

Ignore the voice in the head?

KV

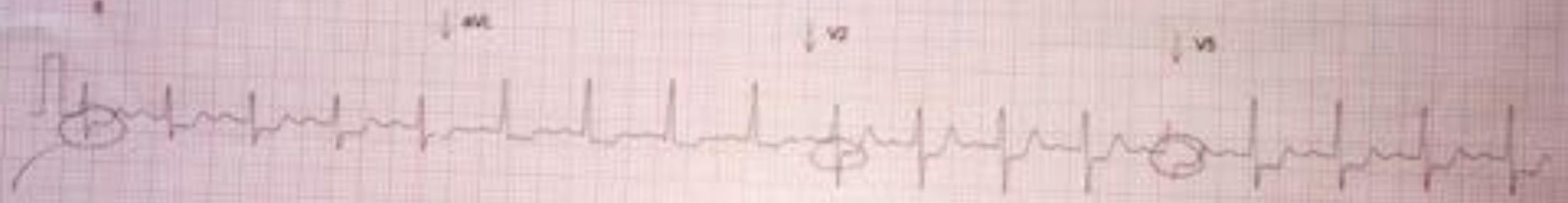
Referred by:

Confirmed By:



9/10 chest tightness

Unconfirmed Report



# LMCA Obstruction

- Widespread horizontal ST depression
- ST elevation in aVR  $\geq 1\text{mm}$
- ST elevation in aVR  $\geq V_1$



Where else?

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

Name: , ID: 999999999 18-Aug-2015 01:16

55 years

Rest. rate

90 BPM

\*\*\* AGE AND GENDER SPECIFIC ECG ANALYSIS \*\*\*

Loc: 1

Room: BED5

KANGAROO FLAT 3555 W. COU  
15/08/15 James, Mr Dugal  
MBP MEM:21191323B CL:All Privat  
CON:SN501553940 M/C:3161532417  
ADM: 891131 / Ref:1 11/2019

BRUS RHYTHM

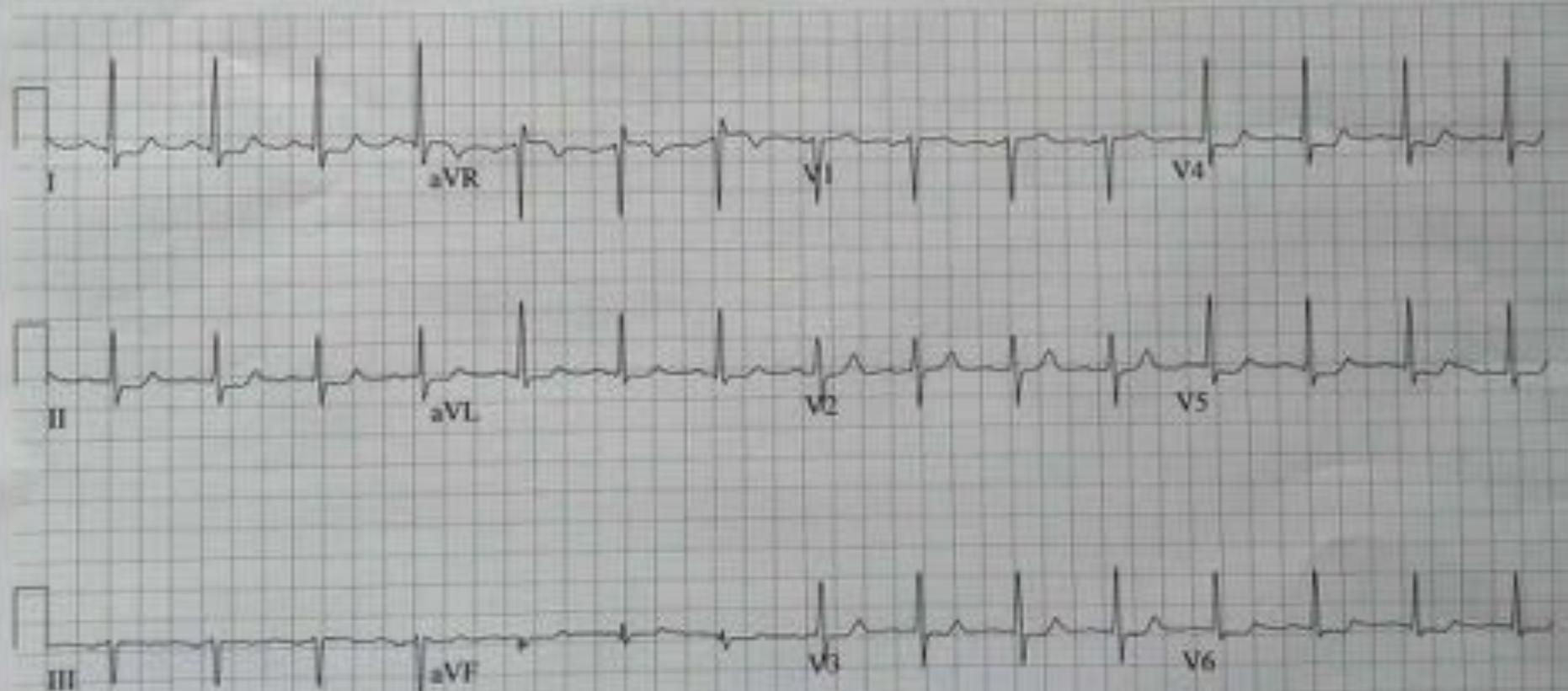
TRICULAR HYPERTROPHY WITH REPOLARIZATION ABNORMALITY

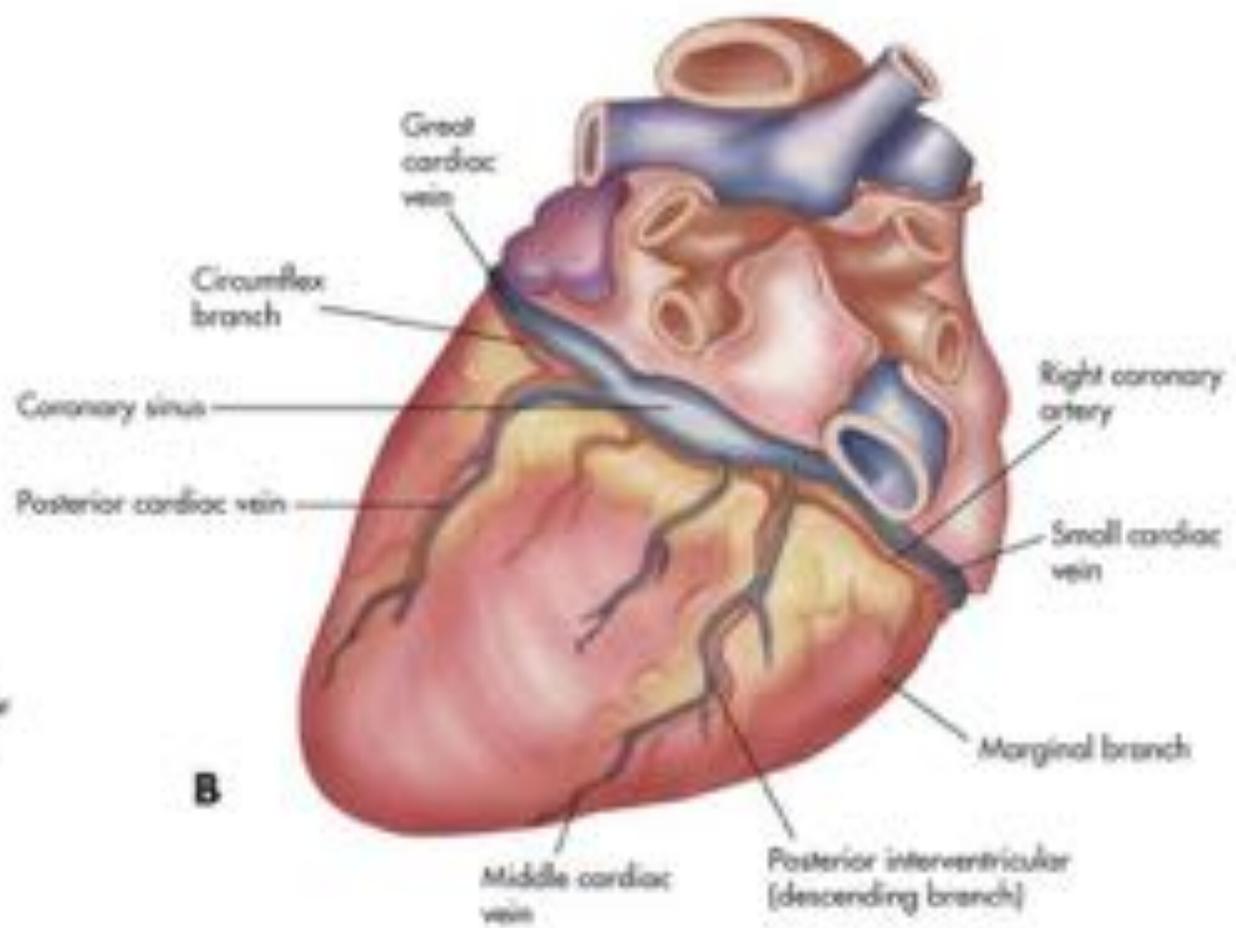
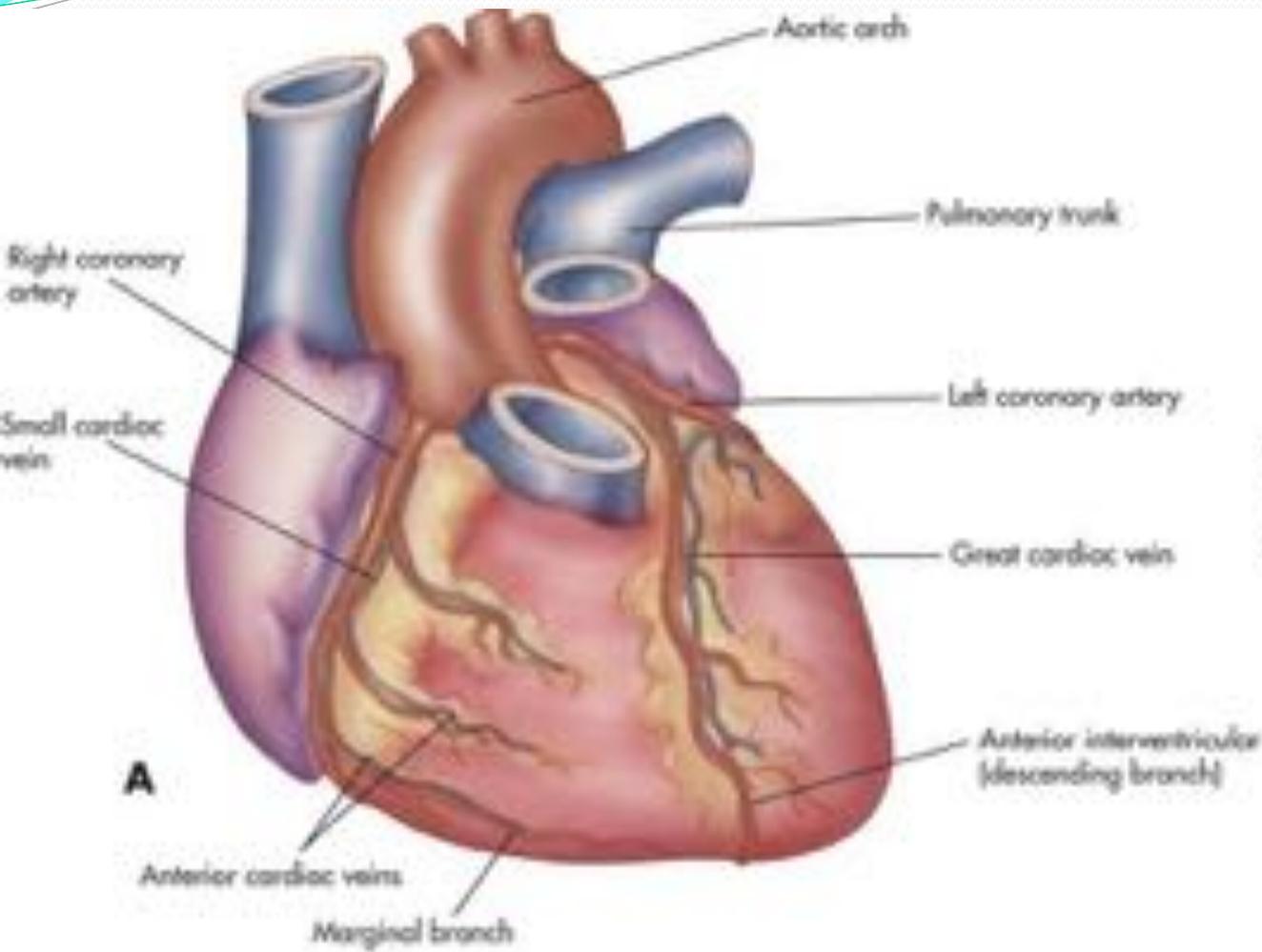
L ECG

O/A to lld

Ni 40 pair

Unconfirmed





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## An Early and Simple Predictor of Severe Left Main and/or Three-Vessel Disease in Patients With Non-ST-Segment Elevation Acute Coronary Syndrome

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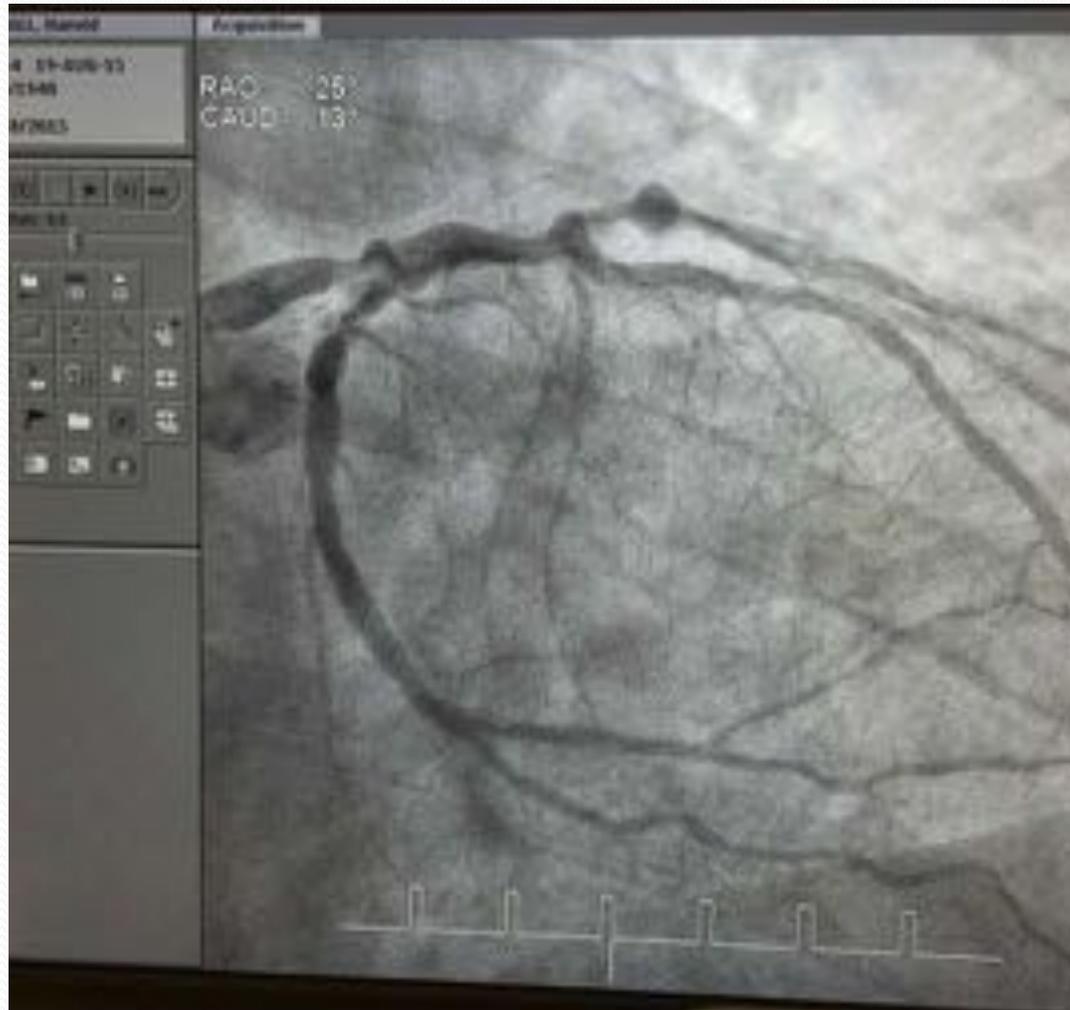
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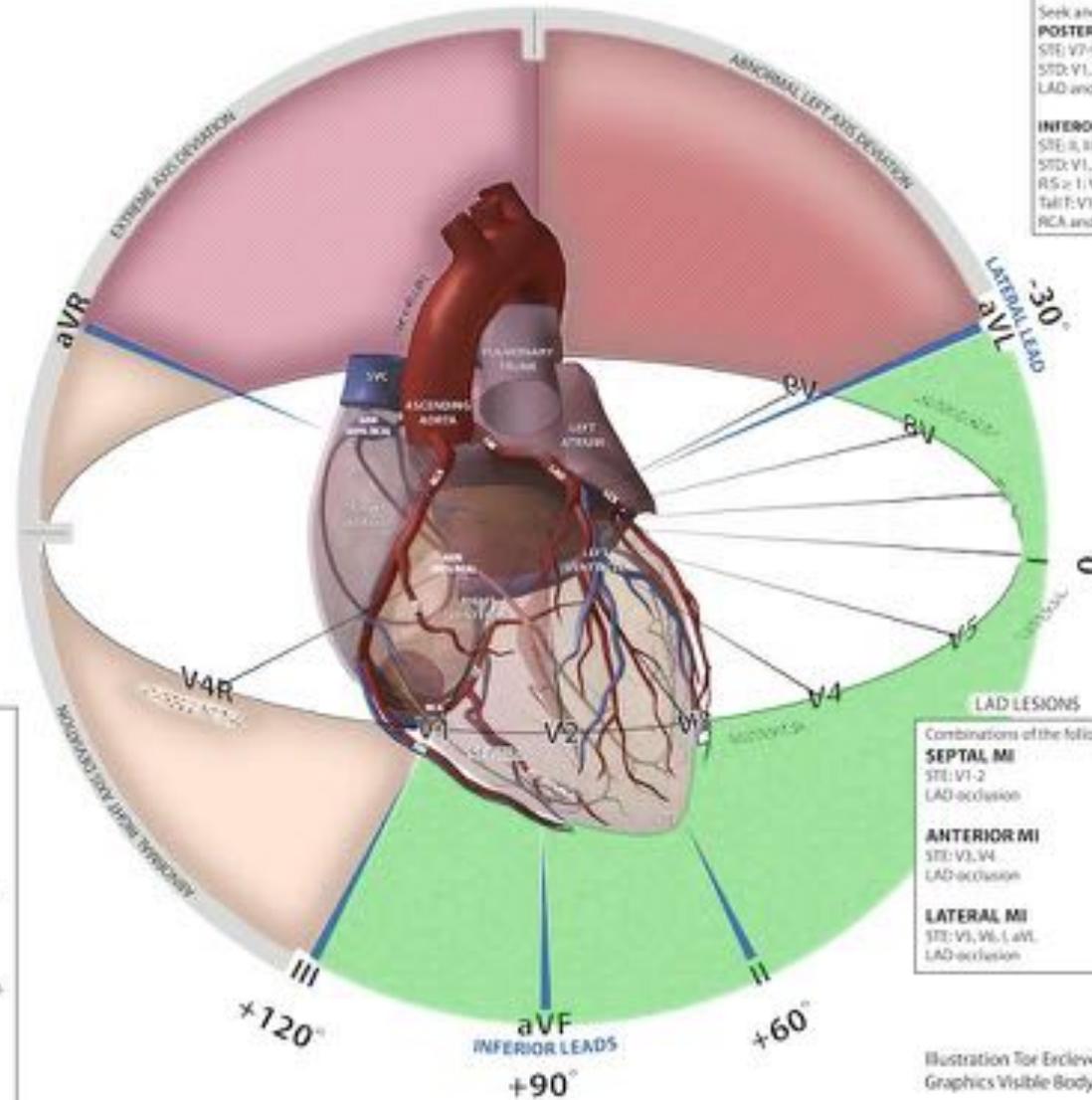
- STE in aVR  $\geq 1$  mm was a strong predictor of severe LMCA / 3VD requiring CABG (Sens 80%, Specf 93%)
- The negative predictive value of STE  $> 1$ mm in aVR was 98% !!
- Conversely, patients with  $< 1$ mm ST elevation in aVR had a negligible risk of severe LMCA / 3VD requiring CABG
- Authors suggest that with the subsequent increased need for CABG, these patients would benefit from withholding clopidogrel

- Angiogram of Left Main ~ 80-90% occluded!



# Mechanism

# AMI ECG, ANATOMY AND PATHOLOGY



## LAD LESIONS ±

- POSTERIOR MI**  
 STE: V7-9  
 STD: V1-2 (reciprocal STE)  
 RS ≥ 1: V1-2  
 Tall T: V1-2  
 RCA and LAD occlusion
- Seek and exclude  
**POSTEROLATERAL MI**  
 STE: V7-9 and I, aVL, V5-6  
 STD: V1, V2  
 LAD and LAD occlusion
- INFEROPOSTERIOR MI**  
 STE: II, III, aVF and V7-9  
 STD: V1, V2 (reciprocal STE)  
 RS ≥ 1: V1-2  
 Tall T: V1-2  
 RCA and LAD occlusion

## RCA 'TYPE' LESIONS ±

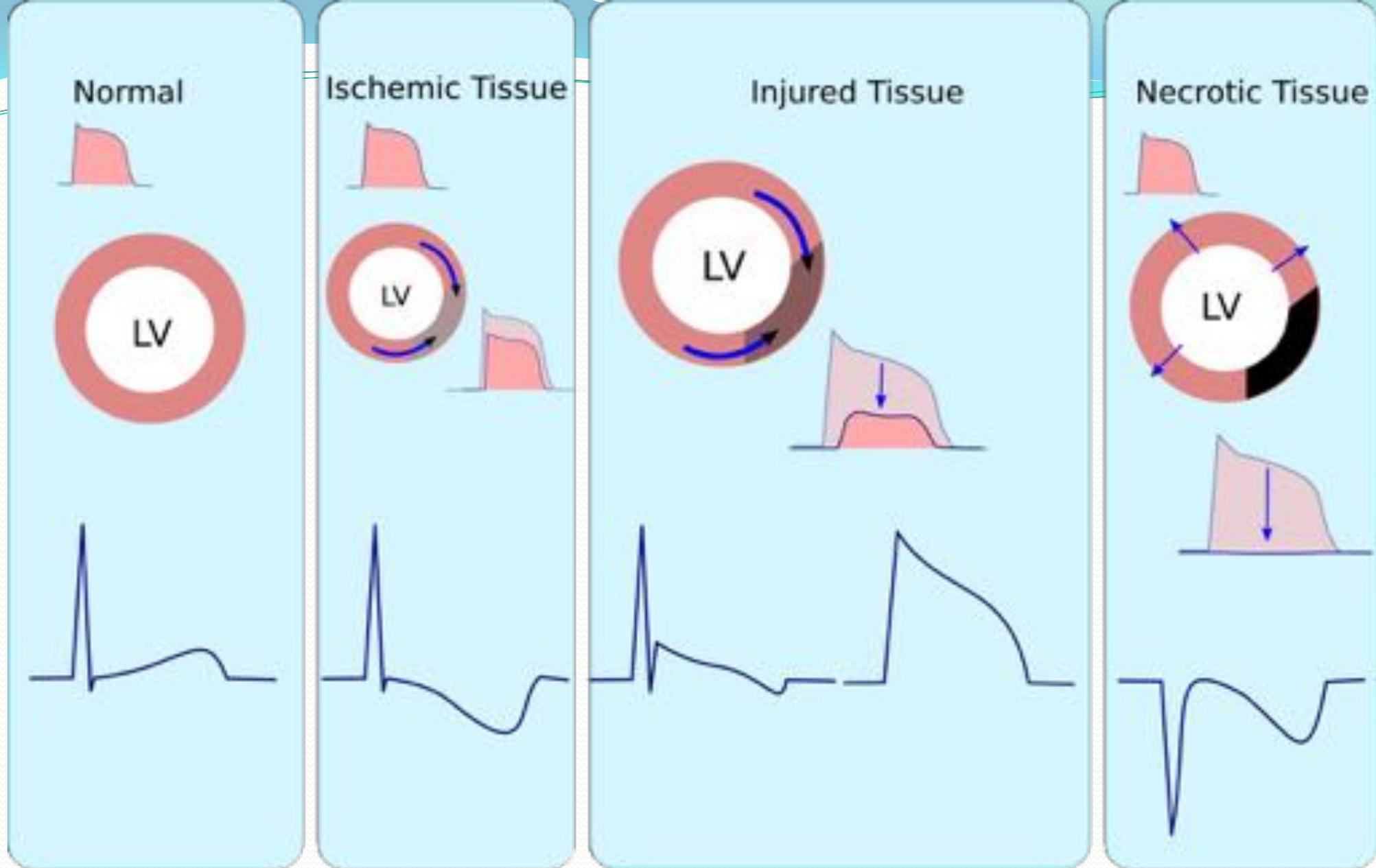
- INFERIOR MI**  
 STE: II, III, aVF  
 STD: aVL (reciprocal STE)  
 RCA occlusion distal to RV  
 50% of MI
- Seek and exclude  
**INFERIOR AND RV MI**  
 STE: II, III, aVF and V1, V4R  
 RCA occlusion proximal to RV  
 40% of inferior MI  
 Increased mortality risk
- INFEROLATERAL MI**  
 STE: I, II, aVL and I, aVL, V5, V6 & V4R  
 LAD and LAD occlusion  
 in a L dominant system
- INFEROPOSTERIOR MI**  
 STE: II, III, aVF and V7-9  
 STD: V1, V2 (reciprocal STE)  
 RS ≥ 1: V1-2  
 Tall T: V1-2  
 RCA and LAD 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

Illustration: Tor Erclevo  
 Graphics: Visible Body 3D

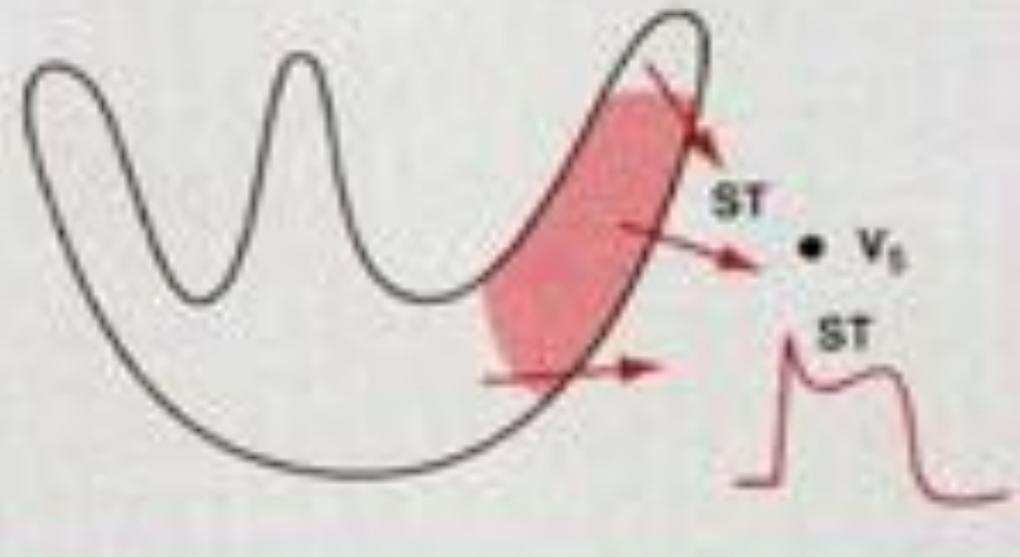
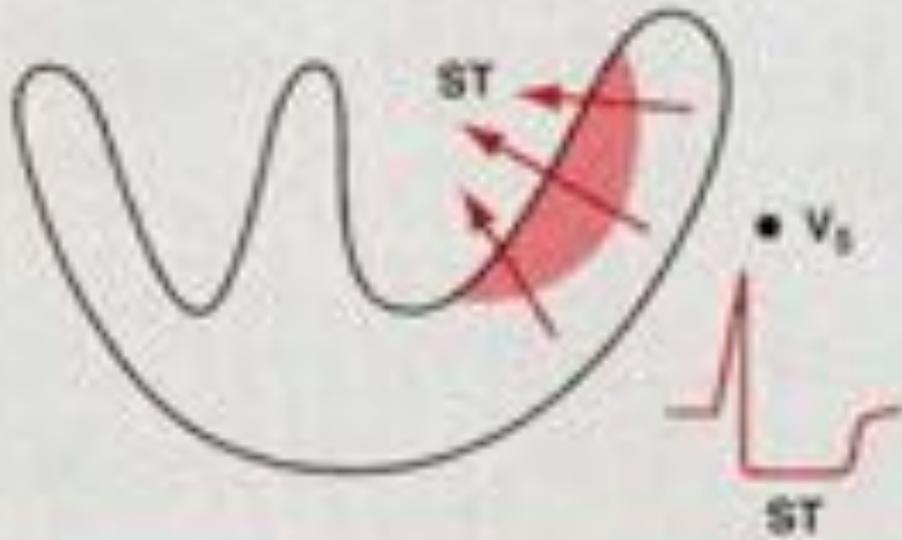
Emergency Department SCGH  
 University of Western Australia  
 UWA GP ABC Course  
 Life in the Fast Lane



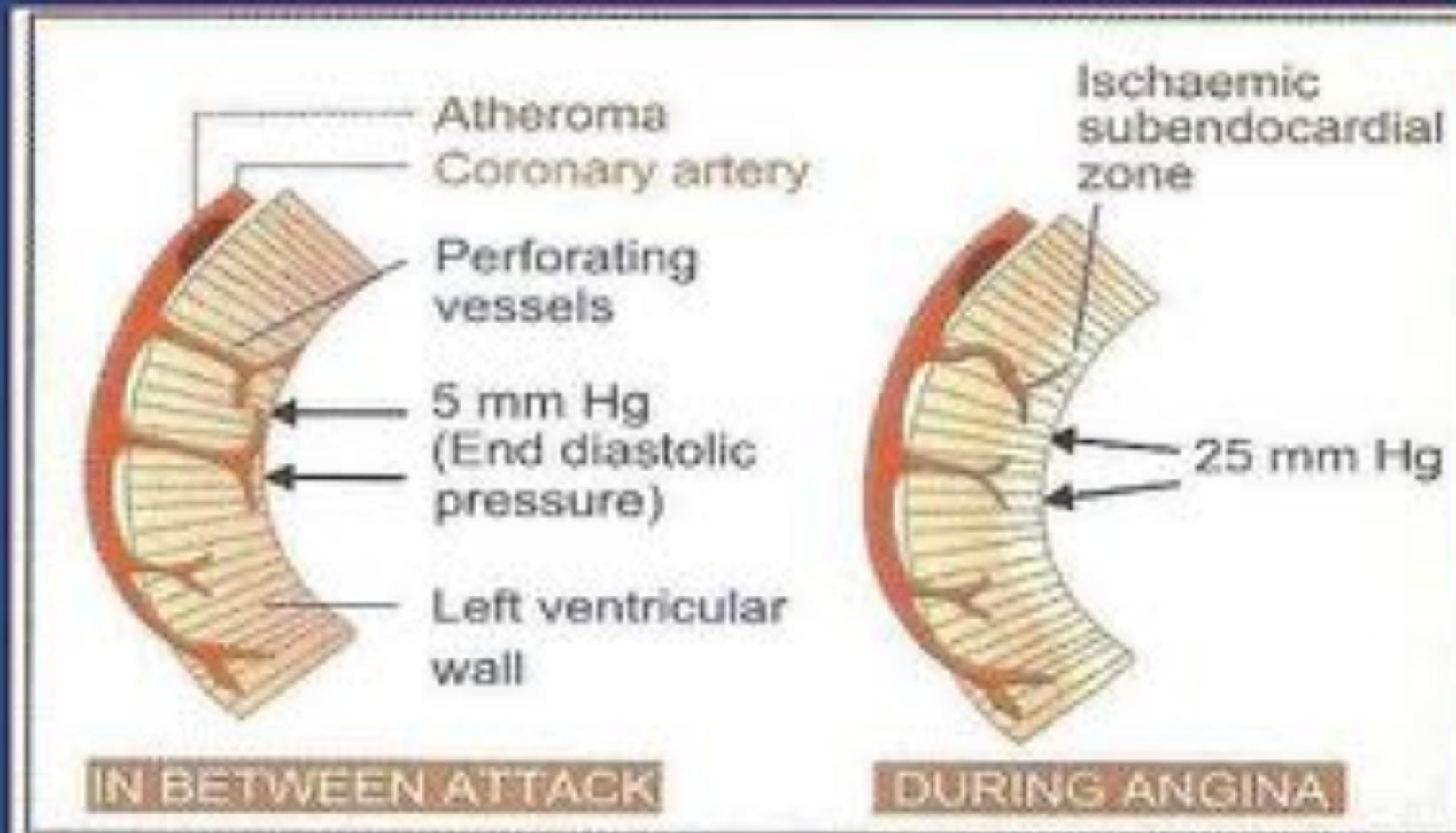
Injury current flows from healthy cardiomyocytes to ischemic cardiomyocytes resulting in ST shift. Necrotic tissue has no electrical activity

**SUBENDOCARDIAL INJURY:  
ST Depression**

**TRANSMURAL (EPICARDIAL) INJURY:  
ST Elevation**



# Subendocardial Crunch - Image



**Fig. 39.1:** Diagrammatic representation of subendocardial 'crunch' during an attack of angina

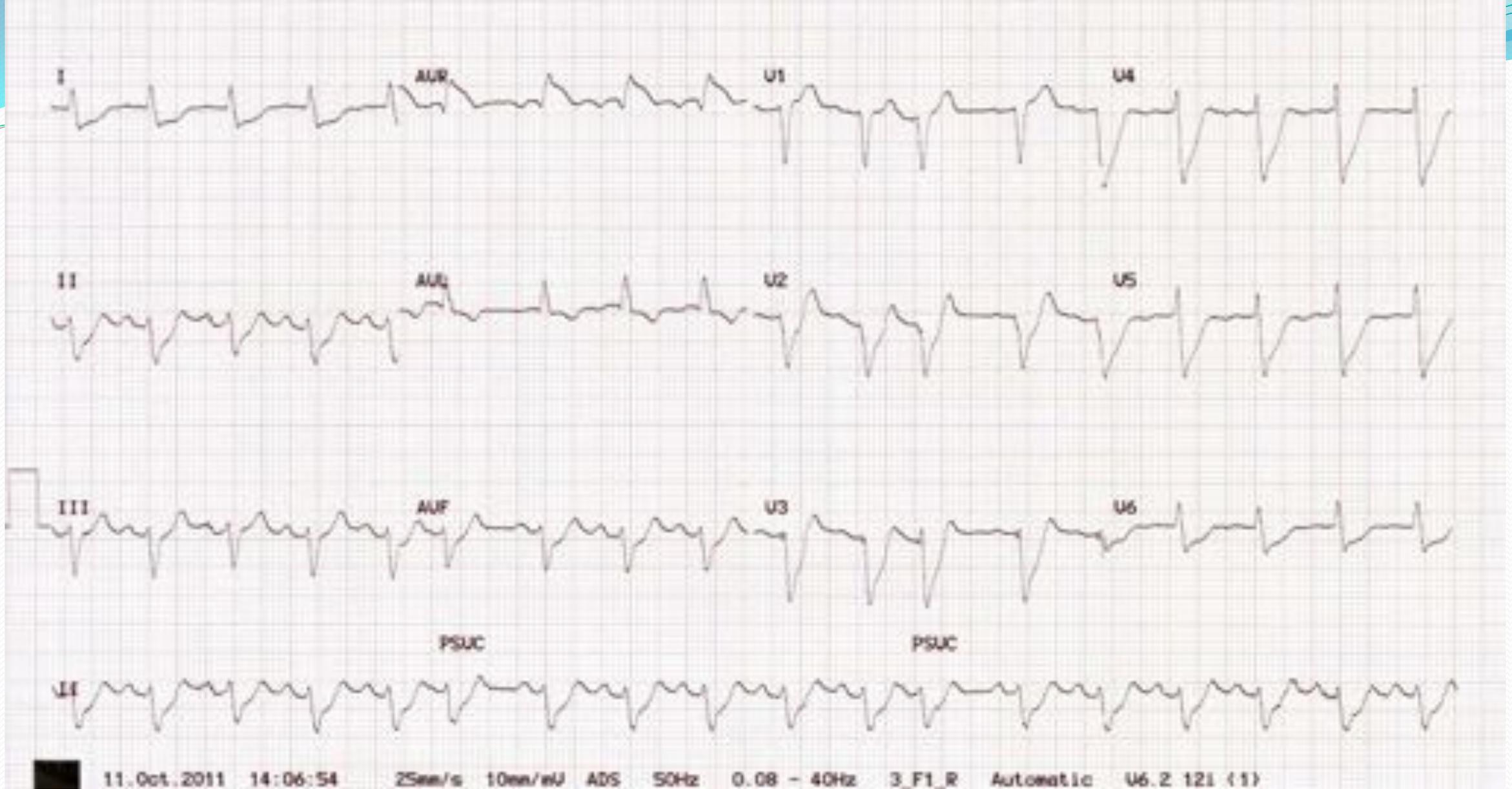
- Diffuse subendocardial ischaemia, with ST depression in the lateral leads producing reciprocal change in aVR

*Opposite (reciprocal) to a negative ST vector towards I, II, and V5*

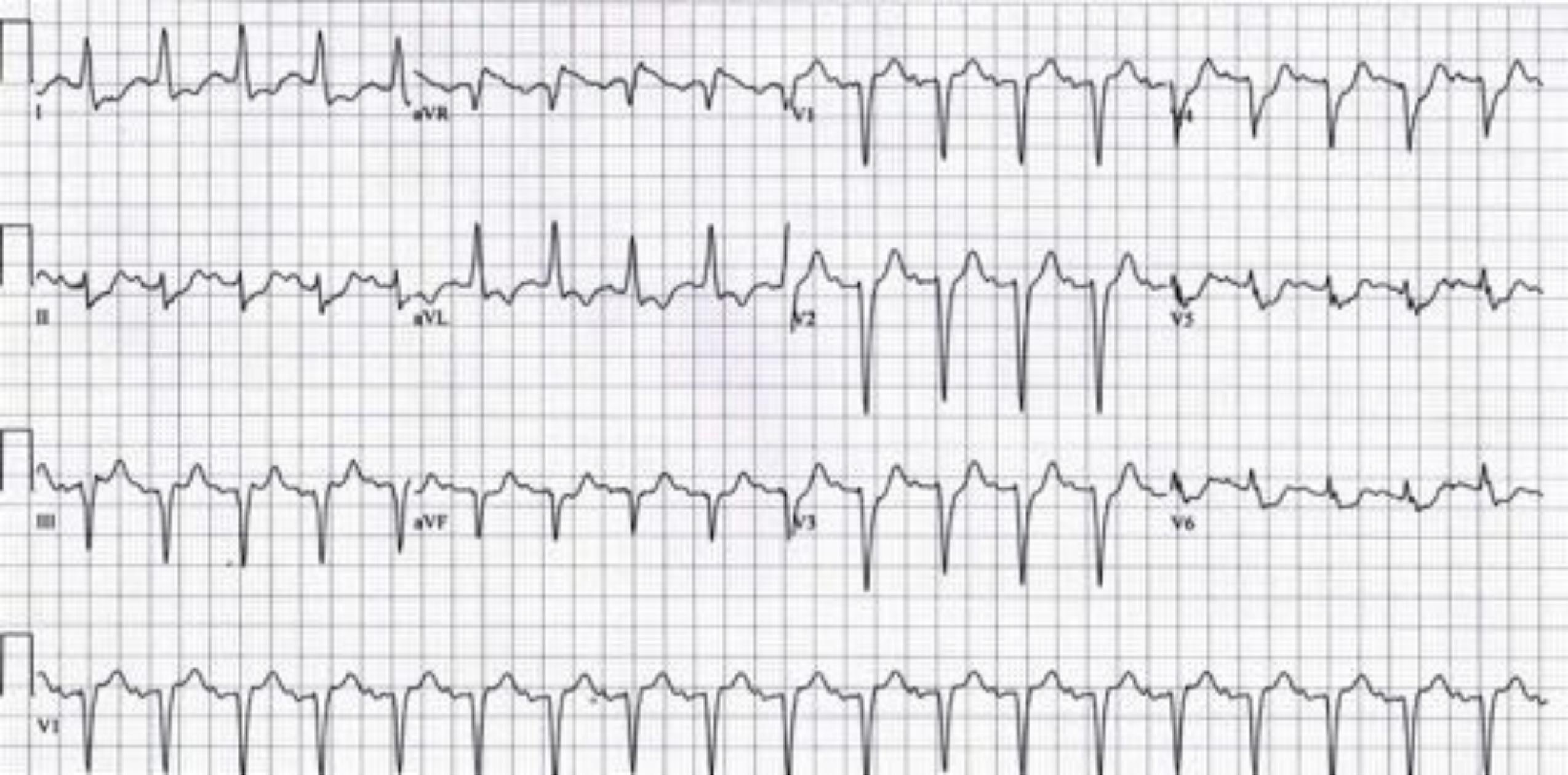
- Infarction of the basal septum, i.e. a STEMI involving aVR.

*The basal septum is supplied by the first septal perforator artery (a very proximal branch of the LAD), so ischaemia / infarction of the basal septum would imply involvement of the proximal LAD or LMCA*

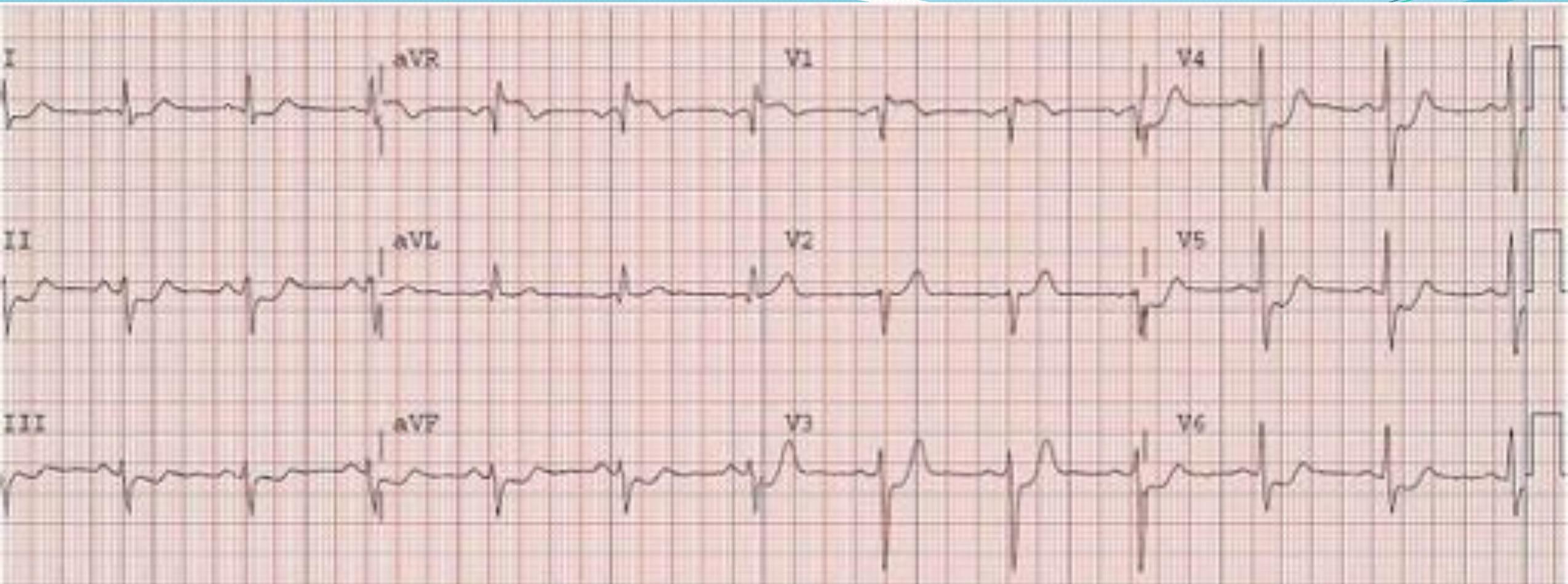
- *In the presence of anginal symptoms,*
- STE in aVR + STE in V<sub>1</sub>
  - - Highly predictive of LMCA or Prox LAD obstruction
- STE in aVR > STE in V<sub>1</sub>
  - - almost always indicates a LMCA obstruction (81% sensitive and 80% specific)



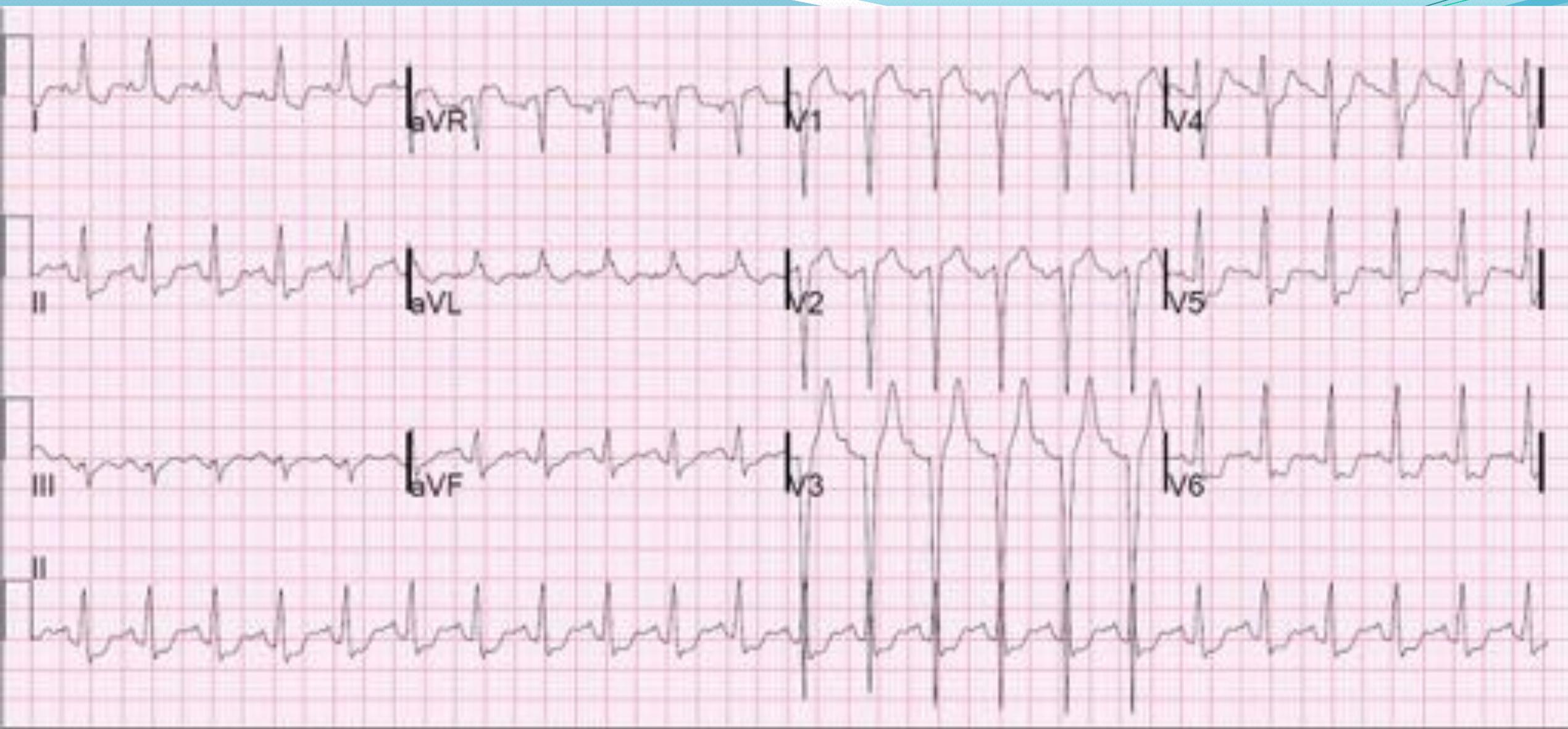
*Severe ischaemic chest pain, vomiting, syncope (due to runs of VT) and cardiogenic shock. Emergent angiography - complete ostial occlusion of LMCA*



*Elderly patient with chest pain and cardiogenic shock (hence the tachycardia). Brief episode of VF whilst being transferred onto the cath lab table. Angiography revealed a LMCA occlusion*



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



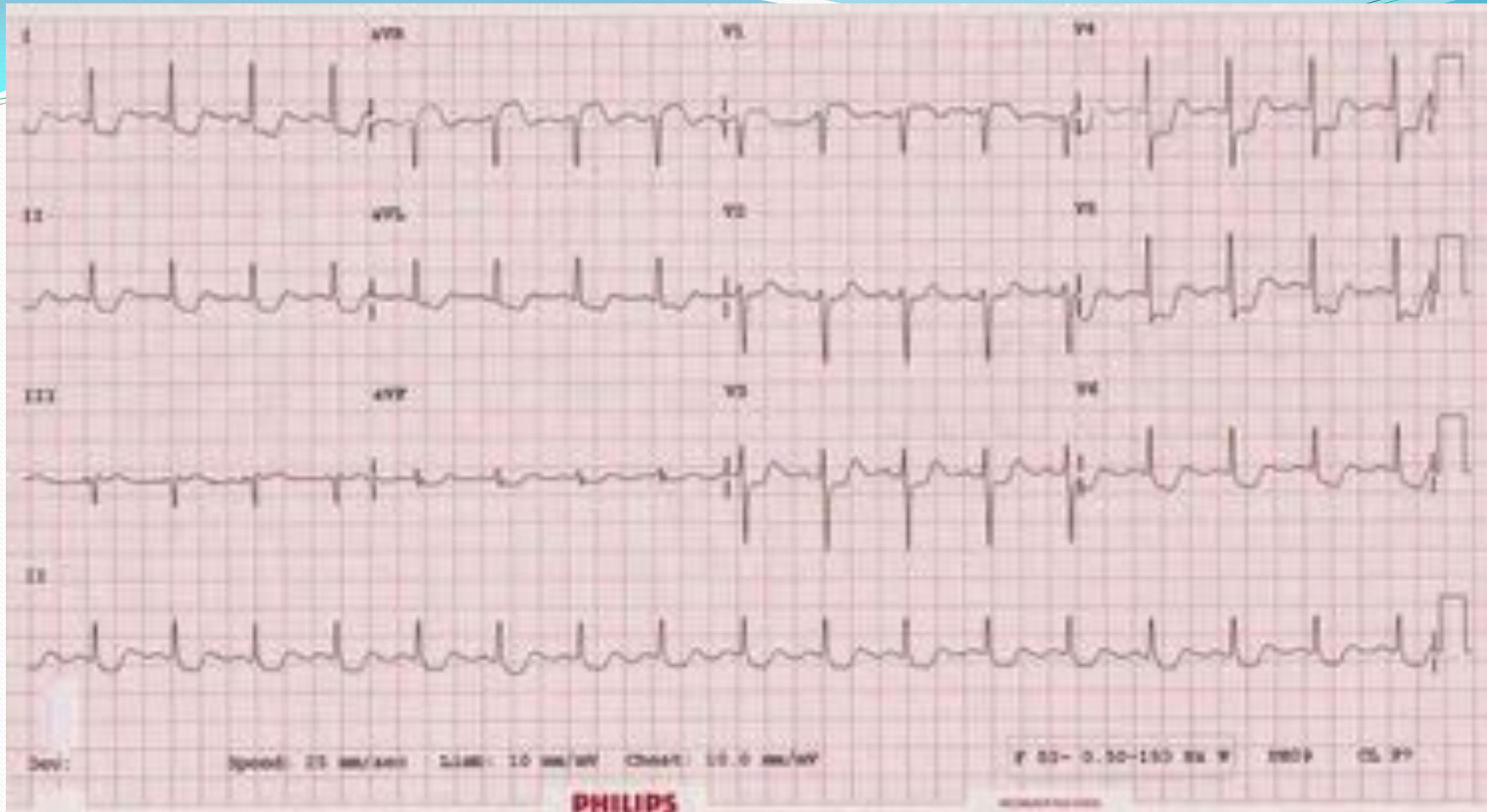
Another classic example of the LMCA / 3VD ECG pattern

# FACEM 2012.2 Exam Q

A 76 year old woman presents to your emergency department with one hour of severe chest pain.

## Observations:

BP	100/55	mmHg	
O <sub>2</sub> Saturation	95	%	on room air
Temperature	36	°C	



Dev: Speed: 25 mm/sec Scale: 10 mm/mV Chart: 10.0 mm/mV F 03-03-10 08 W 1004 05 37

**PHILIPS**



- Question

- a. Describe and interpret the ECG (50%)
- b. Outline your management (50%)

- The overall pass rate for this question was 99/128 (77.3%)
- Pass Criteria Describe
  - Rate 102 bpm (acceptable range 90-110)
  - Rhythm sinus
  - Axis normal
  - PR normal
  - QRS normal
  - QT > 600
  - ST elevation in aVR and V<sub>1</sub>. aVR > V<sub>1</sub>
  - Marked ST depression in V<sub>3</sub>-V<sub>6</sub> Maximum in V<sub>4-5</sub>
  - Better answers showed precision in descriptive items, i.e 3mmST elevation, QRS 100msec
  - Must give good systematic description of ECG
- Pass Criteria Interpret
  - Overall impression: Left main or proximal LAD occlusion with high likelihood cardiovascular compromise, high mortality and morbidity and requiring urgent early reperfusion. Correlation with clinical data suggests early cardiogenic shock

## ● Pass Criteria Management

- Full monitoring
- Oxygen
- Aspirin
- Clopidogrel (+/- heparin)
- Analgesia IV narcotic titrated
- Anticipate problems
- Treat hypotension Fluid challenge +/- Inotropes
- External pacing if required
- Patient requires early reperfusion, Early cardiology consult. PCI ideal. Consider thrombolysis if delay or remote
- Review of all cases and timely Mx – ACHS KPI
- Education of junior staff to significance of ECG
- Disposition: cath lab if available. CCC otherwise



Would you thrombolyse STE in aVR?





What do ACC/AHA say?

- *"Fibrinolytic therapy should not be administered to patients with ST depression except when a true posterior (inferobasal) MI is suspected or when associated with ST elevation in lead aVR"*
- The new 2013 ACC/AHA guidelines give this as an indication for thrombolytic therapy
- This is the first time they have recognised that the studies prohibiting thrombolytics for ST depression did not include this high risk group

**Table 4. Indications for Fibrinolytic Therapy When There is a >120-Minute Delay From FMC to Primary PCI (Figure 2)**

	COR	LOE	References
Ischemic symptoms <12 h	I	A	81, 306–311
Evidence of ongoing ischemia 12 to 24 h after symptom onset and a large area of myocardium at risk or hemodynamic instability	IIa	C	N/A
ST depression, except if true posterior (inferobasal) MI is suspected or when associated with ST elevation in lead aVR	III: Harm	B	10, 11, 81, 312, 313

COR indicates Class of Recommendation; FMC, first medical contact; LOE, Level of Evidence; MI, myocardial infarction; N/A, not available; and PCI, percutaneous coronary intervention.

AMI (Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction) trial,<sup>248</sup> the writing committee considers bivalirudin, in combination with oral DAPT, a reasonable anticoagulant alternative for primary PCI in STEMI, regardless of whether pretreatment was given with UFH, especially for patients at higher risk of bleeding and when avoidance of GP IIb/IIIa antagonists is desired. Bivalirudin in this setting may provide a long-term survival benefit related to decreased bleeding but with a higher risk of early stent thrombosis.<sup>249</sup>

## 5. Reperfusion at a Non-PCI-Capable Hospital

### 5.1. Fibrinolytic Therapy When There Is an Anticipated Delay to Performing Primary PCI Within 120 Minutes of FMC: Recommendations

See Table 4 for a summary of recommendations from this section.

cannot be performed within 120 minutes of FMC.<sup>81,306–311</sup> (Level of Evidence: A)

#### Class IIa

1. In the absence of contraindications and when PCI is not available, fibrinolytic therapy is reasonable for patients with STEMI if there is clinical and/or ECG evidence of ongoing ischemia within 12 to 24 hours of symptom onset and a large area of myocardium at risk or hemodynamic instability. (Level of Evidence: C)

#### Class III: Harm

1. Fibrinolytic therapy should not be administered to patients with ST depression except when a true posterior (inferobasal) MI is suspected or when associated with ST elevation in lead aVR.<sup>10,11,81,312,313</sup> (Level of Evidence: B)

#### 5.1.1. Timing of Fibrinolytic Therapy

The benefits of fibrinolytic therapy in patients with ST elevation or bundle-branch block MI are well established, with a time-dependent reduction in both mortality and morbidity rates during the initial 12 hours after symptom onset.<sup>81,306–311,314–320</sup> As noted in Section 3.2, even when inter-hospital transport times are short, there may be advantages to the immediate delivery of fibrinolytic therapy versus any delay to primary PCI for patients with STEMI and low bleeding risk who present within the first 1 to 2 hours of symptom onset.<sup>320,321</sup> Benefit from fibrinolytic therapy in patients who present >12 hours after symptom onset has not been established,<sup>81,307,309,312,321</sup> although there remains consensus that consideration should be given to administering a fibrinolytic agent in symptomatic patients presenting >12 hours after symptom onset with STEMI and a large area of myocardium at risk or hemodynamic instability if PCI is

# STE in aVR with diffuse ST depressions

- aVR elevation with ST depressions in a patient
  - whose pain is controlled by medical management
  - whose ST deviation largely resolves, and
  - is haemodynamically and electrically stable,can be managed medically with extremely vigilant observation, continuous 12-lead ECG monitoring, and next day Cath

# Should you activate the cath lab?

- Many would call this a “STEMI-equivalent” and should go immediately to the cath lab
- One could argue that, when there is diffuse subendocardial ischemia (which may or may not be left main insufficiency, but could also be LAD insufficiency, or 3VD), it is reasonable to treat medically with GTN, Aspirin, antithrombotics, dual antiplatelet therapy & re-assess.

- 
- Often, the thrombus will cool off and the chest pain and ECG findings will resolve, and the angiogram can be done the next day.
  - *Ischaemia refractory to medical therapy (persistent symptoms or persistent ECG findings) require emergent cath.*

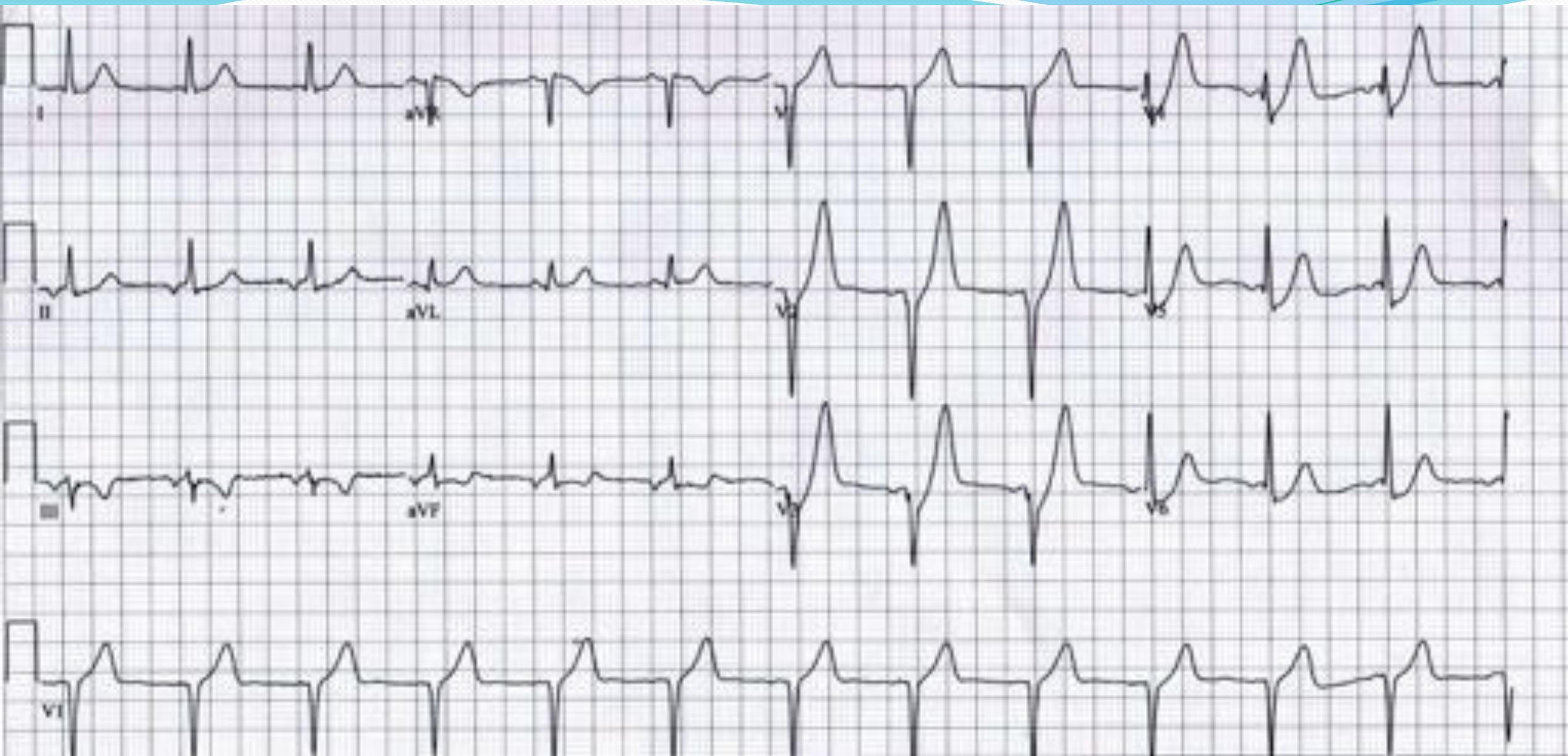


What's the deal with Clopidogrel?

- Clopidogrel treatment  $\leq 7$  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).



- 
- Which is the only other condition with *ST depression* where you would consider emergently activating Cath lab?





- de Winter pattern is *STEMI equivalent*
- How and why is it different to Wellens?

# Take home points

- Don't ever forget the aVR lead!!
- STE with angina indicates LMCA, Prox LAD or 3VD
  - STE in both aVR and V<sub>1</sub> – LMCA or Prox LAD stenosis
  - STE in aVR > V<sub>1</sub> – LMCA Stenosis
- Better outcomes with PTCA, Stent, CABG

Final Take home point

Ignore Lev's voice at your own peril

!!