

Centro de Salud y del Golfo El Hierro Santa Cruz de Tenerife Canarias España



Javier Garcia-Niebla

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https://www.youtube.com/watch?v=dy4--_488b4

Elf cap shape signal a new depolarization ominous marker in the sitting of anterior myocardial infarction

Case report

A 65 years old Caucasian female presented to our emergency department (ED) with 1 hour after a sudden onset of oppressive chest pain and dyspnea at rest (NYHA IV). The pain started in the lower chest and migrated to the upper chest without being worsened by movement or deep respiration. She was in acute distress, cold diaphoresis with a pain intensity rated as 8/10.

Personal history: The patient had type II diabetes mellitus for 4 years, hypertension and tobacco dependence. She was on oral treatment and had no other cardiovascular risk factors.

She was without fever, the blood pressure was 160/102 mmHg in the right arm and 160/120 mmHg in the left, the heart rate 74 bpm, and oxygen saturation 99% on room air.

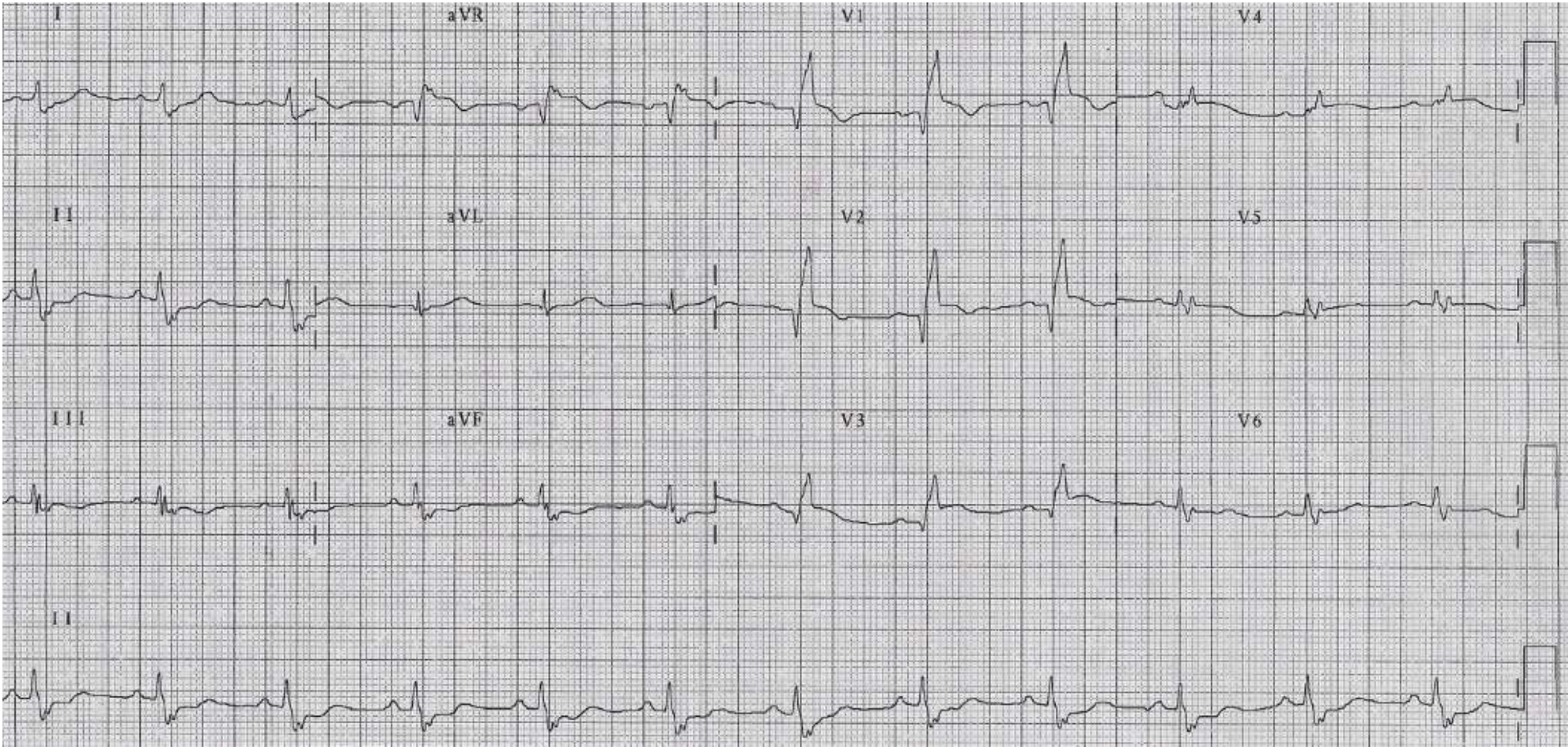
Lung: fine crackles was heard over the patient's posterior lung bases. Heart auscultation showed a third heart sound during early in diastole with gallop cadence. Normal abdominal examination. Peripheral pulses were symmetric.

Lab: Positive biomarkers of myocardial necrosis.

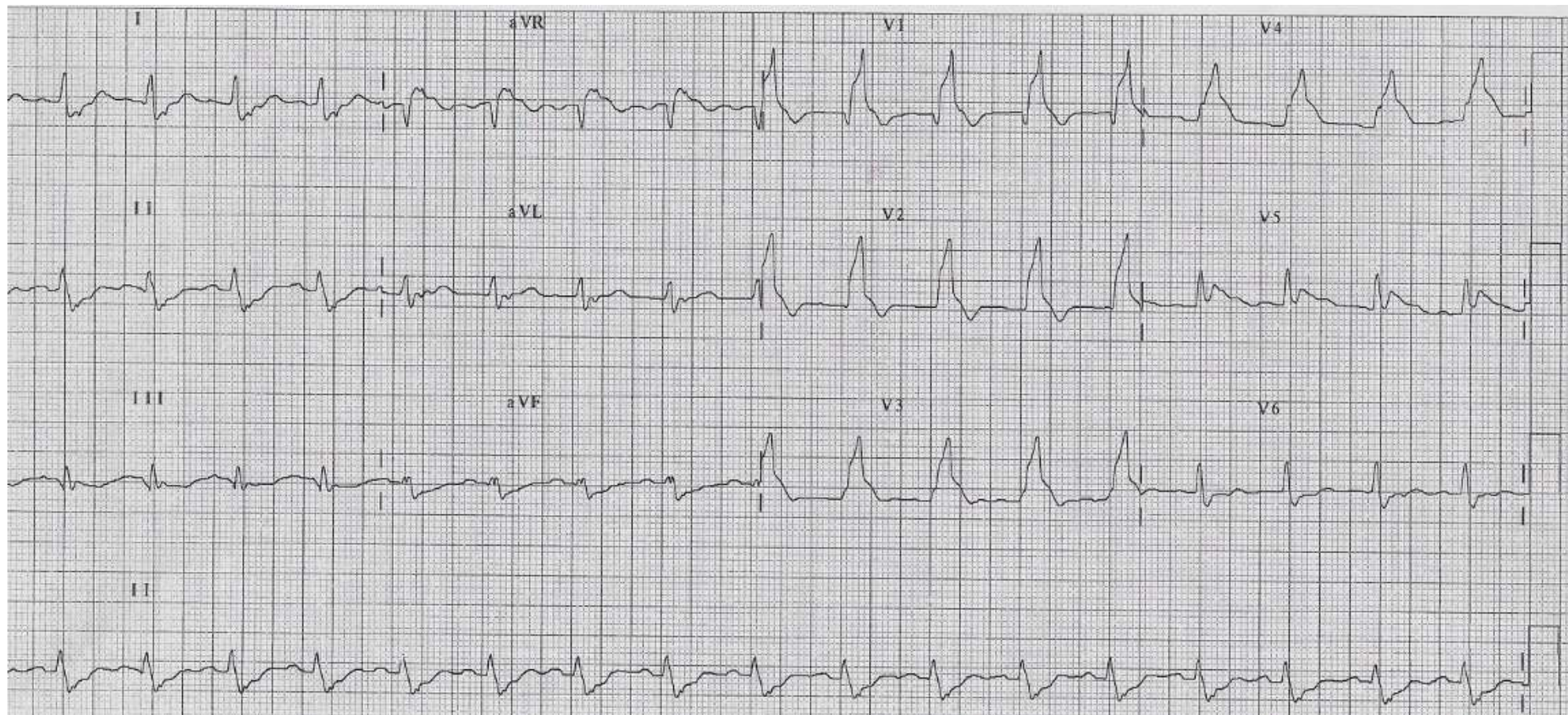
At 17:15 she had suddenly cardiac arrest. Despite the resuscitation measures unfortunately the patient died.

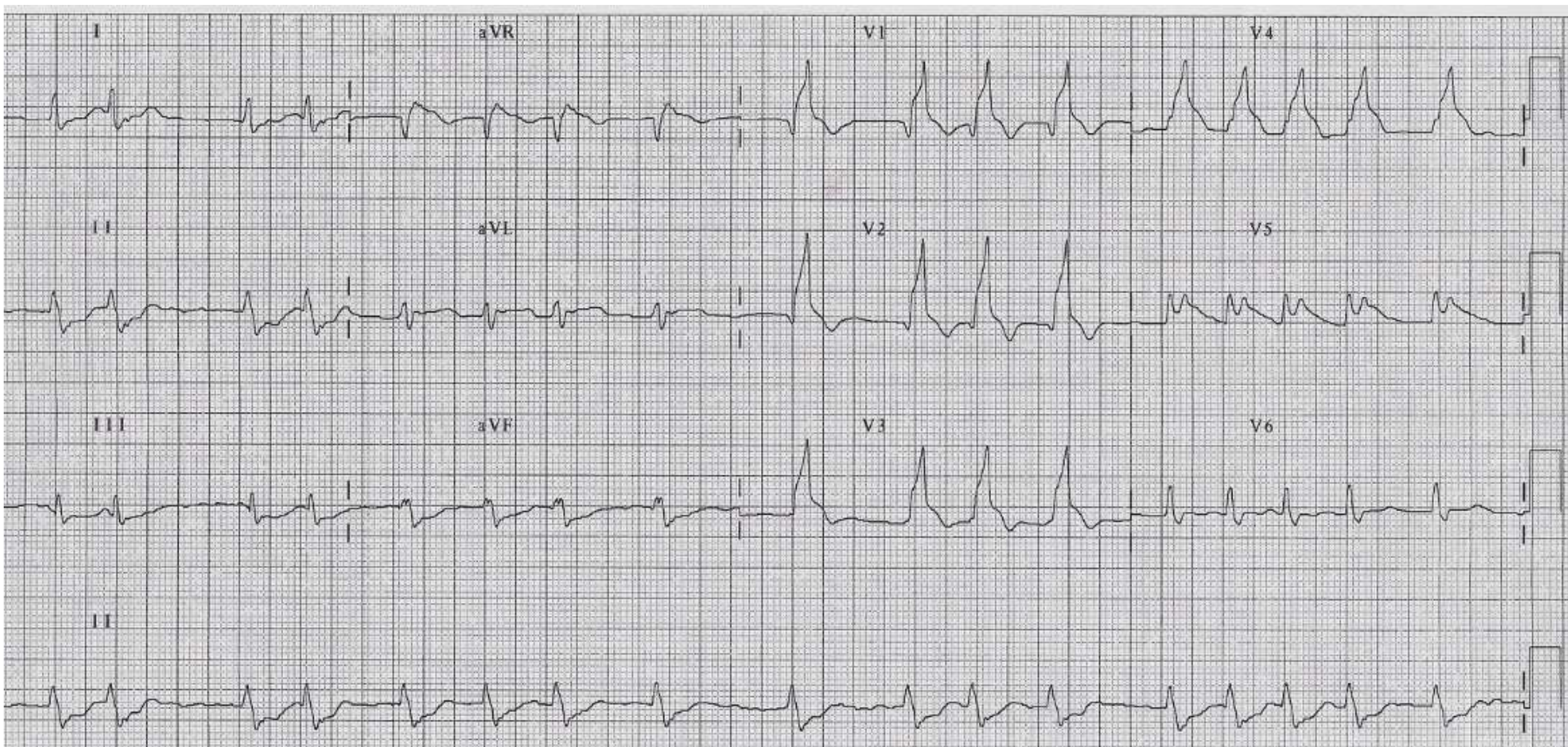
Questions:

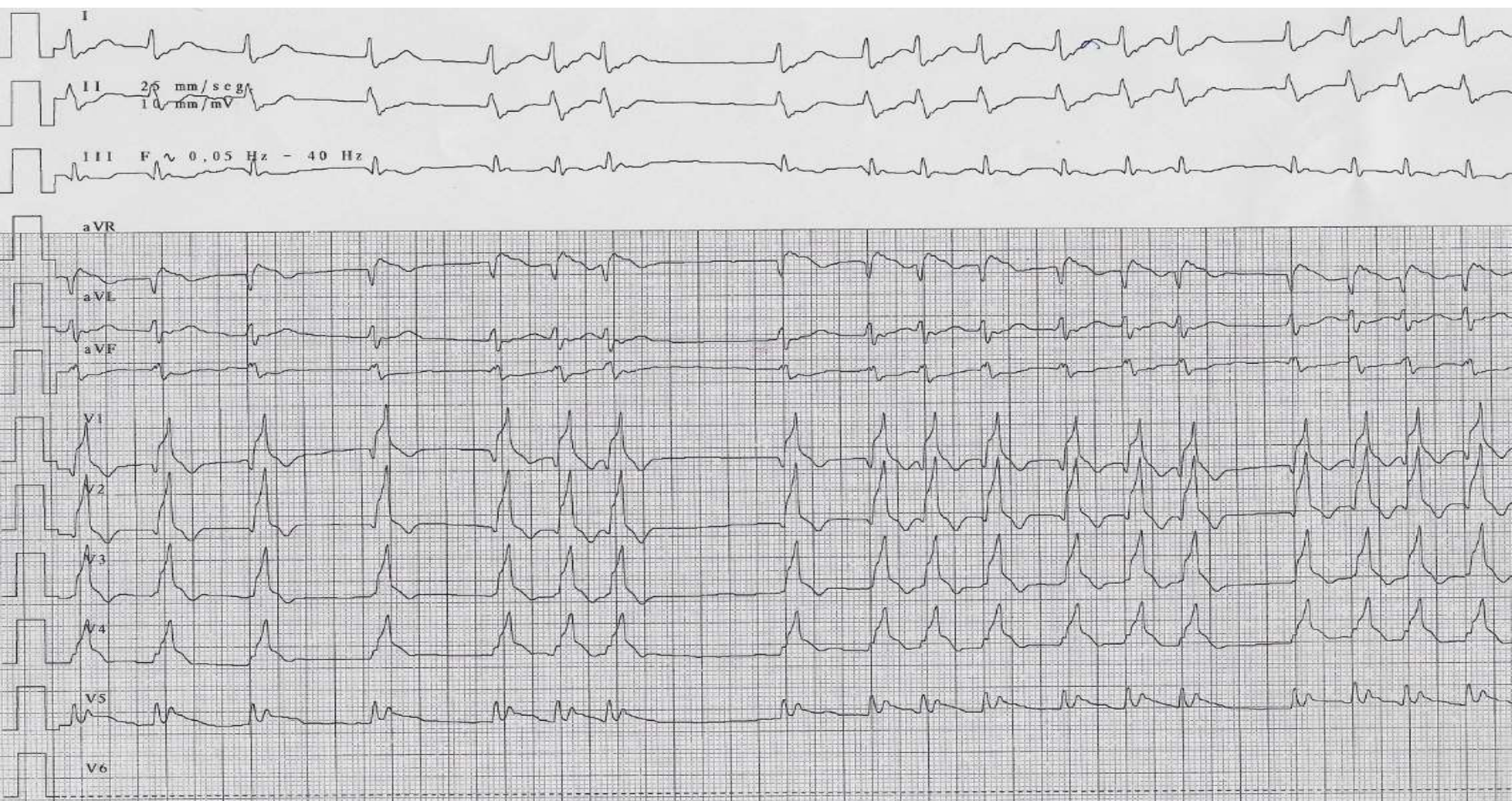
1. What is the electrocardiographic diagnosis?
2. What electrocardiographic changes occurred in this short period of time?



ECG-2 14-01-2019 16:39:12







Colleagues opinions

Question: Dear Andrés what's the difference between this and the "shark fin" sign?

Adrian Baranchuk

Brief trajectory of this pride of Argentina cardiology : Adrian is native of Buenos Aires, Argentina, obtained his MD from the University of Buenos Aires in 1990. After qualifying in Internal Medicine and Cardiology at Sanatorio Mitre, Buenos Aires (1995), he completed a Clinical Fellowship in Cardiac Electrophysiology at the same institution under the supervision of Dr. Claudio Muratore. He completed his Clinical Fellowship (1997) and joined the Arrhythmia Service until 2002 when he immigrated to Spain to join the Arrhythmia Service at the Fundacion Jimenez Diaz, working as a Research Fellow in the Animal Lab under the supervision of Dr. Jeronimo Farre. For his work in this lab, he was awarded the 3rd Chinchona Award, sponsored by 3M Foundation. During his stay in Madrid, Spain, he was PI and received a grant from MAPFRE MEDICINA Foundation (Madrid, Spain) for Ultrasonic Analysis of Radiofrequency Lesion of the Pulmonary Venous Wall through Three-dimensional Intravascular Echography: Therapeutical Implications for Atrial Fibrillation Ablation. Dr. Baranchuk was appointed a Clinical Fellow in Electrophysiology at McMaster University in September 2003, under the supervision of Drs. Carlos Morillo and Stuart Connolly. In 2004 he won first prize at the Fellows Forum, “5th Annual EP Fellows Course” (sponsored by Medtronic), held in Montebello. Dr. Baranchuk was appointed Assistant Professor of Medicine at Queen’s University in the Division of Cardiology in June 2006. He is an active member of the Arrhythmia Service and founded the EP Training Program in 2007. Currently the program has three clinical fellows. He was promoted to Associate Professor in 2010 and to Full Professor in 2016 (Tenure). He is currently the Head of the Heart Rhythm Service. In 2009 he was cross-appointed to the Department of Physiology (now Biomedical and Molecular Sciences). Dr. Baranchuk has presented his work at national and international meetings. In the last four years, he has visited Argentina, Chile, Colombia, Venezuela, Russia, Rumania, Austria, Slovakia, Brazil, Poland, Germany, Spain, USA, and several places in Canada. He is a member of numerous editorial boards and reviewer of several journals. His first book, Atlas of Advanced ECG Interpretation (REMEDICA, UK), was released in 2013 and represents a collaboration effort from recognized electrophysiologists around the world. His second book, Left Septal Fascicular Block (Springer, USA) advances the concept of a trifascicular left hisian system. His first iBOOK, Electrocardiography in Practice: What to do?, written in collaboration with Internal Medicine residents from Queen’s University, remains #1 in iTunes textbooks for the last six months, and has been downloaded more than 1,000 times around the world. His next book about Bayés’ Syndrome, a concept advanced at his lab in Queen’s, will be released Jan. 3, 2017 (Cardiotext, USA).He has published more than 400 articles in prestigious international journals, as well as 35 book chapters, and presented more than 200 abstracts around the world. He is the Vice-President of the Inter American Society of Cardiology (IASC) and the President Elect of the International Society of Electrophysiology (ISE) for the period 2017-2019. He has been also elected as a Member of the Electrophysiology Leadership section of the American College of Cardiology (2017-2020).Dr. Baranchuk is the recipient of several teaching awards, including Outstanding Contribution in



the Core Internal Medicine Program (Queen’s Faculty of Health Sciences), Golden Caliper Award (SOLAECE), Junior Investigator Award (ISHNE), and others. He is a member of the Mentorship Program at Queen’s University. In 2016 he received a Hispanic Business Alliance Award as one the 10 most influential Hispanic Canadians. Congratulation my dear friend!!!! You are amazing. You deserve this mention.

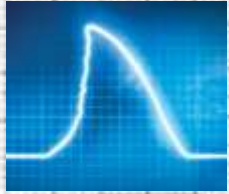
Answer:

Dear Adrian: The Elf cap shape signal is different of Shark Fins morphology also called “Giant R-waves” (**Madias 1981; Faillace 1985; Madias 1993; Testa-Fernandez 2011; Walsh 2015**) or “transient Giant R-waves” (**Chugh 2017**) are very wide phenomenon resultant of a combination of QRS and T-wave. They represent is massive or extreme ST-deviation which encapsulate both STSE as well as ST depression. This is a junctional rhythm with extreme STSE in the inferior leads II, III, and aVF, with concomitant extreme reciprocal or mirror image ST segment depression in leads I and aVL **Old high lateral. The terms posterior and high lateral wall myocardial infarction are incorrect and should be changed to lateral wall and limited anterolateral wall MI (Bayés de Luna 2006 Circulation)**. Additionally, there is also ST-segment depression in the precordial leads maximal in precordial leads from V2-V4 consistent with basal inferior involvement (old dorsal **Bayes de Luna 2006 2007**). It is a massive inferobasal-lateral STEM. Also ST segment depression in aVR a true sign of LMCA occlusion. ST segment elevation in aVR is a sigh of left main or 3 vessel insufficiency (not occlusion), due to diffuse subendocardial ischemia which results in leftward and anterior STS depression and reciprocal STE in aVR LMCA occlusion is identical to simultaneous occlusion of the proximal LAD and LCX The opposing ST vector of anterior and basal can cancel each other out in V1-V4 so that the lateral has the most obvious STE. Lead aVR is opposite an imaginary lead between leads I and II, often called –aVR. LMCA occlusion cause lateral STE (aVL with reciprocal changes in II and III since aVR is reciprocal to this STSE there is ST depression in aVR. The problem is that with this unique morphology, the QRS complex and T-wave merge together as a result of extreme STS deviation, and the two become indistinguishable. But If you can find the end of the QRS complex (the J-point correspondent to phase 1 of monophasic AP) in one lead, you can find the end of the QRS in any lead. ECG territories with Shark Fin reciprocal depression will not have R-waves, but rather S-waves. Shark Fin is an ECG sign of acute coronary occlusion. It is a unique ECG phenomenon consisting of complexes formed by the blurring together of QRS and T-wave as a result of extreme ST-deviation. These complexes manifest in contiguous ECG leads corresponding with coronary anatomy, and represent transmural ischemia. Shark Fin sign should be recognized based on its characteristic morphology, and confirmed by delineating the J-point. While there is a paucity of literature on the topic, the presence of this sign appears to be associated with a significant mortality, underscoring the critical importance of prompt recognition and emergency reperfusion. See ECG in the next slide for ludic explanation.....

ST segment elevation

Shark Fins example

ST segment elevation



ST segment elevation

ST segment elevation

ST segment elevation

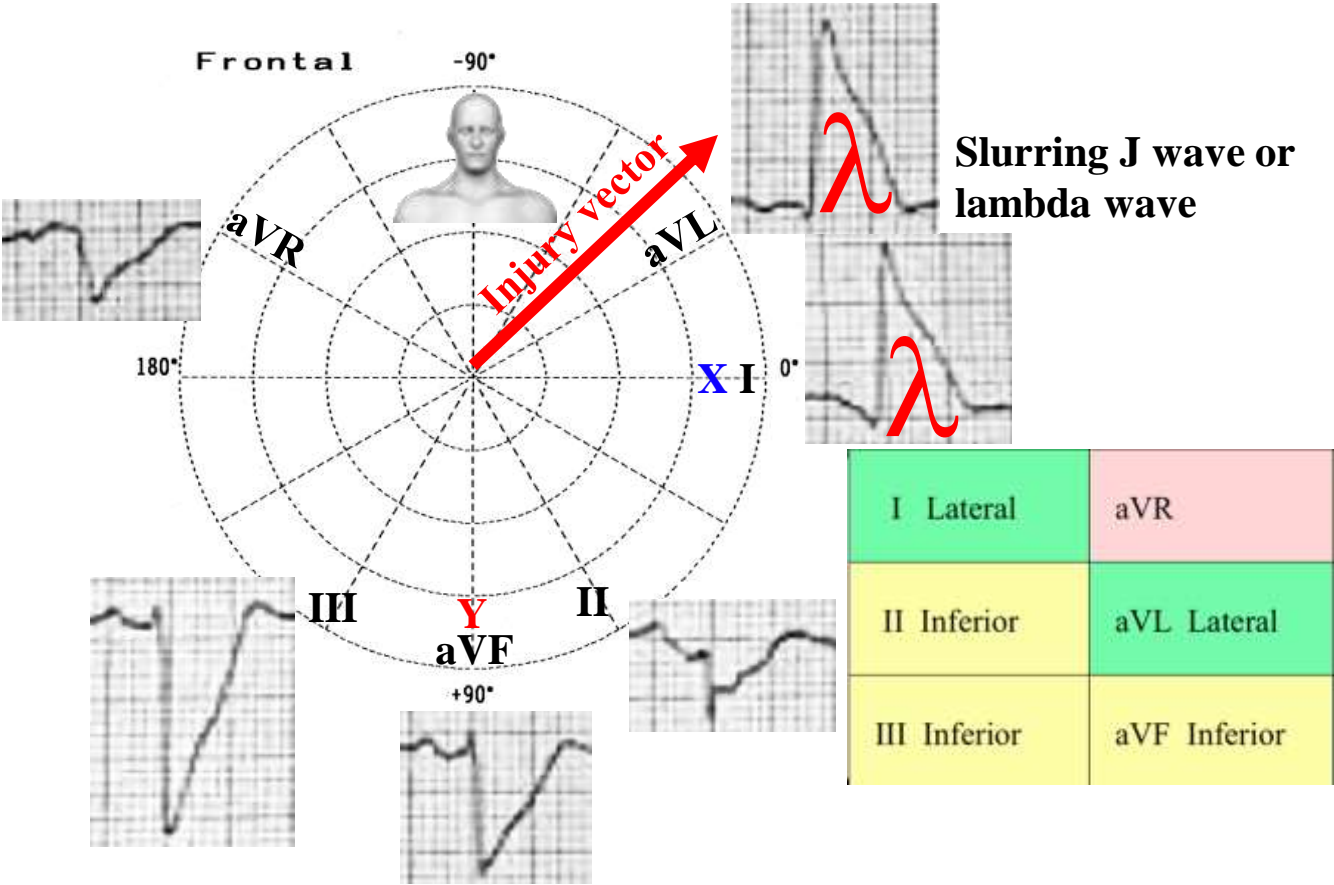
Reciprocal image

Reciprocal image

Reciprocal image

Lead aVL is the only lead facing the superior part of the LV. aVL is the only lead that is opposite the inferior wall of the heart (150° from lead III). 53.3% of patients with inferior wall MI had reciprocal changes \geq STE in inferior leads [1]; 70 – 97.2% of patients with inferior wall MI had reciprocal changes in aVL [2] [3]; 30% of patients with anterior wall MI had reciprocal changes in aVL (**Birnbaum 1993**). ST depression ≥ 1 mM in ≥ 2 lateral leads (I, aVL, V5, or V6) are more likely to: Die (14.9% vs 4.1%); suffer severe HF(14.3% vs 4.1%) [4]. have angina with ECG changes (20.0% vs 11.6%) (**Barrabés 2000**). The most likely culprit artery in inferior MI is RCA (80% of cases): Most likely especially if: STSE III > II and STS depression in lead I and aVL (> 1 mm) Sens 90%, Spec 71%, PPV 94%, and NPV 70%) (**Zimetbaum 2003**). LCX(20% of cases).

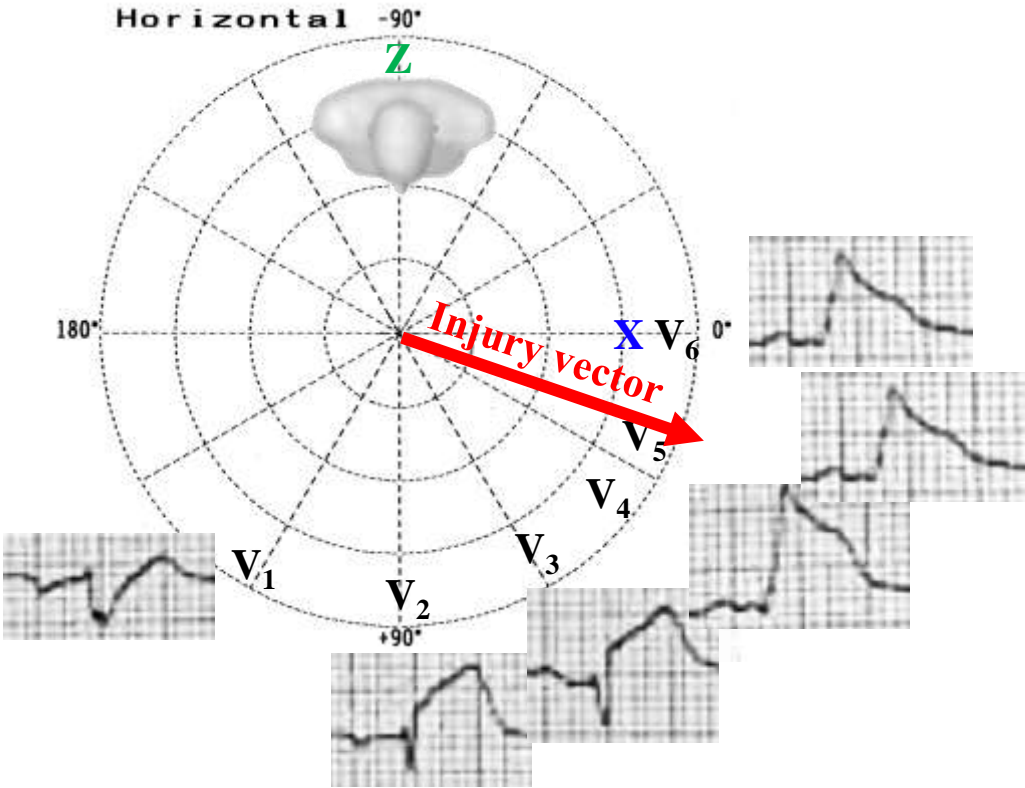
Shark Fins example in the frontal and horizontal planes



Reciprocal leads of I and aVL (**green boxes**) are II, III and aVF (yellow boxes).

I and aVL are mistakenly termed high lateral wall derivations High lateral leads does not exist (**Bayés de Luna 2006; 2007**). Injury vector pointed to the lateral leads and goes away from inferior leads. This direction is indicative of proximal LAD occlusion.

Andrés Ricardo **Pérez-Riera** MD PhD.



In the HP, the injury vector is directed to front and leftward.

V1 Septal	V4 Anterior
V2 Septal	V5 Lateral
V3 Anterior	V6 Lateral

I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

Lateral: I, aVL, V5 and V6: Reciprocal or mirror image leads: II, III and aVF

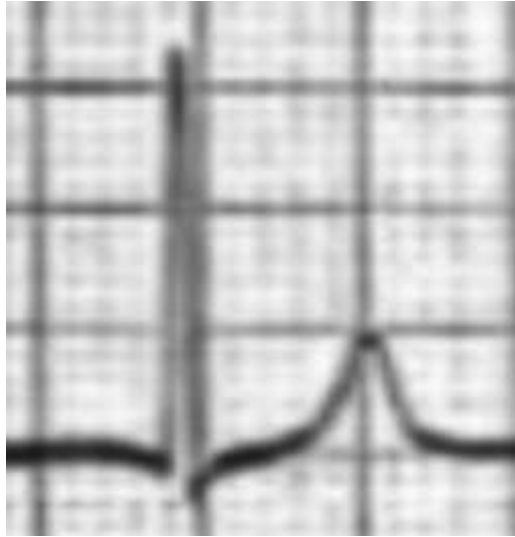
Anterolateral: I, aVL, V4, V5 and V6

Inferior: Reciprocal or mirror image leads: I, aVL

Posterior or dorsal wall: this wall does not exist!

High lateral wall: this wall does not exist

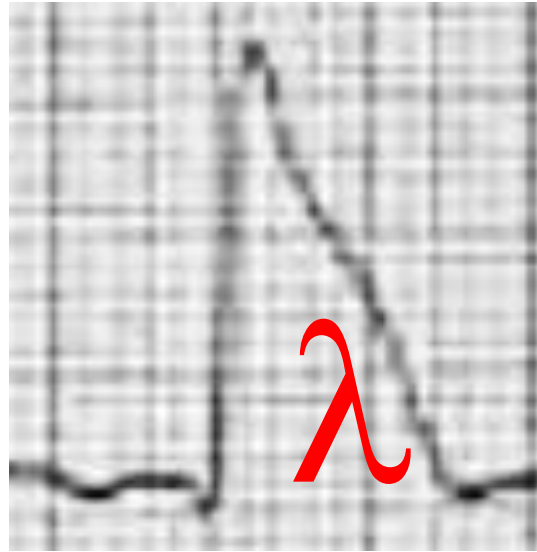
Normal ECG/Normal Action potential



A

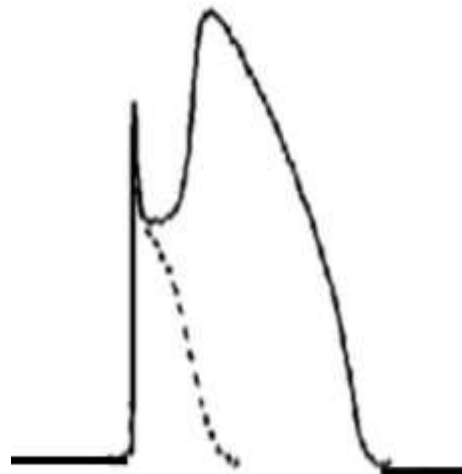


Shark Fins

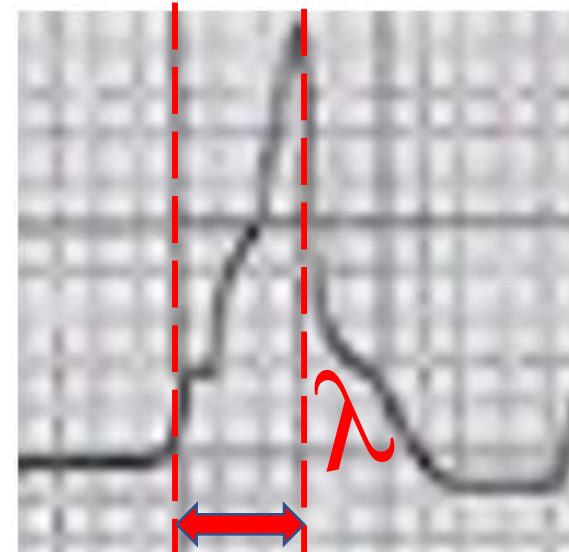


B

**J-slurring or
lambda wave alone**

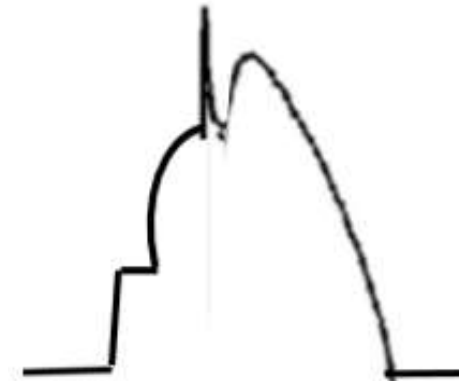


Elf cap shape sign



C

**Very prolonged
R-wave peak time (120ms)
+
J-slurring or lambda**



B. Shark fins

Experimental studies point out that the J wave appearance is the consequence of the presence of transmural gradient in ventricular wall thickness, secondary to the existence in the epicardium but not the endocardium, of significant notch in phase 1, mediated by a greater activity or density of initial transient outward potassium current.

This greater activity and/or density of the Ito channel in epicardial cardiomyocytes, but not endocardial ones, accounts for the characteristic aspect of AP known as “*spike-and-dome configuration of the monophasic action potential*”.

Moreover, the greater initial potassium outflow in the epicardium than the endocardium, causes phase 2 shortening in the epicardium, which conditions transmural dispersion of repolarization and J wave appearance, which carried to a certain level, causes a greater tendency to appearance of ventricular arrhythmia by the mechanism called functional reentry in phase 2 (Yan 1996).

Experimental located cooling of the right ventricular outflow tract (RVOT) *in vivo* in dogs, resembles the electrophysiological alterations that occur in Brugada syndrome, causing J wave appearance secondary to Ito channel activation, and causing the classical aspect of “spike-and-dome configuration” in monophasic action potential of epicardial cells in the RVOT (Nishida 2004).

Several entities are associated to J wave appearance (J wave syndromes) (Hlaing 2005) that include among others, early repolarization syndrome, variant angina, intoxication by tricyclic antidepressants (Bigwood 2005), cocaine abuse (Ortega-Carnicer 2001), hypercalcemia, encephalic lesion, BrS, idiopathic ventricular fibrillation with prominent J wave in inferior wall (Riera 2004), congenital short QT syndrome and the forms called concealed, of arrhythmogenic right ventricular dysplasia.

Experimental evidence supports the hypothesis that one heterogeneous distribution of the Ito channel in the ventricular wall thickness accounts for the spike-and-dome configuration in monophasic AP in the epicardium, and prominent notch in phase 1 and phase 1 shortening, which results in voltage gradient that manifests by J wave (Antzelevitch 2005).

C. Elf cap shape sign

In this case we have severe dromotropic disturbance (depolarization mechanism), consequence of coronary artery disease manifested on ECG by a very prolonged R wave peak time, RBBB, and wide fragmented QRS. When associated with repolarization (J wave) we have eclectic mechanism (depolarization + repolarization) (Pérez-Riera 2012).

Spanish

Se trata de una mujer con factores de riesgo para enfermedad arterial coronaria.

Ingresa en falla de bomba y rápidamente progresa a óbito.

Progresión de ECGs: ritmos sinusal con SCACEST anteroseptal imagen en espejo inferior, probable obstrucción de DA proximal, antes 1° septal o de tronco de CI (R en aVR), presenta fQRS inferior, fuerzas anteriores prominentes con BCRD, probable bloqueo haz anteroseptal, patrón de Aizawa tipo 1 (el casco de delfo) bajo voltaje en derivaciones izquierdas. Luego fibrilación auricular que progresa a FA de alta respuesta ventricular.

Probablemente desencadena TV y FV o ruptura miocárdica, septum alto o rotura externa, causa de óbito.

A la espera de las opiniones de los expertos me despido cordialmente.

Juan Manzzardo Mendoza Argentina

English

It is a woman with risk factors for coronary artery disease. At admission, pump failure and quickly progress to death.

Progression of ECGs: sinus rhythms with an anteroseptal SCACEST reciprocal changes or mirror image, probable obstruction of proximal of LDA, before the first septal perforator branch 1st septal or LMCA (R in aVR), fragmented QRS (fQRS) in the inferior leads, prominent anterior QRS forces with RBBB, probable LSFB, pattern of Aizawa type 1 (delfo helmet) low voltage in left leads. Then atrial fibrillation progresses to AF of high ventricular response.

Probably triggers VT/VF or myocardial rupture, high septum or external rupture, cause of death.

While waiting for the opinions of the experts, I say goodbye cordially.

Juan Manzzardo, Mendoza, Argentina



Andrés and Javier thanks for sharing this interesting and devastating case

Have a lot of ECG sings of bad prognosis:

ECG-1, 14-01-19, 16:24:12

- 1.- Anterior myocardial infarction with RBBB
- 2- Heart rate is 75x' despite signs of heart failure (maybe associated with acute chronotropic dysfunction secondary to acute ischaemia)
- 2.- Early presentation of Q wave in V1-V4 associated with anterior transmural necrosis
- 3.- R-Peak Time in V1-V3 0.08 to 0.12 sec, wide QRS complex 0.12 to 0.14 sec, conduction defect associated with a big necrosis zone
- 3.- ST segment elevation in aVR, that is associated with extended ischemic lesion in left ventricle, and is associated with left main coronary artery occlusion or left main equivalent (coronary obstruction of severe three vessel ischaemia)

ECG-2, 16:39:12

- 1.- Widening of QRS complex 0.20 to 0.26 sec, associated with conduction defect across a big necrosis zone
- 2.- Monofasic R wave in anterior wall (V1-V4), associated with necrosis zone
- 3.- Small P wave, maybe associated with atrial infarction

ECG-3, 17:04:48

- 1- Atrial Fluttering or Atrial Fibrillation with RBBB, wide QRS complex (0.20 to 0.24 sec) and ST elevation of aVF

ECG-4, 17:09:58

- 1- Atrial fibrillation
- 2- Monofasic R wave in V3-V4, with wide QRS complex (0.32 sec) in V4

Is possible to the final rhythm was asystole of pusless electrical activity, secondary to heart failure or mechanical complication such a left ventricular rupture

Thanks for sharing

Dr. Humberto Rodriguez Reyes (FACC, FHRS y AHA Member)

Cardiologia, Electrofisiologia (Arritmias), Medicina Interna

Instructor BLS, ACLS y ACLS-EP de la AHA

Facebook: DrHumberto Rodriguez Reyes, **Facebook:** Sociedad Cardiovascular y Arritmias socaya

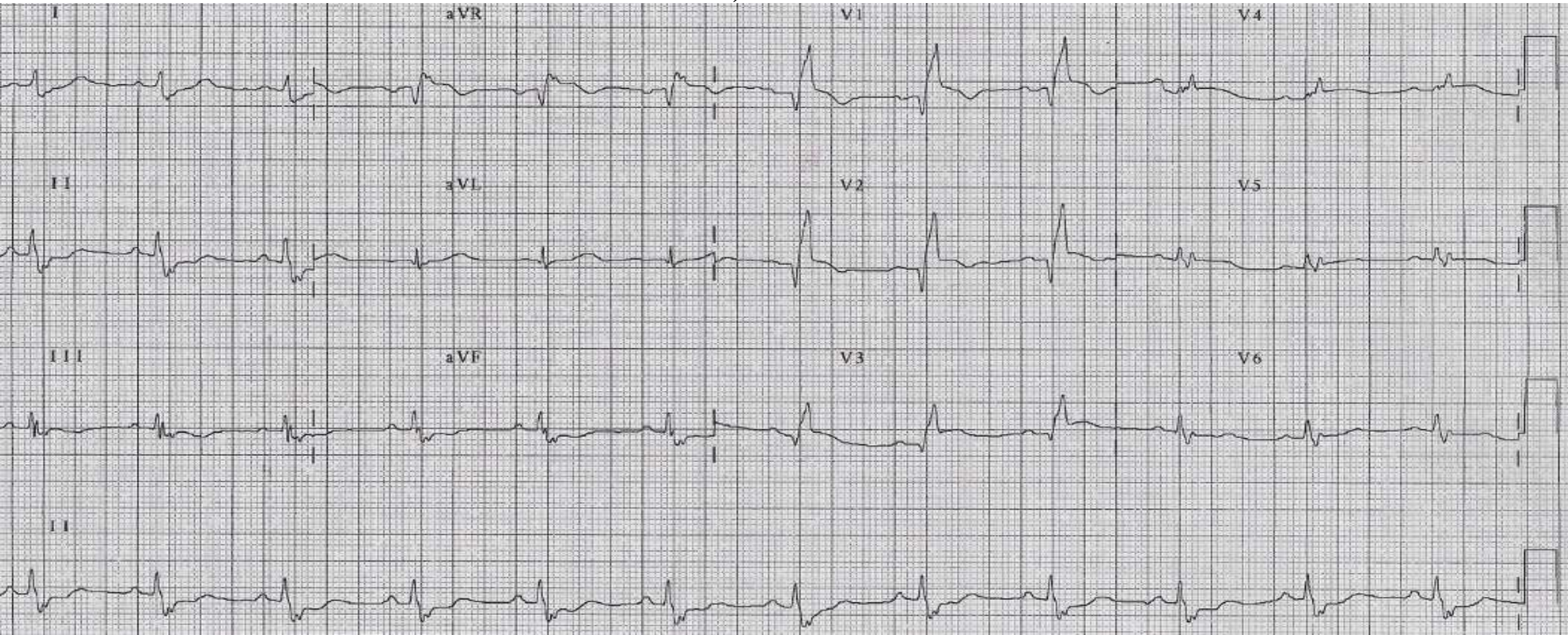
Dirección: República de Argentina 123, Fracc. Las Américas, CP 20230, Aguas Calientes, Ags.

Tel. Consultorio directo: 9162242, 4739745, 4734972, **Cel. Urgencias:** 449-9115962



Dr. Humberto Rodriguez Reyes

ECG-1 14-01-19; 16:24:12 ED admission



Sinus rhythm, HR74bpm, SAP +60°, PR interval: 165, QRS duration (QRSd) 147ms, fragmented wide QRS (fwQRSduration ≥ 120 ms), QT/QTc 422-468, SÂT -27 °, SÂQRS 220. **Conclusion:** CRBBB acute MI with ST segment elevation from V1-V3 and aVR, STSE aVR > V1, discrete ST segment depression in the inferior leads+ fragmented wide QRS (fwQRS) In patients with anterior AMI and RBBB, increasing QRS duration such as the present case is associated with increasing 30-day mortality. Early ST-segment resolution after fibrinolytic therapy despite persisting RBBB is associated with lower mortality rate.



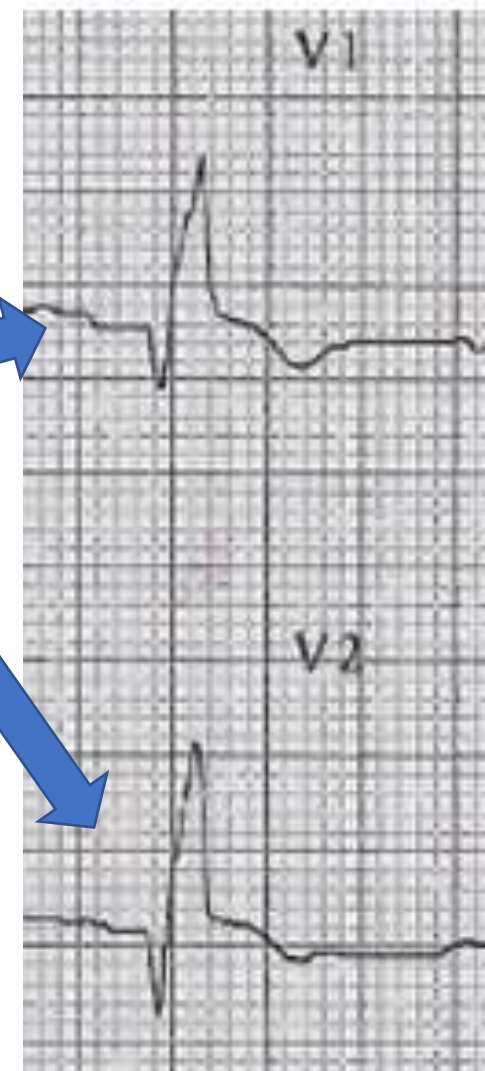
Higher ST segment elevation in aVR compared to V1. This observation suggests very proximal LAD obstruction or LMCA obstruction



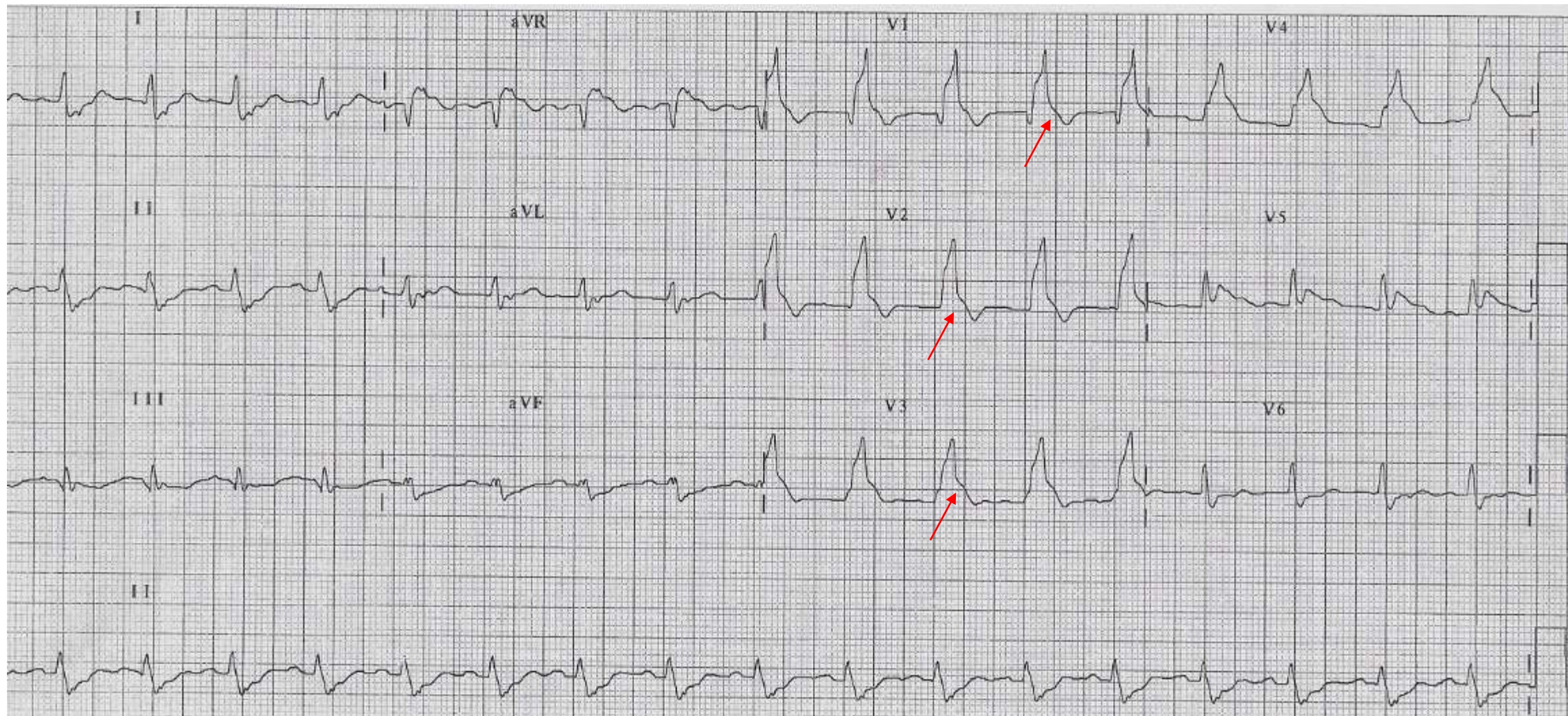
Wide fragmented QRS (W-fQRS) in at least two leads of the same wall (Arrows). f-wQRS on a standard 12-lead ECG is a moderately sensitive and highly specific sign for myocardial scar in patients with known or suspected coronary artery disease. f-wQRS is also an independent predictor of mortality (Das 2008).

QR/qR pattern in the right precordial leads Possible causes

- Severe RVE/RVH (**Gandhi 1962**) (supra-systemic intraventricular pressure inside right ventricle)
- Right Atrial Enlargement: qR pattern in V₁ may be an indirect sign of RAE
- **Complete RBBB complicated with anterior Myocardial Infarction** (**Sodi-Pallares 1952; Rudiakov 1964**)
- Ebstein's anomaly: bizarre and low voltage RBBB with initial q wave (**Kumar 1971**).
- Congenitally Corrected Transposition: Secondary to inversion of septal activation, RAE, by progressive tricuspid regurgitation that occurs with age and associated with deterioration of RV function (**Warnes 2006; Ruttenberg 1966**).
- Endomyocardiofibrosis (**Tobias 1992**).
- MI or ischemia / injury associated with LSFB. S-T elevation and increase in R-wave voltage, “giant R waves”, and concomitant shift of the frontal QRS axis toward the locus of injury is also displayed (**David 1982; Deanfield 1983; Schick 1980; Feldman 1986; Hassapoyannes 1991; Madias 1993; Moffa 1996; Moffa 1997; Uchida 2006**).



ECG-2 14-01-2019 16:39:12



Sinus rhythm, sinus tachycardia (heart rate 102bpm), P axis +59°, PR interval 159 ms, QT/QTc 348/453, T axis -23°

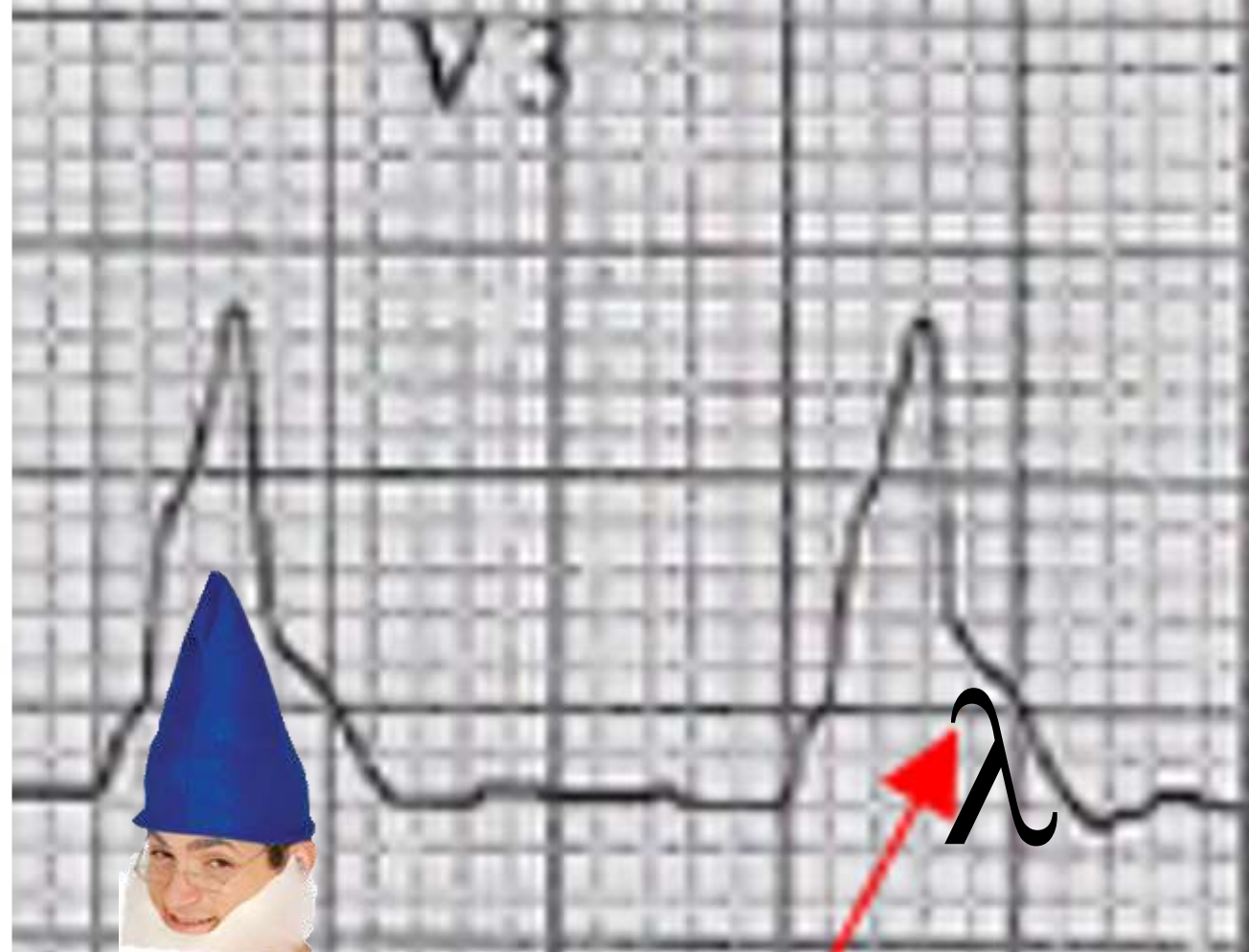
Conclusion: Sinus tachycardia, complete RBBB + STSE acute AMI, reciprocal changes “mirror image” in the inferior leads, very prolonged R-wave peak time in the right precordial, “lambda wave insinuation” or slurring J wave from V1 to V3 (red arrows)



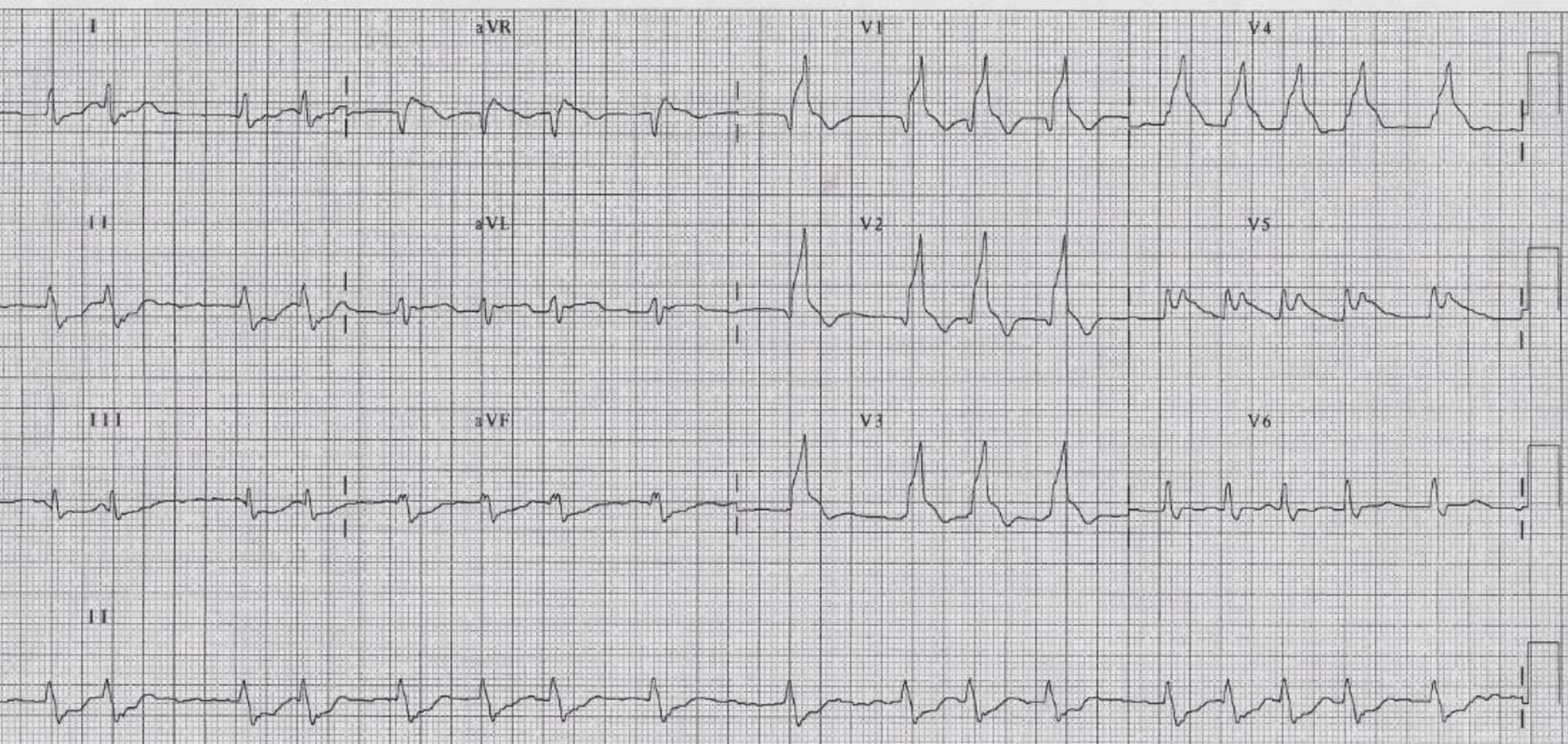
**Very prolonged
R-wave peak time =121ms!!**



Elf cap shape sign

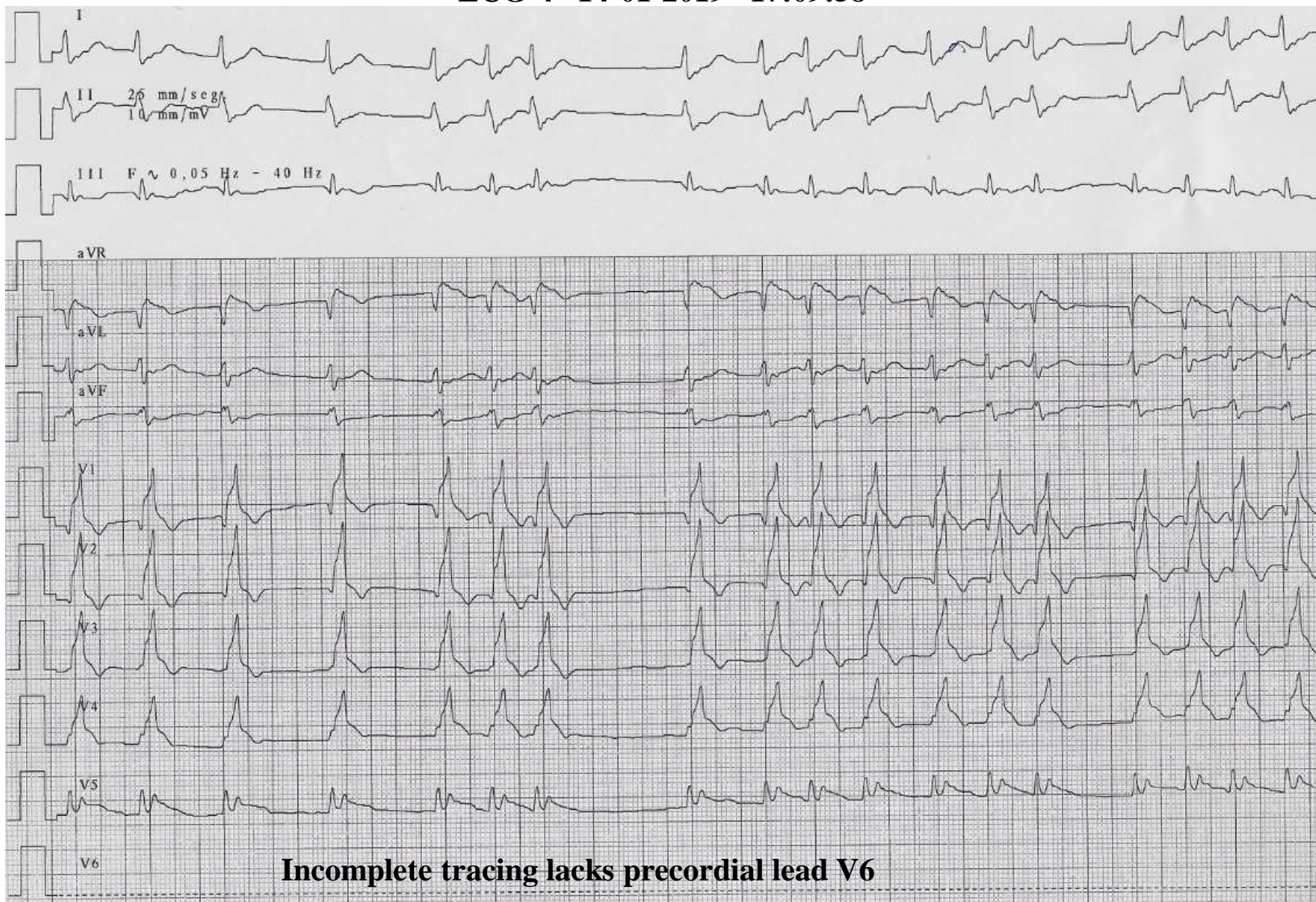


**“Lambdas wave insinuation”
or slurring J wave (arrow)**

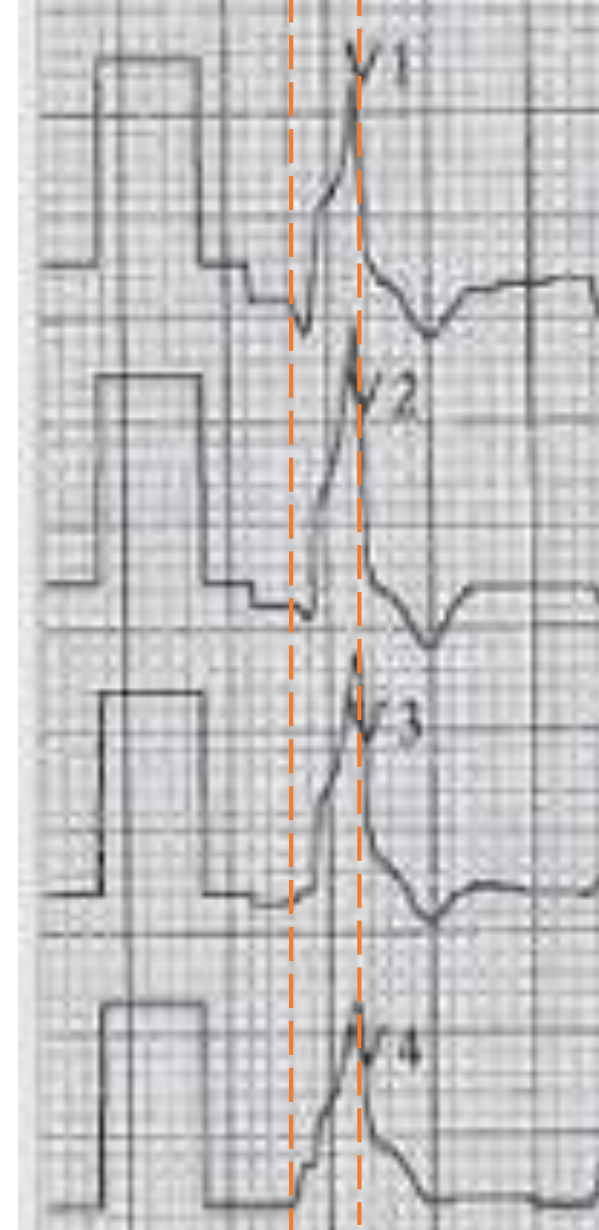


Frec.	117
PR	0
DQRS	155
QT	321
QTc	448
--Eje--	
P	
QRS	247
T	

Atrial fibrillation with rapid ventricular response (>100 bpm), HR 117 bpm, QRS duration 247 ms, wide fragmented QRS (WfRS),



Incomplete tracing lacks precordial lead V6



Atrial fibrillation with rapid ventricular response (mean HR 117 bpm), complete RBBB + anterior myocardial infarction: qR in V1-V2, reciprocal changes in I, aVL, II, III and aVF (inferolateral). Slurring J-wave from V1 to V4, Very prolonged R-wave peak time across precordial leads = 120 ms + slurring J wave or lambda wave.

ECG-1 14-01-19; 16:24:12	ECG-2 14-01-2019 16:39:12	ECG-3 14-01-2019 17:04:48	ECG-4 ECG-4 14-01-2019 17:09:58
Sinus Rhythm	Sinus tachycardia	Atrial fibrillation with rapid ventricular response (>100bpm)	Atrial fibrillation with high ventricular rate
Heart Rate: 74bpm	102bpm	Mean Heart Rate: 117bpm	Mean Heart Rate: 117bpm
P-wave: SAP +60°,	SAP +59°,	0	0
PR interval: 165ms	PR interval: 159ms	0	0
QRS duration:147ms	QRS duration:159ms	QRS duration: 247 ms	275 ms
SÂQRS	226°	247°	
W-fQRS Poor prognosis (Das 2008)	W-fQRS Poor prognosis (Das 2008)	W=fQRS	W=fQRS
CRBBB	CRBBB + acute AMI	CRBBB + acute AMI	CRBBB + acute AMI
QT/QTc: 422-468	QT/QTc: 348/453	QT/QTc: 422-468	QT/QTc: 348/453
Reciprocal changes II, III and aVF	Reciprocal changes II, III and aVF	Reciprocal changes II, III and aVF	Reciprocal changes II, III and aVF
SÂT -27 °	SÂT -23°	SÂT -27 °	SÂT -23°
Conclusion: Acute anterior MI with STSE + W-f QRS + QTc prolongation + complete RBBB STSE aVR>V1 + mirror image inferior leads	Equal	Conclusion: Atrial fibrillation with high ventricular rate Acute AMI with STSE + WfQRS + QTc prolongation + complete RBBB STSE aVR>V1 + very prolonged R wave peak time	Atrial fibrillation with high ventricular rate, extreme QRS prolongation indicative of poor prognosis + very prolonged R wave peak time



Elf cap
shape

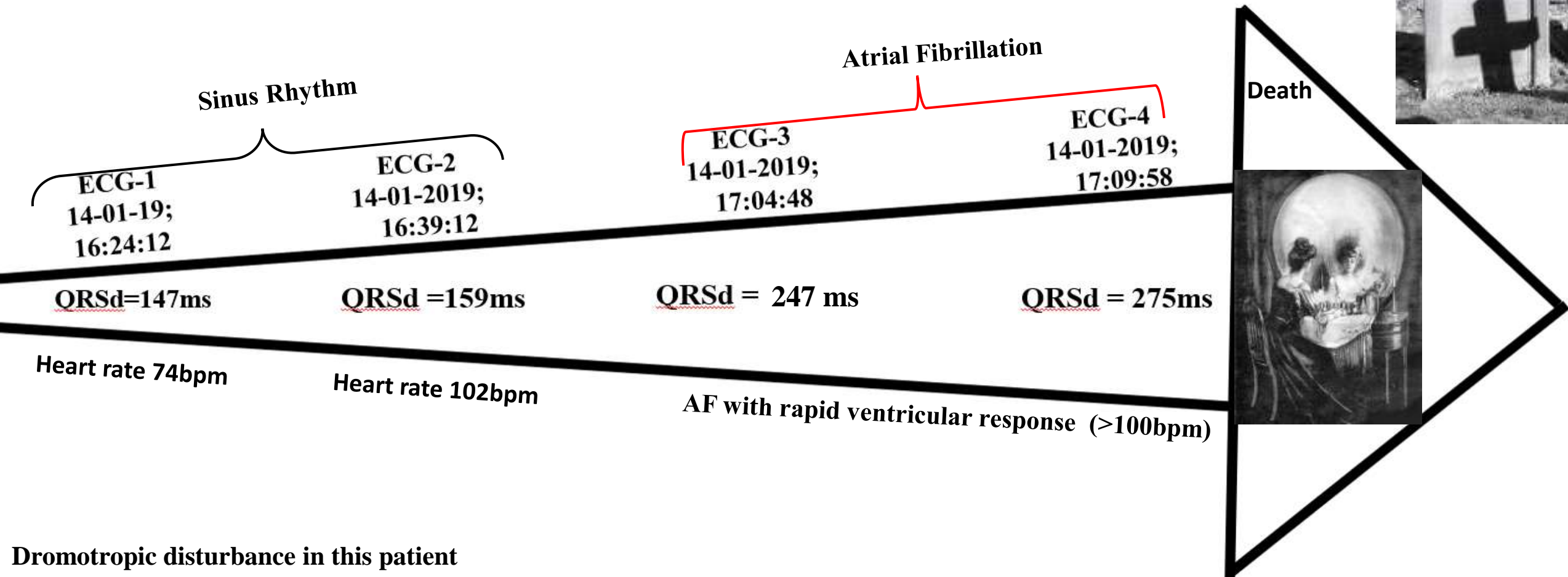
Very prolonged R-wave peak time
+
Lambda wave

Prolonged
R-wave
peak time

+

Very wide QRS complex with
elf cap shape signal
“Signo del gorro de duende”

The QRS complex duration evolution in just 45 minutes: from 147ms to 275ms



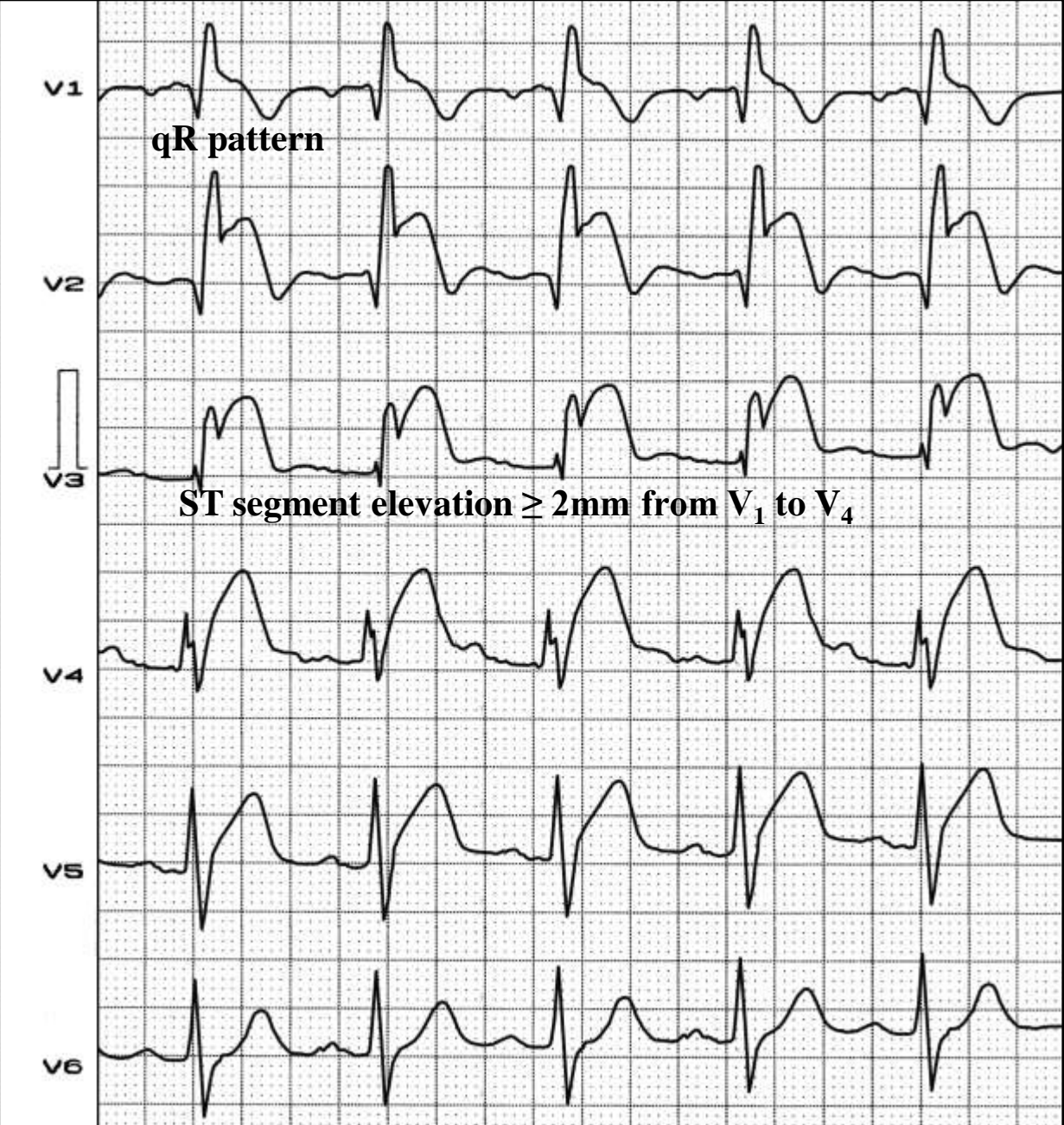
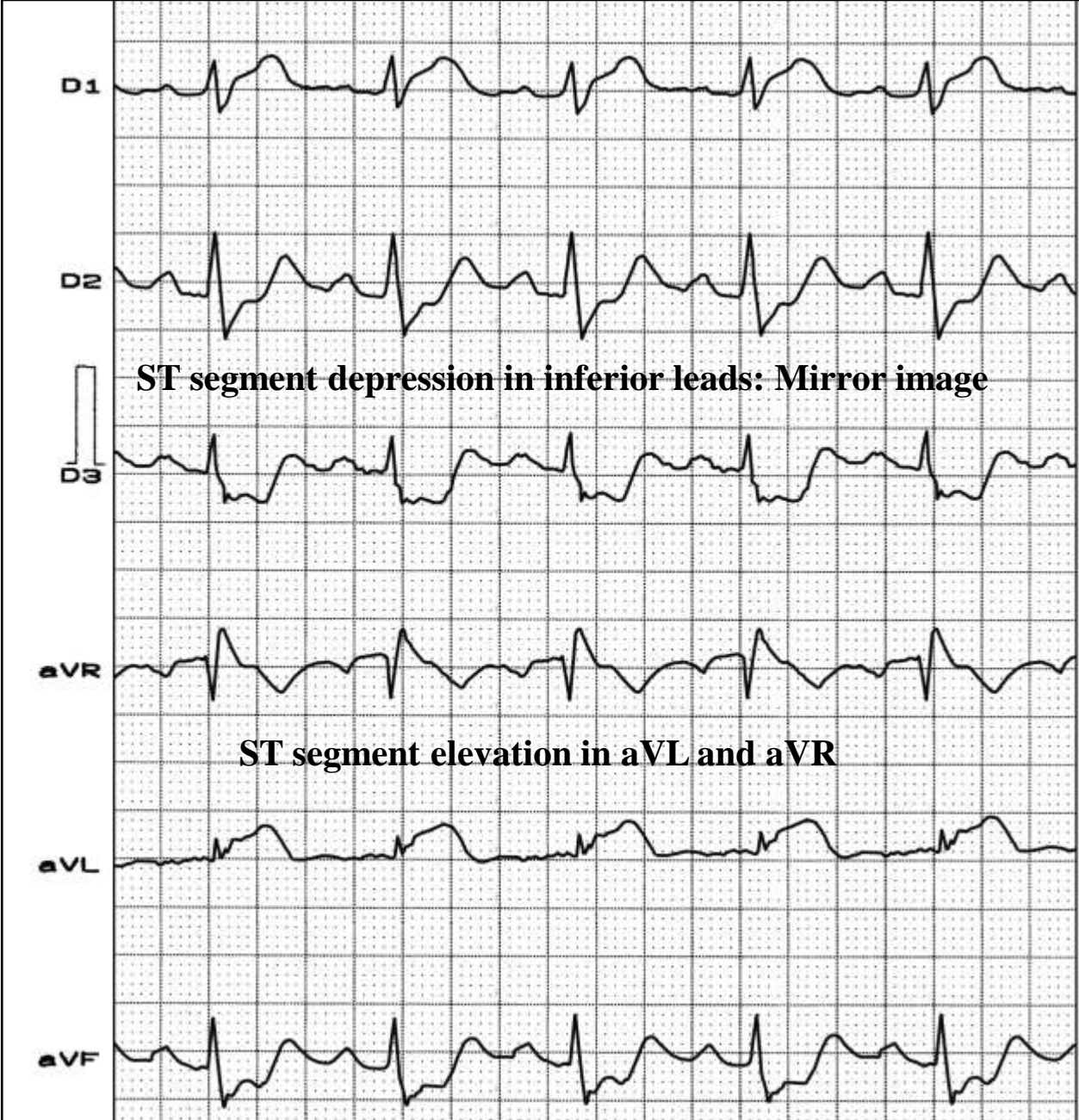
Dromotropic disturbance in this patient

- 1) Complete RBBB
 - 2) Wide fragmented QRS
 - 3) Very prolonged R-wave peak time
 - 4) Slurring J wave at the end of QRS
- 3 + 4 = Elf cap shape sign

Learning points

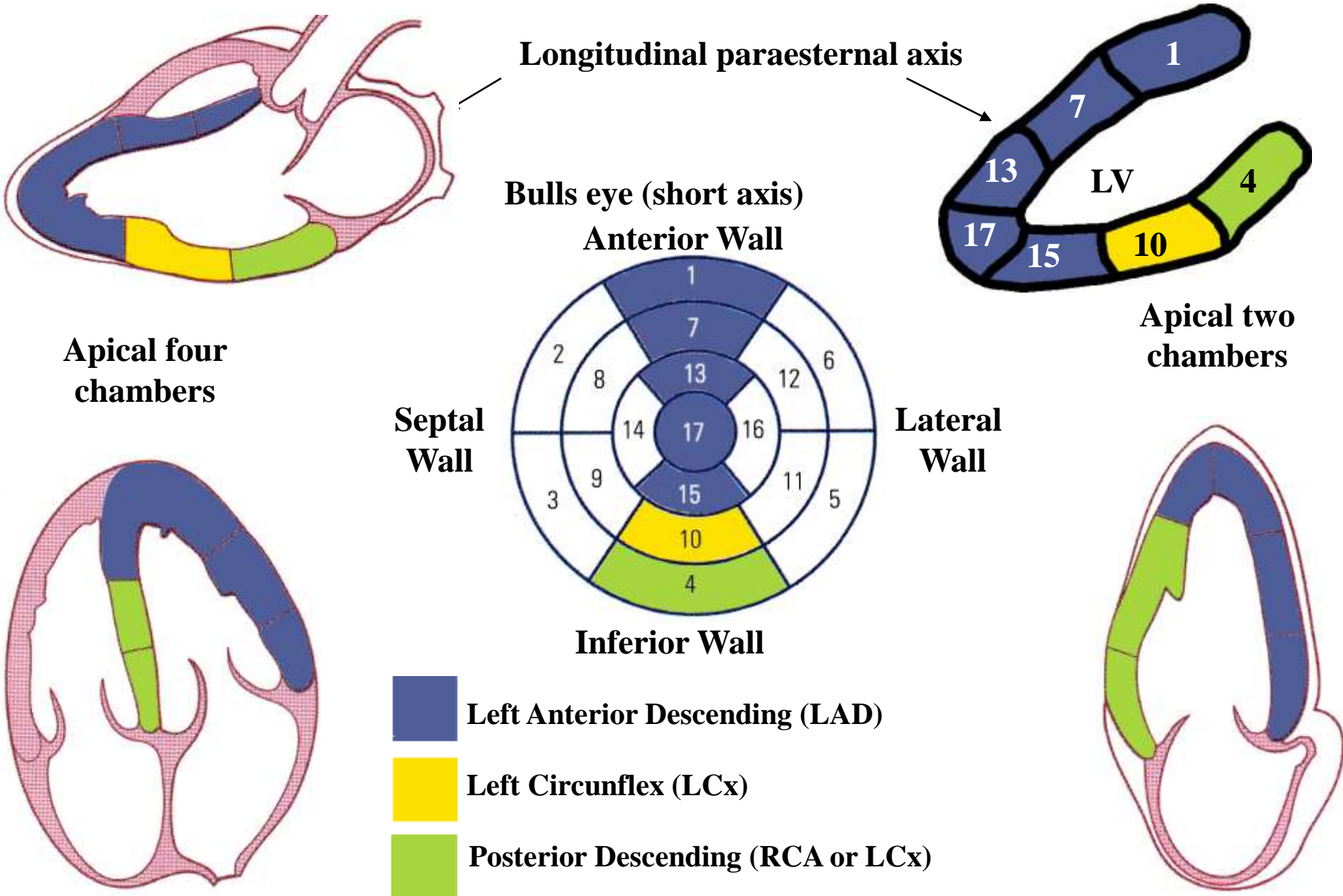
- Right bundle branch block (RBBB) does not usually interfere with the interpretation of ST-segment elevation in acute anterior myocardial infarction (AMI). However, RBBB may be the only ECG sign of an AMI.
- Left ventricular ejection fraction decreases and Killip grade increases in case of RBBB in the setting of acute AMI.
- Culprit artery in patients with RBBB is more commonly a LAD proximal obstruction before the first septal perforator branch and threatened myocardial tissue is larger in patients with RBBB.
- Where a new RBBB is found in a clinical context suggestive of an AMI, clinicians must consider AMI in their differential diagnosis and initiate reperfusion therapy without delay once the diagnosis is confirmed.
- In patients with Left Ventricular Ejection Fraction $\leq 35\%$, RBBB is associated with significantly larger scar size than LBBB is, and occlusion of a proximal LAD septal perforator causes RBBB. In contrast, LBBB is most commonly caused by nonischemic pathologies.
- The HERO-2 trial demonstrated that the co-existence of RBBB and Anterior MI confers a 3-4 times higher 30-day mortality than anterior MI without a conduction abnormality (**Wong 2006**).
- Elf cap shape sign is an ominous signal in the setting of AMI indicative of severe intraventricular conduction disturbance.
- Patients with MI and RBBB have a poor prognosis (**Widimsky 2012**). It may be difficult to detect transmural ischemia in patients with chest pain and RBBB. Therefore, a primary PCI strategy (emergent coronary angiography and PCI if indicated) should be considered when persistent ischemic symptoms occur in the presence of RBBB.

Another example of Acute MI complicated with CRBBB consequence of proximal LAD occlusion before first septal perforator (S₁) complicated with RBBB



Electrocardiographic topographic classification of myocardial infarctions			
Wall Affected	Leads Showing ST Segment Elevation	Leads Showing Reciprocal ST Segment Depression	Suspected Culprit Artery
Septal	V1, V2	None	Left Anterior Descending (LAD)
Anterior	V3, V4	None	LAD
Anteroseptal	V1, V2, V3, V4	None	LAD
Anterolateral	V3, V4, V5, V6, I, aVL	II, III, aVF	LAD, Circumflex (LCX), or Obtuse Marginal
Extensive anterior (Sometimes called Anteroseptal with Lateral extension)	V1,V2,V3, V4, V5, V6, I, aVL	II, III, aVF	Left main coronary artery (LCA)
Inferior	II, III, aVF	I, aVL	Right Coronary Artery (RCA) or Left Circumflex (LCX)
Lateral	I, aVL, V5, V6	II, III, aVF	LCX) or Obtuse Marginal
Right ventricular (Usually associated with Inferior)	II, III, aVF, V1, V4R	I, aVL	Right Coronary Artery (RCA)
Atrial infarction: usually associated with ventricular infarction. Episodes of supraventricular tachycardia or AF. Abnormal P waves: M-shaped, W-shaped, irregular or notched;	Depression of the P-Ta segment of more than 1.5 mm. in precordial leads and 1.2 mm. in leads I, II, and III in the presence of any form of atrial arrhythmia.	Elevation of the P-Ta segment of over 0.5 mm. in V5; and V6 with reciprocal depression of the same segment in V1 and V2; Elevation of the P-Ta segment of over 0.5 mm. in lead I and its depression in leads II or III;	RCA or LCx

Current model of ventricular segmentation and wall/artery irrigation



Wave Myocardial Infarction Based on Correlations With Magnetic Resonance Imaging

Q-wave myocardial infarction (MI) location is generally based on a pathologic correlation first proposed >50 years ago. Despite the proved reliability of contrast-enhanced cardiovascular magnetic resonance (CE-CMR) imaging to detect and locate infarcted areas, no global study has been conducted with the aim of correlating the ECG patterns of Q-wave MI with infarct location. Bayés de Luna et al studied this correlation and divided Q-MI in (**Bayes de Luna 2006; 2006; 2006; 2007; 2007**):

I) Anteroseptal zone

- **Septal MI: Type: A1. Most probable place of occlusion:** Subocclusion of the LADA affecting septal branches. **ECG pattern:** Q in V1-V2. **Infarction area CMR:** 2, 3, 8, 9 and 14. Or only 8, 2 and 14. SE: 100% SP: 97%.
- **Apico-anterior MI: Type: A2. Most probable place of occlusion:** distal occlusion of LADA **ECG pattern:** Q in V1-V2 to V3-V6 **Infarction area CMR:** 13, 14, 15, 16, 17, 7, 8, 9. or only 13, 14, 15, 17. SE: 85% SP: 98%.
- **Extensive anterior MI: Type: A3. Most probable place of occlusion:** Proximal LADA occlusion Q in precordial leads V1-V2 to V4-V6, aVL, and sometimes I. **Infarction area CMR:** 13, 14, 7, 8, 9, 1, 2, 3. SE: 83% SP: 100%.
- **Mid-anterior MI: Type: A4. Most probable place of occlusion:** first diagonal occlusion **ECG pattern:** QS in VL, and sometimes Q in I, without pathological Q in V5-V6. Sometimes small q in V2-V3. **Infarction area CMR:** 7 and 13. Minimal in 1, 12 and 16. SE: 67% SP: 100%.

II) Inferobasal zone

- **Lateral: Type: B1. Most probable place of occlusion:** LCx or its branches oblique marginal(OM). **ECG pattern:** RS in V1-V2 and /or Q wave in I, VL, V5-V6. Diminished R wave in V6. **Infarction area CMR:** 6, 5, 12, 11, and 16. SE: 67% SP: 99%.
- **Inferior: Type: B2. Most probable place of occlusion:** RCA occlusion, sometimes distal LCx occlusion. **ECG pattern:** Q in II, III, VF. **Infarction area CMR:** 3, 4, 10, 15, 3, and 9. SE: 88% SP: 97%.
- **Inferolateral: Type: B3. Most probable place of occlusion:** RCA or dominant LCx, with **ECG pattern:** sign of inferior (Q in II, II, VF: b2) and/or lateral infarct (RS in V1-V2). SE: 73%; SP: 98%.

New conclusions: The predefined ECG patterns we used matched well (86% global concordance) with their corresponding infarction areas as detected by CE-CMR imaging and have real value in clinical practice.

The RS morphology in lead V1 is due to lateral MI and the QS morphology in lead aVL is due to mid-anterior and mid-lateral MI. In other words the RS pattern in V1-V2 is explained by lateral MI and not by MI of inferobasal (posterior) wall.

The terms posterior and high lateral infarction are incorrect and should be changed to lateral wall and limited anterolateral wall MI.

Anterior STEMI results from occlusion of the left anterior descending artery (LAD). Anterior myocardial infarction carries the worst prognosis of all infarct locations, mostly due to larger infarct size.

A study comparing outcomes from anterior and inferior infarctions (STEMI + NSTEMI) found that on average, patients with anterior MI had higher incidences of in-hospital mortality (11.9 vs 2.8%), total mortality (27 vs 11%), heart failure (41 vs 15%) and significant ventricular ectopic activity (70 vs 59%) and a lower ejection fraction on admission (38 vs 55%) compared to patients with inferior MI.

In addition to anterior STEMI, other high-risk presentations of anterior ischemia include left main coronary artery (LMCA) occlusion, Wellens syndrome and De Winter T waves.

How to Recognize Anterior STEMI

ST segment elevation with Q wave formation in the precordial leads (V1-6) \pm I and aVL.

Reciprocal ST depression in the inferior leads (mainly III and aVF).

NB. The magnitude of the reciprocal change in the inferior leads is determined by the magnitude of the ST elevation in I and aVL (as these leads are electrically opposite to III and aVF), hence may be minimal or absent in anterior STEMIs that do not involve the lateral leads I and aVL.

Patterns of Anterior Infarction

The nomenclature of anterior infarction can be confusing, with multiple different terms used for the various infarction patterns. The following is a simplified approach to naming the different types of anterior MI.

The **precordial leads** can be classified as follows:

Septal leads = V1-2

Anterior leads = V3-4

Lateral leads = V5-6

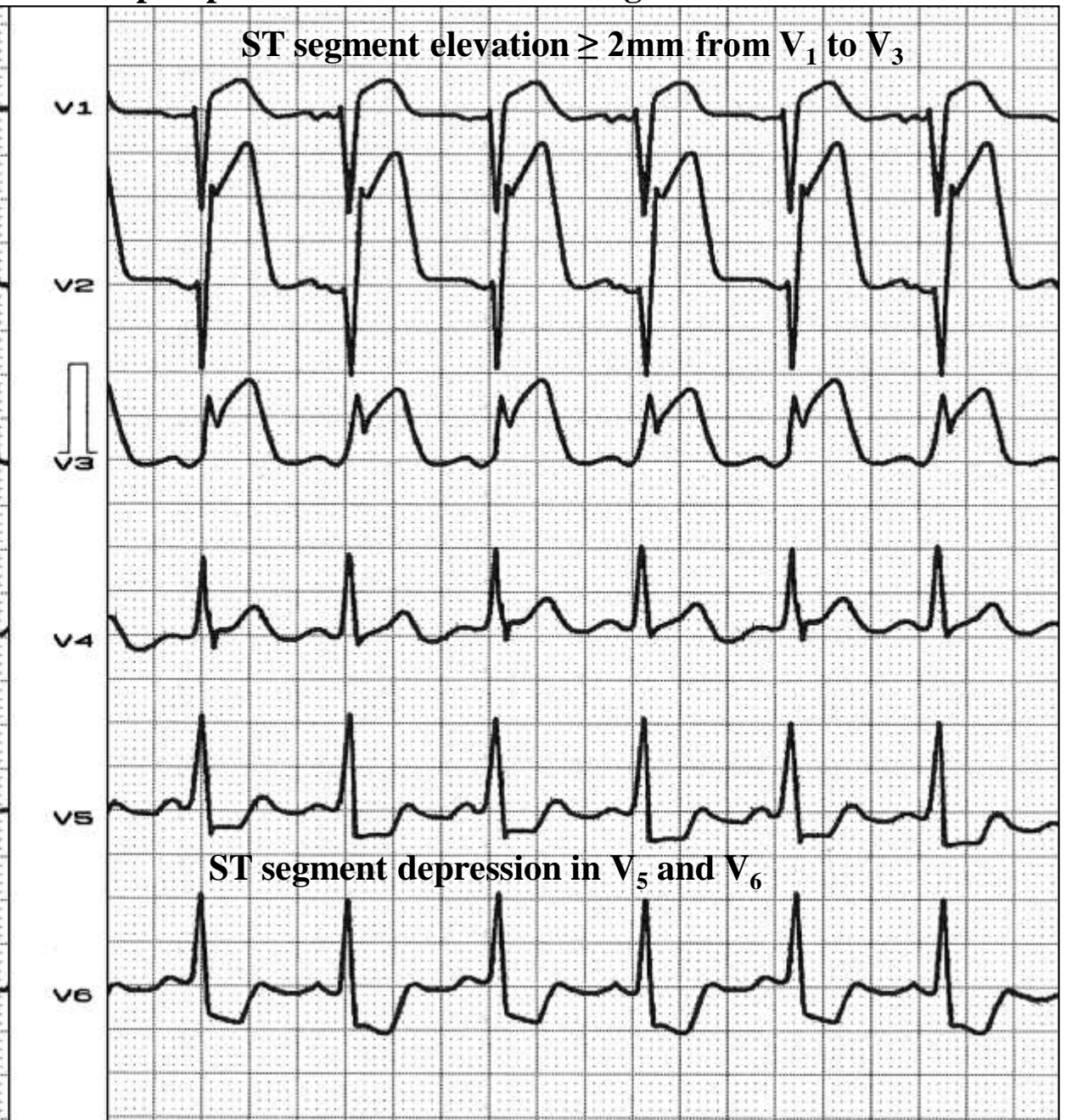
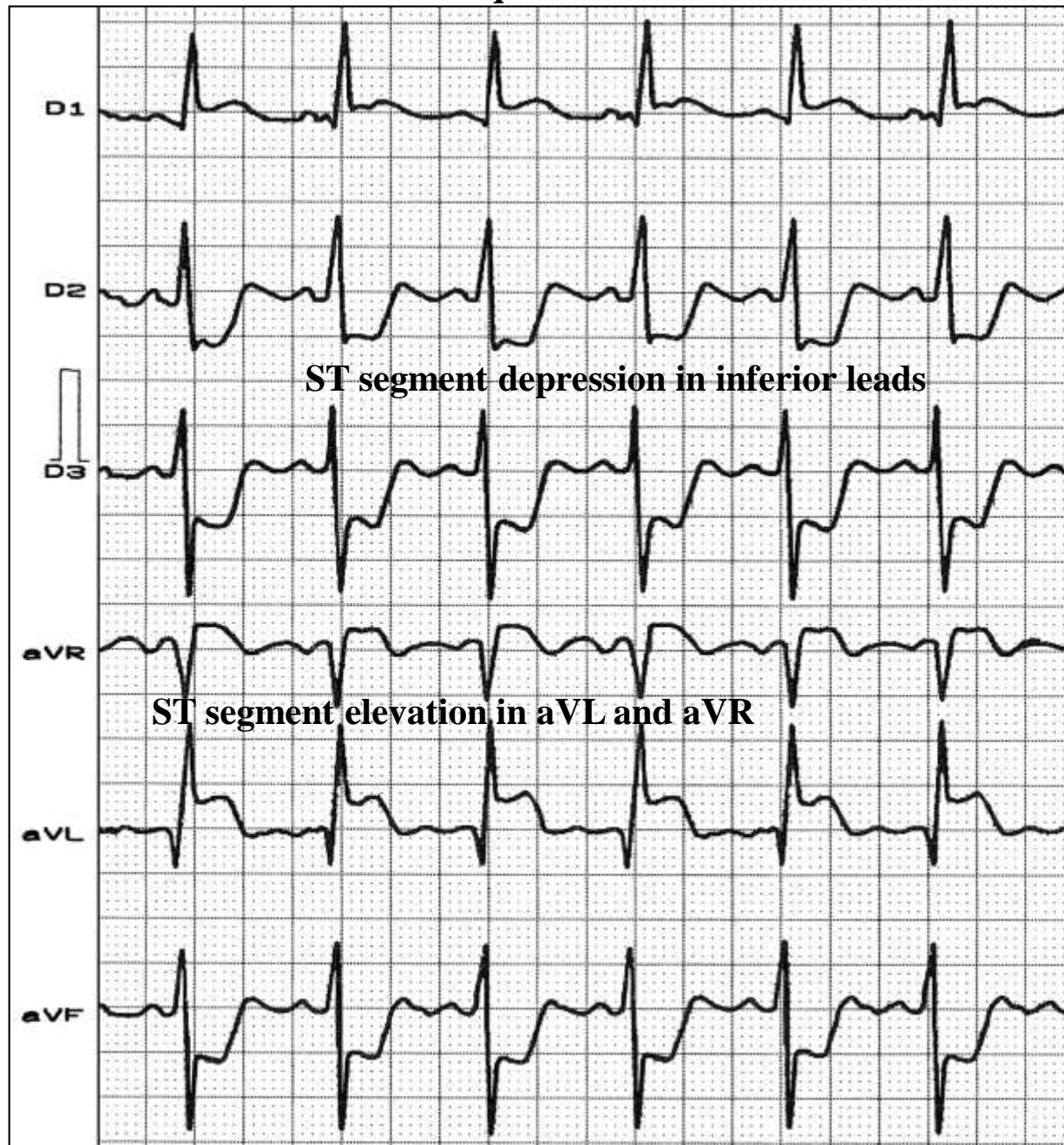
The different **infarct patterns** are named according to the leads with maximal ST elevation:

The different **infarct patterns** are named according to the leads with maximal ST elevation: Septal = V1-2; Anterior = V2-5; Anteroseptal = V1-4

Anterolateral = V3-6, I + aVL; Extensive anterior / anterolateral = V1-6, I + aVL

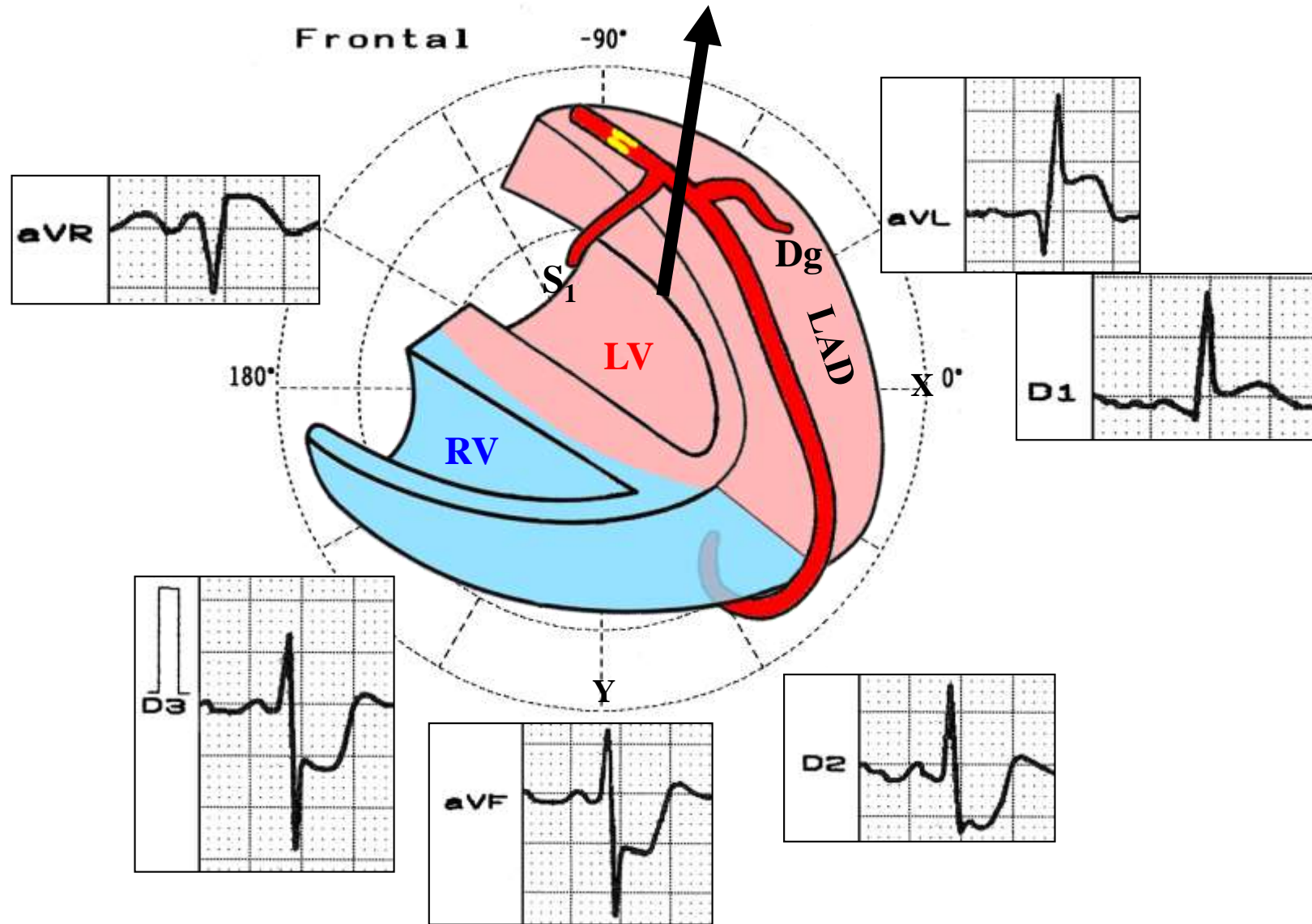
(NB. While these definitions are intuitive, there is often a poor correlation between ECG features and precise infarct location as determined by imaging or autopsy. For an alternative approach to the naming of myocardial infarctions, take a look at this consensus from Circulation 2006 (**Bayés de Luna 2006**)).

AMI consequence of occlusion of LAD before the first septal perforator and the first diagonal branch



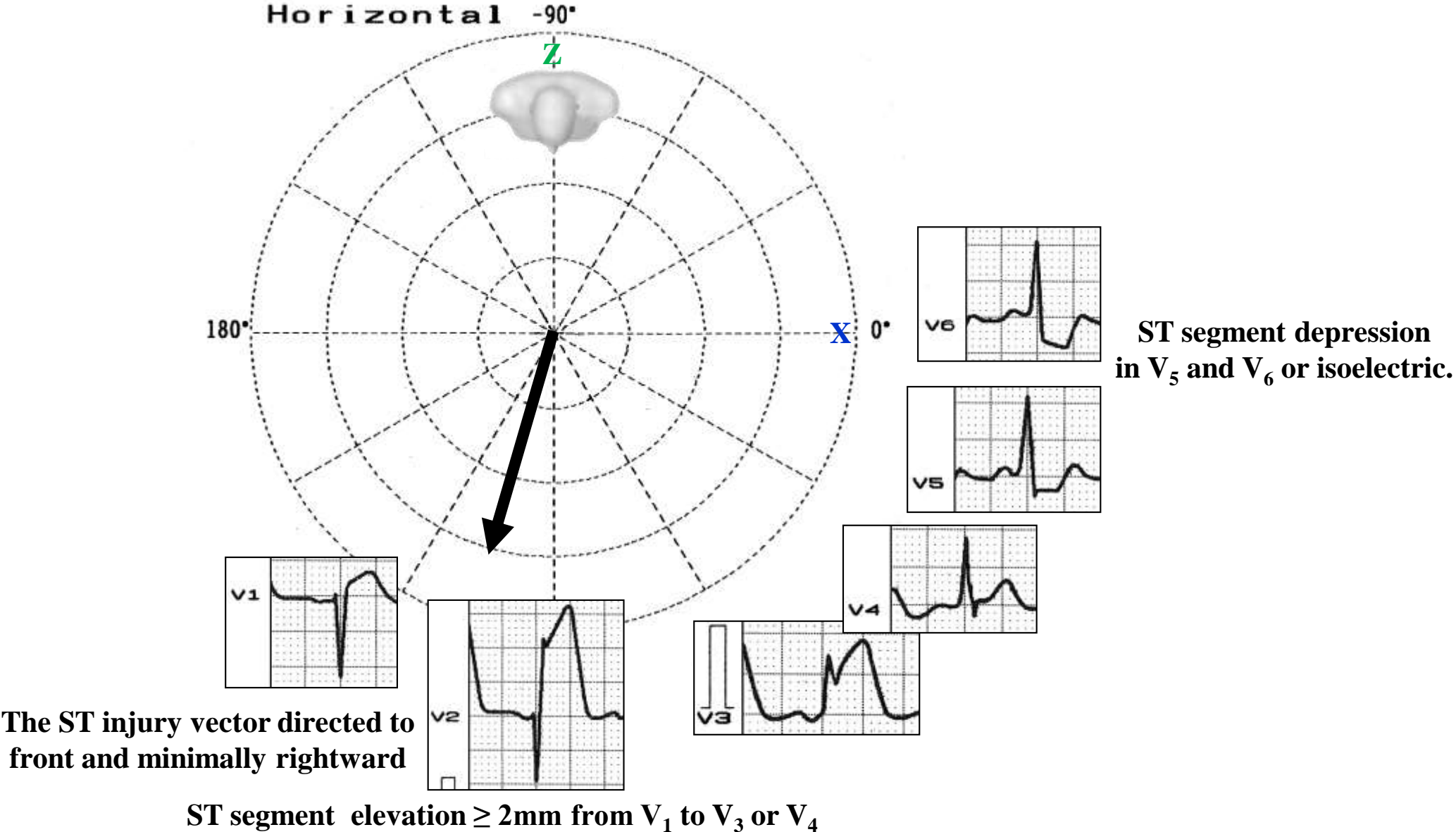
Why do we observe this pattern?

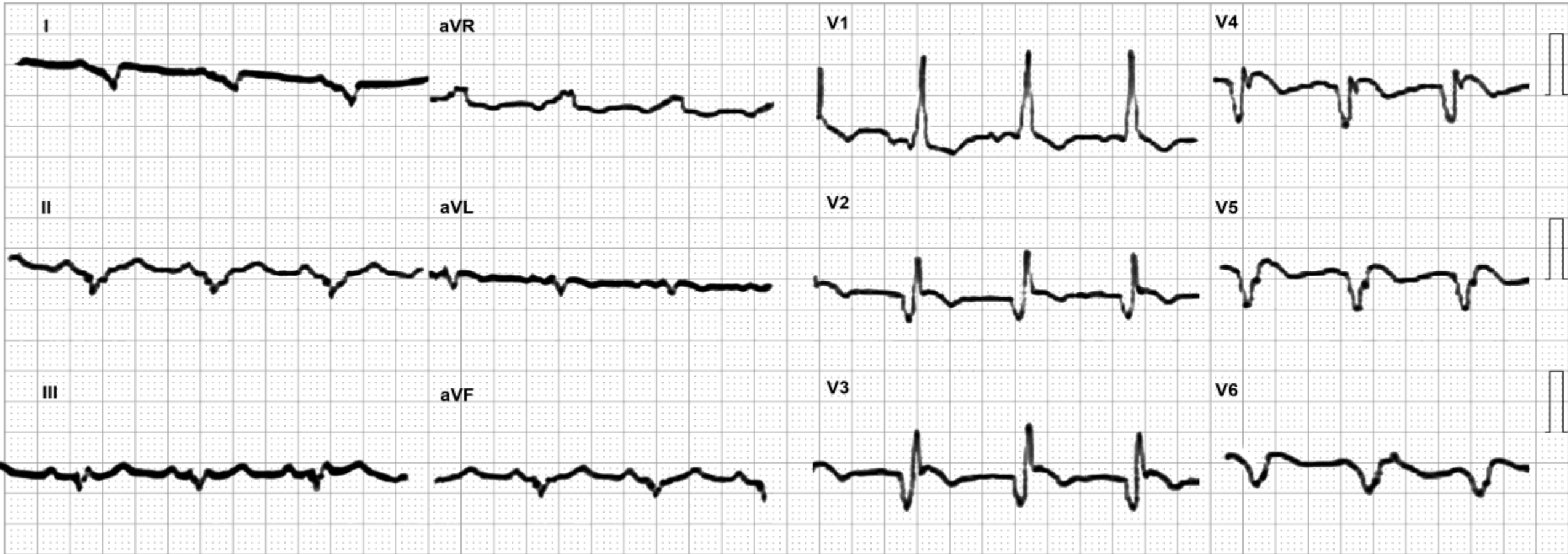
Because the ST injury vector is pointing to up



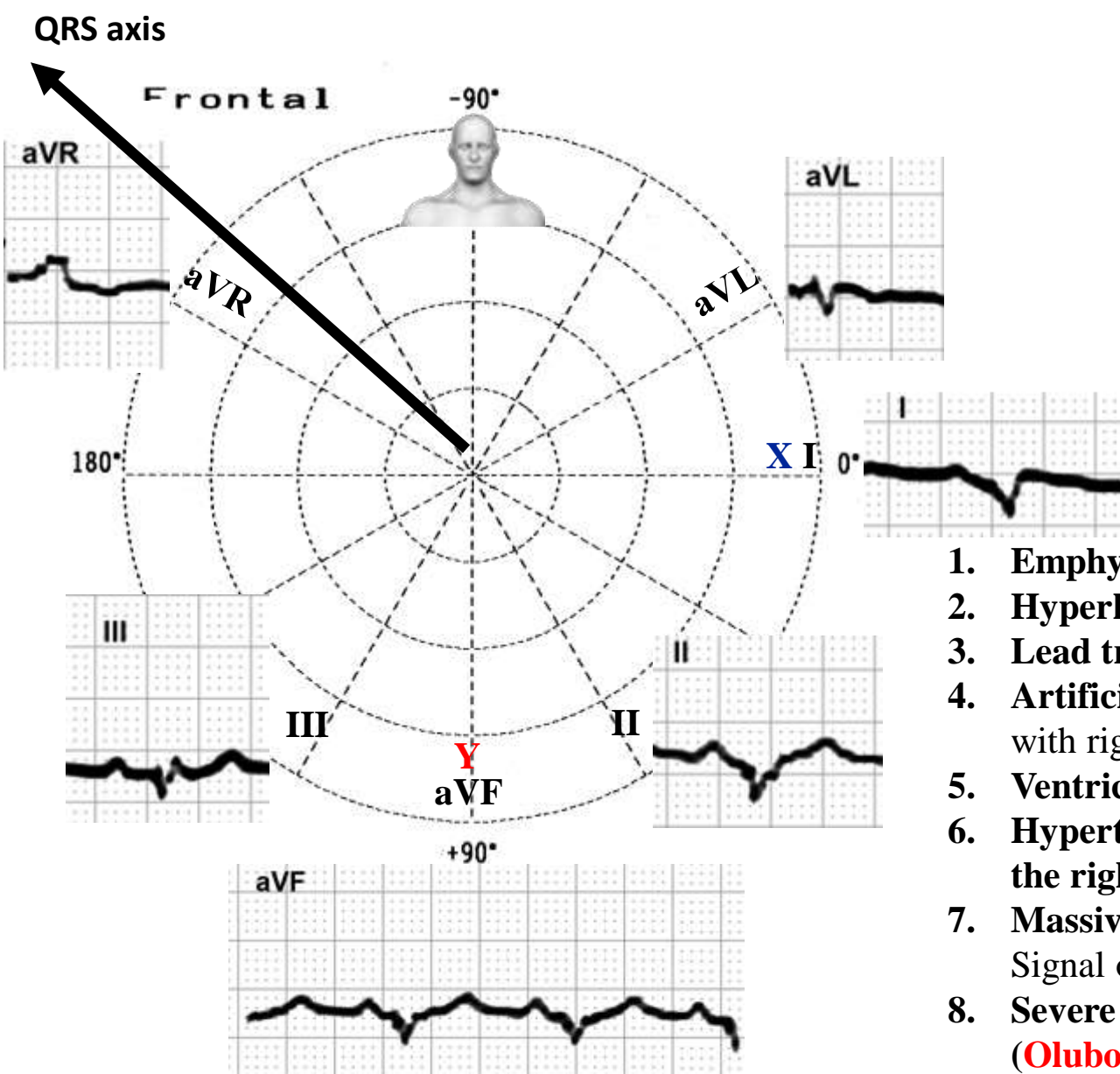
The ST injury vector is pointing to up, causing ST segment elevation in aVL and aVR and ST segment depression in inferior leads.

ST segment elevation $\geq 2\text{mm}$ from V_1 to V_3 or V_4 (ST segment injury vector directed to front). ST segment depression in V_5 and V_6 or isoelectric. Eventually CRBBB and/or LAFB and/or LSFB.





The ECG shows an extreme right axis deviation and RBBB (QRS axis on top right quadrant: 203°). The QRS axis deviation between $+90^{\circ}$ to $+180^{\circ}$ is considered as right axis deviation. It indicates that the right fascicular block, lateral myocardial infarction, right ventricular hypertrophy, ventricular pre-excitation, ventricular tachycardia, and ventricular ectopy are prone to right axis deviation. In this case, we reported RBBB and extreme right axis deviation in a patient with extensive anterior myocardial infarction. The pathological change of anterior infarction and extensive lateral myocardial infarction could change the axis [8]. And extensive lateral myocardial infarction with involvement of basal areas may cause an opposite electric vector, which may explain the presence of a positive main wave direction of QRS in lead aVR. New-onset extreme right axis deviation and right bundle branch block (RBBB) are rare during AMI, and has only been reported in several cases reflecting the severity of AMI. It could predict severe clinical complications and higher risks in CAD. Although there is little electrophysiological explanation, the complications are severe. They should be emphasized in newly diagnosed extreme right axis deviation and RBBB in AMI (**Wang 2018**). See next slide.....



	I	II	aVF
Normal Axis (0° to +90°)	+	+	+
LAD (physiological, 0° to -30°)	+	i	-
LAD (pathological, -30° to -90°)	+	-	-
RAD (+90° to 180°)	-	+	+
Extreme Axis (-90° to 180°)	-	-	-
Indeterminate Axis	Isoelectric (i)		

Causes of a Northwest axis (no man's land)

1. Emphysema
2. Hyperkalaemia
3. Lead transposition
4. Artificial cardiac pacing: Biventricular pacing is associated with right superior quadrant QRS axis (**Refaat 2011**)
5. Ventricular tachycardia
6. Hypertrophic cardiomyopathy with dominant hypertrophy in the right anterobasal region of the ventricular septum (**Matsubara 2000**)
7. Massive anterolateral myocardial infarction (**Liu 2014**) It is an Hazardous Signal of Poor Prognosis (**Wang 2018**)
8. Severe congestive cardiac failure of unidentified etiology in Nigerians (**Olubodun 1991**)

The QRS axis is located on right superior quadrant extreme right axis deviation (between -90° and ± 180°) (AKA “Northwest Axis”) or no Man’s Land axis: QRS complexes negative in I and aVF

Clinical Pearls

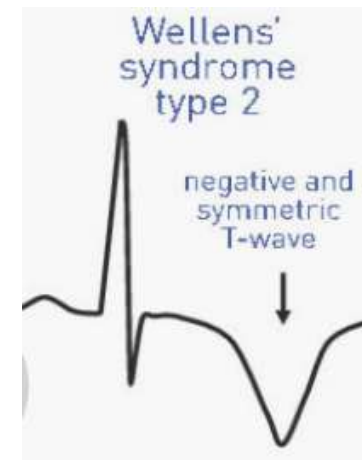
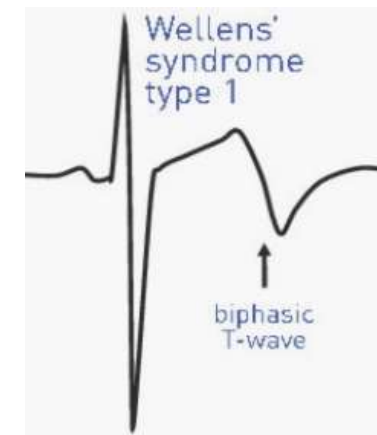
Other important ECG patterns to be aware of:

- a. Anterior-inferior STEMI due to occlusion of a “wraparound” LAD simultaneous ST elevation in the precordial and inferior leads due to occlusion of a variant (“type III”) LAD that wraps around the cardiac apex to supply both the anterior and inferior walls of the left ventricle. The vast majority (~80%) of inferior STEMIs are due to occlusion of the dominant right coronary artery (**RCA**). Less commonly (around 18% of the time), the culprit vessel is a dominant left circumflex artery (**LCx**). Occasionally, inferior STEMI may result from occlusion of a “type III” or “wraparound” left anterior descending artery (**LAD**). This produces the unusual pattern of concomitant inferior and anterior ST elevation.
- b. Left main coronary artery occlusion: widespread ST depression with ST elevation in aVR ≥ V1Wellens syndrome: deep precordial T wave inversions or biphasic T waves in V2-3, indicating critical proximal LAD stenosis (a warning sign of imminent anterior infarction) Wellens syndrome describes a pattern of ECG changes, particularly deeply inverted or biphasic T waves in leads V2-V3, that is highly specific for critical, proximal stenosis of the LAD coronary artery. It is alternatively known as anterior, descending, T-wave syndrome. Typically when patients with Wellens syndrome present to the emergency department they are pain-free, and usually cardiac enzymes are normal or only slightly elevated. However, it is important to recognize the ECG patterns as these patients are at high risk for impending large anterior wall acute myocardial infarction. In fact, when Drs. De Zwaan, Wellens, and colleagues first identified the syndrome in the early 1980s (**de Zwaan 1988**), they noted that 75% of patients with these ECG findings went on to develop acute, anterior, wall, myocardial infarction within weeks if they were treated with only medical management. Definitive treatment typically involves cardiac catheterization with percutaneous coronary intervention (PCI) to relieve the occlusion. Diagnostic criteria for Wellens syndrome are as follows: Deeply inverted T waves in leads V2 and V3 (may also be seen in leads V1, V4, V5, and V6) OR biphasic T waves (with initial positivity and terminal negativity) in V2 and V3 PLUSIsoelectric or minimally elevated ST segment, less than 1 mm (in other words, no signs of an acute anterior wall myocardial infarction), Preservation of precordial R-wave progression AND no precordial Q waves (in other words, no signs of old anterior wall infarct), recent history of angina, ECG pattern present in a pain-free state, normal or slightly elevated cardiac markers.

c. Two patterns of T waves can be seen in LAD occlusion:

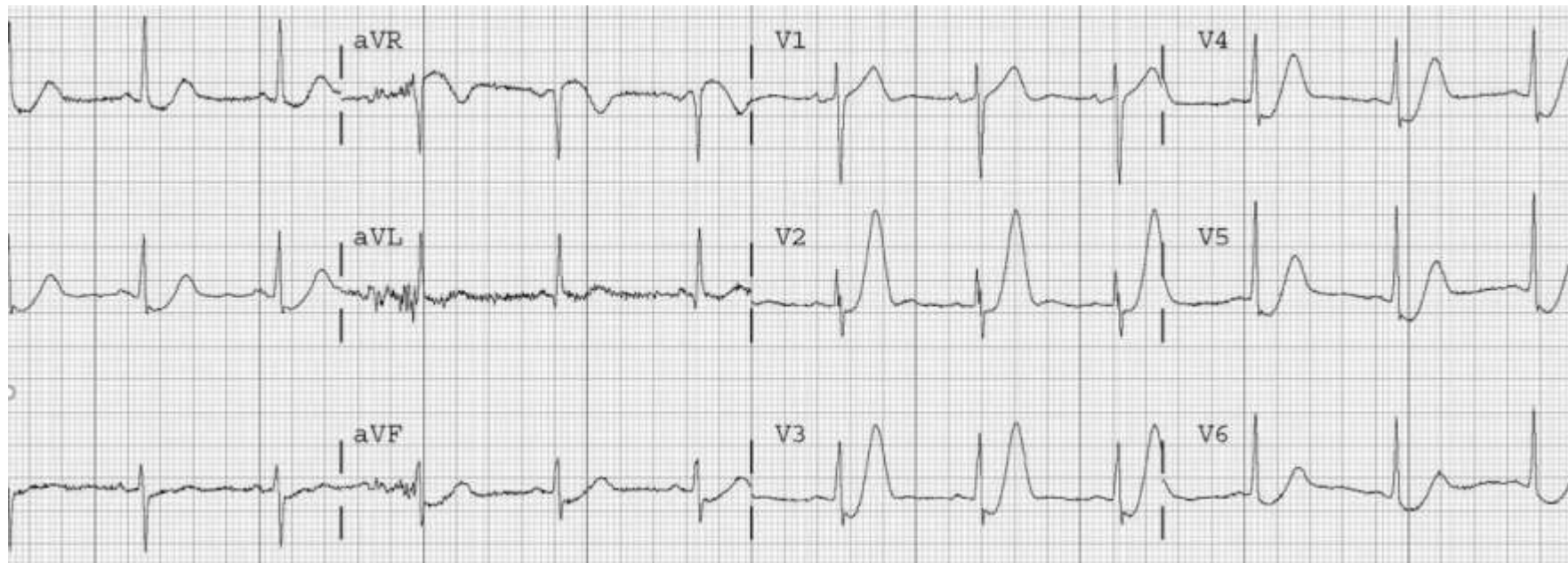
1. **Wellens syndrome:** Type-A T waves are biphasic, with initial positivity and terminal negativity. These T wave findings are present in approximately 25% of cases. Type-B T waves are deeply and symmetrically inverted. These findings are present in approximately 75% of cases. The 2 types of T waves found in Wellens syndrome exist on a spectrum of disease with type-1 T waves evolving into type-2 T waves. The T-wave abnormalities may be persistent, remaining in place for hours to weeks, even when the patient is pain-free. Wellens syndrome is not always an acute process. It can develop over days to weeks. The ECG pattern often develops when the patient is not experiencing chest pain. When the patient does experience chest pain, the ST segment and T-wave pattern can appear to normalize into hyperacute upright T waves (so-called “pseudo-normalization”) or even develop into ST-segment elevations. Cardiac biomarkers including troponin may be falsely reassuring in patients with Wellens syndrome as they frequently result within normal limits. In one prospective study, only 12% of patients with Wellens’ pattern on ECG had elevated cardiac enzymes, and these elevations were less than twice the upper limit of normal. The ECG of Wellens’ syndrome presents certain characteristics: the changes in the T-wave can be observed at rest, when chest pain has disappeared; the electrical abnormalities may be permanent or intermittent and therefore justify performing repeated electrocardiograms; there are usually no signs of prior infarction (Q-waves in the anterior territory or poor R wave progression); the ST-segment is generally isoelectric with no significant elevation or depression (< 1 mm); the changes in T-wave can take 2 forms: 1) type 1: observed in approximately 25% of Wellens’ syndrome cases; biphasic T-waves (initial positivity followed by negativity) most often in V2, V3 but occasionally in V1 and up to V5-V6; 2) type 2: this is the most common form; it is observed in about 75% of cases; negative, deep, rather narrow and symmetrical T-waves, usually in V2, V3 but often in V1 and V4 and sometimes up to V5-V6; in the absence of treatment, the pattern can evolve from type 1 to type 2 and thereafter to evocative signs of acute coronary syndrome with ST-segment elevation;

It is important to recognize a Wellens’ syndrome on the ECG (biphasic or negative T-waves in V2, V3 in absence of pain, without ST-segment elevation) in order to propose aggressive management and avoid the occurrence of anterior infarction by complete occlusion of the proximal LAD.



2. Precordial junctional ST-segment depression with tall symmetric T-waves or De Winter T-waves

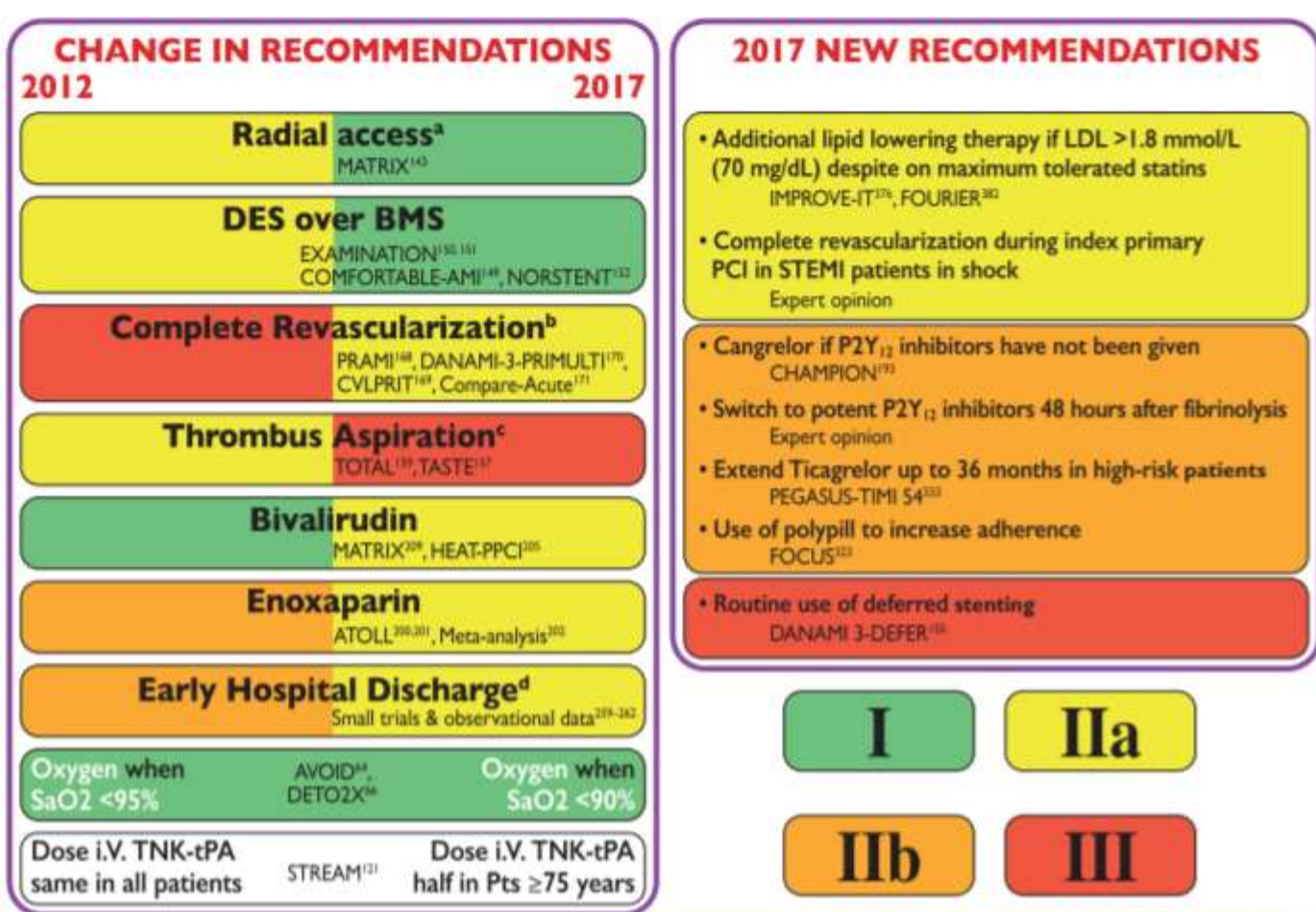
Upsloping ST depression with symmetrically peaked T waves in the precordial leads; a “STEMI equivalent” indicating acute LAD occlusion. Clinical Significance of De Winter T Waves: The de Winter ECG pattern is an anterior STEMI equivalent that presents without obvious ST segment elevation. First reported IN 2008 de Winter in 2008. Key diagnostic features include ST depression and peaked T waves in the precordial leads. The de Winter pattern is seen in ~2% of acute LAD occlusions and is under-recognized by clinicians. Unfamiliarity with this high-risk ECG pattern may lead to under-treatment (e.g. failure of cath lab activation), with attendant negative effects on morbidity and mortality. The prevalence of the junctional ST-depression followed by tall symmetrical T-waves in a field triage system for STEMI is unknown. De Winter et al. prospectively collected all transmitted 12-lead ECGs from the STEMI field triage system in Amsterdam from 2011 to 2013. ECGs with junctional ST-depression with tall symmetrical T-waves were recognized and angiographic documentation and clinical follow up were collected. A total of 5588 patients with at least 1 transmitted field ECG were identified from the database. ST-elevation infarction was present on the field ECG in 1864 patients (33%) and 701 ECGs (12,5%) showed anterior infarction. In 11 patients, junctional ST-depression with tall symmetrical T-waves was identified (0,2% of total transmitted ECGs and 1,6% of anterior infarctions). The 11 angiograms invariably showed involvement of the proximal LAD artery. Mortality was 27% within the first week. An ECG with junctional ST-depression with tall symmetrical T-waves is an infrequent finding. Because this pattern of STEMI equivalent is associated with LAD occlusions, it is important to recognize this pattern, so patients can be transported to the catheterization laboratory without delay (**De Winter 2018**).



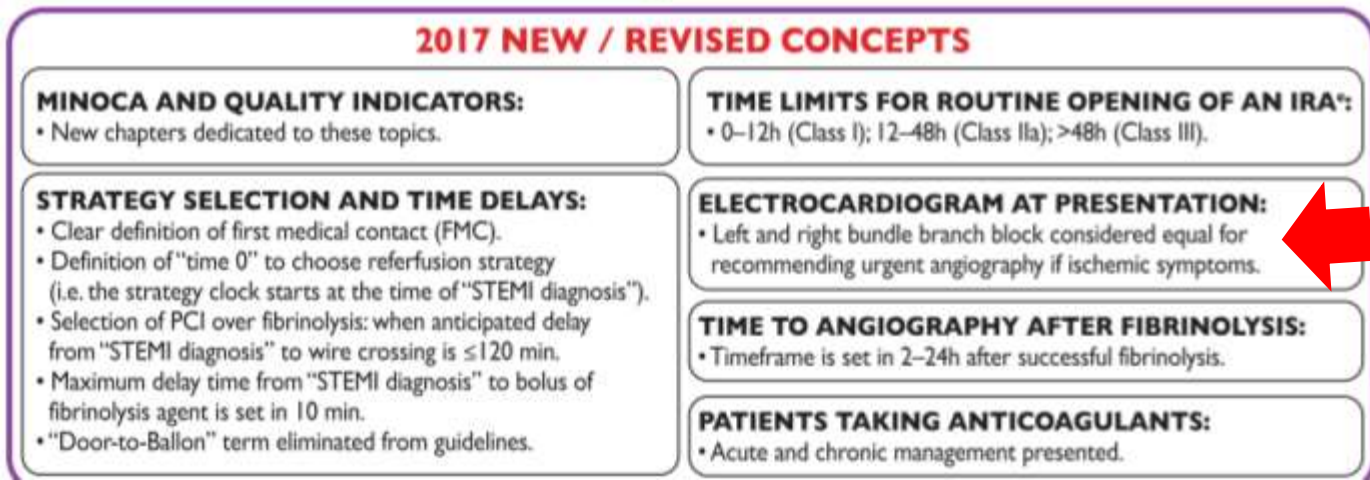
2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

Classes of recommendations	Definition	Suggested wording to use
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.	Is recommended/is indicated
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.	
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy.	Should be considered
Class IIb	Usefulness/efficacy is less well established by evidence/opinion.	May be considered
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective; and in some cases may be harmful.	Is not recommended

Level of evidence A	Data derived from multiple randomized clinical trials or meta-analyses.
Level of evidence B	Data derived from a single randomized clinical trial or large non-randomized studies.
Level of evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.



What is new in 2017 STEMI Guidelines. BMS = bare metal stent; DES = drug eluting stent; IRA = infarct related artery; i.v. = intravenous; LDL = low-density lipoprotein; PCI = percutaneous coronary intervention; SaO₂ = arterial oxygen saturation; STEMI = ST-elevation myocardial infarction; TNK-tPA = Tenecteplase tissue plasminogen activator. For explanation of trial names, see list of. a Only for experienced radial operators. b Before hospital discharge (either immediate or staged). c Routine thrombus aspiration (bailout in certain cases may be considered). d In 2012 early discharge was considered after 72h, in 2017 early discharge is 48–72h. e If symptoms or hemodynamic instability IRA should be opened regardless time from symptoms onset. In left and mid panels, below each recommendation, the most representative trial (acronym and reference) driving the indication is mentioned.



Recommendations	Class ^a	Level ^b
ECG monitoring		
12-lead ECG recording and interpretation is indicated as soon as possible at the point of FMC, with a maximum target delay of 10 min. ^{36,38}	I	B
ECG monitoring with defibrillator capacity is indicated as soon as possible in all patients with suspected STEMI. ^{44,45}	I	B
The use of additional posterior chest wall leads (V ₇ –V ₉) in patients with high suspicion of posterior MI (circumflex occlusion) should be considered. ^{8,46–49}	IIa	B
The use of additional right precordial leads (V ₃ R and V ₄ R) in patients with inferior MI should be considered to identify concomitant RV infarction. ^{8,43}	IIa	B
Blood sampling		
Routine blood sampling for serum markers is indicated as soon as possible in the acute phase but should not delay reperfusion treatment. ⁸	I	C

ECG = electrocardiogram; FMC = first medical contact; MI = myocardial infarction; RV = right ventricle; STEMI = ST-segment elevation myocardial infarction. a Class of recommendation. b Level of evidence.

Relief of hypoxaemia and symptoms

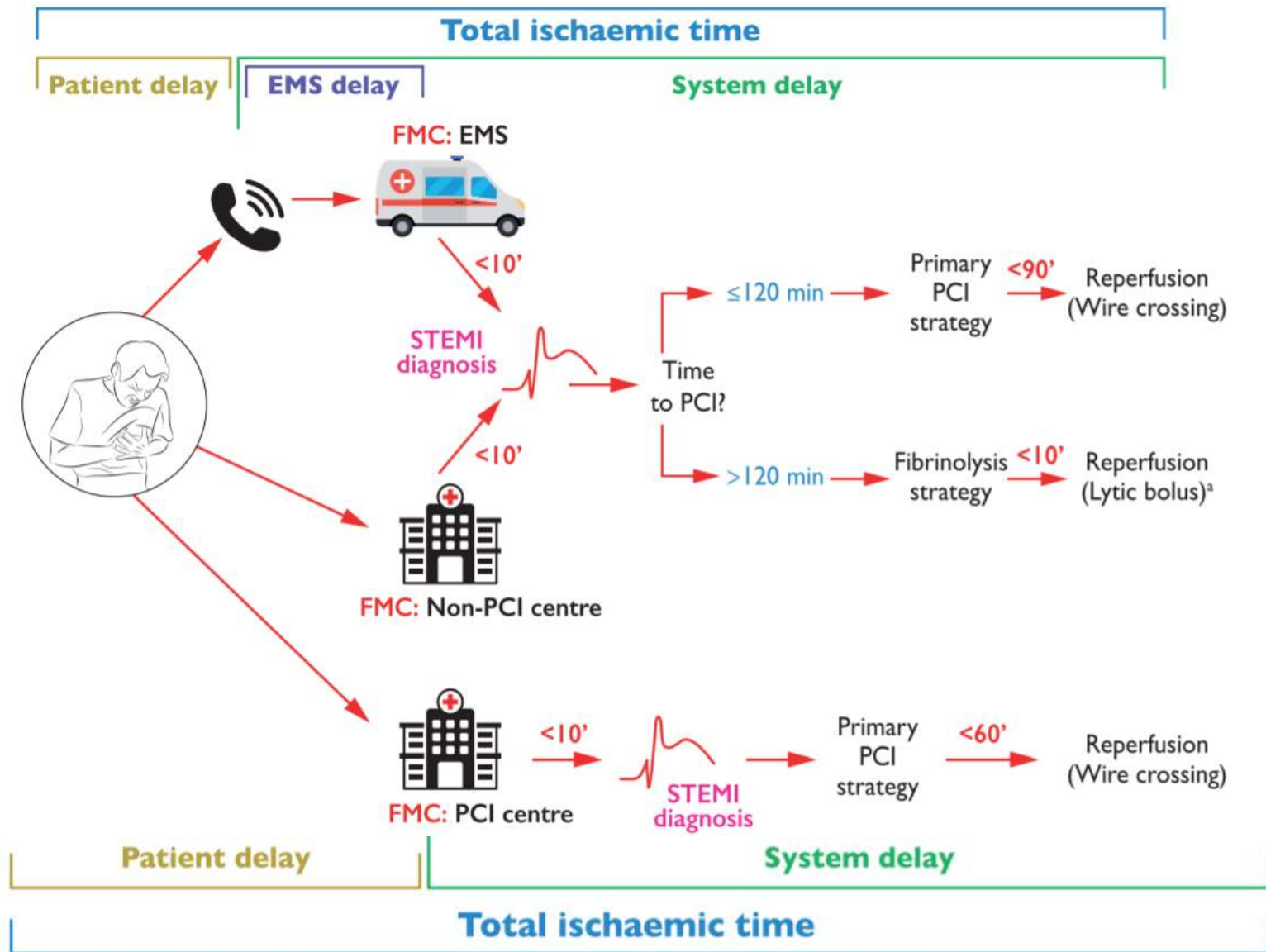
Recommendations	Class ^a	Level ^b
Hypoxia		
Oxygen is indicated in patients with hypoxaemia (SaO ₂ < 90% or PaO ₂ < 60 mmHg).	I	C
Routine oxygen is not recommended in patients with SaO ₂ ≥ 90%. ^{64–66}	III	B
Symptoms		
Titrated i.v. opioids should be considered to relieve pain.	IIa	C
A mild tranquillizer (usually a benzodiazepine) should be considered in very anxious patients.	IIa	C

i.v. = intravenous; PaO2 = partial pressure of oxygen; SaO2 = arterial oxygen saturation. a Class of recommendation. b Level of evidence.

Cardiac arrest

Recommendations	Class ^a	Level ^b
A primary PCI strategy is recommended in patients with resuscitated cardiac arrest and an ECG consistent with STEMI. ^{69–71,85}	I	B
Targeted temperature management ^c is indicated early after resuscitation of cardiac arrest patients who remain unresponsive. ^{77,78,80–82}	I	B
It is indicated that healthcare systems implement strategies to facilitate transfer of all patients in whom a MI is suspected directly to the hospital offering 24/7 PCI-mediated reperfusion therapy via one specialized EMS.	I	C
It is indicated that all medical and paramedical personnel caring for patients with suspected MI have access to defibrillation equipment and are trained in basic cardiac life support.	I	C
Urgent angiography (and PCI if indicated) should be considered in patients with resuscitated cardiac arrest without diagnostic ST-segment elevation but with a high suspicion of ongoing myocardial ischaemia. ^{69–71,73}	IIa	C
Pre-hospital cooling using a rapid infusion of large volumes of cold i.v. fluid immediately after return of spontaneous circulation is not recommended. ⁸⁶	III	B

24/7 = 24 h a day, 7 days a week; ECG = electrocardiogram; EMS = emergency medical system; i.v. = intravenous; MI = myocardial infarction; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction. a Class of recommendation. b Level of evidence. c Targeted temperature management refers to active methods (i.e. cooling catheters, cooling blankets, and application of ice applied around the body) to achieve and maintain a constant specific body temperature between 32 and 36 C in a person for a specific duration of time (most commonly used 24 h).



Modes of patient presentation, components of ischaemia time and flowchart for reperfusion strategy selection. EMS = Emergency Medical System; FMC = First Medical Contact; PCI = Percutaneous Coronary Intervention; STEMI = ST-segment elevation myocardial infarction. The recommended mode of patient presentation is by alerting the EMS (call national emergency number: 112 or similar number according to region). When STEMI diagnosis is made in the out-of-hospital setting (via EMS) or in a non-PCI centre, the decision for choosing reperfusion strategy is based on the estimated time from STEMI diagnosis to PCI-mediated reperfusion (wire crossing). System delay for patients alerting the EMS starts at the time of phone alert, although FMC occurs when EMS arrives to the scene. denotes minutes. a Patients with fibrinolysis should be transferred to a PCI centre immediately after administration of the lytic bolus

Logistics of pre-hospital care

Recommendations	Class ^a	Level ^b
It is recommended that the pre-hospital management of STEMI patients is based on regional networks designed to deliver reperfusion therapy expeditiously and effectively, with efforts made to make primary PCI available to as many patients as possible. ¹⁰⁰	I	B
It is recommended that primary PCI-capable centres deliver a 24/7 service and are able to perform primary PCI without delay. ^{18,103,104}	I	B
It is recommended that patients transferred to a PCI-capable centre for primary PCI bypass the emergency department and CCU/ICCU and are transferred directly to the catheterization laboratory. ^{92,107–110}	I	B
It is recommended that ambulance teams are trained and equipped to identify STEMI (with use of ECG recorders and telemetry as necessary) and administer initial therapy, including fibrinolysis when applicable. ⁹⁵	I	C

It is recommended that ambulance teams are trained and equipped to identify STEMI (with use of ECG recorders and telemetry as necessary) and administer initial therapy, including fibrinolysis when applicable.⁹⁵

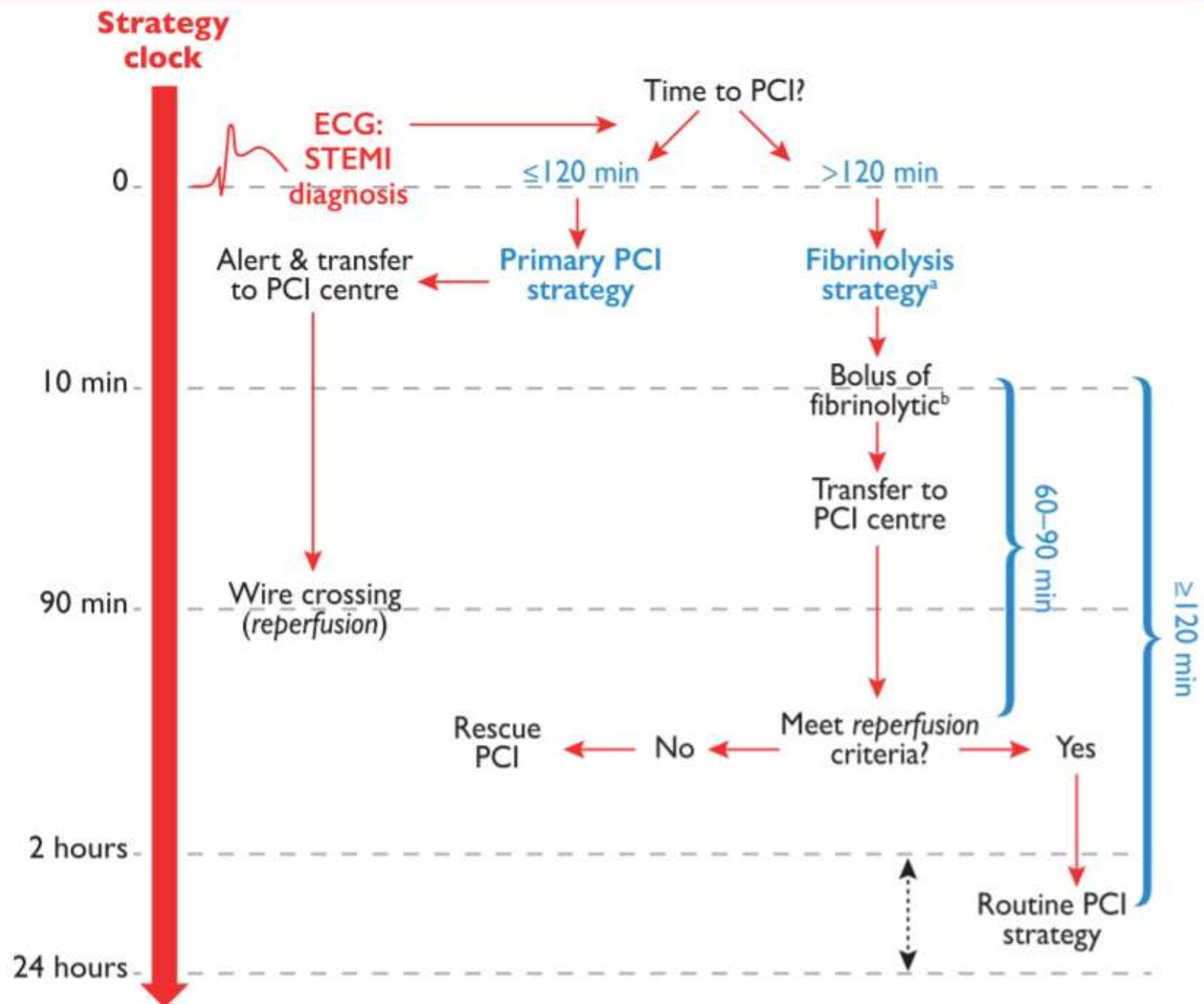
It is recommended that all hospitals and EMS participating in the care of patients with STEMI record and audit delay times and work to achieve and maintain quality targets.^{105–107}

It is recommended that EMS transfer STEMI patients to a PCI-capable centre, bypassing non-PCI centres.

It is recommended that EMS, emergency departments, and CCU/ICCU have a written updated STEMI management protocol, preferably shared within geographic networks.

It is recommended that patients presenting to a non-PCI-capable hospital and awaiting transportation for primary or rescue PCI are attended in an appropriately monitored area (e.g. the emergency department, CCU/ICCU, or intermediate care unit).

ECG = electrocardiogram; EMS = emergency medical system; FMC = first medical contact; IRA = infarct-related artery; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.



Maximum target times according to reperfusion strategy selection in patients presenting via EMS or in a non-PCI centre. ECG = electrocardiogram; PCI = Percutaneous Coronary Intervention; STEMI = ST-segment elevation myocardial infarction. STEMI diagnosis is the time 0 for the strategy clock. The decision for choosing reperfusion strategy in patients presenting via EMS (out-of-hospital setting) or in a non-PCI centre is based on the estimated time from STEMI diagnosis to PCI-mediated reperfusion. Target times from STEMI diagnosis represent the maximum time to do specific interventions. a if fibrinolysis is contra-indicated, direct for primary PCI strategy regardless of time to PCI. b 10 min is the maximum target delay time from STEMI diagnosis to fibrinolytic bolus administration, however, it should be given as soon as possible after STEMI diagnosis (after ruling out contra-indications).

Recommendations for reperfusion therapy				
Recommendation	Class ^a	Level ^b		
Reperfusion therapy is indicated in all patients with symptoms of ischaemia of ≤ 12 h duration and persistent ST-segment elevation. ^{119,138}	I	A	Early angiography (within 24 h) is recommended if symptoms are completely relieved and ST-segment elevation is completely normalized spontaneously or after nitroglycerin administration (provided there is no recurrence of symptoms or ST-segment elevation).	I C
A primary PCI strategy is recommended over fibrinolysis within indicated timeframes. ^{114,116,139,140}	I	A		I C
If timely primary PCI cannot be performed after STEMI diagnosis, fibrinolytic therapy is recommended within 12 h of symptom onset in patients without contraindications. ^{107,120,122}	I	A	In patients with time from symptom onset >12 h, a primary PCI strategy is indicated in the presence of ongoing symptoms suggestive of ischaemia, haemodynamic instability, or life-threatening arrhythmias. ¹⁴¹	Early angiography (within 24 h) is recommended if symptoms are completely relieved and ST-segment elevation is completely normalized spontaneously or after nitroglycerin administration (provided there is no recurrence of symptoms or ST-segment elevation).
In the absence of ST-segment elevation, a primary PCI strategy is indicated in patients with suspected ongoing ischaemic symptoms suggestive of MI and at least one of the following criteria present: - haemodynamic instability or cardiogenic shock - recurrent or ongoing chest pain refractory to medical treatment - life-threatening arrhythmias or cardiac arrest - mechanical complications of MI - acute heart failure - recurrent dynamic ST-segment or T-wave changes, particularly with intermittent ST-segment elevation.	I	C		
			In patients with time from symptom onset >12 h, a primary PCI strategy is indicated in the presence of ongoing symptoms suggestive of ischaemia, haemodynamic instability, or life-threatening arrhythmias. ¹⁴¹	I C
			A routine primary PCI strategy should be considered in patients presenting late (12–48 h) after symptom onset. ^{132,134,142}	IIa B
			In asymptomatic patients, routine PCI of an occluded IRA >48 h after onset of STEMI is not indicated. ^{135,137}	III A

Early angiography (within 24 h) is recommended if symptoms are completely relieved and ST-segment elevation is completely normalized spontaneously or after nitroglycerin administration (provided there is no recurrence of symptoms or ST-segment elevation).	I	C
In patients with time from symptom onset >12 h, a primary PCI strategy is indicated in the presence of ongoing symptoms suggestive of ischaemia, haemodynamic instability, or life-threatening arrhythmias. ¹⁴¹	I	C
A routine primary PCI strategy should be considered in patients presenting late (12–48 h) after symptom onset. ^{133,134,142}	IIa	B
In asymptomatic patients, routine PCI of an occluded IRA >48 h after onset of STEMI is not indicated. ^{135,137}	III	A

IRA = infarct-related artery; MI, myocardial infarction; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction. a Class of recommendation. b Level of evidence.

References

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