ECG and Chest Pain

- A bit of history
- Predict the future
- Some cases

ECG and Chest pain

- The main role of the ECG in a patient with chest pain is to help determine who has an AMI that would benefit from emergent reperfusion therapy

  - lytics (best < 2 hours) or cath lab

- Occasionally, the Hx & ECG's findings may be consistent with another diagnosis such as

  - PE

  - pericarditis

  - pericardial effusion

- But its all about reperfusion therapy

Once Upon A Time

- There was no treatment for an AMI
  - you lived or you died
- For those who lived, the ECG was used to divide them into
  - Q wave infarction
  - non- Q wave infarction

Then we entered the re-perfusion era…..

1994 Meta-analysis

- Who benefits from reperfusion?
- Studies of suspected AMI's who underwent thrombolysis
  - 9 trials totaling over 58000 patients
- For all comers: NNT = 56
- But if look at subgroups according to ECG
  - STEMI: NNT = 42
  - Normal or ST depression no benefit (some were harmed)

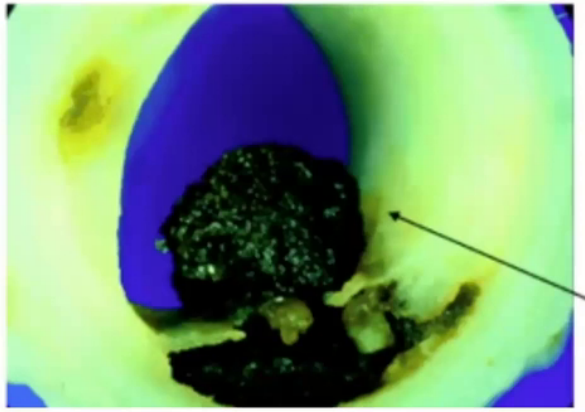
The STEMI Paradigm

- From this developed the theory that a ST elevation identifies an AMI due to an acute coronary artery occlusion
- Thus a STEMI predicts who benefits from emergent re-perfusion

  - thrombolysis

  - cath lab

UA / NSTEMI

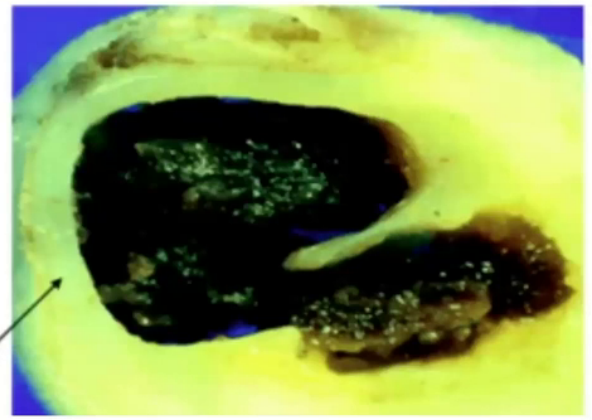


Partial occlusion

Rx: Stabilize

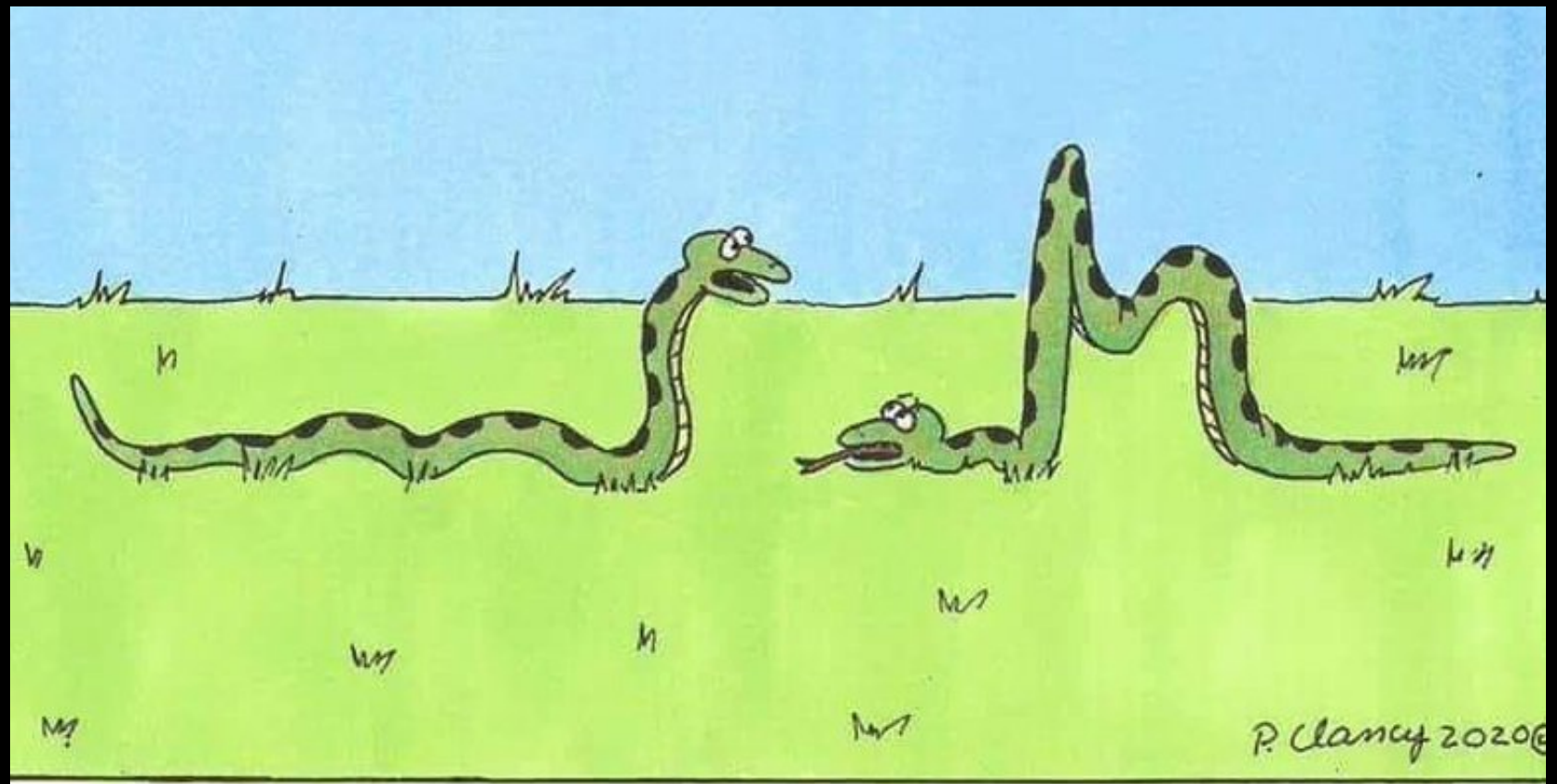
STEMI

Complete occlusion



Rx: Revascularization

In the STEMI paradigm, all that matters is the ST segment



However

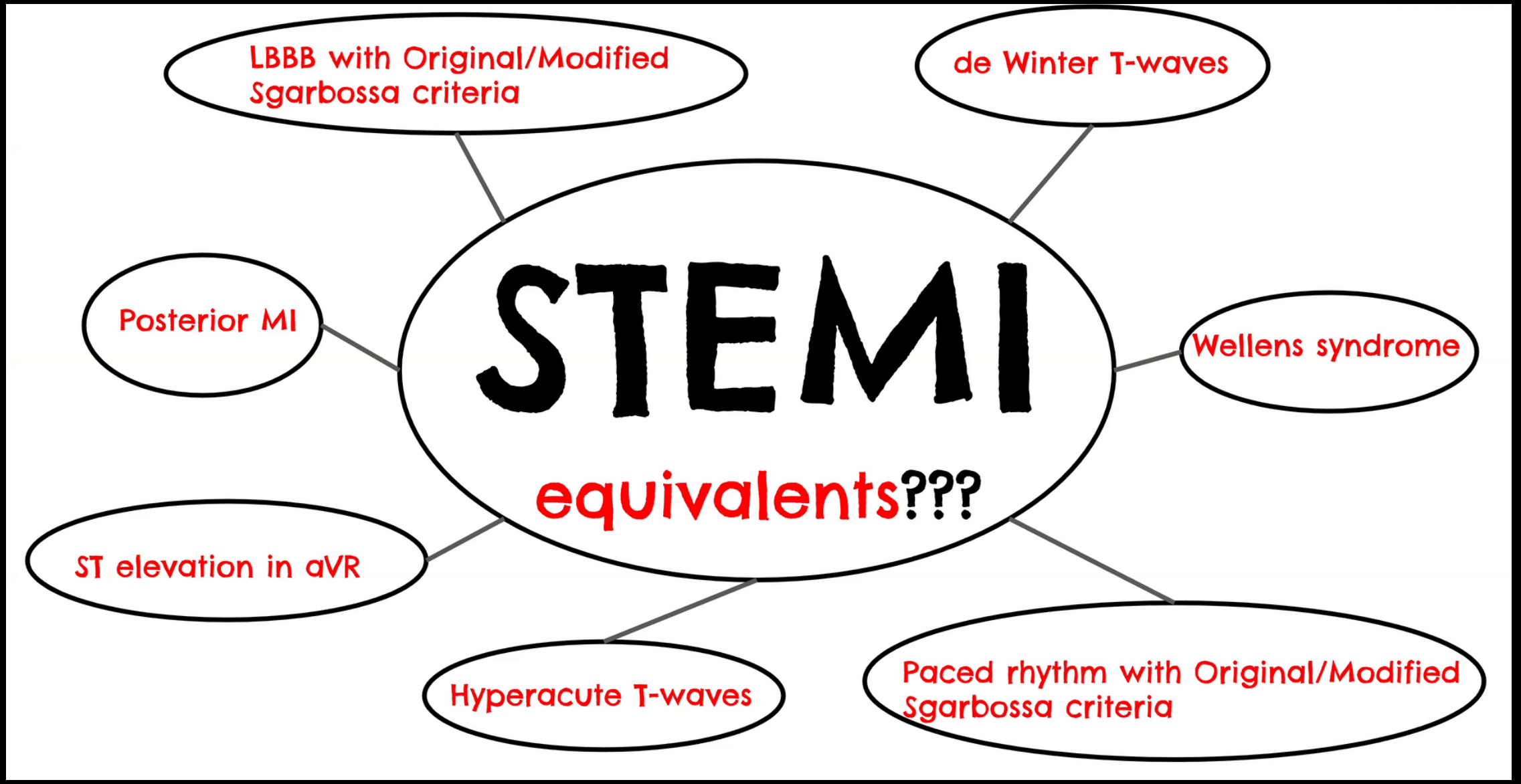
- About 25% of “STEMI’s” are false positives
  - STEMI mimics: LBBB; LVH; BER and many others
- These patients get no benefit from reperfusion and may (rarely) be harmed by the intervention
  - bleeding
  - false aneurysm
  - arterial dissection

Furthermore

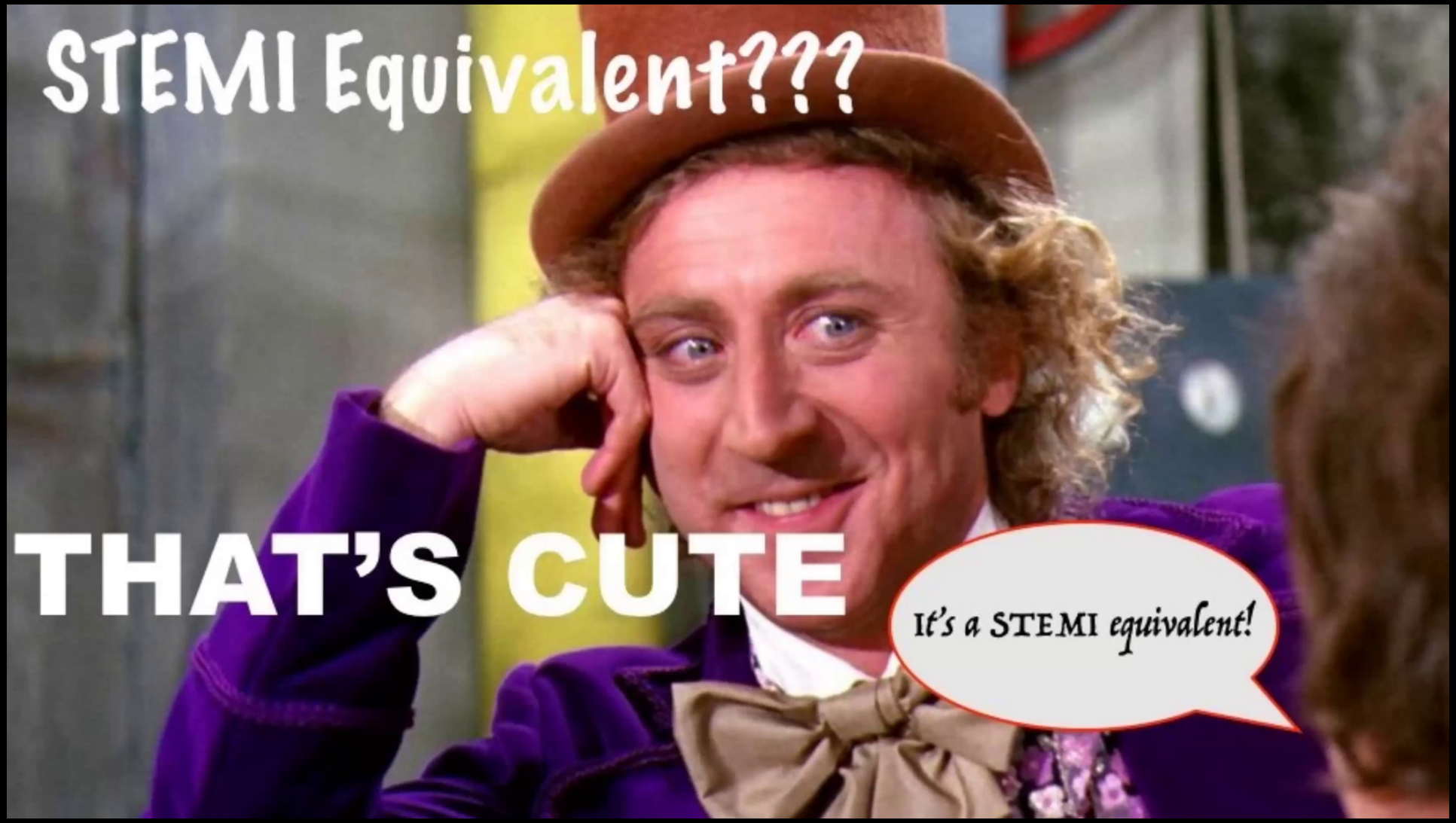
- About 25% of NSTEMI's actually have complete occlusion
- Without reperfusion, these patients have:
  - increased mortality (doubled)
  - increased complications
- Thus like patients with STEMI's, some NSTEMI's also benefit from early reperfusion treatment

So…..

- NSTEMI’s that benefit from emergent reperfusion are called STEMI-equivalents
- The list of STEMI equivalents keeps getting bigger and bigger - sucks to be you (I’m retired – maybe – it’s complicated)


LBBB with Original/Modified Sgarbossa criteria
de Winter T-waves
Posterior MI
STEMI
equivalents???
Wellens syndrome
ST elevation in aVR
Hyperacute T-waves
Paced rhythm with Original/Modified Sgarbossa criteria

Your Friendly Neighborhood Cardiologist



Occlusion Myocardial Infarction Paradigm

- The future?
- An OMI is an acute coronary occlusion resulting in an AMI and would benefit from immediate reperfusion

  - includes STEMI's and STEMI-mimics
- This involves:

  - Hx & Examination
  - ECG: look for more than just ST elevation
  - Bedside US: wall motion abnormalities
  - ?Point of care HS-troponin


GM
GM
GM
Sportsaver
The Test Experts
Sportsaver

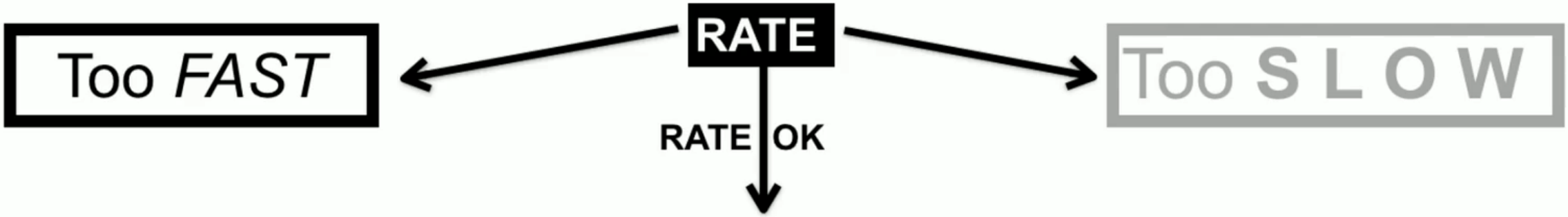
Reading an ECG

- Start with the clinical scenario
- You need a system +/- chest sheet

  - with practice, you will recognize patterns
- There are lots to choose from
- You can make your own
- But you need a system

ECGs by EMRAP

The 30 Second Approach



MOST PROMINENT FEATURE

WIDE QRS	ST ⬆	ST ⬇	BIG Ts	FLIPPED Ts

The 5 Step ECG

- Rate and rhythm
- Axis
- Intervals
- Enlargement
- Ischemia/infarction

Applying the *Systematic* Approach

▶ **Rate** — Divide 300 by # of boxes in R-R Interval (*pg 5*).

▶ **Rhythm** — "*Watch your* ***P****'s &* ***Q****'s — and the* ***3 R's*** *!* "

- Are there **P waves**? If so — ***upright*** in **lead II**?

P waves should *always* be ***upright*** in **lead II** IF there is sinus rhythm (unless *there is lead reversal/dextrocardia*).

- Is the **QRS** wide or narrow?
- What is the **Rate**?
- Is the rhythm **Regular**?
- Are P waves **Related** (ie, *"married" with fixed PR interval*) to the QRS? (*pg 4*).

▶ **Intervals** — Look at intervals *early* in the process!

- The **PR Interval** (*pg 19*) — is *prolonged* IF >0.20-0.21 second (if clearly *more* than a *LARGE* box in duration).
- The **QRS Complex** (*pg 20*) — is *wide* IF >0.10 sec. (if *more* than *HALF* a *large* box).
- The **QT Interval** (*pg 28*) — is *prolonged* IF clearly *more* than *half* the R-R interval (provided that heart rate is *not more* than 100 beats/minute) — *"Drugs-Lytes-CNS"*

KEY **Point** — IF the rhythm is sinus, but the QRS is wide — then ***STOP*** and figure out why (*RBBB, LBBB, IVCD, WPW*) — *before* proceeding further (*See pg 20*).

▶ **Axis** (*pg 31*) — *Look at* **lead I** (*at 0°*) and **aVF** (*at +90°*):

- The axis is ***normal*** — IF *net* QRS deflection is *positive* in leads I and aVF (*defines axis to be between 0° to +90°*).
- There is **RAD** — IF *net* QRS deflection is negative in I, but positive in aVF (*Think RVH, LPHB or normal variant*).
- There is **LAD** — IF the *net* QRS is positive in I, but negative in aVF. There is *pathologic LAD* = **LAHB** — IF *net* QRS is more *negative* than positive in lead II (*See pg 33*).
- The axis is ***indeterminate*** — IF *net* QRS deflection is *negative* in I and aVF (*Think RVH, COPD, obesity*).

▶ **Hypertrophy** (*pp 35 – 40*):

- The "*magic numbers*" for **LVH** are **35** (*deepest* S in V1,V2 — ***plus*** — *tallest* R in V5,V6 — *in a patient at least* ***35*** *years of age*) — and **12** (*for the R in aVL*). True chamber enlargement is more likely IF ***"strain"*** also present!
- There is **RAA** (*P Pulmonale*) — IF P waves are **p**rominent (*≥2.5 mm tall*) and **p**eaked (ie, *"uncomfortable to sit on"*) in the ***p****ulmonary* leads (*II, III, and aVF*). — *pg 37* —
- There is **LAA** (*P Mitrale*) — IF P waves are *notched* (***"m"****-shaped*) in **m**itral leads (*I, II, or aVL*) — or if the P in V1 has a *deep* terminal *negative* component. — *pg 37* —
- Consider ***pulmonary*** **disease** — IF there is RAA, RAD (*or indeterminate axis*), incomplete RBBB (*or rSr' pattern in lead V1*), low voltage, or persistent precordial S waves.
- Consider **RVH** — IF there is also a tall R wave in V1 and right ventricular "strain". — *pp 38-39* —
- With **BBB** — Criteria for LVH/RVH (*See pg 40*).

▶ **Infarct** (= ***Q—R—S—T*** *changes*) — Look at *all* leads (*except perhaps lead aVR*) for the following (*pg 41*):

- **Q Waves** — Small (*normal* ***septal*** **q waves**) are commonly seen in *lateral* leads (***I,aVL,****V4,****V5,V6***); moderate or large-sized Q waves are normal (as an *isolated* finding) in leads III, aVF, aVL, and V_1. — ***pg 45*** —
- **R Wave Progression** — Transition *between* V2-to-V4? (*pg 42*) — *Tall R wave* in V1? (*pg 55*) — *rSr' pattern* in V1?
- **ST Segments** — More than the *amount* of ST segment deviation, focus on *shape* (***"smiley"*** or ***"frowny"*** — *pg 46A*)
- **T Waves** — May *normally* be inverted in leads III, aVF, aVL, and V1 (*pg 45*).
- *Diagnosing* **MI** *with* **BBB** (*pg 26A*).

Suggested Approach: — Use the above as a *guide* for ***descriptive*** **analysis**; then formulate your **clinical** ***impression***. Whenever possible — *WRITE OUT* your findings (but even when time is short — *be systematic*)

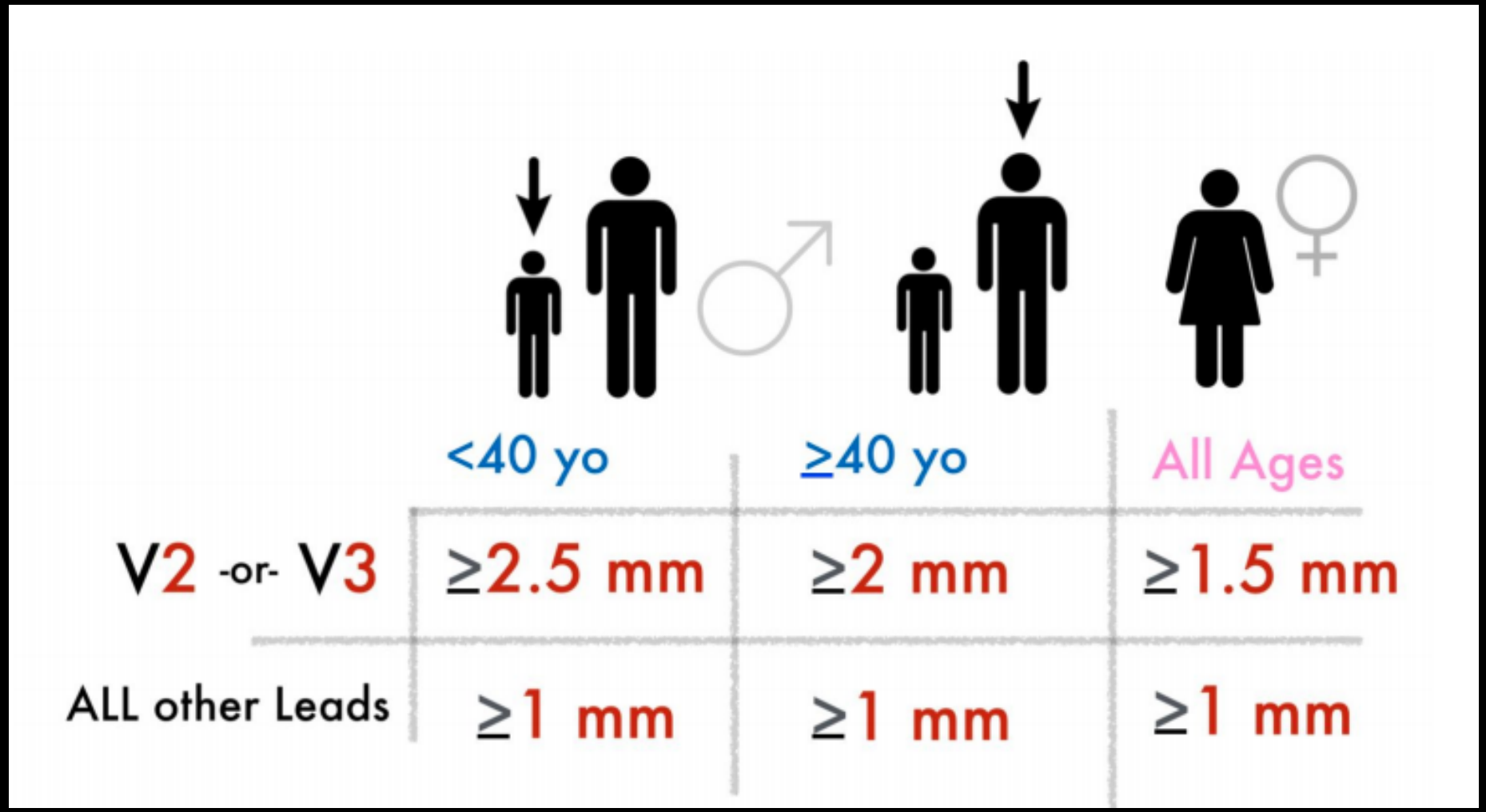
Simplified STEMI Criteria

- Measure ST elevation at the J point vrs the isoelectric line
  - PR or TP segment
- Greater than 1 mm in 2 contiguous limb leads
  - Limb Leads: I, II, III, aVL, aVF and aVR
- Greater than 2 mm in 2 contiguous chest leads
  - Chest Leads: V1 to V6

But the amount of ST elevation may be proportional to the size of the QRS

- A small QRS complex may have < 2mm ST elevation

STEMI Criteria: 2013 ACCF/ AHA Guidelines



	<40 yo	≥40 yo	All Ages
V2 -or- V3	≥2.5 mm	≥2 mm	≥1.5 mm
ALL other Leads	≥1 mm	≥1 mm	≥1 mm

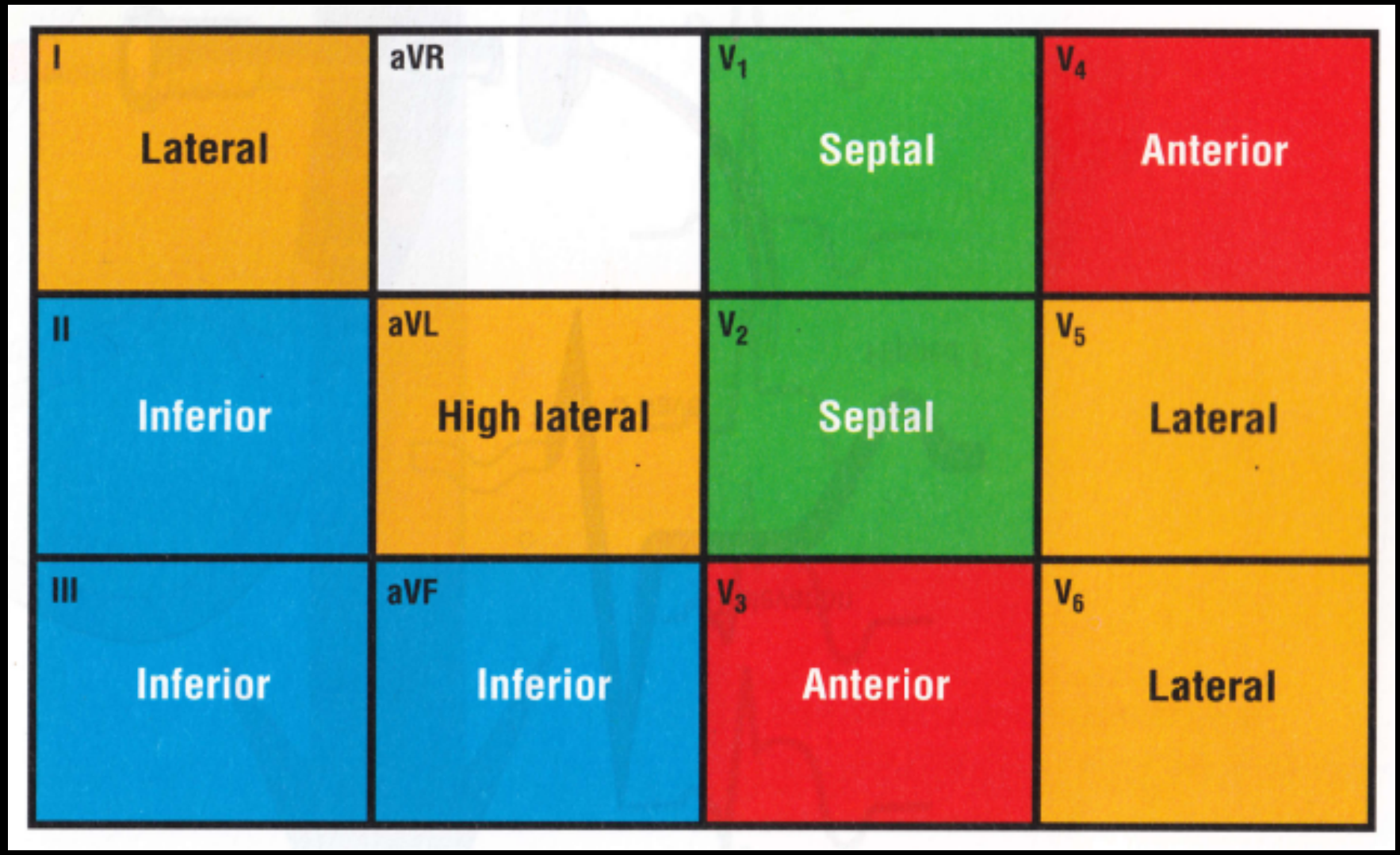
STEMI Criteria Don’t Apply

- LBBB
- Paced
- LVH

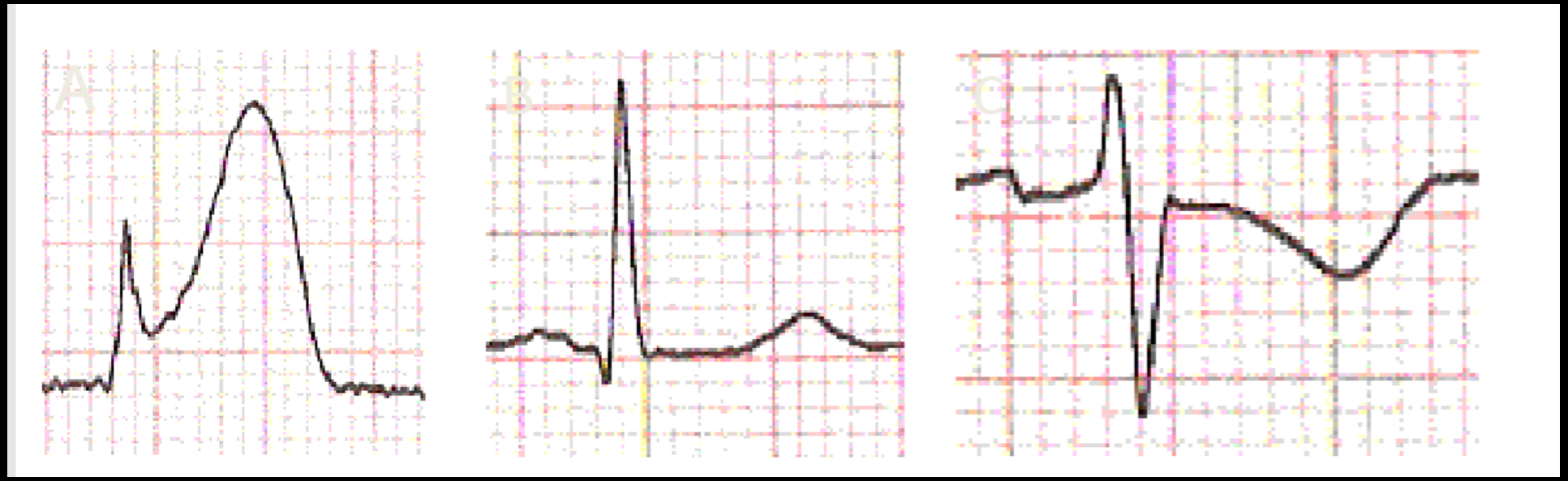
We can use different criteria but

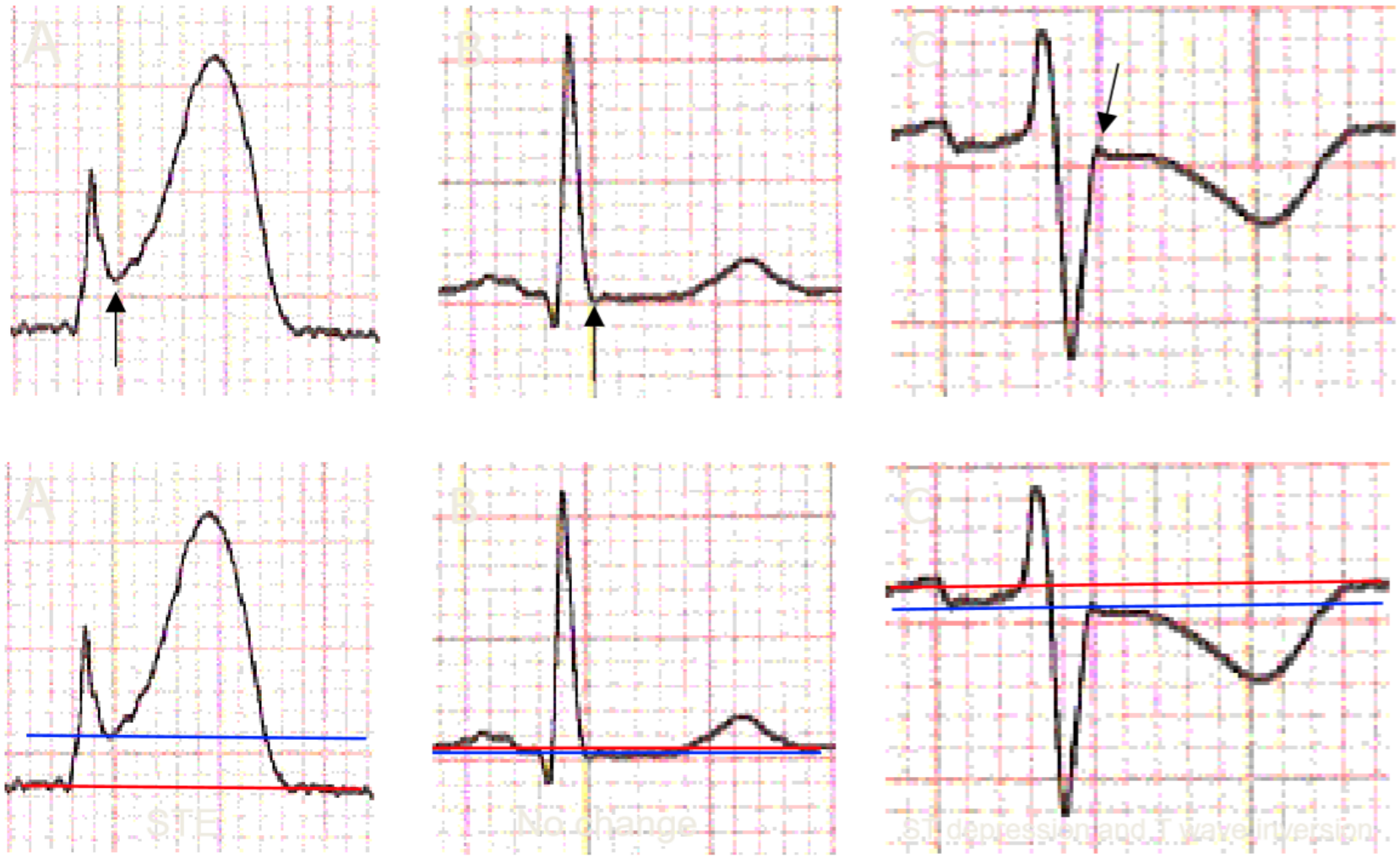
- complicated
- controversial

Contiguous Lead

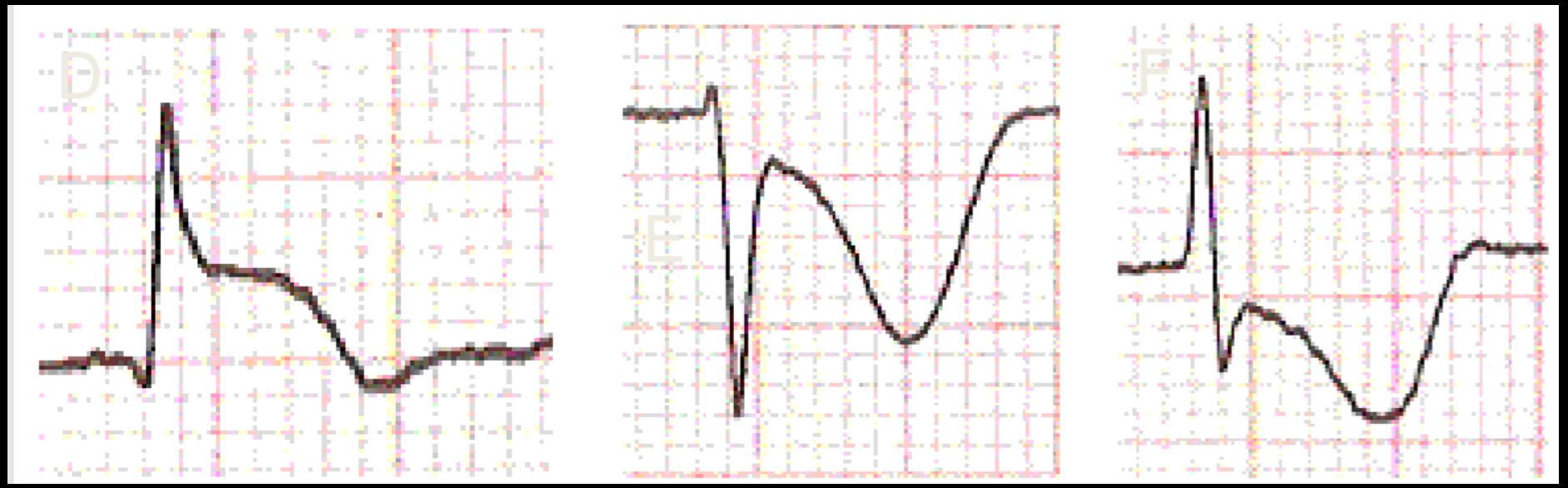


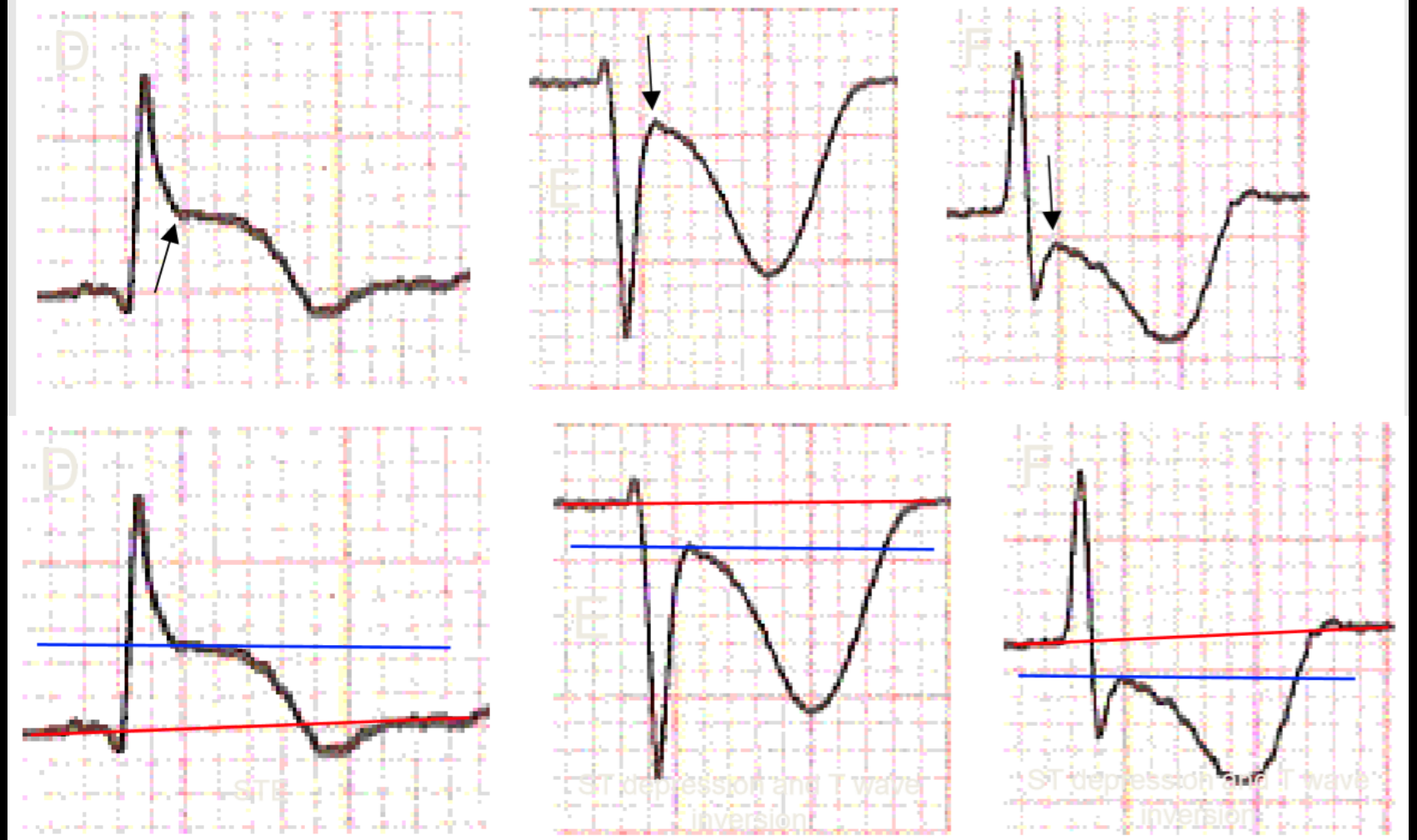
Where Is The J Point?




A
B
C
A
B
C
STE
No change
ST depression and T wave inversion
Red is isoelectric, Blue is J point

Where Is The J Point?



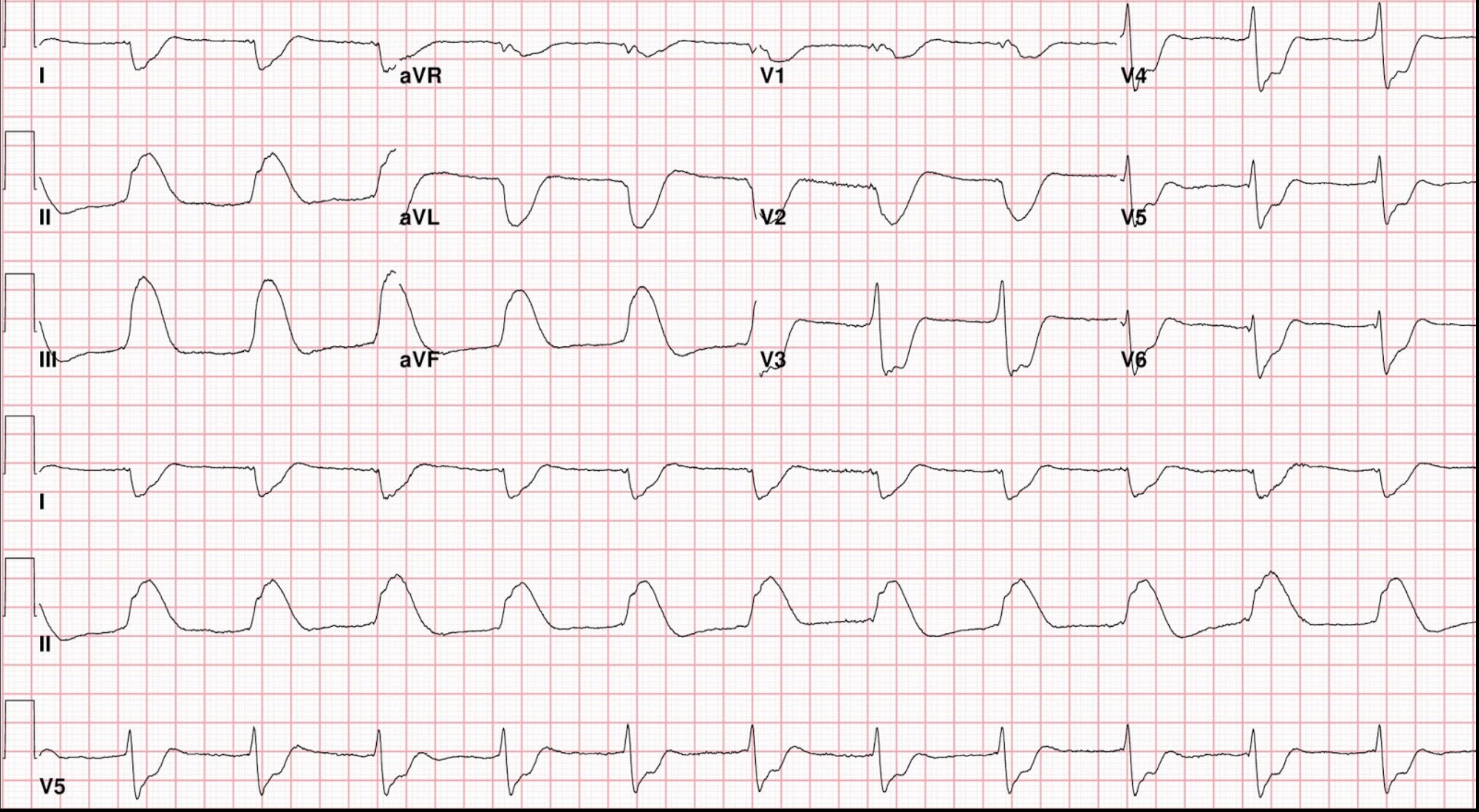


Red is isoelectric, Blue is J point

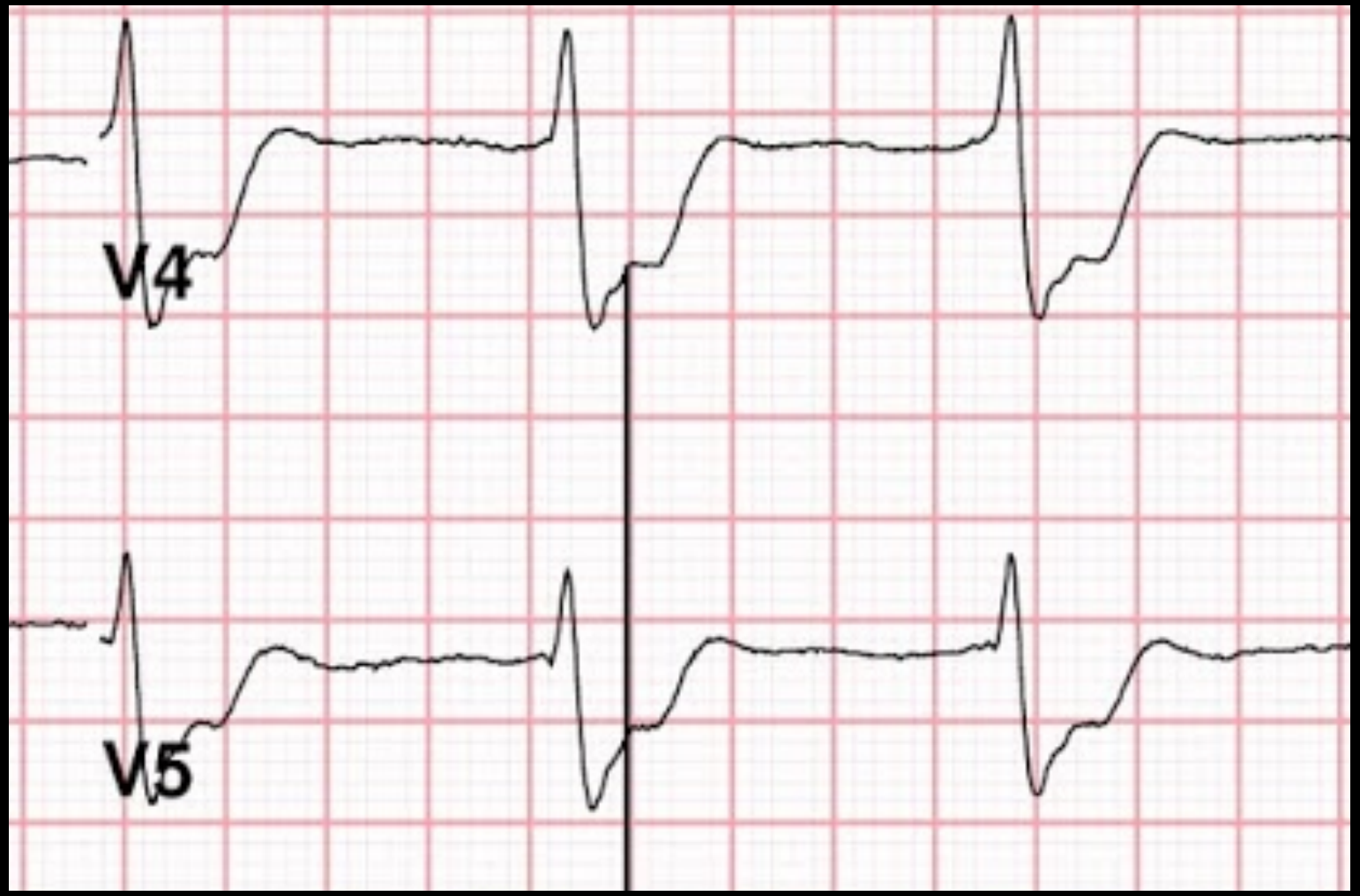
Case: Find The J Point

- 75 year old man arrested at the shopping center
- In VF when the ambo's arrive
- Shocked and intubated

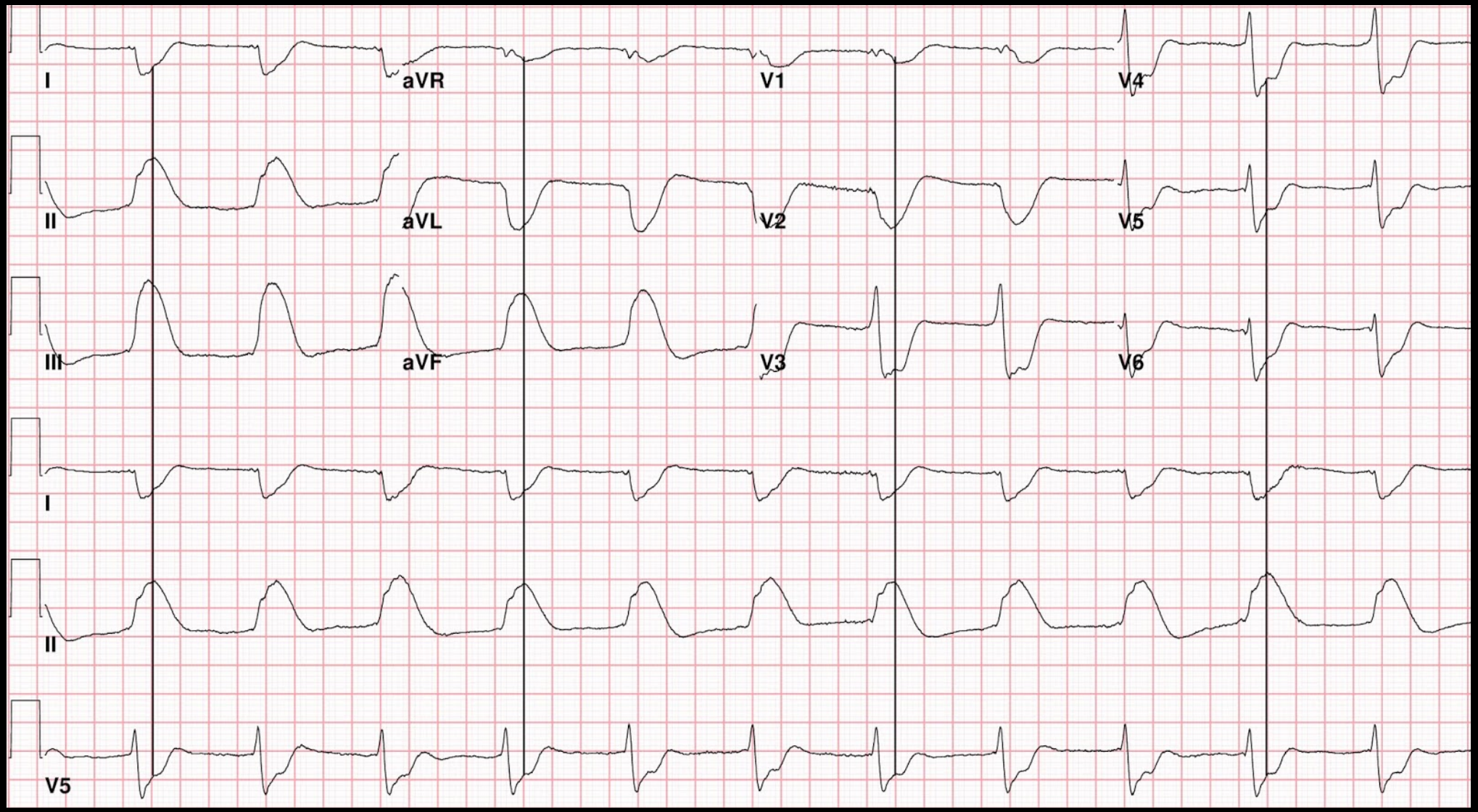
- Does his ECG show
  1) Hyper-kalaemia
  2) VT
  3) Some weird tox shit
  4) ST elevation
  5) No idea

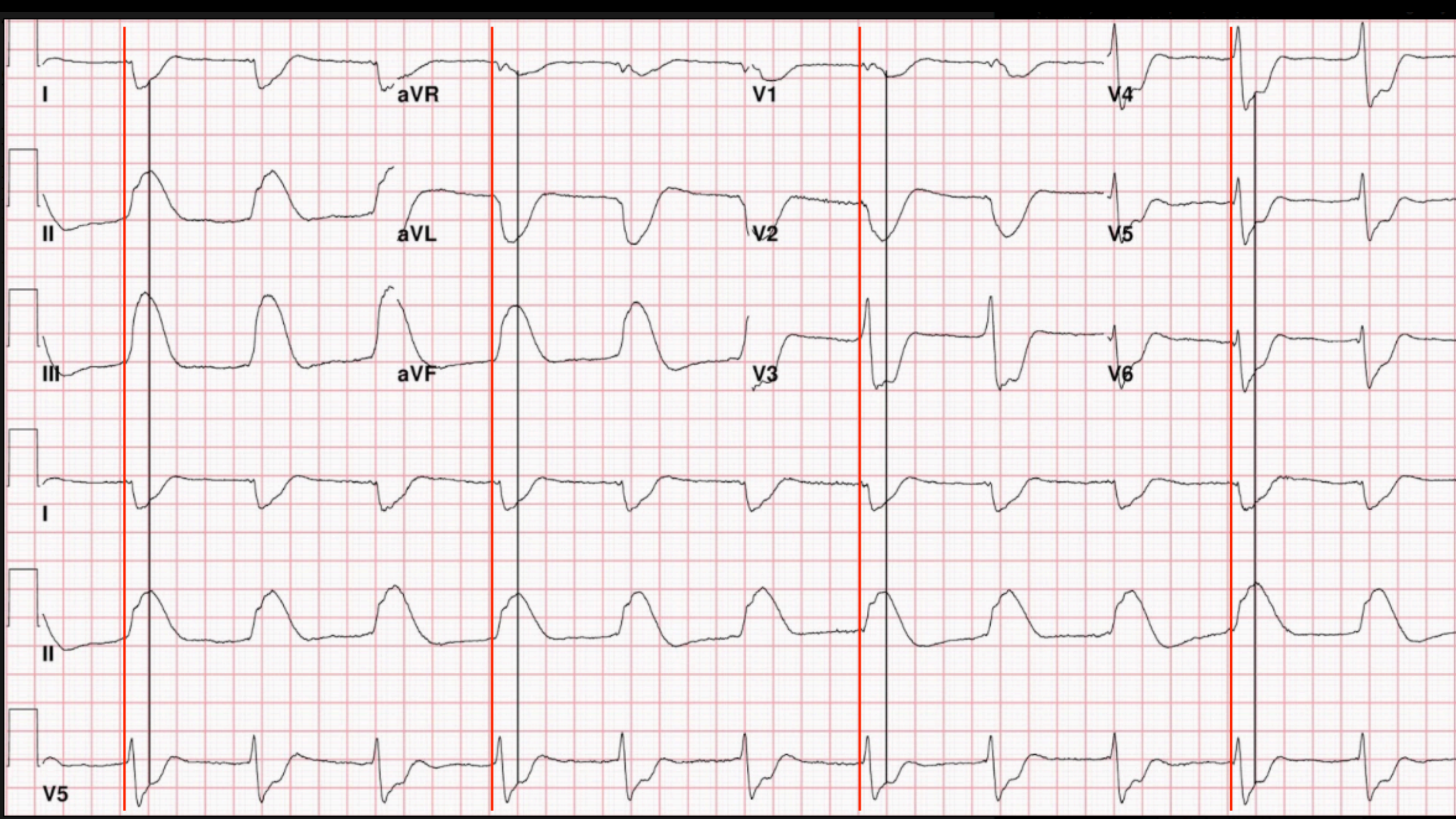

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
I
II
V5

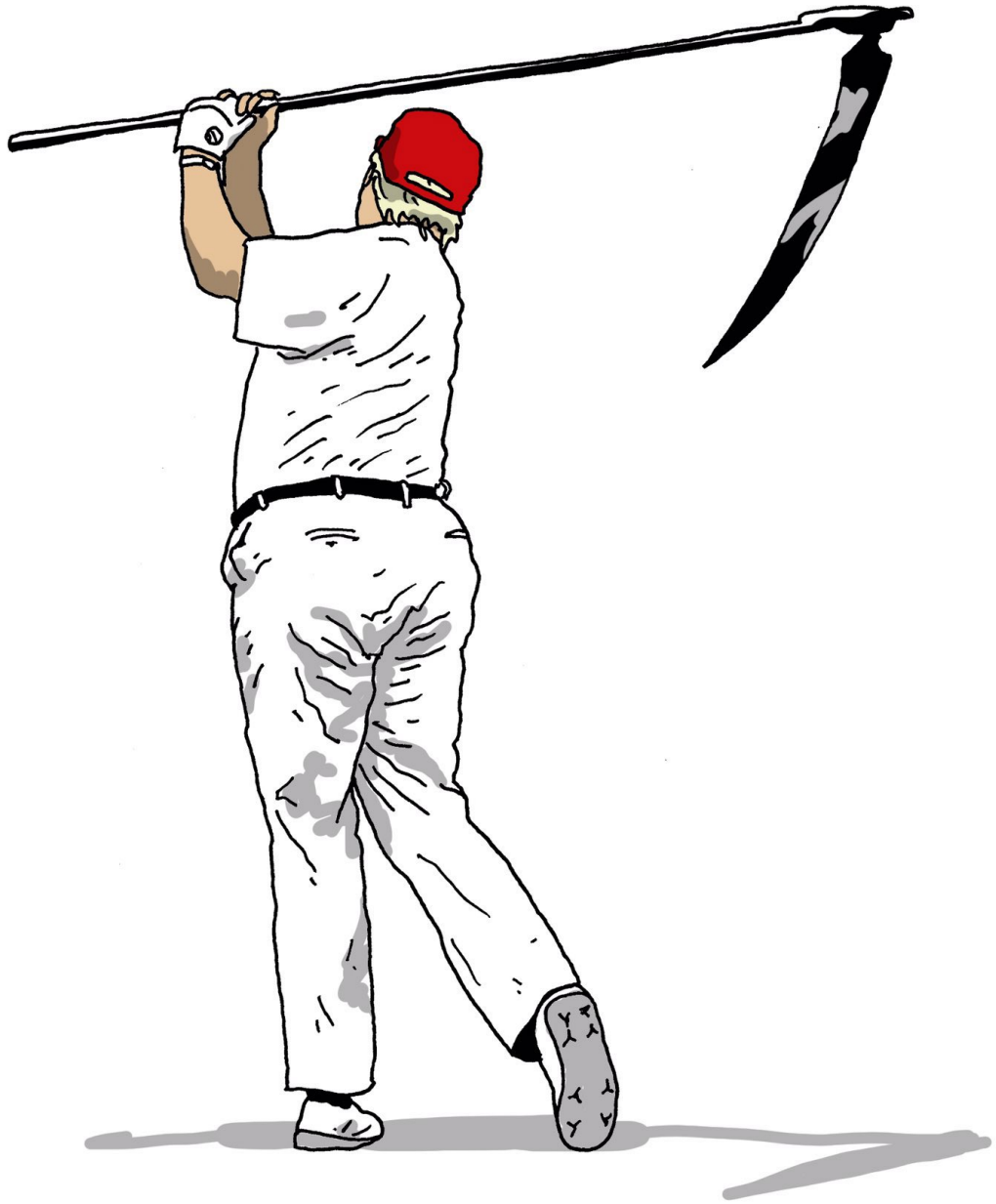
Only Need To Find The J Point In One Lead



Only Need To Find The J Point In One Lead




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



ST Elevation: Is it a STEMI?

- ST elevation does not always mean it is a STE infarction
- Other ECG findings that favour an AMI
  - reciprocal ST depression!! (can occur early)
  - shape of the ST segment
  - hyper-acute T waves (occurs early and evolve into STE)

- Still not sure?
  - serial ECG's
  - get an old ECG for comparison
  - phone a friend

Where do I look for ‘Reciprocal’ Changes

- **“P.A.I.L.I”**
  - **P**osterior: anterior
  - **A**nterior: inferior (about 50%)
  - **I**nferior: lateral (STD in aVL about 95%)
  - **L**ateral: inferior or septal

These changes are not written in stone. You should look in all leads for ST depression

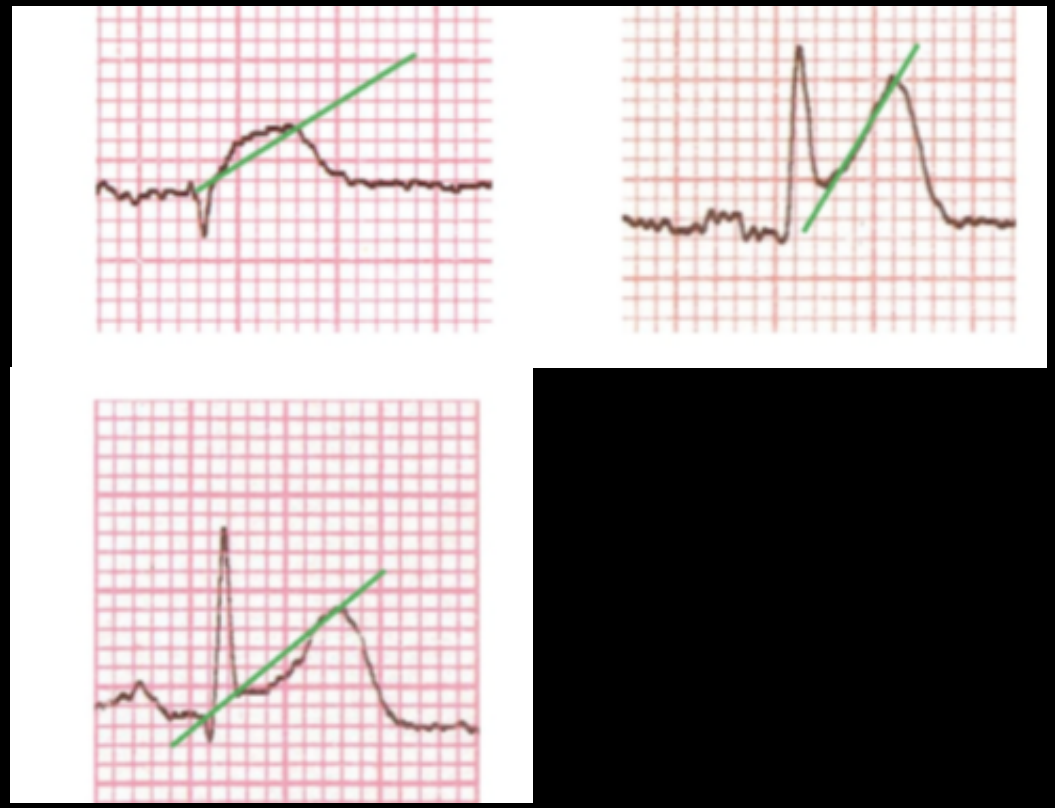
The ST Segment Morphology

Draw a line from the J point to the peak of the T wave and look at the shape of the ST segment

Convex: ishaemia?

Straight: ischaemia?

Concave: benign?



Hyper-acute T Waves

Not well studied & poorly defined

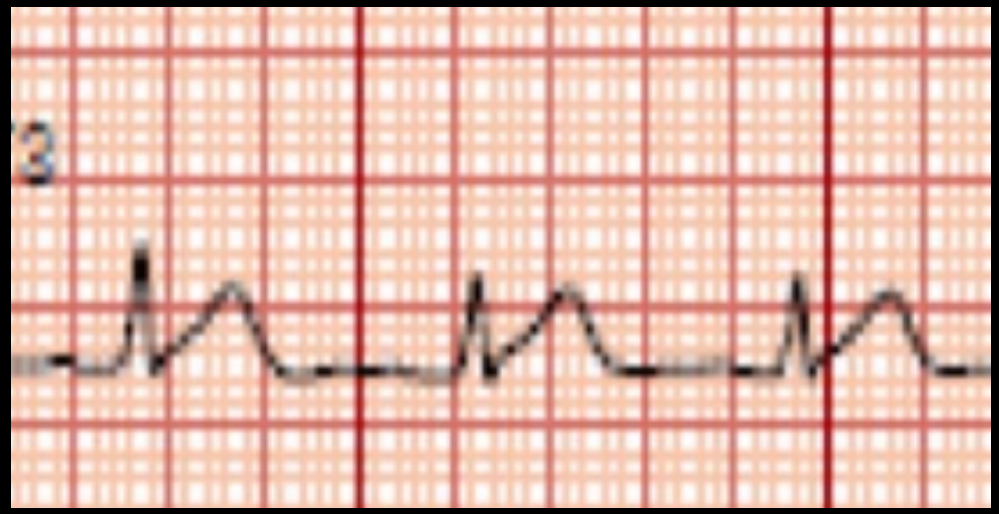
In an anatomic distribution

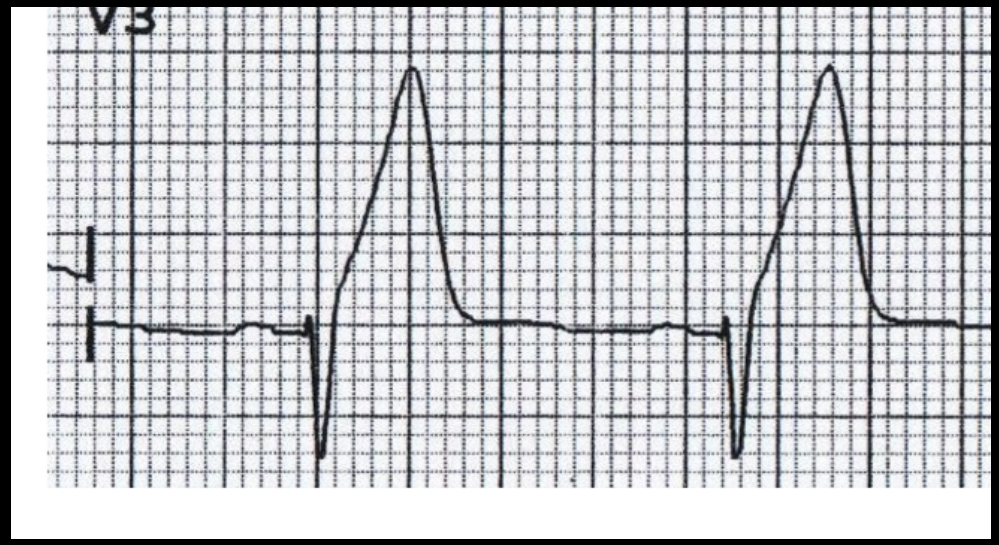
Morphology: R wave straight in to T wave

No ST segment

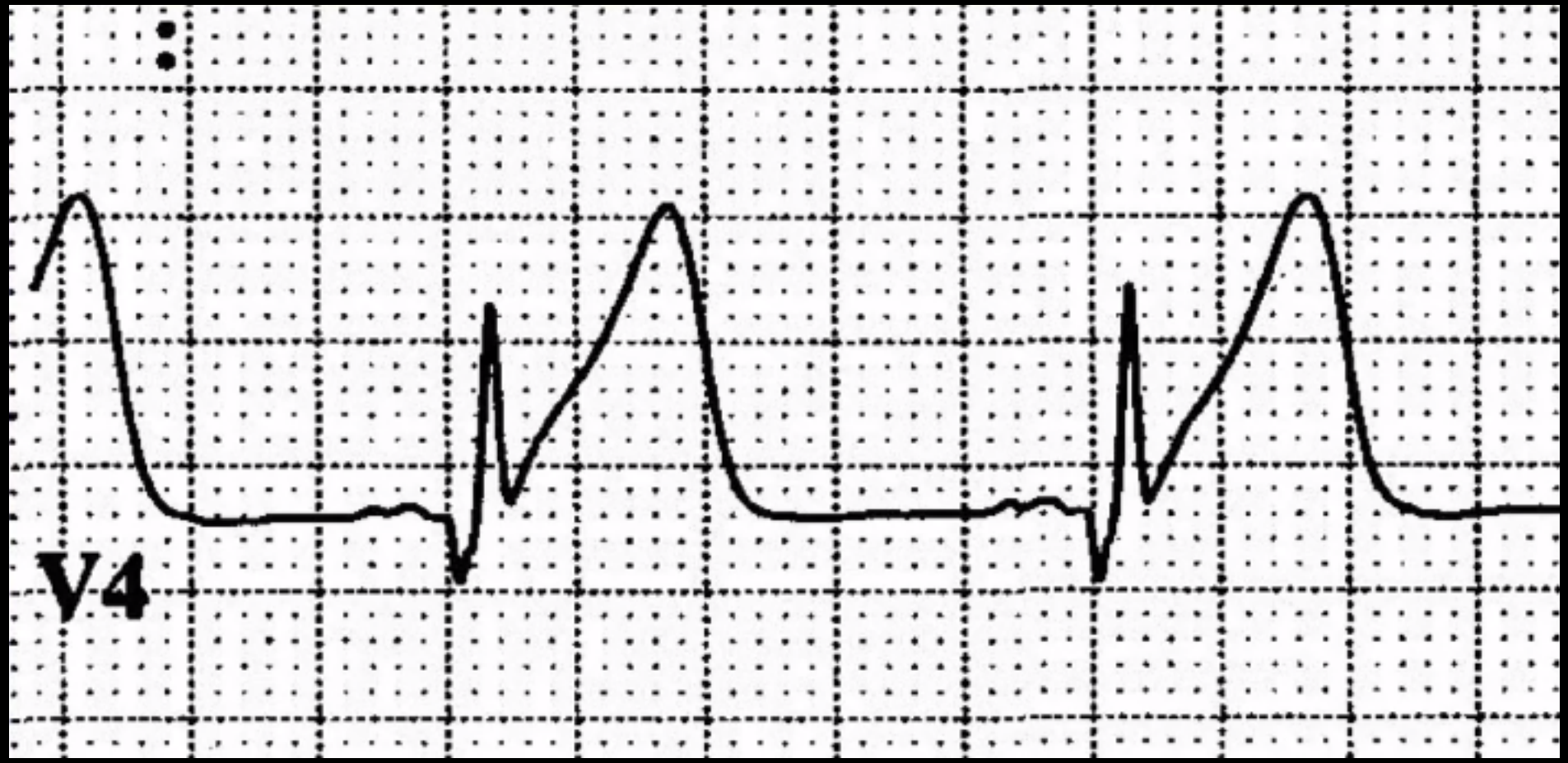
Size: big compared to the size of the QRS

If the entire QRS fits inside the T wave, its big and an infarct till proven otherwise

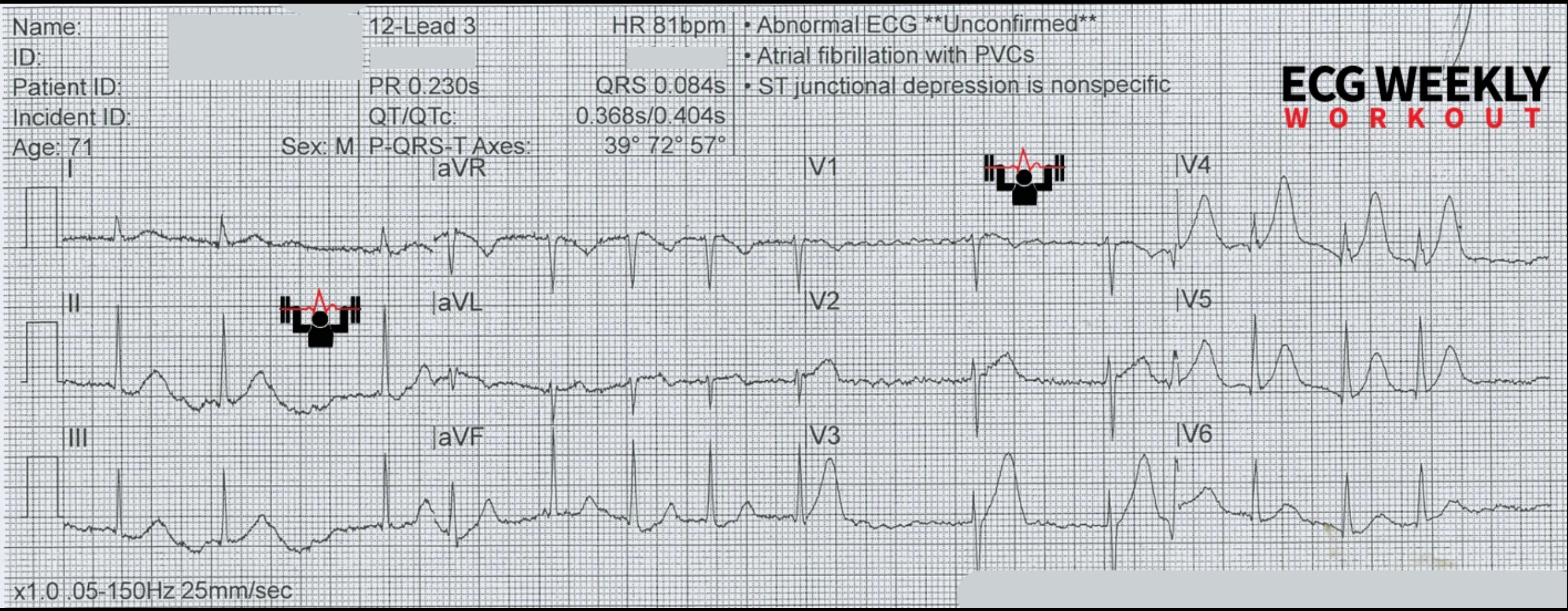


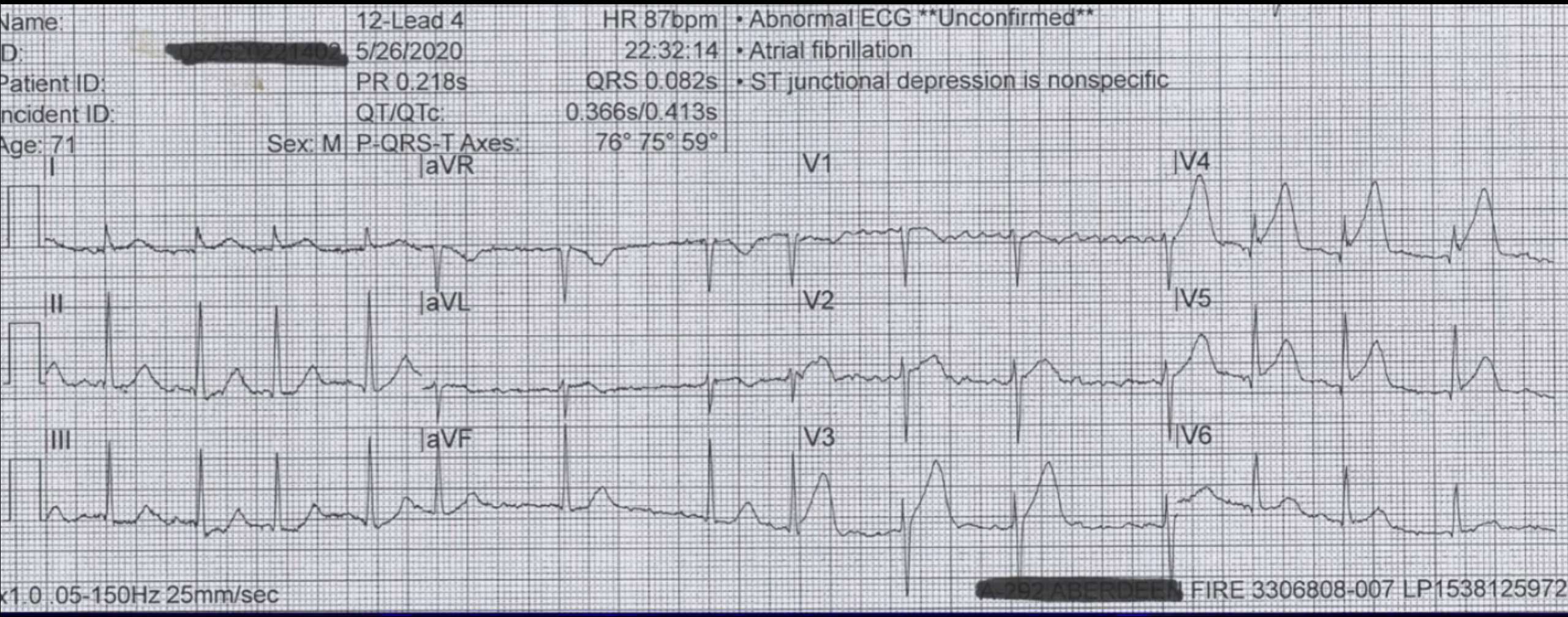


Hyper-acute T Wave: Too Big & Too Straight



Do I Come To You?




Name:
ID: [redacted]
Patient ID:
Incident ID:
Age: 71 Sex: M
12-Lead 4
5/26/2020
PR 0.218s
QT/QTc:
P-QRS-T Axes:
HR 87bpm
22:32:14
QRS 0.082s
0.366s/0.413s
76° 75° 59°
• Abnormal ECG **Unconfirmed**
• Atrial fibrillation
• ST junctional depression is nonspecific
I
aVR
V1
V4
II
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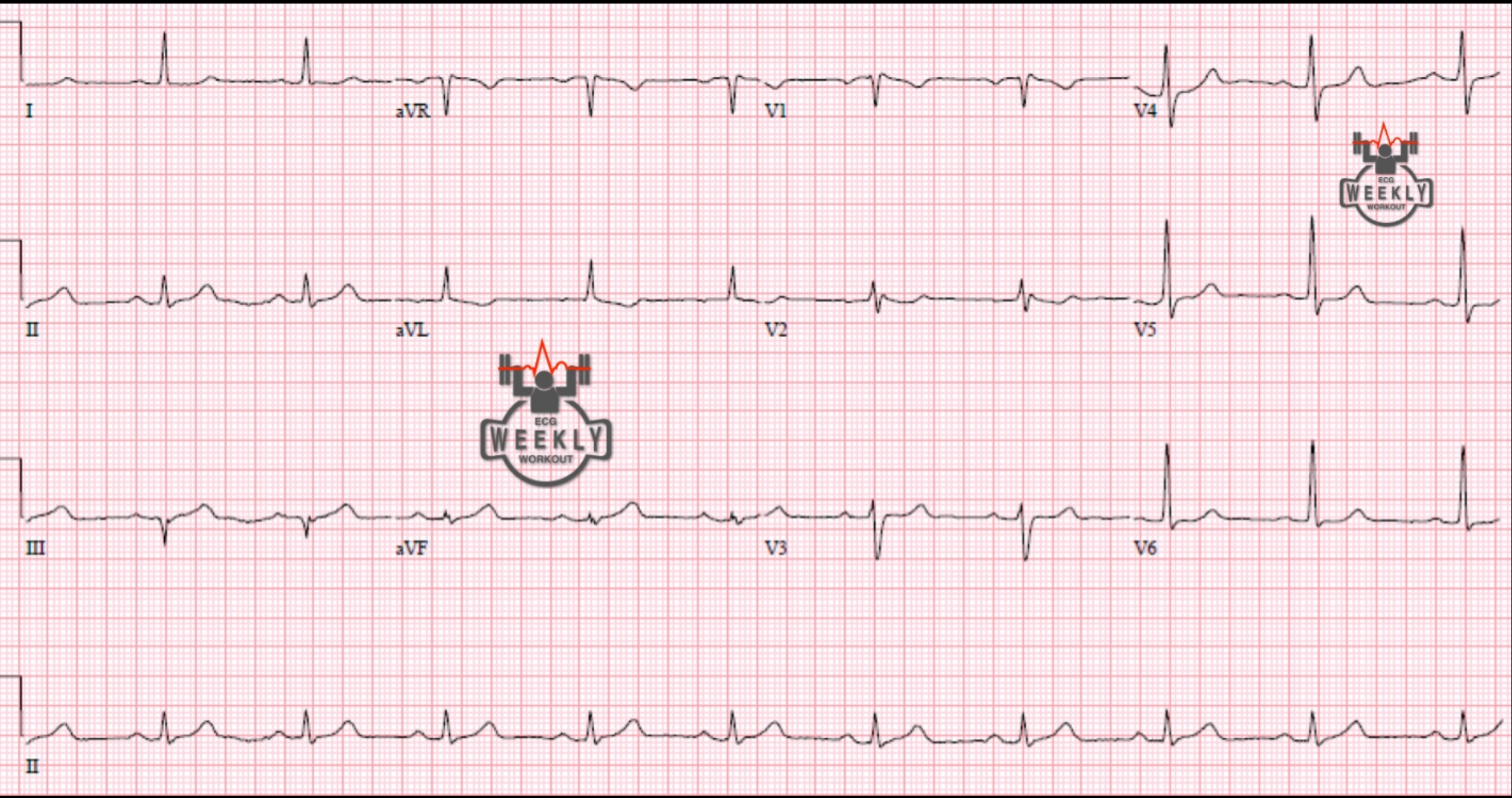
V2
V5
III
aVF
V3
V6
x1.0 .05-150Hz 25mm/sec
[redacted] FIRE 3306808-007 LP1538125972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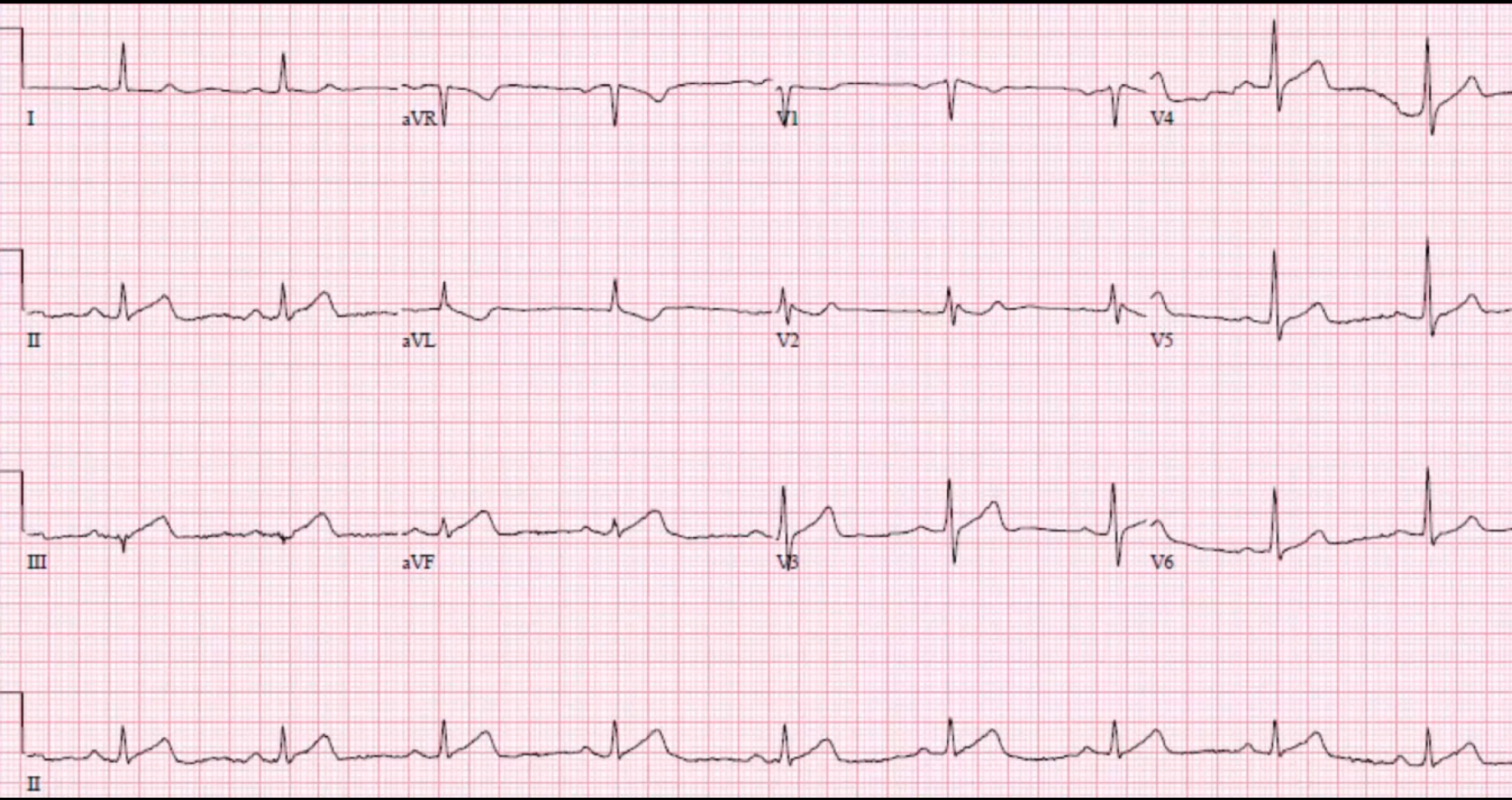
I
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III
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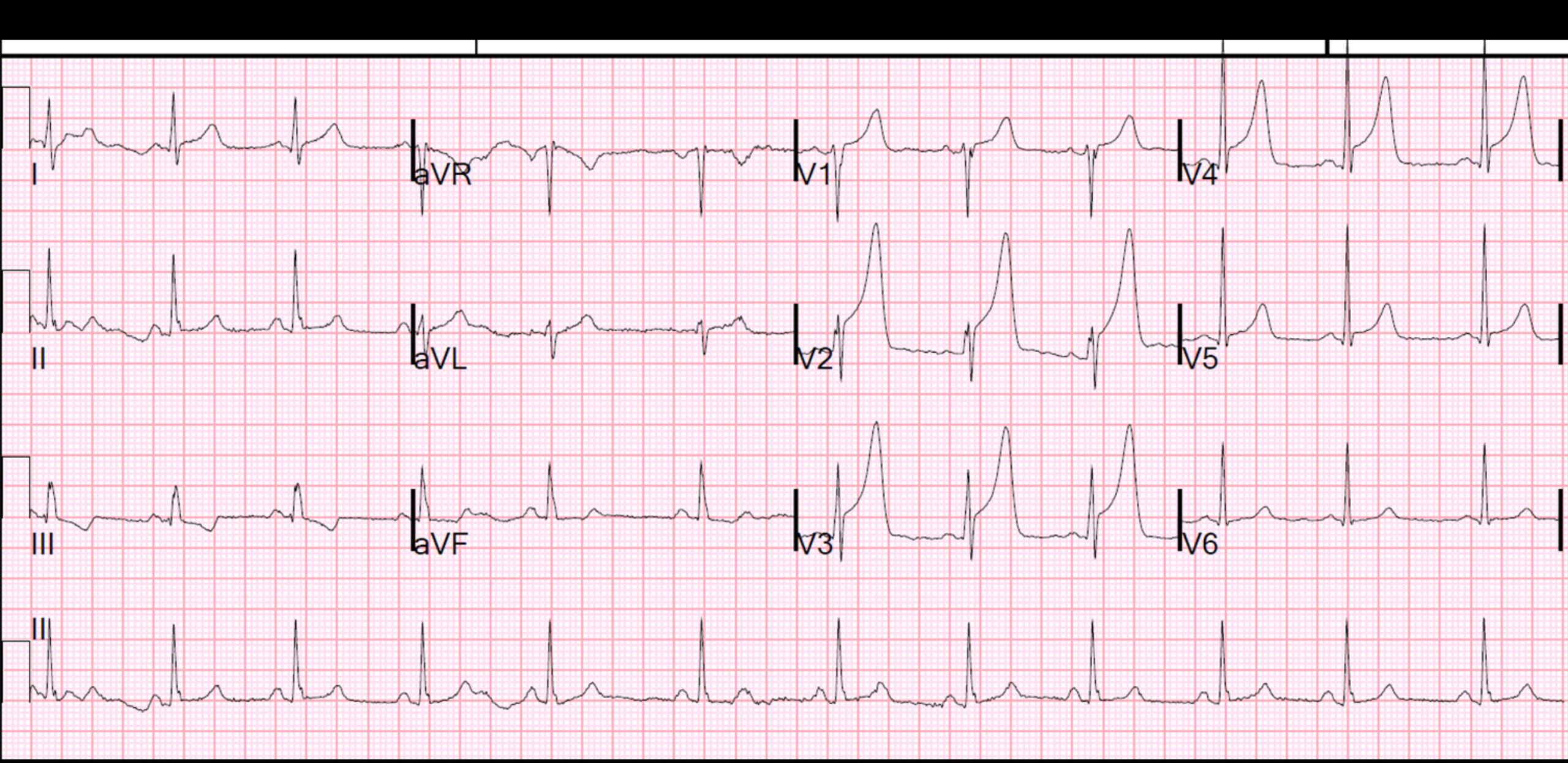
V3
V6
II

Case

62 year old man

Chest pain and diaphoresis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III
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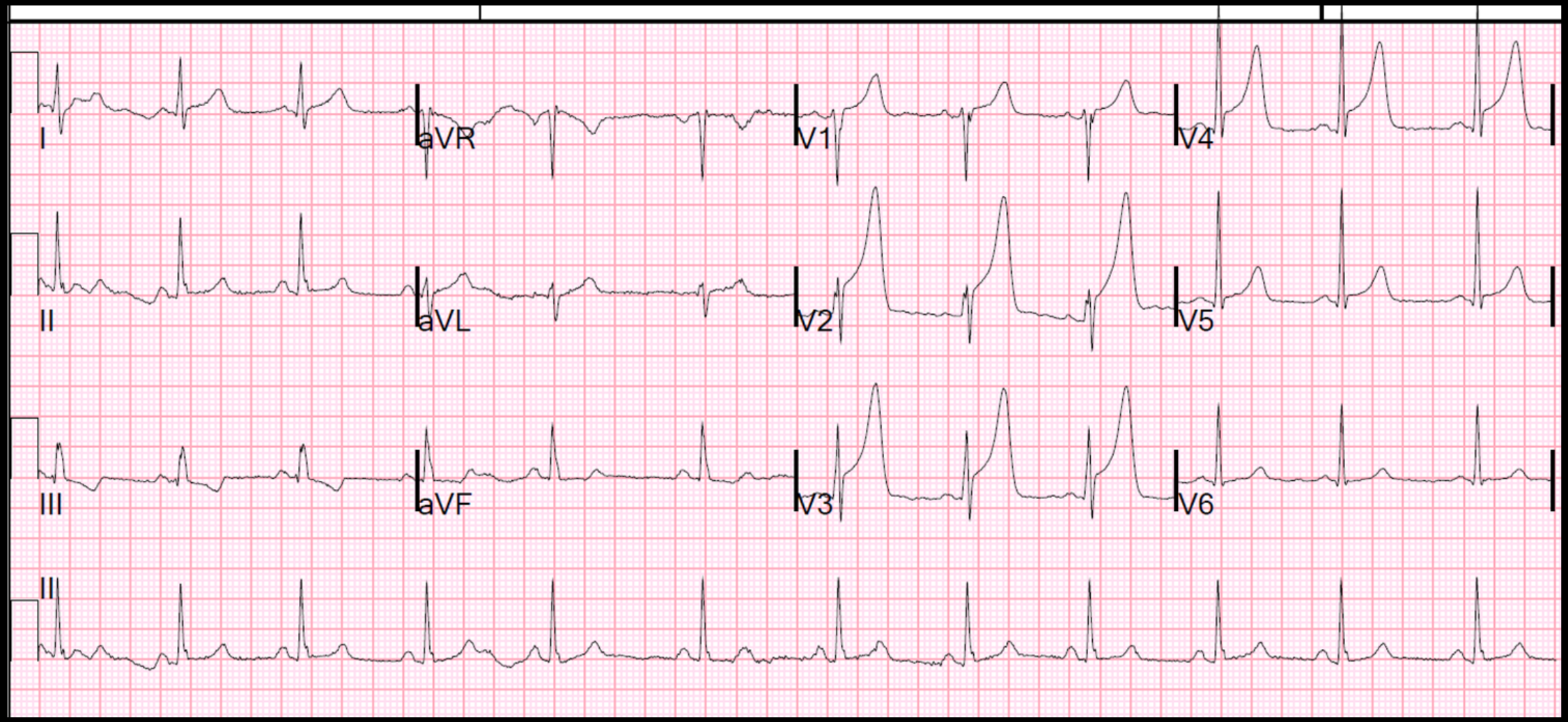
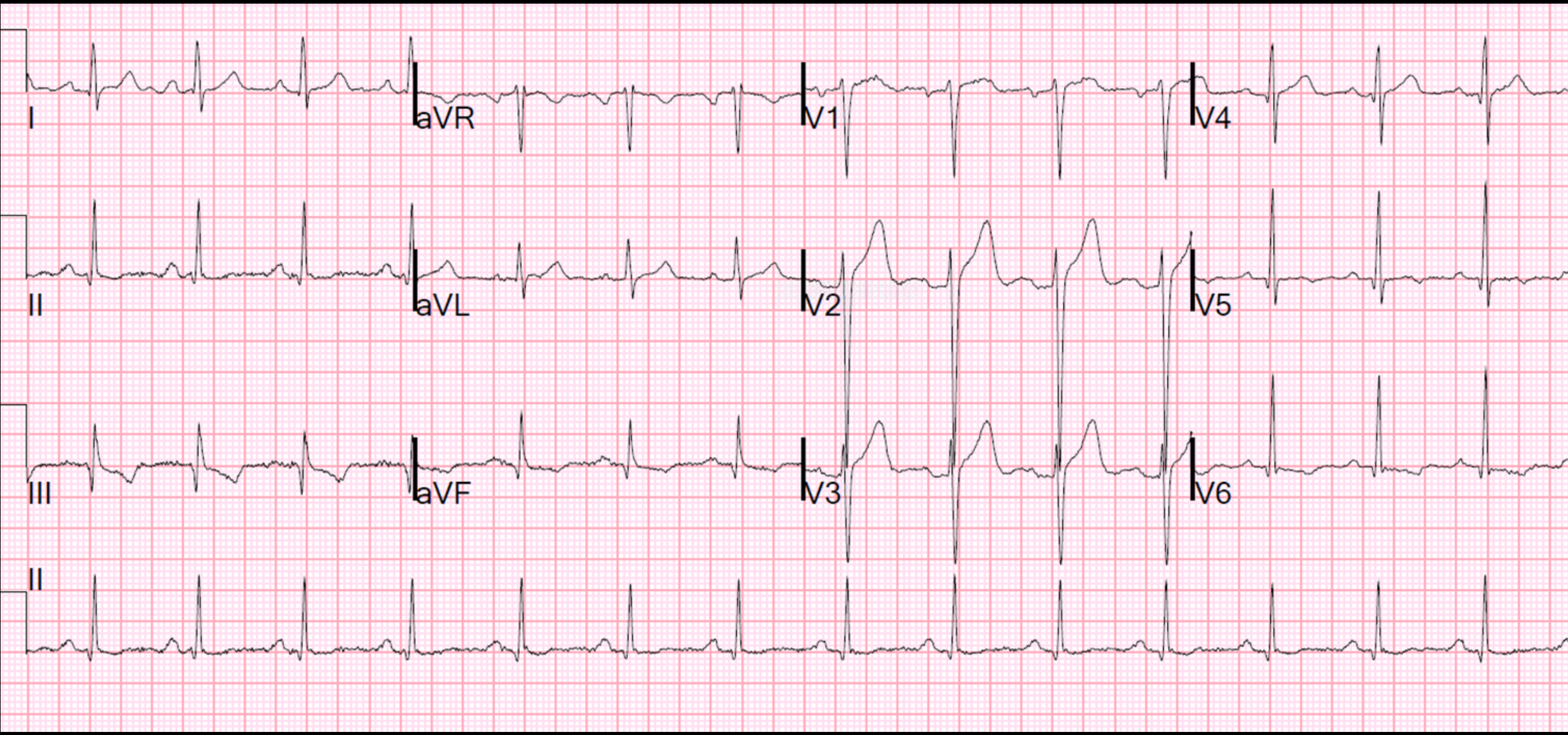
II

Anterior STEMI




I
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V4
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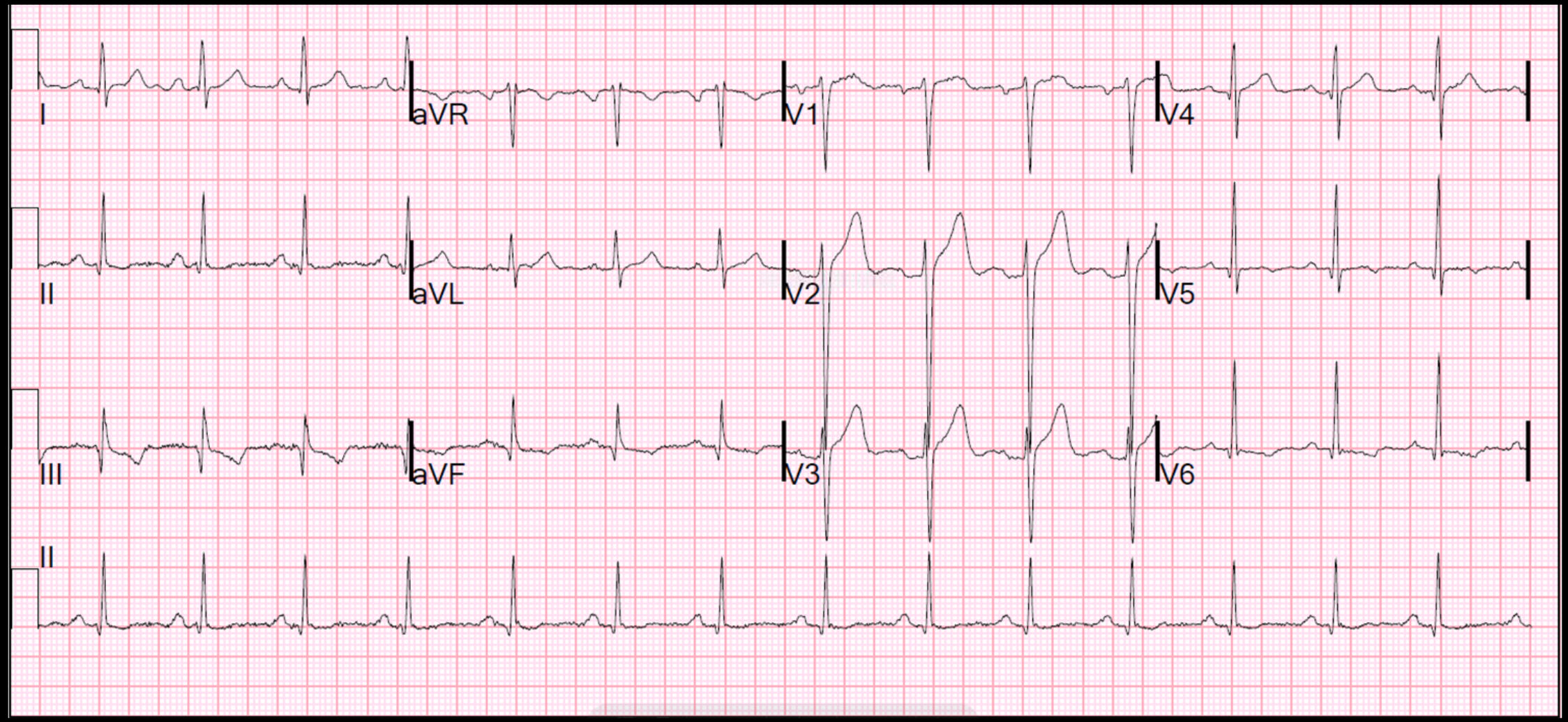
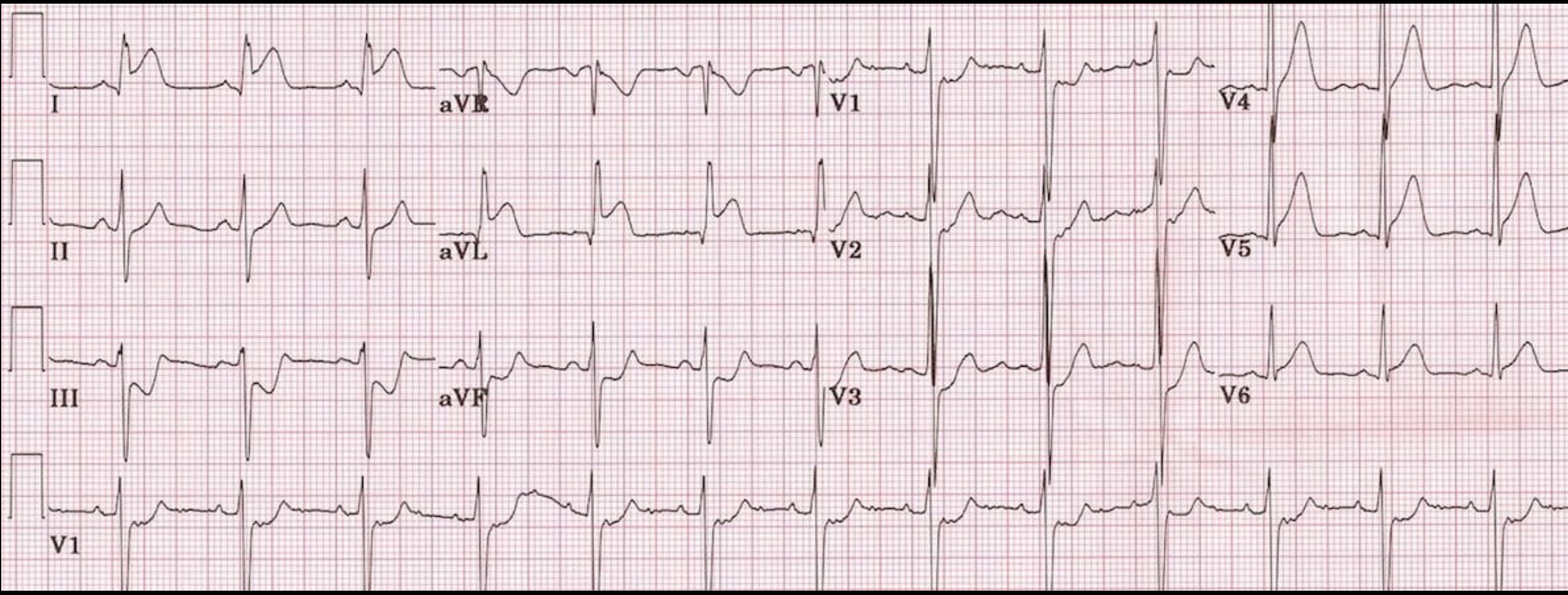
V2
V5
III
aVF
V3
V6
II

Anterior STEMI




I
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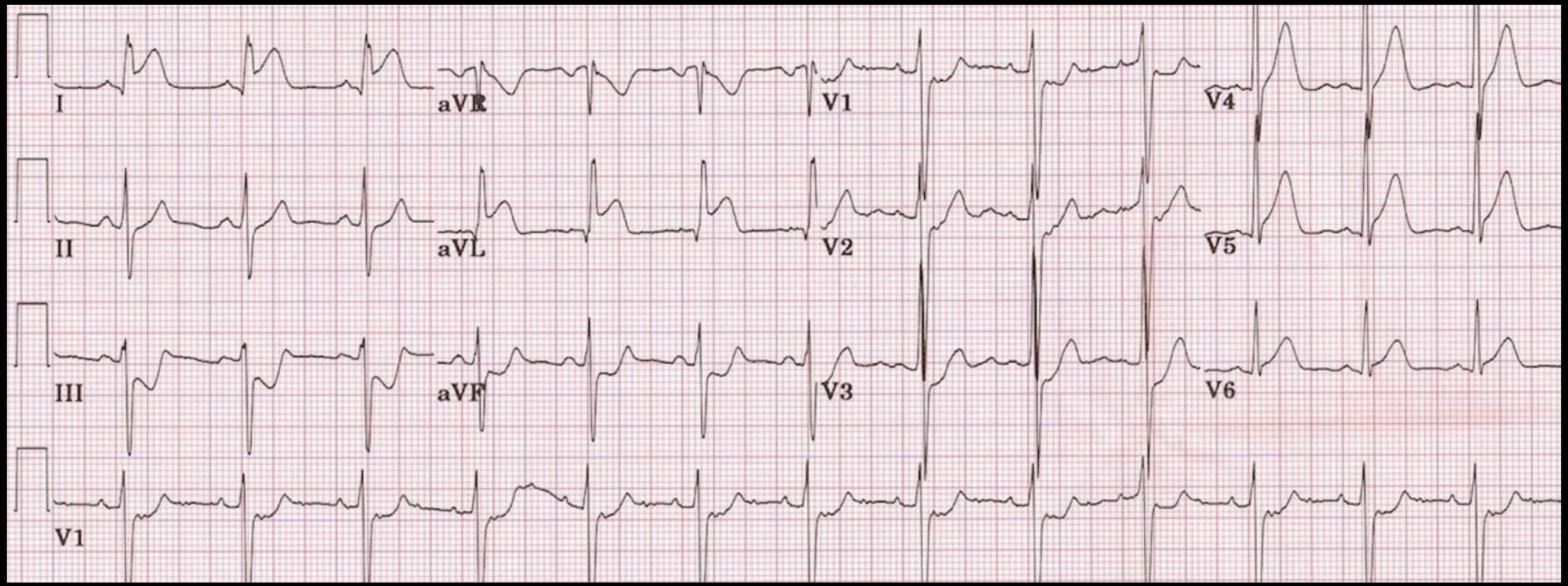
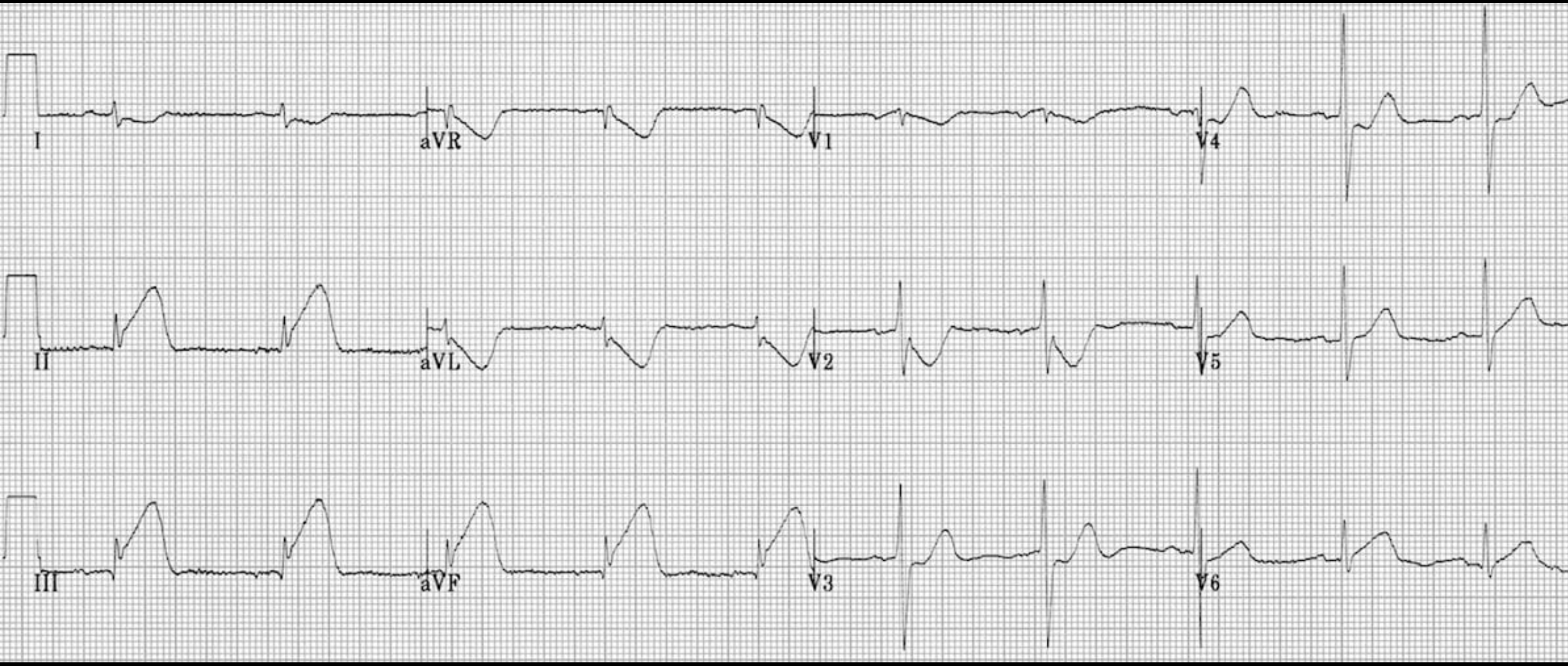
III
aVF
V3
V6
V1

High Lateral STEMI




I
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V4
II
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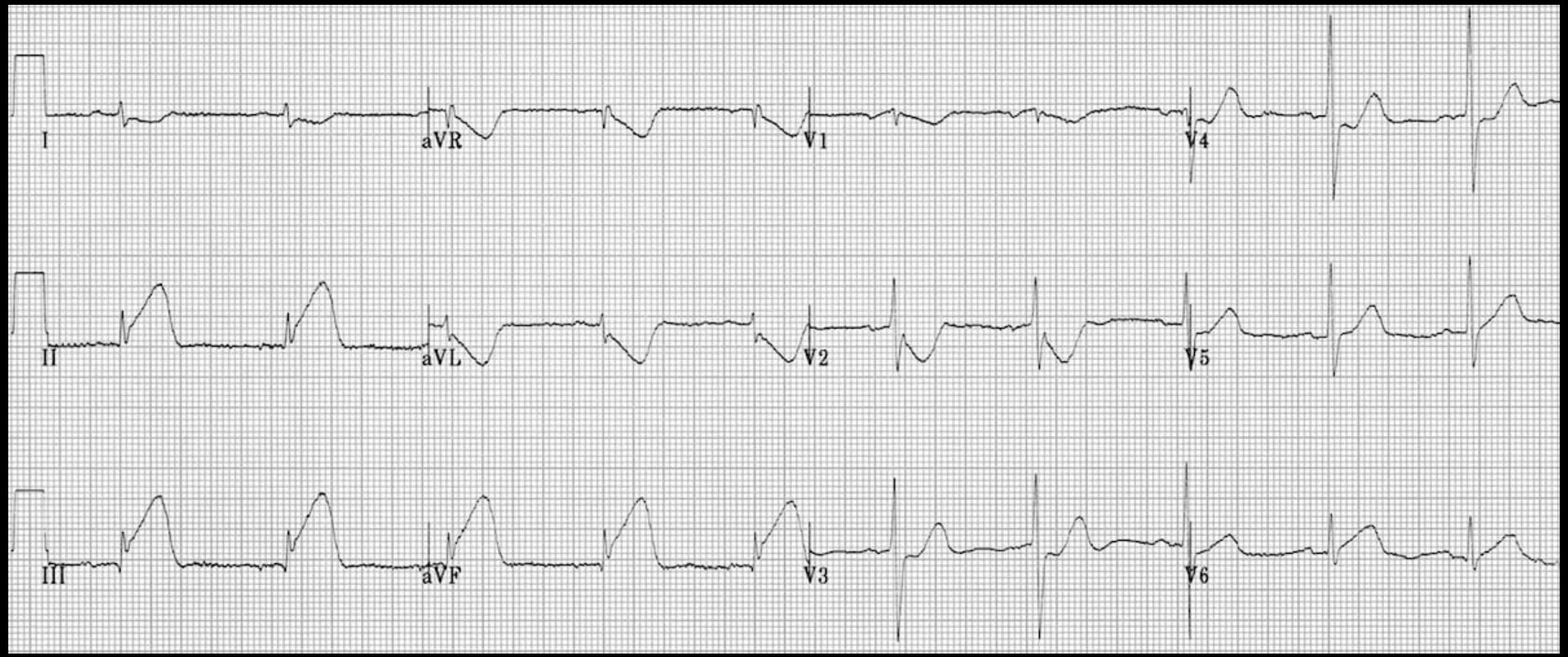
V2
V5
III
aVF
V3
V6

Inferior STEMI



Case

- 48 year old man complaining of chest pain and SOB that began 2 hours prior at work and was becoming progressively worse.
- The pain was constant, pressure-like, substernal, without radiation, and was 10/10 in intensity.
- He is diaphoretic and vomited once
- BP was 213/128.
- Here was his triage ECG: what is worrying?

  what is reassuring?

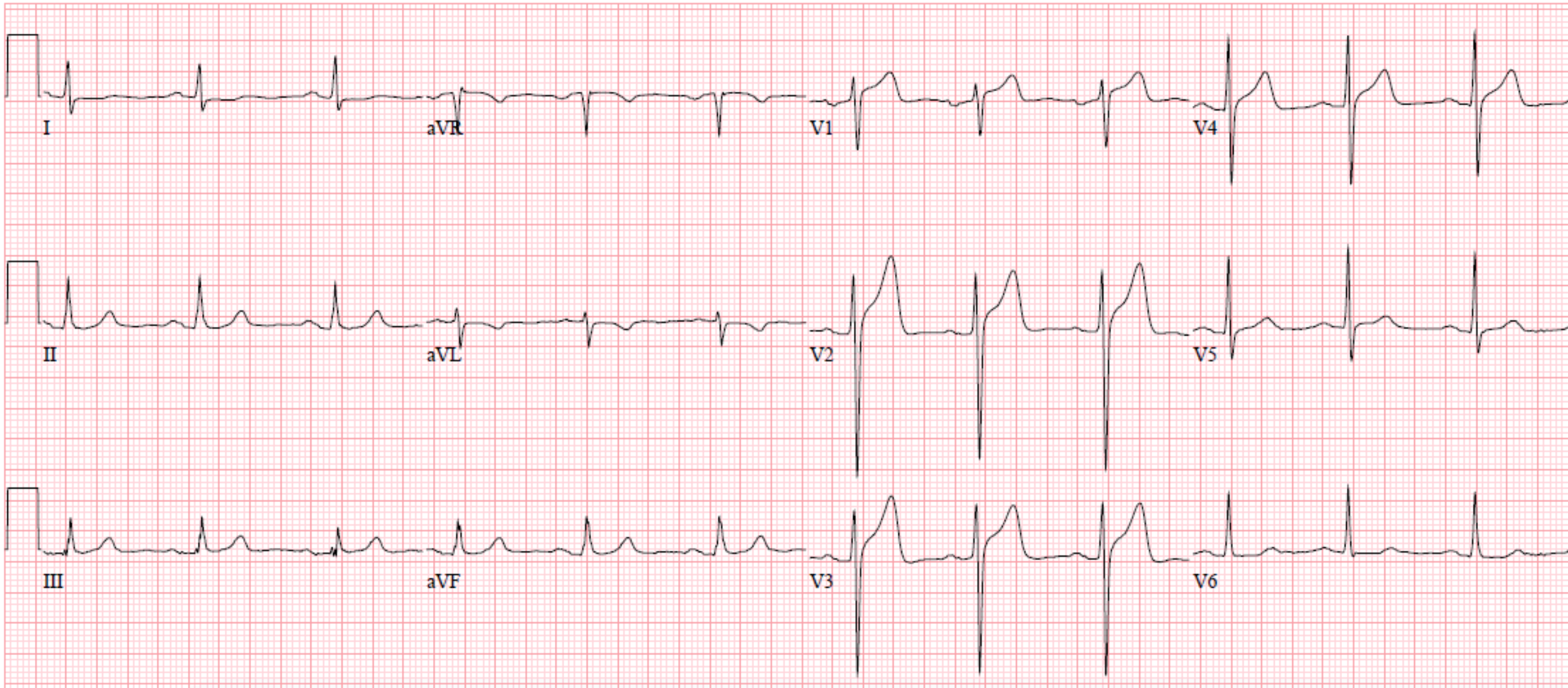
Room:CARD CATH
Loc:1000

Q1/Q1c	382/418	ms	
P-R-T axes	53	74	87

Abnormal ECG
No previous ECGs available
Confirmed by BARBANT, SOPHIE (4244) on 9/29/2020 11:59:52 AM

Technician: B BUSCHELL
Test ind:Chest pain, unspecified

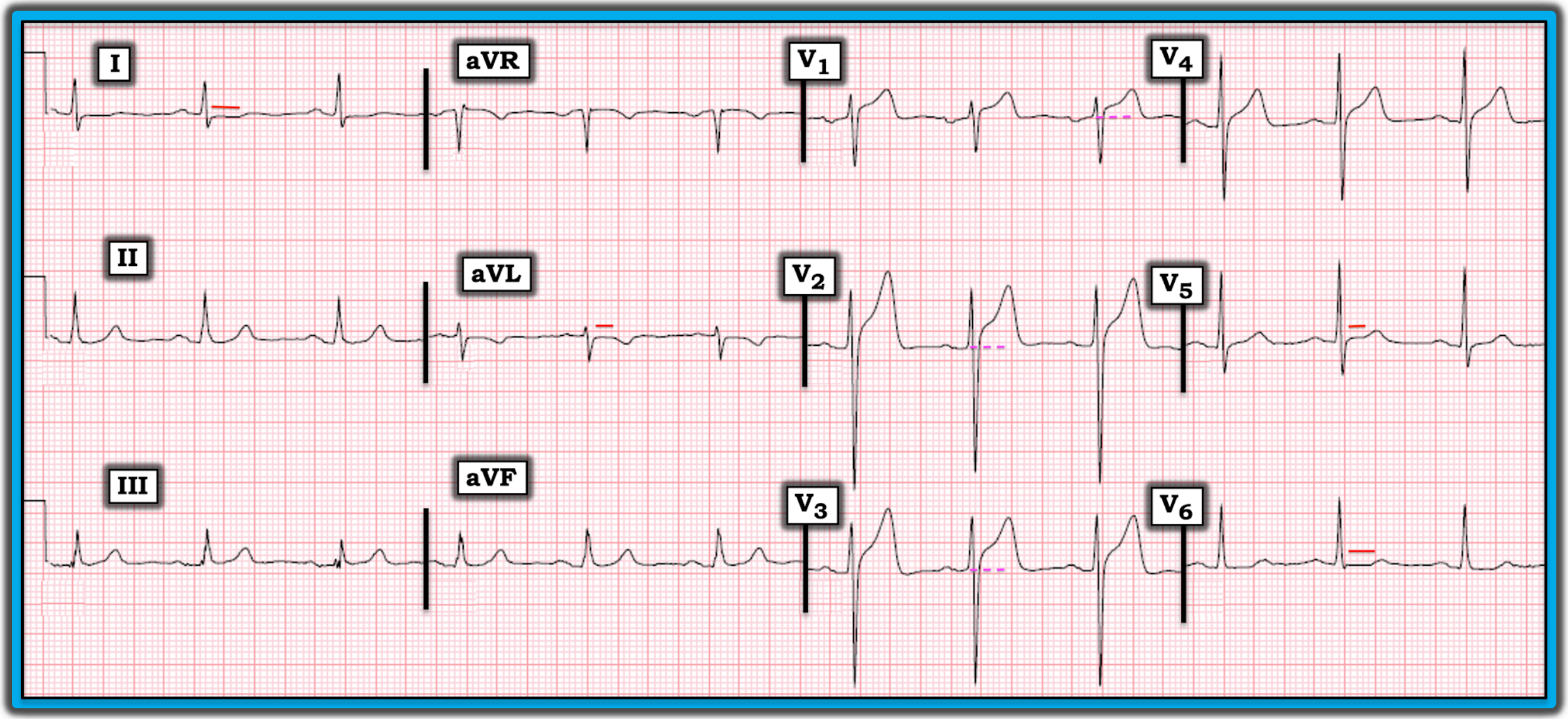
Confirmed By: SOPHIE BARBANT



BER?

- There are asymmetric T-waves (slower upstroke, faster downstroke)
- Upward concavity in all of leads V2-V6 (in this case, even in V1)
- There is no reciprocal ST depression
- There is no terminal QRS distortion (there are S-waves in both V2 and V3)
- There are no Q-waves
- LVH by voltage? (complicates things)

ECG #1 = *initial* ECG in the ED …



Suspicious Findings

- It’s a good history for ischaemia
- There is obvious ST elevation
- There is a big upright T wave in V1 (and its greater than the T wave in V6) and that favours LAD occlusion
- Flat ST segments in several leads
- Flipped T wave in aVL (can be normal if the QRS is predominately negative but I don’t like it)

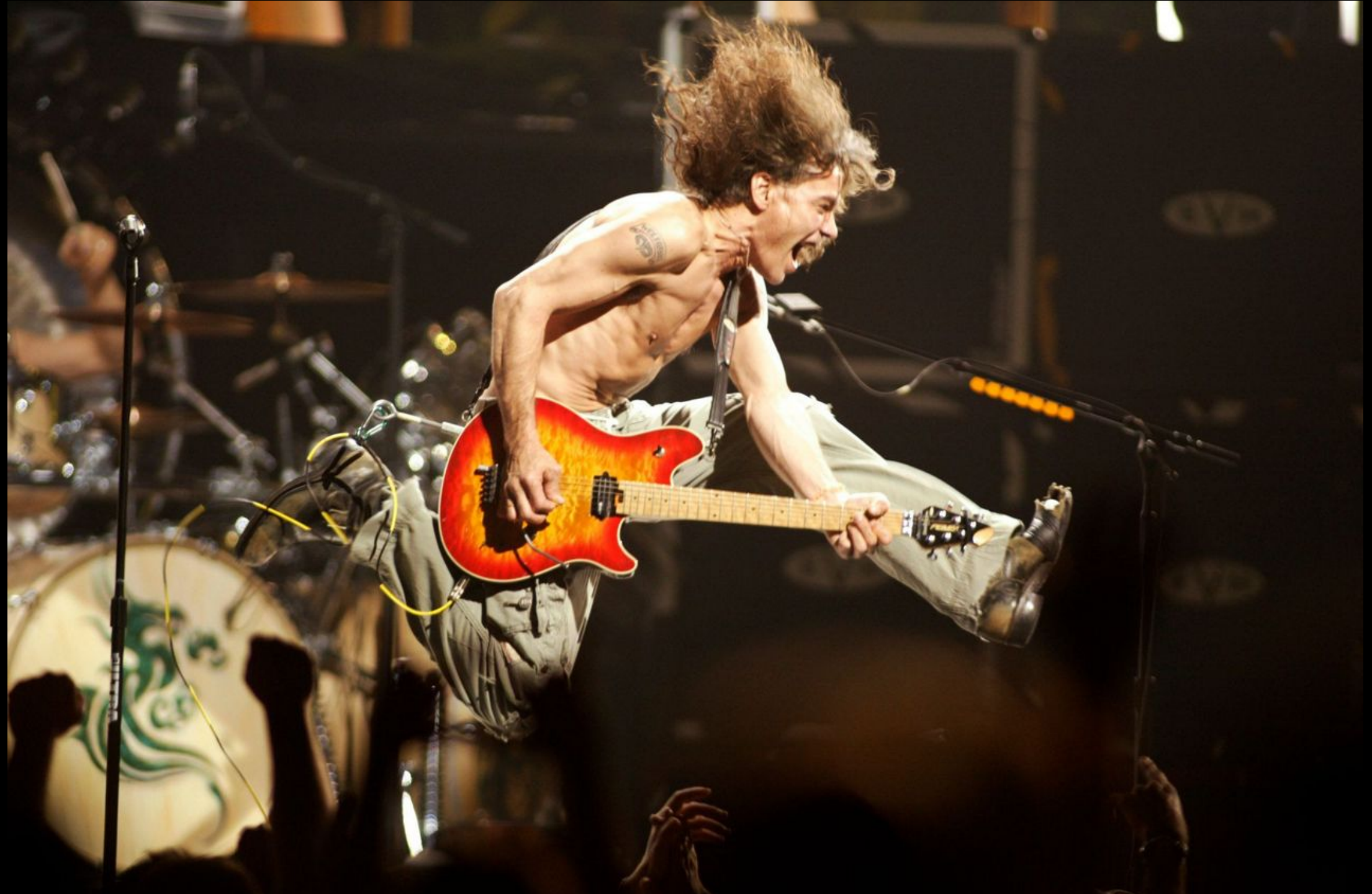
Plan

Plan

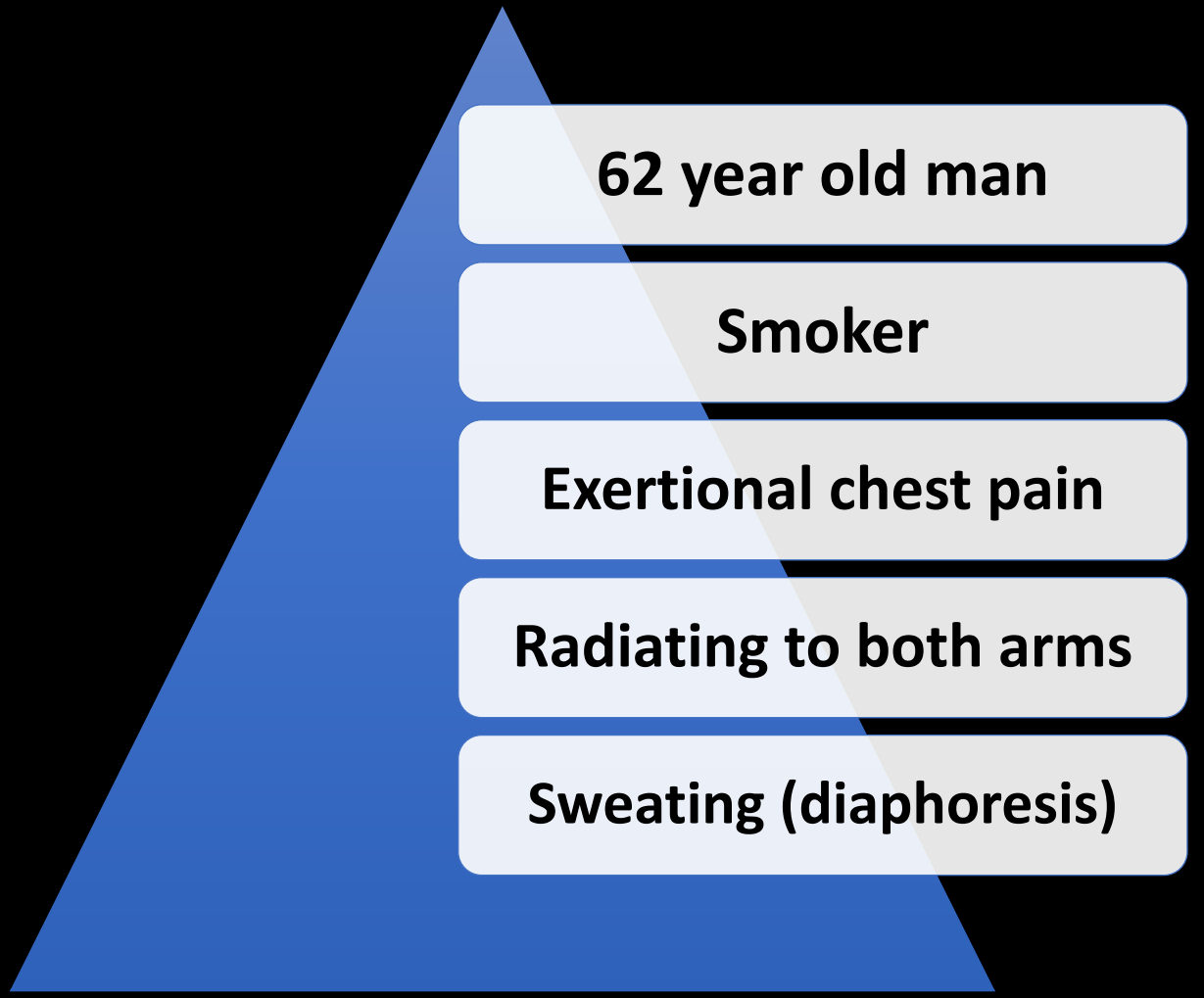
- Based on the history and ECG he was taken to the cath lab
- He had a 100% LAD occlusion

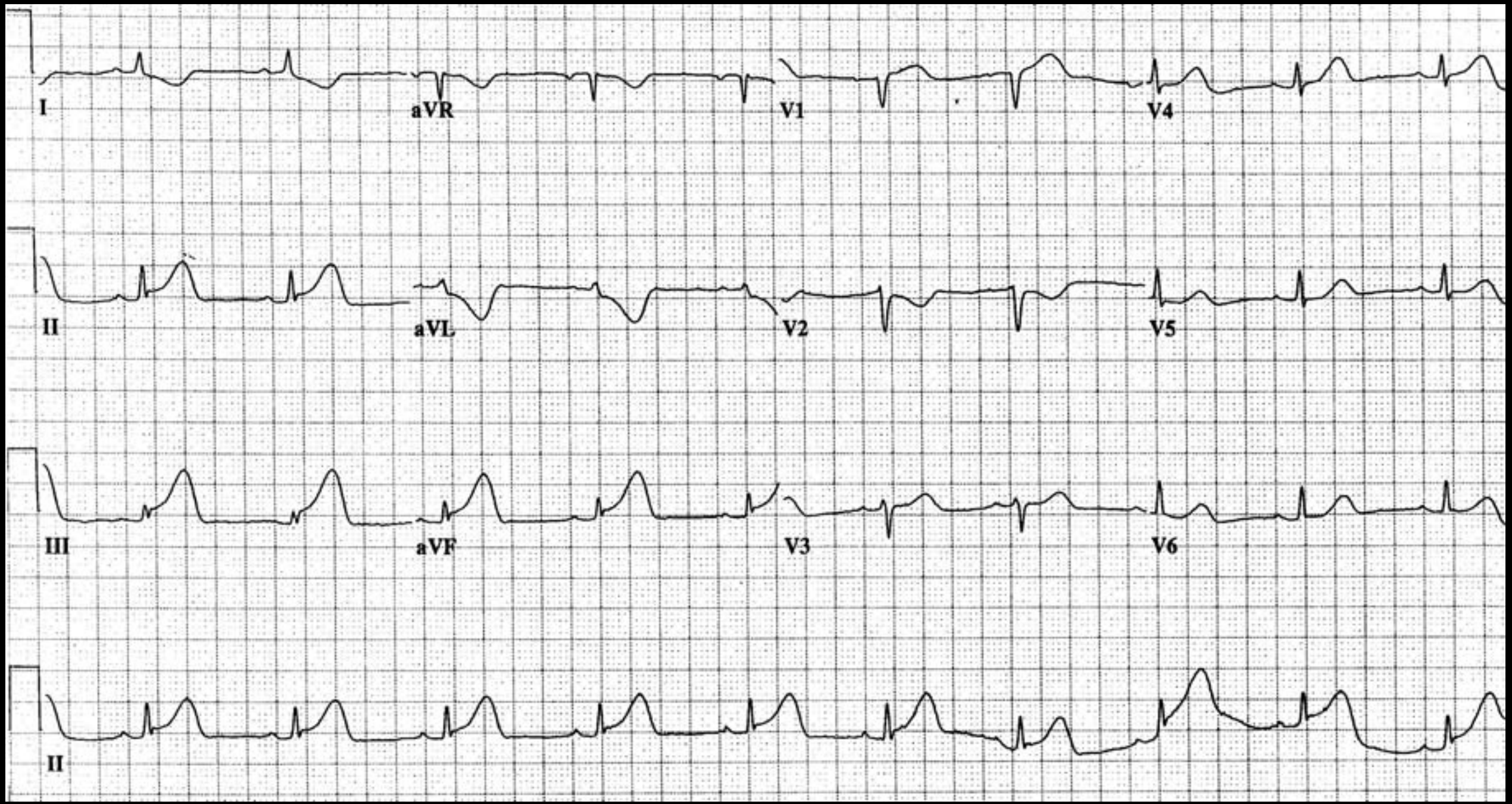
Take Home Points

- You do not have to decide whether or not there is acute AMI on the basis of a single ECG
  - its OK to be unsure
- If in doubt do: serial ECG and look for evolving changes
  find an old ECG
- Bedside ECHO can help
- HS trop (call the lab yell and plead)



Case

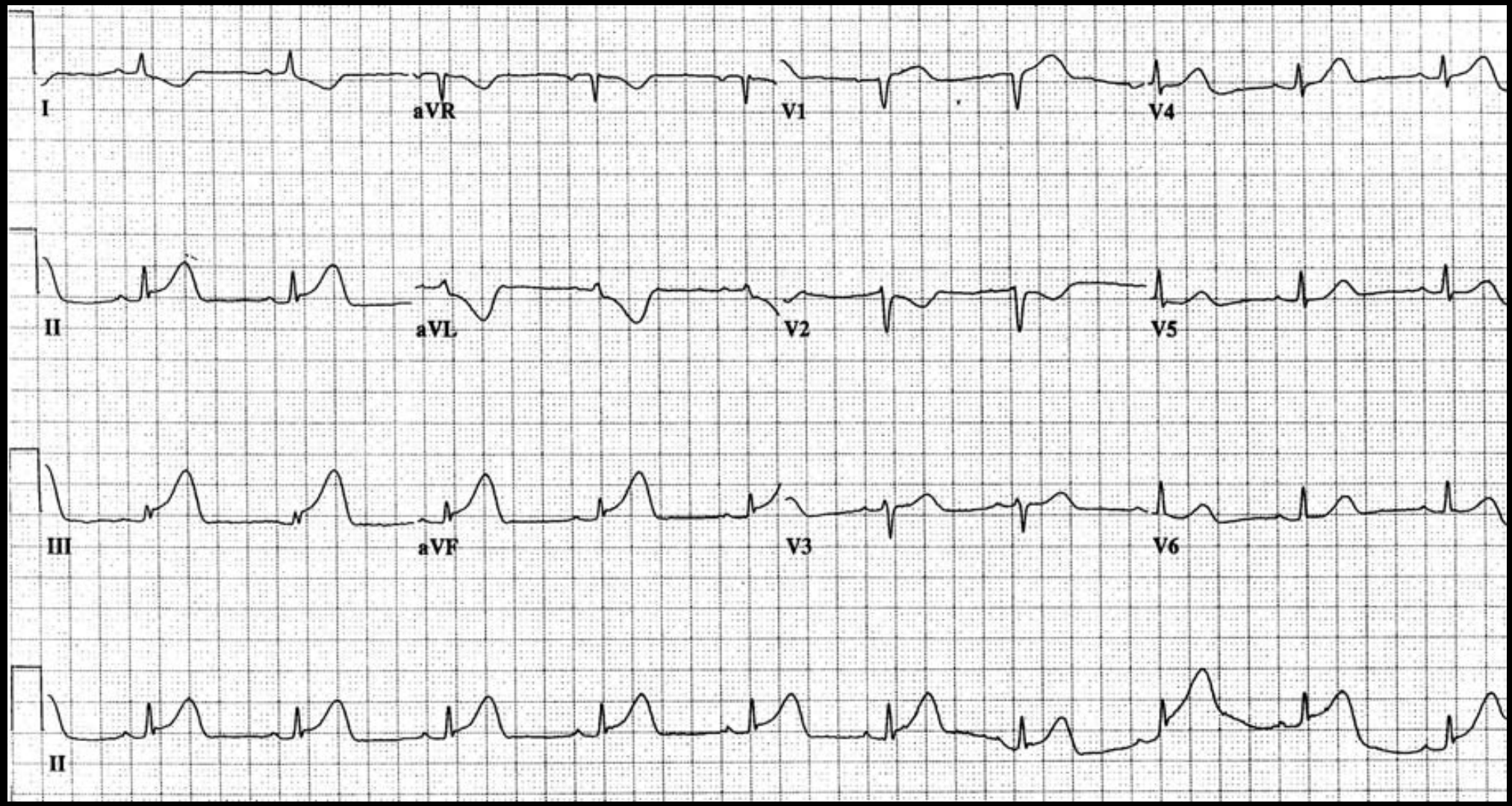

62 year old man
Smoker
Exertional chest pain
Radiating to both arms
Sweating (diaphoresis)


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

ECG Interpretation: Inferior “STEMI”

- Regular Sinus Rhythm
  - at a rate of 66
- Normal Axis
- Subtle ST elevation in inferior leads?
- Reciprocal ST depression in the lateral leads
- Hyperacute T wave sin the inferior leads

What Else could be going on in this ECG?



Inferior STEMI with Likely RV Extension

- Consider RV involvement as well as
  - Subtle ST elevation in V1
  - ST depression in V2
  - ST elevation III > II

RV Infarct: Do I Care?

- Isolated RV infarct is rare
- But always need to think of RV involvement when you have an inferior MI

  - higher Mortality

  - risk of Heart Blocks
- Right Ventricle is “pre-load dependent” and this changes our management
  - Give fluids (RV pre-load dependent to maintain blood pressure)
  - Careful with nitrates
  - Careful with morphine

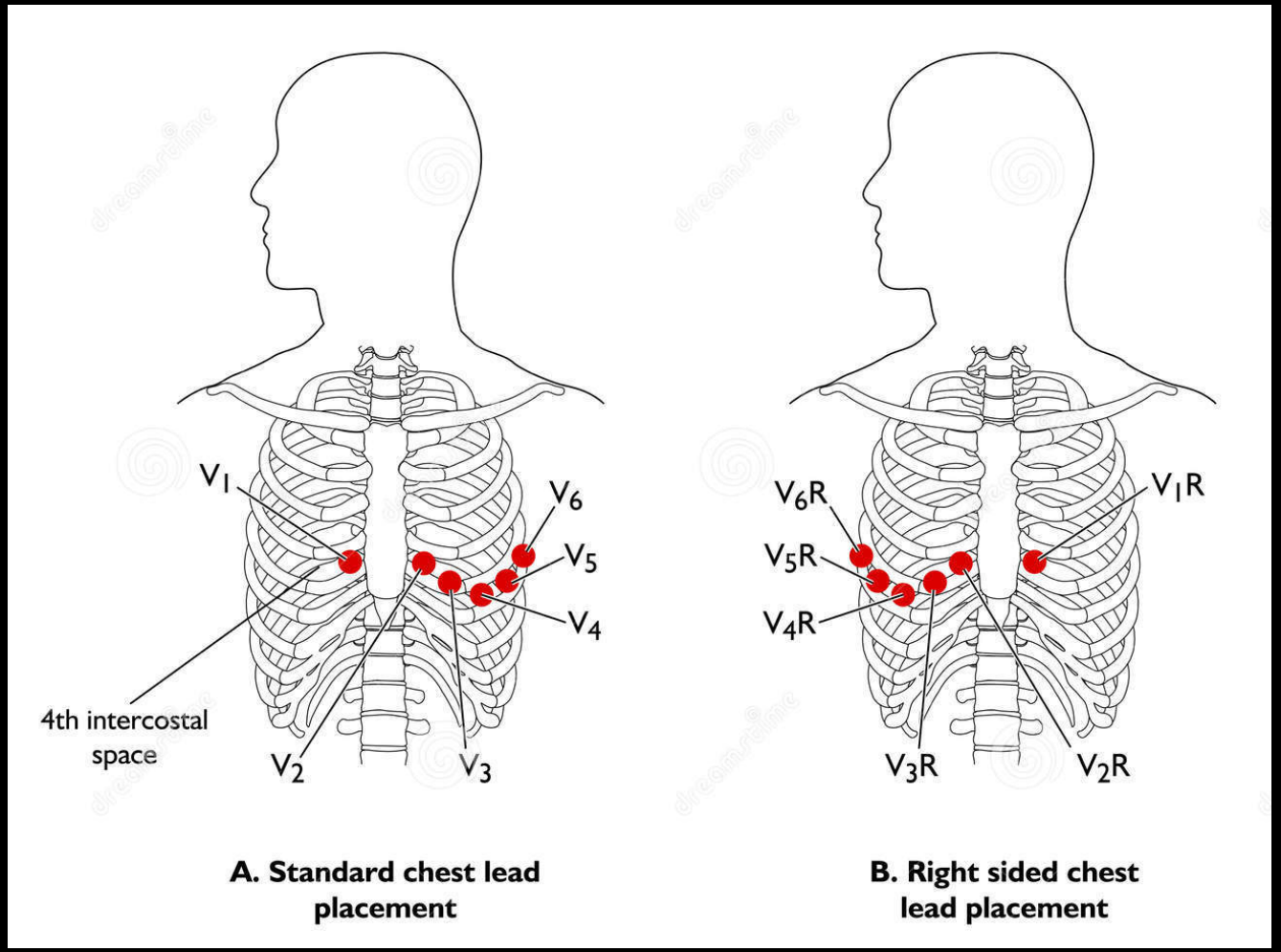
RV Infarct: ECG Changes

- ST elevation in V1
- ST depression in V2 with either
  - ST segment elevation in V1
  - ST segment is isoelectric in both V1 and V3
- ST elevation in III > II

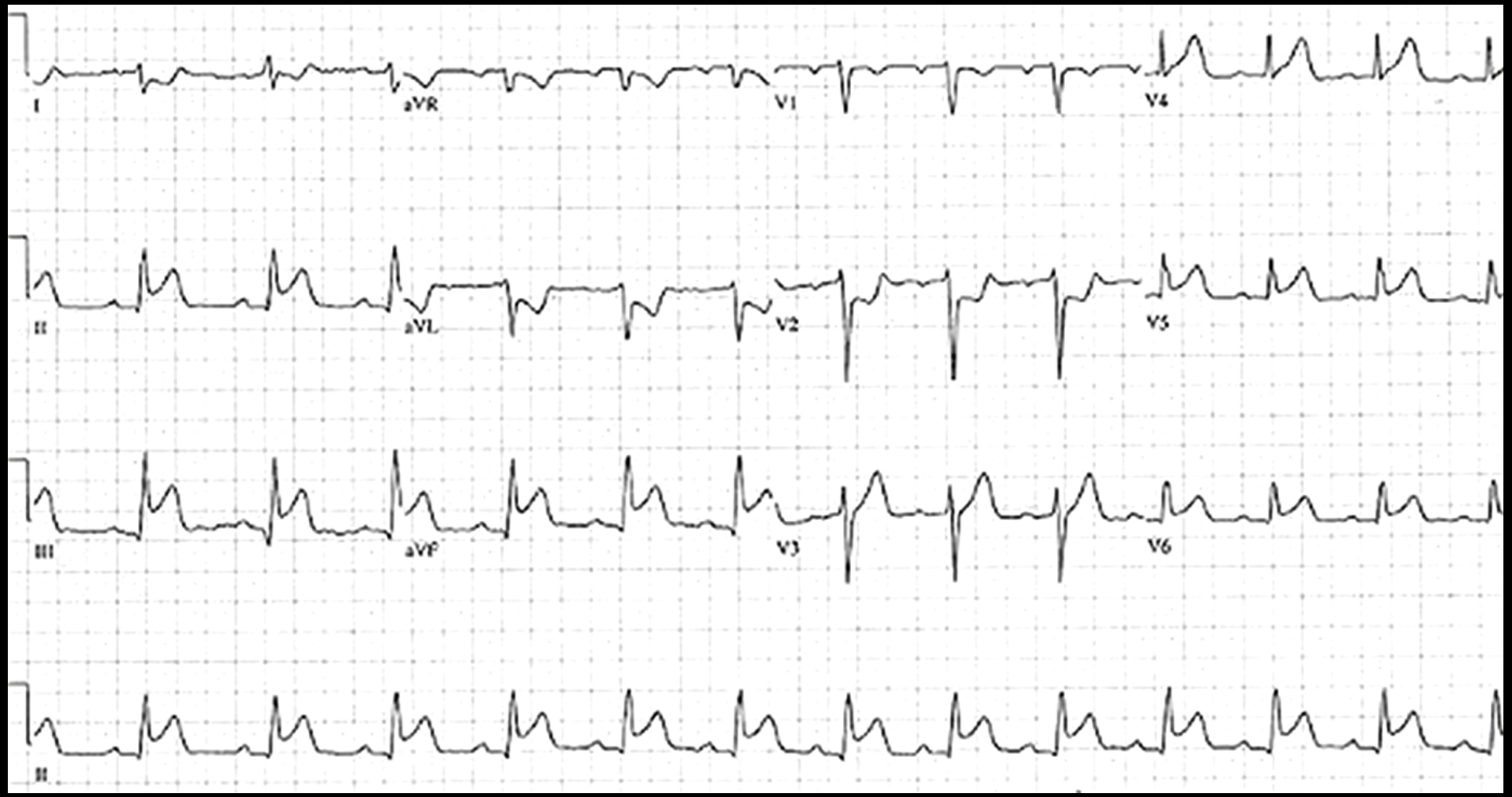
If you suspect RV involvement, get a right-sided ECG

Right Sided ECG

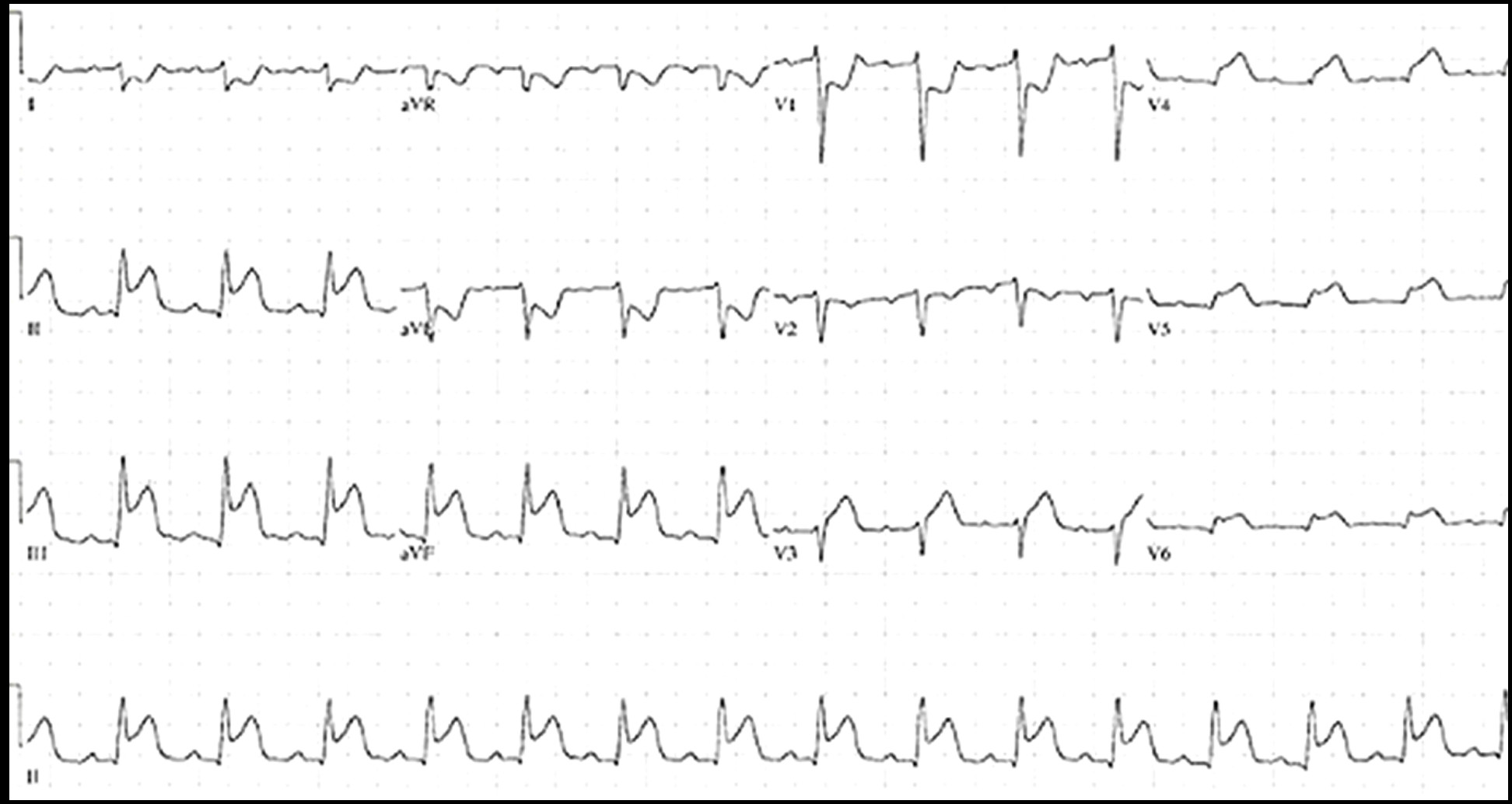
- Any ST elevation in right sided leads
  - RV is small so ST elevation can be subtle
  - rV4 is the money shot



Normal Lead Position



Right-sided Leads



RV Infarction

- Bit hard to remember the rules
- So consider getting “right sided leads” for all your inferior Acute Myocardial Infarctions

  - before you give nitrates and morphine

  - even a hint of ST elevation is likely a Right Ventricular infarction

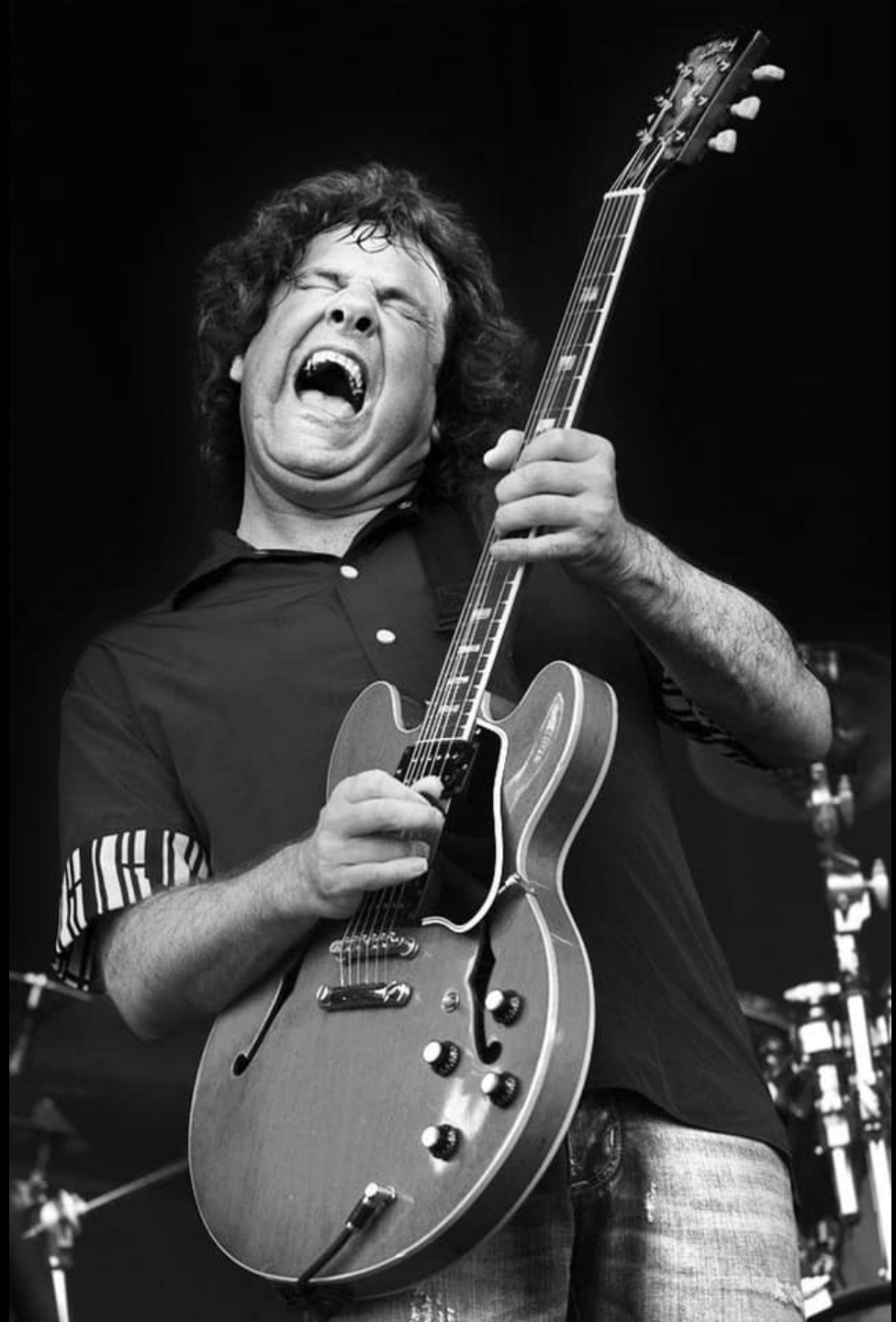
Inferior STEMI: Summary

- ST elevation in inferior leads
- ST depression in lateral leads

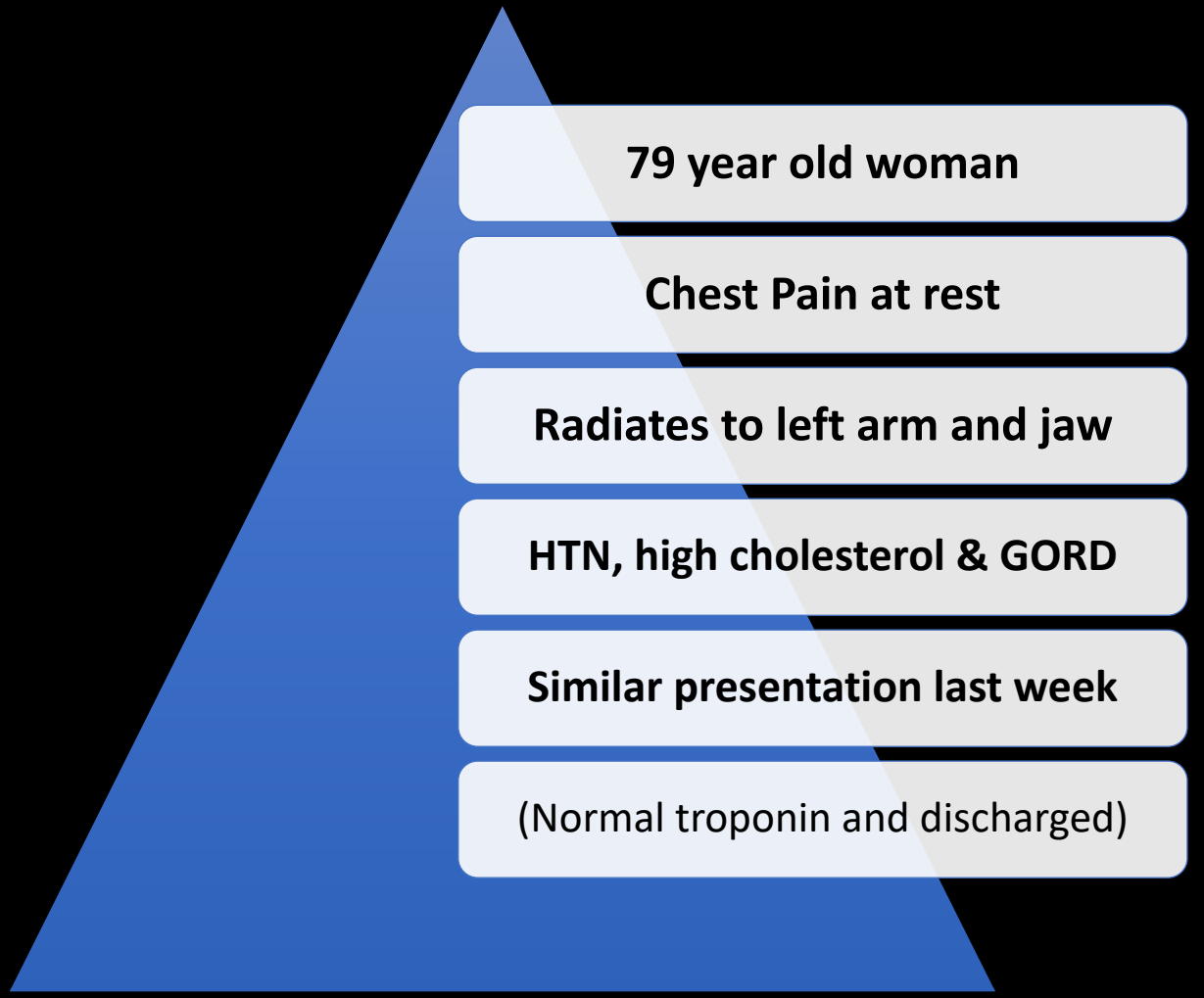
  - especially aVL
- Always consider the possibility of RV or posterior wall of LV involvement

  - a posterior wall infarction does not effect your management

  - get right-sided leads



Case


79 year old woman
Chest Pain at rest
Radiates to left arm and jaw
HTN, high cholesterol & GORD
Similar presentation last week
(Normal troponin and discharged)

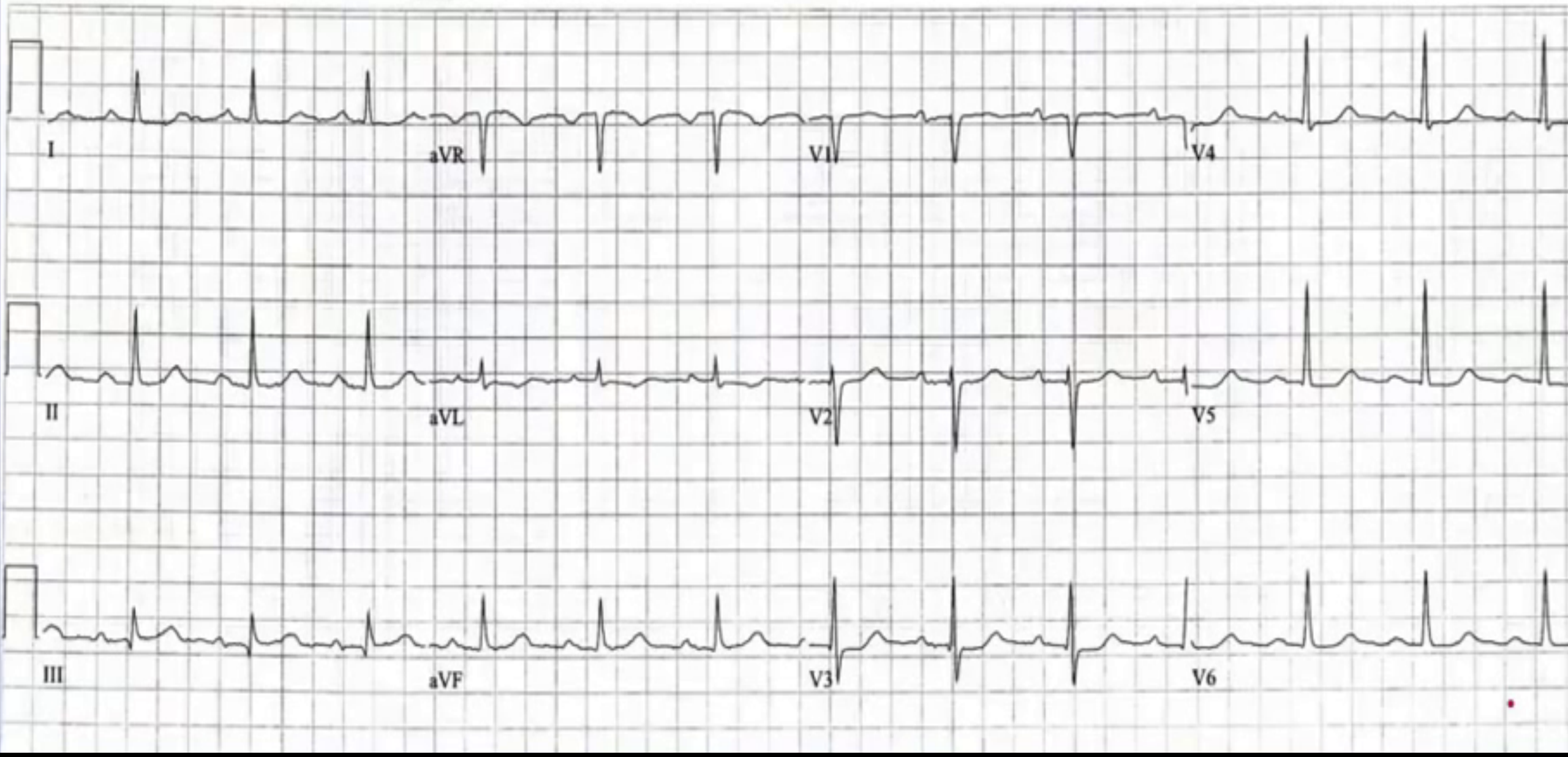
Test ind:

Referred by: [redacted]

Unconfirmed

TECH NOTES:

CRIC:



The ECG

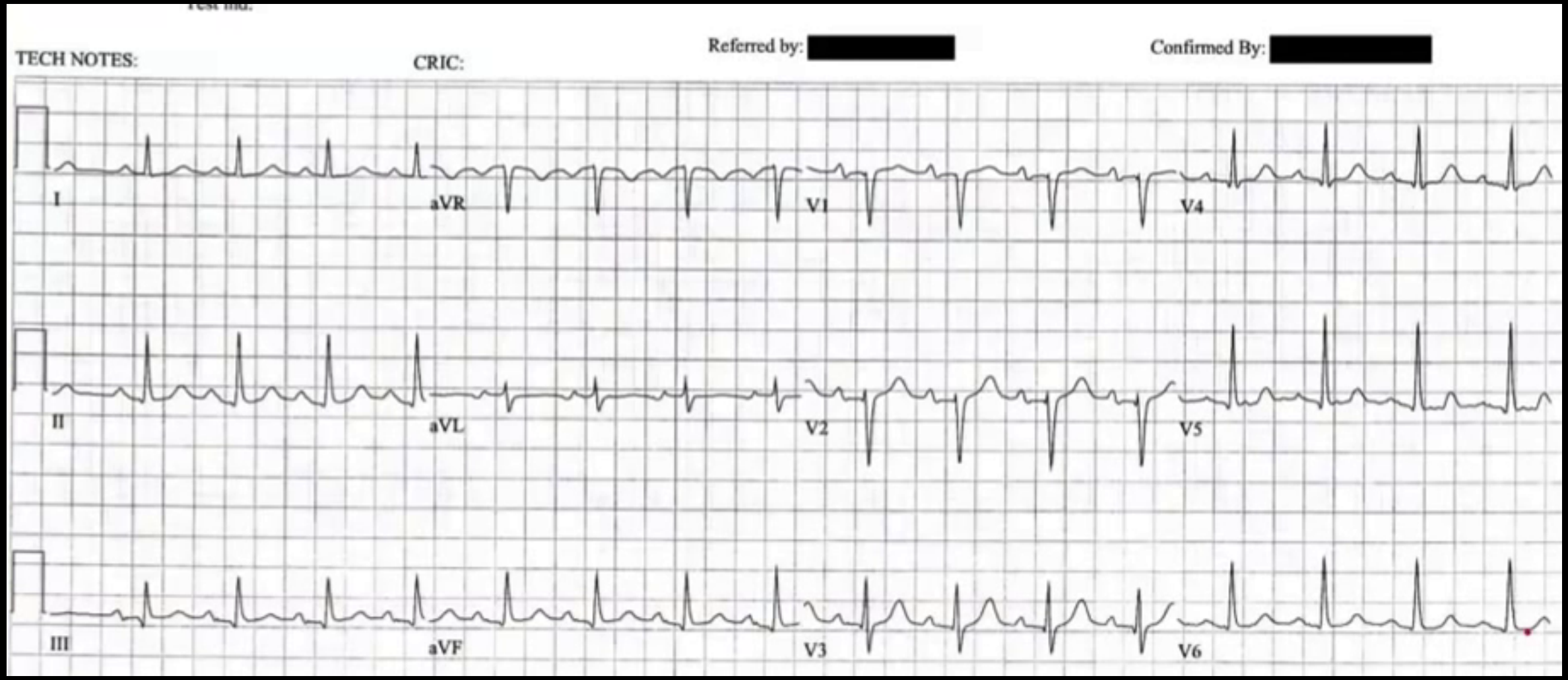
- Subtle ST depression in V2 – V5
- Subtle ST depression in aVL with T wave inversion
  - QRS up and T wave down
- Subtle ST elevation in Lead III?
  - probably not if you use the TP segment as your baseline

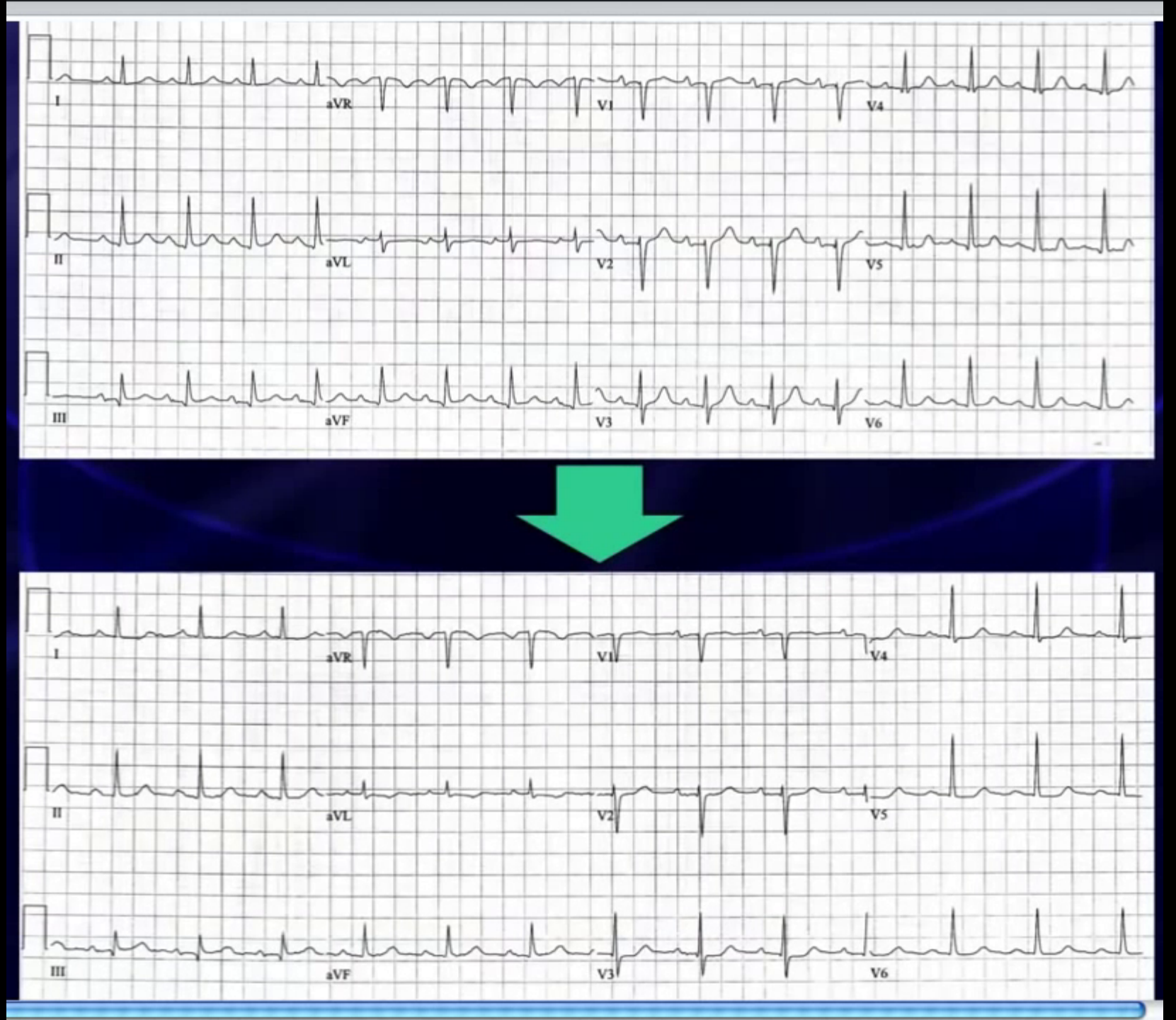
Now what?

Now What

- Get old ECG
- Repeat ECG every 10 minutes
- Follow chest pain algorithm

Old ECG


TECH NOTES:
CRIC:
Referred by: [redacted]
Confirmed By: [redacted]
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

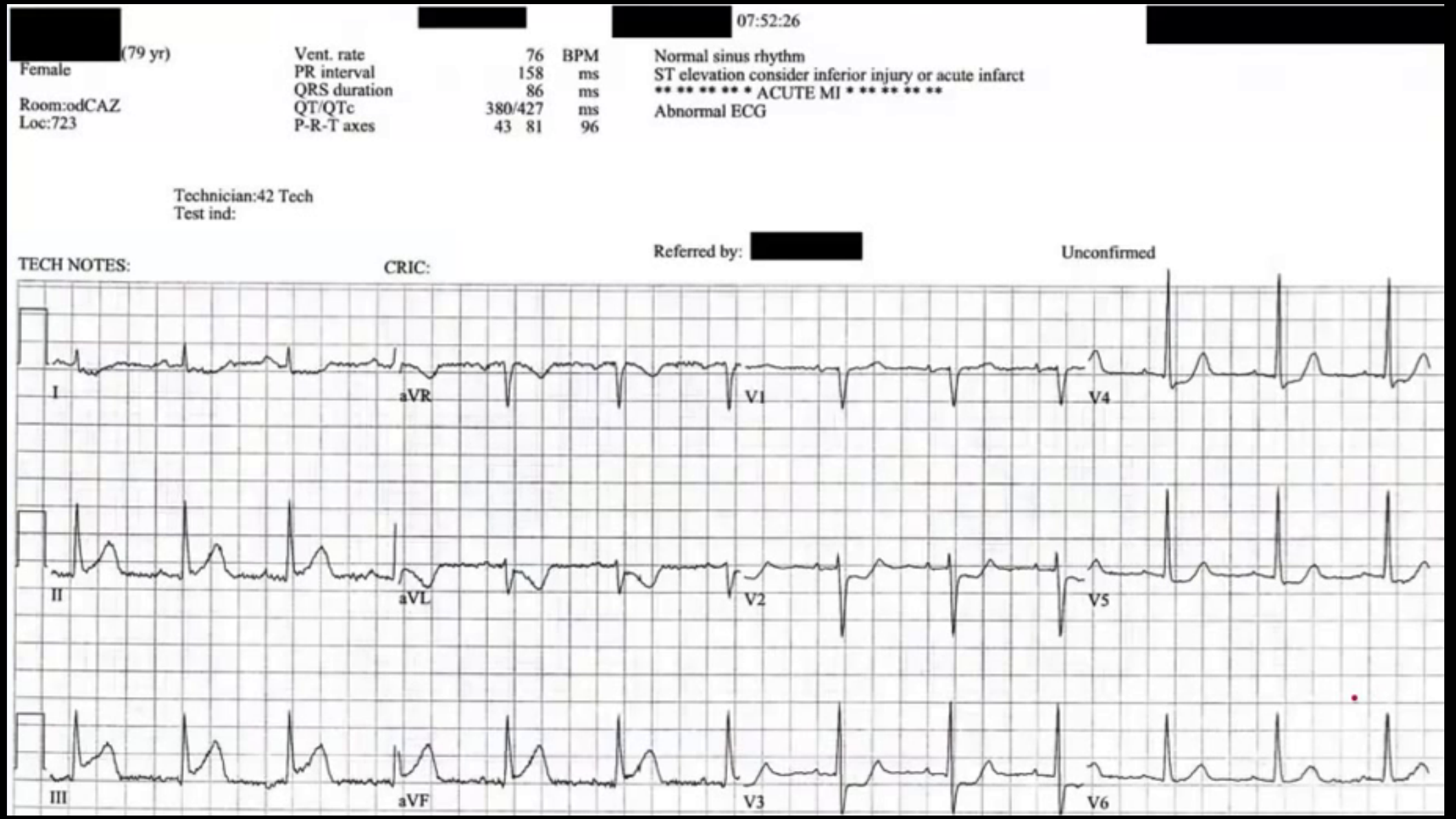

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

The ECG's

- There are new changes
  - The ST depression in aVL is new
  - The ST depression in V2 – V4 is more pronounced
  - subtle ST elevation in III (maybe)

Due to lead placement or something else?

Serial ECG


[redacted] (79 yr)
Female
Room:odCAZ
Loc:723
Vent. rate 76 BPM
PR interval 158 ms
QRS duration 86 ms
QT/QTc 380/427 ms
P-R-T axes 43 81 96
[redacted] 07:52:26
Normal sinus rhythm
ST elevation consider inferior injury or acute infarct
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG
Technician:42 Tech
Test ind:
Referred by: [redacted]
Unconfirmed
TECH NOTES:
CRIC:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

The ECG

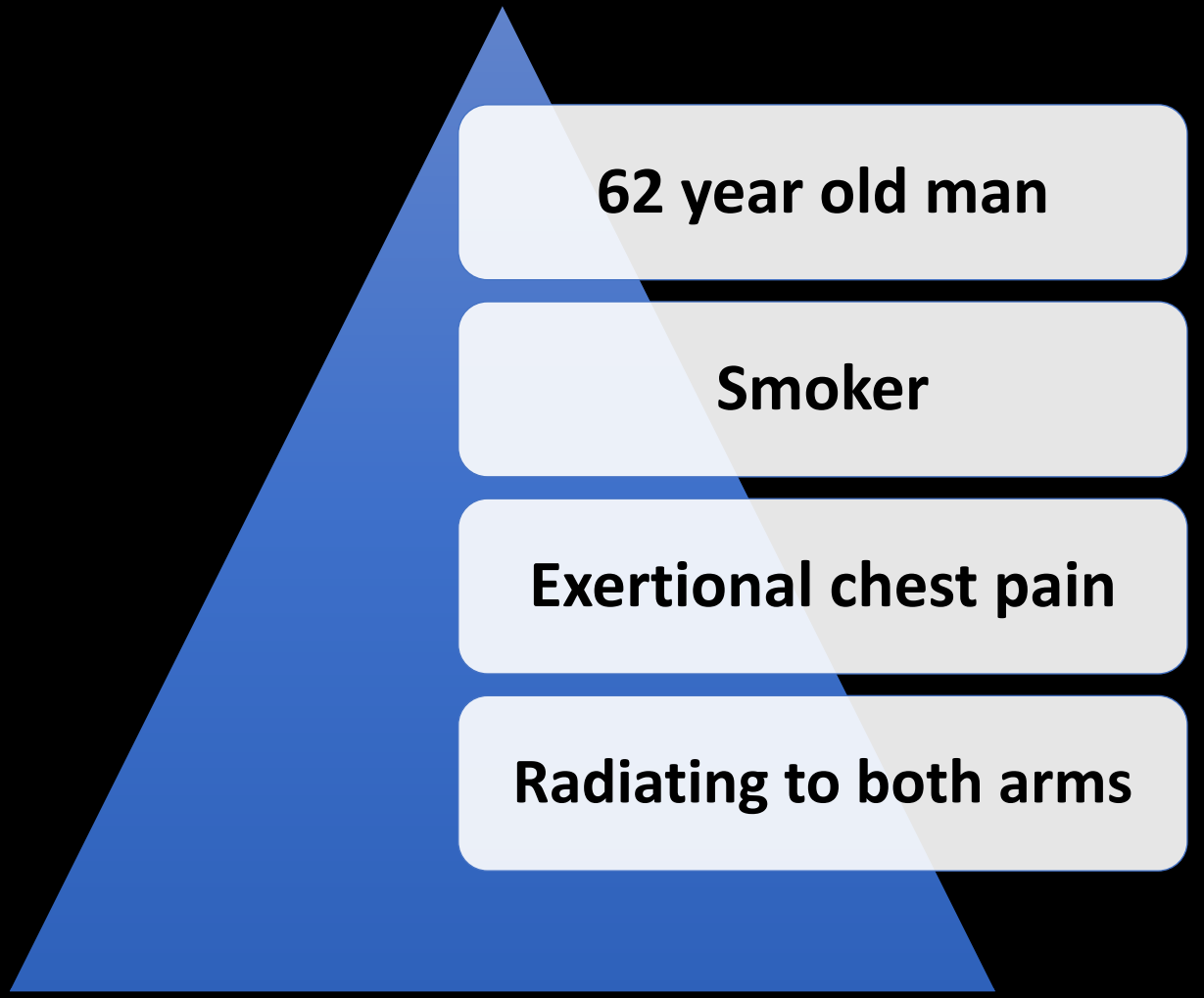
- Inferior STEMI
- And……

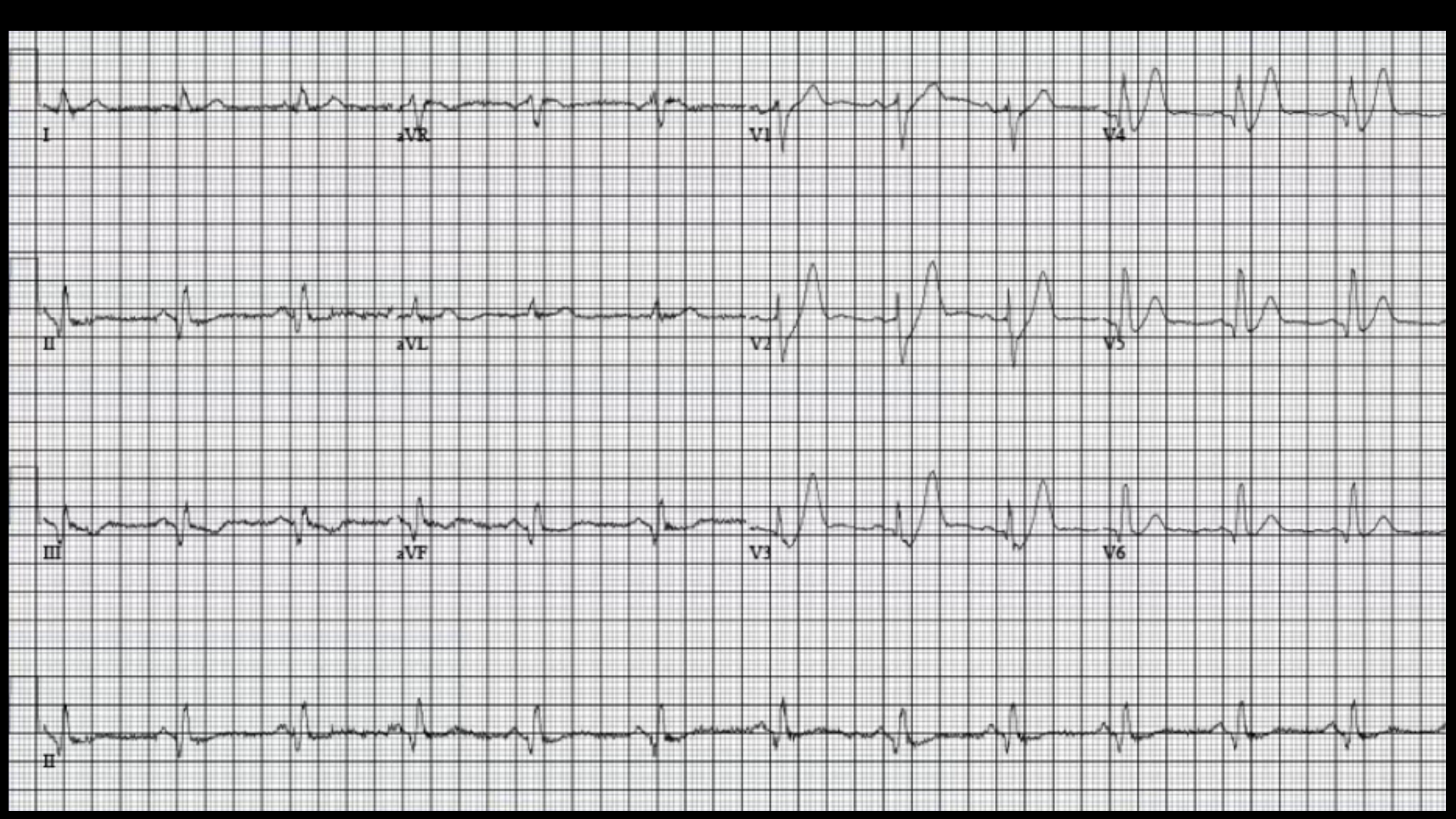
aVL: Summary

- TWI and / or subtle ST depression in aVL needs to be taken seriously
  - can be the first sign of an inferior AMI
    ie reciprocal changes in aVL can precede ST elevation in the inferior leads
- Not enough by itself to activate the cardiac catheter lab?
  - serial ECG's (till the nurse yells at you)
  - find an old ECG


Repost alanpaul66

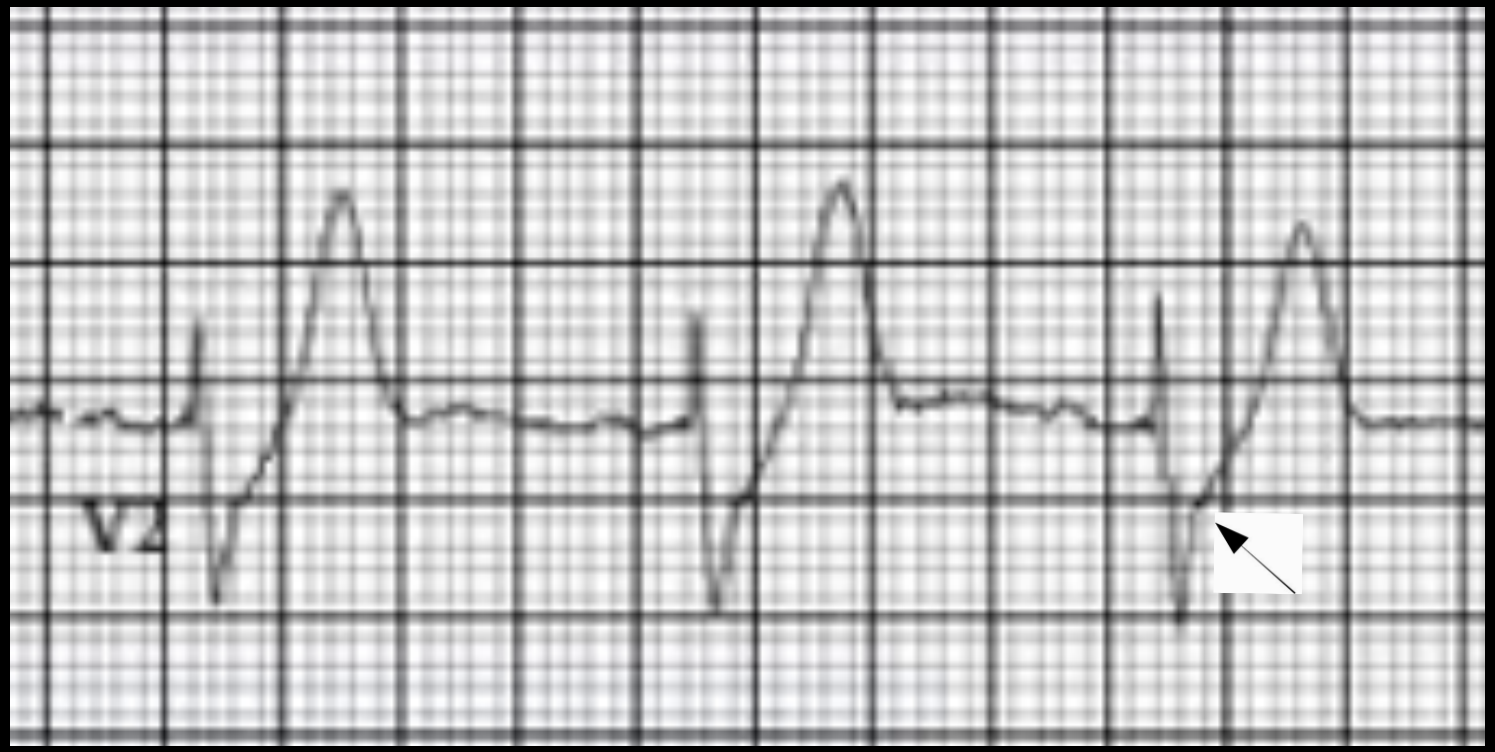
Case


62 year old man
Smoker
Exertional chest pain
Radiating to both arms


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

ECG

- SR at a rate of 72
- Normal axis
- No ST elevation
- TWI inferior leads
- Prominent / chunky / startling T waves in V2 to V4
- Q’s in inferior leads without STE
  - old AMI?



- Up-sloping ST depression
- J point is depressed
- The ST segment seems to run straight into a large, broad based T wave

The ECG

- SR at 72
- No ST elevation
- Normal axis
- DeWinter's T Waves V2 to V4
  - acute LAD occlusion

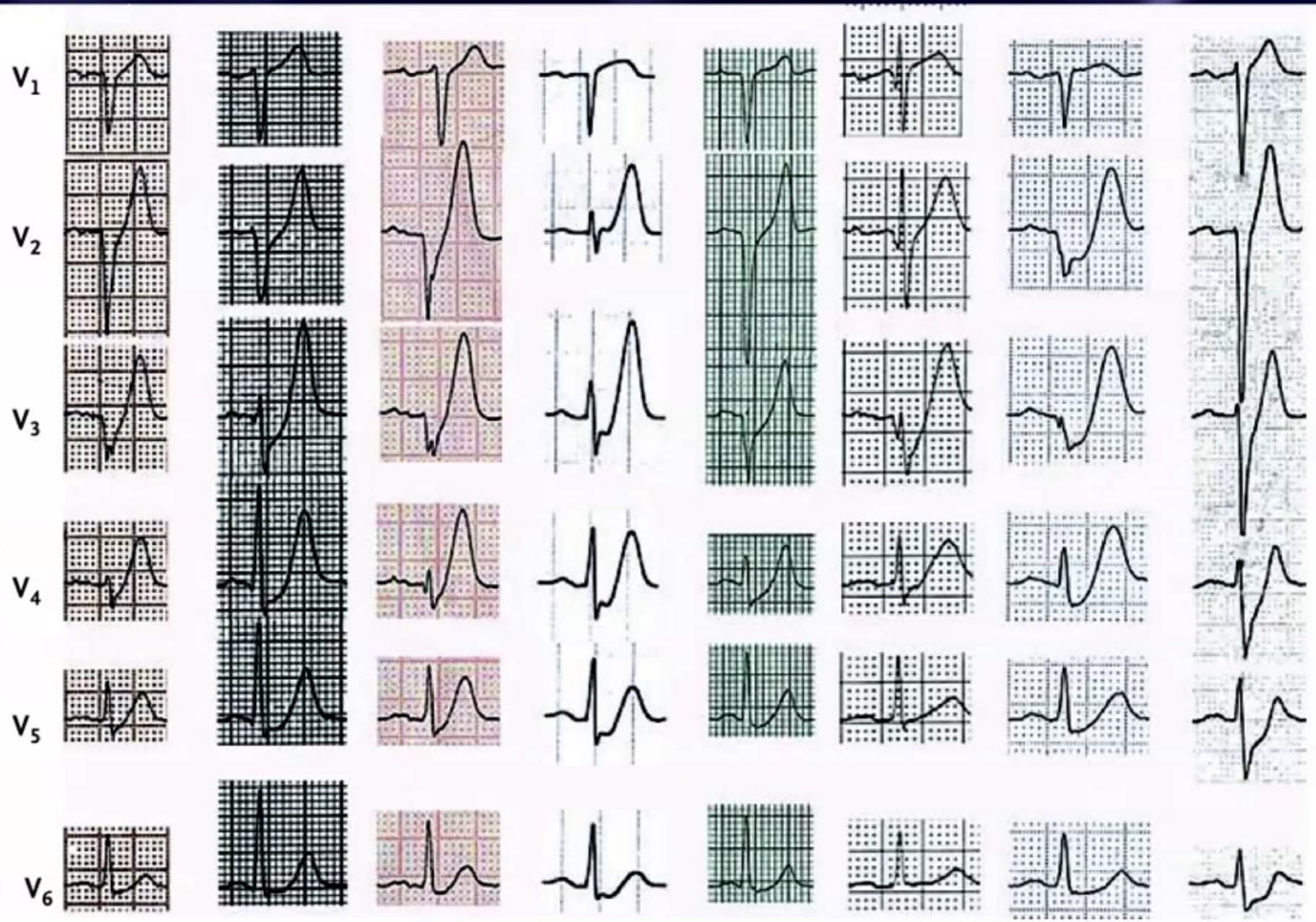
deWinters T waves

- Dressler 1948 rediscovered by DeWinter et al., 2008
- J point depressed 1 to 3 mm below the isoelectric line
- Up-sloping ST depression
- Merges into a tall, symmetric T wave
- Can evolve into ST elevation in the anterior leads
- Found to be due to acute proximal LAD occlusion

De-Winters T waves

- Rokos et al., 2010 proposed that deWinters T waves were a STEMI equivalent and should prompt activation of the cath lab

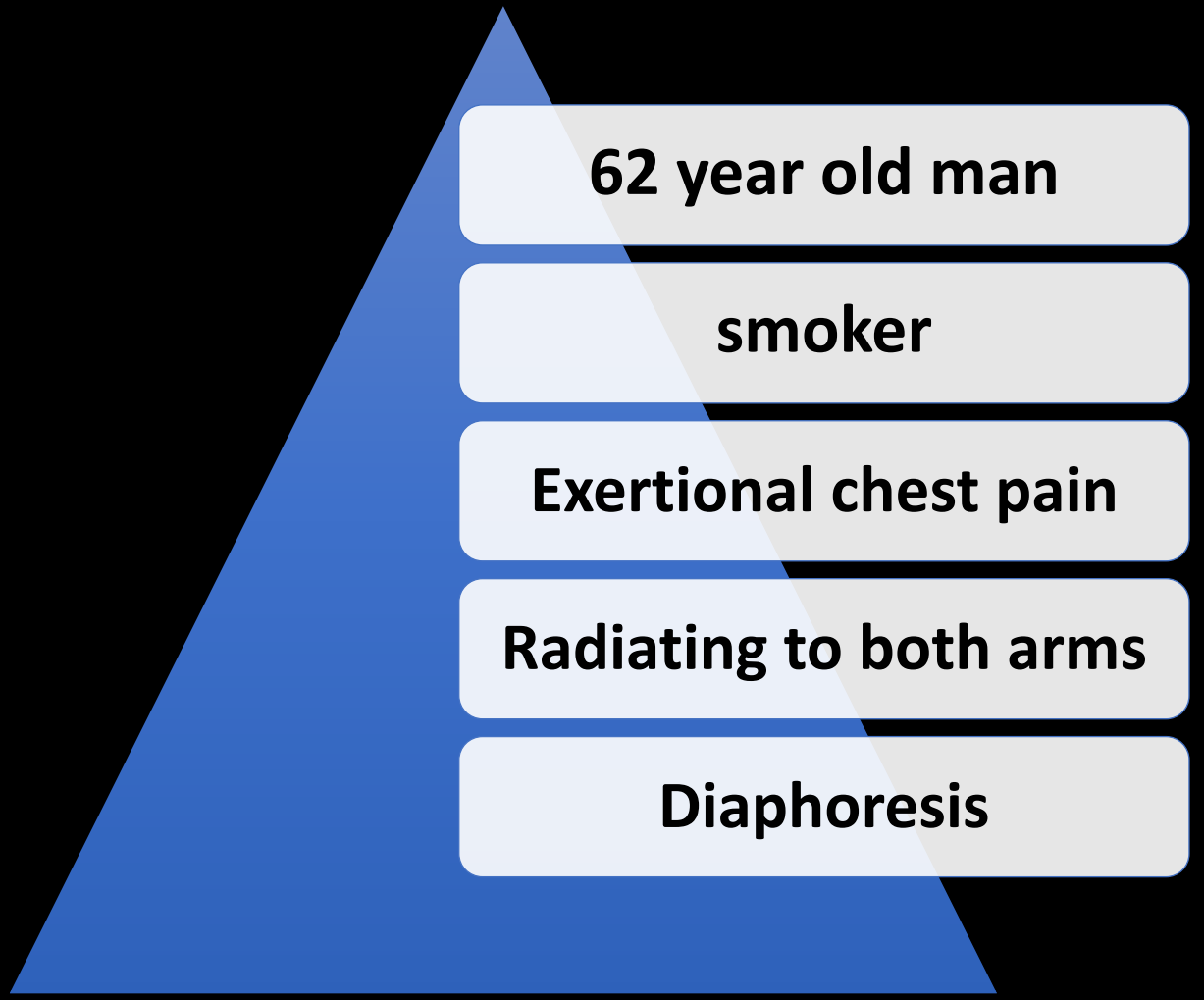
  - controversial
- Call cardiology and let them decide

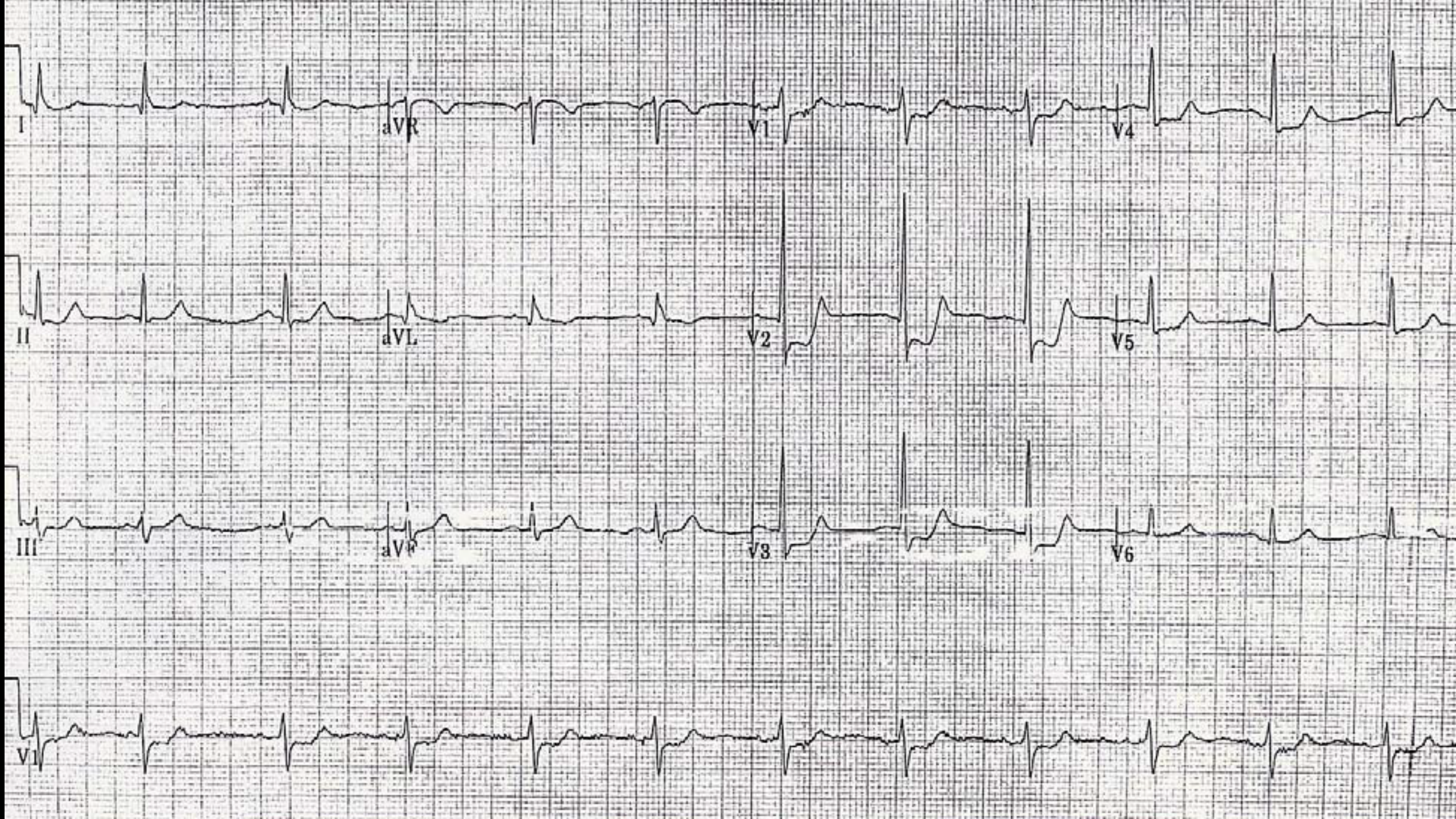

V1
V2
V3
V4
V5
V6

De-Winter’s T Waves: Summary

- Due to acute occlusion of the proximal LAD
- High risk of evolving into an anterior STEMI
- Not yet in the guidelines as a STEMI equivalent
- But they may be soon
- You should call cardiology

Case 3


62 year old man
smoker
Exertional chest pain
Radiating to both arms
Diaphoresis


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

ECG Interpretation

- SR at a rate of 72
- Normal axis
- No ST elevation
- “Early transition” / tall R waves in V2 to V4
- TWI aVL
- Horizontal ST depression in V1 to V5

ST Depression V1 – V4

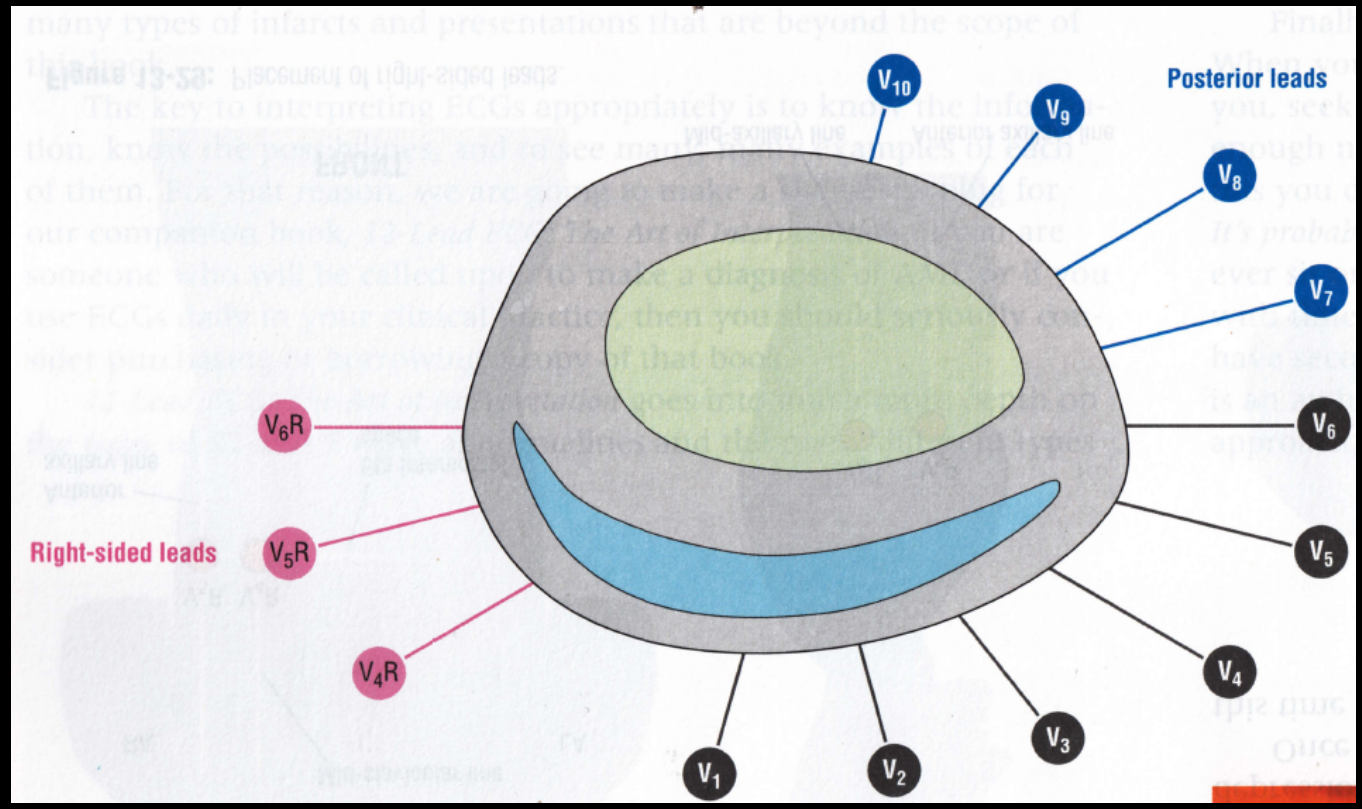
- Reciprocal change
- Ischaemia
- Infarction of the posterior all of the LV

Posterior AMI

- Usually associated with an inferior infarct
  - not change management
  - worse prognosis (it's a bigger area of infarction)
- But about 4 to 10% of AMI's are pure posterior AMI's
- They should got to the cath lab (STEMI-equivalent)
- But only 30% do
- Thus we need to be able to distinguish a posterior MI from anterior ischaemia

Posterior AMI

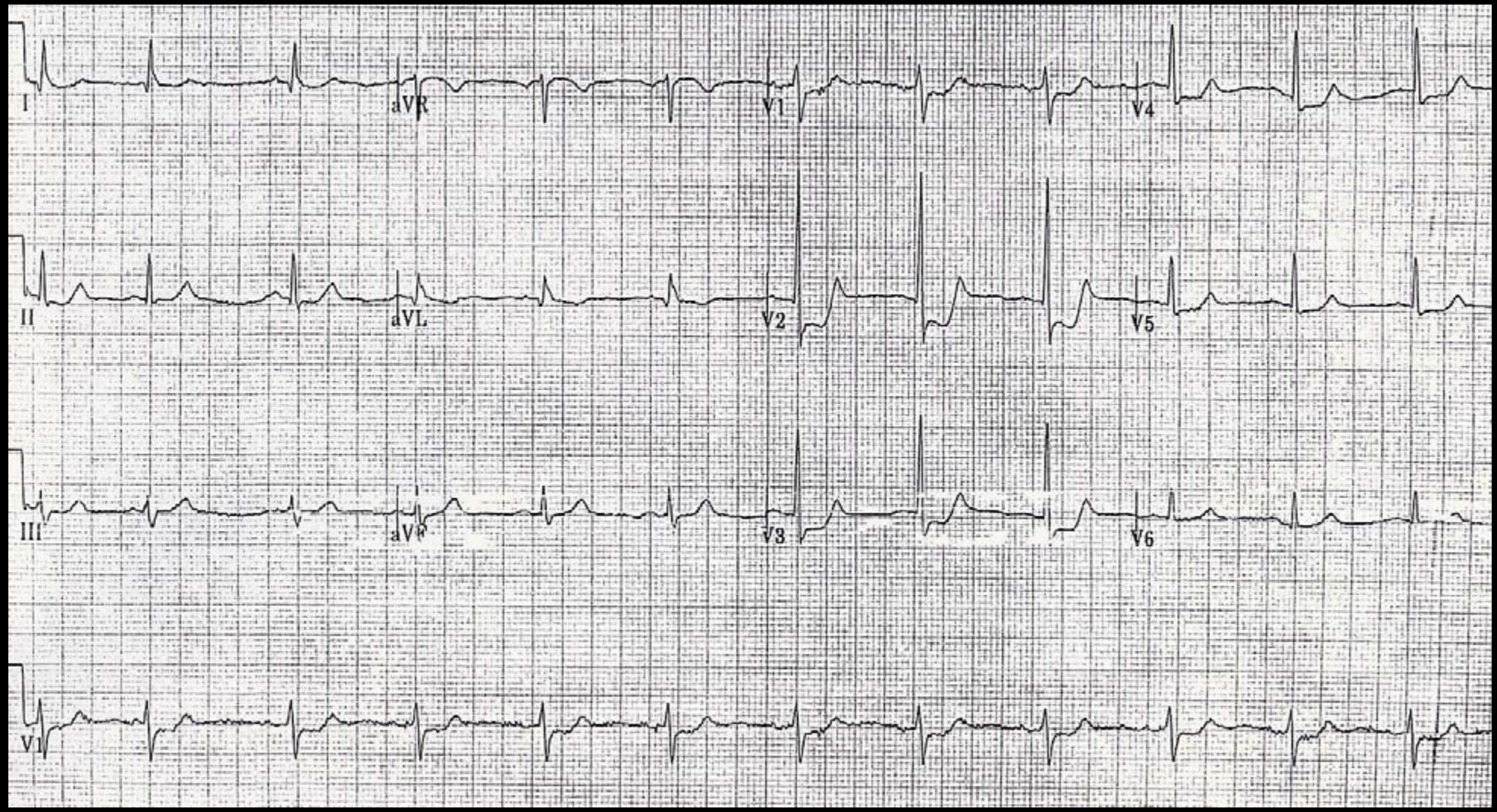
- In a 12 lead ECG, no leads "look at" the posterior wall of the LV
- However, leads V1 to V2 provide a mirror image of the posterior wall of the LV
- Thus a posterior STEMI is the mirror image of a septal (V1 and V2) STEMI



ECG Changes in V1 and V2

Septal MI	STE	Qs develop	Inverted Ts
Posterior MI	STD	Tall Rs develop	Upright Ts

Isolated Infarct of Posterior all of LV



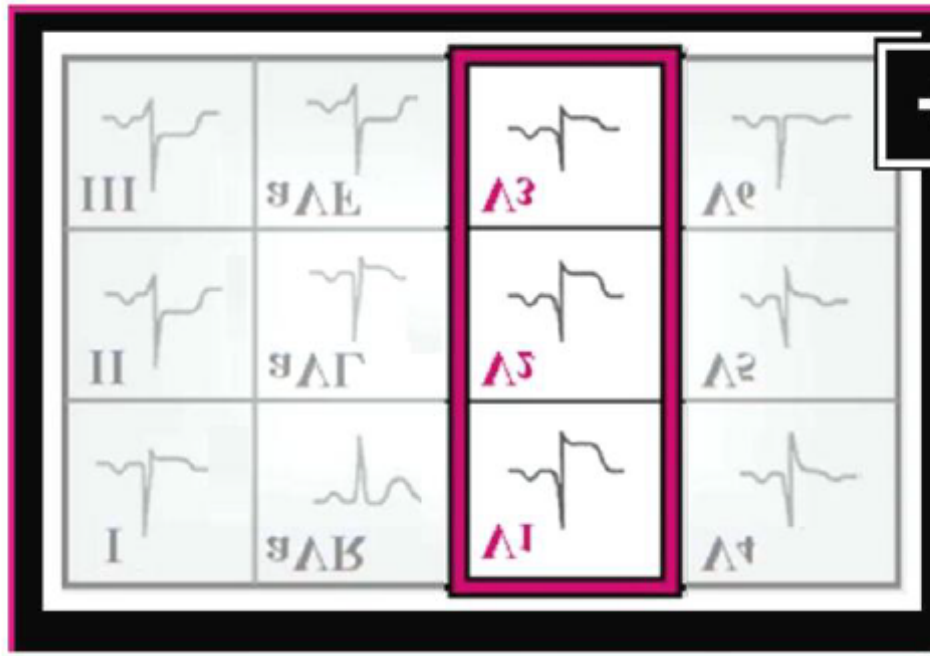
Tall R Wave

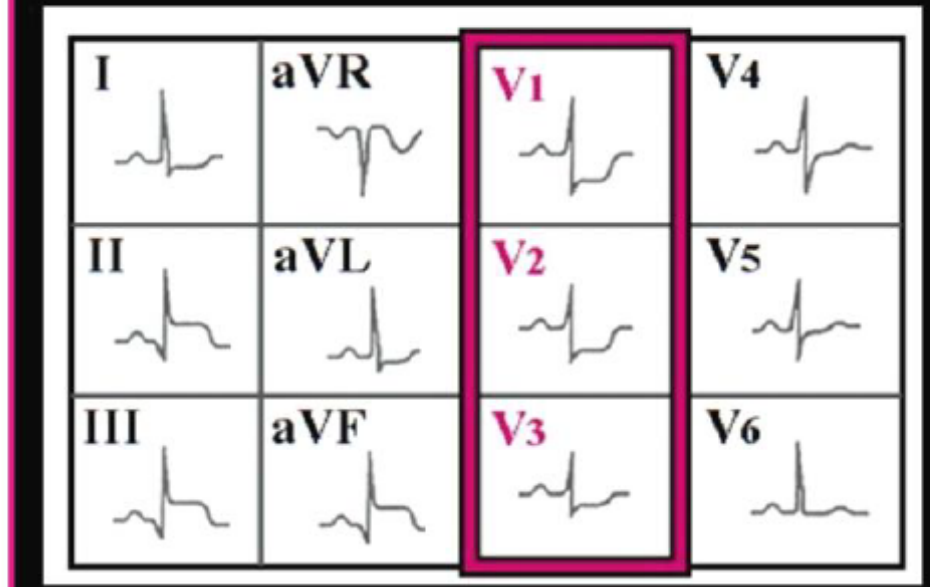
- It’s not the absolute size that is important
- Compare the R wave to the S wave
- If they are about the same size, it is considered a tall R wave

If you suspect a posterior AMI, get an ECG with posterior leads.

Whilst waiting, do the mirror test.

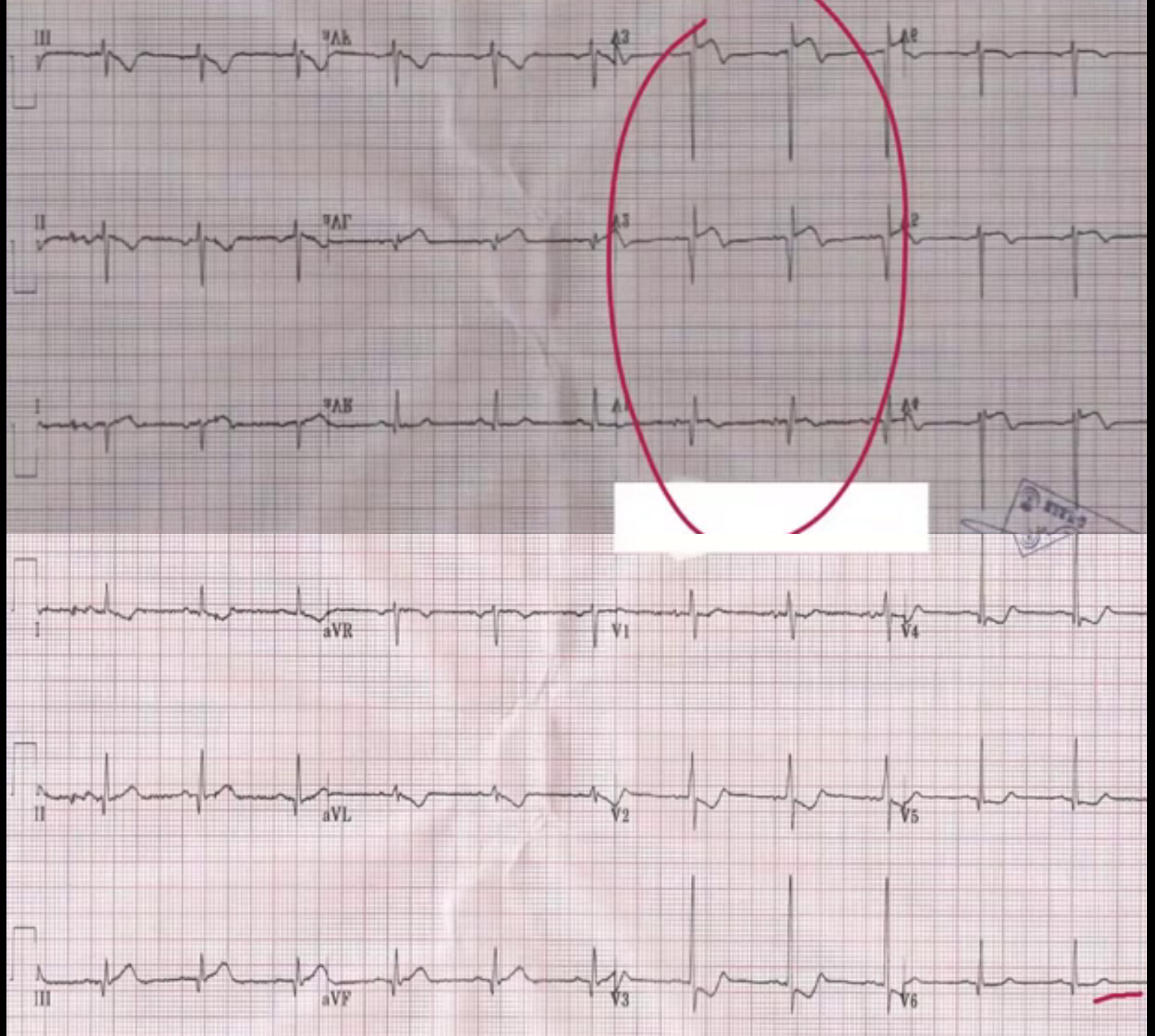
+ Mirror Test





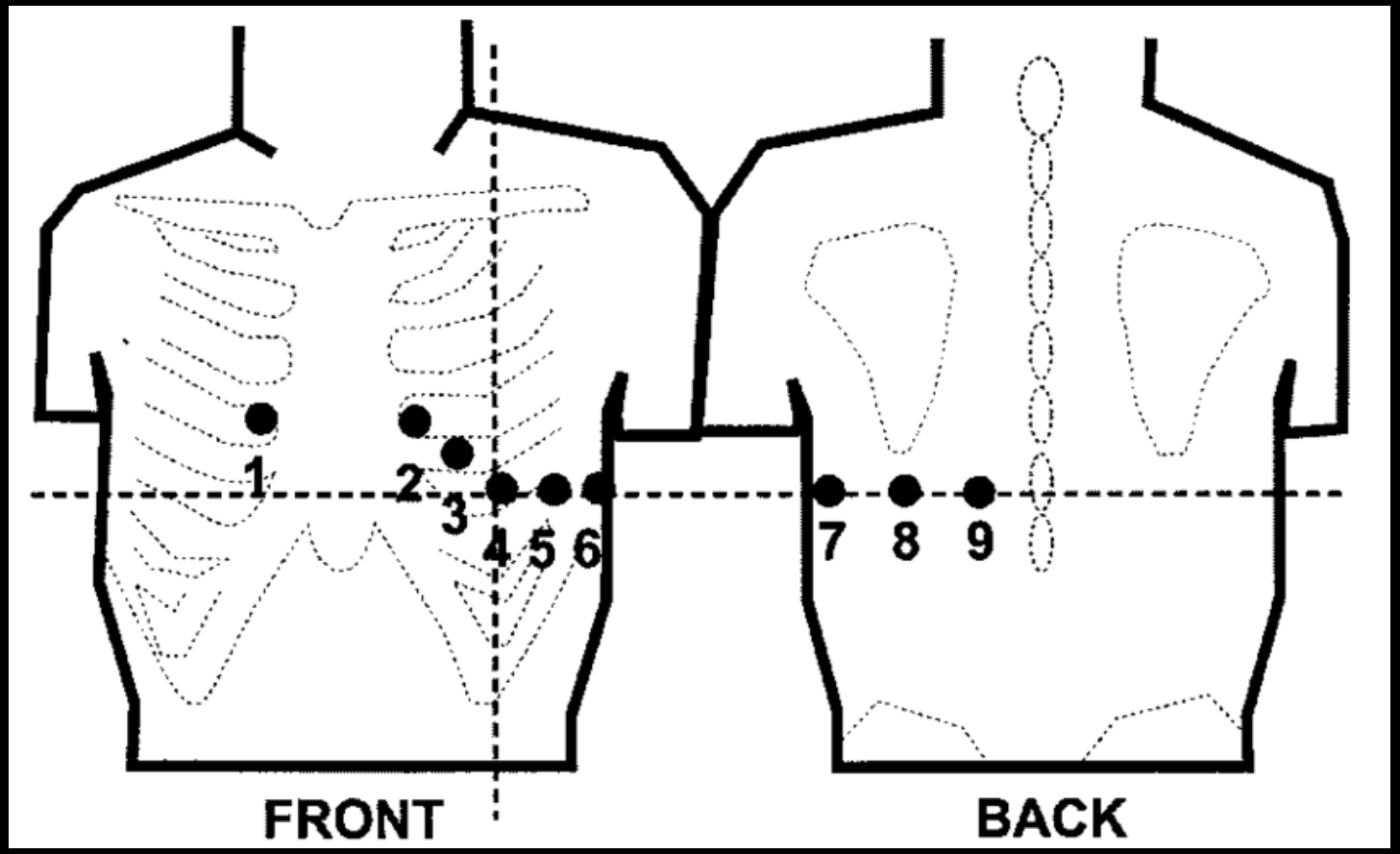
Holding the lower ECG up to the light (and looking through it) you should see a mirror image picture looking like that shown to the left in leads V1, V2, V3.

This defines a "Positive Mirror Test".


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

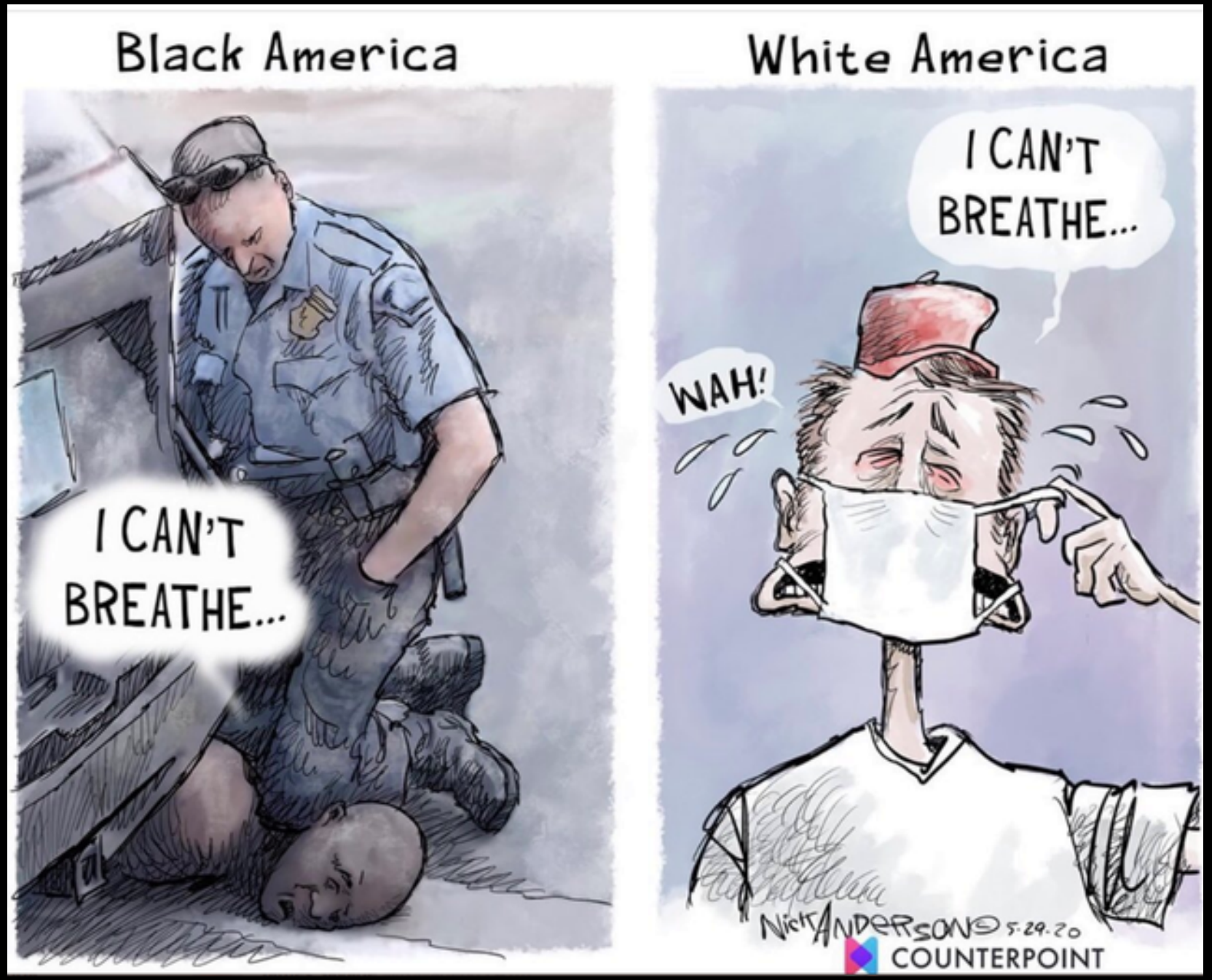
Posterior Leads

- The mirror test is quick and easy
- Right-sided leads are more sensitive
- If you suspect a posterior wall AMI, get an ECG with posterior leads



Posterior Infarct: Summary

- Early on, tall R-waves may not be present
  - may take hours to appear
  - analogous to Q-waves
- Thus whenever isolated ST-segment depression in leads V1-V3 is found, get an ECG using posterior leads
- If these leads show ST elevation, the diagnosis is posterior STEMI.
- If these leads do not show ST elevation, the diagnosis is anterior ischemia


Black America
I CAN'T BREATHE...
White America
I CAN'T BREATHE...
WAH!
NickANDERSON 5.29.20
COUNTERPOINT

Summary

- If, based on the patients history, you suspect an AMI get an ECG and look for ST elevation and then for a STEMI-equivalent
- It is OK to not be sure if the patient needs re-perfusion
- Remember: serial ECG's

  get old ECG's

  additional leads in selected cases

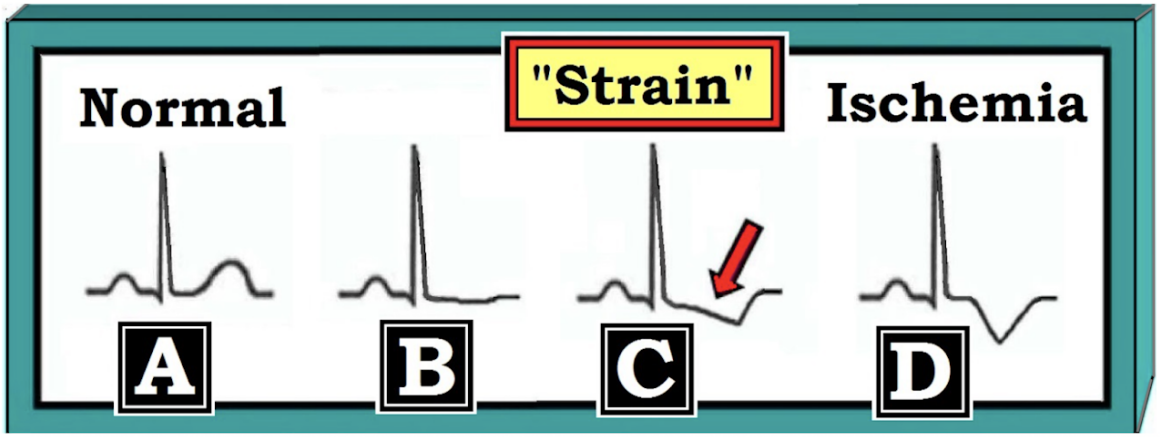
***Simplified* Criteria** for Diagnosing **LVH**

1. Deepest S wave in lead V_1 *or* V_2,
   plus tallest R wave in lead V_5 *or* V_6 ≥ **35**.
   — *and/or* — R in lead aVL ≥**12**.
2. Patient ≥ **35** years old.
3. Left ventricular (LV) ***"strain"***.

***Additional* Voltage Criteria**

No single voltage criterion will identify all patients with LVH. As a result — we occasionally turn to ***additional* voltage criteria**. We favor *any* of the following:

- A ***deep* S wave** (*≥20-25 mm*) in lead **V1** or **V2.**
- A ***tall* R wave** (*≥25 mm in* ***V5*** — or *≥18-20 mm in* ***V6***).
- An **R wave ≥20** in ***any* inferior lead** (*II, III, or aVF*).
- ***Cornell* Criteria** — LVH is present IF sum of **R** wave in lead **aVL + S** in **V3** is **≥20**mm (*female*) or **≥28**mm (*male*).
- ***Peguero* Criteria** — LVH is present IF sum of ***deepest* S** in *any* chest lead + **S** in **V4 ≥ 23**mm (*female*) or **≥28**mm (*male*). NOTE: If the deepest S is in V4, then *double* this value.
- ***Bottom* Line:** In our experience, IF all you remember are the numbers '**35**' and '**12**' for **LVH *voltage* criteria** — then ***~90% of the time*** when it is possible to diagnose LVH on the ECG of an ***adult***, you will be able to do so!
- Satisfying *any* of the above ***additional* criteria** should help to pick up most of the remaining ~10%.
- Incorporating ***Clinical* History** *and* looking for ***"strain"*** or a strain "equivalent" — will further refine and increase accuracy (*specificity*) of your diagnosis.
- Keep in mind that ***competing* conditions** (ie, *hyperkalemia, acute infarction, conduction defects, pulmonary disease*) — may *mask* ECG diagnosis of LVH.
- IF you *really* need to know about chamber size — *Get an* **Echo**. The ECG is simply *not* optimally accurate.



LEGEND: ST-T wave appearance of a *normal* ST segment (**Panel A**) — compared to the ST-T wave changes of **LV "strain"** (*red arrow in* **Panel C**). Typically — LV "strain" is seen in *one or more* of the **lateral leads** (ie, *leads I, aVL; and/or V4,V5,V6*).

- **NOTE:** The presence or absence of LV "strain" assists greatly in determining the likelihood of *true* LV chamber enlargement. Whereas the *specificity* of the ECG for determining LVH is well *under* 50% when voltage criteria alone are met — specificity of the ECG for LVH may increase to ***over* 90%** when ***voltage* criteria** are met in a *middle-aged to older* adult *with* underlying ***heart* disease** who *also* manifests ST-T repolarization changes of **LV "strain"** in one or more leads.
- As one traces the path from a *normal* ST-T wave (Panel A) — to the ***asymmetric*** (ie, *slow downslope; more rapid upslope*) **ST-T wave shape** that is characteristic of **LV "strain"** (*seen in* Panel C) — one passes through an ***"intermediate"*** stage (**Panel B**).
- **Panel B —** In this *intermediate* stage (*which I call a* ***"strain equivalent"*** **pattern**) — the ST segment flattens, and there may be slight ST depression — but the frank *asymmetric* ST depression shape of Panel C is *not* yet seen. While the correlation is not perfect — patients with this "strain equivalent" pattern of Panel B are more likely to have true LV chamber enlargement than patients whose ST-T waves are normal (*corresponding to* Panel A) in lateral leads.
- In contrast to the ***asymmetric* ST-T wave depression** of ***"strain"*** seen in **Panel C** — the ***symmetric* T wave inversion** in **Panel D** is more suggestive of **ischemia.**
- **CAVEAT:** There may be *overlap* between Panels C and D. As a result — patients with *both* true LVH and cardiac ischemia may manifest an ST-T wave that looks like either Panel C or Panel D.

Causes of ST Segment Elevation

- Acute Pericarditis
- Benign Early Repolarization
- Left Bundle Branch Block with AMI (Sgarbossa et al's criteria)
- Left Ventricular Hypertrophy
- Left Ventricular Aneurysm
- Brugada Syndrome
- Hyperkalemia
- Hypothermia
- CNS pathologies
- Prinzmetal Angina
- Post electrical cardioversion