Homem idoso, com Cardiopatia Estrutural, Insuficiência Cardíaca Congestiva, Flutter Atrial e Padrão Eletrocardiográfico Brugada tipo 1

Senior man with Atrial Flutter, Structural Heart Disease, Congestive Heart Failure, Syncope episodes and Type 1 ECG Brugada pattern

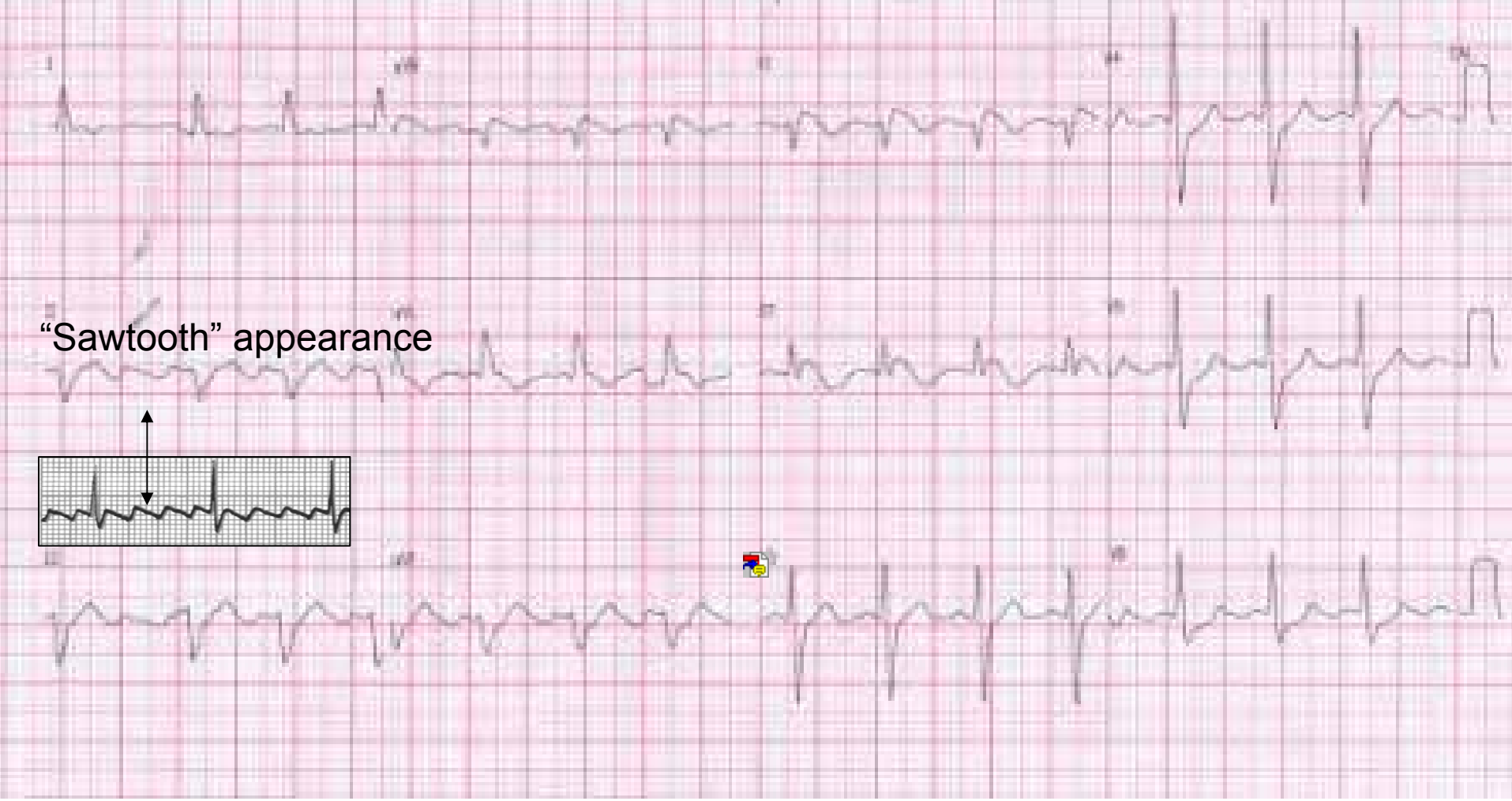
From Raimundo Barbosa Barros MD Nickname " The fox"
Coronary Center Hospital de Messejana Dr. Carlos Alberto Studart Gomes Fortaleza-Ceará-Brazil

Final Comments and ECGs analysis by Andrés Ricardo Pérez-Riera M.D. Ph.D.
In Charge of Electro-vectorcardiogram sector – Cardiology Discipline – ABC Faculty – ABC Foudation – Santo André – São Paulo- Brazil

Holá Professor Andrés:
Homem, 70 anos, com história de insuficiência cardÍaca congestiva. Arritmia supraventricular (flutter atrial) e episódios sincopais.
ECGs exibindo padrão Brugada tipo 1.
História familiar negativa para MS
ECO revela FEVI = 30%.
Atualmente em uso de captopril, carvedilol, espironolactona e warfarina.
Observe a cronologia dos ECGs. Qual o diagnóstico e conduta?
Um abraço a todos do foro
Raimundo Barbosa Barros

Hello Professor Andrés:
70 years old man, with story of congestive heart failure, supraventricular arrhythmia (atrial flutter) and syncopal episodes
ECG showing Brugada type 1 pattern.
Echocardiogram reveals LVEF = 30%.
Currently in use of captopril, carvedilol, spironolactone and warfarin.
Note the timing of the Diagnostic ECGs.
Which is the diagnosis and the appropiate approach?
A hug to forum members

Raimundo Barbosa Barros "The fox"



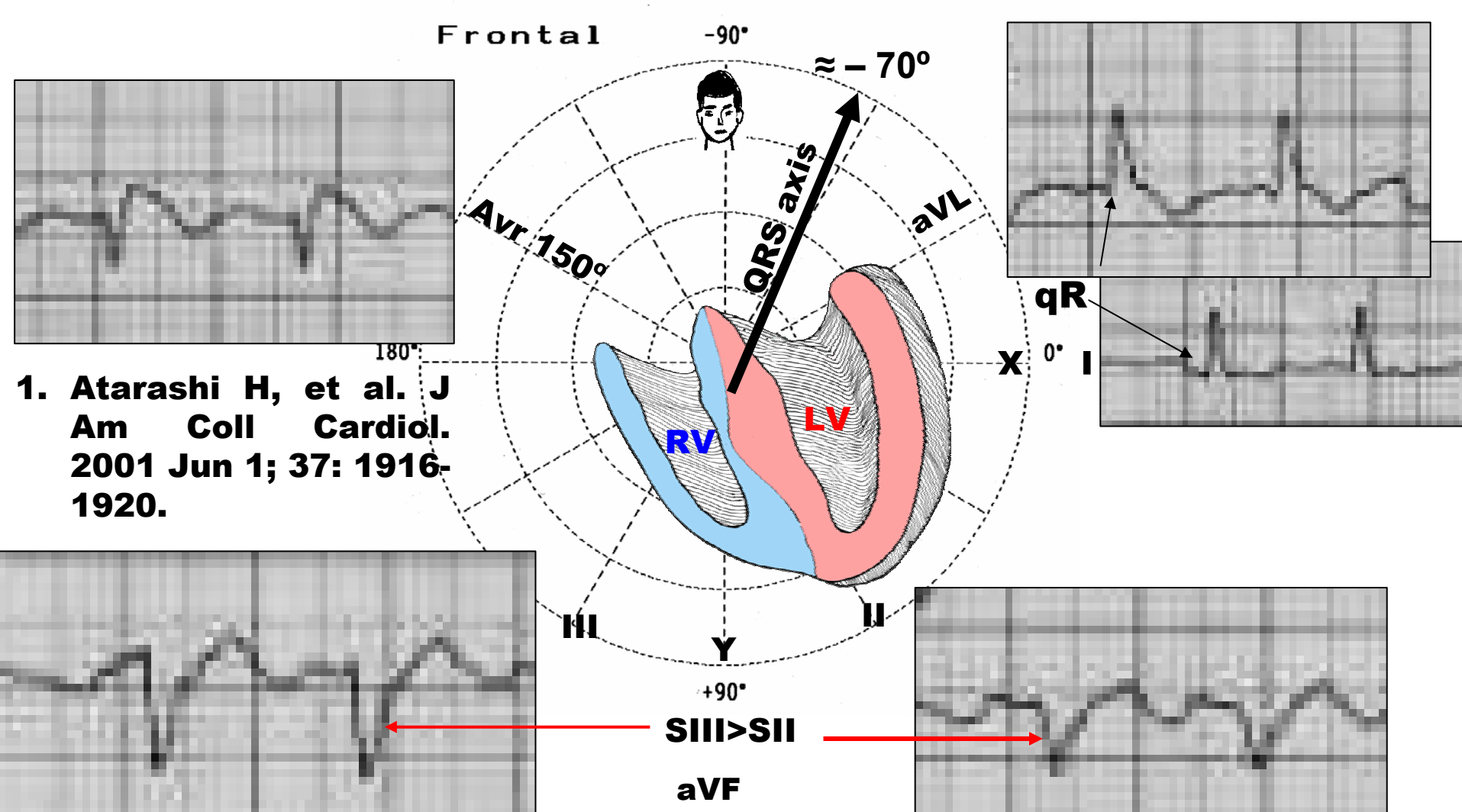
Rhythm: Type I, typical or classical atrial flutter. Negative polarity in II, III and aVF without baseline. (absence of isoelectrical “plateau” between F waves.). The F waves in inferior leads are composed of a negative component (the P´ wave) and a positive component (the Ta wave).

Variable AV conduction ratio HR 66/ 94bpm 2:1/4:1

Extreme left axis QRS deviation, QRS axis ≈ – 70º, qR pattern in I and aVL, SIII >SII and persistent S waves in V5-V6: Left Anterior Fascicular Block

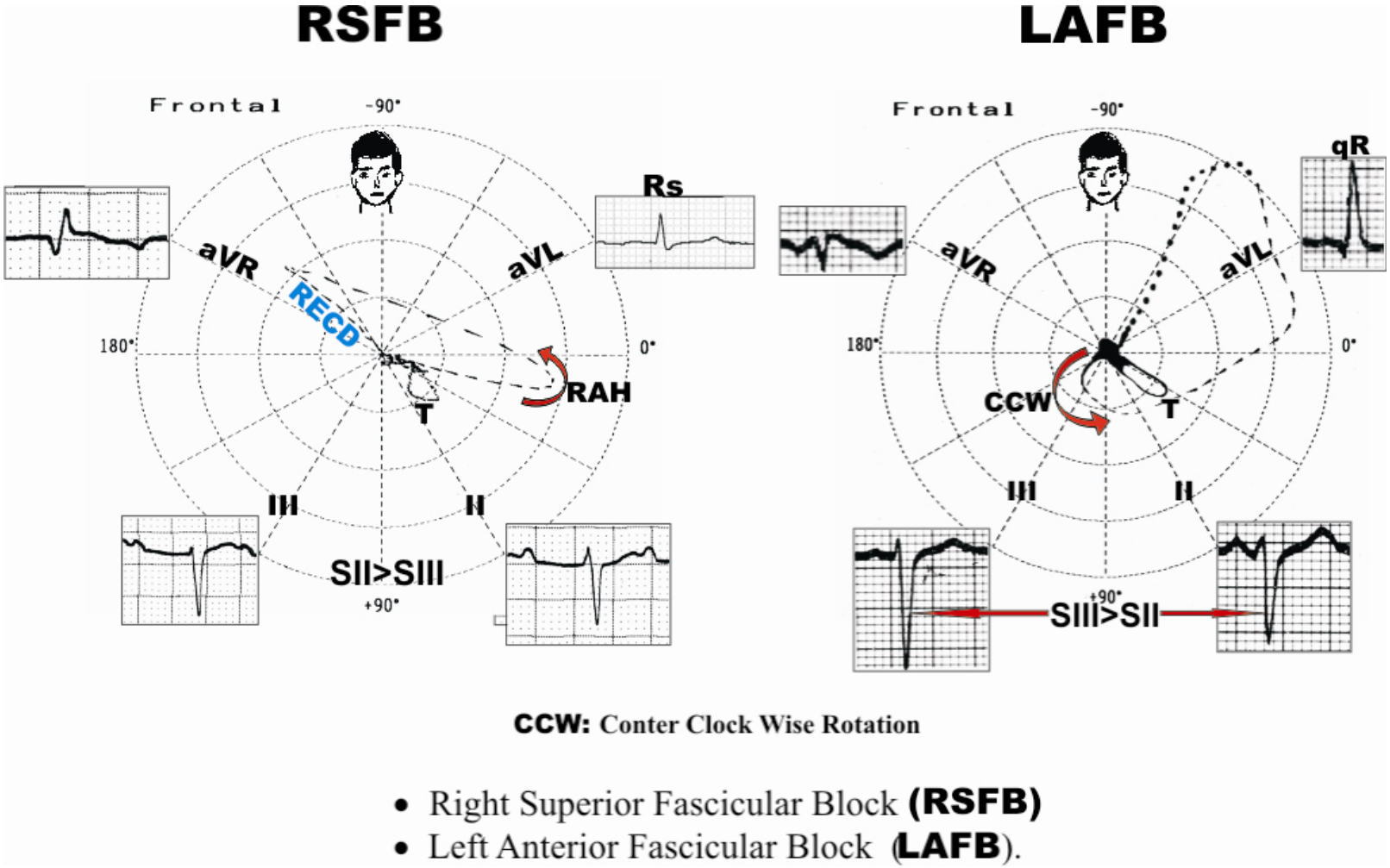
Type 1 ECG Brugada pattern in right precordial leads and unipolar aVR,

ECG on FP: qR in I and aVL, SIII>SII, extreme left axis deviation: Left Anterior Fascicular Block. In BrS, SÂQRS is normal in most cases (90.5%), but in ≈ 9.5% an extreme shift is observed in the left superior quadrant, according to a prospective study carried out in 105 workers from the city of Tokyo, from who 20 had VF, 18 syncope, and 67 were asymptomatic, included from 46 institutions in Japan. **(1)** The presence of extreme electric axis shift to the left (SÂQRS at the left from -30º) suggests the association with Left Anterior Fascicular Block (LAFB)



1. **Atarashi H, et al. J Am Coll Cardiol. 2001 Jun 1; 37: 1916-1920.**

however, a detailed analysis of QRS morphology in the frontal plane may occasionally reveal that this is Right Superior Fascicular Block (RSFB) **35**.The criteria used to differentiate both dromotropic disorders are(1): (see next slide)



CCW: Conter Clock Wise Rotation

- Right Superior Fascicular Block **(RSFB)**
- Left Anterior Fascicular Block (**LAFB**).

1. **Pérez Riera AR, Ferreira C, Schapachnik E. Value of 12 leads electrocardiogram and derived methodologies in the diagnosis of Brugada disease. In The Brugada Syndrome From Bench to Bedside. Editor Antzelevitch C. Blackwell Futura. 2005 Chapter 7, pp: 87-110.**

Initial 10 ms vector of QRS loop

1. LAFB: Heading downward and to the right
2. RSFB: Heading downward and to the left

QRS morphology in I & aVL

1. LAFB: qR pattern
2. RSFB: Rs

SII/SIII ratio

1. LAFB: SIII>SII
2. RSFB: SII>SIII

Location of end conduction delay (ECD)

1. LAFB: In the left superior quadrant when present
2. RSFB: In the right superior quadrant**(1)**.

Prominent R wave in aVR (R-wave ≥ 0.3 mV)

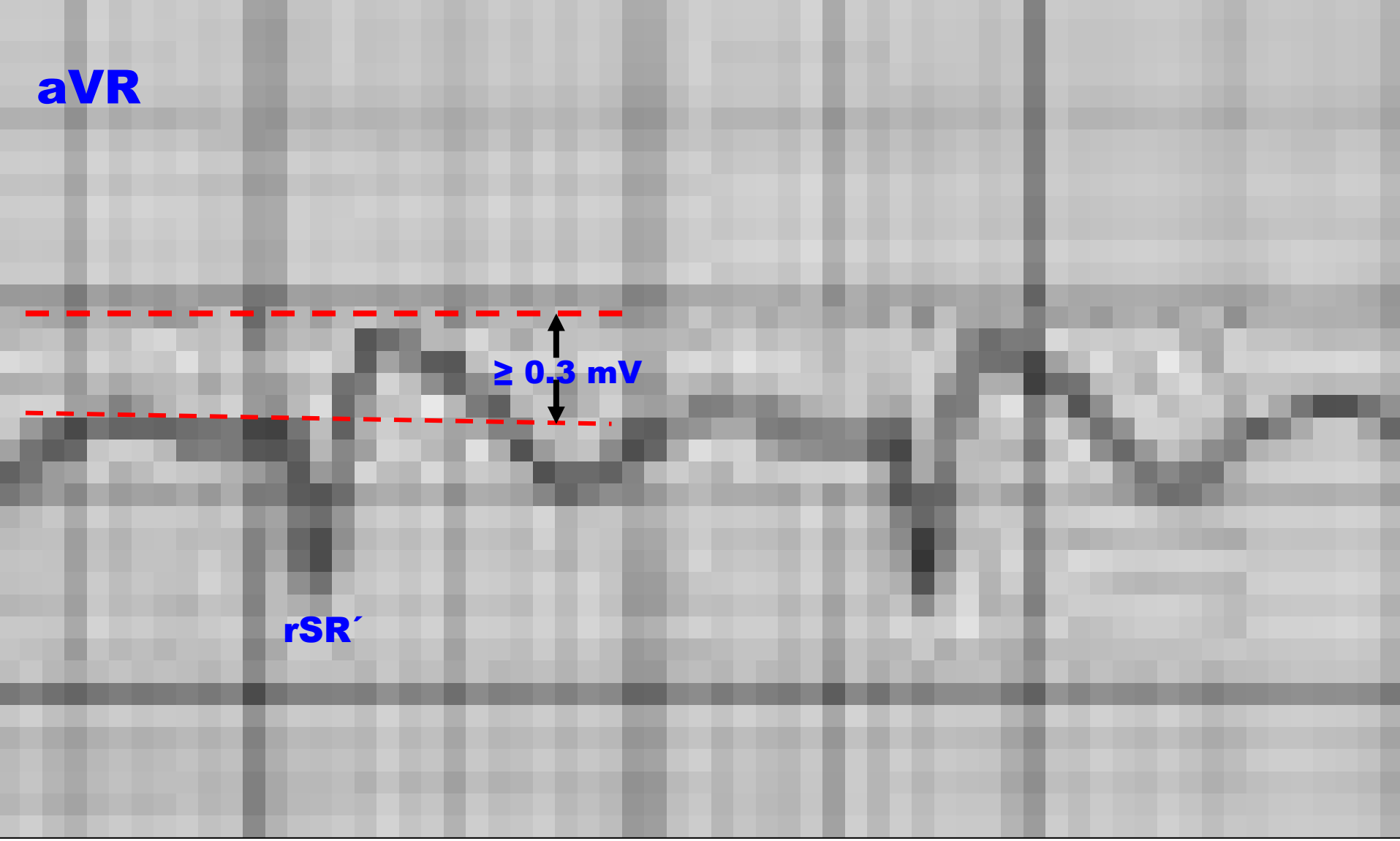
1. LAFB: Absent
2. RSFB: It could be present and it is called aVR sign**(2)**.

1. **Pastore CA, Moffa PJ, Spiritus MO, et al. Fascicular blocks of the right branch. Standardization of vector electrocardiographic findings. Arq Bras Cardiol 1983; 41: 161-166.**
2. **Babai Bigi MA, Aslani A, Shahrzad S. aVR sign as a risk factor for life-threatening arrhythmic events in patients with Brugada syndrome. Heart Rhythm 2007; 4: 1009-1012.**

Morphology of QRS loop of vectorcardiogram in the horizontal plane

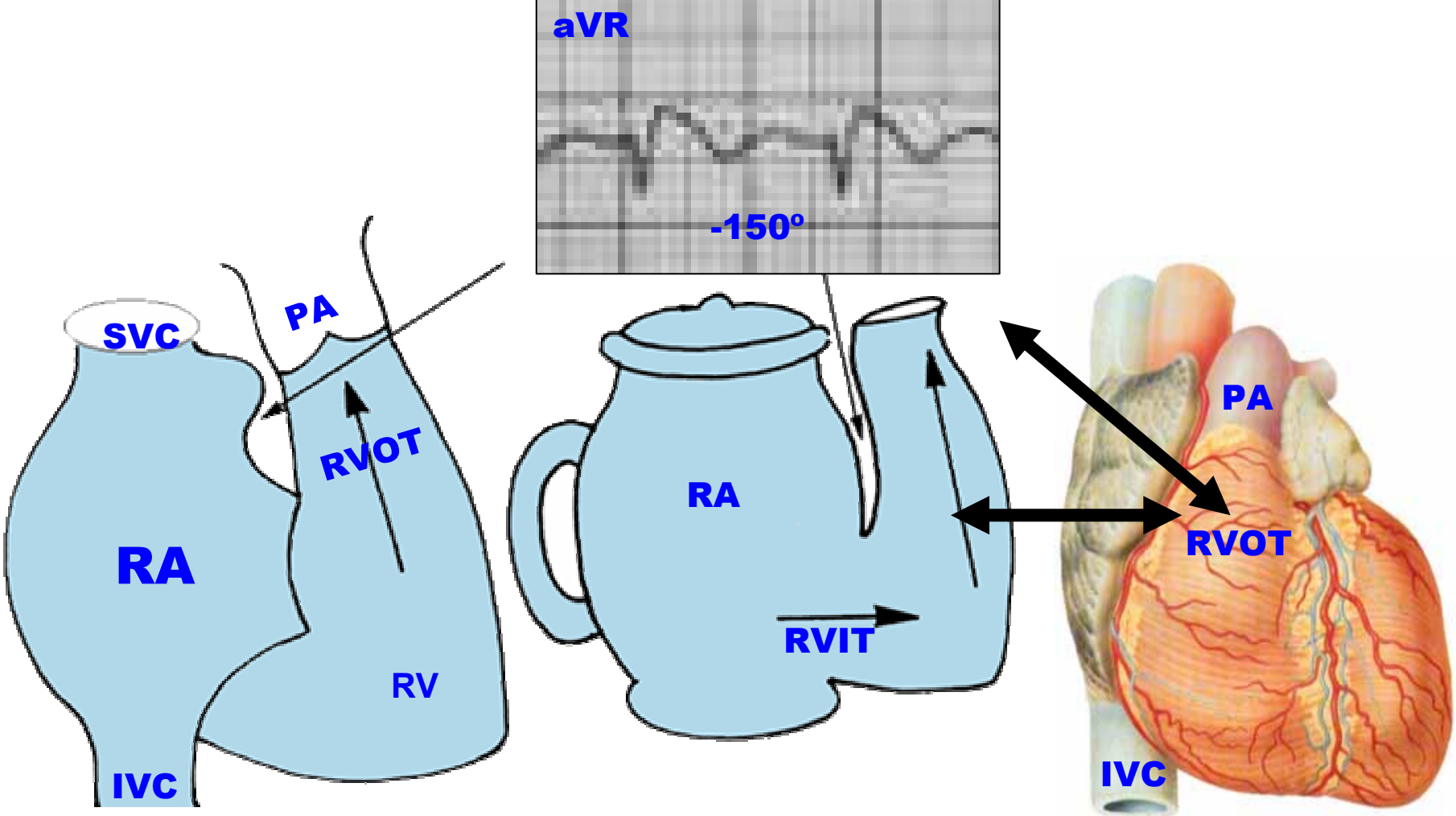
1. LAFB: Similar to normal.
2. RSFB: Similar to type-C right enlargement pattern: initial vector to the front and leftward, counter clockwise rotation and 20% or more of the area of the loop located in the right posterior quadrant in the horizontal plane.(**3)**.

3. **Luna Filho B, Bocanegra JA, Pfeferman A, et al. Fascicular block of the His bundle: critical approach for its identification. Arq Bras Cardiol 1989; 53: 261-265**.



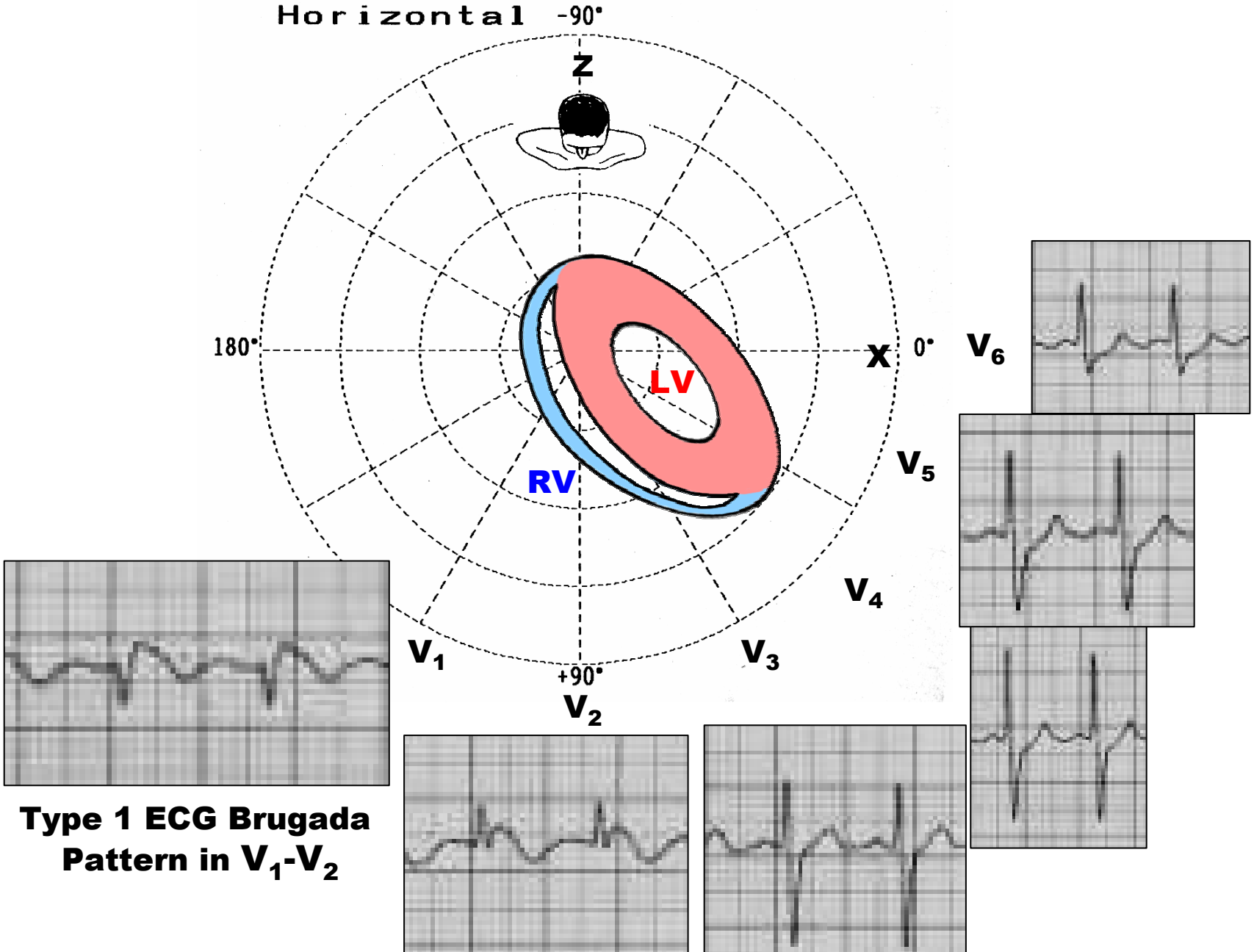
Presence of the so-called aVR sign: final R wave in aVR (R-wave ≥ 0.3 mV). This signal constitutes a risk factor for the appearance of arrhythmic events in patients with BrS.(1)

1. **Babai Bigi MA, et al. Heart Rhythm 2007; 4: 1009-1012.**

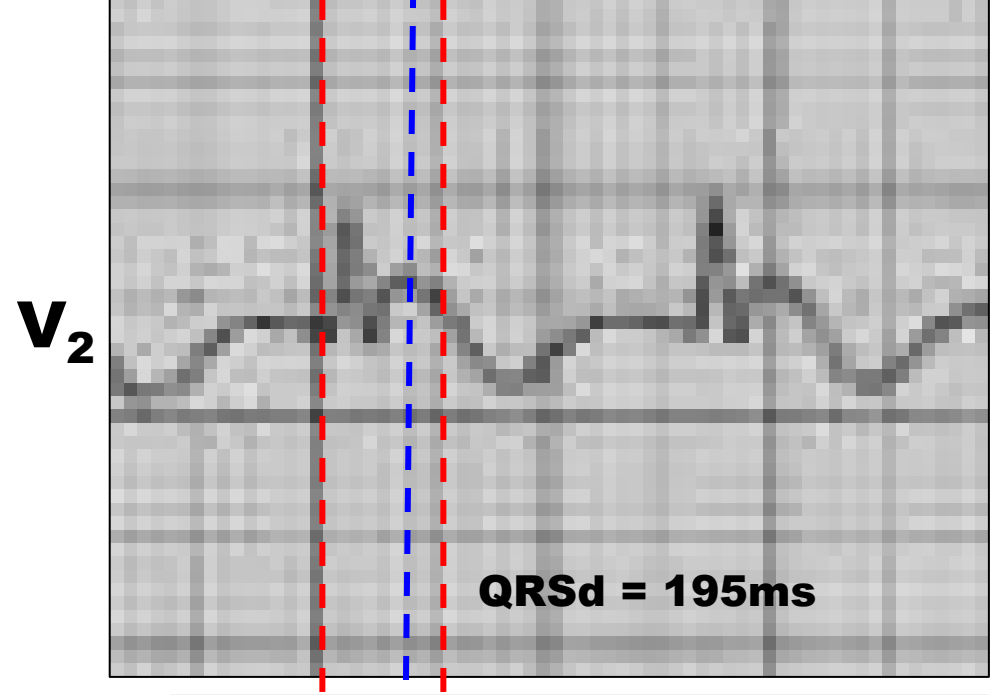


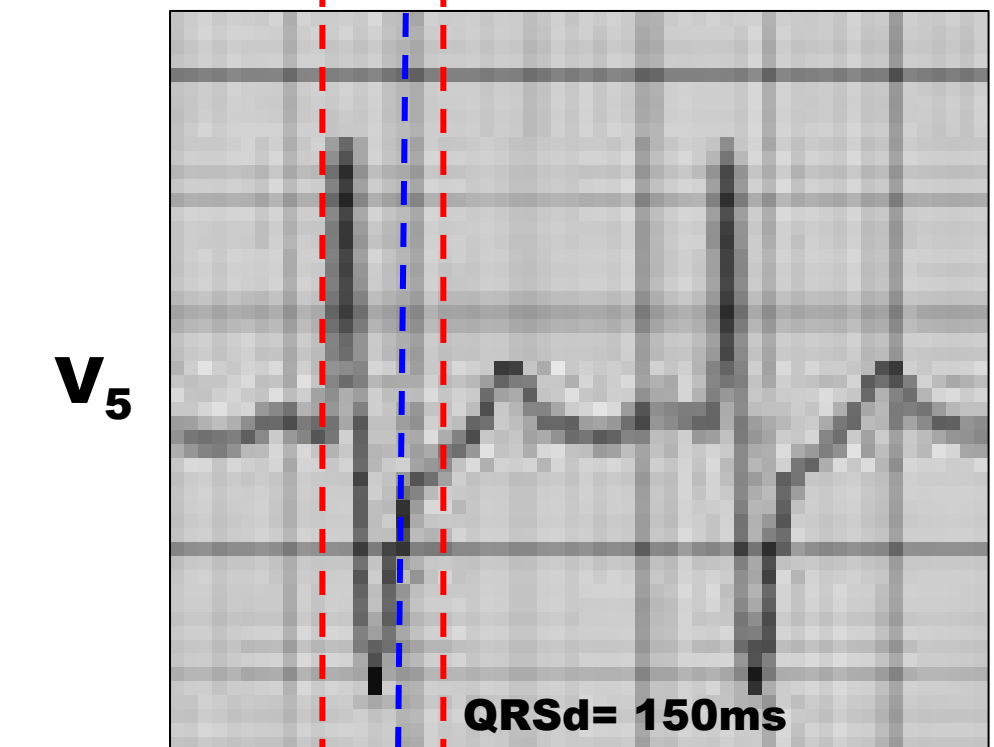
The BrS affect predominantly the right ventricle in the right ventricle outflow tract (RVOT) epicardium.(1) The larger part of clinical evidence supports the presence of right end conduction delay (RECD) as part of the process of BrS pathophysiology in the RVOT, as a consequence of structural abnormalities in the heart as part of BrS.(2). On the other hand, in the concealed forms of arrhythmogenic right ventricular cardiomypathy/ dysplasia (ARVC/D), the RECD pattern can also be observed showing type-1 ECG pattern27. This pattern was shown many years ago by Guy Fontaine et al.(3)

1. Doi A, et al. J Cardiovasc Electrophysiol. 2010 Jun 1;21: 688-696.
2. Coronel R, et al, Circulation 2005; 112: 2269-2277.
3. Hayashi H, et al. Circ J. 2010 Feb; 74: 271-277.



Type 1 ECG Brugada Pattern in V_1-V_2

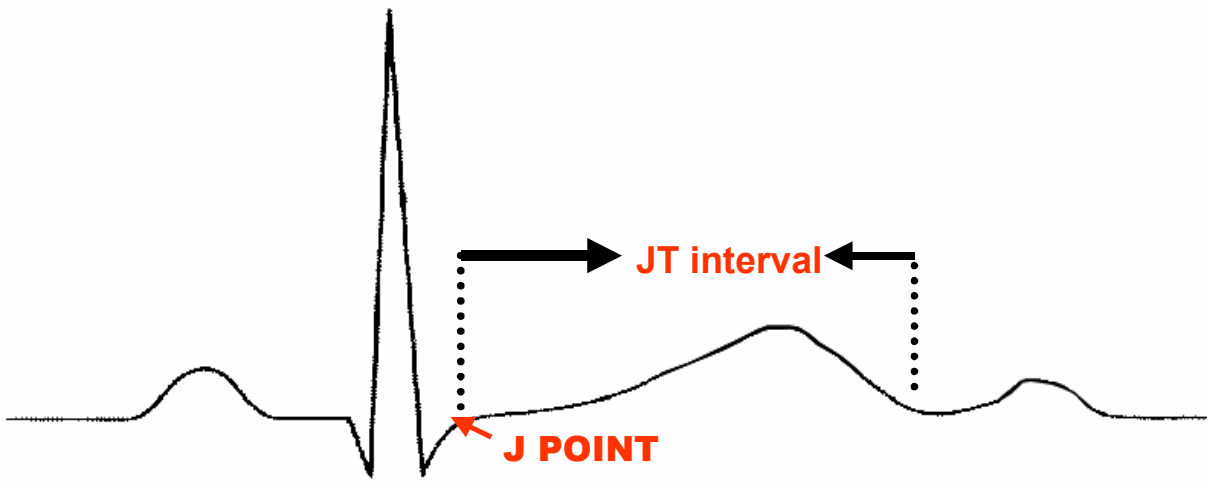




Pitzalis et al (**1)** identified selective prolongation of QT interval duration in the right precordial leads (V_1 to V_3) in comparison to the left ones (V4 to V6). As the QT interval is made up by ventricular depolarization (QRS) plus ventricular repolarization (ST/T) we think that this selective prolongation represents a certain degree of parietal block in the RVOT, as the one observed in ARVC/D. If the QT interval is prolonged only from V1 to V3, being normal or lesser from V4 to V6, it is clear that this increase may be due to prolongation of ventricular depolarization (QRS complex) and/or by ST/T prolongation (repolarization). If we admit that in BrS there is some degree of branch block, clearly the QT interval prolongation is due partly to this. The QTc interval constitutes the classical measurement for ventricular repolarization; however, this parameter includes ventricular depolarization (QRS), and therefore represents the so-called electric systole, which includes depolarization (QRS) and ventricular repolarization (ST/T = JT interval).

1. **Pitzalis MV, Anaclerio M, Iacoviello M, et al. QT-interval prolongation in right precordial leads: an additional electrocardiographic hallmark of Brugada syndrome. J Am Coll Cardiol. 2003 Nov 5; 42: 1632-1637.**

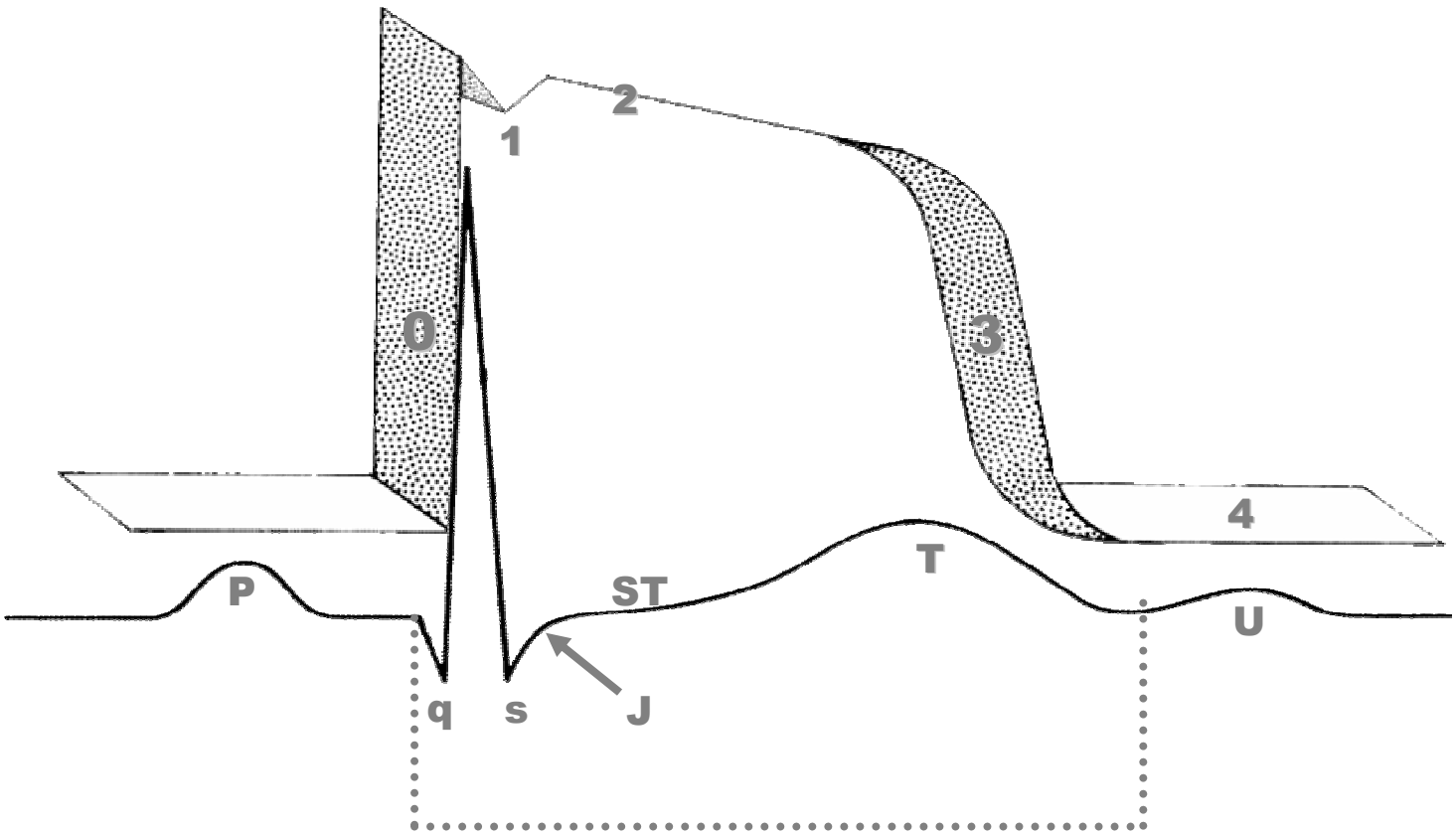
Thus, when there is branch block (as in the some cases of BrS), the measurement of ventricular repolarization through QTc may be incorrect. In these cases, the measurement of the JT interval (JT = QT - QRSD) is more accurate than the QT interval, because it excludes the depolarization that is found prolonged, because the biventricular chamber activates sequentially and not concomitantly as normally. ***This is the reason why it is essential to know accurately the exact point where depolarization ends and repolarization begins***. (We think that this is the mistake of Domenico Corrado manuscript!!!!: erroneous measurement)



JT and JTc INTERVALS: interval that extends from the J point to the end of the T wave.
The QTc interval constitutes the classical measurement of ventricular repolarization; however, the parameter includes ventricular depolarization. The measurement of JTc may be useful to identify LQTS cases with borderline values, where QTc interval could be normal in rest ECG. We find an example in patients carriers of tetralogy of Fallot who underwent surgery, and as a consequence of RV ventriculotomy, developed CRBBB. In these cases, JTc interval measurement is more sensitive than the QTc interval to detect prolonged repolarization.

1. Corrado D, et al. **Right bundle branch block, right precordial st-segment elevation, and sudden death in young people.**Circulation. 2001 Feb 6;103(5):710-7.

NORMAL QT INTERVAL

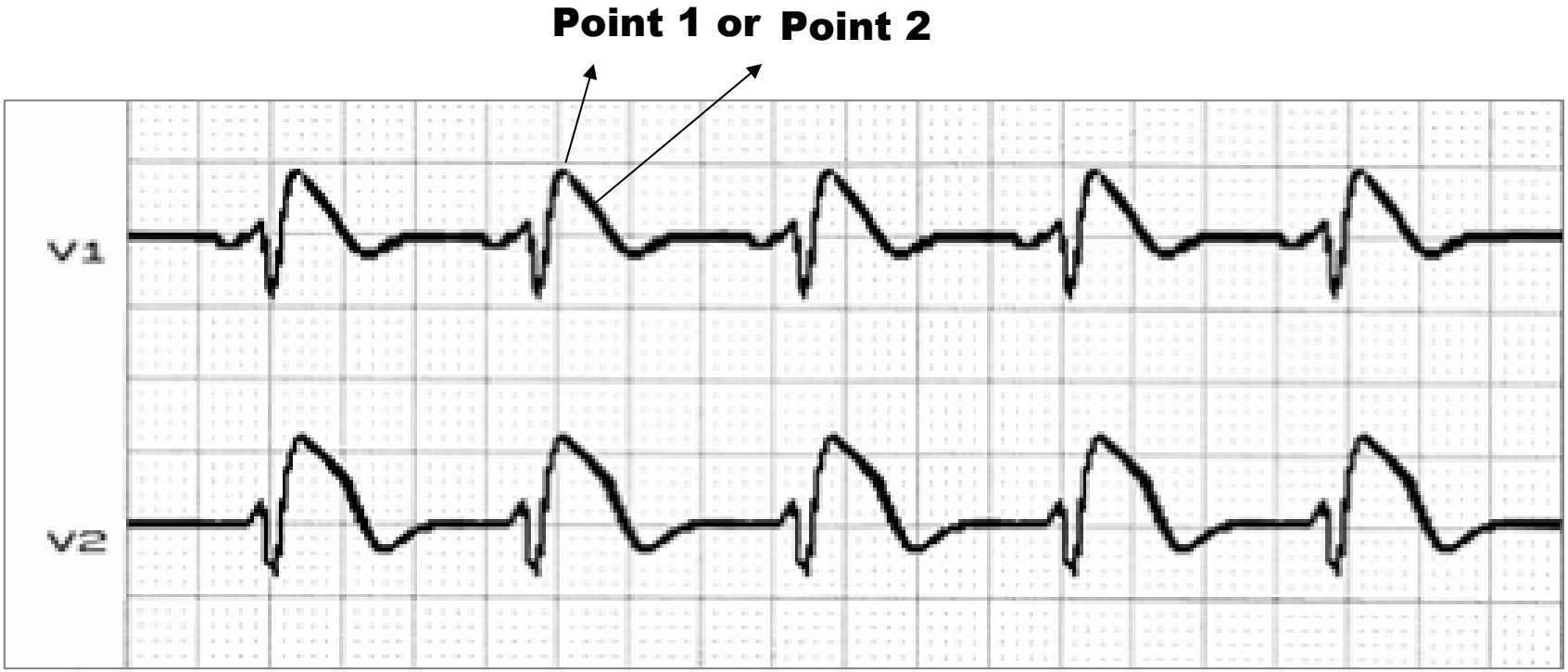


QT interval or electric systole

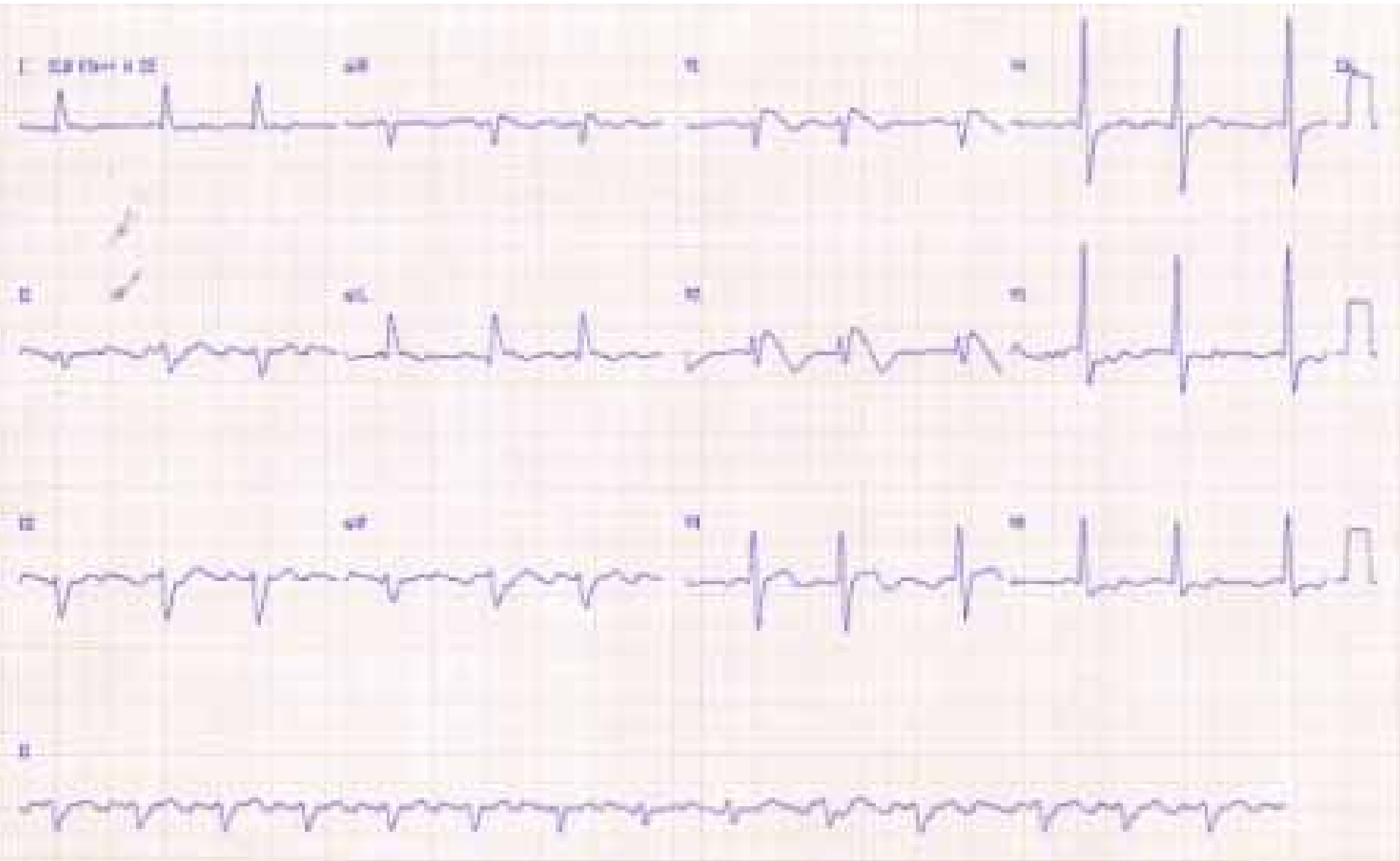
Normal value: 350 to 440 ms or 446 + - 15%
< 330 ms: short QT
> 450 ms: long QT

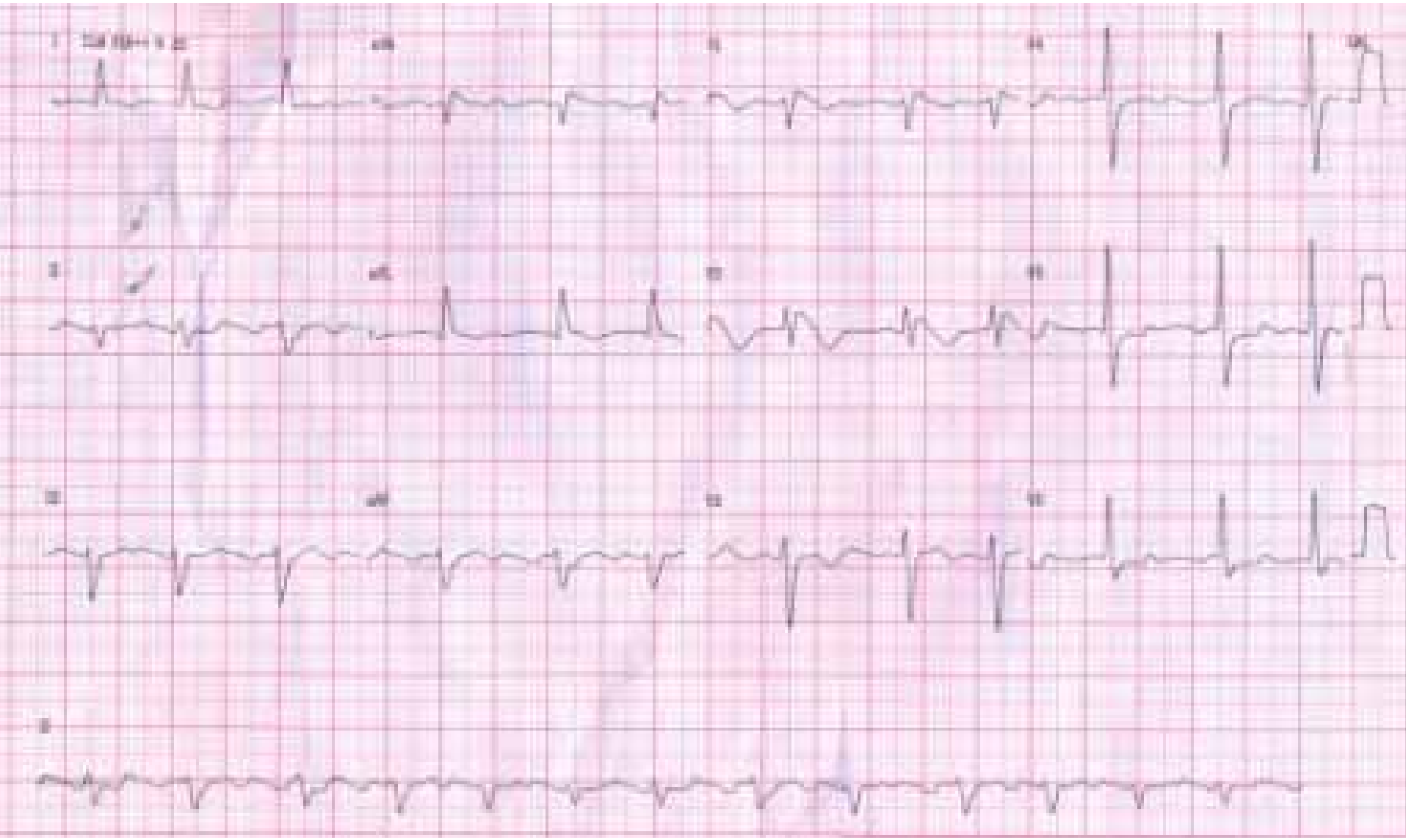
Minimal and maximal value of QT interval and its correlation with monophasic action potential.

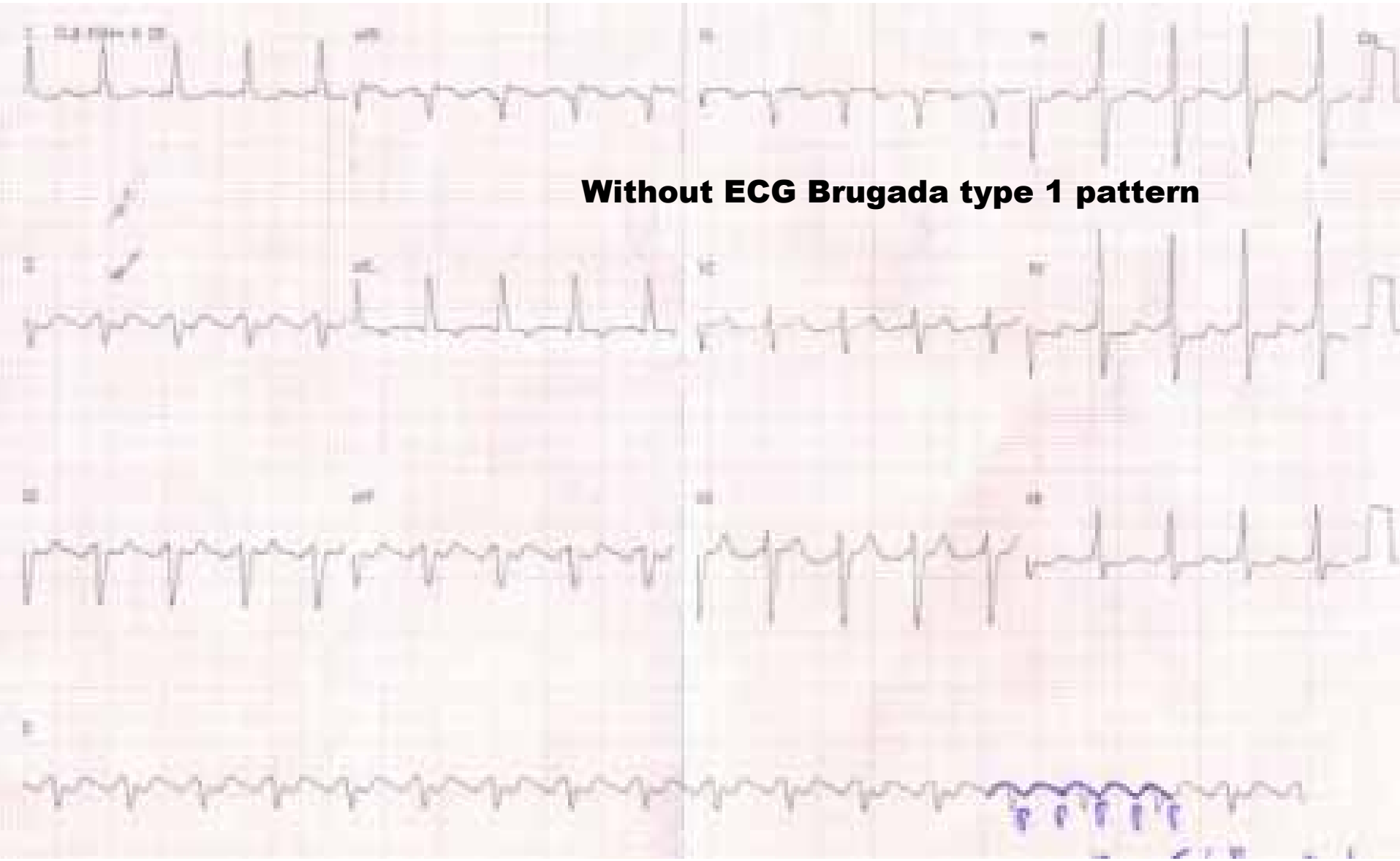
Where is the end of QRS complex?



Answer: point 2






Without ECG Brugada type 1 pattern

Colleagues opinions

Como siempre, hermoso caso el de Raimundo
Aleteo auricular tipo I itsmo dependiente.
Hipertensión arterial + sobrecarga biventricular? Andrés y el ecocardiograma nos dirá la verdad.
Etiologia: a determinar. Una posibilidad podría ser taquicardiomiopatia.
Si se encuentra descompensado, la cardioversión eléctrica se impone, excepto que pueda llevarse rapidamente a ablación del aleteo. Esta última pienso que se impone. Además, el procedimiento determinará si es una taquicardiomiopatía caso la función ventricular se recupere.
El ECG tiene el típico del patrón electrocardiográfico Brugada tipo 1. Aunque para el diagnóstico del síndrome es necesário un "corazón estructuralmente normal", el deterioro de la función ventricular poderia ser consecuencia de taquicardimiopatia.
De todas maneras, la fibrilación auricular es mucho mas frecuente que el aleteo auricular en el sindrome de Brugada.
Otro punto dudoso es la edad avanzada del paciente (70 años.) El sindrome de Brugada ocurre con mayor frecuencia al rededor de los 40 años. No obstante, existen casos descriptos en edades extremas como 80 años.
Yo trataria de descartar patologia estructural porque eventualmente poderiamos estar frente a una fenocopia es decir una causa adquirida que imita el patrón electrocardiográfico de la causa genética.
Queda por esclarecer el sincope. Si confirmamos que se trata de un sindrome de Brugada está indicado el implante del cardiodesfibrilador automático. Si continua con la fracción de eyección deprimida por miocardiopatia dilatada (no taquicardimiopatia), el cardiodesfibrilador es tambien la opcion correcta.
Veremos que opinan los maestros del foro.

Gran abrazo.
Oscar Pellizón.

As always, beautiful case of Raimundo´s case
Type I atrial flutter isthmus dependent.
Hypertension + biventricular overload? Andrés and the echocardiogram will tell the truth.
Etiology: to be determined. One possibility could be tachycardiomyopathy.
If the patient is descompesated(congestive heart failure) electrical cardioversion is the best option, except that it can quickly lead to flutter ablation. I think the latter prevails. In addition, the procedure will determine if a case tachycardiomyopathy ventricular function recovers.
The ECG shows the typical ECG Brugada type 1 pattern. Although for the diagnosis of the syndrome it is necessary "structurally normal heart," the deterioration of ventricular function could be result of tachycardiomyopathy.
Additionally, atrial fibrillation is more common than atrial flutter in Brugada syndrome.
Another doubtful point is the patient's advanced age (70 years). Brugada syndrome occurs most frequently around 40 years old of age. However there are cases described in 80 years.
I would try to rule out structural pathology because eventually we might be facing a phenocopy (an adquired condition that mimics ECG pattern of genetic form.)
Syncope episodes remains to be elucidated. If we confirm that this is a Brugada syndrome is indicated an implantable cardioverter-defibrillator (ICD). If the patient continue with depressed ejection fraction consequence of dilated cardiomyopathy (no tachycardimiopathy), the ICD is also the right choice.

Let´s wait teachers oppinions.
Oscar Pellizón MD from Argentine

FINALS COMMENTS

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

About this patient with structural heart disease, syncope episodes and Brugada type 1 ECG pattern, **First question:** I would like to know: Clinical characteristics of syncope events, i.e., circumstances of occurrence: at rest? Sleeping? During activity? (in BrS, 85% of syncopes occur during night rest) . **Raimundo answer: at rest during awake.**

It would be important to know if syncope events were with or without prodromes? What is the significance of this? Answer: patients carriers of BrS that present syncopes with prodromes, especially cloudy vision, suggests a benign cause of syncope.(1) Patients carriers of BrS may have benign neurally mediated syncope(2), in this case, we cannot consider the patient as being symptomatic, a decisive fact when making a decision (cardioverter defibrillator). Another relevant issue that is not mentioned in the description of the case is the presence or absence of sudden cardiac death in young relatives (≤45 years old) in the first degree. The positivity of this parameter would have a great diagnostic weight. **Raimundo answer: without SCD familial backgroud.**

Raimundo did not mention the ethnicity of the patient, a fact that could be relatively relevant in diagnostic suspicion, since 65% of patients carriers of the syndrome have Asian origins or are Asian. **Raimundo answer: mulatto.**

Does he have high resolution ECG? **Raimundo answer: No, he has not.**

Does he has a NMR? **Raimundo answer Raimundo answer: No he did not accomplish** for NMR

Does he have a “cate”? **Yes he has with a critical obstruction in distal portion of Left Anterior Descending Artery (LAD).**

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2. **Yokokawa M, et al. Neurally mediated syncope as a cause of syncope in patients with Brugada electrocardiogram. J Cardiovasc Electrophysiol. 2010 Feb;21:186-192.**

Conclusion this patient has a Brugada phenocopy. Numerous conditions should be ruled out, which could resemble Brugada type-1 ECG pattern, called "acquired forms of BrS"**(1)**, "Brugada-like electrocardiographic pattern" **(2)**, or the way our group calls them, Brugada phenocopyes (**3;4**) (an environmental condition that imitates or copies one produced by a gene).

BRUGADA PHENOCOPIES RELATED WITH CORONARY ARTERY DISEASE

1. **Acute myocardial ischemia:** ST segment elevation is a manifestation of acute subepicardial or transmural ischemia that occurs in the leads located in front of the lesion. The Ito channel may modulate the manifestation of ECG in acute ischemia, in the same way that in true BrS. Both have similar electrophysiologic substrates**.(5)**.
2. **Atherosclerotic obstruction of the conus artery (branch of the right coronary artery):** this branch irrigates the RVOT and its obstruction may cause type-1 ECG pattern(**6**)
3. **Right ventricular infarction** that involves the RVOT **(7)**
4. **Vasospastic angina, Prinzmetal variant angina.(8)**
5. **Coronary anomaly.(9),**
6. **Type-A dissecting aortic aneurysm**
7. **Antero-septal ventricular aneurysm**
8. **"Primary percutaneous coronary intervention" (10)**. Surawicz et al**11; 12** show an example of "focal" right bundle branch block with selective increase of QRS duration in V1 and V2 when compared to V5**.**

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9 Sharma AK, et al. J Card Surg. 2010 Sep; 25: 614-617.
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12. Surawicz B, et al. J Am Coll Cardiol.1997 Aug; 30: 452-458.

DIFFERENTIAL DIAGNOSIS BETWEEN VARIANT ANGINA OR PRIZMETAL ANGINA AND BRUGADA SYNDROME

Precordial pain:

1. BrS: No.
2. Prizmetal angina: Yes.

Tendency to VT/VF:

1. BrS: High.
2. Prizmetal angina: High.

Structural heart disease:

1. BrS: Apparent absent.
2. Prinzmetal angina: Could exist.

Response to nitrates and nitroglycerine:

1. BrS: Null.
2. Prinzmetal angina: Improves or suppresses clinical/electrocardiographic manifestations.

Permanence of ST segment elevation:

1. BrS: Fluctuating and without pain. Eventually concealed
2. Prizmetal angina: Brief, transitory and accompanied by pain.

Cause:

1. BrS: Genetic alteration of Na^+ channel, and others channels. Sporadic cases are frequents.
2. Prinzmetal angina: Possible alteration in production nitrous oxide in vascular wall.

Presence of mirror image or reciprocal in ECG:

1. BrS: Could be present.
2. Prinzmetal angina: Present.

Topography of ST elevation:

1. BrS: Right precordial leads of V1 to V3; it could rarely be observed in inferiorapicallateral wall and triggered by fever and nocturnal vagotony.
2. Prizmetal: Variable. It could alternate between precordial leads and inferior ones. It could be triggered by hyperventilation.

Dromotropic disorders:

1. BrS: AV block of the first degree by extension of H-V in 50% of cases and in carriers of the mutation. LAFB in near 10% of cases. Fascicular superior right bundle branch block
2. Prinzmetal angina: It could happen in a transitory way until a high degree of AV block during the episode; it is associated with a higher risk of arrhythmia and SCD.

T wave inversion:

1. **BrS:** Negative T wave in precordial leads from V1 to V3, characteristic of type 1.
2. Prinzmetal angina: Inverted and deep T waves from V1 to V4 associated to anterior hypokinesia, suggesting myocardial “stunning” that indicates critical lesion of the anterior descending artery: “LAD-T wave pattern”.

Presence of transitory Q wave:

1. BrS: No.
2. Prinzmetal Angina: It could happen.

Effort test:
BrS: It could normalized the variation during effort.
Prizmetal angina: Variable response.

Myocardial scintigraphy with thallium 201:
BrS: Normal.
Prinzmetal angina: Transitory transmural hypo -uptake.

Response to test of maleate of ergonovine in doses of 0.05 to 0.40 mg (stimulant of alpha adrenergic and seratoninergic receptor)
BrS: There could be a mild diffuse reduction of caliber without spasm when doses are equal to or less than 0.40 mg are used.
Prinzmetal angina: Intense coronary spasm accompanied by pain and ST elevation. Possible cardiac block, asystole and VT.

Response to hyperventilation:
BrS: It does not modify.
Prizmetal angina: Severe spasm and reproduction of clinical electrocardiographic manifestations.

Response to intracoronary acetylcholine, each dose given in a time above one minute, in doses of 10, 25, 50 and 100μg doses separated by five minute intervals:
BrS: It could worse the ST elevation with paradoxical dilation of coronary vessels.
Prinzmetal angina: Severe spasm and reproduction of clinical electrocardiographic manifestations.

Response to magnesium sulfate:
BrS: Not mentioned.
Prinzmetal angina: Suppresses attacks induced by hyperventilation and exercise.

Treatment:
BrS: In symptomatic sutomatic implantable cardioverter defibrillator in association with quinidine, a drug that contributes to diminish the number of shocks. Isoproterenol indicated in electric storm associated with general anesthesia and cardiopulmonary “bypass”
Prinzmetal angina: Calcium antagonists, such as nifedipine, diltiazem, verapamil, and felodipine associated to nitrates. Benefit with prazosin is mentioned.

THE EXTENSIVE LIST OF POSSIBLE BrS PHENOCOPIES.

1. **Early repolarization pattern.(1)**
2. **Athlete's heart(2)**
3. **Vagal tone increase:** during rest and night sleep and early in the morning between 22:00 and 7:00**(3).**
4. **Ingestion of food (4).** The variations of ST segment elevation are frequently associated with meals. ST elevation worsening is observed at night after dinner, being lower in the term between midnight and dawn. This information could help to predict the time of highly risky events of arrhythmias dangerous for life in BrS.
5. **Postprandial variations and in partial gastrectomy (5)**
6. **Reduction of concentration of norepinephrine and AMPc in the heart (adrenergic dysfunction(6)** what would cause a lower beta-adrenergic stimulation and consequently, cardiac tissue decrease of AMPc with possible consequences in arrhythmogenesis by vagal predominance leading to type-1 pattern**.**
7. **Pectus excavatum(7)**.
8. **Left ventricular enlargement(8)**.
9. **Right bundle branch block(9)**
10. **Acute pericarditis (10)**

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11. Acute pulmonary embolism(1)
12. Hypercalcemia(2)
13. Hyperkalemia(3)
14. Hypokalemia: it has been observed in the north-eastern region of Thailand, that the deficit in potassium with hypokalemia, familial hypopotassemic periodical paralysis, distal renal tubular acidosis and kidney stones are endemic and factors contributing to sudden unexpected death syndrome**(4)**. Hypokalemia accentuates Brugada pattern, or makes it appear, besides causing late potentials in SAECG**(5)**.
15. Severe hyponatremia(6).
16. Adrenal insufficiency(7).
17. Familial thyrotoxic periodic paralysis. (8).
18. Glucose-induced hyperinsulinemia: it may trigger type-1 ECG pattern in BrS patients**(9); (10)**. Insulin activates the Na+/K+ pump, stimulating calcium inflow through the slow calcium channel, increasing ST segment elevation.

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19. Hypothermia(1)
20. Hyperthermia(2).
21. Variant 3 of Long QT syndrome and mixed forms.(3)
22. Central nervous system diseases. E.g. subarachnoid hemorrhage.(4)
23. Arrhythmogenic right ventricular cardiomypathy/ dysplasia 5; 6; 7.
24. Chronic chagasic cardiomyopathy(7)
25. Myocarditis(8)
26. Cardiac tumor of the interventricular septum(9).
27. After esophagus cancer reconstructive surgery(10).

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Which is the appropriate approach in this case?

1) Acute treatment: First electrical CD cardioversion for atrial flutter
2) Flutter ablation is the efective option for long term.
3) Angioplaty and stenting. Stent placement in LAD. Percutaneous coronary interventions using stenting techniques can be accomplished with minimal procedural risk, early facilitation of ambulatory discharge, and in the current era of drug-eluting stents, a lower target vessel failure rate.
4) Concomitant medications to treat coronary artery disease and heart failure:
   Calcium channel blockers and/or beta-blockers(carvedilol)
   Angiotensin-converting enzyme (ACE) inhibitors,
   Spironolactone
   Cholesterol lowering medications, such as statins, are useful to decrease the amount of "bad" (LDL) cholesterol
   Nitroglycerin
   Aspirin. Finally, in addition to aspirin, the patient must take an anti-clotting or antiplatelet drug, such as **clopidogrel, prasugrel or ticlopidine** (brand names Plavix, Effient or Ticlid) for a year or more after the stenting, to prevent the blood from reacting to the new device by thickening and clogging up the newly expanded artery (thrombosis). Ideally a smooth, thin layer of endothelial cells (the inner lining of the blood vessel) grows over the stent during this period and the device is incorporated into the artery, reducing the tendency for clotting.
5) If If the patient continue with depressed ejection fraction ≤ 35% CDI is indicate.