Dear Professor Pérez-Riera,

I would like to ask your opinion on the following case which I suspect is one of RBBB combined with new LSFB owing to ischemia related to a proximal LAD artery lesion. The patient was an elderly man with a history of suspected vasospastic angina. He presented to the hospital after he had suffered a short-lasting episode of angina and his ECG recorded when he was free of pain is attached (**ECG 1**). During an episode of angina (**ECG 2**) we noted lambda-like ST elevation in leads V2-V5 and ST-segment elevation in leads II, aVF and III. The rhythm is atrial fibrillation/flutter and also there are signs of RBBB.

Do you agree that ECG 2 displays signs of LSFB? : prominent anterior QRS forces [PAF] with R wave in V2 and V3 ≥ to 15 mm, loss of septal q waves in V4 and V5)

The transient nature of PAF also favored LSFB.

ECG 3 was recorded during a 2nd episode of angina which also resolved promptly after treatment with nitrates.

The case was one of sequential spasm in the proximal LAD artery and the distal RCA at sites of high-grade lesions. Both lesions were tackled by PCI. The patient remained free of CV events 2.5 years after PCI.

Thank you very much

Kind regards

Andreas Y. Andreou, MD, FESC, FACC, FSCAI Clinical Associate Professor, University of Nicosia Medical School

Cardiologist – Interventional Cardiologist Limassol General Hospital, Limassol, Cyprus

y.andreas@yahoo.com

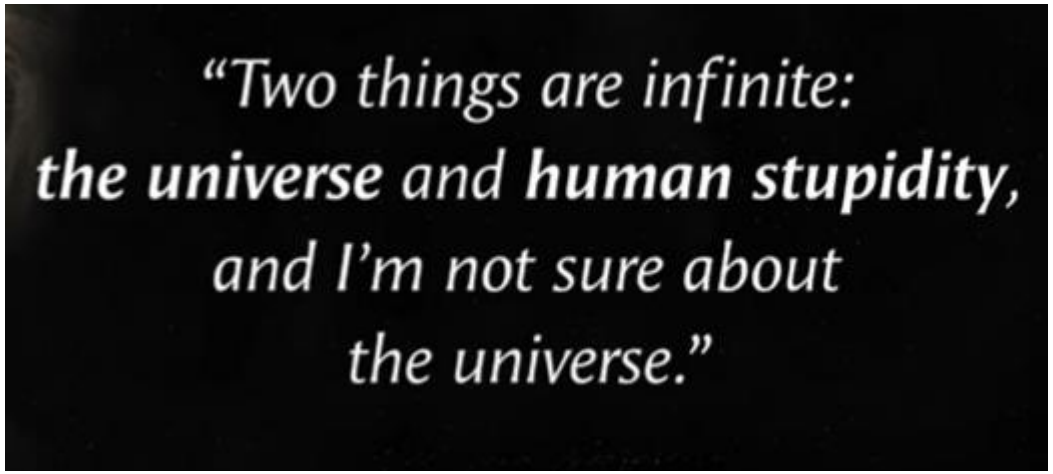
Case Report from

Andreas Y. Andreou, MD, FESC, FACC, FSCAI Clinical Associate Professor, University of Nicosia Medical School Cardiologist – Interventional Cardiologist Limassol General Hospital, Limassol, Cyprus

y.andreas@yahoo.com

[1]Department of Cardiology, Limassol General Hospital, Limassol, Cyprus. y.andreas@yahoo.com

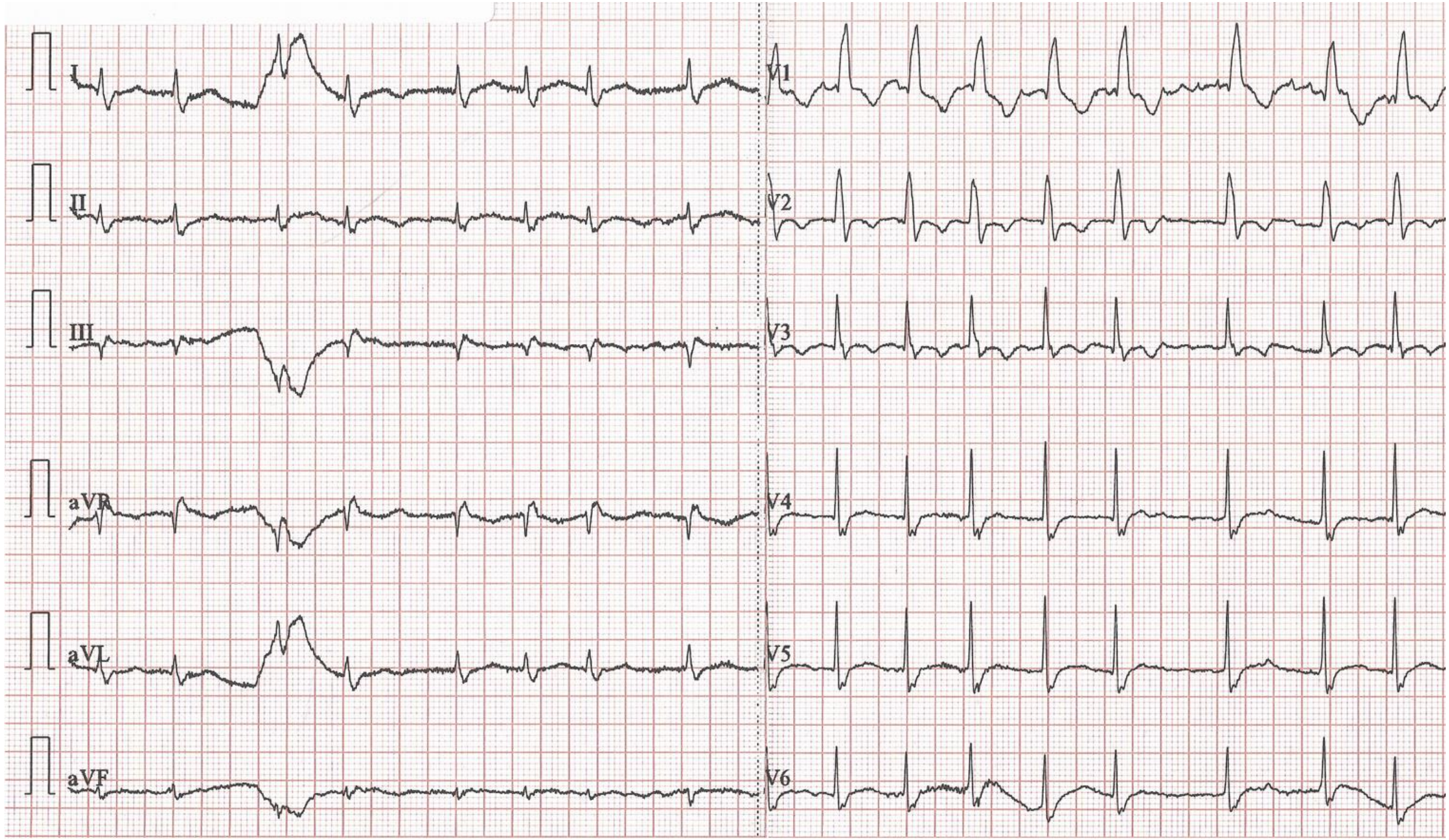
[2]University of Nicosia Medical School, Nicosia, Cyprus. y.andreas@yahoo.com.



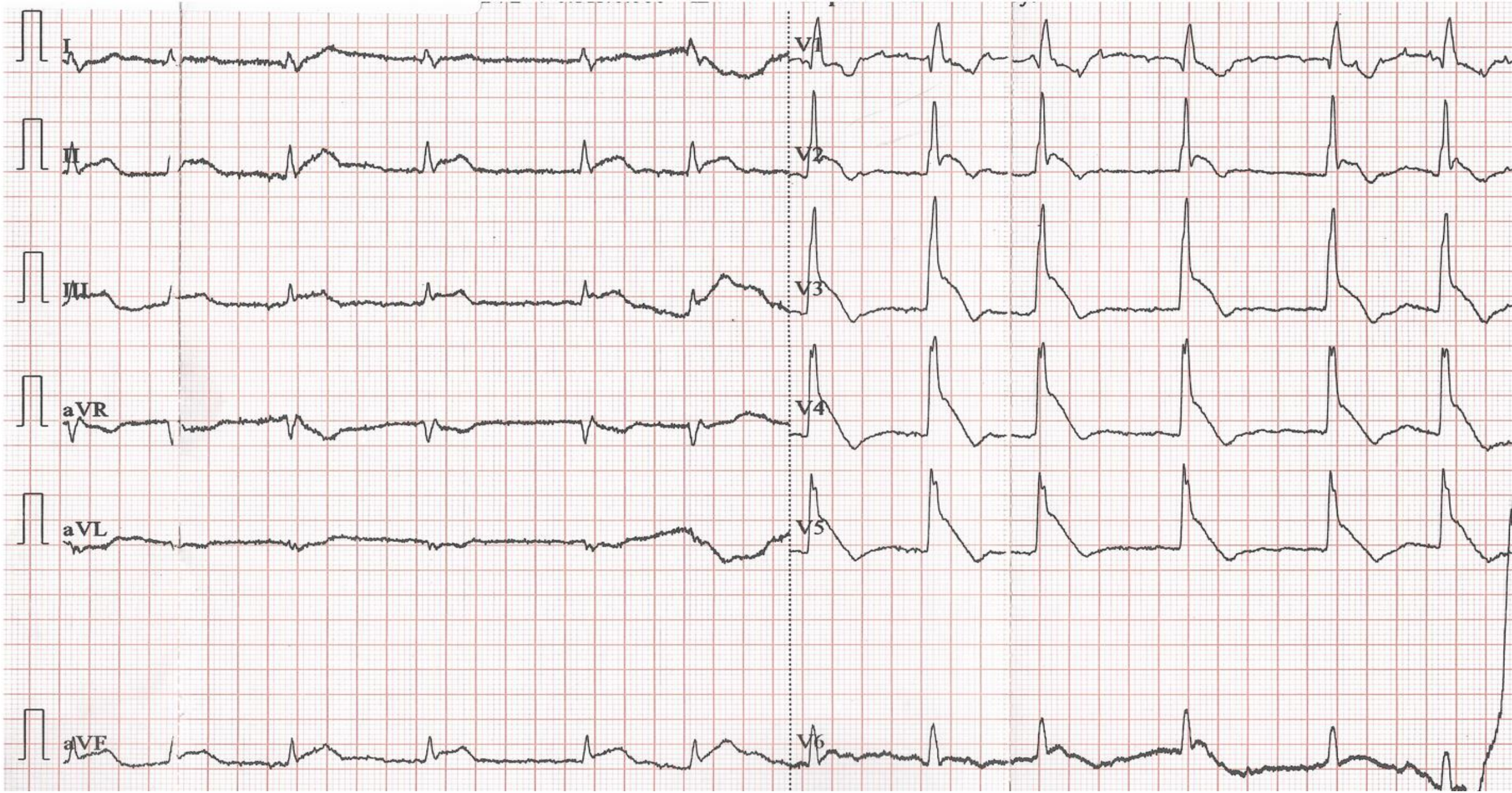
https://www.youtube.com/watch?v=lhJJsnUORhk

Albert Einstein's Quote

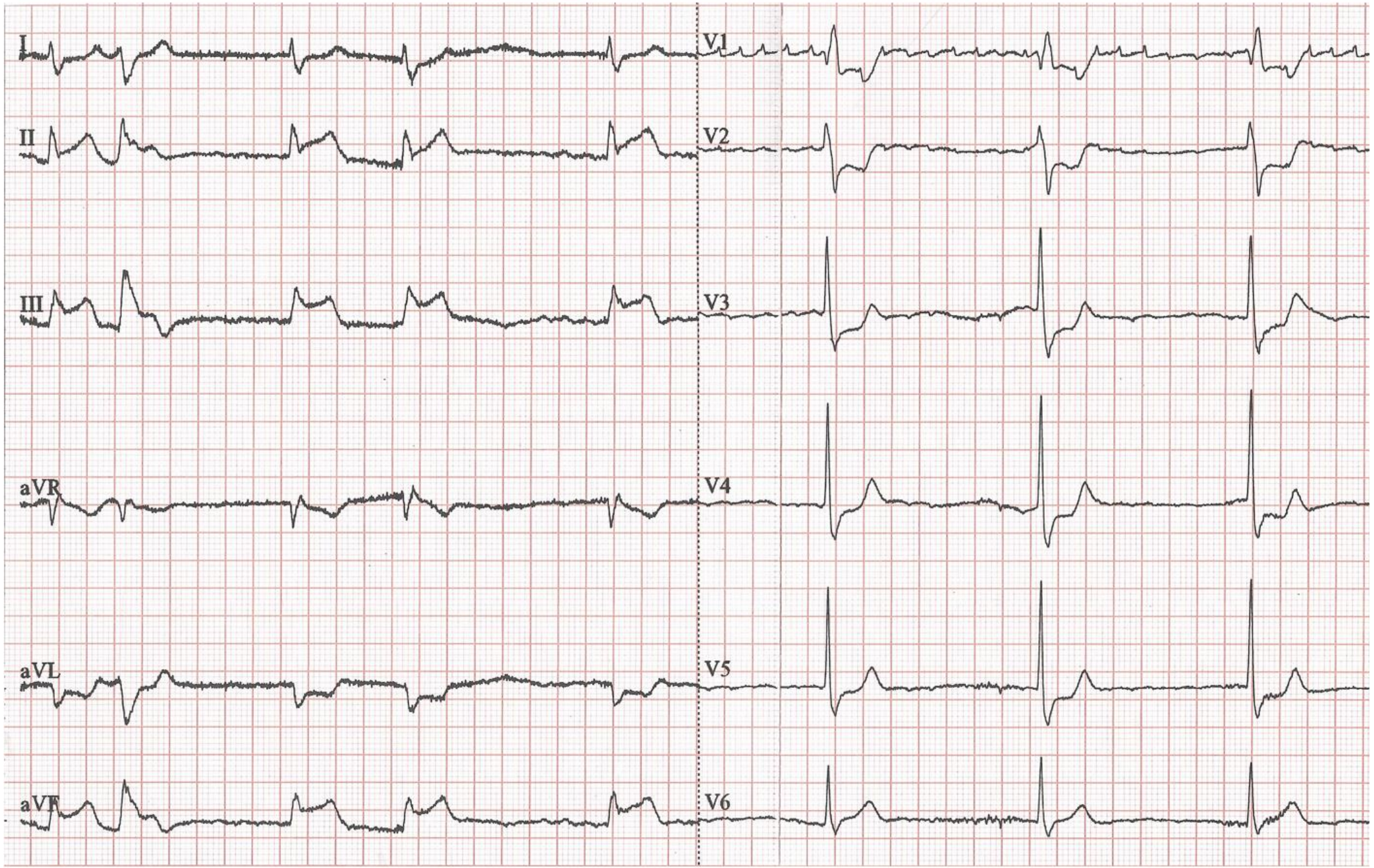
ECG-1



ECG-2 preformed during angina episode



ECG-3 preformed during a 2nd episode of angina which also resolved promptly after treatment with nitrates.



Colleagues opinions

Thank you, Andrés, for sharing the very interesting case of Dr. Andreou's from Cyprus. I agree with Dr. Andreou's assessment of transient left septal fascicular block (LSFB) during an acute episode of myocardial ischemia. (**ECG-2)**

ECG-1 shows underlying atrial fibrillation, RBBB with primary T wave changes suggestive of chronic Atherosclerotic cardiovascular disease (ASCVD); the frontal plane QRS axis is indeterminate.

ECG-2 during apparent coronary spasm shows "lambda like" wave transmural ischemia in the anterior leads along with ST segment elevation in the inferior leads consistent with his underlying coronary anatomy. The growth of anterior R waves and loss of septal q-waves in V5-6 in ECG-2 meets criteria for transient LSFB.

Warmest regards,

Frank G. Yanowitz MD

Professor of Medicine (Retired) University of Utah School of Medicine He is a Cardiology Specialist in Salt Lake City, UT and has over 55 years of experience in the medical field. He graduated from New York State U, College of Medicine -

Spanish

ECG #1 creo que hay ritmo de aleteo o taquicardia auricular con pasaje AV variable, menos fibrilación auricular, porque parece tener ondas regulares en V1, BRD.

ECG #2 registrado durante el dolor, se agregan FAP. Se confunde si hay onda r inicial en V1 por el ritmo supra ventricular superpuesto en el QRS.(si es LSFB no debería tener onda "r" inicial en V1. La onda R en V1 > a 5 mm, R en V2 tiene voltaje de 15 mm, no hay ondas "s" en precordiales derechas ni se puede medir relación R/S por la onda lambda. El TAV en V2 >35 msg. El vértice de R de V2 ocurre antes que R de V3. Si bien no hay complejos qR en V2 V3, son complejos R, la misma disminuye a V5V6. No hay onda "q" en V5V6 y D1. La duración del QRS es > 110 mseg por estar asociado a BRD.

Imagino que el VCG en PH la rotación del bucle QRS deba ser horaria por estar asociado a BRD y se encuentra localizado predominantemente en el cuadrante anterior izquierdo. LSFB transitorio por espasmo de LAD antes de 1° septal (esta arteria irriga el fascículo medio septal en 100% de los casos). Y muestra lo que ud, estimado Potro, siempre comenta en sus disertaciones, que el Profesor Rosenbaum quería ver, la intermitencia del presunto LSFB para solo así admitiría su existencia.

ECG #3 desaparece el LSFB por la acción farmacológica del nitrato cediendo el espasmo y recuperando el flujo de la LAD. Hay compromiso de CD que se observa en la elevación del segmento ST III > II , sin concomitante elevación del ST en aVR, (lo que anula la posibilidad de responder obstrucción del tronco de la coronaria izquierda . En el último trazado se observa mejor el ritmo supra ventricular , creo que no es FA.

Perdón por lo extenso y disperso.

Saludos cordiales

Juan Carlos Manzzardo Hospital Ítalo Perrupato Mendoza Argentina

English

ECG # 1 atrial flutter rhythm or atrial tachycardia with variable AV ventricular response , in V1, RBBB.

ECG # 2 recorded during angina , Prominent anterior QRS forces are added. It is confused if there is an initial r wave in V1 by the superimposed supraventricular rhythm in the QRS. (If it is LSFB it should not have an initial "r" wave in V1. The R wave voltage in V1> at 5 mm, R in V2 has voltage of 15 mm, there are no "s" waves in the right precordial nor can the R / S ratio be measured by the lambda wave. Prolonged ventricular activation time in V2 (> 35 msg). The R-apex of V2 occurs before R of V3. qR complexes in V2 V3, R voltage decreases to V5V6. There is no "q" wave in V5V6 and D1.The duration of the QRS is ≥ 120 msec because it is associated with RBBB.

I imagine that the vectorcardiogram in the Horizontal plane PH has CW QRS loop rotation because it is it is associated with RBBB and is predominantly located in the left anterior quadrant.

In summary, I believe that a transient LSFB is associated with LAD spasm before first septal 1 branch (since this artery is the only one that supplies the left septal fascicle). Intermittent LSFB.

ECG # 3 the LSFB disappears due to the pharmacological action of nitrate, yielding the spasm and the LAD regaining flow. There is CD involvement that is seen in STSE III> II, without ST elevation in aVR, therefore it does not respond to left main coronary artery involvement.

In the l **ECG # 3** tracing, the supraventricular rhythm is better observed, I think it is not AF.

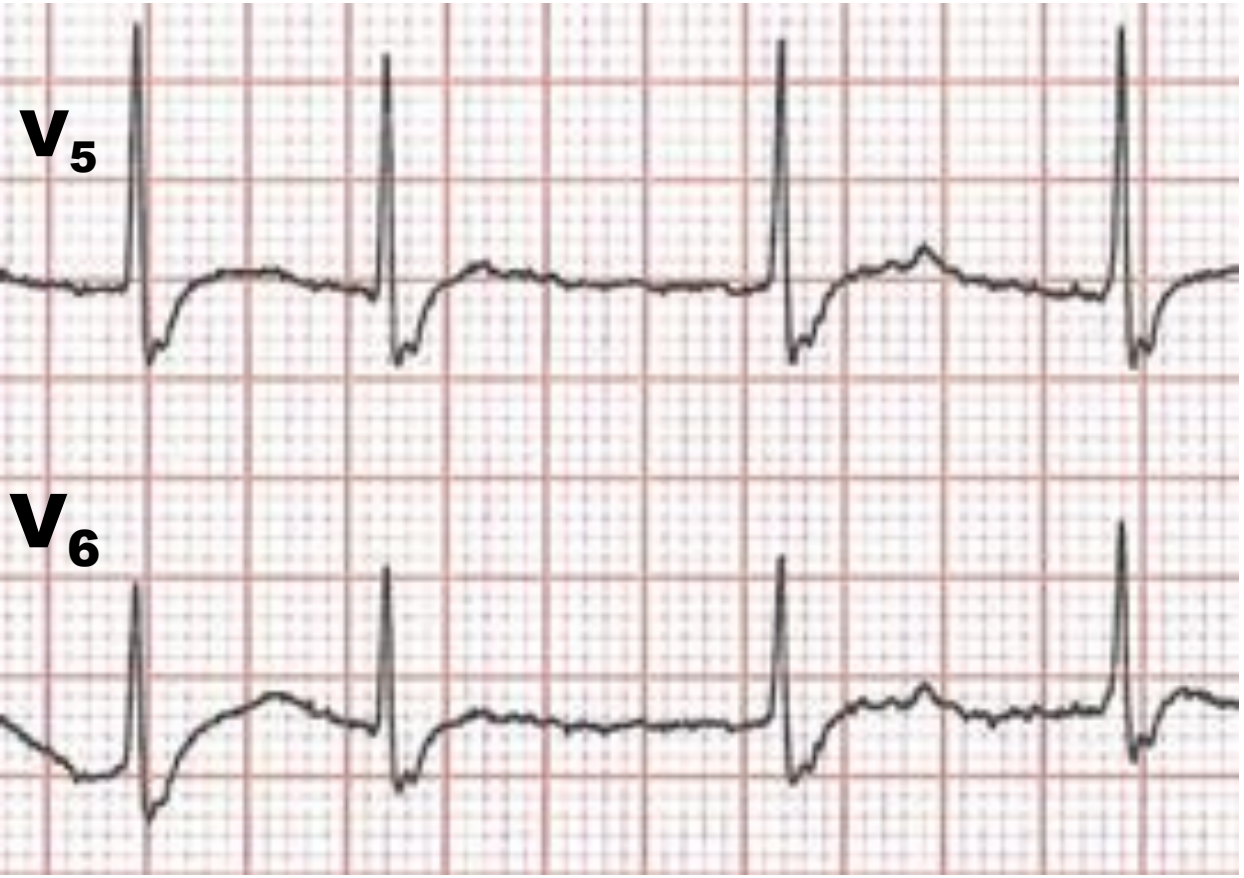
Sorry for the extensive and scattered.

Kind regards

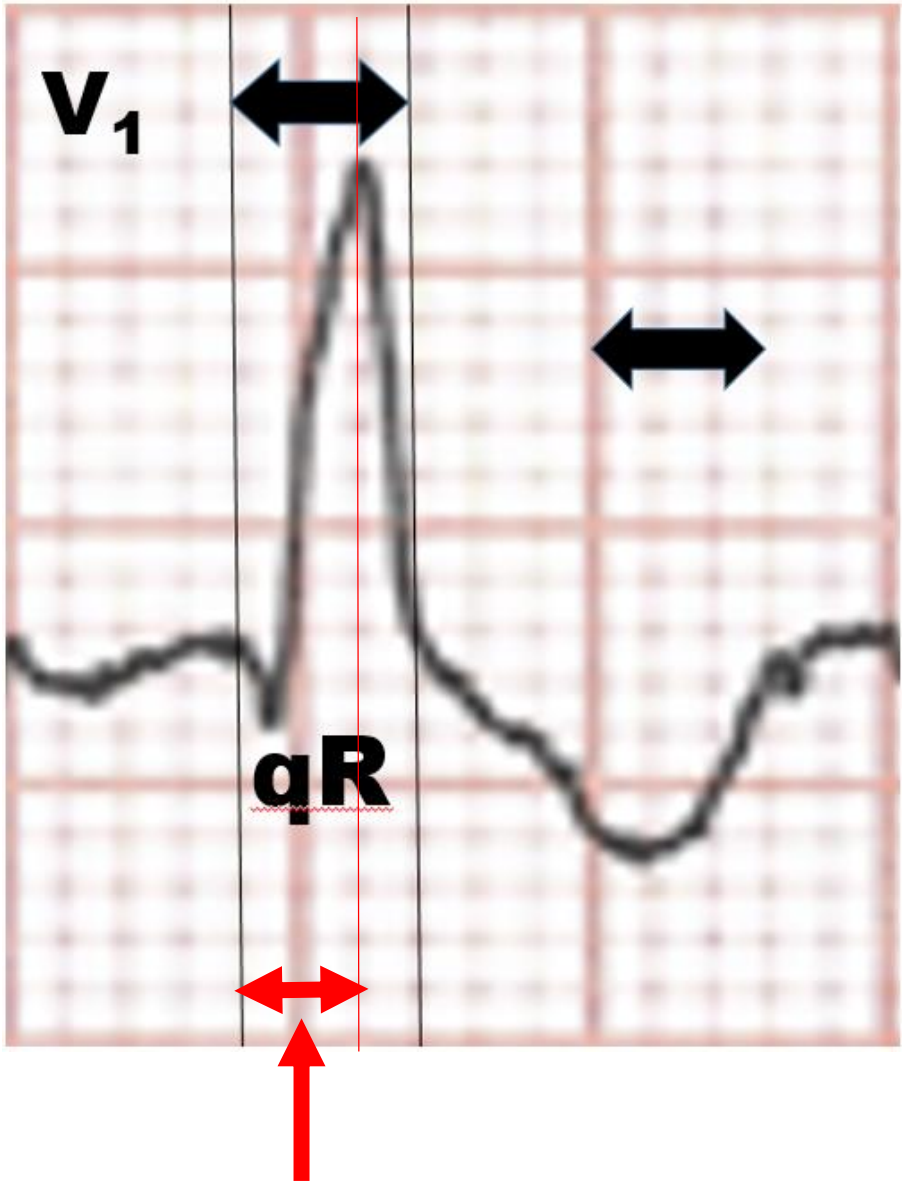
Juan Carlos Manzzardo Hospital Ítalo Perrupato Mendoza Argentina

Dr. Pérez-Riera interpretation

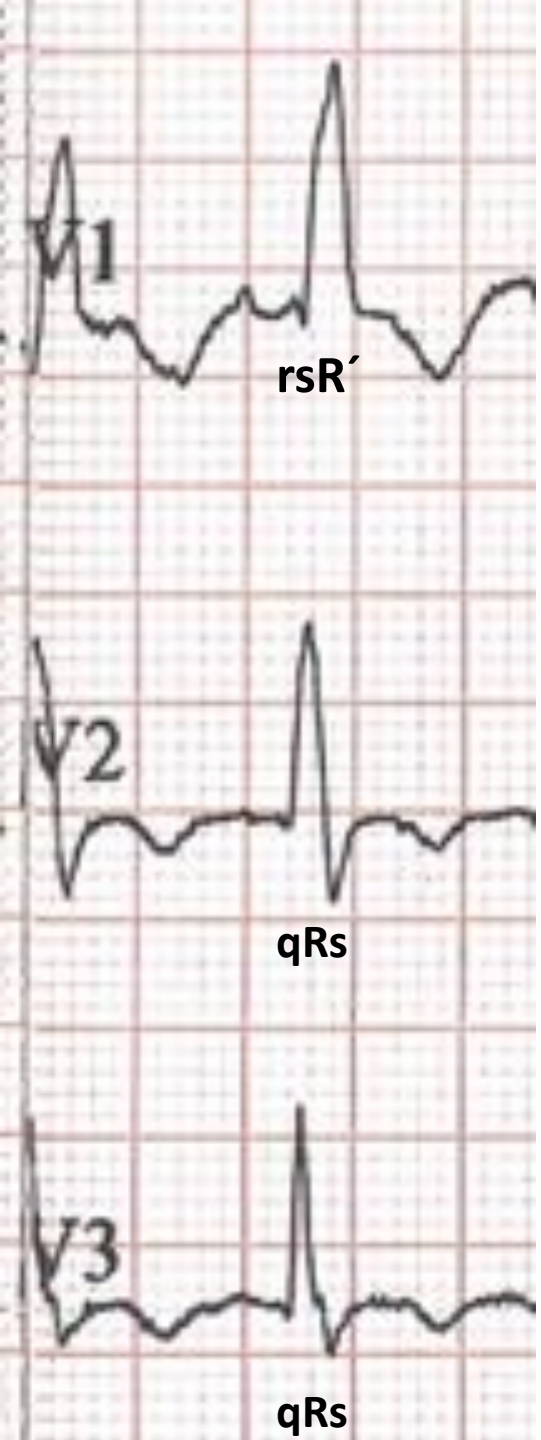
ECG-1: Atrial fibrillation, + Complete RBBB + prolonged VAT ischemic T wave



•Wide, slurred S wave in lateral leads (I, aVL, V5-6)



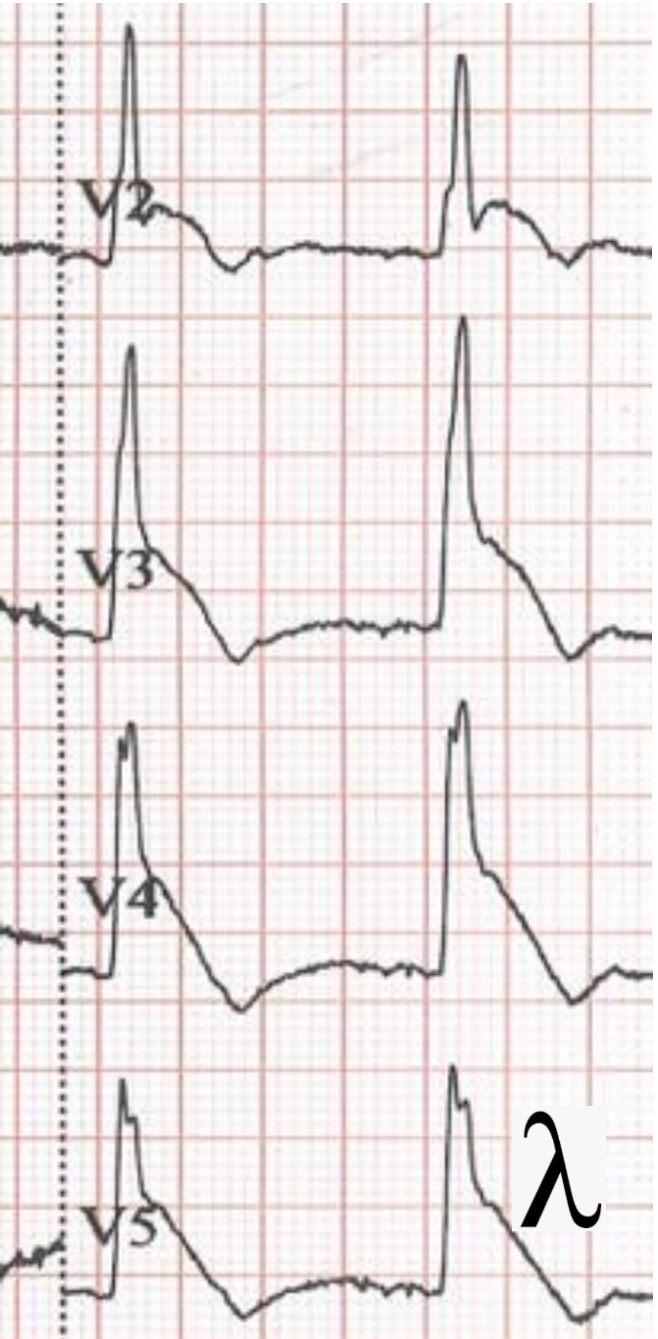
Prolonged Ventricular Activation Time (VAT) , R-Wave Peak Time(RWPT) or intrisecoide deflection

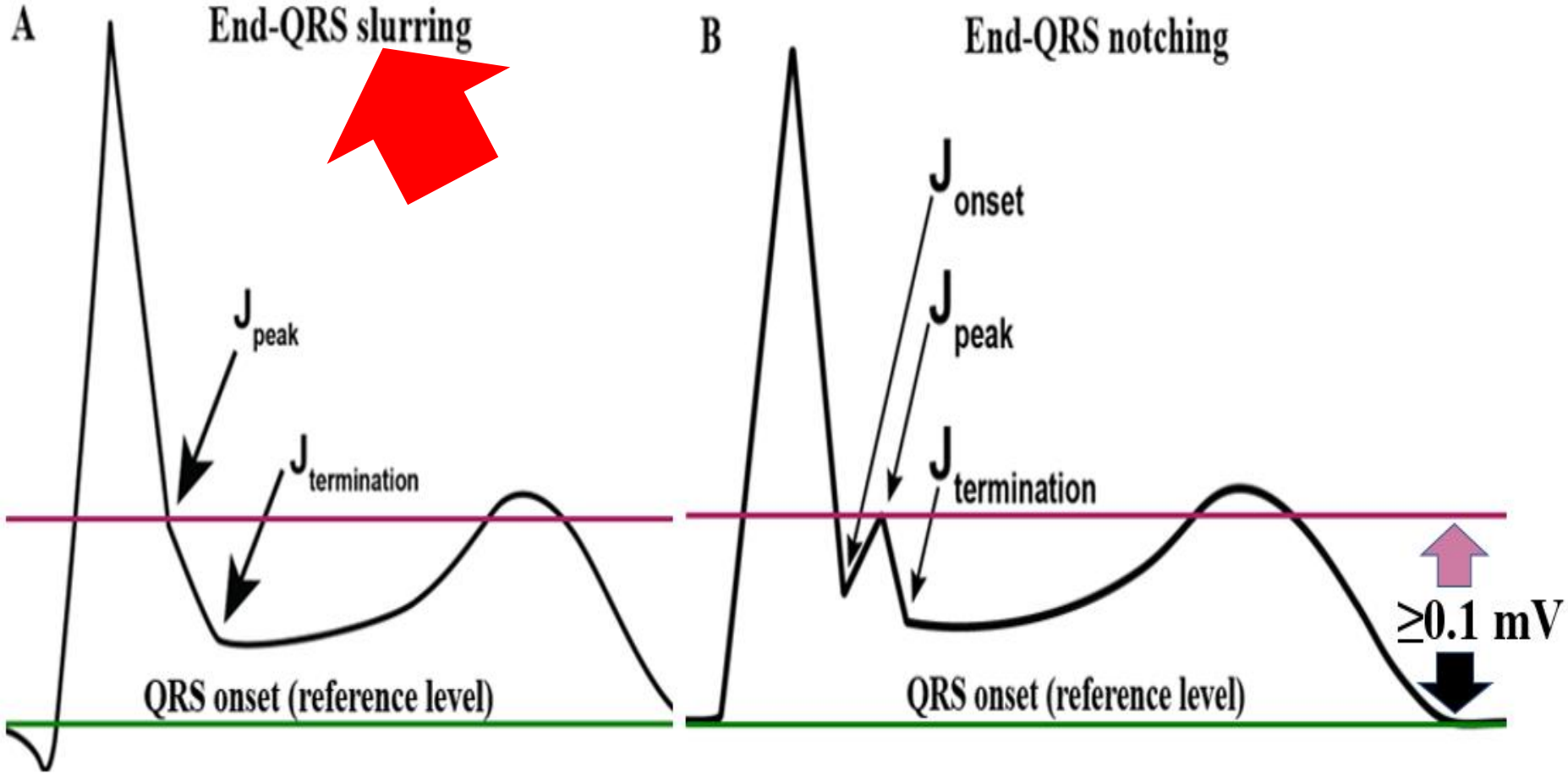


There should be a terminal R wave in lead V_1 (often called "R prime," and denoted by R, rR', rsR', rSR', or qR).

Appropriate discordance: minimal ST depression followed by T-wave inversion (TWI) in right precordial leads (V1-3). Remember that TWI and ST segment depression are normal in leads V1 to V3 in the presence of a RBBB; thus, myocardial ischemia technically cannot be easily determined in these leads. However, unlike in the presence of a LBBB, myocardial ischemia and infarction can easily be detected on ECG when a RBBB is present.

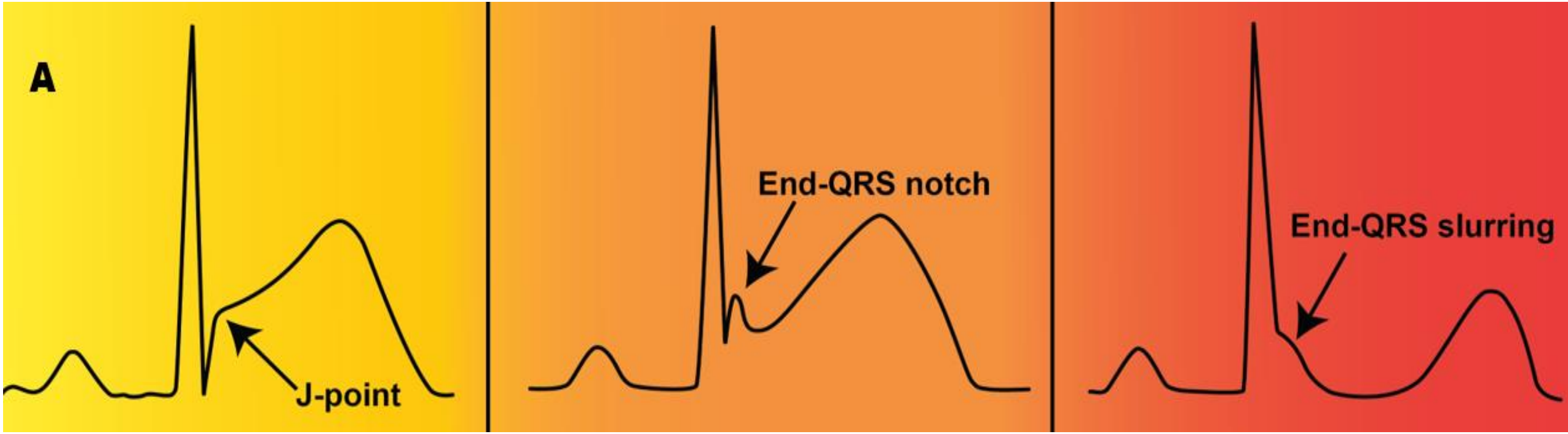
ECG-2 preformed during angina episode J-wave end QRS Slurring or lambda-like





ERP variants: (A) QRS slurring or lambda wave and (B) end-QRS notching. J point is present in all ECGs and marks the transition of QRS complex to ST segment. In both variant, the J-point correspond to J-termination point. The J point is a point in time marking approximately the end of the QRS complex and the onset of the ST segment present on all ECG's. One condition for ERP to be present is Jp ≥0.1 mV, while ST-segment elevation is not a required criterion.

The simple elevation of the J point and ST segment in the absence of a J wave (A) should not be considered an ERP pattern (Figure).



ERS/J wave syndrome is defined as a condition demonstrating an ERP, i.e. a distinct J-point elevation ≥0.1mV from the isoelectric line with a notch or slur at the terminal QRS and/or ST-segment elevation in at least two adjacent leads, in the inferior/lateral leads in a patient resituated from unexplained VF/PVT events. Additionally, ERS can be diagnosed in and SCF victim with a negative autopsy and medical chart review, with previous ECG demonstrating J-point elevation ≥1mm in ≥2 contiguous inferior and/or lateral leads of a standard 12-lead ECG.

The J wave of the ECG theoretical considerations

The J wave is a positive deflection in a normal ECG (present in 2–14% of healthy individuals and is more prevalent in young males, particularly athletic and African/ Afro-Caribbean descent. Additionally, ERP is a common ECG finding in young teenager athletes (the prevalence in the athletic population rises to 20-90%). In this population ERP in both inferior and lateral leads is more common (18.2%) than isolated inferior (9.1%) or lateral (8.2%). ERP, especially the diffuse ST ascending pattern, is common among the young, in those of European ethnicity, found equally in both genders, and with no apparent correlation with atrial nor ventricular arrhythmias.6 Young age might be a contributing factor in causing a more diffuse repolarization abnormality or pathological that occurs approximately (there is an overlap of ≈10 msec) after the junction between the end of the QRS complex and the beginning of the ST segment (also known as the J point, QRS end, J-junction, ST0 [zero msec] or ST beginning to occur after the notch/slur or J wave). It is described as J deflection as slurring/lambda or notching of the terminal portion of QRS complex. Currently, J wave is defined as an elevation of the QRS-ST junction ≥1 mm either as QRS slurring or notching in at least 2 contiguous leads. Additionally, when J wave becomes more accentuated, it may appear as a small R wave (R') or ST segment elevation.

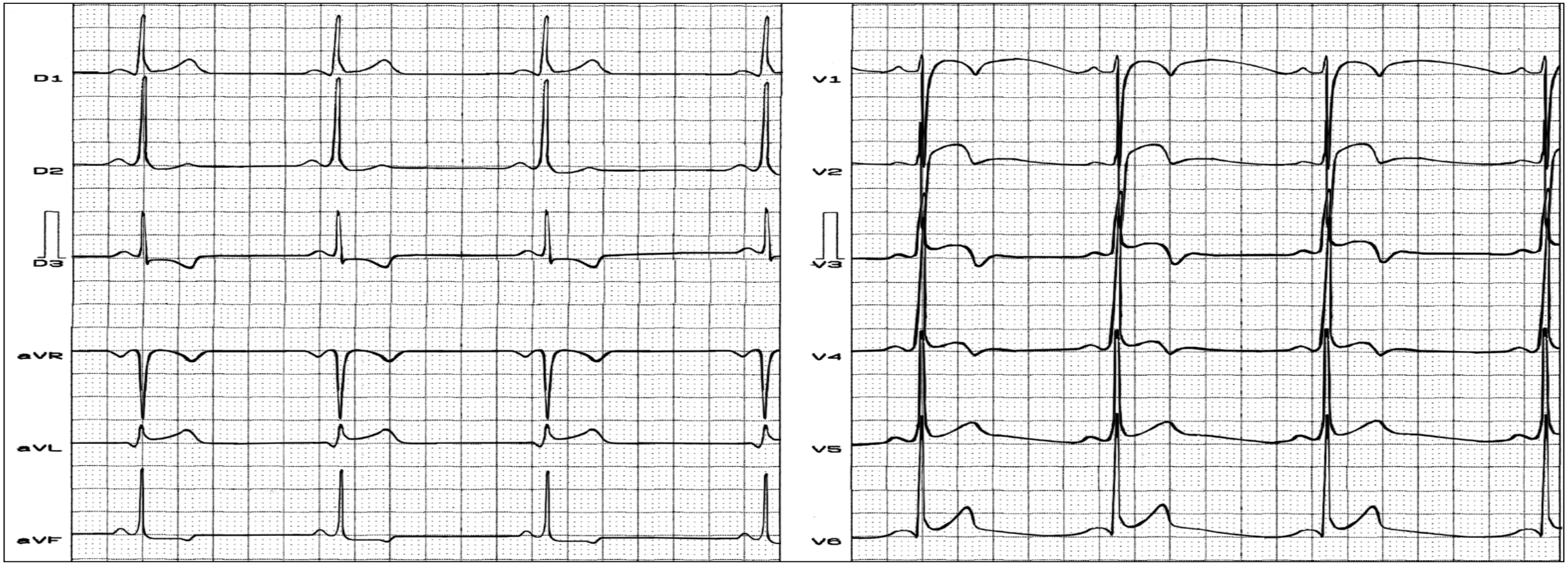
The term J deflection or J wave has been used to designate the formation of the wave produced when there is a large, prominent deviation of the J point from the baseline with two shapes: notching/spike-and-slurring/lambda or dome variety. All J wave deflections do not look alike. Some are elevations of J-point and ST segments ≥2 mm followed by negative symmetrical T wave in leads V1 and/or V2 in at least one lead: BrS whereas others are of the spike-and-dome variety. This suggests that different mechanisms may be responsible for the size and shape of J wave deflections. The J point in the ECG is the point where the QRS complex joins the ST segment. This represents the approximate end of depolarization and the beginning of repolarization as determined by the surface ECG. There is an overlap of ≈10 msec.

Electrocardiographic characteristic of “innocent ERP” or physiological J wave

HR: frequently characteristic sinus bradycardia, frequent phasic or respiratory sinus arrhythmia, QRS, ST segment and T wave axis, oriented in the same direction in the frontal plane, deep and narrow Q waves followed by R wave of great voltage in the left precordial leads, notch or slur of R wave descending branch (J wave), possible but not obligatory, transition QRS precordial zone of sudden occurrence, height of J wave: 1-2 mm, rarely more, no transient/fluctuating global J wave augmentation, ST segment elevation, rapidly ascending/ up sloping "upper concavity” followed by anterolateral ST segment elevation (It is observed in 1-2 % of the general population or only lateral or inferior leads followed by a broad positive tall T wave resembling a "smiling face“, possible reduction in the J point and ST segment elevation by sympathetic action and sympathomimetic drugs, absence of reciprocal or mirror image (with exception in aVR lead), ST segment elevation concave upward "upper concavity", followed by a broad positive pseud symmetric tall T wave, absence of short coupled PVCs (R-on-T phenomenon), unknown syncope and family history of SCD. Rapidly ascending/upsloping ST-segment morphology after the J-point68 followed by tall/symmetric T wave, is generally considered to be benign when there is 0.1 mV elevation of the ST-segment within 100 msec after the J-point and the ST-segment gradually merge with the T-wave. An ST-segment with upward concavity followed by a T-wave is seen in Caucasians, and an elevated ST-segment with upward

convexity and negative T-wave in African-Caribbean athletes. Figure below

Name: BCW; **Age:** 24yo.; **Sex:** Male; **Race:** Black; **Weight:** 86 kg; **Height:** 2.02 m**; Biotype:** Asthenic; **Profession:** professional basketball player; **Date:** 05/01/2019



Clinical diagnosis: healthy patient. Tracing obtained in a periodical evaluation.

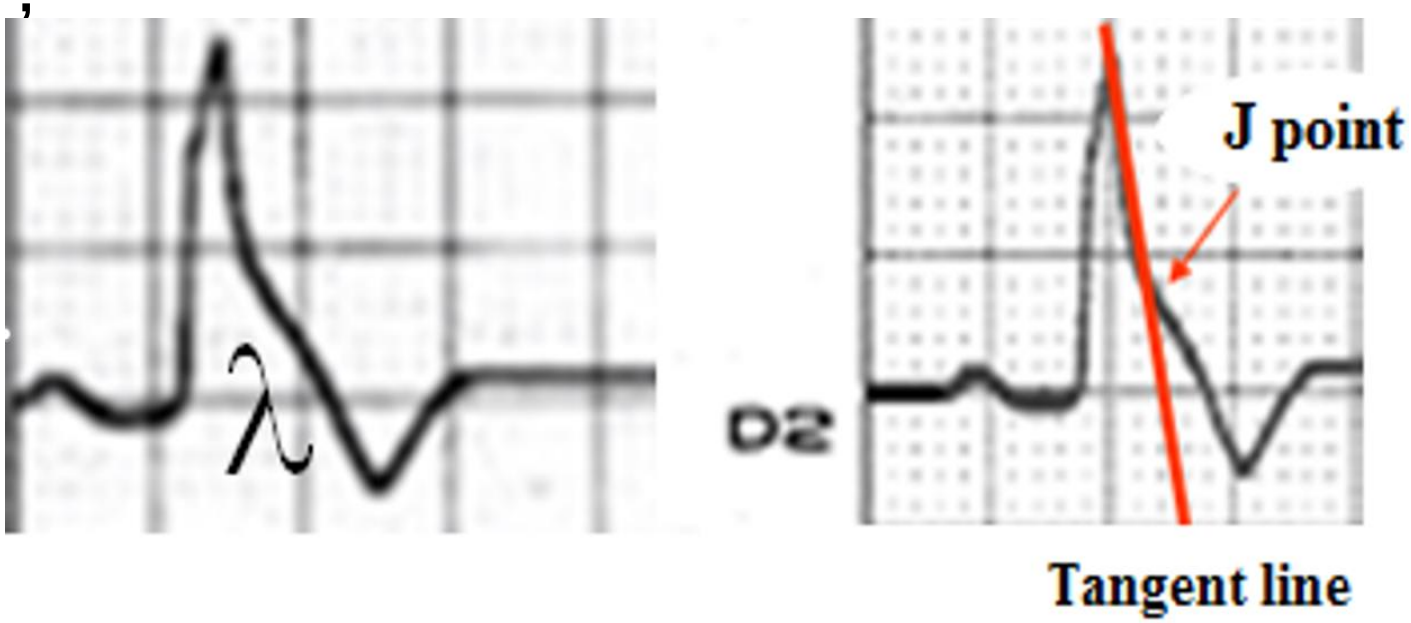
ECG diagnosis: sinus bradycardia, phasic sinus arrhythmia. Positive voltage criterion for LVE. SV1 or V2+RV5 or V6 >35 mm (Index of Sokolow Lyon). ST segment elevation from V2 to V6 and with negative T from V1 to V4. Early repolarization, pattern of pseudo injury and anterior subepicardial ischemia. Normal chest X-rays and echocardiogram. Pattern of pseudo epicardial injury and ischemia in anterior wall in an athlete, professional player of basketball with normal heart.

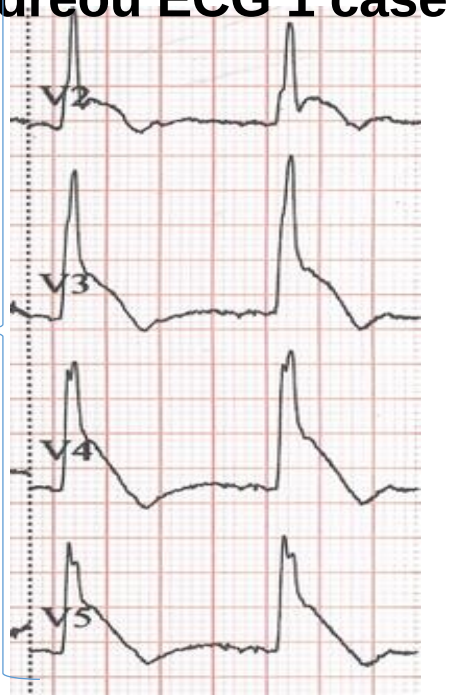
Electrocardiographic characteristic of pathologic J-waves, malignant J waves or evil J-wave

High amplitude of J-point elevation (≥0.2 mV), transient/fluctuating global J-wave augmentation: The occurrence of VF episodes is always accompanied by an accentuation of the J wave amplitude. Isoproterenol (1–4 μM/min) or pacing at rates of 90–100 bpm abolished these ECG changes and prevented the recurrence of VF. Characteristic dynamic amplitude J-wave level portends a high risk for VF in patients with ER and should be closely monitored, since this could signify an imminent risk for the development of ES, J-wave without typically rapidly ascending ST segment horizontal or down sloping when the ST-segment elevation is 0.1 mV within 100 ms after the J-point and continues as a flat(horizontal) ST-segment until the onset of the T-wave. In other words, a combination of J waves with horizontal/descending ST segment, “horizontal/descending” pattern, global J wave or widespread J-wave in inferior, lateral and anterior walls leads was associated with a higher incidence of VF recurrence in patients with JWS (Figure).

Dr **Andreas Y. Andreou ECG 1 case**

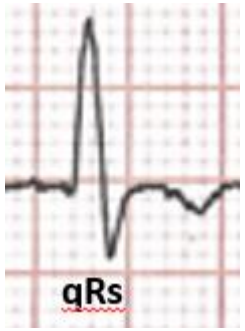
ECG-1



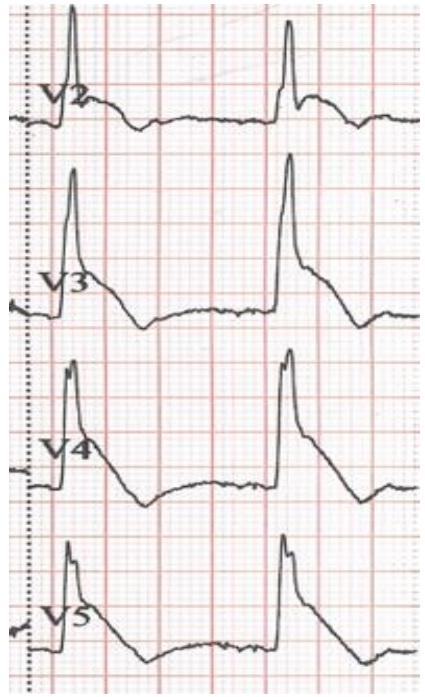


Very high J-point with descending ST segment followed by a negative T-wave. It is considered a malignant or “evil” form.

LSFB Electrocardiographic characterization **(Abrahao, Schwartz et al. 1979) (Dabrowska, Ruka et al. 1978) (MacAlpin 2002) (MacAlpin 2003) Versus ECG-2 Dr Andreas Y. Andreou,**

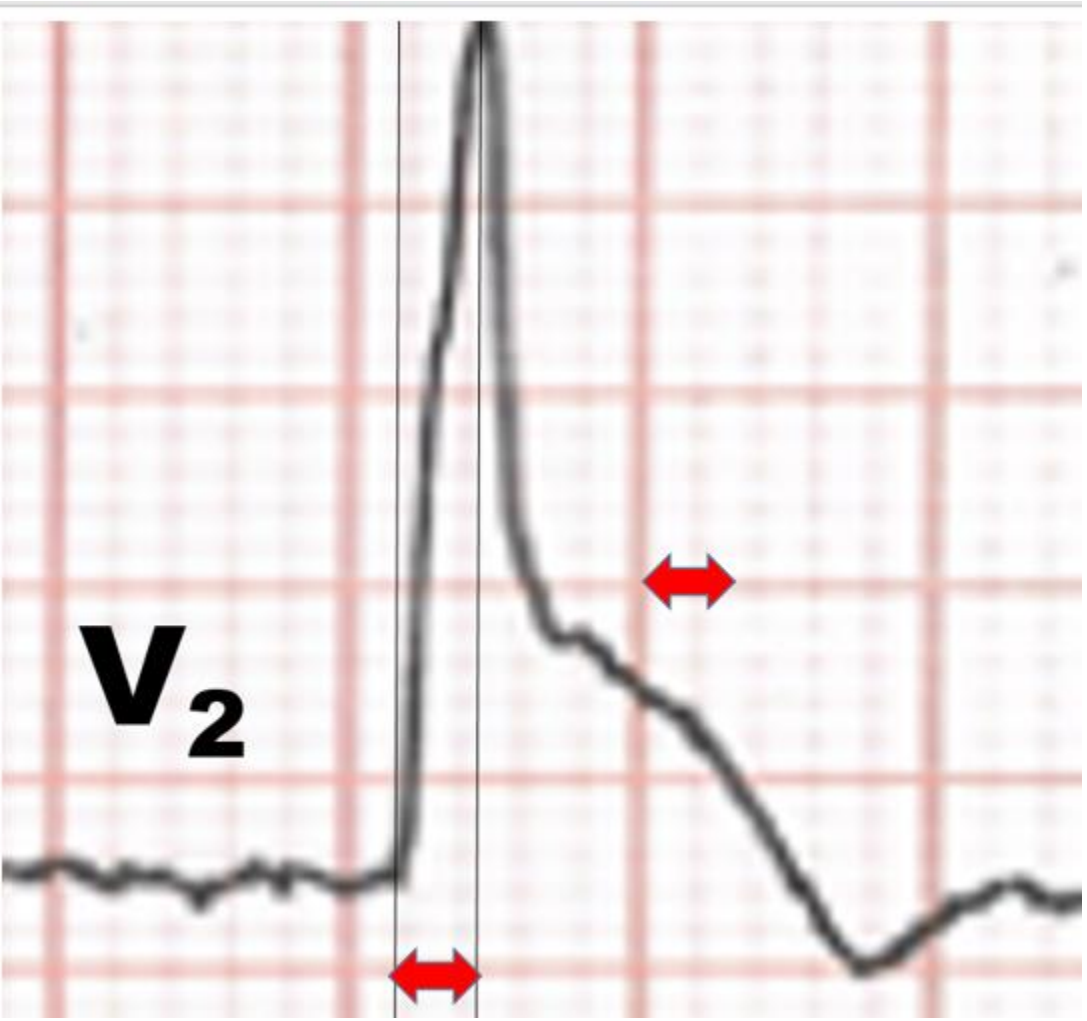
- Normal QRS duration or with a minor increase (up to 110 ms). When associated with other fascicular or bundle blocks it could be ≥ 120 ms. **Present in ECG-2 RBBB+LSFB**
- Frontal Plane leads with no modifications: normal QRS when isolated.
- Increased ventricular activation time (VAT), R-Wave Peak time(RWPT) or intrinsic deflection V1 and V2: ≥ 35 ms ; **Present in ECG-2**; See next slide
- R wave voltage of V1 ≥ than 5 mm ; **Present in ECG-2**;
- R/S ratio in V1 > 2;
- R/S ratio in V2 > 2;
- S wave depth in V1 < 5 mm;
- Possible small (embryonic) INITIAL q wave in V2 and V3 or V1 and V2; ; 

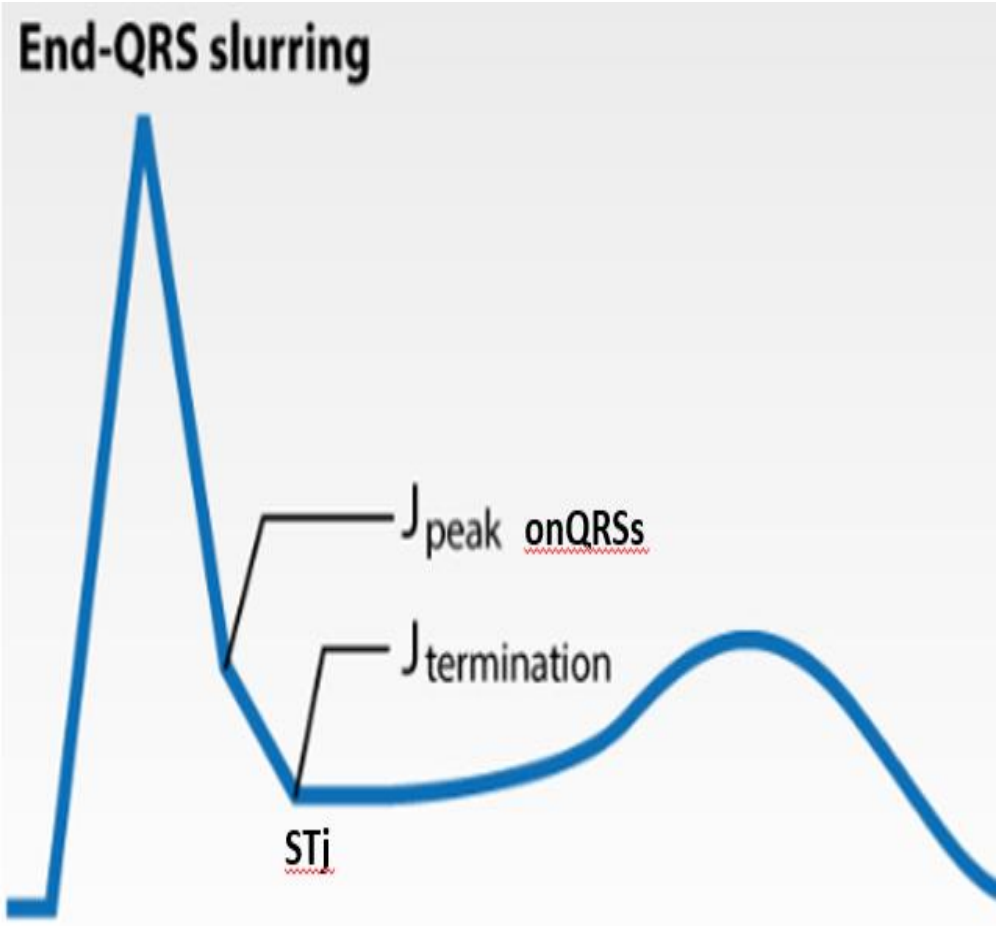
ECG-2 ;
- R wave of V2 > 15 mm: ; **Present in ECG-2**;
- RS or Rs pattern in V2 and V3 (frequent rS in V1) with R wave "in crescendo" from V1 through V3 and decreasing from V5 to V6; **Present in ECG-2**;



LSFB video English/Spanis

https://ekgvcg.files.wordpress.com/2014/09/image00.jpg





- Increased Ventricular Activation Time (VAT), R-Wave Peak Time(RWPT) or intrinsic deflection V1 and V2: **≥ 35 ms**

- Absence of q wave in left precordial leads V_5, V_6 and I (by absence of vector 1_{AM}). One first needs to exclude ILBBB, CLBBB and WPW; **Present in ECG-2 case**
- Intermittent or transient Prominent Anterior QRS forces PAF during hyperacute phase of myocardial infarction (Madias 1993), **vasospasm angina,** (**Present in ECG-2 trace**;)or during an exercise stress test in patients with severe myocardial ischemia (Moffa, Ferreira et al. 1997) (Uchida, Moffa et al. 2006) and during early atrial extrastimuli with some degree of ventricular aberration (Hoffman, Mehta et al. 1976) (Childers 1973)
- Appearance of intermittent, rate-dependent q wave in V_1 and V_2.

The last Brazilian Guidelines for Interpreting Rest Electrocardiogram provided the following criteria for ECG diagnosis of LSFB:

- QRS duration < 120 ms, in general, close to 100 ms. The appearance of LSFB does not increase QRSD by more than 25 ms, due to multiple interconnections between the fascicles of the LBB ("passageway zone" of Rosenbaum). The QRS complex is slightly prolonged between 100 ms to 115 ms. Thus, LSFB pattern with a prolonged QRSD indicates the presence of additional conduction disturbances such as other fascicular blocks, RBBB, MI, focal block, or a combination of these;
- ≥15 mm voltage R waves in V2 and V3 or from V1**Present in this ECG-2 case**;
- Increasing for all intermediary precordial leads and decreasing from V5 to V6 **Present in this ECG-2 case**;
- "r" wave jump may occur from V1 to V2 ("rS" in V1 for R in V2);

Abnormal ECG-waves: This group is eventually registered in pathological circumstances

1. The Delta wave. It is caused by preexcitation of the ventricles via an accessory pathway
2. The J wave (**Gussak, Bjerregaard et al. 1995**) also referred to as the J deflection, "the camel's hump"/ camel-hump sign (**Abbott and Cheitlin 1976**), "late delta wave", elevated J-point (**Yan and Antzelevitch 1996**), hathook junction, hypothermic wave, prominent J wave, K wave, H wave, current of injury or Osborn wave.
   - Hypothermal or cool wave (**Maruyama, Kobayashi et al. 2004**) (**Ortak and Bonnemeier 2007)**;
   - Normotermic J-wave
3. The epsilon wave, right precordial epsilon potentials or Fontaine wave: Its wave constitute a mayor criteria for the diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) (currently polemic) (**Wang, Yang et al. 2009, Wu, Wang et al. 2009**).
4. The lambda or Gussak wave (**Gussak, Bjerregaard et al. 2004**) (**Gussak, Bjerregaard et al. 2004**).

Classification of J waves

I) J wave of hypothermia

II) J wave in normothermal patients:

1. Hypercalcemia
2. Injuries in the central nervous system: subarachnoid hemorrhage, post-heart arrest and in cervical sympathetic system dysfunction
3. Early repolarization syndrome
4. "Brugada "entities":
   - ❑ Familial cases (~17%): true Brugada disease;
   - ❑ Sporadic cases (~63%): Brugada syndrome
   - ❑ Brugada phenocopies (BrPs): are clinical entities that present with an ECG pattern identical to either the type-1 or type-2 Brugada patterns, yet differ etiologically from true congenital BrS. The pattern presents in association with an identifiable condition and, upon resolution of that condition, the ECG pattern normalizes. BrP may not be due to the same sodium channel abnormality as BrS, or may be only transient while the underlying condition persists. Indeed, the defining feature of BrP is the absence of true congenital BrS.
5. Congenital Short QT syndrome;
6. Idiopathic Ventricular Fibrillation;
7. Concealed forms of arrhythmogenic dysplasia of the right ventricle
8. **Variant angina or Prinzmetal angina the present case ECG-2**

Heterogeneous distribution of a transient outward current-mediated spike-and-dome morphology of the action potential across the ventricular wall underlies the manifestation of the electrocardiographic J wave.

The presence of a prominent action potential notch in epicardium but not endocardium is shown to provide a voltage gradient that manifests as a J (Osborn) wave or elevated J-point in the ECG (Yan and Antzelevitch 1996).

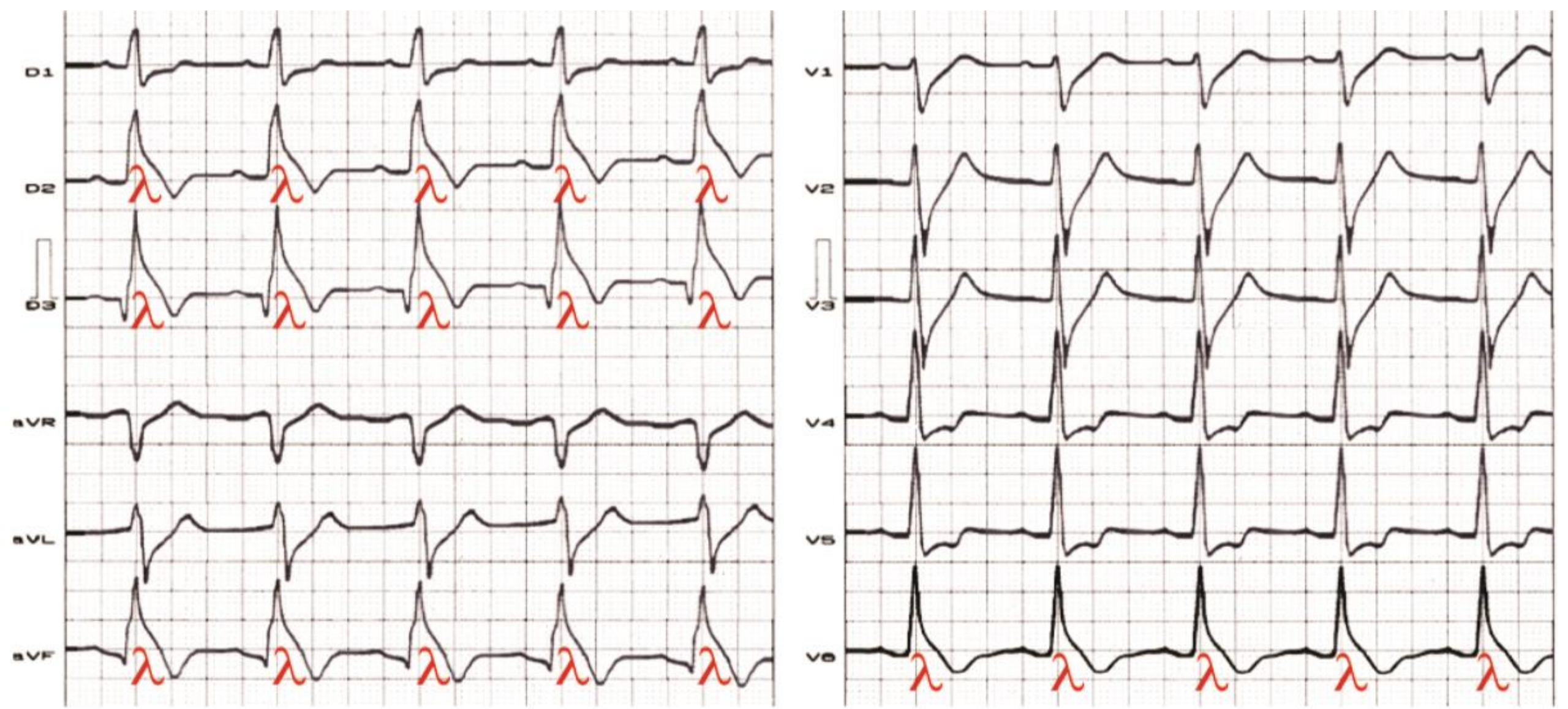
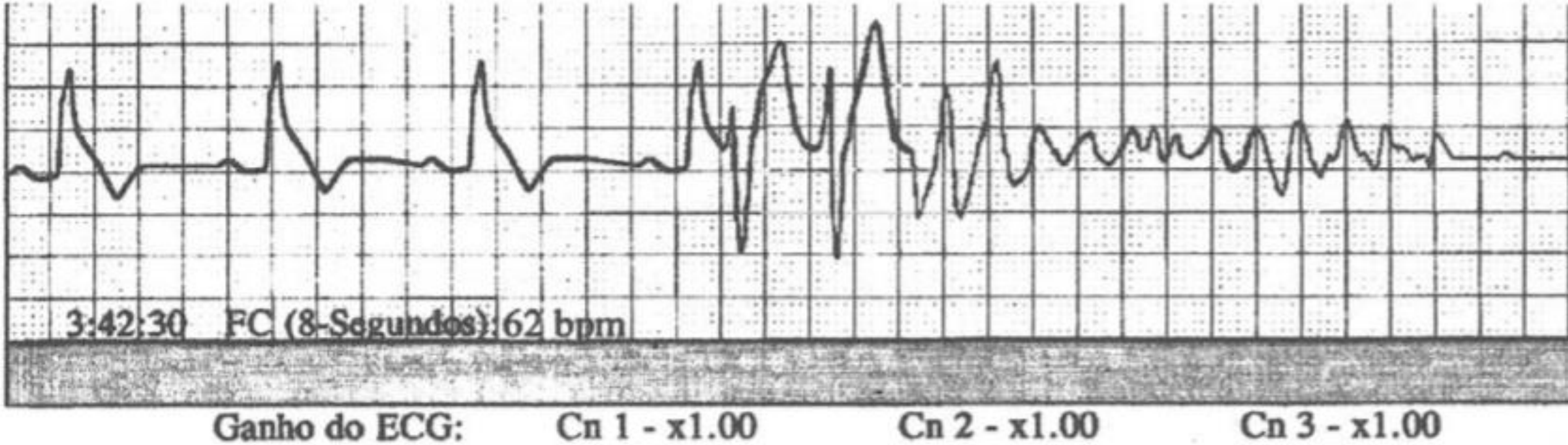


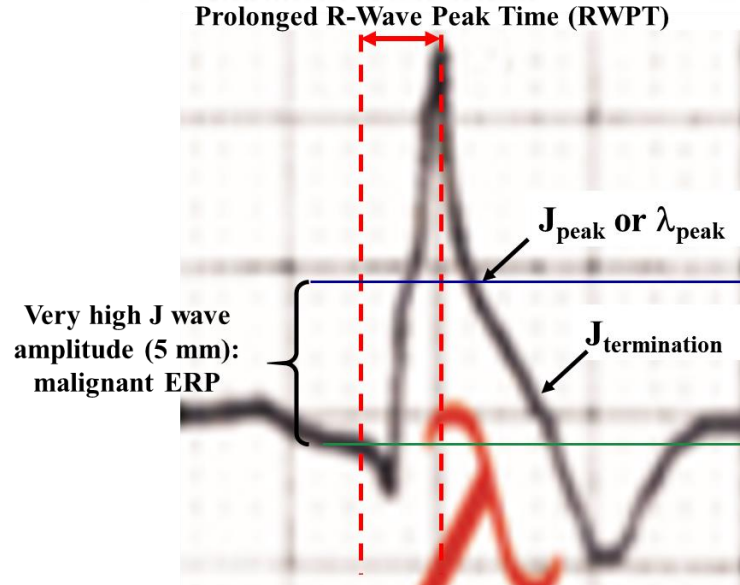
Figure. 12-lead ECG showing persistent ST segment elevation in the inferior and lateral leads, associated with concomitant reciprocal or mirror image in the anterior wall, which was not modified with the use of sublingual nitrate in absence of hypothermia, electrolyte imbalance ischemia or brain injury. **(Riera AR 2004 J Electrocardiology**.)

24-hour Holter monitoring of the same patient shows a very short coupled PVC with early degeneration in VF and CA (Figure).



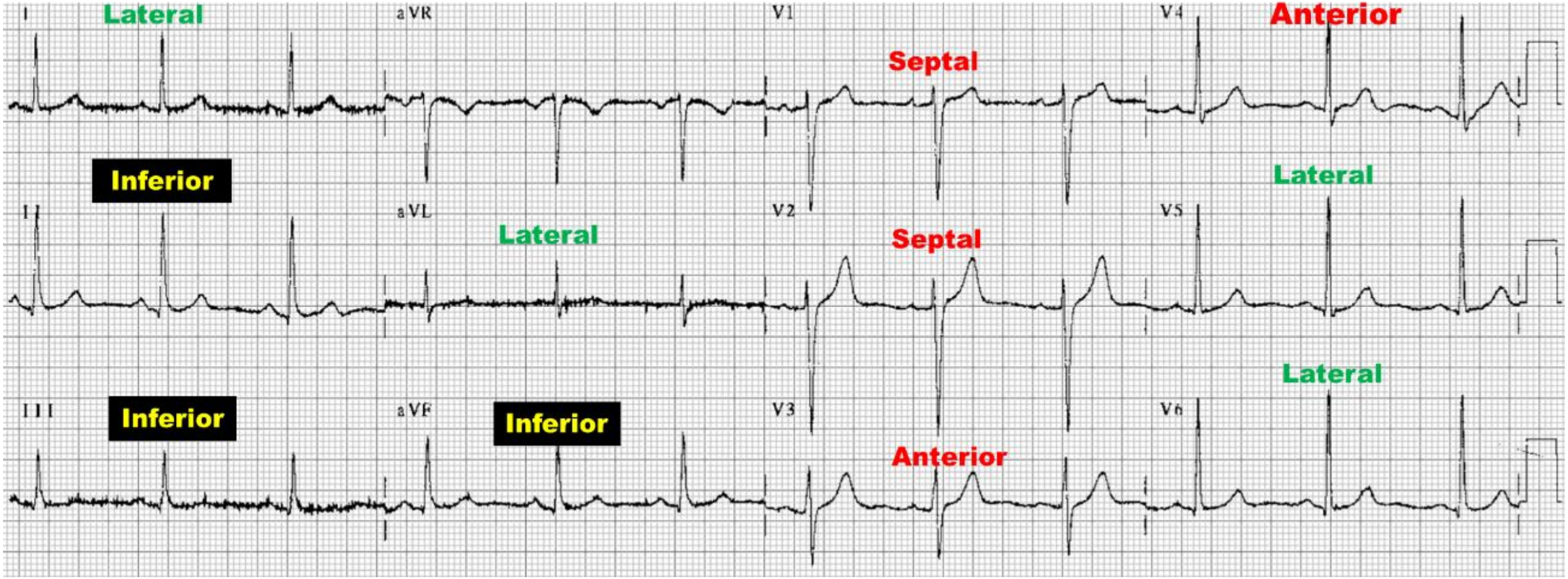
Sudden cardiac death by idiopathic polymorphic ventricular tachycardia/IVF

with short coupling ending in cardiac arrest



Types of ERP

There are three types of ERP: **Type 1** that displays an ERP predominantly in the lateral precordial leads, prevalent among healthy young male athletes; **Type 2** that displays an ERP in the inferior and lateral leads. It is associated with moderate level of risk; **Type 3** that displays an ERP in the inferior, lateral and right precordial leads (anteroseptal). This type has highest level of risk (Figure).



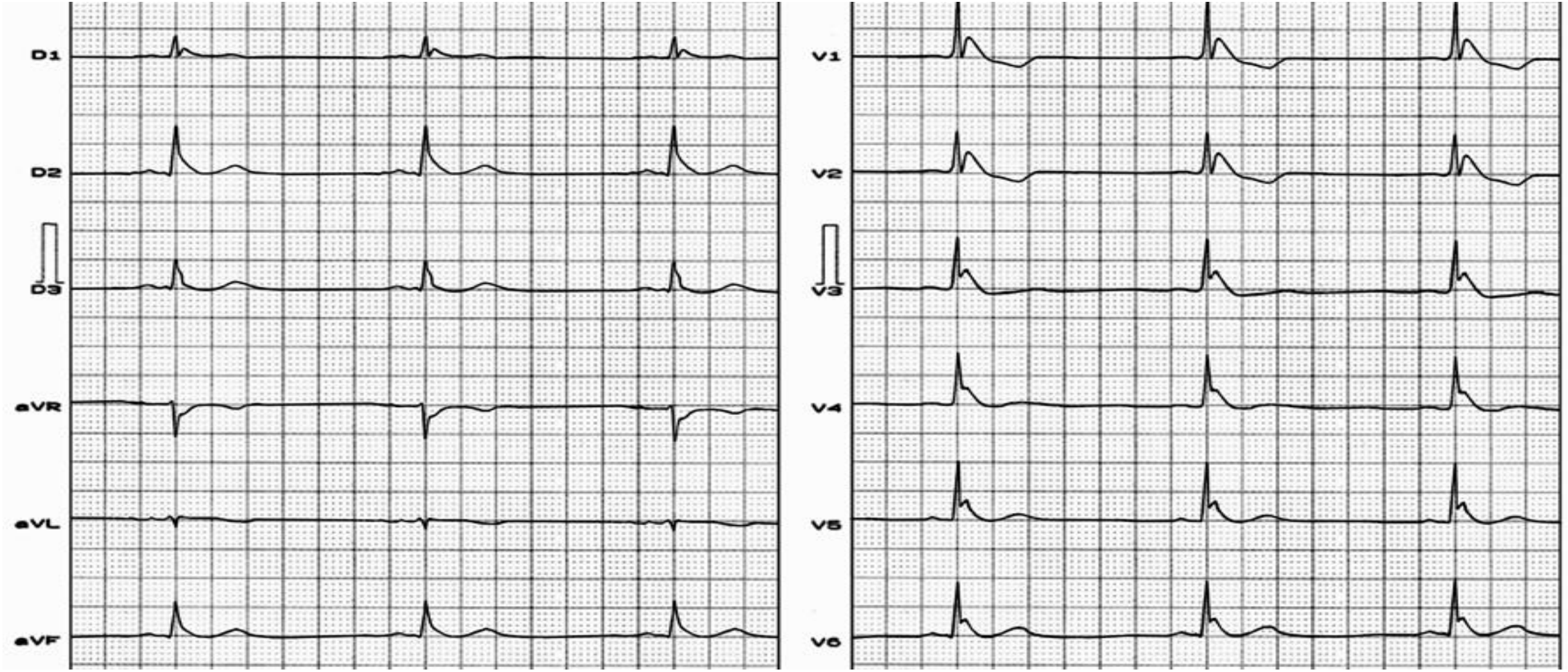
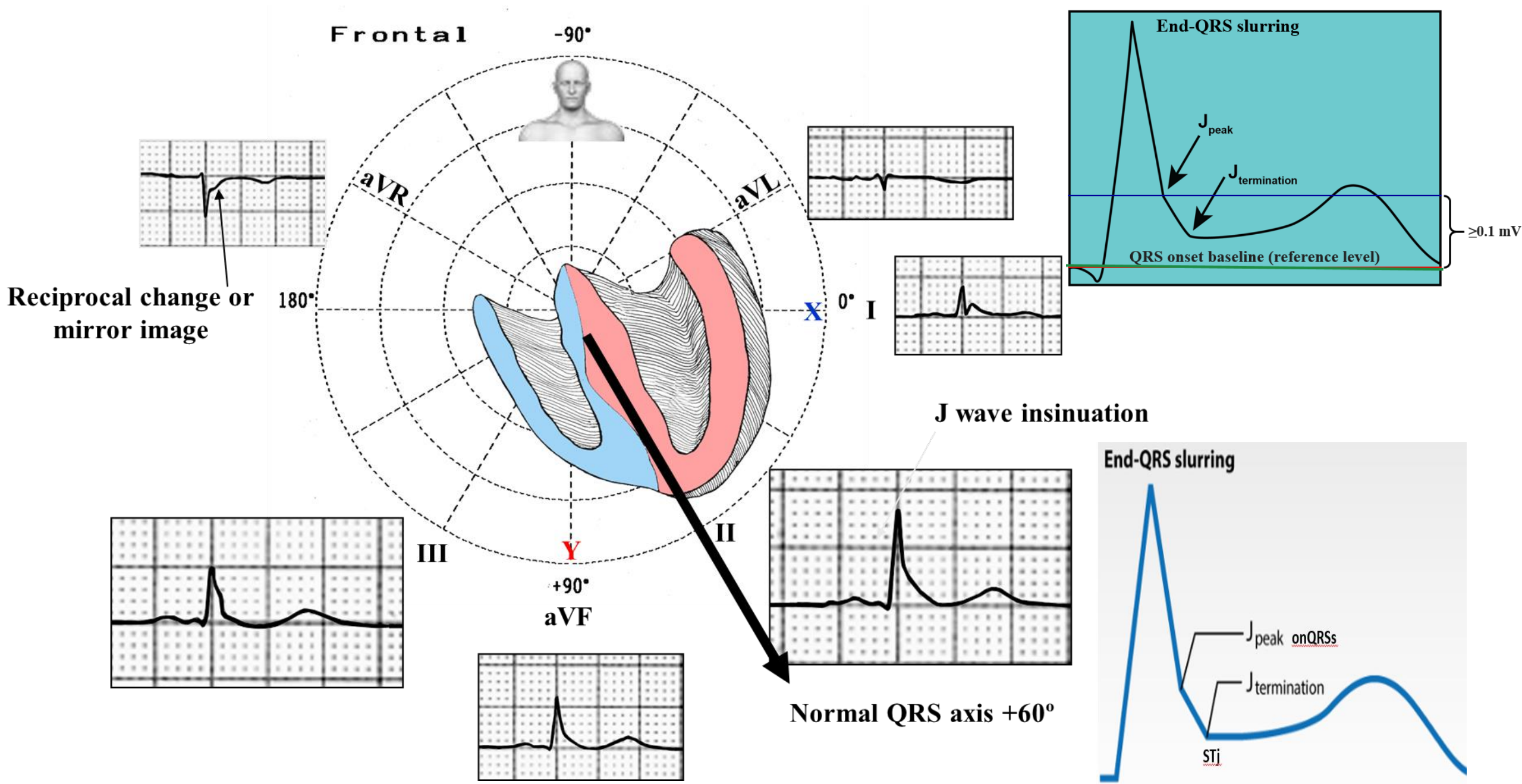
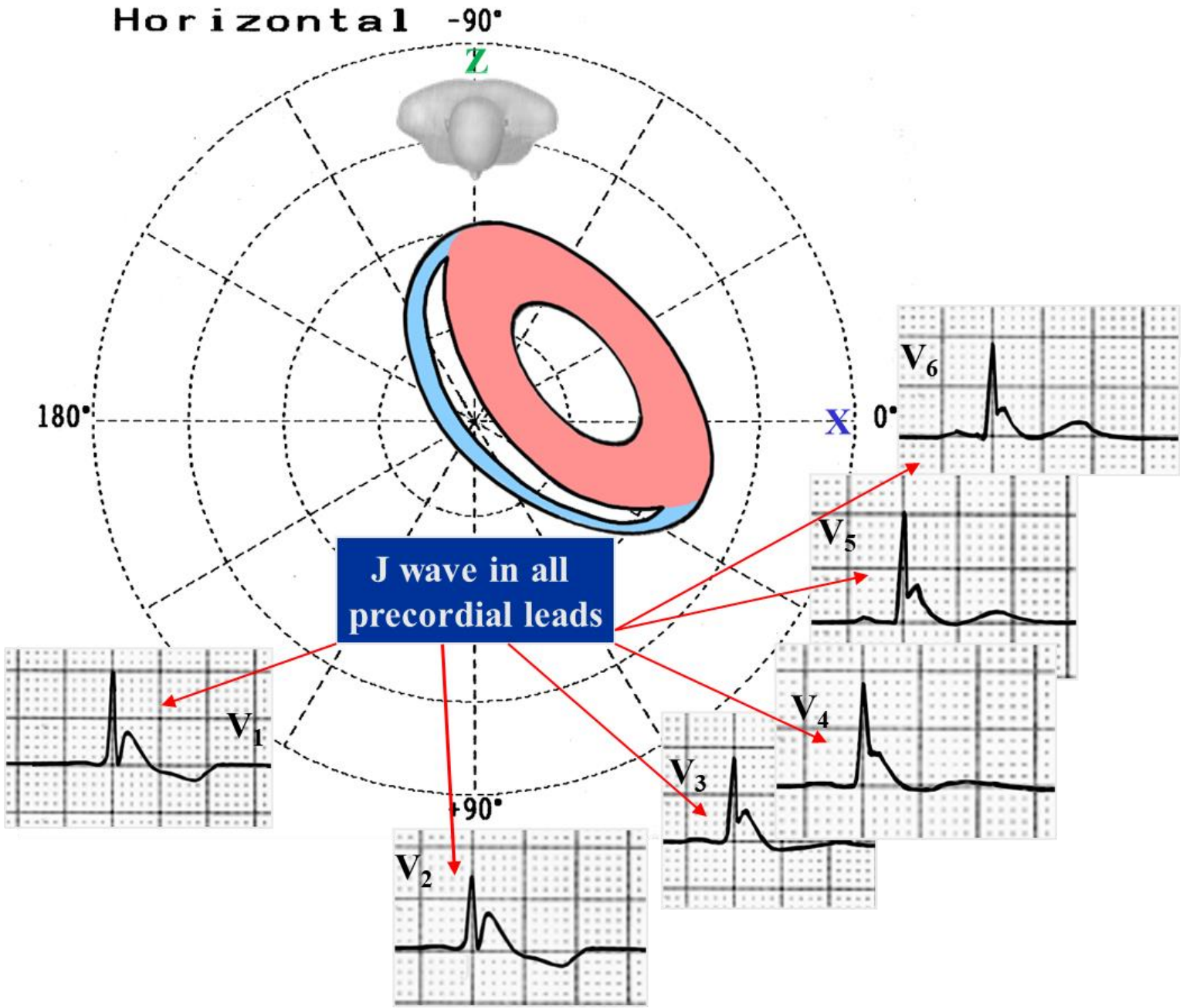
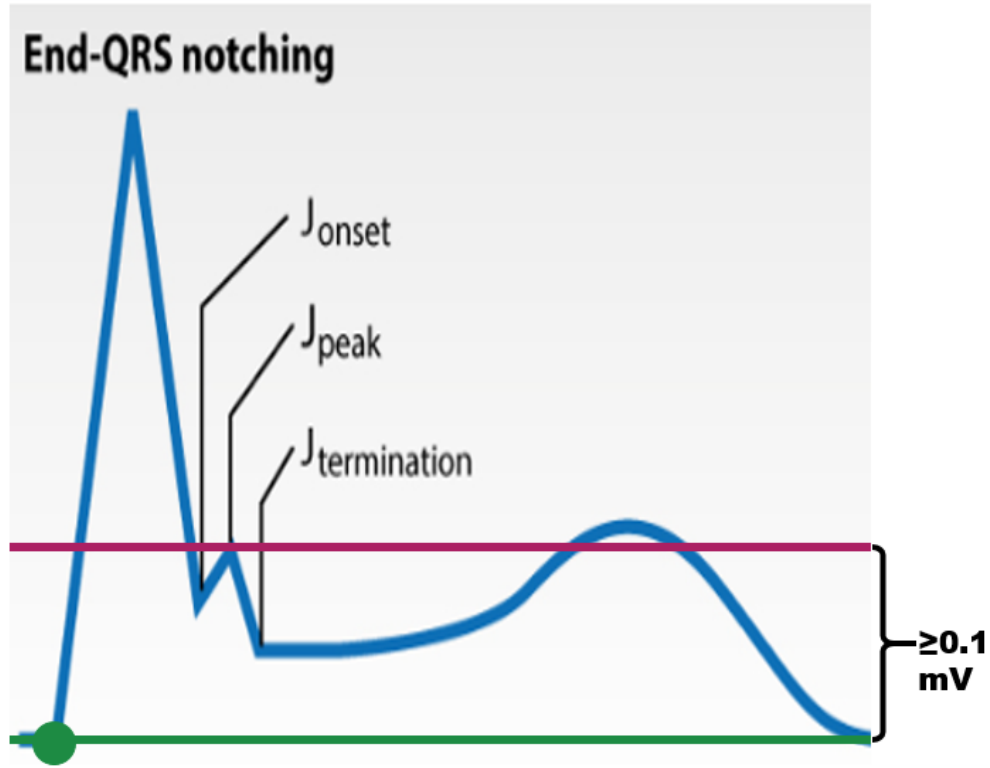


Figure 12 leads ECG of an ERP in the inferior, lateral and right precordial leads (type 3). This variant is associated with the highest level of risk for the development of VF storms. In type 3, the Brugada waves may be seen together with giant J waves in other ECG leads. Although the Brugada waves are not called ER, their underlying mechanism is identical to that of the ERPs. CDI implantation was proposed but the patient refused. Consequently, oral quinidine (1500 mg/day) was administered.



Classical case of Type 3 Early Repolarization Pattern (ERP) (**Yan 1996; Antzelevitch 2005**)





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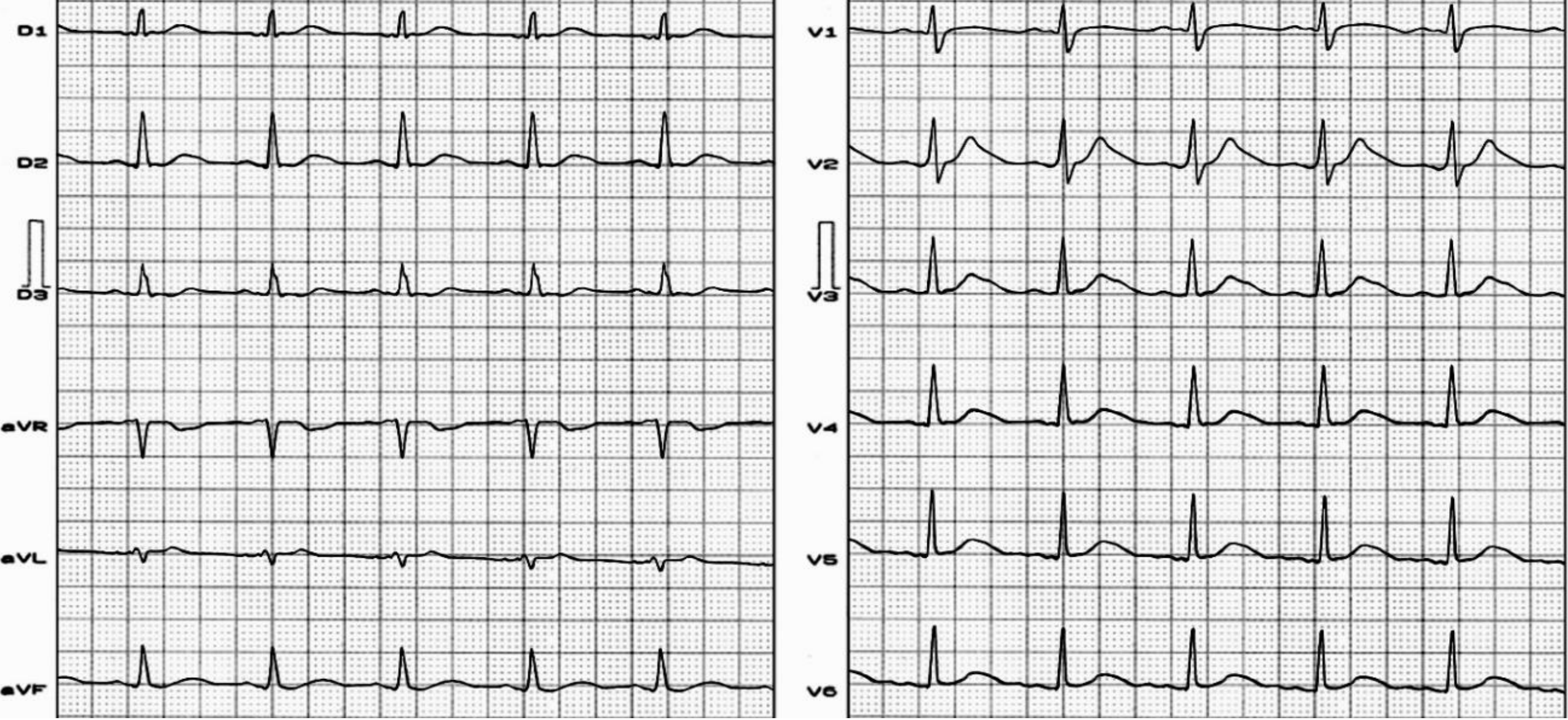
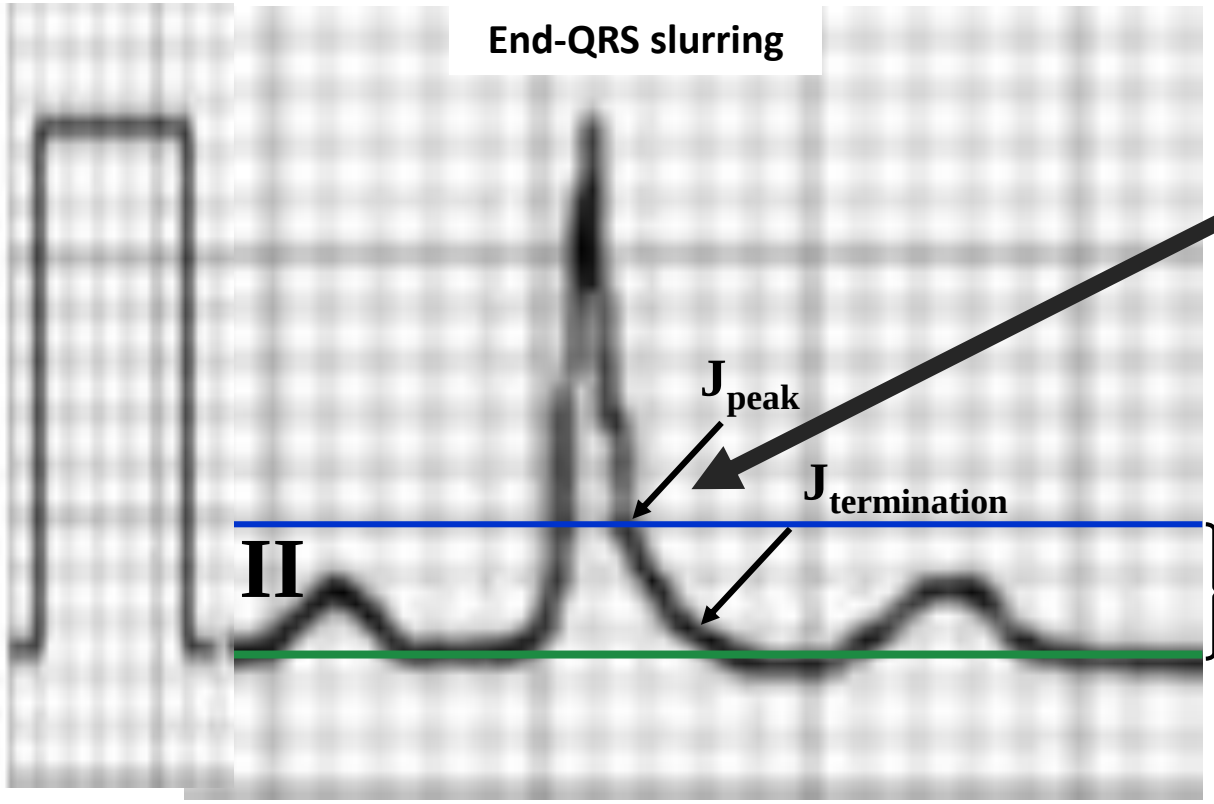
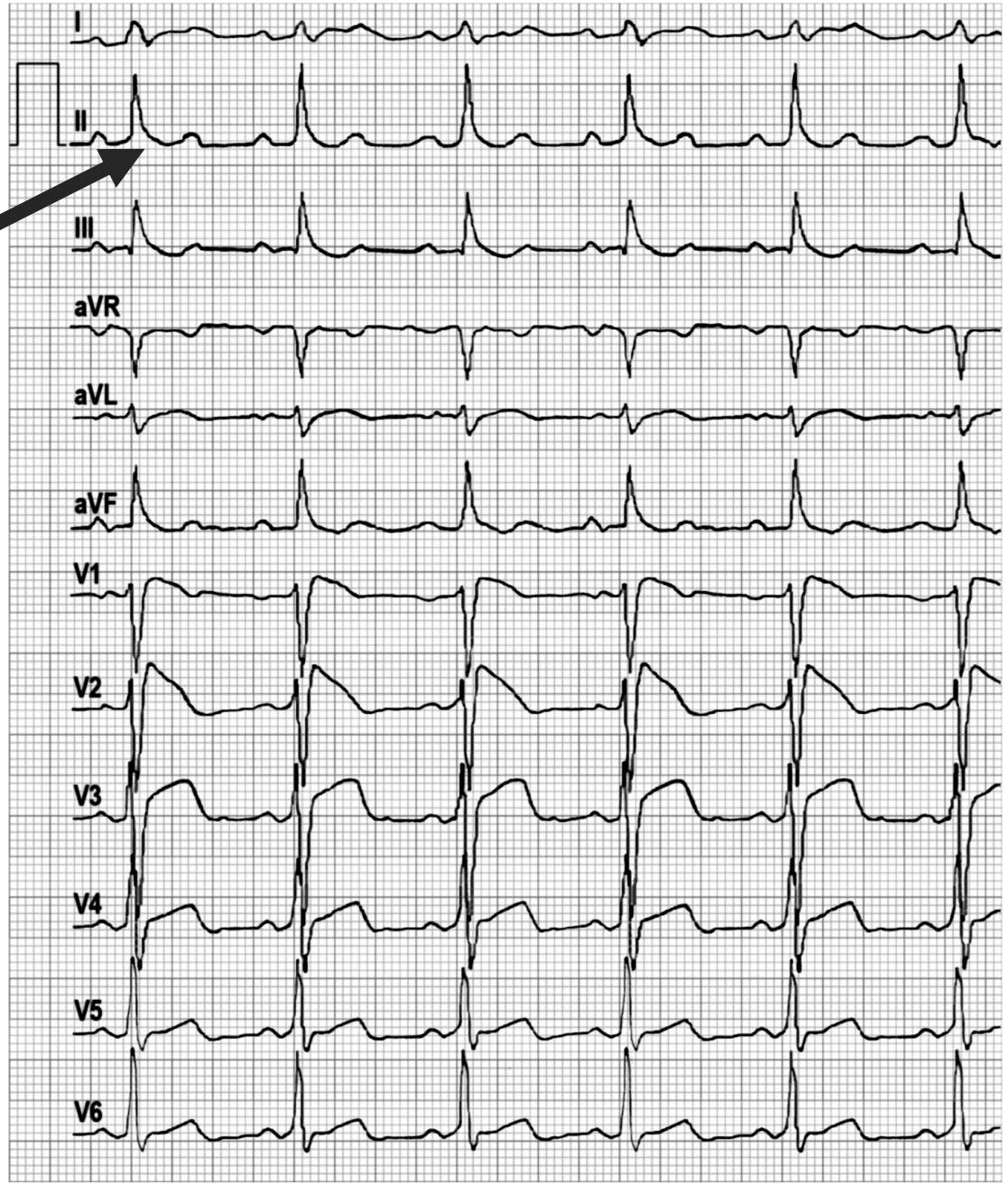


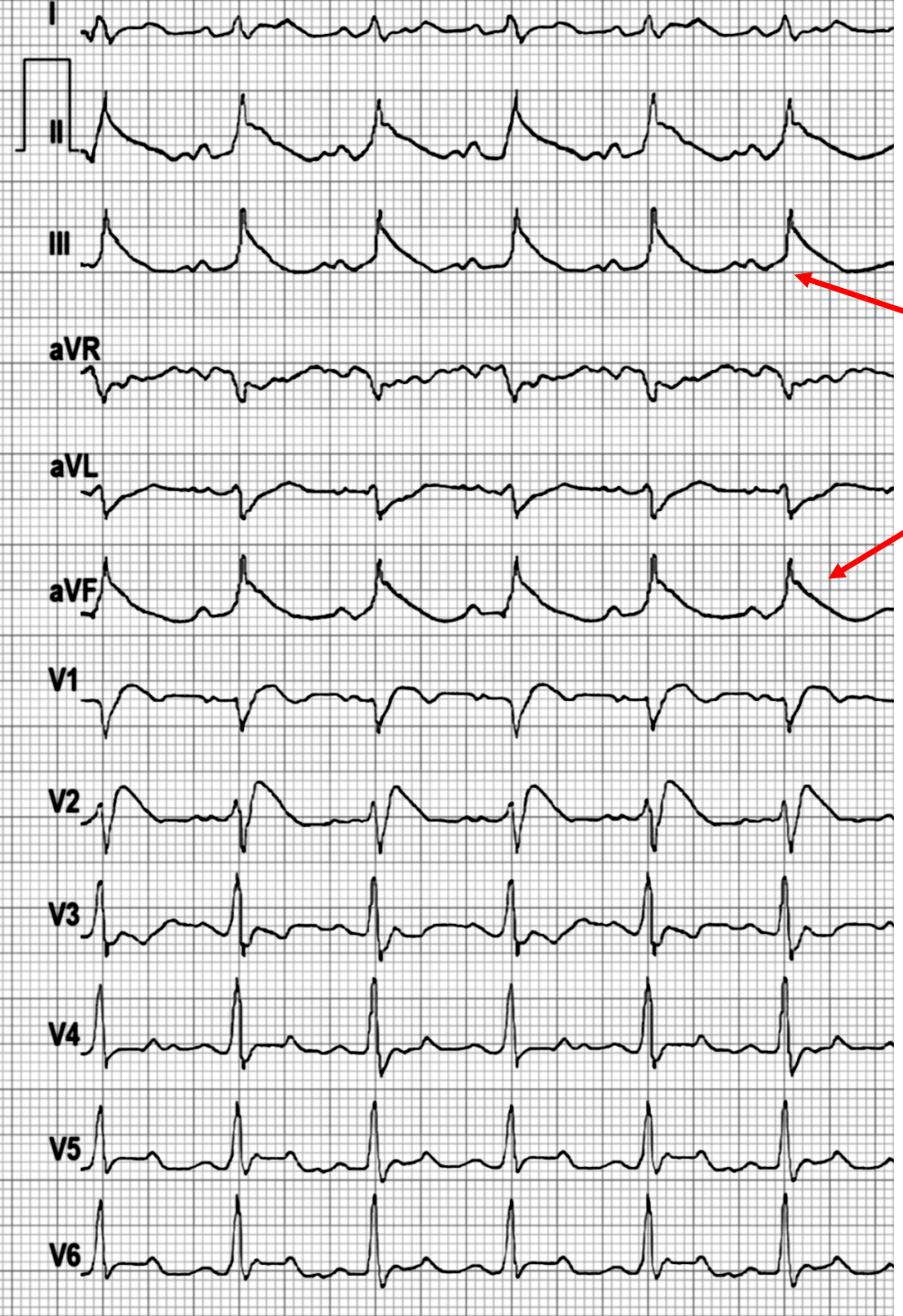
Figure shows an ECG performed two days after quinidine administration. Quinidine reduces the magnitude of the Ito channel – mediator of phase 1 and consequently normalize the ST segment elevation. Additionally, this drug could improve repolarization due to its vagolytic effect (M2 muscarinic receptor block) and to the exacerbation of reflex sympathetic tone).

Before ajmaline test

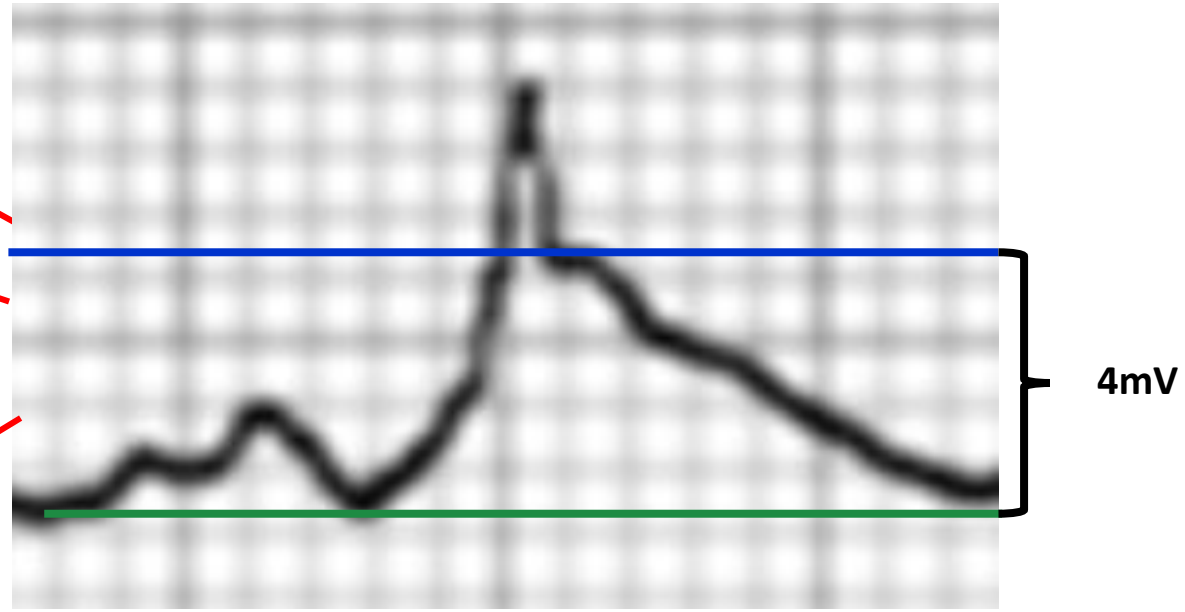


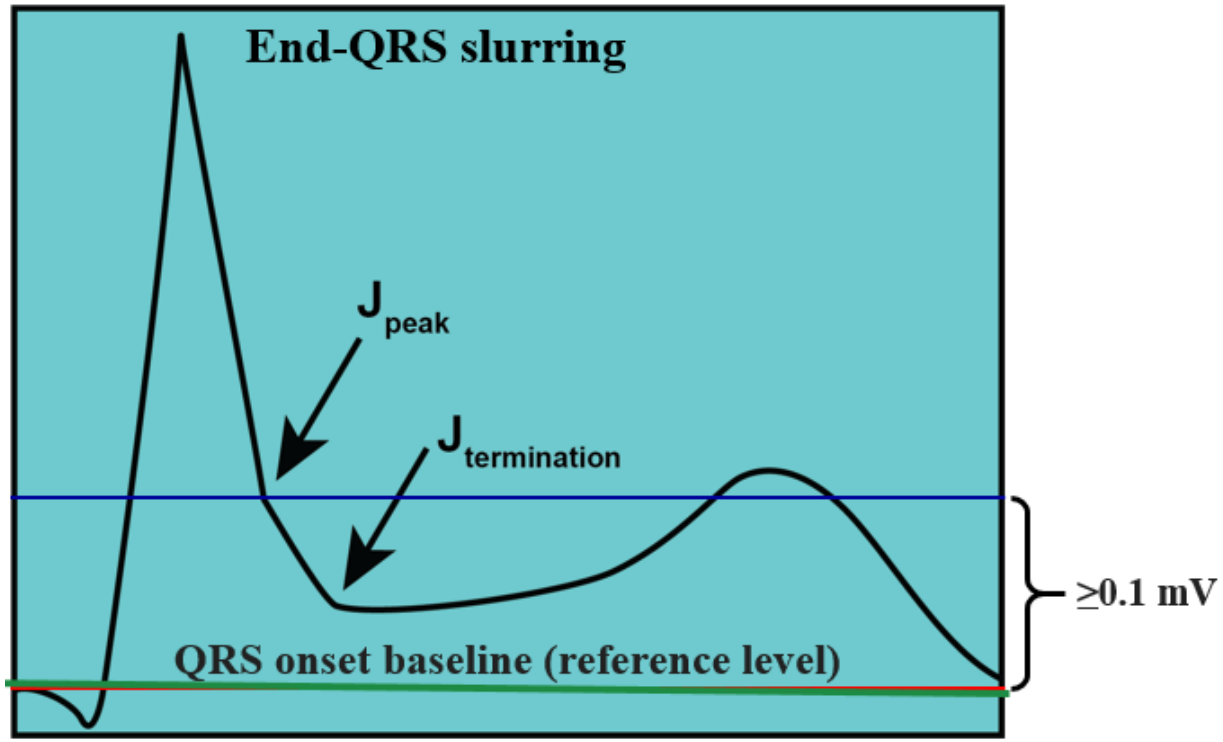


Type 1 Brugada pattern and slurring J-wave insinuation in the inferior leads (before ajmaline injection).



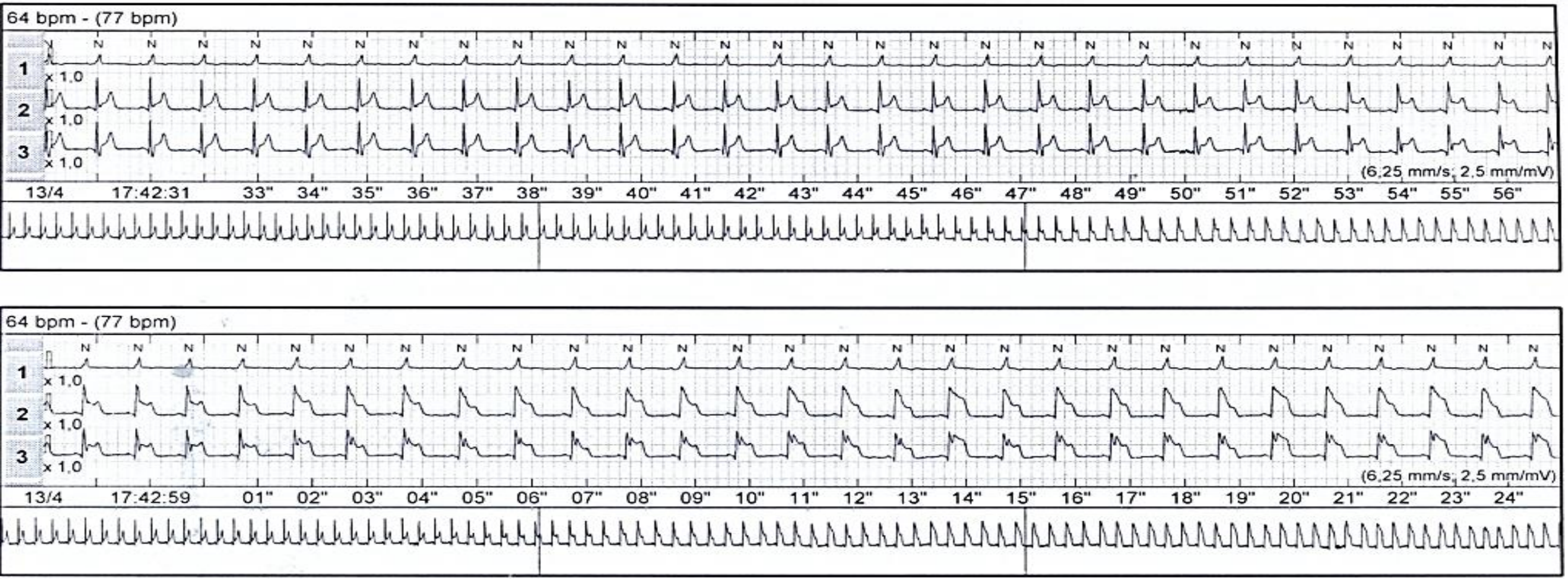
After ajmaline test

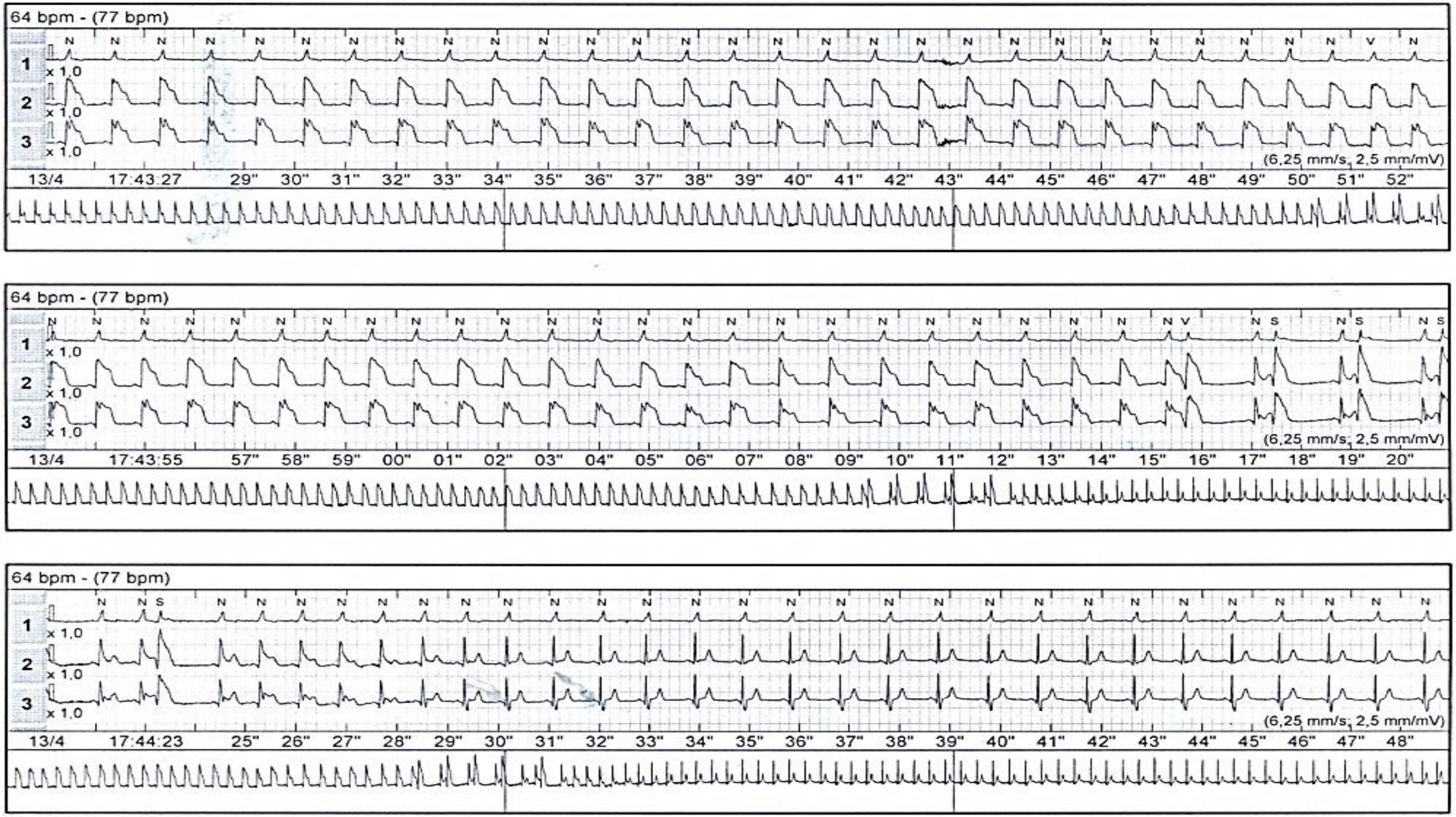


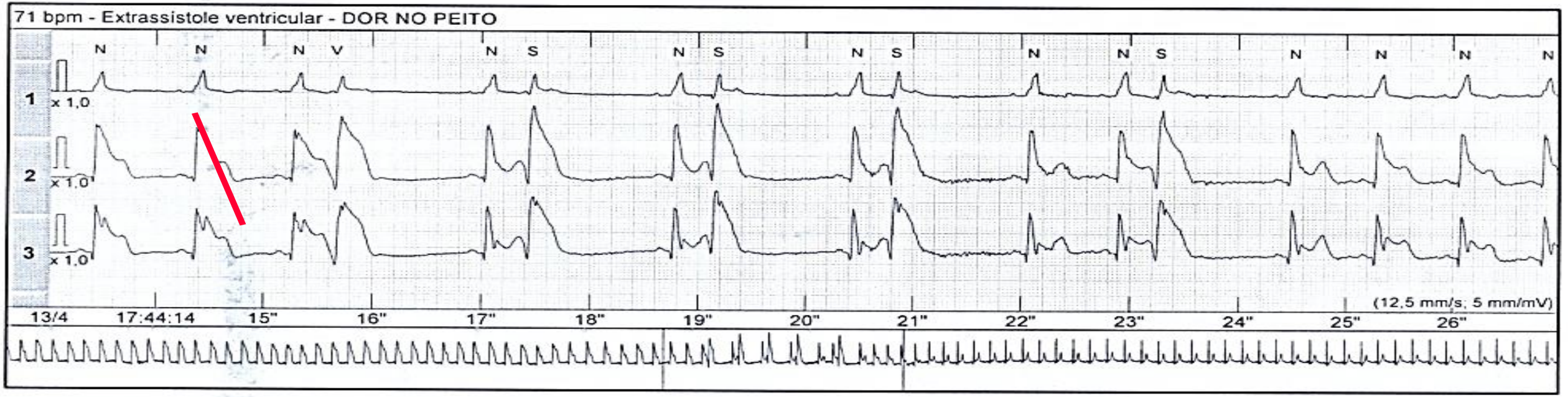


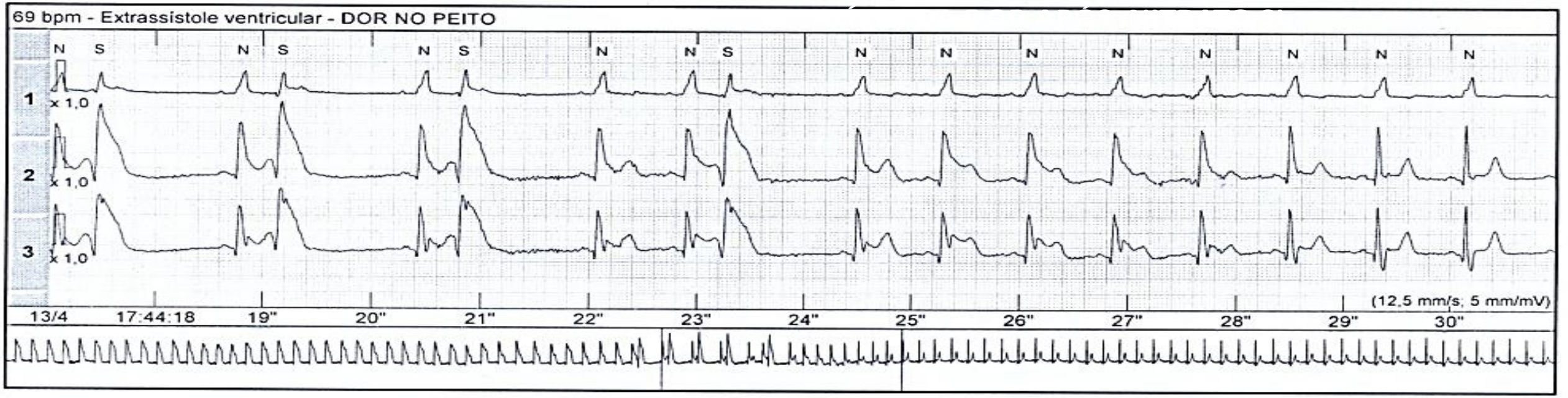
Man who had coronary revascularization a time ago. Continuous Holter monitoring during an episode of angina and concomitant ST segment elevation and ischemic giant J-wave "lambda-like type" associated with Premature Ventricular Contractions with bigeminy sequence and very short coupling. The PVCs disappear immediately after cessation of vasospastic ischemia with administration of sublingual nitrate.

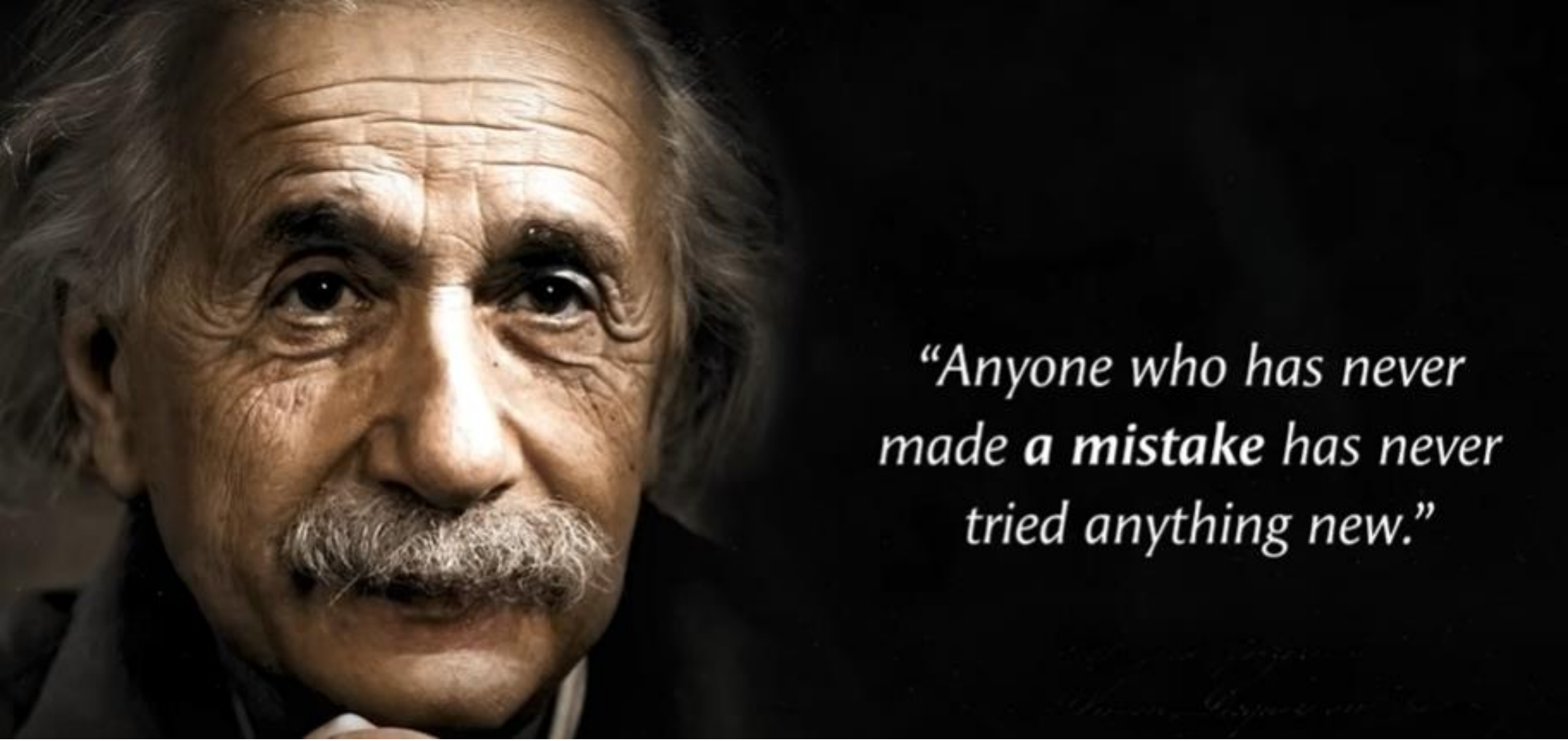
Holter monitoring very similar the present case ECG-2 Prinzmetal angina




64 bpm - (77 bpm)
1
2
3
x 1,0
(6,25 mm/s; 2,5 mm/mV)
13/4 17:43:27 29" 30" 31" 32" 33" 34" 35" 36" 37" 38" 39" 40" 41" 42" 43" 44" 45" 46" 47" 48" 49" 50" 51" 52"
64 bpm - (77 bpm)
1
2
3
x 1,0
(6,25 mm/s; 2,5 mm/mV)
13/4 17:43:55 57" 58" 59" 00" 01" 02" 03" 04" 05" 06" 07" 08" 09" 10" 11" 12" 13" 14" 15" 16" 17" 18" 19" 20"
64 bpm - (77 bpm)
1
2
3
x 1,0
(6,25 mm/s; 2,5 mm/mV)
13/4 17:44:23 25" 26" 27" 28" 29" 30" 31" 32" 33" 34" 35" 36" 37" 38" 39" 40" 41" 42" 43" 44" 45" 46" 47" 48"


71 bpm - Extrassistole ventricular - DOR NO PEITO
N N N V N S N S N S N N S N N N N
1
x 1,0
2
x 1,0
3
x 1,0
(12,5 mm/s; 5 mm/mV)
13/4 17:44:14 15" 16" 17" 18" 19" 20" 21" 22" 23" 24" 25" 26"


69 bpm - Extrassistole ventricular - DOR NO PEITO
N S N S N S N N S N N N N N N N N
1
x 1,0
2
x 1,0
3
x 1,0
(12,5 mm/s; 5 mm/mV)
13/4 17:44:18 19" 20" 21" 22" 23" 24" 25" 26" 27" 28" 29" 30"


“Anyone who has never
made a mistake has never
tried anything new.”