

# Introduction to EKG's The Basics

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# Introduction to EKG's

- August 8<sup>th</sup>: Basics
- August 15<sup>th</sup> : Review of Rate, Axis, Rhythm
- August 22<sup>nd</sup> : Conduction abnormalities;  
Chamber enlargement
- **August 29<sup>th</sup>: Ischemia, Injury, Infarct,  
Practice EKG's**

# Myocardial Infarctions

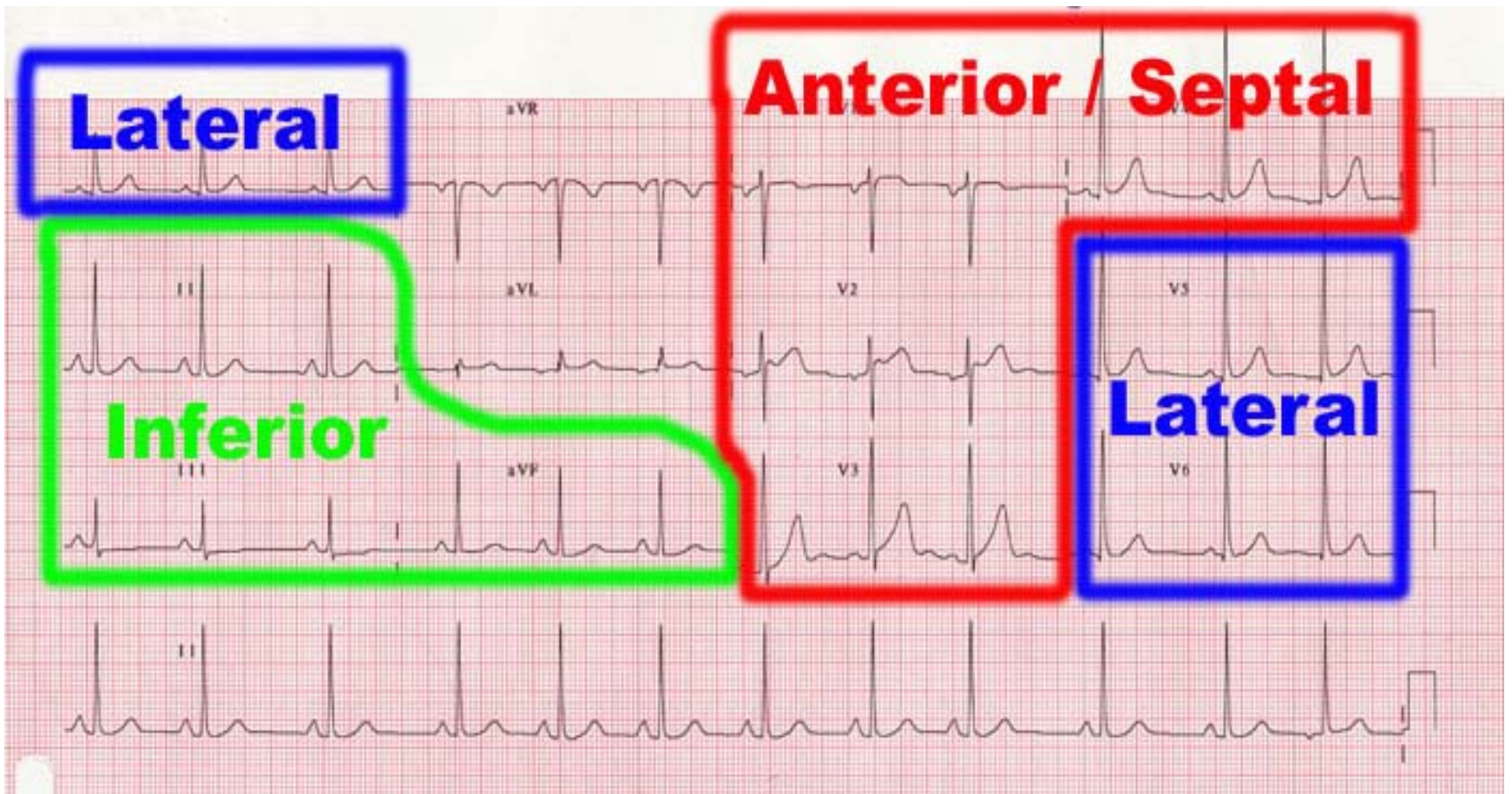
- **EKG designation**
  - Inferior Wall MI: Leads II, III, AVF
  - Anterior Wall MI: Precordial/chest Leads
    - Anteroseptal: V1, V2
    - Anteroapical: V3, V4
    - Anterolateral: V5, V6, Leads I and AVL
  - Posterior Wall MI
    - V1: increased R to S ratio of  $\geq 1$

**Lateral**

**Anterior / Septal**

**Inferior**

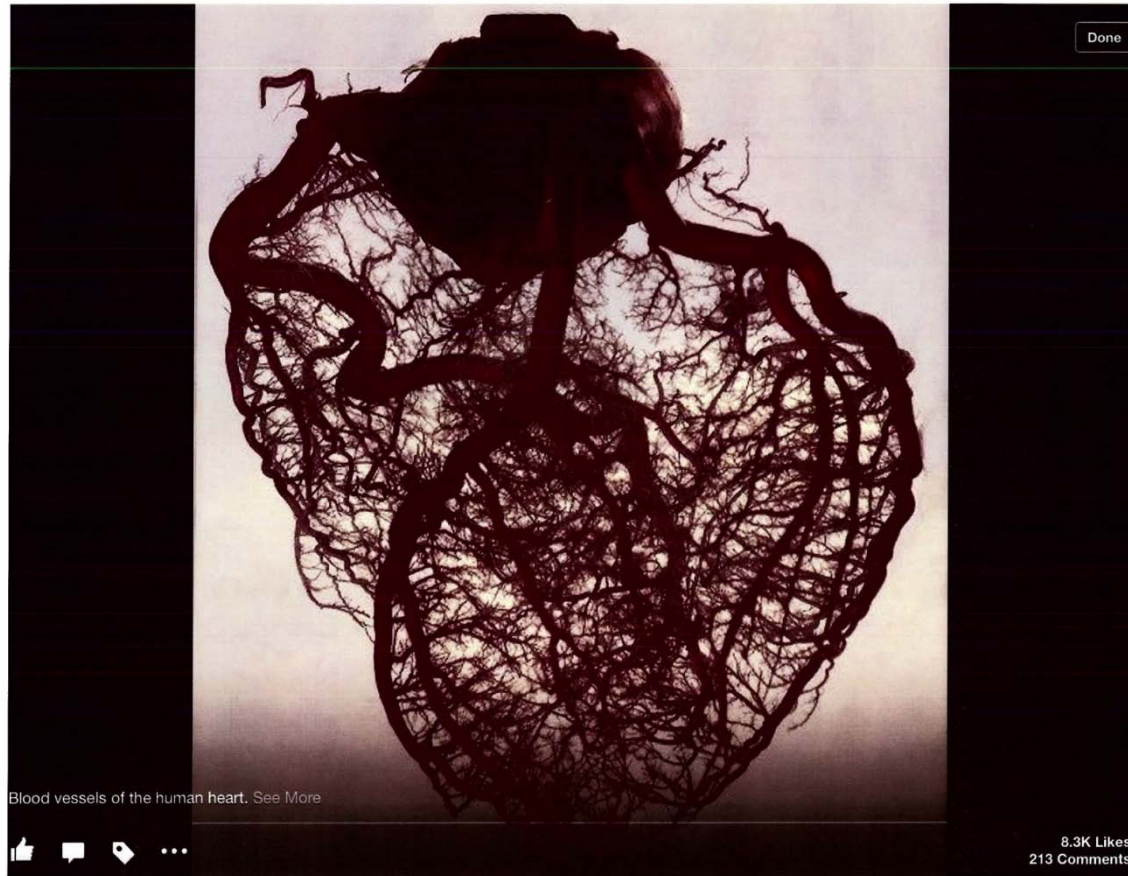
**Lateral**



# Coronary Arteries

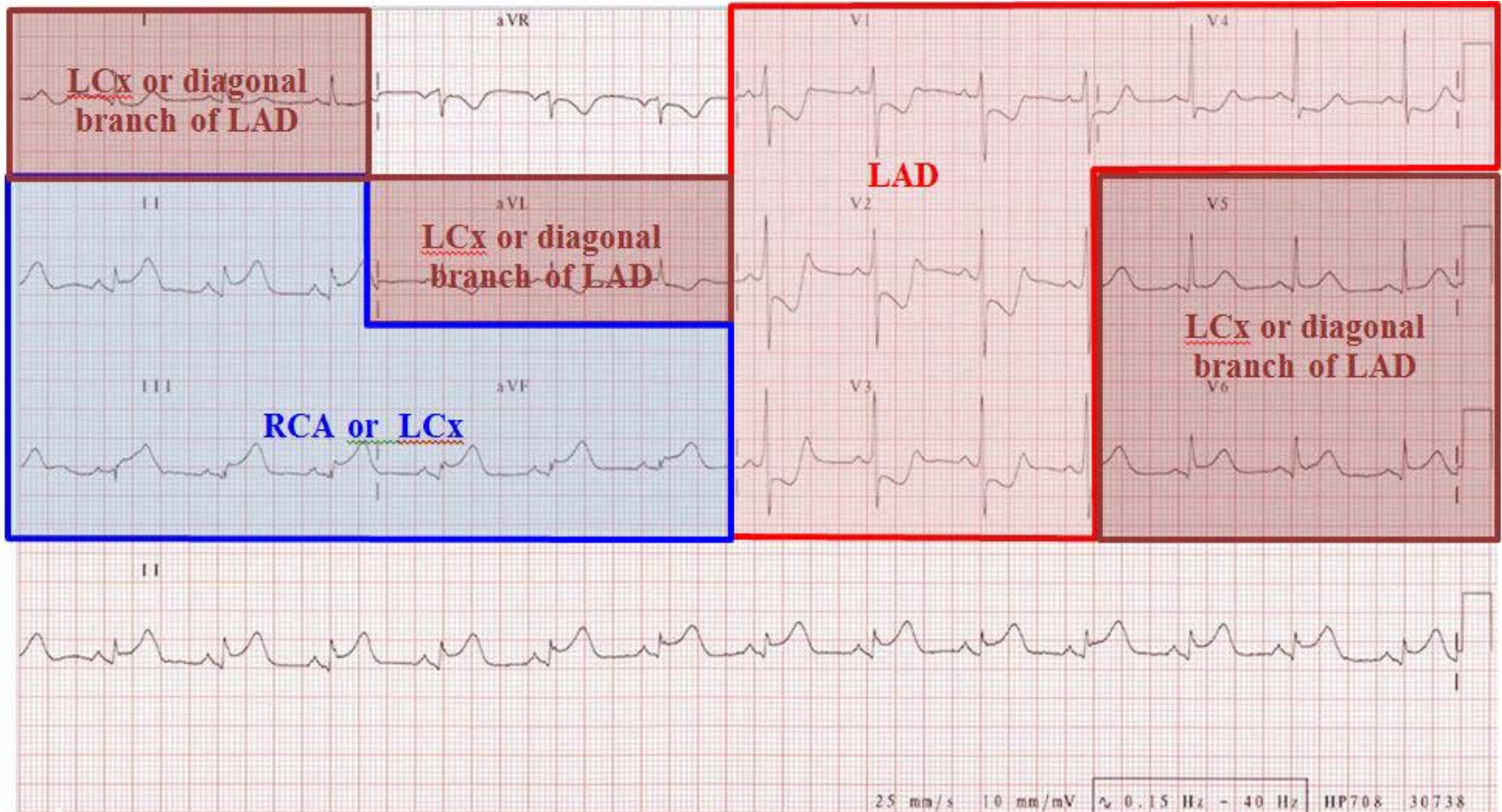
- **LEFT MAIN (widow maker):** Supplies entire anterior wall of the heart  
Bifurcates into the Left Anterior Descending (LAD) and the Circumflex artery.
- **Right Coronary Artery (RCA):** Supplies the inferior wall of the heart

# CORONARY ARTERIES



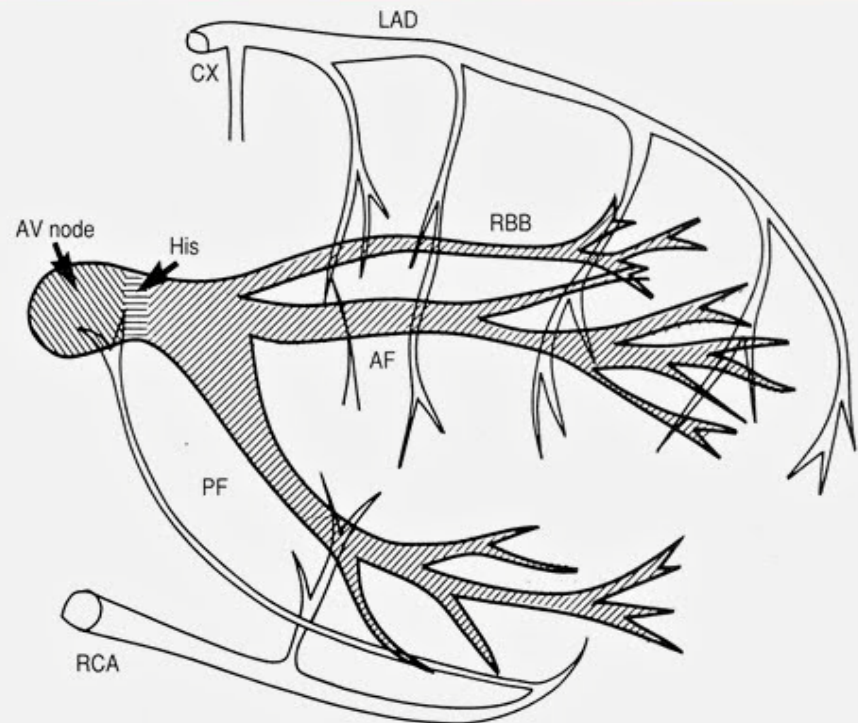
# RCA dominant in most humans:

Dominance determined by vessel giving off branch to the AV node



# Blood Supply to the Conduction

- Sinus node and SA region : 55% by RCA and 45% LCx
- AV node and proximal His: 90 RCA.
- Distal His, RBB and AF by septal branches of LAD
- Posterior fascicle by septal branches of RCA/LAD



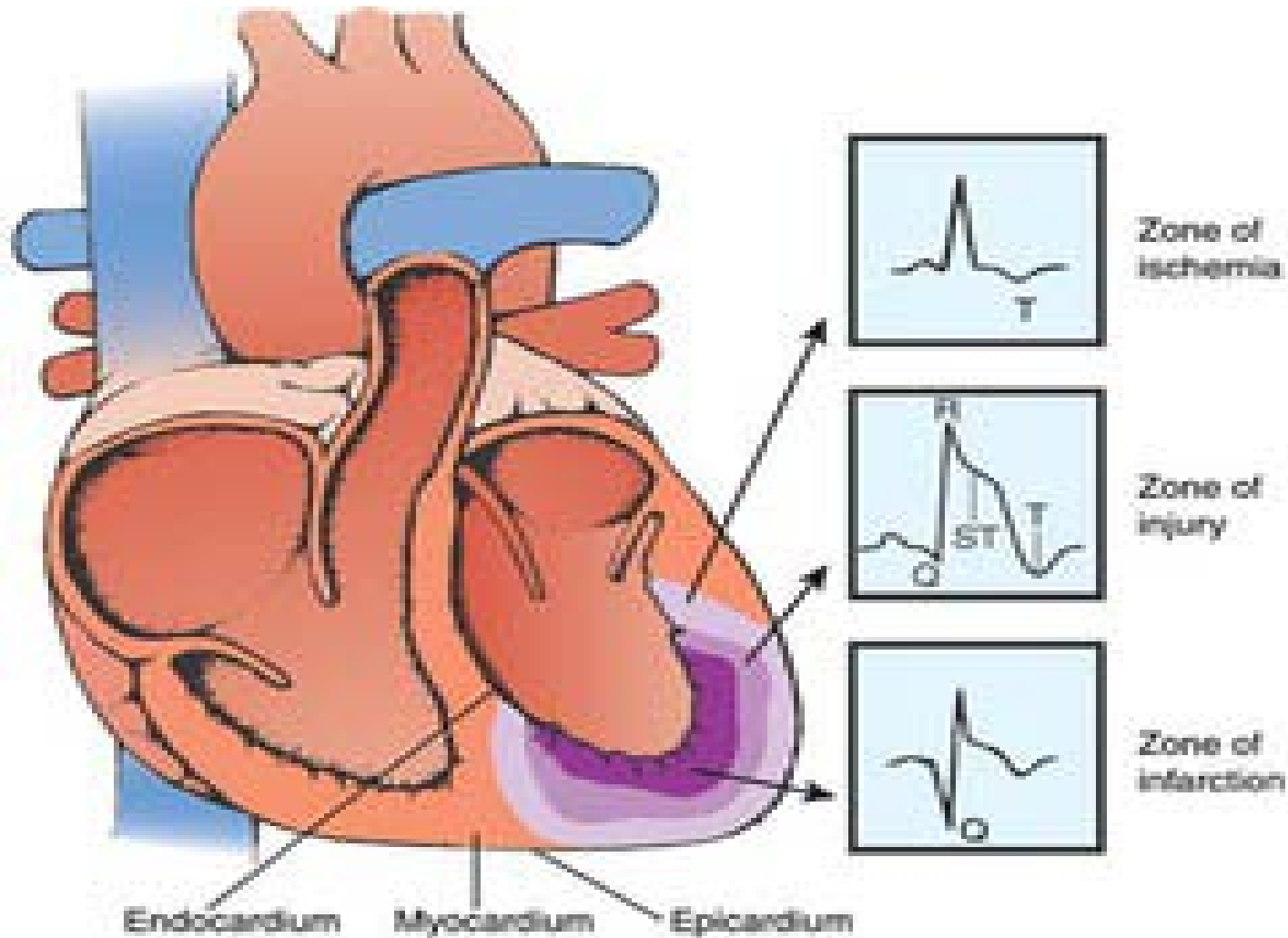
**Figure 1-23** Scheme of the blood supply to the AV conduction system. AF, anterior fascicle; PF, posterior fascicle; His, His bundle; RBB, right bundle branch.



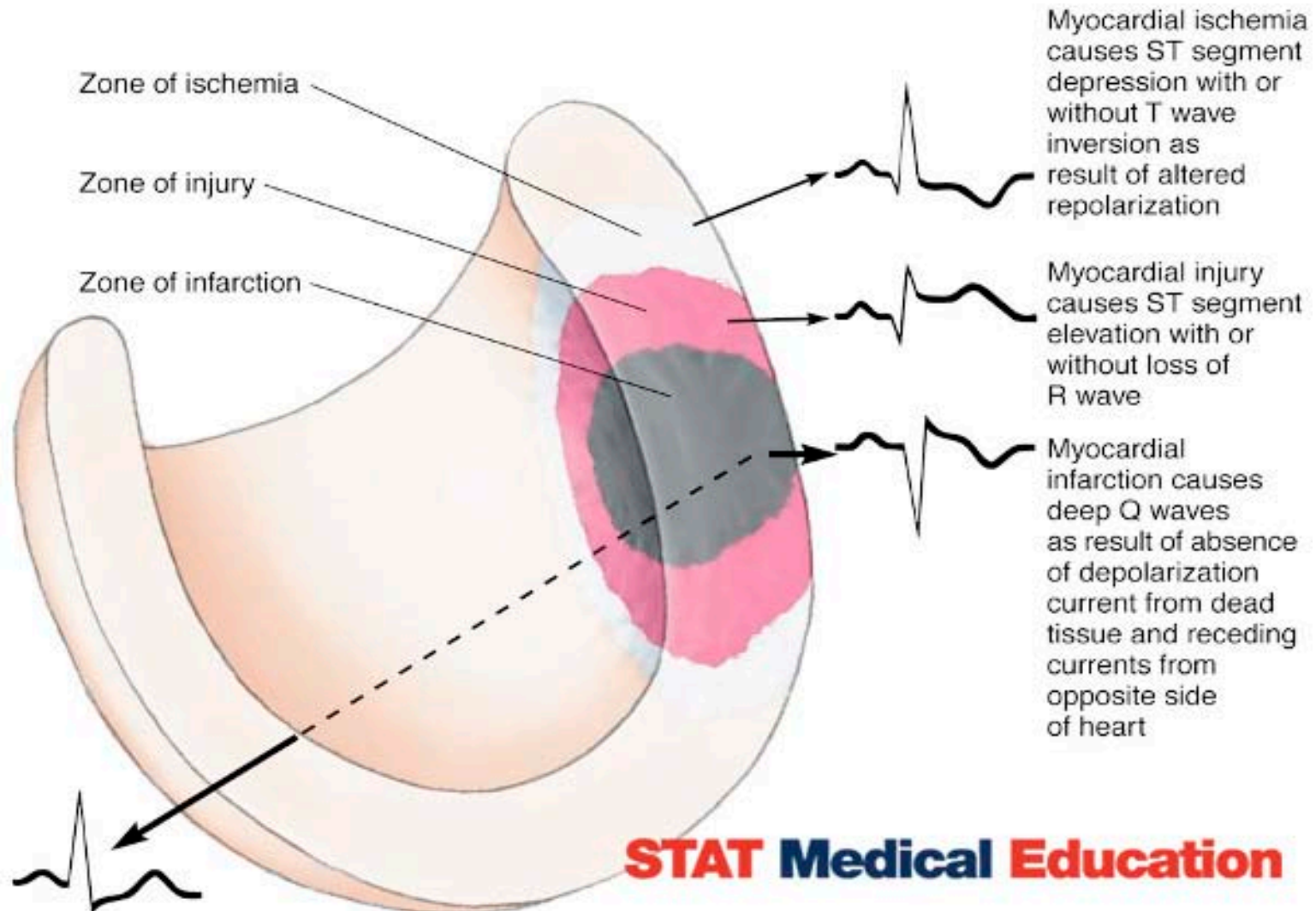
# ISCHEMIA, INJURY, INFARCT

- **Ischemia:** Characterized by J-point depression of 1mm or more below the baseline with Upsloping, horizontal or downsloping ST segment depression of 2mm or more at .08 sec past J-point.
- **Injury:** Characterized by J-point elevation with ST segment elevation
- **Infarct:** ST segment elevation with evolving changes; Q-waves (transmural) or deep, inverted, symmetrical T-waves (Subendocardial)

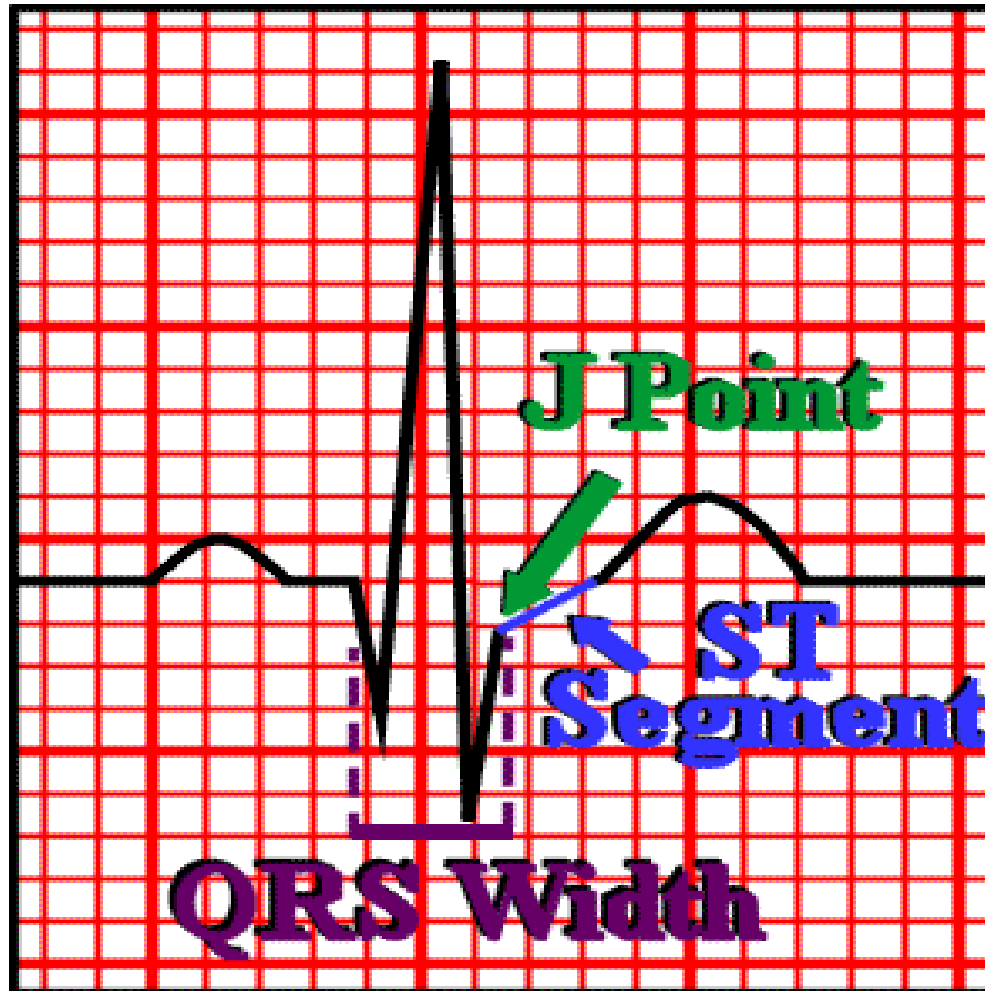
# Ischemia, Injury, Infarct



# ISCHEMIA



# Review of QRS, J-point and ST segment

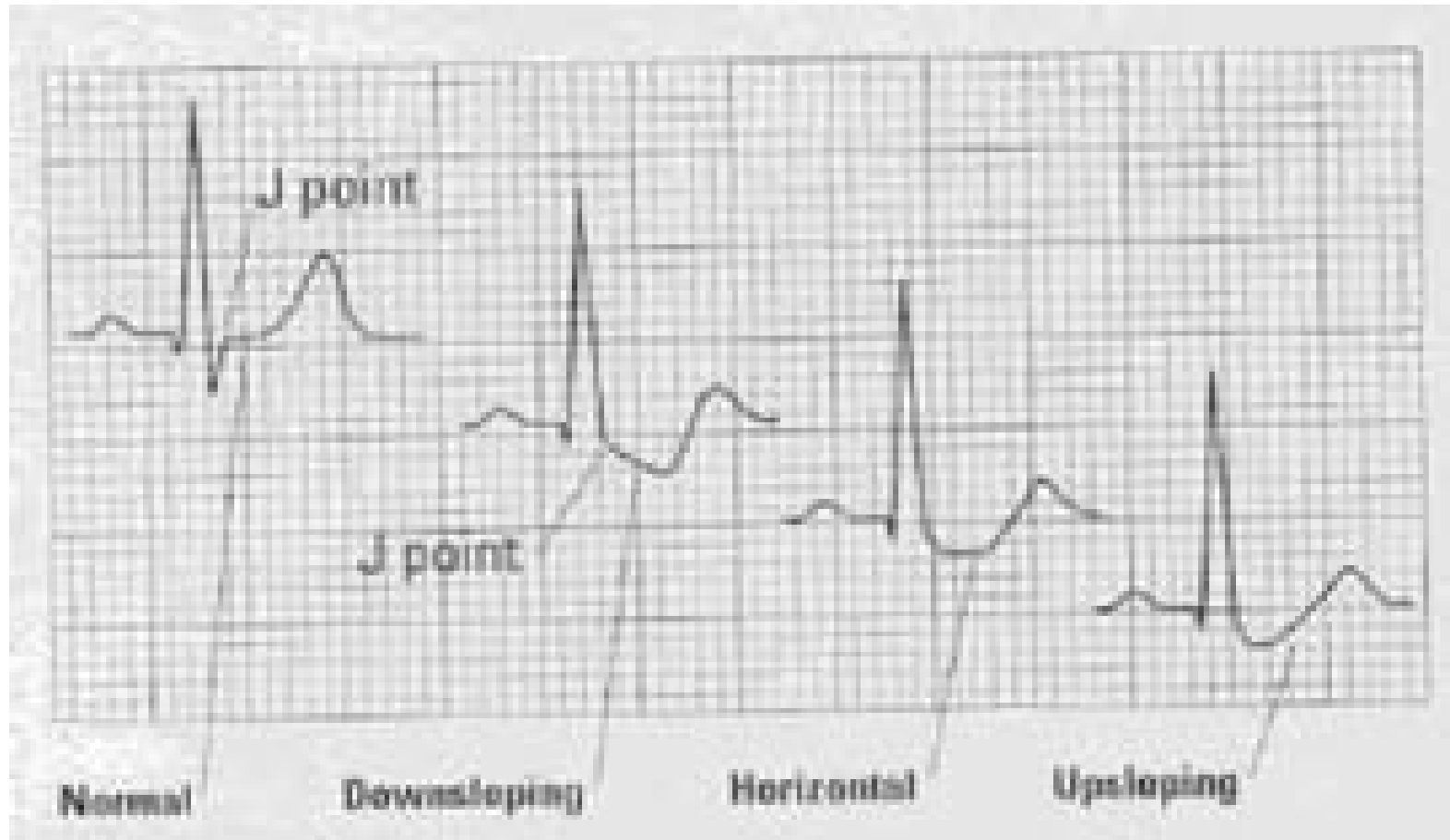


# Determining significance of ST depression

- Find the J-point
- Measure .08 sec (two small boxes) to the right
- If the ST segment is depressed at least 1 mm (1 small box) it is considered significant possible ischemic changes.

# ST Depression

Upsloping, horizontal, or downsloping



# How do I know when to be concerned?

- **Upsloping ST segment depression is of concern**
- **Horizontal ST segment depression is of more concern**
- **Downsloping ST segment depression is of greatest concern**
- **NOTE: clinical correlation is ALWAYS required**

# Additional factors

- ST depression is of greater concern when you see it with minimal amounts of exertion (lower heart rates) when compared to peak exertion
- ST depression is of greater concern when it takes longer to return to baseline after exercise
- ST depression is of greatest concern when changes continue to occur after exertion is terminated



But clinical correlation is ALWAYS required

- **Always monitor the patient**

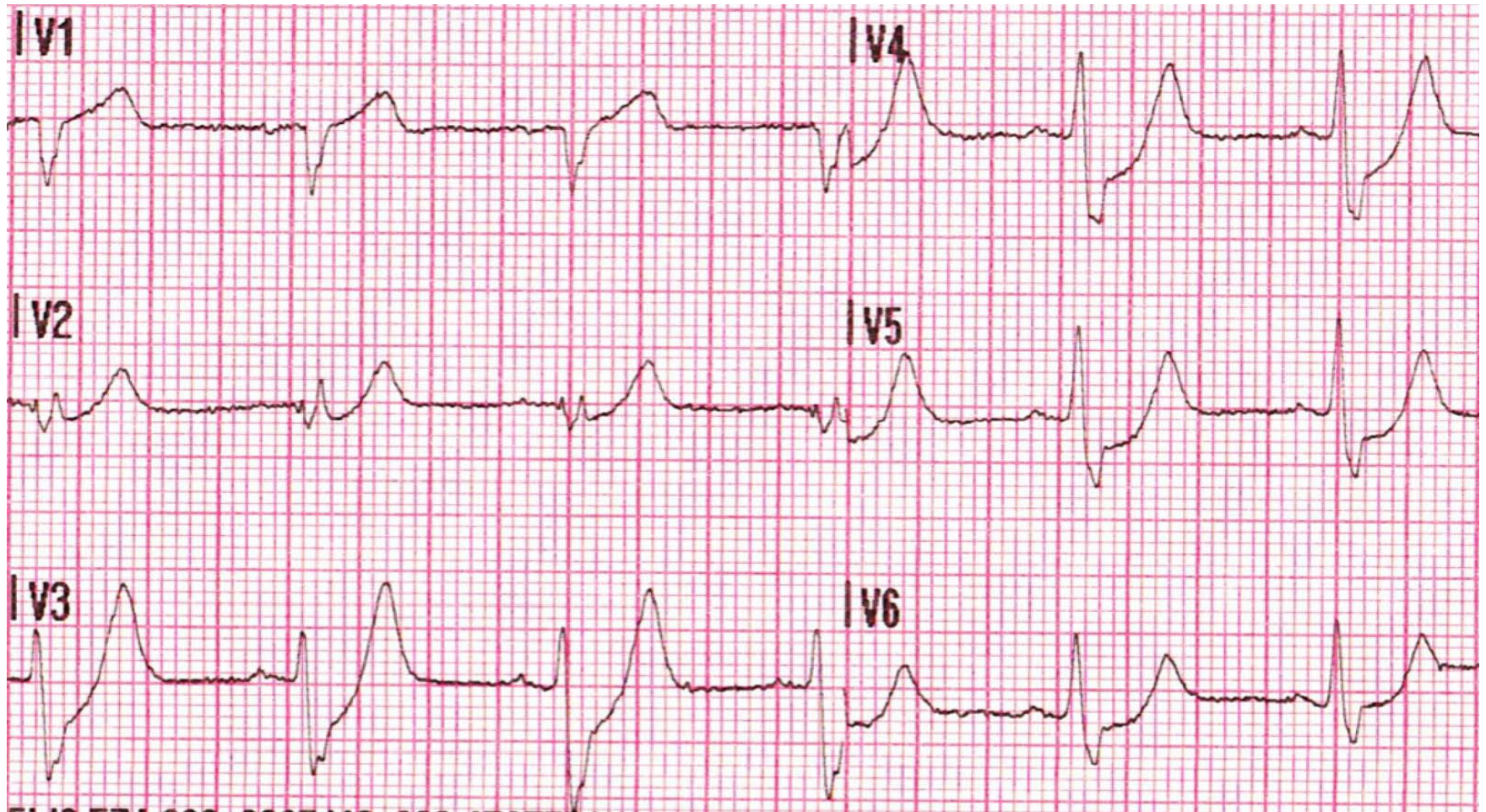
**Vitals**

**Symptoms**

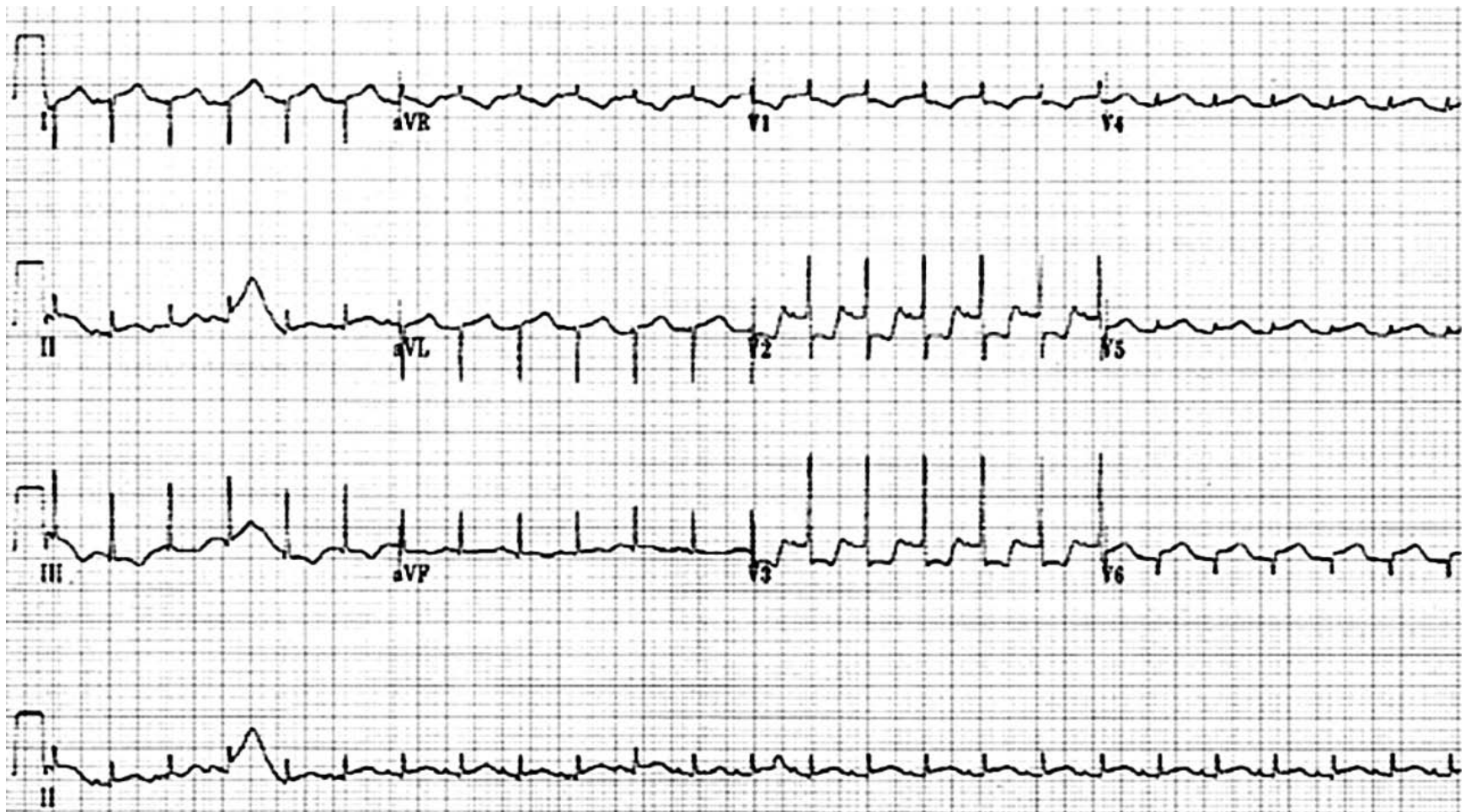
**Just the way the patient looks:**

**Pallor, sweating, ashen**

# Up sloping ST depression

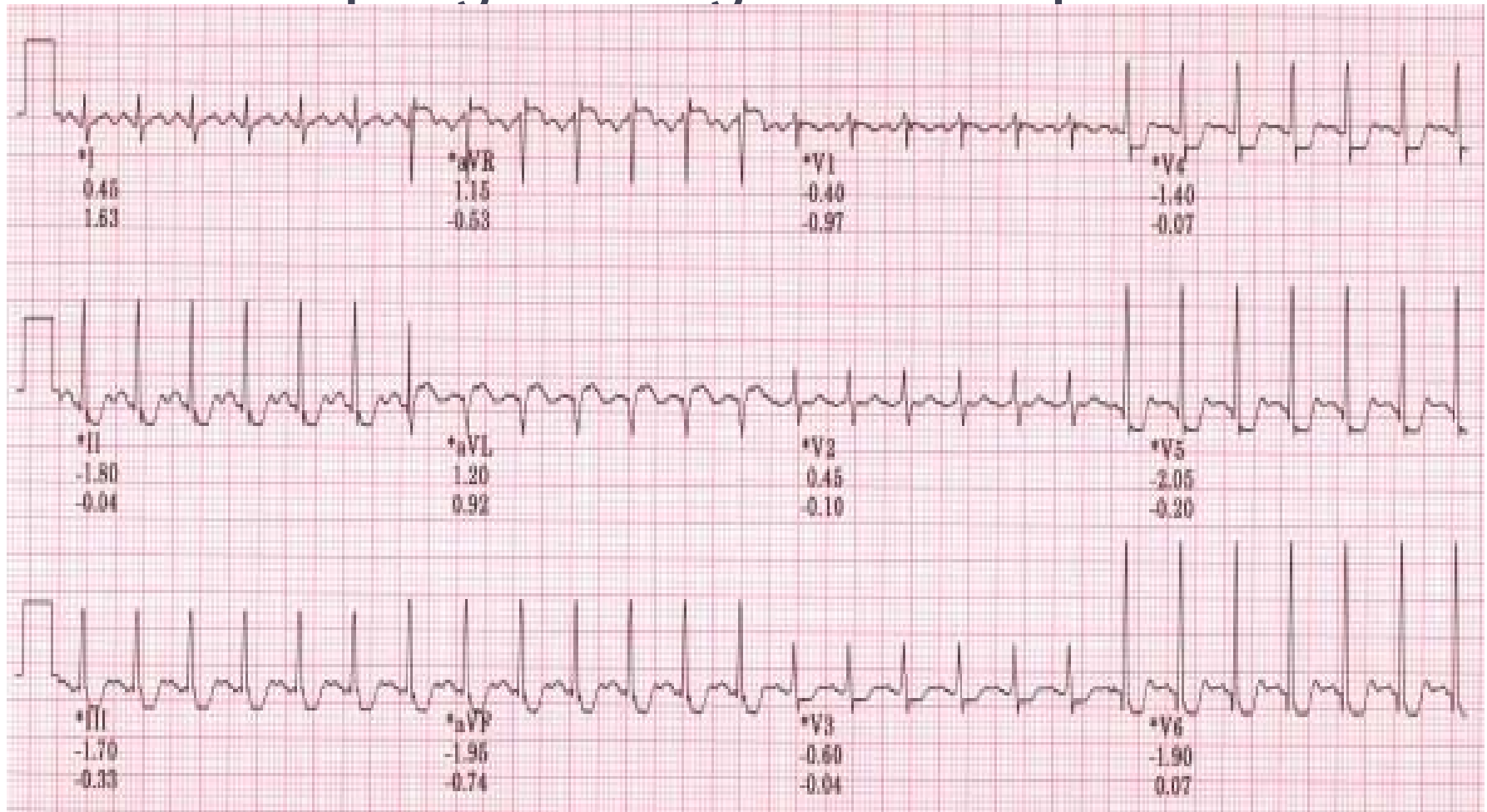


# Horizontal ST Segment Depression

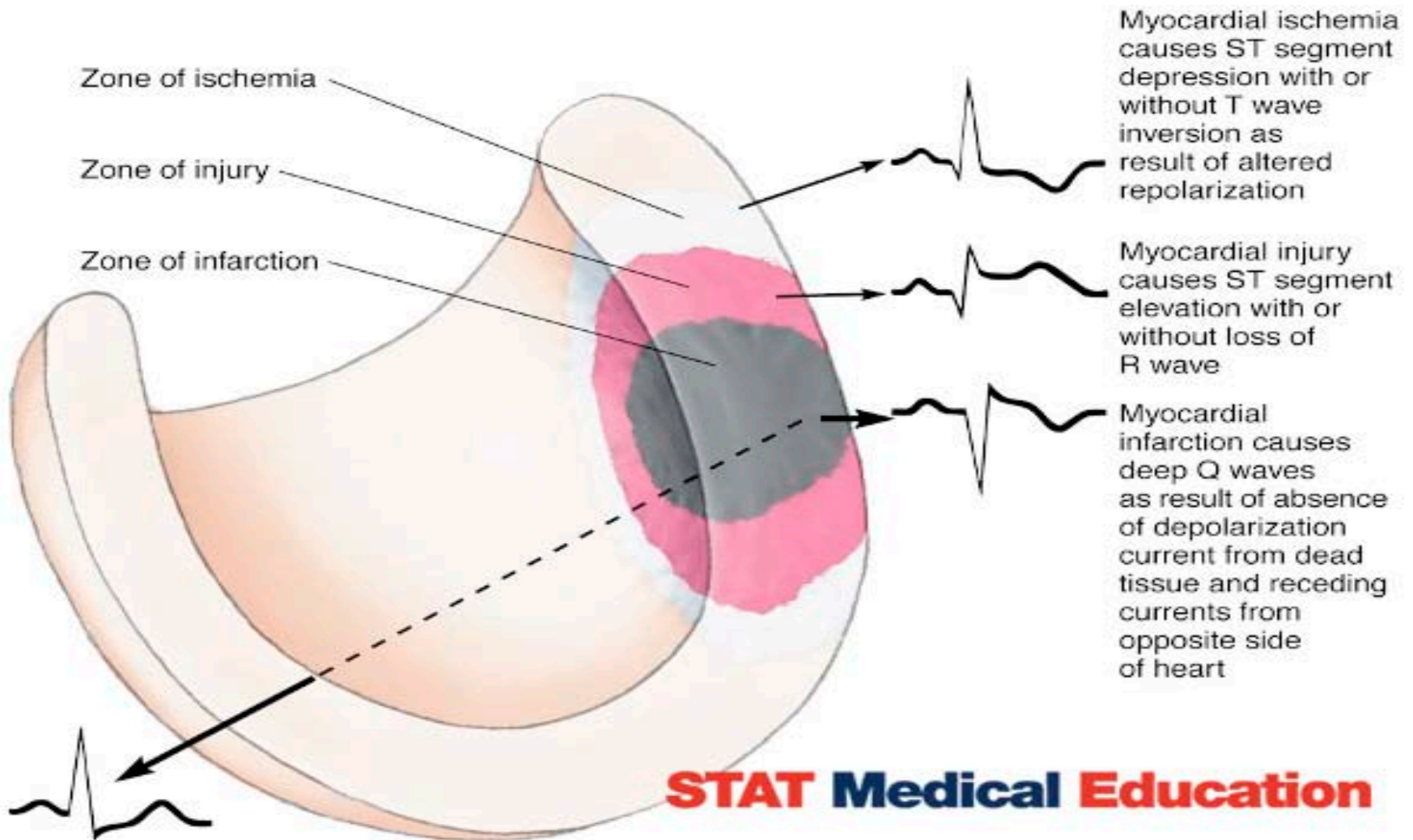


Note posterolateral MI

# Downsloping ST Segment Depression



# INJURY

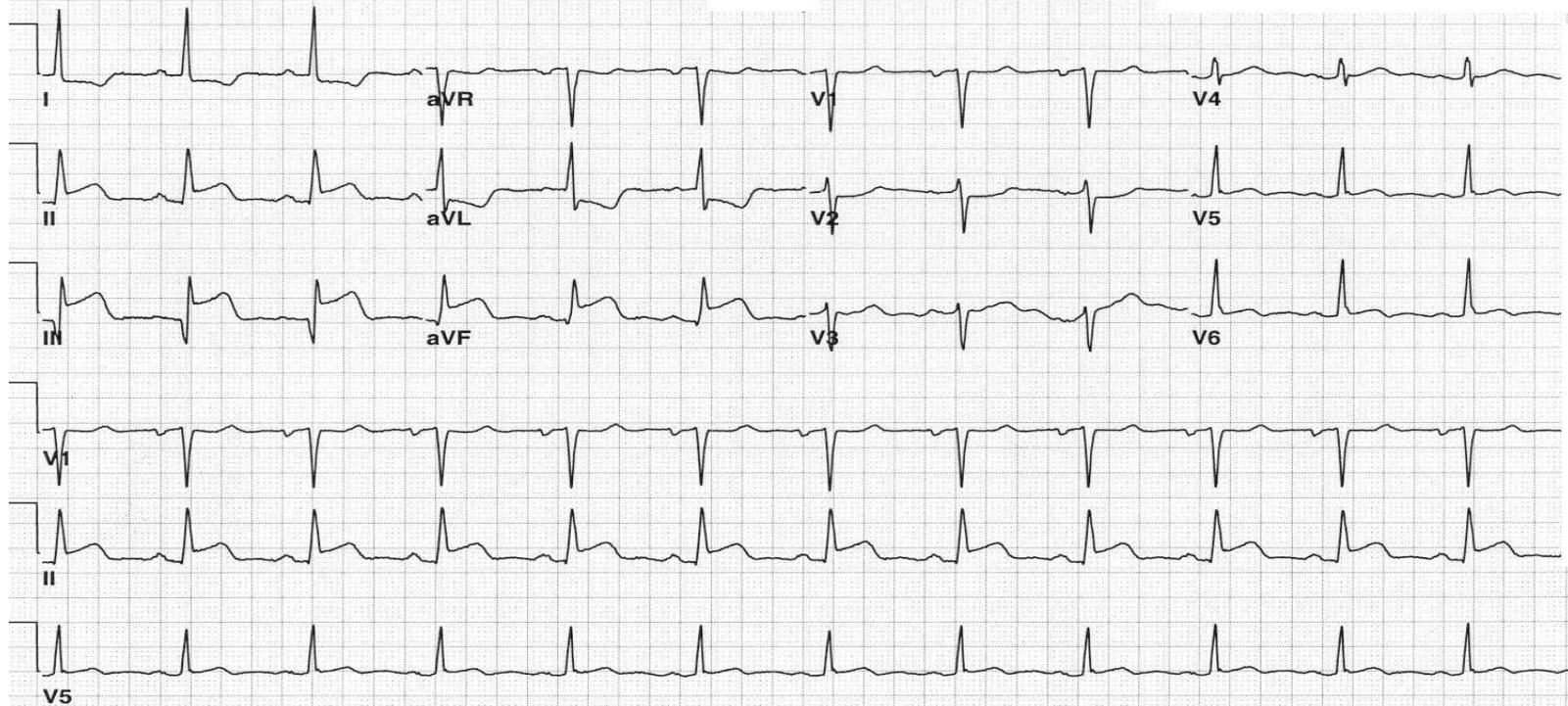


# INJURY

20-JAN 21:40:05

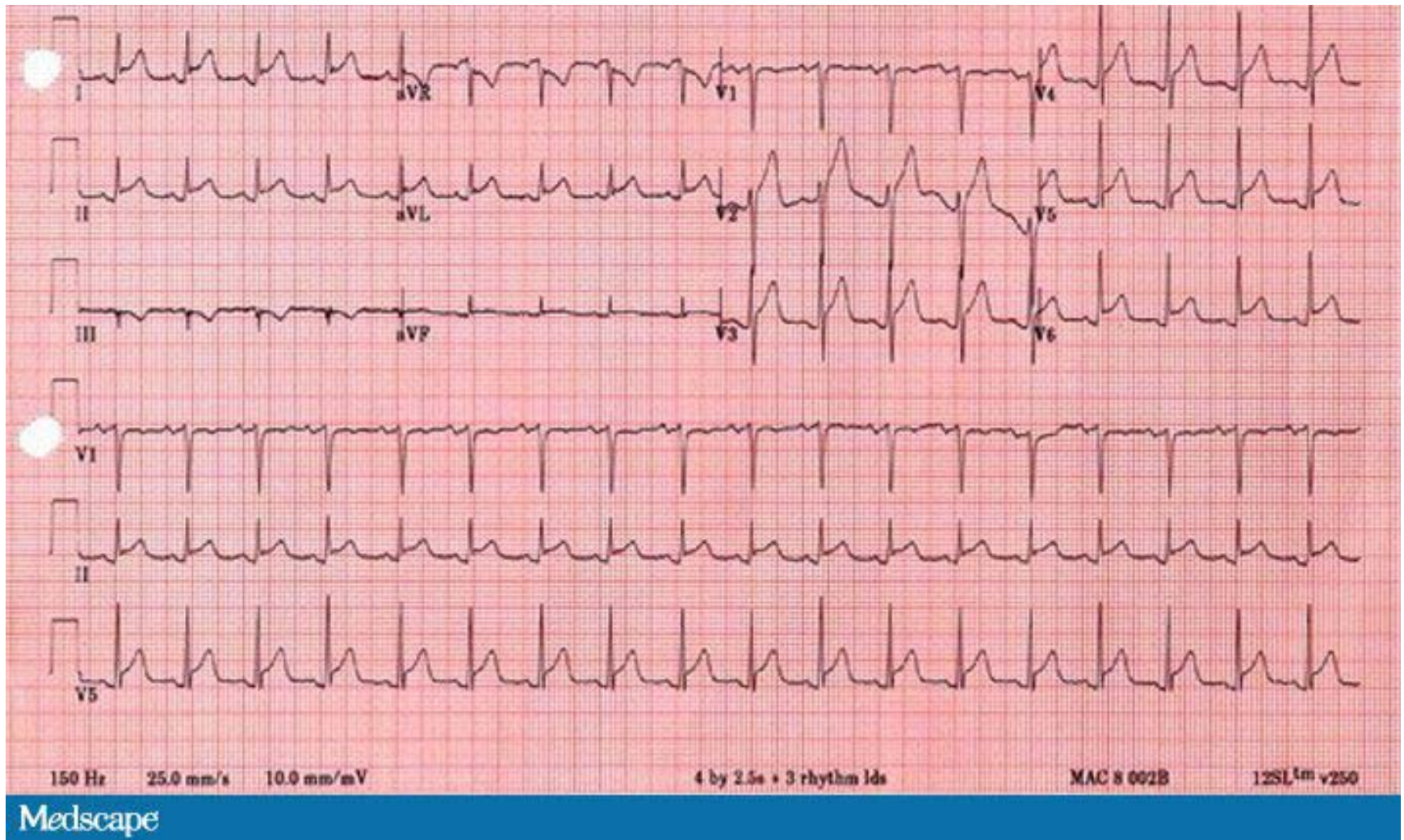
51 yr  
Male Black

Vent. rate	72	BPM
PR interval	198	ms
QRS duration	86	ms
QT/QTc	416/455	ms
P-R-T axes	39 35 109	

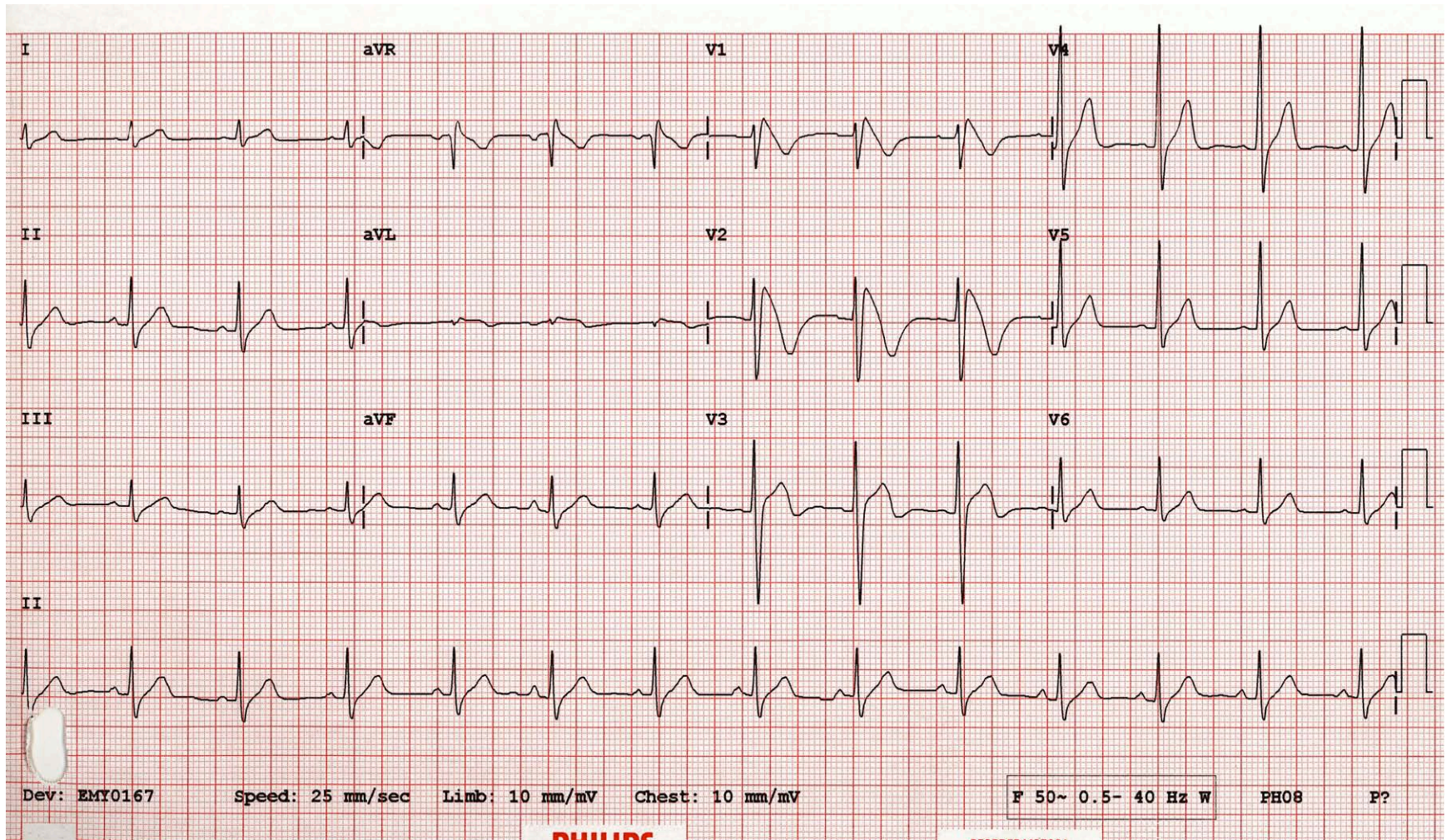


25mm/s 10mm/mV 150Hz 005B 12SL 237 CID: 2

# INJURY due to acute pericarditis

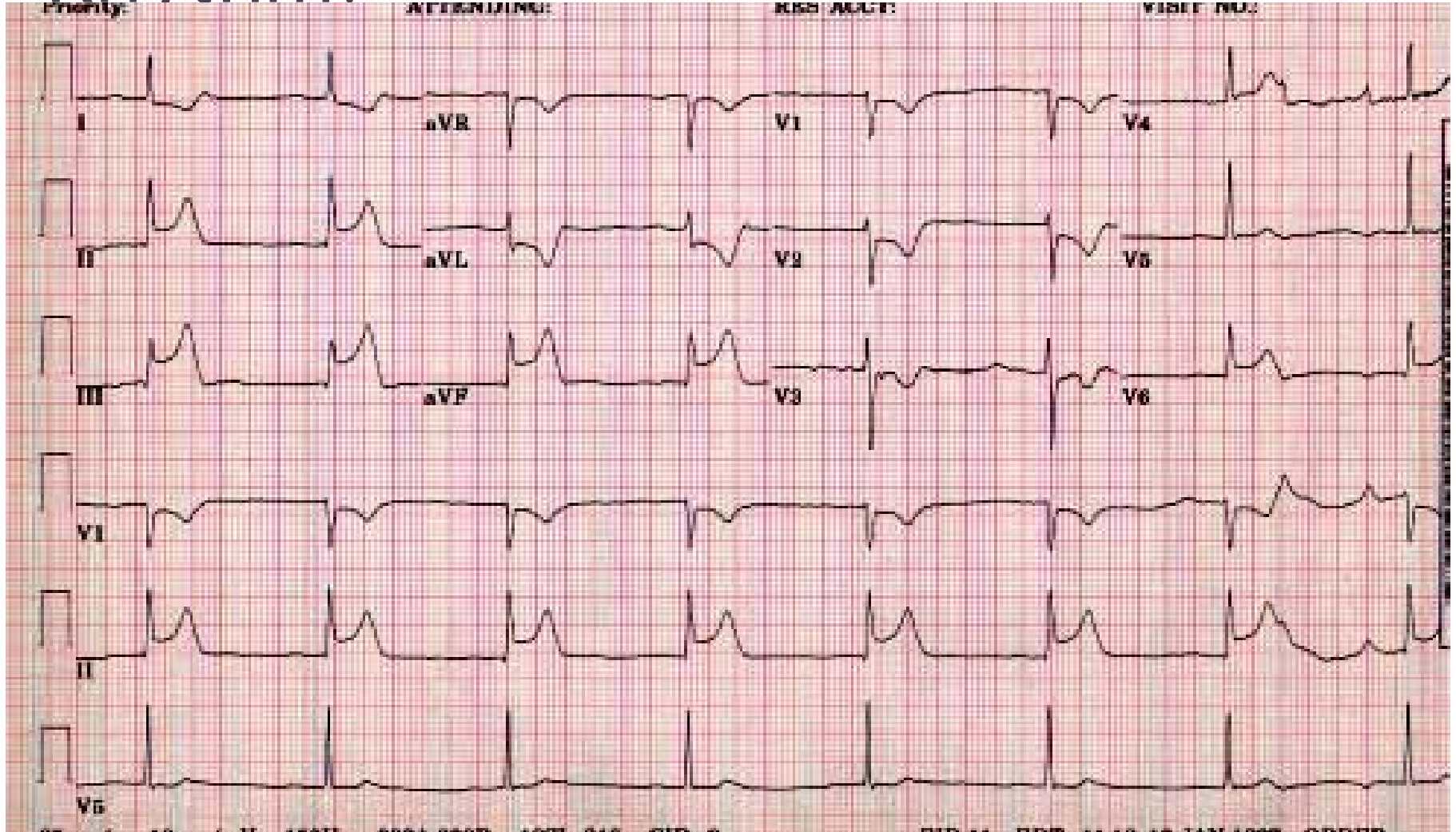


# INJURY

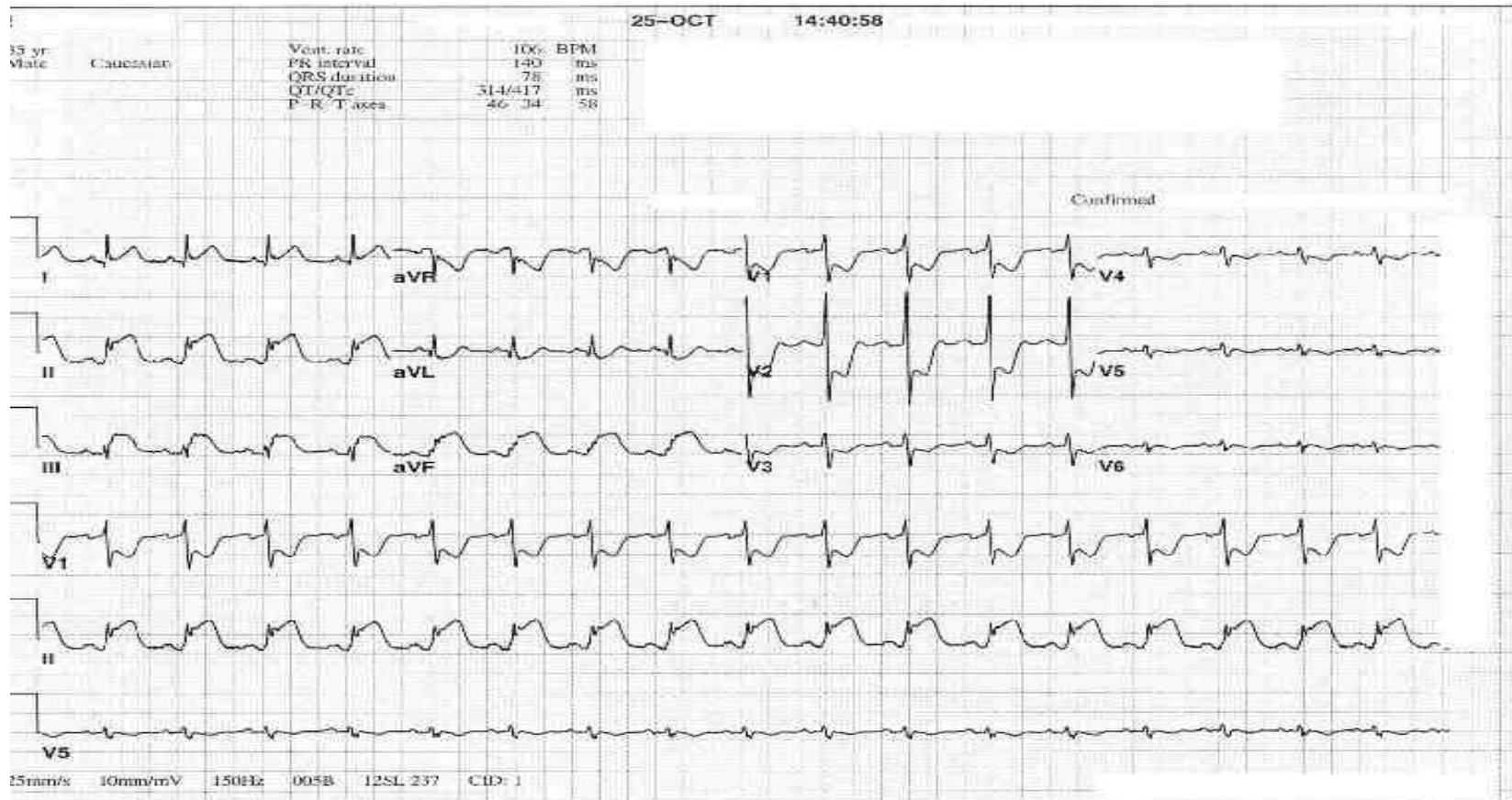




# Acute Inferolateral Injury with Reciprocal Changes: Early inf-lat MI. Rhythm?



# Injury pattern with reciprocal changes

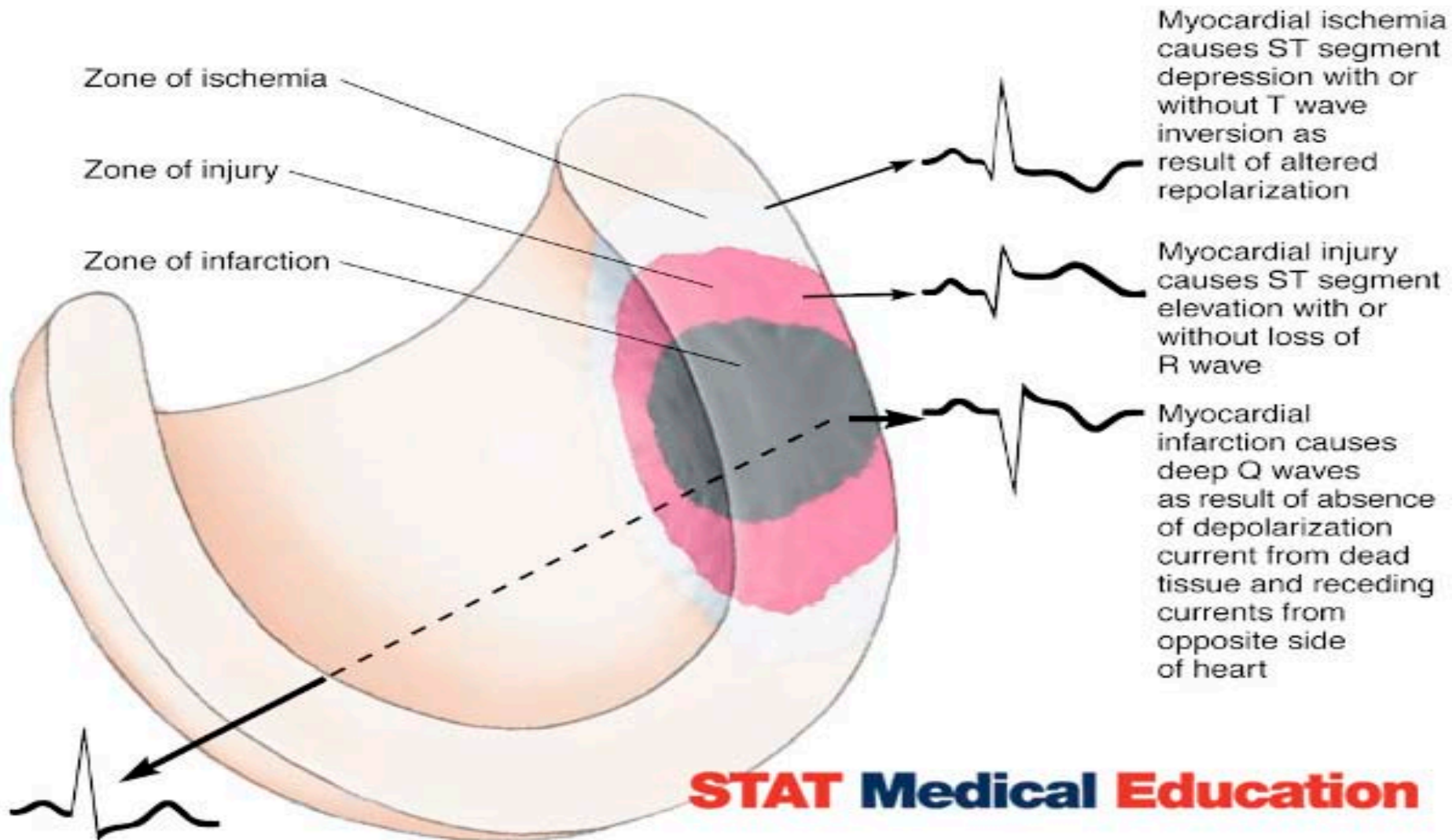


# What are reciprocal changes?

- when ST segment elevation is present in leads that face the acute injury, ST segment depression will often be present in leads that face the “ischemic boundary”.

SITE	FACING	RECIPROCAL
SEPTAL	V1, V2	NONE
ANTERIOR	V3, V4	NONE
ANTEROSEPTAL	V1, V2, V3, V4	NONE
LATERAL	I, aVL, V5, V6	II, III, aVF
ANTEROLATERAL	I, aVL, V3, V4, V5, V6	II, III, aVF
INFERIOR	II, III, aVF	I, aVL
POSTERIOR	NONE	V1, V2, V3, V4

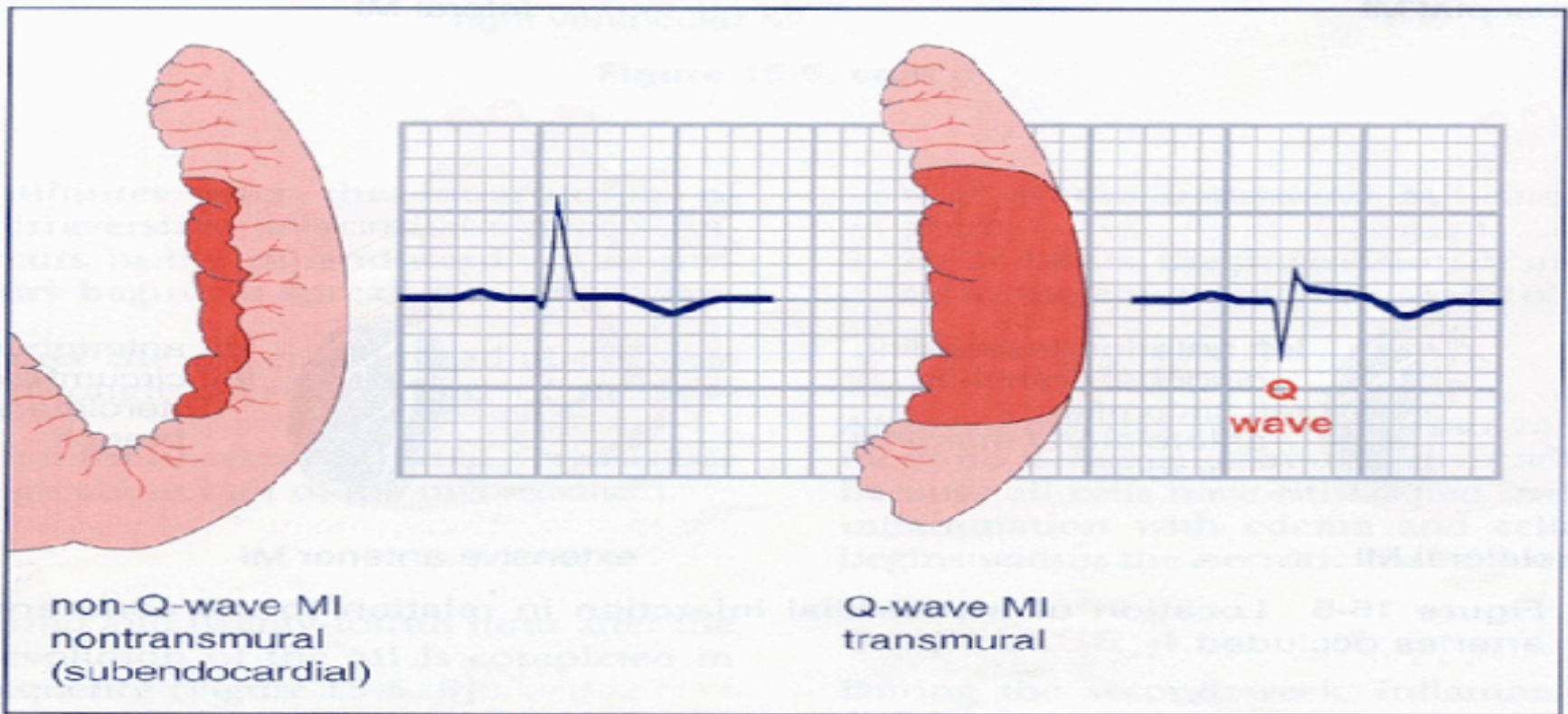
# INFARCT



# MYOCARDIAL INFARCTIONS

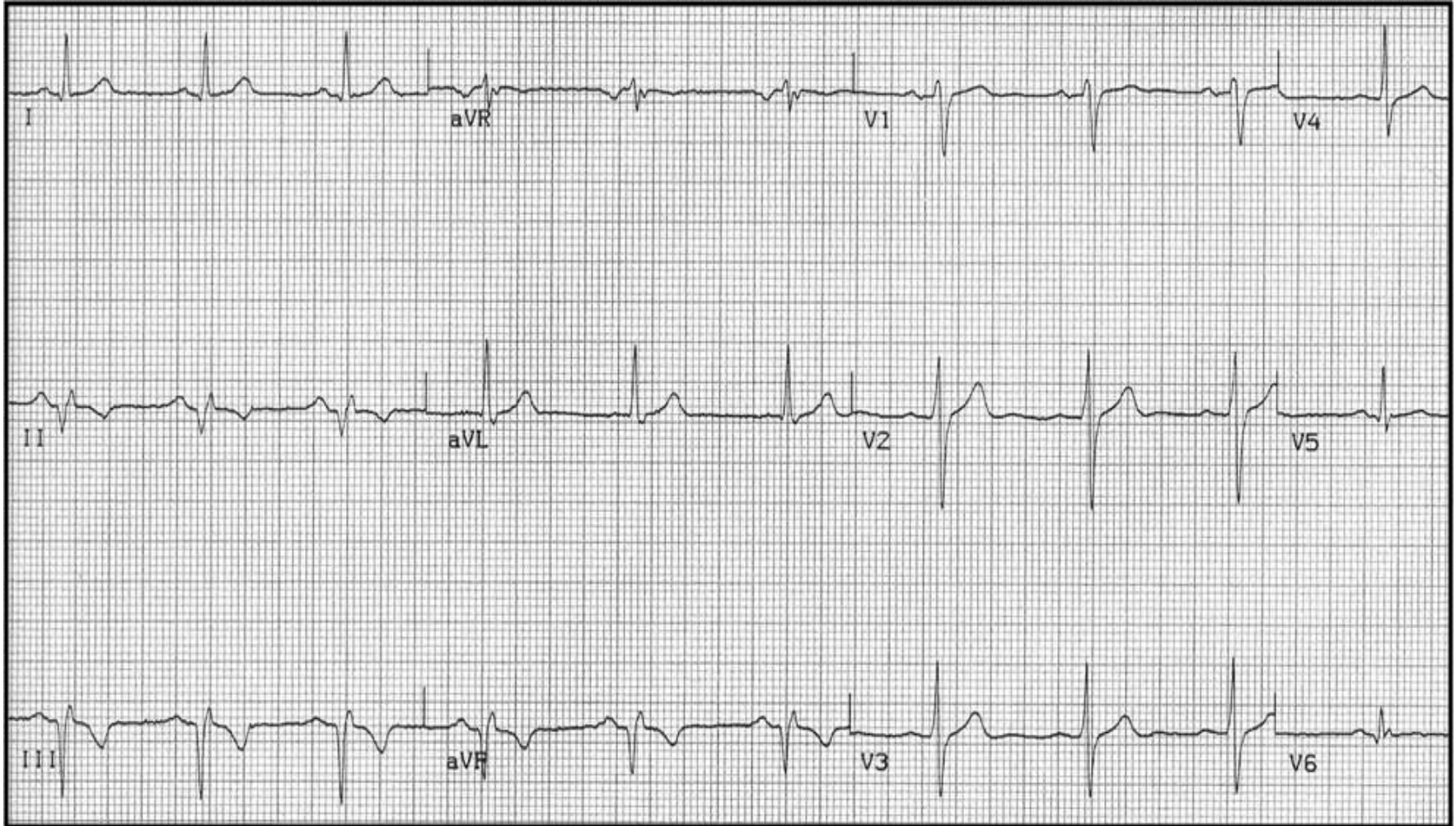
- **Transmural (full thickness)**  
Significant Q waves on EKG (greater than 25% of the height of the R wave)
- **Subendocardial (partial thickness)**  
Symmetrically inverted T waves on EKG

# Subendocardial vs Transmural MI

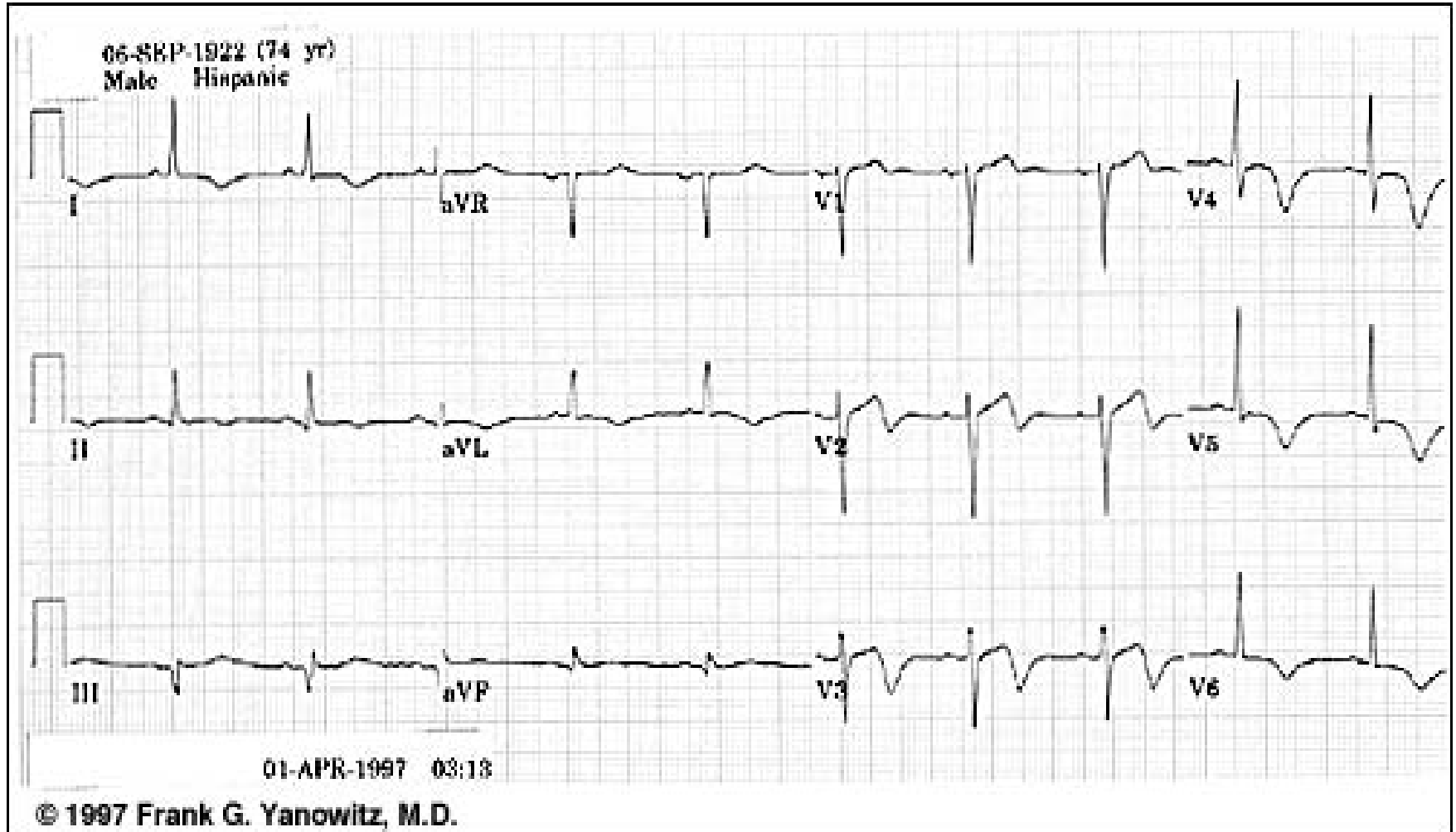


**Figure 15-4** A subendocardial (non-Q-wave) versus a transmural (Q-wave) myocardial infarction.

# Transmural Inferior Wall MI



# Subendocardial (non-Q wave) Anterior Wall MI





# Myocardial Infarctions

- **Locations**

**Inferior Wall**

**Anterior Wall**

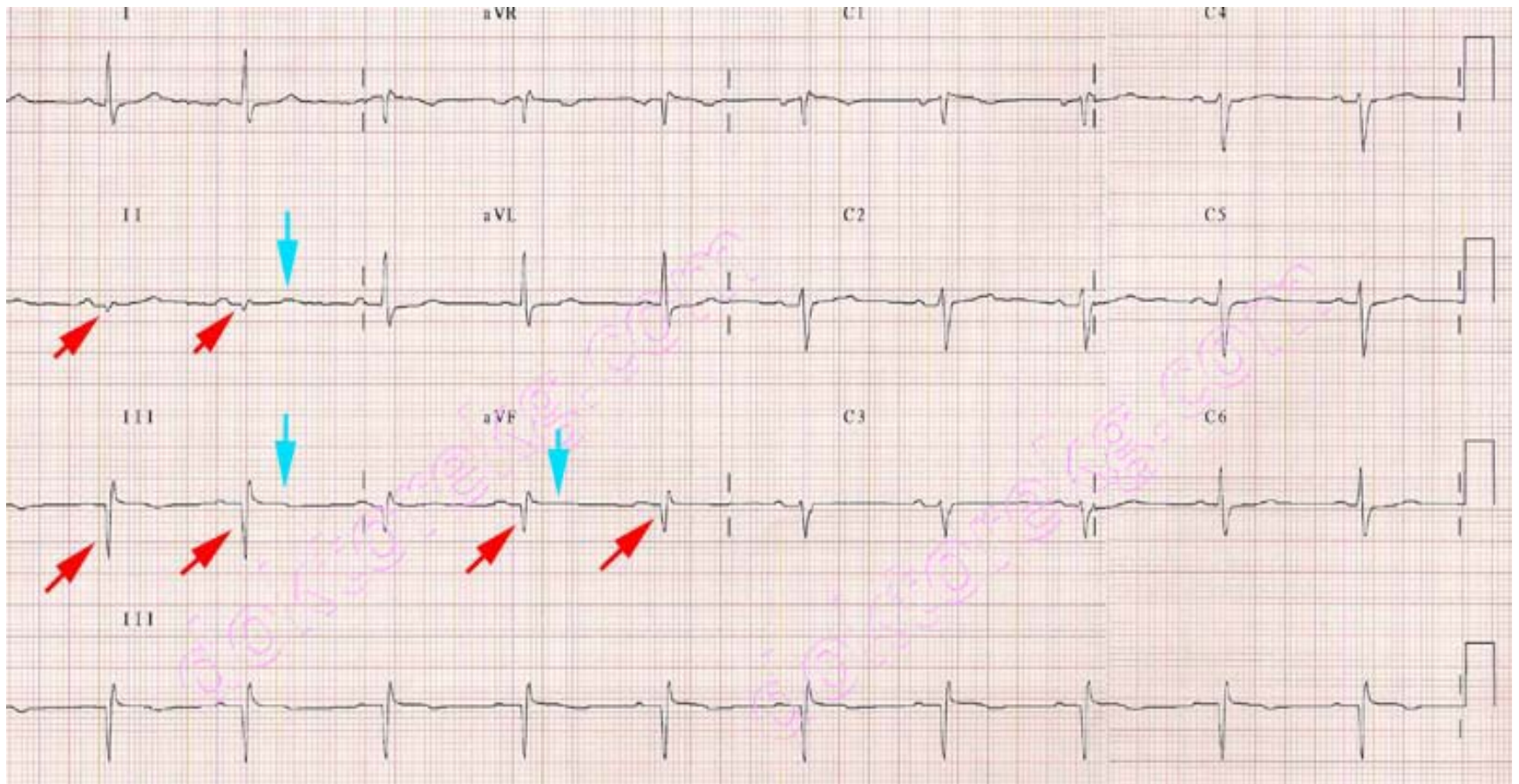
**Lateral Wall**

**Posterior Wall**

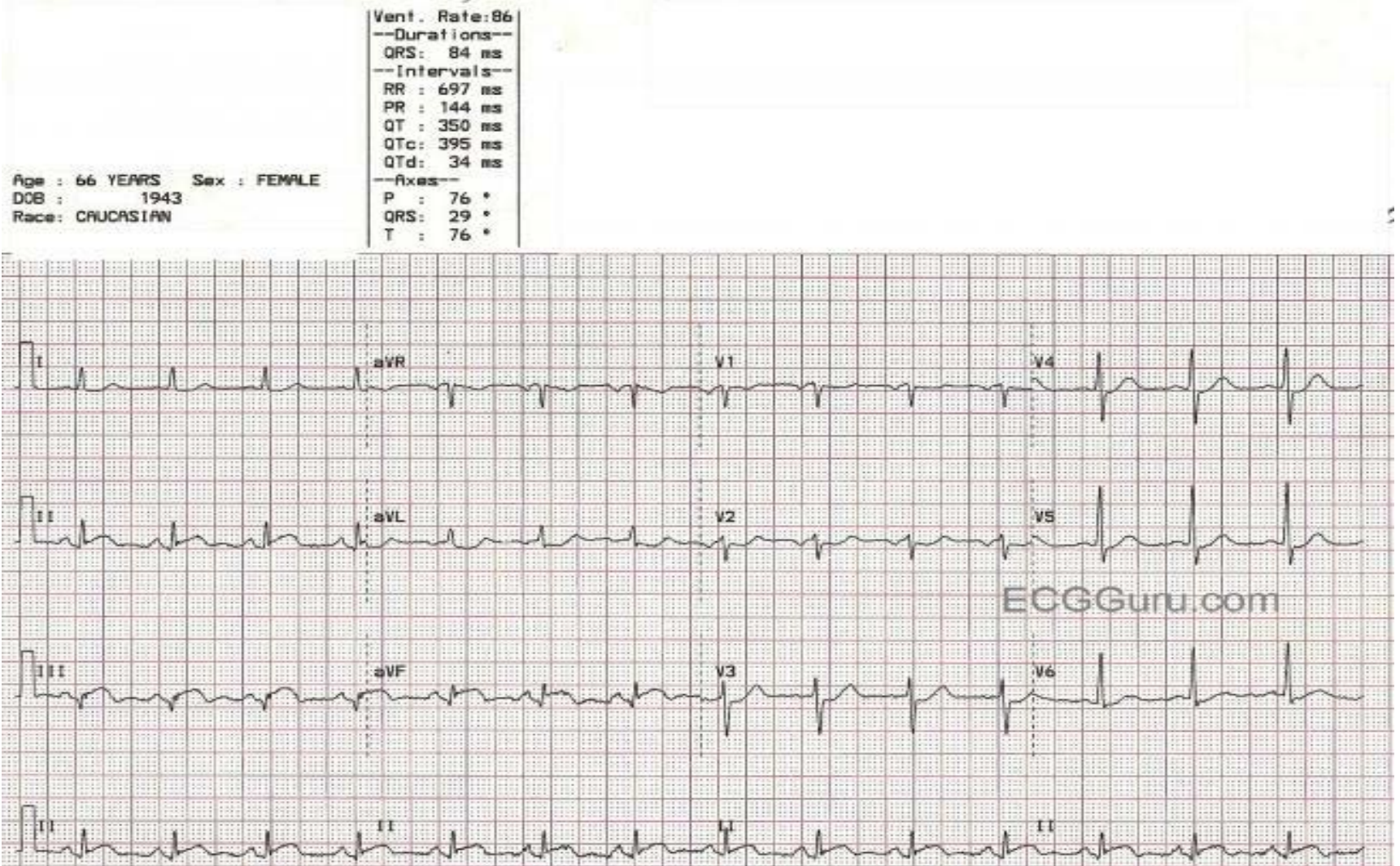
# INFERIOR WALL MI' S

- Usually due to blockage of the Right Coronary Artery (RCA)
- EKG: primary changes seen in Leads II, III and AVF with reciprocal changes in V5 and V6

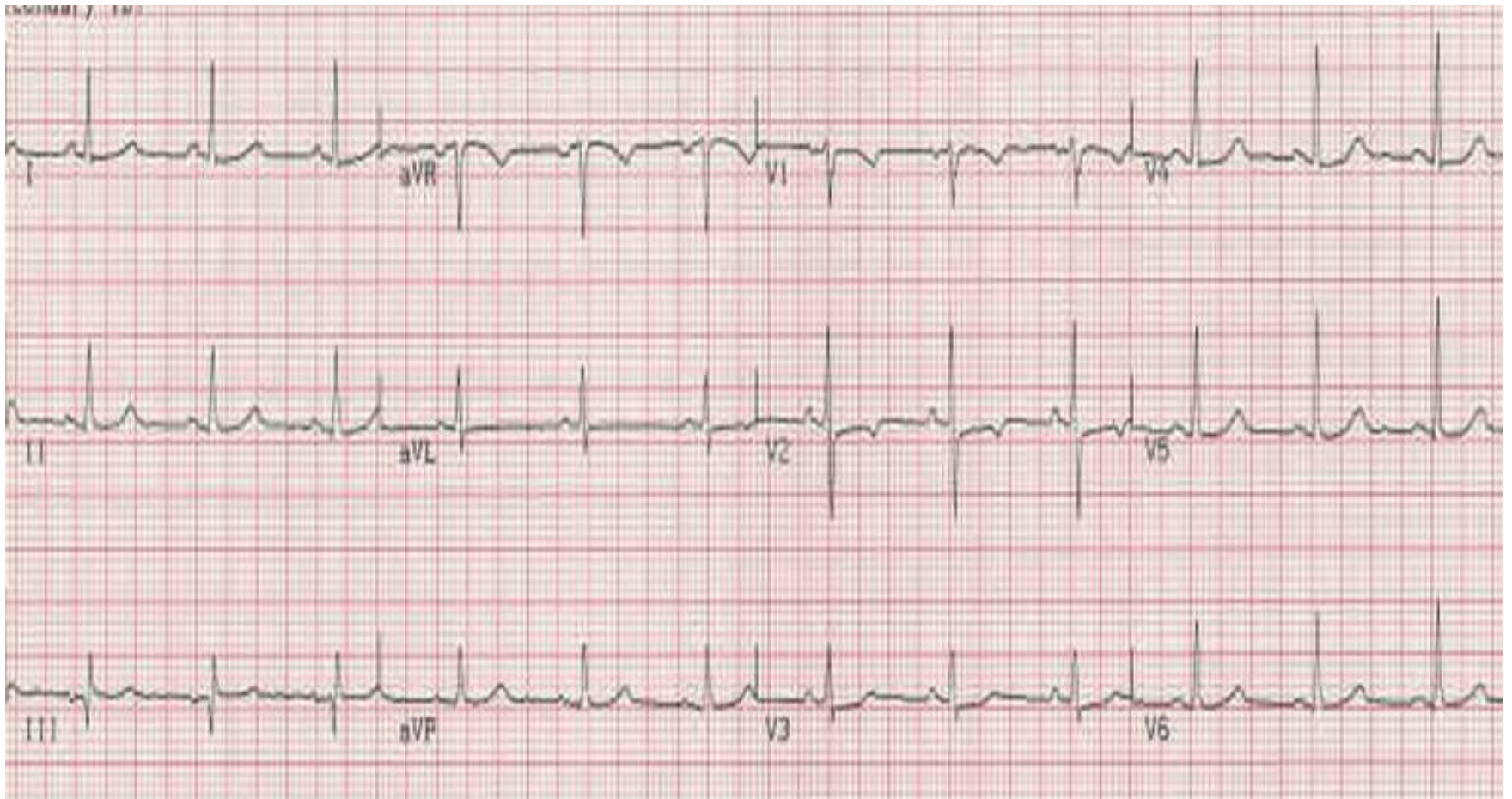
# Old Inferior Wall MI



# Acute Inferior Wall MI



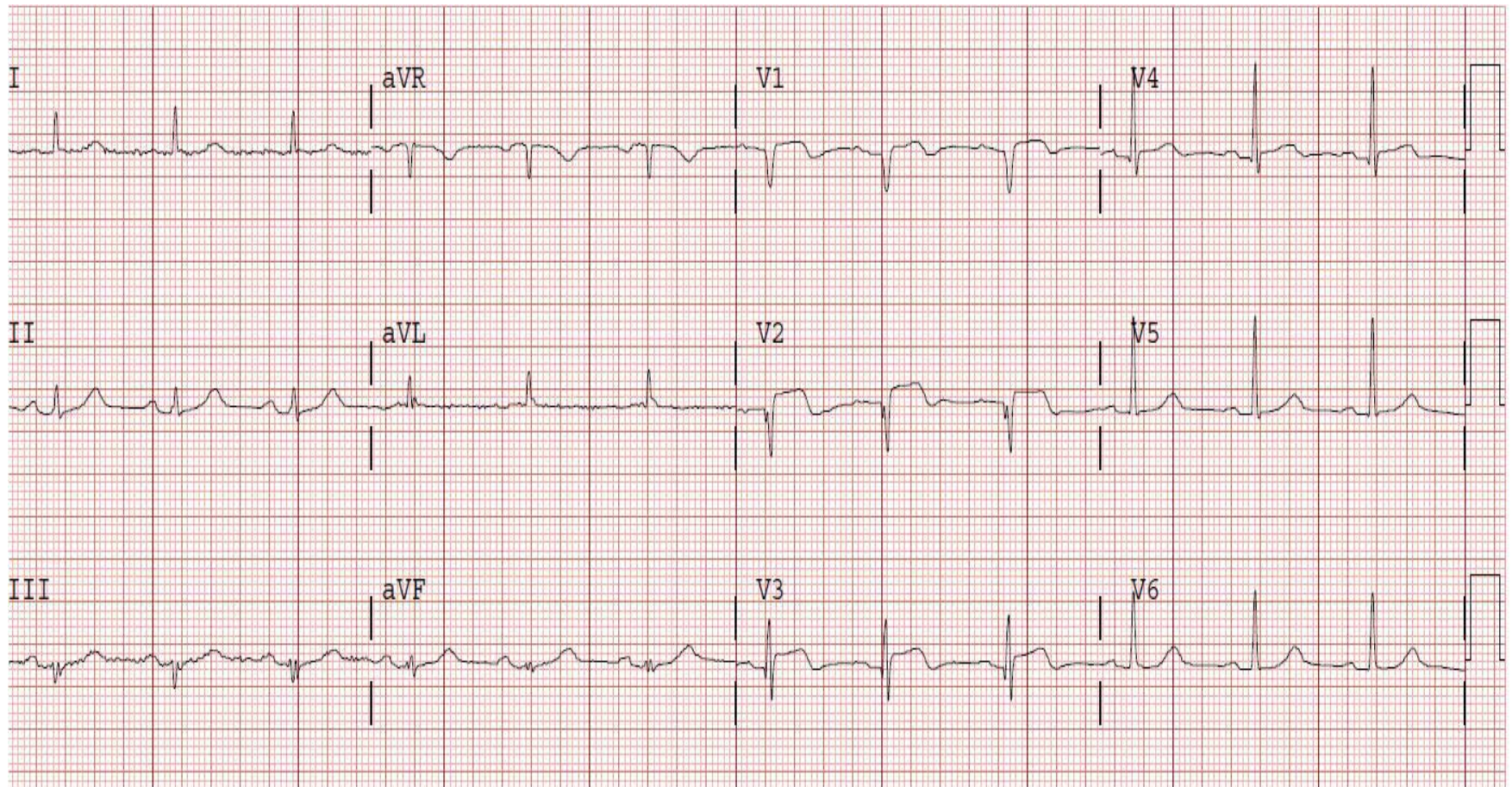
# Normal Sinus Rhythm: NOT an old inferior Wall MI



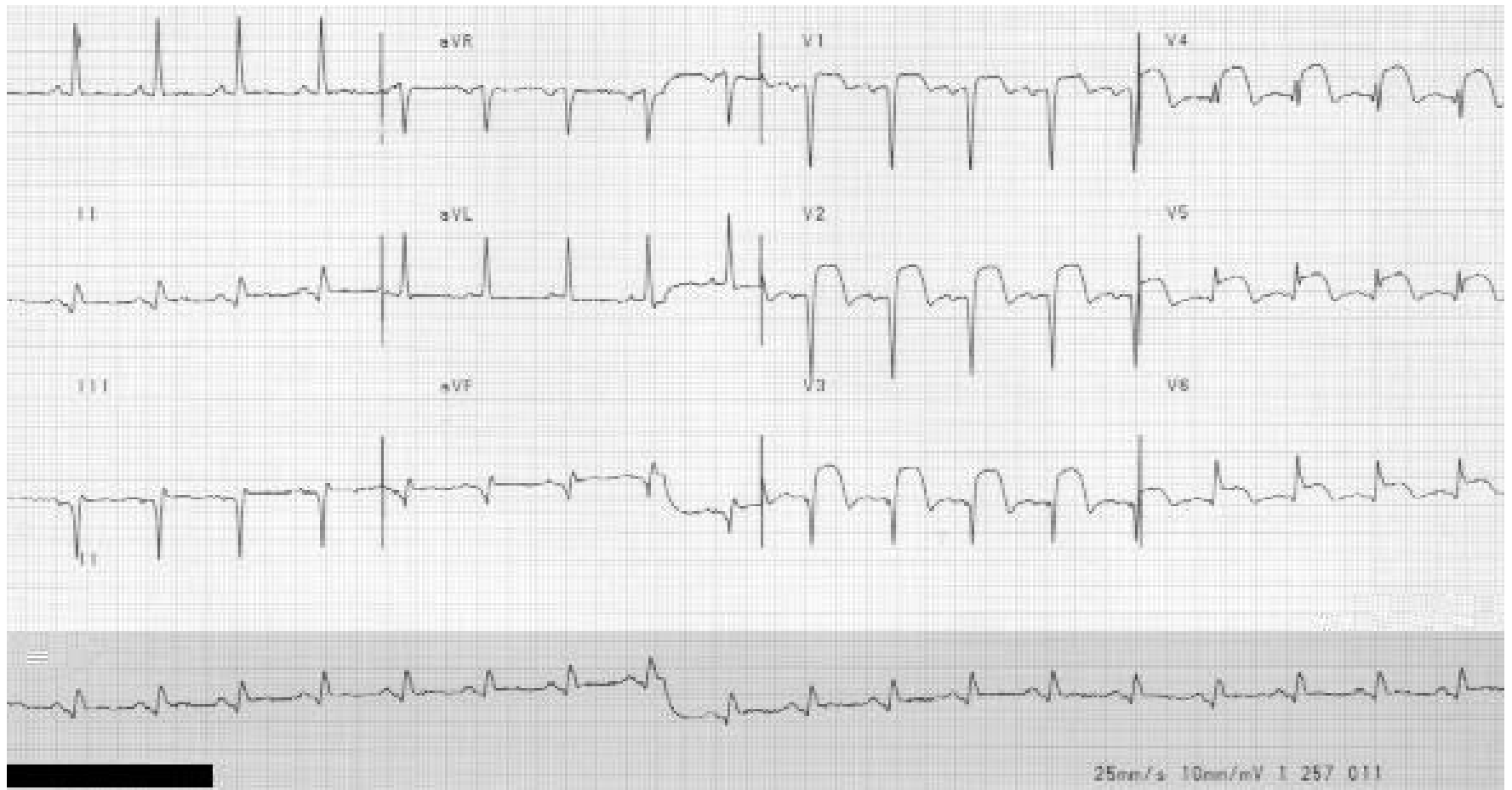
# Anterior Wall MI's

- **Can be due to blockage of Left Main Coronary Artery,**

# Anterior Wall MI

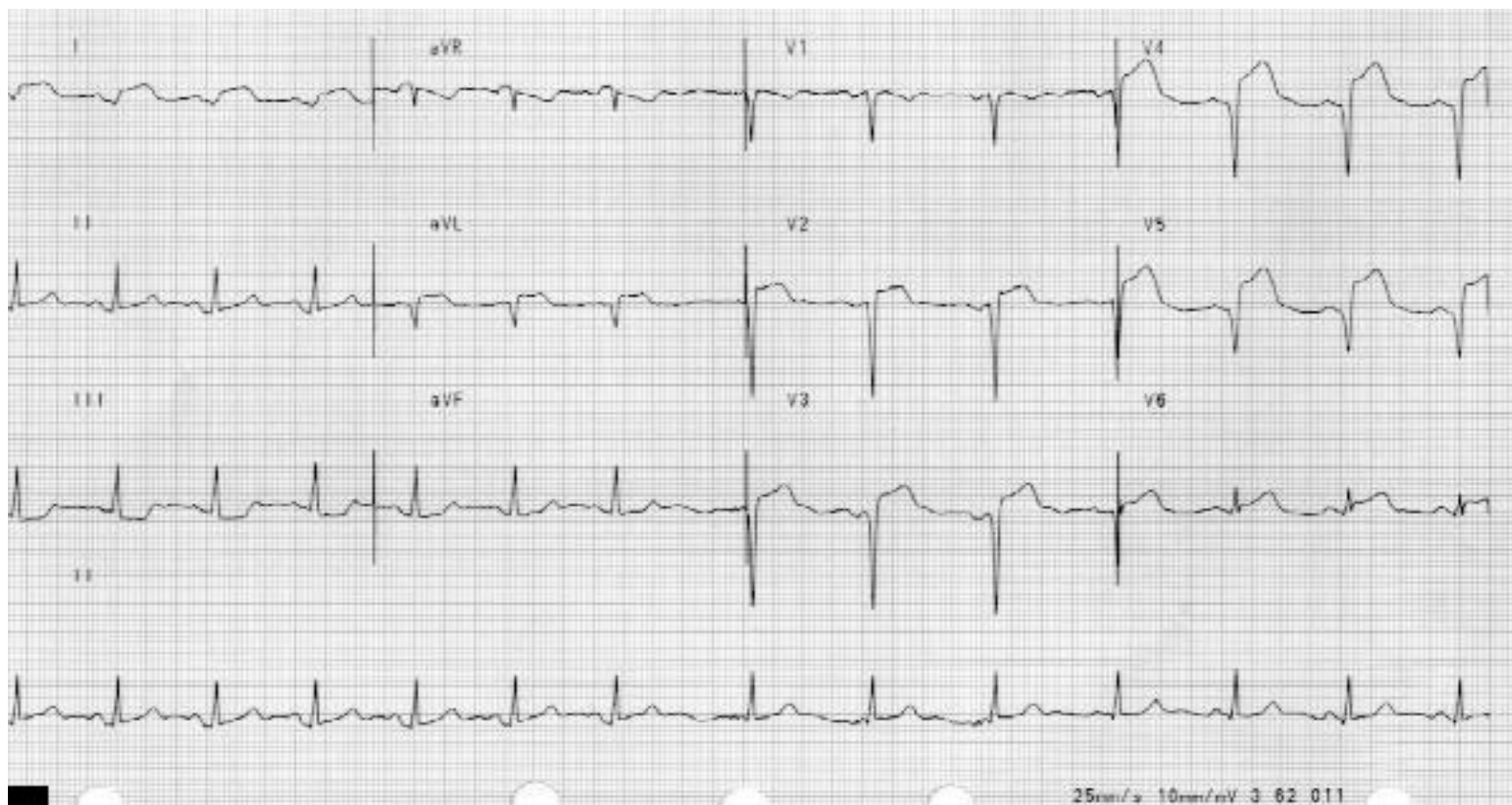


# Acute Anterolateral wall MI (old Inferior wall MI)

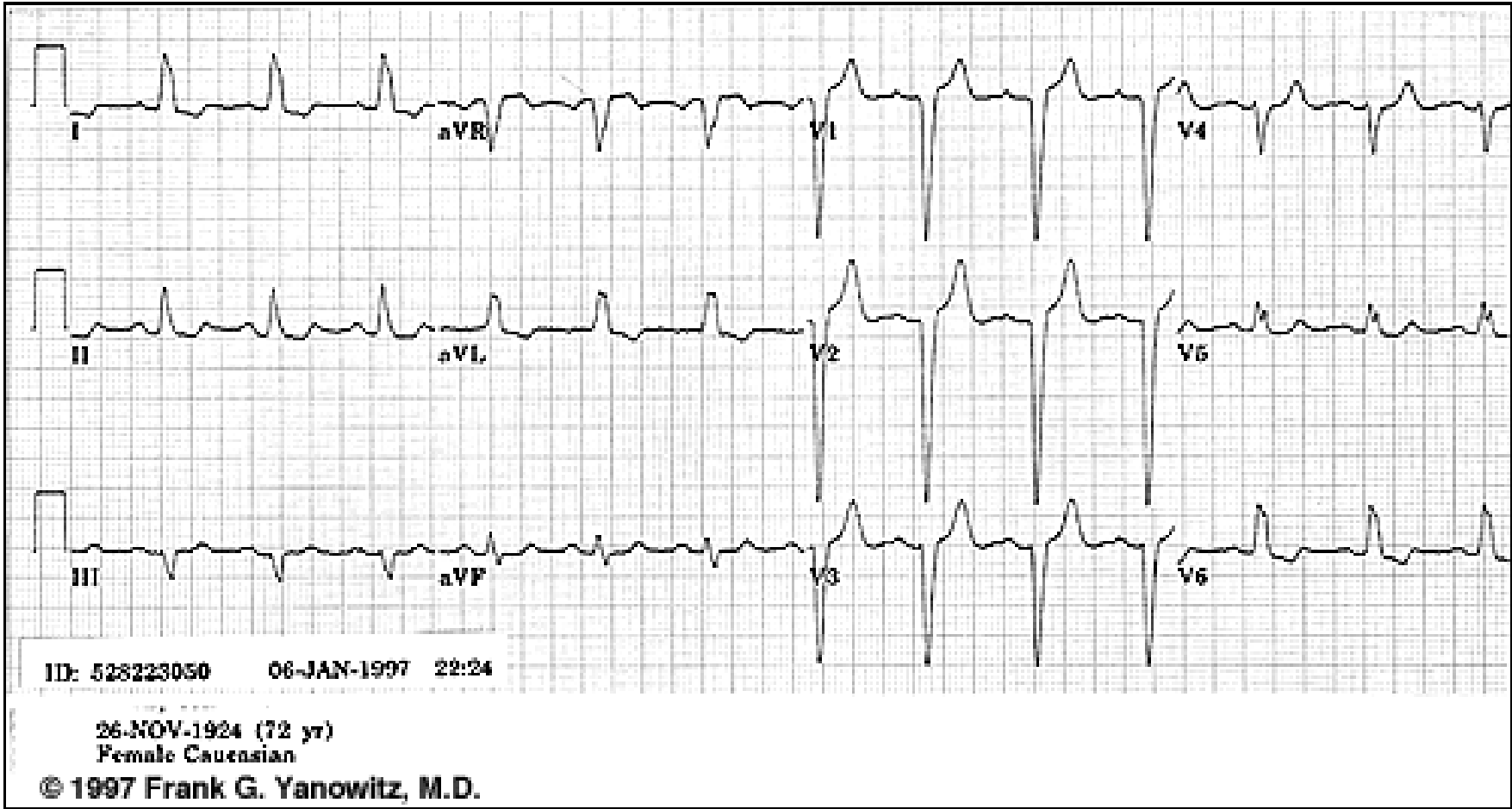




# Acute extensive anterior MI



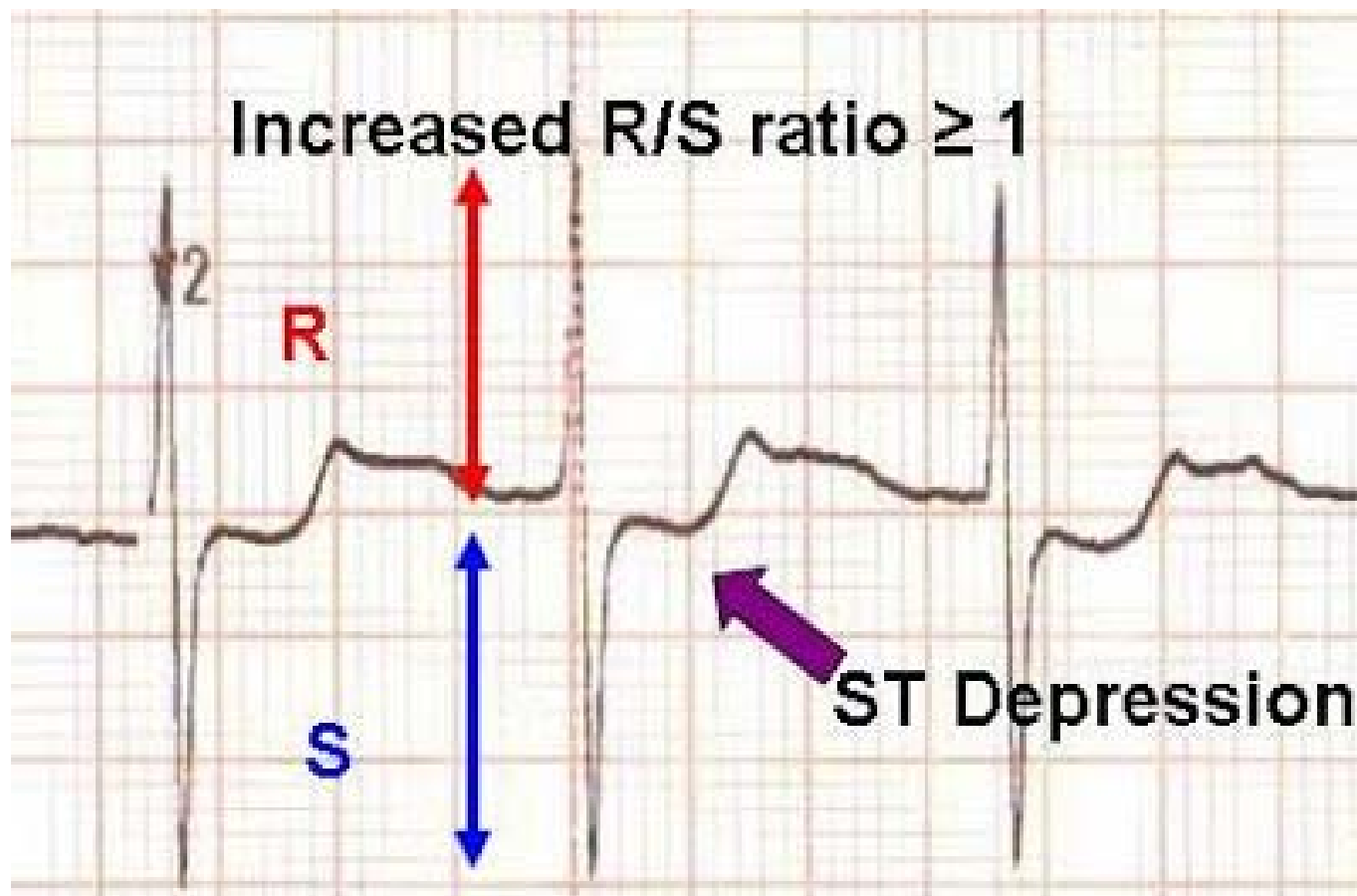
# Pseudoinfarct due to LBBB



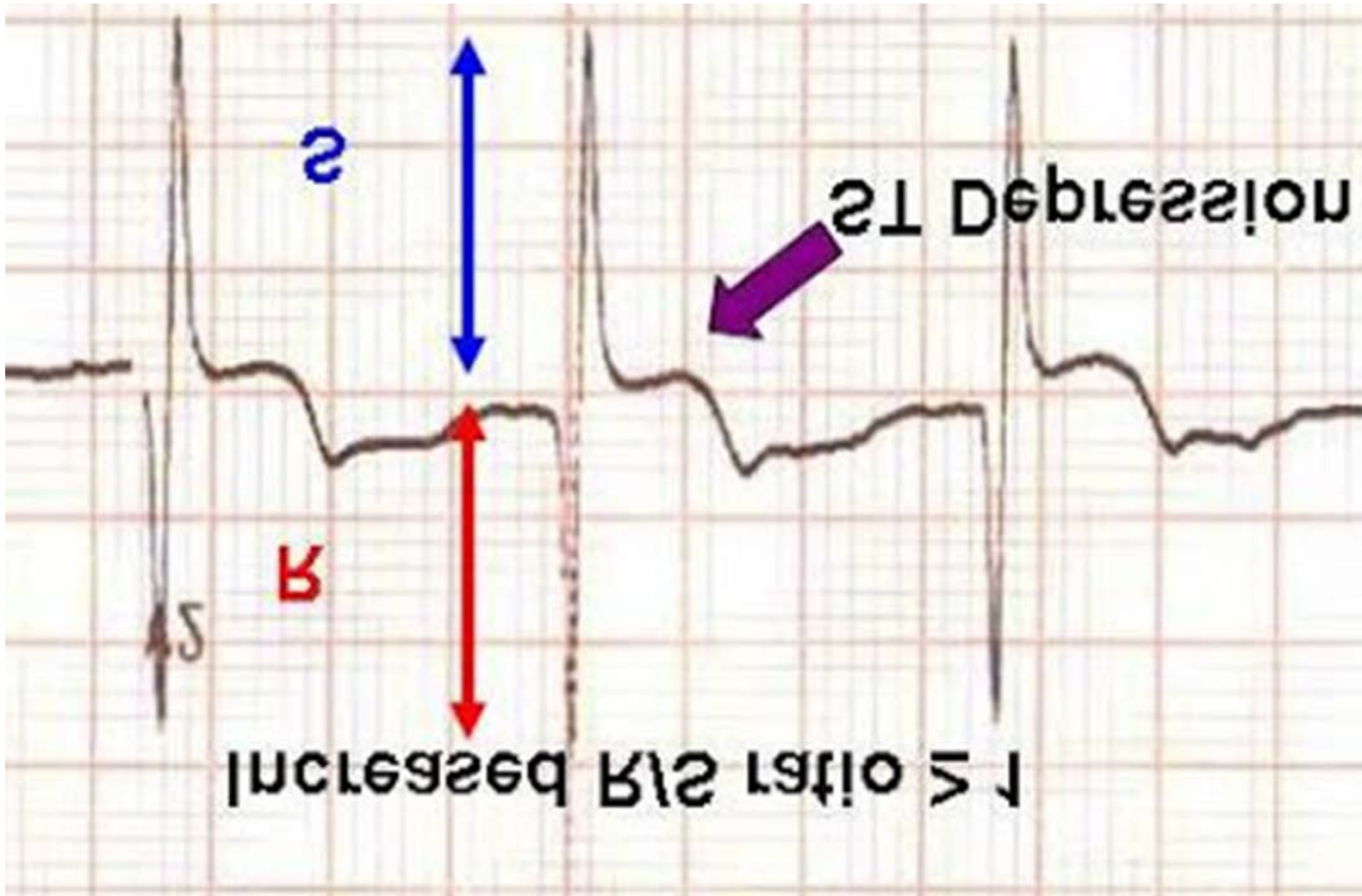
# What about the posterior wall infarction?

- There are no ECG leads overlaying the posterior wall
- Use reciprocal changes in right chest leads. Do additional right chest leads if suspected
- Difficult to diagnose
- Posterior MI not uncommonly associated with inferior or lateral wall MIs

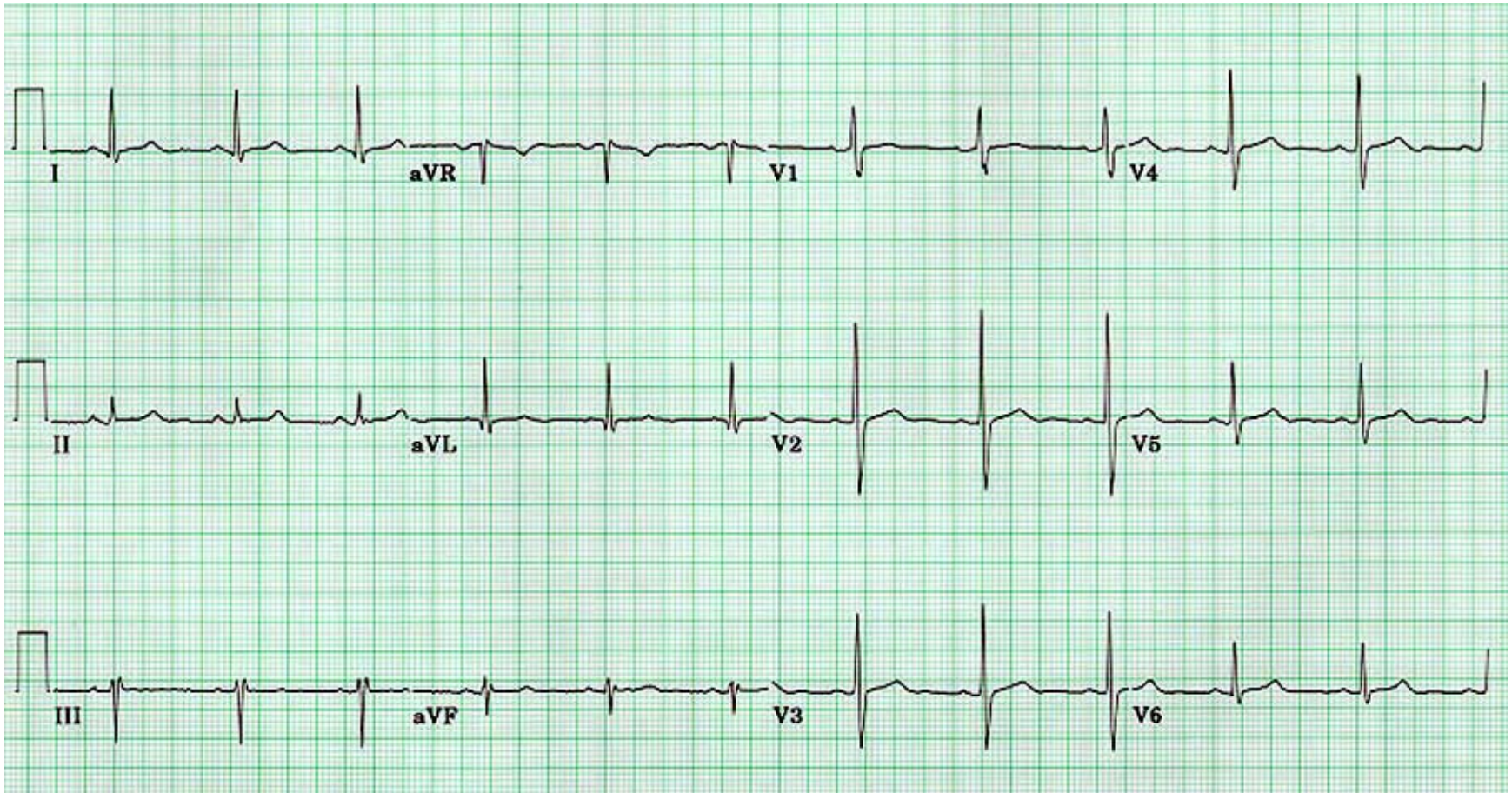
# Posterior Wall MI



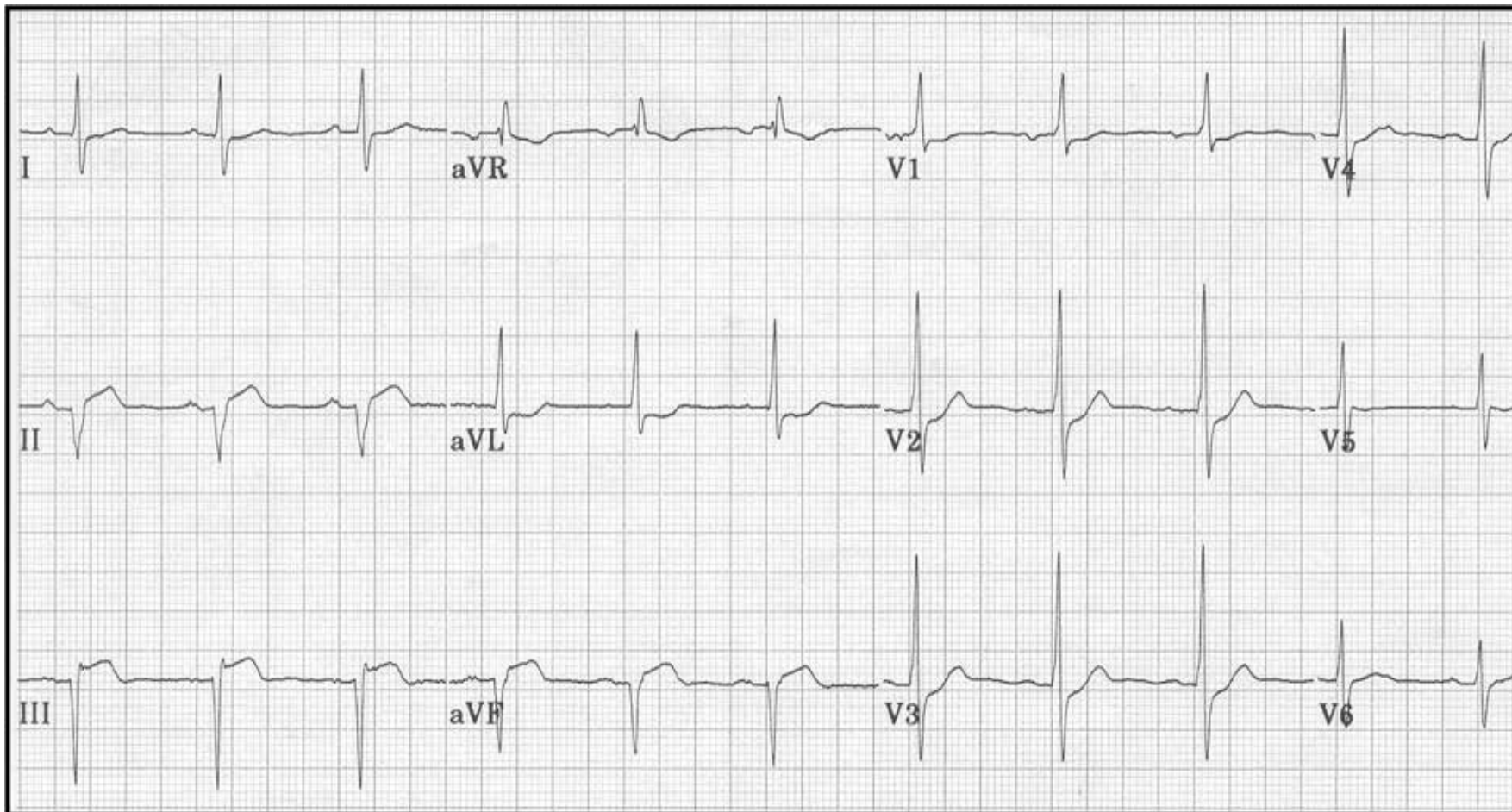
Trick: Look at V1 upside down



# Posterior Wall MI



Note T's upright in right chest leads. In RVH expect inverted T's here.

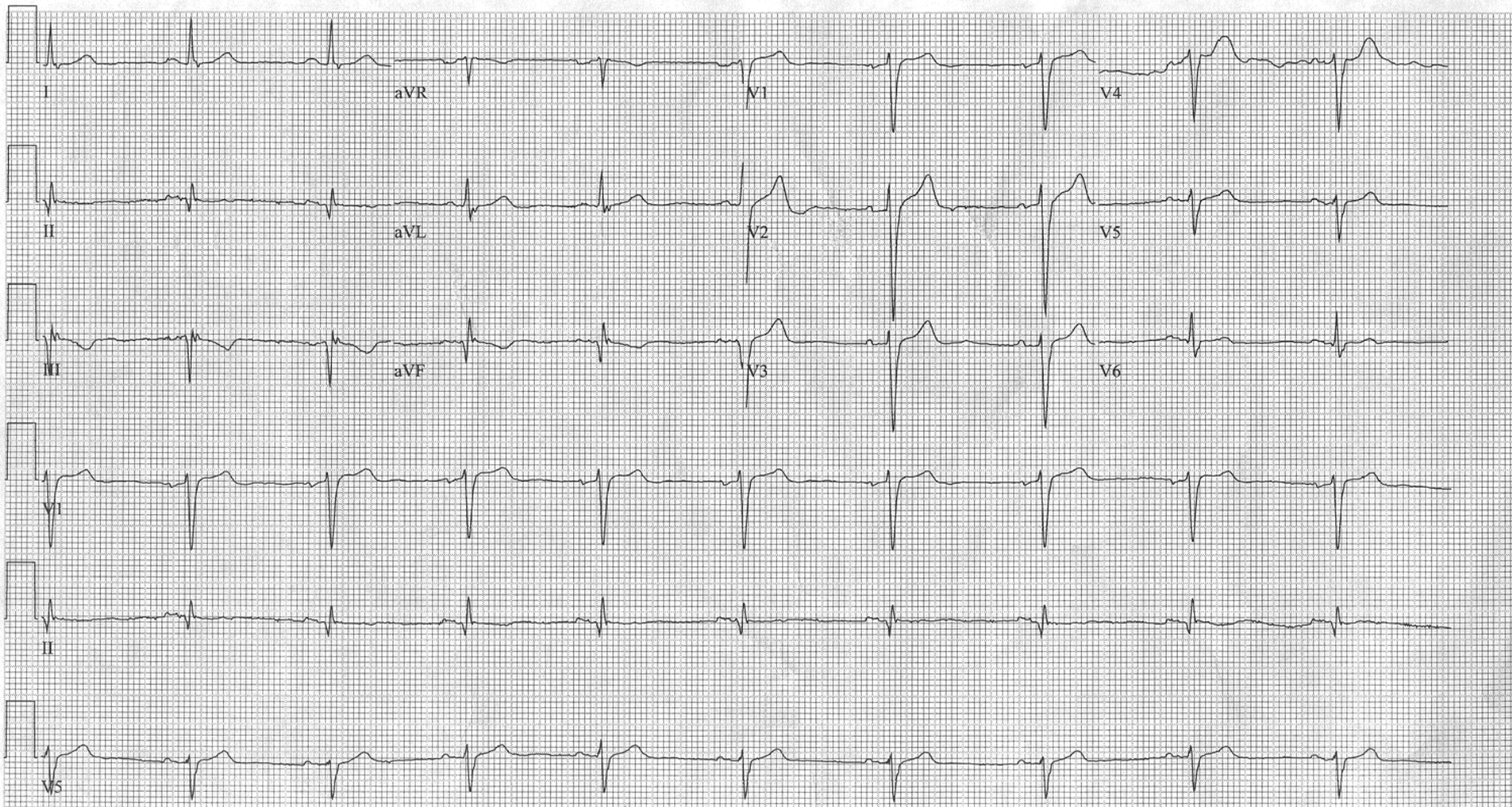


Break Time 😊



# Real Patient Case

A 66 Y/O diabetic with known CAD has a pre-op EKG for a laparoscopic cholecystectomy

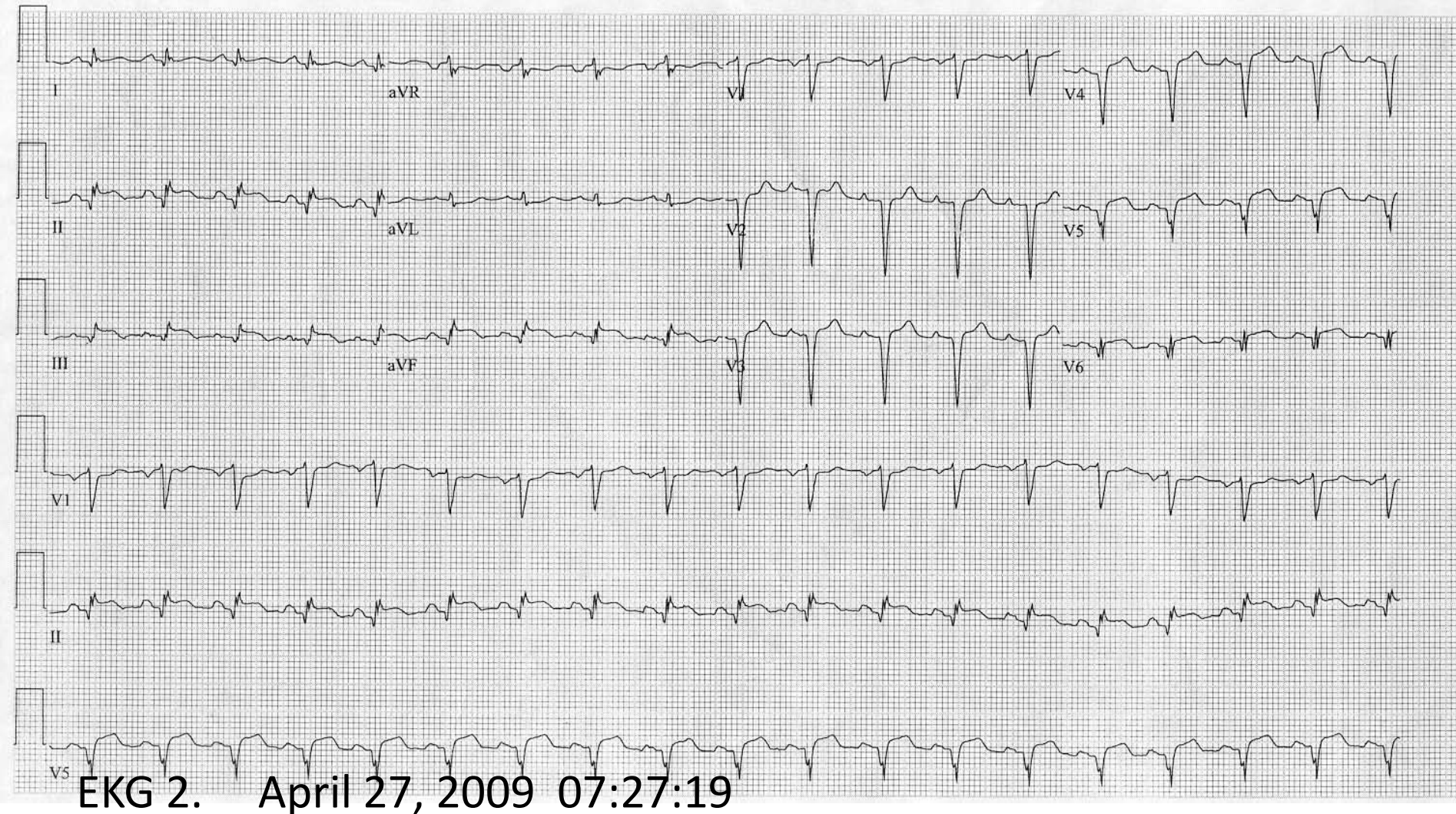


EKG 1. March 27, 2009

**DH is a 66 Y/O diabetic with known CAD** who was “cleared” for a laparoscopic cholecystectomy after an examination and EKG #1. His Plavix & ASA were stopped pre-op. Surgery was uncomplicated.

In the recovery room anesthesia saw him for some PVC's on the monitor. He was then allowed to return to his room. That evening he complained of pain in his left biceps. The nurse gave him Tylenol, which had been ordered for pain, and he did not require another dose.

EKG ordered post op day 1 by surgical PA-C.  
Patient had C/O left biceps pain & had tachycardia.



## Day 1 post op:

On morning rounds a surgical PA noted tachycardia and the previous biceps pain. She obtained EKG 2 and called her supervisor who immediately had a cardiologist review the EKG by fax. He told her the changes showed an extensive acute MI, the kind often associated with complications such as papillary muscle rupture, a ruptured septum or heart, heart failure and/or shock. He urged immediate interventional consultation for treatment and an emergency cardiac catheterization.

## Day 1 post op:

A formal emergency cardiology consult followed, revealing stable hemodynamics but a new systolic murmur. Meanwhile the supervising PA checked the previous hospital notes. She noted that the patient's Plavix & ASA had been stopped pre-op. Reviewed rhythm strips from the recovery room revealed the noted PVCs but also some ST elevation.

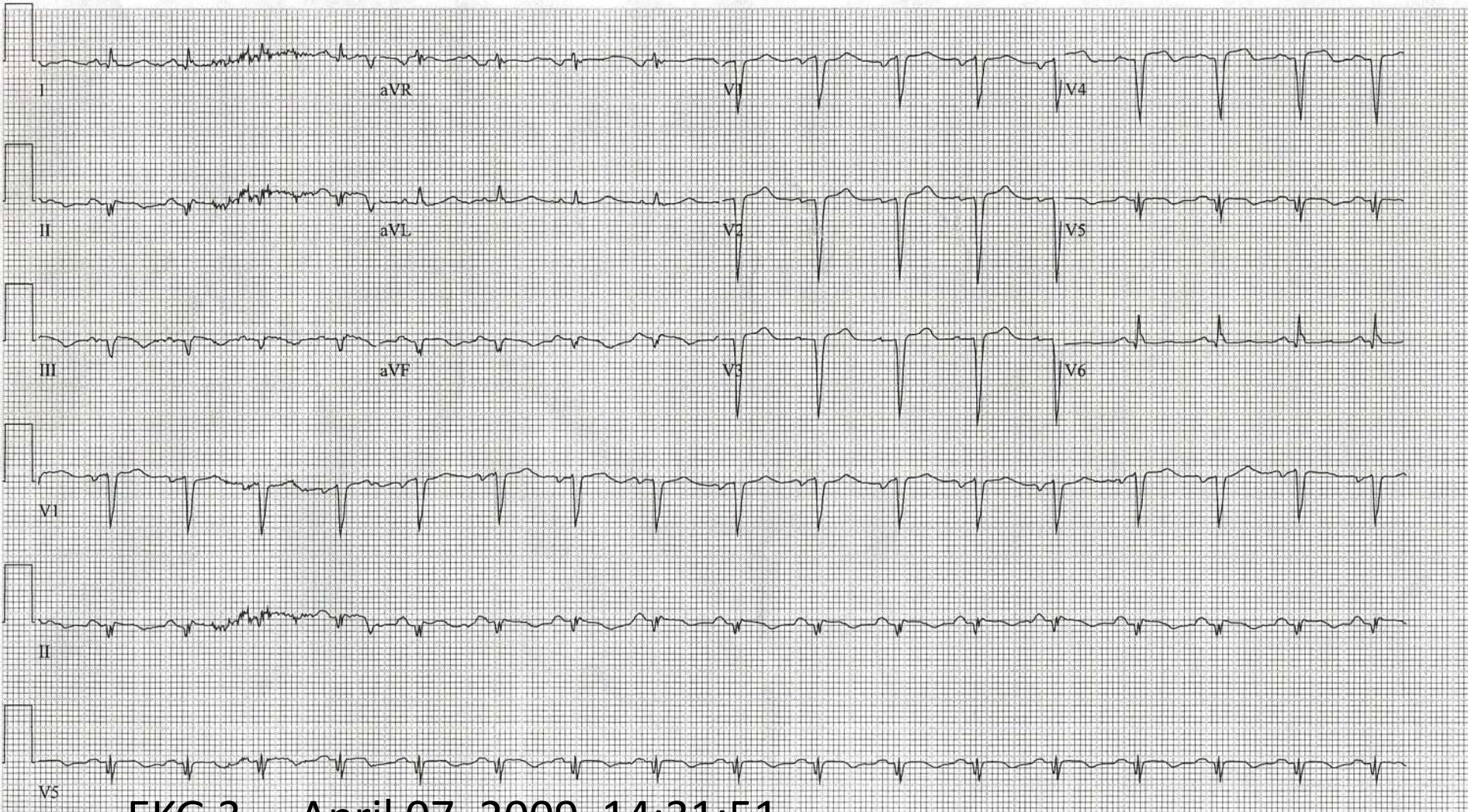
Cardiac markers and an emergency cardiac catheterization were ordered.

## Day 1 post op:

The cardiac catheterization revealed total occlusions of both the LAD and RCA and a VSD consistent with a ruptured septum. Cardiac markers had come back positive. Post cath EKG #3 was obtained.

He underwent emergency CABG and repair of the VSD. His post op EF was 35%. Several weeks later his VSD repair failed and repeat surgery was needed. He tolerated this well.

# Follow-up EKG post op day 1



EKG 3. April 07, 2009 14:31:51

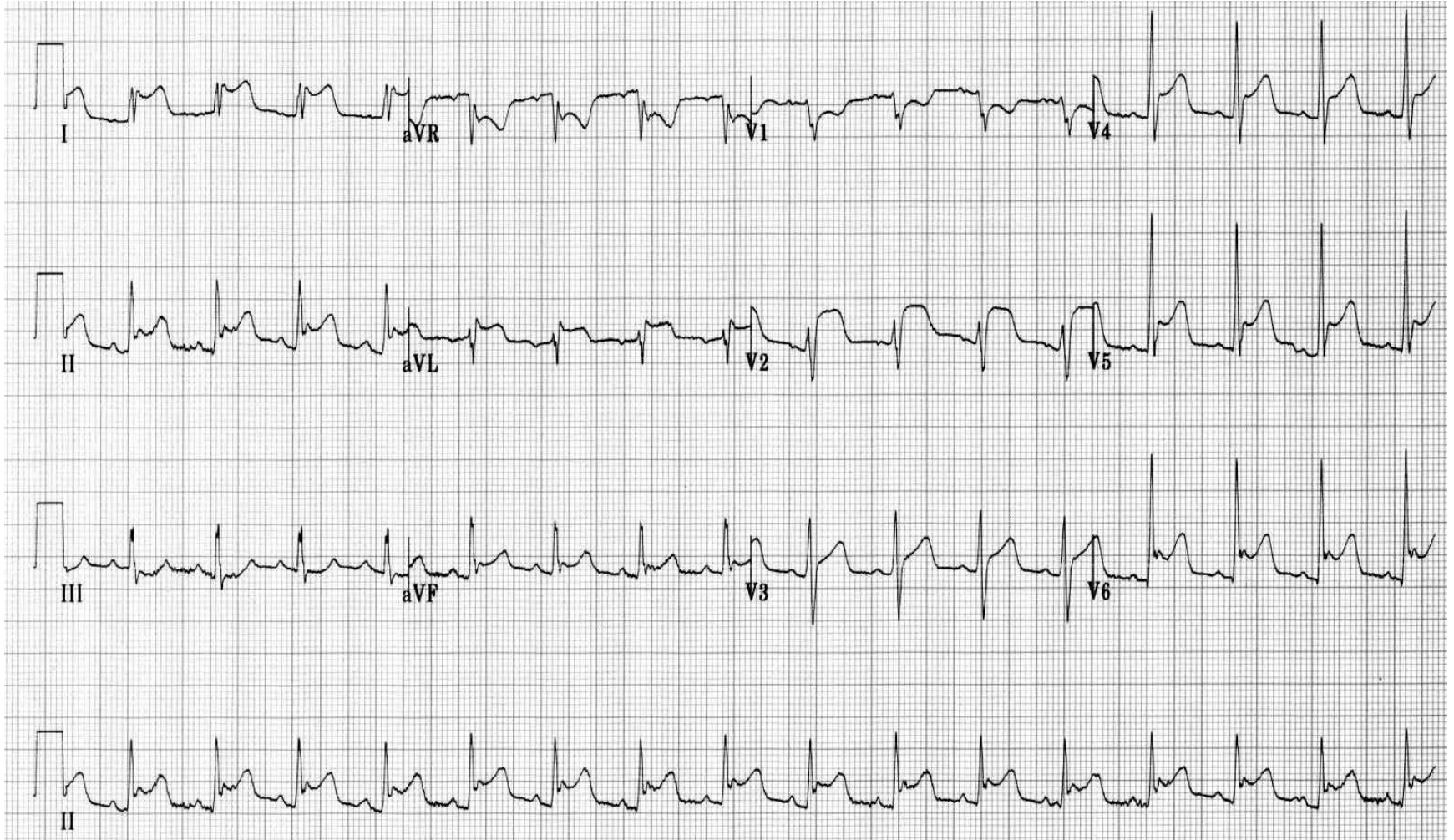


THOUGHTS????

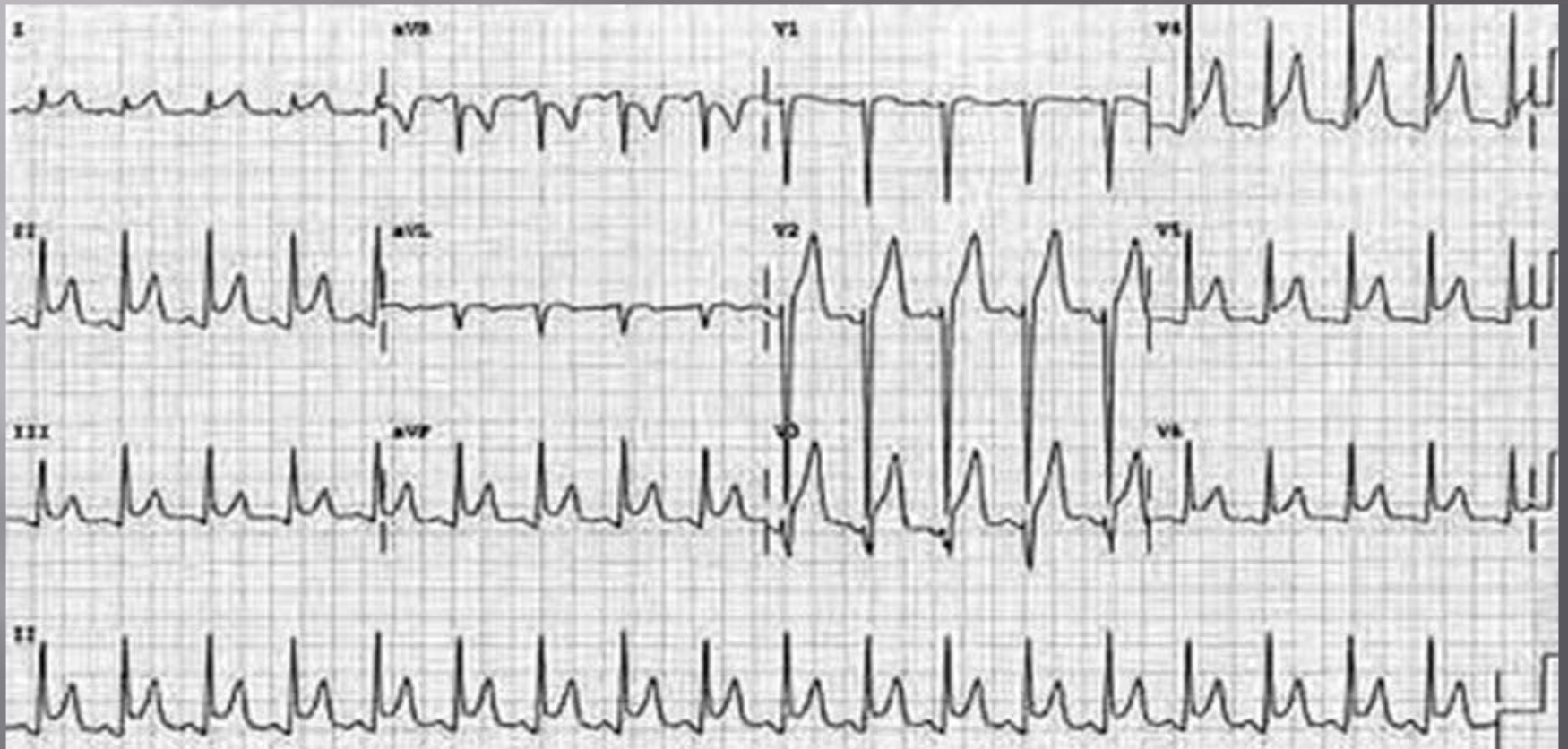
# Acute Pericarditis

- EKG can easily be confused with an evolving MI
- The ST segment and T wave changes in pericarditis tend to be diffuse (although not always) and involve far more leads than the localized effect of infarction
- No reciprocal changes
- Q wave formation does not occur
- Elevated ST returns to baseline before the T wave inverts

# Acute Pericarditis



# Acute Pericarditis



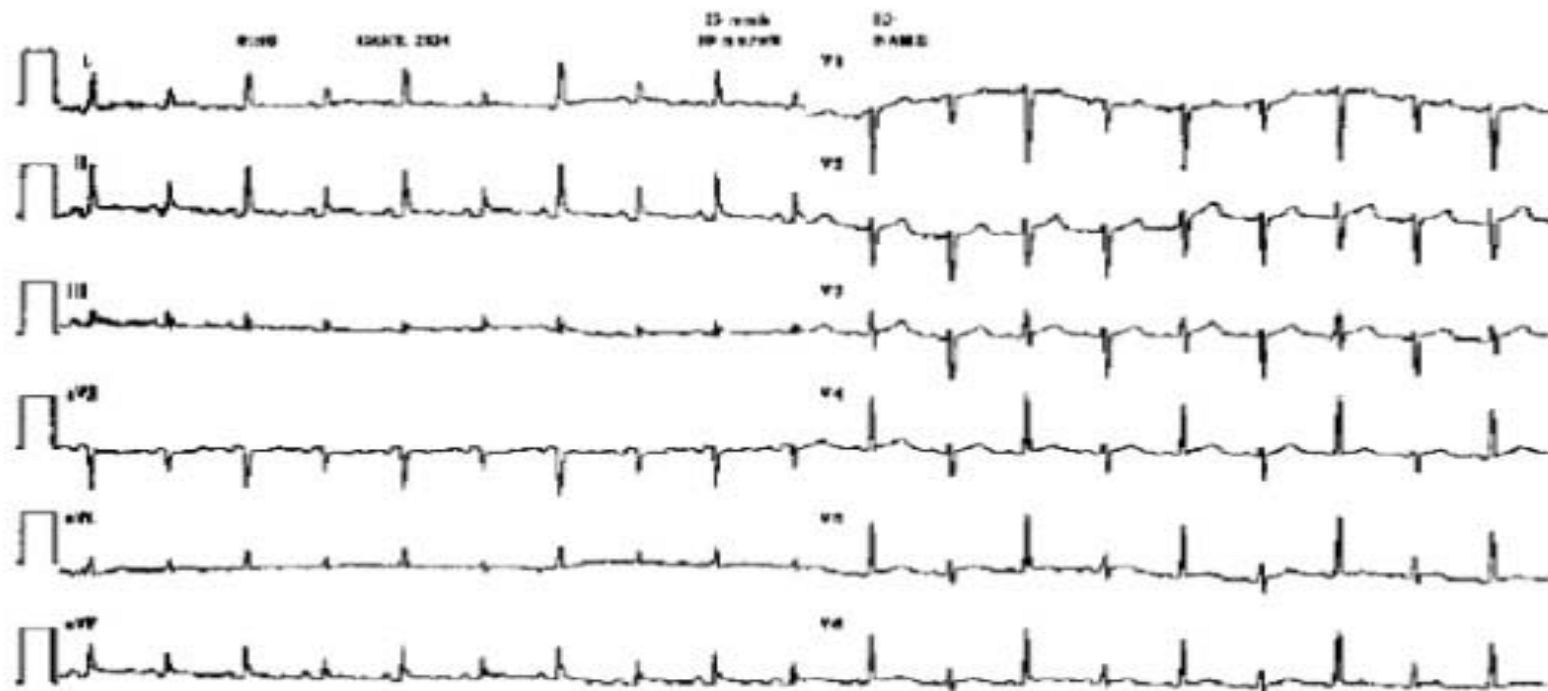
**Pericarditis**

# Cardiac Tamponade

- Low Voltage of the QRS complexes due to pressure from fluid surrounding the heart
- **Electrical Alternans:** Beat to beat shift in QRS axis
- Electrical Alternans with sinus tachycardia is virtually diagnostic of cardiac tamponade

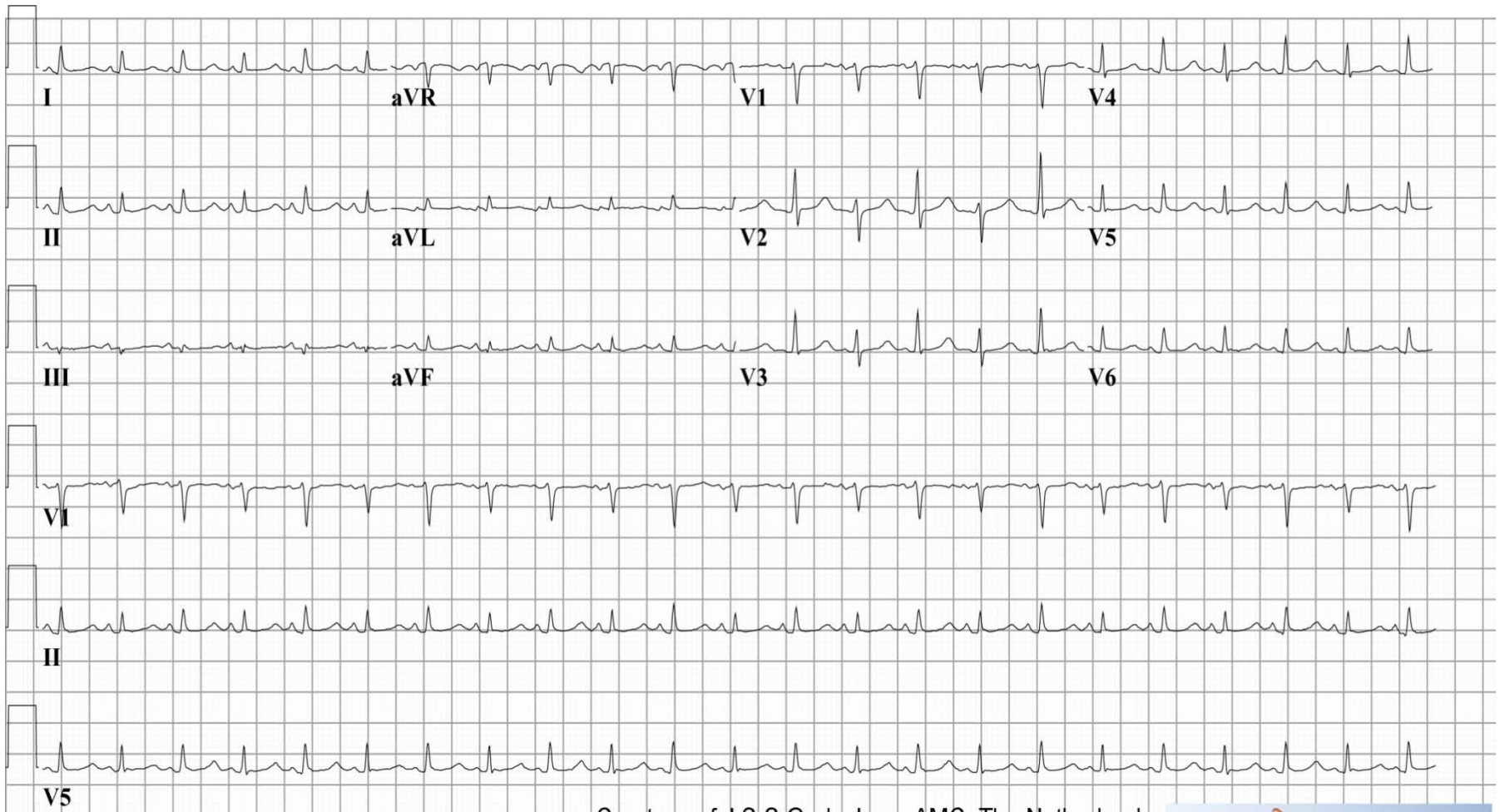


Electrical Alternans  
showing alternating QRS complex axis

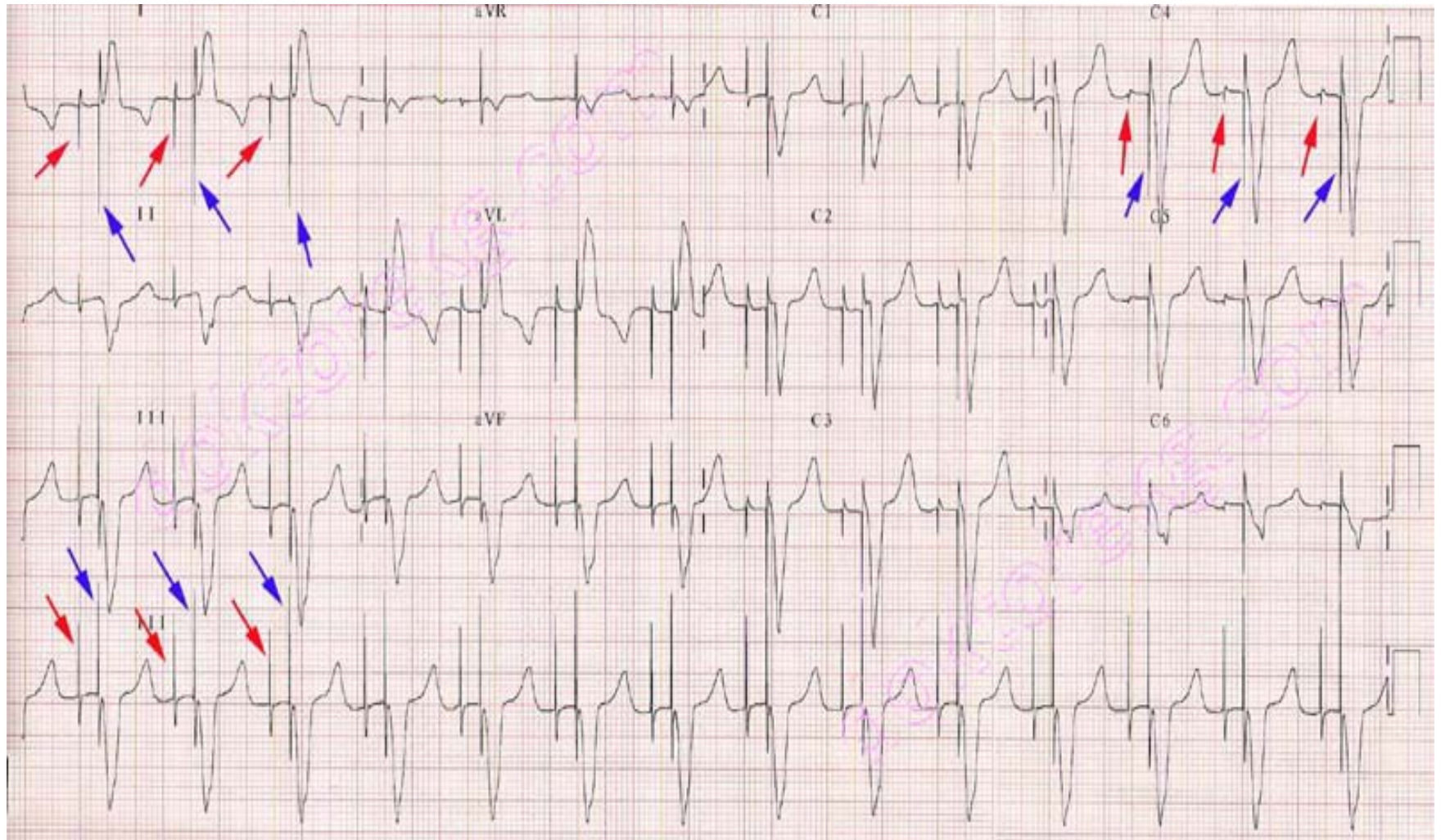


# Cardiac Tamponade

# Cardiac Tamponade



# Pacemaker

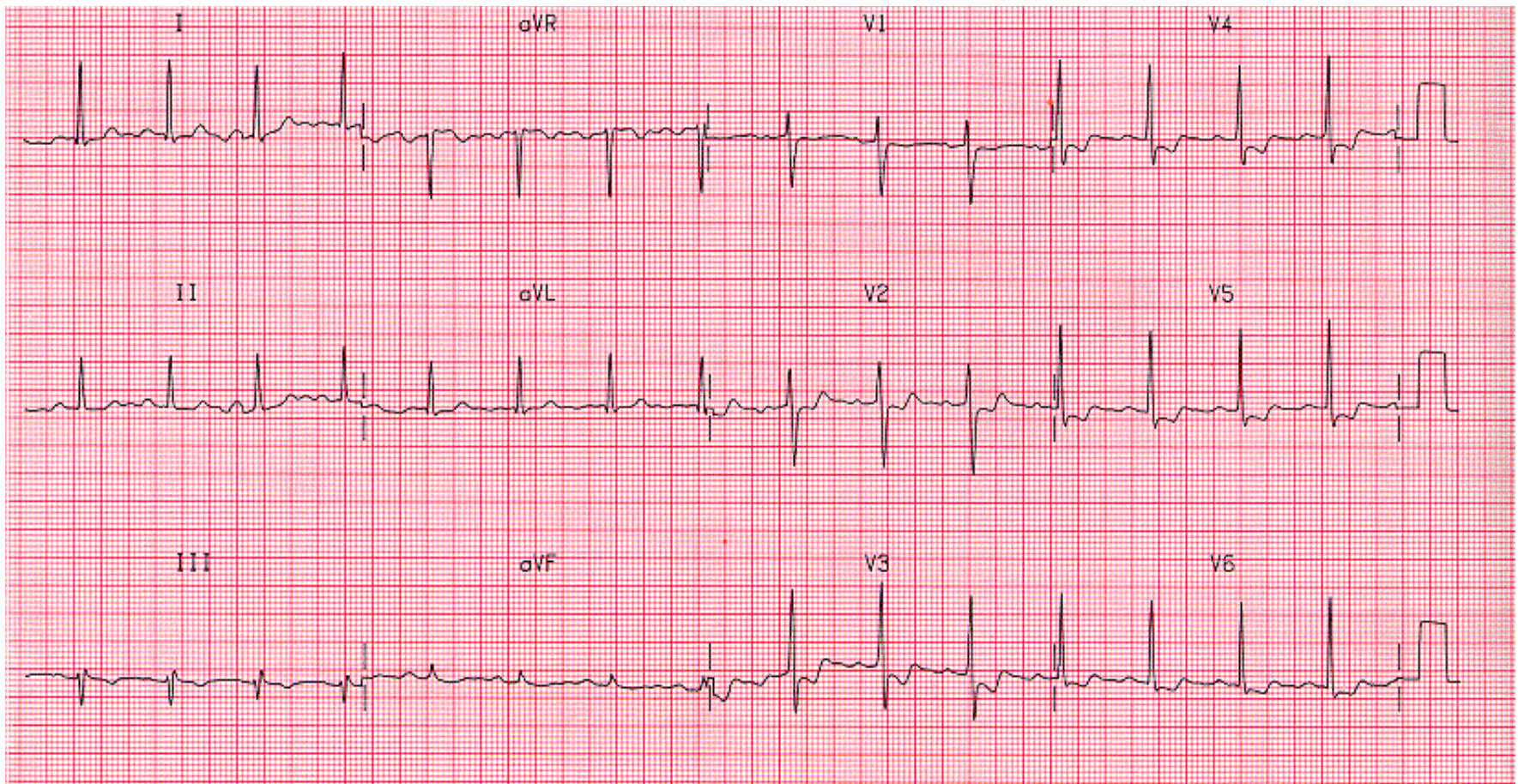




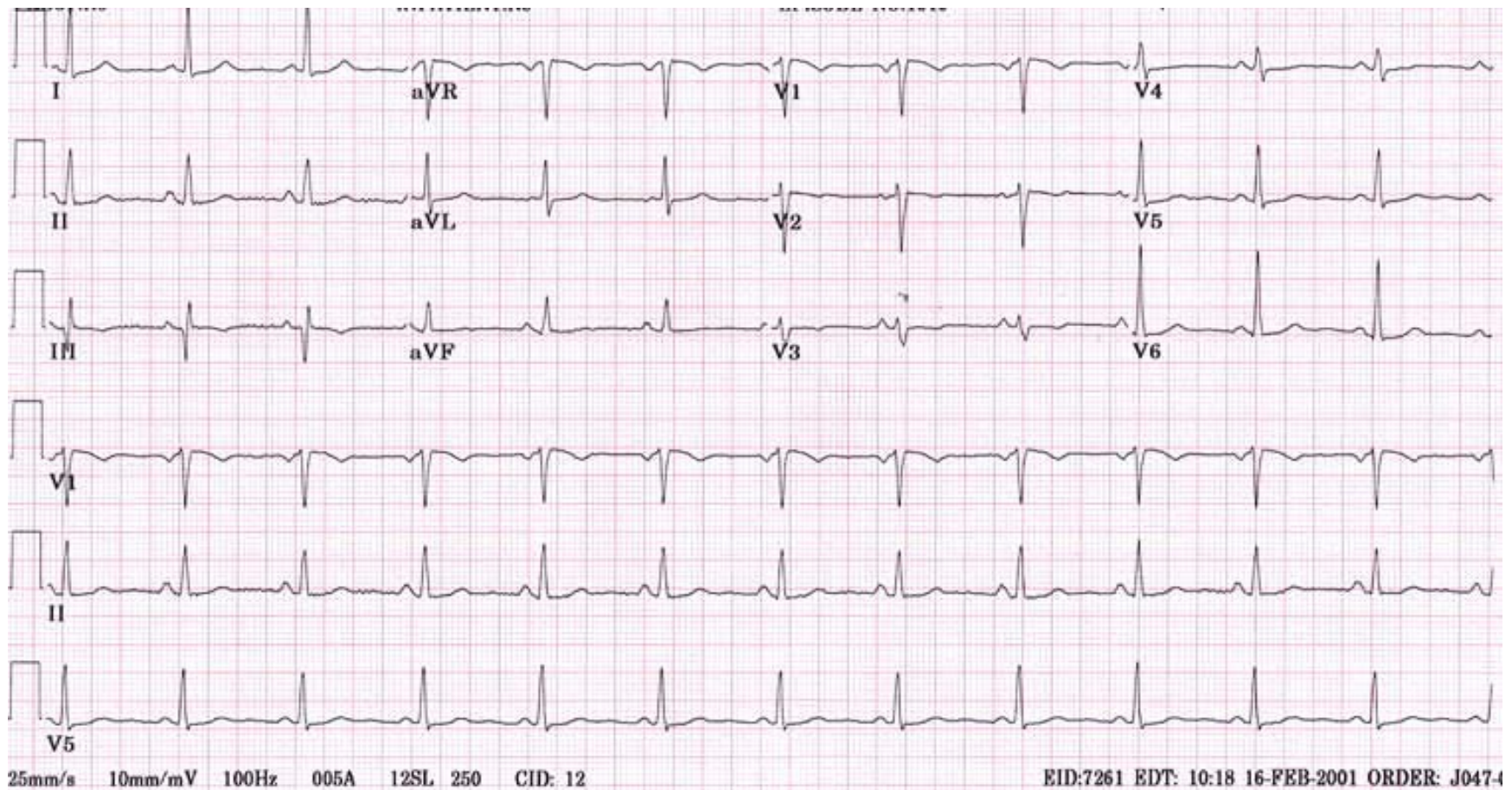


LET' s PRACTICE

# Anterolateral Wall ischemia

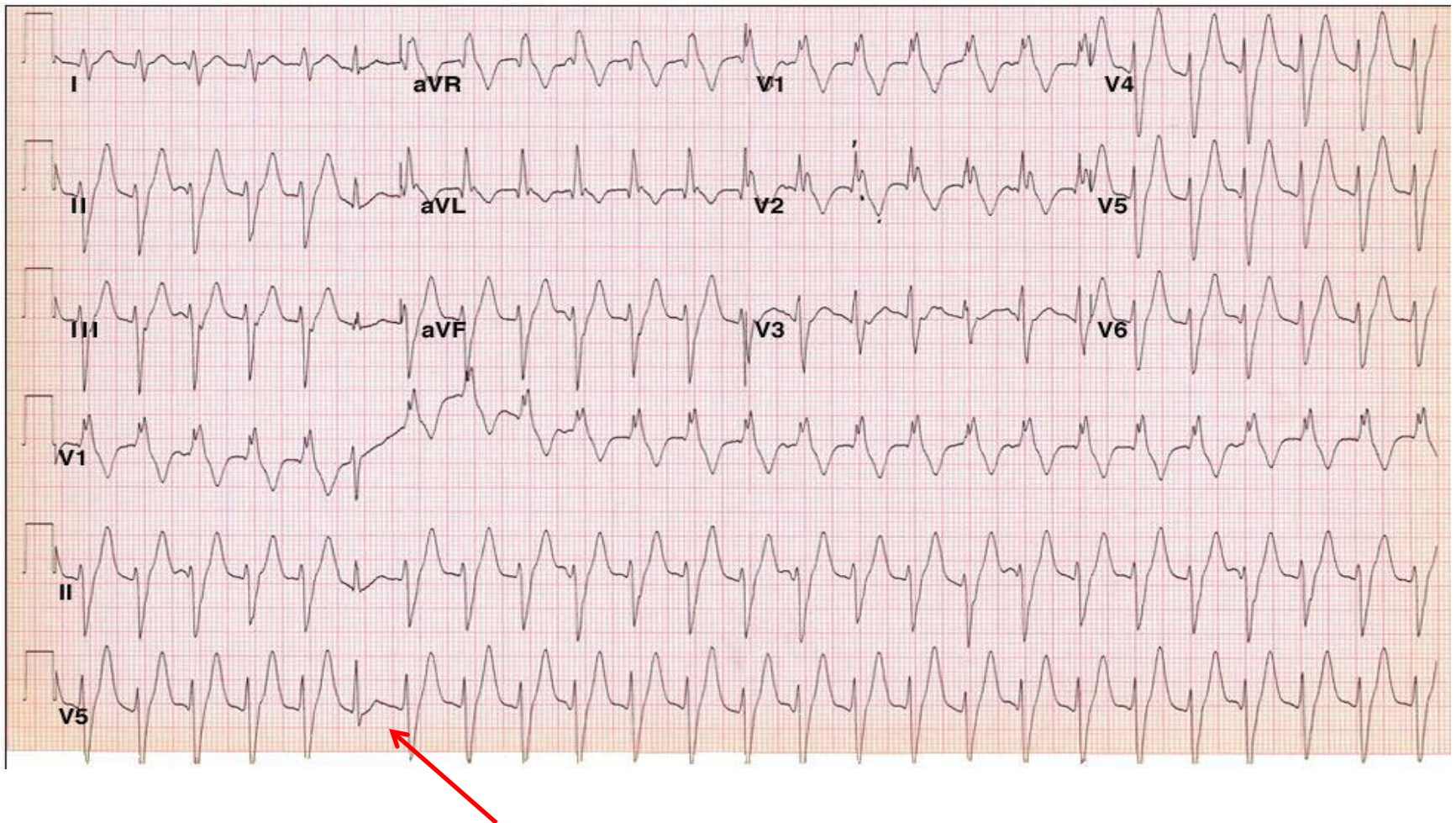


# Normal Sinus Rhythm



# Ventricular Tachycardia

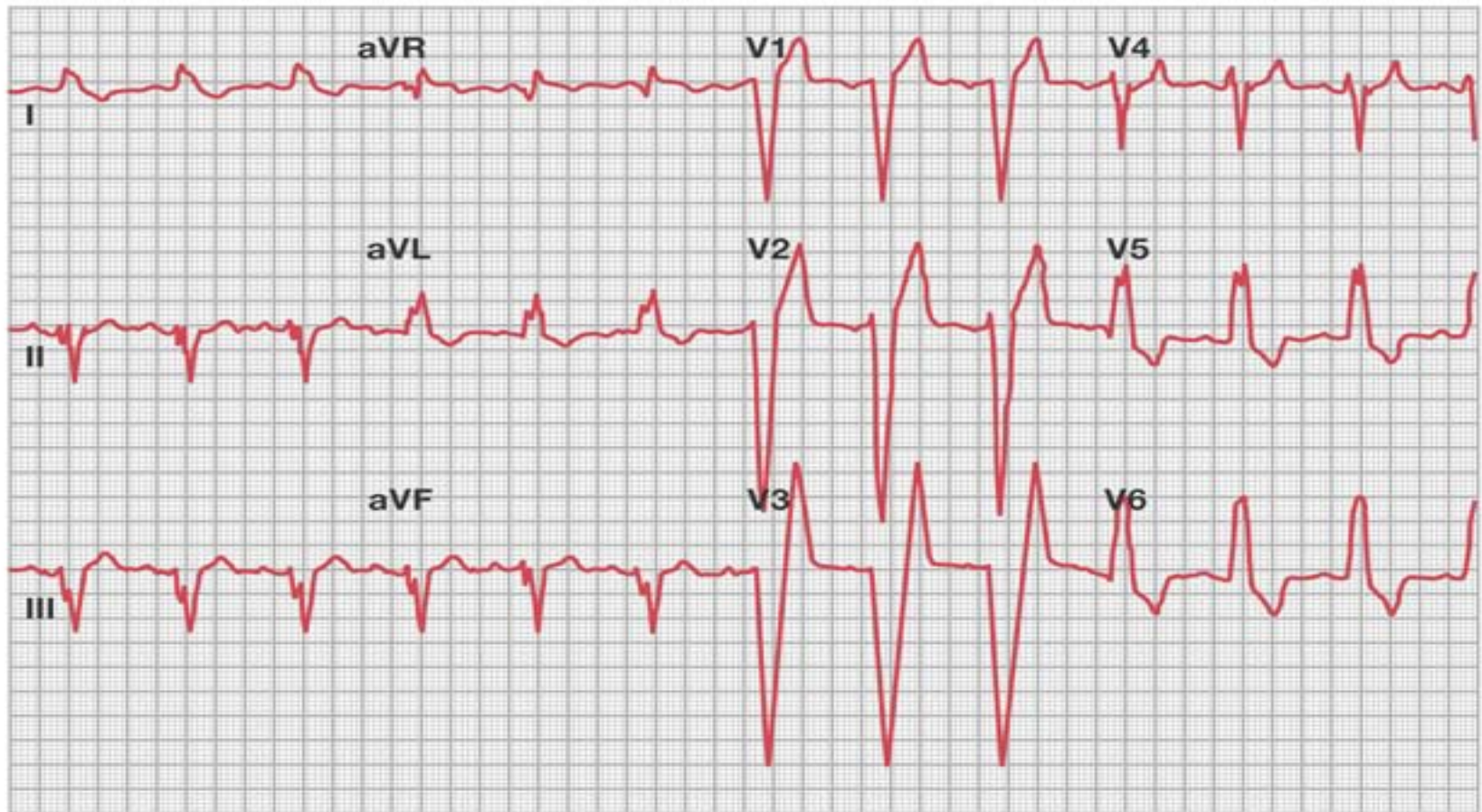
Could suspect hyperkalemia with the tall T waves but the capture beat (6<sup>th</sup> beat) doesn't show a tall T wave.



Sinus Bradycardia with notched wide P wave consistent with intra-atrial conduction defect.

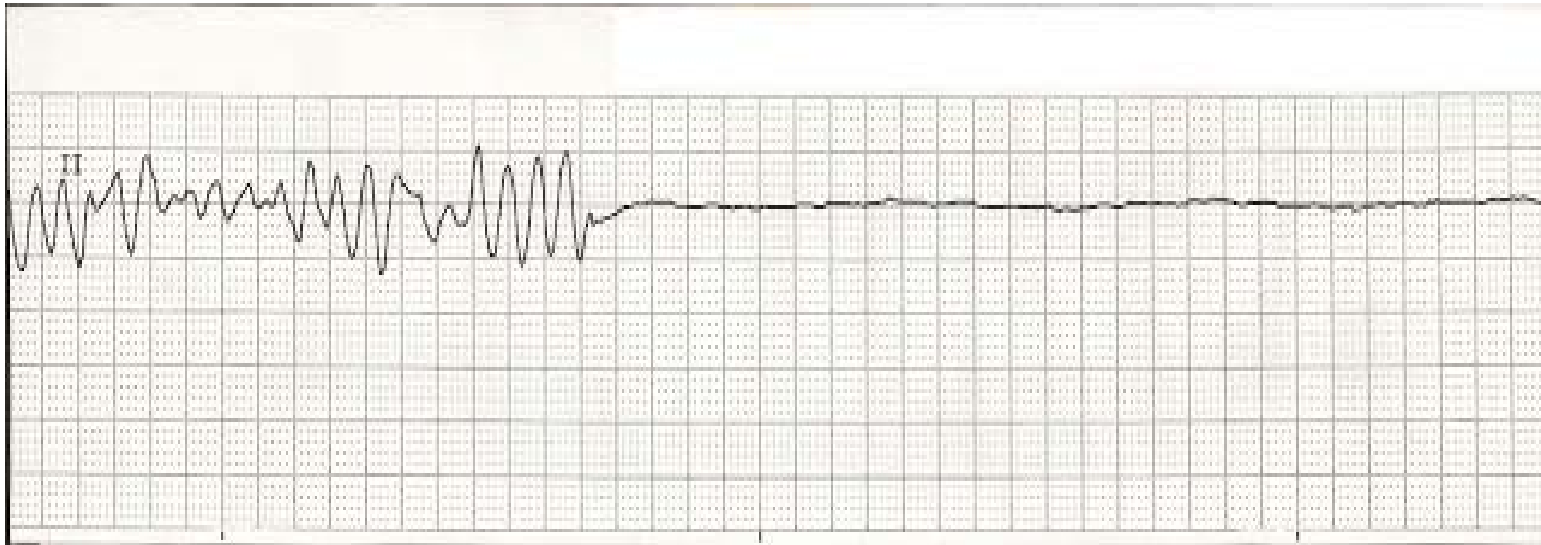


# Left Bundle Branch Block



# Ventricular Fibrillation to Asystole

- **But look at patient to be sure this isn't artifact**



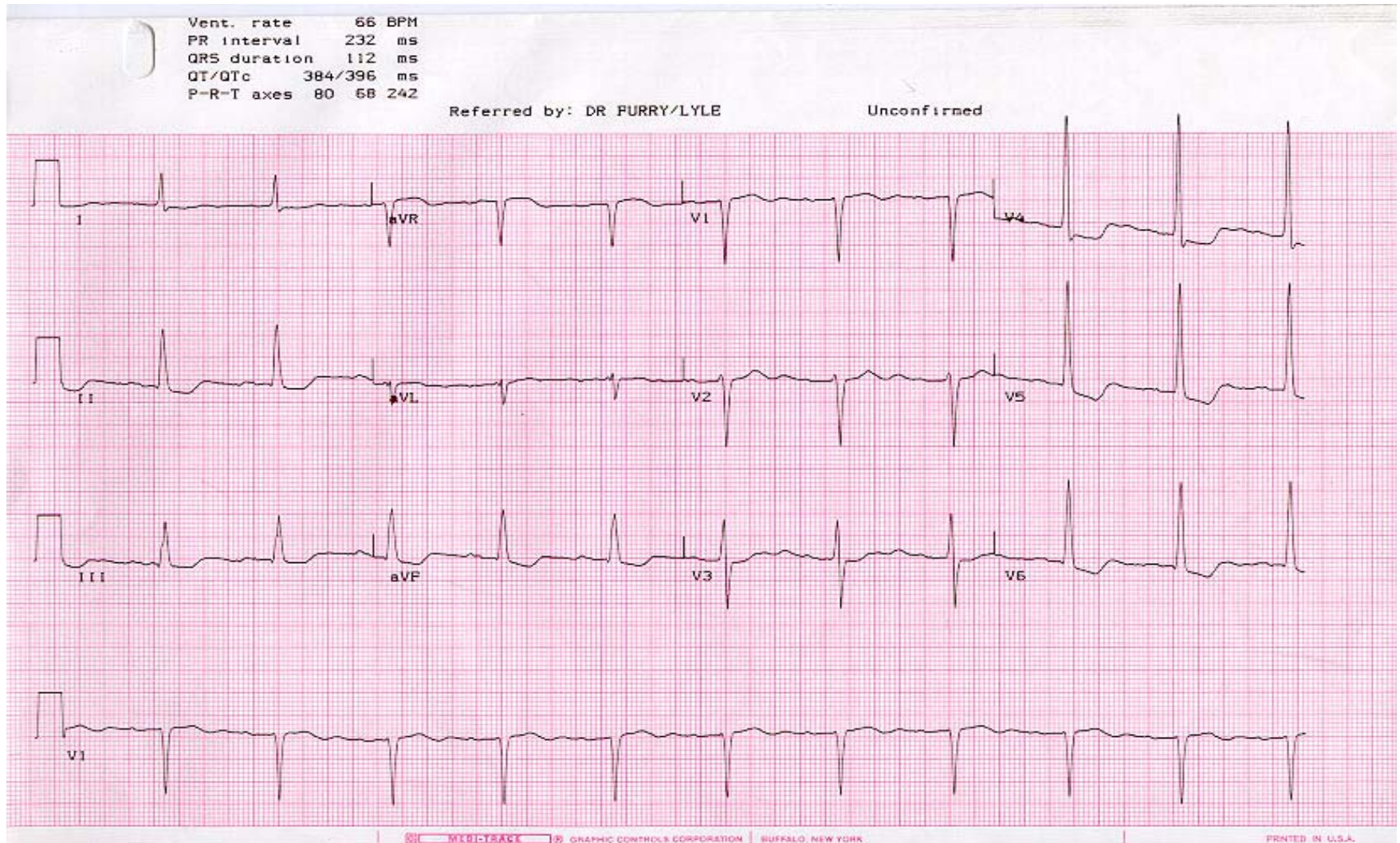
# Second Degree AV Block Type I: Shows the 5 features of Wenckebach

- Group beating (1) with progressive PR lengthening (2) and RR shortening (3).
- Pause less than 2 cycles (4).
- RR after pause longer than RR before pause (5).

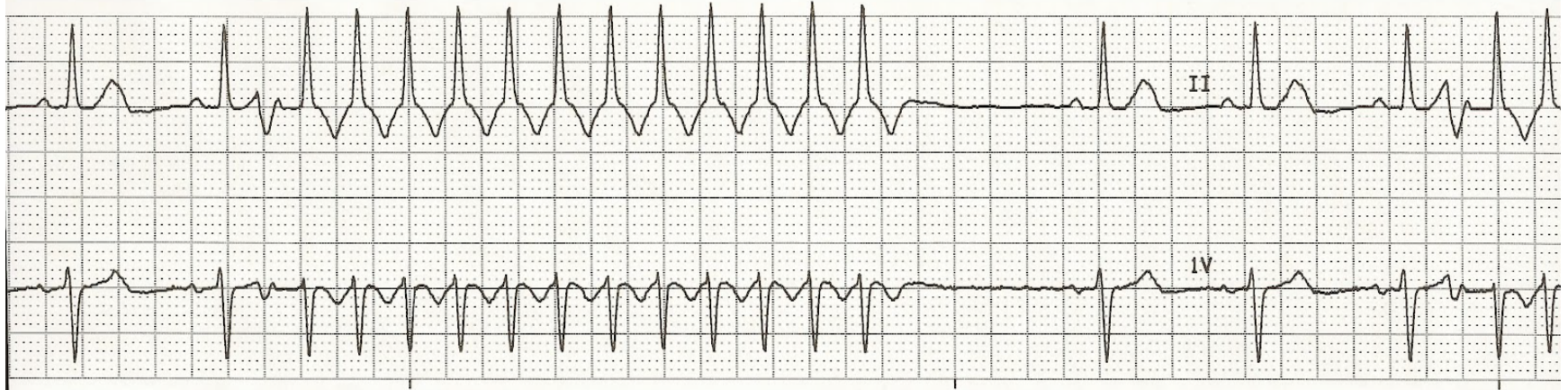




# Left Ventricular Hypertrophy

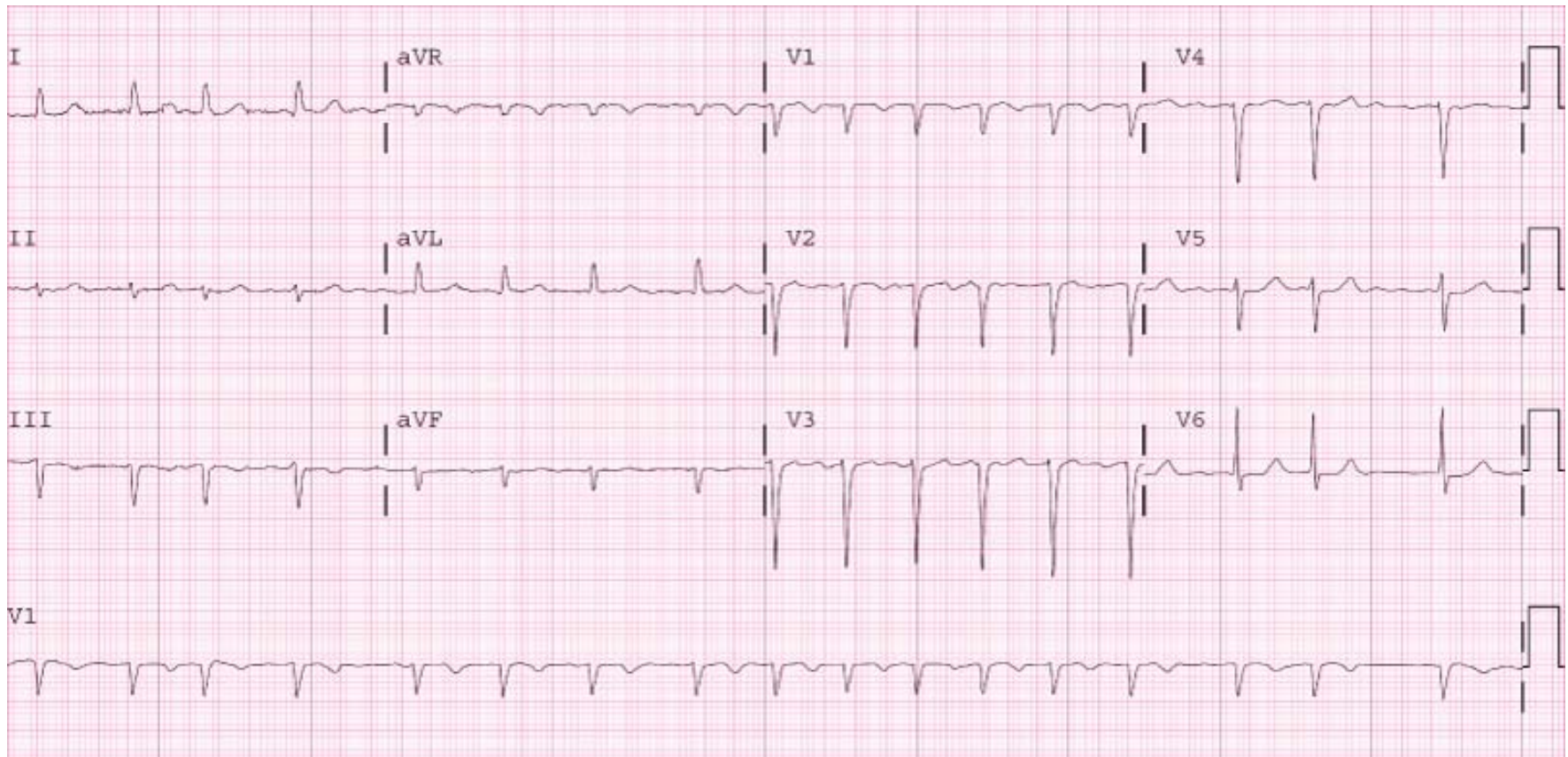


# PSVT with aberrant conduction



Note the tachycardia begins with a PAC and has slightly different QRS morphology and inverted T waves. Also note the short pause after termination of the tachycardia.

# Atrial Fibrillation



Source: Knoop KJ, Stack LB, Storrow AB, Thurman RJ: *The Atlas of Emergency Medicine, 3rd Edition*: <http://www.accessmedicine.com>

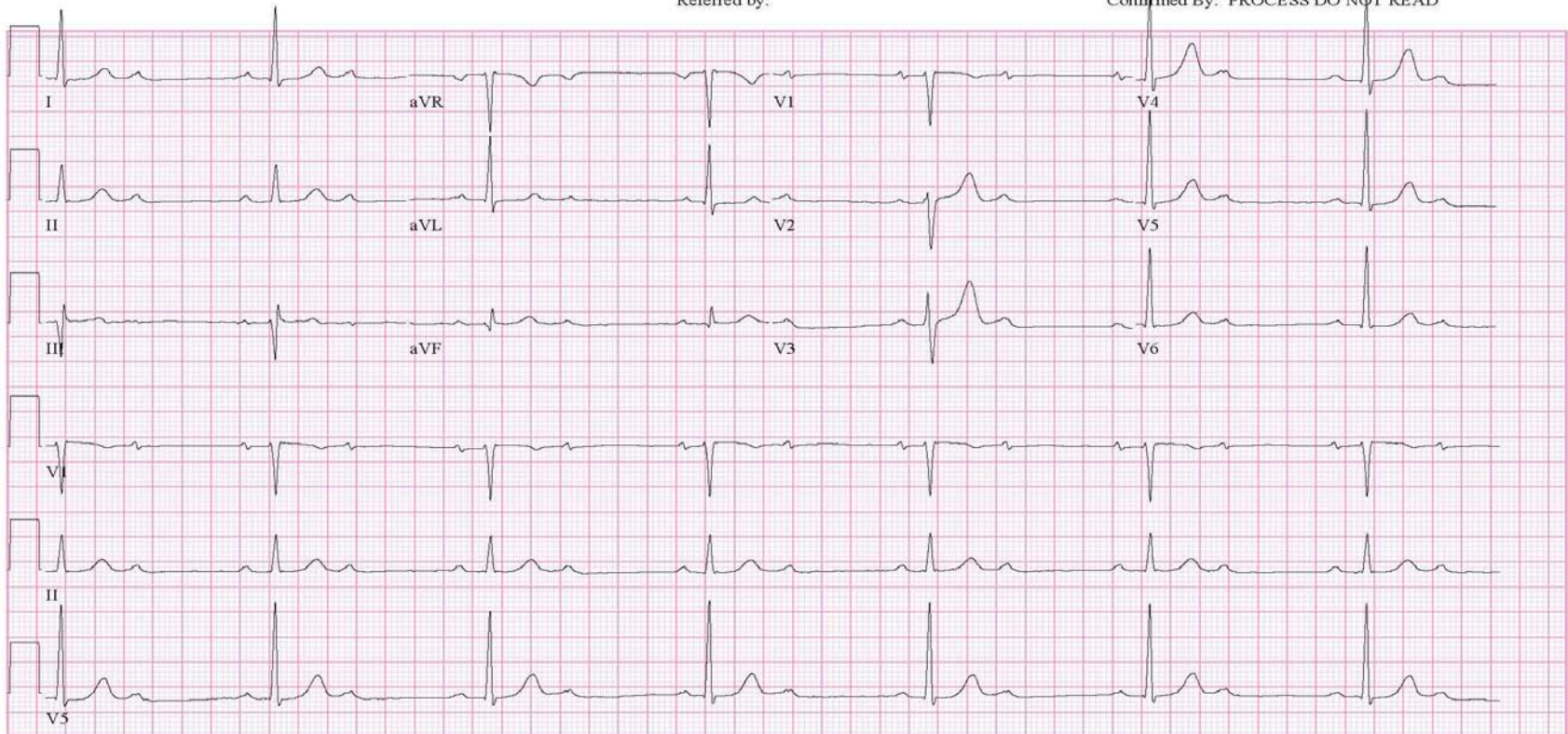
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# Second Degree AV block Type II

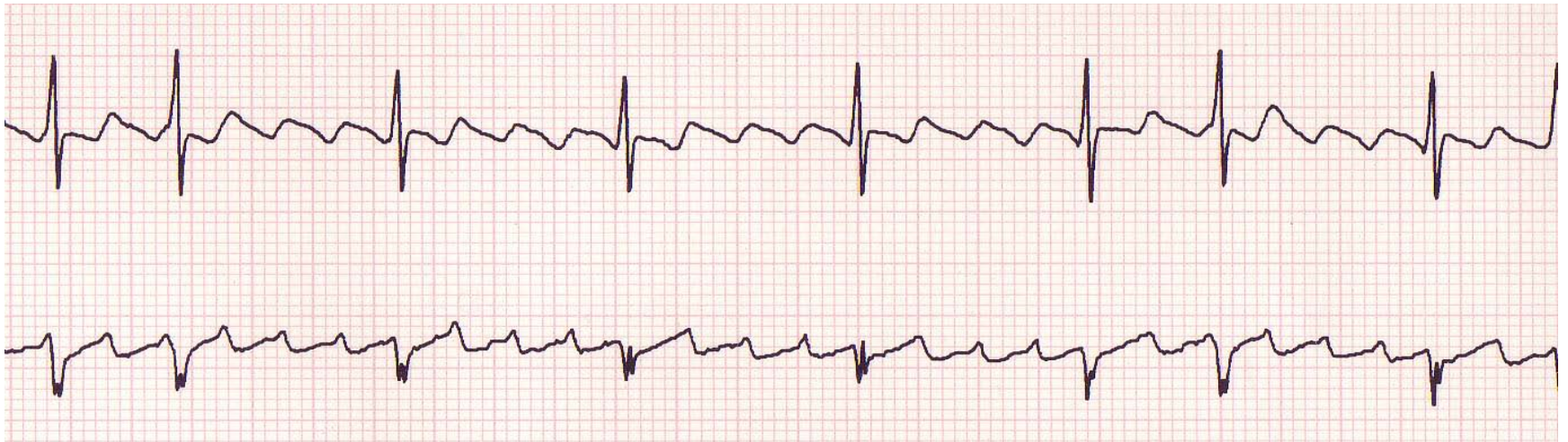
Vent. rate	40	BPM
PR interval	208	ms
QRS duration	88	ms
QT/QTc	432/352	ms
P-R-T axes	43 7	37

Referred by:

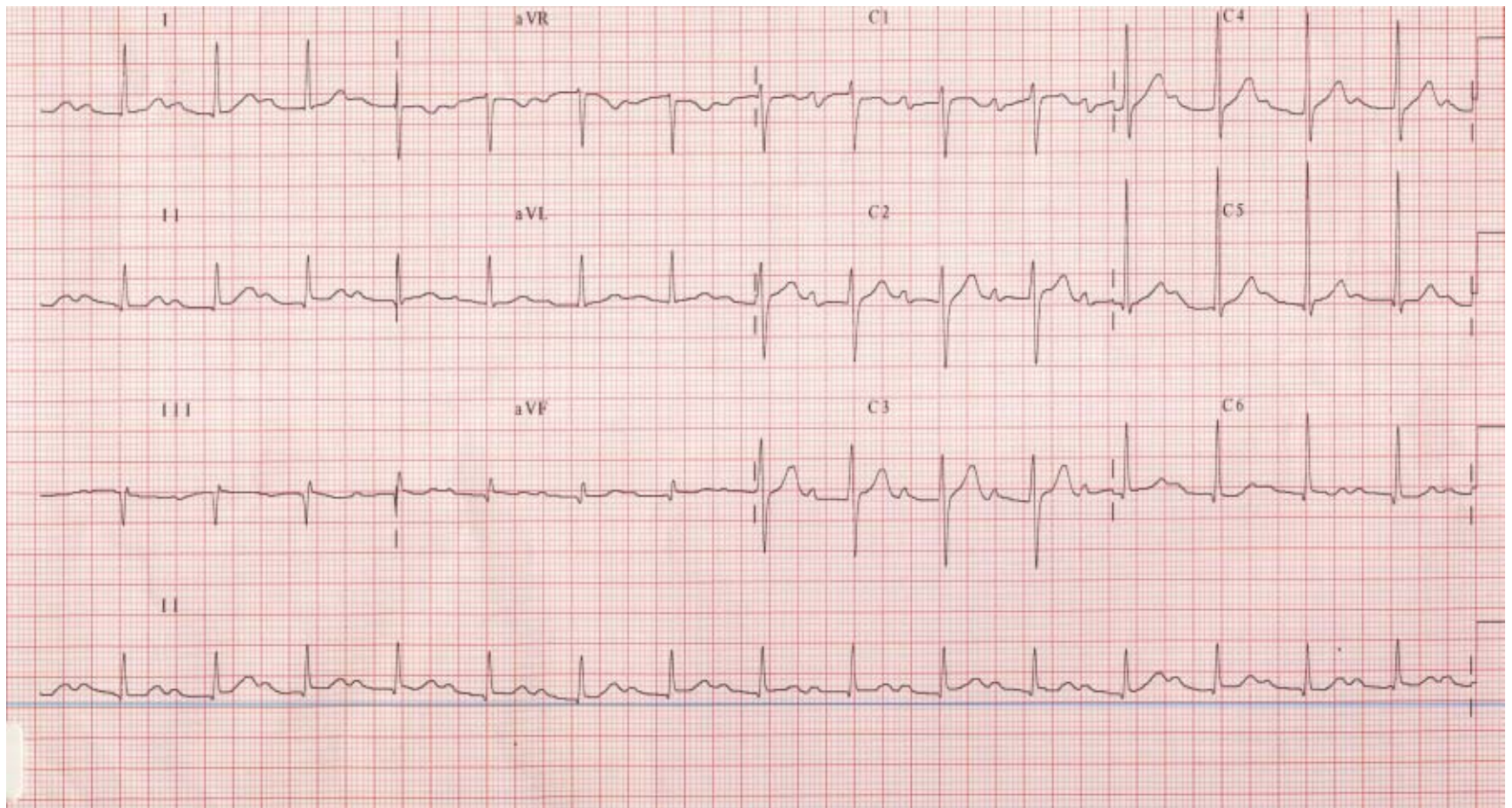
Confirmed By: PROCESS DO NOT READ



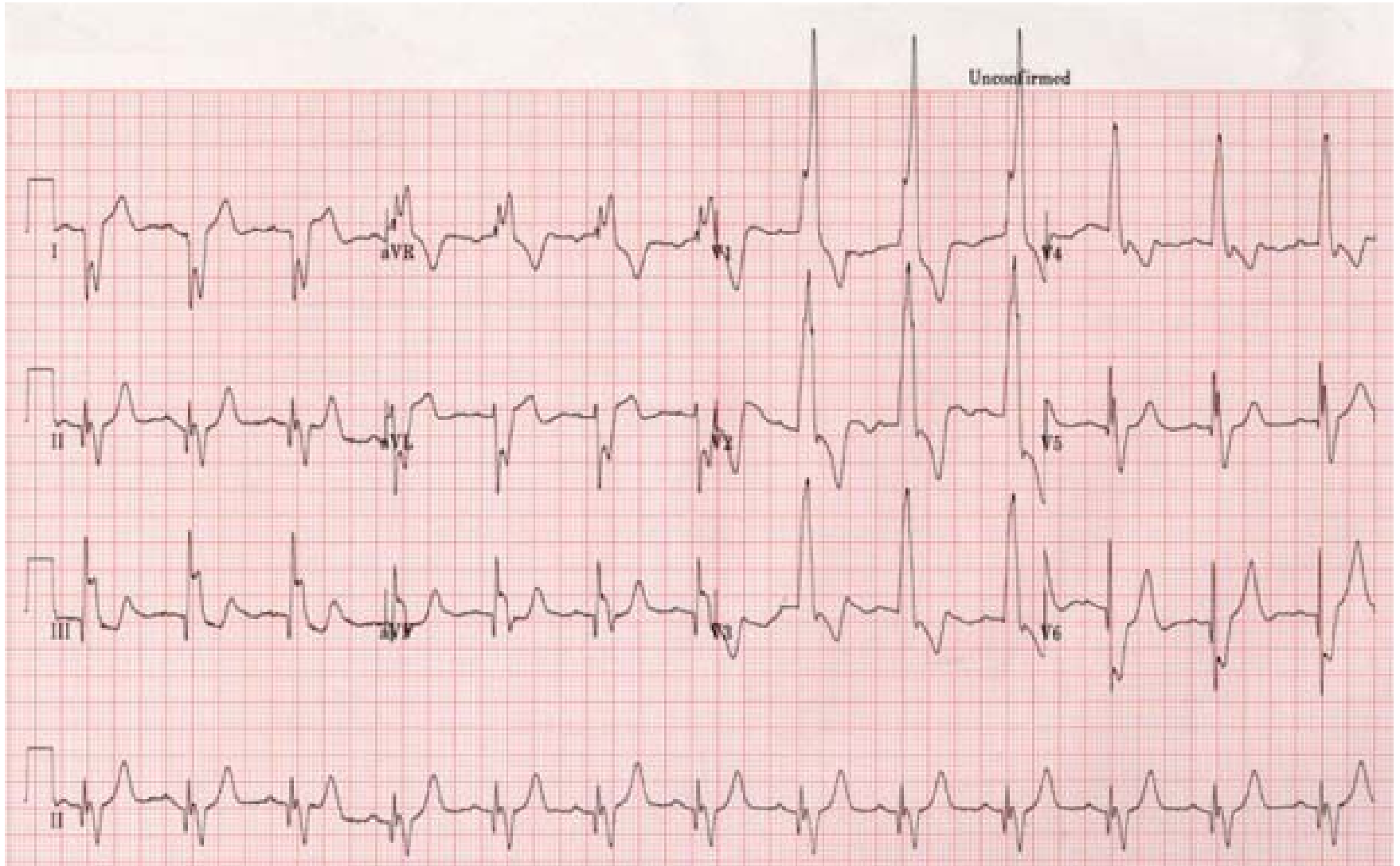
# Atrial Flutter (variable conduction)



# NSR with First Degree AV Block

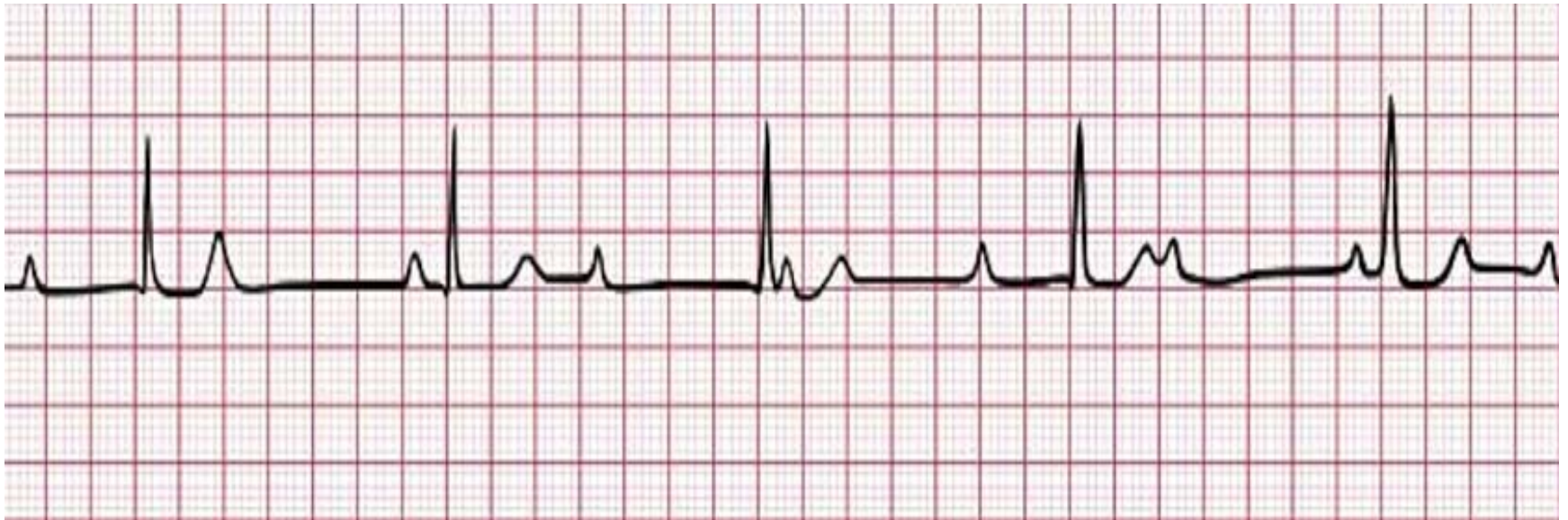


# Right Bundle Branch Block



# Third Degree AV Block

- Note the AV dissociation due to heart block

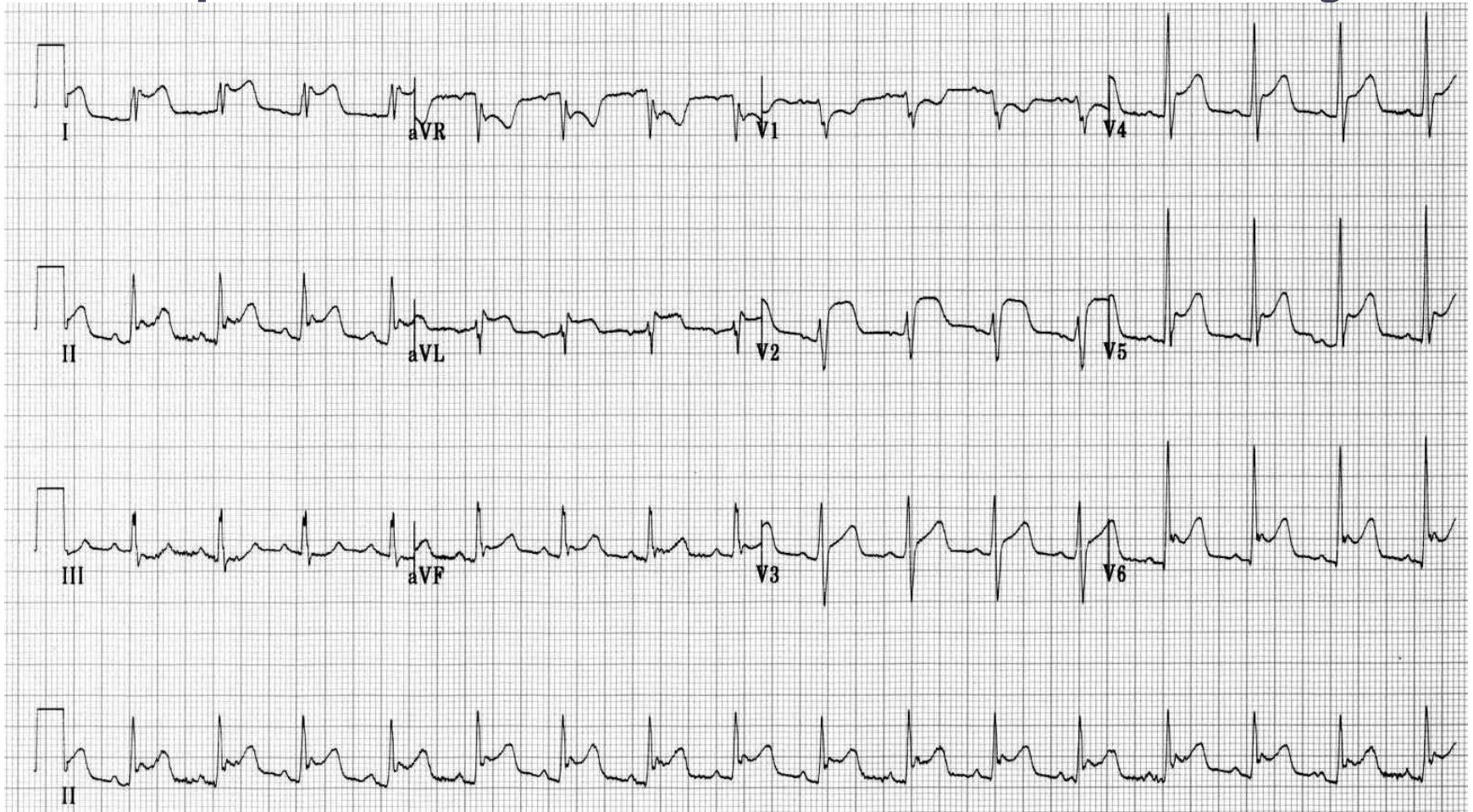


The atrial mechanism is sinus while the ventricles are controlled by a junctional escape rhythm.



# Acute Pericarditis:

Don't forget to look for signs of tamponade on EKG and clinically



# Cardiac Tamponade

Medscape®

www.medscape.com

