Dear Andres:

Thank you for sending me this interesting case. I offer some additional interpretation for the QRS pattern – the alteration of impulse propagation in the ventricles due to alterations of the working myocardium. I found it questionable that the aging/ degenerative processes would affect selectively just the posterior fascicle and the right bundle, and not the working myocardium. As we have shown in our simulation studies, local as well as diffuse alteration of impulse propagation in ventricles can result in QRS patterns consistent with the patterns of a fascicular block or a bundle branch block.

This interpretation could contribute to understanding the relation between the Interatrial block and the altered impulse propagation in the ventricles, not limited to the fascicular and bundle branch blocks.

My very best regards,

Ljuba

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841 04 Bratislava
Slovak Republic
phone: +421 905 401 634
bacharova@ilc.sk

Dear Professor Ljuba Bacharova

Thanks very much for your interest in our work, You commented that *"As we have shown in our simulation studies, local as well as diffuse alteration of impulse propagation in ventricles can result in QRS patterns consistent with the patterns of a fascicular block or a bundle branch block and not the working myocardium"*.

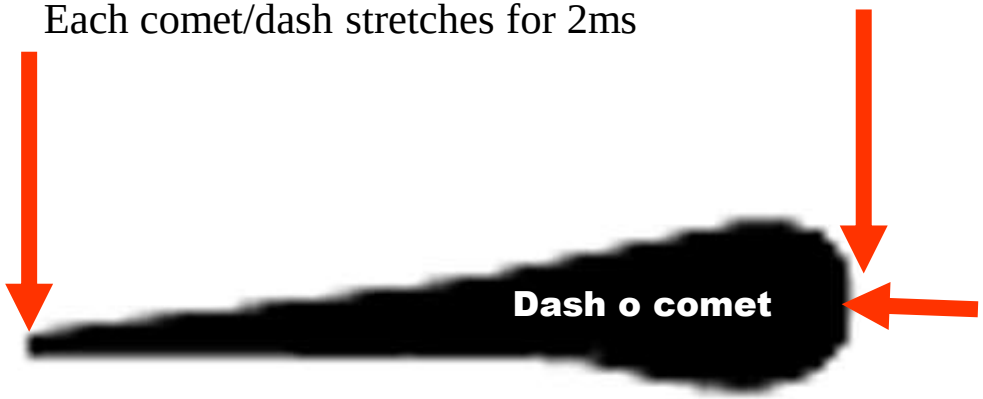
The conduction velocity across the working myocardium should show in the QRS loop of the vectorcardiogram (VCG) diffuse slow conduction(conduction delay) in the totality of the QRS loop. Consequently, the dashes or comets are very close to each other throughout the entire ventricular depolarization because the conduction speed within the ordinary myocardium or working myocardium is much lower and should present comets closer to each other.

Meaning according to the location of the conduction delay in the QRS loop:

- **Initial conduction delay =** *Ventricular preexcitation, WPW syndrome/ delta wave.*
- **Middle and End conduction delay =** *Complete Left Bundle Branch Block.*
- **End conduction delay =** *Complete or incomplete Right Bundle Branch Block.*
- **End conduction delay in the right precordial leads and aVR** = Superior Right fascicular block in the RVOT area.. Typical of Brugada syndrome.
- **Uniform conduction delay (***Non-specific intraventricular conduction delay)* **=** *Hypercalemia; quinidine effect; intra-infarction, etc.*

Manifest Instantaneous Vector

Each dash or comet represents a time of 2 ms or 2.5 ms, depending on the calibration of the device.



Determination of Conduction Velocity of Stimulus

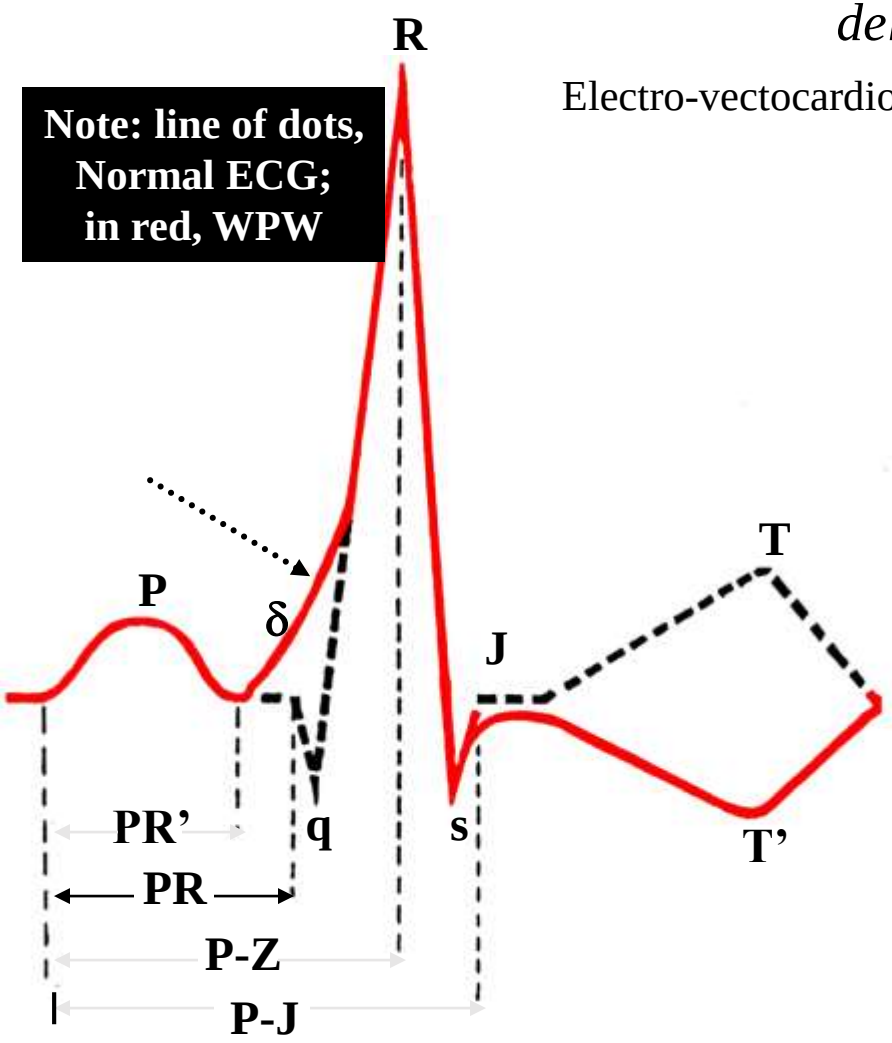
The greater or the lesser distance between dashes or comets indicates the greater or the lesser conduction velocity in the tissue. Thus, when they are very close to each other, it indicates the presence of conduction delay. To consider the phenomenon as true, it is necessary for it to be evident in at least 2 planes.

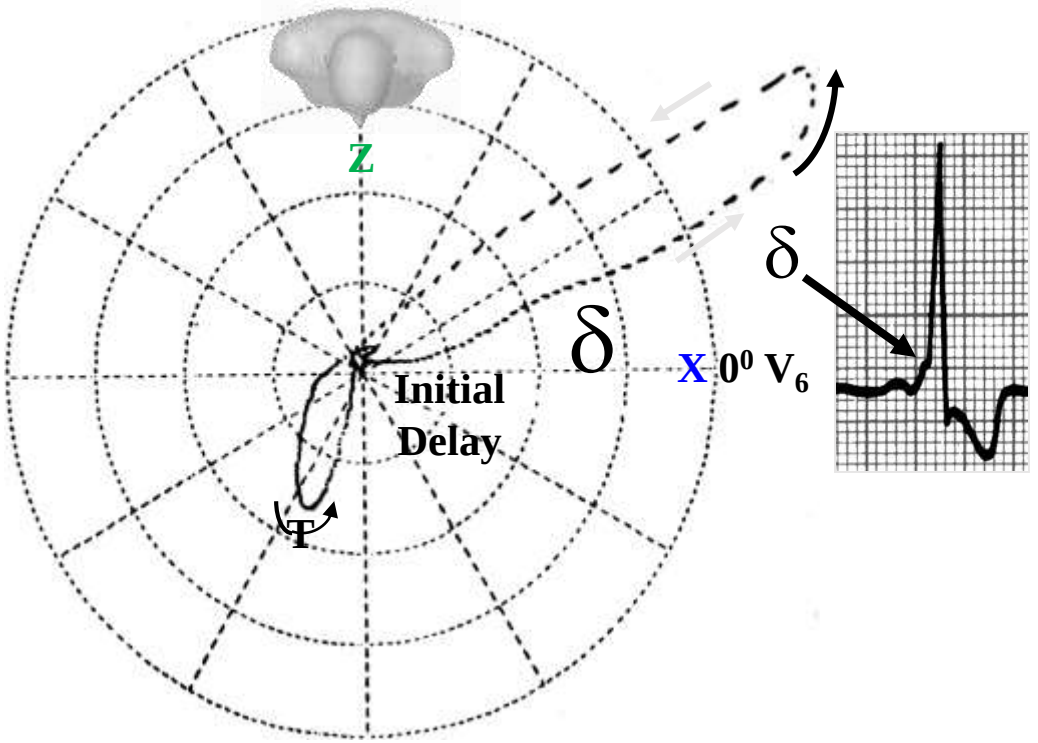
SEPARATE DASHES = MORE DROMOTROPISM

VERY CLOSE DASHES = LESS DROMOTROPISM

WPW ECG/VCG correlation: Initial conduction delay on QRS loop = *Ventricular preexcitation, WPW syndrome/ delta wave.*

Electro-vectocardiographic criteria for WPW type preexcitation.



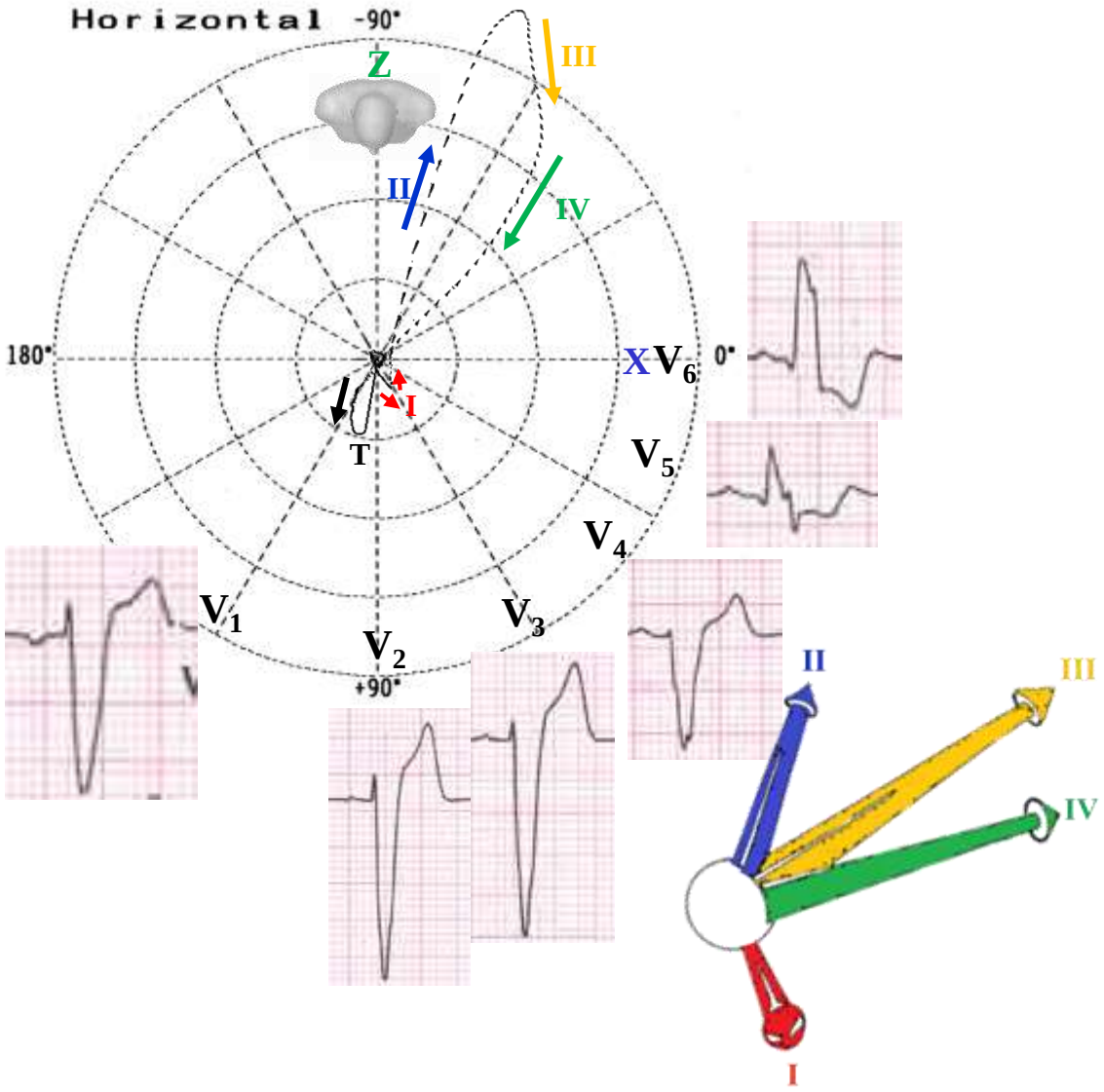


- **PRi or PQ:** since the onset of P up to the onset of QRS. It represents the time the stimulus takes to go from the SA node until reaching the ventricles: 120 ms to 200 ms.
- **PZ:** distance between P wave onset until R apex: 150 to 230 ms.
- **PJ:** distance between P wave onset until j point: 180 to 260 ms.

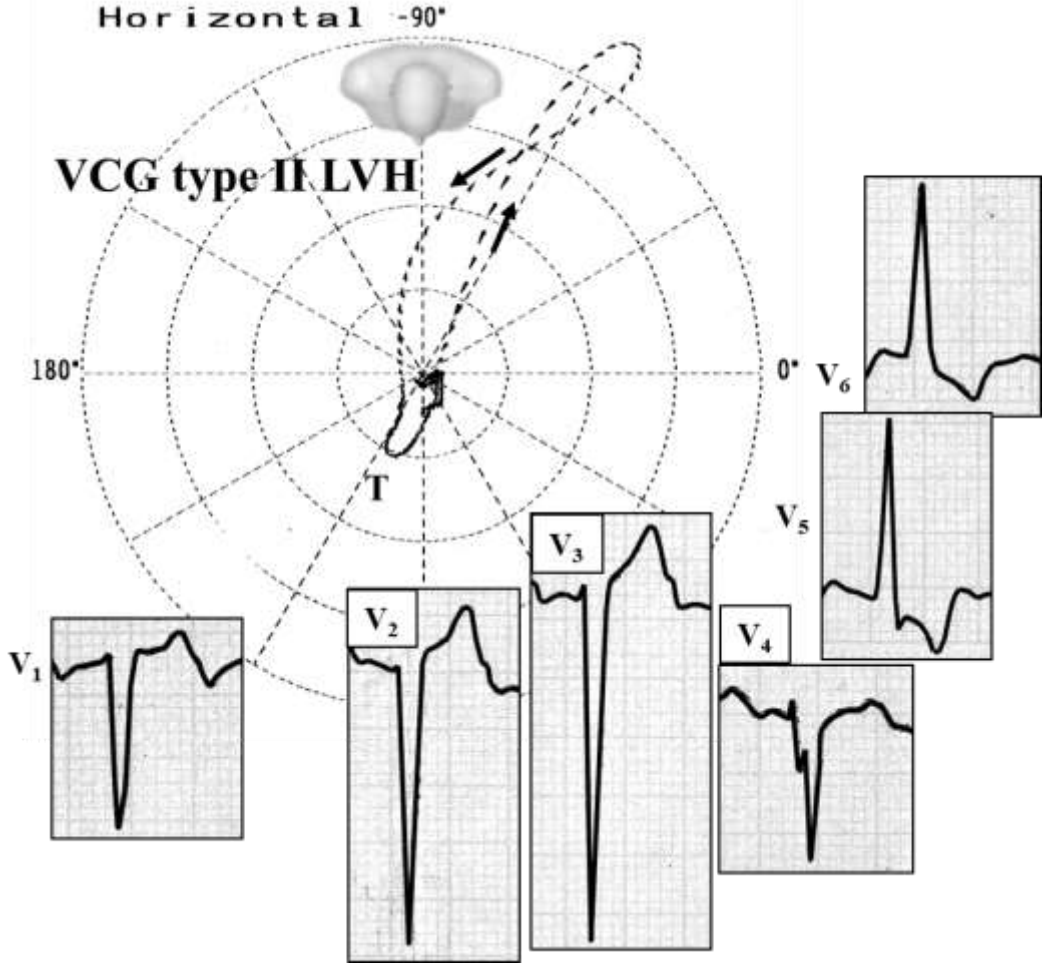
- Initial delay of QRS loop: delta wave.
- T-loop opposite to QRS loop

Middle + End conduction delay =

Truly *Complete Left Bundle Branch Block.*

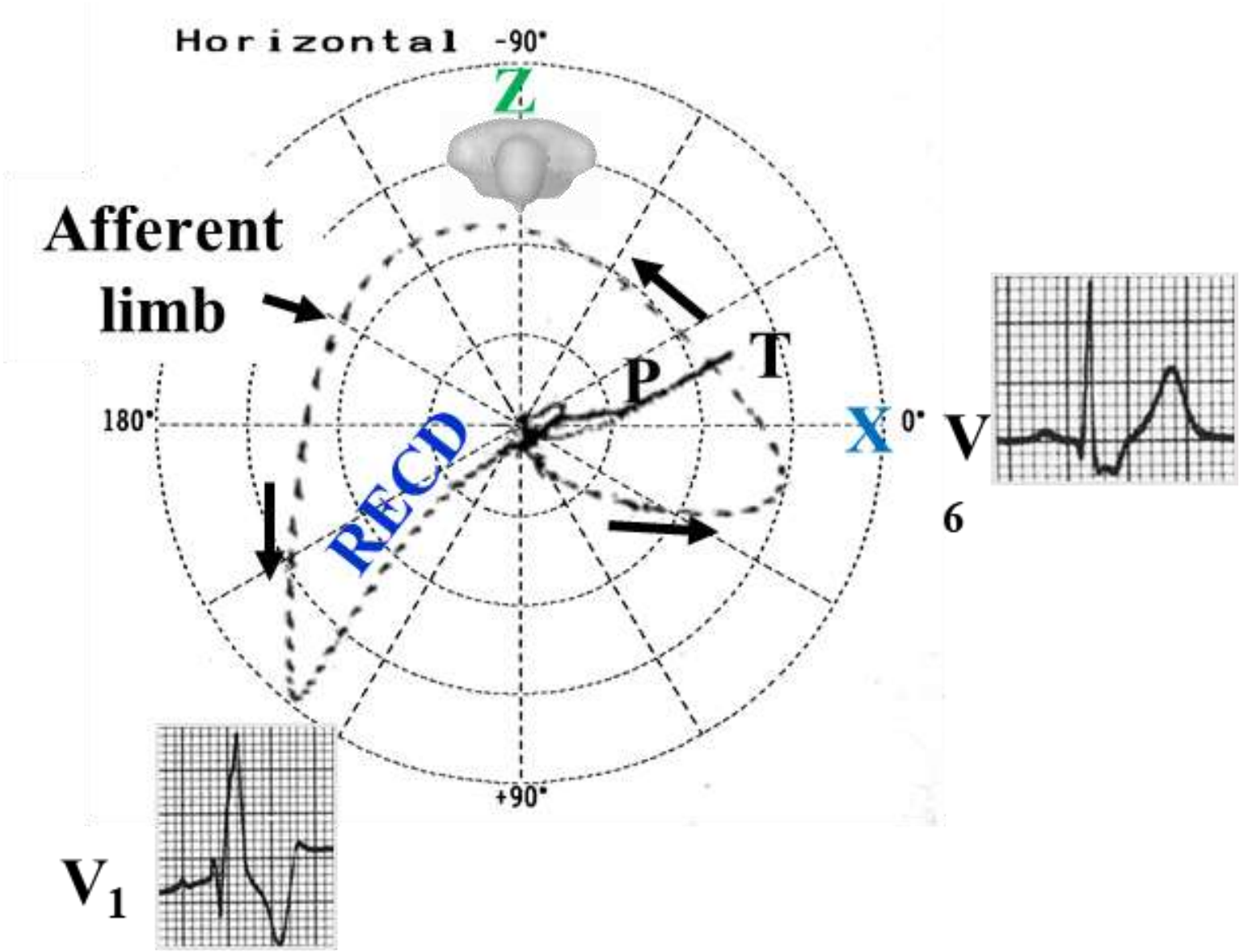


LVH mimicking LBBB: pseudo LBBB



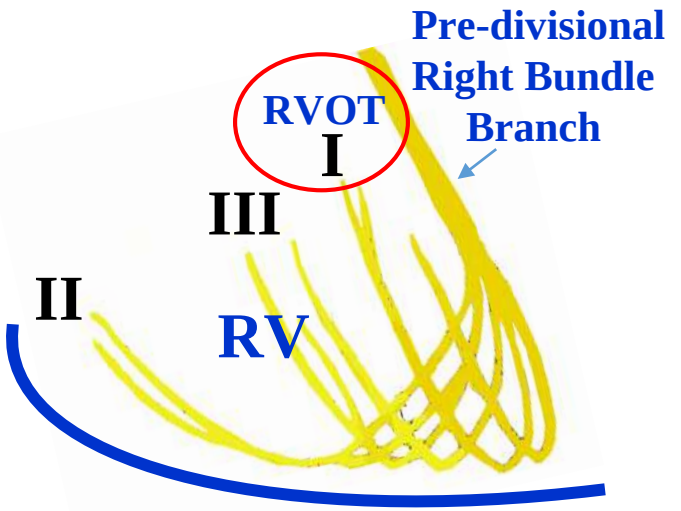
Type II LVH: Very similar to QRS loop of Complete LBBB: Vector of initial 20 ms heading to the front and the left (rarely to the right), rotation in eight with counterclockwise proximal portion and clockwise distal portion, and E point not matching 0 point and located to the front and the right of this. T loop to the front and the right, opposite to QRS loop. The maximal left vector >2 mV. **Note**: it differentiates from Complete LBBB by absence of middle-final delay (non responder).

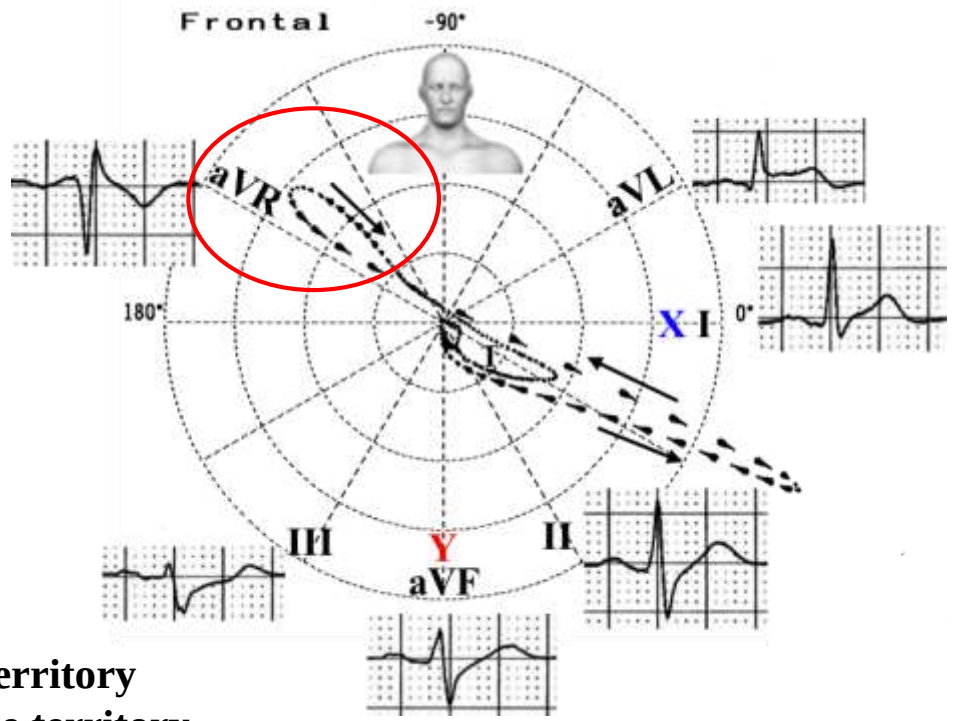
Terminal or right end conduction delay on QRS loop = *Complete or incomplete Right Bundle Branch Block.*

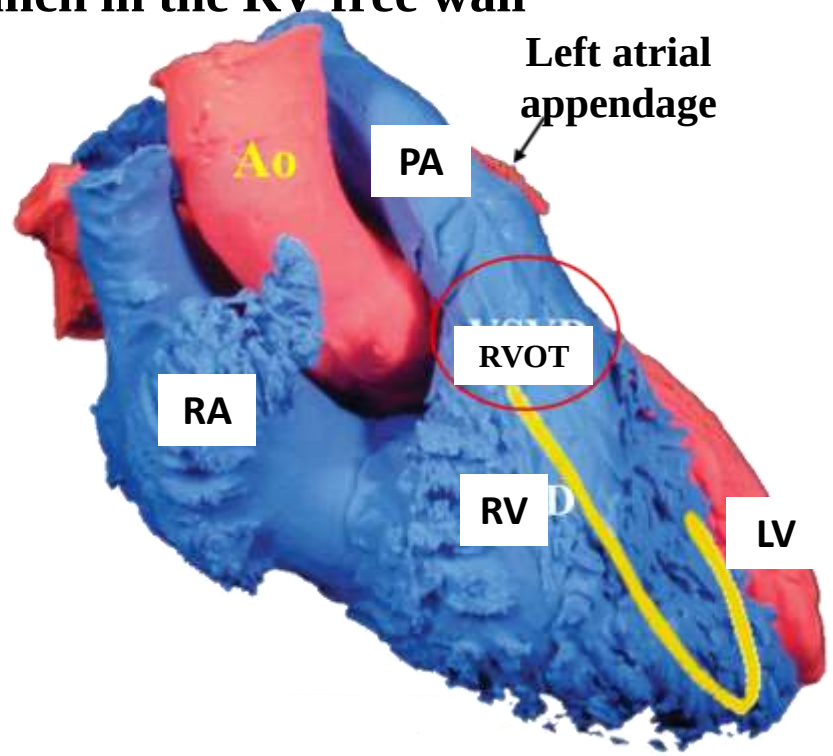


End conduction delay on QRS loop in the right precordial leads and aVR – Superior Right fascicular block in the RVOT area. Typical of Brugada syndrome

Distribution of the three fascicles of the His bundle right branch in the RV free wall



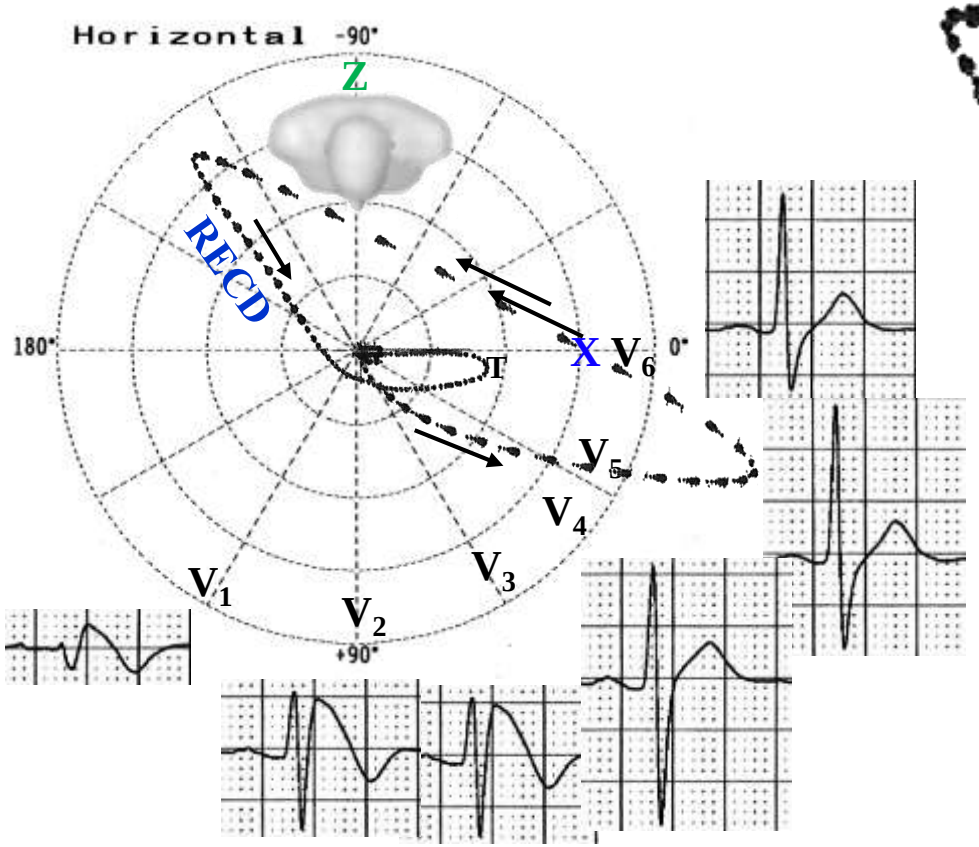




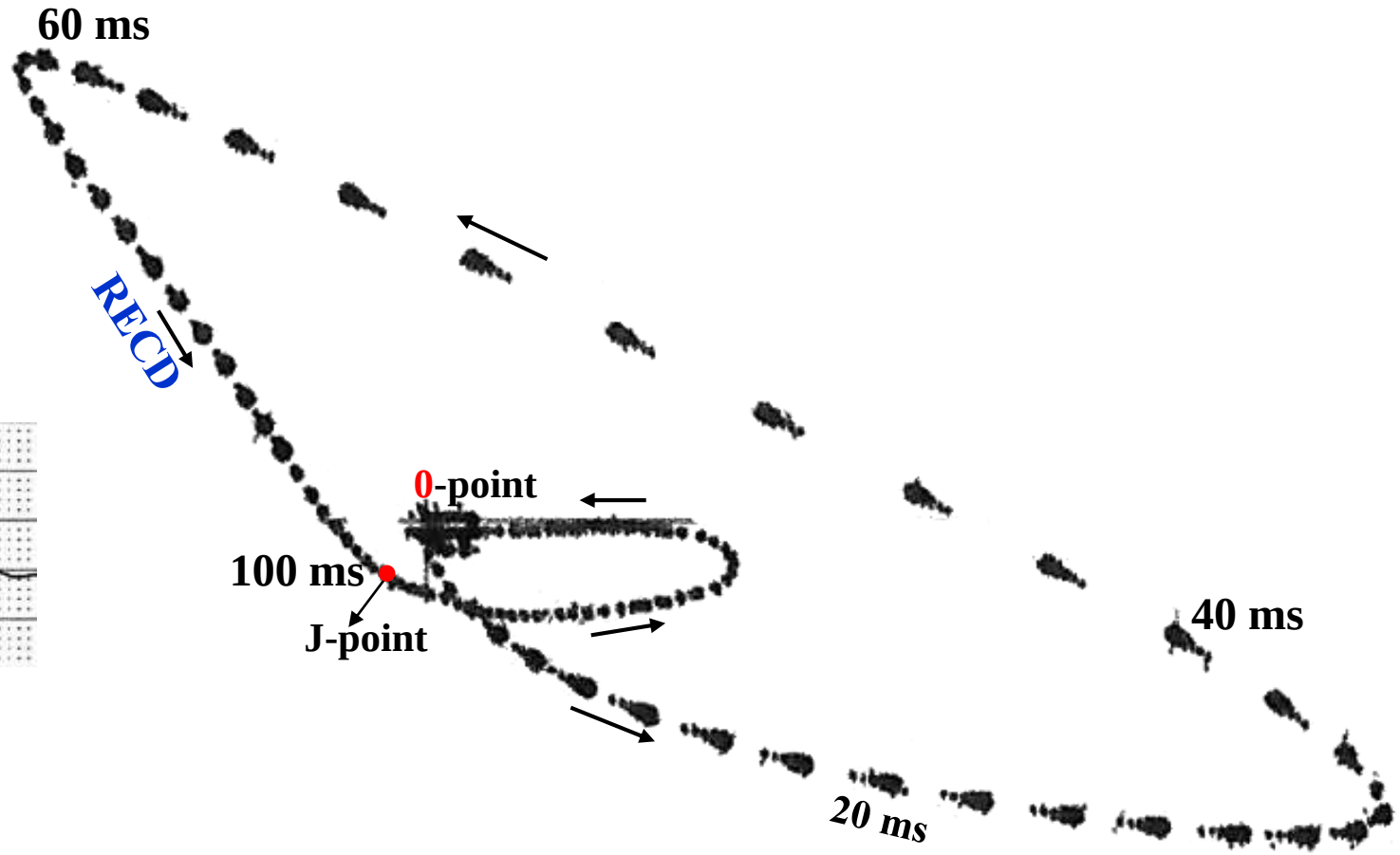
I – Superior or subpulmonary fascicle territory
II – Inferior or posterior-inferior fascicle territory
III – Middle fascicle territory

Structural epicardial alterations in the right ventricular outflow tract (RVOT) are the substrate for the conduction anomalies in Brugada syndrome (BrS). Electroanatomic mapping of endocardial unipolar voltage is an emerging tool that identifies accurately epicardial anomalies in the RVOT in BrS. Endocardial unipolar voltage mapping of the RVOT detects electroanatomical abnormalities in patients with BrS. Wide areas of abnormalities in endocardial unipolar voltage reflect structural epicardial abnormalities in the RVOT of patients with BrS (Letsas KP. Europace. 2018 Jun 1;20(FI1):f57-f63). BrS is associated to interstitial subepicardial fibrosis and a reduction in gap junction expression (connexin-43) in the RVOT, responsible for abnormal potentials, and its ablation abolishes BrS phenotype and arrhythmias risky for life. BrS is also associated to an increase in collagen throughout the heart. Abnormal myocardial structure and conduction are, therefore, responsible for BrS (Nademanee K. J Am Coll Cardiol. 2015 Nov 3;66(18):1976-1986).

ECG/VCG correlation in the horizontal plane



QRS and T loops characteristics in the horizontal plane



Initial 20ms forces directed to front and leftward, rapid passage from left to right between 40ms to 60ms and the final 40ms with Right End Conduction Delay (**RECD**) on posterior right quadrant: **depolarization mechanism.** J-point in the front and the right related the point 0. Both points are very distant from each other, which marks the elevation of point J and the ST segment typical of Brugada type 1 pattern, The T-loop pointing left as a finger, with both efferent and afferent limbs with slow and similar speed inscription. The QRS loop remembers the type C right ventricular overload typical of chronic obstructive pulmonary disease(COPD) or emphysema (**Luna filho B 1989**).

Non specific or unspecified intraventricular conduction disturbance, defect/or delay (NICD)

Other denominations: Non-Specific Intraventricular Conduction Delay (**NSIVCD**) or Non-specific Intraventricular Conduction Defect **(NIVCD)**

Definition

It is defined as an ECG pattern characterized by a wide QRS (≥120 ms in adults, >90 ms in children 8 to 16 years of age, and >80 ms in children younger than 8 years of age) without the appearance of LBBB, RBBB, ventricular pre-excitation, or masquerading bundle branch block (Surawicz 2009).

Observation: Masquerading bundle branch block is characterized by precordial leads that show a RBBB pattern while the limb leads resemble a LBBB+LAFB (extreme superior QRS axis deviation between −80° to −120°) (Elizari 2013). When LAFB is advanced, the QRS axis is ≈ − 60°. It can lead to a very small or even absent final s wave in lead I. In this scenario, ECG masquerading RBBB and it imitates LBBB is limb leads. When RBBB with LAFB resembles LBBB in the limb leads, it is called “standard masquerading bundle branch”. The so called precordial masquerading, the final S wave is absent in the left precordial leads (V5 and V6). In both, RBBB is diagnosed by typical QRS pattern in lead V1. This dromotropic disturbance is a rare but important finding on the ECG that usually indicates severe and diffuse conduction system disease with poor prognosis. This finding on an ECG is almost invariably associated with severe underlying heart disease.

Etiological and morphological classification of Non-specific Intraventricular Conduction Defects/Disturbance (NICD)

1. **Severe hyperkalemia**
2. **Some types of left ventricular enlargement/hypertrophy**.
3. **Sodium Channel Blocking Medications**

   3a Tricyclic antidepressants (TCAs) overdose: e. i. amitriptyline, nortriptyline, trimipramine, despiramine, protriptyline, and dothiepin poisoning. These are caused by blockage of cardiac fast sodium channels leading to disturbances of cardiac conduction and QRS prolongation.

   3b Class Ia antiarrhythmic drugs intoxication (Vaughan-Williams classification): Quinidine, procainamide, and dysopiramide. These are sodium channel blockers with intermediate association/dissociation, prolongation of action potential duration and QRS complex prolongation for values >140 ms or >35% of the baseline tracing, constitutes absolute indication of interruption of the drug (Heissenbuttel 1970).

   3c Class Ic antiarrhythmic drugs -sodium channel blockers with slow association/dissociation, pronounced reduction in phase 0 slope; no effect on action potential duration or effective refractory period (flecainide, propafenone, and moricizine).

   3d Local anaesthetics (bupivacaine, ropivacaine)

   3e Antimalarials (chloroquine, hydroxychloroquine)

   3f Dextropropoxyphene

   3h Carbamazepine

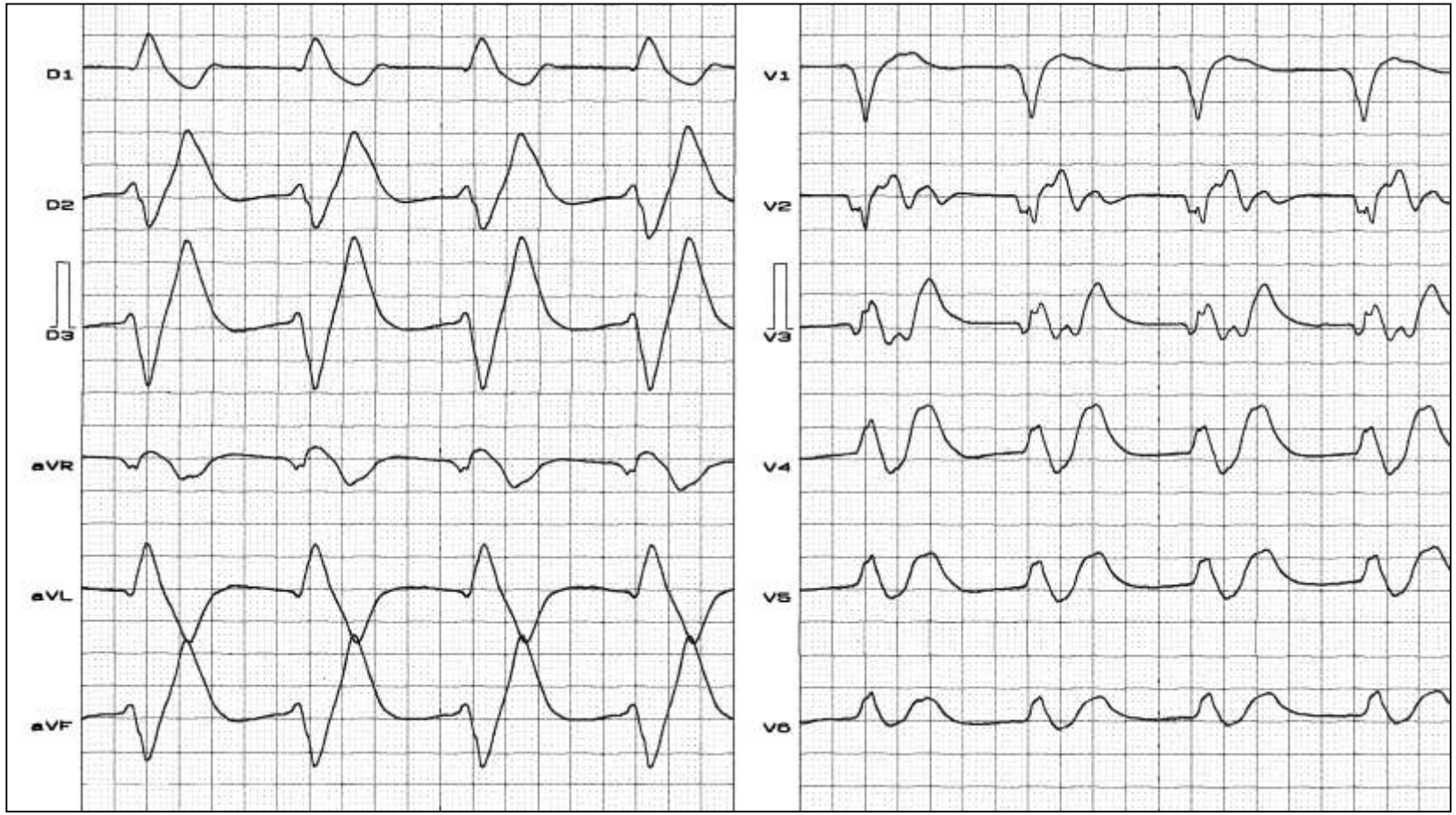
   3g Quinine
4. **Hypothermia**
5. **Peri-ischemic block (this is the present case)**
6. **Peri-infarction block**
7. **Atypical LBBB**
8. **Intraventricular parietal blocks**

Note: To date, no prospective, randomized, blinded trials have been performed to assess the benefit of CRT in patients with NICD. Indeed, the majority of patients included in the major CRT clinical trials were patients with LBBB, because this therapy had been initially proposed to specifically target the detrimental impact of the LBBB ventricular activation sequence (Eschalier 2015).

Posible etiologies of Nonspecific intraventricular conduction disturbance

1) Severe hyperkalemia

Typical ECG example of patient with extremely high level of serum potassium



Clinical diagnosis: chronic renal insufficiency and in dialysis. The patient delayed 72 hours the dialysis session. Severe hyperpotassemia of 9 mEq/L.

ECG diagnosis: absence of P wave, sinoventricular rhythm, 57 bpm, morphology of bizarre intraventricular severe disorder (QRSd: 240 ms) that is similar to complete LBBB. T waves with polarity matching with QRS from V3 to V6. Convergence of QRS with T wave that outlines smooth diphasic wave or sine curve.

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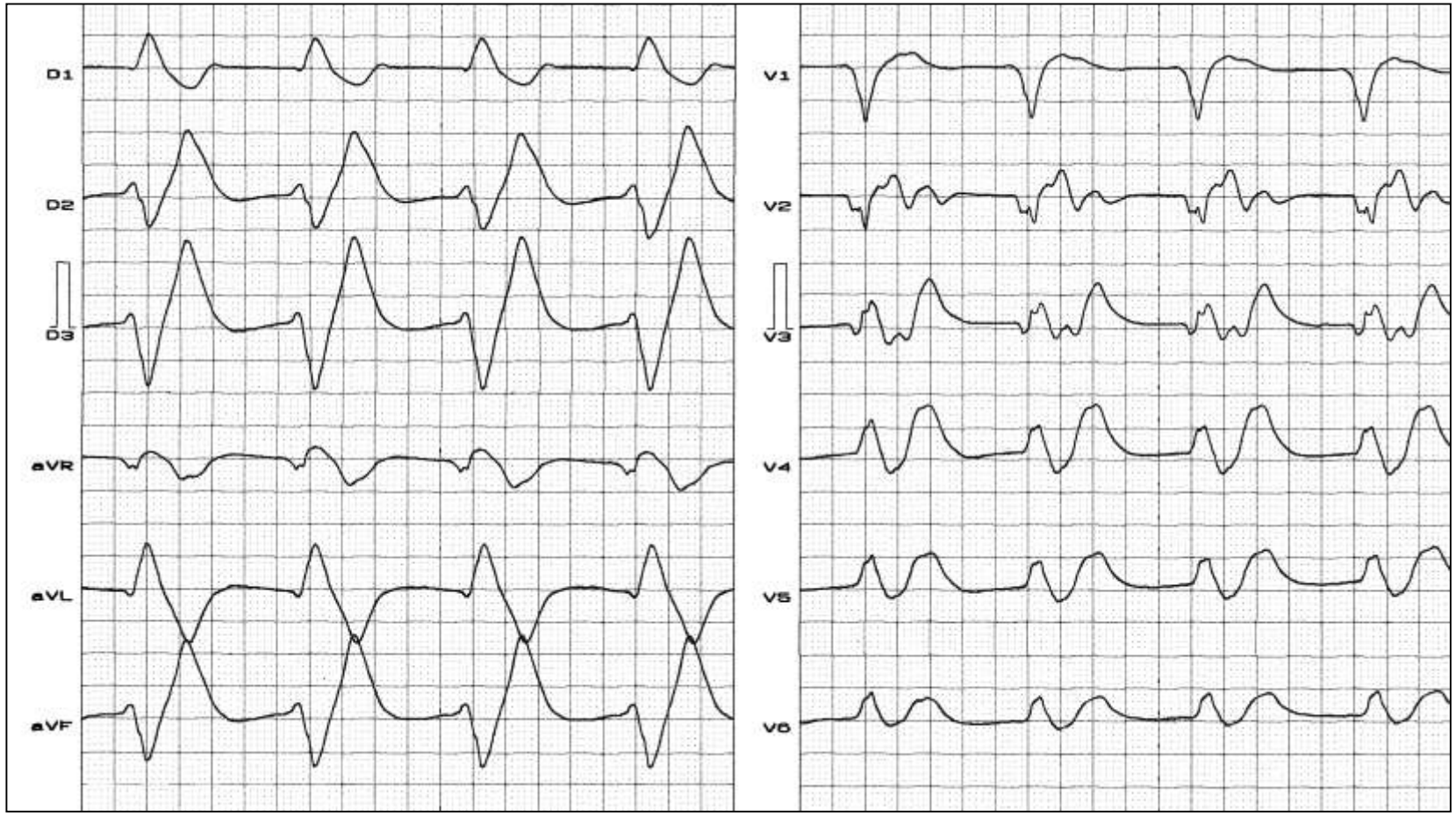

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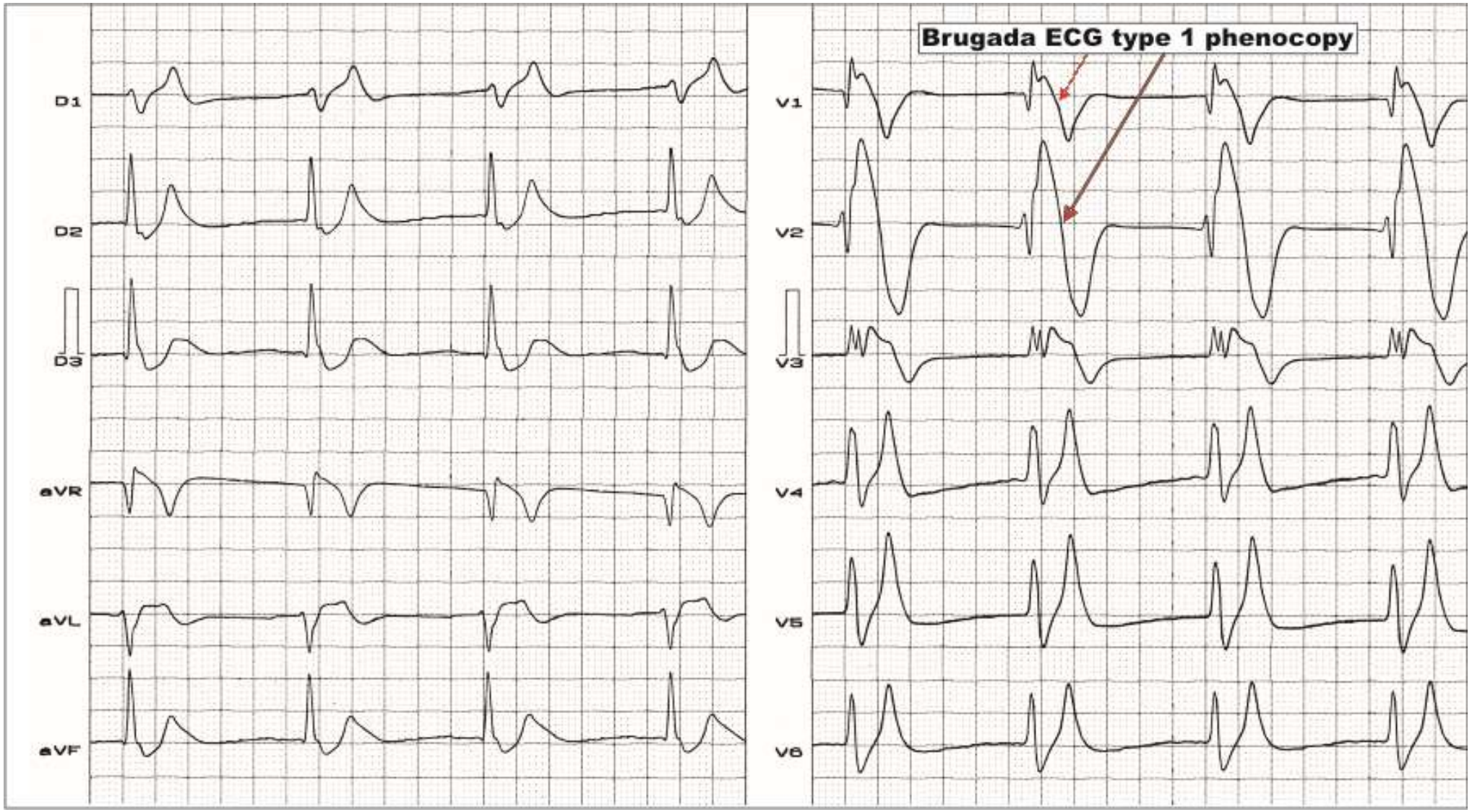
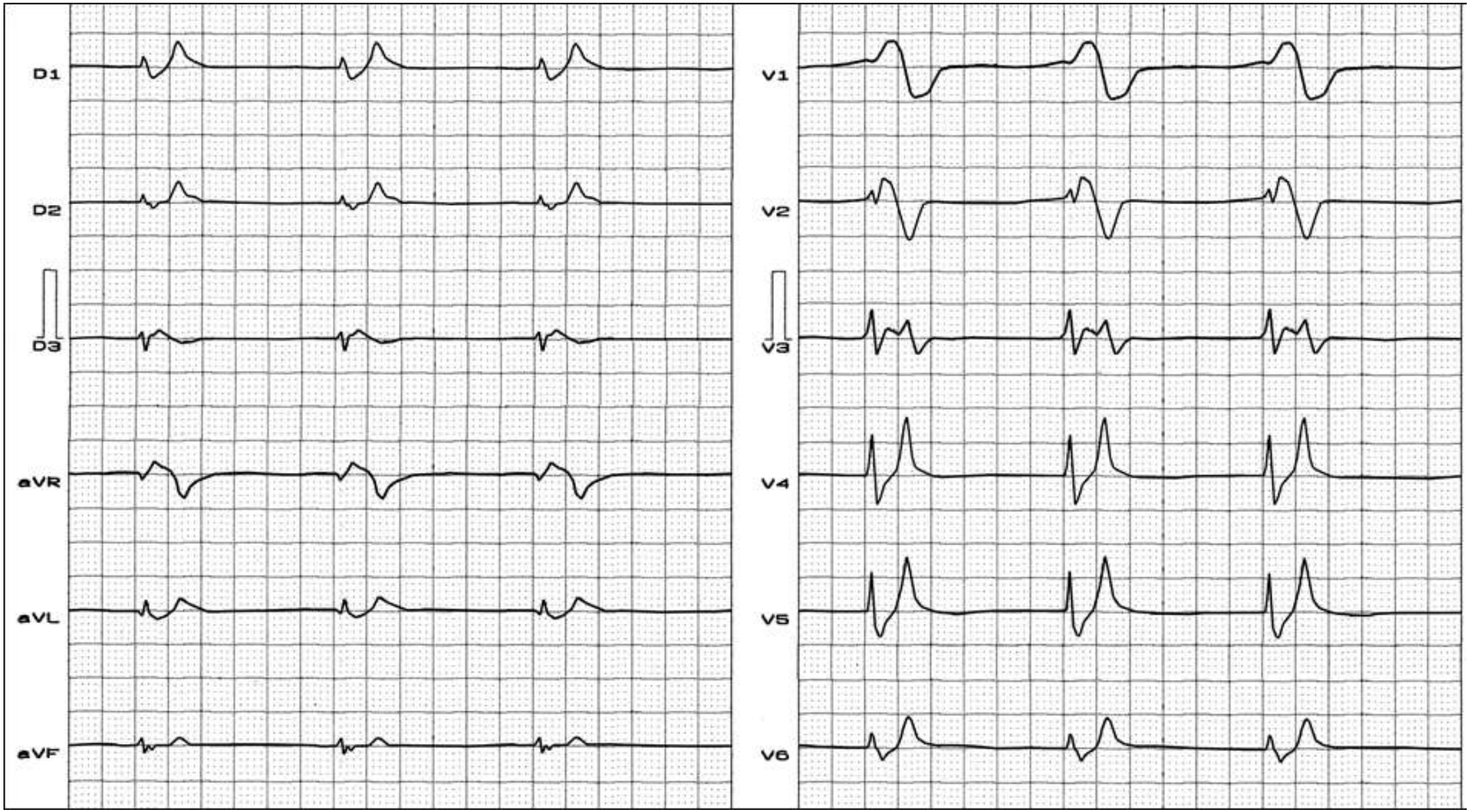
Severe hyperkalemia



Clinical diagnosis: terminal renal insufficiency. Severe hyperkalemia: K^+ 8.7 mEq/L. This sign is known as "dialyzable injury current". ECG diagnosis: very likely, junctional with P waves near the J point, HR: 54 bpm, QRSd: 160 ms, ST segment elevation from V1 to V3 and I, aVL and aVR. V1 to V3 displays ST segment with upwardly convex pattern, similar to Brugada syndrome or Brugada phenocopy", typical T waves in "tent", pointed, and with a narrow base. Numerous conditions which resemble the type-1 BrS pattern should be ruled out. These are called "acquired forms of BrS", "Brugada-like ECG pattern" or Brugada phenocopyes (Nguyen 2011; Riera 2010) (an environmental condition that imitates or mimics one produced by a gene).

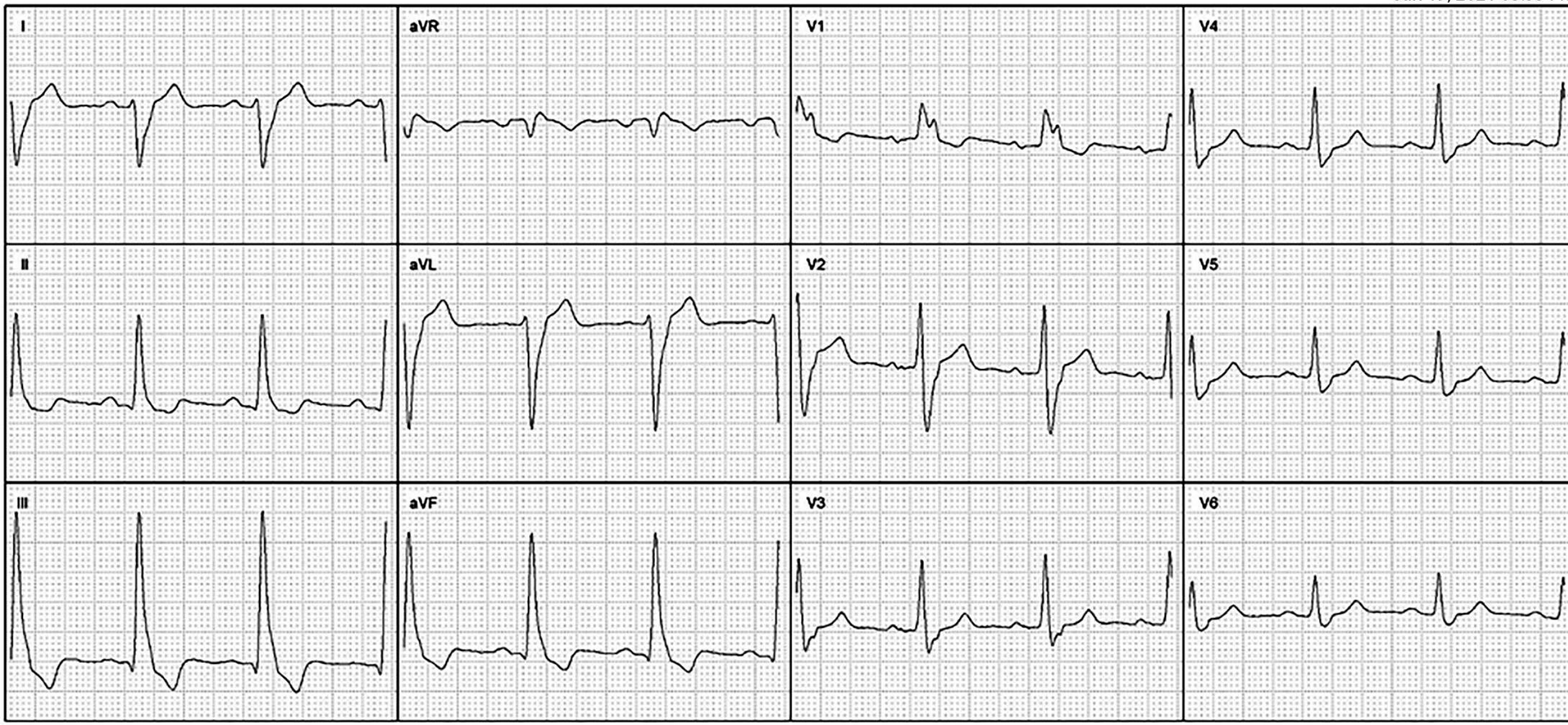
Typical ECG of hyperpotassemia associated to Brugada-like pattern

Name: FHM; **Sex:** M; **Age:** 56 y/o; **Ethnic group**: Mulatto; **Weight:** 65 Kg; **Height:** 1.62 m;**Date:** 05/11/2003

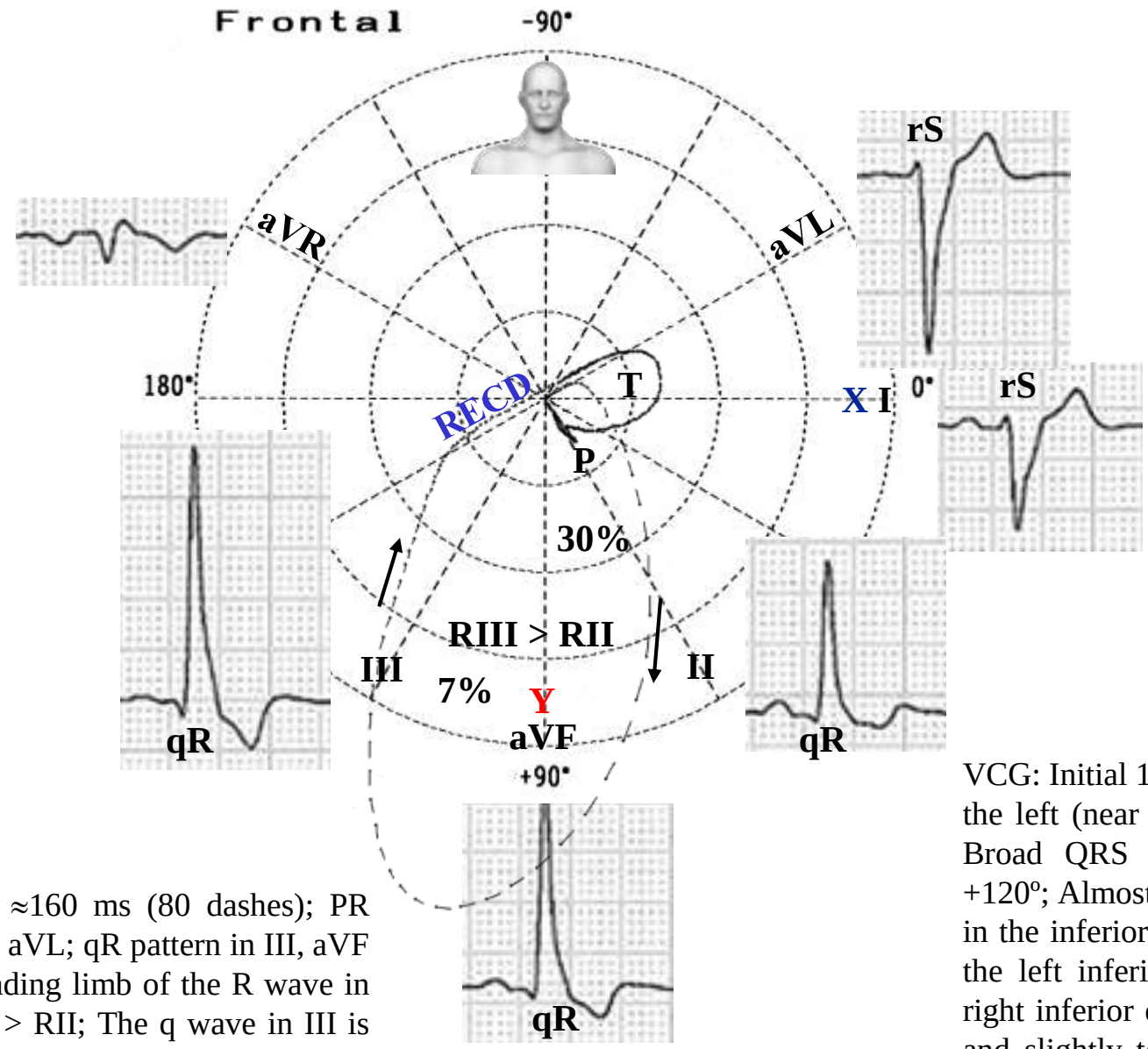


P wave is not identified; sinoventricular rhythm. Severe hypercalcemia with electrocardiographic Brugada-like pattern. Chronic renal insufficiency with hypercalcemia has been described transitorily, and reversed with dialysis. Apiculate T waves in “tent” from V4 through V6.

Truly bifascicular block: LPFB + Complete RBBB



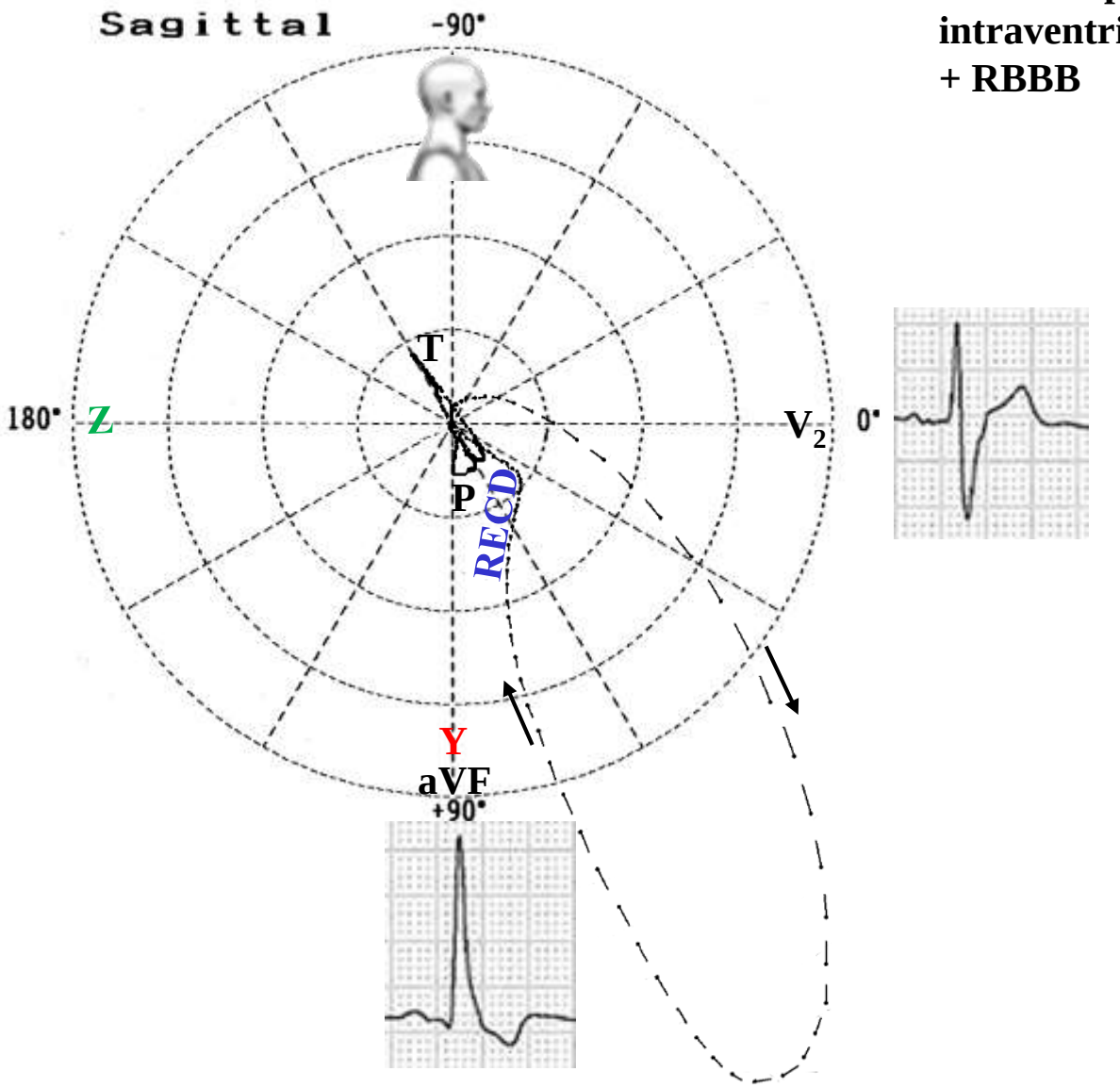
Sinus rhythm, heart rate 71 bpm, QRS axis +120°; QRS duration ≈160 ms; PR interval 180 ms; rS pattern in leads I and aVL; qR pattern in III, aVF and II: qIII > qII; Slurring in the descending limb of the R wave in III, II and aVF (middle-final slur); RIII > RII; The q wave in III is greater than the q wave in II and aVF. If there is association with inferior infarction, the Q wave > 40 ms. Conclusion: LPFB + CRBBB.



ECG: QRS axis +120°; QRS duration ≈160 ms (80 dashes); PR interval 180 ms; rS pattern in leads I and aVL; qR pattern in III, aVF and II: qIII > qII; Slurring in the descending limb of the R wave in III, II and aVF (middle-final slur); RIII > RII; The q wave in III is greater than the q wave in II and aVF. If there is association with inferior infarction, the Q wave > 40 ms.

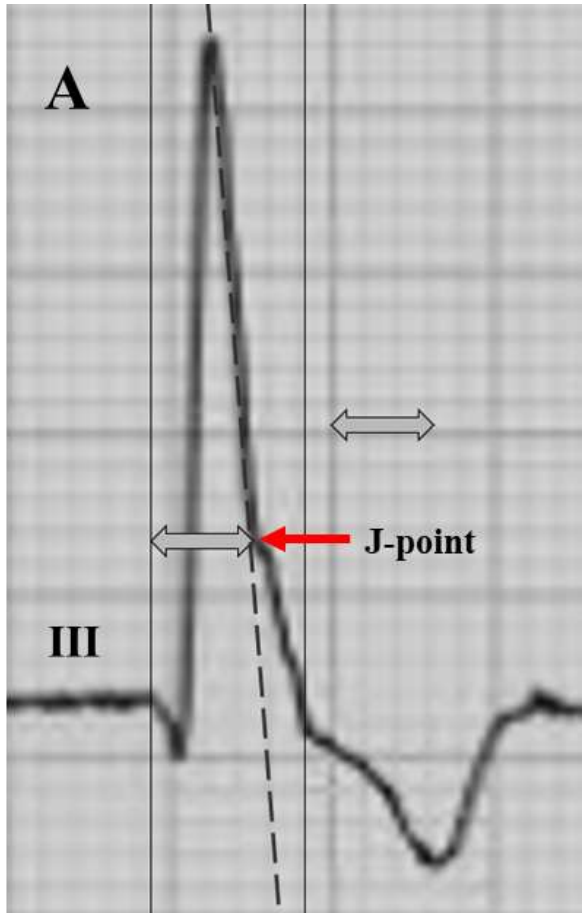
VCG: Initial 10 to 20 ms vector heading above and to the left (near -30°) with delay (initial 10 to 25 ms); Broad QRS loop, CW rotation; Maximal vector +120°; Almost all the loop is located below the X line in the inferior quadrants; 30% of the loop located in the left inferior quadrant and 70% or more in the right inferior quadrant; Afferent limb heading below and slightly to the left, and the efferent one to the right; Middle-terminal portion of the QRS loop (vector of 60 ms to 100 ms) with delay of 40 ms (RECD); T loop with CW rotation, heading to the left: alteration secondary to ventricular

Right Sagittal Plane

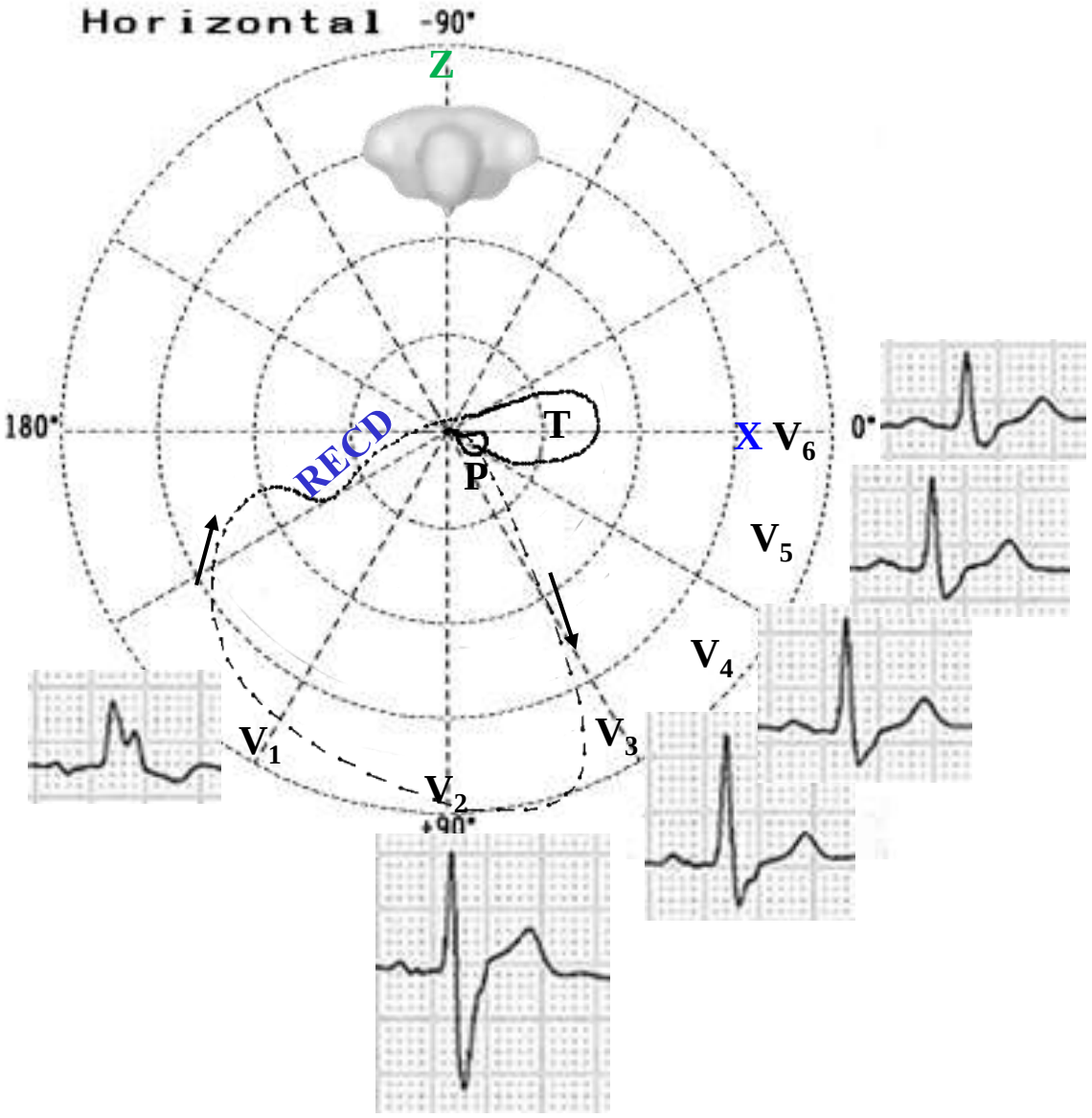


J wave phenocopy (J-wave-like deflection) at the terminal portion of the QRS in a patient with intraventricular conduction disturbance: LPFB + RBBB

End-QRS slurring in II, III and aVF



Vector of initial 10 to 20 ms to above with delay; Most of the QRS loop located in the anteroinferior quadrant; QRS loop of clockwise rotation; Final RECD; T loop heading to above and minimally to back.



CRBBB Kennedy type III or C

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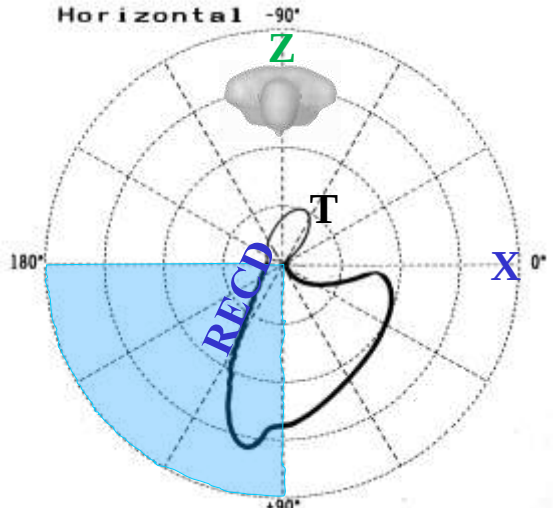
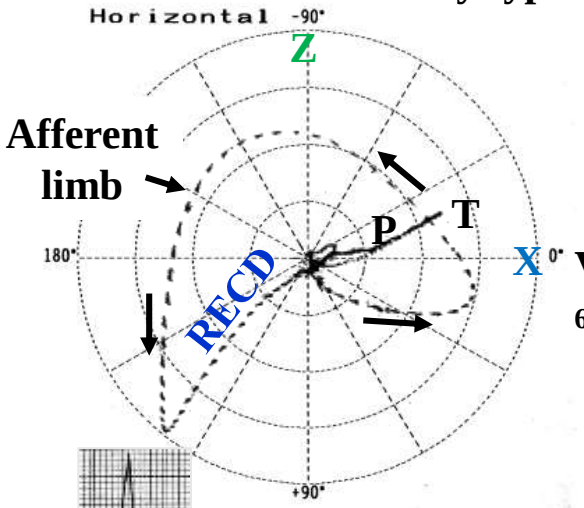
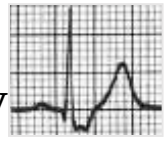


Initial vector to the front, QRS loop of CW rotation and main body located in anterior quadrants and RECD in the anterior right quadrant.

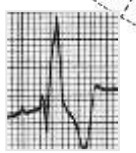
VCG classification of Complete Right Bundle Branch Block in the HP

Grishman or Kennnedy type I



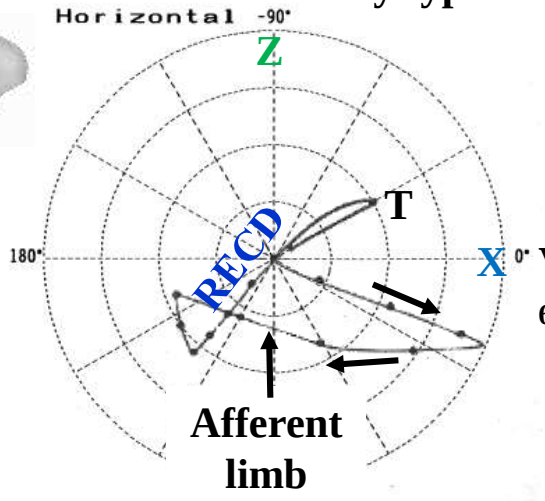


V 6



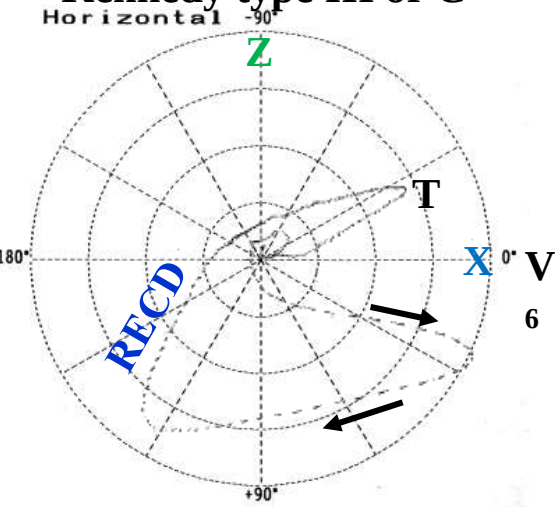
V_1

Cabrera or Kennedy type II



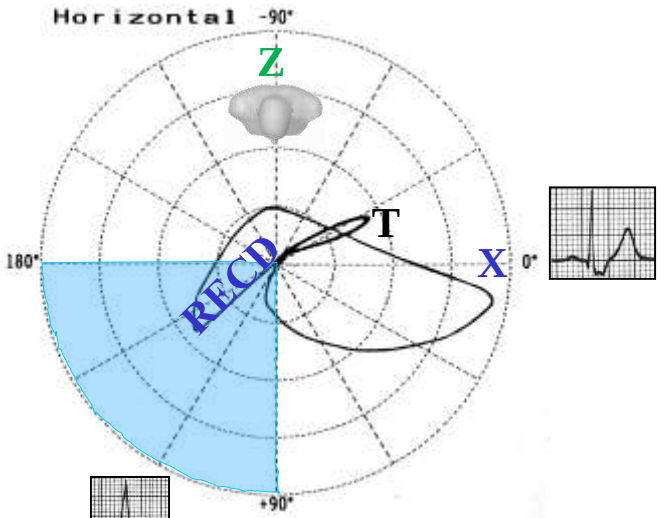
V 6

Kennedy type III or C

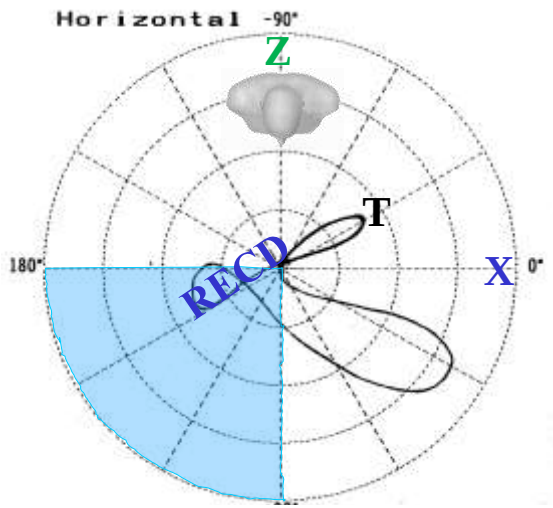


V 6

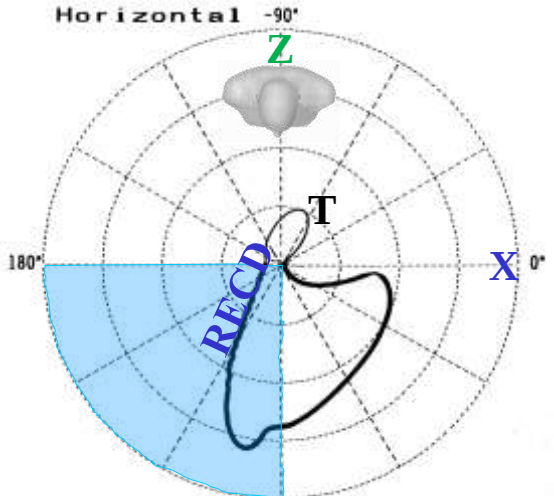
RECD: Right End Conduction Delay



Right Anterior Quadrant



We find type II in ASD, PS, in COPD and more rarely in chronic Chagasic myocardits.



Initial vector to the front, QRS loop of CW rotation and main body located in anterior quadrants.