

Idiopathic Fascicular Left Ventricular Tachycardia with atypical ECG morphology: Which is the mechanism?



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Idiopathic Fascicular Left Ventricular Tachycardia with atypical ECG morphology: Which is the mechanism?

Other denominations: Fascicular Tachycardia (FT), AKA Fascicular or interfascicular VTs, verapamil-sensitive left VT, verapamil-responsive VT, Belhassen-type VT, idiopathic left ventricular tachycardia (IVLT), or borderline-broad complex VT.

Overview

Idiopathic ventricular tachycardia (IVT) is a term that has been used for VT in the absence of clinically apparent structural heart disease (1). It accounts for approximately 10% of all VTs evaluated in specialized arrhythmia services. Several types have been reported according to their clinical presentation, ventricular origin, response to drugs, ECG pattern, among other parameters. Idiopathic fascicular tachycardia (FT) is a rare cause of sustained monomorphic ventricular tachycardia (VT). It is a well described clinical entity (**Okumura 2002**) that typically occurs in the absence of structural heart disease and whose mechanism is usually due to reentry within the left-sided specialized conduction system. (**Zipes 1972; Belhassen 1981; Lerman 1997; Okumura 2002; Nogami 2002**). Detailed anatomic and histopathologic studies of the specialized conduction system have delineated the presence of 3 fascicles in the left ventricle (LV) of the human heart, including the posterior, anterior, and septal fascicles. (**Demoulin 1972; Demoulin 1973; Kulbertus 1975;1976**).

Electrocardiographic features

The baseline electrocardiogram (ECG) is normal in most patients though it may present T-wave inversion immediately after tachycardia (cardiac memory).

Unlike patients with structural heart disease, IFLVT usually shows a QRS complex duration inferior to 140-150 ms and fast initial forces (RS interval of 60-80 ms). Both features can lead to misdiagnosis of aberrant supraventricular tachycardia. The electrocardiographical pattern varies depending on the site of origin of the tachycardia:

Fascicular tachycardia can be classified based on ECG morphology corresponding to the anatomical location of the re-entry circuit in the following variants:

- 1. Left Posterior fascicular VT/Posterior fascicular ventricular tachycardia (P-IFLVT).** (90-95% of cases): RBBB-configuration+ superior axis in the frontal plane (left anterior fascicular block-like pattern) suggesting that the exit of the circuit is located in the inferoposterior septum arises close to the left posterior fascicle. (**Zipes 1972; Belhassen 1981; Nogami 1998; Okumura 2002**);
- 2. Anterior fascicular ventricular tachycardia (A-IFLVT)/Left anterior fascicular VT** (5-10% of cases): The ECG shows RBBB configuration + right axis (left posterior fascicular block-like pattern); arises close to the left anterior fascicle. It is the second variant in terms of frequency. The earliest activation has been described in the anterolateral wall of the left ventricle.

3. **Upper septal fascicular ventricular tachycardia** (rare): that shows a narrow QRS and normal or rightward axis 6; 12 atypical morphology – usually RBBB but may resemble LBBB instead; cases with narrow QRS and normal axis have also been reported. Arises from the region of the upper septum. (**Shimoike 2000**). This presentation is exceptional. As a general rule, it presents RBBB but a few cases with morphology of left bundle branch block have also been described. A narrow QRS and normal frontal axis have been reported too.
4. **Multiform fascicular tachycardia (FT) in the same patient because several circuits are operative** (**Sung 2013**)
- Another component characteristic of FT is borderline QRS complex duration (modest increase in QRS width ($\approx 120\text{-}130$ ms)).

Historical Background

May 1972: Cohen et al (**Cohen 1972**). First description: VT with a relative narrow QRS complexes ($\leq 140\text{ms}$).

1979: Zipes et al (**Zipes 1979**) reported three patients with VT characterized by QRS width of 120 to 140 ms, RBBB-Like pattern and extreme left-axis deviation. These authors described the characteristic triad:

- Induction with atrial pacing
- RBBB-like pattern with extreme left axis deviation
- Without structural heart disease

1981: Belhassen et al (**Belhassen**) were the first to report on the characteristic termination of this VT with intravenous verapamil, hence accounting for the terms Belhassen VT and verapamil-responsive VT to describe the condition.

Differential diagnosis

As in other IFLVT diagnosis requires exclusion of structural heart disease. It is therefore recommended to perform echo, coronary angiography or computed tomography, if deemed necessary depending on the cardiovascular risk profile of the patient. Quite often, the IFLVT can be confused with a **paroxysmal supraventricular tachycardia conducted with aberrancy** because of its relatively narrow QRS, response to verapamil and presentation in young patients without structural heart disease. The presence of ventriculoatrial dissociation in the ECG or during the EPS may clarify the diagnosis.

It must also be distinguished from other forms of VT with narrow QRS. This is the case of **VT related to intramyocardial reentry** close to the conduction system in which the QRS may be narrow due to an early invasion of the His-Purkinje system. As well, verapamil sensitive **VT from the mitral annulus** should be considered. They can present morphology of RBBB, right axis and a relatively narrow QRS, but its clinical and EPS behaviour is usually different. An exceptional entity to consider is the **interfascicular VT**. In this case, the tachycardia circuit is established between the two fascicles, either one or another sense. The QRS morphology is similar in sinus rhythm and tachycardia, RBBB and LAFB, LPFB or LSFB. The ablation of one of the fascicles resolved successfully the event.

Epidemiological Aspects

Age: Usually observed between 15 and 40 year. Gender: Male predominance (70% of cases). Sudden Death Familiar Background: Negative.

Sex: verapamil sensitive fascicular VT has 3:1 male predominance (**Nakagawa 2002**).

Case Report

JSS, 28 years old, Caucasian man with history of recurrent paroxysmal palpitations since age 14 years. Several hospitalizations in the emergency room with recording of regular tachycardia with a borderline-broad complex

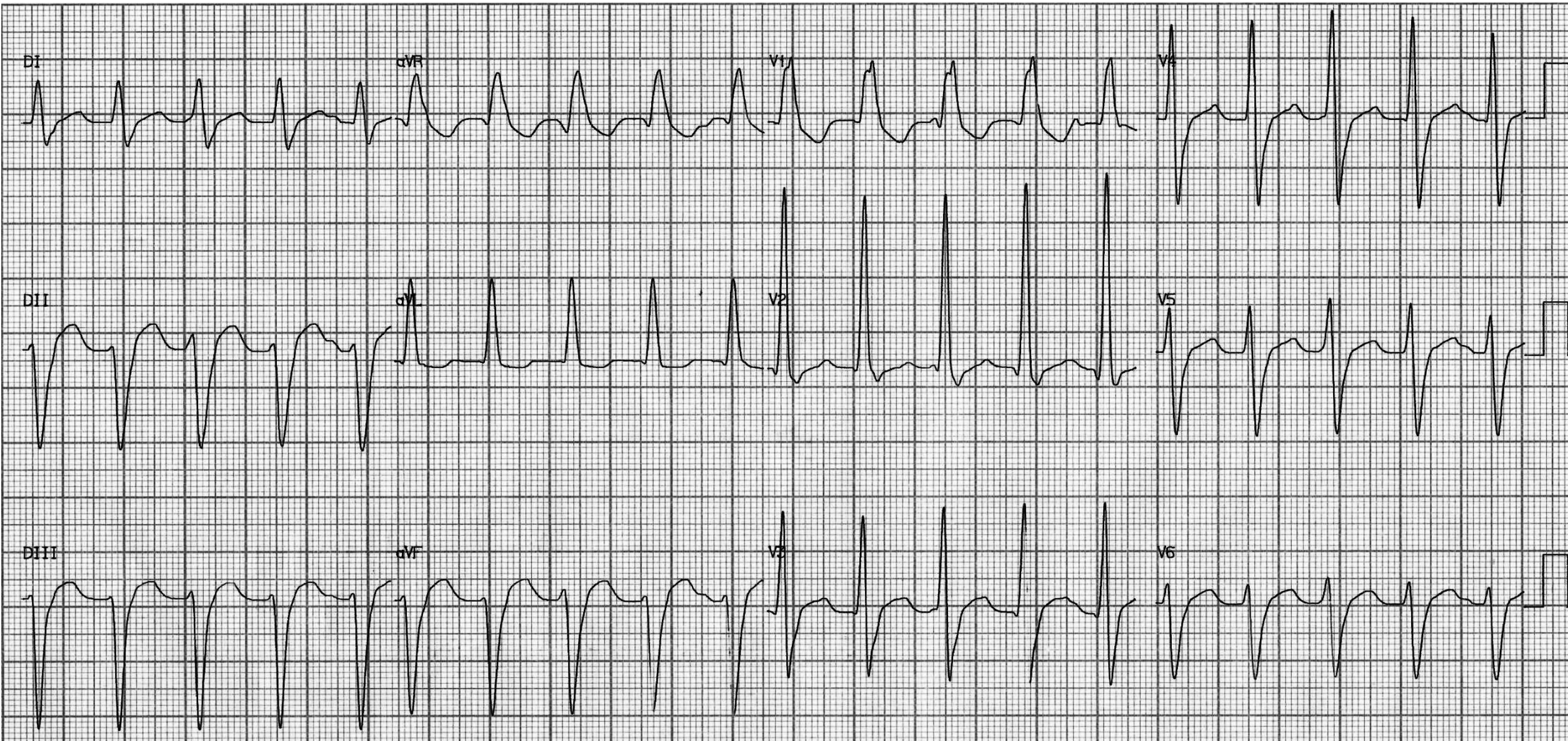
Current admission by dyspnea and precordial pain with recording of ventricular tachycardia with cycle of 500 ms (120 bpm) and ECG with atypical RBBB pattern + Prominent Anterior QRS forces and superior axis deviation: LAFB-like type (suggestive of anterior divisional tachycardia). Little response to treatment with IV amiodarone. Several attempts of cardioversion were successful immediately and early recurrence of tachycardia. We chose to perform electrophysiology study.

Which is the diagnosis of the event?

Which is the mechanism?

Which is probable circuit used as descending pathway and ascending direction?

ECG performed during the event



Colleagues opinions

The QRS width and intrinsicoid deflection do not appear consistent with fascicular mechanism- more like myocardial or papillary muscle.
What did the EPS show?

Best,
Rod
Roderick Tung, MD, FACC, FHRS
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Director, Cardiac Electrophysiology & EP Laboratories
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Center for Arrhythmia Care | Heart and Vascular Center
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1. The diagnostic of VT is 99% correct due to obvious AV dissociation.
2. The origin of the VT is in the posterior LV
3. The mechanism of VT is unclear before we know about the results of EPS. I note 3 important things:
 - a. The effects of verapamil and adenosine have not been tested.
 - b. The QRS morphology during VT is somewhat different from what classically observed in "typical Belhassen VT"
 - c. The authors have certainly some good reason for not sharing with us yet the ECG tracings in sinus rhythm; I am wondering what could be this reason????

Looking forward to learning more about this patient but always remind:

Not all RBBB-LAD VT are "Belhassen VT" !!! (even in patients without obvious heart disease)

Bernard Belhassen

From the Department of Cardiology, Tel-Aviv Sourasky Medical Center,
Sackler Faculty of Medicine, Tel-Aviv University, Israel.

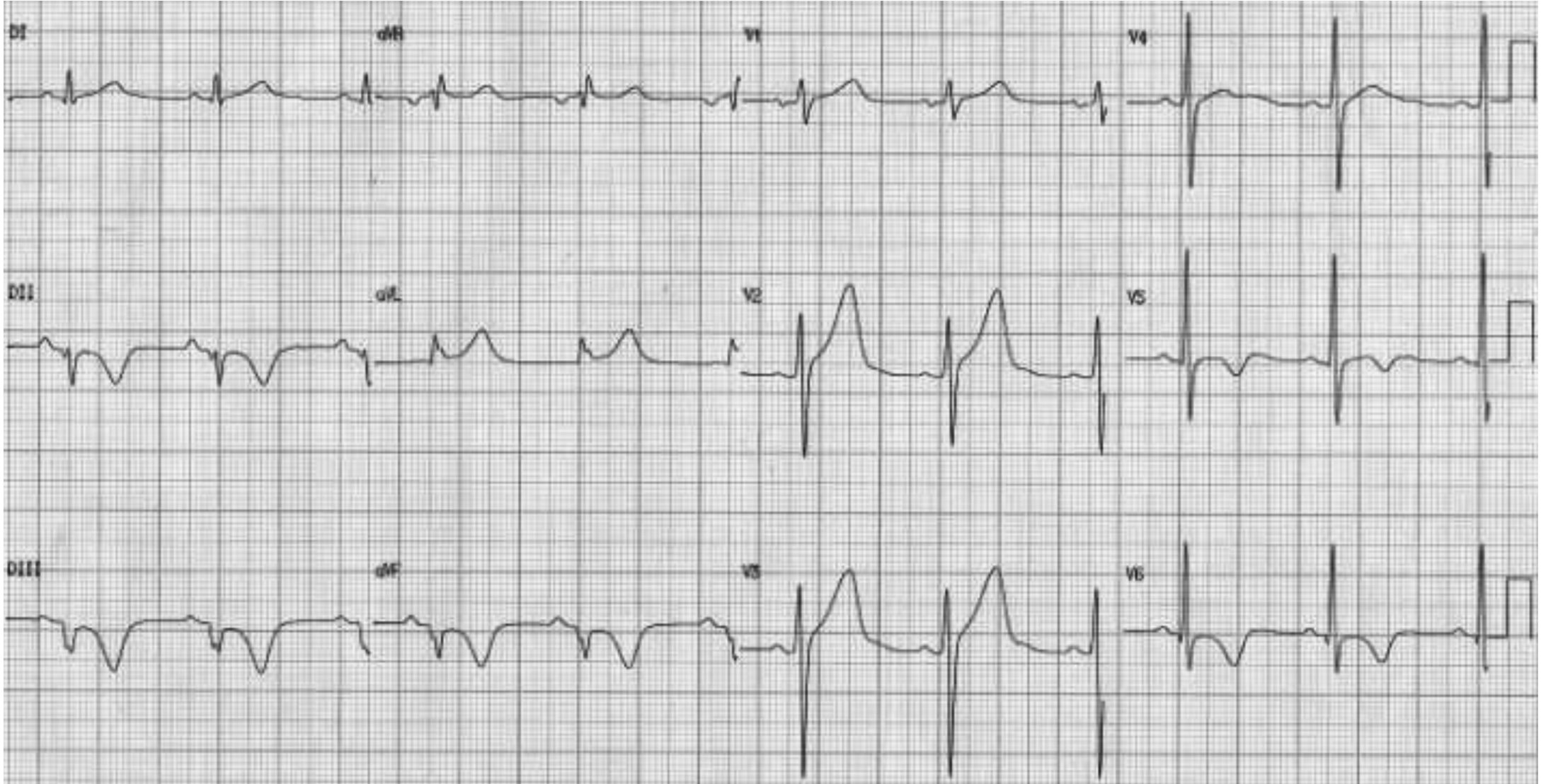
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Tel Aviv Israel.

NB. You might have been surprised by my statement in my previous mail " The diagnostic of VT is 99% correct due to obvious AV dissociation". You certainly asked: why not 100%??? The reason is that I have observed a young patient with BELHASSEN VT and normal resting ECG in whom rapid atrial pacing resulted in intraventricular aberration associating RBBB + LAH with a QRS configuration IDENTICAL to the VT. Therefore it is possible (and this should be absolutely exceptional) that a wide QRS tachycardia with RBBB + LAH in a patient with normal heart and normal baseline ECG could be due to SVT + RBBB + LAH. If during such a tachycardia AV dissociation is present, the diagnosis should be either AVNRT or ectopic junctional/His tachycardia that are associated with RBBB + LAH + VA block (in other words another speculation of cardiac electrophysiologists !!!)



ECG performed immediately after cardioversion



Dear BB: The young emergency physician did not make the correct diagnosis of VT type. He did not think in **Belhanssen's sensitive verapamil VT**. This is the reason why he did not use intravenous verapamil or adenosine

ECG: Characteristic non-specific transient inferolateral T-wave changes immediately after cessation of event (our case).

Andrés& Raiundo

Aduendum

Dear Professor Bernard Belhassen: T-wave inversion In this case is caused by “cardiac memory” Cardiac memory”(CM) is a peculiar variety of cardiac remodeling (**Chiale 2014**) observed after abnormal sequence of ventricular activation/depolarization (**Rosenbaum 1982-1990; Geiger 1943; Katz 1992; Chiale 2014**) manifested by a persistent (for minutes, hours, weeks or months) but reversible T-wave changes on the surface ECG.

- I. Electrocardiographic changes (cardiac memory) subsequente of post pacing or artificial ventricular depolarization(**Chiale 2010; Elizari 1995**)
- II. Cardiac memory after intermittent left bundle branch block (**Rosenbaum 1973-1982; 1990**) “pseudo primary T-wave changes”
- III. Cardiac memory after episodes of tachycardia post-paroxysmal wide tachycardia syndrome (**Kernohan 1969**). This is the present case.
- IV. Cardiac memory after unespecific intraventricular conduction defects (NSIVCD)
- V. Cardiac memory after intermittent preexcitation Wolff-Parkinson–White type (**Kalbfleisch 1991**): example after ablation of anomalous pathway in Wolff-Parkinson-White. T wave inversion in II, III and aVF associated to delta wave disappearance of delta wave after ablation of anomalous accessory pathway in patients carrier of Wolff-Parkinson-White syndrome, is a powerful marker of success of ablation procedure (**Trajkov 2008**).
- VI. In patients who develop significant QRS vector changes during AVB, the effects of cardiac memory lead to excessive QT prolongation. Cardiac memory resulting froernohanm a change in QRS morphology during AVB is independently associated with QT prolongation and may be arrhythmogenic during AVB.(**Rosso 2014**)

The great interest for investigating this topic is due to the impact that recognizing this phenomenon has when making decisions in cardiological clinical practice, since it manifests with T wave alterations generally interpreted mistakenly as of ischemic origin (pseudo-primary T waves) observed in multiple scenarios, mainly in the presence of precordial pain in the ER, as in the first case.

Andrés & Raimundo

BB answer: "MEMORY“ ABSOLUTELY: PLEASE NOTE IN REGARD TO WHAT YOU WROTE " III. Cardiac memory after episodes of tachycardia post-paroxysmal wide tachycardia syndrome (**Kernohan 1969**)“ that KERNOHAN did the wrong diagnosis of SVT + aberration. BUT ABOVE ALL that Mauricio Rosenbaum in his paper on memory published in the AMERICAN JOURNAL OF CARDIOLOGY (**1980-1981-1982??**) showed the tracings of KERNOHAN ... but did not make the diagnosis of VT (this was a typical case of Belhassen VT).

Bernard **Belhassen (BB)**

1. The diagnostic of VT is 99% correct due to obvious AV dissociation.

I wrote this sentence taking in account the ECG showed a wide QRS tachycardia with AV dissociation.

When you have a regular, wide QRS tachycardia and AV dissociation, the first diagnosis should be VT.

I only pointed out that there are exceptional cases of wide QRS tachycardia with AV dissociation in which the final diagnosis is not VT.

I do agree with Dr Baranchuk that the use of “99%” may be inappropriate since I have never studied the issue in a systematic way.

I do accept to delete the “99%” and replace it with “almost always”.

My sentence should read:

“In the presence of regular, wide QRS tachycardia with AV dissociation, the diagnosis of the arrhythmia is “almost always” VT.

NB. I did not read Dr Baranchuk’s e-mail.

Bernard **Belhassen (BB)**

From the Department of Cardiology, Tel-Aviv Sourasky Medical Center,
Sackler Faculty of Medicine, Tel-Aviv University, Israel.

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Tel Aviv Israel.

Hi

My dear colleague, Dr Belhassen makes a profound mistake on his statement that “99% of the correct diagnoses of VT are done due to obvious AV dissociation”. I hope he reads my email and can honestly retract from that comment.

The basis of his comment is that he saw 1 (ONE) patient with this characteristic (AV dissociation, wide complex, not VT) (perfectly explained by him, so no need for me to repeat it). That means that his UNIVERSE is composed by another 99 AV dissociations that were VT. (one case 1%, thus 100 is the total number of cases that he is counting...).

Did Dr Belhassen ONLY see or manage 100 cases of AV dissociations resulting in VT diagnosis? Obviously not. This may be the number he sees in 1 month,...or two (maybe three??). Let’ say 1 or 2 years.

If he had said “The statement that 100% of the correct VT diagnosis are done by finding AV dissociation is false”, I would agree. For the same reason of the case he described.

Managing numbers lightly can induce mistakes in our reasoning.

Best wishes,

Adrian Baranchuk MD FACC FRCPC FCCS

Professor of Medicine (Tenure)

Queen's University

Kingston, Ontario, Canada

Editor-in-Chief, Journal of Electro cardiology



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Bernard **Belhassen (BB)**

Comments (continued)

I had the privilege to get the ECG in sinus rhythm after VT conversion by DC shock. There are marked inferolateral changes as well known after termination (with drug or DC shock) of this type of VT.

The QRS is somewhat bizarre with some Q waves in inferior leads associated with relatively prominent R in V1 with $R/S > 1$.

The possibility of minor preexcitation involving a left sided accessory pathway.

Based on ECG only, we should rule out of course an old inferoposterior myocardial infarction (that can be excluded based on clinical ground of a long history of palpitations in a young otherwise healthy boy).

Thus assuming that we are dealing with a patient with normal heart and LV-VT, 2 diagnoses should be discussed:

1. "Belhassen VT" +++++++
2. Other type of LV-VT +

Answer

Another addendum: Dear BB, the dorsal or posterior wall does not exist; as our "guru" taught us: the great Catalan Professor of electrocardiology Antonio Bayes de Luna.

He wrote in Circulation eleven years ago these **recommendations**

1. Because these ECG patterns matched well with the CE-CMR necrotic areas, although some of them present limited sensitivity, they offer a better global concordance than the classic Q-wave ECG pattern location.
2. The concordance between the ECG patterns and the location of MI by CMR shows that abnormally increased R waves, the Q-wave equivalent, in leads V1 and V2 indicate a **lateral MI** and that abnormal Q waves in leads aVL and I without a Q wave in lead V6 indicate a mid-anterior MI. Therefore, **THE TERMS POSTERIOR AND HIGH LATERAL MI ARE INCORRECT** when applied to these patterns and should be changed to lateral wall MI and mid-anterior wall MI, respectively (**Bayés de Luna 2006**).

Andrés.

My pet "Evita"



Dear Andres,

Thank you for sending this interesting case.

I think that more than 80% of fascicular tachycardia's are involved in the left posterior fascicle. In most of these ECGs, like yours, there is RBBB and left axis deviation. The left posterior fascicular is usually involved and is used as a retrograde part of the reentry circuit. They are generally sensitive to verapamil; however, ablation is very effective once one can localize the reentry circuit. Even though verapamil may work for some time, most do not respond to antiarrhythmic agents; therefore, ablation has become a first-line therapy in these cases with often no structural heart disease. Please let me know if my interpretation is correct. Thank you again.

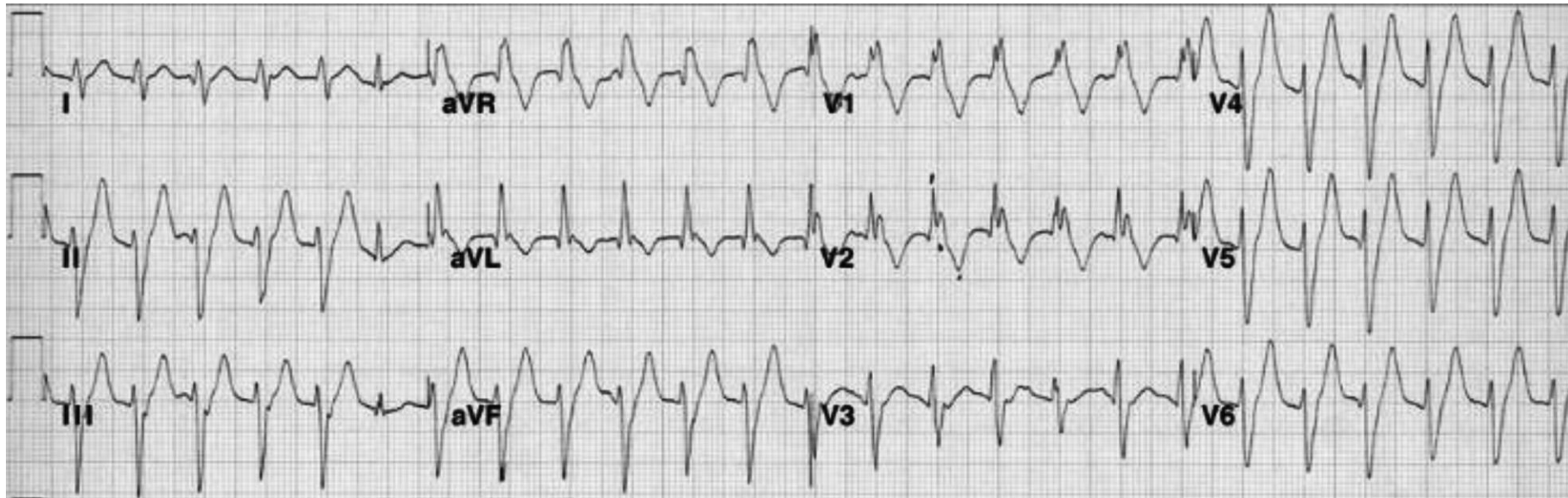
Very best,
Mohammad

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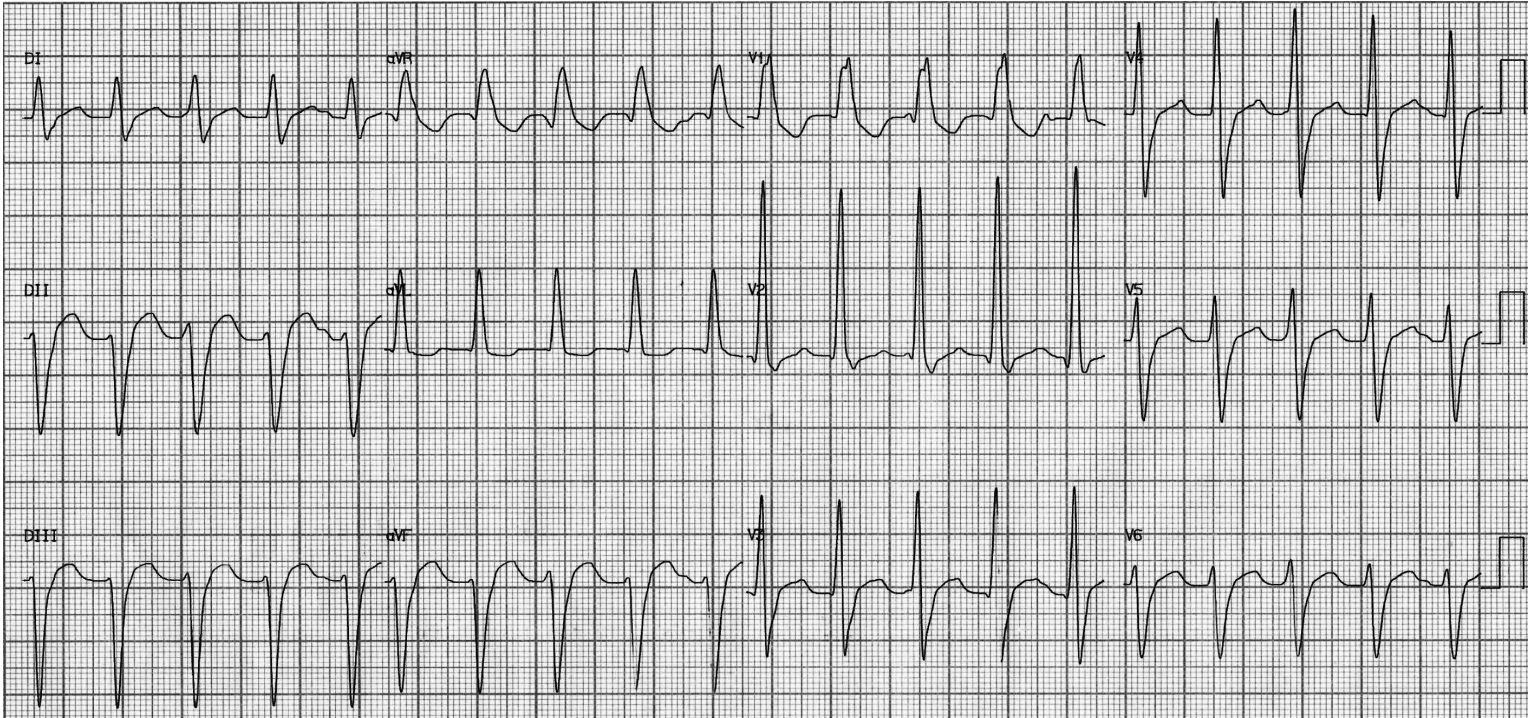
Maya Smith
Assistant to Dr. Mohammad



Final comments



Classical Posterior fascicular VT ($\approx 90\text{-}95\%$ of cases): RBBB-like pattern + extreme left axis deviation; arises close to the left posterior fascicle.



The present case

Right precordial leads comparison

Classic Posterior fascicular VT

V1



V2



QRS duration 120 ms: borderline-broad complex VT ≤ 140 ms

The present case **F**

V1



The fifth beat is narrower and does not have notched at the apex (dissociation) = VT

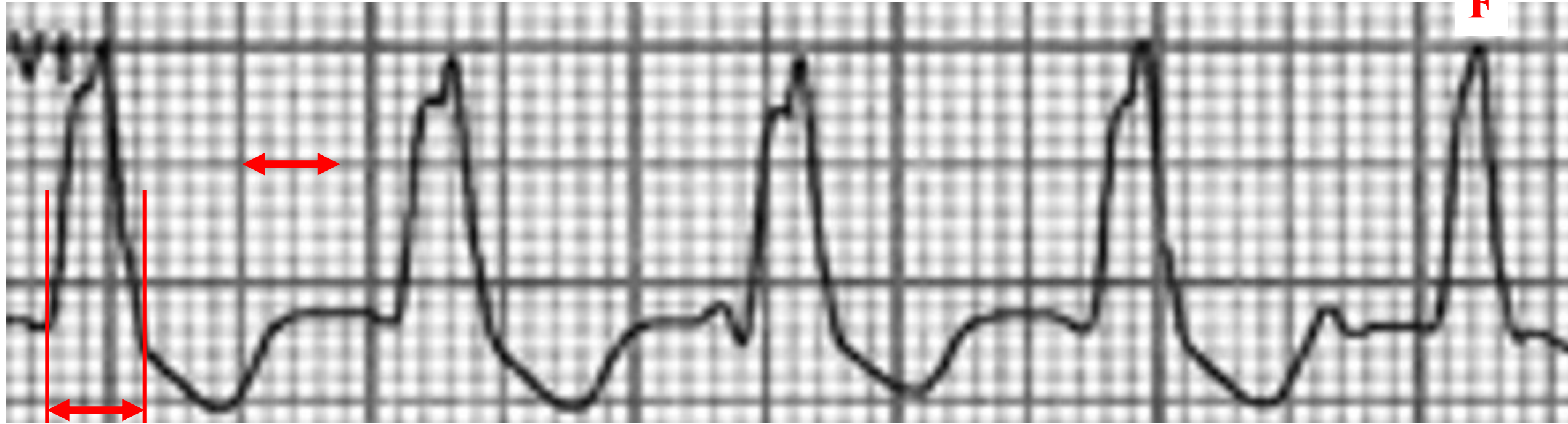
V2



QRS duration 150 ms. Very tall R wave. This pattern is not classical RBBB

The present case

F



QRS duration = 150ms. The fifth beat is a fusion beat (**F**) because it is narrower and does not have notched at the apex (dissociation).

The present case



QRS duration = 150ms

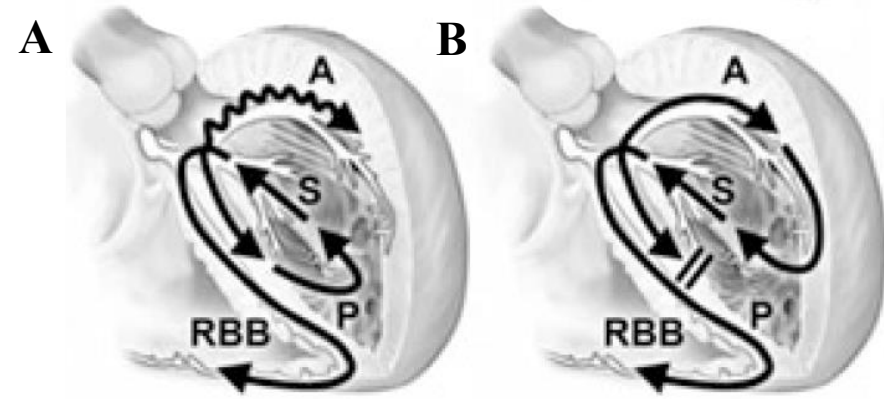
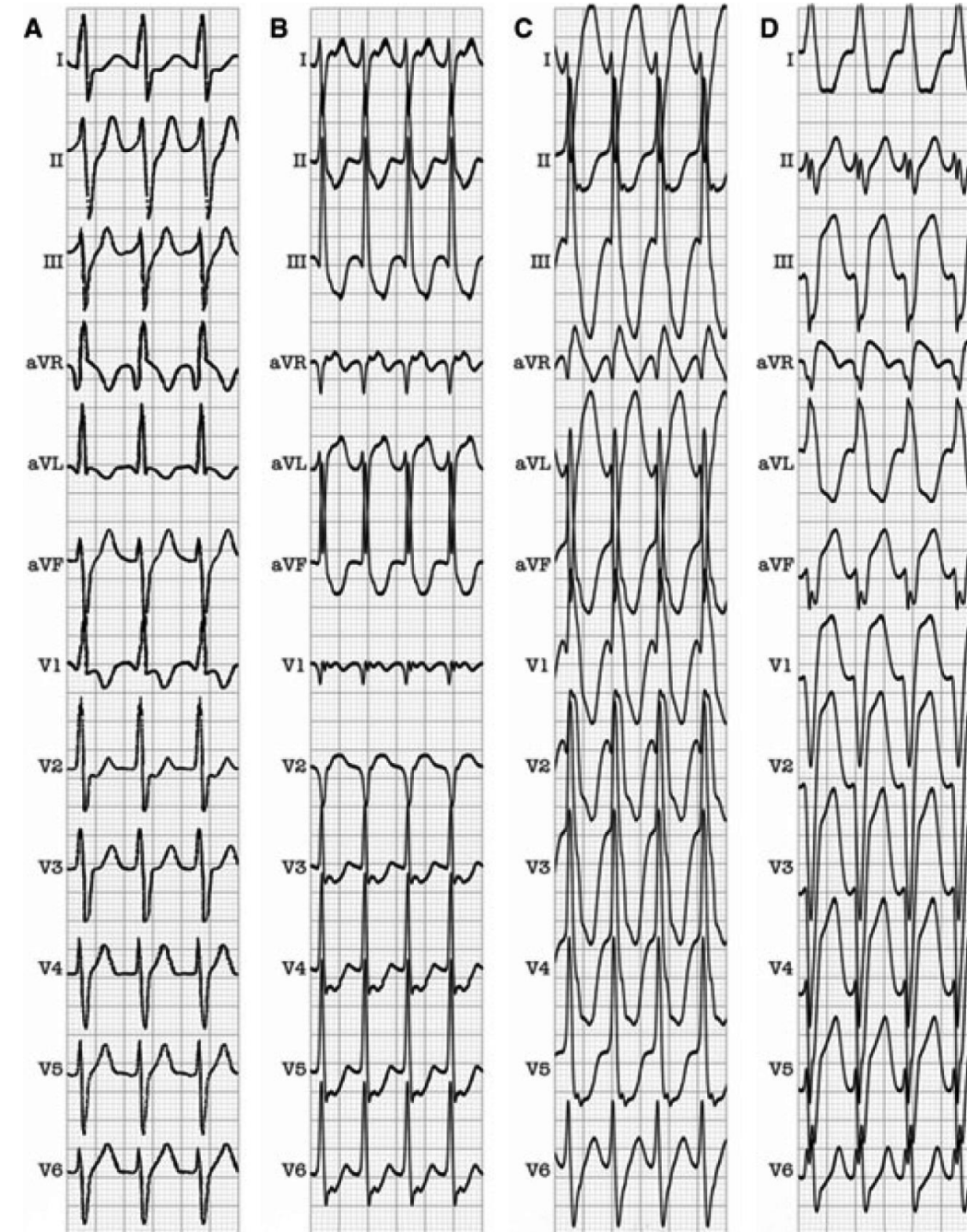
qRS. R voltage = 31 mm!!!. Why is R amplitude too high? (R height = between 31 and 35 mm)

Classical Posterior fascicular VT

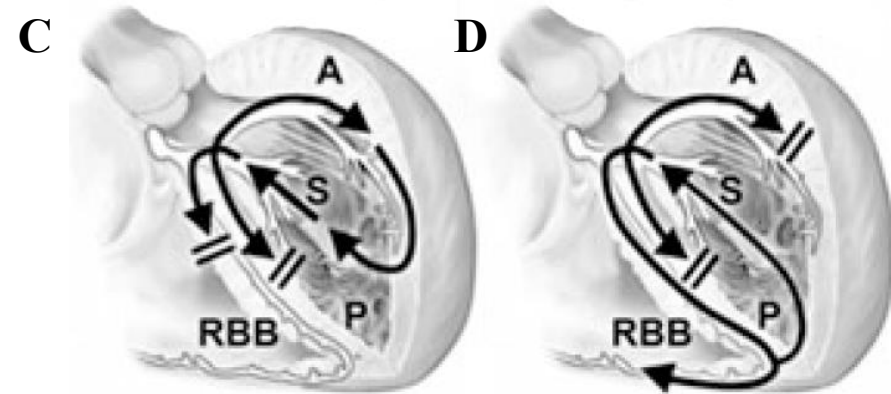


Another component characteristic of idiopathic fascicular VT is borderline QRS complex duration (modest increase in QRS width (≈ 120 - 130 ms)).

Multiform fascicular tachycardia (FT) in the same patient because several circuits are operative (**Sung 2013**)



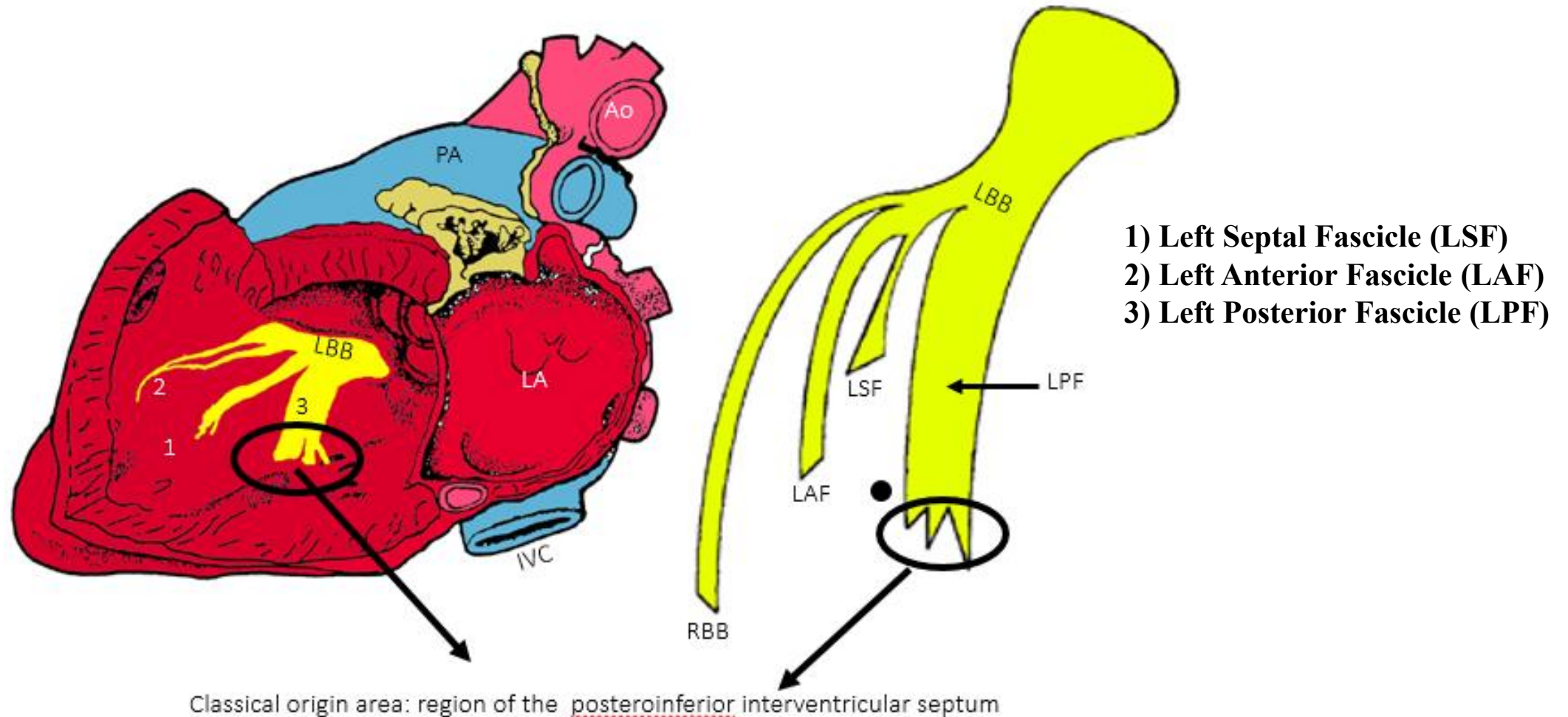
- A. Left posterior fascicular VT morphology prior to ablation. The circuit includes retrograde conduction up the septal fascicle (S) and antegrade conduction down the left posterior fascicle (P), with passive activation of the RBB and likely slow conduction down the left anterior fascicle (A).
- B. VT1 with narrow QRS and right axis deviation. The circuit involves retrograde conduction up S and antegrade conduction down A, with likely block in P. Passive activation of RBB is still present accounting for the narrow QRS.



- C. VT2 with RBBB and right axis deviation. The circuit is the same as in B, with either block or significant delay in the RBB accounting for the new RBB block morphology in comparison to 2B.
- D. VT3 with LBBB and left superior axis. The circuit here is bundle branch reentry, with retrograde conduction likely up S and antegrade conduction down RBB.

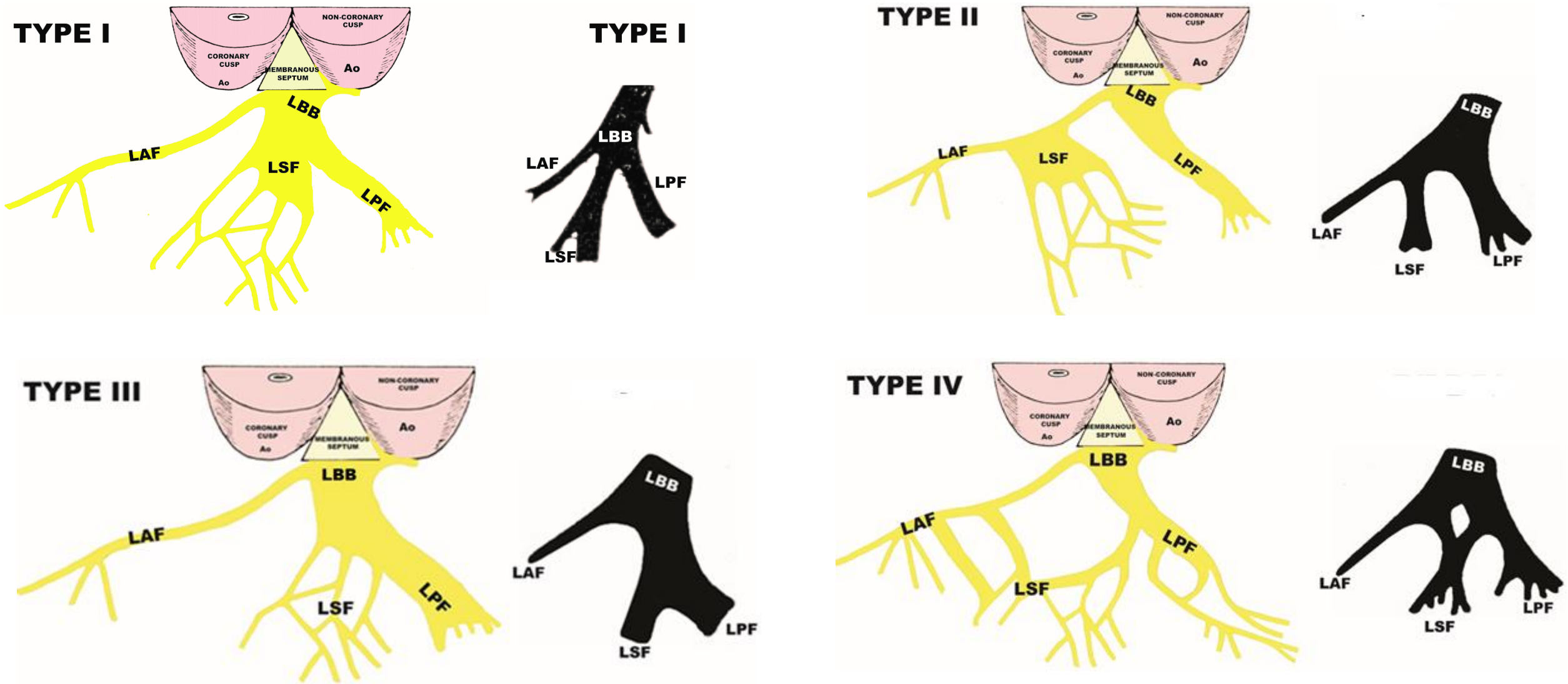
Sung et al present 6 cases of **multiform FT** that suggest functional contribution of a trifascicular left-sided conduction system. Successful ablation therapy relied on suspicion of a fascicular origin on the basis of characteristic surface ECG recordings as well as recording FP in SR and during tachycardia. In addition, the authors emphasize the importance of recording FPs, use of entrainment pacing and 3D electroanatomic maps for successful ablation. For further and complete characterization, they also encourage FP–QRS timing and mapping along both anterior and posterior fascicles to determine interfascicular reentry using the traditional 2 fascicle model versus invocation of a third fascicle (antegrade conduction along both LAF and LPF), and to ablate near the region just apical to the LBB when suspicion of the upper left septal fascicle involvement is present.

Interfascicular Macro-Reentry Mechanism



Supporting evidence of upper septal fascicle or left septal fascicle (LSF)

Anatomic and histopathologic studies by Demoulin and Kulbertus reinforced earlier studies of the trifascicular nature of the left specialized conduction system, demonstrating a third branch in 11 of 20 hearts (**Demoulin 1972; Demoulin 1973**). Kulbertus showed that the majority of examined hearts had a left septal division/fascicle (LSF) easily identifiable, originating from the trunk of the left bundle branch in the majority of cases, or from the LAF and/or LPF in a smaller percentage of cases (**Kulbertus 1975;1976**).



Indeed, extensive ECG and VCG studies have postulated the functional existence of the left septal fascicle (LSF) based on specific ECG/VCG changes consistent with left septal fascicular block (LSFB) (**Iwamura 1978; Dabrowska 1978; Chen 1979; Athanassopoulos 1979; Uchida 2006; Riera AR 2008a,b,c; Pérez-Riera 2011, Pérez-Riera 2016**), and case reports of idiopathic upper septal VT have been previously reported in the literature, with postulated mechanisms including automaticity (**Kottkamp 1995**) and reentry (**Shimoike 2000; Kottkamp 1995**).

Mechanistic and Clinical Implications

While reentry is well established as an underlying mechanism of monomorphic idiopathic VT, the observation that reentry can also result in **multiform VT** has been described for patients with idiopathic VT (**Sung 2013**). In cases of cardiomyopathy or known conduction disease, 3 case reports have been published demonstrating interfascicular reentry between LAF and LPFs resulting in 2 distinct VT forms with alternating morphology (**Lopera 2004; Chien 1992; Crijns 1995**).

In Sung series, 4 of 6 cases had normal HV intervals, and only 2 cases had likely or confirmed HV prolongation. Of the cases with likely baseline HV prolongation, the shift between VT1 and VT2 was spontaneous during tachycardia without tachycardia termination, implicating the presence of a third fascicle. Radiofrequency catheter ablation (RFCA) was successful in terminating fascicular VT for all cases, guided by 3D electroanatomic mapping and segmental modification of the conduction system targeting FPs critical to the circuit (**Nakagawa 1993; Zardini 1995**). Their results are consistent with a prior series describing RF application at a site remote to the VT exit that achieved procedural success. However, only by ablating the auxiliary fascicle did they terminate all VT forms. Ablation of only the antegrade limbs (LPF and LAF) resulted in bundle branch reentrant tachycardia (BBRT) due to persistence of the retrograde limb.

Successful ablation of the auxiliary fascicle in most cases is based on concealed entrainment mapping, localizing concealed entrainment to the region just apical to the LBB. Although this would be the preferred technique, it may not be unreasonable to attempt a line of ablation just distal to the LBB extending from a region close to the LAF to a the region close to the LPF in an attempt to transect the auxiliary fascicle. The entity of **multiform FT** should be differentiated from BBRT and myocardial VT. In all patients, a distinct FP was recorded both in sinus (or atrial paced) rhythm and during VT. When variations in the tachycardia cycle length (CL) were observed, changes in FP–FP drove changes in V–V. In addition, the FP preceded the ventricular electrogram, and the HV interval was very short during FT. Together, these observations serve to exclude BBRT and myocardial VT. The exceptions are the LBBBVTs where BBRT was the likely mechanism for VT after left sided fascicular VT ablation.

Comprehensive maps of all VT circuits and extensive entrainment mapping could not be performed in every case. However, concealed entrainment of one or more VT forms is possible strongly suggestive of a reentrant mechanism. Also, Sung et al. hypothesize use of a separate functioning fascicle (septal) to best explain the observations in this series of cases, but they cannot exclude the possibility of multiple diseased fascicles producing similar patterns. However, the presence of normal HV intervals lending credence to a functional third fascicle rather than disease in the HPS. Previous studies of FT have used multipolar catheters to clearly demonstrate antegrade versus retrograde conduction along individual fascicles (**Nogami 1998; Nogami 2000**). Although Sung et al did not use multipolar mapping of individual fascicles, they believe that the combination of 3D electroanatomic mapping to define spatial location and multipoint fascicular activation mapping with the ablation catheter is sufficient to determine directionality of fascicular conduction.

Conducted

1. With mild sedation and local anesthesia. TWO punctures were made in right femoral vein and ONE puncture in right femoral artery to position catheters: deflectable decapolar catheter in the CS + fixed quadripolar catheter at the RV tip. Through arterial puncture, deflectable decapolar catheter was placed in the left ventricle for activation map construction.
2. Electroanatomic mapping (Ensite Nav’x, SJM) – activation of ventricular tachycardia with emphasis on PRE-PURKINJE (P1) and PURKINJE potentials (P2).
3. Ventricular tachycardia throughout the test. Different spontaneous interruptions of VT during the manipulation of catheters with EASY INDUCTION BY ATRIAL PACING in all attempts.
4. Through activation mapping of P1 and P2 potentials, we observe ventricular tachycardia by reentry, the circuit of which used a descending pathway at the level of medium IVS (area of slow conduction) and ascending direction through the posteroinferior fascicle.
5. 2:1 ventriculo-atrial conduction during tachycardia.

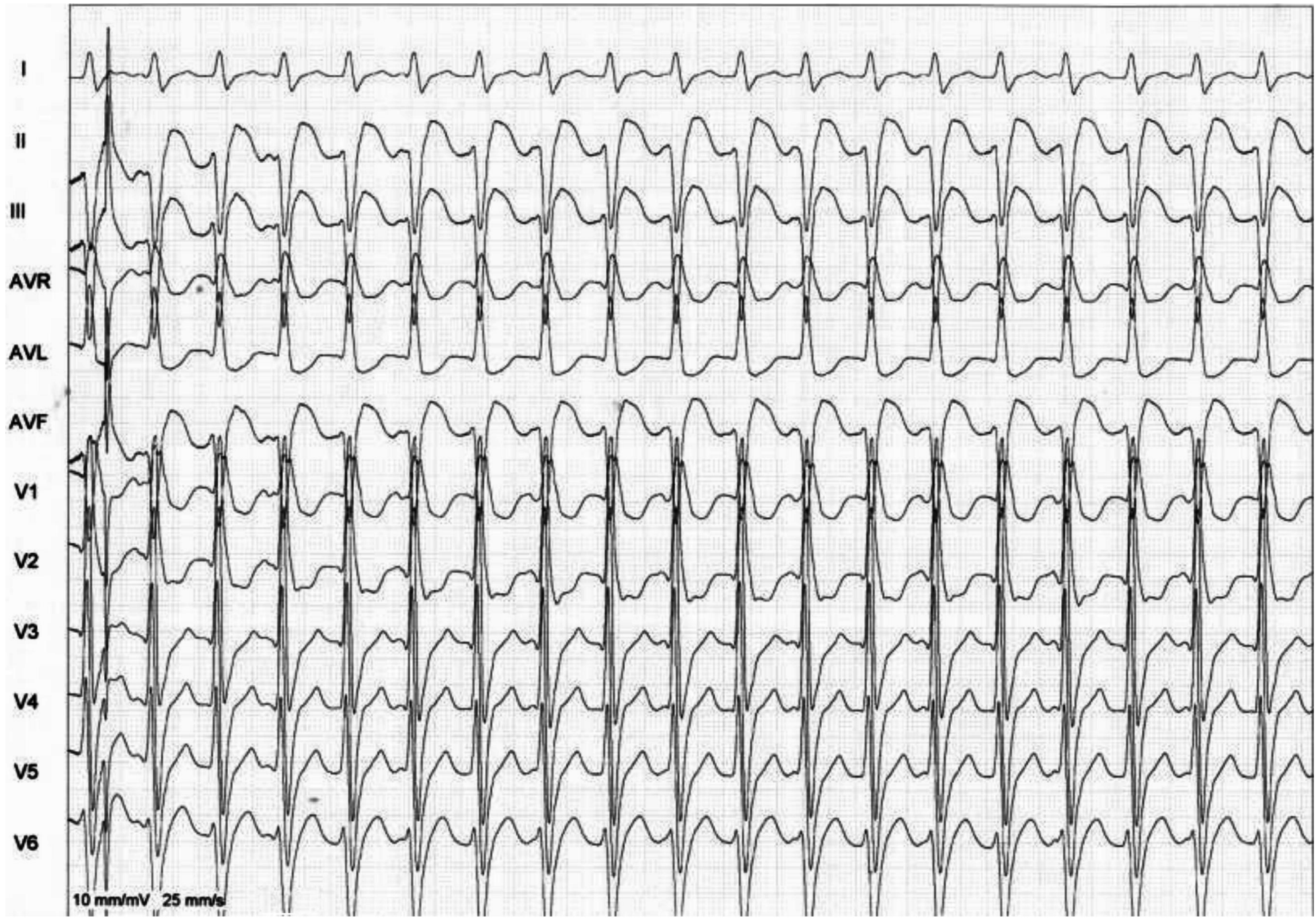
Performed

1. Ablation of tachycardia circuit based on P1 and P2 potentials, seen in the left posterior septal area and confirmed with the activation map and entrainment maneuvers.
2. We performed RF application (50 W, 70°) by 8 mm ablation catheter, in the region with greatest precocity and with evidence of P2 very near the ventricular electrogram. Immediate reversion of ventricular tachycardia preceded by cycle reduction (480 → 510 ms) before interruption. New applications of consolidation increasing the lesion area.
3. By programmed atrial and ventricular pacing with cycles of 500 and 400 ms, besides one and two extra-stimuli, it was NOT possible to induce new episodes of ventricular tachycardia.

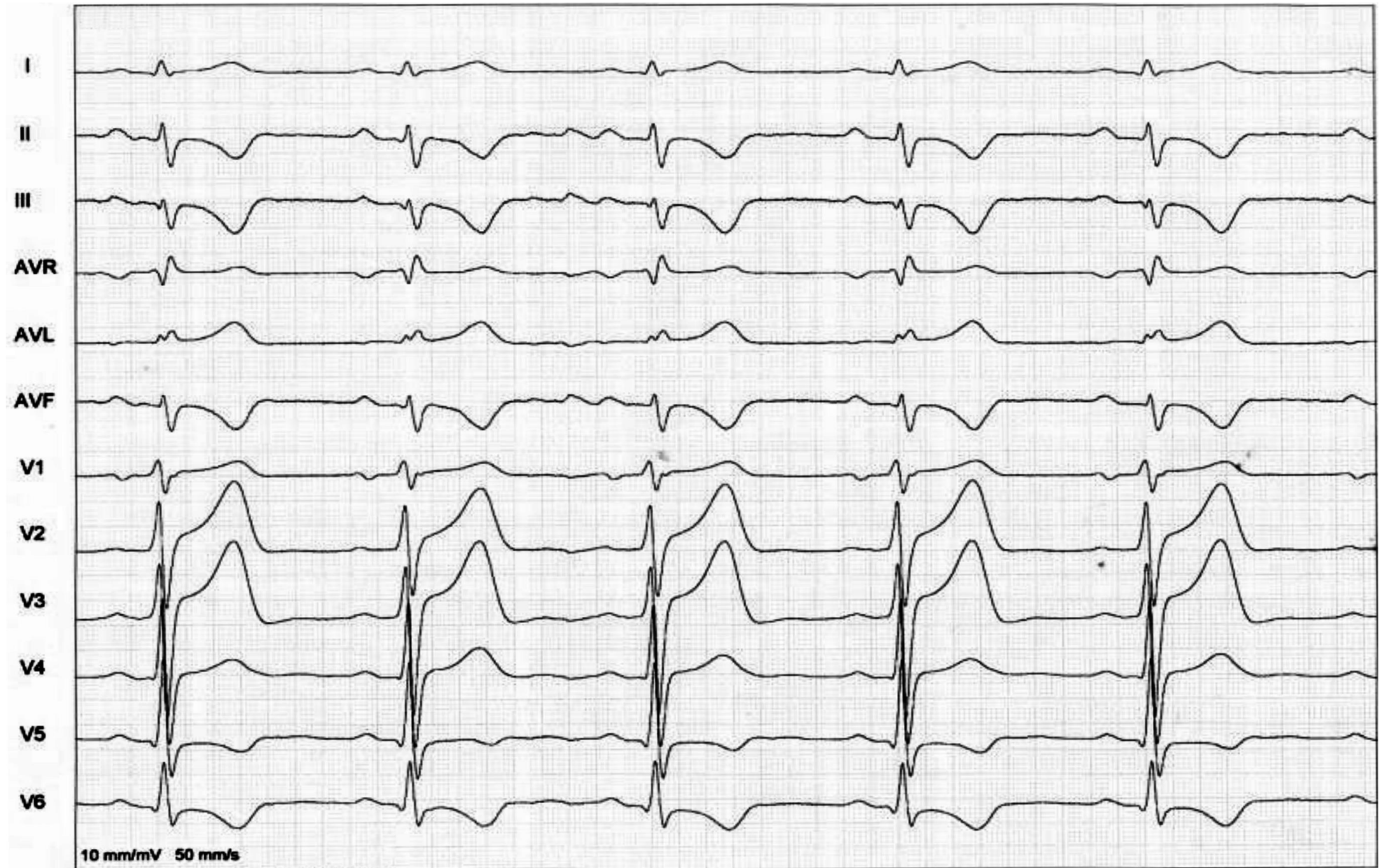
Diagnosis

1. Ventricular tachycardia of the posterior fascicular type
2. Successful ablation of ventricular tachycardia
3. Procedure with no complications

Pre-ablation

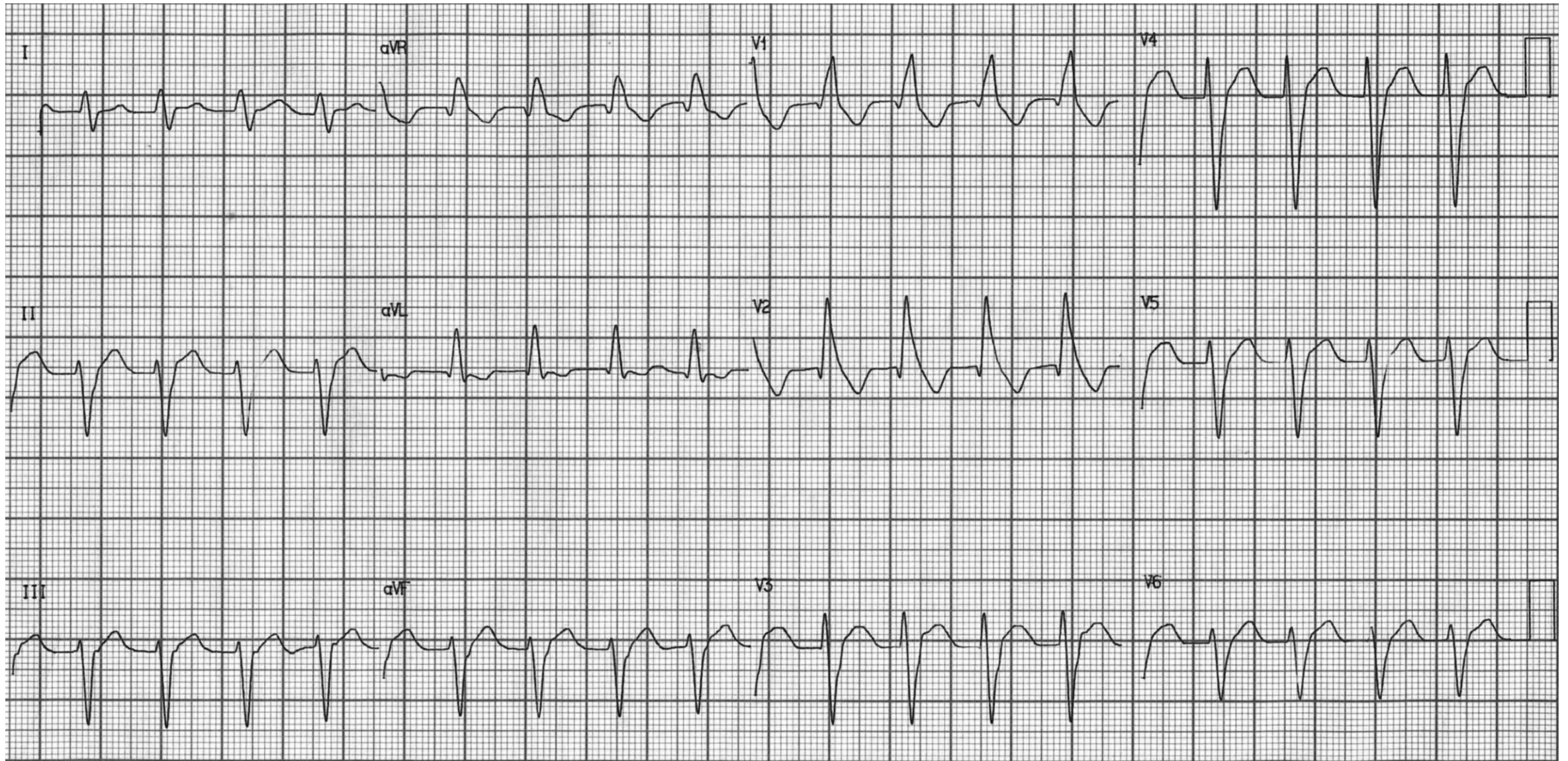


After-ablation



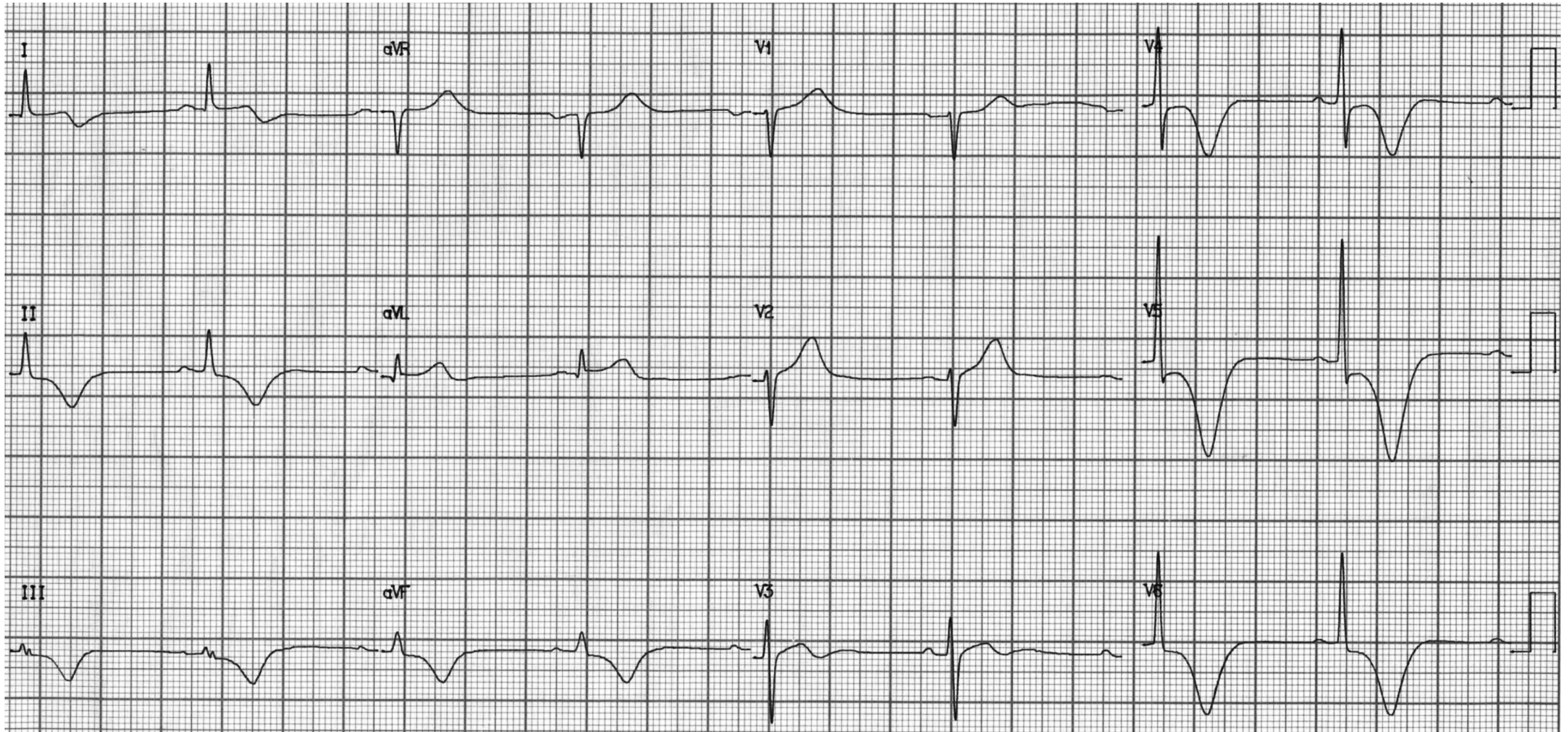
Another example

During the event at Admission



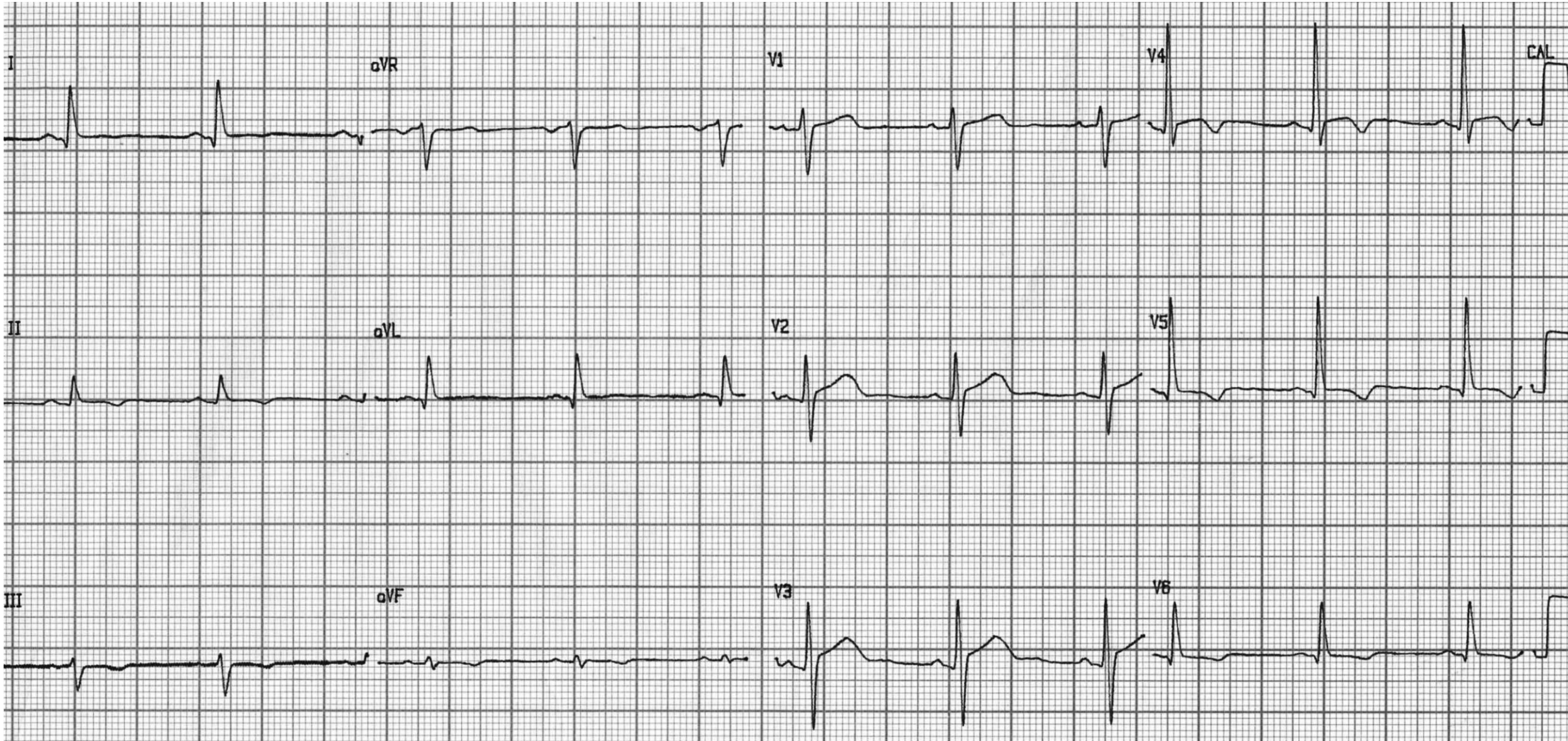
Left Posterior fascicular VT/Posterior fascicular ventricular tachycardia (P-IFLVT). (90-95% of cases): RBBB-configuration+ superior axis in the frontal plane (left anterior fascicular block-like pattern)

ECG performer immediatly after intravenous verapamil

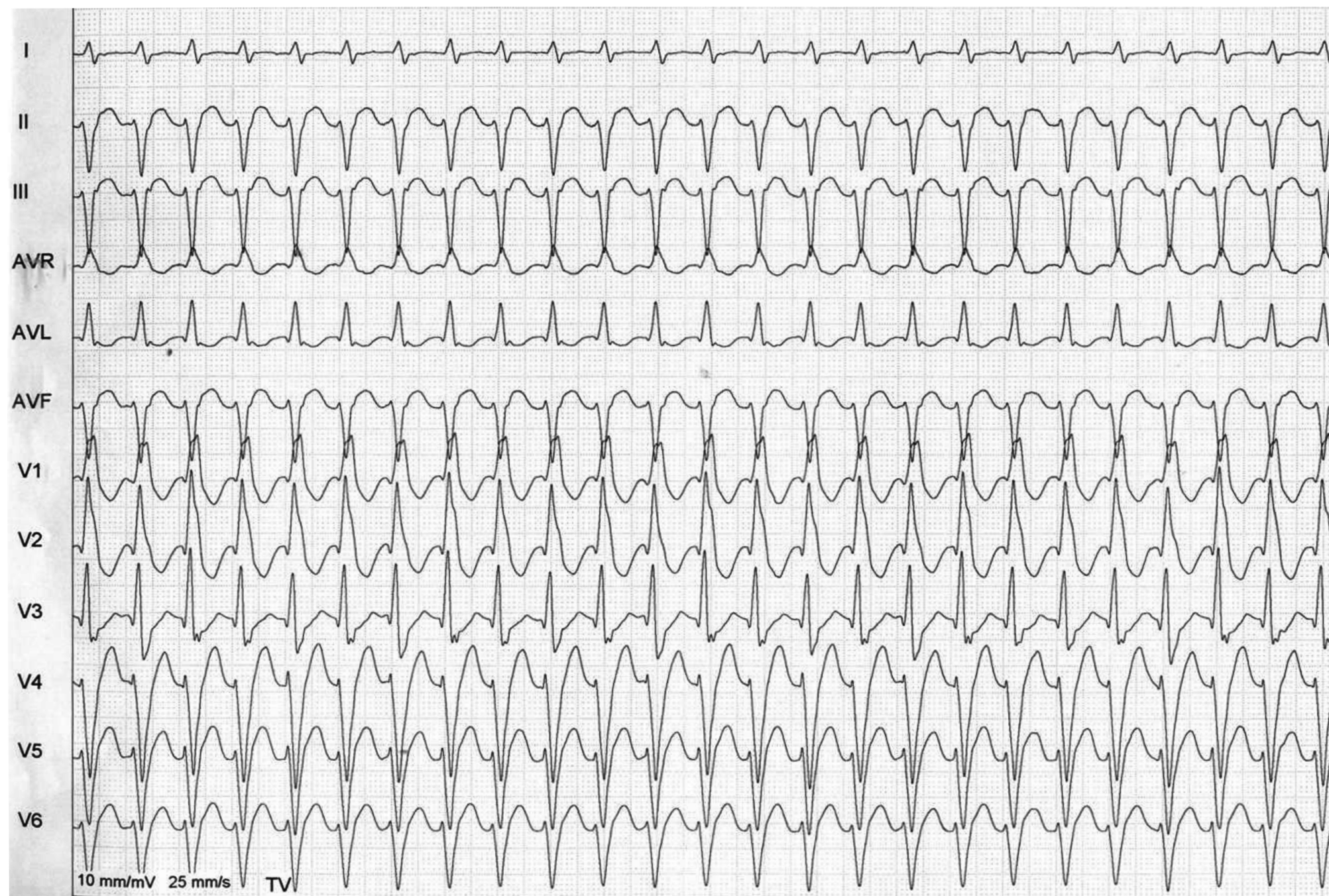


Deep negative T-waves from V4 to V6 ($\geq 10\text{mm}$) and in inferior leads: Cardiac Memory mechanism. Most patients present T-wave inversion immediately after tachycardia (cardiac memory).

24 hours after ending the event



Significant reduction of memory T-wave



After ablation



Negative T-waves from V4 to V6 and in inferior leads: Cardiac Memory mechanism. Most patients present T-wave inversion immediately after tachycardia (cardiac memory).

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