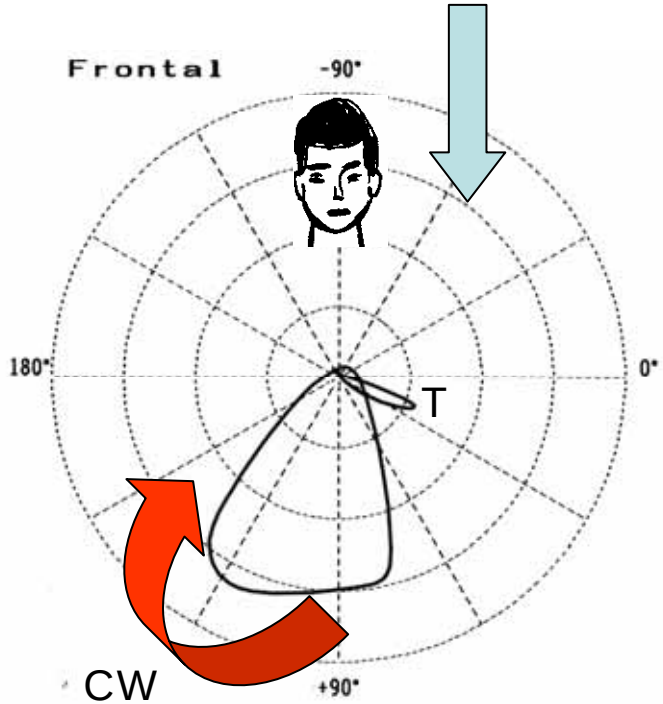




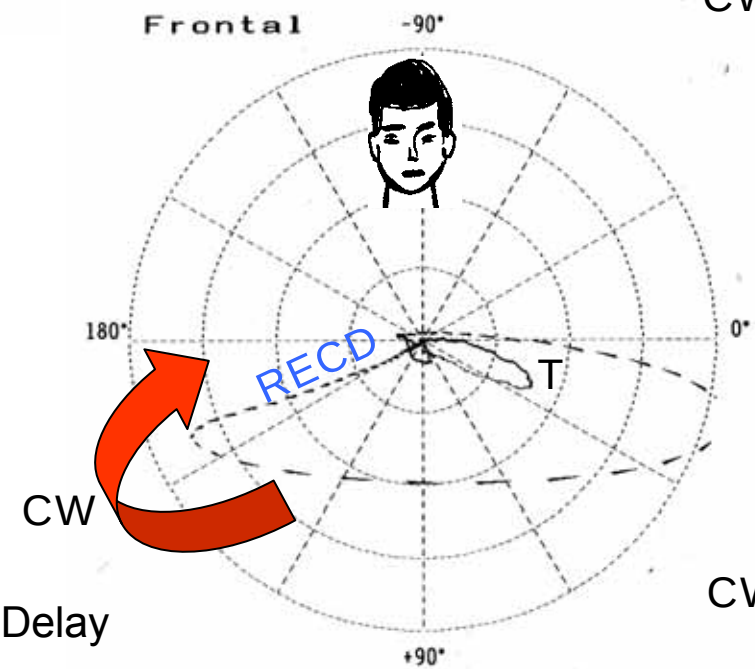


RVH VCG TYPE C, III OR “SPECIAL” IN FRONTAL PLANE





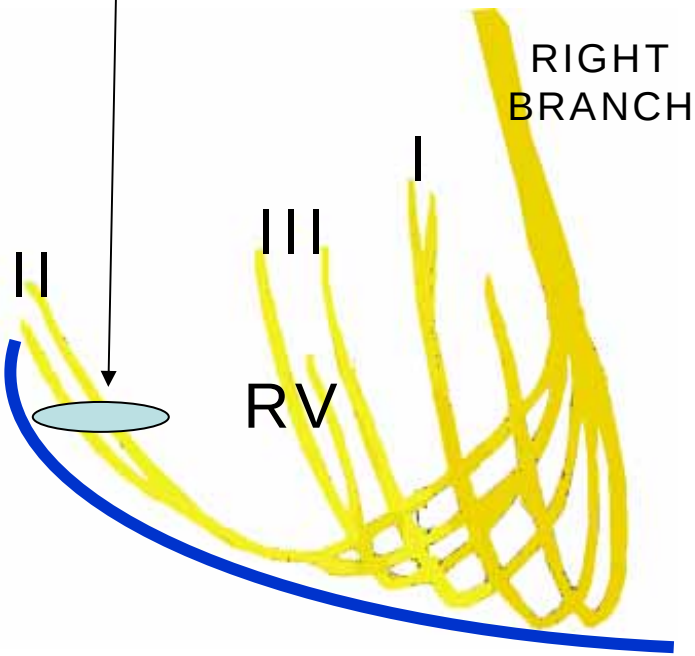
THE PRESENT CASE

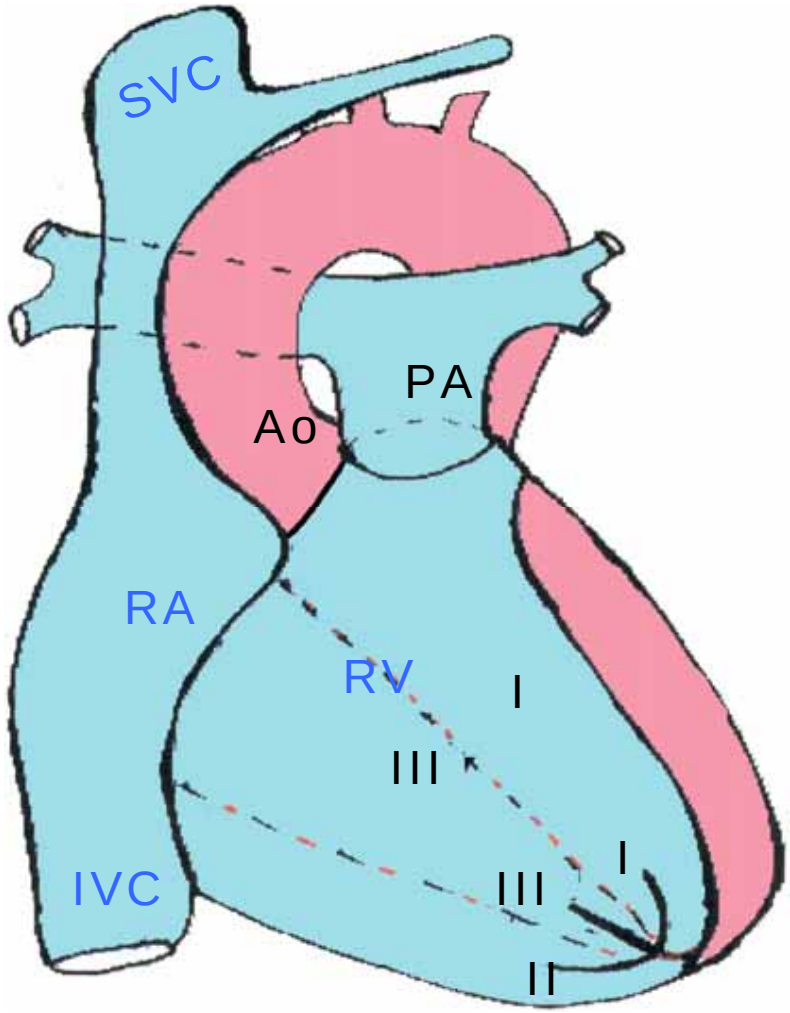


RECD: Right End Conduction Delay

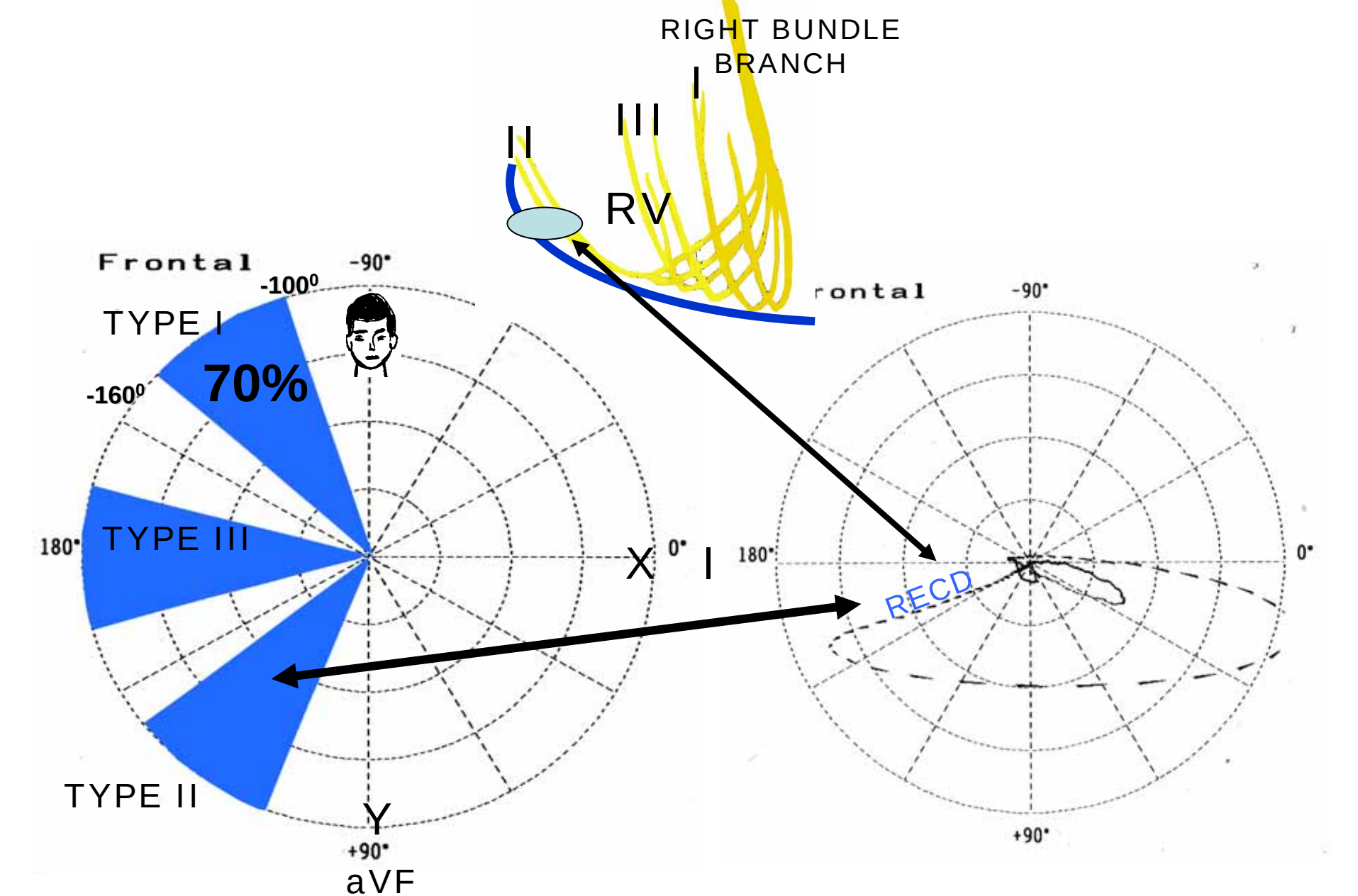
CW: Clock Wise Rotation

Another diagnosis is **inferior or posteroinferior fascicular right bundle branch block**





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



INFERIOR OR POSTEROINFERIOR FASCICULAR RIGHT BUNDLE BRANCH BLOCK

INFERIOR OR POSTEROINFERIOR FASCICULAR RIGHT BUNDLE BRANCH BLOCK

Characterized by presenting RECD located in the right inferior quadrant on FP in the territory of the inferior fascicle of the right branch. It corresponds to the territory of the right inferior fascicle (RIFB). The differential diagnosis occurs with left posterior fascicular block (LPFB) and Type C RVH. Many of the cases described in literature as LPFB are, the way we see it, RECD Type II, and since their electro-vectocardiographic differences are very subtle, the diagnosis must always be clinico-electrovectocardiographic.

A) ELECTROCARDIOGRAPHIC CRITERIA

- SÂQRS between + 70º and + 110º;
- Duration of normal QRS;
- SI RII RIII pattern, with RII and RIII of voltage not increased (usually ≤10 mm), never reaching 15 mm (essential element for the differential diagnosis with LPFB);
- RII ≥ RIII (in LPFB RIII > RII);
- aVR of the QS type;
- Possible notch in the descending ramp of inferior leads;
- S wave of V_2 and/or V_3 of increased depth;
- Persistent S wave until V_5 and/or V_6;
- V_1: rS, or QS, RS or rSR' with S of V_1 and V_2 possibly broadened.

B) VECTOCARDIOGRAPHIC CRITERIA

Right End conduction delay(RECD) in the three planes located to the right and below.

FRONTAL PLANE

Initial vectors always to the left, above and below;
Clockwise rotation;
Predominant location in the inferior quadrants;
Rapid change from left to right between 30ms and 50ms;
RECD to the right and below between +120º and +150º.

HORIZONTAL PLANE

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

RIGHT SAGITTAL PLANE

Initial vectors upward or downward;
Clockwise rotation;
Marked postero-inferior dislocation;
RECD downward and backward.

DIFFERENTIAL DIAGNOSIS BETWEEN RECD TYPE II AND LPFB

	RECD type II or INFERIOR OR POSTEROINFERIOR FASCICULAR RIGHT BUNDLE BRANCH BLOCK	LPFB
PR interval	**Normal**	**Frequent prolonged**
Association with inferior infarction:	**No**	**Frequent**
RII/RIII voltage ratio:	**RII >RIII.**	**RIII > RII.**
Notch in the descending ramp of R wave of inferior leads:	**Absent.**	**Constant middle-final notch.**
Intrinsicoid deflection in aVF, V5 and V6:	**Normal.**	**Increased: up to 30 ms.**
Intrinsicoid deflection in aVL:	**Normal.**	**Decreased: up to 15 ms.**
Aspect of QRS loop in the frontal plane:	**Clockwise and with characteristic rapid passage from left to right between 30 and 50 ms.**	**Clockwise, aspect of “fat” loop and maximal vector close to + 120°.**
Clinical factors that should be excluded:	**Type C RVH.**	**Vertical heart, RVE and lateral infarction.**