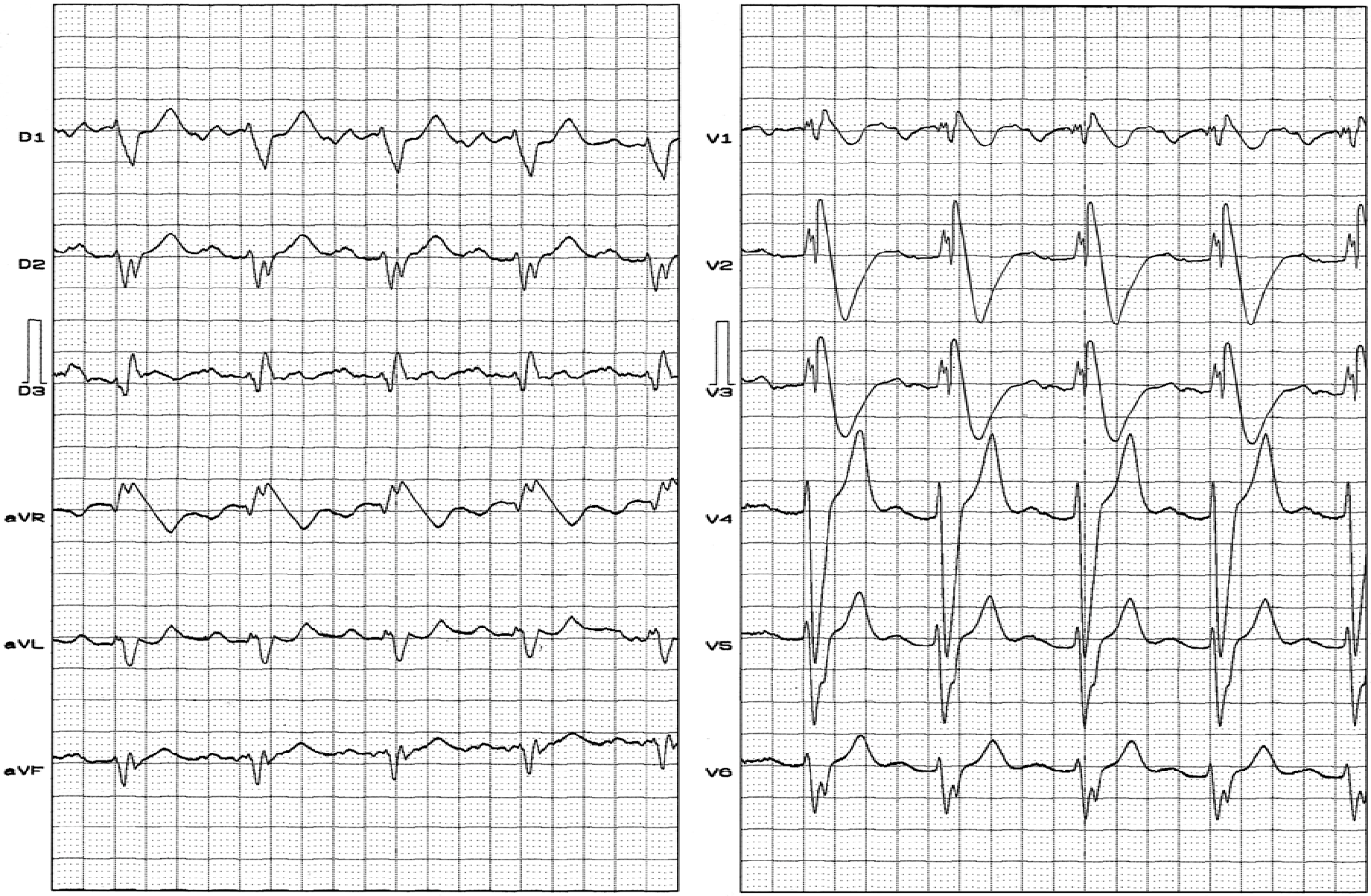
DIAGNOSTIC CHALLENGE:
DESAFIO DIAGNÓSTICO
A BIZARRE ELECTROCARDIOGRAM PREFORMED IN A MIDDLE-AGED MAN
UM ESTRANHO ECG REALIZADO NUM HOMEN DE MÉDIA IDADE

This ECG sent to me the Scottish physician Sir Arthur Conan Doyle.
He created the famous fictional detective Sherlock Holmes.

Sherlock Holmes

By Andrés Ricardo Pérez-Riera MD PhD

Name: PAQ**; Gender:** Male**; Age:** 41 yo**; Ethnic group:** White; **Weight:** 72 Kg**;**
Height: 1.78 m; **Biotype:** Aesthetic **Date:** 01/07/2008



What are the possible clinical and electrocardiographic diagnosis?

Dear Andres,You seem to get the most awful looking electrocardiograms to share with us. My observations include the following - in no particular order.

1. Sinus rhythm with 1st degree AV block
2. Bizarre frontal plane QRS axis in upper right-hand quadrant.
3. Very wide QRS axis approaching 200 ms with a RBBB morphology.
4. QT prolongation with weird STT morphology in right precordial leads.
5. Probable left atrial enlargement

Marked prolongation of all the waveforms looks like major electrolyte abnormalities with at least hyperkalemia and possibly some drug effects on the ECG. Perhaps this is a diabetic patient in ketoacidosis and accompanying electrolyte disturbance.

I will await the expert opinion of others.

Regards,

Frank

Estimado Andrés: Tu pareces tener los ECGs mas terribles para compartir con nosotros. Mis observaciones son las siguientes, sin un orden en particular:

Ritmo sinusal, probable sobrecarga auricular izquierda, bloqueo AV de primer grado, extraño extremo desvio del eje eléctrico del QRS para el cuadrante superior derecho, importante prolongación del intervalo QRS (QRSd ≈200ms), con patrón de BCRD, intervalo QT prolongado con elevación del ST/T en las precordiales derechas.

El diagnóstico etiológico podria ser severa hiperkalemia o posible efecto de drogas en virtud de el extremo aumento en la duración del QRS. Talvez, podria ser un diabético en cetoacidosis acompañado de desequilibrio electrolítico.

Esperaré por las opiniones de otros especialistas.

Yanowitz, Frank MD Professor of Medicine University of Utah School of Medicine Medical Director, ECG DepartmentLDS Hospital Salt Lake City, Utah 8th Ave. and C Street Salt Lake City, Utah 84143 USA

Hyperkalemia range of 7-8 meq/l vs drug overdose essencially. Na channel blockers like tricyclics. Cannot exclude myocarditis.

Hiperkalemia com niveles de potasio entre 7 y 8mEq/L versus overdosis de alguna droga. Ej: bloqueadores de canales de sodio como los antidepresivos tricíclicos.
No podemos excluir miocarditis.

Hiperpotassemia com níveis de potássio entre 7 e 8mEq/L versus over dose de alguma droga do tipo bloqueadores dos canais de sódio como os antidepressivos tricíclicos . Não se pode excluir miocardite.

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Dear Andres: If this patient were from Central/South American the first thing jumped into my mind is Chagas. People don't present this type of ECG for no reason. I'm 100% sure that he has a structural heart disease. From this ECG I see:
1. LAE; 2. CRBBB + LSFB + Partial LPFB and 1st degree LAFB.; 3. Fragmented QRS.; 4. ST-T morphology in aVR is typical to Brugada syndrome though the ST in V1-3 is not elevated. Nevertheless the T wave morphology in V1-3 is certainly suspicious (shall it be called Gussak T?). If he had syncope or evidence of VT/VF, BrS on top of Chagas or other type of structural heart disease is not impossible.
Thank you so much for showing with us this very interesting bizarre ECG tracing, and I look forward to hearing the final Dx.
Li

Prezado Andrés: Se este paciente for procedente da América Central ou do Sul o que primeiro vem na minha mente é a doença de Chagas. As pessoas não apresentam este tipo de traçado sem motivos. Estou 100% segura que ele é portador de alguma doença estrutural do coração.
No ECG eu observo: 1) Sobrecarga atrial esquerda 2) BCRD+BDAM+ BDPI parcial e primeiro grado de BDASE. 3) QRS fragmentado 4) ST/T com padrão em aVR típico da síndrome de Brugada mais o ST de V1 a V3 não está elevado. Porém, a onda T de V1 a V3 é suspeita de Gussak T? Se ele apresenta síncope o evidencia de VT/VF não é impossível que seja portador de doença de Chagas ou outro tipo de doença estrutural.
Obrigado por nos mostrar este muito interessante estranho traçado e esperamos ouvir o diagnóstico final.

Zhang, Li MD ldlzhang@gmail.com Director, Cardiovascular Outcomes Research
Main Line Health Heart CenterLankenau HospitalAssociate ProfessorLankenau Institute for Medical Research558 MOB East100 Lancaster AvenueWynnewood, PA 19096U.S.A.Tel: 610-645-2694Cell: 484-222-1876

Dear Andrès,

Without the symptoms and the family history it may be difficult to give an accurate diagnosis.

1) A myocardial disease with intraventricular conduction disorders (RBBB) with a clear separation of the depolarization wave fronts between the left and the right ventricle. The prolonged PR interval and the abnormal P wave wave (bifid) also favor a myocardial disease. I would help to see the parents ECG and the echocardiography.

2) A channelopathy cannot be rule out. J wave elevation syndrome with the well known but rare Brugada syndrome. I will investigate SCN5 A mutation.

3) An atypical form of RBBB.

4) A congenital inter-ventricular septal defect (Interventricular communication).

5) ARVD is also suggested but not as a first bet.

Holter monitoring and excercise stress test (AV block) will be welcome.

Thank you for sending us these ECG

Kind regards.

Prof Philippe Chevalier from Lyon France **philippe.chevalier@chu-lyon.fr**

Prezado Andrés: Sem os sintomas e sem a história familiar resulta difícil dar um diagnóstico preciso. 1) Cardiomiopatias com distúrbio de condução intraventricular(RBBB) e nítida separação das ondas de despolarização do VE e VD. A onda P bífida e o PR prolongado também estão a favor de cardiomiopatia. Ajudaria ver os ECGs dos pais e o ECO. 2) Uma canalopatia não pode ser descartada Uma síndrome J pode indicar o conhecido mas rara síndrome de Brugada. Eu investigaria a mutação SCN5A. 3) Uma forma atípica de RBBB. 4) CIV congênita. 4) DAVD pode também ser sugerida mas não como primeira escolha. Realizaria um Holter, e TE pelo bloqueio AV. Obrigado por enviar-nos estes ECG.

Con respecto al ECG desafio diagnóstico de nuestro maestro Profesor Dr Andres Ricardo Perez Riera primero diria que este ECG parece ser de un anciano, a menos que sea un chagásico.
La onda P en V_1 es profunda sugeriendo una auricula izquierda ensanchada por presión diastolica final elevada del ventrículo izquierdo. La duración del QRS es de 160 ms y asociado a las S profundas de V4 sugierie que el ventrículo izquierdo esta crescido. Yo sospecho que la dilatacion cardiaca evolucionó a partir de una hipertrofia.
Este ECG es muy probable que sea un bloqueo trifascicular BRD + Hemibloqueo Posterior + Bloqueo de primer grado. No obstante III no se encaja en el patrón hemibloqueo posterior

Con respecto a la etiologia pienso que este paciente no es un coronario con isquemia miocárdica Si fuese un anciano pensaria en amiloidosis de los viejos o en sarcoidosis Si tiene mas de 70 años y no tuvo síncope: haria apenas control. Pero si lo tuvo Marcapasos
Si es menor de esta edad: indicaria MRI y estudio electrofisiologico. Si en este último estudio el intervalo H-V es normal y el A-H prolongado: indicaria apenas control.

Como dijo mi queridisimo amigo Profe Andres Ricardo que hasta de los errores mios se puede aprender algo. Espero no haberme equivocado en grande. Errar es humano, pero siempre meter la pata en los diagnósticos es inhumano.

Un fraternal abrazo Samuel Sclarovsky
Diagnóstico dIferencial: Bloqueo de rama derecha puro con bloqueo del nódulo superior del nódulo A-V en presencia de una miocardioptia hipertrófica y dilatada.

Hola mis saludos me impresiona como un síndrome de Brugada y bloqueo bifascular mas hipertrofia de ventrículo izquierdo esto en lo que respecta a la electrocardiografía.
Sólo algo cuando hizo el infarto a nivel de V1 V2 V3 se dejo efectuar un cateterismo u otro procedimiento como la ablación y por que esa ondonada tan pronunciada en estas derivadas cuantas veces ha sido hospitalizado y quién de acuerdo a la historia clínica hizo FV y LA TV, muerte súbita.
Gregorio Maslivar

RS / 74 x´ / 280 mseg / 160 mseg / 480 mseg /
TPS :
RS con BAV 1er gdo con **BARDHH** con morfología de **S1Q3 T3** y cambios de onda T invertidas asimétricas en derivaciones precordiales der (**V1 a V3**) con desnivel positivo del **ST**
e igualmente un **QT prolongado** , como se aprecia en los pacientes con Hipocalcemias severas
Debo pensar en : Brugada y DAVD , pero les falta algo mas .....

□ □ □ **Mauricio Rondón MD**
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" El que pregunta, tiene 5 min de ignorancia , el que no lo hace , es ignorante toda su vida"

Estimado Maestro Pérez Riera:
1. Bloqueo trifascicular 2. Causa probable miocarditis
Saludos
Martin Ibarrola

……

Estimados colegas Mi opinion : una duda ,la edad del pte ,segun Andrés tiene 41 años ,Dr Samuel interpreta ECG de pte añoso?

Diagnostico EGC : ritmo sinusal sobrecarga de auricula izquierda, sobrecarga auricular derecha bloqueo av de 1er grado eje electrico desviado a la derecha con giro horario en plano frontal y situado en cuadrante sup-izq por un bloqueo de rama derecha por distension du porcion moderator band por sobrecarga volumetrica de VD

Que causas pueden llevar a estas alteraciones electricas ? 1) hipertension pulmonar secundaria a HTVC por : valvulopatia mitral. disfuncion diastolica de VI, miocardiopatia restrictiva, etc 2) hipertension pulmonar por hiperflujo pulmonar por cardiopatia congenita con shunt I-D en el adulto; 3) hipertension arterial pulmonar arterial de larga evolucion (primaria o secundaria , Eisenmegher, parasitarias (bilarziozis,etc) ,EPOC ,EPIC (ENF INTERSTICIAL CRONICA, conectivopatias, etc)

No encuentro caracteristicas tipicas para pensar en un Brugada , DAVD ,

Con el aporte de datos semiologicos podria encuadrar con mayor precision el diagnostico ,para tomar conductas, que seria lo sensato y racional ,pero tratandose de ejercitar lo que aporta el ECG me parece interesante el planteo

Un abrazo

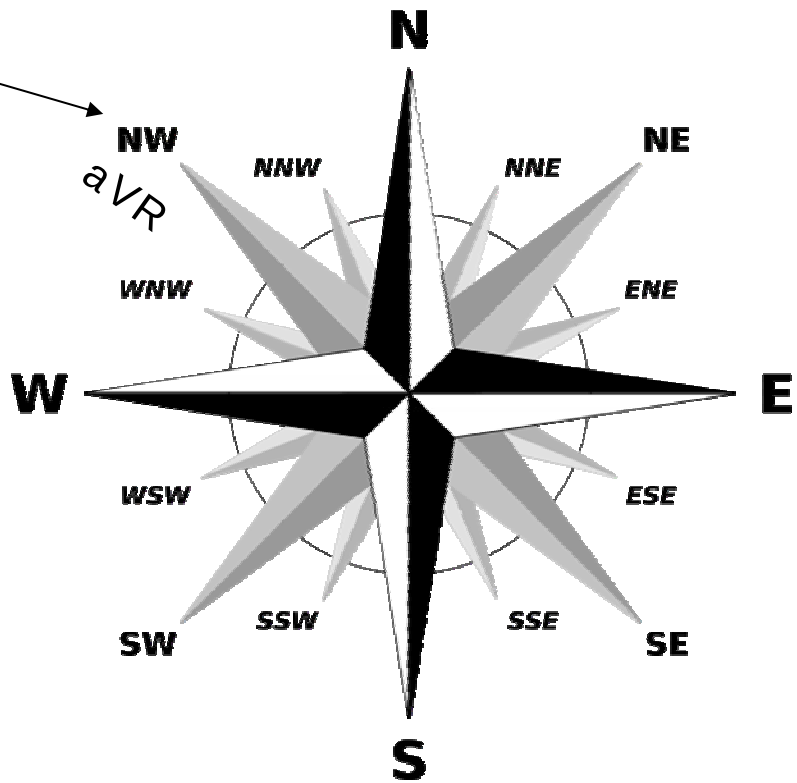
J Sirena

Hola :a simple vista diria....ritmo sinusal con fc de 65 a 70 lat min +BCRD+HBAI+bav de 1°g(bloqueo trifascicular) +QT largo -
Ahora me llama la atencion el fraccionamiento del QRS en precordiales izq,y tambien la morfologia del complejo ventricular en precordiales derechas rsrSR" que tambien esta en cara inferior...
DAVD?
Espero a los expertos
Dra. Maria Elina Ortega

Clinical diagnosis: Accidental plasma concentrations of Propafenone in the toxic range. Acute toxicity of propafenone in a case of suicidal attempt.

ECG diagnosis

P wave: Left atrial enlargement
PR interval: prolonged
QRS axis: extreme right QRS axis deviation on frontal plane. QRS axis near -160º because QRS complexes are only positive in aVR and minimally in III on frontal plane. QRS axis is located on North-west (NW) cardinal direction.



QRS duration: Near 200ms severe prolongation: Nonspecific Intraventricular Conduction disturbance: Pseudo-Complete RBBB. Marked abnormal ventricular activation pattern (bizarre): does not satisfy the criteria of either LBBB or RBBB.

In this particular case Vectorcardiogram could differentiate clearest among truly bundle branch block from pseudo-bundle branch block (our actual case). How? Answer: noting the location and distance between the dashes or tears:

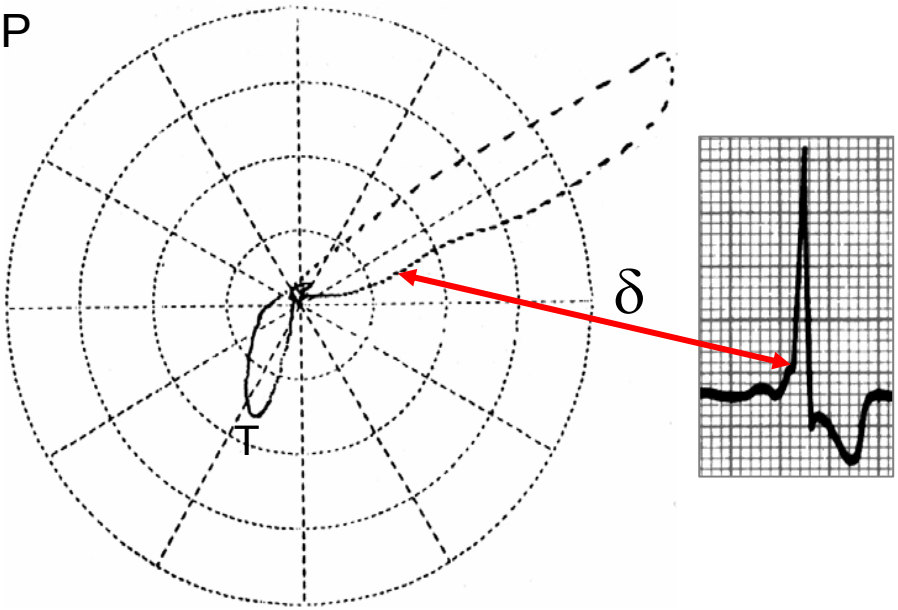
Separate dashes: = more dromotropism:

Very close dashes= less dormotropism

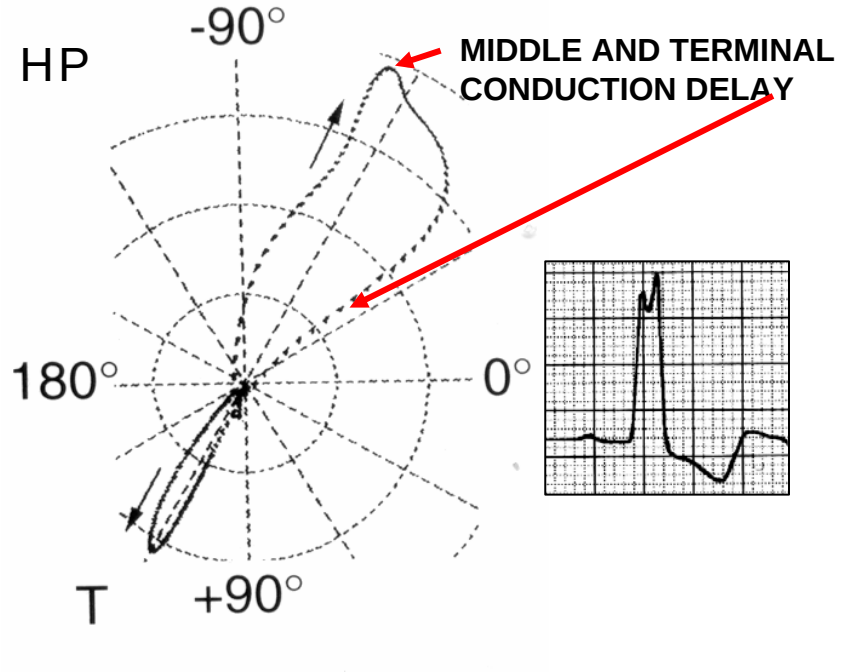
CONDUCTION DELAY INSIDE THE QRS LOOP

DELAY LOCATION: VERY CLOSE DASHES	
I) INITIAL CONDUCTION DELAY	Preexcitation, WPW syndrome/ delta wave on ECG.
II) MIDDLE AND TERMINAL (MIDDLE-END) CONDUCTION DELAY	Complete Left Bundle Branch Block (CLBBB)
III) RIGHT TERMINAL (END) CONDUCTION DELAY WITH GLOVE FINGER PATTERN ON RIGHT ANTERIOR QUADRANT ON Horizontal Plane	Truly Complete Right Bundle Branch Block (CRBBB)
IV) RIGHT TERMINAL (END) CONDUCTION DELAY ON RIGHT POSTERIOR QUADRANT ON Horizontal Plane	Truly Brugada syndrome and fascicular and/ or divisional blocks of RBB on free wall of right ventricle
V) UNIFORM CONDUCTION DELAY (OUR ACTUAL CASE)	Nonspecific intraventricular conduction disturbance hyperkalemia; drugs effect (quinidine, propafenone, antidepressant. etc); intra-infarction, intramural or non-specific block.

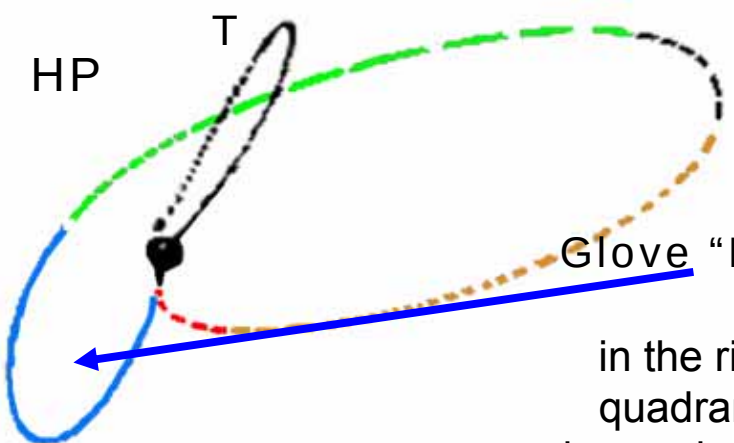
Preexcitation
WPW syndrome



Truly
Complete Left Bundle Branch Block



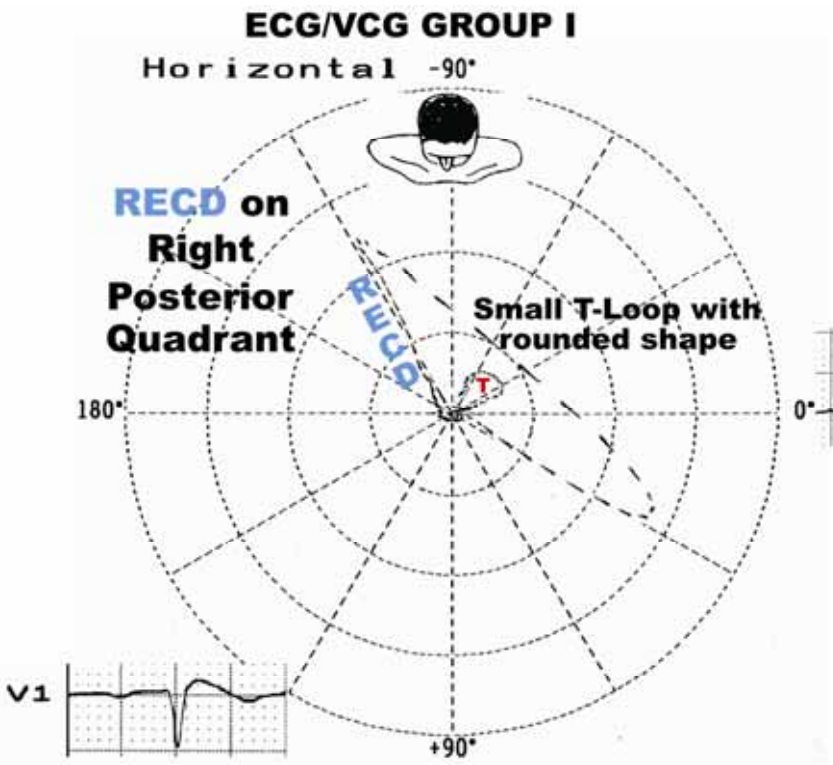
Truly
Complete Right Bundle Branch Block

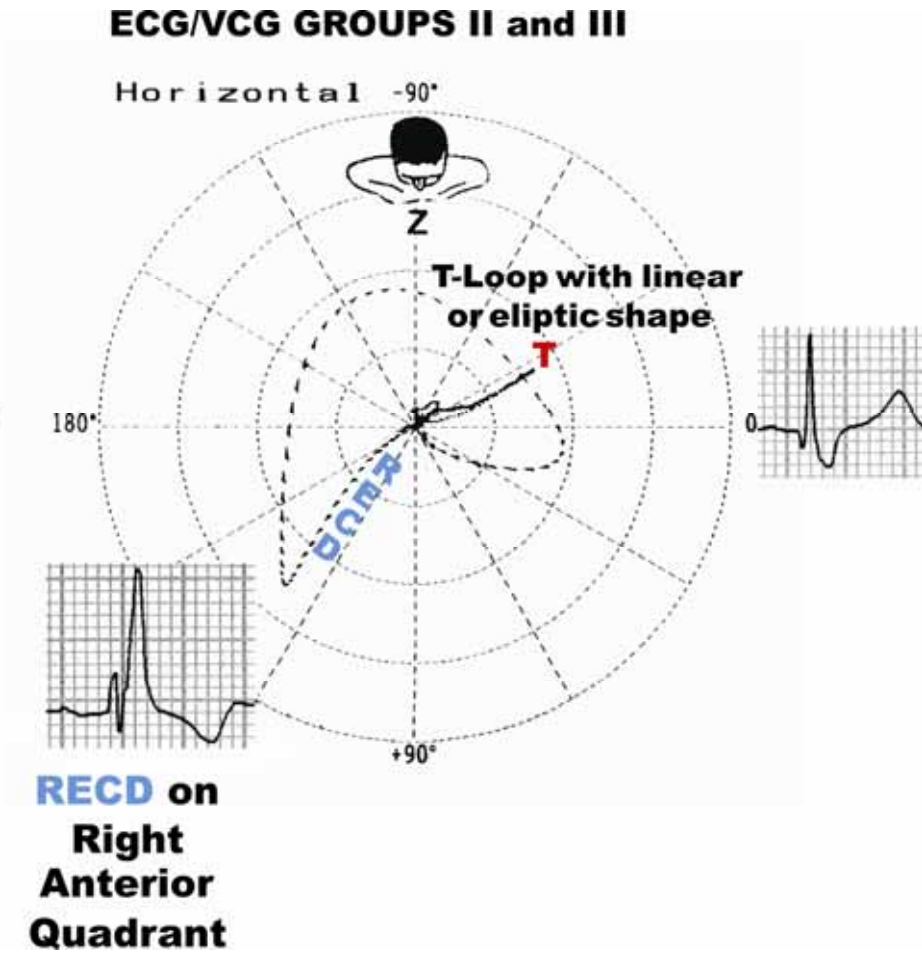


Glove "Finger-like"
Patter
in the right anterior
quadrant in the HP
and recorded slowly (RECD).

Brugada syndrome

Truly
Complete Right Bundle Branch Block





ST segment: ST segment elevation convex to the top from V1 to V3 and aVR lead(RVOT)

T wave: Negative T waves from V1 to V3

It is an example of Brugada-like electrocardiograpic pattern[1]" Brugada phenocopy[2;3] or acquired form of the Brugada syndrome[4].

We think that our denomination the appropiate because PHENOCOPY is an environmental condition that imitates (copies) one produced by a gene. **2.**The person who has an environmentally-produced condition that mimics one produced by a gene.

QT/QTc interval: Prolonged.

1. **Strimel WJ, Woodruff A, Cheung P, Kirmani BF, Stephen Huang SK. Brugada-like electrocardiographic pattern induced by lamotrigine toxicity. Clin Neuropharmacol. 2010 Sep-Oct; 33: 265-267**.
2. **Nguyen T, Smythe J, Baranchuk A. Rhabdomyoma of the interventricular septum presenting as a Brugada phenocopy. Cardiol Young. 2011 May 4:1-4**.
3. **Riera AR, Uchida AH, Schapachnik E, Dubner S, Filho CF, Ferreira C. Propofol infusion syndrome and Brugada syndrome electrocardiographic phenocopy. Cardiol J. 2010;17:130-135**.
4. **Shimizu W. Acquired forms of the Brugada syndrome. J Electrocardiol. 2005 Oct;38(4 Suppl):22-25.**

PROPAFENONE INTOXICATION: MAIN ECG FEATURES

1. PR interval prolongation secondary to augmentation of effective refractory periods of atrioventricular node (> AH interval), His-Purkinje system (> HV interval)
2. Prolongation of the effective refractory period of accessory pathways
3. QRS interval prolongation: in the toxic seric range widening of the QRS-complex
4. Marked abnormal ventricular activation pattern (bizarre): does not satisfy the criteria of either LBBB or RBBB
5. QT interval prolongation with normal JT interval
6. Eventual presence of the “memory phenomenon”: persistent T-wave changes[1].
7. Brugada-like type ECG pattern and extreme QRS complex widening[2;3]
8. Eventually mistaken as acute myocardial infarction[4;5]
9. Induced Brugada-type ECG, is a sign for imminent malignant arrhythmias[6].

1. **Wylie JV Jr, Zimetbaum P, Josephson ME, Shvilkin A. Cardiac memory induced by QRS widening due to propafenone toxicity. Pacing Clin Electrophysiol. 2007 Sep;30:1161-1164.**
2. **Hasdemir C, C, Olukman M, Ulucan C, Roden DM.. Brugada-type ECG pattern and extreme QRS complex widening with propafenone overdose. J Cardiovasc Electrophysiol. 2006; 17: 565-566.**
3. **Guevara-Valdivia ME, Iturralde Torres P, de Micheli A, Huarte Hernandez Y, Galvan L, Lizalde LC, Gonzalez-Hermosillo JA. Disclosure of "Brugada's syndrome" with intravenous propafenone Arch Cardiol Mex. 2002; 72:45-48.**
4. **] Jastrzebski M. Proarrhythmic effect of propafenone in patients with atrial fibrillation and atrial flutter Kardiol Pol. 2008 Nov;66:1221-1224)**
5. **Chutani S, Imran N, Grubb B, Kanjwal Y. Propafenone-induced Brugada-like ECG changes mistaken as acute myocardial infarction.. Emerg Med J. 2008;25:117-118.**
6. **Junttila MJ, Gonzalez M, Lizotte E, Benito B, Vernooy K, Sarkozy A, Huikuri HV, Brugada P, Brugada J, Brugada R. Induced Brugada-type electrocardiogram, a sign for imminent malignant arrhythmias.. Circulation. 2008;117:1890-1893.**

In Brugada syndrome patients the provocation test by Propafenone brought out recurrent spontaneous polymorphic VT and programmed ventricular stimulation test during the electrophysiologic study revealed both monomorphic and polymorphic VT[1].

Proarrhythmic effects are more frequent in patients with previous hepatopathy. Are described suddenly ventricular arrhythmias with the characteristics of a non-responsive electrical storm following the appearance of clinical symptoms of drug intoxication[2].

ELECTROCARDIOGRAPHIC MODIFICATIONS WITH ANTIARRHYTHMIC DRUGS

Toxicity of antiarrhythmic drugs

It suggests the following modifications:

1. **Different degrees of AV block.**
2. **QRS complexes broadening.**
3. **Prolongation of QT interval.**
4. **Torsade de Pointes.**
5. **Significant sinus bradycardia, sinus arrest or sinoatrial block.**

1. **Karaca M, Dinckal MH. Monomorphic and propafenone-induced polymorphic ventricular tachycardia in Brugada syndrome: a case report. Acta Cardiol. 2006;61:481-484.**
2. **Hrovatin E, Piazza R, Brieda M, Dametto E, Zardo F, Burelli C, Cassin M, Nicolosi GL. Proarrhythmic effects of propafenone in a woman with hepatopathy: is it always a simple drug in clinical practice?. Ital Heart J Suppl. 2002; 3: 770-775**.

Propafenone is an effective antiarrhythmic medication that is classified as a Class IC antiarrhythmic drug, although it also has some beta-blocking activity. Propafenone is primarily used for the maintenance of sinus rhythm in patients with a history of atrial fibrillation. While propafenone is approved for the treatment of life-threatening ventricular arrhythmias, its use for this indication is rare due to the potential for proarrhythmia in patients with structural heart disease. The major side effects of propafenone and their management will be presented here.

INTRODUCTION

Propafenone is an effective antiarrhythmic medication that is classified as a Class IC antiarrhythmic drug, although it also has some beta-blocking activity.

Propafenone is primarily used for the maintenance of sinus rhythm in patients with a history of atrial fibrillation. While propafenone is approved for the treatment of life-threatening ventricular arrhythmias, its use for this indication is rare due to the potential for proarrhythmia in patients with structural heart disease.

OVERVIEW

Approximately 15 to 20 percent of patients taking propafenone will have side effects that require drug discontinuation. The most common adverse reactions involve the gastrointestinal, central nervous, and cardiovascular systems. Most of these complications are dose-dependent, particularly central nervous system side effects such as dizziness, nausea, unusual taste, and blurred vision, which are often ameliorated if the propafenone dose is reduced. Potential side effects involving the cardiovascular system are most concerning, as the cardiac side effects are the most life-threatening.

The risk of cardiovascular toxicity is greater in patients with structural heart disease and in those treated for ventricular rather than supraventricular arrhythmias.

The relation between propafenone dosage, concentration, clinical response, and toxicity is complex. The main metabolic route of propafenone is via the cytochrome P450 2D6 isoenzyme. This metabolic pathway, known to have genetic polymorphisms, is functionally absent in about seven percent of whites and African Americans. In these "slow metabolizers," the extent of first-pass hepatic metabolism is much less than in "extensive metabolizers," and increased plasma concentration may be more likely to cause side effects. Moreover, because the oxidative elimination of propafenone is saturable, small dosage increases may cause a rapid and disproportionate increase in plasma concentrations, thus augmenting its potential for causing toxicity.

SIDE EFFECTS

Serious: **chest pain, fast or irregular heartbeat, fatigue, fever, malaise, shortness of breath, swelling of lower legs or feet.**

Common: **change in taste, constipation, dizziness, metallic or bitter taste vomiting or nausea.**

Less common: **blurred vision, diarrhea, dry mouth, fatigue, headache, skin rash**

Possible interactions

Other medicaments:

Propafenone may increase the effects of:

amitriptyline (Elavil, others) and perhaps other tricyclic antidepressants.

antihypertensive medicaments and cause excessive lowering of blood pressure.

beta blockers.

cyclosporine (Sandimmune), leading to toxicity.

quinidine (Quinaglute, various).

theophylline (Theo-Dur, others), leading to theophylline toxicity and seizures.

warfarin (Coumadin, others); more frequent INR tests and dose adjustments are needed.

Propafenone taken concurrently with:

amiodarone (Cordarone) may lead to excessive propafenone levels and toxicity; reduced doses may be required.

digoxin (Lanoxin, others) increases digoxin levels and can cause toxicity.

medicaments that inhibit or are removed by CYP 2D6 (talk to your doctor) may increase propafenone effects.

fluoxetine (Prozac) may lead to increased propafenone blood levels and toxicity if doses are not adjusted.

paroxetine (Paxil) may lead to increased propafenone blood levels and toxicity if doses are not adjusted.

quinidine (Quinaglute) may increase propafenone levels and lead to toxicity if doses are not adjusted.

ritonavir (Norvir) and perhaps other protease inhibitors may lead to propafenone toxicity.

sertraline (Zoloft) may lead to propafenone toxicity

The following medicaments may decrease the effects of propafenone:

carbamazepine (Tegretol).
phenobarbital (various).
rifampin (Rifadin, Rimactane).

Foods:

Ask your physician about the need for salt restriction.

Herbal medicines or minerals:

Kola, St. John's wort, ma huang and yohimbe may cause additive heart rate or rhythm problems. Using St. John's wort, ma huang, or kola while taking this medicine may result in unacceptable heart stimulation. Belladonna, henbane, scopolia, pheasant's eye extract or lily of the valley or squill powdered extracts should not be taken if you have abnormal heart rhythms.

Beverages:

Caffeine may have an effect on heart rate and may not be desirable. Talk to your doctor about caffeine.

Alcohol:

Alcohol can increase the blood-pressure-lowering effects of this medicament.

Tobacco smoking:

Nicotine may irritate the heart, reducing medicament effectiveness.

Occurrence of unrelated illness:

Vomiting, diarrhea or dehydration can affect this medicament's action adversely. Report such developments promptly.

Discontinuation:

Should not be stopped abruptly after long-term use.

POSSIBLE CAUSES OF BRUGADA ECG PHENOCOPIES

1) Early repolarization pattern or variant
2) Highly trained athletes Athlete heart
3) Influence of meals
4) Nocturnal vagotony
5) Sleep-disordered breathing
6) Partial Gastrectomy
7) Autonomic Dysfunction
8) Pectus excavatum
9) Left ventricular enlargement
10) Right Bundle branch block
11) Ischemic-induced ST-segment elevation:
    - Conus branch obstruction (RVOT irrigation)
    - Isolated right ventricular infarction
    - Vasospastic angina
    - Coronary artery anomaly
    - Primary percutaneous coronary intervention
12) Pulmonary embolism
13) Acute pericarditis
14) Hypercalcaemia
15) Hiperkalemia
16) Severe hyponatremia
17) Adrenal insufficiency

18) Thyrotoxic periodic paralysis
19) Effects of glucose-induced insulin secretion
20) Severe hypothermia
21) Fever
22) LQT3
23) Phenotypic overlap of cardiac sodium channelopathies: LQT3+ BrS
24) Non-hypothermic J wave in subarachnoid hemorrhage
25) Arrhythmogenic right ventricular cardiomyopathy/dysplasia
26) Chagasic cardiomyopathy
27) Rhabdomyoma of the interventricular septum
28) Drugs: see inside the fantastic Postema site:
http://www.brugadadrugs.org/avoid/
29) Mediastinal tumor (Compresion RVOT)
30) Haemopericardium (Compresion RVOT)
31) Anterior Mediastinal Infective Mass Lesion (Compresion RVOT)
32) Cardiac contusion
33) Complete left bundle-branch block due to right ventricular pacing.
34) Shoshin beriberi
35) Duchenne-Erb
36) Myotonic dystrophy type 2
37) Friederich ataxia.