Case Report/ Apresentação de Caso

Wide QRS Complex Tachycardia (WCT)
or
Broad QRS Complex Tachycardia (BCT)

Taquicardia com QRS largo

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

Interrogatory

JVC, male, 57-year-old patient. Mulatto, construction worker, protestant. He was born in Virgolândia, Minas Gerais, and currently lives in Santo André, São Paulo, Brazil.

Personal background: he denies suffering high blood pressure, diabetes, dyslipidemia, coronary heart disease, or Chagas disease. Former smoker (for 30 years he smoked a package a day). He stopped smoking ten years ago. He was an old-time alcohol drinker; he stopped drinking five years ago.

Family history: 2 healthy sons. His father died at the age of 72, due to heart problems (he cannot specify what). His mother died at 62 years of age, due to stomach cancer.

Main complaint: palpitations and chest pain.

History of current discomfort: he mentions that on March 5, 2010, while walking, he suddenly felt pre-syncope symptoms, rapid palpitations associated to precordial pain in anguish; dyspnea, vomiting, cold sweating, and headache. Precordial pain was described as very intense, oppressive, which lasted longer than 1 hour, irradiating toward the left shoulder and the back. He sought medical help, and after an ECG was conducted (next slide), an event was detected, being interpreted as VT. He was referred to chemical cardioversion (due to the lack of other resources) with endovenous amiodarone, which caused a quick reversion of the event.

He says that 3 weeks ago he suffered a similar, less intense event. On that occasion, an ECG, and later catheterization were performed. They did not reveal significant alterations.

He is not displaying symptoms presently.

Physical examination

General good physical condition. Conscious and properly oriented, with good colors, hydrated, no fever, acyanotic, anicteric, with no jugular vein distension.

BP: 110x70 mmHg. HR: 88 bpm. Peripheral pulses present.

The rest is not worthy of note.

Interrogatório

JVC, masculino, 57 anos, pardo, pedreiro, evangélico, natural de Virgolândia – Minas Gerais, e residente em Santo André – São Paulo – Brasil.

Antecedentes pessoais: nega hipertensão, diabetes, dislipdemia, coronariopatia ou doença de chagas.

Ex-tabagista (fumou por 30 anos, 1 maço/dia). Há dez anos livre do vício.

Ex-etilista inveterado, há cinco anos abstêmio.

Antecedentes familiares: 2 filhos sadios, pai falecido aos 72 anos por problemas cardíacos (não sabe especificar), mãe falecida aos 62 anos de câncer gástrico

Queixa Principal: “batedeira e dor no peito”

História da Moléstia Atual: refere que no dia 05/03/2010, durante caminhada, fora acometido de súbito de quadro de pre-síncope, palpitações rápidas associada a dor precordial em aperto, dispnéia, vômito, sudorese fria e cefaléia. A dor precordial a referia como de forte intensidade, opressiva, de duração maior que 1h, irradiada para ombro esquerdo e dorso. Procurou auxilio médico, onde após a realização de ECG (Próximo slide) detectou-se evento interpretado como TV, sendo submetido a cardioversão química (por falta de recurso) com amiodarona endovenosa a qual ocasionou rápida reversão do evento.

Refere evento semelhante, de menor intensidade, ocorrido há 3 semanas. Na ocasião, fora realizado ECG e posteriormente cateterismo, o qual não revelou alterações significativas.

No momento assintomático.

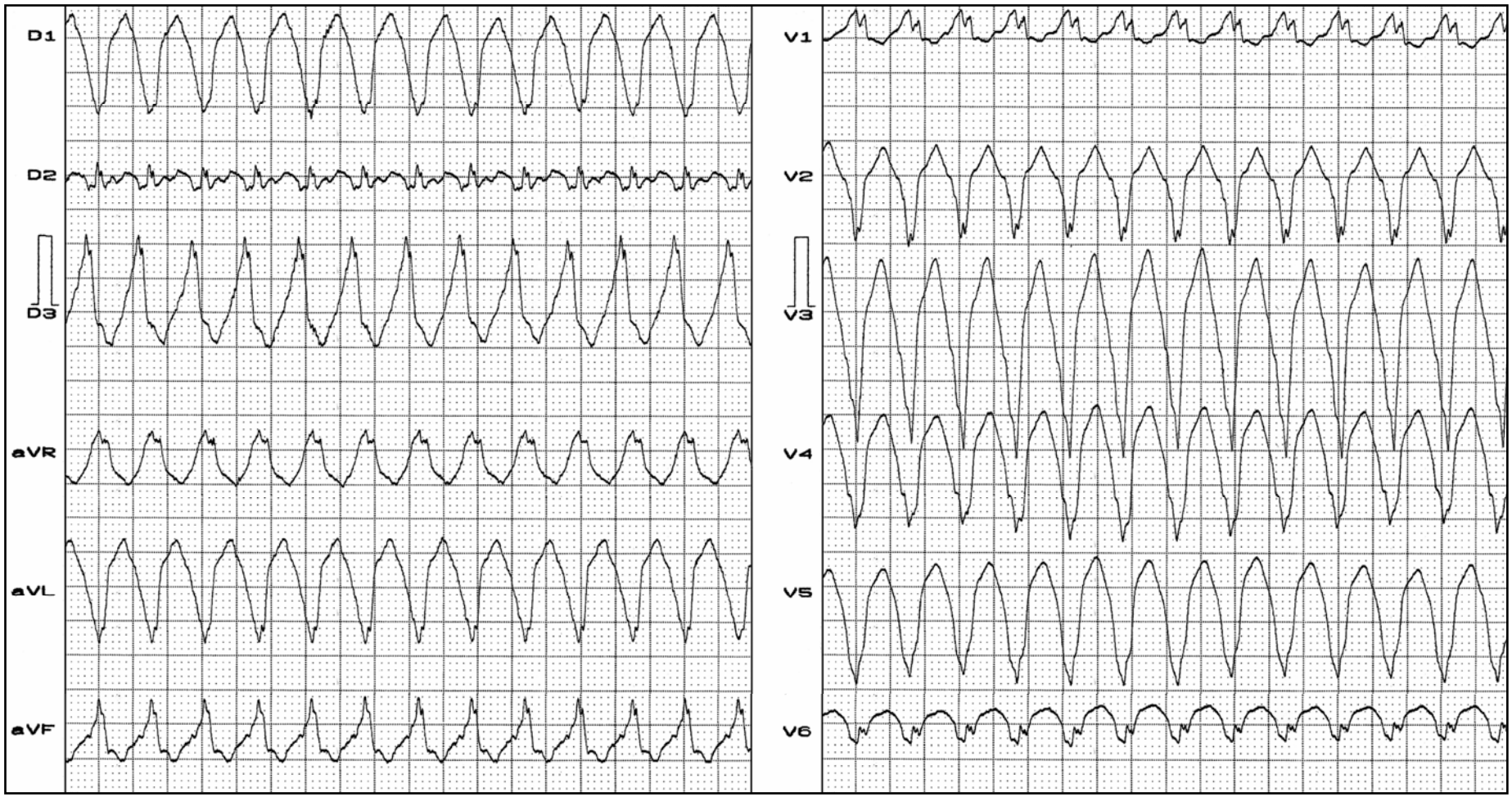
Exame Físico

Bom estado geral, consciente e orientado, corado, hidratado, afebril, acianótico, anictérico, sem turgência jugular.

PA: 110 X 70mmhg FC: 88bpm. Pulsos periféricos presentes.

Resto nada digno de nota.

Date: 05/03/2010



Basic definitions/definições básicas

1. **Wide QRS Complex Tachycardia** (**WCT):** a rhythm with a rate of ≥ 100lpm and QRS duration ≥120ms.
   Taquicardia de QRS largo: um ritmo com frequência ≥ 100lpm e duração do QRS ≥ 120ms.

2. **Ventricular Tachycardia (VT):** a WCT originating below the level o the His bundle.
   Taquicardia ventricular(TV): uma WCT originada abaixo do feixe de His.

3. **Supraventricular thacycardia (SVT):** a tachycardia dependent on participation of structures at or above the level of the His bundle.
   Taquicardia supraventricular(TSV): uma taquicardia dependente ou com participação de estruturas localizadas acima do feixe de His.

4. **Left Bundle Branch Block (LBBB) pattern/morphology or LBBB-like pattern:** QRS duration≥120ms with predominantly negative terminal deflection in ECG lead V_1.
   Padrão ou morfologia de Bloqueio de Ramo Esquerdo: duração do qRS ≥120ms com deflexão terminal do QRS em V_1 negativa.

5. **Right Bundle Branch Block (RBBB) pattern/morphology or RBBB-like pattern:** QRS duration≥120ms with predominantly positive terminal deflection in ECG lead V_1.
   Padrão ou morfologia de Bloqueio de Ramo Direito: duração do qRS ≥120ms com deflexão terminal do QRS em V_1 positiva.

Possible causes of WCT/

1. **Ventricular Tachycardia VT: >80% of cases**
2. **SVT with aberration (SVT-A) right or left aberration conduction**
3. **SVT with preexcitation (WPW-S): accessory pathway conduction. Preexcited SVT: 1% a 5% cases**

- **- Atrial fibrillation with accessory Pathway**
- **- Atrial fibrillation with multiple accessory Pathways**
- **- Atrial flutter with an Accessory pathway:**
- **- Antidromic Circus Movement Tachycardia (CMT): multiple accessory Pathways**
- **in 50% of cases.**
- **- Broad QRS Paroxismal Supraventricular Tachycardia Using Nodoventricular fibers (Mahaim fibers[1])**

4. **SVT with baseline preexisting BBB. Due to abnormal muscle-to- muscle spread of impulse**
5. **SVT with hyperkalemia other electrolytes disturbances or drugs such as IA and IC antiarhtythmic**
6. **Ventricular pacing rhythm: presence of a pacing device on physical examination is a strong clue to pacing as the cause of the WCT. When the pace is in the apex the QRS pattern is LBBB with superior axis.**

---

1. Gallangher JJ, Smith WM, Kasel JH, et al. Role of Mahaim fibers in cardiac arrhtymias in man Circulation 1981, 64: 176.

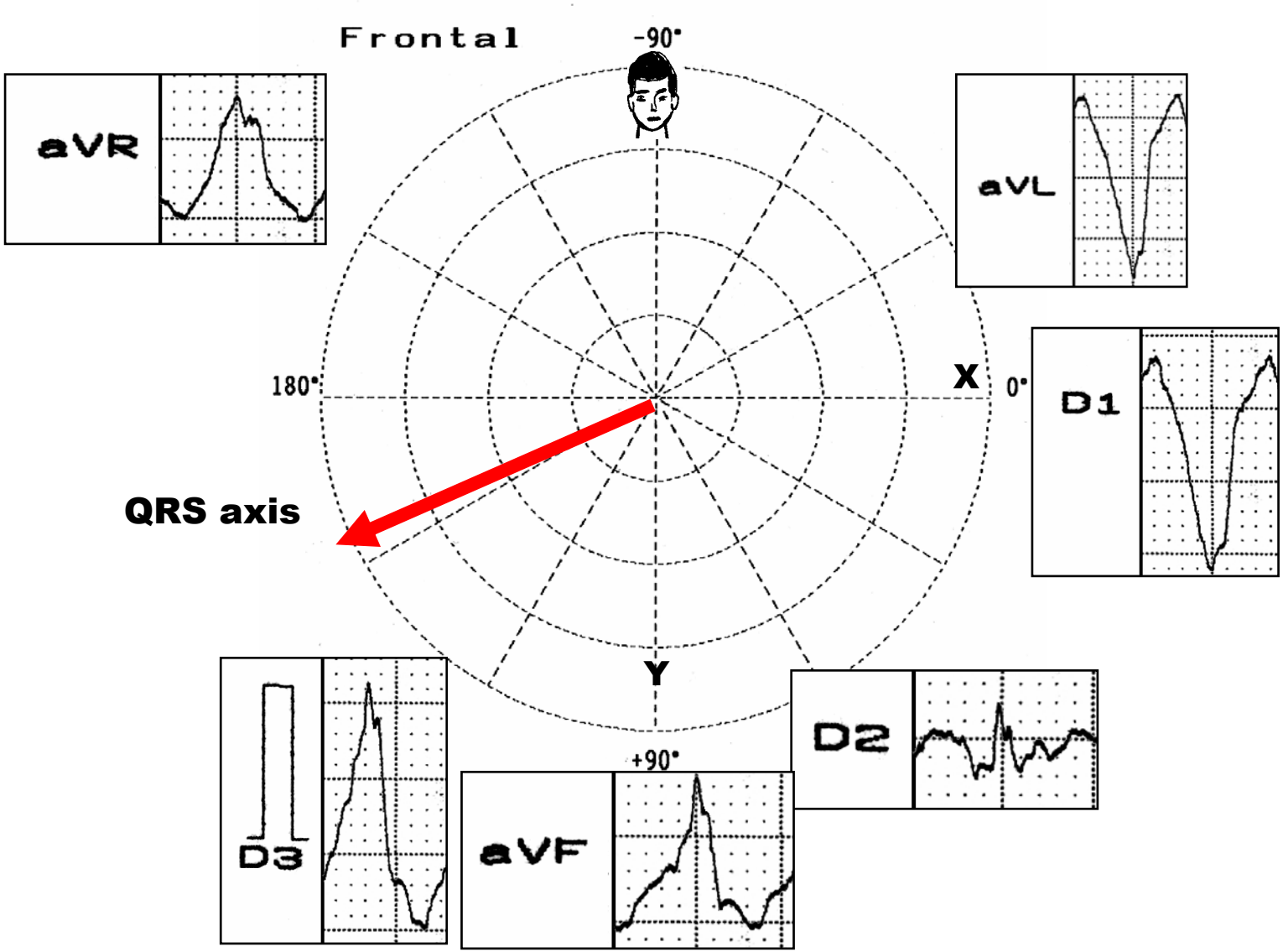
Possíveis Causas de Taquicardia de QRS largo

1. TV: > de 80% dos casos.
2. TSV com condução aberrante(TSV-A). Aberração direita o esquerda
3. TSV com pre-excitation (S-WPW). TSV pre-excitada. 1% a 5% dos casos
   - Fibrilação atrial com via accessorias.
   - Fibrilação atrial com múltiplas vias accessorias.
   - Fluter atrial com via acessória
   - Taquicardia antidrômica com movimento em círculo: possui múliples vias acessórias em 50% dos casos,
   - Taquicardia paroxística supraventricular com QRS largo usando as fibras Nodoventriculares (fibras de Mahaim[1])
4. TSV com ECG de base com bloqueio de ramo pre-existente. Ocasionado por velocidade lenta de condução músculo-músculo
5. TSV com hyperkalemia outro distúrbios eletroliticos ou efeito de drogas
6. Ritmo de marcapasso ventricular. A presença do aparelho no exame físico é uma forte chave para marcapasso como causa da taquicardia com QRS largo. Quando o marcapasso está implantado no ápex do VD o evento possui morfologia de BCRE com eixo superior.

1. Gallangher JJ, Smith WM, Kasel JH, et al. Role of Mahaim fibers in cardiac arrhtymias in man Circulation 1981, 64: 176.

In this case the QRS axis is located on inferior right quadrant.

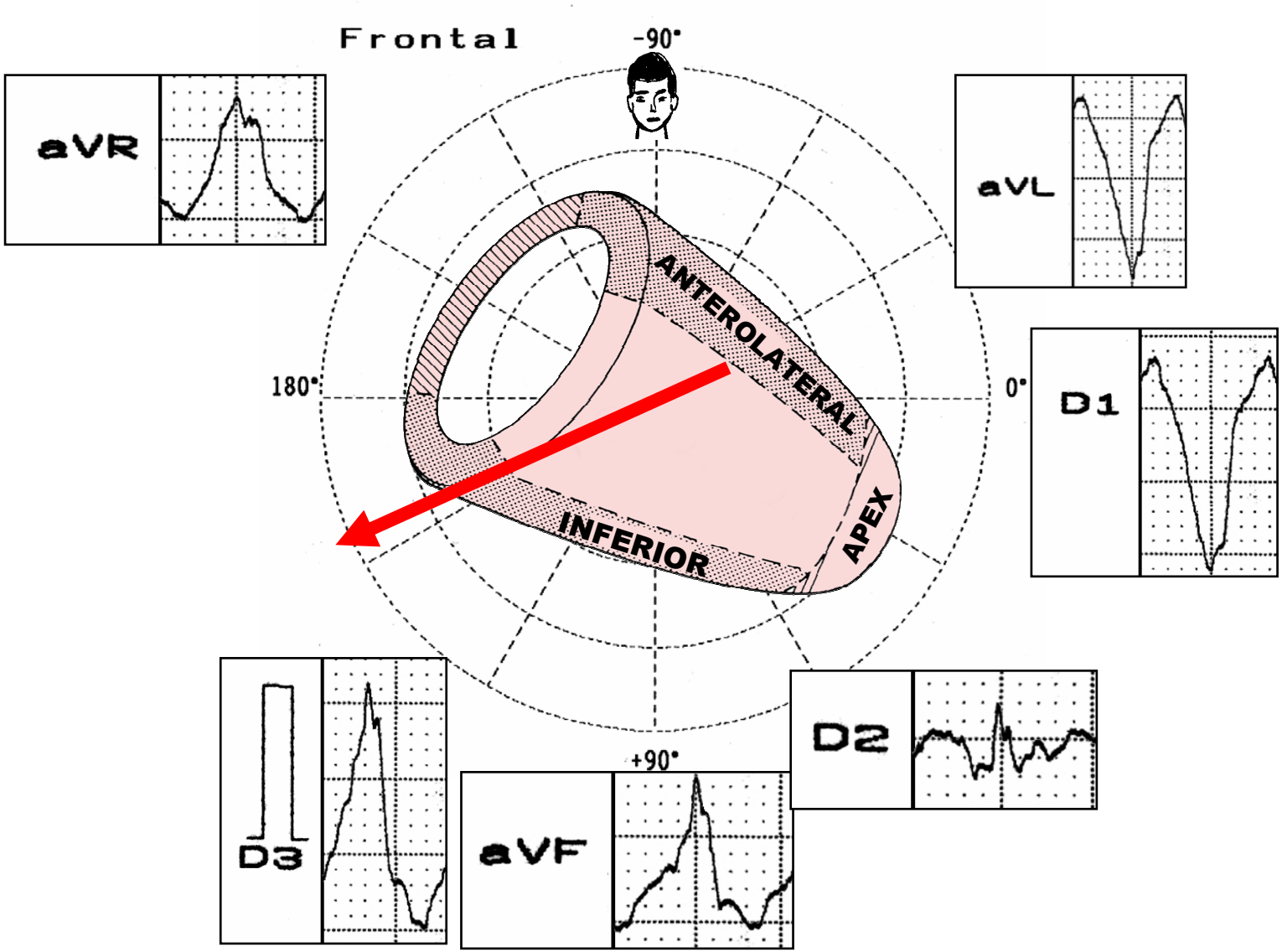
Neste caso, o eixo encontra-se no quadrante inferior direito.



Only WCTs with frontal plane axis in the right superior quadrant are unlikely to be SVT-A.

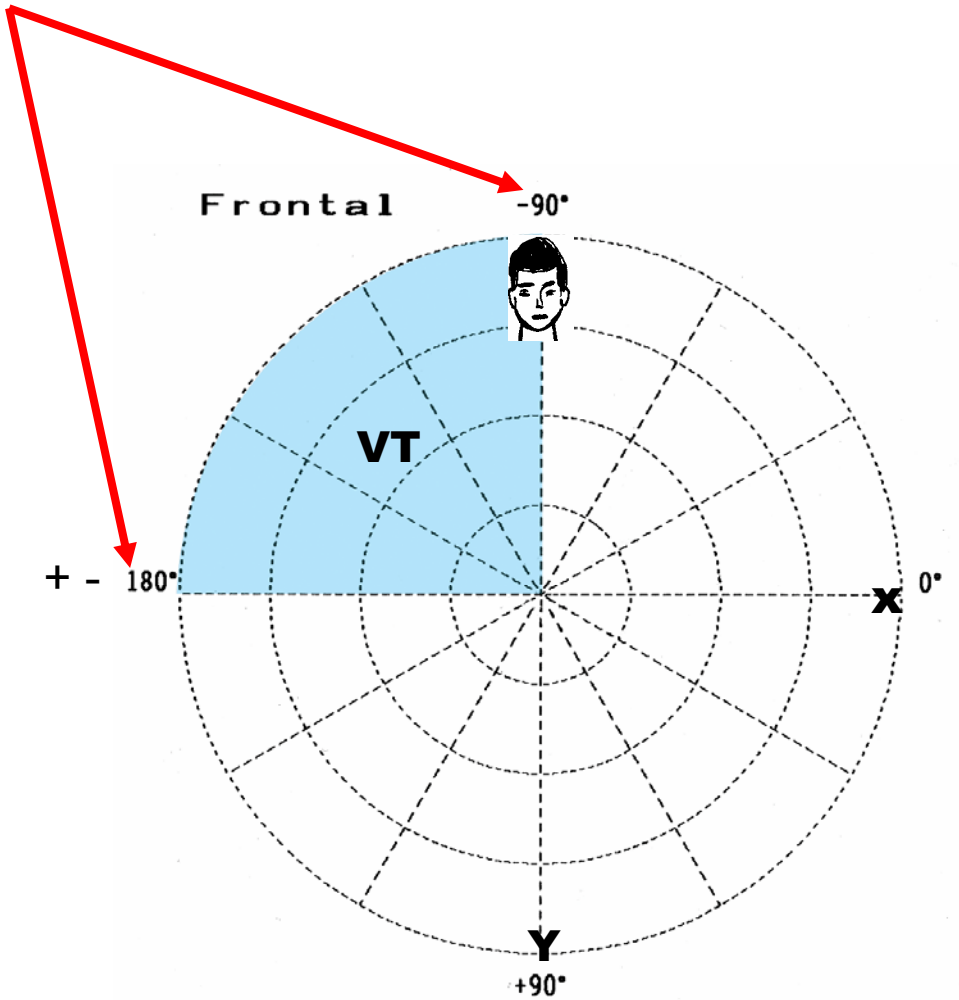
Apenas as taquicardias com QRS largo com eixo no PF no quadrante superior direito não são compatíveis con TSV-A.

POSSIBLE FOCUS LOCATION

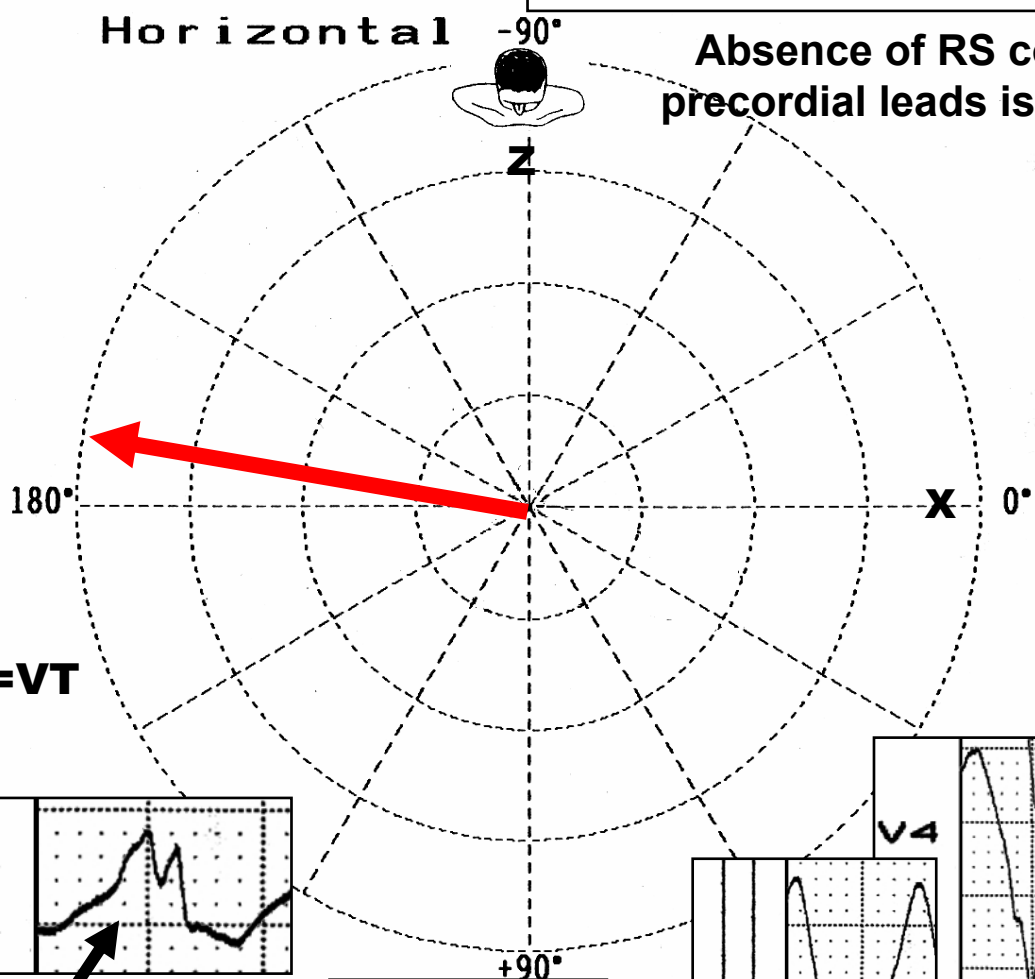


Focus in anaterolateral wall. QRS axis on inferior right quadrant.

QRS axis on superior right quadrant between - 90° and + - 180°, QRS axis "no-man's–lands" or Northwestern axis = VT



Absence of RS complex from V_1 to V_6.

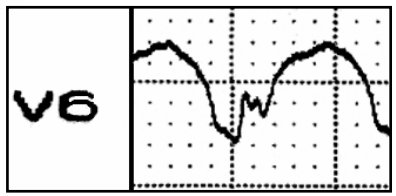


Absence of RS complexes in the precordial leads is suggestive of VT

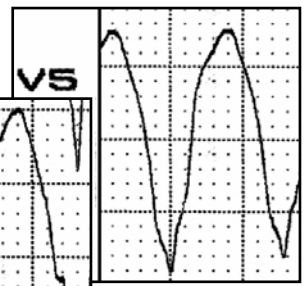
Yes → VT

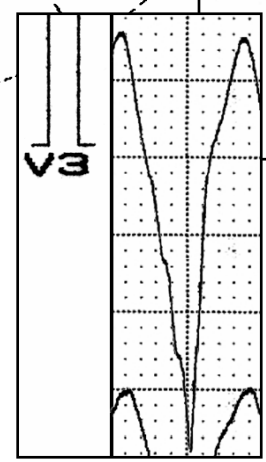
The main QRS vector is oriented from left to right.

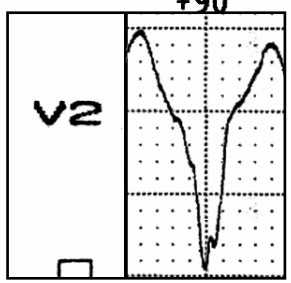
QR or QS in V6 is suggestive of VT



"Atypical" RBBB=VT

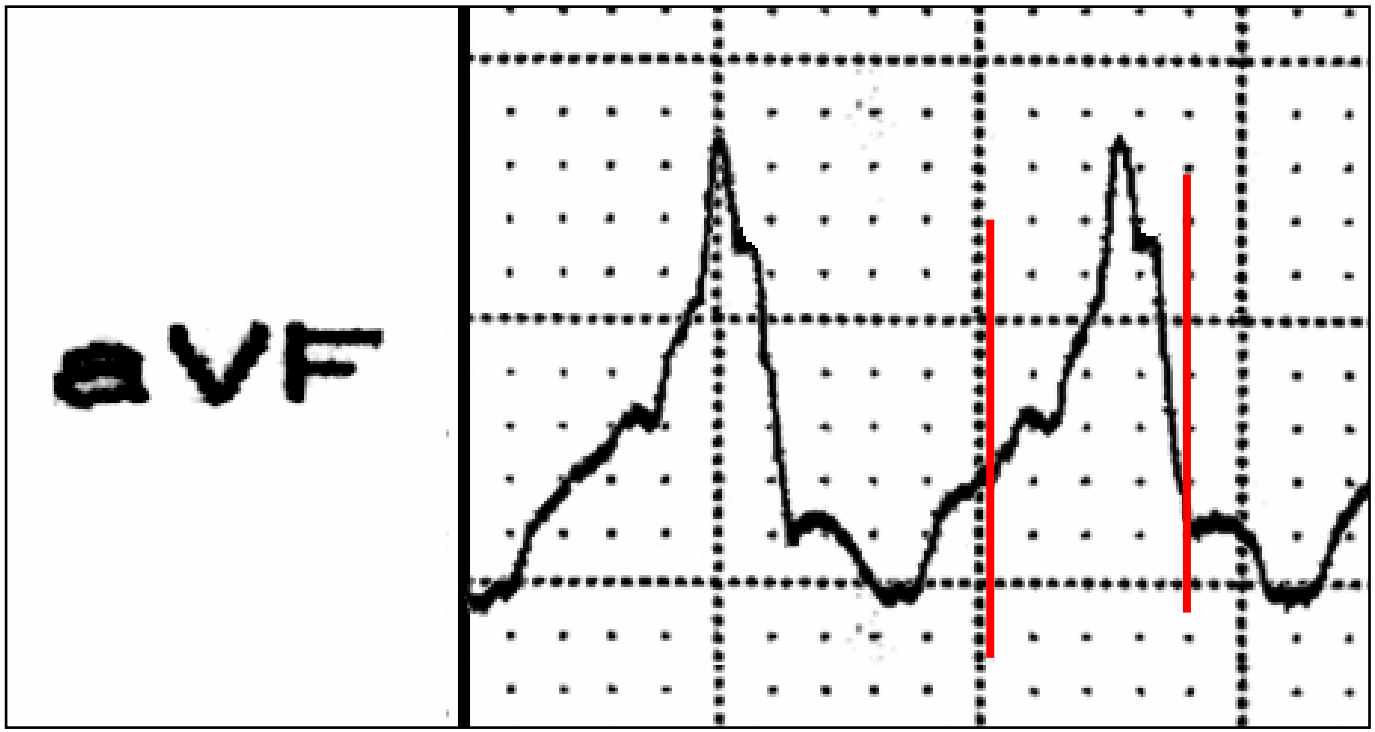






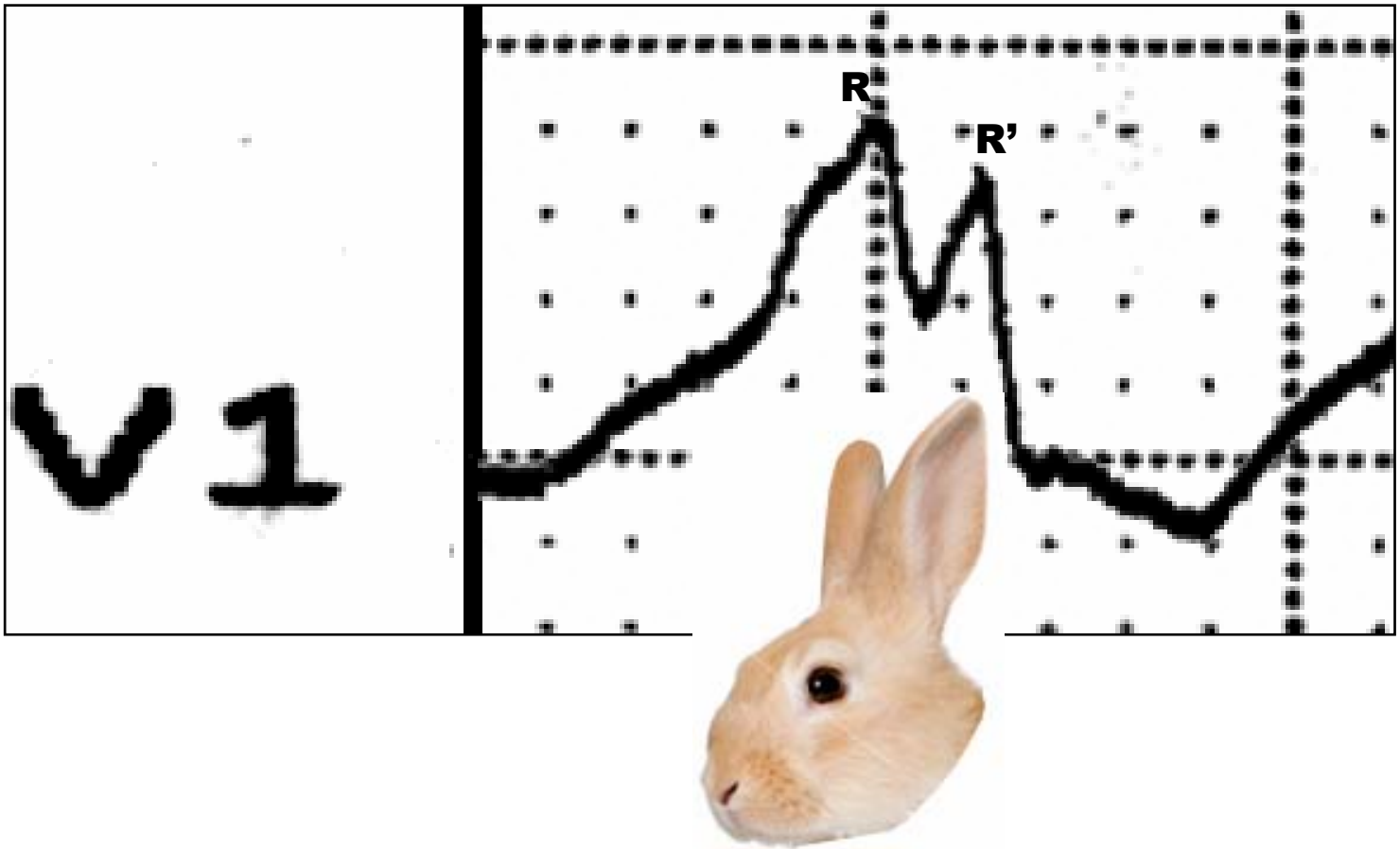
RBBB-like pattern in V_1 indicating that the left ventricle is depolarized before the right one If VT in origin.

Positive precordial Concordance is observed in the VT from an apical focus In this case we have not positive precodial concordance Because V_1 is posiitve.



Heart Rate: near 200bpm. Compatible with VT
QRS Duration: 150ms: compatible with VT **RBBB-pattern >140ms = VT**

LBBB-pattern >160ms



CRBBB is present in V_1, biphasic QRS (RR') = VT.
The voltage of the R wave in V_1 is greater than R': absence of "Rabbit Ears" signal = Rr´. This feature is against VT

1. Gozensky C, Thorne D. Rabbit ears: an aid in distinguishing ventricular ectopy from aberration. Heart Lung. 1974 Jul-Aug;3:634-636.

Step 0: ***Is the patient haemodynamically unstable?:*** YES or NO

Comment: Haemodynamic instability means that the arrhythmia should be addressed immediately. The following algorithm cannot be used where the ventricular response rate is irregular. As an aside, note that one should also include artifact in the differential diagnosis (on a rhythm strip alone), as suggested Knight et al[1].

Step 1: ***Is any one of the following present: CHD, or HF, or past heart surgery, or cardiac enlargement[2]?:*** YES: suggest VT or NO

Comment: Diagnosis of VT is supported by history of prior MI or CHF, physical examination showing cannon A-waves in the jugular venous pulsation, variable S1 heart sounds, chest radiograph showing cardiomegaly or evidence of prior cardiac surgery, and characteristic ECG features: AV dissociation, fusion/capture beats, QRS concordance or typical morphologic features in leads V1 and V6[3].

BRUGADA'S ALGORITHM

Step 2: ***Are there any RS complexes in the V leads?[4]*** YES or NO = VT

Comment: This and the following steps are based on Brugada's algorithm[4] for diagnosis of WCT. Absence of any RS complex in the precordial leads V1..V6 is 100% specific for VT, but is insensitive (26%).

Isenhour et al[5] re-evaluated Brugada's algorithm. These authors observed that the presence of RS complexes in V leads is less effective in differentiating VT and SVT-A than in the original paper, perhaps because the Isenhour´s study included some patients on drug treatment for arrhythmias (excluded from Brugada's study).

1. **Knight BP, Pelosi F, Michaud GF, Strickberger SA, Morady F. Physician interpretation of electrocardiographic artifact that mimics ventricular tachycardia. Am J Med. 2001 Apr 1;110:335-338.**
2. **Gupta AK, Thakur RK. Wide QRS complex tachycardias. Med Clin North Am. 2001 Mar;85:245-266.**
3. **Shah CP, Thakur RK, Xie B, Hoon VK. Clinical approach to wide QRS complex tachycardias. Emerg Med Clin North Am. 1998 May;16:331-360.**
4. **Brugada P, Brugada J, Mont L, Smeets J and Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation. 1991;83:1649-1659**
5. **Isenhour JL, Craig S, Gibbs M, Littmann L, Rose G, Risch R. Wide-complex tachycardia: continued evaluation of diagnostic criteria. Acad Emerg Med. 2000 Jul;7(7):769-73.**

Passo 0: ***Está o paciente hemodinamicamente instável?*** **:** SIM or NÃO

Comentário: a instabilidade hemodinámica assinala que a arritmia deveria ser tratada de imediato. O algaritmo seguinte não pode ser empregado quando a resposta ventricular é irregular. Como uma exceção deveriamos incluir também artefatos no diagnóstico diferencial ou uma tira só como sugere Knight e col [1].

Passo 1: está presente alguma das seguintes condições: ICC, doença coronária, passado de cirurgia, ou cardiomegalia[2]? SIM: sugere TV or NÃO.

Comentários: O diagnóstico de TV é sustentado pela história de IM prévio, ICC, exame físico com em canhão nas veias jugulares, primeira bulha de intensidade variável, Rx de tórax mostrando cardiomegalia ou evidência de cirurgia cardiaca prévia e características do ECG que assinalem dissociação AV como batimentos de fusão e de captura. Também sugere TV a presença de concordância possitiva ou negativa nas precordiais e as típicas morfologias em V1 e V6[3].

ALGORITMO DE BRUGADA

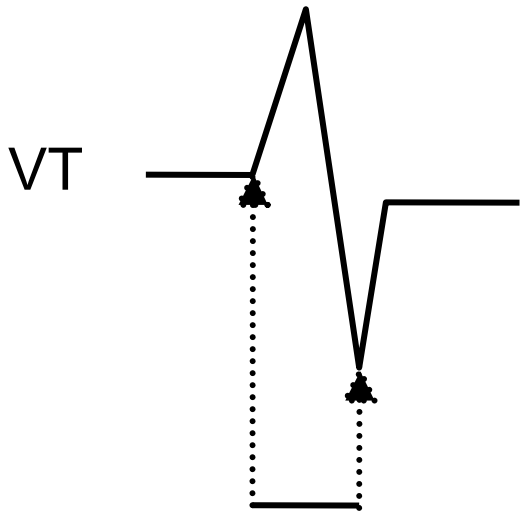
Passo 2: ***Existe algum complexo do tipo RS nas precordiais?*** SIM ou NÃO= TV.

Comentário: Este e os passos seguintes estão baseados no algoritmo de Brugada[4] para o diagnóstico de taquicardia de QRS largo. Ausência de RS nas precordiais é 100% específico para TV, porém pouco sensível (26%). Isenhour e col[5] reavaliaram o algoritmo de Brugada. Os autores observaram que a presença de complexos RS nas precordiais é menos eficaz na diferenciação das TV com as TSV-A provavelmente porque neste último estudo foram incluídos alguns pacientes em uso de drogas excluídos no trabalho de Brugada.

1. **Knight BP, Pelosi F, Michaud GF, Strickberger SA, Morady F. Physician interpretation of electrocardiographic artifact that mimics ventricular tachycardia. Am J Med. 2001 Apr 1;110:335-338.**
2. **Gupta AK, Thakur RK. Wide QRS complex tachycardias. Med Clin North Am. 2001 Mar;85:245-266.**
3. **Shah CP, Thakur RK, Xie B, Hoon VK. Clinical approach to wide QRS complex tachycardias. Emerg Med Clin North Am. 1998 May;16:331-360.**
4. **Brugada P, Brugada J, Mont L, Smeets J and Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation. 1991;83:1649-1659**
5. **Isenhour JL, Craig S, Gibbs M, Littmann L, Rose G, Risch R. Wide-complex tachycardia: continued evaluation of diagnostic criteria. Acad Emerg Med. 2000 Jul;7(7):769-73.**

BRUGADA'S ALGORITHM

Step 3: ***In any of these RS complexes, is the interval from start of R to nadir of S > 100ms?***
YES = VT or NOT



>100ms.

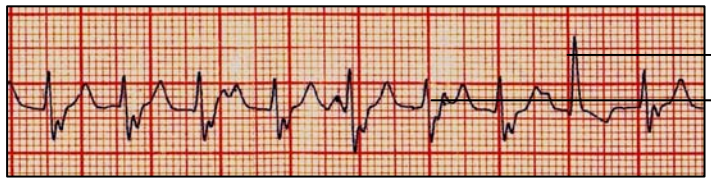
Comment: This criterion is based on the initial observation by Kindwall et al[1]. Whose criteria differ somewhat from those of Brugada in that they used 60ms as the cutoff, and only looked at V_1 and V_2. A duration > 100ms almost excludes SVT-A (98% specific for VT).

Passo 3: qualquer dos complexos RS, o intervalo desde o inicio da R ao nadir da S >100ms. Sim = TV ou Não. Comentário: este critério tem por base a obsevação de Kindwal e col[1]. Os autores empregaram um valor de corte de apenas 60ms e apenas consideraram V_1 e V_2. A duração >100ms exclui praticamente TSV-A uma vez que possui 98% de especificidade.

1. Kindwall KE, Brown J, Josephson ME. Electrocardiographic criteria for ventricular tachycardia in wide complex left bundle branch block morphology tachycardias. Am J Cardiol. 1988 Jun 1;61(15):1279-83.

BRUGADA'S ALGORITHM

Step 4: Is there AV dissociation, or are there fusion or capture beats? YES = VT or NOT
Comment: These are traditional criteria for VT (and pretty reliable when present). In Brugada's study, evidence of AV dissociation was 98% specific for VT.
Capture beat: QRS looks normal because a conducted sinus beat transiently captures the ventricles.**Fusion beat:** QRS is intermediate between normal and the VT complex.



Step 5: Is the QRS complex in V_1 predominantly positive? YES or NO
YES:
Monophasic = VT
Biphasic QR = VT
Triphasic = SVT-A

Next look at V6

Monophasic, QS or QR = VT
RS ratio < 1 = VT
RS ratio > 1 = SVT-A

V1 predominantly negative

R > 30ms or interval to nadir of S > 60ms or a notched or slurred S = VT. NO = SVT-A.

Next look at V6. Is there?

QR or QS or a monophasic R = VT. NO = SVT-A

1. **Wellens HJJ, Bar FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. Am J Med 1978;64:27–33**

ALGORITMO DE BRUGADA

Passo 4: ***Existe dissociação AV ou batimentos de fusão ou captura? SIM= TV ou NÂO***
Comentários: Estes são critérios tradicionais de TV. No manuscrito de Brugada a presença de dissociação AV teve uma especificidade de 98% para TV.
Batimento de Captura: são batimentos que se vem como normais porque o batimento sinusal ativa os ventrículos transitoriamente.
Batimento de fusão: o QRS possui um aspecto intermediário entre o puro batimento sinusal e o puro batimento ectópico.
Passo 5: complexo QRS em V_1 é predominantemente positivo? SIM ou NÃO
SIM
Monofásico = VT
Bifásico QR = VT
Trifásico = SVT-A

A seguir observar V6

Monofásico,QS ou QR = TV
Relação RS < 1 = VT
Relação RS > 1 = SVT-A

V1 predominantemente negativo

R > 30ms ou intervalo até o nadir de S >60ms ou um entalhe na rampa descendente do S = VT. NÃO = SVT-A.

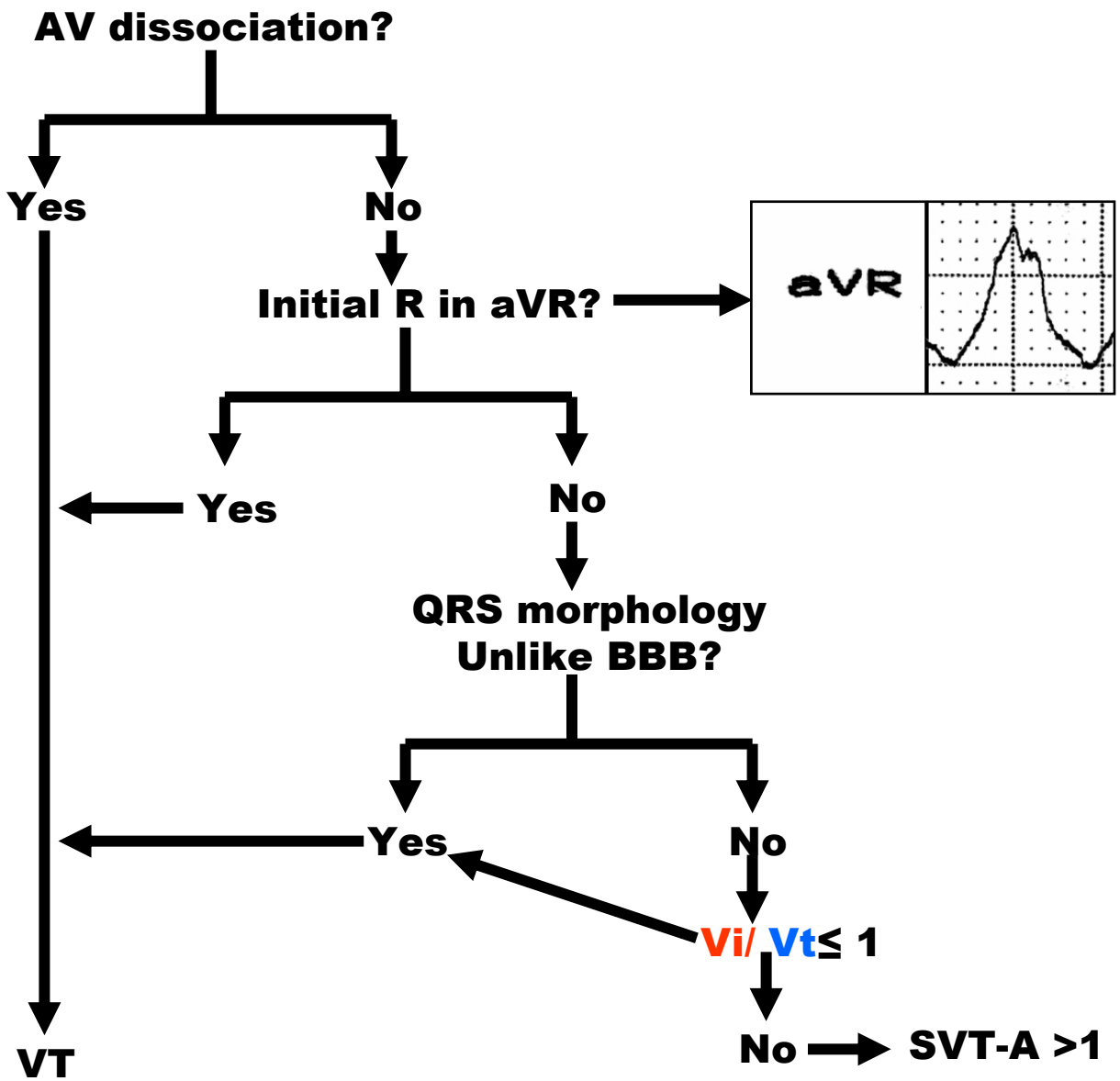
Observar V6

Existe?
QR, QS ou uma R monifásica = VT. NÃO = SVT-A

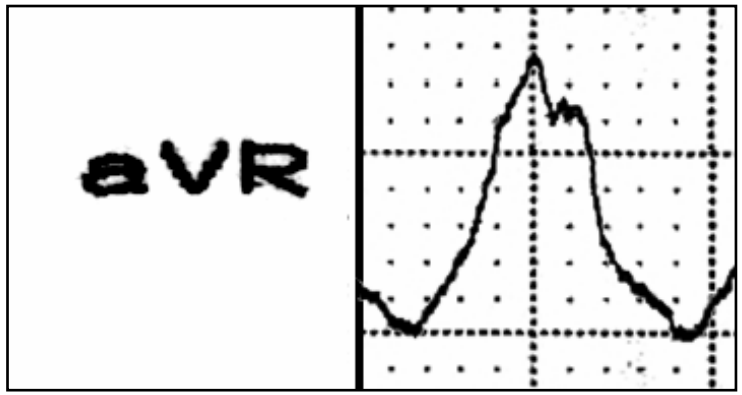
Vereckei´s Algorithm

VENTRICULAR ACTIVATION VELOCITY RATIO

Vi: initial excursion velocity of QRS complex.
Vt: terminal excursion velocity of QRS complex
The ratio between the voltages recorded during the first and the last 40 ms of the QRS complex helps distinction between ectopy and aberration: a ratio ≤ 1 suggests VT whereas a ratio > 1 indicates SVT-A.



1. Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. Heart Rhythm. 2008; 5: 89-98.

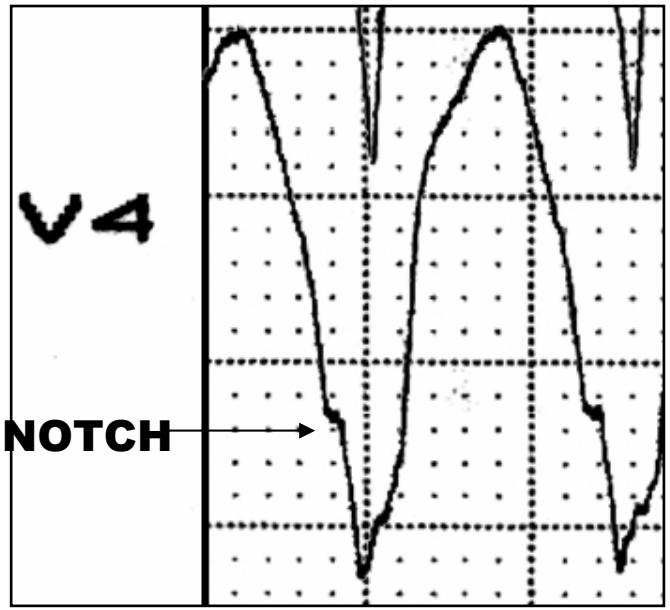


FIRST STEP[1]
PRIMEIRO PASSO

Presence of an initial R wave in lead aVR
Presença de uma R inicial na derivação aVR

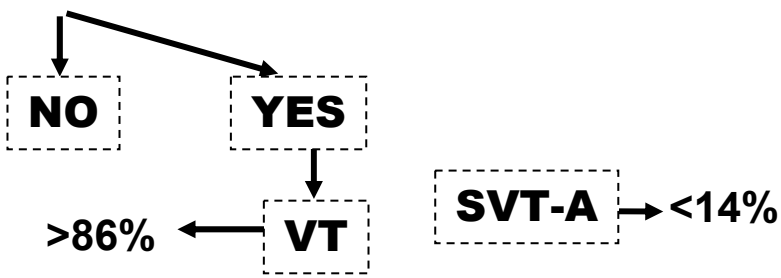
- **NO** → **> 98,6%** **VT**
- **YES** → **VT**, **SVT** → **< 1,5%**

1. Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. Heart Rhythm. 2008; 5: 89-98.

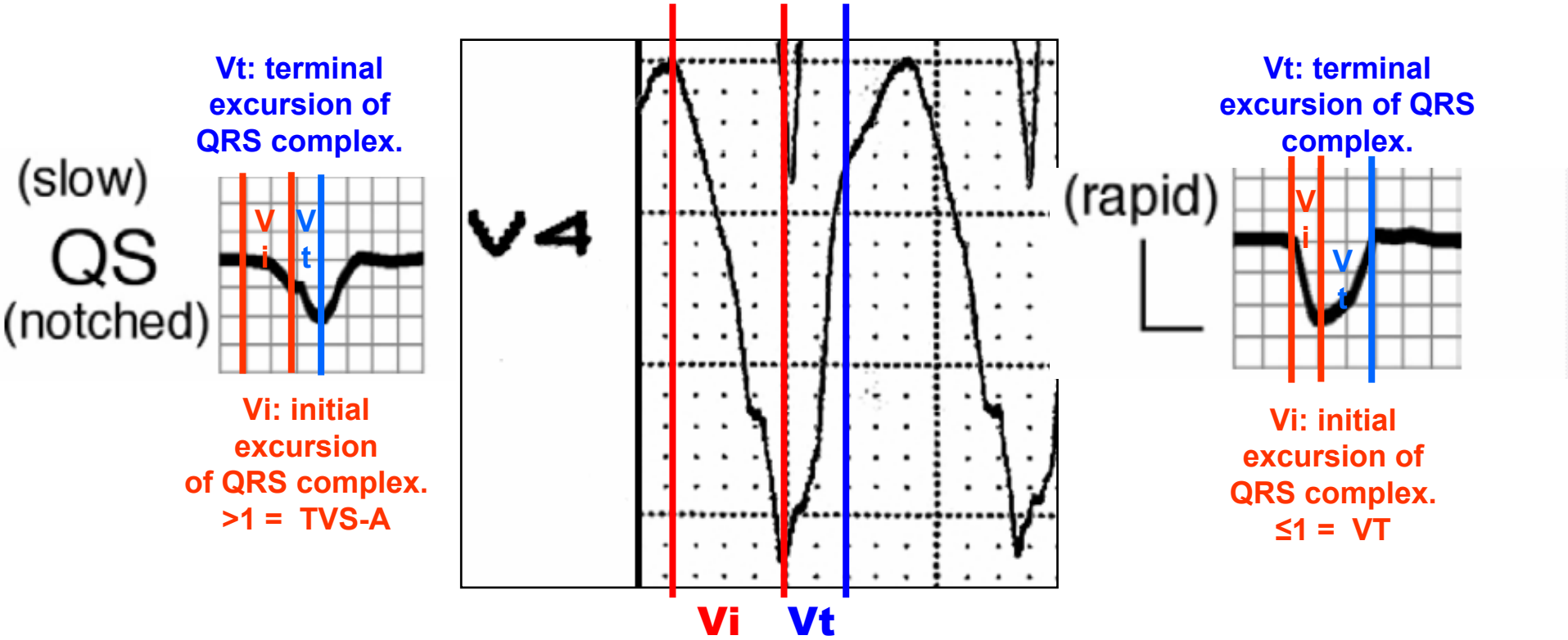


THIRD STEP TERCEIRO PASSO

Presence of a notch or the descending limb of a negative onset and predominantly negative QRS

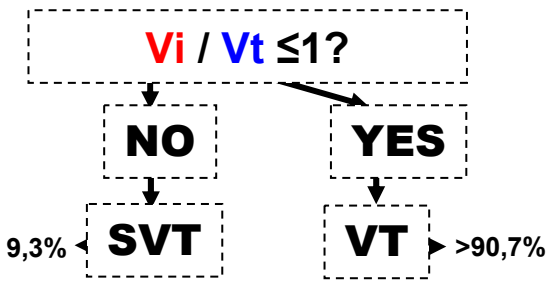


Presença de um entalhe no ramo descendente de um complexo QRS negativo ou predominantemente negativo



FOURTH STEP
QUARTO PASSO

Vi: initial excursion velocity of QRS complex.
Vt: terminal excursion velocity of QRS complex



Ventricular activation-velocity ratio The vertical excursion (in millivolts) recorded during the initial (Vi) and terminal (Vt) 40 ms of the QRS complex.
If ≤1= VT
or >1=SVT.

Ventricular activation-velocity ratio (Vi/Vt) ≤ 1= VT

Relação velocidade de ativação ventricular: relação da velocidade da excursão inicial **Vi**/ velocidade da excursão terminal **Vt**.

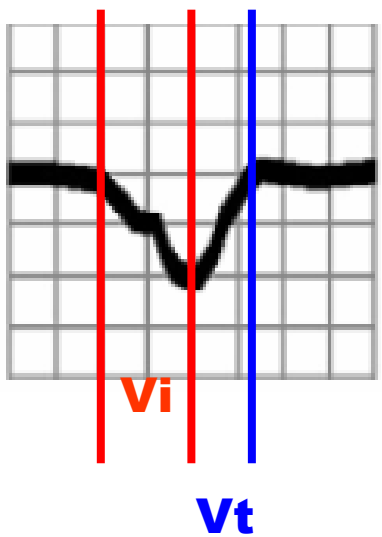
Ventricular activation-velocity ratio (Vi/Vt)

Because in SVT-A, only one portion of the His-Purkinje system is blocked while another mediates initial ventricular activation, the first part of the QRS should have relatively rapid excursion compared with the terminal part of the QRS. The vertical excursion (in milivolts) recorded during the initial **(Vi)** and terminal **(Vt)** 40 ms of the QRS complex. If ≤1= VT or >1=SVT-A.

Relação velocidade de ativação ventricular: relação da velocidade da excursão inicial Vi/ velocidade da excursão terminal Vt.

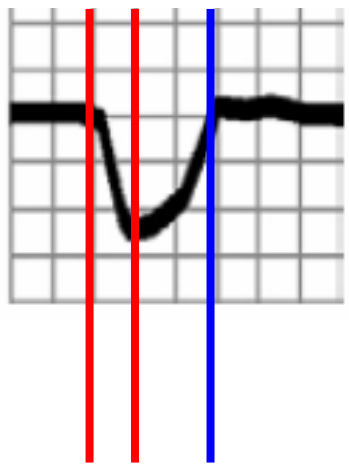
>1=SVT

QS
(notched)



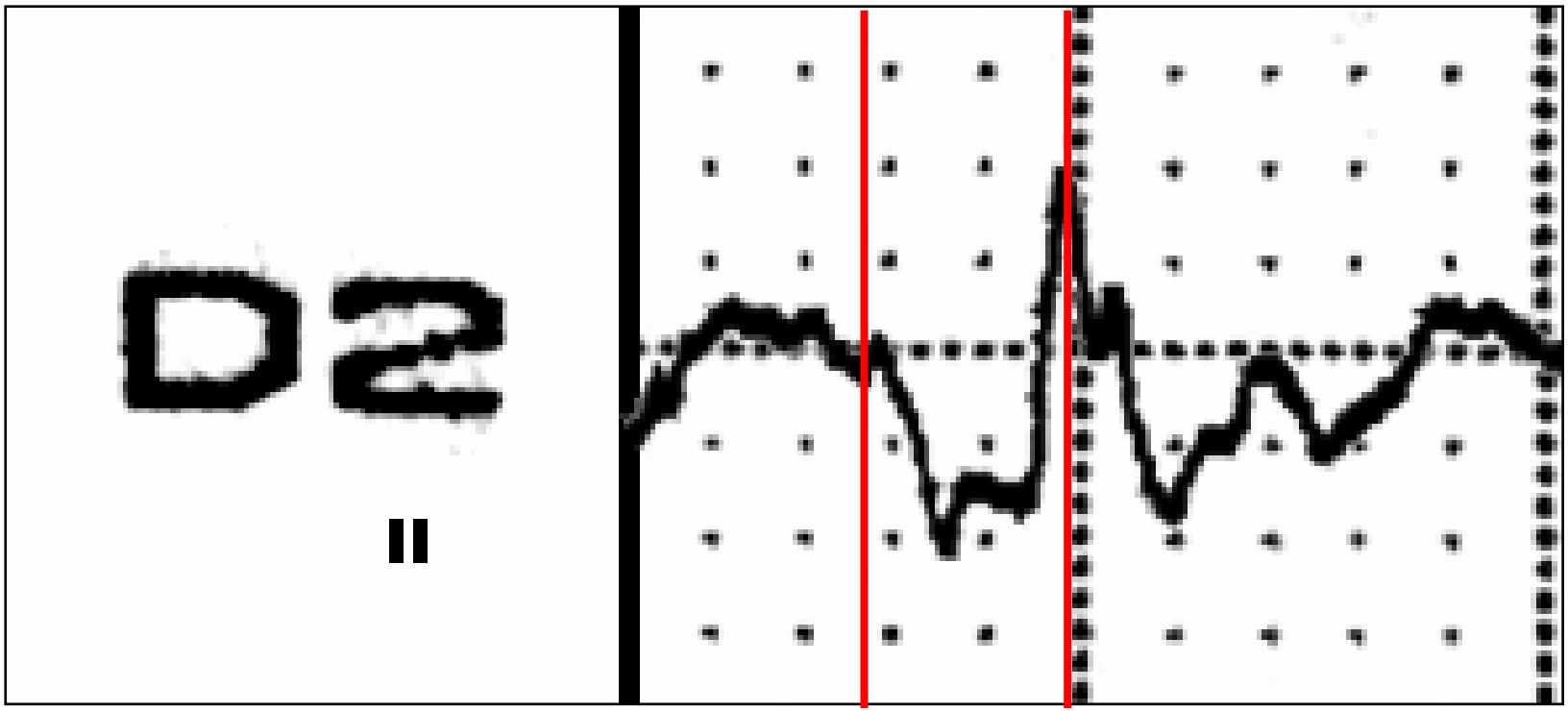
≤1= VT

(rapid)



Pava´s criteria[1]

A R-wave peak time ≥ 50 ms at II is a simple and highly sensitive criterion that discriminates VT from SVT-A in patients with WCTs.



In the present case the R-wave peak time is near 80ms

1. Pava LF, Perafán P, Badiel M, Arango JJ, Mont L, Morillo CA, Brugada J. R-Wave Peak Time at DII: A New Criterion to Differentiate Between Wide Complex QRS Tachycardias. Heart Rhythm 2010, in press

	VT >80% of cases TV >80% dos casos	SVT-A: ≈15% of cases TSV-A: ≈15% dos casos
History / História	Previous MI Angina, CHF, Rarely without Structural heart disease, Older patient, More frequent hemodynamically instable. IM prévio, ICC, raramente sem doença estrutural, paciente de maior idade, mais frequentemente hemodinamicamente instável.	May or may not to has structural heart disease. Younger patient. More often have a history of previous episodes of arrhythmia . Episodes occurring for longer than 3 years. More frequent hermodynamically stable. Pode ou não ter cardiopatia estrutural, paciente mais jovem, mais frequentemente história prévia de episódios arrtimicos, estes por mais de três anos, mais frequentemente hemodinamicamente estável.
Physical finding of AV dissociation Exame físico demonstrando dissociação AV	Frequent: “cannon” A waves in the jugular venous wave forms if the atria are in sinus rhythm. S1 of variable intensity due to loss of AV synchrony, variations in systolic blood pressure unrelated to respiration. Frequente onda A jugular em canhão se o átrio está em ritmo sinusal, primeira bula de intensidade variável, consequência da assincronia AV, variações na pressão arterial sistólica não relacionadas a respiração.	NO Não
Response to vagal maneuvers (Valsalva, carotid sinus pressure) or IV adenosine Resposta as manobras vagais: prova de Valsalva, pressão no seio carotideo e injeção EV de adenosina.	May cause transient retrograde block in the AV node and AV dissociation Pode causar bloqueio retrogrado transitório no nó AV e dissociação AV.	Termination Terminação
Family history of premature (<40 y) sudden death História familiar de morte súbita prematura (< 40 anos)	Any patient with a strong family history of premature (<40 y) SCD should be evaluated for genetic arrhythmia syndromes, including LQTS, SQTS, BrS, CPVT, ARVD, and HCM Qualquer paciente com história familiar de morte súbita prematura (< 40 anos) deveria ser evaluado para síndromes genéticas, incluindo SQTL, SQTC, SBr, TVPC, DAVD e CMH.	Absent Ausente

	VT >80% of cases TV >80% dos casos	SVT-A: ≈15% of cases TSV-A: ≈15% dos casos
QRS axis in right superior quadrant Between -90° and+ - 180° Eixo QRS no quadrante superior direito entre -90° e +- 180°	Exclusive: "no-man's–lands" or Northwestern QRS axis Eixo do QRS no quadrante superior direito.	Not possible. Não possível.
Absence of RS complexes in the precordial leads Ausência de complexos QRS do tipo RS nas precordiais	Characteristic Característico	No Não
RS duration > 100ms in any precordial leads Duração RS > 100ms em qualquer precordial	Characteristic Característico	No Não
QR or QS in V6 Padrão QR ou QS em V6	Suggestive Sugestivo	No Não
QRSd Duração do QRS	LBBB-pattern >160ms RBBB-pattern >140ms QRSd between 120 to 140ms VT without structural heart disease. Padrão de BCRE > 160ms Padrão de BCRD > 140ms Duração do QRS entre 120 e 140ms = TV sem cardiopatia estrutural	LBBB-pattern <160ms RBBB-pattern <140ms Padrão de BCRE < 160ms Padrão de BCRD < 140ms
W or Rs pattern in V1 "Ambiguous patterns" Padrão em W ou Rs em V1 "Padrão ambíguo"	Suggestive Sugestivo	No Não
Presence of multiple WCT configurations Presencia de múltiplas configurações no complexo QRS largo	Suggestive. Present in 50% of cases Sugestivo. Presente em 50% dos casos	Rare. Present in 8% of cases Raro. Presente em 8% dos casos

	VT TV	SVT-A TSV-A
Presence of an initial R wave in aVR Presença de onda R inicial em aVR	Suggestive Sugestivo	Absent Ausente
Width of an initial r or q wave >40 ms in aVR Onda r ou q inicial larga (>40ms) em aVR	Suggestive Sugestivo	Absent Ausente
Notching on the initial downstroke of a predominantly negative QRS complex in aVR Entalhe na rampa descendente quando QRS é nagativo em aVR	Suggestive Sugestivo	Negative Negativo
Presence of completely negative QRS complexes in all three standard limb leads Presença de complexos QRS negativos nas três derivações standard dos membros	Suggestive[1] Sugestivo	No Não
Clean S downstroke in V1-V2 Onda S de V1-V2 sem entalhes "limpa"	No Não	Suggestive Sugestivo
Ventricular activation-velocity ratio (V_i/V_t) Taxa de velocidade da ativação ventricular (V_i/V_t)	≤ 1 Suggestive ≤1 Sugestivo	>1
Concordant pattern: When the precordial leads consist of complexes that are entirely negative or entirely positive during WCT **Padrão concordante:** Quando os complexos QRS das precordiais são totalmente negativos ou positivos durante o taquicardia de QRS largo.	Strong indicator of VT Forte indicador de TV	No Não
V_1 when RBBB-pattern V1 quando padrão de BCRD	Monophasic[2] or diphasic complex R, qR or RS. The voltage of the R wave in V_1 < R':: "Rabbit Ears" signal. Complexo monofásico ou bifásico do tipo R, qR ou RS. A voltagem da R em V1 < R' Sinal da orelha de coelho	Triphasic pattern rSr´, rR´, rsr´, and rSR´. The voltage of the R wave in V_1 > R': absence of "Rabbit Ears" signal. Padrão trifásico rSr', rR', rsr' e rSR'. A voltagem da R em V1 > R': ausência do sinal da orelha de coelho.

1. Reddy GV, Leghari RU. Standard limb lead QRS concordance during wide QRS tachycardia. A new surface ECG sign of ventricular tachycardia. Chest.1987 Oct;92 (:763-765.
2. Gulamhusein S, Yee R, Ko PT, Klein GJ. Electrocardiographic criteria for differentiating aberrancy and ventricular extrasystole in chronic atrial fibrillation: validation by intracardiac recordings. J Electrocardiol. 1985 Jan;18:41-50.

	VT TV	SVT-A TSV-A
V_6 when RBBB-pattern **V6 com padrão de BCRD**	rS, Qrs, QS, QR or a monophasic R wave, R/S ratio <1 rS, Qrs, QS, QR ou R monofásica. Relação R/S <1	qRs, Rs, or RS R/S ratio >1 qRs, Rs ou RS. Relação R/S >1
V_1 when LBBB-pattern **V1 com padrão de BCRE**	rS or QS with broad initia r wave >30ms and slow descendent S wave (≥60ms). A taller R wave during WCT than in sinus rhythm suggest VT. rS ou QS com onda r inicial larga (>30ms) e rampa descendente da S lenta (≥60ms). Uma onda R de maior voltagem do que o complexo de base durante o evento sugere TV.	rS or QS with narrow initia r wave or rapid , smooth to the nadir of the S wave rS ou QS com onda r inicial estreita ou rápida e rampa descendente da S rápida.
V_6 when LBBB-pattern **V6 com padrão de BCRE**	QR, QS, QrS and Rr	RR or monophasic R wave RR ou onda R monofásica
Combination of LBBB pattern and right axis[1] **BCRE com eixo a direita**	Very suggestive Very sugestivo	Absent Ausente
Combination of RBBB with normal axis entre 0° and +90° **BCRD com eixo normal entre 0° e +90°**	No	Very suggestive Very sugestivo
Fusion beats(AV dissociation) [1;2] **Batimentos de fusão**	Strong evidence Forte evidência	Absent Ausente
Capture beats (AV dissociation) **Batimentos de captura**	Strong evidence Forte evidência	Absent Ausente

1. Dressler W, Roesler H. The occurrence in paroxysmal ventricular tachycardia of ventricular complexes transitional in shape to sinoauricular beats; a diagnostic aid. Am Heart J. 1952 Oct; 44: 485-493.
2. Xenakis E, Costeas F. Transitional ventricular complexes in the diagnosis of paroxysmal ventricular tachycardia. Diagnostic contribution of procaine amide. Cor Vasa. 1966; 8: 113-118.

Capture beats(AV dissociation) **Batimentos de captura**	Strong evidence Forte evidência	Absent Ausente
QRS pattern during WCT differs from that in sinus rhythm with BBB **Padrão do QRS durante o evento diferente de aquele do ritmo sinusal com bloqueio de ramo**	Suggestive Sugestivo	Exclude Exclui
1)Had the patient experienced a prior myocardial infarction? **2)Did symptoms of tachyarrhythmia start only after the infarction?** **1) Teve o paciente IM?** **2) O evento ocorrera após IM?**	If yes very suggestive[2] Confirmado sim muito sugestivo	Negative Negativo
Measurement of QRS onset to the predominant peak or nadir of the QRS complex in MCL6 and V6 **Medida do inicio do QRS ao pico da R ou ao nadir da S nas derivações MCL6 e V6**	≥ 70ms	≤ 50ms

1. Akhtar M, Shenasa M, Jazayeri M, Caceres J, Tchou PJ. Wide QRS complex tachycardia. Reappraisal of a common clinical problem.Ann Intern Med. 1988 Dec 1;109:905-912.
2. Tchou P, Young P, Mahmud R, Denker S, Jazayeri M, Akhtar M. Useful clinical criteria for the diagnosis of ventricular tachycardia. Am J Med. 1988 Jan;84:53-56.
3. Drew BJ, Scheinman MM, Dracup K. MCL1 and MCL6 compared to V1 and V6 in distinguishing aberrant supraventricular from ventricular ectopic beats. Pacing Clin Electrophysiol. 1991 Sep;14:1375-1383.

The classic diagnostic differential criteria among WCTs include:

1) **AV dissociation,** characterized by absence of any relationship between QRS complexes and P waves; this phenomenon is at times immediately recognizable but more often can be discovered only by physical examination or a detailed analysis of the tracing: Second degree ventriculo-atrial block, characterized by a relationship between QRS complexes and P waves, but with more ventricular complexes than P waves, fusion and/or capture beats.
2) **Concordant precordial pattern**, a sign that can be also expressed as absence of RS (or even rs, Rs, rS) complexes in the precordial leads. Positive concordant patter is possible to observe in Antidromic CMT and atrial flutter with conduction over a left-sided accessory pathway.
3) An interval > 100 ms from the beginning of the QRS complex to the nadir of S wave in any precordial lead.
4) Vagal maneuvers and analysis of previous ECGs recorded during sinus rhythm, if available, can provide further keys to the diagnosis. Some criteria proposed in the past, such as QRS axis or QRS duration, are nowadays no longer considered; in addition, it has been demonstrated that items such as age, hemodynamic status, heart rate and regularity of R-R intervals may be misleading and should not be taken into account.
5) Analysis of QRS configuration in leads V1 and V6 is a keystone in distinguishing the origin of WCT: diagnostic criteria rely upon the assumption that aberration is due to a functional BBB, whereas ectopy derives from a totally abnormal activation of the ventricles. Aberration, thus, results in a "typical" BBB pattern, whereas ectopy is expressed by an "atypical" BBB. Specific criteria, based on analysis of leads V1 and V6, have been developed to distinguish the two conditions from each other.
6) The criteria based on QRS configuration, however, suffer from limitations since unexpected complicating factors, such as a previous MI, can result in an "atypical" form of BBB even in the presence of SVT.

A algorithm has been introduced, based on analysis of lead aVR only[1]. Any of the following features, observed in this lead, pinpoints a diagnosis of ventricular tachycardia:

- A dominant R wave (R or Rs complexes) in aVR
- An initial q or r wave with duration > 40 ms (qR or rS complexes);
- A notch in the descending Q wave limb in a negative (Qr or QS) complex.

In the absence of these signs…..

- The ratio between the voltages recorded during the first and the last 40 ms of the QRS complex helps distinction between ectopy and aberration: a ratio ≤ 1 suggests VT whereas a ratio > 1 indicates SVT-A.

A hard diagnostic problem is associated with preexcited tachycardia, the condition resulting whenever SVT impulses are conducted to the ventricles over an AP. This situation is far more rare than ectopy and aberration, and can be ruled out in the presence of **negative precordial concordance (QS complexes in all the chest leads) or deep q waves in a precordial lead other than V1. A QRS morphology not consistent with any of the typical patterns observed in the various locations of the APs rules out a preexcited tachycardia, too.**

1. **Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. Heart Rhythm. 2008; 5: 89**

Atrial Fibrillation in Wolff-Parkinson-White Syndrome

Atrioventricular reentry can initiate AF, which can lead to disastrous consequences if the patient is capable of sustaining a very rapid preexcited ventricular response with conduction over the accessory pathway. The rapid heart rate can produce syncope or, more important, AF may cause ventricular fibrillation and sudden cardiac death.1;2;3 Patients who have been resuscitated from ventricular fibrillation have a rapid preexcited ventricular response as demonstrated during induction of AF in electrophysiological study.2;3 Patients with Wolff-Parkinson-White syndrome who are most susceptible to ventricular fibrillation have a history of atrial fibrillation and reciprocating tachycardia, demonstrate rapid conduction over an accessory pathway during atrial fibrillation and have multiple accessory pathways1.

These patients require aggressive therapy, most commonly radiofrequency catheter ablation of the accessory pathway. Sudden cardiac death in patients with ventricular preexcitation who have symptomatic arrhythmias should be preventable, and these persons should undergo electrophysiological evaluation. If they are capable of sustaining a rapid preexcited ventricular response during AF (eg, the shortest preexcited RR interval is less than 260 ms), catheter ablation of the accessory pathway or aggressive antiarrhythmic drug therapy to prevent conduction over the accessory pathway is necessary.

WCT ≥120ms is caused by various mechanisms, either supraventricular or ventricular. It is important to differentiate between ventricular and supraventricular because it will determine treatment and prognosis of patients.

The ECG features are:

1. Irregular WCT
2. Fast irregular rhythm with wide polymorphic QRS tachycardia without the QRS twisting around the isoelectric baseline
3. Heart rate≥ 170-180 beat/minute
4. RBBB-pattern with extreme right axis.

Treatment

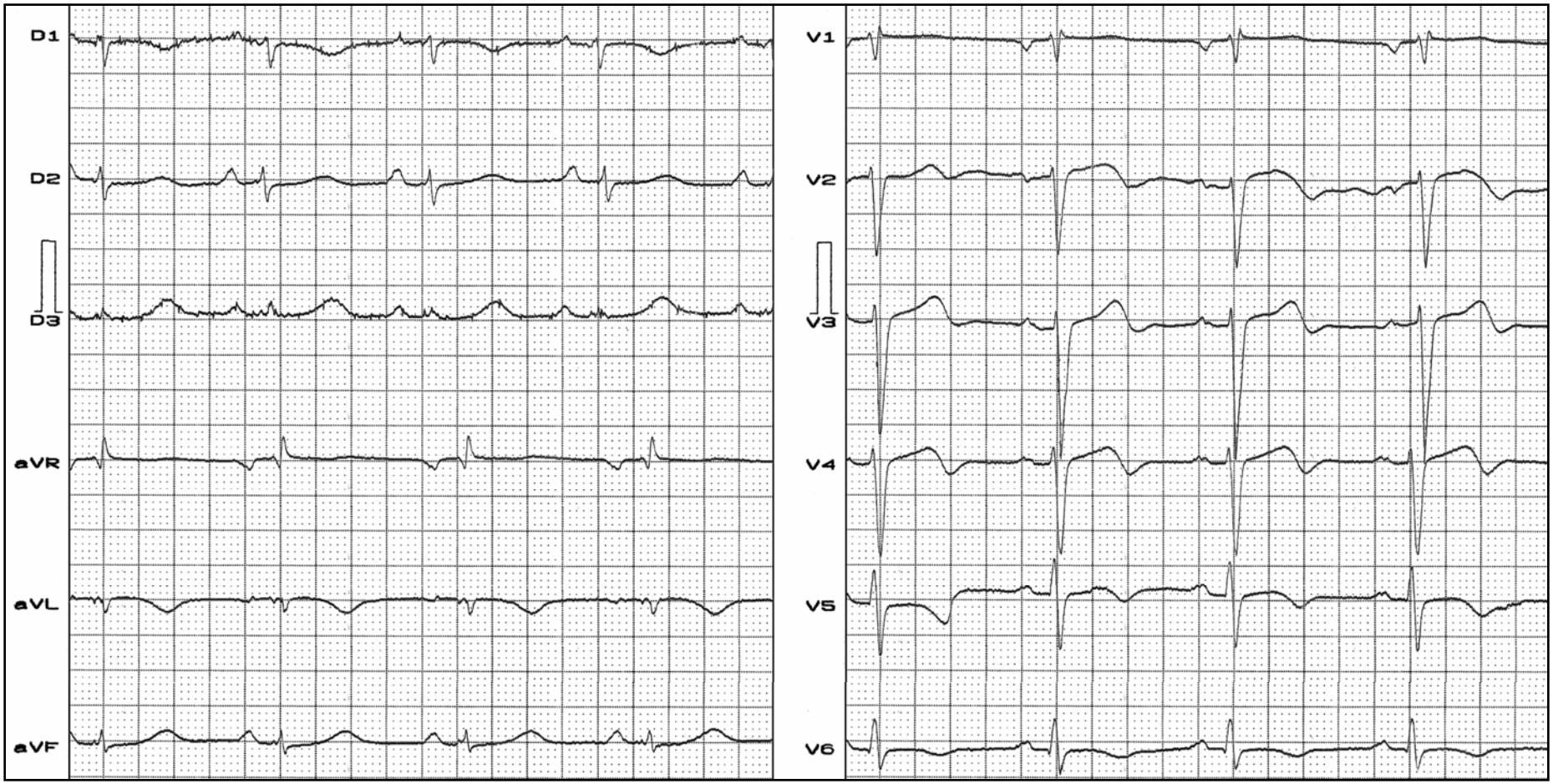
· **Stable:** Intravenous bolus of 300 mg amiodarone administered within 30 minutes and continued with 900 mg/24 hours. During administration of amiodarone, heart rhythm frequently is converted to sinus rhythm with short PR interval and delta wave.

· **Instable:** cardioversion restored sinus rhythm, and the ECG showed a wide QRS complex, short PR interval, and delta wave, indicating the presence of an accessory pathway and pre-excitation.

Sustained AF (greater than 5 minutes or requiring termination due to hypotension). AF is induced during the electrophysiologic study, eventually requiring electrical cardioversion for hypotension. Successful radiofrequency ablation of the accessory pathway completely prevented further inducible AF. Occurrence of sustained AF had a sensitivity of 64% and specificity of 100% for history of syncope. All patients with syncope and AF (12) had a short RR interval between 2 consecutive preexcited QRS complexes during AF at less than or equal to 220). No patient with a short RR interval between 2 consecutive preexcited QRS complexes during AF of greater than 220 ms had a history of syncope. Thus, in young patients with WPW syndrome, occurrence of AF with a rapid ventricular response during EPS correlated well with a history of syncope and may be the cause of synco pe in most patients. EPS may be helpful in identification of young patients with WPW at risk for syncope.

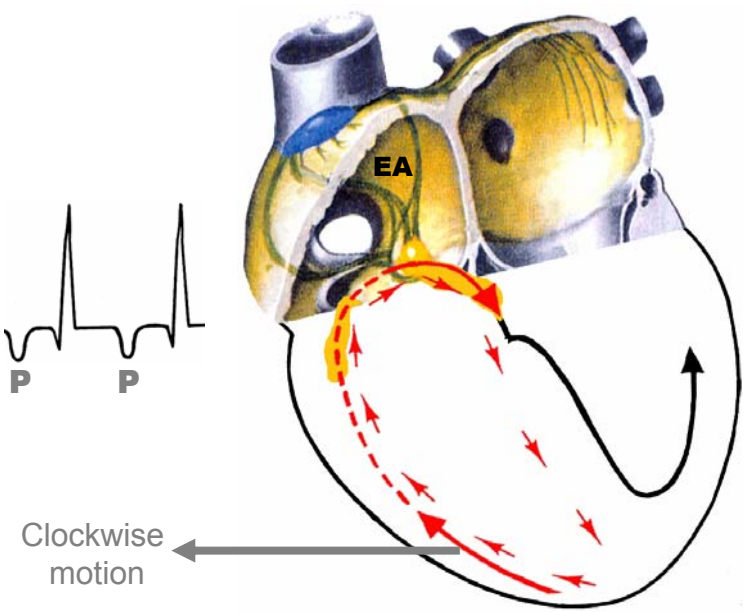
ECG after the event

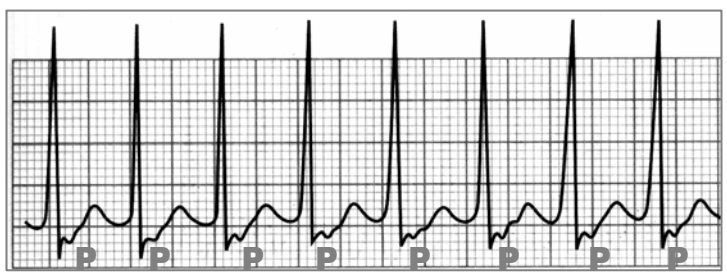
Data: 06/03/2010



Antidromic Circus Movement Tachycardia
Antidromic CMT

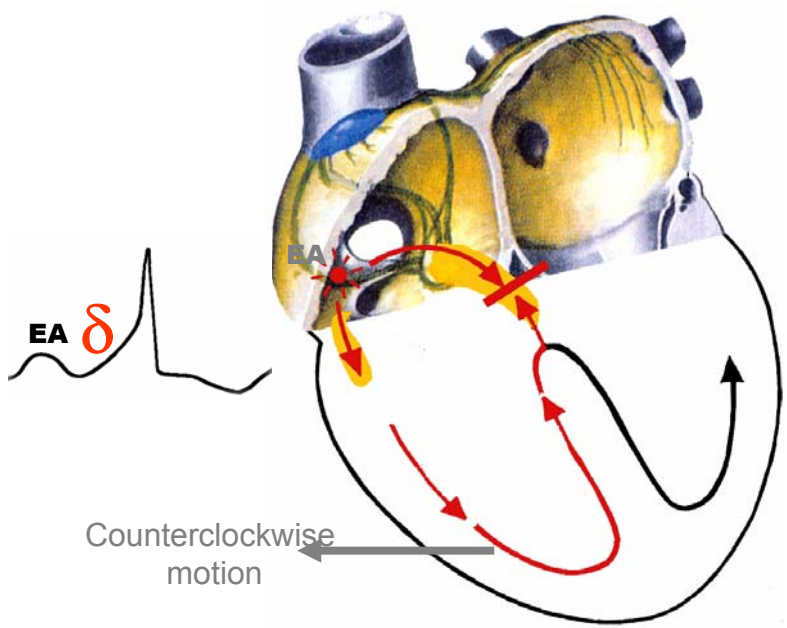
Orthodromic or with narrow QRS: 90%

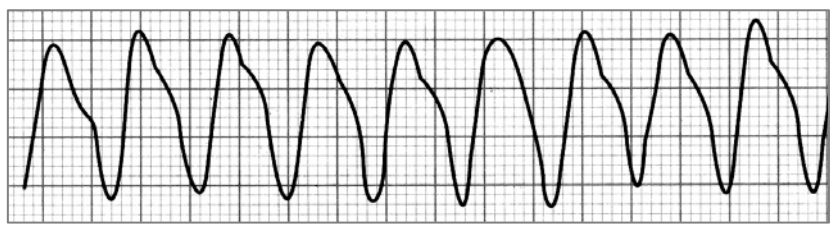




Narrow QRS.
Clockwise macro-reentry motion.
It uses a normal pathway in anterograde fashion
and/or anomalous bundle in retrograde fasion.

Antidromic or with wide QRS: 10%





Wide QRS complex tachycardia (WCT).
Counterclockwise macro-reentry motion.
It uses a normal pathway in retrograde fashion
and/or AP in anterograde fashion.
Identical to VT

Antidromic CMT

Characteristics

1) HR 150 to 250bpm
2) Width QRS complex
3) Usually regular or slightly irregular because retrograde conduction times vary through the fascicles to the atrial: Differential diagnosis with AF with conduction over an APs
4) P´ waves are hidden inside the width of the QRS complexes
5) Strong suspicion when the delta wave during sinus rhythm is the same polarity as the tachycardia in all leads.

Management:

first vagal maneuver …….. Adenosine[1] 6mg IV …..12mg…….. Repeat after 1 minute…….if unsuccessful…..procainamide 10mg/kg/IV …….. If vagal maneuvers and drugs do not terminate ….. Cardioversion.

1. **Marill KA, Wolfram S, Desouza IS, Nishijima DK, Kay D, Setnik GS, Stair TO, Ellinor PT. Adenosine for wide-complex tachycardia: Efficacy and safety*Crit Care Med. 2009 Sep;37:2512-2518.**

Broad QRS paroxysmal Supraventricular Tachycardia Using Nodoventricular fibers (NVFs) (Mahaim fibers)

Mahaim fibers that exhibit decremental AV node-like conduction properties are unusual, comprising < 3% of APs. They are involved in antidromic AV reciprocating (Mahaim) tachycardia with a LBBB pattern. The ECG pattern is very similar to VT. During the sinus rhythym the PR interval is normal or short with or without delta wave. Often the QRS complex is a fusion beats that results from conduction over the extra fiber and over the normal AP.

There is a frequent association of nodoventricular fibers with Ebstein's anomaly, additional APs (Kent) and dual AV nodal pathway conduction. The majority of these APs are long right atriofascicular APs capable of only anterograde conduction.

RFCA of right atriofascicular pathways guided by a distinct Mahaim potential recorded at the anterolateral-to-posterolateral tricuspid annulus or in the RV free wall is safe and highly effective.

True nodoventricular or nodofascicular fibers usually also capable of only anterograde conduction appear to be rare and are associated with pre-excited tachycardia of LBBB morphology not distinguishable from the preexcitation pattern of right atriofascicular pathways. In these APs, a reliable method for mapping and safe RF ablation is lacking because no Mahaim potential could be recorded from the tricuspid annulus in the right midseptal region where ablation may result in elimination of both the slow AV nodal pathway and Mahaim conduction.

Fasciculoventricular Mahaim APs have never been reported to be involved in a tachycardia circuit.

1. **Hluchy J. Mahaim fibers: electrophysiologic characteristics and radiofrequency ablation.Z Kardiol. 2000;89 Suppl 3:136-143.**

Main criteria, references and algorithms for the differential diagnosis of WCTs

1. Dreifus LS, Haiat R, Watanabe Y, Arriaga J, Reitman N. Ventricular fibrillation: a possible mechanism of sudden death in patients with Wolff-Parkinson-White syndrome. Circulation. 1971;43:520-527.
2. Gozensky C, Thorne D. Rabbit ears: an aid in distinguishing ventricular ectopy from aberration. Heart Lung. 1974 Jul-Aug;3:634-636.
3. Wellens HJJ, Bar FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. Am J Med 1978;64:27–33
4. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. N Engl J Med. 1979;301:1080-1085.
5. Wellens HJJ, Bar FW, Vanagl Ej et al. The differentiation between ventricular tachycardia and supraventricular tachycardia with aberrantconduction: the value of the 12-lead electrocardiogram, In Wellens Kulbertus HE, editors: What´s new in electrocardiography? The Hague, 1981, Martinus Nijhoff.
6. Gallangher JJ, Smith WM, Kasel JH, et al. Role of Mahaim fibers in cardiac arrhtymias in man Circulation 1981, 64: 176.
7. Gulamhusein S, Yee R, Ko PT, Klein GJ. Electrocardiographic criteria for differentiating aberrancy and ventricular extrasystole in chronic atrial fibrillation: validation by intracardiac recordings. J Electrocardiol. 1985 Jan;18:41-50.
8. Reddy GV, Leghari RU. Standard limb lead QRS concordance during wide QRS tachycardia. A new surface ECG sign of ventricular tachycardia. Chest.1987 Oct;92 :763-765.
9. Tchou P, Young P, Mahmud R, Denker S, Jazayeri M, Akhtar M. Useful clinical criteria for the diagnosis of ventricular tachycardia. Am J Med. 1988 Jan;84:53-56.
10. Fananapazir L, Packer DL, German LD, Greer GS, Gallagher JJ, Pressley JC, Prystowsky EN. Procainamide infusion test: inability to identify patients with Wolff-Parkinson-White syndrome who are potentially at risk of sudden death. Circulation. 1988;77:1291-1296.
11. Kindwall KE, Brown J, Josephson ME. Electrocardiographic criteria for ventricular tachycardia in wide complex left bundle branch block morphology tachycardias. Am J Cardiol 1988 Jun 1;61:1279-1283.
12. Akhtar M, Shenasa M, Jazayeri M, Caceres J, Tchou PJ. Wide QRS complex tachycardia. Reappraisal of a common clinical problem.Ann Intern Med. 1988 Dec 1;109:905-912.
13. Drew BJ, Scheinman MM, Dracup K. MCL1 and MCL6 compared to V1 and V6 in distinguishing aberrant supraventricular from ventricular ectopic beats. Pacing Clin Electrophysiol. 1991 Sep;14:1375-1383.
14. Brugada P, Brugada J, Mont L, Smeets J and Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation. 1991;83:1649-1659
15. Griffith MJ, de Belder MA, Linker NJ, Ward DE, Camm AJ. Difficulties in the use of electrocardiographic criteria for the differential diagnosis of left bundle branch block pattern tachycardia in patients with a structurally normal heart. Eur Heart J. 1992 Apr;13:478-483.
16. Lee CY, Teo WS. Electrocardiographic case. Irregular broad complex tachycardia. Wolff-Parkinson-White syndrome with atrial flutter-fibrillation. Singapore Med J. 1994 Dec;35(6):635-6, 655-6.
17. Shah CP, Thakur RK, Xie B, Hoon VK. Clinical approach to wide QRS complex tachycardias. Emerg Med Clin North Am. 1998 May;16:331-360.
18. Isenhour JL, Craig S, Gibbs M, Littmann L, Rose G, Risch R. Wide-complex tachycardia: continued evaluation of diagnostic criteria. Acad Emerg Med. 2000 Jul;7:769-773.
19. Isenhour JL, Craig S, Gibbs M, Littmann L, Rose G, Risch R. Wide-complex tachycardia: continued evaluation of diagnostic criteria. Acad Emerg Med. 2000 Jul;7:769-773.
20. Gupta AK, Thakur RK. Wide QRS complex tachycardias. Med Clin North Am. 2001 Mar;85:245-266.

20. Knight BP, Pelosi F, Michaud GF, Strickberger SA, Morady F. Physician interpretation of electrocardiographic artifact that mimics ventricular tachycardia. Am J Med. 2001 Apr 1;110:335-338.
21. Eckardt L, Breithardt G, Kirchhof P. Approach to wide complex tachycardias in patients without structural Heart disease. Heart 2006;92:704-711.
22. Miller JM, Das MK, Yadav AV, Bhakta D, Nair G, Alberte C. Value of the 12-lead ECG in wide QRS tachycardia. Cardiol Clin. 2006 Aug;24:439-445.
23. Zipes DP, Camm AJ, Borggrefe M, M, Buxton AE, Chaitman B, Fromer M, Gregoratos G, Klein G, Moss AJ, Myerburg RJ, Priori SG, Quinones MA, Roden DM, Silka MJ, Tracy C, Smith SC Jr, Jacobs AK, Adams CD, Antman EM, Anderson JL, Hunt SA, Halperin JL, Nishimura R, Ornato JP, Page RL, Riegel B, Priori SG, Blanc JJ, Budaj A, Camm AJ, Dean V, Deckers JW, Despres C, Dickstein K, Lekakis J, McGregor K, Metra M, Morais J, Osterspey A, Tamargo JL, Zamorano JLACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. Circulation. Sep 5 2006;114:e385-484.
24. Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. Heart Rhythm 2008;5:89-98.
25. Sen N, Okuyan H, Türkoğlu S, Tavil Y, Ozdemir M. The missing diagnosis in patients with wide QRS complex tachycardia: WPW syndrome with atrial fibrillation. Anadolu Kardiyol Derg. 2008 Feb 7;8(1):E4-5.
26. Oreto G, Luzza F, Satullo G, Donato A, Carbone V, Calabrò MP. Wide QRS complex tachycardia: an old and new problemG Ital Cardiol (Rome). 2009 Sep;10:580-595.
27. Marill KA, Wolfram S, Desouza IS, Nishijima DK, Kay D, Setnik GS, Stair TO, Ellinor PT. Adenosine for wide-complex tachycardia: Efficacy and safety*Crit Care Med. 2009 Sep;37:2512-2518.
28. Thanavaro JL, Thanavaro S. Clinical presentation and treatment of atrial fibrillation in Wolff-Parkinson-White syndrome. Heart Lung.2010 Mar 4;39:131-136.
29. Pava LF, Perafán P, Badiel M, Arango JJ, Mont L, Morillo CA, Brugada J. R-Wave Peak Time at DII: A New Criterion to Differentiate Between Wide Complex QRS Tachycardias. Heart Rhythm 2010, in press

Colleagues opinions

Very dear friend Prof Andrés R Pérez Riera
This ECG shows a ventricular tachycardia (VT) originated from the left ventricular apex anterior wall.

Why apical VT ?

Because the deep S,s from V2 to V6 and positive R wave in aVR, indicating that the vector is oriented from the lower area of left ventricle toward the upper area.

Why the anterior wall? Because the high R wave in III, indicating that the anterior wall is depolarized before the posterior wall.

Why left ventricle? Because the RBBB-like pattern in V1 indicating that the left ventricle is depolarazide before the righ ventricle.

Why is important to localize the origin of this arrhthmia? Because most of the apical ventricular tachicardia appering in patient without structural heart disease are with left axis deviation, and I think that they are due to a mutation in one of the serine aminoacid fron thre ryanodine as Silvia Priori thought us

Why in the apex ? Because the apex is very sensitive to the adrenergic subtances. In my experience the apical anterior wall is mainly originated from an ischemic event

This monomorphic sustained VT , originated after 14 days of an acute antroapical infartion , after the fibrotic circute is formed(to remember that the soft fibrosis is formed after 14 days but the final strong fibrosis appears between 21 to 28 days)

Which artery are involved in this process ? The LAD or the second marginal

Could be another situation that small emboly stached in the distal arteries and induces a focus of arrhythmia

My friendle regard
Samuel Sclarovsky
Tel aviv

Andrés
Queridisimo Amigo Professor Andrés Haré un análisis morfológico de esta TV

Esta VT proviene del apex del ventrículo izquierdo de la cara anterior

Porque del apex? Porque tiene S desde V2 hasta V6 y R em aVR indicando que los vectores se dirigen de de abajo hacia arriba y de izquierda a la derecha.

Porque del Ventrículo Izquierdo ? Porque V1 es similar a CRBBB

Porque Cara Anterior ? Porque el eje esta a la derecha R altas en III, y S profundas en I, II y aVL indicando que la cara anterior se despolariza antes que la cara posterior

Cual es el significado de esta Taquicardia ? generalmente las taquicardas no sostenidas de la punta cardiaca provienen de la cara posterior, pero este tipo de taquicardia indica que el paciente tiene ya un circuito fibrotico debido, muy probable un infarto apical de mas de 14 dias, debido a una obstrucción de una arteria que irriga esta area, que puede ser una descendente anterior o una segunda marginal

Con un fraternal abrazo y gracias por todos los e-mails que me manda desde arboles de Bs Aires (ahora me voy a fijar con mas detencion sobre esto) y todos los estimulos que ud propaga sobre mi pueblo bastante ultrajado

Samuel Sclarovsky

Amigo

1. Antes de abrir el archivo, tengo el 90% de chances de acertar diciendo que es TV. Porque? Porque el 90% de las taquicardias de QRS ancho son TV. Si hay antecedentes coronarios (infarto) las chances suben al 99%. Pero en este caso se trata de un ex-fumador, en edad de tener enf. coronaria pero sim claro antecedente. entonces solo tengo el 90% de chances de ser TV.
2. ECG: Taquicardia de QRS ancho a 170-180 lpm, eje a la derecha y superior-inferior. V1 con morfologia de BRD, y luego concordancia negativa precordial. No veo clara disociacion AV. Criterios de BRugada positivos para TV salvo disociacion AV.
3. Diagnosticos diferenciales (en orden); 1. TV origen orientado hacia TSVI en pared lateral del VI (aVL y DI negativas) 2. Aleteo Auricular con conduccion 1:1 (el DII es sospechoso), sin embargo, al no tomar antiarritmicos el ciclo del aleteo seria muy lento. Fue el dolor precordial un TEP, y el aumento brusco de presion en AD (eje a la derecha) generó un aleteo de alta respuesta? Podria ser, pero es menos probable.
3. Taquicardia supraventricular con aberrancia: tiene todos los criterios de Brugada en contra (salvo la ausencia de disociacion). Cuando no se ve disociacion hay 2 posibilidades:

a. No la vemos porque no la sabemos encontrar (la mas frecuente en mi experiencia)
b. Porque la TV tiene conduccion VA 1:1 (dependiendo las series es hasta un 50%)

4. Taquicardia antidromica: no presenta la morfologia tipica, casi la descarto.

Le dejo a los estudiosos la aplicacion del algortimo de aVR del amigo Vereckei, y el nuevo del amigo Pava, mencionado hoy mas temprano por uno de los tipos mas lectores que conozco, el amigo Femenia.

Salud

AB

VT from ,anterolateral LV possibly epicardial. MRI may show scar.

TV da parede antero-lateral do VE posivelmente epicárdica. A ressonância nuclear magnética pode revelar cicatriz.

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