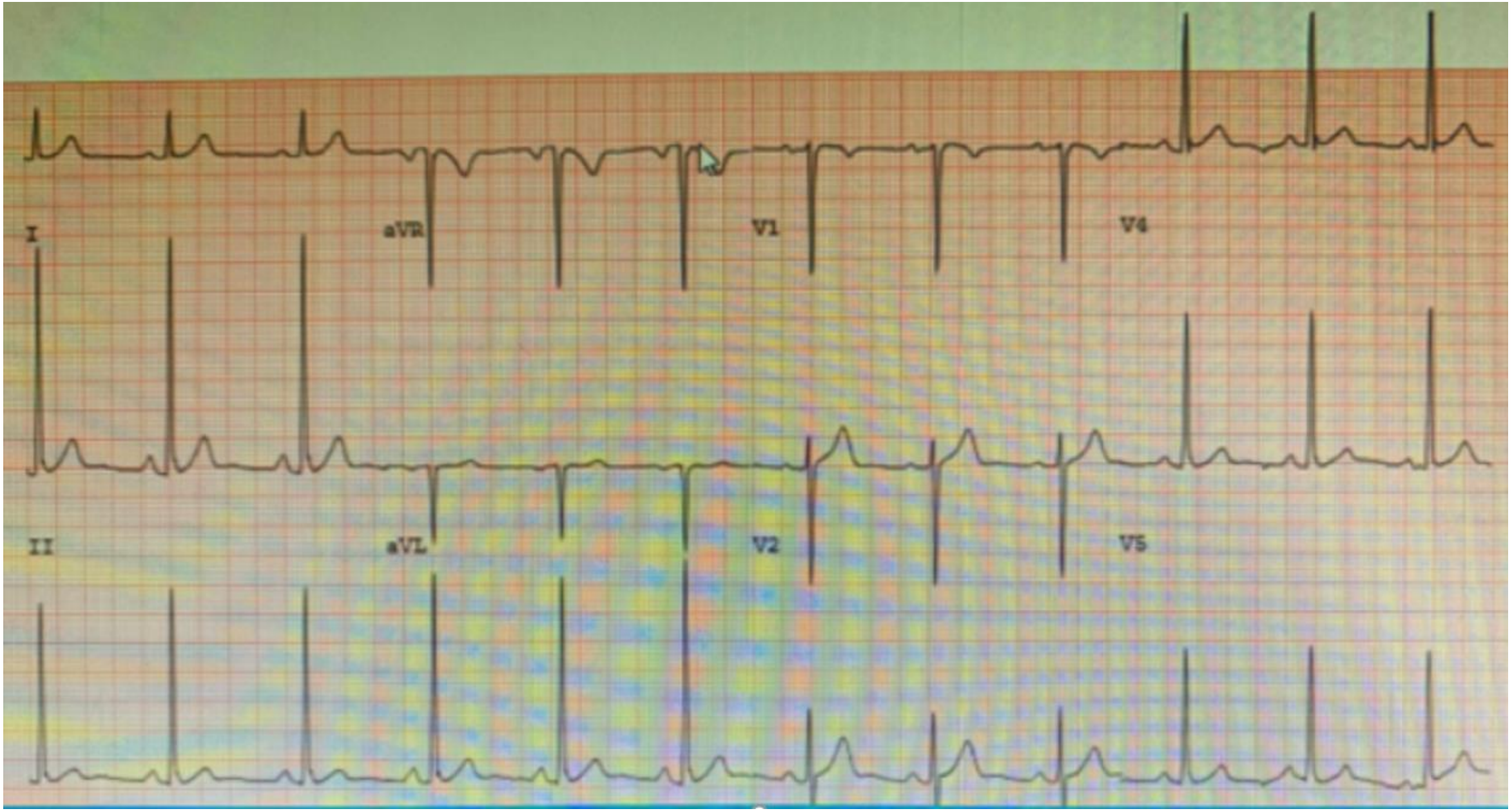
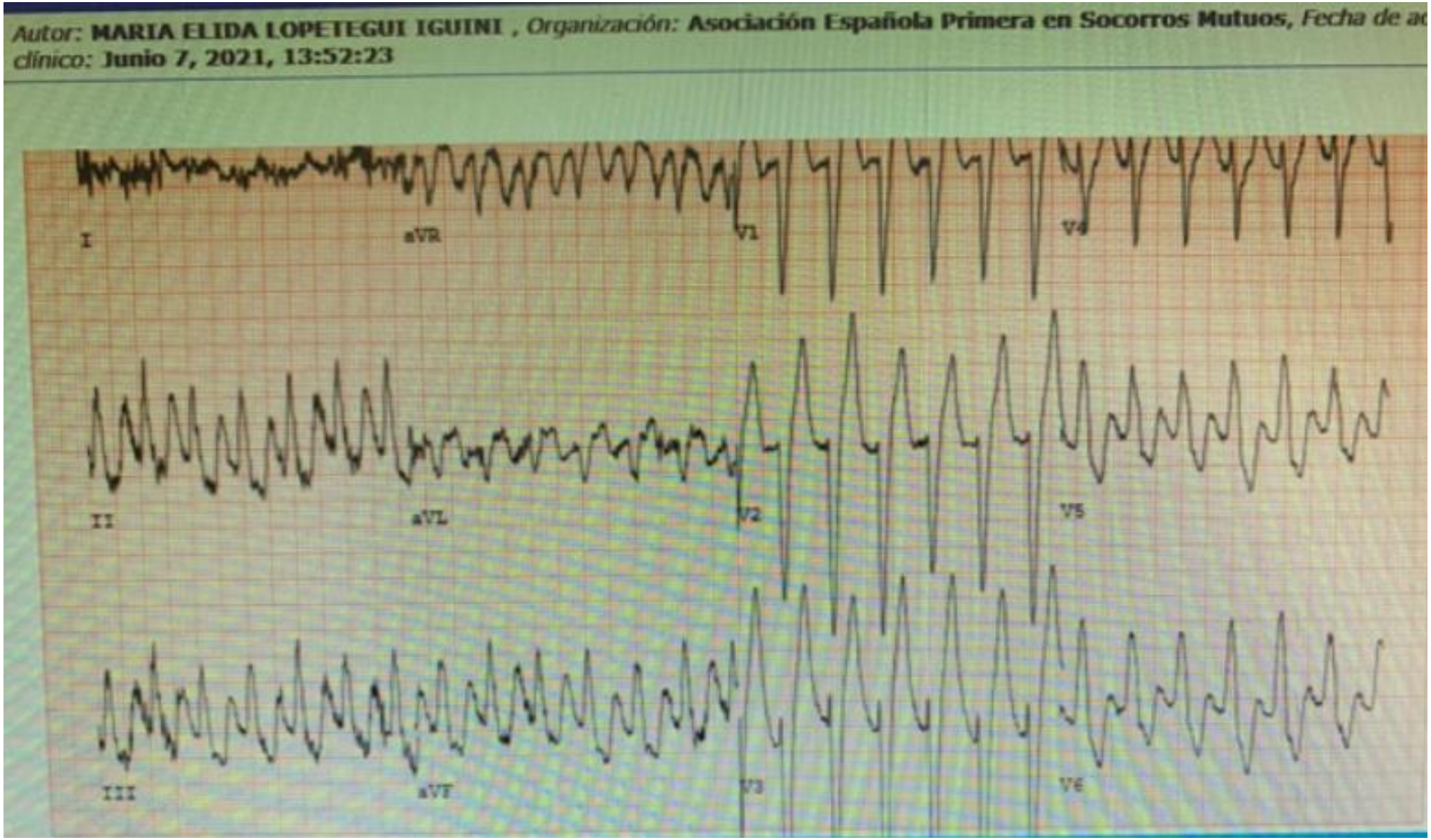
Dr Óscar Díaz Arresto case Mater Atleta odiazarn@GMAIL.COM

58-year-old master marathoner, Trace preformed at rest





58-year-old master marathoner, Trace preformed 1st minute of the post-effort exercise at June 7, 2021 13 hours, 52 minutes and 13 seconds Sustained wide supraventricular tachycardia est (WCTs) with some degree of left bundle branch block HR ≈150 bpm.

Over the last 5-10 years I have become aware of a number of former elite endurance athletes having heart issues. This is counter intuitive given endurance athletes are considered to have strong hearts. However, over the last 15 years research is increasingly showing that the incidence of arrhythmias is higher in athletes, especially in elderly athletes with a lifelong training history in marathons, ultra-marathons, ironman distance triathlons and long distance bicycle races. An arrhythmia is any change from the normal sequence of electrical impulses in the heart. The electrical impulses may happen too fast, too slowly (bradycardia), or erratically so that the heart can't pump blood effectively.

Bradycardia, defined by a resting heart rate <60 beats min , is the most frequent rhythm disturbance in response to endurance training where the resting heart rate can be ~30 bpm and even lower at night. Cyclists Sir Chris Hoy and Tour de France winner Miguel Undrain reportedly had resting heart rates of 30 and 28 beats per minute. Although the bradycardia is usually a harmless adaptation to endurance training, it can become a pathological condition. It was previously thought to affect the electrical activity of the heart that starts in what is called the sinus node (see photo) which is an area of specialized cells in the upper right chamber of the heart that controls the rhythm of your heart.

The most compelling evidence of a link between endurance training and sick sinus syndrome comes from a study of former professional cyclists. Their average heart rate was lower, sick sinus syndrome was more frequent, and pacemaker implantation for bradyarrythmias was more frequent relative to a control group with matched cardiac risk factors. Similarly, a high incidence of pacemaker implantation has been reported in elderly marathon runners.

Historically, this slowing of the heart rate was thought to be the result of a change in the nervous system stimulation of the heart muscle through the sinus node, the pacemaker structure in the heart muscle itself. However, an animal study is the first to show that the heart rate adaption to exercise training is not the result of changes in this nervous system control of the heart, and instead is primarily the result of a training-induced remodelling of the sinus node within the heart itself.

Causes of wide complex tachycardias (WCTs) in patients without structural heart disease

Monomorphic configuration

Supraventricular tachycardia (SVT)

• Bundle branch block

- functional (RBBB more often than LBBB)

- pre-existing

- rate related

• Antidromic (i.e. retrograde conduction over AV node;

• Non-specific conduction delay

- class I or class III antiarrhythmic drugs

- electrolyte imbalance

Ventricular tachycardia (VT)

• LBBB, inferior axis idiopathic right ventricular VT

• RBBB, superior axis: idiopathic left ventricular VT

• Pacemaker mediated VT

Polymorphic configuration

Supraventricular tachycardia

Common Types of Supraventricular Tachycardia and Usual Characteristics

AVNRT: Most common SVT (approximately 50 to60%)[4]Occurs more often in younger women.

Mechanism: Reentry caused by nodal pathways or tracts (two types): atypical (fast/slow) represents 10% and typical (slow/fast) represents 90% of all AVNRT.

Possible ECG changes: Rate: 118 to 264 bpm, Rhythm: regular, narrow QRS complex (< 120 msec); regular, wide QRS complex (≥ 120 msec); may not see any P-wave activity in either type (atypical or typical)Atypical AVNRT: RP interval > PR interval; P waves negative in leads III and Avf, Typical AVNRT: RP interval < PR interval; pseudo R wave in lead V_1 with tachycardia, not with normal sinus rhythm; pseudo S wave in leads I, II, and aVF.

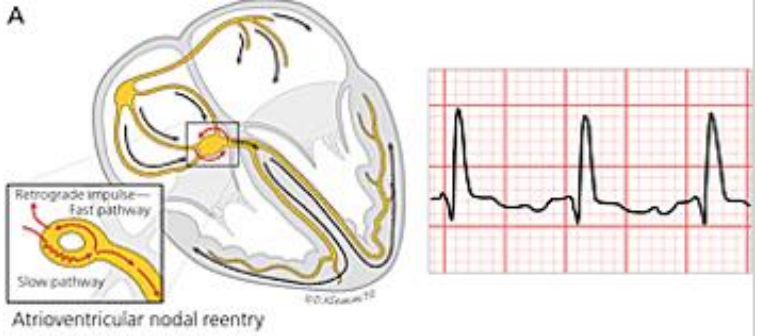
AVRT: Second most common SVT (approximately 30%)[4,5]Orthodromic most common type (81 to 87%)Occurs more often in younger women and children. May be comorbid with Wolff-Parkinson-White syndrome. Reentry caused by accessory pathways (two types): orthodromic (antegrade conduction through atrioventricular node) and antidromic (retrograde conduction through atrioventricular node). Rate: 124 to 256 bpmRhythm: regular, narrow QRS complex common (orthodromic); regular, wide QRS complex uncommon (orthodromic or antidromic) if bundle branch block or aberrancy present. Orthodromic AVRT: RP interval < PR interval or RP interval > PR interval with a slowly conducting accessory pathway; retrograde P waves (leads I, II, III, aVF, V_1); delta wave seen with normal sinus rhythm, not with tachycardia. Antidromic AVRT: short RP interval (< 100 msec); regular, wide QRS complex (≥ 120 ms); delta waves seen with normal sinus rhythm and tachycardia; concealed accessory pathways do not show delta waves.

AT = atrial tachycardia:Third most common SVT (approximately 10%)

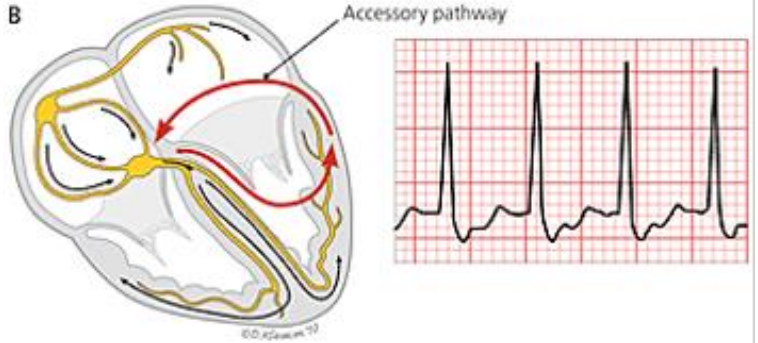
Two types: AT and multifocal ATAT has two forms: focal and macroreentrant Multifocal AT occurs more often in middle age or in persons with heart failure or chronic obstructive pulmonary disease.

Mechanism: Reentry (micro), automaticity, or triggered activity: focal AT (reentry, automaticity, or triggered activity); multifocal AT (automaticity activity)

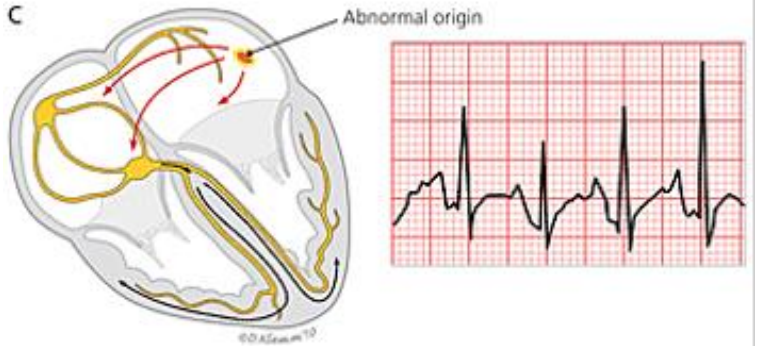
Rate: 100 to 250 bpm (atrial); ventricular varies. Rhythm: regular, narrow QRS complex usually; irregular (ectopic foci) may have wide QRS complex if aberrancy presentFocal AT: long RP interval most common; P-wave shape/polarity variable Multifocal AT: three different P-wave morphologies exist unrelated to each other; RR interval irregularly



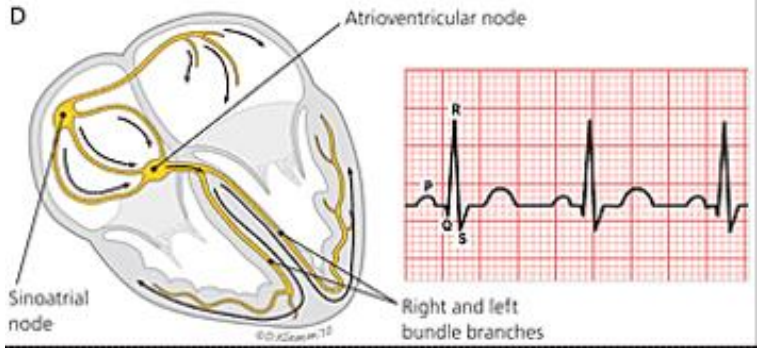
(A) In typical atrioventricular nodal reentrant tachycardia (antegrade conduction down the slow atrioventricular nodal pathway and retrograde conduction up the fast pathway), the retrograde P wave may not be seen or may be visible early after the QRS complex. When visible, it often appears as a pseudo R wave in lead V_1.



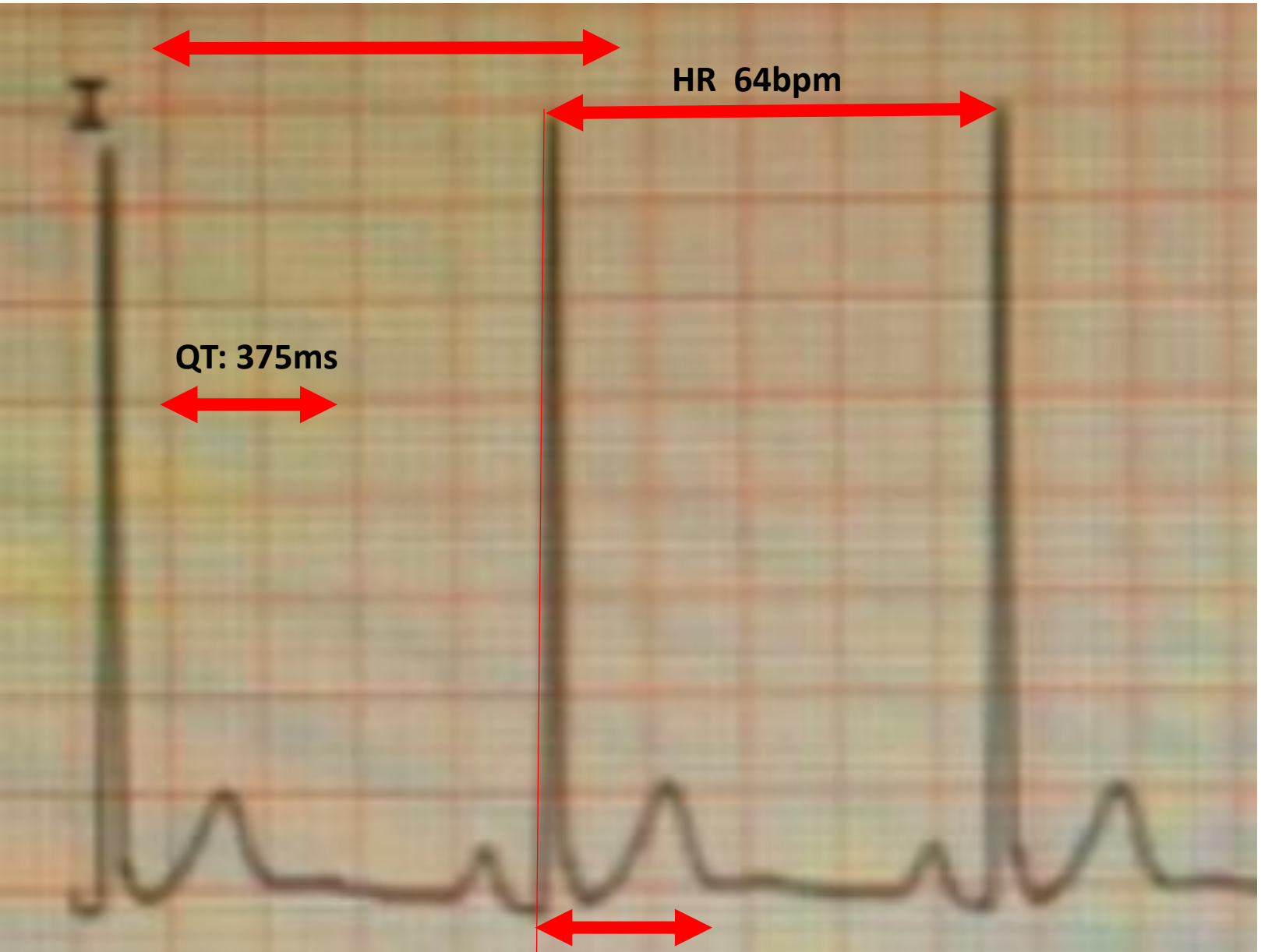
(B) In atrioventricular reciprocating tachycardia, there is typically a short RP interval, with the timing and morphology of the P wave dependent on the site and conduction velocity of the accessory pathway.



(C) Atrial tachycardia typically produces variable RP and PR intervals because atrioventricular conduction depends on atrioventricular nodal properties and the tachycardia rate. In atrial tachycardia, the morphology and axis of the P wave are influenced by atrial site of origin and tachycardia mechanism. Short- and long-term therapies are discussed in the text.



(D) Normal sinus rhythm.



HR 64bpm

QT: 375ms
QT for men in ms:
Mean value: 308
Lower limit: 324
Upper limit: 404
See in next slide mean predicted qt values (and lower and upper 95% limits) at different RR cycle lengths (1)

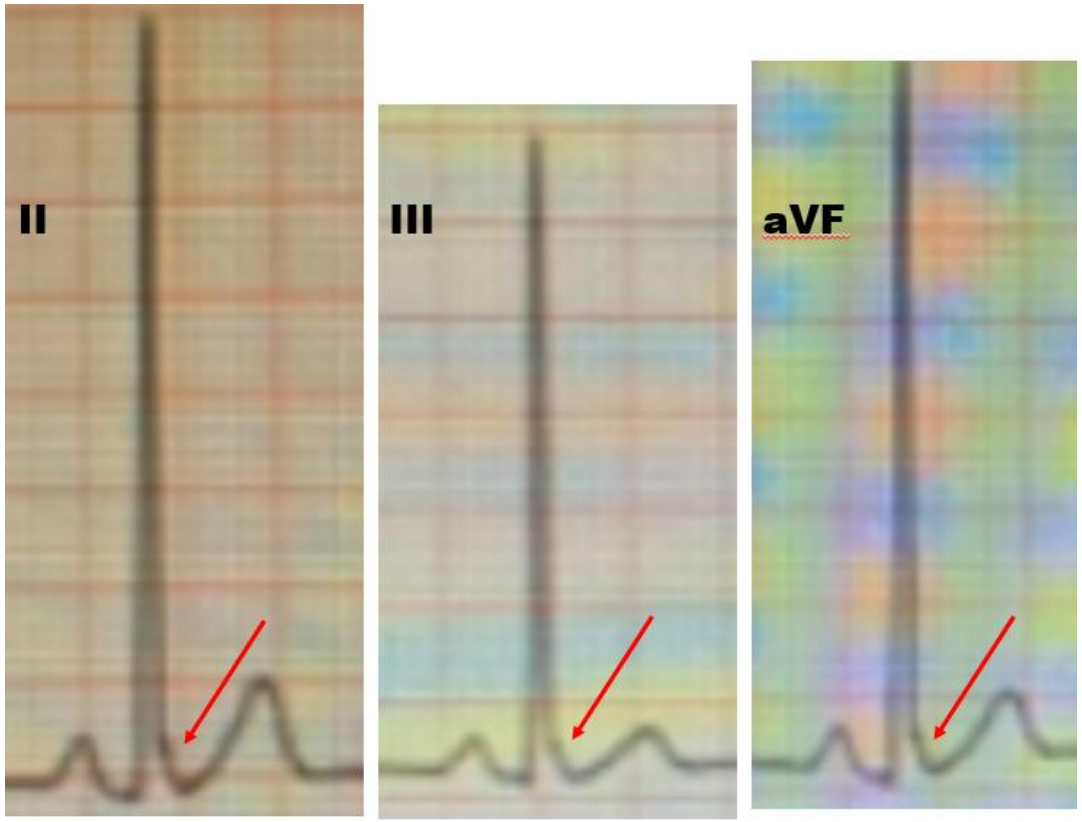
1. Sagie A, Larson MG, Goldberg RJ, Bengtson JR, Levy D.An improved method for adjusting the QT interval for heart rate (the Framingham Heart Study).Am J Cardiol. 1992 Sep 15;70(7):797-801. doi: 10.1016/0002-9149(92)90562-d.

Mean Predicted QT Values (and lower and upper 95% limits) at Different RR Cycle Lengths

RR (sec)	Heart Rate (beats/min)	QT for Men (sec)			QT for Women (sec)		
		Mean Value	Lower Limit	Upper Limit	Mean Value	Lower Limit	Upper Limit
0.50	120	0.299	0.255	0.343	0.311	0.267	0.354
0.55	109	0.307	0.263	0.351	0.318	0.274	0.362
0.60	100	0.314	0.270	0.358	0.326	0.282	0.370
0.65	92	0.322	0.278	0.366	0.334	0.290	0.378
0.70	86	0.330	0.286	0.374	0.341	0.297	0.385
0.75	80	0.337	0.293	0.381	0.349	0.305	0.393
0.80	75	0.345	0.301	0.389	0.357	0.313	0.401
0.85	71	0.353	0.309	0.397	0.364	0.321	0.408
0.90	67	0.361	0.317	0.404	0.372	0.328	0.416
0.95	63	0.368	0.324	0.412	0.380	0.336	0.424
1.00	60	0.376	0.332	0.420	0.388	0.344	0.432
1.05	57	0.384	0.340	0.428	0.395	0.351	0.439
1.10	55	0.391	0.347	0.435	0.403	0.359	0.447
1.15	52	0.399	0.355	0.443	0.411	0.367	0.455
1.20	50	0.407	0.363	0.451	0.418	0.374	0.462
1.25	48	0.414	0.370	0.458	0.426	0.382	0.470
1.30	46	0.422	0.378	0.466	0.434	0.390	0.478
1.35	44	0.430	0.386	0.474	0.441	0.397	0.486
1.40	43	0.438	0.394	0.482	0.449	0.405	0.493
1.45	41	0.445	0.401	0.489	0.457	0.413	0.501
1.50	40	0.453	0.409	0.497	0.465	0.421	0.509

Assessment of a heart rate (HR)-adjusted QT interval is considered to be clinically important, since prolongation of this interval is associated with increased incidence of MACE and SCD. To use HR-corrected QT to predict life-threatening arrhythmias and SCD, it would be appropriate to use a QT correction formula that was developed and validated in a large population-based cohort from Framingham Heart Study subjects, Sagie et al developed a linear regression equation (QTLc) that more accurately corrects QT for HR than Bazett's formula. They found that QTc under corrects QT at slow HRs and overcorrects it at fast HRs, whereas QTLC reliably corrects QT across a wide range of RR cycle lengths. The large number of subjects studied also enabled the authors to subdivide the sample into deciles of RR intervals and to examine the impact of different cycle lengths on QT, QTc and QTLc. Several formulas have been proposed to adjust the QT interval for HR. The most frequently used is Bazett's square root formula which was introduced 91 years ago.(**Bazett HC. An analysis of the time-relations of electrocardiogram. Heart 1920;7:353-370**. However, the adequacy of this nonlinear formula, obtained from data on 39 young men, has been questioned because the QTc overcorrects the measured QT interval at fast HR and undercorrects it at a low HR.(**Van De Water A, Verheyen J Xhonneux, Reneman RS. An improved method to correct the QT interval of the electrocardiogram for changes in heart rate. J Pharmocol Methods 1989;22:207-217**.)

Early repolarization pattern (ERP) Slurring variant?



ERP /J-wave pattern can be diagnosed in the presence of J-point elevation ≥1mm in 2 contiguous inferior and/or lateral leads of a standard 12-lead ECG. ERP is a common ECG finding that was considered benign for a long time. The term was first described in 1947 by Myers et al. and named “normal variations in multiple precordial leads” or normal R-ST segment elevation variant. (**Myers GB, Klein HA, et al. Normal variations in multiple precordial leads. Am Heart J. 1947;34:785-808**) ERP is an ECG finding commonly observed in young, healthy, competitive athletes and appears to be a direct result of exercise training characterized by J-point elevation ≥1 mm in ≥2 contiguous and QRS notching or slurring (J-wave) in inferior and/or lateral leads of a standard 12-lead ECG. This particular aspect was considered a normal ECG variant pattern for a long time.

. Its potential to cause cardiac arrhythmias has been hypothesized from experimental models because of similarities with the ECG manifestations of the highly arrhythmogenic BrS and the potential for misdiagnosis. It has been suggested that some forms of ERP seen in the clinic may not be benign. Sporadic case reports and basic electrophysiological research have suggested a critical role of the J wave or lambda wave in the pathogenesis of idiopathic ventricular fibrillation (IVF). In 2008, when it was linked to sudden cardiac arrest (SCA) due to IVF, Haissaguerre et al reviewed data from 206 individuals from 22 centers who were resuscitated after SCA due to IVF and assessed the prevalence of ECG early repolarization. The latter was defined as an elevation of the QRS-ST junction (end QRS slurring or end QRS notching) of ≥0.1 mV from baseline in the inferior or lateral leads, in at least two contiguous leads manifested as end-QRS slurring or notching (Figure next slide).

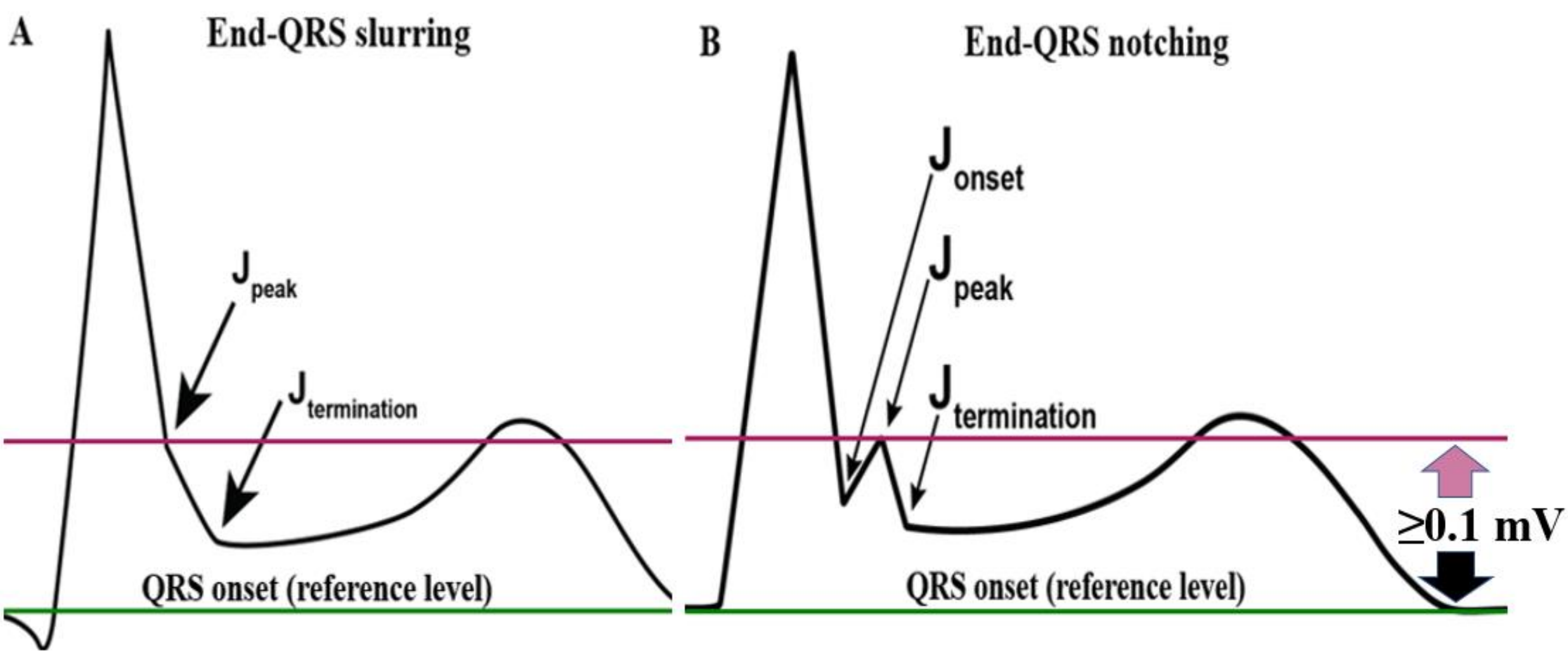
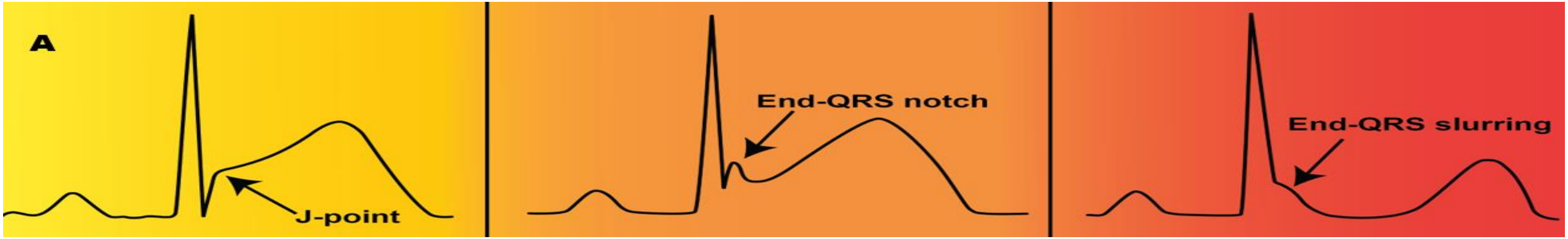


Figure. ERP variants: (A) QRS slurring or lambda wave and (B) end-QRS notching. J point is present in all ECGs and marks the transition of QRS complex to ST segment. In both variant, the J-point correspond to J-termination point. The J point is a point in time marking approximately the end of the QRS 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.

Much confusion over the definition of ERP follows until an expert consensus documented occurred in 2015. The objective of this expert meeting was to prepare an agreed definition to facilitate future research in this area. The different definitions of ERP were reviewed to delineate the ECG characteristics. 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 below). **Macfarlane PW, Antzelevitch C, Haissaguerre M, *et al.* The Early Repolarization Pattern: A Consensus Paper. J Am Coll Cardiol. 2015;66:470-477**.



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. (**Antzelevitch C, Yan GX, Viskin S. Rationale for the use of the terms J-wave syndromes and early repolarization. J Am Coll Cardiol. 2011;57:1587-1590.**)(**Priori SG, Wilde AA, Horie M, *et al.* Executive summary: HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. Europace. 2013;15:1389-1406**.) (**Antzelevitch C, Yan GX. J-wave syndromes: Brugada and early repolarization syndromes. Heart Rhythm. 2015;12:1852-1866**.)

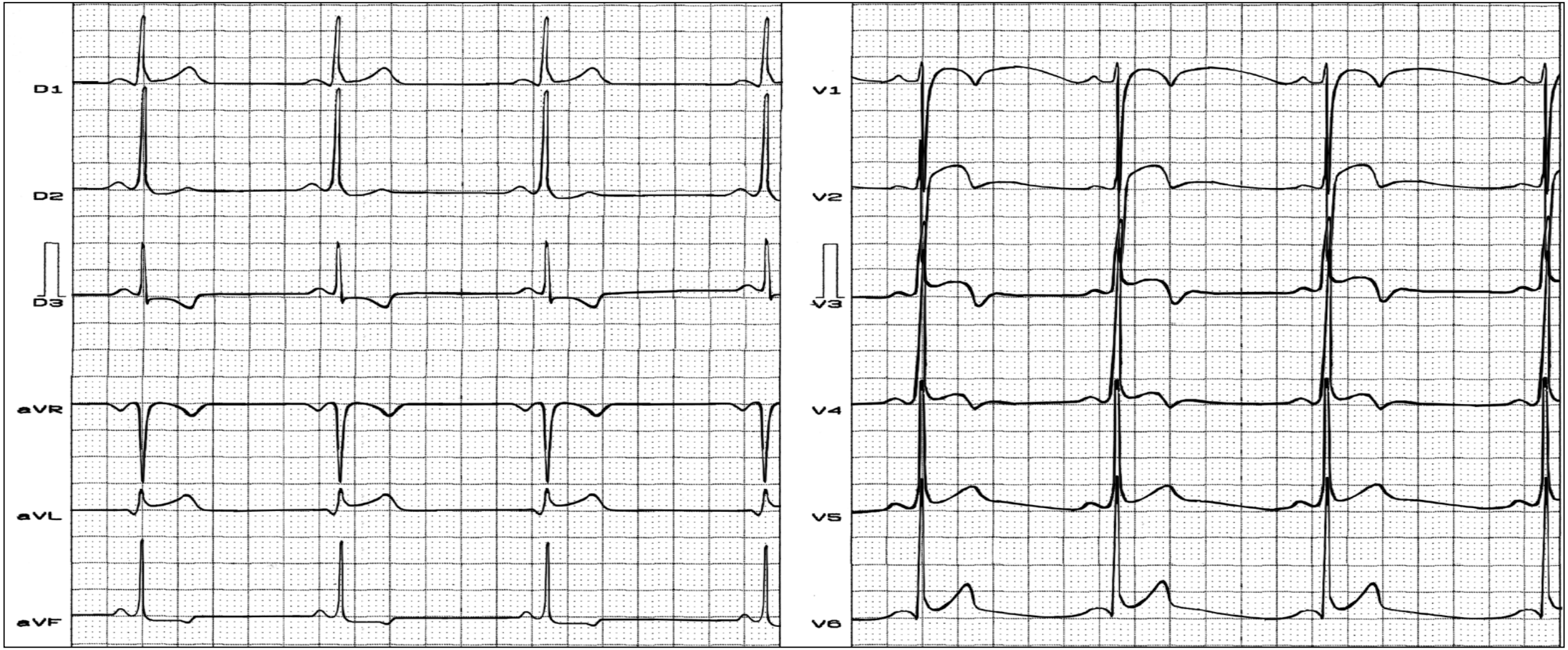
The J wave of the ECG

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 next slide

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. SV_1 or V_2+RV_5 or V_6 >35 mm (Index of Sokolow Lyon). ST segment elevation from V_2 to V_6 and with negative T from V_1 to V_4. 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).

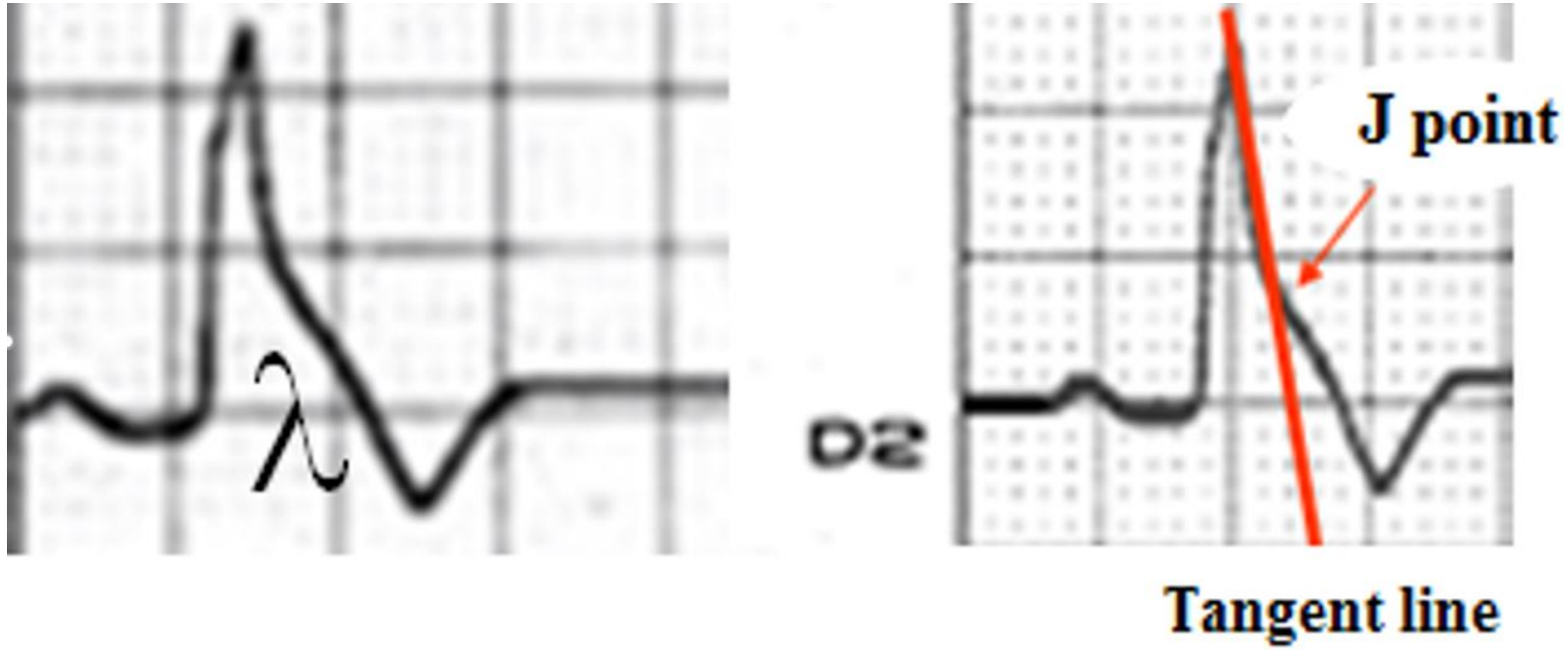


Figure . Very high J-point with descending ST segment followed by a negative T-wave. It is considered a malignant or “evil” form.

Figure shows an example of currently named subtype 2

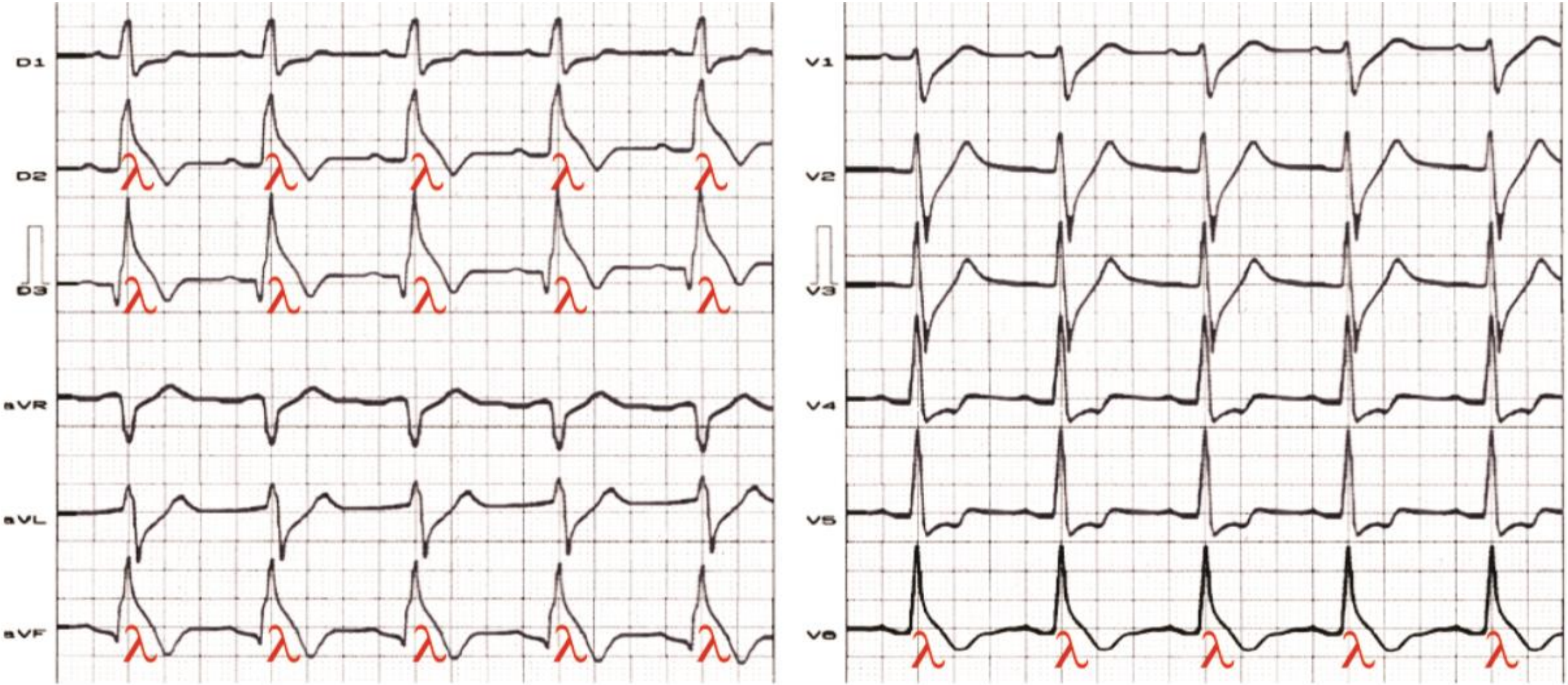


Figure. 12-lead ECG showing persistent ST segment elevation in the inferior and lateral leads, associated with concomitant reciprocal or mirror image in the anterior wall, which was not modified with the use of sublingual nitrate in absence of hypothermia, electrolyte imbalance ischemia or brain injury(**Riera AR, Ferreira C, Schapachnik E*, et al.* Brugada syndrome with atypical ECG: downsloping ST-segment elevation in inferior leads. J Electrocardiol. 2004;37:101-104**.)

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

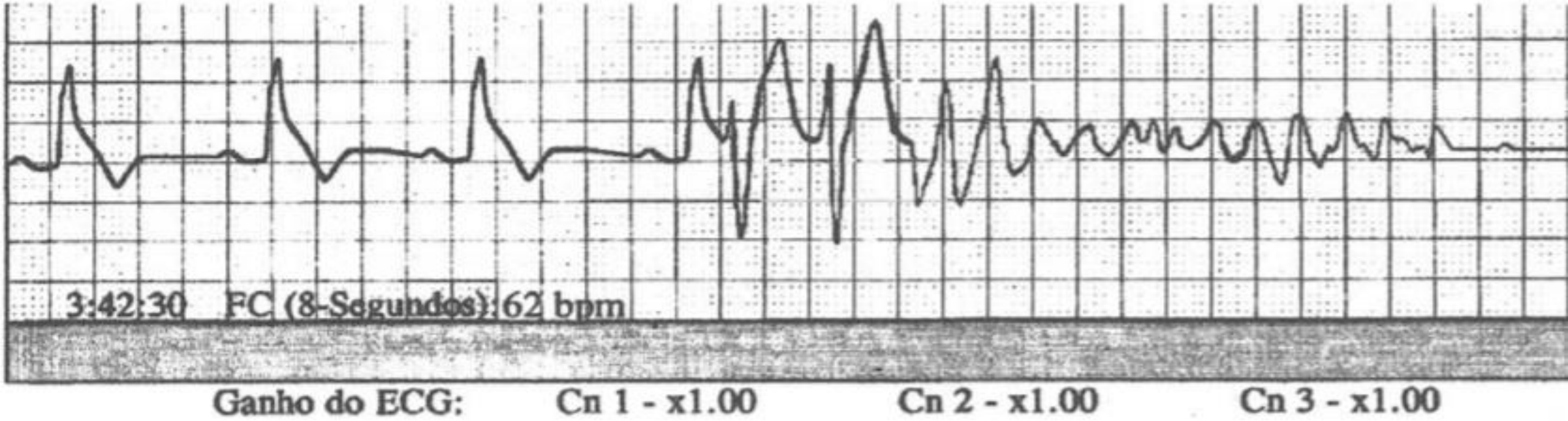


Figure. Sudden cardiac death by idiopathic polymorphic ventricular tachycardia/IVF with short coupling ending in cardiac arrest

ERP types

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 15).

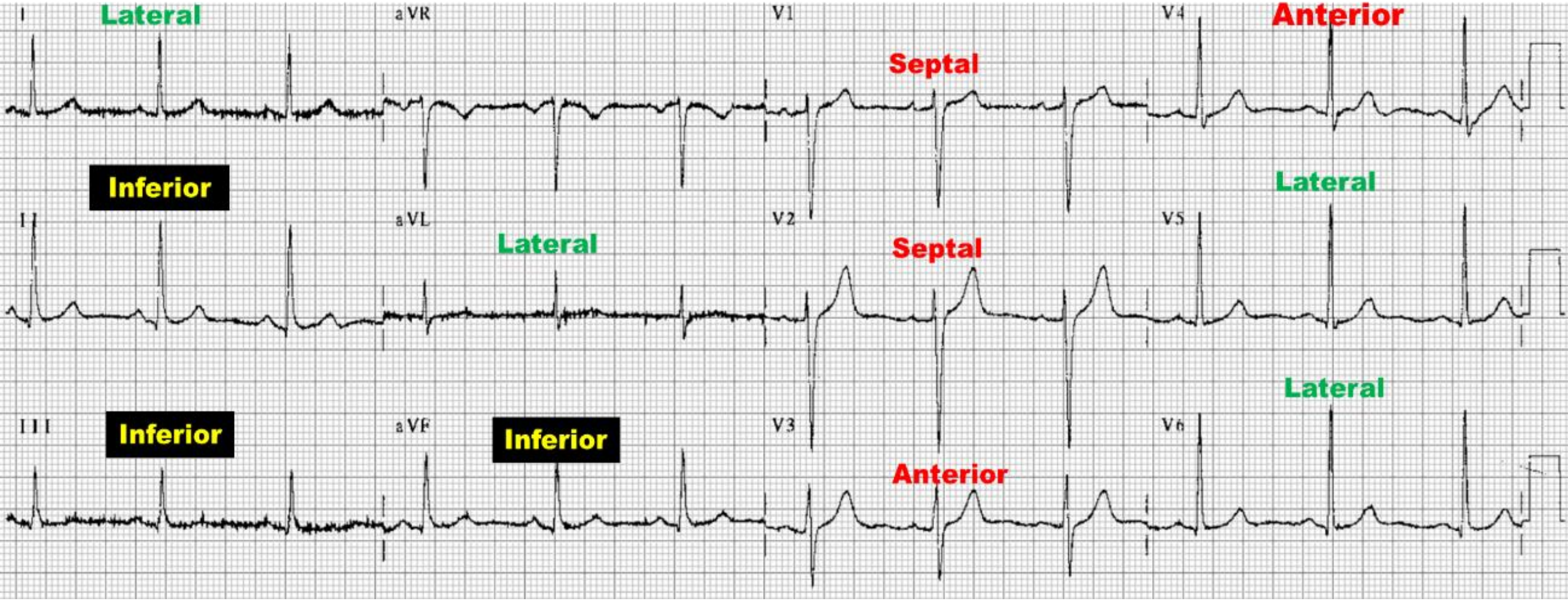


Figure. Basal lead ECG tracing. J-wave across all precordial and inferior leads.

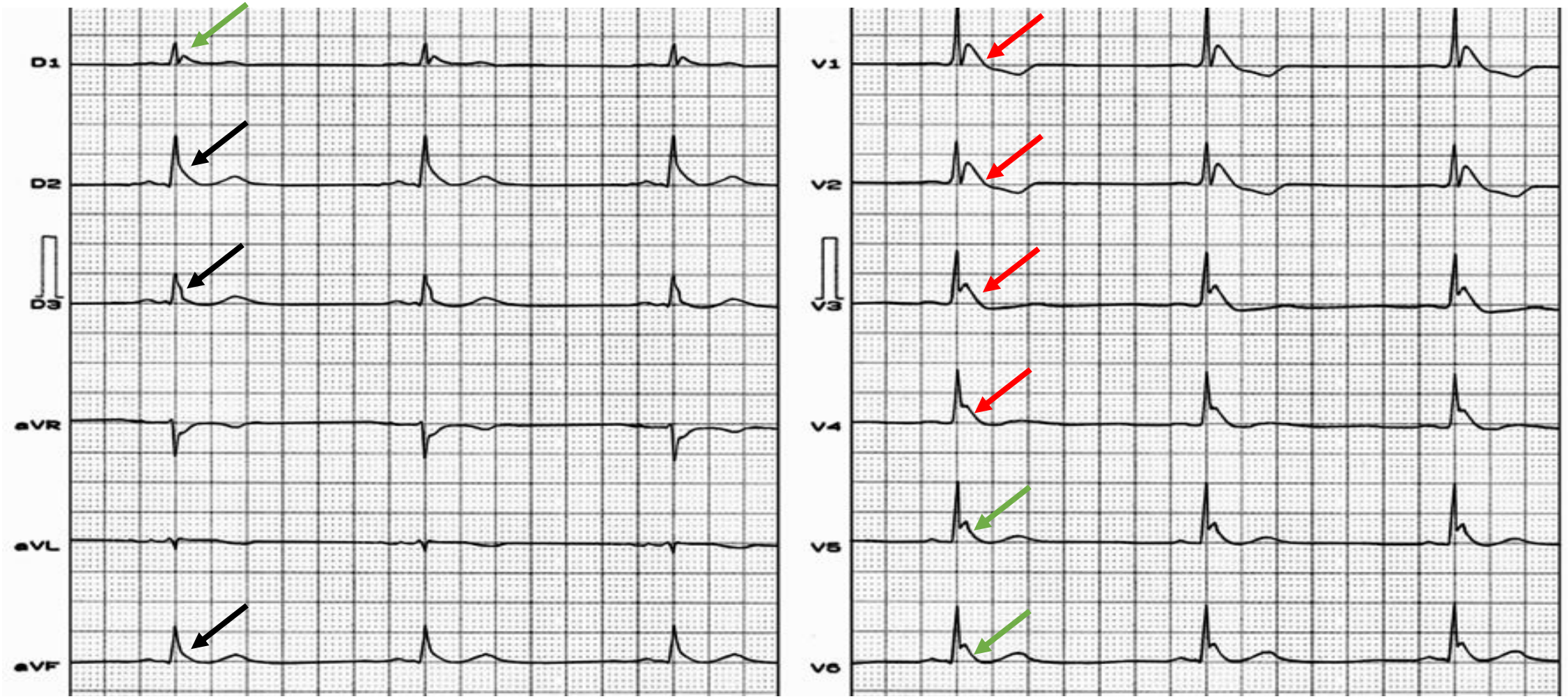
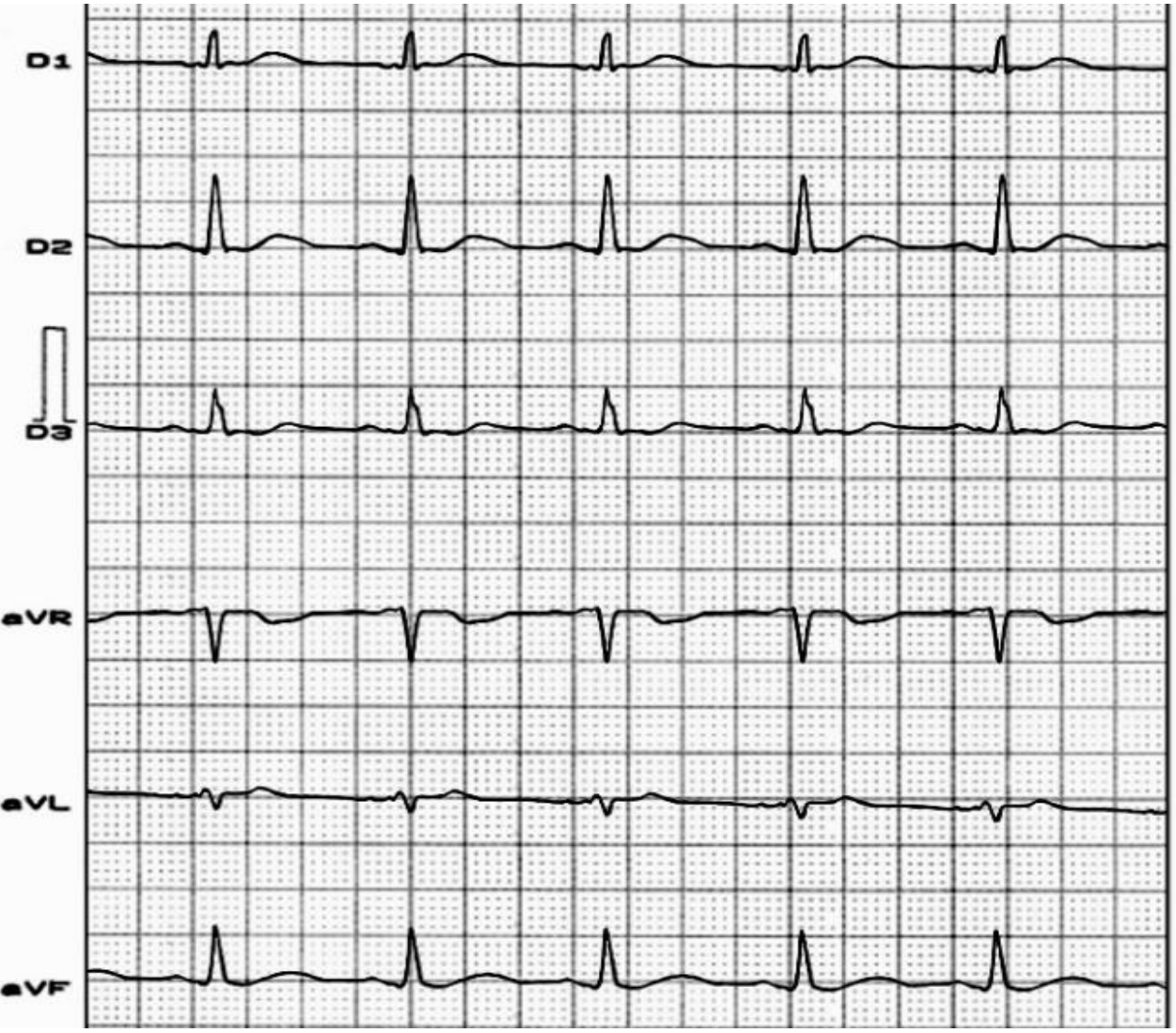


Figure . ECG of an ERP in the inferior, lateral and right precordial leads (type 3). This variant is associated with the highest level of risk for the development of VF storms. In type 3, the Brugada waves may be seen together with giant J waves in other ECG leads. Although the Brugada waves are not 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.



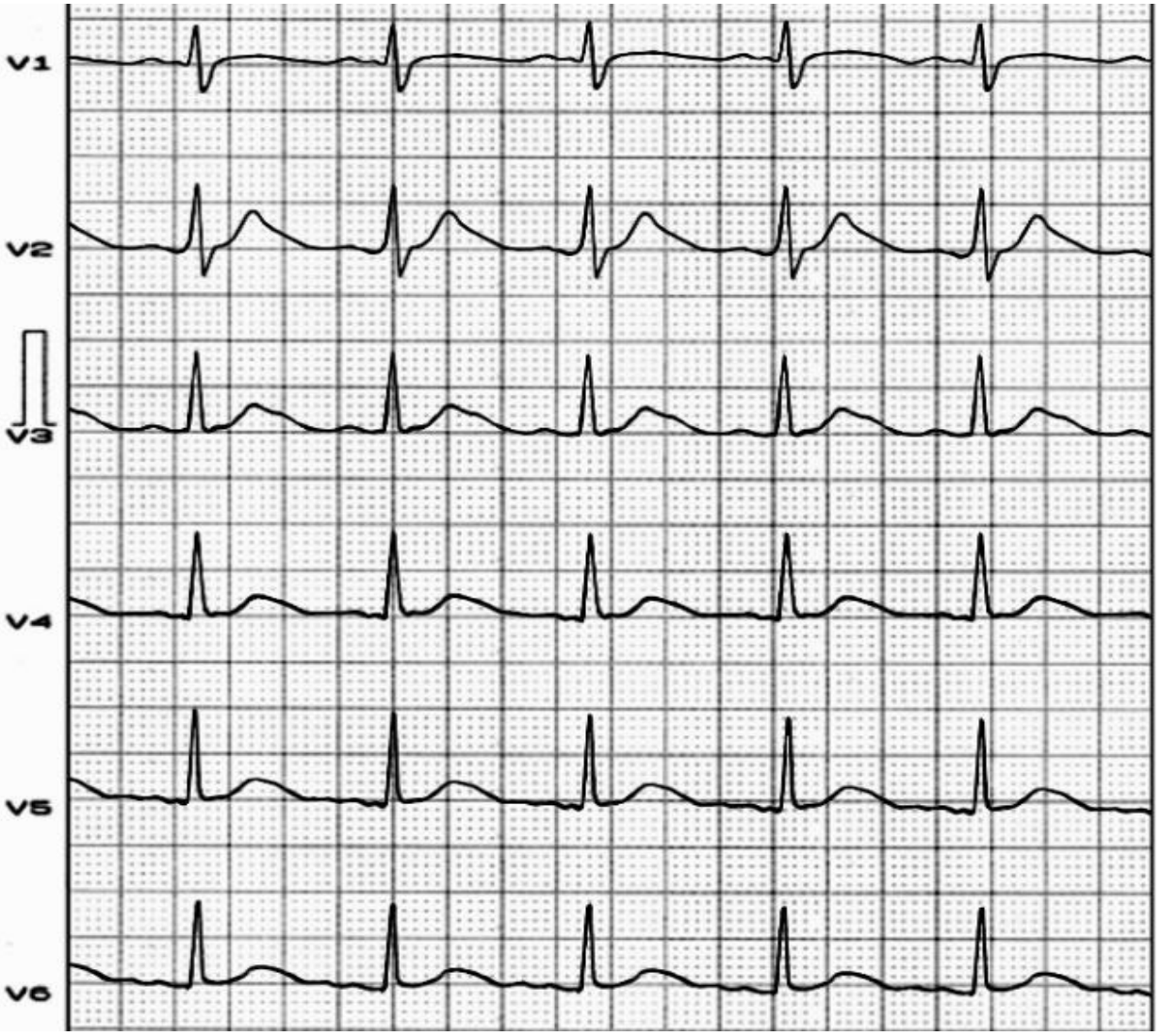
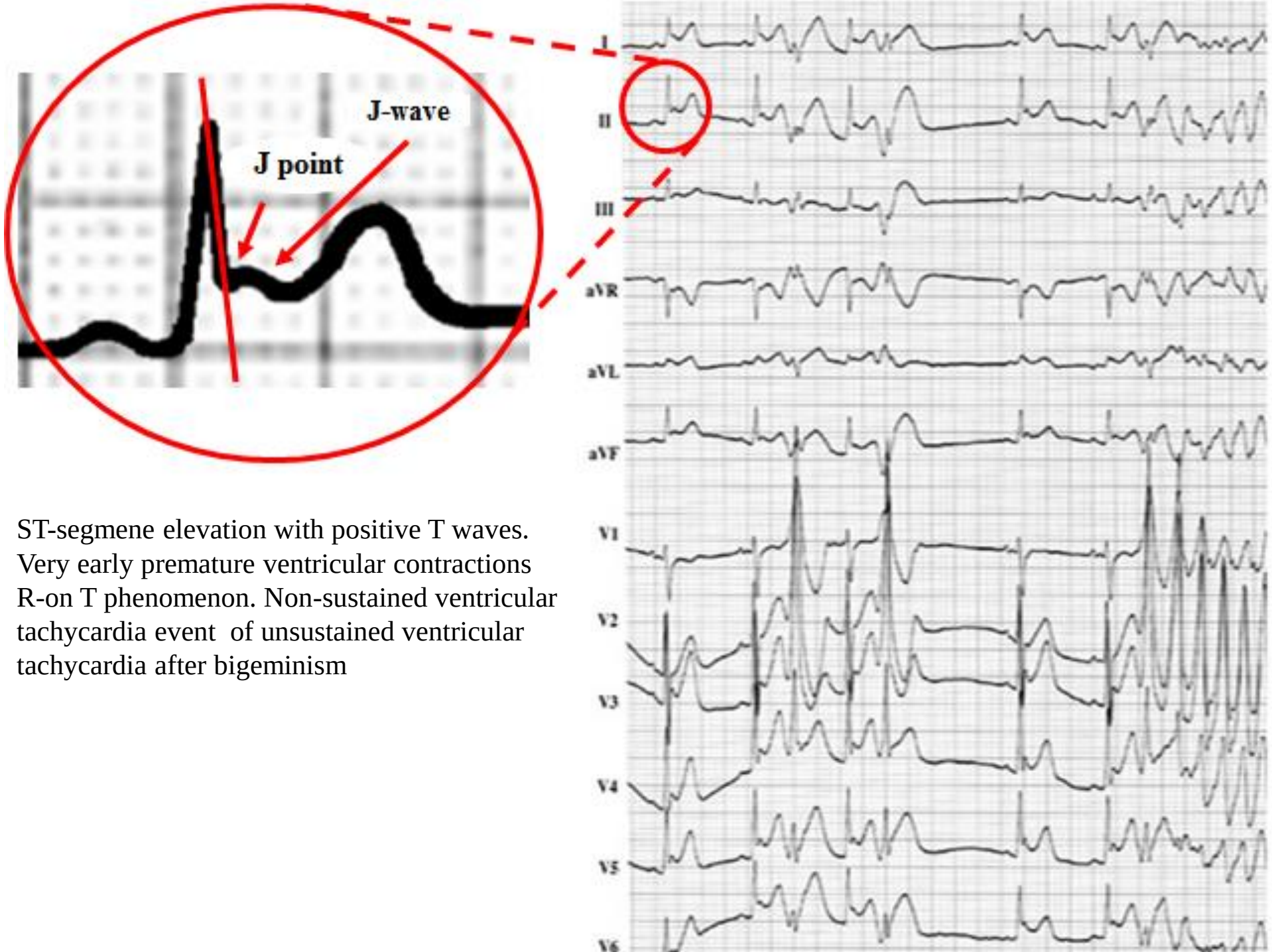


Figure shows an ECG performed two days after oral quinidine administration. Quinidine reduces the magnitude of the Ito channel – mediator of phase 1 and consequently normalize the ST segment elevation. Additionally, this drug could improve repolarization due to its vagolytic effect (M2 muscarinic receptor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



ST-segmene elevation with positive T waves. Very early premature ventricular contractions R-on T phenomenon. Non-sustained ventricular tachycardia event of unsustained ventricular tachycardia after bigeminism