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EKG in STEMI

Consideration of typical EKG patterns in STEMI and STEMI mimickers

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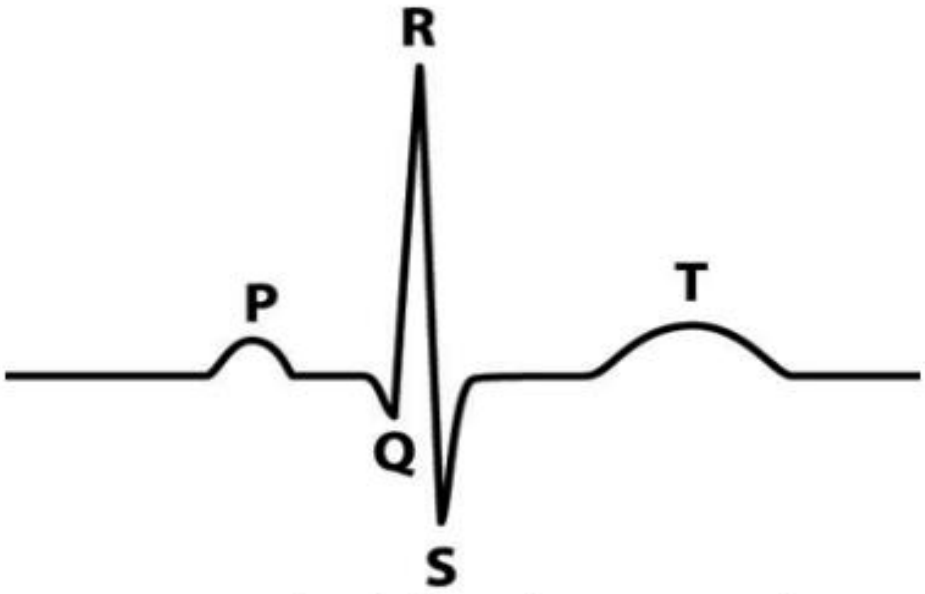
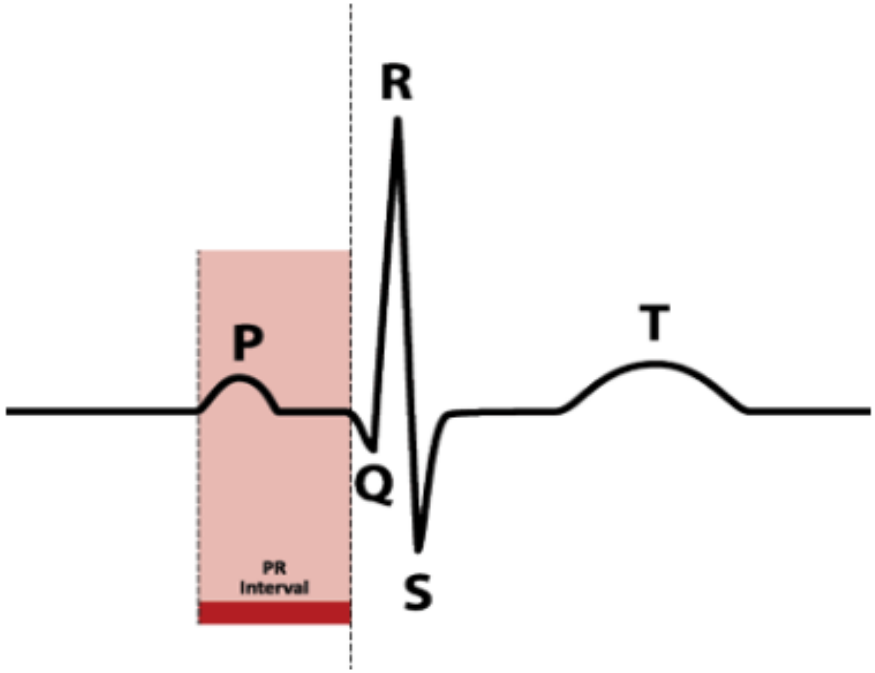
EKG Waveform



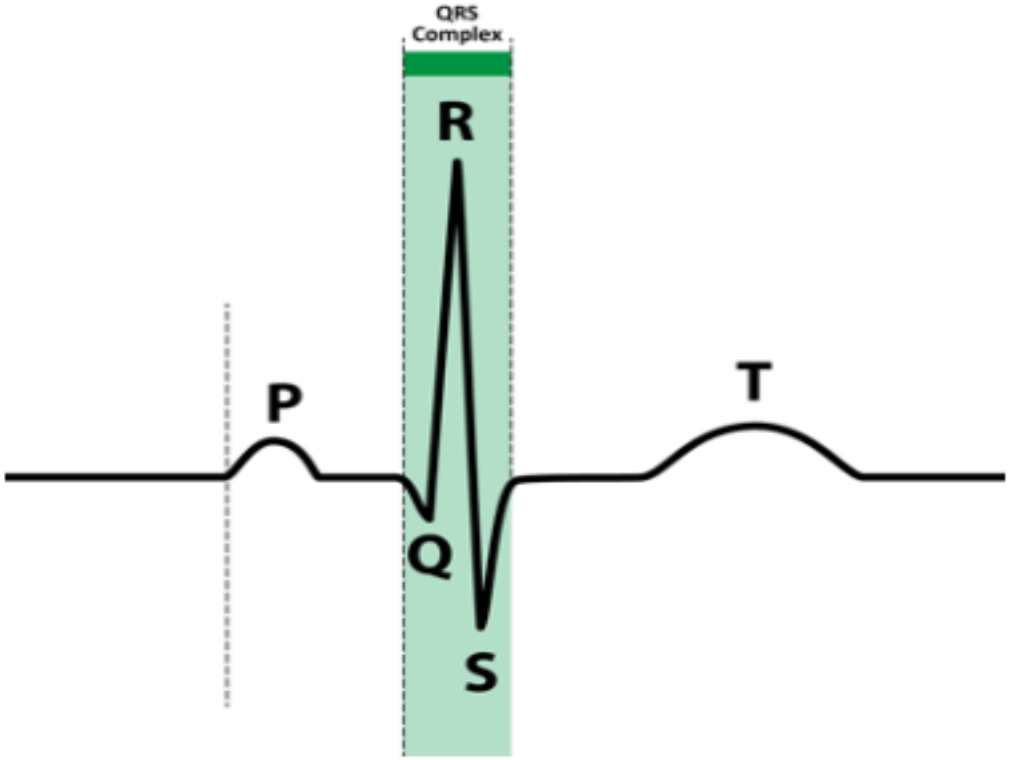
Letters are used to indicate important points on a typical waveform.

PR Interval



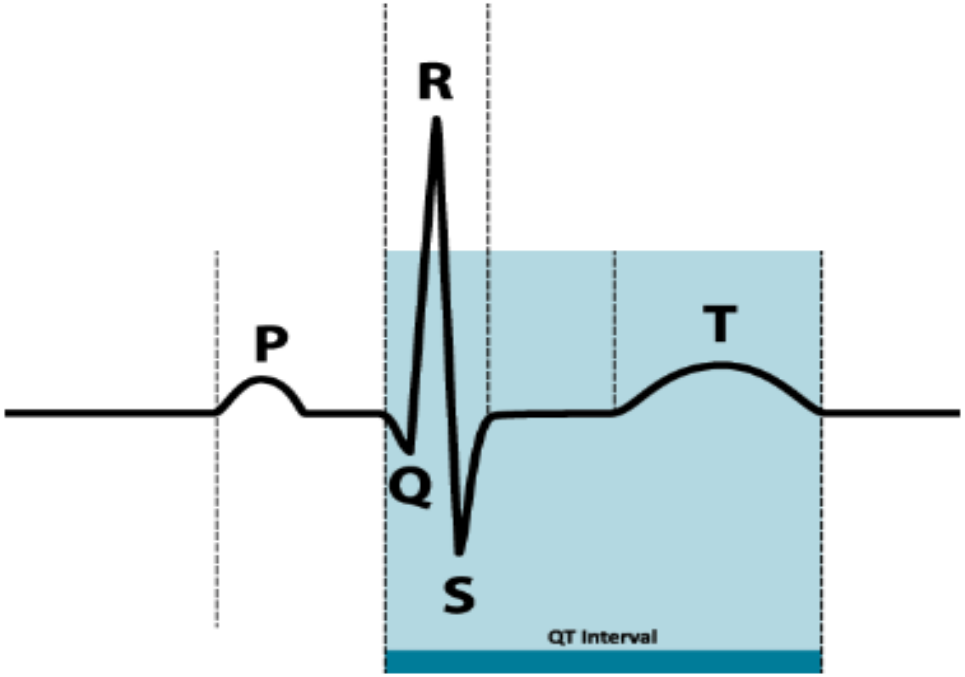
The PR Interval indicates atrioventricular conduction time. The interval is measured from where the P wave begins until the beginning of the QRS complex.

QRS Complex



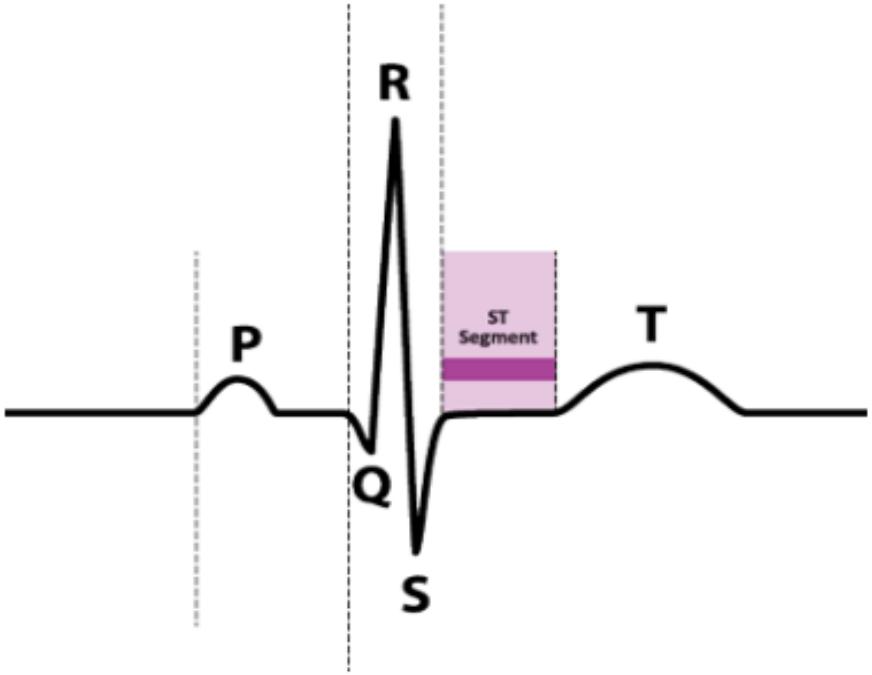
The QRS complex indicates ventricular depolarization. The QRS interval is measured from the end of the PR interval to the end of the S wave.

QT Interval

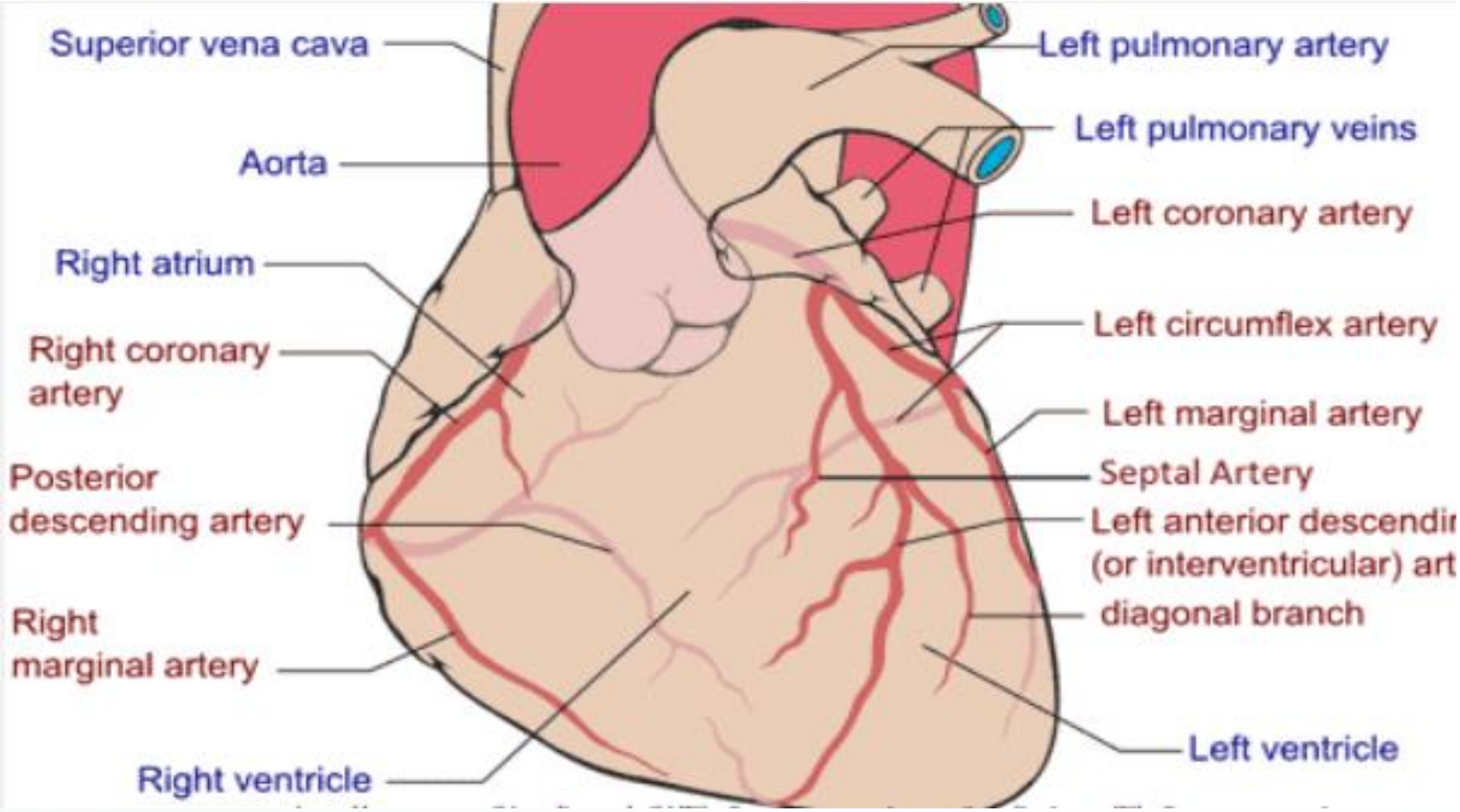


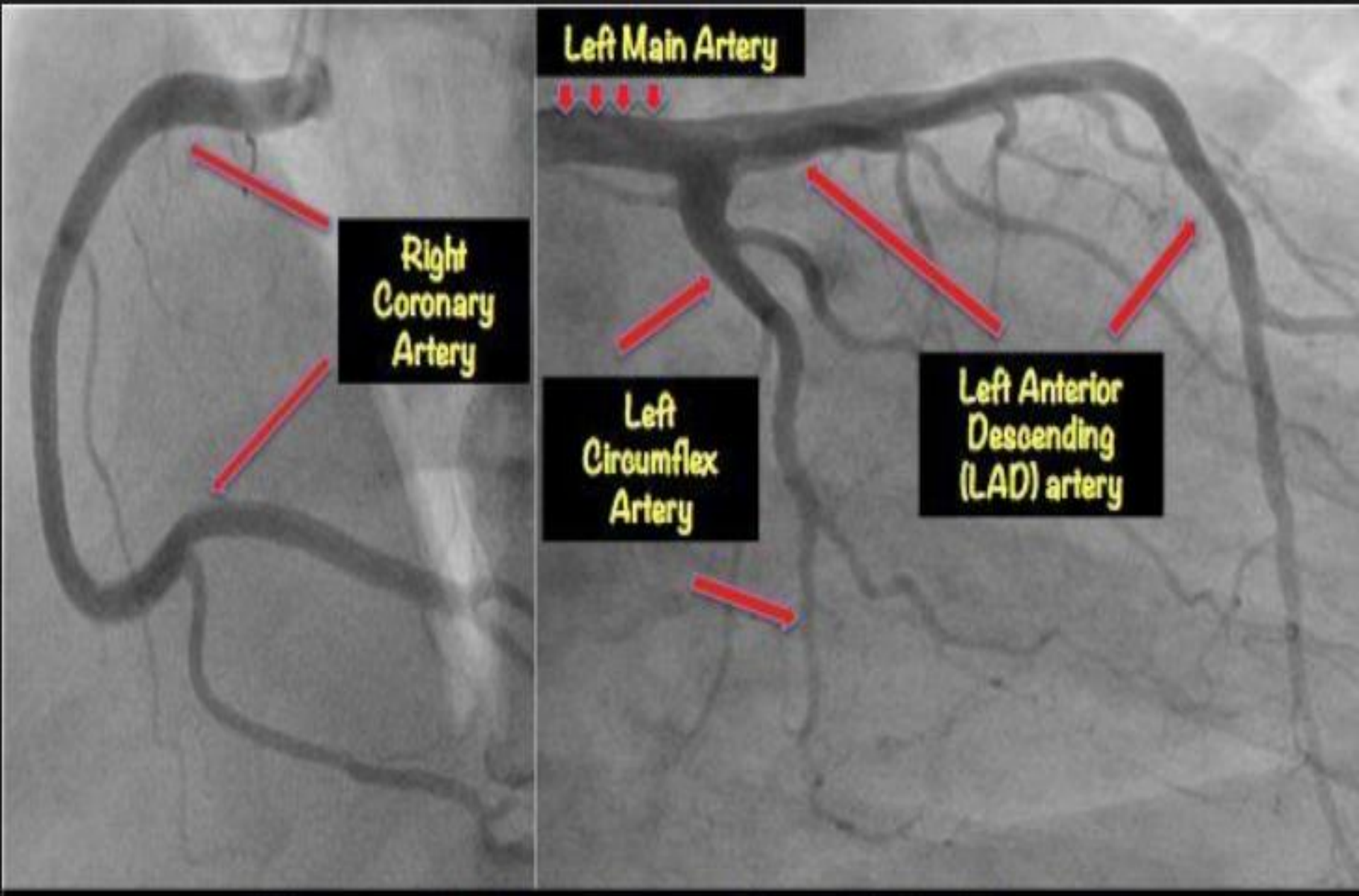
The QT interval indicates ventricular activity, both depolarization and repolarization. Measure the QT interval from the beginning of the QRS complex to the end of the T wave.

ST Interval

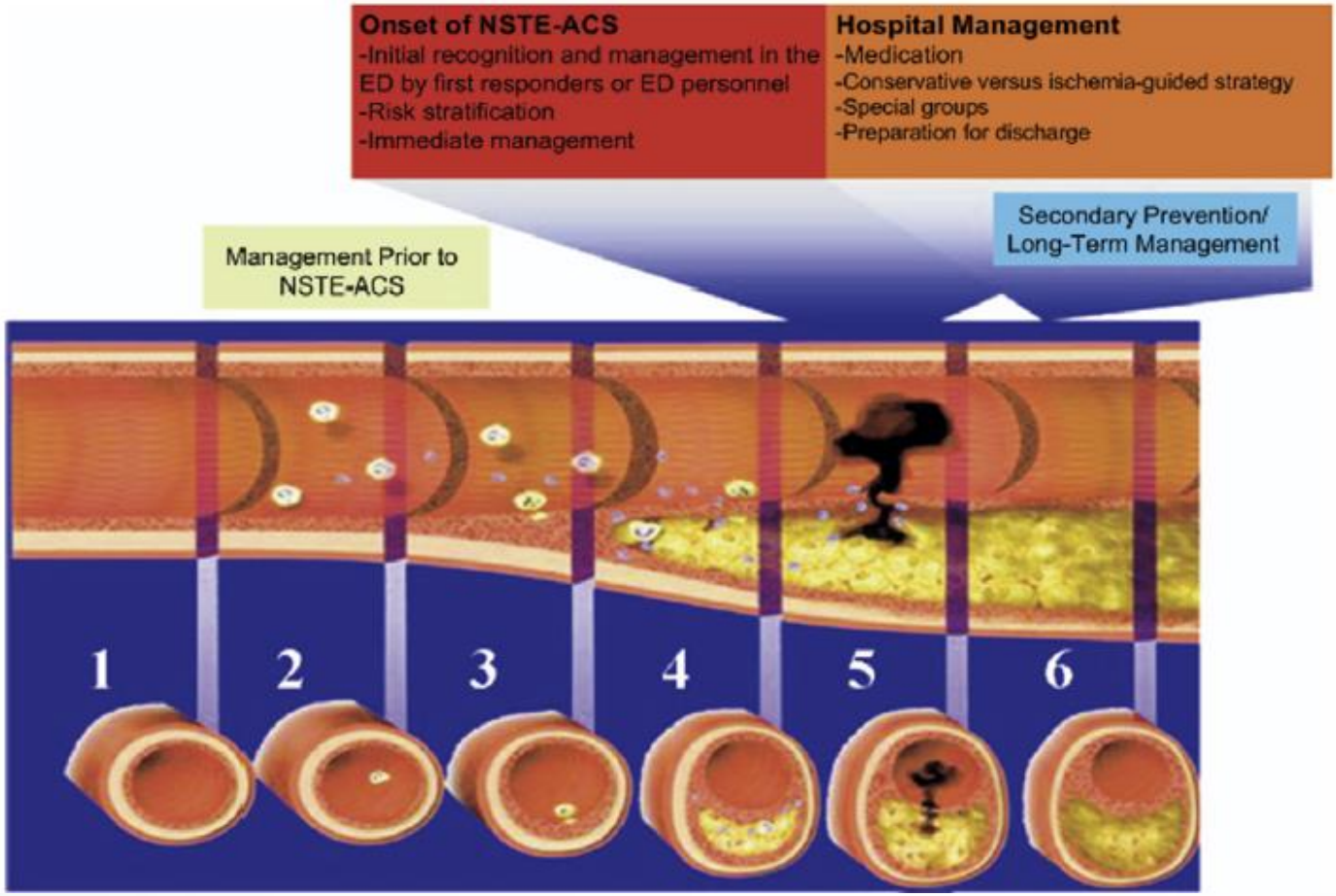


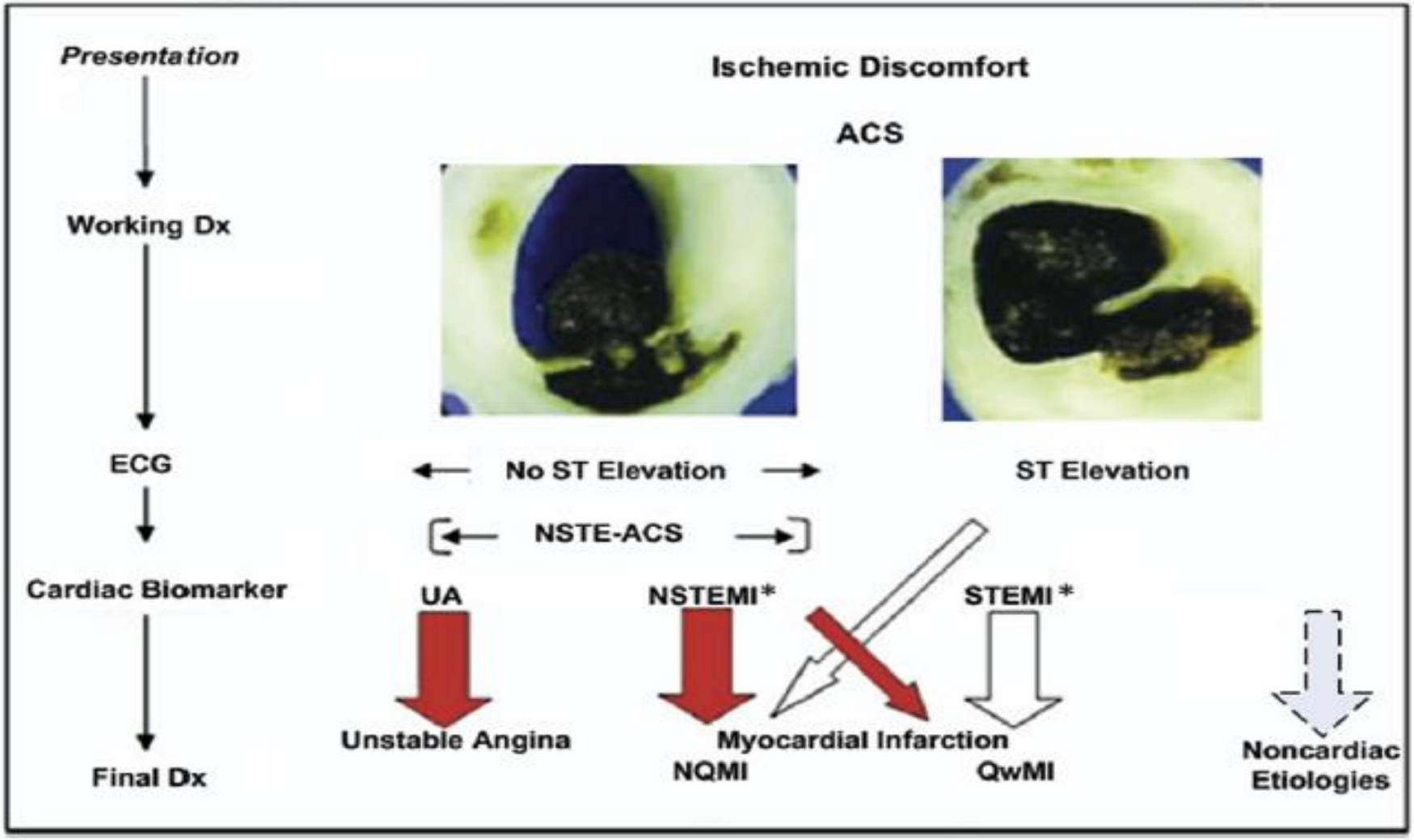
The ST segment traces the early part of ventricular repolarization. The ST segment begins at the end of the QRS complex and continues to beginning of the T wave.


Superior vena cava
Aorta
Right atrium
Right coronary artery
Posterior descending artery
Right marginal artery
Right ventricle
Left pulmonary artery
Left pulmonary veins
Left coronary artery
Left circumflex artery
Left marginal artery
Septal Artery
Left anterior descendi
(or interventricular) art
diagonal branch
Left ventricle



Normal blood supply of the heart



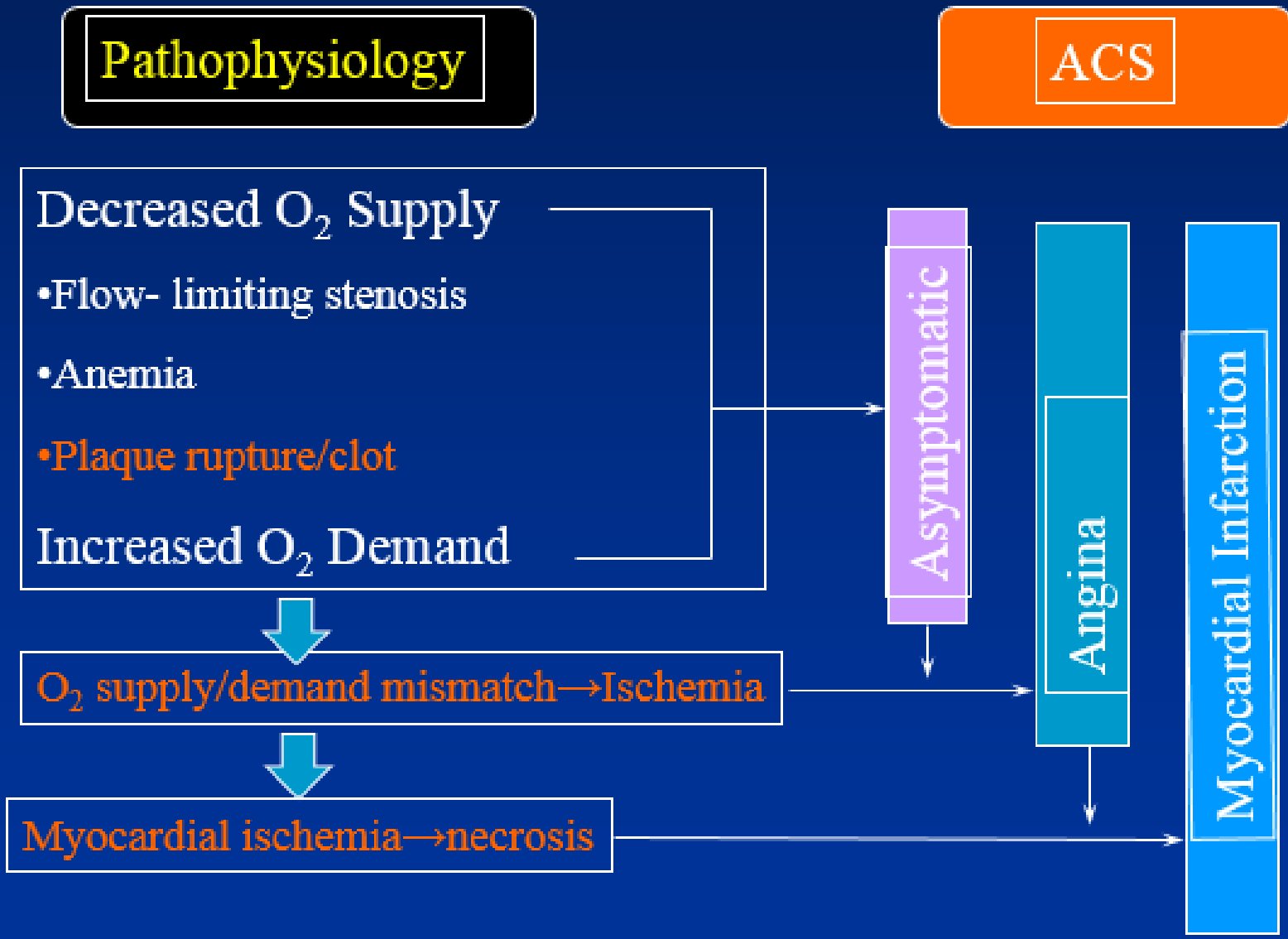


Pathogenesis of acute coronary syndrome

Hallmark of ACS is sudden imbalance between myocardial oxygen consumption and demand

- Usually result of coronary artery obstruction, MI
- Excessive O2 demand (SVT) in setting of noncritical CAD/tortuosity
- Vasospastic (Prinzmetal) angina
- Coronary embolism
- Coronary arteritis

Pathophysiology of Stable Angina and ACS



Pathogenesis of acute coronary syndrome

Noncoronary causes of myocardial oxygen supply demand mismatch :

- Hypotension
- Severe anemia
- Severe hypertension
- Tachycardia
- Hypertrophic cardiomyopathy
- Severe aortic stenosis

Pathogenesis of acute coronary syndrome

Non-ischemic/multifactorial etiologies

- Myocarditis
- Cardiac contusion
- Cardiotoxic drugs
- Takotsubo cardiomyopathy
- Pulmonary embolism
- Severe heart failure
- Sepsis

ORIGINAL ARTICLE ARCHIVE

Prevalence of Total Coronary Occlusion during the Early Hours of Transmural Myocardial Infarction

Marcus A. DeWood, M.D., Julie Spores, C.R.N.A., Robert Notske, M.D., Lowell T. Mouser, M.D., Robert Burroughs, M.D., Michael S. Golden, M.D., and Henry T. Lang, M.D.

N Engl J Med 1980; 303:897-902 | October 16, 1980 | DOI: 10.1056/NEJM198010163031601

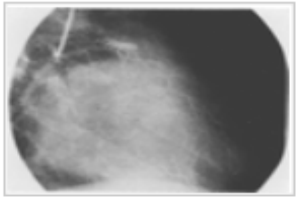
Abstract

To define the prevalence of total coronary occlusion in the hours after transmural myocardial infarction, we used coronary arteriography to study the degree of coronary obstruction in 322 patients admitted within 24 hours of infarction. Total coronary occlusion was observed in 110 of 126 patients (87 per cent) who were evaluated within four hours of the onset of symptoms; this proportion decreased significantly, to 37 of 57 (65 per cent), when patients were studied 12 to 24 hours after the onset of symptoms. Among 59 patients with angiographic features of coronary thrombosis, the thrombus was retrieved by Fogarty catheter in 52 (88 per cent) but was absent in seven (12 per cent false positive). Among an additional 20 patients without angiographic features of thrombosis, a thrombus was discovered in five (25 per cent false negative). Thus, total coronary occlusion is frequent during the early hours of transmural infarction and decreases in frequency during the initial 24 hours, suggesting that coronary spasm or thrombus formation with subsequent recanalization or both may be important in the evolution of infarction. (N Engl J Med. 1980; 303:897–902.)

Supported by the Michael I. Hanneman Memorial Research Fund and by a gift from the Sacred Heart and Deaconess Research Foundations.

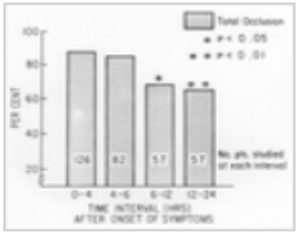
MEDIA IN THIS ARTICLE

FIGURE 1



angiographic Demonstration of Thrombus in a Patient with Anterior Transmural Myocardial Infarction Evaluated within Six Hours of the Onset of Chest Pain.

FIGURE 2



Frequency of Total Occlusion in Patient Groups Evaluated during Discrete Time Intervals over 24 Hours after Onset of Symptoms.

CLASS I

1. In patients with chest pain or other symptoms suggestive of ACS, a 12-lead ECG should be performed and evaluated for ischemic changes within 10 minutes of the patient's arrival at an emergency facility (21). *(Level of Evidence: C)*

2. If the initial ECG is not diagnostic but the patient remains symptomatic and there is a high clinical suspicion for ACS, serial ECGs (e.g., 15- to 30-minute intervals during the first hour) should be performed to detect ischemic changes. *(Level of Evidence: C)*

3.3.2.4. Electrocardiogram

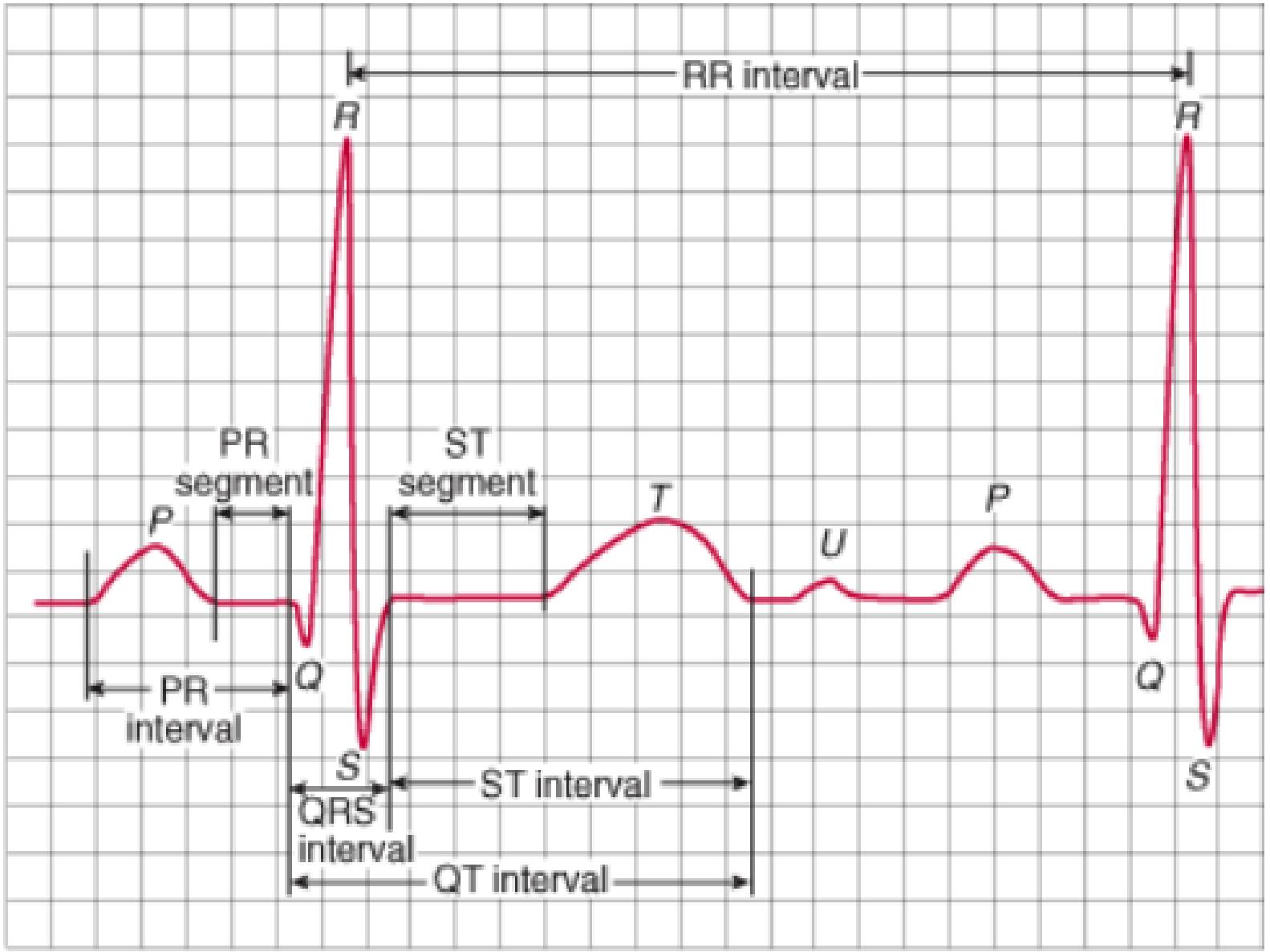
The 12-lead ECG is pivotal in the decision pathway for the evaluation and management of patients presenting with symptoms suggestive of ACS (58,59,85). Transient ST changes (≥0.5 mm [0.05 mV]) during symptoms at rest strongly suggest ischemia and underlying severe CAD.

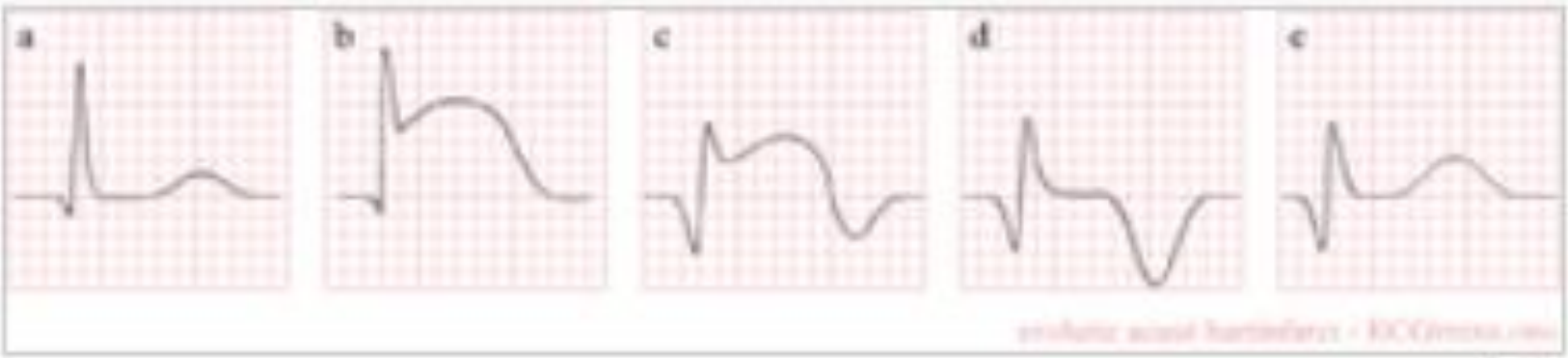
Differential diagnosis of NSTE-ACS includes (41):

- Nonischemic cardiovascular causes of chest pain (e.g., aortic dissection, expanding aortic aneurysm, pericarditis, pulmonary embolism)
- Noncardiovascular causes of chest, back, or upper abdominal discomfort include:
  - Pulmonary causes (e.g., pneumonia, pleuritis, pneumothorax)
  - Gastrointestinal causes (e.g., gastroesophageal reflux, esophageal spasm, peptic ulcer, pancreatitis, biliary disease)
  - Musculoskeletal causes (e.g., costochondritis, cervical radiculopathy)
  - Psychiatric disorders
  - Other etiologies (e.g., sickle cell crisis, herpes zoster)

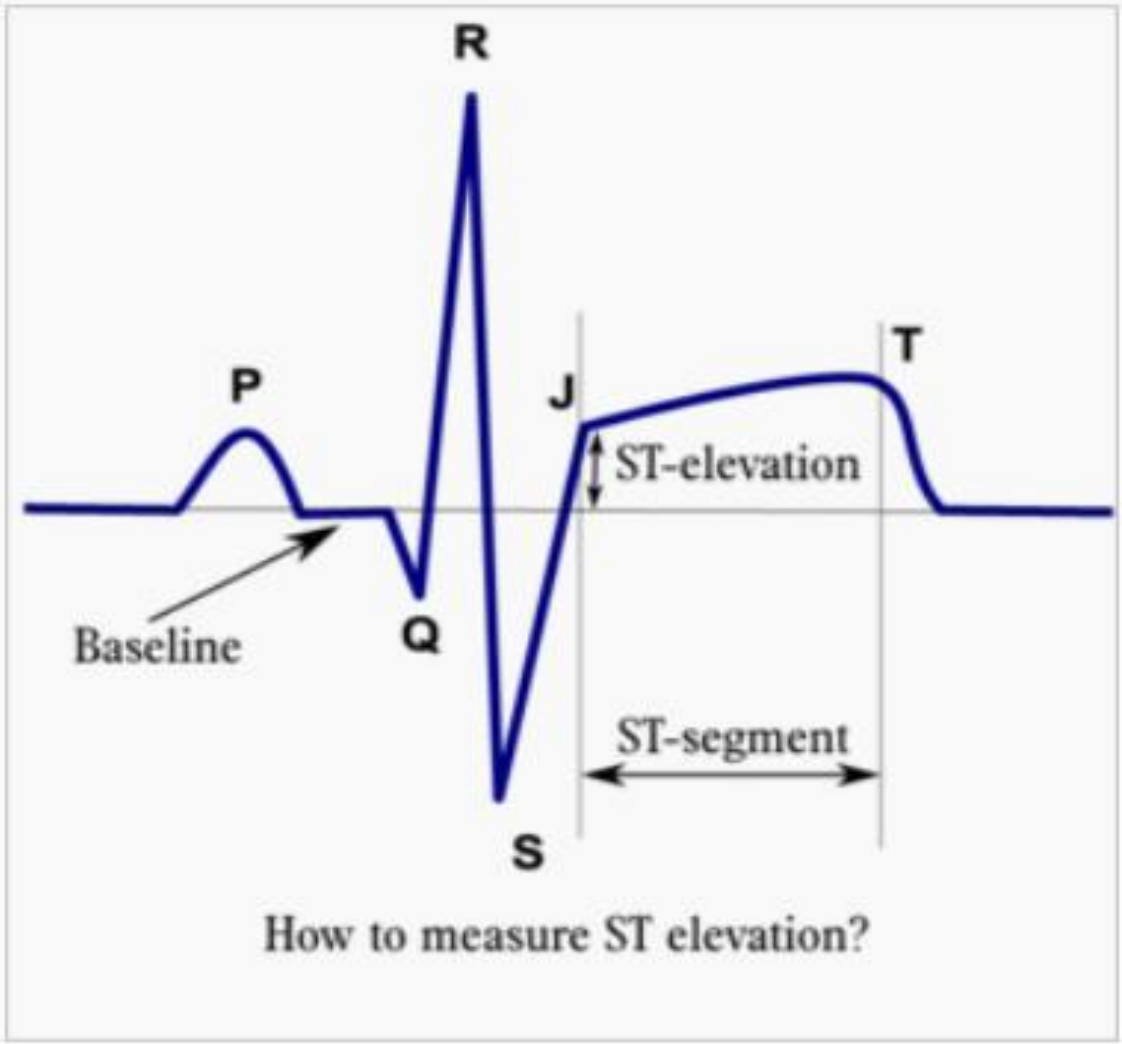
In addition, the clinician should differentiate NSTE-ACS from acute coronary insufficiency due to a nonatherosclerotic cause and noncoronary causes of myocardial oxygen supply-demand mismatch (41) (Section 2.2.2).

Alternative causes of ST-T changes include LV aneurysm, pericarditis, myocarditis, bundle-branch block, LV hypertrophy, hyperkalemia, Prinzmetal angina, early repolarization, apical LV ballooning syndrome (Takotsubo cardiomyopathy, Section 7.13), and Wolff-Parkinson-White conduction. Central nervous system events and therapy with tricyclic antidepressants or phenothiazines can cause deep T-wave inversion.


RR interval
R
R
PR segment
ST segment
P
T
U
P
PR interval
Q
Q
S
S
ST interval
QRS interval
QT interval



The evolution of an infarct on the ECG. ST elevation, Q wave formation, T wave inversion, normalisation with a persistent Q wave



ST elevation is measured at the junctional or J-point

Risk Stratification by ECG

- Simple, quick, noninvasive tool
- Universally available, cheap
- Correlates with risk and prognosis
- Guides treatment decisions
- Can identify alternative causes

Risk Stratification by ECG

- ECG Findings and Associated LR for AMI
  - New ST-E ≥ 1mm — LR 5.7-53.9
  - New Q waves — LR 5.3-24.8
  - Any ST-E — LR 11.2 (7.1-17.8)
  - New Conduction Defect — LR 6.3 (2.5-15.7)
  - New ST-D — LR 3.0-5.2
  - **NORMAL ECG** — **LR 0.1-0.4**

Panju AA. *JAMA.* 1998;280:1256.

Risk Stratification by ECG

CAVEATS

- **1-8% AMI have a normal ECG**

- **Only Approx 50%** of AMI patients have diagnostic changes on their *initial* ECG

Peter J. Zimetbaum, M.D., N Engl J Med 2003;348:933-40.

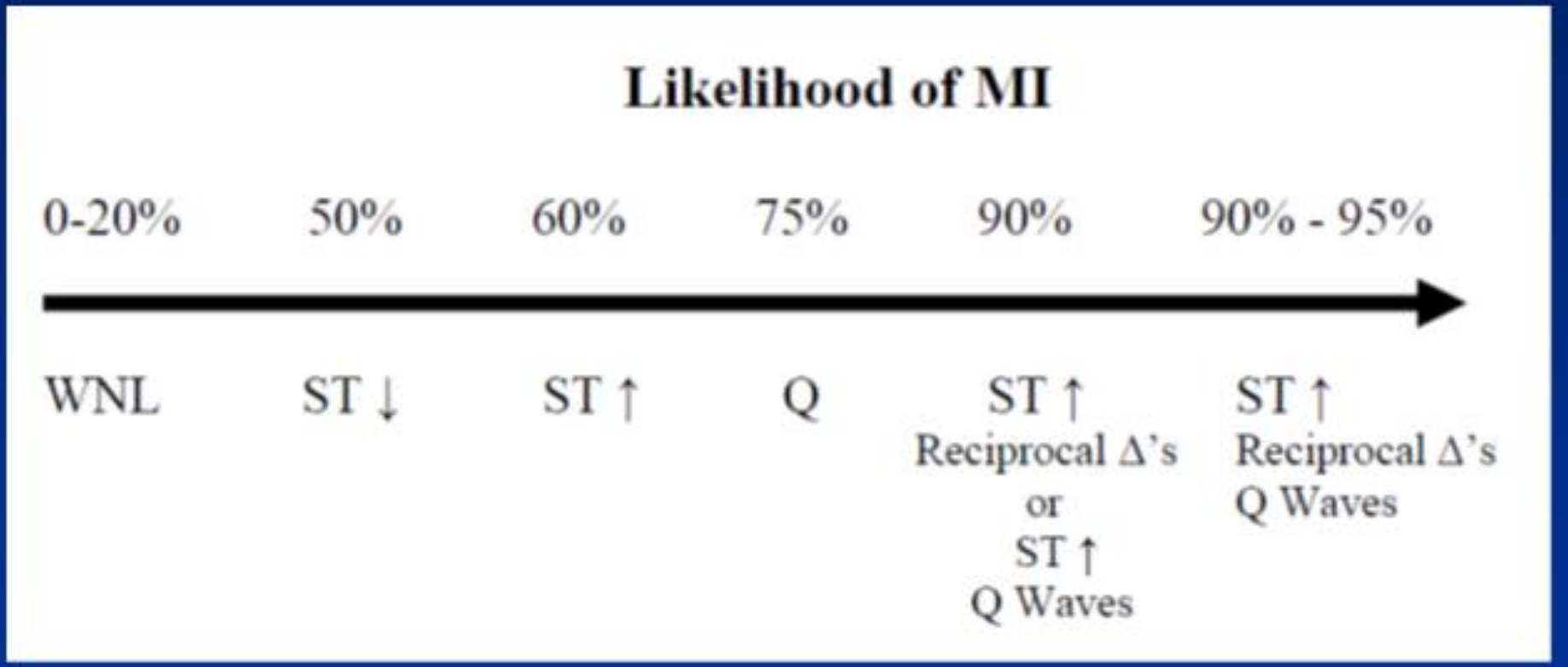
Risk Stratification by ECG

CAVEATS cont.

- 1 ECG cannot exclude AMI
  - Brief sample of a dynamic process
- Small regions of ischemia or infarction may be missed

Peter J. Zimetbaum, M.D., N Engl J Med 2003;348:933-40.

How Sensitive is the ECG Alone?



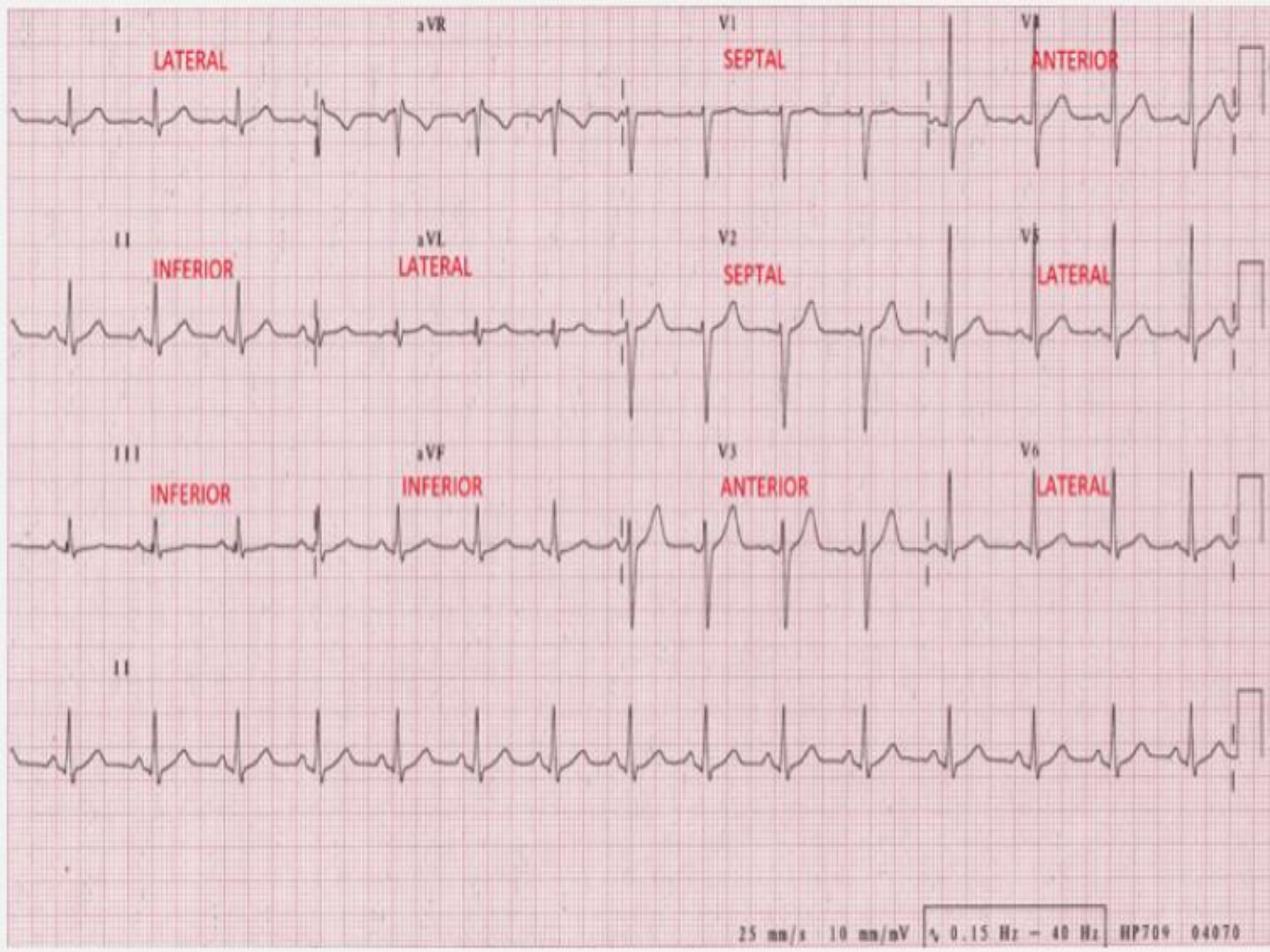
STEMI – EKG CRITERIA

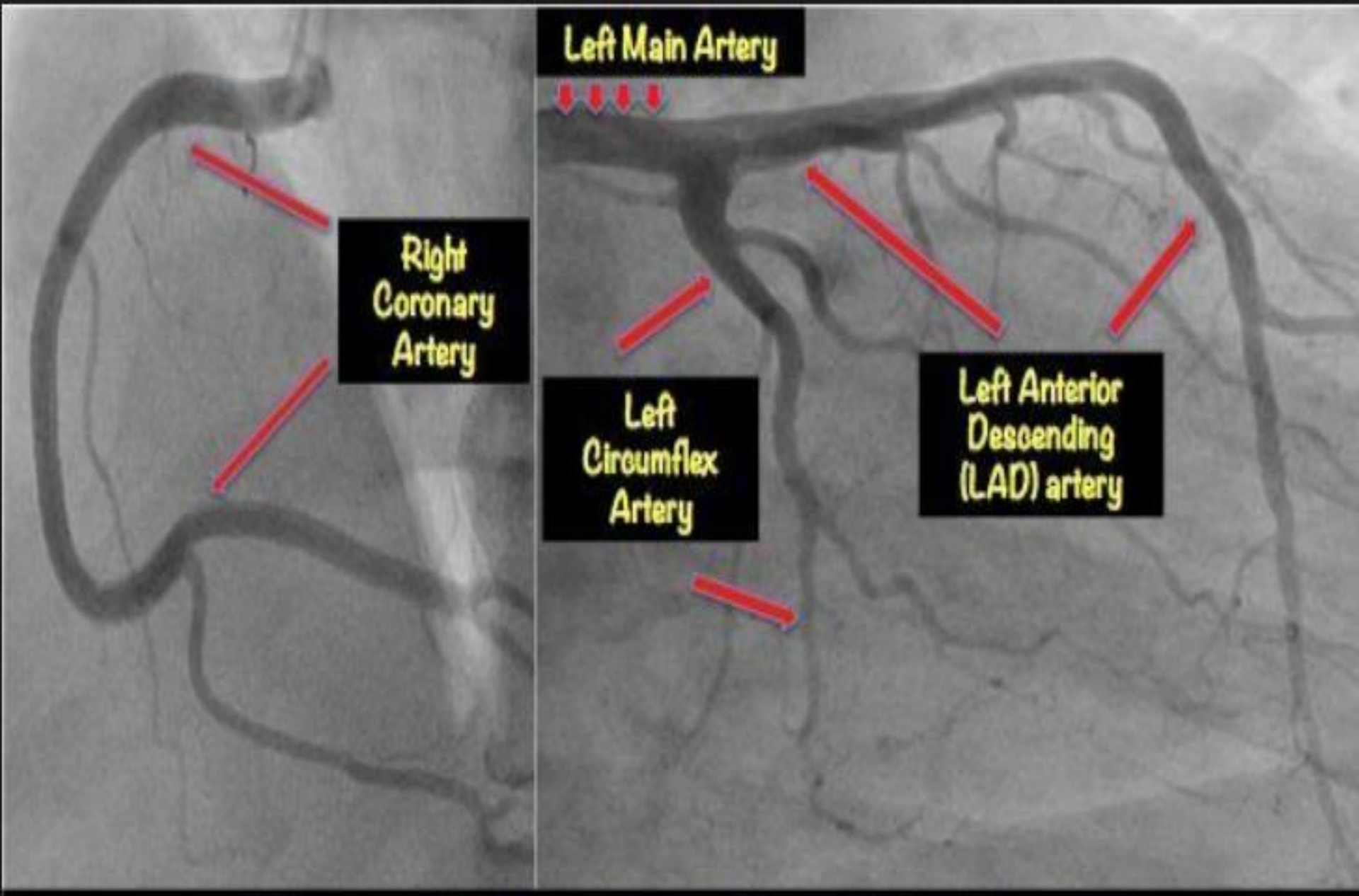
- Diagnostic elevation (in absence of LVH and LBBB) defined as:

- New ST elevation at J point in at least 2 contiguous leads

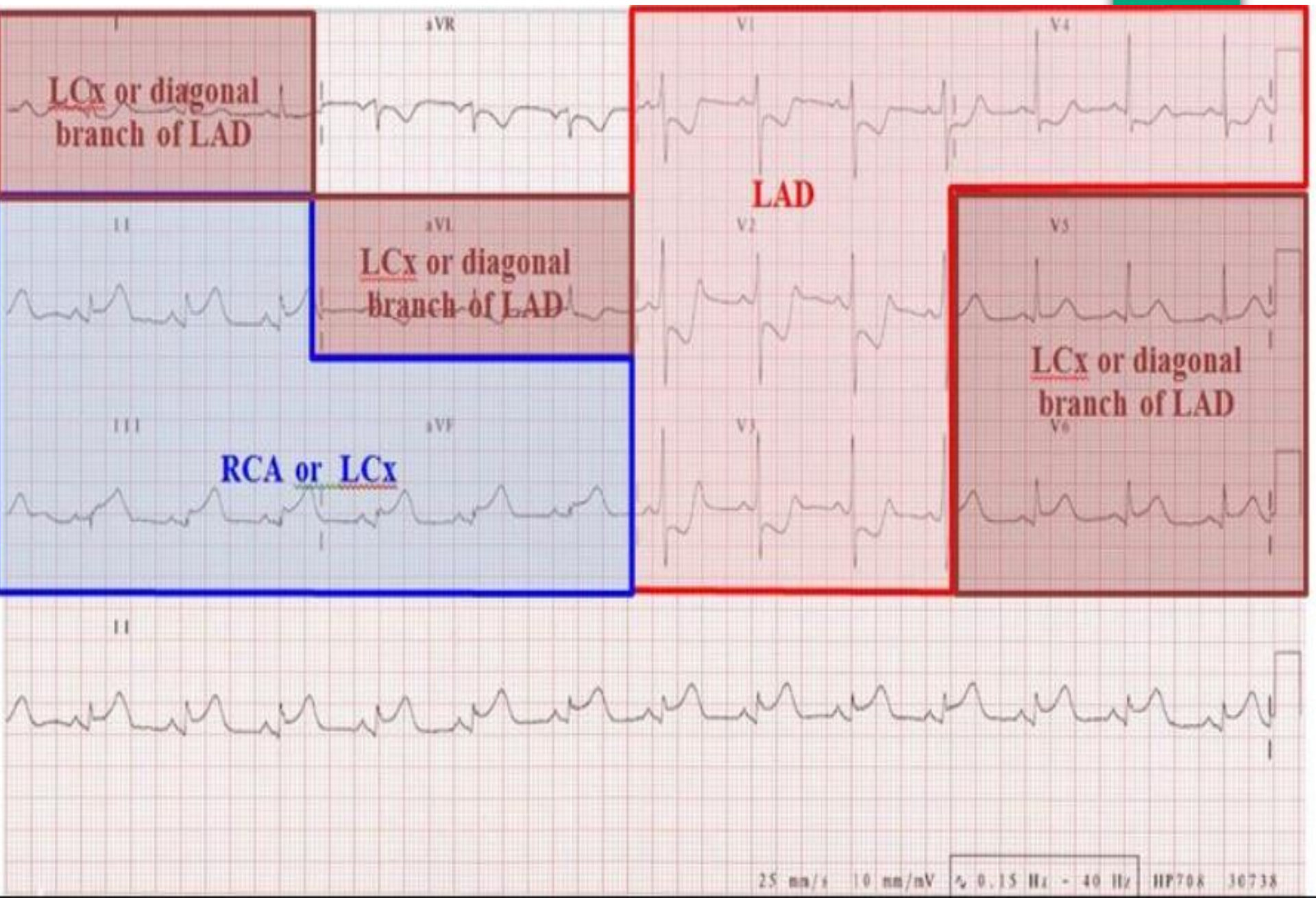
-in leads V2-V3, men >2mm, women > 1.5mm

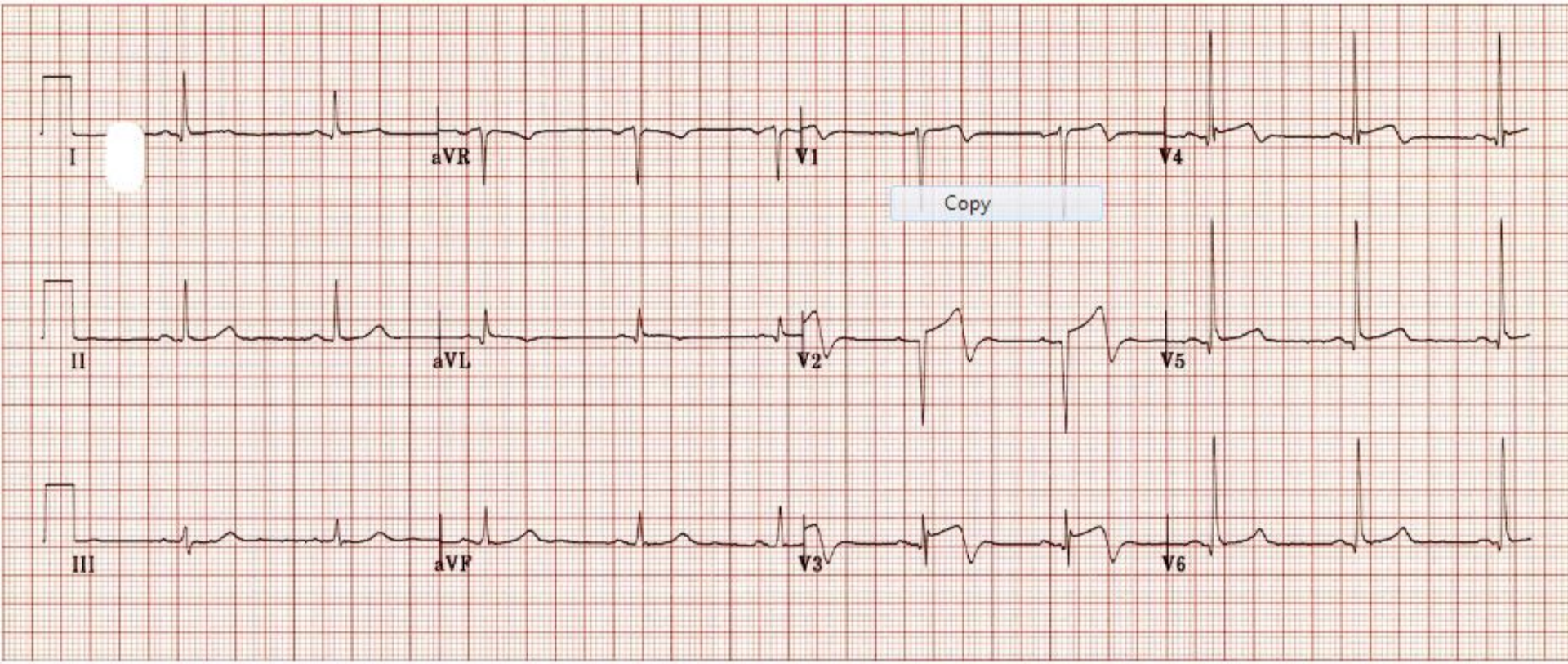
-in other chest leads or limb leads, > 1mm

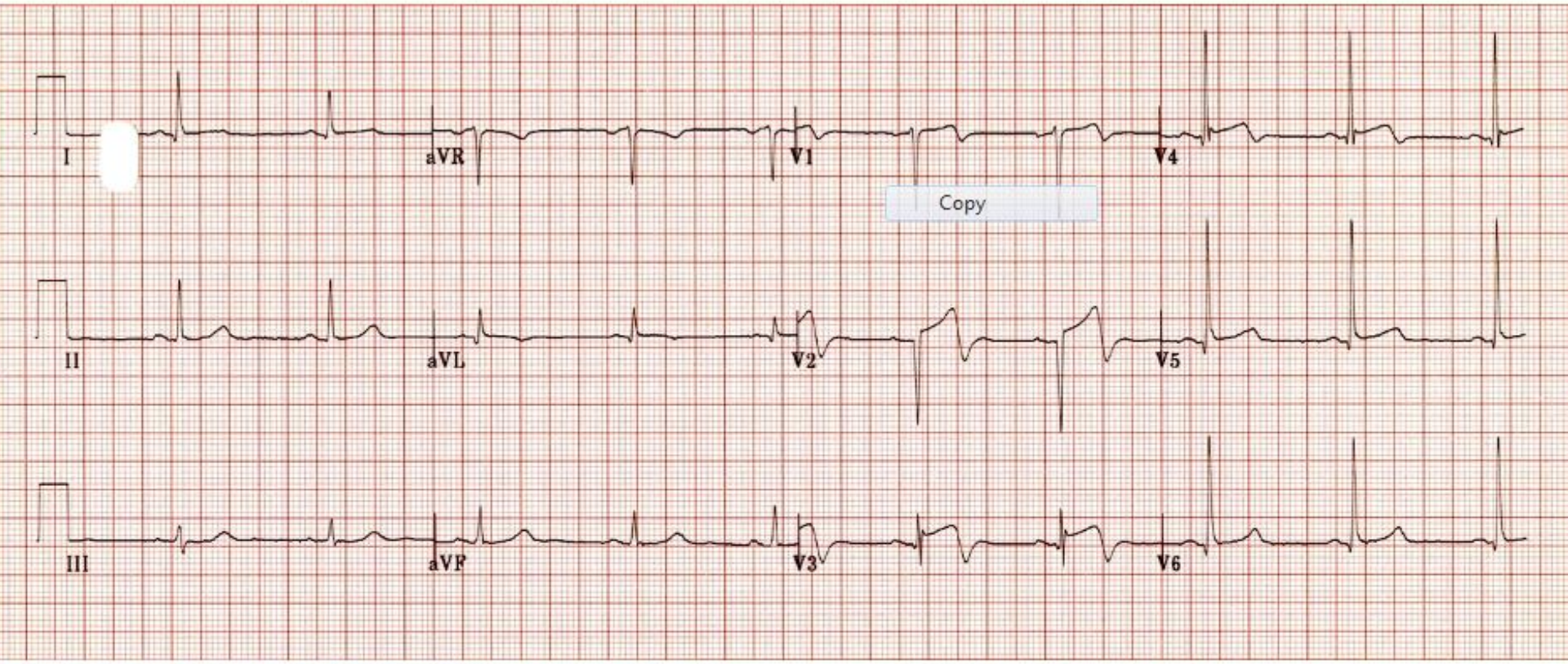

I
LATERAL
aVR
V1
SEPTAL
V4
ANTERIOR
II
INFERIOR
aVL
LATERAL
V2
SEPTAL
V5
LATERAL
III
INFERIOR
aVF
INFERIOR
V3
ANTERIOR
V6
LATERAL
II
25 mm/s 10 mm/mV 0.15 Hz - 40 Hz HP709 04070



Normal blood supply of the heart

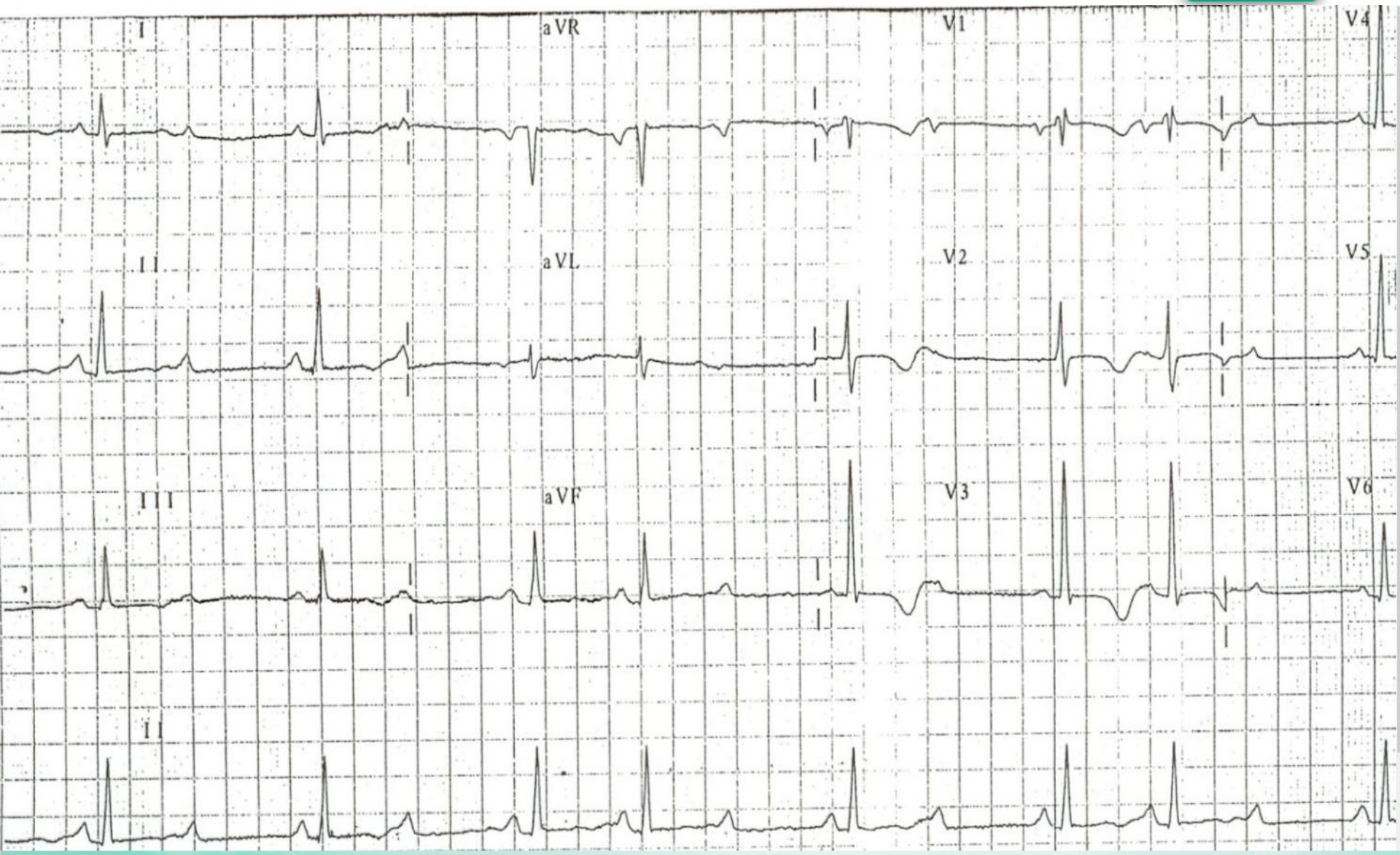

I
aVR
V1
V4
LCx or diagonal branch of LAD
LAD
II
aVL
V2
V5
LCx or diagonal branch of LAD
LCx or diagonal branch of LAD
III
aVF
V3
V6
RCA or LCx
II
25 mm/s
10 mm/mV
0.15 Hz - 40 Hz
HP708
30738


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



Wellens' Syndrome.

Essentially it's an ECG abnormality strongly associated with significant left anterior descending coronary artery stenosis.


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

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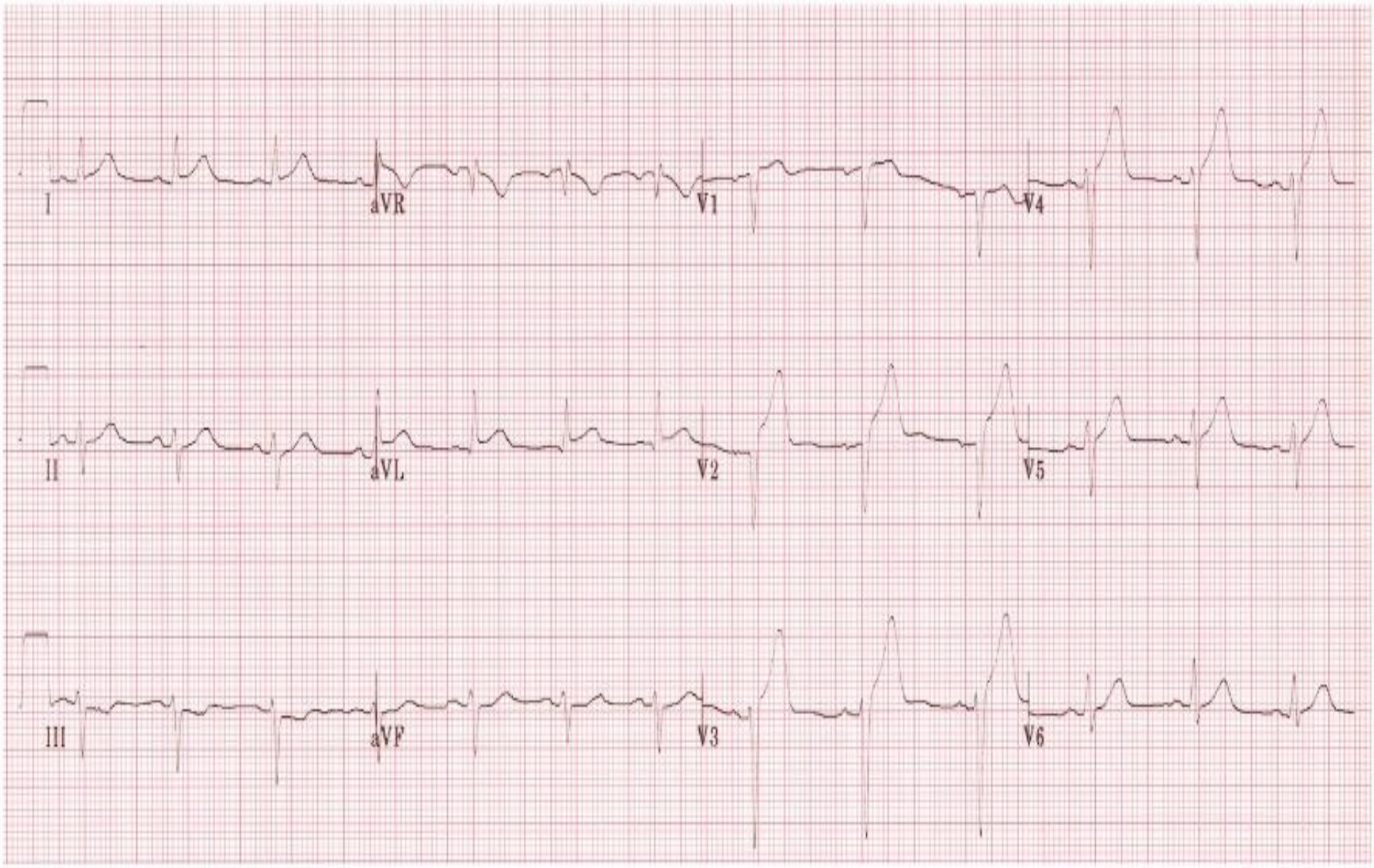
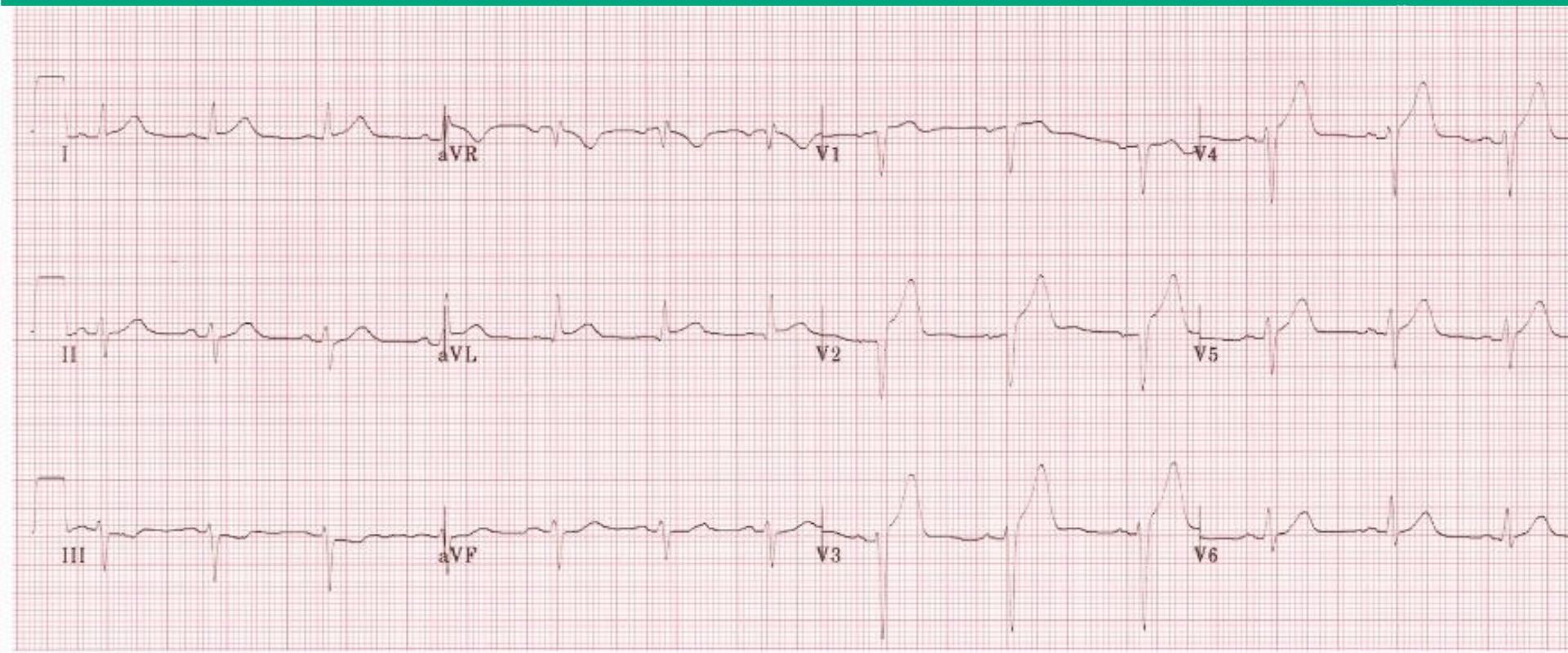
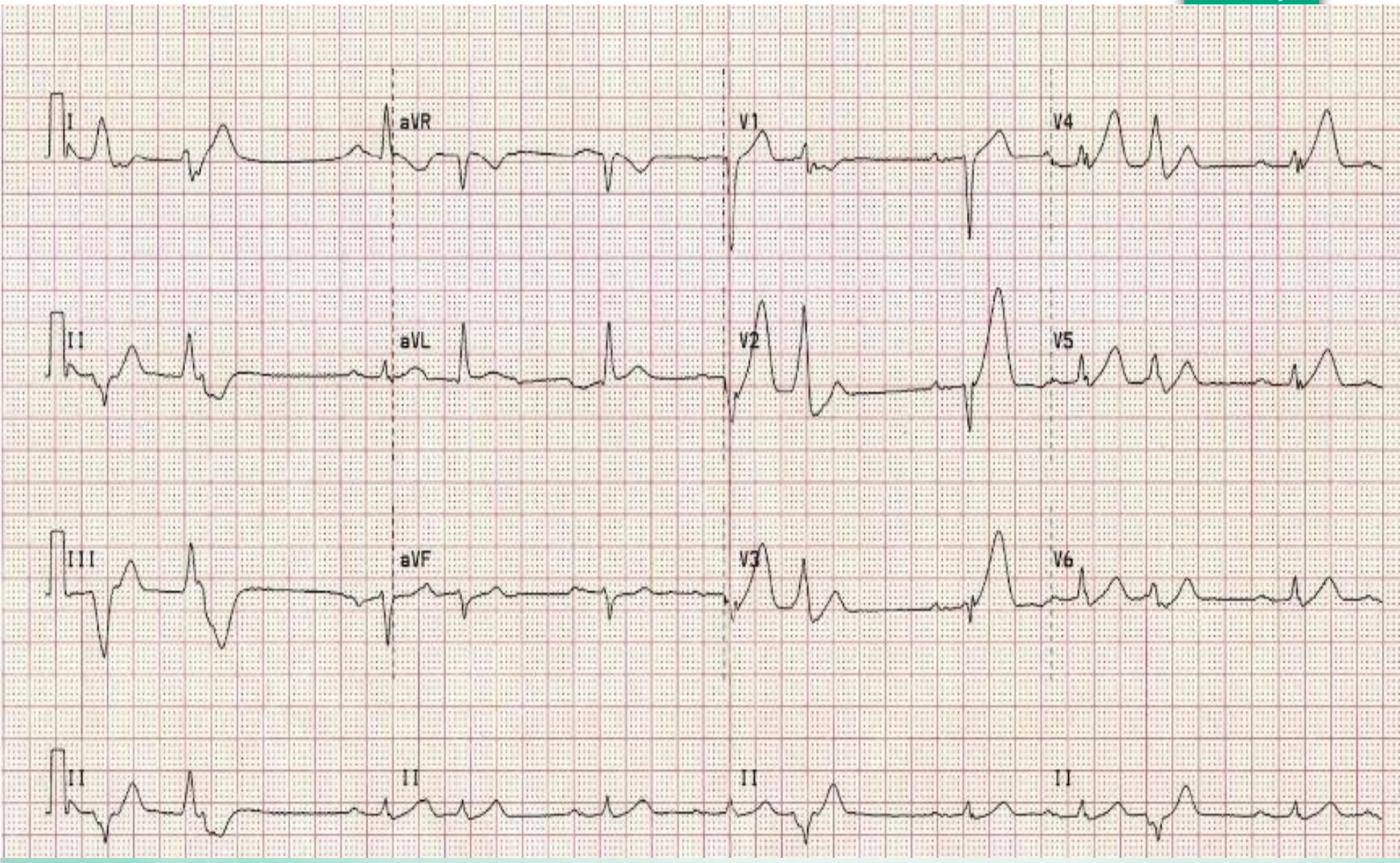
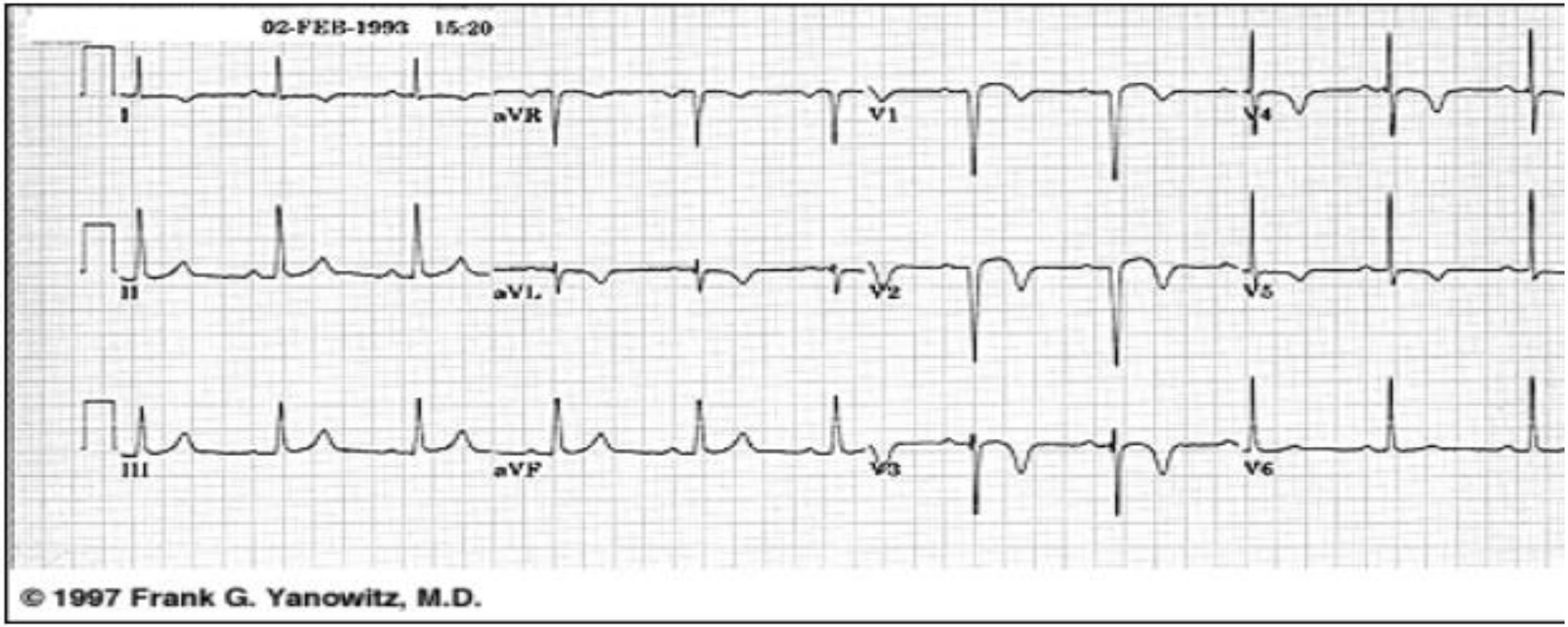




Hyperacute Anteroseptal STEMI

- ST elevation is maximal in the anteroseptal leads (V1-4).
- Q waves are present in the septal leads (V1-2).
- There is also some subtle STE in I, aVL and V5, with reciprocal ST depression in lead III.
- There are hyperacute (peaked) T waves in V2-4.
- These features indicate a hyperacute anteroseptal STEMI


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



Anteroseptal MI: Fully Evolved-KH

Frank G.Yanowitz, M.D.

The QS complexes, resolving ST segment elevation and T wave inversions in V1-2 are evidence for a fully evolved anteroseptal MI. The inverted T waves in V3-5, I, aVL are also probably related to the MI.

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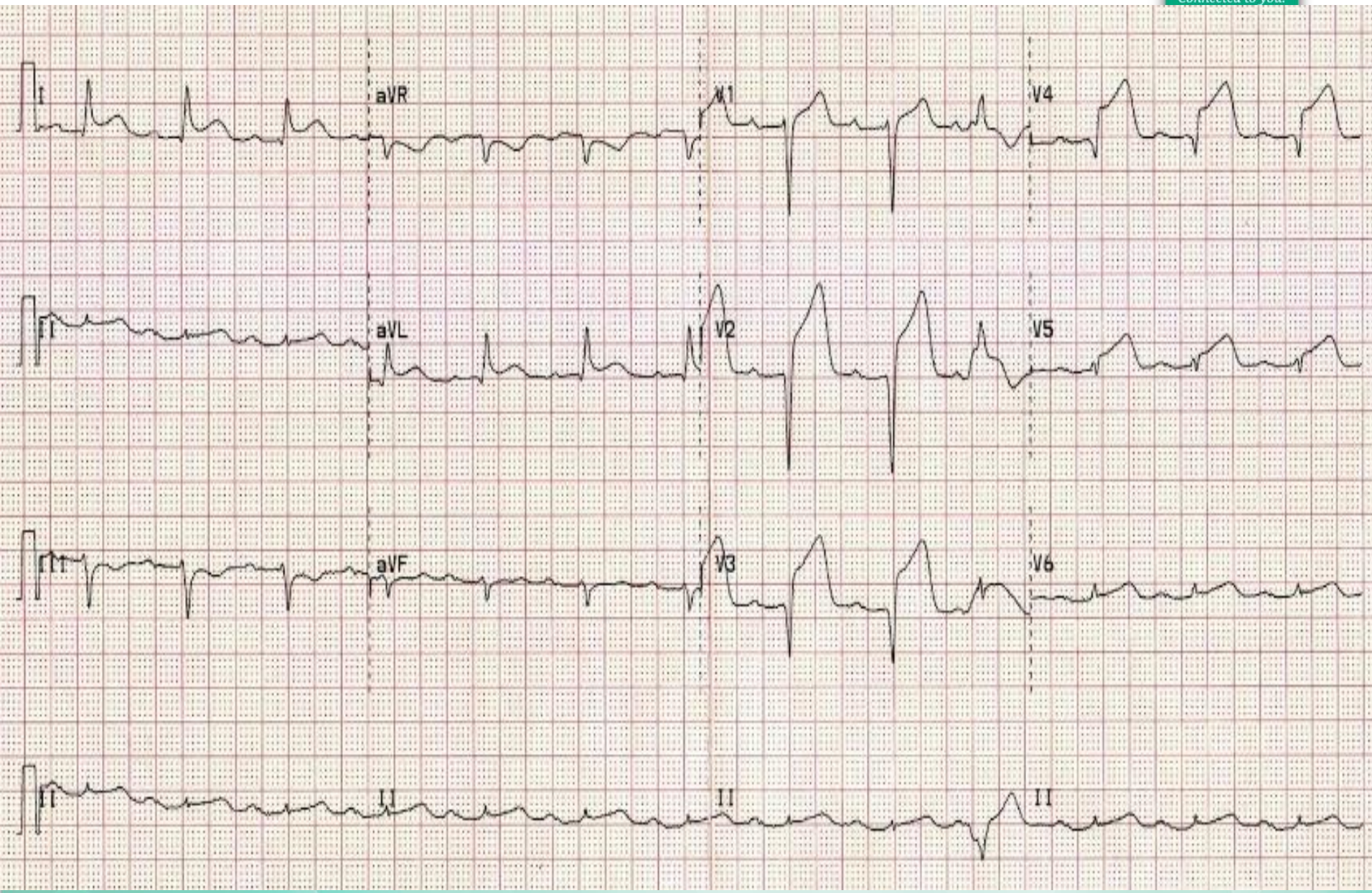

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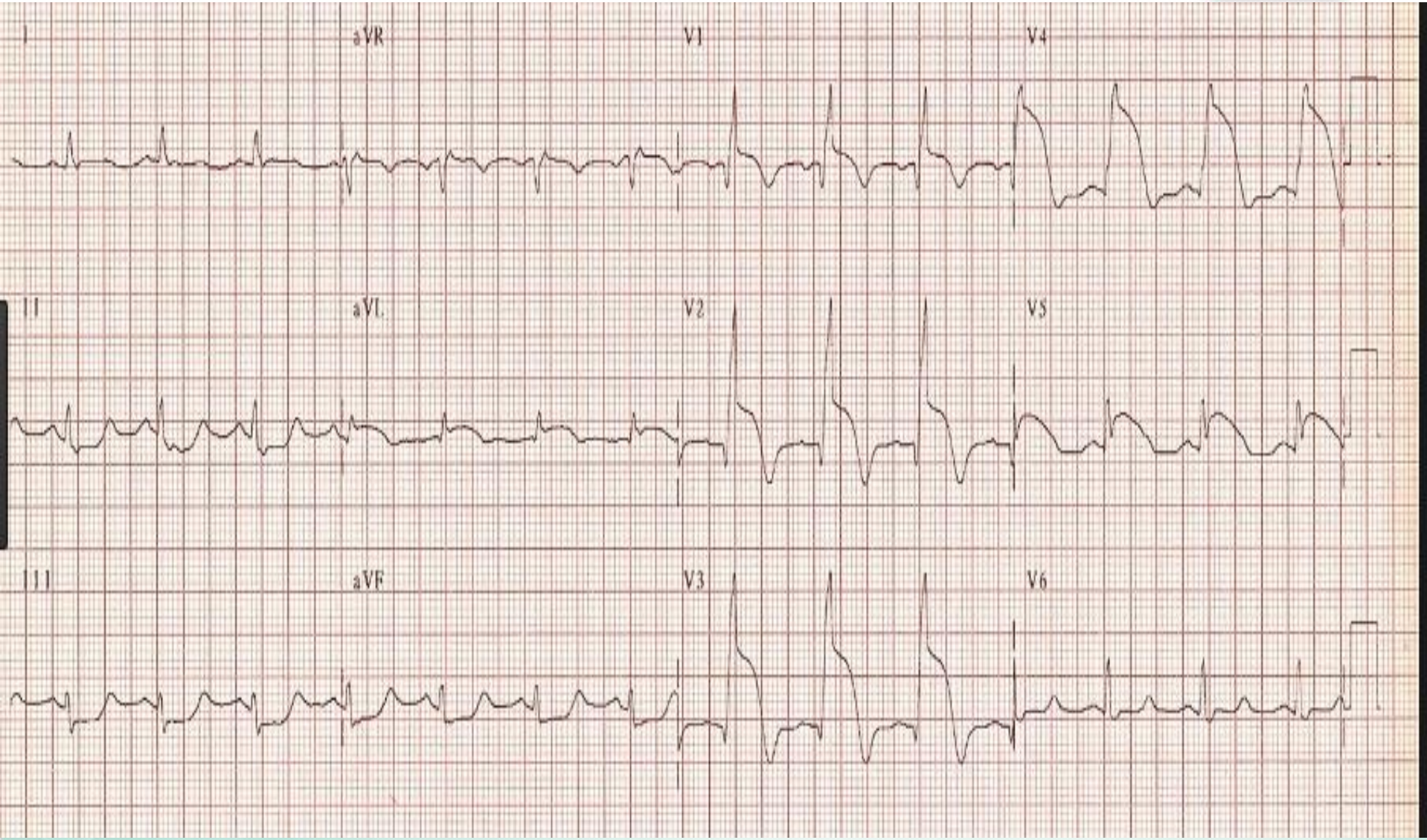
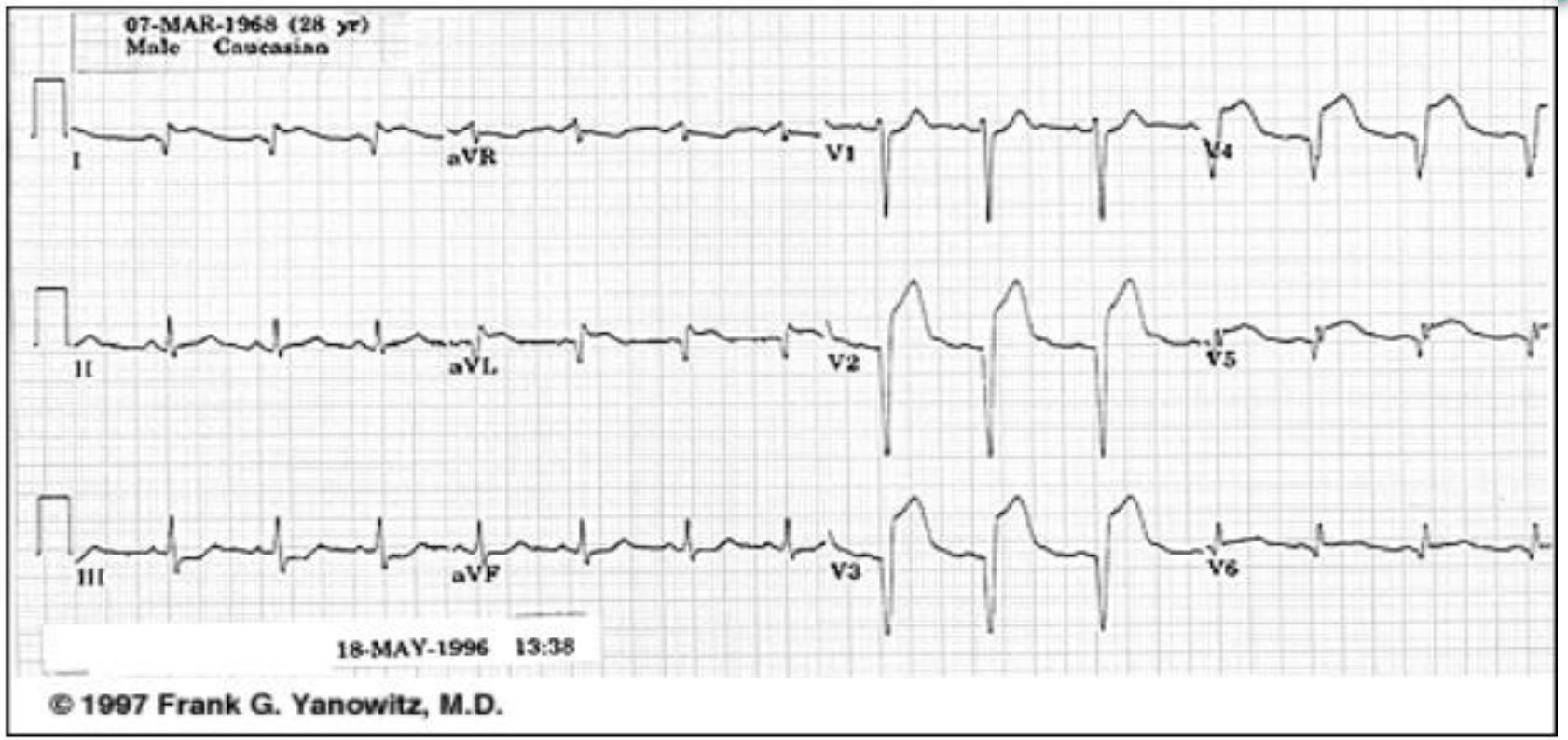
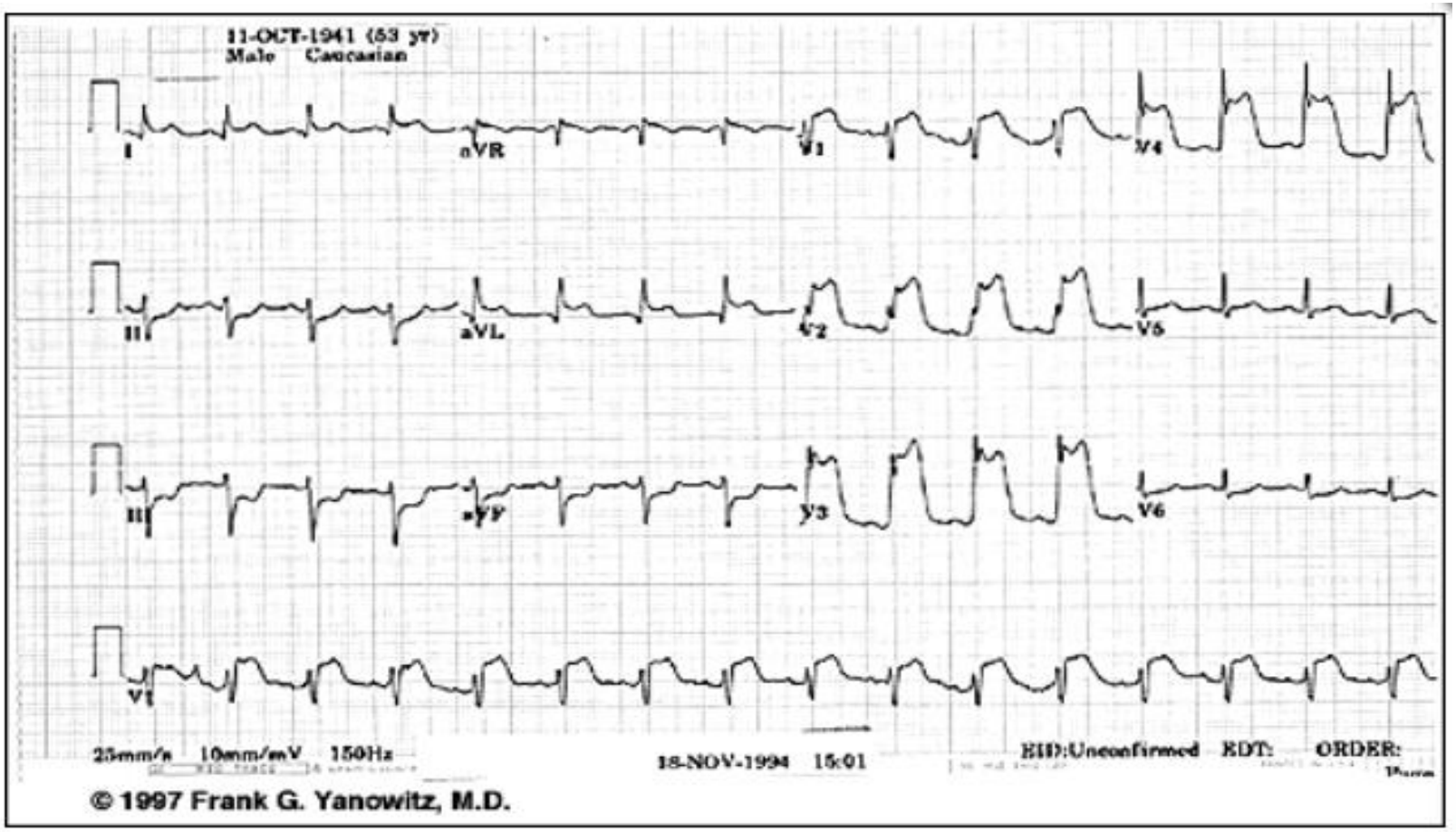


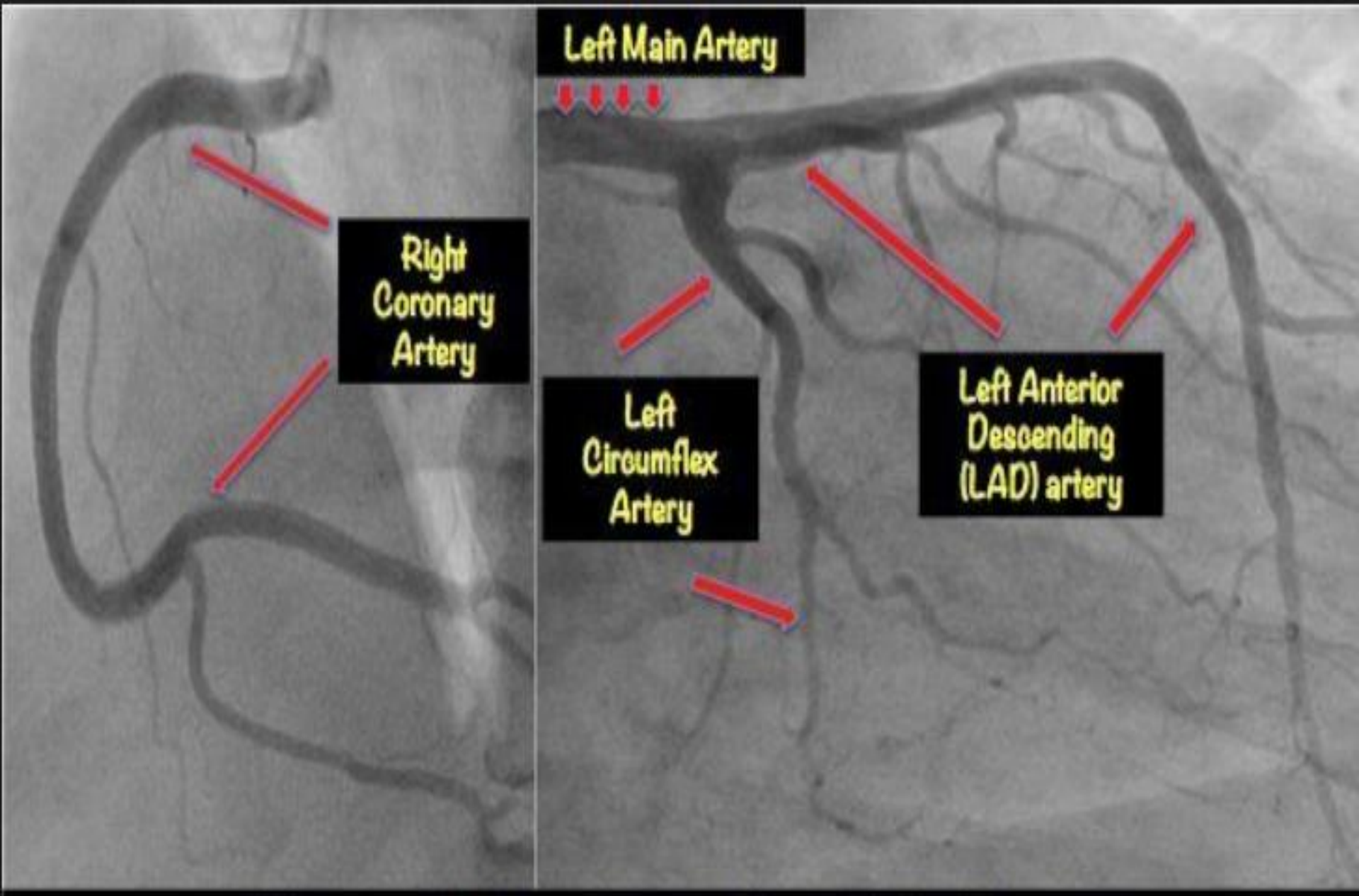


Extensive Anterior/Anterolateral MI: Recent-KH

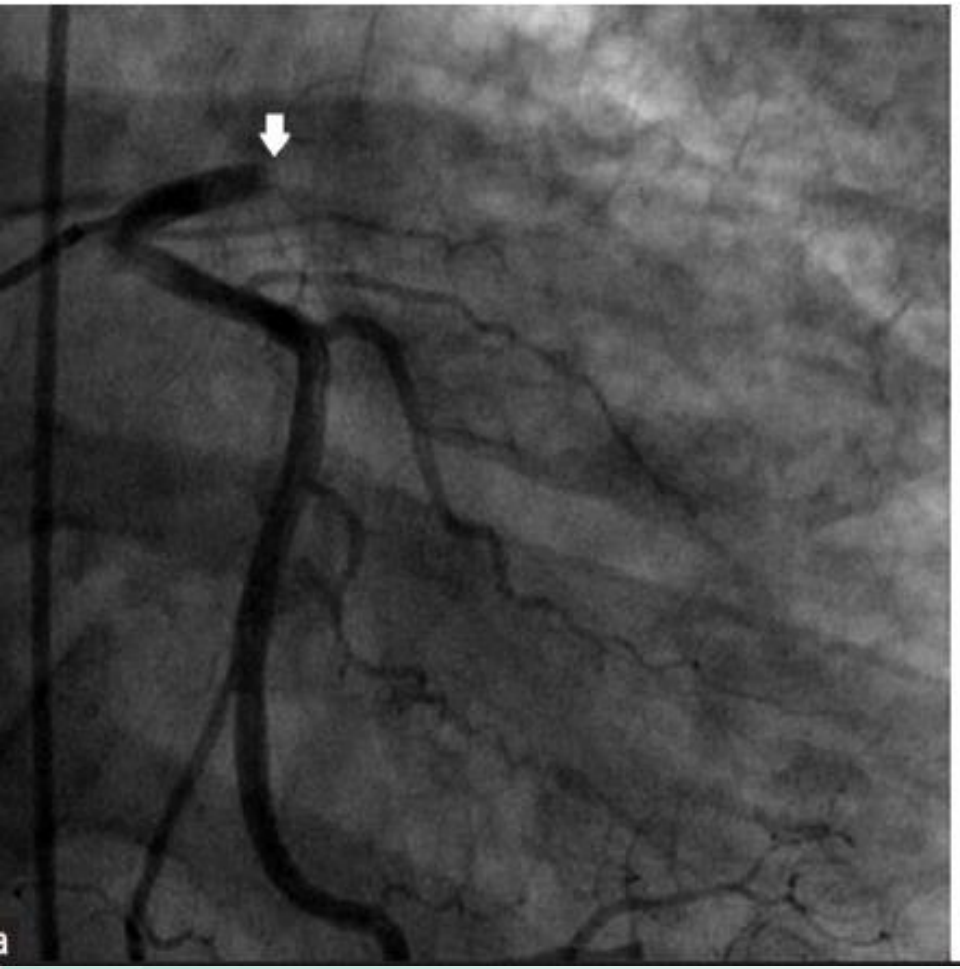
Frank G.Yanowitz, M.D.

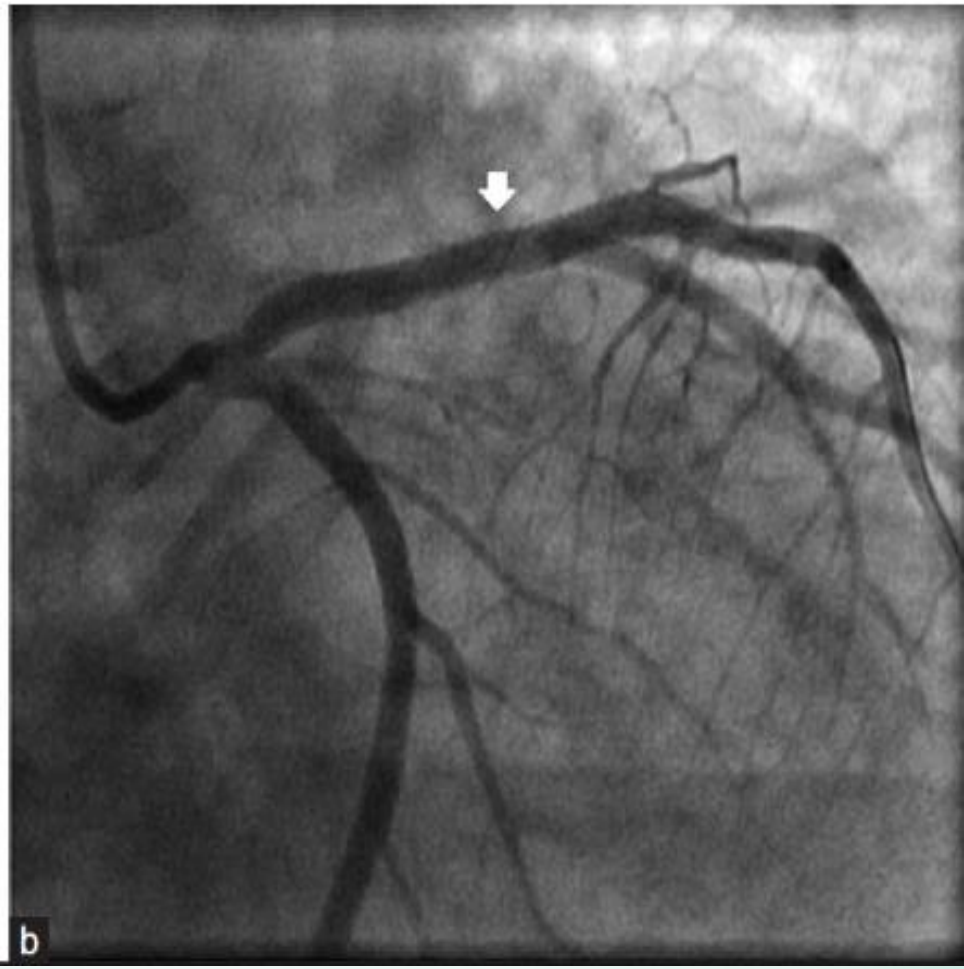
Significant pathologic Q-waves (V2-6, I, aVL) plus marked ST segment elevation are evidence for this large anterior/anterolateral MI. The exact age of the infarction cannot be determined without clinical correlation and previous ECGs, but this is likely a recent MI.


11-OCT-1941 (53 yr)
Male Caucasian
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
25mm/s 10mm/mV 150Hz
18-NOV-1994 15:01
EID:Unconfirmed EDT: ORDER:
© 1997 Frank G. Yanowitz, M.D.

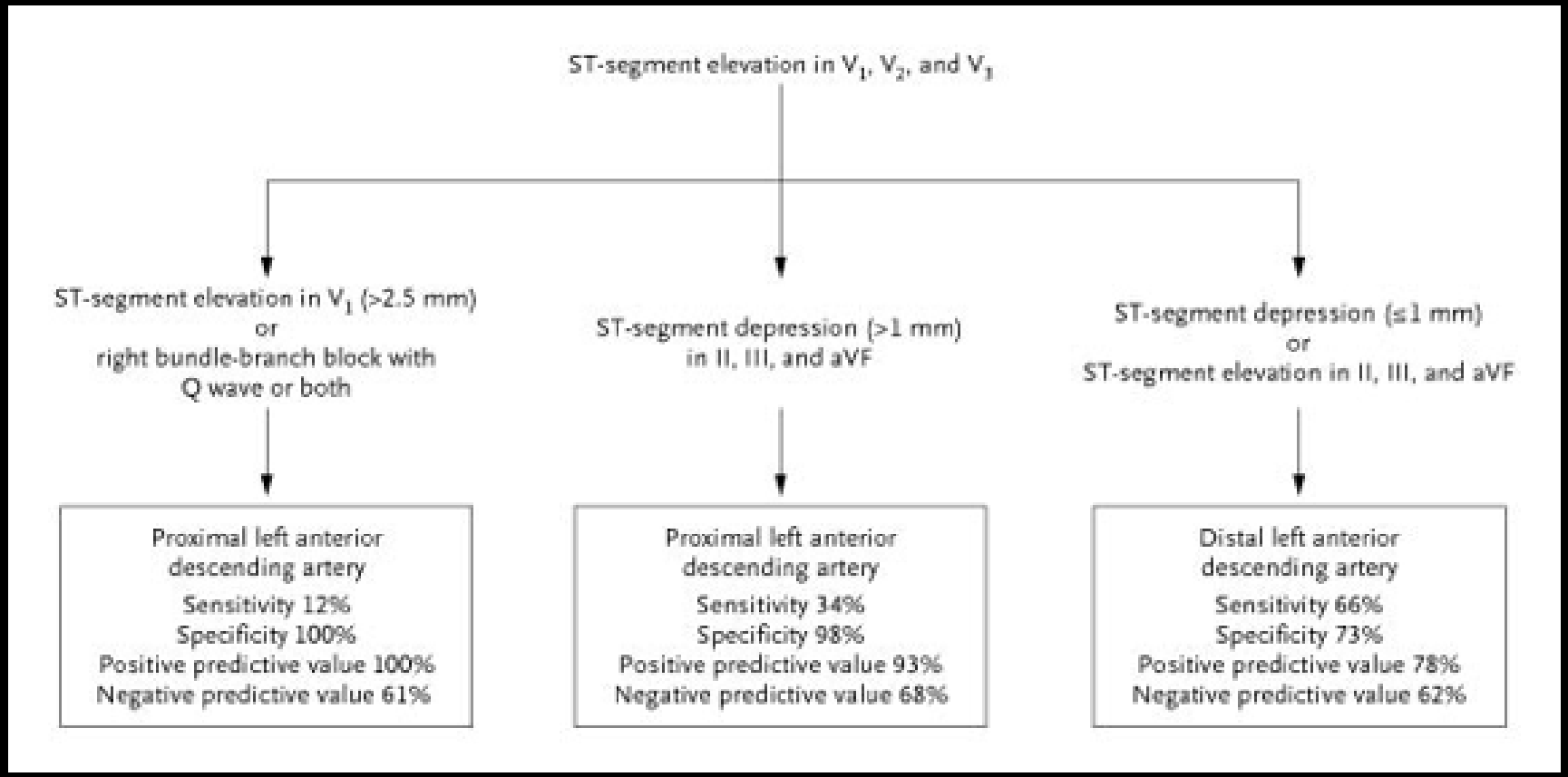


Normal blood supply of the heart

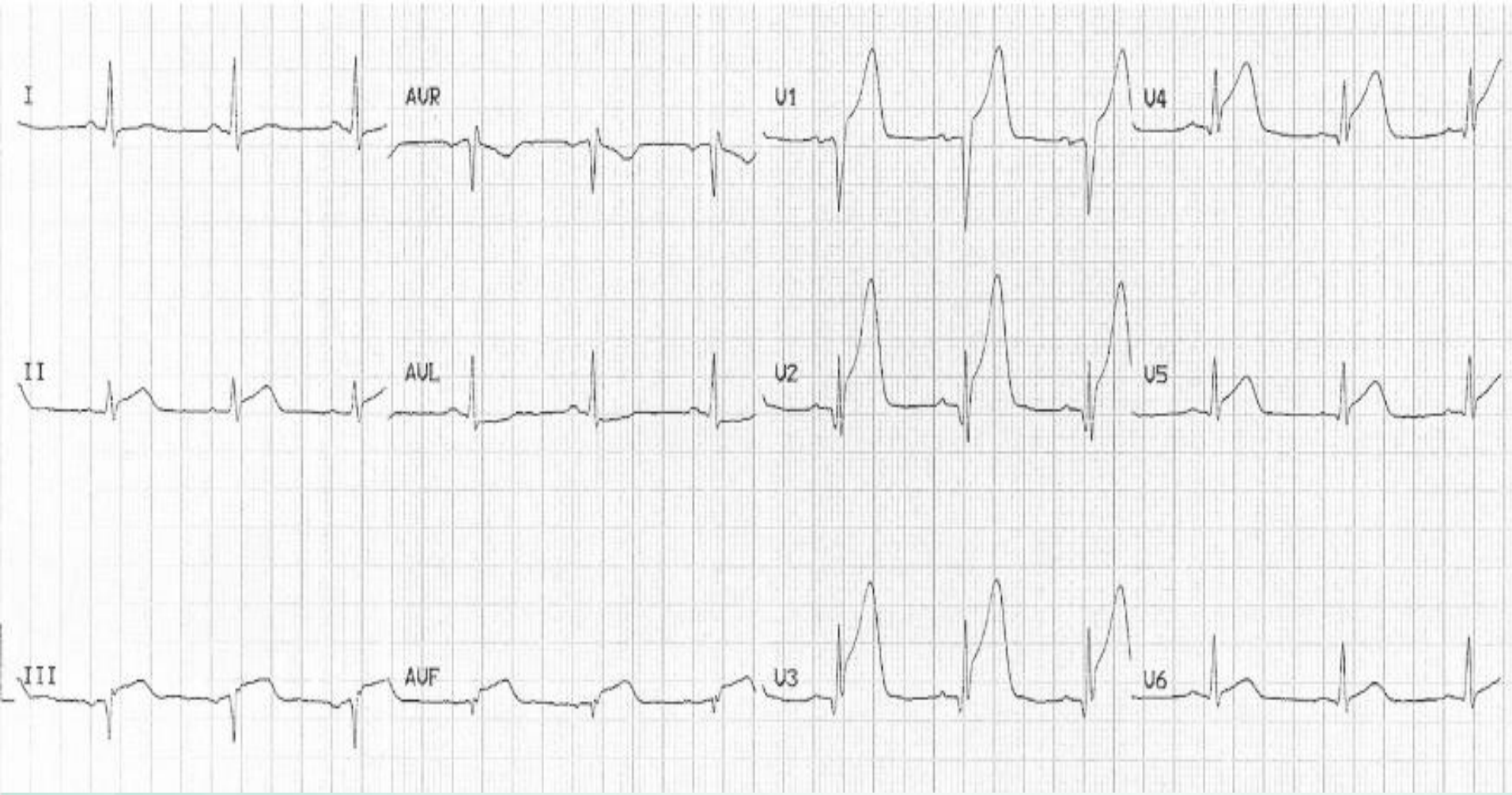




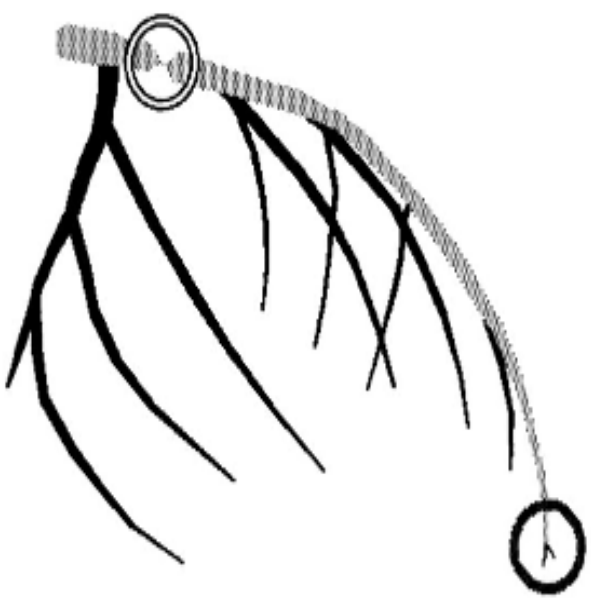
Algorithm for Electrocardiographic Identification of the Infarct-Related Artery in Anterior Myocardial Infarction.



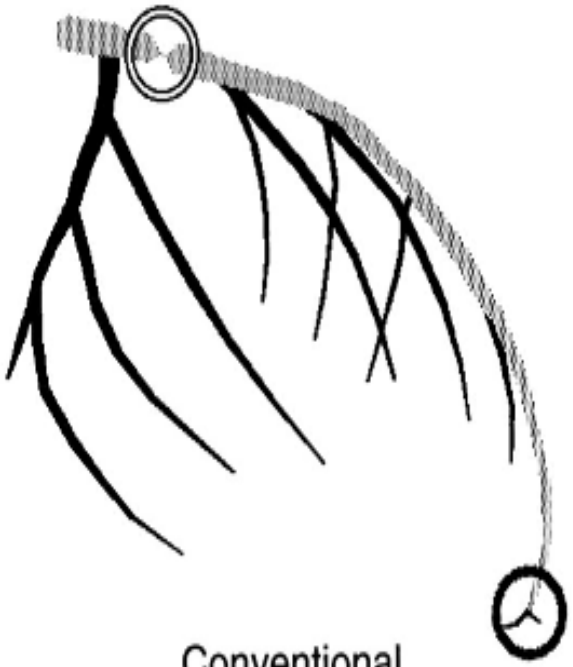
Zimetbaum PJ, Josephson ME. N Engl J Med 2003;348:933-940.


I
AUR
U1
U4
II
AUL
U2
U5
III
AUF
U3
U6

Proximal Culprit Lesion in LAD:

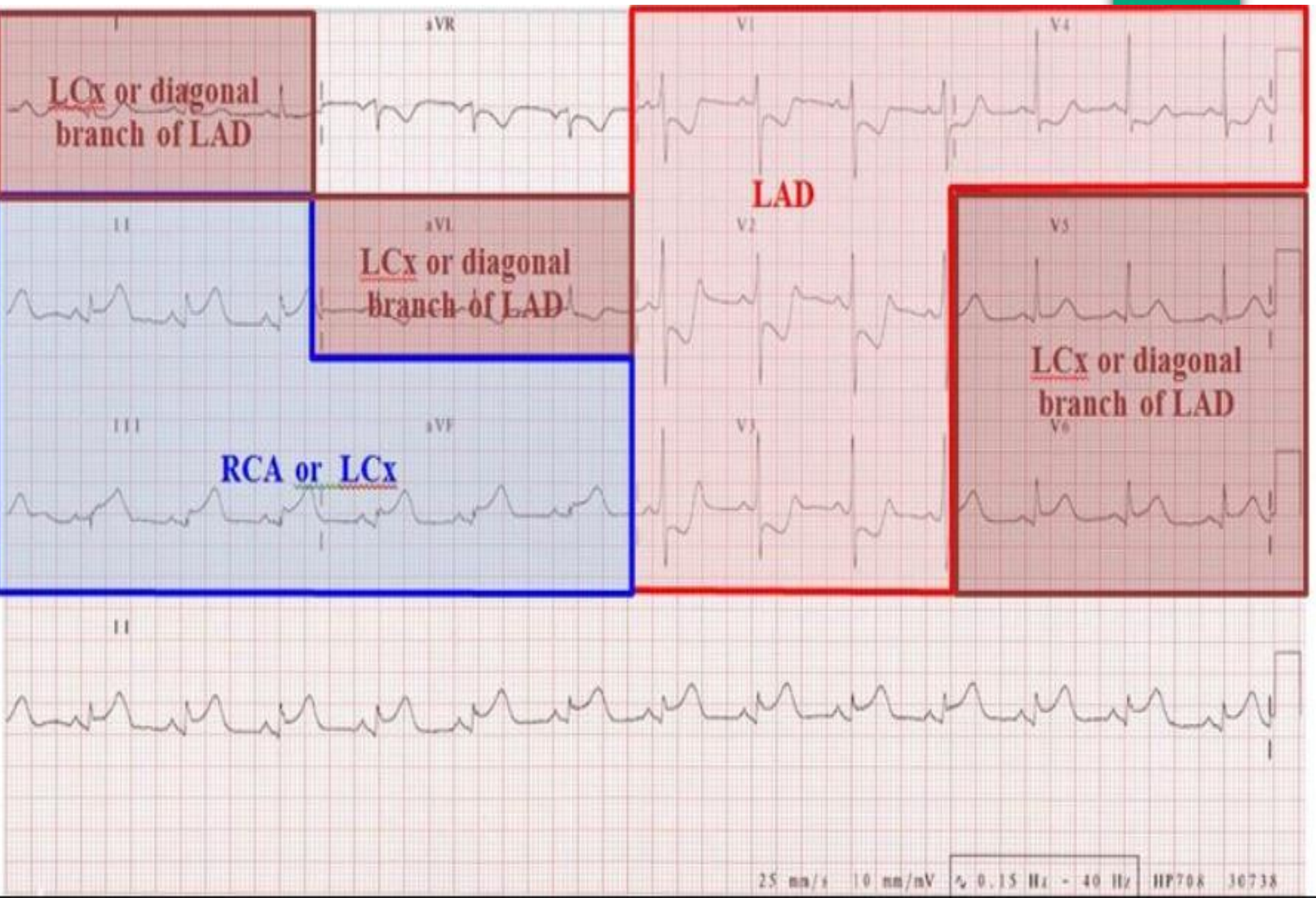


Short



Conventional

Wrap Around


I
aVR
V1
V4
LCx or diagonal branch of LAD
LAD
II
aVL
V2
V5
LCx or diagonal branch of LAD
LCx or diagonal branch of LAD
III
aVF
V3
V6
RCA or LCx
II
25 mm/s
10 mm/mV
0.15 Hz - 40 Hz
HP708
30738

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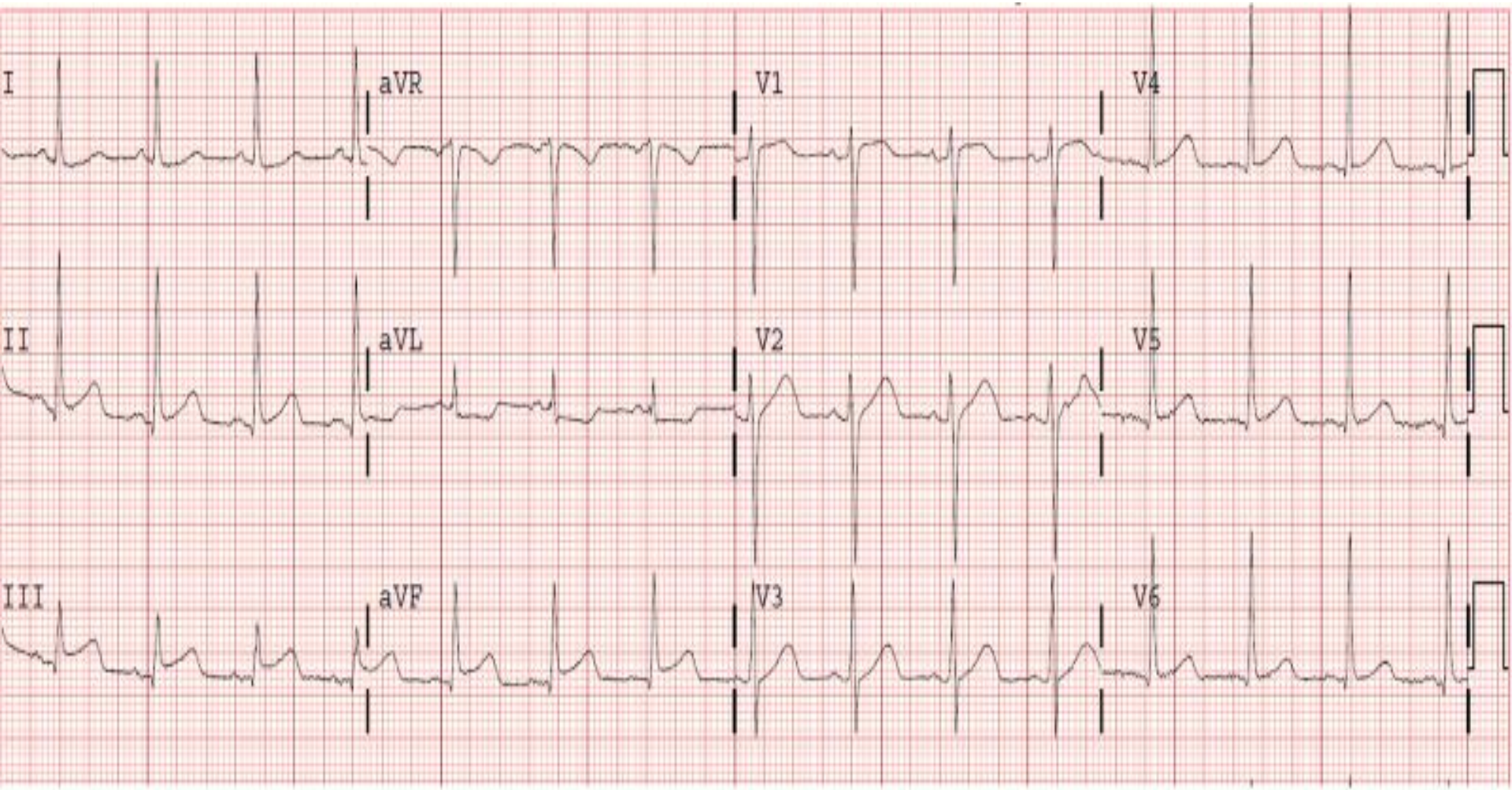

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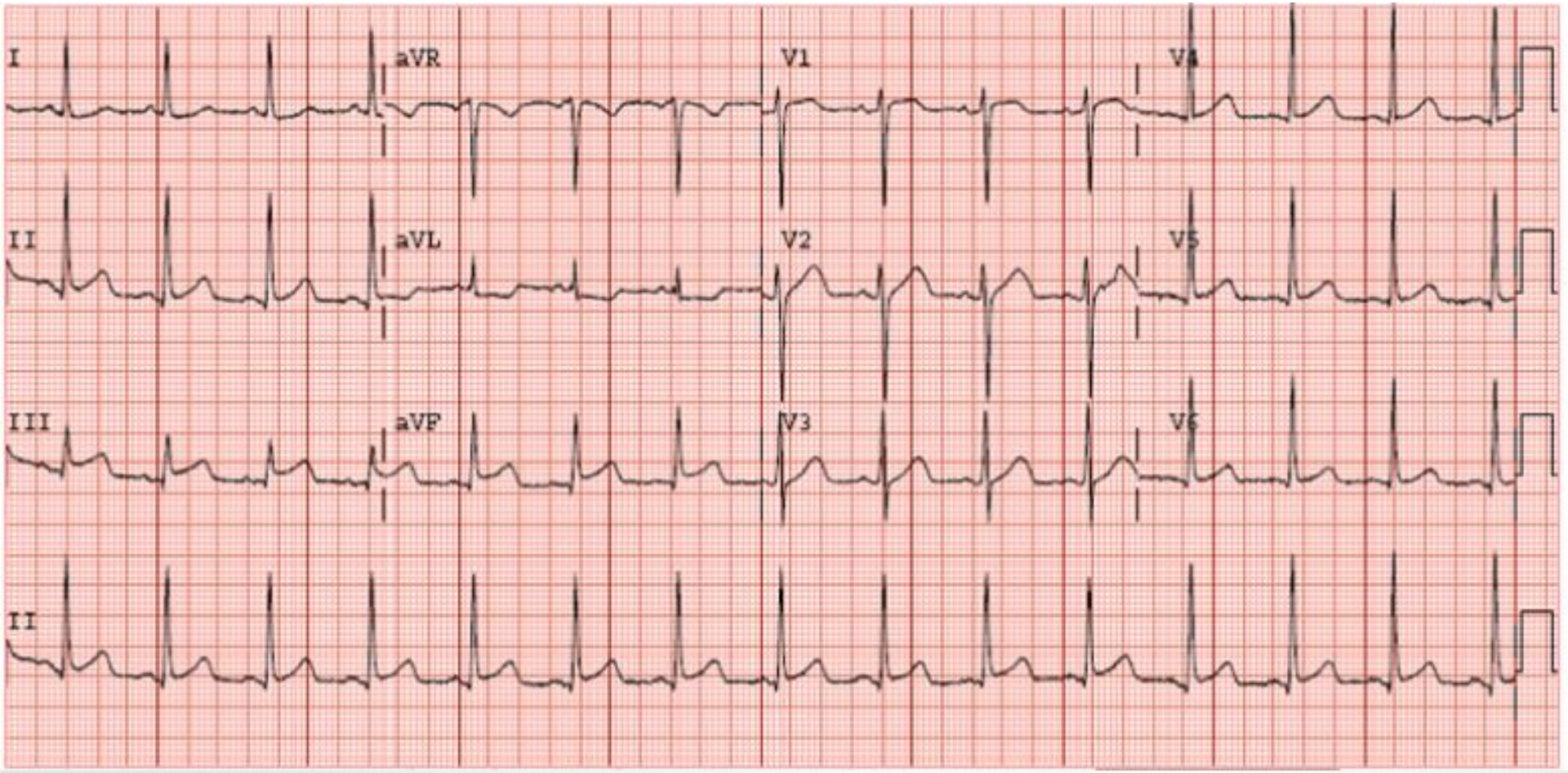

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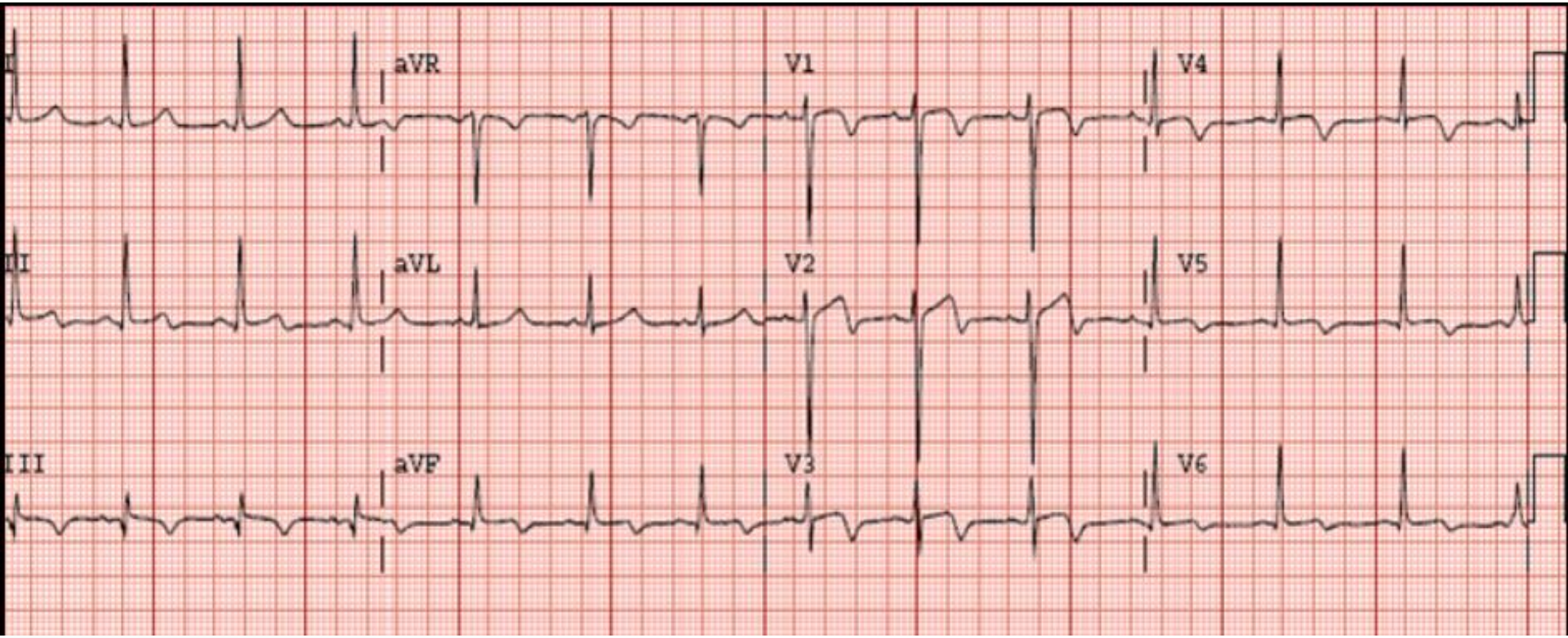
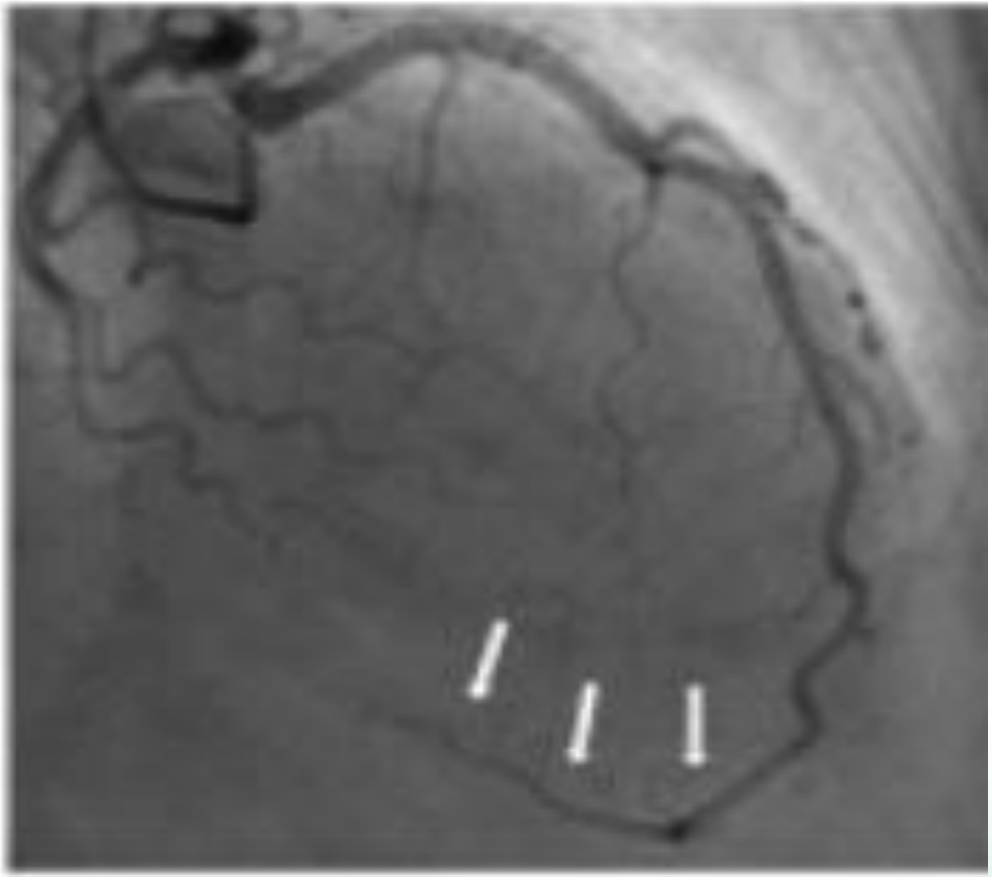
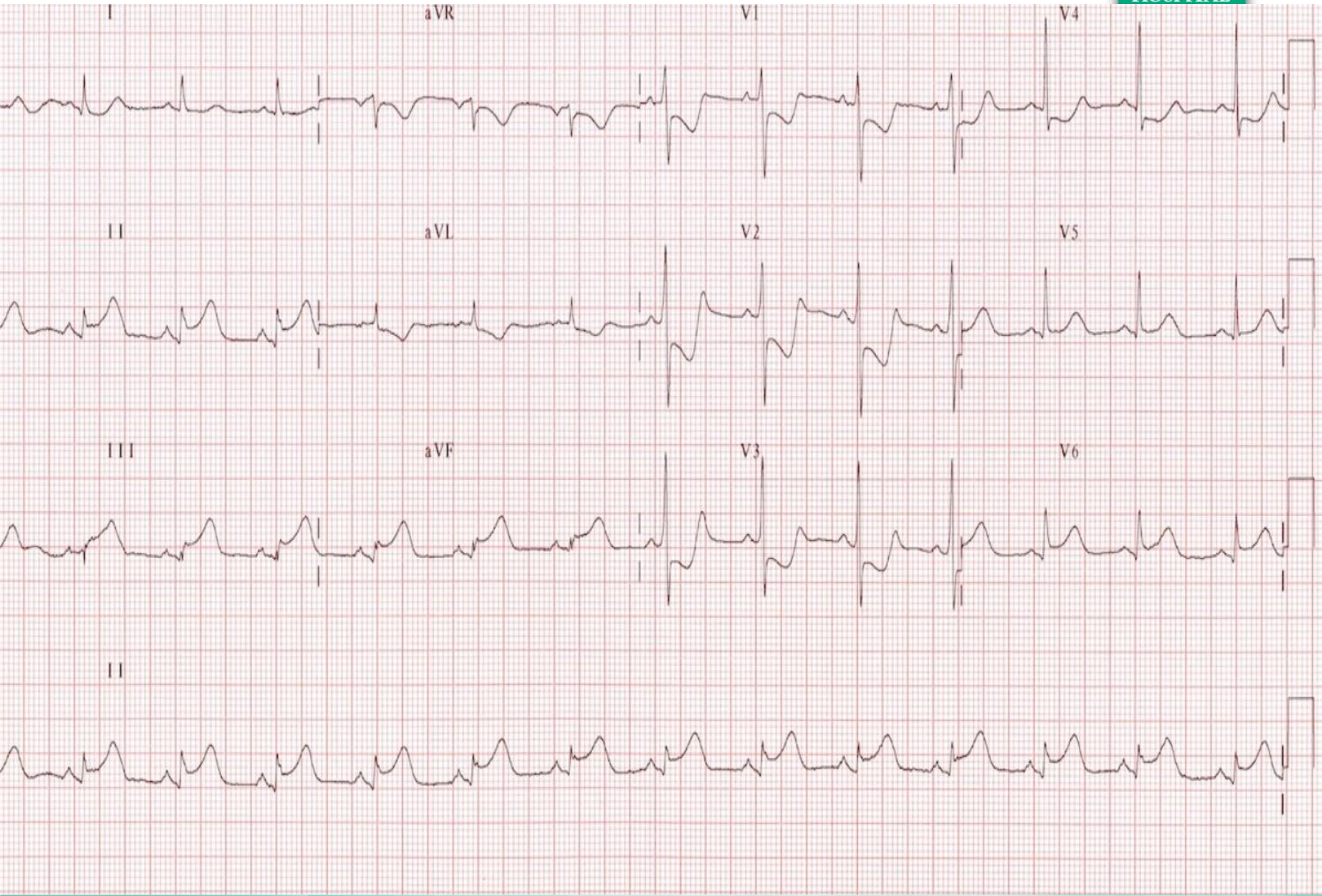
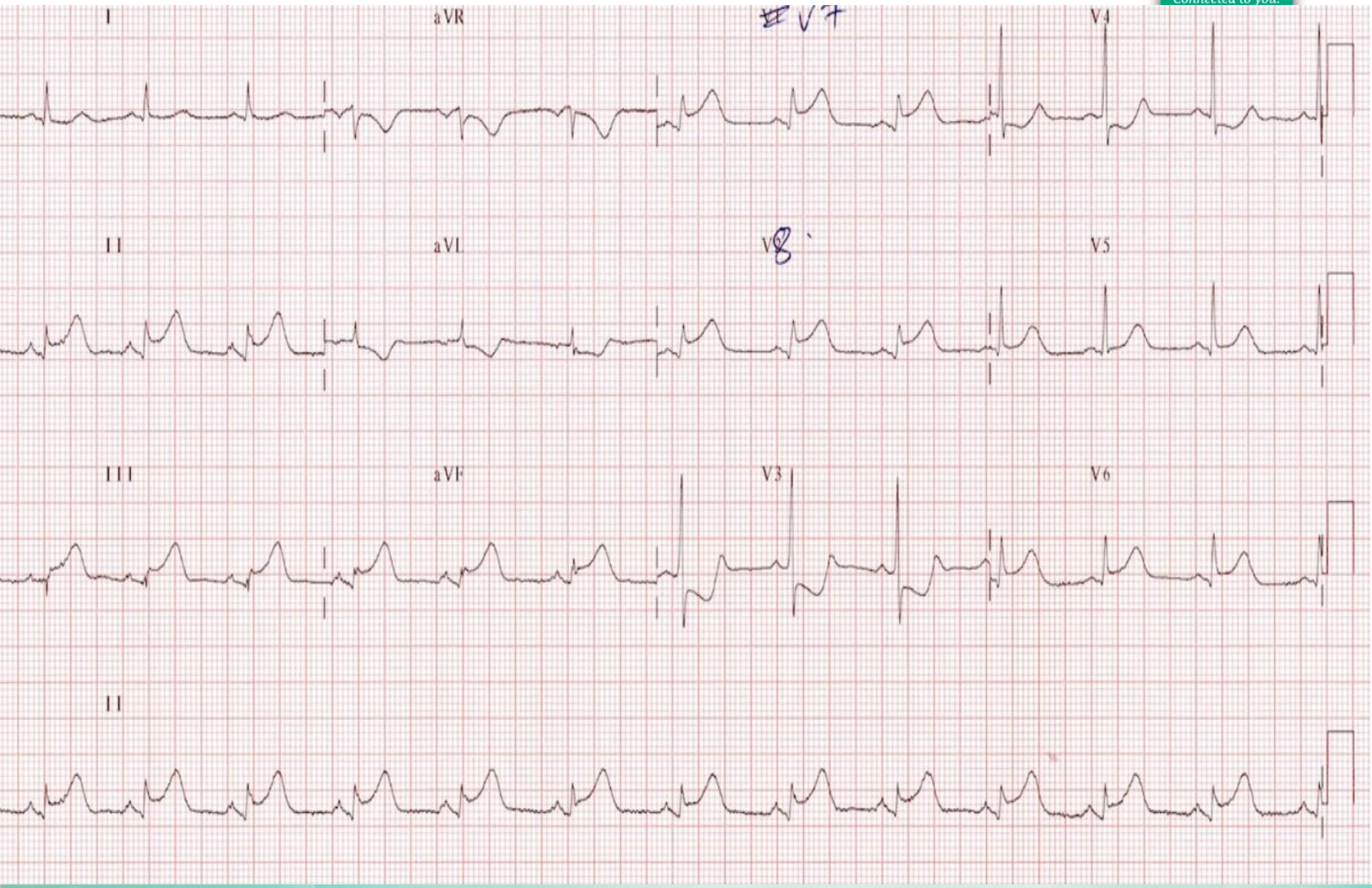
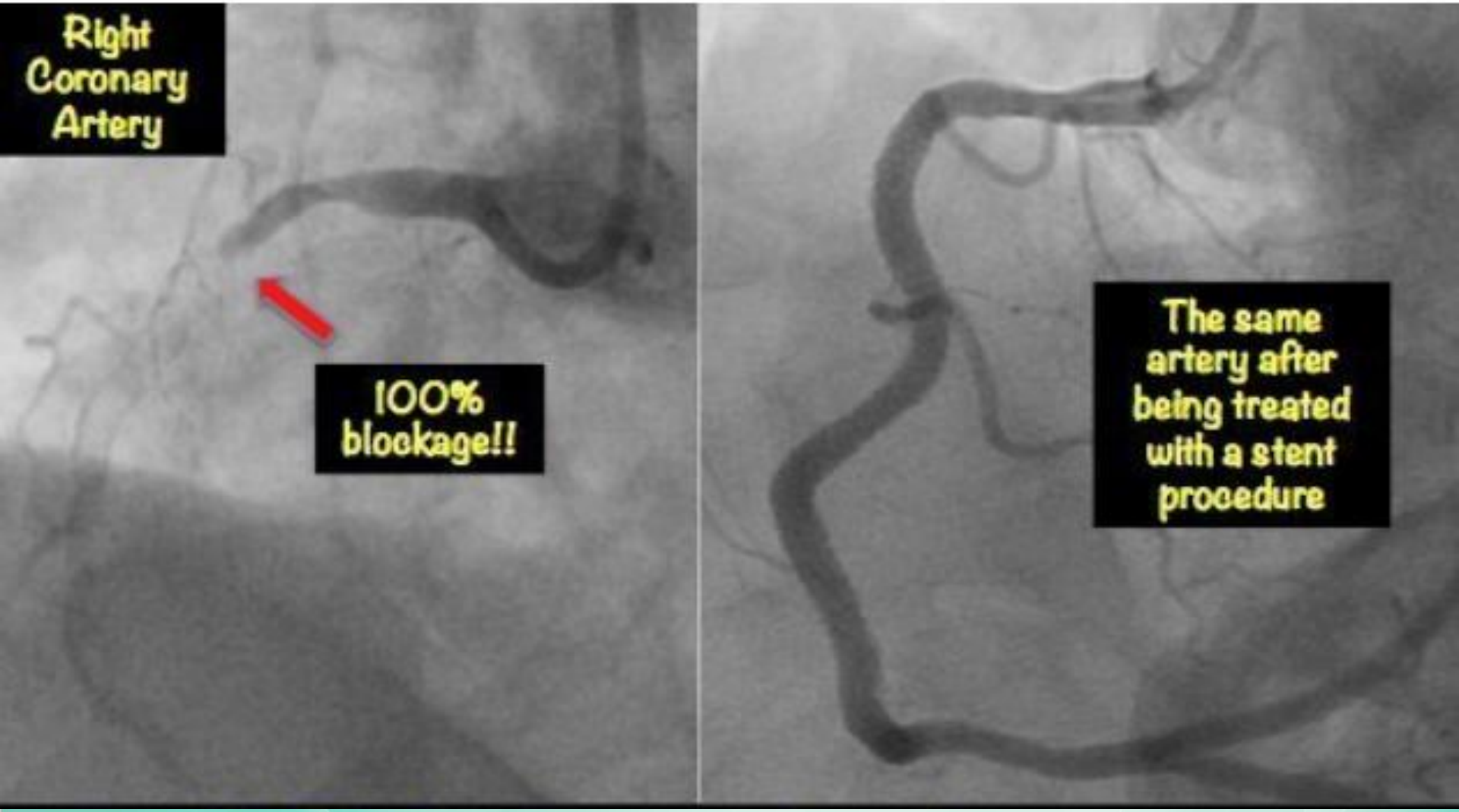


LAD type IV




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

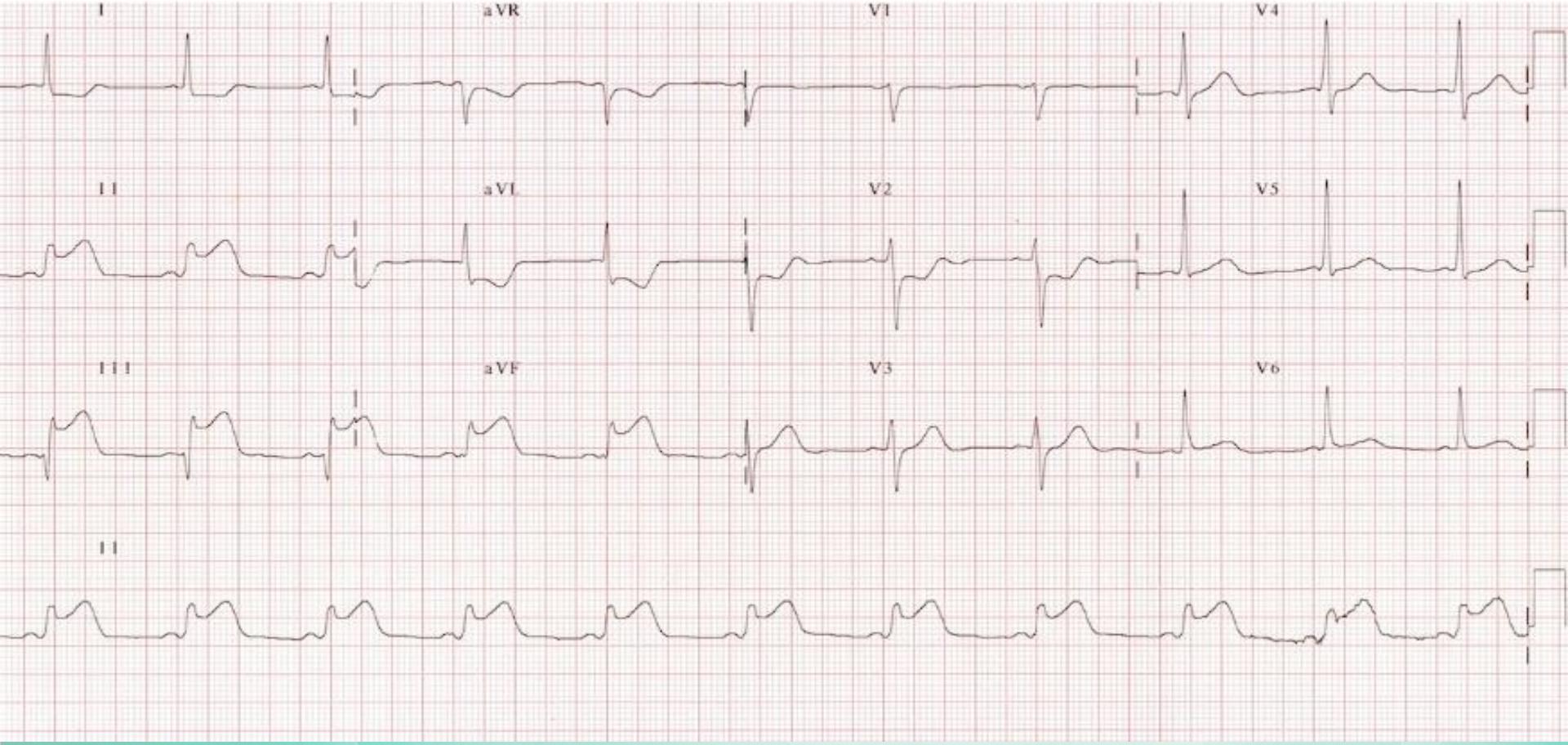

I
aVR
V4
II
aVL
V5
III
aVF
V3
V6
II

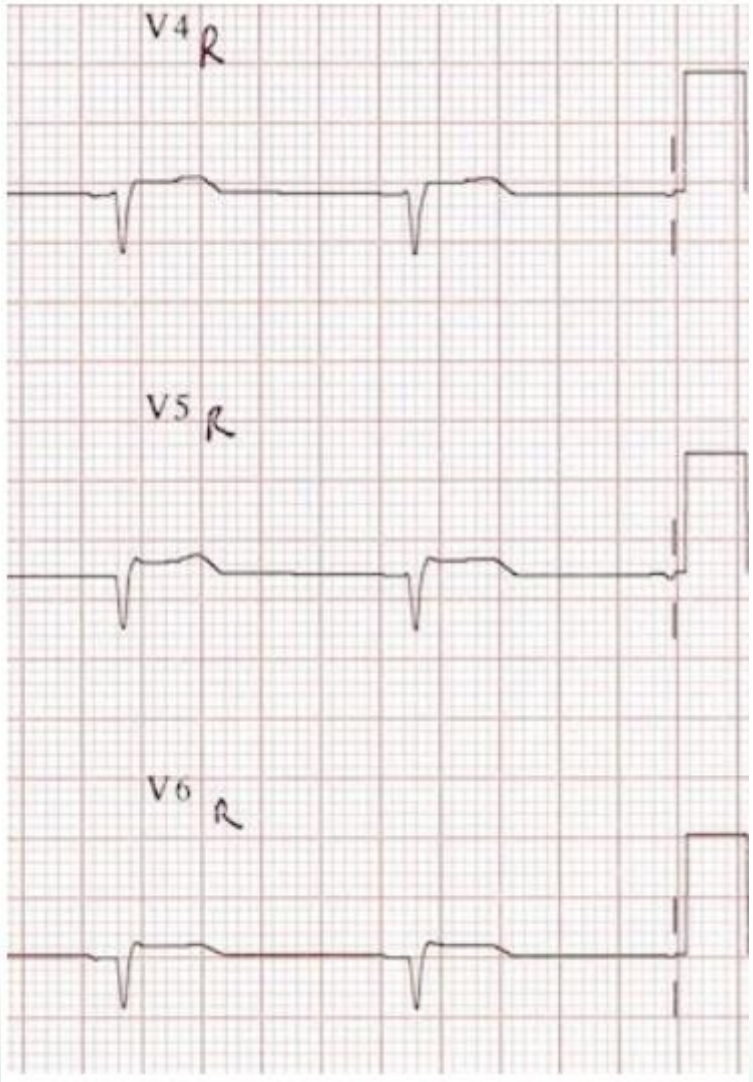

Right Coronary Artery
100% blockage!!
The same artery after being treated with a stent procedure

A 54 year old man presents with one hour of severe central chest pain.

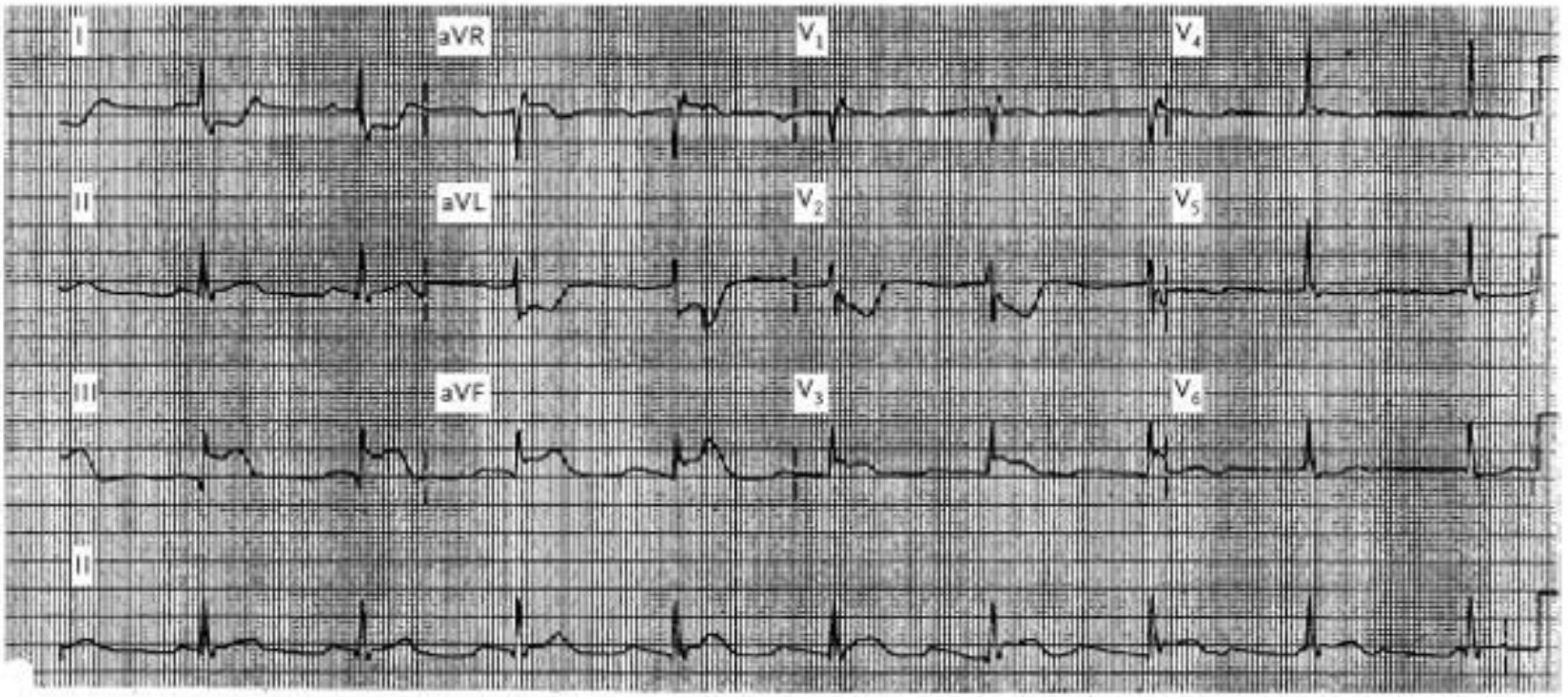
His observations are:

BP	**80/60**	**mmHg supine**
O2 saturation	**97%**	**room air**

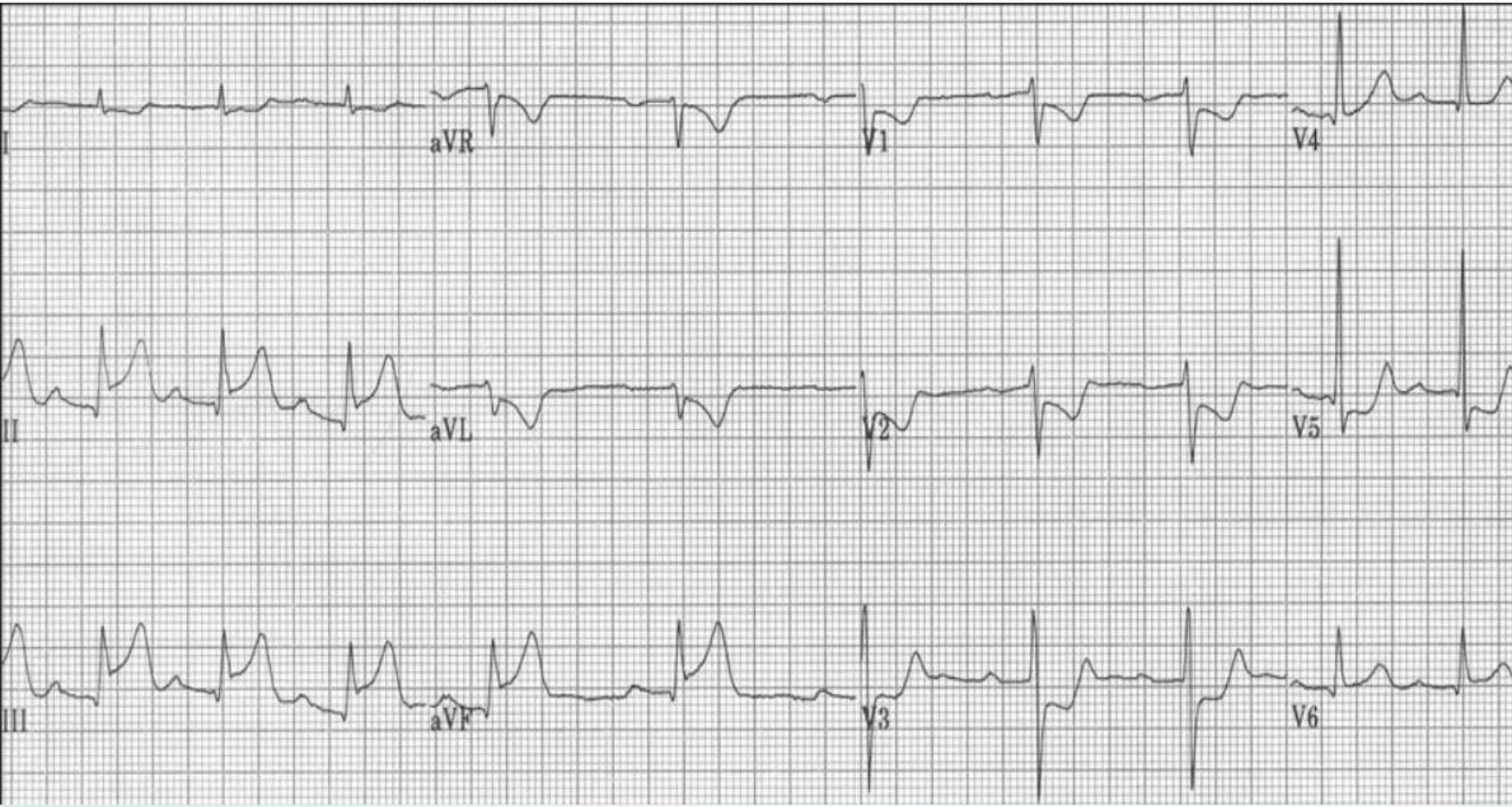




Electrocardiogram Showing Inferior Myocardial Infarction Associated with Complete Heart Block with a Narrow Escape Rhythm.



Zimetbaum PJ, Josephson ME. N Engl J Med 2003;348:933-940.


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

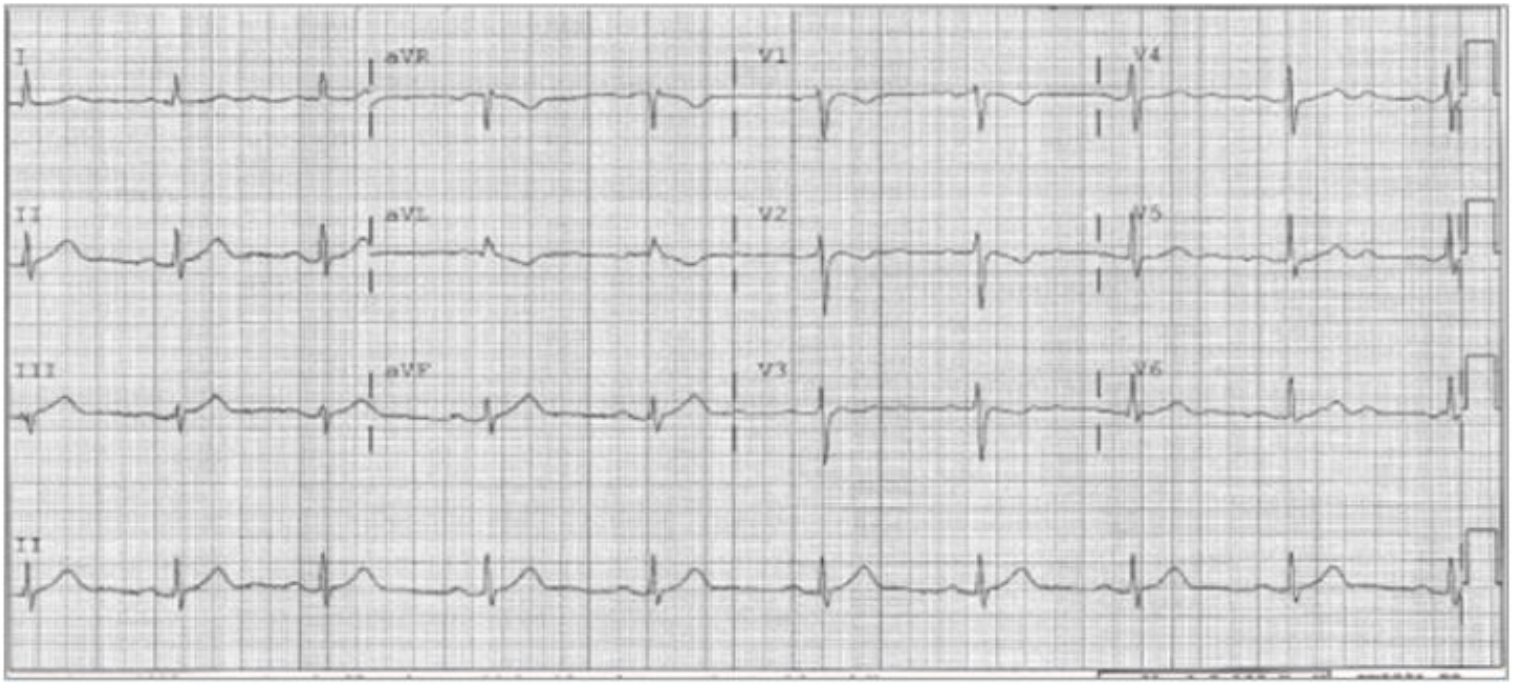
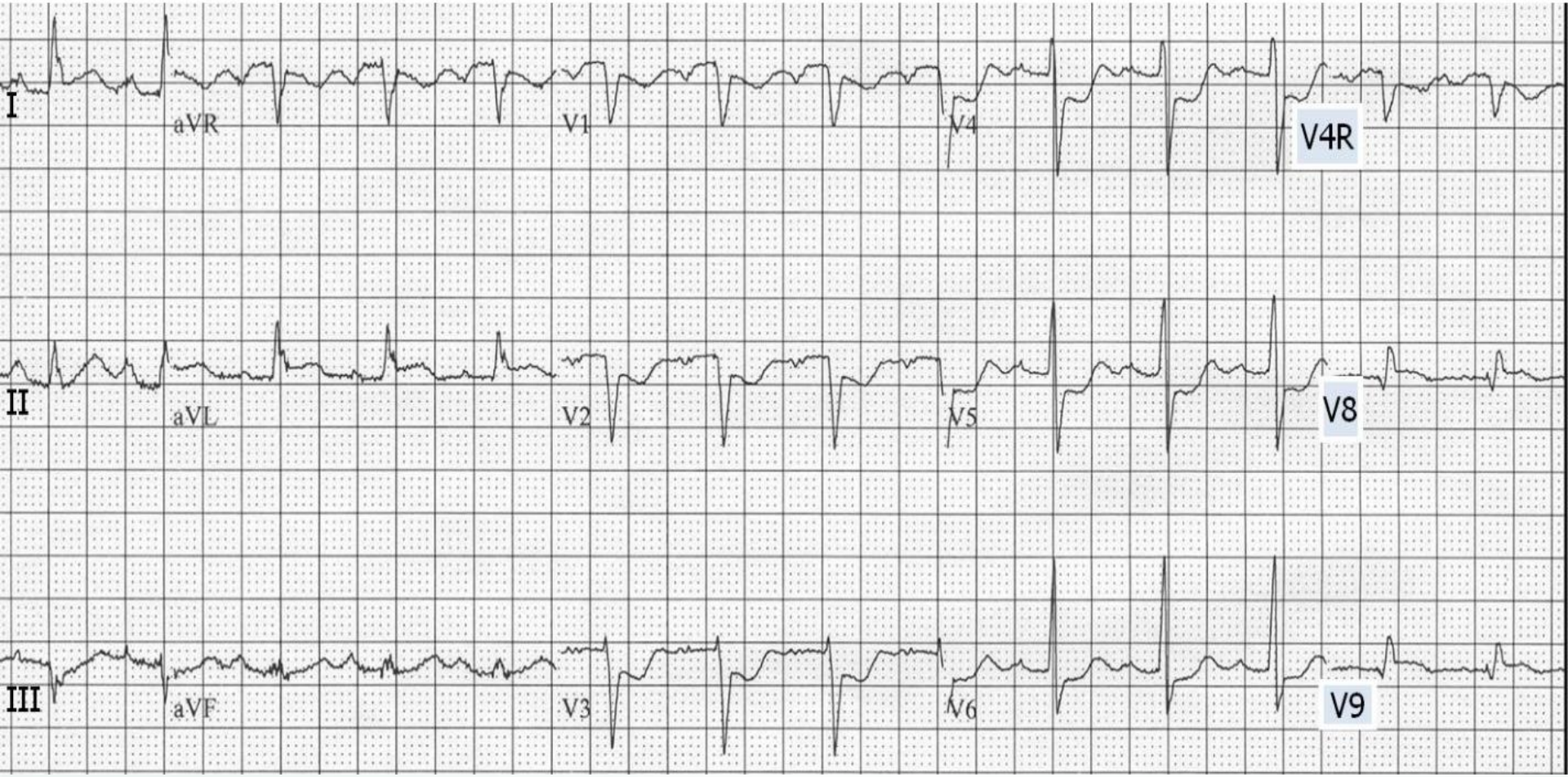
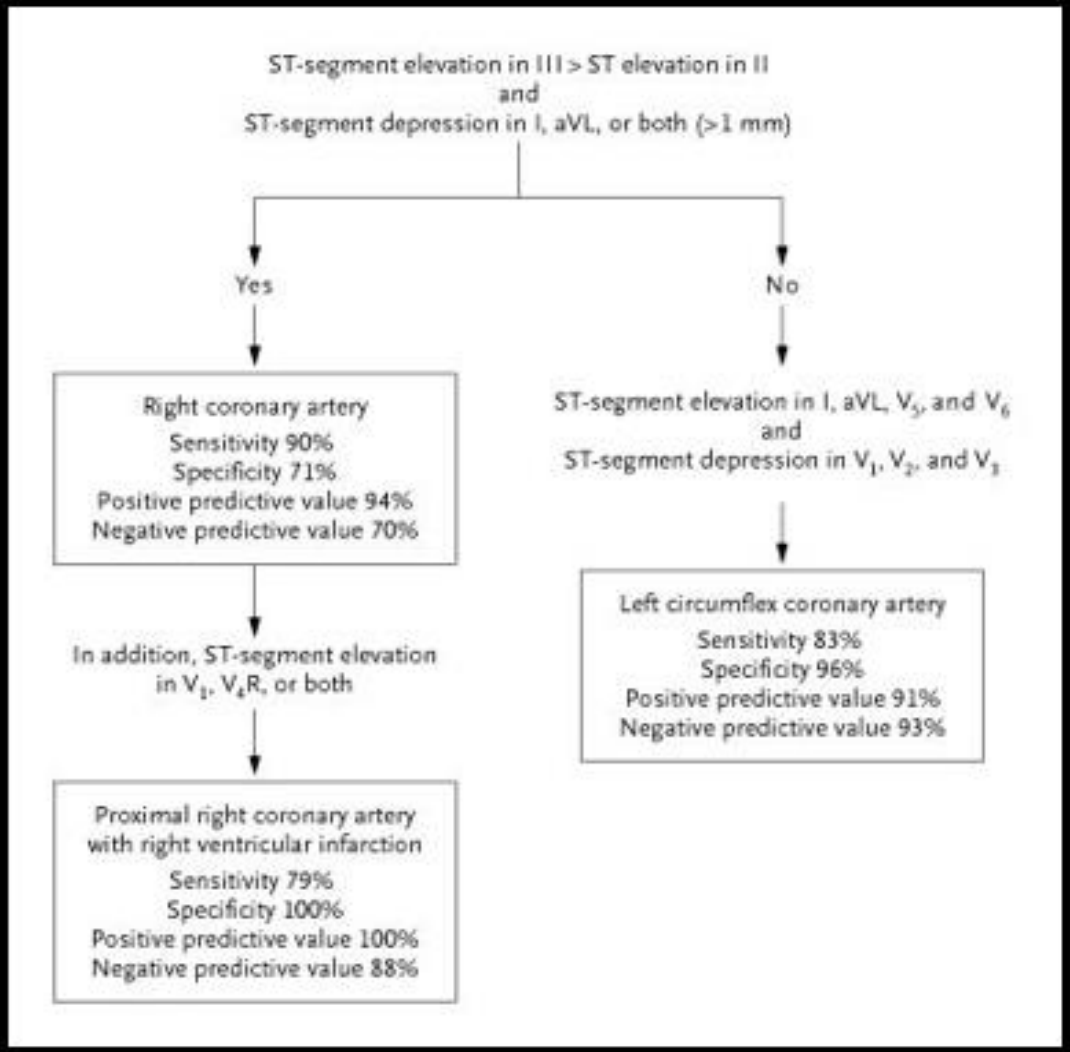


Fig. 2

Example ECG of patient with subtle inferior STEMI, but evidence of ST depression in aVL which could help make this difficult diagnosis. This patient had acute 99% mid-RCA occlusion.

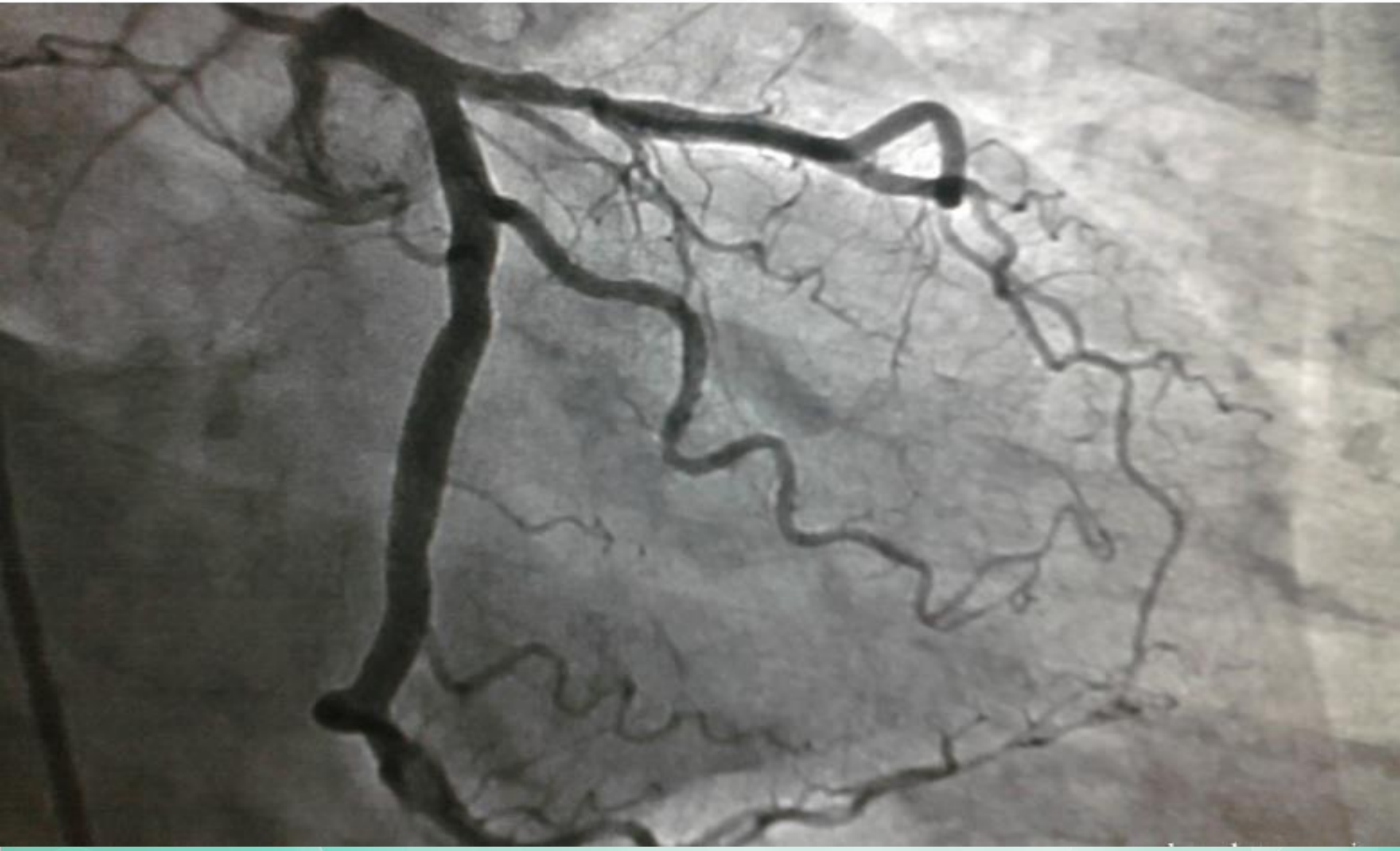

I
aVR
V1
V4
V4R
II
aVL
V2
V5
V8
III
aVF
V3
V6
V9

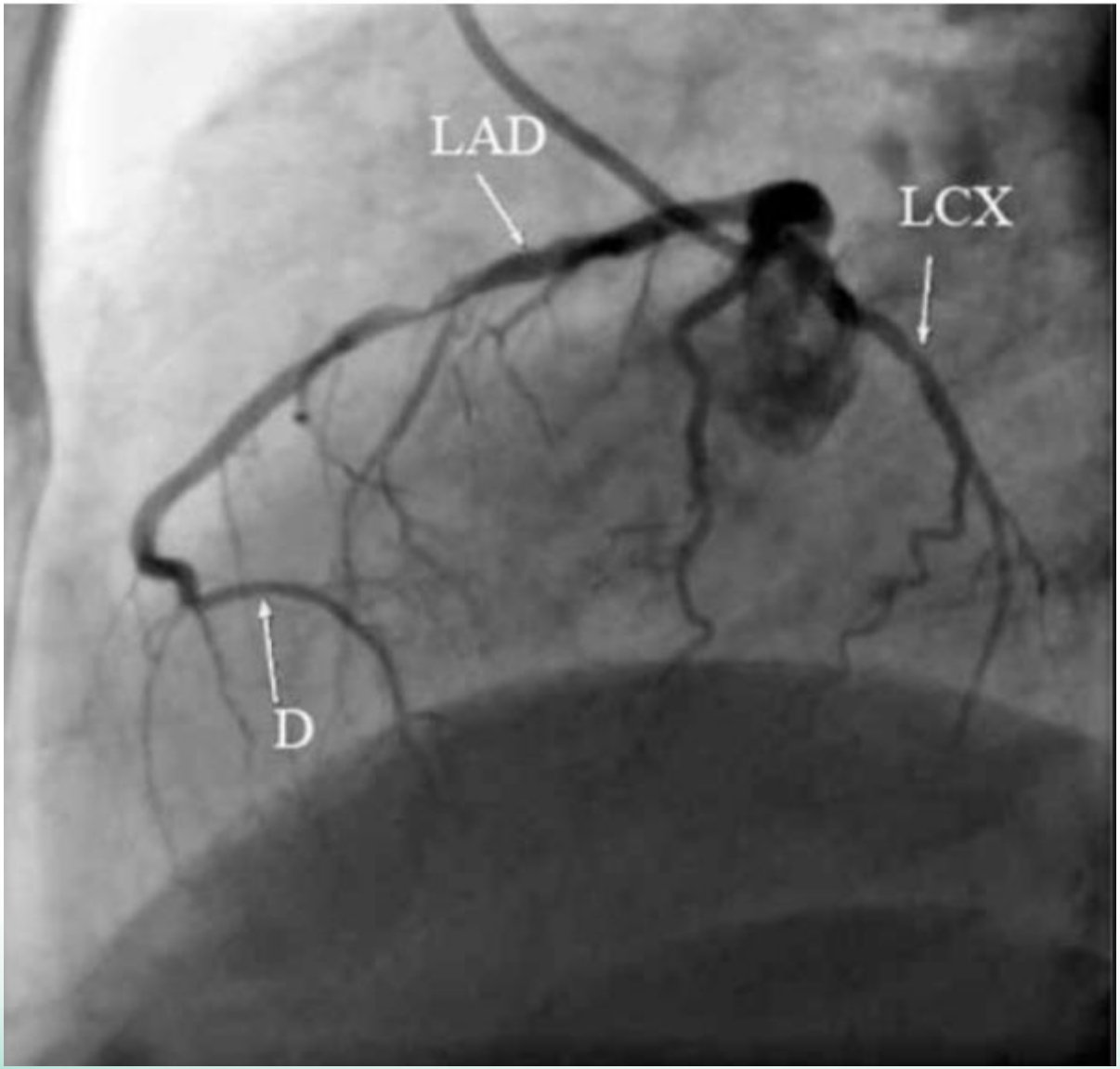
Algorithm for Electrocardiographic Identification of the Infarct-Related Artery in Inferior Myocardial Infarction.

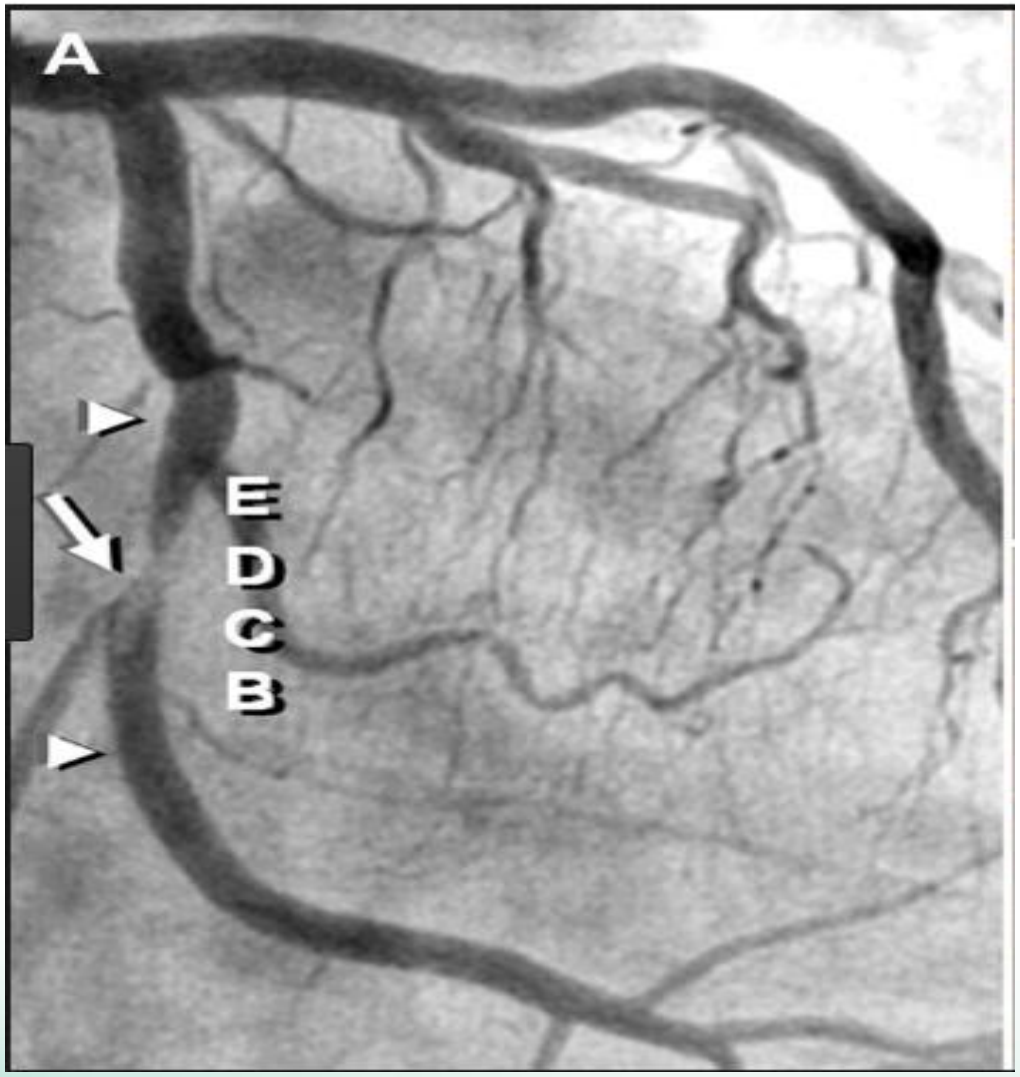


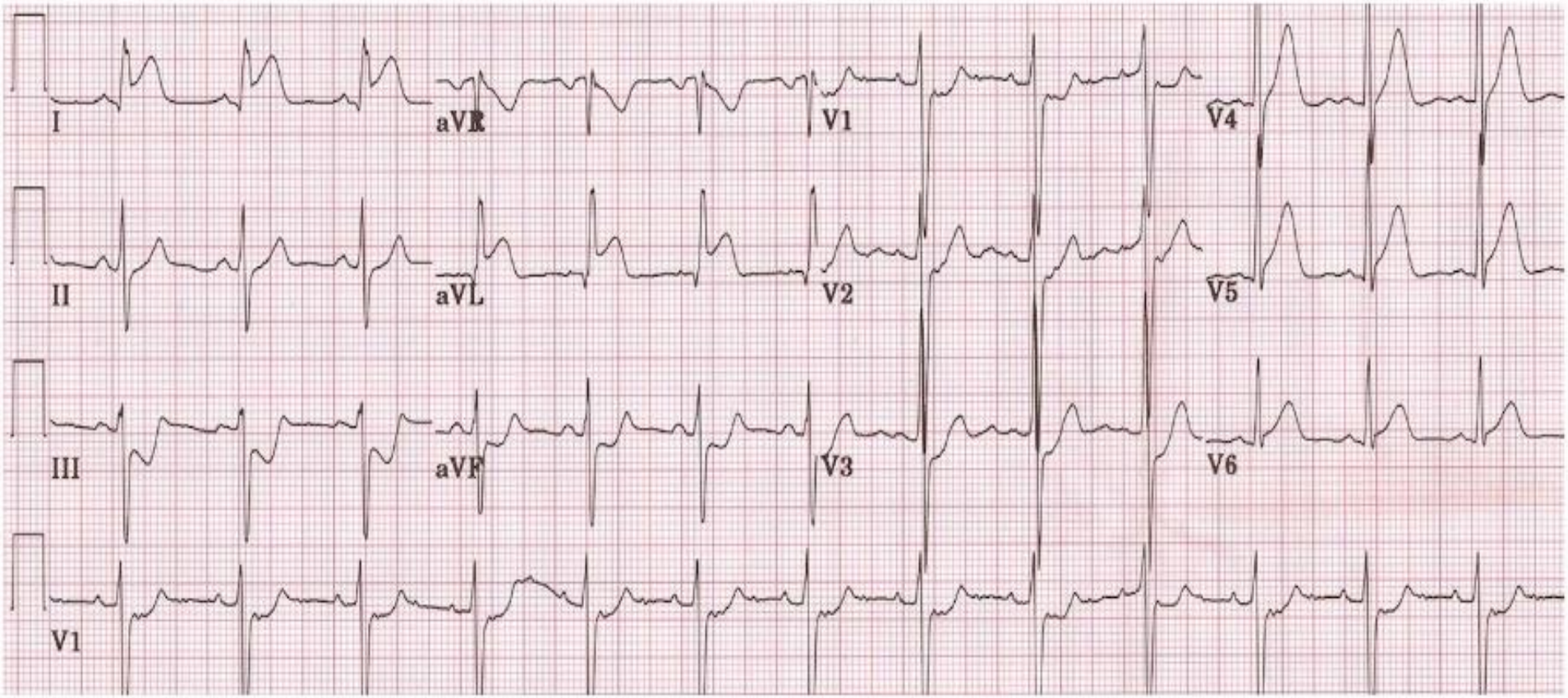
Zimetbaum PJ, Josephson ME. N Engl J Med 2003;348:933-940.

The NEW ENGLAND JOURNAL of MEDICINE

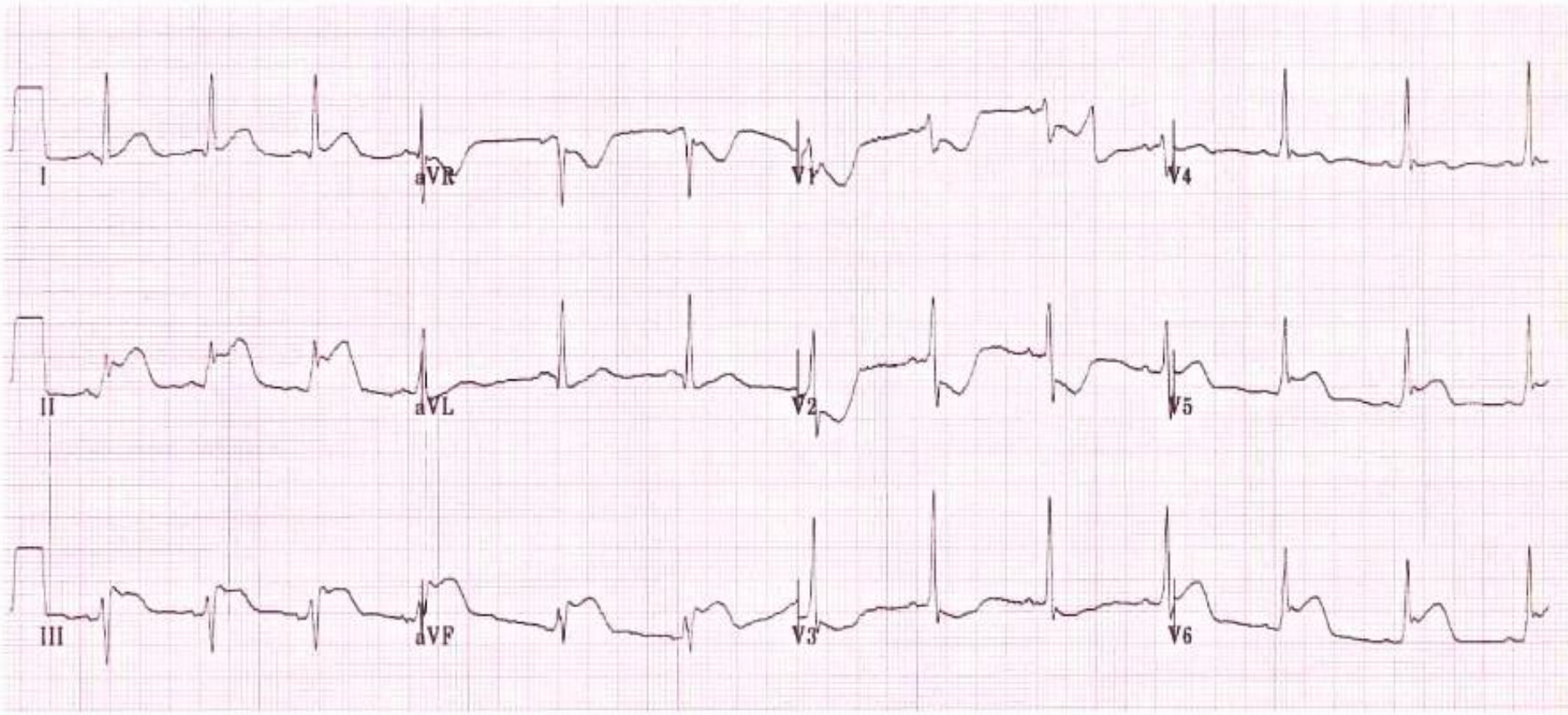



LAD
LCX
D





High Lateral STEMI:



Inferolateral STEMI:

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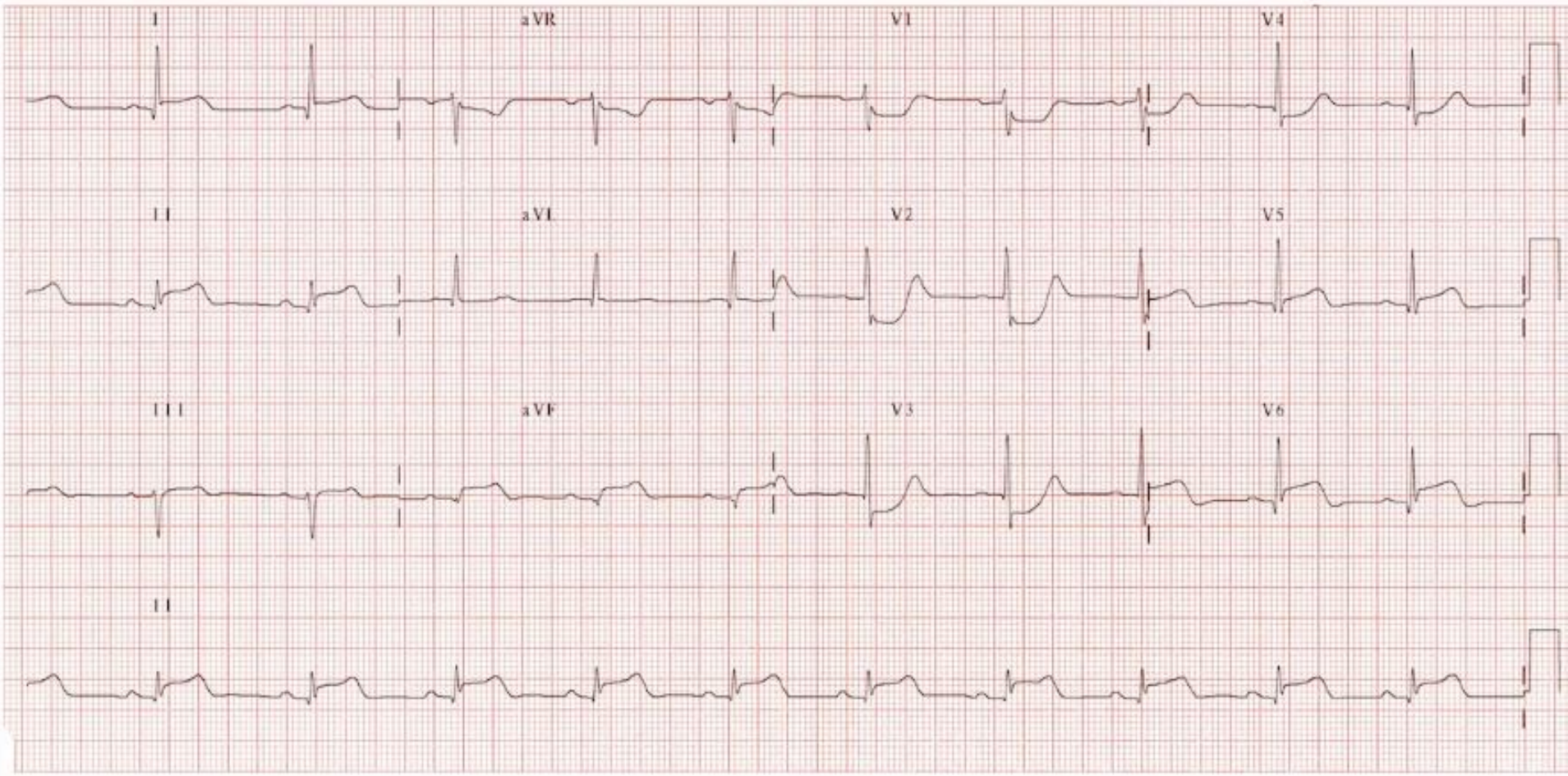
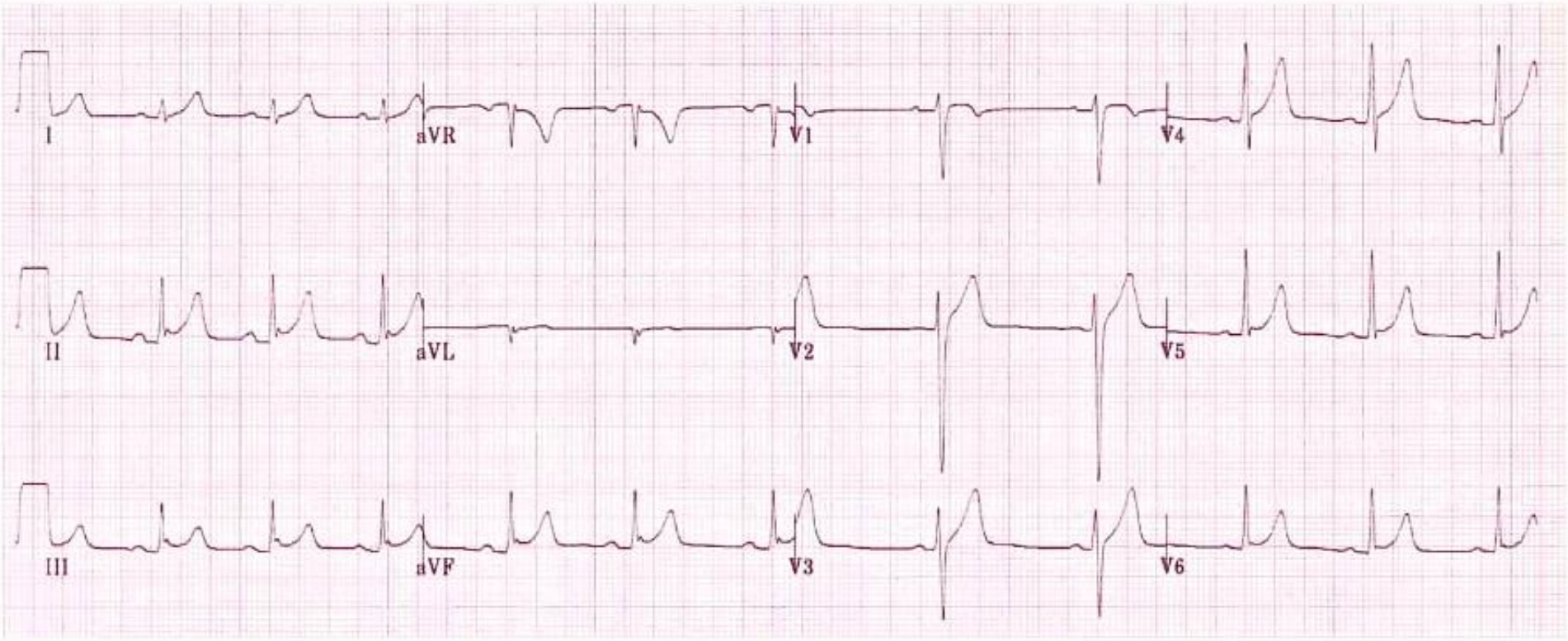


Inferoposterolateral STEMI:

Alternative causes of ST–T changes

- LV aneurysm
- Pericarditis
- Myocarditis
- Bundle-branch block
- Left ventricular hypertrophy
- Hyperkalemia
- Prinzmetal angina
- Early repolarization
- Apical ballooning (Takotsubo cardiomyopathy)
- Wolff-Parkinson-White
- CNS events
- Tricyclic antidepressants

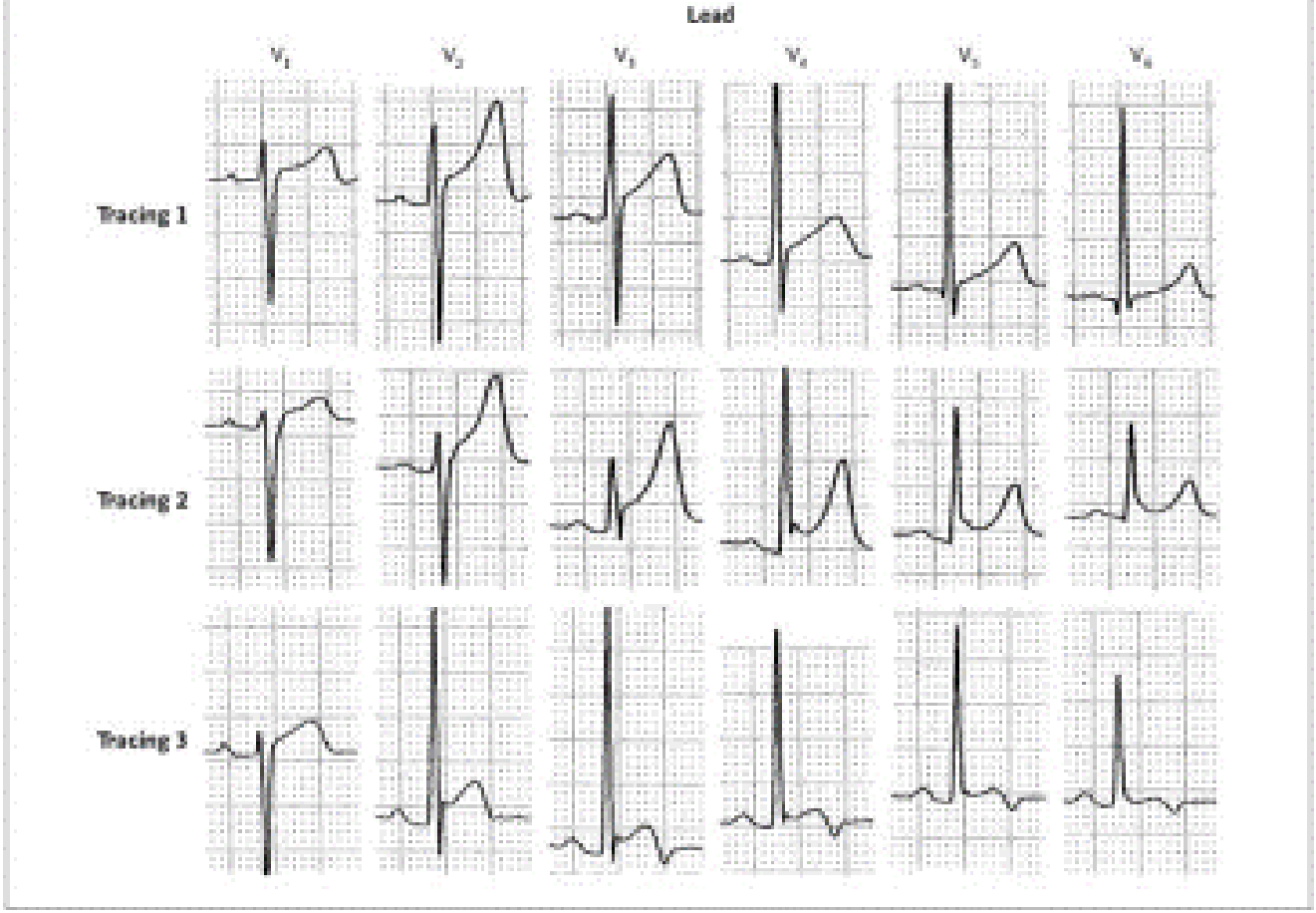


- There is generalised concave ST elevation in the precordial (V2-6) and limb leads (I, II, III, aVF).
- J-point notching is evident in the inferior leads (II, III and aVF).
- There are prominent, slightly asymmetrical T waves that are concordant with the main vector of the QRS complexes.

Table 1. ST-Segment Elevation in Normal Circumstances and in Various Conditions.

Condition	Features
Normal (so-called male pattern)	Seen in approximately 90 percent of healthy young men; therefore, normal Elevation of 1–3 mm Most marked in V_2 Concave
Early repolarization	Most marked in V_4, with notching at J point Tall, upright T waves Reciprocal ST depression in aVR, not in aVL, when limb leads are involved
ST elevation of normal variant	Seen in V_3 through V_5 with inverted T waves Short QT, high QRS voltage

Electrocardiograms Showing Normal ST-Segment Elevation and Normal Variants.

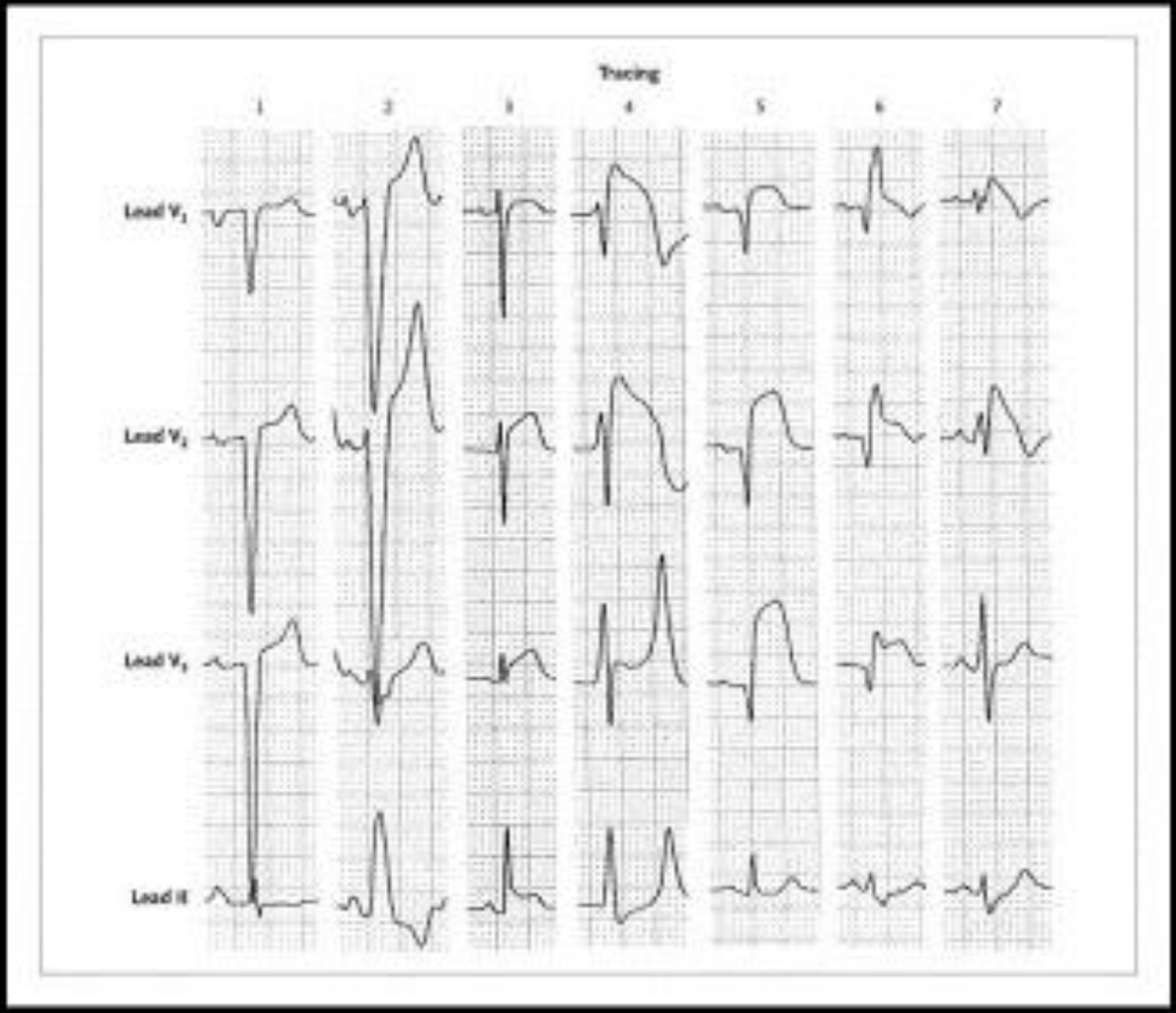


Wang K et al. N Engl J Med 2003;349:2128-2135.

Left ventricular hypertrophy	Concave Other features of left ventricular hypertrophy
Left bundle-branch block	Concave ST-segment deviation discordant from the QRS
Acute pericarditis	Diffuse ST-segment elevation Reciprocal ST-segment depression in aVR, not in aVL Elevation seldom >5 mm PR-segment depression
Hyperkalemia	Other features of hyperkalemia present: Widened QRS and tall, peaked, tented T waves Low-amplitude or absent P waves ST segment usually downsloping

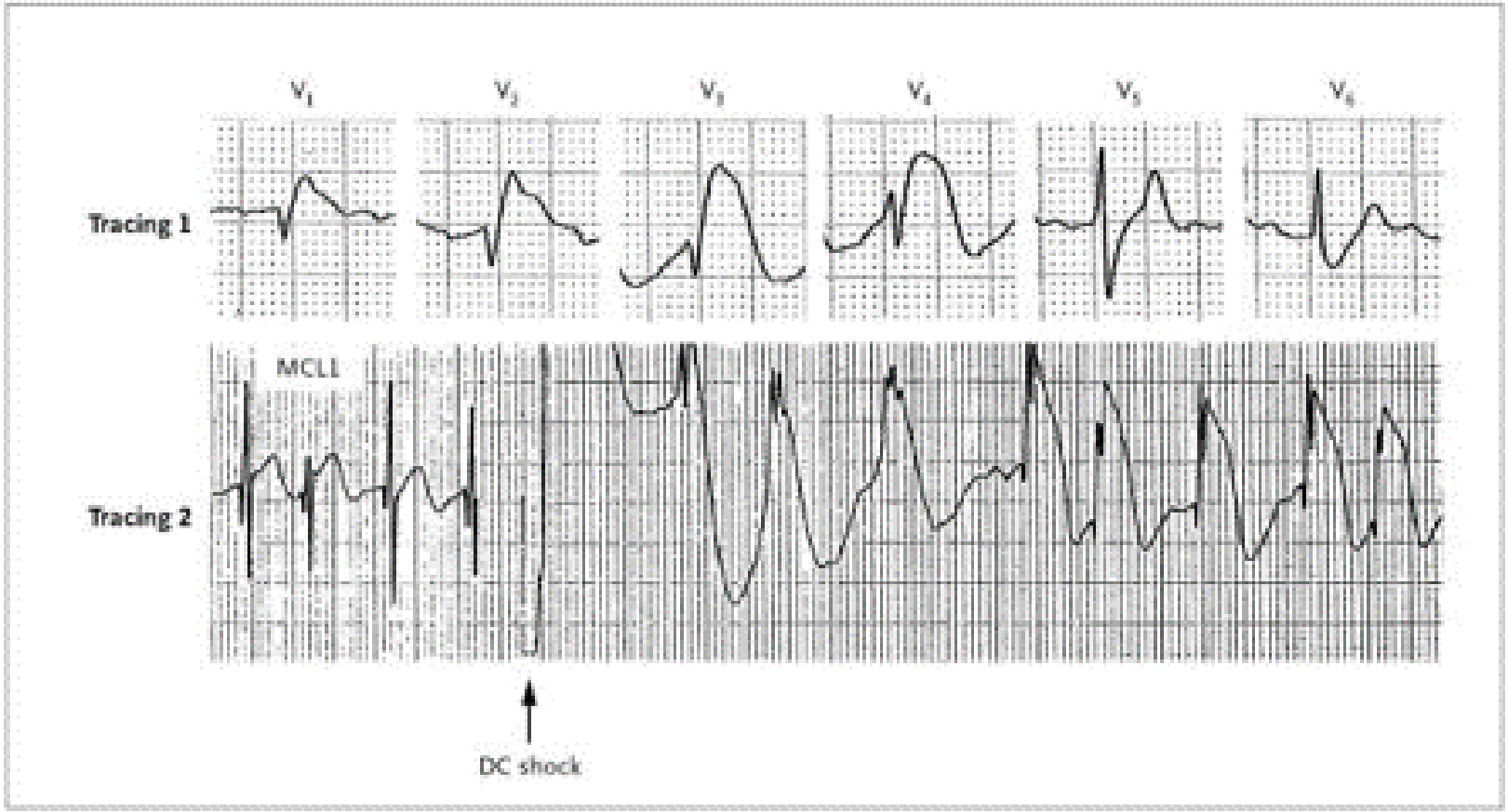
Brugada syndrome	rSR' in V_1 and V_2 ST-segment elevation in V_1 and V_2, typically downsloping
Pulmonary embolism	Changes simulating myocardial infarction seen often in both inferior and antero-septal leads
Cardioversion	Striking ST-segment elevation, often >10 mm, but lasting only a minute or two immediately after direct-current shock
Prinzmetal's angina	Same as ST-segment elevation in infarction, but transient
Acute myocardial infarction	ST segment with a plateau or shoulder or upsloping Reciprocal behavior between aVL and III

Electrocardiograms Showing ST-Segment Elevation in Various Conditions.

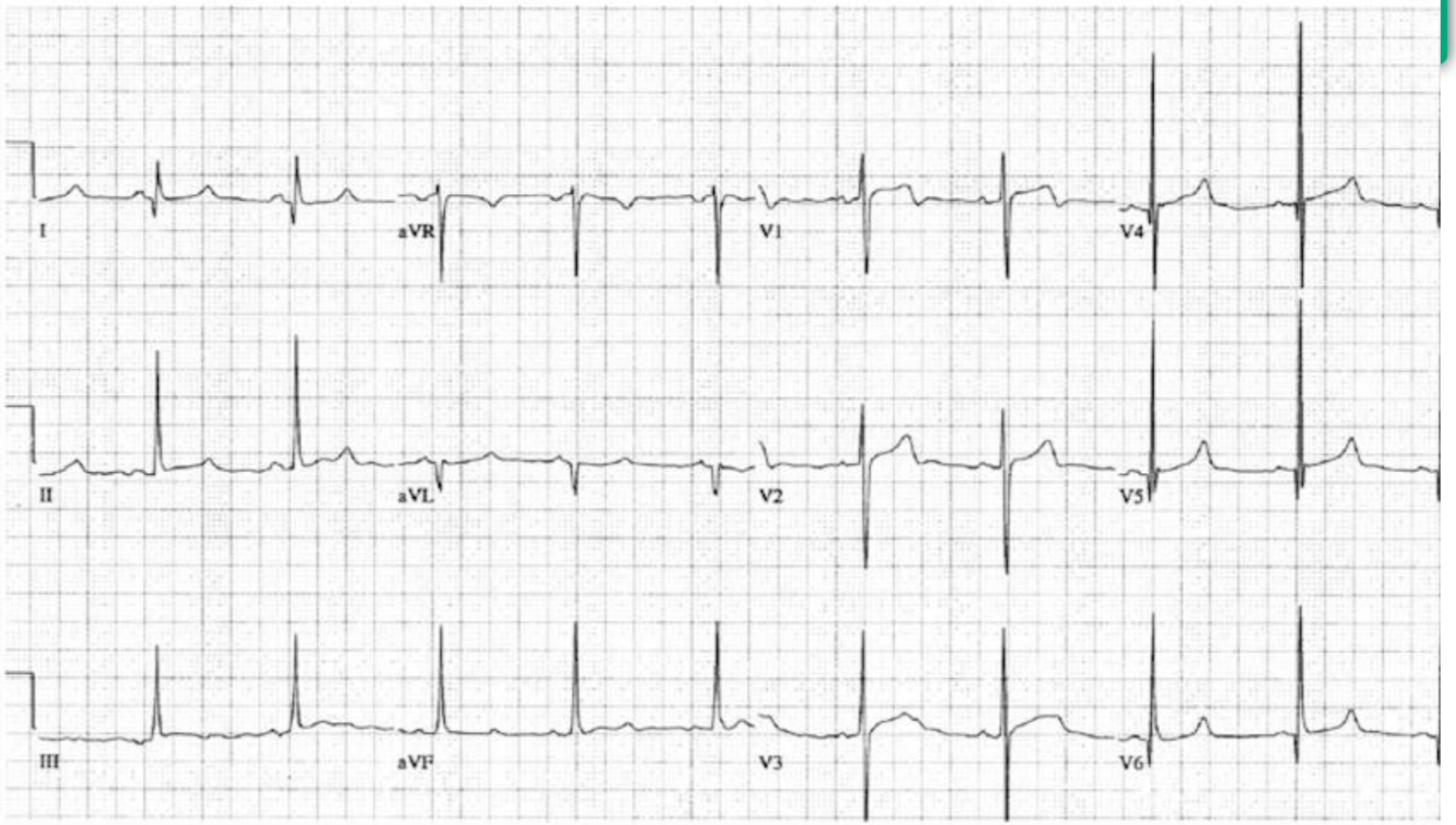


Wang K et al. N Engl J Med 2003;349:2128-2135.

Embolism Who Had a Normal Coronary Angiogram (Tracing 1) and a Patient with Transient ST-Segment Elevation Immediately after Direct-Current (DC) Countershock to the Precordium (Tracing 2)

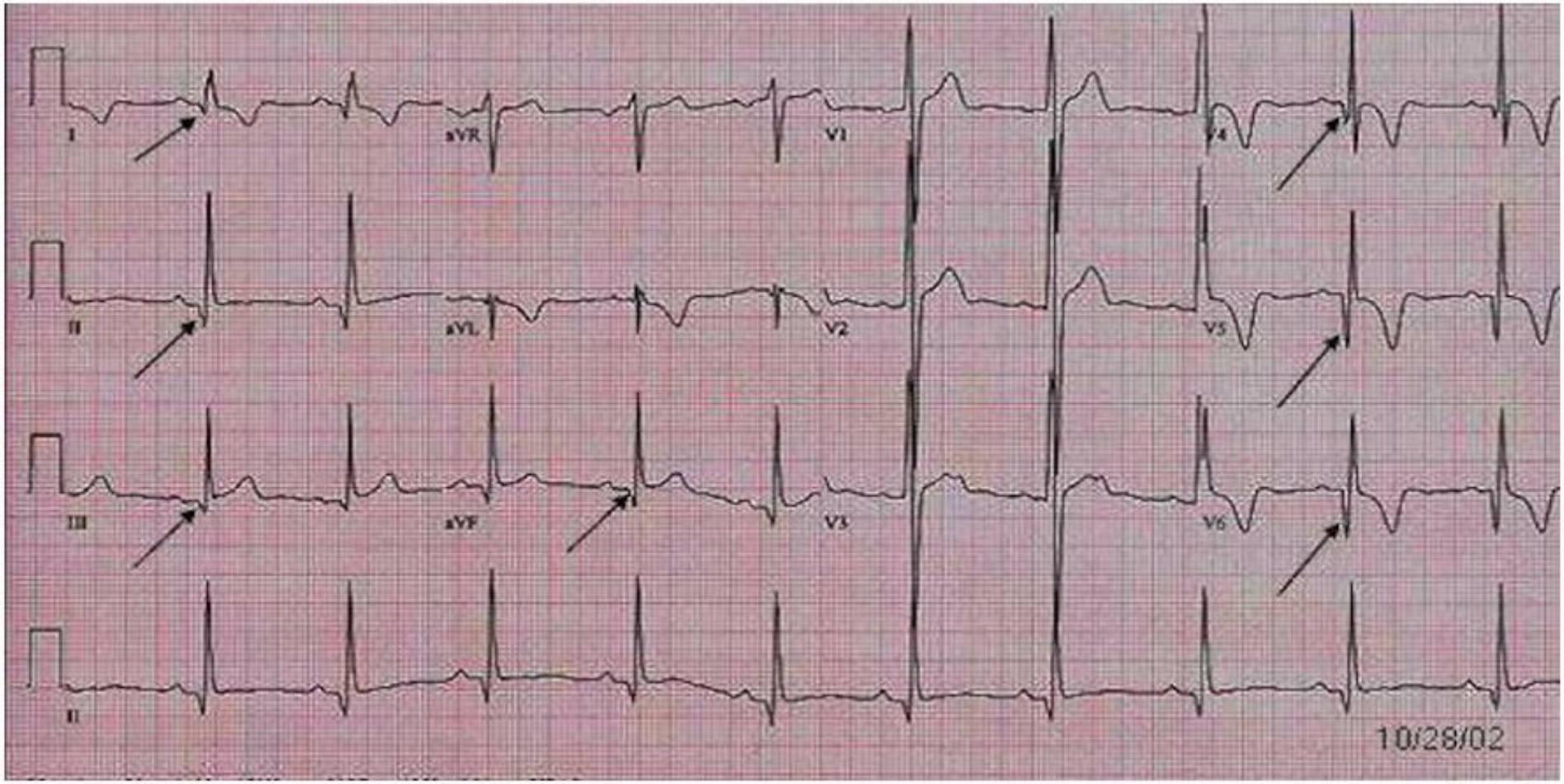


Wang K et al. N Engl J Med 2003;349:2128-2135.



Classic HCM pattern with asymmetrical septal hypertrophy

- Voltage criteria for left ventricular hypertrophy.
- Deep narrow Q waves < 40 ms wide in the lateral leads I, aVL and V5-6.



Dagger-like Q waves

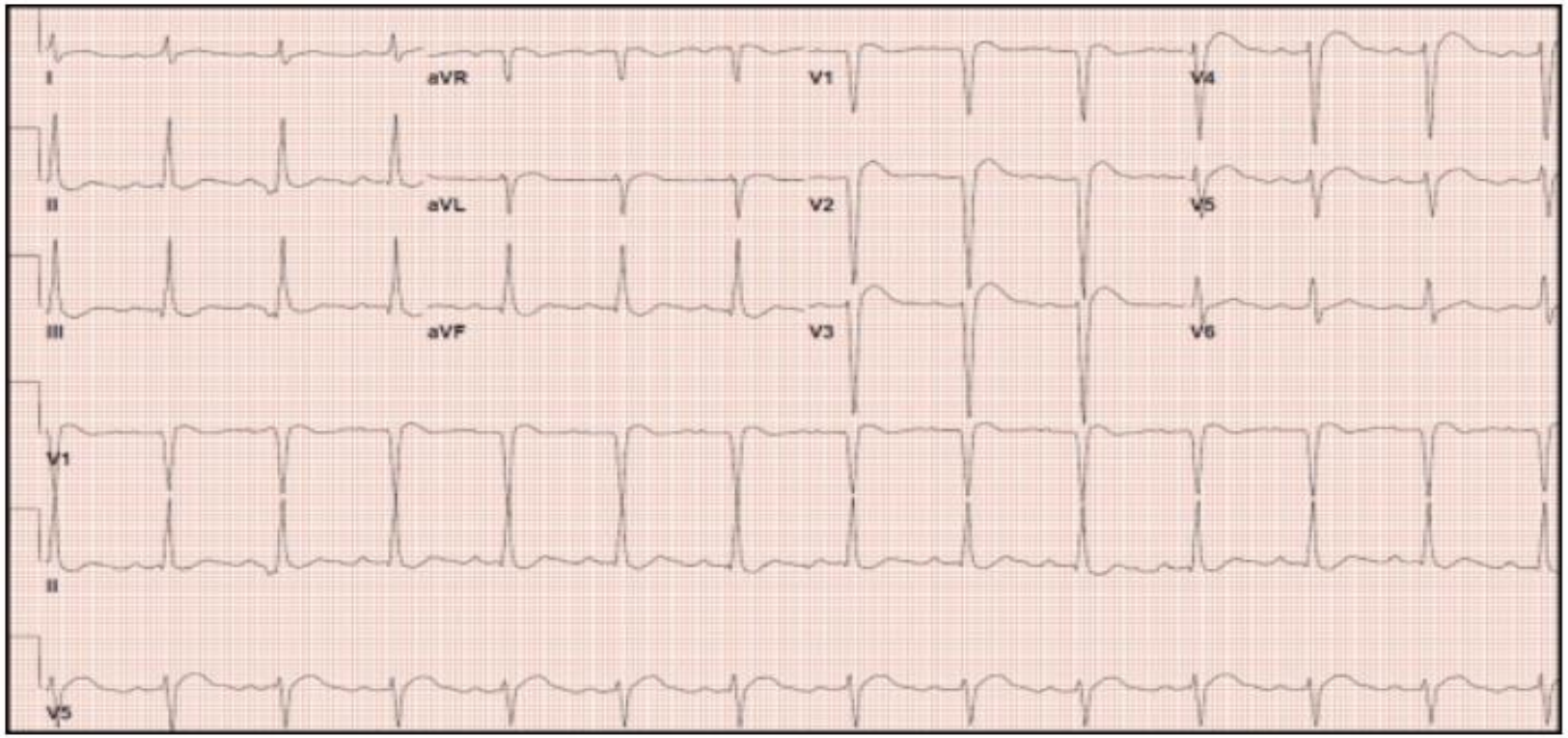


Figure 1

An electrocardiogram ordered on admission showed sinus rhythm, first-degree heart block, normal QT/QT $_C$ intervals, marked scooped ST elevations in leads V1 through V5, and scooped ST depressions in inferior leads.

Milk- alkali syndrome w hypercalcemia

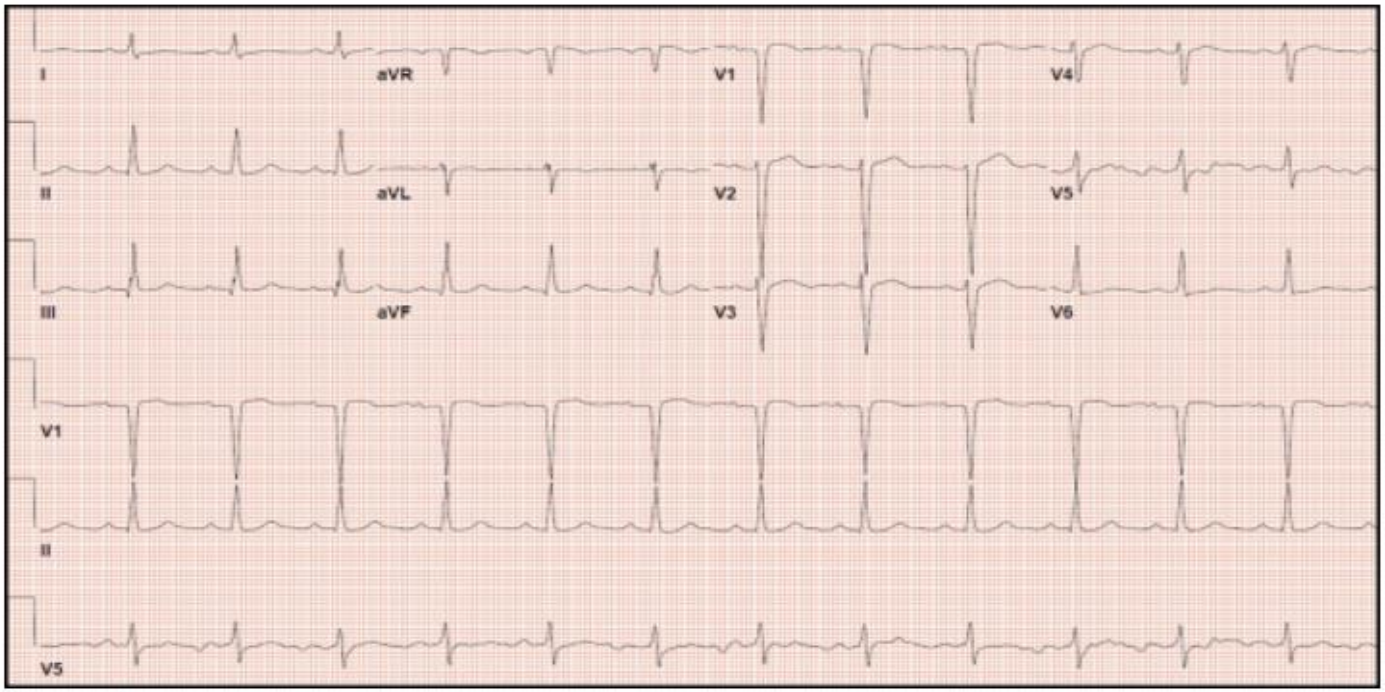
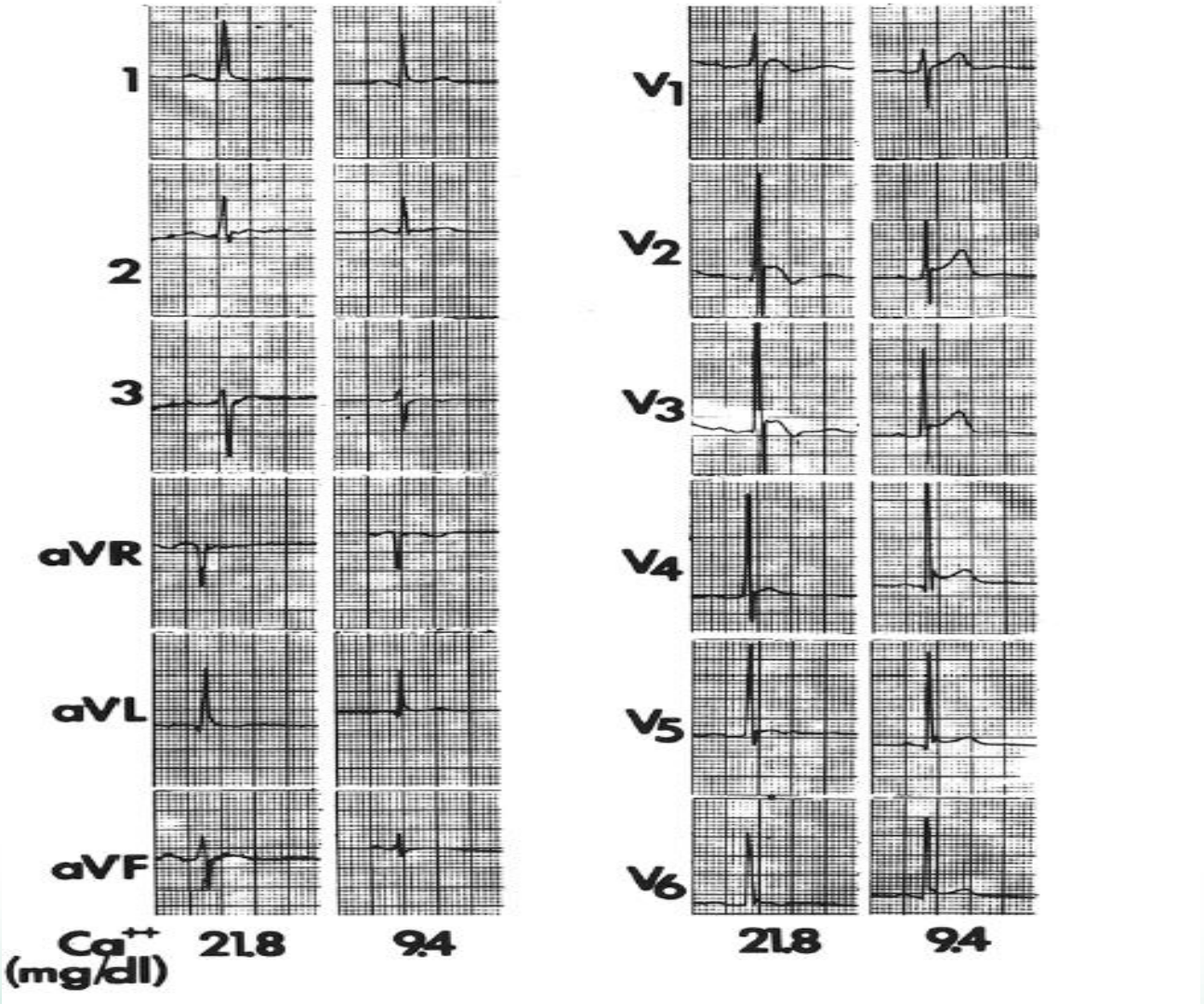
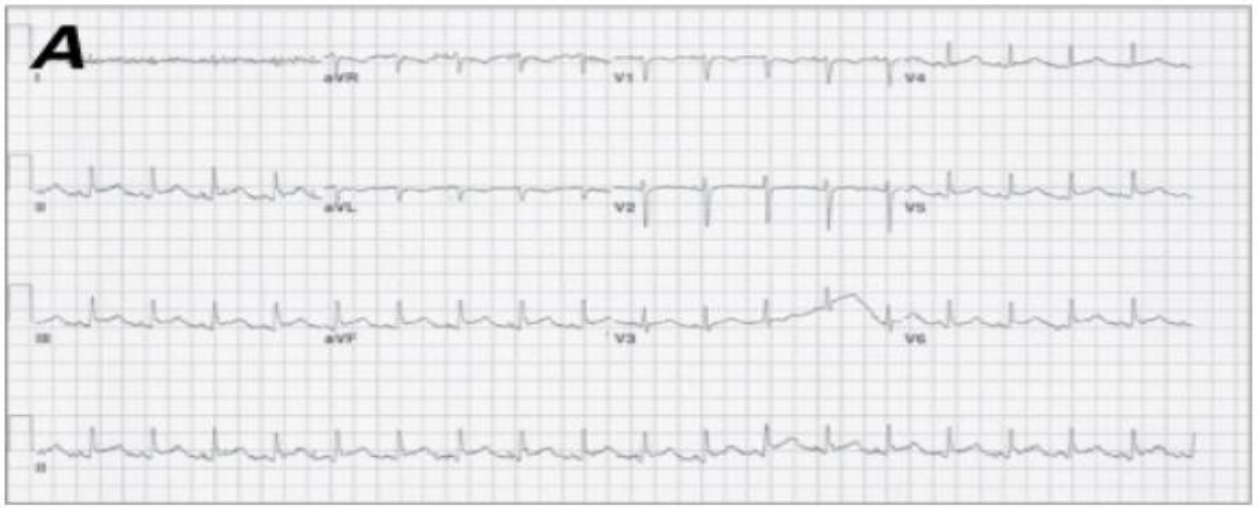
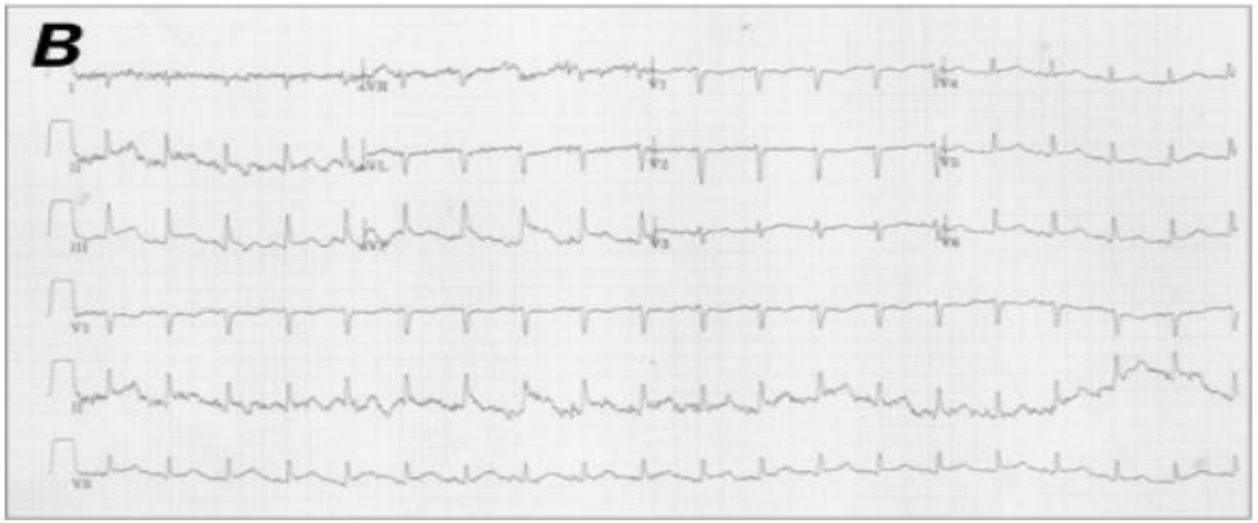


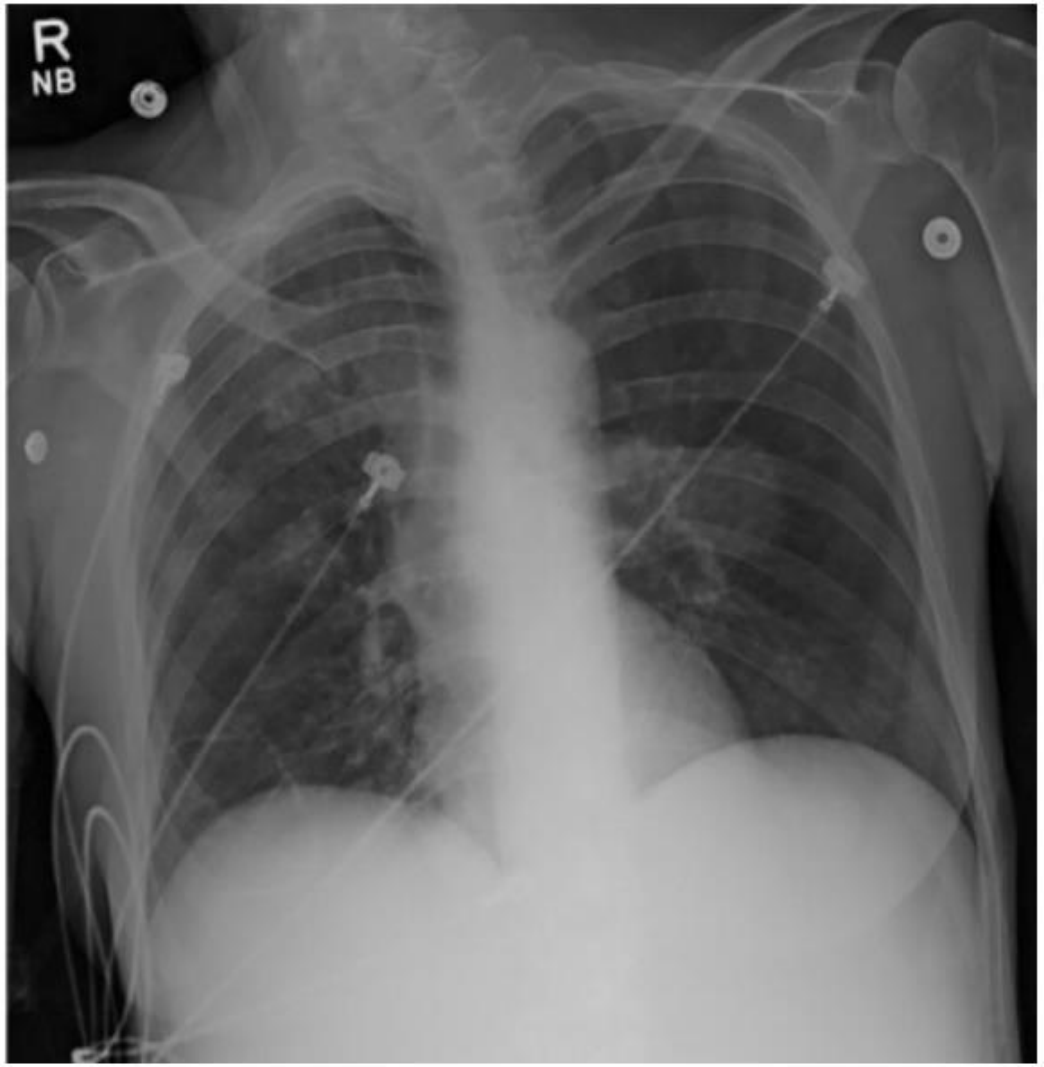
Figure 2

The electrocardiogram obtained prior to discharge demonstrated resolution of first-degree heart block and ST elevations.









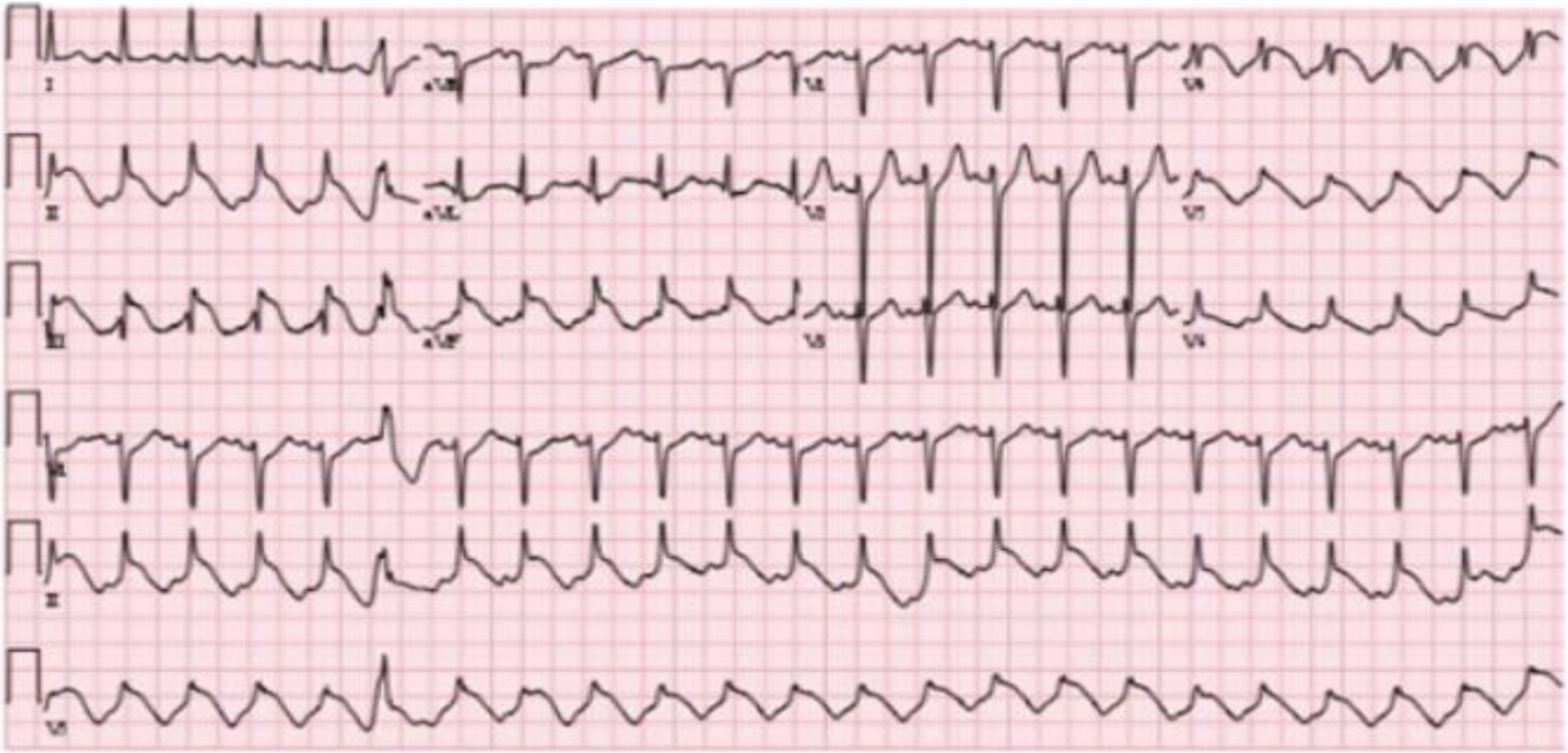
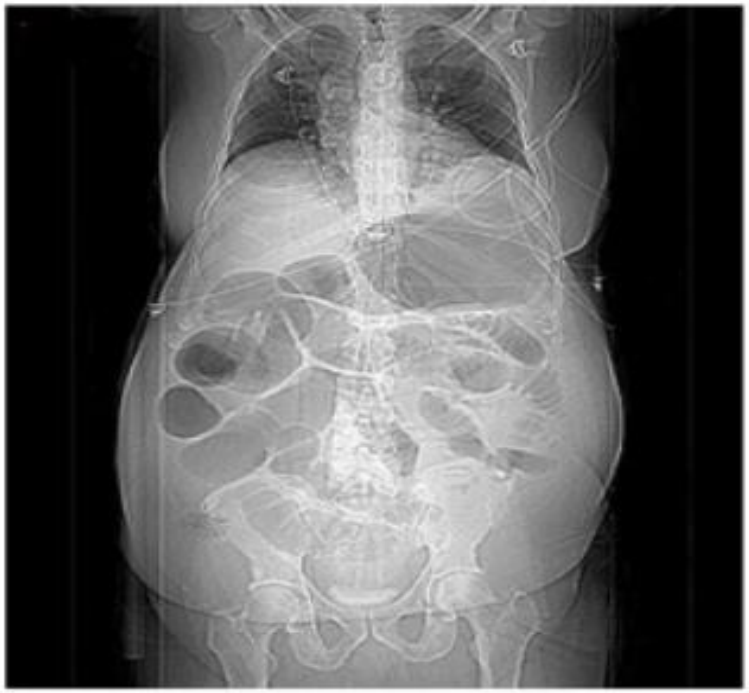


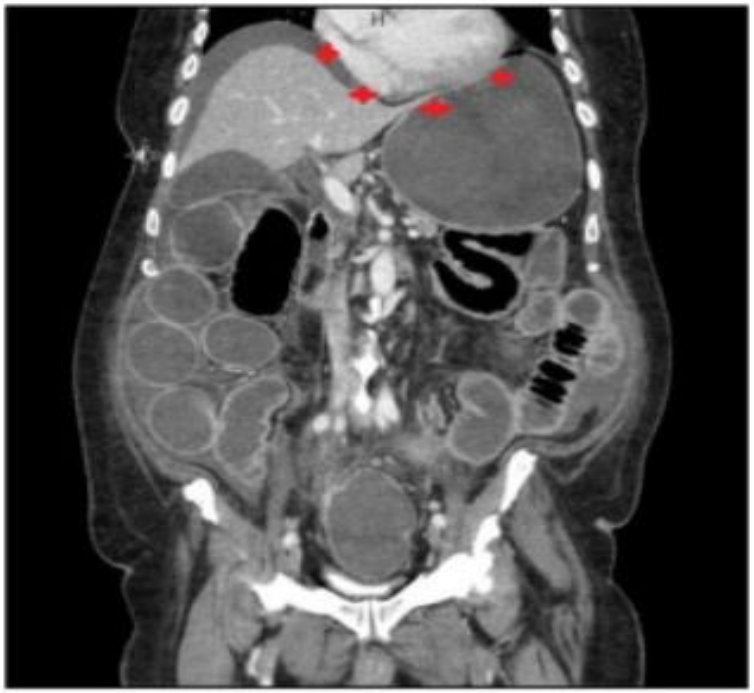
Fig. 1

12 lead ECG showing ST segment elevation in leads II, III, aVF, V-4, V-5 & V-6.

Ogilvie’s syndrome



A) Scout Image



B) CT scan Abd/Pelvis
Coronal view

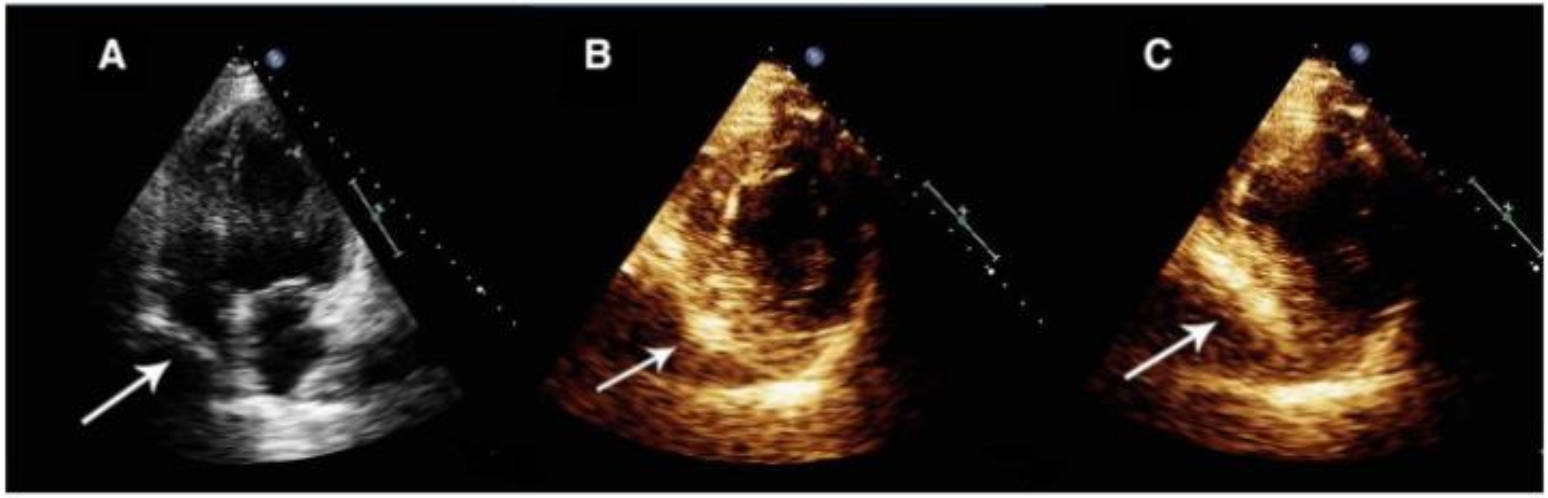
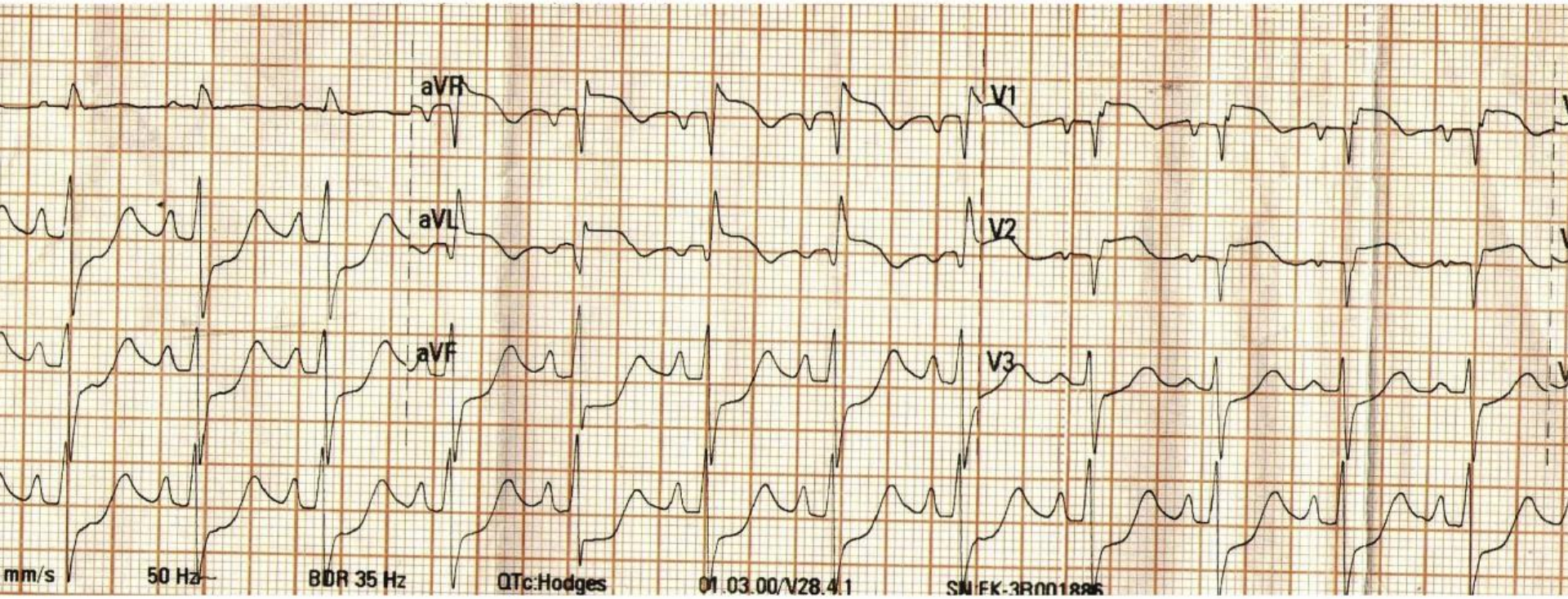
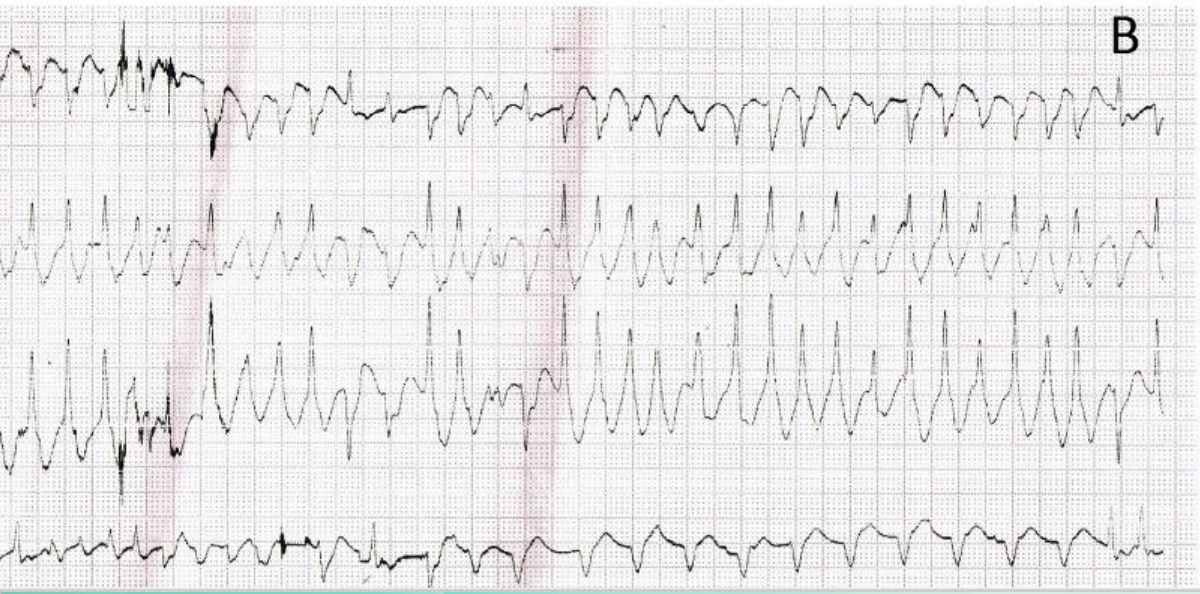
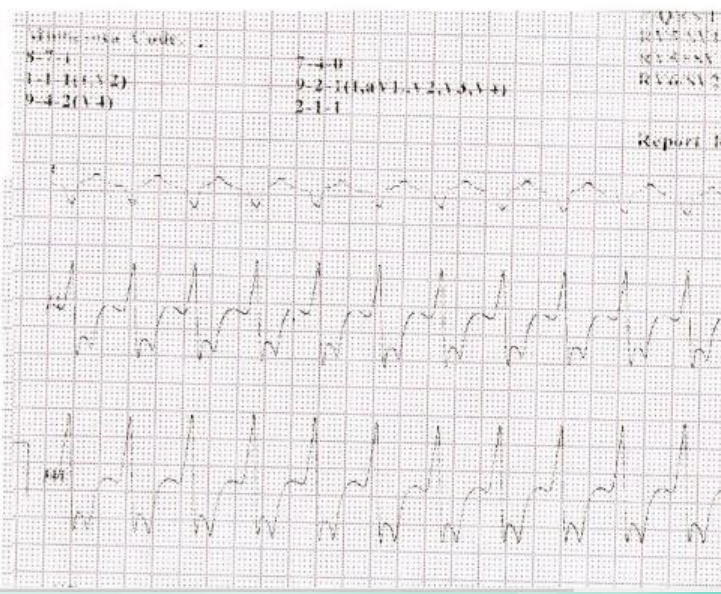


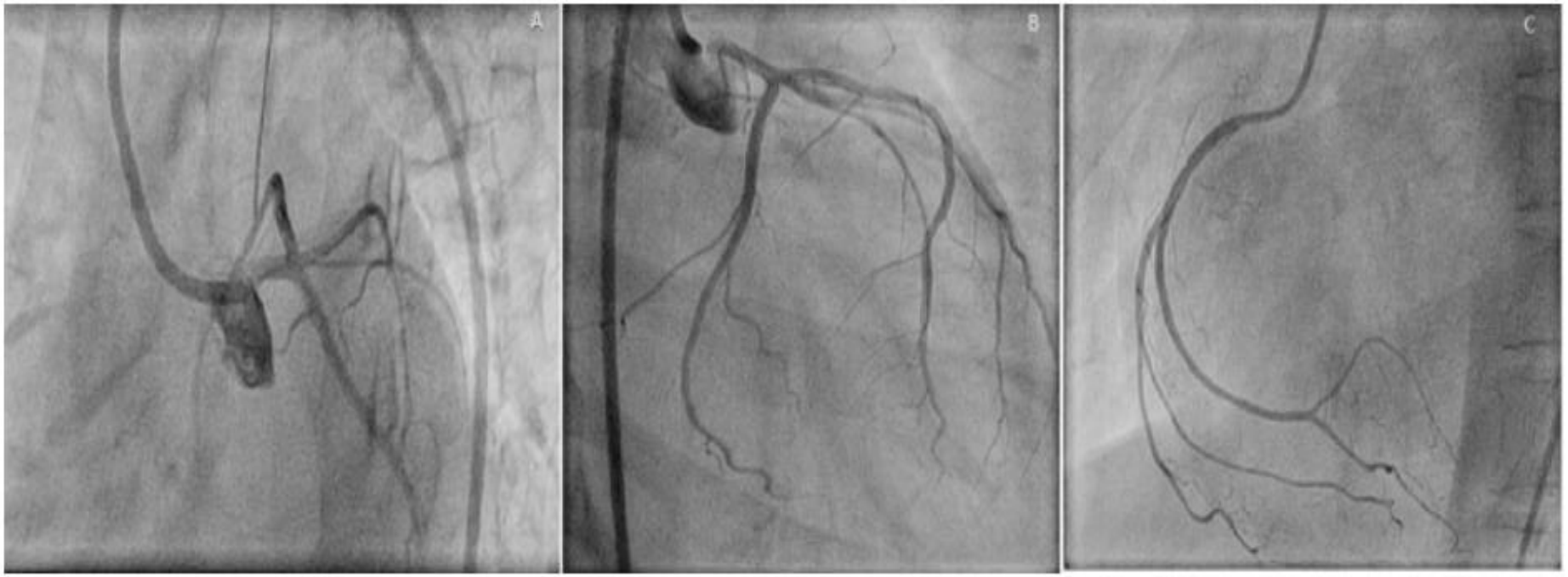
Fig. 2

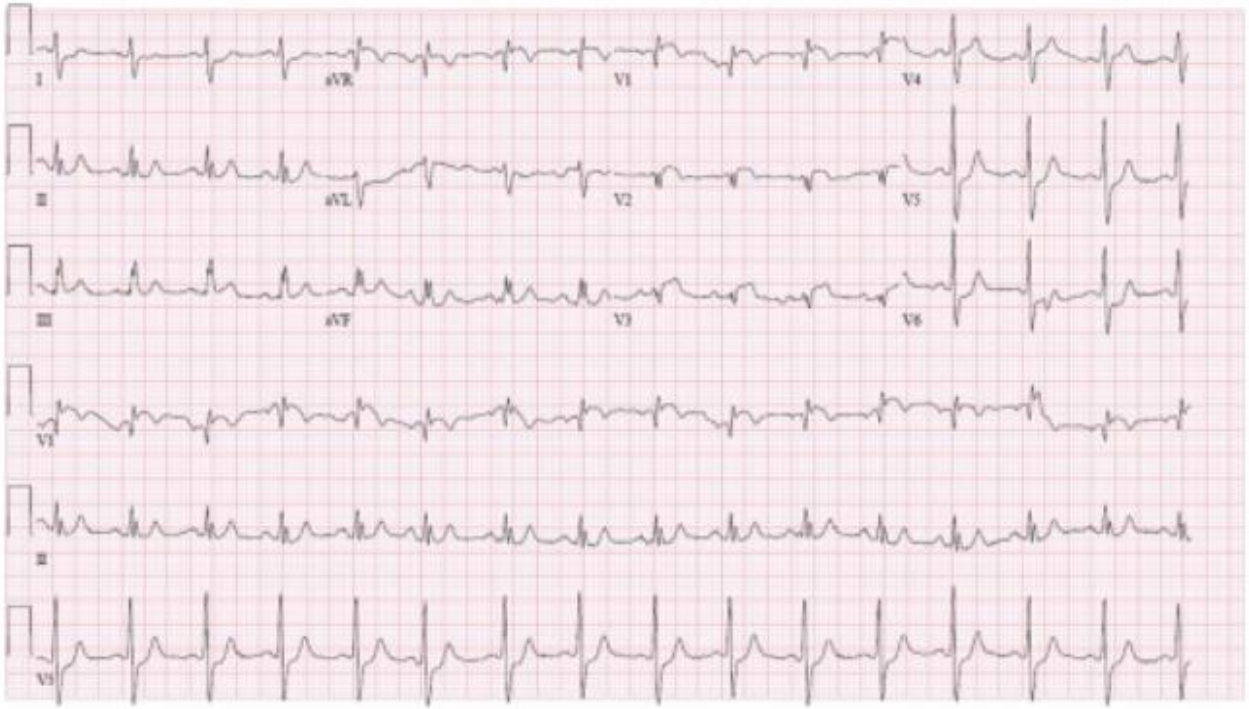
A: White arrows showing indentation of right atrial free wall. B: Preserved geometry of LV cross section during systole. C: Deformation of inferior infero-lateral segment secondary to elevated hemi-diaphragm.


aVR
V1
aVL
V2
aVF
V3
mm/s
50 Hz
BDR 35 Hz
QTc:Hodges
01.03.00/V28.4.1


B

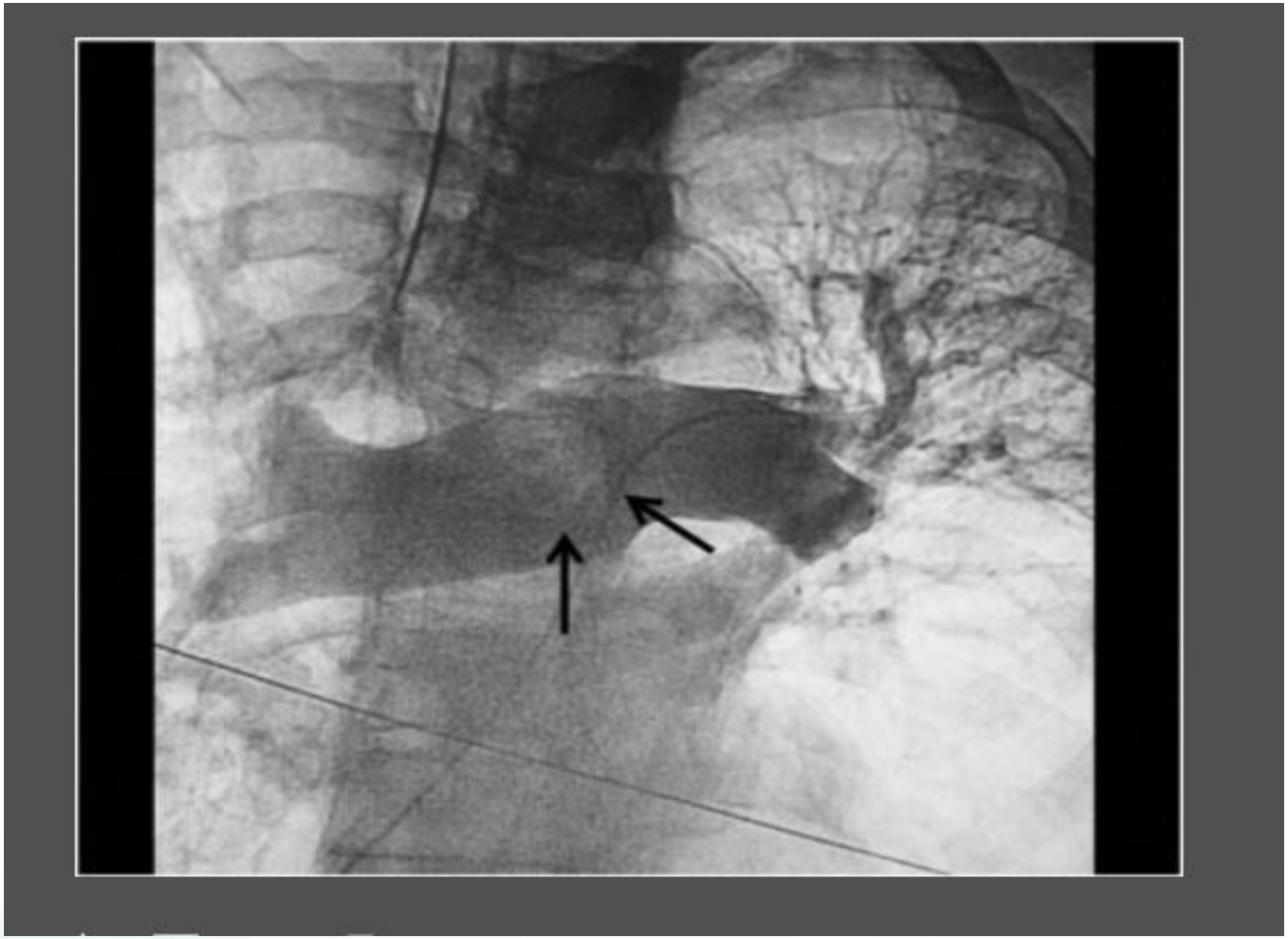



A
B
C



Supplementary Fig. 1

Twelve-lead ECG demonstrating 2-mm ST elevation in V_1 to V_3 and 1-mm ST depression in lead I, along with 2-mm ST depression in V_5 and 3-mm depression in V_6 as well as a new incomplete right bundle-branch block.



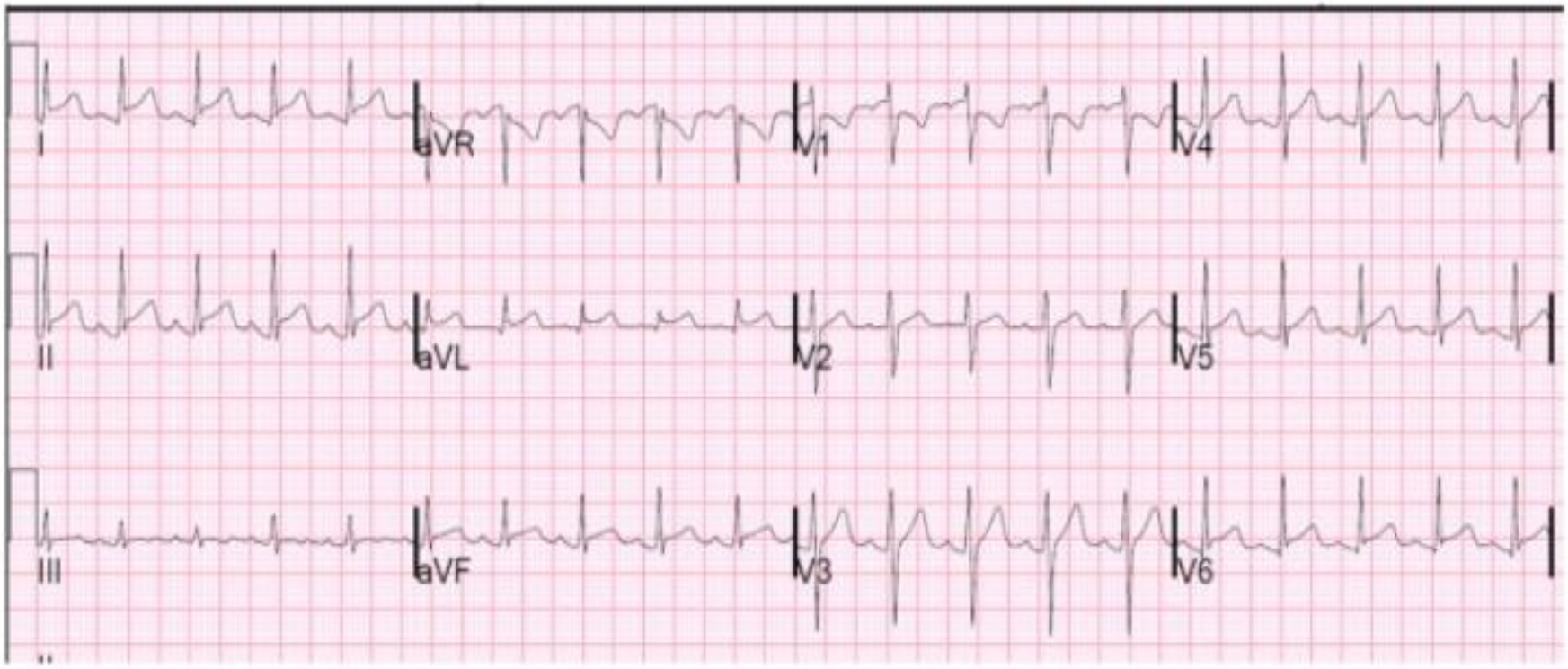


Fig. 3

Example of ECG of patient with pericarditis. Although there is clear ST elevation in the inferior leads, lead aVL lacks ST depression.

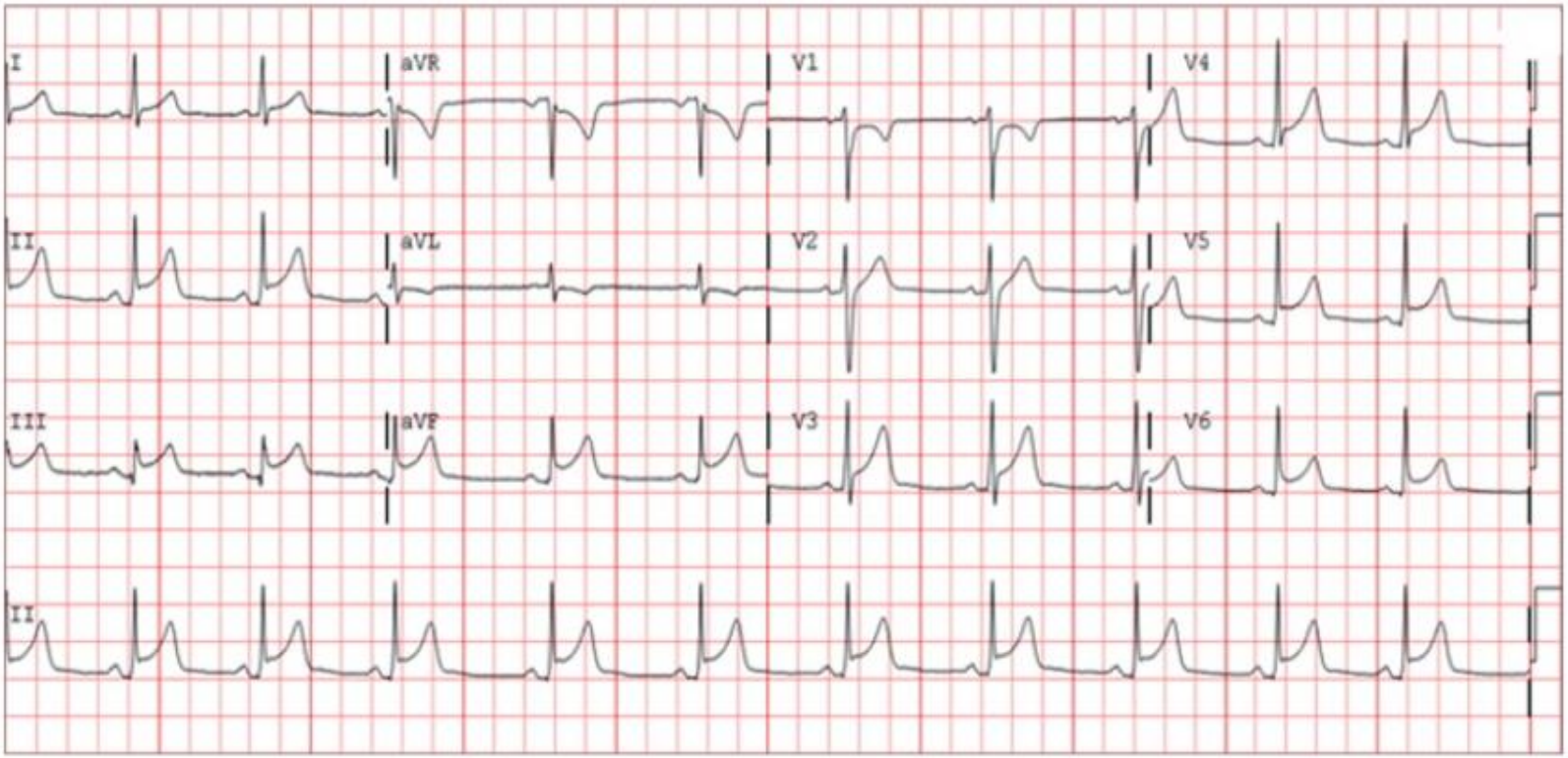
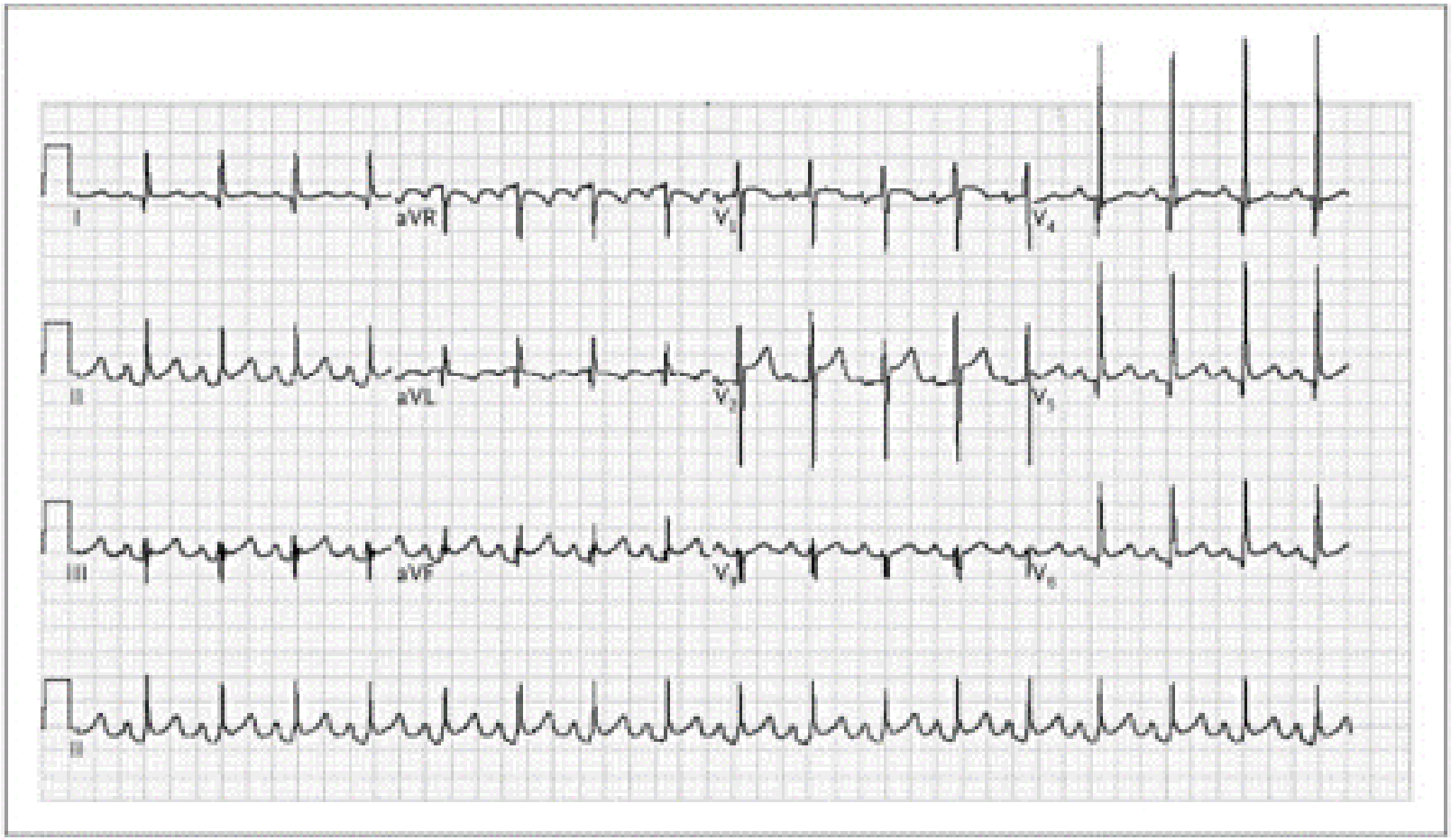


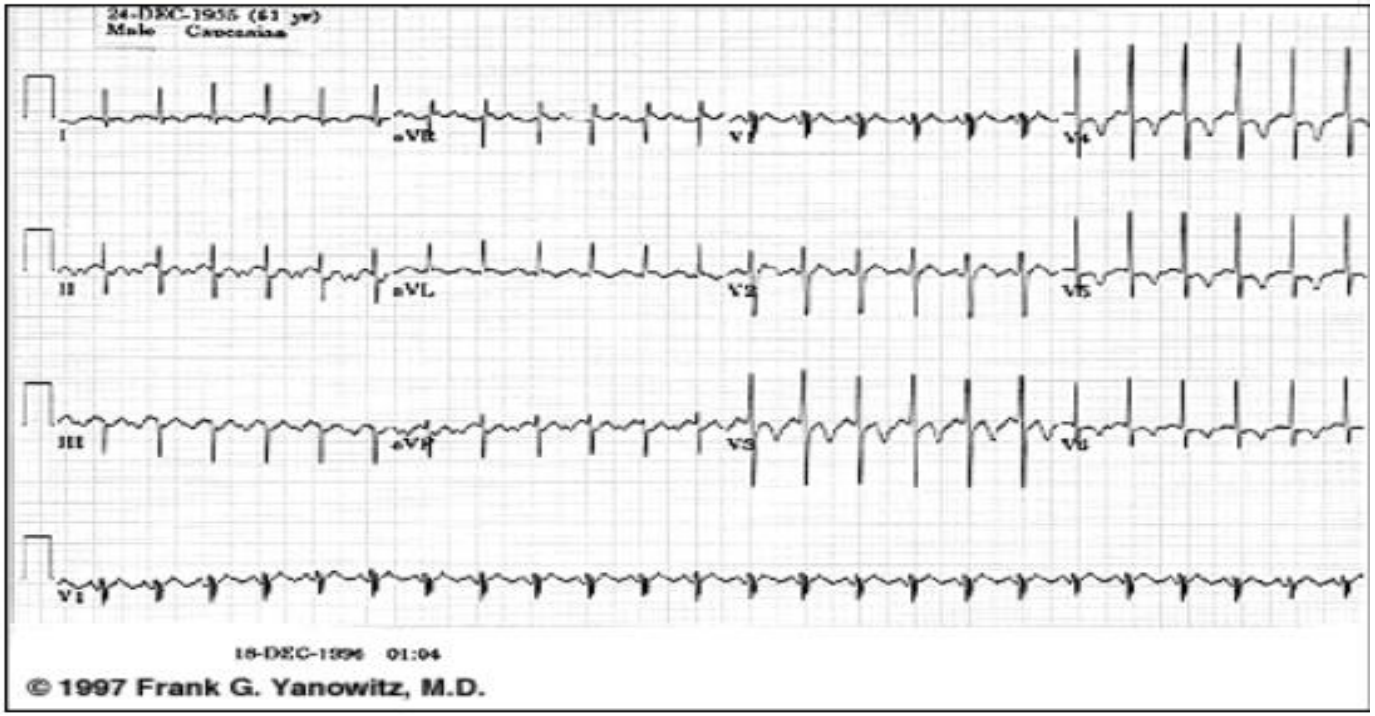
Fig. 4

ECG, few hours later, showing prominent diffuse ST-segment elevation with PR-segment depression.

A 12-Lead Electrocardiogram from a Patient with Acute Pericarditis, Demonstrating Widespread ST-Segment Elevation and PR-Segment Depression.



Lange RA, Hillis LD. N Engl J Med 2004;351:2195-2202.



Atrial Flutter With 2:1 AV Conduction-KH

Frank G.Yanowitz, M.D.

Atrial flutter with 2:1 AV block is one of the most frequently missed ECG rhythm diagnoses because the flutter waves are often hard to find. In this example two

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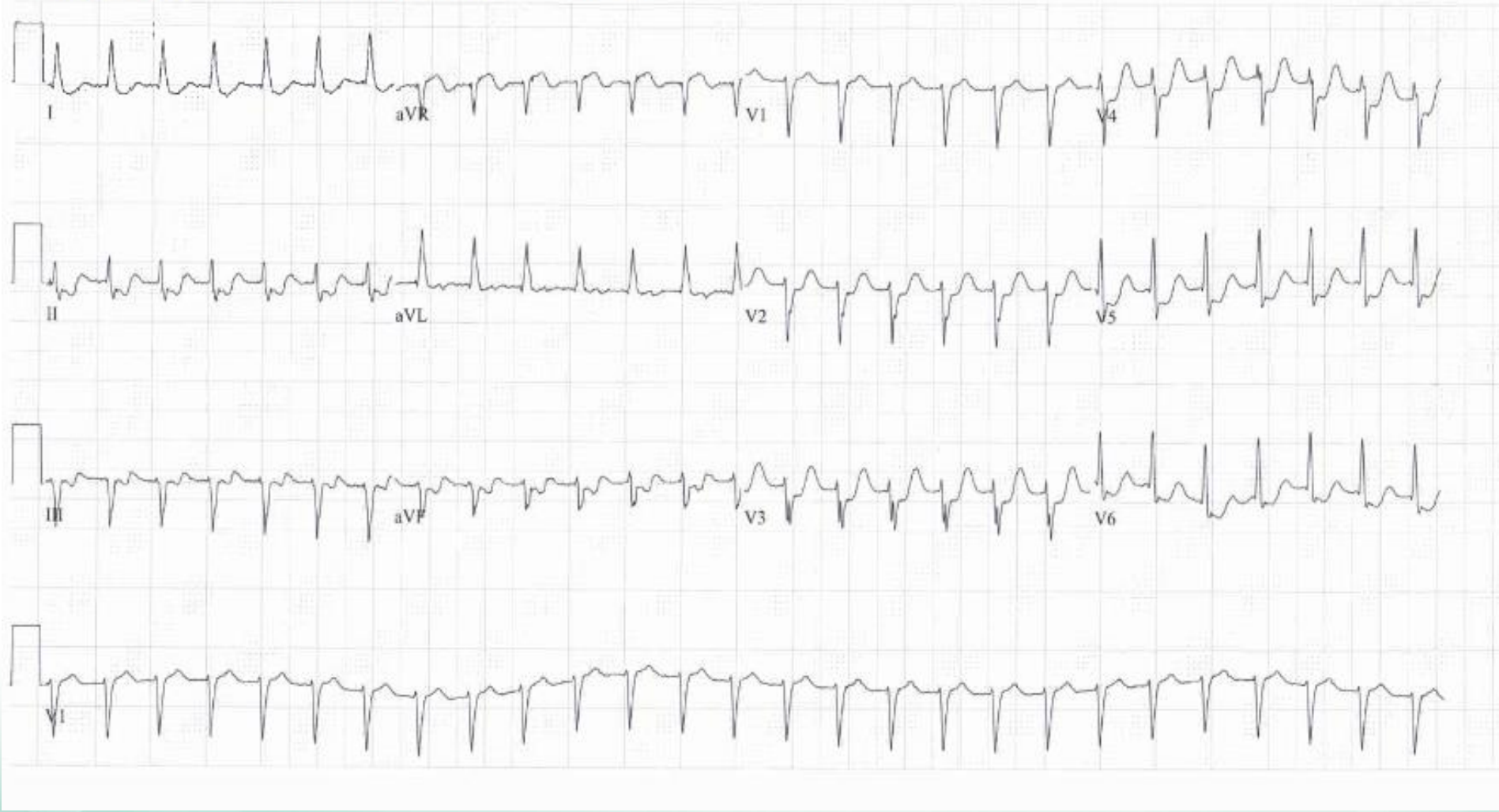
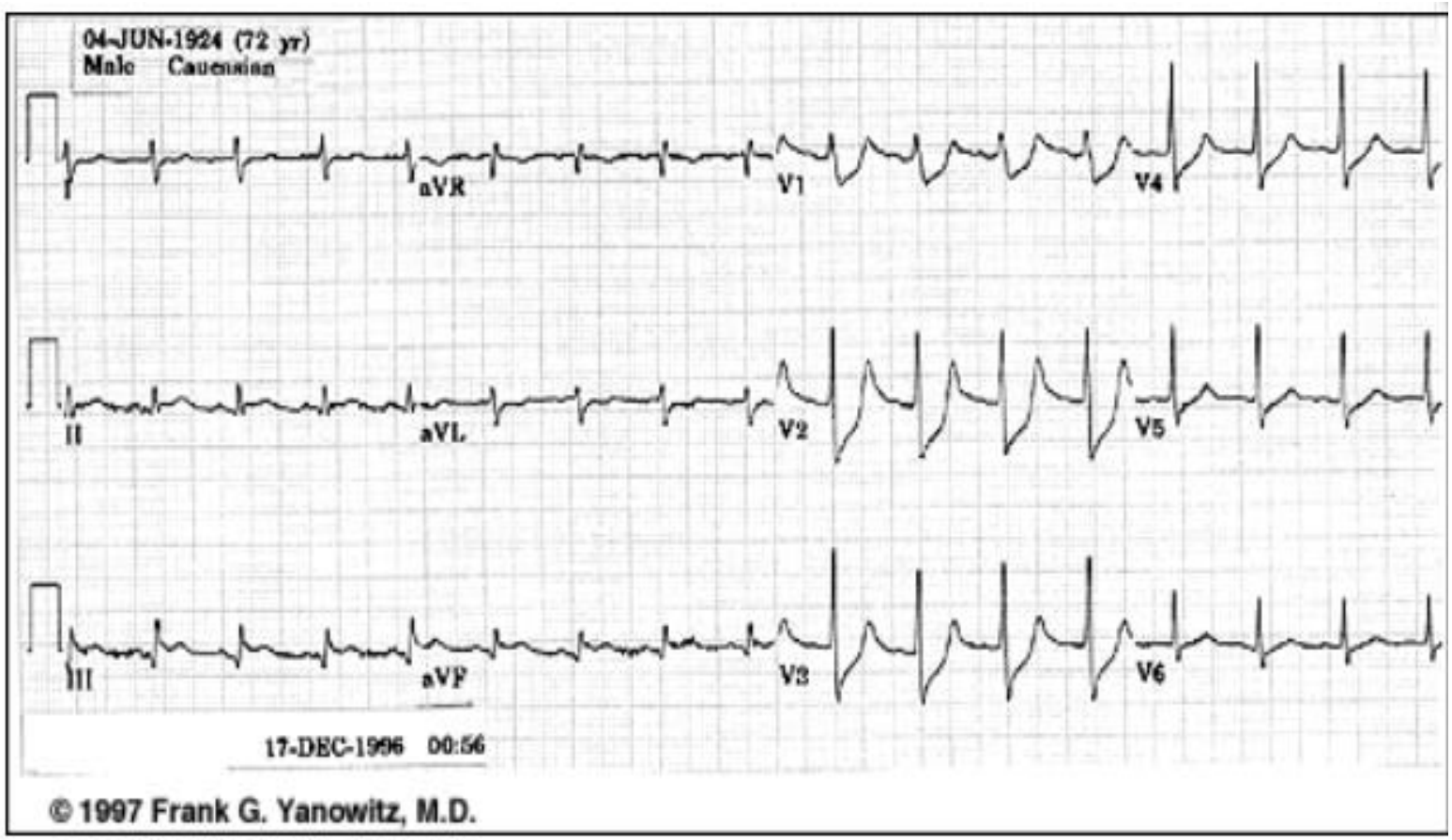
Example (b) – Atrial Flutter with 2:1 Block




04-JUN-1924 (72 yr)
Male Caucasian
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
17-DEC-1996 00:56
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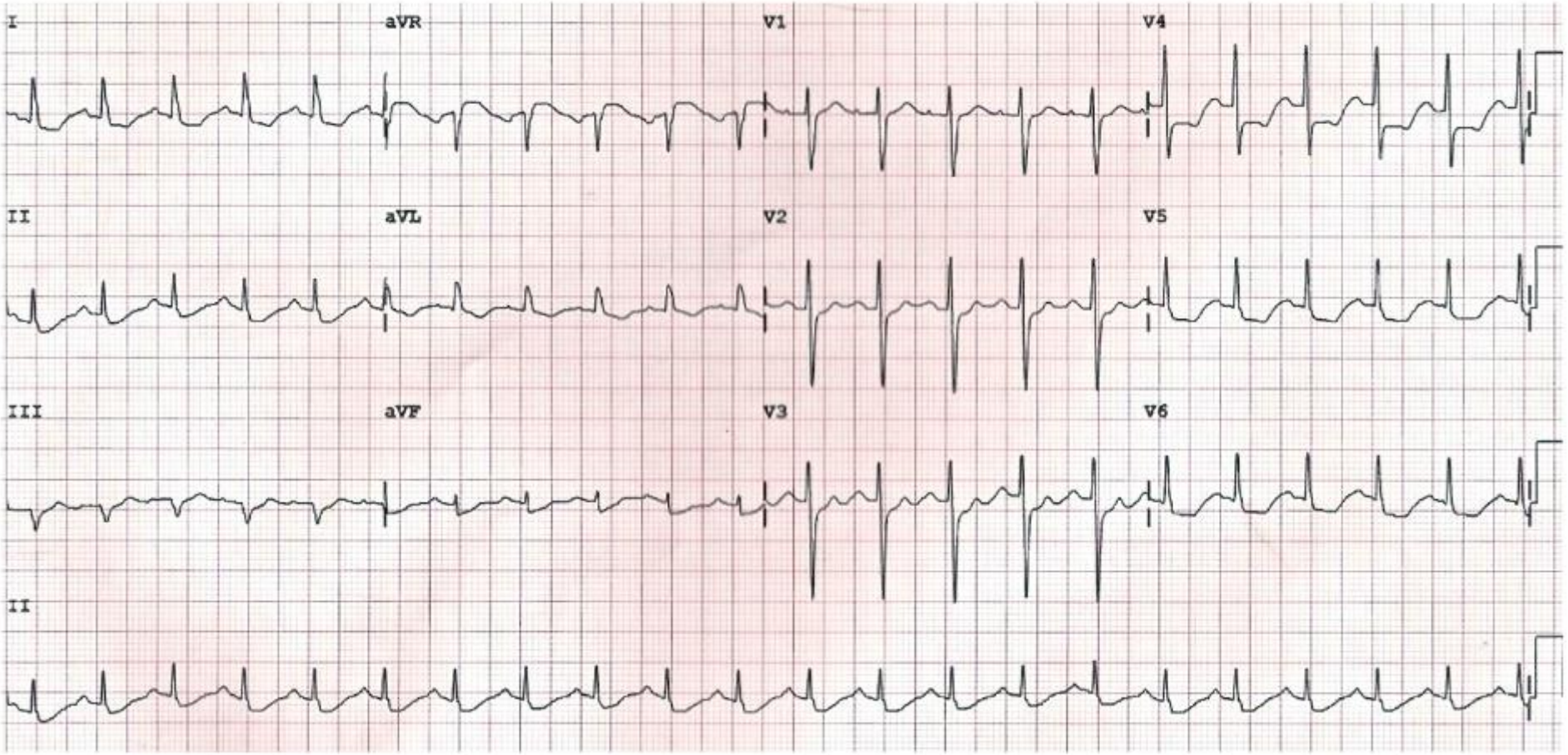

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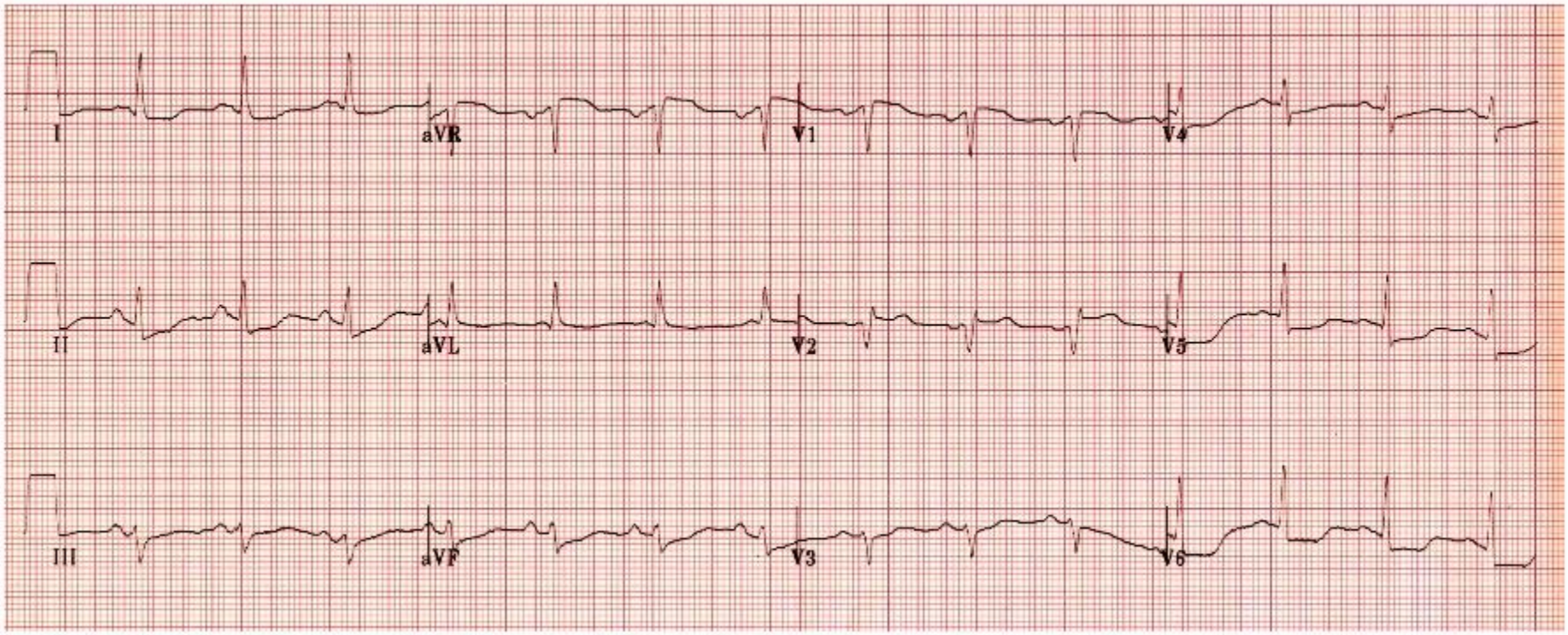

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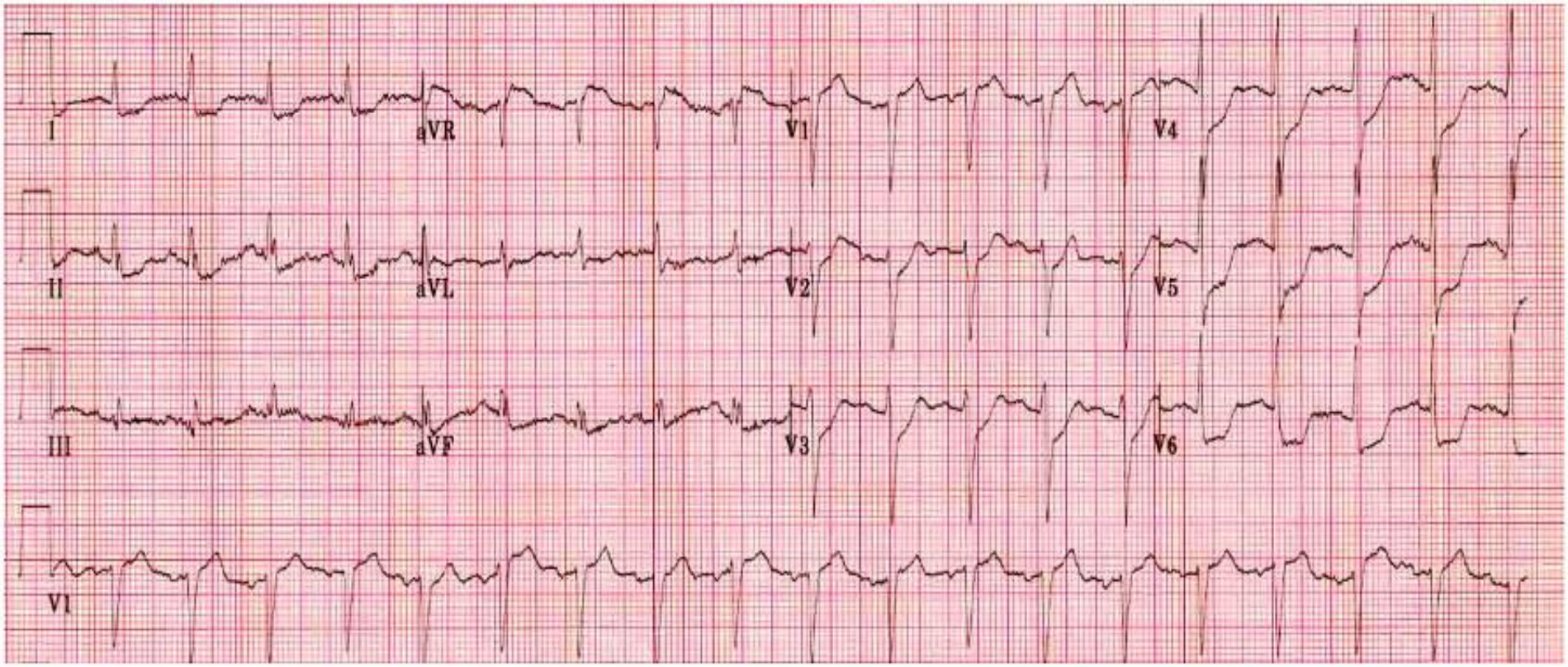

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Typical ECG findings with left main coronary artery (LMCA) occlusion:

- Widespread horizontal ST depression, most prominent in leads I, II and V4-6
- ST elevation in aVR ≥ 1mm
- ST elevation in aVR ≥ V1

ST Elevation in aVR may also be seen with:

- Proximal left anterior descending artery (LAD) occlusion
- Severe triple-vessel disease (3VD)
- Diffuse subendocardial ischaemia – e.g. due to O2 supply/demand mismatch, following resuscitation from cardiac arrest

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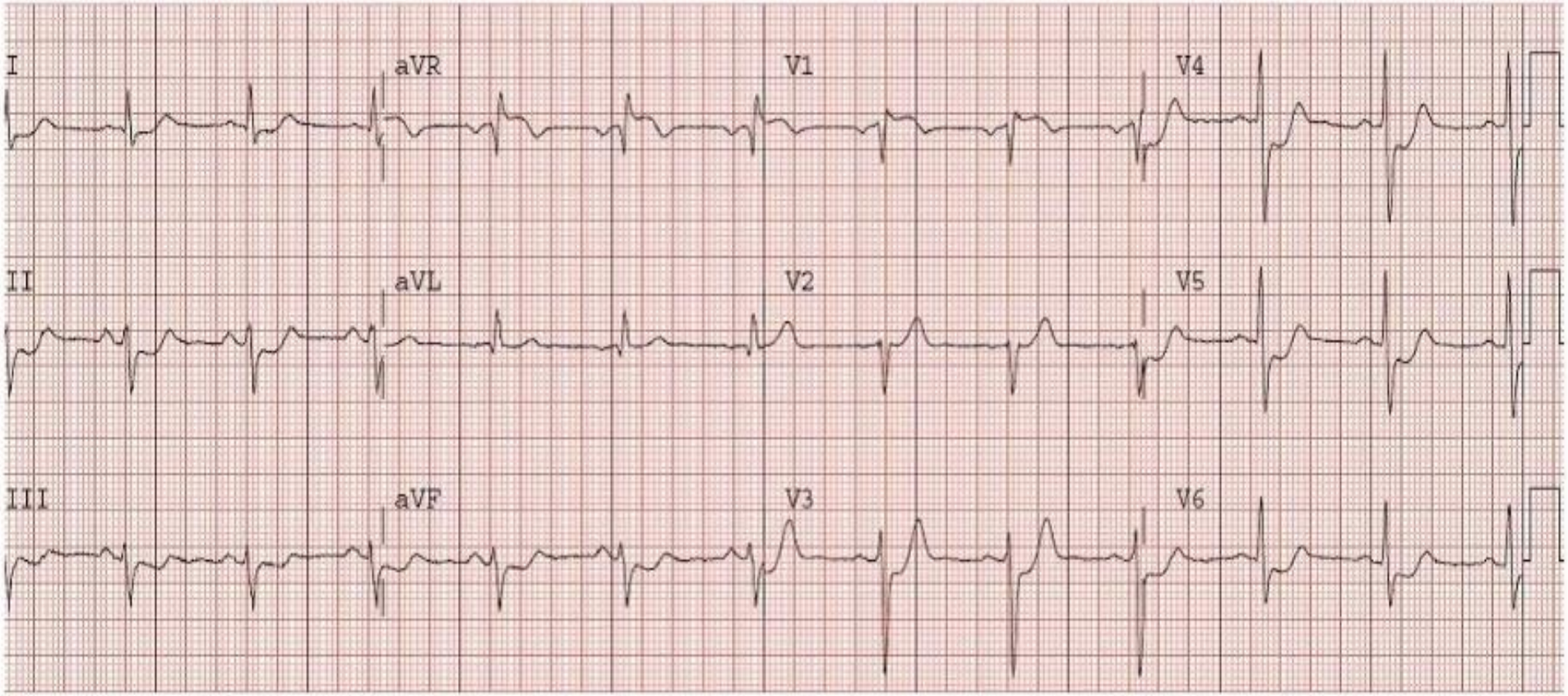
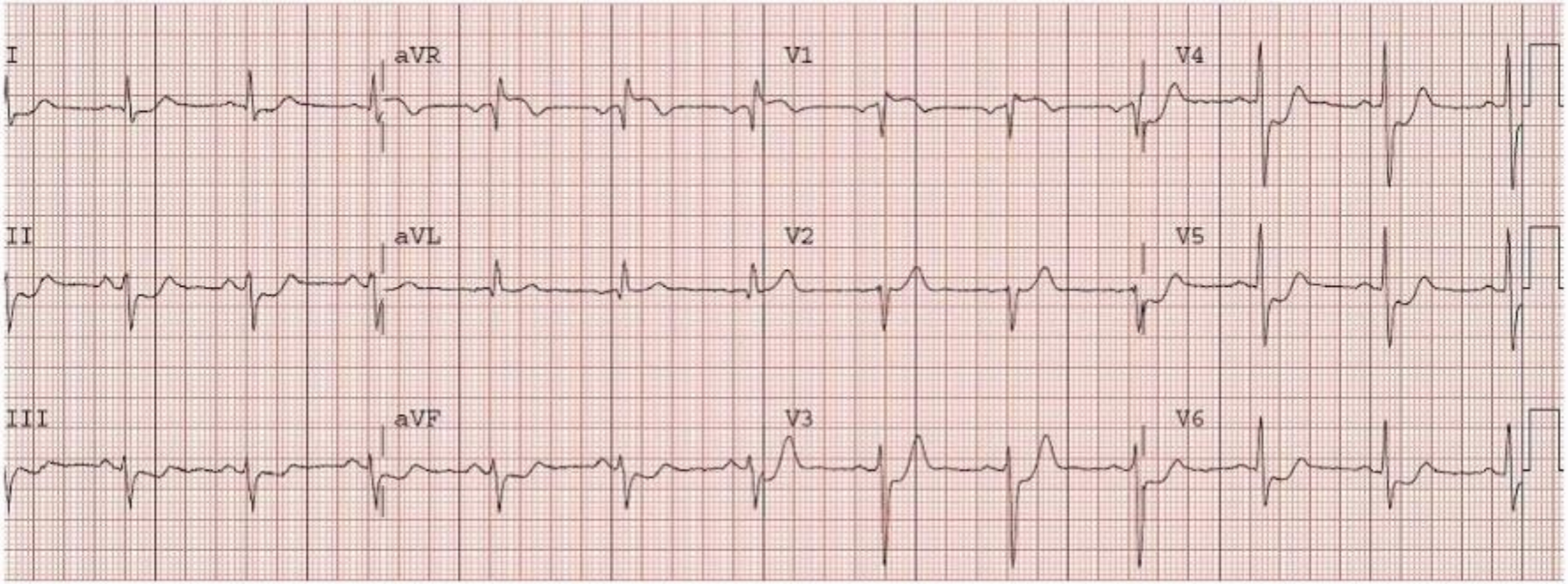
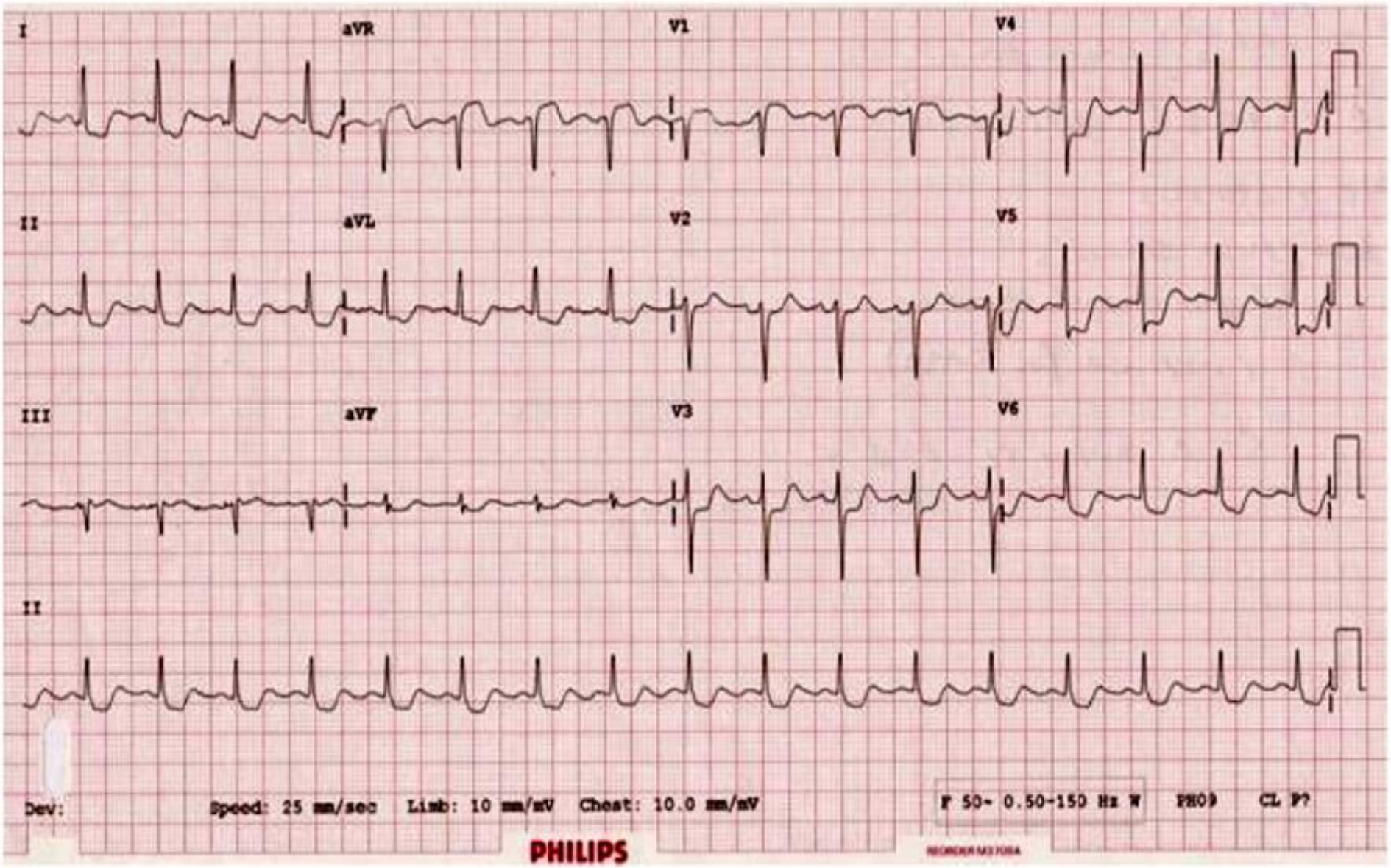


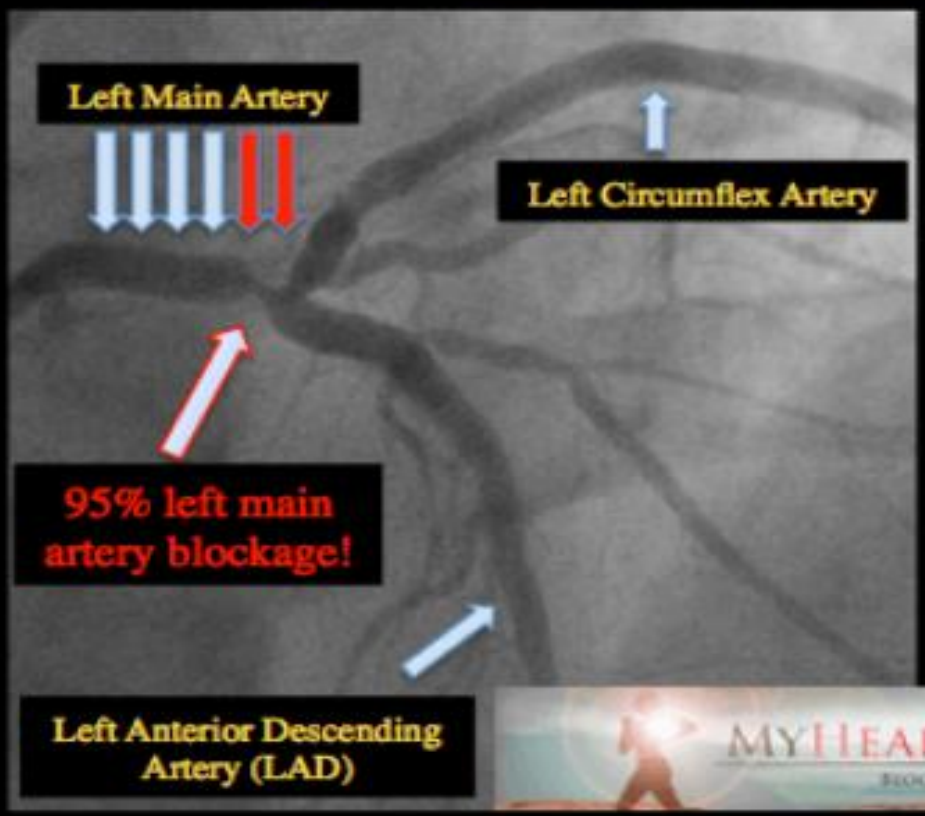


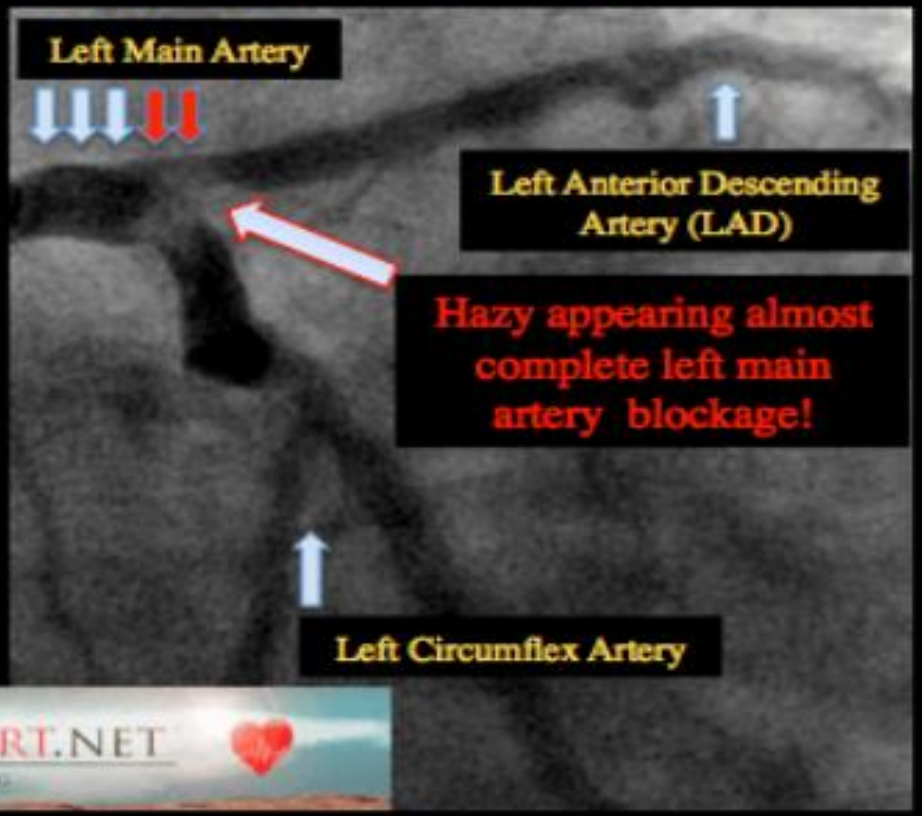
Proximal LAD occlusion

- ST elevation in aVR and V1 of similar magnitude.
- Widespread ST depression (V3-6, I, II, III, aVF)


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Dev:
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10.0 mm/mV
F 50~ 0.50-150 Hz W
PHO
CL F?
PHILIPS

The Widowmaker of All Widowmakers – Critical Left Main Artery Disease





These are images from two different patients. The left main artery, as can been seen above, gives rise to the two main arteries that supplies the heart, the left anterior descending artery (LAD) and the circumflex artery (Lcx). If the left main artery goes totally down, so do the other arteries! Chances of survival are slim with a left main artery heart attack. This is why it's the mother of all Widowmakers.

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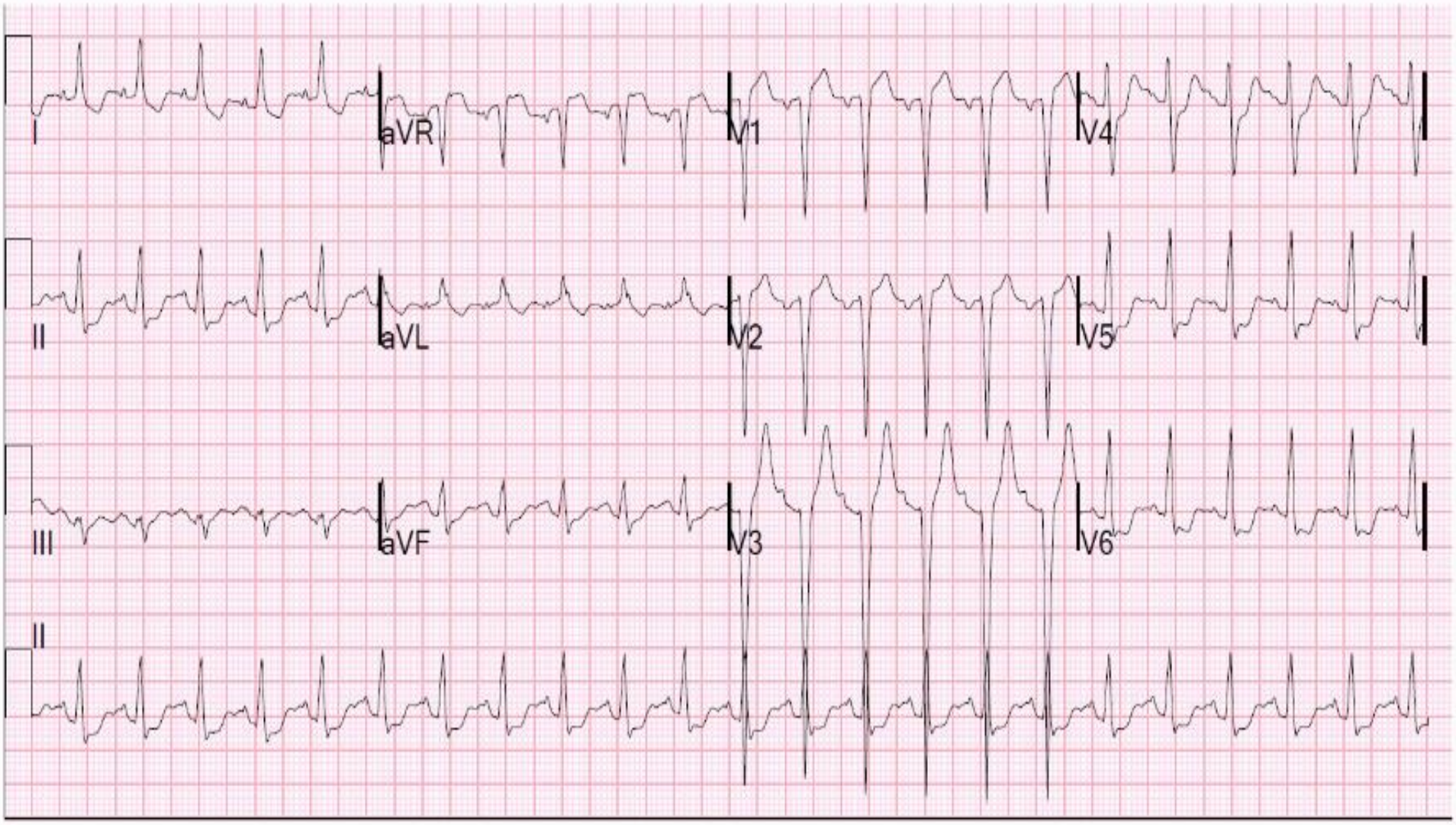
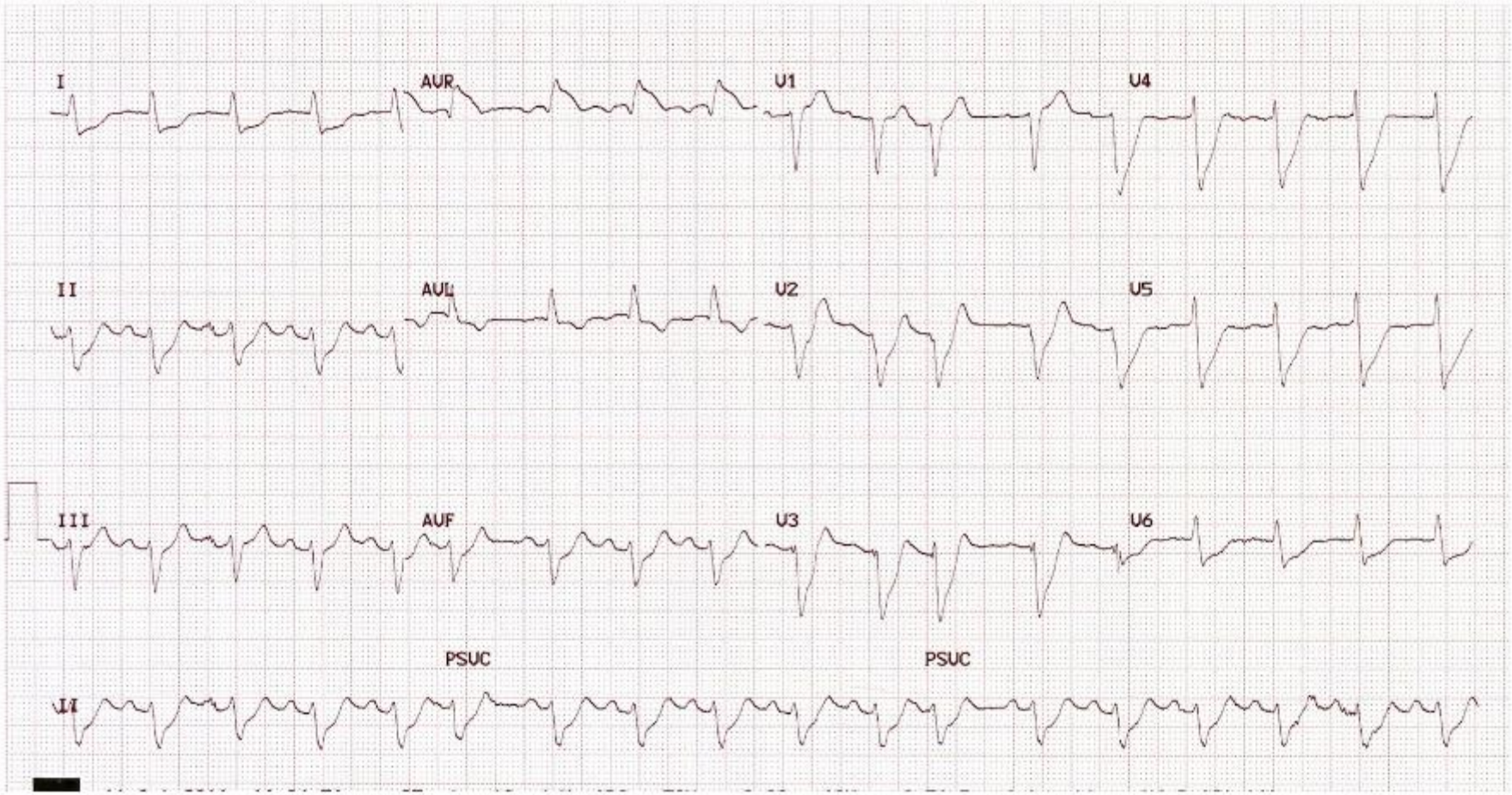
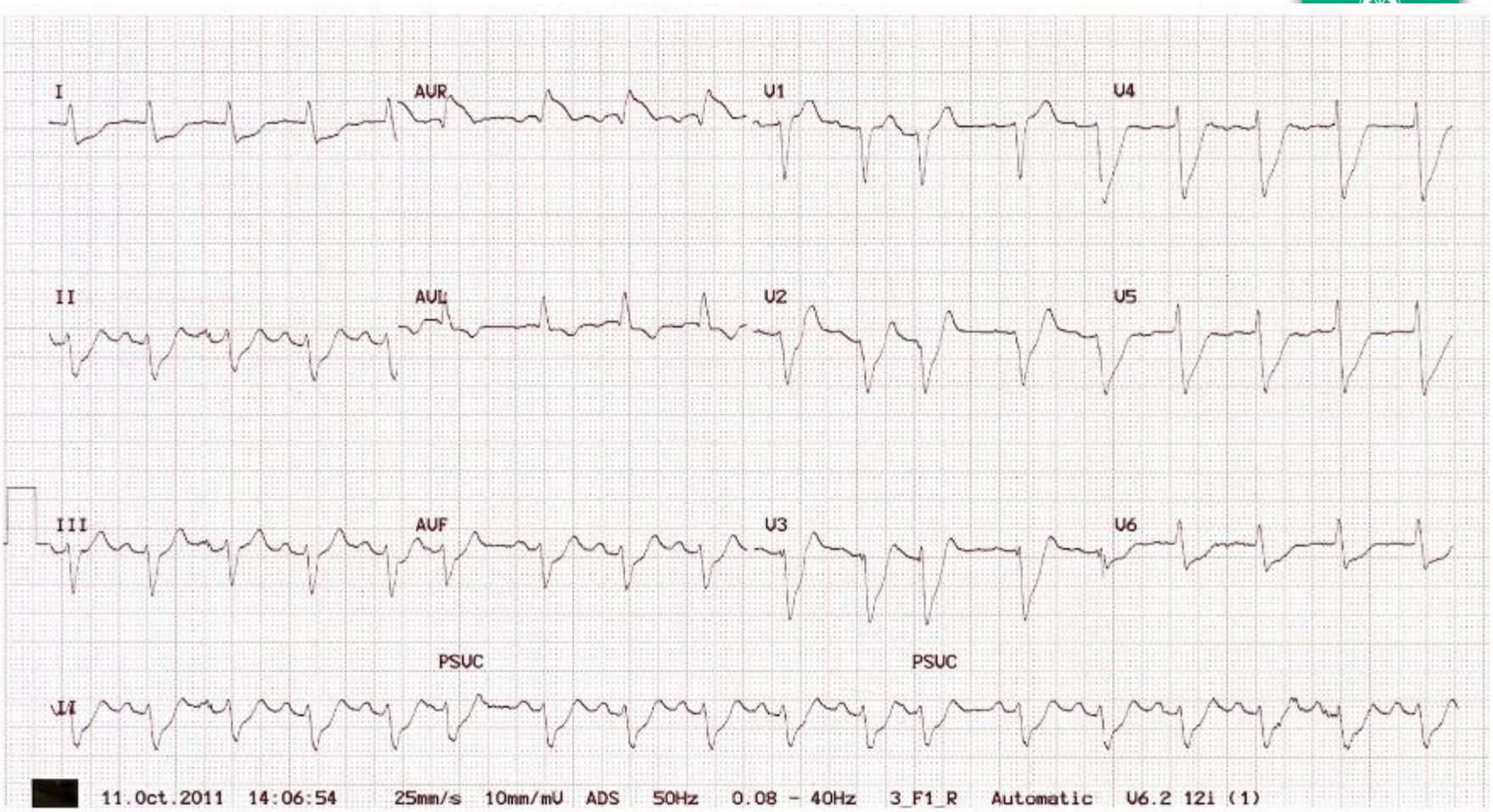
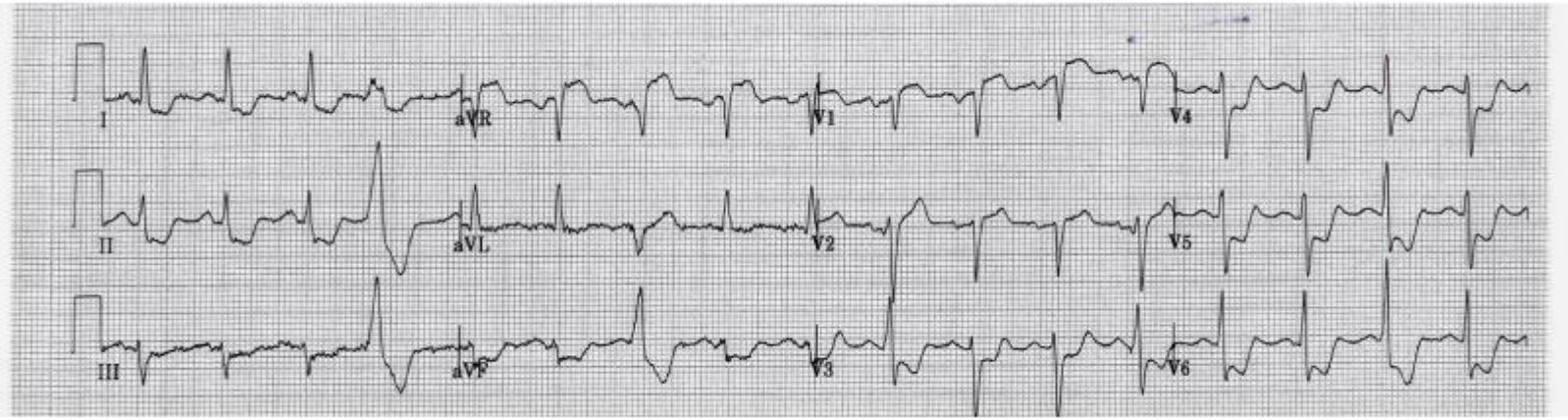


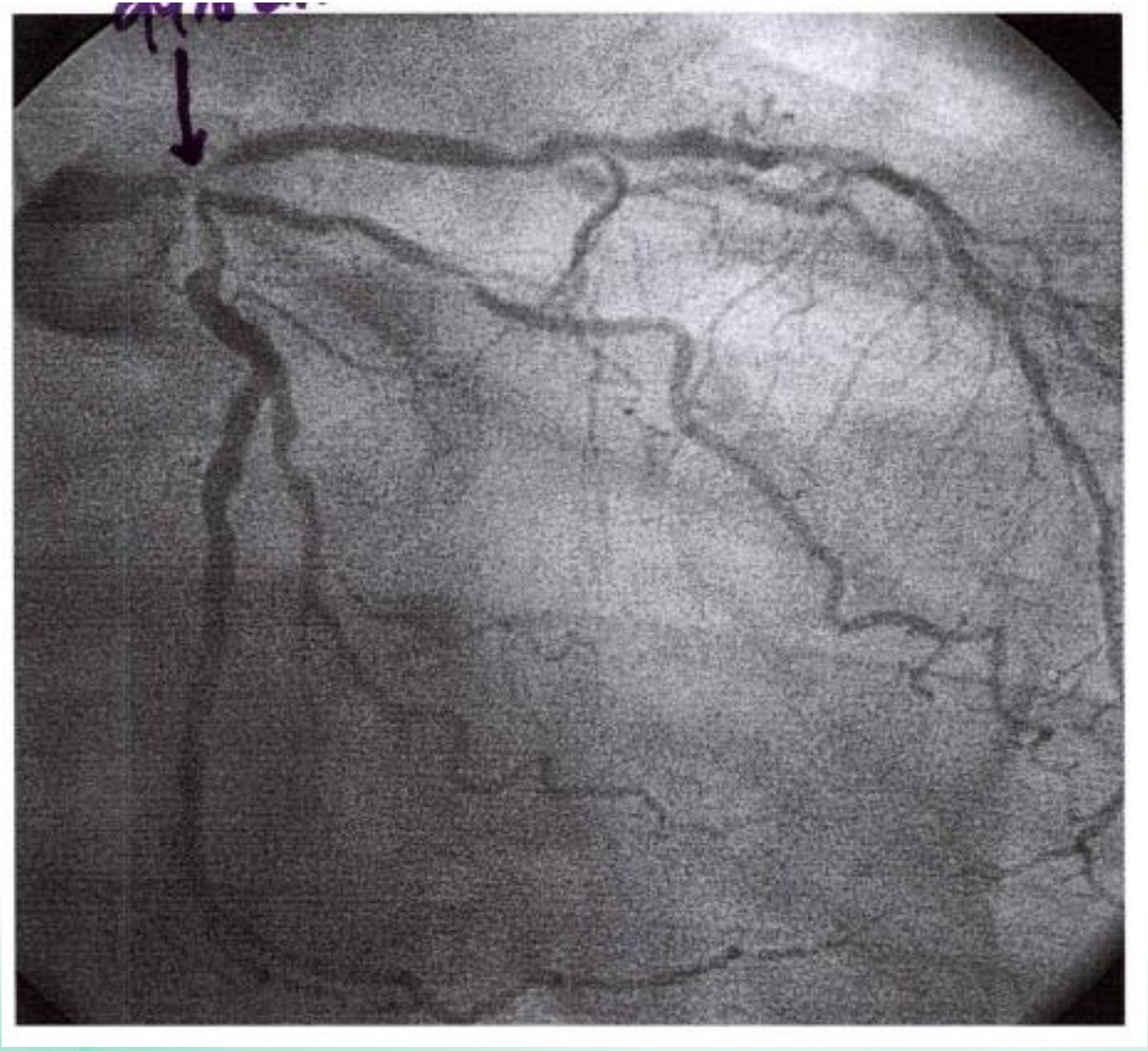

I
AUR
U1
U4
II
AUL
U2
U5
III
AUF
U3
U6
PSUC
PSUC
II

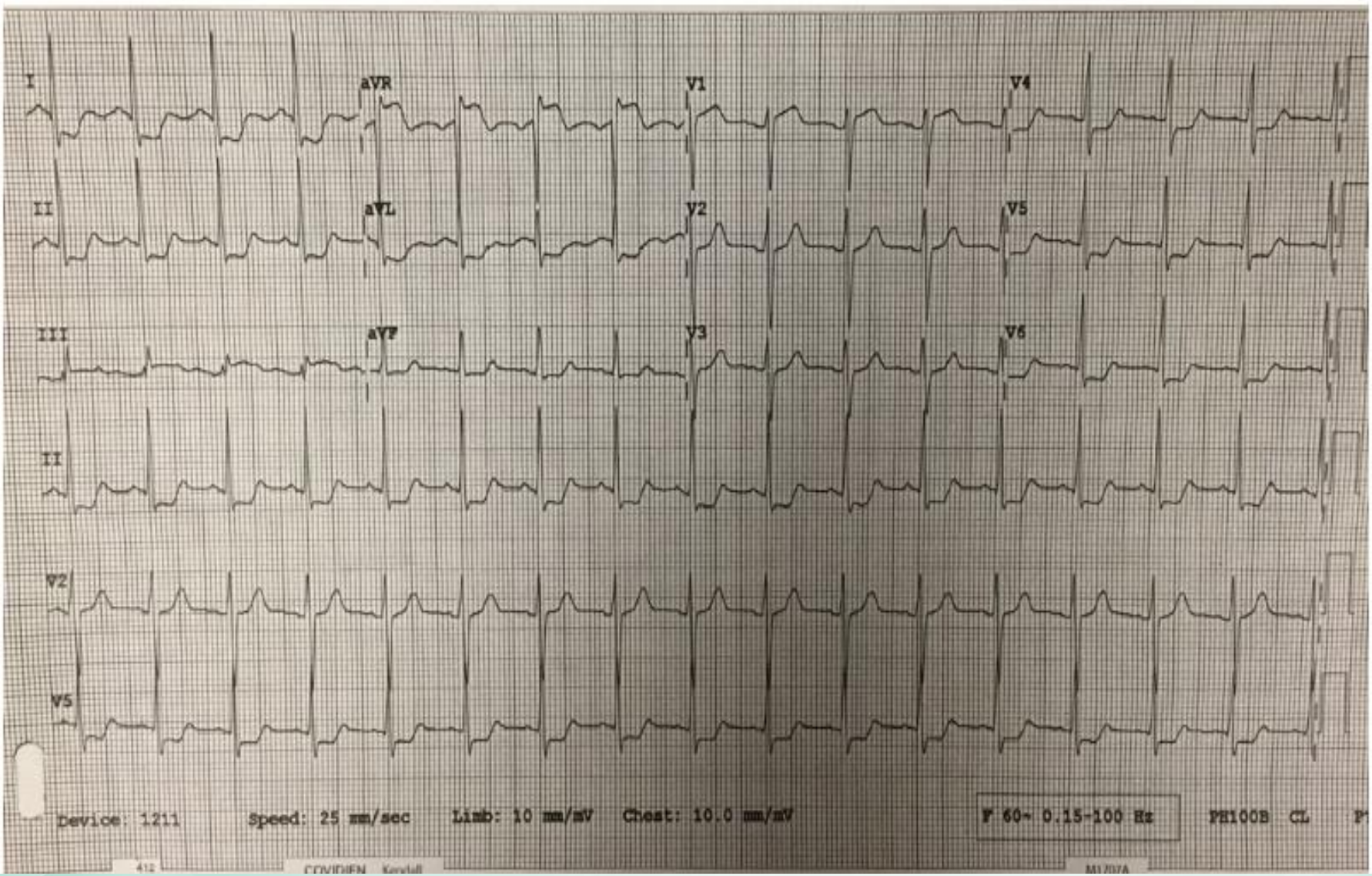


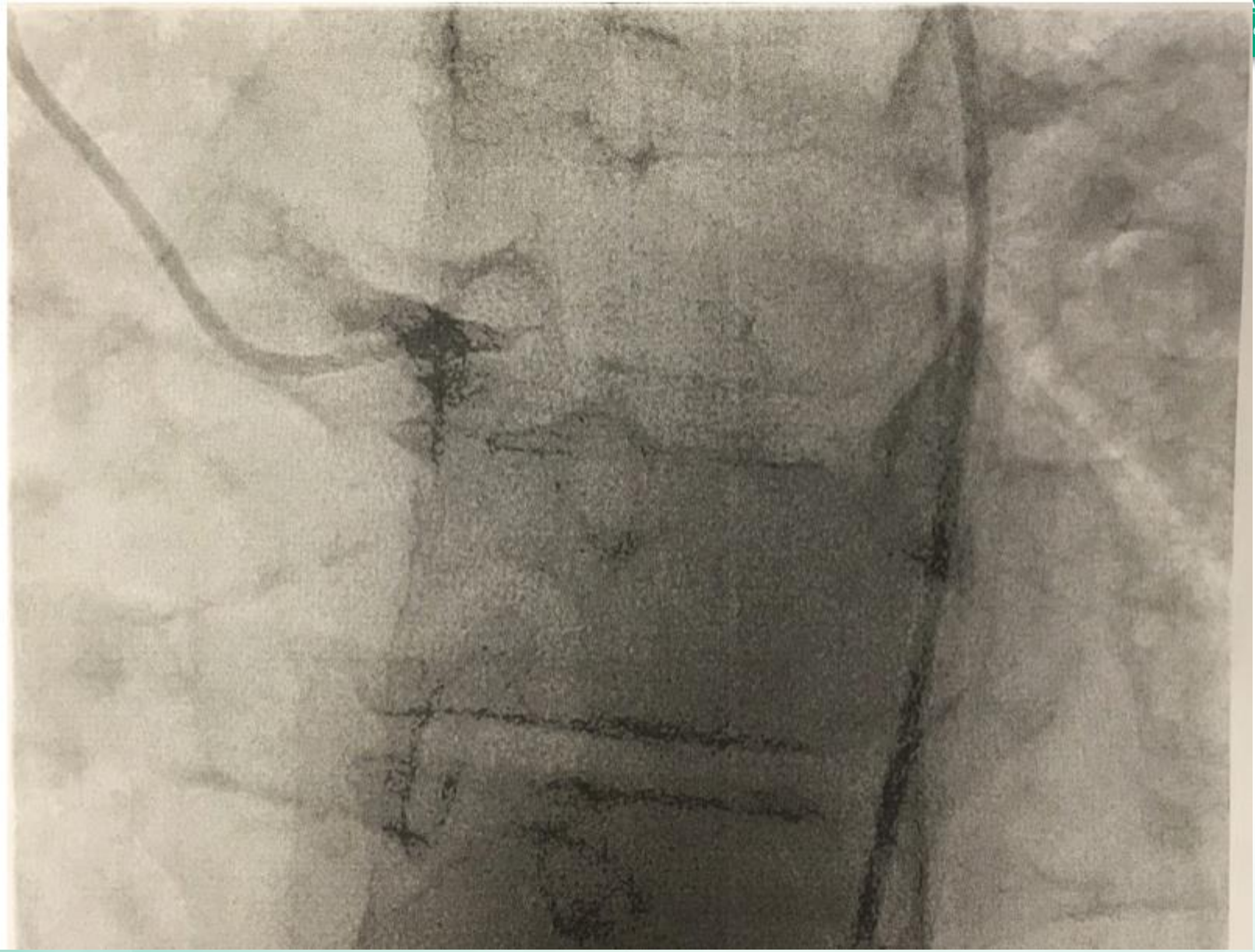
LMCA Occlusion

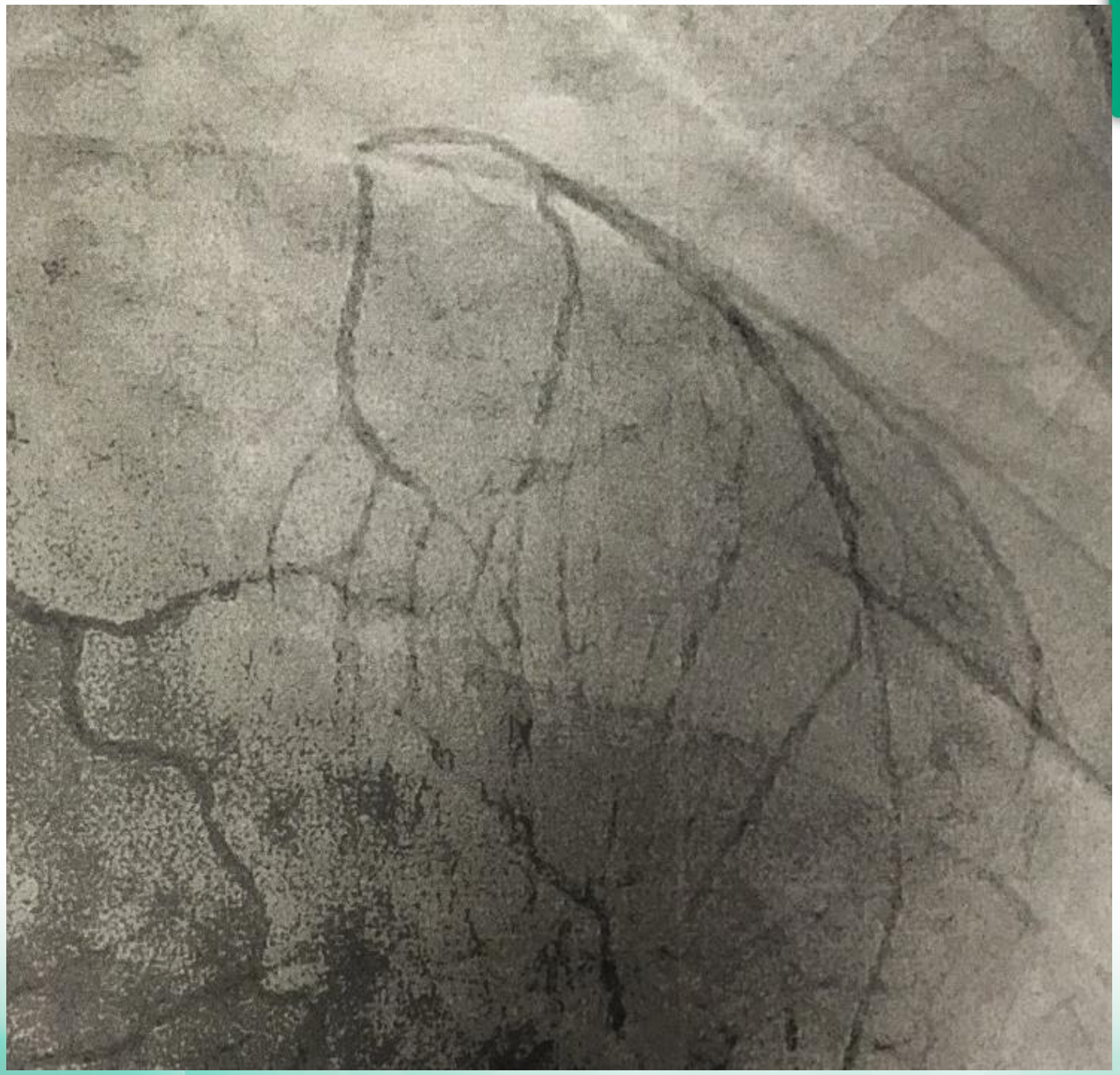
- Marked ST elevation in aVR >> V1
- ST depression in mulitple leads (V2-6, I, II, aVL, aVF), to some extent masked by a non-specific interventricular conduction delay


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




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
V2
V5
Device: 1211
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10.0 mm/mV
F 60~ 0.15-100 Hz
PH100B CL





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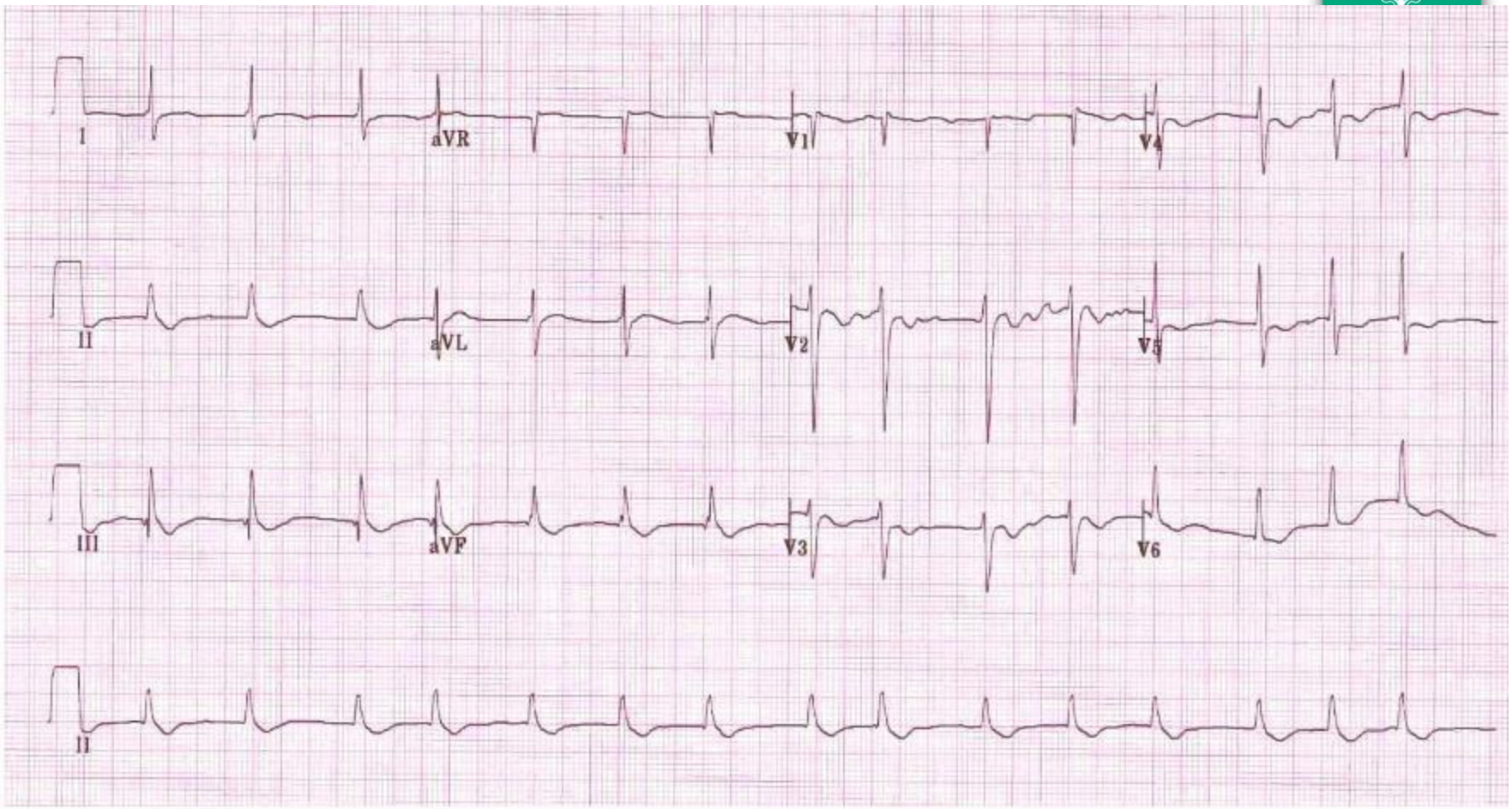
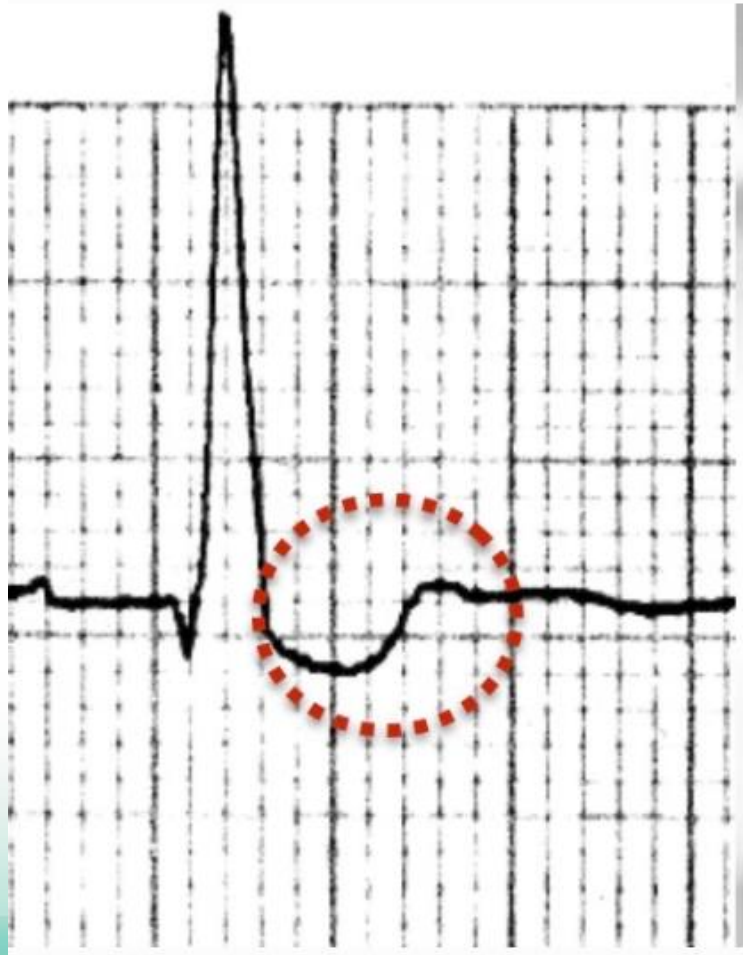
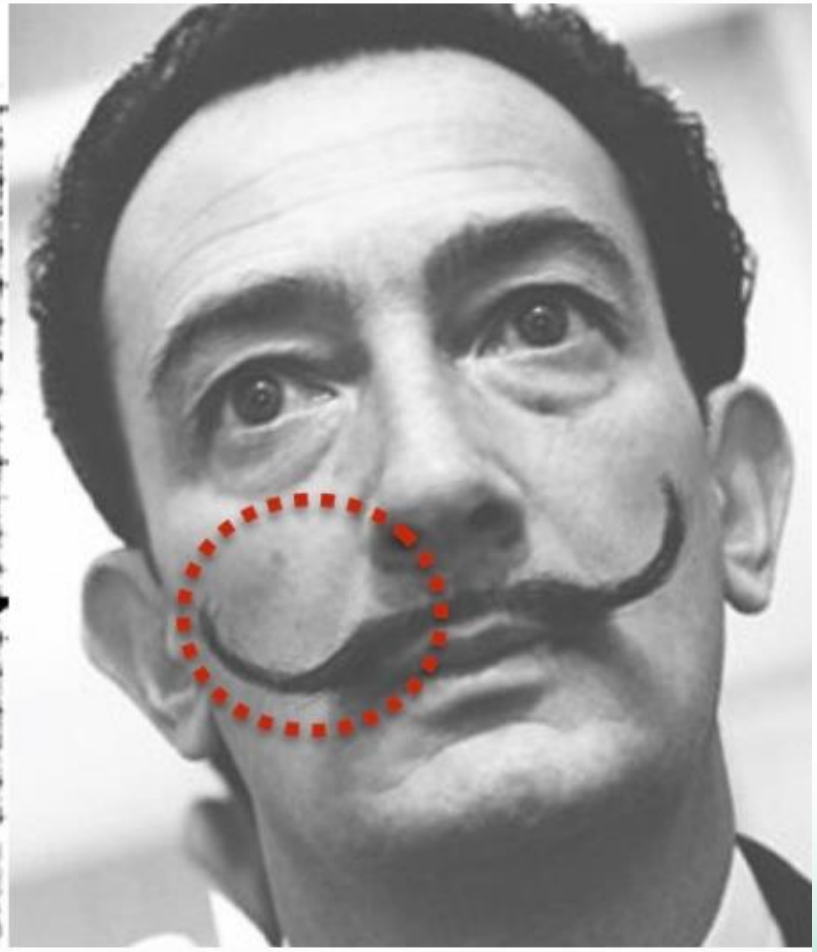


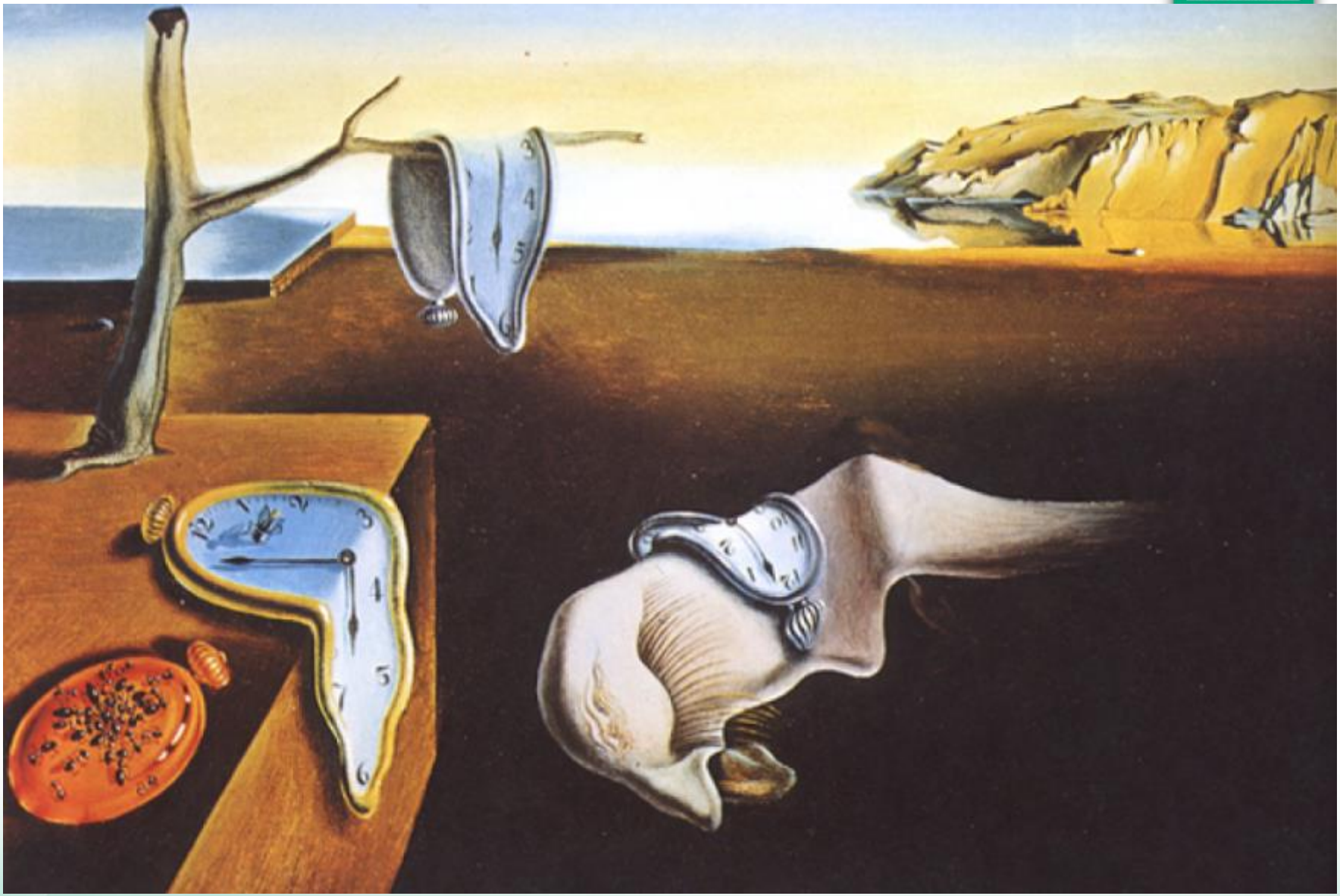
Digoxin effect:

- This is the classic picture of digoxin effect with the "sagging" ST segments and T waves taking on the appearance of "Salvador Dali's moustache".

- The morphology of the QRS complex / ST segment is variously described as either "slurred", "sagging" or "scooped" and resembling either a "reverse tick", "hockey stick" or (my personal favourite) "Salvador Dali's moustache"!







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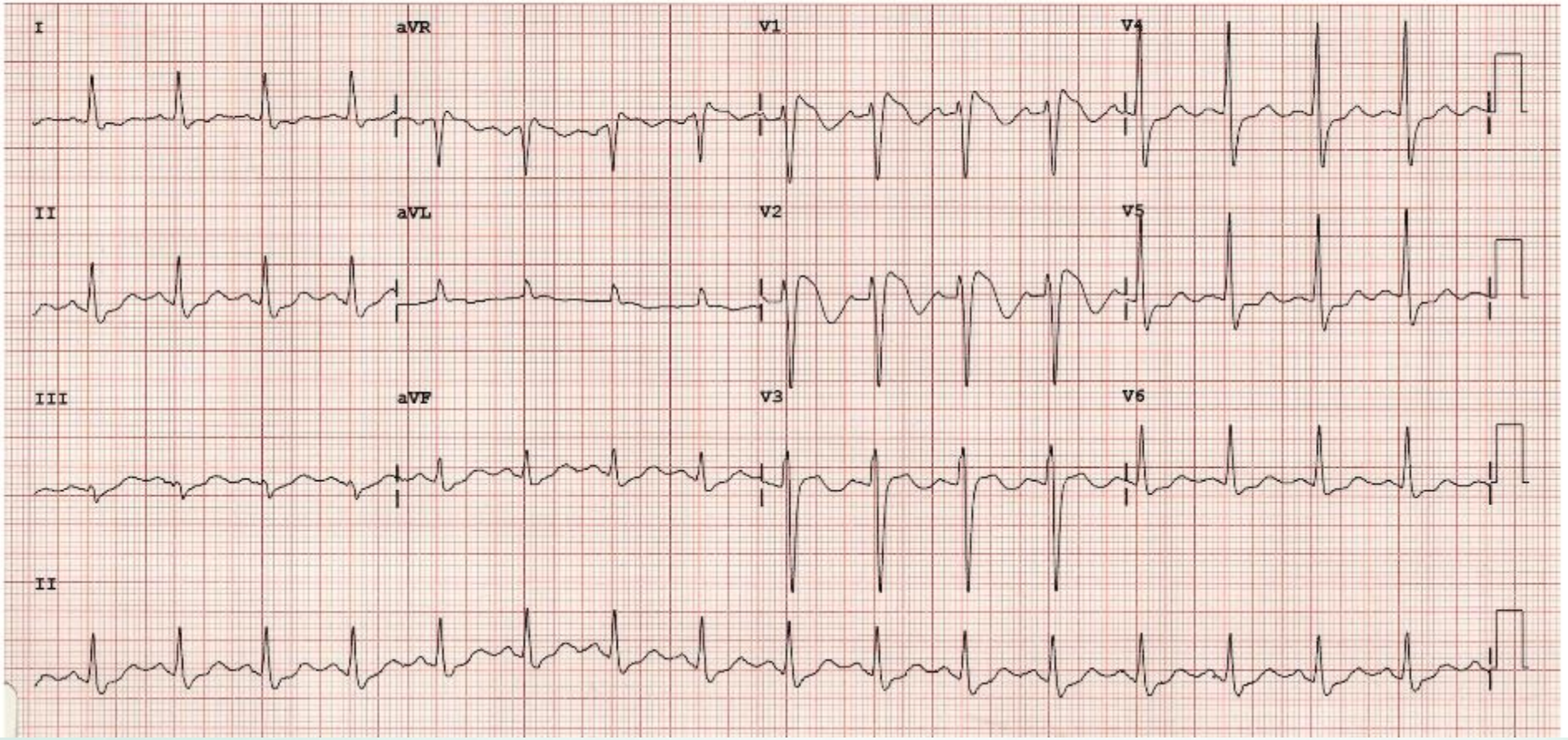
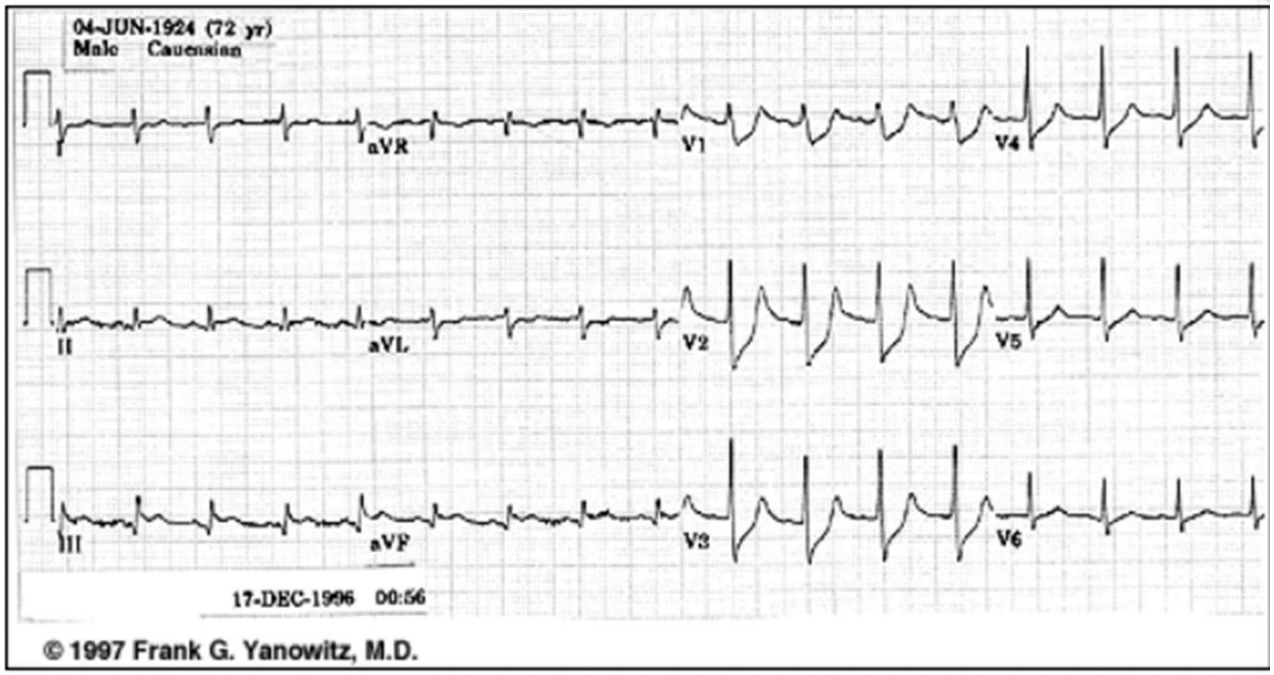
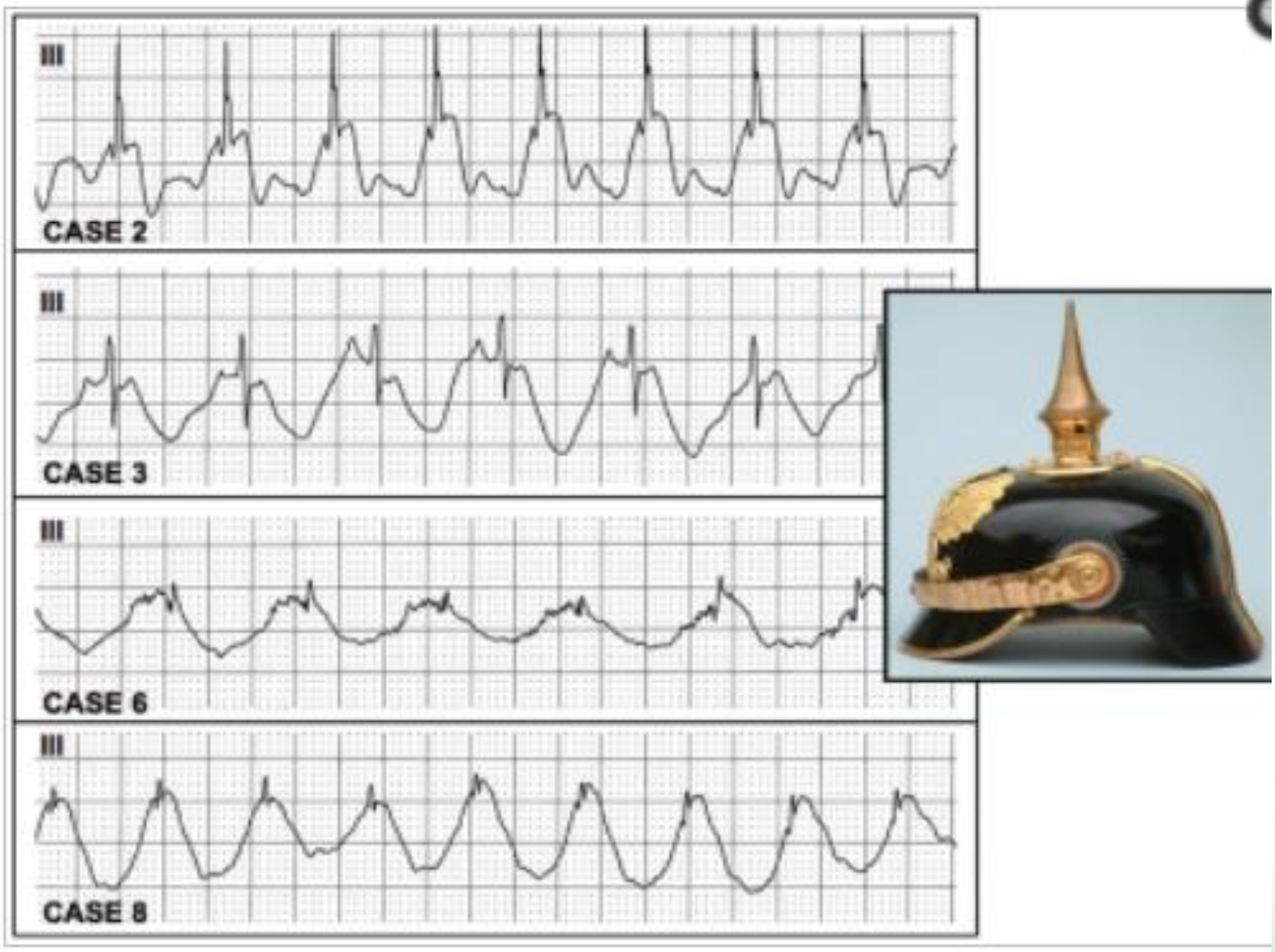
A 21 year old male of Asian descent has presented to your ED following a brief episode of syncope. He feels fine now and wants to go home. His ECG looks like this:



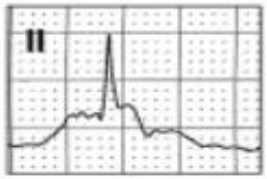
- LV aneurysm

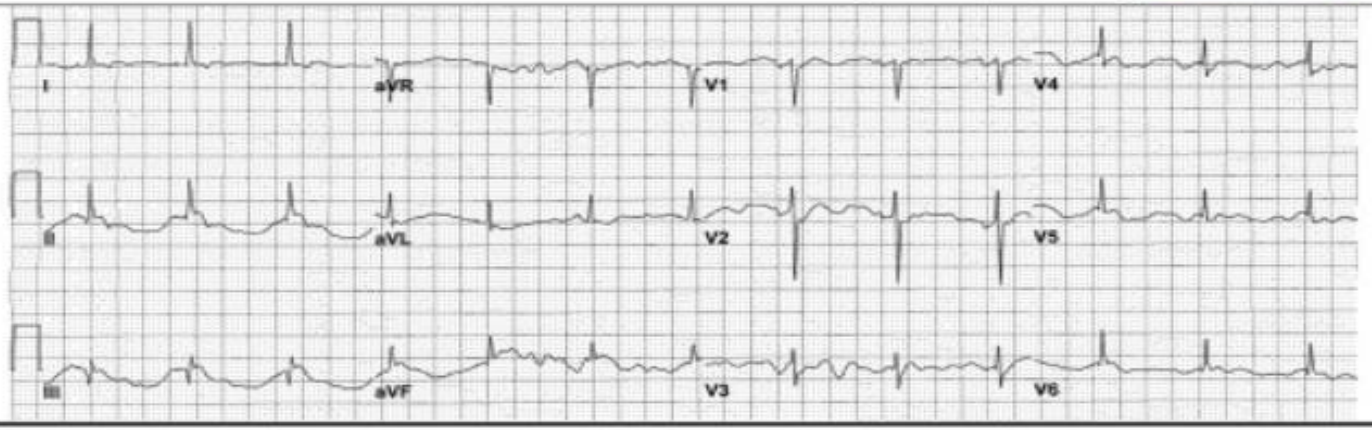

04-JUN-1924 (72 yr)
Male Caucasian
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
17-DEC-1996 00:56
© 1997 Frank G. Yanowitz, M.D.


III
CASE 2
III
CASE 3
III
CASE 6
III
CASE 8

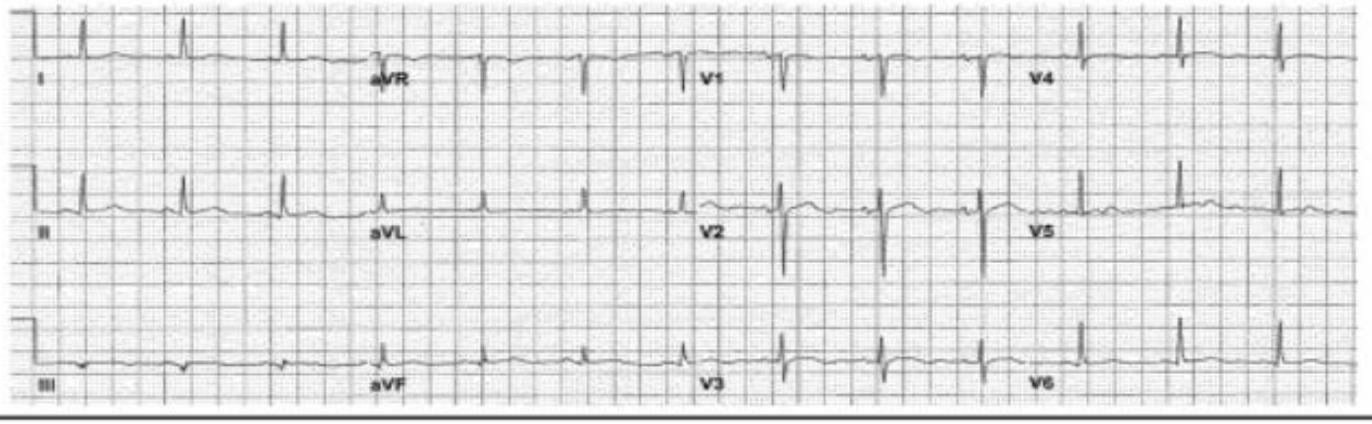
A

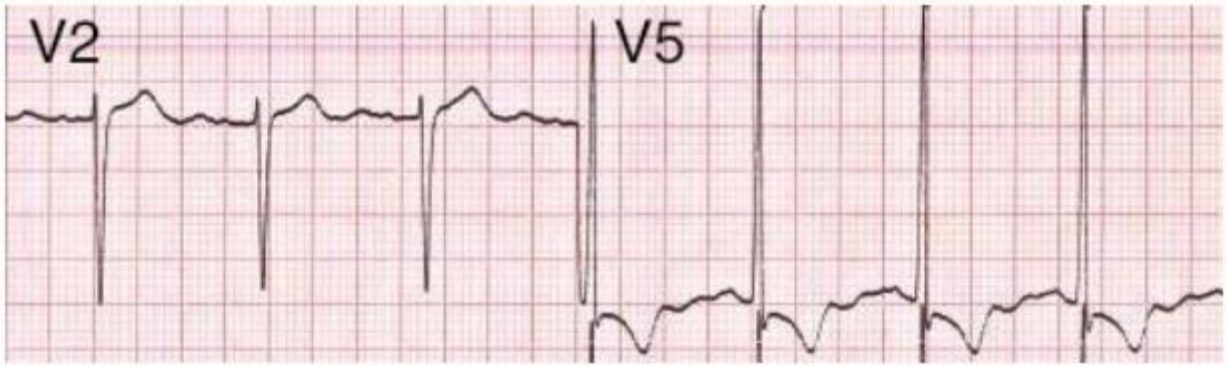
Accelerated Junctional rhythm
ST elevation consider inferior injury or acute infarct
Prolonged QT
*** ** ** ** * ACUTE MI ** ** ** **



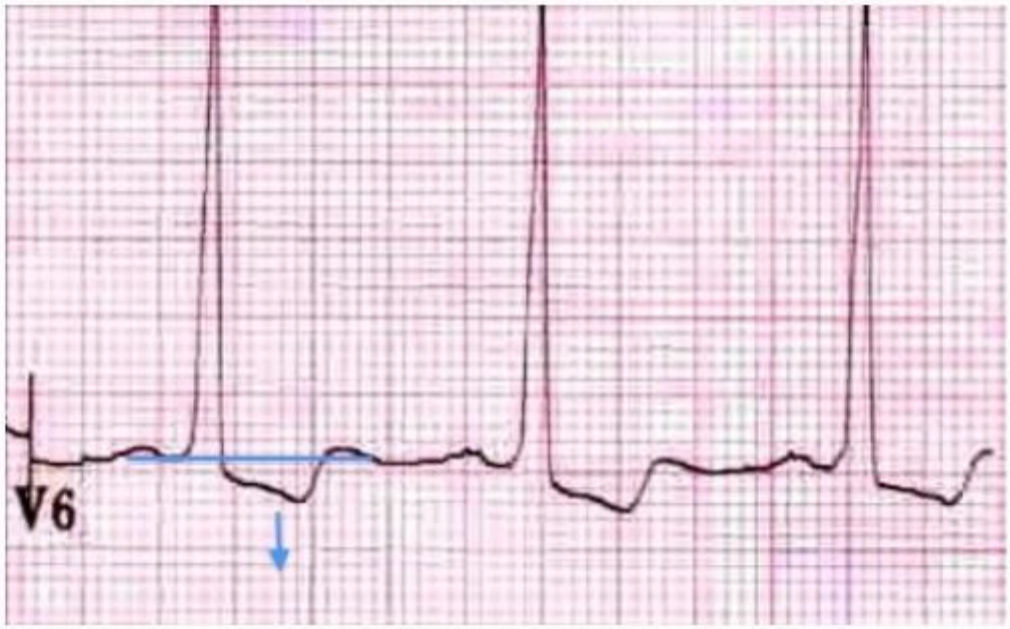


B

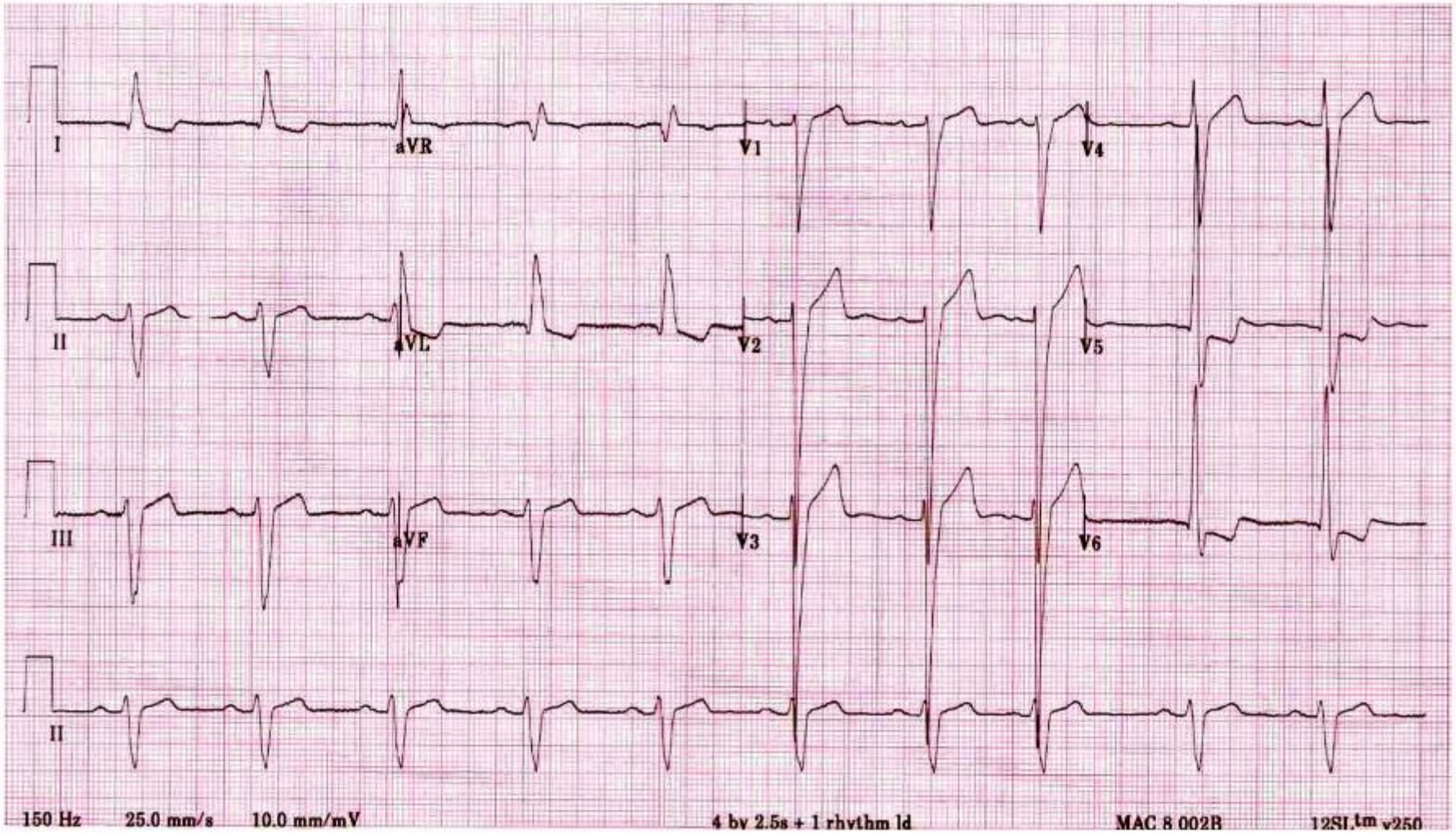


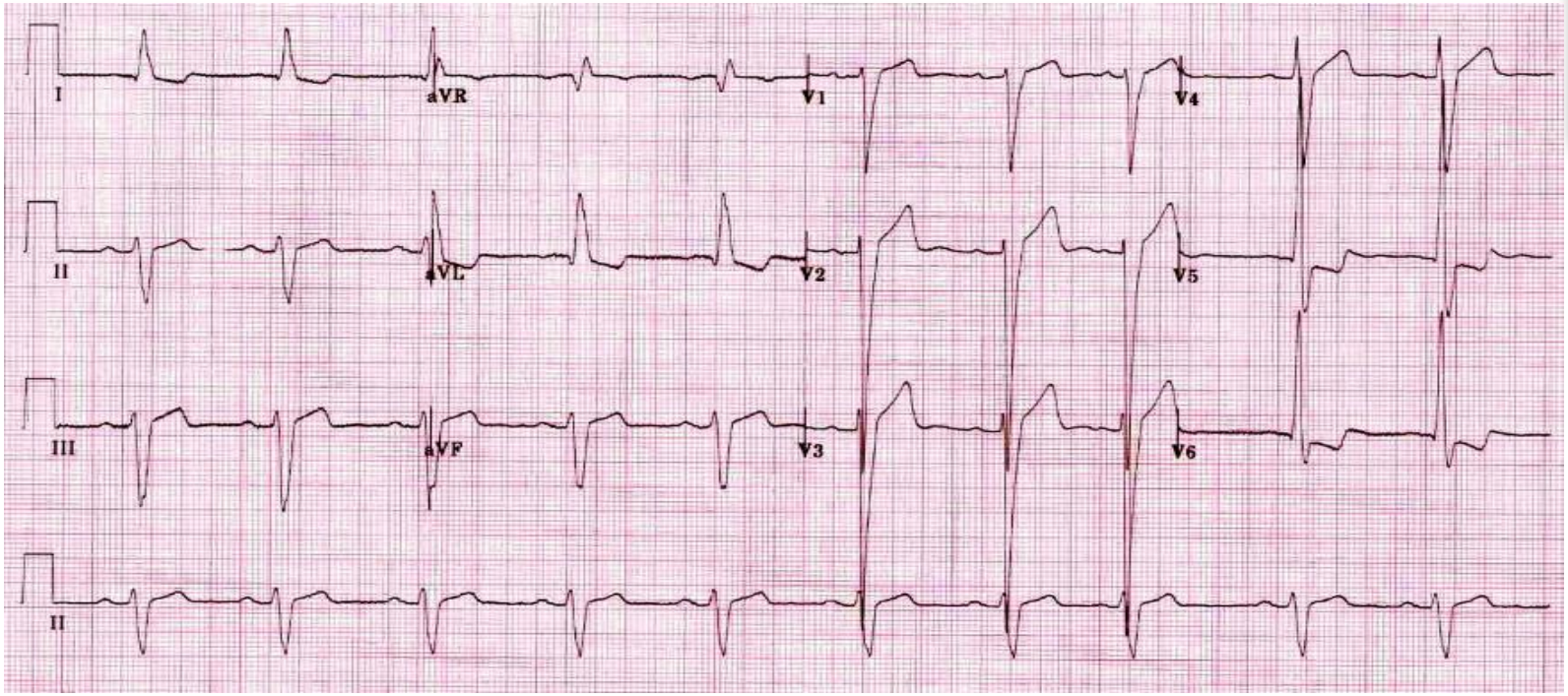


LVH by voltage criteria: S wave in V2 + R wave in V5 > 35 mm



LV strain pattern: ST depression and T wave inversion in the lateral leads


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
150 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm ld
MAC 8 002B
12SL tm v250



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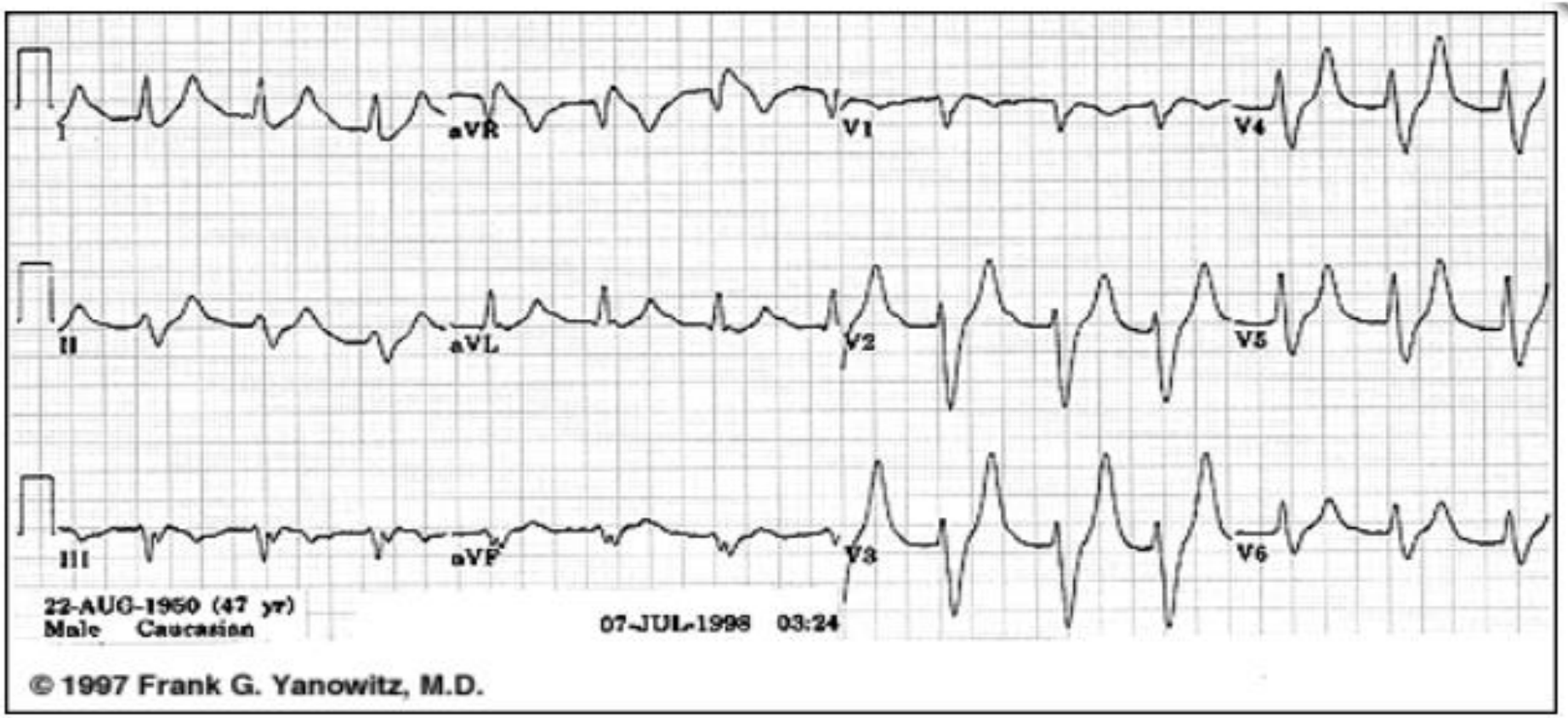
Good example of LVH:

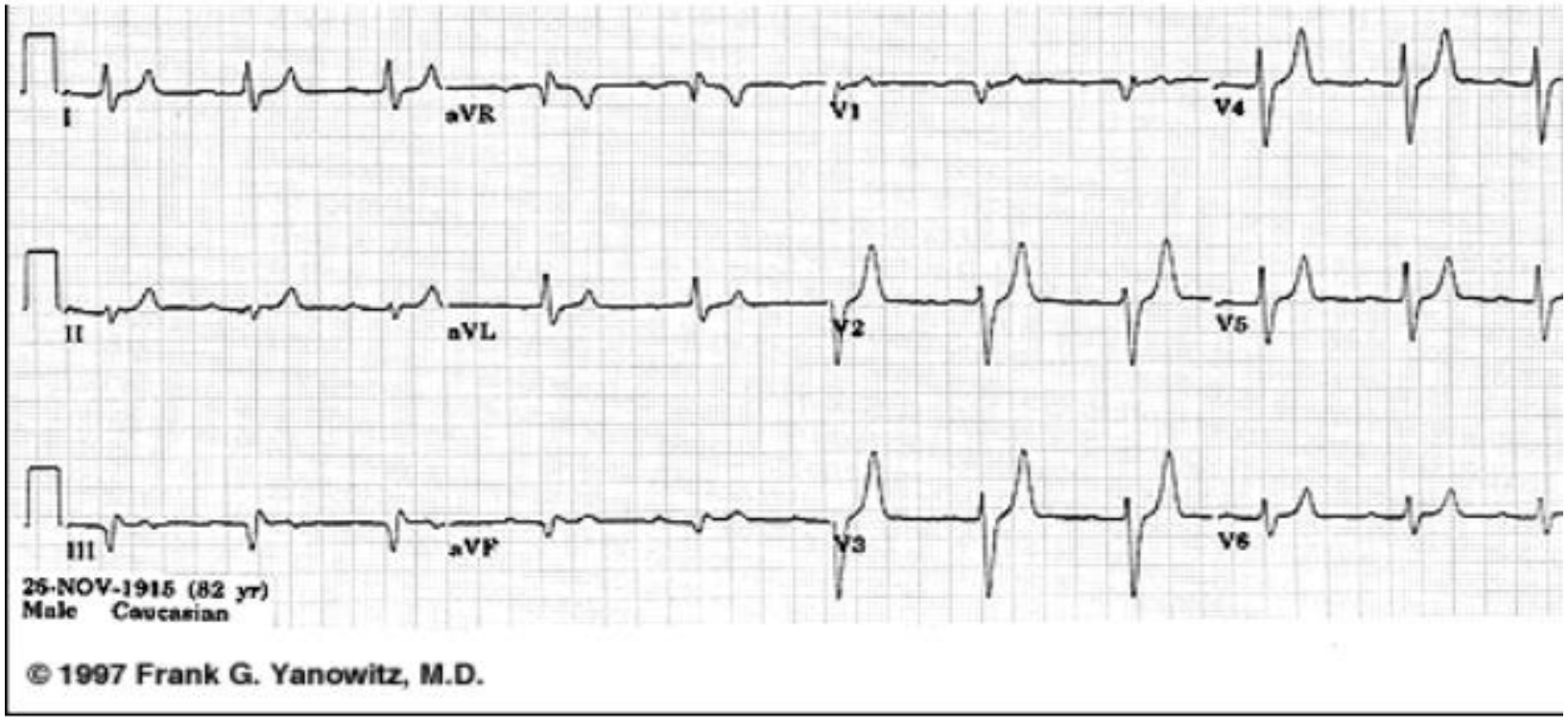
- Markedly increased LV voltages: huge precordial R and S waves that overlap with the adjacent leads (SV2 + RV6 >> 35 mm).
- R-wave peak time > 50 ms in V5-6 with associated QRS broadening.
- LV strain pattern with ST depression and T-wave inversions in I, aVL and V5-6.
- ST elevation in V1-3.
- Prominent U waves in V1-3.
- Left axis deviation.



Advanced Hyperkalemia

Frank G. Yanowitz, M.D. ©1998

Marked widenening of the QRS duration combined with tall, peaked T waves are suggestive of advanced hyperkalemia. Note the absence of P waves, suggesting a junctional rhythm, but in hyperkalemia the atrial muscle may be paralyzed while still in sinus rhythm. The sinus impulse conducts to the AV node through internodal tracts without activating the atrial muscle.



Hyperkalemia and Old Inferior MI

Frank G. Yanowitz, M.D. ©1998

The T waves are tall, peaked and have a narrow base, making them very uncomfortable to sit on! These changes are characteristic of hyperkalemia. The QRS is also slightly widened, another feature of hyperkalemia. Q waves in III and aVF indicate an old inferior MI.

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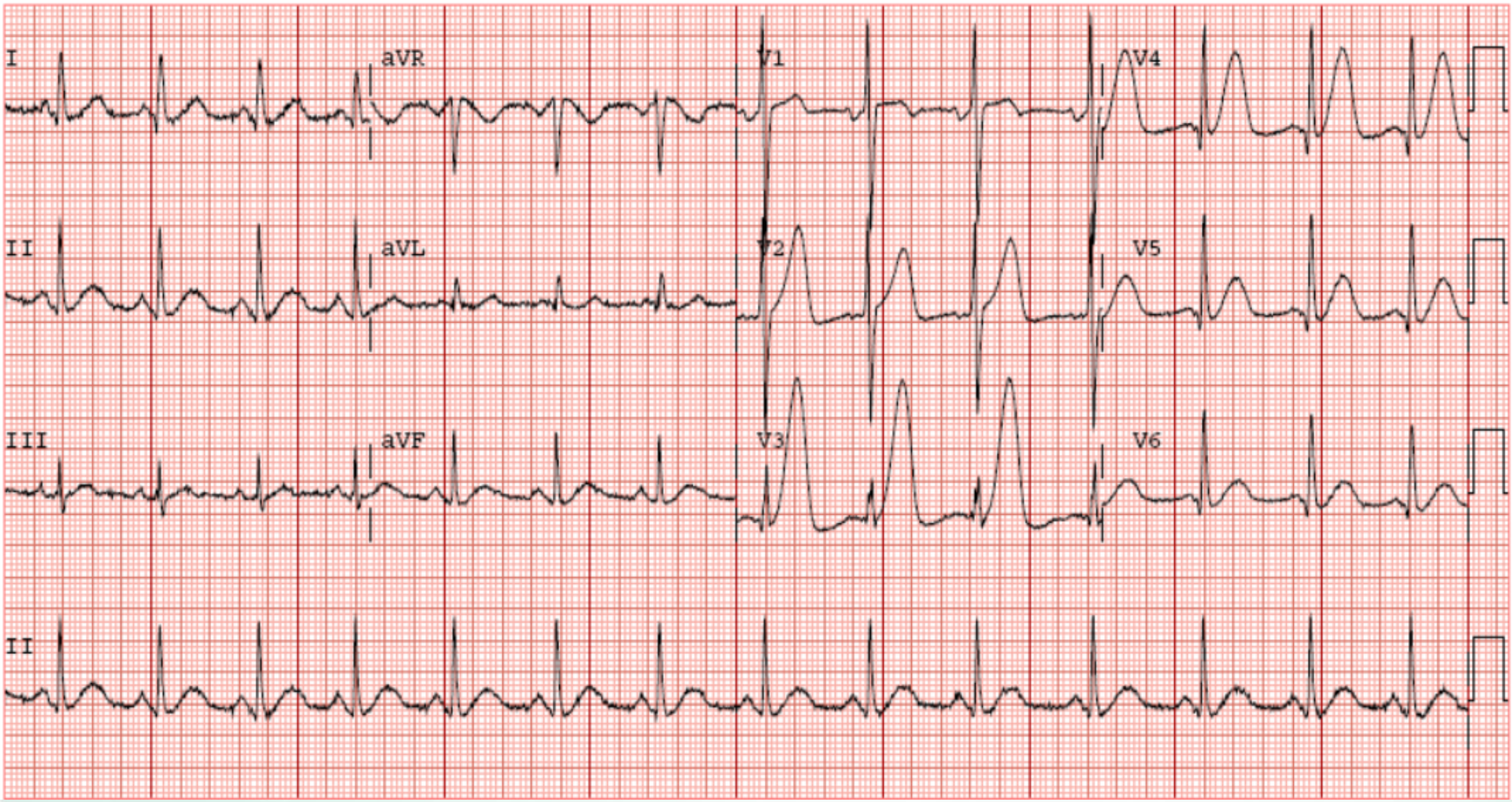


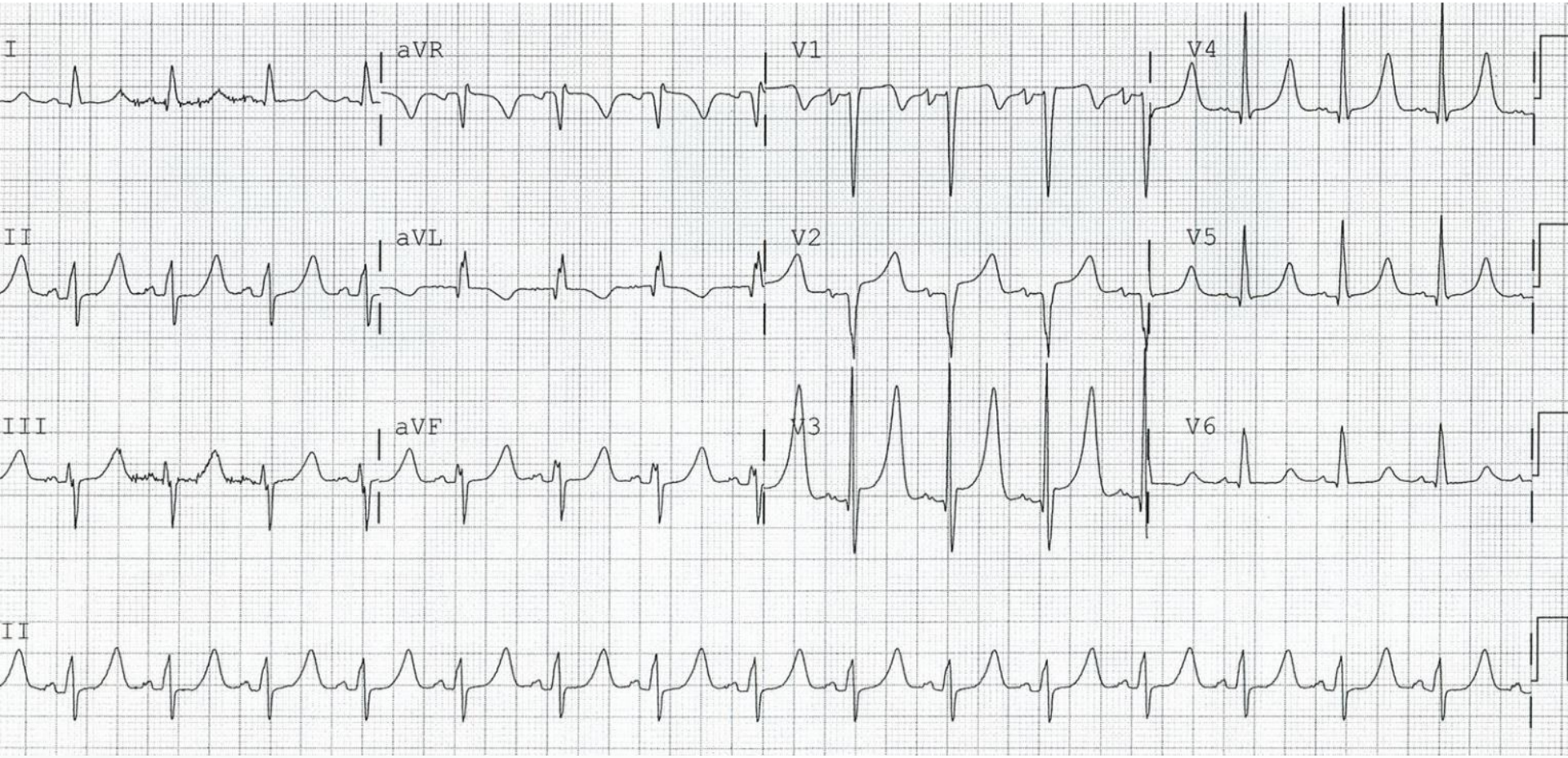
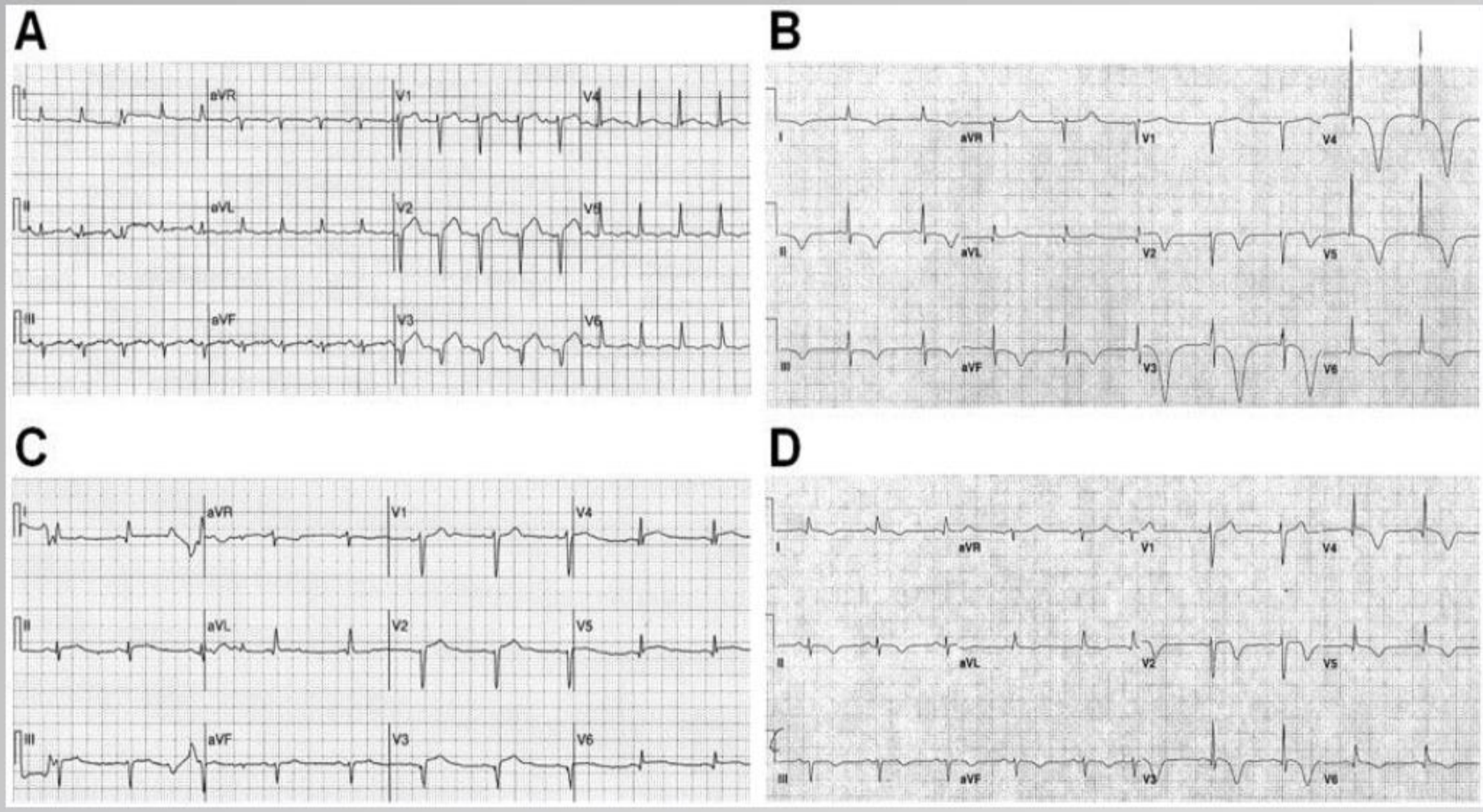
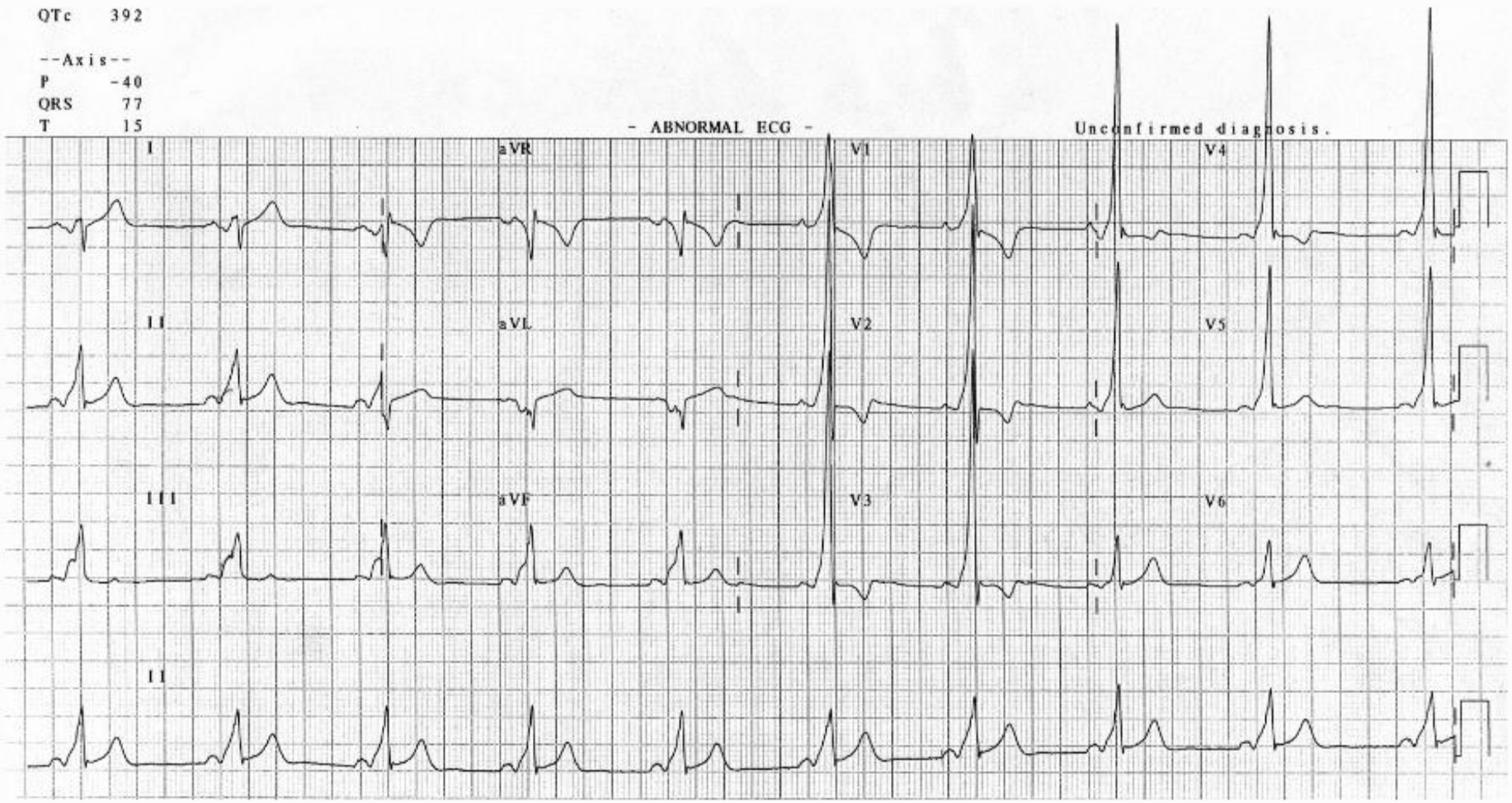
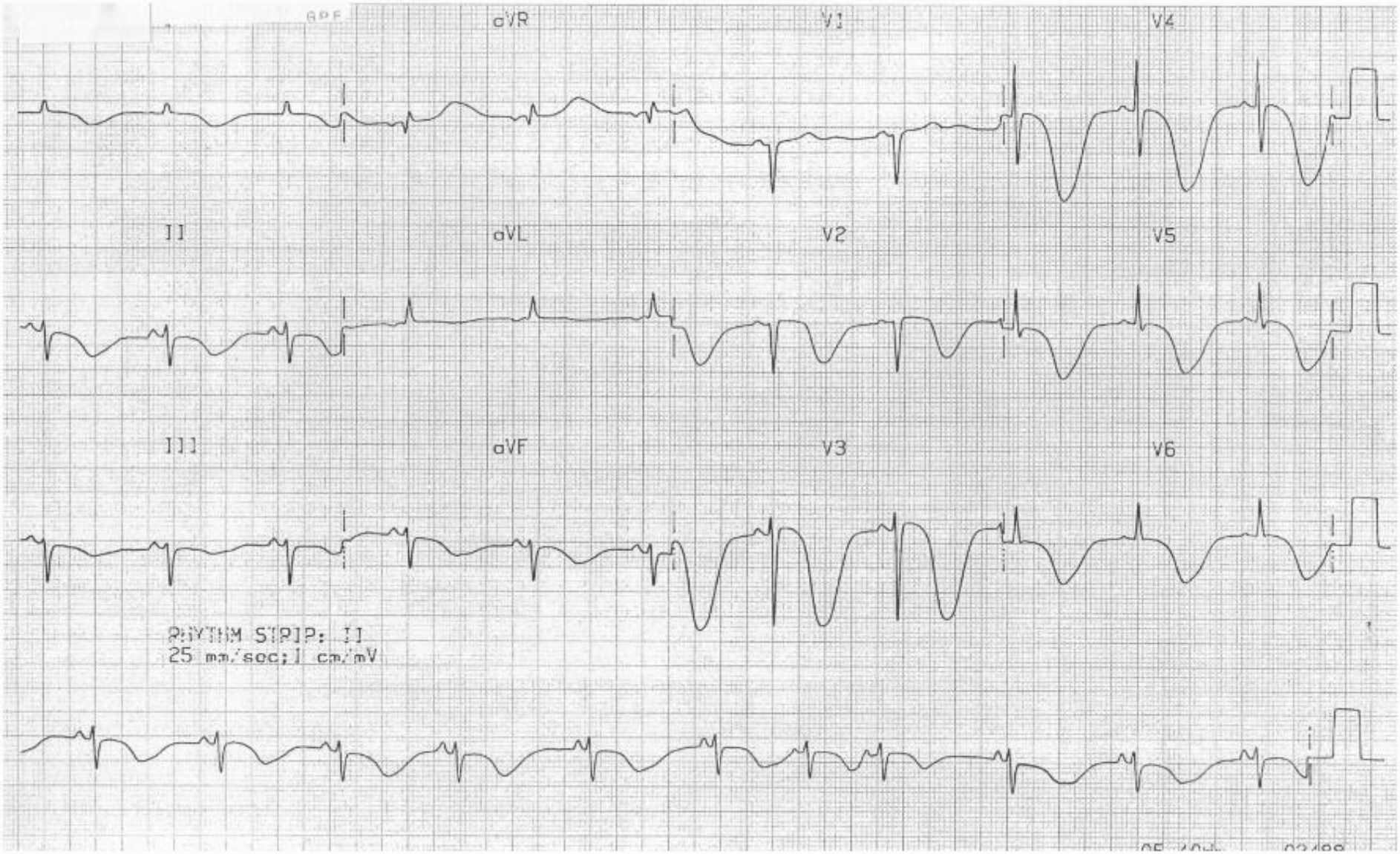

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Figure 1. Spectrum of ECG patterns observed in 3 patients with psychologically triggered, reversible LV dysfunction that mimics myocardial infarction or acute coronary syndrome.



Sharkey S W et al. Circulation. 2005;111:472-479


QTc 392
--Axis--
P -40
QRS 77
T 15
- ABNORMAL ECG -
Unconfirmed diagnosis.
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV