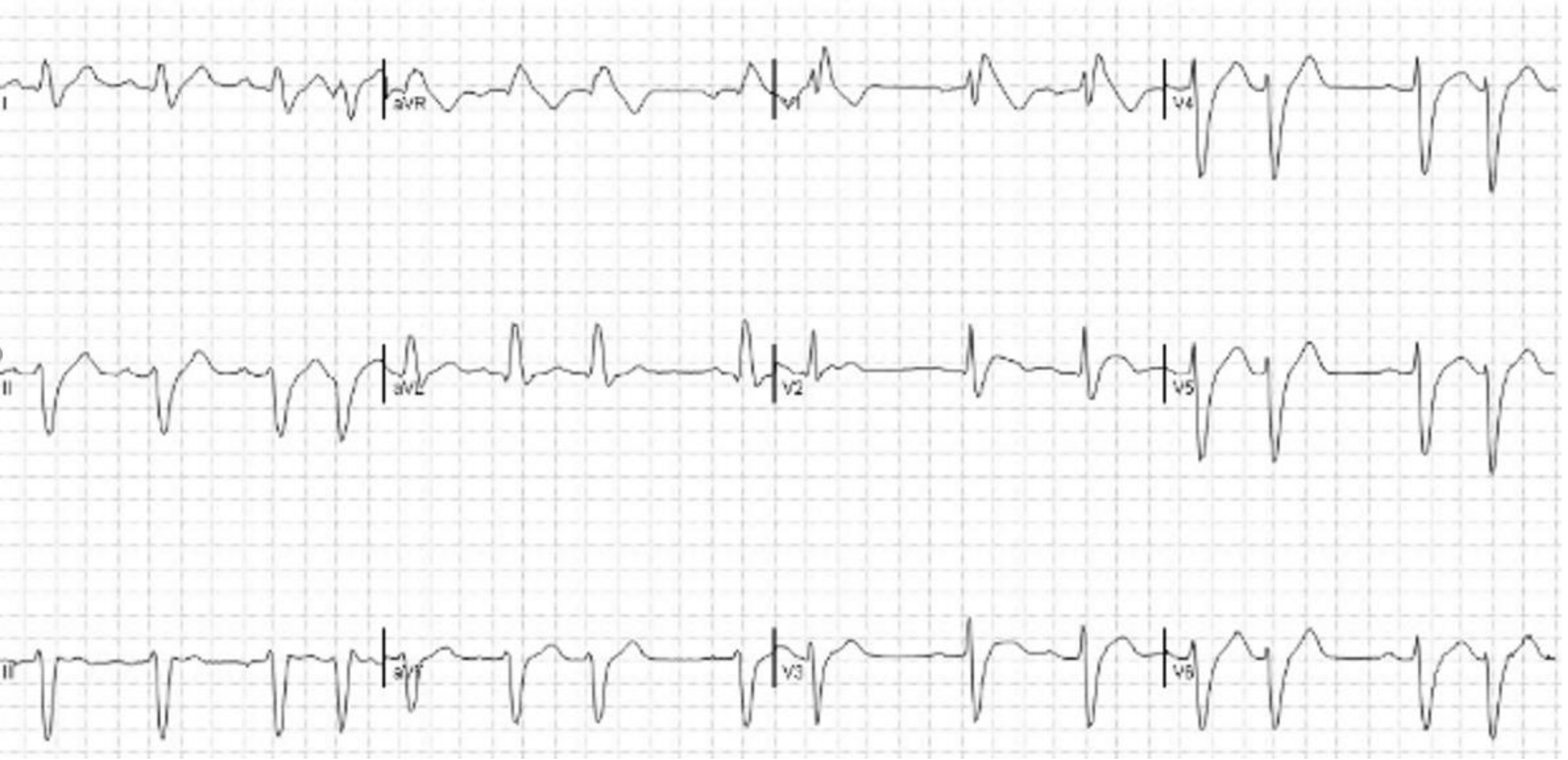
Hombre de 46 años. ACV isquémico sin secuelas motoras a primeros días de agosto. No tengo datos de la TAC. Anda mareado, soñoliento, algo confuso por momentos. Es hipertenso de al menos desde hace cinco años, tratado irregularmente, ahora tratado con Losartan 50 mg/día, no compensado. Ayer, telefónicamente, le indiqué que tomara dos veces por día. Hoy acude al consultorio con PA normal. A la auscultación, ritmo irregular, no soplo. No le habían hecho aún un ECG, llamativamente. No hay antecedentes familiares de MS. Diabético tipo 2. Por eco: HVI, STRAIN severamente reducido.

¿Cual és el posible diagnóstico clínico? ¿ Cual es el diagnóstico electrocardiográfico? ¿Cuales son los próximos pasos a seguir?

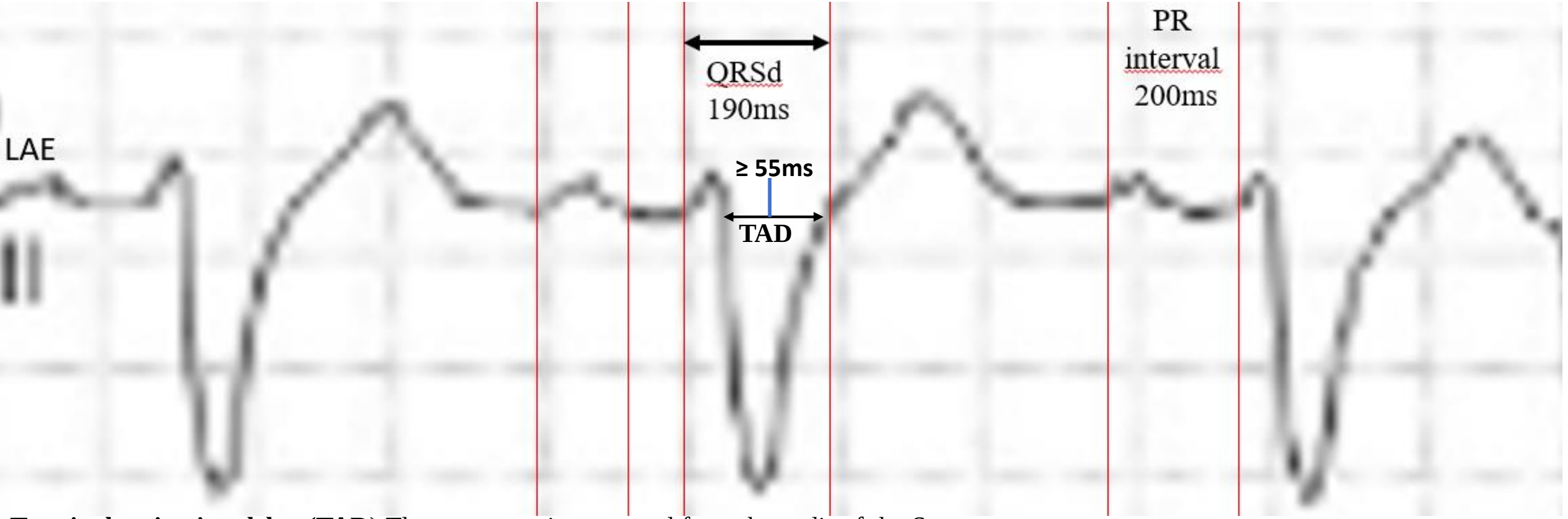
46 year old Man Ischemic stroke without motor sequelae in the first days of August. I do not have Ischemic stroke data. He is dizzy, sleepy, somewhat confused at times. He has been hypertensive for at least five years, treated irregularly, now treated with Losartan 50 mg / day, not compensated. Yesterday, by telephone, I told him to take it twice a day. Today he goes to the office with normal BP. At auscultation, irregular rhythm, I do not blow. He had not yet done an ECG, strikingly. He has not family history of MS in family members . Diabetic type 2. By echo: LVH with strain severely reduced.

What is the possible clinical diagnosis? What is the electrocardiographic diagnosis? What are the next steps to follow?

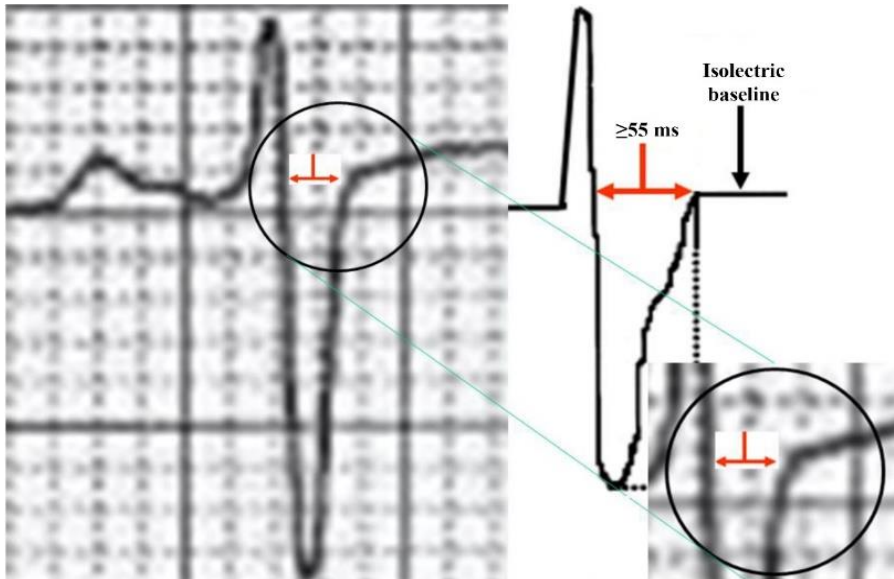
Andrés Ricardo Pérez-Riera M.D.Ph.D
ECG analysis

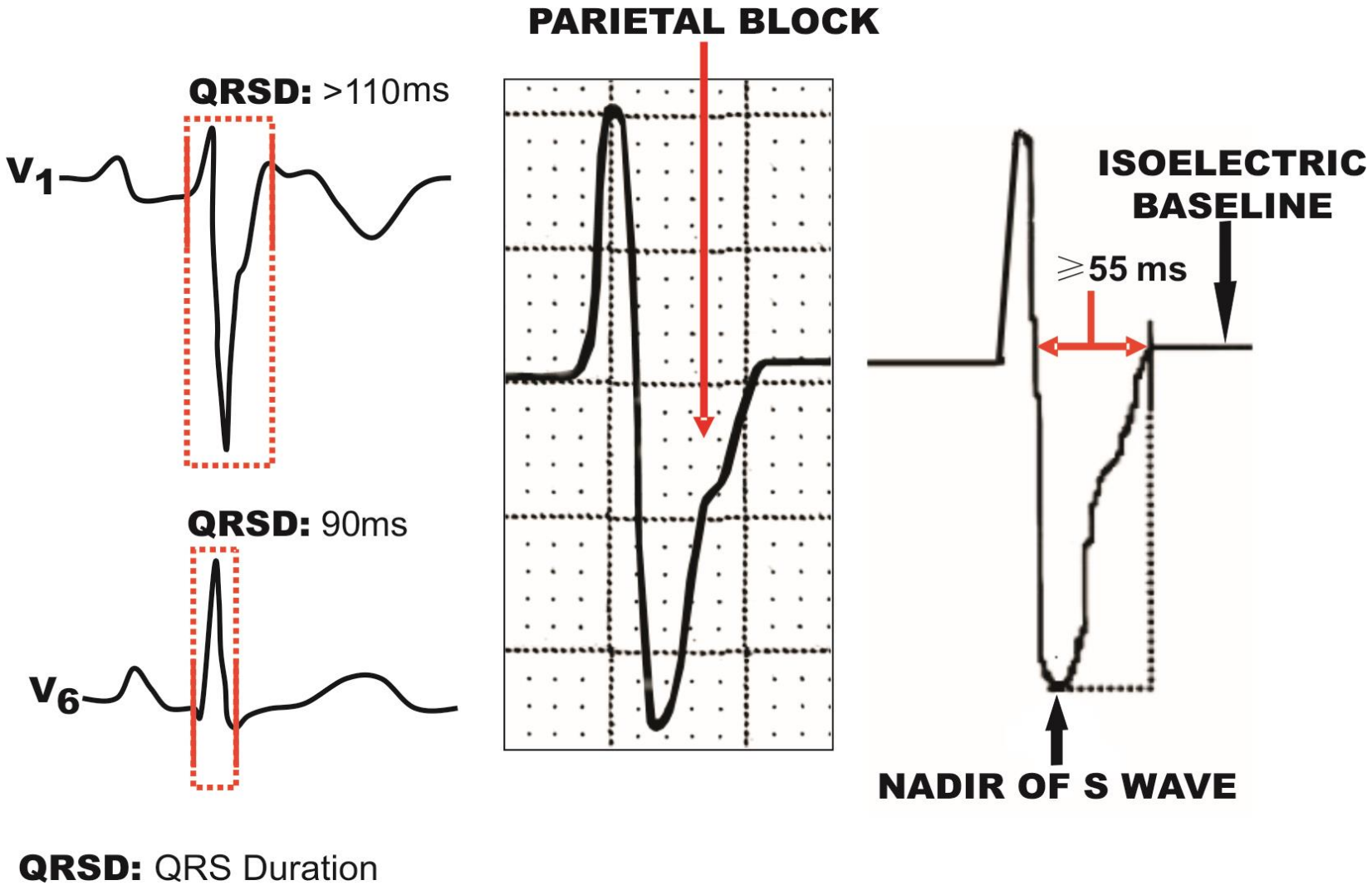


ECG: Left atrial enlargement + PR interval 200ms+ Complete RBBB(QRSd 190ms) + LAFB + Spontaneous type 1 Brugada pattern+ premature contractions + aVR sign: final R wave of aVR lead >3 mm. Presence of prominent final R wave on aVR lead; R wave ≥3 mm or R/q ≥0.75 in lead aVR (aVR sign). Slow conduction at the RVOT may contribute to the induction of VF by PVS.



Terminal activation delay (TAD) The parameter is measured from the nadir of the S wave to the end of the QRS complex. This is considered positive in AC when the QRS duration is ≥55 ms (Nasir et al., 2004) (Figure 3). TAD is registered in □5%–20% of AC cases (Nunes de Alencar Neto, Baranchuk, Bayes-Genis, & Bayes de Luna, 2018).

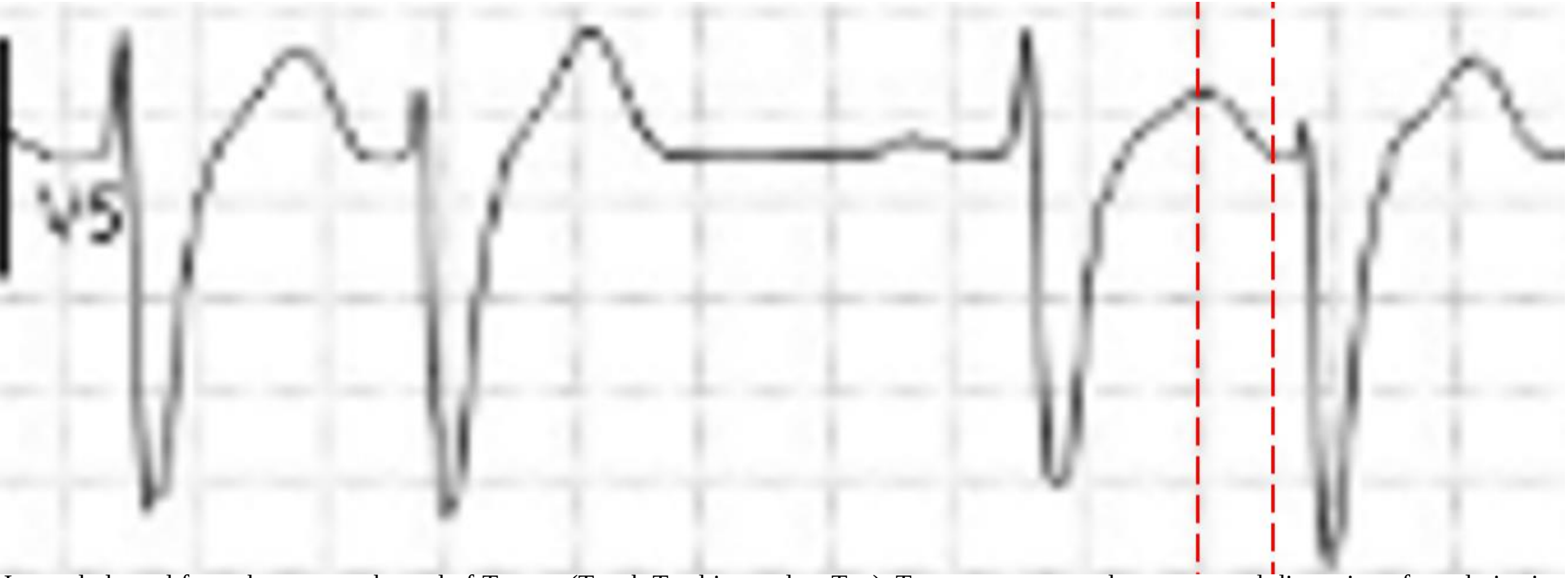




Pitzalis et al (Pitzalis 2003) identified the selective prolongation of QT interval duration in the right precordial leads (V_1 to V_3) in comparison to the left ones (V4 to V6). As the QT interval is made up by ventricular depolarization (QRS) plus ventricular repolarization (ST/T) we think that this selective prolongation represents a certain degree of parietal block in the RVOT, as the one observed in ARVC/D.

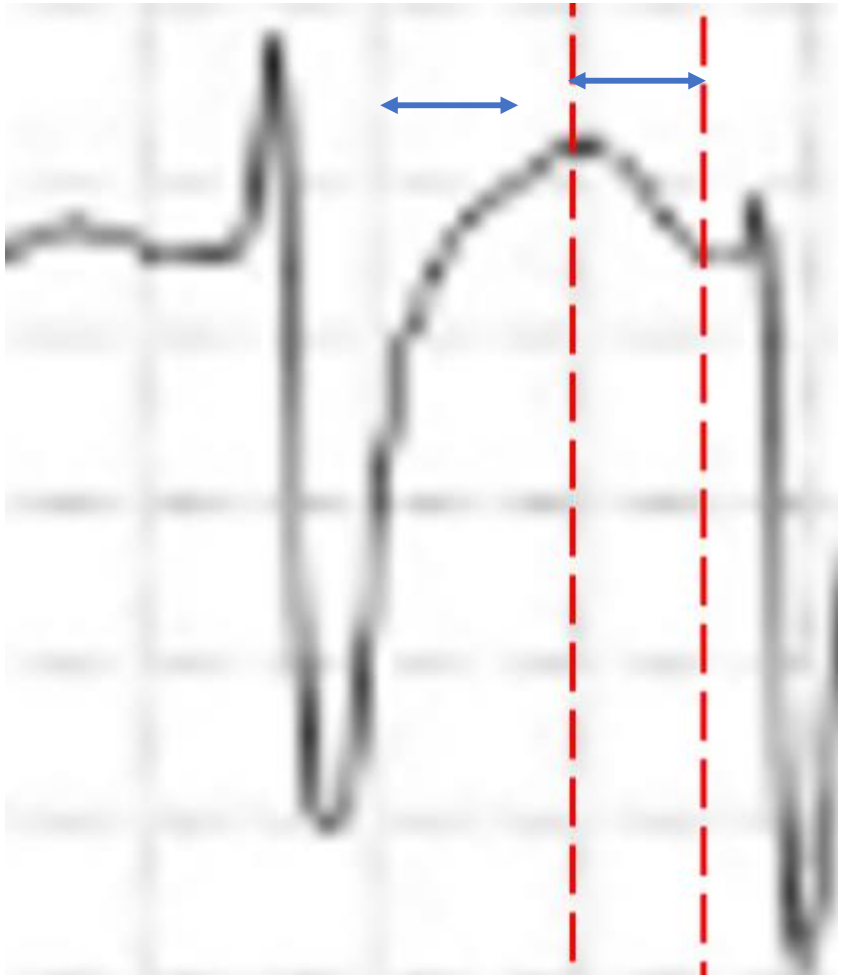
Terminal activation delay (TAD) The parameter is measured from the nadir of the S wave to the end of the QRS complex. This is considered positive in AC when the QRS duration is ≥55 ms (Nasir et al., 2004) (Figure 3). TAD is registered in □5%–20% of AC cases (Nunes de Alencar Neto, Baranchuk, Bayes-Genis, & Bayes de Luna, 2018

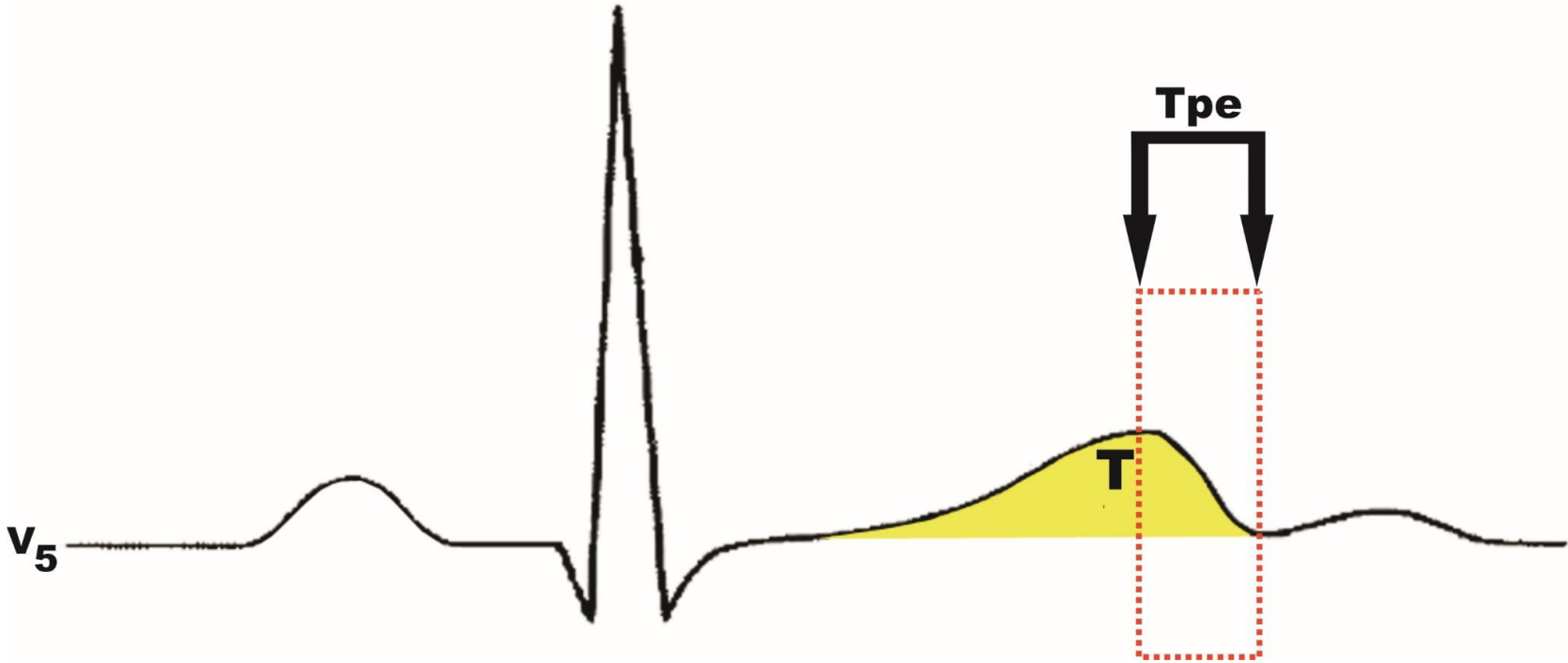
$T_{peak} - T_{end}$ prolongation and $T_{peak} - T_{end}$ dispersion **$T_{peak} - T_{end}$ 110ms**



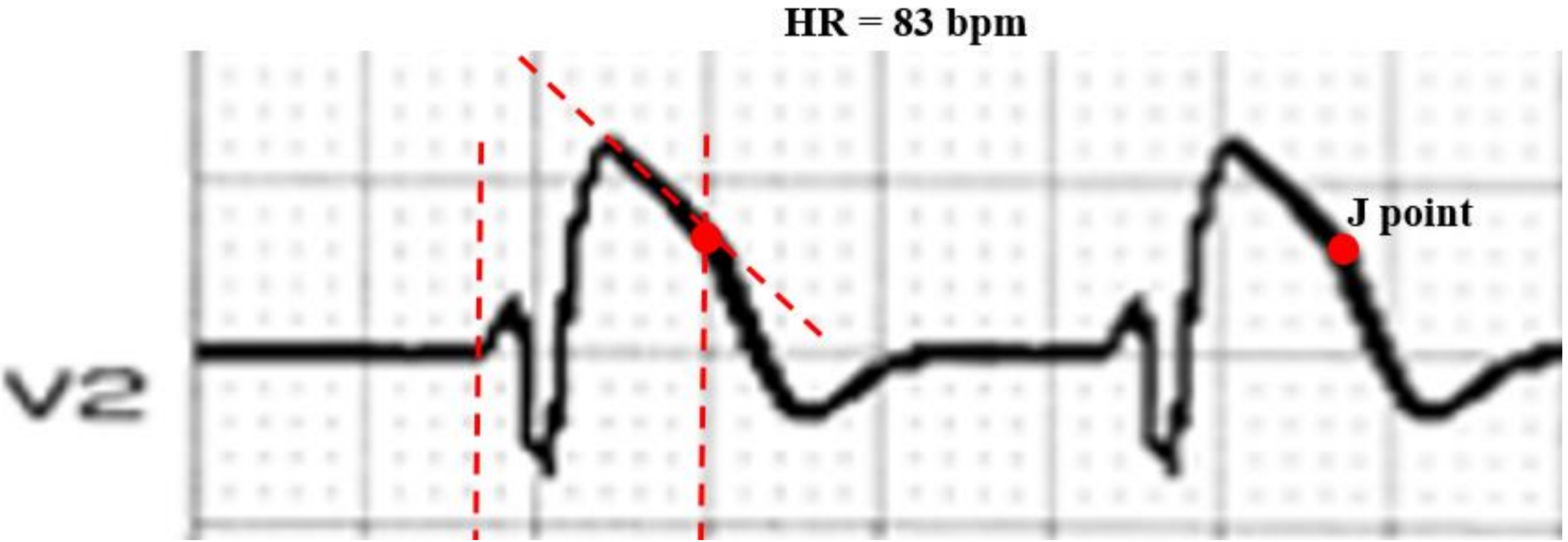
Interval elapsed from the apex to the end of T wave (Tpeak-Tend interval or Tpe). Tpe may correspond to transmural dispersion of repolarization and consequently, the amplification of this interval is associated to malignant ventricular arrhythmias. The normal value of Tpeak/Tend interval (Tpe) is 94 ms in men and 92 in women when measured in the V_5 lead. Tpe prolongation to values ≥120 ms is associated to a greater number of events in patients carriers of BrS

$T_{peak} - T_{end}$ 110ms





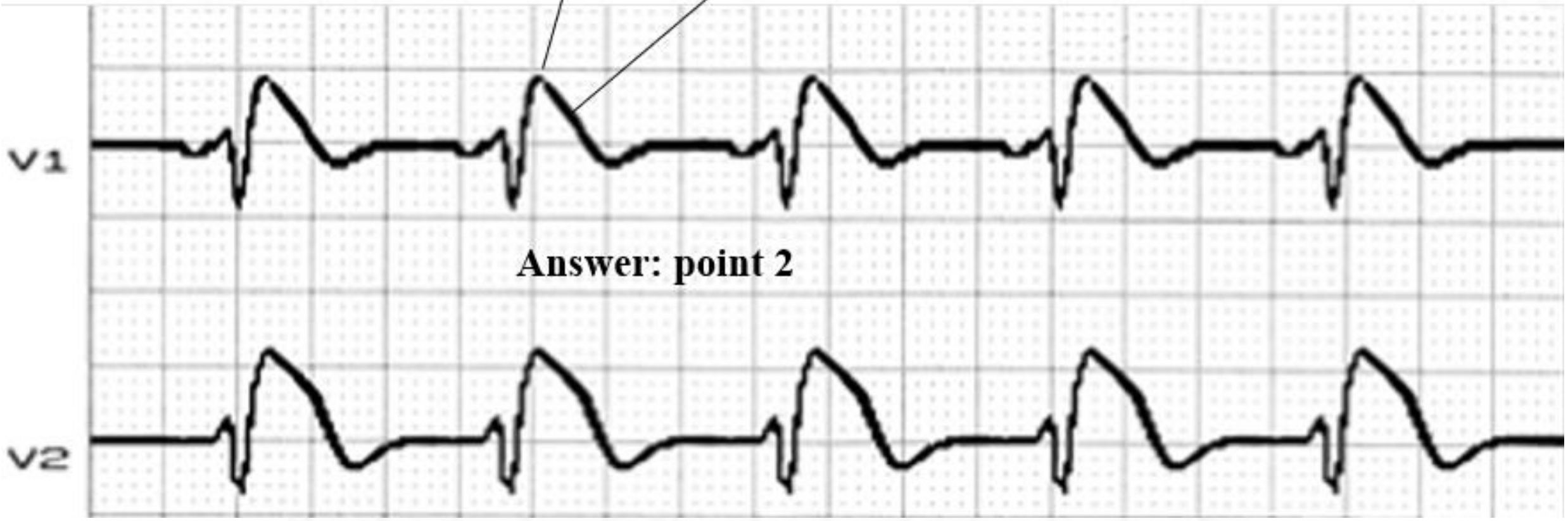
Prolonged QRS duration measured from lead II or lead V2 ≥120 ms (Junttila 2008)



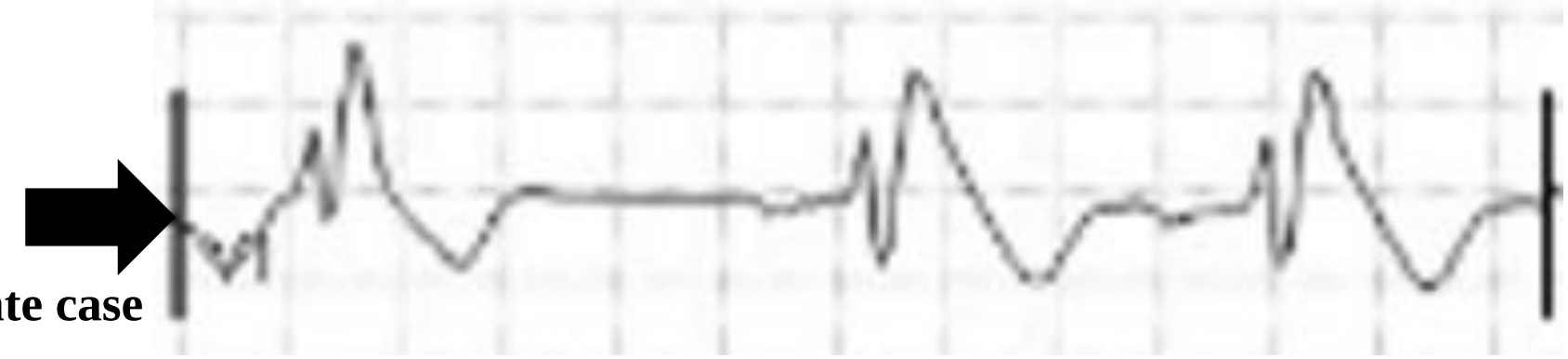
Vertical dotted lines show onset and termination of the QRS complex in V2. In this case QRSd = 165 ms. It is an ECG marker of events.

Where is the end of the QRS complex?

Point 1 or Point 2?



Answer: point 2

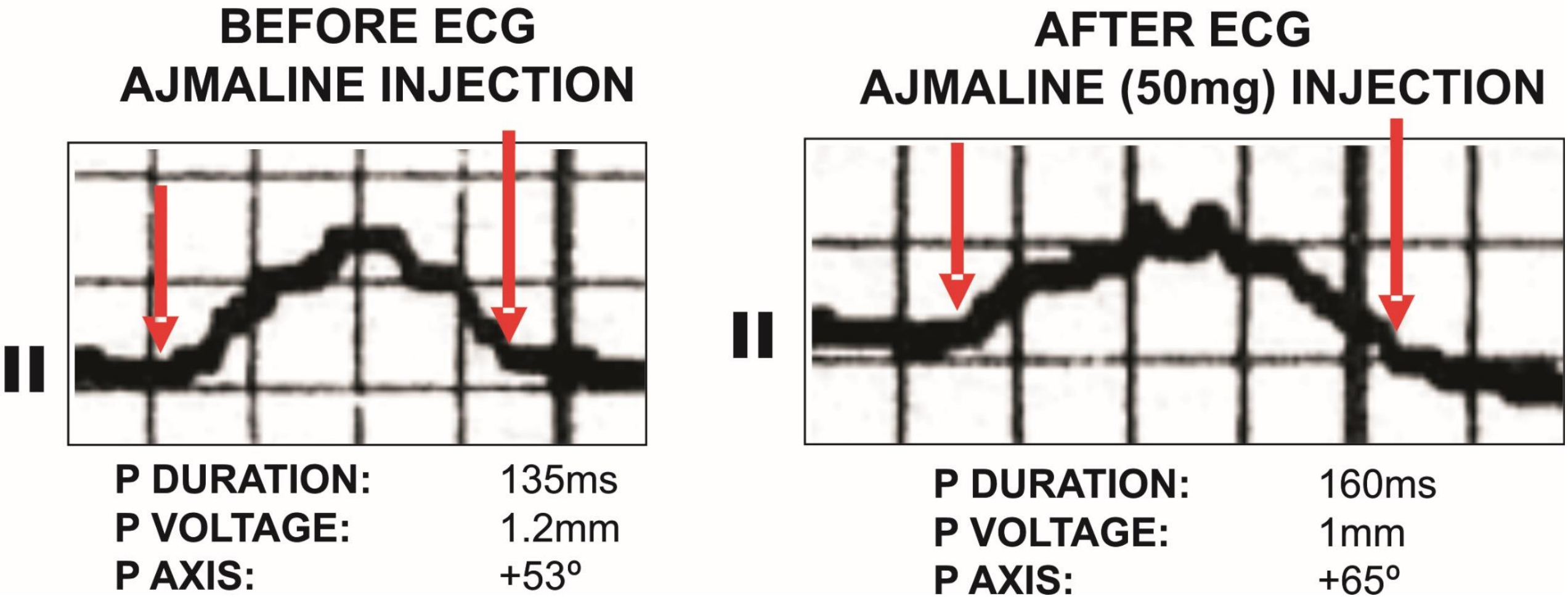


V_1

The presente case

ECG markers in identifying patients at risk in Brugada syndrome

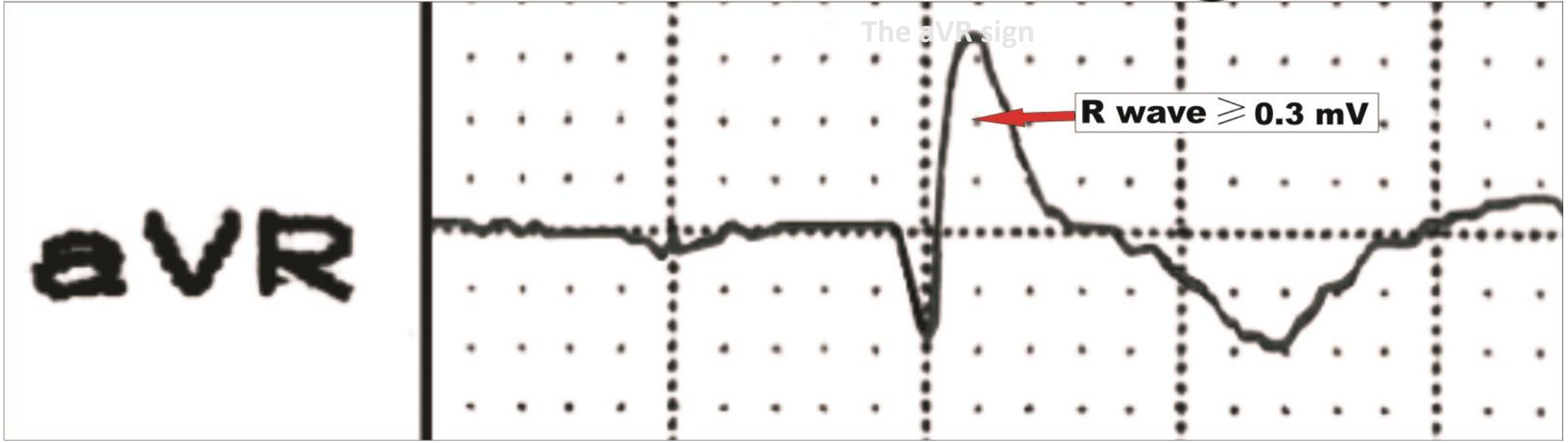
1. Augmented P-wave duration in lead II, P-wave dispersion (Letsas 2009). (**Present in this case**), See next slide.
2. PR prolongation consequence of HV split or HV prolongation (Miyamoto 2011).
3. Presence of prominent final R wave on aVR lead R wave ≥ 3 mm or R/q ≥ 0.75 in lead aVR (aVR sign). Slow conduction at the RVOT may contribute to the induction of VF by PVS (Babai Bigi 2007).**Present in this case. See next slides.**
4. The presence of a spontaneous type I ECG (**Present in this case**), history of syncope, ventricular effective refractory period <200 ms, and QRS fragmentation seem useful to identify candidates for prophylactic ICD (Priori 2012).
5. Inferolateral early repolarization (Kamakura 2009).
6. Prolonged QRS duration measured from lead II or lead V2 ≥120 ms (Junttila 2008). (**Present in this case**),
7. QTc interval more than 460 ms in lead V2 (Take 2011) and QT-interval prolongation in right precordial leads (Pitzalis 2003). Increase in QRS complex duration (>110º) in the right precordial leads, in absence of CRBBB: parietal block.
8. $T_{peak} - T_{end}$ prolongation and $T_{peak} - T_{end}$ dispersion (Castro Hevia 2006).
9. Dynamic alterations in the amplitude of the ST elevation (Take 2011).
10. Loss of rate-dependent QT dynamics (Sangawa 2009).
11. The presence of horizontal (as opposed to rapidly ascending) ST segment after the J point (Takagi 2013).
12. Augmentation of the ST segment elevation during the early recovery phase of exercise test (Makimoto 2010).
13. Deep negative T wave in lead V1 (Miyamoto 2011). (**Present in this case**)
14. The presence of atrial fibrillation (Kusano 2008).
15. The presence of late potentials (LPs) (Ikeda 2001).



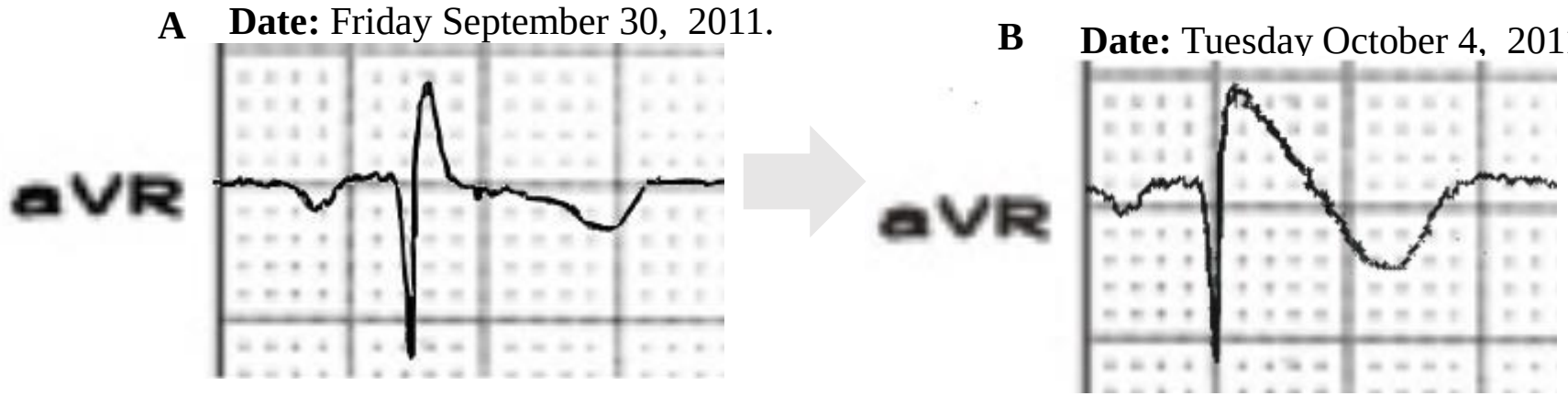
The tracing shows the P wave in a patient with BrS and positive SNC5A mutation, performed before and immediately after ajmaline test (1 mg/kg).

P wave duration (Pd) before the injection is prolonged (Pd=135 ms). After drug administration Pd wave increases more (Pd=162 ms).

These atrial dromotropic disorders could be the substrate for reentrant atrial tachycardias such as AF.

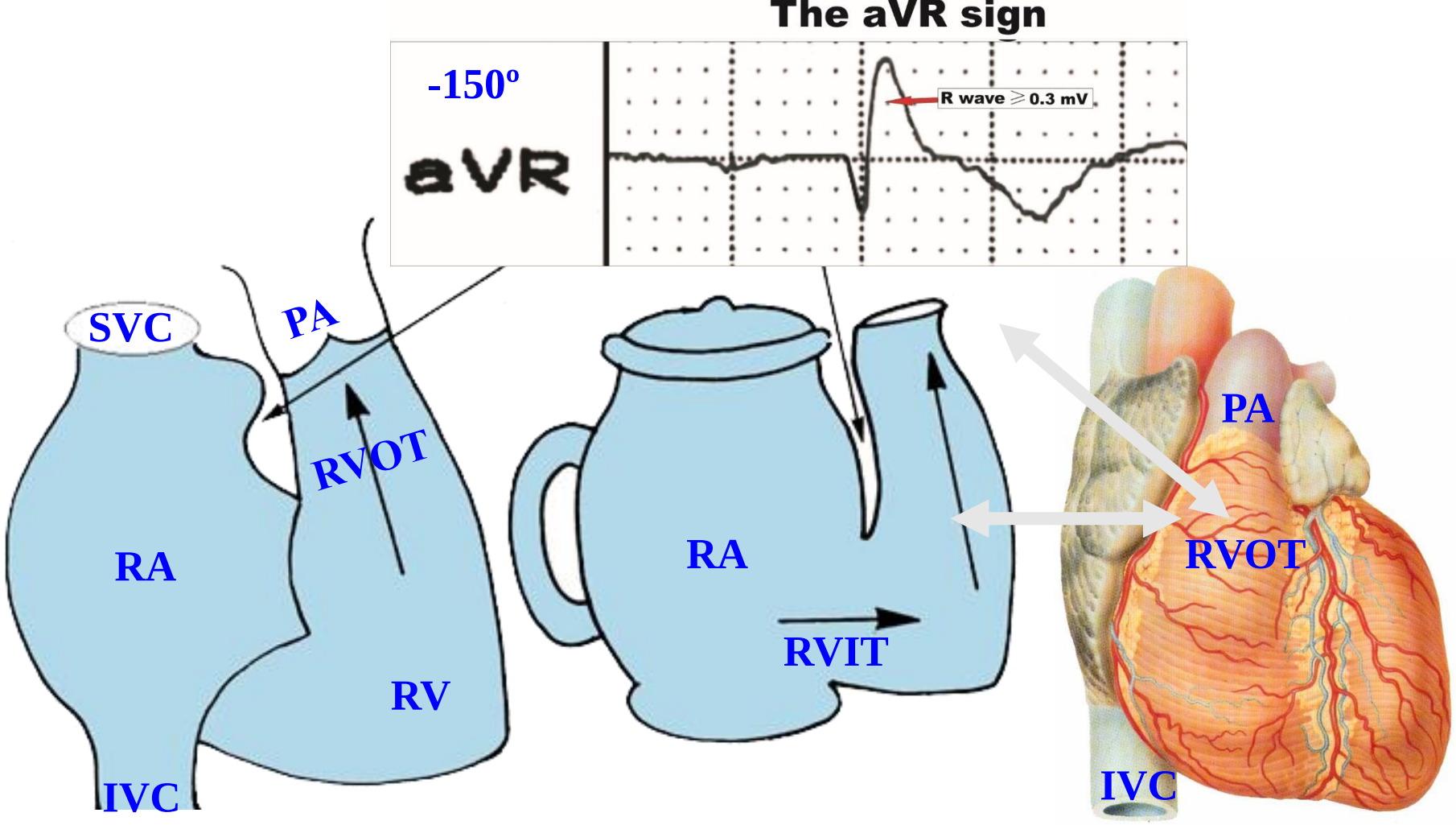


aVR sign: final R wave of aVR lead >3 mm



Terminal broad R wave of the QRS complex in lead aVR (Babai Bigi 2007)

The aVR sign: Presence of prominent final R wave on aVR lead; R wave ≥3 mm or R/q ≥0.75 in lead aVR (aVR sign). Slow conduction at the RVOT may contribute to the induction of VF by PVS.



The BrS affects predominantly the right ventricle in the right ventricle outflow tract (RVOT) epicardium (Doi 2010). 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 (Coronel 2005; Pérez-Riera 2012). 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 pattern. This pattern was shown many years ago by Guy Fontaine et al (Hayashi 2010).

Brugada syndrome: Brugada syndrome diagnosis criteria

1. Absence of apparent structural heart disease: **We don't know**
2. Absence of drugs effects, electrolyte disturbance and CHD **We don't know Brugada phenocophy?**
3. Documented PVT/VF **We don't know**
4. Family history of SCD at <45 years in first-degree relatives **We don't know**
5. Type 1 ECG Brugada pattern (coved-type) in proband and family members Yes
6. Induction of VT/VF with Programmed Electrical Stimulation **We don't know**
7. Syncope, cardiac arrest or nocturnal agonal respiration**. We don't know**

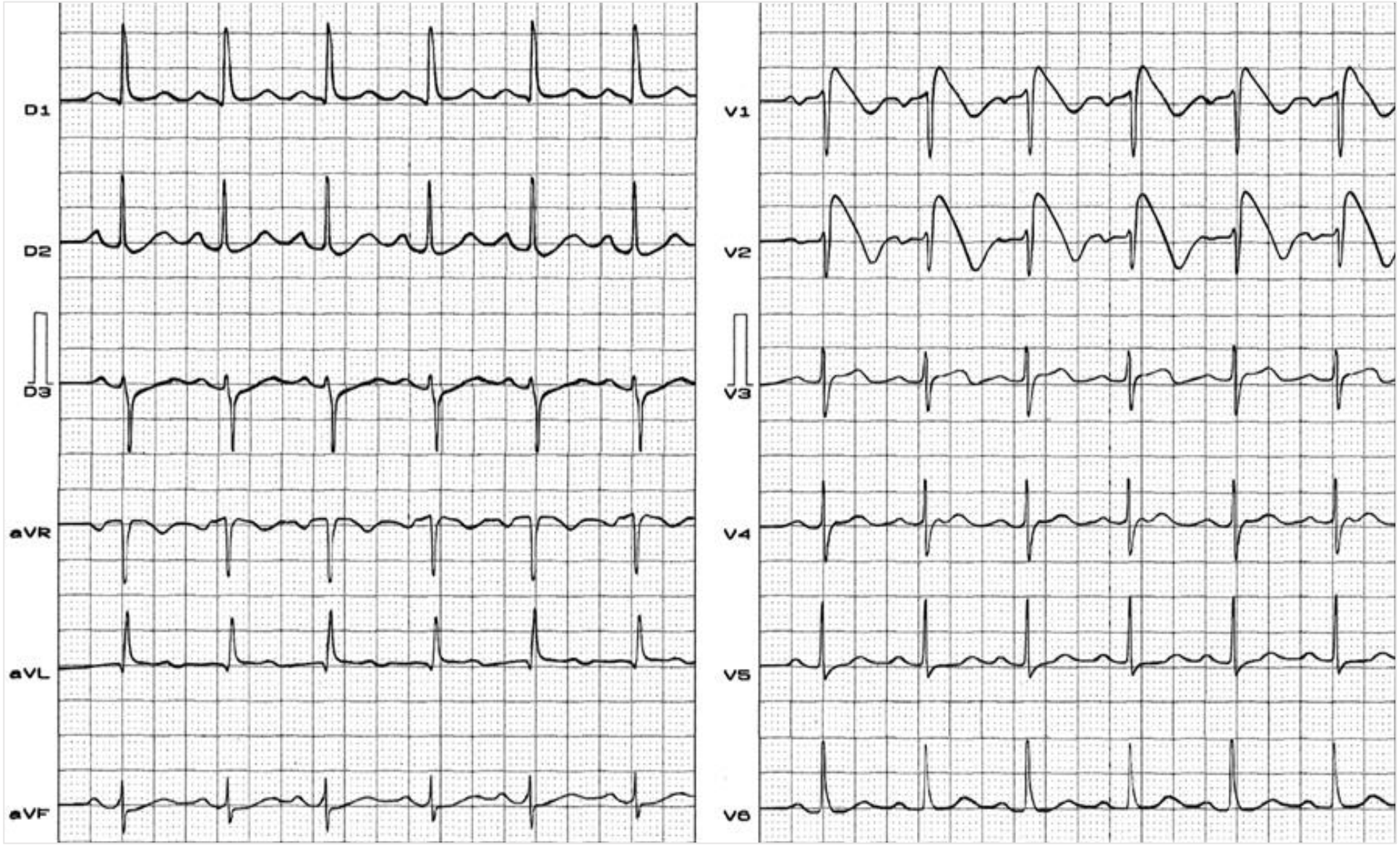
The diagnostic criteria for Brugada phenocopies are (I-V are mandatory) (Baranchuk 2012; Anselm 2013-2014):

An ECG pattern that has a type-1 or type-2 Brugada morphology

I. The patient has an underlying condition that is identifiable
II. The ECG pattern resolves upon resolution of the underlying condition
III. There is a low clinical pretest probability of true Brugada syndrome determined by a lack of symptoms, medical history, and family history
IV. The results of provocative testing with a sodium channel blocker such as ajmaline, flecainide, or procainamide are negative
V. Provocative testing is not mandatory if surgical RVOT manipulation has occurred within the last 96 hours.
VI. The results of genetic testing are negative (desirable but not mandatory because the SCN5A mutation is identifiable in only 20% to 30% of probands affected by true BrS).
VII. Correction of hypokalemia.

1. Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada R, Corrado D, Gussak I, LeMarec H, Nademanee K, Perez Riera AR, Shimizu W, Schulze-Bahr E, Tan H, Wilde A.Brugada syndrome: report of the second consensus conference. Heart Rhythm. 2005 Apr;2(4):429-40. Review. Erratum in: Heart Rhythm. 2005 Aug;2(8):905.
2. Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada R, Corrado D, Gussak I, LeMarec H, Nademanee K, Perez Riera AR, Shimizu W, Schulze-Bahr E, Tan H, Wilde A.Brugada syndrome: report of the second consensus conference: endorsed by the Heart Rhythm Society and the European Heart Rhythm Association.Circulation. 2005 Feb 8;111(5):659-70

Name: SJC; **Sex**: M; **Age**: 32 y/o; **Ethnic group**: Caucasian; **Weight**: 82 Kg; **Height**: 1.76 m;
Date: 09/01/2001



Clinical diagnosis: Symptomatic young man with Brugada syndrome.
ECG diagnosis: Type 1 Brugada pattern without RBBB.