MALE 38

FAMILIAL HCM

ECHO: ASYMETRIC SEPTAL HYPERTROPHY

ECHO: IVS 1.6 LPW 0.6 LA 42mm MVA 3.7cm^2 (slightly reduced)

MVP POSTERIOR LEAFLET – MILD MR – Mildly thickened MV leaflets

FATHER: HYPERTROPHIC CARDIOMYOPATHY

HOMEM 38

PORTADOR DE HCM FAMILIAR

ECO: HIPERTROFIA SEPTAL ASSIMÉTRICA

ECO: IVS 1,6 LPW 0,6; LA 42mm; MVA 3,7cm^2 (ligeiramente reduzido)

FOLHETO POSTERIOR MVP – Regurgitação Mitral LEVE – Folhetos MV levemente espessados

PAI: PORTADOR DE CARDIOMIOPATIA HIPERTRÓFICA

Question
DEAR ANDRÉS:

DOES THE ATTACHED TRACING IS "SUGGESTIVE" For asymmetric septal hypertrophy???

How do you explain the disappearance of the repolarization changes during excercise?

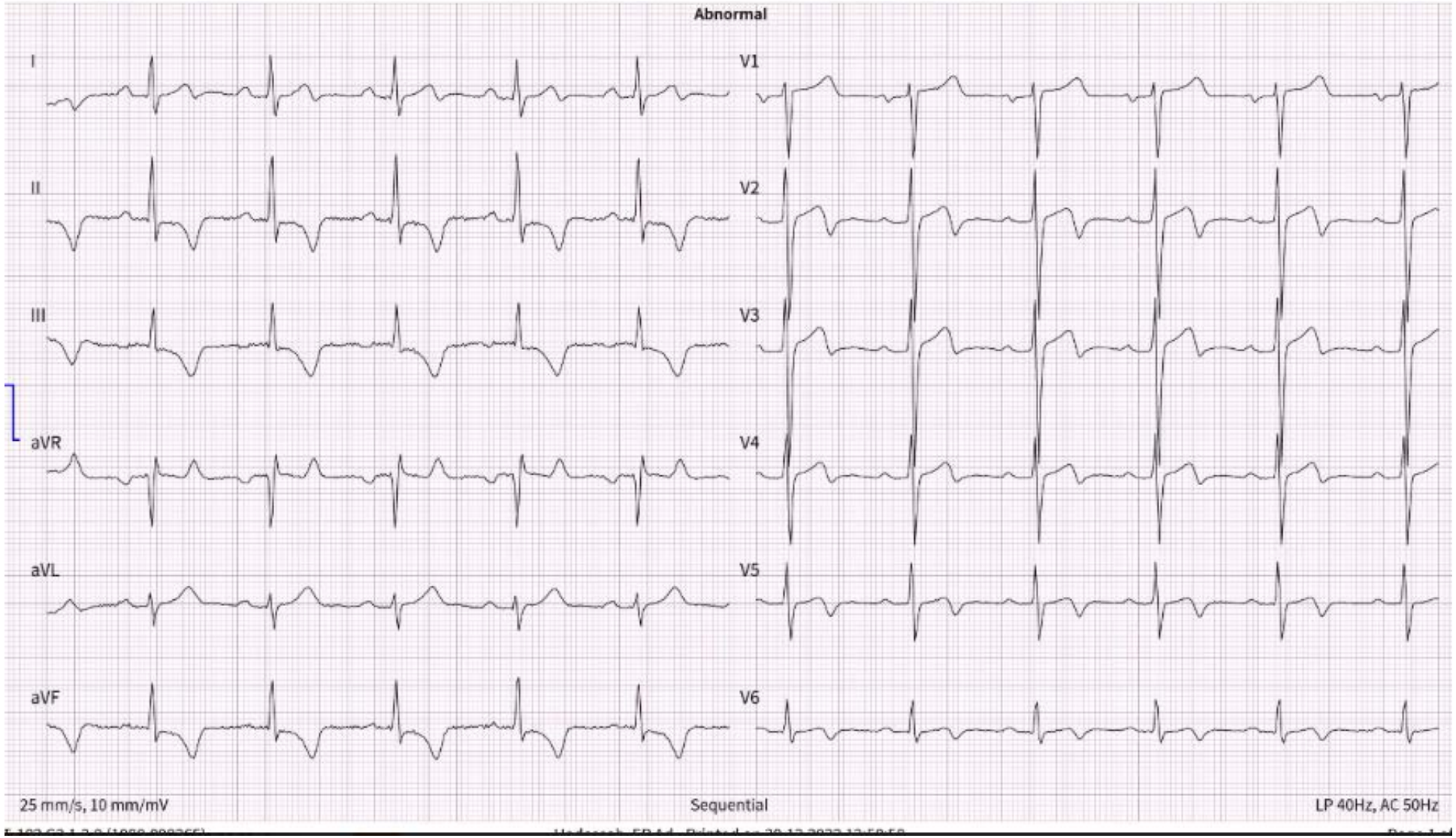
THANKS +++

BB (Bernard Benhansen) Israel

Date of birth	21.09.1984	**Visit ID**		**HR**	**67 bpm**	**RR**	896 ms	Sinus rhythm
Age	38 years	**Room**				**P**	124 ms	Normal electrical axis
Gender	Male	**Order ID**				**PR**	212 ms	S1 S2 S3 pattern
Ethnicity	Undefined	**Ord. prov.**		**P axis**	20 °	**QRS**	101 ms	ST-T abnormality, consider
Height		**Ref. phys.**		**QRS axis**	51 °	**QT**	448 ms	inferior ischemia or left ventricular strain
Weight		**Device ID**	CART4	**T axis**	-75 °	**QTcB**	473 ms	Abnormal ECG

Medication
Remark

Unconfirmed report

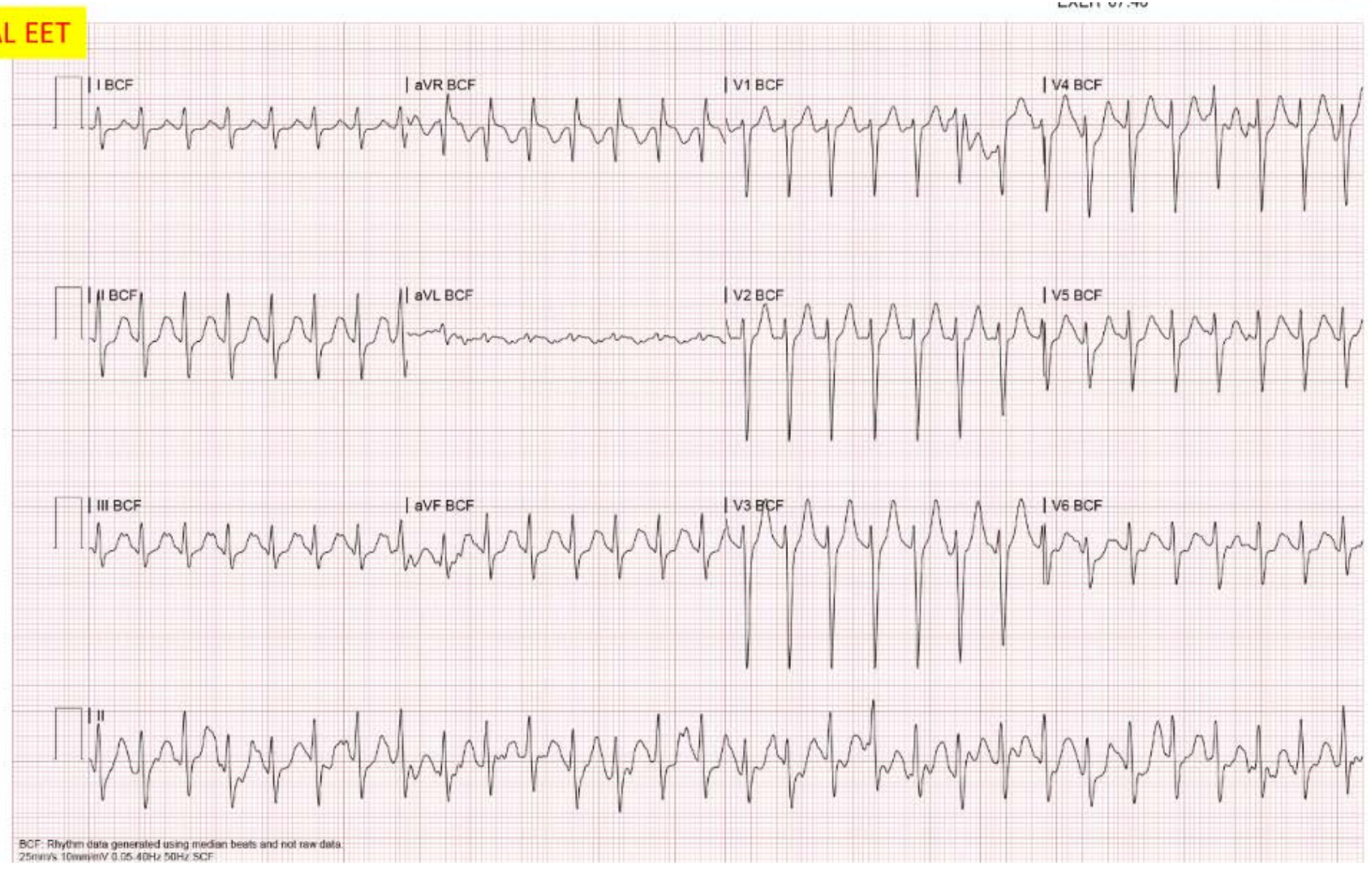


Exam Start: 1/1/2023 11:30:45 AM
Event Time: 01/01/2023 11:40:27
Date of Birth: 9/21/1984
Gender: Male

09:00 EXER 4.2 MPH HR 178
01:02 STAGE 4 16 % BP 116/54

EXER 07:40

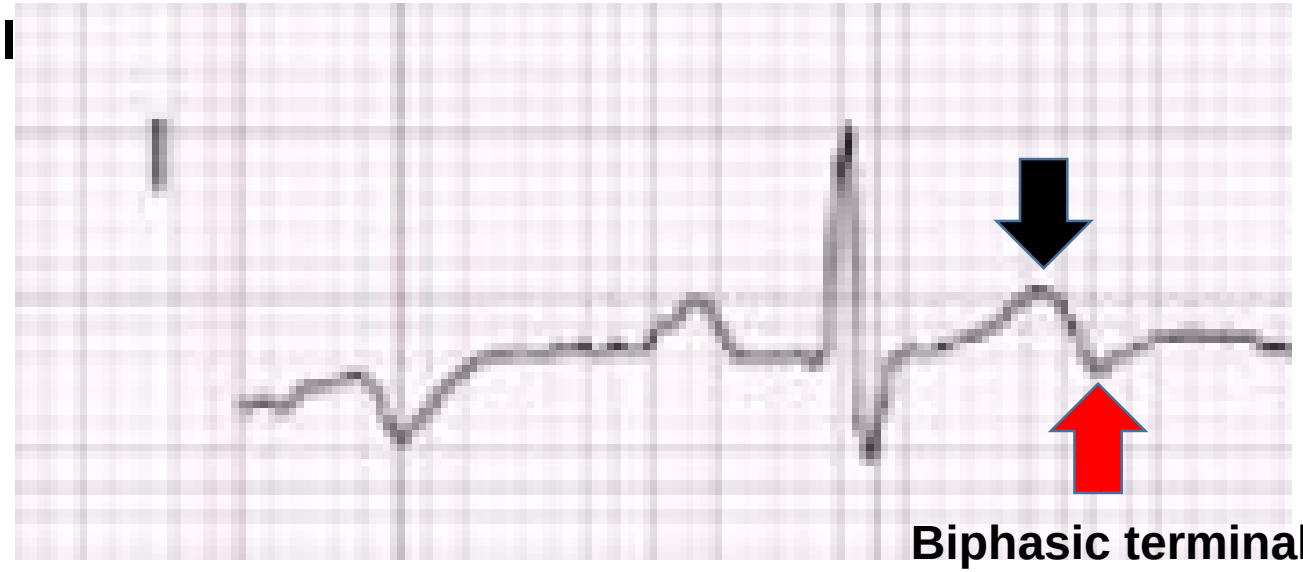
MAXIMAL EET



Reflections on these two ECGs

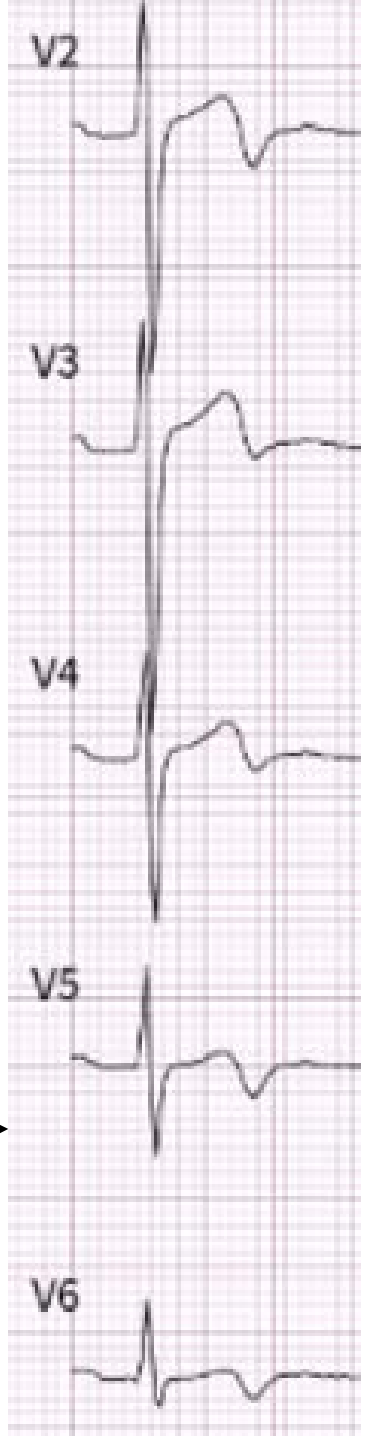
T wave abnormalities

1. Peaked T waves
2. Hyperacute T waves
3. Inverted T waves
4. Camel Hump' T waves
5. Flattened T waves
6. **Biphasic terminal negativity T waves: ECG-1 has Biphasic T waves in anterolateral**



Biphasic terminal negativity T waves

ECG-1



The two main causes of these waves are **myocardial ischemia** and hypokalemia. Example:

Wellens' Syndrome is a pattern of biphasic T waves in V2–3. It is generally present in patients with ischemic chest pain. The biphasic T waves are known for dynamic change in polarity . It may either pull down the or pull up the adjacent ST segment . Prolonged QT interval is a closely related to the biphasic T wave. Some times a U wave can be inscribed in such a way it may mimic a biphasic T wave.

A typical biphasic wave can be two types **Terminal positivity and Terminal negativity**. Terminal negativity (the present case) is more significant than terminal positivity , especially in coronary artery disese CAD. A terminal negativity especially in mid precardial leads would suggest ongoing ischemia in LAD territory .This happens due to dispersion of repolarisation between endocardium and epicardium. The other mechanism could be the altered ventricular gradient between QRS vector and T wave vector. Finally **Myocardial ischemia is present in HCM. Why? Because the presence of Dysfunction of the coronary microvasculature: normal epicardial arteries on coronary angiography, increased wall thickening leading to luminal narrowing, silent myocardial ischemia, myocardial injury and fibrosis.**

(Camici PG, Olivotto I, Rimoldi OE. The coronary circulation and blood flow in left ventricular hypertrophy. J Mol Cell Cardiol. 2012;52:857-864.)

In summary, myocardial ischemia in the HCM could be secondary to:

1) ***Microcirculation disease,***
2) ***Decrease of vasodilator capacity,***
3) ***Systemic compression of septal and subepicardial vessels,***
4) ***Fall of pressure in aorta root,***
5) ***Difficulty in coronary filling by hypertrophy,***
6) ***Coronary atherosclerosis in >50 year-old patients,***
7) ***Offer/demand imbalance by excessive increase of ventricular mass;***
8) ***Striking increase in interstitial connective tissue: replacement fibrosis may be pericellular, patchy, or extensive and often is more prominent in the ventricular septum.*** **(Hughes SE. The pathology of hypertrophic cardiomyopathy. Histopathology. 2004;44:412-427.)(Gutierrez-Barrios A, Camacho-Jurado F, Diaz-Retamino E, *et al.* Invasive assessment of coronary microvascular dysfunction in hypertrophic cardiomyopathy: the index of microvascular resistance. Cardiovasc Revasc Med. 2015;16:426-428.)**

LVH and/or myocardial fibrosis without myocardial disarray are features of uncertain significance.

(Papadakis M, Raju H, Behr ER*, et al.* Sudden cardiac death with autopsy findings of uncertain significance: potential for erroneous interpretation. Circ Arrhythm Electrophysiol. 2013;6:588-596.)

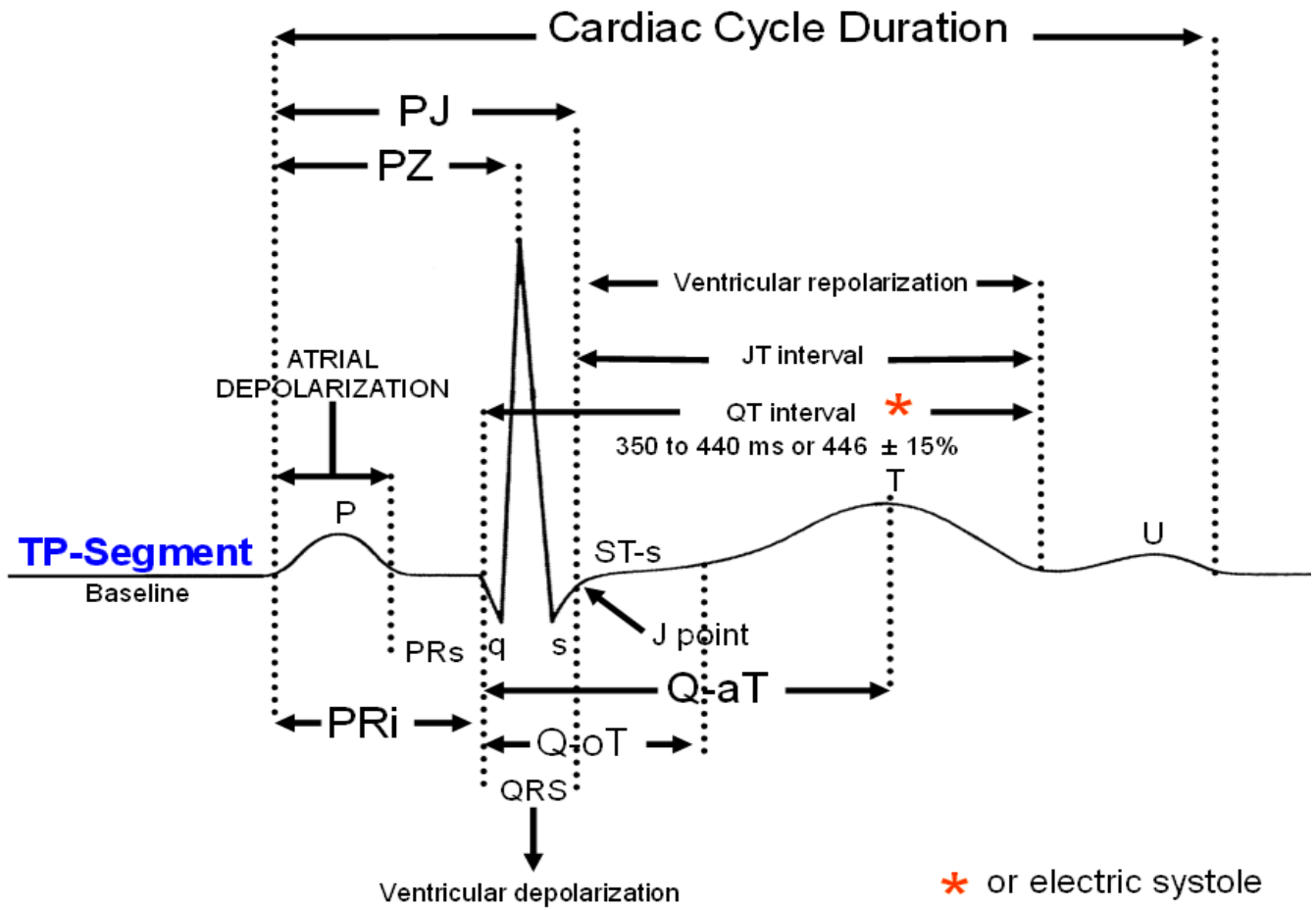
Mitral valve abnormalities, including aberrant papillary muscles, abnormal papillary muscle insertion into the anterior mitral leaflet, and enlargement and elongation of mitral leaflets are common in HCM and responsible for the creation of a pathologic dynamic LVOT gradient.10 In 5 to 10% of patients, in the late phase, they evolve into dilatation and systolic dysfunction resulting from myocardial fibrosis secondary to **microinfarctions** and possible associated to CAD.

The anatomopathological substrate is an important septal cell disorder (95%) and hypertrophy of the middle layer with narrowing of light of the intramural branches responsible of ventricular dysfunction as well as arrhythmias.

Why biphasic T waves are important ?

The biphasic T waves are known for dynamic change in polarity, such as the present case . It may either pull down the or pull up the adjacent ST segment .

Prolonged QT interval is a closely related to the biphasic T wave. In the present case ECG-1 QT= 448ms and QTc 473ms (Normal QT interval is between 350 to 446 ms± n15% figure



Some times a U wave can be inscribed in such a way it may mimic a biphasic T wave. This is especially common in baseline bradycardia.

LVH is one of the common cause of biphasic T wave (Usually terminal positivity)

Biphasic T wave as mode of presentation of NSTEMI

Even though , ST depression is considered the dominant and classical theme of NSTEMI , It is now recognized NSTEMI has another mode of common presentation as biphasic T waves.

Biphasic T waves presenting as acute coronary syndrome(ACS)

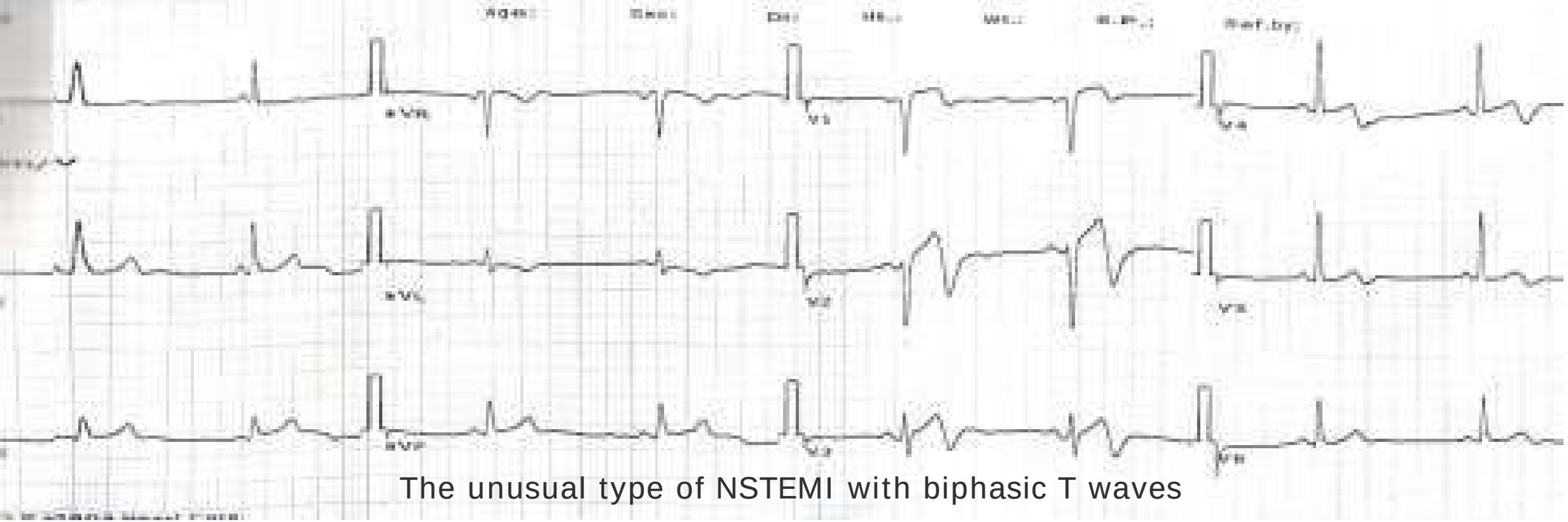
NSTEMI is a common clinical problems in CCU. **When we say NSTEMI it can mean any of the following**

- ❑ NSTEMI with ST depression
- ❑ NSTEMI with T wave Inversion
- ❑ **NSTEMI with Biphasic T wave,** such as the next ECG
- ❑ NSTEMI with normal ECG

The irony called STEMI evolving as NSTEMI**

By default most of us think , if it is NSTEMI . . . there must be ST depression. This thinking is not logical but traditional. Still, ST depression may be the common presentation. NSTEMI with ST depression has much worse outcome than other forms.

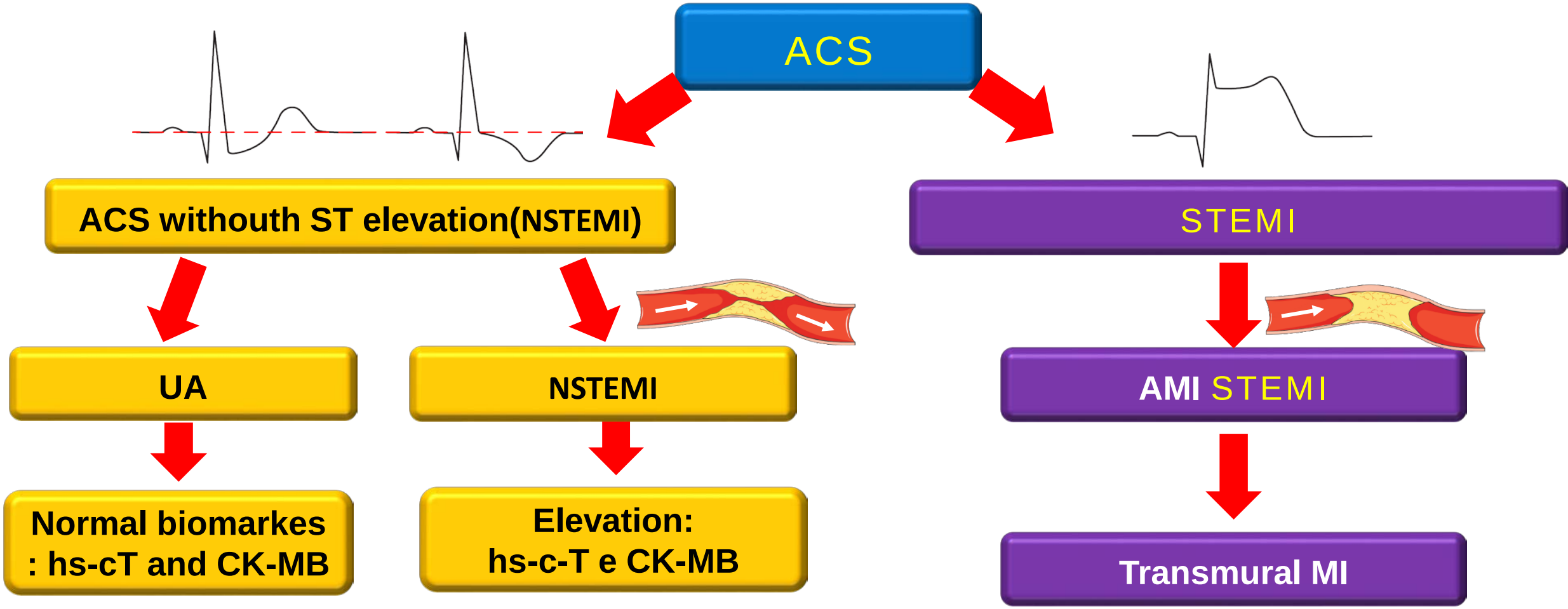
The nex ECG is from a 45 year old man with a vague mid sternal chest pain for 48 hours.



The unusual type of NSTEMI with biphasic T waves

His echo showed wall motion defect in LCX territory .A diagnosis of NSTEMI was made. The predominant finding was biphasic T waves . **One may wonder why can't we call this ECG as a Classical STEMI ? There is a 2mm ST elevation , with a infarct as well ? But , the point here is there is no business for T waves to get biphasic or inverted in the early hours of a classical STEMI . This exactly has happened here. Hence we can not call the above event as STEMI . Instead it is , STEMI evolving into NSTEMI . So a combination of features of STEMI/NSTEMI occur together. The best description for above entity is STEMI in transition to Non Q MI Is the terminology of Non Q MI still relevant or obsolete ? A new paradigm?

Acute coronary syndrome (ACS) is until recently classified as STEMI and NSTEMI.



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, NSTEMI,** and **UA**. 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): Stat Pearls Publishing; 2019**).

Frequency	Bigger	Smaller
In-hospital mortality	Smaller	Bigger
6 month mortality	Similar	Similar

Definitions and Terminology Among Paradigms

STEMI	Refers to AMI with ECG findings meeting the definition of STEMI criteria in the fourth universal definition of MI (. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Circulation 2018;138:e618–51.)
False-positive STEMI	Refers to a patient with ECG features meeting formal STEMI criteria, but the ST elevation is not a result of ischemia, and there is both no culprit lesion and no AMI.

True-positive STEMI =STEMI(+) OMI: Occlusion MI (OMI)	**Refers to a patient with ECG features meeting formal STEMI criteria, who is found to have OMI as the cause of the STE and the AMI. Occlusion MI (OMI) Refers to type 1 acute coronary syndrome involving acute occlusion or near occlusion of a major epicardial coronary vessel with insufficient collateral circulation, resulting in imminent necrosis of downstream myocardium without emergent reperfusion. OMI is the anatomic and pathophysiologic substrate of STEMI, but not all OMI manifests as STEMI.**
Nonocclusion MI (NOMI) = NSTEMI without occlusion	Refers to AMI without angiographic, laboratory, or clinical evidence of OMI.
STEMI(–) OMI = NSTEMI with occlusion	Refers to OMI without the ECG meeting STEMI criteria.

AMI = acute myocardial infarction; ECG = electrocardiogram; STEMI = ST-segment elevation myocardial infarction.

Comparison of STEMI and OMI Paradigms

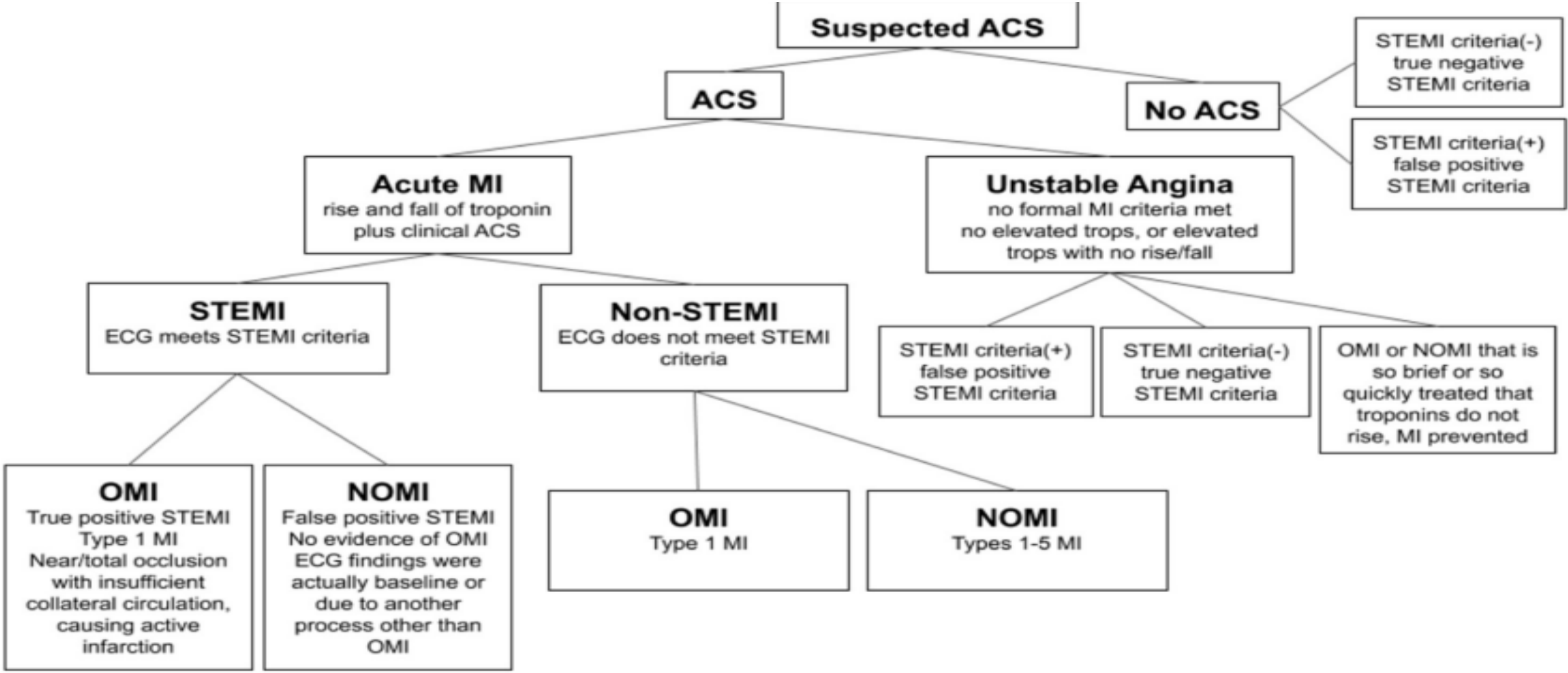
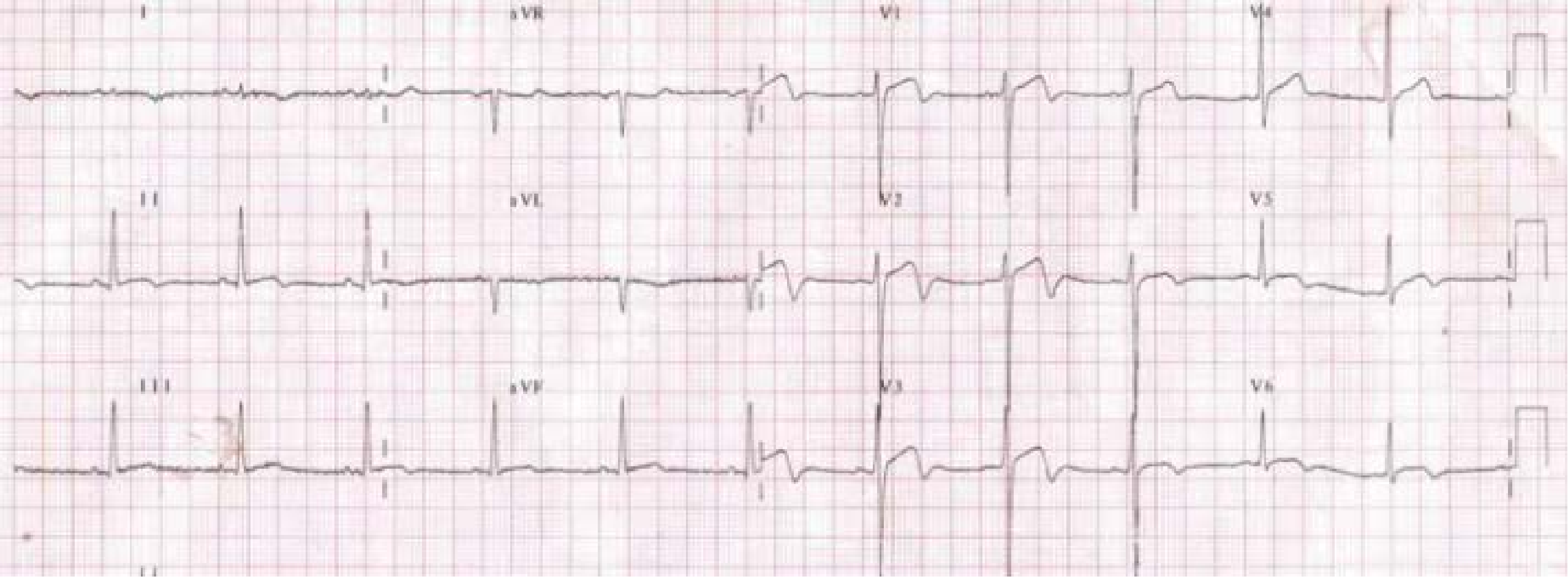


Figure . The acute coronary syndrome (ACS) spectrum using the ST-segment elevation myocardial infarction (STEMI) vs. nonSTEMI paradigm primarily. The current paradigm of MI divides acute MI into STEMI and non-STEMI based on the electrocardiogram (ECG). Occlusion myocardial infarction (OMI) and nonocclusion myocardial infarction (NOMI) are possible in both STEMI and non-STEMI categories

This classification came into vogue primarily to triage patients for thrombolysis eligibility , as ST elevation is the only criteria for thrombolysis. The earlier term ***non q MI*** is largely used to denote the present day NSTEMI. In the past q MI was referring to transmural MI non q MI to non transmural pathologically.(Of course , now we know the relationship between q waves and transmurality is not good)

So when can we still use term non q MI ?

These terminologies of STEMI and NSTEMI are made on admission at the emergency room. ACS being a dynamic entity these patients can have rapidly changing ST shifts , from depression to elevation and vice versa. Fresh T wave changes can also occur .Q waves may or may not develop , depending upon the damage sustained to the myocardium and the efficacy of thrombolysis / PCI. So it should be emphasised here STEMI, NSTEMI, q MI, non q MI are the descriptions of the same group of patients in different time frames. The common mode of evolution of STEMI is to qMI and NSTEMI into non q MI. Cross overs can occur.



A classical “ Non Q MI”, We can´t label this trace as NSTEMI as there is residual ST elevation(In fact it is a STEMI evolving into Non qMI + Plus-minus biphasic T-waves in some anterolateral leads

The problem here is NSTEMI getting converted into STEMI is quiet common and has no nomenclature issues . But when STEMI down grades into NSTEMI there is apparent nomenclature incompatibility .This category of patients have no other labelling option other than “A STEMI evolving into non q MI”. Because one can’t label STEMI evolving into NSTEMI as many of them will have a residual ST elevation as well.

What is the final message ?

The term non q MI is still relevant and is used at discharge , in a patient with STEMI when he or she evolves without a q wave .In the setting of unstable angina , NSTEMI has largely replaced the term non q MI either on admission or at discharge. The important point to remember here is NSTEMI getting converted into STEMI is an adverse outcome and in fact, it is a complication and the patient should get an immediate thrombolysis or PCI , while a STEMI getting converted into non Q MI is generally a major therapeutic success.(Effective salvaging and preventing q waves)