

**RELATIVELY NARROW SUSTAINED TACHYCARDIA  
ASSOCIATED WITH CORONARY HEART DISEASE AND  
NO REVERSION WITH PHARMACOLOGICAL  
APPROACH**

**TAQUIARRITMIA DE QRS RELATIVAMENTE  
ESTREITO RESISTENTE A FÁRMACOS ASSOCIADA A  
DOENÇA CORONÁRIANA**

**From Raimundo Barbosa Barros MD  
Coronary Center Hospital de Messejana Dr. Carlos Alberto Studart Gomes  
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**Comments**

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

**ABC Faculty- ABC Foundation - Cardiology discipline - Santo André - SP - Brazil**

**Paciente J. L., 57 anos, masc., admitido em 09/02/12 a noite, com história de tontura. Apresentava história de ATC com stent para CD há 6 dias. Segundo relatado no prontuário, paciente encontrava-se estável hemodinamicamente.**

**Conduta na SPCR:**

-Adenosina 6-12-12mg

-Amiodarona 300mg

**Paciente começou, então, a evoluir com instabilidade... Sedado com propofol, mas antes da CVE o paciente saiu da taquiarritmia.**

**Evoluiu estável, sem queixas, porém no outro dia apresentou dor precordial e congestão...**

**Encaminhado à hemodinâmica ( TCE e DA sem lesões; MgCx 70% proximal; Stent em CD pérvio)**

I would like to listen to the opinions of the forum in this case.

Male patient, J.L.F., 57 years old, was admitted on the night of February 9th, 2012, complaining of dizziness. He had undergone angioplasty with stent implantation for the right coronary artery 6 days before (hemodynamically stable).

The following management was applied: IV Adenosine-6 12-12 mg, unsuccessful; next IV amiodarone 300 mg, also with no reversion. The patient started evolving with instability. He was sedated with propofol, to conduct cardioversion, but before the electrical procedure, the event reversed spontaneously. He had a stable evolution, with no complaints, until in just

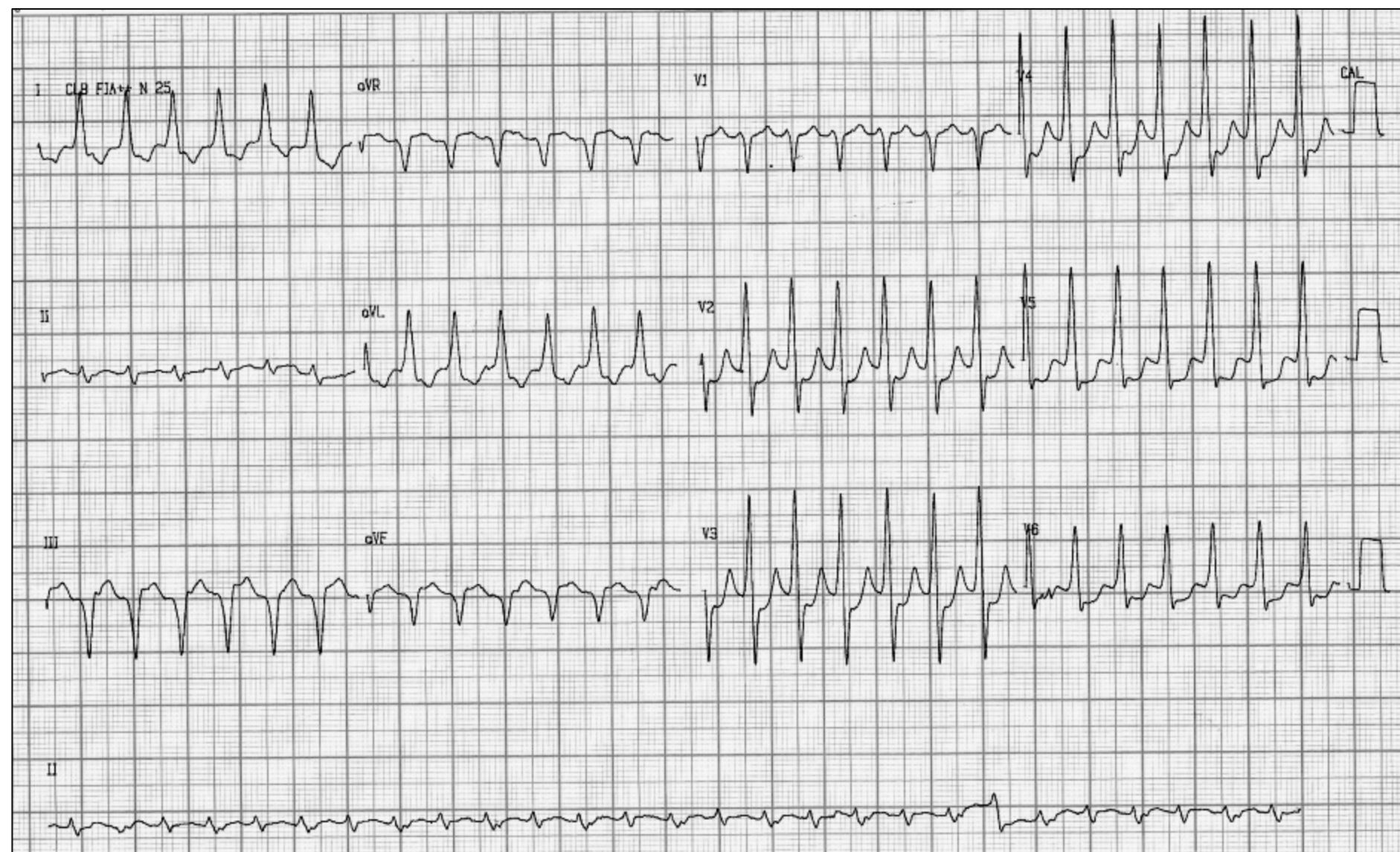
24 hs, he presented symptoms of precordial pain associated to pulmonary congestion. He was submitted to the hemodynamics lab; the new left heart catheterization showed ADA with no lesions, Marginal of the Circumflex artery with 70% of proximal obstruction and previous stent in the RCA.

What is the diagnosis of the arrhythmia and the management to follow?

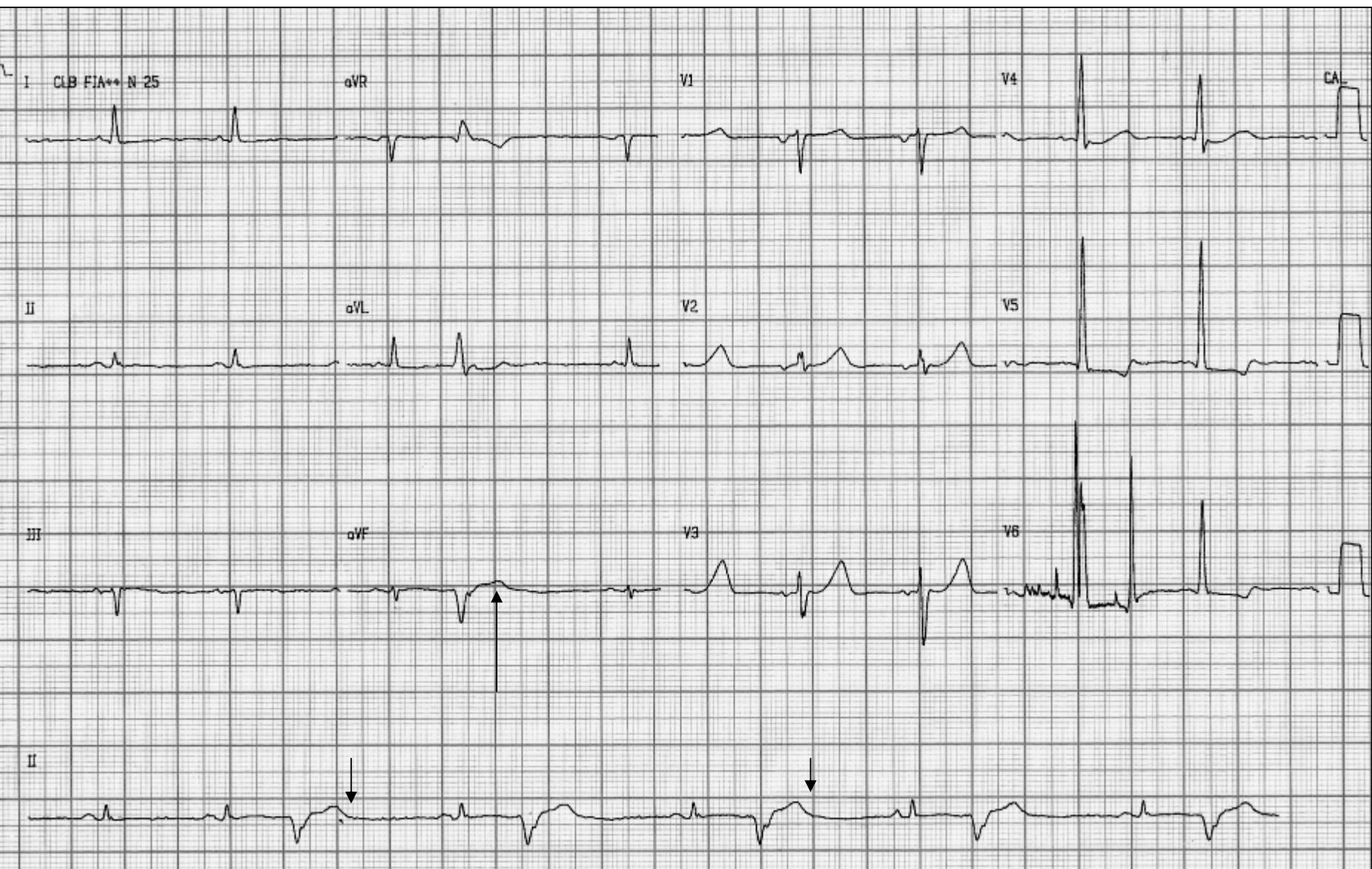
Warm regards,

Raimundo Barbosa Barros MD

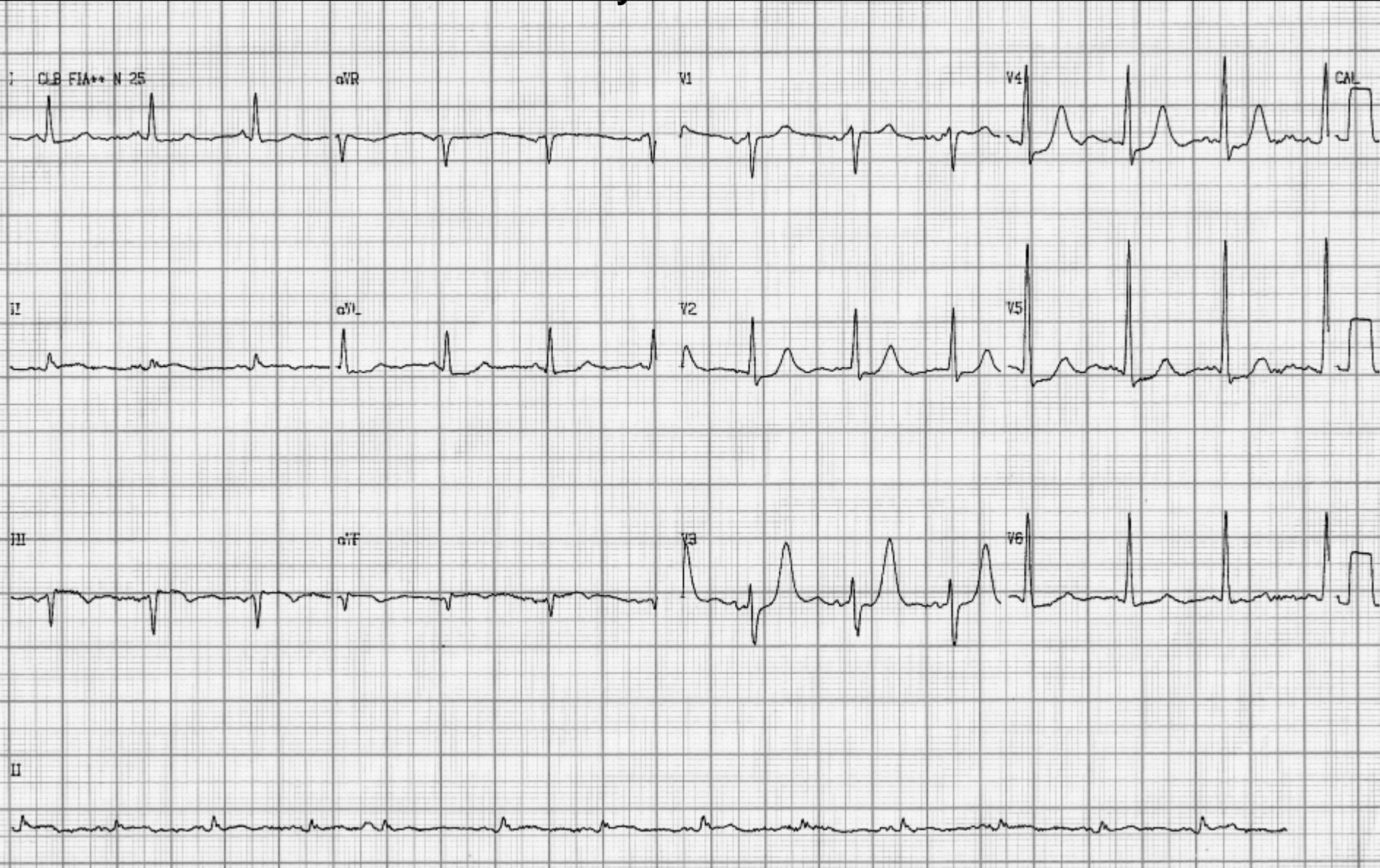
# Admission February 02 2012



# After reversion February 02 2012



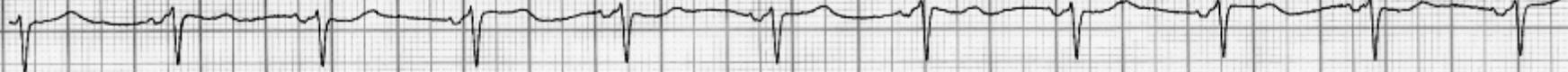
**February 10 2012**



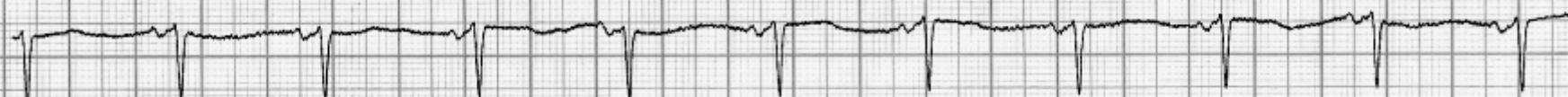
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V1 CLB F1a++ N 25

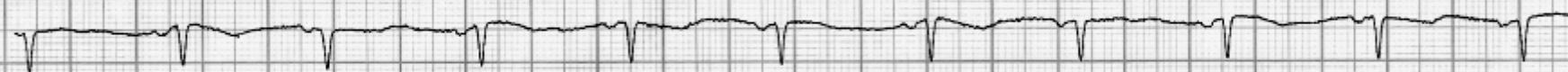
V2 R



V2 V3 R

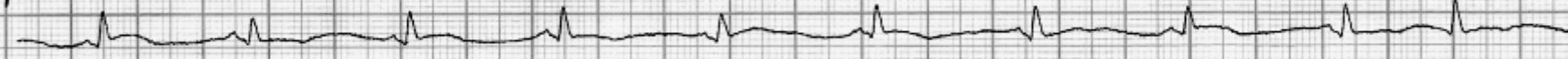


V3 V4 R

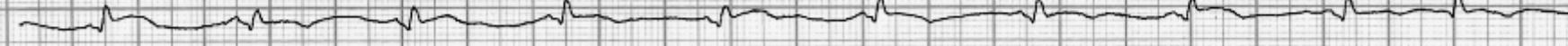


V4 CLB F1a++ N 25

V7



V8



V9



# Colleagues opinions

**Prezado Barbosa:**

A arritmia do ECG inicial me parece Taquicardia por Reentrada Nodal. Acho que os quadros são de origem isquêmica.

O ECG 3 chama atenção por aumento de R e T alta e simétrica (espelho) V2-V4 do chamado antigamente infarto estreito dorsal - (Infero-basal conforme Prof Bayés de Luna) - qR e ST supra de V7-9

Suponho placa 'vulnerável' esta de 70% na Mg1 - Ultra--som intra-coronária confirmaria mas já tenho a clínica - Angioplastia e stent nela.

Abraços - Até agosto. se Deus quiser.

Adail - Bahia - Brasil

**Dear Barbosa:**

The initial ECG arrhythmia seem nodal reentrant tachycardia. I think the pictures are of ischemic origin.

The ECG 3 calls for increased voltage of R and T and symmetrical (mirror) V2-V4 once in the past known strictly dorsal infarction - (inferobasal as Prof Bayes de Luna new nomenclature) - qR and ST above the V7-9

I suppose 'vulnerable' is 70% obstruction in first Mg- intra-coronary Ultra - sound confirm but I already have the clinic - Angioplasty and stenting in it.

Hugs - Until August. God willing.

Adail - Bahia - Brazil

Dear friends:

I will not enter into the possible pathophysiologic mechanism of the arrhythmia but only give my presumptive diagnosis of the tachycardia.

This may surprise you but I am strongly convinced that we are dealing with VENTRICULAR TACHYCARDIA.

I do know that the QRS complexes do not look very wide but it reaches 120msec (see in precordial leads).

Also note the well visible 2:1 VA block on the continuous lead II tracing (at the bottom of fig 1). Of course the very exceptionally rare possibility of AVNRT with 2:1 block should be discussed but I do not think we can explain it taking in account the QRS complex that is unlikely to be consistent with any conduction disturbance.

VT most probably originates from the LV-posterior-inferior area

The last tracing shows suspected acute postero-inferior MI.

I have two questions:

1) Did the patient suffer an MI in the past?

2) Did echocardiogram show any localized LV abnormality at the time VT was documented?

Warmest regards

Professor Bernard Belhassen

Dear Professor BB: thank very much for your valuable and intelligent opinion.

Please, and what about the approach?

Thank in advance

Andrés.

## Spanish

Queridos amigos:

No voy a entrar en el posible mecanismo fisiopatológico de la arritmia, sólo daré mi diagnóstico presuntivo de la taquicardia. Esta opinión puede sorprender, pero estoy firmemente convencido de que se trata de TAQUICARDIA VENTRICULAR. Yo sé que los complejos QRS no son anchos, no obstante casi alcanzan 120 msec (ver en las derivaciones precordiales). También tengan en cuenta el bien visible bloqueo 2:1 VA en la derivación II continua (en la parte inferior de la figura 1). Por supuesto, existe la posibilidad muy excepcionalmente rara de AVNRT con bloqueo 2:1 que se debe discutir, pero no creo que se pueda explicar teniendo en cuenta que el complejo QRS que es poco probable que sea compatible con cualquier alteración de la conducción.

TV probablemente originaria de la zona póstero-inferior del VI

Los últimos trazados tienen sospecha de infarto agudo póstero-inferior.

Tengo dos preguntas:

1) ¿El paciente sufrió un infarto de miocardio en el pasado?

2) ¿ El ecocardiograma no mostró algun movimiento anormal localizado en el momento que la TV fue documentada?.

Saludos cordiales

Professor Bernard Belhassen

## Portuguese

Caros Amigos,

Não vou entrar no possível mecanismo fisiopatológico da arritmia, mas apenas darei o meu diagnóstico presuntivo de taquicardia. Esta minha visão pode surpreendê-lo, mas acredito firmemente que esta é uma taquicardia ventricular. Eu sei que os complexos QRS alargados não são ainda quase chegou a 120 ms (ver nas derivações precordiais). Além disso, note o bloco 02:01 visível em chumbo II VA continuamente (na parte inferior da Figura 1). Claro, as chances são muito excepcionalmente AVNRT rara, com bloqueio 2:1 que deve ser discutido, mas eu não acho que pode ser explicado considerando que o complexo QRS é improvável que seja compatível com qualquer distúrbio de condução.

TV provavelmente se originou na área póstero-inferior do VE.

Os últimos traçados tem suspeita de infarto agudo infero-dorsal.

Tenho duas perguntas:

1) O paciente sofreu um infarto do miocárdio no passado?

2) é o ecocardiograma não mostrou qualquer movimento anormal localizado no tempo que a TV foi documentada?

Professor Bernard Belhassen

Queridos amigos , analizare los trazados interesante que transmitieron nuestros queridos amigos Dr Raimundo Barbosa Barros y el Profesor Andres Ricardo Perez Riera PhD

Estoy de acuerdo con el analisis mi amigo Prof Bernard Belhasan,que la taquicardia es de origen ventricular, se observa bien la conduccion retrógrada ,pegada al QRS

La primera pregunta es; tiene esta taquicardia ventricular alguna relacion con el episodio isquómico ? Y aqui esta la dificultad !! Poque los infartos posteriores desvian el eje muy a la izquierda con QS en DII , DIII, aVF , con morfologia de rama derecha si se originan en el vent izquierdo ,(como la extrasistole en segundo trazado con 12 derivaciones )o con desviacion extrema a la izquierda y morfologia de bloqueo de rama izquierda ,si se originan en el vent derecho , que suele ocurrir en las obstrucciones de CD

Pero esta taquicardia ,no tiene ninguna de estas caracteristicas. Entonces en donde se origina? Pareciera que de un area muy alta del ventriculo antes de las divisiones de las vias de conduccion intraventricular , y es muy temprano que un infarto induzca a taquicardia vent de reentrada , pero puede ocurrir ,no es lo corriente

El ECG es tambien interesante , muestra un infarto inferior, muy probable de la arteria derecha , pero sin reperfusión miocardiaca , todavia el ST esta elevado en DIII , con inversion de la onda T( reperfusión incompleta), pero DII y aVF ,que expresan la cara posterior alta ,igual que la elevación del ST y onda T positiva en v8 ,v9 . indican que esta cara no tiene signo de reperfusión myocardica

Me parece que el 70% de obstrucción CX ,no tuvo influencia en el proceso isquémico

Que recomiendo en estos casos? mantener al paciente en cuidados cardiológicos, (intermedio) hasta que aparezcan los signos de reperfusión miocárdica completa ,que puede llevar entre 7 a 12 días de la apertura de la arteria Como se vera esto? Cuando el ST en las derivaciones inferoposteriores se hagan isoelectricas y las ondas T se inviertan

Un fraternal abrazo a todos los amigos del forum

Samuel Sclarovsky

Dear Andres,

Thanks for sending the interesting case. The tachycardia is likely VT see the A/V dissociation in lead aVL and possible fusion complexes in rhythm strip of Lead 2 as well in the transition to aVR,aVI (? Artifact). The site of origin is the posterior base of LV septum or possibly breakout on right ventricular septum.

Would suggest Echo and if ablation is contemplated an MRI to define the scar area.

The serial ECGs show an evolving posterior inferior MI without strong evidence of RV involvement.

I would be inclined to do EP study and ablate considering the drug resistance. Alternatively, one could insert a Defibrillator and try full loading doses with Amiodarone. Let me know what you did.

Melvin M Scheinman

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Estimado Andrés,

Gracias por enviar este caso interesante. La taquicardia es probable que sea una TV: ver la disociación A / V en la derivación aVL y posibles de fusión en la tira de D2 largo, así como en la transición a aVR, aVL (Artefacto?).

El sitio de origen es la base posterior del tabique del VI o, posiblemente, irrumpir del septo del ventrículo derecho. Sugeriría realizar un ecocardiograma o una resonancia magnética caso se piense en ablación para definir la zona de la cicatriz.

Los ECG seriados muestran una evolución de infarto agudo posteroinferior sin una fuerte evidencia de la participación del ventrículo derecho.

Yo estaría inclinado a hacer un estudio electrofisiológico y la ablación teniendo en cuenta la resistencia a los medicamentos observada.

Alternativamente, se puede implantar un CDI y tratar con dosis plenas de carga con amiodarona.

Déjame saber lo que hiciste.

Melvin

Dear Professor Pérez-Riera

Greetings I am sorry of being late to respond. I was in the International Winter Arrhythmia School in Collingwood, near Toronto. I was lucky to meet Dr. Baranchuk there. It was a great opportunity!!! When I came back to Ottawa last night, I found > 120 e-mails waiting!!!!

Anyway, regarding this case . . . Interestingly, the QRS is relatively narrow, however, it is not like the sinus rhythm. None response to repeated shots of Adenosine, suggests VT [if were given appropriately]. Furthermore, there is more Vs than As. I think this VT is associated with 2:1 VA block and it is most clear in the inferior leads.

The P waves are inverted, which suggests coming from the lower atrium and it is not sinus. It will be great if we can see the effects of the Adenosine on these Ps, because, this may not affect the VT but can affect the VA conduction. Having non-obstructive lesions in the coronary angiography, does not exclude ischemia. However, acute ischemic is rarely associated with SMVT. This is usually scar related VT. I think this patient will benefit from functional study by SPECT thallium scan. This will help to find if there is ischemia or if there is a scar in the RCA territories. It looks the VT is coming from the baso-inferior area. aVL lead is positive, which suggests close to the septum. I think this is close to the normal conduction system and this is the cause of the relatively narrow QRS.

Electrophysiologic study will be very helpful and can put a strategy for management. All my approach was based on being a VT, however, this could be an SVT, but extremely rare to see an AVNRT with upper common pathway block.

Finally, what is this patients LV function and LVEF?

Warmest regards.

Raed Abu Sham'a MD

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***rabushama@ottawaheart.ca***

***Skype name: raedabushama***

Querido Prof. Pérez-Riera

Saludos

Lo siento por responder tarde. Yo estaba en la Escuela Internacional de Invierno de arritmias en Collingwood, cerca de Toronto. Tuve la suerte de conocer al Dr. Baranchuk allí. Fue una gran oportunidad! Cuando volví a Ottawa ayer por la noche, me encontré con más de 120 e-mails esperando!!

En relación con este caso. . . el QRS es relativamente estrecho, sin embargo, creo que es una TV que se asocia con bloqueo 2:1 VA y es más evidente en las derivaciones inferiores.

Las ondas P están invertidas, lo que sugiere que vienen de la parte inferior de la aurícula y no son sinusales. Será genial si podemos ver los efectos de la adenosina porque esta droga no afecta a la TV, pero puede afectar a la conducción AV.

Tener no lesiones obstructivas en la angiografía coronaria, no excluye la isquemia. Sin embargo, isquémico agudo se asocia raramente con SMVT.

Esto es generalmente VT cicatriz-relacionada.

Creo que este paciente se beneficiaría con un estudio funcional mediante SPECT con talio. Esto ayudará a encontrar si hay isquemia o si hay una cicatriz en el territorio RCA.

Se ve que TV viene de la zona baso-inferior. aVL es positivo, lo que sugiere que el foco está cerca del tabique. Creo que está cerca del sistema de conducción normal siendo esta es la causa de la relativamente estrecho complejo QRS.

El estudio electrofisiológico será muy útil y puede ser una estrategia. Todo mi enfoque se basa en ser un VT, sin embargo, esto podría ser una SVT, pero muy raro ver una AVNRT con conducción superior común

Por último,

¿Cuál es la función del VI y la fracción de eyección del paciente?

Raed Abu Sham'a MD

Dear Raed

It was a real pleasure to meet you in Collingwood and to learn you are developing a magnificent EP career in the OHI.

I am happy to report to our mutual forum friends on your successful fellowship.

I do insist in having you and your family in Kingston this summer for an argentinean barbecue.

All the best

(I agree, it looks VT)

Adrian Baranchuk

Andrés Pérez Riera Addendum: Asado (Spanish) is a term used both for a range of barbecue techniques and the social event of having or attending a barbecue in Argentina, Chile, Paraguay, southern Brazil, and Uruguay. In the aforesaid areas, asado is a traditional dish and also the standard word for barbecue. An asado usually consists of beef alongside various other meats, which are cooked on a grill, called a *parrilla*, or open fire.



Asado uruguayo

Shortribs photo("tirita de asado")



Dear Little Adrian (Adriancito)

Do you think you will be able to give to Raed some chinchulines, morcillas and zochori?

Raed: you should come to Buenos Aires and not to Kingston to eat a true barbecue!

Ask to Adrian what is a argentinean zochori!!

Kind regards - Abrazo

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**Adriancito**

**¿Vos crees que podrás darle a Raed algunos chinchulines, morcillas y zochoris?**

**Raed: ¡deberías a Buenos Aires y no a Kindston para comer un vedadero asado argentino!**

**Preguntale a Adrian que es un zochori argentino**

**Abrazo**

**Edgardo**

Dear Edgardo

You should ask Pancho, Samuel and the other friends from all around the world, about the Argentinean barbecue that I made for them at home for the ICE 2011 in June!!!!

There was: tira, zochori, morcilla, molleja (sweet breads), pork, chicken, vacio, chinchulines (tiernizados en leche) and vacio.

Do you think this is enough?

Have you heard about Alberta beef before?

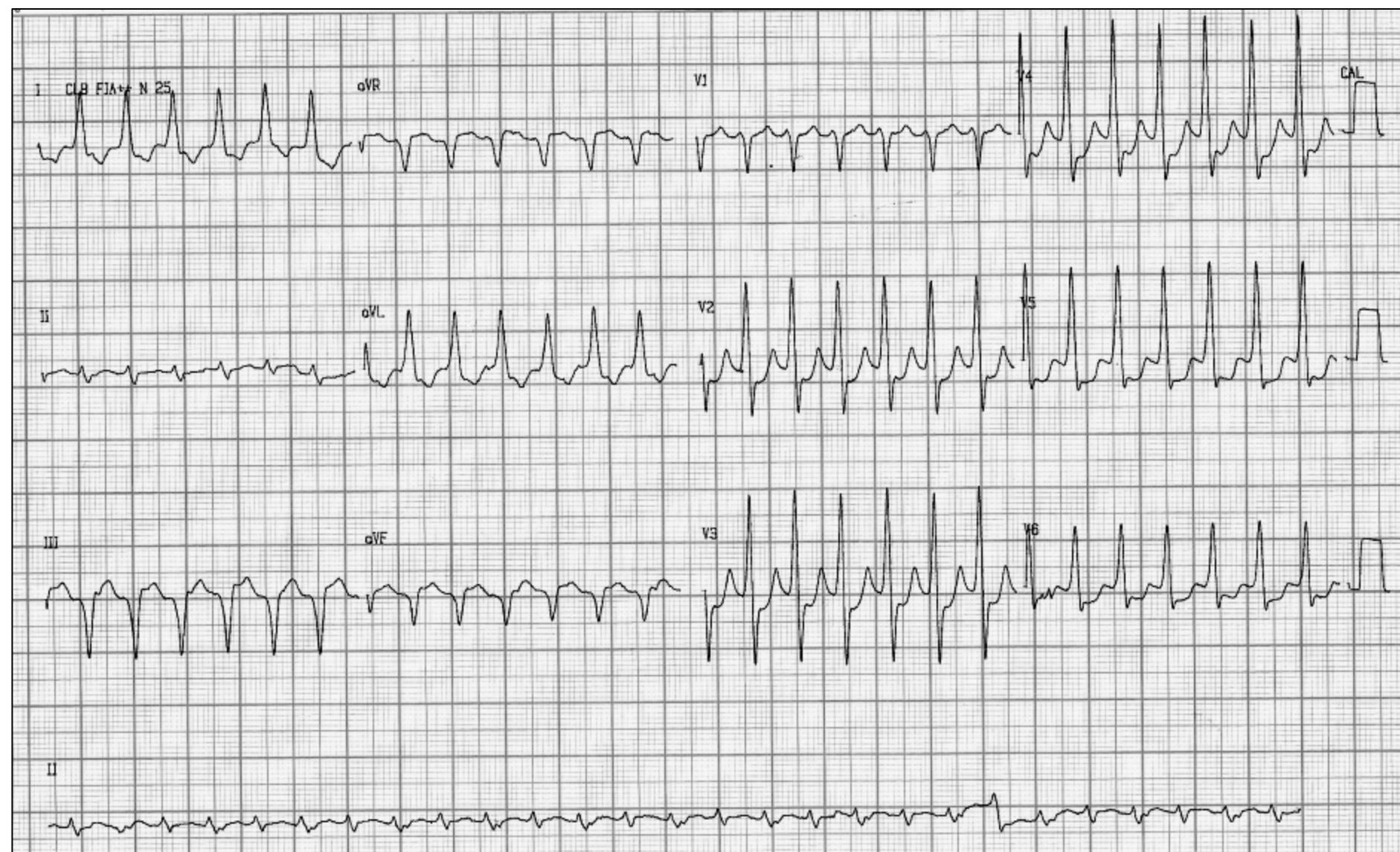
No, you don't, so please do not judge my argentinean barbecue before you experience it.

For more info: go and google: Baranchuk AND argentinean asado. You will notice that my fame as a chef was captured by a relevant food network from Edmonton, Canada.

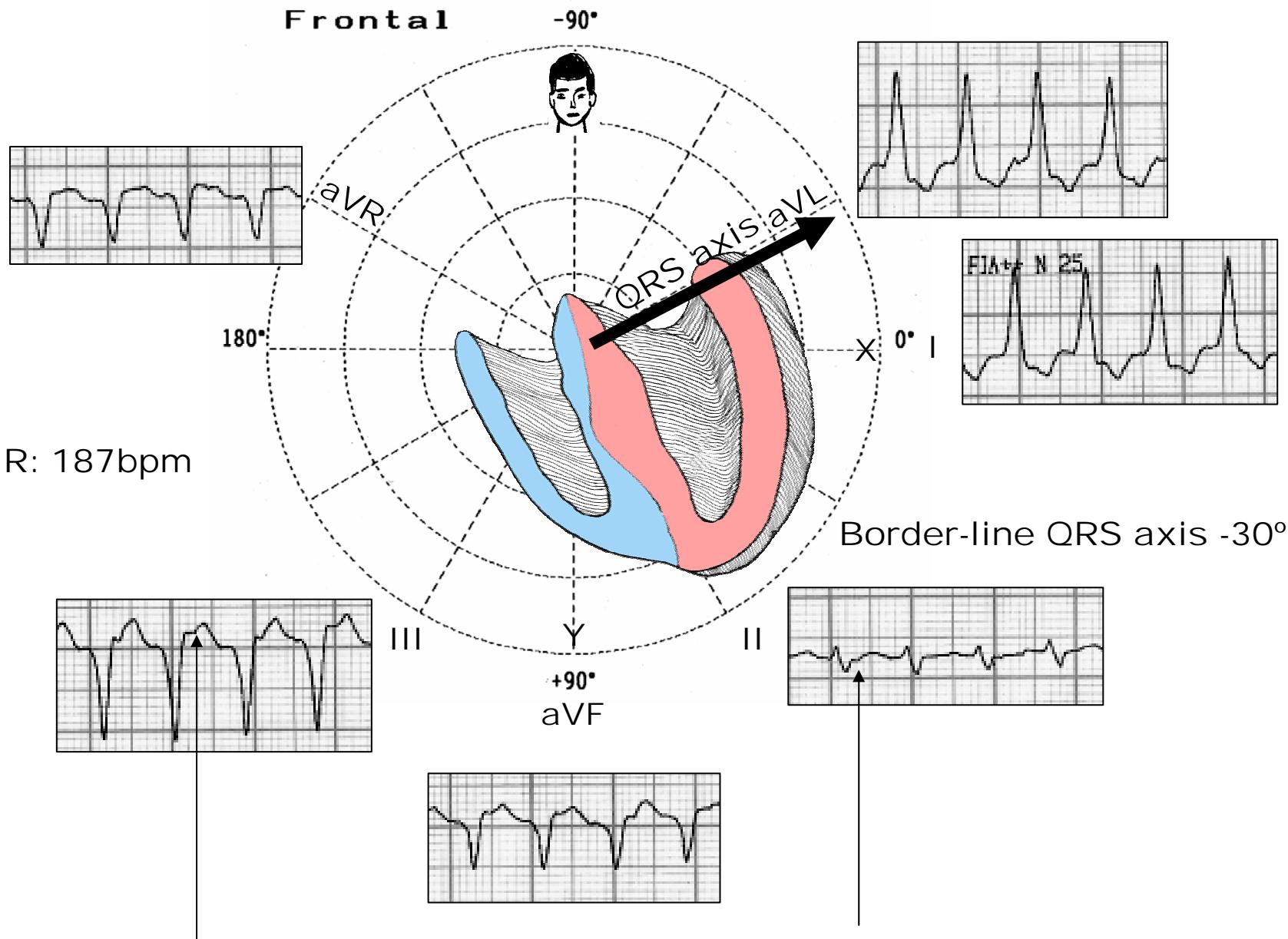
BestAB

# Final comments

# Admission February 02 2012

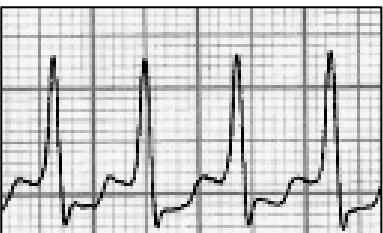
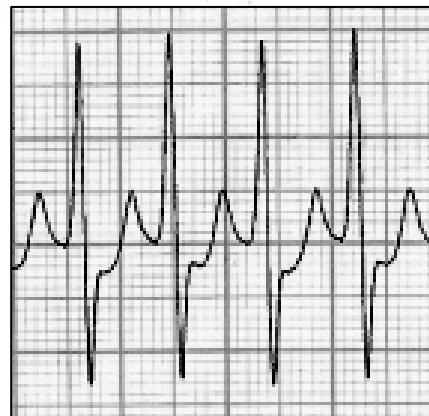
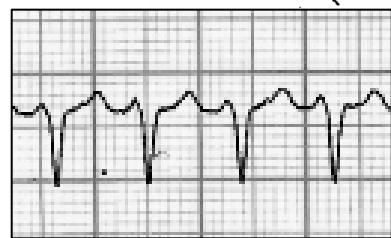
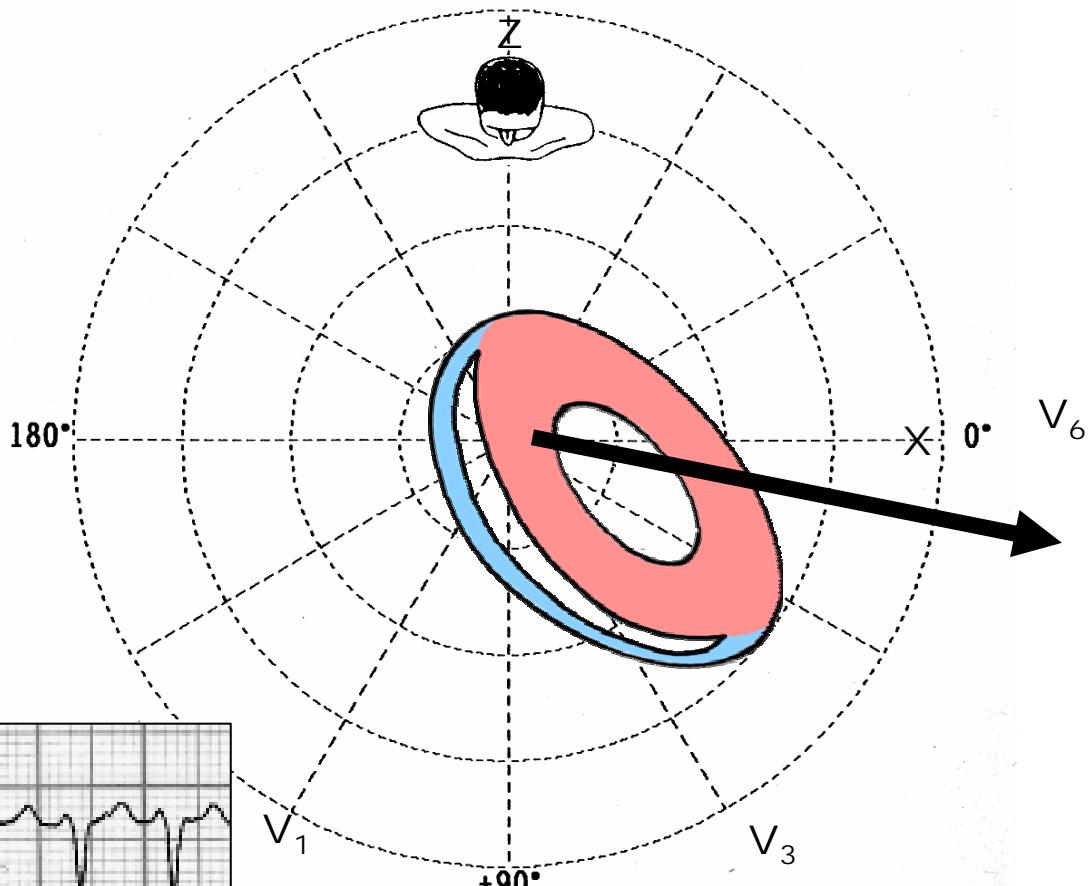


aVL lead is positive, which suggests close to the septum.



The P waves are inverted, which suggests coming from the lower atrium

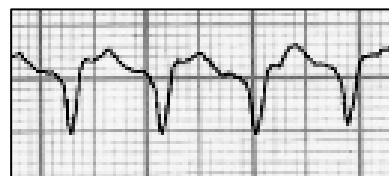
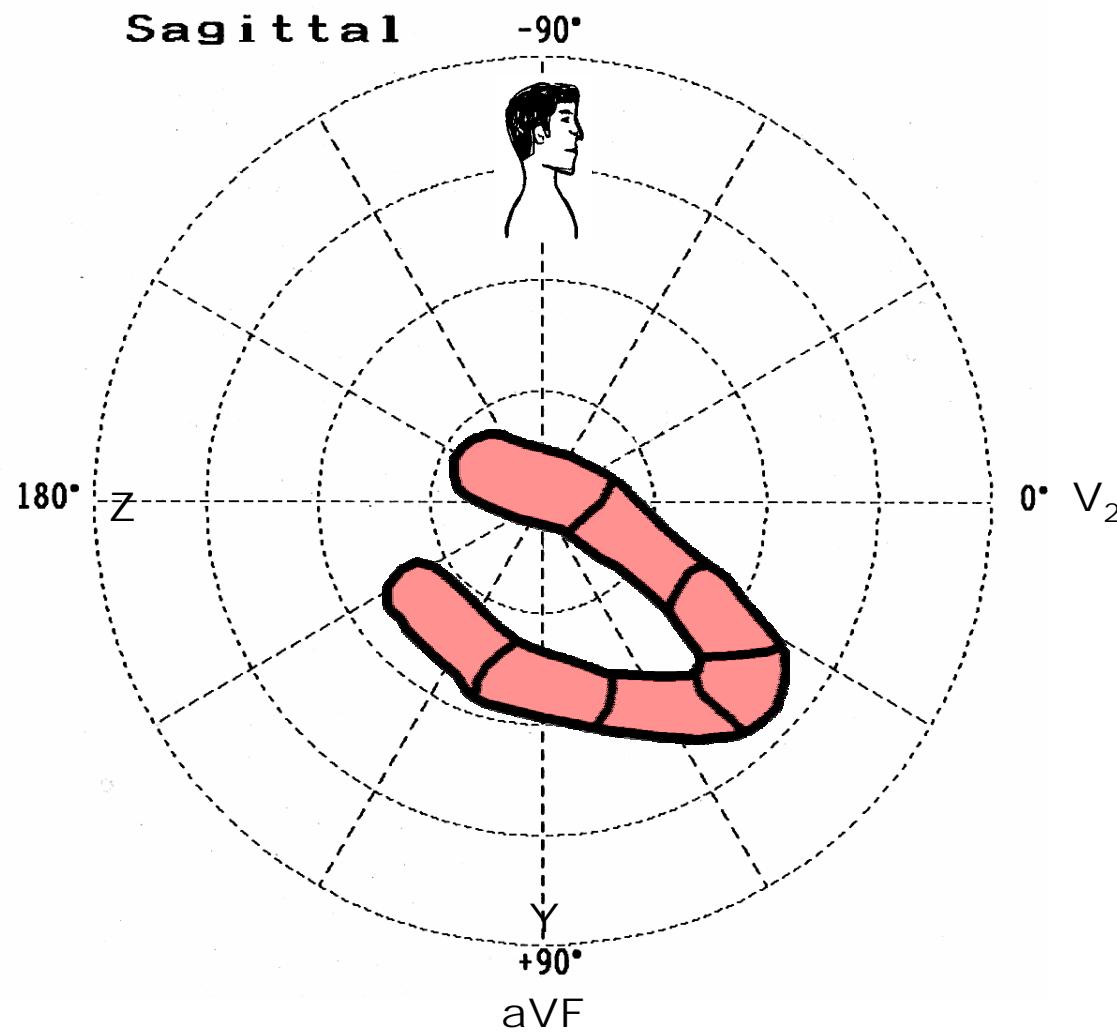
**Horizontal -90°**



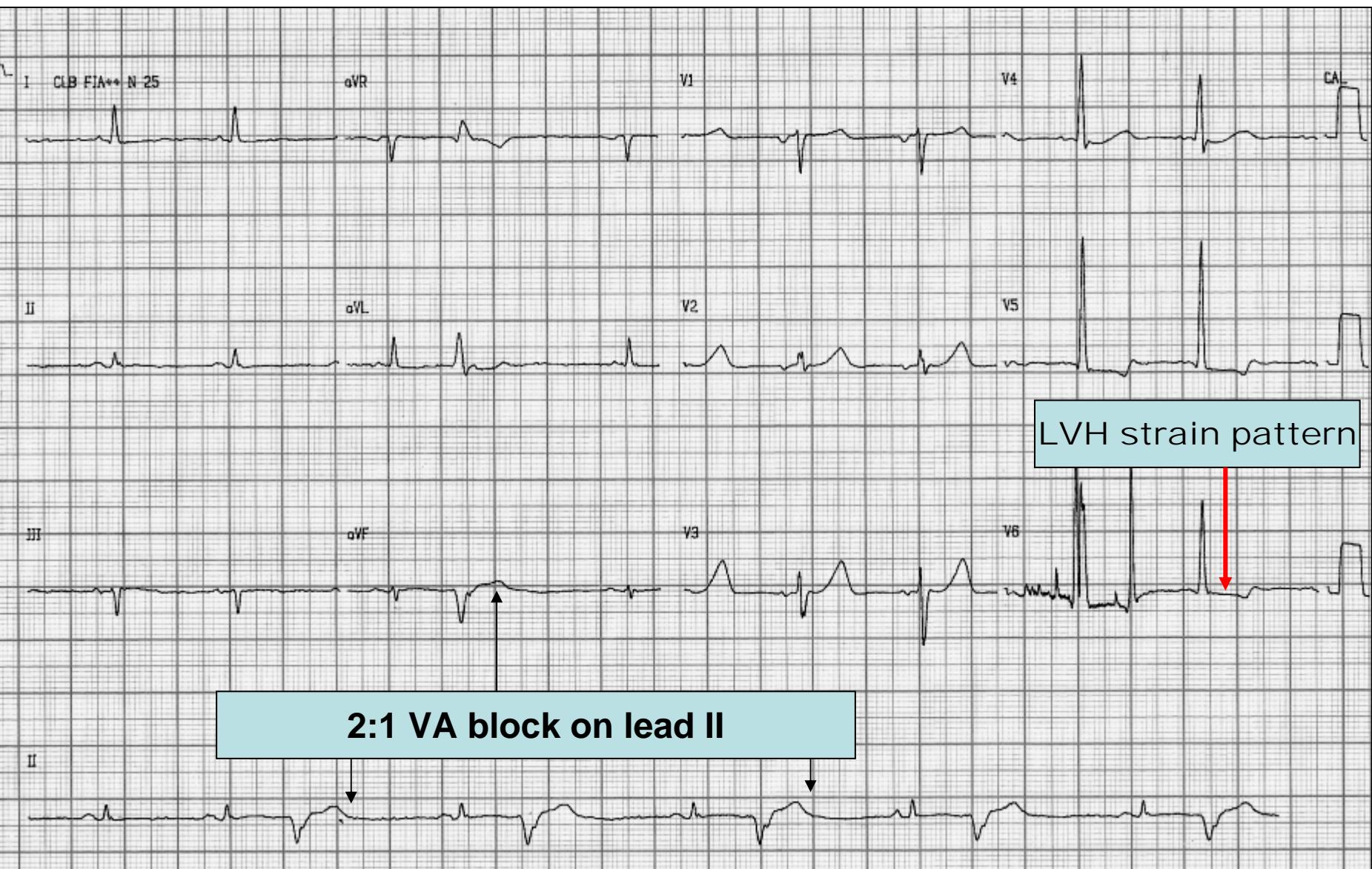


VT with  
narrow  
QRS  
complex  
of near  
120 ms.

QRS duration 122ms: VT that look like  
supraventricular tachycardia

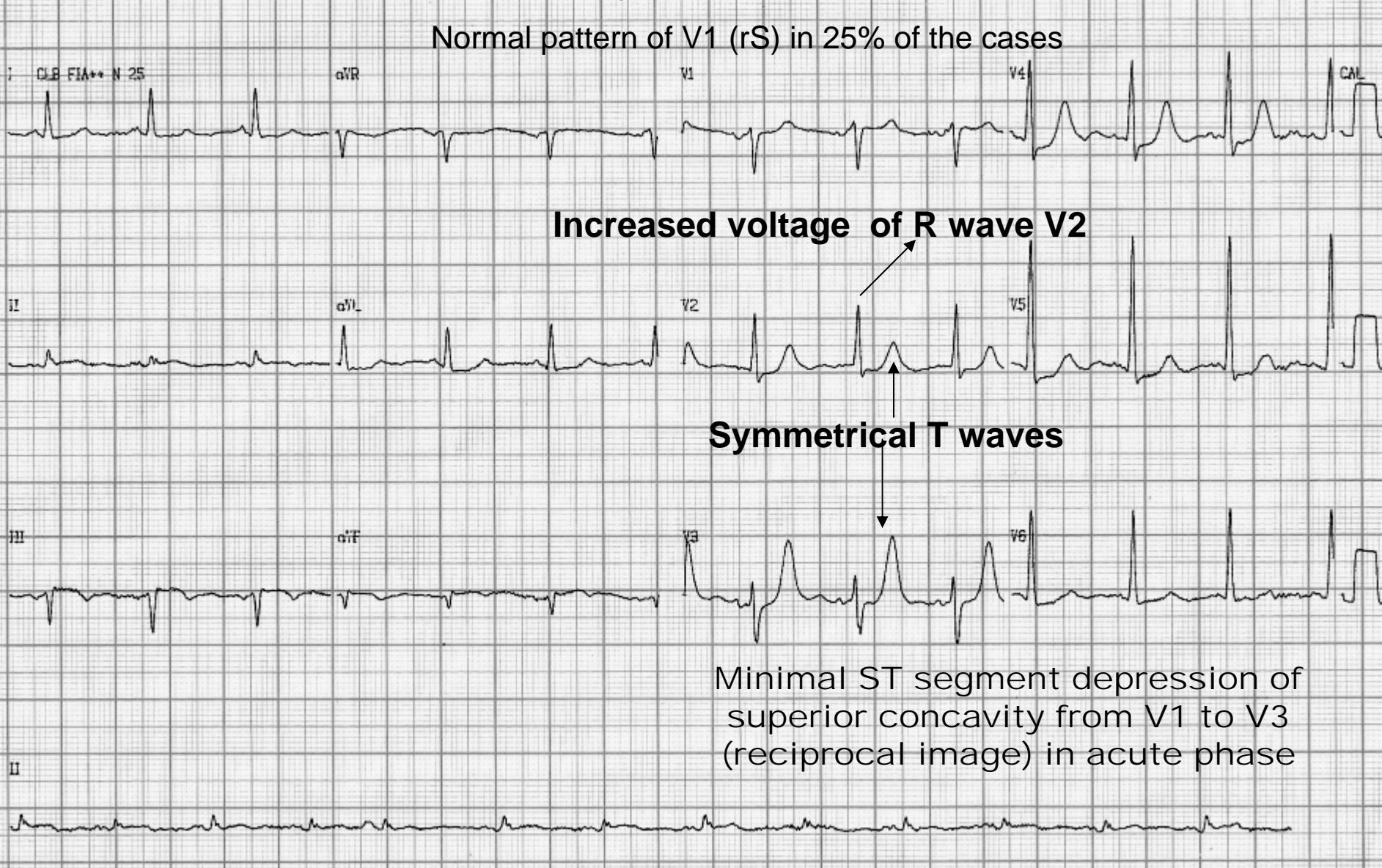


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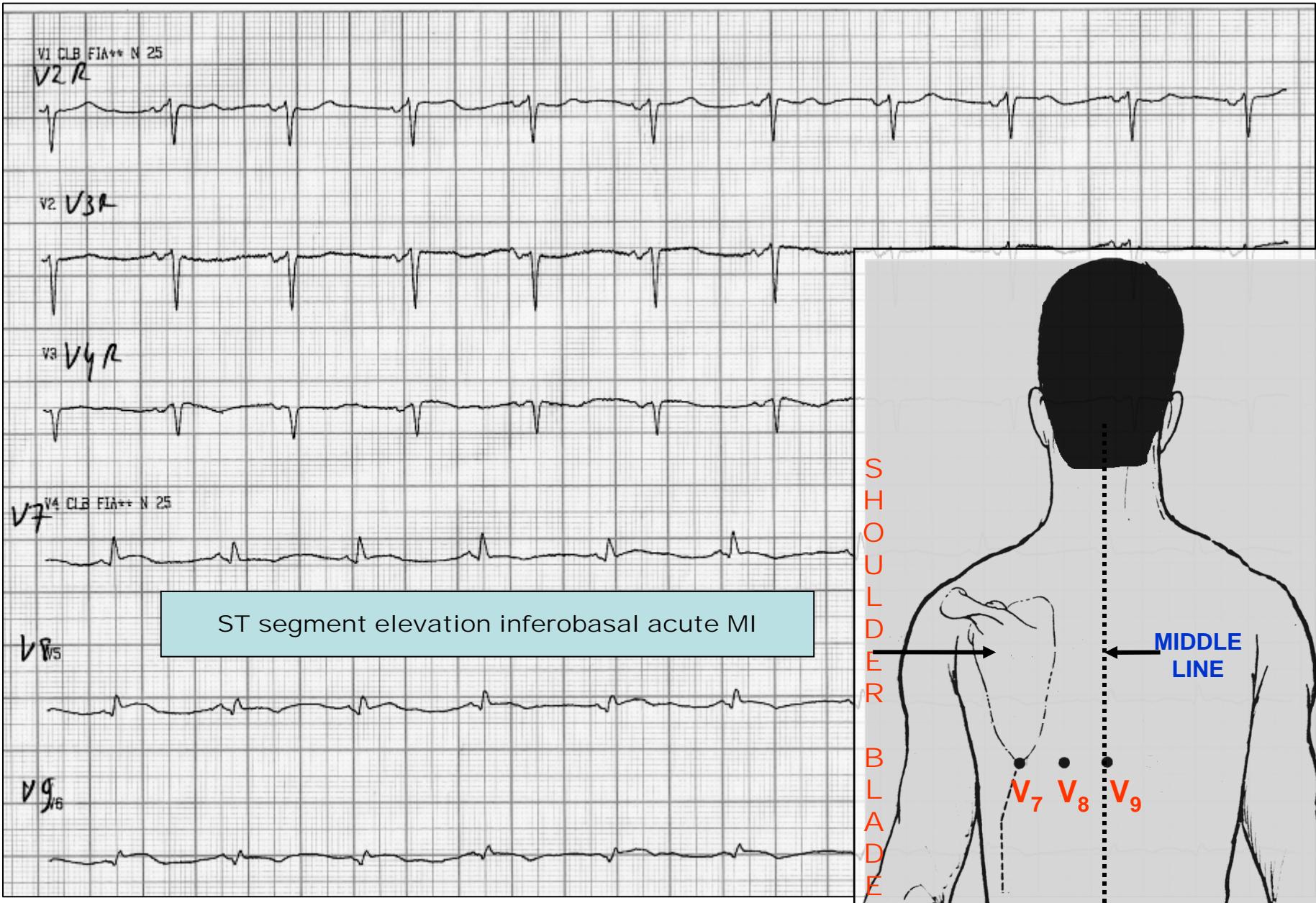


**February 10 2012**

Normal pattern of V1 (rS) in 25% of the cases

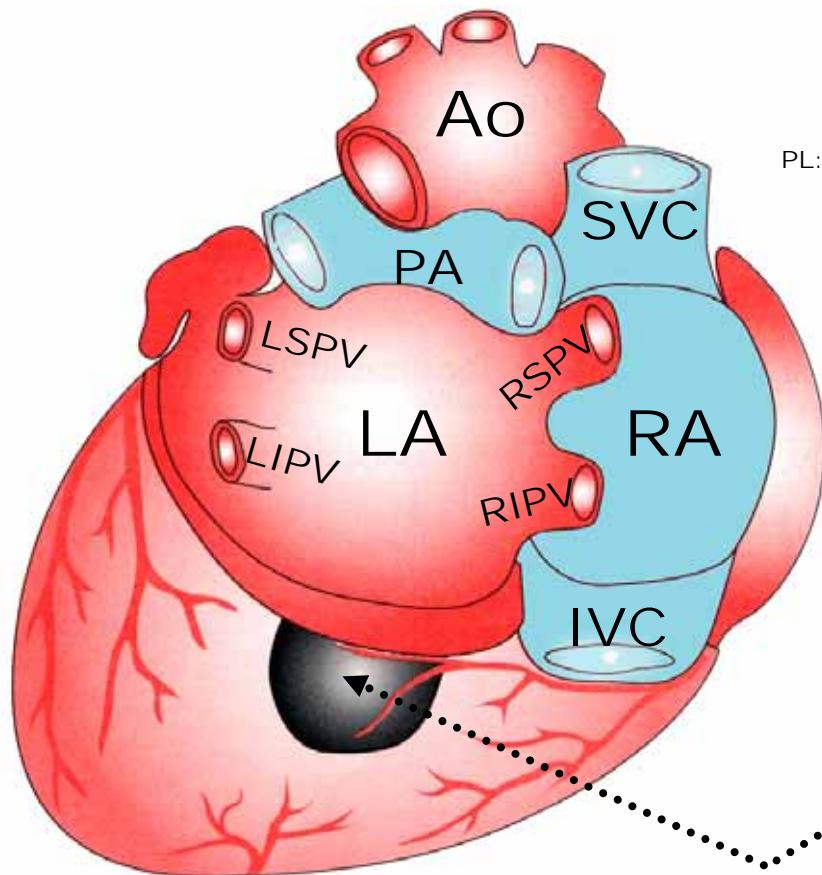


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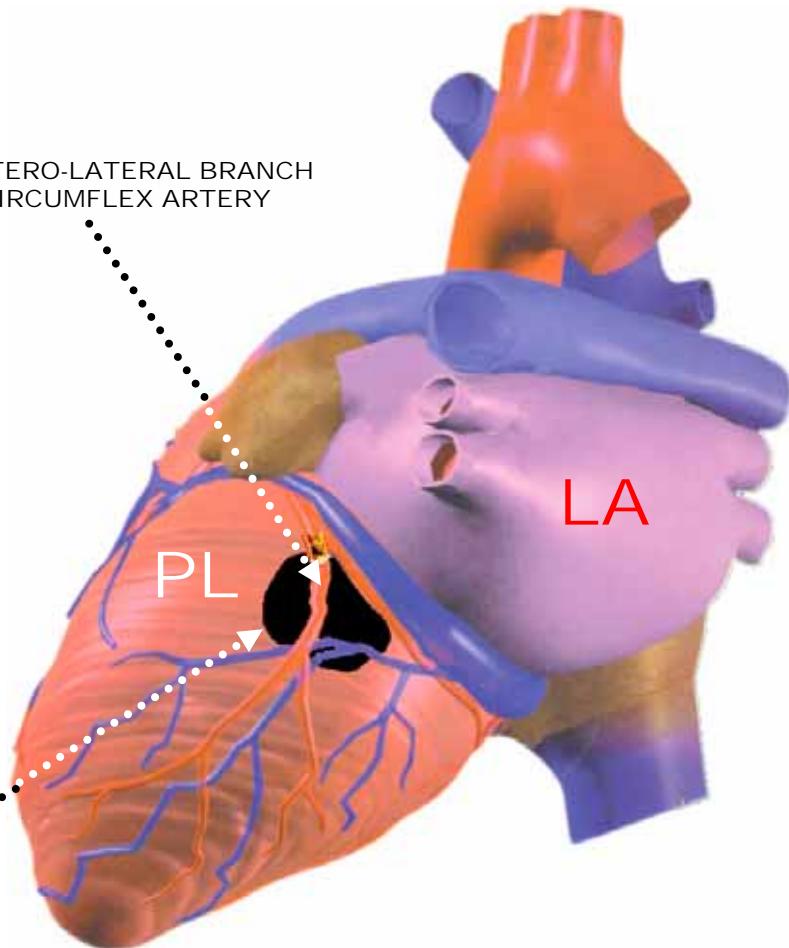


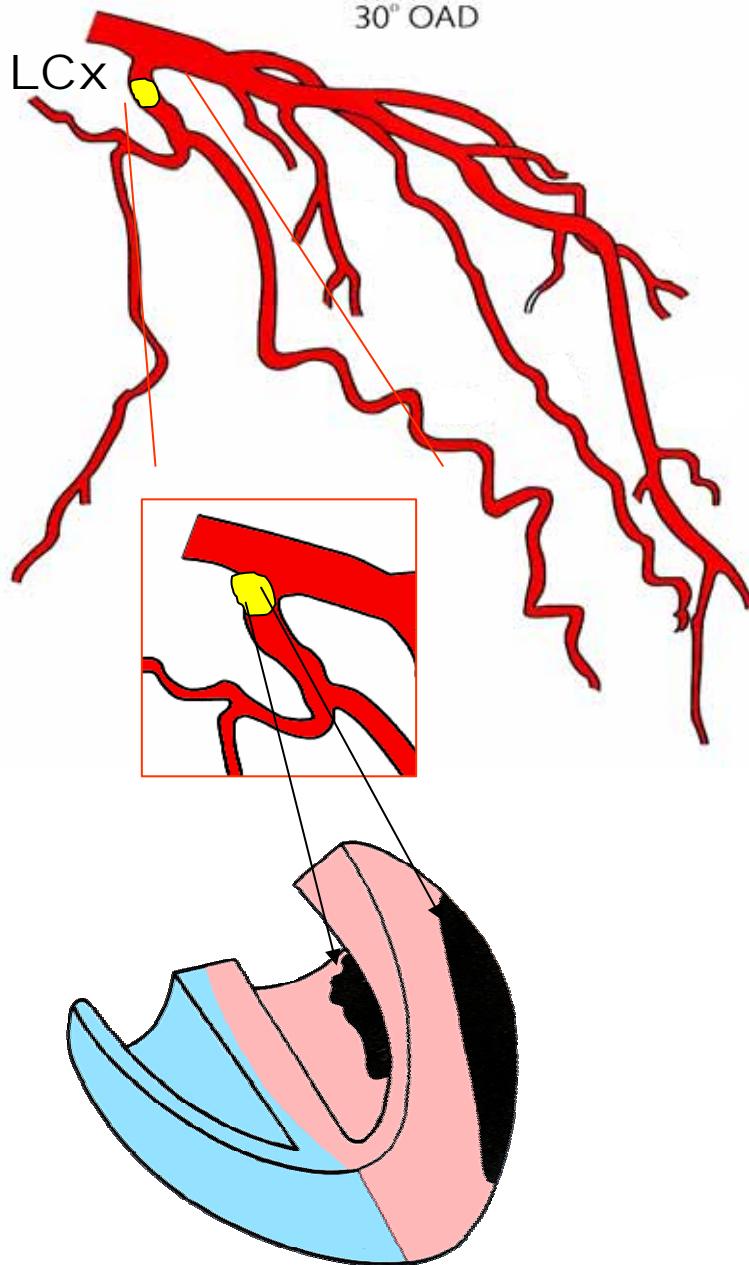
# STRICT DORSAL, POSTERIOR OR INFEROBASAL MYOCARDIAL INFARCTION

POSTERIOR VIEW OF THE HEART



LEFT POSTERIOR OBLIQUE  
VIEW OF THE HEART

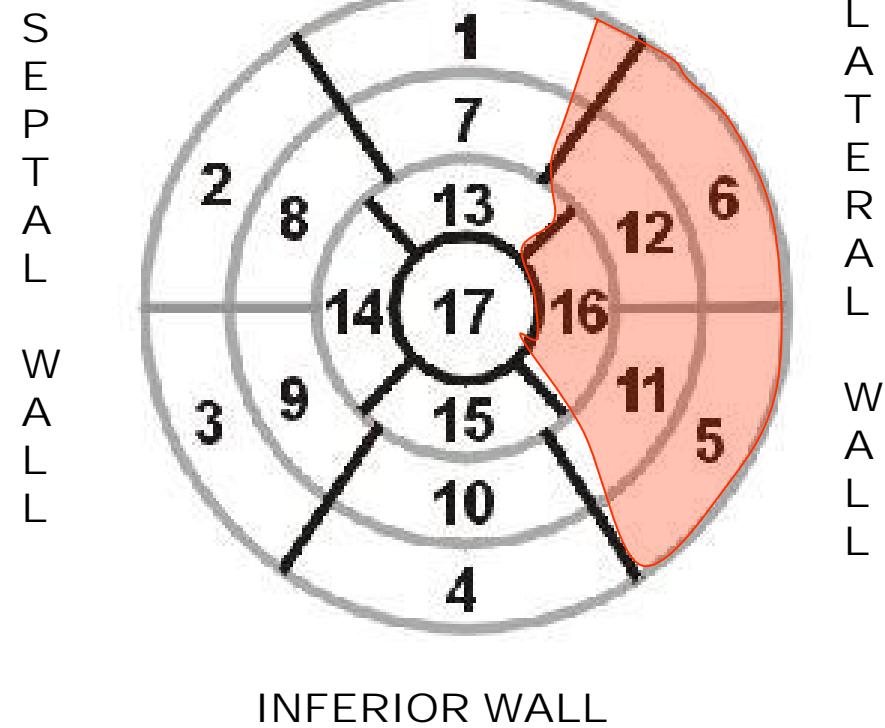




30° OAD

## 1. Lateral wall: A. Mg

## ANTERIOR WALL



There are 3 main types of VT that look like SVT:

- 1) Idiopathic Ventricular tachycardia: Without structural heart disease relatively narrow QRS complex and RBBB pattern. The QRS axis depends on which fascicle is involved in the re-entry. Left axis deviation is noted with left posterior fascicular tachycardia and right axis deviation with left anterior fascicular tachycardia. A left septal fascicular tachycardia with normal axis has also been described. Response to verapamil is an important feature of fascicular tachycardia.
- 2) Bundle branch Reentrant VT in patients with dilated cardiomyopathy
- 3) Fascicular VT : related digitalis toxicity

In the present case there are a clear structural heart disease: episode coronary heart disease prior infarction (He had underwent angioplasty with stent implantation for the RCA 6 days before) VT generally is a consequence of ischemic or structural heart disease or electrolyte deficiencies

Conclusion: scar related VT. The most common setting for VT is ischemic heart disease, in which myocardial scar tissue is the substrate for electrical reentry,

Question: Why the QRS is relatively narrow?

Answer: because the focus is near to the conduction system

Approach: Electrophysiological Study study and radiofrequency catheter ablation (RFCA)

When?: only after stabilization picture and prior evaluation LV ventricular function

Other than bundle branch reentry and interfascicular reentry, monomorphic postmyocardial infarction (post-MI) reentrant VT including the His-Purkinje system has not been reported. Verapamil-sensitive idiopathic left VT includes the left posterior Purkinje fibers but develops in patients without structural heart disease.

Hayasi et al (1) describe a novel mechanism of reentrant VT arising from the left posterior Purkinje fibers in patients with a prior MI. The study consisted of 4 patients with a prior MI and symptomatic HF who underwent EPS and RFCA for VT showing RBBB ( $n = 3$ ) or atypical LBBB ( $n = 1$ ) pattern with superior axis. In two patients, the VT frequently emerged during the acute phase of MI and required emergency RFCA.

Clinical VT was reproducibly induced by EPS. In three patients, both diastolic and presystolic Purkinje potentials were sequentially recorded along the left ventricular posterior septum during the VT, whereas in the fourth patient, only presystolic Purkinje potentials were observed. During entrainment pacing from the right atrium, diastolic Purkinje potentials were captured orthodromically and demonstrated decremental conduction properties, whereas presystolic Purkinje potentials were captured antidromically and appeared between the His and QRS complex. Radiofrequency energy delivered at the site exhibiting a Purkinje-QRS interval of  $58 +/ - 26$  ms successfully eliminated the VTs without provoking any conduction disturbances.

Reentrant monomorphic VT originating from the left posterior Purkinje fibers, which is analogous to idiopathic left VT, can develop in the acute or chronic phase of MI. RFCA is highly effective in eliminating this VT without affecting LV conduction.(1)

1. Hayashi M, Kobayashi Y, Iwasaki YK, Morita N, Miyauchi Y, Kato T, Takano T. Novel mechanism of postinfarction ventricular tachycardia originating in surviving left posterior Purkinje fibers. *Heart Rhythm*. 2006 Aug;3:908-918.