

YOUNG WOMAN WITH REPETITIVE FAST PALPITATIONS
MULHER JOVEM COM PALPITAÇÕES RÁPIDAS DE REPETIÇÃO
MUJER JOVEN CON PALPITACIONES RÁPIDAS REPETITIVAS

Perguntas

1. Qual o diagnóstico da taquicardia?
2. O que aconteceu após adenosina?
3. Qual a conduta?

Questions

1. Which the tachycardia diagnosis?
2. What happened after IV adenosine?
3. Which the appropriate conduct?

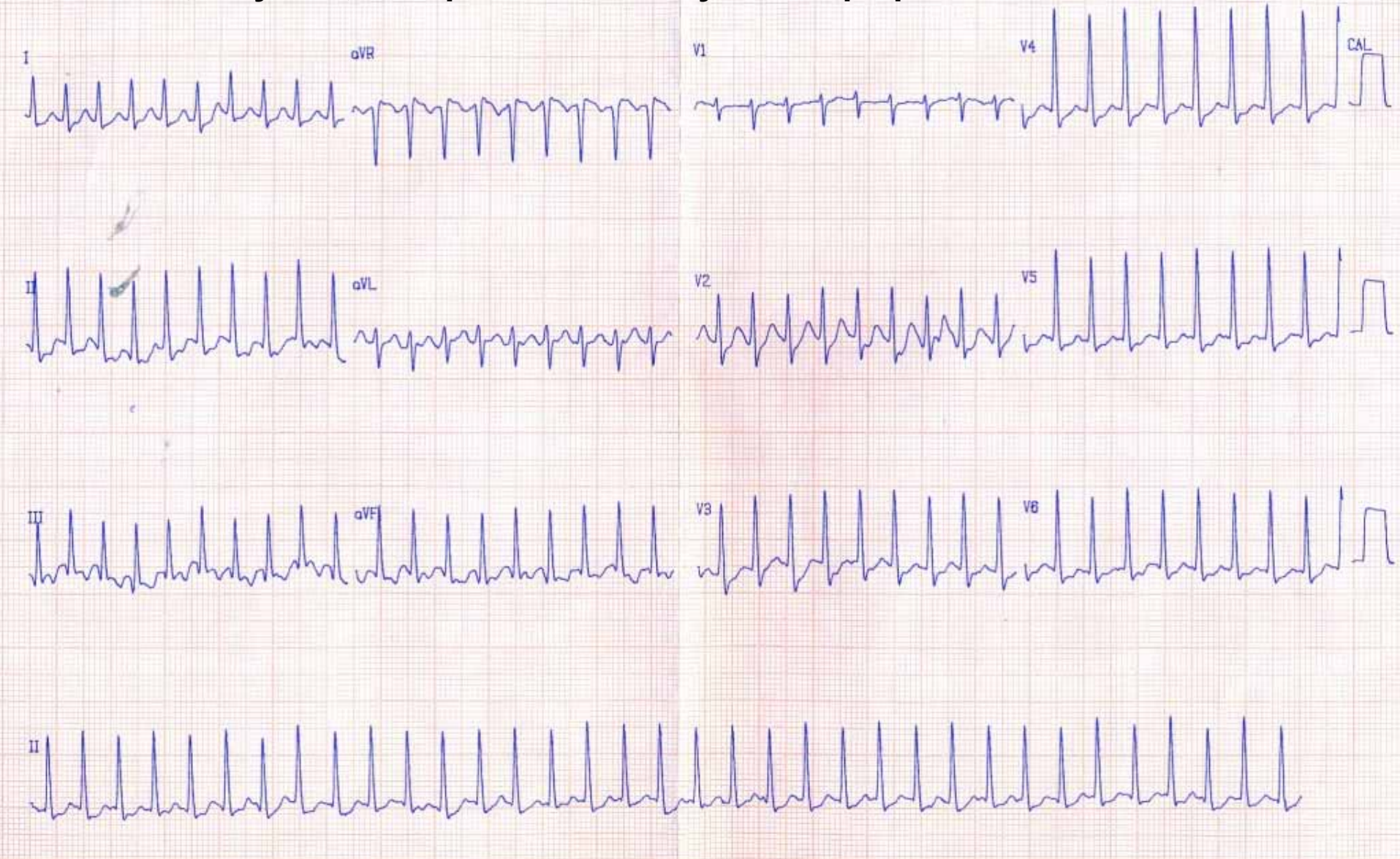
Raimundo Barbosa Barros MD

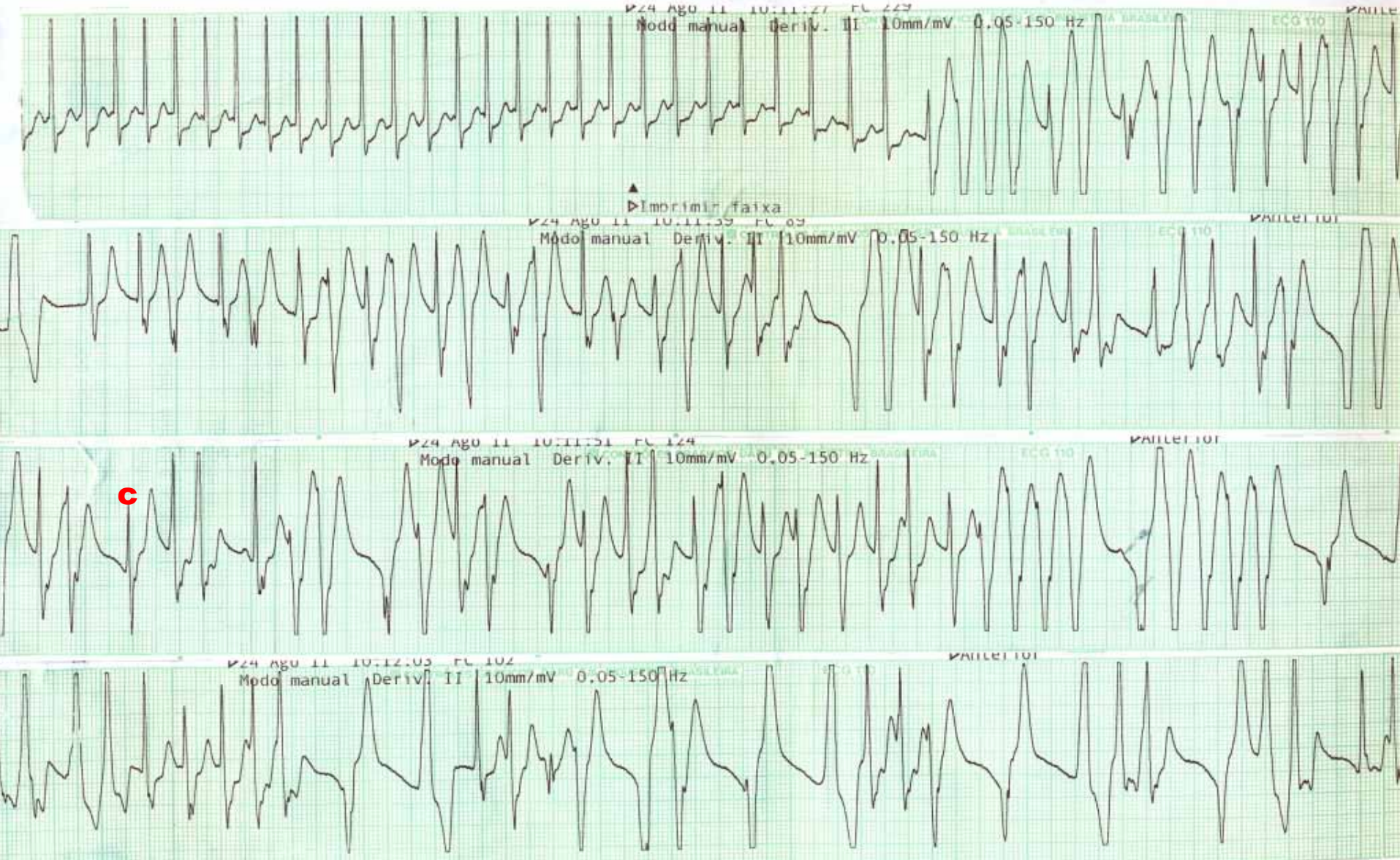
Coronary Center Hospital de Messejana Dr. Carlos Alberto Studart Gomes Fortaleza-Ceará-Brazil

Final commentaries by:

Andrés Ricardo Pérez-Riera M.D. Ph.D. Chief of Electrovectorcardiogram Sector – Cardiology
Discipline – ABC Faculty – ABC Foundation - Santo André – São Paulo-Brazil riera@uol.com.br

Mulher, 22 anos, com história prévia de palpitações rápidas ECG da admissão
Woman, 22 years, with previous history of fast palpitations ECG of the admission



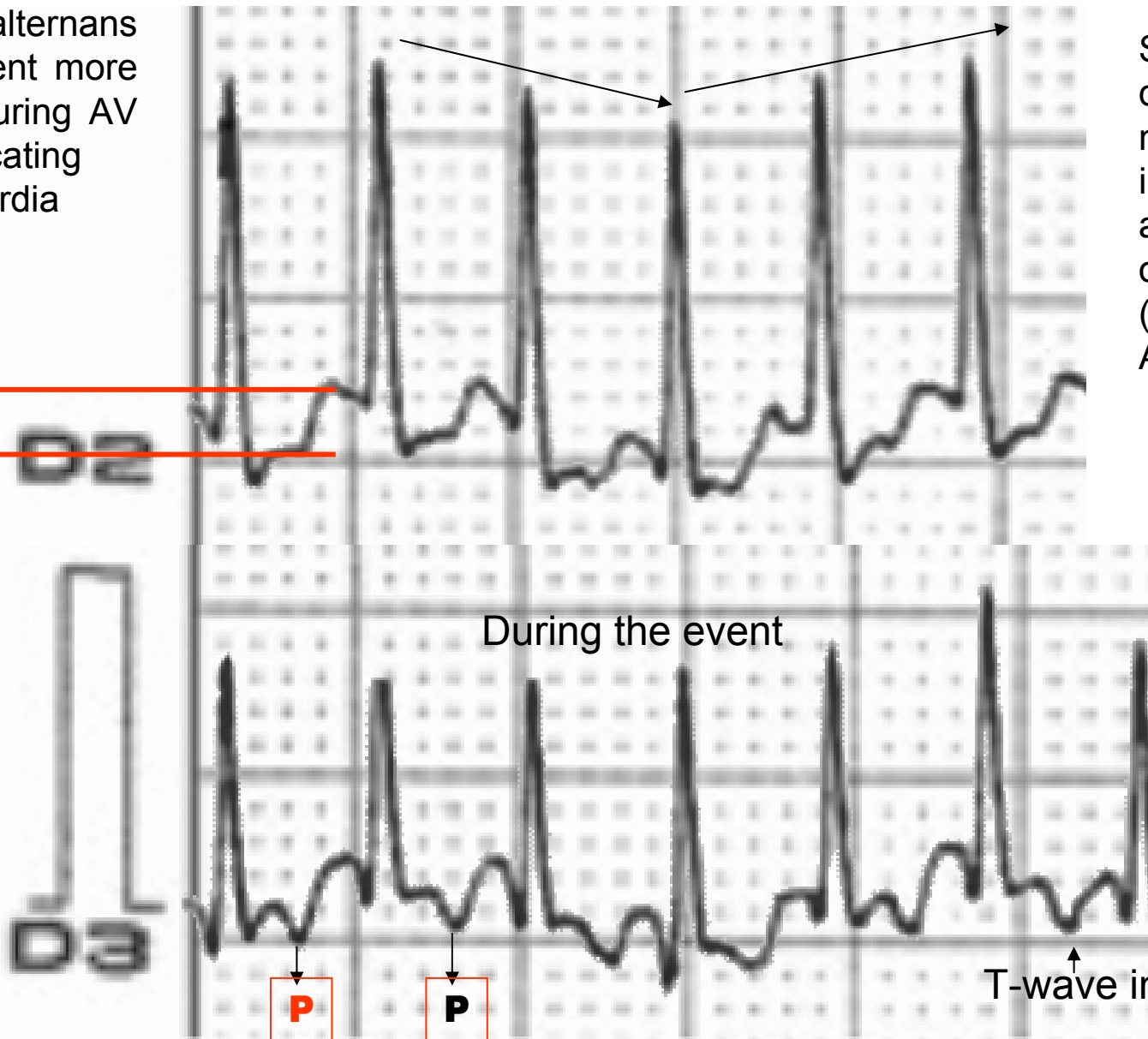


We observe the concertina effect or Öhnell “accordion” phenomenon. It is due to a progressive greater or lesser percentage of ventricular activation by the AP in comparison to the normal pathway, which is translated into a QRS complex with greater or lesser fusion degree. In AF with AV conduction over accessory pathway capture and fusion beats are possible.

QRS alternans is present more often during AV reciprocating tachycardia
AVRT

ST-segment depression (≥ 2 mm) or T-wave inversion, or both, are present more often in AVRT (60%) than in AVNRT (27%).(1)

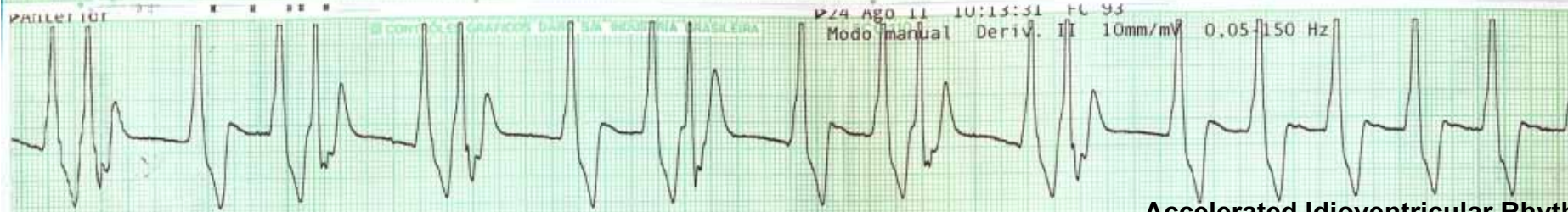
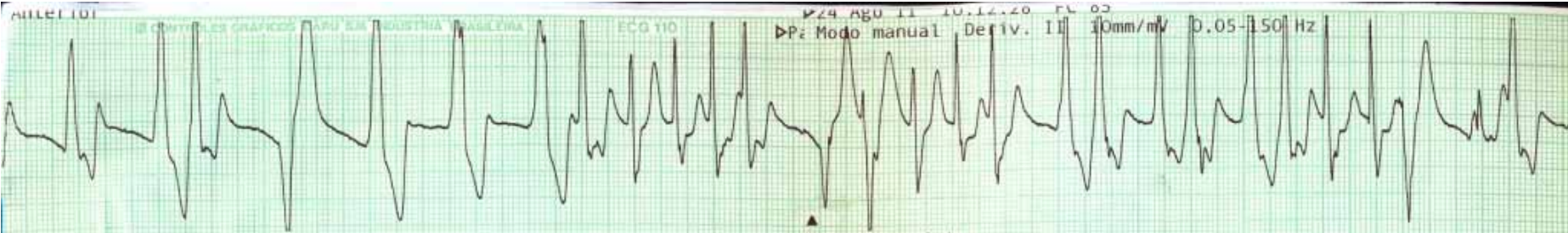
≥ 2 mm



A P wave separate from the QRS complex is observed more frequently in **AVRT** (68%) and atrial tachycardias (75%).

1. Erdinler I, Okmen E, Oguz E, et al. Differentiation of narrow QRS complex tachycardia types using the 12-lead electrocardiogram. Ann Noninvasive Electrocardiol. 2002 Apr;7:120-126.

Adenosine induced AF precipitating polymorphic ventricular tachycardia.



Accelerated Idioventricular Rhythm



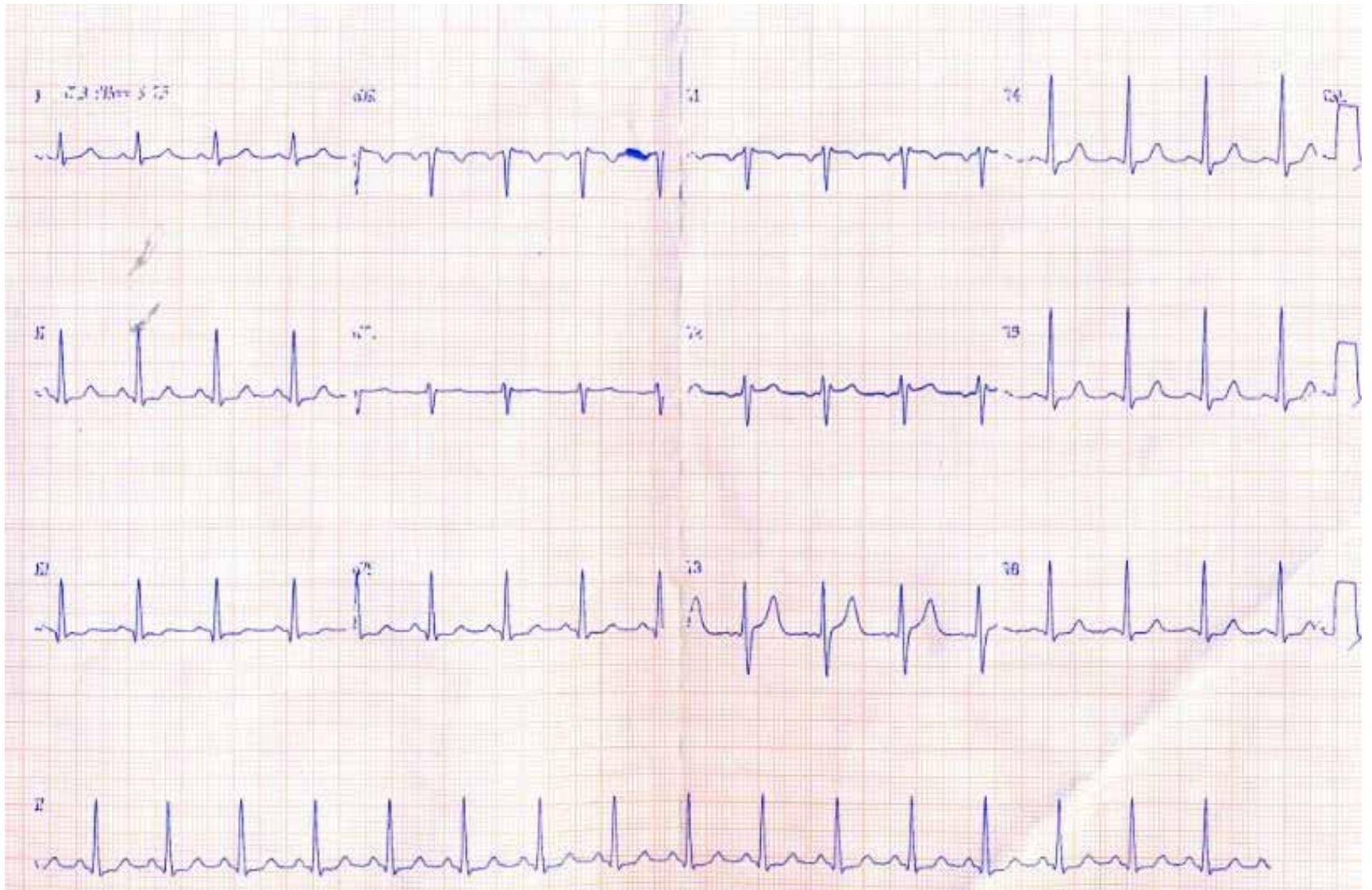
Accelerated Idioventricular Rhythm (AIVR)
HR 88bpm



Junctional rhythm impulses coming from a locus of tissue in the area of the atrioventricular node inverted P wave precede QRS complex.



Após 5 minutos
After 5 minutes



Colleagues opinions

Siempre Raimundiño nos sorprende

- 1. Taquicardia ortodromica por una vía accesoria me impresiona izquierda**
 - 2. durante la al bloquearse el nodo AV el ritmo D**
 - 3. de escape ventricular conduce ventrículo atrial e induce una fibrilacion auricular que conduce alternativamente en forma anterograda por el nodo AV y por la VA**
- sin dudas ablación por catéteres**

Saludos Pancho

Raimundinho is always surprising us.

1. Orthodromic tachycardia through an accessory pathway, which I think is left
2. during the tachycardia, when the AV node blocks, the rhythm of ventricular escape conducts in a ventriculo-atrial way and induces atrialfibrillation that conducts alternatively in an anterograde way through the AV node and the accessory pathway
3. undoubtedly, ablation through catheterization

Regards,

Pancho

Very good case.

The initial ORT seems to conduct retrogradely through a posteroseptal pathway. However, during pre-excited fibrillation, it seems that there could be another anterolateral pathway? Or did I drink too much wine today?

I am surprised at the sudden change from ORT into pre-excited atrial fibrillation with a very low dose of adenosine and a recovery so fast of anterograde conduction of the pathway?? Multiple pathways? Or maybe I'm missing some other detail.

Muy buen caso. El ORT inicial parece conducir en forma retrograda por una via posteroseptal.

Sin embargo, durante la fibrilacion preexitada pareciera que hubiera otra via anterolateral? o a lo mejor tome mucho vino hoy?

Me sorprende el cambio tan brusco de ORT a fibrillation auricular preexitada con una dosis tan baja de adenosina y la recuperacion tan rapida de la conduccion anterograda de la via?? Multiples vias? o a lo mejor me estoy tragando alguna otra detalle.

Dardo Ferrara.

Dear Andres:

PSVT: due to either AVNRT or AVRT involving a concealed accessory pathway.

Wide QRS tachycardia post adenosine-SVT termination: 100% due to polymorphic VT + short-lasting SVT

I exclude an antegradely conducting accessory pathway

TX: RF ablation of SVT

Comment: this is a rare but well known arrhythmia after IV adenosine (and also verapamil) for terminating PSVT

Best regards

Prof. Belhassen, Bernard

Director, Cardiac Electrophysiology Laboratory

Tel-Aviv Sourasky Medical Center

Tel-Aviv 64239, Israel

Tel/Fax: 00.972.3.697.4418

bernardb@tasmc.health.gov.il

Taquicardia paroxística supraventricular de QRS estrecho ocasionada o por una AVRT (taquicardia AV reentrante nodal) o por una (AVNRT) que involucra una vía accesorio cancelada

Taquicardia de QRS ancho post-adenosina terminando el evento supraventricular: 100% dado por taquicardia ventricular polimórfica + seguido por un controlado evento de taquicardia supraventricular. Yo excluyo vía accesorio anterógrada conductora

Tratamiento: Radiofrecuencia ablativa de la taquicardia paroxística supraventricular

Comentario esta es una rara pero bien conocida arritmia producida después de Adenosina (y también después de verapamil) que ocurre al terminar una taquicardia paroxística supraventricular

Mis mejores saludos

Bernard

Dear colleagues, There are antagonical points of view in relation to our three questions:

1.What is the diagnosis of tachycardia? Both Pancho and Dardo thought without hesitation that this was "orthodromic circus movement tachycardia"(CMT) that uses a cancelled, retrogradely conducted accessory pathway. Already Professor Bernard thinks that it could be one or the other: AVNRT or CMT (circus movement tachycardia).

2.What happened after adenosine? In this item there is an open disagreement: Dardo and Francisco thought this was pre-excited AF, conducting by 2 alternating pathways; professor Bernard thinks decisively, that this is polymorphic VT!!!!

3.What is the appropriate approach? In this item, the three agree.

If it was not for the enormous scientific weight of Bernard, I would say he is wrong. What do the others think???? Dardo and Pancho insist on their diagnoses? If so, what are your respective reasoning?. Andrés.

Queridos colegas existen puntos de vistas antagónicos a nuestras tres preguntas:

Cual es el diagnóstico da taquicardia? Tanto Pancho cuanto Dardo pensaron sin dudar que se trataba de una "Orthodromic Circus Movement Tachycardia"(CMT) que usa una via accesoria cancelada conducida retrogradamente. Yá el Profesor Bernard piensa que tanto puede ser una cuanto otra: (AVNRT) o una CMT(Circus Movement Tachycardia)

Que ocurrió póst adenosina? En este item hay franca discordância: en cuanto Dardo y Francisco pensaron que era una FA pre-excitada conduciendo por 2 vias alternadamente el profesor Bernard sostiene en forma contundente que es una TV polimórfica!!!!

Cual es la conducta adecuada? En este item los tres coinciden.

Si no fuera el enorme peso científico de Bernard yo diria que está equivocado. Que piensan otros????. Dardo y Pancho permanecen con sus diagnósticos?. Caso positivo cuales son sus argumentos respectivos.

Andres.

El diagnostico diferencial de la ORT/CMT is siempre AVNRT atipica. No hay dudas de esto. El segundo punto es muy interesante. Por eso me habia preguntado antes si estaba perdiendo algunos detalles. La forma de comienzo de la tachicardia de complejo ancho es muy extrana para FA preexitada. Y ahora que veo bien, pareciera haber una captura sinusal evidente. Tampoco sabia de la posibilidad de TV polimorfa con adenosina? Alguien tiene la referencia? Creo que el Dr Belhassen debe tener razon. Muy buen caso. Felicitaciones EPS y ablacion de PSVT. Ver que se induce y la conduccion retrograda despues de la ablacion. Dardo

Estimado Dr Dardo: le envio algunos de los articulos y links de pubmed que ud pidio.

Dear Dr Dardo: I'm sending some manuscripts and links from pubmed that you request

Un cordial saludo Regards

Martin Ibarrola

Emerg Med J. 2004 Jul;21(4):408-10. Proarrhythmic effects of adenosine: a review of the literature. Mallet ML. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1726363/pdf/v021p00408.pdf>

Smith JR, Goldberger JJ, Kadish AH. Adenosine induced polymorphic ventricular tachycardia in adults without structural heart disease. *Pacing Clin Electrophysiol*. 1997;201:743-5.

Tan HL, Spekhorst HH, Peters R, Wilde A. Adenosine induced ventricular arrhythmias in the emergency room. *Pacing Clin Electrophysiol*. 2001;24:450-5. Ann Noninvasive Electrocardiol. 2008 Oct;13(4):386-90.

Adenosine-induced ventricular arrhythmias in patients with supraventricular tachycardias. Ertan C, Atar I, Gulmez O, Atar A, Ozgul A, Aydinalp A, Müderrişoğlu H, Ozin <http://onlinelibrary.wiley.com/doi/10.1111/j.1542-474X.2008.00245.x/abstract;jsessionid=A3B9E19BE34F8DCBE1D67EDD223D542F.d03t01>

5. Harrington GR, Froelich EG. Adenosine-induced torsade de pointes. *Chest*. 1993;103:1299 <http://chestjournal.chestpubs.org/content/103/4/1299.full.pdf>

Estimado Maestro Andres y Raimundo;

1. Taquicardia regular 150 latidos por min con QRS angosto.

Diagnostico TPSV por reentrada nodal. El RP menor de 40 mseg que presenta y es mas evidente n DII y DIII, es sugerente de esta arritmia y del mecanismo de una doble via nodal con fenomeno de reentrada.

Diagnostico diferencial en este caso Taquicardia ortodromica por haz accesorio.

Se encontraba hemodinamicamente estable al ingreso Dr Raimundo? Si presentaba inestabilidad hemodinamica al ingreso pienso que la cardioversion electrica se podria haber considerado.

2. La administracion de adenosina, que bloquea transitoriamente el NAV y deprime el NS.

En este caso desencadena una TV polimorfa.

3, Tratamiento: el efecto de la adenosina es transitoria rapidamente metabolizada, por lo que no me parece necesaria la cardioversion electrica. No esperaria que degenera en una FV, sino que revierte al metabolizarse la misma.

4. Tratamiento definitivo: EEF y ablacion

Un saludo

Espero las opiniones de los expertos

Martin Ibarrola

Estimado Maestro Andres:

Voy a abusar de su buena voluntad pero me encantaria me expliquen los Dres Dardo o Pancho si tiene mas de una via accesorio, ambas vias conducen solo retrogramente?

En el trazado posterior no presenta signos de WPW, o sea la/s via/s no conducen en forma anterograda. Esto descarta la FA preexcitada a mi criterio, ya que alguna de estas deberia conducir en forma anterograda por lo descripto por ambos y observarse en el trazado posterior signos de esta (no presenta ninguno).

Un saludo y perdón por no incluirlo en el mensaje anterior.

Marin Ibarrola

Dear Master Andres and Raimundo: 1. Regular tachycardia, 300 bpm with narrow QRS. Diagnosis of SVT by nodal reentry (AVNRT- Atrioventricular nodal reentry tachycardia). The RP lower than 40 ms that she presents and more evident in DII and DIII, is suggestive of this arrhythmia and the mechanism of a double nodal pathway with reentry phenomenon. Differential diagnosis in this case, orthodromic tachycardia by accessory pathway. Was she hemodynamically stable when Dr. Raimundo admitted her? If hemodynamically unstable at admittance, I think that electric cardioversion could have been considered. 2. The administration of adenosine, which transiently blocks the AV node and depresses the sinus node, in this case triggers polymorphic VT. 3. Treatment: the effect of adenosine is transient and quickly metabolized, so that I don't think electric cardioversion is necessary. I would not wait for it to degenerate into VF, but for it to revert when the drug gets metabolized. 4. Definitive treatment: EPS and ablation.

I would love to read the concepts by Dr. Dardo and Pancho.

Regards,
Martin Ibarrola

Adenosine is used to revert paroxysmal supraventricular tachycardia by blocking the atrioventricular node. Administered endovenously, it depresses the activity of the sinus node and is used for a quick conversion to sinus rhythm of supraventricular arrhythmias by reentry. It acts as a neuroprotector by inhibiting excitatory transmission of A1 receptors. This endogenous purine nucleotide, when stimulating cardiac A1 receptors, activates a K⁺ outward current, sensitive to acetylcholine in the atria, sinus node, atrioventricular node, which produces action potential duration shortening, hyperpolarization and slows normal automaticity down.

It also inhibits the electrophysiologic effects of increased intracellular AMPc, which occurs with sympathetic activation. Thus, it inhibits inflow of Ca²⁺ stimulated by AMPc, which also depresses the frequency of SA node cells and the conduction velocity of through the AV node, while prolonging its refractory period.

Dear Master Andrés: I copy what I had previously expressed about these concepts. I agree with the line of thought by Professor Bernard. I don't agree with the sequence of thoughts by Drs. Dardo and Pancho. Why? The way I see it, this is atypical nodal reentry (AV nodal reentrant tachycardia – AVNRT or atrioventricular nodal reentrant tachycardia), and due to the evidently unusual high frequency for this type of tachycardias, it leads to think about orthodromic circus tachycardia. They leaned toward pre-excited AF, and as the complexes have several morphologies in the monitoring tracing, the only explanation would be more than one accessory pathway involved. Evidently, from the first initial diagnosis, the diagnosis of the tracing of posterior arrhythmia arises, or the other way round of this arrhythmia (for me polymorphic VT), pre-excited AF for the Drs., is that with the initial HR and this, they immediately thought about WPW and its different arrhythmias. The tracing of the monitoring of post-adenosine polymorphic VT has been widely described in bibliography. My greatest disagreement with both is if they thought on pre-excited AF, and following their line of thought, they should have considered that the management should be electric cardioversion. It does not match with their previous concepts. Their diagnosis is contradictory with the waiting management. Maybe they can explain why did they think about an AF that conducts through accessory pathway and they didn't propose cardioversion. If there is more than one accessory pathway, do both pathways conduct only retrogradely? In the posterior tracing there are no signs of WPW, i.e. the pathway(s) do not conduct anterogradely. This makes pre-excited AF very unlikely the way I see it, since some these should be conducted in an anterograde way because of what has been described by both, and check in the posterior tracing for signs of this (there is none). Dear Pancho, you are so very patient with me, and maybe you can clarify these issues.

Regards,

Martin Ibarrola

Estimado Maestro Andres: Copio lo que habia expresado previamente a estos conceptos. Comparto la line de pensamiento del Profesor Bernard. No comparto la secuencia de pensamiento de los Dres Dardo y Pancho. Porque? mi impresion es una reentrada nodal atipica (AV nodal reentrant tachycardia-AVNRT, or atrioventricular nodal reentrant tachycardia), por la alta frecuencia obviamente inusual para este tipo de taquicardias es que induce a pensar en una TOR ("Orthodromic Circus Tachycardia".CMT) Se han inclinado por una FA preexcitada, en el trazado del monitoreo como tienen varias morfologias los complejos la unica explicacion seria mas de un haz accesorio involucrado. Obviamente del primer diagnostico inicial surge el diagnostico del trazado de la arritmia posterior, o al reves de esta arritmia (para mi una TV polimorfica), para los Dres FA preexcitada, es que con la FC inicial y esto pensaron inmediatamente en WPW y sus diferentes arritmias. El trazado del monitoreo TV polimorfica post adenosina, ampliamente descripto en la bibliografia.

Mi mayor desacuerdo con ambos es si pensaron en una FA preexcitada, siguiendo su linea de pensamiento deberian haber pensado que la conducta deberia ser la cardioversion electrica. No concuerda con sus conceptos anteriores. Contradictorio su diagnostico con la conducta espectante. Tal vez puedan explicar porque pensaron en una FA que conduce por via accesorio y no plantearon cardioversion. Si tiene mas de una via accesorio, ambas vias conducen solo retrogramente? En el trazado posterior no presenta signos de WPW, o sea la/s via/s no conducen en forma anterograda. Esto hace poco probable la FA preexcitada a mi criterio, ya que alguna de estas deberia conducir en forma anterograda por lo descripto por ambos y observarse en el trazado posterior signos de esta (no presenta ninguno).

Querido Pancho vos que me tenes mucha paciencia tal vez me aclares estos puntos. Un saludo

Martin Ibarrola

El diagnostico diferencial de la ORT/CMT is siempre AVNRT atipica. No hay dudas de esto. El segundo punto es muy interesante. Por eso me habia preguntado antes si estaba perdiendo algunos detalles. La forma de comienzo de la tachicardia de complejo ancho es muy extrana para afib preexitada. Y ahora que veo bien, pareciera haber una captura sinusal evidente. Tampoco sabia de la posibilidad de TV polimorfa con adenosina? Alguien tiene la referencia? Creo que el Dr Belhassen debe tener razon.

Muy buen caso. Felicitaciones

EPS y ablacion de PSVT. Ver que se induce y la conduccion retrograda despues de la ablacion.

Dardo Ferrara

Undoubtedly, we are facing a proarrhythmic effect from adenosine, and maybe professor Bernard (who am I to say to say maybe no?) is right; I think that the tip of the diagnosis is at the end of the recording of events. If it was pre-excited AF, with anterograde conduction favored by concealed accessory pathway after adenosine, maybe the behavior would have been different, unless the accessory pathway has a very poor AV conduction (there is no evident pre-excitation in posterior ECG) and conduction is quickly exhausted through it, and what we may see is a controlled heart rate through the AV node, and with a certain degree of aberration, until it reverts. Sinus rhythm with posterior sinus tachycardia by adrenergic modulation. Undoubtedly, as a proarrhythmic effect of adenosine, the appearance of VT of the polymorphic type is also described, just as Bernard says. I have no doubts that this patient should go to ablation by catheterization of her paroxysmal SVT. Regards, Pancho

Sin dudas estamos ante un efecto proarritmico de la adenosina, y tal vez el profesor Bernard (quien soy yo para decir tal vez no?) tenga razón, creo que el tip del diagnostico esta al final del registro de eventos. Si fuera una FA preexcita, favorecida la conducción Anterograda por una VA oculta luego de la adenosina, tal vez el comportamiento hubiera sido otro, excepto que la VA tenga muy mala conducción AV (no hay preexcitacion patente en el ECG posterior) y rápidamente se agote la conducción por la misma y lo que veamos es una frecuencia cardiaca controlada a través del nodo AV, y con cierto grado de aberrancia, hasta que revierte. Ritmo sinusal con taquicardia sinusal posterior por modulación adrenergica.

Sin dudas como efecto proarritmico da adenosina también esta descripto la aparición de TV de tipo polimorfica como bien dice Bernard.

No me quedan dudas que esta paciente debe ir a ablación por cateres de su TPSV, saludos Pancho

This young woman has a supraventricular tachycardia, either AVNRT or AVRT. It is abruptly terminated by adenosine. I am not convinced that I can clearly identify P waves to provide a clue as to its mechanism. There is also a possibility that it is an automatic tachycardia in the atria but even if adenosine terminated it, I would expect to first see a couple of cycles of AV block before the tachycardia ended. During the tachycardia, there is extensive myocardial ischemia as the rate is simply too fast markedly increasing metabolic demand and the diastolic period too short to allow for adequate coronary blood flow. Ischemia need not imply atherosclerotic heart disease and/or coronary vasospasm – I would not suspect either in this 22 year old. Immediately post-termination with a slower rate reducing metabolic demand and the longer diastolic period allowing for myocardial reperfusion, one sees malignant ventricular “reperfusion” arrhythmias. This is potentially life threatening. Either she should be placed on a medication such as a calcium channel blocker to prevent the arrhythmias in the first place which was the only option some 20+ years ago or today, referred to an EP lab, have the tachycardia induced and mapped followed by ablation of the critical pathway.

Paul A. Levine MD, FHRS, FACC, CCDS

25876 The Old Road #14

Stevenson Ranch, CA 91381

Cell: 661 565-5589

Fax: 661 253-2144

Email: paul91321@gmail.com

Esta jovem tem uma taquicardia supraventricular de QRS estreito, quer seja uma AVNRT ou uma Taquicardia AV nodal reentrante. A mesma é abruptamente terminada por adenosina. Não estou convencido de que eu possa identificar claramente as ondas P para fornecer uma pista sobre o seu mecanismo. Existe também a possibilidade de que se trate de uma taquicardia automática atrial, mas mesmo que adenosina tenha terminado ela, eu esperaria ver primeiro um par de ciclos de bloqueio AV antes da taquicardia terminar.

Durante o evento, se observa isquemia miocárdica extensa consequência de uma FC muito rápida condicionante de uma acentuada e crescente demanda metabólica e, como o período diastólico é extremamente curto no permite um adequado fluxo sanguíneo coronário. Isquemia não implica necessariamente em doença aterosclerótica e / ou vasoespasmismo coronariano – Eu não suspeitaria disto numa paciente de 22 anos.

Imediatamente apos terminar do evento com a menor FC e a redução das demandas metabólicas e o mais longo período diastólico se permite a reperfusão miocárdica, deflagradora da arritmia maligna de reperfusão a qual é potencialmente uma ameaça à vida. Por isto, antigamente se administaba um bloqueador dos canais de cálcio para prevenir estas arritmias malignas. Esta era a única opção há 20 anos atrás mas hoje, se encaminha a um laboratório de eletrofisiologia onde poderá mapear se e ablacionar a via crítica.

Paul A. Levine MD, FHRS, FACC, CCDS

25876 The Old Road #14

Stevenson Ranch, CA 91381

Cell: 661 565-5589

Fax: 661 253-2144

Email: paul91321@gmail.com

Hello, professor. Very interesting case, and -at least to me- very difficult:

- 1. Tachycardia by nodal reentry (AVNRT), slow-rapid -pr>rp, and I say nodal because the RP is less than 0.08 sec.**
- 2. After adenosine, I see three types of complexes that repeat with different paces. Difficult to interpret, but the explanation that suits me is VT - I see what seems to be AV dissociation. This rhythm becomes irregular with fusion beats (1st and 5th complex after reversion, for instance) and capture beats - 2nd and 4th complexes of the inferior strip to the previous one. There is a fourth form that appears at the end of this strip, that has a morphology similar to the delta wave, for instance, the last beat.**
- 3. Sinus EKG, incomplete right bundle branch block, the QT seems normal. Since this is a young girl, supposedly without structural heart disease, nor coronary disease, I would send her to electrophysiologic study.**

Diego Fernandez

Hola profesor. Un caso muy interesante y -al menos para mí- muy difícil.

- Taquicardia por reentrada nodal (AVNRT) lenta rápida -PR > RP y digo nodal porque el RP mide menos de 0,08 seg-**
- Luego de la Adenosina veo tres tipos de complejos que se repiten con distintas cadencias. Difícil de interpretar, pero la explicación que me cuadra es decir TV -aprecio lo que parece ser una discociación AV, este ritmo se irregulariza con latidos de fusión (1° y 5° complejo tras reversión por ej) y de captura-2° y 4° complejos de la tira inferior a la previa. Hay una cuarta forma que aparece al finalizar esta tira que tiene una morfología similar a onda delta, por ejemplo el último latido.**
- EKG sinusal bloqueo incompleto de rama derecha, QT impresiona normal Al tratarse de una chica joven, en principio sin cardiopatía estructural, ni enfermedad coronaria, iría al estudio electrofisiológico.**

Diego Fdez

Greetings. She presents: supraventricular tachycardia with narrow QRS of 0.06 seconds. HR of 150 bpm. Right after the use of adenosine, premature contractions stand out, mainly in the post-adenosine continuity (tracing No. 1).

The origin of the premature contractions is at the level of the sinoatrial node or at atrioventricular level, right after the use of 6 mg of IV adenosine and as a result it produces this form of premature contraction. With background of branch block in AVR, D2, D3, V2, V3 prior to QRS complex. She presented something similar to 3rd degree AV block in continuity (in relation to tracing No. 2). QRS complex in AVR, V1, V2; what branch of the His bundle was affected and evolved with the T wave in V3, V4, V5, V6 right after the use of adenosine?

About the management of this case, what is the original focus at the level of the sinoatrial node and the atrioventricular node? In the marked tracing of D2, D3, there is right bundle branch block. When did she present tachycardia in her history and noticed something strange? In brief, related to an arrhythmia as the current one. What is the condition of the His bundle after adenosine with just 6 mg? What is the focus at ventricular level?

Another aspect is an orthodromic supraventricular tachycardia between the AV node and the sinoatrial node with anterograde conduction in this case.

Regards,

Gregorio Maslivar

Mis saludos. PRESENTA:

TAQUICARDIA SUPRAVENTRICULAR CON QRS ESTRECHO DE 0.06 SEGUNDOS. FC DE 150 lpm. JUSTO DESPUÉS DEL USO DE ADENOSINA LLAMA LA ATENCIÓN LAS EXTRASÍSTOLES SOBRE TODO EN LA CONTINUACIÓN POST ADENOSINA (EL TRAZO # 1). EL ORIGEN DE LAS EXTRASÍSTOLES ES A NIVEL DEL NODO SINOAURICULAR O A NIVEL AURICULOVENTRICULAR JUSTO DESPUÉS DEL USO DE ADENOSINA 6mg IV Y

COMO RESULTADO DA ESTA FORMA DE EXTRASÍSTOLES.

CON ANTECEDENTES DE BLOQUEO DE RAMA EN ,AVR, D2, D3,V2,V3 PREVIO AL COMPLEJO QRS.

PRESENTO ALGO PARECIDO A UN BLOQUEO AV DE 3ER GRADO EN LA CONTINUACIÓN.(ES CON RESPECTO AL TRAZO#2).

EL COMPLEJO QRS EN AVR,V1,V2 QUE RAMA DEL HAZ DE HIZ QUEDÓ AFECTADA Y EVOLUCIONÓ CON LA ONDA T EN V3,V4,V5,V6,JUSTO DESPUÉS DEL USO DE ADENOSINA.

CON RESPECTO AL MANEJO DE ESTE CASO CUÁL ES EL FOCO DE ORIGEN A NIVEL DEL NODO SINOAURICULAR Y NODO AURICULO VENTRICULAR. EN EL TRAZO MARCADO DE D2,D3 TIENE BLOQUEO DE RAMA DERECHA.CUANDO EN SU HISTORIA HIZO ALGUNA TAQUICARDIA Y NOTÓ ALGO RARO. EN CONCLUSIÓN RELACIONADA A UNA ARRITMIA COMO A LA ACTUAL.

COMO QUEDÓ EL HAZ DE HIZ DESPUÉS DEL ADENOSINA CON SÓLO 6mg. CUAL ES EL FOCO A NIVEL VENTRICULAR. OTRO ASPECTO ES UNA TAQUICARDIA SUPRAVENTRICULAR ORTODROMICA ENTRE EL NODO AV Y EL SINOAURICULAR CON LA CONDUCCIÓN ANTERÓGRADA EN ESTE CASO.

MIS SALUDOS.

Gregorio Maslivar

Comparative clinical and electrophysiologic effects of adenosine triphosphate and verapamil on paroxysmal reciprocating junctional tachycardia

BERNARD BELHASSEN, M.D., AARON GLICK, M.D., AND SHLOMO LANIADO, M.D.

Circulation 77, No. 4, 795-805, 1988.

Dear Andres: I would like to continue discussing the case of the Polymorphic VT after termination of SVT with adenosine. Of course, I am reiterating that the diagnosis I suggested for explaining that tracing is the good one (I would emphasize "the only good one"). Please look to the figure included in a paper I published many years ago when I was younger (I am still young !). In the same patient with AVRT associated with a manifest WPW, I compared the effects of ATP (adenosine triphosphate) and Verapamil. A nonsustained wide QRS tachycardia was observed upon termination of SVT with ATP but not with verapamil. Despite the fact that the surface leads tracings during this wide QRS tachycardia could have suggested the possibility of antegrade conduction over the accessory pathway (i.e. a supraventricular origin of the arrhythmia), a careful analysis of the intracardiac tracings actually showed that all the wide QRS complexes had a ventricular origin and were associated with a 1:1 VA relationship over the accessory pathway. In other words, even in patients with OVERT PREEXCITATION, the wide QRS tachycardia that may occur following IV adenosine are not obligatory antegradely preexcited complexes.

Best regards

Bernard

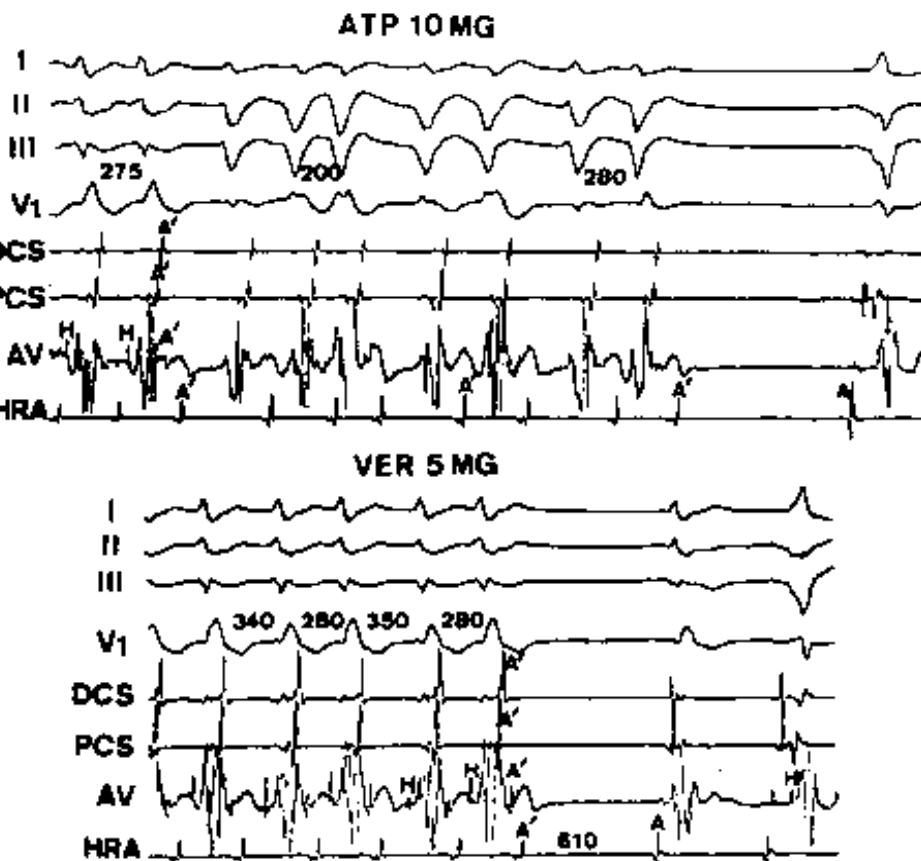


FIGURE 3. Ventricular excitability after ATP but not verapamil in a patient with a left posteroseptal accessory pathway (patient 1). Shown are electrocardiographic leads I, II, III, and V₁ and intracardiac electrograms from the distal coronary sinus (DCS), proximal coronary sinus (PCS), atrioventricular junction (AV), and high right atrium (HRA). PRJT is terminated 18 sec after administration of 10 mg ATP due to AV nodal block. Immediately after conversion, nonsustained irregular ventricular tachycardia (7 beats) with 1:1 retrograde conduction through the accessory pathway is observed. In contrast, 5 mg verapamil (VER) terminates PRJT 30 sec after administration due to AV nodal block after cycle length alternans but with no ventricular arrhythmia.

Caro Andrés: Gostaria de continuar a discutir o caso da TV polimórfica após o término da SVT com adenosina. Por favor, olhe para a figura incluída neste artigo que publiquei há muitos anos quando eu era mais jovem. No mesmo paciente com TAVR associado a um WPW manifesto, comparei os efeitos da adenosina trifosfato (ATP) e Verapamil. Uma NS-TV de QRS largo foi observada após o término do SVT com ATP, mas não com verapamil. Apesar do fato de que a superfície conduz traçados durante este taquicardia de QRS largo poderia ser sugerido a possibilidade de condução anterógrada na via acessória (ou seja, uma origem supraventricular da arritmia), porém, uma análise cuidadosa dos traçados intracardiácos mostrou que todos os complexos QRS largos eram ventriculares e estavam associados com uma relação de 1:1 sobre a via VA acessória. Em outras palavras, mesmo em pacientes com pré-excitação evidente, a taquicardia de QRS largo, que pode ocorrer após adenosina EV não sendo obrigatoriamente complexos anterógrados preexcitados.

Cumprimentos

Bernard

Dear Andrés,

It is a very interesting ECG. My observations are the following:

1) Tachycardia with narrow QRS, very high HR (300 bpm), RP 120 ms, in LV there seems to be electrical alternans. My first diagnosis: orthodromic atrioventricular reciprocating tachycardia mediated by accessory pathway.

2) Adenosine reverts the OAVRT by the mechanism we all know, and there a tachyarrhythmia occurs with QRS with different morphologies and widths:

A. Pre-excited AF. One of the unwanted effects of adenosine is producing AF and by blocking the AV node exclusively through the accessory pathway, as it presents several morphologies, we should consider there is more than one pathway.

B. Polymorphic VT is also described as an unwanted effect of adenosine. We have seen it, but for a few beats, not as prolonged as here.

C. I am curious about the rhythm being regular before the tachycardia with broad QRS ends, as if it was idioventricular rhythm, by AVB induced by adenosine.

3) Undoubtedly, EPS and RF ablation.

Regards, and we'll see what is the final diagnosis.

Oscar Pellizon

Estimado Andres,

es un ECG muy interesante, mis observaciones serian las siguientes.

- 1) Taquicardia con QRS angosto, FA muy elevada (300 lpm), rp 120 mseg, en vi impresiona alternancia electrica, mi primer diagnostico: t r av ortodromica mediada por una via accesoria.
- 2) La adenosina revierte la trazo por el mecanismo que todos conocemos y alli sobreviene una taquiarritmia con QRS con diferentes morfologias y anchos:
 - a. FA preexcitada. uno de los efectos indeseables de la adenosina es producir FA y al bloquear el NAV conduce exclusivamente a traves de la via Accesoria como presenta varias morfologias, habria que pensar que existe mas de una via.
 - b. la TV polimorfa tambien esta descripta como efecto indeseable de la adenosina. la hemos visto, pero por pocos latidos, no tan prolongado como aca.
 - c. me llama la atencion antes de terminar la taquicardia con QRS ancho, que el ritmo es regular, como si fuera un ritmo idioventricular, por BAV inducido por la adenosina
- 3) sin duda EEF y ablacion RF.

Saludos y veremos cual es el diag. final. abrazo.

Oscar a. Pellizzon

Mi opinion estimados colegas amigos

1-Diagnóstico: taquicardia paroxistica por reentrada VA por presencia de via accesorio oculta . Porque ?

por la alternancia del QRS en V 1

frecuencia de 300 min

onda P que sucede a R ,siendo RP menor que PR y con distancia RP de mas de 80 ms

por polaridad de onda P negativa en DIII sugestiva de via accesorio posteroseptal derecha

2- post adenosina fibrilacion auricular preexcitada!!

3-Conducta EEF y ARF de la via en cuestion

Saludos

J J SIRENA

Dear colleagues my opinion: Atrioventricular reciprocating tachycardia (AVRT) supported by atrioventricular reentry circuit that use the AV node anterogradely and a occult rapidly conduction accessory pathway retrogradely.

Why?

Alternance of QRS complexes in V1 lead

HR very high 300bpm

P wave after R wave with $RP' > PR$ and $RP > 80ms$.

P with polarity in III negative suggest accessory pathway right posteroseptal

2) After adenosine preexcited AF

3) Management electrophysiological study + Radiofrequency catheter ablation (RFCA)

Regards

Juan José Sirena

Amigos

1. La literatura en Adenosina se lee con los papers originales de Brignole (Italia) y de Blanc (Francia). Ellos son los que desarrollaron la investigación mas extensa sobre los efectos de Adenosina en el sistema de conduccion (ver Moore tambien) 2. La respuesta post Adenosina ha sido ampliamente descripta, y la presencia de TVNS es muy frecuente. La arritmia polimorfa es muy frecuente. He escrito un capitulo con JC Guzman sobre las características electrocardiograficas de las pruebas autonomicas, en un libro publicado por Uribe y Duque para la Soc Colombiana de Cardiologia.
3. Este caso se embarro porque 2 buenos EP (Pancho y Dardo) vieron vias accesorias y uno de ellos, multiples (lo que es mas raro que mono verde!!!)

The green monkey (*Chlorocebus sabaeus*), also known as the Callithrix monkey, is an Old World monkey with golden-green fur and pale hands and feet. The tip of the tail is golden yellow as are the backs of the thighs and cheek whiskers. It does not have a distinguishing band of fur on the brow, like other *Chlorocebus* species, and males have a pale blue scrotum. Some authorities consider this and all of the members of the genus *Chlorocebus* to be a single widespread species, *Cercopithecus aethiops* — confusingly, the name "green monkey" has also been used for this single species. The green monkey is found in West Africa from Senegal to the Volta River. It has been introduced to the Cape Verde islands off north-western Africa, and the West Indian islands of Saint Kitts, Nevis, Saint Martin, and Barbados. It was introduced to the West Indies in the late 17th century when slave trade ships travelled to the Caribbean from West Africa.

It occurs in a wide range of wooded habitats, ranging from very dry Sahel woodland to the edge of rainforests. It is also commonly seen in coastal regions, where known to feed on seashore foods such as crabs. It also takes a wide variety of other foods, including fruits and invertebrates. As other members of the genus *Chlorocebus*, the green monkey is highly social and usually seen in groups.



siendo que cuando se reestablece el RS, no hay evidencia alguna de preexcitacion. Por otro lado, el paciente presentaba taquicardia de QRS angosto (o sea, que si hubiera via, esta conduciria retrogradamente) pero luego de la Adenosina, siguiendo el pensamiento de Dardo y Pancho, la via empieza a conducir en forma anterograda!!!!

Esta confusion es notable ya que es una mezcla de 3 cosas:

Pattern recognition: lo mas dificil, porque los complejos parecen preexcitados, pero no lo son

Deductive mechanism: aqui fallaron los 2 amigos, porque mecanisticamente, era muy dificil sostener su teoria, y era mucho mas posible el mecanismo explicado por mi inicialmente (e ignorado en el foro, ya que raimundo no lo subio!!!) y luego sustentado por Bernard. Miren que facil (asi se lo dije a Raimundo, ojala conserve ese email: Typical AVNRT que revierte con Adenosina mediante arritmia ventricular (super frecuente). Nada mas, sencillito.

3. Experiencie: este mecanismo no solo esta descrito,sino que ademas es de muy frecuente observacion. La primera vez lo vi como residente de 2 anio de cardio y casi me muero de un ataque cardiaco; y llame al staff en medio de la noche y le conte que luego de darle adenosina, el tipo hizo una especie de torsion de punta que cedio sola, pero que duro como 5 segundos etc etc.

Salud AB

FINAL CONCLUSIONS

ECG This patient has a regular Paroxysmal Supraventricular Tachycardia (**PSVT**) with narrow QRS complexes (< 110 ms) and HR 250bpm ($HR \geq 120$ beats/min). Epidemiologic studies have indicated that the prevalence of PSVT is approximately two to three of 1000 persons, of whom 50-60% have slow-fast atrioventricular node reentrant tachycardia or **AVNRT**. (1) and the remained 30% **AVRT**. Which variety? Answer Orthodromic Circus Movement Tachycardia. (**CMT**) or atrioventricular reciprocating tachycardia (**AVRT**): supported via a concealed accessory pathway by atrioventricular reentry circuit that use the AV node anterogradely and a concealed rapidly conduction accessory pathway retrogradely. ST-segment depression (≥ 2 mm) or T-wave inversion, or both, are present more often in **AVRT** (60%) than in AVNRT (27%), clear QRS alternans. and P-wave that follows each of its regular, narrow QRS complexes, due to retrograde conduction.

After adenosine: Both preexcited atrial fibrillation AF and Polymorphic VT(PVT) after termination of SVT with adenosine. Adenosine induced first AF precipitating non-sustained polymorphic ventricular tachycardia.(2): proarrhythmic effect of this drug. Following adenosine IV administration, PVCs or VT developed in near 50 to 65% of cases. All VT episodes are short (in this case is not short apparently because first AF +PVT), and self-terminating. often induces nonsustained VT or PVCs that are clinically insignificant in the absence of other accompanying heart disease.(3). Physicians are finding increased applications for adenosine as a diagnostic and therapeutic modality for a variety of cardiac dysrhythmias. Its short half life and lack of reported major complications make it an ideal pharmacologic agent to utilize for diagnosis and treatment.

Approach: Electrophysiological Programmed Studies followed by RFCA.

1. Goldberger JJ, Passman R, Arora R, A higher than expected prevalence of AV nodal reentrant tachycardia in patients receiving implantable cardioverter-defibrillators. Pacing Clin Electrophysiol. 2011 May;34:584-586.
2. Kaplan IV, Kaplan AV, Fisher JD. Adenosine induced atrial fibrillation precipitating polymorphic ventricular tachycardia. Pacing Clin Electrophysiol. 2000 Jan;23:140-141.
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CLASSIFICATION OF NARROW QRS PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA(PSVT)

I) Common Forms (Narrow QRS)

- **AV Nodal Reentrant Tachycardia (AVNRT) the so-called "common, usual, typical" form of AVNRT or Slow-Fast AVNRT:** utilizes the slow AV nodal pathway as the anterograde limb of the circuit and the fast AV nodal pathway as the retrograde limb. 50% to 60% of all PSVT cases. The retrograde P wave is seen in close proximity to the QRS complex.
- **Orthodromic Circus Movement Tachycardia. (CMT) or atrioventricular reciprocating tachycardia (AVRT):** supported by atrioventricular reentry circuit that use the AV node anterogradely and a rapidly conduction accessory pathway retrogradely

II) Uncommon Forms (Narrow QRS)

- **Fast-Slow AVNRT: AV nodal reentrant tachycardia "uncommon, unusual, atypical" form of AVNRT:** The reentry circuit is reversed: the fast AV nodal pathway is the anterograde limb and the slow AV nodal pathway is the retrograde limb: the retrograde P wave occurs late, within or following the T wave. RP' interval longer than the P'R interval. Negative P wave in inferior leads, positive or isoelectric in I and negative in V5-V6.
- **Slow-Slow AVNRT:** this utilizes the slow AV nodal pathway as the anterograde limb and left atrial fibers that approach the AV node from the left side of the inter-atrial septum as the retrograde limb.
- **Incessant Junctional reciprocating tachycardia** (atypical orthodromic circus movement tachycardia using a slow accessory pathway)
- **Sinoatrial (SA) nodal reentrant tachycardia**
- **Intraatrial reentrant tachycardia.**

AV Nodal Reentrant Tachycardia (AVNRT)

CONCEPT: regular paroxysmal tachyarrhythmia (sudden onset and end), with anatomical base in intranodal reentry or in perinodal atrial tissue by double conduction in the junctional region (longitudinal dissociation) by the existence of two pathways with different electrophysiological properties with differential refractoriness (α and β pathways) determining a rapid pathway (β) and a slow one (α), which causes functional unidirectional block, initially in the β pathway, by possessing a more prolonged refractory period and greater velocity: rapid or reciprocating pathway. Dual AV nodal physiology is a substrate for the development of AVNRT). As we see, this is a reciprocating rhythm, the initial impulse of which may originate in the sinus node, the atrial, the junction or the ventricles, which establishes a motion of atrioventricular activation as a consequence of the existence of the double nodal pathway.

Prevalence: 1 to 1,5 of 1000 persons

Gender differences: It is more common in women than men (approximately 75% of cases occurring in females). In the anterograde and retrograde AV nodal electrophysiology were noted in the patients with AVNRT. Women had a significantly younger age of onset, higher incidence of multiple jumps, shorter AH interval, atrial effective refractory period (ERP), anterograde fast pathway ERP, anterograde slow pathway ERP, and retrograde slow pathway ERP, and longer ventricular ERP than men. The incidence of baseline ventriculoatrial dissociation is lower in women than in men. Women needed less isoproterenol/atropine to induce AVNRT. Both typical and atypical AVNRT are more predominant in women. In incidence of baseline ventriculoatrial dissociation the retrograde slow pathway ERP is significantly shorter in women than in men. Women of premenopausal age (≤ 50 years old) have a significantly higher incidence of anterograde multiple jumps and a retrograde jump phenomenon, and a shorter anterograde slow pathway ERP and retrograde slow pathway ERP than those of women over 50 years old.(1)

1. Suenari K, Hu YF, Tsao HM, et al. Gender differences in the clinical characteristics and atrioventricular nodal conduction properties in patients with atrioventricular nodal reentrant tachycardia. J Cardiovasc Electrophysiol. 2010 Oct;21:1114-9.

In the common type of AVNRT the reentrant circuit consists of the slow pathway in the anterograde direction and the fast pathway retrogradely.

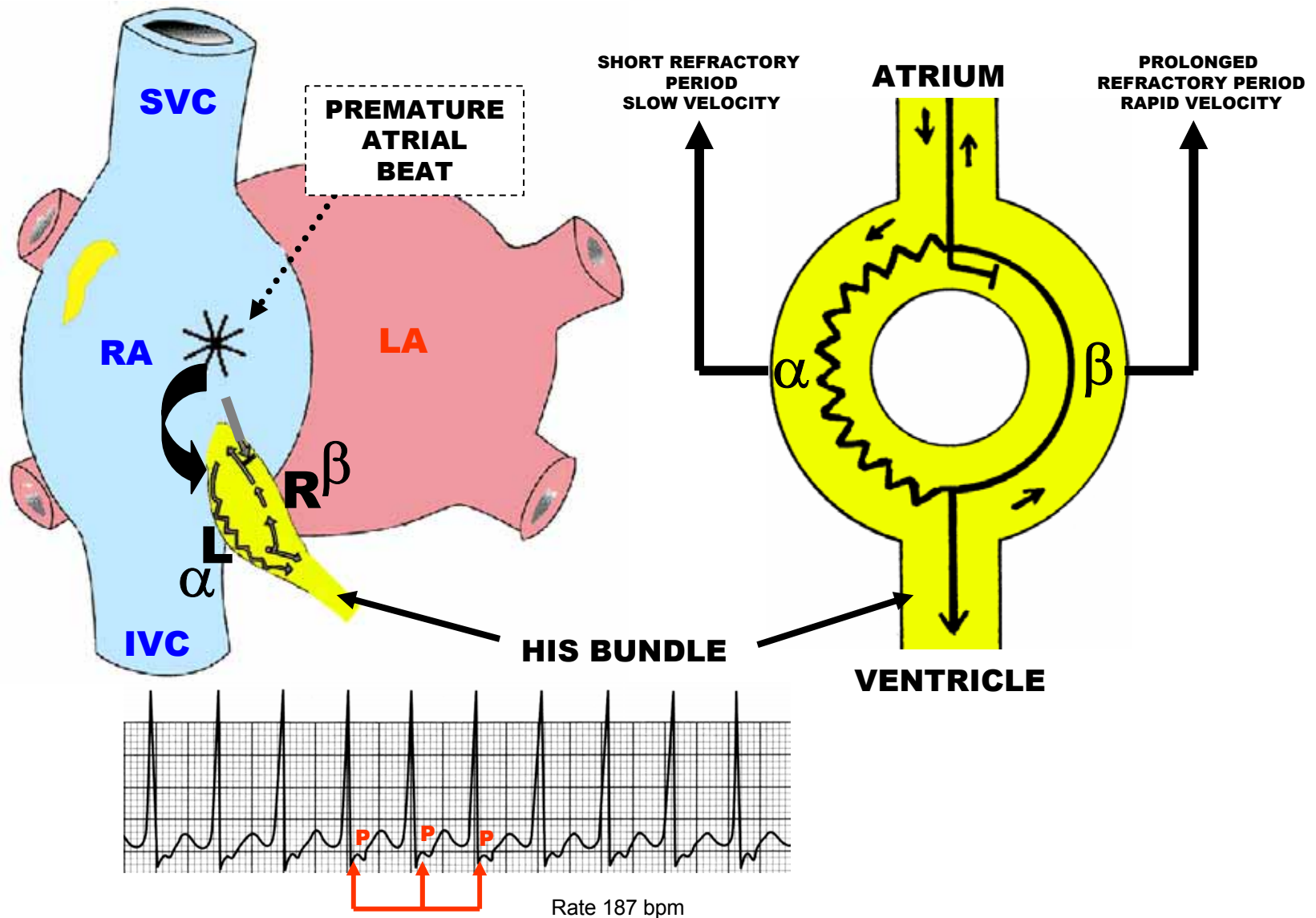
In the uncommon type of AVNRT the slow pathway is utilized retrograde and a relatively fast pathway is utilized in the anterograde direction.(1)

ETIOLOGIES OF AVNRT

1. Without underlying heart disease. The most frequent one;
2. Mitral valve prolapse;
3. Hyperthyroidism: an overactive thyroid increases the risk of AVNRT
4. Atherosclerotic coronary insufficiency;
5. Hypertensive heart disease;
6. Cardiomyopathies;
7. Pericarditis;
- 8) Electrolytic disturbances in potassium, calcium and magnesium may predispose to AVNRT
- 9) Congenital heart disease: ASD, Eisenmenger complex;
- 10) Gestational: gestation may trigger runs of PSVT as a first event in 34% of the cases, or it may increase the frequency of the crises in 28%. Estrogens, by increasing the number of peripheral autonomous receptors, increase the sensitivity to catecholamines, which are released in a greater degree in gestation, causing nodal reentry in 60% and macro-reentry by concealed or manifest accessory pathway (WPW) in 15% to 30%.

1. **Lelakowski J, Rydlewska A, Kuniewicz M. Atrioventricular nodal reentrant tachycardia-arrhythmias mechanism, clinical feature and electrocardiographic recordings.Pol Merkur Lekarski. 2010 Jun;28:429-437.**

PHYSIOPATHOGENIC MECHANISM OF ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA



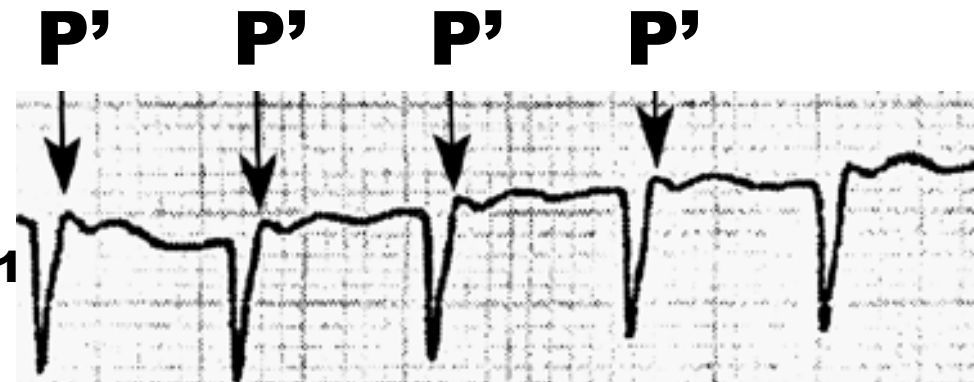
Pathophysiological mechanism

I) ECG RECOGNITION OF SLOW-FAST AVNRT (COMMON AVNRT)

1. Incidence: Accounts for 80-90% of all AVNRT.; **2. Mechanism:** Associated with Slow AV nodal pathway for anterograde conduction and Fast AV nodal pathway for retrograde conduction.; **3. Heart rate:** between 140 to 280 bpm.; **4. Rhythm:** regular or slightly irregular in cases of changing conduction velocities through the AV node.; **5. Location of the P wave:** The retrograde P wave is seen in close proximity to the QRS complex.; **6. The retrograde P wave is obscured in the corresponding QRS or occurs at the end of the QRS complex as pseudo r' in V1 or S waves in II, III, and aVF.** The P waves are often hidden – being embedded in the QRS complexes. Pseudo r' wave may be seen in V1 Pseudo S waves may be seen in leads II, III or aVF. In most cases this results in a 'typical' SVT appearance with absent P waves and tachycardia.



In the inferior leads, P' distorts the end of QRS, and it may originate a false S.



In V1, the P' wave may distort the end of QRS, resembling an r' or R': Pseudo R' or r'

II) ECG RECOGNITION OF FAST-SLOW NARROW ATYPICAL AVNRT (UNCOMMON AVNRT)

1. Accounts for 10% of **AVNRT**.;
2. **Heart rate:** 170 to 250bpm
3. **Rhythm:** Regular or slightly irregular in cases of changing conduction velocities through the AV node
4. Associated with Fast AV nodal pathway for anterograde conduction and Slow AV nodal pathway for retrograde conduction.
5. The retrograde P wave appears after the corresponding QRS. P waves are visible between the QRS and T wave. RP' interval longer than the P'R interval.
6. Negative P wave in inferior leads II, III, and Avf and in V5-V6. P positive or isoelectric in I. (Positive P wave in lead I differentiates atypical AVNRT from incessant junctional tachycardia.)
7. **Natural history:** usually benign and self-limiting. Usually terminated by a vagal maneuver
8. **Management:** if the tachycardia cannot be controlled RFCA of the slow atrionodal pathway.

III) ECG RECOGNITION OF SLOW-SLOW AVNRT (ATYPICAL AVNRT)

1. 1-5% AVNRT. ;
2. Associated with Slow AV nodal pathway for anterograde conduction and Slow left atrial fibres approaching the AV node as the pathway for retrograde conduction.;
3. ECG: Tachycardia with a P-wave seen in mid-diastole... effectively appearing 'before the QRS complex'...
4. Confusing as a P wave appearing before the QRS complex in the face of a tachycardia might honestly be read as a sinus tachycardia.

INVESTIGATIONS

ELECTROCARDIOGRAM

The ECG will typically show a tachycardia of 140-280 bpm with normal and regular QRS complexes. The P/QRS time ratio is very important and it may be:

No visible P-waves (hidden within the QRS complex) P concomitant with QRS: 60% of the cases. Atria and ventricles activated simultaneously. P remains concealed by QRS or it may modify the onset of the complex (30%). In these cases, the esophageal lead enables to identify the P wave and the R-P' interval (V-A time) always <70 ms. **or**

P-waves immediately before the QRS or

P-waves immediately after the QRS complex P preceding QRS: 3%. P waves of retrograde activation precede QRS complexes, which may resemble q waves in inferior leads.

For recurrent episodes of palpitations, a Holter monitor and EPS may be useful in identifying rhythms typical of AVNRT.

The polarity of ectopic P that triggers tachycardia (the first one) is different from the rest of subsequent Ps: if its origin is atrial, the polarity is positive in inferior leads, while posterior Ps are of inverted polarity because their activation is retrograde. This element is important to differentiate it from automatic paroxysmal tachycardia.

ECHOCARDIOGRAM

An echocardiogram may be useful in evaluating for structural heart disease

ELECTROPHYSIOLOGICAL STUDIES

Electrophysiological studies may be necessary if considering ablative therapy.

BLOOD TESTS

Blood tests that may be appropriate in patients experiencing palpitations include cardiac markers (to investigate for myocardial infarction), urea and electrolytes (to identify imbalances in potassium, magnesium or calcium) or thyroid function tests (hyperthyroidism may trigger AVNRT or other arrhythmias).

Clinical Features of AVNRT

AVNRT is typically paroxysmal and may occur spontaneously in patients or upon provocation with exertion, coffee, tea or alcohol.

It is more common in women than men (~75% of cases occurring in women) and may occur in young and healthy patients as well as those suffering chronic heart disease.

Patients will typically complain of the sudden onset of rapid, regular palpitations.

The patient may experience a brief fall in blood pressure causing presyncope or occasionally syncope.

If the patient has underlying coronary artery disease the patient may experience chest pain similar to angina (tight band around the chest radiating to left arm or left jaw).

The patient may complain of shortness of breath, anxiety and occasionally polyuria due to elevated atrial pressure releasing atrial natriuretic peptide.

The tachycardia typically ranges between 140-280 bpm and is regular in nature. It may cease spontaneously (and abruptly) or continue indefinitely until medical treatment is sought.

The condition is generally well tolerated and is rarely life threatening in patients with pre-existing heart disease.

MANAGEMENT

Patients may be instructed to undertake vagal manoeuvres upon the onset of symptoms which can be effective in stopping the AVNRT. This may involve carotid sinus massage or valsalva manoeuvres, which will both stimulate the vagus nerve. Alternative strategies include:

Adenosine, beta-blockers or calcium channel blockers can suppress an AVNRT event by blocking or slowing the AV node.

Other second-line therapies may include amiodarone or flecainide.

Cardioversion is rarely used on patients with AVNRT, usually when the tachycardia is refractory to other medical therapies or the tachycardia is causing haemodynamic instability (falling blood pressure, development of heart failure etc.)

Radiofrequency catheter ablation (RFCA) can be offered to patients with frequent attacks for whom medical therapy isn't appropriate in the long term, and can be curative. RFCA for AVNRT is highly successful but carries a risk for inadvertent atrioventricular block. Cryoablation (cryo) has the potential to assess the safety of a site before the energy is applied. Cryo and RFCA with 4-mm tip catheters for AVNRT are equally effective, even after long-term follow-up.(1)

- 1. Schwagten B, Knops P, Janse P, et al; Long-term follow-up after catheter ablation for atrioventricular nodal reentrant tachycardia: a comparison of cryothermal and radiofrequency energy in a large series of patients. J Interv Card Electrophysiol. 2011 Jan;30:55-61.**

Atrioventricular Reciprocating Tachycardia, AV reentrant tachycardia (AVRT) or Orthodromic Circus Movement Tachycardia. (CMT)

AVRT is the second most common form of PSVT.

Incidence: The incidence rate of AVRT in the general population is 0.1-0.3%.

Gender: AVRT is more common in males than in females (male-to-female ratio of 2:1),

Age: patients with **AVRT** commonly present at a younger age than patients with **AVNRT**.

Structural heart disease in association: AVRT is associated with the Ebstein anomaly, although most patients with **AVRT** do not have evidence of structural heart disease. **AVRT** occurs in the presence of accessory pathways, or bypass tracts. Accessory pathways are errant strands of myocardium that bridge the mitral or tricuspid valves.(1;2;3;4)

AVRT is the result of 2 or more conducting pathways: the AV node and 1 or more bypass tracts. In a normal heart, only a single route of conduction is present. Conduction begins at the sinus node, progresses to the AV node, and then to the bundle of His and the bundle branches.

1. Josephson ME, Kastor JA. Supraventricular tachycardia: mechanisms and management. *Ann Intern Med.* Sep 1977;87:346-358
2. Murdock CJ, Leitch JW, Teo WS, Sharma AD, Yee R, Klein GJ. Characteristics of accessory pathways exhibiting decremental conduction. *Am J Cardiol.* Mar 1 1991;67:506-510.
3. Ganz LI, Friedman PL. Supraventricular tachycardia. *N Engl J Med.* Jan 19 1995;332:162-173.
4. Xie B, Thakur RK, Shah CP, Hoon VK. Clinical differentiation of narrow QRS complex tachycardias. *Emerg Med Clin North Am.* May 1998;16:295-330

However, in **AVRT**, 1 or more accessory pathways connect the atria and the ventricles. The accessory pathways may conduct impulses in an anterograde manner, a retrograde manner, or both.(1). When impulses travel down the accessory pathway in an anterograde manner, ventricular preexcitation results. This produces a short PR interval and a delta wave as is observed in persons with Wolff-Parkinson-White (WPW) syndrome. Importantly, note that not all accessory pathways conduct in an anterograde manner. Concealed accessory pathways are not evident during sinus rhythm, and they are only capable of retrograde conduction. A reentry circuit is most commonly established by impulses traveling in an anterograde manner through the AV node and in a retrograde manner through the accessory pathway; this is called orthodromic **AVRT**. A reentry circuit may also be established by a premature impulse traveling in an anterograde manner through a manifest accessory pathway and in a retrograde manner through the AV node; this is called antidromic **AVRT**. Patients with WPW syndrome can develop AF and atrial flutter. The rapid nondecremental conduction via the accessory pathways can result in extremely rapid rates, which can degenerate to VF and cause SD. Patients with preexcitation syndromes with AF must not be administered an AV nodal blocking agent; these agents can further increase conduction via the accessory pathway, which increases the risk of VF and SD. (2;3;4;5)

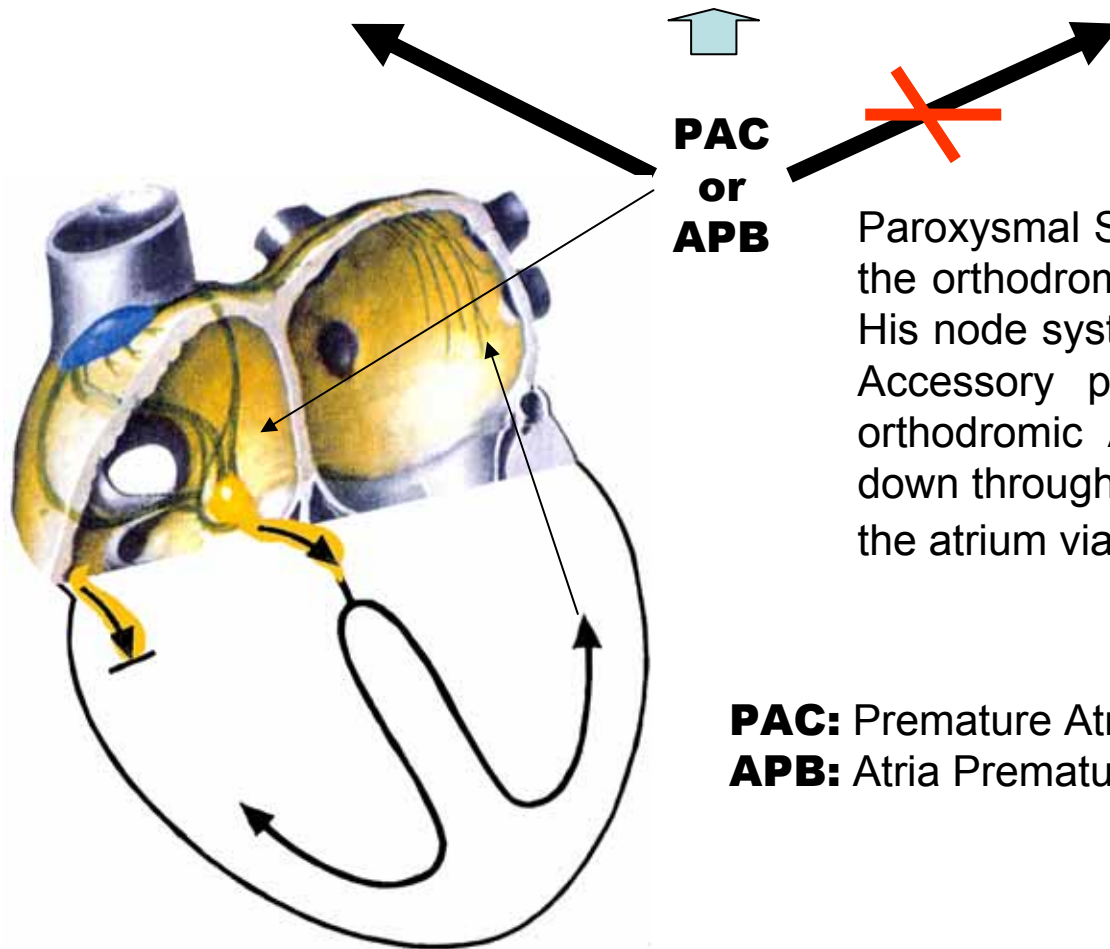
1. Wolff L, Parkinson J, White PD. Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. *Am Heart J*. 1930;5:685-704.
2. Oren JW 4th, Beckman KJ, McClelland JH, Wang X, Lazzara R, Jackman WM. A functional approach to the preexcitation syndromes. *Cardiol Clin*. Feb 1993;11:121-49.
3. Wolff L, Parkinson J, White PD. Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. *Am Heart J*. 1930;5:685-704.
4. Bardy GH, Packer DL, German LD, Gallagher JJ. Preexcited reciprocating tachycardia in patients with Wolff-Parkinson-White syndrome: incidence and mechanisms. *Circulation*. Sep 1984;70(3):377-91.
5. Obel OA, Camm AJ. Supraventricular tachycardia. ECG diagnosis and anatomy. *Eur Heart J*. May 1997;18 Suppl C:C2-11

ORTHODROMIC CIRCUS MOVEMENT TACHYCARDIA

This is the second most frequent form of PSVT with narrow QRS complex. It is usually

Initiated by an Premature Atrial Contraction (PAC) and the following structures are activated in sequence;

Atria → AV node → Ventricles → Accessory Pathway → Atrium



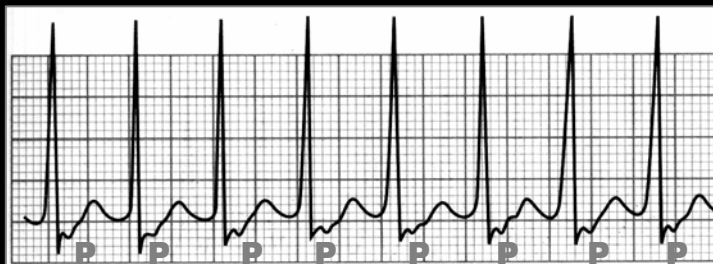
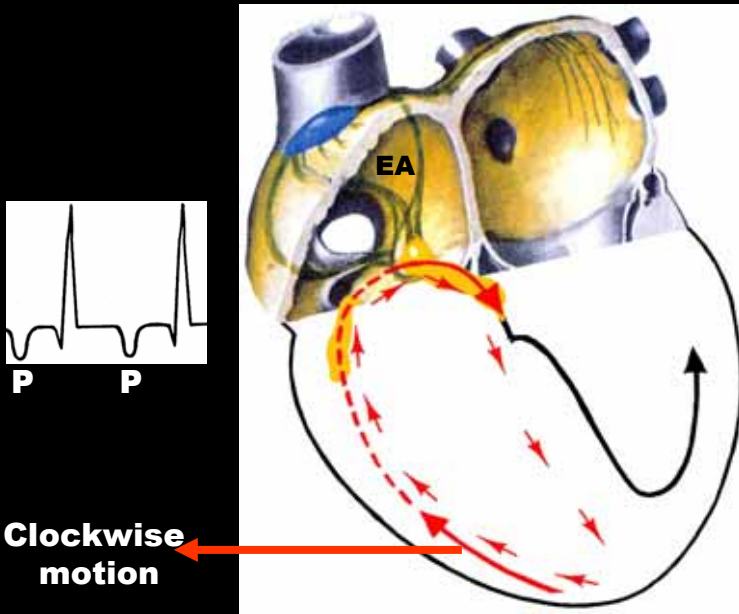
**PAC
or
APB**

Paroxysmal Supraventricular Tachycardia (PSVT) of the orthodromic macro-reentry type, which uses the His node system in an anterograde fashion, and the Accessory pathway in a retrograde fashion. In orthodromic AVRT, atrial impulses are conducted down through the AV node and retrogradely re-enter the atrium via the accessory pathway.

PAC: Premature Atrial Contraction
APB: Atria Premature Beat

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIAS (PSVT) BY RECIPROCAL MACRO-REENTRY OR REENTRANT IN WPW

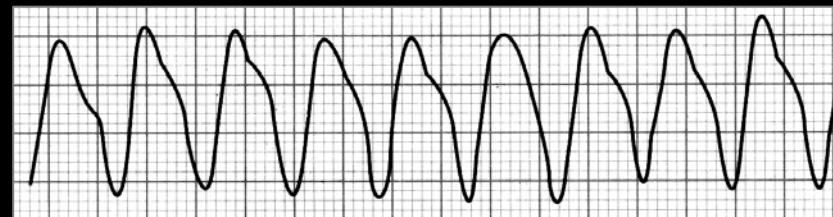
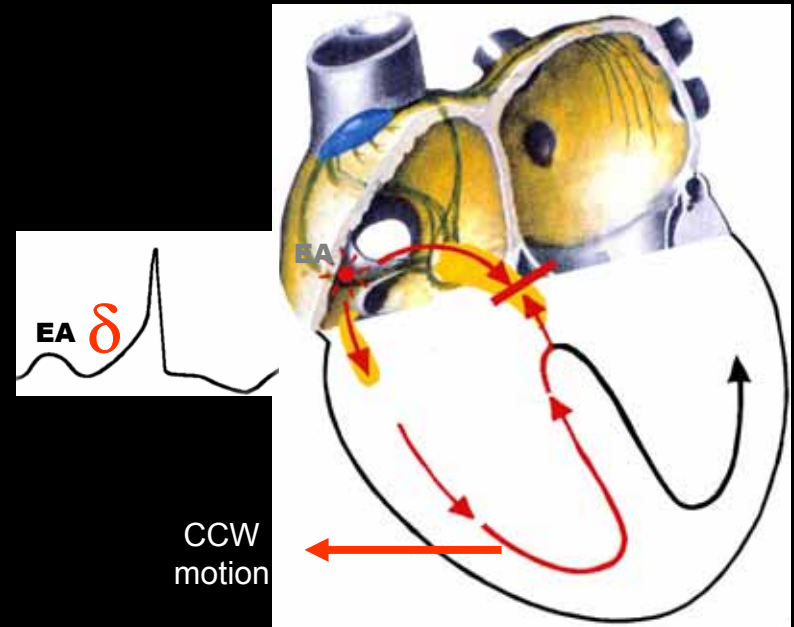
Orthodromic or with narrow QRS: 90%



Narrow QRS.

Clockwise macro-reentry motion.
It uses a normal pathway in anterograde fashion
and/or anomalous bundle in retrograde fashion.

Antidromic or with wide QRS: 10%



Wide QRS.

Counterclockwise macro-reentry motion.
It uses a normal pathway in retrograde fashion
and/or anomalous bundle in anterograde fashion.

Outline of the two modalities of macro-reentry in ventricular pre-excitation.

ELECTROCARDIOGRAM IN AVRT OR CMT

- **Heart rate:** 170 to 290bpm
- **Rhythm:** Regular, but may be slightly irregular because of changing conduction through the AV node
- **Begins and end:** Abruptly
- **Initial P'R interval:** no prolonged
- **Location of P' wave:** closely follow QRS complex. Always separated from QRS. A distinguishing characteristic of orthodromic AVRT can therefore be a P-wave that follows each of its regular, narrow QRS complexes, due to retrograde conduction.
- **P wave polarity:** Variable, depending on the accessory bundle location. If the P wave is negative in I, the diagnosis is left accessory bundle.
- **QRS complex:** Narrow
- **Conduction ratio:** Always 1:1
- **Aberrant ventricular conduction:** Common
- **Heart rate during aberrancy:** Slower with aberrancy than without if the accessory pathway is on the same side as the BBB; this is a diagnostic sign ; if accessory pathway is located on opposite ventricle from the BBB, rate is the same with and without aberrancy.
- **QRS alternans:** Common

ECG diagnosis: paroxysmal narrow QRS complex tachycardia (PSVT):

242 ECGs demonstrating narrow PSVT (< 110 ms and HR \geq 120 beats/min) were analyzed by Kalbfleisch et al(1). All ECGs were analyzed by an observer who had no knowledge of the mechanism of the tachycardia. There were 137 atrioventricular (AV) reciprocating tachycardias, 93 **AVNRT** and 12 atrial tachycardias. Six criteria were found to be significantly different between tachycardia types by univariate analysis:

- 1) A P wave separate from the QRS complex was observed more frequently in **AVRT** (68%) and atrial tachycardias (75%).
- 2) A pseudo r' deflection in lead V₁ and a pseudo S wave in the inferior leads were more common in **AVNRT** (58% and 14%, respectively);
- 3) QRS alternans was present more often during AV reciprocating tachycardia **AVRT** (27%).
- 4) When a P wave was present, an RP/PR interval ratio ≥ 1 was more common in atrial tachycardias (89%).
- 5) During sinus rhythm, manifest pre-excitation was observed more often in patients with AV reciprocating tachycardia **AVRT** (45%).
- 6) By multivariate analysis, the presence of a P wave separate from the QRS complex, pseudo r' deflection in lead V₁, QRS alternans during tachycardia and the presence of pre-excitation during sinus rhythm were independent predictors of narrow PSVT type. These criteria correctly identified 86% of **AVNRT**, 81% of **AVRT** and incorrectly assigned the tachycardia type in 19% of cases.

The authors concluded that several features on the ECG are useful for differentiating narrow PSVT type. However, approximately 20% of tachycardias may be incorrectly classified on the basis of analysis of the ECG; therefore, the ECG should not serve as the sole means for determining tachycardia mechanism(1).

1. Kalbfleisch SJ, el-Atassi R, Calkins H, et al. Differentiation of paroxysmal narrow QRS complex tachycardias using the 12-lead electrocardiogram. J Am Coll Cardiol. 1993 Jan;21:85-89.

Studies have shown that only 80% of narrow PSVT types can be differentiated by standard 12-lead ECG) criteria. Erdinler et al(1) studied the value of some new ECG criteria in differentiating narrow QRS SVT.

120 ECGs demonstrating paroxysmal narrow QRS complex tachycardia ($QRS \leq 0.11$ ms and rate > 120 beats/min) were analyzed. 40 atrioventricular reciprocating tachycardia (AVRT), 70 atrioventricular nodal reentrant tachycardia (AVNRT), and 10 atrial tachycardia defined with electrophysiologic study (EPS) consisted the study group. 8 surface ECG criteria were found to be significantly different between tachycardia types by univariate analysis. P waves separate from the QRS complex were observed more frequently in AVRT (70%) and atrial tachycardia (80%). Pseudo r' deflection in lead V(1), pseudo S wave in inferior leads, and cycle length alternans were more common in AVNRT (55, 20, and 6%, respectively). QRS alternans was also present during AVRT (28%). ST-segment depression (≥ 2 mm) or T-wave inversion, or both, were present more often in AVRT (60%) than in AVNRT (27%). During sinus rhythm, manifest preexcitation was observed more often in patients with AVRT (42%). When a P wave was present, RP/PR interval ratio > 1 was more common in atrial tachycardia (90%).

By multivariate analysis, presence of a P wave separate from the QRS complex, pseudo r' deflection in lead V(1), QRS alternans, preexcitation during sinus rhythm, ST-segment depression > 2 mm or T-wave inversion, or both, were independent predictors of tachycardia type.

Several new ECG criteria may be useful in differentiation of SVT types. Prediction of mechanism prior to EPS may provide additional benefits concerning the fluoroscopic exposure time and cardiac catheterization procedure.

- 1. Erdinler I, Okmen E, Oguz E, et al. Differentiation of narrow QRS complex tachycardia types using the 12-lead electrocardiogram. Ann Noninvasive Electrocardiol. 2002 Apr;7:120-126.**

Letsas et al(1) evaluate the diagnostic value of specific ECG markers in the differentiation of common type atrioventricular nodal reentrant tachycardia (**AVNRT**) and atrioventricular reentrant tachycardia (**AVRT**) via a concealed accessory pathway.

110 ECGs with paroxysmal narrow PTV, short RP tachycardia were evaluated. Subjects with overt ventricular pre-excitation during sinus rhythm were excluded from the study. The mechanism of arrhythmia was established during the EPS and confirmed by the efficacy of RFCA.

Of the 110 patients, 74 displayed common type **AVNRT** and 36 **AVRT**. Predictors of **AVNRT** were the presence of pseudo r'-waves in lead V₁ [sensitivity 39.19%; specificity 97.14%; positive predictive value (PPV) 96.67%; negative predictive value (NPV) 43.04%] and pseudo S-waves in inferior leads (sensitivity 28.38%; specificity 94.29%; PPV 91.30%; NPV 38.37%). In the setting of visible P-waves, an RP interval ≤90 ms favored the diagnosis of **AVNRT** (sensitivity 57.14%; specificity 80.65%). Predictors of **AVRT** were QRS alternans (sensitivity 50%; specificity 89.19%; PPV 69.23%; NPV 78.57%) as well as ST-segment alterations during tachycardia. The overall sensitivity, specificity, PPV and NPV of ST-segment depression for discriminating **AVRT** from **AVNRT** were 97.22%, 58.11%, 53.03%, 97.73%, respectively. Similarly, the sensitivity, specificity, PPV and NPV of ST-segment elevation in lead aVR were 94.44%, 58.11%, 52.31%, and 95.56%, respectively. Multiple logistic regression analysis showed that ST-segment depression [(odds ratio (OR): 12.67, 95% confidence interval (CI): 1.77-90.81, P = 0.011)] and QRS alternans (OR: 9.43, 95% CI: 1.38-64.37, P = 0.022) displayed the highest predictive ability favoring the diagnosis of **AVRT**.

Twelve-lead ECG parameters may help to differentiate the mechanism of supraventricular tachycardia prior to the RFCA procedure.

1. Letsas KP, Weber R, Siklody CH, et al. Electrocardiographic differentiation of common type atrioventricular nodal reentrant tachycardia from atrioventricular reciprocating tachycardia via a concealed accessory pathway. *Acta Cardiol*. 2010 Apr;65:171-176.

The noninvasive differentiation between **AVNRT** and **AVRT** mediated by concealed accessory pathway conduction is clinically important, as it helps in counseling and potentially facilitates RFCA. 148 ECGs showing narrow QRS complex SVT were obtained from children before successful RFCA. An initial 102 ECGs were analyzed by 3 blinded observers to assess the utility of various ECG findings. No ECG criteria were found to discriminate between SVT mechanisms on 1- to 3-channel Holter/event recorder tracings (n = 32); their interpretation mainly (55%) resulted in an incorrect SVT diagnosis. On 12-lead ECGs (n = 70), the 2 arrhythmias were accurately diagnosed in 76% of patients; 5 findings were found to be discriminators of tachycardia mechanism. Predictors of **AVRT** were:

1. Visible P waves in 74% of cases (sensitivity 92%; specificity 64%),
2. RP intervals of ≥ 100 ms in 91% (sensitivity 84%; specificity 91%),
3. ST-segment depression of ≥ 2 mm in 73% of cases (sensitivity 52%; specificity 82%).
4. Pseudo r' waves in lead V₁
5. Pseudo S waves in the inferior leads during the event predicted **AVNRT** in 100% of cases (sensitivity 55% and 20%, respectively; specificity 100% for both).

Based on these results, Jaeggi et al (1) developed a diagnostic ECG algorithm for pseudo r'/S waves, RP duration, and ST-segment depression during tachycardia. Two observers tested the algorithm in 46 (21 **AVNRT**; 25 **AVRT**) additional cases; they correctly diagnosed the SVT mechanism in 91% and 87%, respectively. Thus, the stepwise use of diagnostically relevant ECG parameters helps to more accurately differentiate mechanisms of reentrant SVT.

1. Jaeggi ET, Gilljam T, Bauersfeld U, Chiu C, Gow R. **Electrocardiographic differentiation of typical atrioventricular node reentrant tachycardia from atrioventricular reciprocating tachycardia mediated by concealed accessory pathway in children.** Am J Cardiol. 2003 May 1;91:1084-1089.

DIFFERENTIAL DIAGNOSIS BETWEEN ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA (AVNRT) AND ATRIOVENTRICULAR REENTRANT TACHYCARDIA (AVRT) OR CIRCUS MOVEMENT TACHYCARDIA (CMT)

ECG SIGNS	AVNRT	AVRT
QRS complexes alternation	Rare	Common
Initial P'R	Prolonged	Normal
Location of P' wave	Within QRS. It may cause pseudo S wave in inferior leads or pseudo r' wave in V ₁	Always separated from QRS
P' wave polarity	Negative in II, III and aVF.	Variable, depending on the accessory bundle location. If the P wave is negative in I, the diagnosis is left accessory bundle.
Aberrance	Rare	Common
Heart rate during aberrance compared with non-aberrance	It is not modified	It may decrease with aberrance; branch block on the same side of the accessory bundle
AV conduction	Usually 1:1	Always 1:1

Incessant Junctional reciprocating tachycardia (atypical orthodromic circus movement tachycardia using a slow accessory pathway) Permanent junctional reciprocating tachycardia (PJRT)

Junctional reciprocating tachycardia is an atrioventricular reentrant tachycardia whose anterograde conduction occurs via the His Purkinje and the retrograde conduction via an accessory pathway with slow conduction. The accessory pathways are typically located in the posteroseptal region. The most common form is incessant tachycardia but a paroxysmal form also exists. Permanent junctional reciprocating tachycardia (PJRT) is an incessant or almost incessant supraventricular tachycardia with a long RP interval, usually occurring in children and young adults.

ECG CHARACTERIZATION

Heart rate: 130 to 200bpm

Rhythm: Regular

Location of P' wave: $RP' > P'R$

P wave polarity: negative in II, III, aVF, V5 and V6

QRS duration: narrow

Main characteristic:

1. The most common form is permanent or almost incessant narrow QRS tachycardia but a paroxysmal form also exists. Paroxysmal
2. Negative P waves in II, III, aVF, V5 and V6
3. $RP' > P'R$
4. Present more than 12hours a day
5. The persistent nature of the tachycardia can lead to heart failure secondary to left ventricular dysfunction and the arrhythmia is often refractory to drug control. Result of tachycardia-related cardiomyopathy

The differential diagnosis of PJRT includes an atrial tachycardia and atypical atrioventricular nodal reentrant tachycardia (AVNRT). (1)

PJRT is an infrequent form of reciprocating tachycardia, almost incessant from childhood and usually refractory to drug therapy.

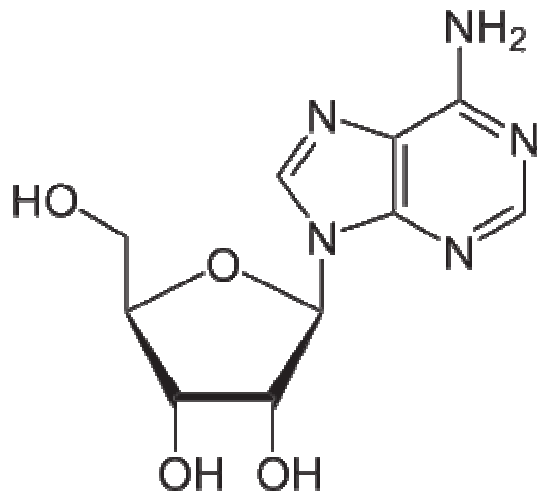
PJRT is the commonest incessant tachycardia in childhood.

RFCA currently is the first-line therapy for PJRT, but its application in the septal region may be associated with complications. In contrast, cryoenergy has several advantages, such as the ability to test the effects of ablation while the lesion is still forming, thus reducing the number of ineffective, useless, and potentially harmful lesions. Cryoablation is a safe, effective, and pain-free technique for treating pediatric patients with PJRT.(2)

1. **Kose S, Iyisoy A, Barcin C. A permanent junctional reciprocating tachycardia with atypical location, treated with radiofrequency catheter ablation. Acta Cardiol. 2002 Oct;57:371-375.**
2. **Gaita F, Montefusco A, Riccardi R, et al. Cryoenergy catheter ablation: a new technique for treatment of permanent junctional reciprocating tachycardia in children. J Cardiovasc Electrophysiol. 2004 Mar;15:263-268.**

ADENOSINE

Adenosine is an endogenous nucleoside occurring in all cells of the body. It is chemically 6-amino-9-β-D-ribofuranosyl-9-H-purine and has the following structural formula:



Adenosine is a white crystalline powder. It is soluble in water and practically insoluble in alcohol. Solubility increases by warming and lowering the pH. Adenosine is not chemically related to other antiarrhythmic drugs. Adenosine injection, USP is a sterile, nonpyrogenic solution for rapid bolus intravenous injection. Each mL contains 3 mg Adenosine and 9 mg sodium chloride in water for injection. The pH of the solution is between 4.5 and 7.5.

Adenosine is a purine nucleoside comprising a molecule of adenine attached to a ribose sugar molecule (ribofuranose) moiety via a β-N9-glycosidic bond.

Adenosine plays an important role in biochemical processes, such as energy transfer—as adenosine triphosphate (ATP) and adenosine diphosphate (ADP)—as well as in signal transduction as cyclic adenosine monophosphate, cAMP. It is also an inhibitory neurotransmitter, believed to play a role in promoting sleep and suppressing arousal, with levels increasing with each hour an organism is awake.

Adenosine - Clinical Pharmacology

Mechanism of Action: Adenosine injection slows conduction time through the AV node, can interrupt the reentry pathways through the AV node, and can restore normal sinus rhythm in patients with PSVT, including PSVT associated with WPW-S. This is mediated via the A1 receptor, inhibiting adenylyl cyclase, reducing cAMP and so causing cell hyperpolarization by increasing outward K⁺ flux. It also causes endothelial dependent relaxation of smooth muscle as is found inside the artery walls. This causes dilatation of the "normal" segments of arteries; i.e. where the endothelium is not separated from the tunica media by atherosclerotic plaque. This feature allows physicians to use adenosine to test for blockages in the coronary arteries, by exaggerating the difference between the normal and abnormal segments. Adenosine is antagonized competitively by methylxanthines such as caffeine and theophylline, and potentiated by blockers of nucleoside transport such as dipyridamole. Adenosine is not blocked by atropine. Adenosine is an endogenous purine nucleotide that modulates many physiological processes. Cellular signaling by adenosine occurs through four known adenosine receptor subtypes (A1, A2A, A2B, and A3). Extracellular adenosine concentrations from normal cells are approximately 300 nM; however, in response to cellular damage (e.g. in inflammatory or ischemic tissue), these concentrations are quickly elevated (600–1,200 nM). Thus, in regard to stress or injury, the function of adenosine is primarily that of cytoprotection preventing tissue damage during instances of hypoxia, ischemia, and seizure activity. Activation of A2A receptors produces a constellation of anti-inflammatory response. The different adenosine receptor subtypes (A1, A2A, A2B, and A3) are all seven transmembrane spanning G-protein coupled receptors. These four receptor subtypes are further classified based on their ability to either stimulate or inhibit adenylyl cyclase activity.

The A1 and A2A receptors of endogenous adenosine are believed to play a role in regulating myocardial oxygen consumption and coronary blood flow. Stimulation of the A1 receptor has a myocardial depressant effect by decreasing the conduction of electrical impulses and suppressing pacemaker cell function, resulting in a decrease in heart rate.

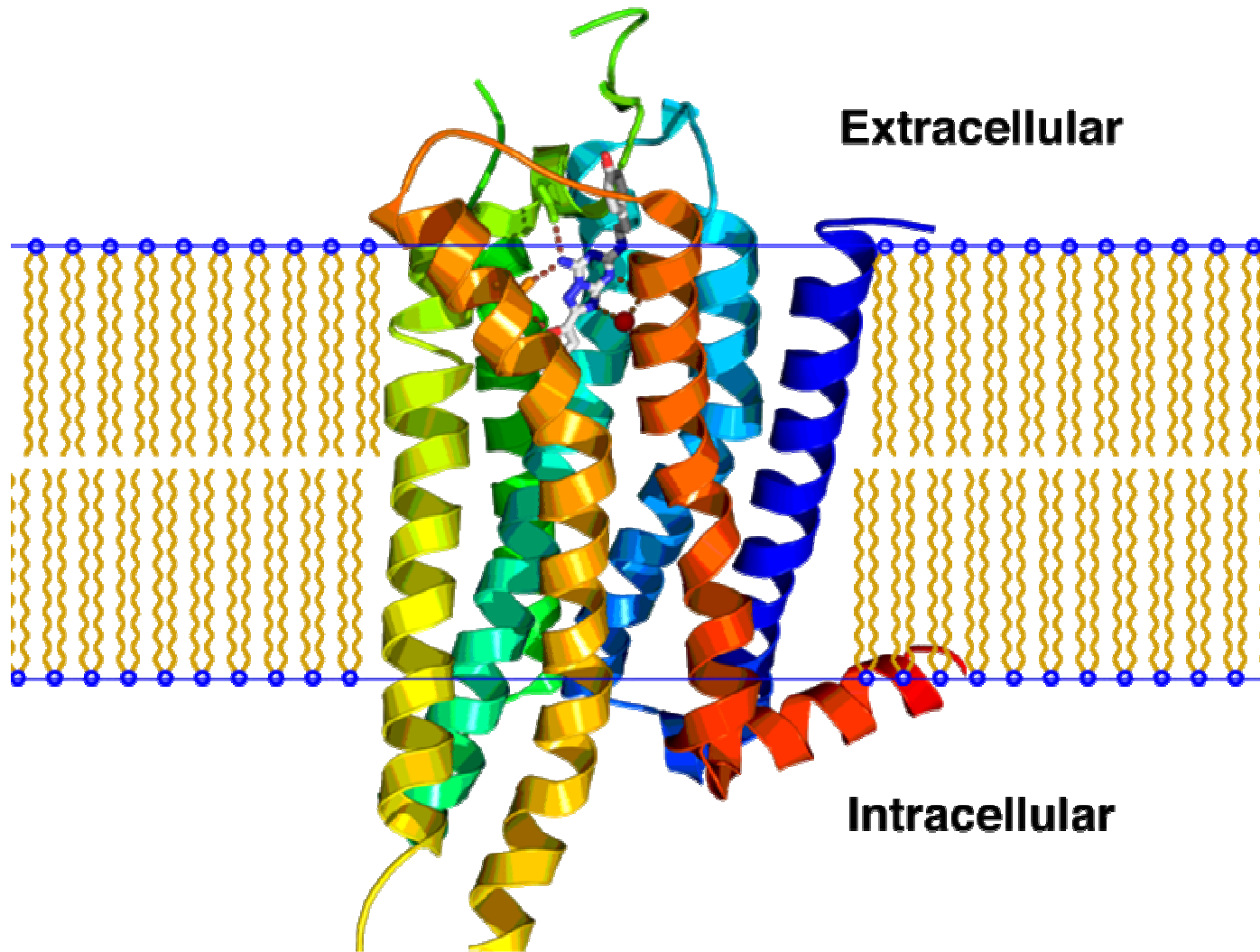
This makes adenosine a useful medication for treating and diagnosing *tachyarrhythmias*, or excessively fast heart rates. This effect on the A1 receptor also explains why there is a brief moment of cardiac standstill when adenosine is administered as a rapid IV push during cardiac resuscitation. The rapid infusion causes a momentary myocardial stunning effect.

In normal physiological states, this serves as protective mechanisms. However, in altered cardiac function, such as hypoperfusion caused by hypotension, heart attack or cardiac arrest caused by nonperfusing bradycardias, adenosine has a negative effect on physiological functioning by preventing necessary compensatory increases in heart rate and blood pressure that attempt to maintain cerebral perfusion. All four adenosine receptor subtypes have been shown to play a role in cardioprotection, and there is evidence that all four subtypes may be expressed in cardiomyocytes. There is also increasing evidence that optimal adenosine cardioprotection requires the activation of more than one receptor subtype. Both adenosine A(2A) and A(2B) receptors are required for adenosine A(1) receptor-mediated cardioprotection, implicating a role for interactions among receptor subtypes.(1)

Selective adenosine A1 receptor antagonists targeting renal microcirculation are novel pharmacologic agents that are currently under development for the treatment of acute HF and chronic HF. Despite several studies showing improvement of renal function and/or increased diuresis with adenosine A1 antagonists, particularly in CHF, these findings were not confirmed in a large phase III trial in acute HF patients. The role of adenosine A1 receptors in the regulation of renal function, is emerging data regarding the safety and efficacy of A1 adenosine receptor antagonists based on all available completed and reported clinical trials using A1 adenosine receptor antagonists. The majority of trials were done in HF patients. However, there is clear clinical evidence for a role of this new class in hepatorenal syndrome, hypotension on dialysis, and radiocontrast media-induced nephropathy.

1. Zhan E, McIntosh VJ, Lasley RD. Adenosine A2A and A2B receptors are both required for adenosine A1 receptor-mediated cardioprotection. *Am J Physiol Heart Circ Physiol*. 2011 Sep;301:H1183-9.

Adenosine A2A receptor



As with the A1, the A2A receptors are believed to play a role in regulating myocardial oxygen consumption and coronary blood flow. In addition, A2A receptor can negatively regulate overreactive immune cells, thereby protecting tissues from collateral inflammatory damage. The A2A receptor is responsible for regulating myocardial blood flow by vasodilating the coronary arteries, which increases blood flow to the myocardium, but may lead to hypotension. Just as in A1 receptors, this normally serves as a protective mechanism, but may be destructive in altered cardiac function.

The A2A and A2B receptors couple to Gs and mediate the stimulation of adenylate cyclase, while the A1 and A3 adenosine receptors couple to Gi which inhibits adenylate cyclase activity. Additionally, A1 receptors couple to Go, which has been reported to mediate adenosine inhibition of Ca²⁺ conductance, whereas A2B and A3 receptors also couple to Gq and stimulate phospholipase activity. Adenosine is believed to be an anti-inflammatory agent at the A(2A) receptor. Topical treatment of adenosine to foot wounds in diabetes mellitus has been shown in lab animals to drastically increase tissue repair and reconstruction. Topical administration of adenosine for use in wound healing deficiencies and diabetes mellitus in humans is currently under clinical investigation. Methotrexate's anti-inflammatory effect may be due to its stimulation of adenosine release.

In individuals suspected of suffering from SVT, adenosine is used to help identify the rhythm. Certain SVTs can be successfully terminated with adenosine. This includes any re-entrant arrhythmias that require the AV node for the re-entry (e.g., AVRT, AVNRT). In addition, atrial tachycardia can sometimes be terminated with adenosine. Adenosine has an indirect effect on atrial tissue causing a shortening of the refractory period. When administered via a central lumen catheter, adenosine has been shown to initiate AF because of its effect on atrial tissue. In individuals with accessory pathways, the onset of AF can lead to a life-threatening VF. Fast rhythms of the heart that are confined to the atria (e.g., AF, atrial flutter) or ventricles (e.g., MVT) and do not involve the AV node as part of the re-entrant circuit are not typically converted by adenosine. However, the ventricular response rate is temporarily slowed with adenosine in such cases. Because of the effects of adenosine on AV node-dependent SVTs, adenosine is considered a class IV antiarrhythmic agent. When adenosine is used to cardiovert an abnormal rhythm, it is normal for the heart to enter ventricular asystole for a few seconds. This can be disconcerting to a normally conscious patient, and is associated with angina-like sensations in the chest. Caffeine's principal mode of action is as an antagonist of adenosine receptors in the brain.

By nature of caffeine's purine structure it binds to some of the same receptors as adenosine. The pharmacological effects of adenosine may therefore be blunted in individuals who are taking large quantities of methylxanthines (e.g., caffeine, found in coffee and tea, or theobromine, as found in chocolate).

HEMODYNAMICS

The intravenous bolus dose of 6 or 12 mg Adenosine injection usually has no systemic hemodynamic effects. When larger doses are given by infusion, Adenosine decreases blood pressure by decreasing peripheral resistance.

PHARMACOKINETICS

Intravenously administered Adenosine is rapidly cleared from the circulation via cellular uptake, primarily by erythrocytes and vascular endothelial cells. This process involves a specific transmembrane nucleoside carrier system that is reversible, nonconcentrative, and bidirectionally symmetrical. Intracellular Adenosine is rapidly metabolized either via phosphorylation to Adenosine monophosphate by Adenosine kinase, or via deamination to inosine by Adenosine deaminase in the cytosol. Since Adenosine kinase has a lower K_m and V_{max} than Adenosine deaminase, deamination plays a significant role only when cytosolic Adenosine saturates the phosphorylation pathway. Inosine formed by deamination of Adenosine can leave the cell intact or can be degraded to hypoxanthine, xanthine, and ultimately uric acid. Adenosine monophosphate formed by phosphorylation of Adenosine is incorporated into the high-energy phosphate pool. While extracellular Adenosine is primarily cleared by cellular uptake with a half-life of less than 10 seconds in whole blood, excessive amounts may be deaminated by an ecto-form of Adenosine deaminase. As Adenosine requires no hepatic or renal function for its activation or inactivation, hepatic and renal failure would not be expected to alter its effectiveness or tolerability.

CLINICAL TRIAL RESULTS

In controlled studies in the United States, bolus doses of 3, 6, 9, and 12 mg were studied. A cumulative 60% of patients with paroxysmal supraventricular tachycardia had converted to normal sinus rhythm within one minute after an intravenous bolus dose of 6 mg Adenosine (some converted on 3 mg and failures were given 6 mg), and a cumulative 92% converted after a bolus dose of 12 mg. Seven to sixteen percent of patients converted after 1-4 placebo bolus injections. Similar responses were seen in a variety of patient subsets, including those using or not using digoxin, those with Wolff-Parkinson-White Syndrome, males, females, blacks, Caucasians, and Hispanics.

Adenosine is not effective in converting rhythms other than PSVT, such as atrial flutter, atrial fibrillation, or ventricular tachycardia, to normal sinus rhythm.

INDICATIONS AND USAGE FOR ADENOSINE

Intravenous Adenosine injection is indicated for the following.

Conversion to sinus rhythm of PSVT, including that associated with accessory bypass tracts (WPW Syndrome). When clinically advisable, appropriate vagal maneuvers (e.g., Valsalva maneuver), should be attempted prior to Adenosine administration.

It is important to be sure the Adenosine solution actually reaches the systemic circulation. Adenosine does not convert atrial flutter, atrial fibrillation, or ventricular tachycardia to normal sinus rhythm. In the presence of atrial flutter or AF, a transient modest slowing of ventricular response may occur immediately following Adenosine administration.

SIDE EFFECTS

Many individuals experience facial flushing, a temporary rash on the chest, lightheadedness, diaphoresis, or nausea after administration of adenosine due to its vasodilatory effects. Metallic taste is a hallmark side effect of adenosine administration. These symptoms are transitory, usually lasting less than one minute. It is classically associated with a sense of "impending doom", more prosaically described as apprehension. This lasts a few seconds after administration of a bolus dose, during transient asystole induced by intravenous administration. In some cases adenosine can make patients' limbs feel numb for about 2–5 minutes after administration intravenously depending on the dosage (usually above 12 mg). produces transient heart block by slowing conduction through the AV node and thus terminates supraventricular tachycardias that involve the atrioventricular node. Bradyarrhythmias of short duration are common side effects of the use of this drug. Premature atrial and PVCs have also been reported. Adenosine is a frequently used pharmacologic stress agent in myocardial perfusion imaging. Its safety profile is well established, and most of its side effects are mild and transient. Coronary vasospasm occurs occasionally during or after adenosine stress test in rare cases, which may lead to seriously adverse outcomes. (1). Adenosine often induces NS-VT or PVC that are clinically insignificant in the absence of other accompanying heart disease.(2) The adenosine induced PVCs and VT were transient and self-terminating. More than half had a RBBB pattern with a superior axis that suggested an origin in the inferior left ventricular septum. **This drug induced PVCs and VT in 65% of the patients. The high incidence of ventricular arrhythmias following adenosine infusion not require intervention. These arrhythmias appeared to frequently originate from the inferior left ventricular septum, suggesting that this area may be particularly susceptible to the proarrhythmic effects of adenosine.**(3)

1. Han PP, Tian YQ, Wei HX, Coronary spasm after completion of adenosine pharmacologic stress test. *Ann Nucl Med*. 2011 May 15. [in press]
2. Ertan C, Atar I, Gulmez O, et al. Adenosine-induced ventricular arrhythmias in patients with supraventricular tachycardias. *Ann Noninvasive Electrocardiol*. 2008 Oct;13:386-390.
3. Tan HL, Spekhorst HH, Peters RJ, Wilde AA. Adenosine induced ventricular arrhythmias in the emergency room. *Pacing Clin Electrophysiol*. 2001 Apr;24:450-455.

The very short half-life and lack of serious adverse effects generally lead to the consideration that adenosine is a safe drug. Interruption of the supraventricular tachycardia is followed frequently by non-sustained polymorphic ventricular tachycardia.(1)Eventually, polymorphic VT is observed during adenosine stress perfusion imaging in the setting of resting pre-excitation ECG pattern(2) The occurrence of a coronary spasm after an adenosine stress test is an exceptional event after termination of adenosine infusion on abrupt withdrawal of vasodilation. Ischemia induced bursts of polymorphous ventricular tachycardia respond well to antispastic therapy. (3). Chest pain and ST-segment elevation 3 minutes after completion of adenosine pharmacologic stress testing was described.(4) The phenomenon is observed mainly in patients with coronary artery disease.(5).Dipyridamole and adenosine cause frequent side effects as a result of nonspecific adenosine receptor stimulation. Selective agonism of the adenosine A2A receptor should result in a similar degree of coronary vasodilation (and thus similar perfusion images) with fewer side effects. The selective adenosine A2A receptor agonist binodenoson results in an extent and severity of reversible perfusion defects on SPECT imaging similar to nonselective adenosine receptor stimulation, accompanied by a dose-related reduction in the incidence and severity of side effects.(6)

1. Romer M, Candinas R. Adenosine-induced non-sustained polymorphic ventricular tachycardia. *Eur Heart J.* 1994 Feb;**15**:281-282.
2. Mao J, Fang K, Ananthasubramaniam K. Nonsustained polymorphic ventricular tachycardia during adenosine stress perfusion imaging in the setting of resting pre-excitation electrocardiographic pattern: should we be avoiding adenosine pharmacologic stress testing in pre-excitation syndromes? *J Nucl Cardiol.* 2008 May-Jun;**15**:469-472.
3. Rosenberg T, Perdrisot R. Coronary spasm after an adenosine stress test: an adverse effect of a vasodilator. *Acta Cardiol.* 2008 Jun;**63**:401-404.
4. Golzar J, Mustafa SJ, Movahed A. Chest pain and ST-segment elevation 3 minutes after completion of adenosine pharmacologic stress testing. *J Nucl Cardiol.* 2004 Nov-Dec;**11**:744-746.
5. Raza JA, Khan NU, Mustafa JS, et al. ST segment elevation during adenosine pharmacological stress testing in a patient with coronary artery disease. *Am Heart Hosp J.* 2009 Winter;**7**:E122-124.
6. Udelson JE, Heller GV, Wackers FJ, et al. Randomized, controlled dose-ranging study of the selective adenosine A2A receptor agonist binodenoson for pharmacological stress as an adjunct to myocardial perfusion imaging. *Circulation.* 2004 Feb 3;**109**:457-464.

Rarely, after IV adenosine developed **AF and polymorphic VT following administration of IV adenosine is possible**.⁽¹⁾ The PVT cause hemodynamic instability with no response to cardioversion, but termination with procainamide. The heart is vulnerable to hemodynamically unstable, possibly lethal, PVT early under some circumstances. This vulnerability may be exposed following administration of adenosine. Extra caution is warranted when using adenosine mainly in the post-MI period.

Compounds that increase gap junctional communication, thereby increasing the conduction velocity and decreasing the risk of arrhythmias. Some of these compounds also inhibit connexin 43 (Cx43) hemichannels, thereby limiting adenosine triphosphate loss and volume overload following ischaemia/reperfusion, thus potentially increasing the survival of cardiomyocytes.

The family of peptides called antiarrhythmic peptides (AAP's) gap junction openers are:

- 1) Antiarrhythmic peptide (AAP), AAP10, ZP123;
- 2) GAP-134;
- 3) RXP-E;
- 4) The Cx mimetic peptides Gap 26 and Gap 27.

None of these compounds have effects on Na(+) , Ca(2+) and K(+) channels, and therefore have no proarrhythmic activity associated with currently available antiarrhythmic drugs. GAP-134, RXP-E, Gap 26 and Gap 27 are pharmacological agents with a favorable clinical safety profile, as already confirmed in phase I clinical trials for GAP-134. These agents show an excellent promise for treatment of arrhythmias in patients with ischaemic cardiomyopathy⁽²⁾.

1. Kaplan IV, Kaplan AV, Fisher JD. Adenosine induced atrial fibrillation precipitating polymorphic ventricular tachycardia. Pacing Clin Electrophysiol. 2000 Jan;23:140-141.
2. De Vuyst E, Boengler K, Antoons G, et al. Pharmacological modulation of connexin-formed channels in cardiac pathophysiology. Br J Pharmacol. 2011 Jun;163:469-83.