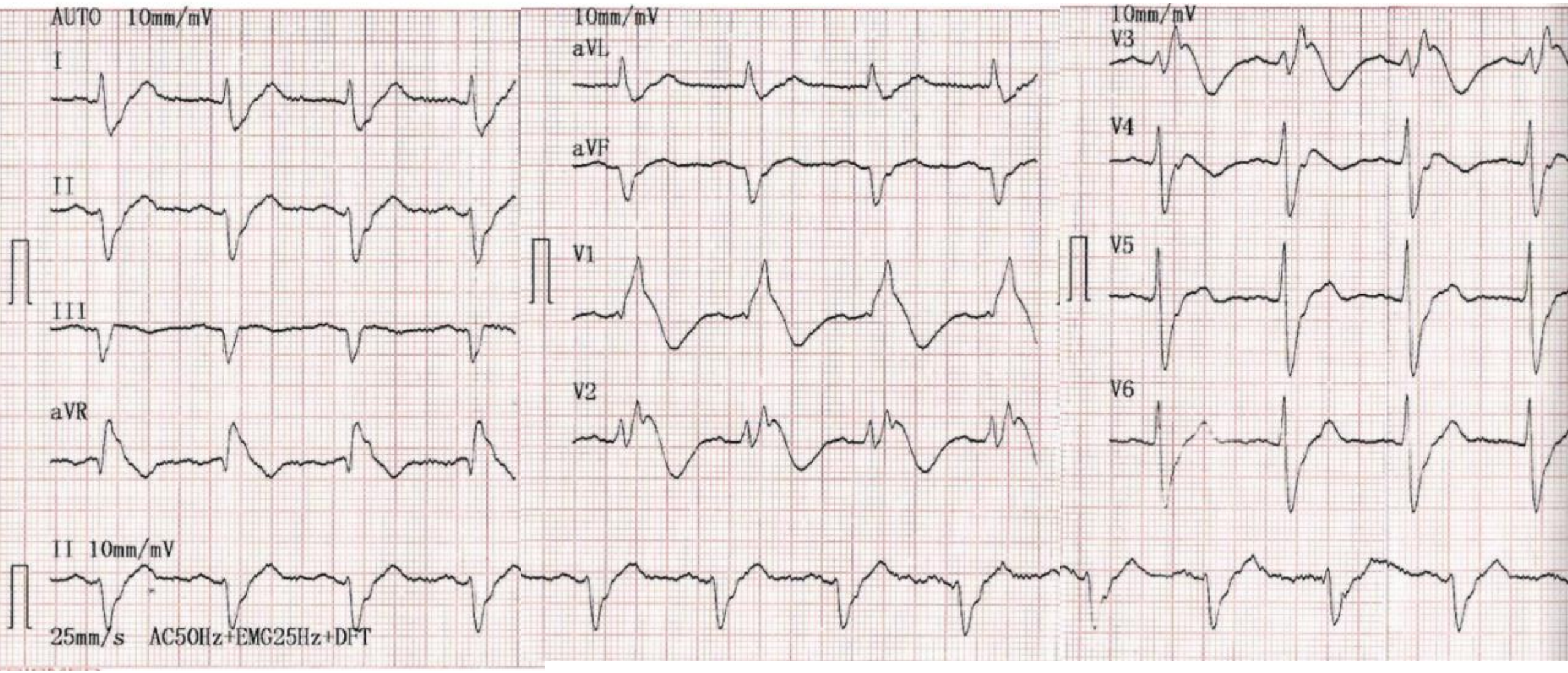
Dr, Solon Navarrete´s Case report

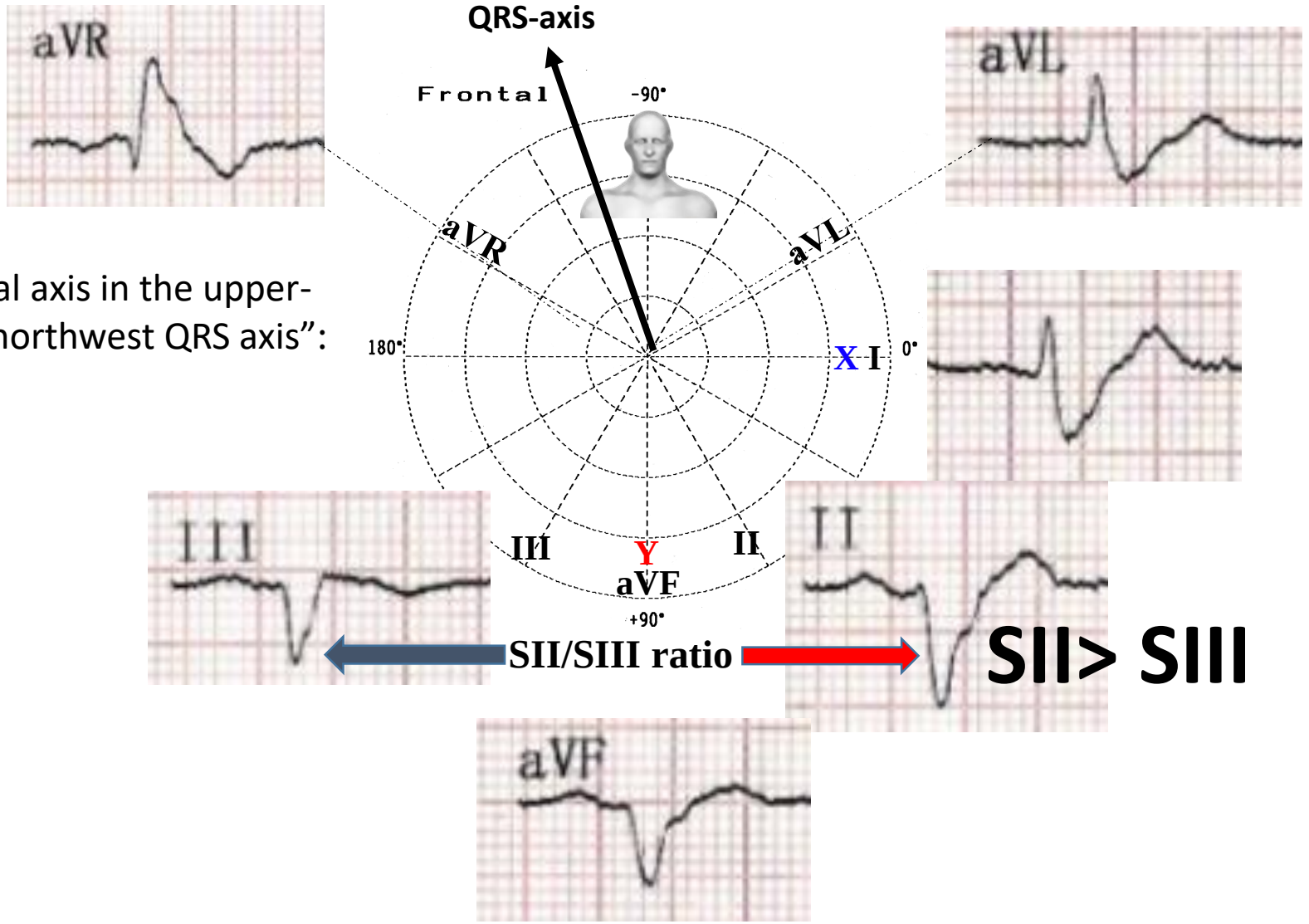
Estimados amigos y colegas buenos días. Me gustaría saber sus opiniones de este EKG de un paciente masculino de 53 años derivado a valoración por cardiología por un aparente episodio sincopal estudiado previamente por dolor torácico al que le realizaron estudio de perfusión miocárdica reportado como normal y ecocardiograma también normal. Antecedente familiar padre fallecido de “un problema cardíaco” el cual desconoce con precisión el diagnóstico a la edad de 60 años.

Dear friends and colleagues. I would like to know your opinions about this ECG of a 53-year-old male patient referred to cardiology evaluation for an apparent syncope episode previously studied for chest pain who underwent a myocardial perfusion study reported as normal and echocardiogram also normal. Family history of a deceased father of "a heart problem" which the diagnosis is not precisely known at the age of 60 years.

Solon Navarrete Case report



The QRS electrical axis in the upper-right quadrant “northwest QRS axis”:

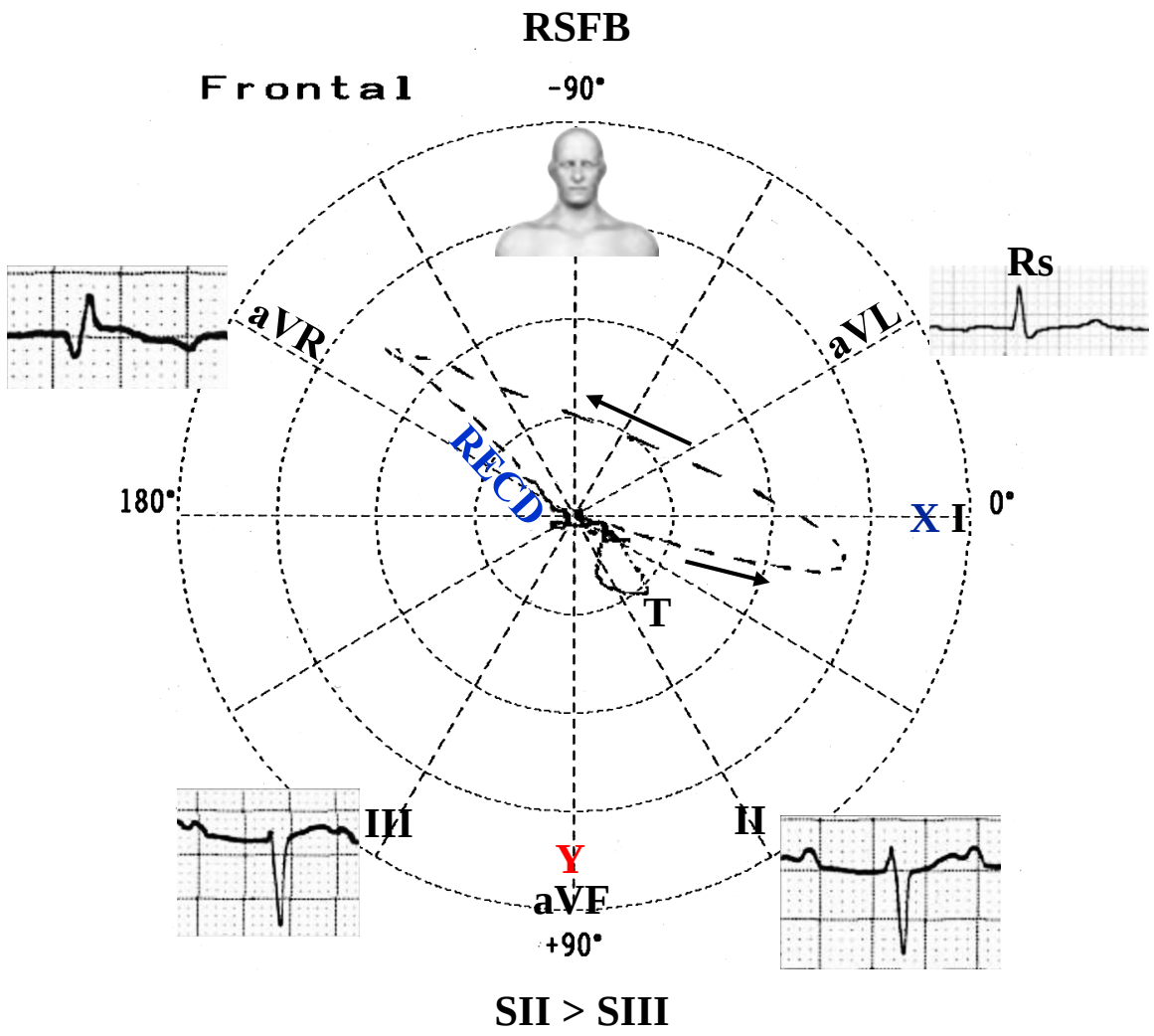


1. “**Norwest QRS axis”** in the Brugada Syndrome it is potential marker to predict poor outcome. This it the clinical significance of the QRS electrical axis in the upper-right quadrant “northwest QRS axis”: https://www.sciencedirect.com/science/article/pii/S2666084920309670

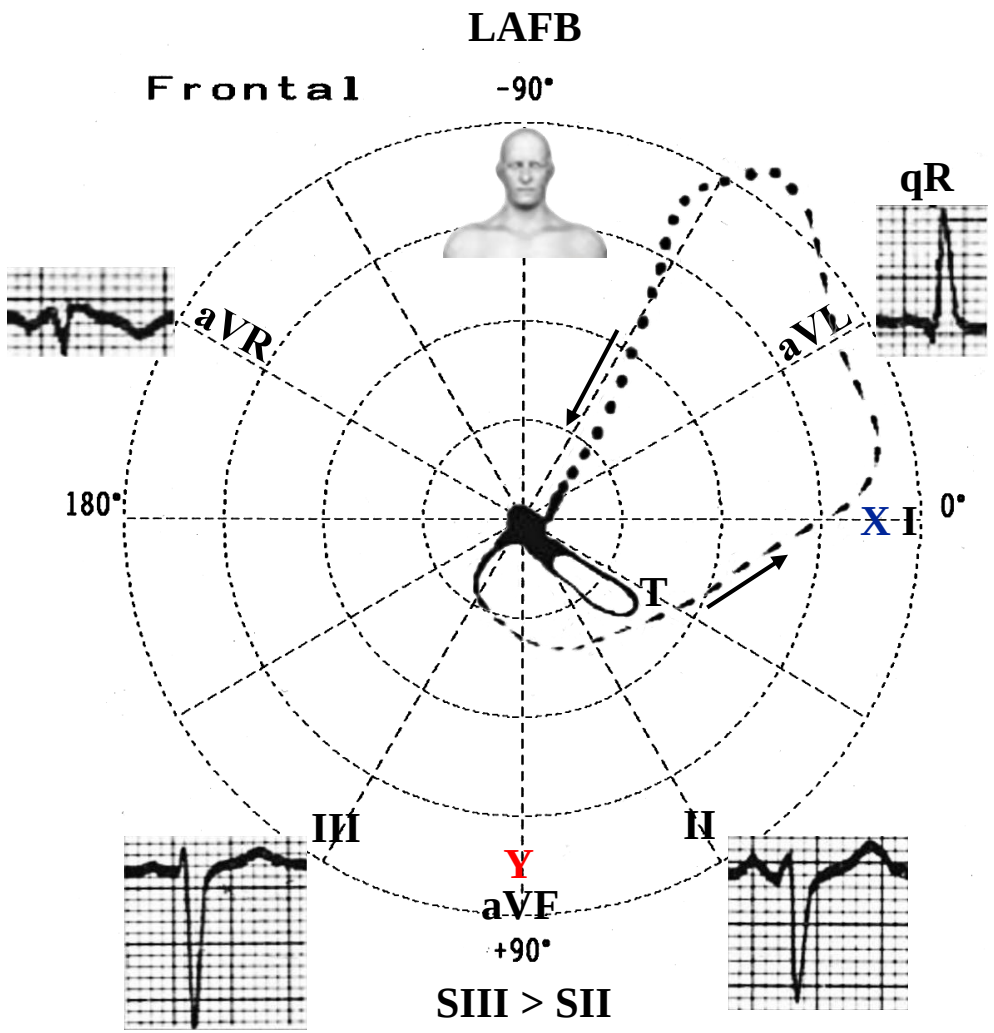
Possible Clinical Scenarios in Which Right-Axis Deviation Can Be Observed

Clinical Scenario	***Details***
ECG lead misplacement	*Reversal of right arm and left leg cables*
Altered position of the heart in the chest	*Dextrocardia*
Ventricular rhythm	*Ventricular tachycardia* *Accelerated idioventricular rhythm* *Ventricular escape rhythm*
Severe hyperkalemia	
Drug intoxication	*Tricyclic antidepressant, sodium channel blocker*
Pulmonary emphysema	*"Type C" right ventricular hypertrophy*
*Right superior fascicular block**	Please see the next two slides

ECG/VCG differential diagnosis between right superior fascicular block (RSFB) and left anterior fascicular block (LAFB) (Pérez-Riera AR 2005)

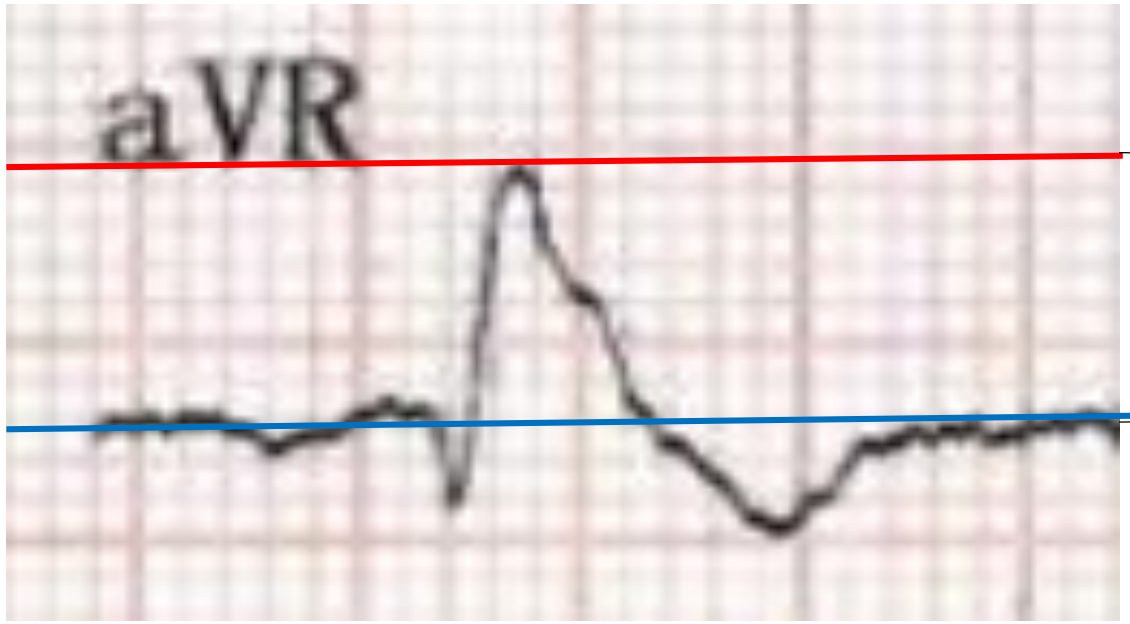


SII > SIII



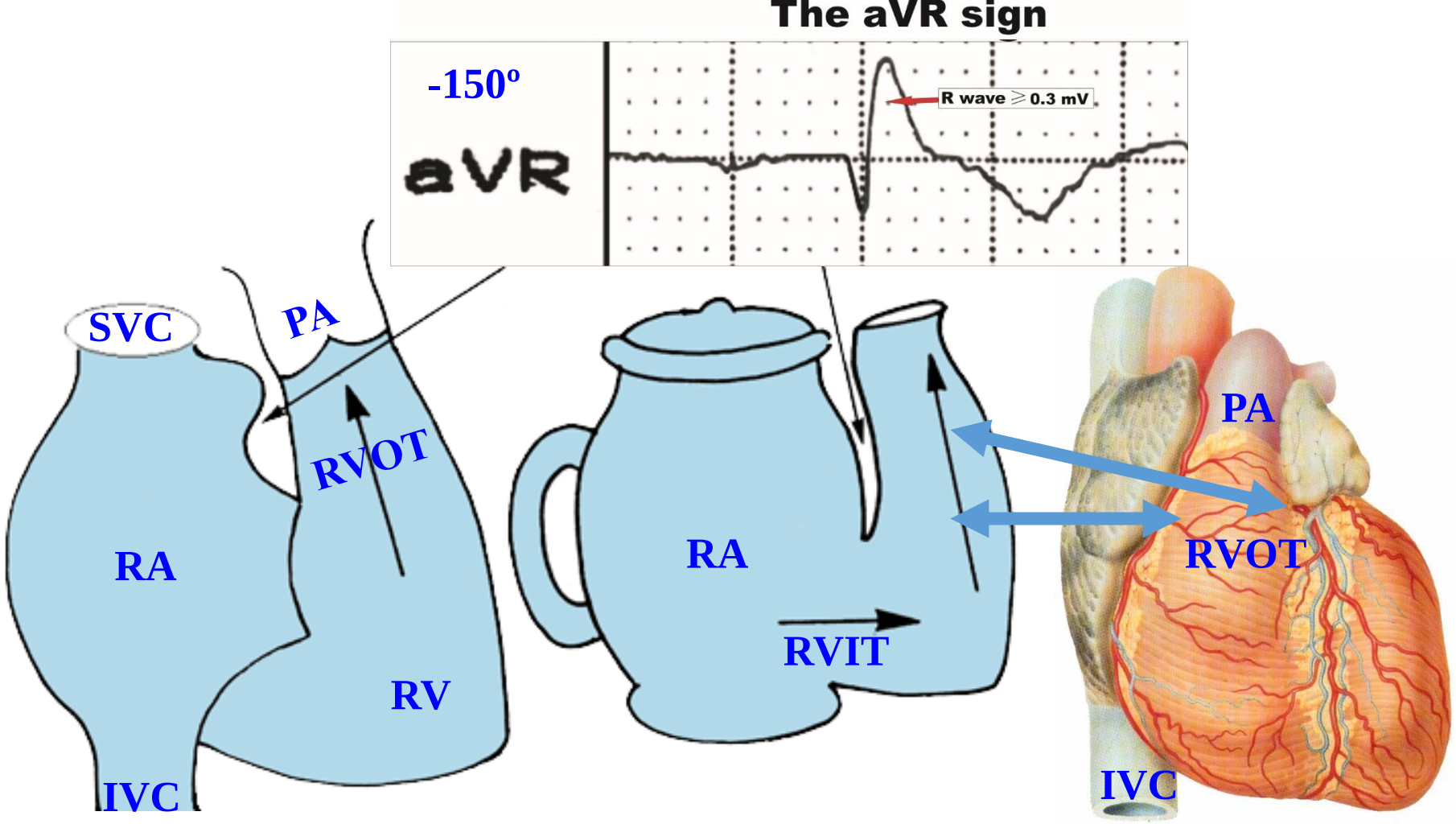
SIII > SII

	LAFB	RSFB
Initial 10 ms vector of QRS loop	Heading downward and to the right	Heading downward and to the left
QRS morphology in I & aVL	qR pattern	Rs
SII/SIII ratio	SIII>SII	SII>SIII
Location of end conduction delay (ECD)	In the left superior quadrant when present	In the right superior quadrant (Pastore 1983)
Prominent R wave in aVR (R-wave ≥ 0.3 mV)	Absent	It could be present and it is called aVR sign (Babai Bigi 2007).
Morphology of QRS loop of vectorcardiogram in the horizontal plane	Similar to normal	Similar to type-C right enlargement pattern: initial vector to the front and leftward, counterclockwise rotation and 20% or more of the area of the loop located in the right posterior quadrant in the horizontal plane (Luna Filho 1989)

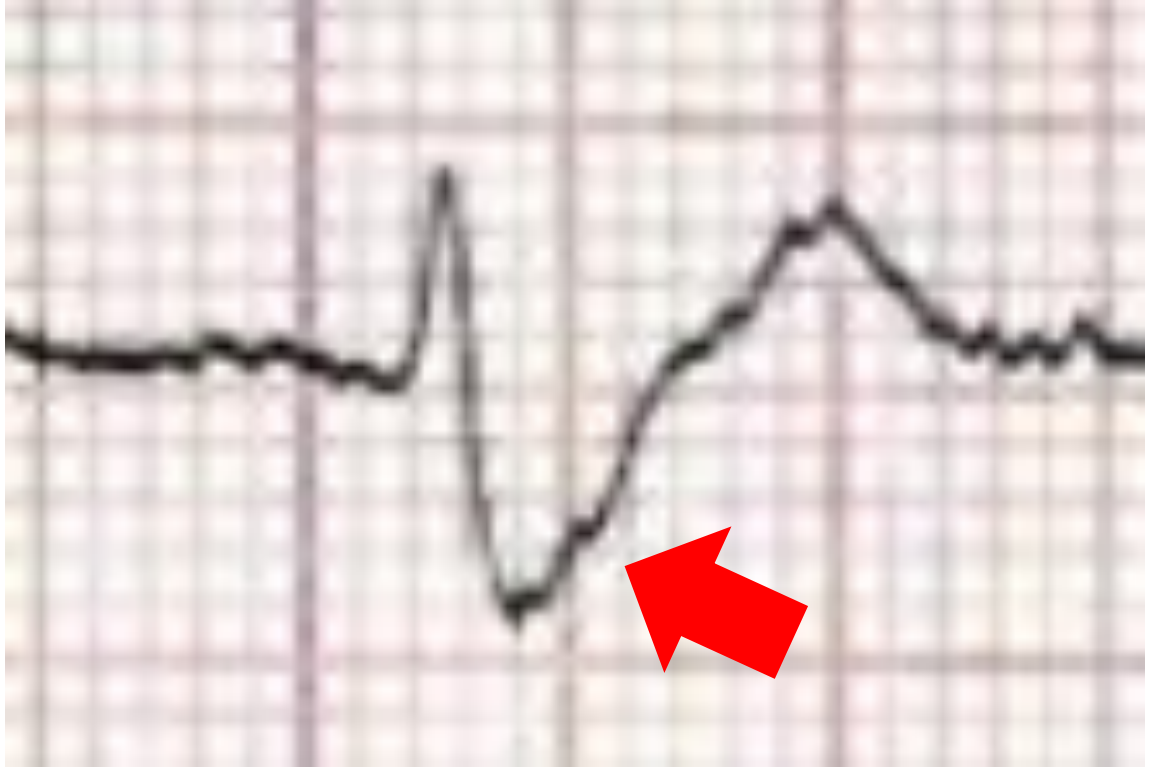


Final R wave on aVR lead; R wave **6mm!!!!**

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).



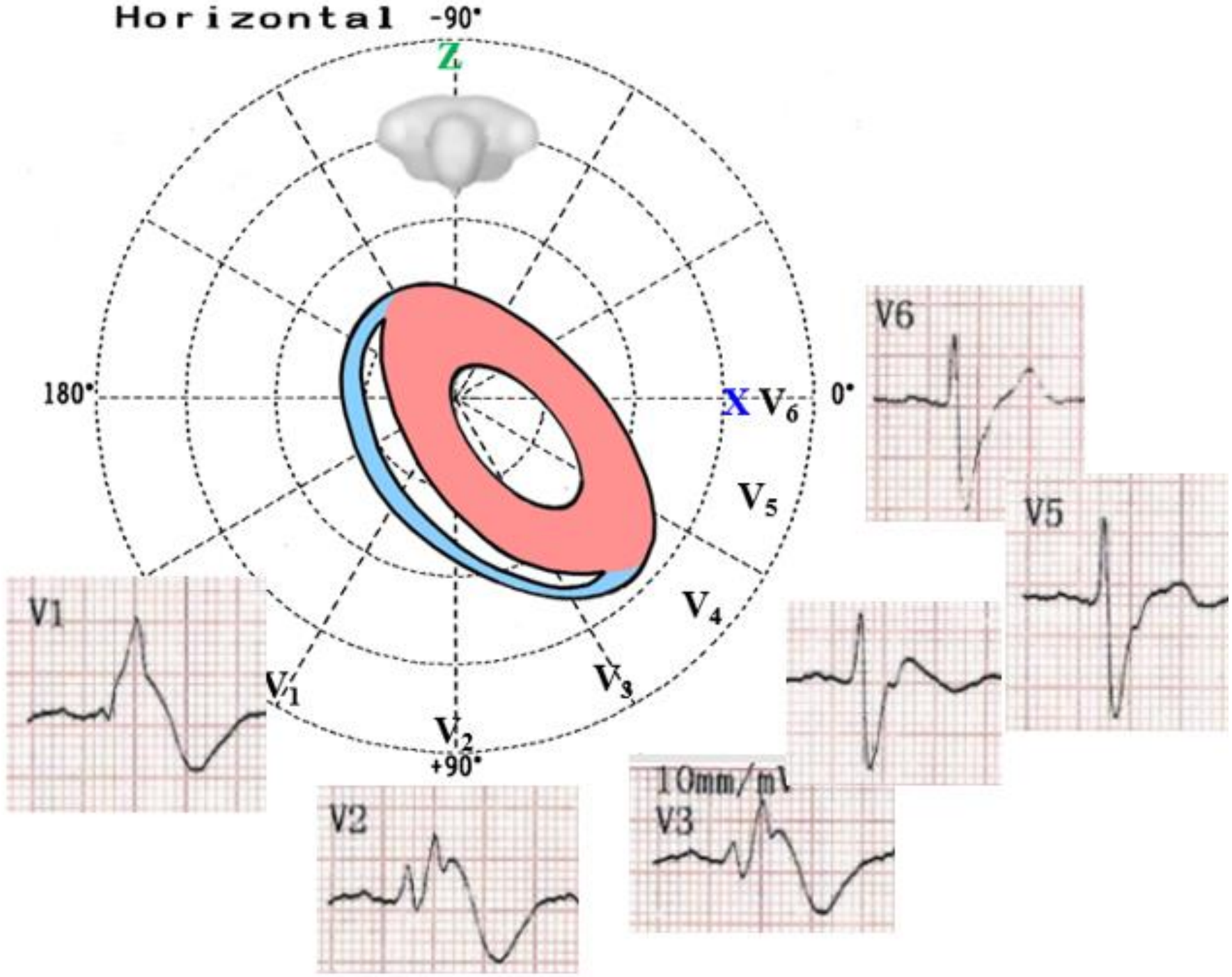
Leonardo Calò

Wide and/or large S wave in lead I: **Calò Sign**

Presence of a wide and large S-wave in lead I is a powerful predictor of life-threatening ventricular arrhythmias in BrS patients. Calò and colleagues note that “The so-called third vector, which is directed upward and somewhat to the right and backward, generates the S-wave in lead I. This vector is determined by electrical activation of the basal region of both ventricles and by depolarisation of the RVOT. A prominent S-wave in lead I is typically present in cases of congenital heart disease, valvular heart disease, and *cor pulmonale* that cause right ventricular enlargement and fibrosis. Thus, we hypothesised that a deep and/ or large S-wave in lead I in Brugada Syndrome would reveal a conduction delay over the RVOT and could be used to identify high-risk patients.”

The study analysed data from 347 consecutive patients (78.4% male; mean age 45±13.1 years) at four Italian centres with spontaneous type I

BrS by ECG parameters but with no history of cardiac arrest (including 91.1% asymptomatic at presentation, 5.2% with a history of atrial fibrillation, and 4% with a history of arrhythmic syncope). Electrocardiographic characteristics at the first clinic visit were analysed to predict VF or SCDduring follow-up. During follow-up (48±38 months), 276 (79.5%) patients remained asymptomatic, 39 (11.2%) developed syncope, and 32 (9.2%) developed ventricular fibrillation or sudden cardiac death," the authors report. Patients who developed ventricular fibrillation or sudden cardiac death had a lower prevalence of SCN5A gene mutations and a higher prevalence of positive electrophysiological study results ($p<0.0001$), a family history of sudden cardiac death ($p=0.03$), and atrial fibrillation. The most powerful marker for VF or SCD was a significant S-wave (≥0.1mV and/or ≥40ms) in lead I," the authors write. "In the multivariate analysis, the duration of S-wave in lead I ≥40ms and AF were independent predictors of VF or SCD during follow-up," they continue. Electroanatomic mapping in 12 patients showed an endocardial activation time "significantly longer" in patients with an S-wave in lead I, which Calò *et al* attribute to *"*a significant delay in the anterolateral RVOT". Speaking about the relevance of these findings in clinical practice, Calò told *Cardiac Rhythm News*: "The presence of a wide and/or large S wave in lead I in BrS, expression of a delayed activation in the RVOT, can be used as a potential novel marker of SCD risk stratification. But the most important finding of this study is that the absence of S wave in Brugada type can reassure physician and patient." In an accompanying editorial comment, Arthur Wilde, University of Amsterdam, Amsterdam, The Netherlands, and Pieter G Postema, Princess Al-Jawhara Al-Brahim Centre of Excellence in Research of Hereditary Disorders, Jeddah, Saudi Arabia, write that "the study by Caló *et al* suggests the prognostic value of ECG parameters of terminal right ventricular conduction delay in so far asymptomatic BrS patients with a spontaneous type I electrocardiogram. In particular, the absence of a deep and wide S-wave in lead I is a potentially reassuring parameter with a very high negative predictive value that should make one even more reluctant to continue with primary prevention implantable cardioverter-defibrillator implantation."


Horizontal
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Z
180°
X
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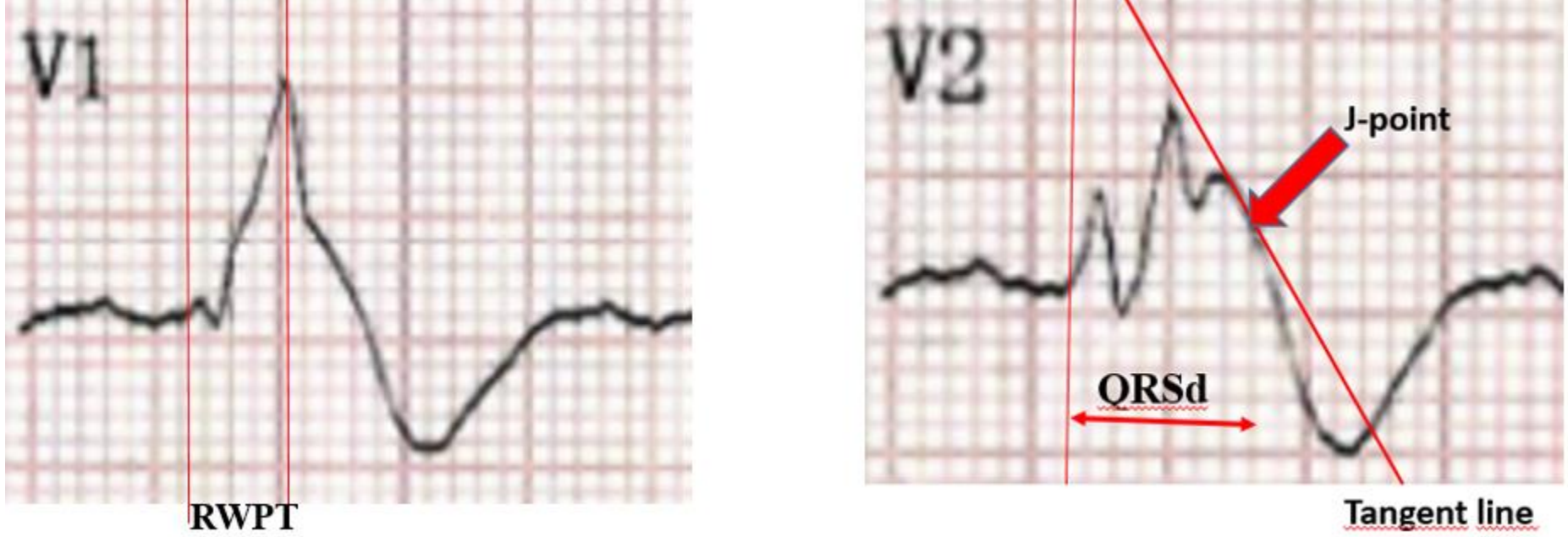
V2
+90°
V6
V5
V1
V2
10mm/mV
V3

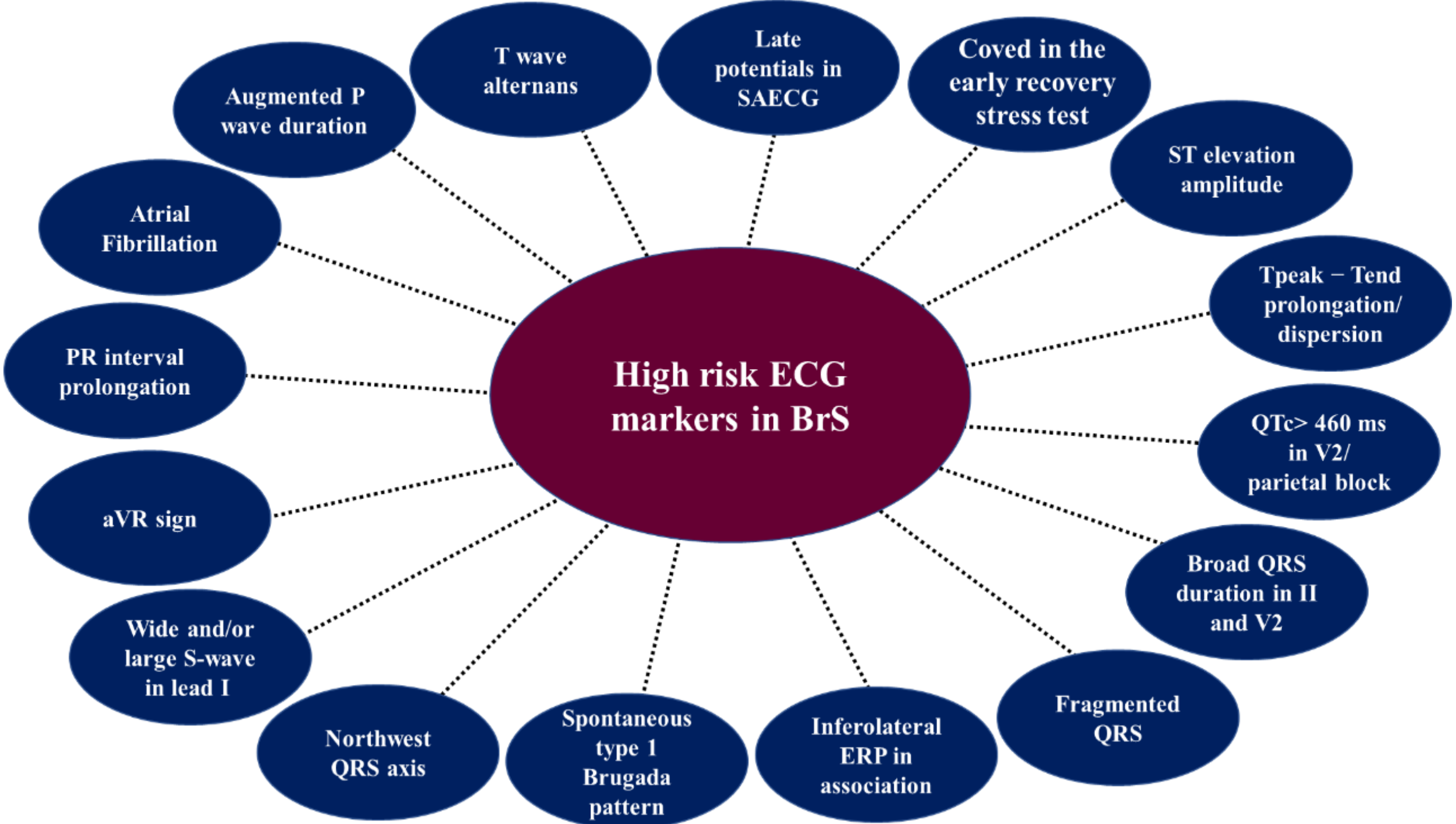


I. *Prolonged QRS duration (**QRSd**) measured from lead II or V2 ≥120 ms.[37] Prolonged QRS duration as measured on a standard 12-lead ECG is associated with VA and could serve as a simple noninvasive marker of vulnerability to life-threatening cardiac events in patients with BrS.[38] The duration of the QRS in leads V1 and V2 is greater than in the middle and left precordial leads.* ***Peters S. Low amplitude ECG and QRS fragmentation in provocable coved-type ST-segment elevation on surface ECG are strong predictors of a continuum between arrhythmogenic cardiomypathy and Brugada syndrome. Int J Cardiol. 2016;214:148-150.***

II. *Extremely prolonged R-Wave Peak Time (**RWPT**) or Ventricular Activation Time (It is not recommended intrinsic deflextion nomenclature) It is indicative of severe dormotropic disturbance in RVPT*

III. *Spontaneous Type 1 Brugada pattern history of syncope, ventricular effective refractory period <200 ms, and fQRS seem useful to identify candidates for prophylactic ICD.(**Priori SG, Gasparini M, Napolitano C, et al. Risk stratification in Brugada syndrome: results of the PRELUDE (PRogrammed ELectrical stimUlation preDictive valuE) registry. J Am Coll Cardiol. 2012;59:37-45**.)*

IV. QRS fragmentation, or fQRS: defined as ≥ 2 notches of the R wave or in the nadir of the S wave in at least two consecutive leads. fQRS is a powerful depolarization marker for VF/SCD. **Priori SG, Gasparini M, Napolitano C*, et al.* Risk stratification in Brugada syndrome: results of the PRELUDE (PRogrammed ELectrical stimUlation preDictive valuE) registry. J Am Coll Cardiol. 2012;59:37-45. Morita H, Kusano KF, Miura D*, et al.* Fragmented QRS as a marker of conduction abnormality and a predictor of prognosis of Brugada syndrome. Circulation. 2008;118:1697-1704.**


High risk ECG markers in BrS
Augmented P wave duration
T wave alternans
Late potentials in SAECG
Coved in the early recovery stress test
ST elevation amplitude
Atrial Fibrillation
Tpeak − Tend prolongation/ dispersion
PR interval prolongation
QTc> 460 ms in V2/ parietal block
aVR sign
Broad QRS duration in II and V2
Wide and/or large S-wave in lead I
Fragmented QRS
Northwest QRS axis
Spontaneous type 1 Brugada pattern
Inferolateral ERP in association