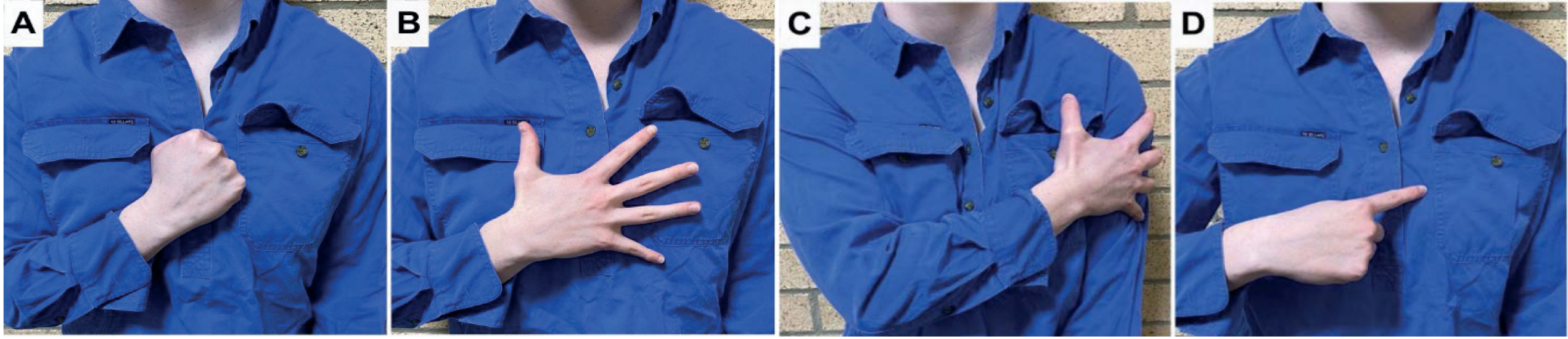
Case report

Paroxysmal regular narrow supraventricular tachycardia associated with typical MI chest pain, Levine´s signs#, repolarization disturbances and increased serum troponin

A 46-year-old woman without a history of recurrent palpitation was admitted in our emergency department. The patient complained of sudden, regular, rapid-onset palpitation associated with oppressive retrosternal precordial pain, without radiation or concomitants (dyspnea, nausea, vomiting or sweating). She places her clenched fist on her breastbone to describe her pain(positive Levine's sign). She denied hypertension, diabetes, not obese, not sedentary, no positive family history for CAD and no dependence on illegal substances use. Her 12-lead ECG at admission showed complex regular narrow sustained tachycardia associated with conspicuous ST segment elevation in aVR lead and diffuse ST segment depression in multiple leads. Figure 1 Uma mulher de 46 anos sem história de palpitações recorrentes foi admitida em nosso departamento de emergência. A paciente queixava-se de palpitações de inicio súbito, associadas a dor precordial retroesternal opressiva, sem irradiação ou concomitantes (dispneia, náuseas, vômitos ou sudorese). ela colocou o punho cerrado sobre seu esterno para descrever sua dor (sinal de Levine +). Negava hipertensão, diabetes, não era obesa, sedentária, sem história familiar positiva para DAC e não usava drogas ilícitas. Seu ECG de 12 derivações na admissão mostrava taquicardia sustentada estreita regular complexa associada a elevação conspícua do segmento ST na derivação aVR e depressão difusa do segmento ST em múltiplas derivações. figura 1 Mujer de 46 años sin antecedentes de palpitaciones previos recurrentes ingresó en nuestro servicio de urgencias. Refería palpitaciones de aparición súbita, asociadas a dolor torácico opresivo retroesternal, sin irradiación ni concomitantes (disnea, náuseas, vómitos o sudoración). Colocó su puño cerrado sobre su esternón para describir su dolor (signo de Levine +).Negaba hipertensión, diabetes, no era obesa, sedentaria, sin antecedentes familiares positivos para coronariopatia y no consumía drogas ilícitas. Su ECG de 12 derivaciones al ingreso mostró una taquicardia sostenida estrecha regular compleja asociada con una elevación del segmento ST notoria en la derivación aVR y depresión difusa del segmento ST en múltiples derivaciones. Figura 1



Patient gestures indicate origin of pain: a patient demonstrating A (the Levine sign), B (palm sign) or C (arm sign) is more likely to be experiencing ischemic cardiac pain than one showing D (pointing sign)
Source: adapted from Marcus et al (2007) 1
Gestures suggesting deep, poorly localized visceral pain (the Levine and palm signs) or pain radiating to the left arm (the arm sign) show CAD is more probable, whereas gestures indicating well-localized somatic pain (the pointing sign) mean ischemic cardiac pain is less likely (1).

1. **Marcus GM, Cohen J, Varosy PD, Vessey J, Rose E, Massie BM, Chatterjee K, Waters D. The utility of gestures in patients with chest discomfort. Am J Med. 2007 Jan;120(1):83-9. doi: 10.1016/j.amjmed.2006.05.045. PMID: 17208083**

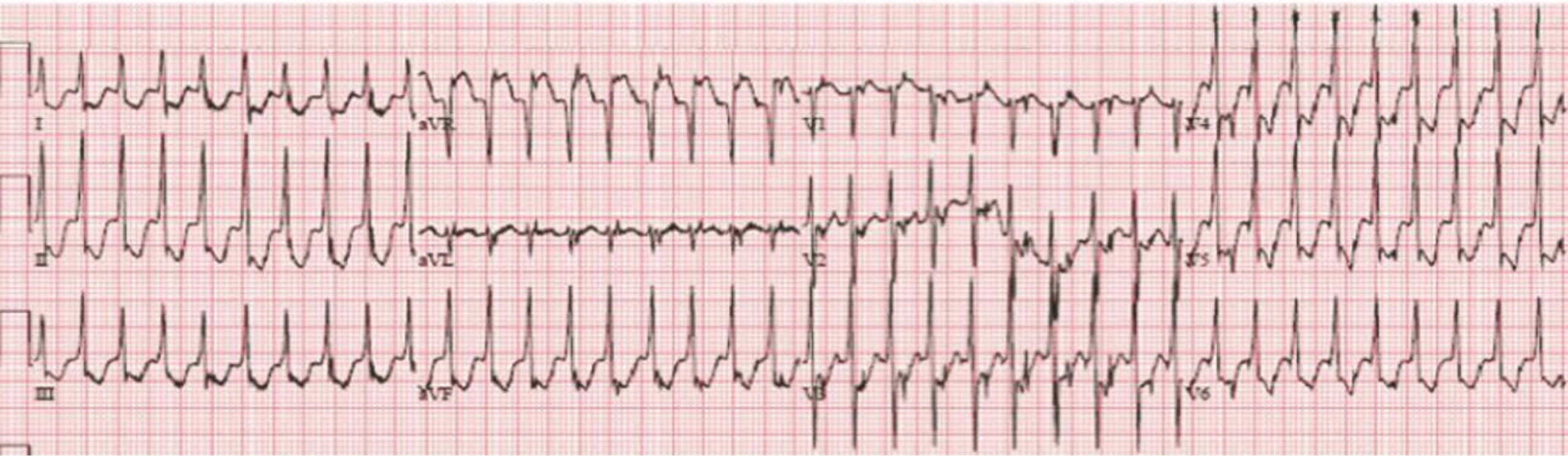
Levine's sign is **a universal sign of ischemic chest pain**, defined as an individual holding a clenched fist over the chest that has a low sensitivity but is relatively specific for ischemia

El signo de Levine es un signo universal de dolor torácico isquémico, definido como un individuo que sostiene un puño cerrado sobre el pecho. El signo tiene una sensibilidad baja pero es relativamente específico para la isquemia

 # O sinal de Levine é um sinal universal de dor torácica isquêmica, definido como um indivíduo segurando um punho cerrado sobre o peito. O sinal possui baixa sensibilidade, mas é relativamente específico para isquemia

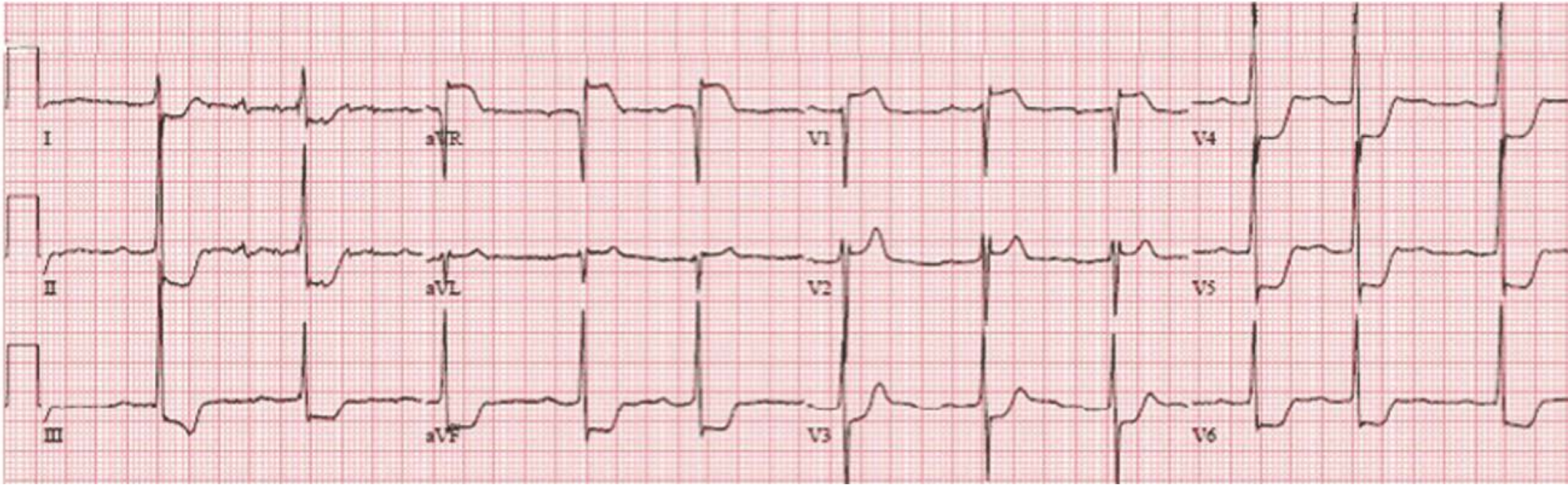
 # Mahsa Mohammadian 1, Dhaval Shah 2, Melvin Santana 2, Sherif Elkattawy 1, Shruti Jesani 3 Levine's Sign Points to Spontaneous Coronary Artery Dissection in a Healthy Young Male Cureus . 2022 May 10;14(5):e24893. doi: 10.7759/cureus.24893. eCollection 2022 May.

12-lead ECG at admission



After 10 minutes of admission the patient spontaneously reverted to sinus rhythm first degree AV block, however the chest pain did not resolve. A post resolution ECG was done

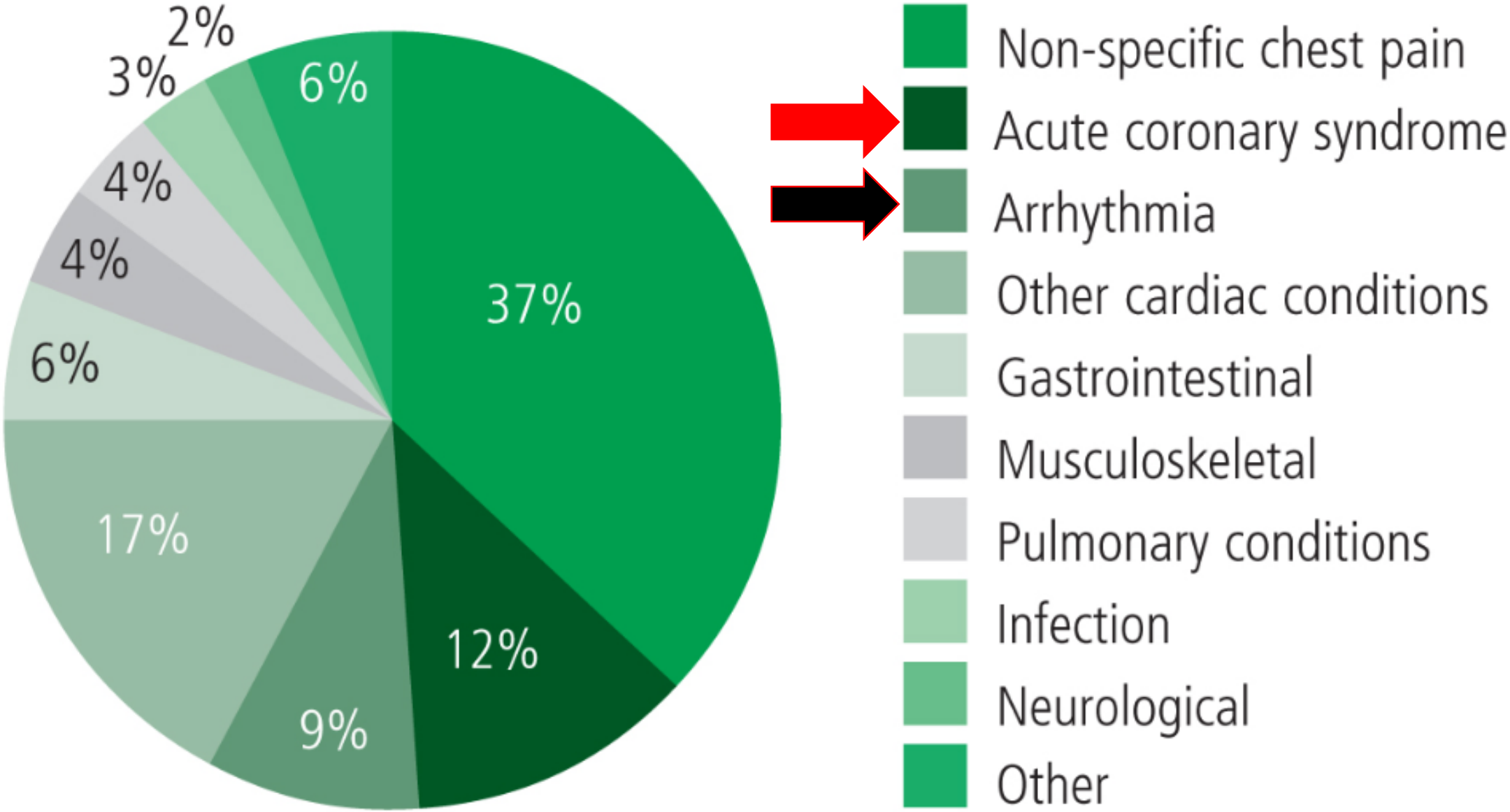
Figure 2.



What is the possible explanation about theses repolarization disturbance?

Qual a possível explicação para esse distúrbio de repolarização?

¿Cuál es la posible explicación de estas perturbaciones de la repolarización?



Distribution of chest pain conditions in an emergency department (adapted from Bjørnsen et al, 2019 .

Bjørnsen LP, Naess-Pleym LE, Dale J, Grenne B, Wiseth R. Description of chest pain patients in a Norwegian emergency department. Scand Cardiovasc J. 2019 Feb;53(1):28-34. doi: 10.1080/14017431.2019.1583362.)

Colleagues oppinions

Dear Andrés, Thank you for sharing this interesting case. The narrow complex, regular supraventricular tachycardia (230 bpm) suggests atrial flutter with 1:1 conduction. The ST-T wave pattern of multiple ST segment depression in ≥ 7 leads with ST elevation in aVR greater than in Vl is the pattern of circumferential sub endocardial ischemia. This pattern has been reported in persons with partial left main occlusion (or severe three vessel coronary artery disease). The absence of coronary risk factors in this 46 year old woman raises the possibility of coronary artery spasm, but the increased serum troponin and continued ST abnormalities after returning to sinus rhythm indicates she has had an acute myocardial infarction. Acute myocardial infarction can also be due to a sudden reduction in oxygen delivery to the myocardium. In this case the duration of SVT at 230 bpm must also be considered as the precipitating event. I look forward to your comments and those of our colleagues.

I'm wishing you and your family and our colleagues a wonderful holiday season.

Regards,

Frank G. Yanowitz MD Salt Lake City, UT

Comment: Frank is my virtual friend for many years, I consider him to be one of the most important electrocardiographers around the world

Andrés

Dear Andres:

I would like to be intrepid and propose a single diagnosis (I do recognize that there are several possible diagnoses for explaining this case).
But as I told you, I would like to suggest a single diagnosis as the tachycardia is concerned.:
Rapid (#240/min) AV Reentrant tachycardia involving a left posterolateral accessory pathway
Is CAD also present for explaining the major “pseudo-ischemic” changes?
This is possible but not mandatory.
I have already seen such pseudo-ischemic changes associated with normal coronary arteries.
Warm regards,
Prof. Bernard Belhassen,. Director, Cardiac Electrophysiology Laboratory.
Tel-Aviv Sourasky Medical Position: ProfessorEmeritus Location: Jerusalem, IL

Prof. Bernard Belhassen graduated at University Paris VII (France) and completed his training in cardiac electrophysiology in Fernand Widal Hospital (Paris) under the direction of Prof. Gilbert Motté. Between 1978-2015, he directed the cardiac electrophysiology laboratory at Tel-Aviv Medical Center. In 1983-1984 he was visiting Professor at Hahnemann University, Philadelphia. He is presently working at the Heart Institute, Hadassah University Hospital, Jerusalem, and serving as Professor Emeritus at Tel-Aviv University. He is the author of over 236 medical publications in leading journals including those on SABRUS with the outstanding cooperation of Drs Anat Milman, Yoav Michowitz and many co-authors.

Hello. I completely agree with Frank's interpretation. The ECG post-tachycardia represents a typical example of global ischemia, or, as named by our friend Samuel Sclarovsky, circumferential subendocardial ischemia: clear ST depression in eight leads with the maximal ST depression (and inverted T waves) in V4-V5, ST elevation greater in aVR than V1. Post-tachycardia ST depression does not necessarily indicate coronary artery disease, but in my opinion, such severe ST changes as in this case, are very rare as post-tachycardia changes. I think the patient had left main or left main equivalent disease, or 3-vessel disease. At least, this has to be excluded. Are we sure that the patient had not undergone radiotherapy for breast cancer??

Best regards **Kjell Christer Nikus MD PhD**

Dear collages Dr Nikus is fantastic Finland Professor of internal medicine/Professor in Cardiology at Heart Center, Tampere University Hospital Tampere, Finland. He has 324 Publications (Nikus CK), 40,309.Reads 6, 273 Citations 8, 8,337 We have wrote together 44 publications t indexed to Pubmed. I feel very honored with this partnership. I learned a lot from the "ice man"

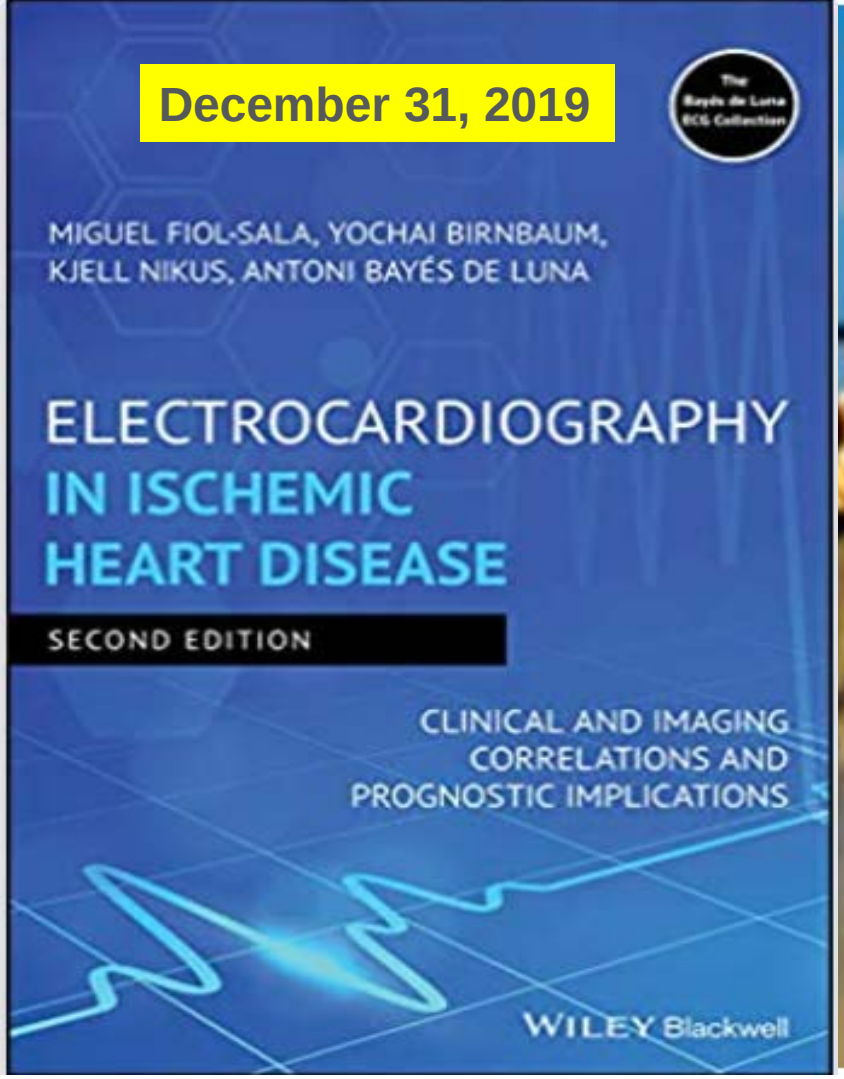
Thank you Dear Nikus for your friendship

Andrés Perez-Riera

Observation: The "man from the land of ice"

and with the happiest inhabitants on the planet

Pictures of Romantic Walk in Tampere



Skills and Expertise

Acute Myocardial Infarction | Interventional Cardiology | ECG | Electrocardiography | Myocardial Infarction | Electrocardiogram | Cardiovascular | Cardiology | Arrhythmias | Cardiovascular Disease

Final comments by
Andrés Ricardo Pérez-Riera

The ECG shows ST elevation in aVR, V_1 and V_2, consequently ischemic vector pointed to “Norwest quadrant”. There is also significant ST depression in multiple leads.

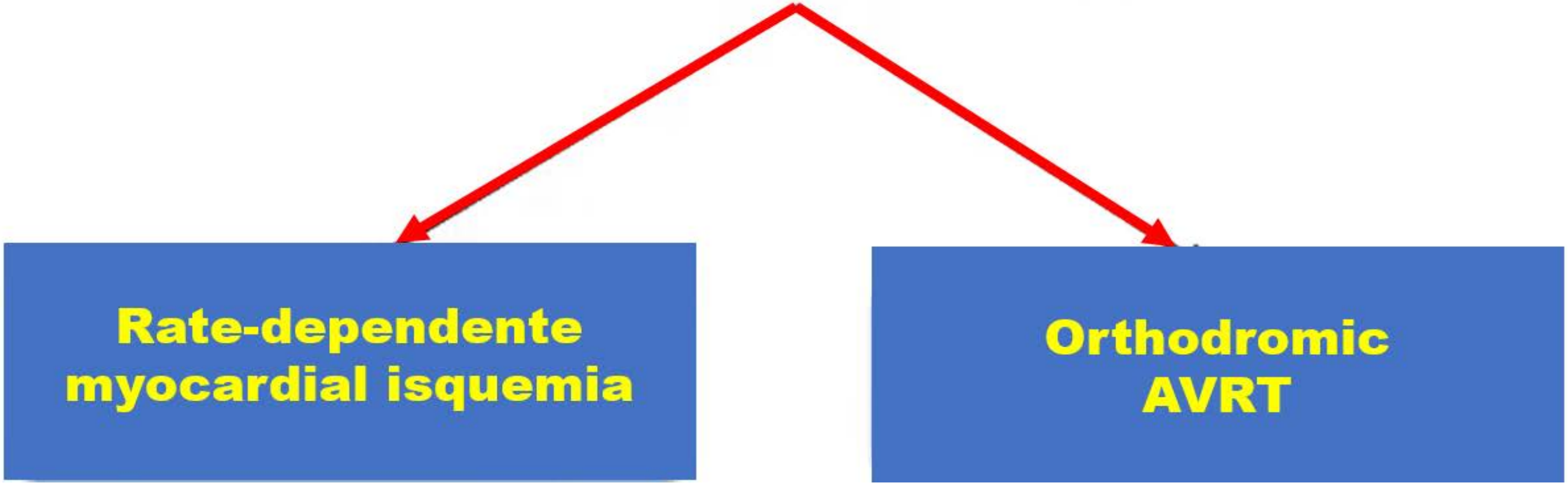
Is this a ST elevation MI? Remember the pain was ongoing. Usually ST elevation in aVR and V1 with other ST depressions may indicate LMCA critical occlusion or very proximal LAD or or severe three vessel coronary artery disease .

ST depression during SVT

- Oxygen supply-demand mismatch
- Unmasking of underlying CAD (Poor man stress ECG **Poor-man's stress test?**) 1
- Repolarization abnormality (especially with orthodromic AVRT)

1 **Martinez-Lopez JI. Poor-man's stress test? J La State Med Soc. 1980 Jan;132(1):15-6.**

ST depression during SVT



- ❑ Is there chest pain or not?
- ❑ Check resting ECG after termination of PSAVT:
- ❑ Persistent ST segment depression?
- ❑ Monthly secondary to ongoing myocardial ischemia?
- ❑ Right ventricular hypertrophy?

- ❑ **Há dor no peito ou não?**
- ❑ **Verifique o ECG em repouso após o término da PSVT Depressão persistente do segmento ST?**
- ❑ **Mensalmente secundário a isquemia miocárdica em curso?**
- ❑ **Hipertrofia ventricular direita?**

What would you do?

In this case the cath lab was activated. The patient had normal coronary arteries and a LVEF of 70%. There was no dissection of the ascending aorta. (**Congratulation Bernard!!!**) The troponin (cTnI) was elevated. The ECG resolved and was normal by the next day. Increase of serum troponin I and ST-segment depression are both markers of myocardial ischemia/injury. Abnormalities of the 2 indicators have been associated with SVT but their relevance for diagnosing ACS and the presence of CAD in this setting have not been clarified. Therefore, Mahajan N et al sought to evaluate the frequency of CAD based on increased troponin I and ST-segment depression during SVT. During a 5-year period, 104 patients were admitted with a diagnosis of SVT, 80 of whom had troponin I testing, and 70 of these patients could be assessed for ST-segment changes. 37 patients (48%) had increased troponin I (mean 1.54 +/- 2.7 ng/dl, normal <or=0.07 ng/dl) and 46 patients (57%) had ST-segment depression ≥1.0 mm. There were no

significant differences in baseline characteristics and clinical presentation of patients with and without troponin I increase or ST-segment depression. There was no difference in the diagnosis of CAD by noninvasive or invasive testing in patients with and without increased troponin I. More patients with than without ST-segment depression had evidence of CAD (22% vs none, p = 0.01), but after adjusting for covariates, ST-segment depression was not a significant predictor of CAD. In conclusion, increased troponin I and ST-segment depression are not significant markers of ACS in patients with SVT. A high cTnI level however is not synonymous to ACS (**Mahajan N, Mehta Y, Rose M, Shani J, Lichstein E. Elevated troponin level is not synonymous with myocardial infarction.** ***Int J Cardiol.*** **2006; 111:442–449. doi: 10.1016/j.ijcard.2005.08.029.**). Patients with normal coronary arteries may have elevated cTnI because of other diseases such as severe congestive heart failure, myocarditis, cerebrovascular accident, renal insufficiency and so on (**Bukkapatnam RN, et al. Relationship of myocardial ischemia and injury**

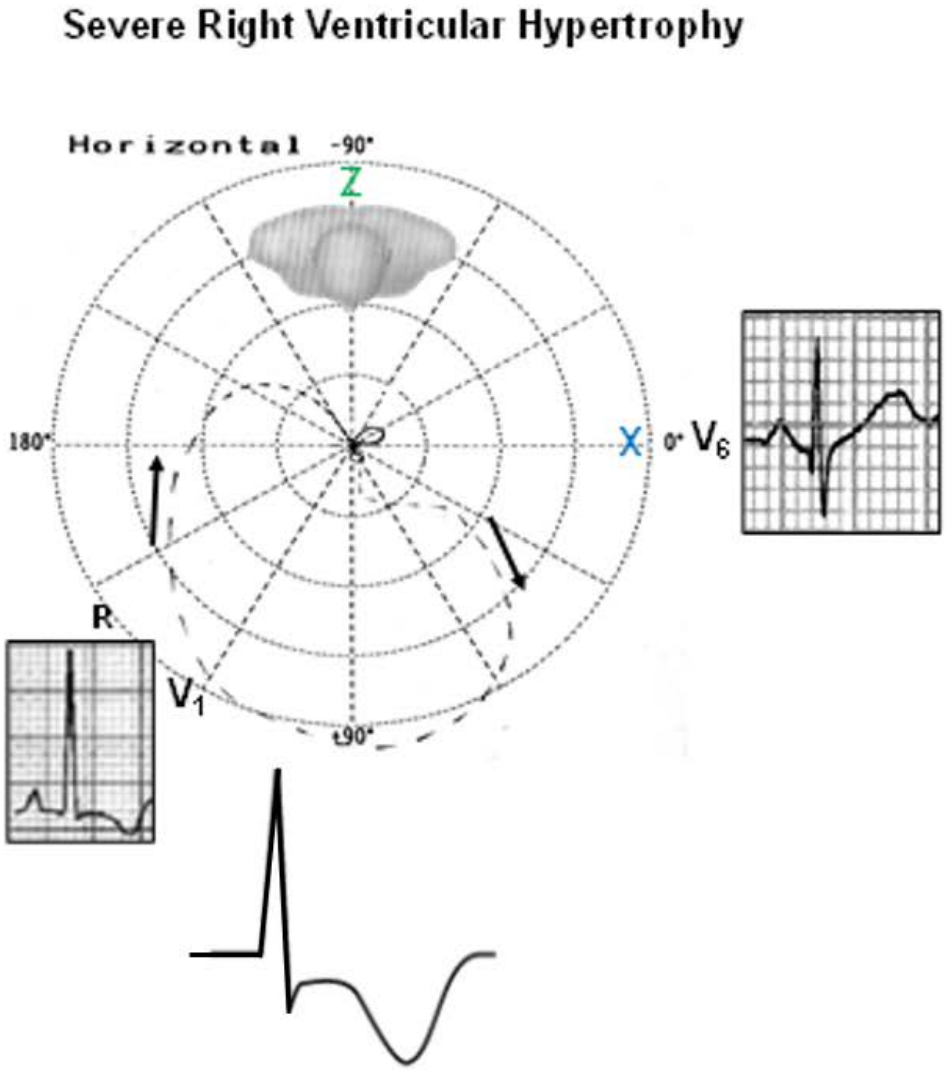
to coronary artery disease in patients with supraventricular tachycardia. 2010 Aug 1;106(3):374-7. doi: 10.1016/j.amjcard.2010.03.035.). Zellweger MJ et al has been reported that patients suffering from paroxysmal

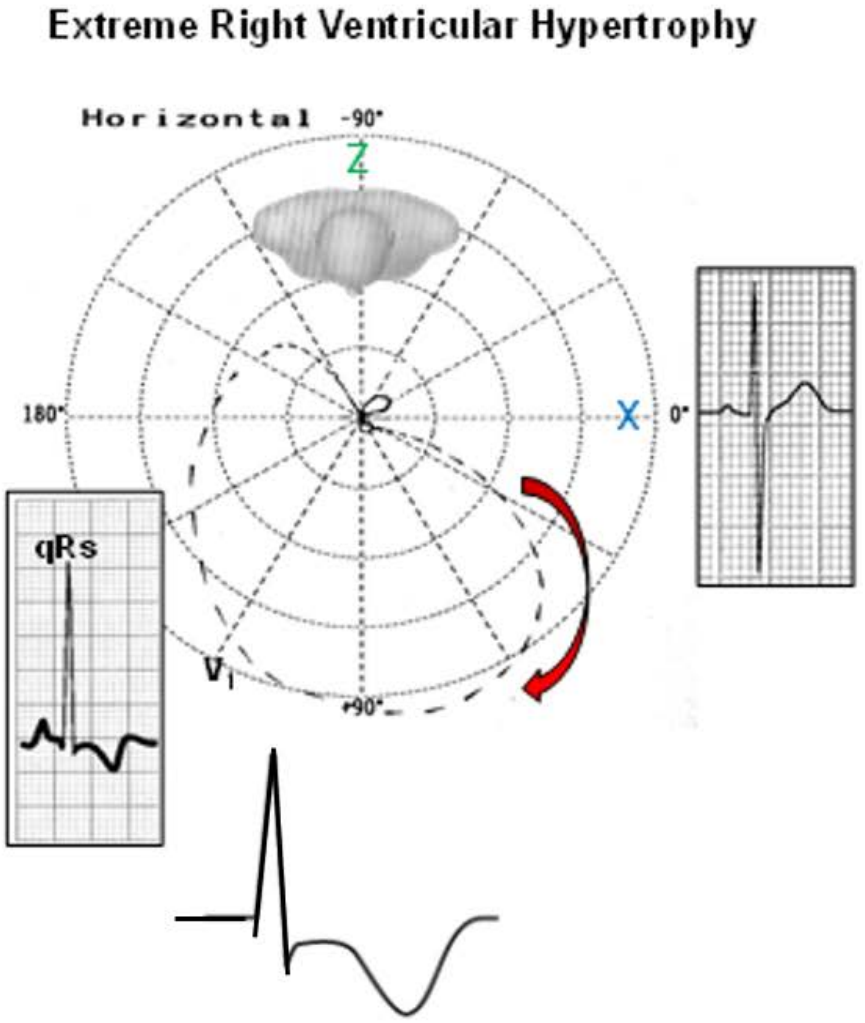
PSVT had elevated troponin levels (**Zellweger MJ, Schaer BA, Cron TA, Pfisterer ME, Osswald S. Elevated troponin levels in absence of coronary artery disease after supraventricular tachycardia. *Swiss Med Wkly.* 2003; 33:439–441**.). Coronary angiography is often performed in some patients presenting with Paroxysmal SVT because they have chest pain and high troponin levels.

Here, we reported one cases of PSVT-induced elevations in cTnI diffuse ST segment depression in multiples leads associated with ST segment elevation in aVR in the absence of CAD.

Conclusion: ST depression and a Troponin leak are not unusual in SVT. However, chest pain may signify a different patient group, ie., those with CAD. ST elevation, other than in aVR, should always be a cause for concern.

- **Right ventricular hypertrophy (RVH) on right precordial leads**

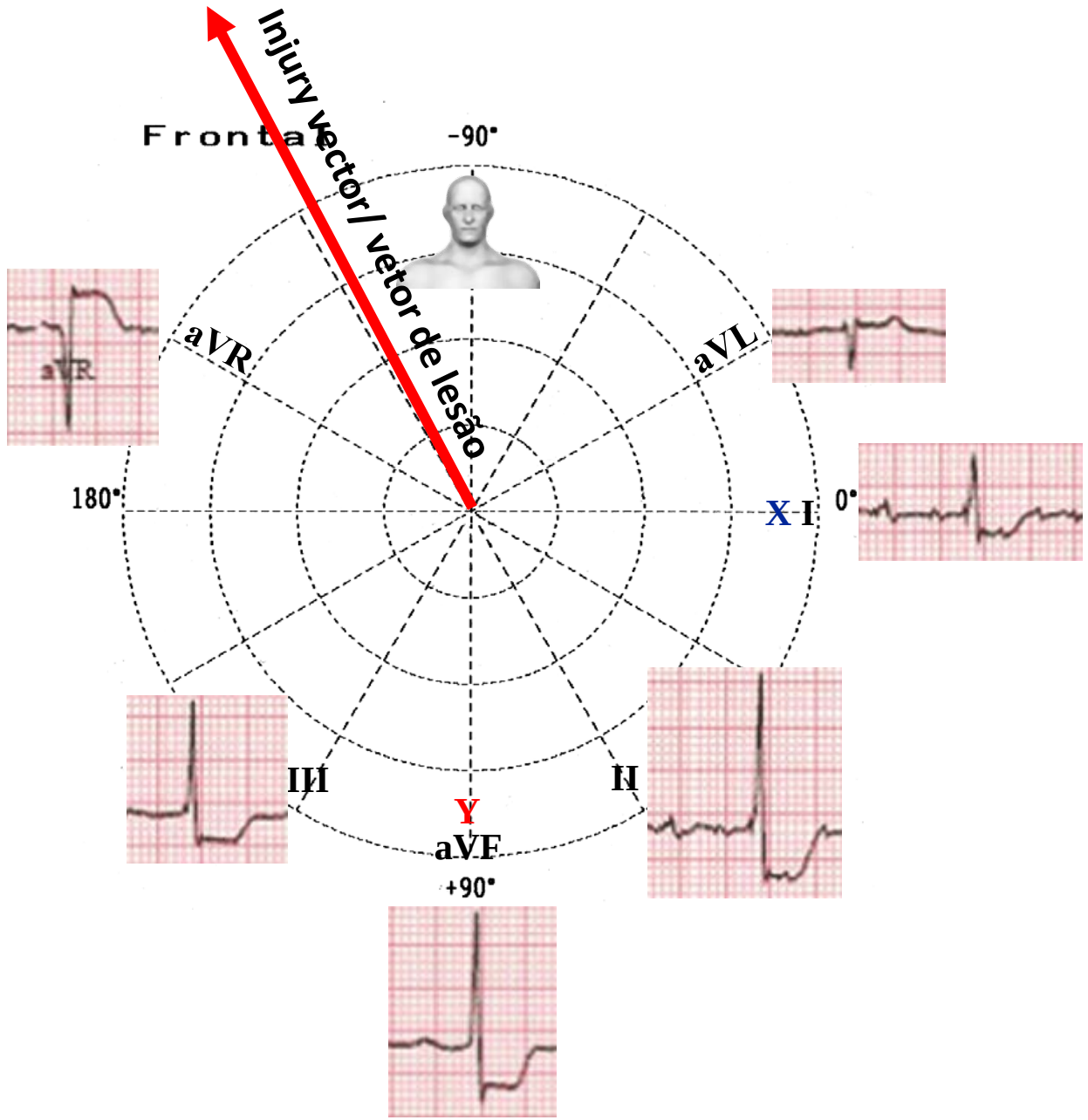


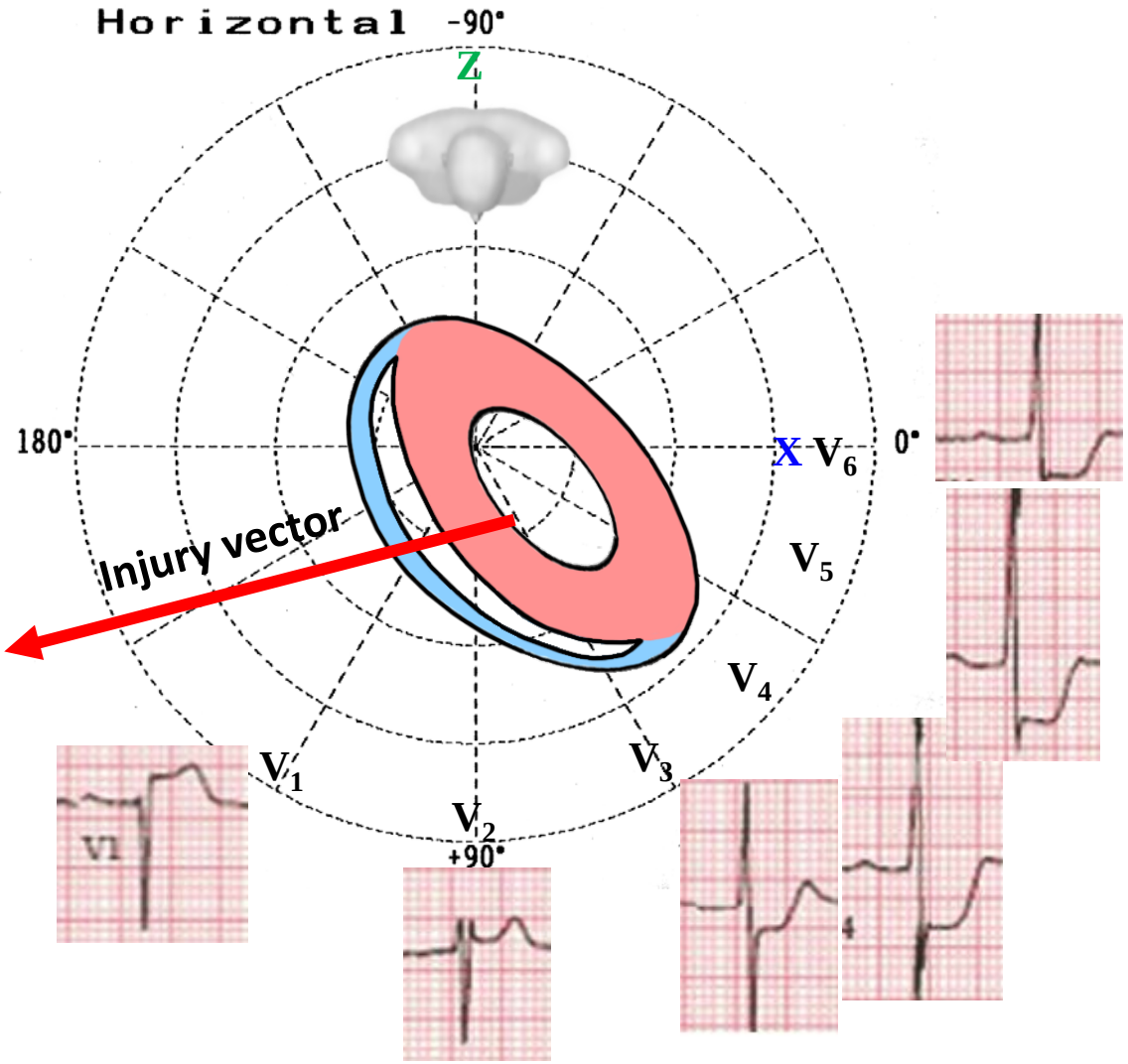


RVH with large R-waves, ST-segment depression V_1 to V_3 followed by negative T-waves. In case of chest discomfort, one must consider possibility of reciprocal image of lateral and high lateral transmural ischemia/infarction as a differential diagnosis.

Injury vector located on rigth superior quadrant

ECG after event





ST elevation in aVR with co-existent multi-lead ST depression indicates subendocardial ischaemia due to O_2 supply/demand mismatch.

Clinical causes include:

1. Left Main Coronary Artery (LMCA) suboccusion
2. LADA suboccusion proximal to the first septal perforator branch(S_1),
3. Severe triple vessel disease
4. Hypoxia or hypotension following resuscitation from cardiac arrest
5. Diffuse sub endocardial ischemia, with ST depression in the lateral leads producing reciprocal change in aVR (most common)
6. Infarction of the basal septum, i.e. a STEMI involving aVR. *The basal septum is supplied by the first septal perforator artery* S_1 *(a very proximal branch of the LAD), so ischemia / infarction of the basal septum would imply involvement of the proximal LAD.* The lead aVR is oriented to 'look' at the right upper side of the heart, and can **provide specific information about the RVOT and basal part of the septum**
7. **Atrial flutter with rate-related ST depression:** Tachycardia and flutter waves in V2 indicate atrial flutter with a 2:1 block ST depression is seen in I, aVL and V4-6, with reciprocal ST elevation in Avr
8. The degree of elevation of the ST segment in aVR correlates with mortality and its relationship to the ST elevation in V1 can give clues as to the location of the lesion. ST elevation in aVR of $\geq$ 1mm in the presence of non-ST elevation in the precordial leads has a sensitivity of 80% and a specificity of 93% for LMCA or triple vessel **disease(Kosuge M et al. An early and simple predictor of severe left main and/or three vessel disease in patients with non-ST-elevation acute coronary syndrome. Am J Cardiol Feb 15 2011Vol 107, issue 4:495-500**)

When the ST elevation in aVR ≥ V1, it is more likely to be a LMCA rather than LAD **occlusion(Yamaji H et al. Prediction of acute left main coronary artery obstruction by 12 lead electrocardiography: ST segment elevation in lead aVR with less ST segment elevation in lead V1. J Am Coll Cardiol November 2001. Vol 38, Issue 5: pp1348-1354**)

LMCA "occlusion": a misnomer

ST elevation in aVR with coexistent multi-lead ST depression can be a sign of Non-Occlusion Myocardial Infarction (NOMI) due to severe single or multi-vessel disease, but does not usually represent acute LMCA occlusion as once thought. Such acute occlusion most often causes SCD due to simultaneous anterior, lateral and basal STEMI. Ahmed et al.. recognise this ECG pattern as consistent with LMCAS sub or complete occlusion with well-developed collateral circulation. A 2019 single-centre retrospective analysis identified patients presenting with STE-aVR with multilead ST depression. Coronary occlusion was found only in 10% of patients, and none of these lesions were involving the LAD or LMCA STE-aVR with multilead ST depression was associated with acutely thrombotic coronary occlusion in only 10% of patients. Routine STEMI activation in STE-aVR for emergent revascularization is not warranted, although urgent, rather than emergent, catheterization appears to be important.(**Ahmed A Harhash 1, Jennifer J Huang 1, Sridhar Reddy 1, Balaji Natarajan 1, Mahesh Balakrishnan 1, Ranjith Shetty 1, Mathew D Hutchinson 1, Karl B Kern 2aVR ST Segment Elevation: Acute STEMI or Not? Incidence of an Acute Coronary Occlusion. Am J Med. 2019 May;132(5):622-630. doi: 10.1016/j.amjmed.2018.12.021**.)

STE in the precordial leads predicted the absence of collateral circulation while STE in aVR and STE in both aVR and aVL predicted different collateral filling territories in ULM occlusion. STE in I, non-STE in aVR, and STEMI predicted in-hospital mortality in these patients.(**Chunwei Liu # 1 2, Fan Yang # 3, Jingxia Zhang 2, Yuecheng Hu 2, Jianyong Xiao 2, Mingdong Gao 2, Le Wang 2, Ximing Li 2, Zhigang Guo 4, Hongliang Cong 5, Yin Liu 6 Electrocardiographic patterns predict the presence of collateral circulation and in-hospital mortality in acute total left main occlusion BMC Cardiovasc Disord 2022 Apr 2;22(1):144. doi: 10.1186/s12872-022-02585-x**.)

In patients with anterior STEMI, admission aVR STE ≥1 mm was found to be a strong and independent predictor of Maior Adverse Cardiac Events (MACE) within 30 days of discharging. On the other hand, in patients with inferior STEMI, aVR ST depression ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging.(**Sogol Sedighi 1, Mustafa Fattahi 1, Pooyan Dehghani 1, Amir Aslani 1, Zahra Mehdipour Namdar 1, Mani Hassanzadeh 1) aVR ST-segment changes and prognosis of ST-segment elevation myocardial infarction. Health Sci Rep. 2021 Oct 1;4(4):e387. doi: 10.1002/hsr2.387**.).

References

1. Mahajan N, Mehta Y, Rose M, Shani J, Lichstein E. Elevated troponin level is not synonymous with myocardial infarction. *Int J Cardiol.* 2006; 111:442–449. doi: 10.1016/j.ijcard.2005.08.029.
2. M J Zellweger 1, B A Schaer, T A Cron, M E Pfisterer, S Osswald. Elevated troponin levels in absence of coronary artery disease after supraventricular tachycardia. Swiss Med Wkly. 2003 Aug 9;133(31-32):439-41.
3. David J Carlberg 1, Sarah Tsuchitani, Kevin S Barlotta, William J Brady. Serum troponin testing in patients with paroxysmal supraventricular tachycardia: Outcome after ED care. *Am J Emerg Med*, vol. 29, no. 5, pp. 545–548, 2011. 2011 Jun;29(5): 545-8.doi: 10.1016/j.ajem.2010.01.041.
4. Radhika Nandur Bukkapatnam 1, Melissa Robinson, Samuel Turnipseed, Daniel Tancredi, Ezra Amsterdam, Uma Narasimhan Srivatsa. Relationship of myocardial ischemia and injury to coronary artery disease in patients with supraventricular tachycardia. 2010 Aug 1;106(3):374-7. doi: 10.1016/j.amjcard.2010.03.035.

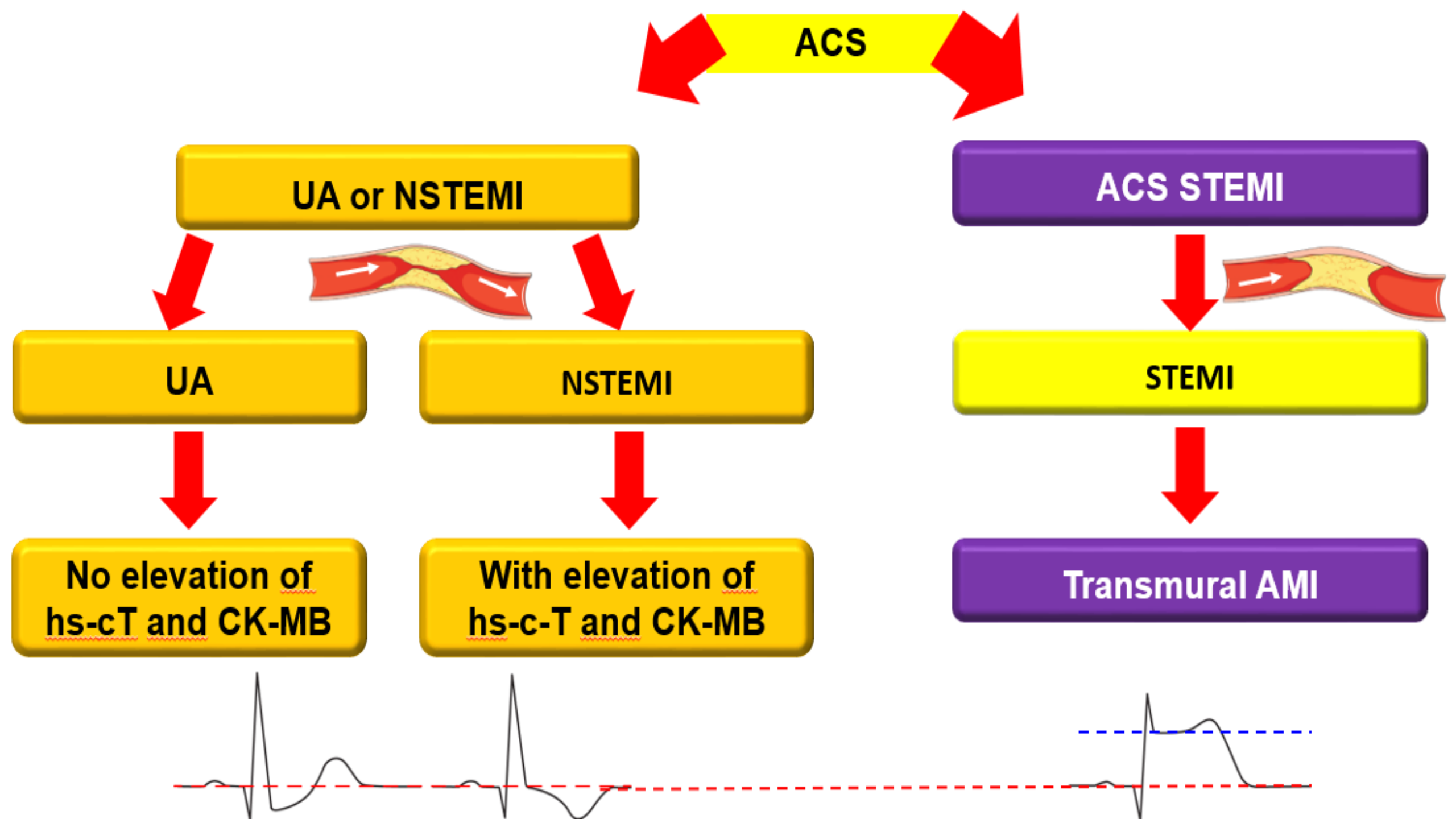
Theoretical concept for understanding this topic

Acute Coronary Syndrome (ACS)

is a term that describes an acute ischemic insult to the myocardium resulting from sudden reduction in coronary blood flow. ACSs include three clinical pictures: **STEMI(**ST-Segment Elevation Myocardial Infarction**),** **NSTEMI(**non-STEMI**),** and **UA**(Unstable Angina) The findings on the ECG will help to categorize patients into two major subdivisions of major diagnostic and therapeutic consequences (**Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes. J Am Coll Cardiol. 2014 Dec 23;64(24): e139-e228. doi: 10.1016/j.jacc.2014.09.017.**). In other words, ACSs are (set of signs and symptoms or conditions) due to decreased blood flow in the coronary arteries such that part of the heart muscle is unable to function properly or dies. (**Petrovic L, Chhabra Selecting A Treatment Modality In Acute Coronary Syndrome. SourceStatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2019**).[2]

According to the 4th universal definition of MI, ECG criteria for **STEMI(**: *Fourth* Universal Definition of Myocardial Infarction**)** diagnosis are STE ≥1 mm in two contiguous leads, except leads V_2-V_3 where the following cut-points apply: ≥2 mm in men ≥40 years; ≥2.5 mm in men <40 years or ≥1.5 mm in women regardless of age (**Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018) *J Am Coll Cardiol.* 2018;72(18):2231–64.).**

These current formal criteria are based on the modification of the 2000 ACC/ESC criteria by Macfarlane et al. (**Macfarlane PW, Browne D, Devine B, Clark E, Miller E, Seyal J, et al. Modification of ACC/ESC criteria for acute myocardial infarction. *J Electrocardiol.* 2004; 37:98–103**.) However, some patients present with ECG patterns that are associated with ATO (Acute Total Occlusion.) but do not fulfill the above-mentioned STEMI criteria.


ACS
UA or NSTEMI
ACS STEMI
UA
NSTEMI
STEMI
No elevation of hs-cT and CK-MB
With elevation of hs-c-T and CK-MB
Transmural AMI

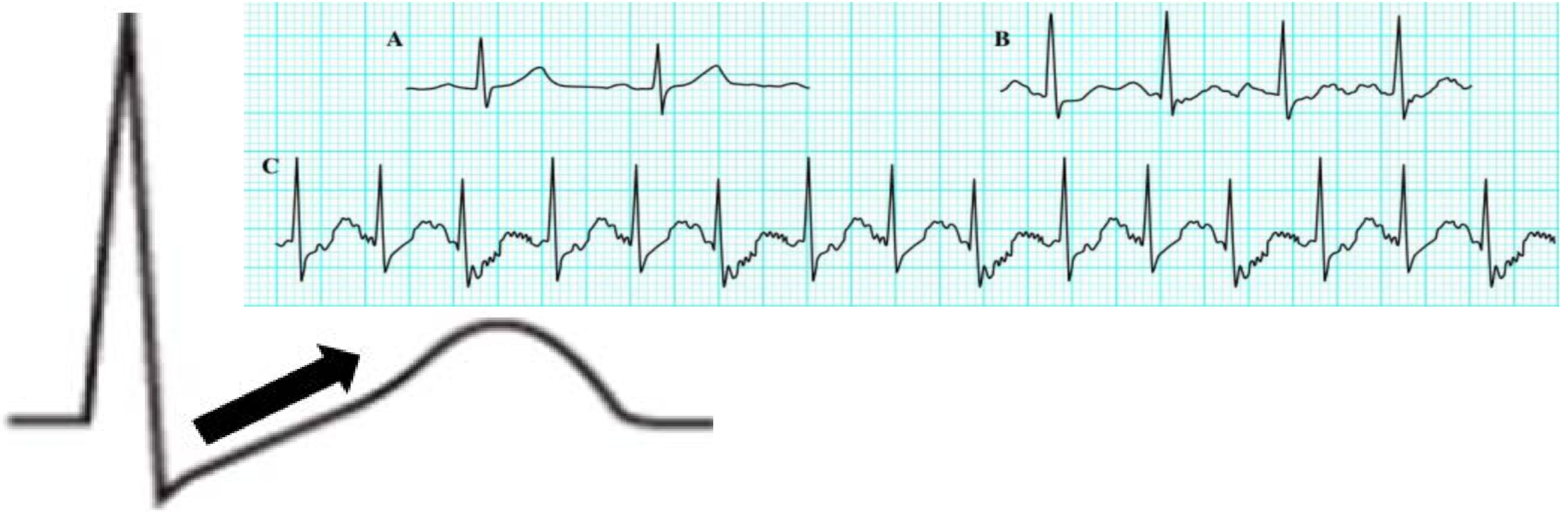
Main characteristic of Incomplete partial or intermittent coronary artery occlusion versus Complete or near complete coronary artery occlusion

(Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction: executive summary and recommendations A report of the American College of Cardiology/American Heart Association task force on practice guidelines (committee on the management of patients with unstable angina) *Circulation . 2000 Sep 5;102(10):1193-209. doi: 10.1161/01.cir.102.10.1193.*) (Brian A Bergmark 1, Njambi Mathenge 2, Piera A Merlini 3, Marilyn B Lawrence-Wright 4, Robert P Giugliano 5. Acute coronary syndromes Lancet. 2022 Apr 2;399(10332):1347-1358. doi: 10.1016/S0140-6736(21)02391-6.)

NSTEMI	STEMI
Incomplete partial or intermittent coronary artery occlusion	Acute Total coronary Occlusion: (ATO)
Account for ≈70% of all ACSs	Account for ≈30% of all ACSs
ST depressions: present in ≈ 30%	
T-Wave Inversion (TWI): present in ≈ 12%	Complete or near complete coronary artery occlusion:
ST segment depressed* + TWI ≈ 16%	Account for ≈30% of all ACSs
Absence of ST depression and TWI ≈ 41%	
Subendocardial myocardial infarction without ATO, However, coronary angiograms reveal that a significant proportion of patients with NSTEMI have ATO.	Transmural myocardial necrosis

Causes of ST Depression

- **Normal variant during exercise or hyperventilation**



The normal ST segment depressed is unsloping followed by a positive T-wave. The J point (the point of inflection at the junction of the S wave and ST segment) becomes depressed during exercise, with maximum depression at peak exercise. The normal ST segment during exercise therefore slopes sharply upwards. Normal changes from rest (A), after three minutes' exercise (B), and after six minutes' exercise (C). Note the upsloping ST segments followed by positive T-wave

Normal electrocardiographic changes during exercise

P wave increases in height, R wave decreases in height, J point becomes depressed, ST segment becomes sharply upsloping, QT/QTc interval shortens and T wave decreases in height

Abnormal changes during exercise

The standard criterion for an abnormal ST segment response is horizontal (planar) or downsloping depression of >1 mm. If 0.5 mm of depression is taken as the standard, the sensitivity of the test increases and the specificity decreases (vice versa if 2 mm of depression is selected as the standard).

Reasons for stopping a test

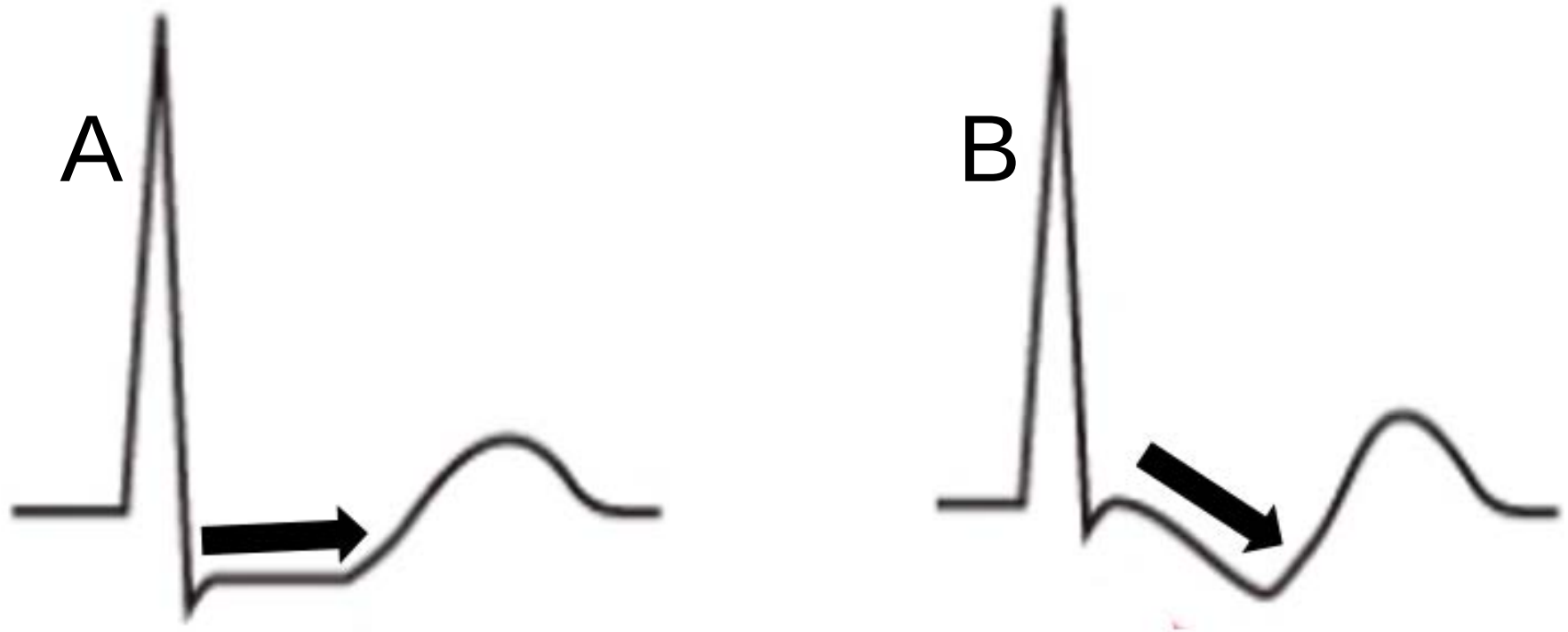
Electrocardiographic criteria

Severe ST segment depression (>3 mm), ST segment elevation >1 mm in non-Q wave lead, frequent PVCs(unless the test is to assessment ventricular arrhythmia), onset of ventricular tachycardia, new atrial fibrillation or supraventricular tachycardia, development of new bundle branch block (if the test is primarily to detect underlying CAD), new second or third degree heart block and cardiac arrest

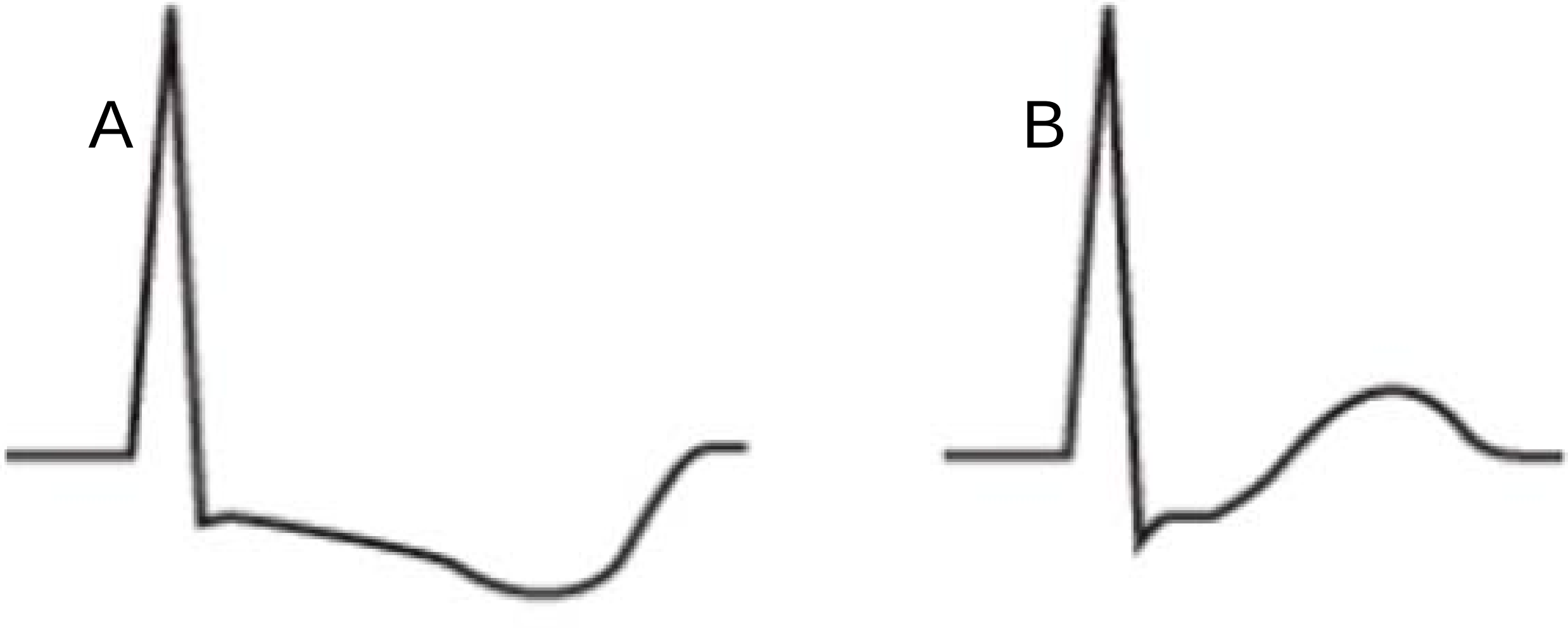
Symptoms and signs

Patient requests stopping because of severe fatigue, severe chest pain, dyspnea, or dizziness, fall in systolic blood pressure (>20 mm Hg), rise in blood pressure (systolic >300 mm Hg, diastolic >130 mm Hg) and ataxia

- **Myocardial ischemia / NSTEMI. Myocardial ischemia** is the term for an imbalance of O_2 supply to the heart. **NSTEMI:** is an acute ischemic event causing myocyte necrosis. ST segment depression horizontal is very typical of ischemia Figure



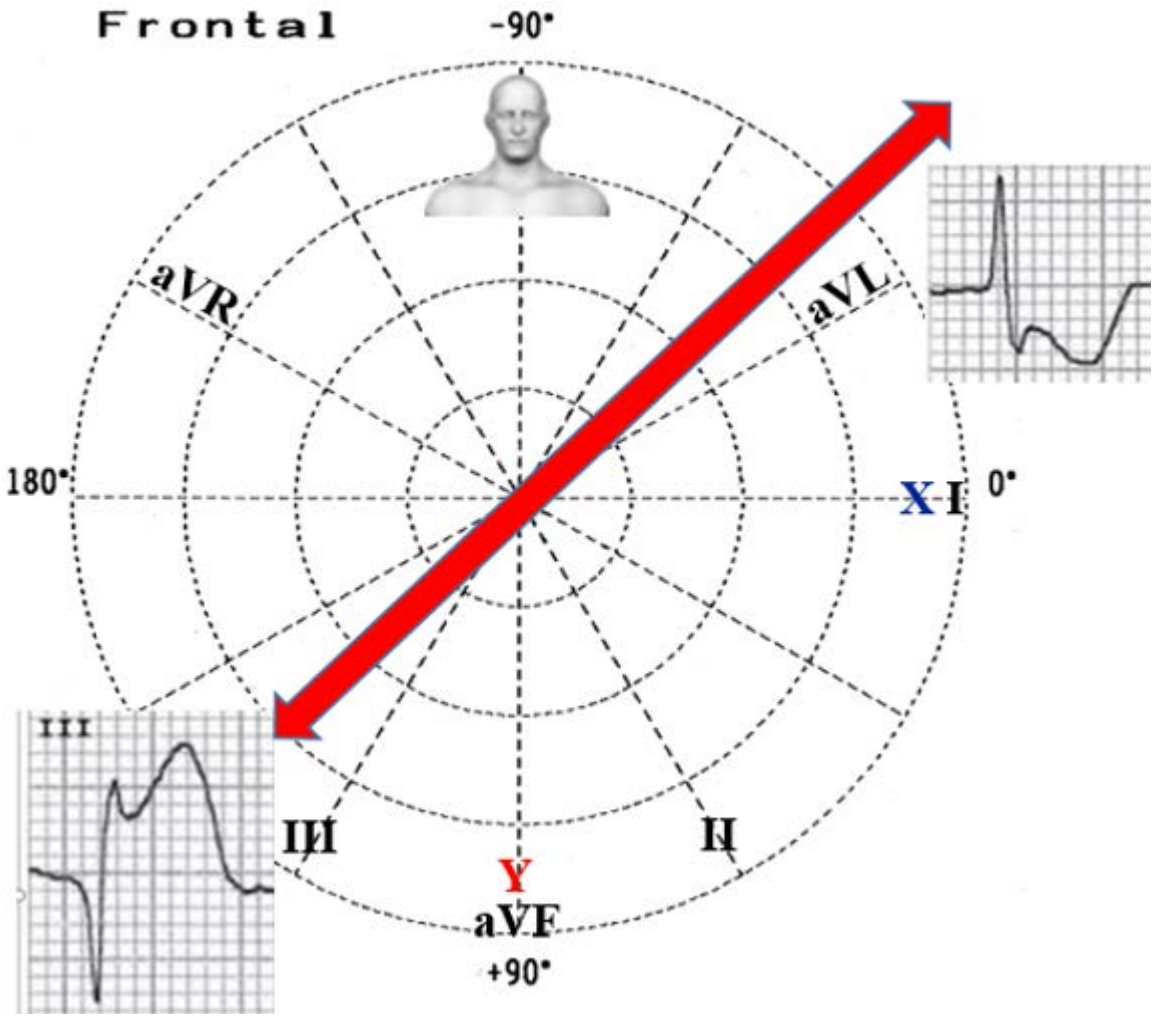
A：Horizontal ST depression followed followed by positive T-wave
B：Downsloping ST depression followed followed by positive T-wave



A: Downsloping ST depressionfollowed by negative T-wave

B: Downsloping ST depression followed by positive T-wave

Reciprocal change or "mirror image" in STEMI: it is defined as ST-segment depression occurring on an ECG which also has ST-segment elevation in at least 2 leads in a single anatomic segment. (**W J Brady, et al. Cause of ST segment abnormality in ED chest pain patients. Am J Emerg Med. 2001 Jan;19(1):25-8. doi: 10.1053/ajem.2001.18029**.). Reciprocal change has a pattern that resembles "upside down" ST elevation and is seen in leads electrically opposite to the site of acute myocardial infarction.

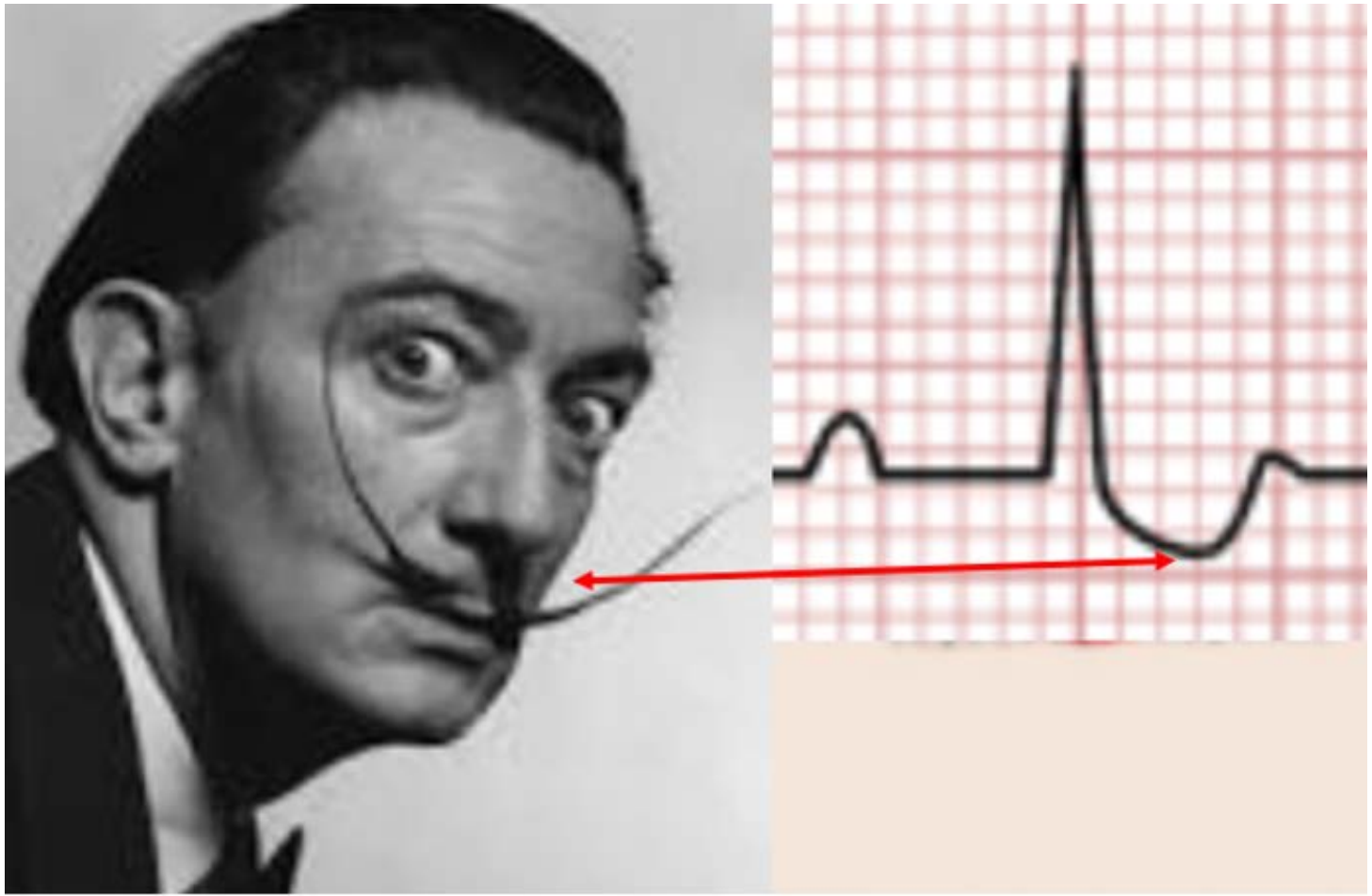


ST Segment elevation during acute inferior MI and reciprocal change in lateral lead aVL

Lateral AMI manifests as horizontal ST depression in V1-3 and is associated with upright T waves and tall R waves in the right precordial leads.

Lateral AMI, erroneously called posterior or dorsal myocardial infarction. The posterior wall of the heart does not exist! **Bayés de Luna A et al. The end of an electrocardiographic dogma: a prominent R wave in V1 is caused by a lateral not posterior myocardial infarction-new evidence based on contrast-enhanced cardiac magnetic resonance-electrocardiogram correlations. Eur Heart J, 2015 Apr 21;36(16):959-64. doi: 10.1093/eurheartj/ehv035.**) (**Bayés de Luna A, Wagner G, Birnbaum Y, Nikus K, Fiol M, Gorgels A, Cinca J, Clemmensen PM, Pahlm O, Sclarovsky S, Stern S, Wellens H, Zareba W; International Society for Holter and Noninvasive Electrocardiography. Circulation. 2006 Oct 17;114(16):1755-60. doi: 10.1161/CIRCULATIONAHA.106.624924**) Figure

Digoxin effect: It is characterized by downsloping ST depression with a characteristic “reverse tick” or “Salvador Dali sagging” appearance, J point depression (usually in leads with tall R waves) flattened, inverted, or biphasic T waves with peaking of the terminal portion, shortened QT interval and mild PR interval prolongation due to increased vagal tone, up to 240 ms. This drug is used in some cases of heart failure with atrial fibrillation



Salvador Dalí, in full Salvador Felipe Jacinto Dalí y Domenech, (**born** May 11, 1904, Figueras, Spain—died January 23, 1989, Figueras), Spanish Surrealist ...
Born: May 11, 1904 Spain
Died: January 23, 1989 (aged 84)

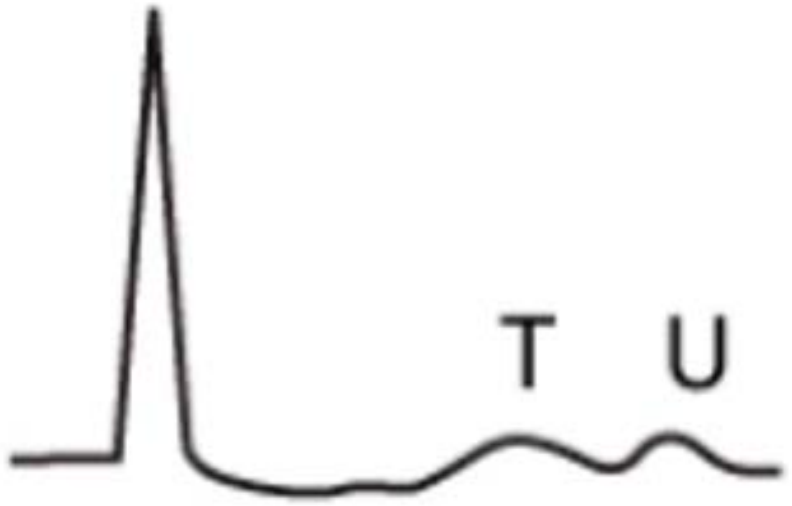
Hypokalemia:

Hypokalemia is defined as a serum potassium level of K^+ ≤4.0 mmol/l,(**João Pedro Ferreira 1, Javed Butler 2, Patrick Rossignol 3 et al. Abnormalities of Potassium in Heart Failure: JACC State-of-the-Art Review,J Am Coll Cardiol. 2020 Jun 9;75(22):2836-2850. doi: 10.1016/j.jacc.2020.04.021**.).

Hypokalemia is classified by its severity in

- Mild: ≤4.0 mmol/l and >3.0 mmol/L
- Moderate: potassium level of <3.0 mmol/L and ≥2.5 mmol/L
- Severe potassium level <2.5 mmol/L.

ECG changes generally do not manifest until there is a moderate degree of hypokalemia (2.5-2.9 mmol/L). The earliest ECG manifestation of hypokalemia is a decrease in T wave amplitude. **ECG features of hypokalemia (K < 2.7 mmol/L),** Increased P wave amplitude: the "P pulmonale" pattern is occasionally observed in patients with hypokalemia, and it is transient and concomitant, (**Weaver WF, Burchell HB. Serum potassium and the electrocardiogram in hypokalemia. *Circulation* 1960; 21: 505–521. 1960/04/01. doi: 10.1161/01.cir.21.4.505.**) (**Chou TC, Helm RA. The pseudo P pulmonale. *Circulation* 1965; 32: 96–105. 1965/07/01. doi: 10.1161/01.cir.32.1.96.**) prolonged PR interval, widespread ST depression followed by T wave flattening/inversion Prominent U waves (best seen in the precordial leads V2-V3) Apparent long QT interval due to fusion of T and U waves (= long QU interval). Hypokalemia and high sympathetic tone causes ST-segment depression followed by flat T-waves and prominent m marked U waves. High sympathetic tone also causes tachycardia Figure

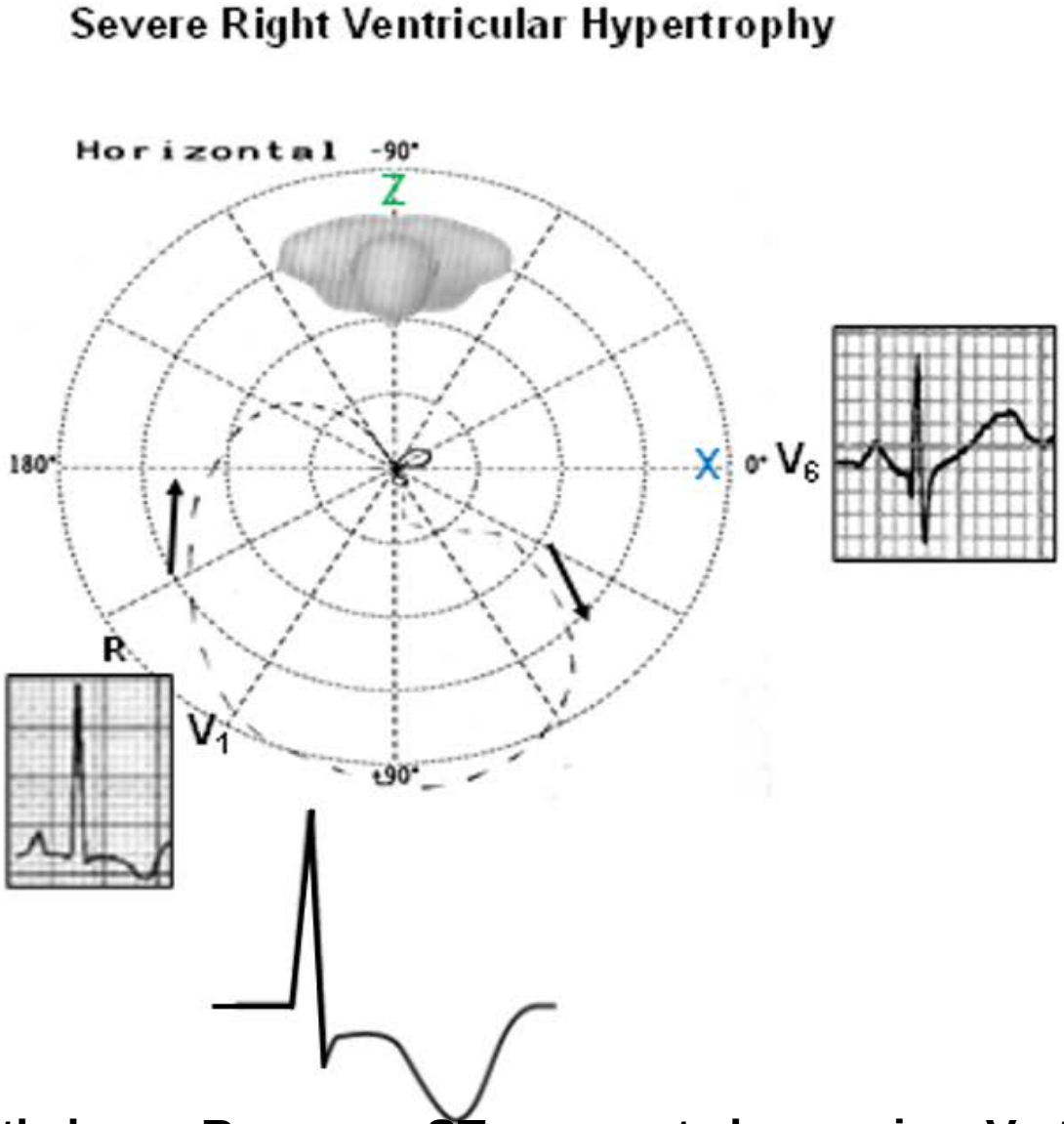


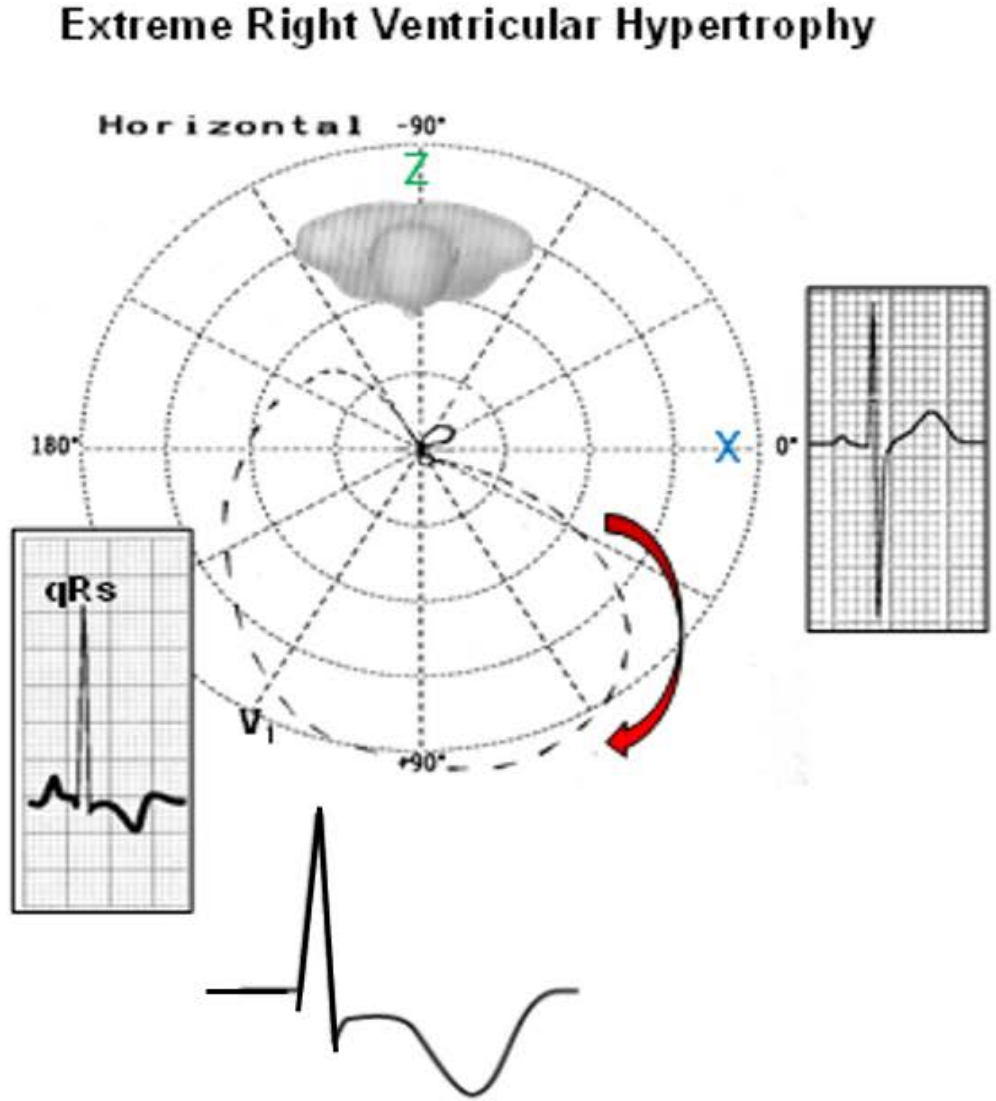
ECG Score for hypokalemia

Electrocardiographic sign	Value
T/U ≤ 1 in lead II	1
T/U ≤ 1 in lead V2	1
U-wave voltage ≥1,5 mm	2
U-wave voltage 1.1 -1.9 mm	1
U-wave voltage ≥ 2.0	2
ST-depression ≥ 0.5mm in II or V1 to V3	1

Electrocardiographic score 0-1, no diagnostic, 2: suggestive. 3-7 characteristic

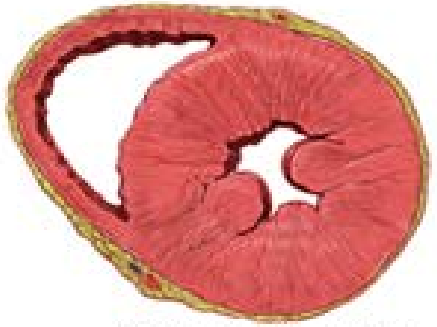
Right ventricular hypertrophy (RVH) on right precordial leads



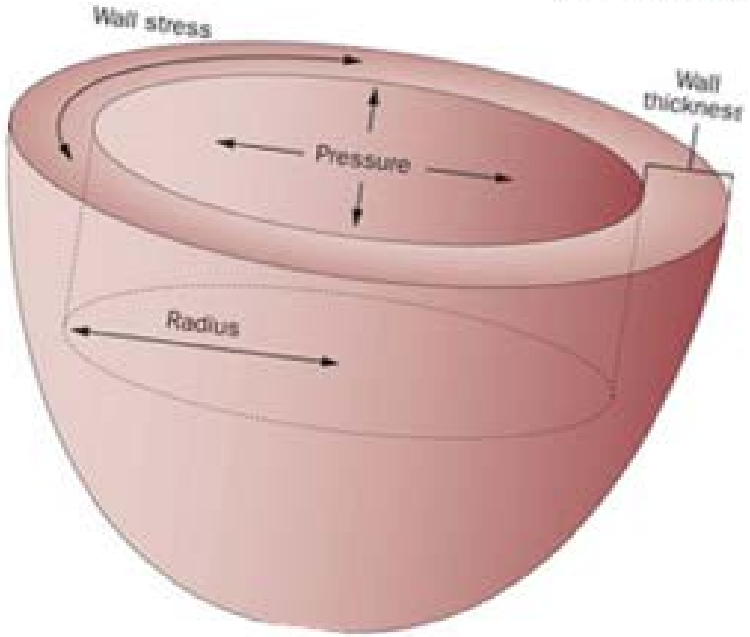


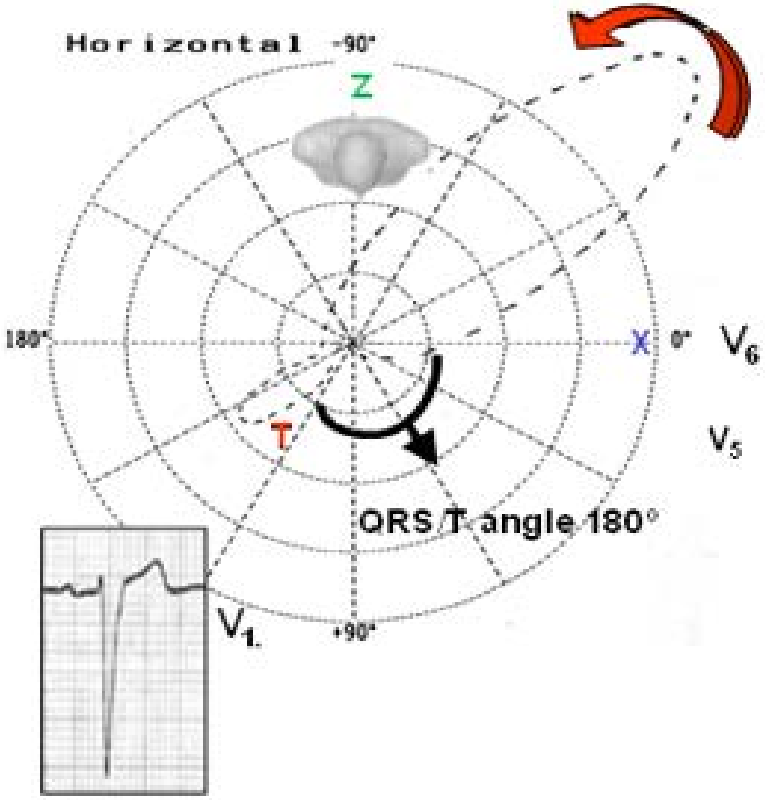
RVH with large R-waves, ST-segment depression V_1 to V_3 followed by negative T-waves. In case of chest discomfort, one must consider possibility of reciprocal image of lateral and high lateral transmural ischemia/infarction as a differential diagnosis.

Left Ventricular hypertrophy



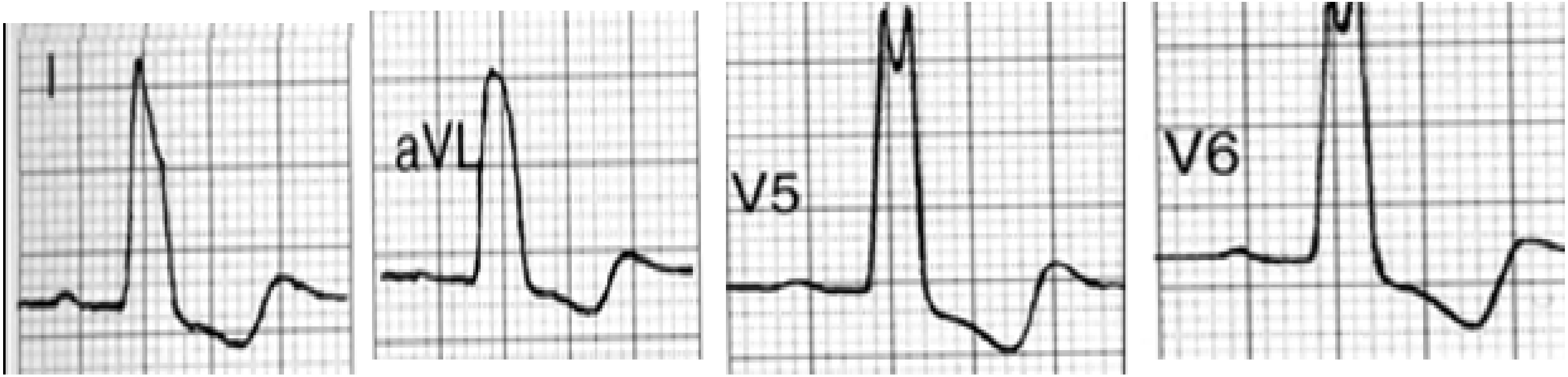
Concentric LVH





Repolarization abnormalities: Deviation of the ST segment and the T wave in the opposite direction to the main QRS vector causes widening QRS amplitude and wide QRS/T angle.

Left Bundle Branch Block(LBBB)/ CLBBB



LBBB is characterized by ST-segment depression followed by a negative asymmetric T wave in leads I, aVL, V5 and V6. In discordant LBBB there is ST-segment depression followed by negative asymmetrical T waves in at least two of the lateral leads I and V5 or V6 and concomitant positive T-waves in the right precordial leads.

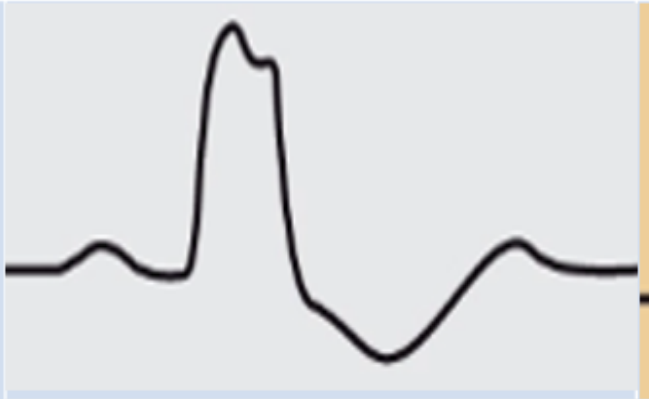
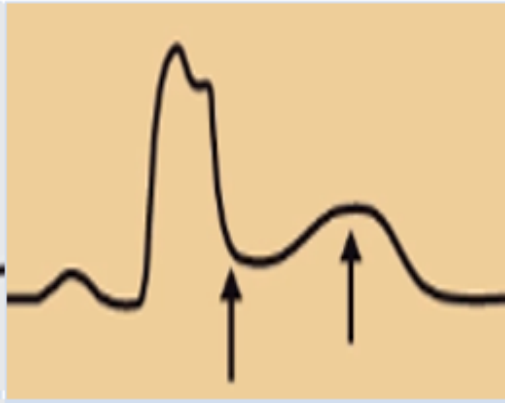
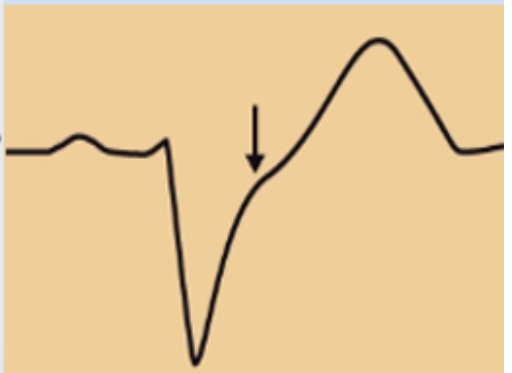
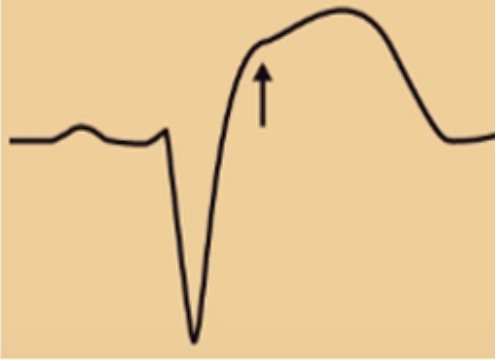
For patients presenting with possible ACS, ECG should be performed within 10 minutes of ED arrival and can distinguish between STEMI and NSTE-ACS. Complains suggestive of myocardial ischemia (Chest pain/angina) or discomfort, often described as aching, pressure, tightness or burning, pain spreading from the chest to the shoulders, arms, upper abdomen, back, neck or jaw, nausea and or vomiting, dyspnea, diaphoresis) and presumed new ischemic ECG changes or new or presumably new CLBBB. **Sgarbossa Criteria.** These criteria are used in the diagnosis of an AMI when a LBBB is present. (it is a complicated CLBBB). Traditionally, it has been taught that MI is not able to be diagnosed via ECG in the presence of a LBBB. However, Sgarbossa et al described **(Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. *N Engl J Med.* 1996 Feb 22;334(8):481–7. doi: 10.1056/NEJM199602223340801.)** some ECG changes seen in patients with LBBB and concomitant AMI and devised a point scoring system. This is called the Sgarbossa criteria, and they are:

ST segment elevation > 1 mm and in the same direction (concordant) with the QRS complex = 5 points. In other words, STE ≥1 mm for leads with a predominantly positive QRS complex (sensitivity 18%, specificity 94%);

STD ≥ 1 mm in leads V_1, V_2, or V_3 (sensitivity 29%, specificity 82%) = 3 points.;

STE ≥5 mm in any lead with negative (discordant) QRS (V1, V2, V3) complexes (sensitivity 55%, specificity 88%).

Table Genuine Sgarbossa criteria for STEMI diagnosis in the setting of CLBBB

Criteria	Points	Leads	Uncomplicate CLBBB	CLBBB+ AMI
ST segment elevation > 1 mm and in the same direction (concordant) with the QRS complex (sensitivity 18%, specificity 94%)	5	V4-V6, aVL, I		
ST segment depression > 1 mm in leads V1, V2 and/or V3 (sensitivity 29%, specificity 82%)	3	V1-V3		
STE ≥5 mm in any lead with negative (discordant) QRS (V1, V2, V3) complexes (sensitivity 55%, specificity 88%).	2	V1-V3		

A score of 3 points is required to diagnose an acute MI. Criteria #3 is under debate as to its usefulness; therefore, either criteria 1 or criteria 2 are essentially required. When ECG suggests STEMI, fast reperfusion with PCI within two hours reduces mortality from 9% to 7%. If PCI within two hours is not possible, fibrinolytic therapy with alteplase, reteplase, or tenecteplase at full dose should be administered for patients < 75 years without contraindications and at 50% dose for patients ≥75 years (or streptokinase at full dose if cost is a consideration), followed by transfer to a facility with the goal of PCI within the next 24 hours. hs-cTnI measurements are the endorsed test to assess NSTEMI. In high-risk patients with NSTE-ACS and no contraindications, prompt invasive coronary angiography and PCI or CABG within 24 to 48 hours are associated with a reduction in death from 6.5% to 4.9% and reduction of the incidence of heart failure.

High risk ECG patterns associated with Acute Total coronary Occlusion (ATO) (Grigorios Avdikos,1,* George Michas,2 and Stephen W. Smith3 From Q/Non-Q Myocardial Infarction to STEMI/NSTEMI: Why It's Time to Consider Another Simplified Dichotomy; a Narrative Literature Review. Arch Acad Emerg Med. 2022; 10(1): e78 . doi: 10.22037/aaem.v10i1.1783)

Smith's modified Sgarbossa criteria:

- STD≥1 mm in any of V1-V3 or
- STE ≥1 mm concordant with QRS in any lead or
- STE/S wave ≥ 25% in any lead **(Smith SW, Dodd KW, Henry TD, Dvorak DM, Pearce LA. Diagnosis of ST-elevation myocardial infarction in the presence of left bundle branch block with the ST-elevation to S-wave ratio in a modified Sgarbossa rule. *Ann Emerg Med.* 2012;60(6):766–76.) (Meyers HP, Limkakeng Jr AT, Jaffa EJ, Patel A, Theiling BJ, Rezaie SR, et al. Validation of the modified Sgarbossa criteria for acute coronary occlusion in the setting of left bundle branch block: A retrospective case-control study. *Am Heart J.* 2015;170(6):1255–64.)**

Sensitivity/specificity: 80%/99%

Culprit artery: LAD, LCx or RCA

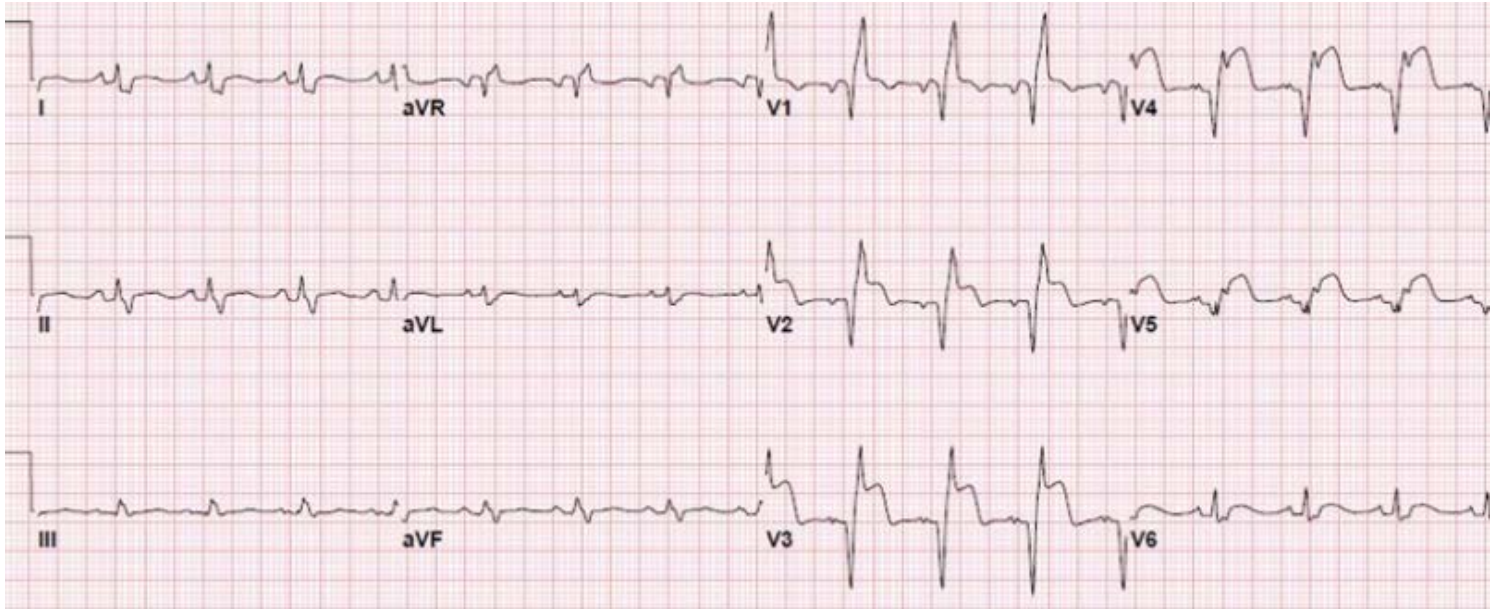
Right Bundle Branch Block (RBBB/CRBBB)

Normally, RBBB has a bit of ST depression in V1-V3 that is discordant (in the opposite direction of) the R'-wave. So that bit of ST Depression in V1 is normal. Right bundle branch block occurs when electrical activity of the heart, specifically the His-Purkinje system, is interrupted or altered, resulting in a widened QRS and ST-segment changes. The ECG findings of RBBB can sometimes lead to an over-interpretation of the ST-segment changes as acute myocardial infarction.

Can you see a STEMI in RBBB?

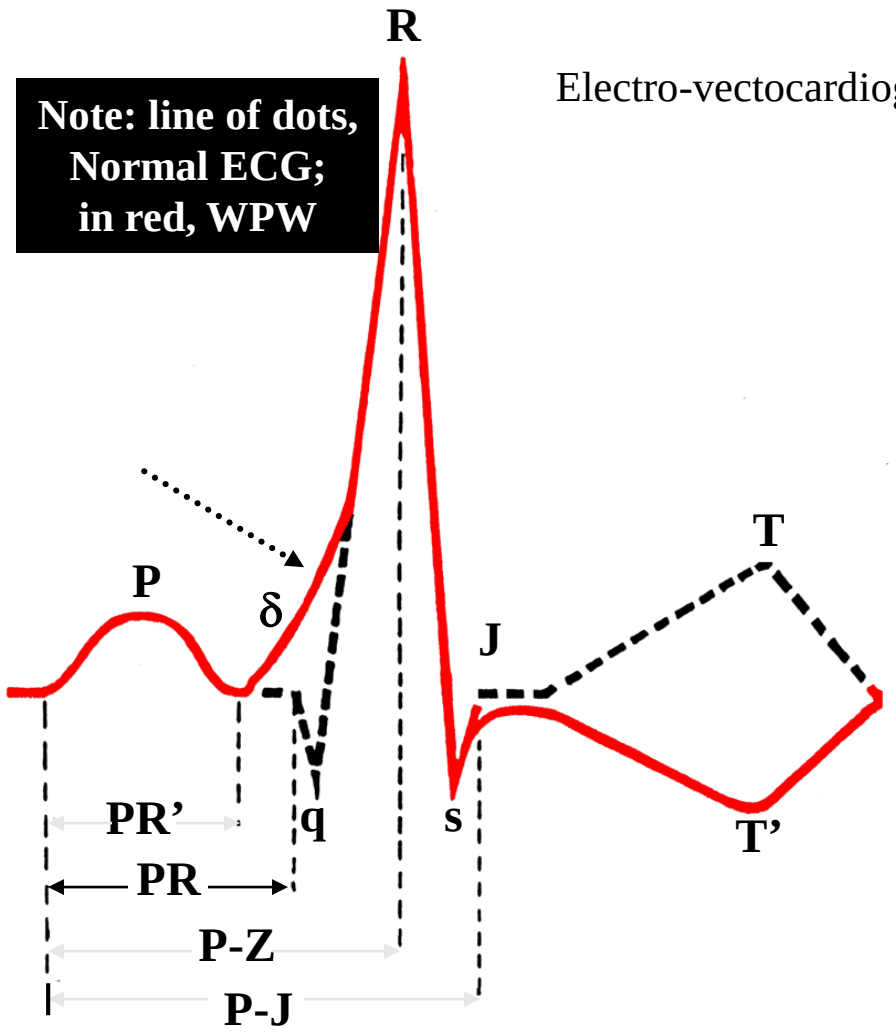
ST elevation — RBBB does not usually interfere with the diagnosis of an acute ST-elevation MI (STEMI). The reason is that MI most often involves the left ventricle and therefore affects the initial phase of ventricular depolarization, sometimes producing abnormal Q waves.

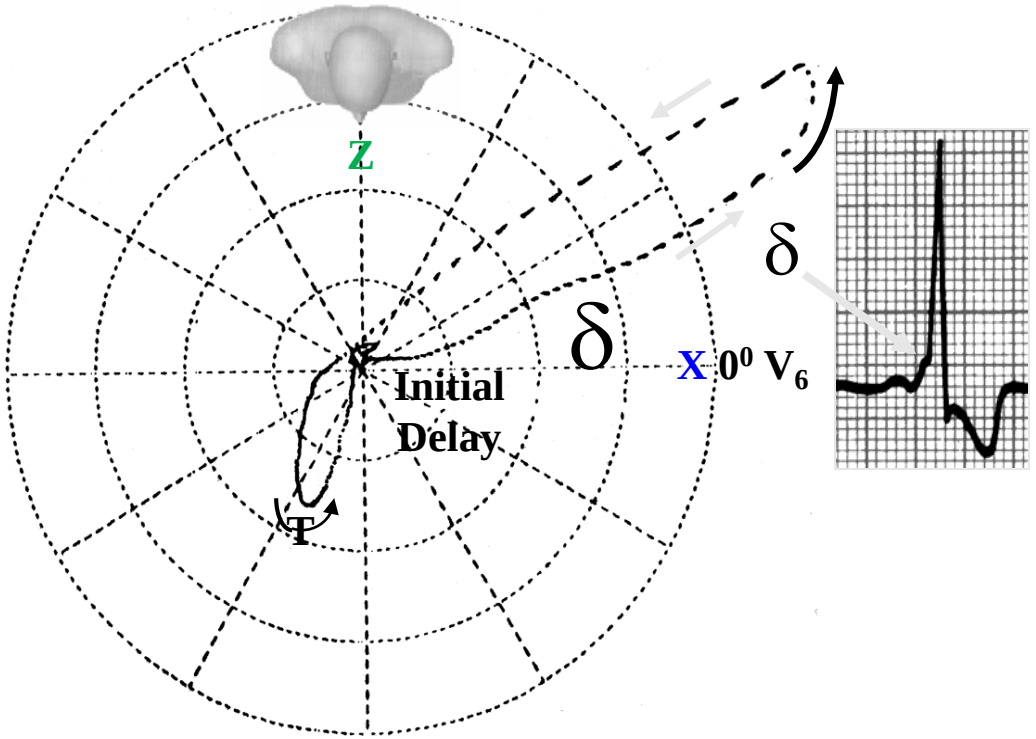
Anterior Wall ST elevation MI with RBBB ECG



WPW ECG/VCG correlation

Electro-vectocardiographic criteria for WPW type preexcitation.

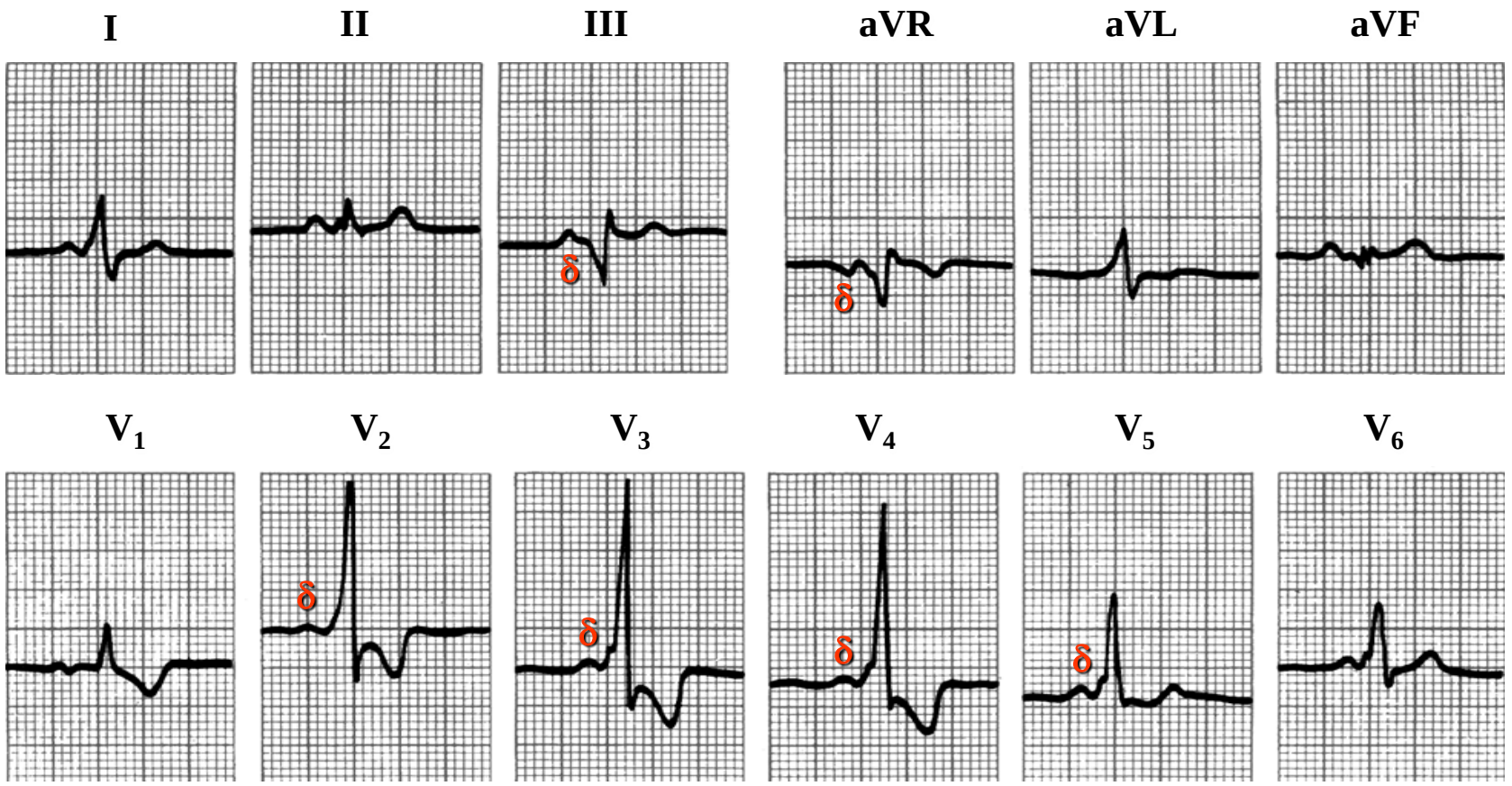




- **PRi or PQ:** since the onset of P up to the onset of QRS. It represents the time the stimulus takes to go from the SA node until reaching the ventricles: 120 ms to 200 ms.
- **PZ:** distance between P wave onset until R apex: 150 to 230 ms.
- **PJ:** distance between P wave onset until j point: 180 to 260 ms.

- Initial delay of QRS loop: delta wave.
- T-loop opposite to QRS loop

Classical ventricular pre-excitation



Female, white, 36-year-old patient. Asymptomatic. She came to the office for a pre-operative evaluation for an otorhinolaryngological surgery.

Sinus rhythm, short PR (100 ms), notch at the base of the ascending ramp of R from V_2 to V_6 (δ wave). Prominent anterior forces (PAF) wall from V_2 to V_4 that indicates the posterior location of the anomalous pathway. The broad Q from III may originate the false suspicion of electrically inactive area in inferior wall.

Typical Wolff-Parkinson-White tracing, with anomalous pathway of right posterior location, pseudo- RVH with strain pattern, prominent anterior forces.