Paciente femenino 40 años, sin antecedentes personales de destaque , internada por hemorragia subaracnoidea con mala evolución. Comatosa, por hemorragia, solo con gatillo respiratorio, resto de los reflejos ausentes

Nos consultan del servicio de terapia intensiva por este ECG que mostramos en la próxima diapositiva

El monitor, desde hace varias horas previas a la interconsulta presentaba el mismo patrón electrocardiográfico.

Hemodinamicamente estable, solo con bajas dosis de noradrenalina que no cambiaron en las ultimas 48hs, perfusión

adecuada, sin trastornos electrolíticos e oximetricos (K, Ca, Mg, pG, HCO3 y lactato normales).

Dr Franco Di Prinzio

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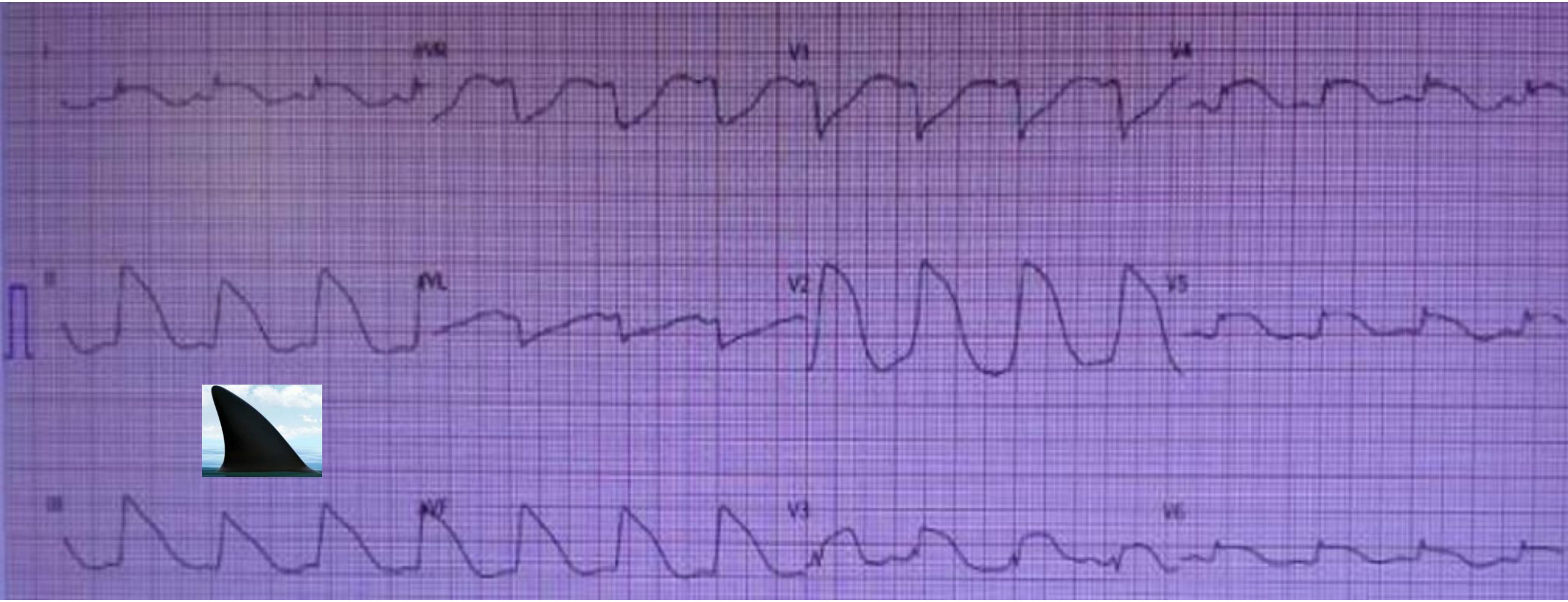
English

40-year-old female patient, with no notable personal history, hospitalized for subarachnoid hemorrhage with poor evolution. Comatose, due to hemorrhage, only with respiratory trigger, other reflexes absent. We are consulted by the intensive care service for this ECG that we show on the next slide. The monitor, for several hours prior to the consultation, had the same electrocardiographic pattern. Hemodynamically stable, only with low doses of norepinephrine that did not change in the last 48 hours. Adequate, perfusion , without electrolyte and oximetric disturbances (normal K^{+}, Ca^{++}, Mg^{++}, pG, HCO^{3-} and lactate).



Shark fin appearance, triangular waveform ECG pattern, or “lambda-like ST, conspicuous pattern in the inferior leads and V2 caused by fusion of QRS, ST-segment, and T waves, is another high-risk pattern reflecting presence of large area of **transmural ischemia**. This pattern predicting significant mortality Possible cause; subarachnoid hemorrhage causing transient stress-induced apical ballooning cardiomyopathy, The literature on this distinct ECG phenomenon is scant, consisting essentially of case reports. Therefore, its incidence is unknown. Presumably many cases go unrecognized and are mistaken for conduction abnormalities, metabolic derangements, **adrenergic stimulus**, or toxicological insult. From the cases that have been described, Shark Fin appears to be an ominous sign with a strikingly poor prognosis.

Possible condition: Transient stress-induced apical ballooning cardiomyopathy, **1, 2** Takotsubo cardiomyopathy (TTC)3 or broken heart syndrome.**4** adrenergic cardiomyopathy, and with the eponymous Gebrochenes-Herz syndrome.**5** This entity is characterized as a transient segmental cardiac dysfunction mimicking ACS triggered by emotional or physical stress. Although neurological disorders, infection, malignant diseases, trauma and surgery are known triggers for the development of TC, role of cardiac diseases as underlying conditions for the development of TTC is uncertain. TCM may develop in critically ill cardiac diseases but are often underdiagnosed. Careful echocardiographic examination is needed to unveil these "hidden" TTC. Women are more prone than men to experience the symptoms of broken heart syndrome, particularly Asian and Caucasian postmenopausal women.

1. **Bybee KA, Kara T, Prasad A, *et al.* Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. Ann Intern Med. 2004;141:858-65. doi: 10.7326/0003-4819-141-11-200412070-00010**
2. **Tsuchihashi K, Ueshima K, Uchida T, *et al.* Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Angina Pectoris-Myocardial Infarction Investigations in Japan. J Am Coll Cardiol. 2001;38:11-8. doi: 10.1016/s0735-1097(01)01316-x**
3. **Kurisu S, Sato H, Kawagoe T, *et al.* Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. Am Heart J. 2002;143:448-55. doi: 10.1067/mhj.2002.120403**
4. **Sancar F. For "Broken Heart" Syndrome, Brain May Hold the Key. JAMA. 2019;321:2270-71. doi: 10.1001/jama.2019.5198**
5. **Ahmad SA, Brito D, Khalid N, Ibrahim MA. Takotsubo Cardiomyopathy. StatPearls. Treasure Island (FL)2021.**

Les cuento como siguió el caso.
La paciente no presentó alteraciones hemodinámicas en las siguiente horas. El dosaje de troponina ultra sensible ligeramente positiva, sin curva.
Ecocardiograma con leve deterioro de la función ventricular izquierda, aquinesia apical con contractilidad conservada en el resto de los segmentos (Takotsubo?) A las 12 horas se repitió nuevo ECG ver en la próxima diapositiva
En virtud que no presentaba trastornos de la motilidad de distribución epicárdica, troponina sin curva, y además por el estado de la paciente, se decidió no realizar la coronarografia.

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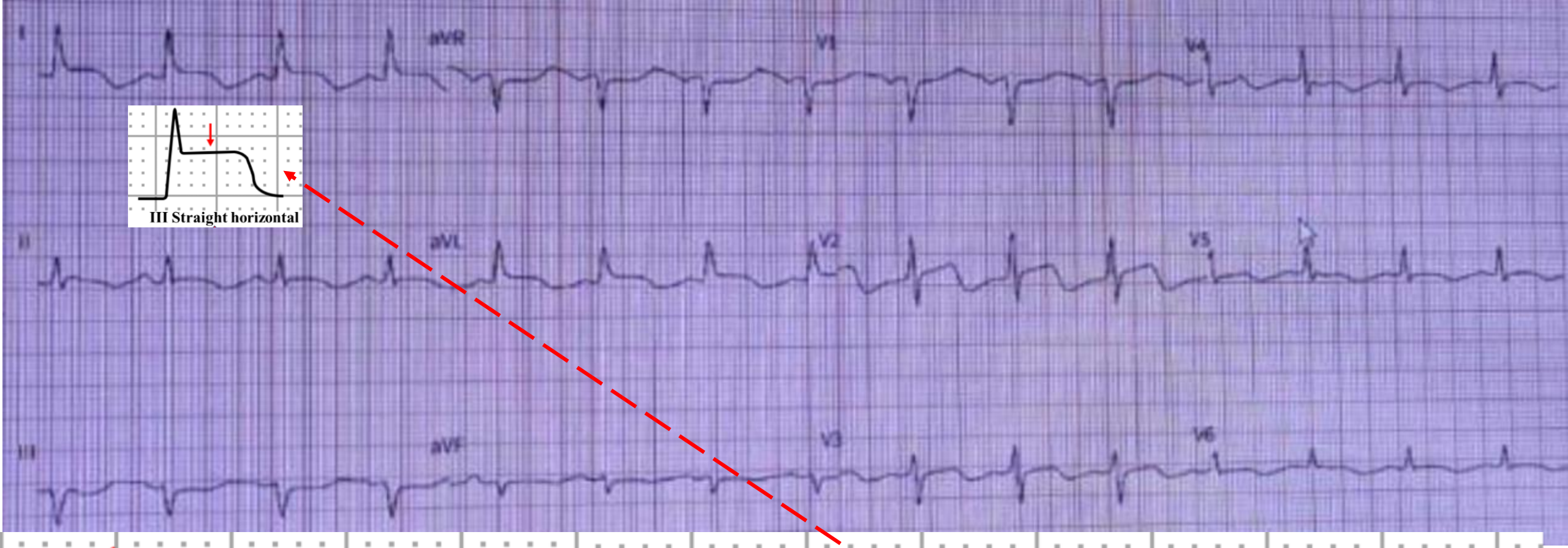
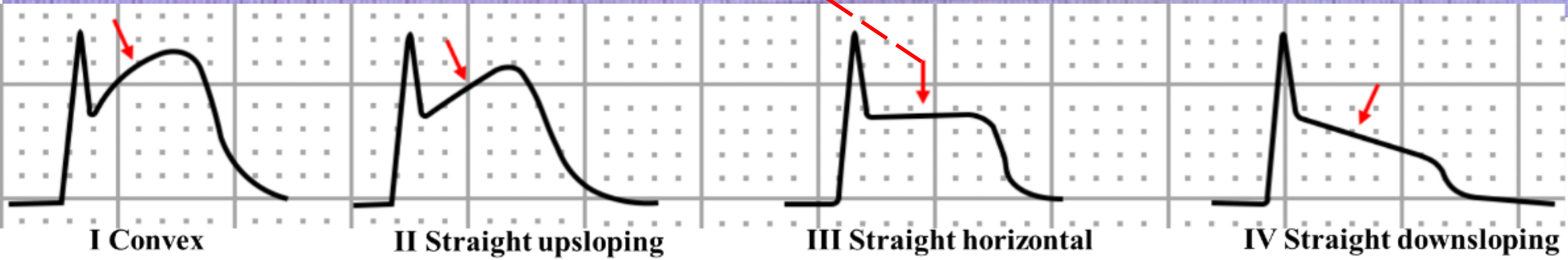
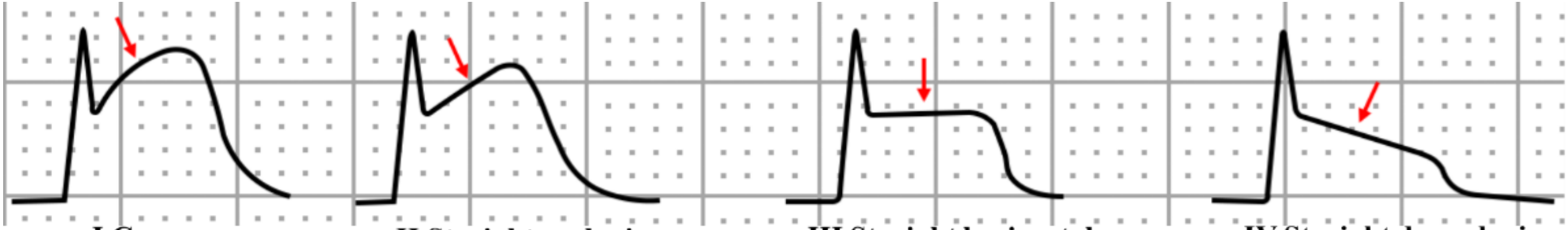
I tell you how the case continued. The patient did not present hemodynamic alterations in the following hours. Ultra-sensitive troponin dosage slightly positive, no curve. Echocardiogram with slight deterioration of left ventricular function, apical akinesia with conserved contractility in the rest of the segments (takotsubo?) At 12 hours a new ECG was repeated, see on the next slide Since there were no epicardial distribution motility disorders , troponin without curve, and also due to the patient's condition, it was decided not to perform coronariography.



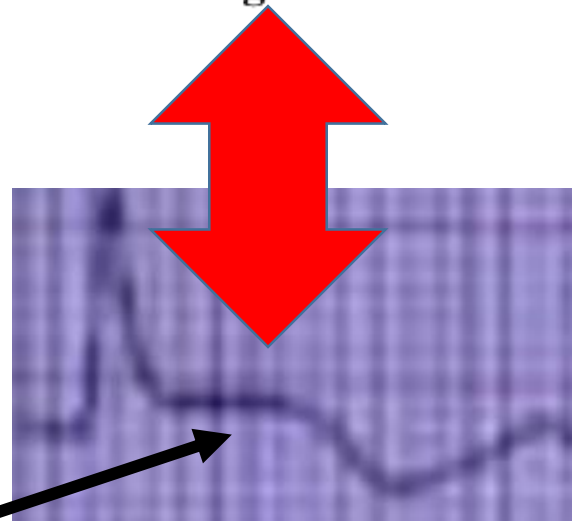


Patterns characteristics of STE caused by acute transmural myocardial ischemia/myocardial infarction

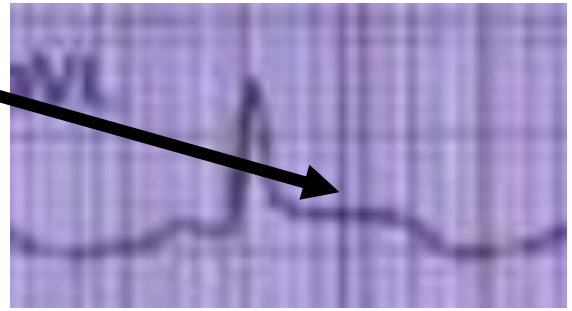
Patterns of STE caused by acute transmural myocardial ischemia/myocardial infarction



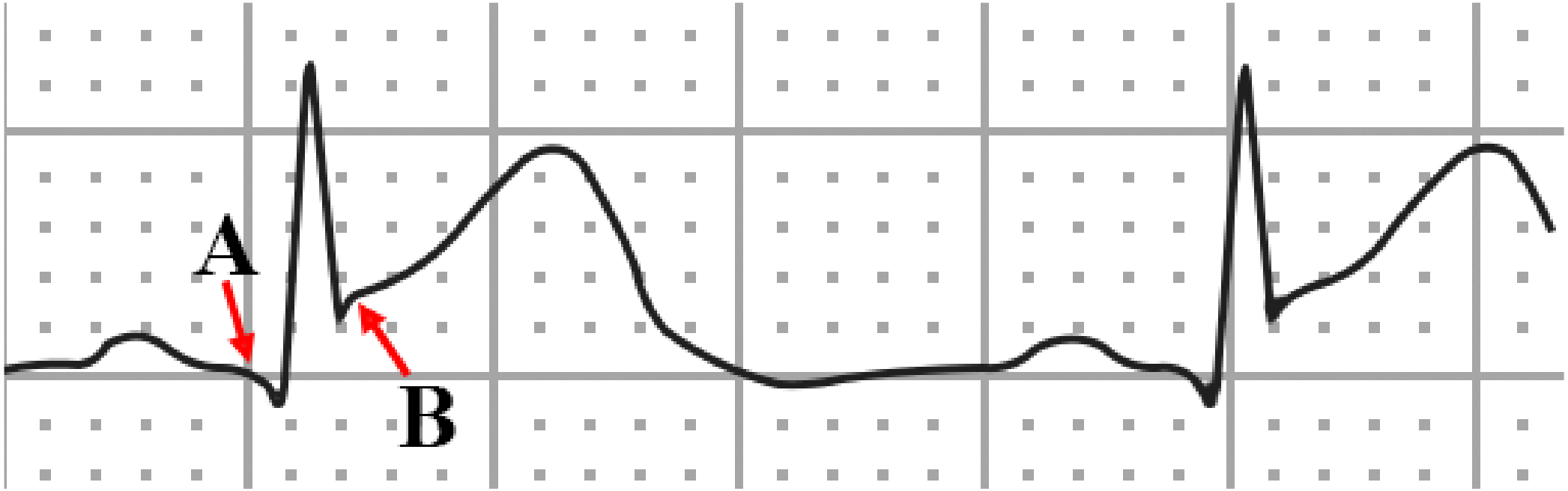
I Convex | II Straight upsloping | III Straight horizontal | IV Straight downsloping



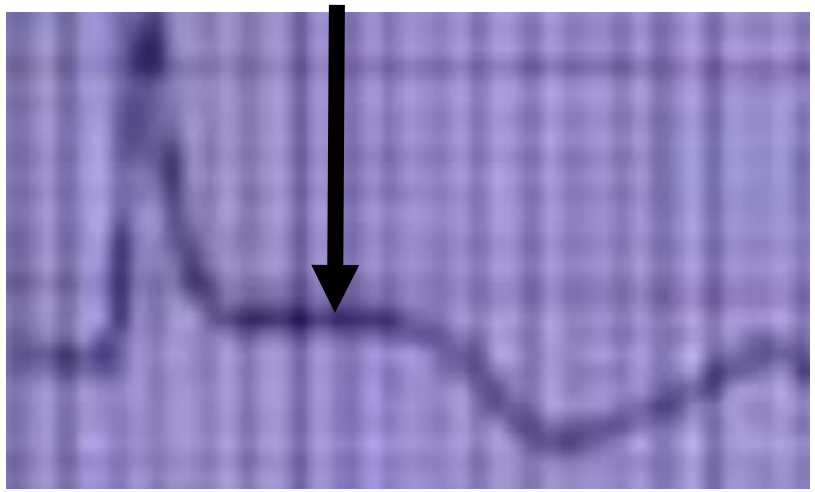
Franco Di Prinzio´s case

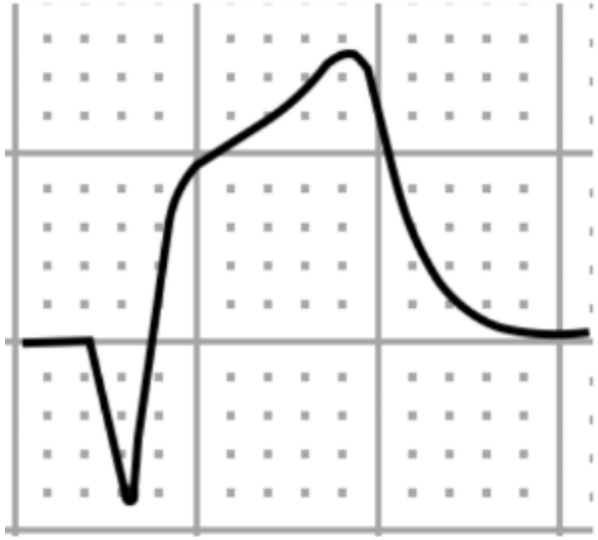
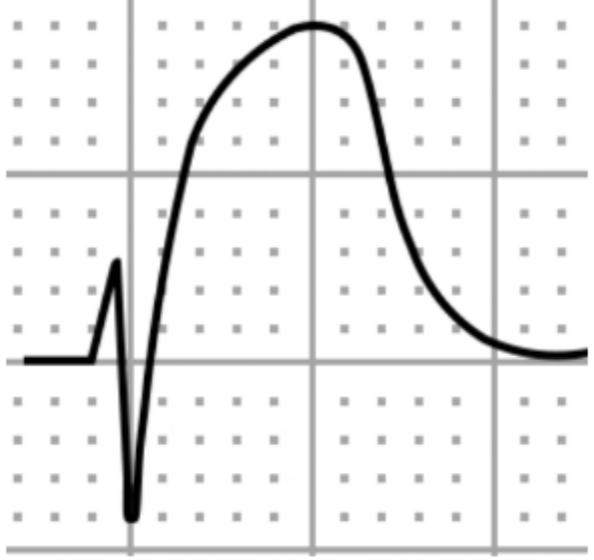
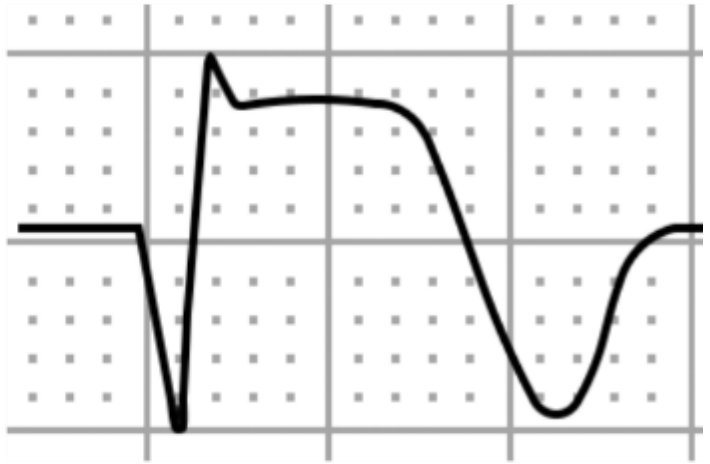
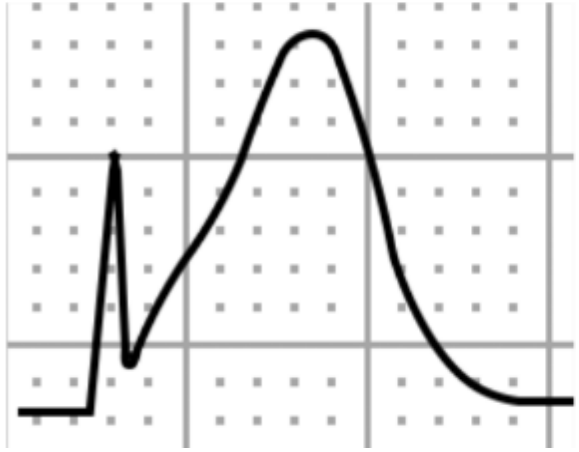
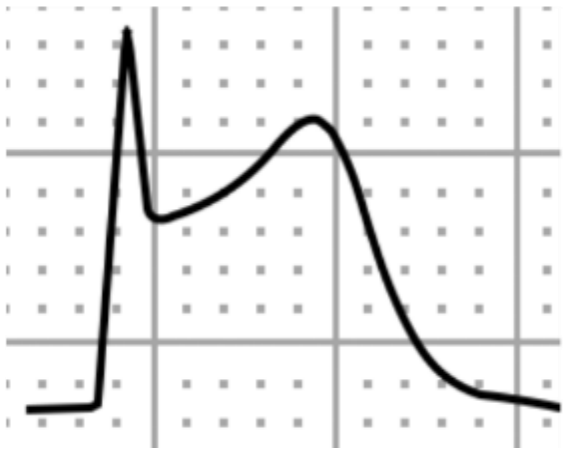
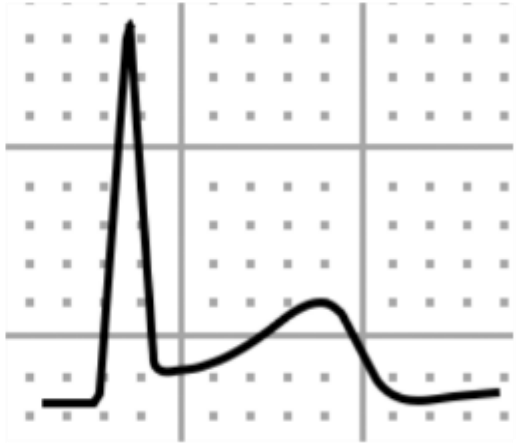


Typical example of ischemic STEMI

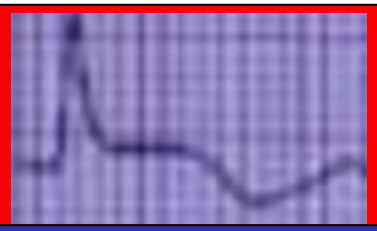
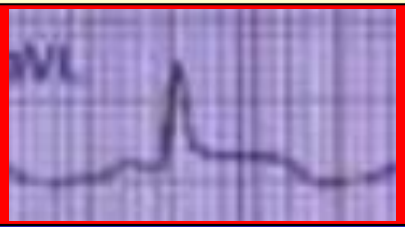
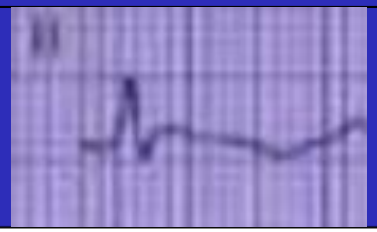
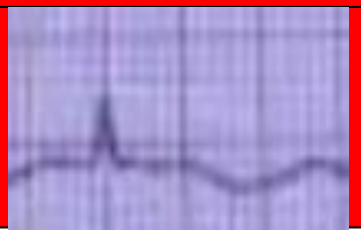


The initial onset of the Q wave shown by arrow A serves as the reference point and arrow B shows the onset of the ST-segment or J-point. The difference between the two identifies the magnitude of displacement. Measurements of both arrows should be made from the top of the ECG line tracing.STE with straight horizontal, down loping or convex ST segment strongly suggest acute transmural ischemia



ST-segment elevation can vary markedly in appearance. These six examples were retrieved from six different patients with STEMI.

	aVR	V1 SEPTAL	V4 ANTERIOR
II INFERIOR		V2 SEPTAL	V5 LATERAL
	aVF INFERIOR	V3 ANTERIOR	

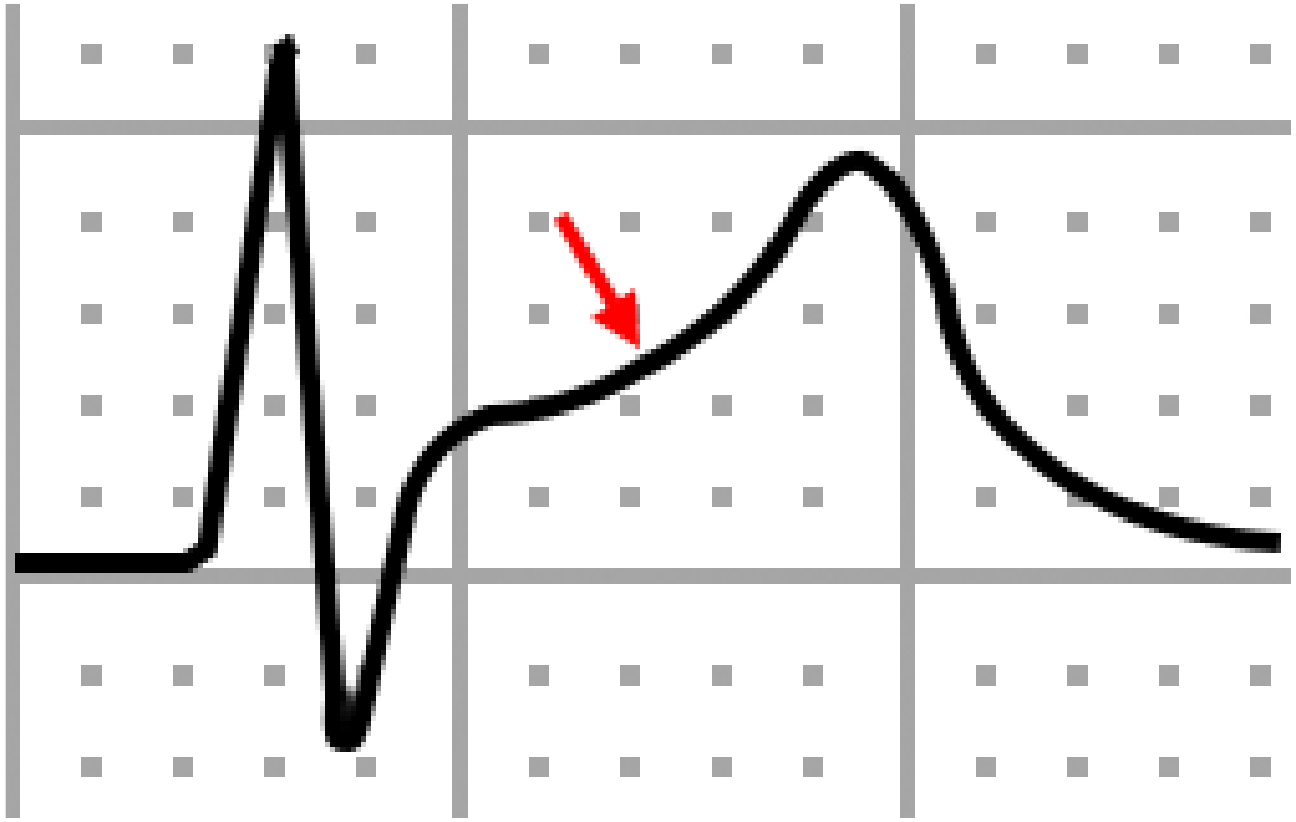
I LATERAL	aVR	V1 SEPTAL	V4 ANTERIOR
II INFERIOR	aVL LATERAL	V2 SEPTAL	V5 LATERAL
III INFERIOR	aVF INFERIOR	V3 ANTERIOR	V6 LATERAL

Note:

The dorsal/posterior, high lateral walls do not exist!!!!!! **Bayes de Luna A, Wagner G, Birnbaum Y, *et al.* A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. Circulation. 2006;114:1755-60. doi: 10.1161/CIRCULATIONAHA.106.624924**

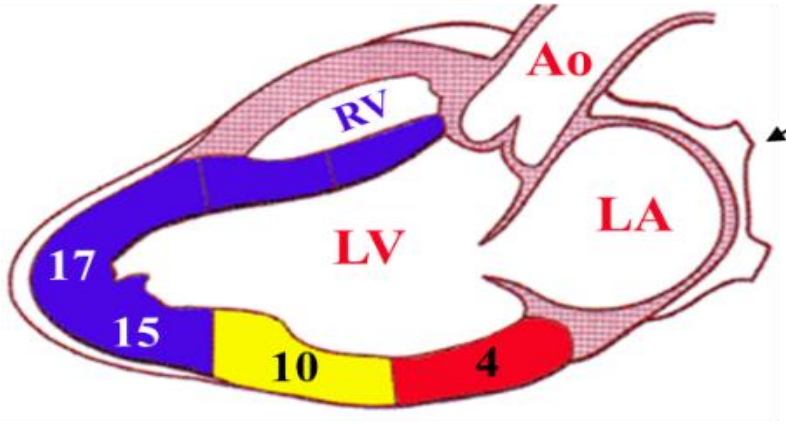
See explanation in next slides……

Concave STEs, on the other hand, are much less likely to be caused by ischemia however, a concave STE does not rule out ischemia, it merely reduces the probability of ischemia as the underlying cause (Figure).

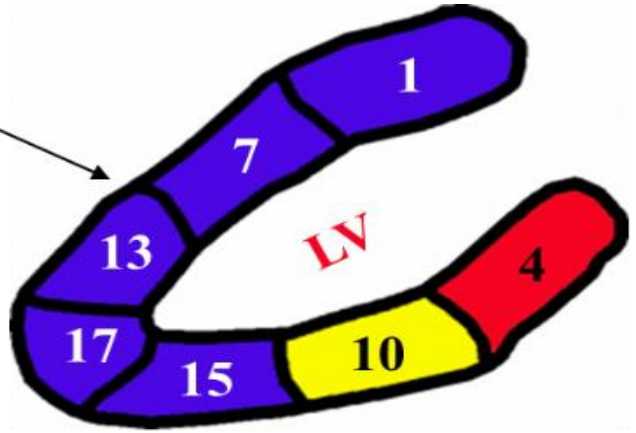


Typical non-ischemic ST-segment elevation. Non-ischemic STEs are very frequent in heathy population. They are characterized by a concave STE and greater distance between the J point ant the T wave apex.

Ventricular segmentation heart walls with CE-CMRI



Longitudinal paraesternal



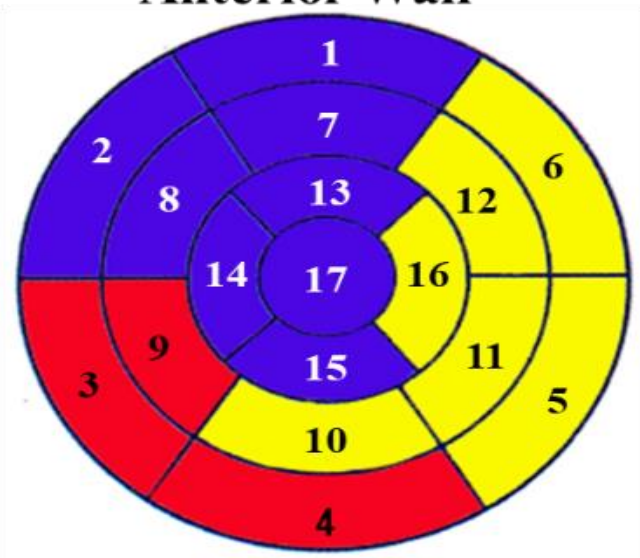
Apical four chambers

Apical two chambers

Bulls eye (apical short axis)

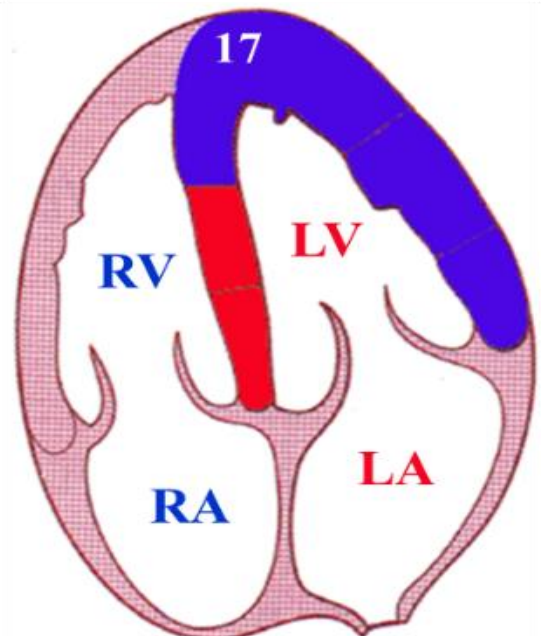
Anterior Wall

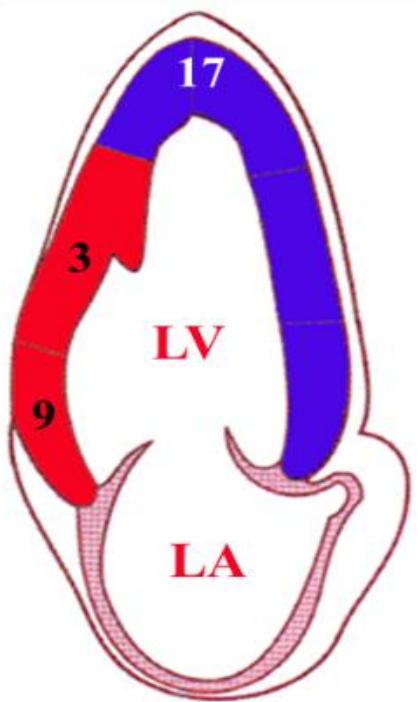
Septal Wall



Lateral Wall

Inferior Wall



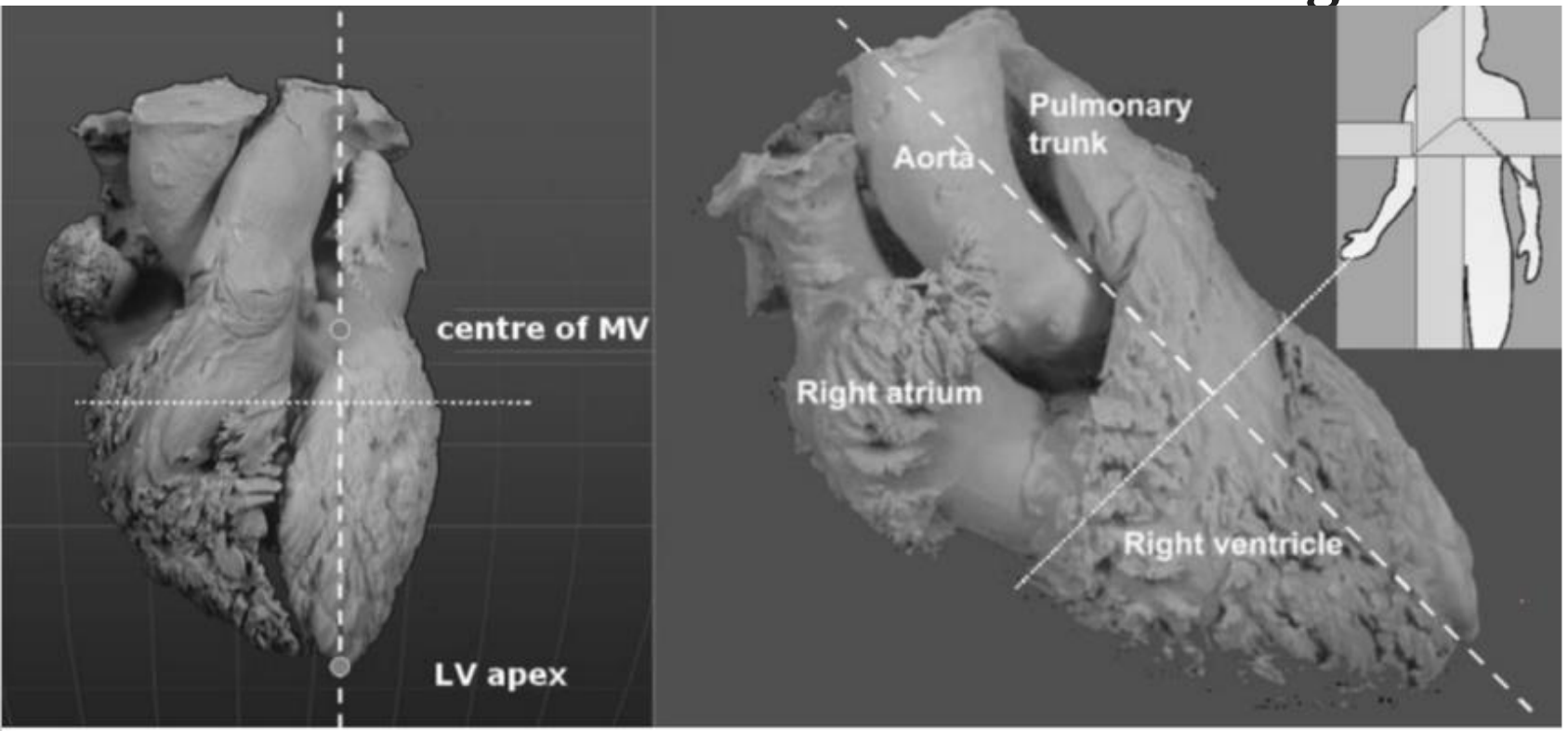


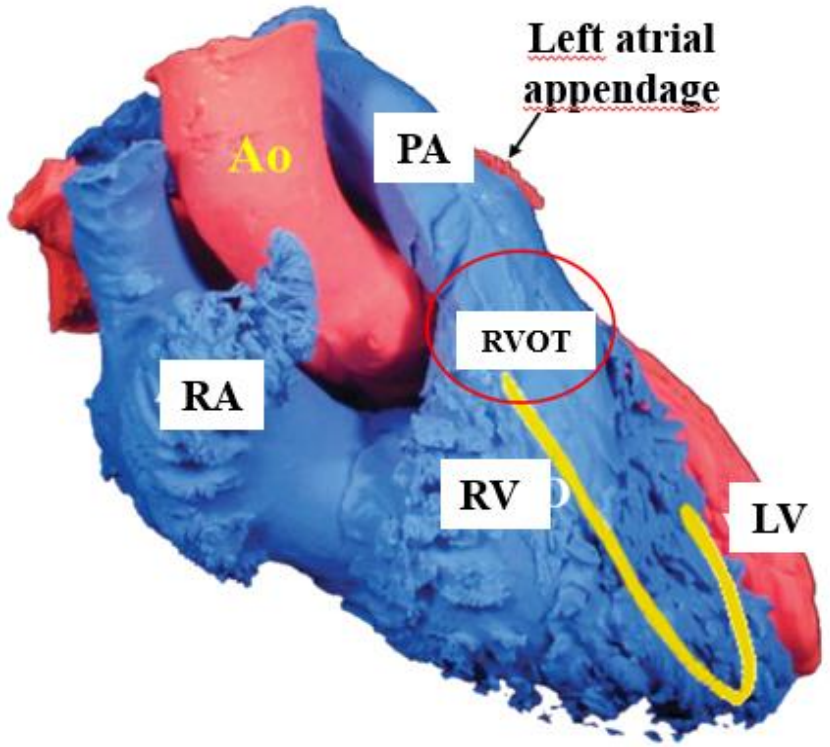
Left Anterior Descending (LAD)

Left Circunflex (LCx)

Posterior Descending (RCA or LCx)

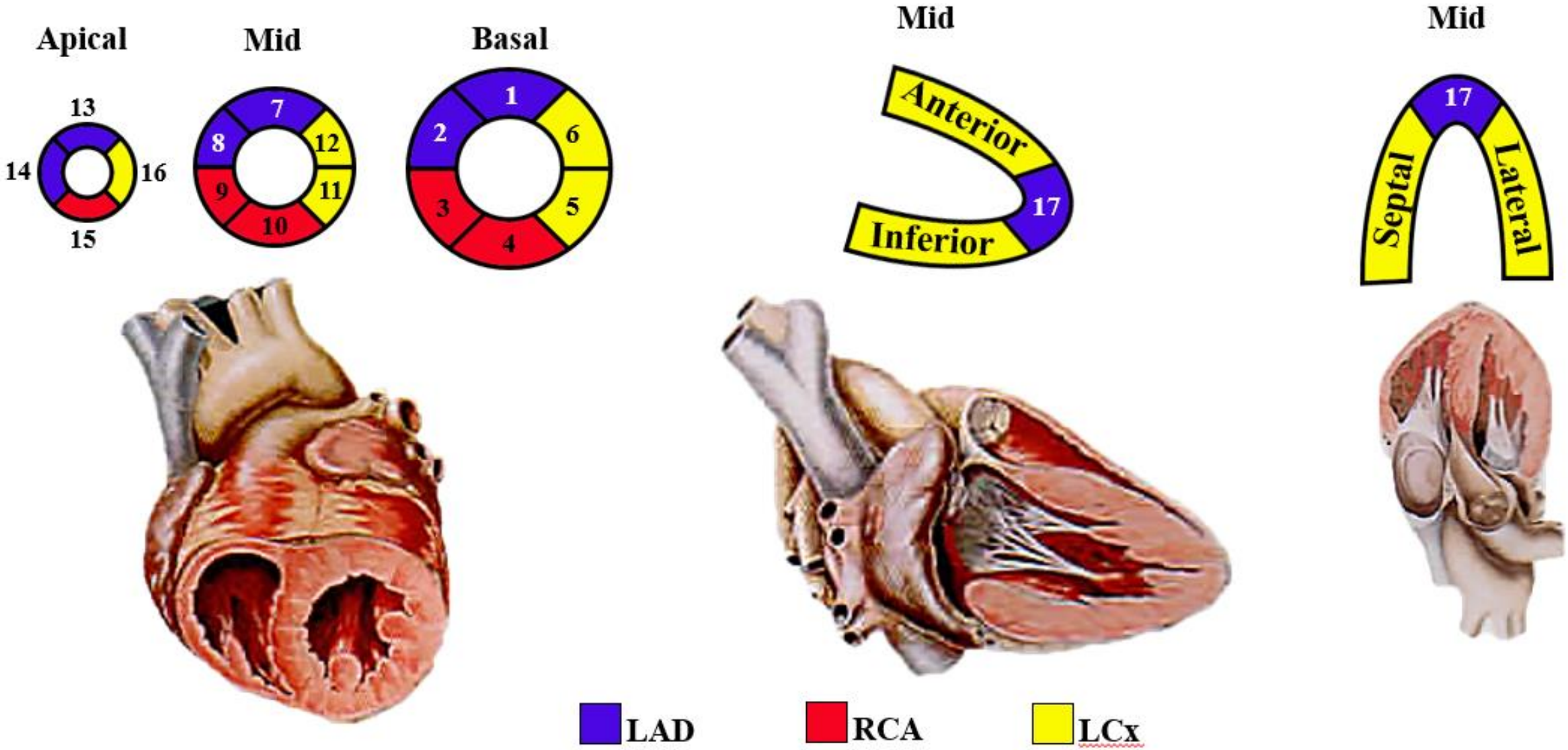
Current nomenclature of the heart wall segmentation with CE-CMRI



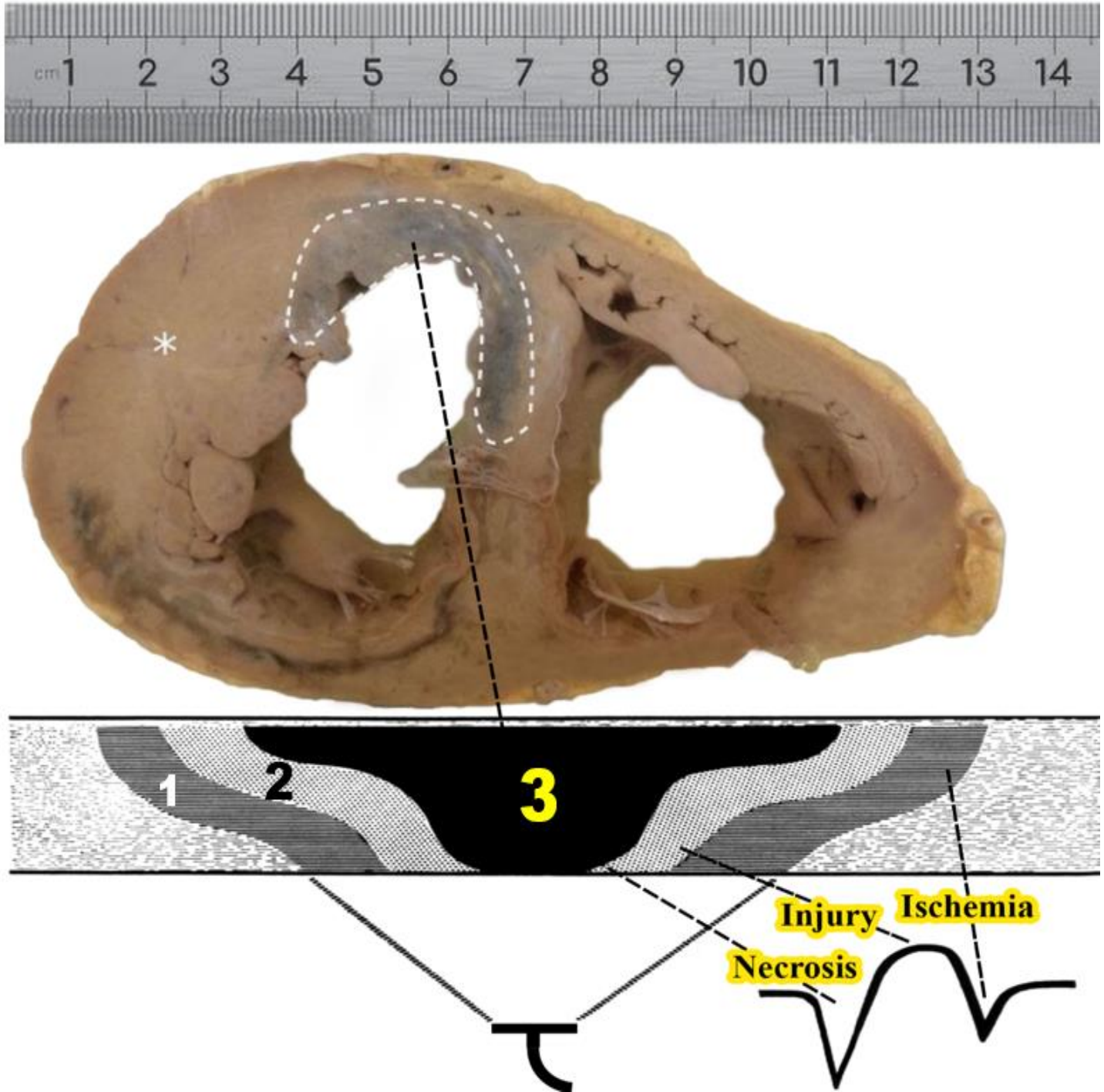


The left panel shows the heart in its ‘‘Valentine’’ position, with the long axis of the left ventricle and its defining points (dashed line) and a short axis (dotted line). In the right panel, we have positioned the heart in attitudinally appropriate fashion, showing the angulation of the ventricular axes relative to the axes of the body.

Coronary artery territories[1]



1. Cerqueira MD, Weissman NJ, Dilsizian V*, et al.* Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Circulation. 2002;105:539-42. doi: 10.1161/hc0402.102975



Specimen showing myocardial infarction in the LV and the interventricular septum. The asterisk (*) also indicates LVH. After 20 minutes of experimental or natural total obstruction of a coronary branch, three concentric areas are delimited in the affected myocardium:

1. **Peripheral or ischemic area:** it modifies the T wave. In it, there is a discrete circulatory deficit, and only metabolic and functional alterations without ultrastructural injuries.
2. **Intermediate injury area:** it modifies the ST segment. There is a greater circulatory deficit than in the previous one, and ultrastructural modifications; however, without cell death and still reversible.
3. **Central or necrosis area:** it modifies the QRS complex (Q wave > 40 ms). There is maximal circulatory deficit and structural injuries, nearly always irreversible.

New hs-cTn tests are currently available and if used appropriately can substantially improve management. Because of their high sensitivity and accuracy, these tests allow measurement of very low serum troponin levels, such as those present in healthy individuals and can detect small changes in troponin concentration within a short time frame. These tests are thus, very useful for the early diagnosis of AMI but can also be elevated in several other conditions that result in myocardial injury. A good understanding of the analytical characteristics of these assays is of uppermost importance for their appropriate use in clinical practice.

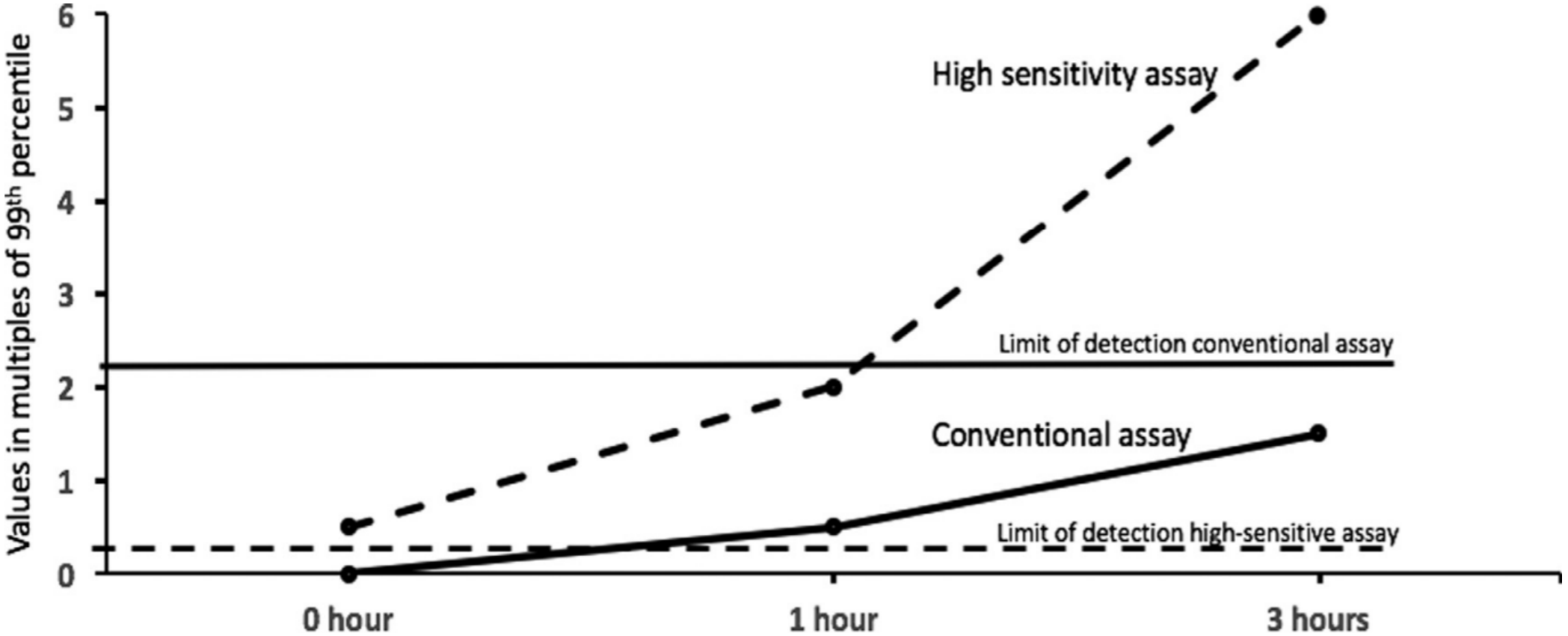


Figure . Comparison of high-sensitivity and conventional troponin assays. With conventional assay, levels of troponin are below the lower detection limit during the first 3 hours after onset of AMI. Measurements should be repeated 6 to 12 hours later in order to detect a significant rise. With hs-cTn assays, very low levels can be measured at time of admission and any minor change can be detected after 1 hour. This allows a much earlier diagnosis of AMI. Rabih R Azar 1, Antoine Sarkis 2, Evangelos Giannitsis 3 A Practical Approach for the Use of High-Sensitivity Cardiac Troponin Assays in the Evaluation of Patients With Chest Pain Am J Cardiol. 2021 Jan 15;139:1-7. doi: 10.1016/j.amjcard.2020.10.037

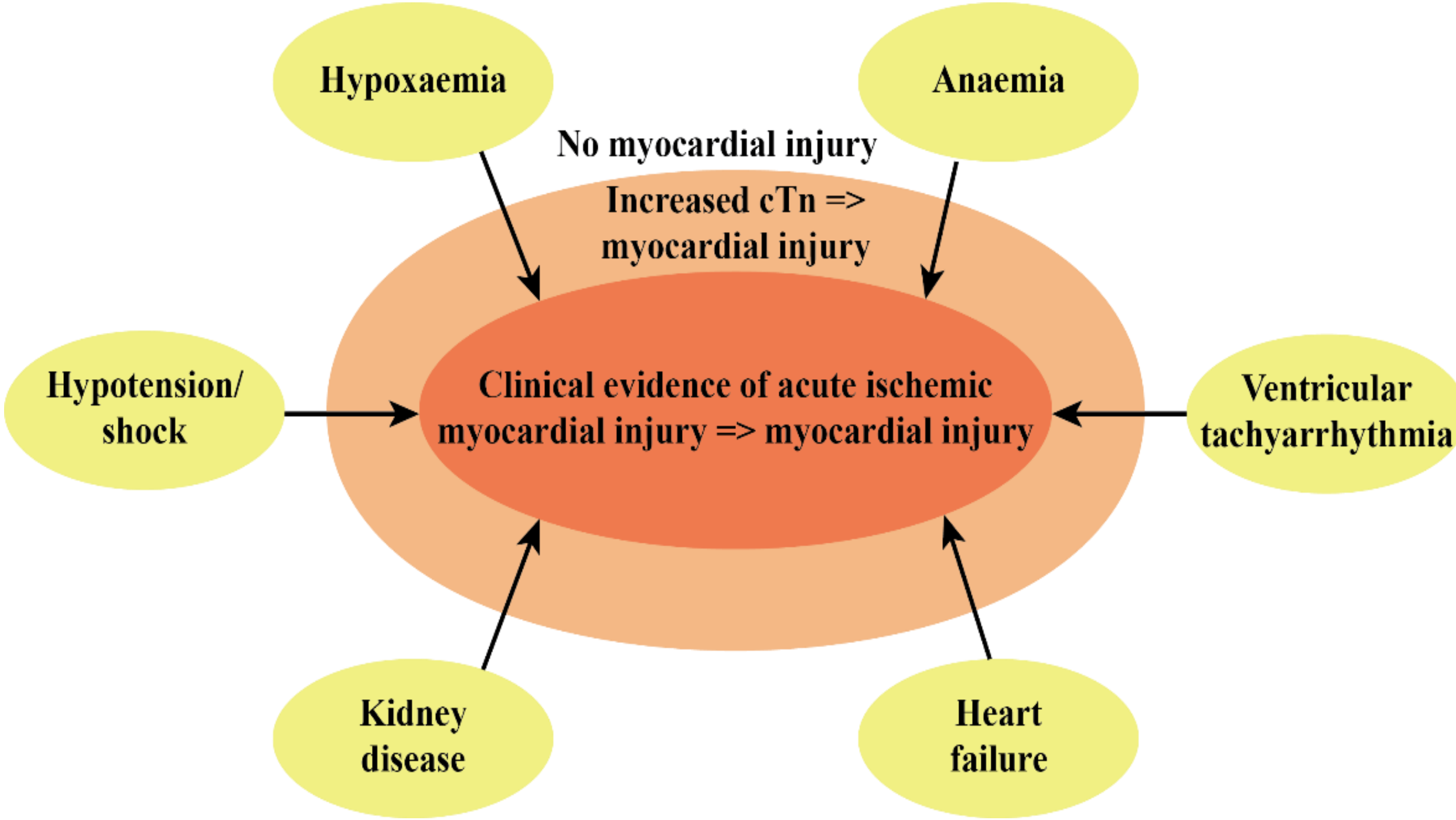
Value of high-sensitivity cardiac troponin T test hs-cTnT

hs-cTnT: high-sensitivity cardiac troponin T test. cTnI and cTnT are the preferred biomarkers for the evaluation of myocardial injury (hs-cTnT),**1-3** and high-sensitivity (hs)-cTn assays. Troponin assays are recommended for routine clinical use.67 Creatine kinase MB isoform (CK-MB) is less sensitive and less specific.**1** Myocardial injury is defined as being present when blood levels of cTn are increased above the 99th percentile upper reference limit (URL).**4-7-** The injury may be acute, as evidenced by a newly detected dynamic rising and/or falling pattern of cTn values above the 99th percentile URL, or chronic, in the setting of persistently elevated cTn levels. Criteria for myocardial injury. Detection of an elevated cTn value above the 99th percentile URL is defined as myocardial injury. The injury is considered acute if there is a rise and/or fall of cTn values. Although elevated cTn values reflect injury to myocardial cells, they do not indicate the underlying pathophysiological mechanisms, and can arise following preload-induced mechanical stretch or physiological stresses in otherwise normal hearts.**8-10** Various causes have been suggested for the release of structural proteins from the myocardium, including normal turnover of myocardial cells, apoptosis, cellular release of cTn degradation products, increased cellular wall permeability, the formation and release of membranous blebs, and myocyte necrosis. **11;12** Yet, it is not clinically possible to distinguish which increases of cTn levels are due to which mechanisms. **13** However, regardless of the mechanism, acute myocardial injury, when associated with a rising and/or falling pattern of cTn values with at least one value above the 99th percentile URL and caused by myocardial ischemia, is designated as an AMI. .**4-7** Histological evidence of myocardial injury with myocyte death can be detected in clinical conditions associated with non-ischemic mechanisms of myocardial injury as well76, **14;15** (Figure).

References

1. **Goodman SG, Steg PG, Eagle KA*, et al.* The diagnostic and prognostic impact of the redefinition of acute myocardial infarction: lessons from the Global Registry of Acute Coronary Events (GRACE). Am Heart J. 2006;151:654-60. doi: 10.1016/j.ahj.2005.05.014**
2. **Siriwardena M, Campbell V, Richards AM, Pemberton CJ. Cardiac biomarker responses to dobutamine stress echocardiography in healthy volunteers and patients with coronary artery disease. Clin Chem. 2012;58:1492-4. doi: 10.1373/clinchem.2012.187682**

3. Turer AT, Addo TA, Martin JL, *et al.* Myocardial ischemia induced by rapid atrial pacing causes troponin T release detectable by a highly sensitive assay: insights from a coronary sinus sampling study. J Am Coll Cardiol. 2011;57:2398-405. doi: 10.1016/j.jacc.2010.11.066
4. Apple FS, *et al.* IFCC educational materials on selected analytical and clinical applications of high sensitivity cardiac troponin assays. Clin Biochem. 2015;48:201-3. doi: 10.1016/j.clinbiochem.2014.08.021
5. Thygesen K, Alpert JS, Jaffe AS, *et al.* Third universal definition of myocardial infarction. Circulation. 2012;126:2020-35. doi: 10.1161/CIR.0b013e31826e1058
6. Thygesen K, *et al.* How to use high-sensitivity cardiac troponins in acute cardiac care. Eur Heart J. 2012;33:2252-7. doi: 10.1093/eurheartj/ehs154
7. Thygesen K, *et al.* Recommendations for the use of cardiac troponin measurement in acute cardiac care. Eur Heart J. 2010;31:2197-204. doi: 10.1093/eurheartj/ehq251
8. Siriwardena M, et al Cardiac biomarker responses to dobutamine stress echocardiography in healthy volunteers and patients with coronary artery disease. Clin Chem. 2012;58:1492-4. doi: 10.1373/clinchem.2012.187682
9. Turer AT, Addo TA, Martin JL, *et al.* Myocardial ischemia induced by rapid atrial pacing causes troponin T release detectable by a highly sensitive assay: insights from a coronary sinus sampling study. J Am Coll Cardiol. 2011;57:2398-405. doi: 10.1016/j.jacc.2010.11.066
10. Weil BR, et al . Troponin Release and Reversible Left Ventricular Dysfunction After Transient Pressure Overload. J Am Coll Cardiol. 2018;71:2906-16. doi: 10.1016/j.jacc.2018.04.029
11. Mair J, Lindahl B, Hammarsten O, *et al.* How is cardiac troponin released from injured myocardium? Eur Heart J Acute Cardiovasc Care. 2018;7:553-60. doi: 10.1177/2048872617748553
12. White HD. Pathobiology of troponin elevations: do elevations occur with myocardial ischemia as well as necrosis? J Am Coll Cardiol. 2011;57:2406-8. doi: 10.1016/j.jacc.2011.01.029
13. Jaffe AS, Wu AH. Troponin release--reversible or irreversible injury? Should we care? Clin Chem. 2012;58:148-50. doi: 10.1373/clinchem.2011.173070
14. Eggers KM, Lindahl B. Application of Cardiac Troponin in Cardiovascular Diseases Other Than Acute Coronary Syndrome. Clin Chem. 2017;63:223-35. doi: 10.1373/clinchem.2016.261495
15. Giannitsis E, Katus HA. Cardiac troponin level elevations not related to acute coronary syndromes. Nat Rev Cardiol. 2013;10:623-34. doi: 10.1038/nrcardio.2013.129


Hypoxaemia
Anaemia
No myocardial injury
Increased cTn =>
myocardial injury
Hypotension/
shock
Clinical evidence of acute ischemic
myocardial injury => myocardial injury
Ventricular
tachyarrhythmia
Kidney
disease
Heart
failure

Another Case report example of TTC takotsubo.

Case Presentation: A 71-year-old woman was admitted to the emergency room with typical symptoms of acute retrosternal chest pain that radiated into the upper arms. The pain was accompanied by perfuse sweating and occurred immediately after a very stressful family conflict.

Physical examination: Hemodynamically stable. BP = 140/90 mmHg; HR = 75 bpm; regular heart rhythm and without murmurs; clear lungs; warm limbs; and no peripheral edema.

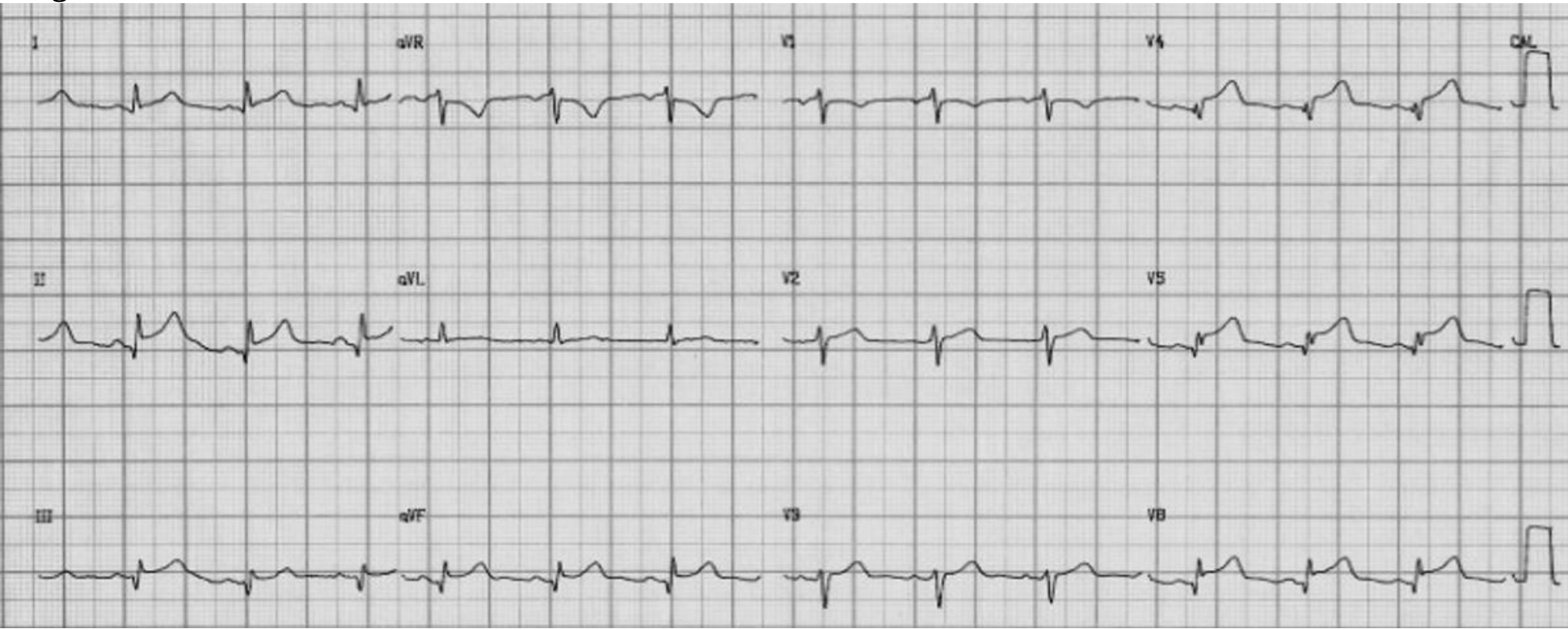
The admitting ECG is shown in **Figure 1**. After the initial evaluation she underwent coronary angiography which revealed normal coronary arteries. Left ventriculography showed apical hypokinesis/akinesis, and in the mid-ventricular region there was regional ballooning (**Figure 2**).

Necrosis markers (troponin) showed minimal elevation.

Echocardiogram made on the day of admission revealed LV mid-apical akinesis with a LVEF of 41% (**Figure 3A**).

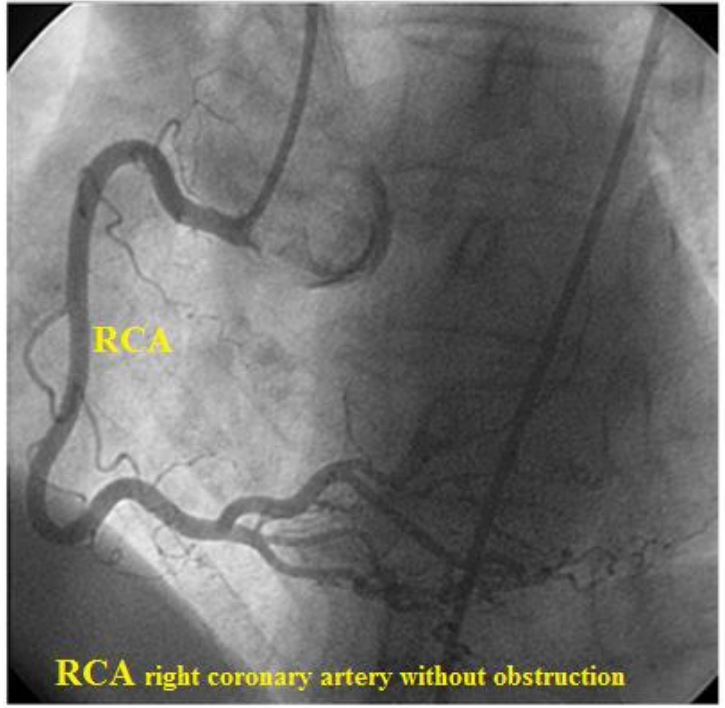
A repeat echocardiogram one week later showed recovery of contractile dysfunction and a LVEF of 57% indicative of the transitory nature of this phenomenon (**Figure 3B**).

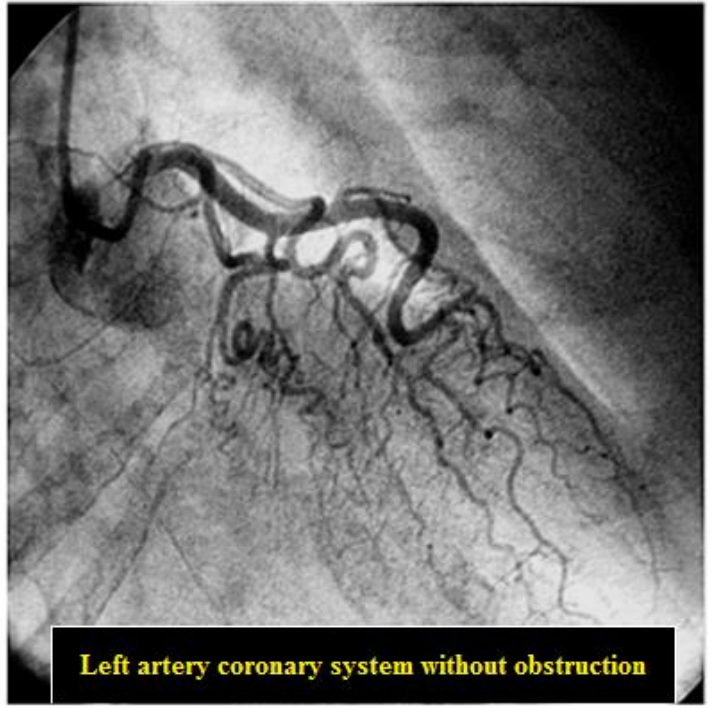
Figure 1

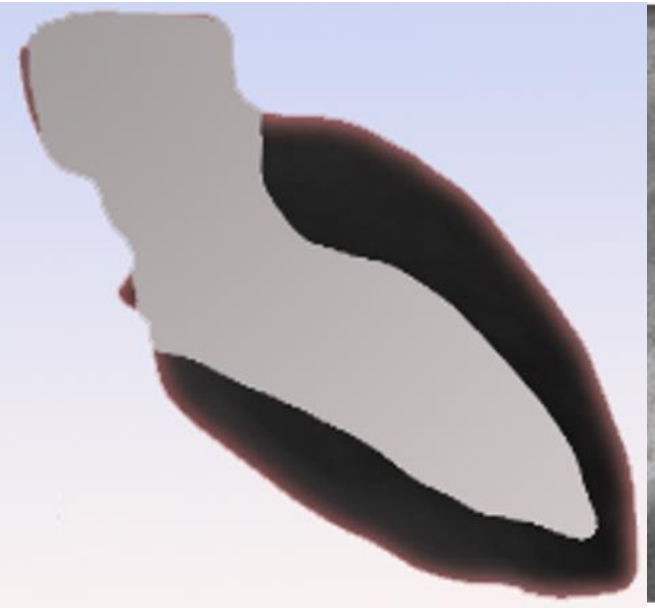


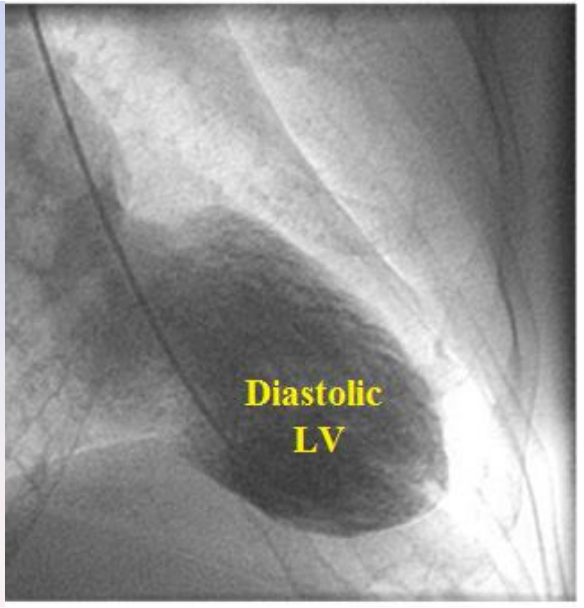
Electrocardiographic diagnosis: sinus rhythm, rate 75 bpm, QRS axis +30°, wide-spread ST segment elevation with positive T wave in inferior (II, III and aVF), antero-apical (from V3 through V6) and high lateral wall (I and aVL) with reciprocal change in aVR (subepicardial circumferential ischemic syndrome). The lesion vector is directed inferior, anterior and leftwards. The combination of ST segment depression in aVR with absence of ST elevation in V1 is diagnostic of takotsubo with 91% sensitivity, 96% specificity and 95% predictive accuracy.(**Kosuge M, Ebina T, Hibi K, *et al.* Simple and accurate electrocardiographic criteria to differentiate takotsubo cardiomyopathy from anterior acute myocardial infarction. J Am Coll Cardiol. 2010;55:2514-6. doi: 10.1016/j.jacc.2009.12.059**)

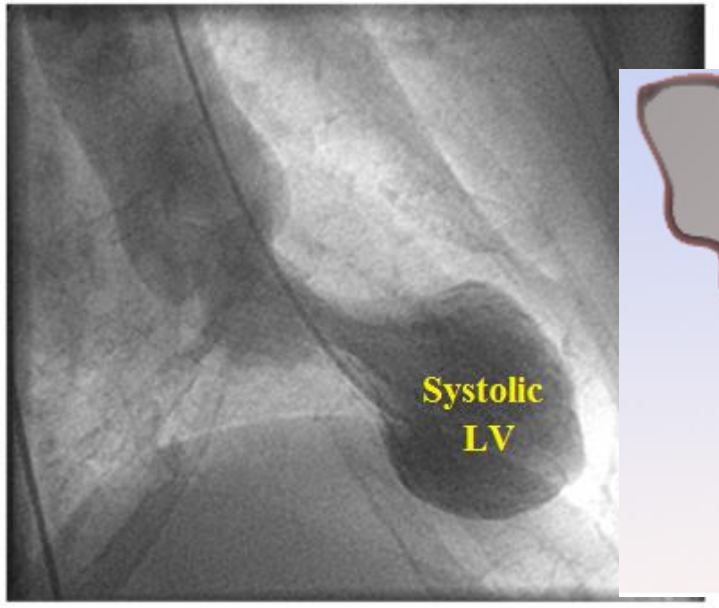
Fig 2


RCA
RCA right coronary artery without obstruction


Left artery coronary system without obstruction




Diastolic
LV


Systolic
LV

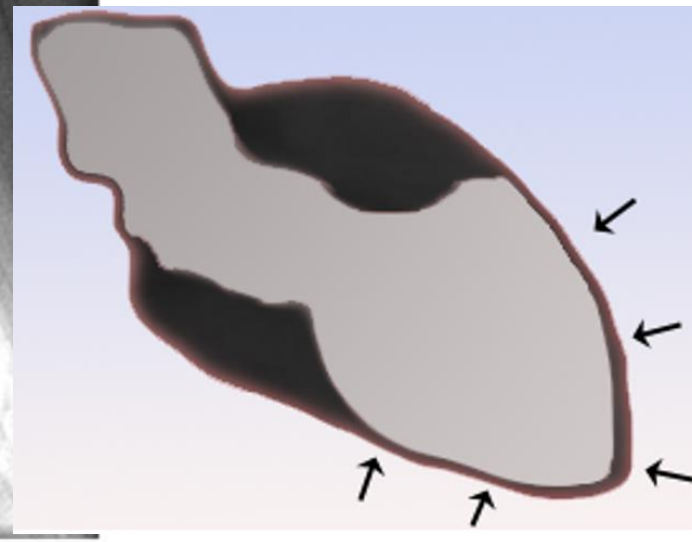
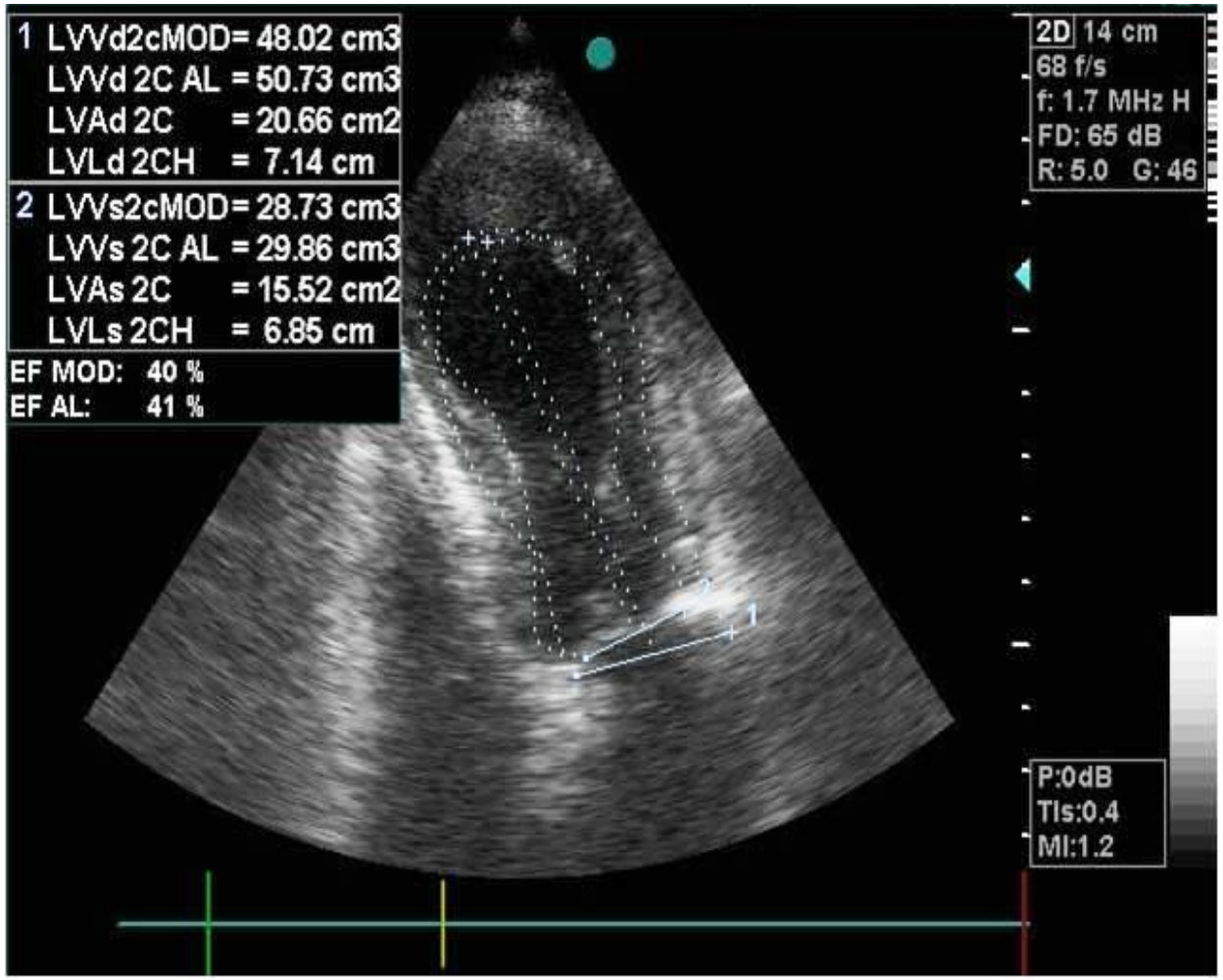
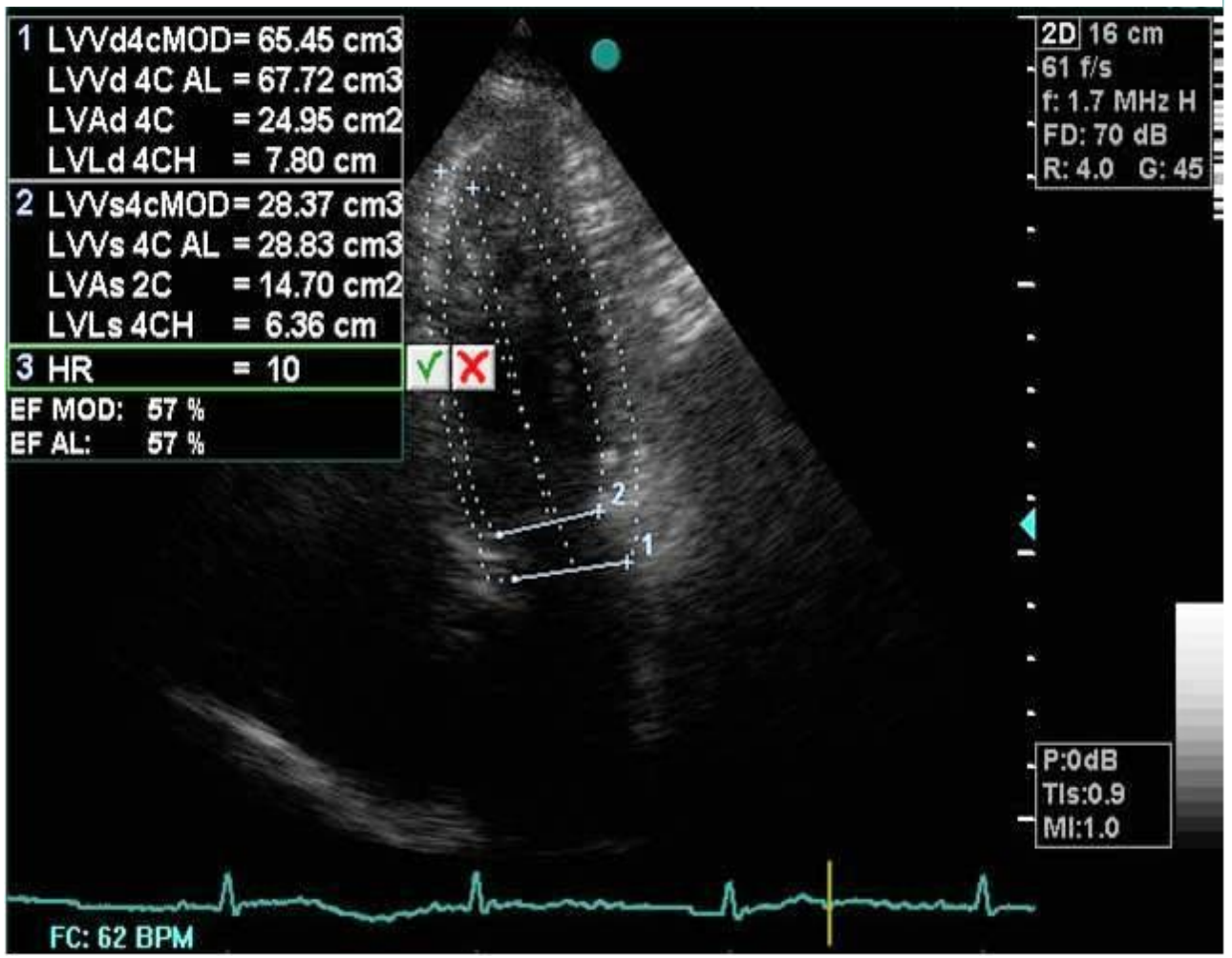


Figure 3A



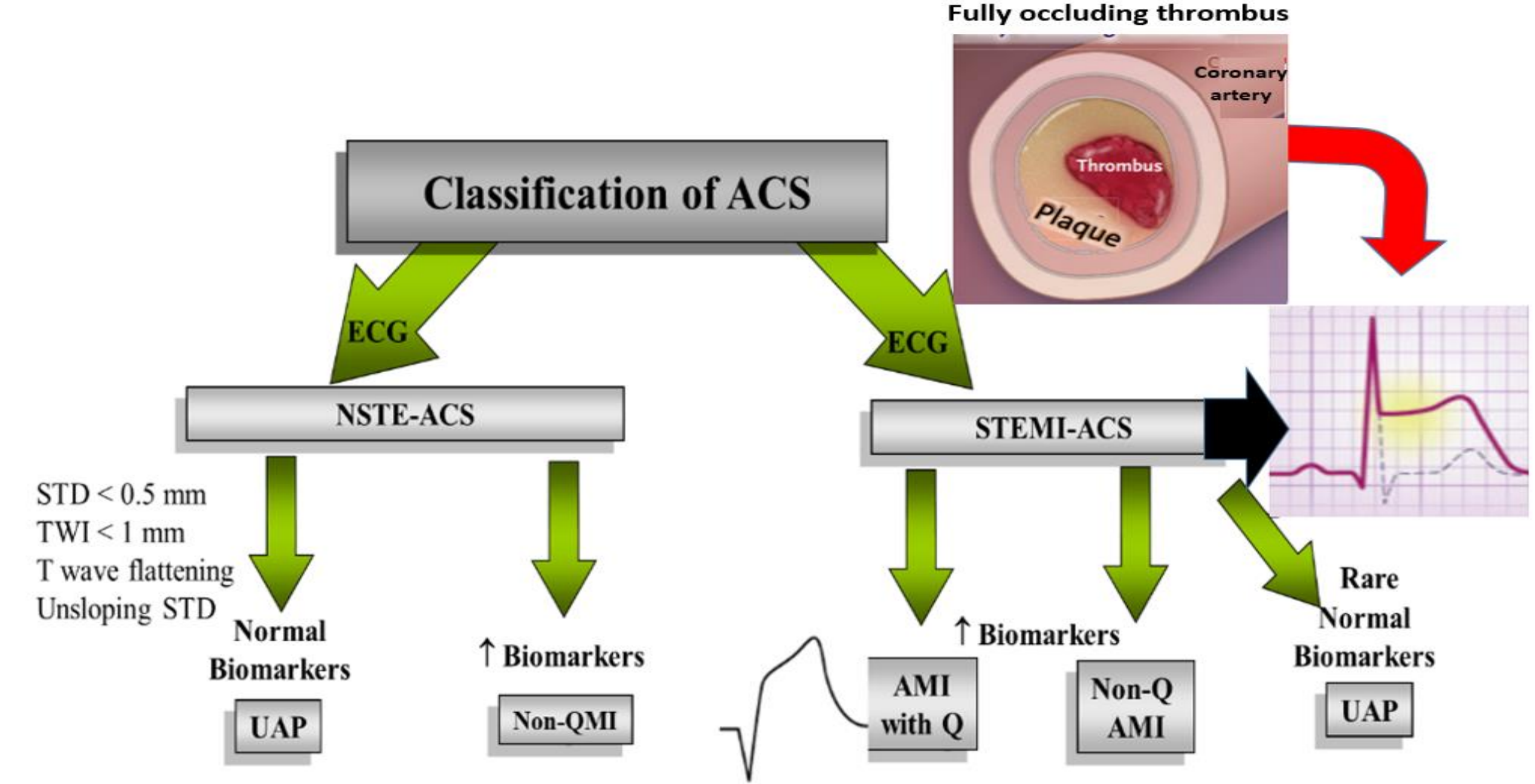
Echocardiogram on admission

Figure 3B



Echocardiogram 1 week later

Classical classification of ACSs: Unstable Angina Pectoris, NSTEMI and STEMI



UAP (Unstabe Angina Pectoris) and NSTEMI

The basis for UA or UAP and NSTEMI differentiation is the presence of biomarkers of myocardial damage (cardiac-specific troponins) in the blood sample of patients with NSTEMI and the lack of those in patients with UAP.

An ACSs without significant (Non-specific). STE are classified as NSTE-ACS. In most case there are STDs and/or T-Wave Inversion (TWI). A minority of patients with NSTE-ACS display normal ECG through the course.

Pathophysiology: Partial occlusion causing ischemia located to the sub endocardium. NSTEMI as the presence of suggestive symptoms, cTnI≥0.2ng/ dL and/or dynamic ST-segment changes (STD ≥1 mm or non-persistent elevation in ≥2 contiguous derivations).

UA or UAP is defined as the presence of suggestive thoracic pain with or without repolarization abnormalities in the baseline ECG, and whit serum levels of **cTnI <0.2 ng/dL 24** hours after the first symptoms appear.

STEMI

Diagnostic criteria: all ACSs with significant STEs are classified as STE-ACS. The ECG will usually also display ST-segment depression (STD) and/or T-Wave Inversion (TWI).

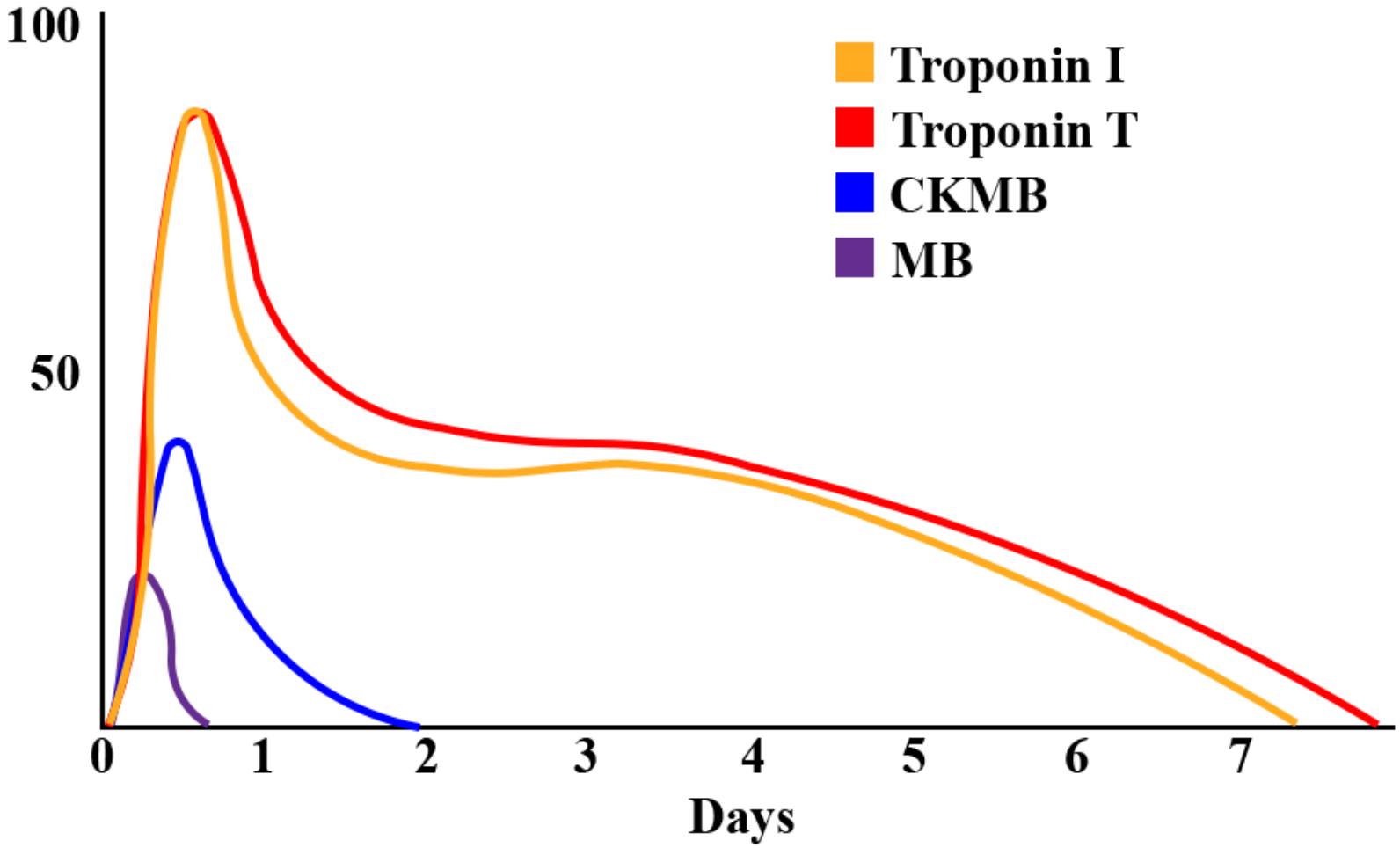
Pathophysiology: Total occlusion in a coronary artery. This causes extensive ischemia which is transmural (i.e. stretches from the endocardium to the epicardium). These infarctions are large and usually leads to development of pQ-waves in leads with STE.

Acute Coronary Syndrome

Unstable Angina Pectoris (UAP)	NSTEMI	STEMI
The plaque ruptures and a thrombus forms around the ruptured plaque, causing **partial** occlusion of the vessel. Angina pain occurs at rest or progresses rapidly over a short period of time.	During an NSTEMI, the plaque rupture and thrombus formation causes **partial** occlusion to the vessel that results in injury and infarct to the **subendocardial** myocardium.	A STEMI is characterized by **complete** occlusion of the blood vessel lumen resulting in **transmural** injury and infarct to the myocardium, which is reflected by ECG changes and rise in troponin.
Normal biomarkers	**Augmented biomarkers↑**	**Augmented biomarkers ↑**

Stable angina

Angina pain develops when there is increased demand in the setting of stable atherosclerotic plaque. The vessel is unable to dilate enough to allow adequate blood flow to meet the myocardial demand.



Levels of myocardial proteins in the circulation following MI. Note that cardiac troponins peak after 24 to 28 hours after initiation of MI. Troponin levels increase within 2 or 3 hours after onset of myocardial necrosis. Levels are normalized within 7 days. The slow normalization is due to slow ongoing leakage of troponin from necrotic cells. A negative (i.e normal) troponin 6 hours after the last episode of symptoms rules out myocardial infarction (it does not rule out UA). With high-sensitive troponin assays it is possible to rule out myocardial infarction after 3 hours. Troponin levels at 24hours after onset of symptoms may be sued to estimate the size of the infarction.

Chronic Total Occlusion (CTO)

Chronic total occlusion (CTO) refers to complete luminal diameter stenosis with resultant thrombolysis in myocardial infarction (TIMI) grade flow 0 or 1.**1** In such, there is no anterograde flow due to collaterals.**1** The occlusion should be of at least three months' duration to be labeled as chronic.**2** The main vessels that are affected due to CTO are the RCA (43% to 55%), the LAD artery (approximately 24%), and the LCx artery (17%-20%). **3;4** Functional CTOs represent severe occlusion, but not a complete occlusion of the coronary arteries. Such functional CTOs have collateral circulations presenting as anterograde flow.**5** However, even such collateral flow is unable to sustain future ischemic events in functional CTOs, indicating a need for revascularization.6

References

1. **Fefer P, *et al.* Current perspectives on coronary chronic total occlusions: the Canadian Multicenter Chronic Total Occlusions Registry. J Am Coll Cardiol. 2012;59:991-7. doi: 10.1016/j.jacc.2011.12.007**
2. **Stone GW, *et al.* Percutaneous recanalization of chronically occluded coronary arteries: a consensus document: part I. Circulation. 2005;112:2364-72. doi: 10.1161/CIRCULATIONAHA.104.481283**
3. **Tajti P, *et al.* The Hybrid Approach to Chronic Total Occlusion Percutaneous Coronary Intervention: Update From the PROGRESS CTO Registry. JACC Cardiovasc Interv. 2018;11:1325-35. doi: 10.1016/j.jcin.2018.02.036**
4. **Tsai TT, *et al.* Contemporary Incidence, Management, and Long-Term Outcomes of Percutaneous Coronary Interventions for Chronic Coronary Artery Total Occlusions: Insights From the VA CART Program. JACC Cardiovasc Interv. 2017;10:866-75. doi: 10.1016/j.jcin.2017.02.044**
5. **Ivanhoe RJ,., *et al.* Percutaneous transluminal coronary angioplasty of chronic total occlusions. Primary success, restenosis, and long-term clinical follow-up. Circulation. 1992;85:106-15. doi: 10.1161/01.cir.85.1.106**
6. **Werner GS, et al . Immediate changes of collateral function after successful recanalization of chronic total coronary occlusions. Circulation. 2000;102:2959-65. doi: 10.1161/01.cir.102.24.2959**

FUDMI: The **F**ourth **U**niversal **D**efinition of **M**yocardial **I**nfarction. The FUDMI Expert Consensus Document updates the definition of MI to accommodate the increased use of high-sensitivity cardiac troponin (hs-cTn). Detection of an elevated cTn value above the 99th percentile upper reference limit (URL) is defined as myocardial injury.