Pattern recognition

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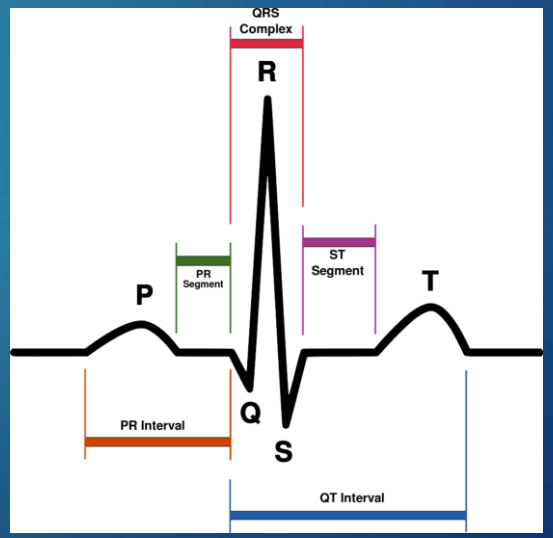
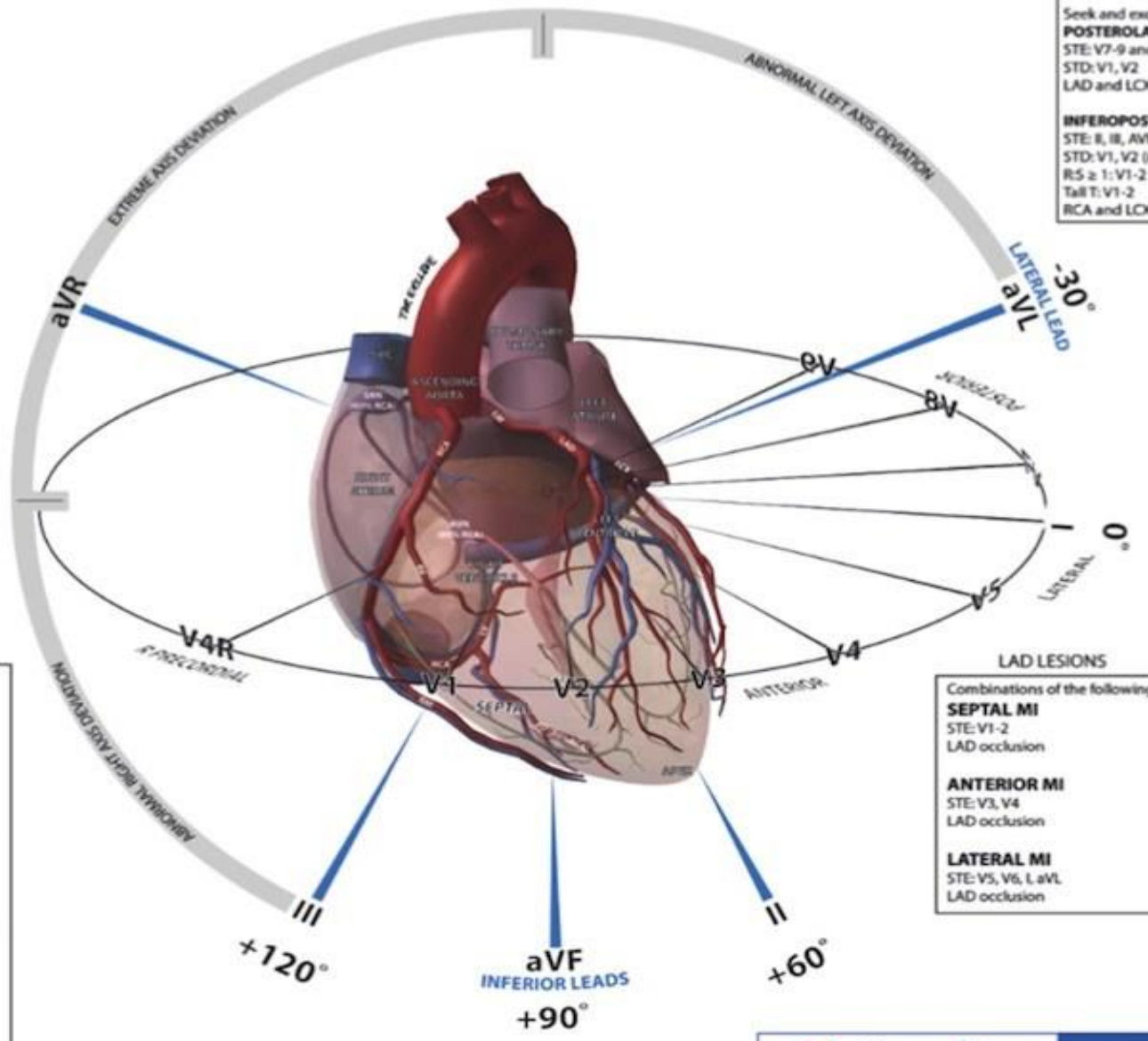
ED PHYSICIAN AND CO-DEMT
BENDIGO HEALTH



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

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

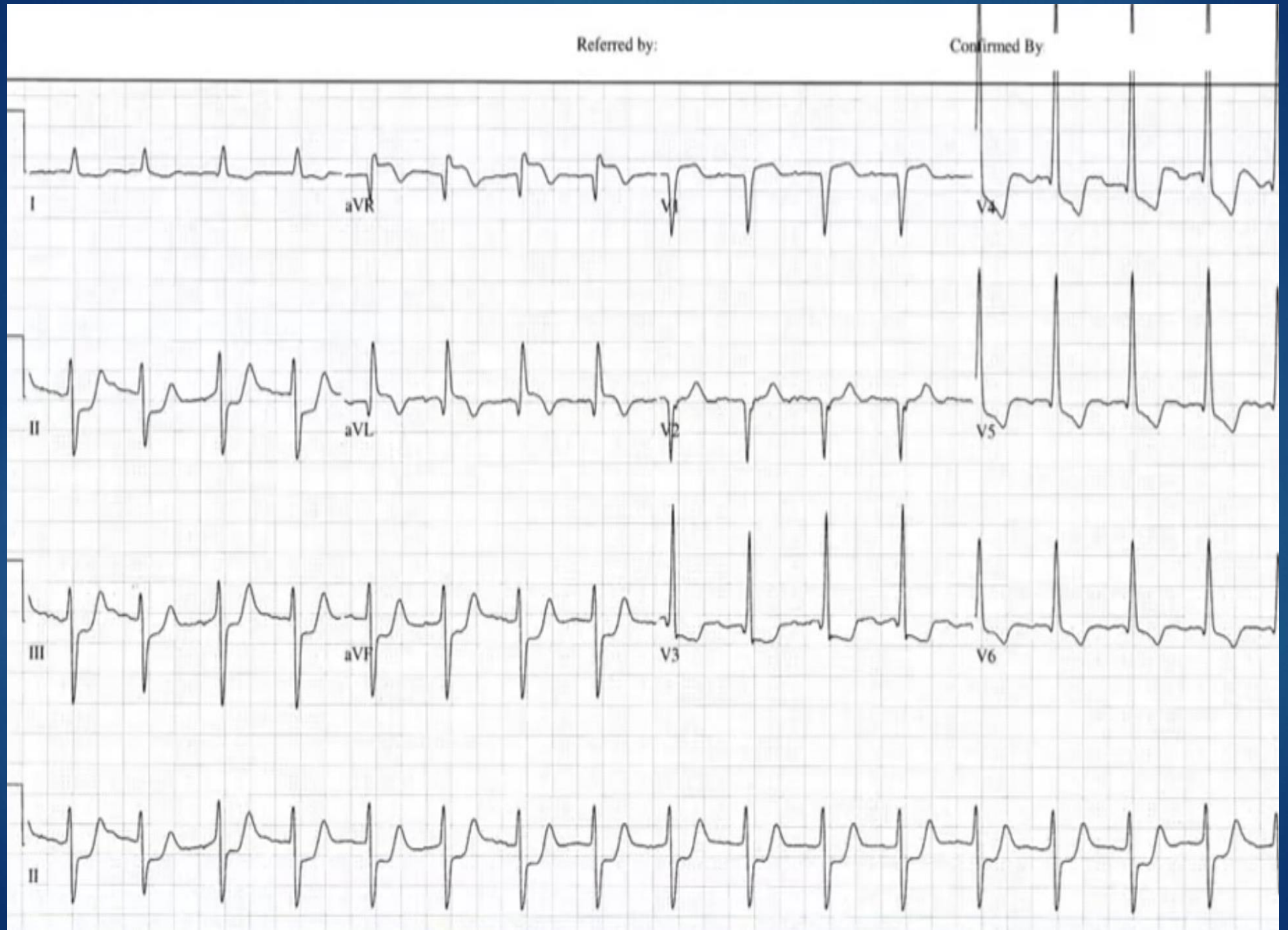
Combinations of the following
SEPTAL MI
STE: V1-2
LAD occlusion

ANTERIOR MI
STE: V3, V4
LAD occlusion

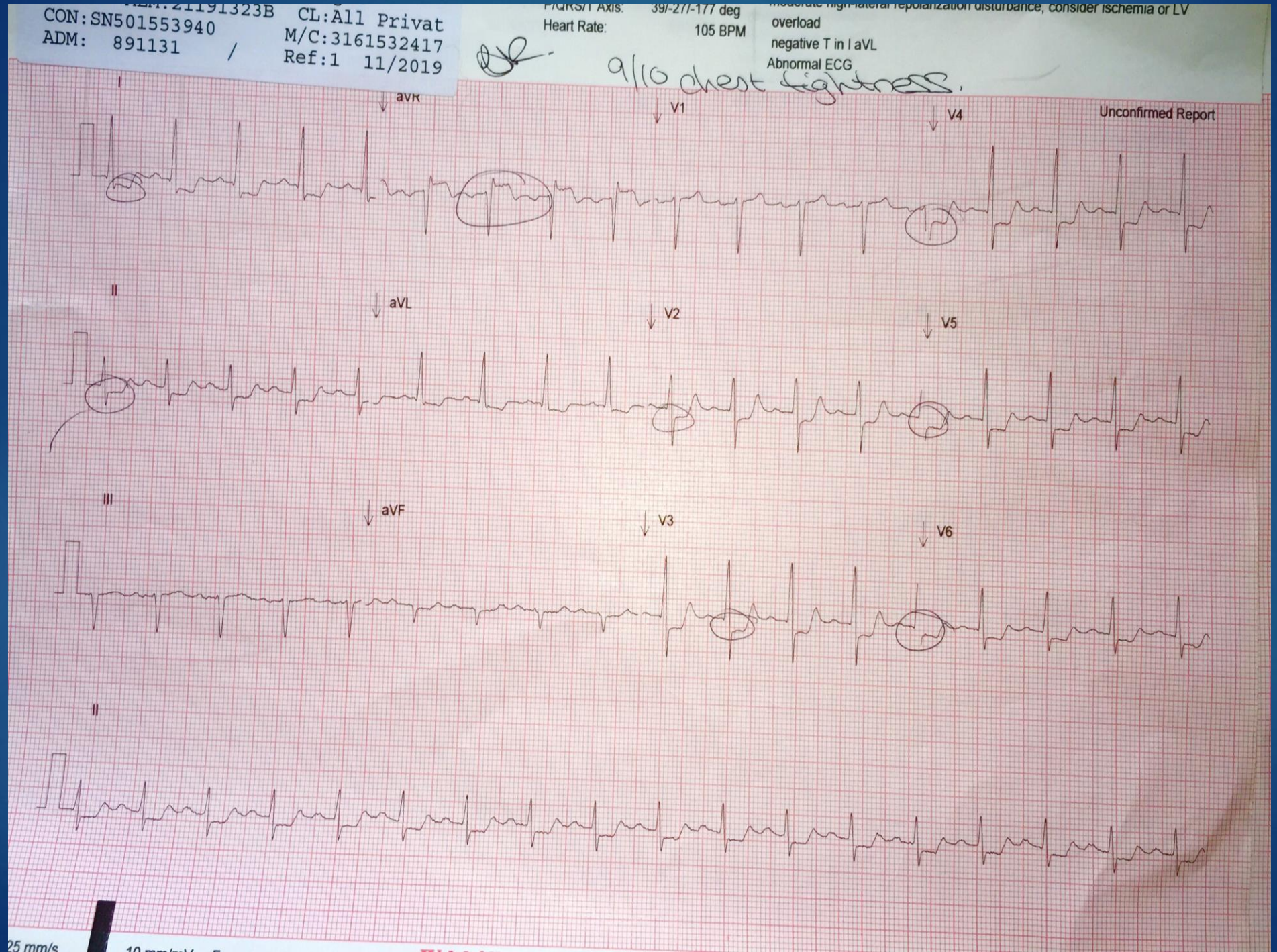
LATERAL MI
STE: V5, V6, I, aVL
LAD occlusion

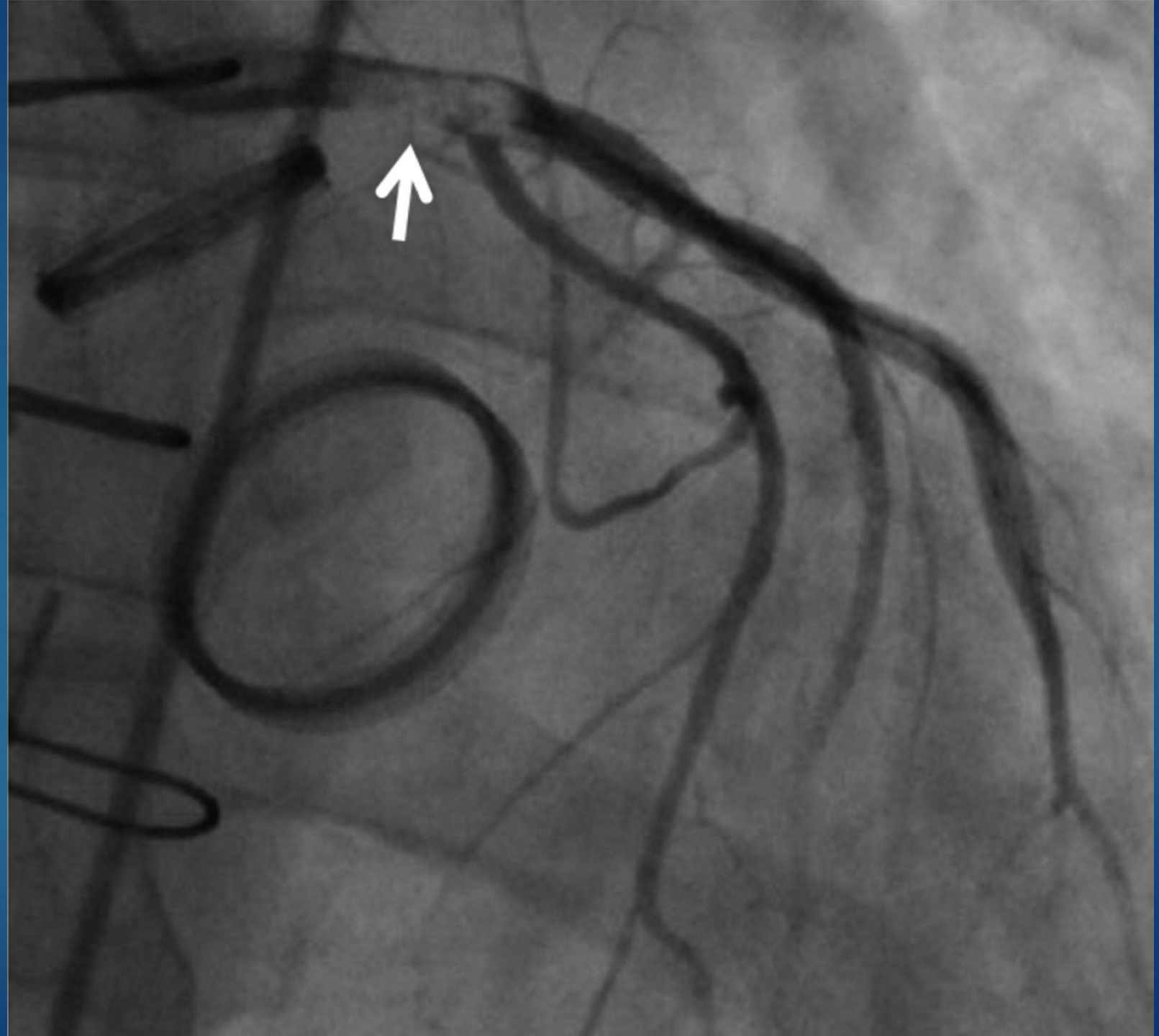
- 75yo male with HTN, DM-2 complains of angina

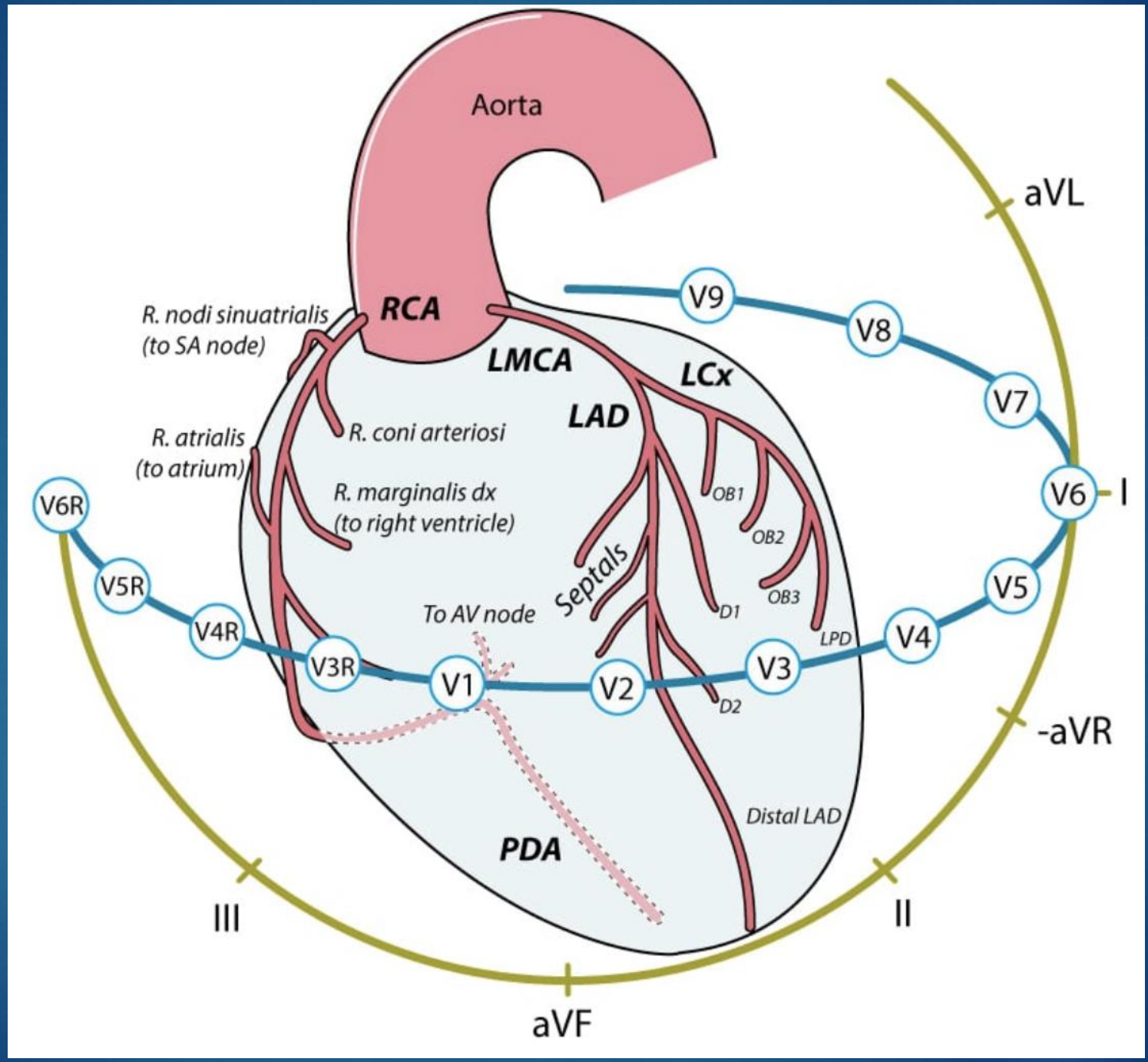
- What does this ECG show?


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

LMCA Obstruction


CON:SN501553940
ADM: 891131
CL:All Privat
M/C:3161532417
Ref:1 11/2019
P/QRS/T Axis: 39/-27/-177 deg
Heart Rate: 105 BPM
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




Aorta
RCA
R. nodi sinuatrialis
(to SA node)
LMCA
LCx
LAD
R. atrialis
(to atrium)
R. coni arteriosi
R. marginalis dx
(to right ventricle)
OB1
OB2
OB3
D1
D2
LPD
Septals
To AV node
Distal LAD
PDA
V9
V8
V7
V6
V5
V4
V3
V2
V1
V3R
V4R
V5R
V6R
aVL
I
-aVR
II
aVF
III

LMCA Obstruction

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

Also seen in

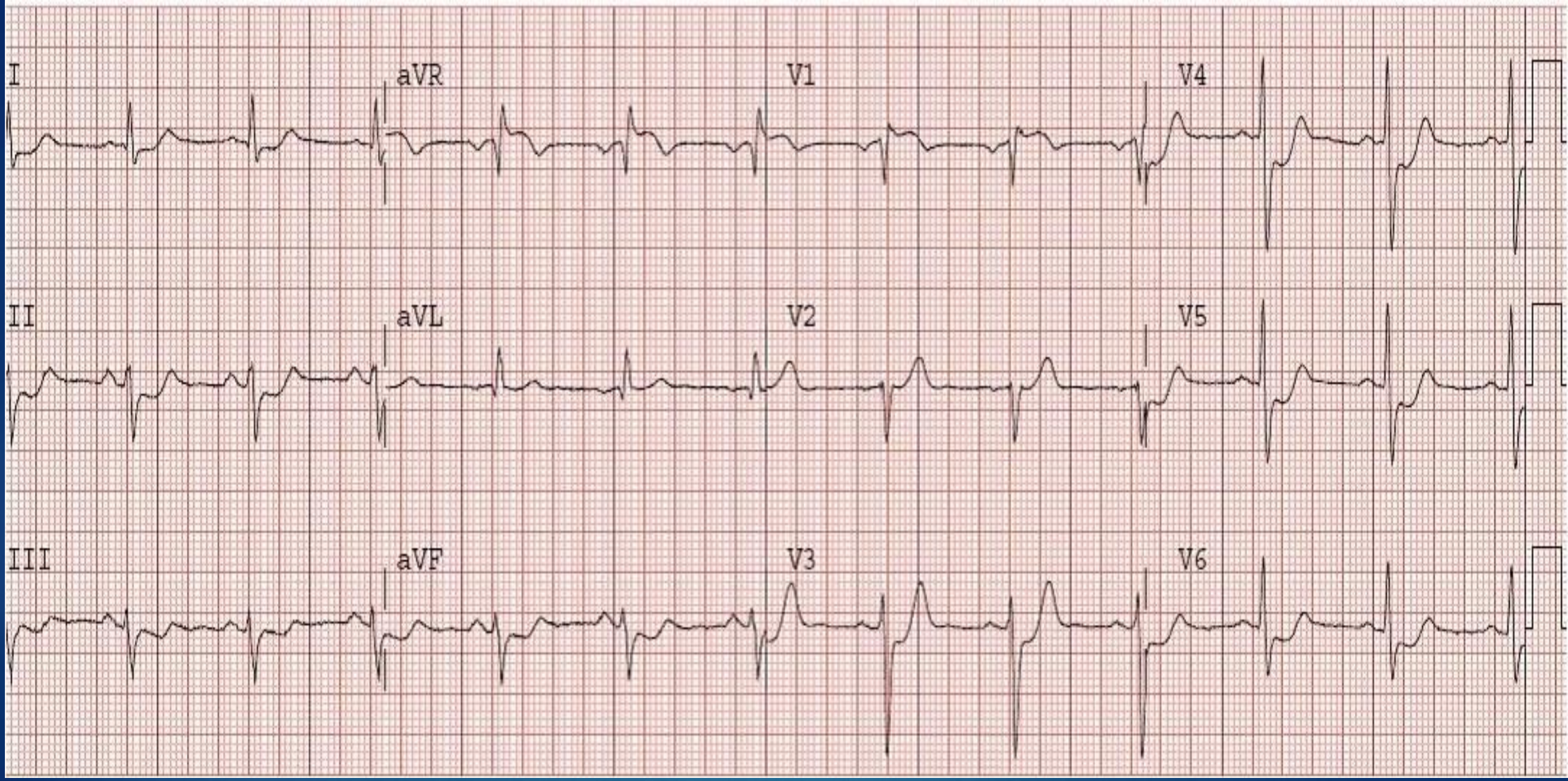
- Prox LAD obstruction
- Severe Triple vessel disease
- Diffuse subendocardial ischaemia

PE, LVH with strain, LBBB(including PPM), SVTs, hypok+, aortic dissection, Na ch pathology (TCA, Brugada etc), severe anaemia

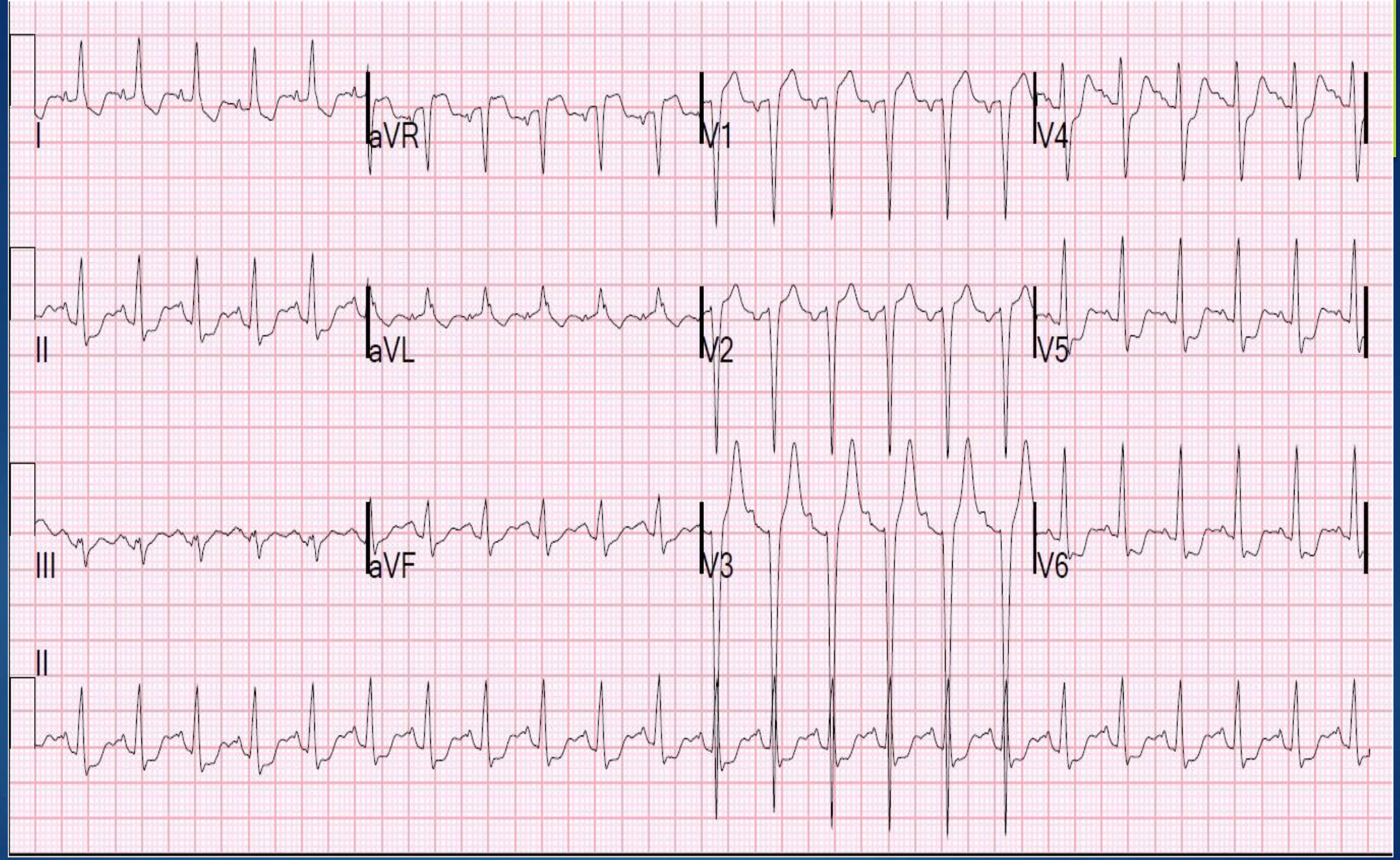
In the presence of anginal symptoms,

- 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)



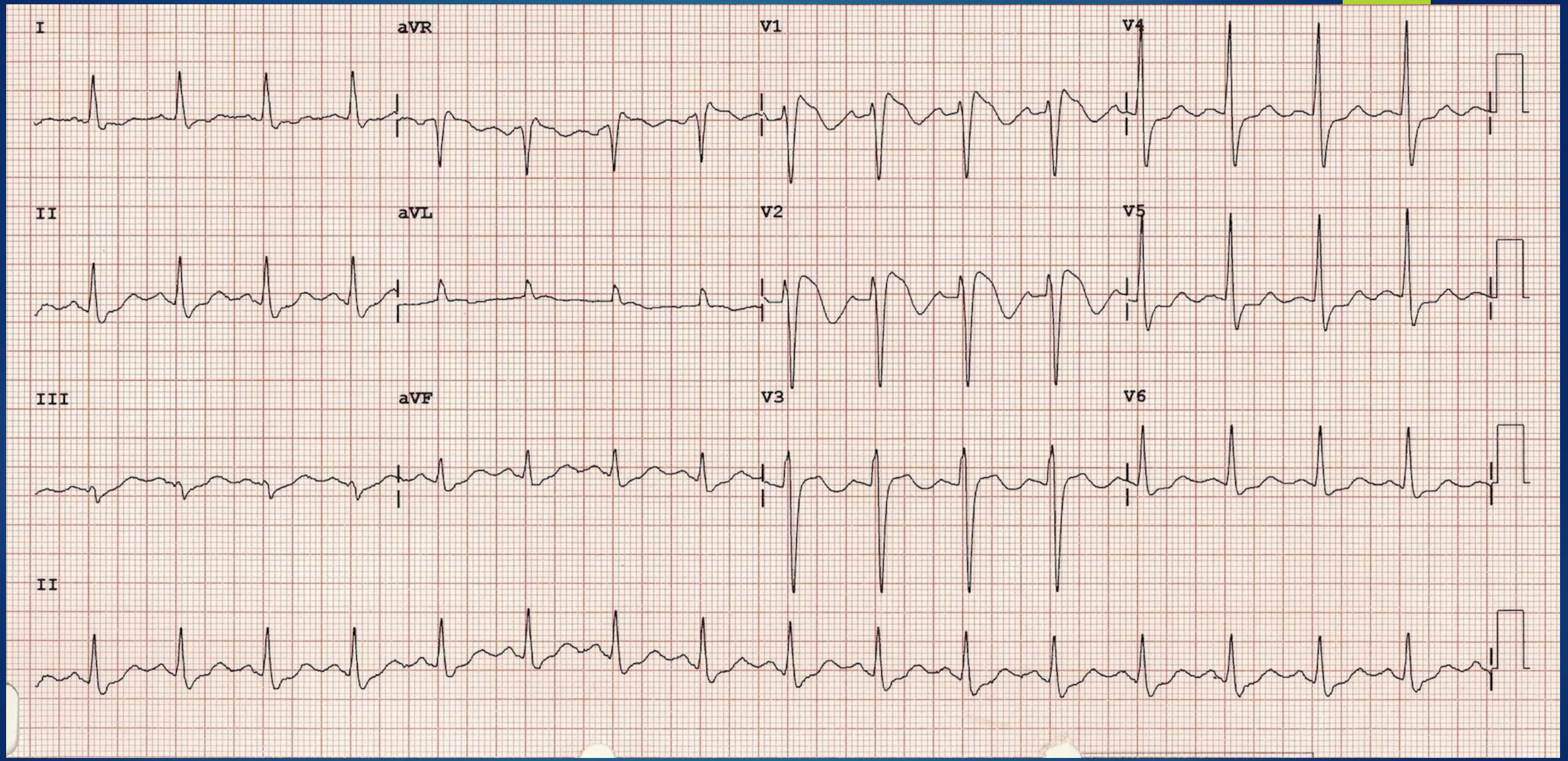
Patient had a severe ostial LAD thrombus that was close to the left main.



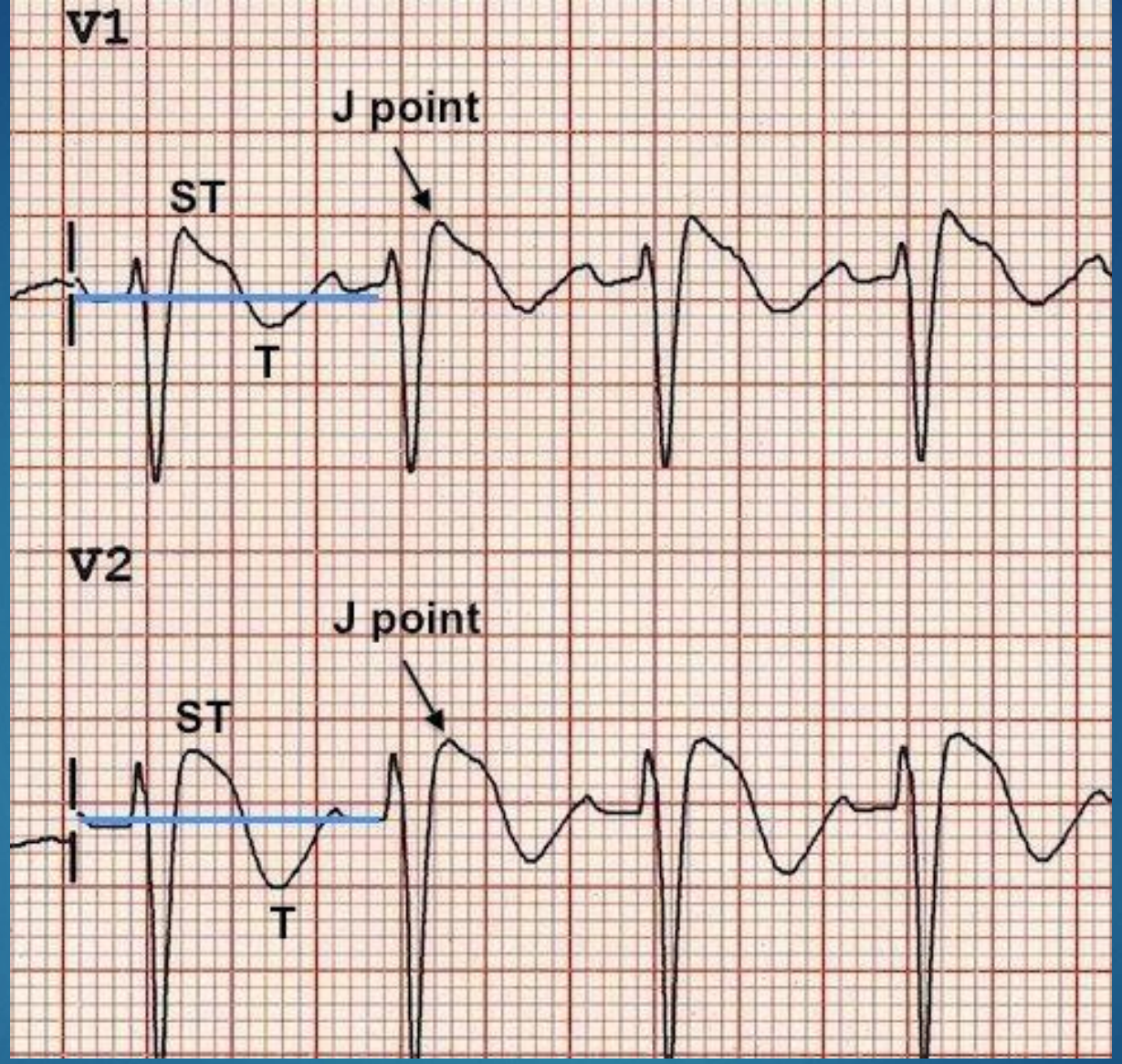
Another classic example of the LMCA / 3VD ECG pattern

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

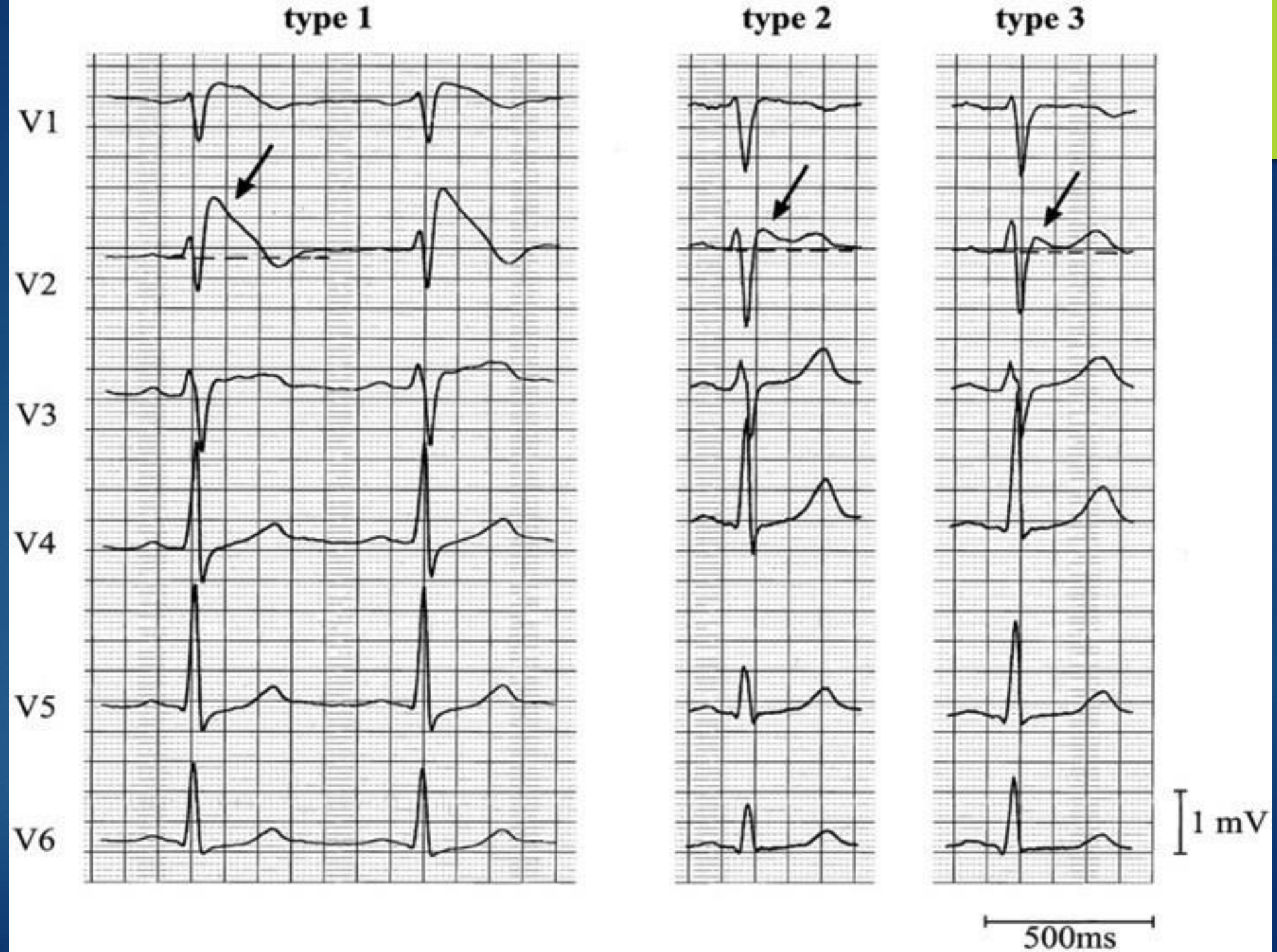
40yo female with anxiety, palpitations and pseudoseizures

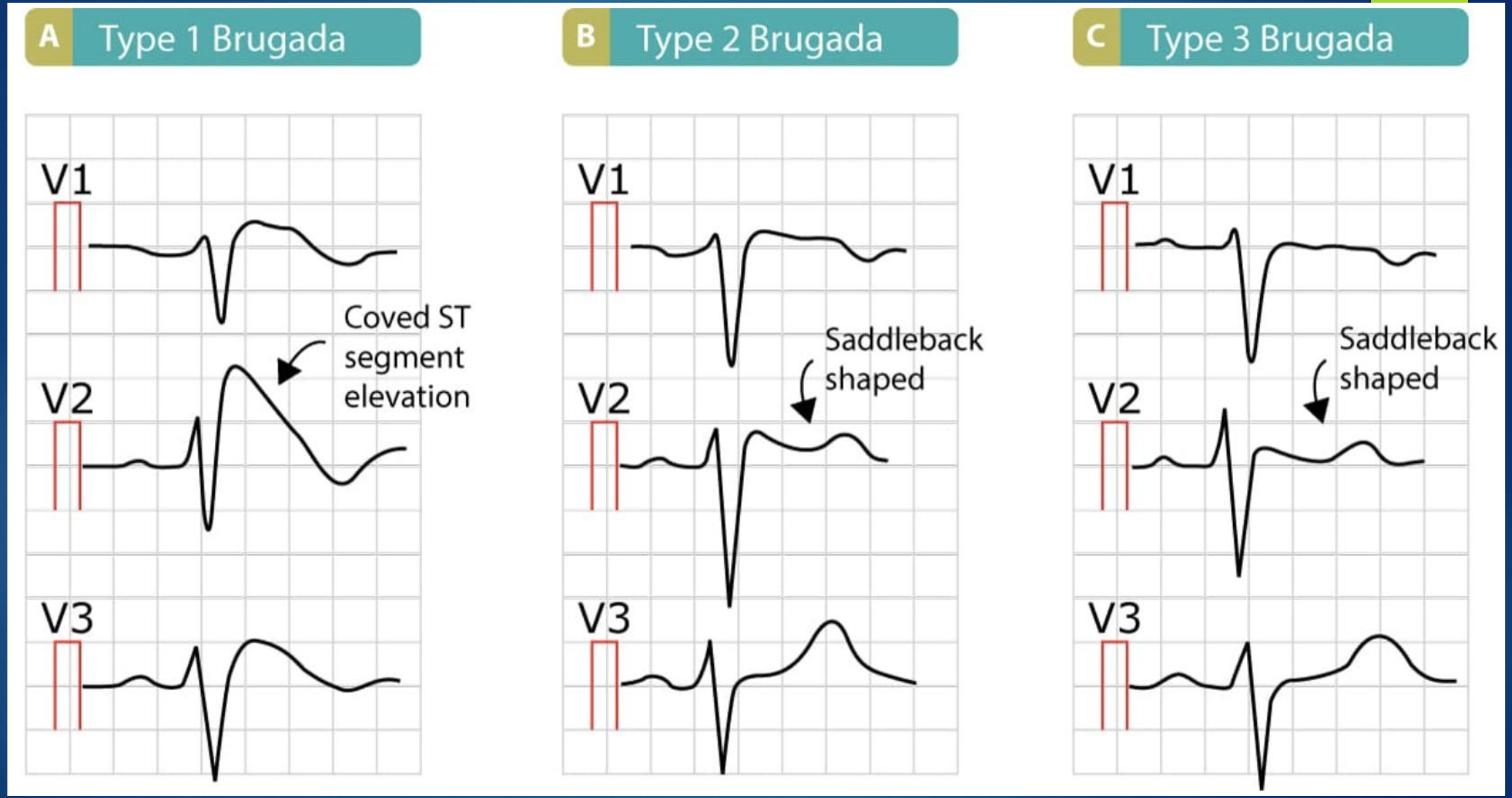

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

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


A Type 1 Brugada
V1
Coved ST segment elevation
V2
V3
B Type 2 Brugada
V1
Saddleback shaped
V2
V3
C Type 3 Brugada
V1
Saddleback shaped
V2
V3

Diagnosis

ECG plus one of the following:

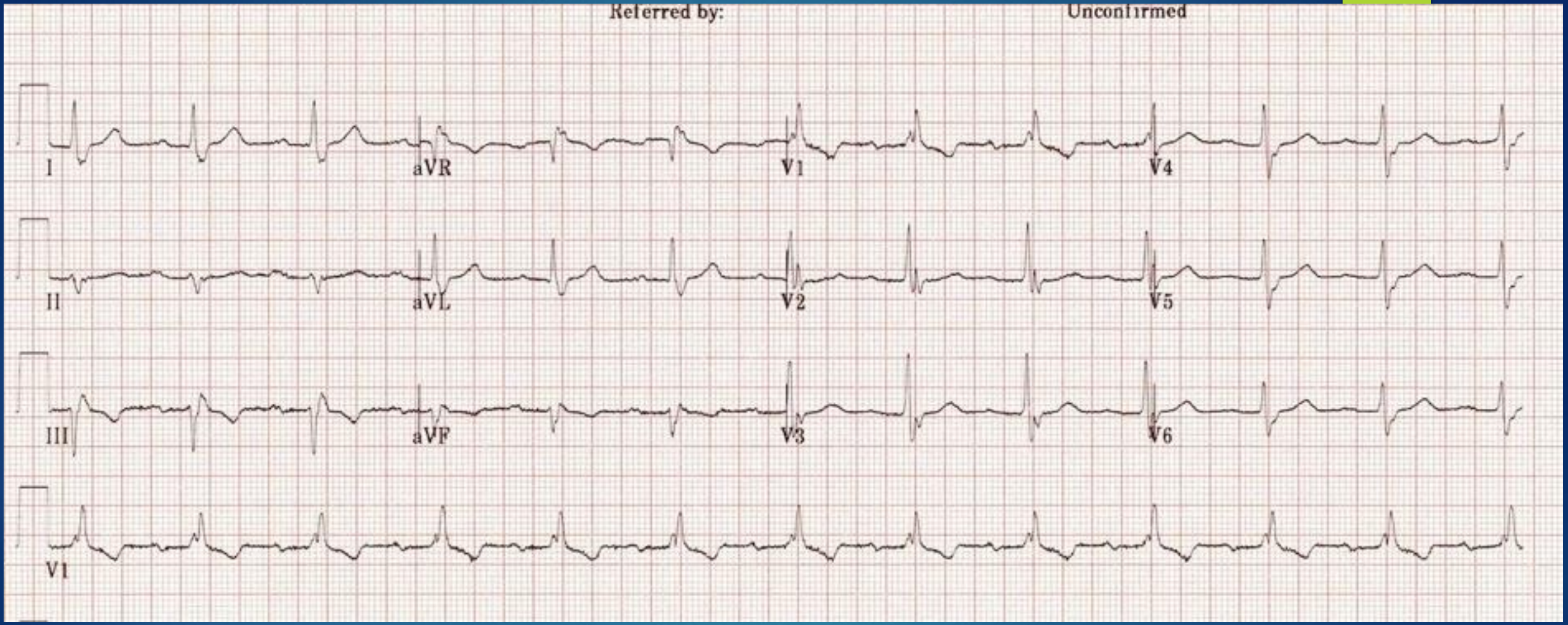
- Documented VF or VT
- Family history of SCD at <45 years old
- Coved-type ECGs in family members
- Syncope
- Nocturnal agonal respiration

Only proven therapy is ICD

Take Home points

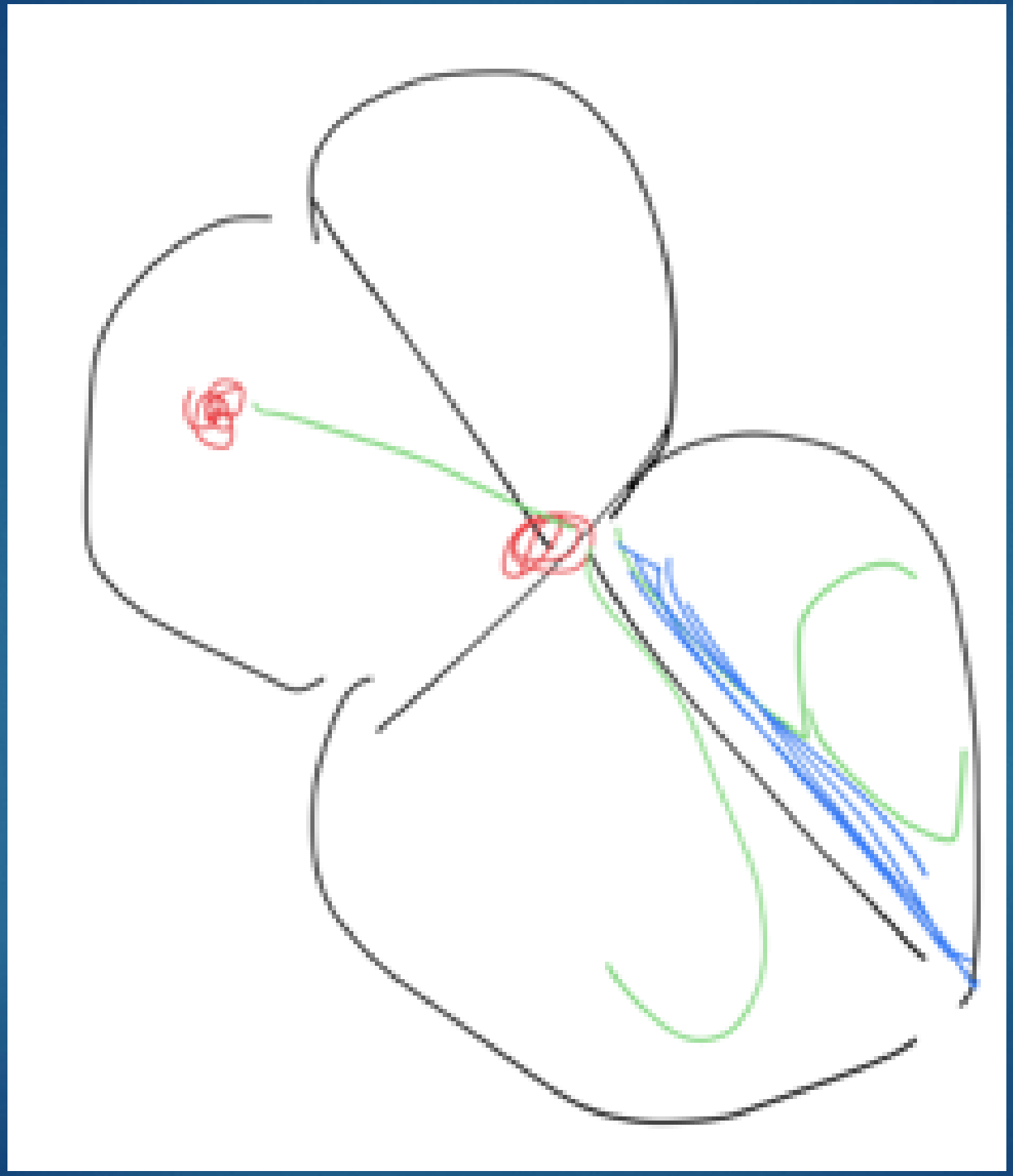
- Consider Brugada syndrome in any patient presenting after syncope
- ECG: (I)RBBB + STE in V1 - V2
  - Coved STE is most concerning
- Discuss/ refer to electrophysiologist

50yo male with syncope

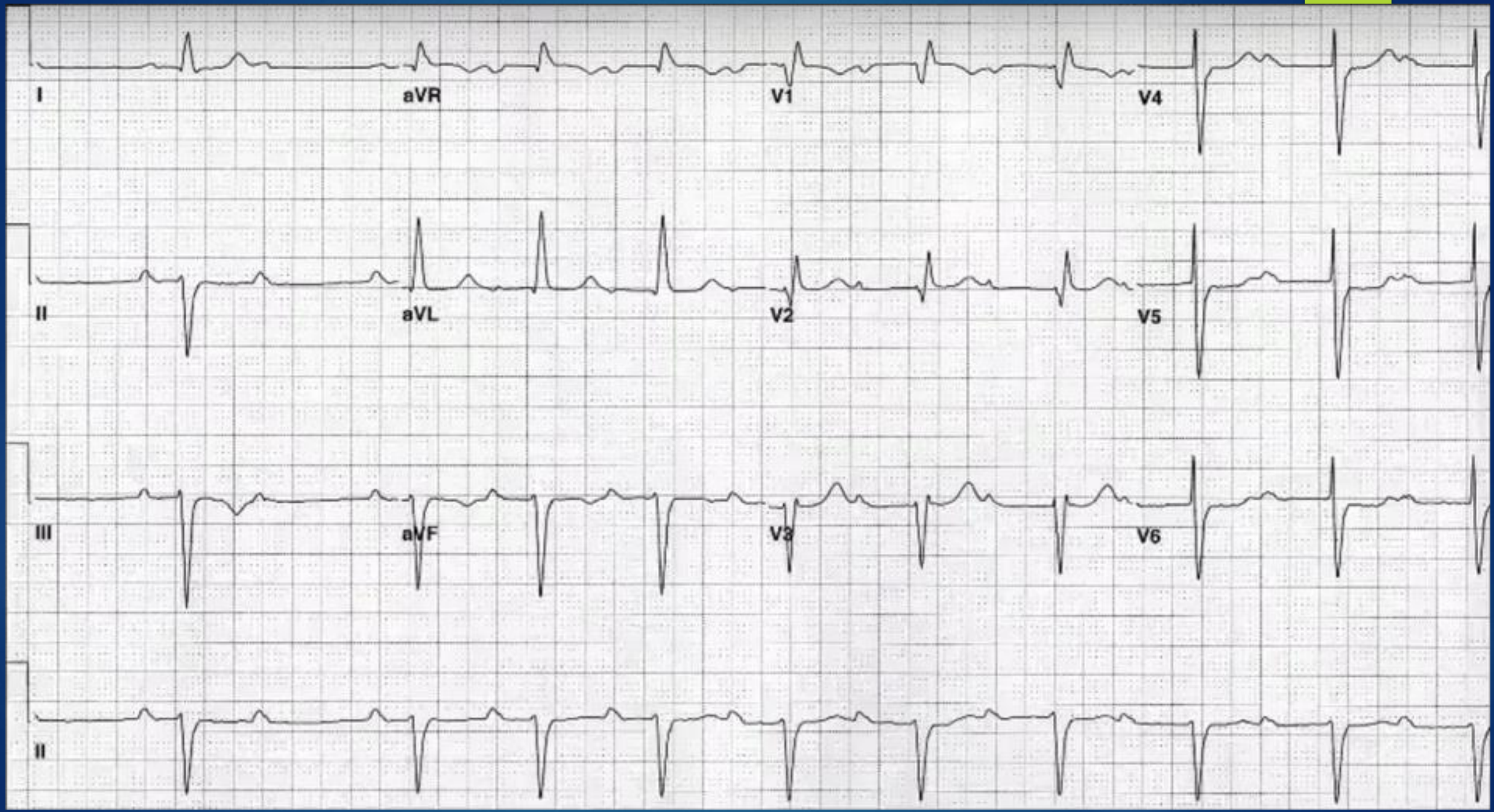

Referred by:
Unconfirmed
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

Trifascicular block

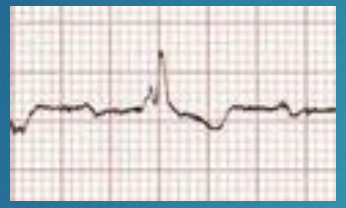
- But AVN is not a fascicle - why is it a trifascicular block?

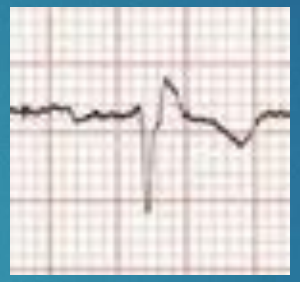


Trick ECG


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

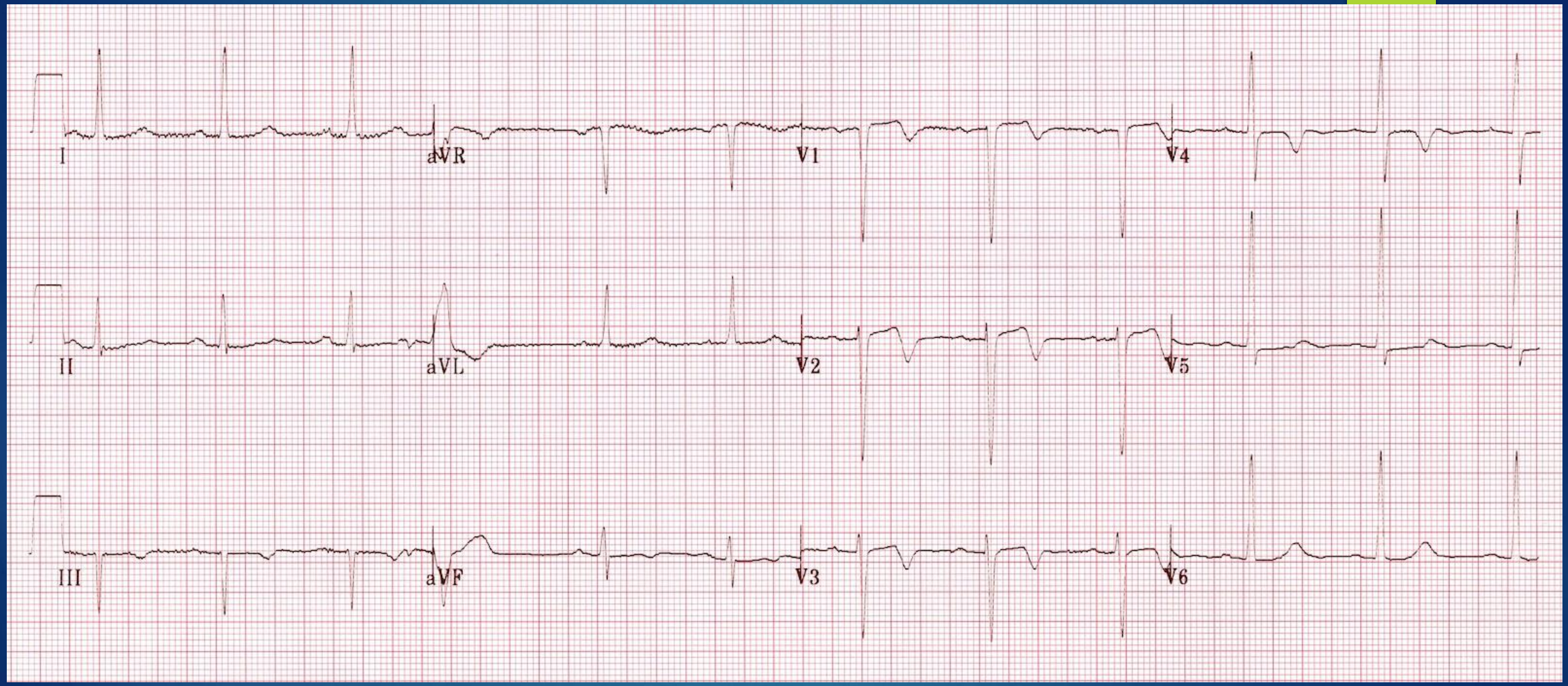
Take home points

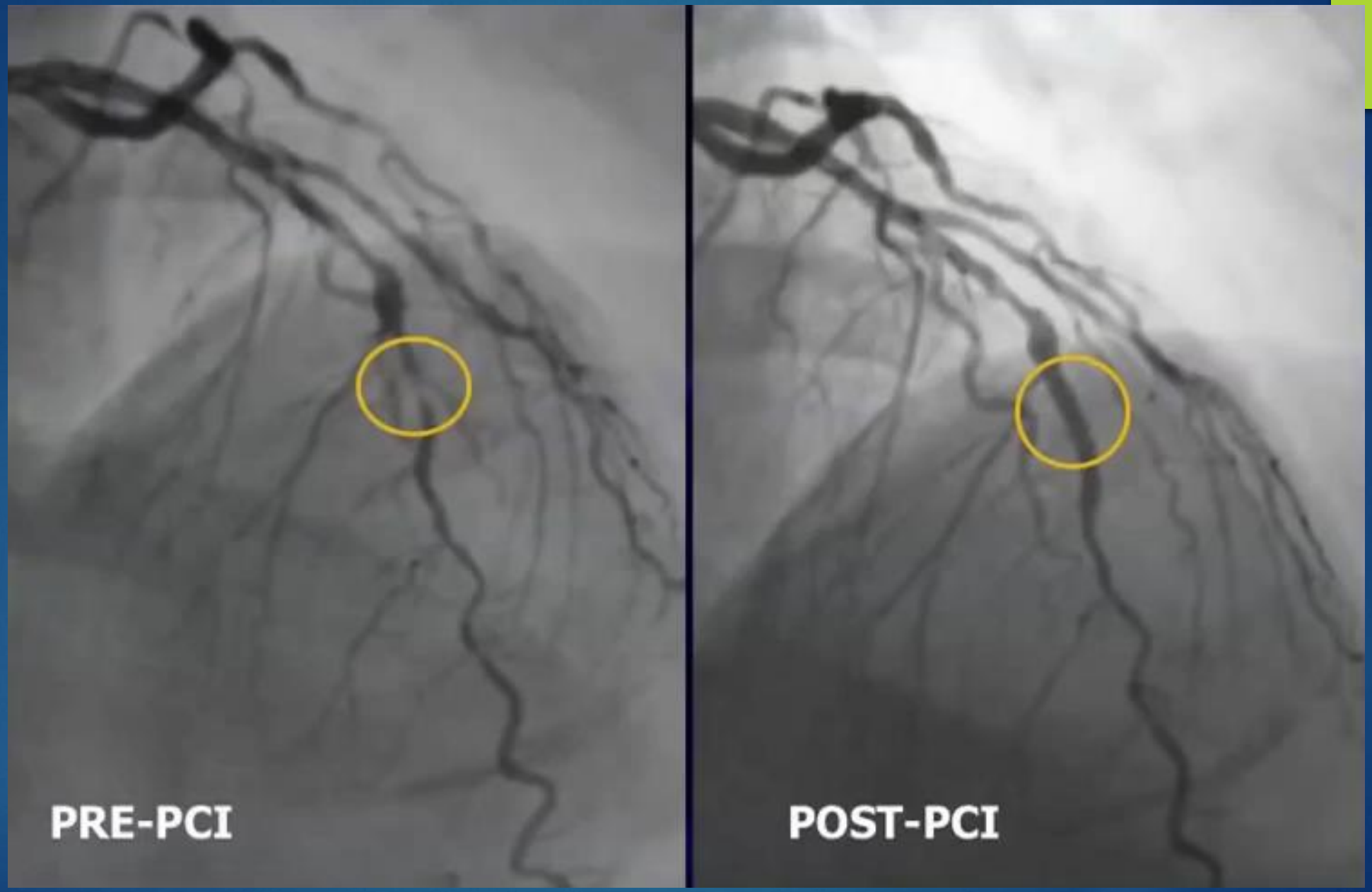




- RBBB + LAFB + I° AV Block = TFB
- 50% of them will need a PPM in future
- More commonly prevalent than previously thought

- 54yo male
- History of chest pain for 20mins – resolved with antacids
- Now pain free and in your clinic
- 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*

- 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

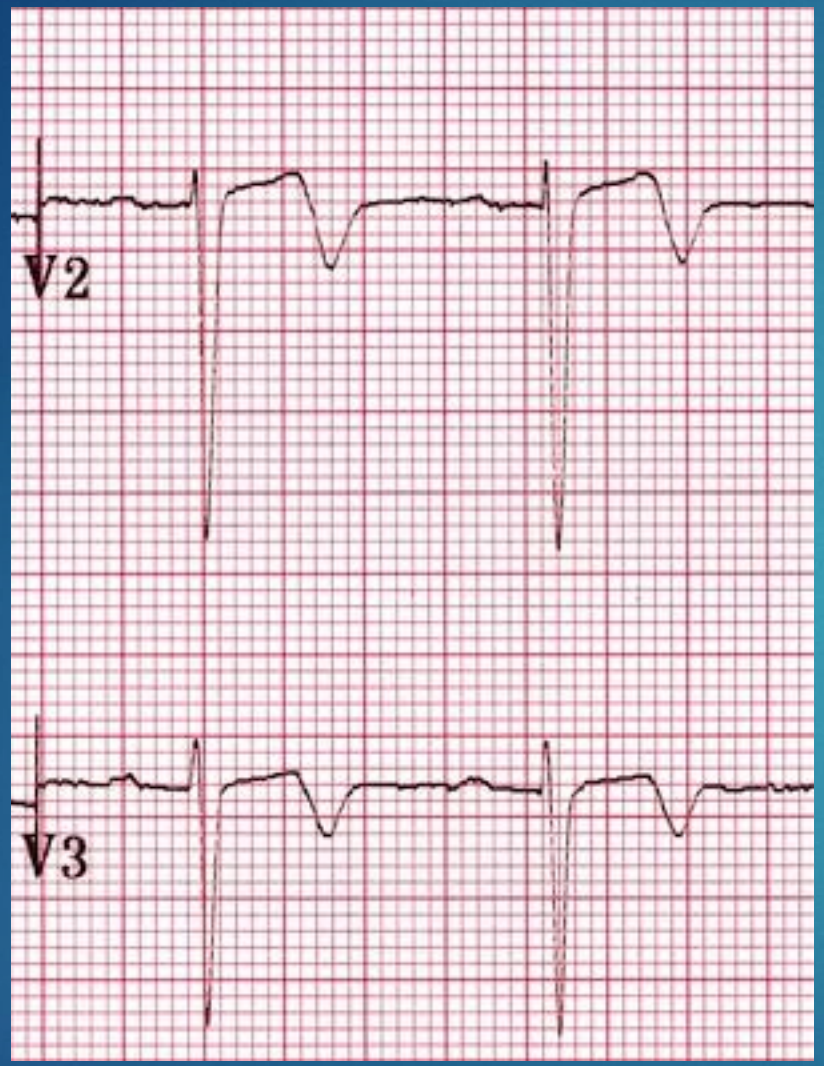
- Normal or slightly elevated serum cardiac marker

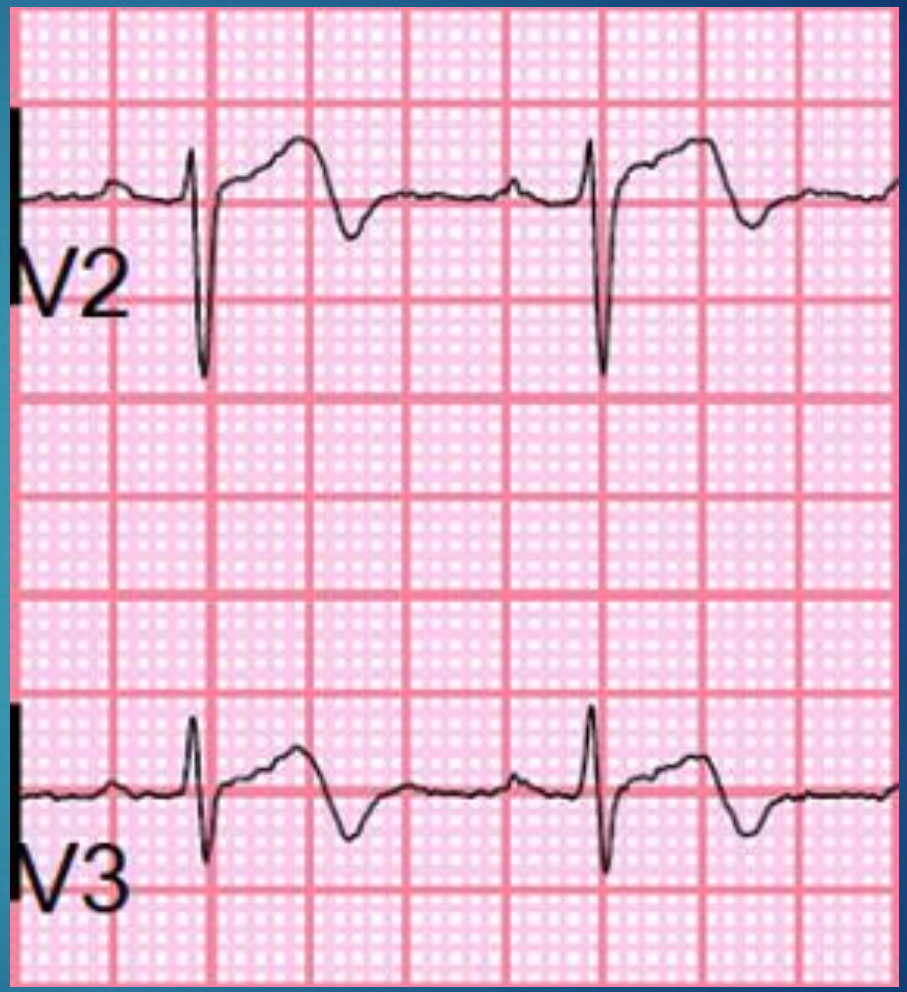
Two patterns of T-wave abnormality:

- *Type A* = Biphasic, with initial positivity & terminal negativity (25% of cases)

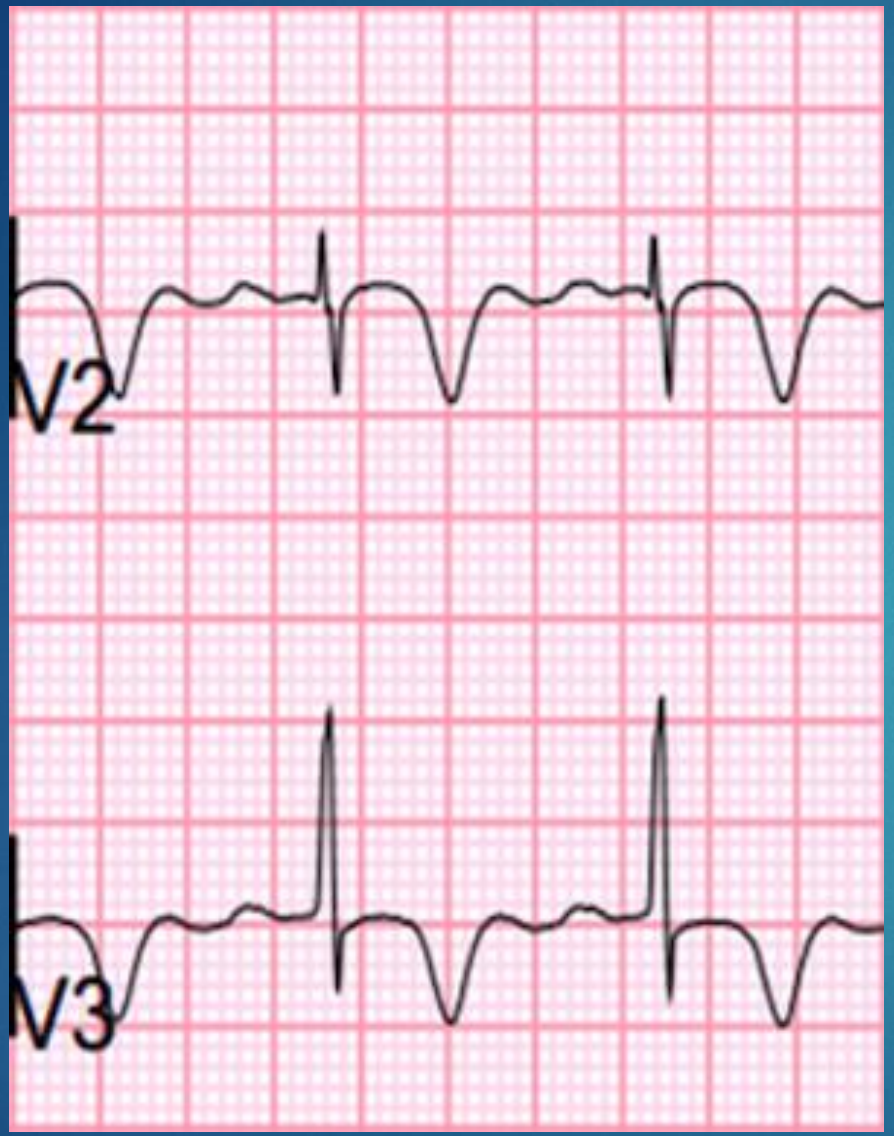
- *Type B* = Deeply and symmetrically inverted (75% of cases)

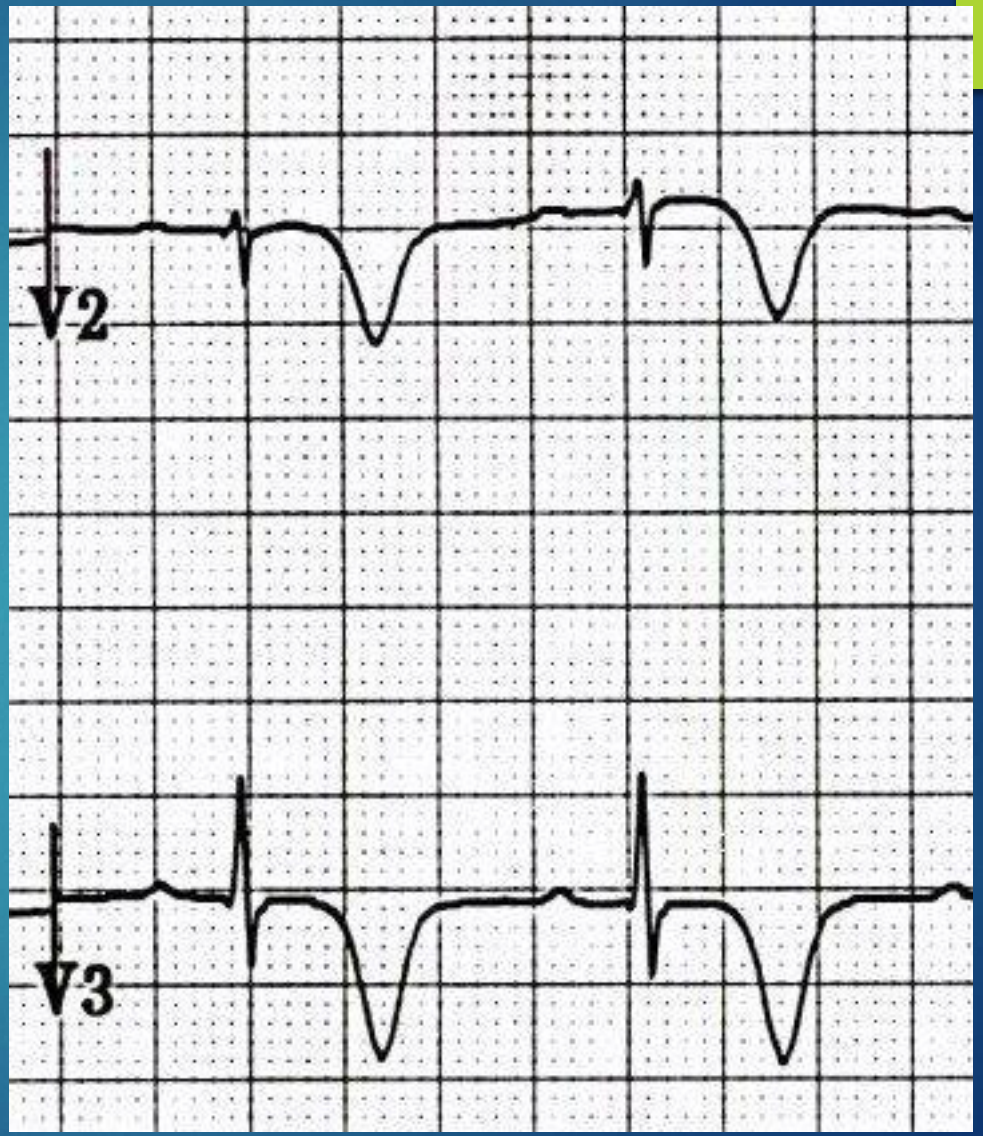
Computer often calls it non-specific T-wave abnormality



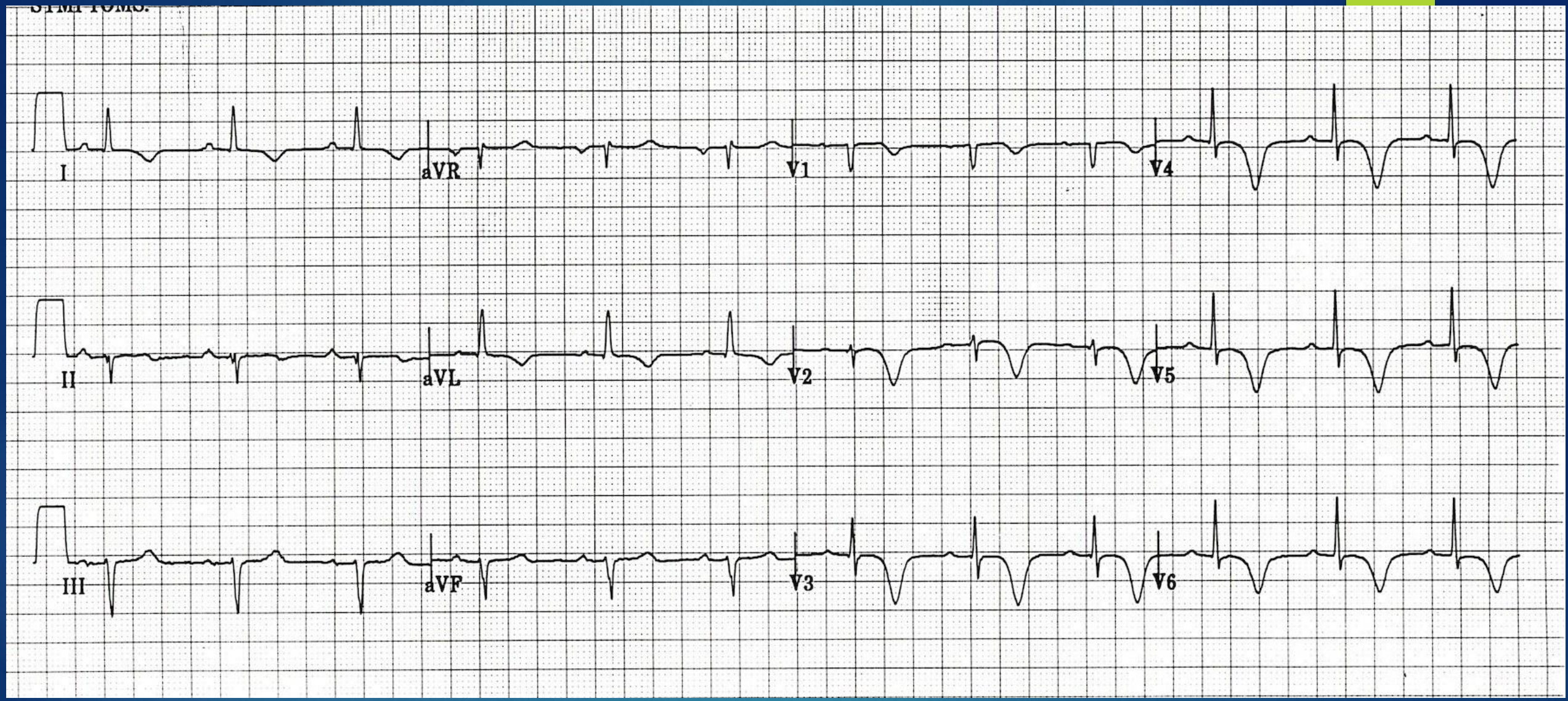


Biphasic T Waves (Type A)

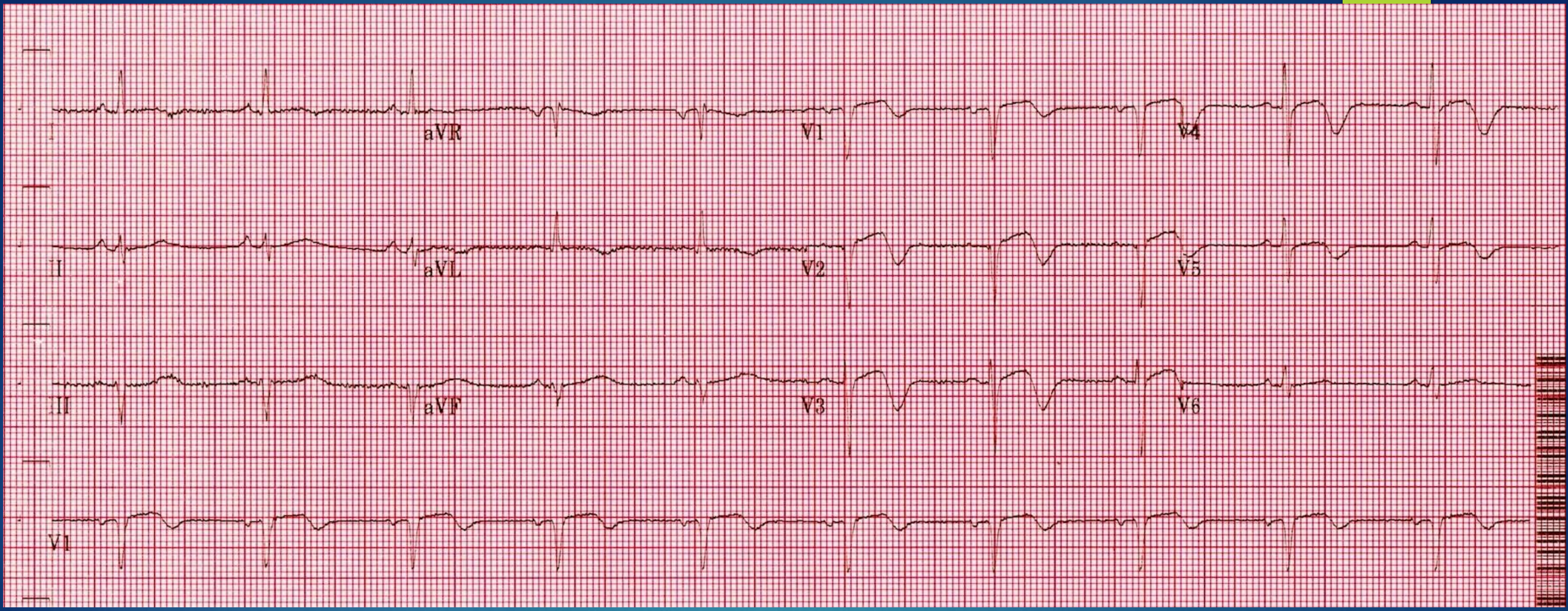




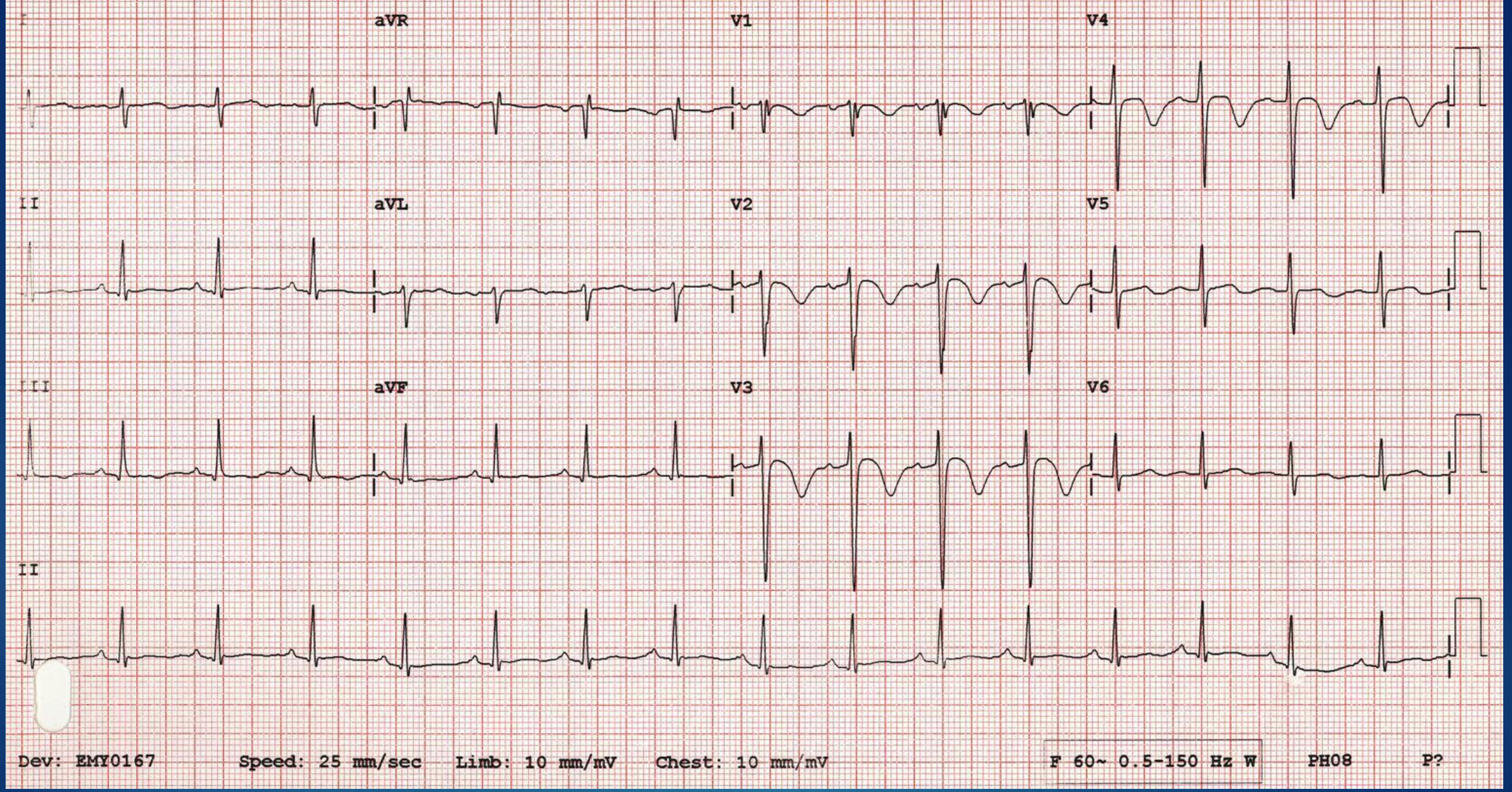
Deeply Inverted T Waves (Type B)



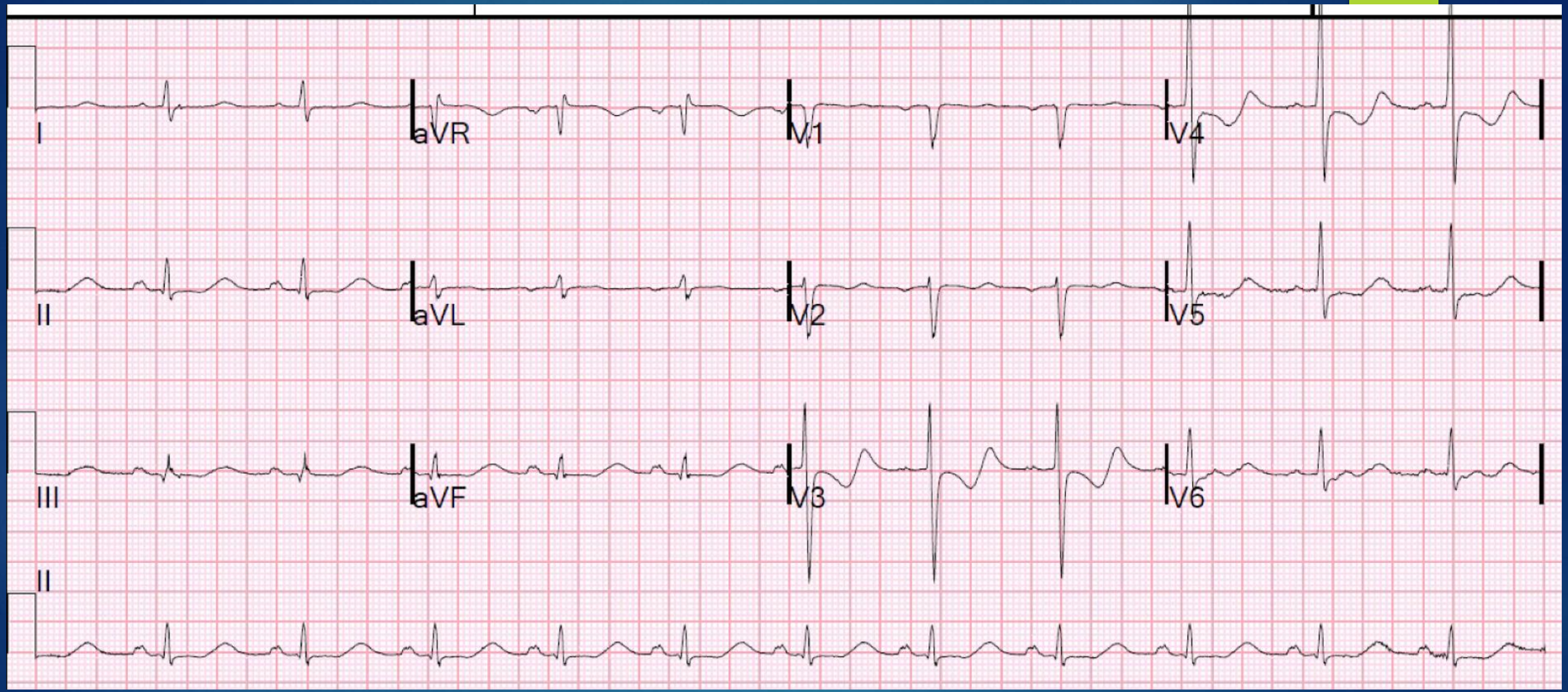
There are deep, symmetrical T wave inversions throughout the anterolateral leads (V1-6, I, aVL)



- The patient had experienced angina immediately prior to arrival in hospital and was pain free at the time the ECG was taken



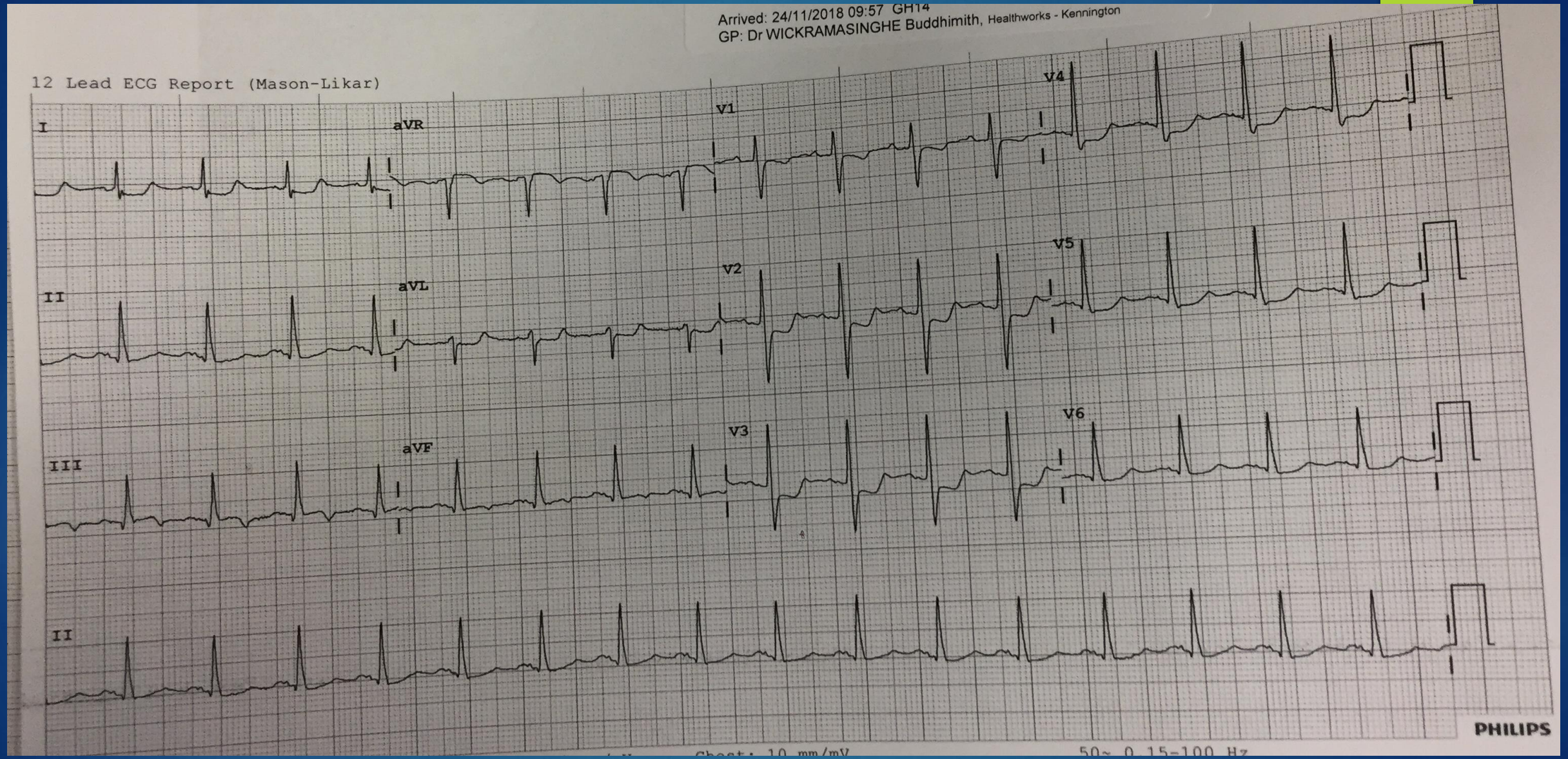
Is this Wellens?

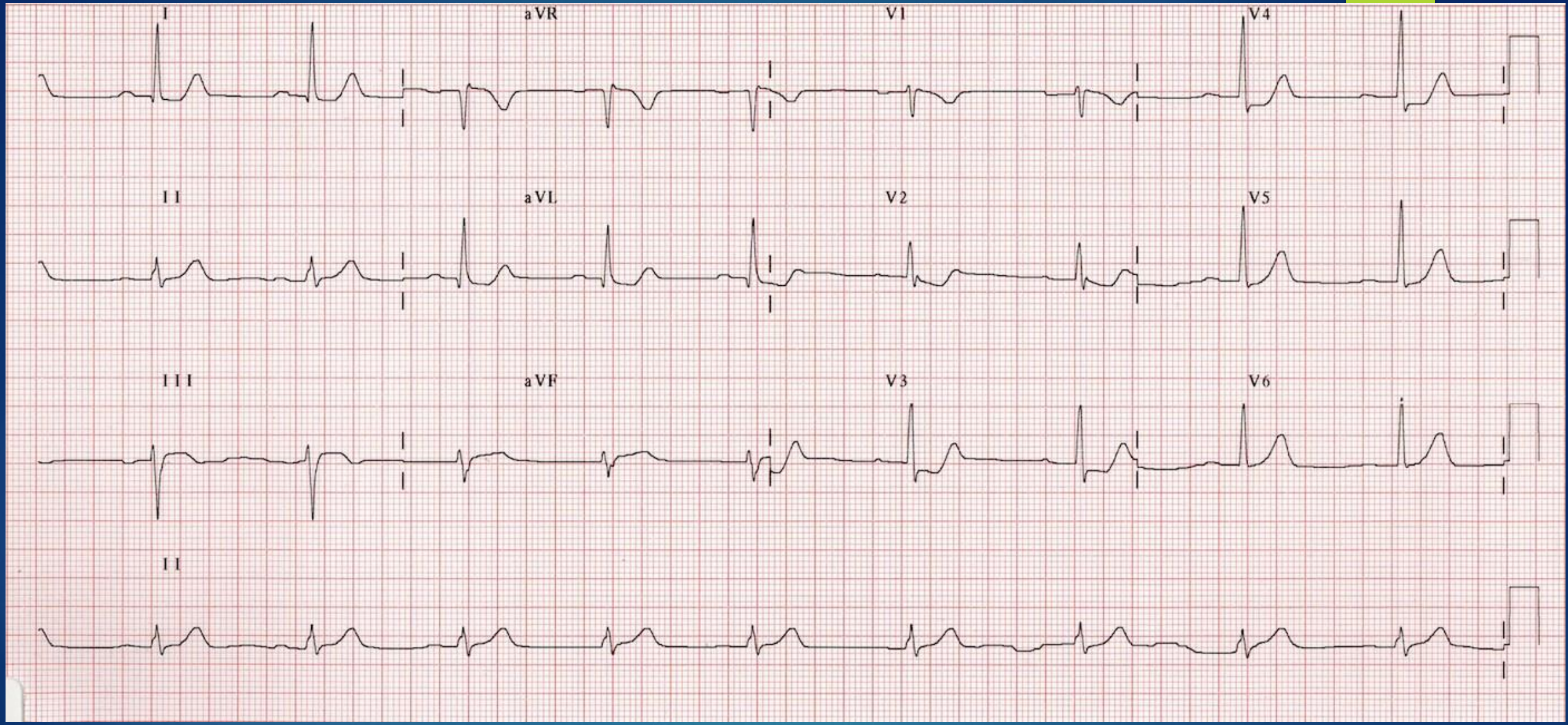


Is this Wellens?

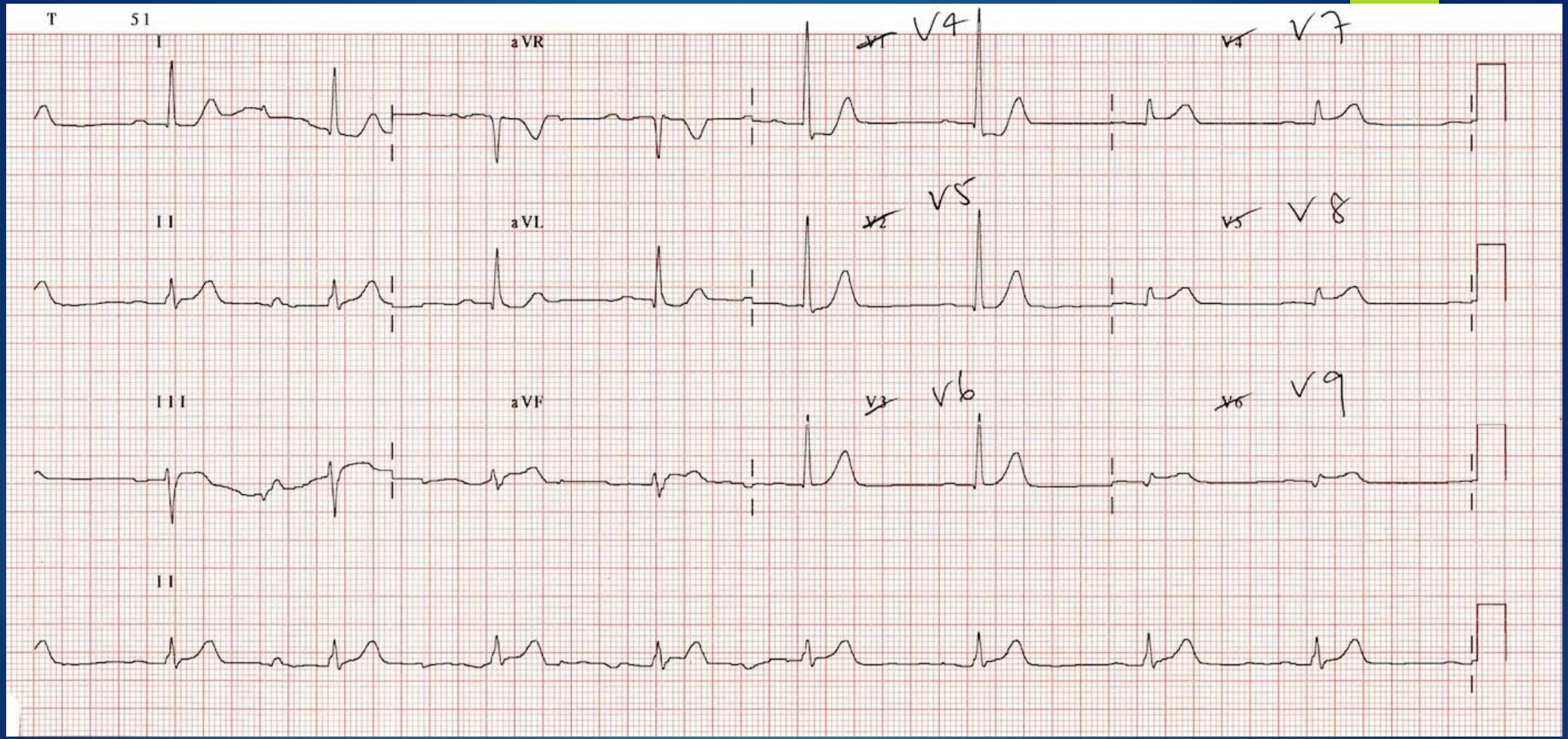
Take home Points

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


Arrived: 24/11/2018 09:57 GH14
GP: Dr WICKRAMASINGHE Buddhimith, Healthworks - Kennington
12 Lead ECG Report (Mason-Likar)
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
PHILIPS

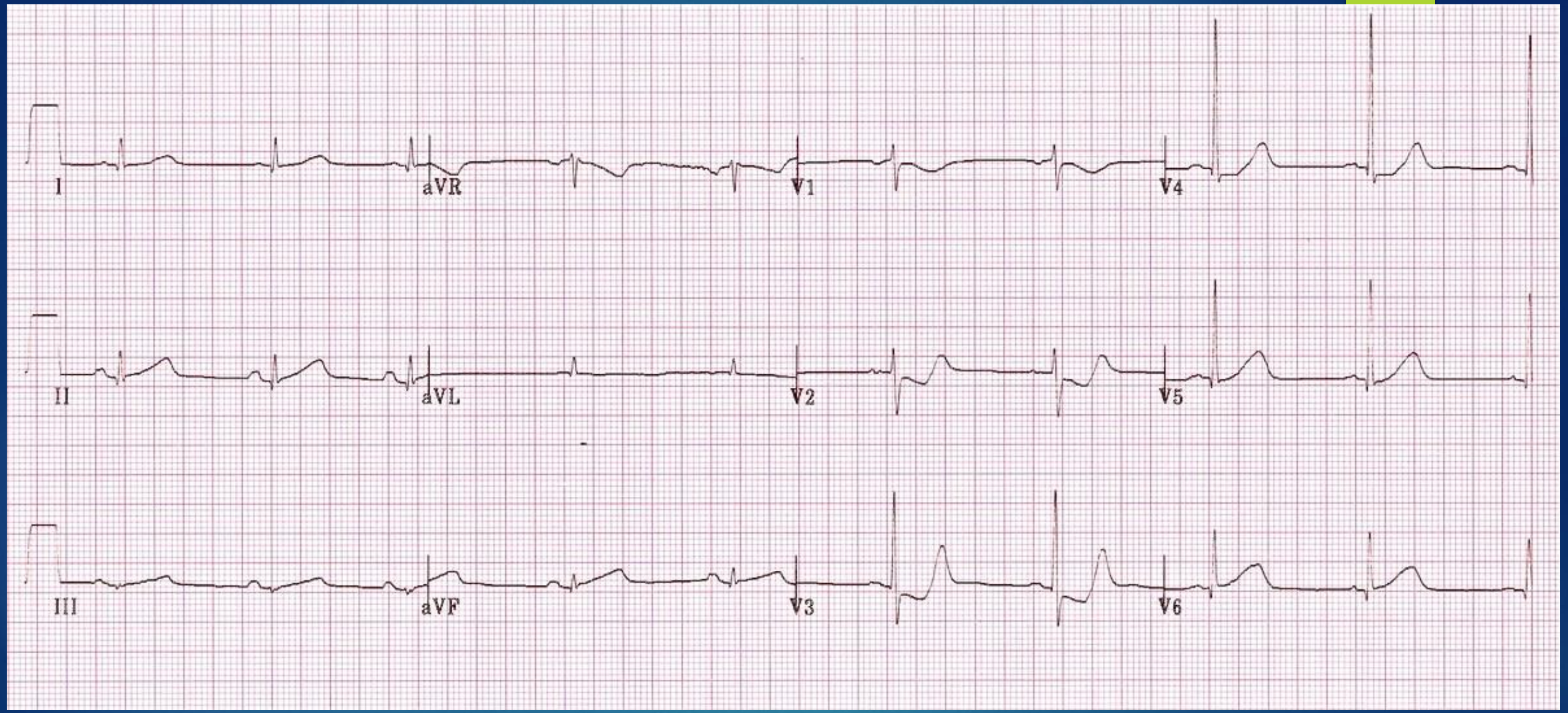


52yo male with central chest pain



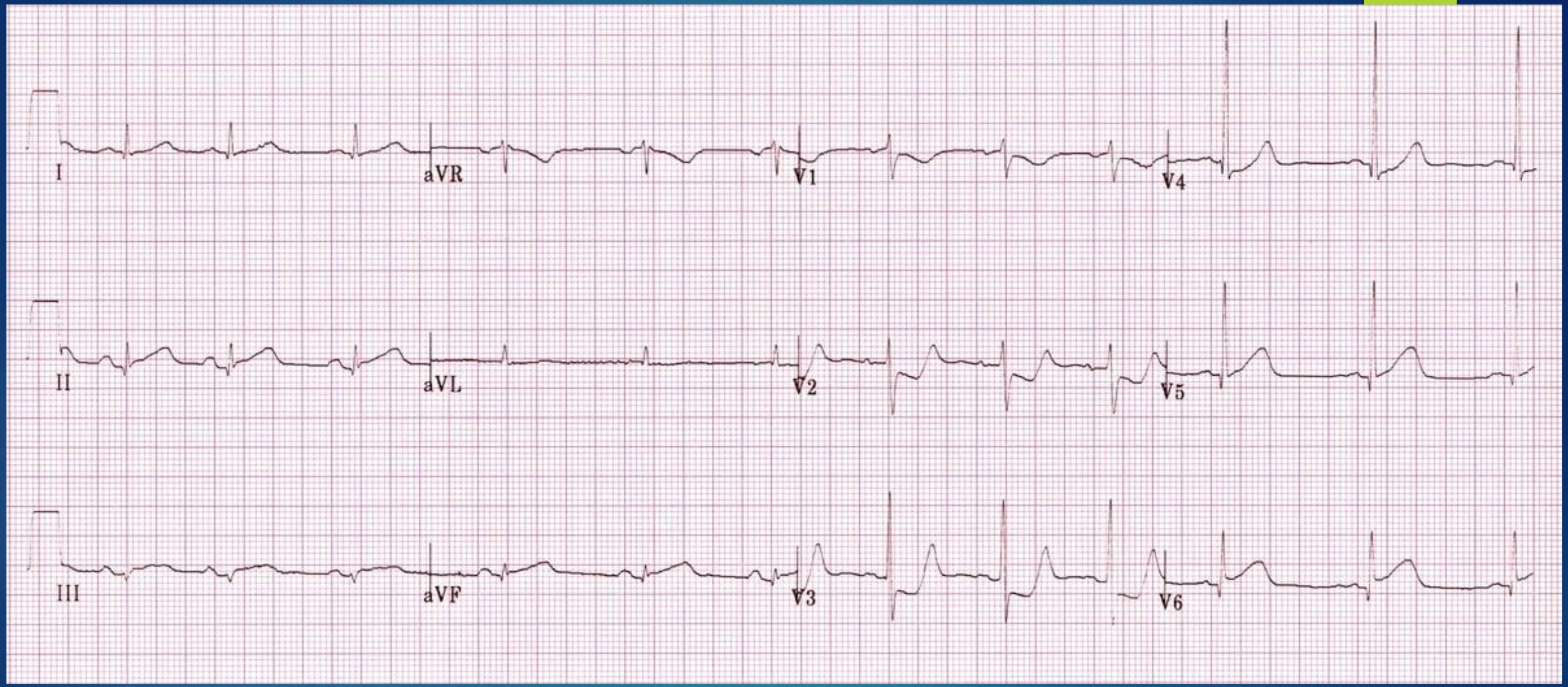
Posterior leads

Posterior MI

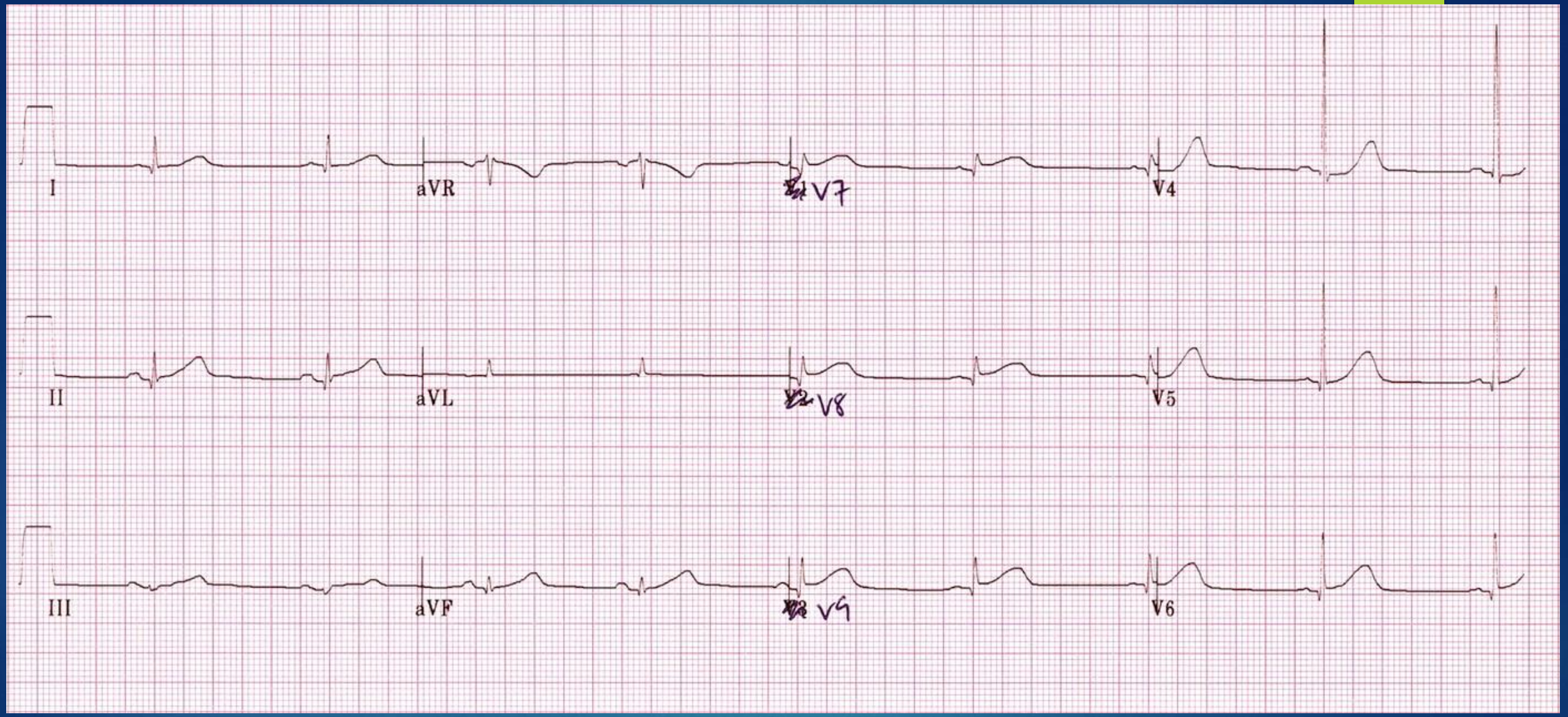


- Findings can be very, very subtle

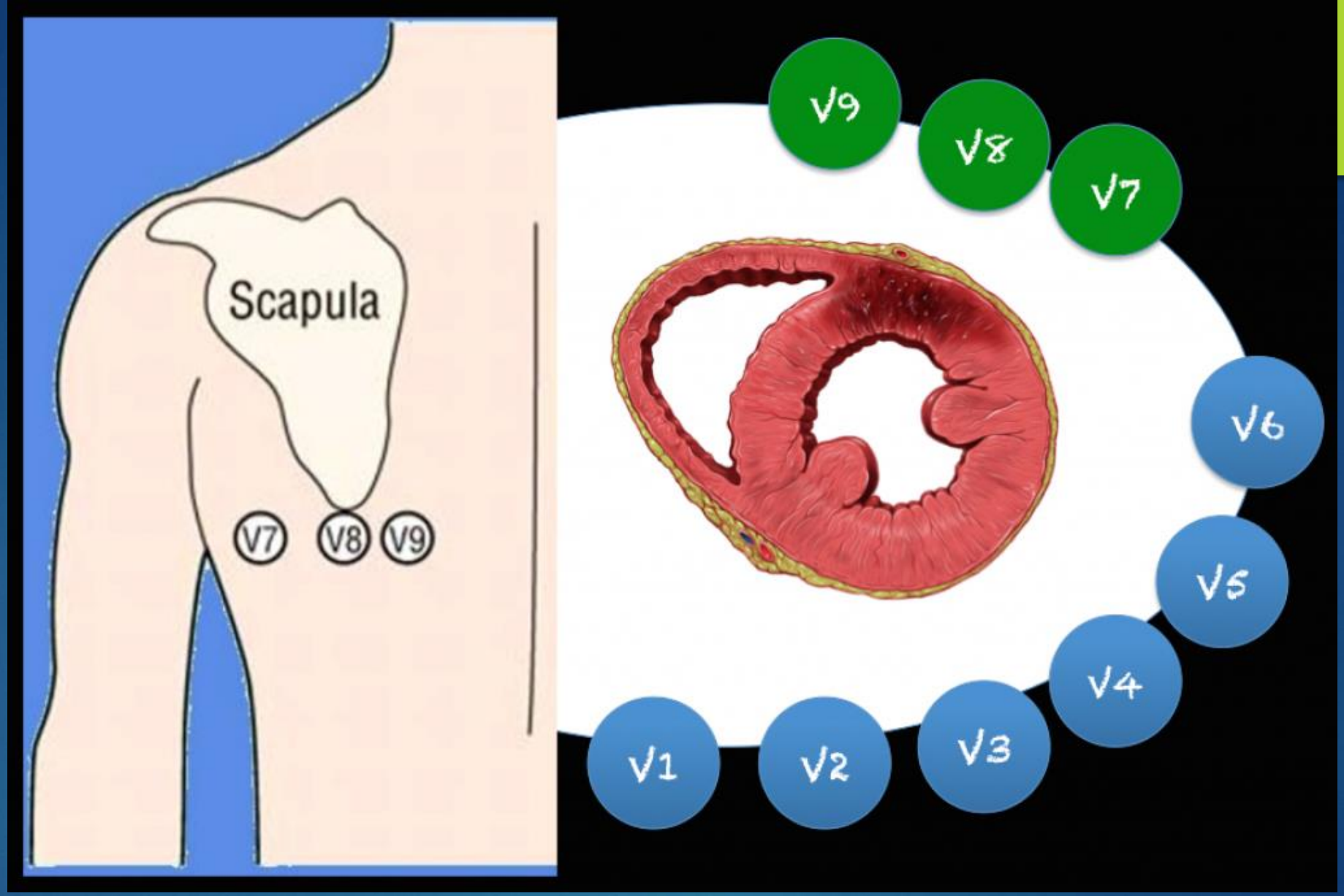
30mins later



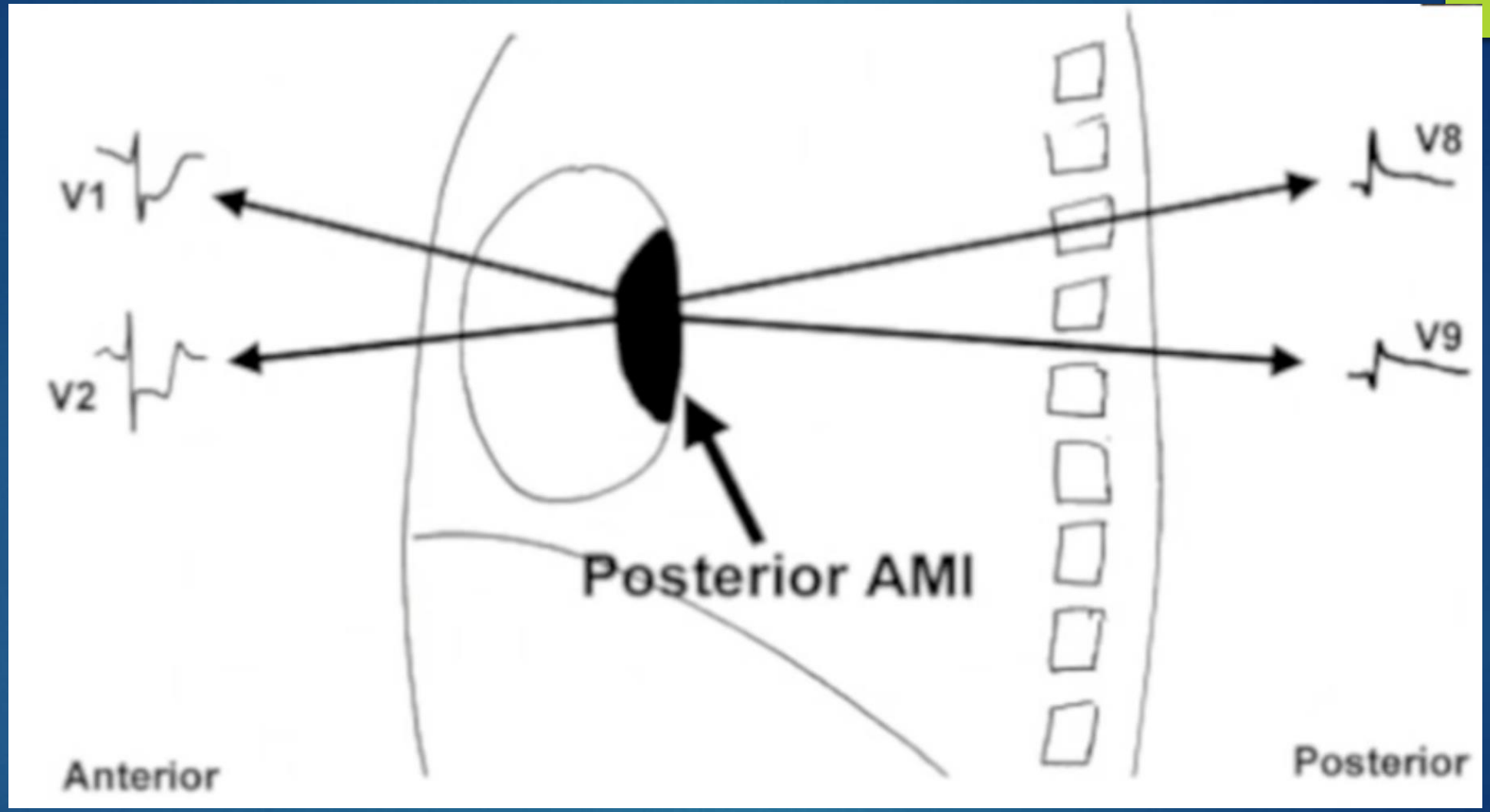
- There is now some ST elevation developing in V6.
- With an eye of faith there is perhaps also some early STE in the inf leads (lead III looks particularly abnormal)



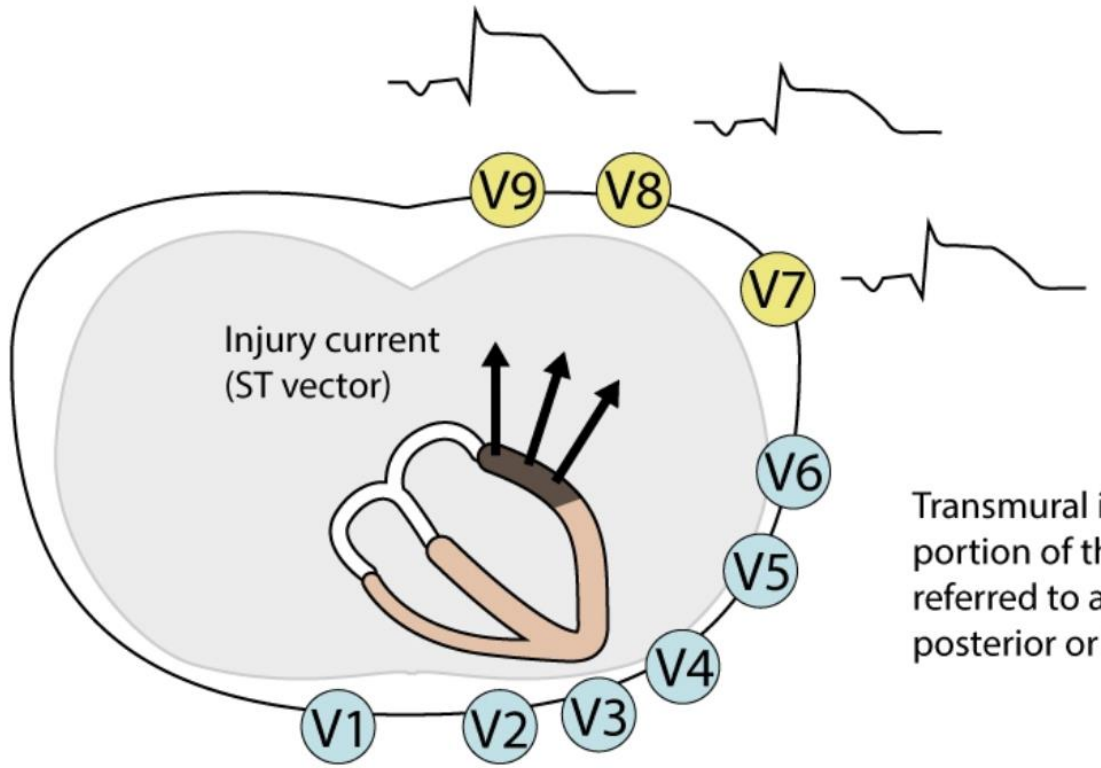
Posterior infarction is confirmed by the presence of STE >0.5mm in leads V7-9



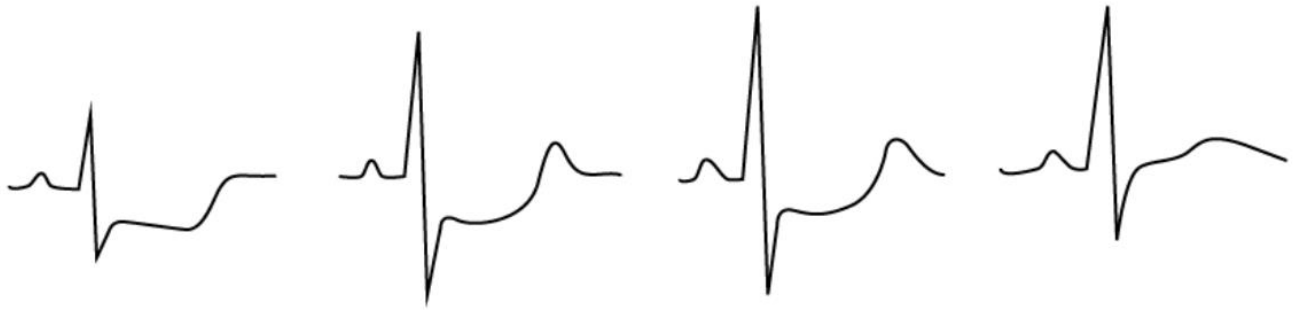
V7 – Left posterior axillary line, in the same horizontal plane as V6.
V8 – Tip of the left scapula, in the same horizontal plane as V6.
V9 – Left paraspinal region, in the same horizontal plane as V6.


V1
V2
V8
V9
Posterior AMI
Anterior
Posterior

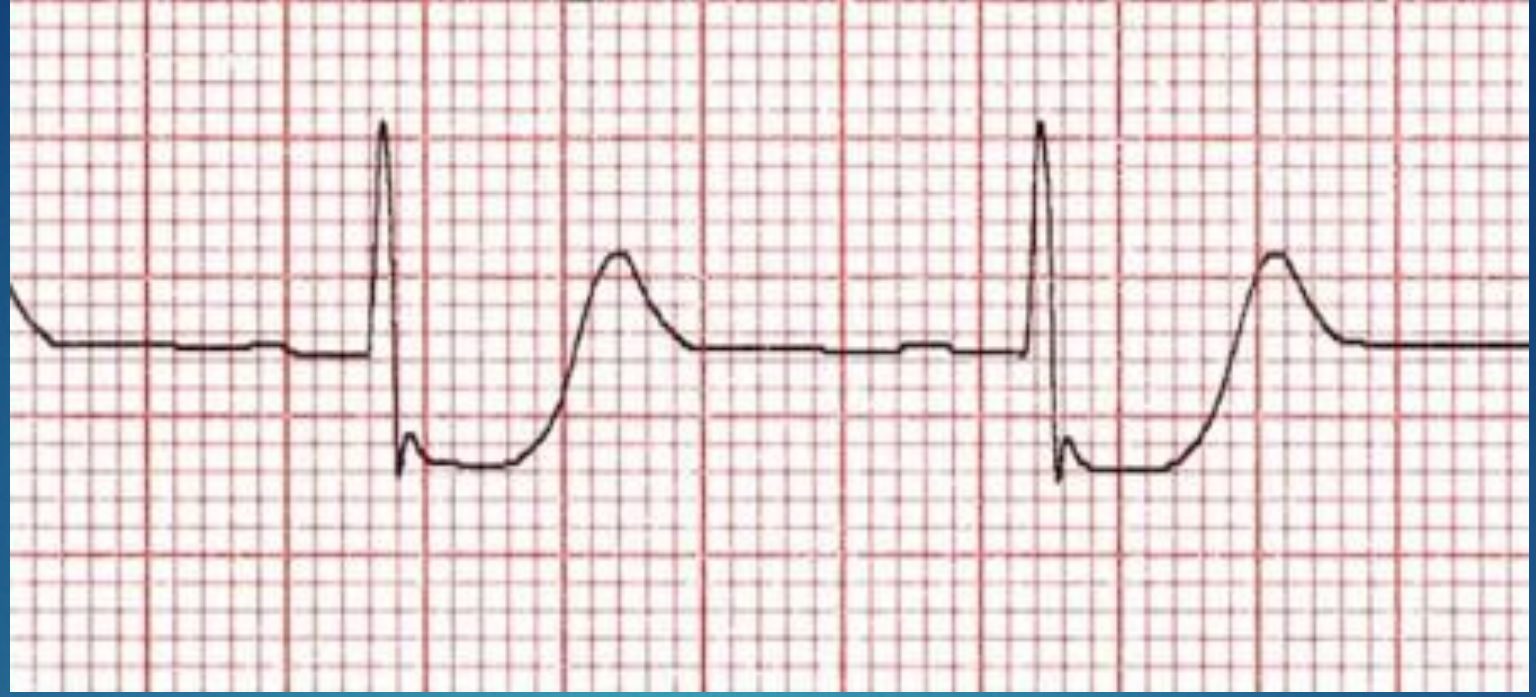
Posterolateral (posterior, inferobasal) transmural ischemia

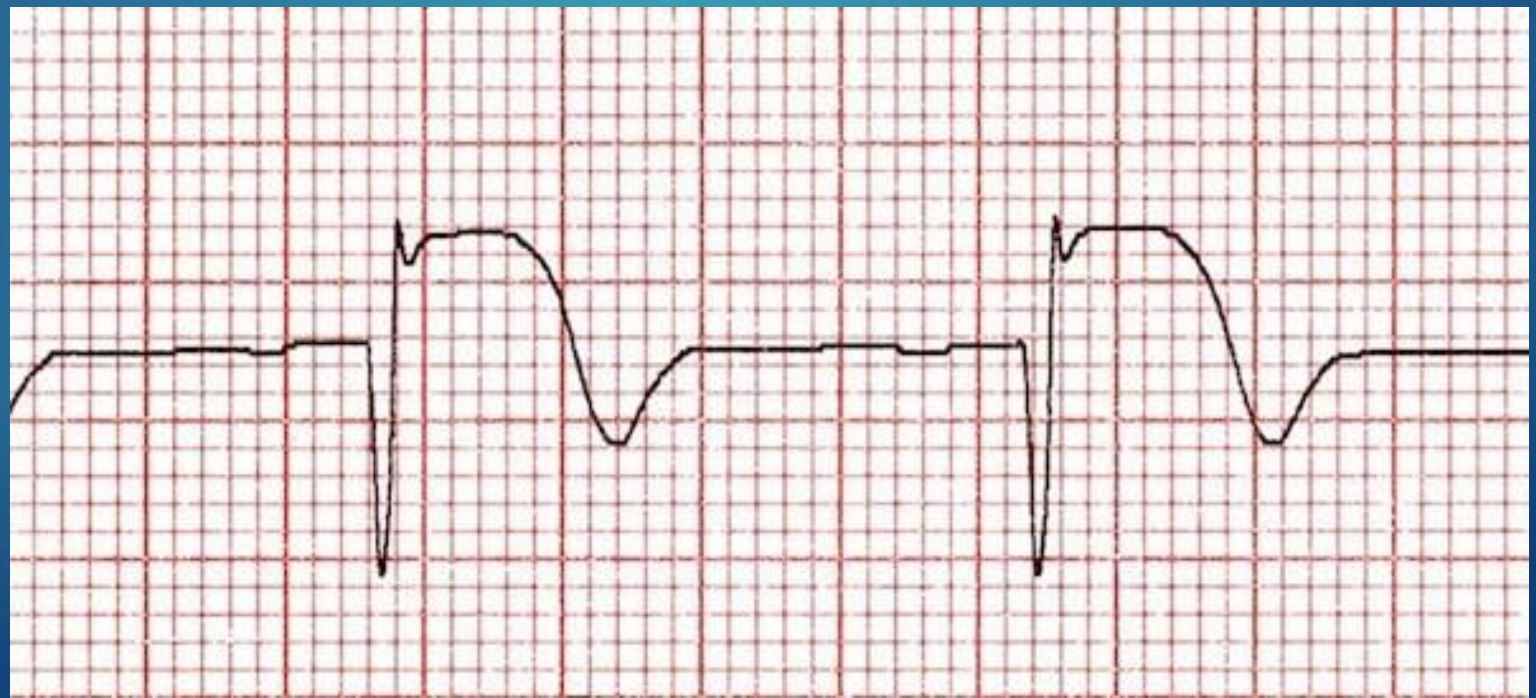


Transmural ischemia in the basal portion of the lateral wall. This is referred to as posterolateral, posterior or inferobasal ischemia.



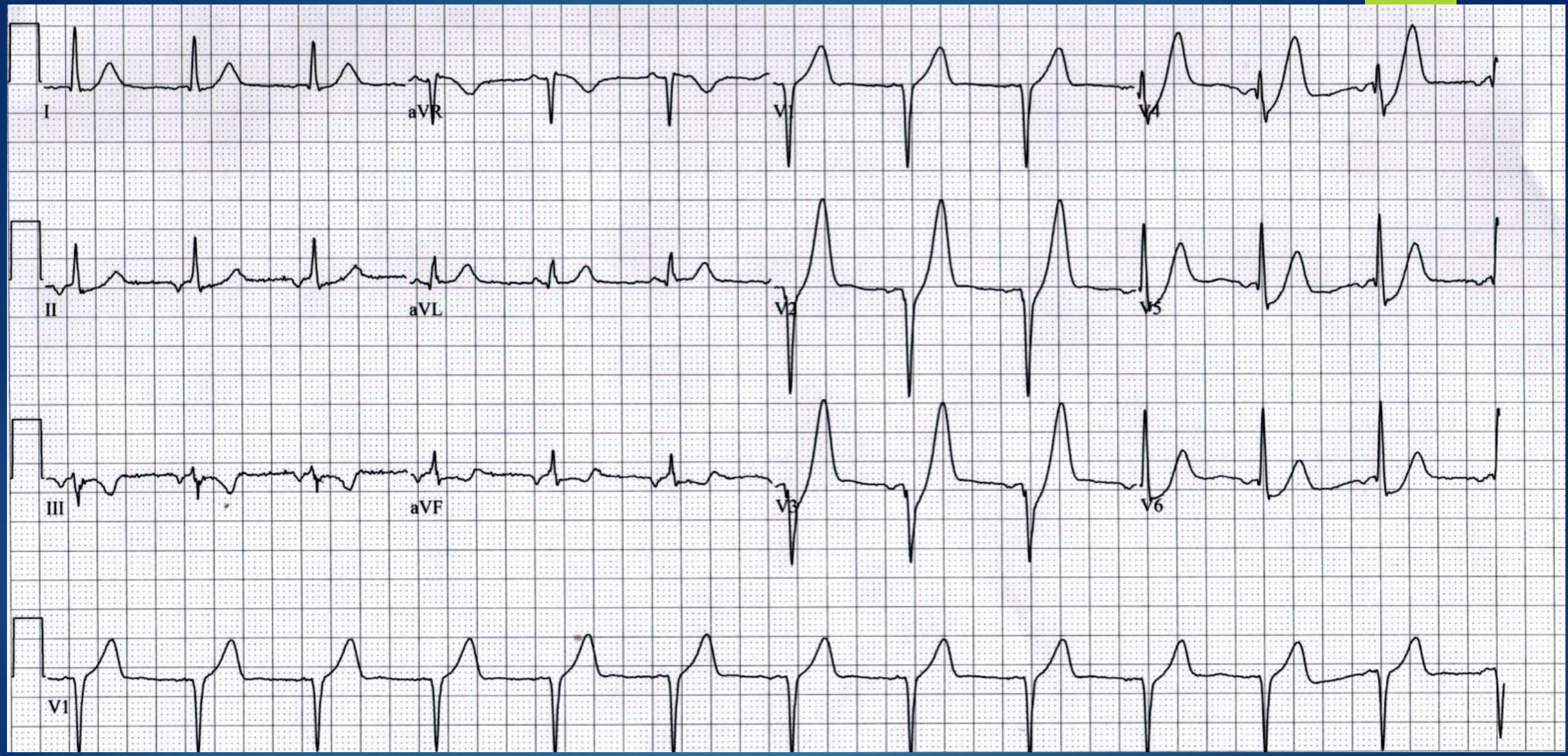
Reciprocal ST-segment depressions





- 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

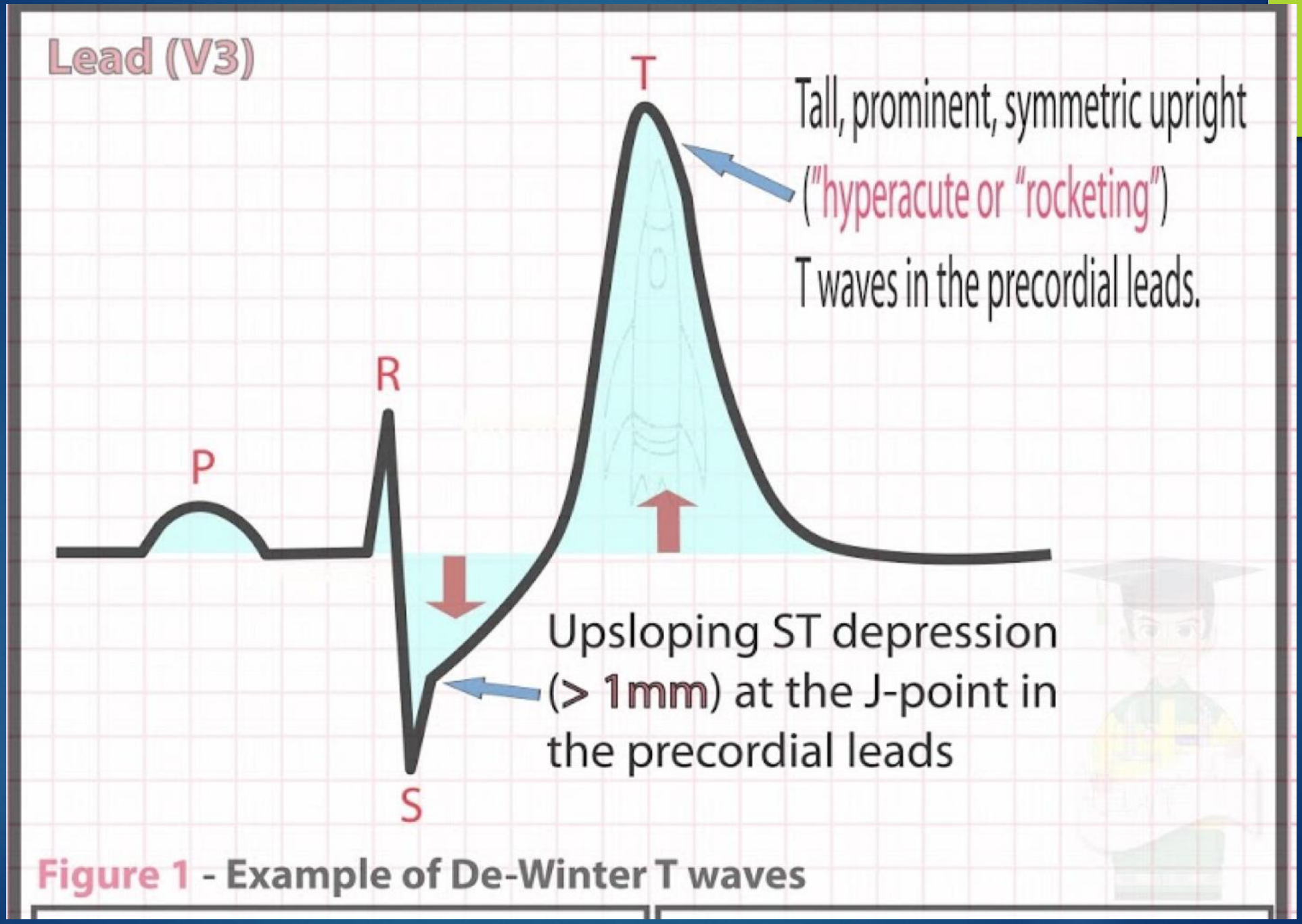
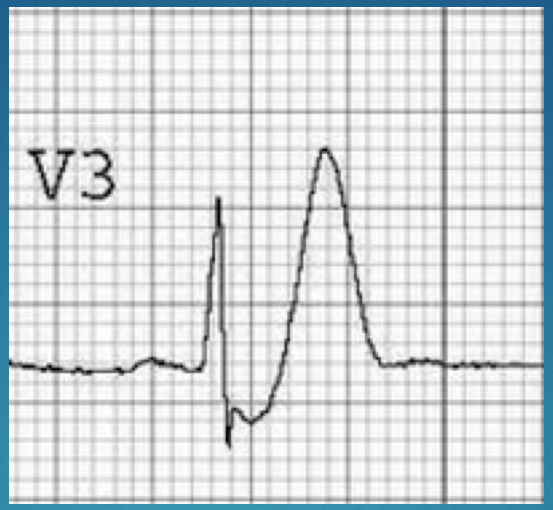


Figure 1 - Example of De-Winter T waves



- De Winter ECG pattern is an *Anterior STEMI equivalent*
- Presents without obvious ST segment elevation
- Key diagnostic features include ST depression and prominent T waves in the precordial leads

Rate	74
PR	166
QRSd	110
QT	427
QTc	474

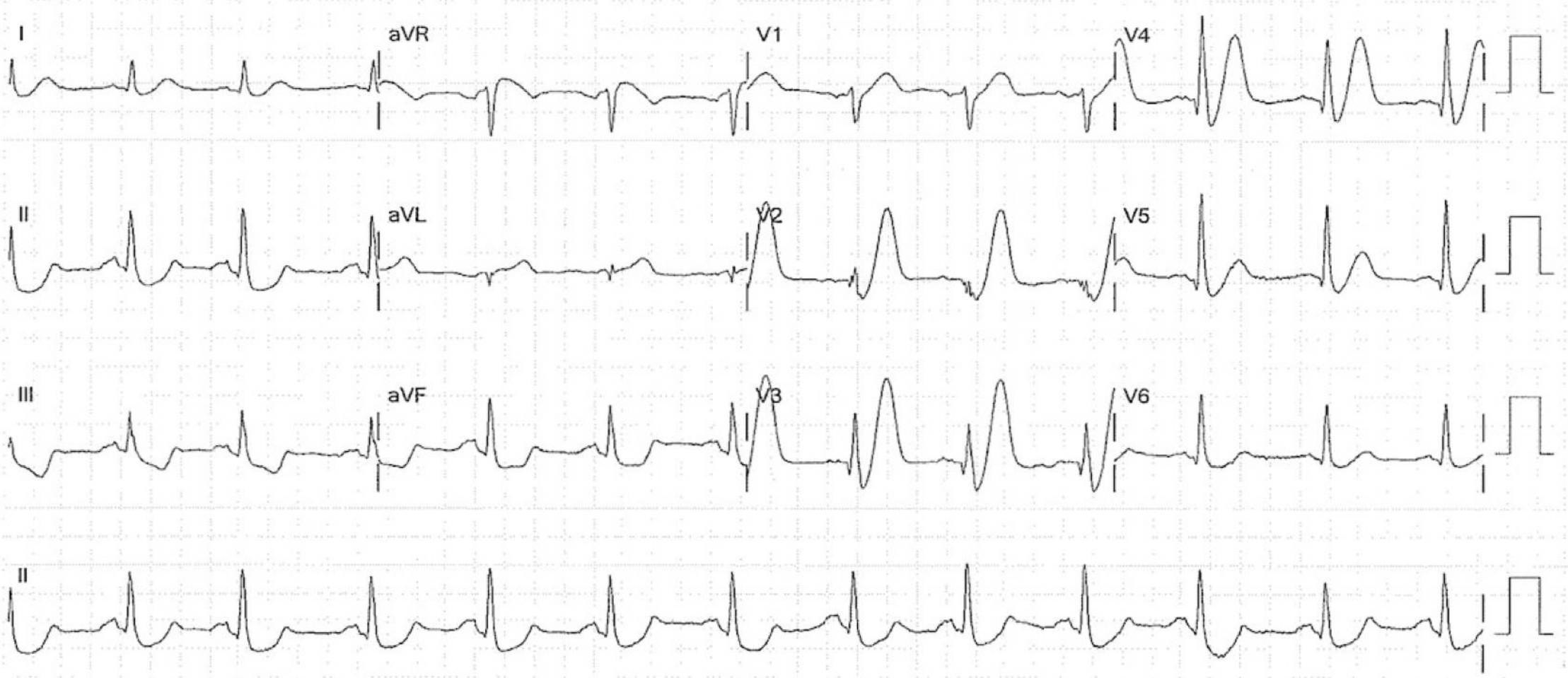
Axes	
P	77
QRS	66
T	-61

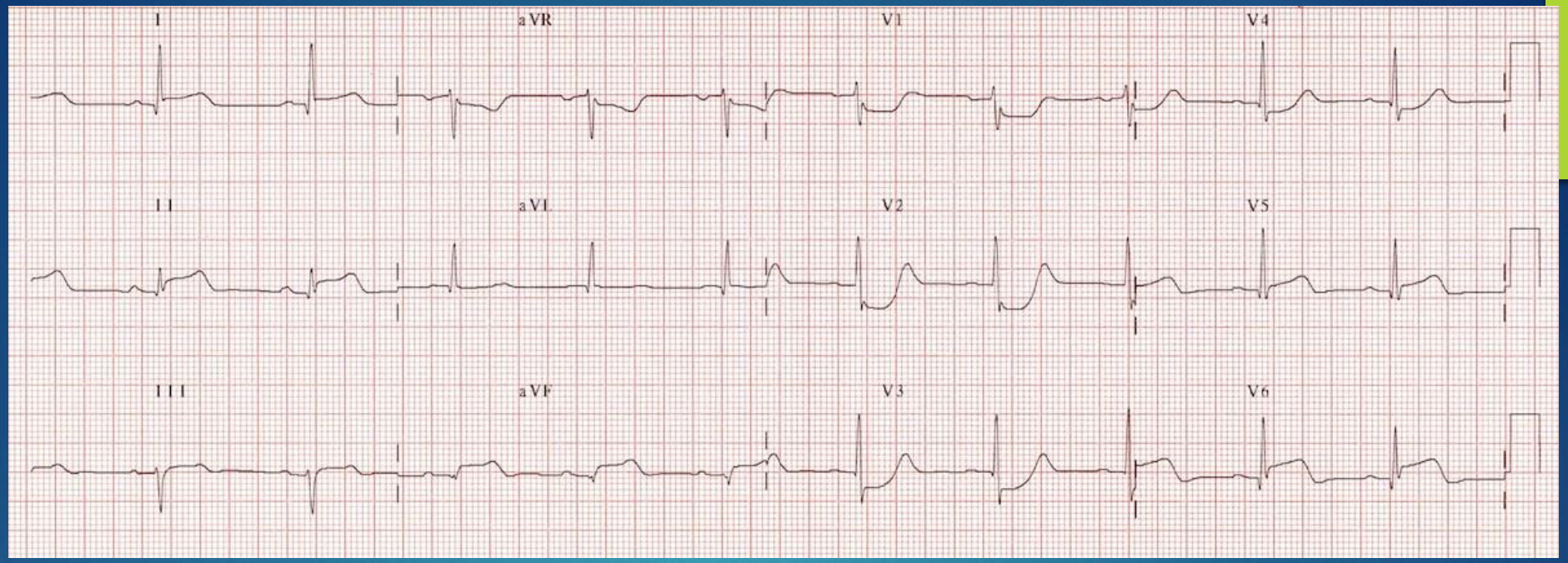
- Sinus rhythm ---------- normal P axis, V-rate 50- 99
- Repol abnrm, severe global ischemia (LM/MVD) ---------- STe aVR, STd & Tneg, ant/lat/inf
- Tall T, consider metabolic/ischemic abnrm ---------- T >1.2mV
- >>> Acute Ischemia <<<

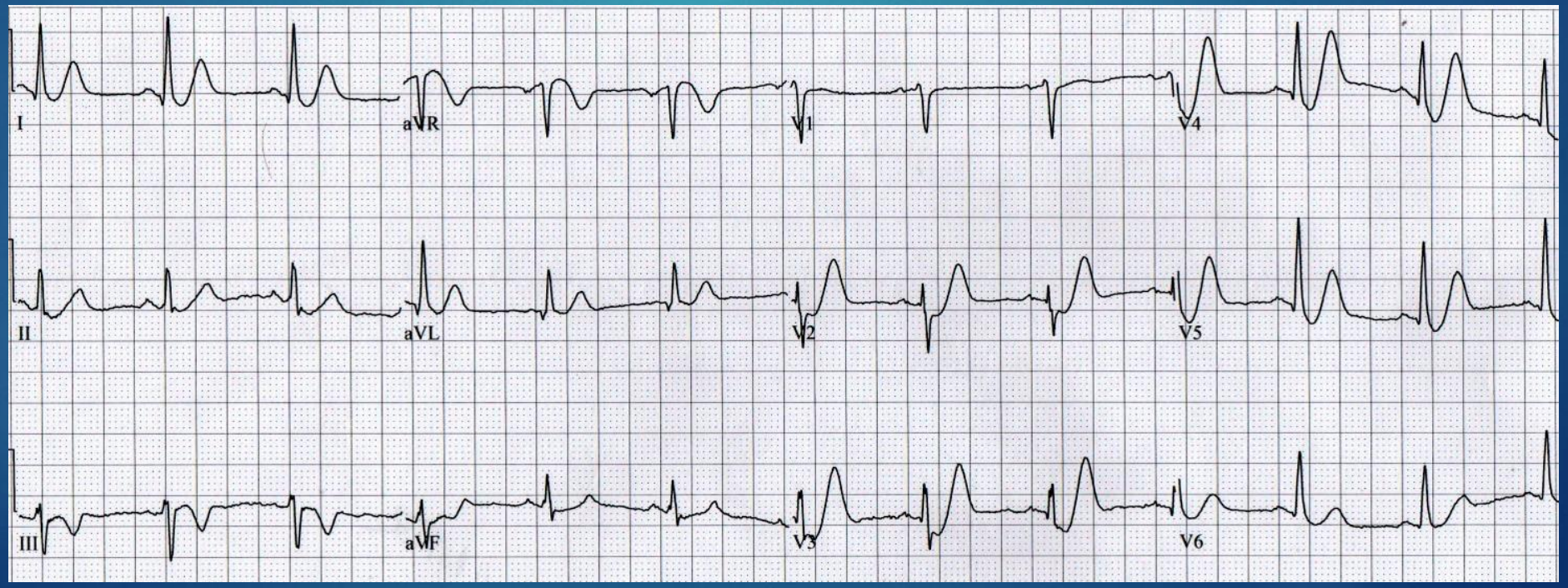
Requested by:

- ABNORMAL ECG -

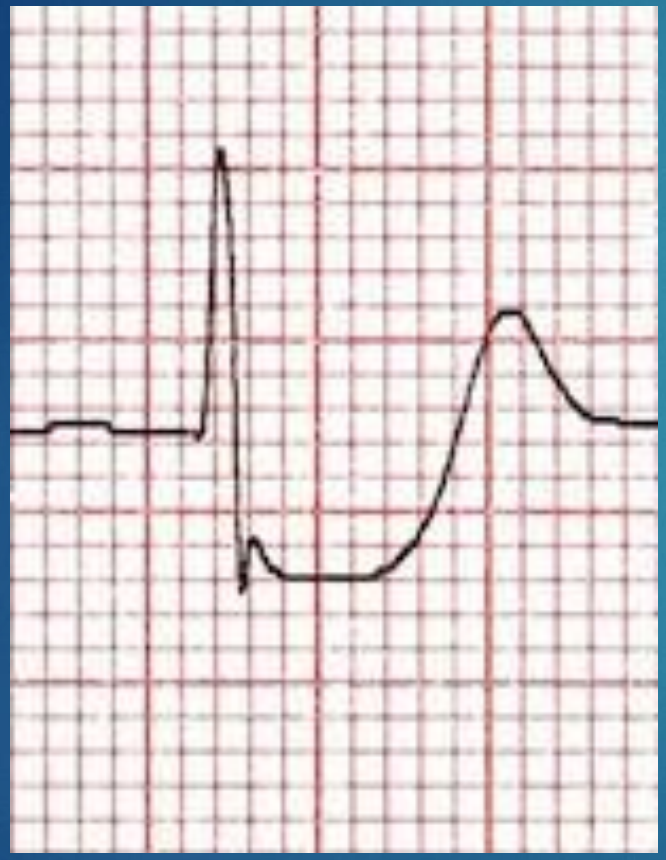
Unconfirmed diagnosis

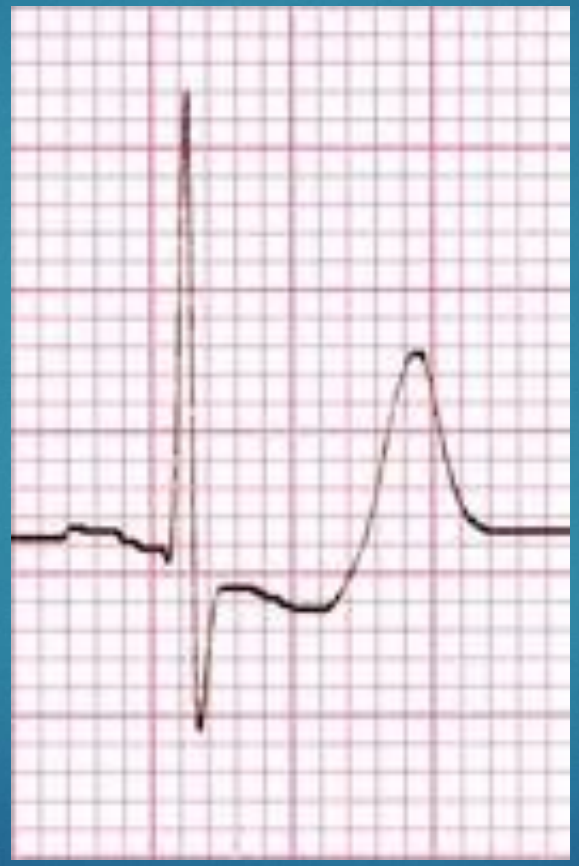


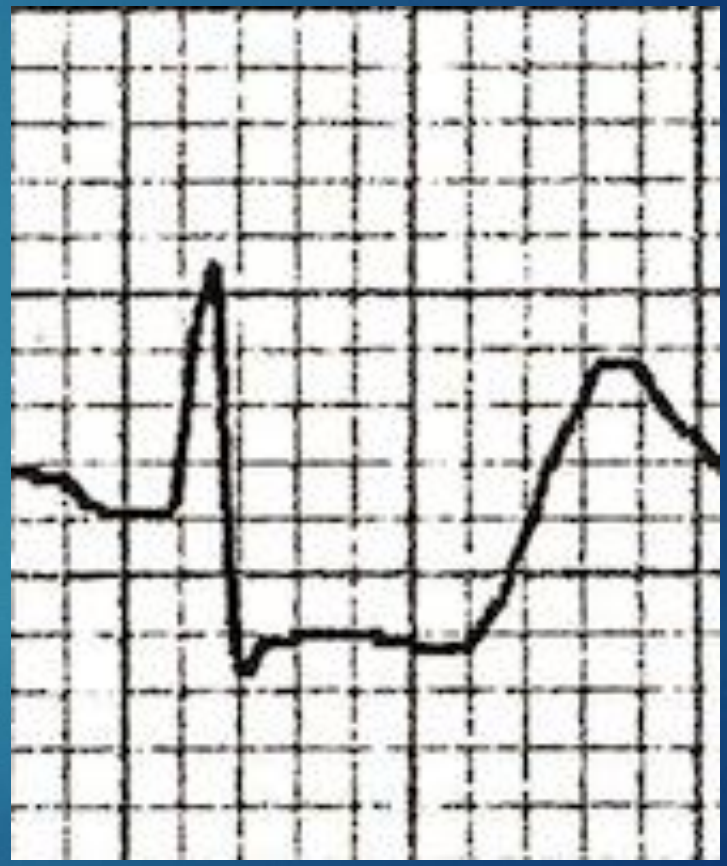

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

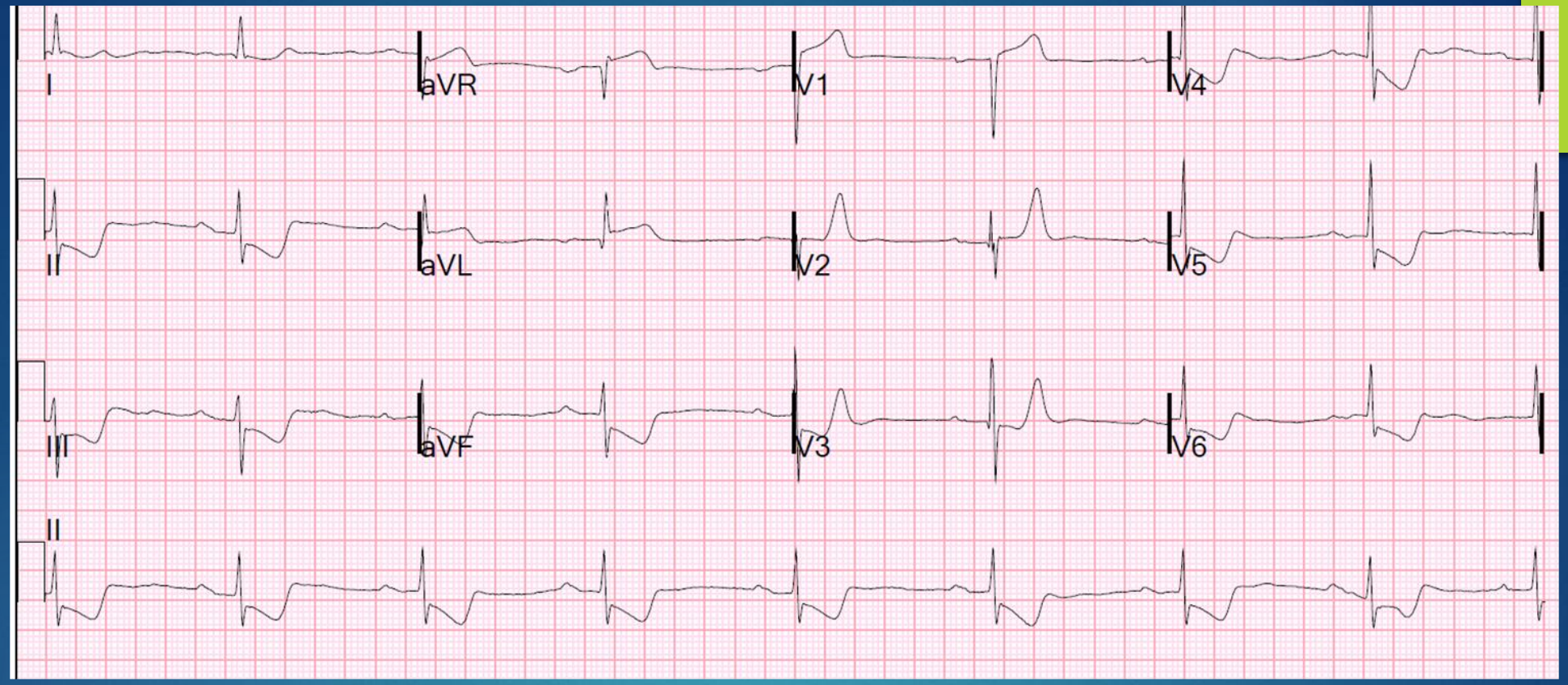

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

ST segment morphology in post MI








I
aVR
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II
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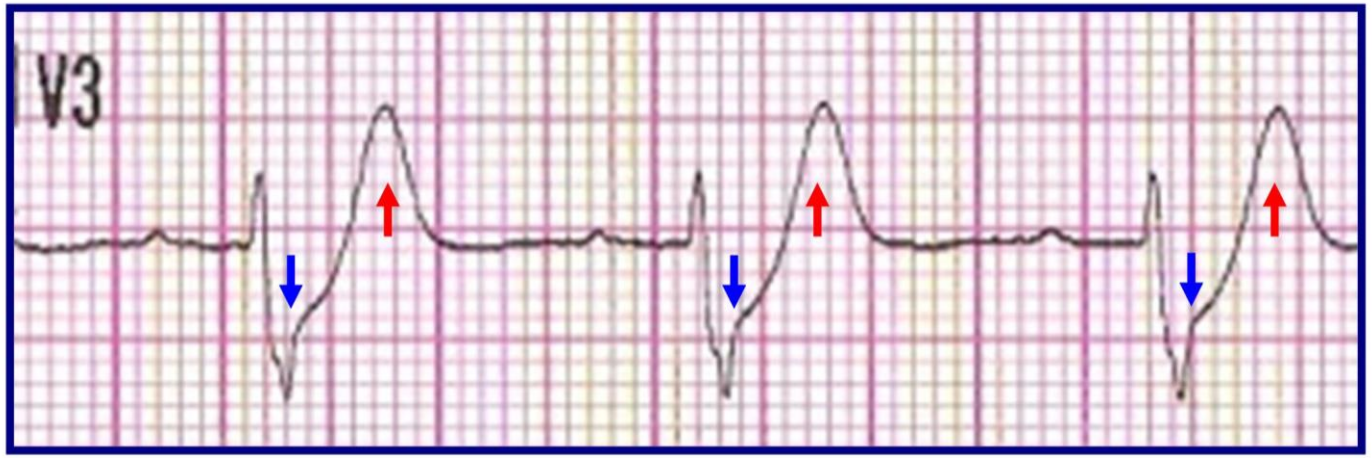
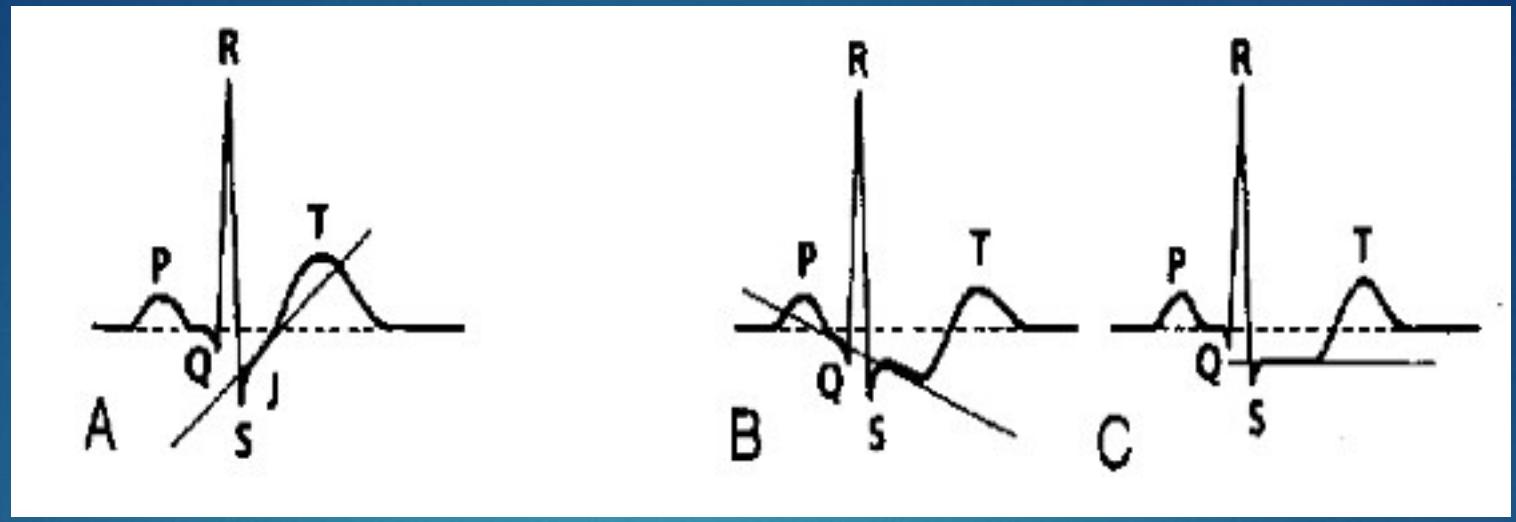
V2
V5
III
aVF
V3
V6
II


V3


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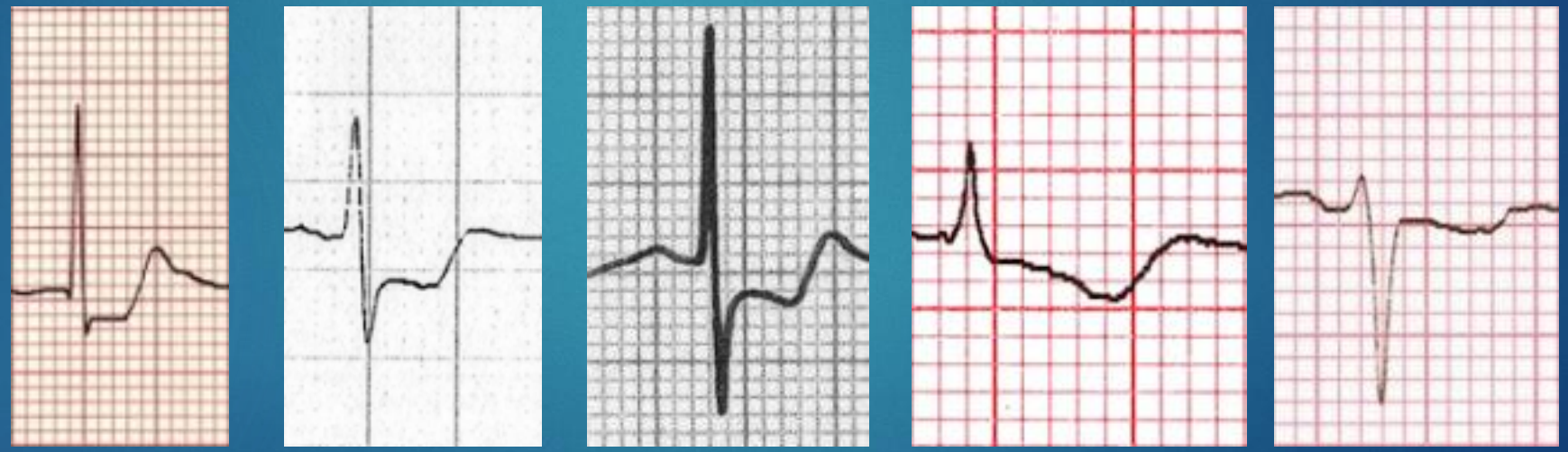
Q
S
B
R
P
T
Q
S
C



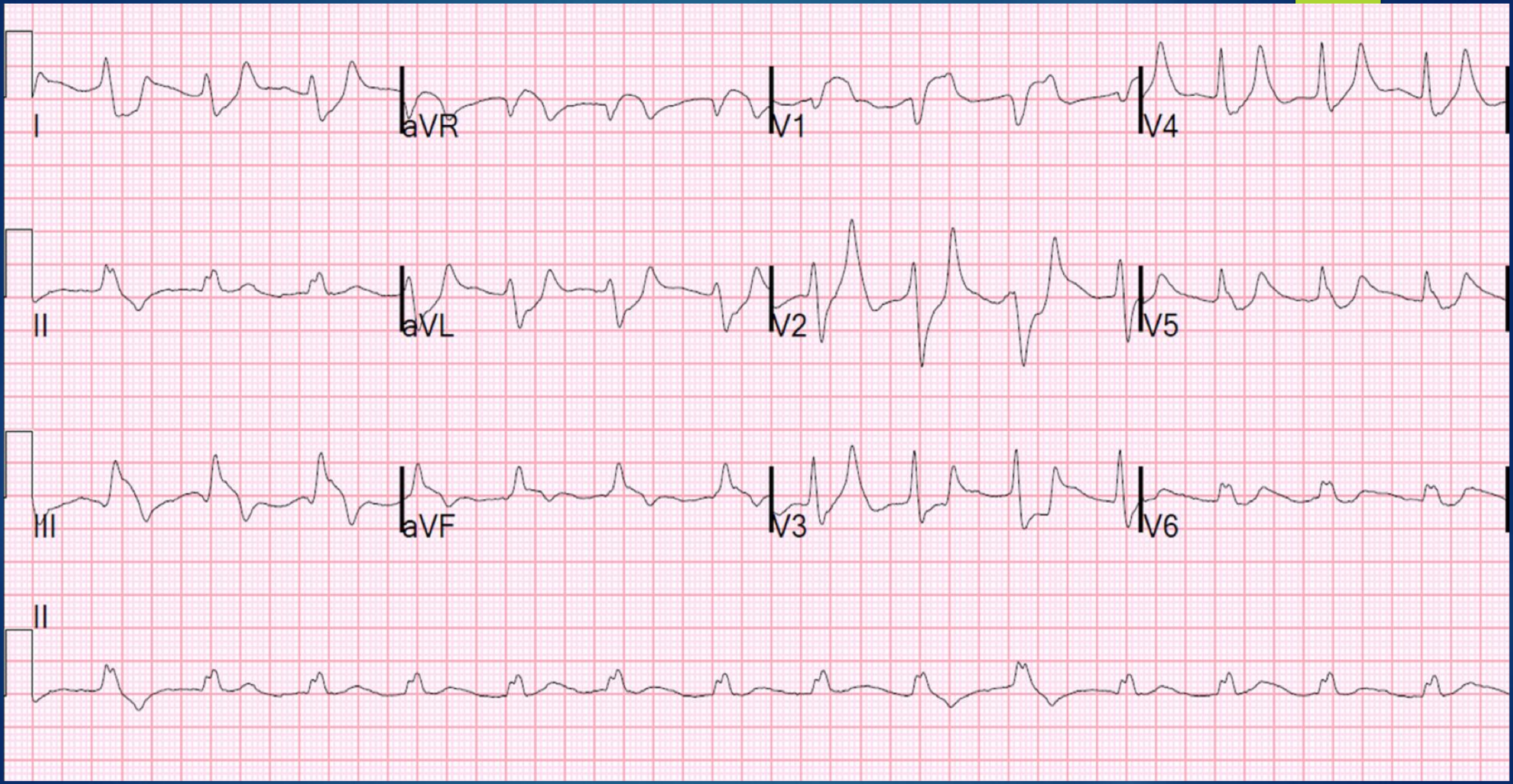
Posterior MI

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

De-Winter T waves

- Tall, prominent, symmetric, "rocketing" T waves in precordial leads
- Upsloping ST depression (>1mm) at the J-point in precordial leads
- STE (0.5-1mm) in aVR

Is this De-Winter or Post MI?


I
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III
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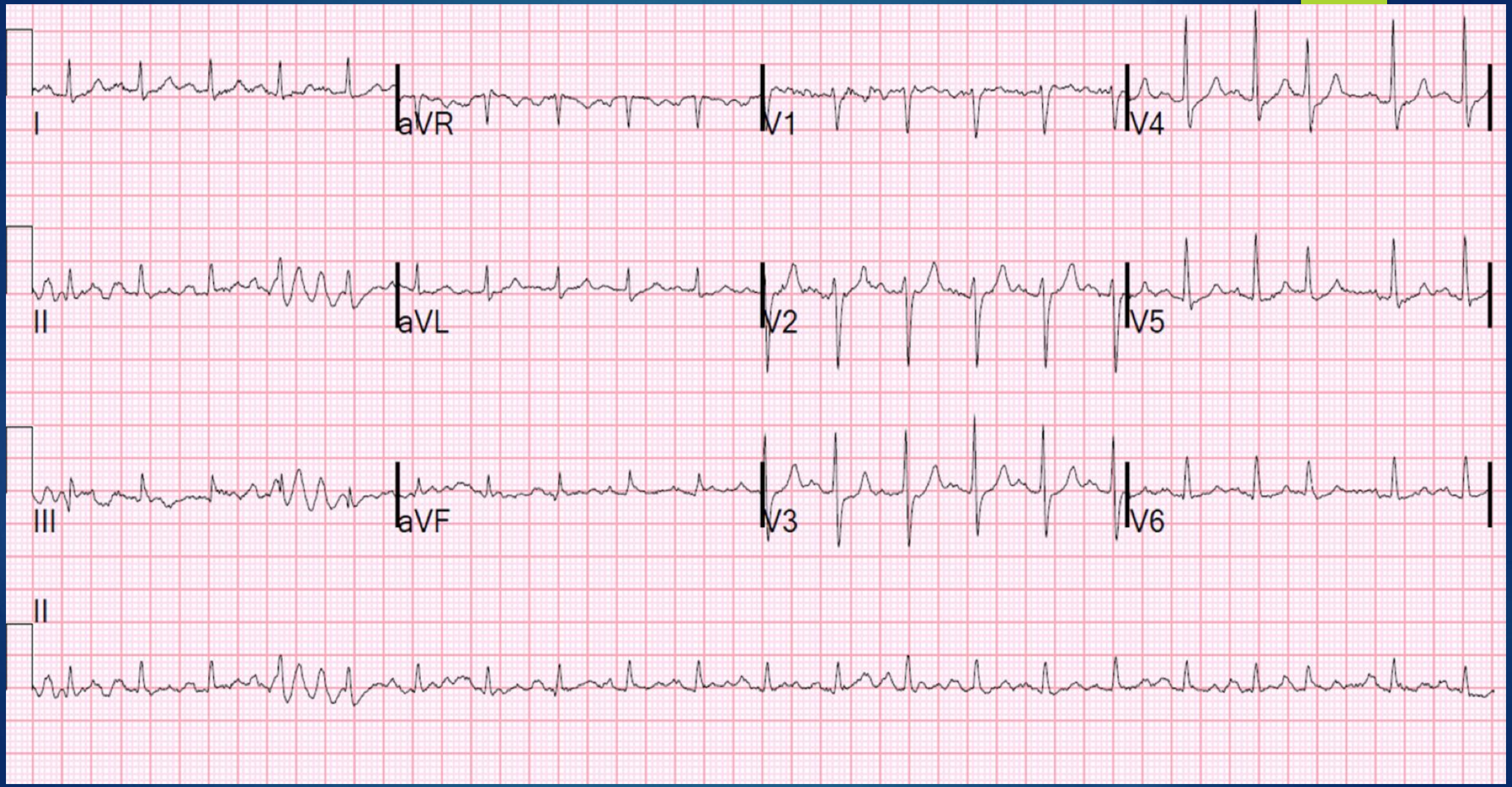
V3
V6
II


I
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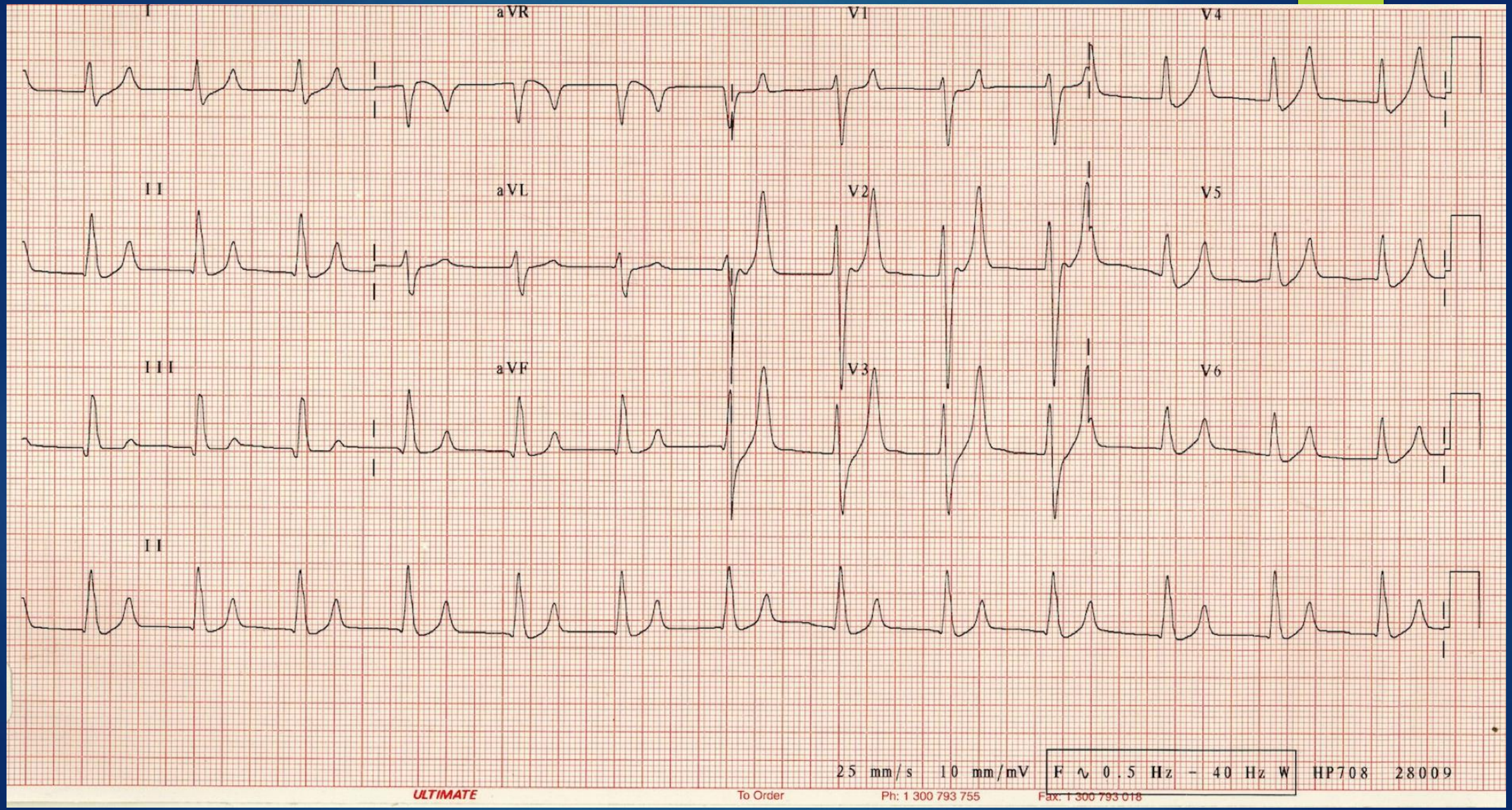
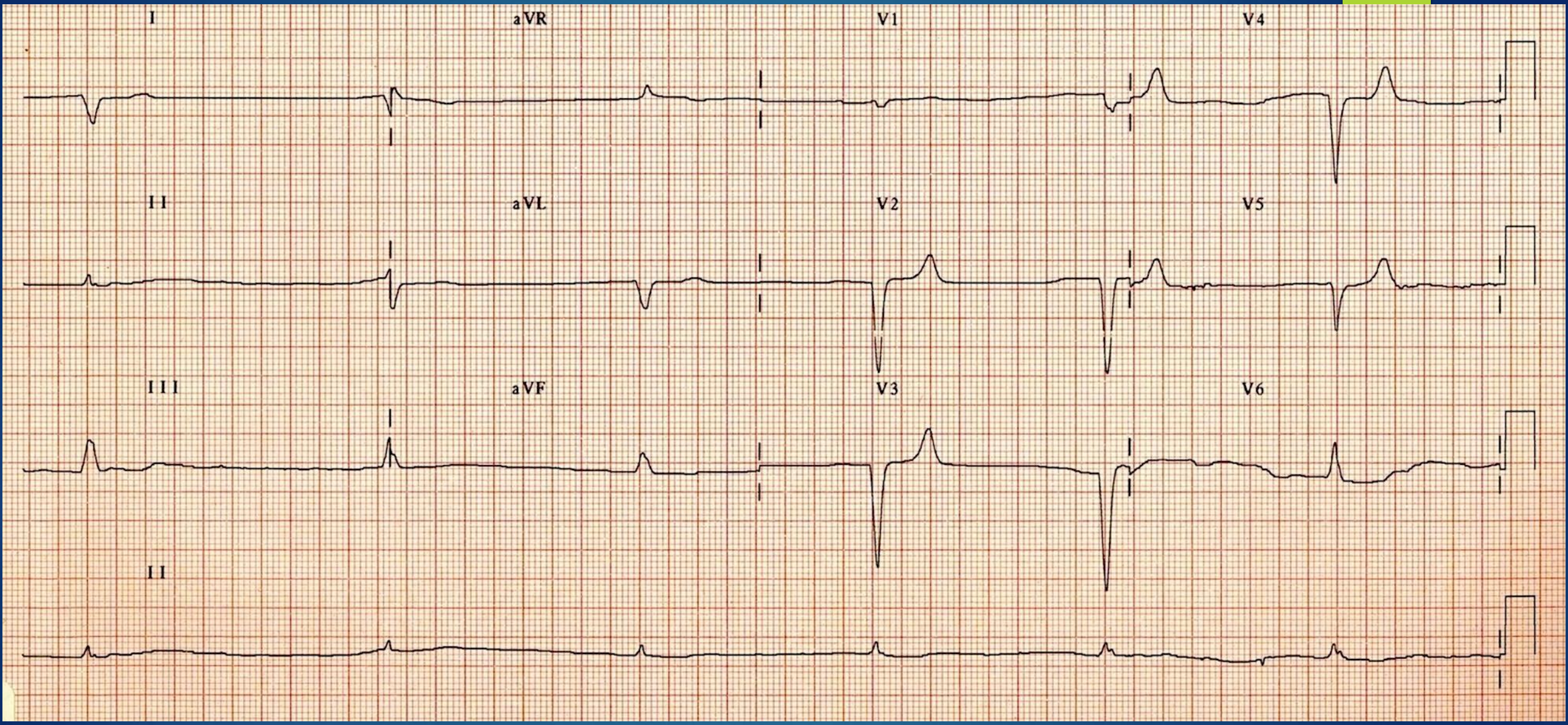

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s 10 mm/mV
F ∿ 0.5 Hz - 40 Hz W
HP708 28009
ULTIMATE
To Order
Ph: 1 300 793 755
Fax: 1 300 793 018


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

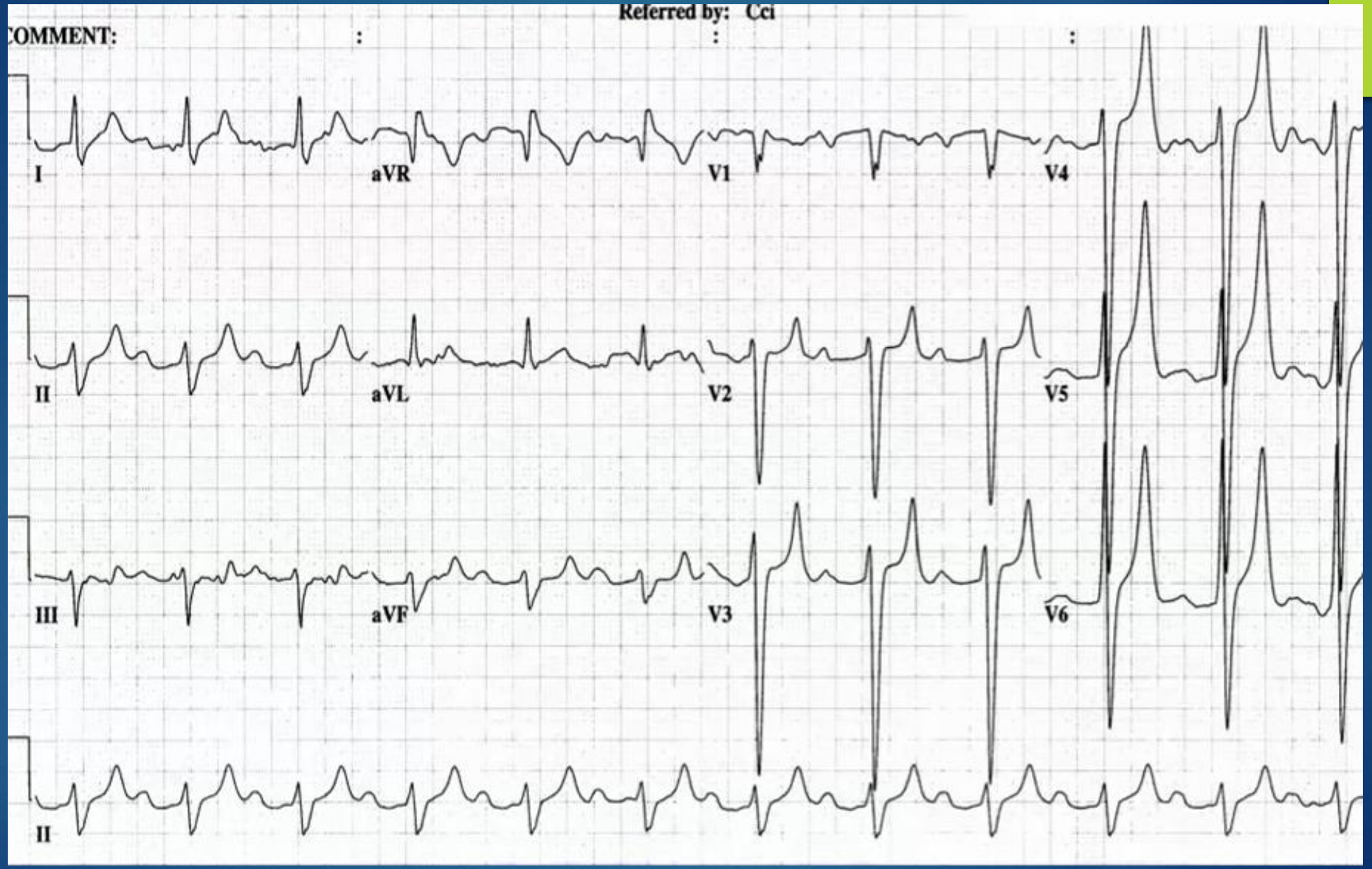
- Presented with APO after missing several dialysis sessions
- Cardiac arrest shortly after presenting
- K^+ 7.9

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

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
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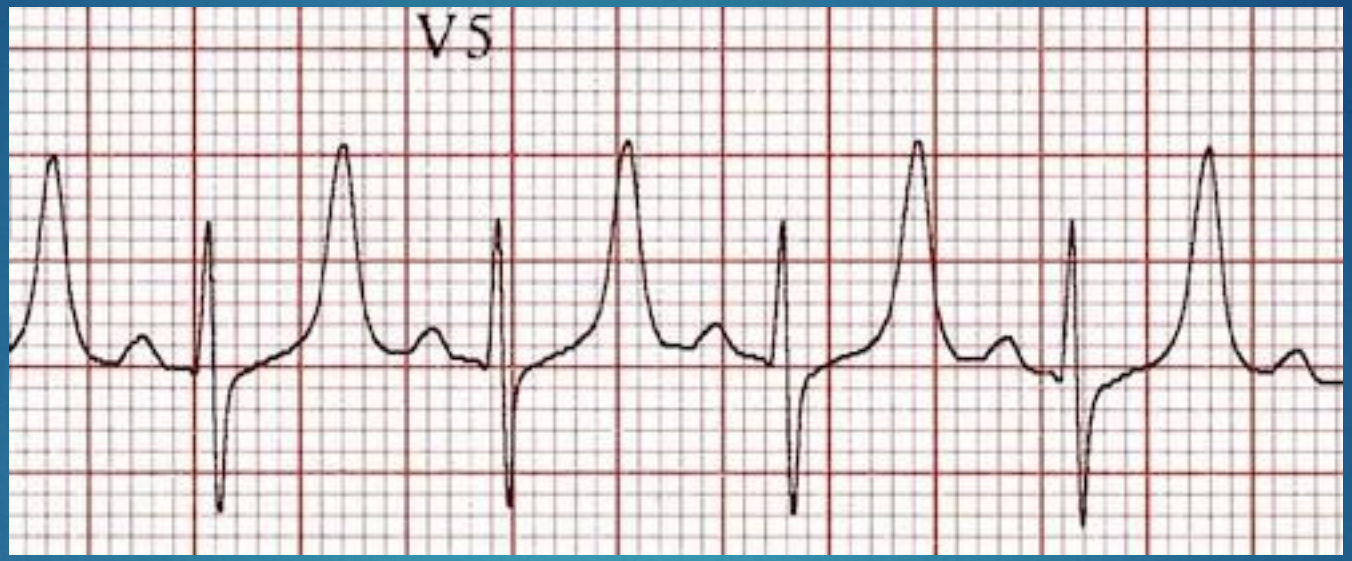
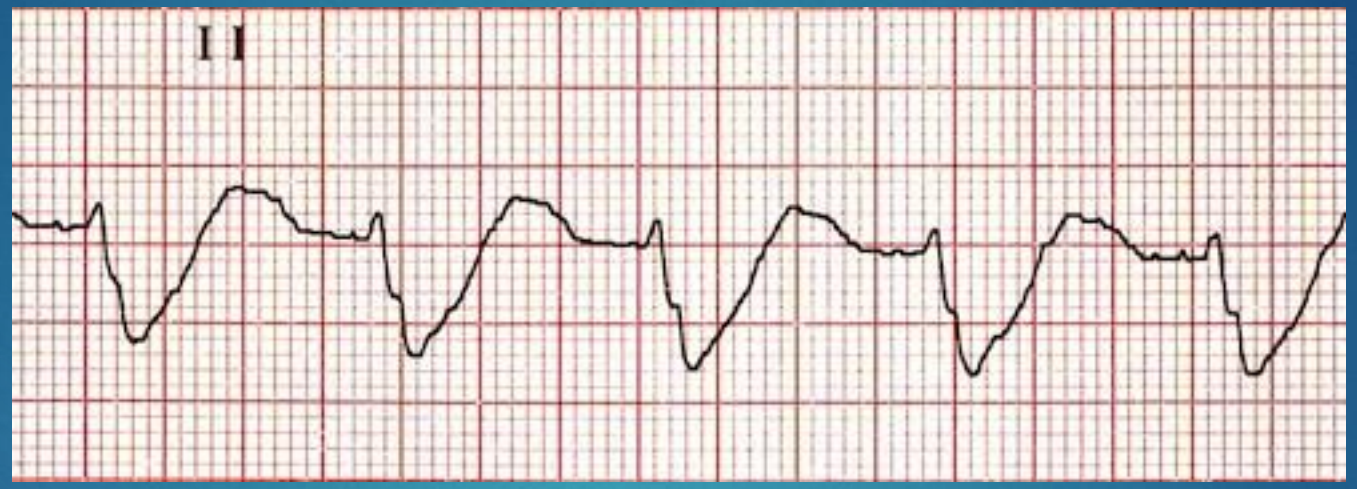
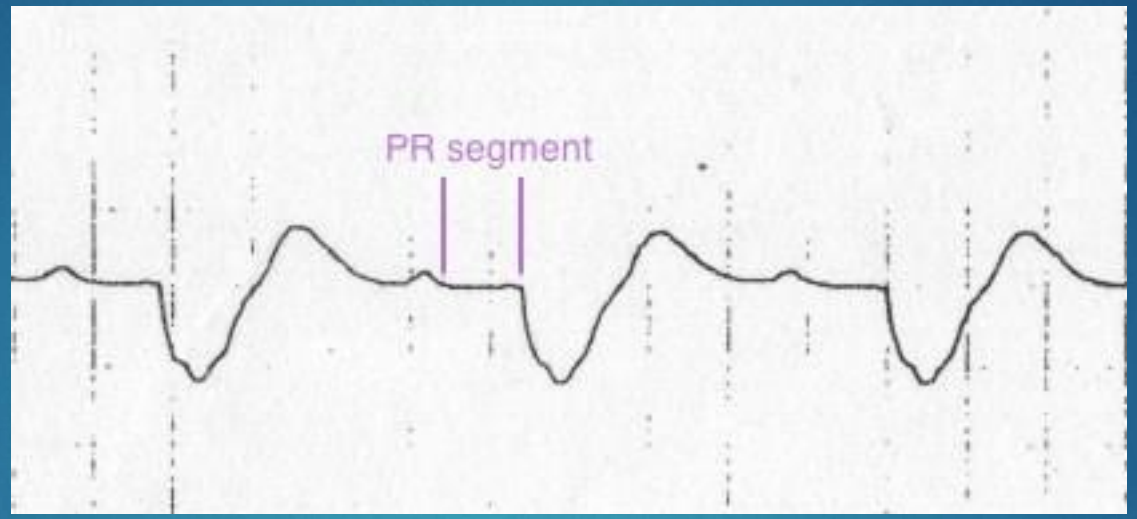
V2
V5
III
aVF
V3
V6
II



Peaked T waves



Broad QRS



Prolonged PR segment

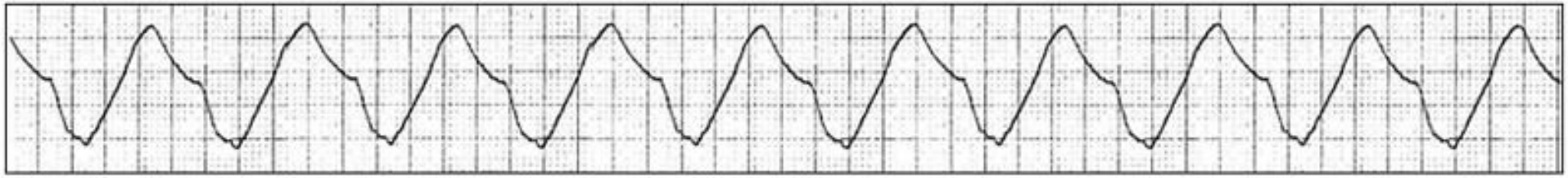
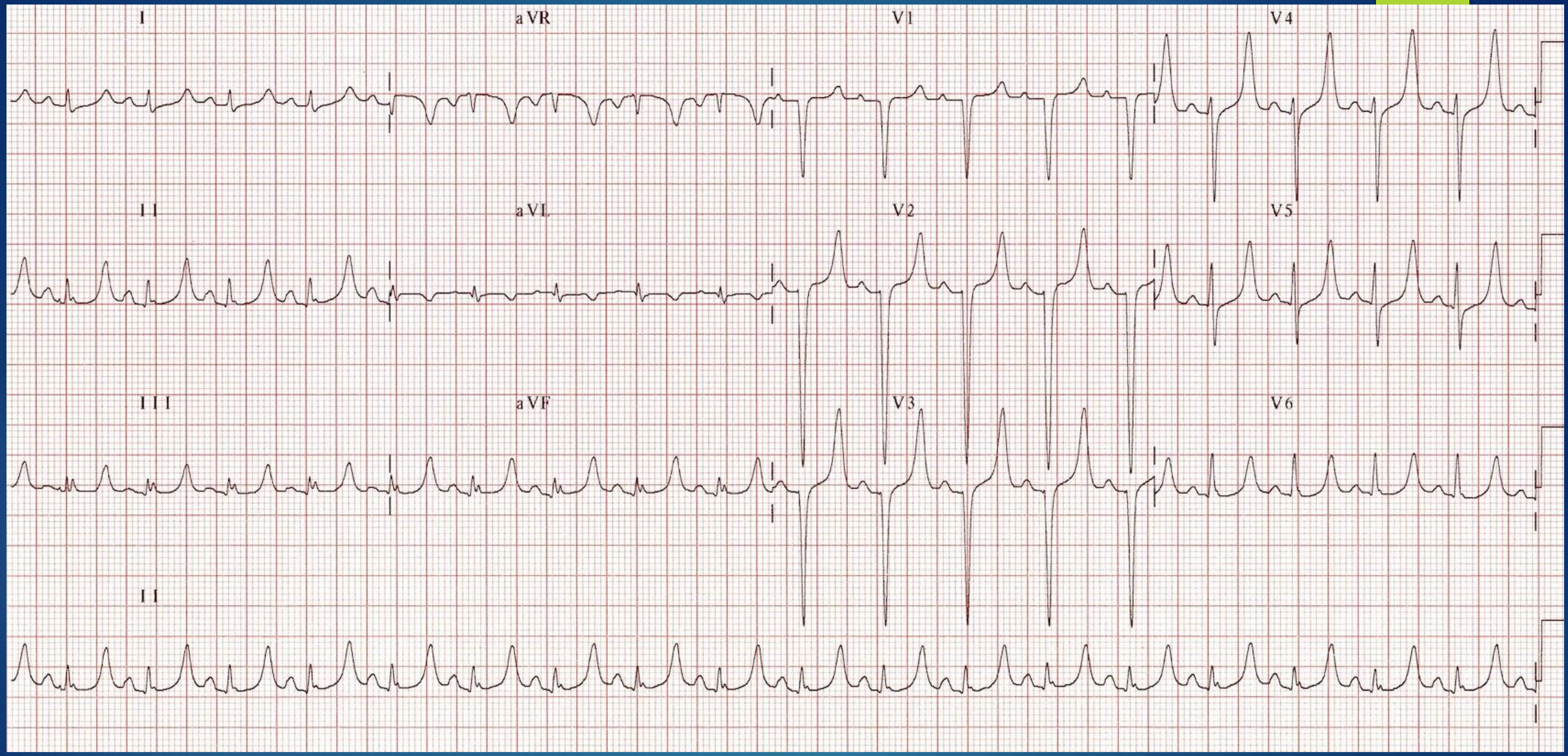
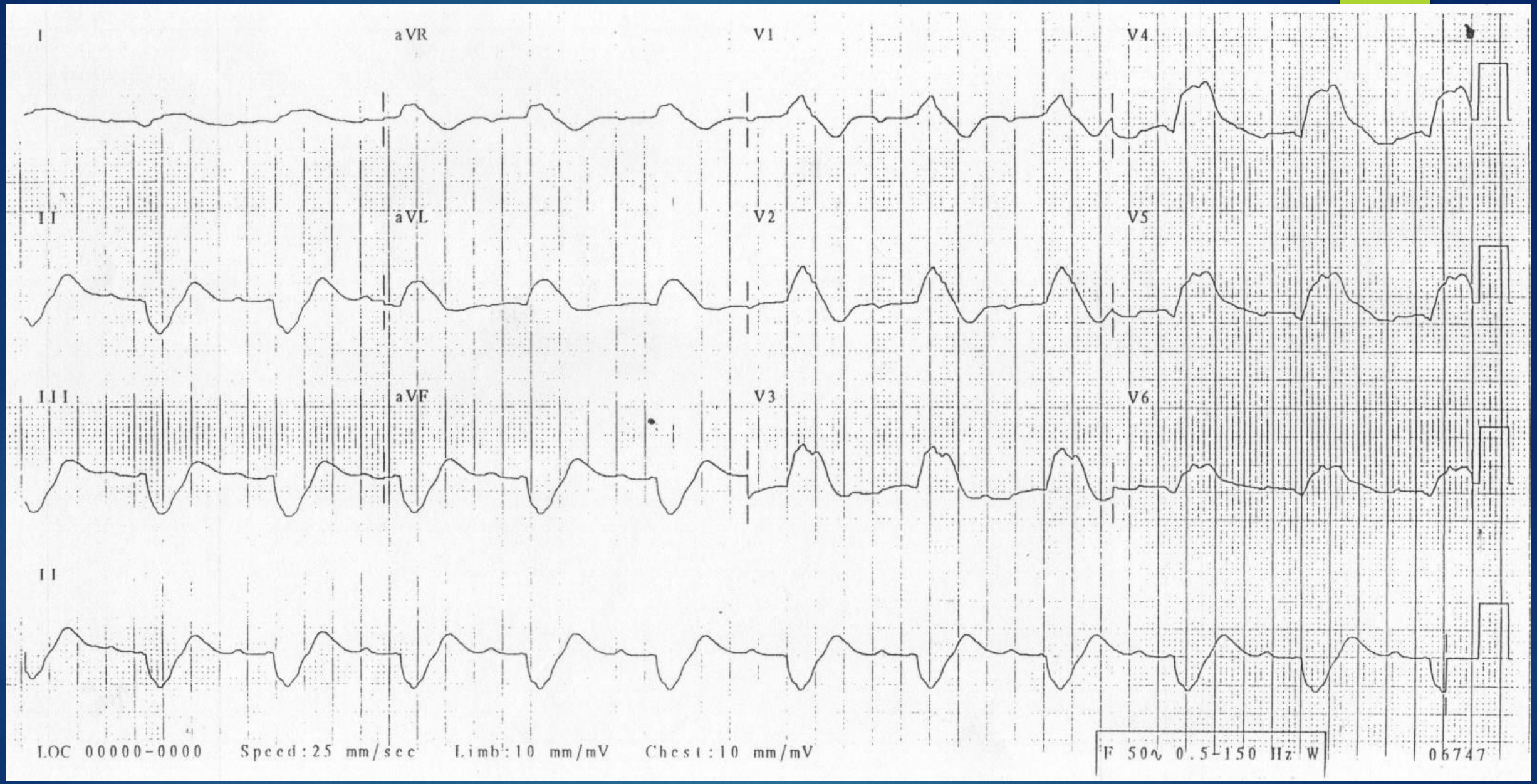


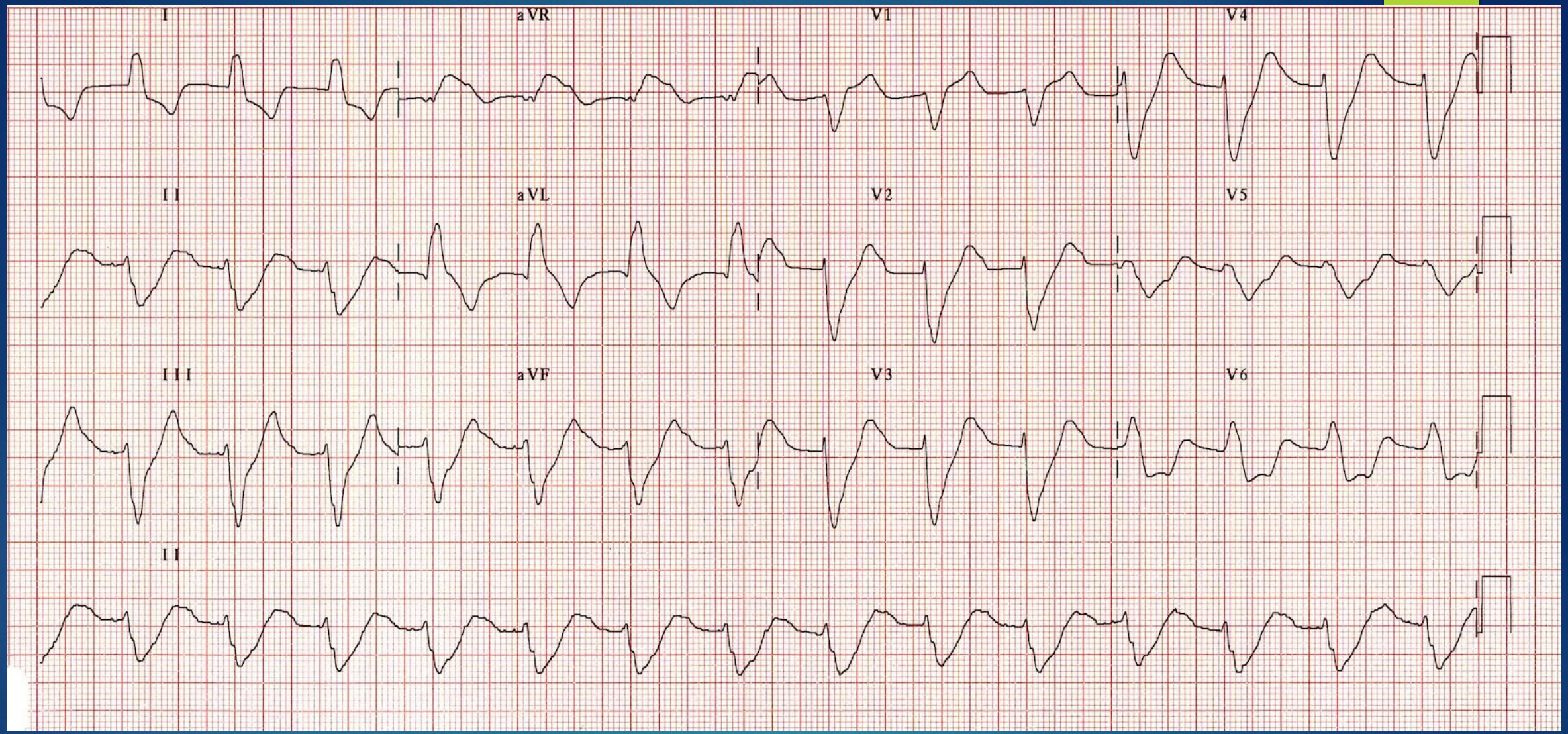
Figure 1 – This ECG rhythm strip shows a wide-complex bradycardic rhythm with sine-wave configuration and the absence of discernible P waves. These findings are consistent with pronounced hyperkalemia.



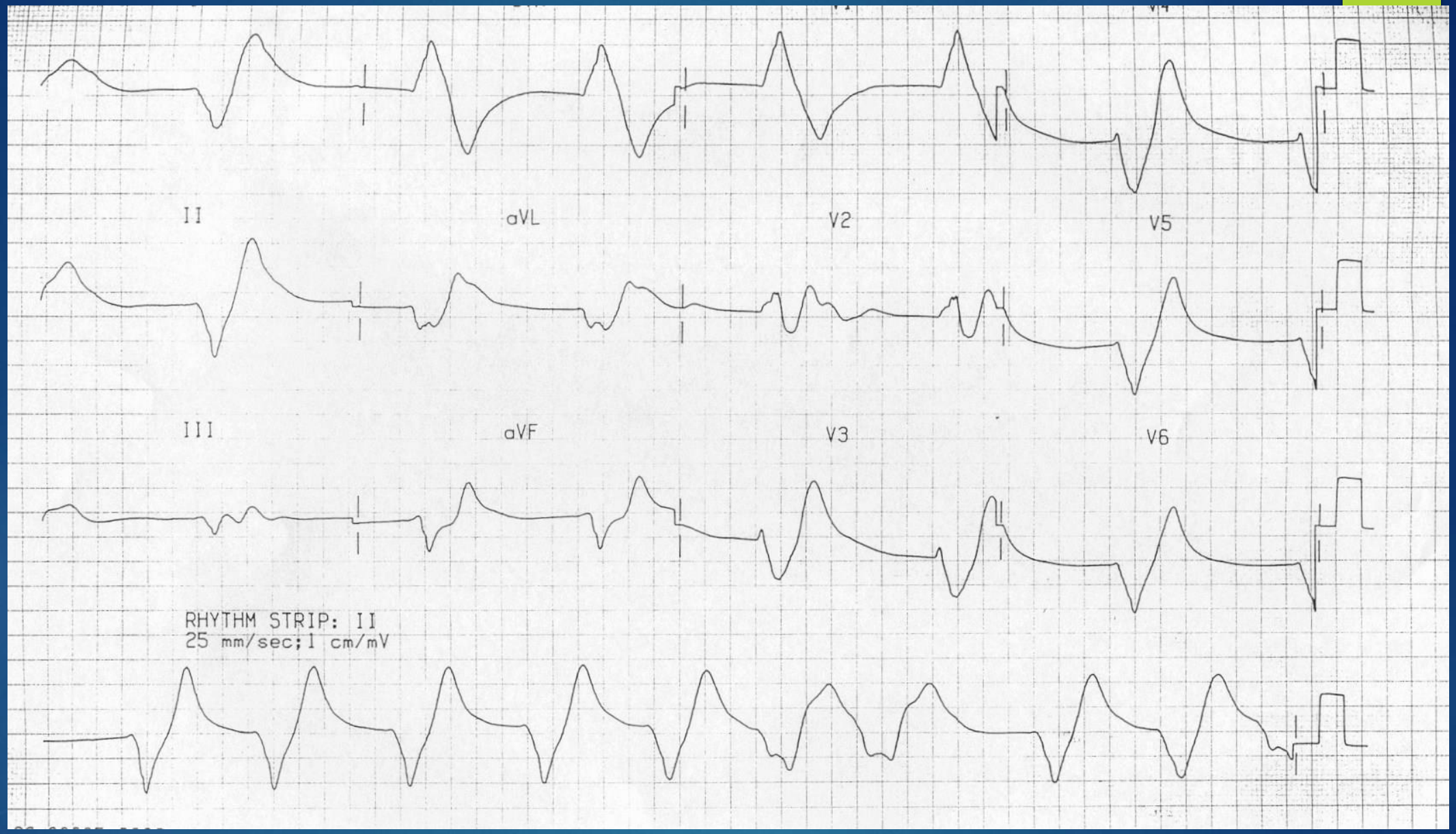
This patient had a serum K+ of 7.0



Long PR segment.
Wide, bizarre QRS.



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

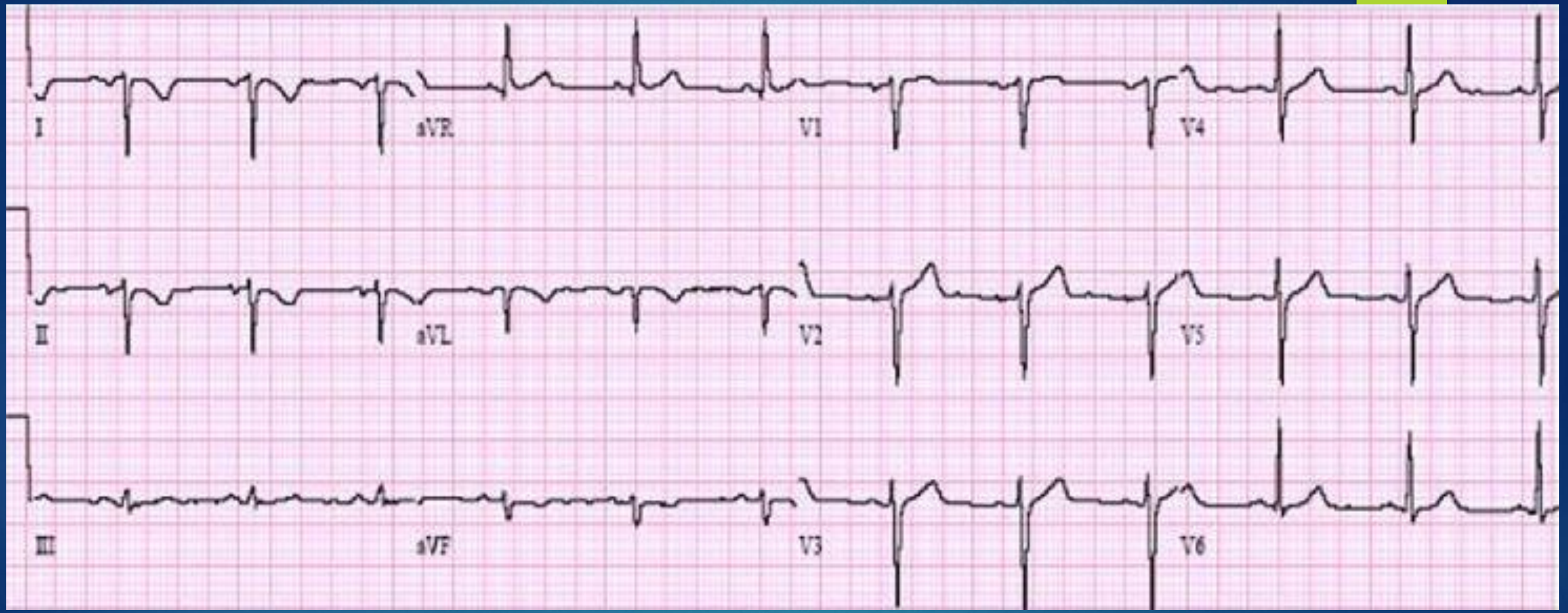


Sine wave appearance with severe hyperkalaemia (K+ 9.9 mEq/L)

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

What's this?


I
aVR
V1
V4
II
aVL
V2
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III
aVF
V3
V6