https://youtu.be/06MqF6nL_GA

When is a positive Troponin not an NSTEMI?!

"When Troponin was a lousy assay it was a great test. Now that it's a great assay, it's a lousy test."

On the relative value of an assay versus a test: a history of troponin for the diagnosis of myocardial infarction. Jesse R L, J Am Coll Cardiol 55:2125-2128, 2010.

Objectives

- Distinguish myocardial injury from myocardial infarction.
- Review symptoms that would warrant ordering Troponin and EKG.
- Offer a differential diagnosis of myocardial injury.
- Review ST- and T-wave changes and renew a differential diagnosis, including ischemia.

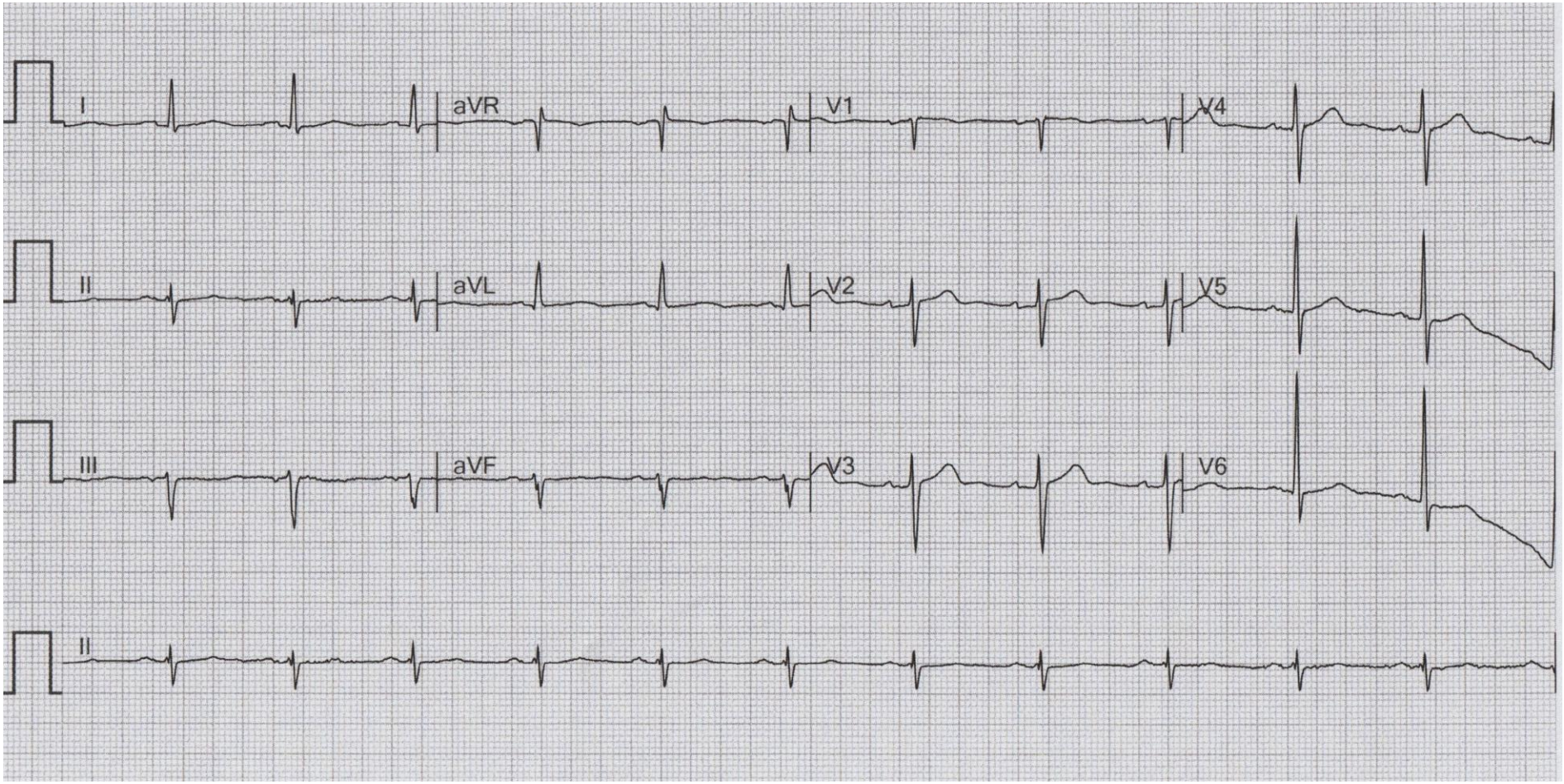
62-yo male

- PMH: +HTM, +HChol, -DM, -CAD/MI, + pancreatic CA, newly Dx liver mets post chemotherapy, +PE three months earlier;
- PSH: Whipple, pancreatectomy 3 years earlier;
- Presents to his oncologist office on 2/11/2019 CC: “throwing up yellow stuff,” denies chest pain, DOE, - leg swelling/pain/redness;
- Admitted with NSTEMI based on first Troponin

✓		Reg Date ▲	Type	Loc	Dis Date	Account Num	Provider	Reason for Vi
☐		03/18/19	DIS ONCRx	VONCMED	03/26/19	V00030774335	Naseri,Hussain M	PANCREATIC METASTATIC, R51,
☐	🚑	03/08/19	DEP ERx	VER	03/08/19	V00031177496	Aulick,Neal	seizure ? / pt a&o x3
☐	🚑	03/02/19	DEP ERx	VER	03/02/19	V00031163223	Rudis,Steven P	WEAKNESS
☐		03/01/19	SCH RCRx	EOUT		E00031766488	Suders,Daniel J	klebsiella bacteremia
☐	🚑	02/22/19	DIS INx	V5W	02/28/19	V00031144918	Greco,Rick	Leukocytosis, Hyponatremia, Me
☐		02/22/19	**REG RCRx**	EOUT		E00031699846	Crake,Robert J	R78.81
☐		02/19/19	DEP REFx	EOR	02/19/19	E00031759699	Petersen,Joseph M	No Longer Needed
☐	🚑	02/11/19	DIS INx	VTCU	02/20/19	V00031116932	Suders,Daniel J	NSTEMI
☐		02/08/19	REG CLIx	VCT		V00031105216	Naseri,Hussain M	cardio toxic chemo Z51.81
☐		01/27/19	DEP CLIx	EOUT	01/27/19	E00031711245	Das,Anup K	IV THERAPY

Troponin I

Collected	Result	Units	Range	Group
02/13/2019 17:30	0.210 H*	NG/ML	0-0.034	
02/13/2019 07:15	0.246 H*	NG/ML	0-0.034	
02/12/2019 20:15	0.298 H*	NG/ML	0-0.034	
02/12/2019 12:40	0.345 H*	NG/ML	0-0.034	
02/12/2019 07:00	0.290 H*	NG/ML	0-0.034	
02/11/2019 22:50	0.243 H*	NG/ML	0-0.034	
02/11/2019 18:40	0.196 H*	NG/ML	0-0.034	
02/11/2019 15:00	0.162 H*	NG/ML	0-0.034	


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

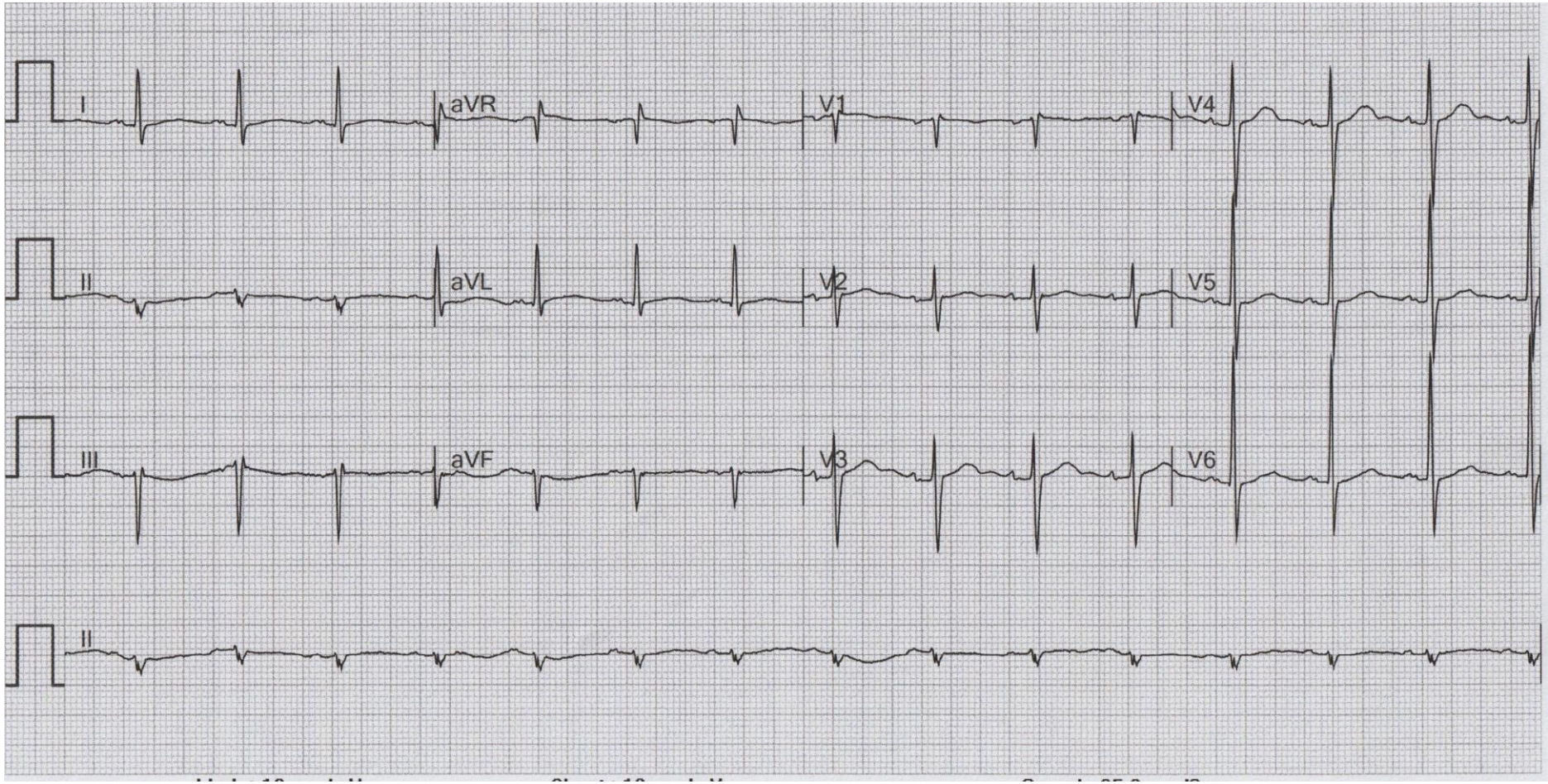
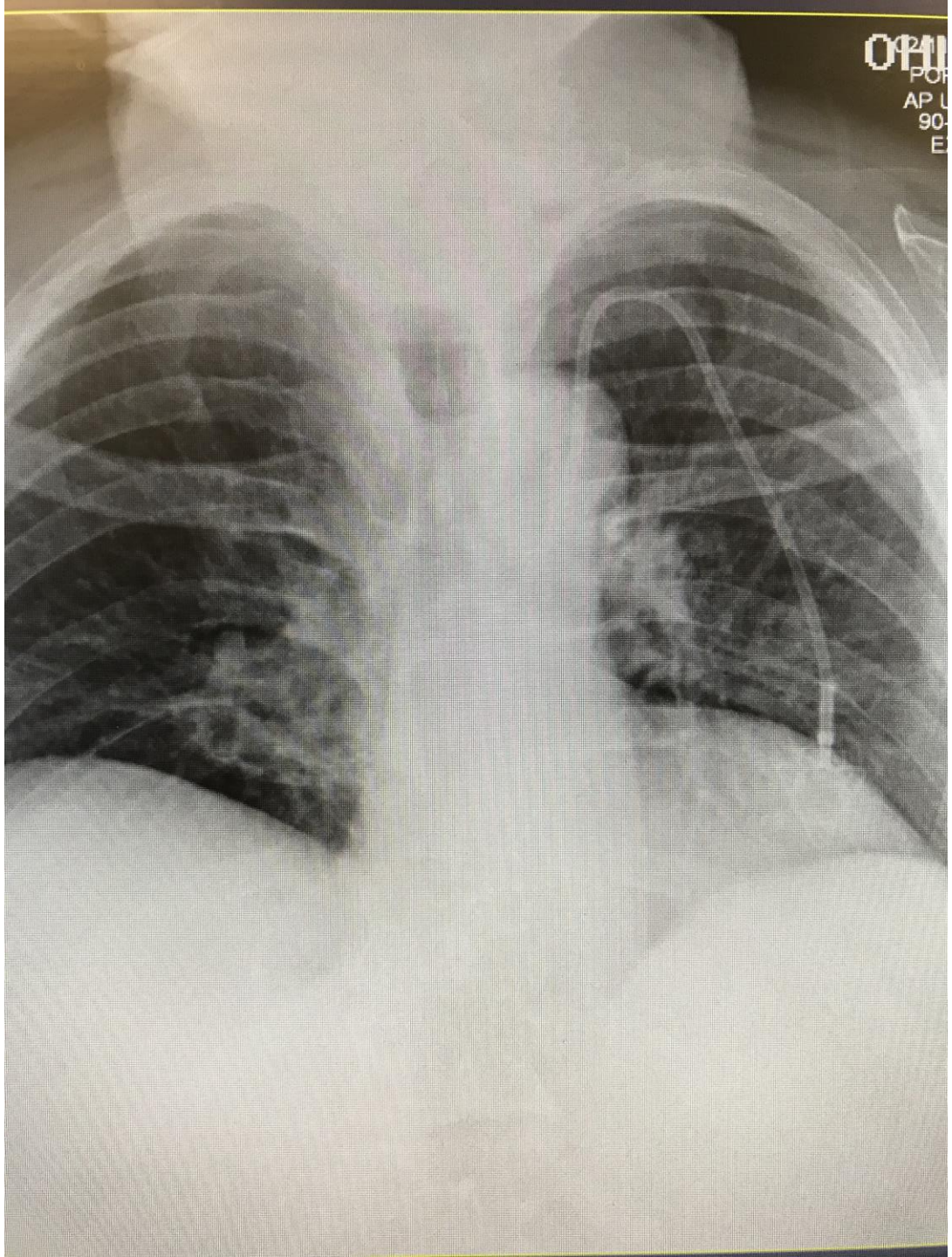

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

TABLE 2 **Electrocardiographic manifestations suggestive of acute myocardial ischaemia (in the absence of left ventricular hypertrophy and bundle branch block)**

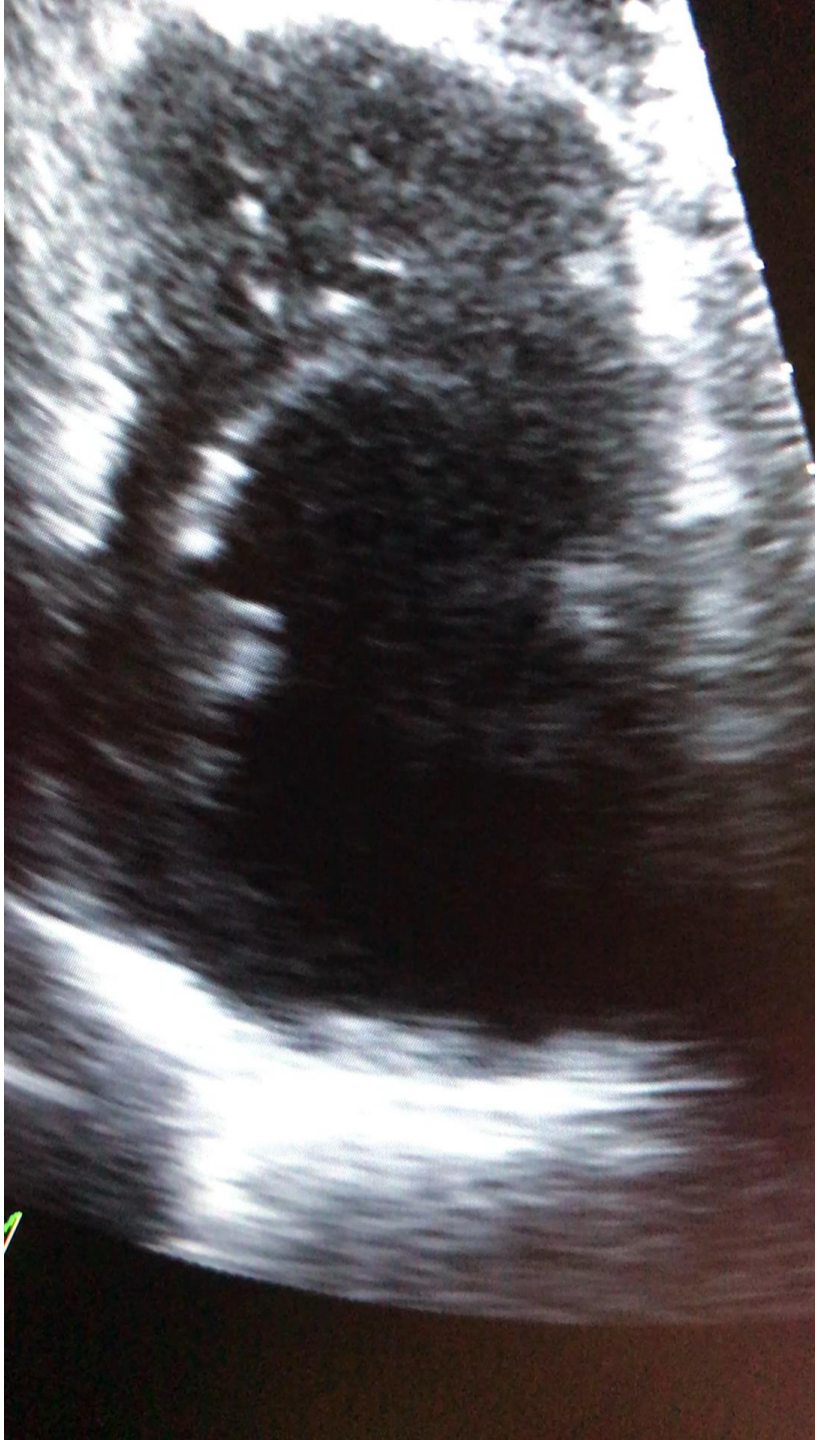
ST-elevation
New ST-elevation at the J-point in two contiguous leads with the cut-point: ≥ 1 mm in all leads other than leads V_2–V_3 where the following cut-points apply: ≥ 2mm in men ≥ 40 years; ≥ 2.5 mm in men < 40 years, or ≥ 1.5 mm in women regardless of age.[a]
ST-depression and T wave changes
New horizontal or downsloping ST-depression ≥ 0.5 mm in two contiguous leads and/or T inversion > 1 mm in two contiguous leads with prominent R wave or R/S ratio > 1.

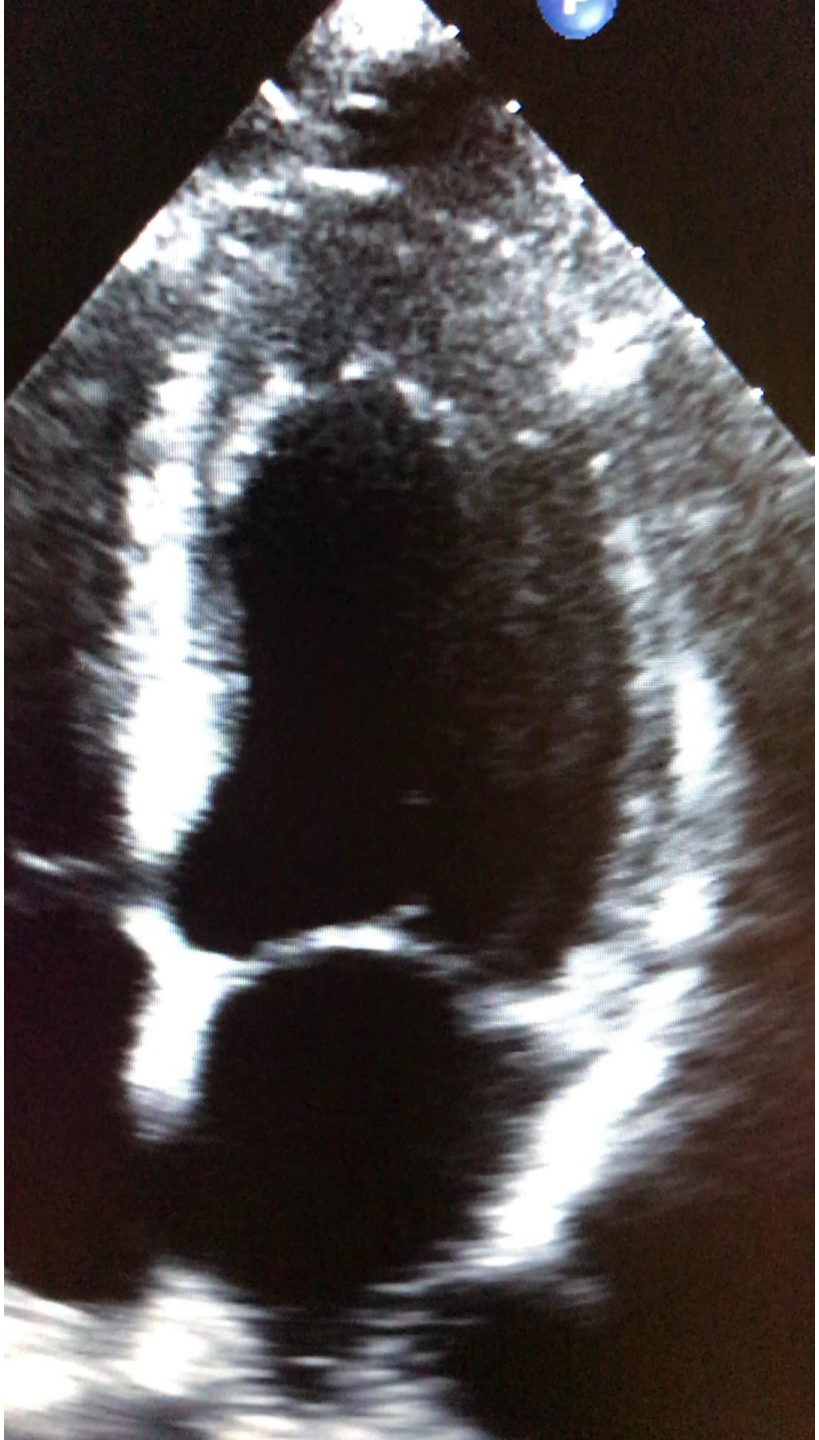
[a]When the magnitudes of J-point elevation in leads V2 and V3 are registered from a prior electrocardiogram, new J-point elevation ≥ 1 mm (as compared with the earlier electrocardiogram) should be considered an ischaemic response. For bundle branch block, see section below.



Hospital Course

- ECHO ordered to eval for new RWMA,
- Cancelled because an echo had been done 4 days prior to presentation!
- Rescheduled, showed normal LVEF and no new RWMA.
  - Echo tech tells me patient is acting weird comparing before to during admission
- Day three resident calls to ask when the patient should be seen by the cardiologist on discharge on ASA, beta blocker and statin.



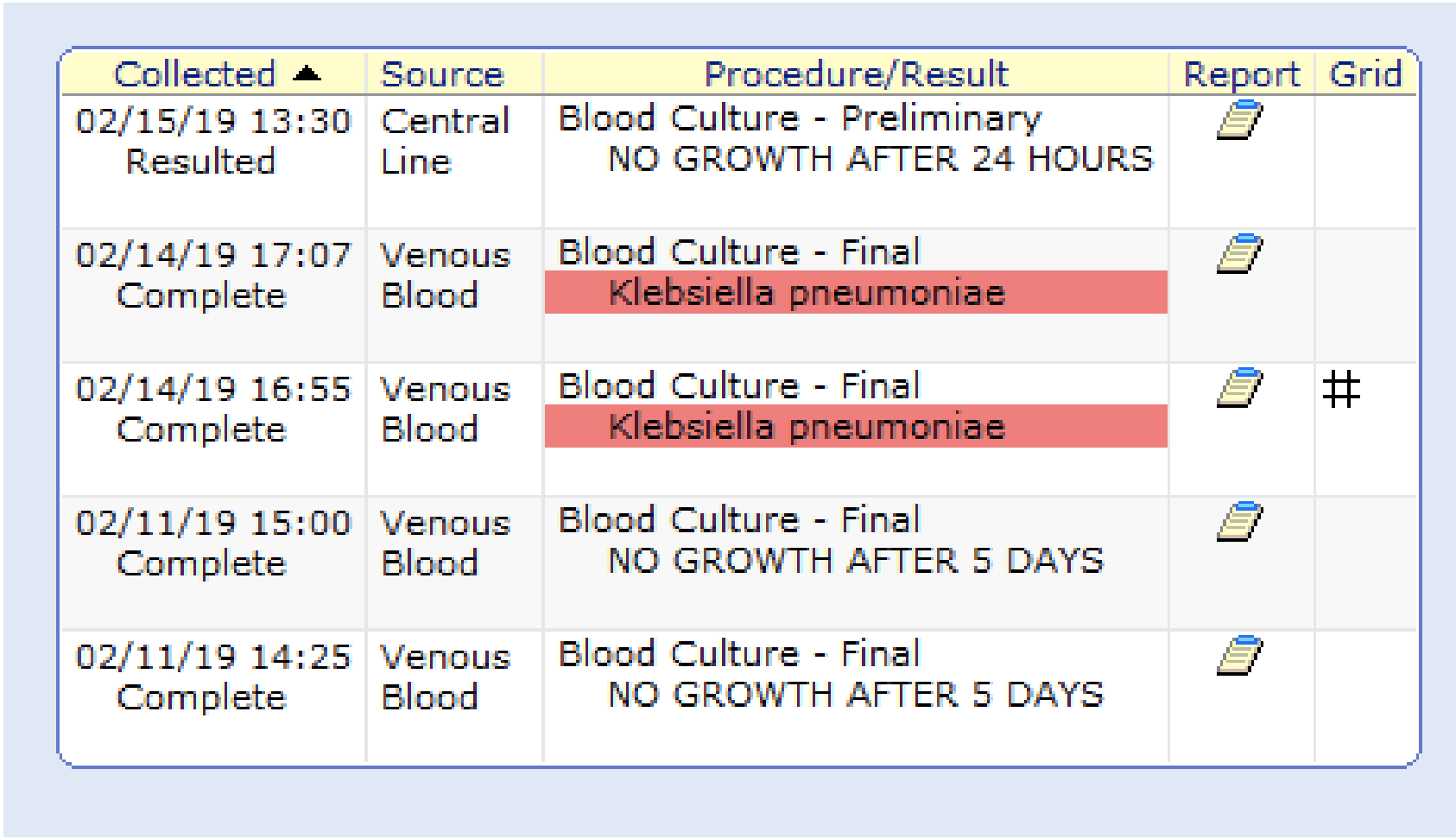


White Blood Count

Collected	Result	Units	Range	Group
02/16/2019 05:35	21.8 H	K/uL	4.5-11.0	
02/15/2019 06:50	18.9 H	K/uL	4.5-11.0	
02/14/2019 16:55	21.0 H	K/uL	4.5-11.0	
02/14/2019 06:45	19.0 H	K/uL	4.5-11.0	
02/13/2019 08:15	16.8 H Δ	K/uL	4.5-11.0	
02/12/2019 07:00	13.0 H	K/uL	4.5-11.0	
02/11/2019 15:00	15.8 H	K/uL	4.5-11.0	

Procalcitonin

Collected	Result	Units	Range	Group
02/15/2019 06:50	2.23 H*	NG/ML	0-0.09	
02/14/2019 16:55	2.11 H*	NG/ML	0-0.09	
02/11/2019 15:00	0.24 H	NG/ML	0-0.09	



Collected ▲	Source	Procedure/Result	Report	Grid
02/15/19 13:30 Resulted	Central Line	Blood Culture - Preliminary NO GROWTH AFTER 24 HOURS		
02/14/19 17:07 Complete	Venous Blood	Blood Culture - Final Klebsiella pneumoniae		
02/14/19 16:55 Complete	Venous Blood	Blood Culture - Final Klebsiella pneumoniae		#
02/11/19 15:00 Complete	Venous Blood	Blood Culture - Final NO GROWTH AFTER 5 DAYS		
02/11/19 14:25 Complete	Venous Blood	Blood Culture - Final NO GROWTH AFTER 5 DAYS		

- **Consultation**
Date of Service: 02/19/19
Requesting Physician:
Daniel J Suders, DO

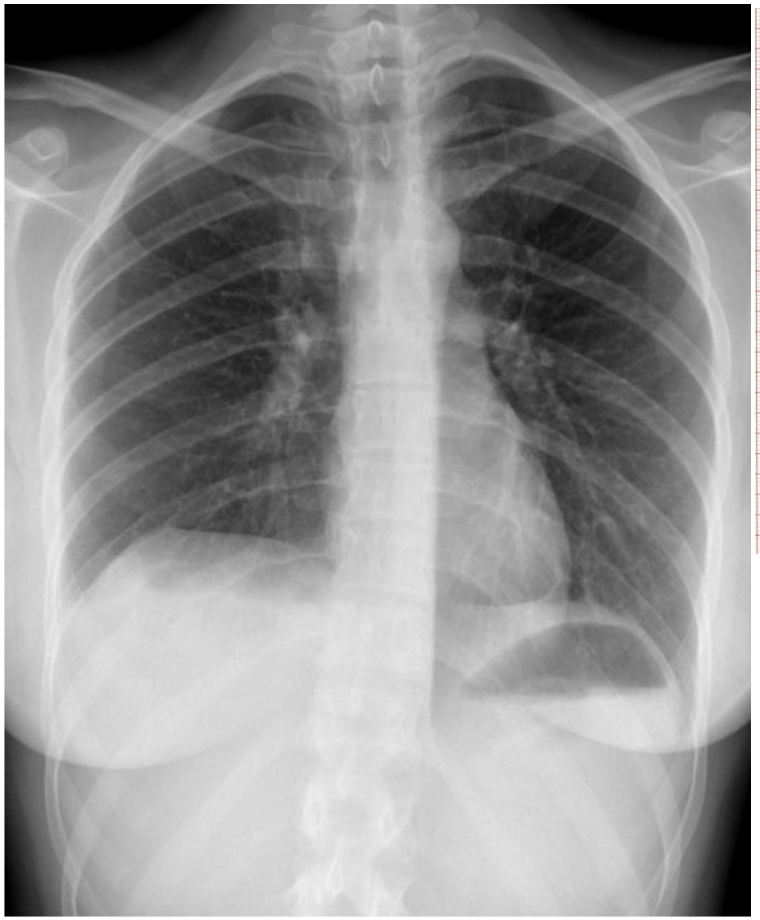
Primary Care Provider:
Dan Jones, DO

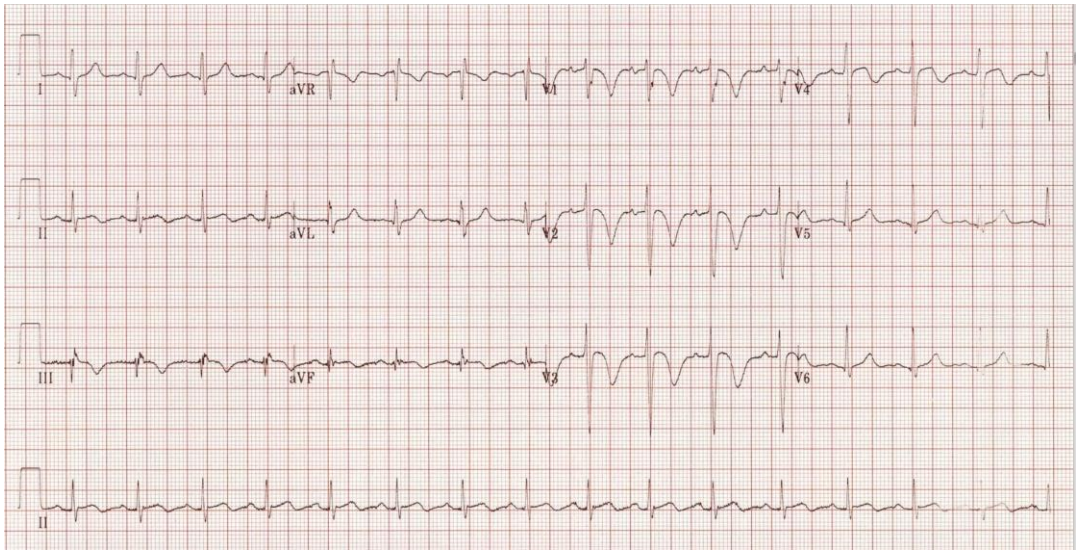
- **HPI**

This 62-year-old male who is dying from metastatic pancreatic cancer has an infected MediPort which needs to come out. If it has not taken out and he will die of infected MediPort instead of metastatic pancreatic cancer. He is on the schedule to remove the MediPort in the morning.

In another vein…..

- 47-yo hypertensive, diabetic female smoker
  - Presents with 12 h new onset chest pain
  - Associated dyspnea, palpitations

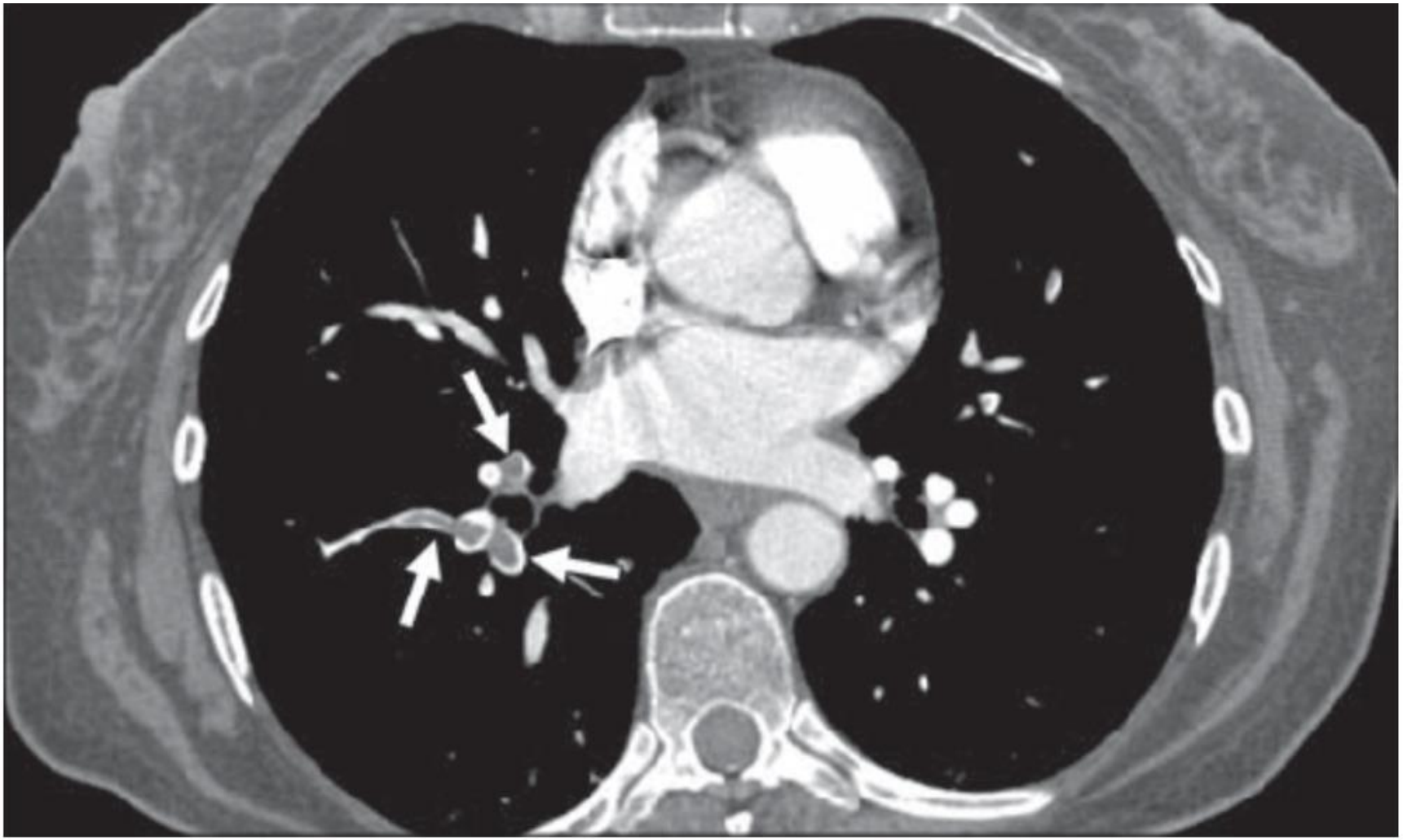




CBC: Hgb/Hct/WBC/Plts	13.1/37/12.1/375
INR/PTT	1.0/24
ABG/pCO2/pO2	7.48/27/55
Troponin	1.2

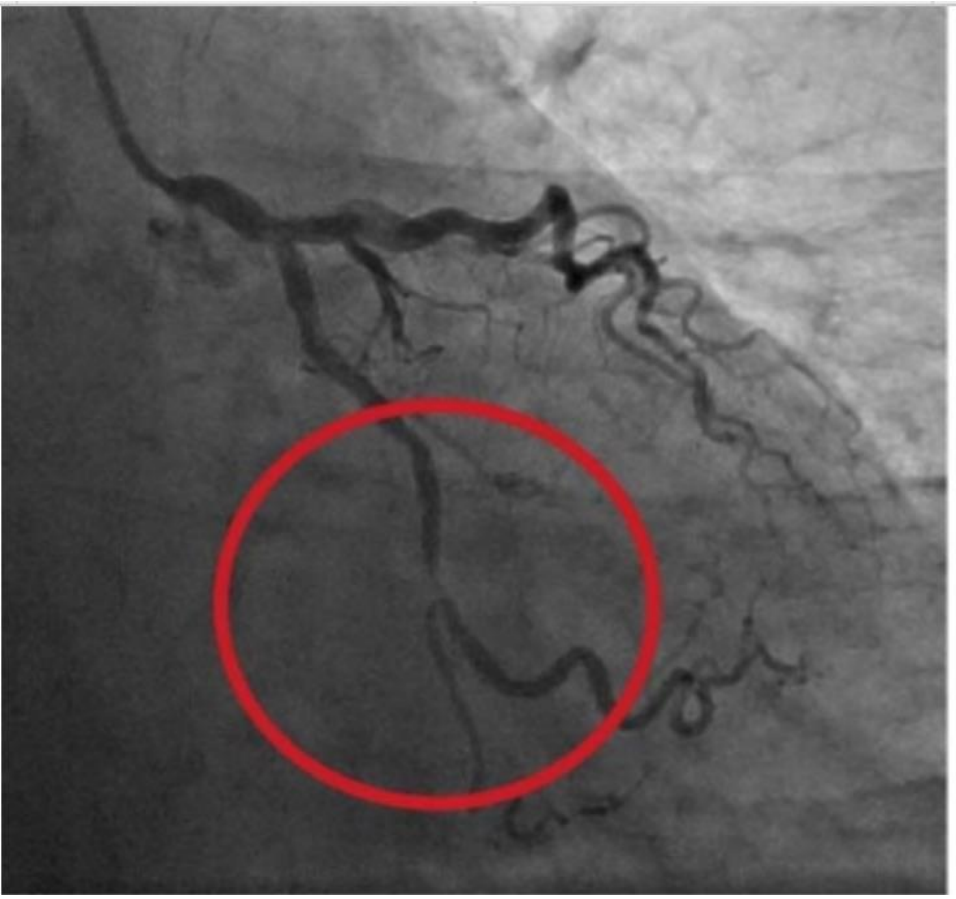
ED Course

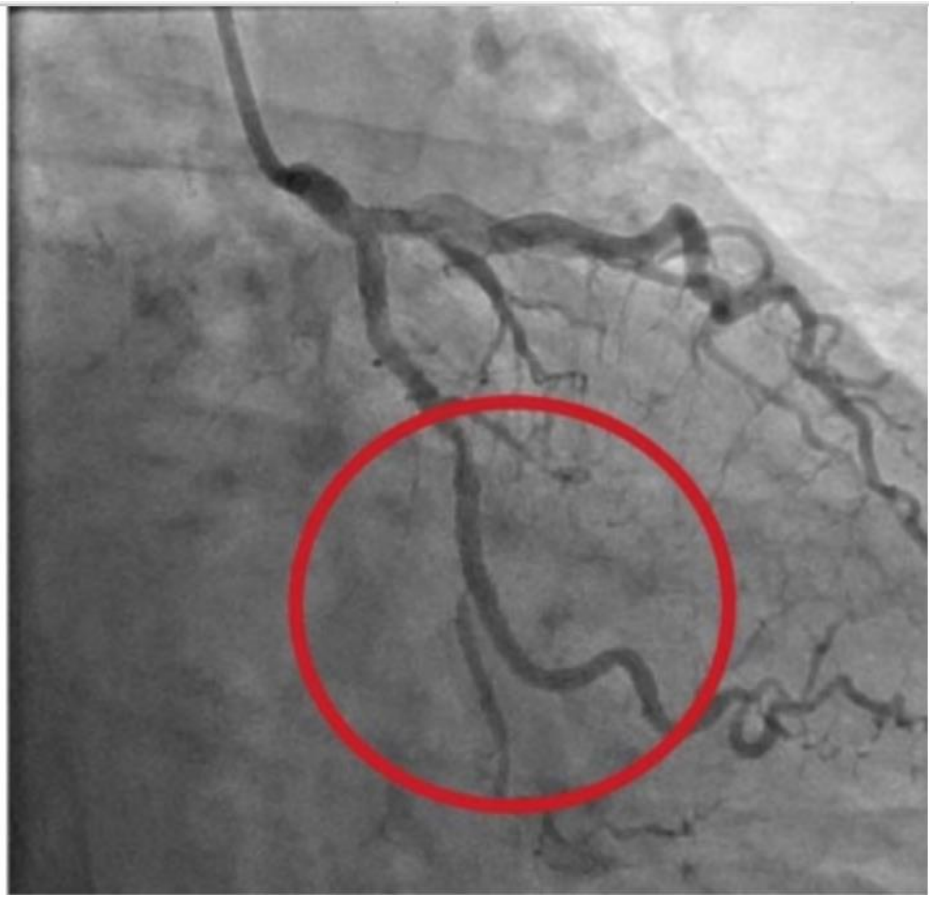
- Patient admitted with NSTEMI
- Interventional cardiology consulted
  - Busy in the cath lab, patient hydrated and kept NPO for 12:30 pm cath
- Patient requests another cardiologist:
  - Who is also busy in the cath lab;
  - PA examines patient and orders CTA chest



Hospital Course

- Treated with unfractionated heparin
- Venous Doppler study negative
- Dyspnea resolved in 3-days.
- Because she was told she had an MI, patient insisted upon cardiac catheterization.
  - (Cardiologist now available.)





Important Questions

- How do we define NSTEMI?
- How is myocardial injury different from myocardial infarction?
- What is the prognosis in elevated Troponin without infarction?
  - And why?

  How dangerous is it to label the patient with Dx: NSTEMI on admission based on one Troponin?

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Myocardial Infarction Redefined—A Consensus Document of The Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction

The Joint European Society of Cardiology/
American College of Cardiology Committee**

This document was developed by a consensus conference initiated by Kristian Thygesen, MD, and Joseph S. Alpert, MD, after formal approval by Lars Ryde´n, MD, President of the European Society of Cardiology (ESC), and Arthur Garson, MD, President of the American College of Cardiology (ACC).

> "Thus, the current diagnosis of acute MI is a clinical diagnosis based on patient symptoms, ECG changes and highly sensitive biochemical markers, as well as information gleaned from various imaging techniques."

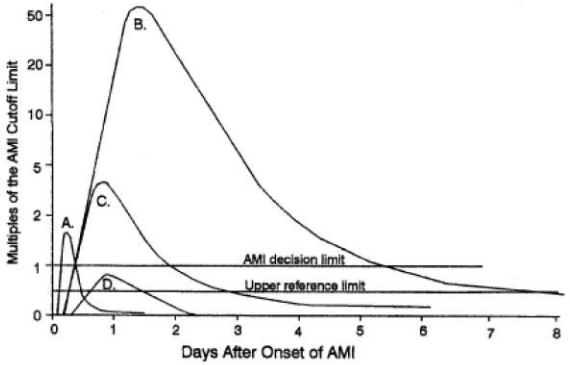

Figure 2. Timing of release of various biomarkers following acute, ischemic myocardial infarction.

Peak A, early release of myoglobin or CK-MB isoforms after AMI; **peak B,** cardiac troponin after AMI; **peak C,** CK-MB after AMI; **peak D,** cardiac troponin after unstable angina. Data are plotted on a relative scale, where 1.0 is set at the AMI cutoff concentration.

AMI = acute myocardial infarction; CAD = coronary artery disease; CK = creatine kinase.

Reproduced with permission, WU AH, et al. Clin Chem 1999;45: 1104–1121.

ARTICLE IN PRESS

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EXPERT CONSENSUS DOCUMENT

Fourth Universal Definition of Myocardial Infarction (2018)

Kristian Thygesen,* *Denmark*
Joseph S. Alpert,* *USA*
Allan S. Jaffe, *USA*
Bernard R. Chaitman, *USA*
Jeroen J. Bax, *The Netherlands*
David A. Morrow, *USA*
Harvey D. White,* *New Zealand*, the Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction

"Many doctors have not understood that elevated troponin levels in the blood are not sufficient to diagnosis a heart attack and this has created real problems," said **Kristian A. Thygesen, MD, DSC, FACC**, co-chair of the writing committee.

2. UNIVERSAL DEFINITIONS OF MYOCARDIAL INJURY AND MYOCARDIAL INFARCTION: SUMMARY

Universal definitions of myocardial injury and myocardial infarction

Criteria for myocardial injury

The term myocardial injury should be used when there is evidence of elevated cardiac troponin values (cTn) with at least one value above the 99th percentile upper reference limit (URL). The myocardial injury is considered acute if there is a rise and/or fall of cTn values.

Criteria for acute myocardial infarction (types 1, 2 and 3 MI)

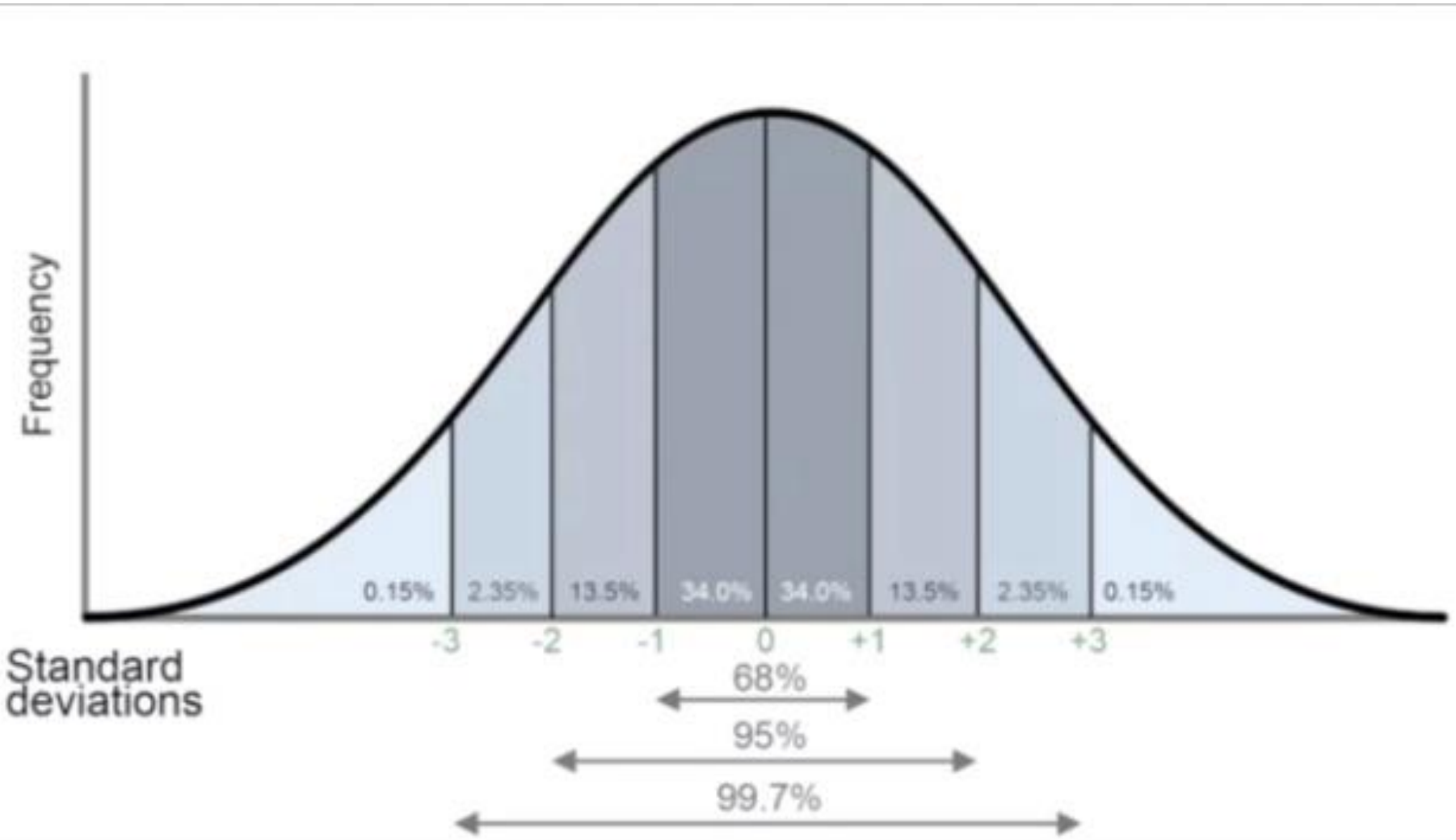
The term acute myocardial infarction should be used when there is acute myocardial injury with clinical evidence of acute myocardial ischaemia and with detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL and at least one of the following:

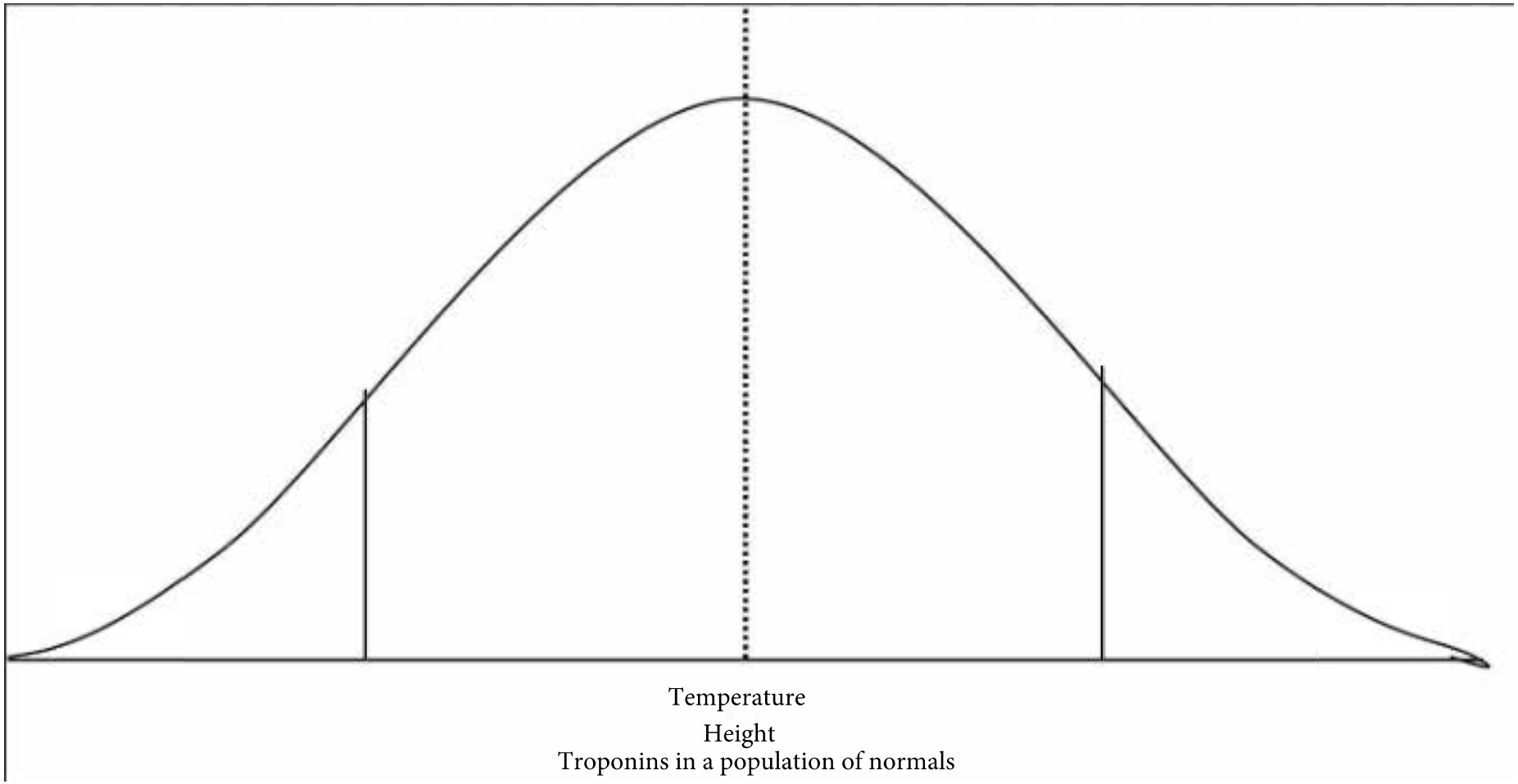
- Symptoms of myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology;
- Identification of a coronary thrombus by angiography or autopsy (not for type 2 or 3 MIs).

Post-mortem demonstration of acute athero-thrombosis in the artery supplying the infarcted myocardium meets criteria for *type 1 MI*.

Evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute athero-thrombosis meets criteria for *type 2 MI*.

Cardiac death in patients with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes before cTn values become available or abnormal meets criteria for *type 3 MI*.


Frequency
0.15%
2.35%
13.5%
34.0%
34.0%
13.5%
2.35%
0.15%
-3
-2
-1
0
+1
+2
+3
Standard deviations
68%
95%
99.7%

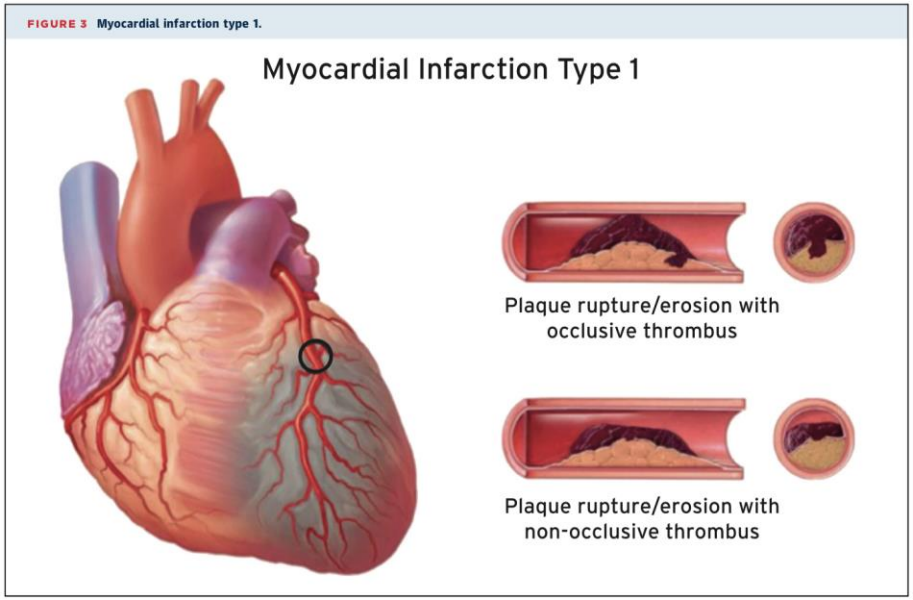


Temperature

Height

Troponins in a population of normals

FIGURE 3 Myocardial infarction type 1.

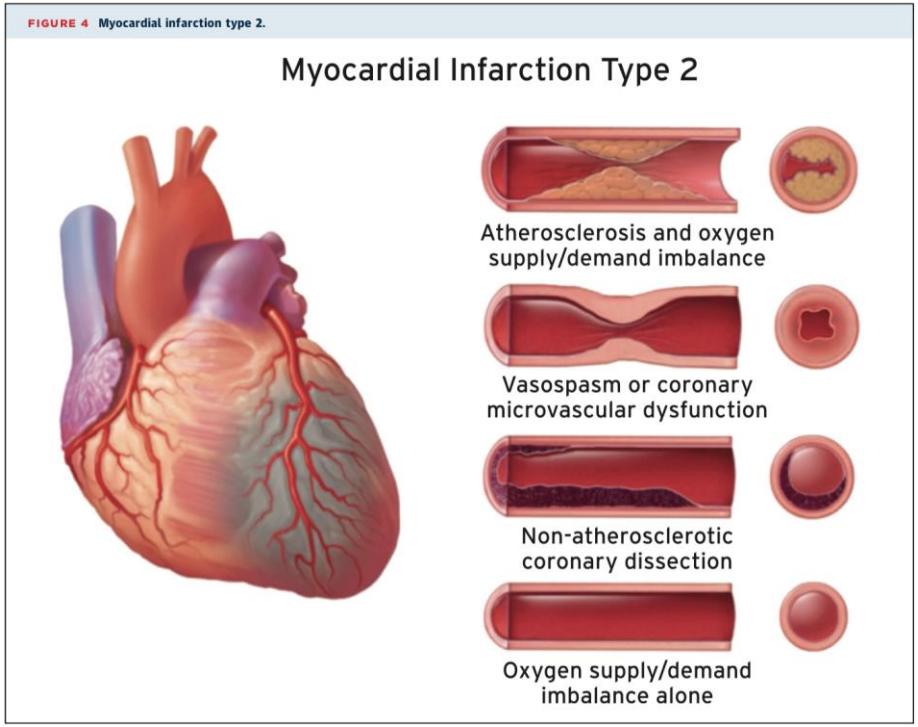


Criteria for type 1 MI

Detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL and with at least one of the following:

- Symptoms of acute myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology;
- Identification of a coronary thrombus by angiography including intracoronary imaging or by autopsy.[a]

FIGURE 4 Myocardial infarction type 2.



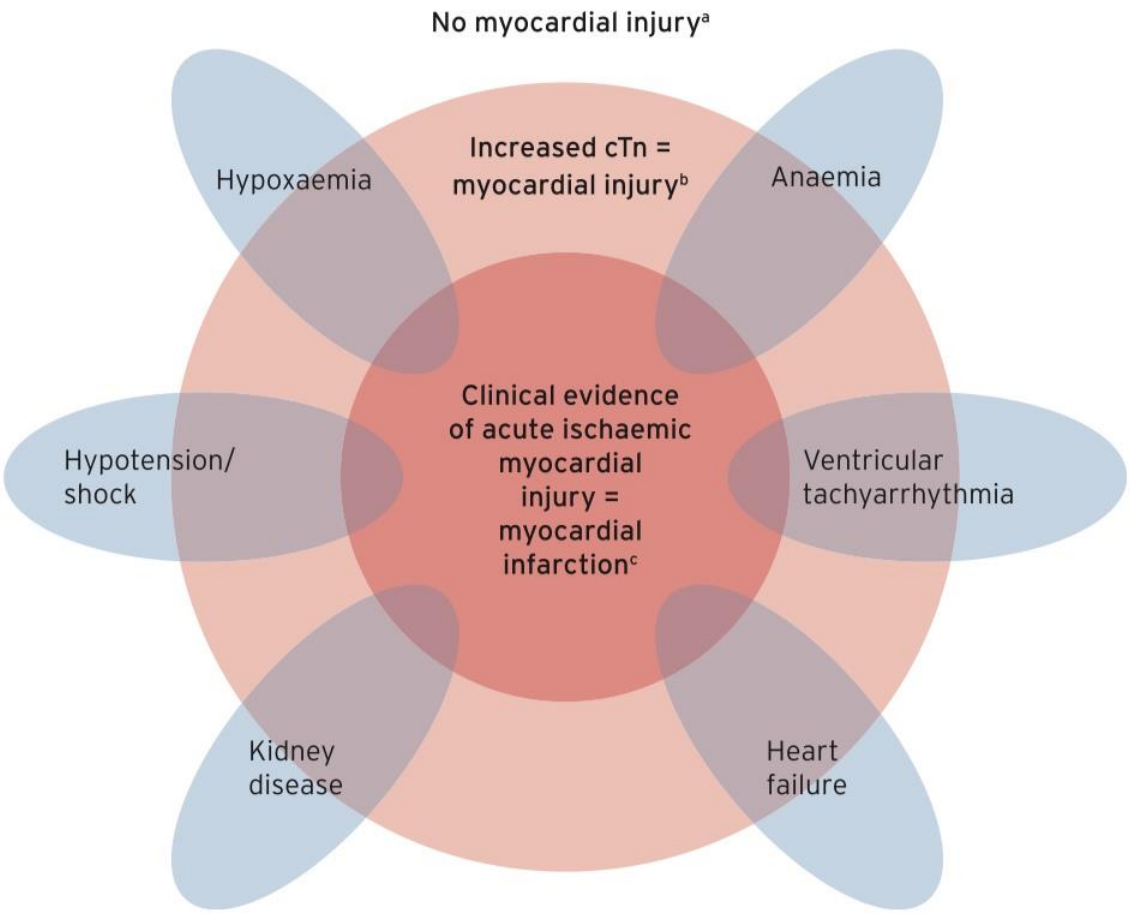
Criteria for type 2 MI

Detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL, and evidence of an imbalance between myocardial oxygen supply and demand unrelated to coronary thrombosis, requiring at least one of the following:

- Symptoms of acute myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology.

What's the difference between Type I and Type II MI, between Type II NSTEMI and myocardial injury?

FIGURE 2 Spectrum of myocardial injury, ranging from no injury to myocardial infarction. Various clinical entities may involve these myocardial categories, e.g. ventricular tachyarrhythmia, heart failure, kidney disease, hypotension/shock, hypoxaemia, and anaemia. cTn = cardiac troponin; URL = upper reference limit. [a]No myocardial injury = cTn values ≤ 99th percentile URL or not detectable. [b]Myocardial injury = cTn values > 99th percentile URL. [c]Myocardial infarction = clinical evidence of myocardial ischaemia and a rise and/or fall of cTn values > 99th percentile URL.



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.

Clinical criteria for MI

The clinical definition of MI denotes the presence of acute myocardial injury detected by abnormal cardiac biomarkers in the setting of evidence of acute myocardial ischaemia.

TABLE 1 Reasons for the elevation of cardiac troponin values because of myocardial injury

Myocardial injury related to acute myocardial ischaemia

Atherosclerotic plaque disruption with thrombosis.

Myocardial injury related to acute myocardial ischaemia because of oxygen supply/demand imbalance

Reduced myocardial perfusion, e.g.

- Coronary artery spasm, microvascular dysfunction
- Coronary embolism
- Coronary artery dissection
- Sustained bradyarrhythmia
- Hypotension or shock
- Respiratory failure
- Severe anaemia

Increased myocardial oxygen demand, e.g.

- Sustained tachyarrhythmia
- Severe hypertension with or without left ventricular hypertrophy

Other causes of myocardial injury

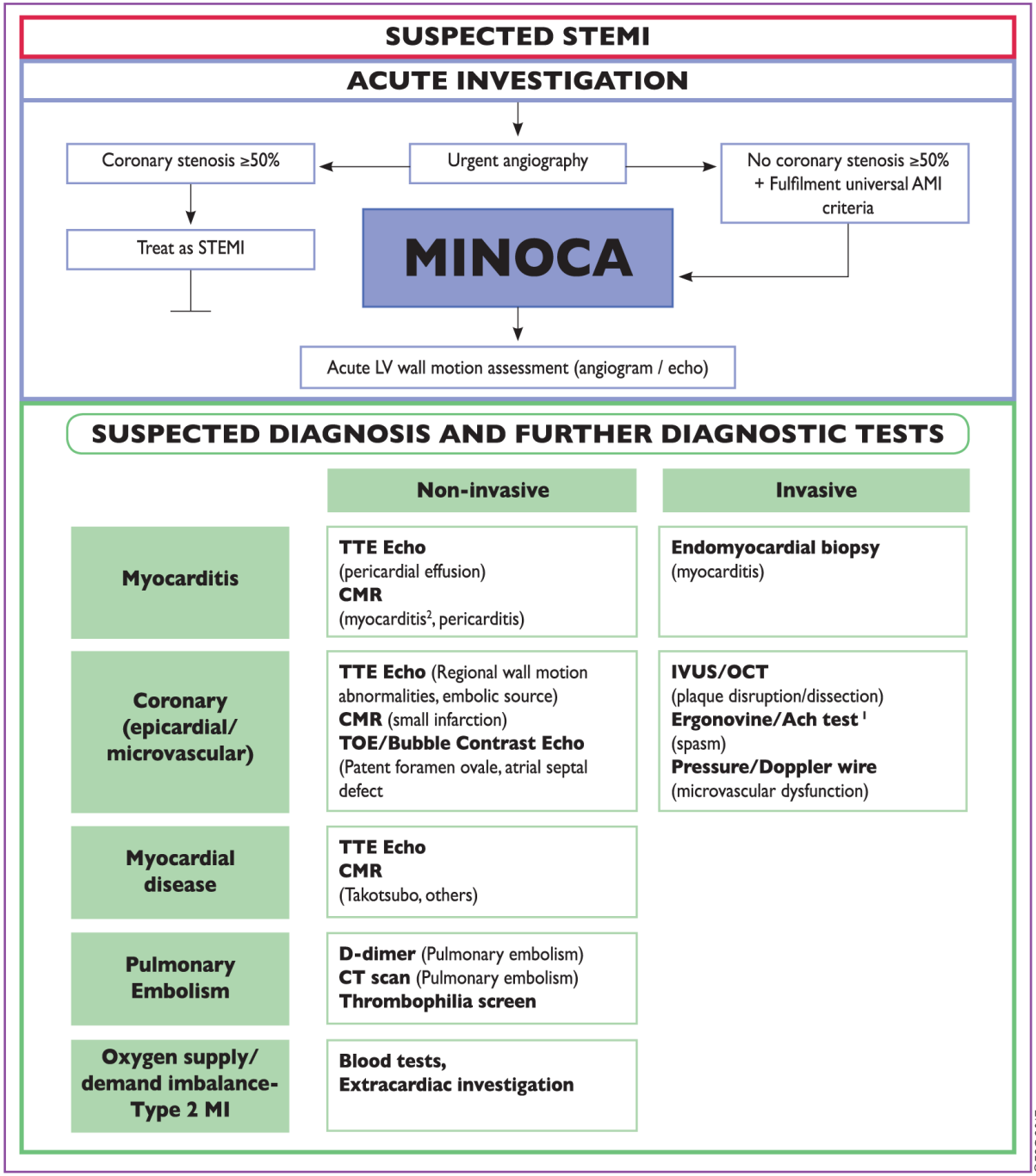
Cardiac conditions, e.g.

- Heart failure
- Myocarditis
- Cardiomyopathy (any type)
- Takotsubo syndrome
- Coronary revascularization procedure
- Cardiac procedure other than revascularization
- Catheter ablation
- Defibrillator shocks
- Cardiac contusion

Systemic conditions, e.g.

- Sepsis, infectious disease
- Chronic kidney disease
- Stroke, subarachnoid haemorrhage
- Pulmonary embolism, pulmonary hypertension
- Infiltrative diseases, e.g. amyloidosis, sarcoidosis
- Chemotherapeutic agents
- Critically ill patients
- Strenuous exercise

For a more comprehensive listing, see (39–41)


SUSPECTED STEMI
ACUTE INVESTIGATION
Coronary stenosis ≥50%
Urgent angiography
No coronary stenosis ≥50% + Fulfilment universal AMI criteria
Treat as STEMI
MINOCA
Acute LV wall motion assessment (angiogram / echo)
SUSPECTED DIAGNOSIS AND FURTHER DIAGNOSTIC TESTS
Non-invasive
Invasive
Myocarditis
TTE Echo (pericardial effusion)
CMR (myocarditis[2], pericarditis)
Endomyocardial biopsy (myocarditis)
Coronary (epicardial/ microvascular)
TTE Echo (Regional wall motion abnormalities, embolic source)
CMR (small infarction)
TOE/Bubble Contrast Echo (Patent foramen ovale, atrial septal defect
IVUS/OCT (plaque disruption/dissection)
Ergonovine/Ach test [1] (spasm)
Pressure/Doppler wire (microvascular dysfunction)
Myocardial disease
TTE Echo
CMR
(Takotsubo, others)
Pulmonary Embolism
D-dimer (Pulmonary embolism)
CT scan (Pulmonary embolism)
Thrombophilia screen
Oxygen supply/ demand imbalance- Type 2 MI
Blood tests,
Extracardiac investigation
©ESC 2017

Coding for Myocardial Injury

Codes

- S26 Injury of heart
  - S26.0 Injury of heart with hemopericardium
    - S26.00 Unspecified injury of heart with hemopericardium
      - S26.00XA ..... initial encounter
      - S26.00XD ..... subsequent encounter
      - S26.00XS ..... sequela
    - S26.01 Contusion of heart with hemopericardium
      - S26.01XA ..... initial encounter
      - S26.01XD ..... subsequent encounter
      - S26.01XS ..... sequela
    - S26.02 Laceration of heart with hemopericardium
      - S26.020 Mild laceration of heart with hemopericardium
        - S26.020A ..... initial encounter
        - S26.020D ..... subsequent encounter
        - S26.020S ..... sequela
      - S26.021 Moderate laceration of heart with hemopericardium
        - S26.021A ..... initial encounter
        - S26.021D ..... subsequent encounter
        - S26.021S ..... sequela
      - S26.022 Major laceration of heart with hemopericardium
        - S26.022A ..... initial encounter
        - S26.022D ..... subsequent encounter
        - S26.022S ..... sequela
    - S26.09 Other injury of heart with hemopericardium
      - S26.09XA ..... initial encounter
      - S26.09XD ..... subsequent encounter
      - S26.09XS ..... sequela
  - S26.1 Injury of heart without hemopericardium
    - S26.10 Unspecified injury of heart without hemopericardium
      - S26.10XA ..... initial encounter
      - S26.10XD ..... subsequent encounter
      - S26.10XS ..... sequela
    - S26.11 Contusion of heart without hemopericardium
      - S26.11XA ..... initial encounter
      - S26.11XD ..... subsequent encounter
      - S26.11XS ..... sequela
    - S26.12 Laceration of heart without hemopericardium
      - S26.12XA ..... initial encounter
      - S26.12XD ..... subsequent encounter
      - S26.12XS ..... sequela
    - S26.19 Other injury of heart without hemopericardium
      - S26.19XA ..... initial encounter
      - S26.19XD ..... subsequent encounter
      - S26.19XS ..... sequela
  - S26.9 Injury of heart, unspecified with or without hemopericardium
    - S26.90 Unspecified injury of heart, unspecified with or without hemopericardium
      - S26.90XA ..... initial encounter
      - S26.90XD ..... subsequent encounter
      - S26.90XS ..... sequela
    - S26.91 Contusion of heart, unspecified with or without hemopericardium
      - S26.91XA ..... initial encounter
      - S26.91XD ..... subsequent encounter
      - S26.91XS ..... sequela
    - S26.92 Laceration of heart, unspecified with or without hemopericardium
      - S26.92XA ..... initial encounter
      - S26.92XD ..... subsequent encounter
      - S26.92XS ..... sequela
    - S26.99 Other injury of heart, unspecified with or without hemopericardium
      - S26.99XA ..... initial encounter
      - S26.99XD ..... subsequent encounter
      - S26.99XS ..... sequela

- I21.4 non-ST-elevated MI
- I74.8 elevated other serum enzyme

Brief Report

ONLINE FIRST FREE

March 17, 2019

Misclassification of Myocardial Injury as Myocardial Infarction

Implications for Assessing Outcomes in Value-Based Programs

Cian McCarthy, MB, BCh, BAO[1]; Sean Murphy, MB, BCh, BAO[1]; Joshua A. Cohen, MD[1]; et al

» Author Affiliations | Article Information

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From: **Misclassification of Myocardial Injury as Myocardial Infarction: Implications for Assessing Outcomes in Value-Based Programs**

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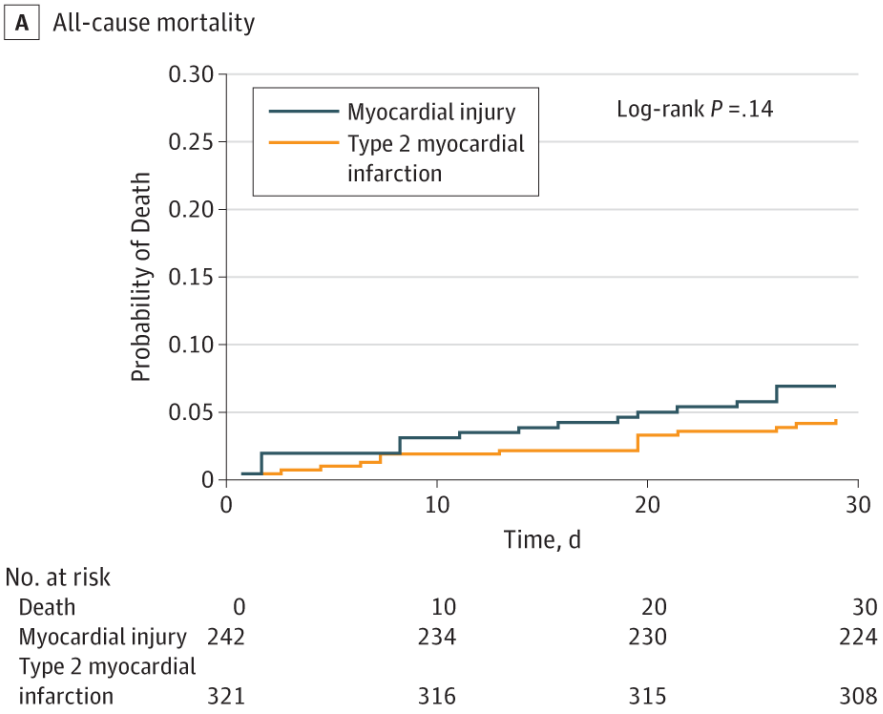


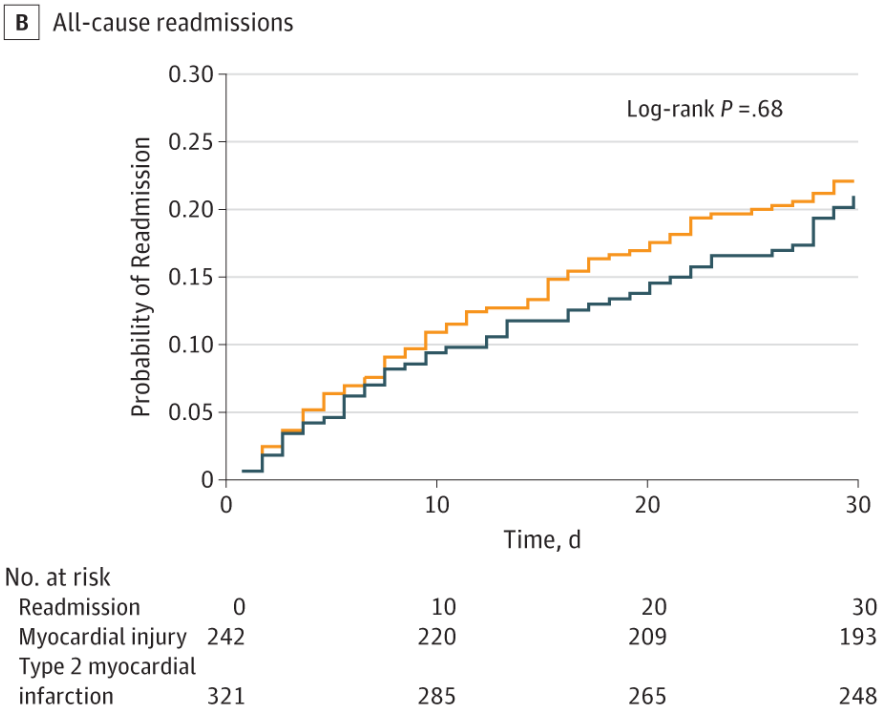


Figure Legend:

Kaplan-Meier Survival CurvesThe curves depict time to mortality (A) and readmission (B) over 30 days among patients with nonischemic myocardial injury and type 2 myocardial infarction who were discharged alive (n = 563) and were compared using log-rank tests.

Key Points

Question To what extent are patients with myocardial injury being misclassified as having type 2 myocardial infarction (T2MI) and what are the possible implications for 30-day readmission and mortality rates?

Findings This study used the new *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision* code to identify 633 patients who were coded as having T2MI. After strict adjudication, only 57% of patients met the criteria for T2MI and 42% had myocardial injury; both groups had similar in-hospital mortality rates and 30-day mortality and readmission rates.

Meaning A substantial proportion of patients who are coded as having T2MI actually have myocardial injury, which likely has implications for hospital reimbursement under current policy programs.

FIGURE 6 A model for interpreting myocardial injury. Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease. MI = myocardial infarction; URL = upper reference limit. [a]Stable denotes ≤ 20% variation of troponin values in the appropriate clinical context. [b]Ischaemia denotes signs and/or symptoms of clinical myocardial ischaemia.

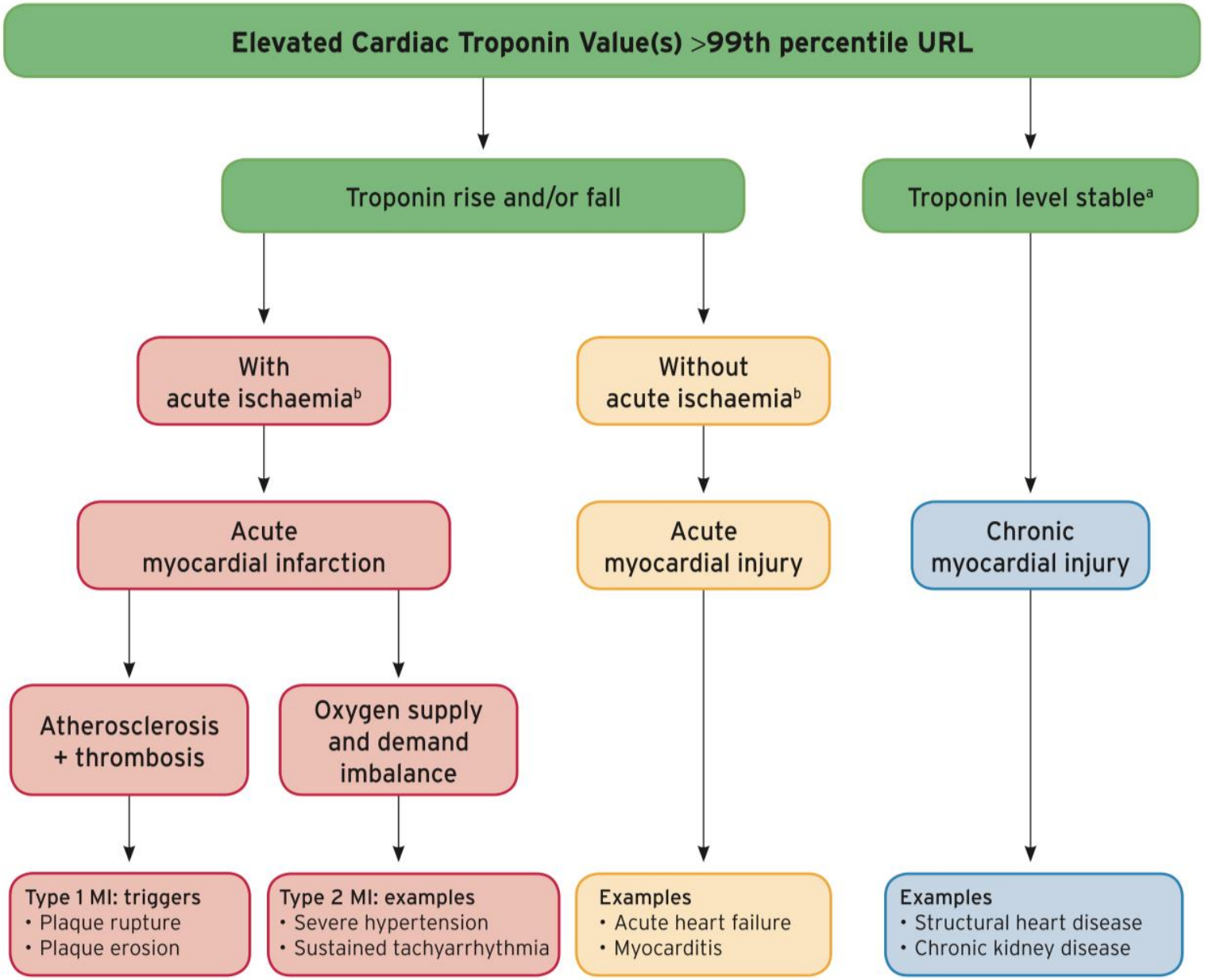
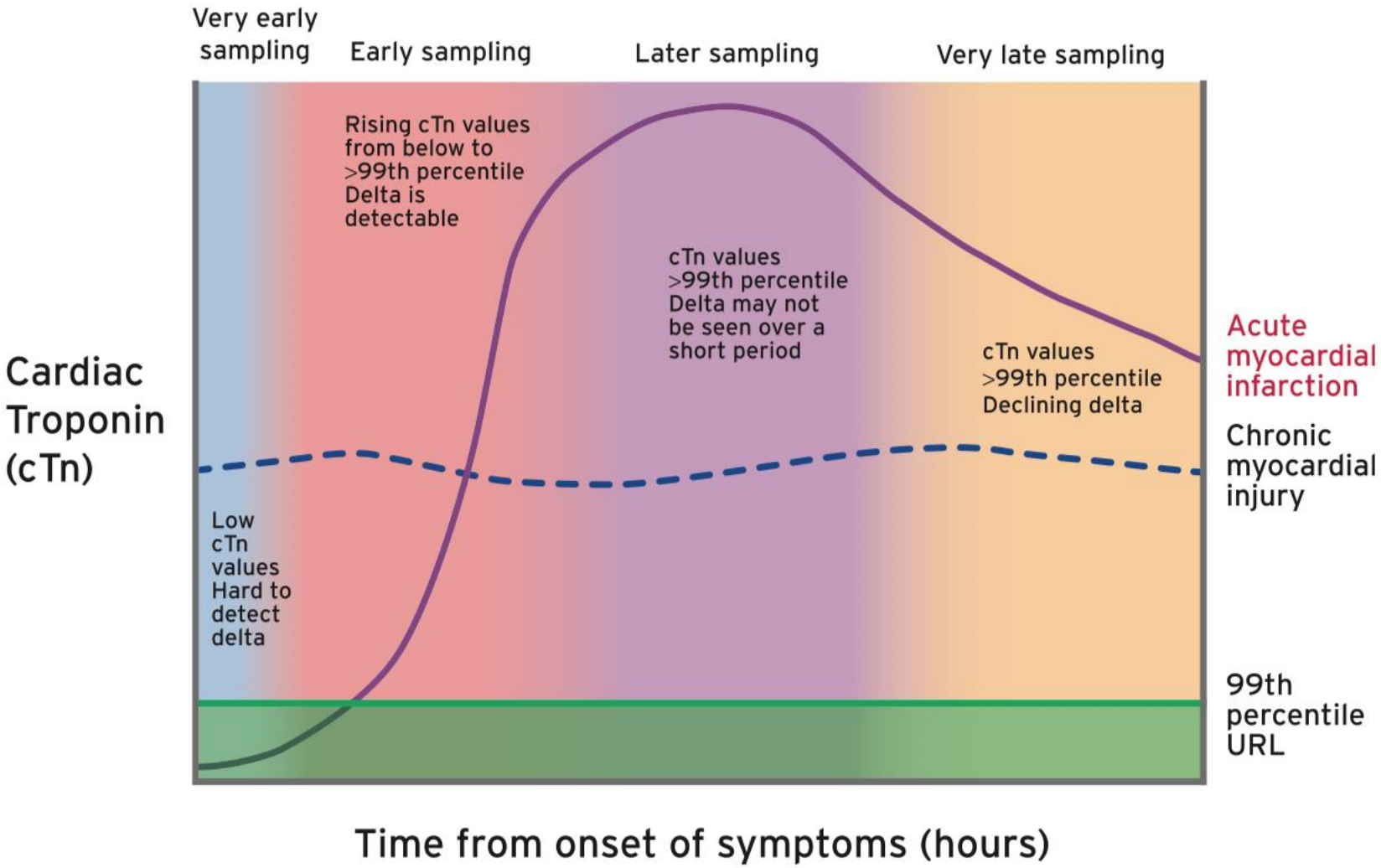


FIGURE 7 Illustration of early cardiac troponin kinetics in patients after acute myocardial injury including acute myocardial infarction. The timing of biomarker release into the circulation is dependent on blood flow and how soon after the onset of symptoms samples are obtained. Thus, the ability to consider small changes as diagnostic can be problematic. In addition, many comorbidities increase cTn values and, in particular, hs-cTn values, so that elevations can be present at baseline even in those with myocardial infarction who present early after the onset of symptoms. Changes in cTn values or deltas can be used to define acute compared with chronic events, and the ability to detect these is indicated in the figure. Increased cTn values can often be detected for days after an acute event. cTn = cardiac troponin; URL = upper reference limit.



JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY VOL. 73, NO. 1, 2019

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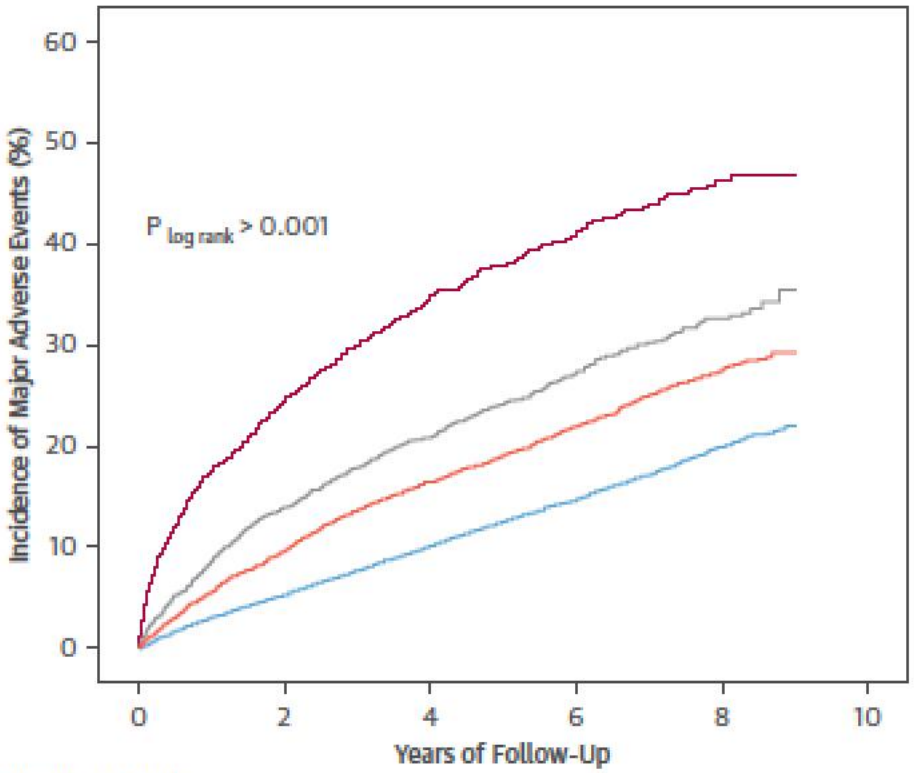
ORIGINAL INVESTIGATIONS

Cardiac Troponin Elevation in Patients Without a Specific Diagnosis

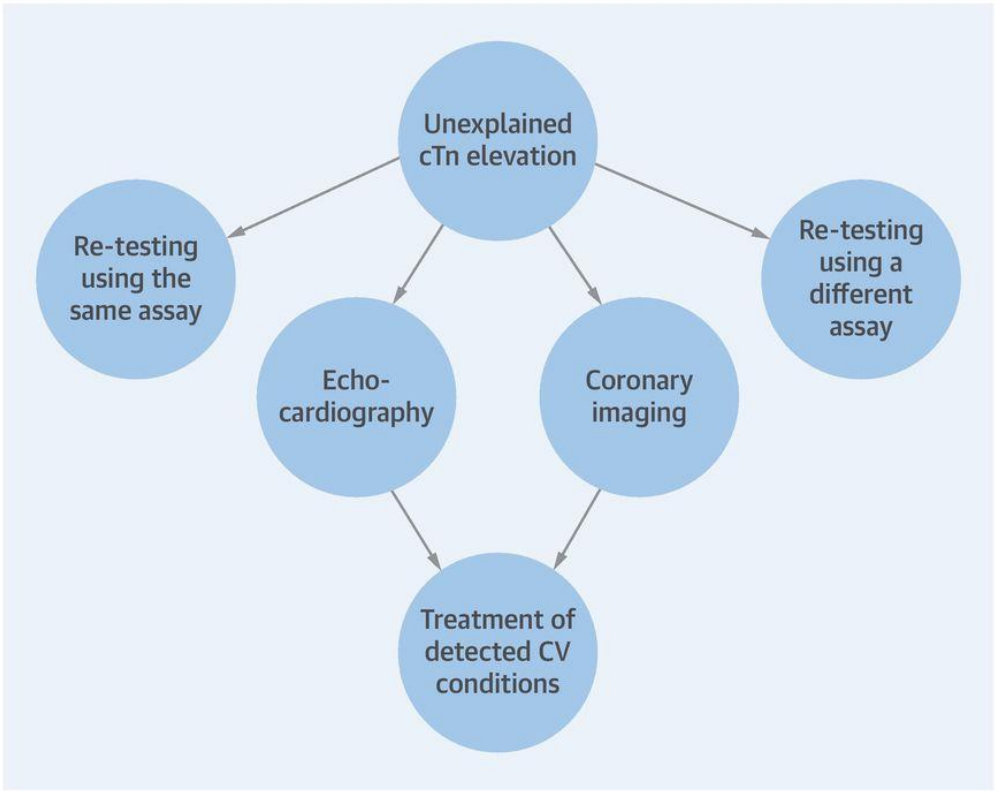
Kai M. Eggers, MD, PhD,[a] Tomas Jernberg, MD, PhD,[b] Bertil Lindahl, MD, PhD[a]

FIGURE 2 **Unadjusted Kaplan-Meier Estimates of the Cumulative Incidence of Major Adverse Events**



Unadjusted Kaplan-Meier analysis demonstrated that the rates of major adverse events increased across strata with higher cardiac troponin (cTn) levels. The event curves diverged early and constantly during the follow-up period (median 4.9 years). The **blue line** represents patients with cTn levels below the assay-specific 99th percentile. **Orange, gray, and red** lines represent patients with cTn levels in the assay-specific tertiles 1, 2, and 3, respectively.

CENTRAL ILLUSTRATION: Work-Up of Patients With Unexplained Cardiac Troponin Elevation



Eggers, K.M. et al. J Am Coll Cardiol. 2019;73(1):1-9.

Kai M. Eggers et al. JACC 2019;73:1-9

JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY VOL. 73, NO. 1, 2019

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EDITORIAL COMMENT

Trivializing an Elevated Troponin

Adding Insult to Injury?*

James L. Januzzi, JR, MD,[a,b] Cian P. McCarthy, MB, BCH, BAO[c]

"Myocardial injury is not a benign condition and should not be trivialized with awkward and nonsensical monikers such as "troponinemia"; it is injury, and should be referred to as such. Information on definitive management for these high-risk patients will depend on outcome of much-needed clinical trials currently planned or ongoing."

Classification of Chest Pain

- Typical anginal chest pain:
  - Onset with exertion or emotional distress;
  - Of a typical character, duration and location;
  - Relieved with rest or SL NTG.
- Atypical chest pain:
  - 2 of the above
- Non-anginal chest pain:
  - 0-1 of the above.

Grading of Angina Pectoris according to the Canadian Cardiovascular Society (CCS)[107]

CLASS	DESCRIPTION
I	*Ordinary physical activity does not cause angina* such as walking or climbing stairs. Angina occurs with strenuous, rapid or prolonged exertion at work or recreation.
II	*Slight limitation of ordinary activity.* Angina occurs on walking or climbing stairs rapidly; walking uphill; walking or climbing stairs after meals; in cold, in wind, or under emotional stress; or only during the few hours after awakening. Angina occurs on walking > 2 blocks on the level and climbing > 1 flight of ordinary stairs at a normal pace and under normal conditions.
III	*Marked limitation of ordinary physical activity.* Angina occurs on walking 1 to 2 blocks on the level and climbing 1 flight of stairs under normal conditions and at a normal pace.
IV	*Inability to carry on any physical activity without discomfort - angina symptoms may be present at rest.*

Grocery Store Index

I	II	III	IV
COSTCO WHOLESALE	GIANT EAGLE	ACME	ALDI
Sam's Club	Publix	GIANT	DOLLAR GENERAL
	Walmart	Kroger	FOOD LION
		piggly wiggly	Riesbeck's Food Markets
		Wegmans	TRADER JOE'S
			weis


Walmart
24 hours
Pickup
Walmart.com/grocery
Order. Schedule. Pick up.
Pick up here.
Grocery pick up only at this location.

Three Principal Presentations of Unstable Angina

- Resting angina: resting onset of > 20 minutes.
- New onset: of at least CCS III severity.
- Crescendo angina:
  - Distinctly more frequent, longer duration or lower threshold;
  - Increasing by at least one CCS class;
  - Of at least CCS III severity.

2002 AHA/ACC Unstable angina/NSTEMI guideline update

Analysis of Probability as an Aid in the Clinical Diagnosis of Coronary-Artery Disease

George A. Diamond, M.D., and James S. Forrester, M.D.

Appendix Table 4. **Pretest Likelihood of Coronary Artery Disease in Symptomatic Patients According to Age and Sex***

Age, *y*	Nonanginal Chest Pain, %		Atypical Angina, %		Typical Angina, %	
	Men	Women	Men	Women	Men	Women
30–39	4	2	34	12	76	26
40–49	13	3	51	22	87	55
50–59	20	7	65	31	93	73
60–69	27	14	72	51	94	86

* Combined data from Diamond and Forrester's study (13) and Coronary Artery Surgery Study (14). Each value represents the percentage with significant coronary artery disease on catheterization. Adapted from Diamond and Forrester (13) with permission.

June 14, 1979

N Engl J Med 1979; 300:1350-1358

THE AMERICAN JOURNAL of MEDICINE®

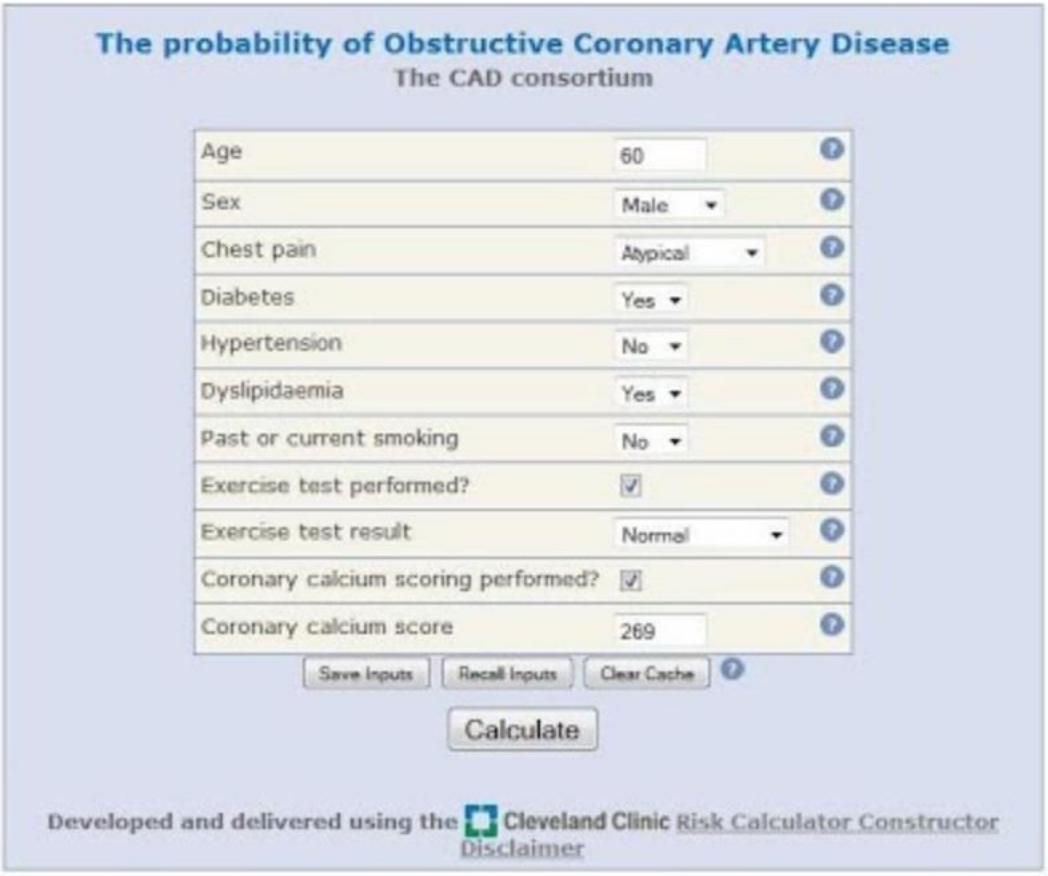
Official Journal of the Alliance for Academic Internal Medicine

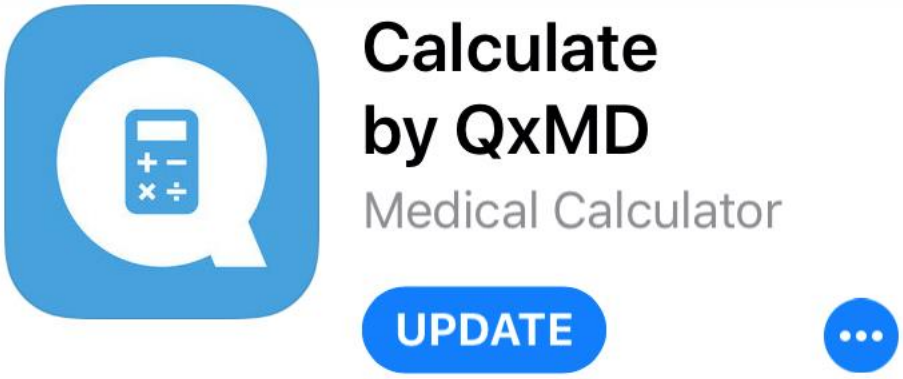
Appendix Table 5. **Comparing Pretest Likelihoods of Coronary Artery Disease in Low-Risk Symptomatic Patients and High-Risk Symptomatic Patients***

Age, *y*	Nonanginal Chest Pain, %		Atypical Angina, %		Typical Angina, %	
	Men	Women	Men	Women	Men	Women
35	3–35	1–19	8–59	2–39	30–88	10–78
45	9–47	2–22	21–70	5–43	51–92	20–79
55	23–59	4–21	45–79	10–47	80–95	38–82
65	49–69	9–29	71–86	20–51	93–97	56–84

* From the Duke Database. Each value represents the percentage with significant coronary artery disease. The first is the percentage for a low-risk, mid-decade patient without diabetes, smoking, or hyperlipidemia. The second is that of the same-age patient with diabetes, smoking, and hyperlipidemia. Both high- and low-risk patients have normal results on resting electrocardiography. If ST-T wave changes or Q waves had been present, the likelihood of coronary artery disease would be higher in each entry of the table. Reprinted from Pryor et al (16) with permission.

November 1983 Volume 75, Issue 5, Pages 771–780





ORIGINAL RESEARCH ARTICLE

European Society of Cardiology–Recommended Coronary Artery Disease Consortium Pretest Probability Scores More Accurately Predict Obstructive Coronary Disease and Cardiovascular Events Than the Diamond and Forrester Score

The Partners Registry

BACKGROUND: The most appropriate score for evaluating the pretest probability of obstructive coronary artery disease (CAD) is unknown. We sought to compare the Diamond-Forrester (DF) score with the 2 CAD consortium scores recently recommended by the European Society of Cardiology.

METHODS: We included 2274 consecutive patients (age, 56±13 years; 57% male) without prior CAD referred for coronary computed tomographic angiography. Computed tomographic angiography findings were used to determine the presence or absence of obstructive CAD (≥50% stenosis). We compared the DF score with the 2 CAD consortium scores with respect to their ability to predict obstructive CAD and the potential implications of these scores on the downstream use of testing for CAD, as recommended by current guidelines.

RESULTS: The DF score did not satisfactorily fit the data and resulted in a significant overestimation of the prevalence of obstructive CAD ($P<0.001$); the CAD consortium basic score had no significant lack of fitness; and the CAD consortium clinical provided adequate goodness of fit ($P=0.39$). The DF score had a lower discrimination for obstructive CAD, with an area under the receiver-operating characteristics curve of 0.713 versus 0.752 and 0.791 for the CAD consortium models ($P<0.001$ for both). Consequently, the use of the DF score was associated with fewer individuals being categorized as requiring no additional testing (8.3%) compared with the CAD consortium models (24.6% and 30.0%; $P<0.001$). The proportion of individuals with a high pretest probability was 18% with the DF and only 1.1% with the CAD consortium scores ($P<0.001$)

CONCLUSIONS: Among contemporary patients referred for noninvasive testing, the DF risk score overestimates the risk of obstructive CAD. On the other hand, the CAD consortium scores offered improved goodness of fit and discrimination; thus, their use could decrease the need for noninvasive or invasive testing while increasing the yield of such tests.

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*Drs Bittencourt and Hulten contributed equally to this work.

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Sources of Funding, see page 209

Key Words: chest pain ■ coronary artery disease ■ risk assessment ■ prognosis

The HEART Score for Chest Pain Patients in the ED

History	• Highly Suspicious • Moderately Suspicious • Slightly or Non-Suspicious	• 2 points • 1 point • 0 points
ECG	• Significant ST-Depression • Nonspecific Repolarization • Normal	• 2 points • 1 point • 0 points
Age	• ≥ 65 years • > 45 - < 65 years • ≤ 45 years	• 2 points • 1 point • 0 points
Risk Factors	• ≥ 3 Risk Factors or History of CAD • 1 or 2 Risk Factors • No Risk Factors	• 2 points • 1 point • 0 points
Troponin	• ≥ 3 x Normal Limit • > 1 - < 3 x Normal Limit • ≤ Normal Limit	• 2 points • 1 point • 0 points

Risk Factors: DM, current or recent (<one month) smoker, HTN, HLP, family history of CAD, & obesity

Score 0 – 3: 2.5% MACE over next 6 weeks → Discharge Home
Score 4 – 6: 20.3% MACE over next 6 weeks → Admit for Clinical Observation
Score 7 – 10: 72.7% MACE over next 6 weeks → Early Invasive Strategies

Clinical characteristics	Score
A) Age (please circle single best answer)	
18-45	+2
46-50	+4
51-55	+6
56-60	+8
61-65	+10
66-70	+12
71-75	+14
76-80	+16
81-85	+18
86+	+20
B) Male sex (please circle if true)	+6
C) This component is to be used only for ages 18-50 with either	
Known CAD (previous AMI, CABG, or PCI in men <55 years or women <65 year)	OR
≥3 risk factors present (family history premature CAD, diabetes, hypertension, dyslipidemia, and current smoker)	+4
D) Signs and symptoms (circle each that present)	
Diaphoresis (in association with pain)	+3
Pain occurs or worsened with inspiration	−4
Pain radiates to the arm or shoulder	+5
Pain reproduced by palpation	−6
EDACS total (please add score of all circled figures)	[ ]

EDACS: <16 (Low Risk), 16-21 (Intermediate Risk), ≥21 (High Risk). EDACS: Emergency Department Assessment of Chest Pain Score, CAD: Coronary Artery Disease, CABG: Coronary Artery Bypass Graft, PCI: Percutaneous Coronary Intervention, AMI: Acute Myocardial Infarction

TIMI RISK SCORE for UA/NSTEMI

HISTORICAL	POINTS
Age ≥ 65	1
≥ 3 CAD risk factors (FHx, HTN, ↑ chol, DM, active smoker)	1
Known CAD (stenosis ≥ 50%)	1
ASA use in past 7 days	1
PRESENTATION	
Recent (≤24H) severe angina	1
↑ cardiac markers	1
ST deviation ≥ 0.5 mm	1

RISK SCORE = Total Points (0 - 7)

RISK OF CARDIAC EVENTS (%) BY 14 DAYS IN TIMI 11B*

RISK SCORE	DEATH OR MI	DEATH, MI OR URGENT REVASC
0/1	3	5
2	3	8
3	5	13
4	7	20
5	12	26
6/7	19	41

*Entry criteria:UA or NSTEMII defined as ischemic pain at rest within past 24H, with evidence of CAD (ST segment deviation or +marker)

For more info go to wwwtimi.org

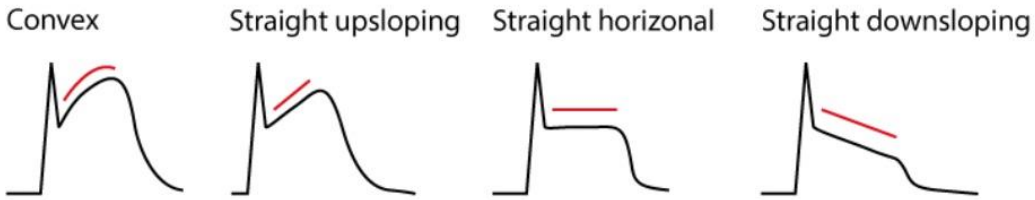
Antman et al *JAMA 2000*; 284: 835 - 842

ESC 2017 Guidelines

- In the proper clinical context, ST-segment elevation (measured at the J-point) is considered suggestive of ongoing coronary artery acute occlusion in the following cases:
  - at least two contiguous leads with ST-segment elevation ≥ 2.5 mm in men < 40 years, ≥2 mm in men ≥ 40 years, or ≥ 1.5 mm in women in leads V_2–V_3 and/or ≥ 1 mm in the other leads [in the absence of left ventricular (LV) hypertrophy or left bundle branch block LBBB)].[8]
  - In patients with inferior MI, it is recommended to record right precordial leads (V_3R and V_4R) seeking ST-segment elevation, to identify concomitant right ventricular (RV) infarction.[8,43]
  - Likewise, ST-segment depression in leads V_1–V_3 suggests myocardial ischaemia, especially when the terminal T-wave is positive (ST-segment elevation equivalent), and confirmation by concomitant ST-segment elevation ≥ 0.5 mm recorded in leads V_7–V_9 should be considered as a means to identify posterior MI.[8]
  - The presence of a Q-wave on the ECG should not necessarily change the reperfusion strategy decision.

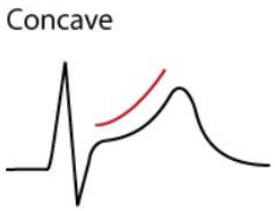
STEMI

A Characteristics of ST-segment elevations caused by ischemia



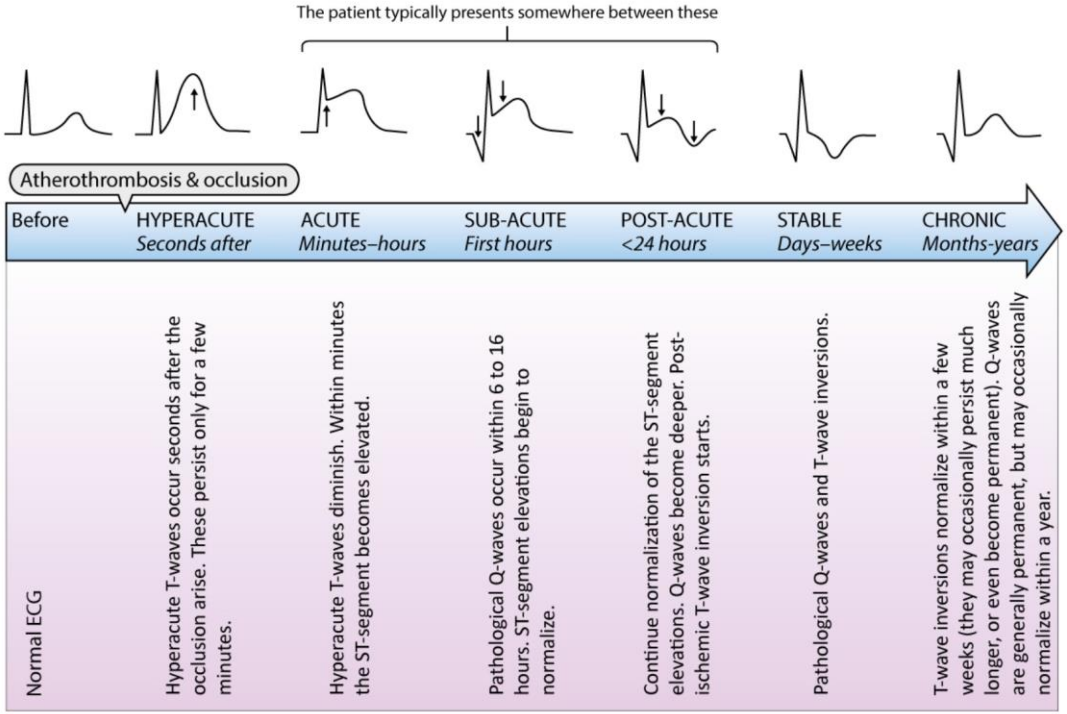
ST-segment elevations caused by ischemia typically displays a convex or straight ST-segment. Such ST-segment elevations in presence of chest discomfort are strongly suggestive of transmural myocardial ischemia. Note that the straight downsloping variant is unusual.

B Typical non-ischemic ST-segment elevation



Non-ischemic ST-segment elevations are extremely common in all populations. They are characterized by a concave ST-segment and a greater distance between the J point and the T wave apex.

The electrocardiographic natural course of ST-elevation myocardial infarction (STEMI)



de Winter's sign

de Winter's sign is an exception to the rule that upsloping ST-segment depressions are not ischemic. de Winter's sign implies the presence of upsloping ST-segment depressions with prominent T-waves in the majority of the precordial (chest) leads. This is a sign of acute ischemia, most often caused by a proximal occlusion of the left anterior descending (LAD) artery.

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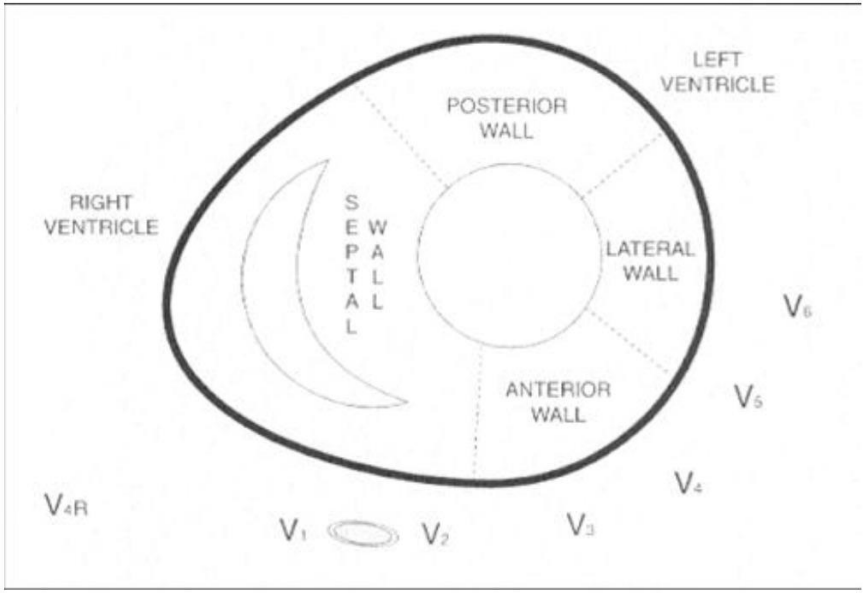
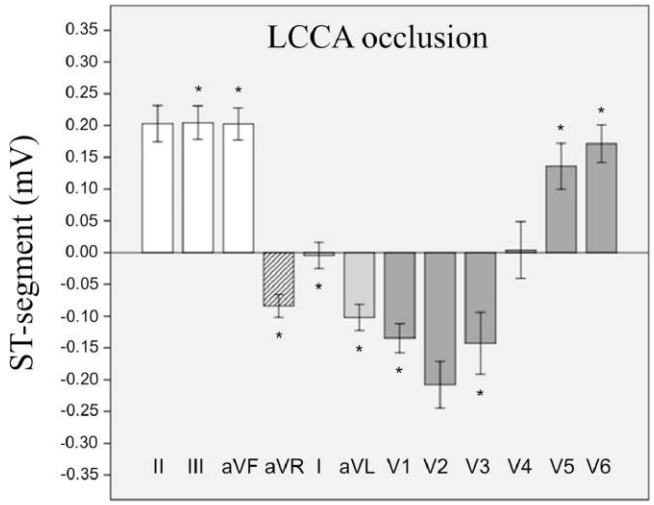
I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

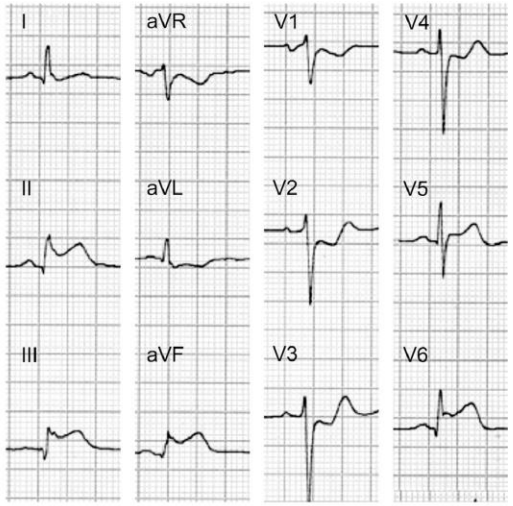
EMS12Lead.com

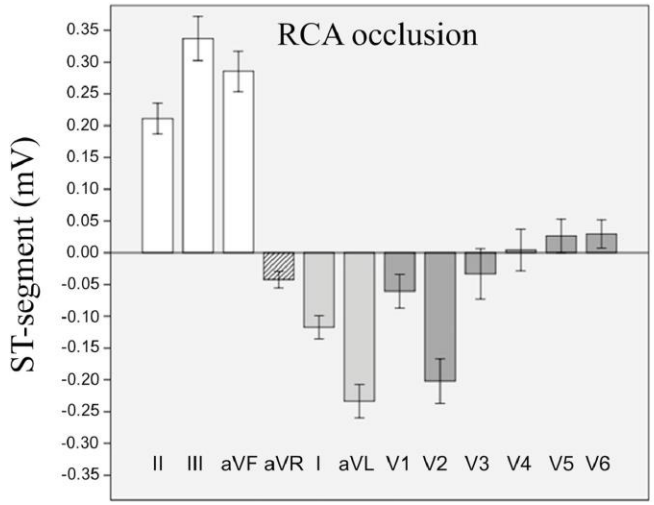
That's the reason we sometimes miss acute STEMI in the distribution of the circumflex artery.

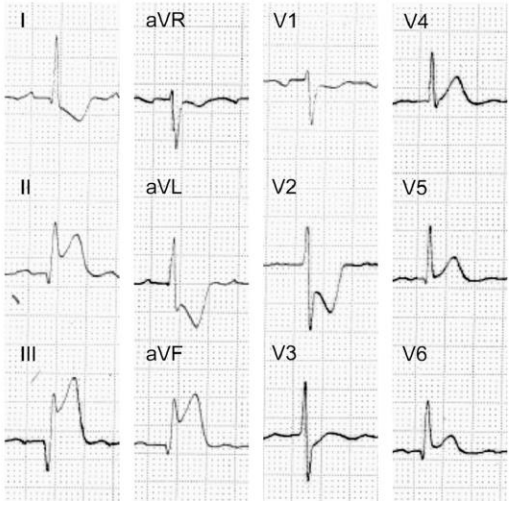


But what about the Cx?

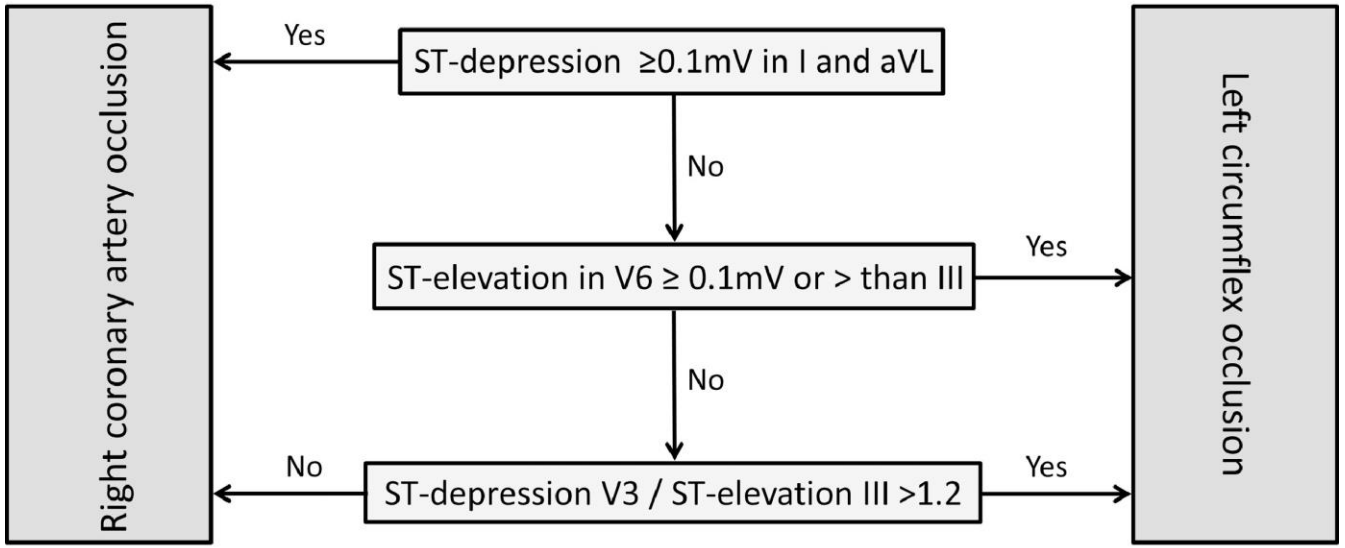








ST-segment elevation in inferior ECG leads

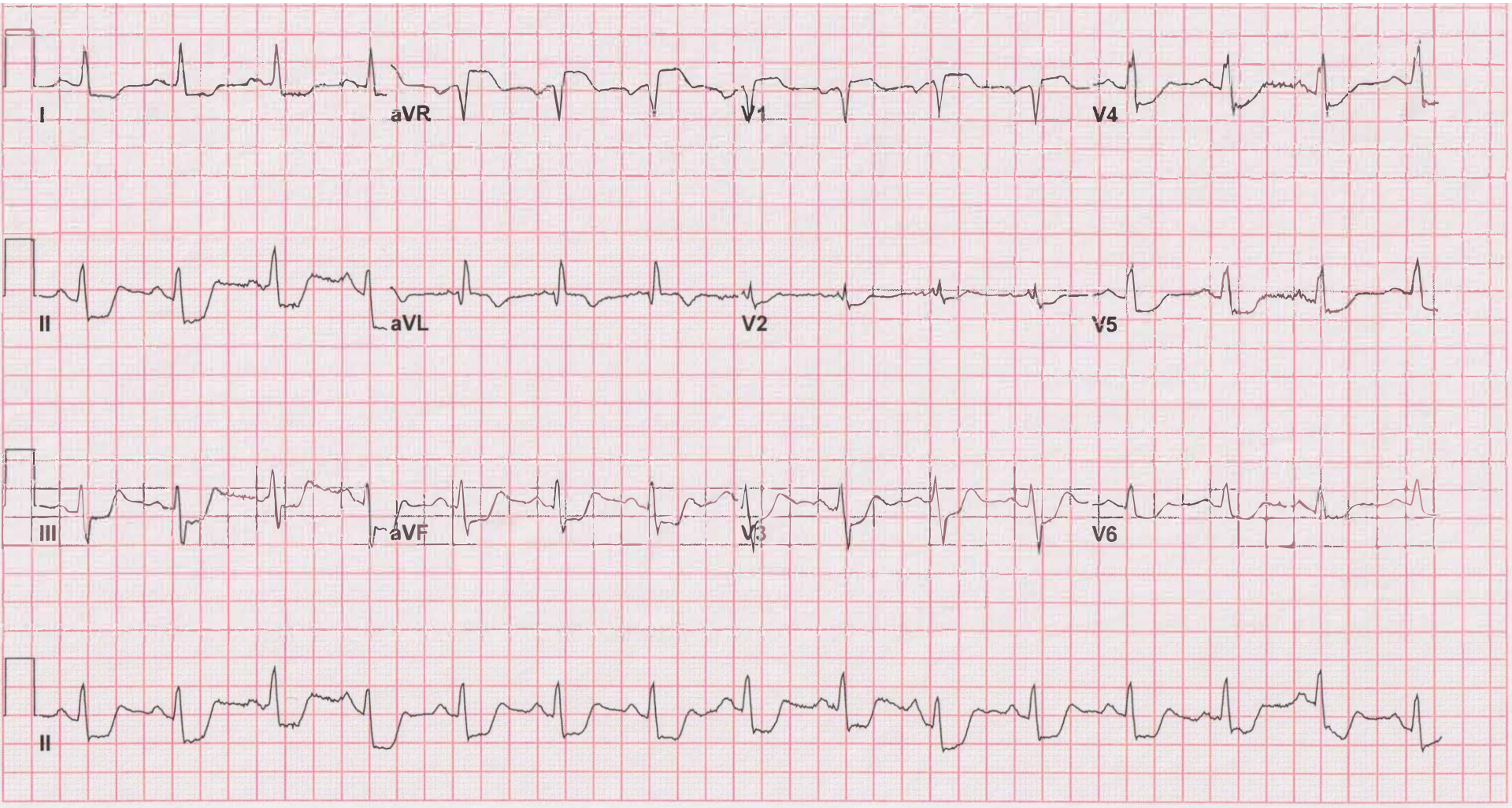


Sensitivity 77% Specificity 86% Accuracy 82% Youden Index 0.63

Apparent AUC 0.85 Validated AUC (10-fold cross-validation) 0.82

(Am J Cardiol 2019;123:1019−1025)
April 1, 2019

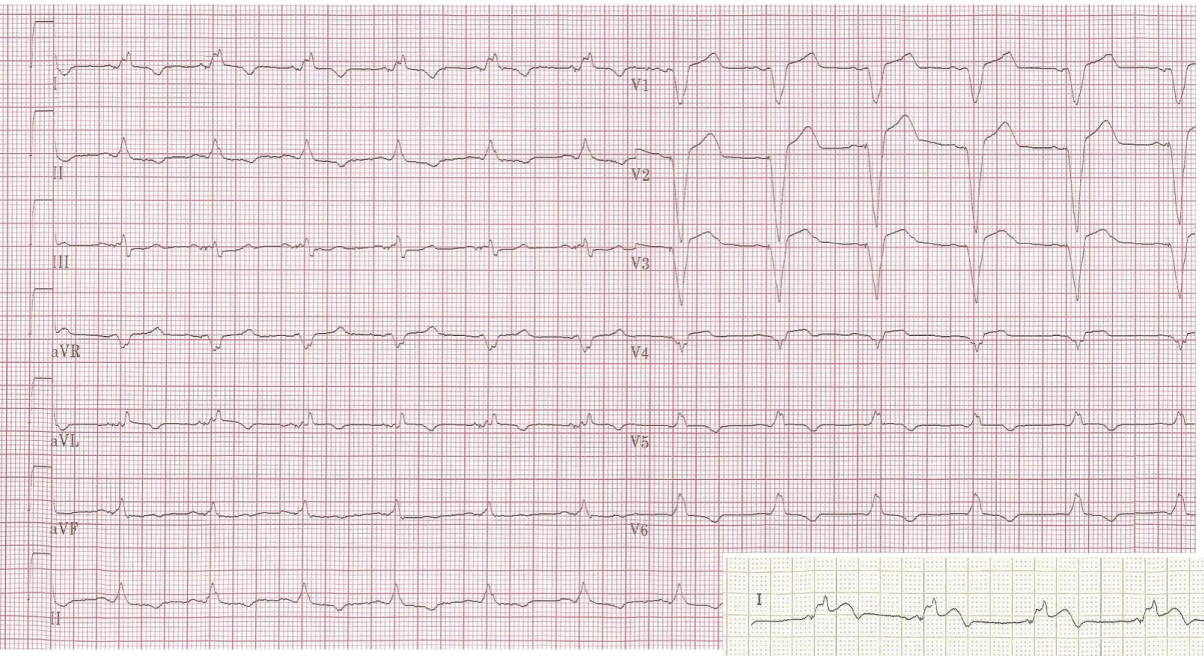
LM STEMI EKG

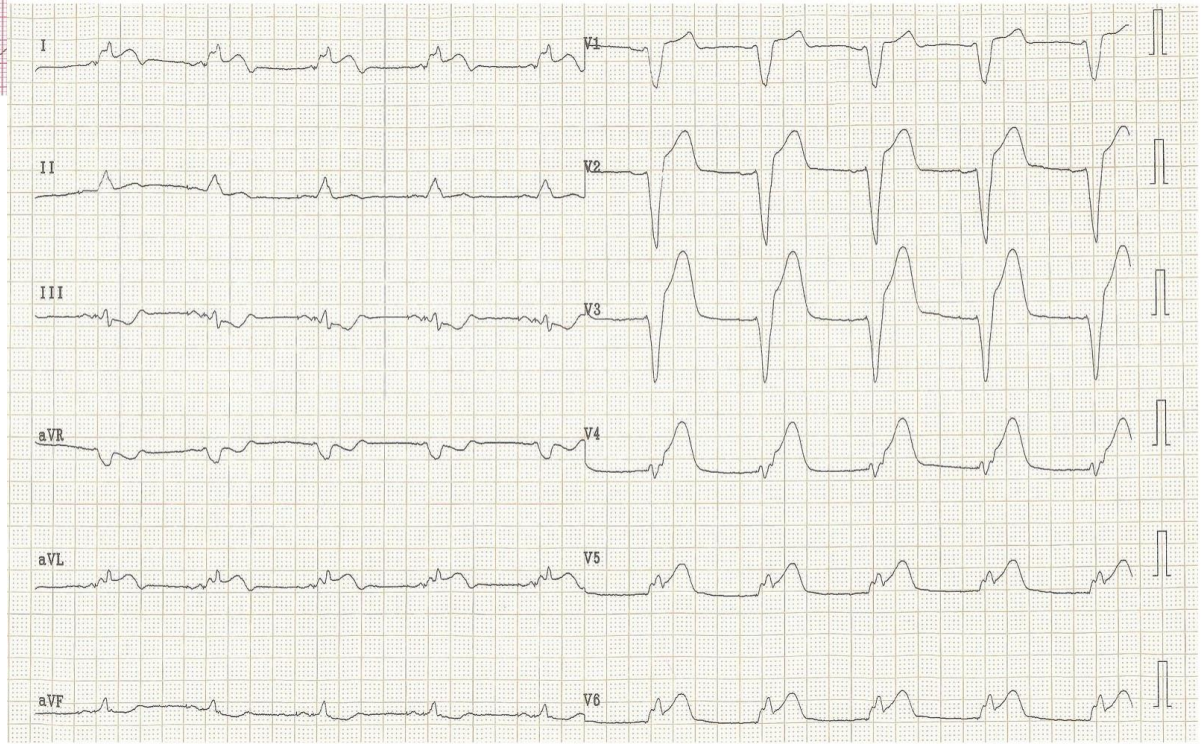


Value of reciprocal changes

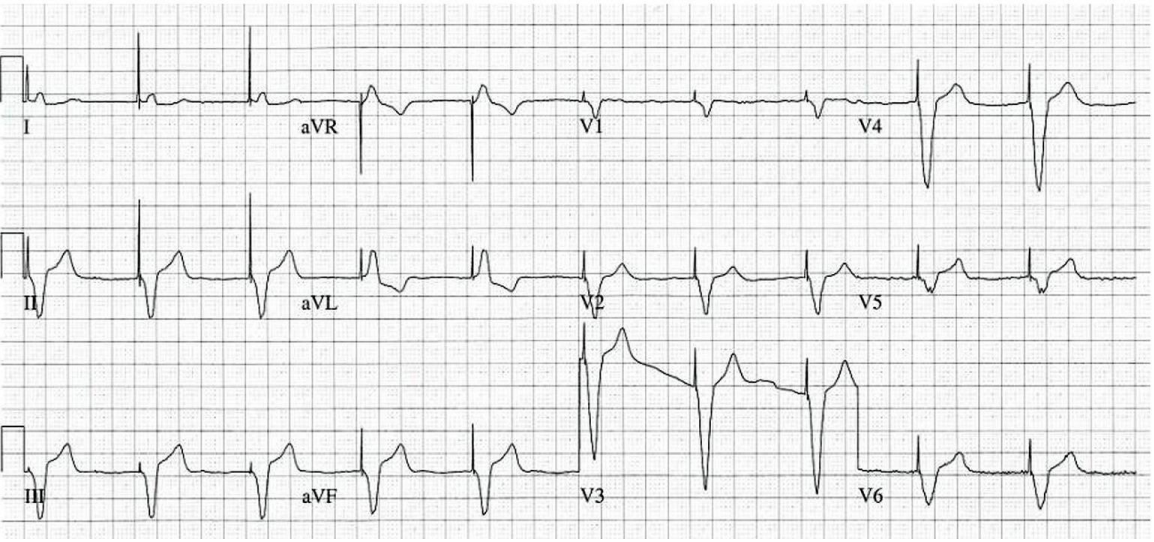
- Not of value in LBBB, paced rhythm or LVH/strain
- Defined and horizontal or down-sloping ST-depression in at least one lead “opposite” to ST-elevations
- Present in 75% of IWMI, only 30% of anterior MI’s, “frequent” in lateral MI’s
  - Presence of reciprocal changes is supportive of diagnosis of STEMI, but absence does not exclude STEMI
- By CMR predict greater area of myocardium at risk
- Predicts increase risk of cardiogenic shock or high degree AV-block

Left Bundle Branch Block

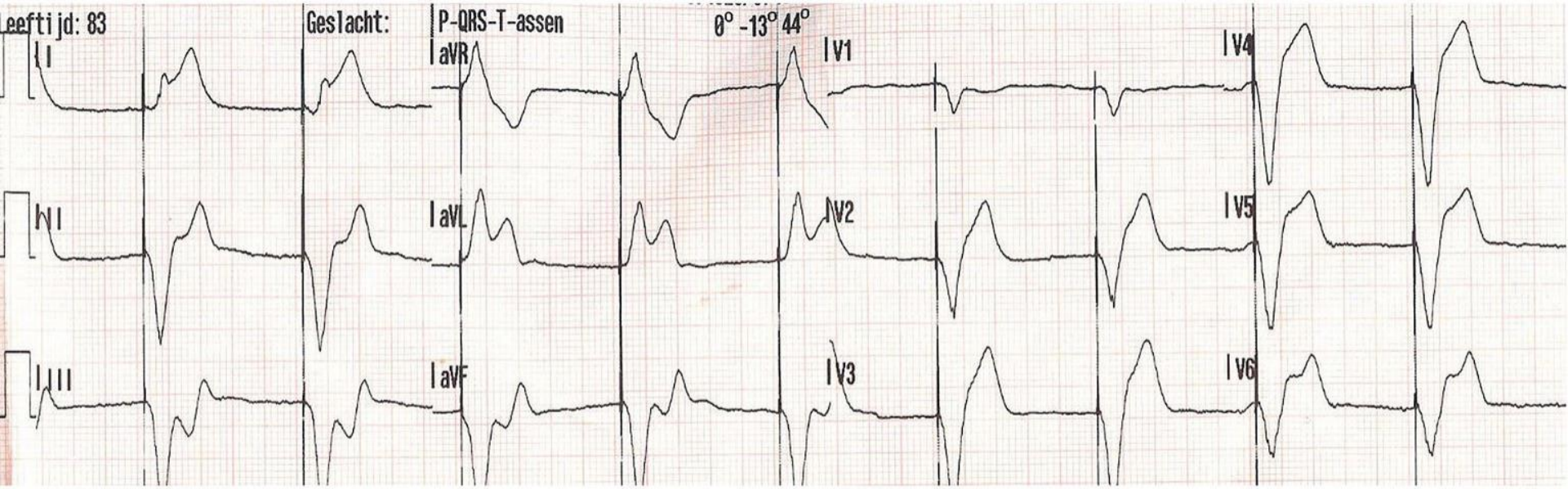


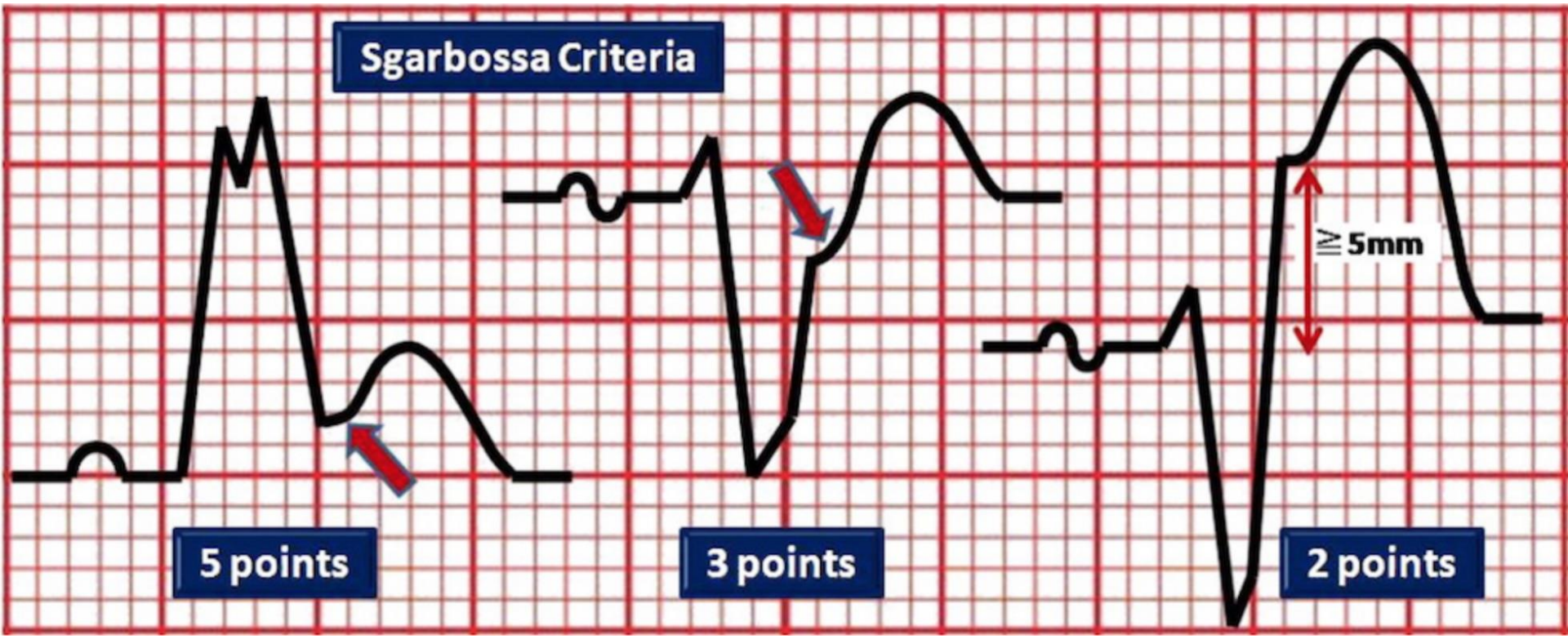


RV-pacemaker



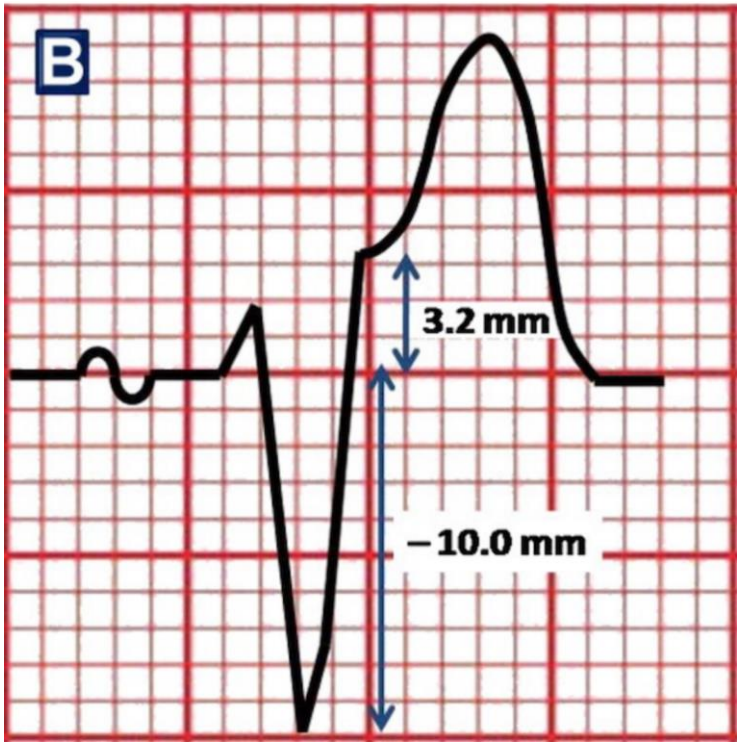
ECG PEDIA.ORG
part of cardionetworks.org





- **Sgarbossa Criteria** (**≥3** points is considered **positive**):
  - **CONCORDANT** ST elevation ≥1mm that is **5 points**
  - **CONCORDANT** ST depression ≥1mm in V1-V3 that is **3 points**
  - **DISCORDANT** ST elevation ≥5mm that is **2 points**

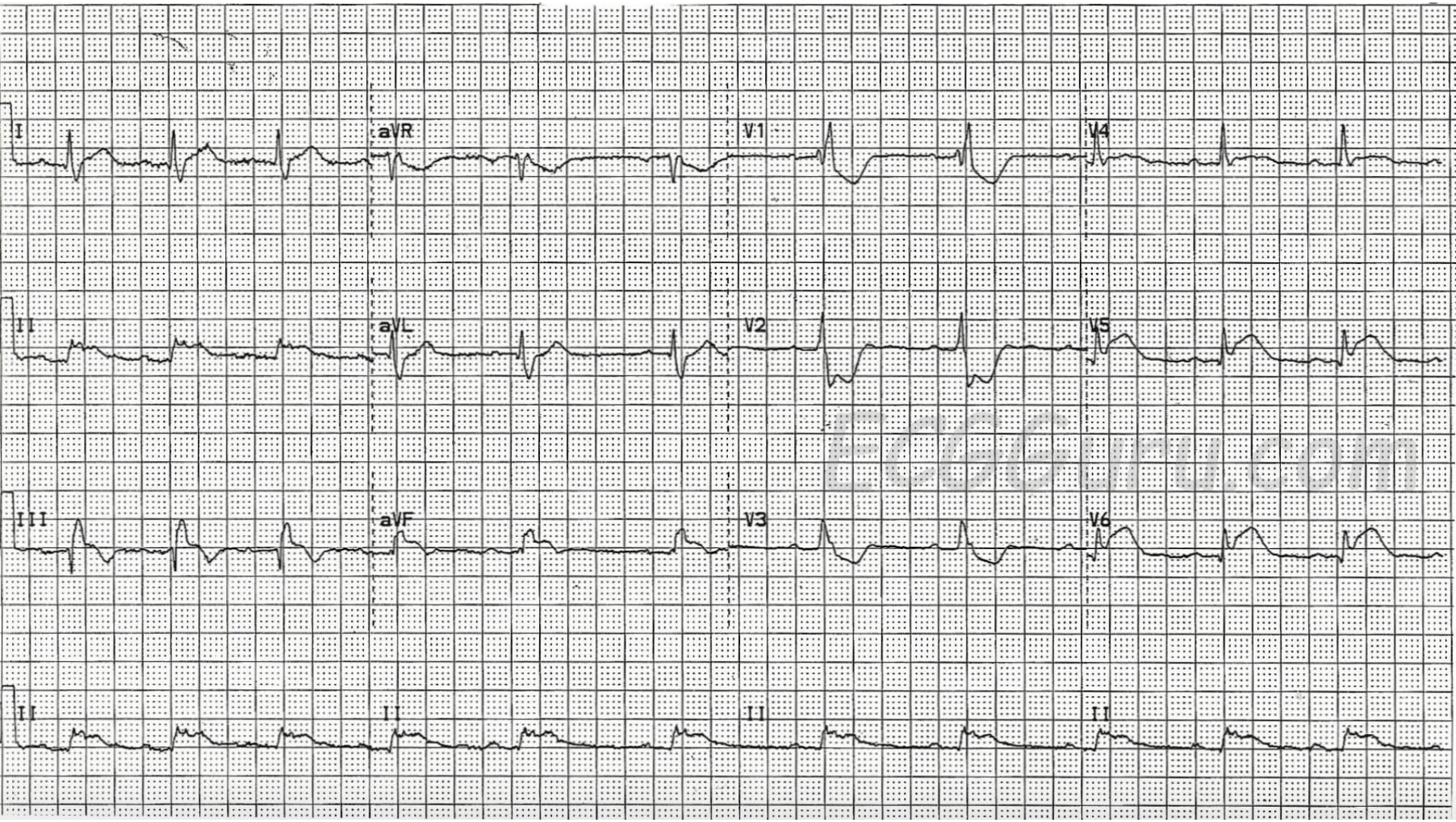
Sgarbossa E et al. Electrocardiographic Diagnosis of Evolving Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block. NEJM 1996; 334: 481-487



- **Modified Sgarbossa** (any **one** criteria is considered **positive**):
  - First and second criteria are the same.
  - The third criterion is changed to ST elevation to S- wave amplitude ratio ≥ 0.25.
- Modified Sgarbossa Criteria vs Original Weighted Sgarbossa Criteria:
  - Sensitivity: 80% vs 49 %
  - Specificity: 99% vs 100%
- Modified Sgarbossa Criteria vs Unweighted Sgarbossa Criteria:
  - Sensitivity: 80% vs 56%
  - Specificity: 99% vs 94%

Smith S et al. 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

RBBB STEMI



Normal ST-variants

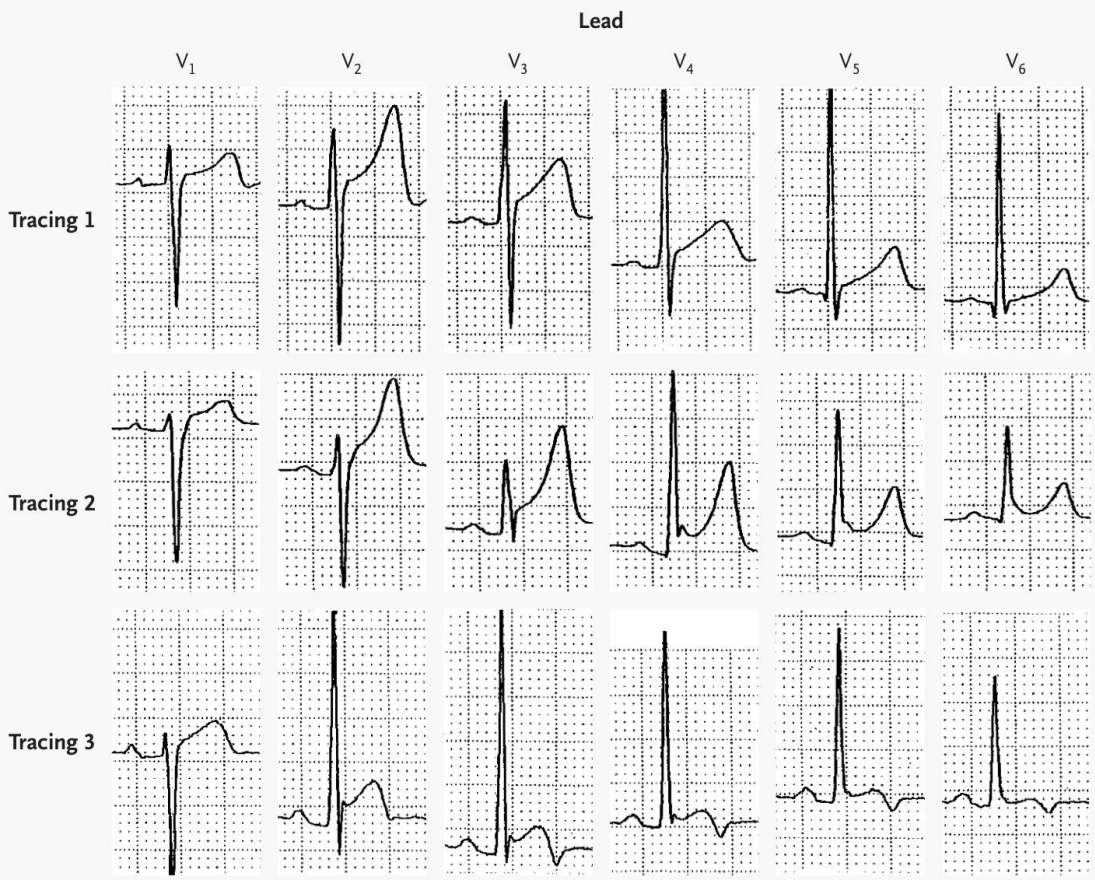


Figure 1. Electrocardiograms Showing Normal ST-Segment Elevation and Normal Variants.

Tracing 1 shows normal ST-segment elevation. Approximately 90 percent of healthy young men have ST-segment elevation of 1 to 3 mm in one or more precordial leads. The ST segment is concave. Tracing 2 shows the early-repolarization pattern, with a notch at the J point in V_4. The ST segment is concave, and the T waves are relatively tall. Tracing 3 shows a normal variant that is characterized by terminal T-wave inversion. The QT interval tends to be short, and the ST segment is coved.

Normal ST-elevation

- J-point far < 50% T-wave height
- Concave up
- Males under 60-yo up to 3 mm most prominent in V2, < 1 mm V5-V6
  - Rare > 70-yo
- Rare and usually < 1mm in II, III, aVF
- Females usually < 1 mm, less pronounced in limb leads, no age differences

N Engl J Med 2003;349:2128-35.

Early Repolarization

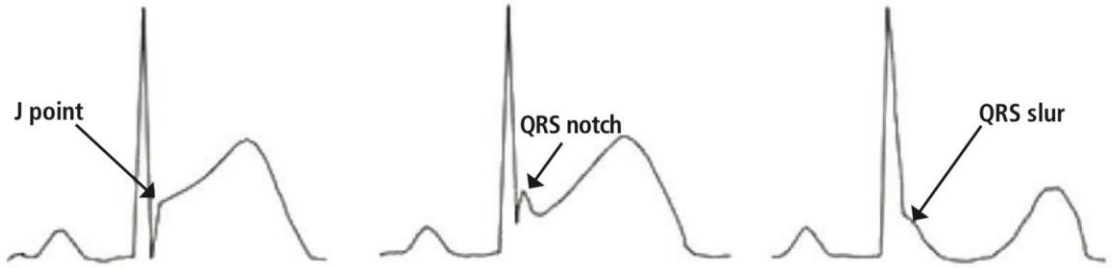
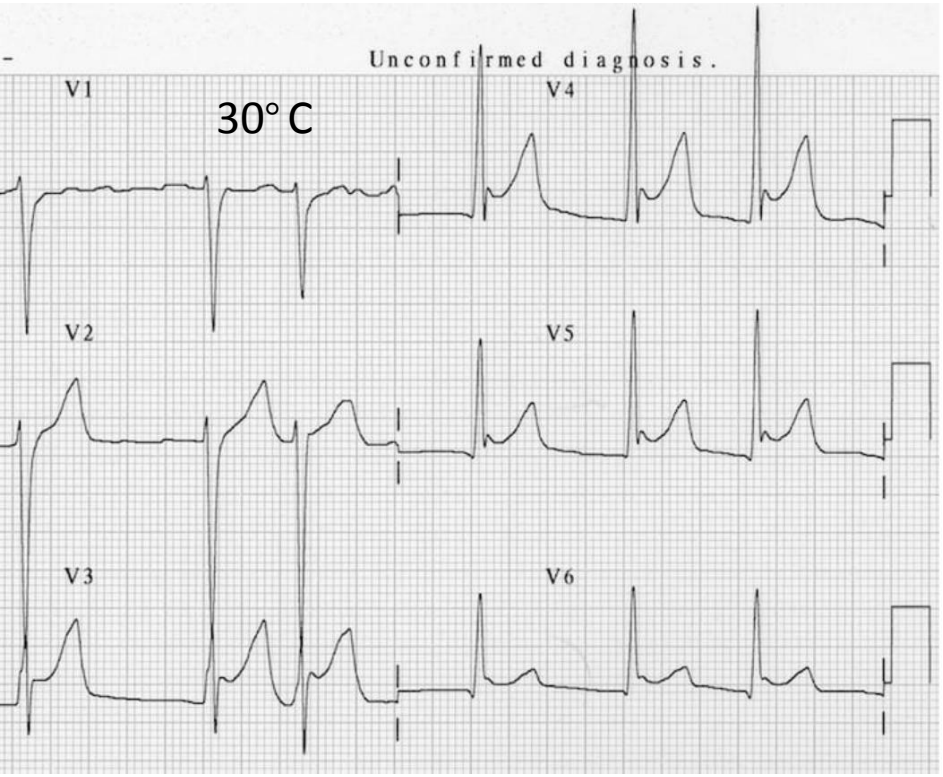


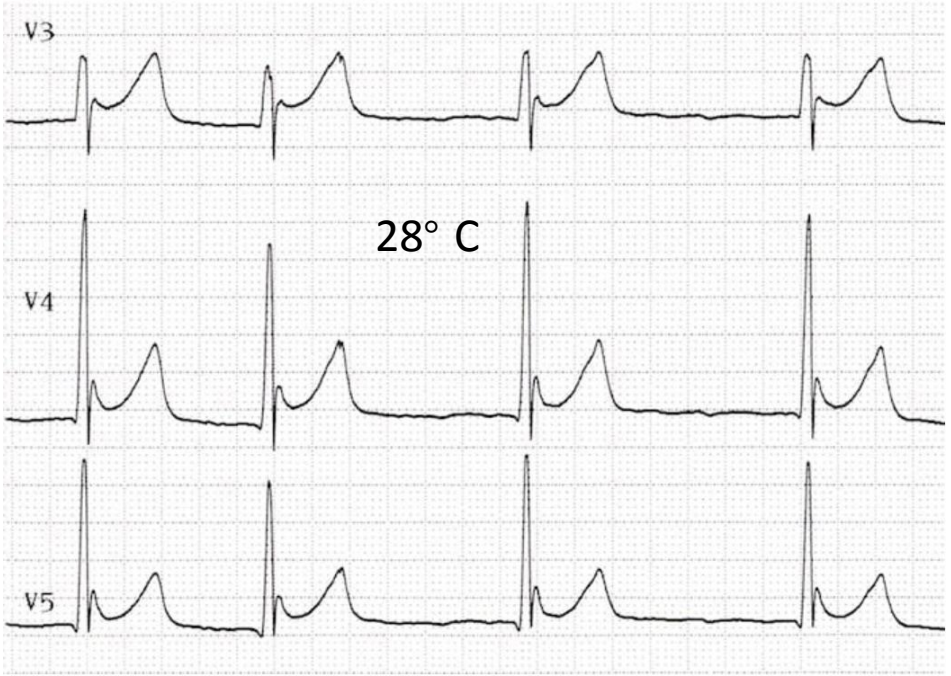
Figure 1. Early repolarization with and without QRS notch or slur.

- J-point elevation ≥ 0.1 mV in 2 or more contiguous leads on 12-lead ECG, excluding leads V1 to V3, with the presence of terminal QRS notch or slur and QRS duration less than 120 msec (J Am Coll Cardiol 2015; 66(4):470–477).
- Present in 5-15% of general population, more common in younger patients and in African American males.
- In the absence of syncope or family history of sudden cardiac death, early repolarization does not merit further workup.
- Warrants EP-study after unexplained syncope
- High risk features include:
  - Inferior or global as opposed to lateral distribution;
  - J-wave ≥ 2 mm;
  - Notching of the terminal QRS

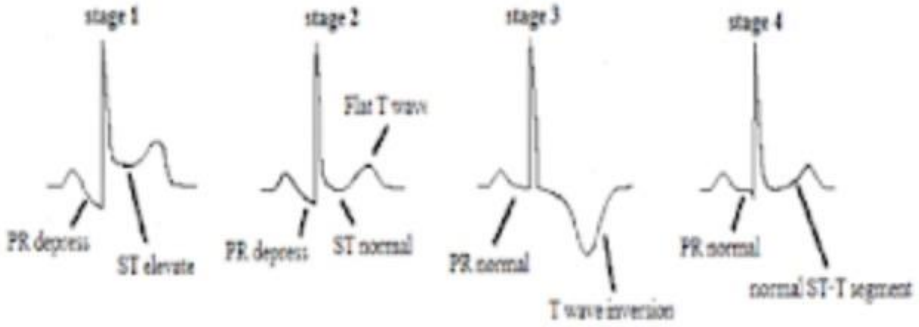
CLEVELAND CLINIC JOURNAL OF MEDICINE VOLUME 86 • NUMBER 3 MARCH 2019

J-waves of Osborne



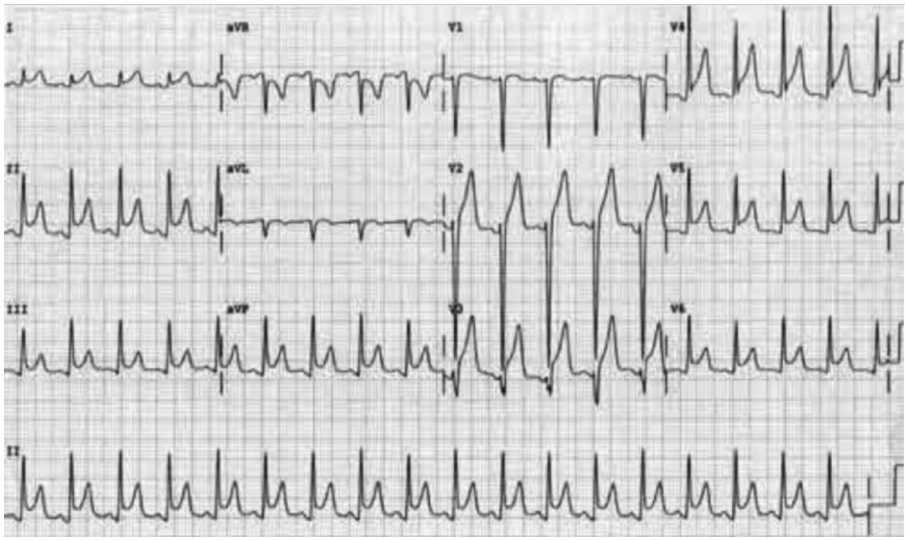


Pericarditis



- **Stage 1:** This stage spans over the first two weeks. There is a general **concave up** ST-elevation in all leads with the exception of aVR, V1 and III. PR-depression is also generally seen throughout the same leads too. Reciprocal changes are seen only in lead aVR.
- **Stage 2:** This stage spans over the next 1 to 3 weeks. In this stage, there is a normalization of ST changes. There is also a generalized T-wave flattening in all the leads. This stage is also called the pseudonormalization stage as the ECG gets normal in this transition period before more changes are observed.
- **Stage 3:** This stage starts after three weeks for the next several weeks. It is characterized by diffuse T-wave inversion of the previously flattened T-waves.
- **Stage 4:** After the next several weeks, the ECG goes back to normalization and this marks the fourth stage of pericarditis.

Myocarditis

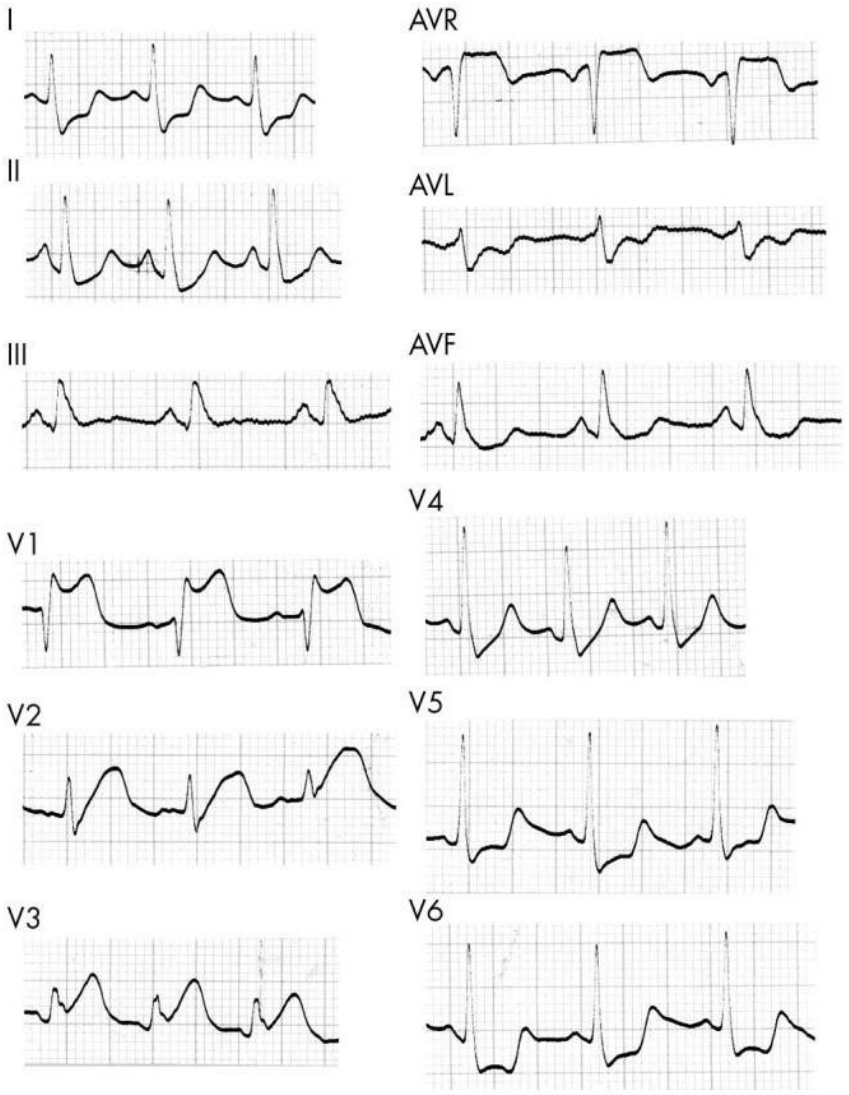


- Sinus tachycardia
- Afib
- QRS, QT prolongation
- Diffuse T-wave inversion
- AV block
- Ventricular arrhythmias

Improved outcomes with ACEI, statins
Equivocal benefit with beta blockers
No benefit/increased bleeding with DAPT

(Review the cath film!)

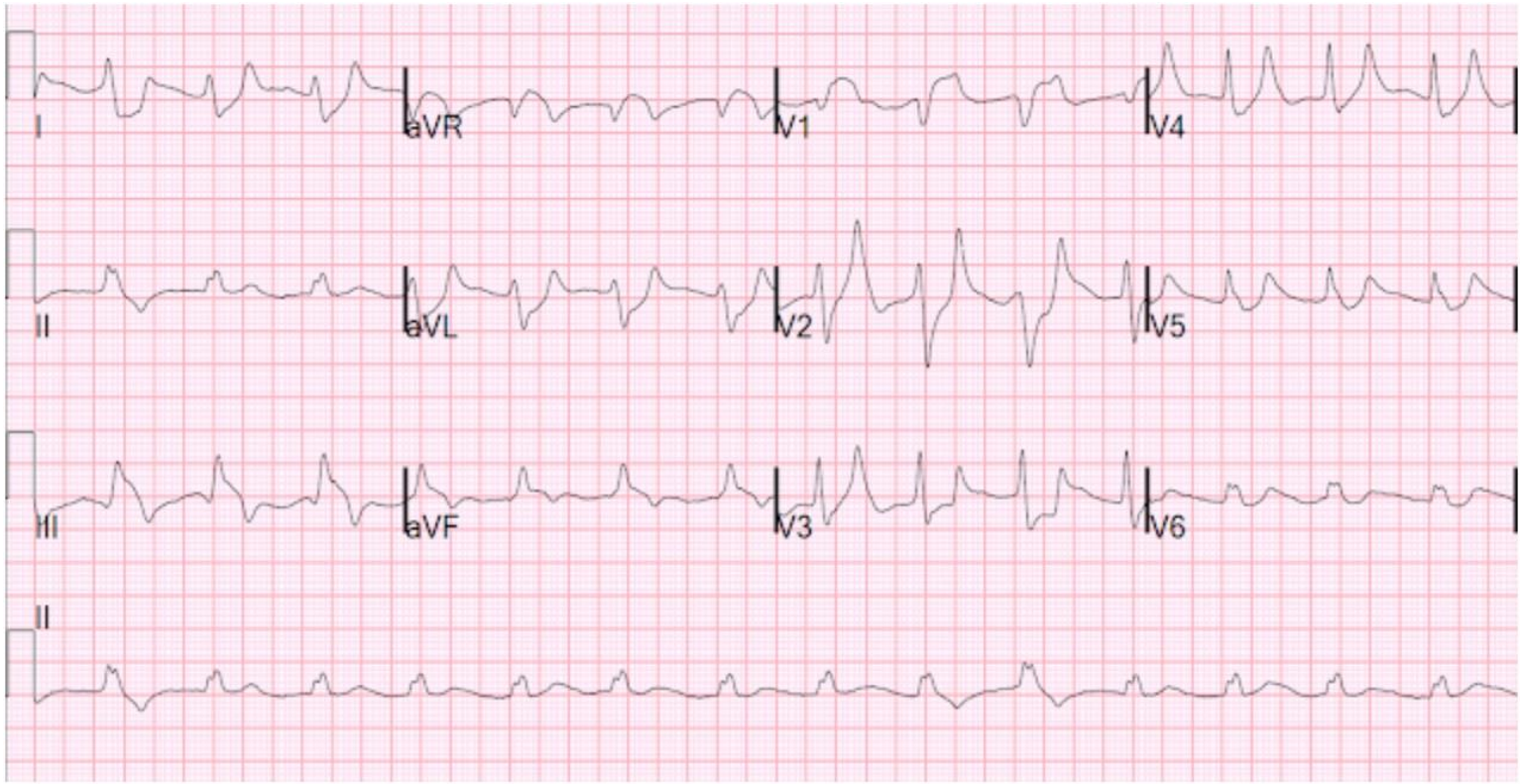
Pulmonary Embolism ST-elevation



- EKG normal 18%.
- ST 44%
- RV strain (TWI V1-V3 ± inferior leads) 34%
- RAD 16%
- P-pulmonale 9%
- S1Q3T3 < 20%
- Afib/flutter/tachy 8%

Hyperkalemia

- Bradycardia
- Peaked T-waves: > 50% of R-wave
- Loss of P-waves, but regular rhythm
- QRS widening, slurring of ST- into T-wave
- Short QT



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WILEY CLINICAL CARDIOLOGY

REVIEW

Lithium-induced electrocardiographic changes: A complete review

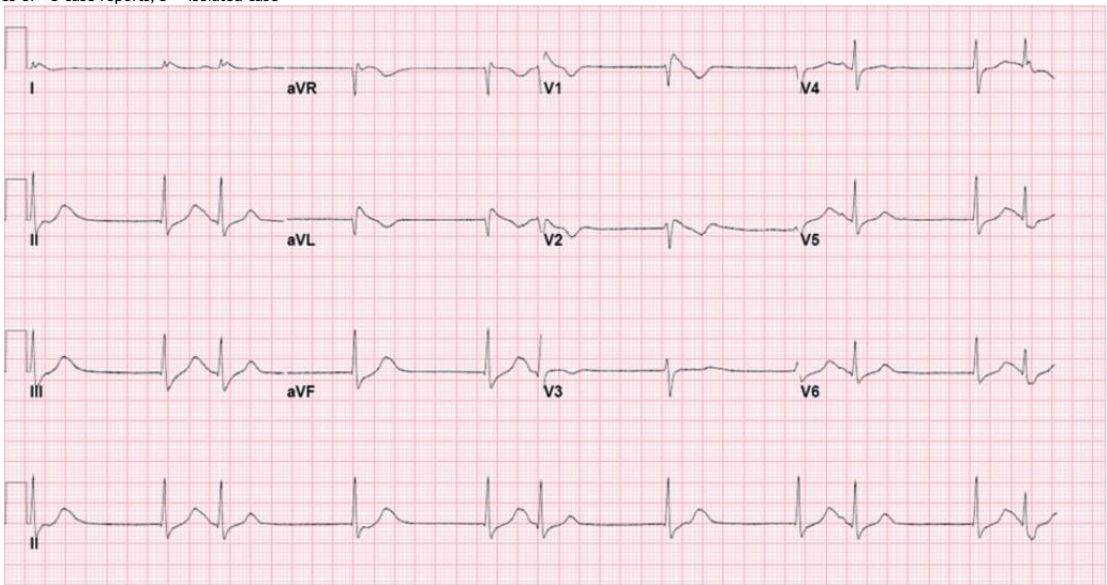
Nikhil Mehta MD[1] | Robert Vannozzi MD[2]

TABLE 1 Summary of ECG findings in patients on lithium therapy

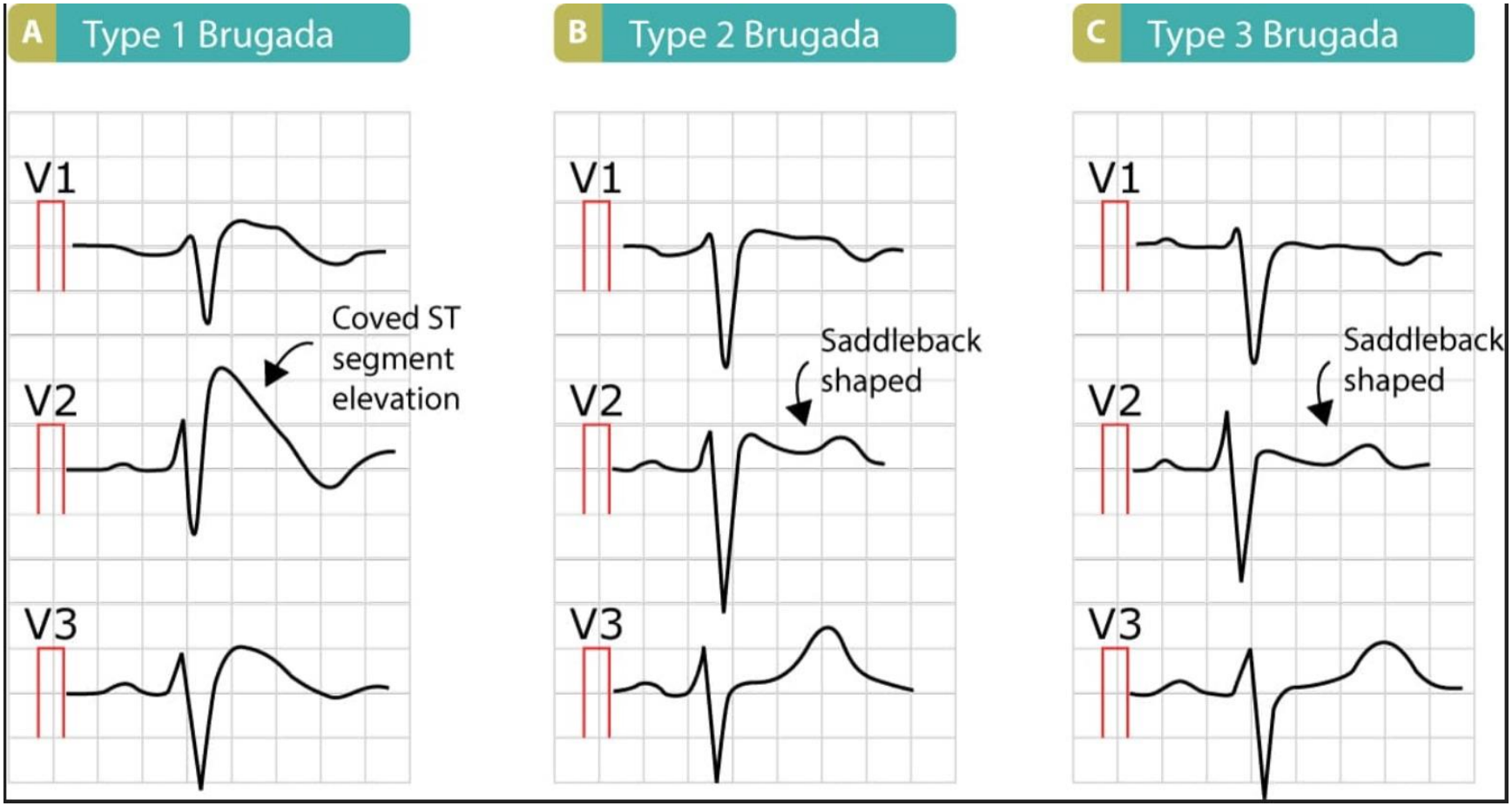
ECG Component	Findings	Level of Ev		
Rate and rhythm	Sinus node dysfunction and bradycardia	2a		
	Increased atrial conduction time	2b		
	Atrial flutter	5		
	Sick sinus syndrome	5		
	Cardiac asystole	5	Human	14
P and PR	Sinoatrial blocks	4	Human	15–17
	PR prolongation and atrioventricular blocks	3b	Human	10,19–21
QRS	Incomplete bundle branch blocks	4	Human	20
	Right bundle branch block	5	Human	20
	Nonspecific intraventricular conduction delay	5	Human	20
	Left bundle branch block	5	Human	23
ST	Depression	4	Human	24
	Elevation	4	Human	25–27
	Brugada pattern	5	Human	28,29
T and QTc	T wave flattening or inversion	2a	Human	10,32–34
	QTc prolongation	5	Human	36
	Higher QT dispersion ratio	2a	Human	18,35
	Ventricular tachyarrhythmias	4	Animal	36

Abbreviations: ECG, electrocardiogram.

[a] Levels of evidence: 1a = multiple/homogenous RCTs, 1b = individual RCT, 2a = multiple/homogenous prospective cohort studies, 2b = individual cohort study, 3a = multiple/homogenous retrospective studies, 3b = individual retrospective study, 4 = case series or >3 case reports, 5 = isolated case reports or expert opinion.



Brugada Variations



ECG Criteria to Differentiate Between Takotsubo (Stress) Cardiomyopathy and Myocardial Infarction

Antonio H. Frangieh, MD, MPH;* Slayman Obeid, MD;* Jelena-Rima Ghadri, MD; Yoichi Imori, MD; Fabrizio D'Ascenzo, MD; Marc Kovac; Frank Ruschitzka, MD; Thomas F. Lüscher, MD; Firat Duru, MD;* Christian Templin, MD, PhD, FESC;* on behalf of the InterTAK Collaborators†

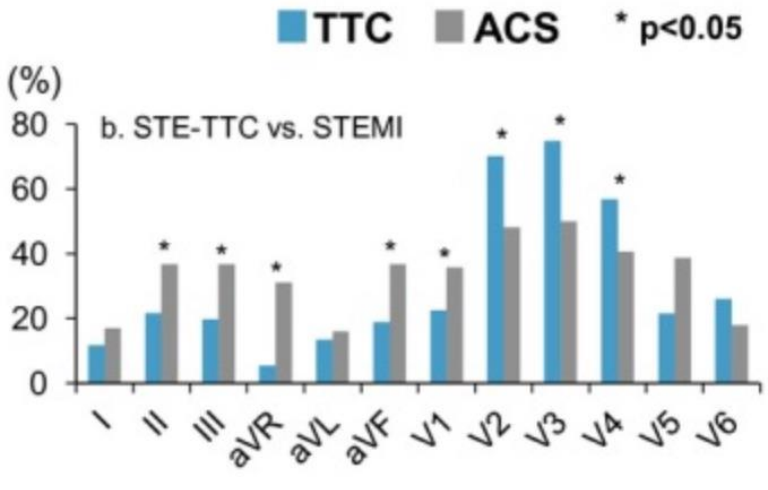
Characteristics of the Total Study Population; Comparison Between MI and TTC

	Total TTC	Total MI	*P* Value
	N=200	N=200	
Baseline characteristics			
Age, y[a]	65.5±12.1	65.8±12.3	0.62
Female	182 (91)	53 (27)	<0.001
BMI, kg/m²[a]	24.5±4.4	28.4±6.0	<0.001
Cardiovascular risk factors and cardiovascular history			
Hypertension	109 (55)	125 (64)	0.10
Diabetes mellitus	18 (9)	37 (19)	0.006
Ever-smoker	71 (36)	114 (57)	<0.001
Current smoker	38 (19)	86 (43)	<0.001
Dyslipidemia	52 (26)	109 (56)	<0.001
Positive family history of cardiovascular disease	50 (25)	53 (28)	0.65
Known CAD	11 (6)	31 (16)	0.001
Clinical and laboratory parameters			
EF at admission (%)[a]	43±10 (N=193)	51±11 (N=151)	<0.001
Peak troponin level (ULN)[a]	21.0±27.7 (N=187)	36.4±63.5 (N=200)	0.002
Peak CK level (ULN)[a]	2.5±7.3 (N=164)	7.5±10.2 (N=200)	<0.001
Peak CRP level, mg/L[a]	35.4±54.5 (N=173)	67.5±109.9 (N=192)	0.001
In-hospital complications			
Cardiogenic shock	16 (8)	19 (10)	0.60
All-cause mortality	8 (4)	10 (5)	0.64

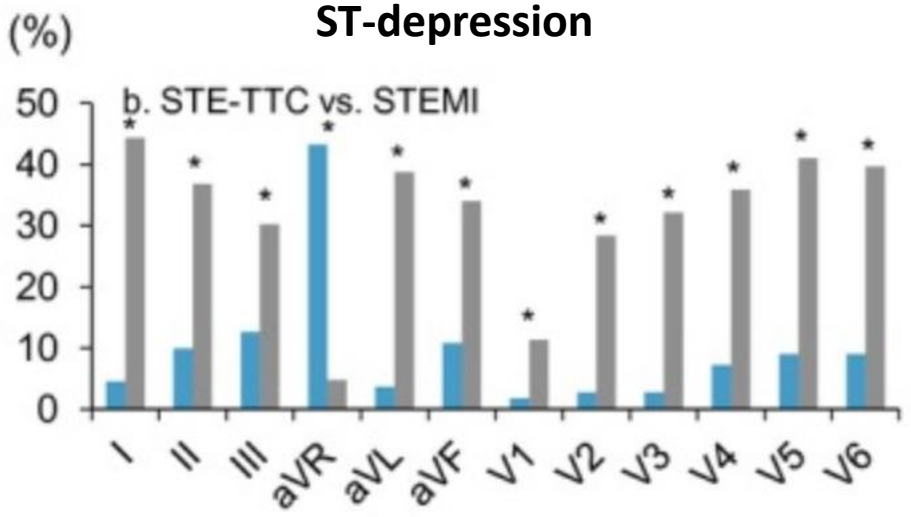
J Am Heart Assoc. 2016;5: e003418

Takatsubo v STEMI EKG

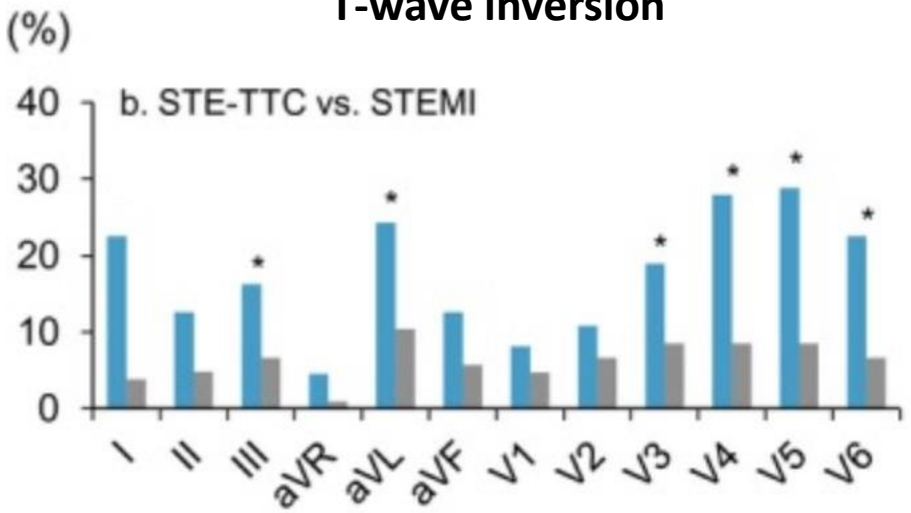
ST-elevation



ST-depression

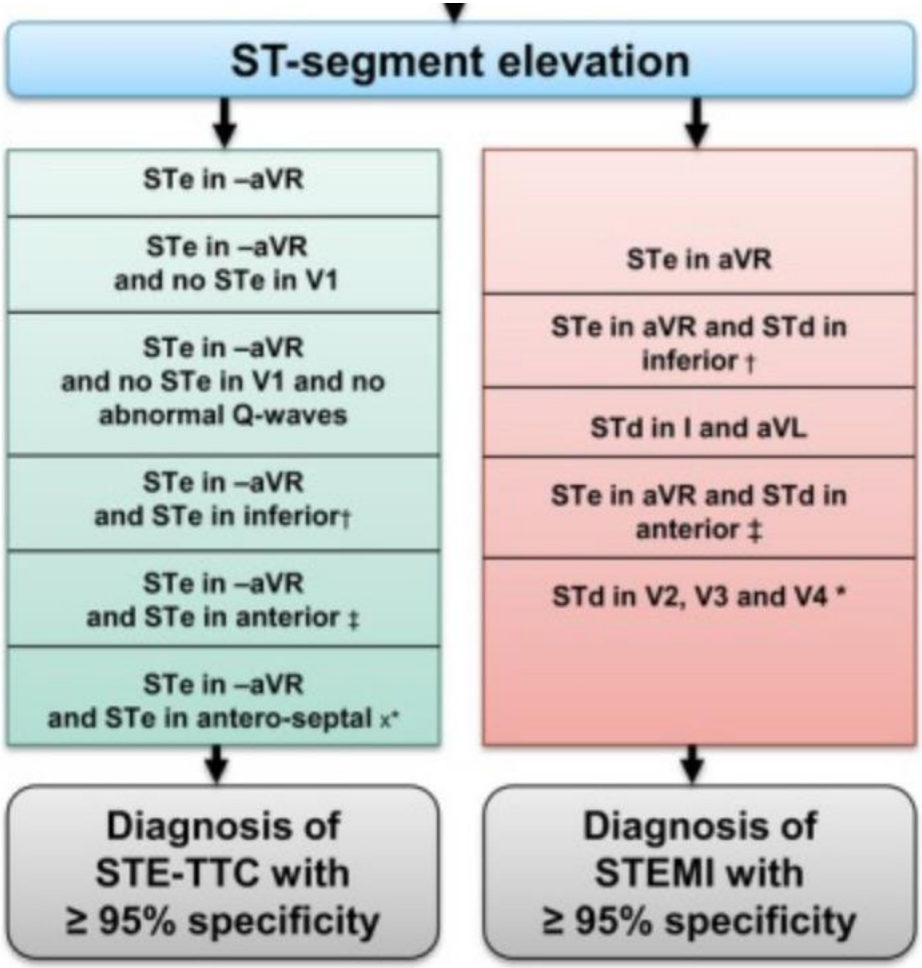


T-wave inversion

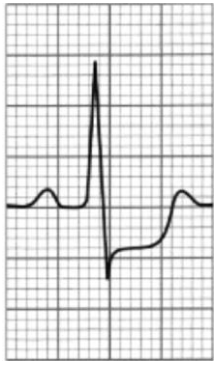


Takatsubo v STEMI by EKG

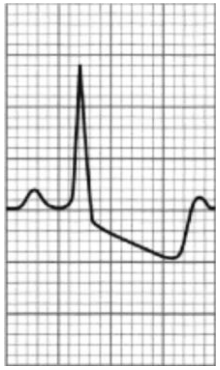
Distinguish -aVR from aVR!



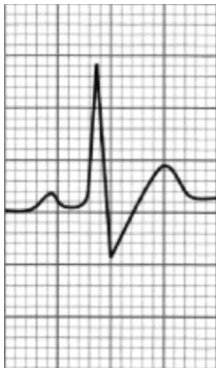
ST-depression



- Ischemia
- Reciprocal changes
- In V1-V3 posterior MI
- Hypokalemia
- Hyperventilation



- Ischemia
- Reciprocal changes
- In V1-V3 posterior MI
- LVH with strain



- Exercise
- Tachycardia
- deWinters sign (with peaked T-wave)
- Quinidine

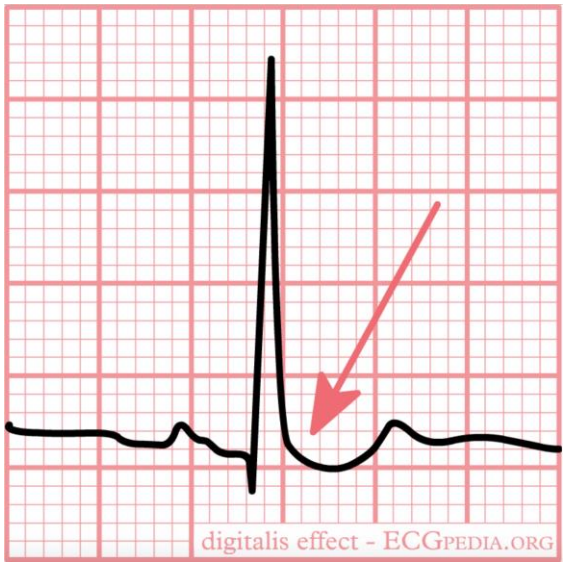
Myocardial ischemia

ST depression and T wave changes

- New horizontal or down-sloping ST-depression ≥0.05 mV in two contiguous leads and/or T inversion ≥0.1 mV in two contiguous leads with prominent R wave or R/S ratio ≥1.

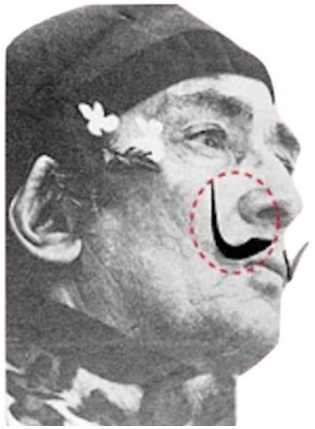
LVH

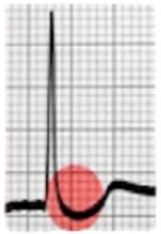
- The **Sokolow-Lyon index**:
  - S in V_1 + R in V_5 or V_6 (whichever is larger) ≥ 35 mm
  - R in aVL ≥ 11 mm
- The **Cornell voltage criteria**:
  - S in V_3 + R in aVL > 28 mm (men)
  - S in V_3 + R in aVL > 20 mm (women)
- The **Romhilt-Estes point score system** ("diagnostic" >5 points; "probable" 4 points):
  - Voltage Criteria (any of): (3 points)
    - R or S in limb leads ≥20 mm
    - S in V_1 or V_2 ≥30 mm
    - R in V_5 or V_6 ≥30 mm
  - ST-T Abnormalities:
    - ST-T vector opposite to QRS without digitalis (3 points)
    - ST-T vector opposite to QRS with digitalis (1 point)
  - Negative terminal P mode in V_1 1 mm in depth and 0.04 sec in duration (3 points)
  - Left axis deviation (QRS of -30° or more) (2 points)
  - QRS duration ≥0.09 sec (1 point)
  - Delayed intrinsicoid deflection in V_5 or V_6 (>0.05 sec) (1 point)



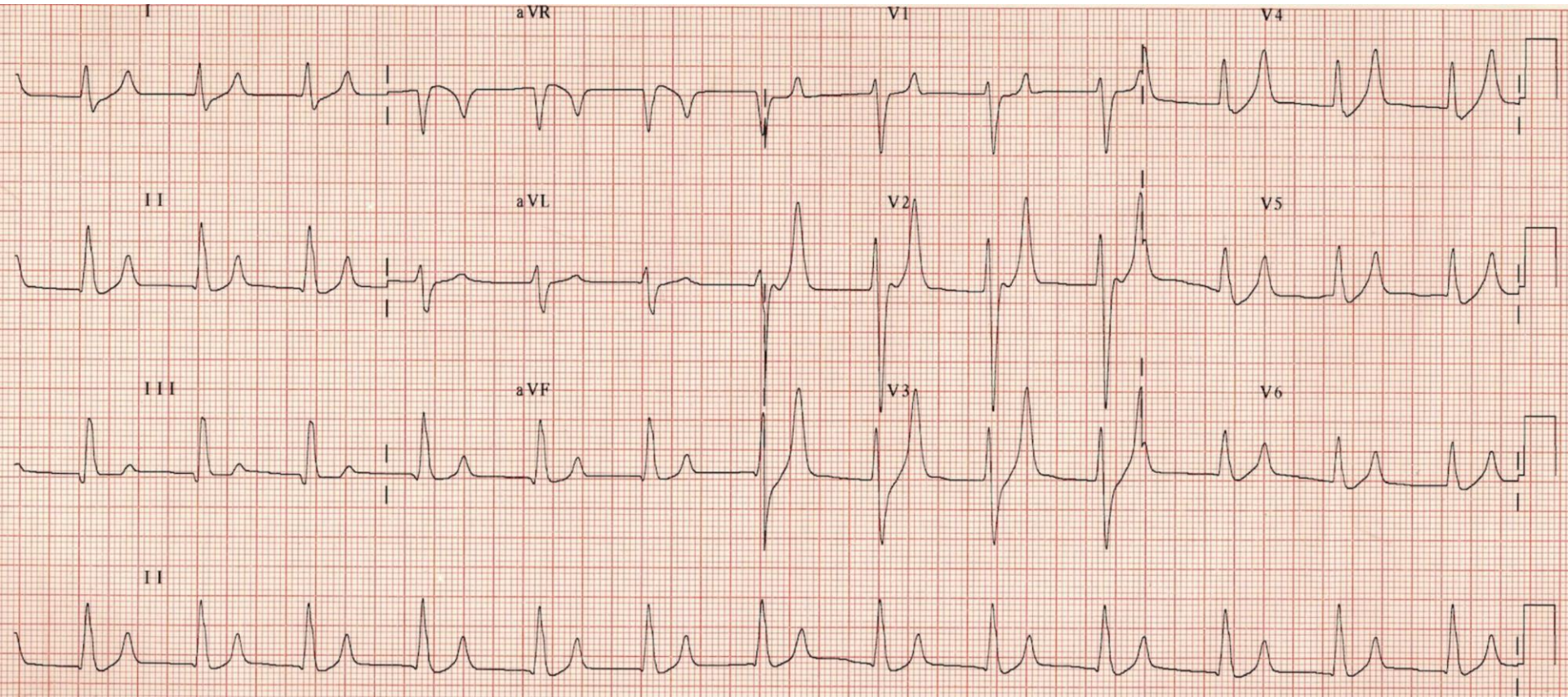
Digoxin

- Coved ST-changes
- Peaked T-waves
- Short QTc
- Flattened P-waves

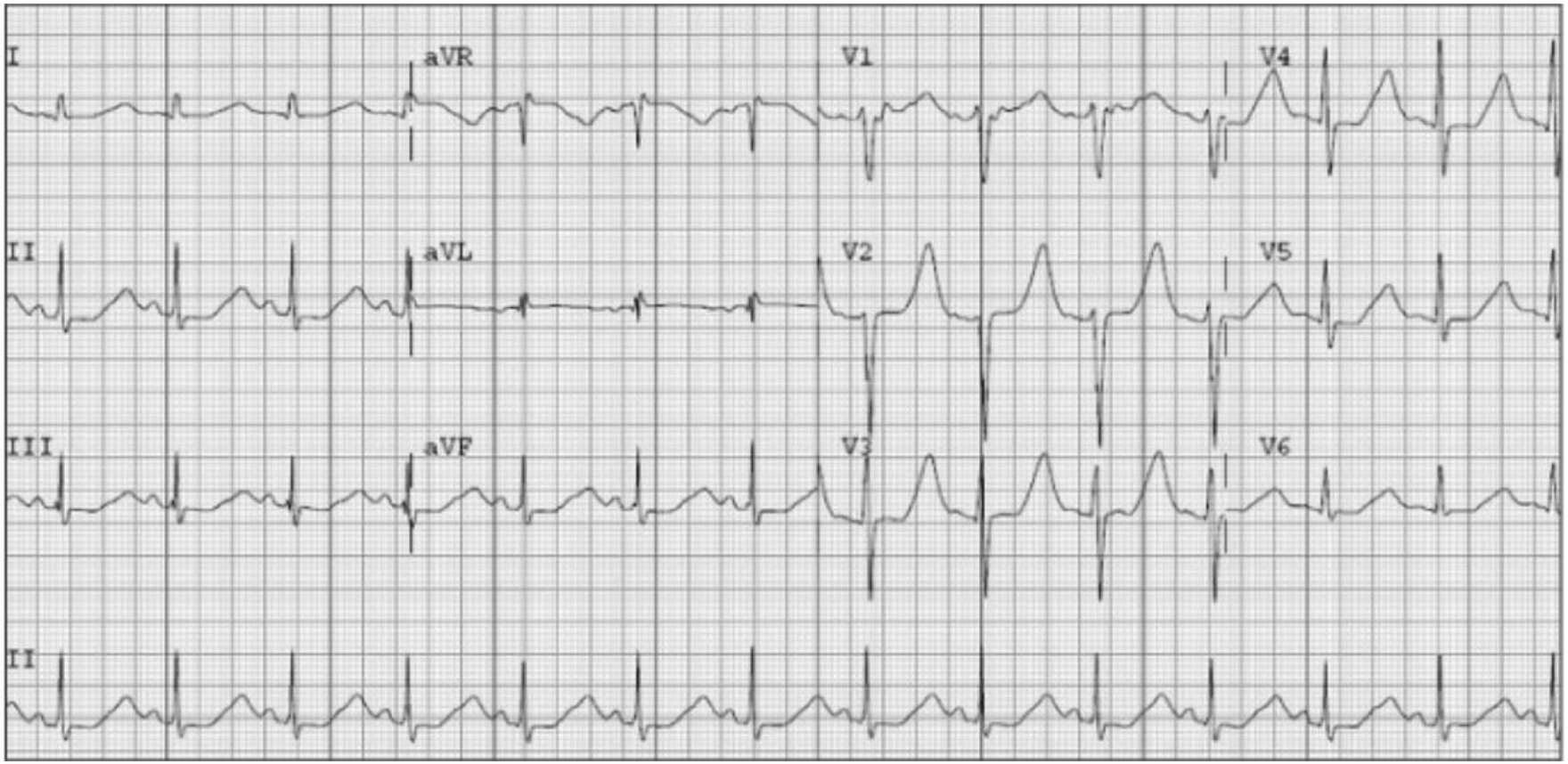




Digitalis Effect



Hypokalemia



Inverted T-waves

Primary T-wave inversion

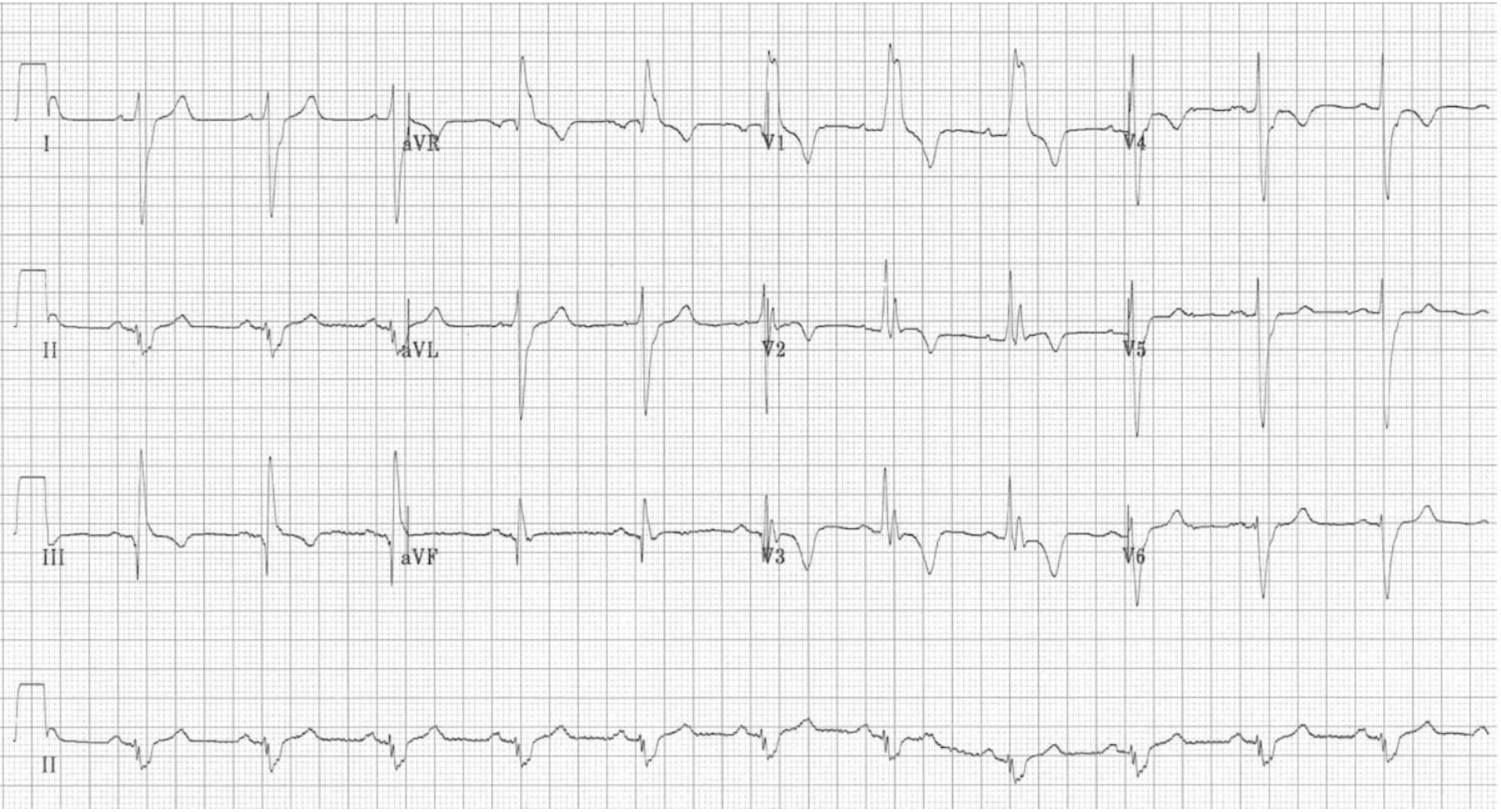
- Post myocardial ischemia
- Persistent juvenile TWI
- Contrast dye injection
- Intermittent BBB-memory
- Post pacing-memory
- Post tachycardia-memory
- Hypertrophy, LV (including apical), RV
- Pericarditis
- Cardiomyopathy
- Cerebral

Secondary Changes

- LBBB
- RBBB
- IBBB/IVCD
- PVC's
- WPW
- RV-strain: PE

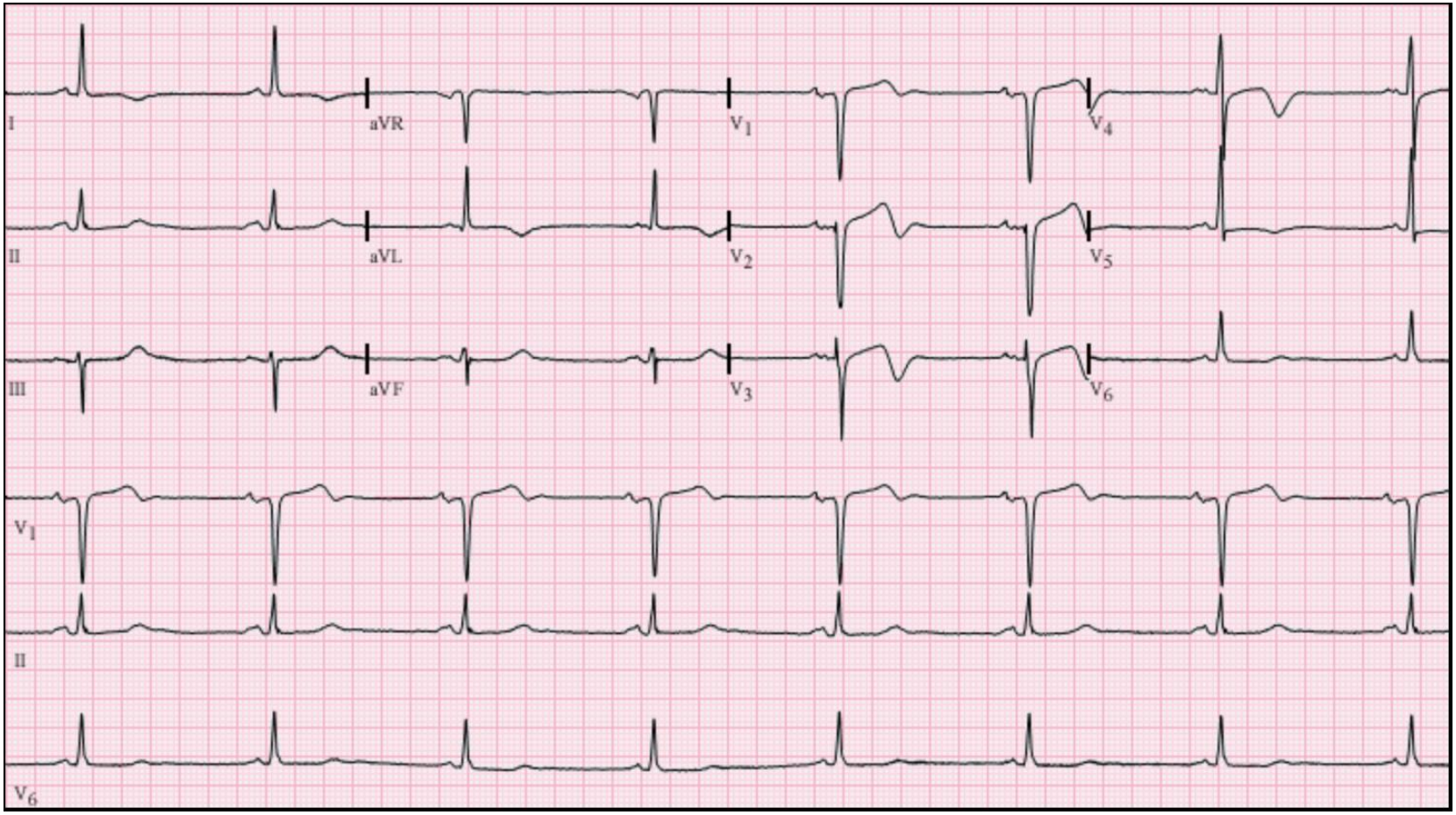
(And then there's pseudonormalization….)

Pulmonary Embolism redoux



(RBBB, RAD, “clockwise rotation” right precordial T-wave inversion, S1Q3T3)

Wellens Syndrome



So what are Non-specific ST- and T- Changes?

"NONSPECIFIC" ST AND T-WAVE CHANGES

Table 3

Possible Etiologies of One-Hundred and Six Cases with Nonspecific ST and T-Wave Abnormalities

	No known cause	ASHD	HBP 170/90 by history	HBP and ASHD	Anemia	Pulmonary embolus	CNS disease	Post-op. shock	Clinical pericarditis	Clinical cor pulmonale	Rheumatic valvular	Electrolytes	Total
T	25	2	6	1	2	1	4	2	1	0	1	0	45
ST and T	19	4	6	2	0	2	5	0	2	2	4	2	48
ST	5	2	2	2	0	0	1	1	0	0	0	0	13
Totals	49	8	14	5	2	3	10	3	3	2	5	2	106

ASHD, Arteriosclerotic (coronary) heart disease; HBP, hypertension; CNS, central nervous system.

Circulation, Volume XXIII, May 1961

ECG Changes
Associated With Prior Myocardial Infarction

- Any Q-wave in leads V2–V3 ≥0.02 sec or QS-complex in leads V2 and Vr.
- Q-wave ≥0.03 sec and ≥0.1 mV deep or QS complex in leads 1, II, aVL, aVF or V4–V6 in any two leads of a contiguous lead grouping (1, aVL; V1–V6; II, III, aVF).
- R wave ≥0.04 sec in V1–V2 and R/S ≥1 with a concordant positive T-wave in absence of conduction defect.

PLEASE……

- Use Troponin judiciously, for real symptoms, even marginal symptoms, but not for non-specific symptoms.
- Read the squiggly lines on the pink paper before labeling someone with a diagnosis that will change the course of their admission.
- Don't diagnose/admit/ a patient with NTEMI on the basis of one Troponin.
- Repeat Troponin in three hours and again next scheduled blood draw….
  - Look for a rise and fall
  - Look for one reading greater than 3 SD above a mean
    - Check your lab's assay for mean and standard deviation and make sure label on results accurately reflects the medical standard.
    - Detected but less than 3 SD above mean is negative, not "equivocal, borderline or indeterminant"
- Distinguish between myocardial injury and myocardial infarction despite the coders.
- Know a differential for a positive Troponin and consider an alternative diagnosis
  - Especially where there is no rise and fall
- Consider alternative means of confirming NSTEMI (RWMA by echo, nuclear scintigraphy) before ushering the patient to the cath lab for a stent.
- Remember the art of reading, interpreting and teaching EKG's.