SUSTAINED WIDE QRS COMPLEX TACHYCARDIA (WCT)
TAQUICARDIA COMPLEXA SUSTENTADA DE QRS LARGO

Case Dr Raimundo Barbosa Barros Fortaleza Ceará - Brazil

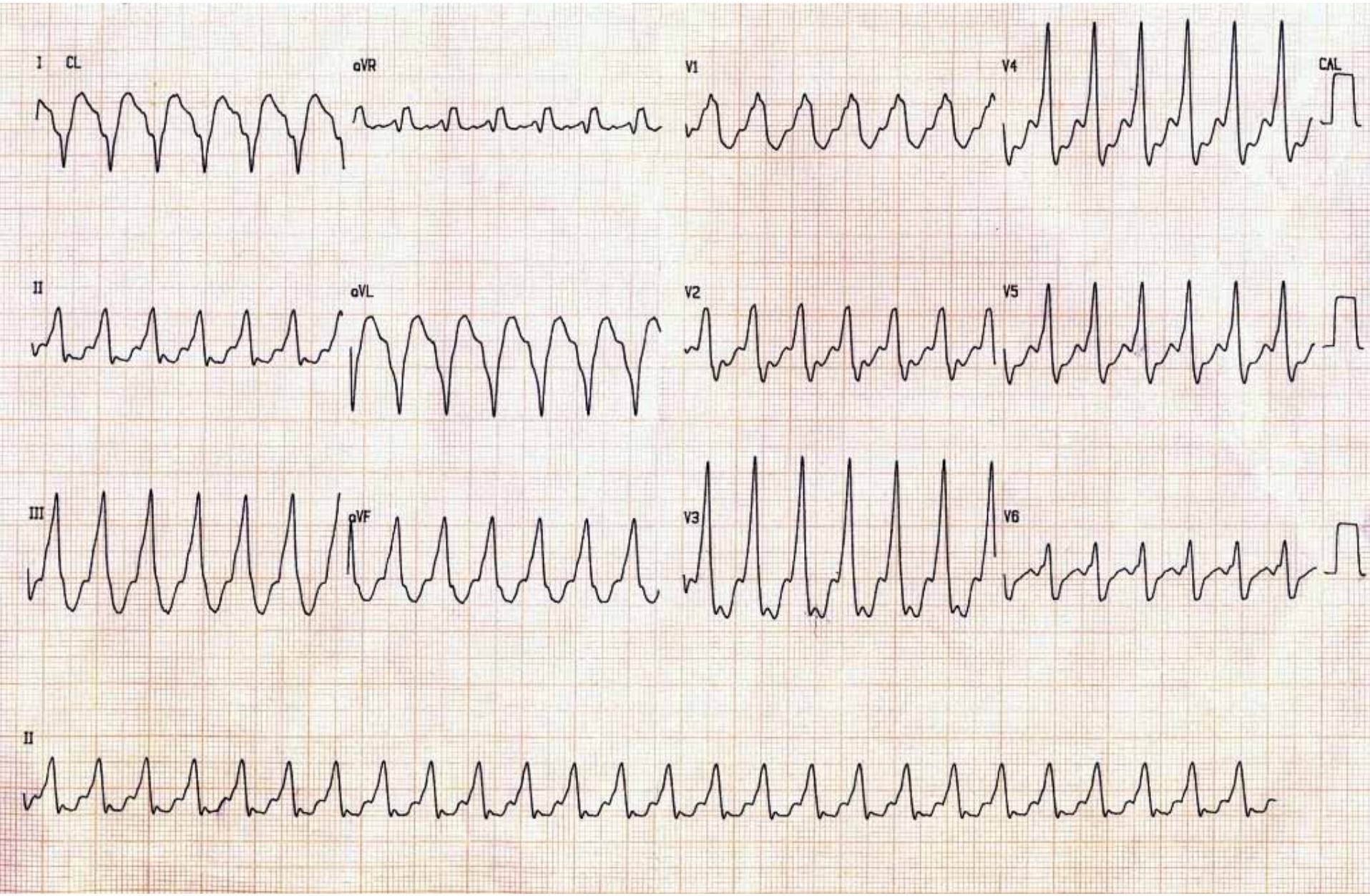
Prezado professor Andrés, estou enviando os traçados com melhor resolução. Homem, 40anos, atendido no HM com história de dispnéia e dor precordial tipo "pressão" há 48 horas. Realizou ECG que revelou taquicardia sustentada complexa com QRS largo. Como estava hipotenso (PA= 90/70 mmHg) foi realizado cardioversão elétrica com 200J a qual estabilizou o quadro (PA=140/80). Nesta ocasião o colega fez o diagnóstico de WPW. Foi prescrito amiodarona e solicitado ECO que revelou hipocinesia ínfero-lateral, FE=57%. Sem fatores de risco para DAC. Não há relato de palpitações prévias. Não foram solicitados biomarcadores. No dia 30/01/11, acredito que algum colega interpretou a onda delta negativa em D1 e AVL (via acessória lateral esquerda) como onda Q (pseudo Q) e solicitou coronariografia a qual não mostrou lesões coronárias.

Dear Prof. Andrés, I am sending these sequential traces with better resolution.
Man, 40 yo., history of dyspnea and prolonged chest pain (48hrs) with pressure character. We preformed the first ECG that revealed Sustained Wide Complex Tachycardia. He had hypotension (BP= 90/70 mmHg) consequently, the option was synchronized electrical cardioversion (200J). Immediately his blood pressure returned to normal values (BP = 140/80). The fellow makes diagnosis of tachyarritmia secondary to WPW. He prescribed amiodarone and requested ECHO that revealed inferolateral hypokinesis and LVEF=57%. The patient has not CAD risk factor. Also he has not previous palpitations episodes. We don't request biomarkers. I believe that some colleague interpreted the initial broad negative delta wave in high leads I and aVL (left lateral accessory pathway) as necrosis Q-waves and requested a coronary angiography which resulted normal.

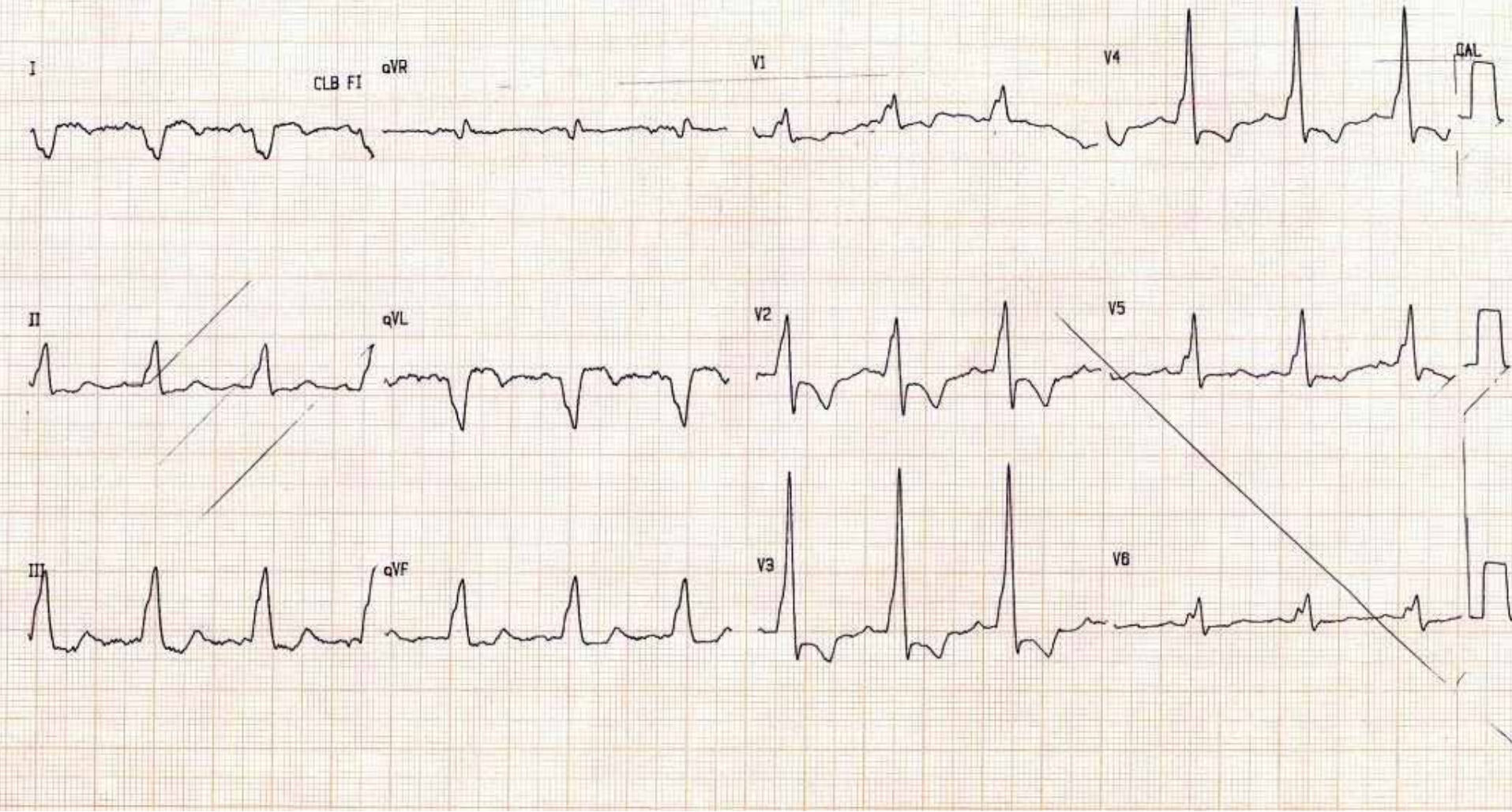
Questions:

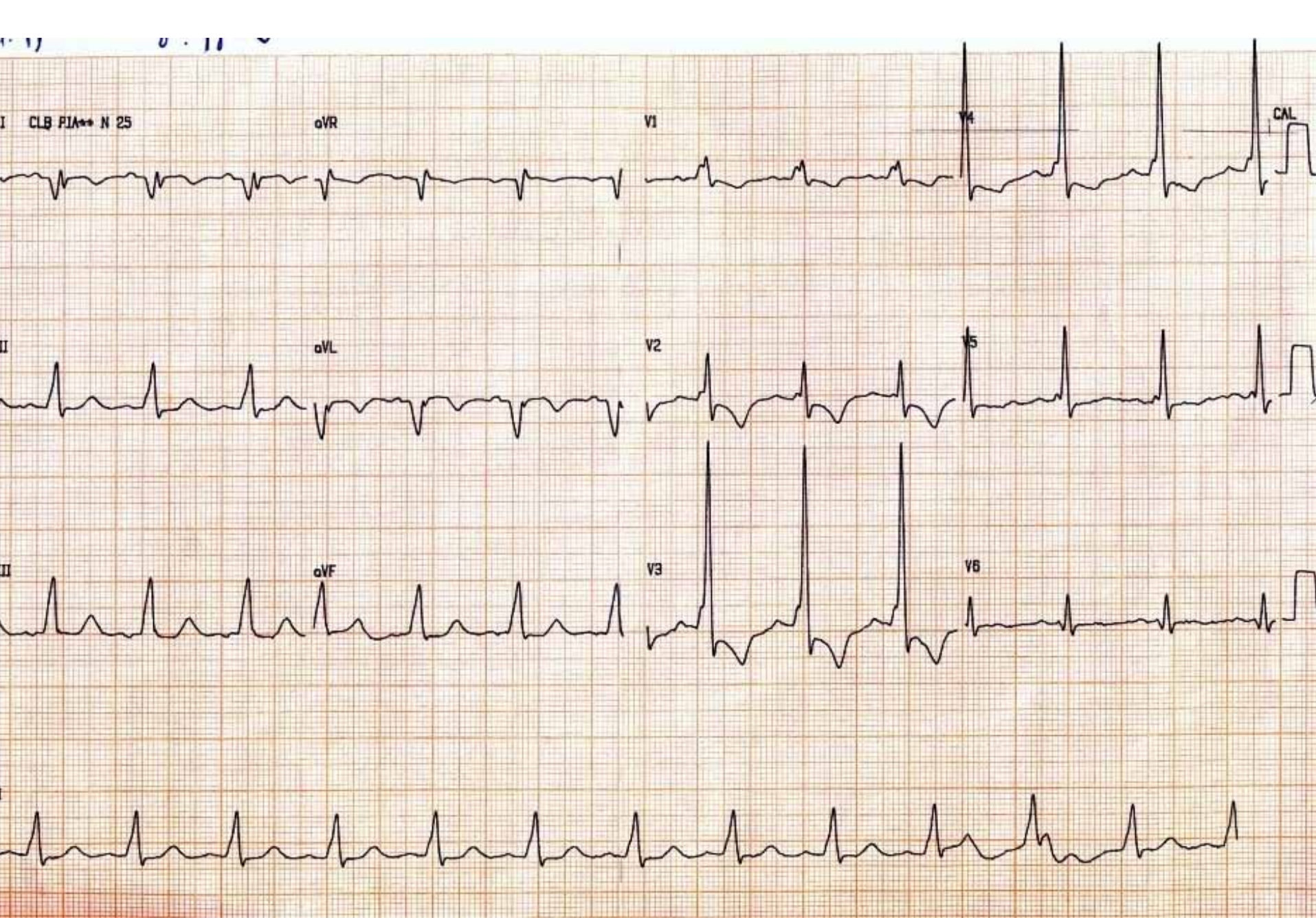
Which is the event diagnosis? And why? Qual o diagnóstico durante o evento? E porque?
Which is the basal ECG diagnosis? Qual o diagnóstico do ECG de base?
Witch is the appropriate approach during the event and out of it? Qual a abordagem mas adequada durante o evento e fora dele?
We are waiting your valuable opinions. Estamos aguardando suas valiosas opiniões.

First ECG



12 lead ECG during the event


I
CLB FI
aVR
V1
V4
CAL
II
aVL
V2
V5
III
aVF
V3
V6



Basal 12-lead ECG

Pt with left lateral AP baseline with preexcited tachycardia from same pathway. Ablation of AP is indicated.

Paciente com feixe anômalo lateral esquerdo de base e taquicardia pré-excitada com o mesmo feixe anômalo. Ablação está indicada

Melvin M Scheinman, MD PhD
Department of Cardiac Electrophysiology, University of California San Francisco, San Francisco, California, USA. **scheinman@medicine.ucsf.edu**
Professor of Medicine

Address:
UCSF
Electrophysiology Service
500 Parnassus Avenue
San Francisco, CA 94143-1354

Antidromic tachycardia
Left lateral accessory pathway
RF ablation

Baranchuk,Adrian MD FACC
Assistant Professor of Medicine
Cardiac Electrophysiology and Pacing
Director, EP Training Program
Kingston General Hospital
FAPC 3, 76 Stuart Street
K7L 2V7, Kingston ON
Queen's University
Ph: 613 549 6666 ext 3801
Fax: 613 548 1387
barancha@kgh.kari.net

Taquicardia antidrómica
Feixe anômalo lateral esquerdo
Energia de radiofrequencia

Andrés y Raimundo queridos, nuevamente en el ruedo.
Taquicardia antidrómica. El estímulo atrioventricular conduce en forma anterógrada por una via anómala lateral izquierda.
Alguien duda que habra que ablacionarla? Yo no.
Ademas de la VA, tiene algo mas este paciente?

Saludos,

Dr. Francisco Femenia Unidad de Arritmias, Departamento de Cardiología, Hospital Español de Mendoza.Mendoza- Argentina

---

Dear Andrés and Raimundo: again in together

Antidromic tachycardia. Anterograde conduction by left lateral accessory pathway.

Someone doubt that appropriate approach is radiofrequency catheter ablation?

Has this patient another think?

Greeting
Francisco Femenia MD. Mendoza- Argentina

Very nice tracing.
Antegradely conducting left accessory pathway (left lateral) is highly suspected in sinus rhythm.
The tachycardia may be either antidromic tachycardia involving the pathway antegradely or another accessory pathway retrogradely or atrial tachycardia with 1"1 conduction; atrial flutter looking less likely due to the heart rate (170/min)

Traçado muito bonito
Anterogradamente conduzindo por uma via acessória lateral esquerda é altamente suspeito em ritmo sinusal. A taquicardia pode ser antidrômica envolvendo o feixe anômalo anterogradamente ou outro feixe retrogradamente ou taquicardia atrial 1:1. Flutter atrial parece menos possível pela FC de 170bpm

Prof. Bernard Belhassen,
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

FINAL CONCLUSIONS
COCLUSÕES FINAIS

Andrés Ricardo Pérez-Riera MD

First ECG:

Sustained Wide or broad QRS Complex Tachycardia (WCT), HR: 166bpm, preexcited antidromic reciprocating macro-reentry tachycardia or antidromic Circus Movement Tachycardia with anterograde conduction over a left lateral Accessory Pathway (AP). Region I of Linsay, left lateral: negative delta wave in I or aVL, not shifted SÂQRS and precordial transition shifted to the right or early. Points 8 or 9 of Gallanger or Type C WPW

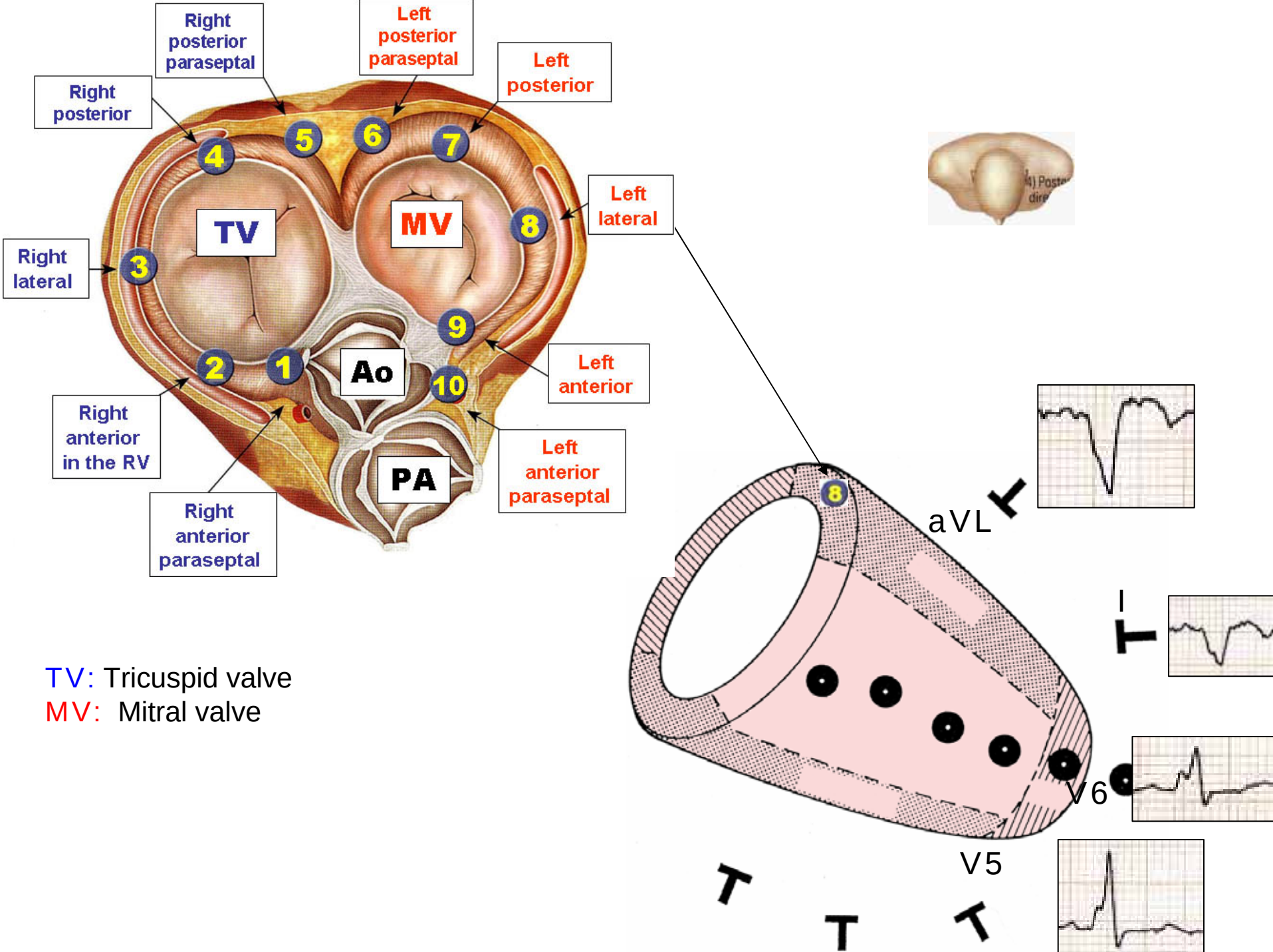
The presence of an antidromic tachycardia should prompt a careful search for a second bypass tract. Patients with WPW syndrome are potentially at an increased risk of dangerous ventricular arrhythmias due to extremely fast conduction across the bypass tract if they develop atrial flutter or atrial fibrillation. These wide tachycardias are difficult to differentiate from VTs and often have a slurred R wave upstroke with QRS duration >160 ms.

Overall, sudden death occurs rarely, with an estimated frequency rate of 0.1%.

Other factors that appear to influence risk are the presence of multiple bypass tracts, short accessory pathway refractory periods (<240 ms), atrial fibrillation and atrial flutter, or a family history of premature SD. SCD is unusual without preceding symptoms.

Morbidity may be related to rapid near syncopal or syncopal arrhythmias. Even when syncope is absent, the arrhythmia episodes may be highly symptomatic. The potential for syncope, hemodynamically compromising rhythms, or sudden death may prevent patients with WPW from participating in competitive sports or hazardous occupations until the substrate is definitively addressed and cured by a catheter ablation procedure.

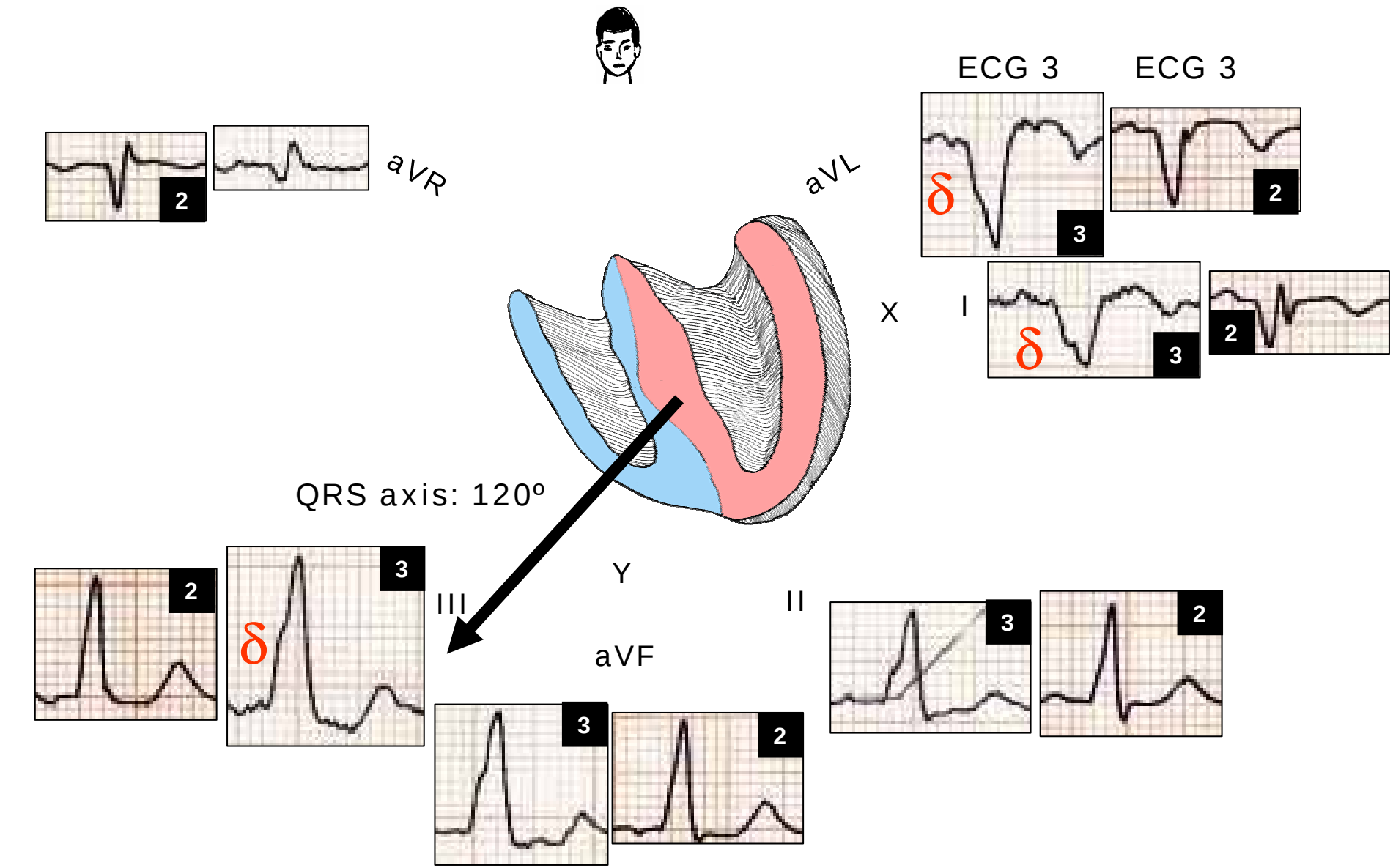
1. **Marcus GM, Lee BK, Scheinman MM.. Initiation of antidromic reciprocating tachycardia: what is the mechanism?Heart Rhythm. 2006 Jul;3:865**



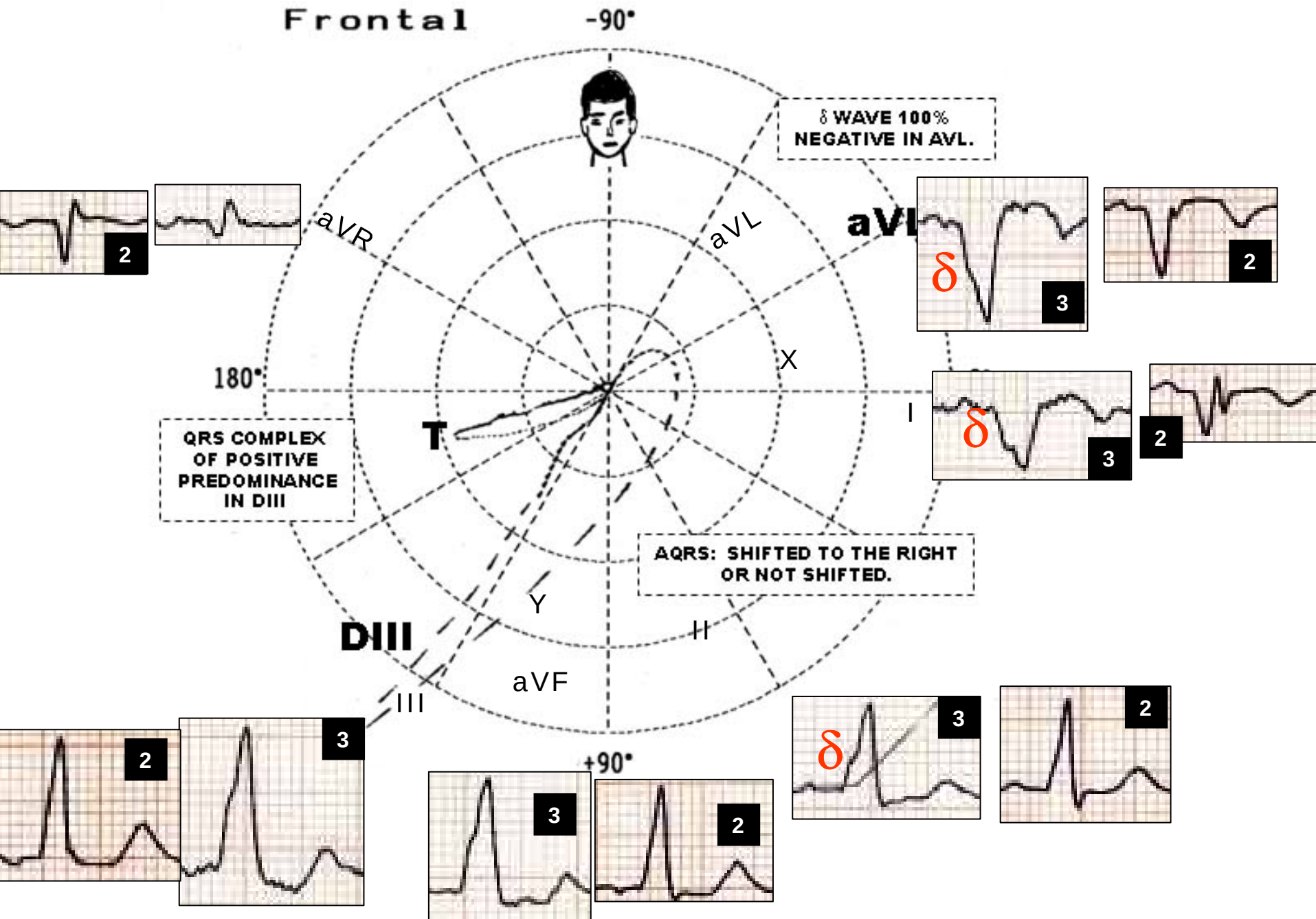
TV: Tricuspid valve

MV: Mitral valve

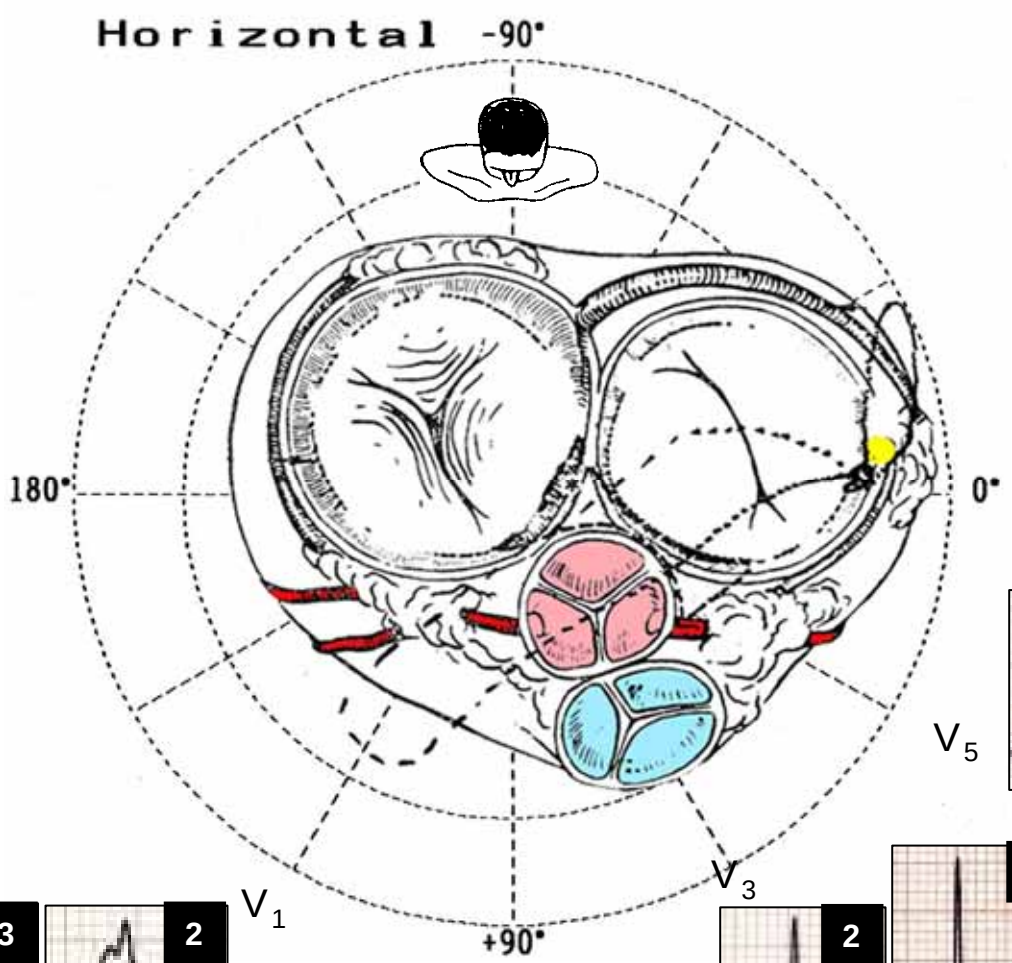
ECG 2 AND ECG 3 ON FRONTAL PLANE

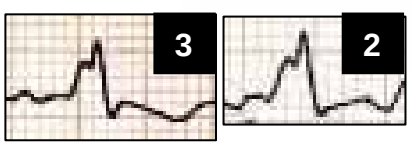


HYPOTHETICA ECG/VCG CORRELATION ON FRONTAL PLANE

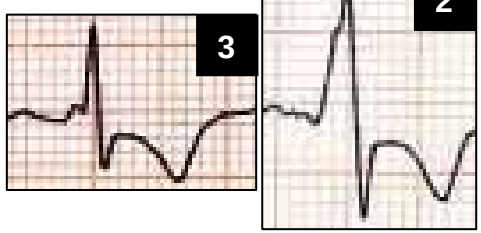


HYPOTHETICAL ECG VCG CORRELATION ON HORIZONTAL PLANE

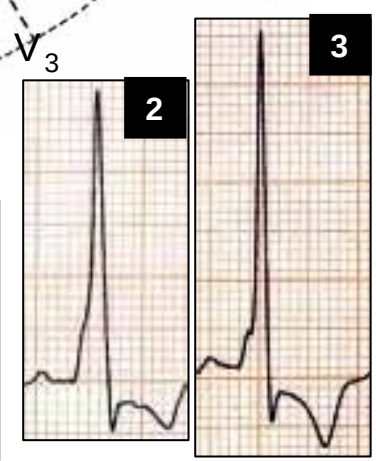




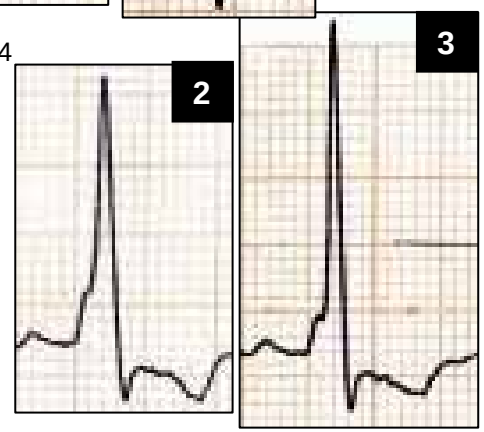
V_1



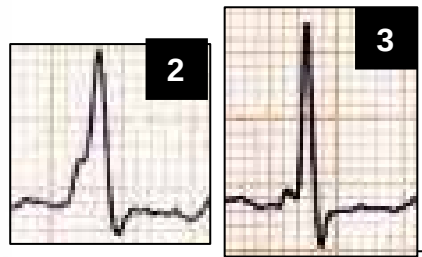
V_2



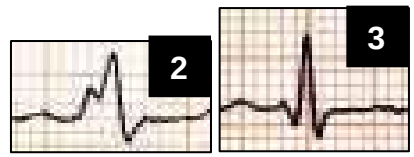
V_3



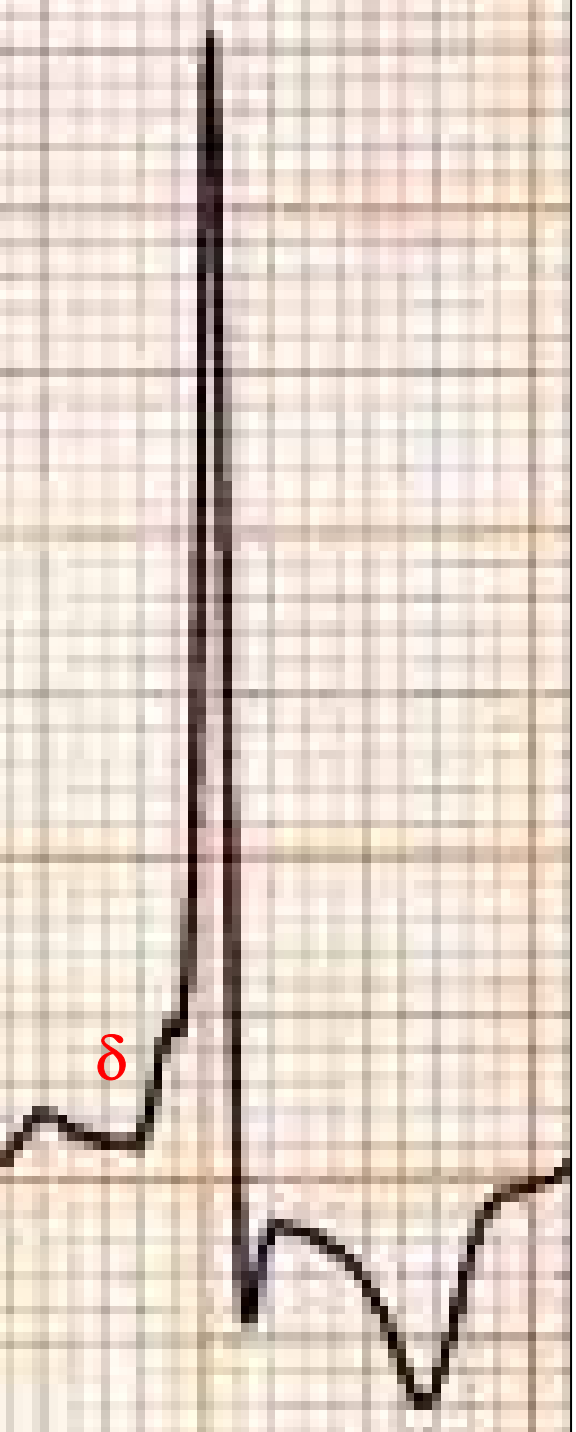
V_4



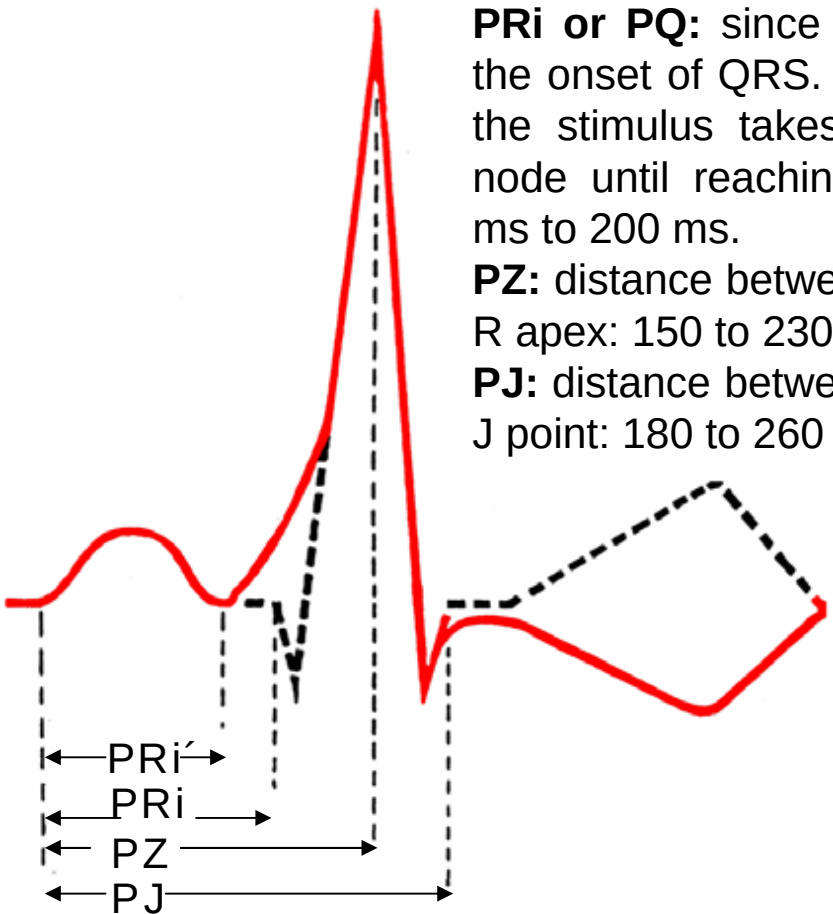
V_5



V_6



1. Short PRi interval: <120 ms
2. Wide QRS complex: 160 ms
3. Notch at the onset of QRS complex: delta (δ) wave, duration 30 ms to 60 ms and voltage of up to 4 mm, which corresponds to early depolarization by ventricular mass;
4. Unaltered P-J interval (normal): 180 to 260 ms;
5. Unaltered P-Z interval (normal): 230 ms (150 to 230 ms);
6. Secondary alterations of ventricular repolarization (ST-T): depending on aberrant depolarization;

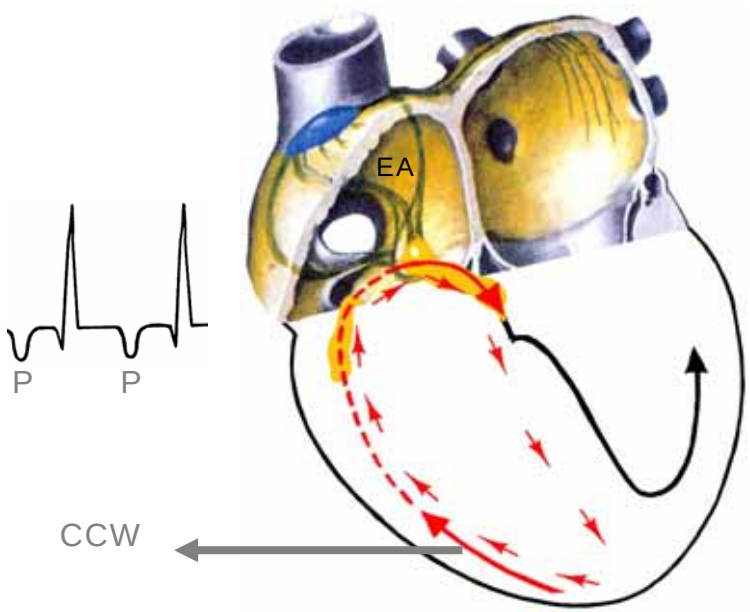


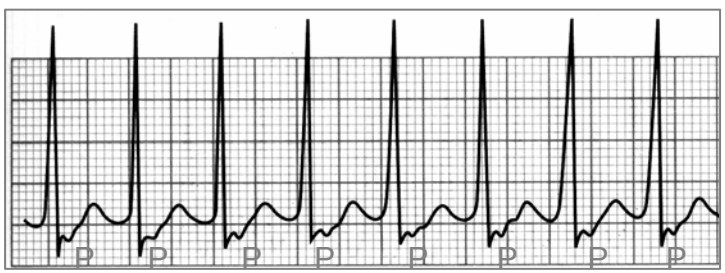
PRi or PQ: since the onset of P up to the onset of QRS. It represents the time the stimulus takes to go from the SA node until reaching the ventricles: 120 ms to 200 ms.

PZ: distance between P wave onset until R apex: 150 to 230 ms.

PJ: distance between P wave onset until J point: 180 to 260 ms.

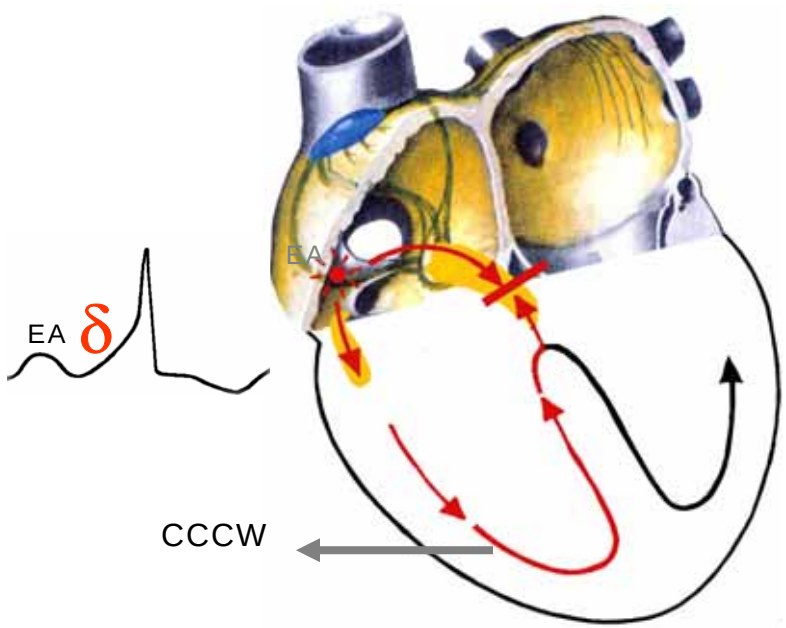
Orthodromic or with narrow QRS: 90%

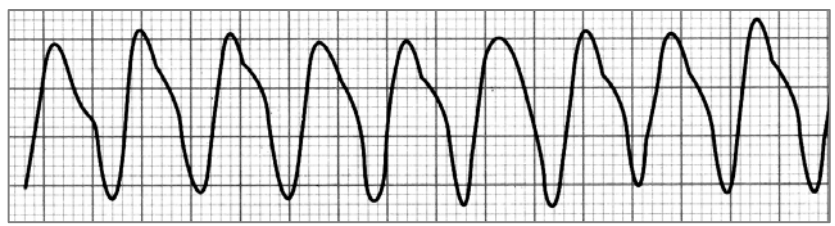




Narrow QRS.
Clockwise macro-reentry motion.
It uses a normal pathway in anterograde fashion and/or AP in retrograde fashion.

Antidromic or with wide QRS: 10%





Wide QRS.
Counterclockwise macro-reentry motion.
It uses a normal pathway in retrograde fashion and/or AP in anterograde fashion.

Background

In 1930, Wolff, Parkinson, and White originally described bundle-branch block with a short PR interval in patients prone to paroxysmal tachycardia[1]. Pre-excitation was defined by Durrer et al in 1970 when they wrote, "Pre-excitation exists, if in relation to atrial events, the whole or some part of the ventricular muscle is activated earlier by the impulse originating from the atrium than would be expected if the impulse reached the ventricles by way of the normal specific conduction system only."

Wolff-Parkinson-White (WPW) syndrome is a congenital abnormality involving the presence of abnormal conductive tissue between the atria and the ventricles in association with supraventricular tachycardia (SVT). It involves activation of the ventricles that occurs earlier than anticipated (pre-excitation), which occurs because of conduction of an atrial impulse not by means of the normal conduction system, but via an extra atrioventricular (AV) muscular connection, termed an accessory pathway(AP), that bypasses the AV node[2]. Patients with WPW syndrome are potentially at an increased risk of dangerous ventricular arrhythmias due to extremely fast conduction across the bypass tract if they develop atrial flutter or atrial fibrillation. While they have an accessory AV connection, it lacks antegrade conduction, and accordingly they do not have the classical abnormalities of the surface ECG. Only a small percentage of patients with WPW syndrome are at risk for SCD (<1%). In patients who present with pre-excited AF, cardiac EPS and RFCA may be curative. Other presentations include symptomatic SVT, which can also be cured by catheter ablation. Asymptomatic patients need periodic observation. The onset of cardiac arrhythmias, and possibly the SCD, may be eliminated by prophylactic catheter ablation as well[3].

1. **Wolff, L., Parkinson, J., White, PD. Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. *American Heart Journal*. 1930/08;5:685-704.**
2. **Calkins H, Sousa J, el-Atassi R, et al. Diagnosis and cure of the Wolff-Parkinson-White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. *N Engl J Med*. Jun 6 1991;324:1612-1618.**
3. **Pappone C, Santinelli V, Manguso F, Augello G, Santinelli O, Vicedomini G. A randomized study of prophylactic catheter ablation in asymptomatic patients with the Wolff-Parkinson-White syndrome. *N Engl J Med*. Nov 6 2003;349:1803-1811**

ANTIDROMIC CIRCUS MOVEMENT TACHYCARDIA

Less commonly, a shorter refractory period in the accessory tract may cause block of an ectopic atrial impulse in the normal pathway, with antegrade conduction down the accessory tract and then retrograde reentry of the normal AV nodal pathway. This type of tachycardia is called antidromic Circus Movement Tachycardia. (Antidromic CMT)

On ECG findings, the QRS is wide, which is an exaggeration of the delta wave during sinus rhythm (ie, wide-QRS tachycardia). Such tachycardias are difficult to differentiate from VTs and often have a slurred R wave upstroke with QRS duration >160 ms.

The mechanism underlying most (95%) of the tachycardias in patients with WPW syndrome is macro-reentry caused by antegrade conduction over the AV node His bundle pathway and retrograde conduction over an accessory pathway (orthodromic).

Antidromic CMT is less common (5%) in patients with WPW syndrome. Even when the AP conducts only in retrograde fashion, it can still participate in the reentrant circuit and produce an orthodromic AV reciprocating tachycardia with a narrow QRS morphology. The presence of an antidromic tachycardia should prompt a careful search for a second bypass tract. Multiple APs are observed in 33% of cases of antidromic CMT[1].

SVT due to re-entry in WPW is typically orthodromic CMT in 95% and antidromic CMT in 5%.

Light-headedness and near syncope appear to occur more commonly in persons with WPW syndrome who have paroxysmal SVT (PSVT) or atrial fibrillation than in those with AV nodal reentry.

Syncope can occur because of inadequate cerebral circulation due to a rapid ventricular rate or because the tachyarrhythmia is depressing the sinus pacemaker, causing a period of asystole at the point of tachycardia termination.

1. **Atié J, Brugada P, Brugada J, Smeets JL, Cruz FS, et al. Clinical and electrophysiologic characteristics of patients with antidromic circus movement tachycardia in the Wolff-Parkinson-White syndrome. Am J Cardiol. 1990 Nov 1;66:1082-1091.**

LOCATION OF ACCESSORY PATHWAYS (APs)

LOCATION	Relative percentage
Left free wall	50%
Posterior septum	30%
Right free wall associated congenital heart disease	13%
Anteroseptal	7%

CLASIFICATION OF APs

Manifested	**65%**
Concealed or latent without delta wave during sinus rhythm	**35%**

High –risk patients are identified from the duration of the refractory period of the APs in the anterograde direction. This duration varies considerably among patients and is influenced by sympathetic tone. Most atrioventricular APs) exhibit Kent bundle physiology characterized by fast and non-decremental conduction properties. In contrast, atriofascicular APs, which are only capable of reaching slow levels of long antegrade decremental conduction, are uncommon. APs with unusual decremental properties that are either latent, demonstrable only during CMT or overt, exhibiting functional longitudinal dissociation. These APs could be identified and successfully ablated after detailed electrophysiological analysis[1].

1. **Hluchy J, Schickel S, Schlegelmilch P, Jörger U, Brägelmann F, Sabin GV. Decremental conduction properties in overt and concealed atrioventricular accessory pathways. Europace. 2000 Jan;2(1):42-53.**

Tachycardia may also involve multiple APs which may provide both antegrade and retrograde conduction and may alternate antegradely or retrogradely. Tachycardia may occur in which the AP simply acts as a bystander, and does not participate in the tachycardia mechanism. When AF is conducted to the ventricles via and AP, the resultant ventricular rate may be extremely rapid, placing the patient at risk of developing VF and cardiac arrest.

The normal AV annulus is composed exclusively of electrically inert fibrous tissue. The AV node and His bundle normally act as the sole route of electrical conduction. APs occur at all points along the AV ring, and usually occur as isolated abnormalities, although a proportion of patients have associated congenital heart disease. This is particularly true of right-sided APs. Most APs exhibit non-decremental conduction properties, and conduct faster than normal AV conduction tissue.

In many patients with APs the surface ECG reveals clear evidence of pre-excitation, and a good idea of AP localization is possible using one or more of several algorithms which have been developed. Patients with latent pre-excitation, intermittent pre-excitation, and patients with concealed APs have not evidence of pre-excitation on a proportion or all of their surface ECGs. Patients present with a history of paroxysmal palpitations, often with associated symptoms such as chest discomfort. Syncope is a rare presenting symptom. Unless BBB is present, patients with orthodromic CMT exhibit a narrow complex tachycardia on the surface ECG. Patients with pre-excited tachycardia including antidromic CMT, and other forms of SVT in which the AP conducts to the ventricles as a bystander but does not participate in the tachycardias mechanism, present as WCT on the surface ECG which may be difficult to distinguish from VTs. Adenosine is increasingly used for this purpose since it is highly efficacious and has an extremely short half-life. Adenosine is also very useful in the diagnosis of WCT, and in unmasking latent pre-excitation during sinus rhythm. EPS in these patients is frequently performed at the same time as an attempt at RFCA; it aims to diagnose, localize and determine the functional characteristics of an AP, and to characterize the role of the AP in tachycardia. CMT can be reliably terminated by effective AV nodal blockade. Drug therapy for the prevention of CMT is useful for temporary control whilst awaiting more definitive measures and in certain cases as long-term management.

No class of drug stands out as 'therapy of choice', and physician preference, pro-arrhythmic effects and associated conditions need to be taken into account such that an individual choice can be made in each patient. The management of patients with CMT has been revolutionized in recent years with the advent of catheter-based techniques for their cure. Whilst this method of treatment is highly effective and has low complication rates, APs in particular locations such as the septal region remain challenging.

The typical and most common tachycardia in patients with atriofascicular pathways is a macro reentrant tachycardia, with anterograde conduction over the decrementally conducting AP tract and retrograde conduction over the right bundle branch-His-AV node axis resulting in a short V-right bundle branch and short V-H interval. Rate changes in antidromic tachycardia in patients with atriofascicular fibers can be based on a shift in VA conduction from one bundle branch to the other. This may be accompanied by changes in the frontal plane QRS axis because of a change in ventricular activation sequence[1].

1. **Sternick EB, Rodriguez LM, Timmermans C, Sosa E, Cruz FE, Gerken LM, et al. Effects of right bundle branch block on the antidromic circus movement tachycardia in patients with presumed atriofascicular pathways. J Cardiovasc Electrophysiol. 2006 Mar;17:256-260.**