ACUTE INFERIOR MYOCARDIAL INFARCTION SECONDARY TO PROXIMAL OBSTRUCTION OF RIGHT CORONARY ARTERY MIMICKING TYPE 1 BRUGADA PATTERN AND ATYPICAL LAMDA-LIKE WAVE IN INFERIOR LEADS

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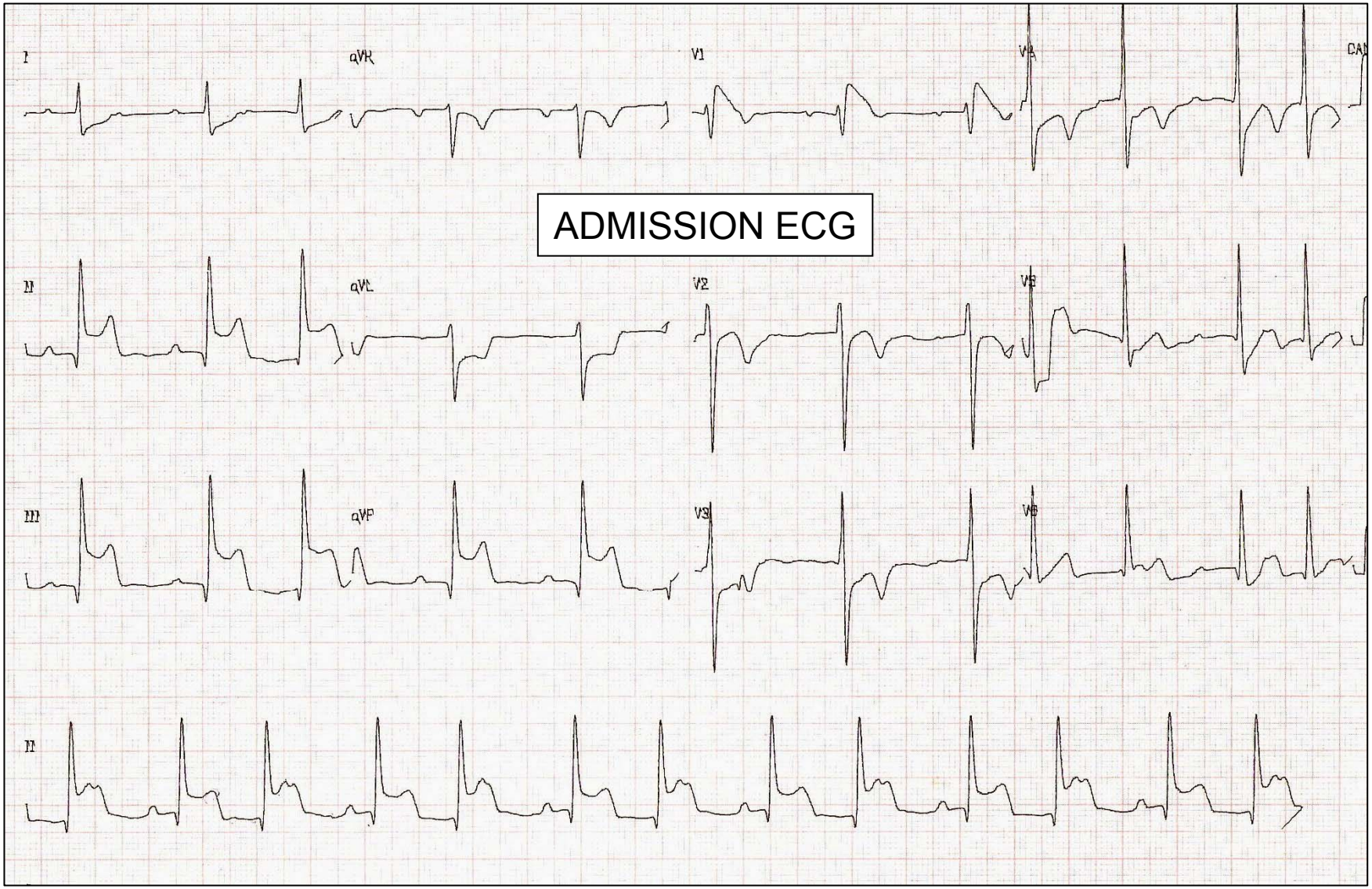
Case Report

- Male, 64-year-old patient, who was admitted with symptoms of chest pain typical of acute coronary syndrome.

- ECG (admission) shows a pattern of inferior AMI with RV compromise (confirmed by V4R) + second degree AVB, type I. Associate typical type 1 Brugada pattern in V1 lead. As there was no hemodynamic room available, the patient underwent fibrinolytic therapy with streptokinase (1 hour infusion).

- ECG (post STK) shows a pattern resembling that of a variant of Brugada Syndrome or atypical Brugada pattern: "Lambda like wave" in inferior leads.

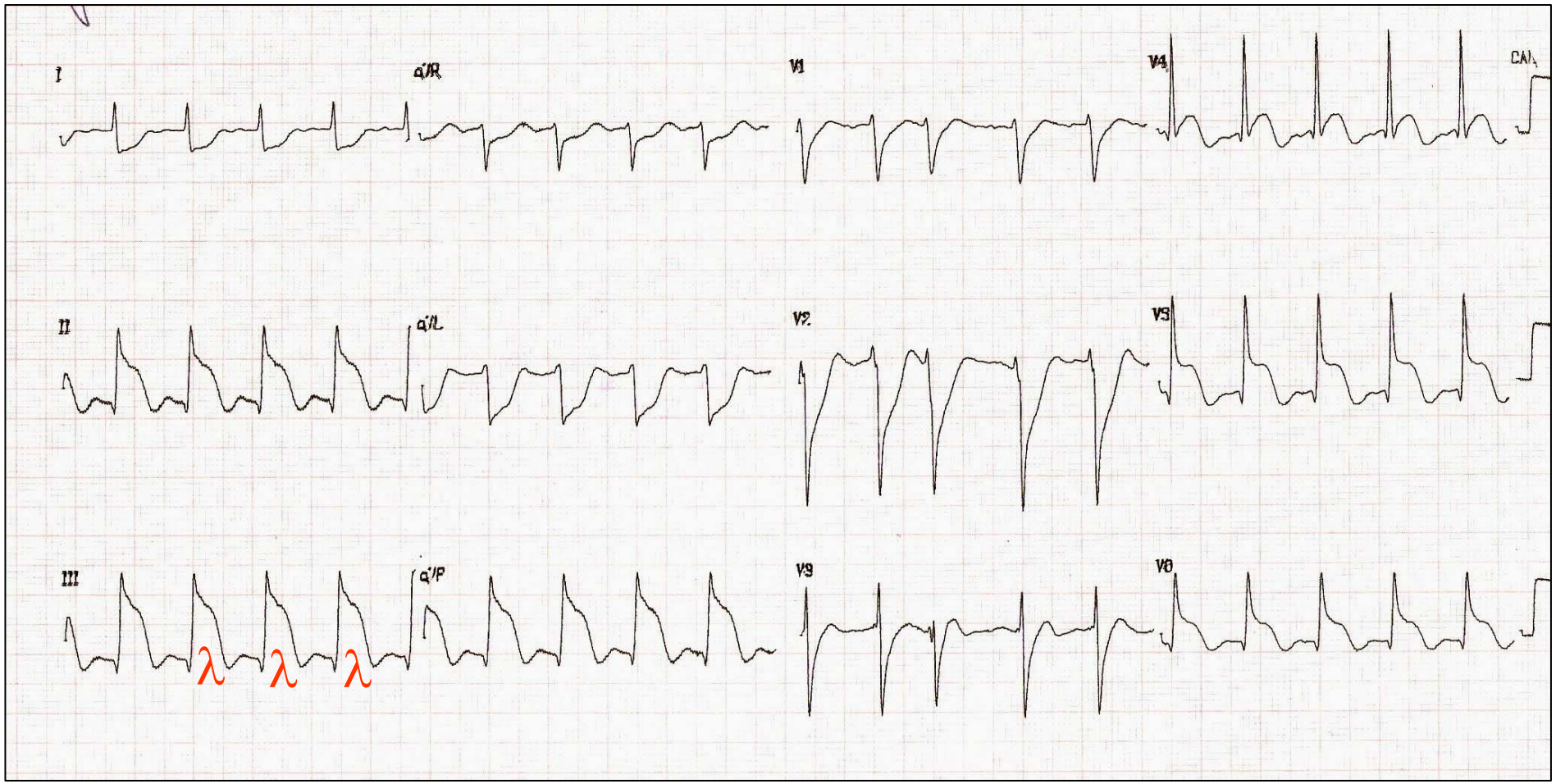
- Later, the patient underwent coronary angiography, which showed critical residual lesion of proximal RCA, which in turn was treated with STENT implant.



Right Coronary Artery occlusion proximal to the right ventricular side branches (=STsegment elevation in V1 Brugada like type 1ST-Segment elevation is a common ECG manifestation of acute transmural myocardial ischemia in leads facing the injury. Acute myocardial ischemia involving the RVOT of RV is known to induce a Brugada-like ECG pattern. Ito can modulate the ECG manifestation of acute ischemia as well as that of the Brugada syndrome, and that both clinical entities are the result of a similar electrophysiological substrate[1]. ST segment elevation in inferior leads. Mirror image in I, VL, Type I second degree AV block.

1) Di Diego JM, et al J of Electrocardiol. 2005;38(4 Suppl):14-17.

ECG POST-STREPTOKINASE



Characterization of proximal RCA occlusion in acute inferior MI

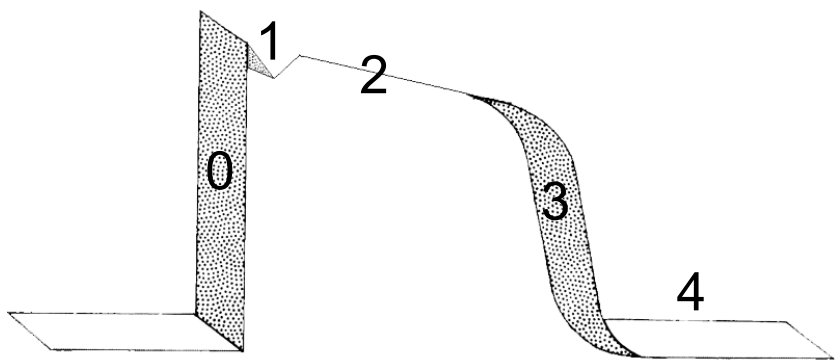
ST-segment depression in lead V1, The progress of the ischemic process like this, we consider the counteracting inferoposterior injury vectors to attenuate the ST-elevations caused by right ventricular involvement (=no ST elevation in V1). ST-segment depression in leads V1-V3, maximum ST-segment depression in the precordial leads, ST-segment depression in lead V3 of ≤ 50% of the magnitude of ST-segment elevation in lead III, absence or minimal of ST-segment depression in lead V1 in combination with ST-segment depression in lead V2 and the arithmetic sum of the ST-segment: III + V3 > 1, more ST elevation in the lateral precordial leads. Severe progression of ischemia (grade 3). Lamda-like wave in inferolateral wall: High risk for ventricular fibrillation.

Nomenclature challenge

- **1920**
  - Dr. Harold Pardee, from New York, publishes the first electrocardiogram of an acute myocardial infarction in a human and describes the T wave as being tall and "starts from a point well up on the descent of the R wave[1]
  - “The Pardee complex”. This is observed also during the hyperacute early injury phase of MI (next slide). This complex looks like the monophasic action potential of rapid fibers.

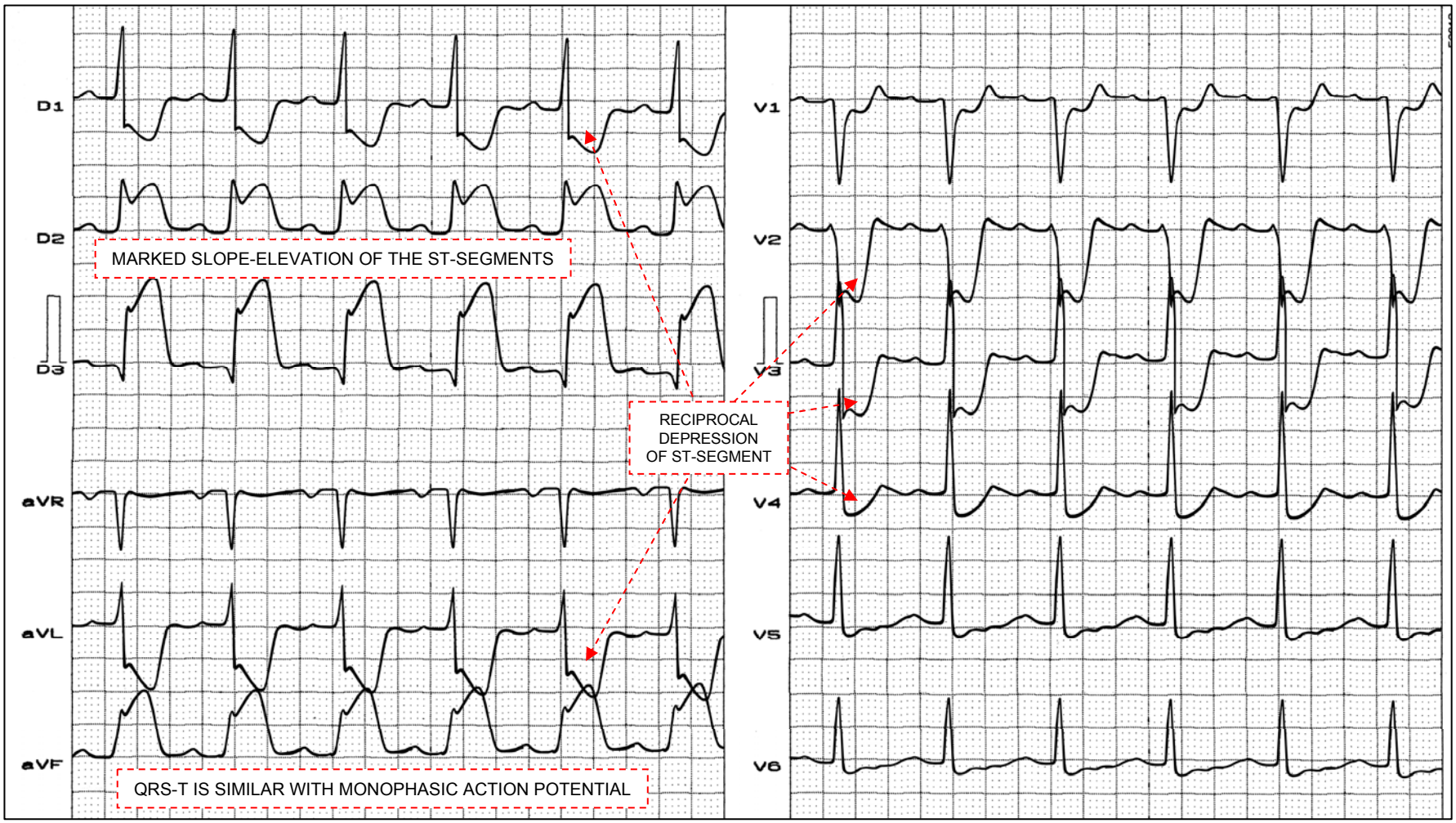
OUR CASE (2nd ECG)

MONOPHASIC ACTION POTENTIAL OF RAPID FIBERS



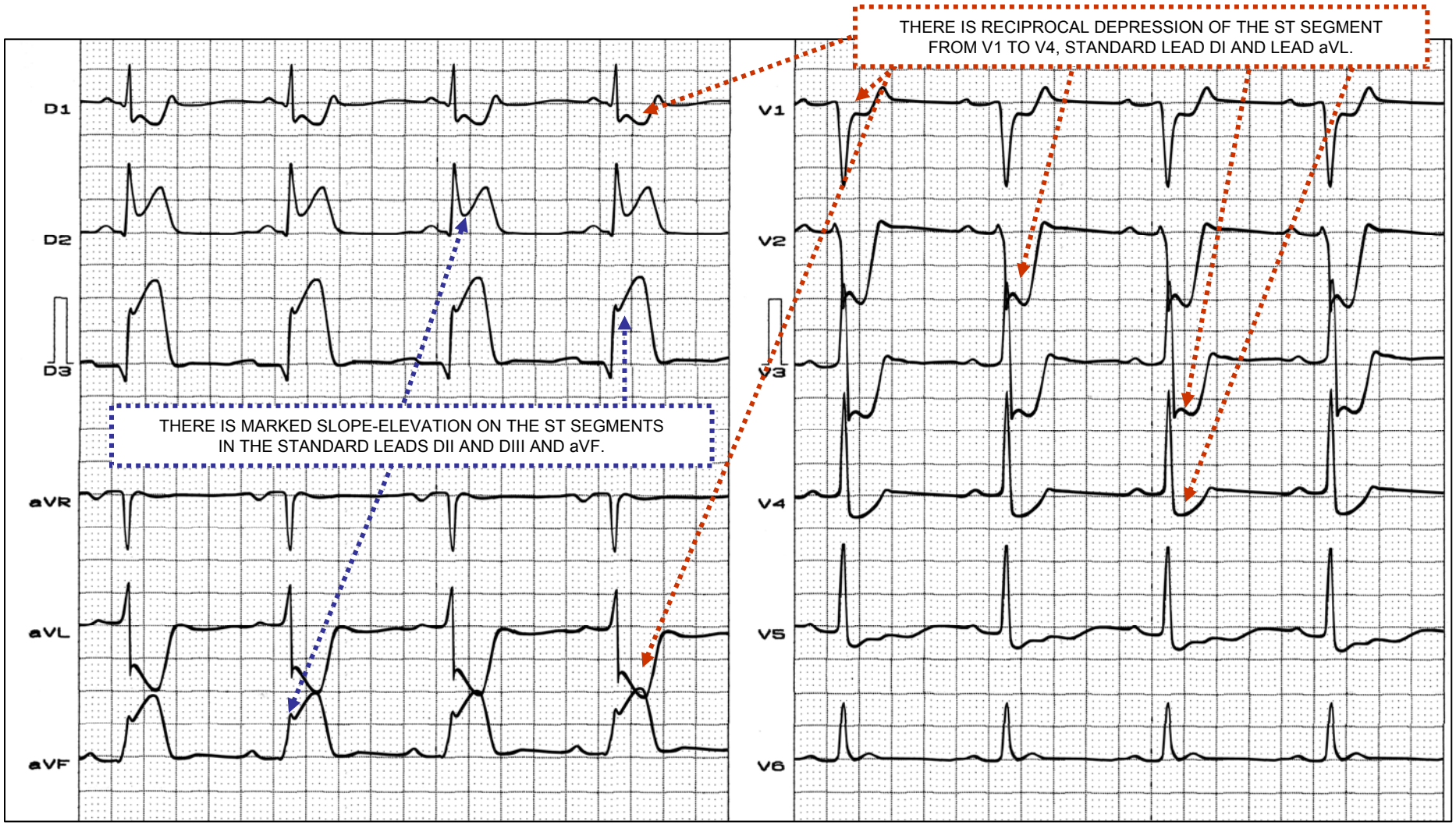
1) Pardee HEB. An electrocardiographic sign of coronary artery obstruction. Arch Int Med 1920;26:244-257.

THE ‘HYPERACUTE EARLY INJURY PHASE’ IN INFERIOR MYOCARDIAL INFARCTION



Man, 55 years old, 3 hours onset chest pain, first ECG in his home.

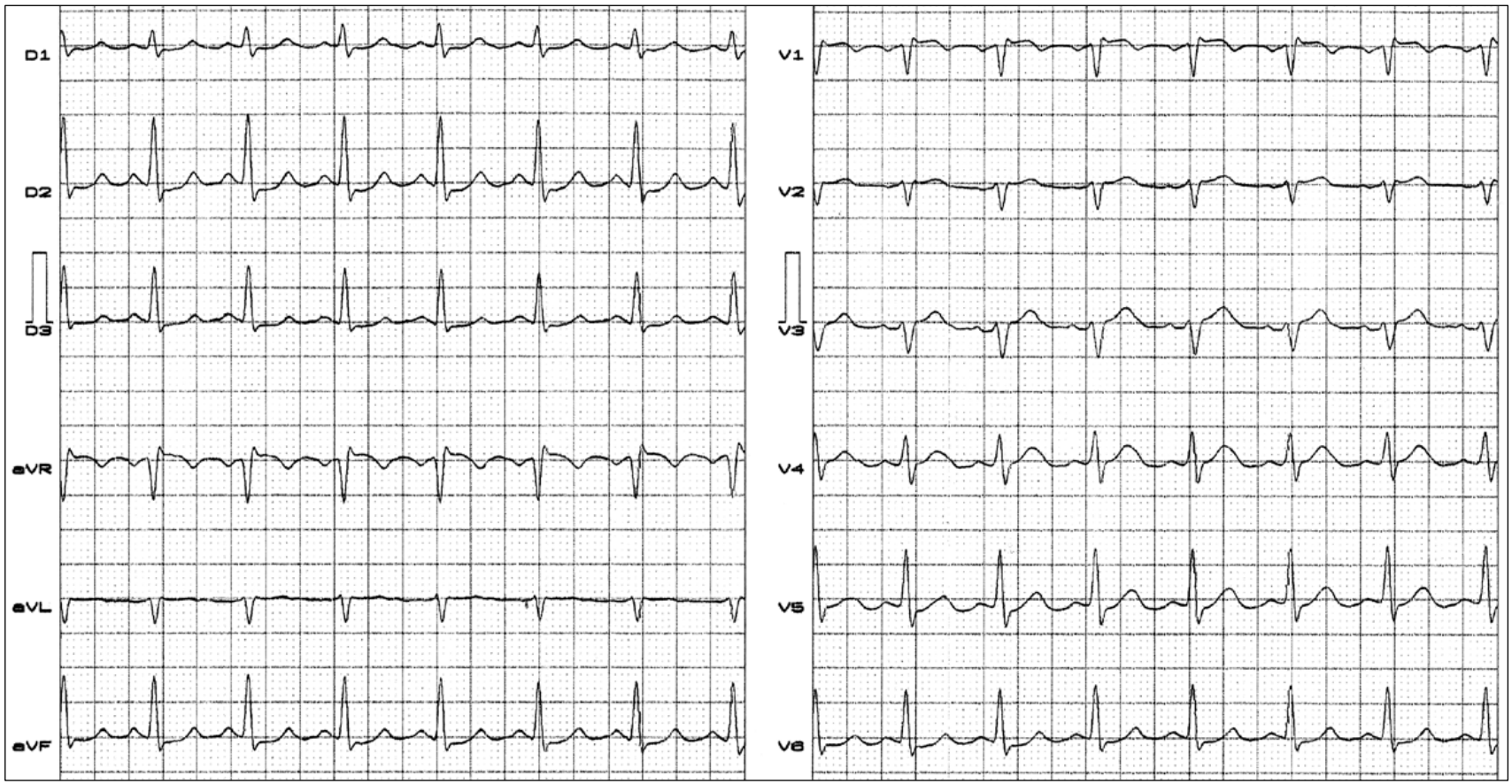
THE ‘HYPERACUTE EARLY INJURY PHASE’ IN INFERIOR MYOCARDIAL INFARCTION



There is a increased in VAT with delay in the inscription of the ID (60ms in DIII and aVF).

ERGOMETER TEST: STAGE 1 ONSET OF STRAIN

NAME: P. R. T **DATE:** 04/11/2002 **AGE:** 48 Y. **NUMBER:** 616
SEX: M. **RACE:** M. **WEIGHT:** 82 Kg . **HEIGHT:** 1.84 m. **BIOTYPE:** ATHLETIC
MEDICATION IN USE: NOTHING STATED.

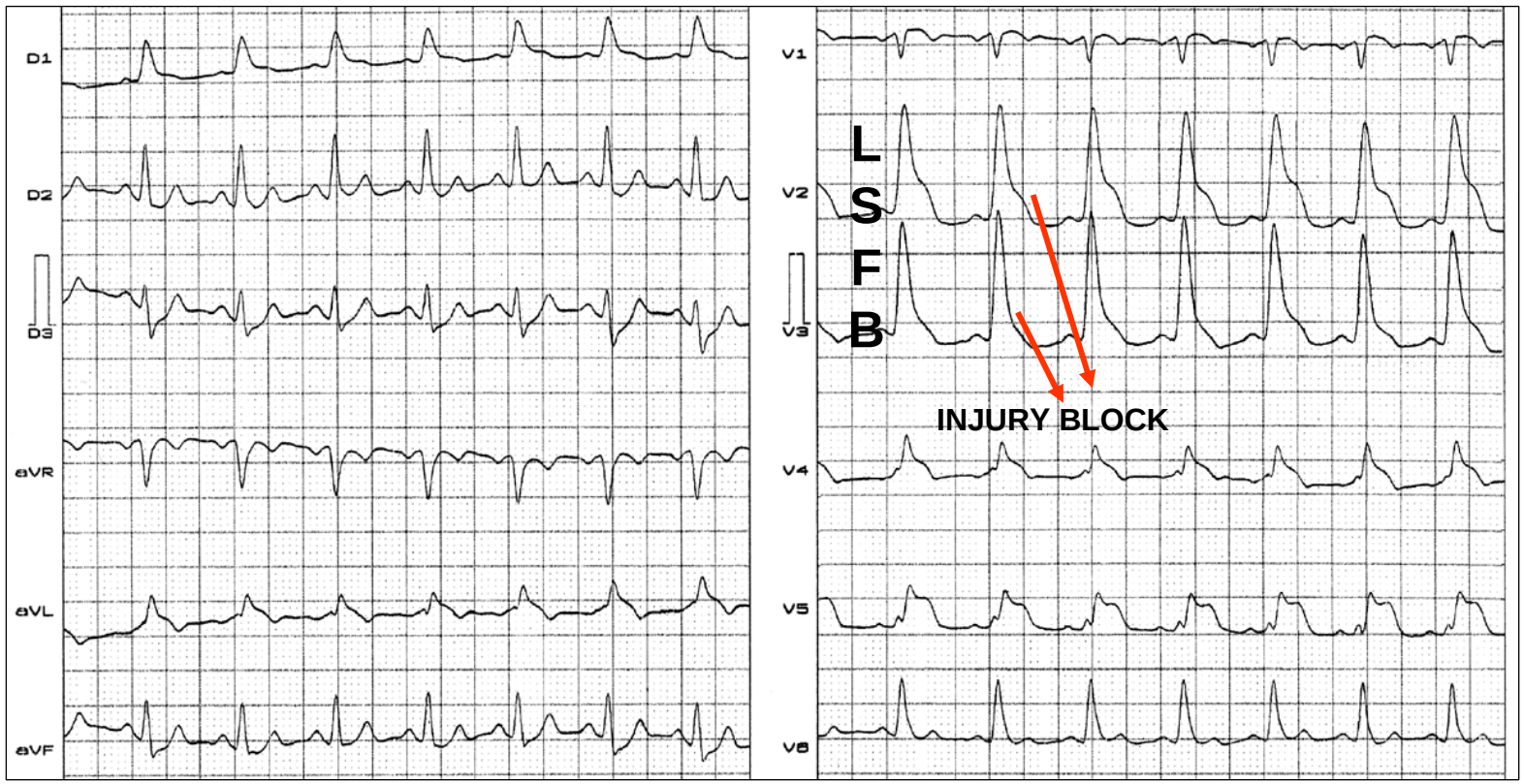


Clinical Diagnosis: angor pectoris of strain, inveterate smoker (30 cigarettes per day), stress. Absence of diabetes, high blood pressure, dyslipidaemias and others.
ECG diagnosis: tracing of onset of strain, HR 109 bpm, discrete end conduction delay by one of the RB fascicles: aVR and V_1 Qr.

ERGOMETER TEST

STAGE 2 INTRASTRAIN: INJURY BLOCK ASSOCIATED TO LSFB

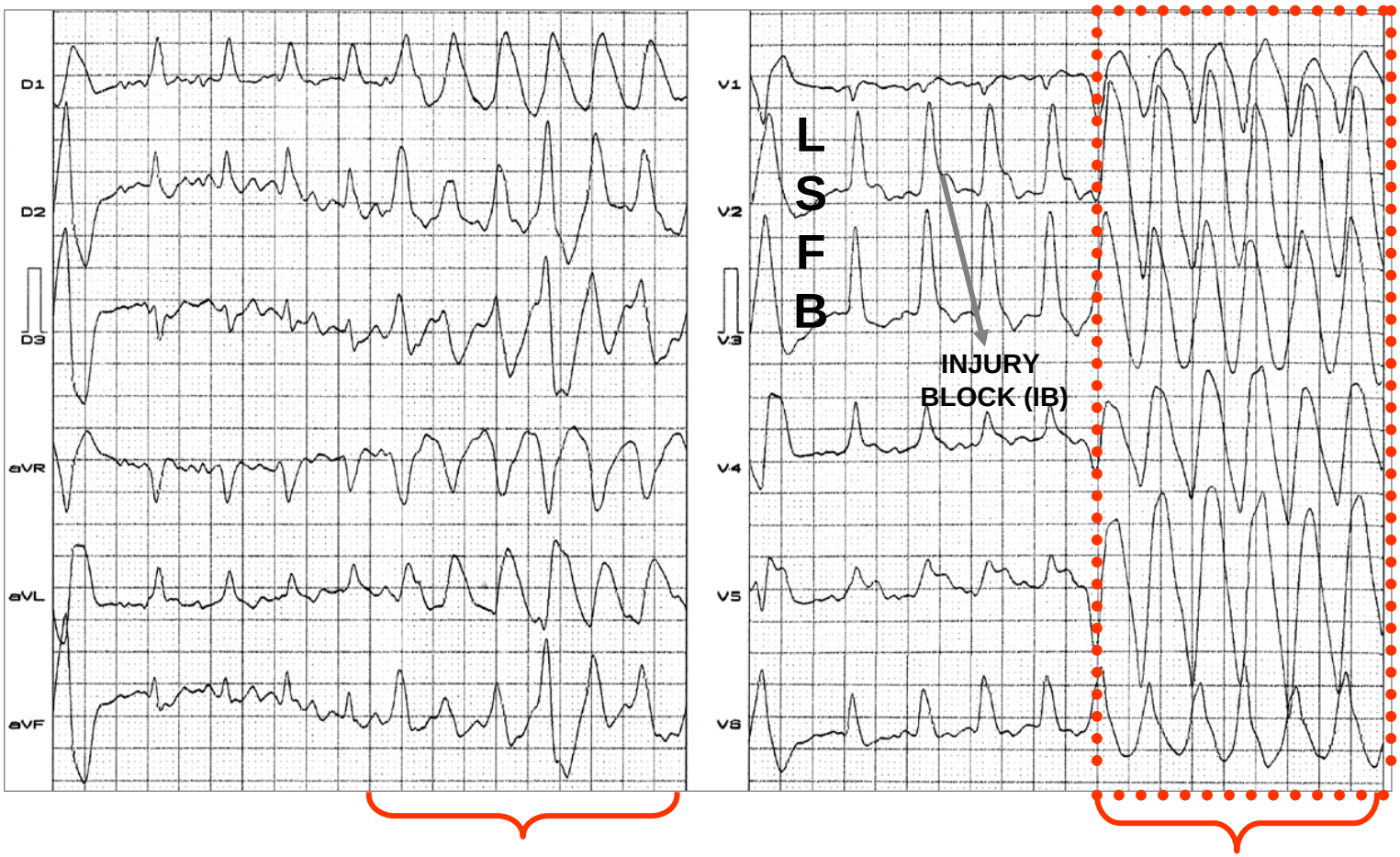
NAME: P. R. T **DATE:** 04/11/2002 **AGE:** 48 Y. **NUMBER:** 616
SEX: M. **RACE:** M. **WEIGHT:** 82 Kg . **HEIGHT:** 1.84 m. **BIOTYPE:** ATHLETIC
MEDICATION IN USE: NOTHING STATED.



In the second stage of the ergometer test, the typical pattern known as injury block (IB) appeared in the anterior wall, which is characterized by the distortion of the terminal portion of the QRS complex. This IB is characterized by the emergence of the J point at a level above the inferior half of the R wave, the disappearance of the S wave in leads with RS configuration, as in this case V_2 and V_3. Besides, a significant increase of R wave voltage is observed in V_2 and V_3 prominent anterior forces (PAF), indicating the appearance of LSFB. The hemodynamic study revealed proximal critical injury of the ADA before the first septal perforating artery.

ERGOMETER TEST
STAGE 3 INTRASTRAIN: LSFB + IB + VT

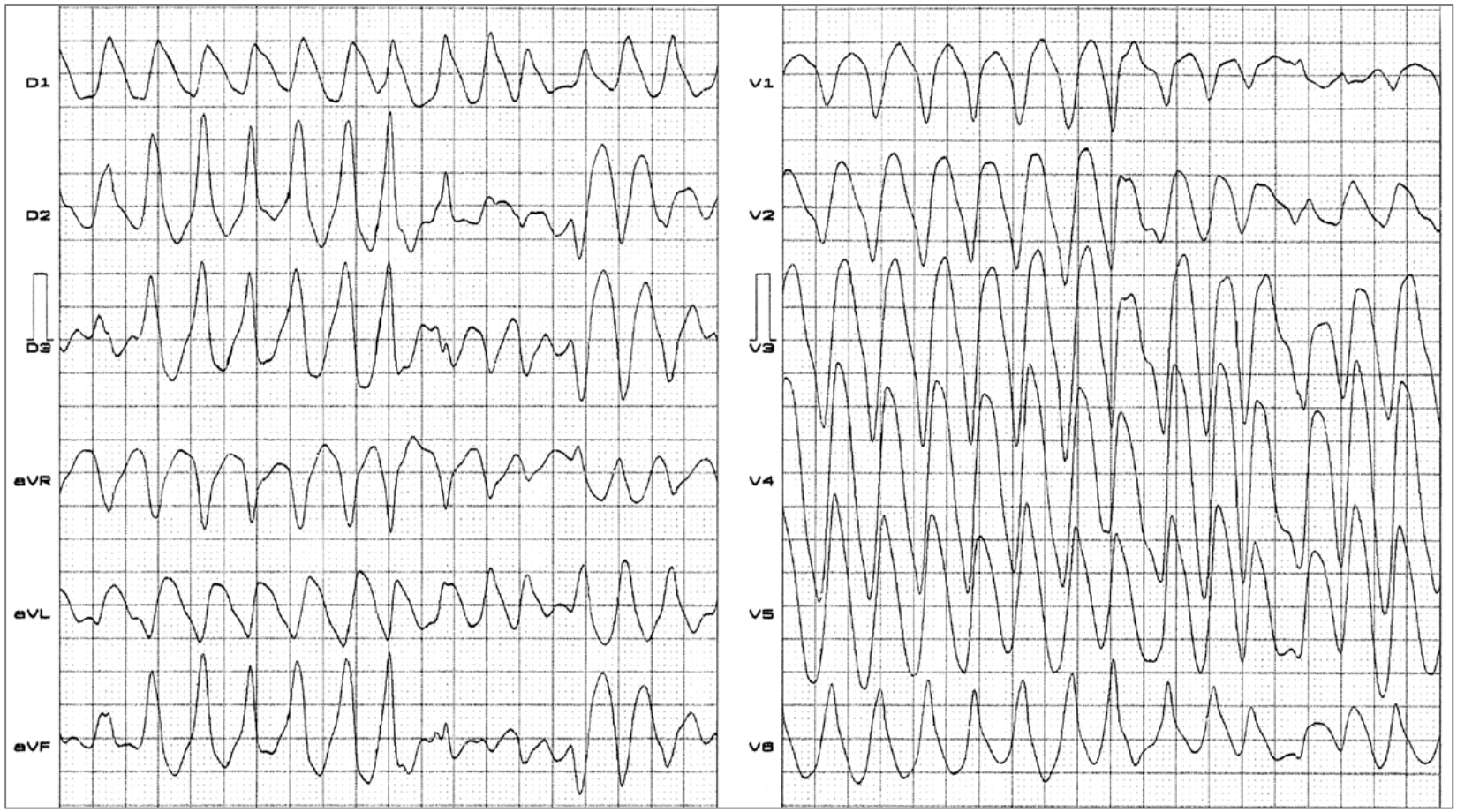
NAME: P. R. T **DATE:** 04/11/2002 **AGE:** 48 Y. **NUMBER:** 616
SEX: M. **RACE:** M. **WEIGHT:** 82 Kg . **HEIGHT:** 1.84 m. **BIOTYPE:** ATHLETIC



Tracing of a patient during the stress test, which shows LSFB in the sequence, with prominent anterior forces and injury block followed by a run of monomorphic sustained ventricular tachycardia, which disappears at recovery.

ERGOMETER TEST
STAGE 4 INTRASTRAIN: VT

NAME: P. R. T **DATE:** 04/11/2002 **AGE:** 48 Y. **NUMBER:** 616
SEX: M. **RACE:** M. **WEIGHT:** 82 Kg . **HEIGHT:** 1.84 m. **BIOTYPE:** ATHLETIC

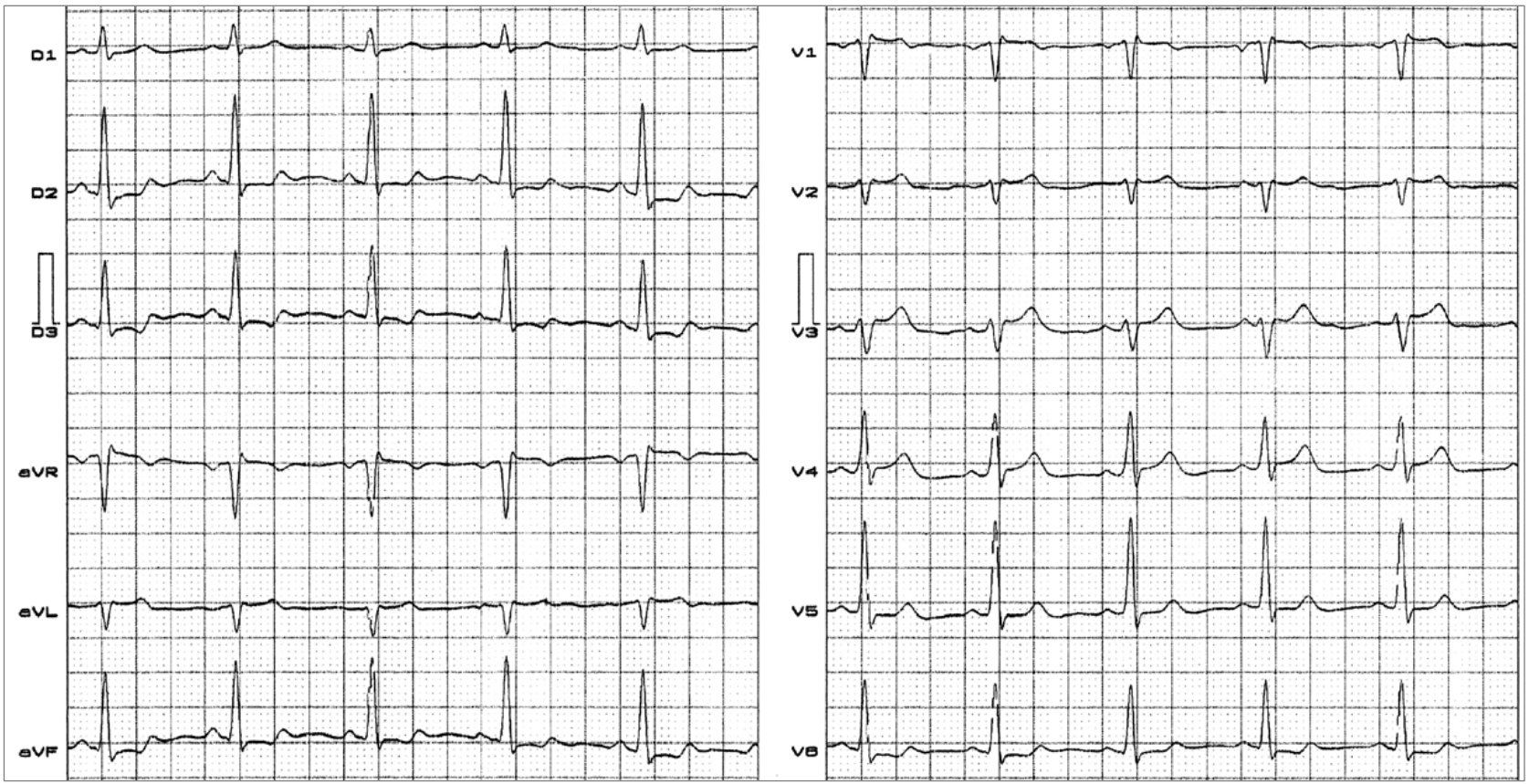


Tracing of a patient during the stress test, which shows LSFB in the sequence, with prominent anterior forces and injury block followed by a run of monomorphic sustained ventricular tachycardia, which disappears at recovery.

ERGOMETER TEST

STAGE 5 2-MINUTE RECOVERY:
NORMALIZATION, THE PAF BY LSFB AND THE VT DISAPPEARED

NAME: P. R. T **DATE:** 04/11/2002 **AGE:** 48 Y. **NUMBER:** 616
SEX: M. **RACE:** M. **WEIGHT:** 82 Kg . **HEIGHT:** 1.84 m. **BIOTYPE:** ATHLETIC

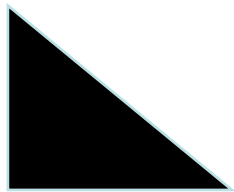


CONCLUSIONS: after 2 minutes of interruption of strain, the run of VT disappears (NS-VT), as well as the injury block (IB) and the prominent anterior forces (PAF) secondary to the left septal fascicular block (LSFB). This case reveals irrefutably, the intermittent form of LSFB. The phenomenon os intermittence has a great diagnostic value, since it rules out the possibility of other causes for the PAF.

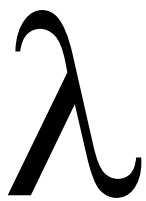
PROPOSAL OF CLASSIFICATION OF TYPE 1 BRUGADA ECG PATTERN

TYPE 1A: COVED SHAPE OR “BULL TERRIER” WAVE

TYPE 1B: TRIANGULAR SHAPE



TYPE 1C: “LAMBDA” (λ) OR GUSSAK WAVE

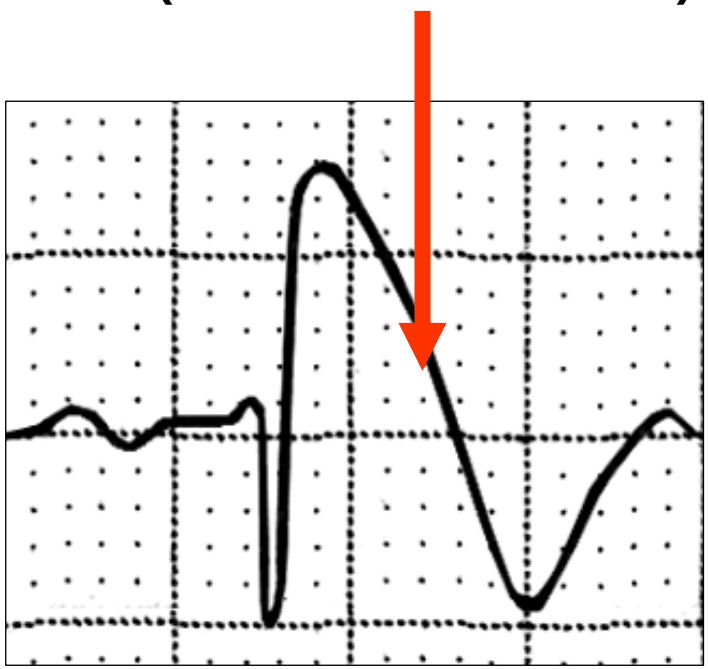


GREEK SMALL
LETTER LAMBDA

PROPOSAL OF CLASSIFICATION OF TYPE 1 BRUGADA ECG PATTERN

TYPE 1A

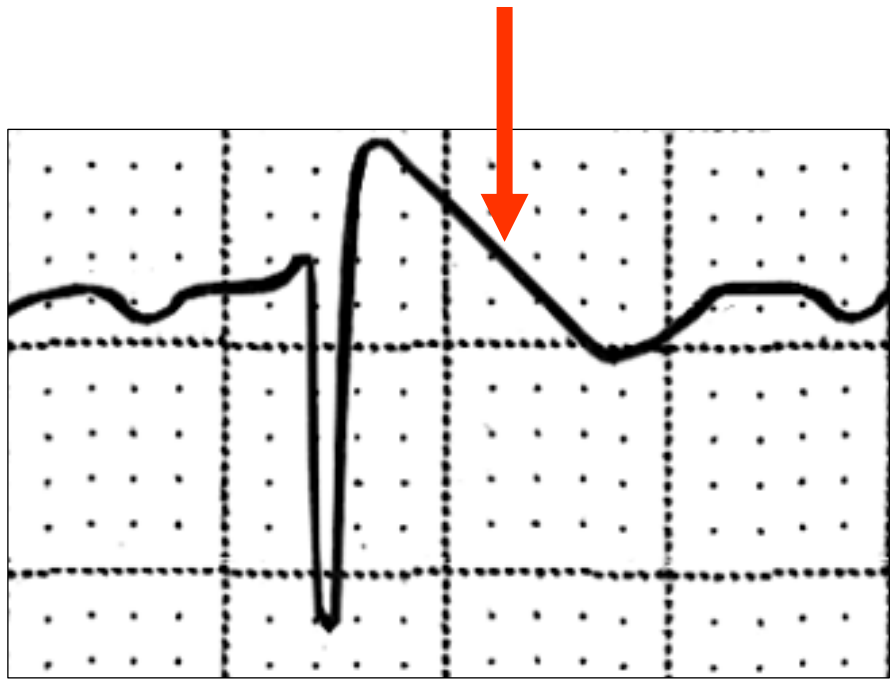
COVED TYPE
(CONVEX TO THE TOP)



V_1 to V_2 or V_3

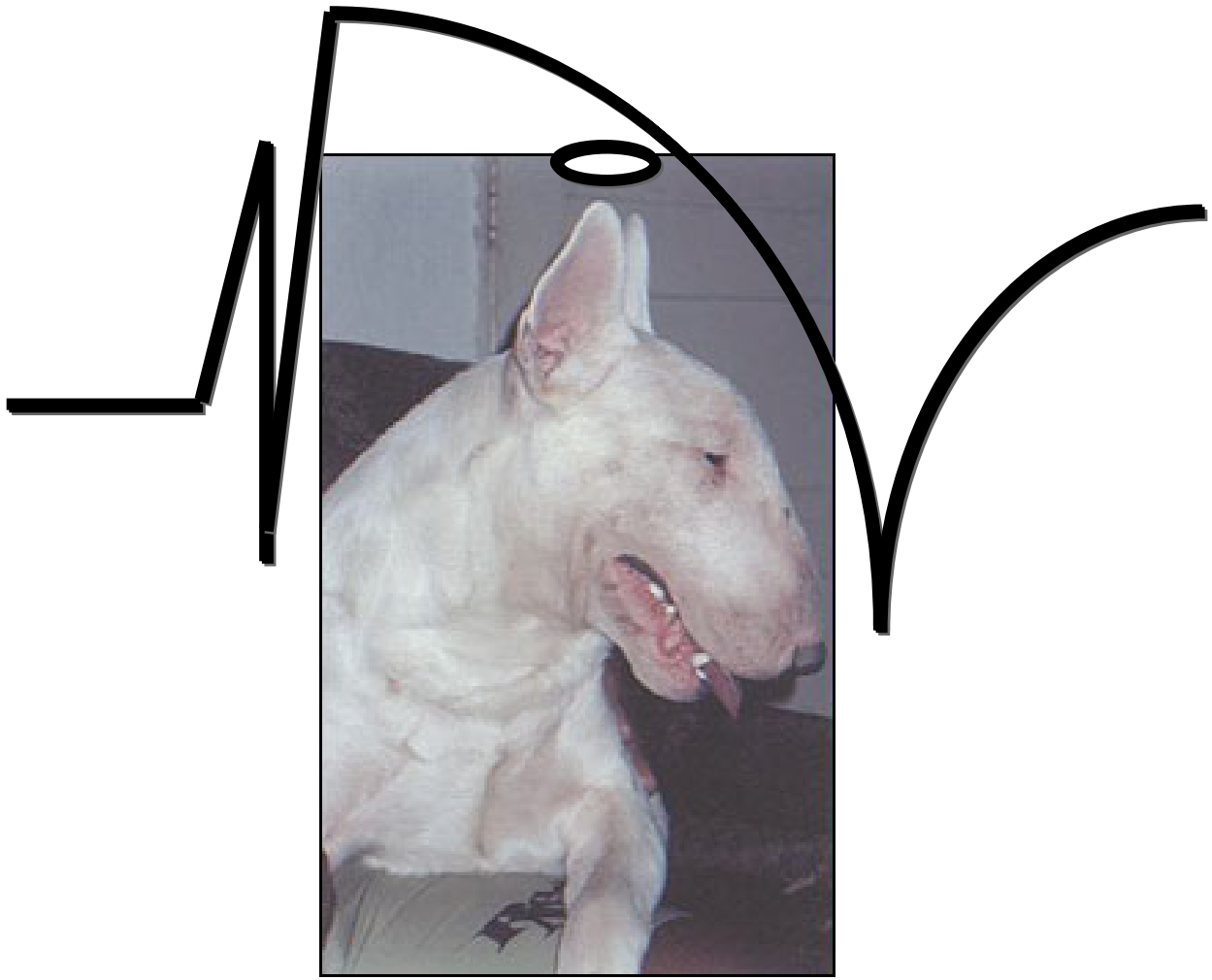
TYPE 1B

TRIANGULAR SHAPE

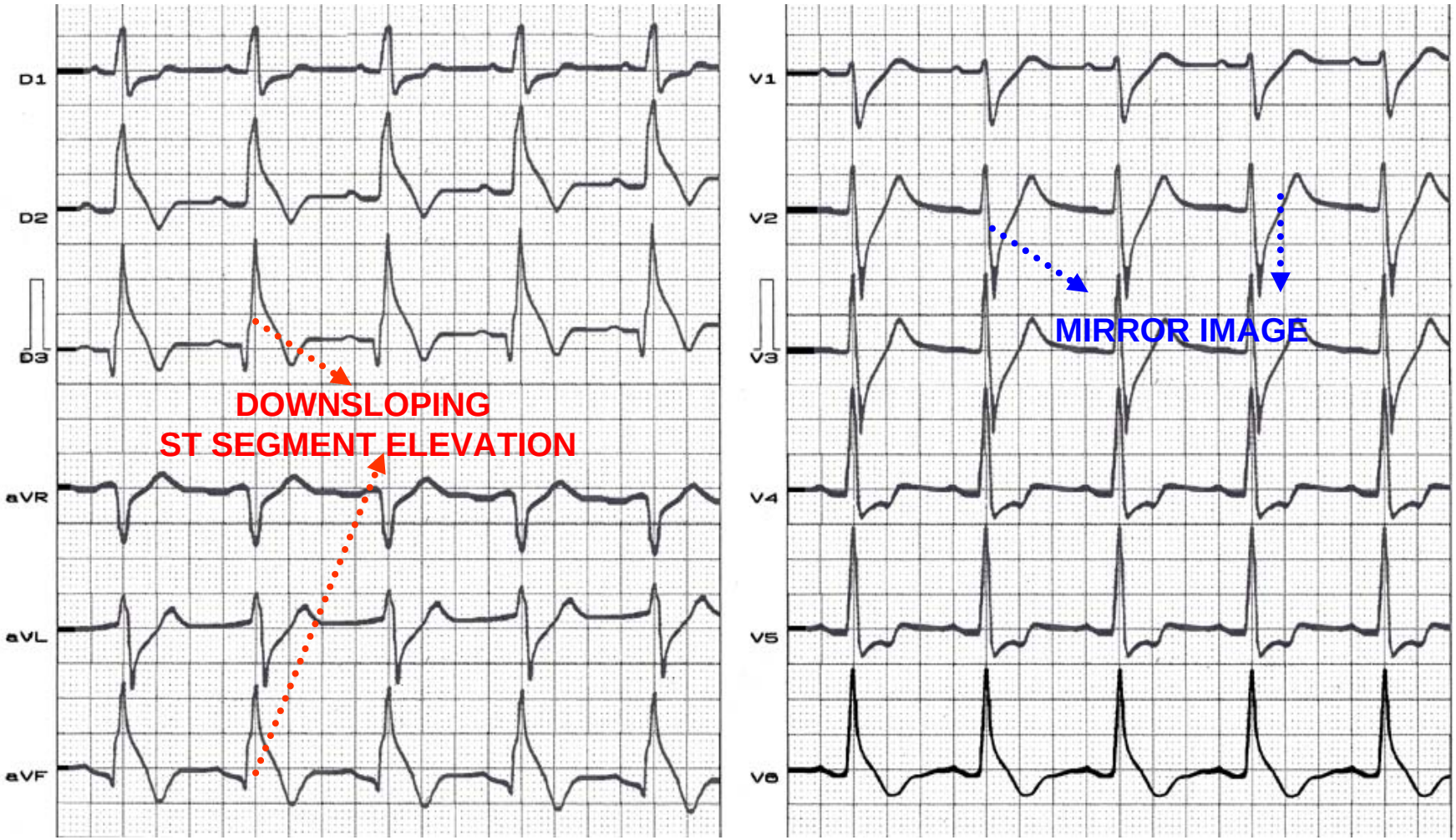


V_1 to V_2 or V_3

“BULL TERRIER” OR TYPE 1A BRUGADA PATTERN
CONVEX TO THE TOP

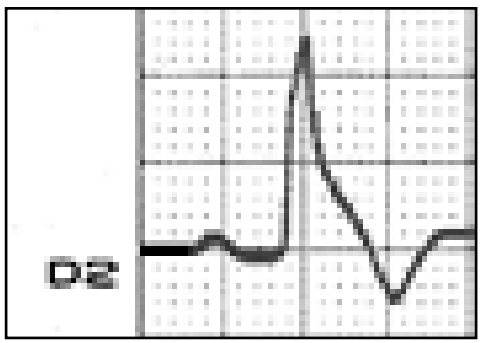


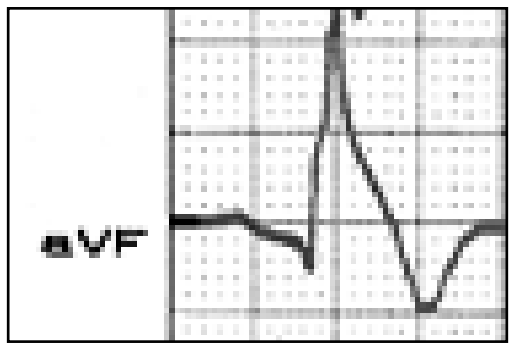
PROPOSAL OF CLASSIFICATION OF TYPE 1C BRUGADA ECG PATTERN

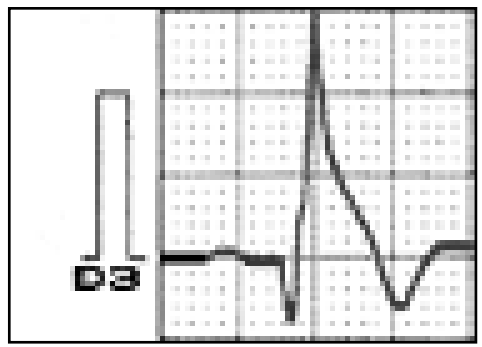


Riera AR, et al. J Electrocardiol 2004; 37:101-104.
Kukla P, et al. Kardiol Pol. 2008;66:873-877

TYPE 1C WAS DENOMINATED “LAMBDA” WAVE BY GUSSAK I ET AL







λ

Type 1C: ST-segment elevation is triangular or coved to the top (“coved type”) ≥2mm (0.2mV), and followed by negative T wave located in inferior leads.

Gussak I, et al. J Electrocardiol 2004;37:105-107.

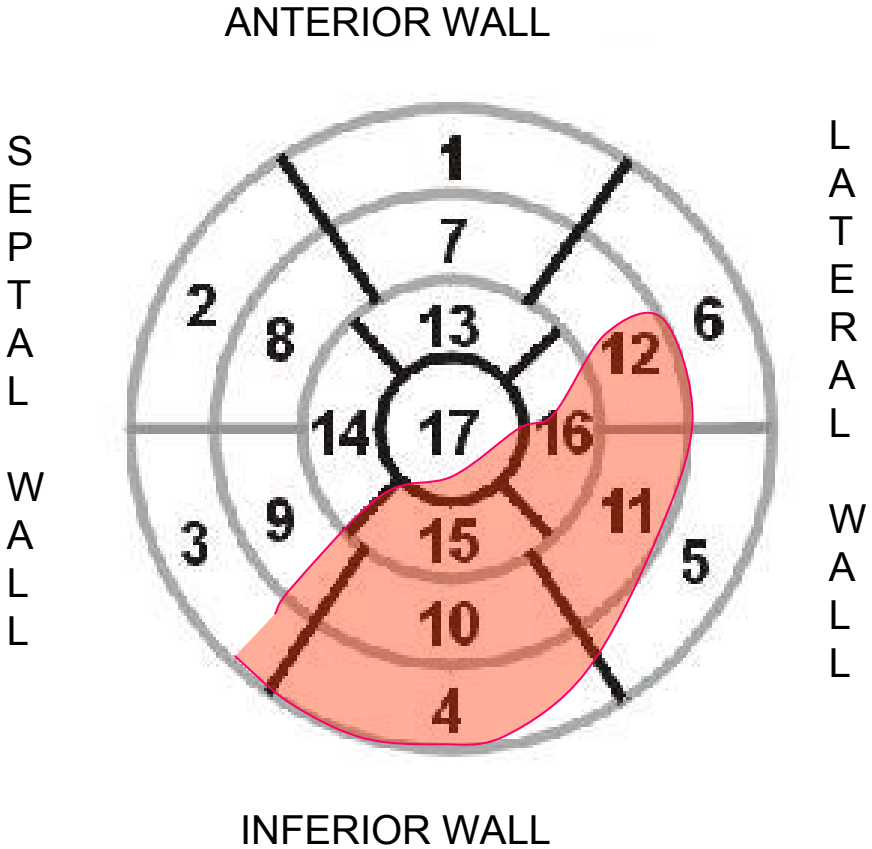
NEW ELECTROCARDIOGRAPHIC TERMINOLOGY FOR Q-WAVE INFARCTIONS BASED ON THE CORRELATION WITH CE-CMR

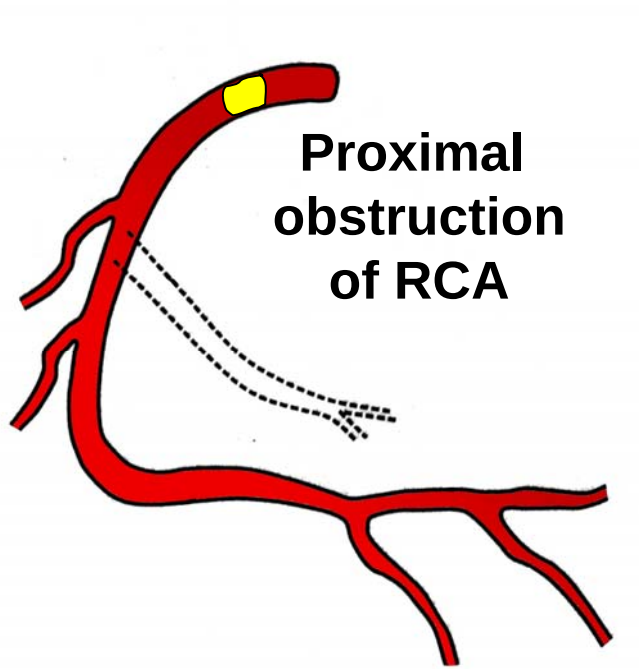
INFEROLATERAL ZONE

- **Inferolateral**
- **Type:** B-3
- **Most likely site of occlusion:** RCA or dominant LCx
- **ECG pattern:** signs of inferior (Q in II, II, VF: B2) and/or lateral infarction (RS in V_1).
- **Segments compromised by infarction in CE-CMR:** image in the next slide.
- **SE:** 73%.
- **SP:** 98%.

1) Bayés de Luna A, et al.Am J Cardiol. 2006;97:443-451.
2) Bayés de Luna A, et al. Circulation 2006; 114:1755-1760.
3) Bayés de Luna A, et al. J Electrocardiol. 2006; 39 (4 Suppl):S79-81.
4) Bayés de Luna A, et al. J Electrocardiol. 2007;40:69-71.
5) Bayés de Luna A,et al. Ann Noninvasive Electrocardiol. 2007; 12:1-4.
6) Bayés de Luna A, et al. Cardiology Journal 2007;14 : 417-419.
7) Cino JM, et al. J Cardiovasc Magn Reson. 2006;8:335-44.
8) Pons-Lladó G, et al. J Cardiovasc Magn Reson. 2006;8:325-6.

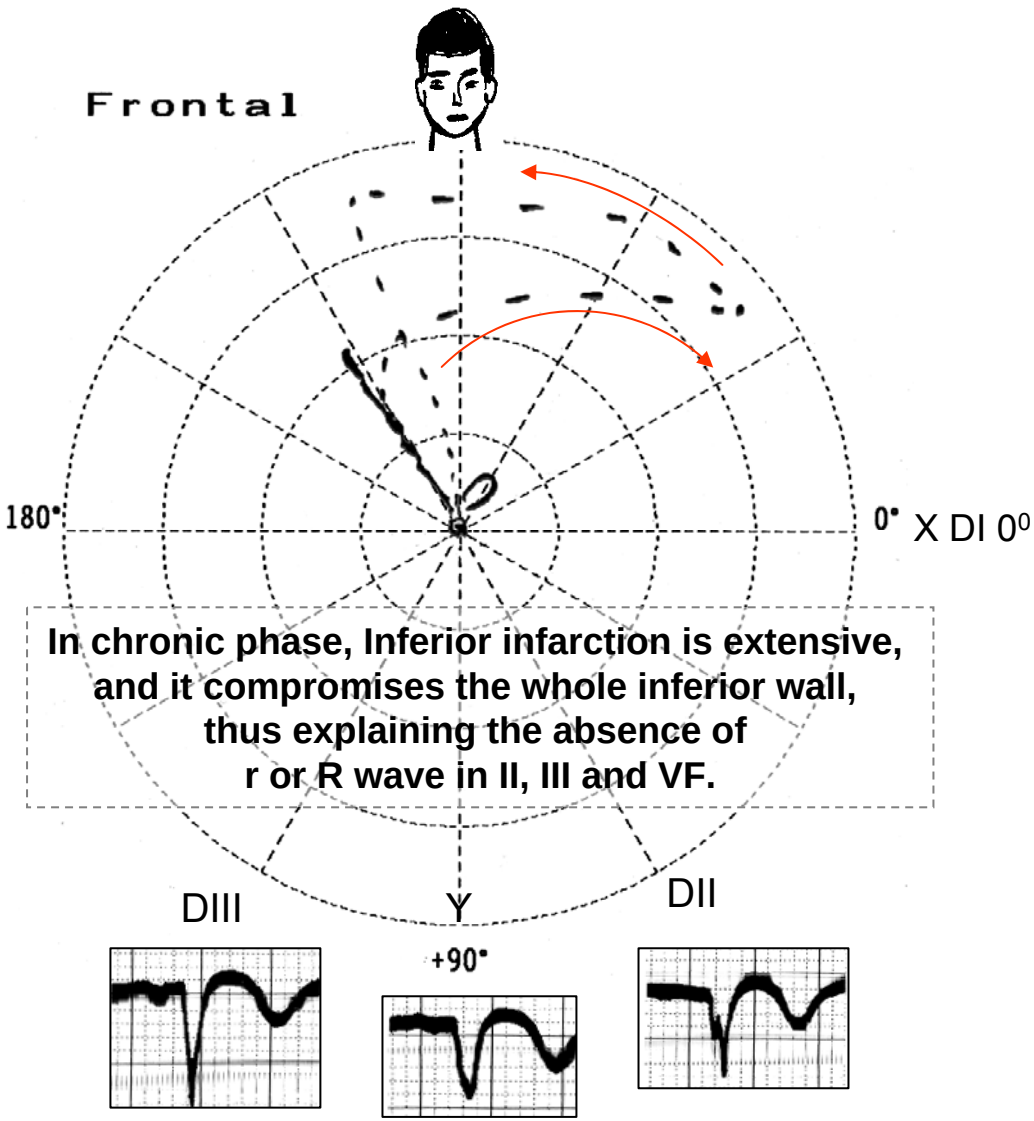
LATERAL INFARCTION
B-3





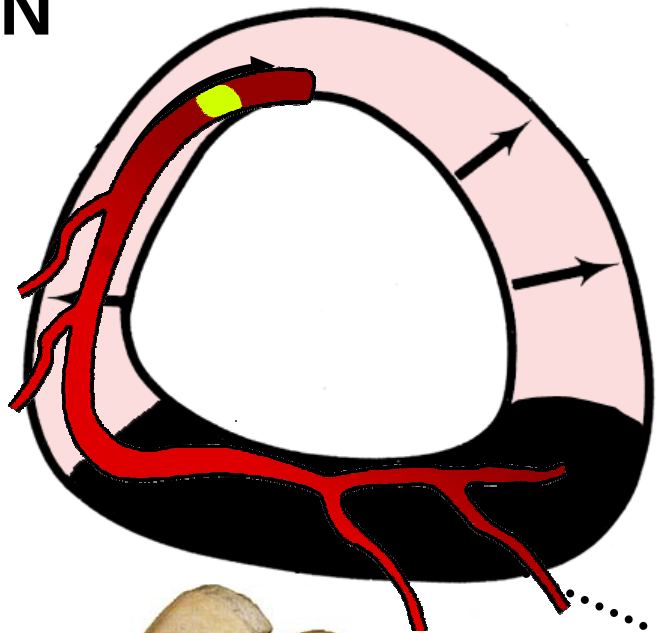
ECG pattern: signs of inferior (Q in II, II, VF: B2) and/or lateral infarction (RS in V1).

INFERO-LATERAL INFARCTION

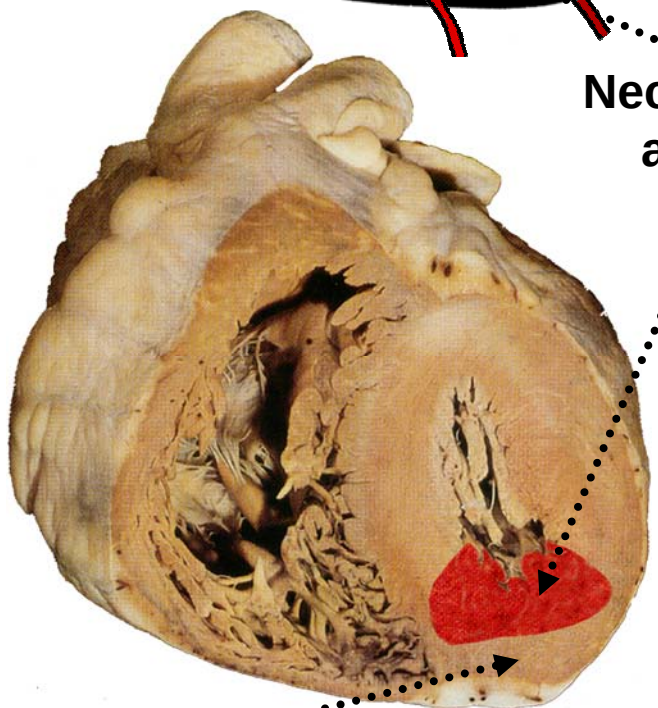


In chronic phase, Inferior infarction is extensive, and it compromises the whole inferior wall, thus explaining the absence of r or R wave in II, III and VF.

QS in the three inferior leads



Necrosis area



Apical region

ECG criteria for identifying patients with acute inferior MI (ISTEMI) caused by proximal obstruction of the RCA[1]

1) ST-segment depression in lead V1
2) ST-segment depression in leads V1-V3
3) Maximum ST-segment depression in the precordial leads
4) ST-segment depression in lead V3 of ≤ 50% of the magnitude of ST-segment elevation in lead III
5) The absence of ST-segment depression in lead V1 in combination with ST-segment depression in lead V2
6) The arithmetic sum of the ST-segment: III + V3 > 1.
7) The arithmetic sum of the ST-elevation in V3/ST-elevation in III < 0.5

1) Styliadis I, Ziakas A, Karvounis H, Giannakoulas G, Efthimiadis GK, Parisiadou A, Anifanti M, Dalamanga E, Parcharidis G, Louridas GThe utility of the standard 12-lead electrocardiogram in the prediction of proximal right coronary artery occlusion in acute inferior myocardial infarction. J Emerg Med. 2008;35:67-72

Inferior wall acute myocardial infarction ECG characterization[1]

- In inferior wall acute myocardial infarction patients with maximum ST depression in leads V4 to V6 more often had 3-vessel disease than those without precordial ST depression or those with ST depression in leads V1 to V3, and they had a lower EF.
- ST depression in leads V4 to V6, but not V1 to V3, confers a greater likelihood of multivessel coronary artery disease.
- Patients with maximum ST depression in leads V1 to V3 less often had AMIs due to proximal RCA obstruction than patients without precordial ST depression or those with ST depression in leads V4 to V6 and had larger AMIs as estimated by peak creatine kinase.
- Different patterns of precordial ST depression are associated with distinctive coronary anatomy.

1) Birnbaum Y, Wagner GS, Barbash GI, Gates K, Criger DA, Sclarovsky S, Siegel RJ, Granger CB, Reiner JS, Ross AM. Correlation of angiographic findings and right (V1 to V3) versus left (V4 to V6) precordial ST-segment depression in inferior wall acute myocardial infarction. Am J Cardiol. 1999;83:143-148.

Acute inferior myocardial infarction with right ventricular involvement[1]

- The ST changes in acute inferior myocardial infarction (AIMI) give indications regarding the site, extension, and extent of AIMI.
- RV involvement can mask posterior extension, points to the right coronary as the culprit vessel (100%), and, with high probability, indicates the proximal segment as the site of the lesion.
- The ECG signs of isolated AIMI indicate a peripheral obstruction; and a collateral circulation may appear relatively early.

1) Correale E, Battista R,. Martone A, Pietropaolo F, Ricciardiello V, DiGirolamo D, Barlera S, Maggioni AP.Electrocardiographic patterns in acute inferior myocardial infarction with and without right ventricle involvement: classification, diagnostic and prognostic value, masking effect. Clin Cardiol. 1999;22:37-44

Proximal RCA and LCx obstruction [1]

- Significant ST segment depression (ST≥ 1 mm) in leads I and aVL is more common in RCA obstruction associated inferior wall MI with a sensitivity of 70% and 100%, and a specificity of 63% and 38%, respectively.
- The absence of significant ST segment depression in lead VL is most common in proximal left circumflex (LCx) obstruction, with a similar trend for lead I.
- ST segment depression patterns in leads V5 and V6 is not indicative of the infarct-related artery or the site of obstruction. The lack of ST segment depression in these leads indicates proximal LCx with a sensitivity of 71% and 86%, and a specificity of 65% and 100%, respectively.

1) Hasdai D, Birnbaum Y, Herz I, Sclarovsky S, Mazur A, Solodky A.ST segment depression in lateral limb leads in inferior wall acute myocardial infarction. Implications regarding the culprit artery and the site of obstruction. Eur Heart J. 1995;16:1549-1553

Clinical conditions mimicking STEMI in patients referred for primary PCI[1]

1) Coronary aneurysm
2) Myo/pericarditis
3) Cardiomyopathy
4) Brugada syndrome
5) Aortic stenosis
6) Aortic dissection
7) Subarachnoidal haemorrhage
8) Pneumonia
9) Chronic obstructive pulmonary disease
10) Mediastinal tumour
11) Peritonitis after recent abdominal surgery.

1) Gu YL, Svilaas T, van der Horst IC, Zijlstra F. Conditions mimicking acute ST-segment elevation myocardial infarction in patients referred for primary percutaneous coronary intervention. Neth Heart J. 2008;16:325-331

ACQUIRED FORMS OF THE BRUGADA SYNDROME; PSEUDO-ECG BRUGADA PATTERN OR PSEUDO BRUGADA PHENOTYPE[1]

- Experimental studies have suggested that an intrinsically prominent transient outward current (Ito)-mediated action potential (AP) notch and a subsequent loss of AP dome in the epicardium, but not in the endocardium of the RVOT, give rise to a transmural voltage gradient, resulting in ST segment elevation in leads V1 - V3 and induction of subsequent VF due to the mechanism of phase 2 reentry.

- Because the maintenance of the AP dome is determined by the balance of currents active at the end of phase 1 of the AP, any interventions that increase outward currents (e.g. Ito, adenosine tri-phosphate sensitive potassium current [IK-ATP], slow and fast activating components of delayed rectifier potassium current [IKs, IKr]) or decrease inward currents (e.g. L-type calcium current [ICa-L], fast sodium current [INa]) at the end of phase 1 of the AP can accentuate or unmask ST segment elevation, similar to that found in Brugada syndrome.

- A number of drugs and conditions, which cause an outward shift in current active at the end of phase 1, have been reported to induce transient Brugada-like ST segment elevation. This is the so-called “acquired” form of Brugada syndrome similar to the “acquired” form of LQTS.

1) Shimizu W. Acquired forms of the Brugada syndrome. J Electrocardiol. 2005; 38:22-25

CONDITIONS THAT CAN LEAD TO ST SEGMENT ELEVATION IN THE RIGHT PRECORDIAL LEADS

- Young ECG pattern[1]
- Technical problem of inertia with the recording device[2]
- Acute phase of myocardial infarction[3-4]
- Acute myocardial ischemia[5-6]
- Dissecting aortic aneurysm[7]
- Acute pulmonary embolism[8]
- Hypothermia[9]

1) Rosso R, et al. J Am Coll Cardiol. 2008;52:1231-1238.
2) Rudiger A, et al. Am J Emerg Med. 2007;25:174-178.
3) Brugada J, et al. Circulation. 2002;105:73-78.
4) Priori SG, et al. Circulation. 2002; 105: 1342-1347.
5) Atarashi H, et al. Circ J. 2003;67:8-10.
6) Morita H, et al. Circ J. 2003; 67:312-316
7) Kanda M, et al. J Am Coll Cardiol. 2002;39:1799-1805
8) Eckardt L, et al. PACE. 2001;24:1423-1424
9) RuDusky BM. Am J Cardiol. 2004; 93:671-67

CONDITIONS THAT CAN LEAD TO ST SEGMENT ELEVATION IN THE RIGHT PRECORDIAL LEADS

- Hyperkalemia[1-2]
- Hypercalcemia[3]
- Left Ventricular Enlargement[4]
- LQTS 3 variant[5]
- Autologous peripheral blood stem cell transplantation for acute myeloid leukemia[6]
- Primary lung cancer[7]
- Anti-depressants overdose [8]

1) Littmann L, et al. J Electrocardiol. 2007; 40:53-59.
2) Ansari E, et al. J Electrocardiol. 2003; 36: 257-260.
3) Wu LS, et al. Europace2007;9 :172-174.
4) Bathya B et al. J Assoc Physicians India. 2007; 55 Suppl:7-9
5) Shimizu W, et al. Curr Pharm Des. 2005;11:1561-72
6) Matsubara E, et al. Rinsho Ketsueki. 2004;45:481-483.
7) Kaneda Y, et al. Surg Today. 2001;31:817-819.
8) Akhtar M, et al. Electrocardiol. 2006;39:336-339.

CONDITIONS THAT CAN LEAD TO ST SEGMENT ELEVATION IN THE RIGHT PRECORDIAL LEADS

- Pericarditis[1]
- Duchenne-Erb paralysis or Duchenne muscular dystrophy[2]
- Friederich's ataxia[2]
- Acute Myocarditis[3]
- Myocardial involvement of hematologic diseases[4].
- Arrhythmogenic right ventricular dysplasia[5]
- Cocaine intoxication: acute cocaine poisoning[6]
- Profound Electrolyte Disturbance induced by diabetic ketoasidosis[7]
- Epidural bupivacaine[8]
- Intraventricular conduction defects, RBBB LBBB[9]
- Severe hypothermia[10].

1) Hermida JS, et al. J Electrocardiol. 2007; 40: 67
2) Antzelevitch C, et al. Heart Rhythm. 2005;2:429-440.
3) Satish OS, et al. Chang Gung Med J. 2005;28:69-76
4) Kim YH et al. Pacing Clin Electrophysiol. 2008;31:761-764.
5) Grigorov V, et al. Cardiovasc J S Afr. 2004; 15:139-142
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CONDITIONS THAT CAN LEAD TO ST SEGMENT ELEVATION IN THE RIGHT PRECORDIAL LEADS

- Hypothyroidism[1]
- Central and autonomic nervous system abnormalities[2]
- Mediastinum tumor that compresses the RVOT[3]
- Thiamine Deficiency[2]
- Atypical early repolarization pattern[4]

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