Repetitive episodes of paroxismal lone atrial fibrillation associated with “cardiac memory” or “Chatterjee phenomenon”

https://ekgvcg.wordpress.com/

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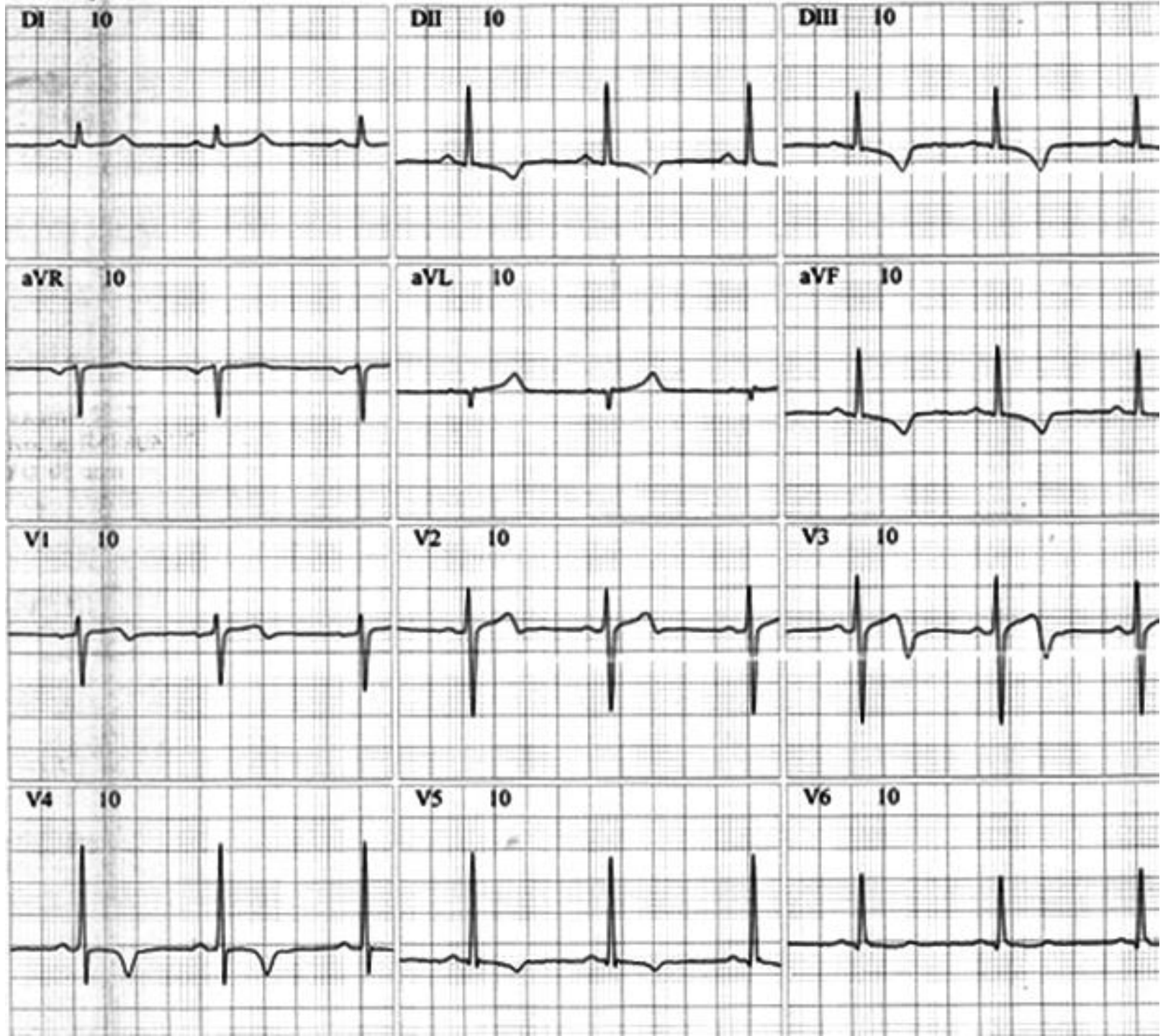
Português - Reporte de caso

Masculino, branco, 51 anos, , mecânico, natural de Fortaleza, Ceará - Brasil.
Antecedentes de vários surtos sucessivos de fibrilação atrial paroxística de fácil reversão espontânea. Num destes surtos ocorreu episódio de acidente vascular cerebral isquêmico transitório sem sequelas.
Antecedentes: ausência de fatores de risco ou causa aparente: diabetes, hipertensão, dislipidemia, cardiopatia estrutural, hipertiroidismo, antecedentes familiares, sobrepeso ou obesidade, fumo, álcool ou drogas. Em virtude destes achados clínicos e elétricos mostrados nos ECG, VCG e Holter, foi realizado ecocardiograma transtorácico e transesofágico, que resultaram totalmente normais. Evidencias clínicas e ecocardiográficas de doença cardiovascular ou pulmonar foi afastado.
Foi realizado cineventriculocoronariografia há três anos, motivado pelos fatos clínicos e as alterações eletrocardiográficas.
Atualmente em uso de warfarina em dose de acordo ao INR.
Pergunta:
Quais os diagnósticos eletrovetorcardiográficos?

English - Case report

Male, Caucasian, 51 years old, mechanic, native from Fortaleza, Ceará - Brazil.
History of successive episodes of paroxysmal atrial fibrillation with easy spontaneous reversion. In one of these episodes occurred transient ischemic attack (TIA) without sequelae.
Background: absence of risk factors or apparent cause: diabetes, hypertension, dyslipidemia, structural heart disease, hyperthyroidism, family history, overweight or obesity, smoking, alcohol or drugs use.
Because of these clinical and electrical findings shown in the ECG, VCG and Holter was performed transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE), which resulted totally normal. Clinical and echocardiographic evidence of cardiovascular or pulmonary disease has been ruled out.
Ventricular coronary angiography was held three years ago, motivated by clinical features and electrocardiographic changes.
Currently taking warfarin at a dose according to the range of International Normalized Ratio (INR).
Question:
What are the electrovectorcardiographic diagnosis?

Basal ECG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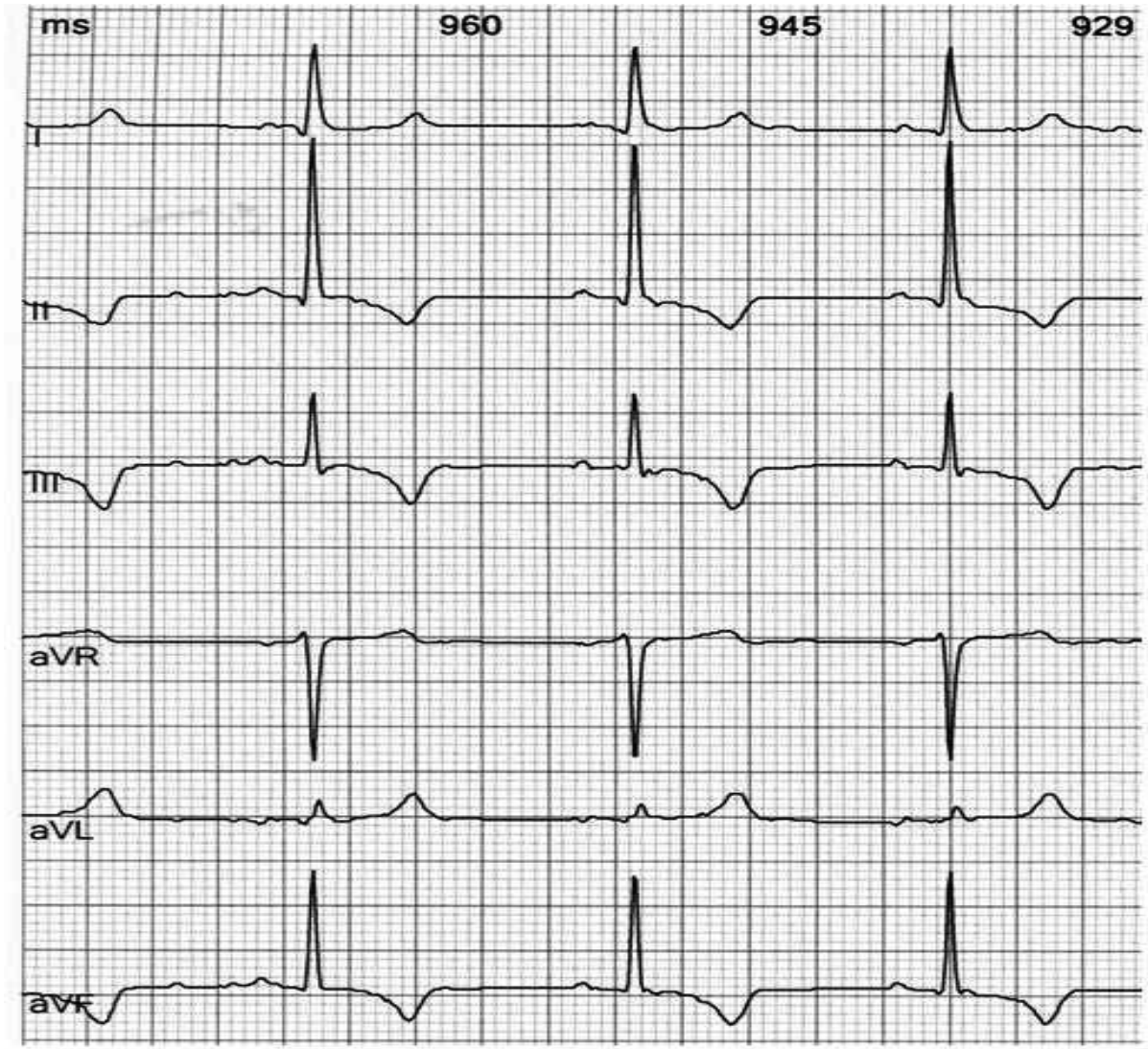
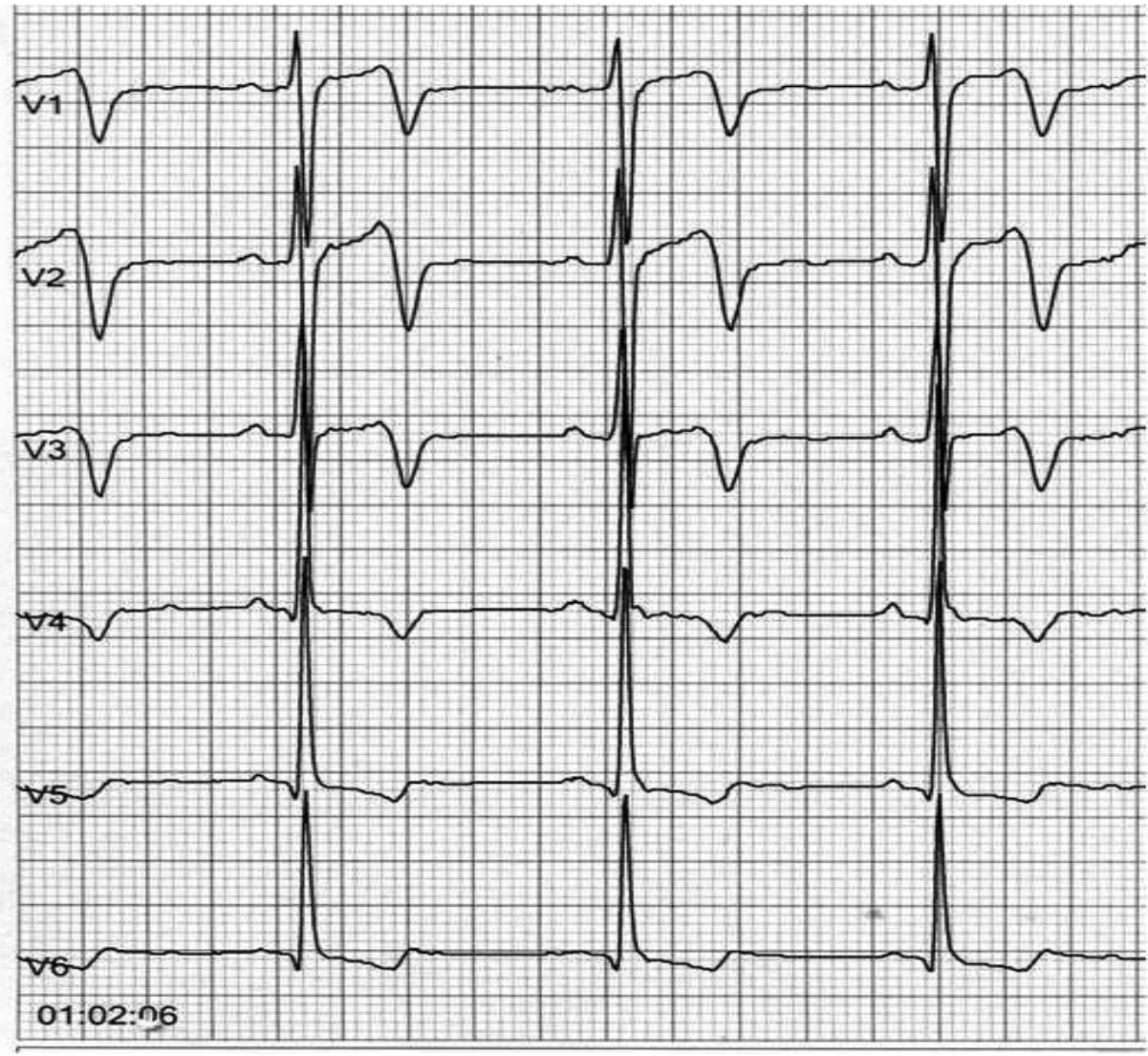
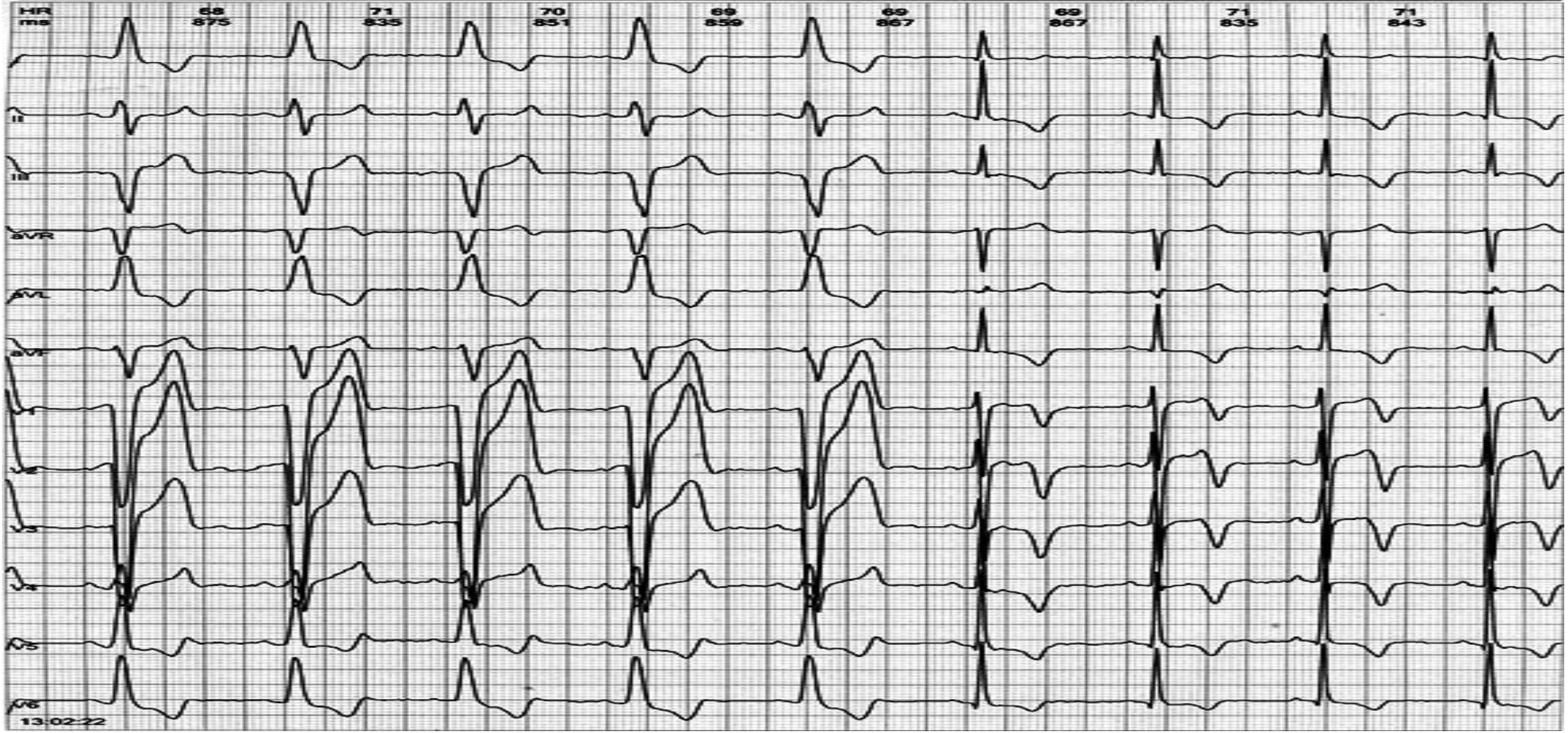
ECG during Holter Monitoring register



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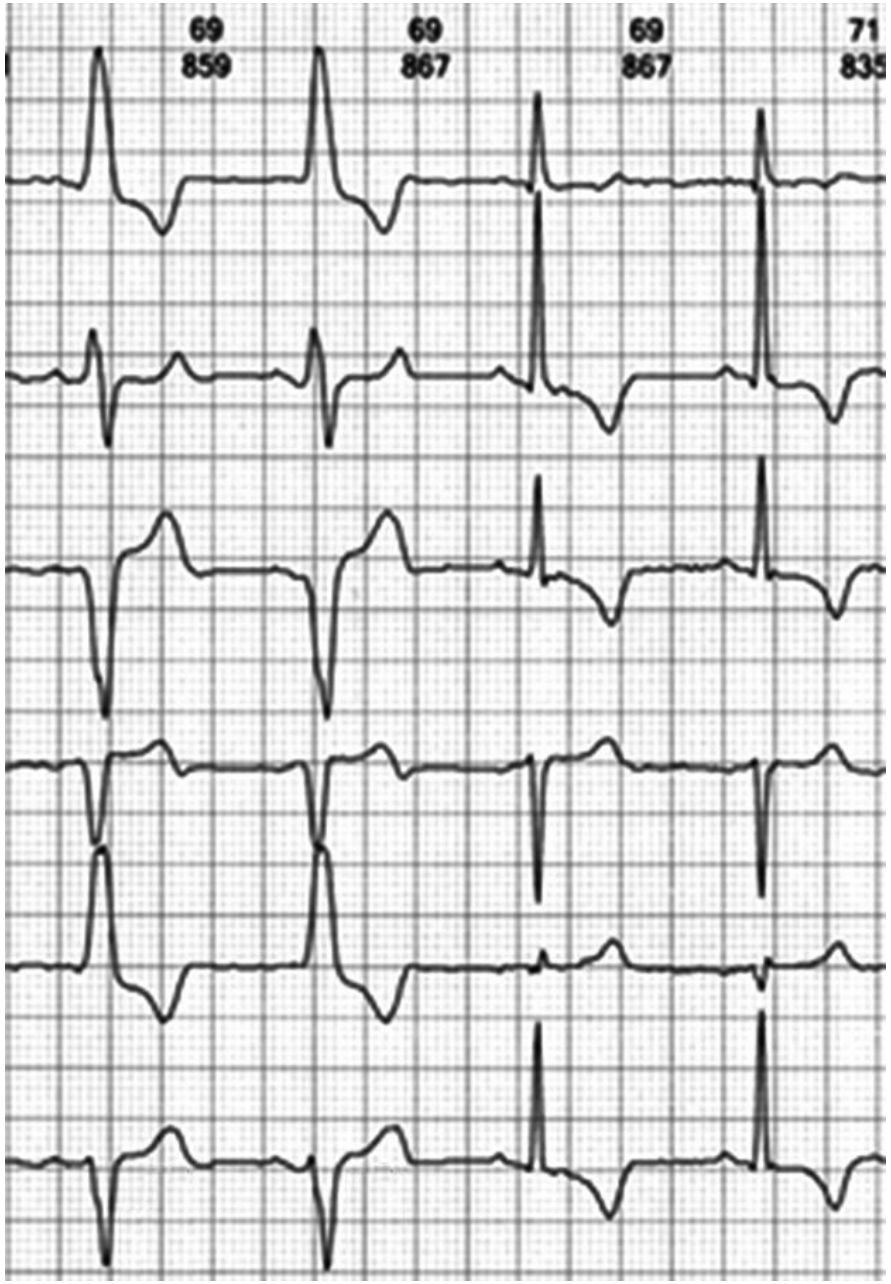
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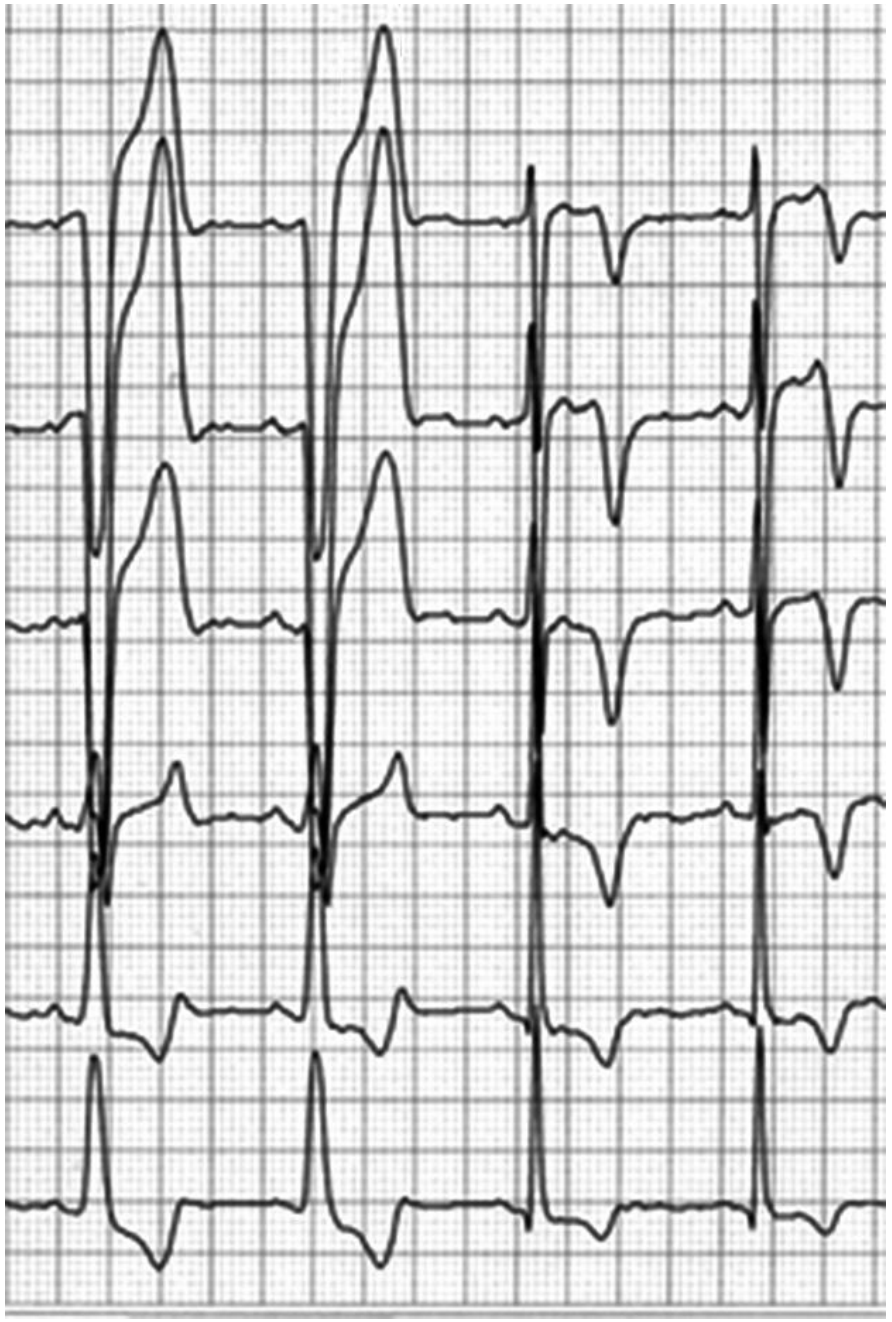
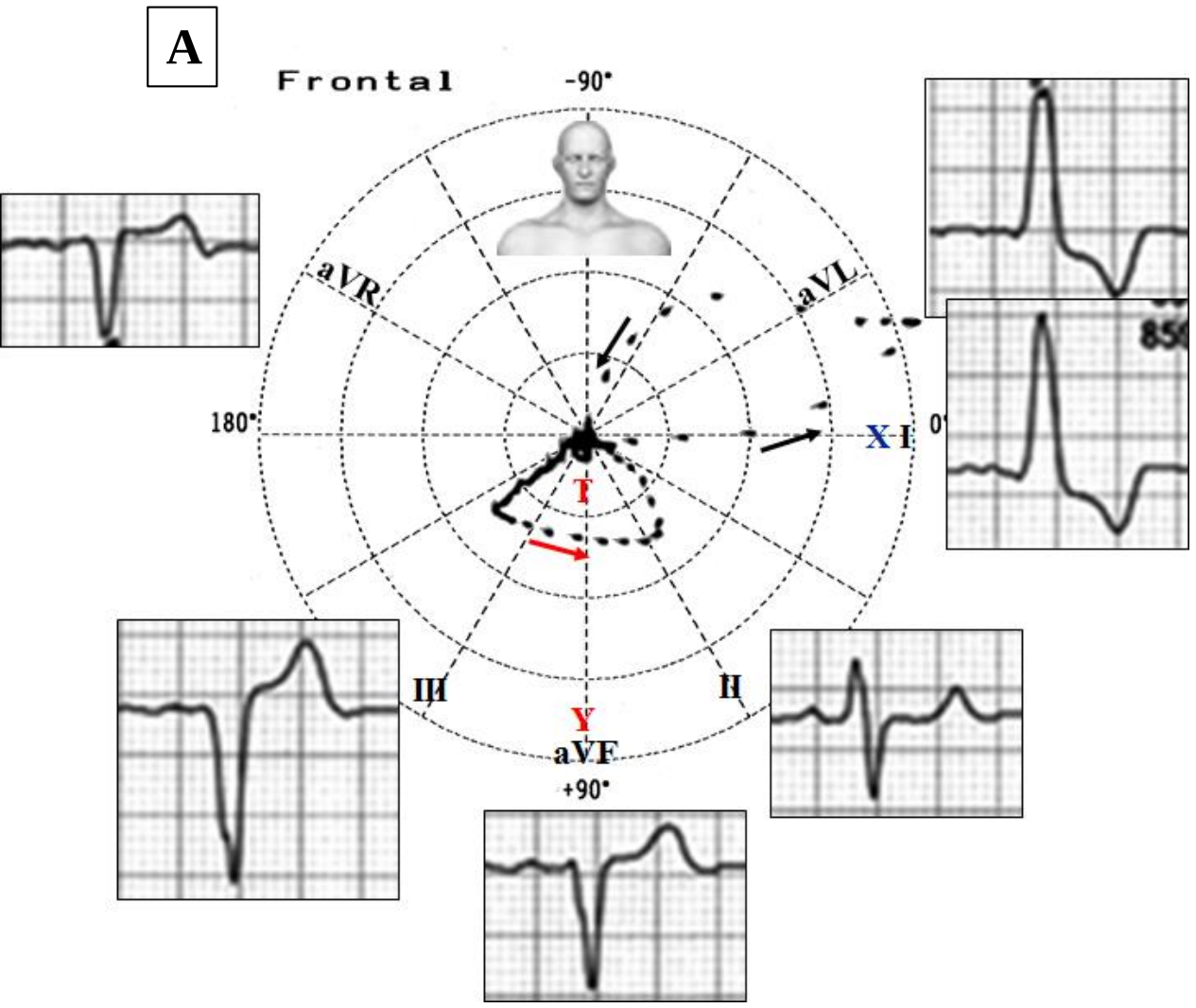
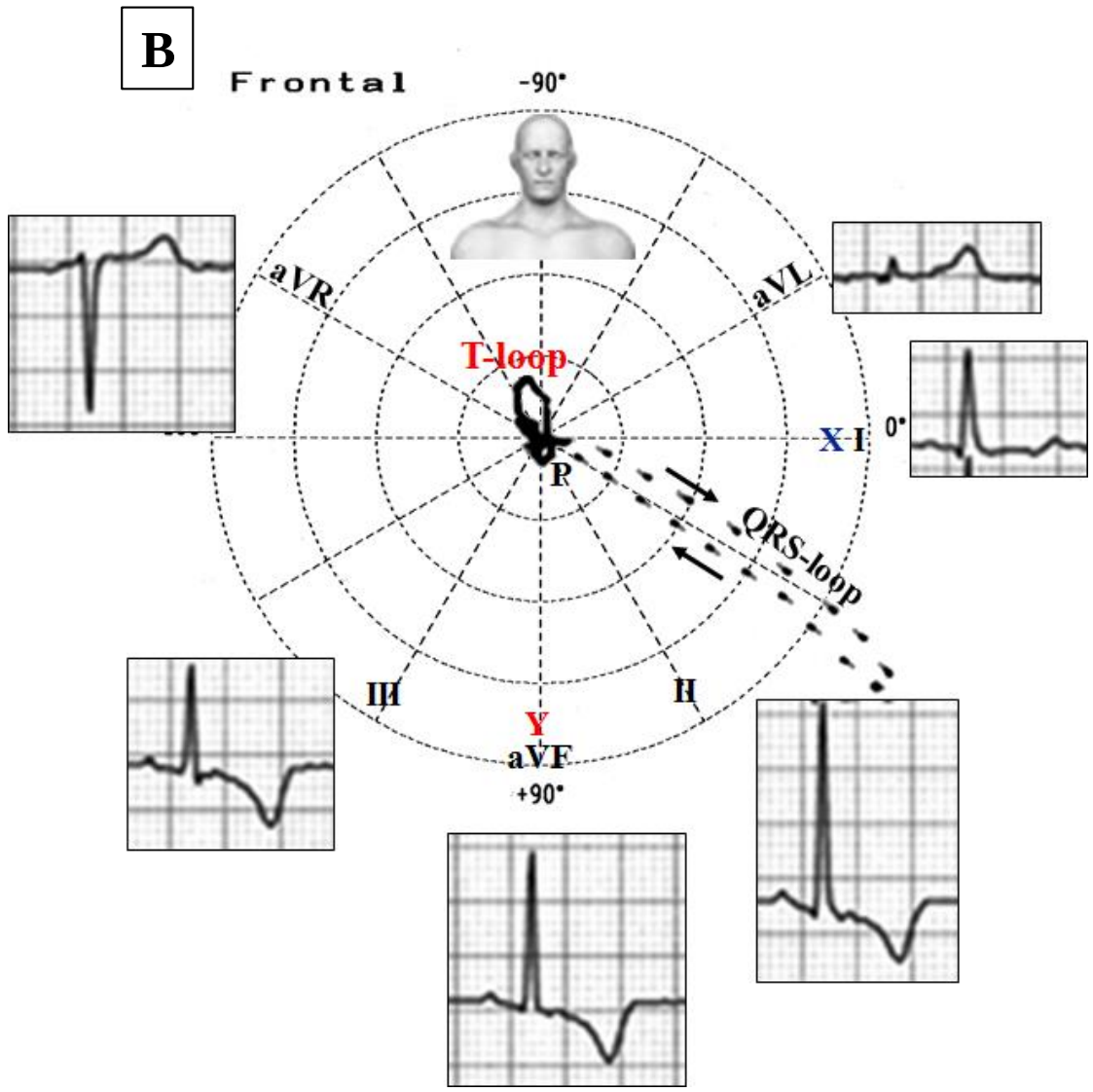
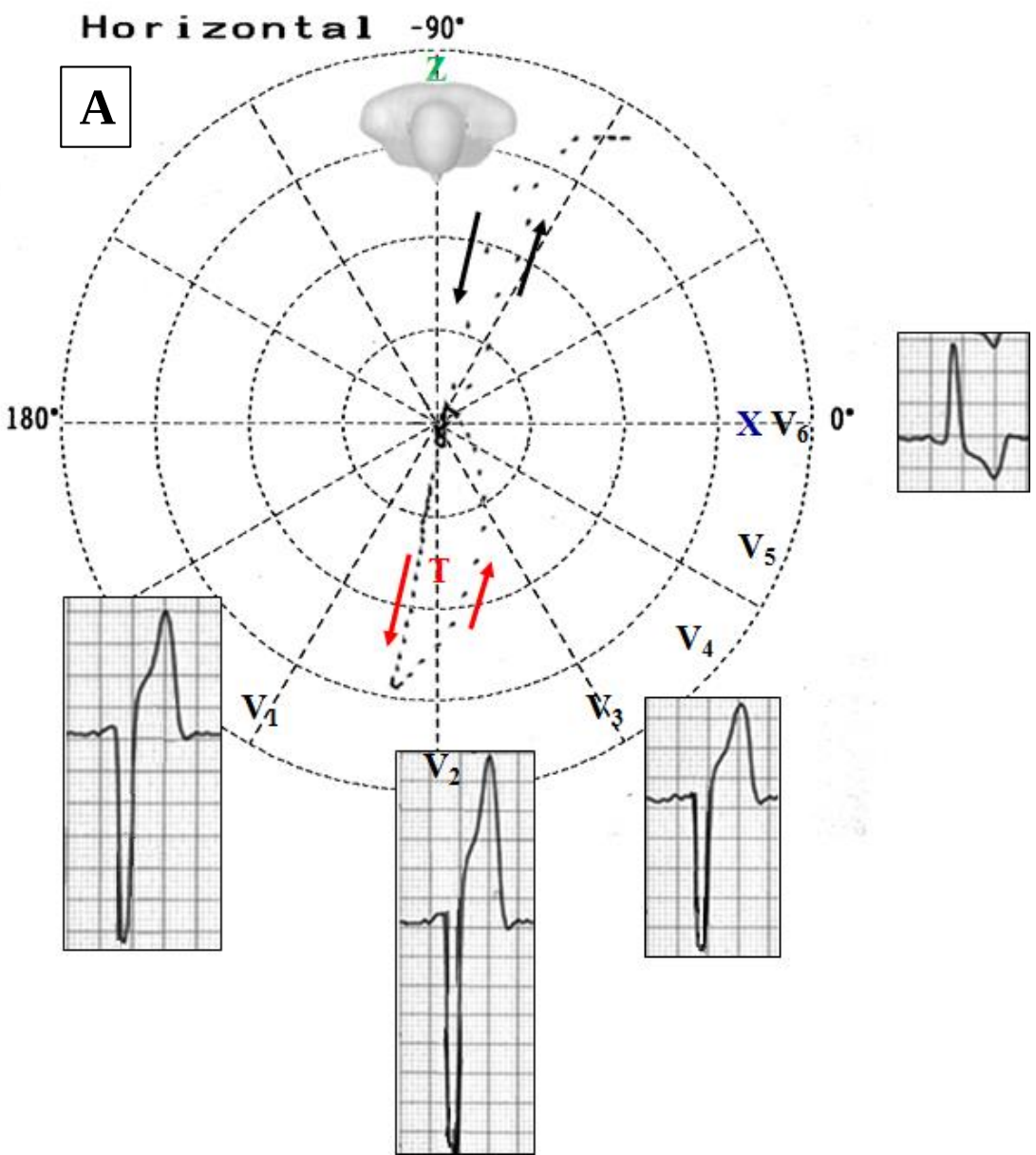


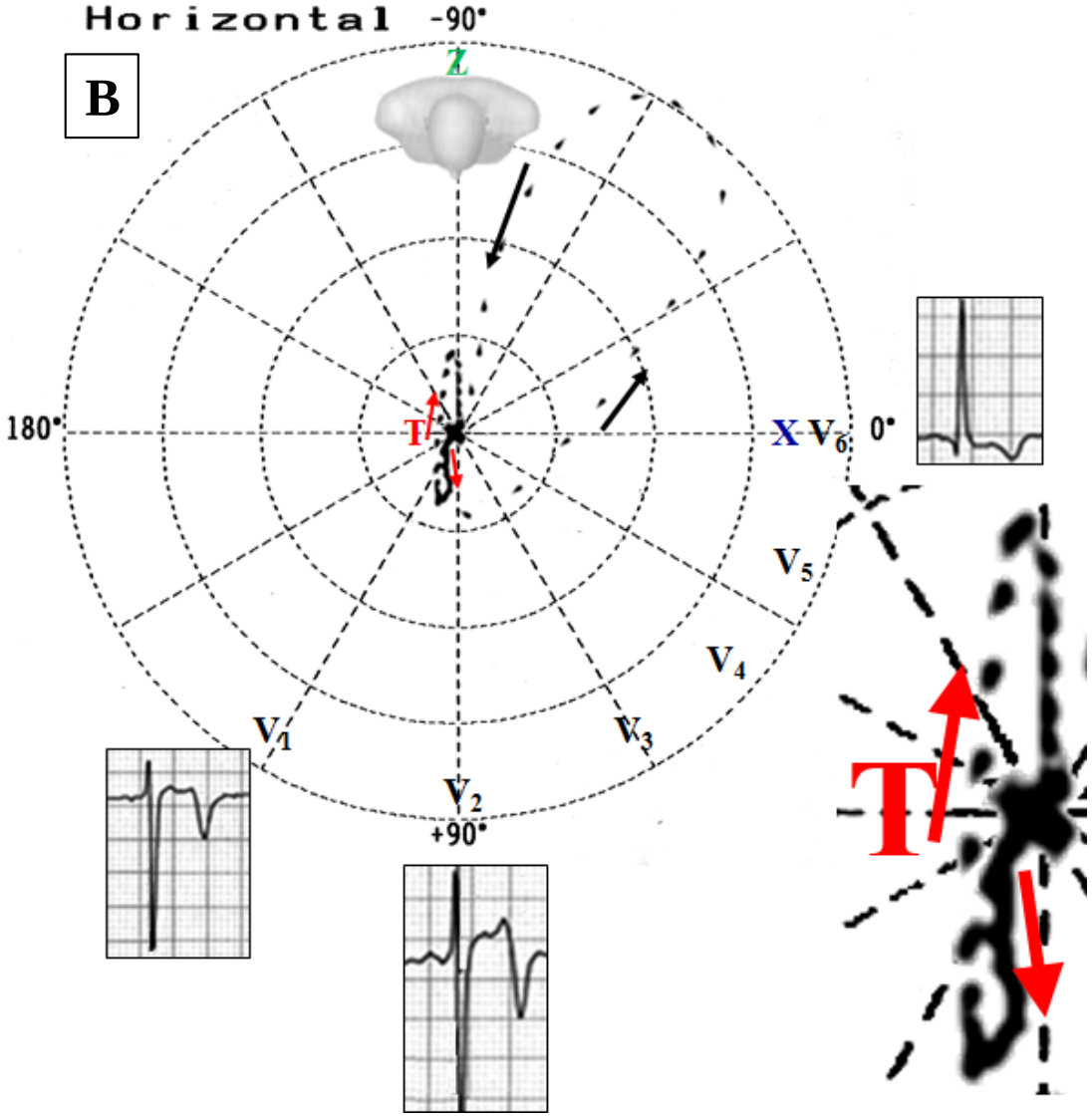
ECG/VCG correlation in the Frontal Plane with (A) and without (B) conduction disturbance



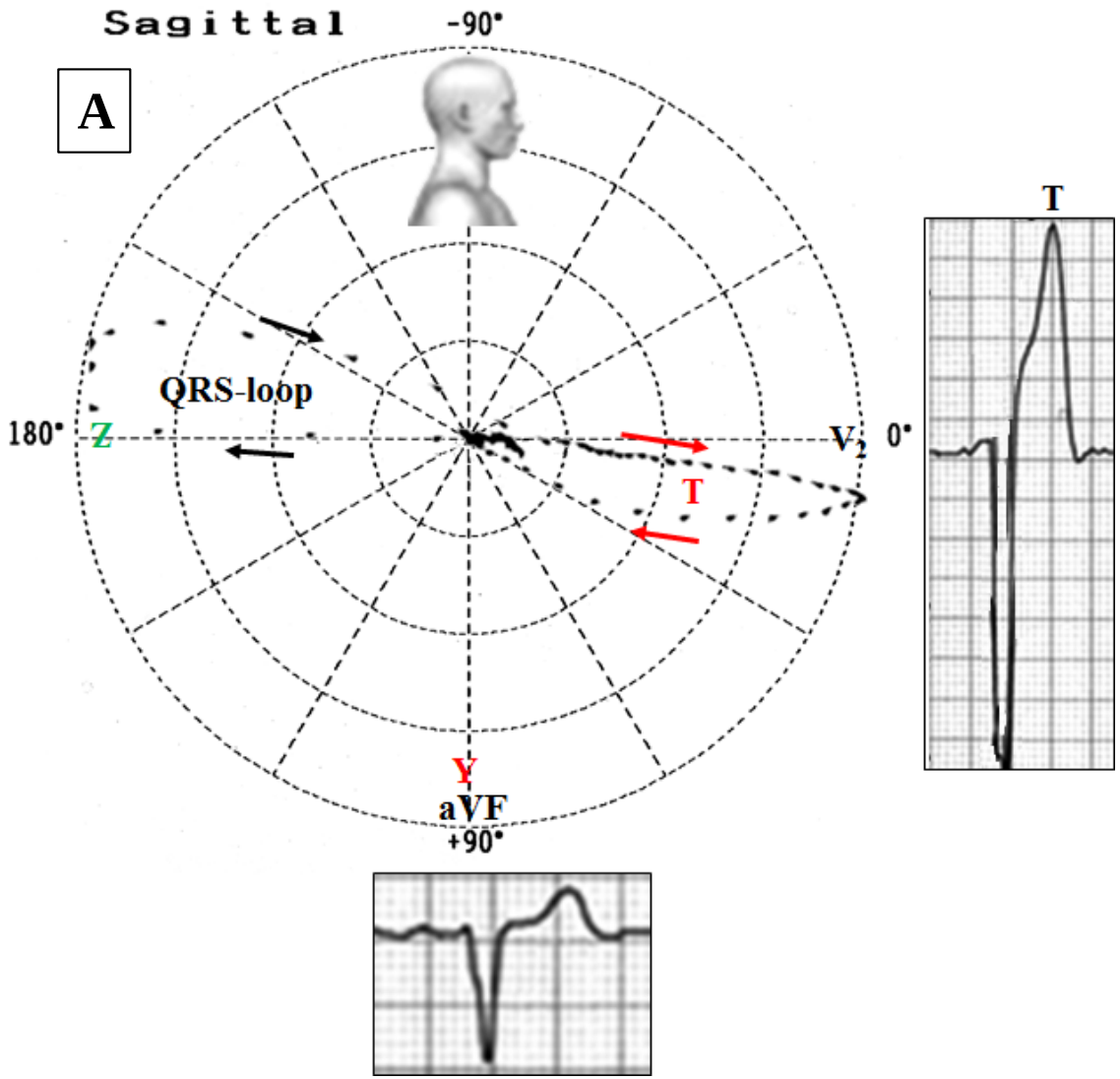


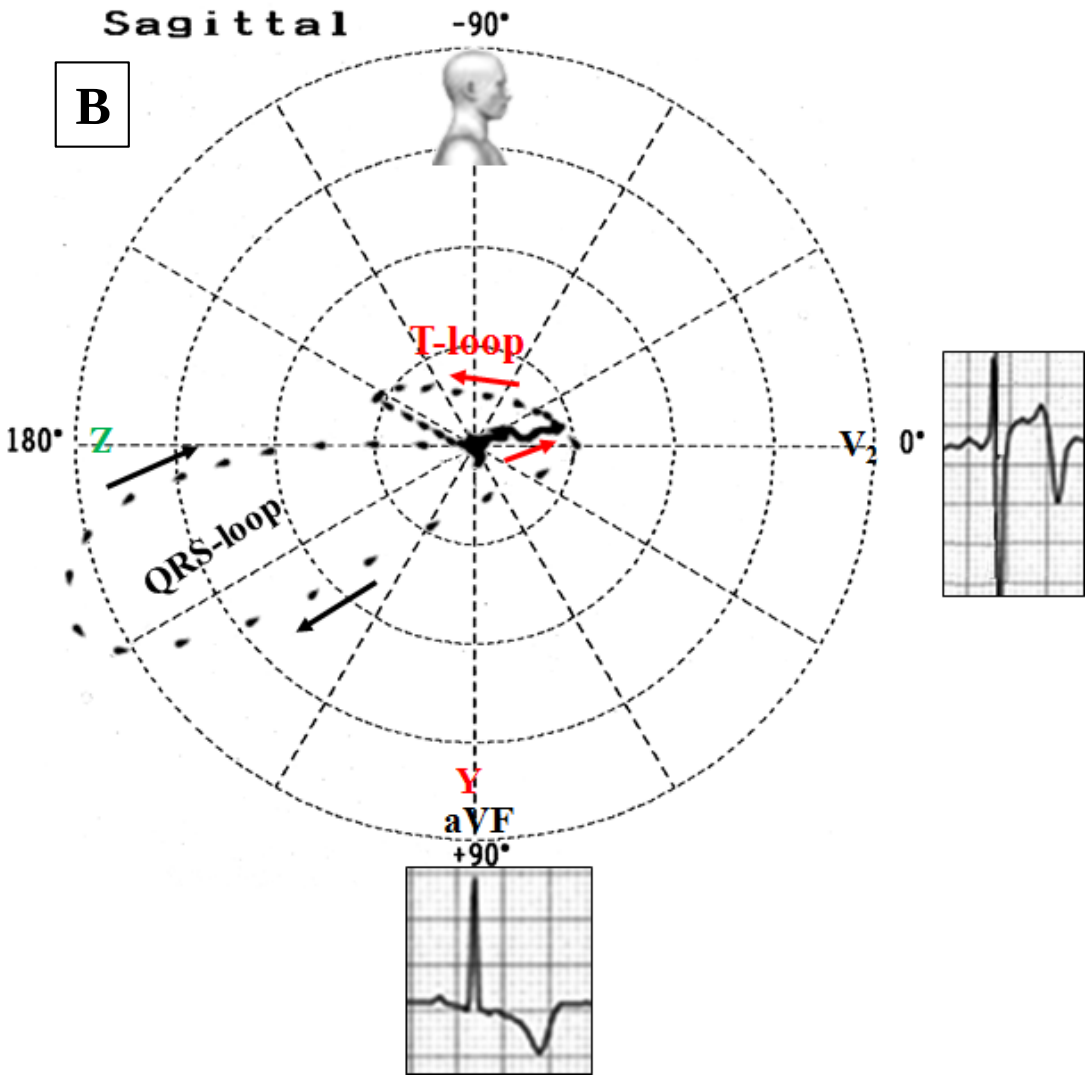
ECG/VCG correlation in the Horizontal Plane with (A) and without (B) conduction disturbance





ECG/VCG correlation in the Right Sagittal Plane with (A) and without (B) conduction disturbance





Opinion of colleagues

This patient has intermittent left bundle branch block. There are not too many sinus cycle lengths to measure, but it appears that LBBB occurs with longer cycle length (bradycardia or phase-4 BBB). Initially, I thought that the negative T waves when the QRS normalizes were only due to cardiac memory. However, the T wave axis is slightly different than the QRS axis during LBBB. In cardiac memory there is perfect concordance between the QRS axis during abnormal depolarization and the T wave axis when depolarization normalizes. Therefore, I suspect there is an intrinsic abnormality in addition to the intermittent conduction disease. This can be coronary artery disease or myocardial disease.

Thank you,

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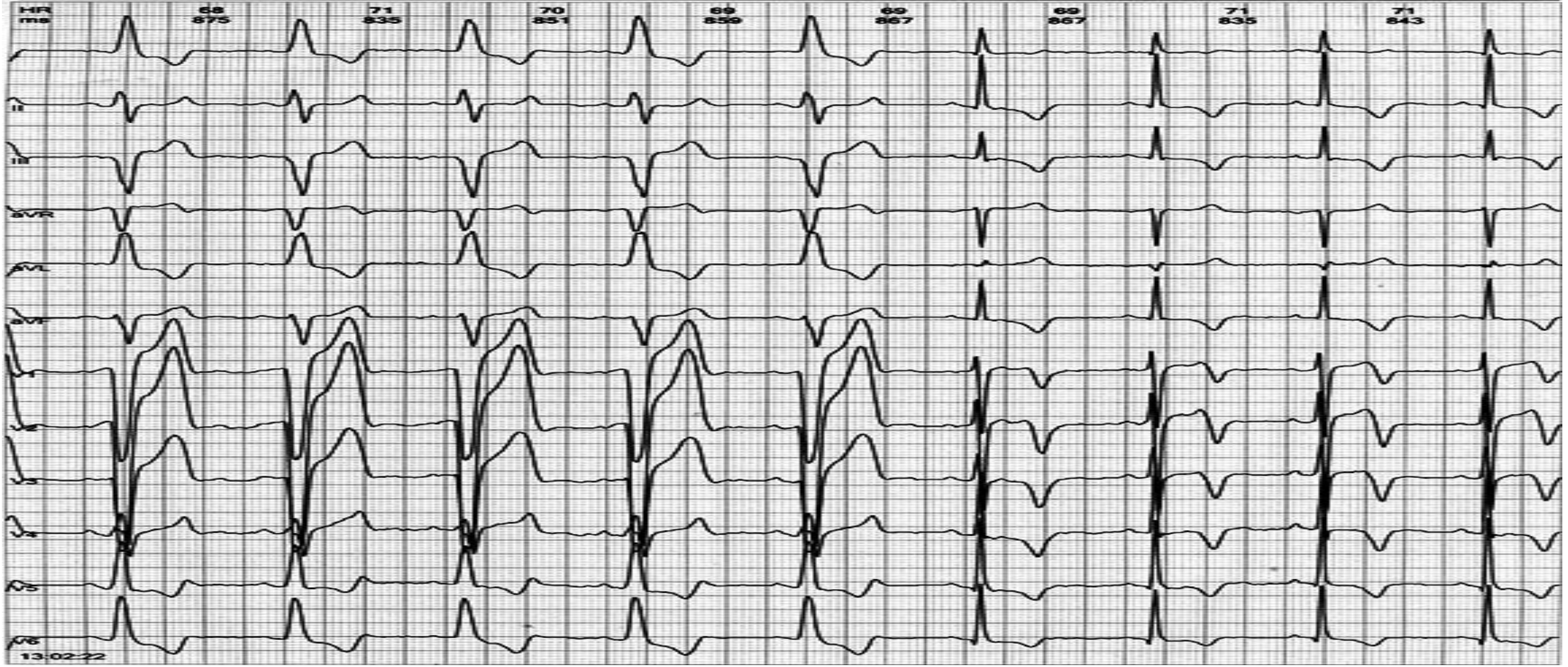
Final comments and conclusions



The ECG pattern of high-grade LAD stenosis has proven to be an important marker of high-risk patients with chest pain (Wellens syndrome, “acute coronary T-wave syndrome” or “LAD coronary T-wave syndrome”). False-positive ECG changes that mimic this pattern may also be seen in patients with intermittent LBBB. This ECG pattern called cardiac memory(CM) may mimic anterior ischemia due to LAD high-grade stenosis (**Kershaw 2014**) of so called Wellens syndrome characterized by symmetric and deeply inverted T waves or positive-negative biphasic T wave in leads V2 and V3, occasionally V1, V4, V5, and V6 plus Isoelectric or minimally elevated (< 1-mm) ST segment, no precordial Q waves, prolonged QT interval and history of chest pain in the last hours to days (Pattern present in pain-free state) and normal or slightly elevated cardiac serum markers.

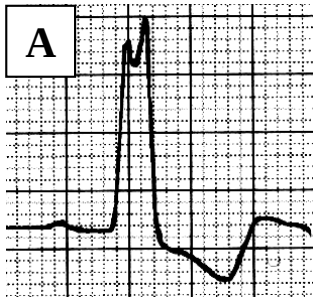
Abnormalities in the ST segment and T wave/loop, which are the result of changes in the shape and/or duration of the repolarization phases of the transmembrane action potential (AP) and occur in the absence of changes in depolarization, are referred to as primary repolarization abnormalities. They may be localized or diffuse and may be caused by ischemia, myocarditis, drugs, toxins, electrolyte disturbance (e.i. Ca^{2+} and K^{+} alterations), sudden change in heart rate, hyperventilation, changes in body position, sympathetic stimulation or ablation of the stellate ganglion, and temperature changes also can cause primary repolarization abnormalities (**Surawicz 2001-1972**). Abnormalities in the ST segment and T wave that occur as the direct result of changes in the sequence and/or duration of ventricular depolarization, manifested as changes in QRS shape and/or duration, are referred to as APs of individual cells. Rather, they may be due to voltage gradients that are normally largely canceled but become manifest when the changes in the sequence of depolarization alter the repolarization sequence. The ST- and T-wave changes that occur in association with bundle-branch blocks (BBBs), ventricular preexcitation (WPW), ectopic and paced ventricular complexes (Chatterjee phenomenon or "cardiac memory"). The classic ventricular gradient concept introduced by Wilson et al (**Wilson 1931**) in 1931 is of some theoretical interest concerning primary versus secondary repolarization abnormalities. Ventricular gradient in a single ECG lead is the net time integral of the ECG voltage from the beginning of the P wave to the end of the U wave. Its spatial counterpart is the ventricular gradient vector determined from the orthogonal XYZ leads. The practical utility of the ventricular gradient in differentiating primary from secondary repolarization abnormalities has not been demonstrated (**Surawicz 1988**). When the direction of the QRS axis is normal, an abnormal direction of the T-wave axis is generally an indication of primary repolarization abnormalities. Recognition of secondary repolarization abnormalities is usually not difficult. In LBBB, the ST- segment and T-wave vectors are generally directed opposite to the mean QRS vector. In RBBB, they are directed opposite to the slow terminal component of the QRS complex. In ventricular preexcitation, ST-T changes are directed opposite to the δ wave of the QRS complex. The magnitude of the ST-T change is dependent on the magnitude of the QRS-waveform changes when the excitation pathways change. The secondary ST- and T-wave changes associated with transiently altered ventricular conduction such as those that occur with ectopic ventricular complexes or transient BBBs usually revert promptly to the pattern that existed before the ventricular conduction changes developed. However, some secondary repolarization changes take longer (hours or days) to develop and to dissipate. The repolarization changes associated with prolonged ventricular pacing are examples of Chatterjee phenomenon (**Chatterjee 1969**). Primary and secondary repolarization abnormalities may occur concomitantly. Left ventricular hypertrophy is associated with changes in the shape and duration of the ventricular AP of isolated ventricular cells, particularly on the endocardium (**Nordin 1989**). These changes may contribute to ST- and T-wave changes and are independent of the changes that are secondary to QRS-amplitude changes and prolongation of the QRS complex. Both primary and secondary repolarization abnormalities should also be considered when T-wave polarity does not change as anticipated by the changes in the QRS complex. The distinction between primary and secondary repolarization abnormalities is clinically relevant because primary abnormalities indicate changes in the repolarization characteristics of ventricular myocytes whereas secondary changes do not.

Differential diagnosis of ST-segment depression or T-wave inversion

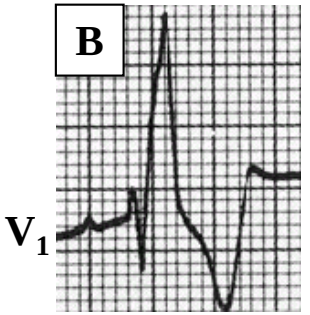
Secondary repolarization abnormalities or secondary ST-T wave changes: these are *normal* ST-T wave changes solely due to alterations in the sequence of ventricular activation or ST segment and T wave move in the same direction, discordant to QRS. The ST segment and T wave are directed opposite to the QRS: this is called discordance between the QRS complex and the ST-T abnormalities:

- ST-T changes seen in bundle branch blocks (BBBs): generally the ST-T polarity is opposite to the major or terminal deflection of the QRS. In the case of LBBB, left precordial leads show ST and T waves opposite to R wave (A). In the case of RBBB, the ST and T are directed opposite to the terminal portion of the QRS, ie, the part of the QRS deformed by the conduction abnormality (B).

LBBB



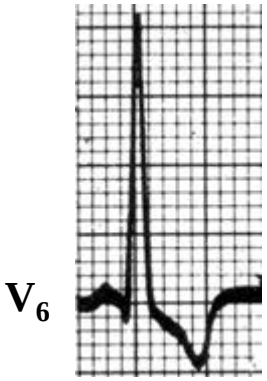
RBBB



- ST-T changes seen in cases of left ventricular hypertrophy (LVH) and right ventricular hypertrophy (RVH), since the QRS complex is upright in the left lateral leads I, aVL, V5, and V6, the ST segment is characteristically depressed and the T wave is inverted in these leads for LVH and vice-versa for RVH in right precordial leads.

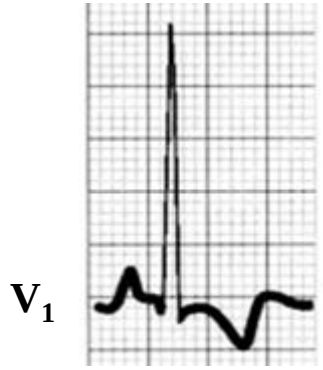
Observation: LVH and RVH are not always associated with ST-T abnormalities, but when these are present, they correlate with more severe hypertrophy or ventricular systolic dysfunction, and have been called strain pattern. In addition, while these morphologic features are consistent with secondary abnormalities, they do not rule out ischemia in a patient with angina

LVH



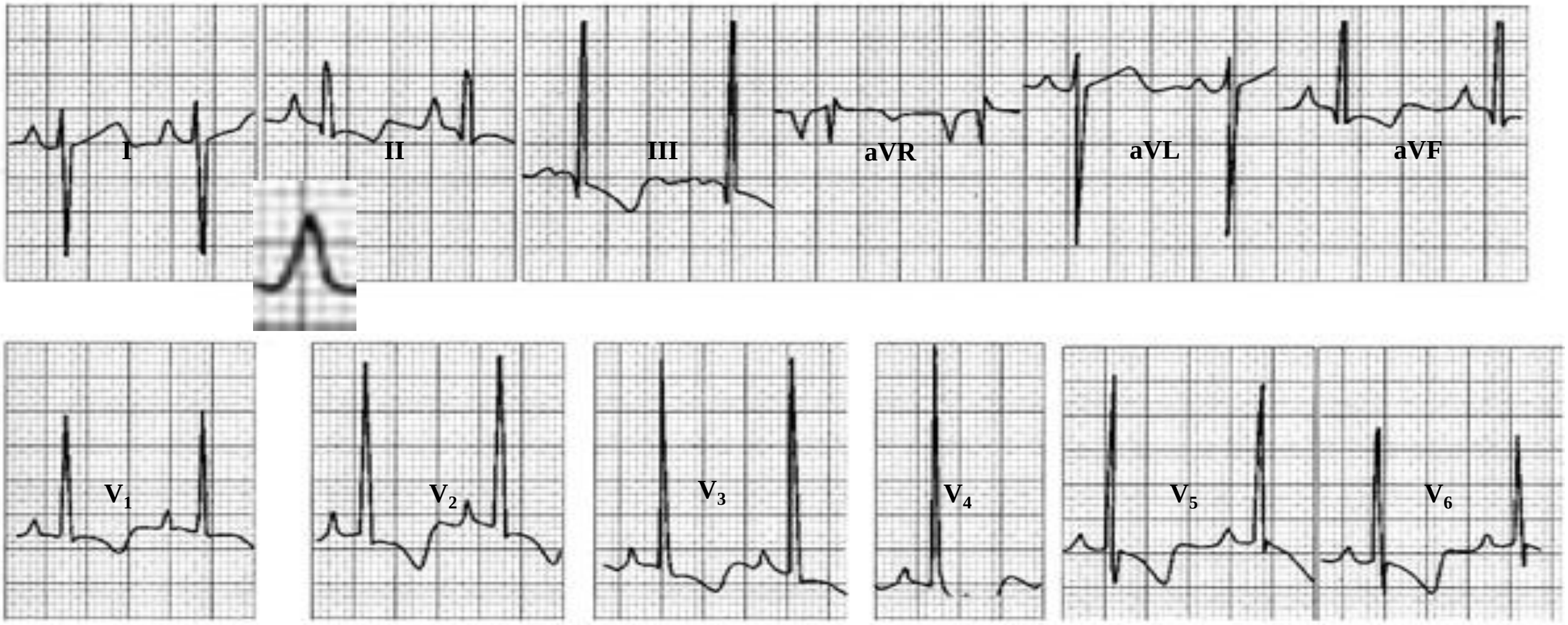
Strain pattern

RVH



Strain pattern

ST-T changes seen in cases of RVH or RBBB, T waves are characteristically inverted in the right precordial leads V1, V2, and V3.



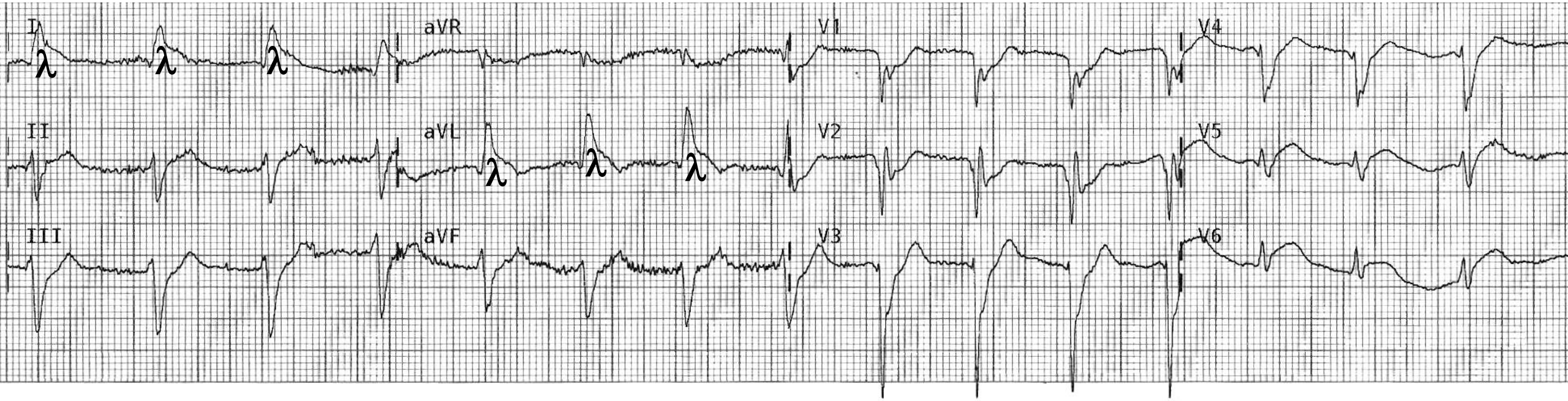
Clinical diagnosis: Severe Pulmonary Stenosis

ECG diagnosis: Right Atrial Enlargement: P voltage 4 mm in II and prominent P-waves on right precordial leads.

Severe systolic RVH pattern: QRS axis + 115°, rS pattern in I and aVL, pure R wave from V_1 through V_3 with negative ST segment and T-wave from V_1 to V_6.

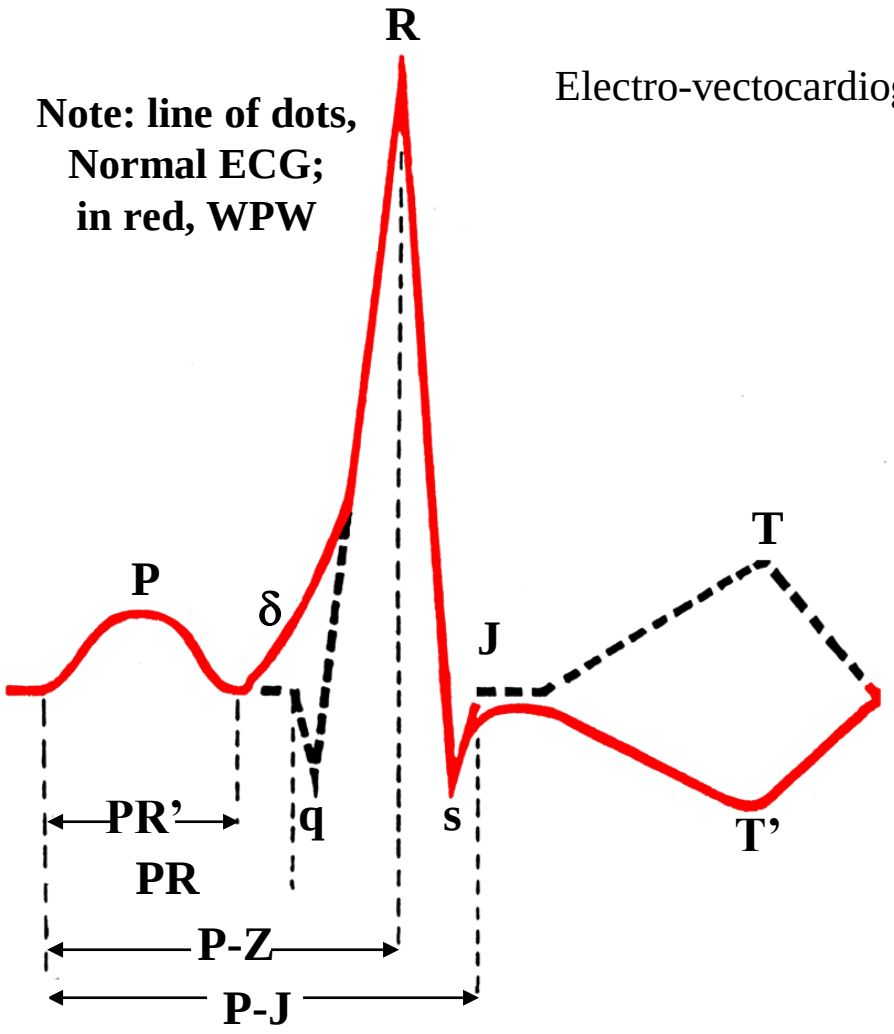
Marked clockwise rotation without transitional zone on precordial leads.

- ST-T changes seen in fascicular block
- ST-T changes seen in nonspecific intraventricular conduction delay (IVCD)

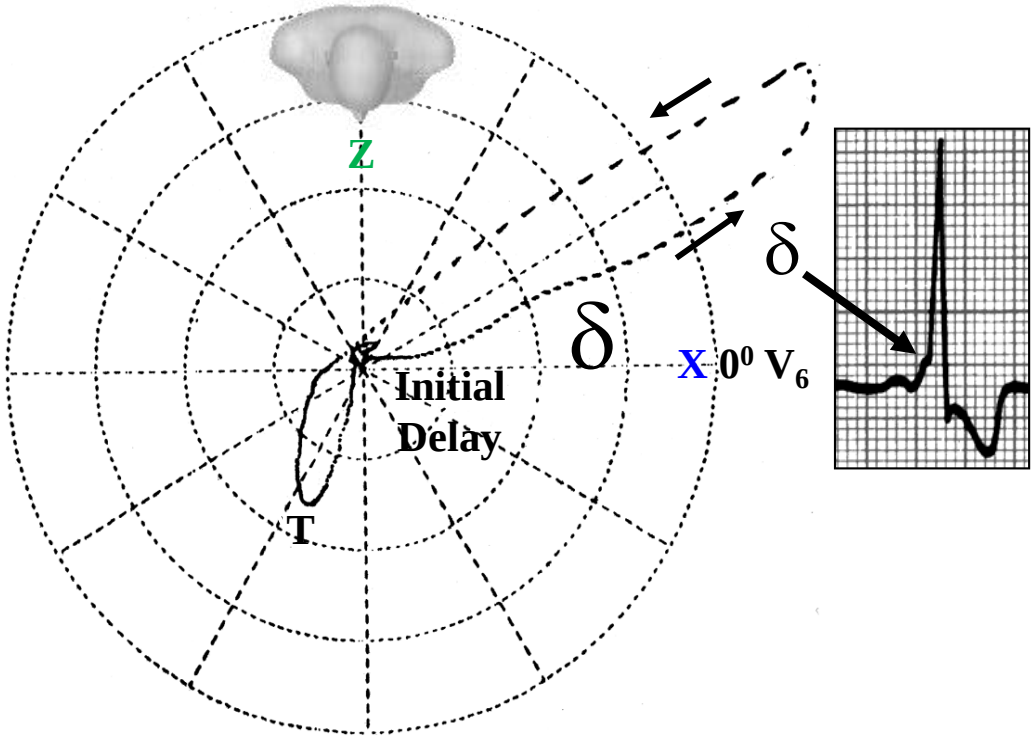


Regular rhythm with no clearly discernible P-waves, heart rate about 90 bpm, wide QRS complexes with atypical or bizarre morphology, especially in the right precordial leads. If this is a supraventricular? **Conclusion:** Non-specific intraventricular conduction defect or atypical LBBB? Regardless of what kind of conduction delay is present (even if it's an accelerated idioventricular rhythm) we know that the depolarization is abnormal. ST/T-wave discordance is present throughout the majority of the 12-lead ECG and has lambda wave pattern (λ) in I and aVL. This pattern is marker for sudden cardiac death in both atypical Brugada pattern and coronary artery disease scenario. Here we can see significant ST-elevation that is concordant with (in the same direction as) the majority of the QRS complex. It is also concordant with the terminal (last) wave of the QRS complex. leads I and aVL show pathological initial Q-waves. Inferior leads III and aVF show mirror image of I and aVL. Additionally, right precordial leads show ST segment depression (Sgarbossa's criteria). Finally, V5 and V6 show ST segment elevation (STSE). This patient had SCD without possibility of resuscitation.

- ST-T changes seen in WPW preexcitation



Electro-vectocardiographic criteria for WPW type preexcitation.

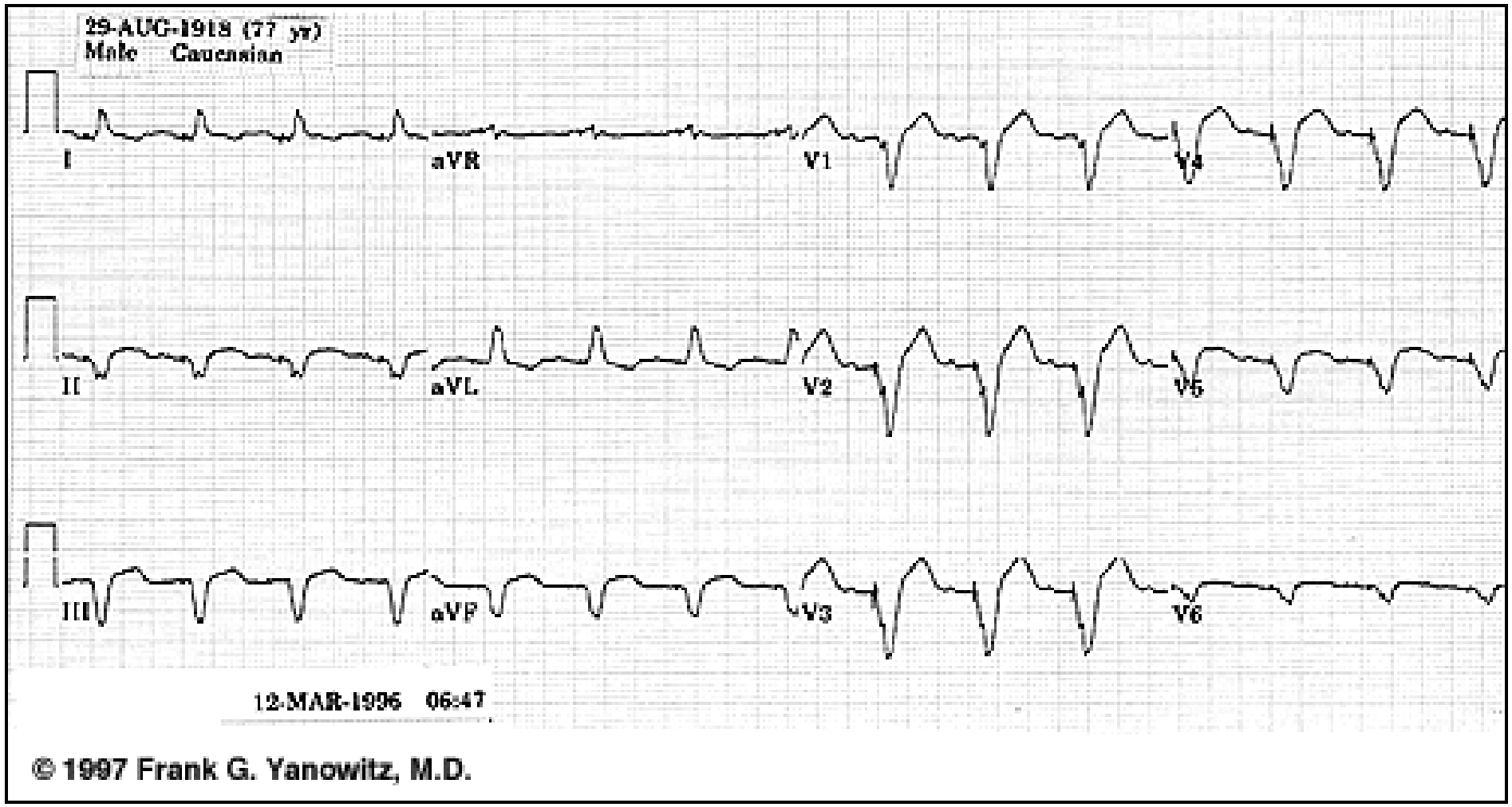


PRi or PQ: since the onset of P up to the onset of QRS. It represents the time the stimulus takes to go from the SA node until reaching the ventricles: 120 ms to 200 ms.

PZ: distance between P wave onset until R apex: 150 to 230 ms.

PJ: distance between P wave onset until j point: 180 to 260 ms.

- ST-T changes in PVCs, ventricular arrhythmias, and ventricular paced beats



Ventricular Pacemaker Rhythm-KH

Frank G.Yanowitz, M.D.

Note the small pacemaker spikes before the QRS complexes in many of the leads. In addition, the QRS complex in V1 exhibits ventricular ectopic morphology; i.e., there is a slur or notch at the beginning of the S wave, and >60ms delay from onset to QRS to nadir of S wave. This rules against a supraventricular rhythm with LBBB.

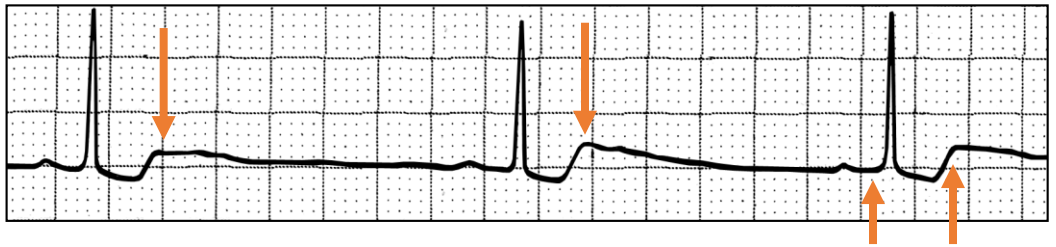
Primary ST-T wave abnormalities: the ST-T wave changes that are independent of changes in ventricular activation and that may be the result of global or segmental pathologic processes that affect ventricular repolarization:

Drug effects (e.g., digoxin, amiodarone, etc)

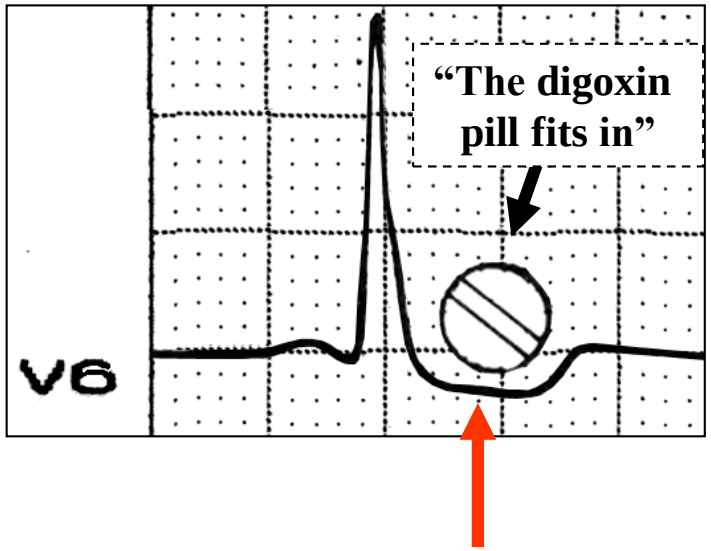
The earliest modification of digitalis effect on ECG or "digitalis action" are:

- Prolonged PR interval.
- ST segment: shortening and superior convexity ("in spoon") by shortening of phases 2 and 3 of action potential (AP);
- QT and QTc intervals shortening: main cause of acquired short QT;
- T wave flattening with apiculate form of terminal portion in 10% of cases. Possible symmetrical inversion of T wave (pseudo-ischemic T wave);
- Prominent U wave.

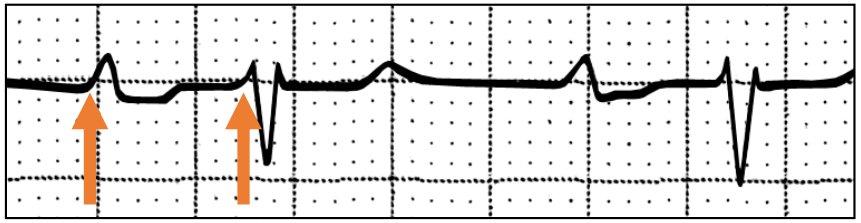
Apiculate form of terminal portion of T wave



QTc interval shortening

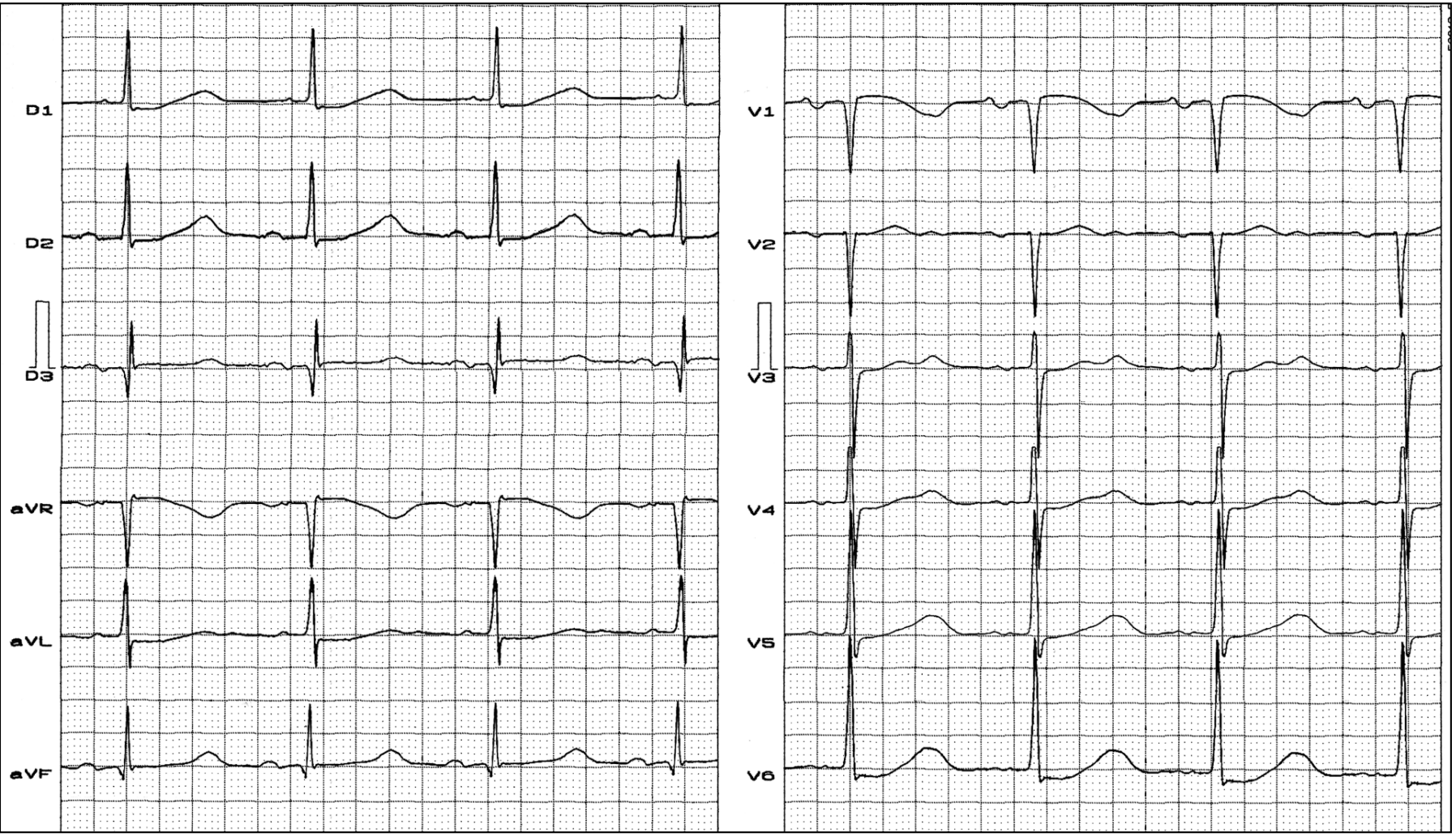


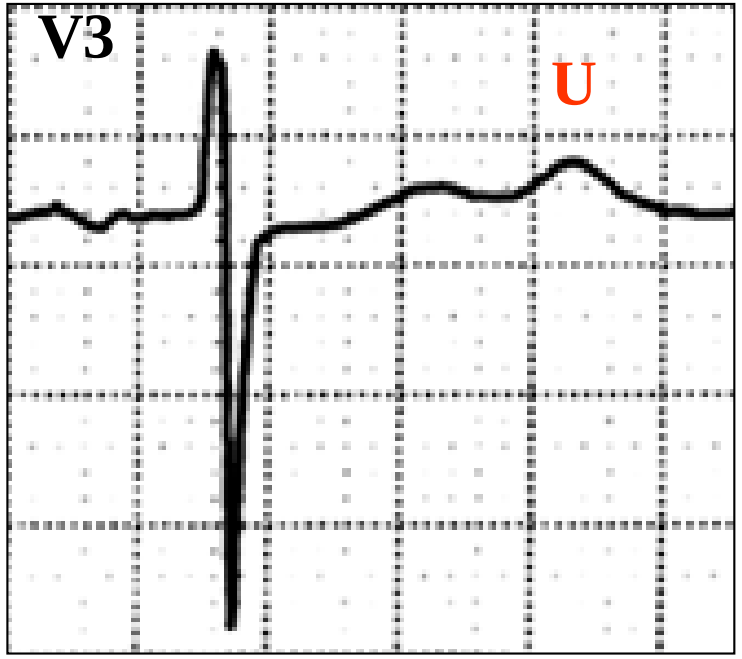
Short ST segment "in spoon"



Prolonged PR interval

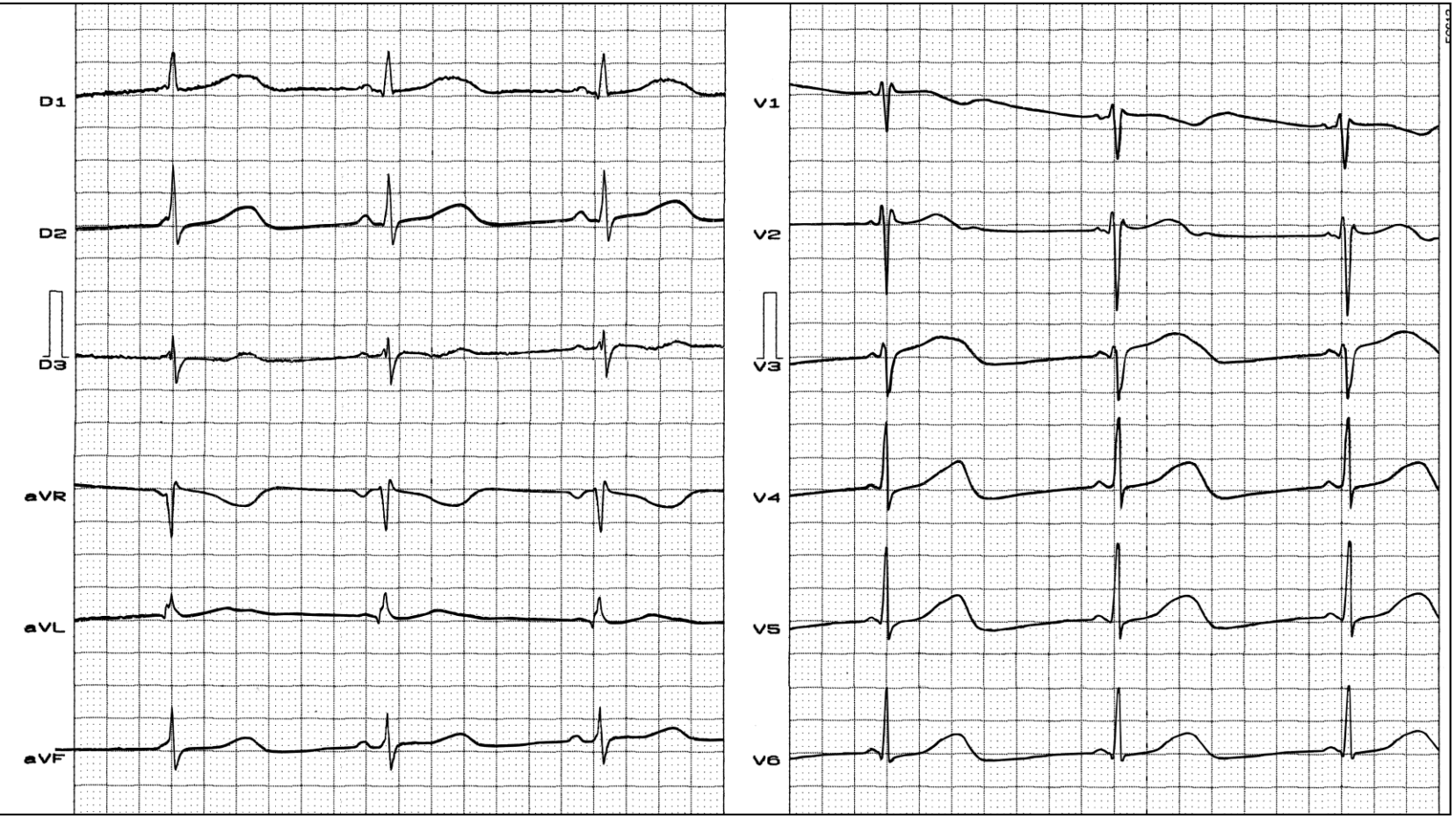
Secondary or iatrogenic forms of LQTS by amiodarone





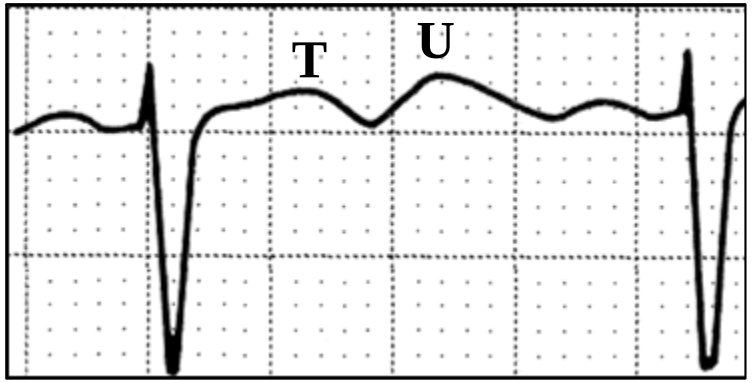
LAE; PR interval: 190 ms; QRS: 104 ms; long QT of 520 ms; QT + U: 716 ms; T and U waves hard to differentiate; notch between the end of T wave and the onset of U wave.

Electrolyte abnormalities: hypokalemia



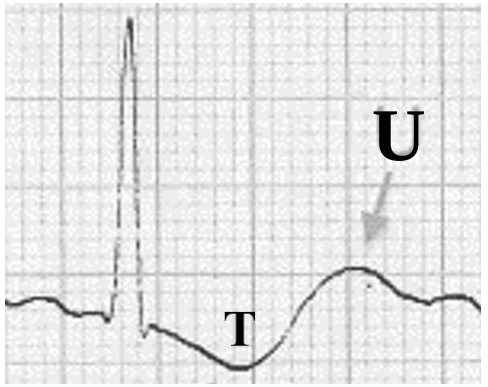
Clinical diagnosis: Hypopotasemia.
ECG diagnosis: isorhythmic AV dissociation, HR: 42 bpm, IRBBB, total fusion of T waves with U waves, prolonged QT (U) interval: 620 ms, QTc (U) interval: 521 ms.

T/U ratio ≤ 1 in II and/or V_3



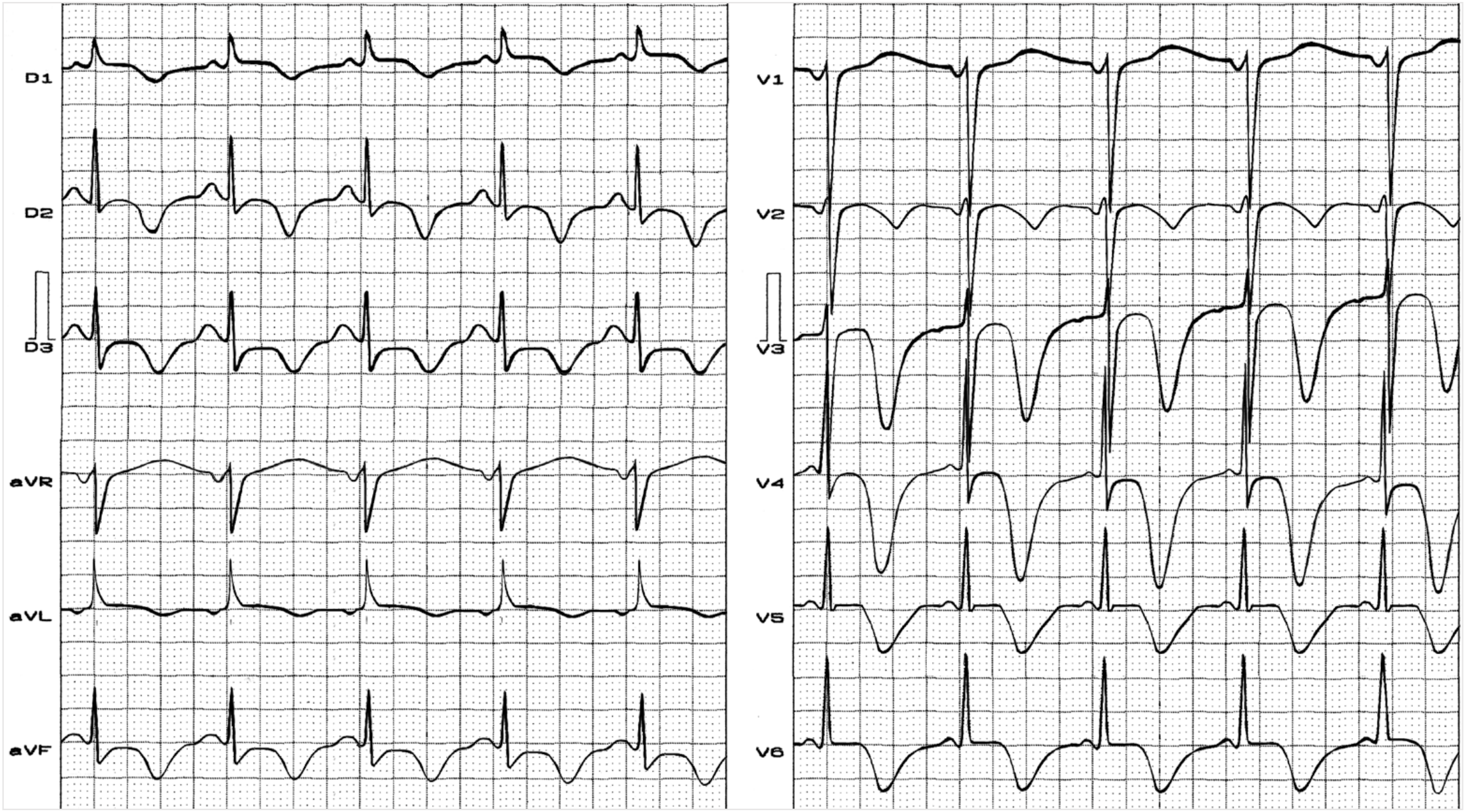
U wave > 0.5 mm in II or > 1 mm in V_3

- Gradual ST segment depression ≥ 0.5 mm in II or from V_1 to V_3.
- Decrease of T wave amplitude (flat T wave).
- Possible T wave inversion.
- Prominent U enhancement. wave.
- QTc interval prolongation.
- Digitalis action



ST depression, inverted T-wave, prominent U-wave, QT prolongation: hypokalaemia

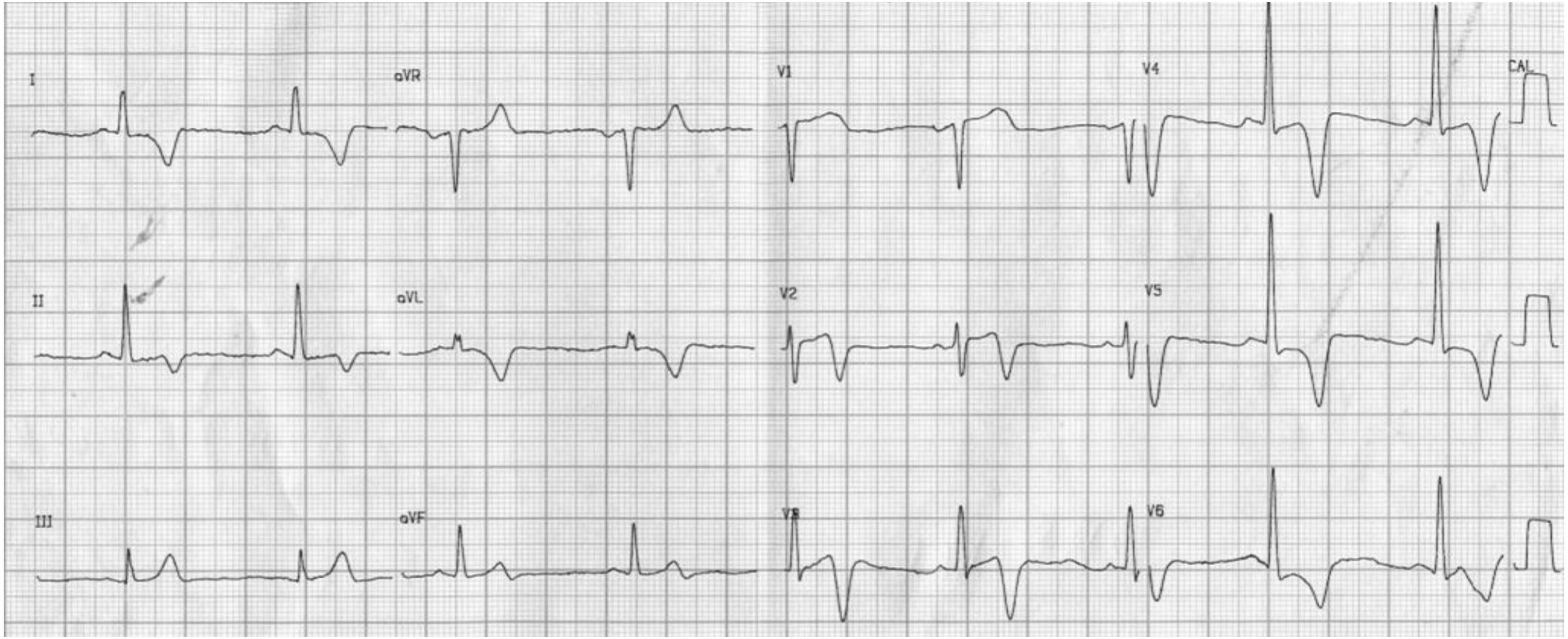
Neurogenic effects (e.g., subarrachnoid hemorrhage causing long QT)



Clinical diagnosis: Subarachnoid bleeding.

ECG diagnosis: long QT interval, largely wide and inverted T waves: “giant T waves”.

Apical hypertrophic cardiomyopathy (ApHCM)

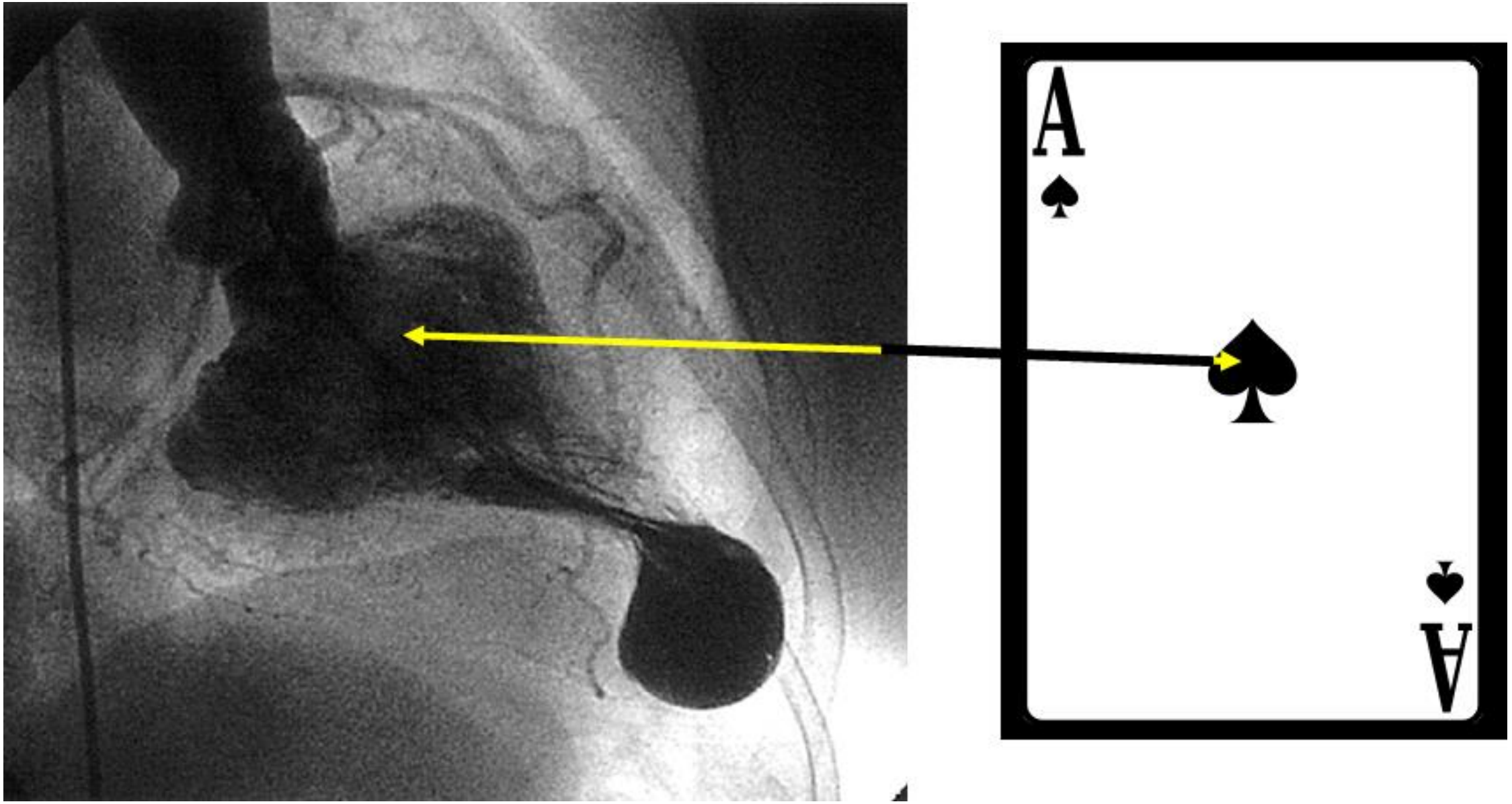


Giant negative T waves in the precordial ECG leads ≥ 1.0 mV (10 mm). The significant posterior and rightward shift of the ST/T vector is responsible for the characteristic giant negative T wave (>10 mm) in the leads of the horizontal plane from V2 to V6, and I, II and aVL. Three hypotheses aroused to explain these negative T waves: 1) apical subendocardial ischemia.; 2) apical cell disorder; 3) greater duration of action potential of hypertrophied cells, thus conditioning the area to have a slower repolarization.

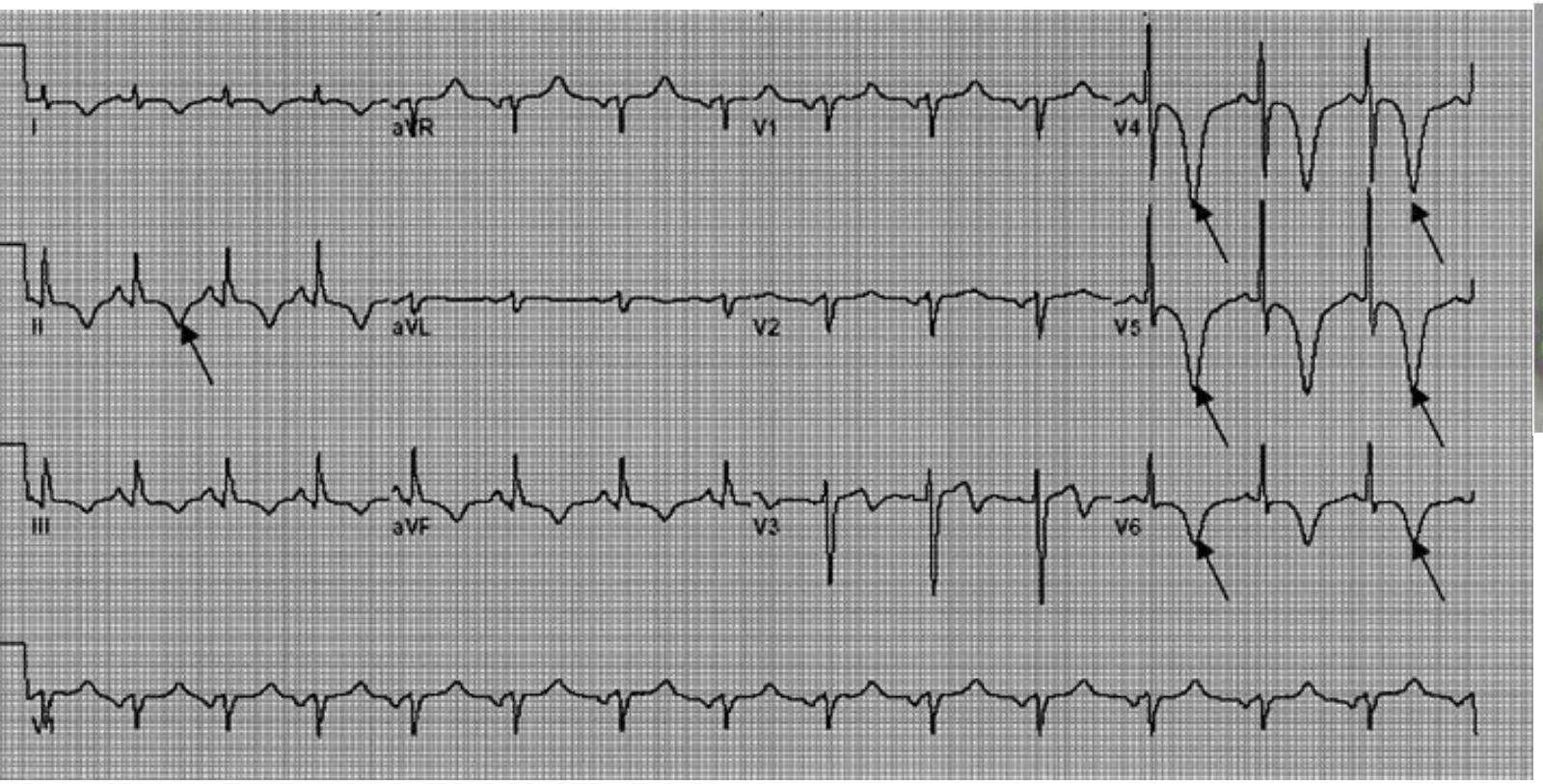
The prevalence in the western world of this form of HCM is approximately 0.02 to 0.2% and it constitutes 8% of the cases of the entity. In Japan, the apical form of HCM constitutes 25% of HCM.

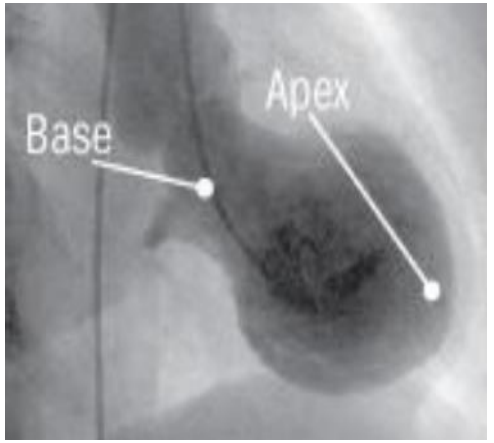
Left Ventriculography in ApHCM

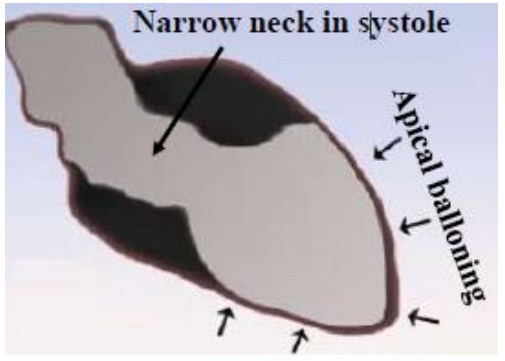
The "ace-of-spades" sign on left ventriculography being pathognomonic (**Olearczyk 2008**).



Takotsubo cardiomyopathy "octopus trap", transient apical ballooning syndrome, apical ballooning cardiomyopathy, stress-induced cardiomyopathy, Gebrochenes-Herz-Syndrome, broken heart, and simply stress cardiomyopathy a bulging out of the left ventricular apex with a hypercontractile base of the LV is often noted. It is the hallmark bulging out of the apex of the heart with preserved function of the base that earned. Summary of ECG criteria for diagnosis: **absence of ST segment elevation in V1, absence of reciprocal changes in inferior leads, presence of ST segment elevation in inferior leads, especially in II, sum of ST segment elevation in V4-6 ÷ V1-3 ≥1, ST segment depression in aVR, deep negative T waves associated with prolonged QTc.**







ECG taken 42 hours from admission (third stage), revealing dramatic, deep T wave inversions (black arrows) and QT prolongation typically resolves after 3-4 months, but in some cases these changes may last up to 1 year. Resolution of changes may sometimes occur earlier after 3-4 weeks. The name Takotsubo comes from the shape of left ventricle seen on ventriculography showing transient apical and/or mid-ventricular ballooning with compensating hyperkinesis in the LV basal segment (narrow neck), resembling a Japanese takotsubo. This is a ceramic pot or trap pot (tako: octopus and tsubo: clay pot) with a rounded aspect and narrow base.

During systole the midsection and tip (apex) of the left ventricle balloon out, while the area above called the base, contracts normally. The shape is similar to that of Takotsubo cardiomyopathy a round-bottomed, narrow-necked vessel used to catch octopuses.

The criteria proposed by the Mayo Clinic for the diagnosis of TCM and the Japanese guidelines for the diagnosis of this entity include the following:

- Transient hypokinesis, dyskinesis or akinesis of the LV middle segment with or without apical involvement and usually triggered by physical or emotional stress.
- Segmental alterations of contractility in absence of significant epicardial coronary artery obstruction.
- Absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture.
- New electrocardiographic changes of transient ST segment elevation and/or diffuse T wave inversion with slight elevation of troponins.
- Absence of proven pheochromocytoma or myocarditis.

The electrocardiogram in TCM is characterized by circumferential subepicardial ischemia. These ECG changes are significantly different from those that occur in acute segmental transmural ischemia characteristic of ST segment elevation MI (STEMI). Although some segmental contractile alterations (apical dyskinesis and basal hyperkinesis) occur in TCM, ST segment elevation is more diffuse in comparison to STEMI. This paradox can be explained by considering the electrophysiological and molecular alterations.

The electrocardiographic pattern of TCM has 3 successive stages or phases:

I. **First stage:** characterized by discrete ST segment elevation, usually in the precordial leads but also sometimes in the lateral and inferior leads. The magnitude of ST elevation is usually less than ST segment elevation in STEMI. T waves are tall but do not exceed 12-15 mm as is sometimes seen in STEMI where they may exceed 18 mm. The maximal ST segment alteration usually occurs in leads V3-5.

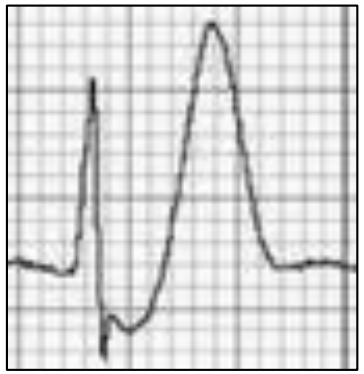
II. **Second stage**: seen after 2-3 days; ST segment elevation resolves with appearance of diffuse, deep and inverted T waves except in lead aVR where T waves are positive. The presence of positive T waves in aVR is a valuable sign in differentiating TCM from MI. The non-segmental distribution of T wave alterations is a characteristic of this syndrome. The QT and QTc intervals may also be prolonged. Pathological Q waves are rarely seen.

III. **Third stage:** T wave inversion and QT prolongation typically resolves after 3-4 months, but in some cases these changes may last up to 1 year. Resolution of changes may sometimes occur earlier after 3-4 weeks.

ST-segment depression or T-wave inversion is consistent with ischemia if any of the following is true:

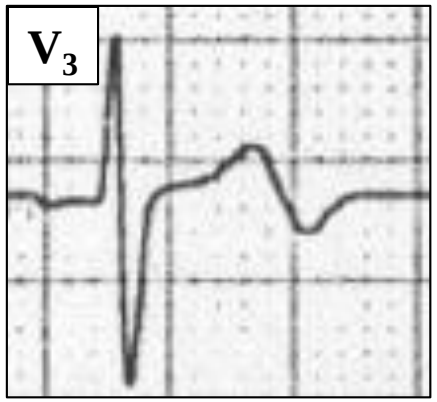
- The ST segment is depressed concave upwards followed by peaked prominent symmetric anterior T wave is upright(V2-V6). So-called hyperacute T waves with the ascending limb of the T wave commencing below the isoelectric line Additionally, sometimes sublte ST segment elevation (≥0.5mm-1mm) in aVR. Lead aVR shows slight ST-segment elevation in most cases = It is the so-called the De Winter ECG pattern. This is a sign of acute LAD occlusion and should be treated as a STEMI equivalent. The de Winter pattern is seen in ≈ 2% of acute LAD occlusions and is under-recognized by clinicians. There is also some high lateral involvement, with subtle ST elevation in aVL plus reciprocal change in III + aVF. This is consistent with LAD occlusion occurring proximal to the first diagonal.

V2-V6

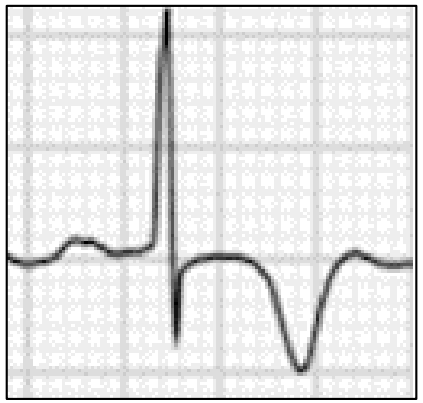


De Winter's T Waves - a STEMI equivalent

- The T wave has a positive-negative biphasic pattern or deep negative T wave.



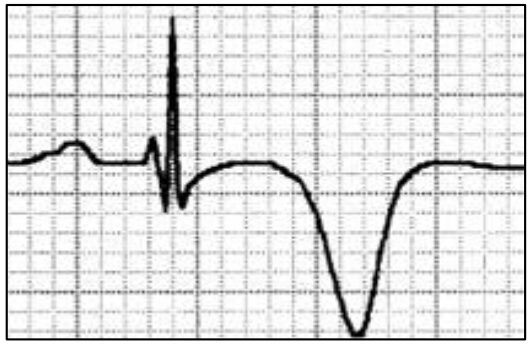
Biphasic T waves due to ischemia, plus-minus T wave or **Type A or 2 Wellens syndrome**



Wellens' Type B or 1 syndrome

Wellens syndrome Symmetric and deeply inverted T waves or Positive-negative biphasic T wave in leads V2 and V3, occasionally V1, V4, V5, and V6 PLUS Isoelectric or minimally elevated (< 1-mm) ST segment No precordial Q waves Prolonged QT interval History of chest pain in the last hours to days Pattern present in pain-free state Normal or slightly elevated cardiac serum markers.

- The ST-segment depression or T-wave inversion is directed in the same direction as the QRS complex: this is called concordance between the QRS complex and the ST or T abnormality.
- The T wave is symmetrically inverted and has a pointed configuration, while the ST segment is not deviated or is upwardly bowed (coved) or horizontally depressed.



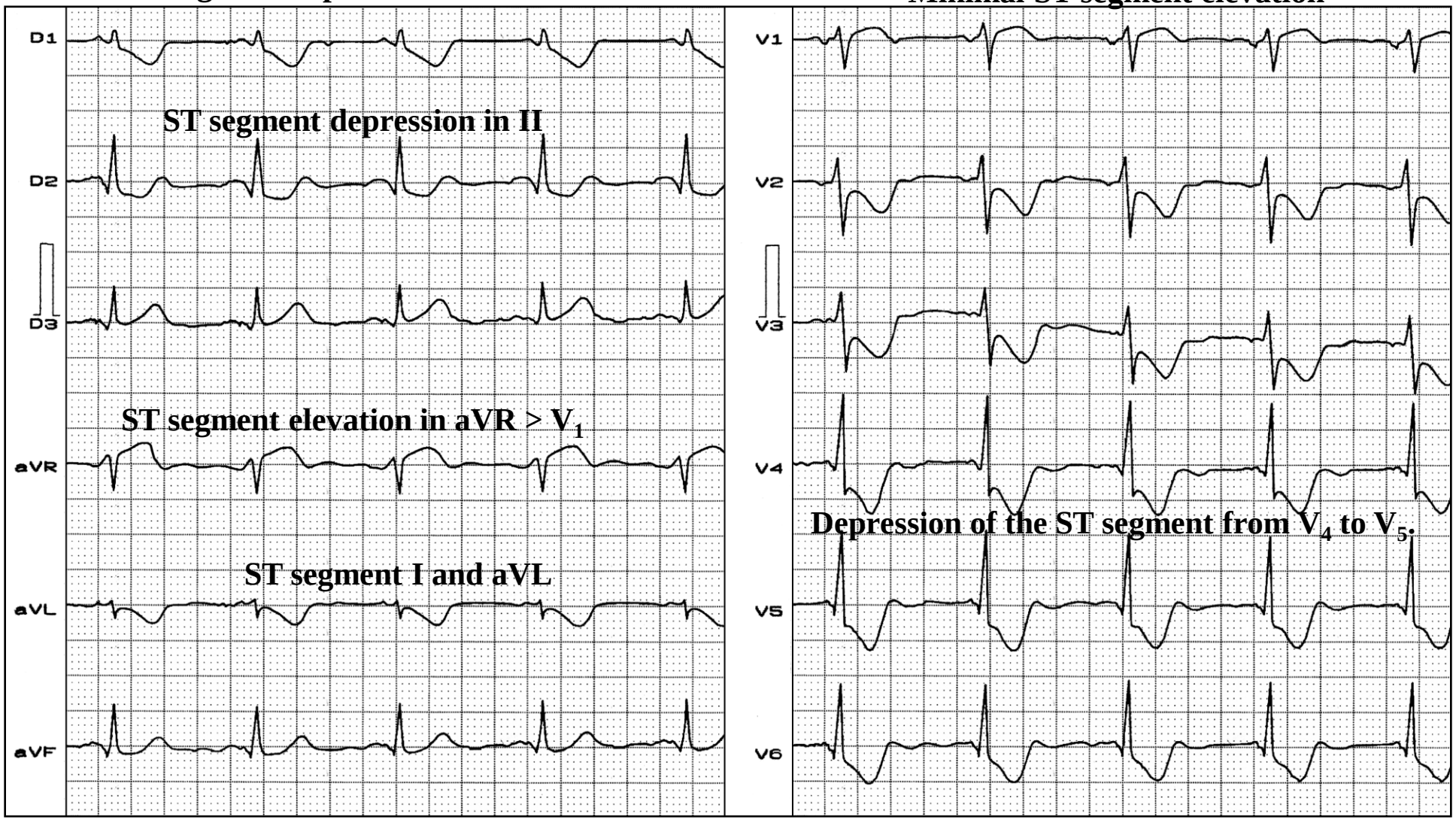
Ischemic negative, wide base, symmetrical, acute nadir T wave in “seagull wings”.

- The magnitude of ST-segment depression progresses or regresses on serial tracings, or ST-segment depression progresses to T-wave abnormality during ischemia-free intervals (dynamic ST-segment depression).
- Unlike ST-segment elevation, ST-segment depression does not localize ischemia. However, the extent and the magnitude of ST-segment depression correlate with the extent and the severity of ischemia.

- ST-segment depression in eight or more leads, combined with ST-segment elevation in leads aVR and V1 and occurring during ischemic pain, is associated with a 75% predictive accuracy for LMCA or three-vessel disease. This finding may also be seen in cases of proximal stenosis of the LAD.

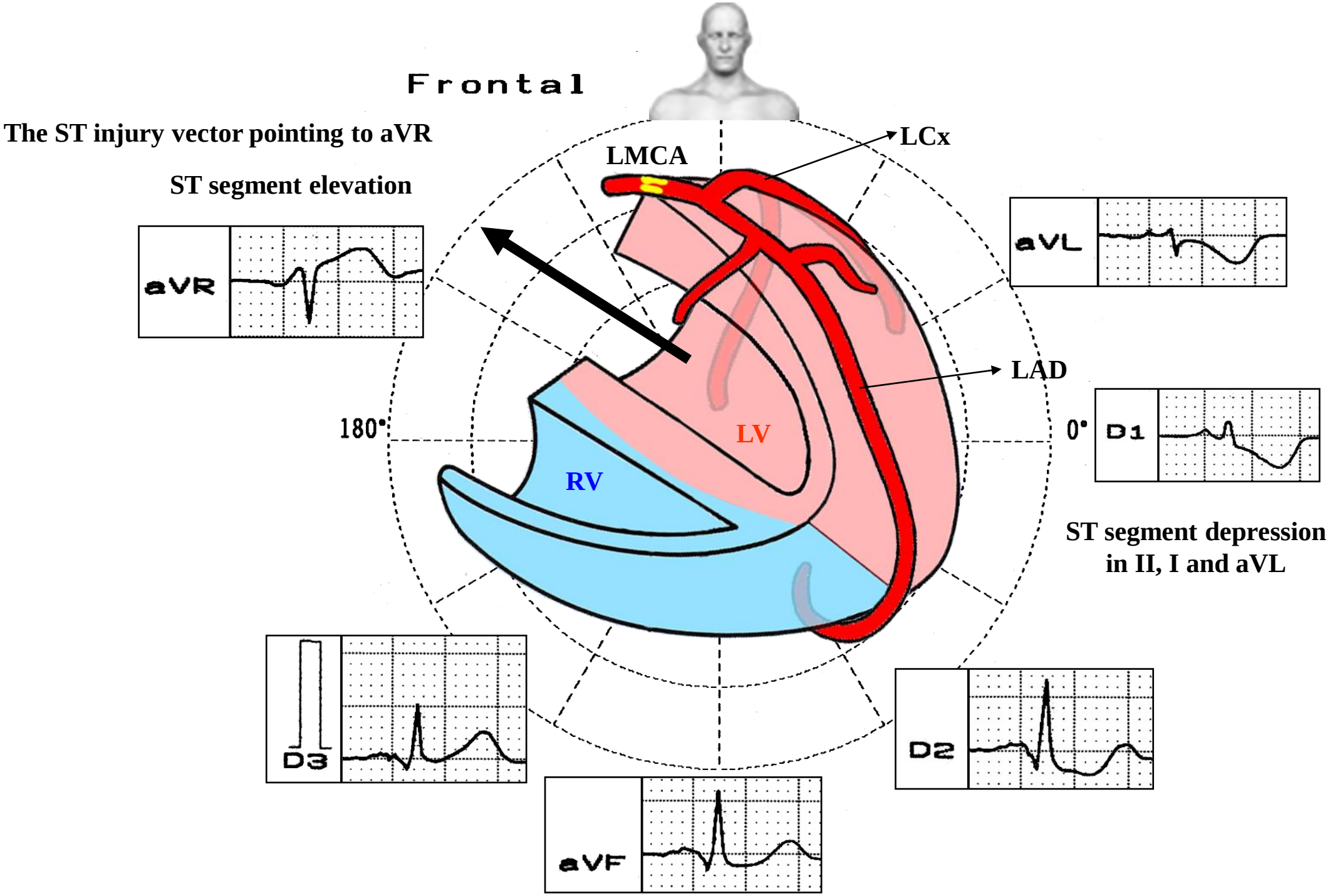
Diffuse ST segment depression in the inferolateral leads

Minimal ST segment elevation

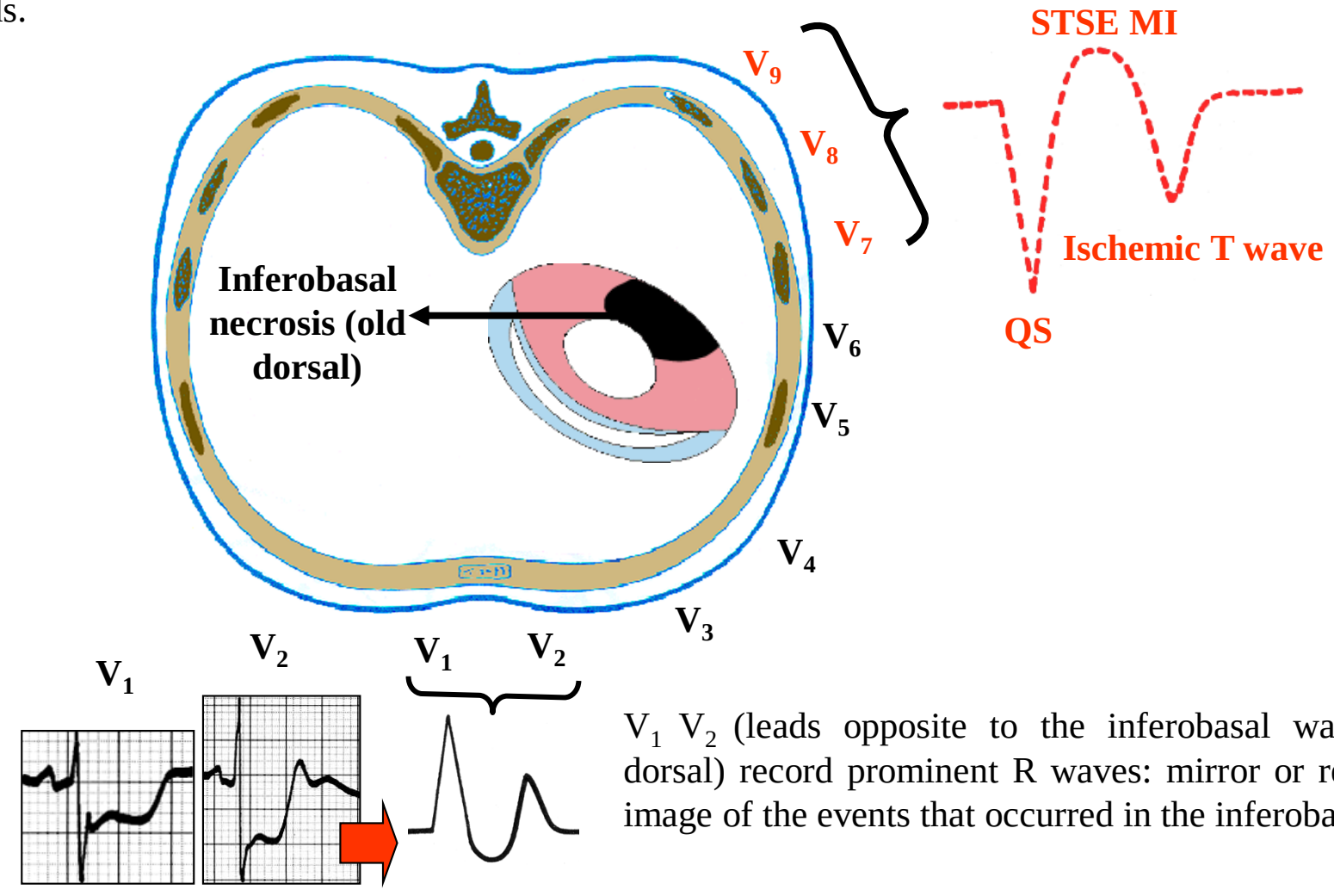


Why this pattern is observed?

ST segment depression in V_6 > ST segment elevation in V_1.


Frontal
The ST injury vector pointing to aVR
ST segment elevation
LMCA
LCx
LAD
LV
RV
180°
0°
aVR
aVL
D1
D2
D3
aVF
ST segment depression
in II, I and aVL

Inferobasal STSEMI (old true posterior STEMI): Maximal ST-segment depression in V1–V3 ST-segment elevation in V7–V9 ST-segment depression reciprocal to subtle ST-segment elevation Subtle ST-segment elevation concomitant to a more marked ST-segment depression in the reciprocal leads.



V_1 V_2 (leads opposite to the inferobasal wall – old dorsal) record prominent R waves: mirror or reciprocal image of the events that occurred in the inferobasal wall.

The terms posterior or dorsal(for V7 to V9) and high lateral MI (for I and aVL) are incorrect and should be changed to lateral wall MI and inferobasal respectively.

Myocardial Infarcts with Q wave based on the standard of Cardiac Magnetic Resonance Imaging

A Statement for Healthcare Professionals From a Committee Appointed by the International Society for Holter and Noninvasive Electrocardiography (**Bayés de Luna A 2006**)

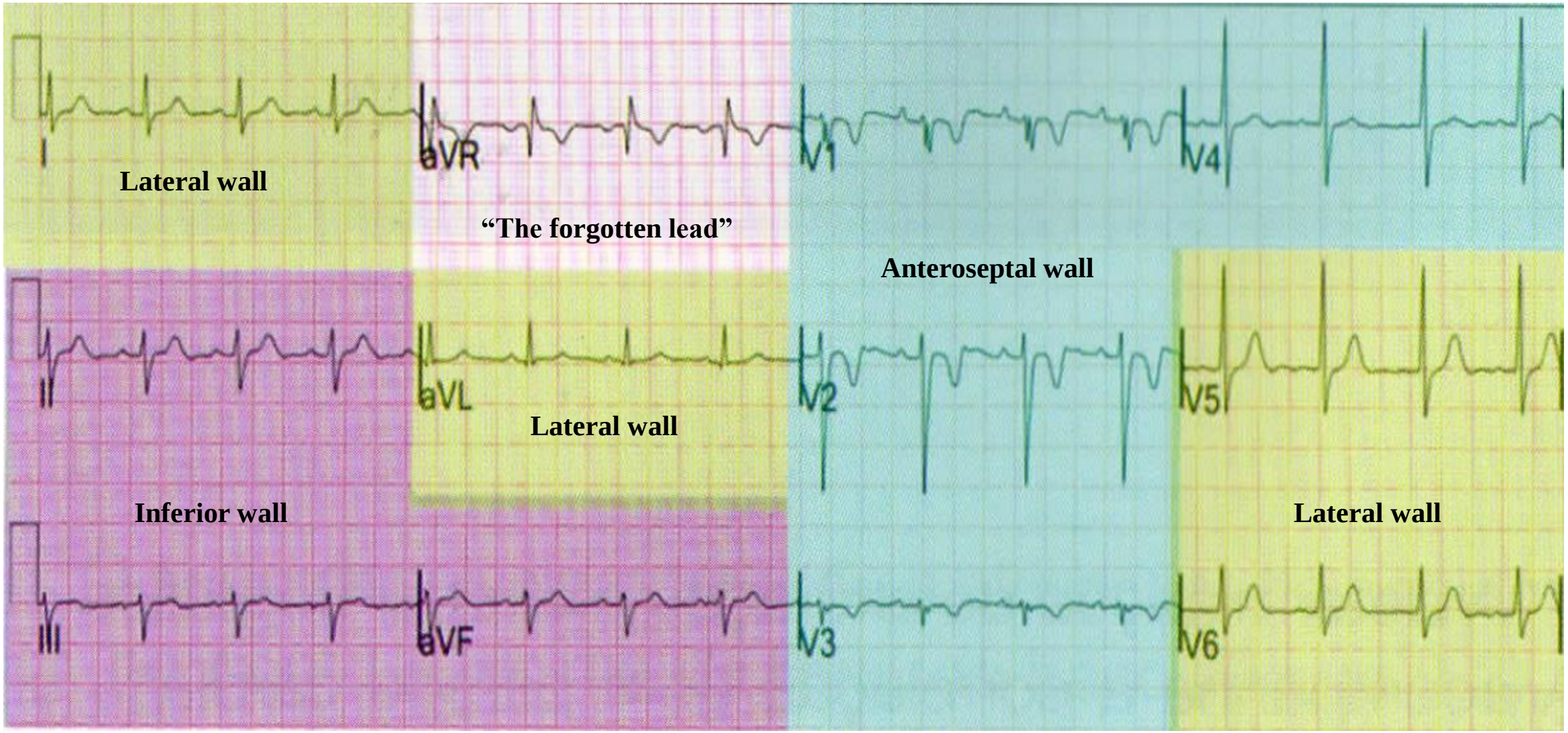
I. Anterior Myocardial infarctions (MI)

- **Septal MI:** Q waves in leads V_1 and V_2. The CMR with involvement of the septal wall and often a small part of the adjacent anterior wall. It is caused by occlusion of septal branches or LAD distal to origins of the diagonal branches.
- **Mid-Anterior MI:** abnormal Q waves in leads aVL and sometimes I but not in leads V_5 and V_6. A Q wave in leads V_2 and V_3 may be present. CMR shows that the infarction encompasses especially the mid-low segments (7 and 13) of the anterior wall. It is usually caused by occlusion of the first diagonal branch of the LAD.
- **Apical-Anterior MI:** Compared with septal infarction, the abnormal Q waves extend into the more leftward precordial leads: typically V_3 and V_4 and sometimes V_5 and V_6. Absence of abnormal Q waves in leads aVL and I. The CMR documents MI in the LV apex, often with extension into both the anterior and septal walls but not into the lateral wall. The infarct is caused usually by mid-LAD occlusion.
- **Extensive Anterior MI:** Abnormal Q waves from V1 to V6, aVL and sometimes I. The CMR documents that the infarct extensively involves the anterior, septal, and mid-low lateral walls. It is caused by occlusion of the LAD proximal to both the first septal and diagonal branches.

II. Lateral MI: may produce the Q-wave equivalents of abnormally prominent R waves in leads V_1 and V_2. There may also be abnormal Q waves in lead I, aVL, and/or V_5 and V_6. The CMR documents infarction in the lateral walls. It is caused by occlusion of a non dominant LCX or of its marginal branch.

III. Inferior MI: Q waves in leads II, III, and VF but without increased R waves in leads V_1 and V_2. The CMR shows involvement of the inferior wall, frequently including the basal segment. There may be involvement of the inferior part of the septal wall because the posterior descending artery has “perforating” branches that supply part of the inferior portion of the septum. The infarct is caused by occlusion of the dominant coronary artery that supplies the LPD. This is the RCA in ≈90% and the LCX in ≈10%. When the RCA or LCX is very dominant and the occlusion is proximal, the infarction encompasses both the inferior and the lateral wall, and then the ECG pattern is the association of criteria of inferior and lateral MI (inferolateral MI).

Current clinical electrocardiographic walls



As we see, the dorsal or posterior wall and high lateral wall do not exist. The presence of prominent the R wave in V1 is due to the lateral MI and not to the involvement of inferobasal segment of inferior wall (old posterior wall) (**Goldwasser 2015**). We must abandon this wrong and old nomenclature.

In the present case clearly during the conduction disturbance T waves/loops have a secondary pattern, because the efferent and afferent limbs show different speeds: slow in starting and faster in the final portion. The intermittence of the intraventricular conduction disturbance occurs virtually without changes in the heart rate (minimum increase in HR) Although the quality of the QRS loop not be good, we do not think there is **middle-end delay in the QRS loop** that should exist in the case of the existence of a genuine complete left bundle branch block (LBBB). On the other hand, the so-called " stricter criteria Strauss ECG criteria for complete LBBB " in the present case are absent because there are not mid-QRS notching or slurring(in the apex of the waves R.) in at least 2 contiguous leads. Straus et al review the pathophysiologic and clinical evidence supporting why only patients with complete LBBB benefit from CRT. This author review how the threshold of 120 ms to define LBBB was derived subjectively at a time when criteria for LBBB and right bundle branch block were mistakenly reversed. Three key studies over the past 65 years have suggested that 1/3 of patients diagnosed with LBBB by conventional electrocardiographic criteria may not have true complete LBBB, but likely have a combination of left ventricular hypertrophy and left anterior fascicular block. This author and colleagues propose stricter criteria for complete LBBB that include a QRS duration ≥ 140 ms for men and ≥ 130 ms for women, along with mid-QRS notching or slurring in ≥ 2 contiguous leads. When disappear the conduction disturbance we observe the so called Chatterjee phenomenon or cardiac memory in all leads because it records in the anterolateral wall (V1 to V6 I and aVL) as well as the diaphragmatic wall (II, III and aVF). Additionally, we know that intermittent LBBB is always a second degree LBBB never complete LBBB.

Classification of LBBB according to steadiness:

a) Permanent or definite: most of them.

b) Intermittent or of second degree that could be:

- **Rate-dependent intermittent LBBB**

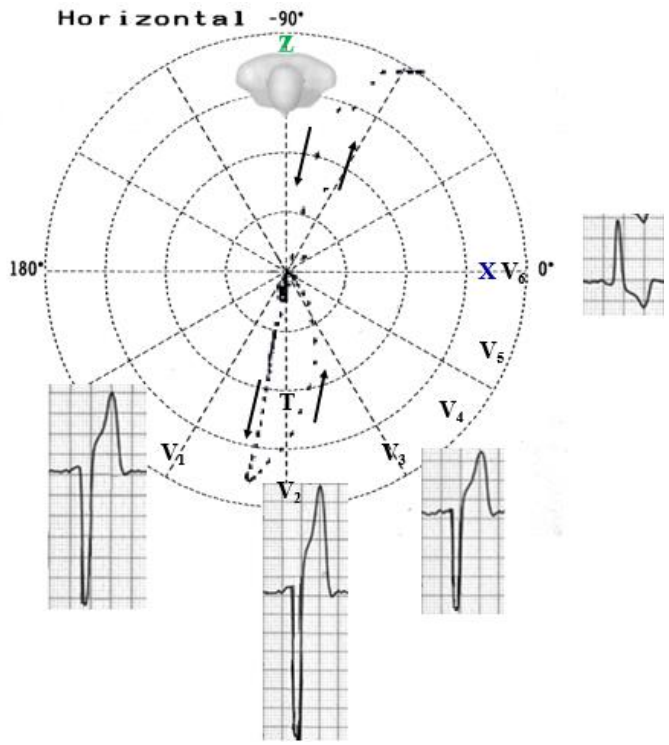
  - Tachycardia-dependent or in “phase 3”;
  - Bradycardia-dependent or in “phase 4”.

- **Independent from heart rate:**

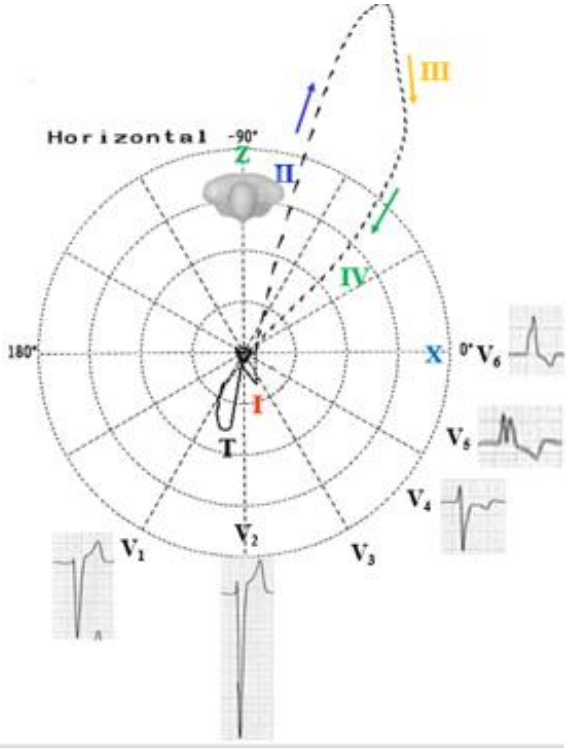
  - Mobitz type I;
  - Mobitz type II by Wenckebach phenomenon;
  - By significant hypopolarization.

ECG/VCG correlation in the Horizontal Plane

The present case



Genuine LBBB pattern



	The present case	Genuine LBBB pattern
QRS-loop rotation	CCW rotation may indicate parietal CLBBB, complicated with lateral infarction, severe LVH or nonspecific intraventricular conduction delay.	Main portions of QRS loop has clockwise(CW) rotation.
QRS-loop speed	Absence of conduction delay	**Middle-end delay** (III and IV) It is the hallmark of truly LBBB
Magnitude of the max QRS vector	Normal.	The magnitude of the max QRS vector is increased above normal (> 2mV).

Cardiac memory concept

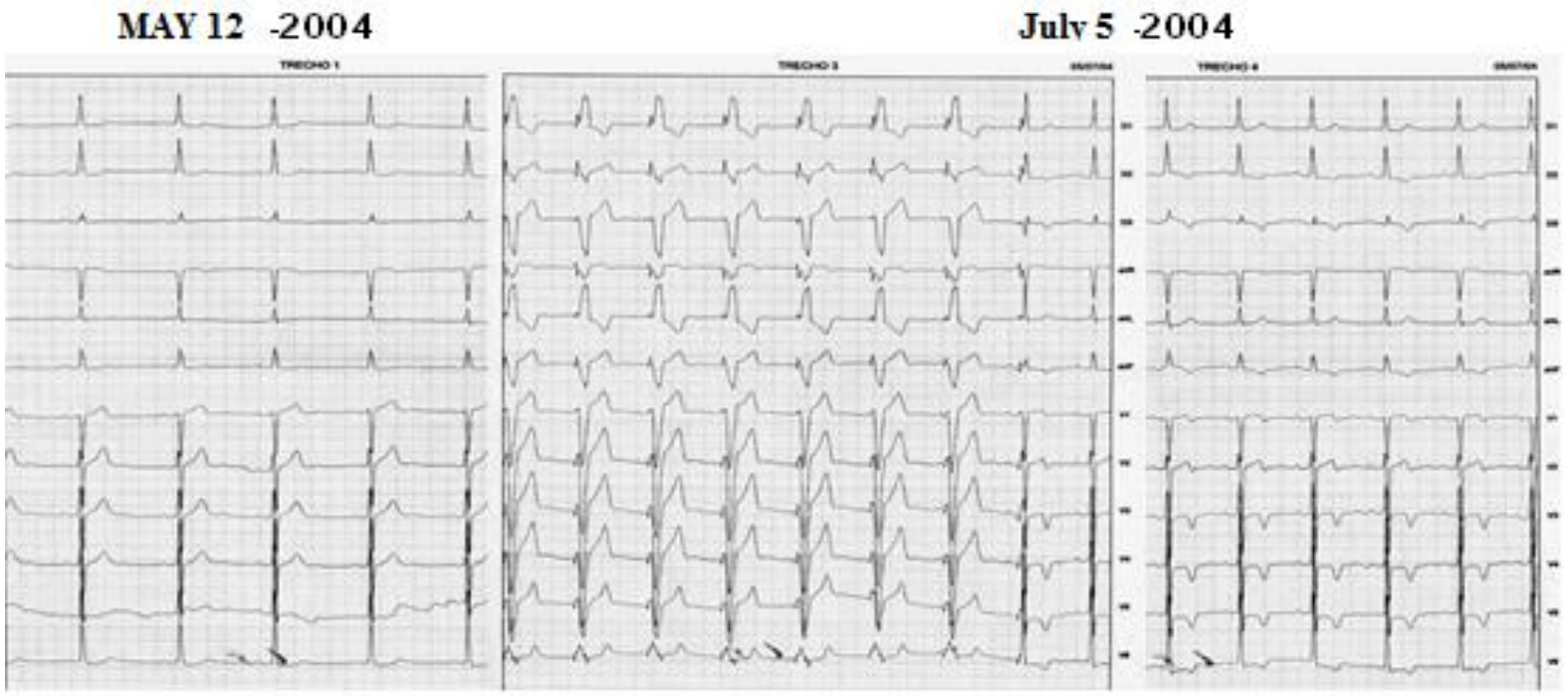
"Cardiac memory"(CM) is a peculiar variety of cardiac remodeling **(Chaile 2014)** observed after abnormal sequence of ventricular activation/depolarization (**Rosenbaum 1982-1990; Geiger 1943; Katz 1992; Chaile 2014)** manifested by a persistent (for minutes, hours, weeks or months) but reversible T-wave changes on the surface ECG.

I. **ECG changes (cardiac memory) subsequent of post pacing or artificial ventricular depolarization**
II. **Cardiac memory after intermittent left bundle branch block (Rosenbaum 1973-1982) "pseudoprimary T-wave changes"**
III. **Cardiac memory after episodes of tachycardia post-paroxysmal wide tachycardia syndrome (Kernohan 1969).**
IV. **Cardiac memory after unespecific intraventricular conduction defects (NSIVCD)**
V. **Cardiac memory after intermittent preexcitation Wolff-Parkinson –White type (Kalbfleisch1991): 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).**

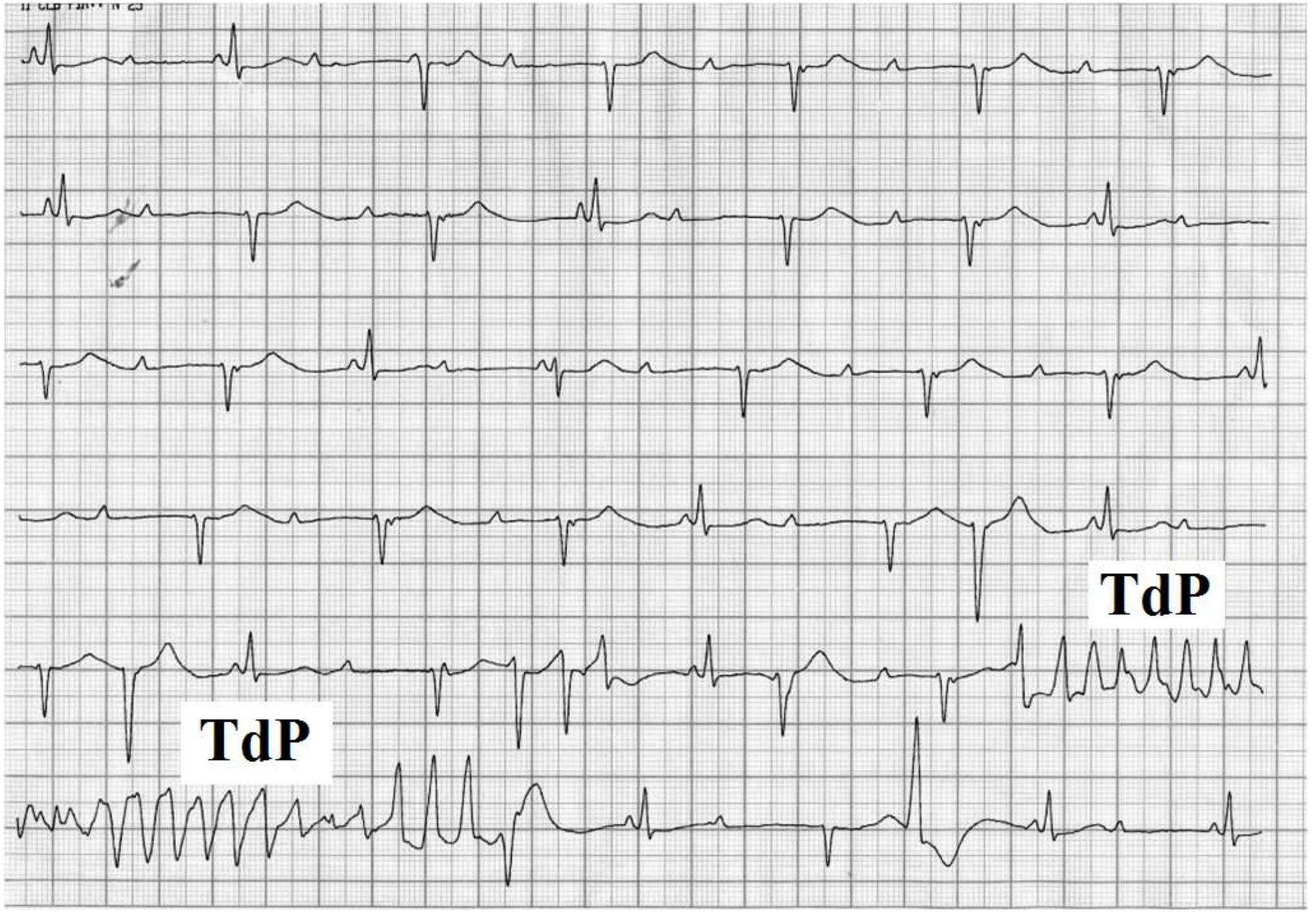
The great interest for investigating this topic is due to the impact that recognizing this phenomenon has when making decisions in cardiological clinical practice, since it manifests with T wave alterations generally interpreted mistakenly as of ischemic origin (pseudo-primary T waves) observed in multiple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

I. ECG changes (cardiac memory) subsequent of post pacing or artificial ventricular depolarization

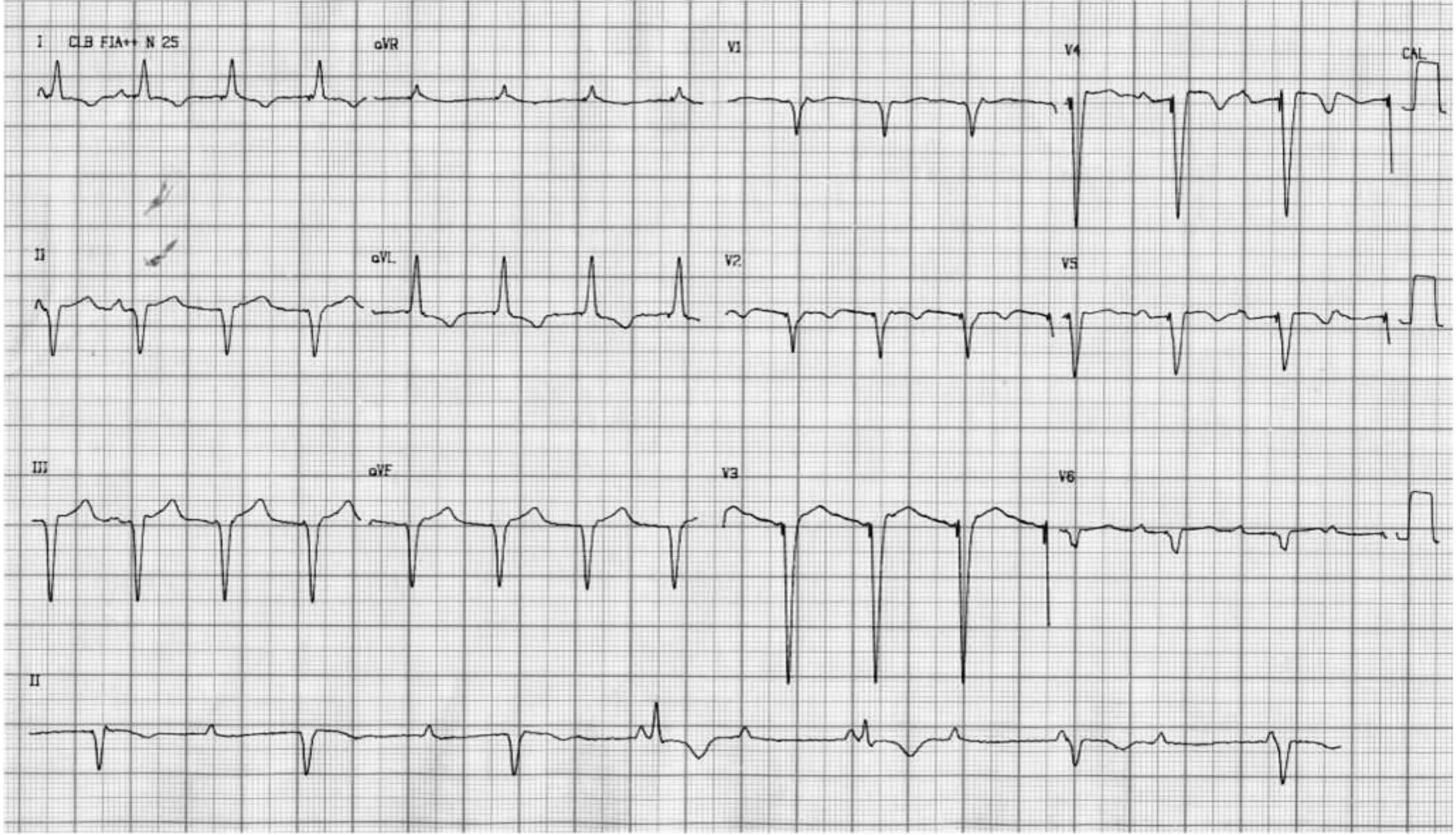
In the following example we show the case of a male patient, carrier of single-chamber permanent pacemaker, implanted by sinus node disease, who was admitted in the ER with atypical precordial pain and ECG displaying PM rhythm in VVI mode, alternating with his own rhythm, with T wave inversion in II, III, aVF and V1-V6, that were mistakenly interpreted as being of ischemic origin. In spite of the negative markers (troponin), the patient was referred for coronary angiography that revealed normal coronary arteries. Check that these alterations in ventricular repolarization occur after a period of ventricular stimulation, in the leads where QRS was negative. Moreover, the previous ECG shows normal QRS without repolarization alterations. This electrocardiographic evolution clearly configures the presence of CM.



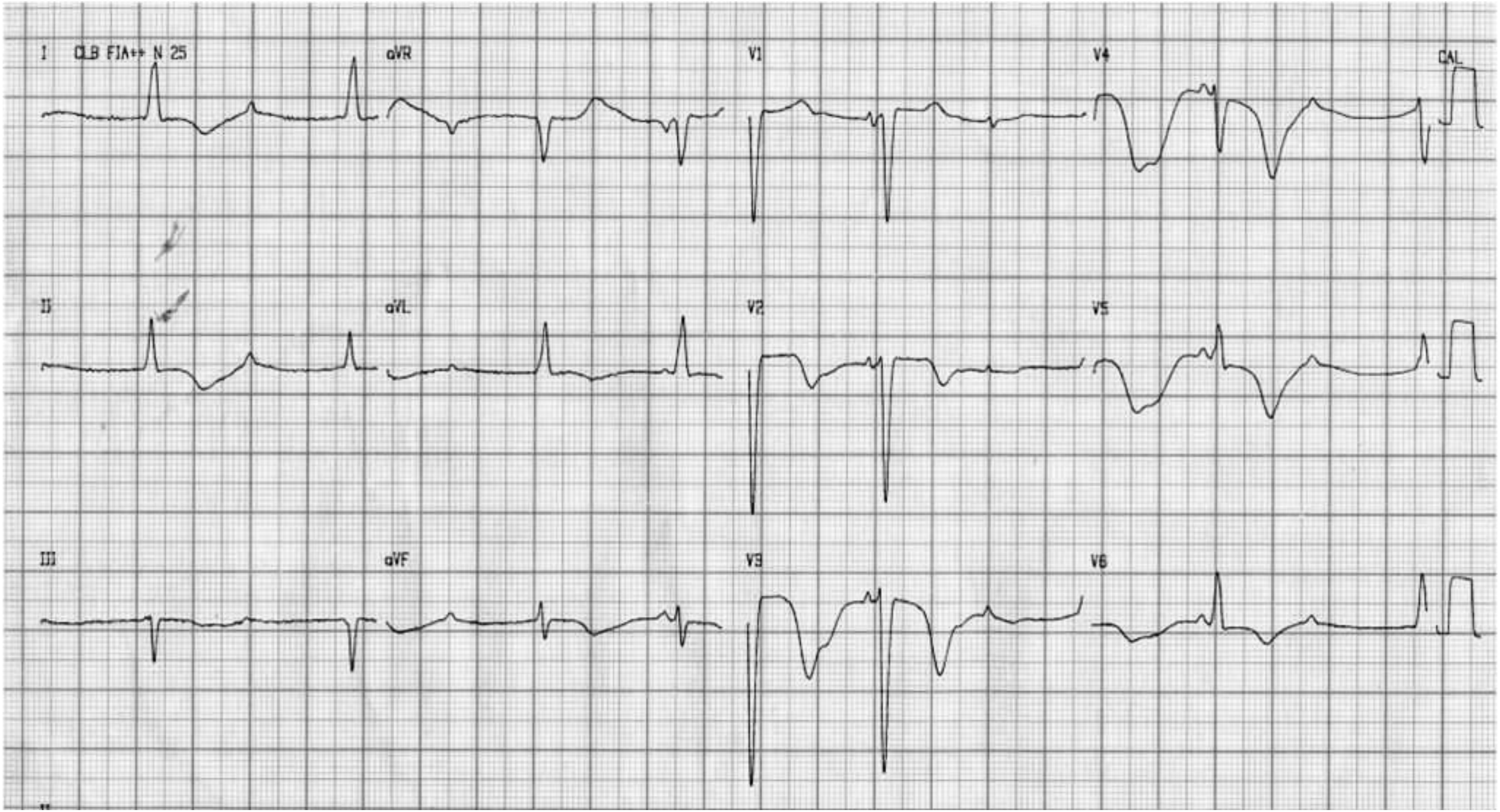
Although these electrocardiographic findings are attributed to ischemia, T wave inversion in the case of CM, does not correlate to myocardial ischemia. These alterations may arise early, and may persist for weeks after the onset of abnormal ventricular depolarization. Although the exact time relation has not been figured out yet, new investigations have shown that the time of permanence of T wave alterations depend on the so-called accumulation phenomenon. The phenomenon of CM may also occur after a brief period of temporary ventricular stimulation, in which ventricular repolarization alterations appeared after 24 hours of cardiac pacing in a patient admitted with syncope and ECG displaying the presence of complete AVB and runs of Torsades de Pointes (see next slide).



ECG long/continuous II in admittance.



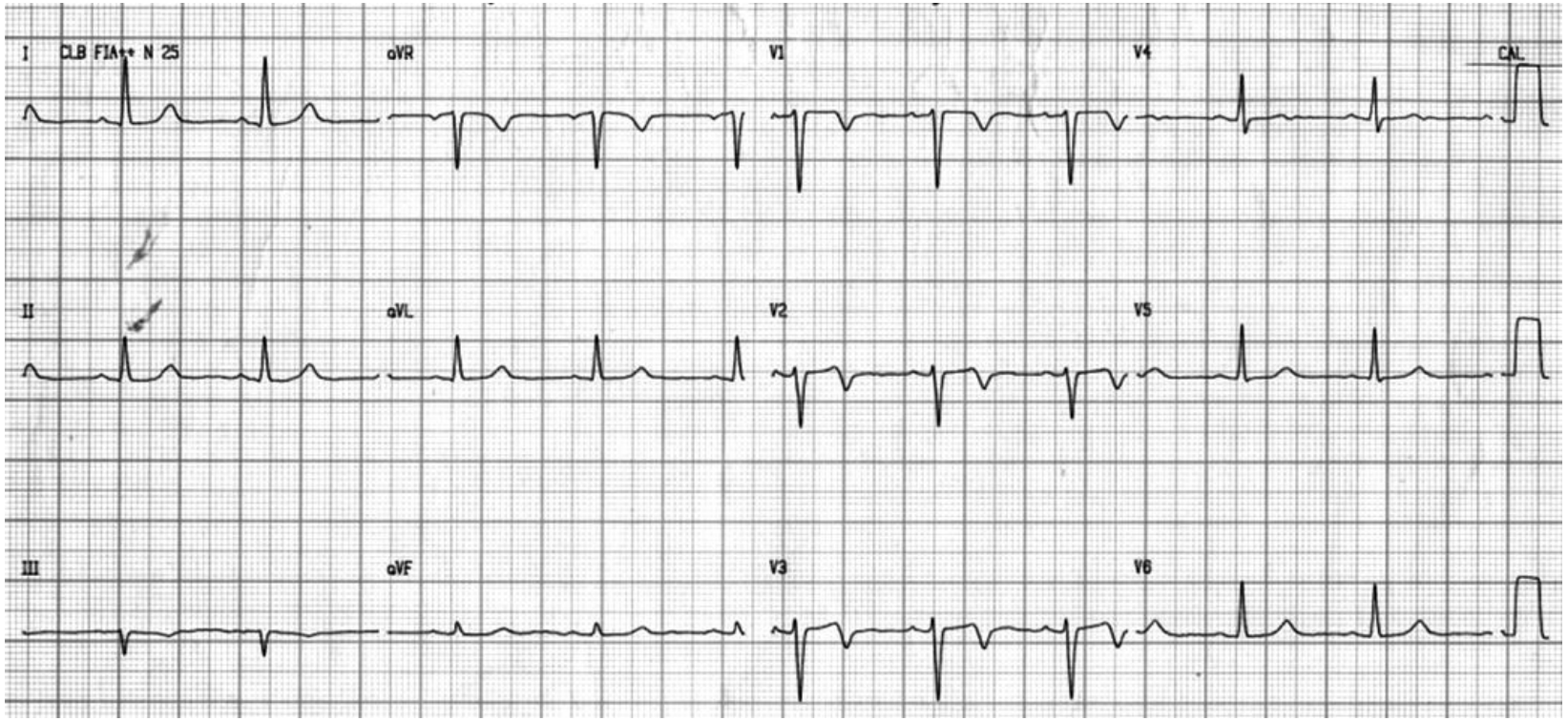
ECG after temporary pacemaker implant. Inferior tracing (long II) was recorded with the PM off.



Complete ECG with PM off after 24 hours, showing AV dissociation rhythm with long QT, ventricular repolarization alterations (negative T waves) in the same leads where QRS complexes were previously negative (II, III, aVF and V2-V6), configuring the effect of CM.

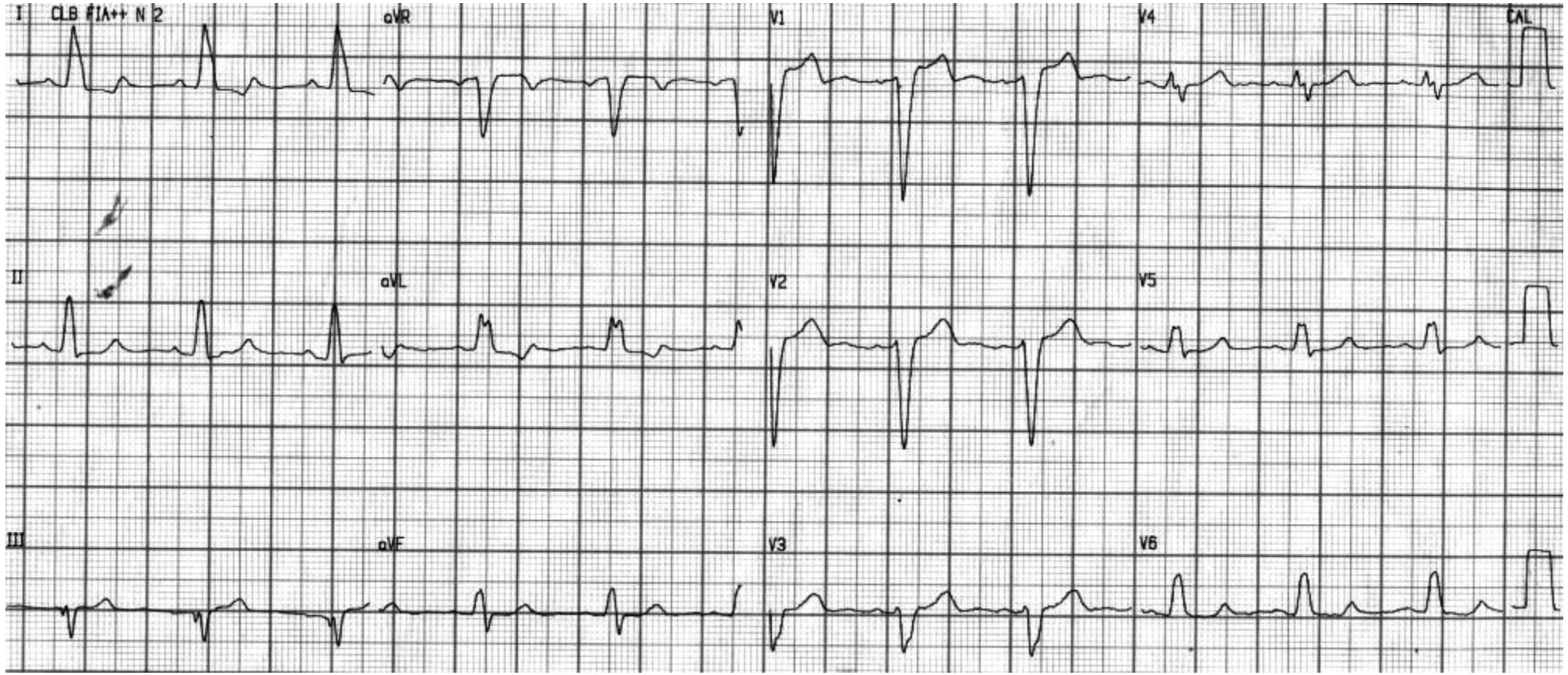
II. Cardiac Memory or T-Wave Memory after intermittent left bundle branch block

Case report: Female patient, 61 years of age, with history of presenting in her successive electrocardiograms, left bundle branch block (LBBB) of intermittent presentation (she brought previous tracings with her) and normal coronary angiography. Readmitted with oppressive precordial pain with no irradiation or concomitant symptoms. Physical examination: normal. Normal biochemical markers of necrosis. In the emergency room, she was medicated with acetylsalicylic acid, sublingual nitrate, clopidrogel, simvastatin, and enoxaparin. Admission ECG.



Electrocardiographic diagnosis: sinus rhythm, HR 65 bpm, SAP +55° and to the front, PR interval 140 ms, SAQRS +10° and backward, QRS duration 90 ms, SAT +30° in the FP and backward and the left in the HP with negative T wave from V1 through V3. **Note:** In normal adults invariably, the ventricular repolarization vector (T vector) is heading to the left and below, and usually discretely to the front around +10° in the HP. Consequently, normal T wave polarity is always positive from V3 to V6; generally positive in V2 and frequently negative in V1. In the frontal plane, normal SAT in the FP is between +15° and +80° consequently, T wave will always be positive in II and aVF and I; variable (biphasic or inverted) in aVL and III; and negative in aVR. **Conclusion:** negative T wave in the three right precordial leads. The previous ECG showed CLBBB pattern (figure next slide).

Cardiac memory after intermittent left bundle branch block (Rosenbaum 1973; 1982) “pseudoprimary T-wave changes”



Electrocardiographic diagnosis: Sinus rhythm, HR 74 bpm, PR interval 140 ms, SAQRS +10°, QRS duration 120 ms, QRS complexes of right precordial leads (V1 to V3), predominantly negative of the rS type, pure and monophasic R waves of slow inscription in the left leads: I, aVL and V6, QRS complex of the QS type in aVR, ST-T opposite to greater QRS deflection: positive from V1 to V3, but not opposite in the left leads I, aVL, V5 and V6. Positive T wave in aVL and positive or minus-plus in I, constitutes a criterion that leans in favor of cardiac memory (CM) (**Shvilkin 2005**). Conceptually, by presenting QRS duration = 120 ms from the merely morphological point of view she meets the concept of complete left bundle branch block (CLBBB) or advanced left bundle branch block (ALBBB); however, this is incomplete second degree block. Why? Answer: because following the concept of Professor Antoni Bayés de Luna, any intermittent left bundle branch block is necessarily of a second degree, even when reaching the arbitrary criterion of 120 ms for QRS duration (**Antoni Bayés de Luna 1998**). Thus, according to permanence, left bundle branch blocks could be classified in the following manner:

- **Permanent or definitive:** most of them. These are troncular or of a third degree, advanced or complete (ALBBB or CLBBB).
- **Intermittent or of the second degree:** which in turn, could be:

1) Dependent on heart rate:
- Tachycardia-dependent or in “phase 3”;
- Bradycardia-dependent or in “phase 4”.

1) Independent from heart rate:
- Mobitz type I;
- Mobitz type II by Wenckebach phenomenon;
- By significant hypopolarization.

In this group of intermittent or transient blocks, the phenomenon of Cardiac Memory (CM) or T-wave memory may occasionally be observed, manifested by changes in T wave polarity, following the intermittent alterations of ventricular activation or depolarization in a given time after conduction without dromotropic disorder.

Normal echocardiogram

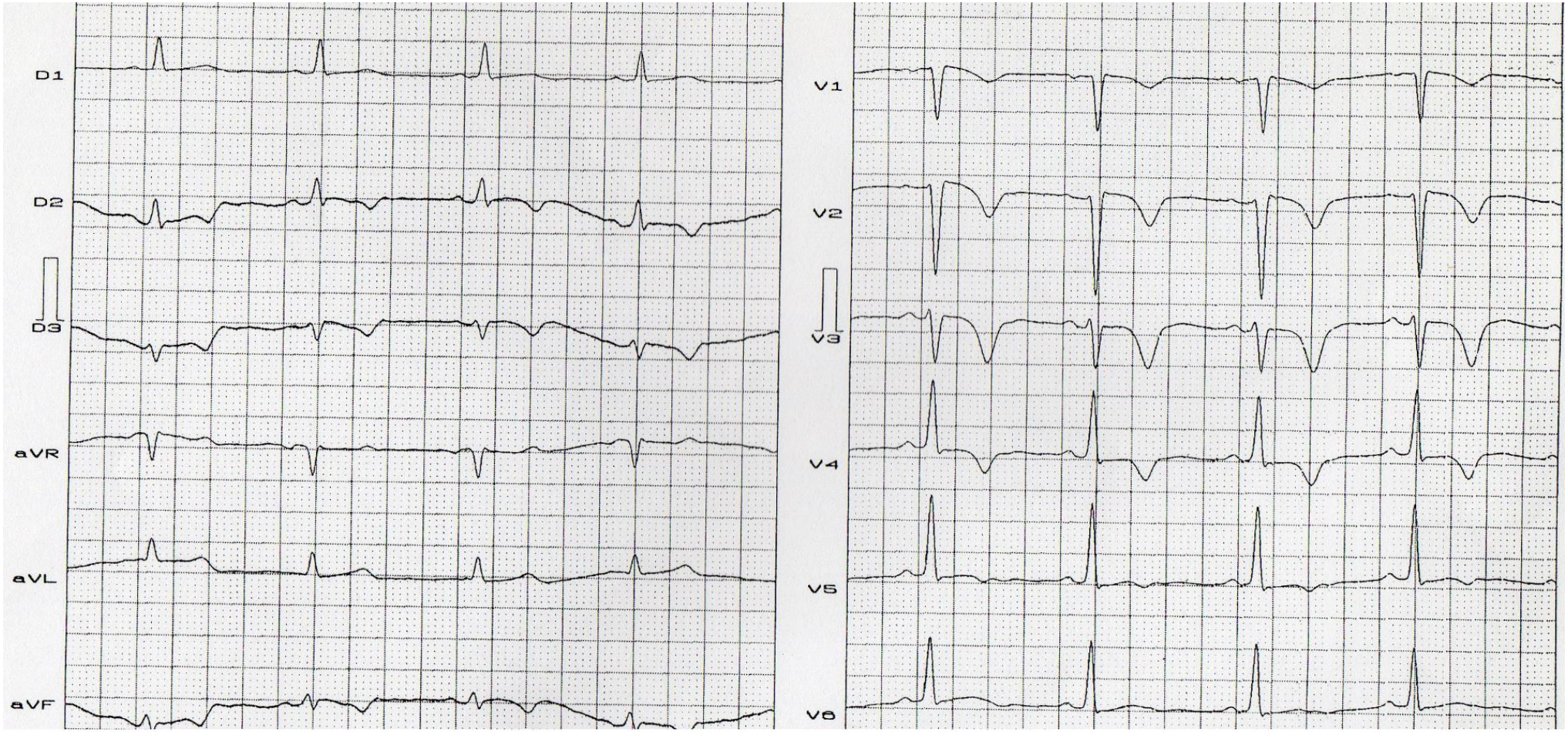
New cardiac catheterization revealed normal coronary arteries and ventriculography.

The most convincing and studied example with ECG and cardiogram vector (VCG), all belonging to the same patient. The ECG was made approximately 3 days before the ECG/VCG of the tracings, all made at the same time.

This is a 38-year-old lady, who came to our clinic 3 days before and our colleague made the ECG, with the aim of a pre-participation evaluation for a gym academy. Negative and irrelevant family history, novices, she denies having any previous disease. She always had normal blood pressure.

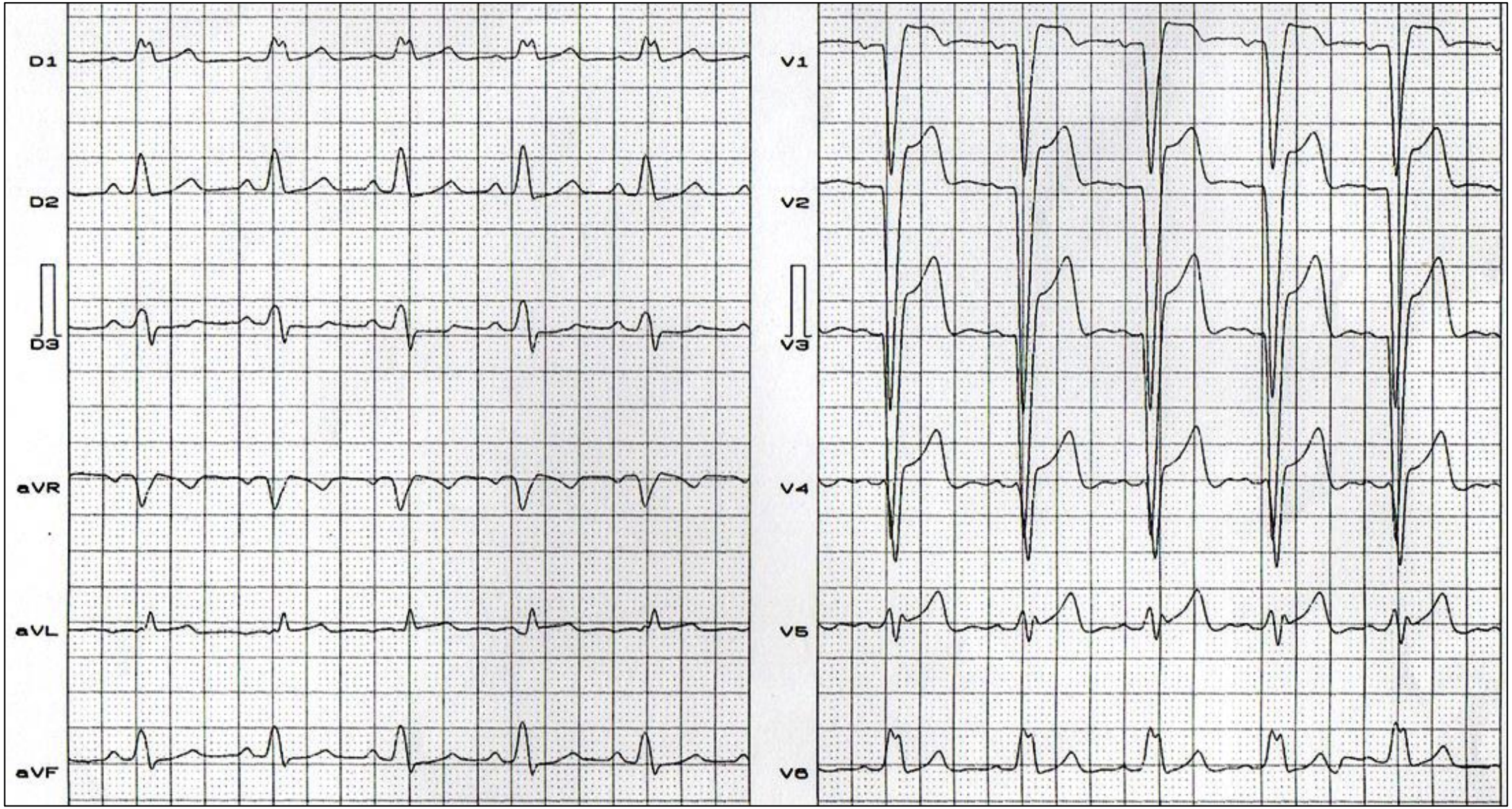
Normal transthoracic echocardiogram and a tomography of the coronary arteries made later, revealed normal calcium score and permeable coronary arteries with no anomalies.

Date: Feb 27, 2013; **Time**: 17 h; **Age**: 38 y/o; **Sex**: Female; **Ethnic group**: Caucasian.



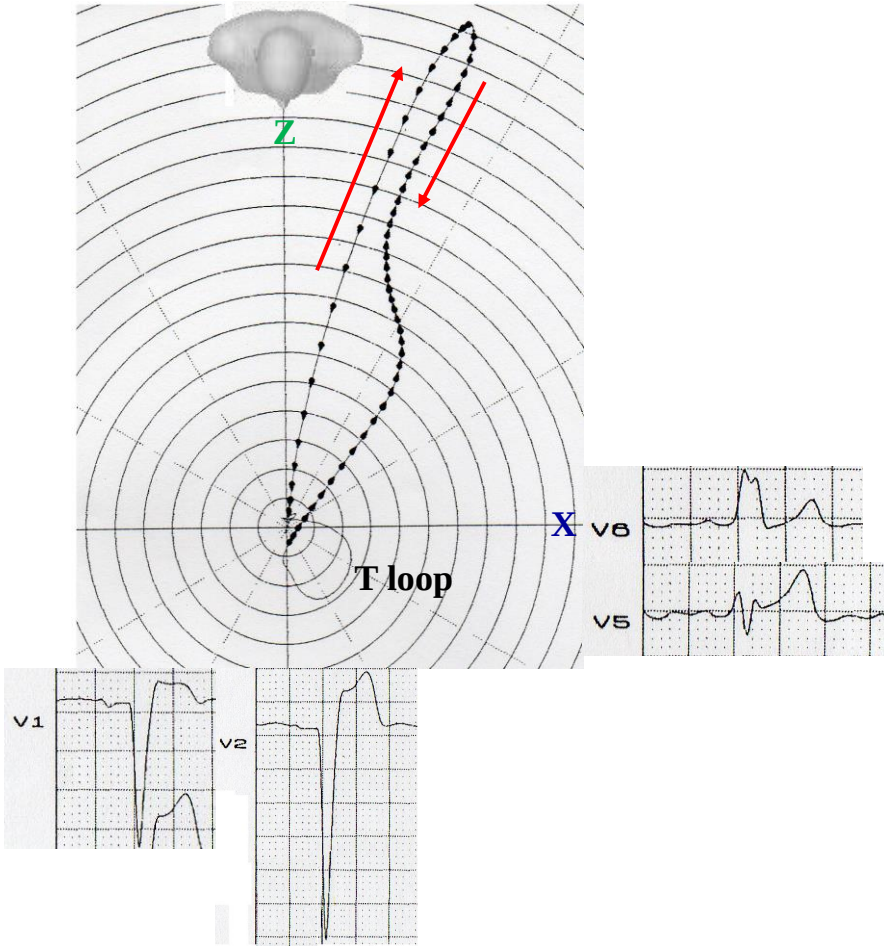
ECG diagnosis: Sinus rhythm, heart rate 83 bpm; SÂQRS +15° and backward with QRS complexes of the QS type in V1 and rS with small initial r in V2-V3 and sudden passage of the transition area in V4, where the QRS complex is of the pure R or Rs type. This sudden passage from complexes of the rS type to complexes of the Rs type from V3 to V4 without recording R/S transition complex, eventually occurs in the presence of LVH by posterior shift of the QRS loop in the HP. Negative T waves in II, III and aVF and in V1 and deeply negative from V2 though V4, lead to the doubt of anterior and inferior ischemia or a chance of non-obstructive Ap-HCM by the presence of deep, giant, negative T waves from V2 to V4 in a totally asymptomatic person. In athletes with anamnesis and normal physical examination, the presence of deep, inverted T waves in ≥2 or more contiguous leads in the anterior or lateral wall should lead to the suspicion of HCM or ARVC/D (**Wilson 2012**).

Date: March 2, 2013; **Time**: 17 h; **Age**: 38 y/o; **Sex**: Female; **Ethnic group**: Caucasian.



ECG diagnosis: Typical CLBBB pattern by QRS duration of 120 ms and characteristic pattern; however, followed by positive T waves in left precordial leads.

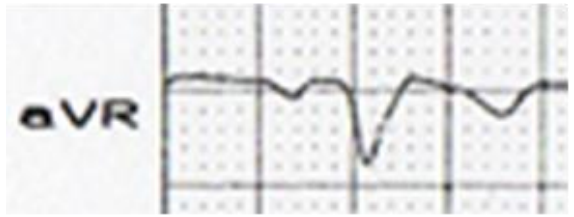
ECG/VCG correlation in the Horizontal Plane

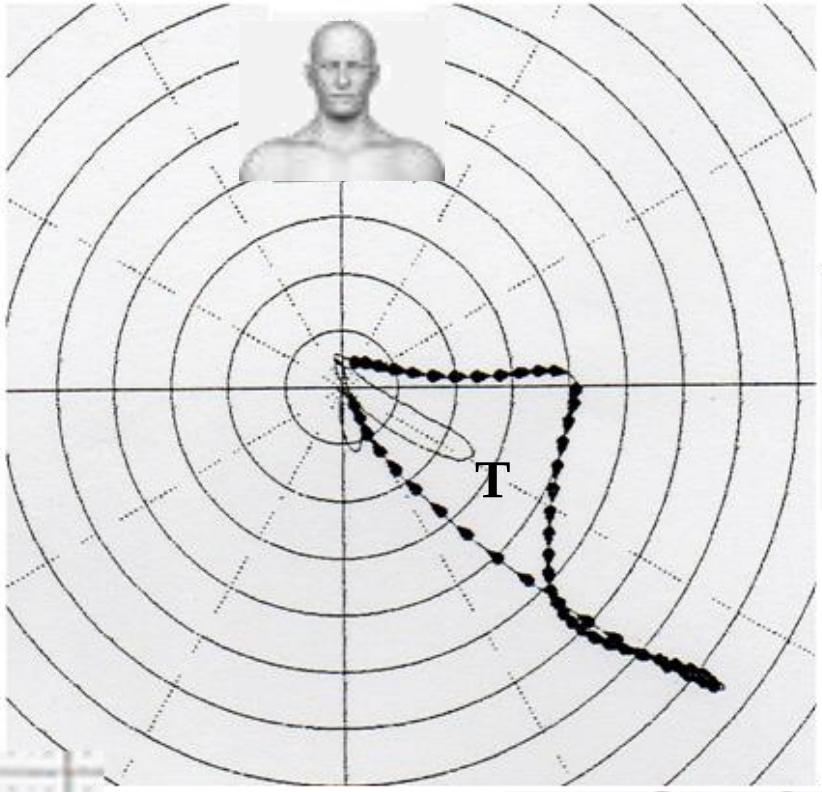


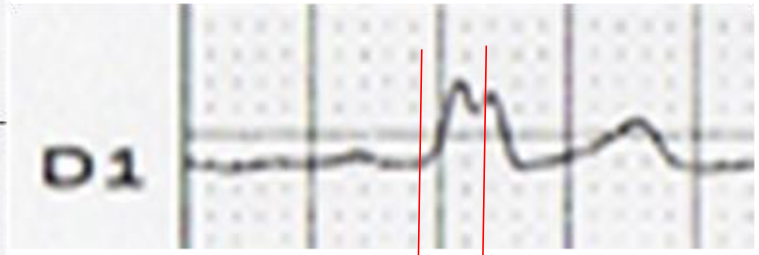
VCG diagnosis: narrow, long QRS loop, with increased magnitude (>2 mV), of clockwise rotation with maximal QRS vector located in the left posterior quadrant (between -70° and -60°). Efferent branch located at the right and afferent branch located at the left and of slow **middle-final** inscription (dashes very close to each other, characteristic of LBBB). T loop of non-secondary or abnormal (rounded) aspect and located to the front and left (in the left anterior quadrant). Regrettably, this device does not allow inferring conduction velocity in the T loop afferent and efferent branches.

ECG diagnosis: QRS complexes of the QS type in V1 and V2 and pure monophasic R in V6 with wider QRS. Ventricular repolarization opposite to depolarization only in the right precordial leads and matching in V5-V6.

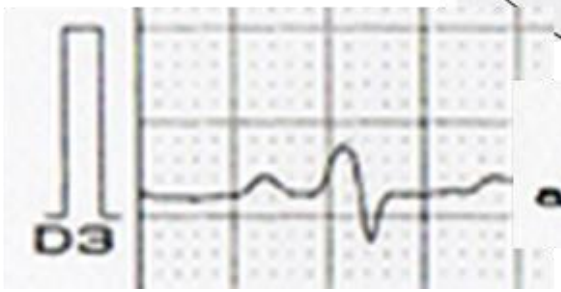
ECG/VCG correlation in the Frontal Plane

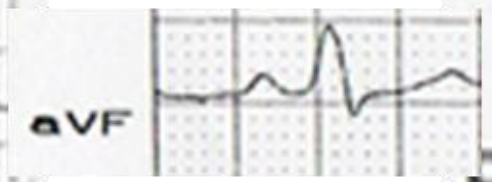


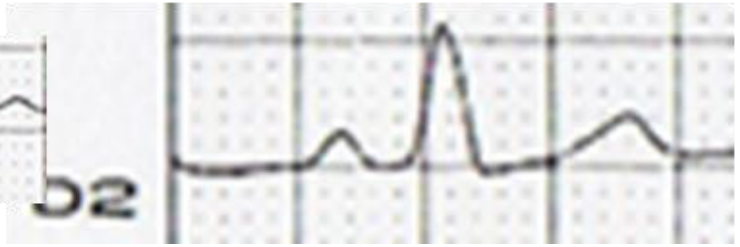




Prolonged Ventricular Activation Time (VAT) >50 ms







VCG diagnosis: P loop in +70°, SÂQRS close to +35° and QRS loop of counterclockwise rotation.
T loop is not opposite to QRS loop, a fact considered atypical in the classical CLBBB. (T loop located in +30° and matching QRS loop in +35°).
ECG diagnosis: SÂP = +30°, PR interval = 140 ms, QRS duration = 120 ms, pure, wide and monophasic R wave "in tower", with notch in I, aVL and ventricular activation time >50 ms in I, SAT in +30°; i.e. not opposite to QRS.

ECG/VCG correlation in the Right Sagittal Plane

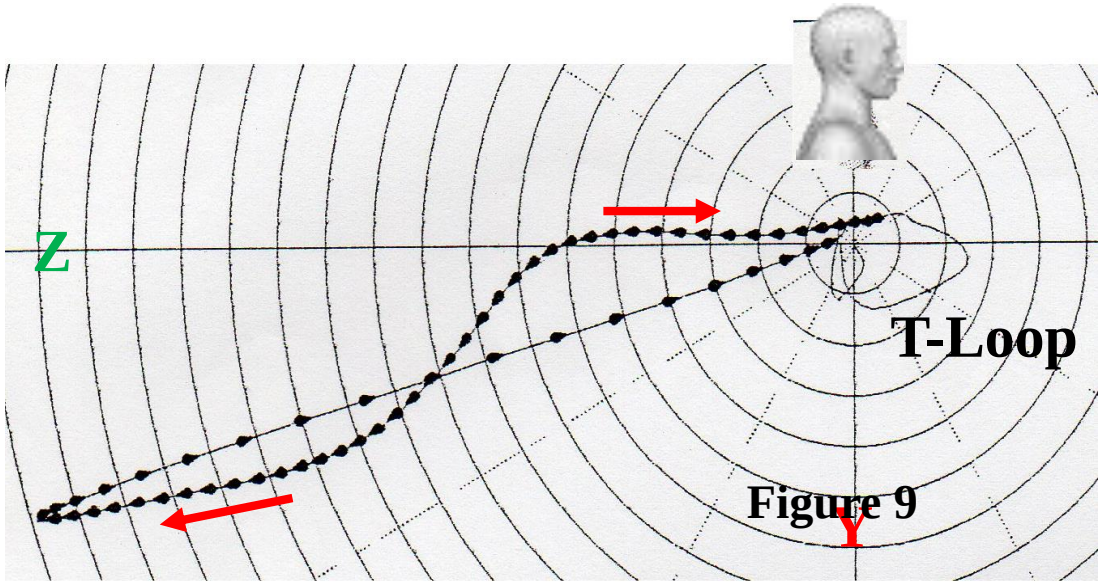
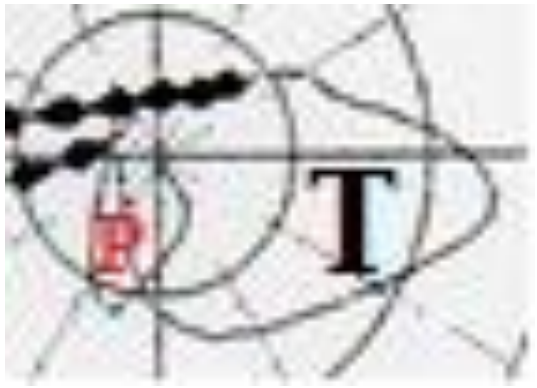
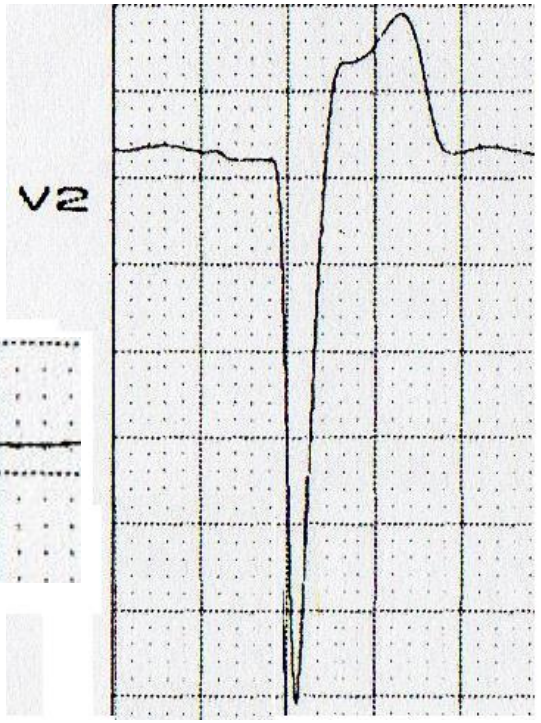
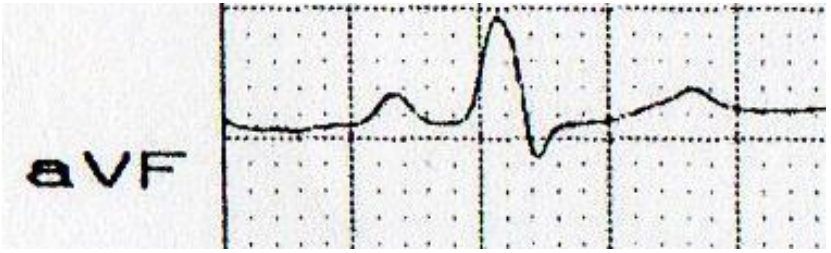


Figure 9

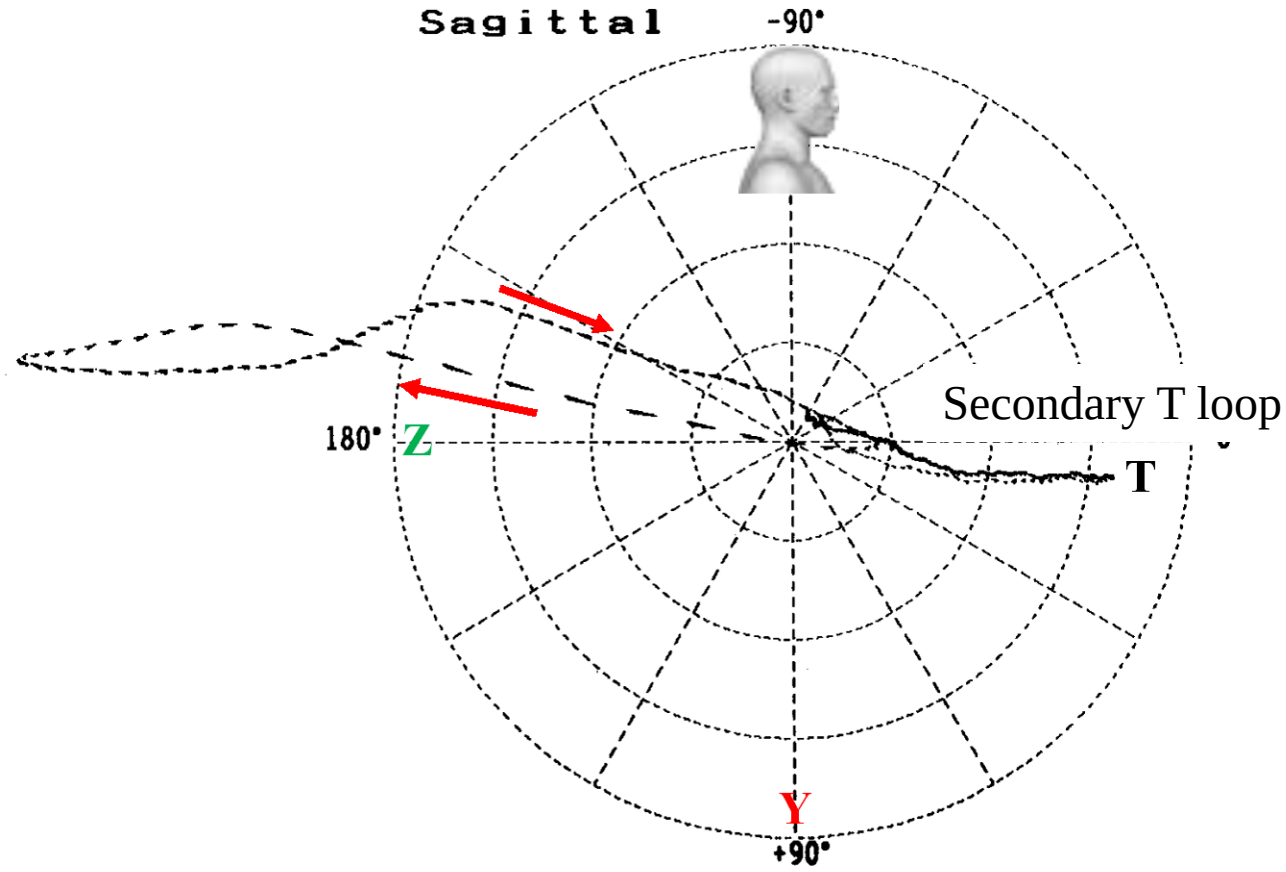




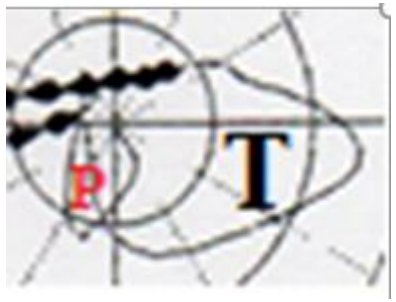


VCG diagnosis: QRS loop is located in the posterior-inferior quadrant, rotation in eight, and with afferent loop of slow **middle-final** conduction. T loop of opposite direction to QRS loop heading to the front and with altered (rounded) morphology. In uncomplicated CLBBB, T loop is fusiform or elongated as shown the T-loop of the next slide.

Uncomplicated LBBB on Right Sagittal Plane

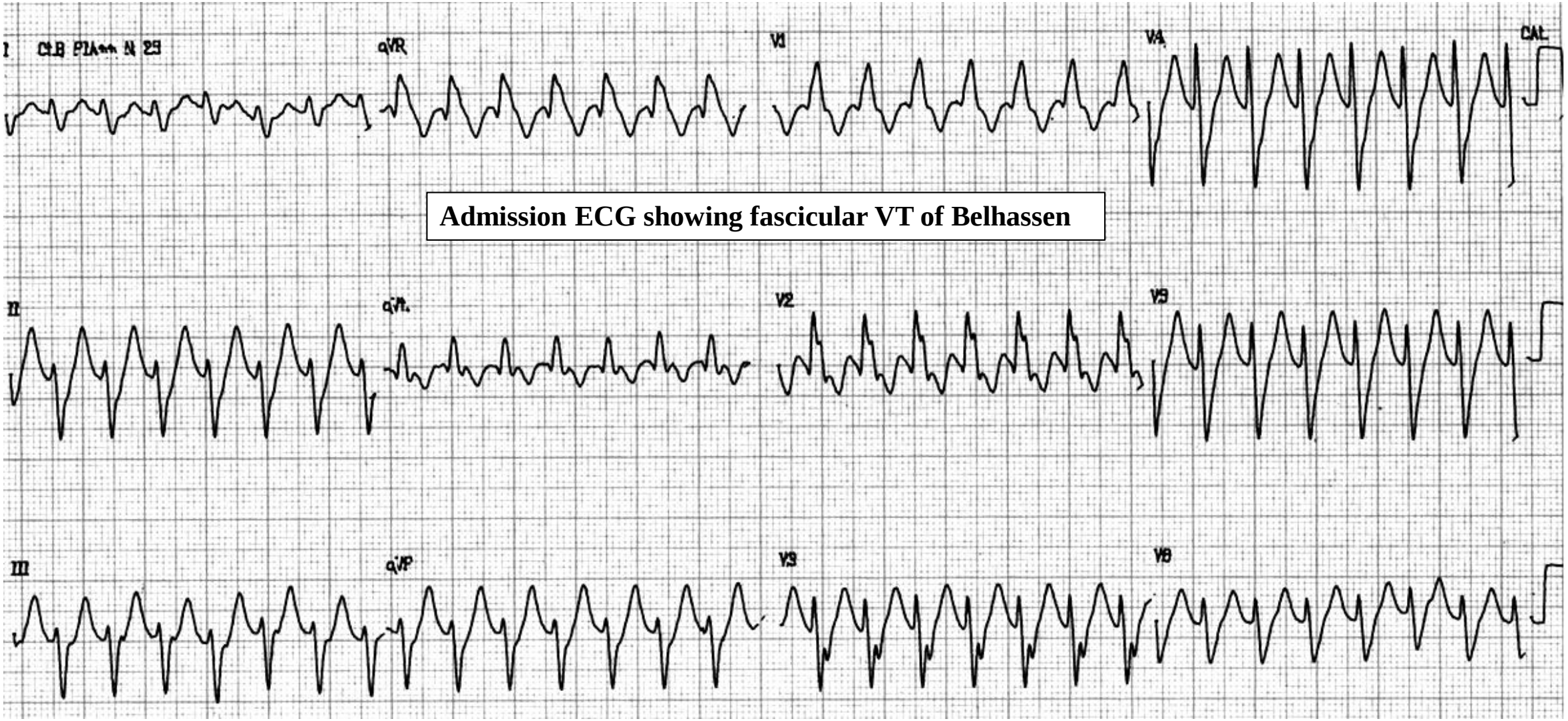


Primary T-loop



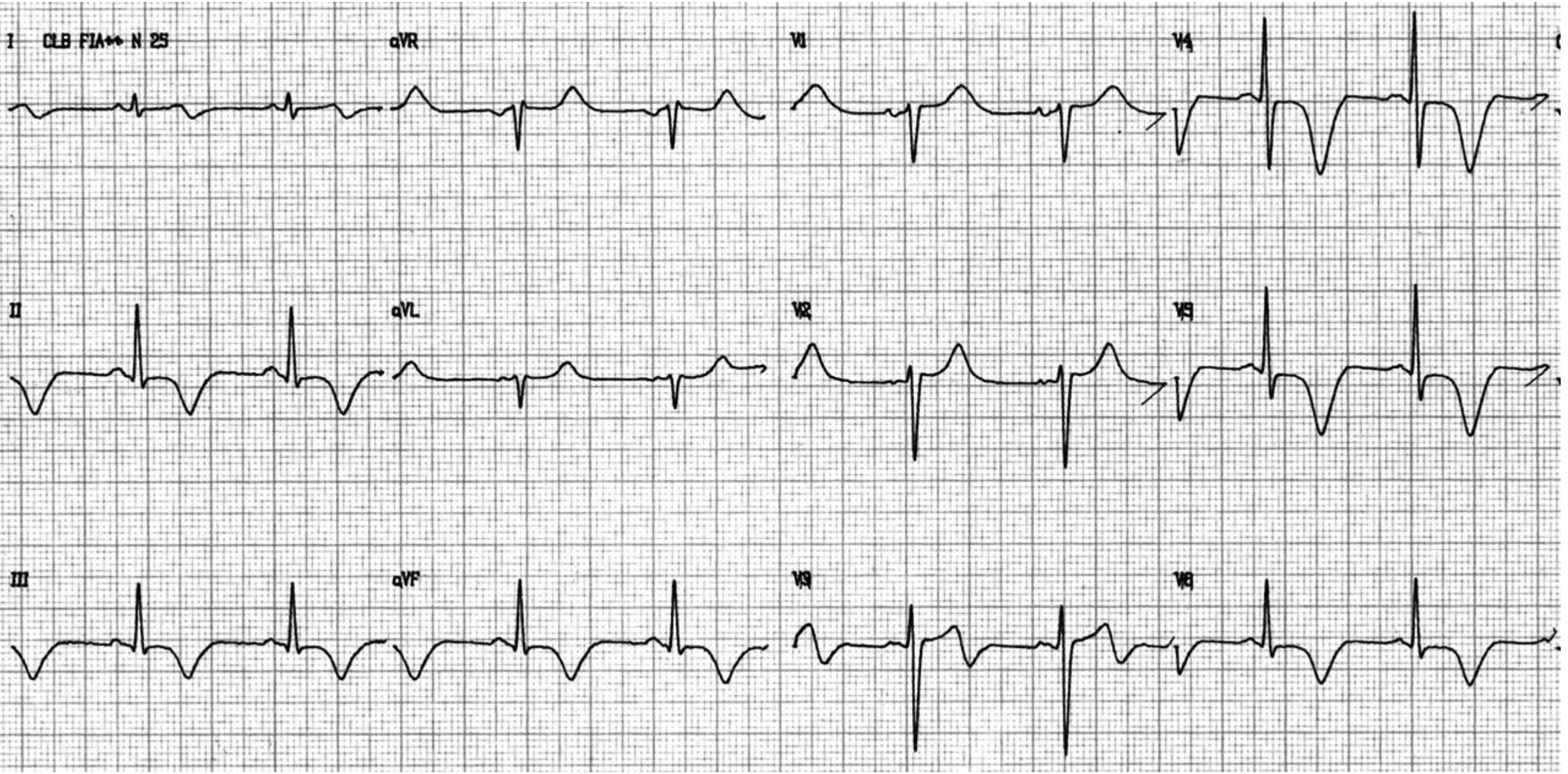
VCG of a patient carrier of uncomplicated CLBBB in the right sagittal plane. Check the thin and long aspect of the T-loop heading to the front, close to +3°. In this case, the device allows seeing that the efferent branch of the T loop presents a slower recording than the afferent branch. This T loop is very different from the T loop aspect of the primary T-loop, which is rounded, small and with symmetric branches

III. Cardiac Memory(CM) after episodes of wide QRS tachycardia post-paroxysmal wide tachycardia syndrome (Kernohan 1969)

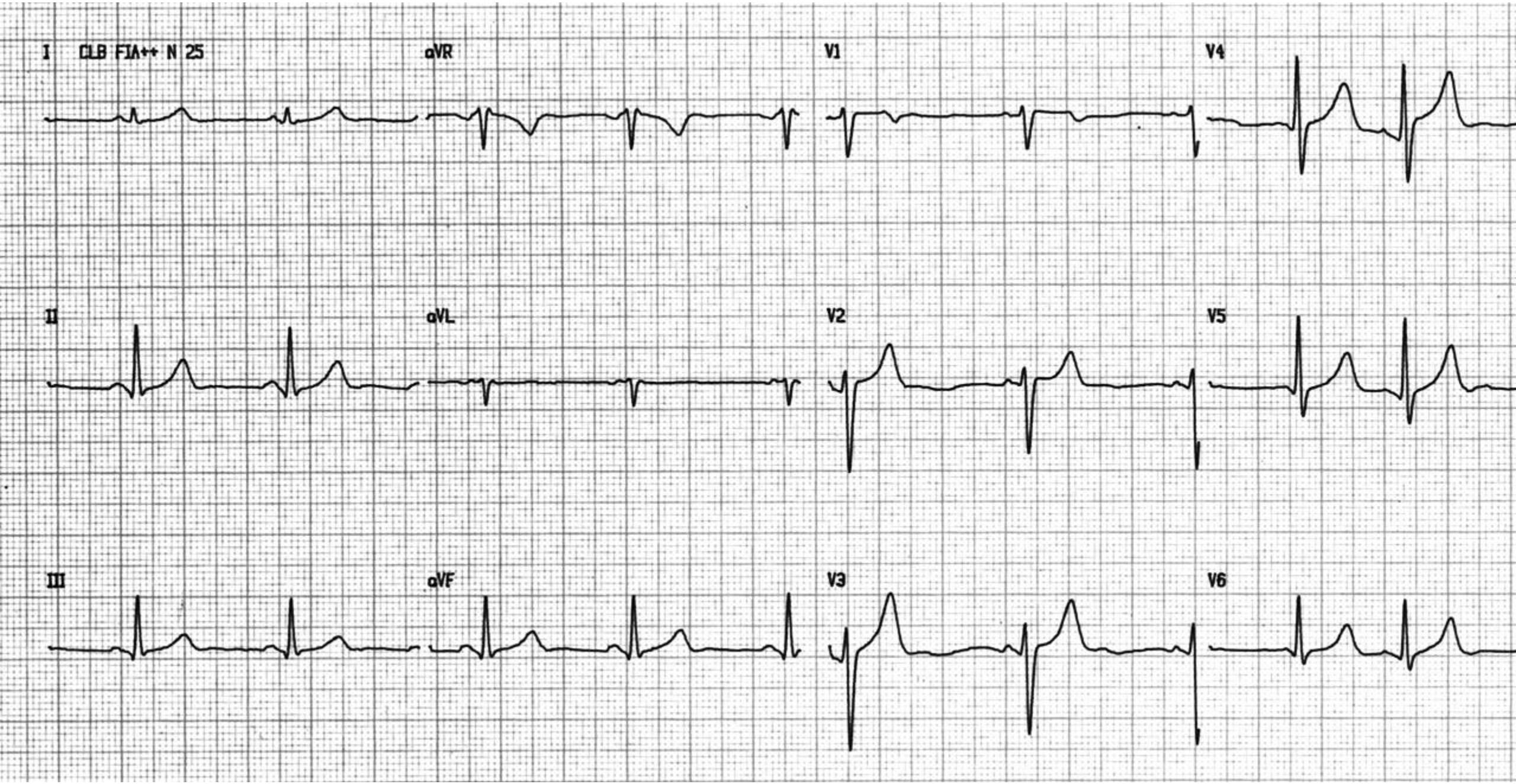


Admission ECG showing fascicular VT of Belhassen

This is the case of a young male, 22-year-old patient, admitted during event of fascicular ventricular tachycardia, in whom ECG after reversion with IV verapamil, shows negative T waves suggesting CM. Such alterations normalized after 1 week, according to the electrocardiographic evolution described below.



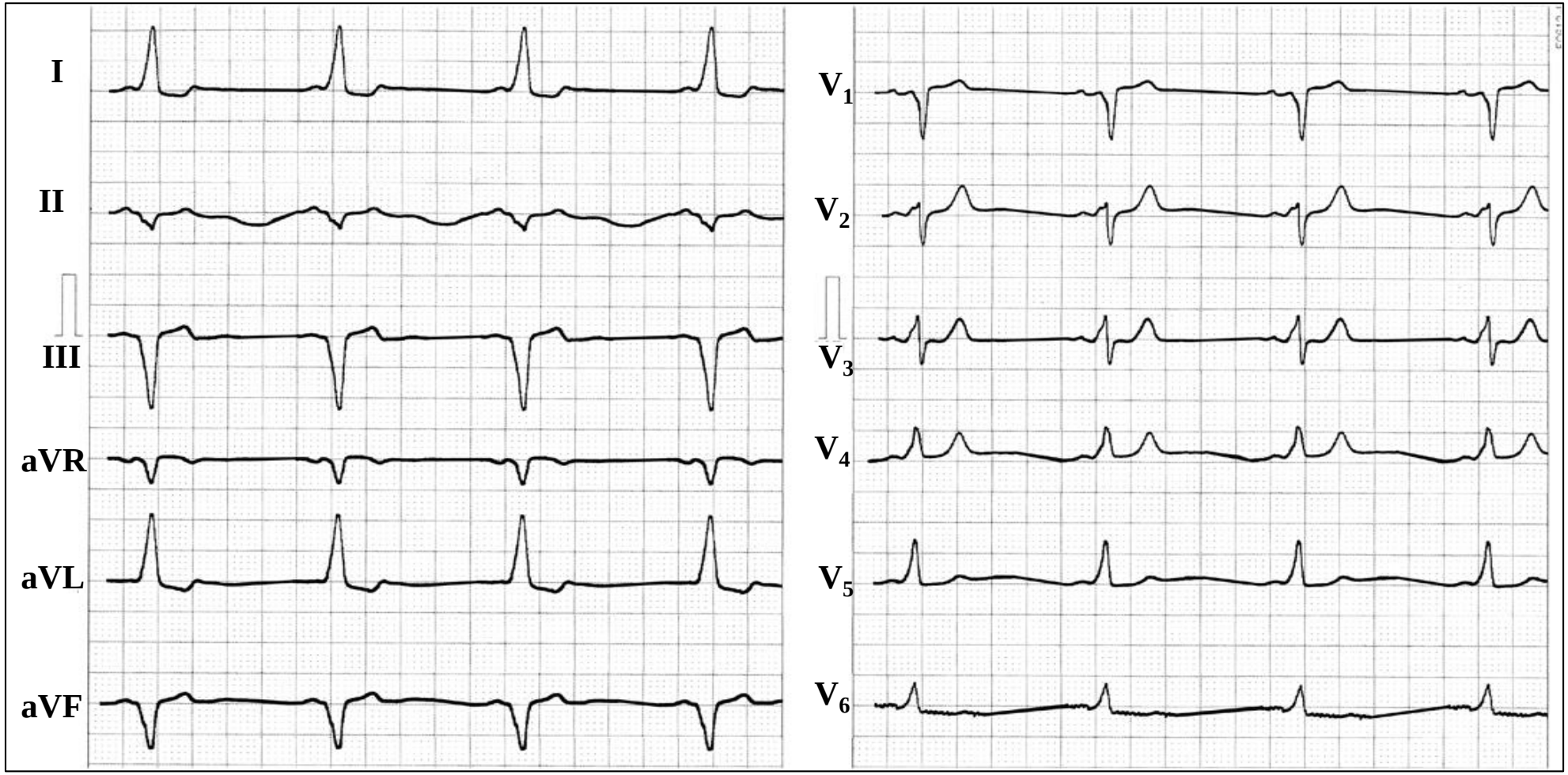
ECG after reversion to sinus rhythm with verapamil, showing negative T waves in II, III, aVF and from V4 through V6 and plus-minus in I, typical of CM phenomenon.



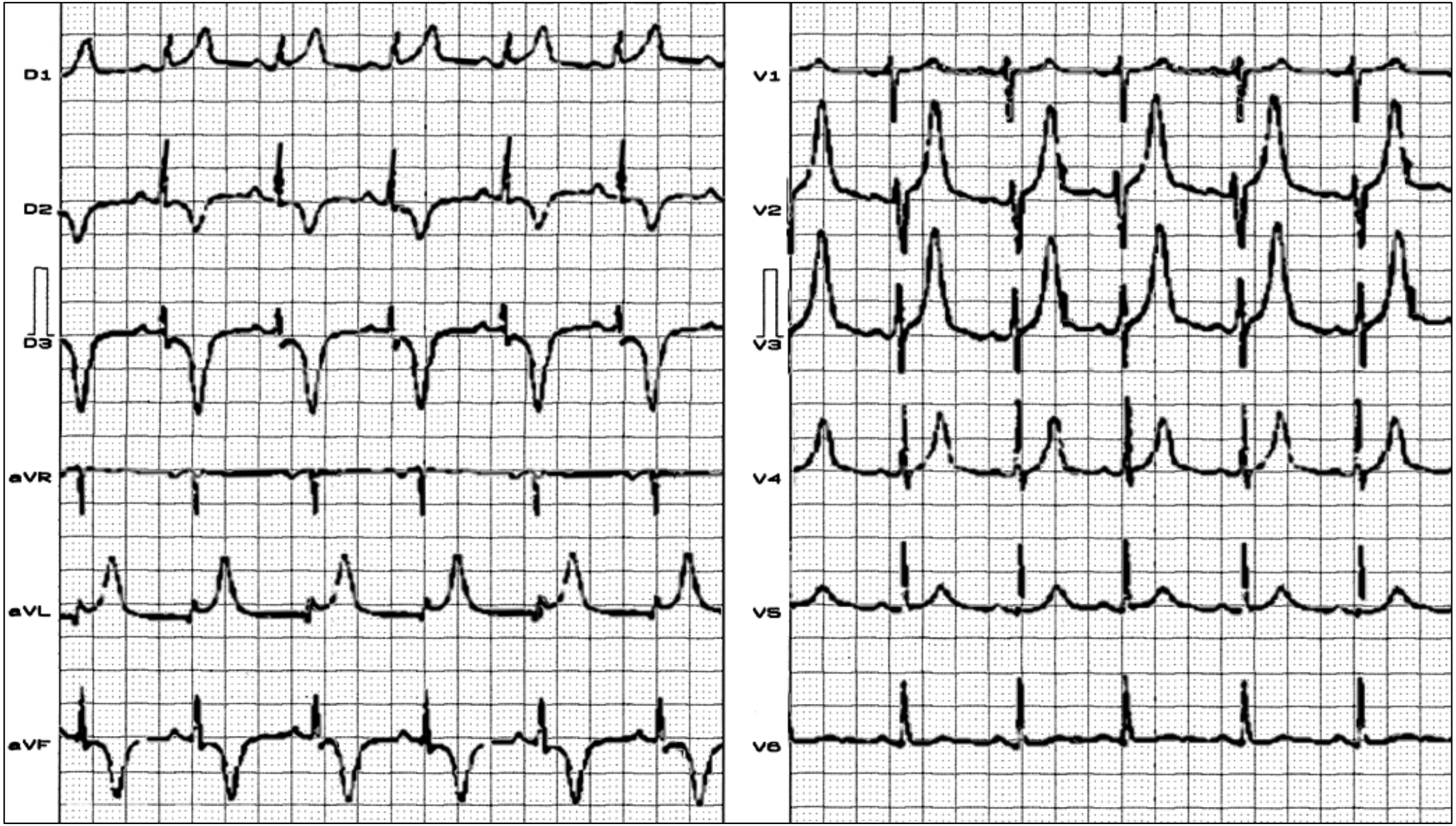
ECG performed after 1 week, showing normalization of alterations in ventricular repolarization.

IV. Cardiac memory after unespecific intraventricular conduction defects (NSIVCD): The present case

V. Cardiac memory after ablation of anomalous pathway in Wolff-Parkinson-White



ECG diagnosis before ablation: WPW right anterior free wall of the RV, point 2 of Gallagher, B or European I type WPW, and Region 5 of Lindsay. It may resemble electrically inactive area in inferior wall (QS in II, III and aVF), inferior infarction and LBBB – pure R in (I, aVL, V_5 and V_6) and QS or rS in V_1 and V_2.



ECG immediately after ablation, there are marked T wave abnormalities with deep T wave inversion in the inferior leads and T wave peaking in leads I and aVL and V2 to V4: cardiac memory. In each lead, the directional change of the T wave from before to after ablation is in the same direction as that of the δ wave on the baseline ECG.

Possible causes for Cardiac Memory

The phenomenon of cardiac memory (CM) is characterized by the presence of negative T waves preceded by normal QRS complexes after a conditioning period of abnormal ventricular depolarization in the same ECG leads. The term CM was coined by Mauricio Rosenbaum and his school (**Rosenbaum 1982**) in an experimental fashion in dogs to describe electrocardiographic alterations in ventricular repolarization induced by abnormal ventricular activation by:

1. Intermittent left bundle branch block (**Byrne 2010**).
2. After ventricular pacemaker (**Kolb 2002**). In this case, CM that induces negative T waves, is caused by the presence of transmural gradients of repolarization manifest during atrial pacing, which is maximum near the site of ventricular stimulation (**Coronel 2007**).
3. Following an episode of ventricular tachyarrhythmia (**Omidvar 2013**).
4. After ablation of anomalous pathway in Wolff-Parkinson-White or transitorily in an intermittent fashion. 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**).

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.

Concept of CM accumulation

Accumulation: duration and frequency of depolarization alteration is a predictor in relation to the time of the phenomenon of CM, which will remain present after the end of ventricular depolarization alteration. Consequently, when CM is observed after a short event of tachyarrhythmia, the phenomenon of CM will last shortly, and on the contrary, when depolarization alteration was prolonged as it happens after preexcitation removed by ablation, T wave changes are observed for a long period of hours or days (**Takada 2002**). Inden et al (**Inden 2001**) found that the prolongation in the duration of action potential of the epicardium was present before, and persisted after preexcitation ablation. Gradual changes on repolarization properties include action potential prolongation that continues after ablation procedure and may be justified by the phenomenon of accumulation proper of CM.

Mechanisms attempting to explain the phenomenon of cardiac memory.

A) Hypothesis of modification in the expression of sarcolemmal ion channels, of connexin 43 and calcium percentage in the intracellular sarcoplasmic reticulum.

In CM, two early and late regions could be activated.

I) Region of early activation located proximally to the site of altered activation, causing changes in the electrotonic flow, mediated by angiotensin II receptors. The block of angiotensin I receptor attenuates short-term CM, which in the last case modifies the sarcolemmal potential. Three important channels have been identified as being responsible for the early CM phenomenon and the expression of connexin 43:

   a. Decrease in transient outward potassium channel activity in phase 1, causing decrease in notch of epicardial cells. This decrease is due to the regulation in the expression of Kv4 channels and stability of Kv4.3 mRNA channel. LV pacing causes a loss in the epicardium notch and T vector shift mentioned as CM initiated by local increase in angiotensin I in the Hek293 cells, in which the Kv4.3 and KchlP3 subunits contribute to a greater Ito expression with the angiotensin I receptor (AT1R) (**Özgen 2012**). The CM phenomenon could be abolished using the transient outward potassium channel blocker, Ito 4-aminopyridine (4-AP) channel (**Geller 1993**). Changes in Ito concentration affect action potential, modify vulnerability to arrhythmias and influence excitation-contraction coupling. A decrease in Ito density is observed in immature hearts, in elderly people, cardiomyopathies and heart failure. A decrease in Ito density causes action potential duration prolongation, and less calcium efflux by the Na^{+}/Ca^{2+} exchanger channel. Both facts favor calcium increase in the sarcoplasmic reticulum with accumulation of this cation in the intracellular medium, thus favoring triggered arrhythmias.
   b. Decrease in activity of the L-type calcium current. Pharmacological block of the slow calcium channel attenuates the appearance of CM, both early and late. On the other hand, and rapidly, it decreases the activation of the delayed outward-rectifier I_{Kr} channel.
   c. Decrease in the activation of the delayed rapid rectifier I_{Kr} channel in phase 3, and consequently reduction in I_{Kr} transmural gradient (**Obreztchikova 2006**).
   d. Reduction in the expression of connexin 43. The proteins that make up Gap junctions are known as connexins. The most numerous connexin found in the heart is connexin 43, and in a lower number, connexins 40 and 45. In the ventricles, there is a large amount of connexins 43 and 45, and very little of connexin 40. In the atria, there is a large amount of the three types. Connexin 43 is the greatest determinant of the electrical properties of the cardiac muscle. The closure of gap junction is at the level of this connexin, causes negative dromotropism and its decrease may contribute to the CM phenomenon (**Patel 2001**).

II. Late-activated region, distal to the site of altered activation. Characterized by significant prolongation of action potential due to increase in mechanical strain. In spite of a marked action potential duration (APD) prolongation, there was a surprisingly minimum variation in the densities of ion channels in the sarcolemma and a significant increase in cytosolic calcium concentration (twice the concentration of cytosolic CA) responsible for the prolongation in APD in the region of late activation, increasing the activity of the Na^{+}/Ca^{2+} exchanger channel (**Jeyaraj 2013**). In summary, the basis for memory is caused by an increase in the electrophysiological regional gradients.

B. Hypothesis that supports the basis of CM as the presence of a slow acting mechano-electric feedback mechanism.

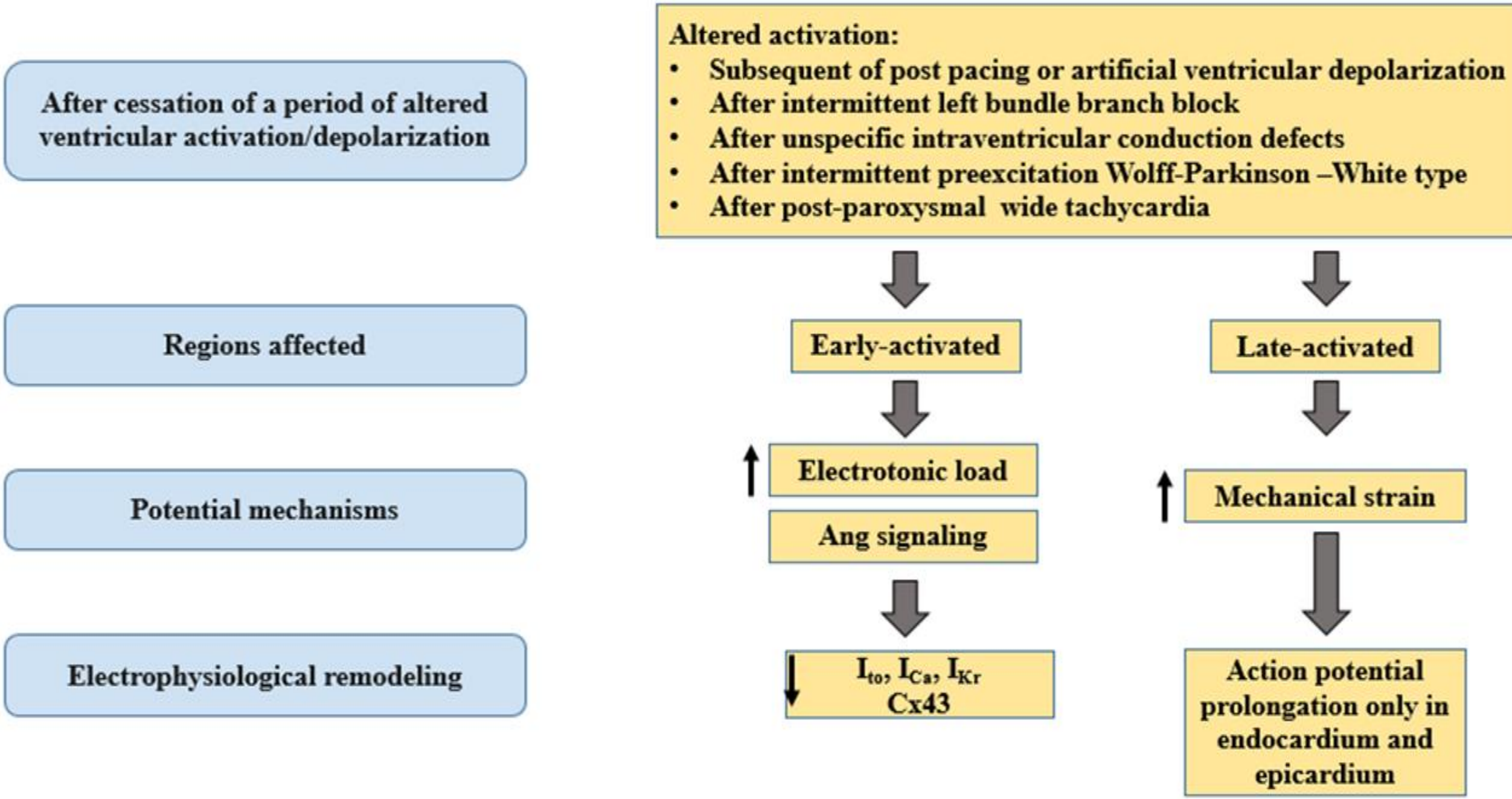
C. In ECG, T and R waves are matching during normal sinus rhythm, but mismatching after a period of ventricular pacing. This phenomenon called CM, could be mediated by mechanic stimulus. Hermeling et al (**Hermeling 2012**), using a mathematical model investigated if this phenomenon of slow acting mechano-electric feedback explains CM. The authors built a model that resembled the behavior of the left ventricle using serial coupled mechanic and electric segments. Every segment was equipped with ion currents of the membrane, of calcium handling channels and slow excitation-contraction coupling of slow acting mechano-electric feedback. The model showed T wave concordance with R wave in normal sinus rhythm and acute mismatching T waves after restoration of sinus rhythm. These results of LV simulation indicate that the slow coupling of excitation-contraction of mechano-electric feedback in the LV may explain:

   a. The relatively small differences in systolic shortening and mechanic work during sinus rhythm;
   b. The small dispersion in repolarization time;
   c. Matching T waves during sinus rhythm;
   d. The phenomenon of CM.

The physiological distribution in the electrophysiology properties, reflected by matching T waves, may be useful to optimize the cardiac pump function.

The figure of next slide summarizes the main mechanisms of CM.

Summary of cardiac memory features



How to suspect, in the daily clinical practice, that we are facing an effect of CM?

First, we should bear in minds which are the circumstances in which this phenomenon occurs (LBBB, cardiac stimulation, presence of accessory pathways and ventricular arrhythmias). If there was presence of precordial pain associated to some of the previously mentioned conditions, the diagnosis of this phenomenon becomes more complex. In this situation, it is imperative to make a clinical analysis of the characteristic of the pain (typical or atypical), along with T wave morphology, and whenever possible, to make a comparison with previous tracings. The analysis of cardiovascular risk factors is important too, since we have to rule out the presence of true ischemic episode. Diffuse T wave inversion may be frequently observed in the presence of lesion in the anterior descending artery, but also in the CM effect after ventricular pacing, making the differential diagnosis between these 2 phenomena difficult and complex. A recent study (**Rosenbaum 1982**) proposes a series of criteria that favor the diagnosis of CM and not of myocardial ischemia in this scenario:

- Positive T wave in aVL
- Positive or isodiphasic T wave in I
- T waves with maximum negativity in precordial leads in relation to III.
- In the case of being secondary to CLBBB, a duration not much greater than 120 ms is a constant, as well as repolarization in the left leads, not opposite to the preceding R waves.
- T loop of VCG in the case of CLBBB during CM is shown as rounded, unlike the T loop of uncomplicated CLBBB, which is fusiform or elongated.

The results showed a sensibility of 92% and specificity of 100% for the diagnosis of CM. Although not yet validated, this tool could be very helpful in the differentiation between a benign condition (CM) from another potentially severe, such as myocardial ischemia.

Altered mechanical load of the heart leads to ventricular hypertrophy, decompensated heart failure and fatal arrhythmias. Nevertheless, the molecular mechanisms that point to mechanic and electric dysfunction remain very little known. Increasing evidence suggest that ventricular electrical remodeling is a process that could be induced by altered mechanic stress, creating persistent electrophysiological alterations that predispose the heart to fatal arrhythmias. As to ventricular electrical remodeling, it is clearly a physiological property of the human heart manifest by T-wave memory and a variety of pathological states associated to altered ventricular activation, as bundle branch block, preexcitation, pacemaking, etc. Animal models that are being used to investigate ventricular electrical remodeling induced by stretching, present significant limitations. The model of the zebra-fish recently arose as an attractive animal model to study cardiovascular diseases and could overcome some of such limitations. The zebra-fish model could provide new clarifications on the molecular mechanisms that lead to negative electrical remodeling in response to stretching and mechano-electric feedback. The data suggest that the model of the zebra-fish is a powerful platform to investigate the molecular mechanisms of mechano-electric feedback in the heart (**Werdich 2012**).

1969 Dr. Kanu Chatterjee, the pioneer of cardiac memory phenomenon observer

In this year, Dr. Kanu Chatterjee et al (**Chatterjee 1969**) observed massive T-wave inversion and ST depression occurred in unpaced ECGs of 31 patients after ventricular pacing, whether endocardium or epicardium, and were related to the power of the artificial electric stimulus and not to the presence of the electrode alone. They continued as long as pacing was continued but were always reversible. They should not be mistaken for evidence of myocardium ischemia or underlying CAD. This phenomenon later (1982) was called "cardiac memory (CM)" by Rosenbaum school (**Rosenbaum 1982**).

Dr. Kanu Chatterjee (1934-2015)

Dr. Chatterjee was born in Bangladesh in 1934 and moved to Calcutta, India, as a refugee. He completed medical school at the R.G. Kar Medical College in Calcutta, India, while still living in a refugee camp, and moved to England in 1964 to complete his training in internal medicine and cardiology. At St. George's Hospital. He was the first to describe post pacing T wave changes on the ECG, an effect that was later termed CM.

He moved to Los Angeles and worked with Drs. Jeremy Swan and William Ganz in Cedars Sinai Medical Center and described, along with his colleagues, the hemodynamic subsets of acute coronary syndrome and provided insights into hemodynamically guided drug therapy. He was the first to report the beneficial effect of sodium nitroprusside in severe mitral regurgitation. The major portion of Dr. Chatterjee's career was spent at the University of California in San Francisco, where he taught several generations of innovative, successful cardiologists who became national and international leaders in their field. From 2001–2009 he directed the UCSF Chatterjee Center for Cardiac Research, named in his honor. He moved to Iowa in 2009 with his wife, Docey Edwards Chatterjee, an Iowa native. At the University of Iowa, he continued his teaching, clinical work, and writing as the first UI Kanu and Docey Edwards Chatterjee Chair in Cardiovascular Medicine. Dr. Chatterjee wrote more than 380 peer reviewed publications and 80 reviews and chapters relating to the management of patients with cardiovascular diseases and with the assistance of several Iowa colleagues, he edited the book *Cardiology – An Illustrated Textbook*. He received many teaching and achievement awards including the Gifted Teacher Award from the ACC, and the Lifetime Achievement Award of the Heart Failure Society of America. In 2014, he received the Herrick Award, the highest honor in clinical cardiology available from the American Heart Association.

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