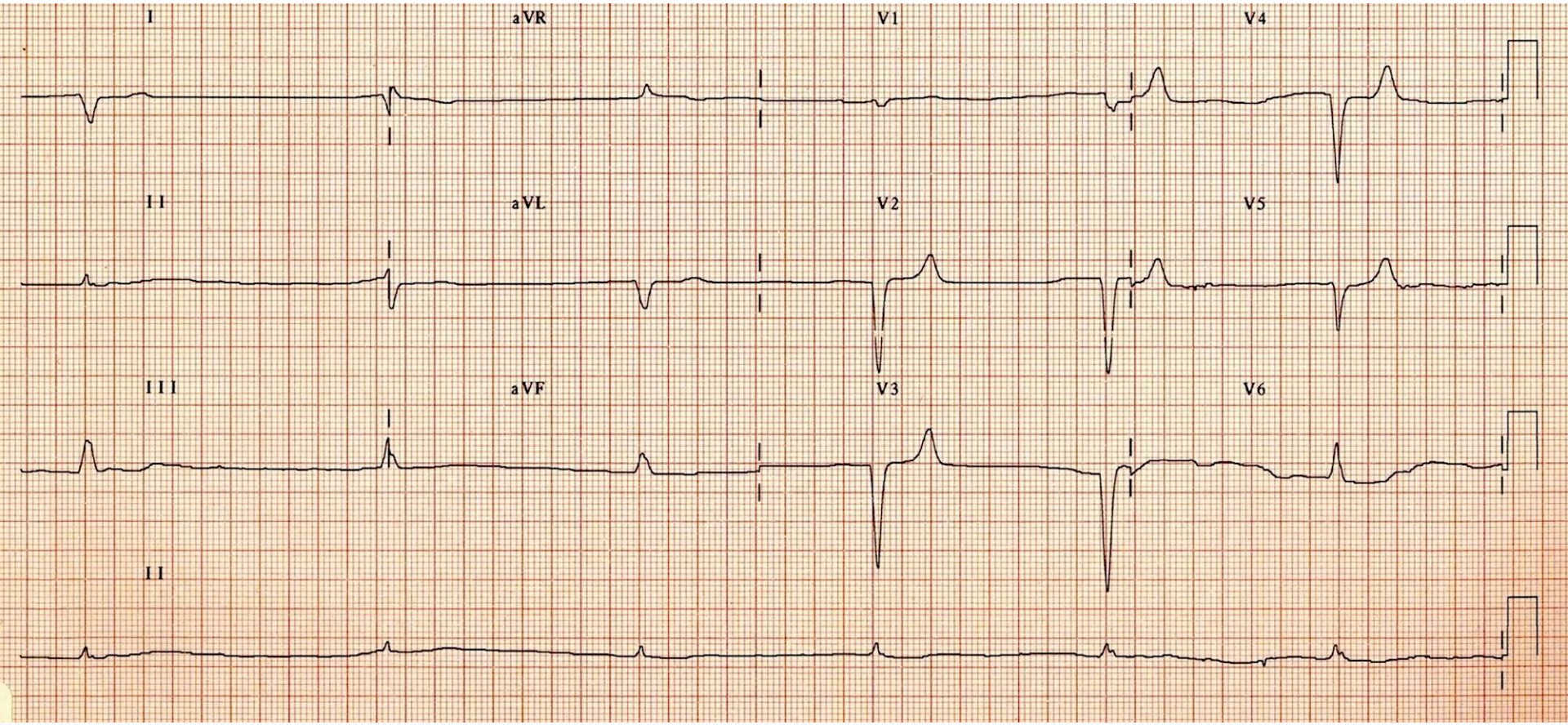
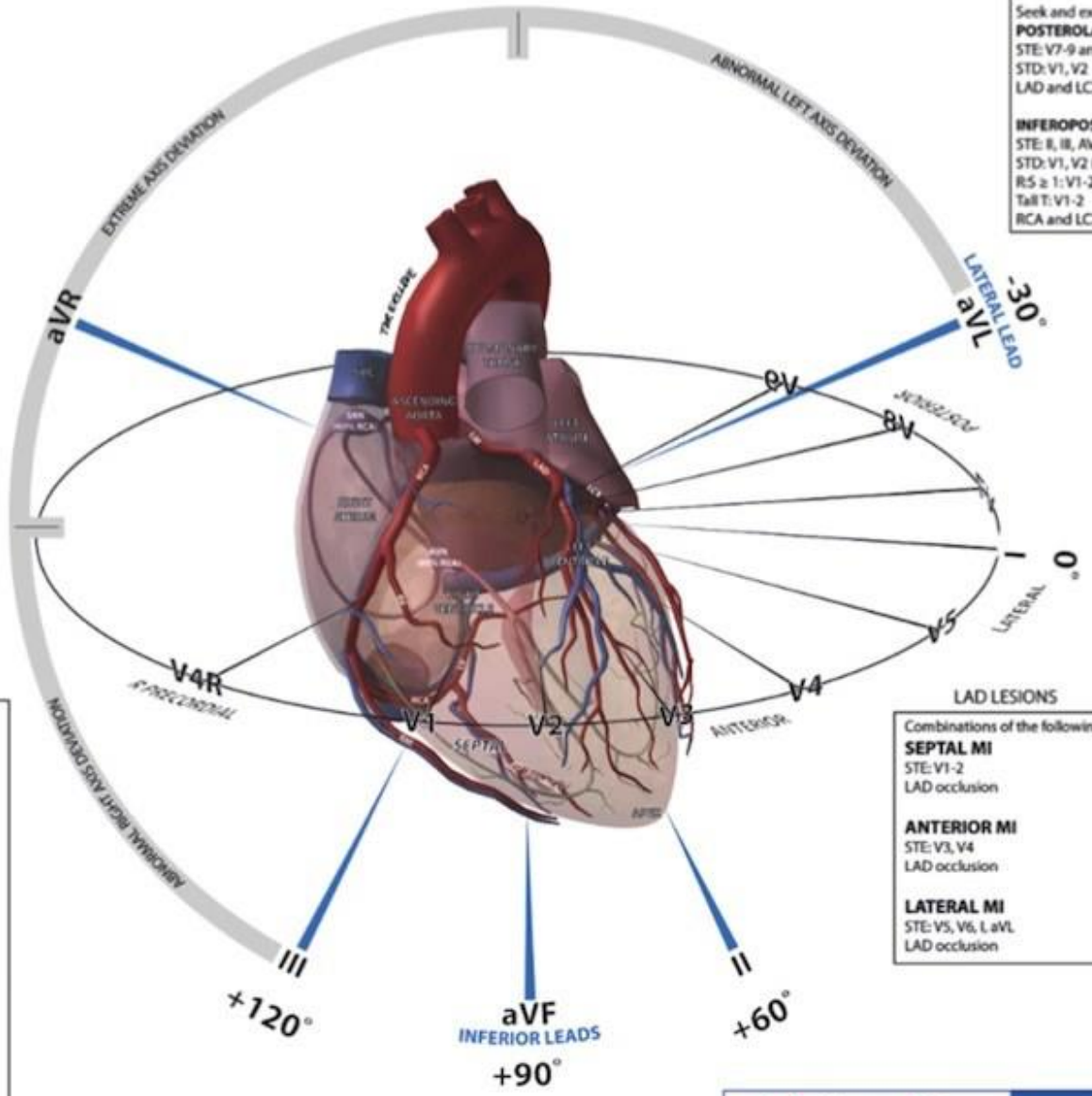
Killer ECG Patterns

20th Oct 2016



60-year old with shortness of breath

AMI ECG, ANATOMY AND PATHOLOGY



LCX LESIONS ±

POSTERIOR MI
STE: V7-9
STD: V1-2 (reciprocal STE)
R:S ≥ 1: V1-2
Tall T: V1-2
RCA and LCX occlusion

Seek and exclude
POSTEROLATERAL MI
STE: V7-9 and I, aVL, V5-6
STD: V1, V2
LAD and LCX occlusion

INFEROPOSTERIOR MI
STE: II, III, AVF and V7-9
STD: V1, V2 (reciprocal STE)
R:S ≥ 1: V1-2
Tall T: V1-2
RCA and LCX occlusion

RCA 'TYPE' LESIONS ±

INFERIOR MI
STE: II, III, aVF
STD: aVL (reciprocal STE)
RCA occlusion distal to RV
58% of MI

Seek and exclude
INFERIOR AND RV MI
STE: II, III, aVF and V1, V4R
RCA occlusion proximal to RV
40% of Inferior MI
Increased mortality risk

INFEROLATERAL MI
STE: II, III, AVF and I, aVL, V5, V6 ± V4R
LAD and LCX occlusion
in a L dominant system

INFEROPOSTERIOR MI
STE: II, III, AVF and V7-9
STD: V1, V2 (reciprocal STE)
R:S ≥ 1: V1-2
Tall T: V1-2
RCA and LCX occlusion

LAD LESIONS

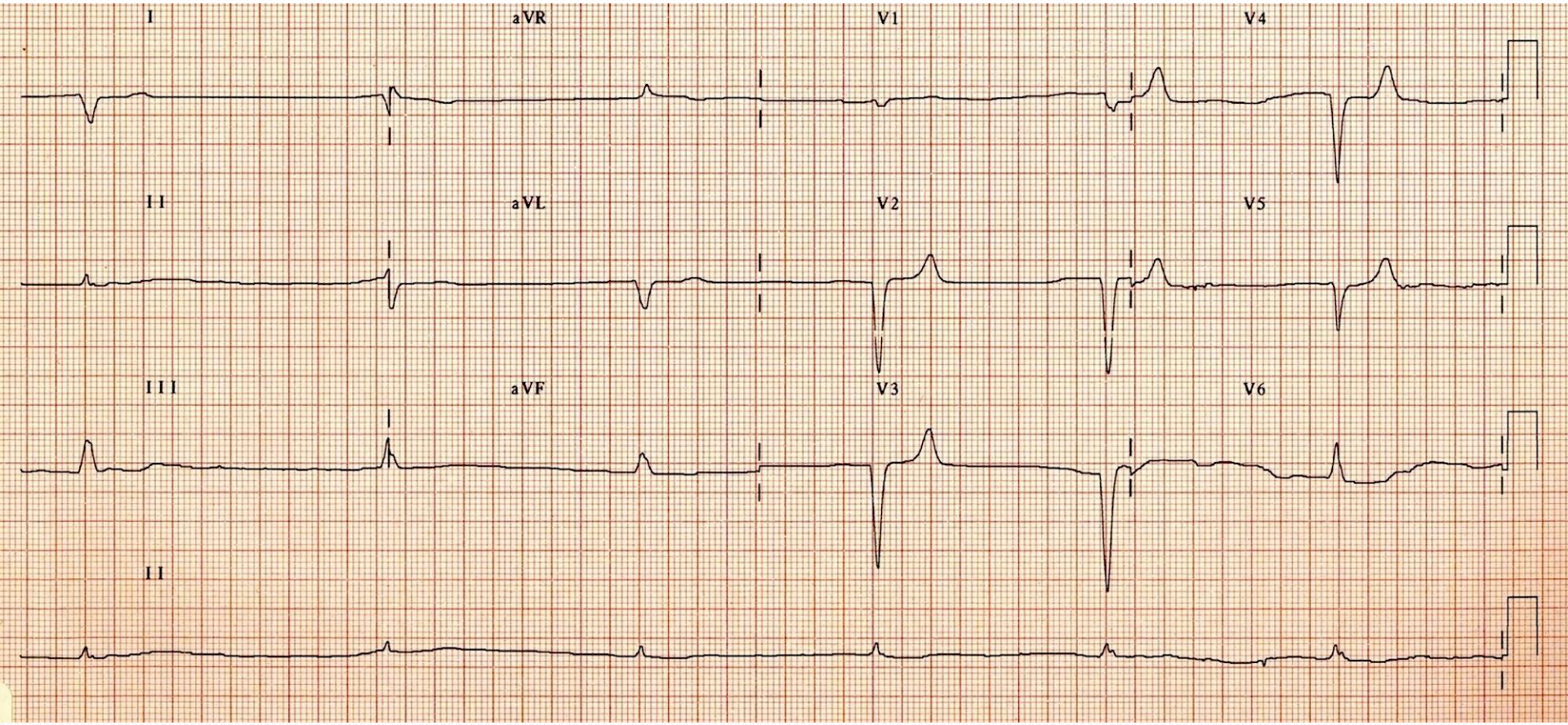
Combinations of the following

SEPTAL MI
STE: V1-2
LAD occlusion

ANTERIOR MI
STE: V3, V4
LAD occlusion

LATERAL MI
STE: V5, V6, I, aVL
LAD occlusion

- Rate
- Rhythm
- Axis
- Intervals
- Chamber enlargements
- Ischaemia



60-year old with shortness of breath

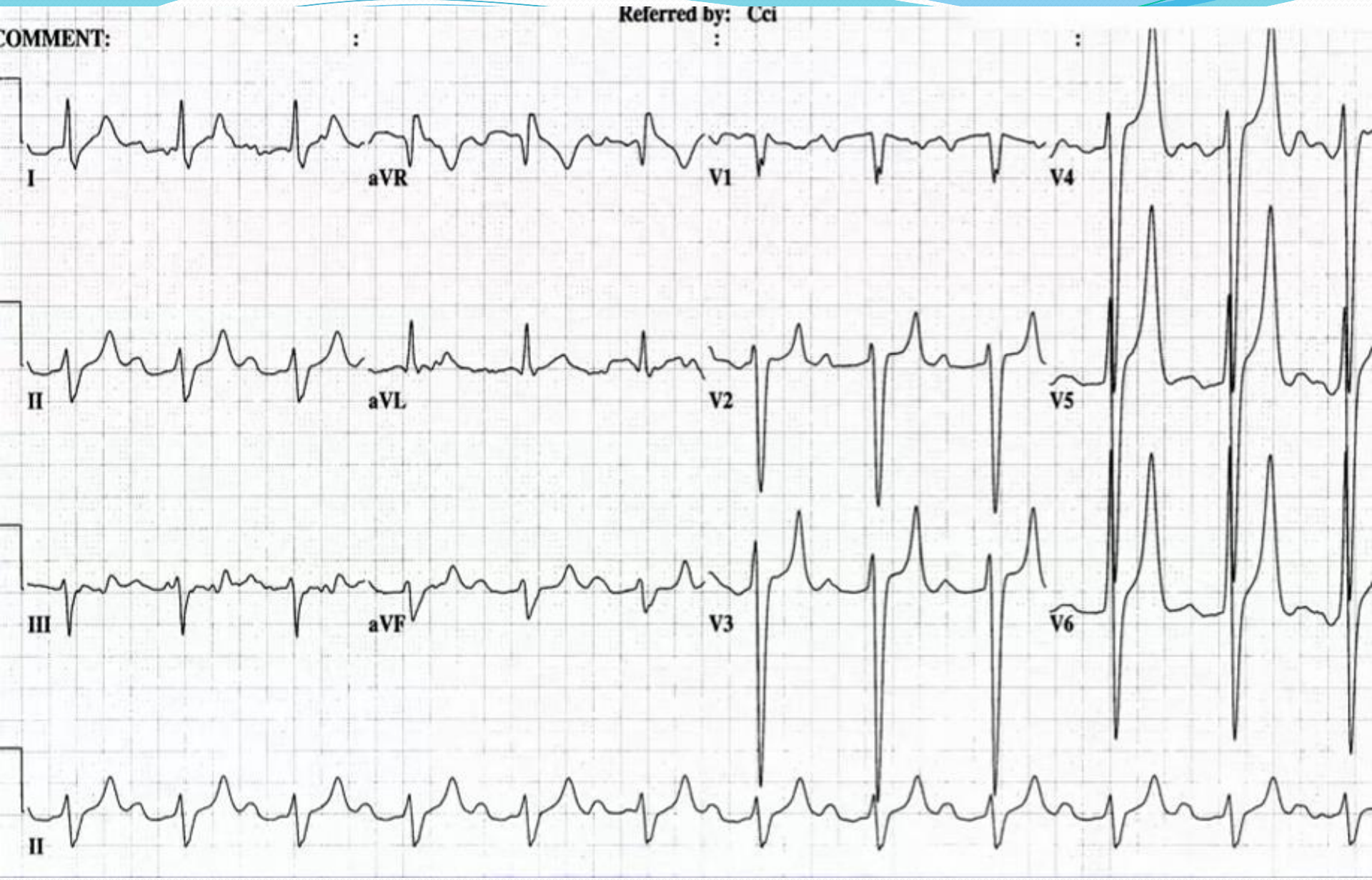
- Presented with APO after missing several dialysis sessions
- Cardiac arrest shortly after presenting
- K^+ 7.9

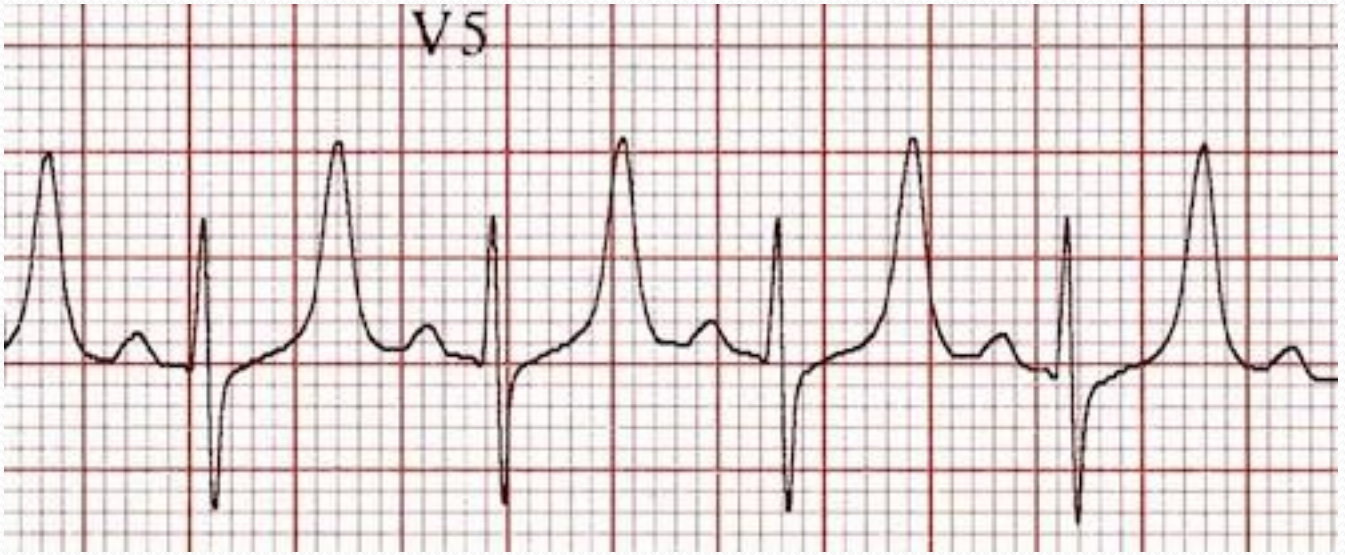
- Differential for severe bradycardia with I^o AV Block?
  - Ischaemia
  - B-blocker toxicity
  - Ca Channel blocker toxicity

Severe Hyperkalaemia

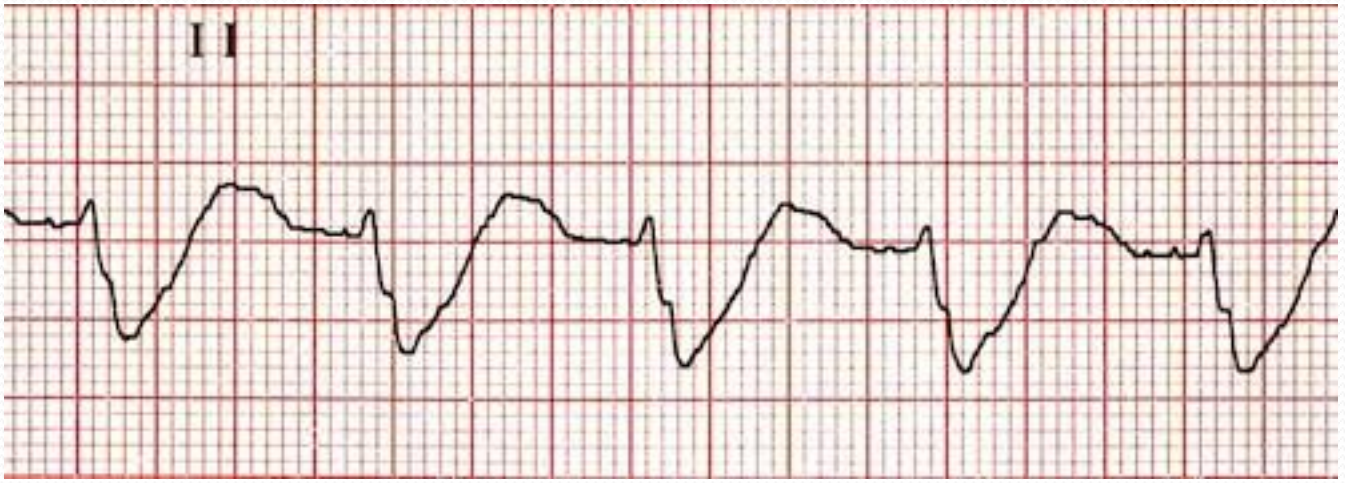
ECG Findings in Hyperkalemia

- - Peaked Ts
- - Widening of the QRS
- - Prolonged PR
- - Flattening and eventual loss of Ps
- - Tachycardias
- - Advanced AV blocks and sinus pauses
- - Pseudo-ACS - new BBBs, ST changes
- - Sine wave (preterminal)

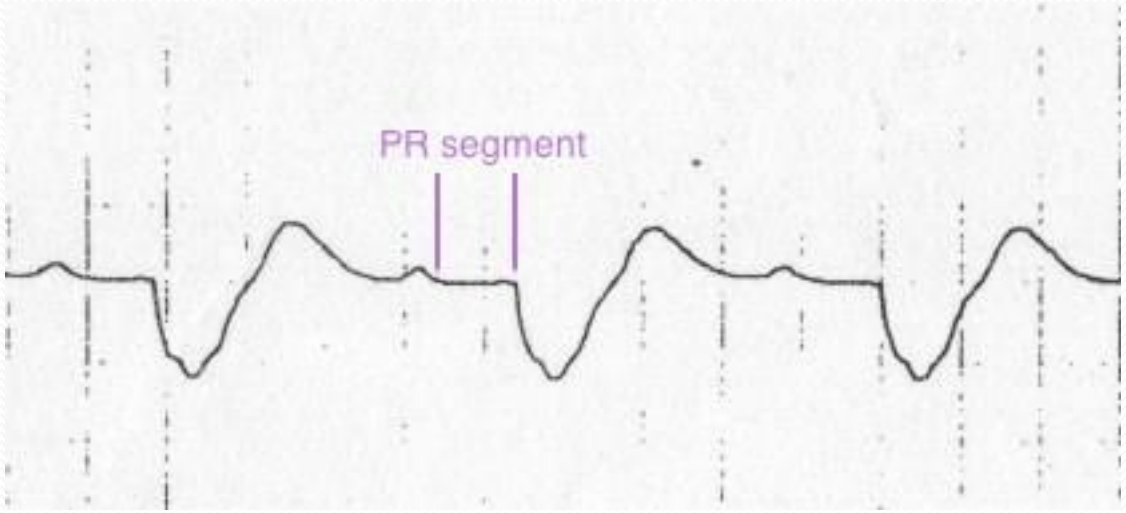

Referred by: Cci
COMMENT:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II



Peaked T waves



Broad QRS



Prolonged PR segment

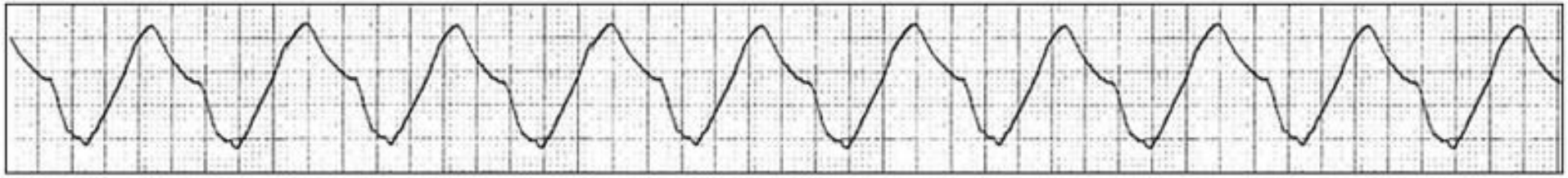
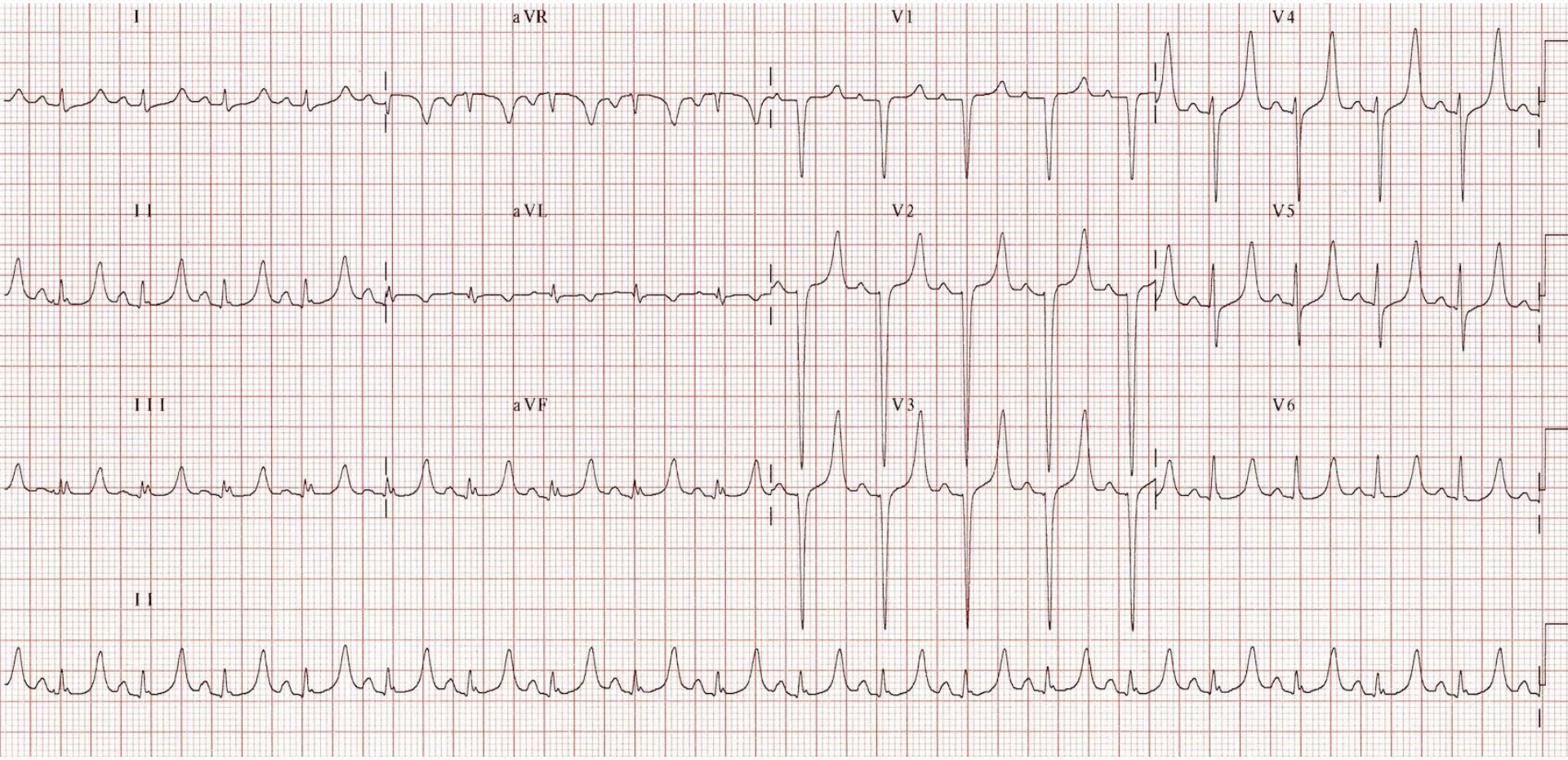
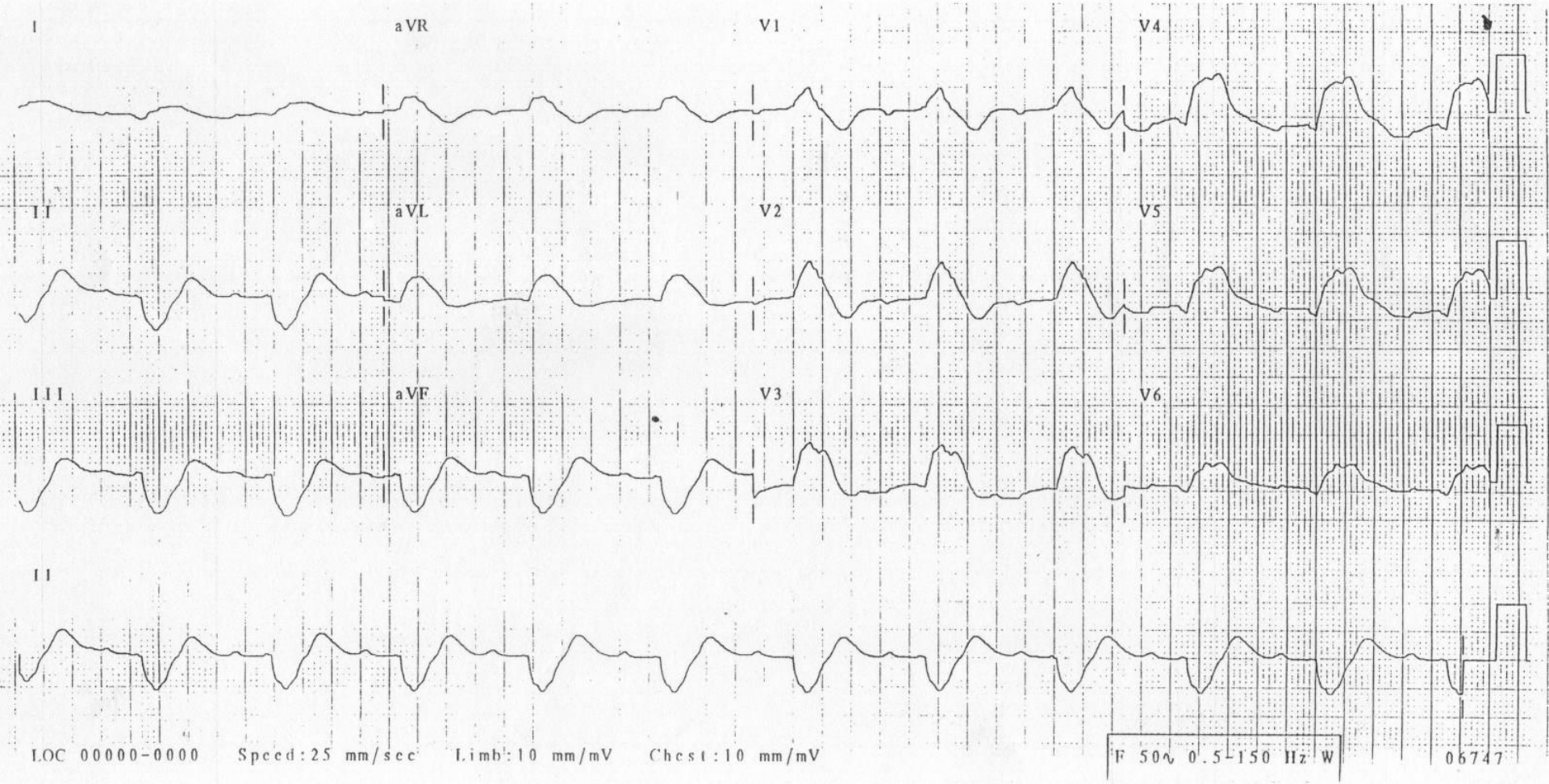


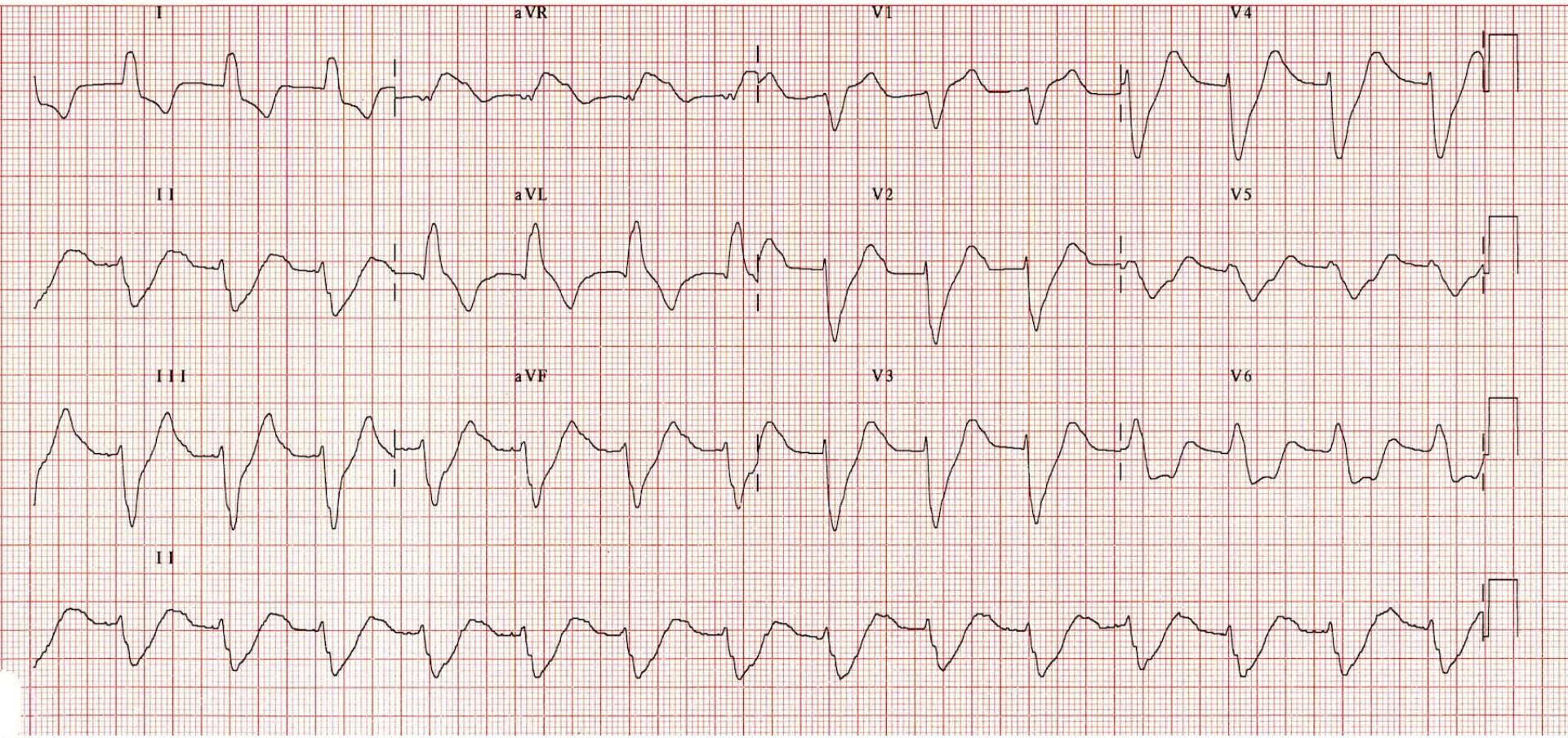
Figure 1 – This ECG rhythm strip shows a wide-complex bradycardic rhythm with sine-wave configuration and the absence of discernible P waves. These findings are consistent with pronounced hyperkalemia.



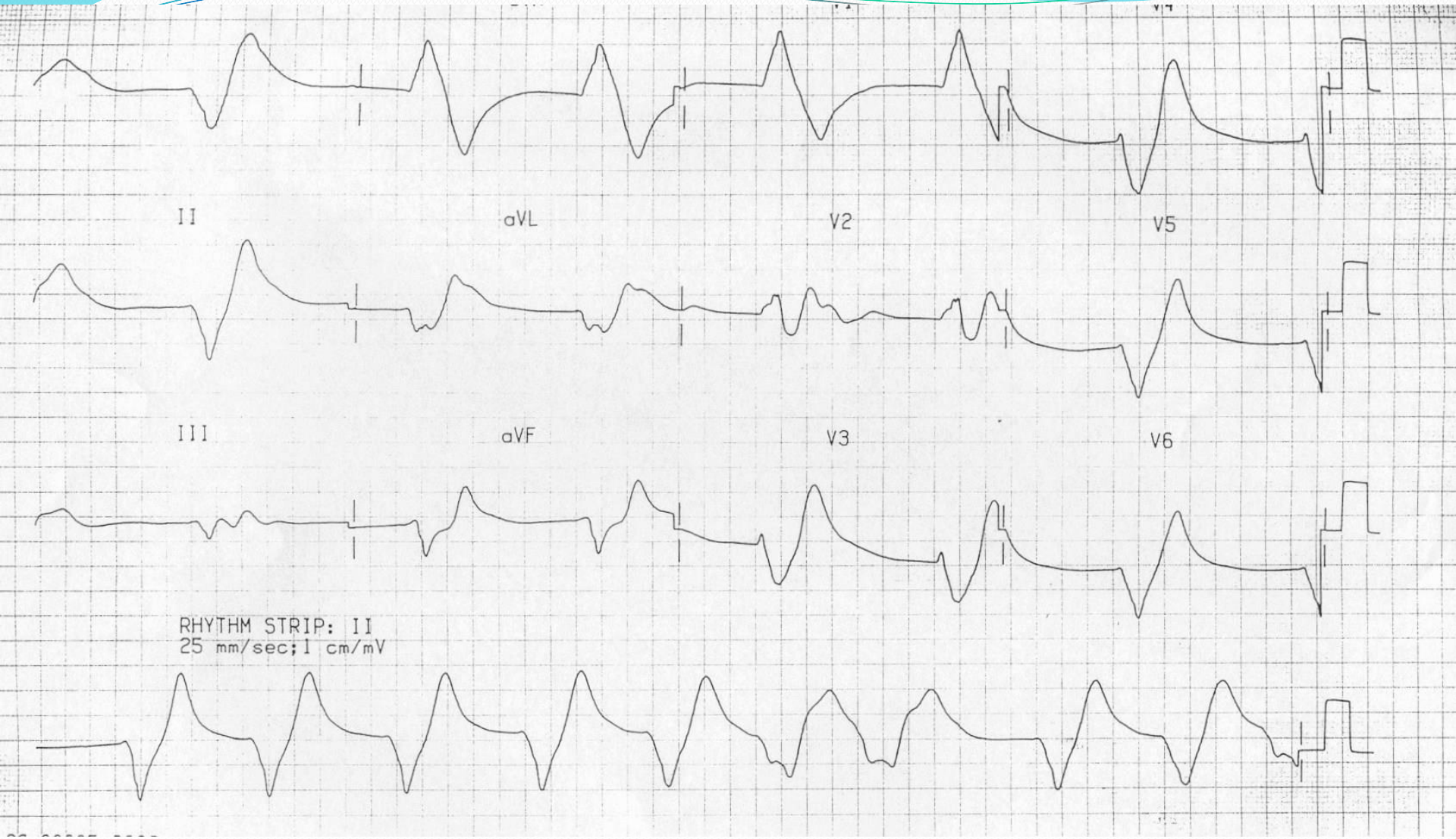
This patient had a serum K+ of 7.0



Long PR segment.
Wide, bizarre QRS.



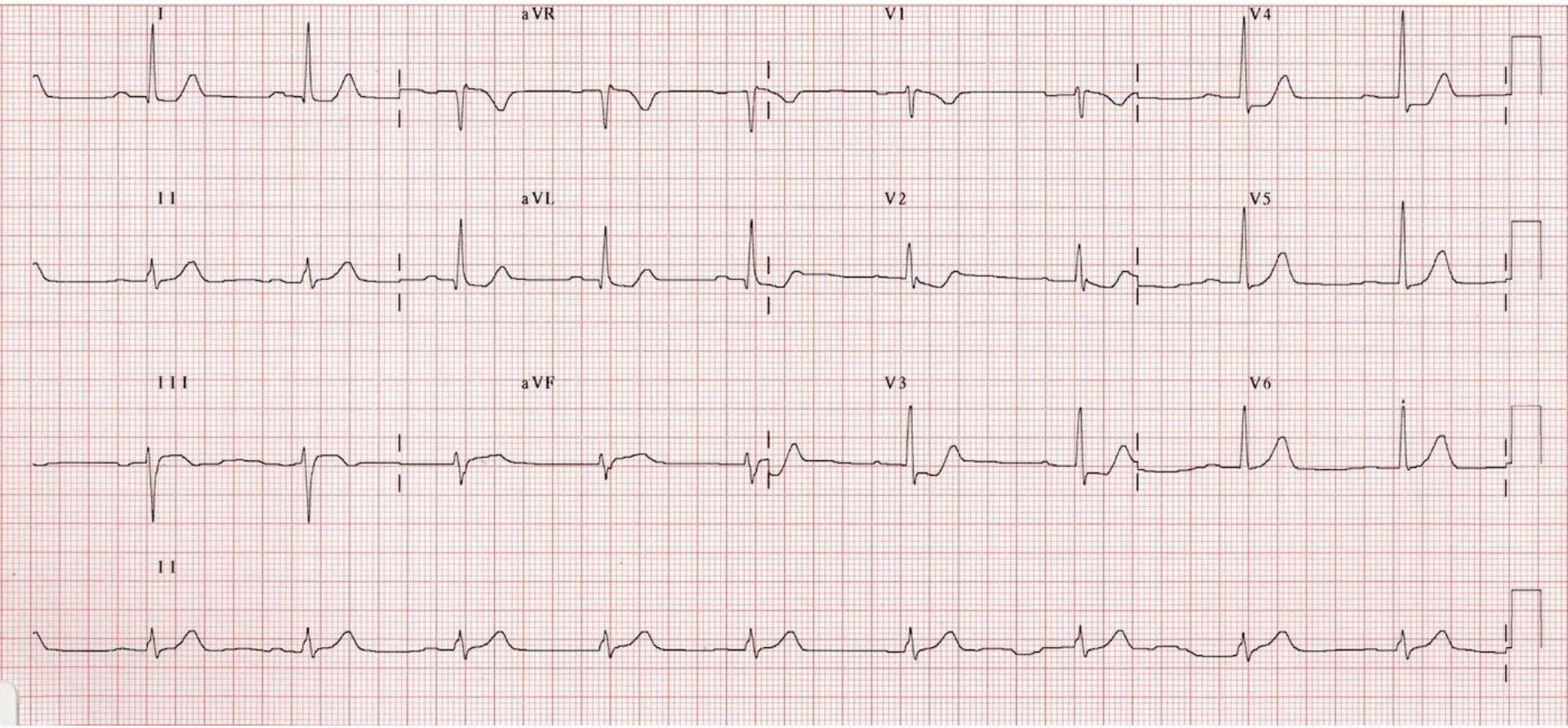
Broad complex rhythm with atypical LBBB morphology.
Left axis deviation.
Absent P waves



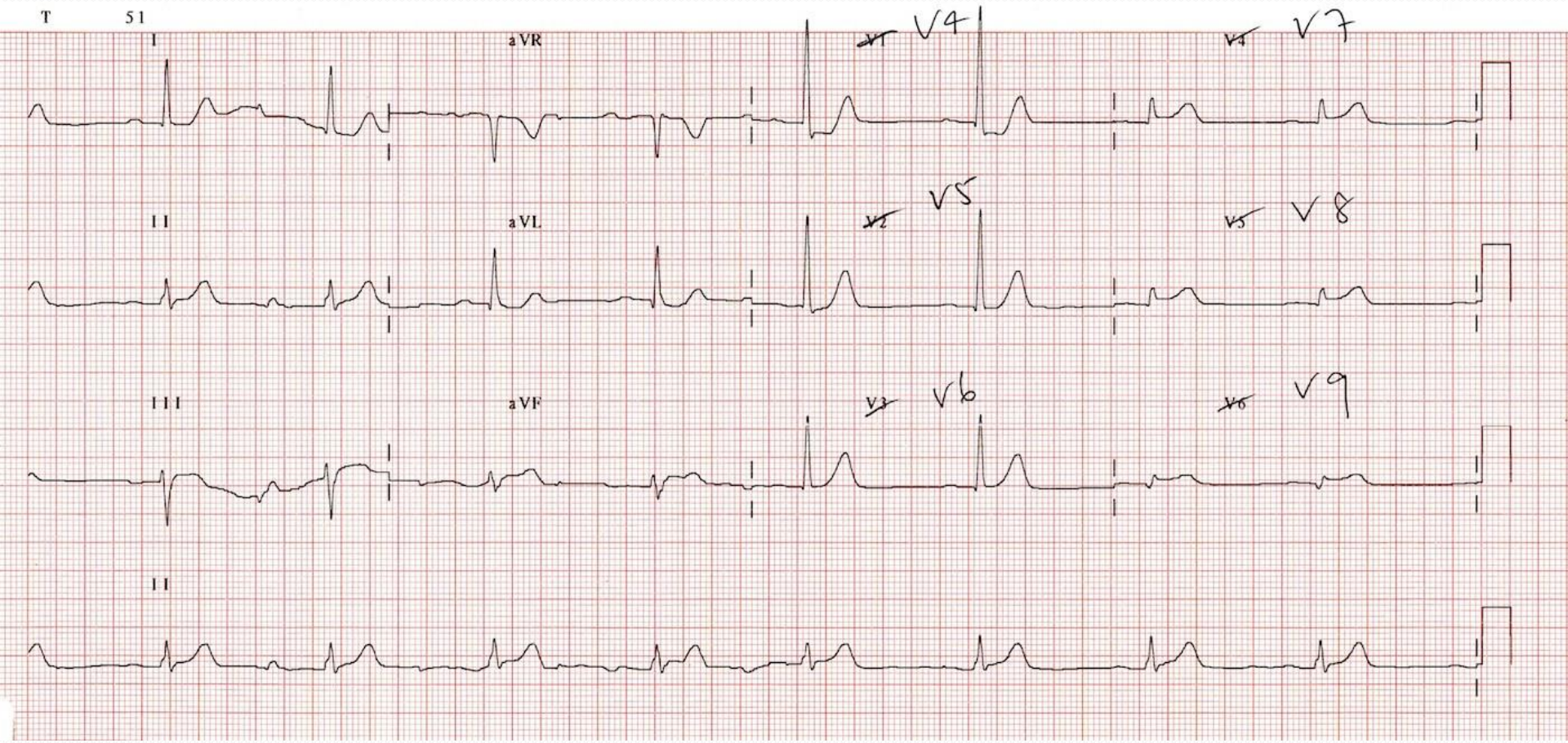
Sine wave appearance with severe hyperkalaemia (K+ 9.9 mEq/L)

Take home points

- Beware of Hyper K in any sick patient with potential metabolic problems
  - Get an ECG early
- Hyper K is the fastest cause of death in DKA
  - Early ECG in pts with hyperglycaemia that look sick
- Always consider Hyper K in pts presenting with bradycardia or CHB

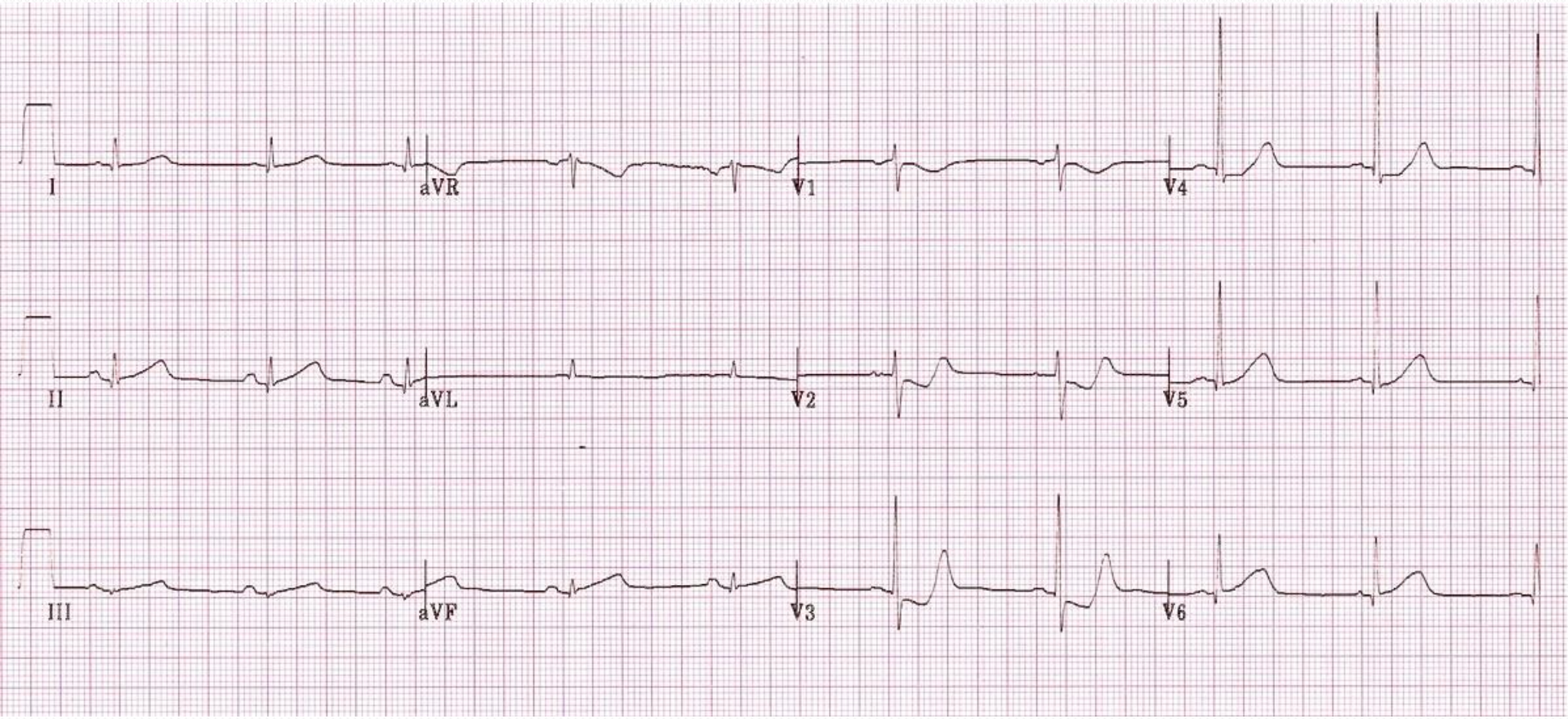


52yo male with central chest pain



Posterior leads

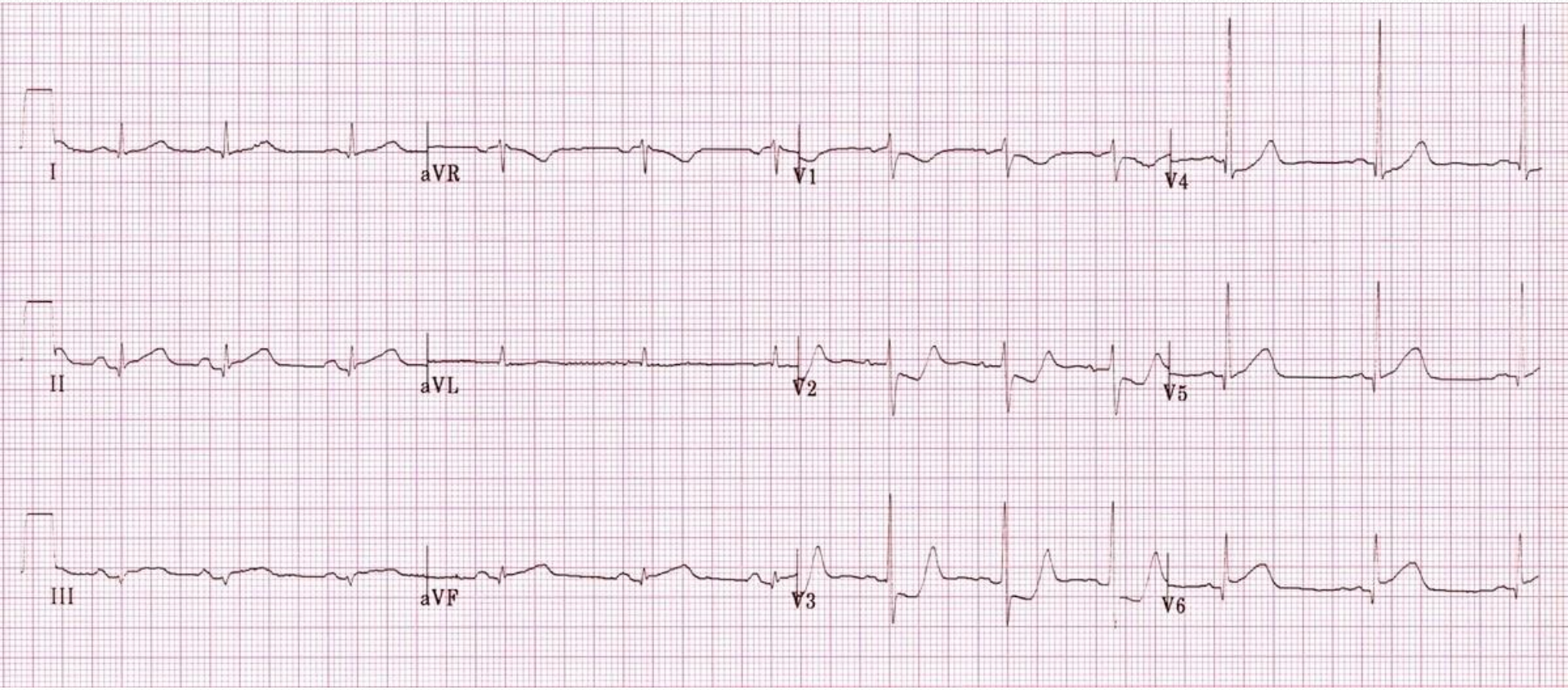
Posterior MI



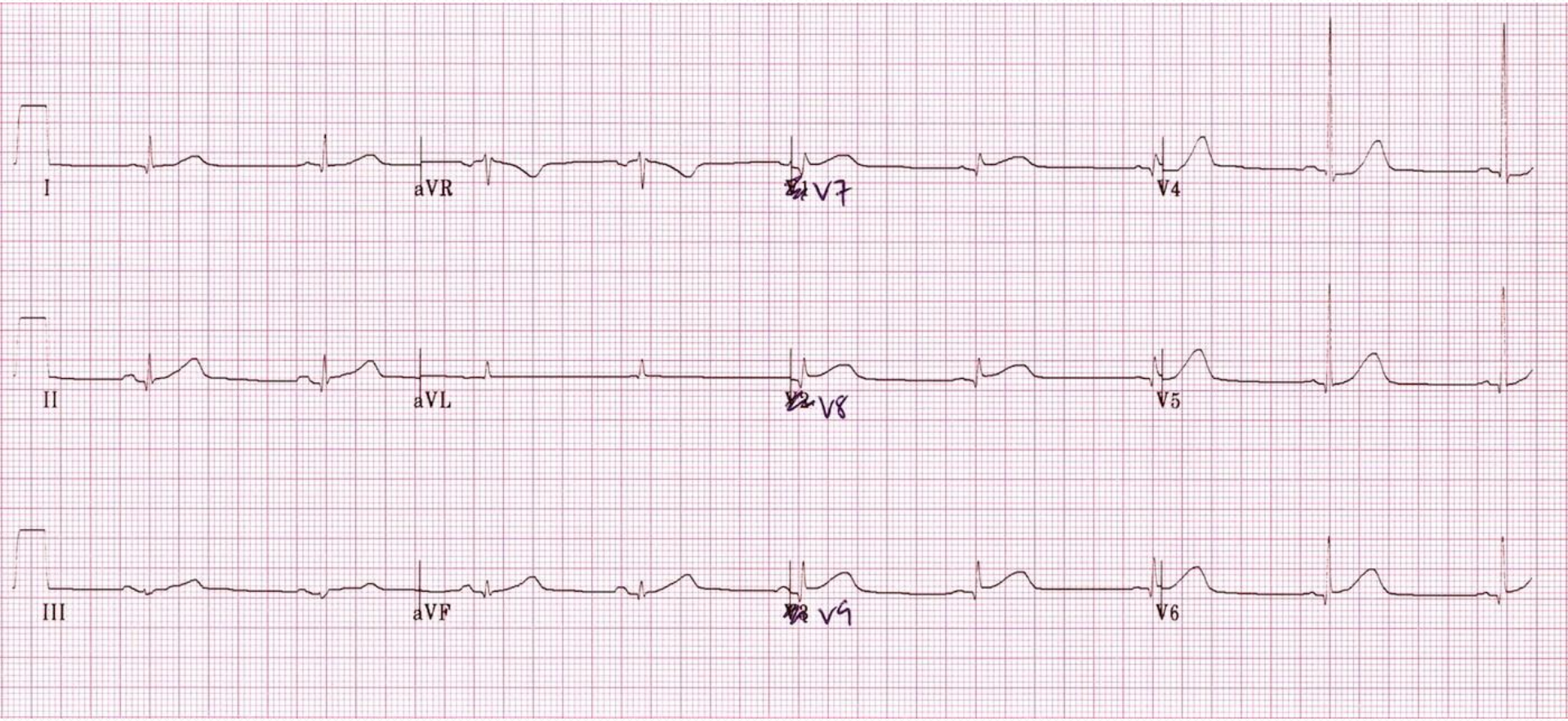
- Findings can be very, very subtle

- The ST depression and upright T waves in V2-3 suggest posterior MI.
- There are no dominant R waves in V1-2, but it is possible that this ECG was taken early in the course of the infarct, prior to pathological R-wave formation.
- There are also some features suggestive of early inferior infarction, with hyperacute T waves in II, III and aVF.

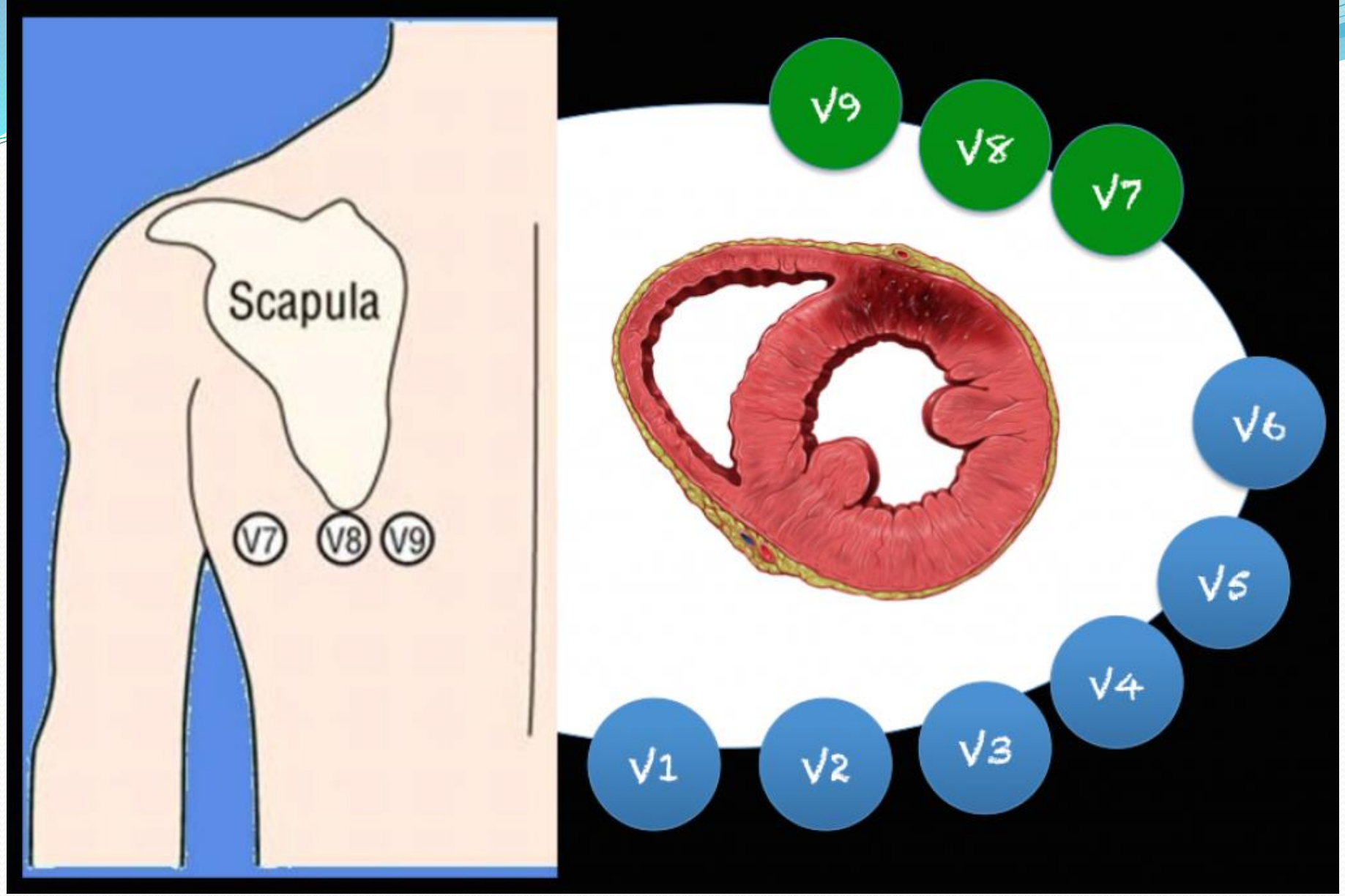
30mins later



- There is now some ST elevation developing in V6.
- With the eye of faith there is perhaps also some early ST elevation in the inferior leads (lead III looks particularly abnormal)



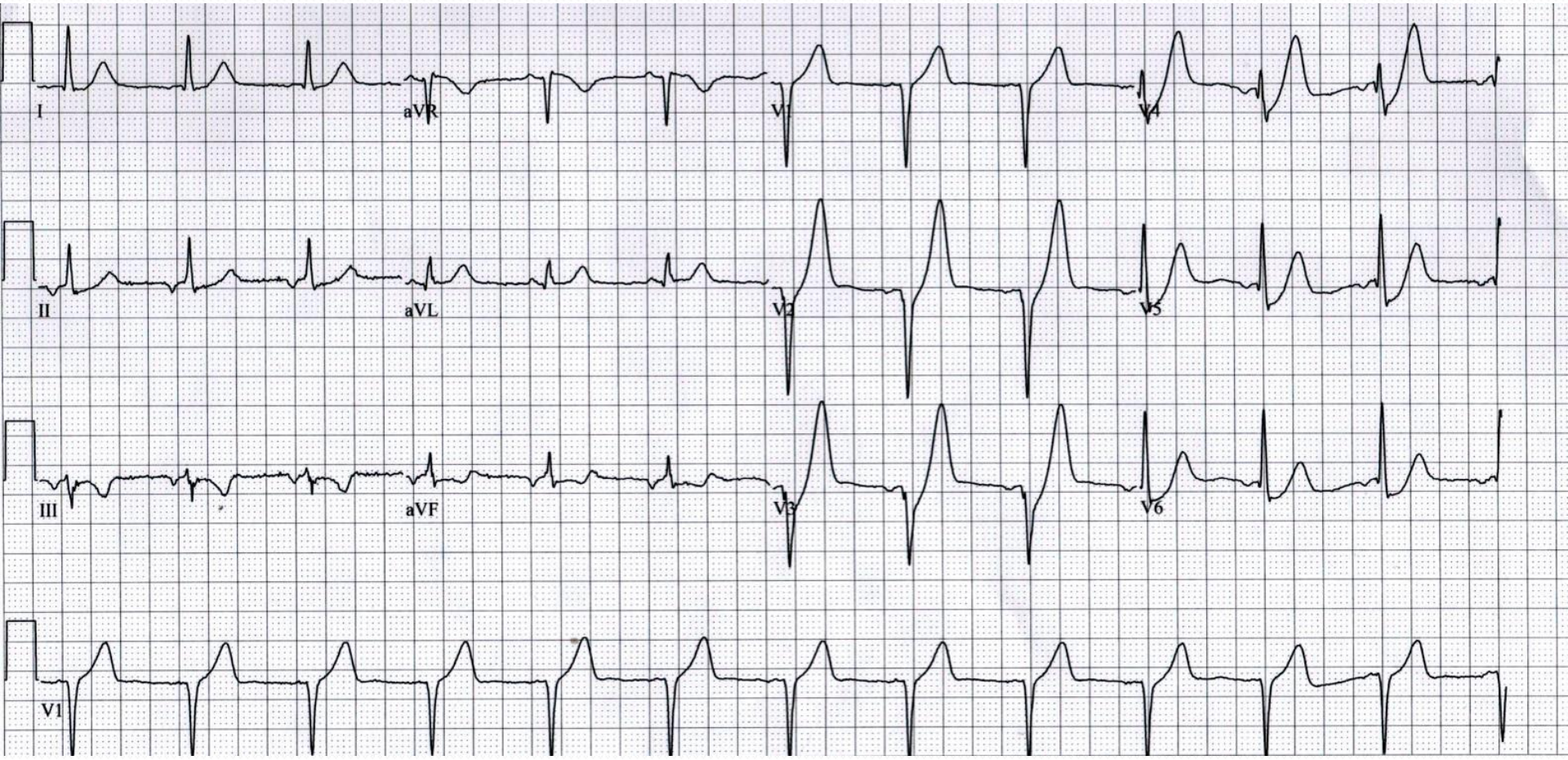
Posterior infarction is confirmed by the presence of ST elevation >0.5mm in leads V7-9.



V7 – Left posterior axillary line, in the same horizontal plane as V6.
V8 – Tip of the left scapula, in the same horizontal plane as V6.
V9 – Left paraspinal region, in the same horizontal plane as V6.

- Isolated posterior infarction is an indication for emergent coronary reperfusion.

- However, the lack of obvious STE in this condition means that the diagnosis is often missed



Contrast with this ECG

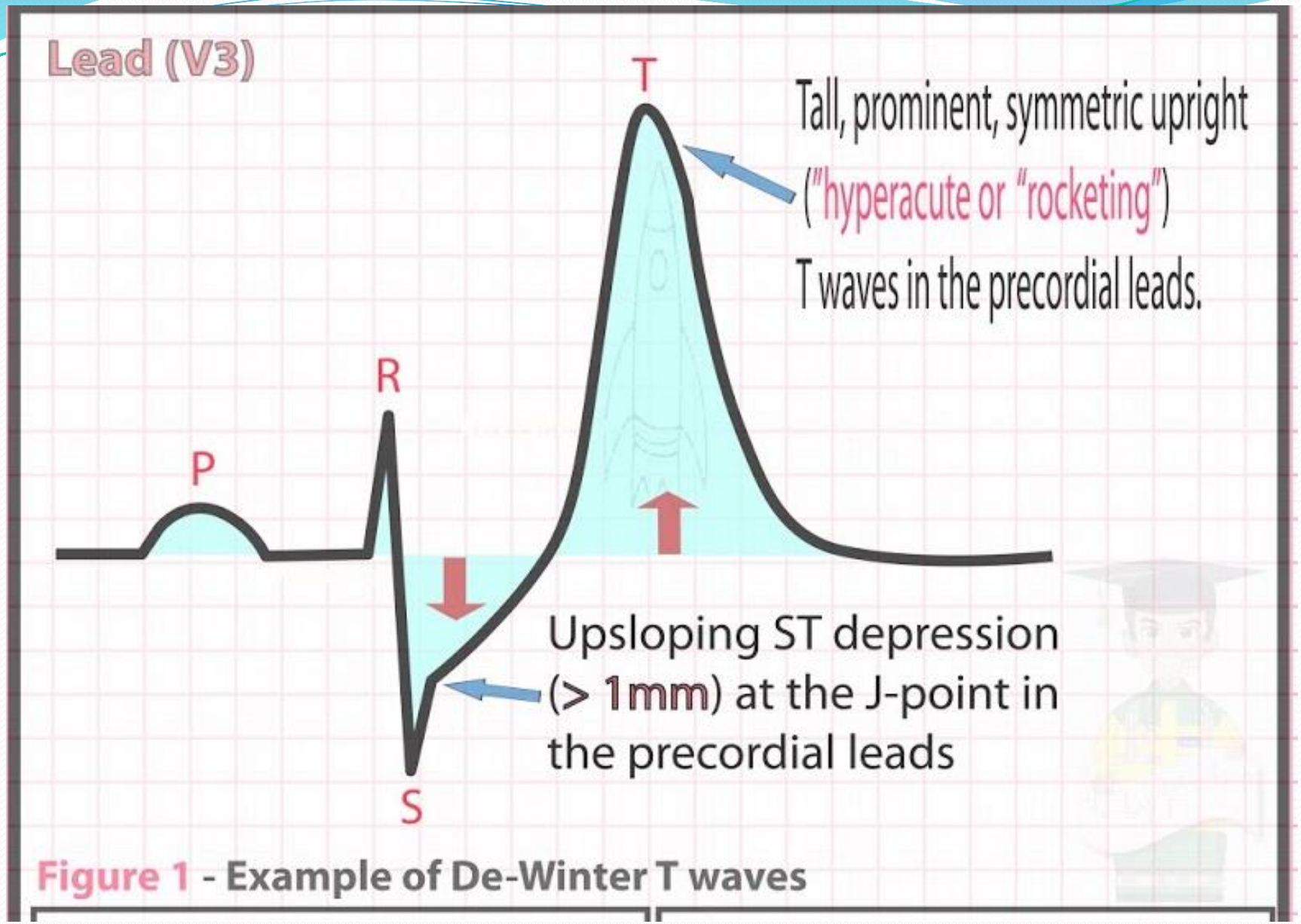
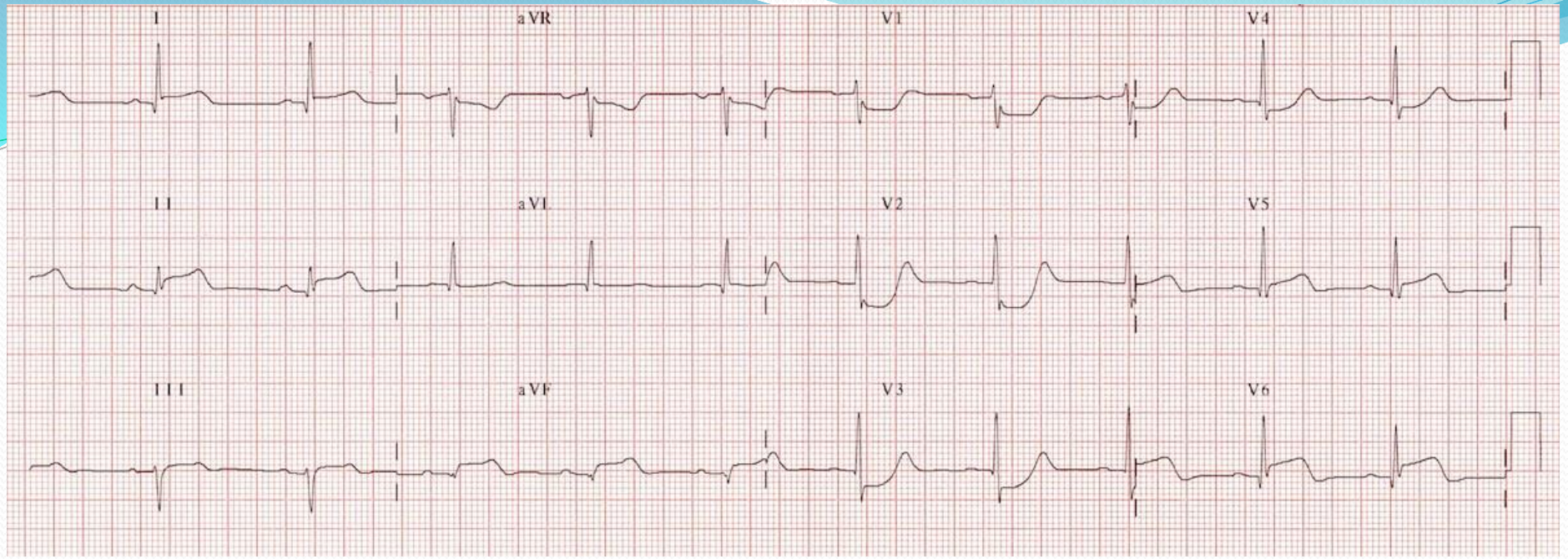
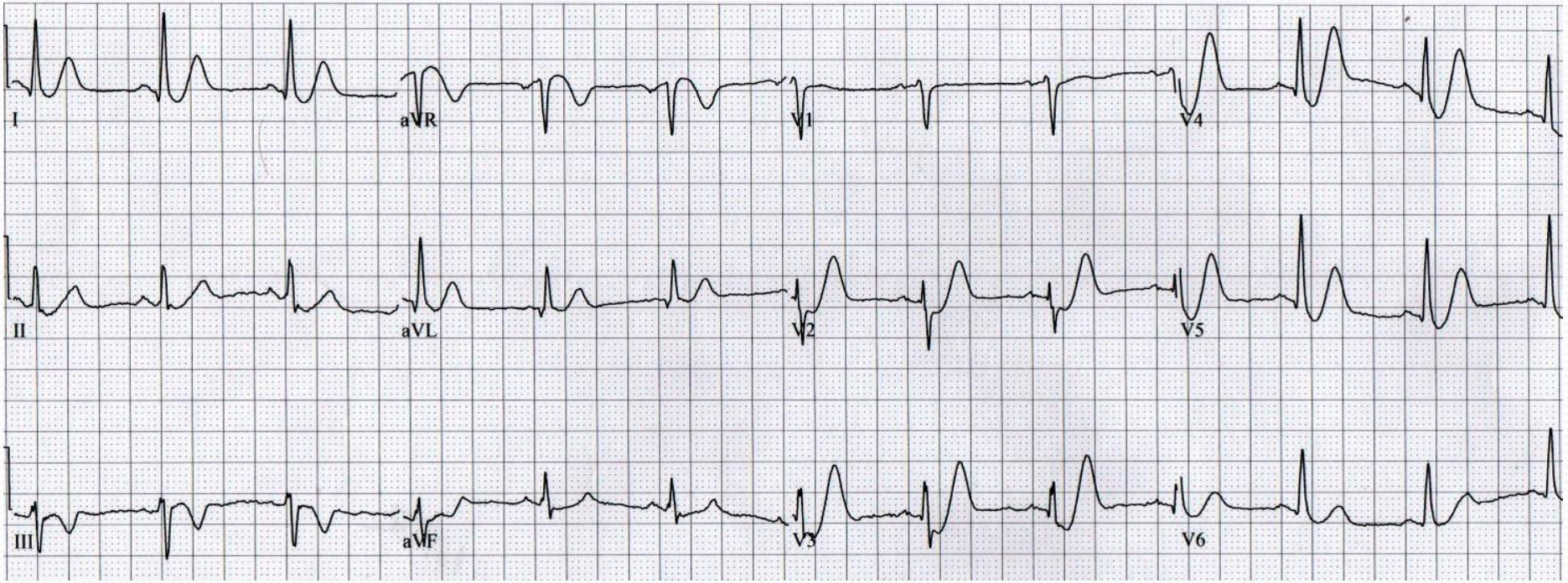
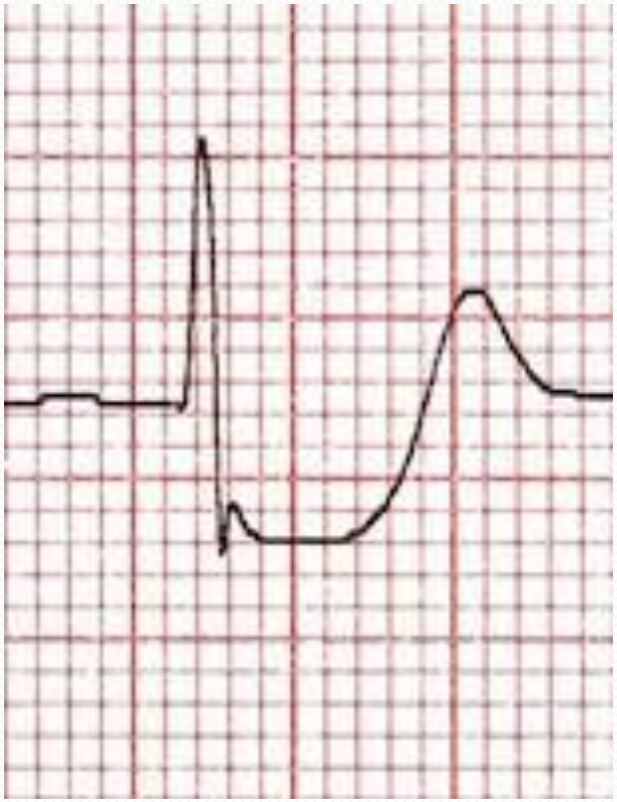


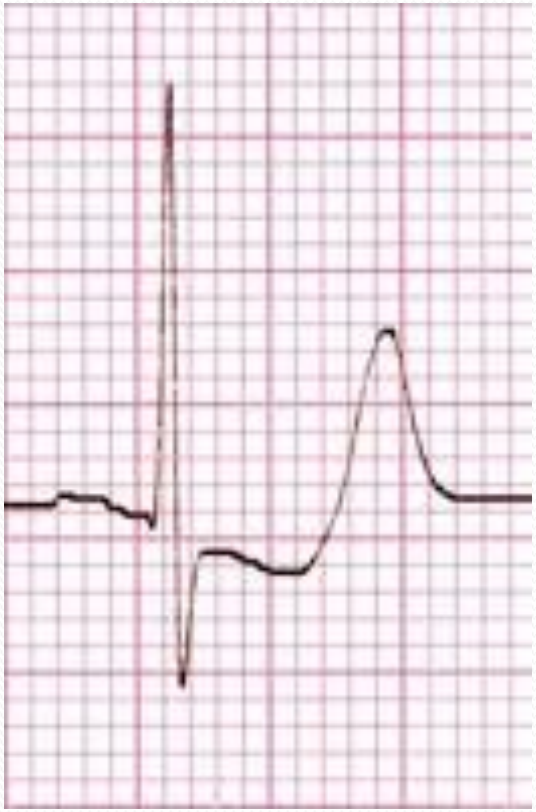
Figure 1 - Example of De-Winter T waves

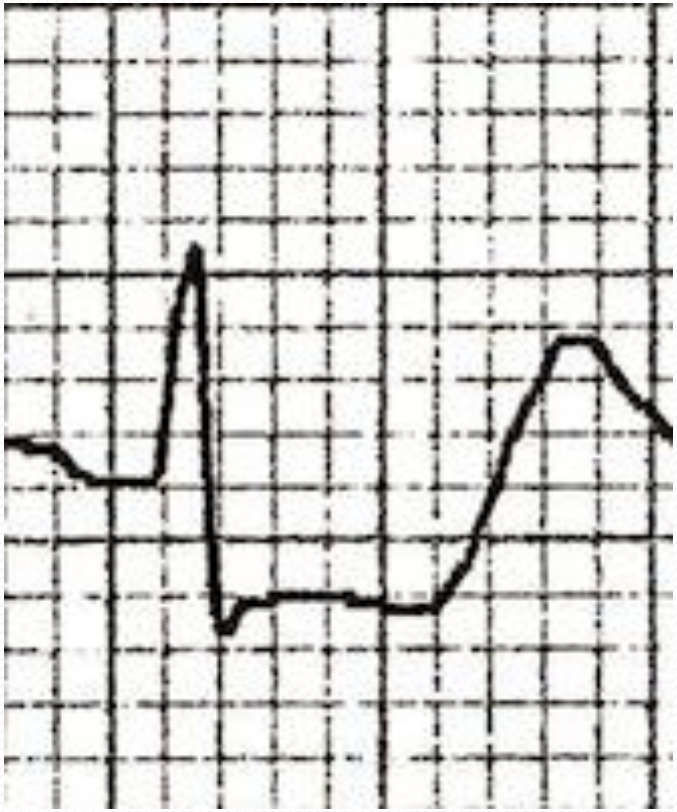

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

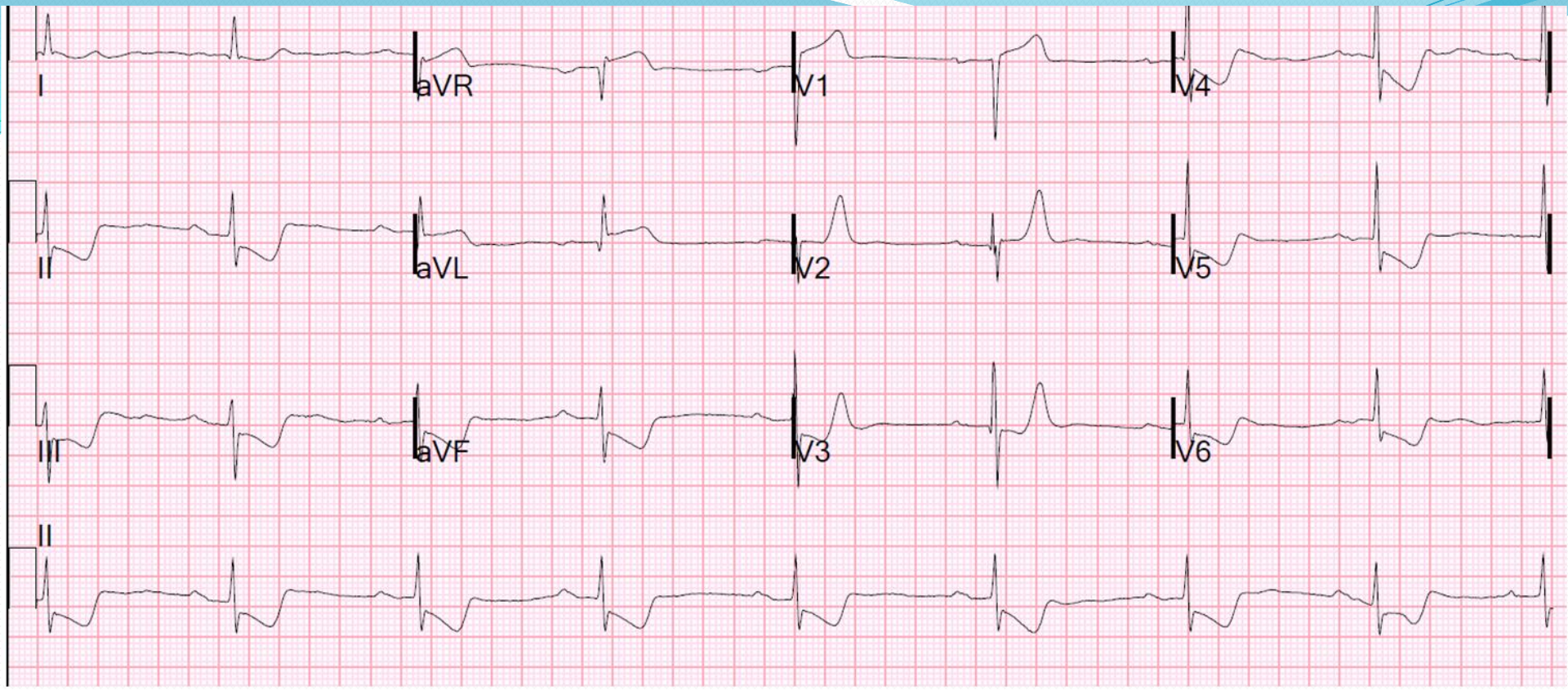

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

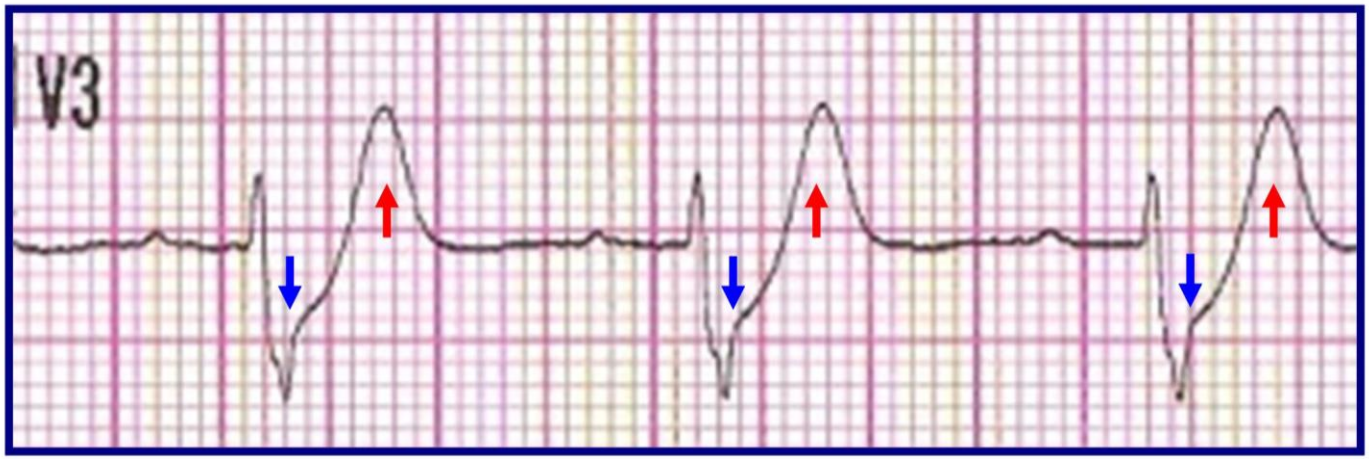
ST segment morphology in post MI

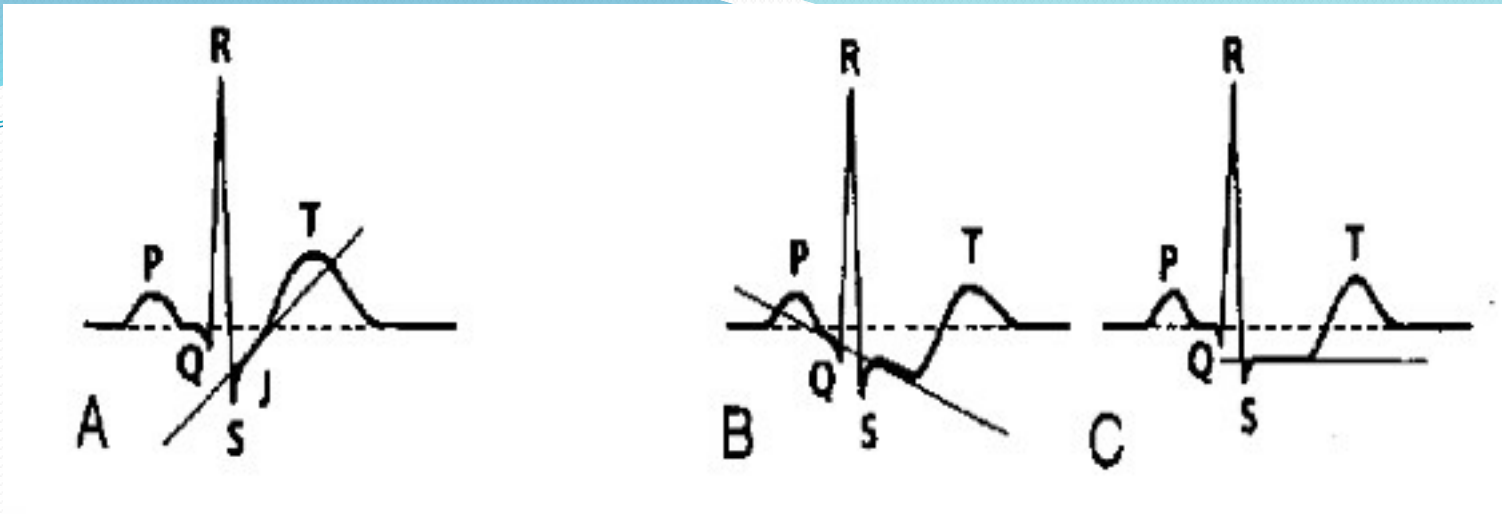


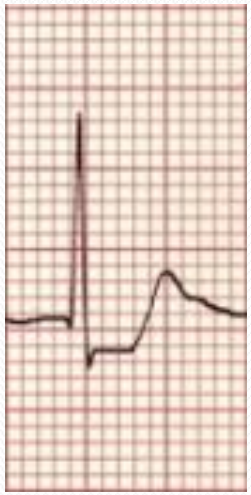


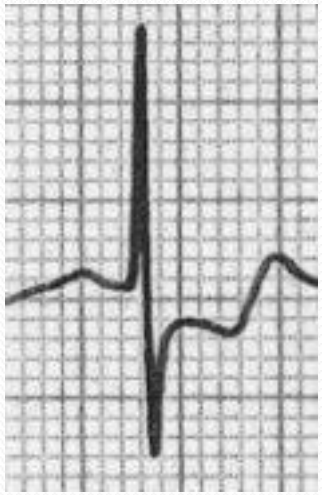


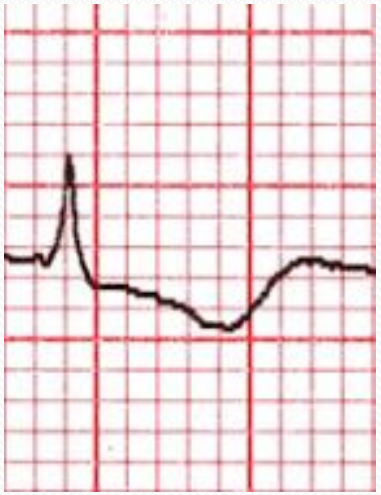

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

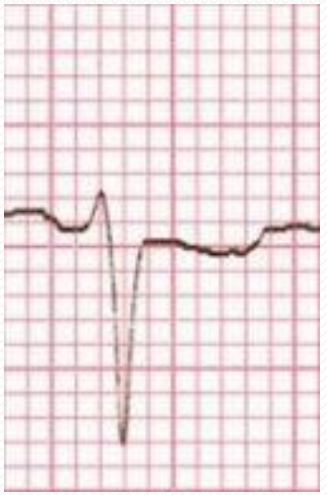

V3


R
P
T
Q
J
S
A
R
P
T
Q
S
B
R
P
T
Q
S
C



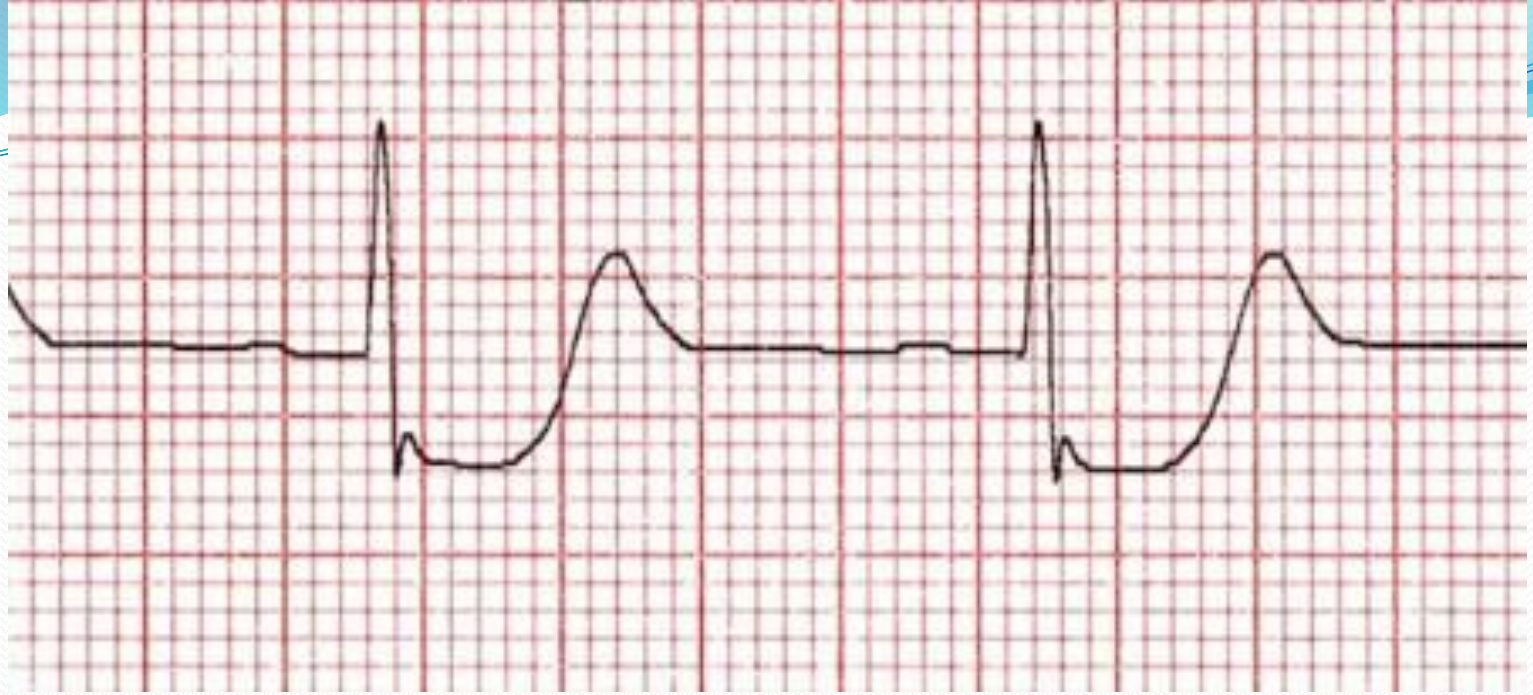


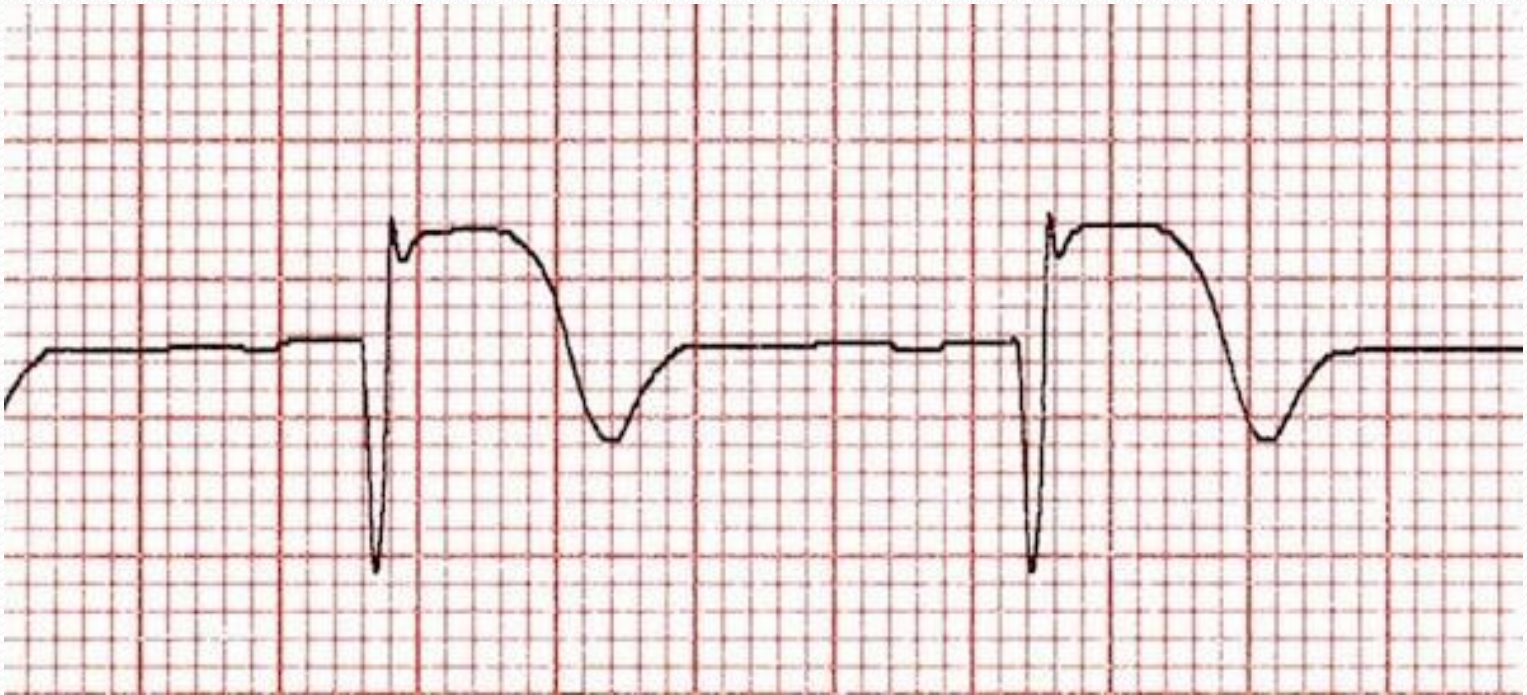


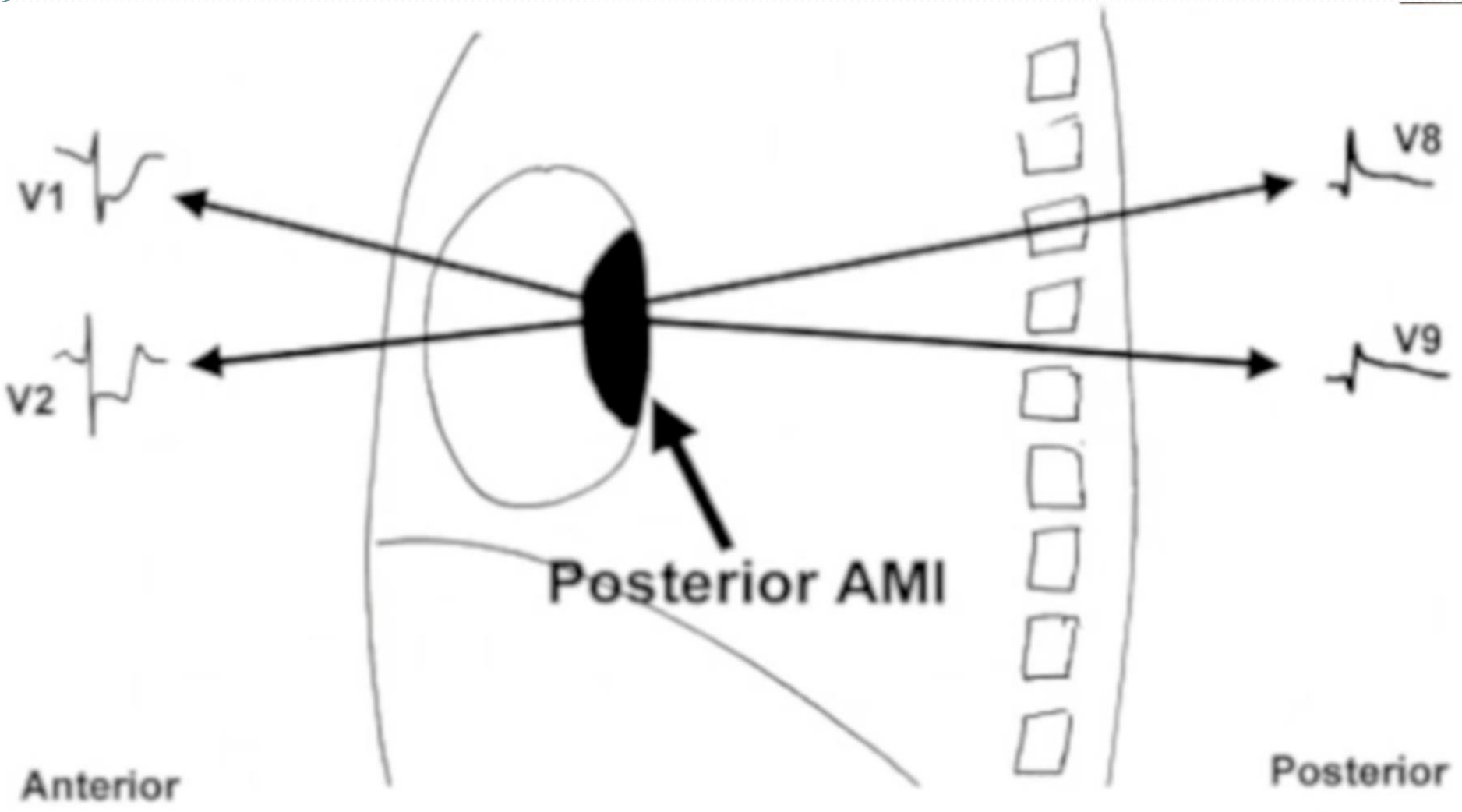


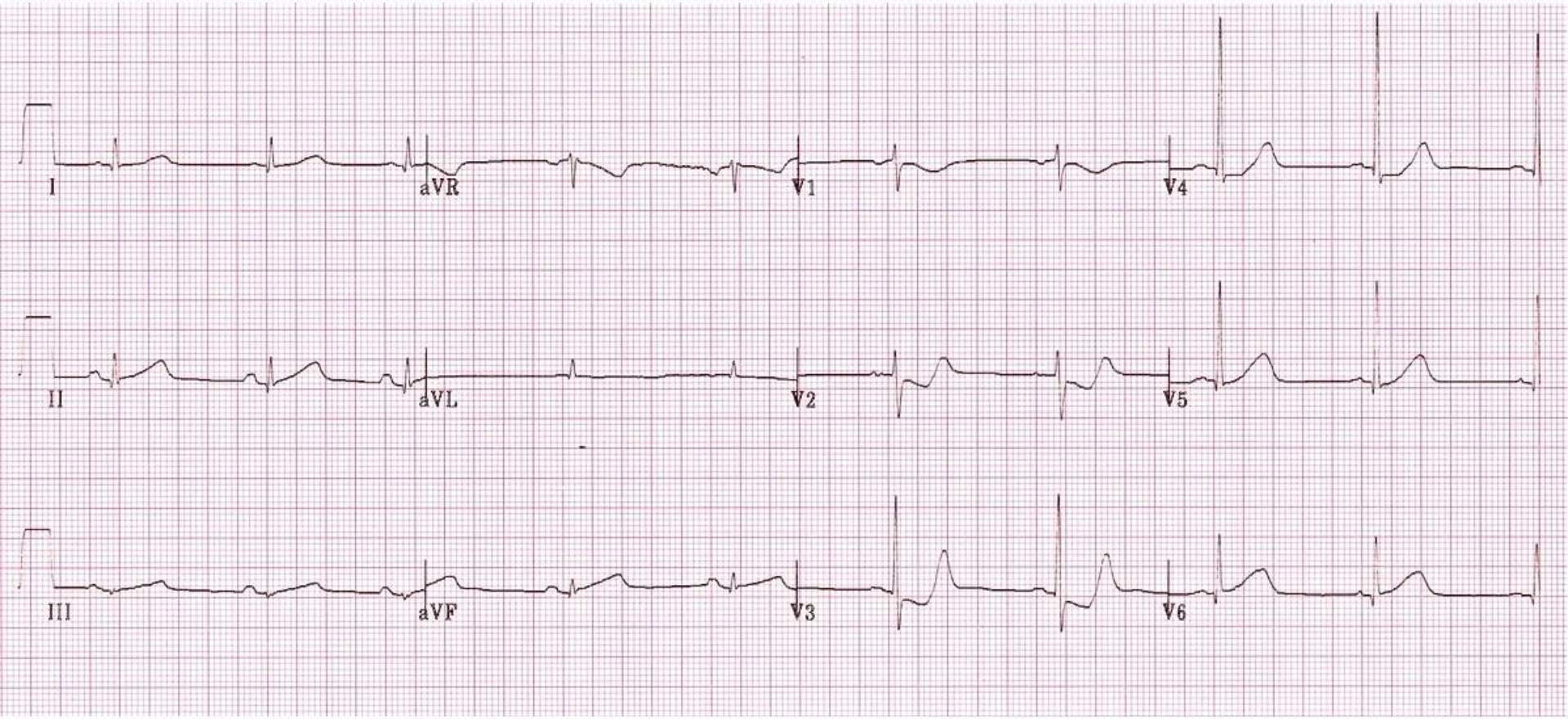
Posterior MI

- Horizontal ST depression
- Tall, broad R waves (>30ms)
- Upright T waves
- Dominant R wave (R/S ratio > 1) in V2

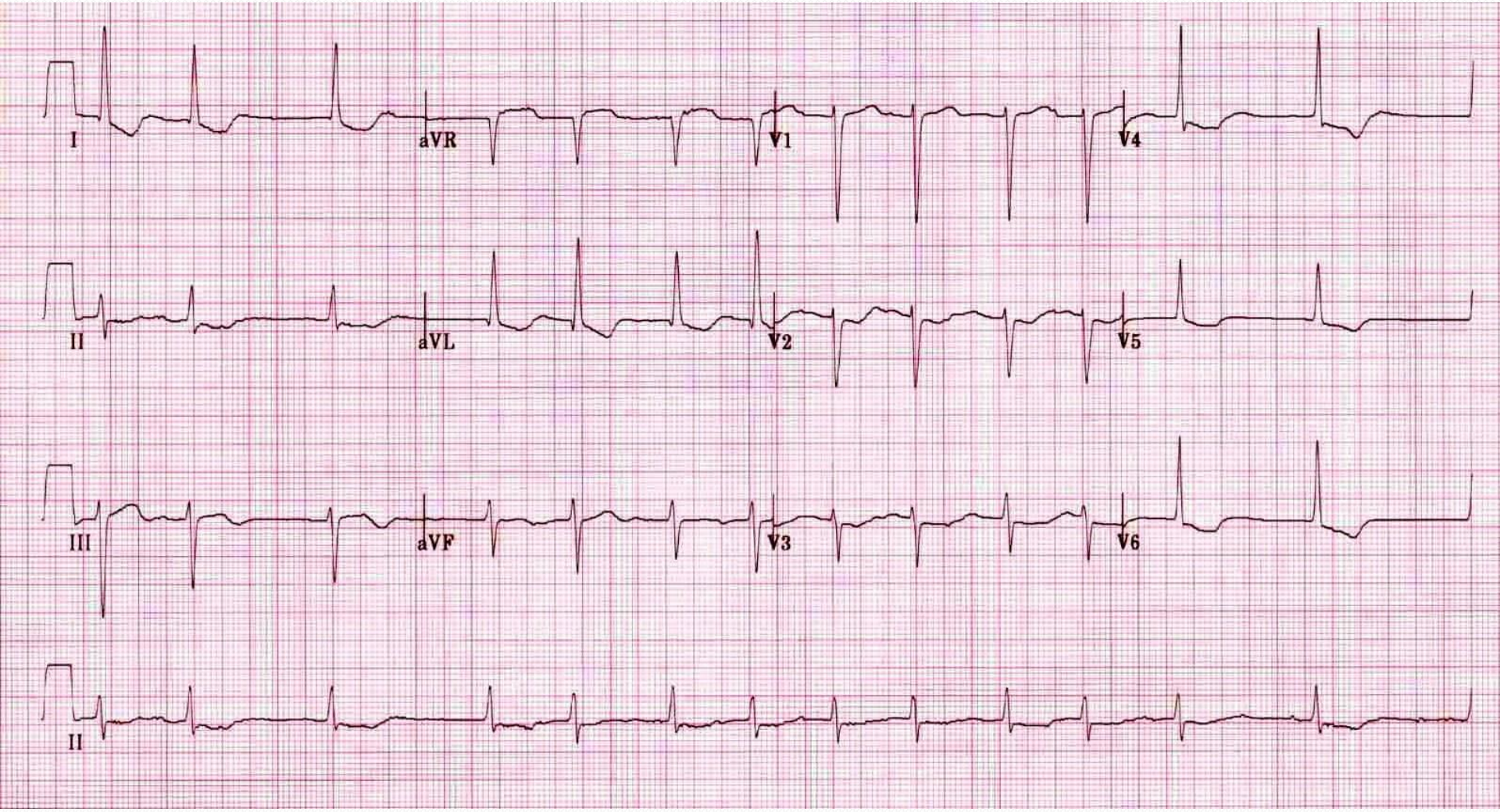




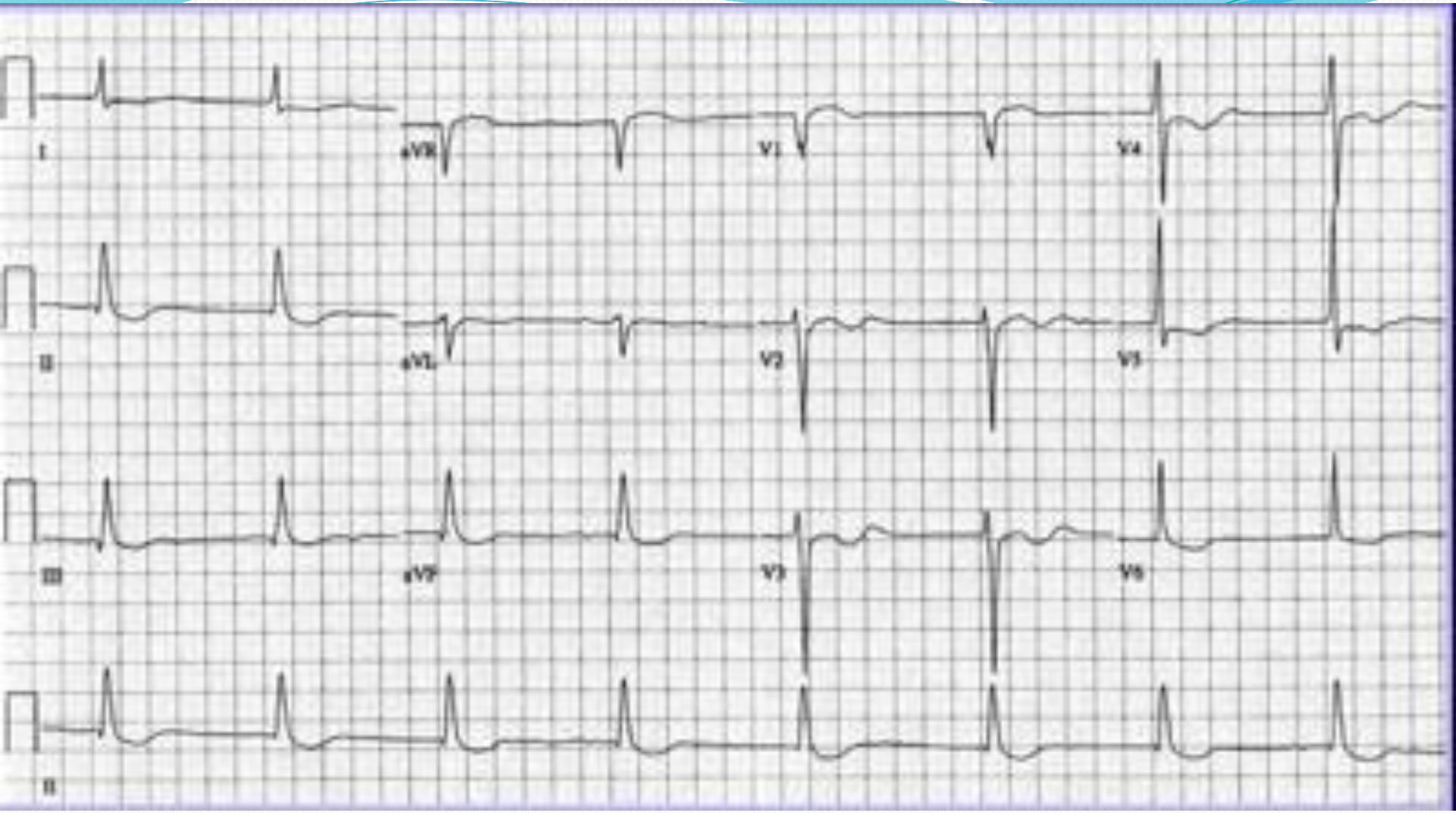

V1
V2
V8
V9
Posterior AMI
Anterior
Posterior


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

- ACS is high risk – but high pay off!
  - Very good outcome vs Very bad outcome
- Missed ACS – 30% mortality
  - In elderly – 50% 3 day mortality
- Isolated posterior infarction is an indication for emergent coronary reperfusion.
- However, the lack of obvious STE in this condition means that the diagnosis is often missed

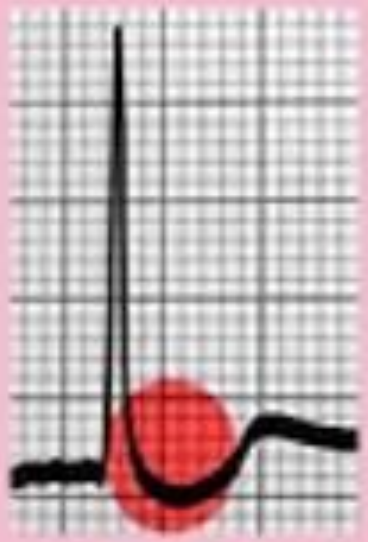


What is this?

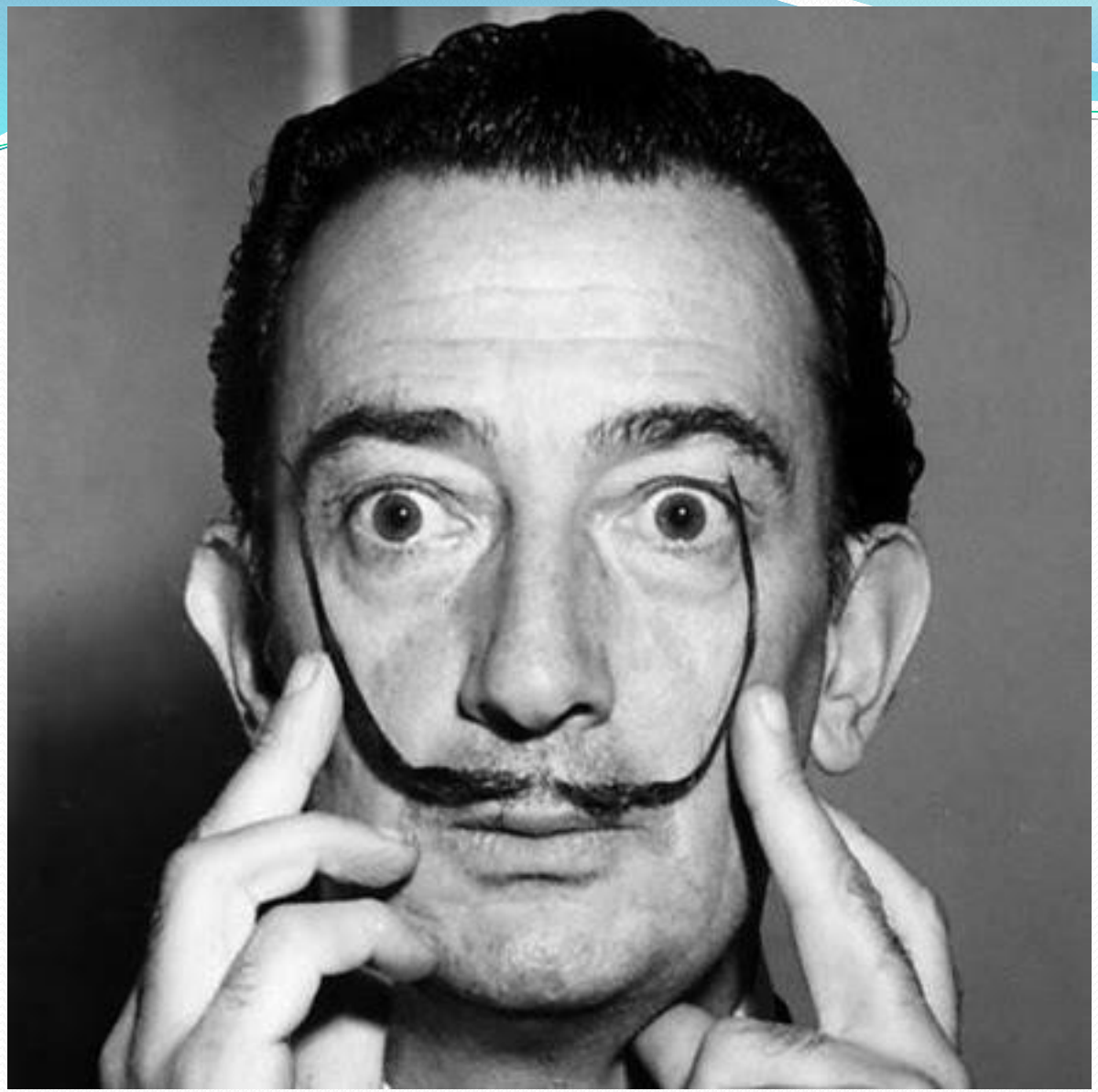


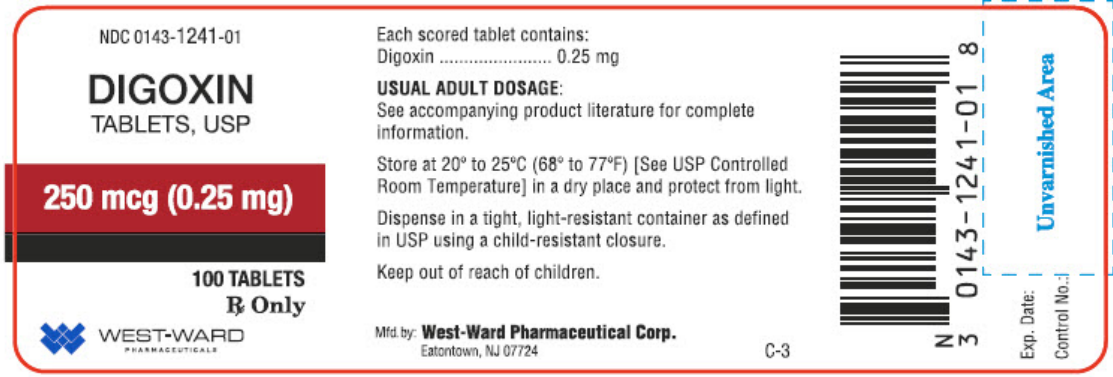
Is this the same?

Digoxin toxicity



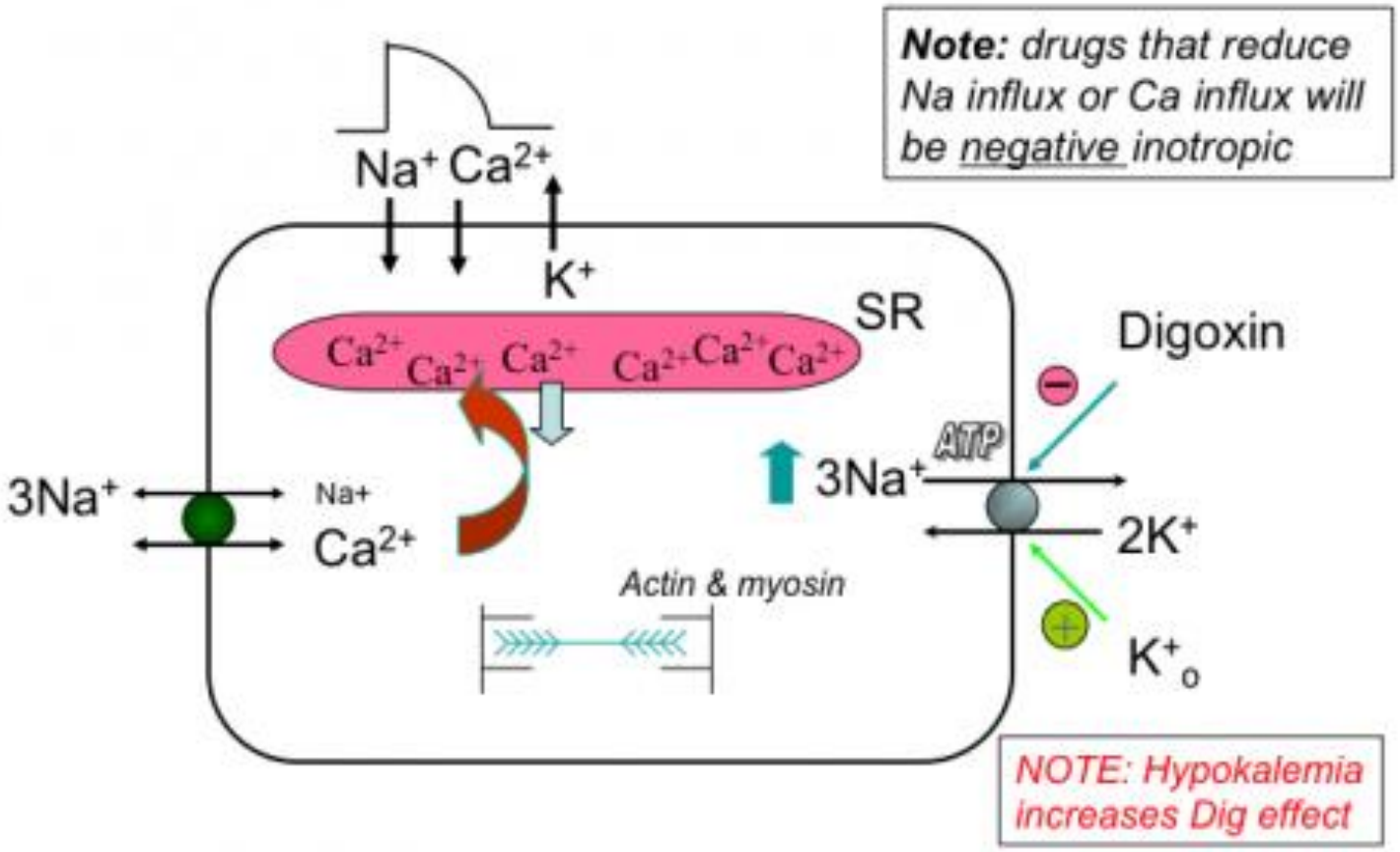
Digitalis effect





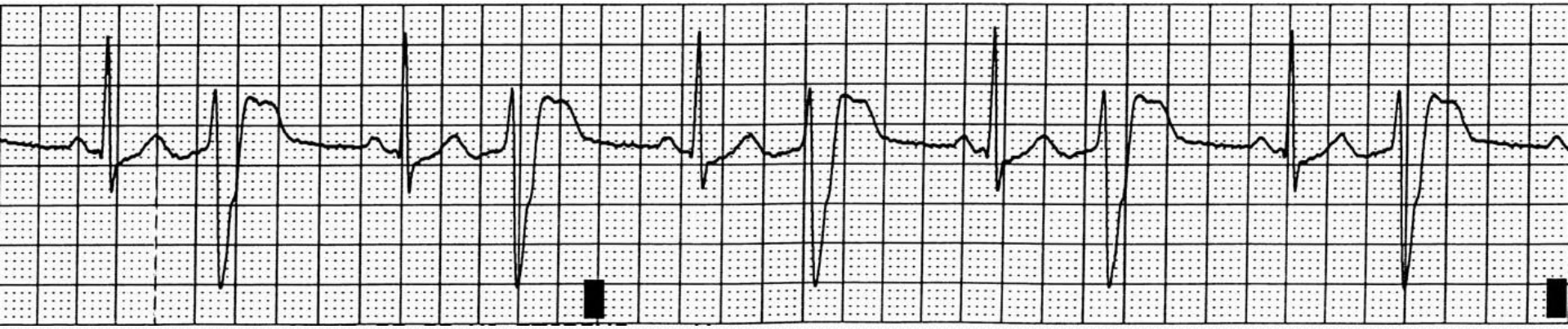
- Acute vs Chronic
- Acute if >10 times the daily dose ingested
- Potentially lethal dose predicted by:
  - >10mg in adult, >4mg in a child
  - S Digoxin level >15nmol/L
  - K > 5.5mmol/L

Mechanism of Positive Inotropic Action

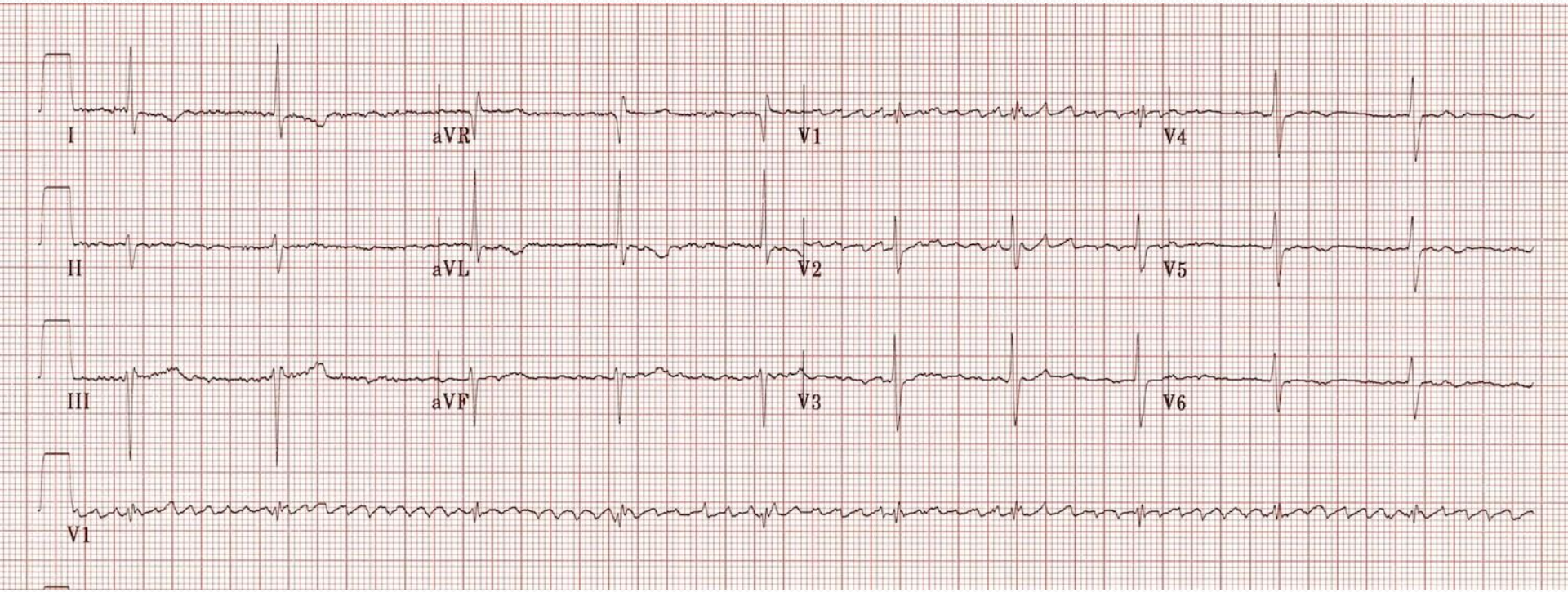


ECG features

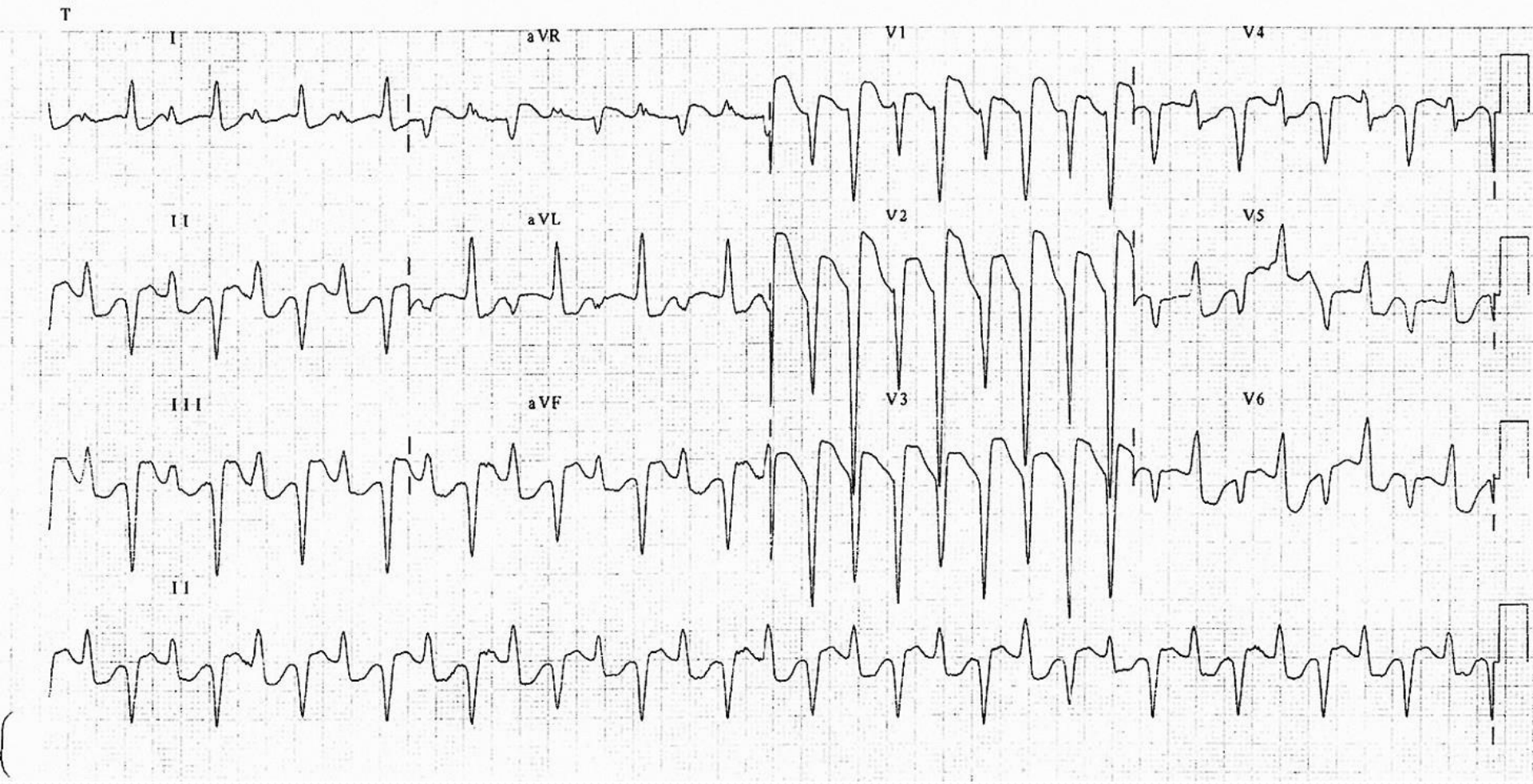
- Frequent PVCs (most common abnormality), including ventricular bigeminy and trigeminy
- Sinus bradycardia or slow AF
- Any type of AV block (1st , 2nd & 3rd degree)
- Regularised AF = AF with complete heart block and a junctional or ventricular escape rhythm
- VT, including polymorphic and bidirectional VT



Sinus rhythm with frequent PVCs in a pattern of ventricular bigeminy



Regularised AF: Coarse atrial fibrillation with 3rd degree AV block and a junctional escape rhythm.


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

- In cardiac arrest – std resuscitation measures futile
- ACLS + 20 ampoules of Digibind

38 mg NDC 0173-0230-44

DIGOXIN IMMUNE FAB (OVINE)

DIGIBIND®

NEUTRALIZES 500 µg DIGOXIN

Sterile Lyophilized Powder for Intravenous Injection

℞ only

Each vial contains 38 mg Digoxin Immune Fab (Ovine), 75 mg sorbitol and 28 mg of sodium chloride. No preservative. For indications, dosage, precautions, etc., see enclosed package insert. Refrigerate at 2 to 8°C (36 to 46°F).

Manufactured by
GlaxoSmithKline, SpA
Parma, Italy
US License No. 129

Distributed by
GlaxoSmithKline
Research Triangle Park, NC 27709

Made in Italy

When to give Digibind?

- Cardiac arrest
- Life threatening Cardiac dysrhythmia
- Ingested dose > 10mg (>4mg in children)
- S Dig level >15nmol/L (>12ng/mL)
- S K^+ > 5mmol/l

Pt with CP who said “Yes” to every question!

Name: , ID: 99999999 05 Dec 2015 02:20

55 years
Male

Loc:1
Room: 12

Vent. rate	74	/min
PR interval	186	ms
QRS duration	100	ms
QT/QTc	362/402	ms
P–R–T axes	71	59 69

*** Age and gender specific ECG analysis ***
Normal sinus rhythm
ST elevation, consider anterolateral injury or acute infarct
ST elevation, consider inferior injury or acute infarct
** ** ACUTE MI ** **
Abnormal ECG

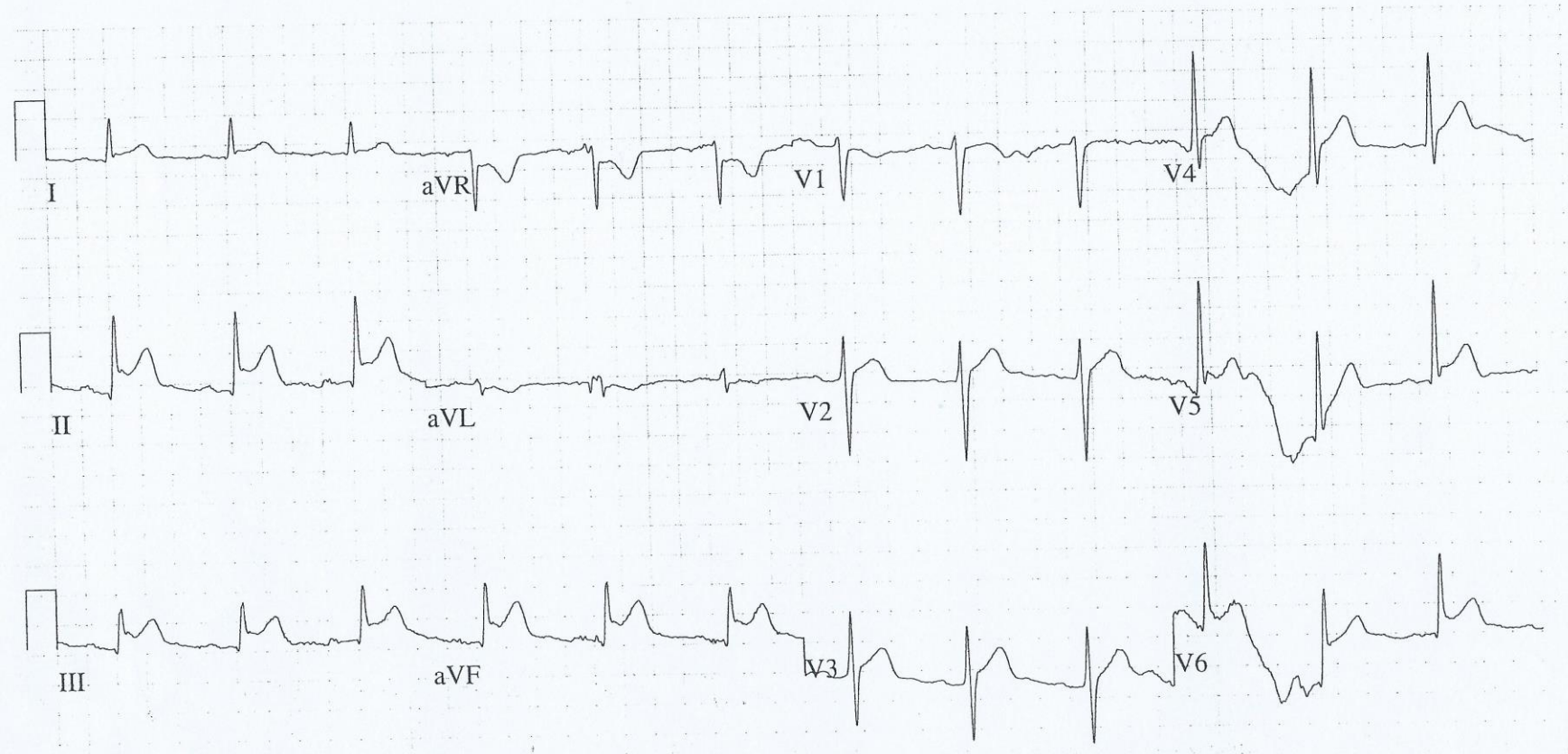
(1)

15/01/1958
Male 57 years, 10 months
Ward: EMERG
MC: 31330049311

3 Yvonne Court
MAIDEN GULLY VIC, 3551
Ph: 54496796 M: 0448064175
Public - Eligible
Cardiology
Adm: Saturday 5th December 2015
GP: Dr BABOVIC Avram, Bendigo Medical Centre - Bridge Street
Time:
Sign: .....

pre

CCP 9/10



25mm/s 10.0mm/mV 100H
GEMS Information Technologies

55 years
Male

Loc:1
Room: 12

Vent. rate	75	/min
PR interval	*	ms
QRS duration	100	ms
QT/QTc	372/415	ms
P–R–T axes	* 61	72

*** Age and gender specific ECG analysis ***
Undetermined rhythm
ST elevation, consider inferolateral injury or acute infarct
** ** ACUTE MI ** **
Abnormal ECG

2

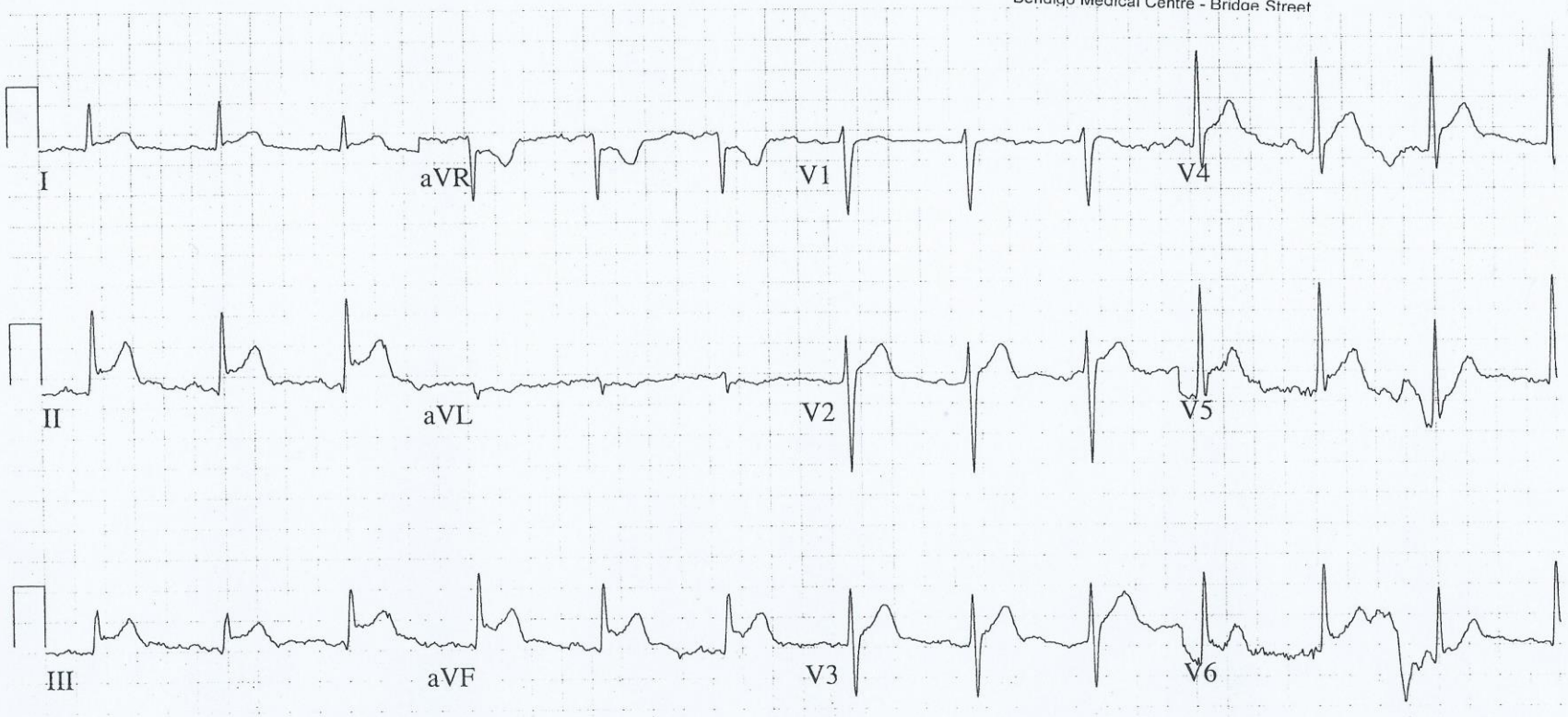
ST I – III
V3 – V6
inf & V6 – L47

[redacted]
MAIDEN GULLY VIC,3551
Ph: 54496796 M: 0448064175

15/01/1958
57 years, 10 months
Male

Public MC: 31330049311

GP: Dr BABOVIC Avram
Bendigo Medical Centre - Bridge Street



25mm/s 10.0mm/mV 100H
GEMS Information Technologies

Name: , ID: 999999999 03-Dec-2013 03:30

8

55 years

Loc:5
Room: RES-3

Vent. rate	69	/min
PR interval	190	ms
QRS duration	104	ms
QT/QTc	372/398	ms
P-R-T axes	68 60 68	

*** Age and gender specific ECG analysis ***
Normal sinus rhythm
ST elevation, consider anterolateral injury or acute infarct
ST elevation, consider inferior injury or acute infarct
** ** ACUTE MI ** **
Abnormal ECG

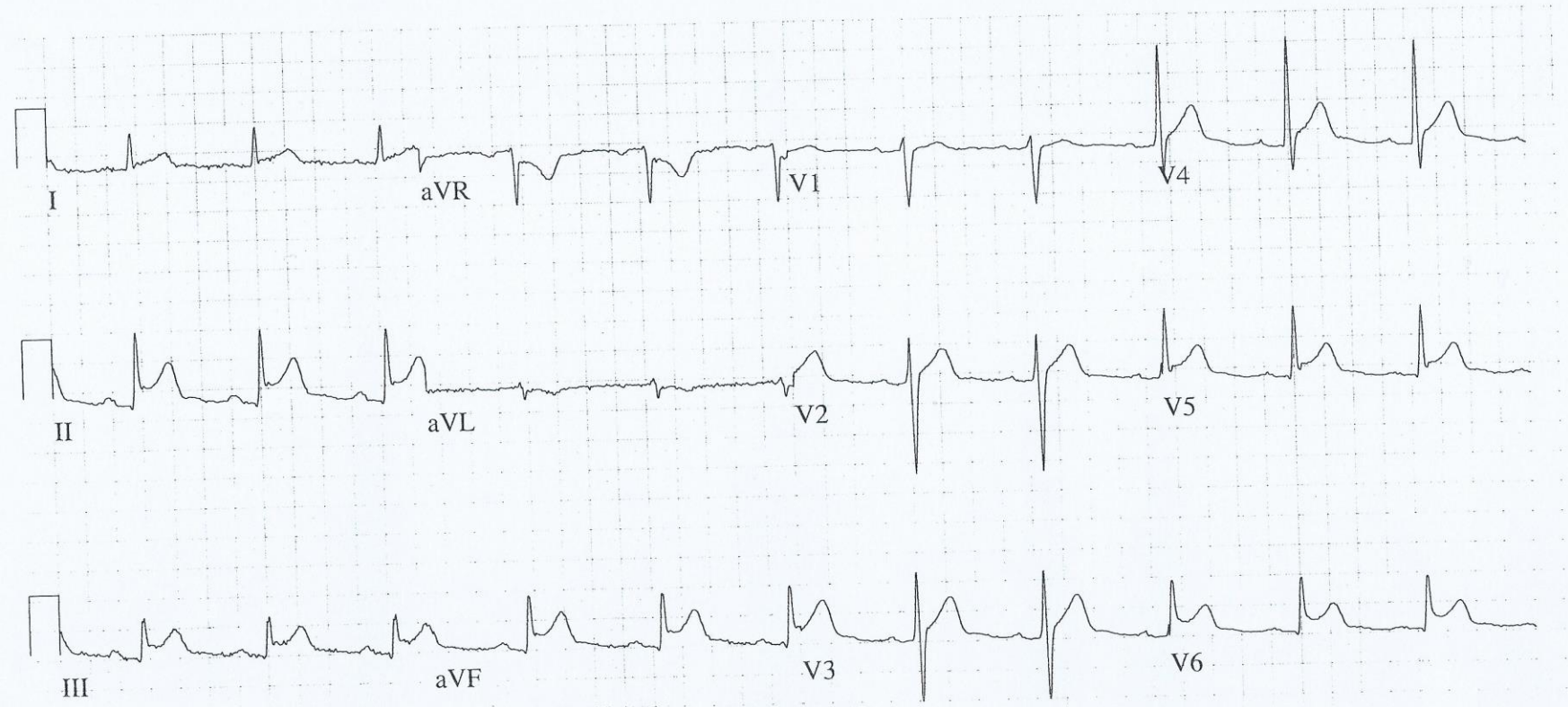
Post Thrombolysis.
CCP 5/10

3 YVONNE COURT
MAIDEN GULLY VIC,3551
Ph: 54496796 M: 0448064175

15/01/1958
57 years, 10 months
Male

Public MC: 31330049311

GP: Dr BABOVIC Avram
Bendigo Medical Centre - Bridge Street



25mm/s 10.0mm/mV 100H
GEMS Information Technologies

Name: , ID: 99999999 05 Dec 2015 05:40

11

55 years

Loc:5
Room: RES-3

Vent. rate	76	/min
PR interval	*	ms
QRS duration	100	ms
QT/QTc	364/409	ms
P-R-T axes	84 67 74	

*** Age and gender specific ECG analysis ***
Sinus tachycardia with 2nd degree AV block with 2:1 AV conduction
ST elevation, consider anterolateral injury or acute infarct
ST elevation, consider inferior injury or acute infarct
** ** ACUTE MI ** **
Abnormal ECG

[redacted]

3 YVONNE COURT
MAIDEN GULLY VIC,3551
Ph: 54496796 M: 0448064175

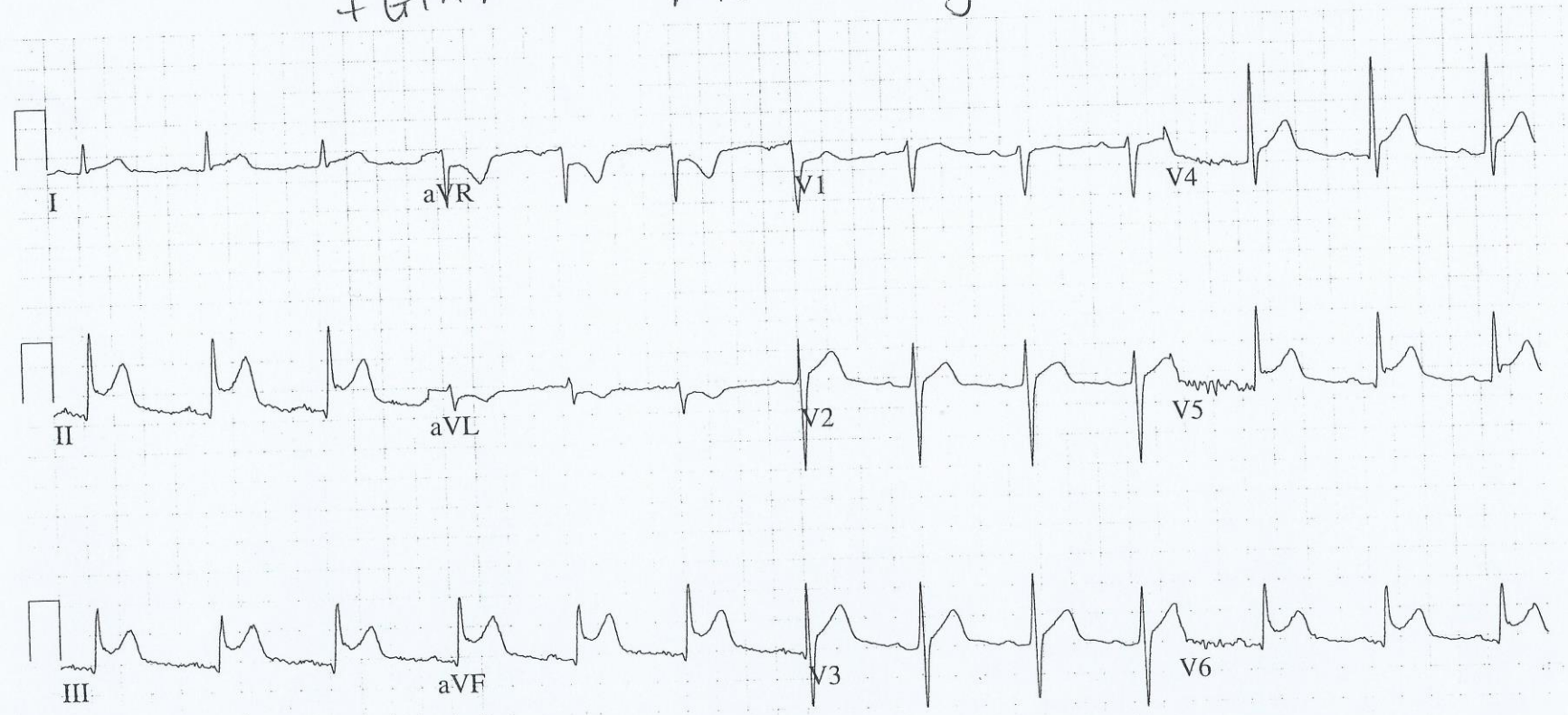
15/01/1958
57 years, 10 months
Male

Public MC: 31330049311

GP: Dr BABOVIC Avram
Bendigo Medical Centre - Bridge Street

T 20 min post thrombolysis + GTN patch

Post thrombolysis ↑↑ pain 10/10 pain + clammy



25mm/s 10.0mm/mV 100H
GEMS Information Technologies

13

55 years

Loc:5
Room: RES-3

Vent. rate	82	/min
PR interval	178	ms
QRS duration	96	ms
QT/QTc	364/425	ms
P-R-T axes	76 59 67	

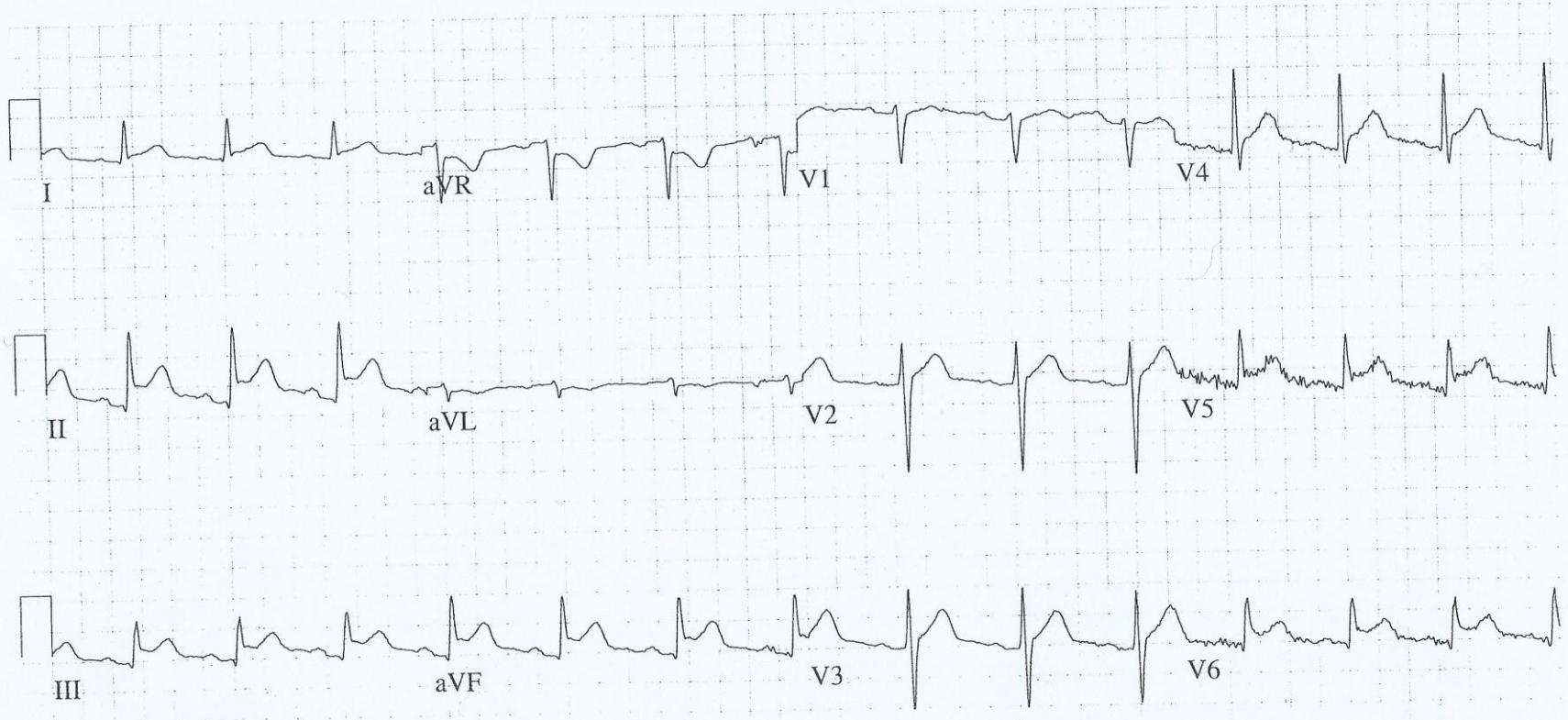
*** Age and gender specific ECG analysis ***
Normal sinus rhythm
ST elevation, consider inferolateral injury or acute infarct
** ** ACUTE MI ** **
Abnormal ECG

DOB: 15/01/1958
Date: Time:

Unconfirmed

Sign: .....

± 1hr post thrombolysis Painfree.
on GTN infusion



25mm/s 10.0mm/mV 100H
GEMS Information Technologies

Name: , ID: 99999999 05 Dec 2015 01:46

15

55 years

Loc:5
Room: RES-3

Vent. rate	83	/min
PR interval	176	ms
QRS duration	96	ms
QT/QTc	358/421	ms
P-R-T axes	71 54	65

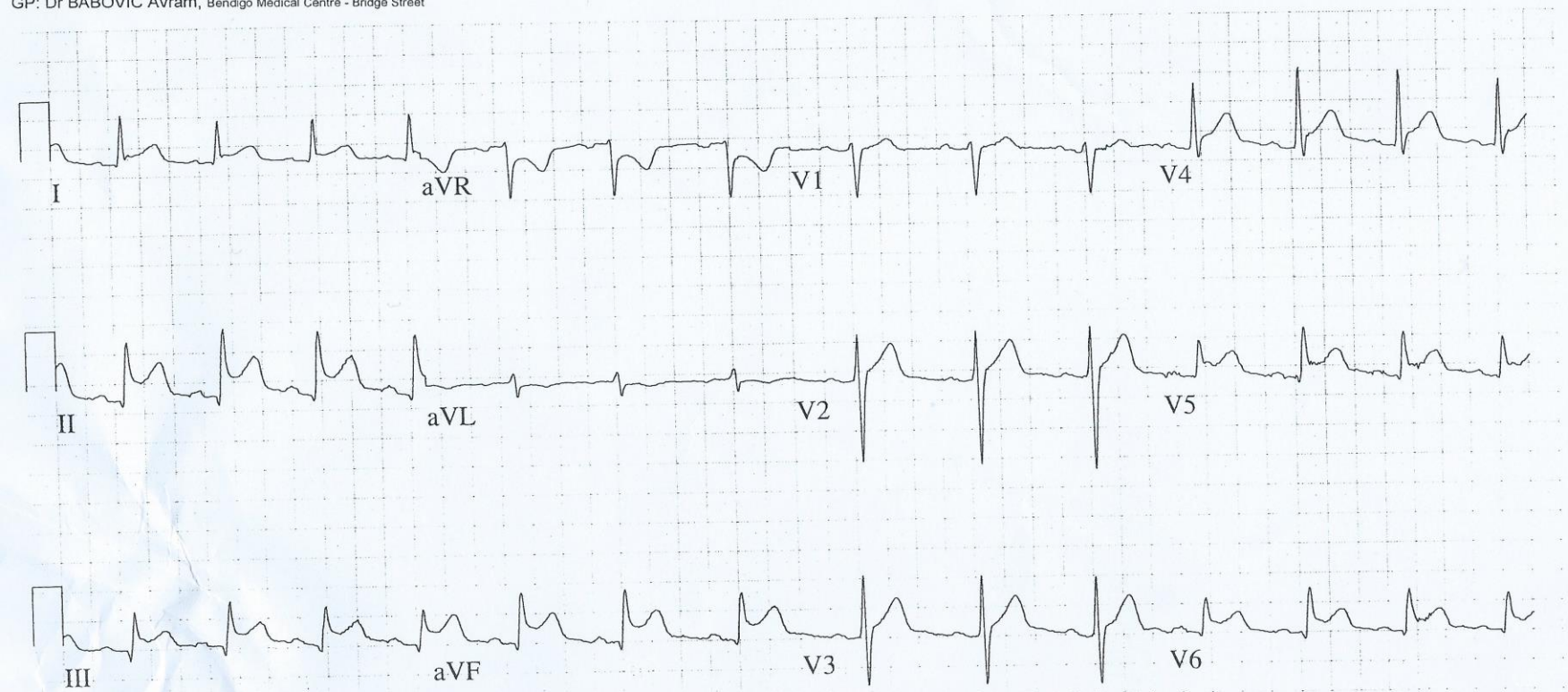
*** Age and gender specific ECG analysis ***
Sinus rhythm with sinus arrhythmia
ST elevation, consider inferolateral injury or acute infarct
** ** ACUTE MI ** **
Abnormal ECG

[redacted]
3 Yvonne Court
MAIDEN GULLY VIC, 3551
Ph: 54496796 M: 0448064175
Public - Eligible
Cardiology
Adm: Saturday 5th December 2015
GP: Dr BABOVIC Avram, Bendigo Medical Centre - Bridge Street

15/01/1958
Male 57 years, 10 months
Ward: EMERG
MC: 31330049311

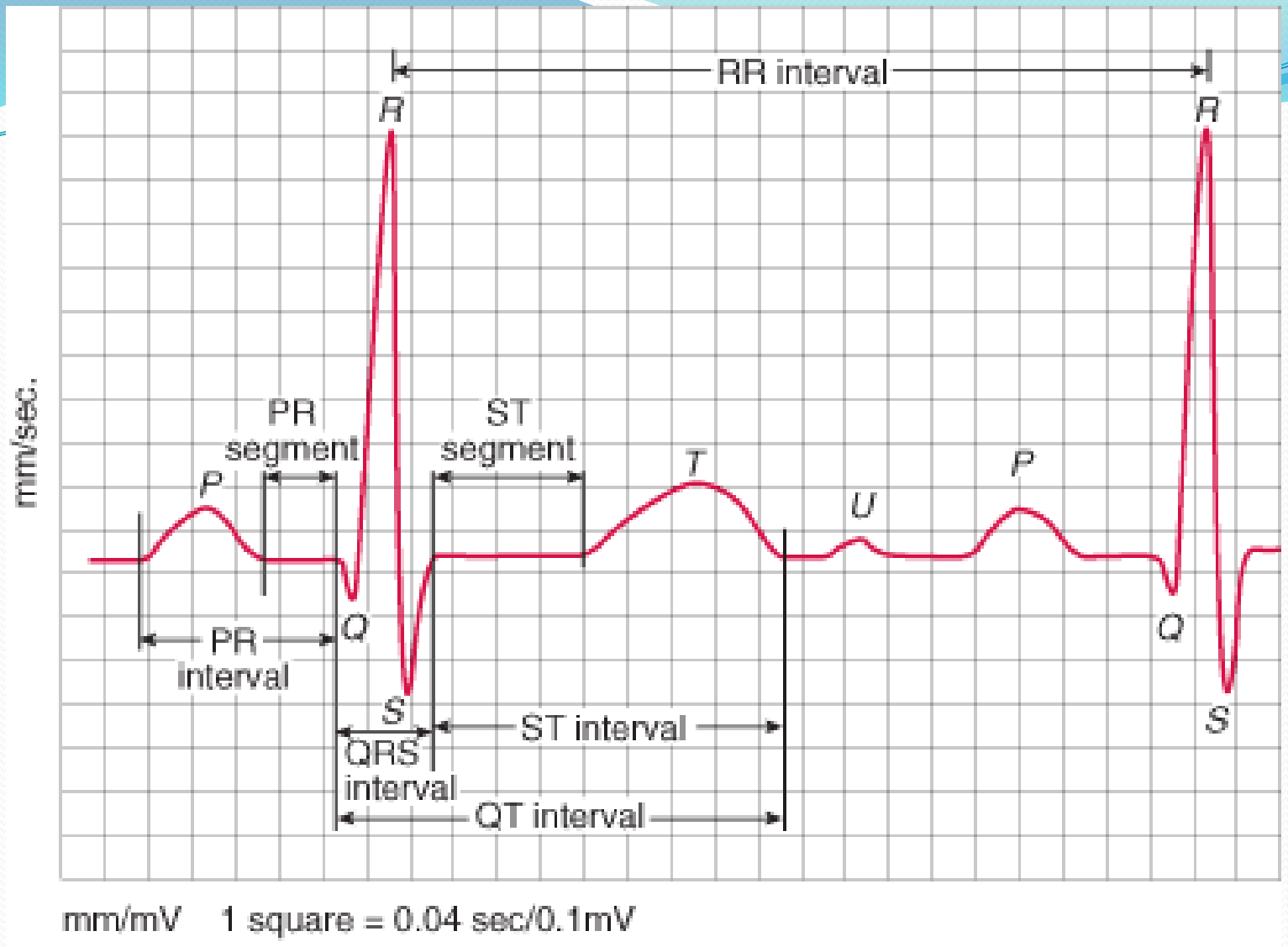
GTN infusion 1.5 mls/hr.
Heparin Infusion 1000 units/hr.
Chest discomfort on deep breathing 2/10

Unconfirmed



25mm/s 10.0mm/mV 100H
GEMS Information Technologies

Pericarditis vs STEMI



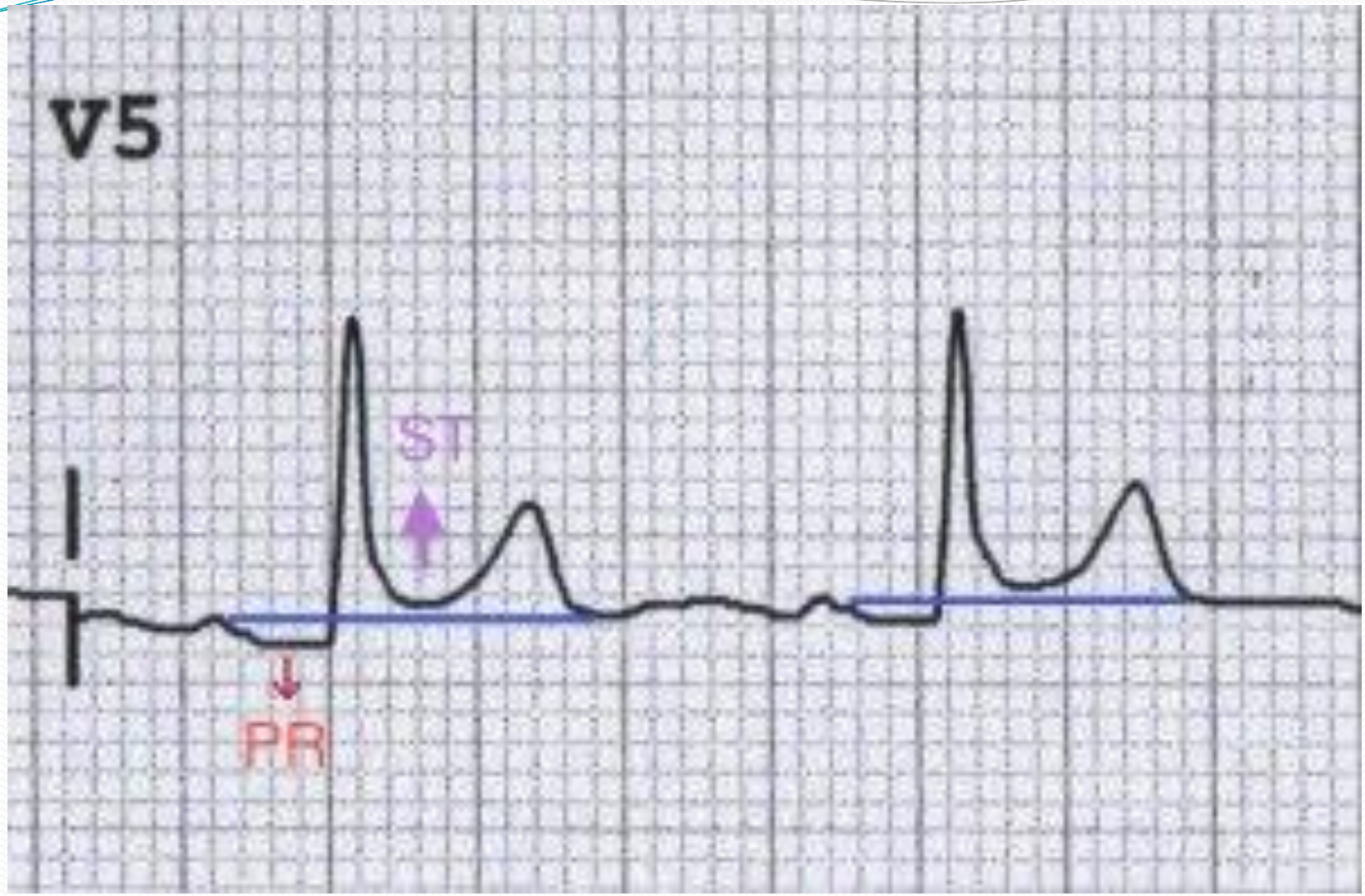
Its all about the ST segment, right?!

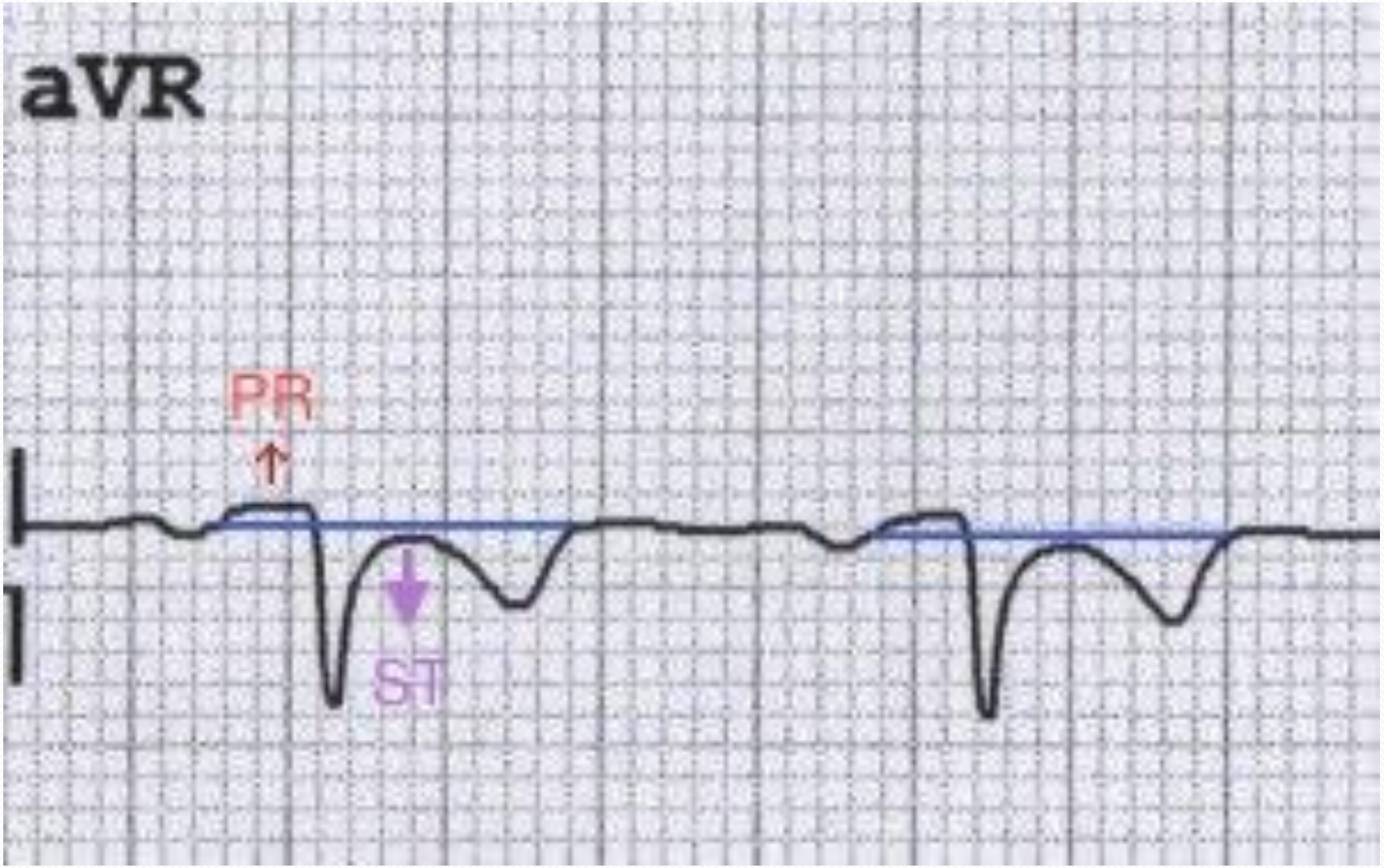
Yes and No!

Classic Teaching!

- Widespread concave ST elevation and PR depression throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6)

- Reciprocal ST depression and PR elevation in lead aVR (± V1)


V5
ST
PR

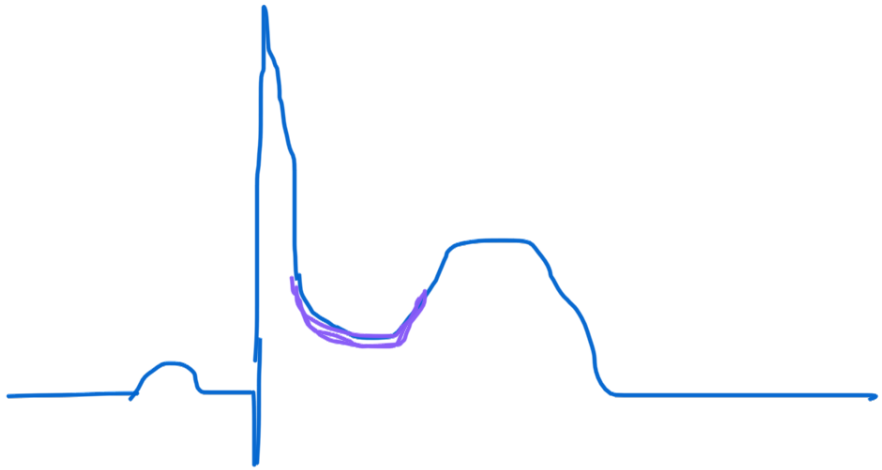

aVR
PR
ST

- That's not all!
- Classic teaching at best is….
- Over simplified!

What is it that we really want to know when we see STE?

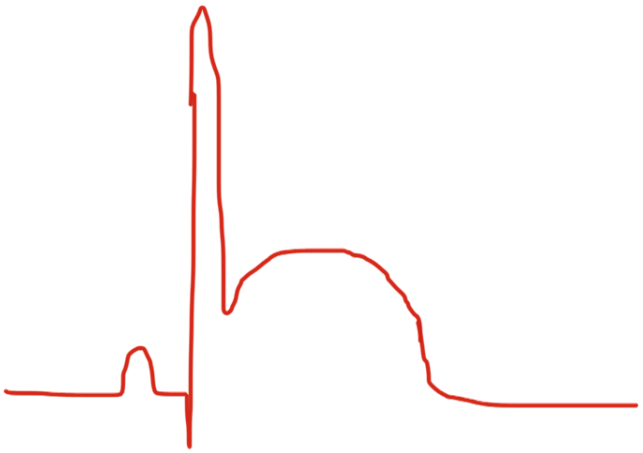
Factors that strongly favour STEMI:

- Reciprocal ST depression in any leads (except aVR & V1) – *It's a STEMI*
- STE in lead III > STE in lead II – *It's a STEMI*
- Horizontal or convex upward ST-segment morphology – *It's a STEMI*



WINNING!!

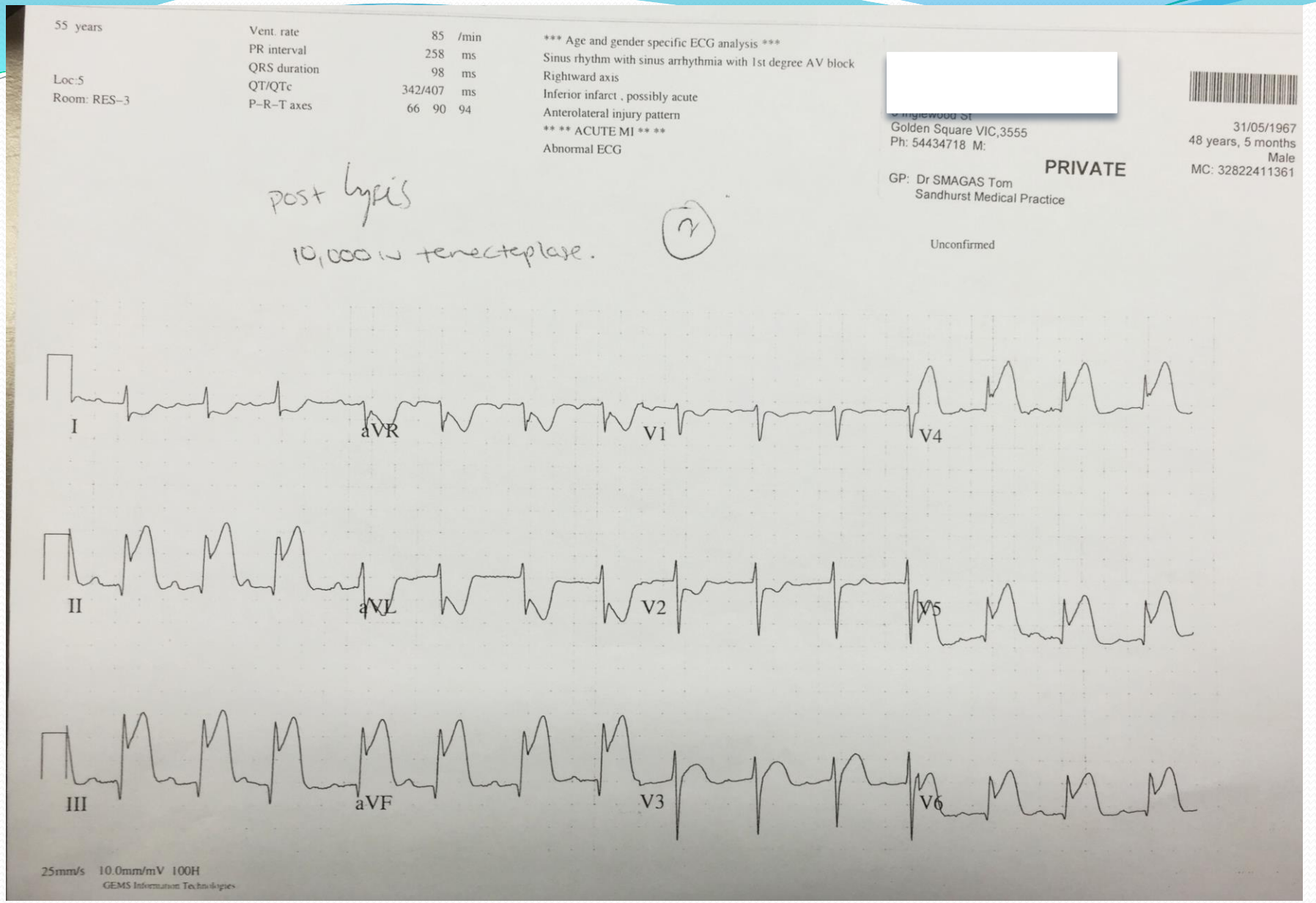
PERICARDITIS



NOT HAPPY

STEMI


"CHECKMARK"
"NIKE"!


55 years
Loc:5
Room: RES-3
Vent. rate 85 /min
PR interval 258 ms
QRS duration 98 ms
QT/QTc 342/407 ms
P-R-T axes 66 90 94
*** Age and gender specific ECG analysis ***
Sinus rhythm with sinus arrhythmia with 1st degree AV block
Rightward axis
Inferior infarct , possibly acute
Anterolateral injury pattern
** ** ACUTE MI ** **
Abnormal ECG
Golden Square VIC,3555
Ph: 54434718 M:
PRIVATE
GP: Dr SMAGAS Tom
Sandhurst Medical Practice
Unconfirmed
31/05/1967
48 years, 5 months
Male
MC: 32822411361
post lysis
10,000 iv tenecteplase.
?
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s 10.0mm/mV 100H
GEMS Information Technologies

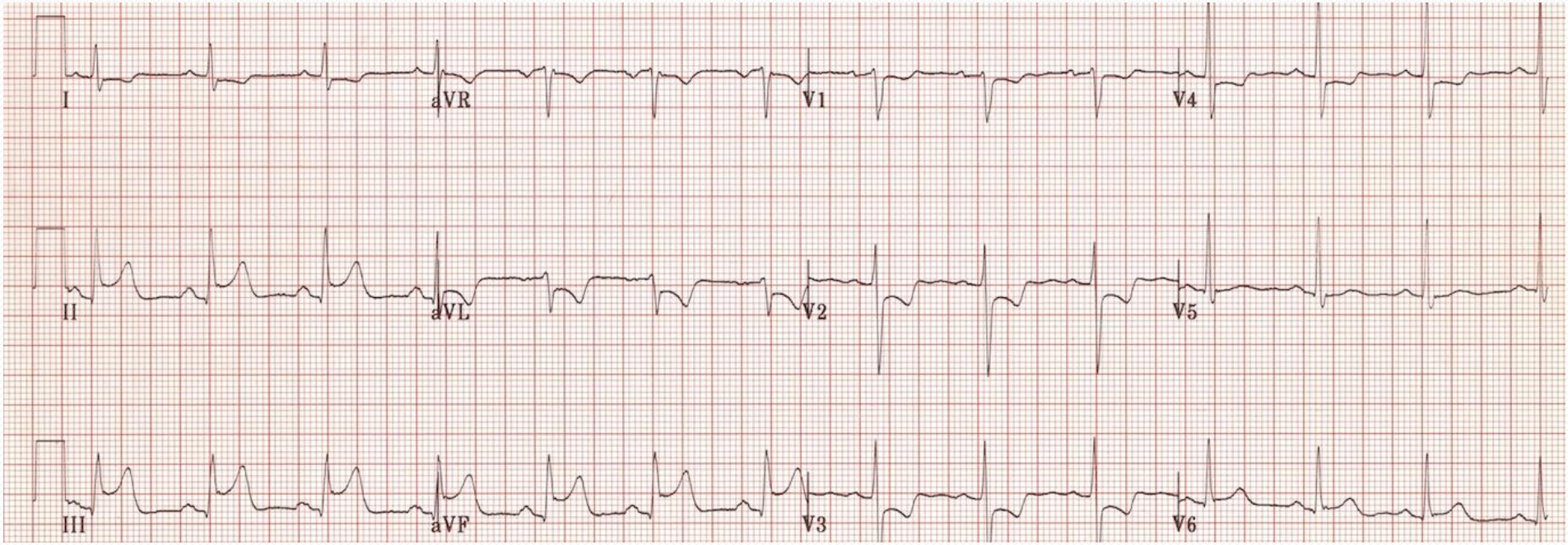
- If the patient has new Q waves
- Or
- If he develops new Q waves

- – *It's a STEMI*

Can…

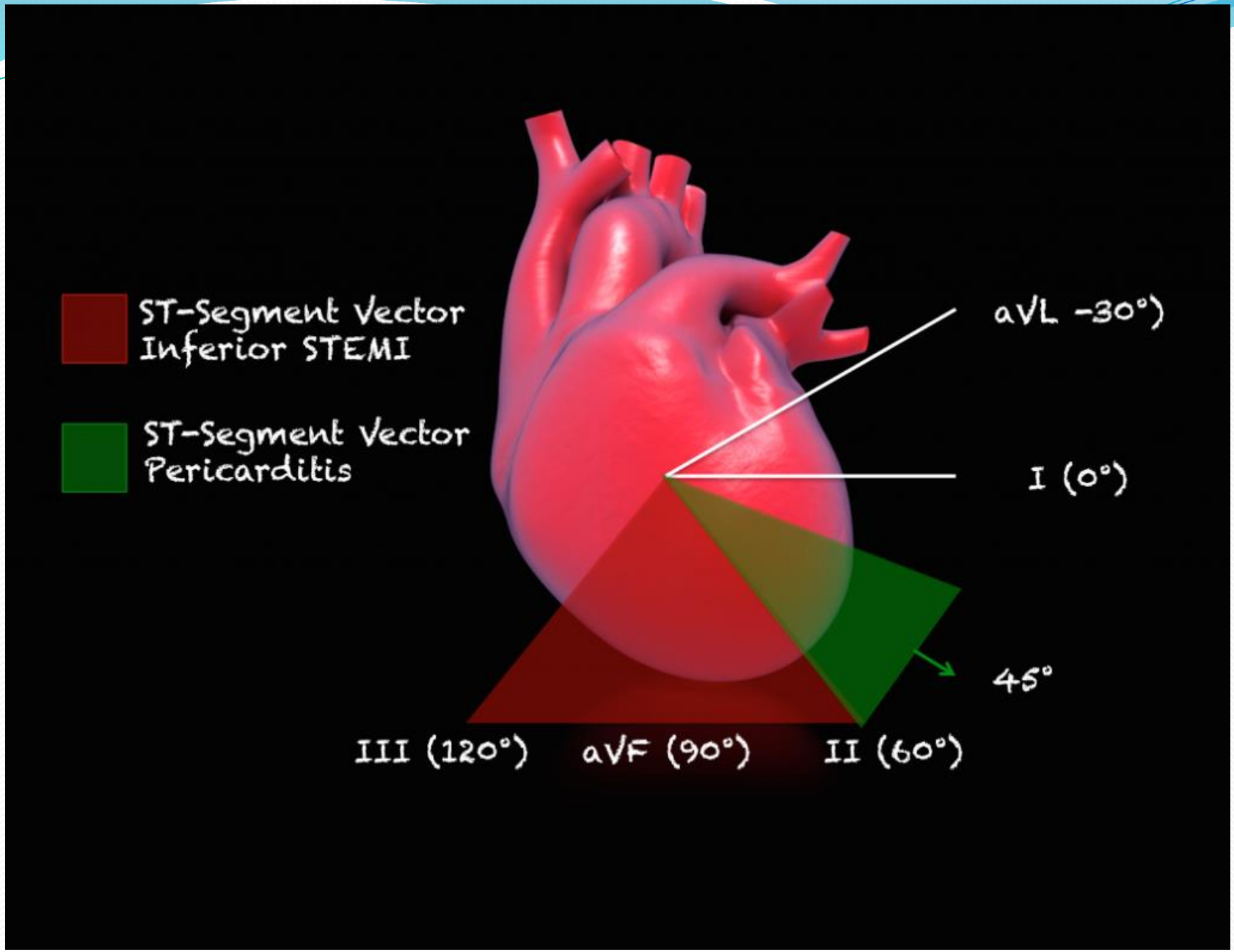
- Pericarditis can cause localised ST elevation?
- *YES! – but there should be no reciprocal ST depression (except in AVR and V1)*

- STEMI, like pericarditis, cause concave up ST elevation?
- *YES! – but will have 'in your face' reciprocal changes!!*

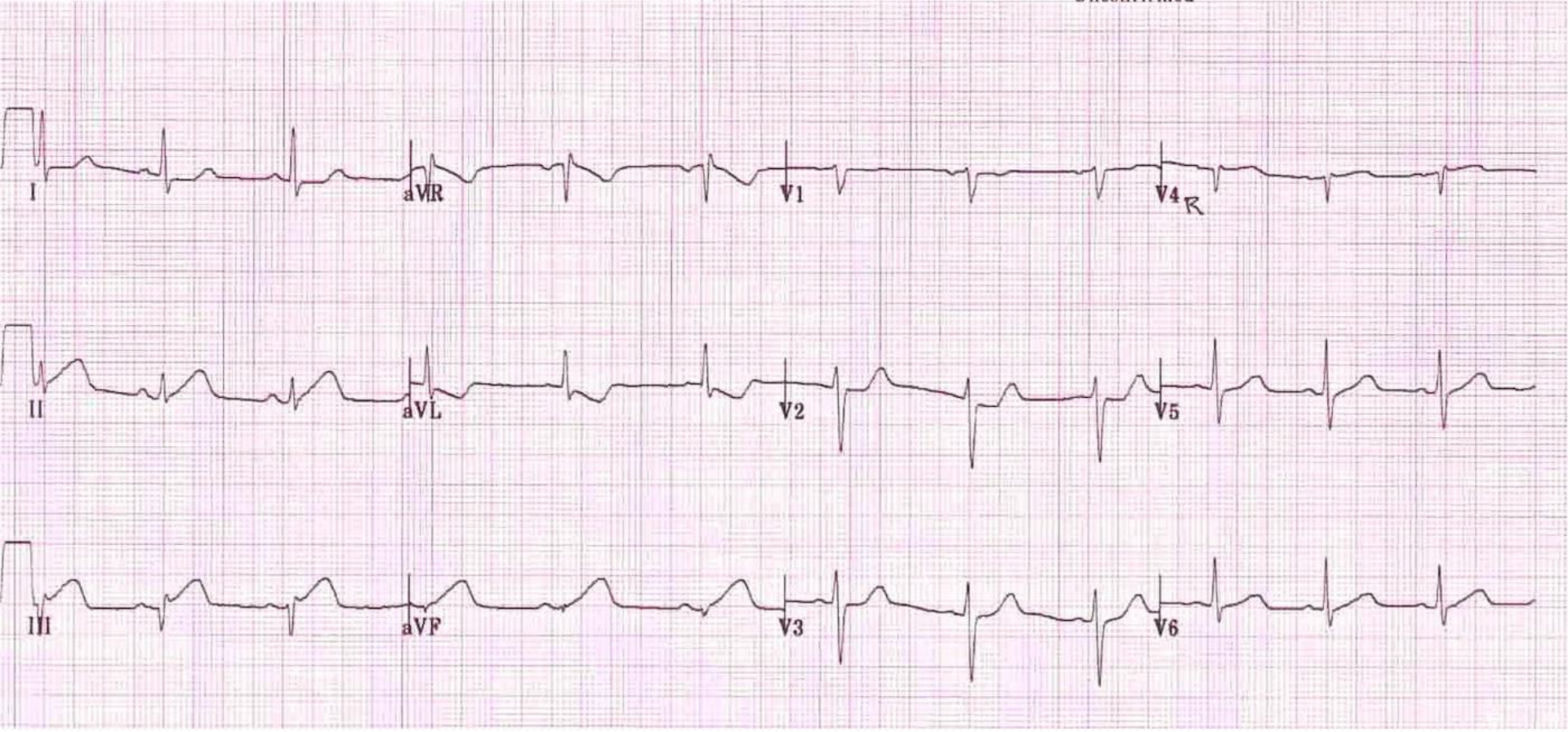


STEMI, like pericarditis, can also cause concave up ST elevation

Why STE in III > II ?


ST-Segment Vector
Inferior STEMI
ST-Segment Vector
Pericarditis
aVL -30°)
I (0°)
45°
III (120°)
aVF (90°)
II (60°)

What’s this?



- *Hyperacute T waves in II, III and aVF with relative loss of R wave height*
- *Early ST elevation and Q-wave formation in lead III*
- *Reciprocal ST depression and T wave inversion in aVL*
- *ST elevation in lead III > lead II*

"If there is inferior ST elevation, but ***no reciprocal ST depression in aVL****, you should be* ***skeptical*** *of the diagnosis of STEMI -* ***even if there is ST elevation in V5 and V6****"*

- You wonder if he is actually talking about our ECG!!

Name: , ID: 99999999 05 Dec 2015 01:46

15

55 years

Loc:5
Room: RES-3

Vent. rate	83	/min
PR interval	176	ms
QRS duration	96	ms
QT/QTc	358/421	ms
P-R-T axes	71 54	65

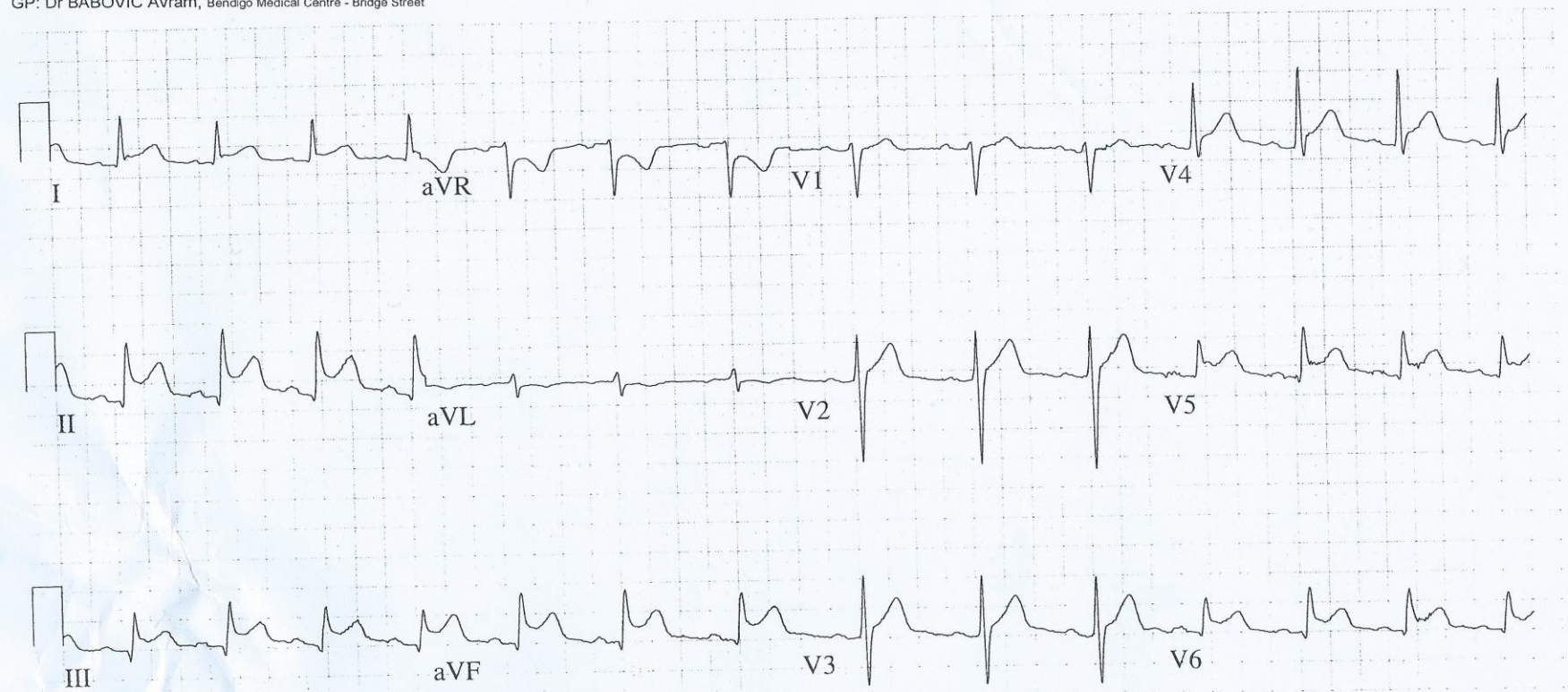
*** Age and gender specific ECG analysis ***
Sinus rhythm with sinus arrhythmia
ST elevation, consider inferolateral injury or acute infarct
** ** ACUTE MI ** **
Abnormal ECG

[redacted]
3 Yvonne Court
MAIDEN GULLY VIC, 3551
Ph: 54496796 M: 0448064175
Public - Eligible
Cardiology
Adm: Saturday 5th December 2015
GP: Dr BABOVIC Avram, Bendigo Medical Centre - Bridge Street

15/01/1958
Male 57 years, 10 months
Ward: EMERG
MC: 31330049311

GTN infusion 1.5 mls/hr.
Heparin Infusion 1000 units/hr.
Chest discomfort on deep breathing 2/10

Unconfirmed



25mm/s 10.0mm/mV 100H
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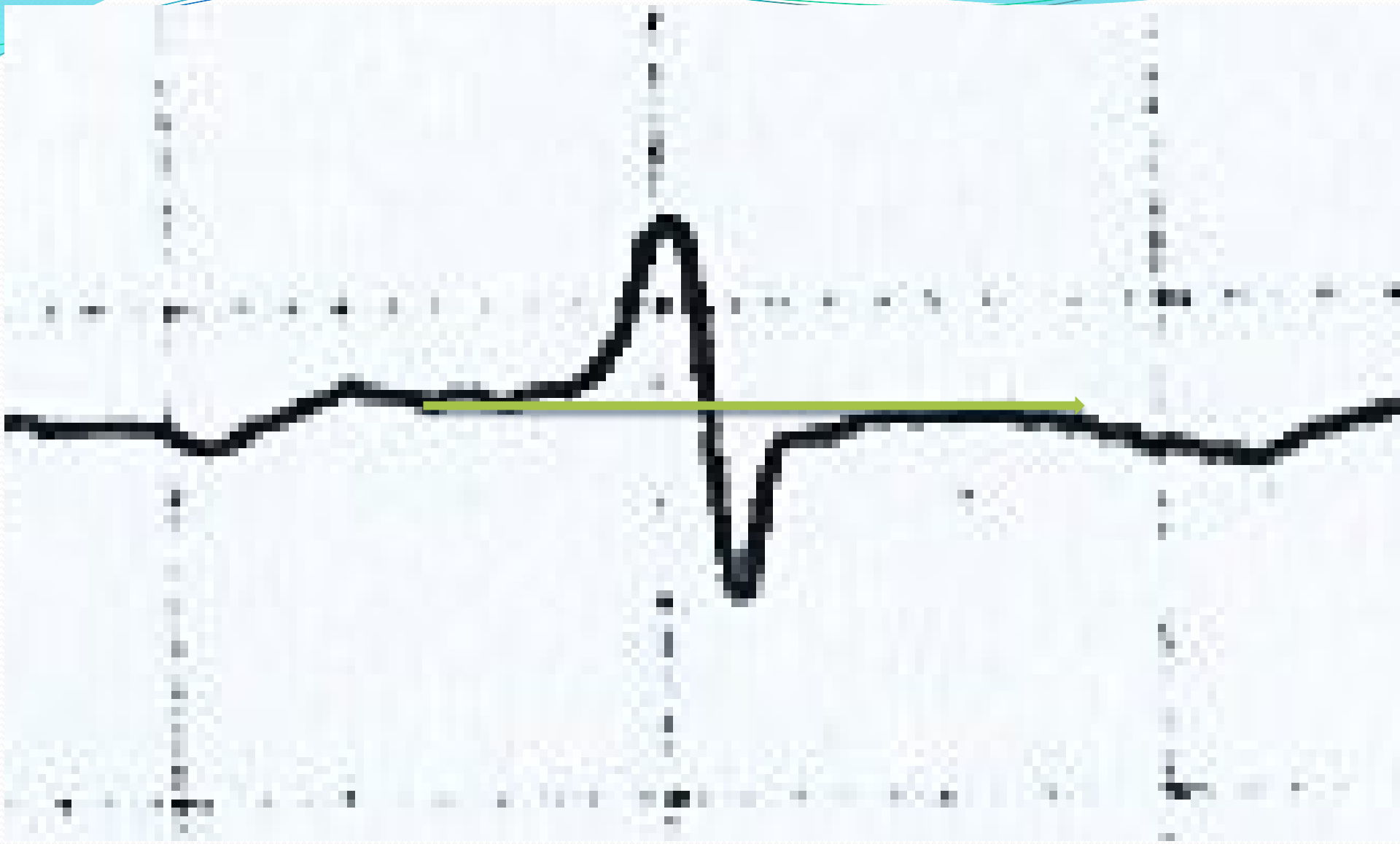
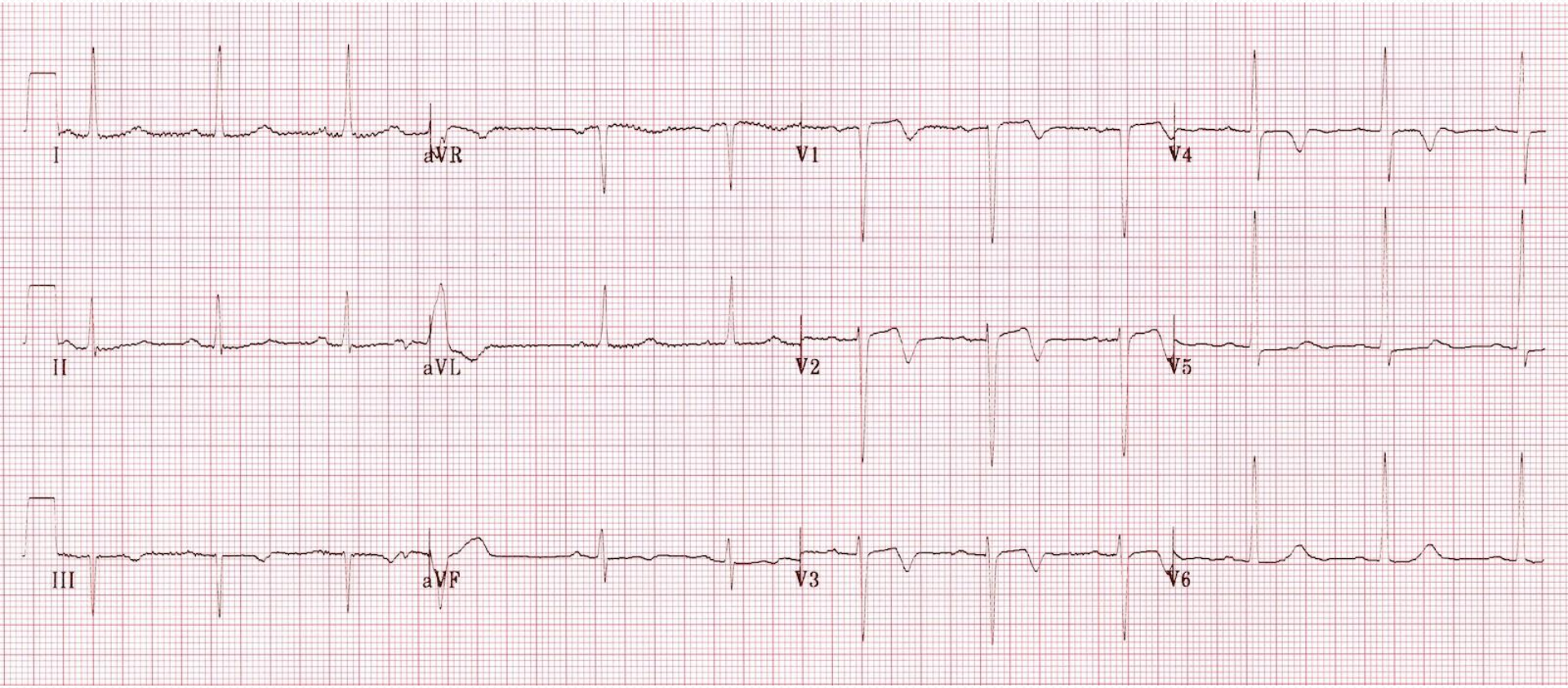
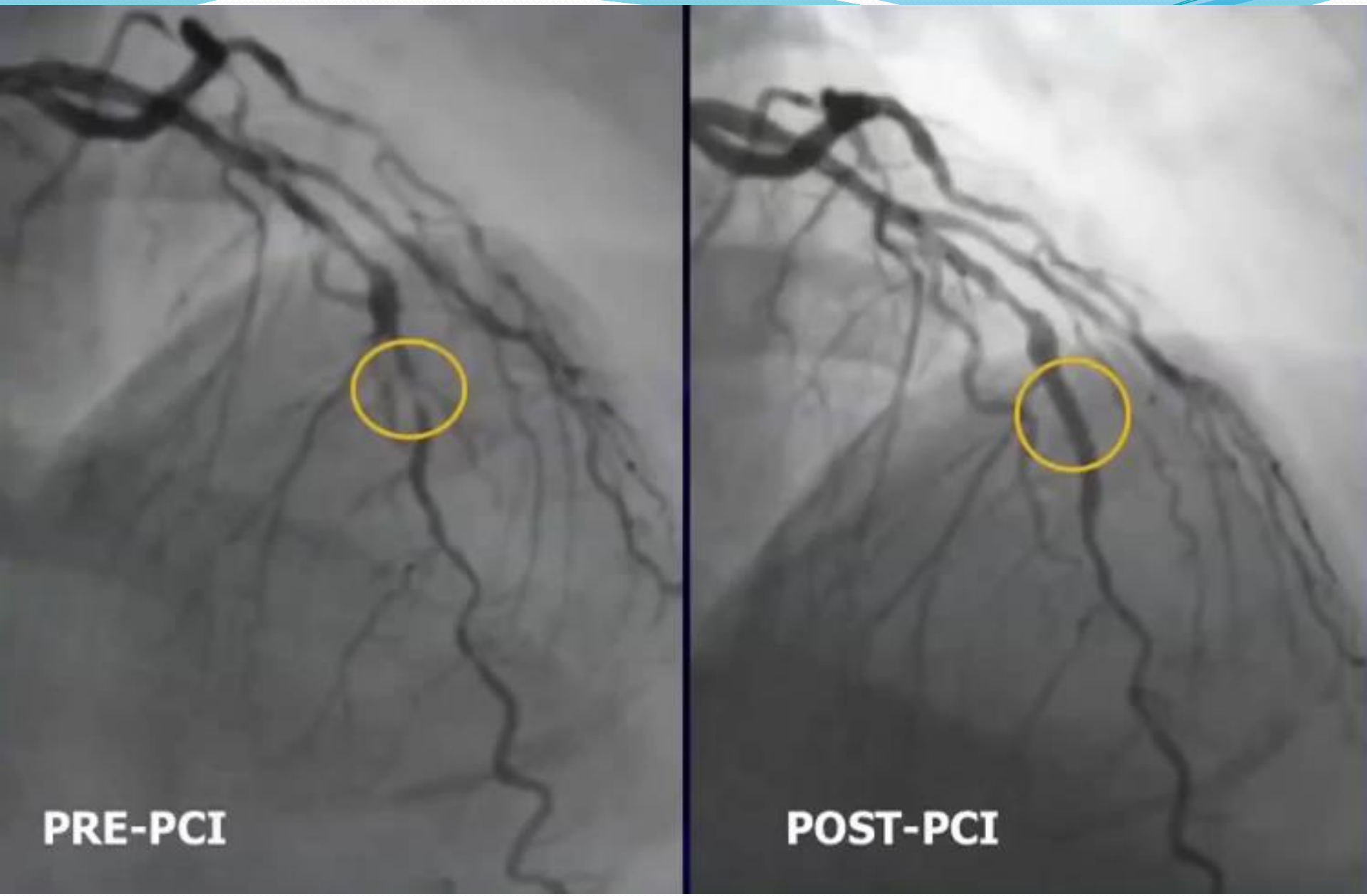
Relook

- Is there:
  - Reciprocal ST depression in any leads (except aVR & V1) ?
  - STE in lead III > STE in lead II ?
  - Horizontal or convex upward ST-segment morphology ?
  - Any new Q waves?



- 54yo male
- History of chest pain for 20mins – resolved with antacids
- Now pain free and in your office
- GORD right?


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


PRE-PCI
POST-PCI

Wellens' Syndrome

- Pattern of deeply inverted or biphasic T waves in V2-3
- Highly specific for a critical stenosis of LAD
- Patients may be pain free at the time of ECG
- Have normal cardiac enzymes
- Extremely high risk for extensive anterior wall MI within the next few days to weeks.

- Due to the critical LAD stenosis, these patients:
  - require invasive therapy,
  - do poorly with medical management

- *May suffer MI or cardiac arrest if stress tested!*

Absolute contraindication

Diagnostic criteria

- Deeply-inverted or biphasic T waves in V2-3 (may extend to V1-6)
- Isoelectric or minimally-elevated ST segment (< 1mm)
- No precordial Q waves

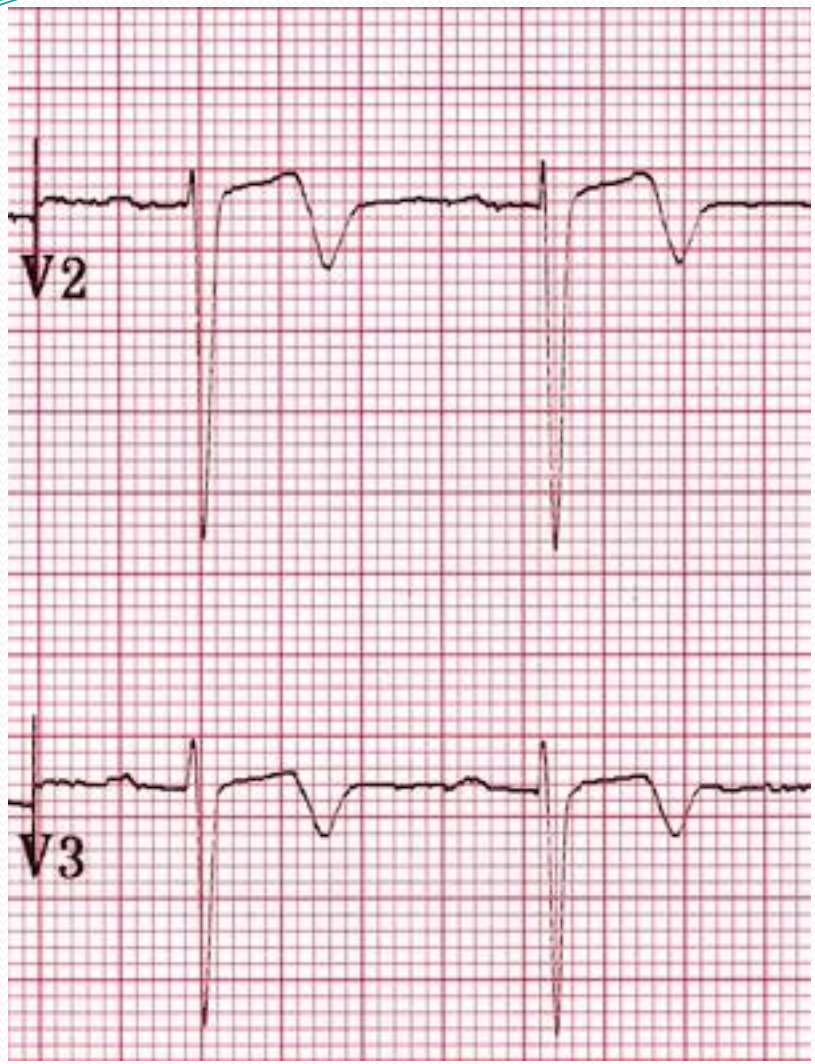
- Recent history of angina
- ECG pattern present in pain-free state

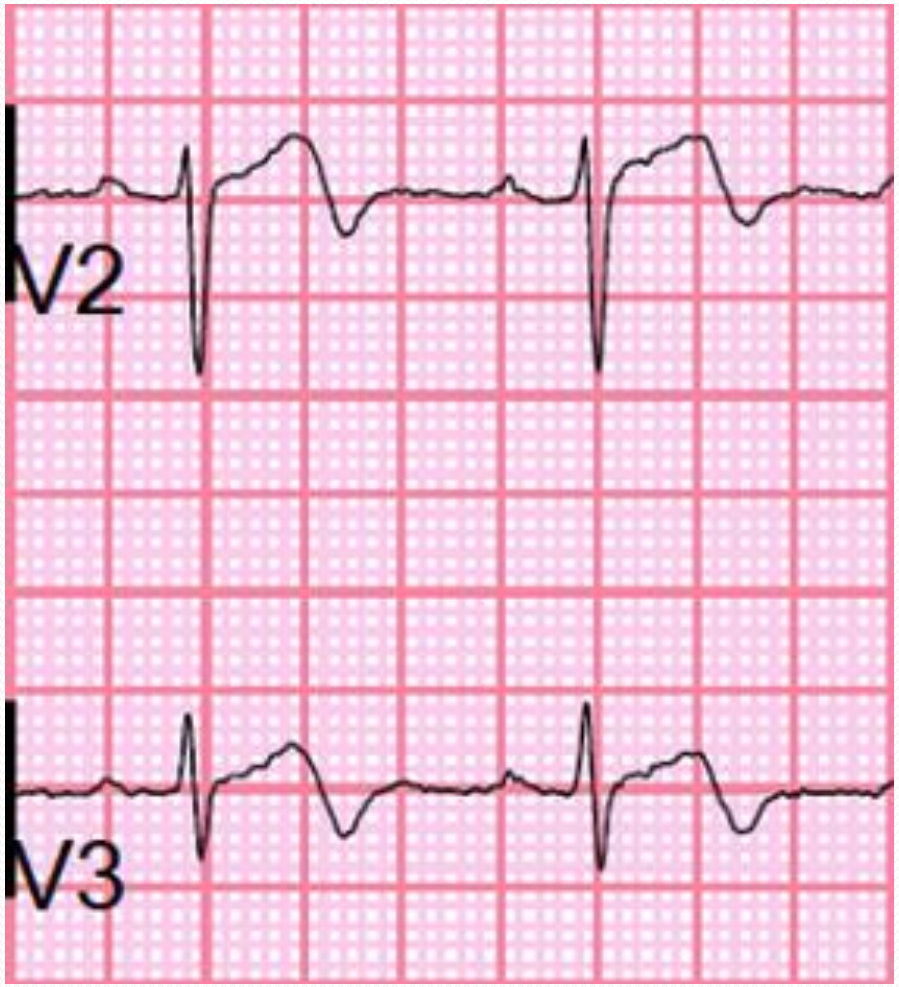
- Normal or slightly elevated serum cardiac marker

Two patterns of T-wave abnormality:

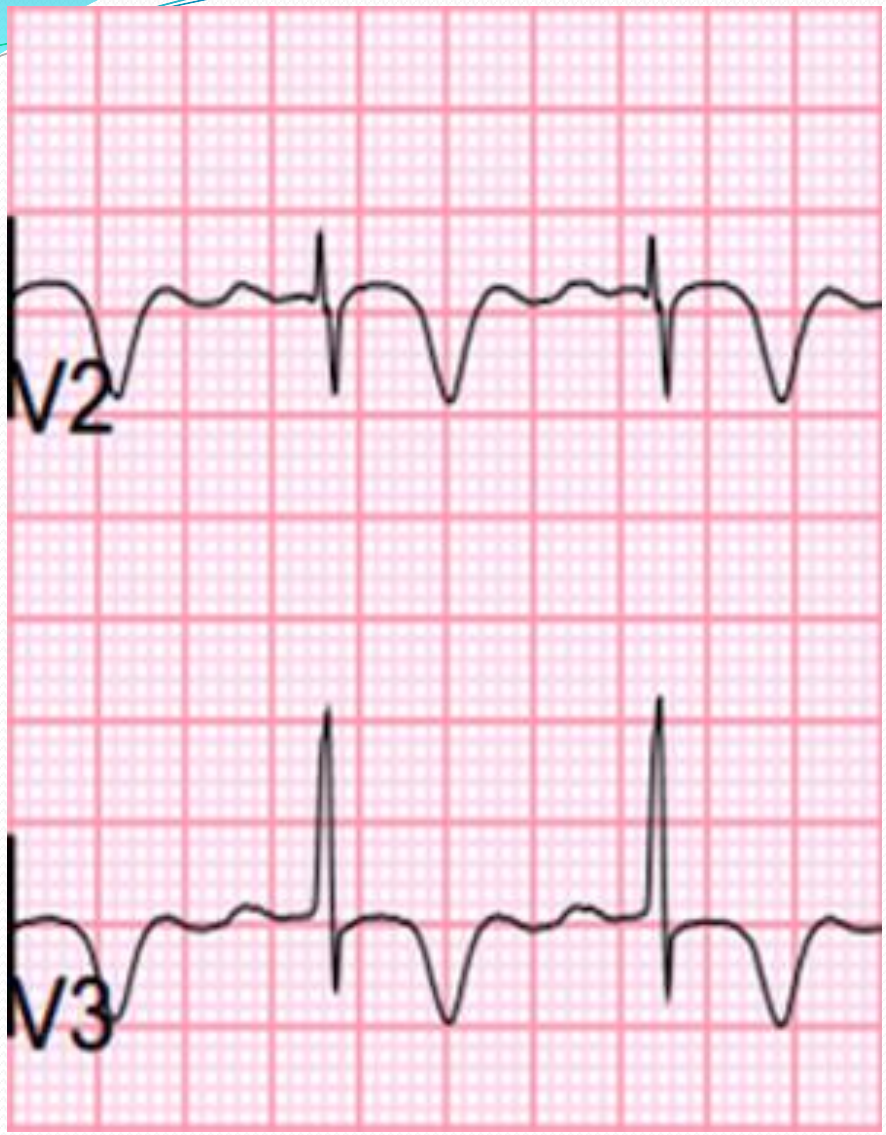
- *Type A* = Biphasic, with initial positivity & terminal negativity (25% of cases)
- *Type B* = Deeply and symmetrically inverted (75% of cases)

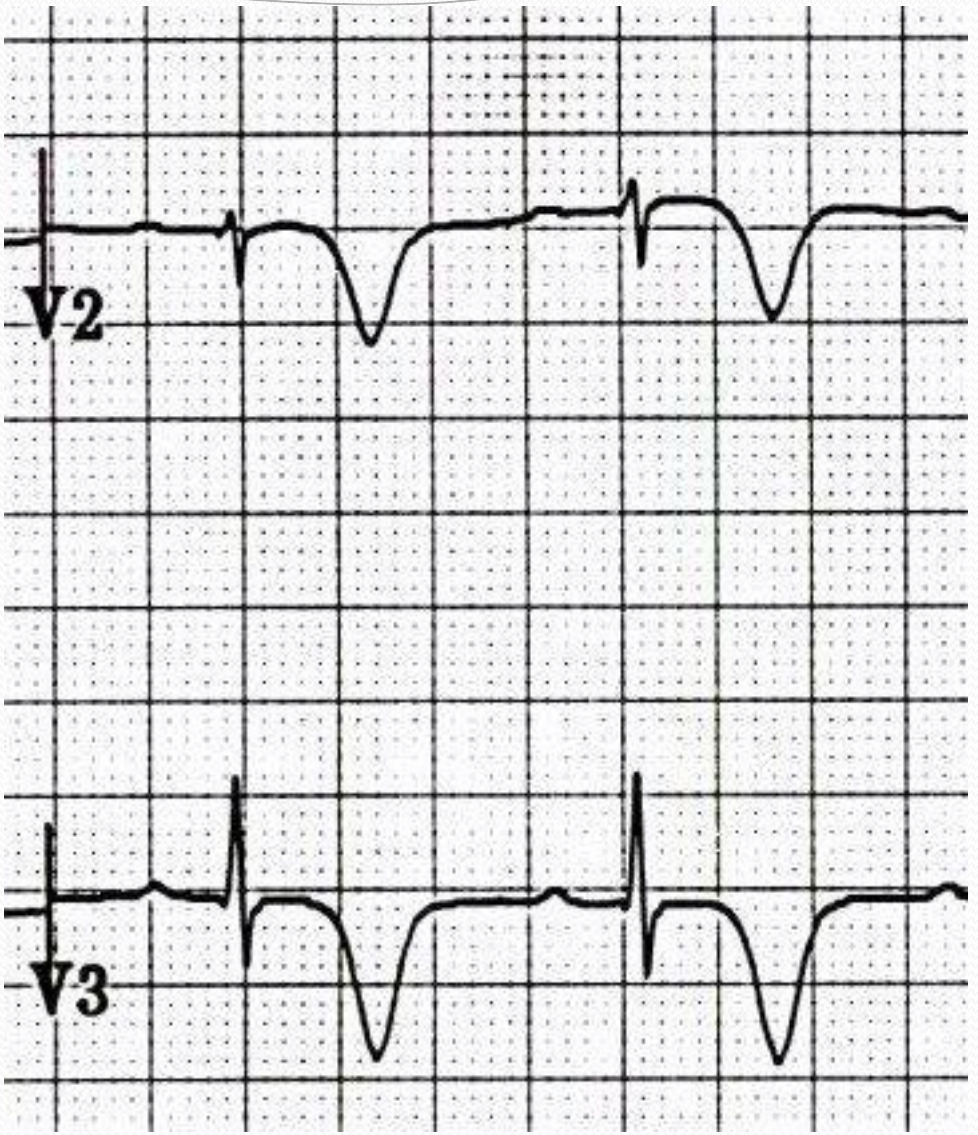
Computer often calls it non-specific T-wave abnormality



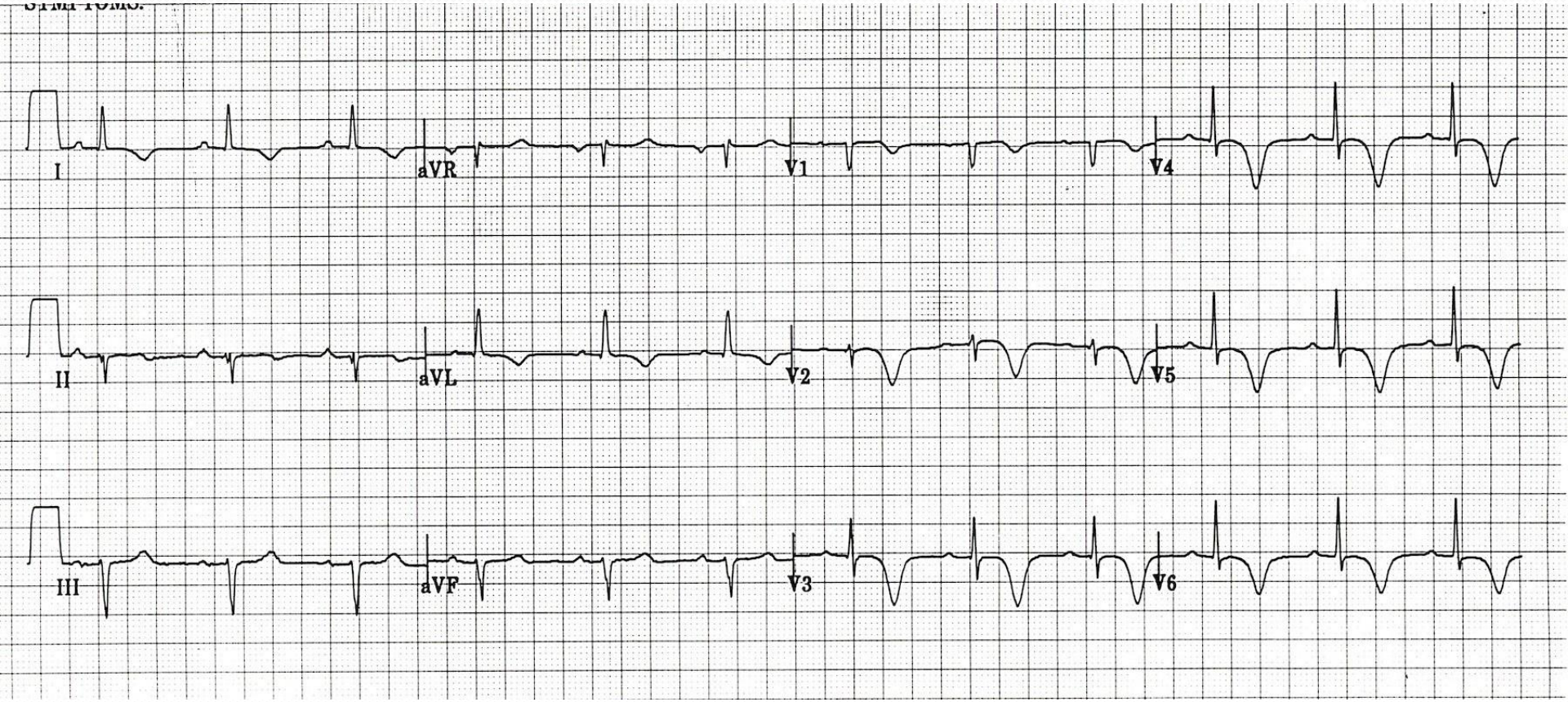


Biphasic T Waves (Type A)

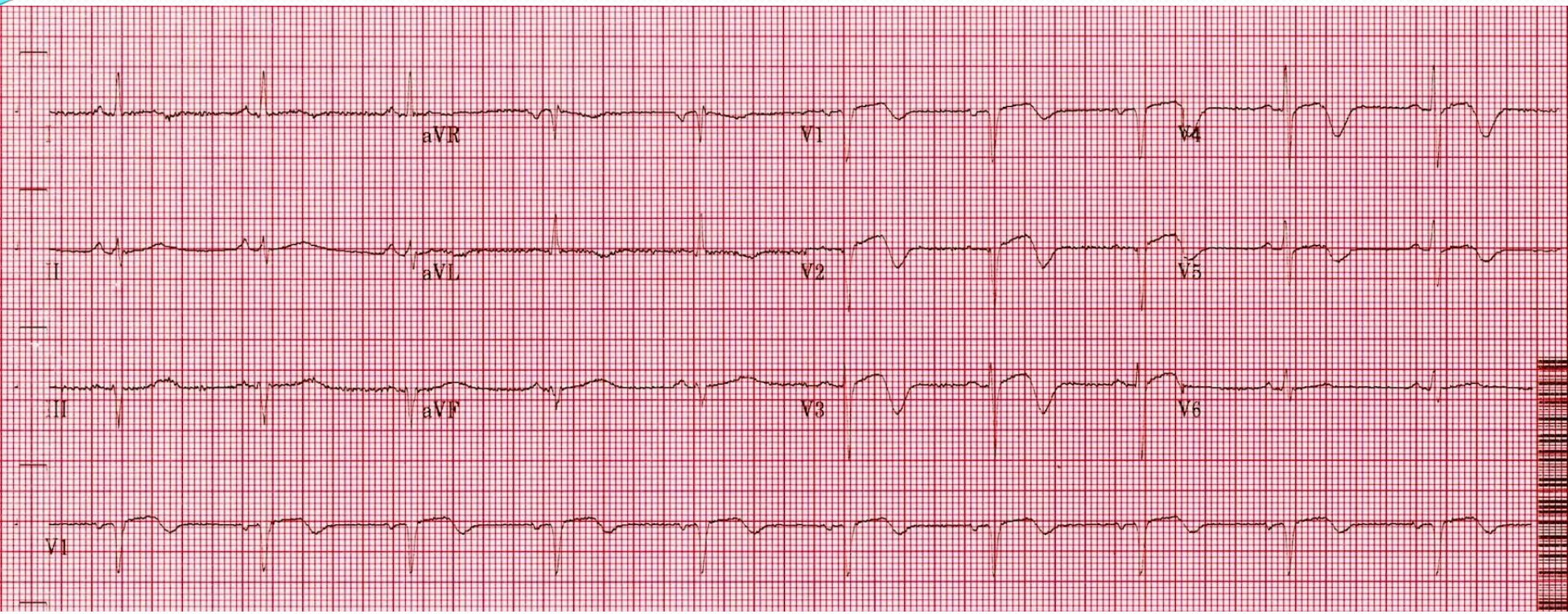




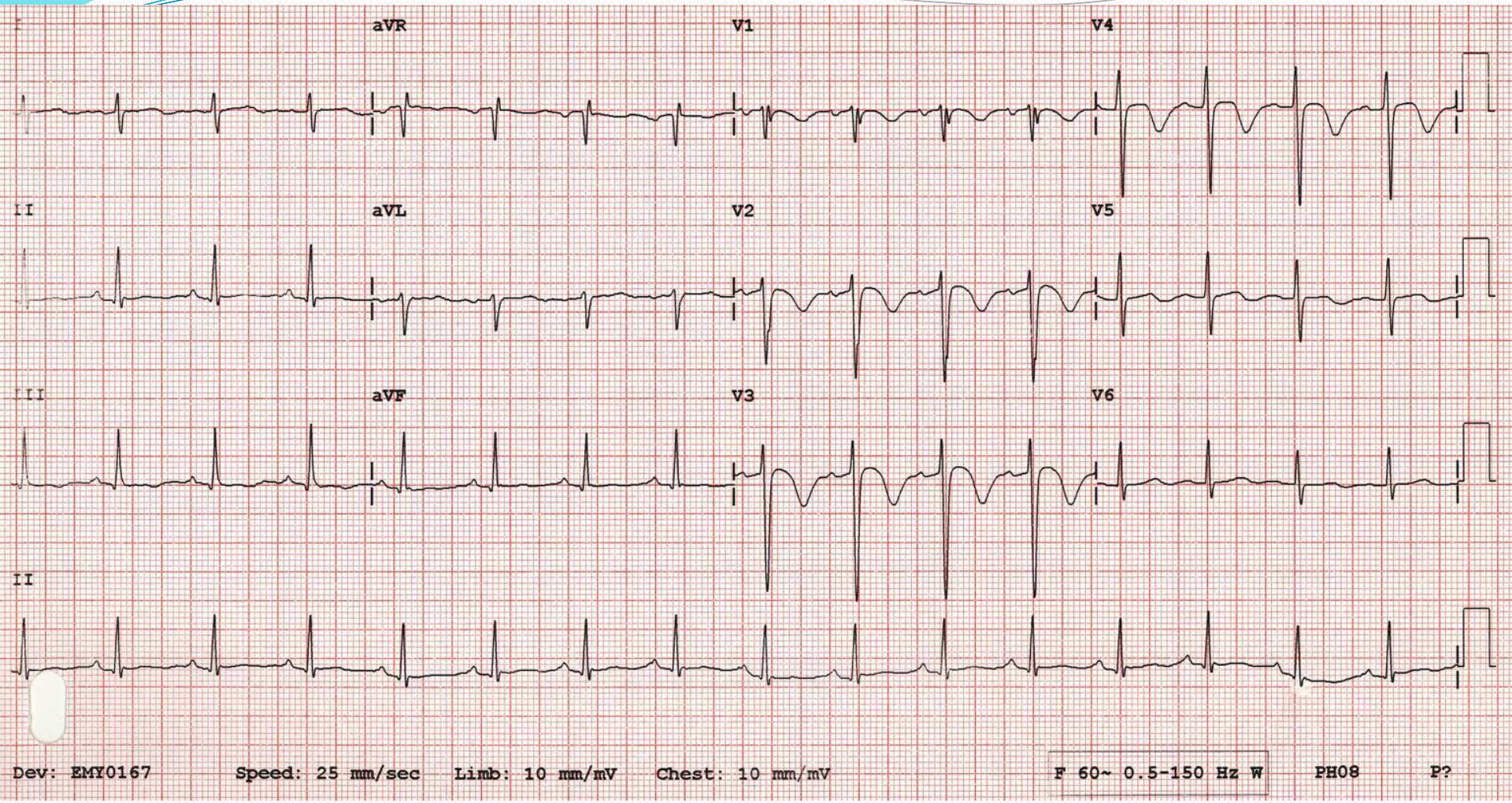
Deeply Inverted T Waves (Type B)



There are deep, symmetrical T wave inversions throughout the anterolateral leads (V1-6, I, aVL)



- Biphasic T waves with minimal ST elevation in V1-5, consistent with Wellens' syndrome.
- The patient had experienced ischaemic chest pain immediately prior to arrival in hospital and was pain free at the time the ECG was taken.

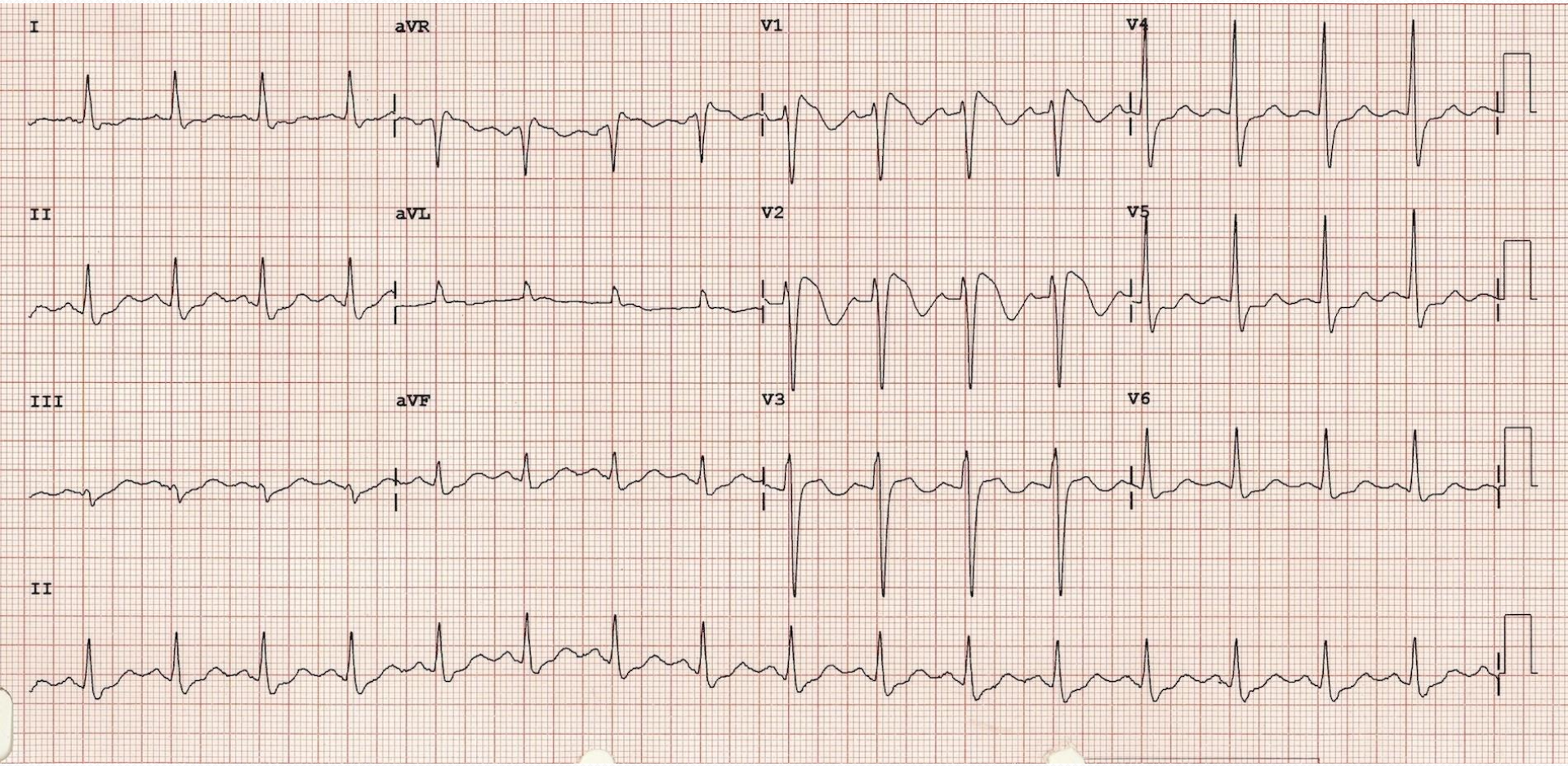


Is this Wellens?

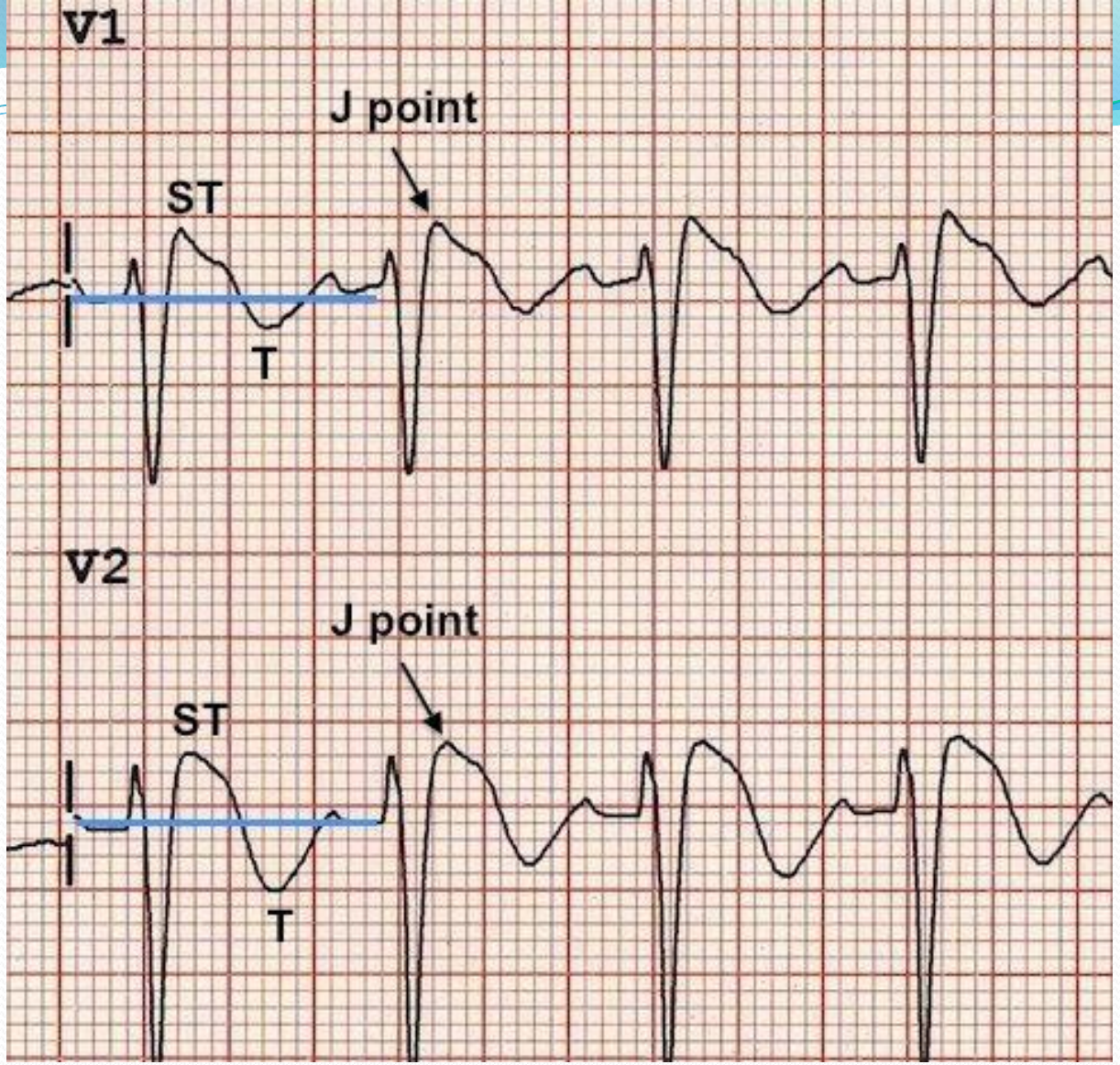
Take home Points

- - Get serial ECGs
- - Don't rely on your computer interpretations
- - Beware Wellens' waves!
- - No exercise stress testing for Wellens'!

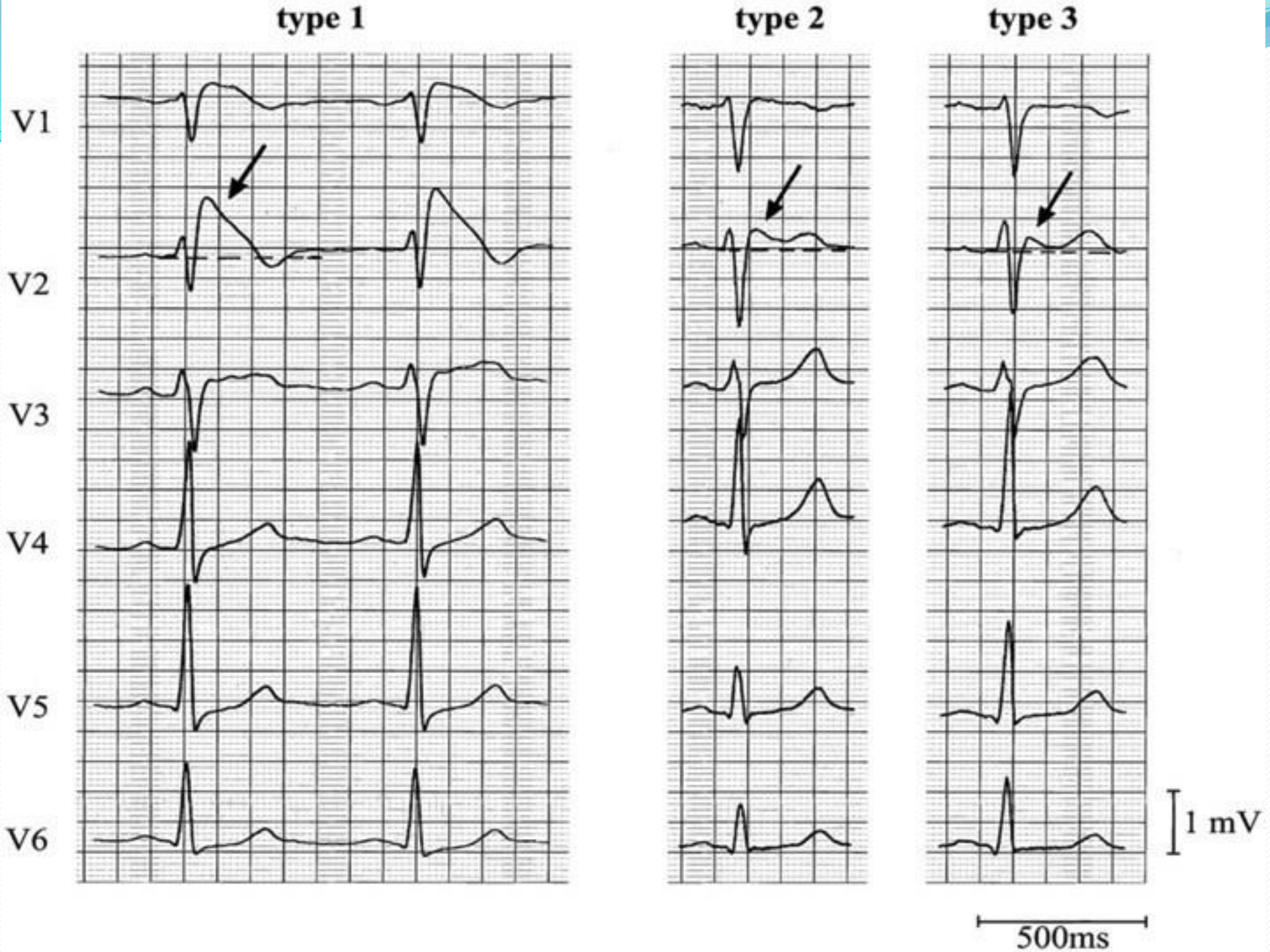
- 40yo female with syncope


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

Brugada Syndrome



- RBBB-like pattern with secondary R' wave following the QRS complex.
- ST elevation at the J point > 2mm with a "coved"
- T wave inversion


type 1
type 2
type 3
V1
V2
V3
V4
V5
V6
1 mV
500ms

Diagnosis

ECG plus one of the following:

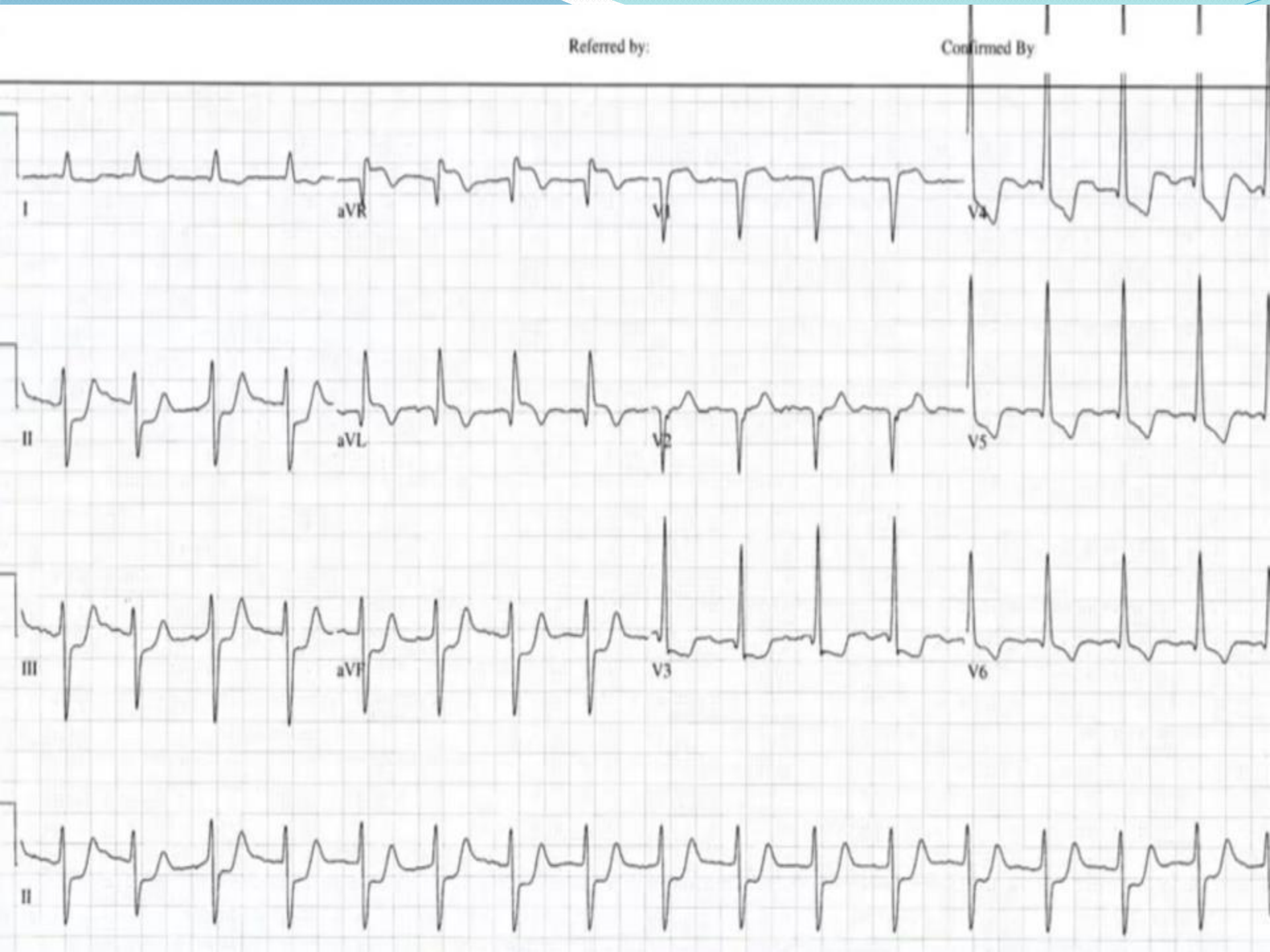
- Documented VF or VT
- Family history of SCD at <45 years old
- Coved-type ECGs in family members
- Syncope
- Nocturnal agonal respiration.

Only proven therapy is ICD

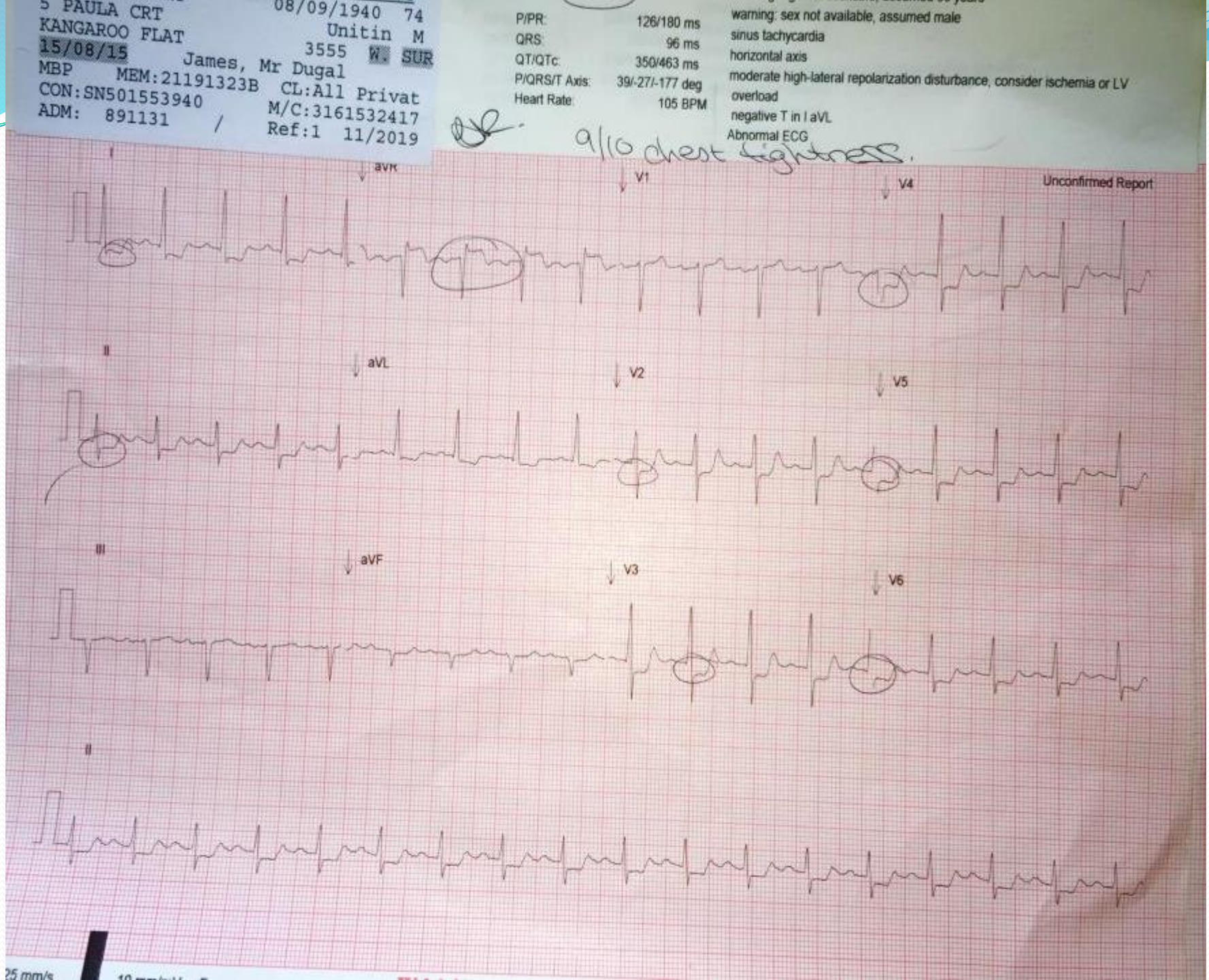
Take Home points

- Consider Brugada syndrome in any patient presenting after syncope
- ECG: (I)RBBB + STE in V1 - V2
  - Coved STE is most concerning
- Discuss/ refer to electrophysiologist

- 75yo male with HTN, DM-2 complains of angina
- What does this ECG show?


Referred by:
Confirmed By
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

LMCA Obstruction


5 PAULA CRT
KANGAROO FLAT
15/08/15
James, Mr Dugal
MBP MEM:21191323B
CON:SN501553940
ADM: 891131 /
08/09/1940 74
Unitin M
3555 W. SUR
CL:All Privat
M/C:3161532417
Ref:1 11/2019
P/PR: 126/180 ms
QRS: 96 ms
QT/QTc: 350/463 ms
P/QRS/T Axis: 39/-27/-177 deg
Heart Rate: 105 BPM
warning: sex not available, assumed male
sinus tachycardia
horizontal axis
moderate high-lateral repolarization disturbance, consider ischemia or LV overload
negative T in I aVL
Abnormal ECG
9/10 chest tightness.
I
aVR
V1
V4
Unconfirmed Report
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s

LMCA Obstruction

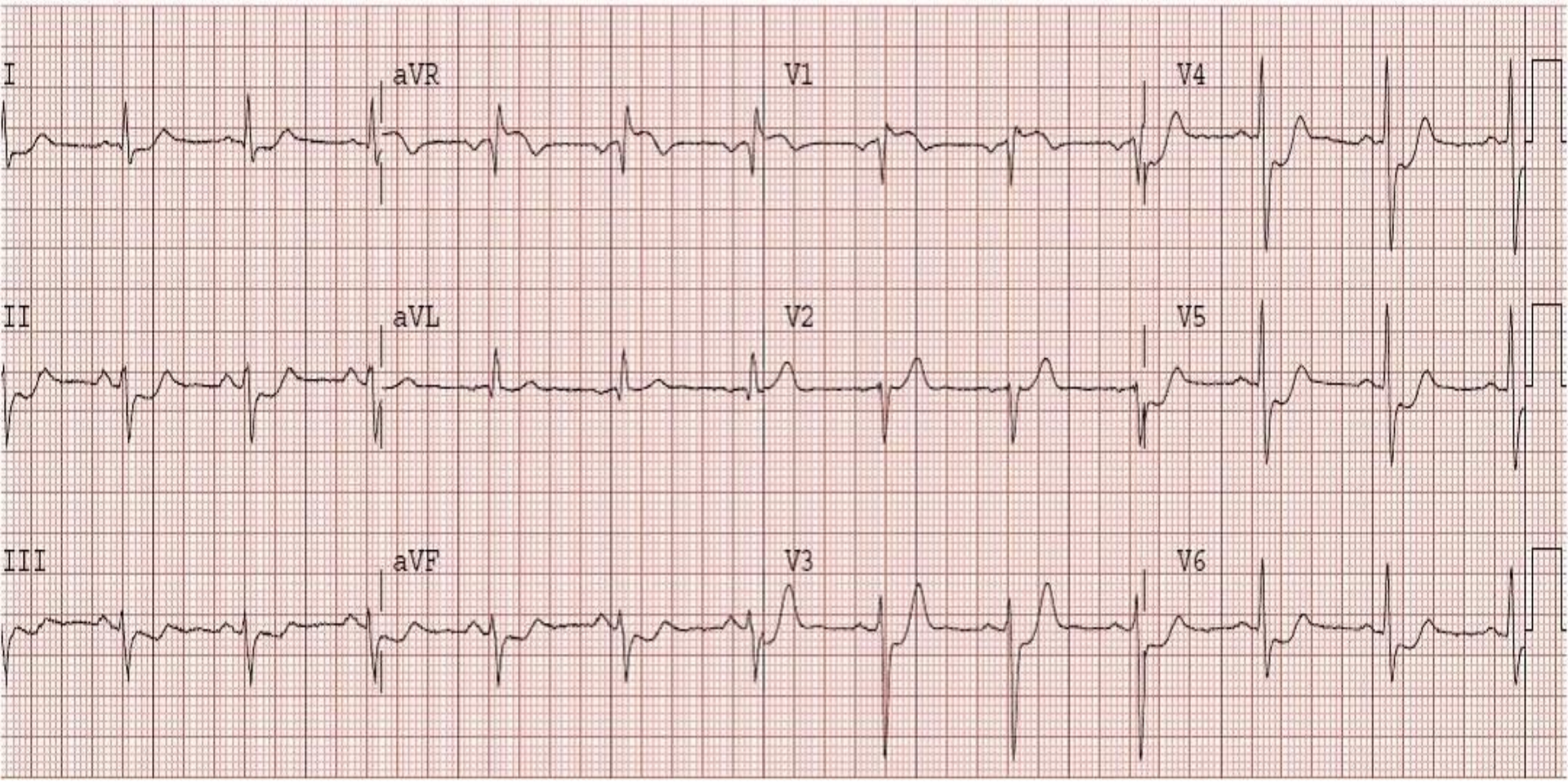
- Widespread horizontal ST depression
- ST elevation in aVR ≥ 1mm
- ST elevation in aVR ≥ V1

- Seen in
  - Prox LAD obstruction
  - Severe Triple vessel disease
  - Diffuse subendocardial ischaemia

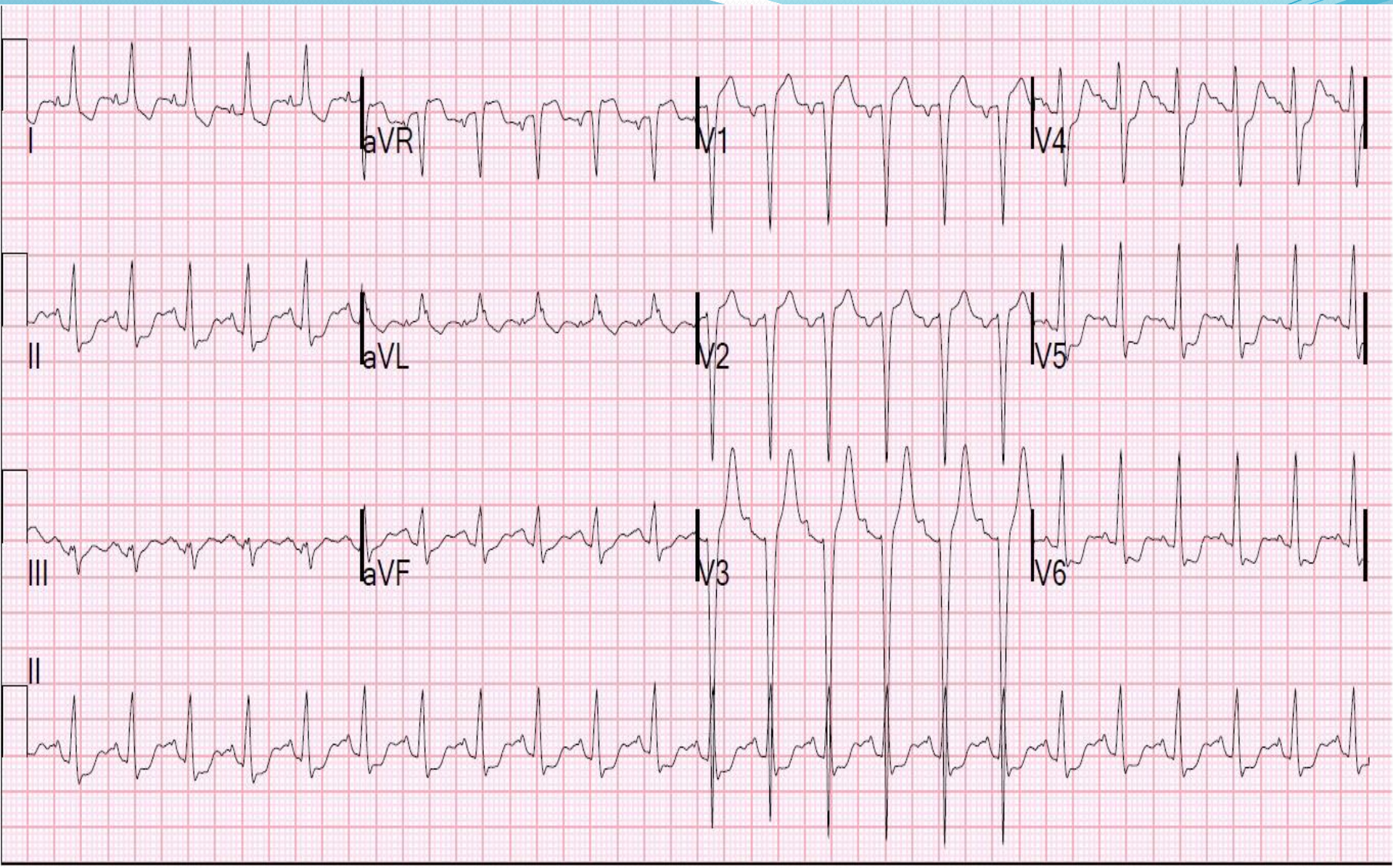
- *In the presence of anginal symptoms*,

- STE in aVR + STE in V1
  - - Highly predictive of LMCA or Prox LAD obstruction

- STE in aVR > STE in V1
  - - almost always indicates a LMCA obstruction (81% sensitive and 80% specific)



Patient had a severe ostial LAD thrombus that was close to the left main.



Another classic example of the LMCA / 3VD ECG pattern

- I would treat a patient with LMCA obstruction with all the following *except*:
  - Aspirin
  - Clopidogrel
  - Heparin
  - Early Cath lab

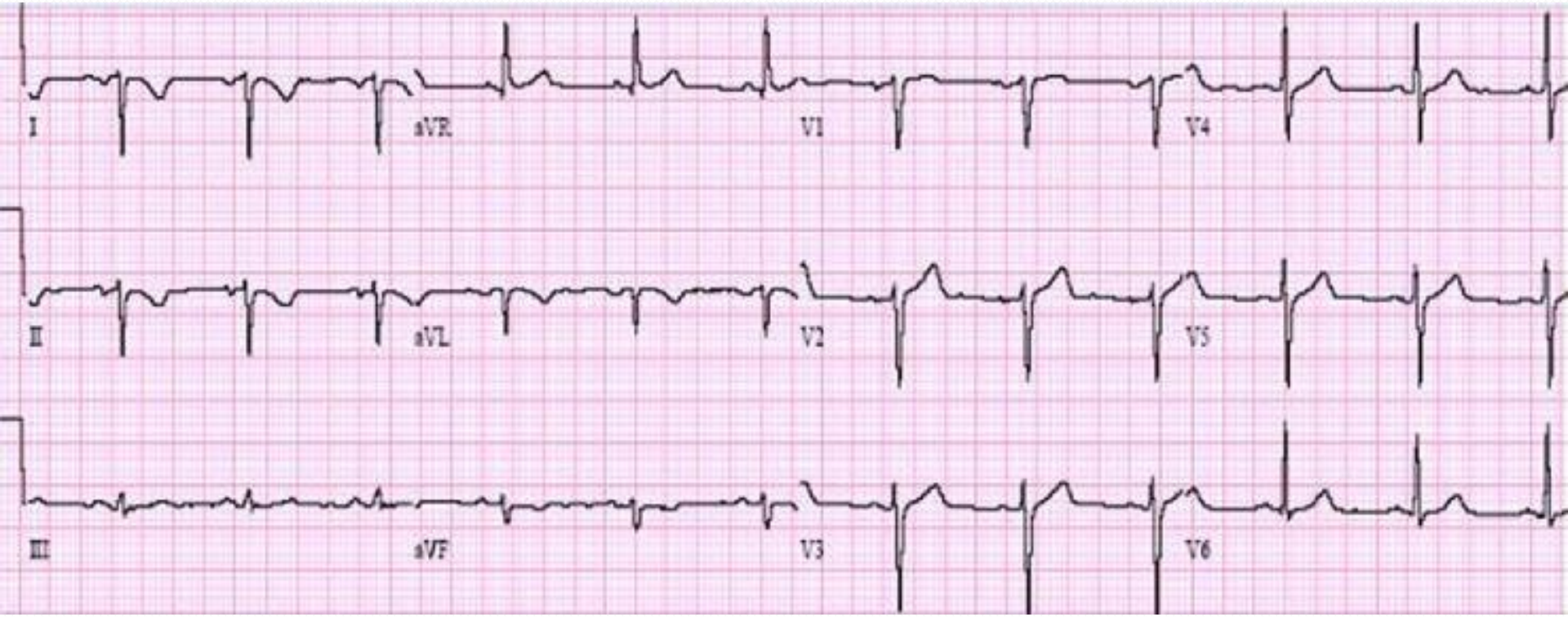
What’s the deal with Clopidogrel?

- Clopidogrel treatment ≤ 7 days before CABG is associated with an increase in major bleeding, haemorrhage-related complications, and transfusion requirements

- Prasugrel is associated with even more bleeding than Clopidogrel

- If urgent CABG (within 7 days) is likely, then there is an argument for omitting thienopyridines during the initial management of an acute coronary syndrome (or at least using Clopidogrel instead of Prasugrel).

Win a champagne!


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