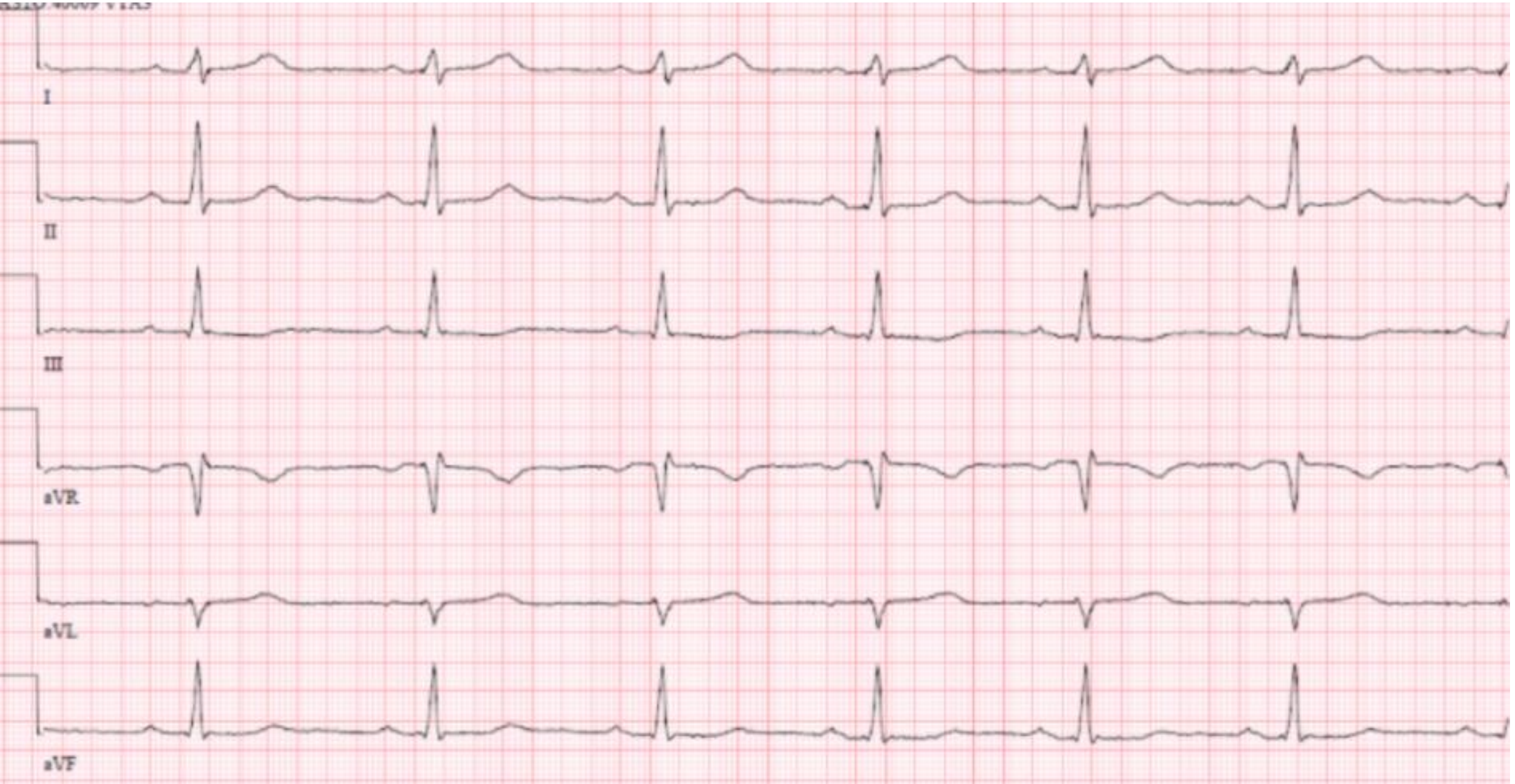
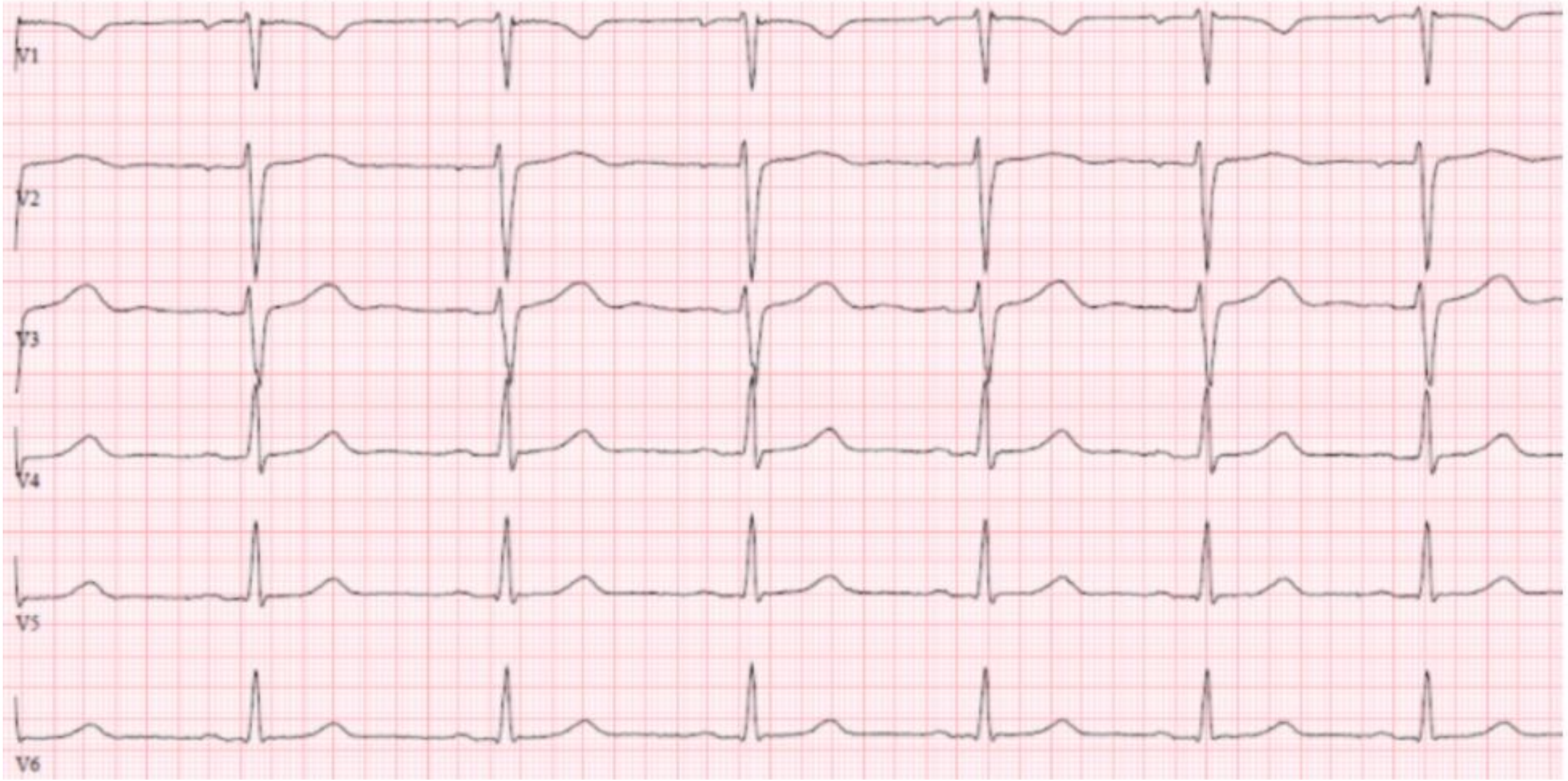
18 Years old woman No known heart disease Twice presyncope during exercise

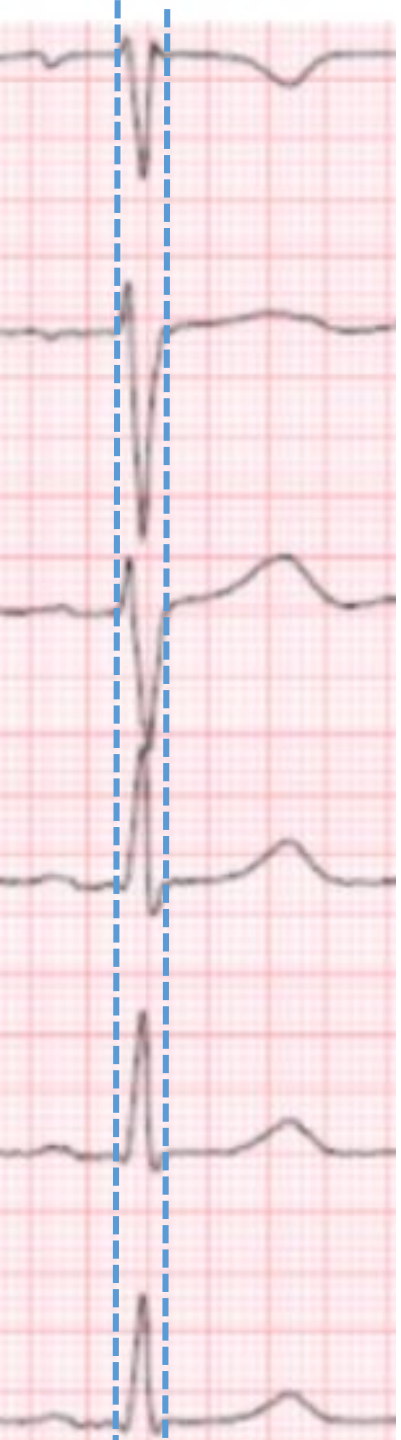
Number 1A Resting ECG 50 mm/sec frontal plane leads

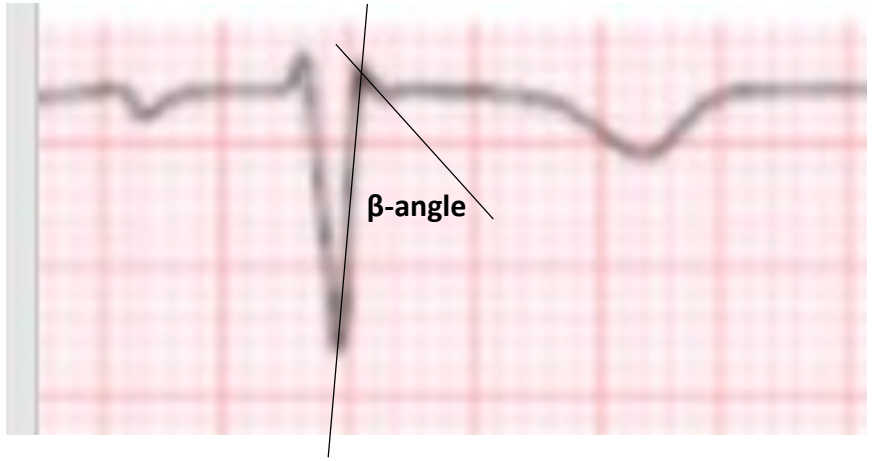


Resting ECG, 50 mm/sec, V1 and V2 negative (or biphasic) P wave – electrodes maybe placed a bit high?

Number 1B **Resting ECG 50 mm/sec Precordial plane leads**

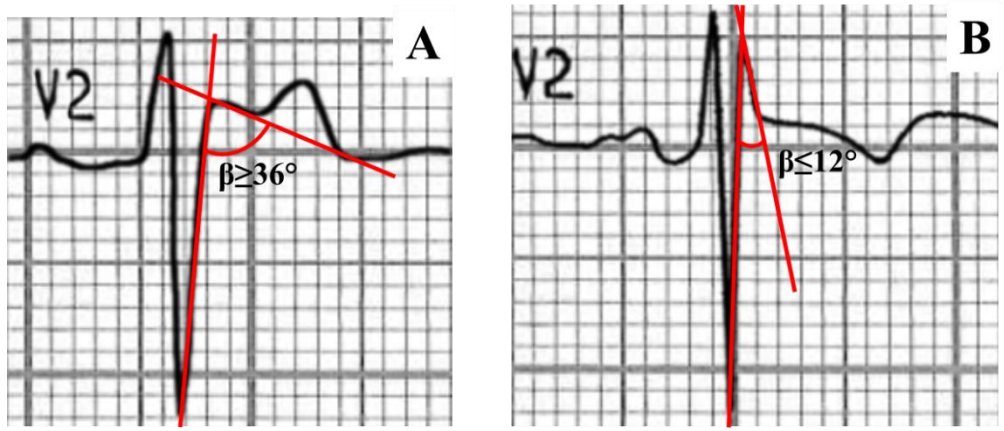






The present case:

Typical IRBBB pattern in the precordial leads V1. Note the r´ final wave with acute contour (“high take-off”) and narrow β-angle ≤36°. angle is formed by the ascending and descending ramps of r´/R final wave in V1-V2,



Type 2 Brugada pattern versus ordinary IRBBB.

A) Typical type 2 Brugada pattern in the precordial lead V2. Note the ST with saddleback appearance and followed by positive T wave in V2. The βangle formed by the ascending and descending ramp of the final r`/ R' wave with blunt contours and the so-called beta angle always is ≥36.8°.

B) Typical IRBBB pattern in the precordial leads V1-V2. Note the r´/R´ final wave with acute contour (“high take-off”) and narrow angle ≤12°. angle is formed by the ascending and descending ramps of r´/R final wave in V1-V2,

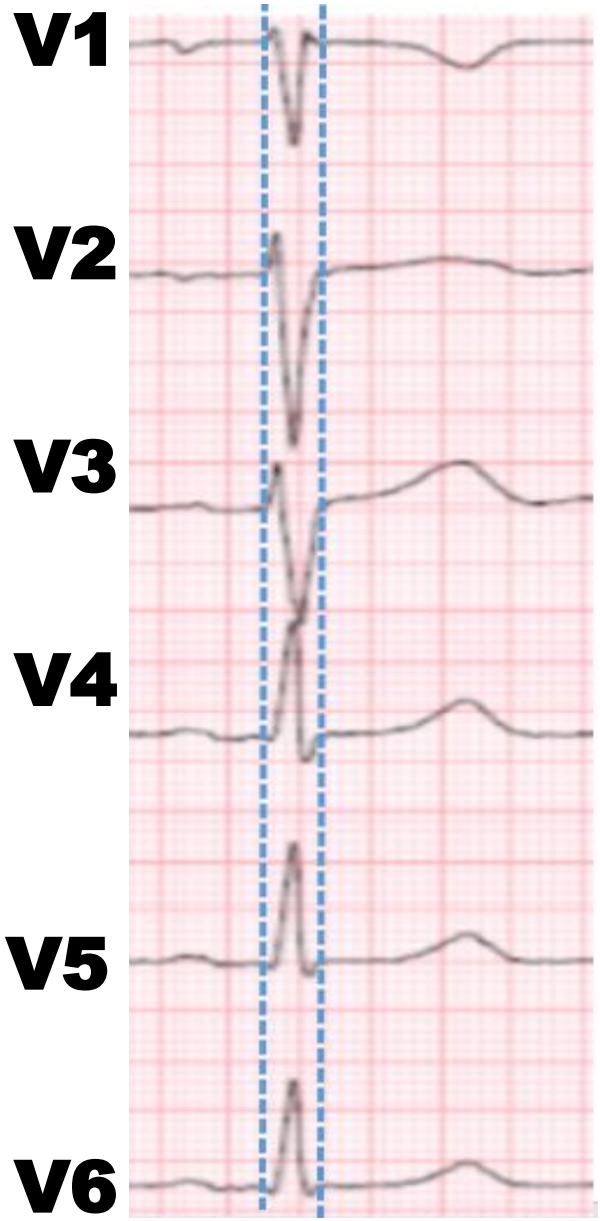
Table 1. Differential diagnosis between the type 2 Brugada pattern and "innocent" IRBBB. PPV: positive predictive value; NPV: negative predictive value.

	Type 2 Brugada pattern	"innocent" IRBBB
High take-off angle	**Approximately 36° blunt contours**	**Acute narrow β angle ≤12°**
β angle	≥36.8° (sensitivity: 86%, specificity: 94.7%, PPV: 93.5%, NPV: 88.5%)[5]	Minor ≤12°
ST segment	Bimodal with camel hump shape	ST depression convex upward followed by asymmetric negative T wave in the right precordial leads
T-wave	Positive or plane	Negative
Duration of the base of the triangle at 5 mm from the peak of r′ wave[6]	≥160 ms (4 mm)	Minimal
Duration of the base of the triangle at the isoelectric line from the peak of r' wave	≥60 ms (1.5 mm)	
High take-off contour	Wide/ blunt	Acute

Crea et al studied the inter-observer and intra-observer agreement in the diagnosis of type 2 BP in a cohort of 14 clinical cardiologists, 14 arrhythmologists and 14 electrophysiologists. They collected 14 ECGs with a triphasic QRS complex in lead V1-V2 at the 4th intercostal space. The authors proposed these ECGs, specifying to use 2012 Consensus conference criteria for diagnosis of type 2 BP,. The same 14 ECGs, with a different order, were proposed fifteen days later to the same cohort to assess intra-observer variability. Authors analyzed all 14 ECGs in order to assess whether or not 2012 Consensus Conference criteria for BP were fulfilled. All patients underwent provocative test with flecainide in order to exclude or confirm the diagnosis of Brugada Syndrome (BrS). Slight inter-observer agreement (Fleiss K<0.20) in the diagnosis of type 2 BP was observed in all three categories of cardiologists. Considering five operators per class, intra-observer agreement is variable (k ranging from 0,000 to 0,857), with a slight superiority of arrhytmologists (k minimum value 0,276; k maximum value 0,857). The authors verified a low inter-observer agreement in diagnosis of type 2 BP in categories of cardiologists with different abilities. Reproducibility of type 2 BP diagnosis (intra-observer agreement) is poor, even among experts. These findings show that the mere isolated ECG analysis is not enough, making personal and family questioning data essential.1, 2

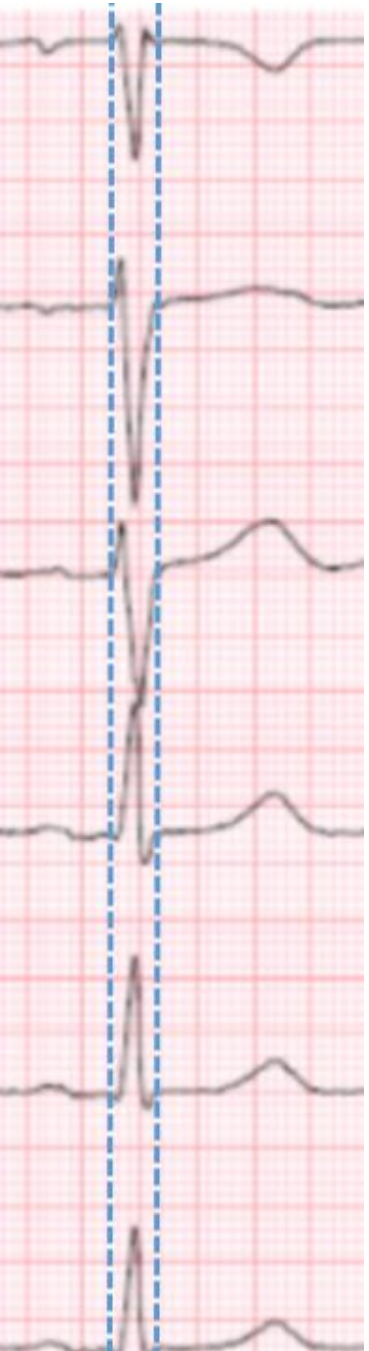
1. **Crea P, Rivetti L, Bitto R, *et al.* Diagnosis of type 2 Brugada pattern: insights from a pilot survey. Minerva Cardioangiol. 2020.**
2. **Sciarra L, Moriya M, Robles AG, *et al.* Type 2 Brugada pattern: more doubts than certainties. Minerva Cardioangiol. 2020.**

The present case
Number 1B
Resting ECG 50
mm/sec
Precordial leads leads

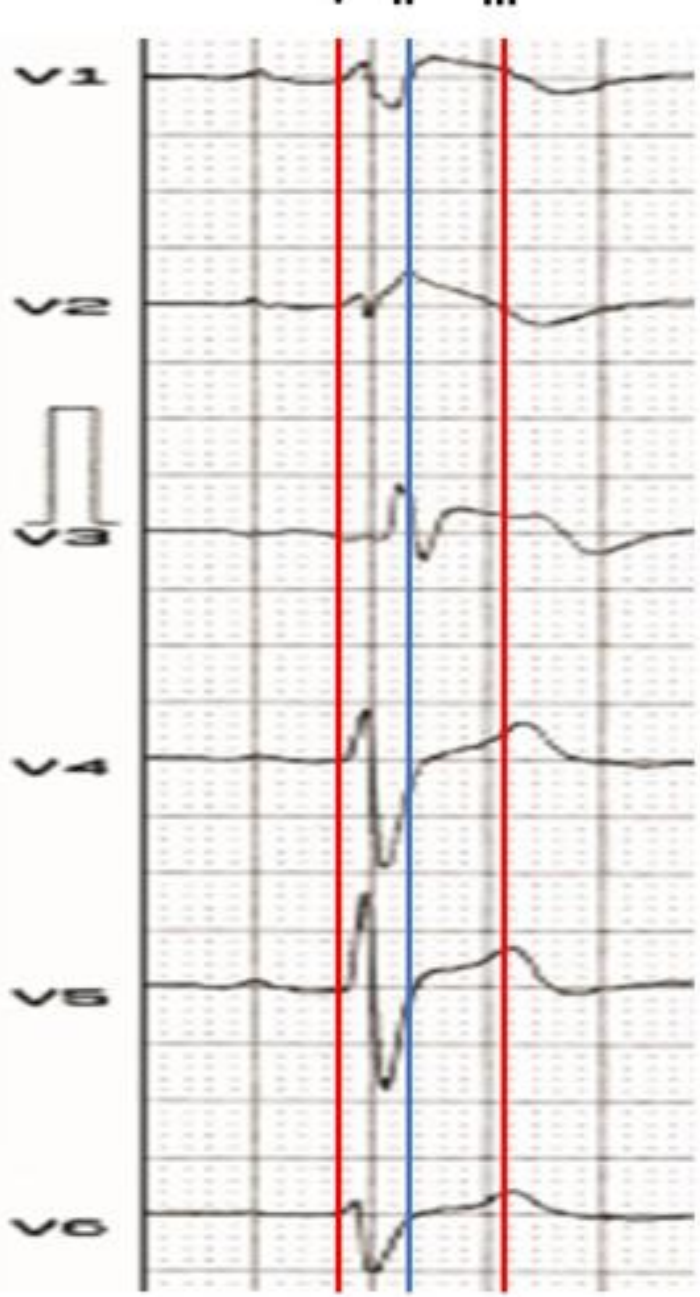


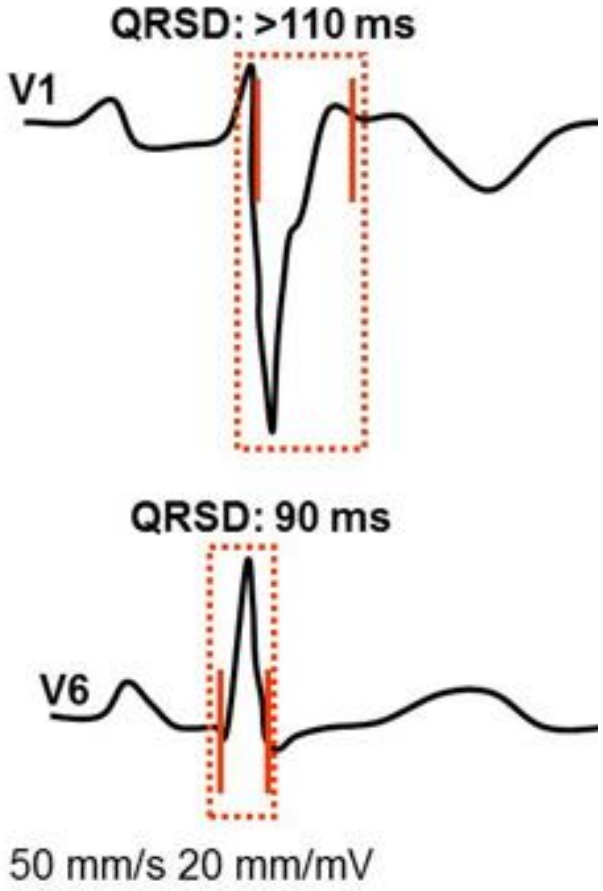
. QRS Duration (QRSd) of $^{V1+V2+V3}/_{V4+V5+V6}$ = 1

A) The present case



B) Brugada syndrome

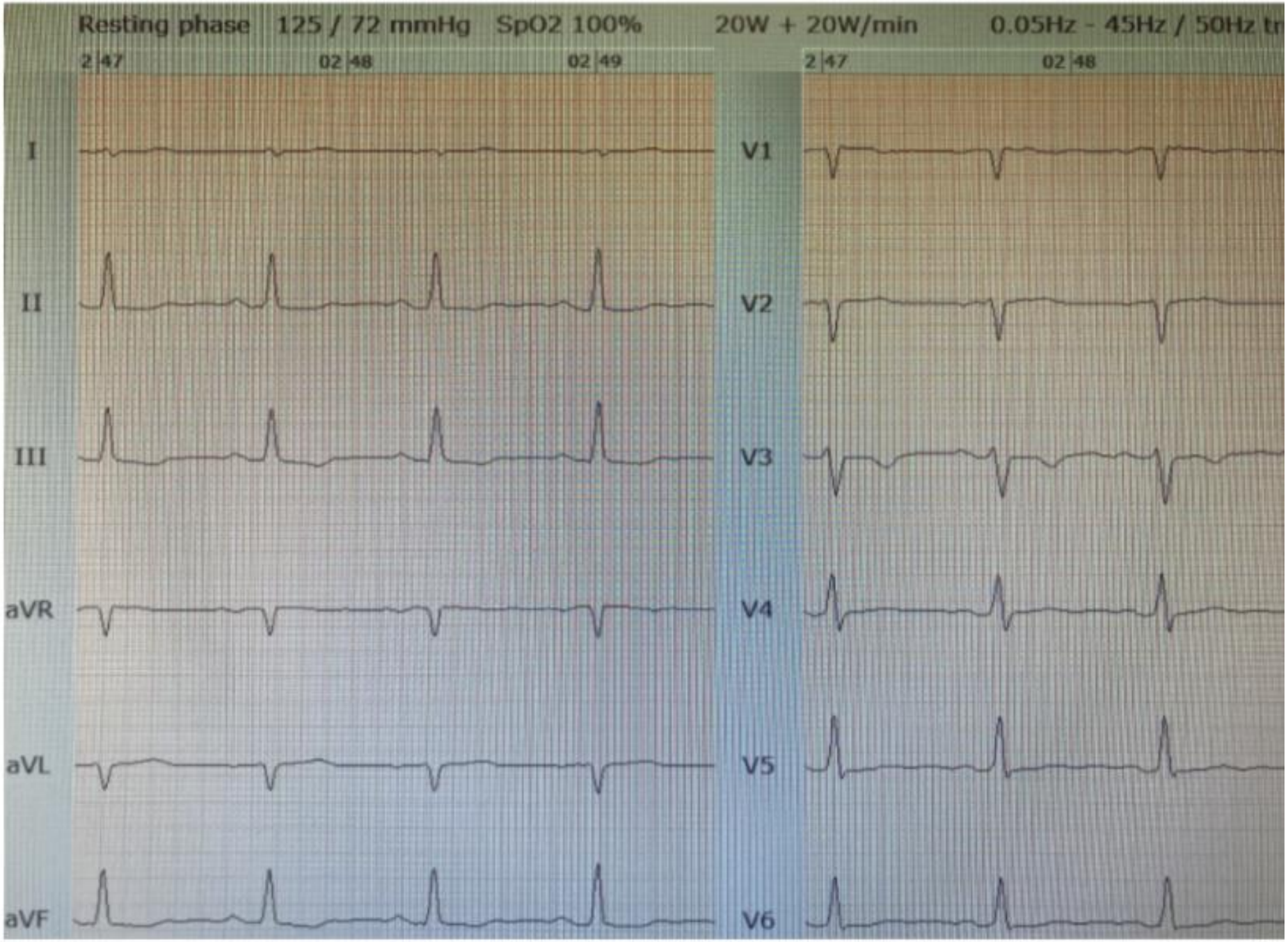




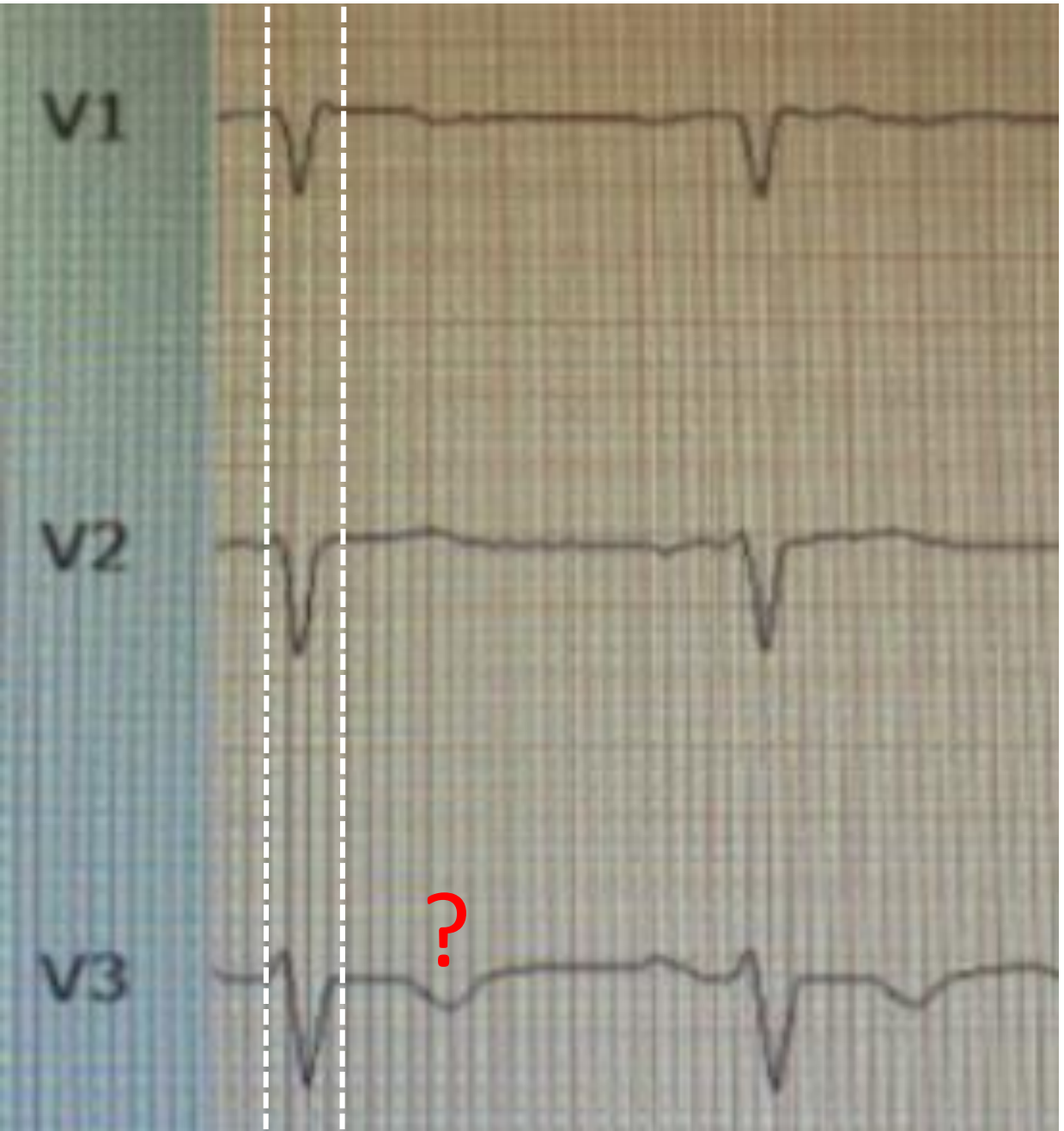
In BrS (B) Increase in QRS complex duration (>110º) in right precordial leads, in absence of CRBBB: **parietal block**; I: Onset of the QRS complex; II: Termination of the QRS complex from V4-V6; III: Termination of the QRS complex from V1-V3. QRSD of V1+V2+V3 /V4,V5, V6 > 1.2. This feature is considered typical of ARVC/D, but it is also observed in BrS leads . Pitzalis et al 30identified the selective prolongation of QT interval duration in the right precordial leads (V1 to V3) 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 (1)

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;42:1632-1637.**

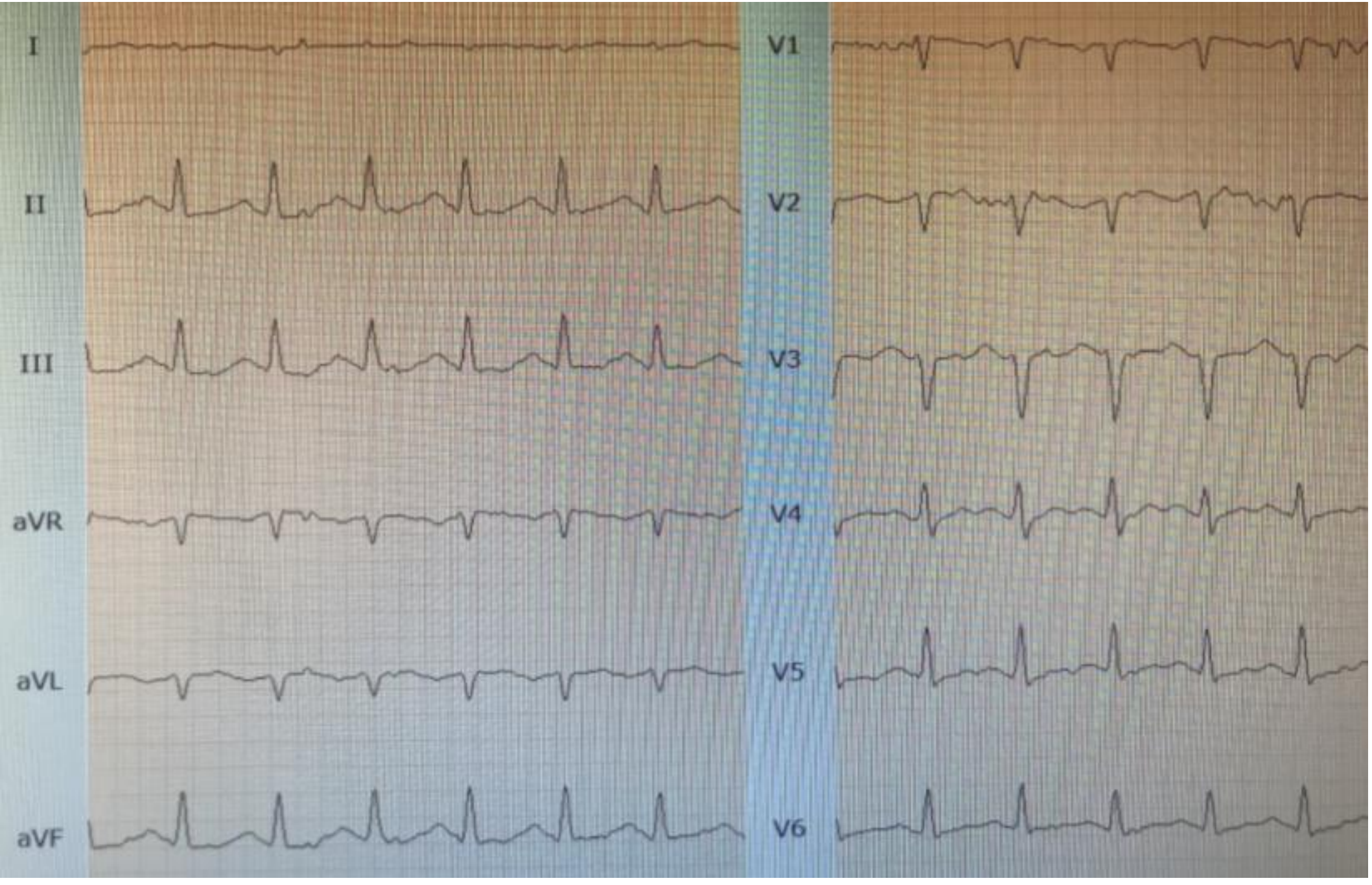
Number 2A ECG Before start of exercise, Mason-Likar lead system



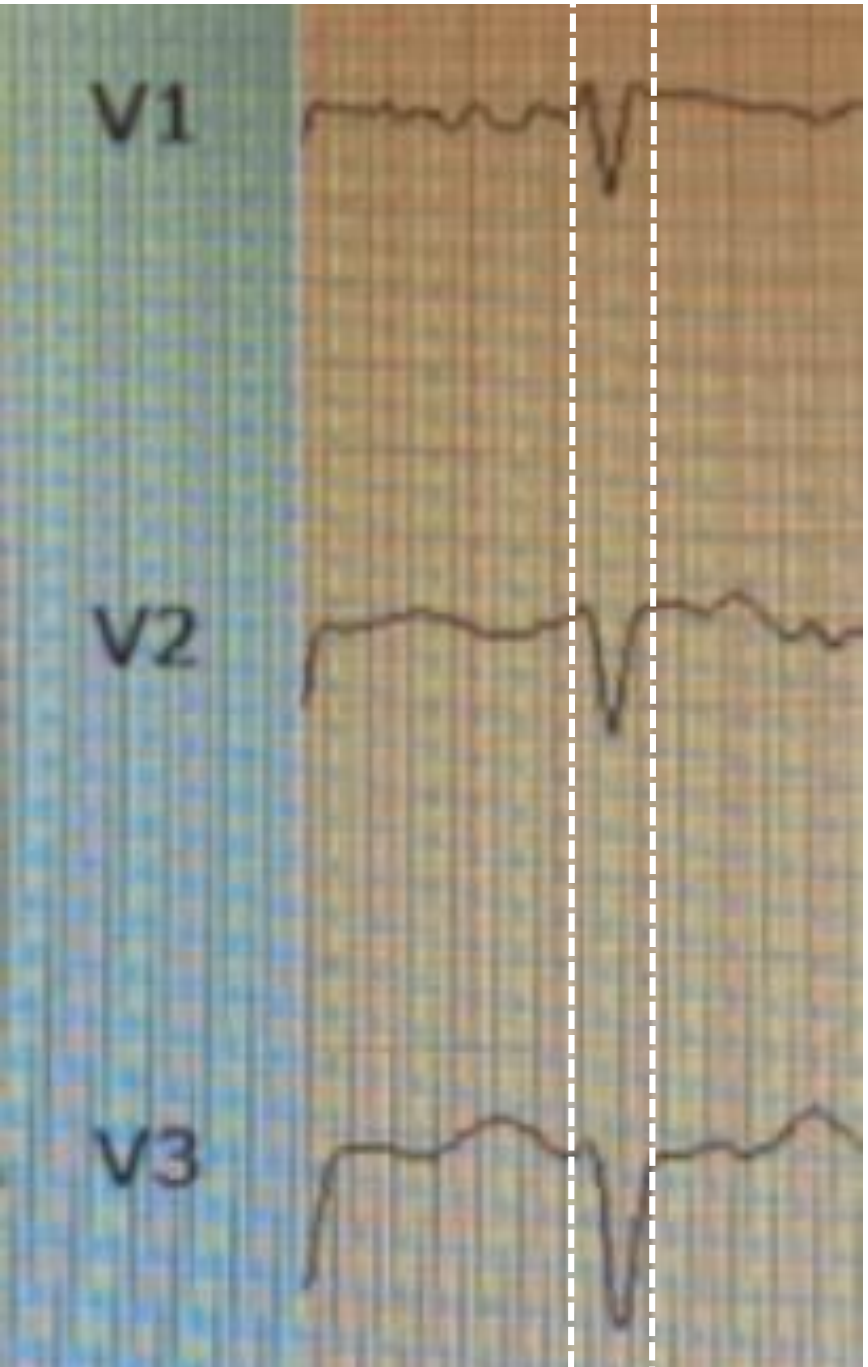
Number 2B ECG only rigt precordial leads Before start of exercise, Mason-Likar lead system



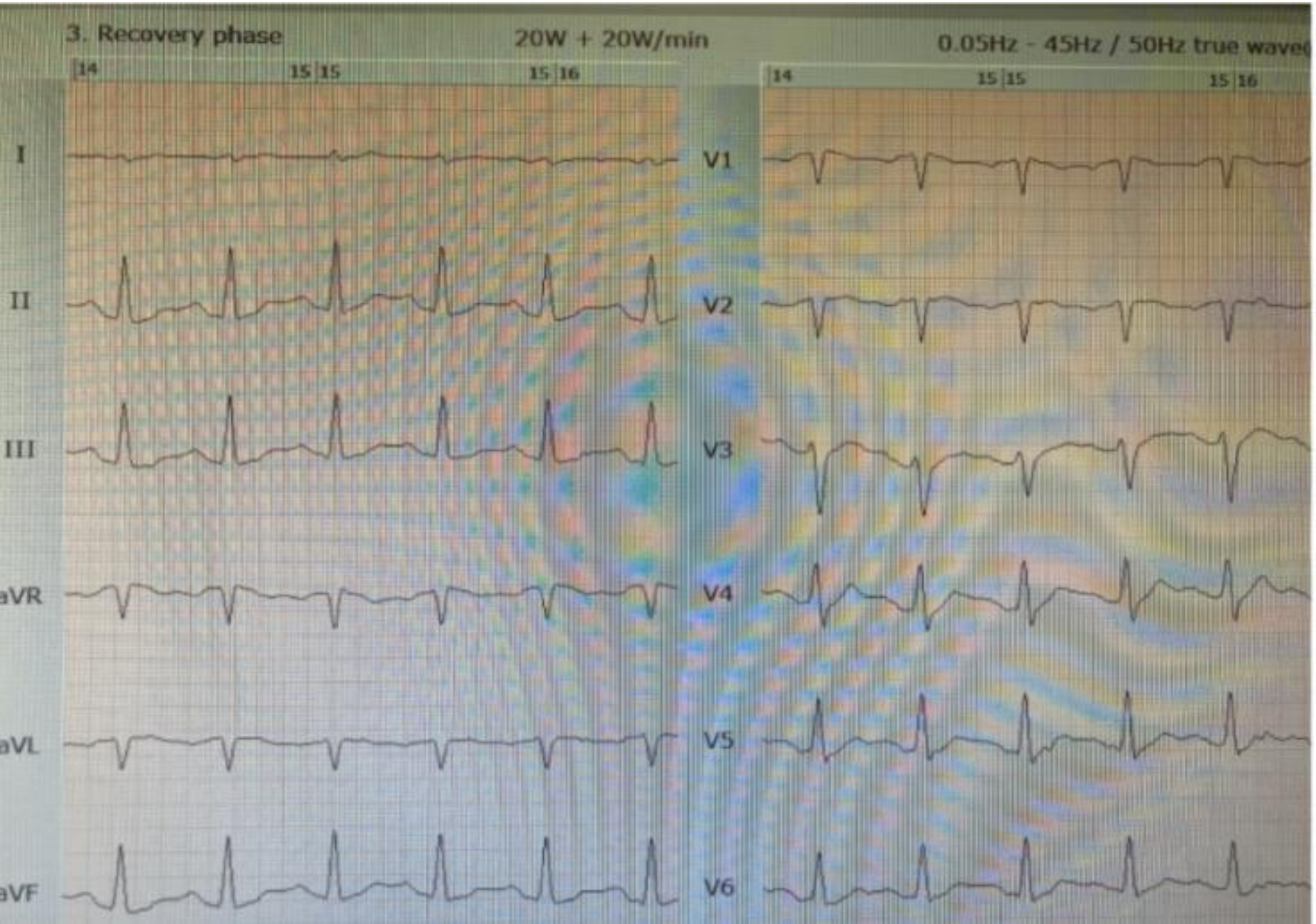
Number 3A During exercise test, 100 watt, heart rate 160/min



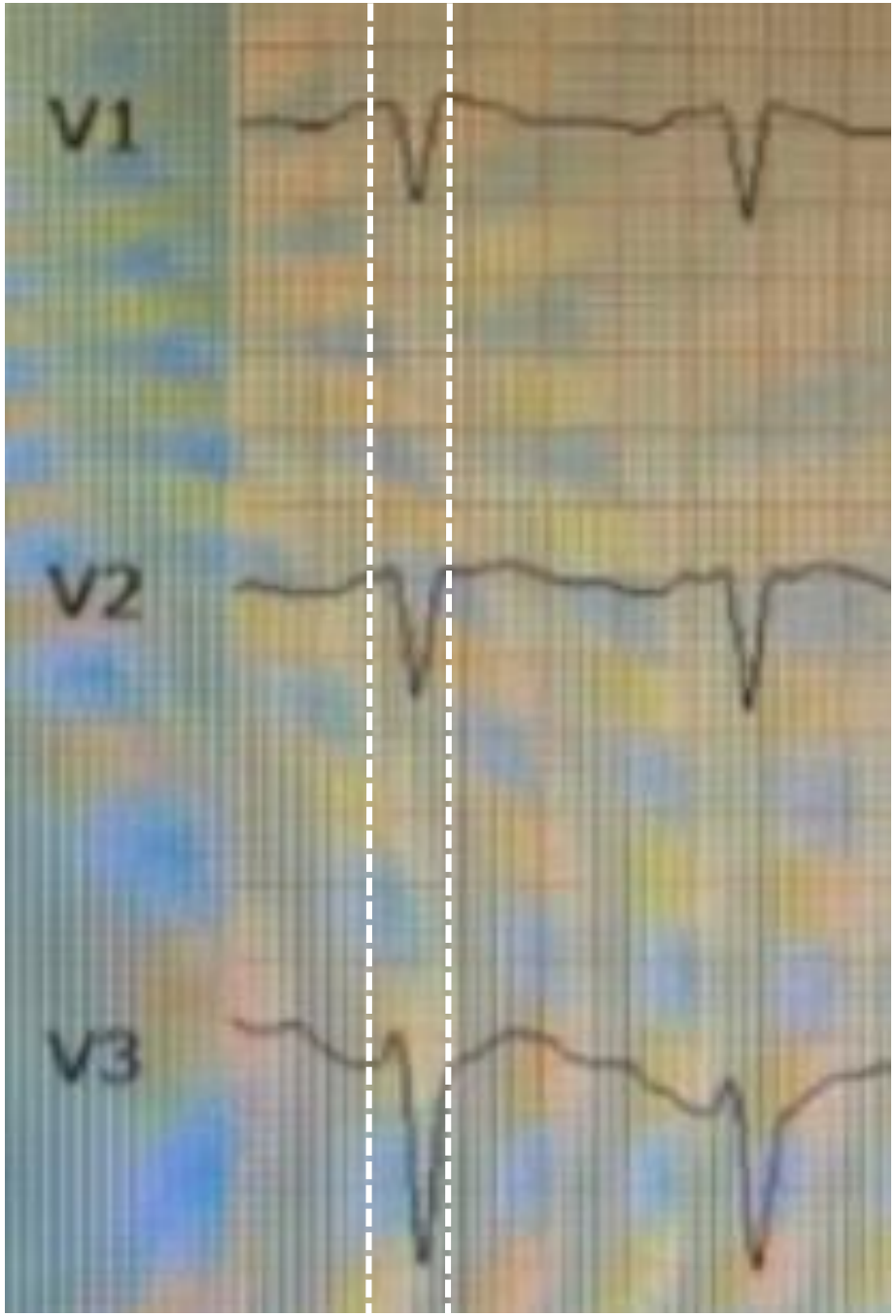
Number 3B Only right precordial leads During exercise test, 100 watt, heart rate 160/min

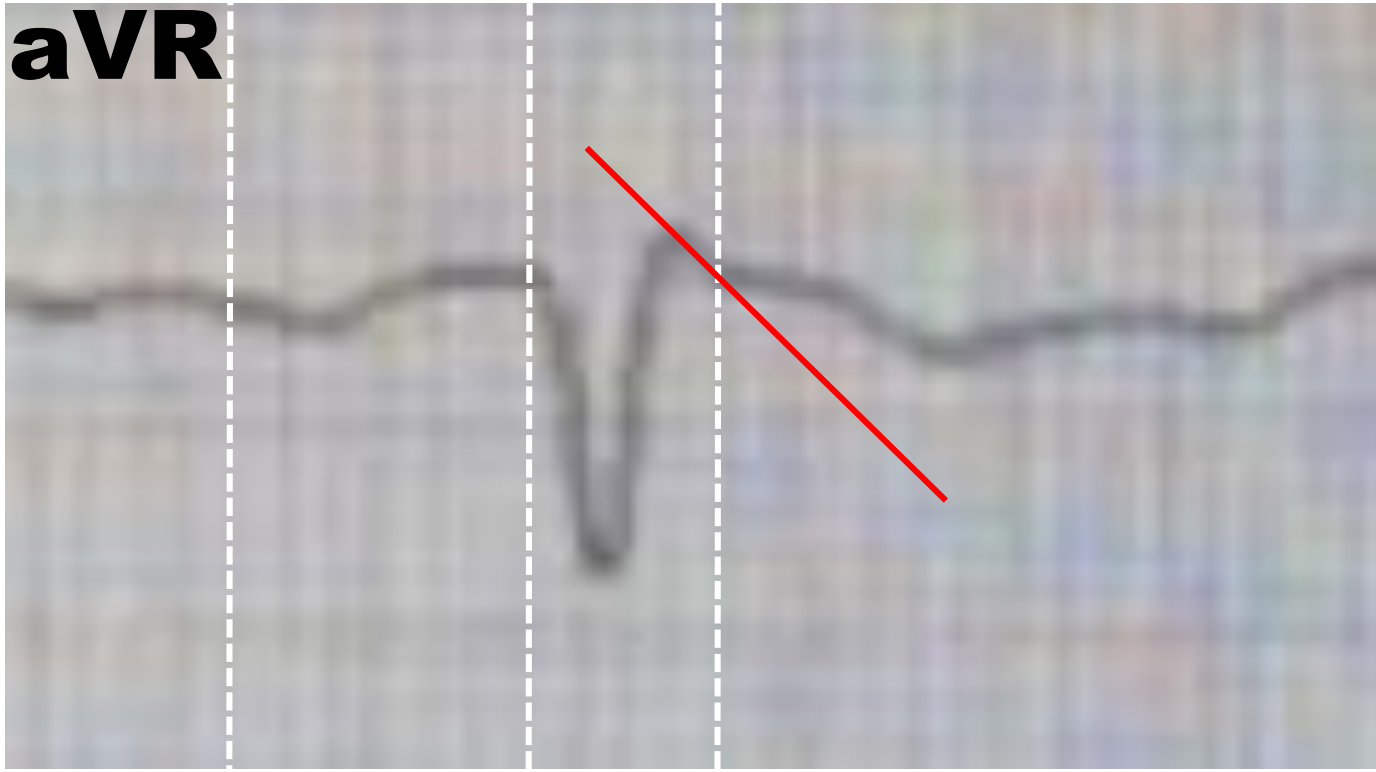


4A: Recovery phase

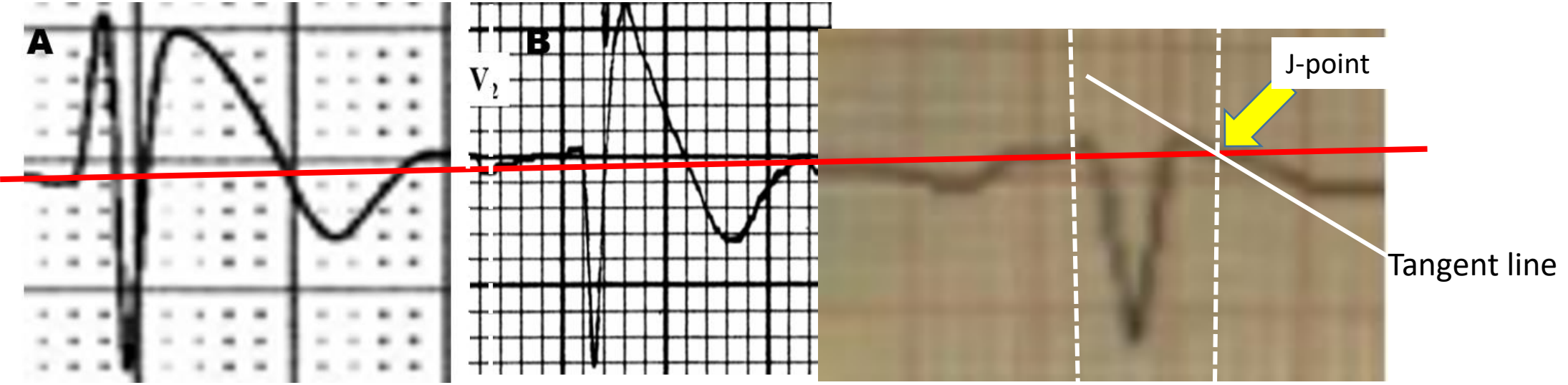


4B: Only Right precordial leads Recovery phase





Criterial for Type 1 Brugada pattern. J point and ST elevation ≥2 mm, with upper convexity (A) or descending oblique rectilinear (B), followed by symmetric negative T wave in the right precordial leads (V1-V2 or from V1 through V3) and/or High lead placement, 2nd ICSs (V_{2H}) and . the 3rd ICSs (V_{3H}) The present case ABSENT **Type 1 Brugada pattern**



More thinks What about family background ?. It is very important near syncope characterization:

This is a near-syncope of unclear mechanism/unclear etiology.

Suspected arrhythmic syncope or reflex neurally mediatedl?

syncope associated with prodromes. specific triggers ?

Main categories of causes of syncope grouped by common pathophysiology, presentation and risk

I) **Cardiac syncope (generally high risk)**
   a. Arrhythmia, eg, bradycardia (BrS) or tachycardia.
   b. Structural, eg, aortic stenosis, hypertrophic cardiomyopathy, pulmonary embolus.

II) **Reflex (neurally mediated) syncope (generally low risk)**
   a. Vasovagal
      I. Orthostatic vasovagal syncope that is triggered by standing.
      II. Emotional, eg, triggered by fear or venepuncture.
      III. Pain triggered.
   b. Situational
      I. Micturition.
      II. Gastrointestinal, eg, swallow syncope, defaecation syncope.
      III. Coughing/sneezing.
      IV. Post exercise. V. Other, eg, laugh syncope. Your case
   c. Carotid sinus syncope.

d. Atypical, ie, without prodrome/triggers. The above can be predominantly

► Cardioinhibitory reflex syncope—leads to a low cardiac output.

► Vasodepressor reflex syncope—leads to a low peripheral resistance.

► Mixed— combination of cardioinhibitory and vasodepressor. Orthostatic syncope (generally low risk) a. Drug-induced. Volume depletion. c. Primary autonomic failure, eg, Parkinson's disease. d. Secondary autonomic failure, eg, diabetes. T above can be exacerbated after exercise, meals or prolonged bed rest due to venous pooling. OH can be

► Classic (time from upright position to abnormal BP response 3min).

Orthostatic syncope (generally low risk)

a. Drug-induced.
b. Volume depletion.
c. Primary autonomic failure, eg, Parkinson's disease.
d. Secondary autonomic failure, eg, diabetes. The above can be exacerbated after exercise, meals or prolonged bed rest due to venous pooling. OH can be
    ► Classic (time from upright position to abnormal BP response 3min)
    ► Delayed (time from upright position to abnormal BP response >3min).

ED risk stratification as recommended by the 2018 ESC guidelines for the diagnosis and management of syncope

I) Syncopal event Low risk

► Associated prodrome typical of reflex syncope (eg, lightheadedness, feeling of warmth, sweating, nausea, vomiting).

► After sudden unexpected unpleasant sight, sound, smell or pain.

► After prolonged standing or crowded, hot places.

► During a meal or postprandial.

► Triggered by cough, defecation or micturition.

► With head rotation or pressure on carotid sinus (eg, tumor, shaving, tight collars).

► Standing from supine/sitting position.

II) Medical history Low risk

► Long history (years) of recurrent syncope with low-risk features with the same characteristics of the current episode.

► Absence of structural heart disease.(except **BrS, ERS, JWS, LQTS, CPVT, SQTS, and LQTS**)

III) Physical examination Low risk

► Normal examination. .(except **BrS, ERS, JWS, IVF, LQTS, CPVT, SQTS, and LQTS**)

IV) ECG Low risk

► Normal ECG. .(except **CPVT, IVF**)

High risk (red flag) Major

- ECG changes consistent with acute ischaemia.
- Mobitz II second-degree and third-degree atrioventricular (AV) block.
- Slow atrial fibrillation (AF) (3 s in awake state and in absence of physical training.
- Bundle branch block, intraventricular conduction disturbance, ventricular hypertrophy or Q waves consistent with ischaemic heart disease or cardiomyopathy.
- Sustained and non-sustained ventricular tachycardia.
- Dysfunction of an implantable cardiac device (pacemaker or implantable cardioverter defibrillator)
- ST-segment elevation with type 1 morphology in leads V1− V3 (Brugada pattern).
- QTc >460 ms in repeated 12-lead ECGs indicating long QT syndrome. Minor (high risk only if history consistent with arrhythmic syncope)
- Mobitz I second-degree AV block and first-degree AV block with markedly prolonged PR interval.
- Asymptomatic inappropriate mild sinus bradycardia (40– 50 bpm), or slow AF (40–50 bpm).
- Paroxysmal supraventricular tachycardia or AF.
- Pre-excited QRS complex.
- Short QTc interval (≤ 340 ms).
- Atypical Brugada patterns.
- Negative T waves in right precordial leads, epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy.