Asymptomatic 19-year old male professional bicycle rider

Jovem assintomático 19-anos ciclista profissional do sexo masculino

Joven asintomático 19 años masculino ciclista profesional

Dear friend. I would like to ask for an expert opinion about this ECG.
Sport heart?
Echo needed?

Kind regards
Kjell Nikus M.D. from Tampere Finland

Portuguese
Caro amigo. Eu gostaria de pedir um parecer sobre este ECG.
Coração de atleta?
É necessário solicitar Ecocardiograma?

Saudações

Kjell Nikus M.D. from Tampere Finland

\Spanish

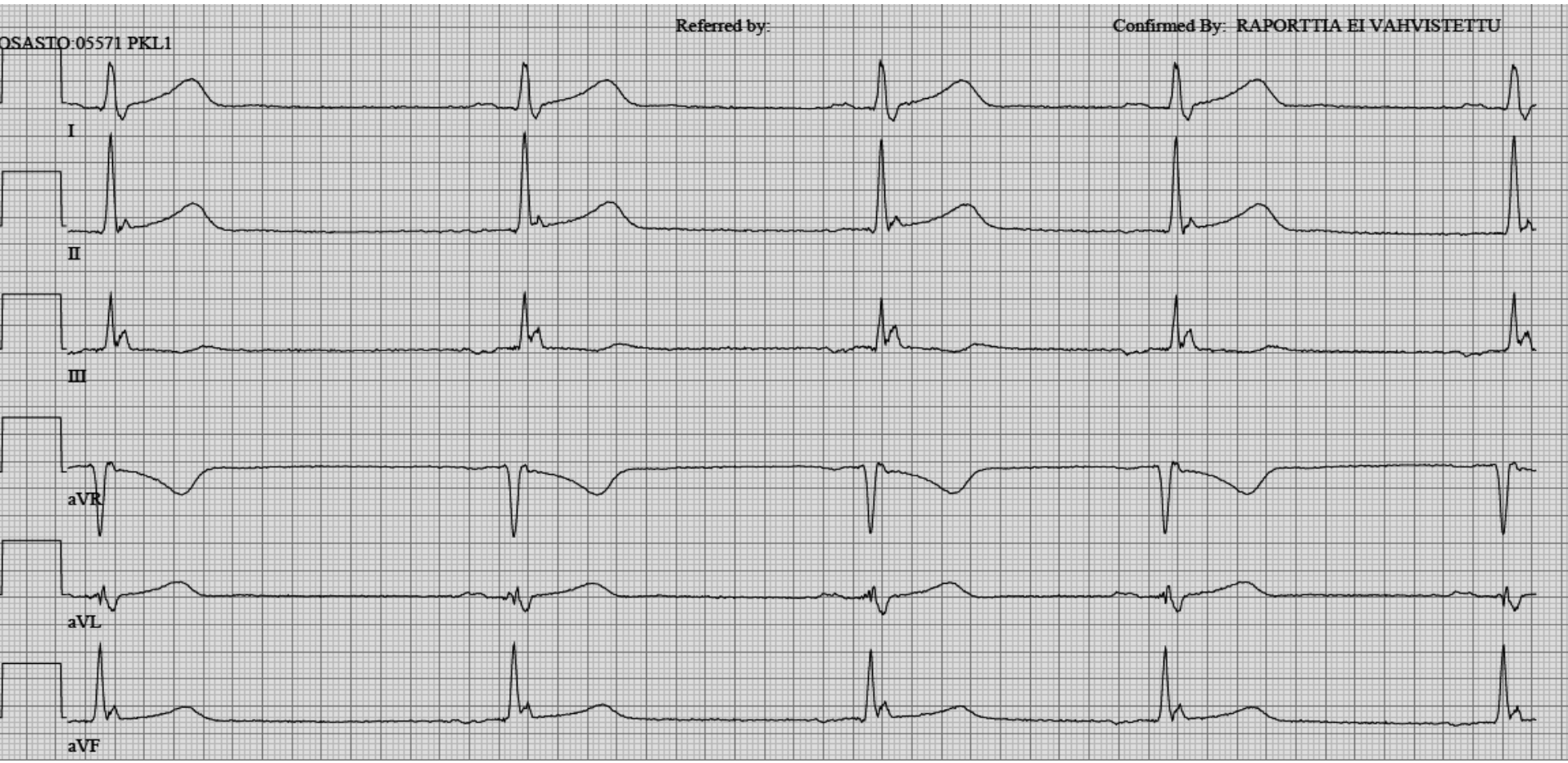
Querido amigo. Me gustaria saber la opinión de los expertos sobre este ECG
¿Considederan que sea un mero corazón de atleta?
¿Consideran necesario solicitar un Ecocardiograma?

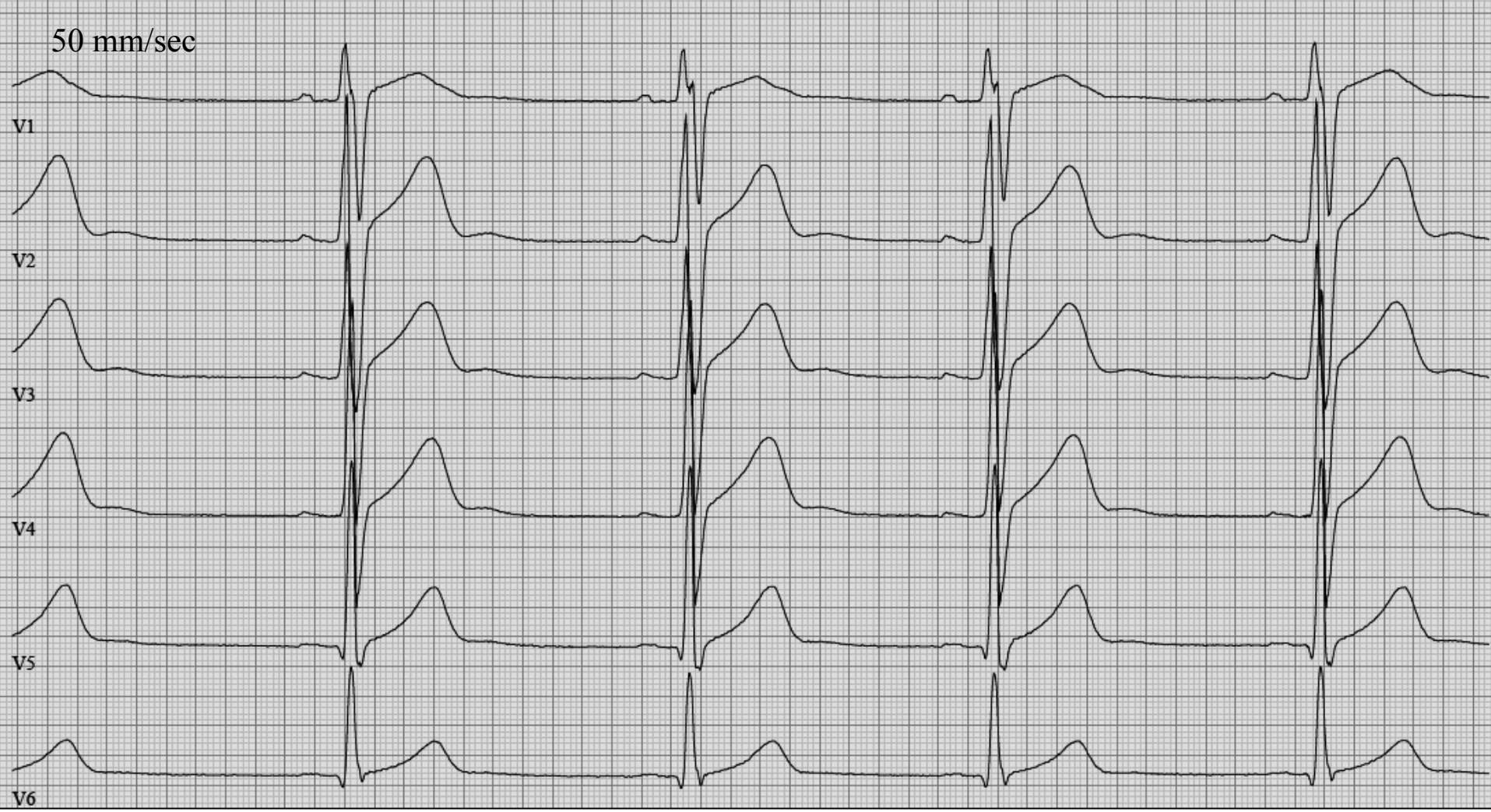
No symptoms. No family history
Routine ECG
No murmurs on auscultation
Two ECGs 1 month apart 50 mm/sec!

Asintomático. Sin historia familiar de relevancia.
ECG de rutina. Ausencia de soplos a la auscutación
Las 2 primeras diapositivas del registro fueron realizadas a doble velocidad

50 mm/sec

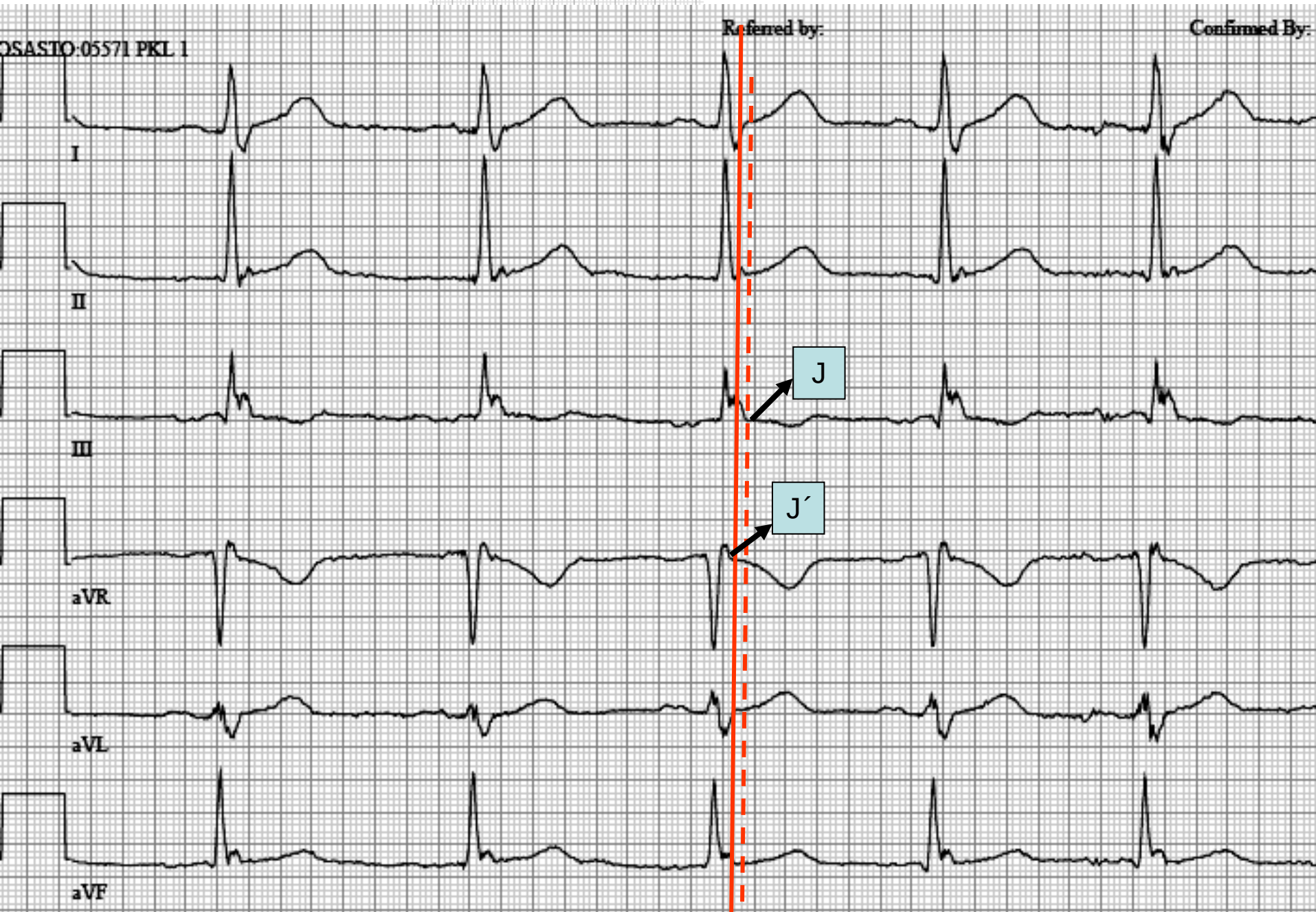
Vent. rate	52	BPM
PR interval	136	ms
QRS duration	112	ms
QT/QTc	432/401	ms
P-R-T axes	-20 66 31	

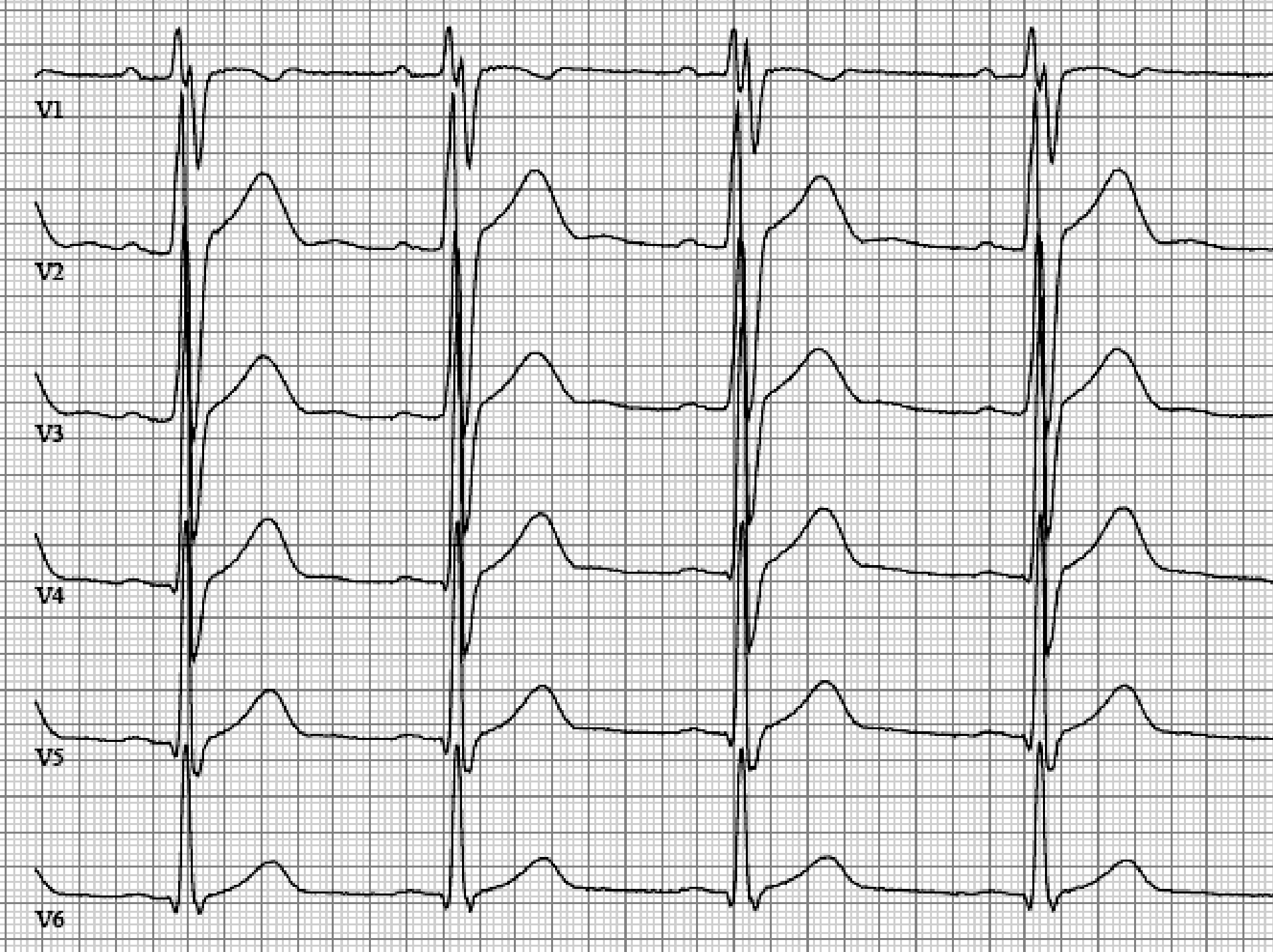



50 mm/sec
V1
V2
V3
V4
V5
V6

Vent. rate		81	BPM
PR interval		132	ms
QRS duration		114	ms
QT/QTc	392/455		ms
P-R-T axes	-4	61	26

Where is the real J wave? In J or J´point?




V1
V2
V3
V4
V5
V6

Colleagues opinions

Dear Prof. Andres Ricardo Perez Riera

My opinion to very interesting ECG send from Dr Kjell Nikus from Finland

1. this QRS pattern - QRS fragmentation in leads II, III, aVF and V1 should be suspected ASD typ 2
2. this pattern could be also similar to the controversial pattern of J wave in lead III, aVF, but this fragmentation seems to be delayed depolarization, pseudo J wave in leads III, aVF correspondes with S wave in lead I. Beside it the QRS fragmentation can be seen in lead aVL
3. there is positive T wave in lead V1 in the first ECG but T wave is negative in the second one - maybe other place of electrodes in position V1 in the first and in the second ECG.

All the best

Peter Kukla

My opinion to very interesting ECG send from Dr Kjell Nikus from Finland:
1. this QRS pattern - QRS fragmentation in leads II, III, aVF and V1 should be suspected ASD typ 2
2. this pattern could be also similar to the controversial pattern of J wave in lead III, aVF, but this fragmentation seems to be delayed depolarization, pseudo J wave in leads III, aVF correspondes with S wave in lead I. Beside it the QRS fragmentation can be seen in lead aVL
3. there is positive T wave in lead V1 in the first ECG but T wave is negative in the second one - maybe other place of electrodes in position V1 in the first and in the second ECG.

All the best

Peter Kukla M.D. Ph.D. from Poland

Envio mi opinion de este interesante ECG enviado por el Dr Kjell Nikus de Finland

1. Este patrón de QRS fragmentado en la pared inferior y V1 deberia hacer sospechar de CIA tipo ostium secundum.

2 Este patrón podria ser tambien similar al controvertido patrón de onda J en III y aVF, no obstante esta fragmentación parece ser despolarización atrasada, Osea pseudo onda J en III y aVF corresponde con la S de DI.

3. Existe onda T positiva em V1 en el primer ECG pero la T es negativa en el segundo ECG talvez pueda ser consecuencia de diferente colocación de los electrodos en V1 en cada trazado.

Dear Kukla Your ECG observation is very cleaver because in ostioum secundum atrial septal defect (OS-ASD) is frequent to observe a notch near the apex of the R wave of inferior leads: "Crochetag sign or crochetage pattern" (notch).(1) The sign correlates with severity.

The specificity of this sign for the diagnosis was remarkably high when present in all three inferior limb leads (≥ to 92%), even when comparison was limited to patients with an incomplete RBBB (≥ 95.2%). Early disappearance of this pattern was observed in 35.1% of the operated-on patients although the RBBB pattern persisted. A crochetage pattern of the R wave in inferior limb leads is frequent in patients with ASD, correlates with shunt severity and is independent of the RBBB pattern. Sensitivity and specificity of this sign are remarkably high when it is associated with an incomplete RBBB or present in all inferior limb leads.(2)

Unfortunately in Kjell Nikus description he wrote that there are no murmurs. In ASD the patient can have a hyperdynamic right ventricular impulse due to increased diastolic filling and large stroke volume. Palpable pulsation of the pulmonary artery and an ejection click can be detected because of a dilated pulmonary artery. S1 is typically split, and the second component may be increased in intensity, reflecting forceful RV contraction and delayed closure of the tricuspid leaflets. S2 is often widely split and fixed because of reduced respiratory variation due to delayed pulmonic valve closure (seen only if pulmonary artery pressure is normal and pulmonary vascular resistance is low). This characteristic abnormality is found in almost all patients with large left-to-right shunts. Blood flow across the ASD does not cause a murmur at the site of the shunt because no substantial pressure gradient exists between the atria. However, ASD with moderate-to-large left-to-right shunts result in increased RV stroke volume across the pulmonary outflow tract creating a crescendo-decrescendo systolic ejection murmur. This murmur is heard in the second intercostal space at the upper left sternal border.Patients with large left-to-right shunts often have a rumbling middiastolic murmur at the lower left sternal border because of increased flow across the tricuspid valve.Auscultatory findings of the ASD may resemble those of mild valvular or infundibular pulmonic stenosis and idiopathic dilatation of the pulmonary artery. These disorders all manifest as a systolic ejection murmur, but they differ from the ASD by movement of the S2 with respiration, a pulmonary ejection click, or the absence of a tricuspid flow murmur.

Andrés Ricardo Pérez-Riera

Spanish

Estimado Kukla Su observación es muy inteligente porque en la comunicación interauricular tipo ostium secundum (CIA-OS) en el ECG es frecuente observar una muesca cerca de la cúspide de la onda R derivaciones inferiores: "signo Crochetag o patrón crochetage" (1). El signo se correlaciona con la gravedad de la CIA.
La especificidad de este signo para el diagnóstico era muy alta cuando está presente en las tres derivaciones inferiores (≥ 92%), aun cuando se limita a pacientes con un bloqueo de rama derecha incompleto (≥ 95,2%). La desaparición temprana de este patrón se observó en el 35,1% de los pacientes operados en-aunque el patrón de BRD persistió. Un patrón crochetage de la onda R en las derivaciones inferiores se correlaciona con la gravedad y es independiente del patrón de BRD. La sensibilidad y especificidad de este signo son muy altas cuando se asocia con un bloqueo de rama derecha incompleto o cuando está presente en todas las derivaciones inferiores. (2) No obstante, en la descripción que hizo Kjell Nikus escribió que no hay soplos. En la CIA-OS el paciente puede tener un impulso hiperdinámico del ventrículo derecho debido a un llenado diastólico aumentado y volumen sistólico grande. Pulsación palpable de la arteria pulmonar y un clic de eyección puede ser escuchado a causa de una arteria pulmonar dilatada.
S1 está típicamente dividido, y el segundo componente puede ser incrementado en intensidad, lo que refleja la contracción RV enérgica y el cierre retardado de las valvas tricúspide. S2 es a menudo muy desdoblado fijamente debido debido a retraso en el cierre de la válvula pulmonar (aparece solamente si la presión arterial pulmonar es normal y la resistencia vascular pulmonar es baja). Esta anormalidad característica se encuentra en casi todos los pacientes con grandes CIAs cortocircuitos de izquierda a derecha. El flujo de sangre a través de la CIA no es la causa un soplo porque ningún gradiente de presión sustancial existe entre las aurículas. Sin embargo, la CIA, con moderada a grande de izquierda a derecha resultado derivaciones en mayor volumen tiempos RV a través de la arteria pulmonar la creación de un soplo sistólico de eyección crescendo-decrescendo. Este soplo se oye en el segundo espacio intercostal en la parte superior izquierda del esternón.Patientes con grandes shunts de izquierda a derecha a menudo tienen un soplo sordo en la parte inferior mesodiastólico en el borde esternal izquierdo, debido a aumento del flujo a través de la tricúspide.

Las manifestacioens auscultatorias de la CIA-OS pueden parecerse a los de la estenosis infundibular pulmonar idiopática y la dilatación de la arteria pulmonar. Todos estos trastornos se manifiestan por un soplo sistólico de eyección, pero se diferencian de la CIA-OS porque el segundo ruido se modifica con la respiración es decir no es fijamente desdoblado

1. Cohen JS, Patton DJ, Giuffre RM. The crochetage pattern in electrocardiograms of pediatric atrial septal defect patients.Can J Cardiol. 2000 Oct;16:1241-1247.
2. Heller J, Hagège AA, Besse B, Desnos M, Marie FN, Guerot C. "Crochetage" (notch) on R wave in inferior limb leads: a new independent electrocardiographic sign of atrial septal defect.J Am Coll Cardiol. 1996 Mar 15;27:877-882

Dear Prof. Dr Andres Riera
Thank you very much for your ultra-speed comments and responds to my "first-glance-opinion" on this ECG.
I agree that this patient has no cardiac murmur so in the clinical context of that case - ASD is almost "impossible" I would like to mention that my proposed opinion was only based on ECG findings.
By the way of this case (a pattern that could resemble the early repolarization) I would like to share with you my own opinion concerning the problem of the early repolarization.
Mainly there are too many confusions and misunderstandings with this problem !!!
Nowadays the term "the early repolarization " is used improperly...We cannot call everything what is "after -or-inside QRS" as the early repolarization. In my modest opinion most of this is.....delayed depolarization impressed on ECG as QRS notching or fragmentation of the downslope ramp of the QRS comples in the inferior or lateral leads.
Last days I have prepared from case publications what different authors called the "early repolarization" !!!!Please look at next slide !!!! I gathered 21 cases from last publications described as the Early repolarization or J wave.There is very very heterogenous ECG changes and I only some of them fit with the true early repolarization !I wonder of your opinion. Do you agree with my approach to this problem ?
Maybe we could together write a statement paper concerning the proper naming what the true early repolarization is.I think that this "new definition of early repolarization " proposed and used by Michel Haissaguerre put confusion in the ECG world.and the J wave is for me as a "Trojan horse". The classic definition was based mainly on "ST elevation", the new one is based on the presence of the J wave but the ST elevation is not mandatory.But what we consider as J wave .....could not always be the J wave.....the same could not always be the early repolarization at all
I will be waiting for your opinion on the ER !!!
All the best from Poland
Peter Kukla MD PhD **Dear colleagues the next 13 slides show Peter Kukla inerpretation**

Estimado Prof. Dr. Andrés Riera Muchas gracias por tus comentarios ultra-rápidos y por responder a mi "primer vistazo de opinión" Estoy de acuerdo que este paciente no tiene soplo cardíaco y en este contexto clínico el diagnóstico de CIA es casi "imposible“. Me gustaría mencionar que la opinión expuesta se basó únicamente en el ECG sin tener en cuenta los datos clínicos.

A propósito de este caso el patrón podría parecerse a la repolarización precoz. Me gustaría compartir con ustedes mi personal opinión sobre el tema de la repolarización precoz. Debo decir que pienso que existen demasiada confusión conceptual y malos entendidos con este tópico. Pienso que hoy en día el término "repolarización precoz" se utiliza mal. No podemos llamar a lo que ocurre dentro del complejo QRS, como la repolarización. En mi modesta opinión la mayor parte de losfemnómenos en el QRS son despolarización retardada que se traducen en el ECG, como muescas del QRS o la fragmentación de la rampa descendente de los complejos QRS en las derivaciones inferiores o laterales.

Los últimos días he preparado - a partir de publicaciones de la literatura casos lo que diversos autores - llamados de "repolarización precoz"!! Por favor, mire en mi archivo adjunto!! Reuní 21 casos de las publicaciones de los últimos que se describen como siendo repolarización precoz . Existen cambios electrocardiográficos extremamente heterogéneos y apenas algunos de ellos encaja en el concepto de repolarización precoz verdadera.

Me pregunto cual seria su opinión. ¿Está de acuerdo con mi enfoque a este problema?

Tal vez así podría escribir un manuscrito de la denominación correcta cuál es la verdadera de la repolarización precoz pensar que esta "nueva definición de la repolarización precoz" propuesto y utilizado por Michel Haissaguerre puso confusión en el mundo.y ECG la onda J es para mí como un "caballo de Troya". La definición clásica se basó principalmente en "la elevación del ST", el nuevo se basa en la presencia de una onda J, pero la elevación del segmento ST no es mandatoria. Mas lo que consideramos como una onda J no siempre seria ser la onda J el mismo no siempre podría ser la repolarización precoz en todos

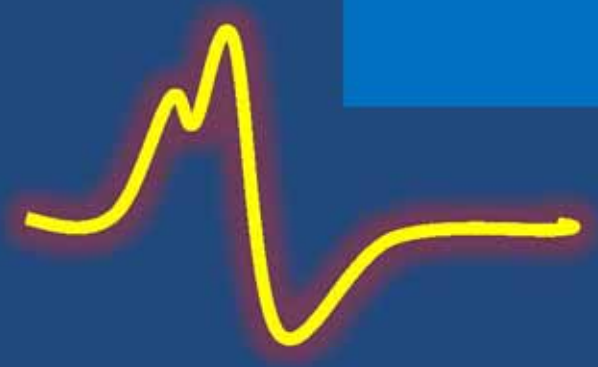
Voy a estar esperando su opinión en la sala de emergencias!

Todo lo mejor de Polonia

Confusions and misunderstandings terms

Early repolarisation syndrome

Early repolarisation



J wave

All 3 nowadays are used as the synonims !

Early repolarisation

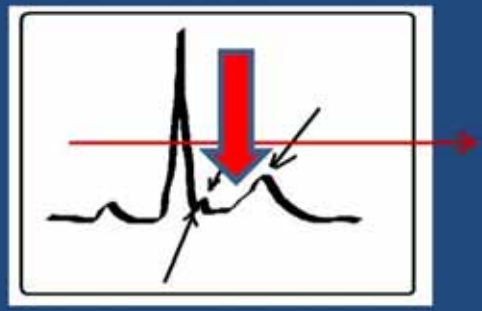
Classic definition by Wasserburger

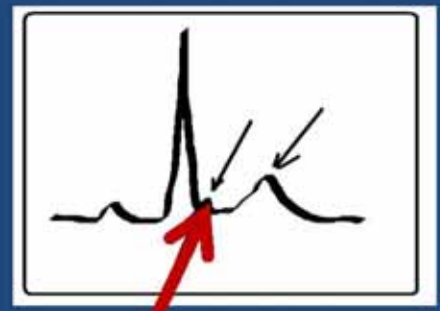
New definition by *Haissaguerre*

„Main Hero"

ST segment elevation

J wave





„Classic ER"

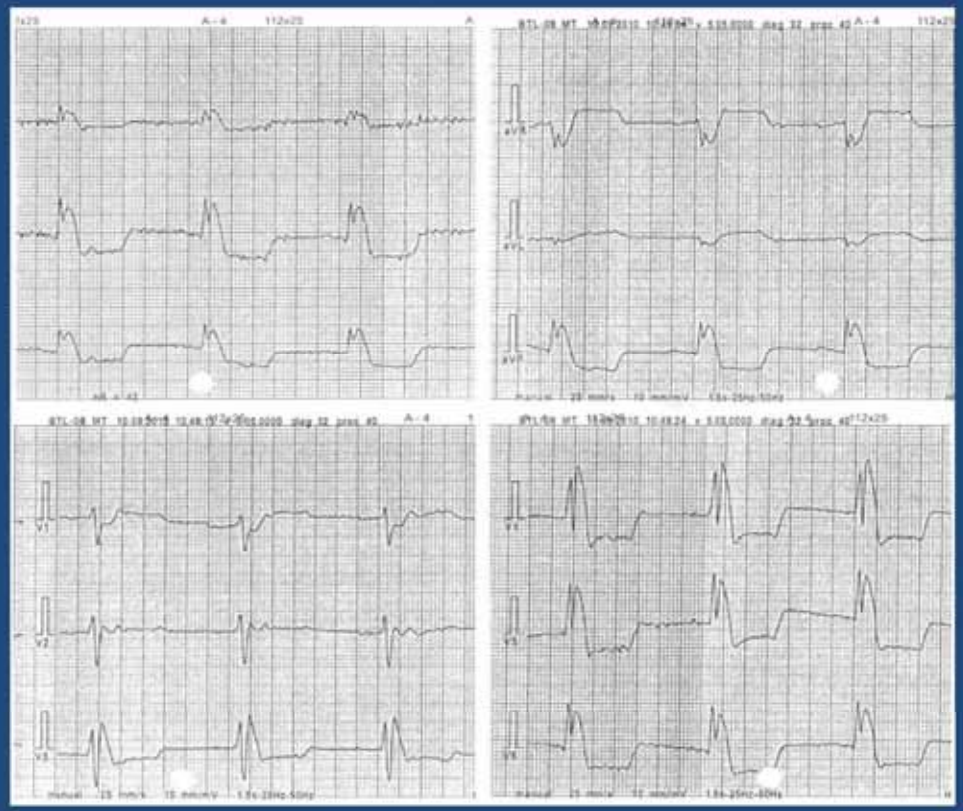
„New ER"

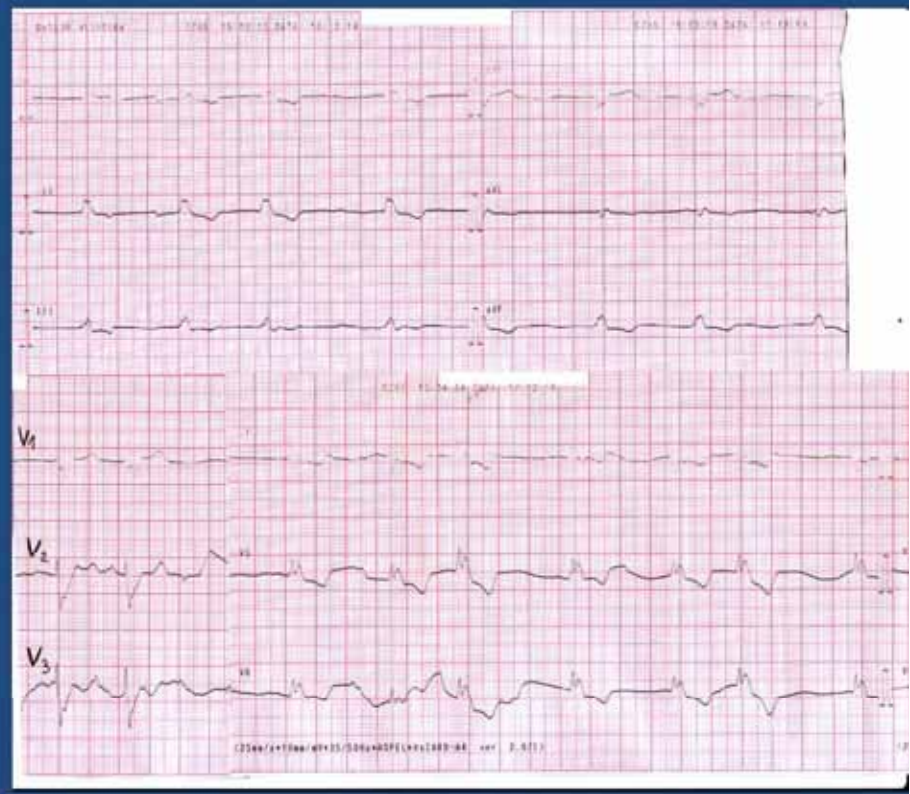
J wave – trojan horse

„Classic ER"

J wave in malignant hypothermia

ST depression but not ST elevation



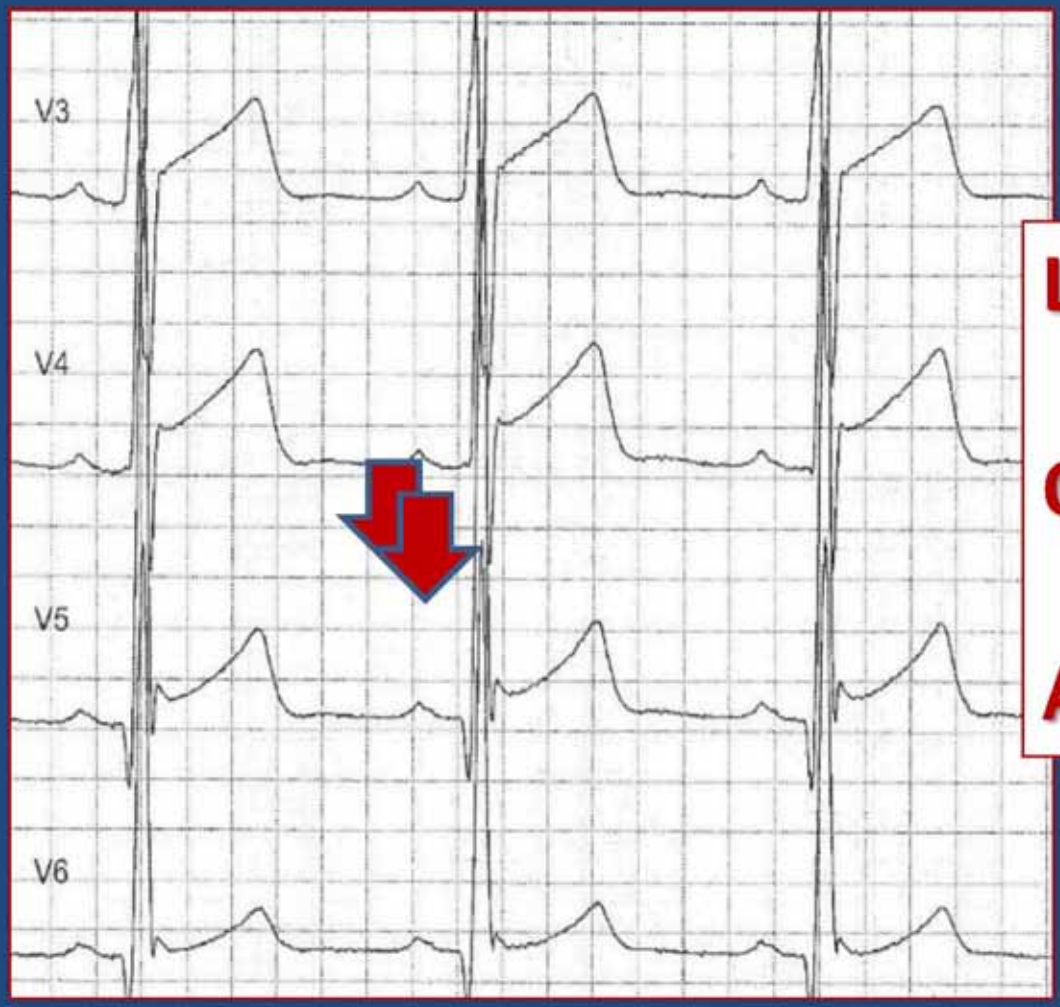


In many cases like these above malignant hypothermia with VF present J wave but with ST depression not elevation +except aVR + mimicking LBBB not ERS !!!

ER

- **ER by the new definition (and not only)**
- **can be face in additionally in all arhythmic disorders !**
- **e.g. HCM, ARVD/C, LQTS, and in STEMI……**
- **See next pictures…..**

ER in HCM



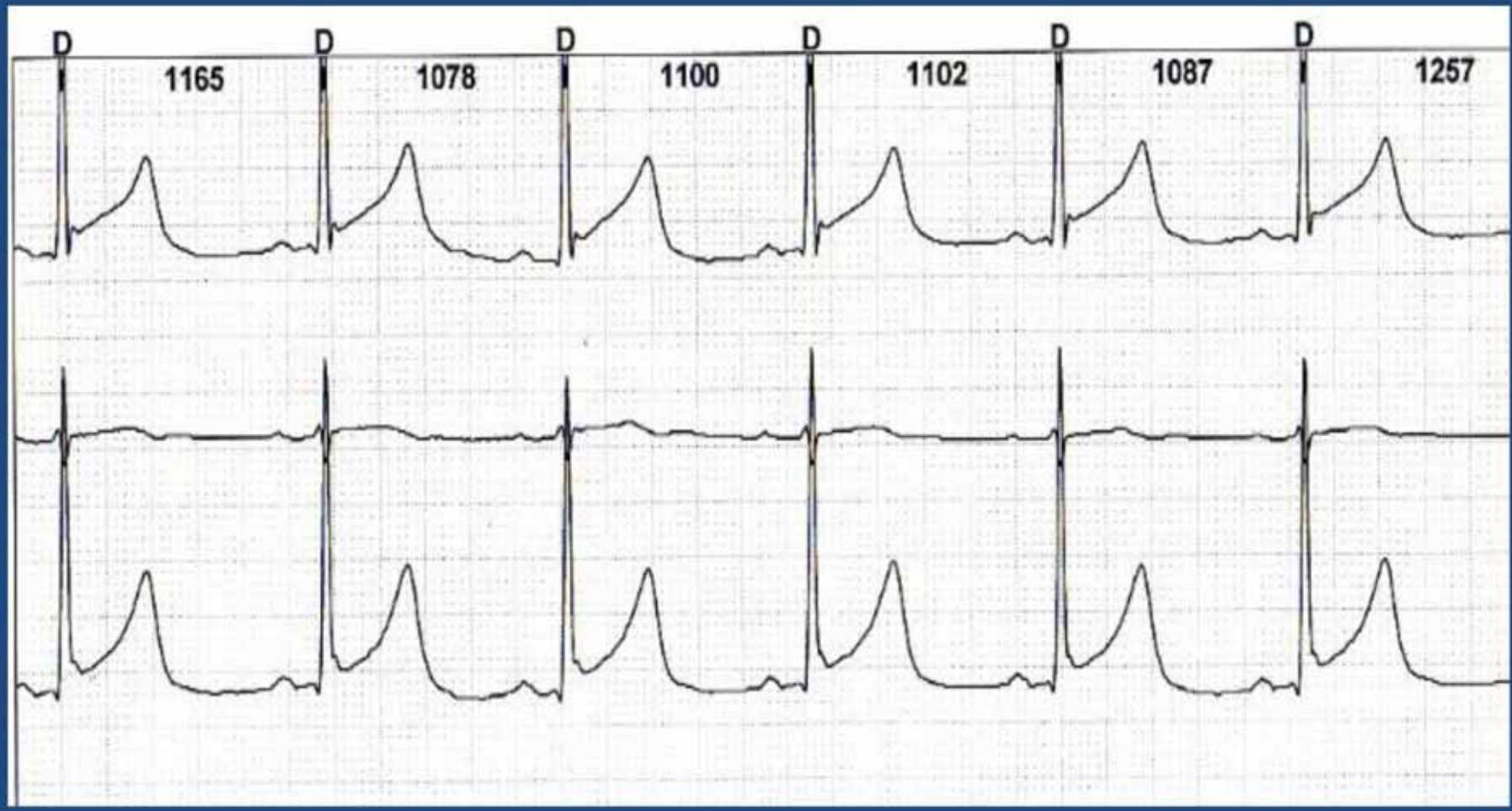
Classic ER

LVH

QTc „shorter”

Ascending ST

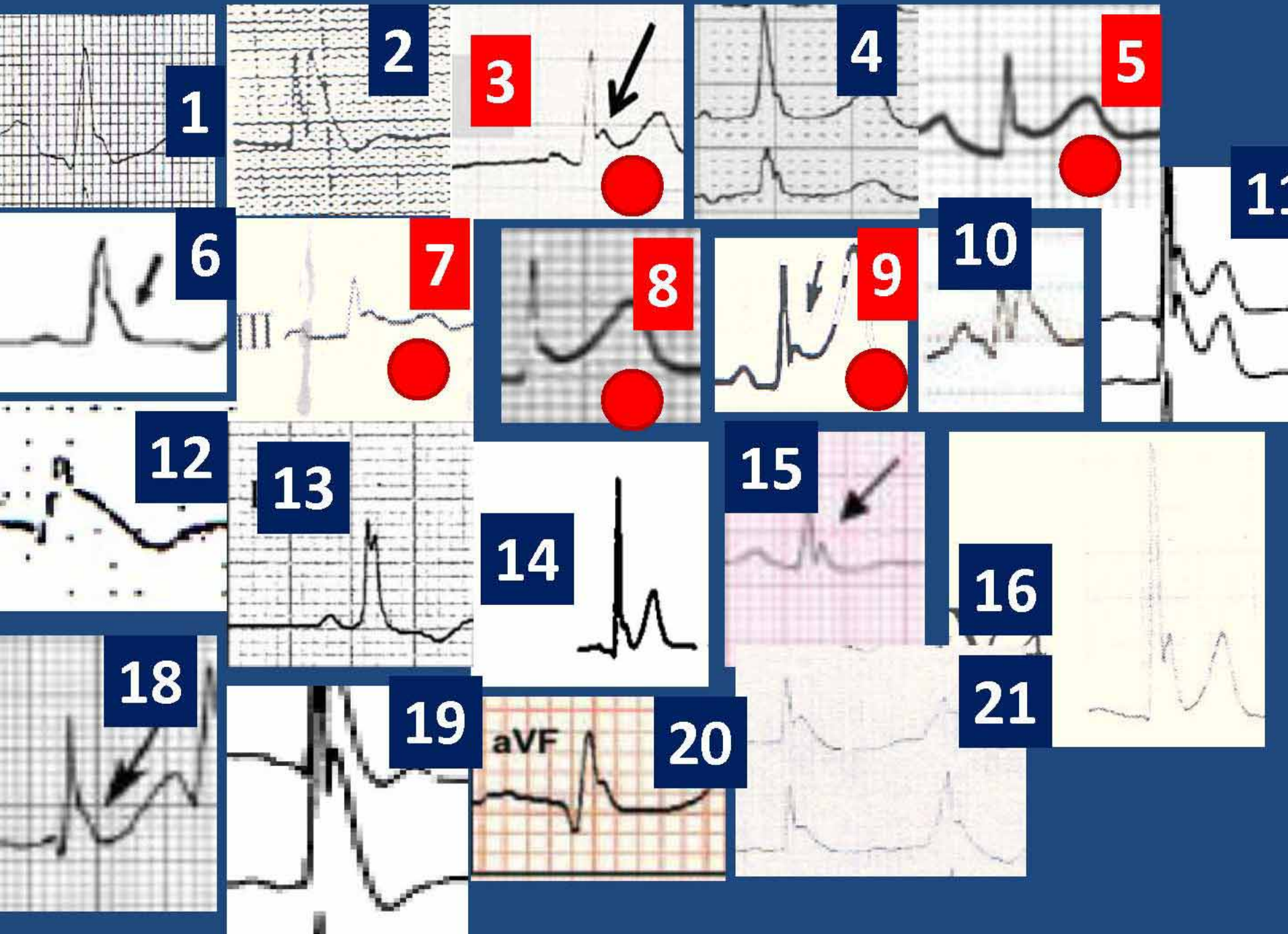
ER in LQTS

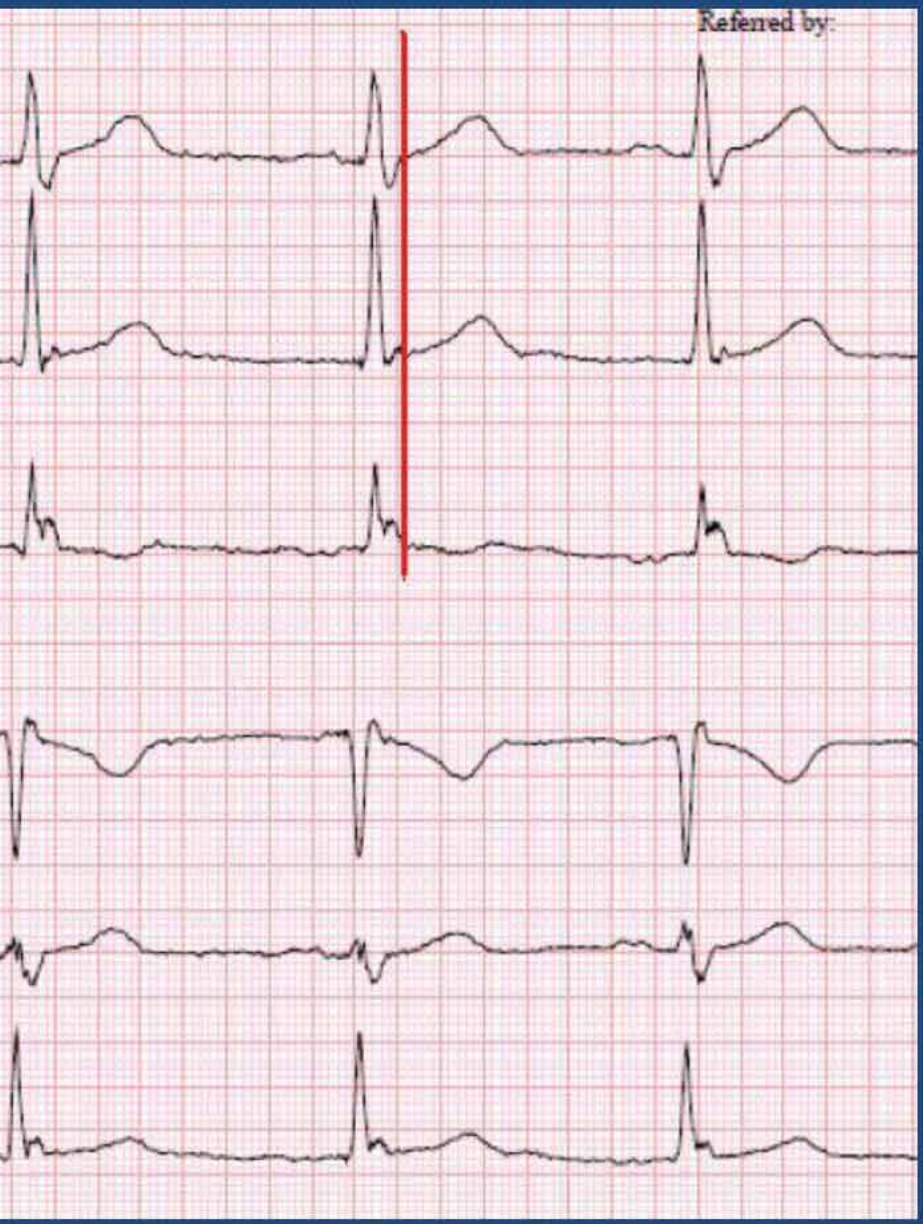


ER

- **Next slide pictures from the last 21 publications cases where all these cases were named as The early repolarisation in the context of clinical VF or SCD or syncope**

- **Let`s look at this –**
- **Really all of these cases we shpuld called ER ?**


1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
18
19
aVF
20
21



ER ? NOT for me

We should not use term J wave properly in this

J wave cannot beging before J point

This hump –ends here in lead III

Corresponds with end of

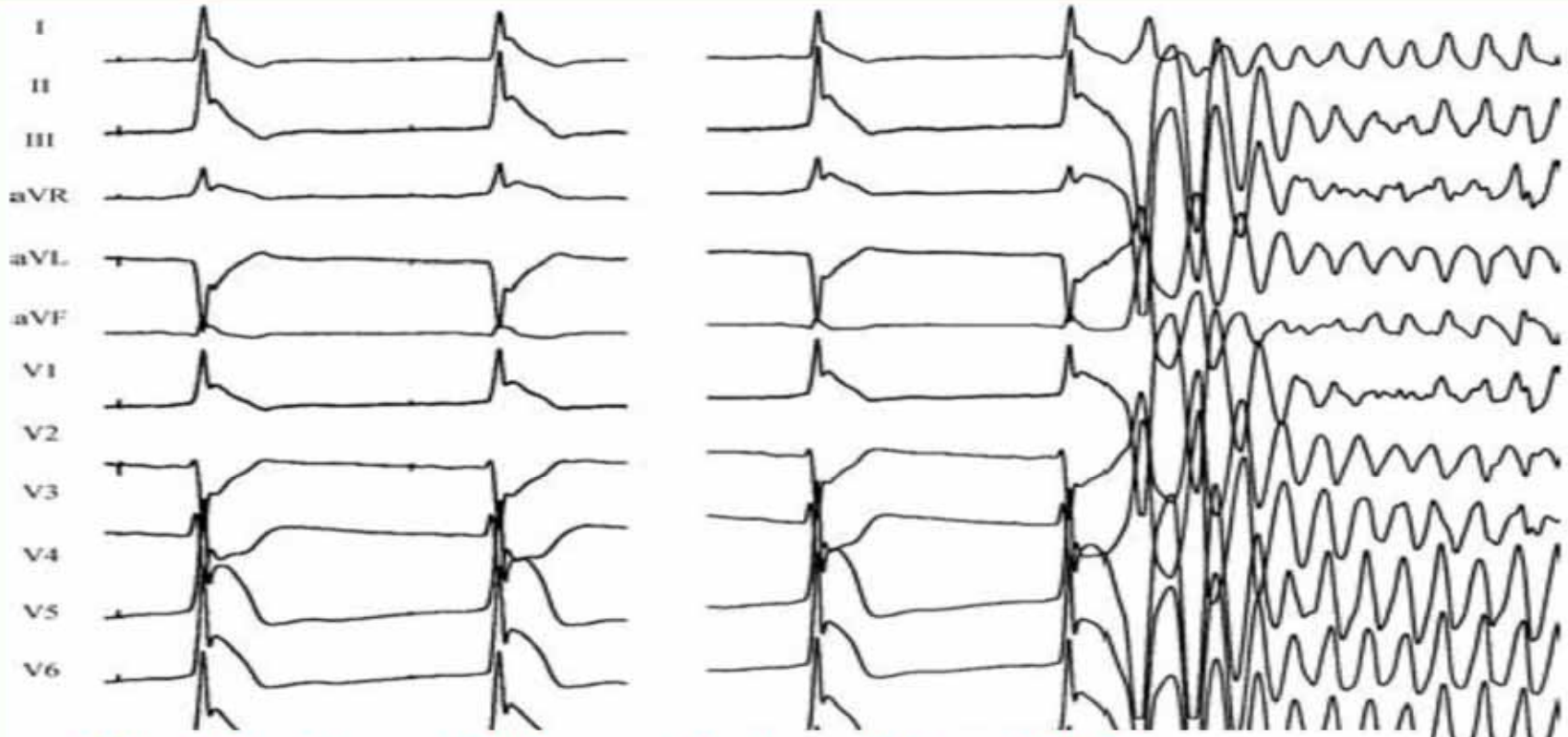
QRS – S wave end in lead I

For me this is depolarization abnormalities

And we should name it QRS fragmentation but not ER

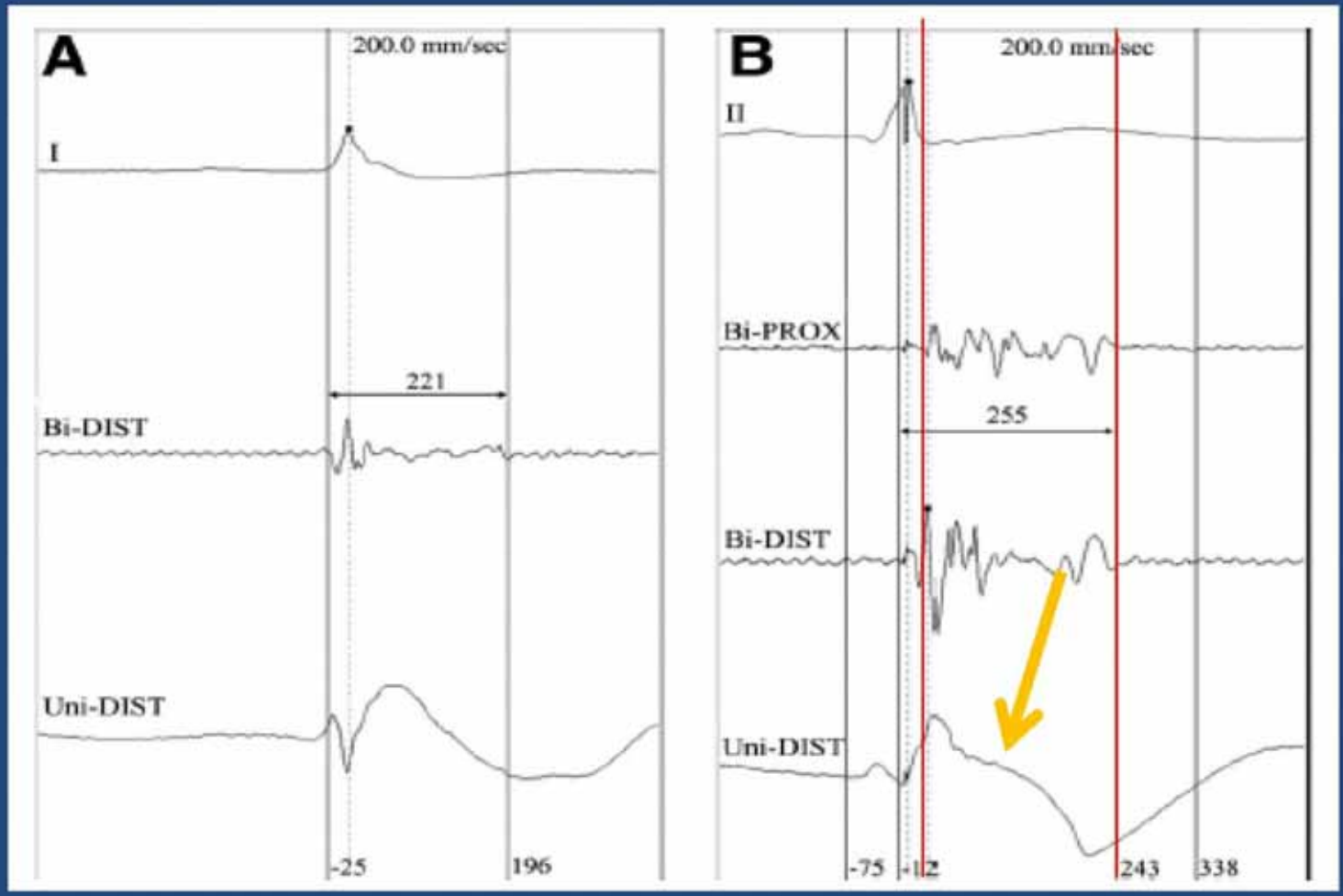
Ventricular Fibrillation with Prominent Early Repolarization Associated with a Rare Variant of KCNJ8/K_{ATP} Channel

MICHEL HAÏSSAGUERRE, M.D.,* STÉPHANIE CHATEL, M.S.,†,‡,§
FREDERIC SACHER, M.D.,* RUKSHEN WEERASOORIYA, M.D.,*



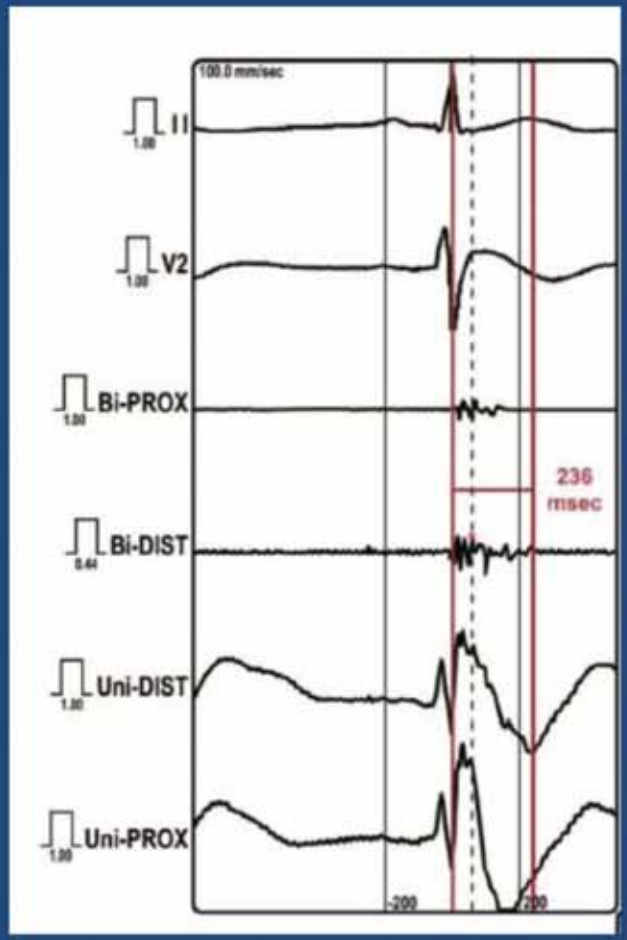
What this pictures of ER has got similiar with classic ER ?
Most of all it resembles ST before VF in patients with Prinzmetal Angina or ST shape after cardioversion !!!

Delayed depolarization
or
early Repolarisation

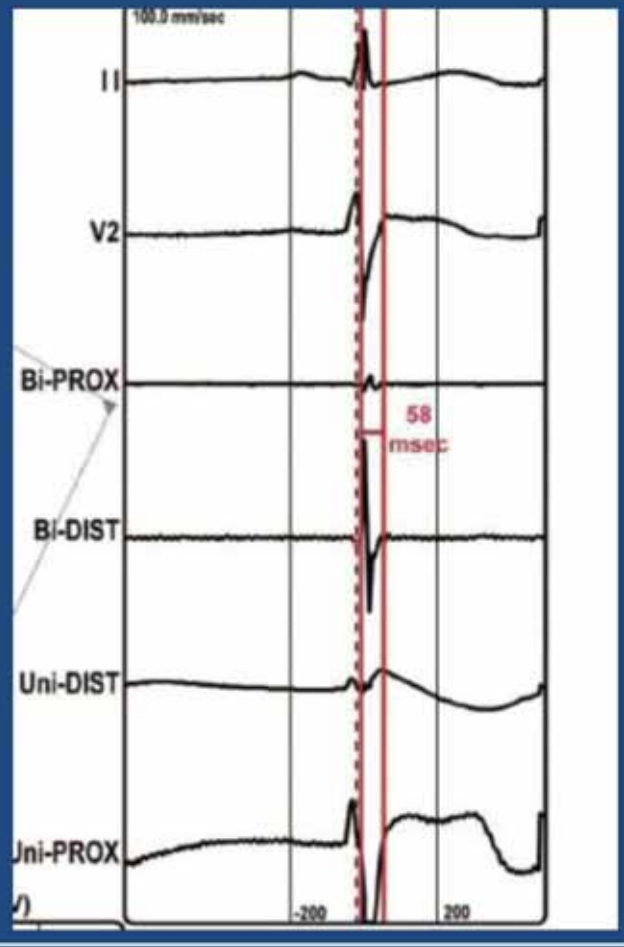


Nadamanee K.

delayed depo or early repo ?



RV epikardium



RV endokardium

Professor Melvin M Scheinman opinion

The ECG shows LVH as well as striking J waves in the inferior leads. Certainly would get Echo to exclude hypertrophic cardiomyopathy and get a detailed family history to exclude early repolarization syndrome.

El ECG muestra SVI como también impresionantes ondas J en las derivaciones de la pared inferior. Ciertamente yo realizaria un ECO para excluir cardiomiopatia hipertrófica y veria en detalle la historia familiar para excluir el sindrome de repolarización precóz.

Melvin M Scheinman,
Department of Cardiac Electrophysiology, University of California San Francisco, San Francisco, California, USA. scheinman@medicine.ucsf.edu
Professor of Medicine
Address:
UCSF
Electrophysiology Service
500 Parnassus Avenue
San Francisco, CA 94143-1354
Telephone/FAX/E-mail:
Phone: (415) 476-5706
Fax: (415) 476-6260

Professor **Arthur Wilde opinion**

Dear Andrés, first of all my compliments for your nice paper in Europace concerning the vectorcardiograms in BrS. Very nice paper, congratulations. Thank you also for mentioning my group at the end! As to this ECG I tend to localize the J-point at the J mark (that you have indicated). In the precordial leads the QRS width is increased and apparently that reflects in the inferior leads as a secondary bump. If imaging is normal (and if there is no family history for SCD) I would not do anything special
I wish very best

Arthur Wilde, MD, Ph.D. ***Academic Medical Center***
University of Amsterdam – Department of Cardiology
Professor, Division of Cardiology
Chair, Department of Clinical and Experimental Cardiology
Academic Medical Centre, Amsterdam/Interuniversity Cardiology Institute of the Netherlands, Utrecht, the Netherlands
Co-Director, Fondation Leducq for Sudden Cardiac Death

Estimado Andrés, en primer lugar mis felicitaciones por tu lindo manuscrito publicado en el Europace relativo a vectorcardiogramas en el sindrome de Brugada. Muy bonito trabajo, felicitaciones. Gracias también por mencionar mi grupo al final!
En cuanto a este ECG mi tendencia es localizar el punto J en la marca del punto J (como tu lo has indicado). En las derivaciones precordiales el ancho del QRS es mayor y aparentemente, se refleja en las derivaciones inferiores como una joroba secundária. Si imaginamos que es un joven normal (y si no hay historia familiar de SCD) yo no haría nada especial

Deseo lo mejor para ti

Arthur Wilde

Prof Bernard Belhassen opinion

It is unfortunate that the standard ECG leads are not synchronous (while the precordial leads are fine). This will not influence substantially my feeling about the present ECG. I do not see any "repolarization" problem but rather a "depolarization" one. The terminal notch visible in inferior leads is synchronous with the end of the QRS complex. I have not seen before. It should be the part of some intraventricular conduction disturbance. I do not see obvious RBBB nor any "late potential" suggesting epsilon wave in V1.

It is not actually not easy to give an opinion about sport clearance when you are faced in front such an atypical ECG. But I would probably be indulgent in the case where there is no family history of SCD and normal cardiac evaluation (including echocardiogram and ECG testing during maximal exercise).

Prof Bernard Belhassen

Tel-Aviv, Israel

Es lamentable que las derivaciones estándar del ECG no son sincrónicas (mientras que las derivaciones precordiales están muy bien). Esto no va a influir sustancialmente en mi sensibilidad sobre el presente del ECG. Yo no veo ningun problema de "repolarización", sino más bien de "despolarización". La muesca de terminal visible en derivaciones inferiores es síncrona con la parte final del complejo QRS. No he visto antes. Debe ser parte de algún trastorno de la conducción intraventricular. No veo BRD obvio ni ningún "potencial de tardio" que sugiera onda épsilon en V1.

En realidad no es fácil dar una opinión cuando nos enfrentamos ante estos ECGs atípico. No obstante probablemente sería indulgente en caso de ausencia antecedentes familiares de MSC y que la evaluación cardíaca sea normal (incluyendo un ecocardiograma y pruebas de esfuerzo con ejercicio máximo).

Profesor Bernard Belhassen

Tel-Aviv, Israel

Dear Andrés,

Interesting ECG sent by Dr. Kjell Nikus. I think it presents left ventricular hypertrophy with septal predominance. The changes in repolarization and the J point observed in the precordial and inferior leads may be considered usual in sportsmen and may not correspond to a pathological repolarization pattern implying a risk.

Dr. Kjell Nikus has asked if it would be indicated to perform an echo, taking into account this ECG and this history.

My answer is that I would perform an echo, not due to the electrocardiographic findings, but due to the currently existing evidence that echo should be made systematically along with personal and family history and ECG in all high competition sportsmen.

Echo is easily available, cheap and efficient, and it has proven to be additionally useful to show structural alterations and thus, it provides an additional contribution, helping to decrease the incidence of sudden cardiac death in high competition sportsmen. The method is not meant to exclude ECG. I completely agree with the thoughts by Dr. Michelle A. Grenier et al, presented in a poster in the American Society of Echocardiography this year(1).

Maybe you will disagree with my position, but I think it is necessary to review the evaluation guidelines for apparently healthy high performance athletes, in whom competitive sports practice enhances 6 times the risk of sudden cardiac death in comparison to the general population (in the general population the risk of SCD is 0.5% and in this group 3%). This means that high performance sports entails a risk in itself, so I think it is fully justified to perform an echocardiogram as a routine, besides a thorough clinical history, physical examination and ECG.

Best regards,

Martin Ibarrola

Estimado Andrés: interesante ECG ha enviado el Dr Kjell Nikus. El mismo me impresiona que presenta hipertrofia ventricular izquierda a predominio septal. Los cambios en la repolarización y punto J observados en las derivaciones precordiales e inferiores podrian considerarse como habituales en deportistas y no corresponder con patrón de repolarización patológicos que impliquen un riesgo.
El Dr Kjell Nikus ha preguntado si esta indicado realizar un eco teniendo en cuenta este ECG y esta história.
Mi respuesta es que yo le haría un ecocardiograma no por los hallazgos electrocadiográficos sino por la evidencia actualmente existente que el ecocardiogama deberia ser realizado en forma sistemática junto la história clinica personal y familiar y al ECG en todos los deportistas de alta competencia.
El ecocardiograma es un método accesible, de bajo costo y eficaz que ha demostrado ser de utilidad adicional por evidenciar alteraciones estructurales y asi brinda un aporte adicional contribuyendo a la disminución de la incidencia de muerte súbita en deportistas de alta competencia. El método no pretende ser excluyente del ECG. Comparto plenamente a lo presentado por el Dr Michelle A Grenier y colaboradores en un poster e la Sociedad Americana de Ecocardiografia este año(1).
Talvez van a discordar con mi posición, pero creo necesario revisar las guias de evaluación en atletas de alto rendimiento aparentemente sanos, en los que la práctica deportiva competitiva aumenta 6 veces el riesgo de muerte súbita con repecto a la población general (en la poblacion general el riesgo de MS es e 0,5% y en este grupo de 3%) Esto quiere decir que el deporte de alto rendimiento implica e un riesgo en si mismo, por lo que encuentro plenamente justificado la realización de un ecocardiograma en forma rutinaria adicionalmente a la minuciosa historia clínica, examen físico y ECG.
Mis cordiales saludos

Martin Ibarrola

1. Grenier MA, Hinton R, Knilans TJ, et al. An echo screening tool for sudden cardiac death in young athletes. American Society of Echocardiography 23rd Annual Scientific Sessions; June 30 to July 3, 2012; National Harbor, MD. Abstract P1-105

En 2007, la legislatura de Texas disponibilizó recursos para la realización de un amplio estudio piloto con el objetivo de identificar estudiantes-atletas que pudieran estar en riesgo de MS utilizando un cuestionario, examen físico, ECG y ECO limitado. Los autores trataron de determinar

1) La viabilidad de un amplio programa de rastreamiento cardiovascular;
2) La capacidad de identificar de forma confiable los grupos de riesgo, y
3) Los problemas en la aplicación de un rastreamiento amplio.

Los datos fueron analizados utilizando ECG pediátrico. Los resultados positivos fueron confirmados por un revisor en forma ciega.

En 31 salas (con 2.506 estudiantes), los hallazgos del ECG levantaron sospecha de enfermedad cardiovascular en 57 (2,3%), siendo 33 sugestivos de HCM, 14 con SQTL, 7 con síndrome de WPW, y 3 con potencial presencia de anomalía coronaria.

De los 2.051 ECHO realizados, 10 tuvieron resultados relativos a la enfermedad (9 de MCH y 1 con miocardiopatía dilatada).

De los 33 jóvenes con hallazgos electrocardiográficos compatibles con HCM, los ECOs fueron normales en 24 de los 33. De los 33 que permanecieron en situación de riesgo de MSC en el ECG o ECO 25 (65,8%) seguido de la evaluación recomendada, se confirmó SQTL en 4, síndrome de WPW en el 7, y miocardiopatía dilatada en 1. La concordancia interobservador fue del 100% con el ECG y de 79% para el ECHO. El cuestionario identificó 895 (35% del total) expuestos a riesgo, de los cuales tuvieron enfermedad confirmada apenas 11 (1,23%).

En este gran proyecto financiado por el estado, la detección de ECG y ECO identificó 11 de los 2.506 pacientes expuestos a los riesgos de enfermedad cardiovascular. El cuestionario mostró tener un valor limitado con gran número falsos positivos. La variación interobservador fue significativa para ECHO y podría crear problemas empleando el ECO limitado. Muchos sujetos con resultados anormales en el rastreamiento declinaron de seguir un acompañamiento.

1. **Zeltser I, Cannon B, Silvana L, et al. Lessons Learned from Preparticipation Cardiovascular Screening in a State Funded Program.Am J Cardiol. 2012 Jun 15. [Epub ahead of print]**

In 2007, the Texas legislature appropriated money for a pilot study to evaluate cardiovascular screening of student athletes to identify those who might be at risk of SCD using a questionnaire, physical examination, ECG, and limited ECHO. The authors sought to determine (1) the feasibility of a state-wide cardiovascular screening program, (2) the ability to reliably identify at-risk subjects, and (3) problems in implementing screening state wide. The data were analyzed using established pediatric ECG and ECHO criteria. Positive results were confirmed by a blinded reviewer. In 31 venues (2,506 students), the ECG findings met the criteria for cardiovascular disease in 57 (2.3%), with 33 changes suggestive of HCM, 14 with LQTS, 7 with WPW syndrome, and 3 with potential coronary anomaly. Of the 2,051 ECHOs, 11 had findings concerning for disease (9 with HCM and 1 with dilated cardiomyopathy). In patients with ECG findings consistent with HCM, the limited ECHOs were normal in 24 of 33. Of the 33 who remained at risk of SCD on the ECG or ECHO 25 (65.8%) pursued the recommended evaluation, which confirmed LQTS in 4, WPW syndrome in 7, and dilated cardiomyopathy in 1. The interobserver agreement was 100% for ECG and 79% for ECHO. The questionnaire identified 895 (35% of the total) potentially at-risk students, with disease confirmed in 11 (1.23%).

In this large state-funded project, ECG and ECHO screening identified 11 of 2,506 patients potentially at risk of cardiovascular disease. The questionnaire was of limited value and had a large number of false-positive results. Interobserver variation was significant for ECHO and might create problems with limited ECHO screening. Many subjects with abnormal screening results declined a follow-up evaluation.

1. **Zeltser I, Cannon B, Silvana L, et al. Lessons Learned from Preparticipation Cardiovascular Screening in a State Funded Program. Am J Cardiol. 2012 Jun 15. [Epub ahead of print]**

Professor Charles Antzelevitch opinion

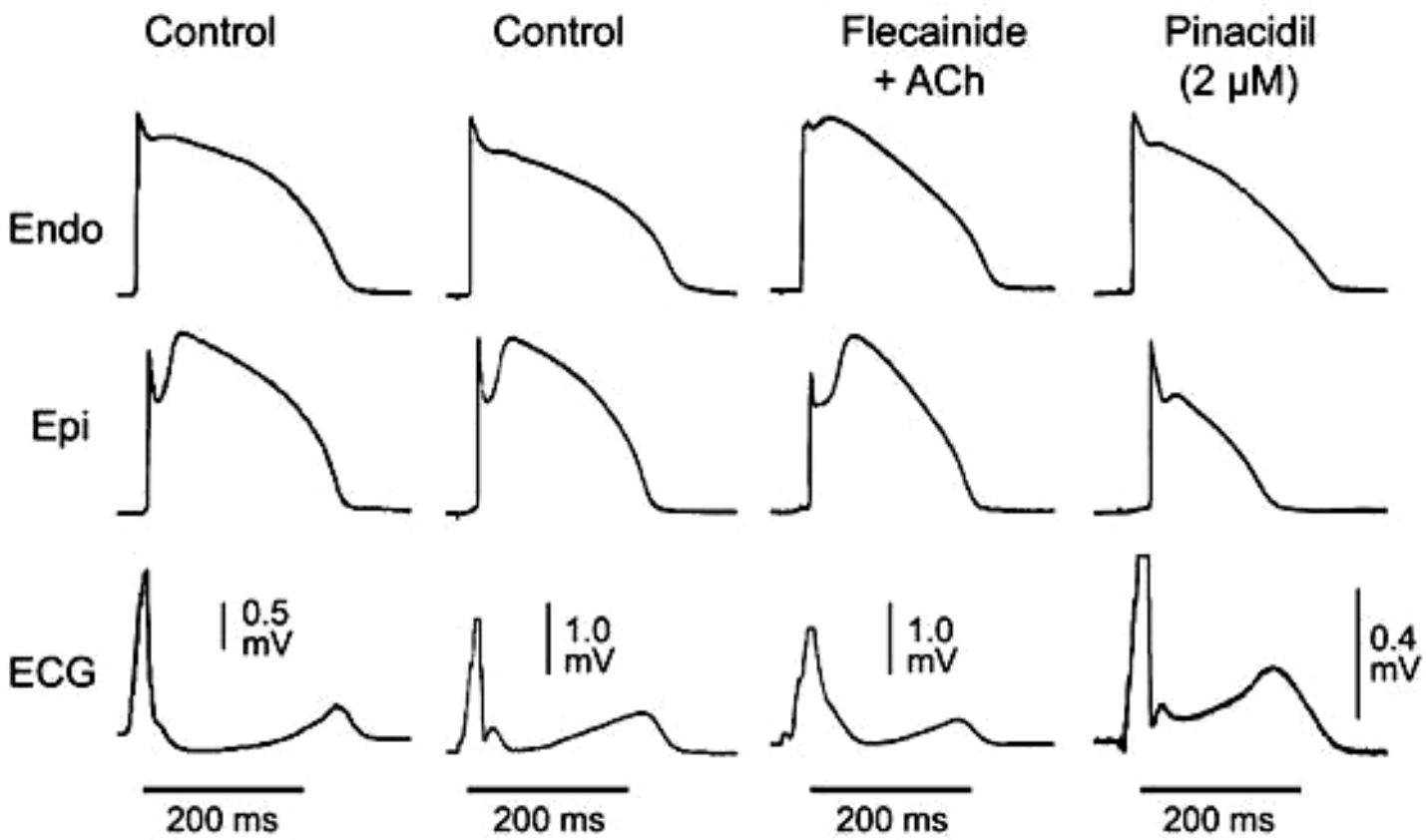
Andres

Thanks for the interesting discussion relative to Kjell's case. I would be inclined to agree that both depolarization and repolarization abnormalities may contribute to the electrocardiographic manifestations in this case, although repolarization abnormalities can largely account for the J wave manifestations (please see Figure below). As we and others have repeatedly maintained, these possibilities can be sorted out by gauging the response to an atrial premature beat (which may be observed spontaneously) or atrial pacing, obviating the need for excessive speculation. As you are aware, early repolarization pattern (ERP) is the rule rather than the exception in well-trained athletes. In this setting, I believe there is good agreement that ERP is due to accentuation of early repolarization of ventricular epicardium and that the pattern diminishes or normalizes as heart rate is accelerated. Conduction problems interrupting the QRS, on the other hand, show the opposite response to an acceleration of rate. We would do well to keep in mind that conduction problems can at times mask repolarization abnormalities, which may underlie the true arrhythmogenic substrate, as recently reported for a case of Brugada syndrome by Chiale and co-workers (**Heart Rhythm.** 2012 Jun;9(6):974-6). The interesting report by Chiale et al showed that a coved-type ST-segment elevation diagnostic of BrS could be induced by anticipated right ventricular apex pacing causing fusion QRS complexes to minimize the right ventricular activation delay and RBBB present during spontaneous atrioventricular conduction.

Kjell, we would be pleased to do a genetic screen on this patient to see if there is a genetic component contributing to exacerbation of the increased vagal tone in this well-trained athlete.

Best regards to all

Charlie



From Antzelevitch C. et al. J Am Coll Cardiol. 2011 Apr 12;57(15):1587-90

Gracias por la interesante discusión en relación al caso del Dr. Kjell. Yo me inclinaría a concordar que en el presente caso presenta tanto problemas de conducción dentro del QRS cuanto alteraciones de la repolarización que puede contribuir con las manifestaciones electrocardiográficas de las "ondas J", aunque las alteraciones de la repolarización en gran medida puede dar cuenta de las manifestaciones de una onda J (por favor vea la figura). Como nosotros y otros han sostenido en reiteradas oportunidades, esta posibilidad se puede demostrar en el laborarotio de electrofisiologia observando la respuesta a un ritmo auricular prematuro por la estimulación auricular programada (que puede ser espontáneo) y asi evitando la necesidad de una especulación excesiva.

Como sabemos, el patrón de repolarización precoz (RP) es la regla y no la excepción en atletas bien entrenados. En este contexto, creo que existe consenso que el mencionado patrón obedece a la acentuación de la repolarización precoz en el epicardio ventricular y que el mismo disminuye o se normaliza cuando la FC aumenta. (ejemplo usando isoproterenol)

Problemas de conducción dentro del QRS, por otro lado, ocasionan respuestas opuestas ante el aumento de la FC. Ademas, deberiamos tener en mente que los problemas de conducción pueden, enmascarar las alteraciones de repolarización, que puede ser la base del sustrato arritmogénico, como informaron recientemente en un caso de síndrome de Brugada Chiale y col (Heart Rhythm 2012 jun;. 9 (6) :974-6). En este interesante caso reportado fue demostrado que el patrón tipo 1 puede ser inducido por la estimulación anticipada ápex del ventrículo derecho la cual produce complejos QRS simultáneos y no secuenciales (de fusión) que reducen al mínimo el retraso de activación ventricular derecha y el BRD presente durante la conducción auriculoventricular espontánea. En otras palabras la anulación o atenuación del BRD pone de manifiesto el patrón tipo 1 en el Brugada.

Kjell nosotros nos dispondriamos a realizar el rastreamiento genético en este paciente para ver si el componente genético contribuye para exacerbar el tono vagal en este atleta bien entrenado.,

Abrazo a todos

Charlie

Profess Borys Surawic comments

Dear Professor Riera: In your recent discussion of the ECG of a 19-year old Finnish boy, whose ECG I consider normal I am in agreement with Professor Wilde ,my name was mentioned by a discusser calling me "guru". This obliges me to state my view in greater detail on the subject of terminology,in particular the use of terms early repolarization, J point, J wave and J wave syndrome-all of which are unnecessary challenging the users of these terms whom I acknowledge as respected investigators

.This also brings up the question why these terms have not entered official nomenclature and have not been used in the past by pioneering authorities e.g. Thomas Lewis, Franklin Wilson, Lepeshkin, Goldreyer, Durrer, Selvester, Chou, Janse, Castellanos, Pipberger, Kossman, Mirvis, Bellet etc etc

Safely assuming that electrical system of the heart has not changed with the global warming, and the construction of recording machines, I *do not know why the small "bumps" or splintering at the end of QRS have become of such interest rather recently,*

I can give you my take: In the early nineteen fifties Eugene Lepeschkin and I investigated ventricular gradient in ECG magnified several times and stumbled on difficulty of identifying the end of QRS complex. In our paper in **Am,Heart J 44:80-88,1952** we concluded that "there is no exact objective definition of the point where the QRS ends and the ST-segment begins, We proposed several methods to deal with this seemingly insoluble problem but left it at that

We know that in a single ventricular action potential the point at which depolarization ends and repolarization begins is clearly marked, but in the ECG where depolarization continues when the earlier depolarized fibers begin to repolarize, we find at or near QRS end a mixture of both processes which I believe to account for terminal notches or small bumps with variable expression at different heart rates and in different leads.

This does not explain why it is called ER.Repolarization can be short or long and followed by afterdepolarization, but it cannot be early or late (then what? J point is not mentioned because it is beginning of ST-segment,J wave occurs in severe hypothermia and calcium overload.,J syndromes in the imagination of their proponents.

Sincerely, Borys Surawic

Estimado profesor Riera: En el reciente debate sobre el ECG de un joven finlandés de 19 años de edad, cuyo ECG que considero normal, estoy de acuerdo con el profesor Wilde, mi nombre fue mencionado consderandome su "gurú". Esto me obliga a expresar mi punto de vista con mayor detalle sobre el tema de la terminología, en particular el uso de la repolarización precóz términos, la letra J, J y la onda de la onda J-síndrome de todos los cuales son innecesarios desafiando a los usuarios de estos términos que me reconozcan como investigadores respetados. Esto también trae a colación la pregunta de por qué estos términos no han entrado en la nomenclatura oficial y no se han utilizado en el pasado por las autoridades pioneras, por ejemplo, Thomas Lewis, Franklin Wilson, Lepeshkin, Goldreyer, Durrer, Selvester, Chou, Janse, Castellanos, Pipberger, Kossman, Mirvis, Bellet, etc, etc Con seguridad el supuesto de que el sistema eléctrico del corazón no ha cambiado con el calentamiento global, y la construcción de máquinas de grabación, no sé por qué los pequeños "baches" o grietas en la final del QRS se han convertido en tema de tanto interés recientemente.

Te puedo dar mi opinión: A principios de los años cincuenta Eugene Lepeschkin y yo investigando gradiente ventricular en el ECG ampliado varias veces y tropezamos con la dificultad de identificar el final del complejo QRS. En nuestro trabajo en **Am, Heart J 44:80-88,1952** llegamos a la conclusión de que "no existe una definición objetiva exacta del punto donde el QRS termina y comienza el segmento ST, hemos propuesto varios métodos para hacer frente a este problema aparentemente insoluble Sabemos que en una sola acción ventricular potencial del punto en que termina la despolarización y repolarización comienza está claramente marcada, pero en el ECG en la despolarización continúa cuando las fibras despolarizadas anteriores comienzan a repolarizar, se encuentra en o cerca del final del QRS una mezcla de ambos procesos que creo que dan cuenta de las muescas de terminales o protuberancias pequeñas, con expresión variable en diversos ritmos cardíacos y en distintas derivaciones.

Esto no explica por qué se llama Repolarization precóz puede ser corto o largo y seguido por posdespolarización, pero no puede ser temprano o tarde (¿entonces qué? Punto J no se menciona porque es el comienzo de segmento ST, onda J se presenta en la hipotermia severa y sobrecarga de calcio., síndromes de J en la imaginación de sus autores

Final comments

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

In young competitive athletes SCD frequently occurs as a tragic first manifestation of clinically unapparent underlying structural or electrical cardiac disorders (channelopathies). An increased risk may be reflected by typical ECG alterations preceding symptoms but a correct interpretation is often challenging due to a high prevalence of training-related ECG alterations in competitive athletes mimicking such disorders. Misinterpretation may thus result in either unnecessary disqualification from competitive sports or continuation despite an increased risk or extensive diagnostic work-ups yielding additional equivocal findings. However, as observed in large athlete cohorts in recent years a variety of ECG alterations, such as:

1) Sinus bradycardia: (observed in ≈ 75% of cases) Heart rates between 30 to 40 bpm at rest are not rare. In highly trained athletes, there are descriptions of HR of 25 bpm.
2) Phasic or respiratory sinus arrhythmia: present in 60% (in the population in athletes in 2.4%).
3) Sinus pauses.
4) Junctional rhythm present in 0.31% (in the general population in 0.02%).
5) Variable pacemaker or rhythm of left atrium;
6) rarely ventricular rhythm;
7) Junctional escape beats;
8) Long sinus pauses: they are frequent (> 2 seconds);
9) Supraventricular ectopic beats;
10) Right axis deviation and frequent occurrence of left posterior fascicular block, which is thought to be 'potentially malignant', requires further investigation(3);

1. **Le VV, Wheeler MT, Mandic S, et al. Addition of the electrocardiogram to the preparticipation examination of college** athletes**. Clin J Sport Med. 2010 M:98-105.**
2. **Sharma S, Whyte G, Elliott P.et al. Electrocardiographic changes in 1000 highly trained junior elite** athletes**. Br J Sports Med. 1999 Oct;33:319-324.**
3. **Swiatowiec A, Król W, Kuch M, et al. Analysis of 12-lead electrocardiogram in top competitive professional athletes in the light of recentguidelines. Kardiol Pol. 2009 Oct;67:1095-10102.**

11. P waves: increase of voltage and notches are described
12. *First degree AV block*: obsrveded in 5% to 30% (in non athletes, 0.65%). When the PR interval does not reach the value as a criterion for 1st degree AV block, it is relatively prolonged. The PR interval normalizes or even gets smaller after exercise;
13. *Second degree atrioventricular block (Mobitz type I)* it is observed in 10% (in non athletes < 1 in 30,000 or 0.003%), and it disappears invariably during exercise and atropine;
14. *Rarely Mobitz type II, AV dissociation and third degree AV block(*5 each 12,000 athletes.)
15. *Isolated increased right and left ventricular voltage: Augmented QRS voltage is observed in ≈ 40%-50% of cases), positive Sokolow-Lyon criteria for LVH ; however, only 27% had a Romhilt-Estes score of ≥4.(1) Isolated Sokolow-Lyon voltage criterion for LVH is common; however, associated abnormalities that indicate pathological hypertrophy are absent;*
16. *Incomplete right bundle branch block. (observed in ≈ 71% of cases) or non-specific intraventricular conduction delay;*
17. *Early repolarization pattern;*
18. *Vagotonic or high T wave voltages(1), diphasic and inverted T waves, and*
19. *Prominent U waves.(2)*

These ECG features, called group I - 'benign', common - thought to be consistent with the athlete's heart syndrome represent common variants of ECGs of athletes reflecting physiological and training-related cardiac adaptations. These ECG modifications do not usually require further diagnostic evaluation.

1. **Boraita Pérez A, Serratosa Fernández L. "The athlete's heart": most common electrocardiographic findings. Rev Esp Cardiol. 1998 May;51:356-368.**
2. **Ferst JA, Chaitman BR. The electrocardiogram and the athlete. Sports Med. 1984 Sep-Oct;1:390-403.**

A significant negative correlation was found between heart rate and PR duration in athletes. In the sinus tachycardia group, the PR duration was shorter and the ST depression more prominent than in the other groups. Parameters of LVH were markedly increased in athletes with PR ≥ 220ms, while the heart rate was only slightly decreased, suggesting an association between prolonged atrioventricular conduction time and LVH.(1)

In contrast, group II - 'suspected', uncommon ECG features - which may occur due to organic heart disease are:

1) Repolarization abnormalities: Strain pattern in left leads, T-wave inversion in right precordial leads in absence of RBBB after 16 yo. Minor T wave inversions in leads other than V2 and V3 may be present in *athletes* ***and non-*** *athletes* ***less than 16 but should be an indication for further investigation in older*** *athletes***.**(2)

2 Complete bundle branch block

3) Prolonged QT interval

4) Pathological Q waves

5) Ventricular arrhythmia

6) Inverse T wave

7) Pathological QRS axis deviation.

These features are strongly suggestive of underlying disorders and require further evaluation even in asymptomatic athletes. Thus, the ECG plays a pivotal role in the prevention of SCD in competitive athletes.

1. ***Bjørnstad H, Storstein L, Dyre Meen H, Hals O. Electrocardiographic findings of heart rate and conduction times in athletic students and sedentary control subjects. Cardiology. 1993;83:258-267.***
2. ***Sharma S, Whyte G, Elliott P.et al. Electrocardiographic changes in 1000 highly trained junior elite*** *athletes***. Br J Sports Med. 1999 Oct;33:319-324.***

Arrhythmias in the hearts of athletes and comparative incidence with the general population

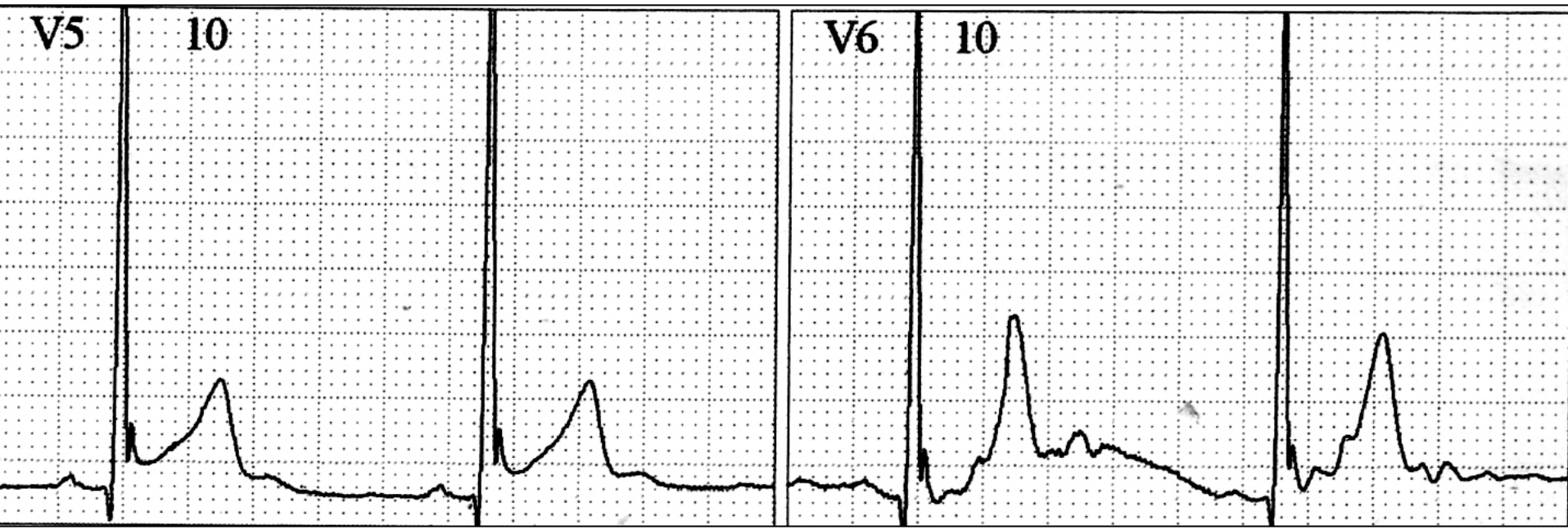
ARRHYTHMIA	GENERAL POPULATION	ATHLETES
Sinus Bradycardia	23.7	50-85
Sinus Arrhythmia	2.4-20	13.5-69
Atrial Variable Pacemaker	NOT AVAILABLE	7.4-19
1st degree AV block	0.65	6-33
2nd degree AV block		
Mobitz Type 1	0.003	0.125-10
Mobitz Type II	0.003	NOT REPORTED
3rd degree AV block	0.0002	0.017
Junctional Rhythm	0.06	0.31-7.0

Table comparing the incidence of arrhythmias in the general population and in athletes.

Dear Peter Kukla: You used the appropiate term: “in this context the presence of OS-ASD is almost impossible”. Why almost and not totally impossible? Because, as has just written Kjell Nikus: ***It is possible that a murmur could have been missed by the physician who did the examination.*** Currently young doctors do not learn properly physical examination.

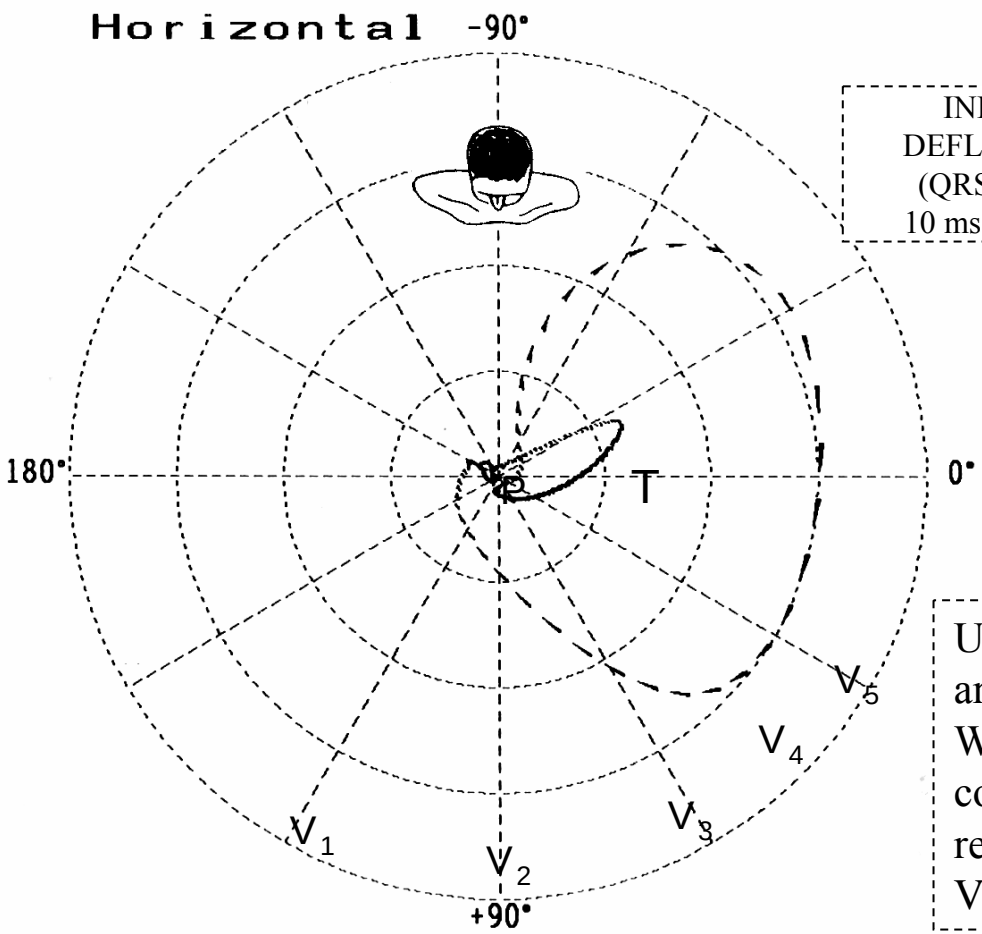
Our “gurú” Professor Borys Surawicz wrote: the term early repolarization lacks precise definition because there are no defined criteria for the upper limits of ST segment elevation in normal young males.

The universal criteria used in trials for ER is the presence of at least two adjacent precordial leads show ST segment elevation, with values ≥1 mm. Additionally, notching, irregular or slurring contour of the terminal QRS complex (J point). Figure



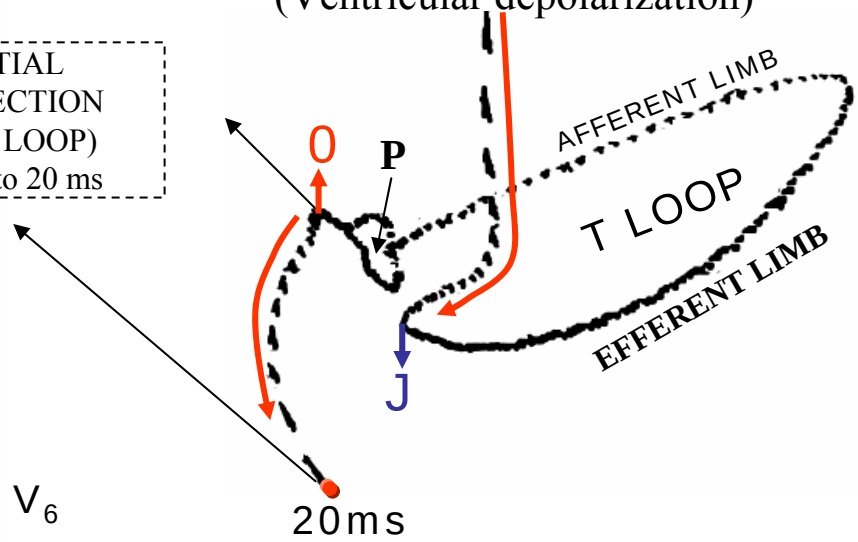
There is a period of overlap between the potentials that arise from the last portions of muscle to be excited and repolarization potentials from other regions. This period my last for 10ms or more. However, in a normal sequence both the potentials at the end of depolarization and the beginning of repolarization are small, so the ECG does return to a voltage near the baseline. The J point sometimes used as a marker for the end of excitation only approximately. Again the approximation occurs because of the overlap of potentials

VCG HORIZONTAL PLANE CORRELATION



INITIAL DEFLECTION (QRS LOOP) 10 ms to 20 ms

End of QRS loop
(Ventricular depolarization)



Usually 0 & J points are coincident. In this case, they are not: ST-segment elevation

When ST is elevated, the J and 0 points do not coincide: we observe this phenomenon in early repolarization pattern, Brugada syndrome, Idiopathic VF, and short QT syndrome

0 point: it corresponds to the end of biatrial chamber activation, QRS loop onset (because PR segment does not exist, it is only a point) and the end of ventricular repolarization (T loop).

J point: in vectorcardiography, it corresponds to 3 elements: end of ventricular depolarization (QRS complex); beginning of repolarization (ST segment) when it does not present depression or elevation, and T wave onset.

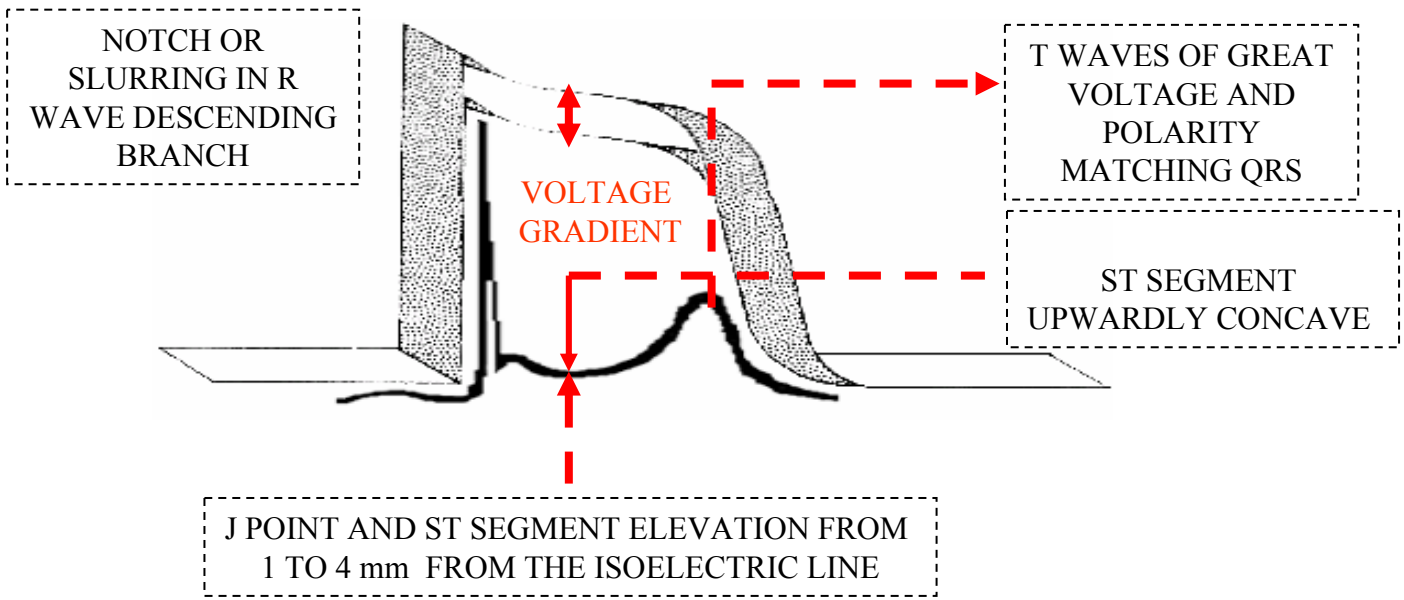
at the ending of depolarization and the beginning of repolarization(1). The duration of overlap between the onset of ventricular recovery and the end of the excitation was determined from isopotential maps and ranged rom 4 to 16 ms. There are no significant correlation between these values and either torso or V lead potentials at either 40 or 80 ms into S-T segment.

1) Standard precordial leads do not accurately predict maximal torso potentials during the S-T segment, and 2) The degree of overlap between repolarization and depolarization is not a major determinant of precordial voltage. Hence, the rationale for use of the term "early repolarization" to describe this clinical condition is not substantiated.(2;3)

Gender differences(4): In comparison to men, women have a higher incidence of TdP and a greater lengthening of QT-interval after administration of class III antiarrhythmic drugs. Before puberty, the QT intervals and the patterns of ventricular repolarization in boys and girls are similar. At puberty, in boys the QT interval shortens, and a typical male pattern of ventricular repolarization develops. This pattern is characterized by a higher amplitude of the J-point, a shorter and steeper ST segment course, a steeper ascent, and a higher amplitude of the T wave. This pattern is prevalent in >90% of young males. With increasing age the prevalence of the male pattern in males declines gradually and drops to 14% in the oldest age group. The rise and fall of the prevalence of the male pattern appears to parallel the rise and decline of testosterone in males. The female pattern of ventricular repolarization is prevalent in about 80% of females in all age groups. The females have greater divergence of L calcium current among different layers of the myocardium and a lower density of the repolarizing IKr and IKs currents. The males with female pattern are at the same risk of TdP as the females or whether the females with male pattern are at lower risk of TdP than the females with female pattern. S-T segment elevation is commonly observed in the ECG of normal persons.

1. Spach MS, Barr RC, Benson W, et al. Body surface low-level potentials during ventricular repolarization with analysis of the ST segment: variability in normal subjects.Circulation. 1979 Apr;59:822-836.
2. Mirvis DM. Evaluation of normal variations in S-T segment patterns by body surface isopotential mapping: S-T segment elevation in absence of heart disease. Am J Cardiol. 1982 Jul;50:122-128.
3. Surawicz B, Macfarlane PW. Inappropriate and confusing electrocardiographic terms: J-wave syndromes and early repolarization. J Am Coll Cardiol. 2011 Apr 12;57:1584-1586.
4. Surawicz B, Parikh SR. Prevalence of male and female patterns of early ventricular repolarization in the normal ECG of males and females from childhood to old age. J Am Coll Cardiol. 2002 Nov 20;40:1870-1876.

In early repolarization, there is a voltage gradient, however, no dispersion of duration of action potentials in ventricular wall thickness. For this reason, these patients showed ST segment elevation with no tendency to develop arrhythmias.



Theoretical electrophysiological explanation for ST segment elevation in ECGs in athletes.

ECG CRITERIA THAT SUGGEST EARLY REPOLARIZATION PATTERN (ERP)

✓ HR: sinus bradicardia is frequent;

✓ Axes of QRS, ST segment and T wave, are oriented in the same direction in the FP;

✓ Deep and narrow Q waves followed by R wave of great voltage in left precordial leads;

✓ Notch or slurring of R wave descending branch;

✓Transition area in precordial leads of sudden occurrence;

✓Presence of at least two adjacent precordial leads show ST segment elevation, with values ≥1 mm. J point and ST segment elevation, usually < 2 mm (exceptionally it may be > 5 mm) of superior concavity in middle and/or left precordial leads and possibly in inferior leads;

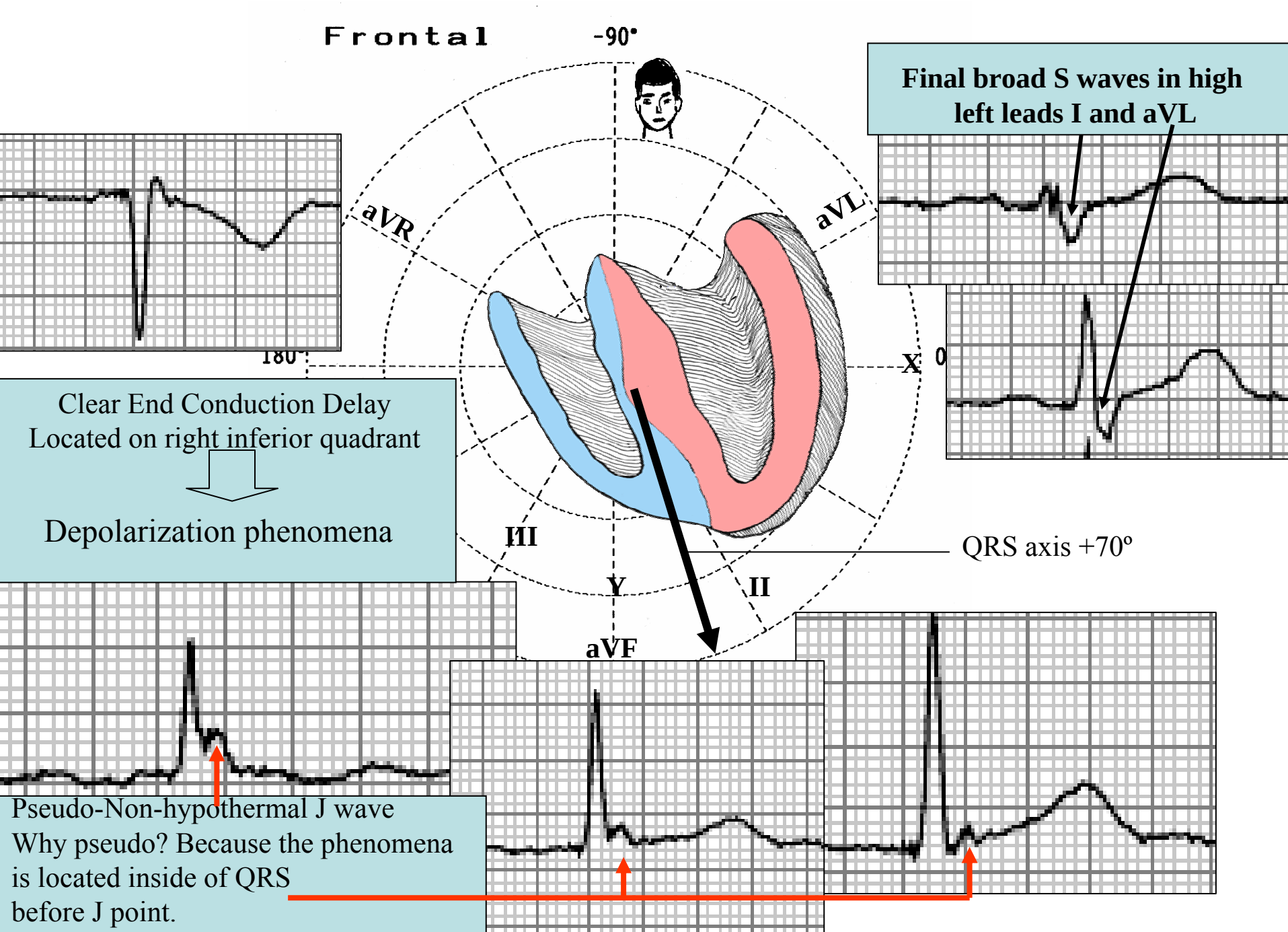
✓Notch, irregular or slurring contour of the terminal QRS complex (J point).

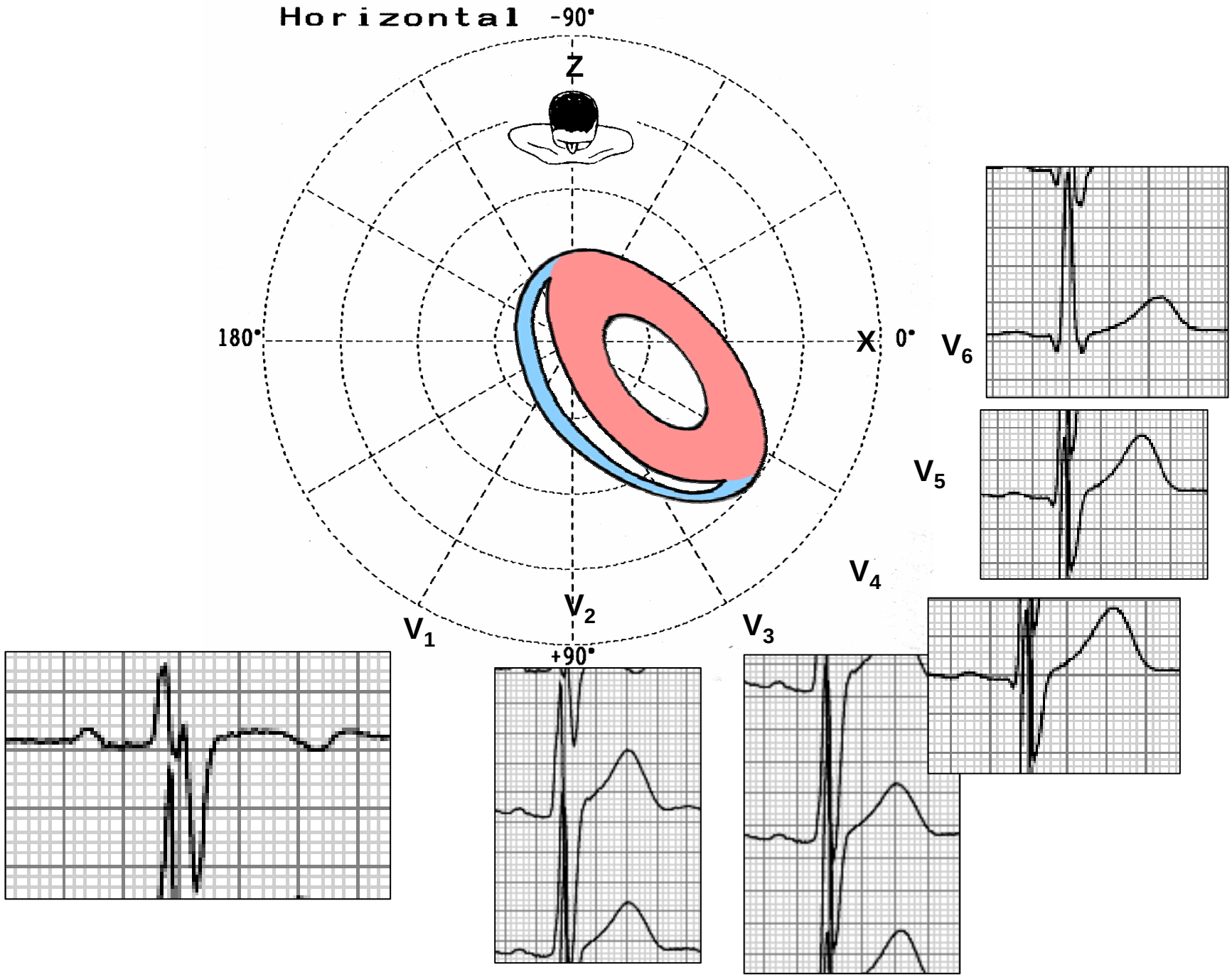
✓Possible reduction in J point and ST segment elevation by sympathetic action and sympathomimetic drugs;

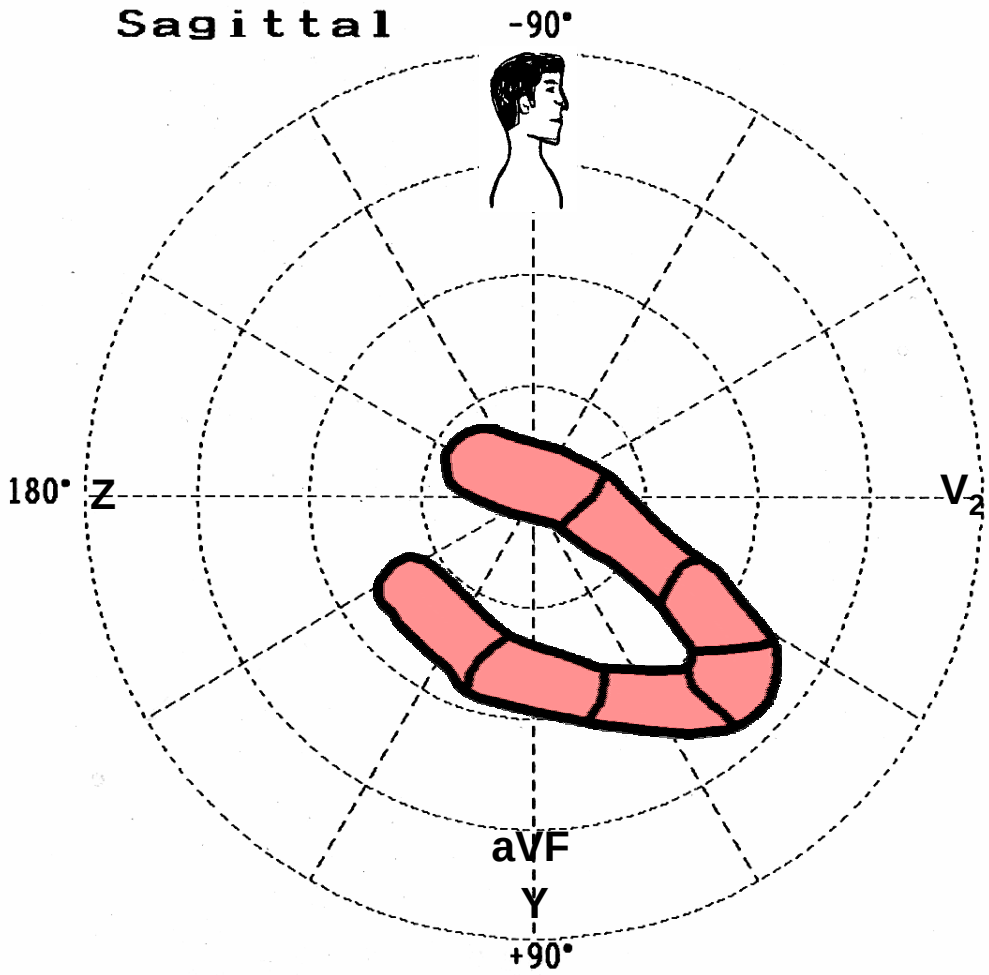
✓Absence of reciprocal or mirror image (exception in VR lead);

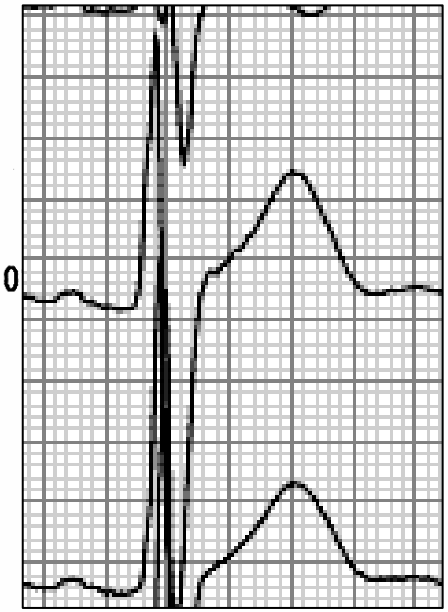
✓Pseudo- symmetrical T waves, with great width and polarity matching QRS;

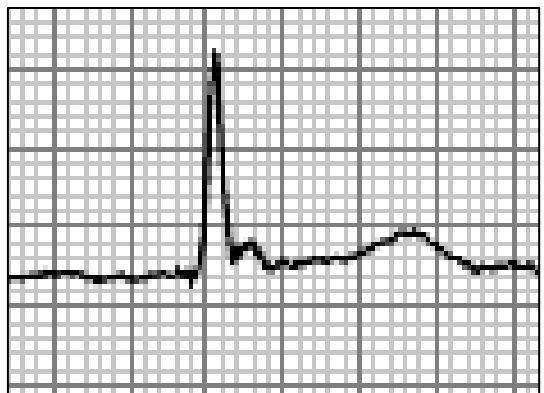
Electrocardiographic criteria of early repolarization syndrome.


Frontal
-90°
aVR
aVL
180°
0
X
III
II
Y
aVF
Final broad S waves in high left leads I and aVL
Clear End Conduction Delay
Located on right inferior quadrant
Depolarization phenomena
QRS axis +70º
Pseudo-Non-hypothermal J wave
Why pseudo? Because the phenomena is located inside of QRS before J point.


Horizontal
-90°
Z
180°
X
0°
+90°
V1
V2
V3
V4
V5
V6


Sagittal
-90°
180°
Z
V2
aVF
Y
+90°


0



J wave on ECG : Topographic Classification proposal

1. **Real or truly J wave:** when observed only after J point. It observed on J point and ST segment (Phase 1 and 2 of monophasic action potential: early and middle repolarization phase). Eventually, this wave mimic a fragmented QRS. All extra ECG waves or deflections of slow inscription located between the end of QRS and the beginning of the ST segment, in the location corresponding to the J point, It called also **junctional wave** or **J deflection**. Since this is a wave and not a point, it also affects the initial portion of the ST segment, causing in it a coved-type elevation, and for this reason it is known as well as injury potential. This upward convexity profile is the cause of its denomination as **camel-hump sign** or the **camel's hump**, or **hump-like defection**. Since it is similar in its aspect to the ventricular pre-excitation delta waves (δ), the **late delta wave** term has been rarely used. The δ wave of ventricular pre-excitation is located in the Ja point (end of the PR segment and beginning of the QRS complex). Finally, there is an eponymous denomination, the **Osborn wave**, which the way we see it is an unfair choice, since the first one to describe it was Tomaszewski1. Five years later, after the first description, the German Grosse-Brockhoff, F. et al., described it in an experimental work.(2) The description by Osborn happened fifteen years later, in 1953(3) Ironically, valuable Cardiology texts called the wave as Osborne, adding an e vowel to the original name of the author(4).
2. **Pseudo J wave:** When the J wave is located before the J point, inside of QRS complex. It is a depolarization phenomena. Correspond to Phase 0 of monophasic action potential.
3. **J wave located before and after J point:** eclectic mechanism depolarization and repolarization is present Example Brugada syndrome where we observed end conduction delay at the end of QRS loop (depolrization mechanism) and additionally associated rounded T wave (repolarization mechanism)

1. Tomaszewski W. Changements electrocardiographiques observes ches un homme mort de froid. Arch Mal Coeur 1938; 31:525-528.
2. Grosse-Brockhoff F, Schoedel W. Das Bild der akuten Unterkuhlung in Tierexperiment. Arch. Exp. Pathol Phramakol.1943; 201: 417.
3. Osborn JJ. Experimental hypothermia: respiratory and blood pH changes in relation to cardiac function. Am J Physiol 1953; 175:388-398.
4. Braunwald E. Heart Disease. A Texbook of Cardiovascular Medicine. 5th Edition, 1997; pg 140-141.

ETYOLOGICAL CLASSIFICATION PROPOSAL FOR ECG J WAVES

I) J wave of hypothermia

II) J wave in normothermal patients:

(IIa) Hypercalcemia[1]

(IIb) Injuries in the central nervous system: subarachnoid hemorrhage, post-heart arrest and in cervical sympathetic system dysfunction[2].

(IIc) Rarely in early repolarization syndrome[3].

(IId) Brugada "entities":

(IId1) Familial cases (≈17%): true Brugada disease;

(IId2) Sporadic cases (≈63%): Brugada syndrome[4].

(IId3) Brugada phenocopies: they are those clinico-pharmacological entities or circumstances, where Brugada phenotype or sign in ECG, may be found as a consequence of causing increase in Ito channel function in the ventricular epicardium or decrease of slow calcium channel[5].

(IIe) In concealed forms of arrhythmogenic dysplasia of the right ventricle[6];

(IIf) In variant angina of Prinzmetal[7].

1) Topsakal R, et al. Jpn Heart J. 2003; 44:1033-1037.
2) Carrillo-Esper R, et al. Cir Cir. 2004; 72:125-129.
3) Nava A, et al. Mises a Jour Cardiologiques 1988;17:157-159.
4) Schulze-Bahr E, et al. Hum Mutat. 2003;21:651-652.
5) Shimizu W. J Electrocardiol. 2005; 38:22-25.
6) Corrado D, et al. J Am Coll Cardiol 1996, 27: 443-448.
7) Aizawa Y, et al. Intern Med. 2006; 45:43-44.

Proposal for hypothermal and normothermal J waves classification in electrocardiogram.

ELECTROPHYSIOLOGICAL SUBSTRATE OF J WAVE

Experimental studies point out that J wave appearance is the consequence of the presence of transmural gradient in ventricular wall thickness, secondary to existence in the epicardium but not the endocardium, of significant notch in phase 1, mediated by a greater activity or density of initial transient outward potassium current. This greater activity and/or density of the Ito channel in epicardial cardiomyocytes, but not endocardial ones, accounts for the characteristic aspect of AP known as *"spike-and-dome configuration of the monophasic action potential"*. Moreover, the greater initial potassium outflow in the epicardium than the endocardium, causes phase 2 shortening in the epicardium, which conditions transmural dispersion of repolarization and J wave appearance, which carried to a certain level, causes a greater tendency to appearance of ventricular arrhythmia by the mechanism called functional reentry in phase 2[1].

Experimental located cooling of the right ventricular outflow tract (RVOT) in vivo in dogs, resembles the electrophysiological alterations that occur in Brugada syndrome, causing J wave appearance secondary to Ito channel activation, and causing the classical aspect of "spike-and-dome configuration" in monophasic action potential of epicardial cells in the RVOT[2].

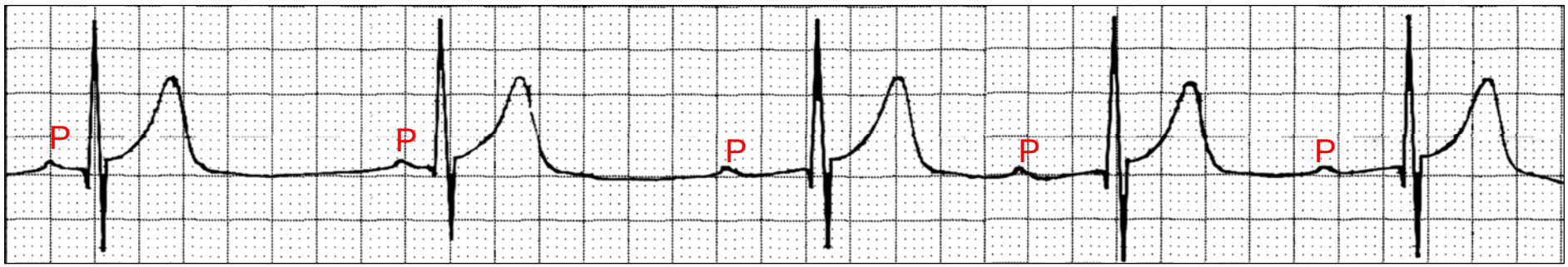
Several entities are associated to J wave appearance (J wave syndromes)**[3]** that include among others, early repolarization syndrome (rare), variant angina, intoxication by tricyclic antidepressants**[4]**, cocaine abuse**[5]**, hypercalcemia, encephalic lesion, Brugada syndrome, idiopathic ventricular fibrillation with prominent J wave in inferior wall**[6]** and the forms called concealed, of arrhythmogenic right ventricular dysplasia.

Experimental evidence support the hypothesis that one heterogeneous distribution of the Ito channel in the ventricular wall thickness accounts for the spike-and-dome configuration in monophasic AP in the epicardium, and prominent notch in phase 1 and phase 1 shortening, which results in voltage gradient that manifests by J wave**[6].**

1) Yan GX, et al. Circulation, 1996; 93: 372-379.
2) Nishida K, et al. J Cardiovasc Electrophysiol. 2004; 15: 936-941.
3) Hlaing T, et al. Ann Noninvasive Electrocardiol. 2005;10:211-223.
4) Bigwood B, et al. Anaesth Intensive Care. 2005; 33:266-270.
5) Riera AR, et al. J Electrocardiol. 2004; 37:101-104.
6) Antzelevitch C. Ann N Y Acad Sci 2005; 1047: 314-323

HOLTER RECORDING 1ST DEGREE AV BLOCK

Name: B . C. **Sex:** Male **Age:** 22 **Race:** Black **Weight:** 74 Kg.
Height: 1.82 m **Biotype:** Athletic **Date:** 01/04/2002 **Time:** 2:50:12 AM Patient sleeping.
Profession: Marathon runner



Heart rate of 38 bpm.
1st degree AV block usually observed for a few seconds, as in this case, where it is present only in the three last beats.

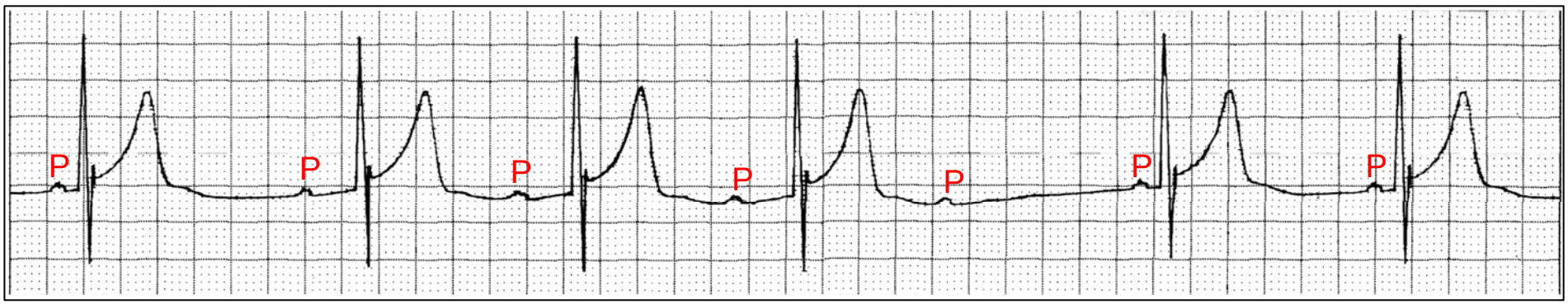
1st degree AV block is observed in average between 10% and 33% of athletes (1), generally very briefly. In non-athletes it is around 0.65%.

1) Smith WG, et al. Br Heart J 1964:469-476.

1st-degree AV block in an elite athlete in Holter monitoring.

HOLTER RECORDING

Name: A . S. **Sex:** Male **Age:** 26 **Race:** Black **Weight:** 64 Kg.
Height: 1.68 m **Biotype:** Athletic **Date:** 05/01/2003
Time: 3:42:30 AM Patient sleeping. **Profession:** long distance runner



Gradual prolongation of PR interval until the 5th P wave is not conducted: 2nd degree AV block; Wenckebach or Mobitz Type I.

This modality of dromotropic disorder is observed in more than a 20% of elite athletes (1). In the general population, 2nd degree AV block Type I & II is observed and 1 each 30,000 people or 0.003 %

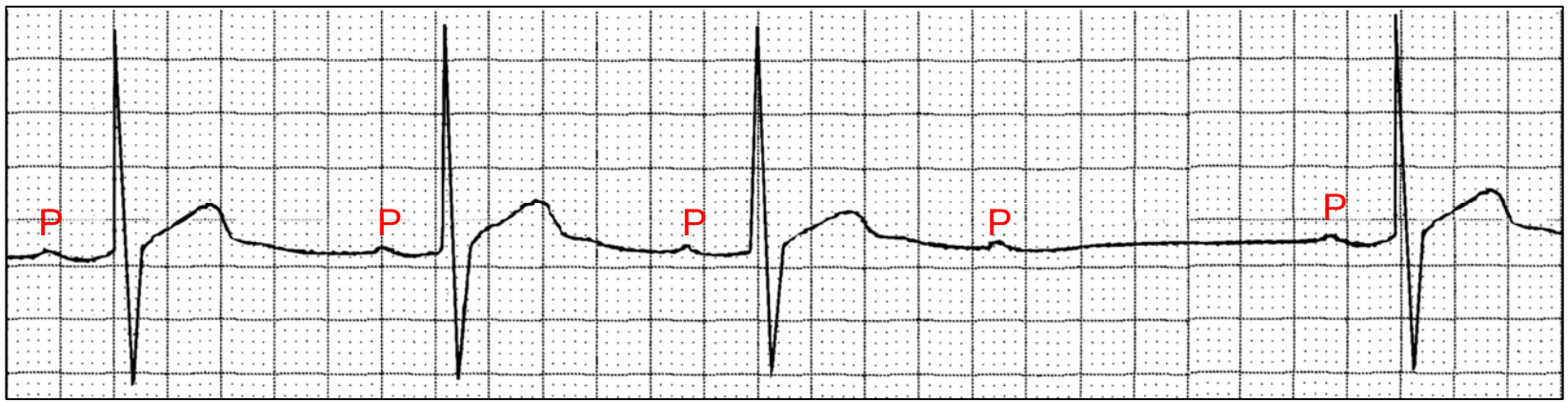
1) Viitasalo MT, et al. Br Heart J. 1982;47:213-220.

2nd-degree AV block, Wenckebach type or Mobitz Type I in an elite athlete.

HOLTER RECORDING

2ND DEGREE AV BLOCK, MOBITZ TYPE II WITH NARROW QRS

Name: E . J. **Sex:** Male **Age:** 26 **Race:** White **Weight:** 70 Kg.
Height: 1.72 m **Biotype:** Athletic **Date:** 25/01/2001
Time: 1:52:10 AM Patient sleeping **Profession:** Long distance runner

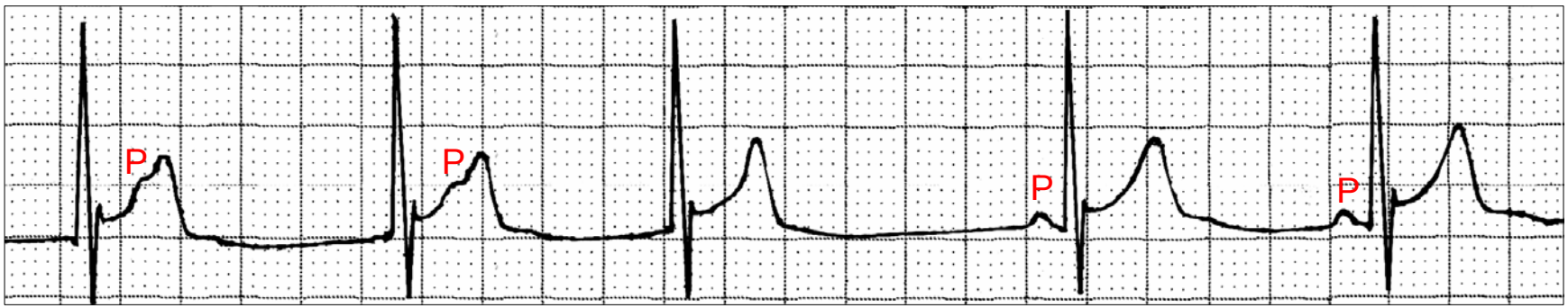


PR interval remains constant until a P wave is not conducted. This type of block is observed in 7% of the cases in athletes of enduro. Fixed or constant PR interval: it does not exist, progressive prolongation of PR, with the block occurring suddenly. In general, 2nd degree AV block type II with narrow QRS is observed in 35% of the cases and in the remaining 65%, the QRS is long.

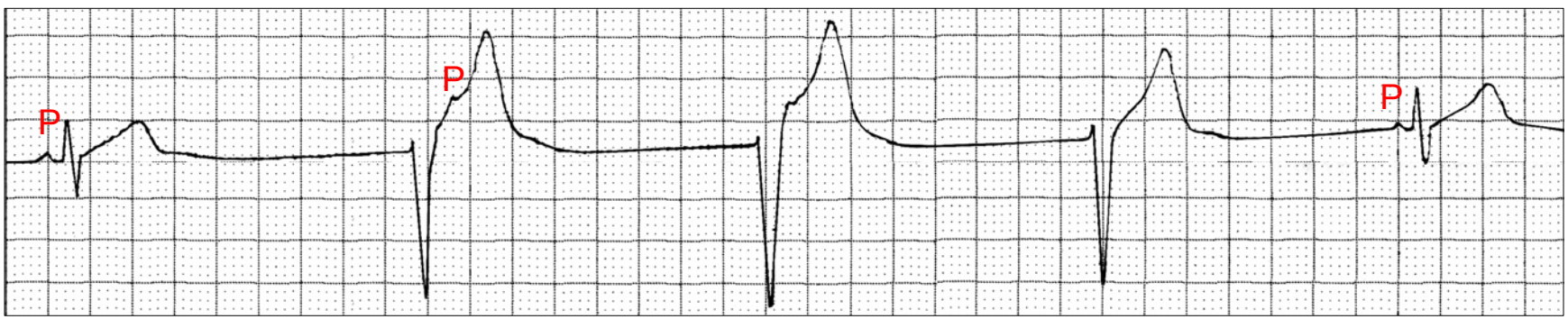
2nd-degree AV block, Mobitz type II with narrow QRS.

HOLTER RECORDING

ATRIOVENTRICULAR DISSOCIATION (DISSOCIATION BY INTERFERENCE) WITH JUNCTIONAL SCAPE RHYTHM

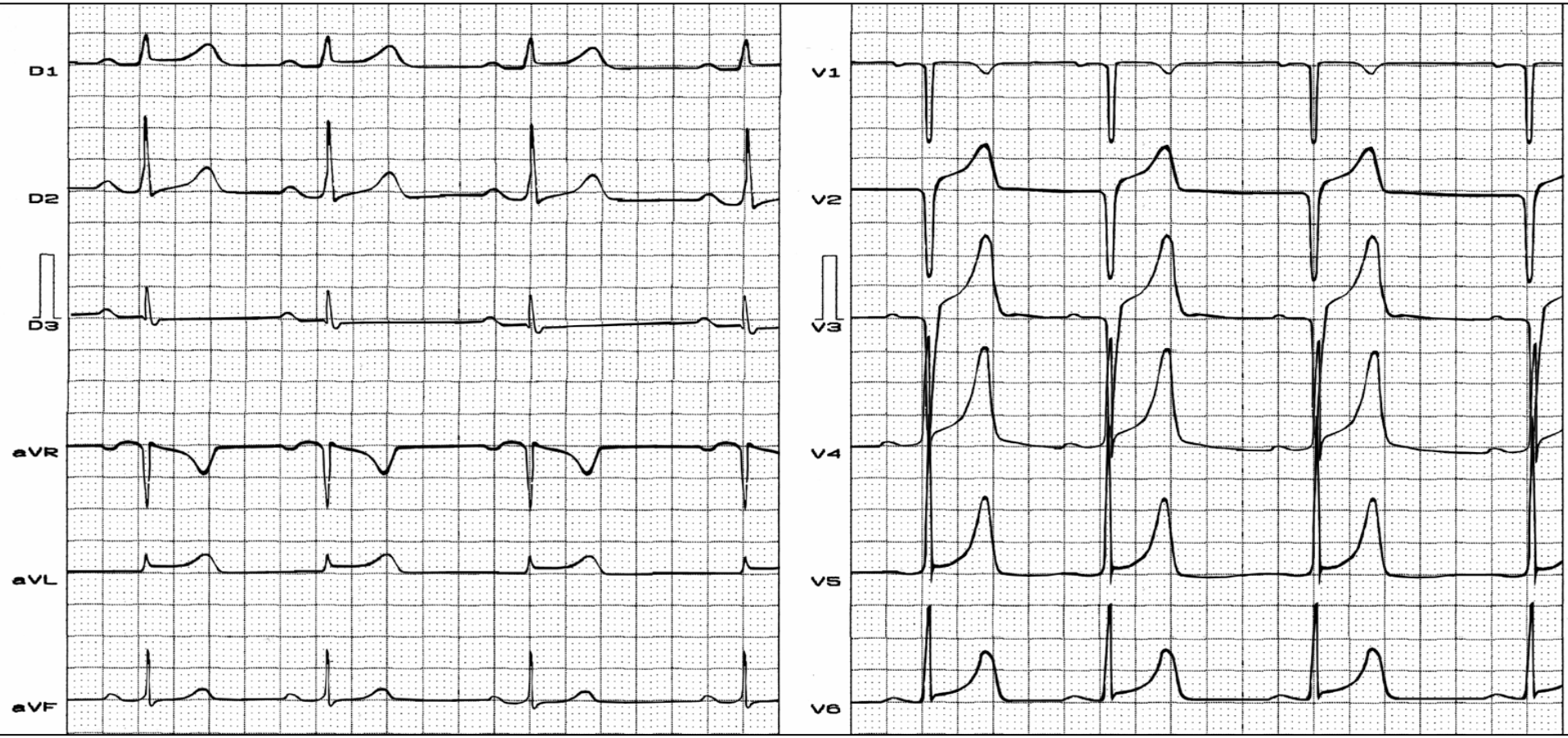


ATRIOVENTRICULAR DISSOCIATION (DISSOCIATION BY INTERFERENCE) WITH SCAPE VENTRICULAR RHYTHM



Atrioventricular dissociation (dissociation by interference) with junctional scape rhythm and atrioventricular dissociation (dissociation by interference) with scape ventricular rhythm in an elite athlete in Holter.

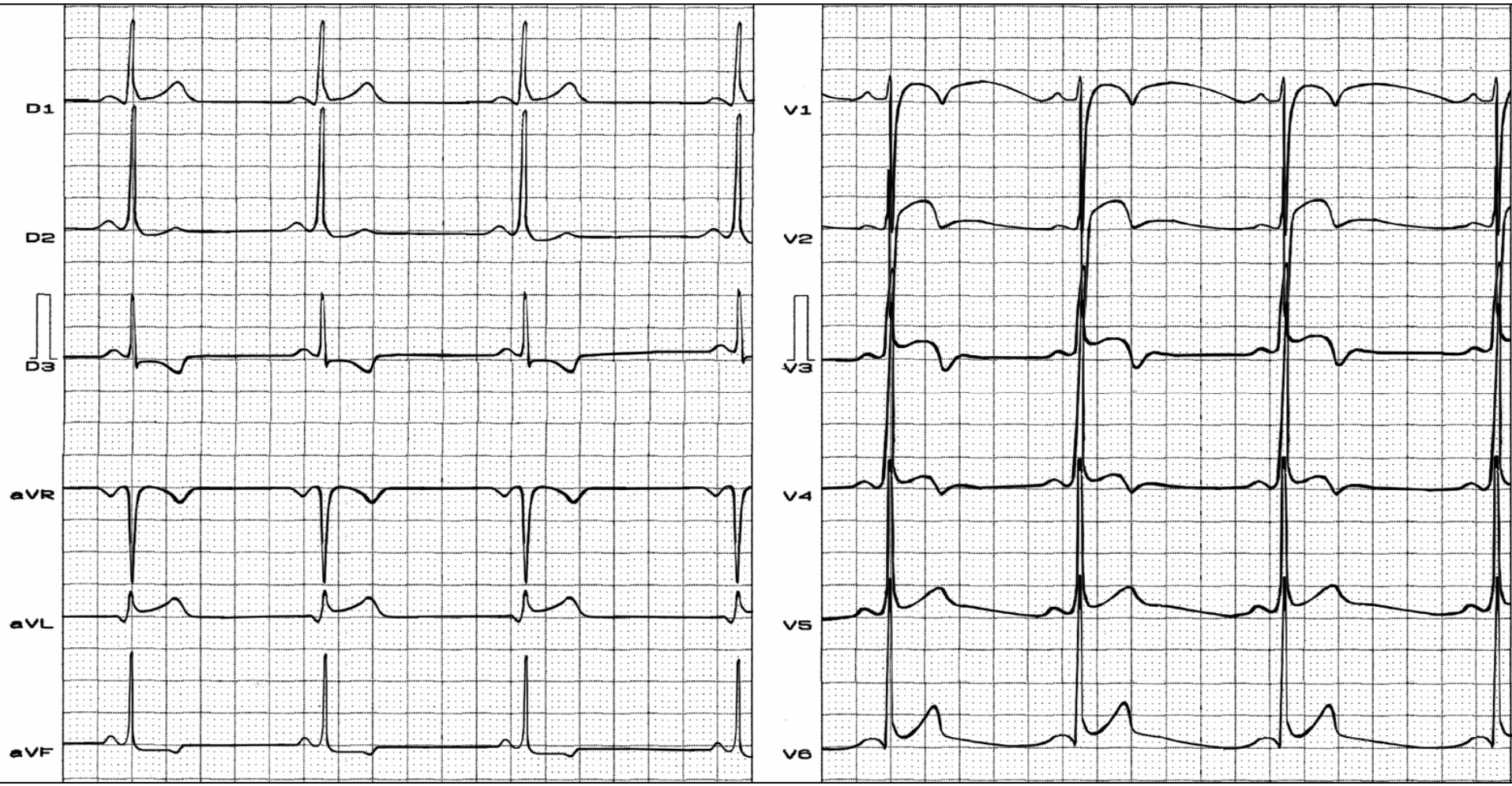
Name: BCA **Age:** 22y **Sex:** Male **Race:** Black **Weight:** 82 kg
Height: 1.96m **Biotype:** Athletic Profession: professional basketball player **Date:** 2/09/2001



CLINICAL DIAGNOSIS: athlete's heart. Normal variant.
ECG DIAGNOSIS: sinus rhythm; HR: between 50 bpm and 57 bpm: Phasic or respiratory sinus bradyarrhythmia; QS from V1 to V3: pattern of pseudo anteroseptal myocardial infarction
Peaked T waves from V3 to V6. Normal X-rays of chest and echocardiogram.

Pseudo anterior myocardial infarction pattern of in an athlete, professional player of basketball with normal heart.

Name: BCW **Age:** 24y **Sex:** Male **Race:** Black **Weight:** 86 kg**Height:** 2.02 m**. Biotype:** Asthenic
Profession: professional basketball player. **Date:** 05/01/1999



CLINICAL DIAGNOSIS: healthy patient. Tracing obtained in a periodical evaluation.

ECG DIAGNOSIS: sinus bradycardia, phasic sinus arrhythmia. Positive voltage criterion for LVE. SV_1 or V_2+RV_5 or V_6 >35 mm (Index of Sokolow Lyon). ST segment elevation from V_2 to V_6 and with negative T from V_1 to V_4. Early repolarization, pattern of pseudo injury and anterior subepicardial ischemia. Normal chest X-rays and echocardiogram. Pseudo subepicardial injury pattern and ischemia in anterior wall in an athlete, professional player of basketball with normal heart.

CONCEPT OF SUDDEN DEATH (SD) AND PREVALENCE IN ATHLETES

SD is defined as the death that is not traumatic, not violent, unexpected, which occurs within the first 6h without a prior manifestation of cardiac disease[1].

PREVALENCE

Estimated between young athletes of secondary school, as 1 in 200,000 per year. [2,3]

1) Maron BJ, et al.. J Am Coll Cardiol 1986;7:204-214.
2) Maron BJ, et al. J Am Coll Cardiol 1998;32:1881-1884.
3) Maron BJ, et al. Circulation 1996;94:850-856 [Addendum appears in Circulation 1998;97:2294].

CAUSES OF ARRHYTHMIC SUDDEN DEATH IN YOUNG ATHLETES (AVERAGE AGE: 17 YEARS)

I) ENTITIES WITH STRUCTURAL HEART DISEASE (98%)

1) Hypertrophic cardiomyopathy (HCM) whether in its obstructive form or in its non-obstructive form (36%);
2) Congenital anomalies of coronary arteries with increase of ventricular mass (19%);
3) Tumors or cardiac masses (10%);
4) Aorta rupture due to Marfan syndrome (5%). Mutation in the gene in fibrillin-1 (FBN1), in chromosome 15q21.1 and Marfan-like syndrome with no eye anomalies, mapped in chromosome 3p24;
5) Arrhythmogenic right ventricular dysplasia/cardiomyopathy (3%). Prevalence of 1 in 15,000;
6) Early atherosclerotic coronary artery disease (2%) by familial hypocholesterolemia and dominant mixed hyperlipidemia by alteration in chromosome 6;
7) Mitral valve prolapse syndrome (MVPS) (2%);
8) Myocarditis (2%);
9) Familial arrhythmogenic syndrome: ventricular and tachyarrhythmia association (syndrome of Wolff-Parkinson-White), progressive disease of conduction system and cardiac hypertrophy by involvement of regulatory subunit gamma-2 (PRKAG2) of AMP- activated by protein kinase (1);
10) Aortic stenosis.

Description of the causes for arrhythmic sudden death in young athletes (average age: 17 years old), secondary to structural heart disease.

1) Gollob MH, et al. Curr Opin Cardiol. 2002;17:229-234.

CAUSES OF ARRHYTHMIC SUDDEN DEATH IN YOUNG ATHLETES (AVERAGE AGE: 17 YEARS)

II) ENTITIES WITHOUT STRUCTURAL HEART DISEASE (2%);

1) Drug abuse, e.g. anabolic agents,
2) Ventricular pre-excitation of the Wolff-Parkinson-White syndrome type, with anomalous pathway of short refractory period, not detected previously;
3) Cardiac concussion or commotio cordis;
4) Channelopathies or primary electrical diseases.

4-A) OF THE SARCOLEMMA OR EXTERNAL CHANNELOPATHIES:

- Congenital long QT syndrome;
- Congenital SQTS
- Brugada syndrome;
- Progressive familial heart block type I; progressive "idiopathic" disease of the His-Purkinje system or Lenègre;
- Genuine idiopathic ventricular fibrillation (GIVF);
- Mixed forms or with overlapped phenotypic aspects:
  - 5a) Brugada disease and variant 3 of congenital LQTS;
  - 5b) Brugada disease and Lenègre disease;
  - 5c) Brugada disease and sinus node dysfunction;
  - 5d) Association of Brugada disease, LQTS and progressive conduction disorder;
- Some sudden unexpected nocturnal death syndromes (SUNDS);
- Some sudden infant death syndromes (SIDS).

4-B) OF THE CHANNELS OF THE ENDOPLASMIC RETICULUM OR INTRACELLULAR CHANNELOPATHIES:

1) Catecholaminergic polymorphic ventricular tachycardia (CPVT).

STRUCTURE OF PHYSIOLOGICAL VENTRICULAR HYPERTROPHY ATHLETE'S HEART

"Possibly, the Differentiation Between Physiological Ventricular Hypertrophy of Athletes and the Pathological One (Ventricular Remodeling) May Become A Challenge".

Difference between physiological ventricular hypertrophy of athletes and the pathological one (ventricular remodeling).

DIFFERENCES BETWEEN PHYSIOLOGICAL VENTRICULAR HYPERTROPHY OF THE ATHLETE AND THE PATHOLOGICAL ONE (VENTRICULAR REMODELING)

	PHYSIOLOGICAL VENTRICULAR HYPERTROPHY	PATHOLOGICAL VENTRICULAR HYPERTROPHY VENTRICULAR REMODELING
Location:	Symmetrical, however, it may be asymmetrical.	Asymmetrical, however, it may be symmetrical.
Relative Ischemia:	Absent.	Present.
Myocitic/Non-myocitic Component Relationship:	Maintained.	Loss of balance in favor of the non-myocitic component (fibrosis).
Energetic Cycle:	Aerobiosis.	Anaerobiosis.
Renin-angiotensin-aldosterone Mechanism	Normal.	Increased.
Norepinephrine	Normal.	Increased.
Atrial Natriuretic Peptide	Normal.	It may be increased.
Pump Function	Normal.	Depressed.
Heart Rate:	Tendency to sinus bradyarrhythmia by vagotony.	Frequent tachycarrhythmia and sympathotony.
LV Pd2:	Normal.	Increased.
Pulmonary Artery Pressure And Central Venous Pressure:	Normal.	It may be increased.
ANS:	Parasympathetic predominance.	Sympathetic predominance.
Curve Of Dissociation of Hb:	Deviation to the right.	Deviation to the left.
Echocardiogram:	Proportional growth between the diameter and the thickness of walls. Normal LA.	Loss of walls thickness/diameter ratio. Increased LA.

DIFFERENCES BETWEEN PHYSIOLOGICAL VENTRICULAR HYPERTROPHY OF ATHLETES AND HYPERTROPHIC CARDIOMYOPATHY (HCM) WHEN BOTH PRESENT WALL THICKNESS BETWEEN 13 mm & 15 mm

The concentric or symmetrical form of HCM (5%), may be confused with the athlete's heart with physiological hypertrophy of its walls, since the increase is not asymmetrical. For the differential diagnosis, the following criteria could be used:

	ATHLETE	**HCM**
Bizarre ECG pattern of LVE	No.	Yes.
LV cavity < 45 mm	No.	Yes.
LV cavity > 55 mm	Yes.	No.
LAE	No.	Yes.
Female Gender	Negative.	Positive.
Decrease Of Hypertrophy With Less Physical Training	Positive.	Negative.
Family History Or Provable Genetic Mutation	Negative.	Positive.

Difference between physiological ventricular hypertrophy in athletes and hypertrophic cardiomyopathy (HCM), when both present wall thickness between 13 mm and 15 mm.

STUDY METHODOLOGY IN DETECTING PATIENTS IN RISK

- ✓ **Periodical Evaluations:** each 2 years.

I) Personal and Family Clinical History

II) Physical Examination: mandatory for any candidate.

III) Non-invasive Supplementary Tests: (only in selected cases)

Study methodology for screening candidates to athletes in risk.

I) PERSONAL AND FAMILIAL CLINICAL HISTORY

1) Ask questions about SD in first-degree relatives under 45 years old;
2) Ask questions about the knowledge in the family about HCM, LQTS, Marfan-type somatic habit, sindactily, etc;
3) Personal history of murmur in childhood;
4) Personal history of dizziness, syncope, palpitations, intolerance to exercise, precordialgia, dyspnea, etc.
5) Dizziness or syncope during or after exercise, may indicate the presence of: HCM, dromotropic disorder, MVP, aortic stenosis or arrhythmia;
6) Precordialgia intra- or post-strain may indicate early coronary atherosclerosis;
7) Excessive/progressive dyspnea may indicate valvular diseases, pulmonary disease, or structural anomalies;
8) Palpitations during or after exercise may be a sign of arrhythmia.
9) Ask questions about the current or past use of legal (tobacco, alcohol) and illegal drugs;
10) Recent history of virus infection may lead to symptoms compatible to myocarditis;
11) History of congenital heart disease or cardiac surgery;
12) Any background that may imply greater risk for congenital heart disease. E.g.: Maternal rubella, exposition to toxics used or environmental.

Significance of personal and familial clinical history.

II) PRE-PARTICIPATION PHYSICAL EXAMINATION

1) Anthropometrical evaluation: weight, height, BP and percentage of body fat;
2) Identification and characterization (intensity, location and time of cycle) of murmurs and arrhythmias, standing and in supine position;
3) Recognize phenotypes: e.g. Marfand, Noonan and Holt-Oram syndrome, supravalvular aortic stenosis, Williams syndrome.
4) Measurement of BP in superior and inferior limbs, and assessment of femoral, radial and foot pulses to exclude Aorta coarctation.
5) Auscultation must be performed in decubitus and standing to identify murmurs influenced by dynamic obstruction in the LV outflow tract; detection of extracardiac clicks and sounds;
6) Musculo-skeletal aptitude. Try to detect medical conditions or skeletal muscles that may predispose to injuries or diseases during a competition.

Value of physical examination.

III) NON-INVASIVE METHODS IN PRE-PARTICIPATION IN YOUNG CANDIDATES TO ATHLETES

Although they may increase the diagnostic power of history and physical examination, they are not advised as a routine "screening" ;

- ✓ ECG
- ✓ Echocardiogram Echocardiogram M-module, two-dimensional with transthoracic doppler.
- ✓ Three-dimensional echocardiogram.
- ✓ Transesophageal or biplanar echocardiogram.
- ✓ Ergometer test; it is an electric recording of the heart while the organism is undergoing physical stress. The most used ergometers are the bicycle ergometer and the treadmill. It should be performed in all preventive medicine check-ups, but regrettably, it is not so. But it is convenient for all athletes or at least sportsmen with a regular activity, to do this test. Only thus it is possible to know how the heart behaves regarding physical strain. In sedentary people older than 30 years old, who begin a program of exercises, it should be mandatory. It is known that 70% of people with chest angina and who hadn't experienced myocardial infarction, present rest ECG without abnormalities, and that many myocardial ischemias appear in a strain test even before the person has displayed any symptom.
- ✓ Ergometer test with spirometery.
- ✓ Transesophageal cardiostimulation,
- ✓ Tilt-Test,
- ✓ Looper,
- ✓ RR variability,
- ✓ QT dispersion
- ✓ Microvolt T wave alternans, etc.
- ✓ High resolution ECG.
- ✓ Holter monitoring.
- ✓ RR variability.
- ✓ Microvolt T wave alternans
- ✓ Nuclear Magnetic Resonance.
- ✓ Ultra-fast computed tomography.
- ✓ Radioisotopic ventriculography

INVASIVE SUPPLEMENTARY TESTS

- Programmed electrophysiological stimulation.
- Cineangiography, coronary angiography, and ventricular angiography.
- Endomyocardial biopsy.

Invasive tests.