The 12-Lead EKG

Ben Taylor PA-C, PhD, DFAAPA

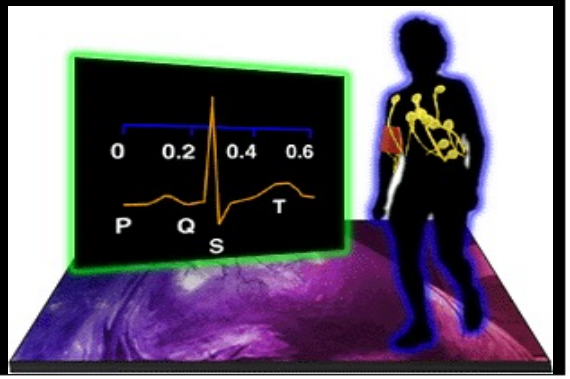
Disclosures

No relevant relationships with ineligible companies to disclose within the past 24 months

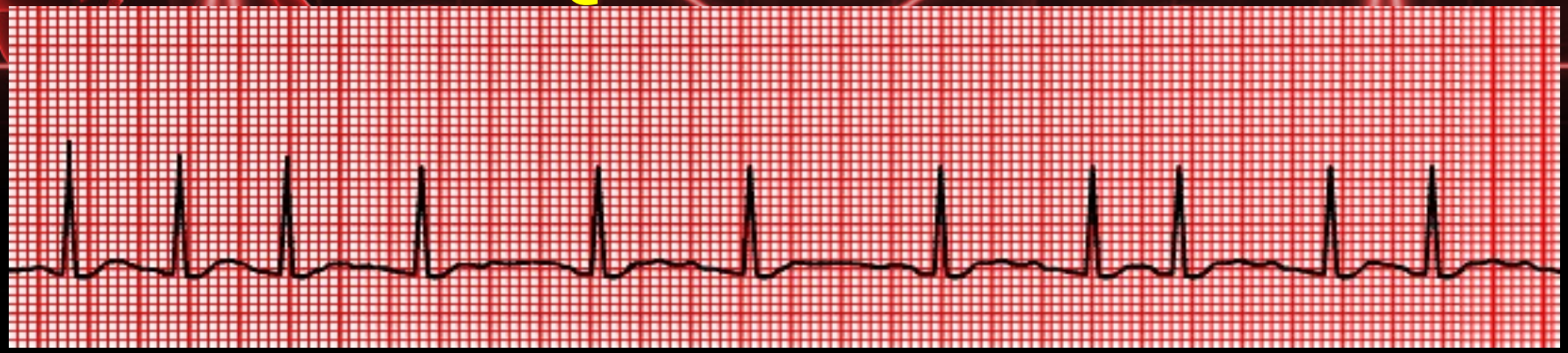
Course Objectives

At the conclusion of this session, participants should be able to:

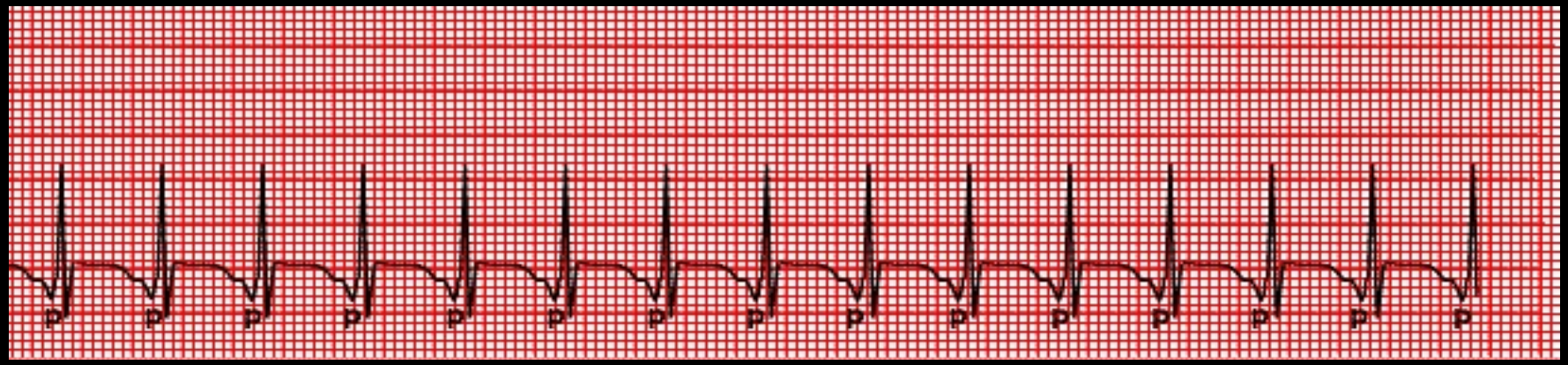
- Apply a systematic approach to the interpretation of EKG's
- Identify dysrhythmias and their relevant implications
- Determine the mean QRS axis and state the common causes of axis deviation
- Describe the 12-Lead EKG features of ischemia, injury, and infarction
- Recognize ventricular hypertrophy, atrial enlargement, and bundle branch blocks



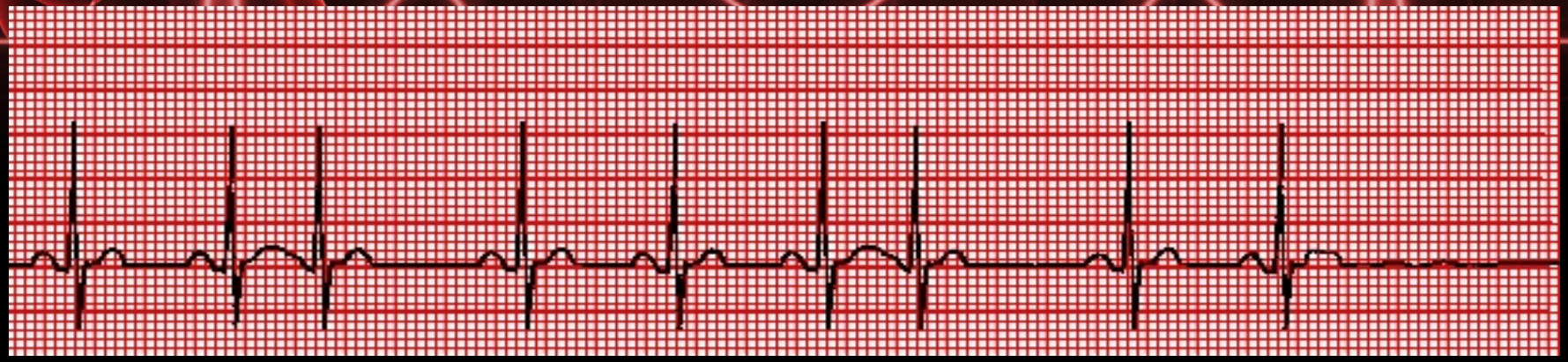
A Quick Review



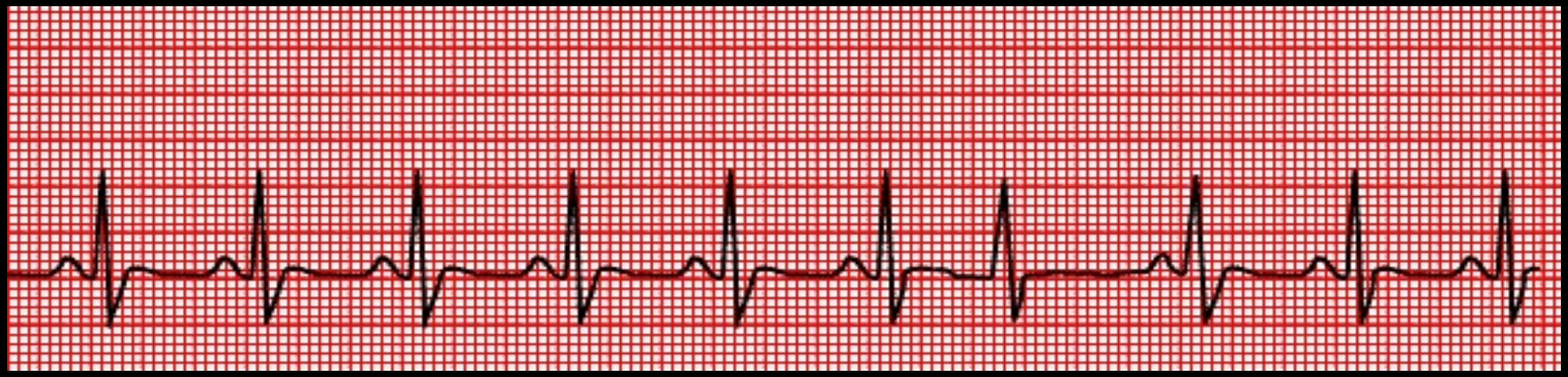
A-fib



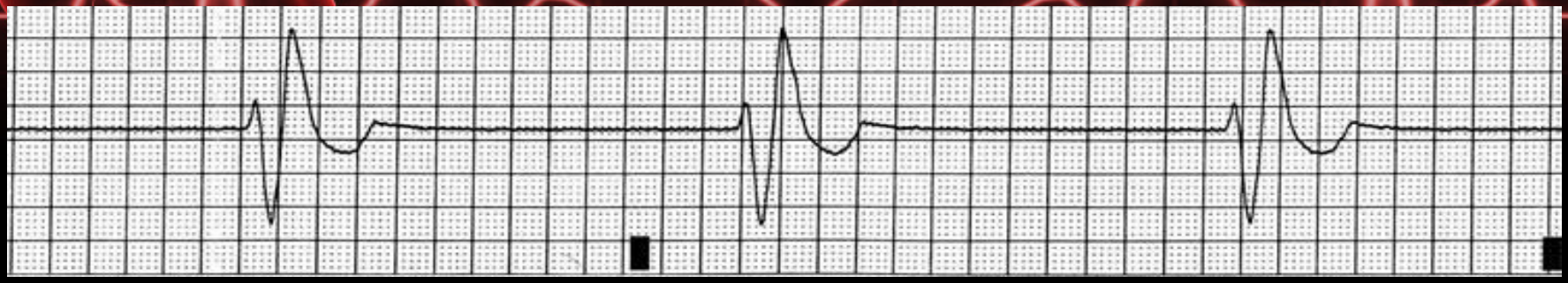
Junctional Tachycardia



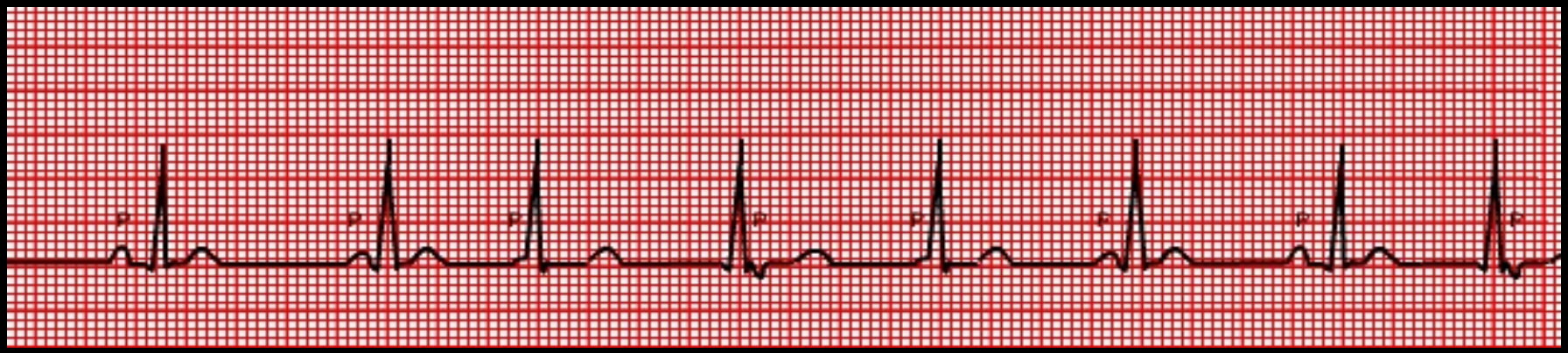
PAC



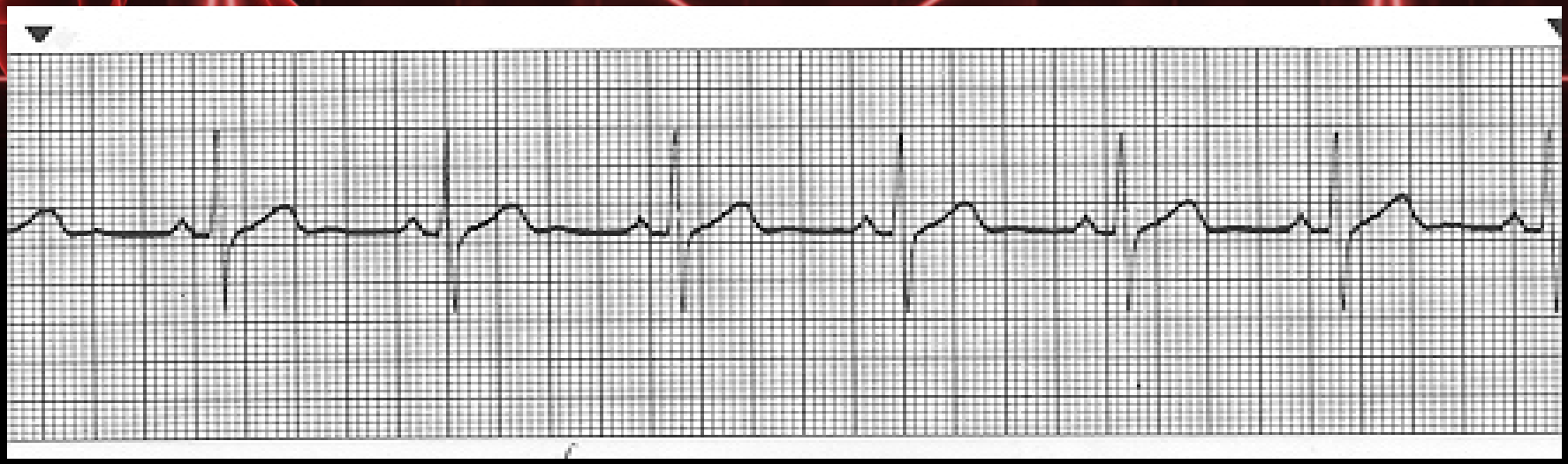
PJC



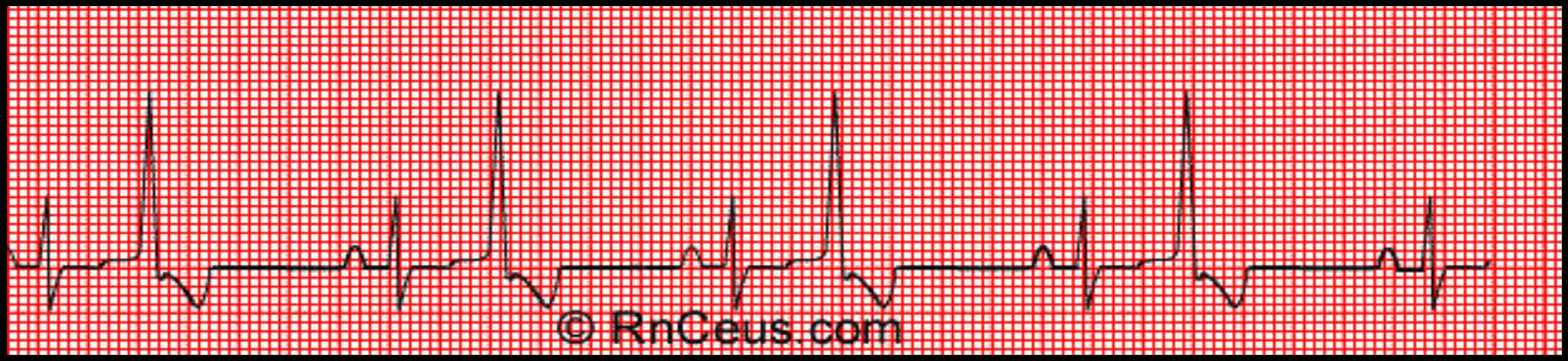
Idioventricular Rhythm



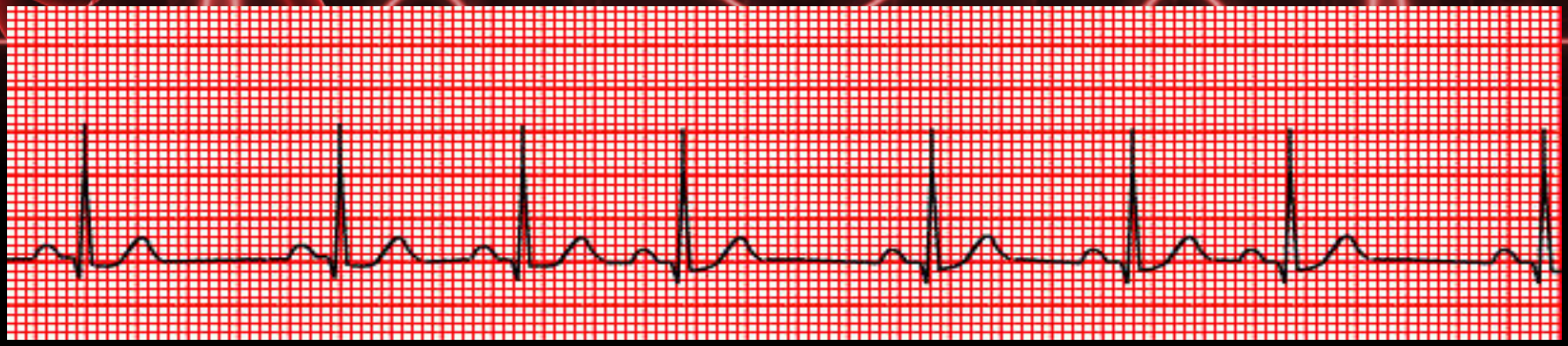
Wandering Pacemaker



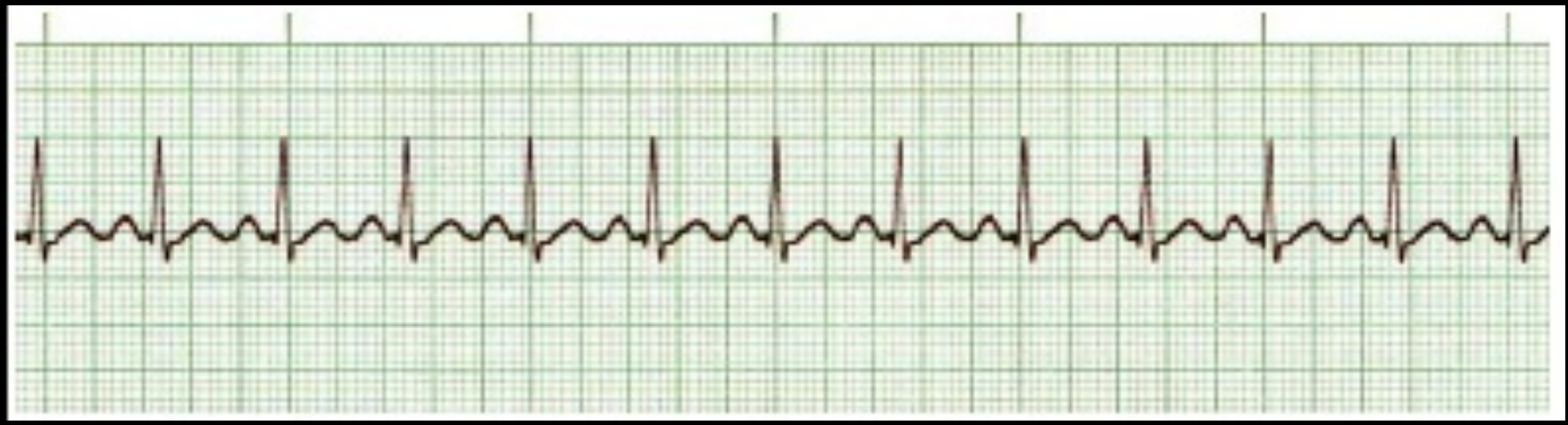
Normal Sinus Rhythm



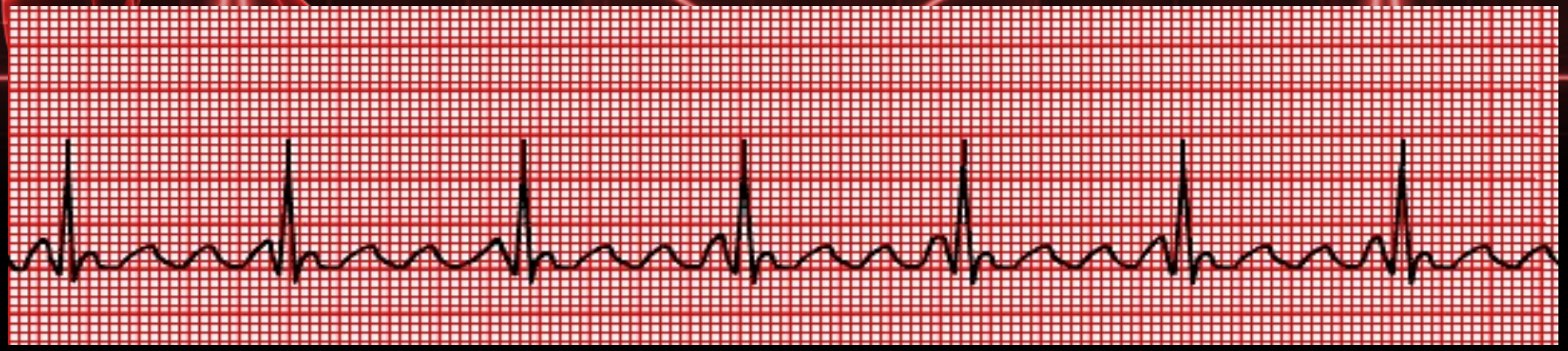
Sinus Rhythm with Bigeminy



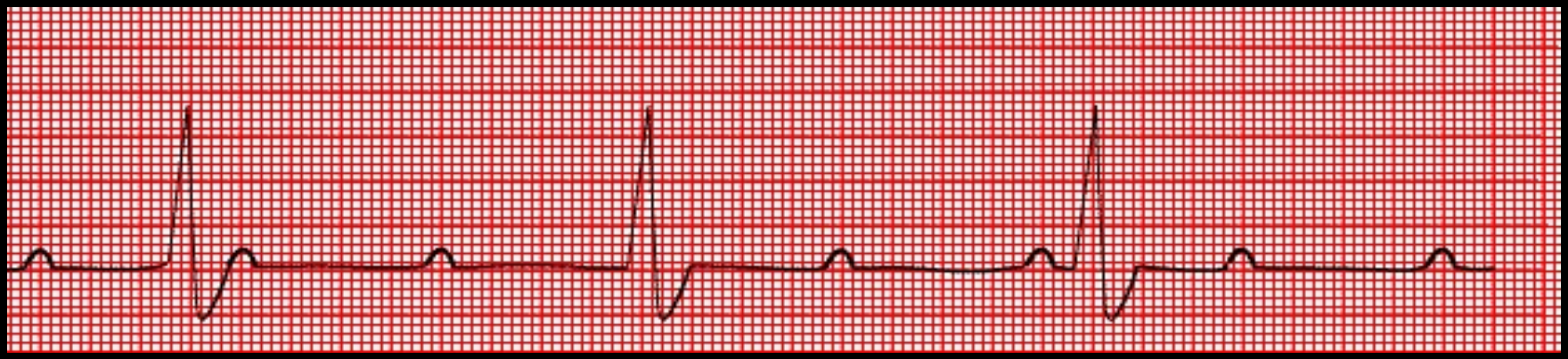
Sinus Arrhythmia



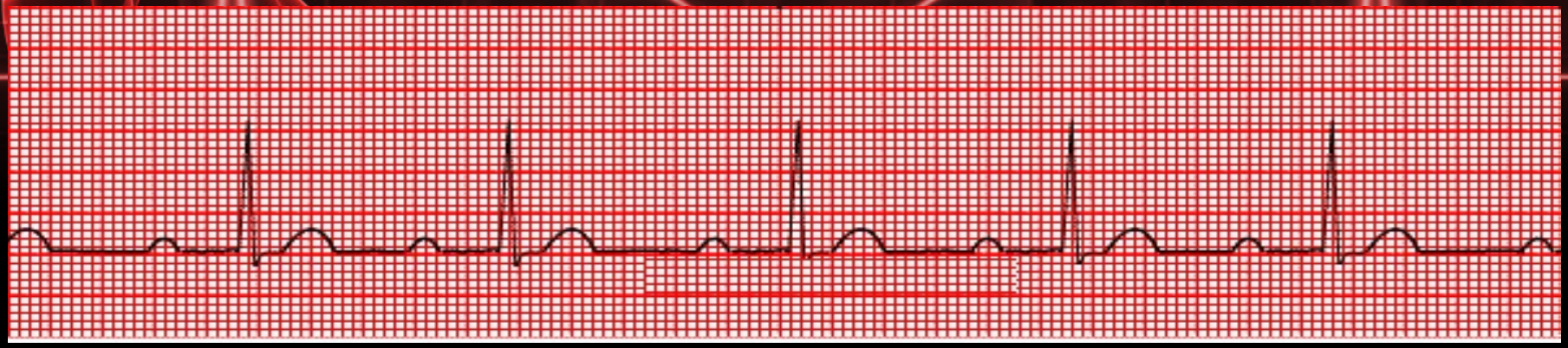
Sinus Tachycardia



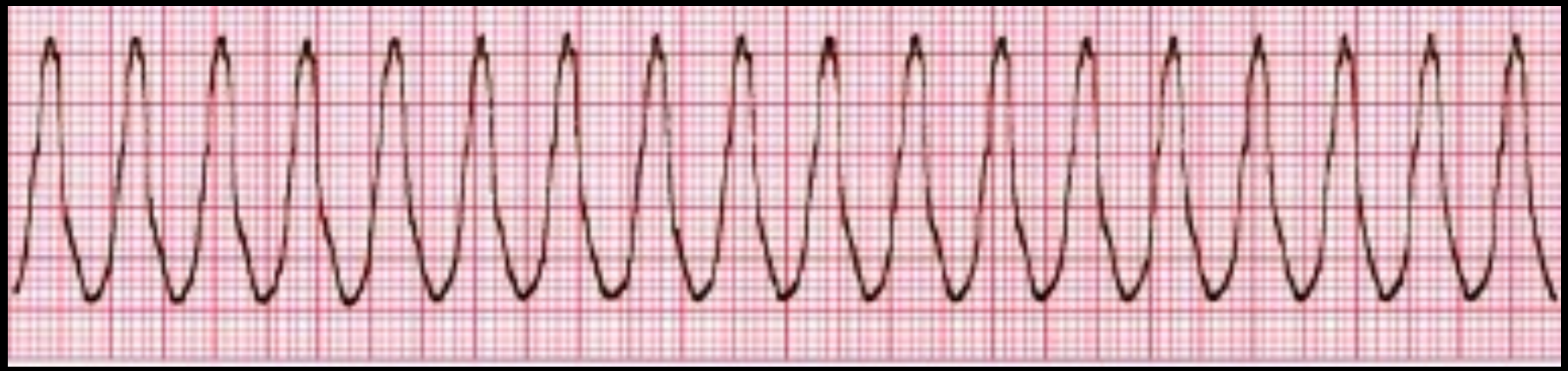
Atrial Flutter



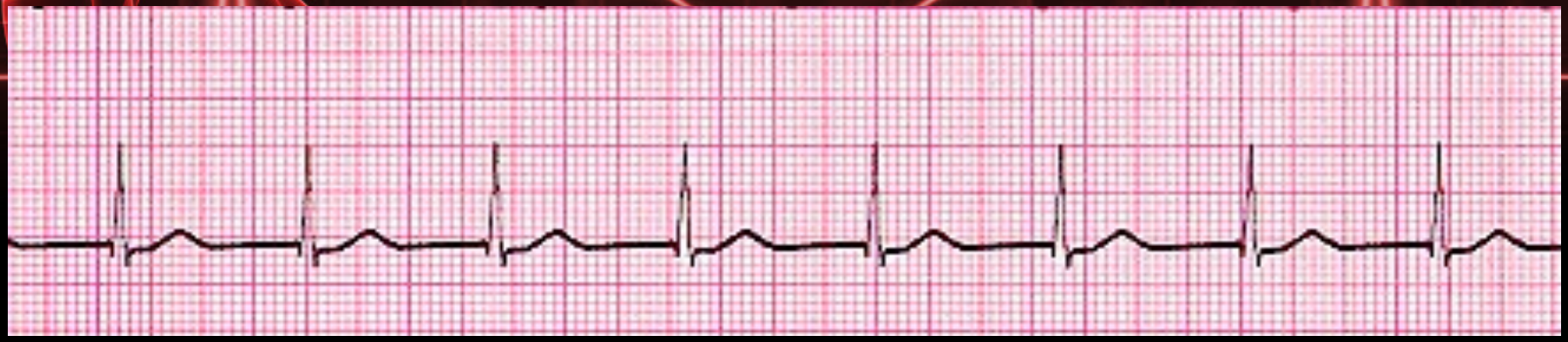
3rd Degree AV Block



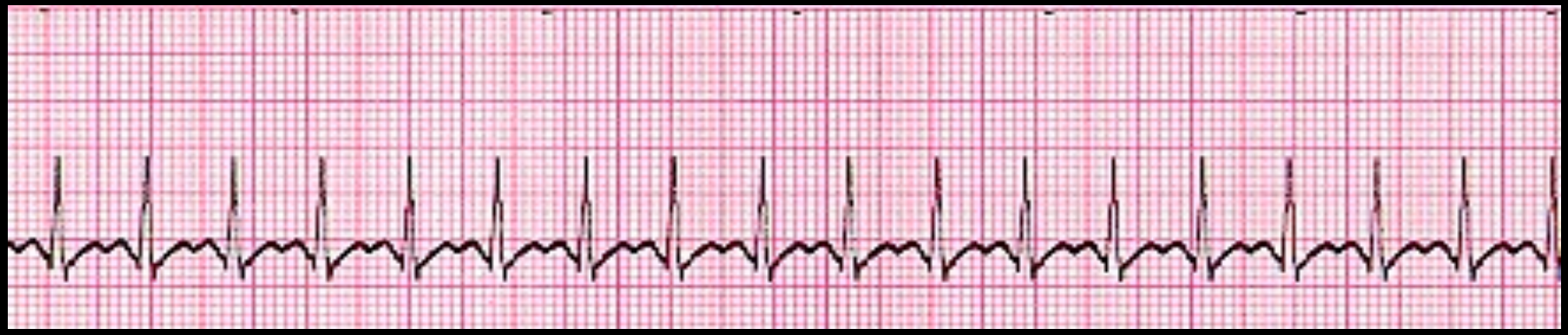
1st Degree AV Block



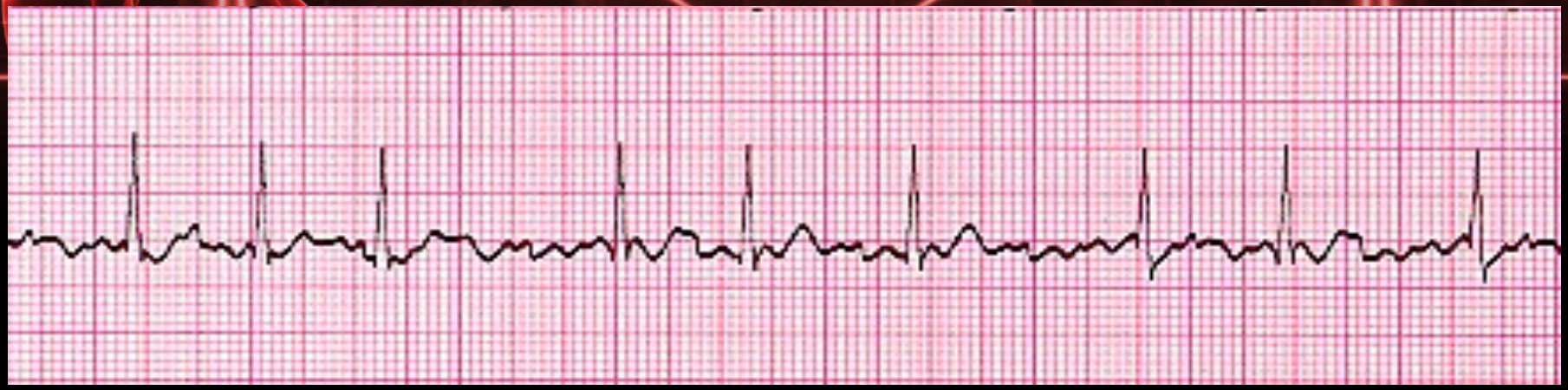
Ventricular Tachycardia



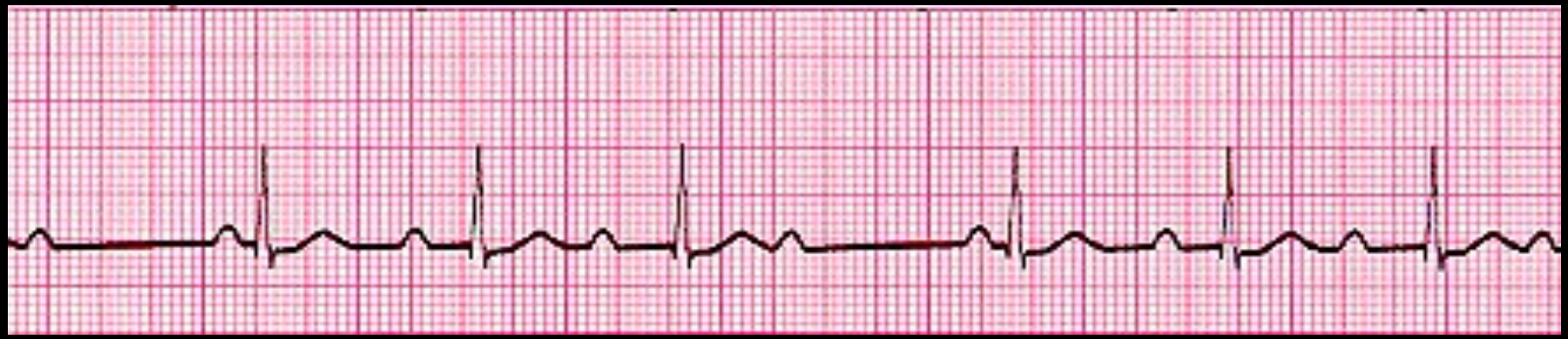
Accelerated Junctional Rhythm



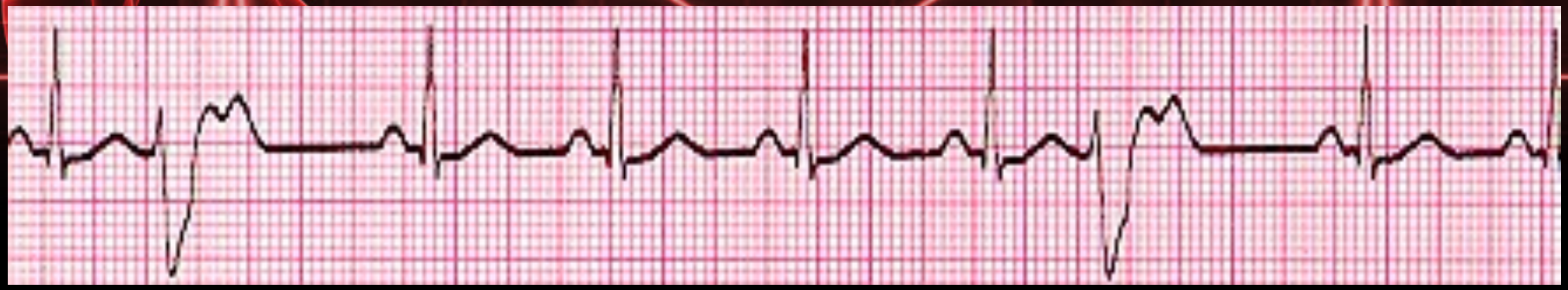
Supraventricular Tachycardia



Atrial Fibrillation (again)

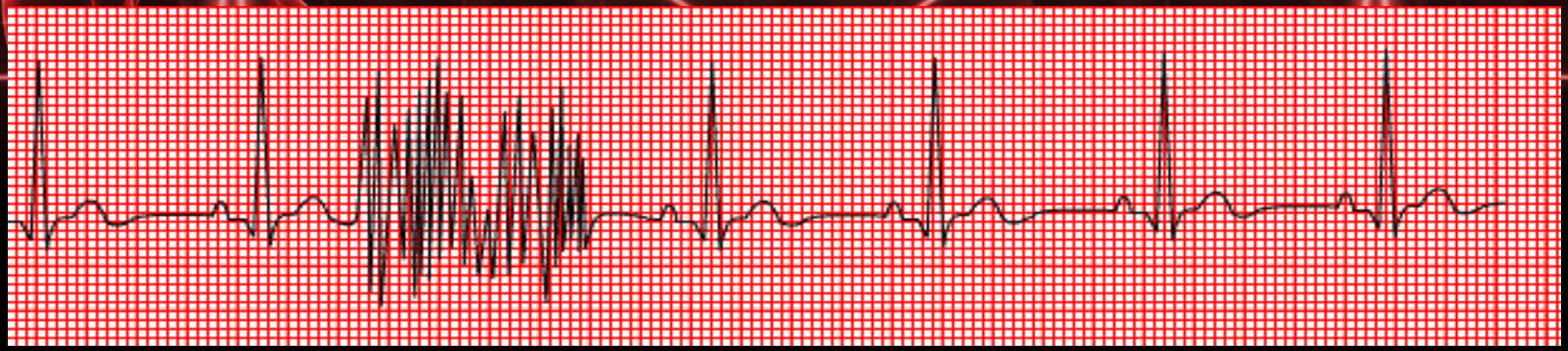


2nd Degree AV Block type I (**Wenchebach)**

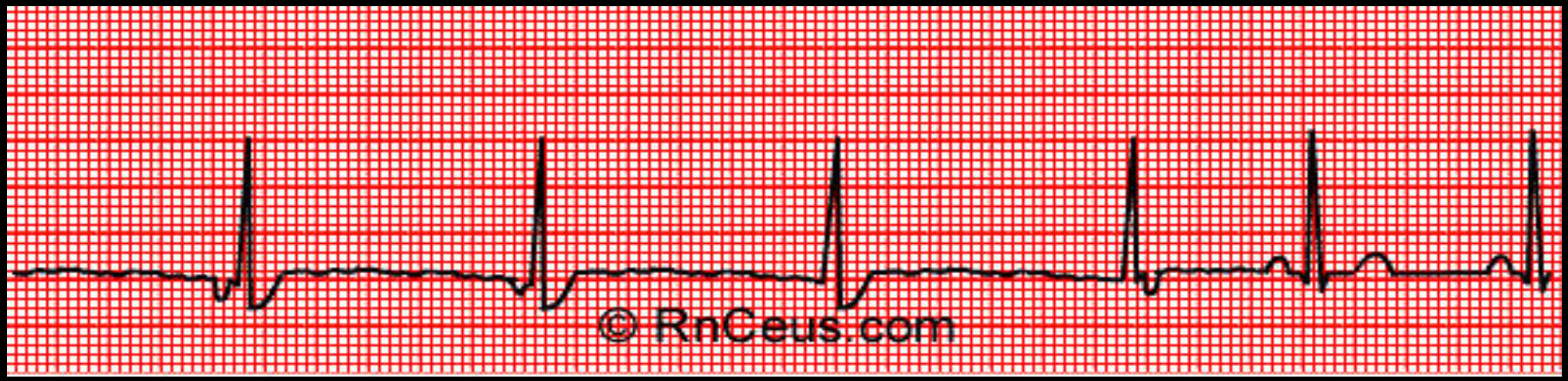


Sinus Rhythm with Unifocal PVC's

Torsade de Pointes



Sinus Rhythm with Artifact

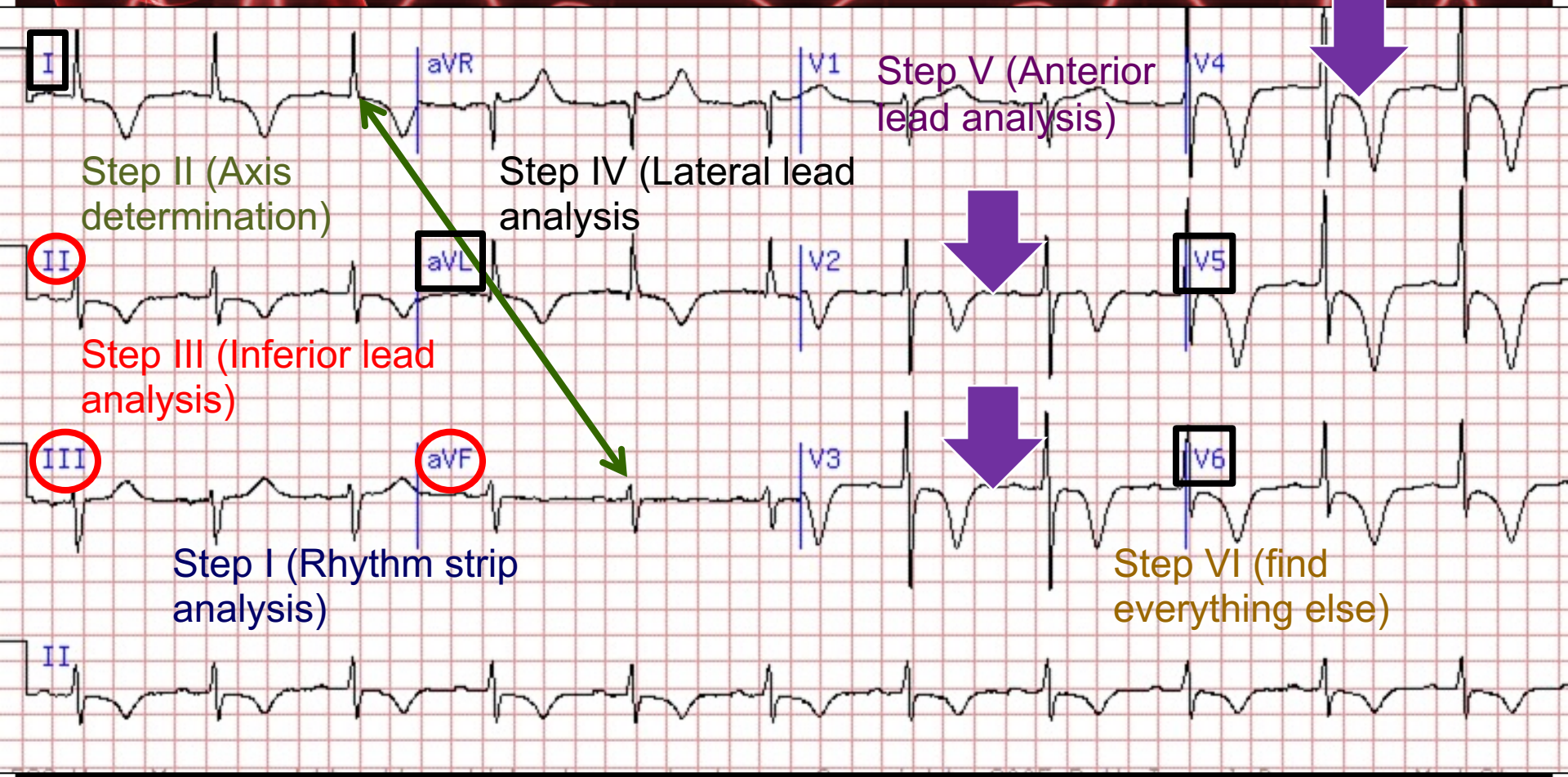


Junctional Escape Rhythm

Steps in Interpreting the 12-Lead

1. Assess the rate (atrial and ventricular) and regularity of the underlying rhythm.
   a) Assess the usual intervals and widths: PR interval, QRS width, QT interval.
   b) Interpret the rhythm itself.
2. Determine the axis.
3. Grouped lead analysis
   a) Look for signs of infarct vs. ischemia in all grouped leads
4. Look for any other abnormality

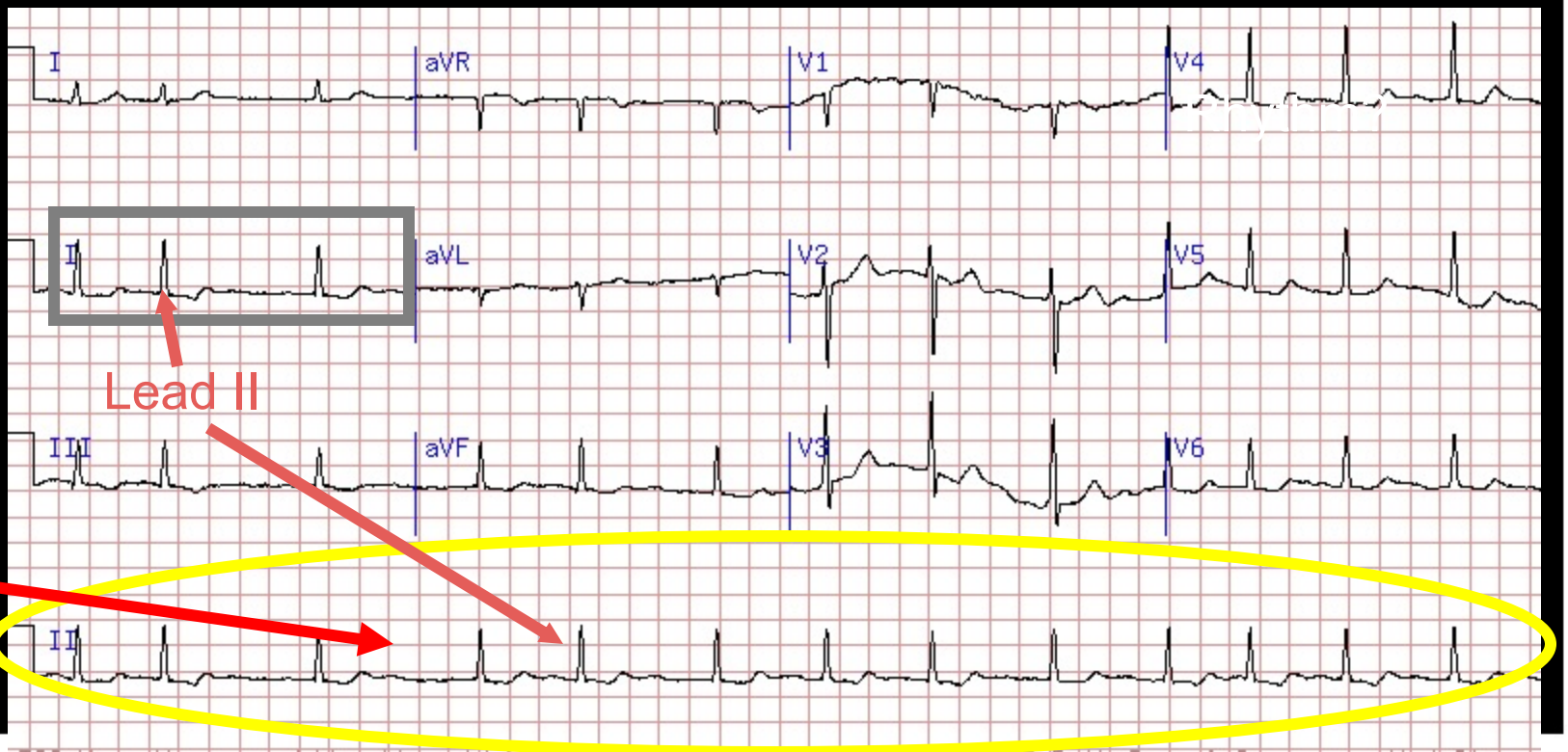
Assessing the EKG



Assessing the EKG Rhythm

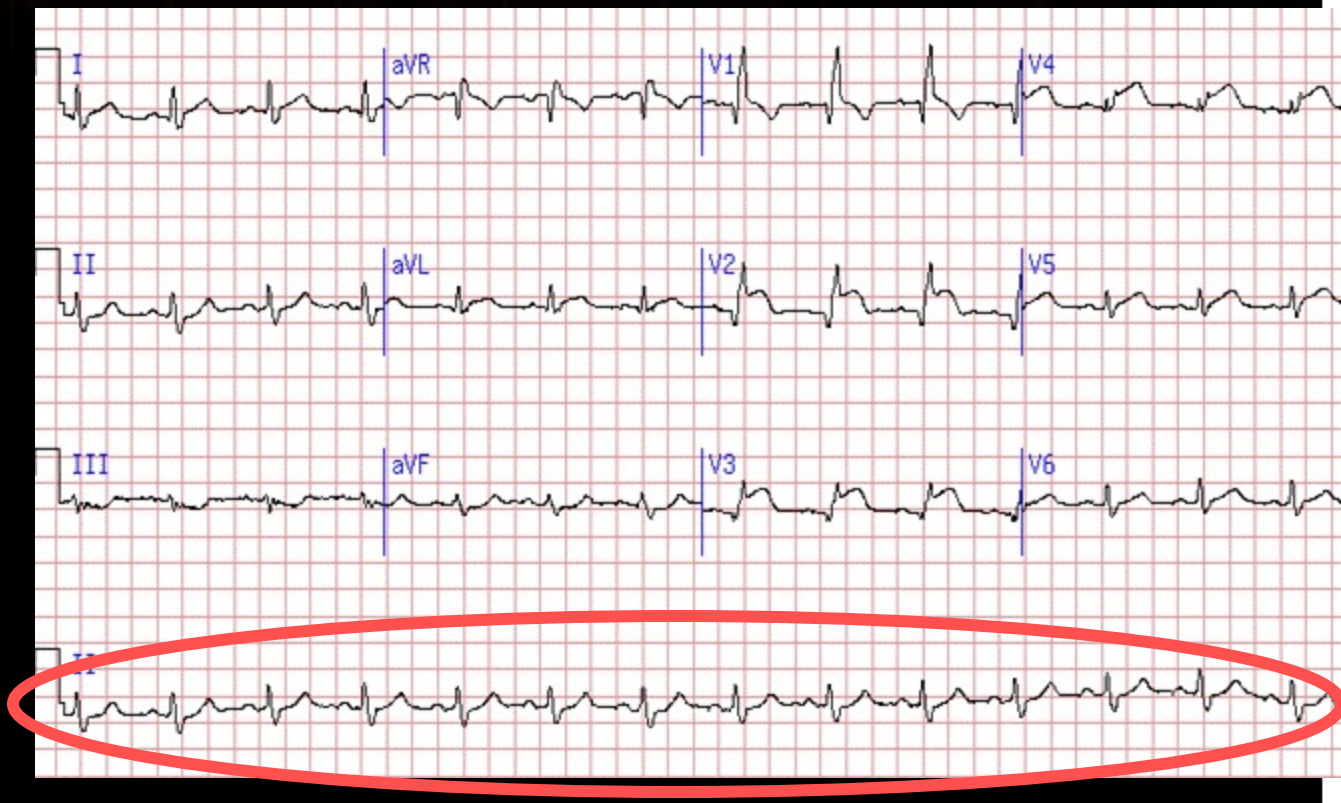
Tip: the rhythm strip portion of the 12-lead EKG is a good place to look at when trying to determine the rhythm because the 12 leads only capture a few beats.

Atrial fibrillation



Assessing the EKG Rate

If you use the rhythm strip portion of the 12-lead EKG the total length of it is always 10 seconds long. So you can count the number of R waves in the rhythm strip and multiply by 6 to determine the beats per minute.

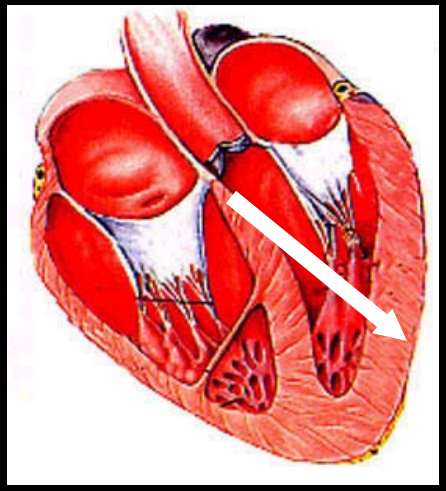


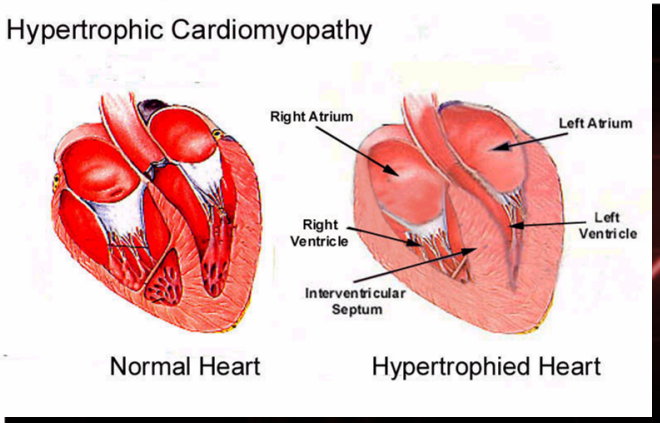
Rate?

14 (R waves) x 6 = 84 bpm

Assessing the Axis

Axis refers to the mean QRS axis (or vector) during ventricular depolarization. As you recall when the ventricles depolarize (in a normal heart) the direction of current flows leftward and downward because most of the ventricular mass is in the left ventricle. We like to know the QRS axis because an abnormal axis can suggest disease such as pulmonary hypertension from a pulmonary embolism.

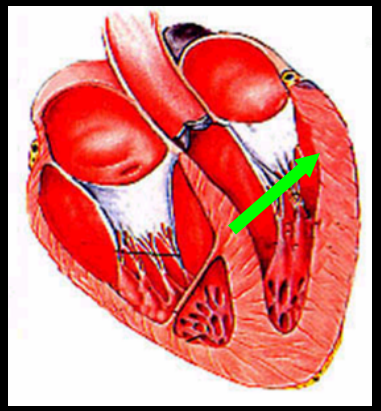


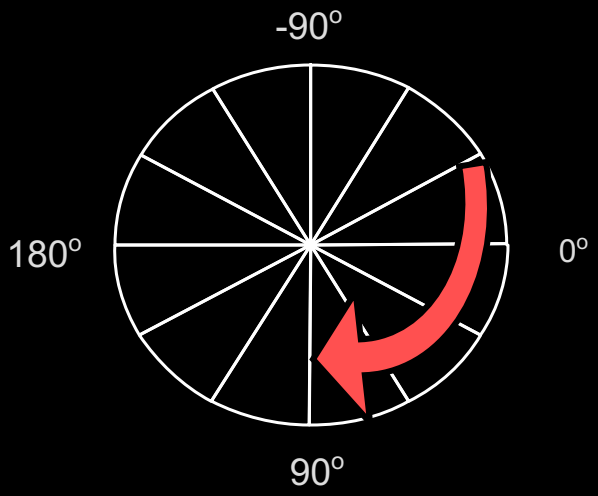


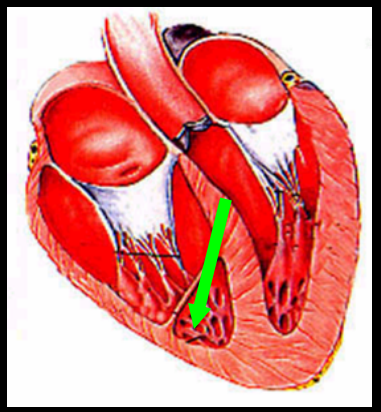
Assessing the Axis

- Causes of left axis deviation include:
  - **Left ventricular hypertrophy**
  - **Inferior wall MI**
  - **Left bundle branch block**
  - **Left anterior fascicular block**

- Causes of right axis deviation include:
  - **Right ventricular hypertrophy**
  - **Lateral wall MI**
  - **Right bundle branch block**
  - **Pulmonary hypertension**





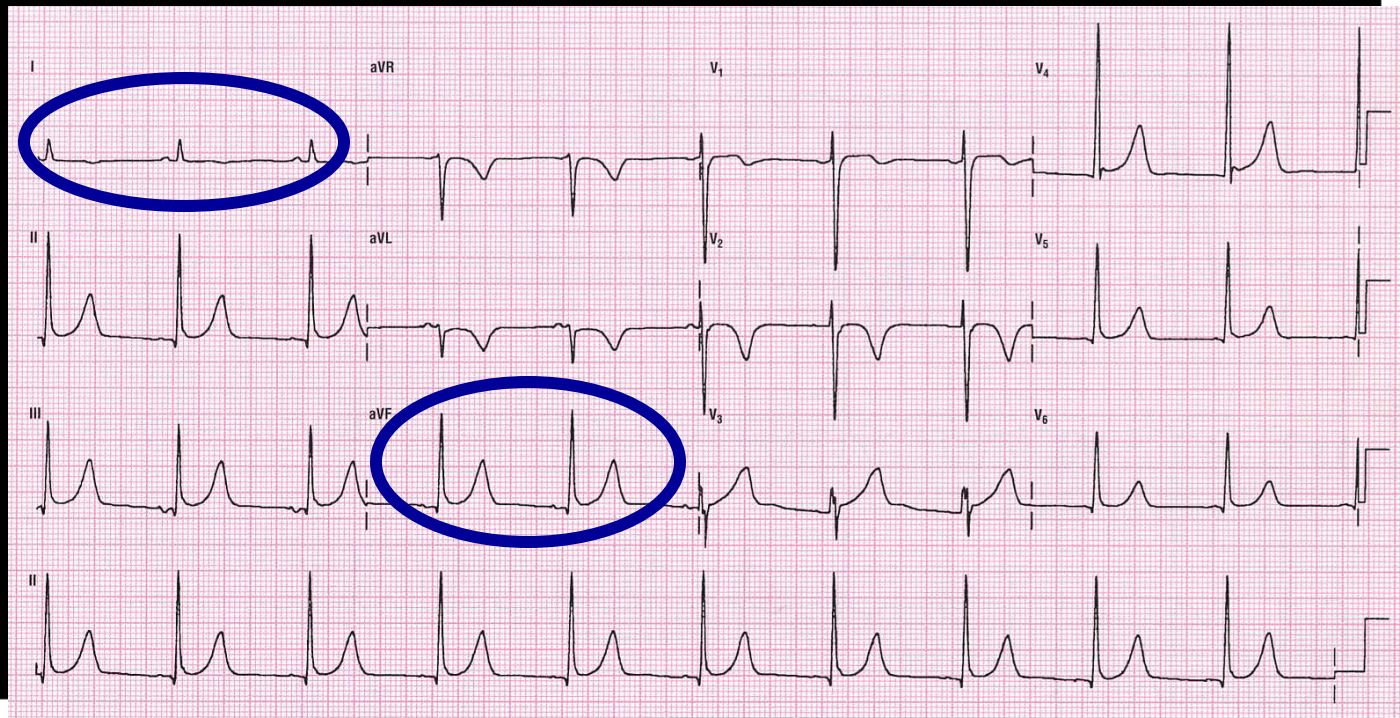


Assessing the Axis

We can quickly determine whether the QRS axis is normal by looking at leads I and aVF.

If the QRS complex is overall positive in leads I and aVF, the QRS axis is normal.

Both R-waves are more + than the S-waves



Assessing the Axis

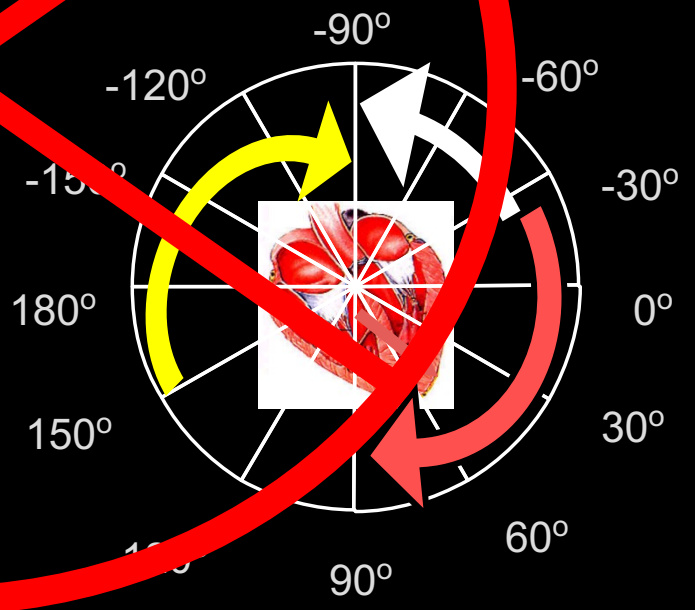
The QRS axis is determined by overlying a circle, in the frontal plane. By convention, the degrees of the circle are as shown

The normal QRS axis lies between -30º and +90º.

A QRS axis that falls between -30º and -90º is abnormal and called left axis deviation.

A QRS axis that falls between +90º and +150º is abnormal and called right axis deviation.

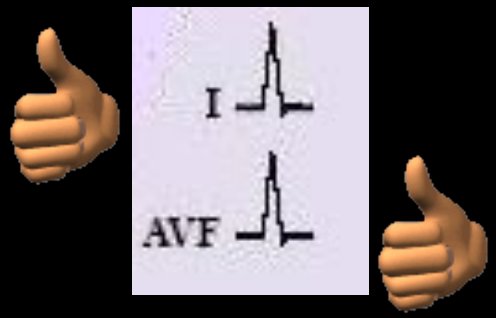
A QRS axis that falls between +150º and -90º is abnormal and called extreme right axis deviation.



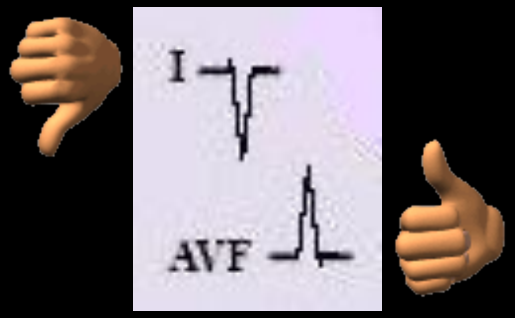
Assessing the Axis

The thumb method

Always go with the positive thumb!!!!



Normal Axis

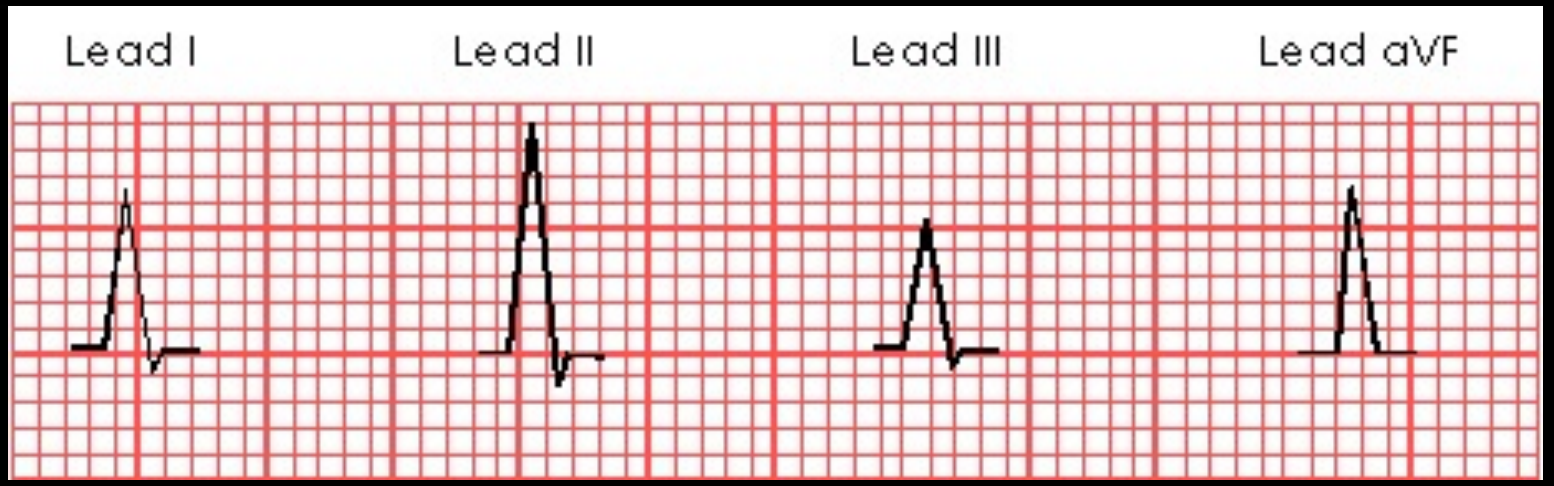


Right Axis

Left Axis

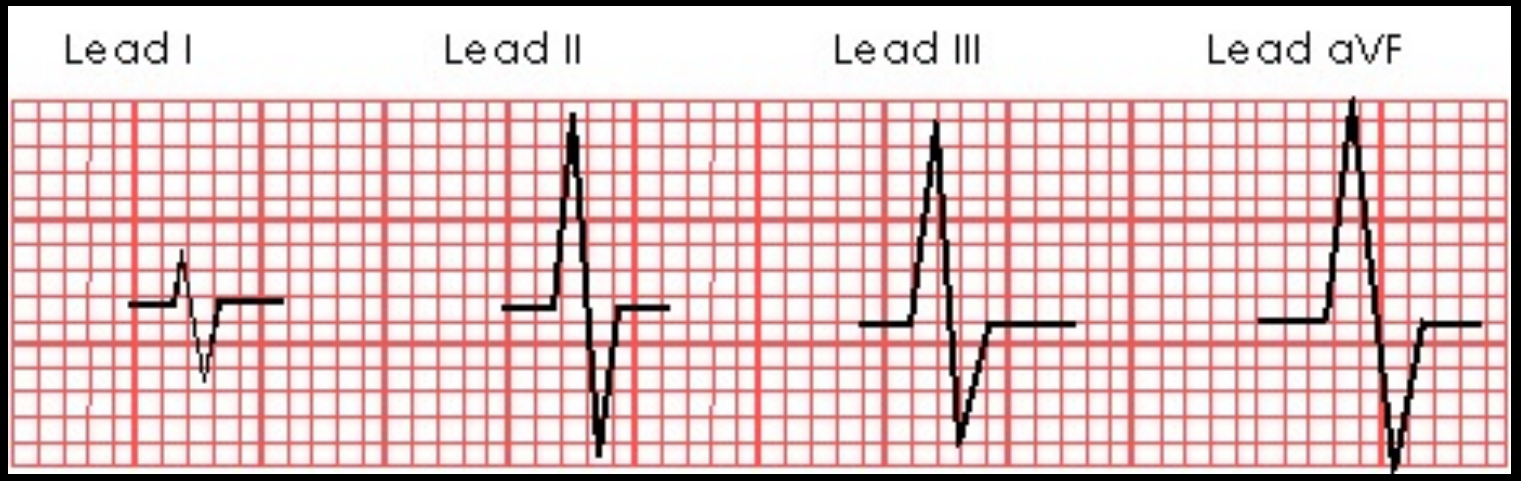
Extreme Right Axis

Normal, Right or Left Axis?



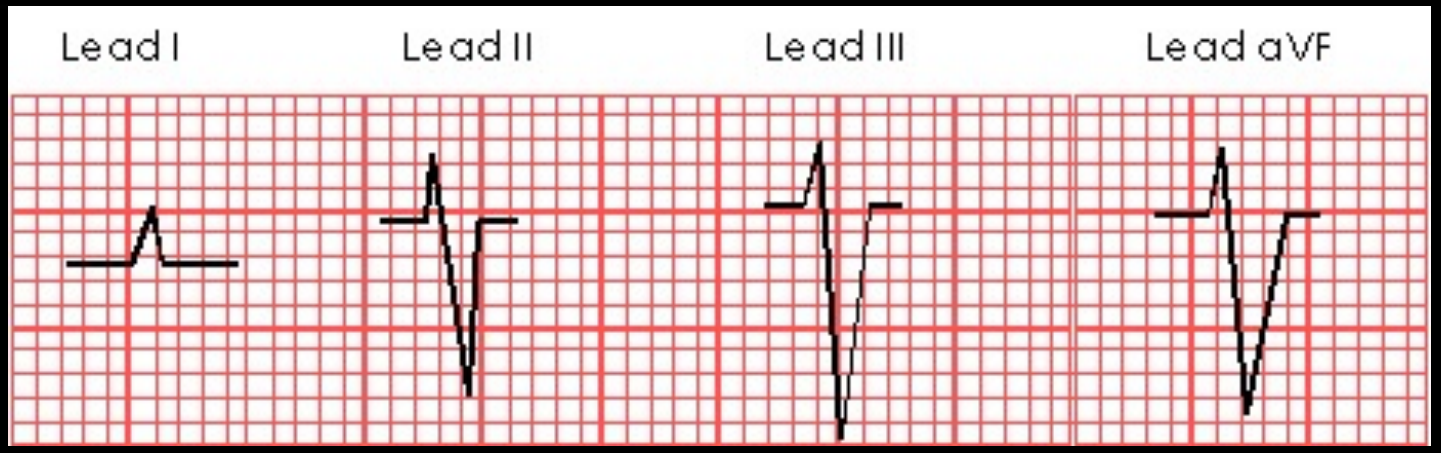
Normal

Normal, Right or Left Axis?



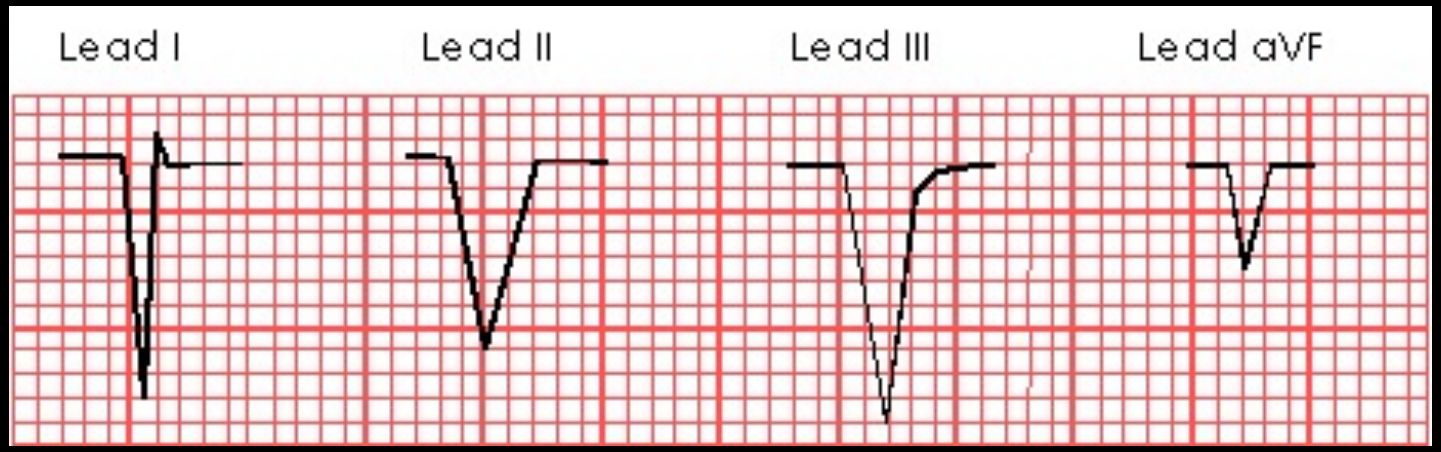
Right

Normal, Right or Left Axis?



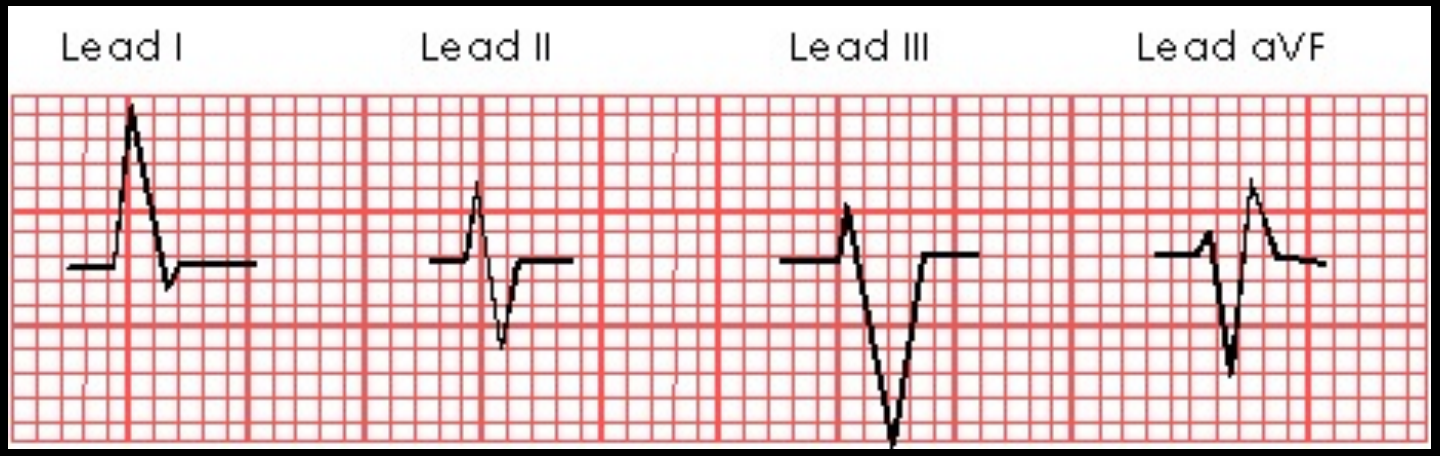
Left

Normal, Right or Left Axis?



Extreme Right

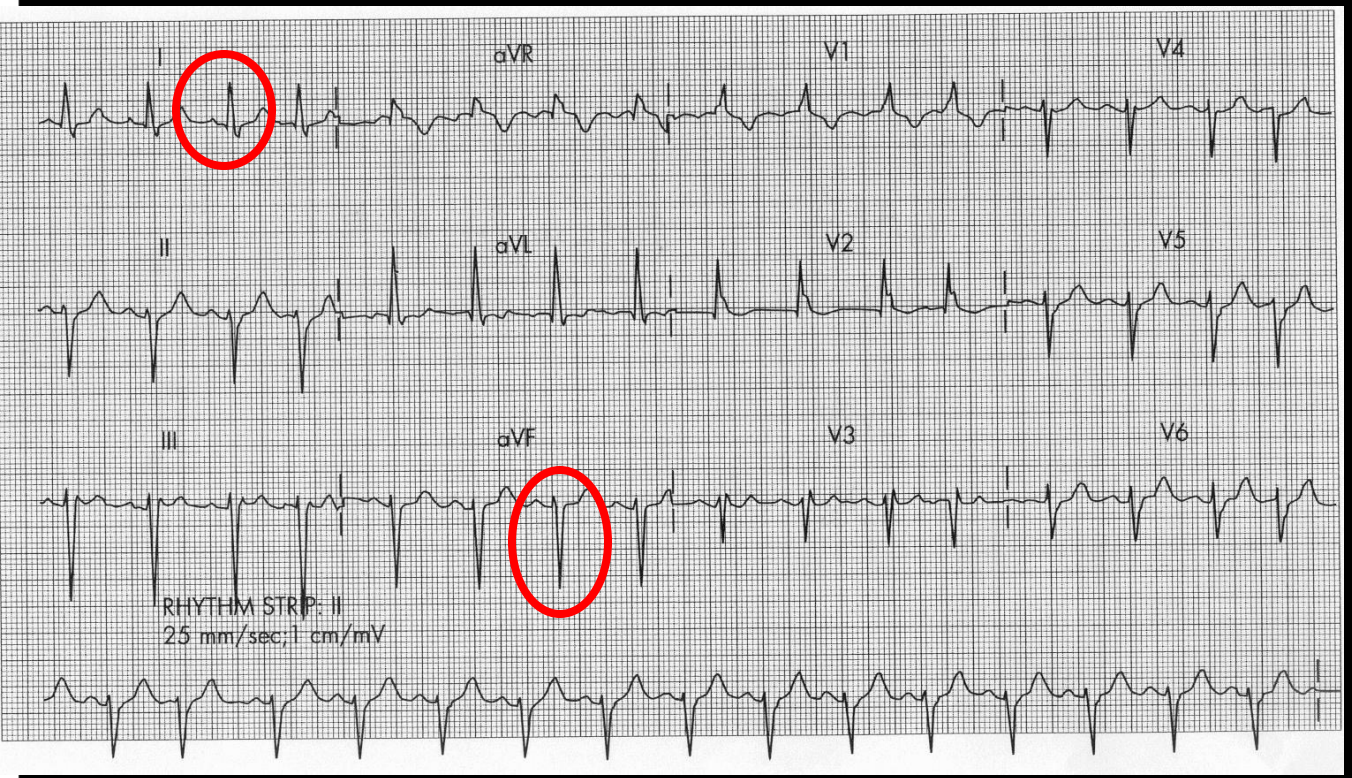
Normal, Right or Left Axis?



Left

Assessing the Axis

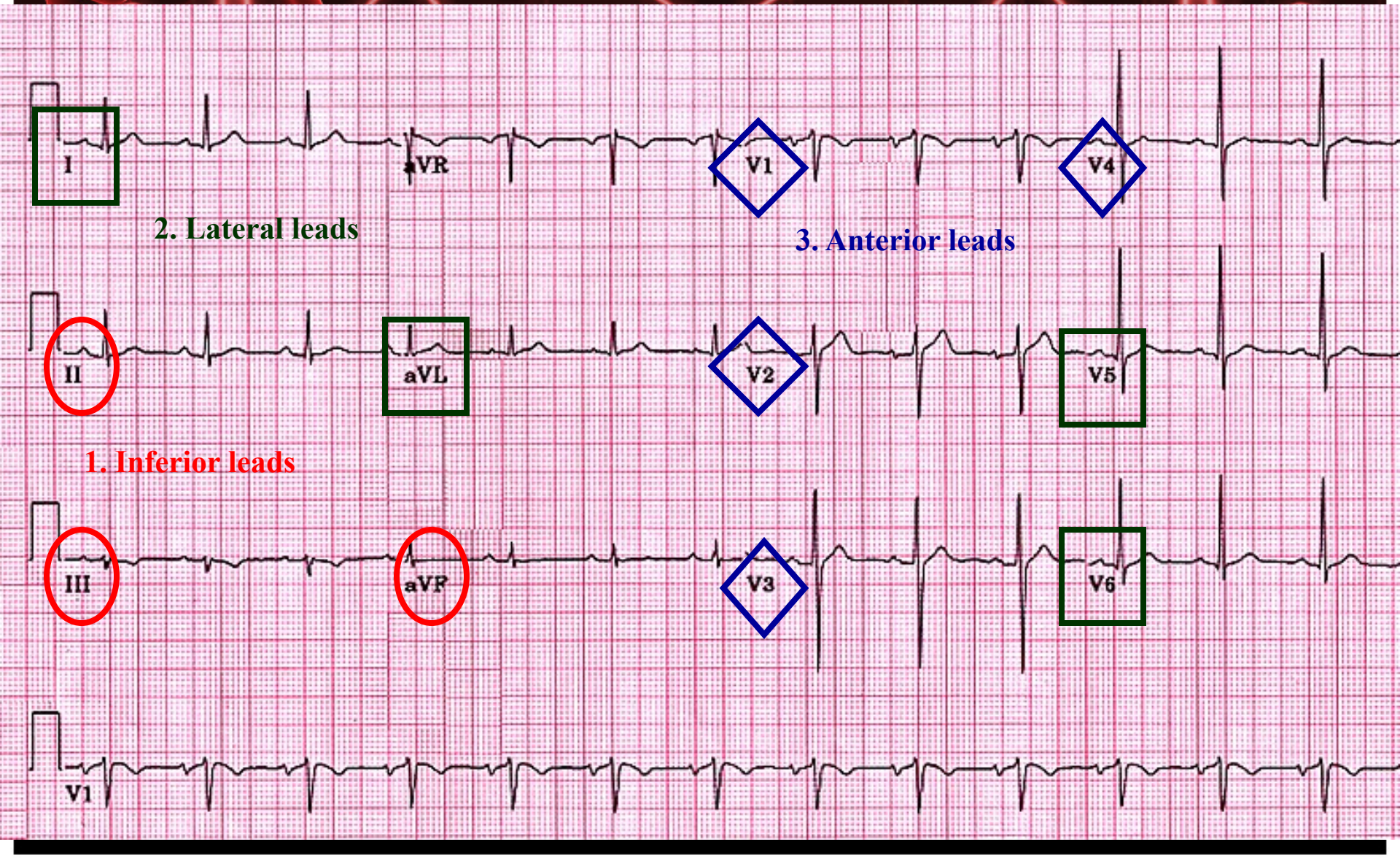
Is the QRS axis normal in this EKG?

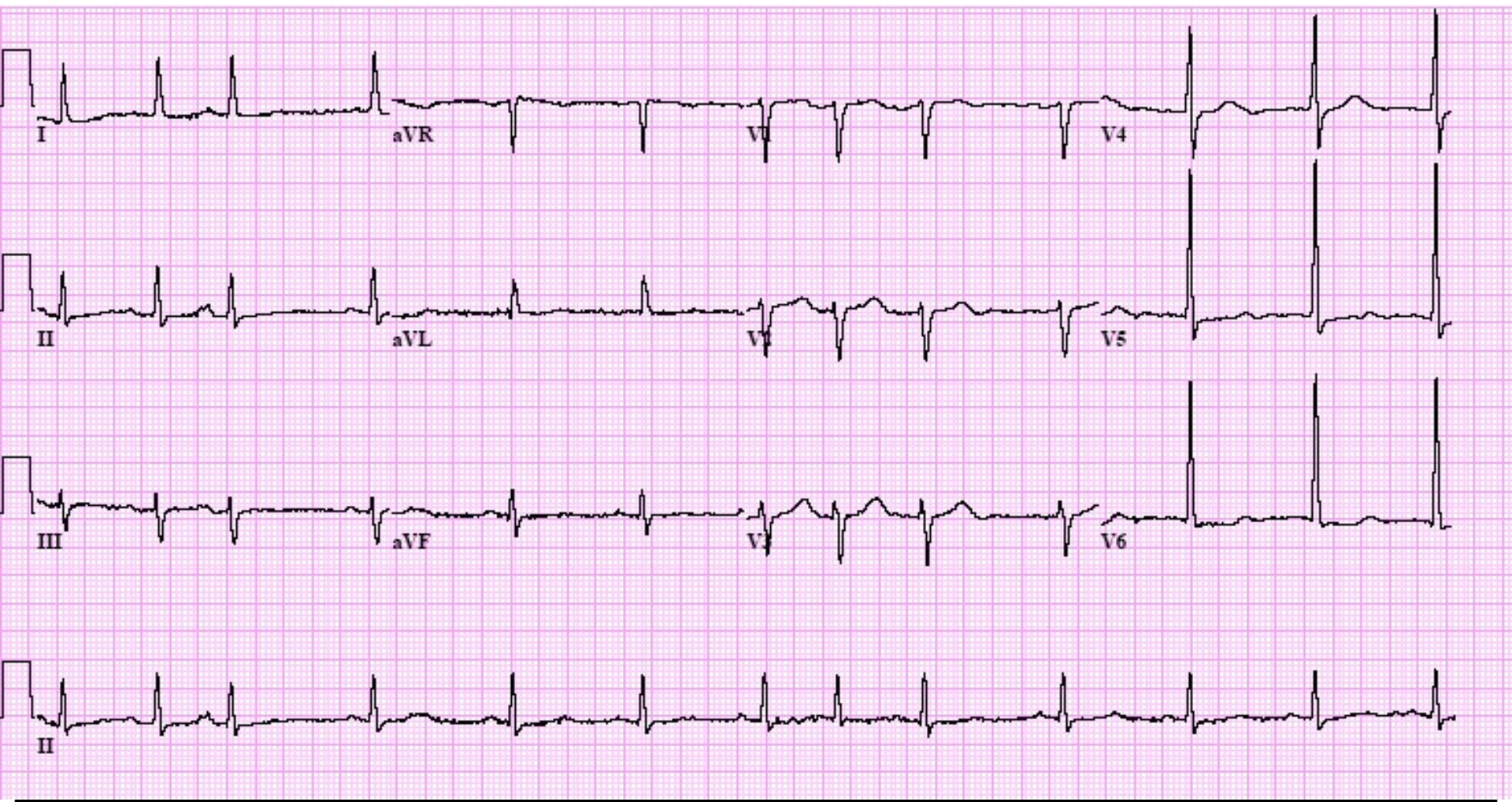


No, there is left axis deviation.

The QRS is positive in I and negative in aVF.

Grouped lead analysis





Rate_Variable___ Rhythm Regular? _Irregular__ P-Waves Upright? _Yes____

Significant Q-Waves?_No_ Any ectopic beats?_Yes, but caused by the rhythm__

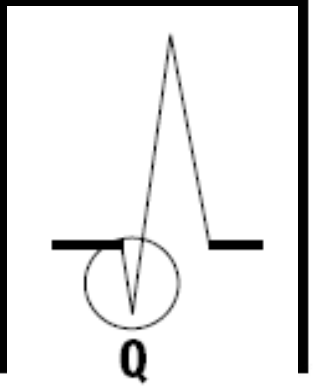
Rhythm is __Sinus Arrhythmia_____

-Waves

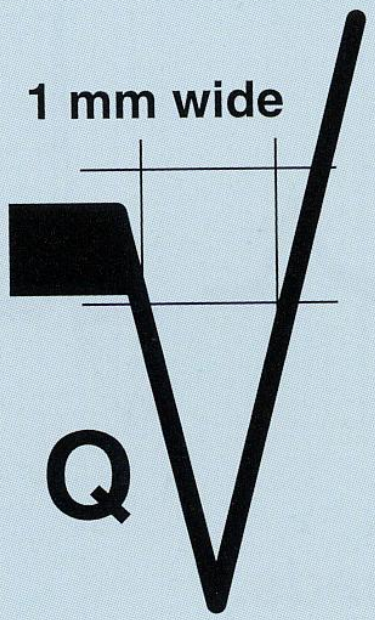
Q wave = Necrosis (significant Q's only)

- Significant Q wave is one millimeter (one small square) wide, which is .04 sec. in duration…
- … or is a Q wave 1/3 the amplitude (or more) of the QRS complex.
- Note those leads (omit AVR) where significant Q's are present

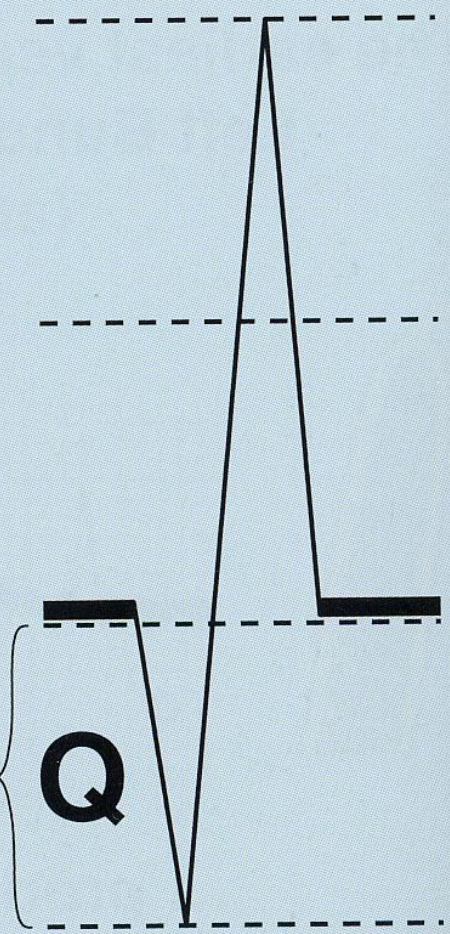
  * A Q wave in lead III alone is not diagnostic of infarction, even if it is otherwise "significant" in size and width. Qs in III are ignored unless other abnormalities are seen b/c they usually represent…..
- Old infarcts: significant Q waves (like infarct damage) remain for a lifetime.



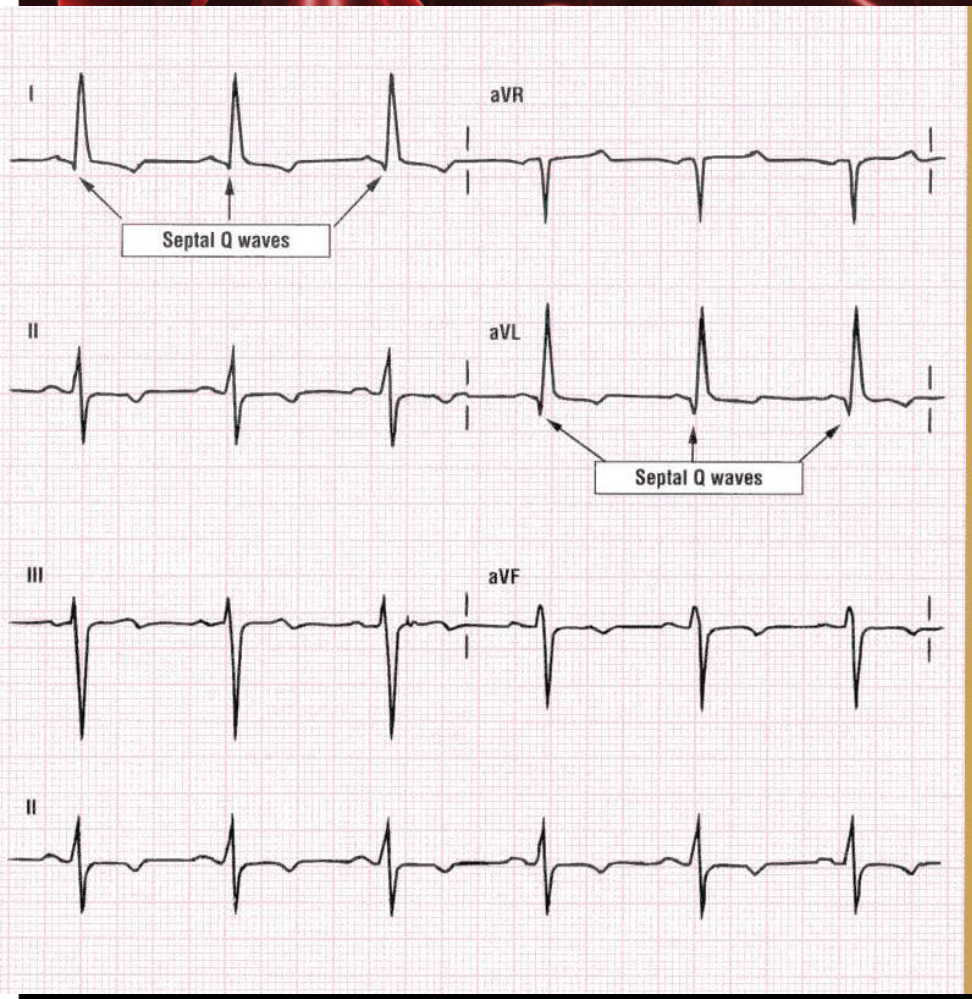
Significant Q waves

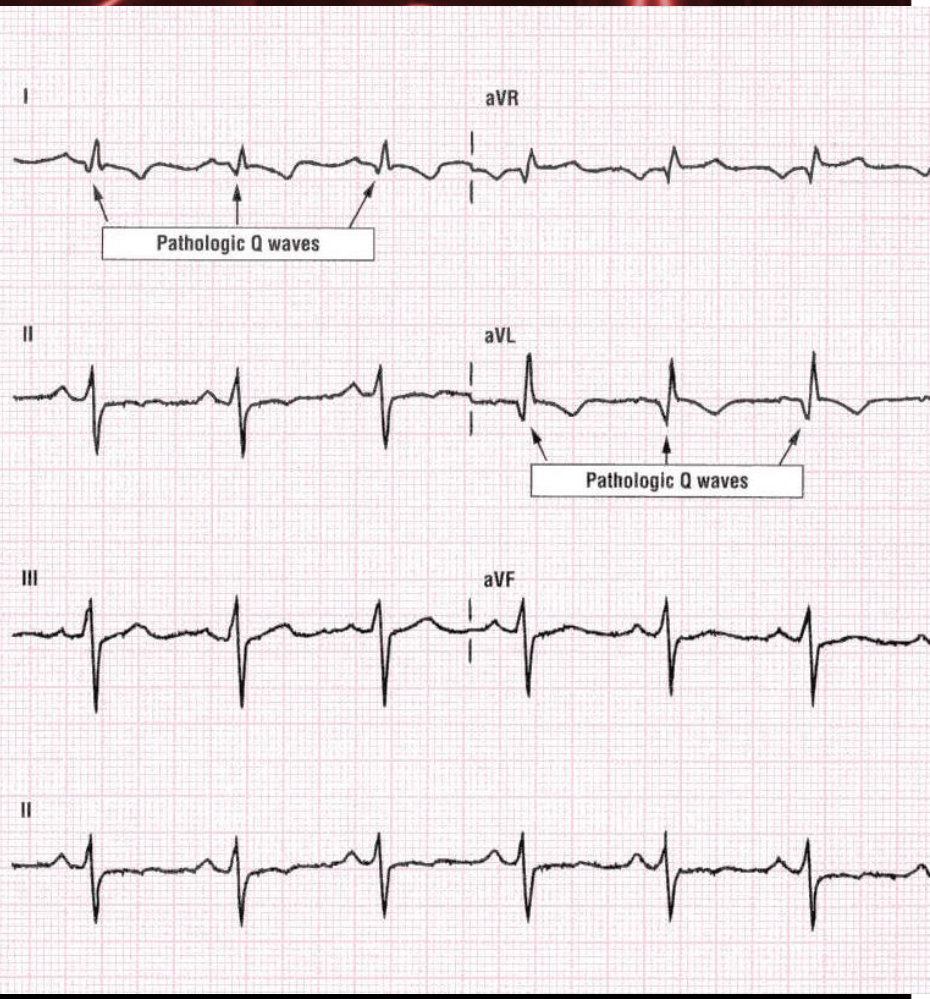


or

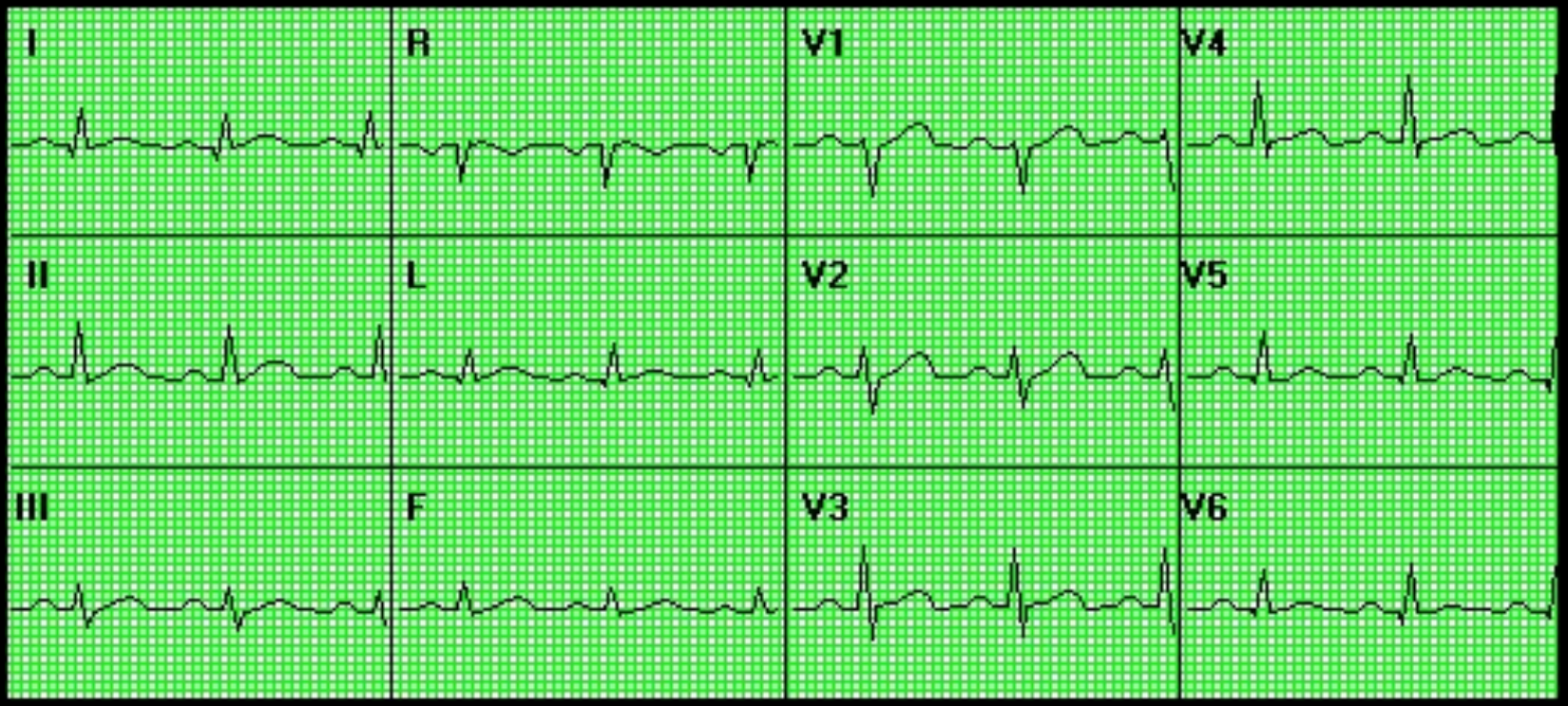


Normal vs. Abnormal Q-waves

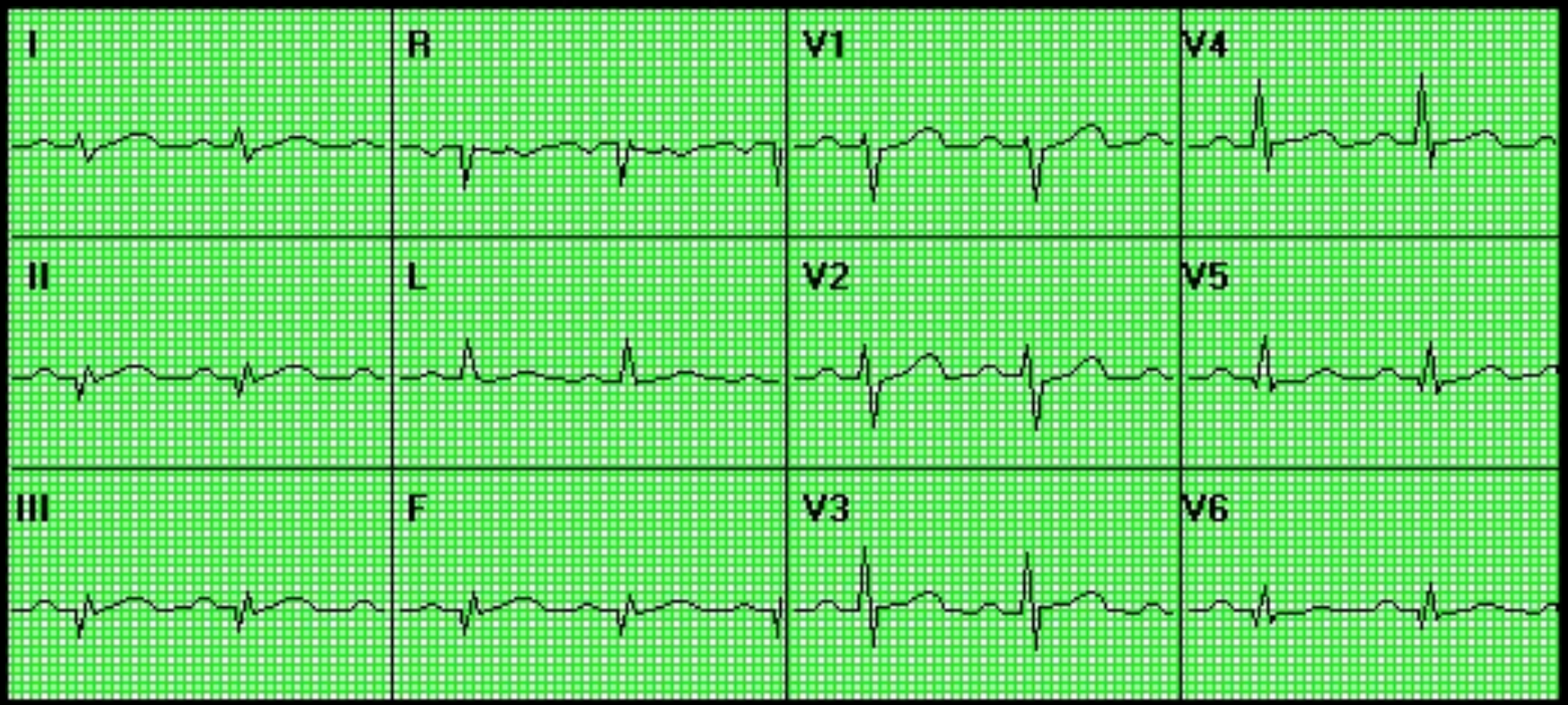




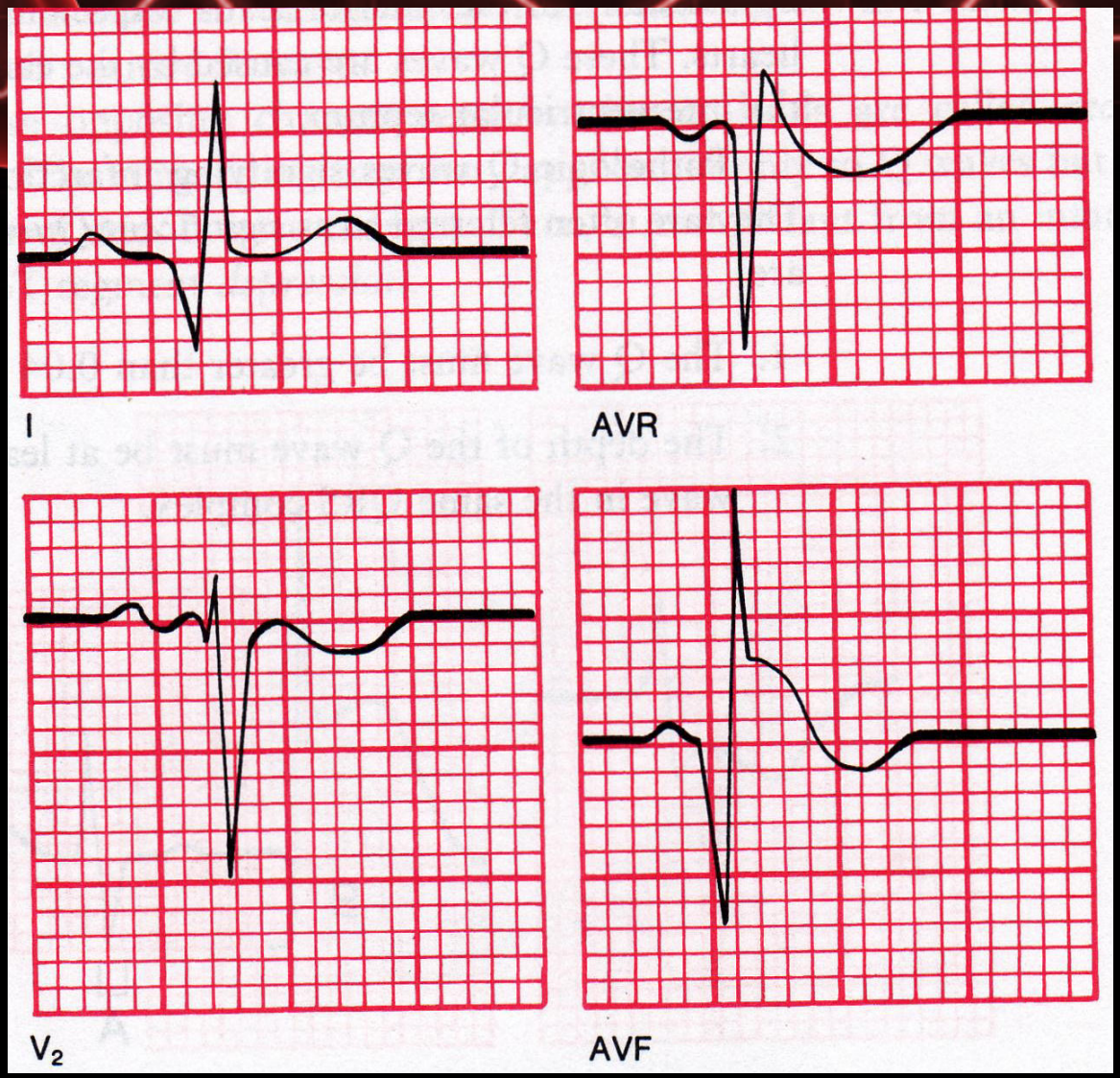
EKG showing normal Qs in I, AVL, V5 and V6



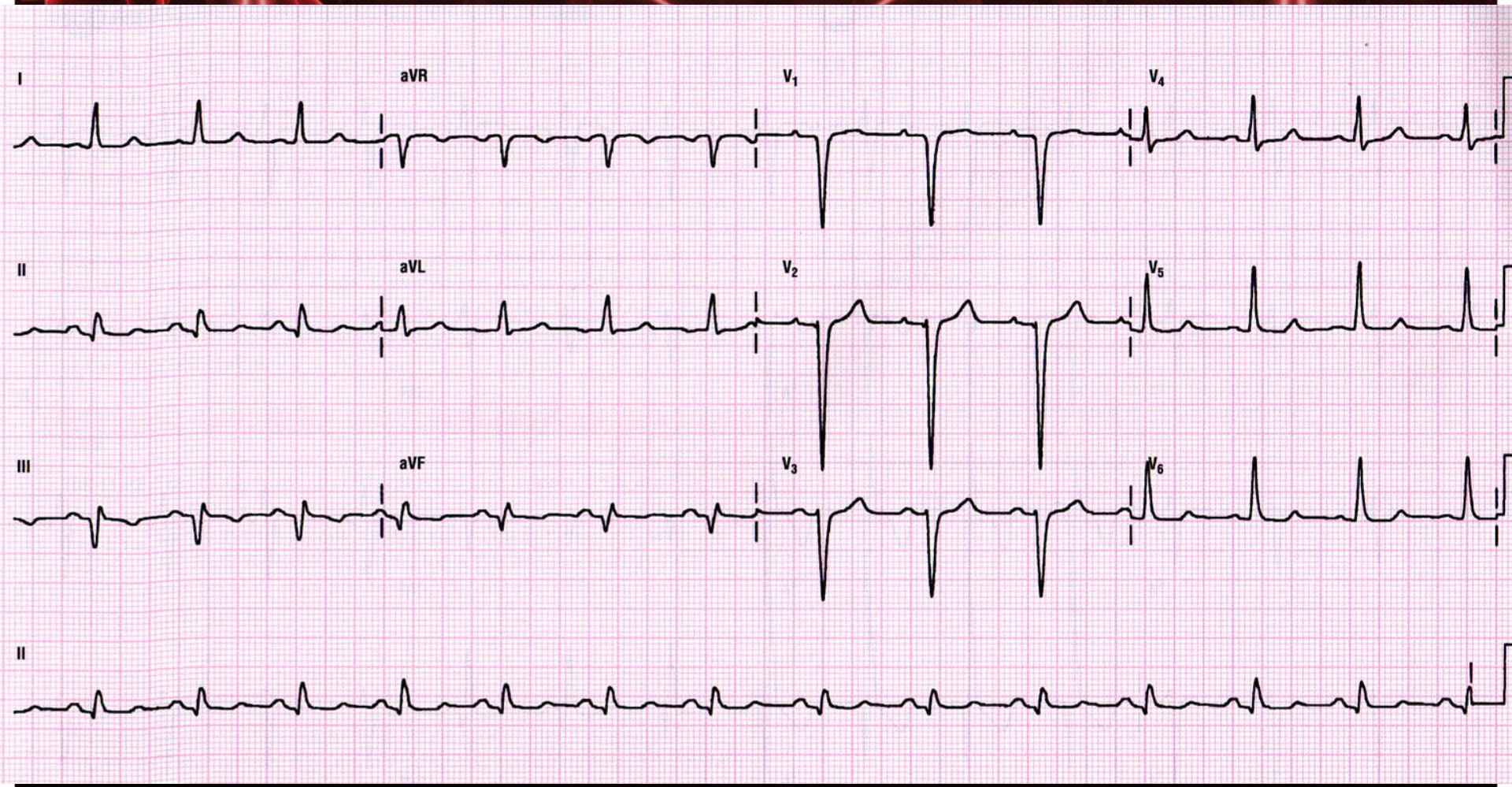
Q waves of old infarction in II, III, and AVF



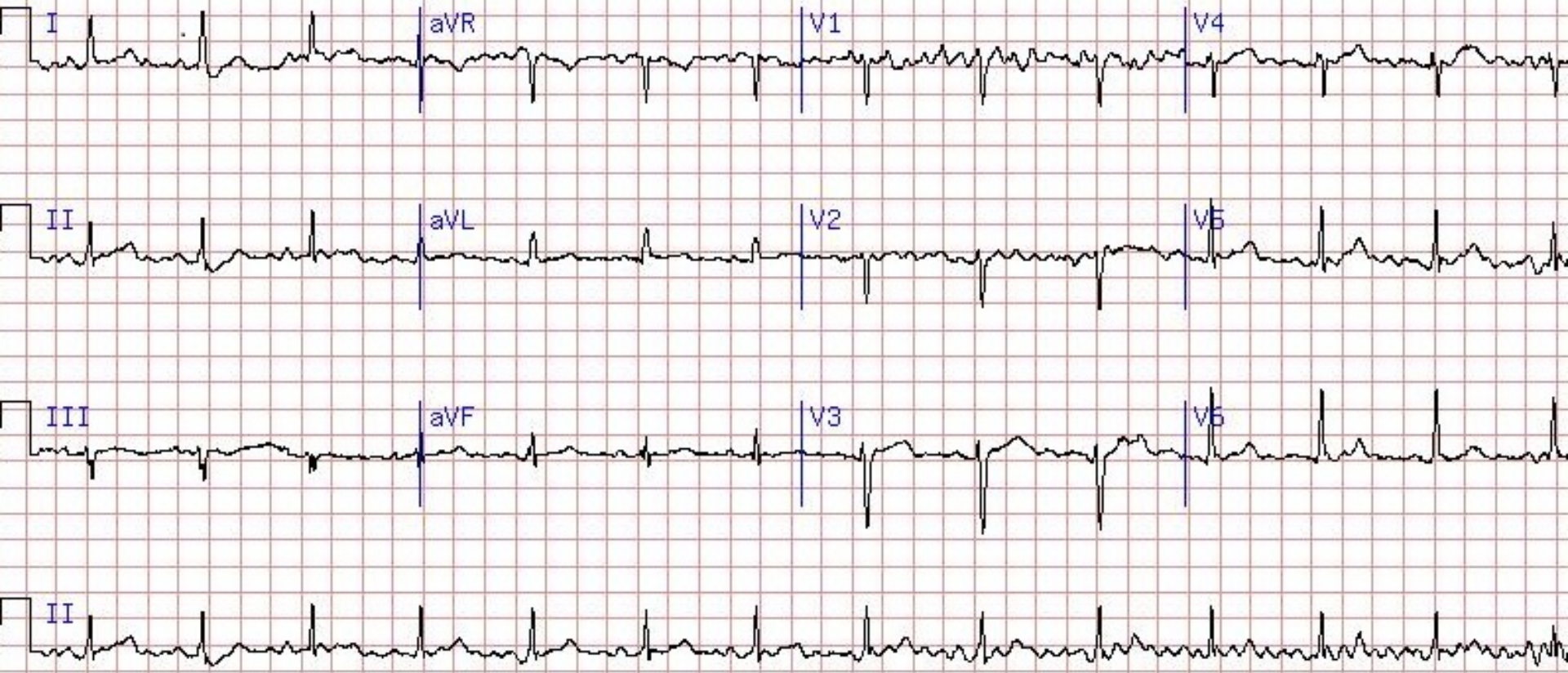
Significant or Not?



Any Significant Q's?



Yes in leads III & AVF only



Rate Variable__ Rhythm Regular? __NO_____

P-Waves Upright? _Indiscernible_ Significant Q-Waves? No_____

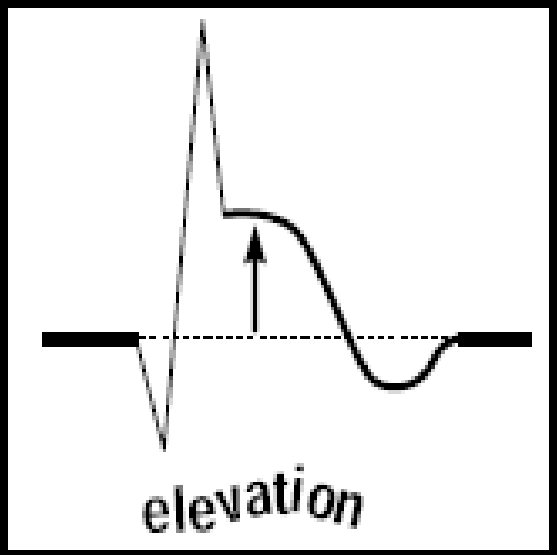
Any ectopic beats? No_____

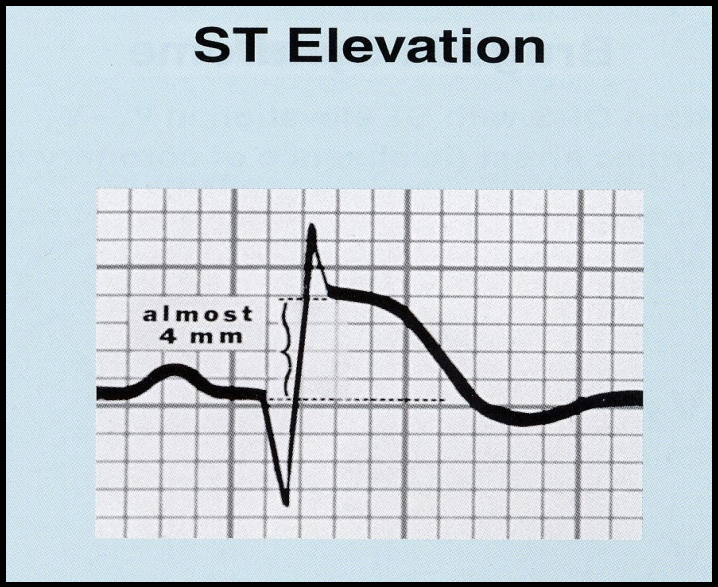
Rhythm Diagnosis is _A-Fib_____


ST - Elevation

ST Elevation

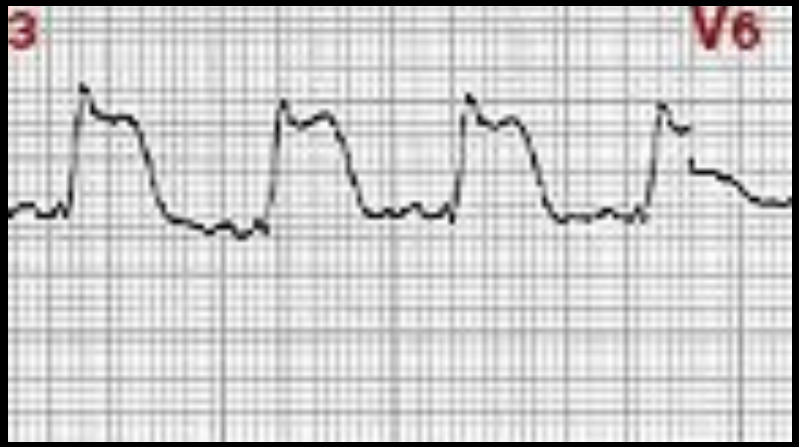
- Signifies an acute process, ST segment returns to baseline with time.
- ST elevation associated with significant Q waves indicates an acute (or recent) infarct.

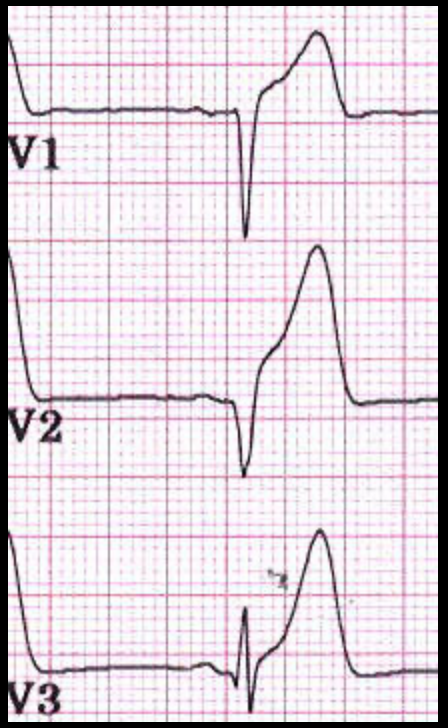




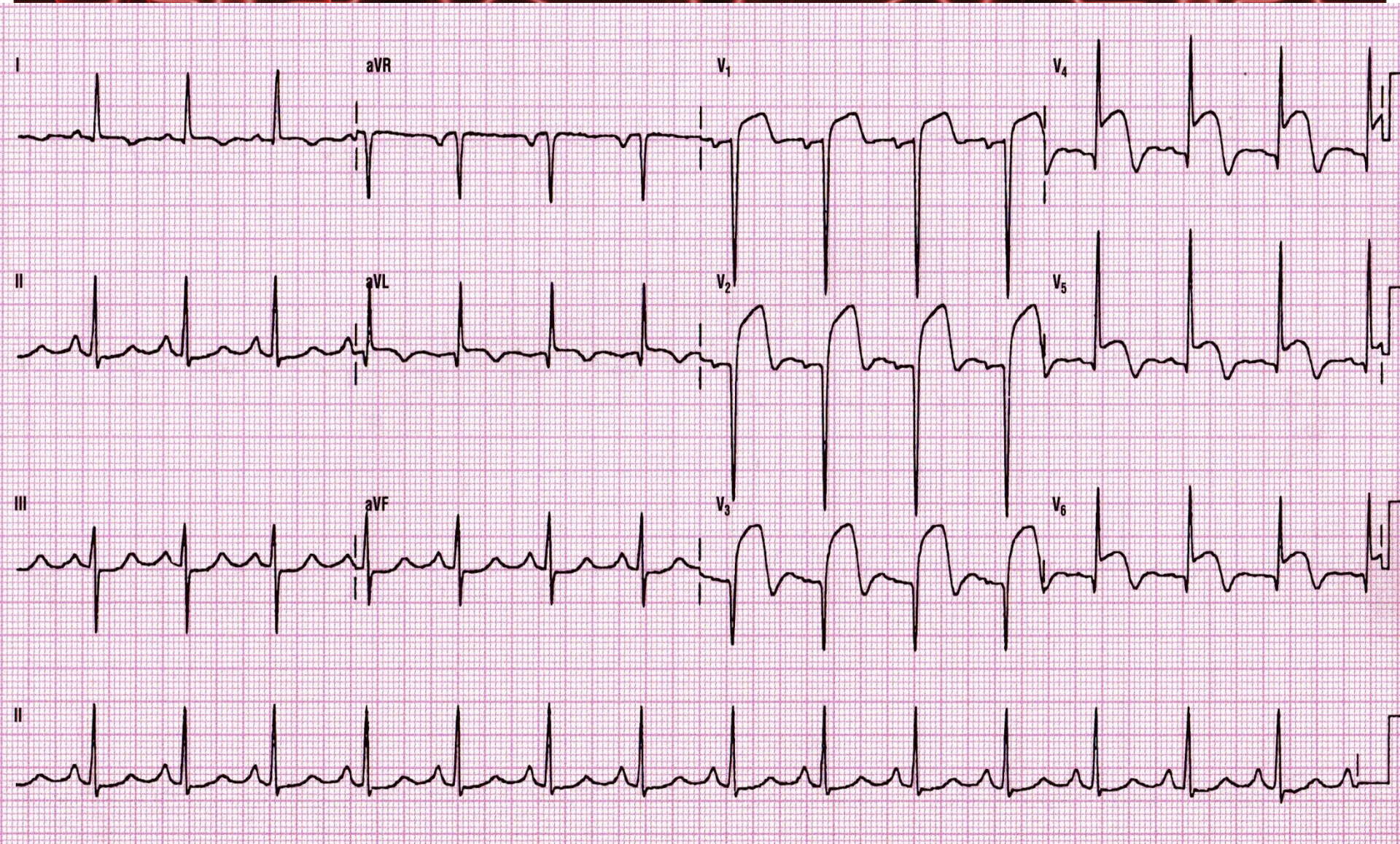
Causes of ST Elevation

- Infarction
  Vasospastic angina
  Pericarditis
  Early repolarization

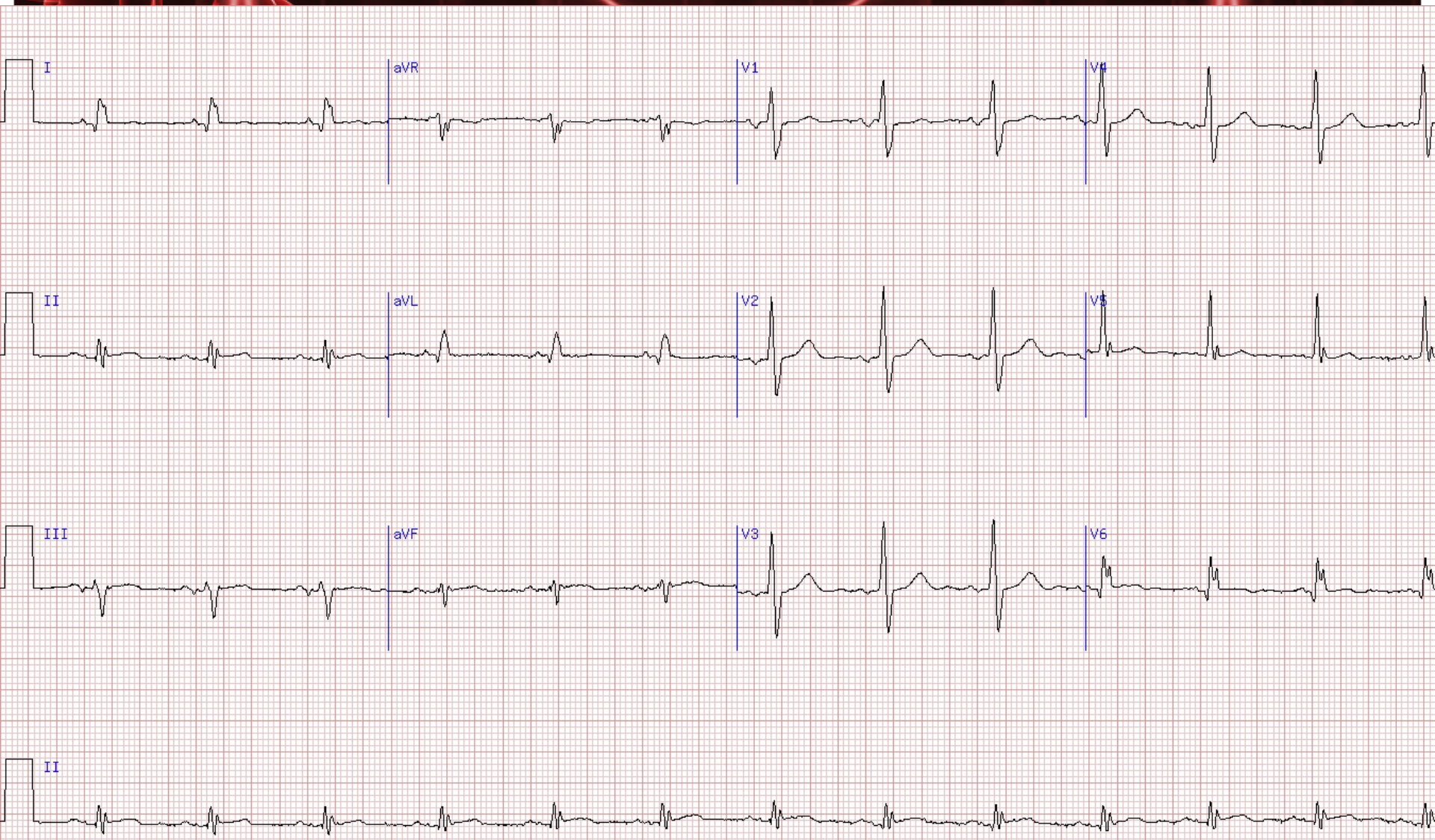




Is this ST Elevation?



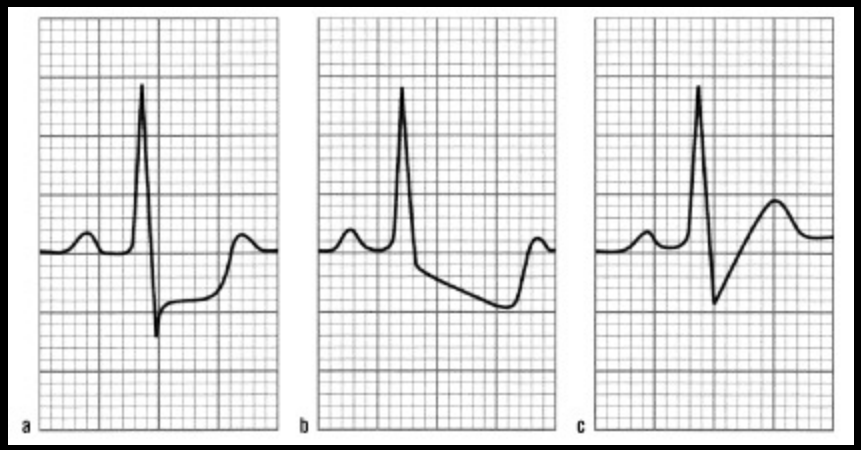
What about this?




ST Depression

ST Depression Significance

- ST segment depression is considered significant if the ST segment is at least two boxes below baseline.
- With infarction, the location of the ischemia is reflected in the leads in which the ST depression occurs.

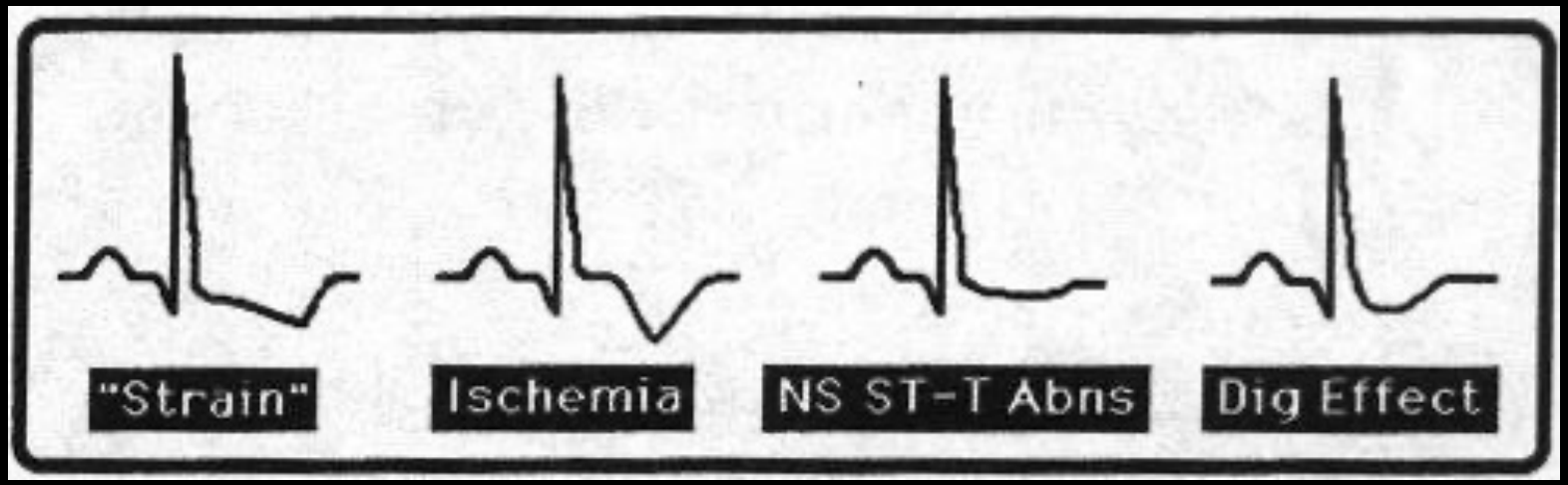


Common Causes of ST Segment Depression

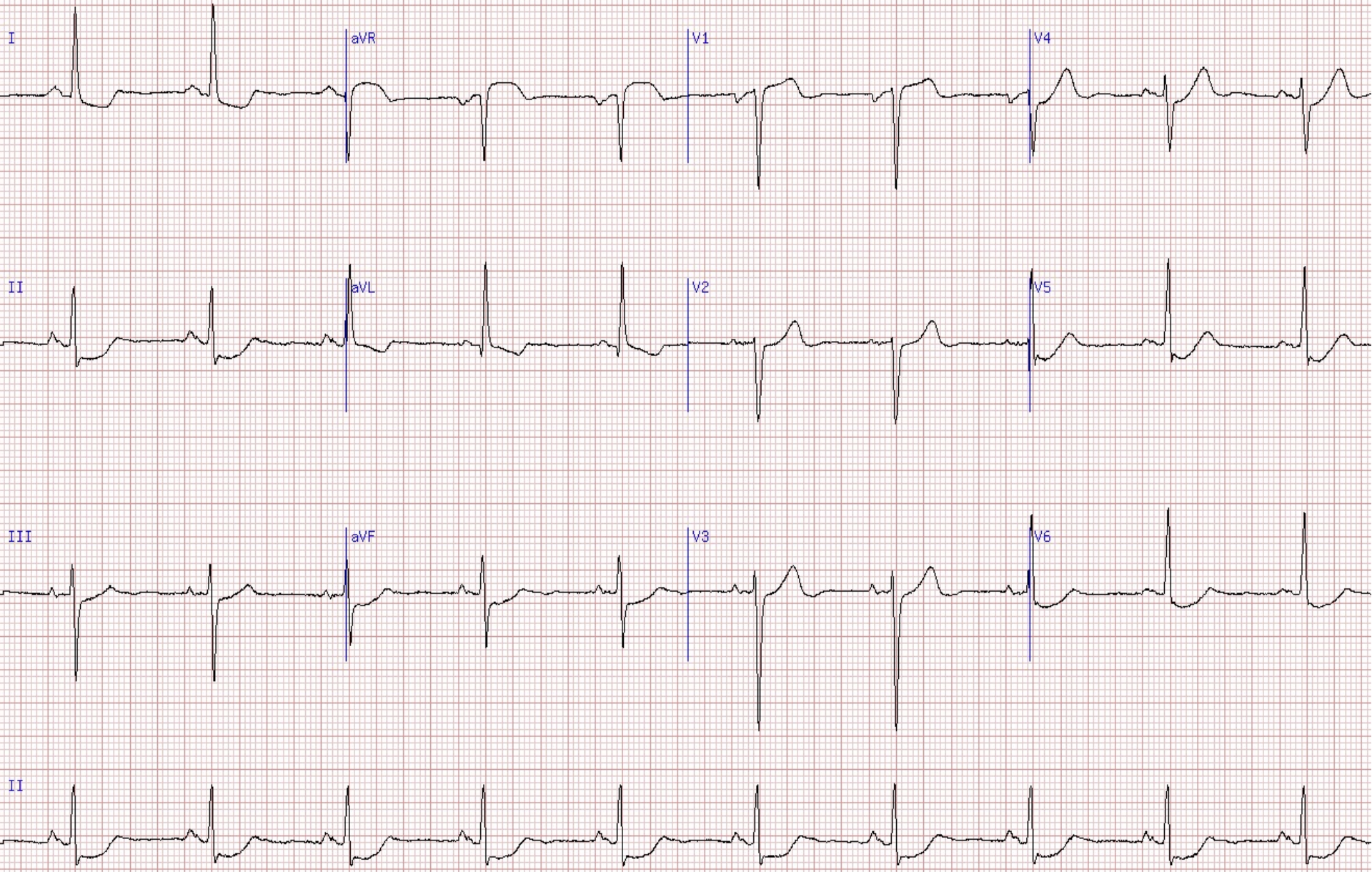
- Ischemia
- "Strain" in LVH
- Digitalis effect
- Bundle branch block
- Hypokalemia/Hypomagnesemia
- Reciprocal ST elevation
- Any combination of the above

Digoxin Toxicity

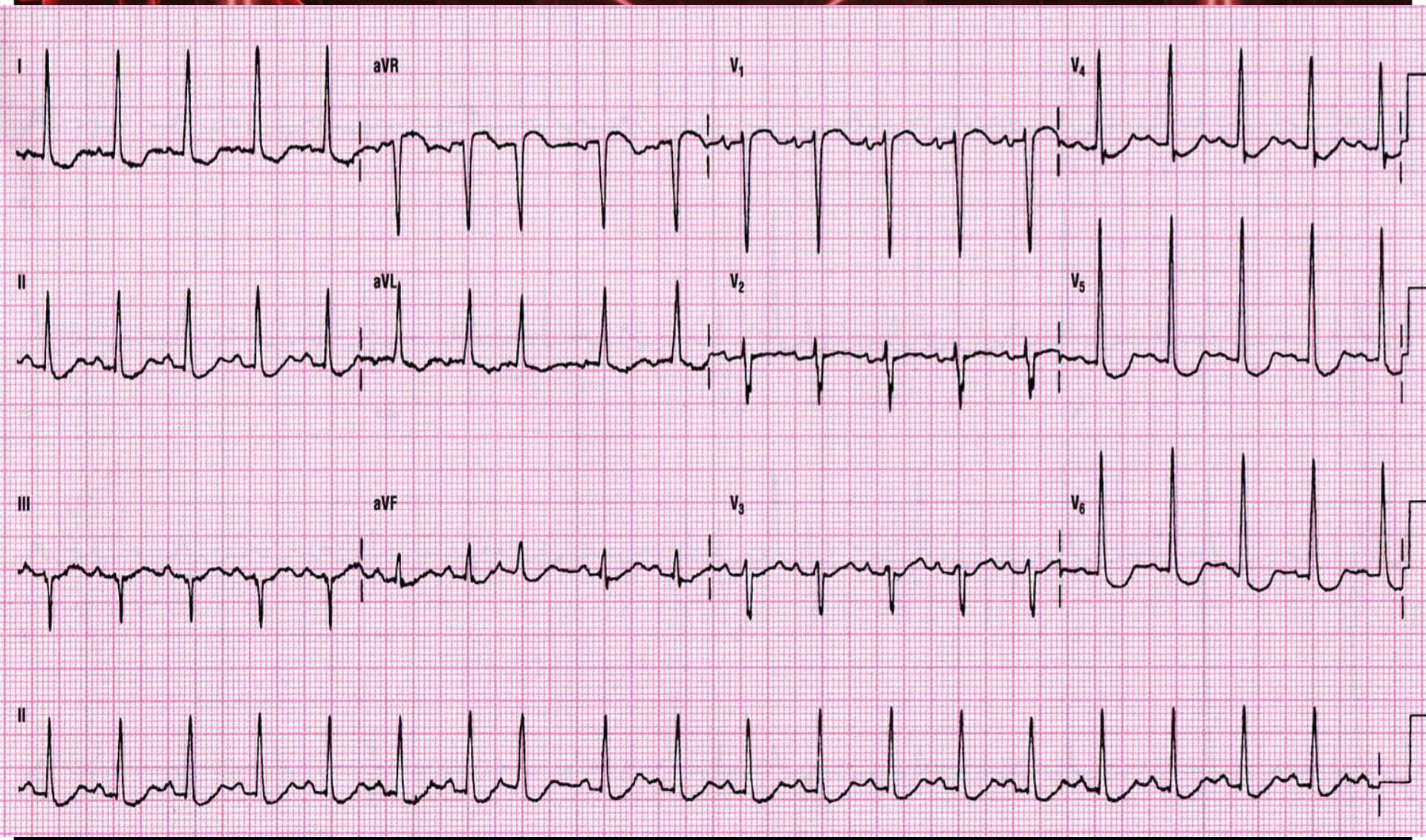
- Digoxin ("**Dig effect**") may produce either ST "scooping" or a "strain"-like pattern or no change at all.
- Can be seen in levels greater than 2.5mg/ml



Where's the Depression?



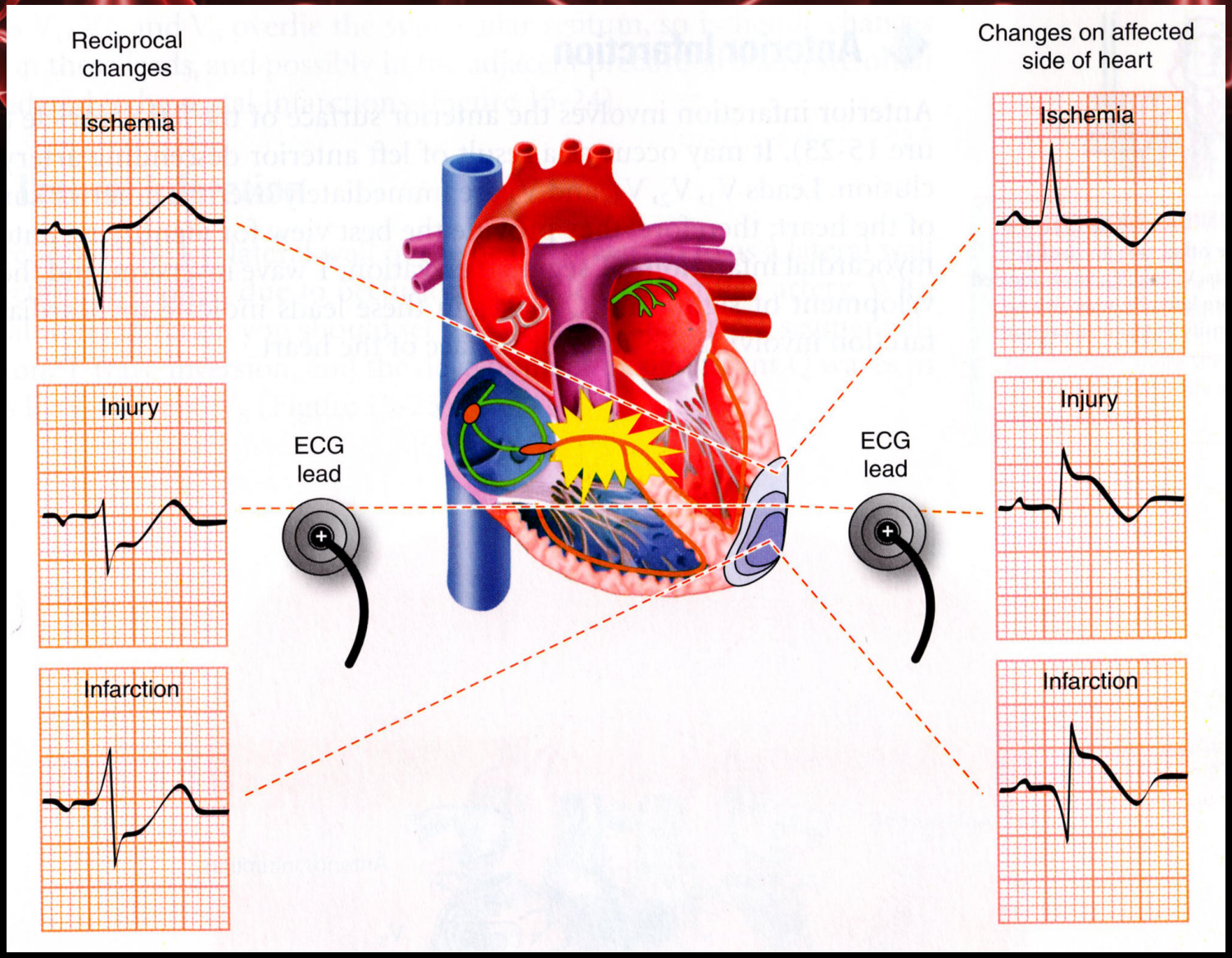
Dig Toxicity



What is "Electrical Reciprocity"?

- Electrical Reciprocity is like a mirror image. On the EKG, this mean that ST elevations in one group will show up as ST depressions in another.
- When you see this, it is not considered ischemia.
- For example, a set of inferior ST elevations in II, III and AVF can produce lateral ST depressions in I, AVL, V5 and V6. You'd think that this would mean lateral ischemia, but no – it's a reflection, bouncing electrically across the heart from the inferior injury, showing up in reverse.

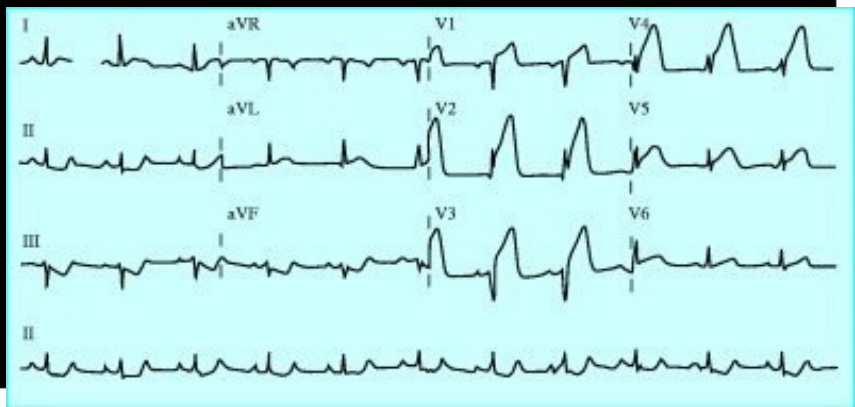
Reciprocal Changes



T Wave Categories

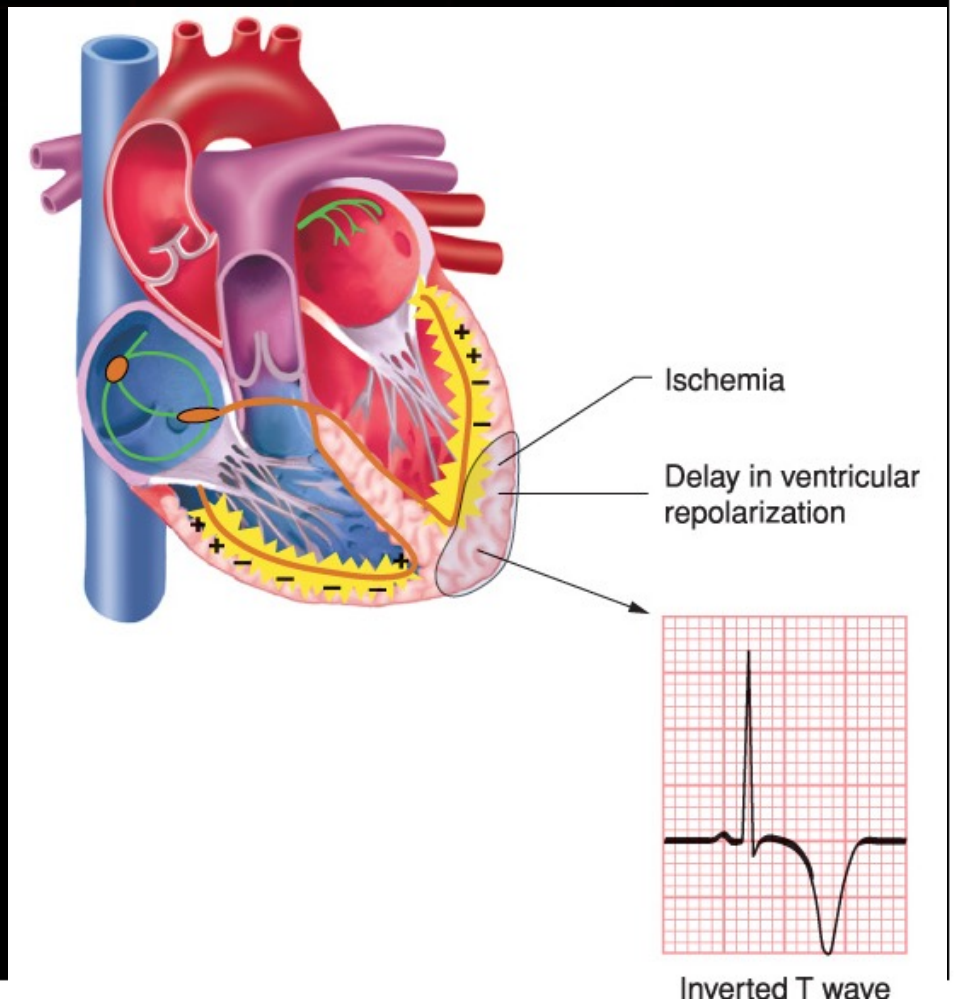
- Tall, peaked = hyperkalemia if generalized
  infarction if localized
- Inverted = evolving infarction
  chronic pericarditis
  conduction block
  ventricular hypertrophy
  acute cerebral disease
  other cardiac disease
- Flattened = nonspecific

Hyperacute T-wave

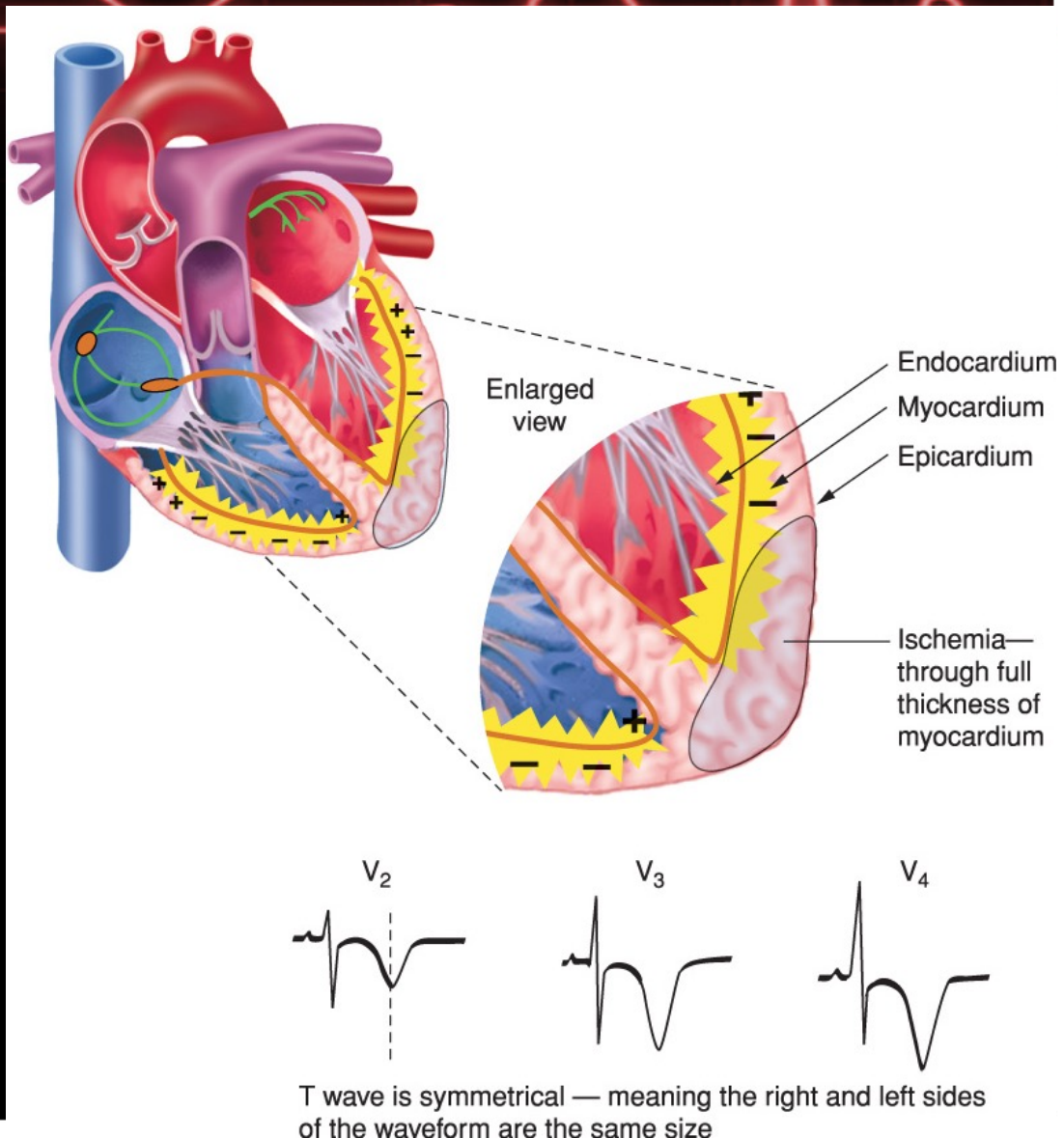


T Wave Inversion

- Occurs because ischemic tissue does not repolarize normally

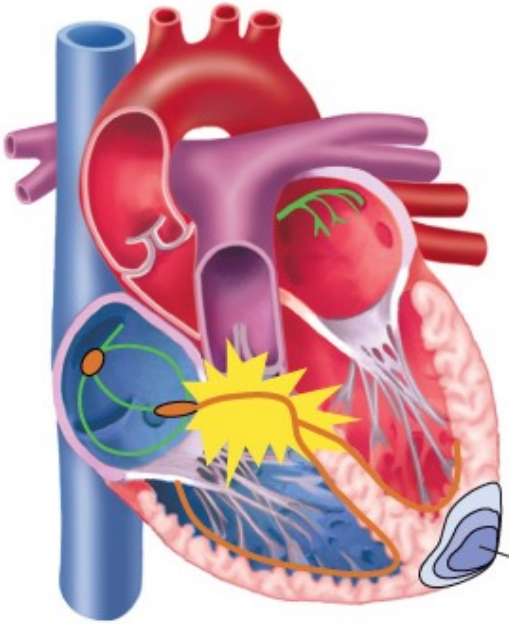


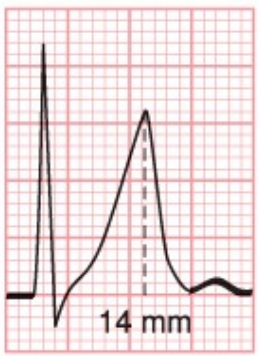
T Wave Inversion



Peaked T Waves

- May be seen in early stages of acute myocardial infarction
- Within a short time (two hours) T waves invert





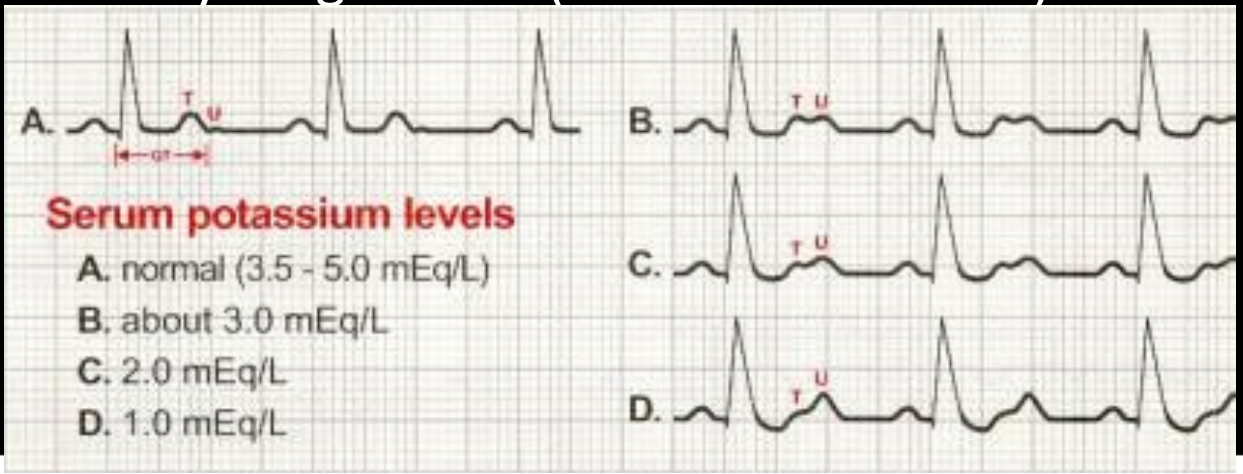
T wave is more than 6 mm high in limb leads and 12 mm in precordial leads

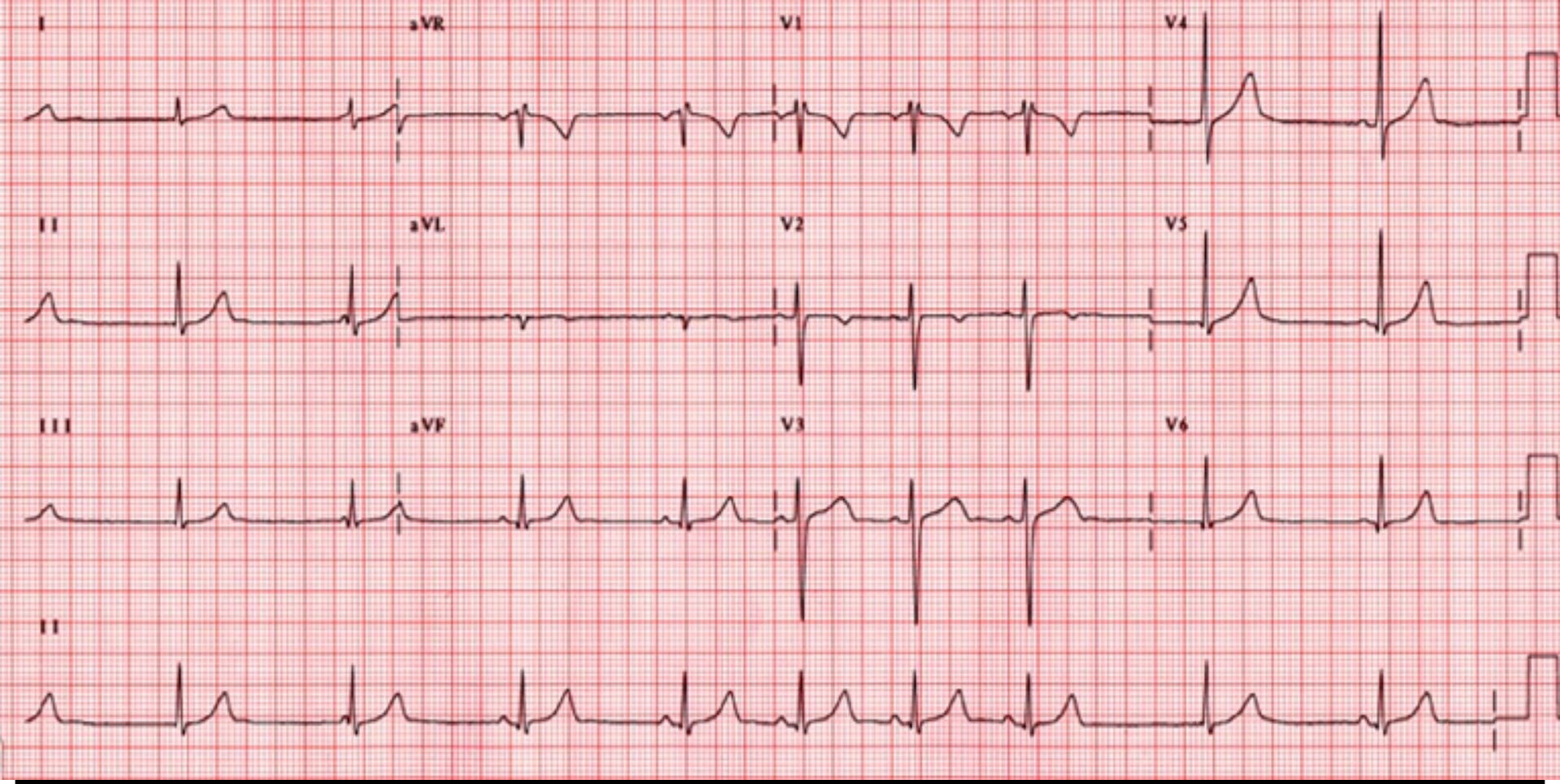
or

T wave is more than two-thirds the height of the R wave

Flattened T-Waves

- Flat T waves can be seen in many conditions, including ischemia, evolving infarction, and electrolyte abnormality (such as hypokalemia).
- In acute cerebral disease, such as intracranial hemorrhage, elongated or bizarre T waves may be seen. These Ts are often biphasic or deeply and sharply inverted. The QT interval is often dramatically lengthened (0.5 to 0.7 seconds).





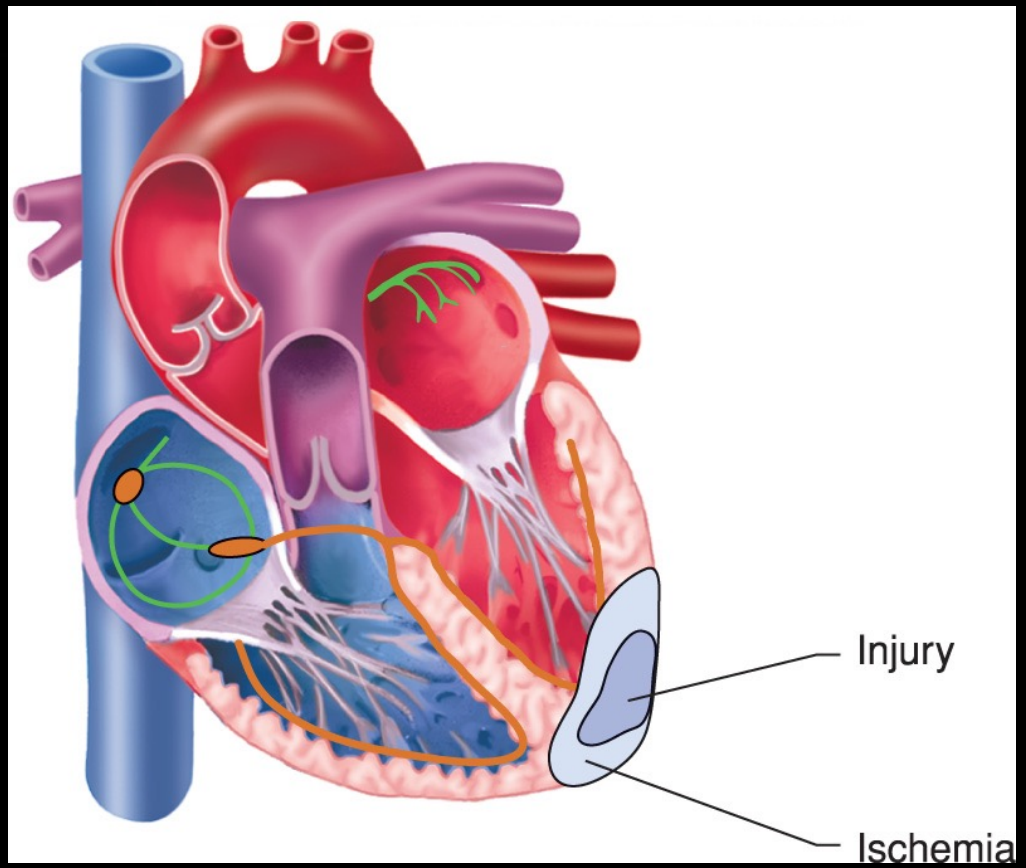
Rate_Variable_ Rhythm Regular? No P-Waves Upright? _Intermittanly__

Significant Q-Waves?_No_ Any ectopic beats? _No__

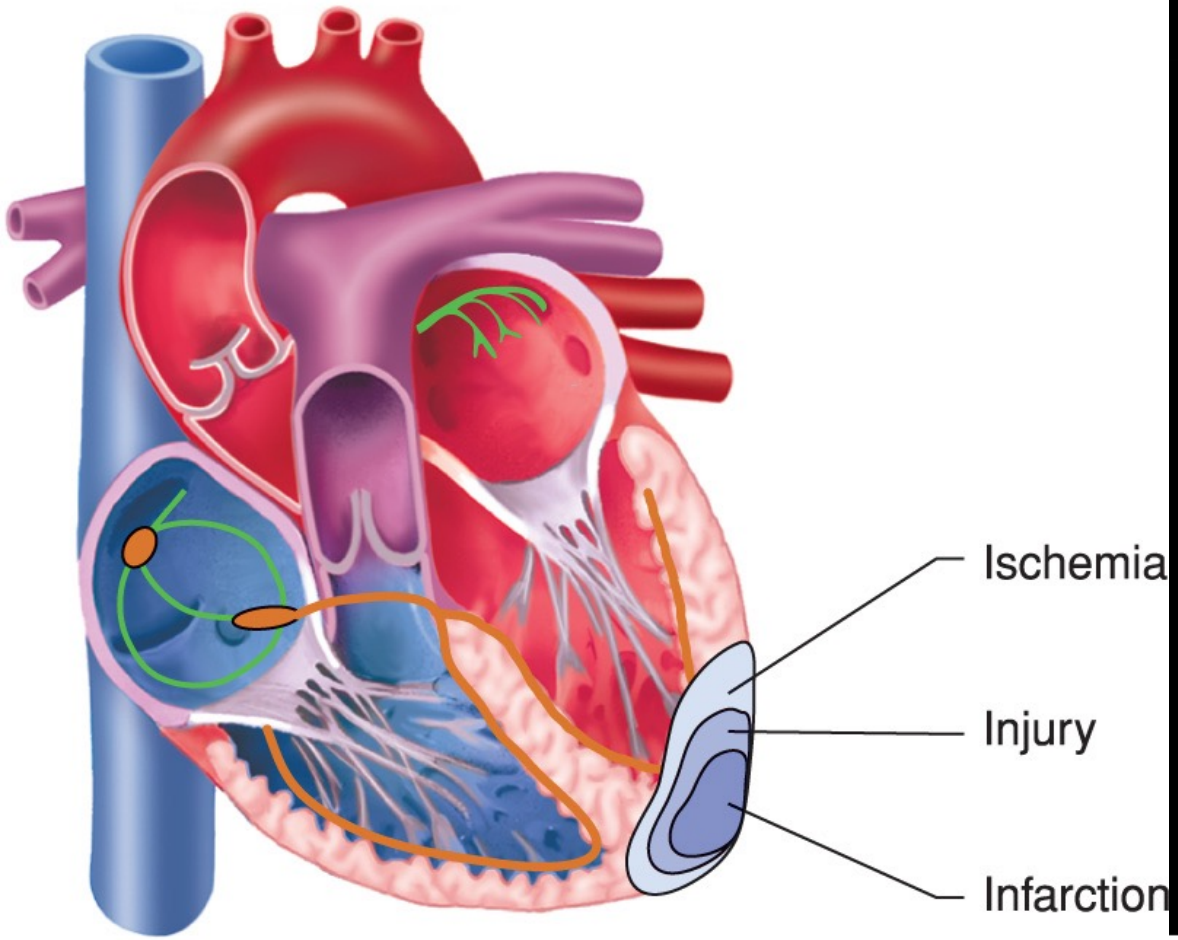
Rhythm Diagnosis is: Sinus Rhythm with Junctional Escape Rhythm Complexes

Myocardial Injury

- Results if ischemia progresses unresolved or untreated

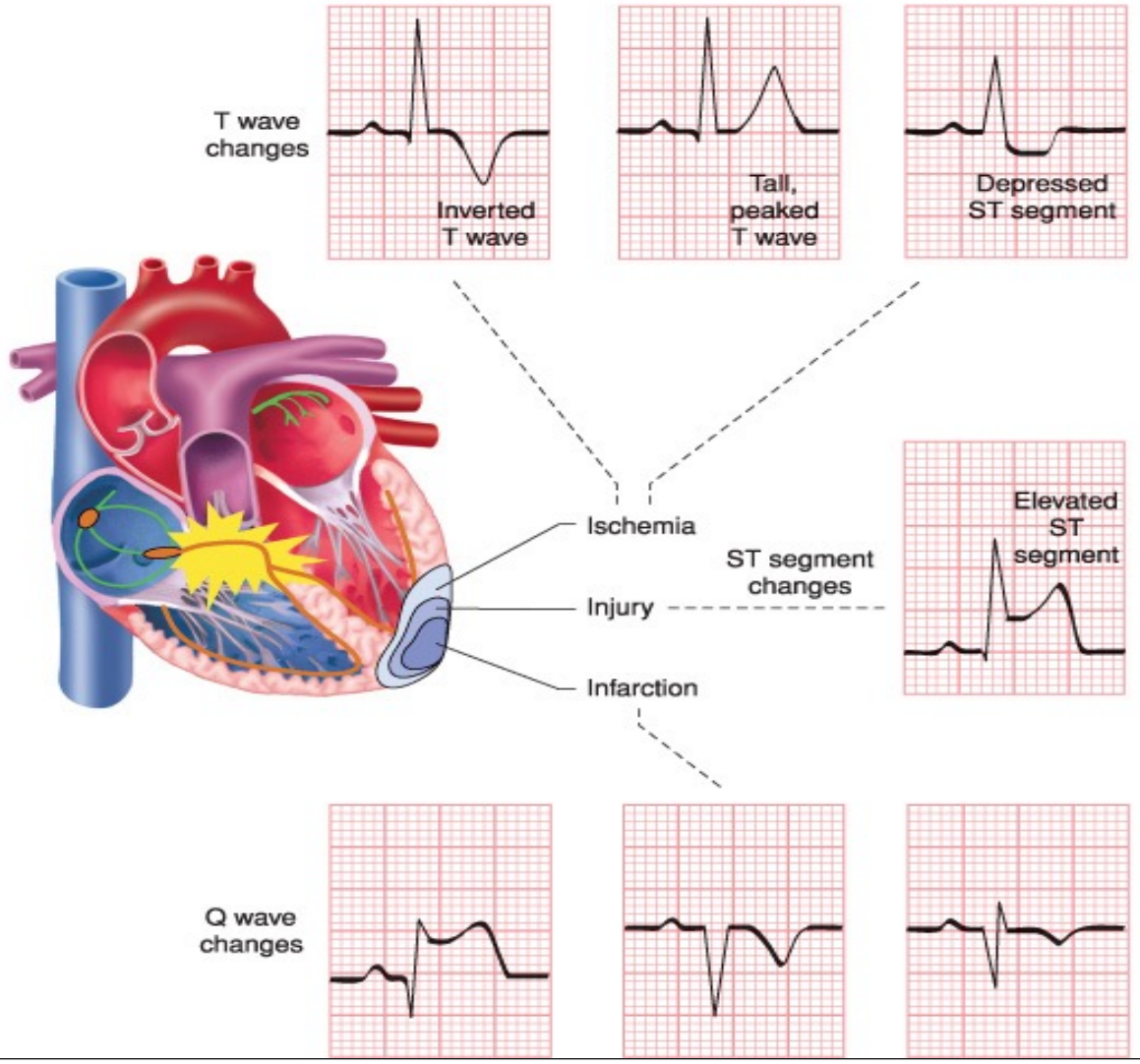


Myocardial Infarction

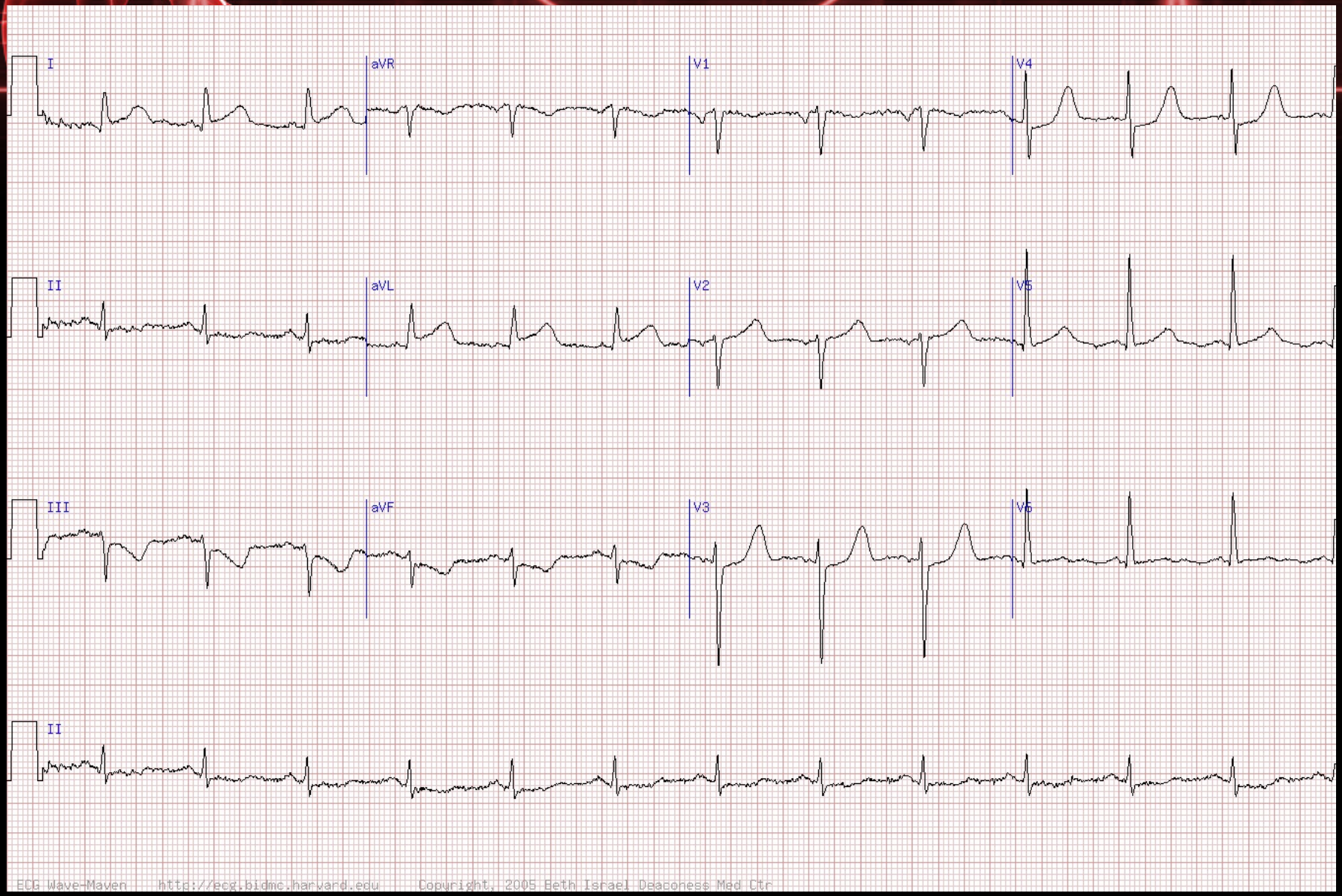


Death of myocardial cells

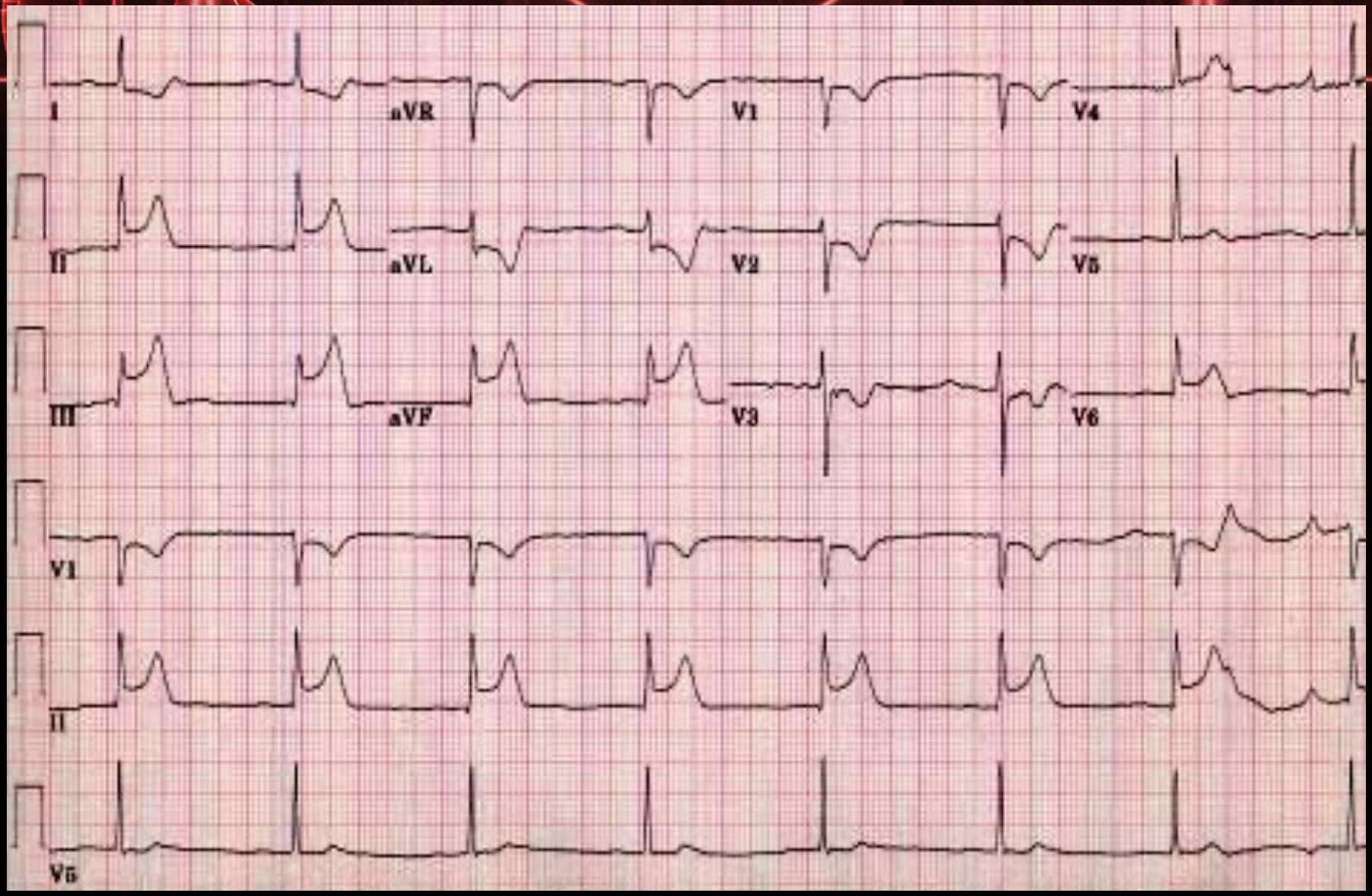
EKG Indicators



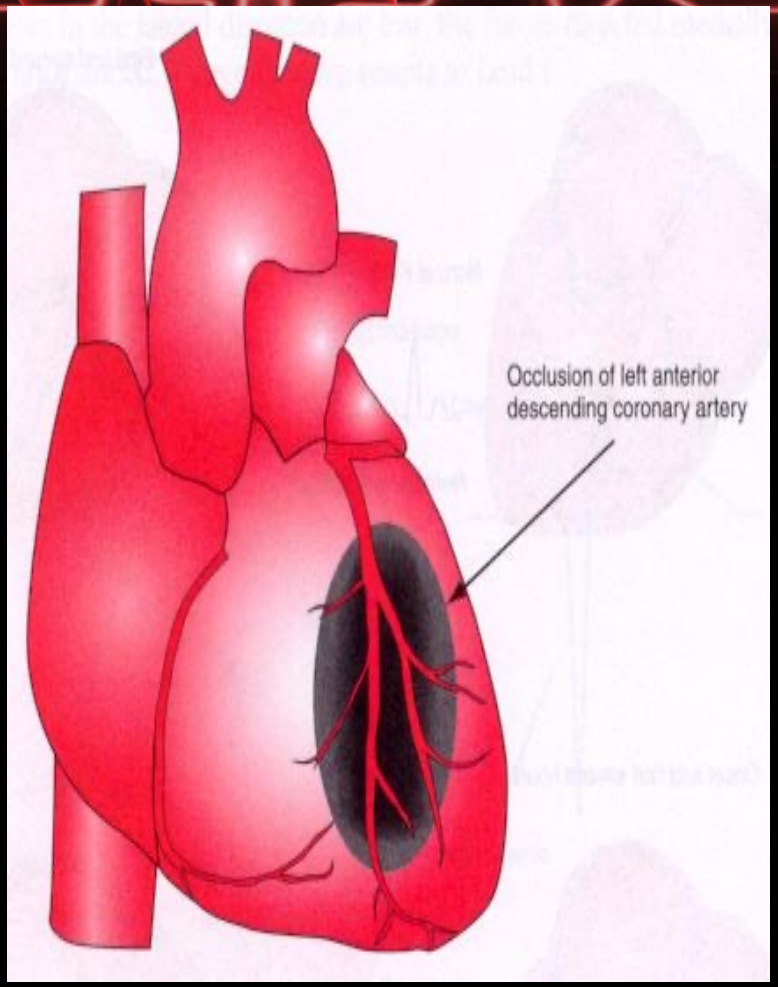
Lateral MI, Inferior Reciprocity

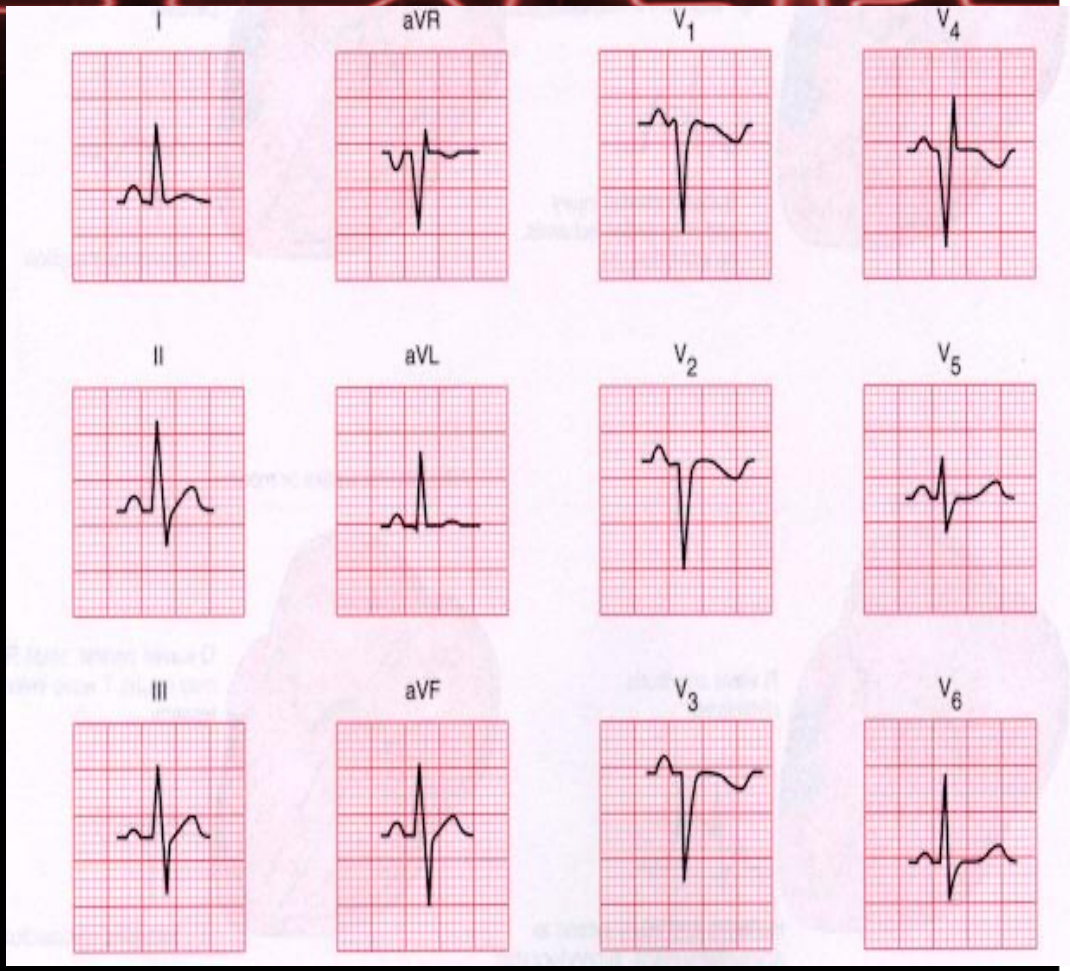


Inferior MI, Lateral Reciprocity

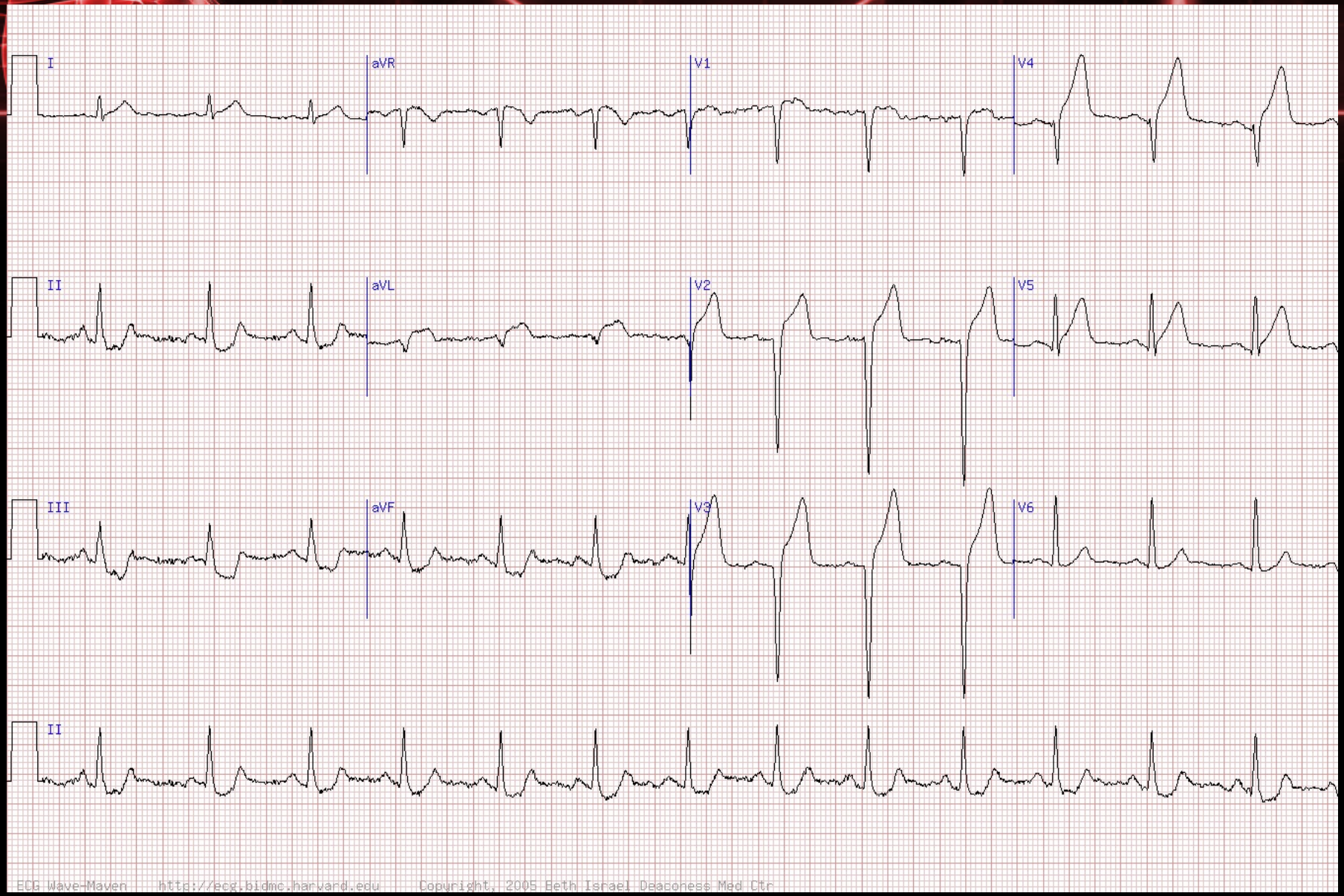


Anterior MI

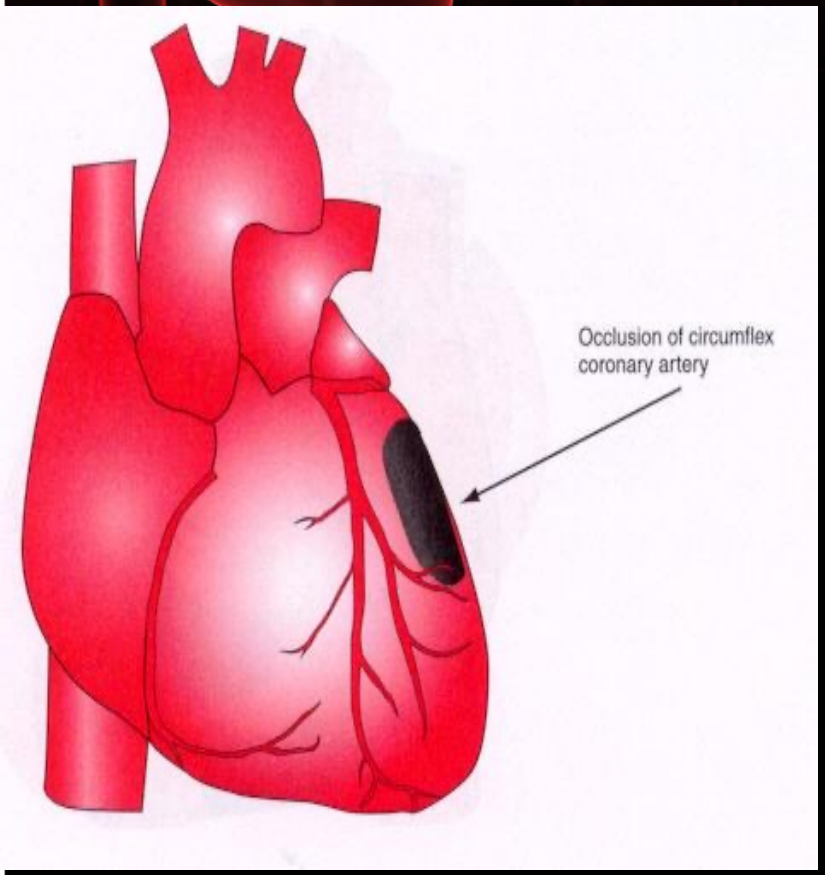

Occlusion of left anterior descending coronary artery

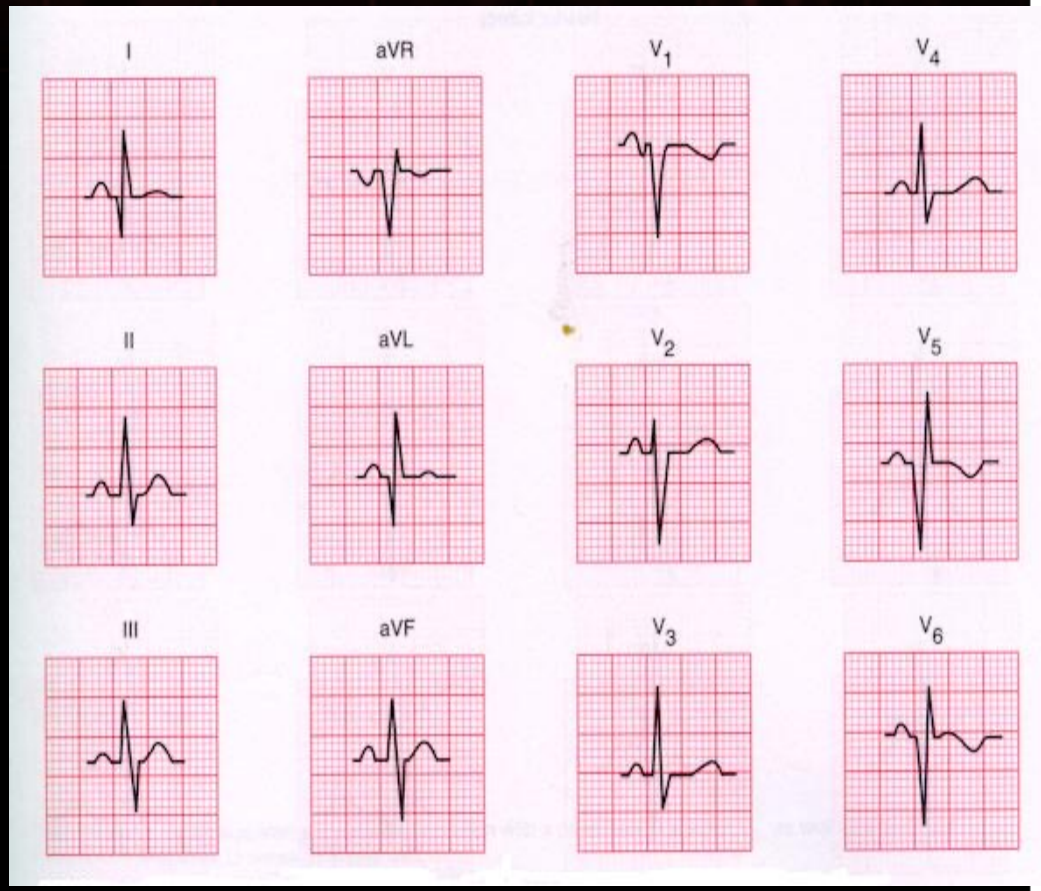

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Anterior Wall MI

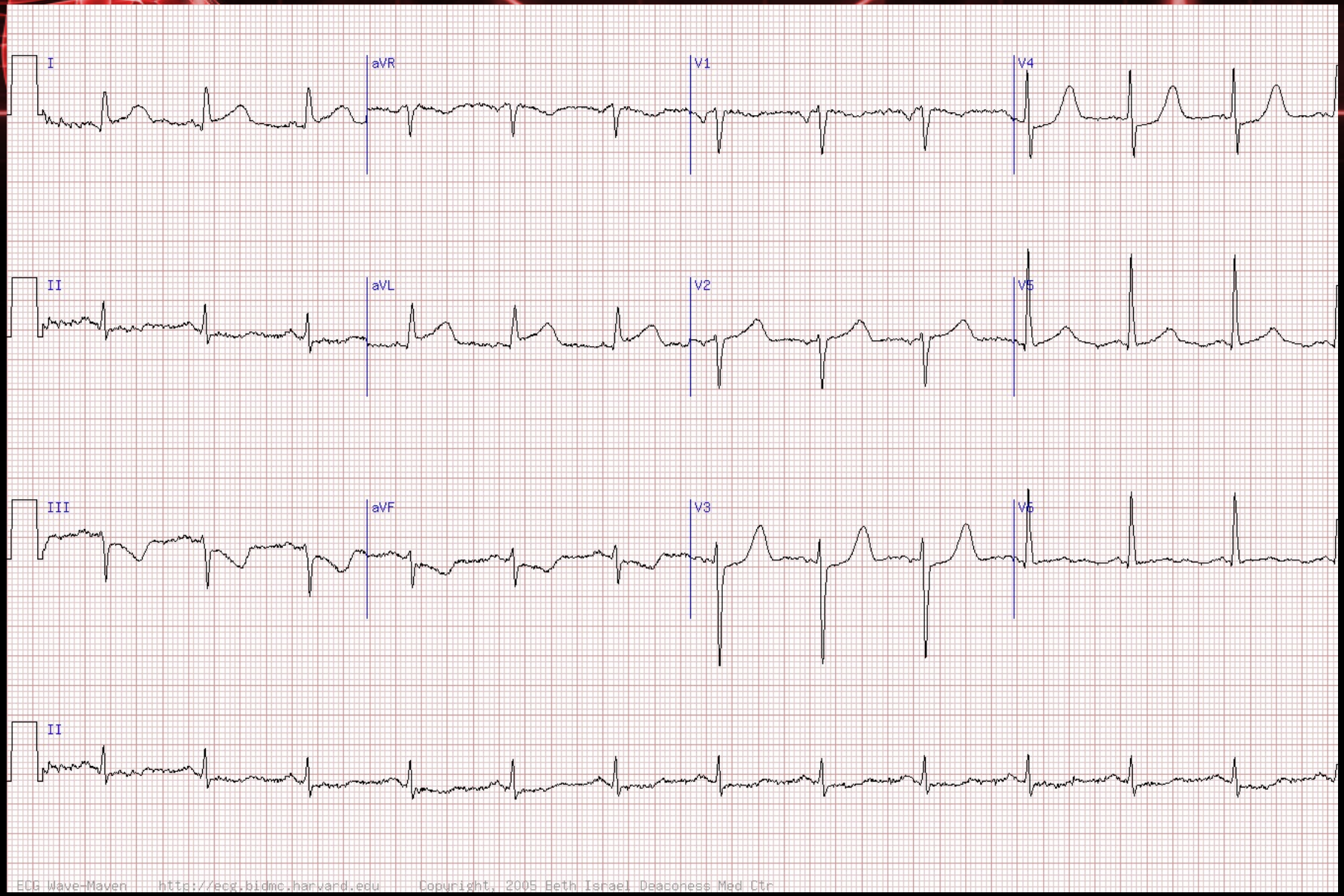


Lateral MI

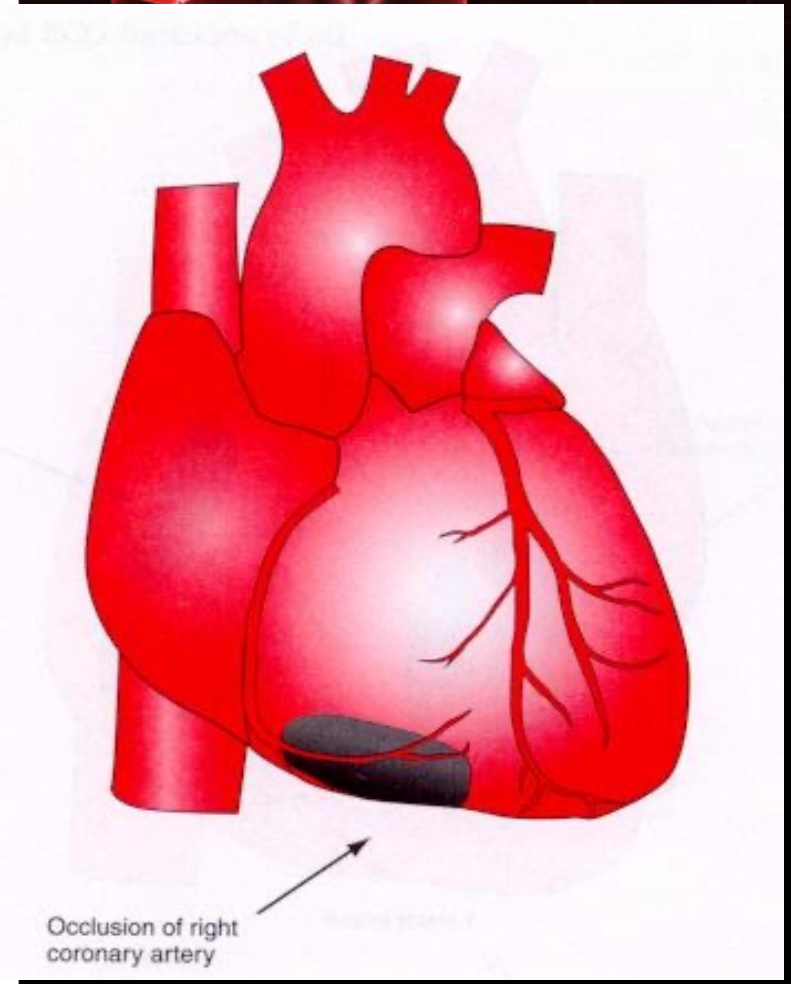

Occlusion of circumflex coronary artery

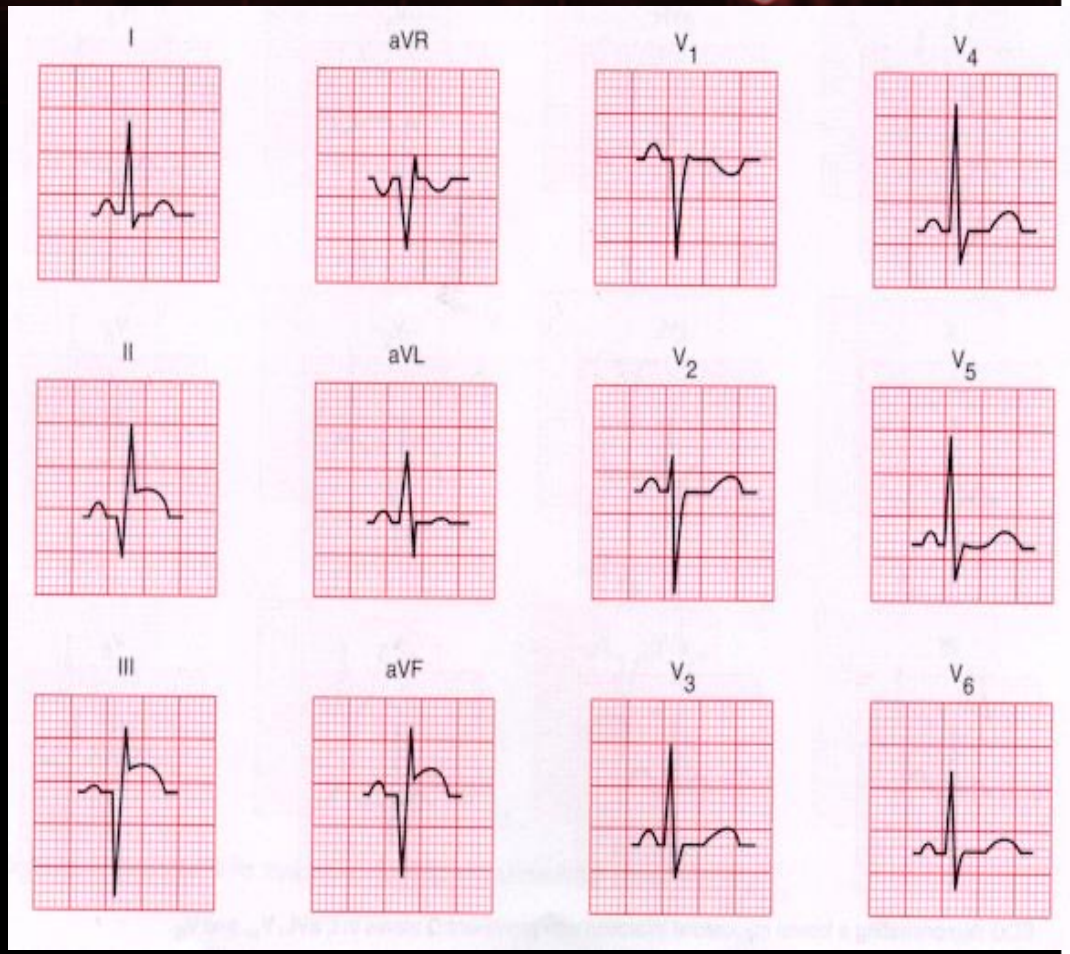

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Lateral Wall MI

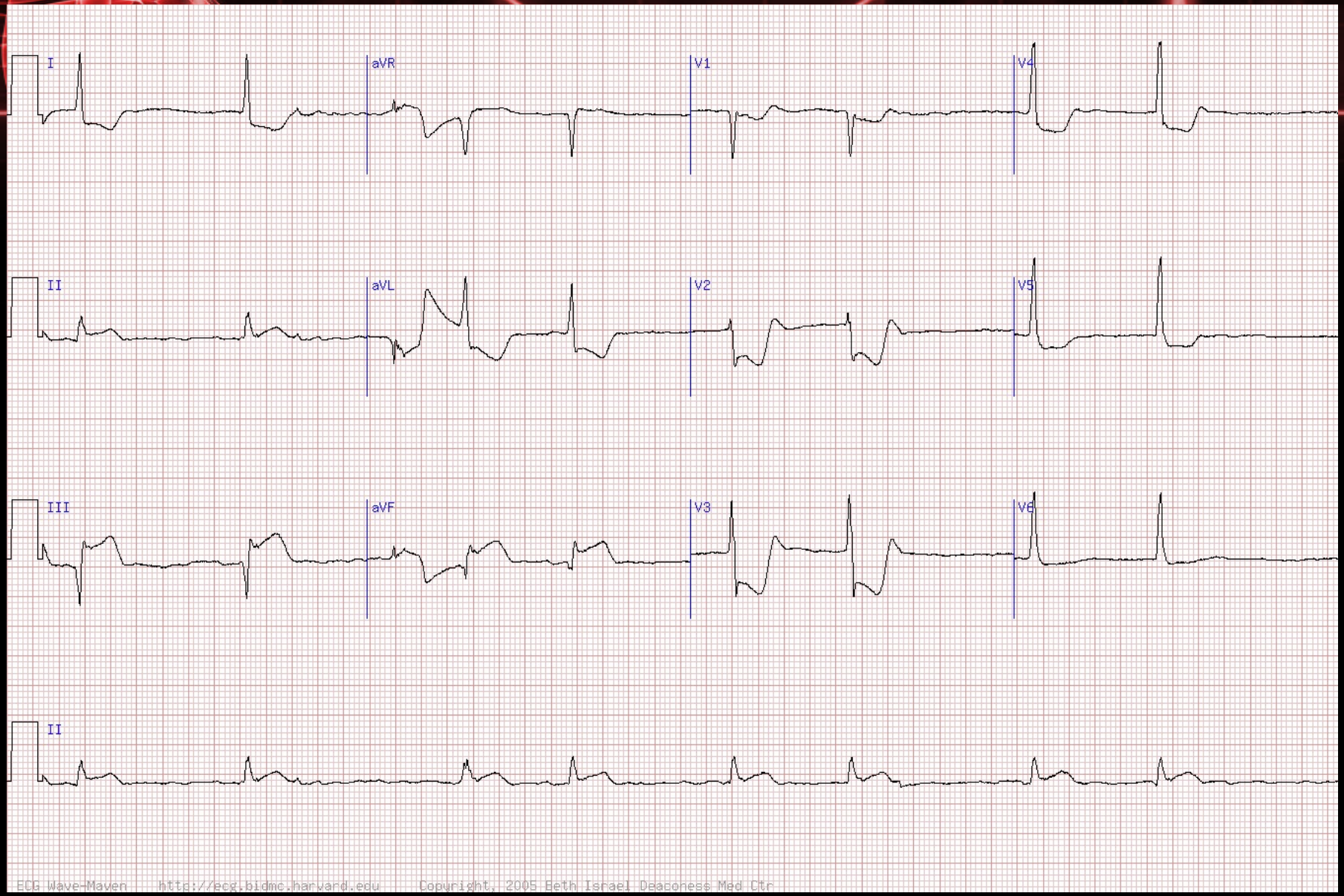


Inferior Infarct

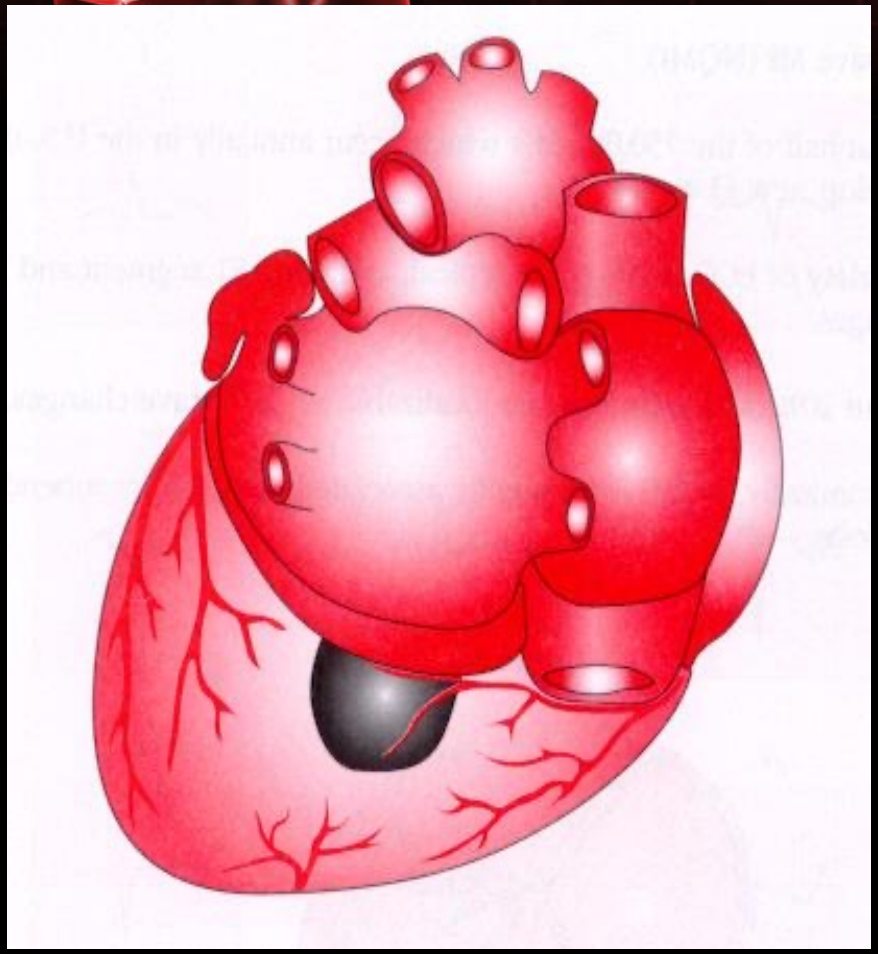


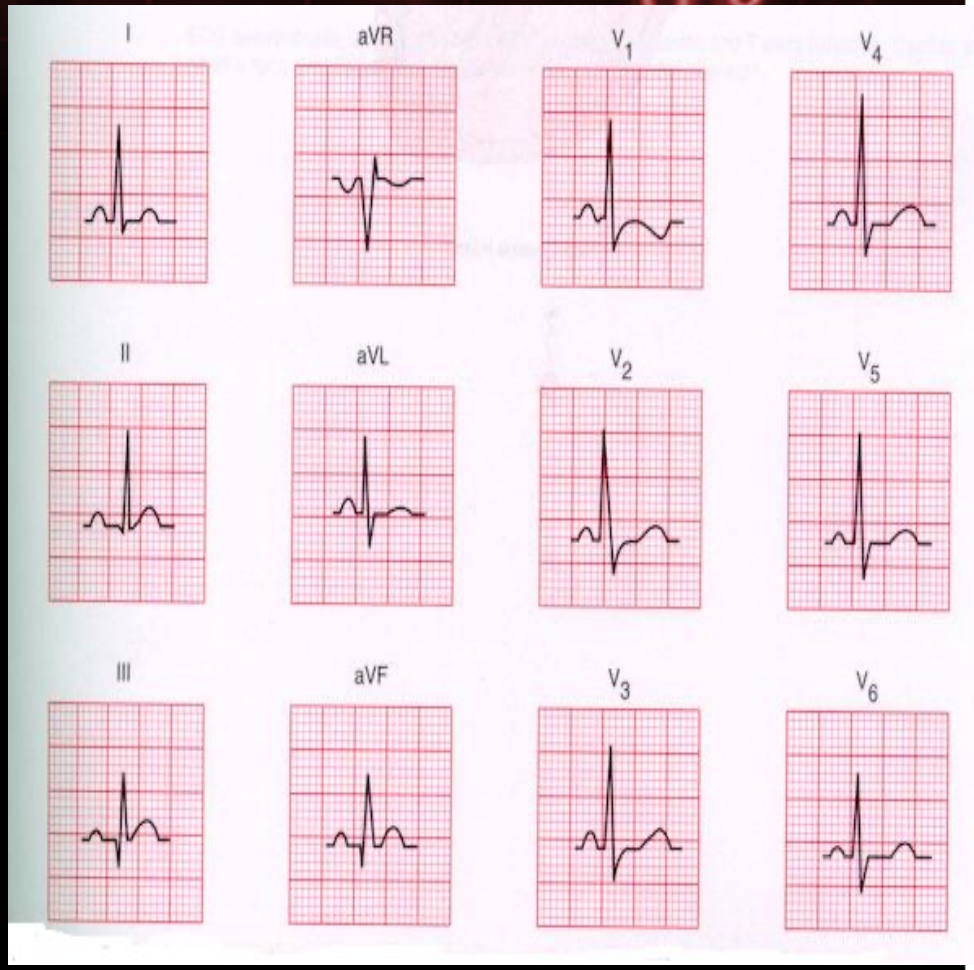


Inferior Wall MI

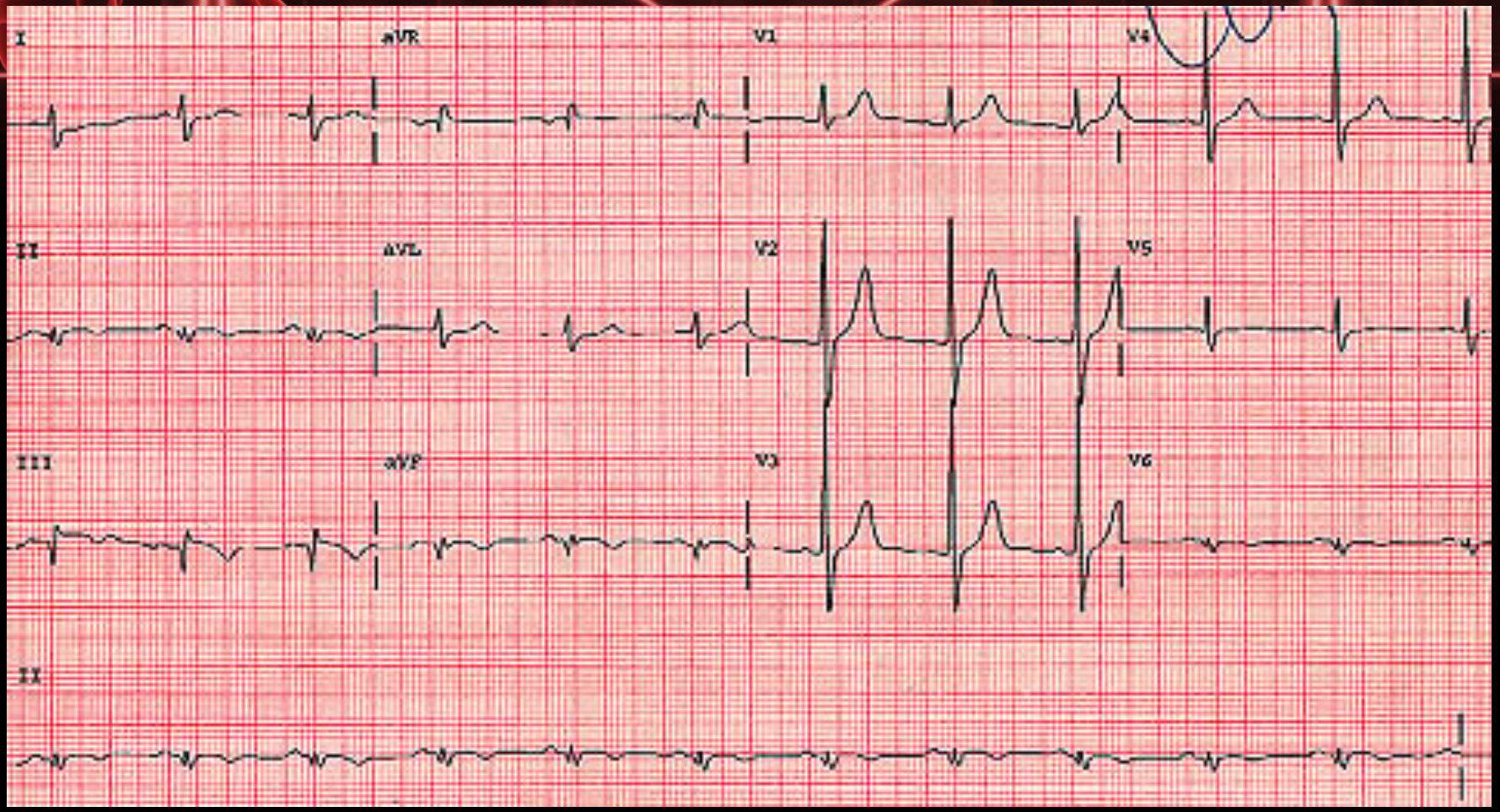


Posterior Infarct



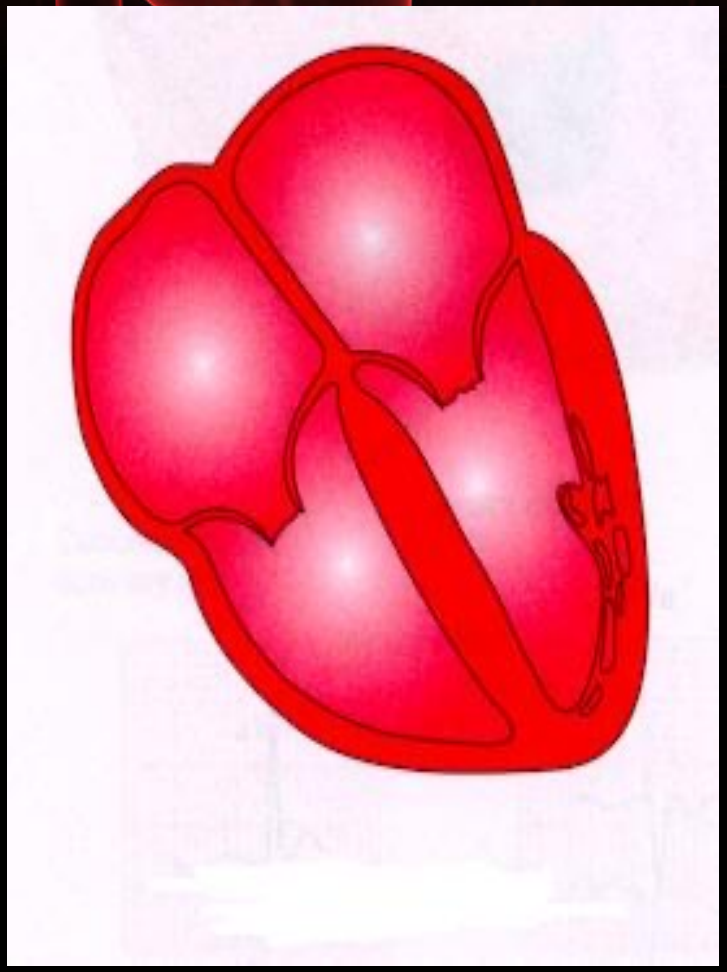


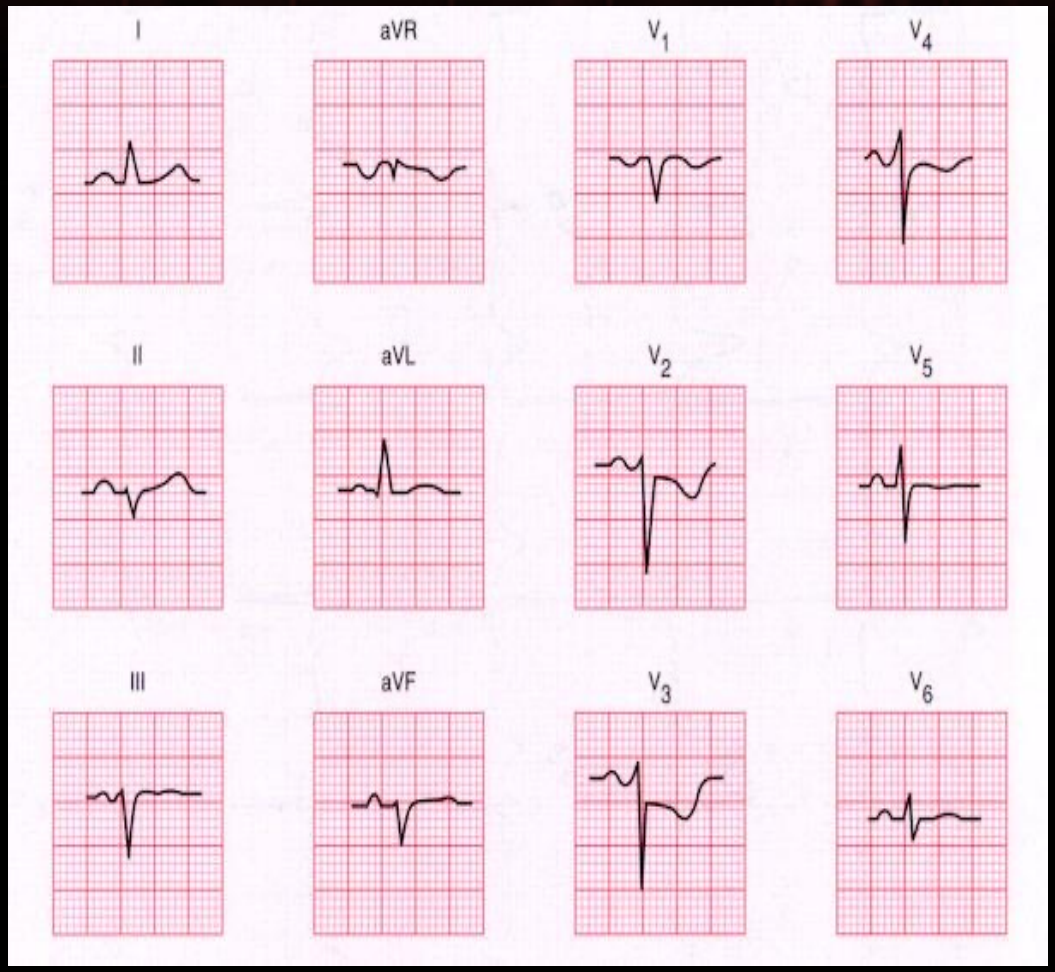
Posterior Wall MI



Notice tall R wave in V1. Posterior wall infarcts are often associated with inferior wall infarcts (Q waves in II, III and aVF).

Non Q-Wave Infarct





Your Next Patient……

- **A 58 year old male calls EMS complaining of chest discomfort that awoke him out of his sleep. Upon EMS arrival, the patient is found sitting on the edge of the bed. He is anxious but alert and oriented to person, place, time, and event.**
- **Onset: 30 minutes ago while sleeping**
- **Provoke: Nothing makes the pain feel better or worse**
- **Quality: Severe pressure or "ache"**
- **Radiate: The pain does not radiate**
- **Severity: 10/10**
- **Time: He has had chest pain before but "not this bad"**

- **Past medical history: HTN, dyslipidemia**

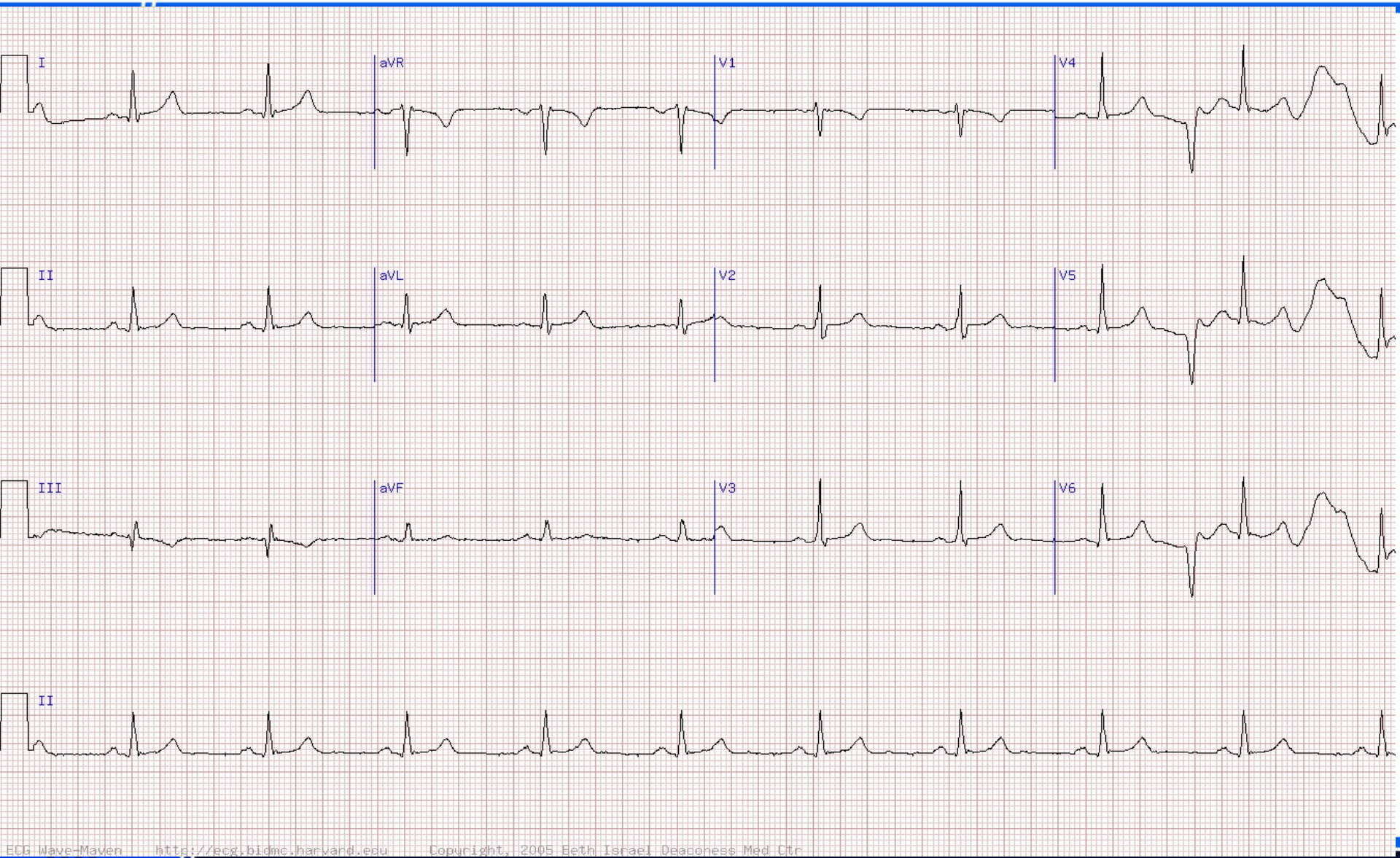
- **Medications: Lipitor, Norvasc, ASA**

- **Vital signs:**
- **RR: 24**
- **Pulse: 60**
- **BP: 160/98**
- **SpO2: 96 on RA**
- **Temp: 99.1**
- **BGL: 138**

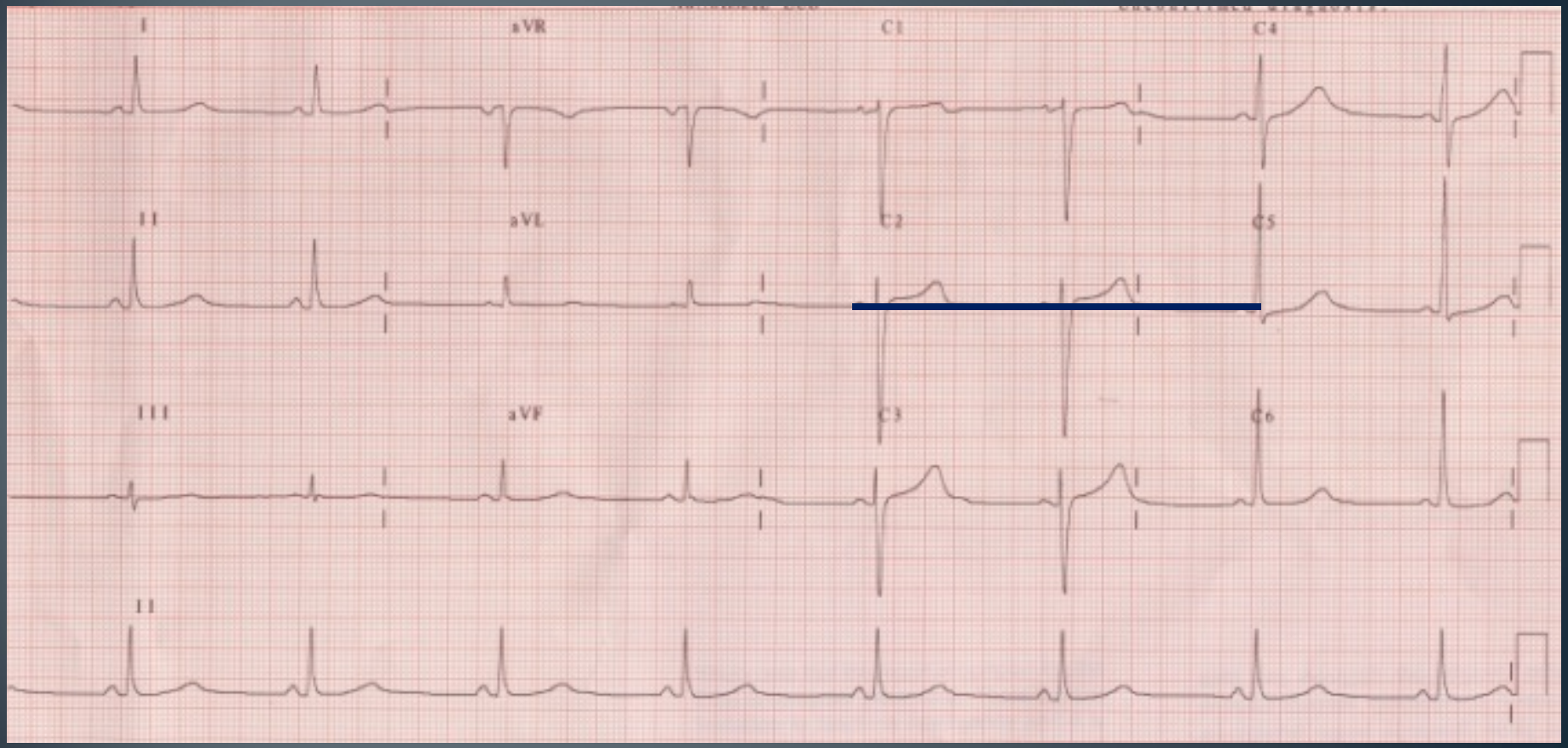
- **Breath sounds: basilar rales**

- **The patient admits to mild dyspnea. He states that he has "gained a little weight" recently and his doctor was getting ready to put him "on a water pill."**

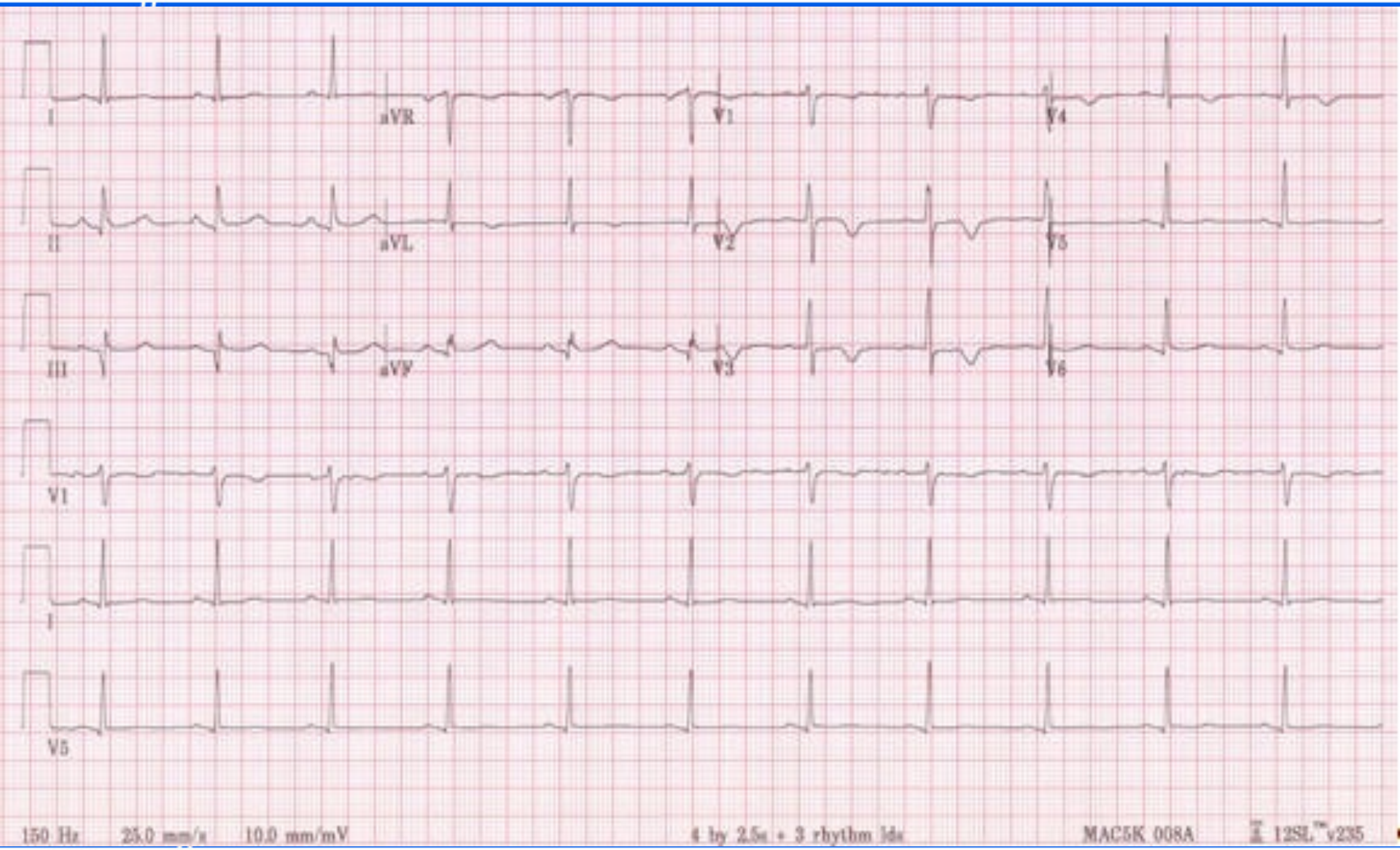
Can I Send Him Home?



One Hour Later

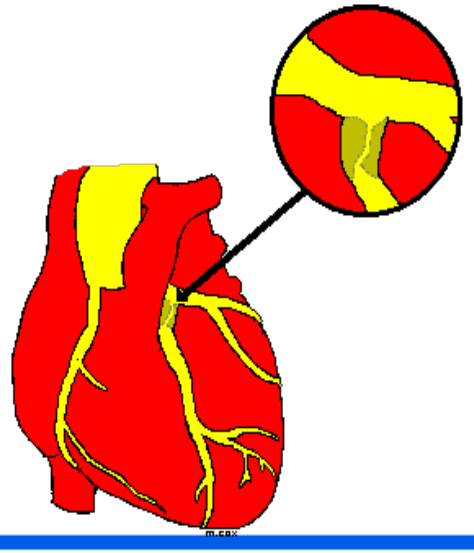


His EKG 4 days Later
What Do You See?



Wellens Syndrome

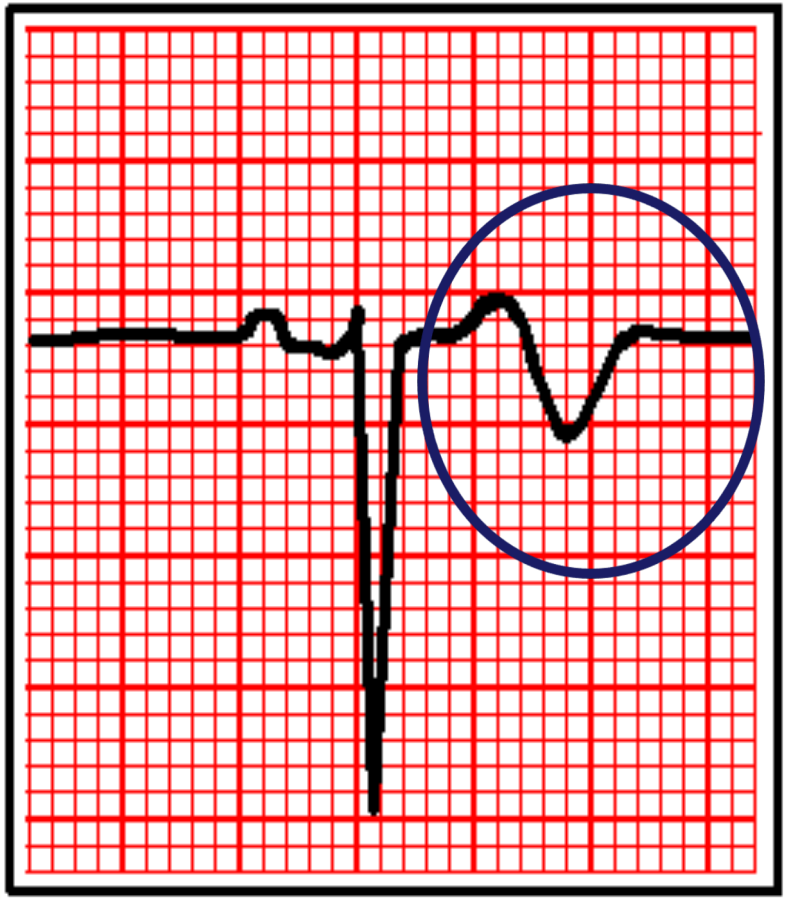
Wellens Syndrome is an easy to identify cardiac syndrome which indicates a critical high grade occlusion of the proximal LAD. If not identified and properly treated the mean time from onset of symptoms to extensive anterior wall MI is 8.5 days



Criteria of Wellens Syndrome

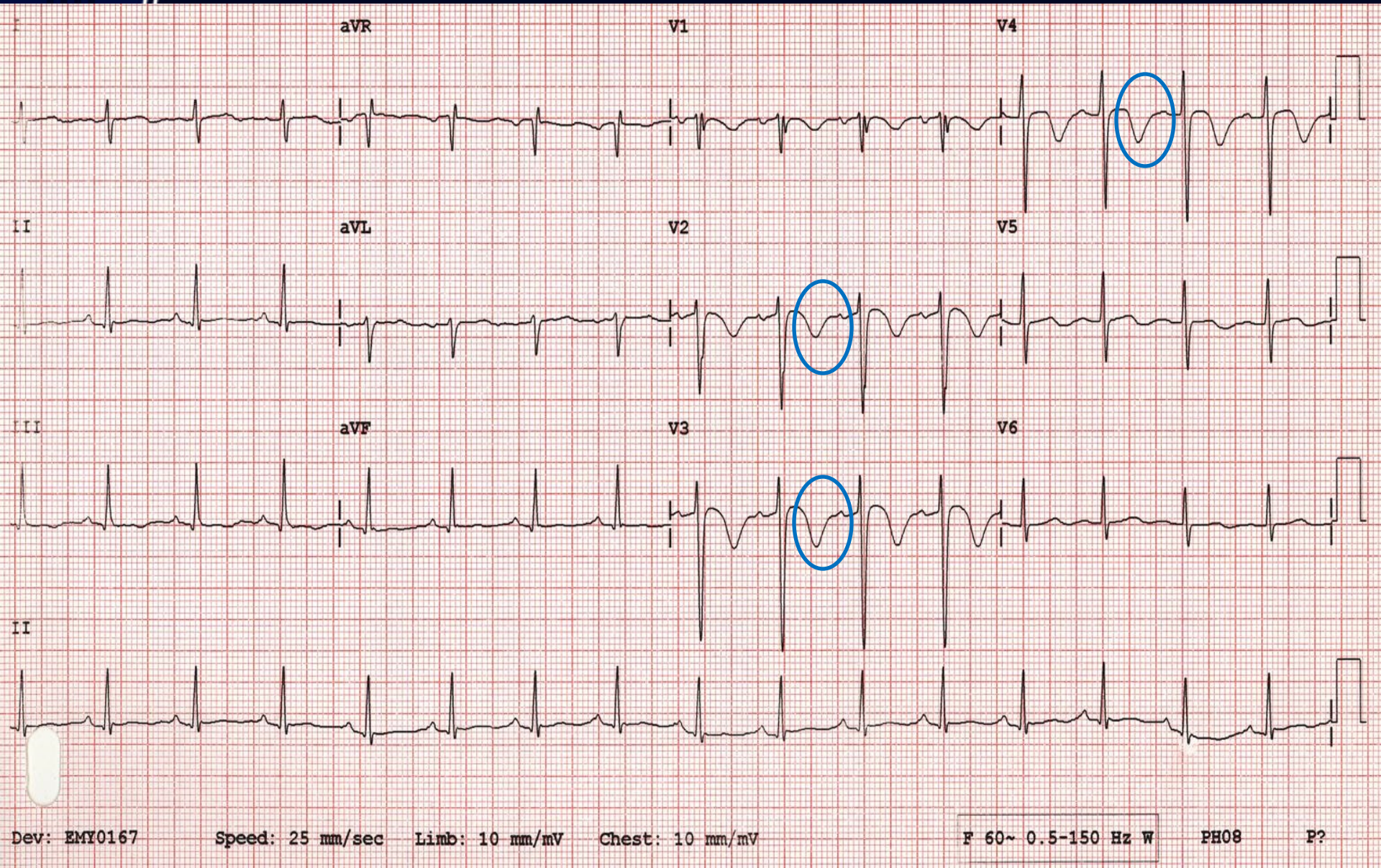
- Prior history of chest pain/angina
- Chest pain with normal EKG
- Normal or minimally elevated cardiac enzymes
- No pathologic precordial Q waves or loss of R waves
- ST segment in V2 and V3 that is isoelectric or minimally elevated (1mm), concave or straight
- Symmetric and deep T-wave inversion or biphasic T-waves in V2-V5 or V6 in pain free periods
- Proximal LAD stenosis

Wellens Syndrome

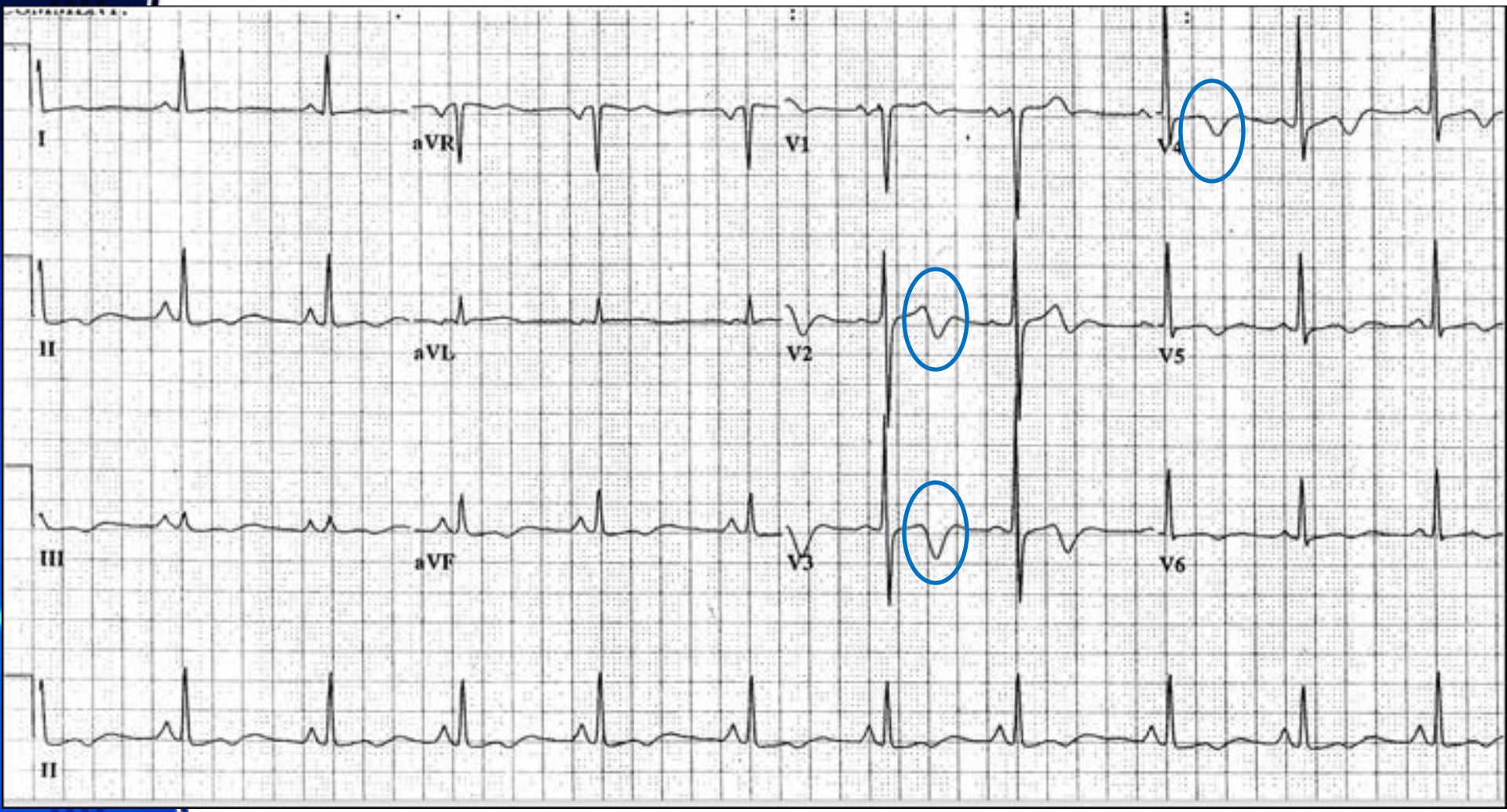


Biphasic T-wave

Wellens Syndrome Type I



Wellens Syndrome (Type II)



Wellens Syndrome Management

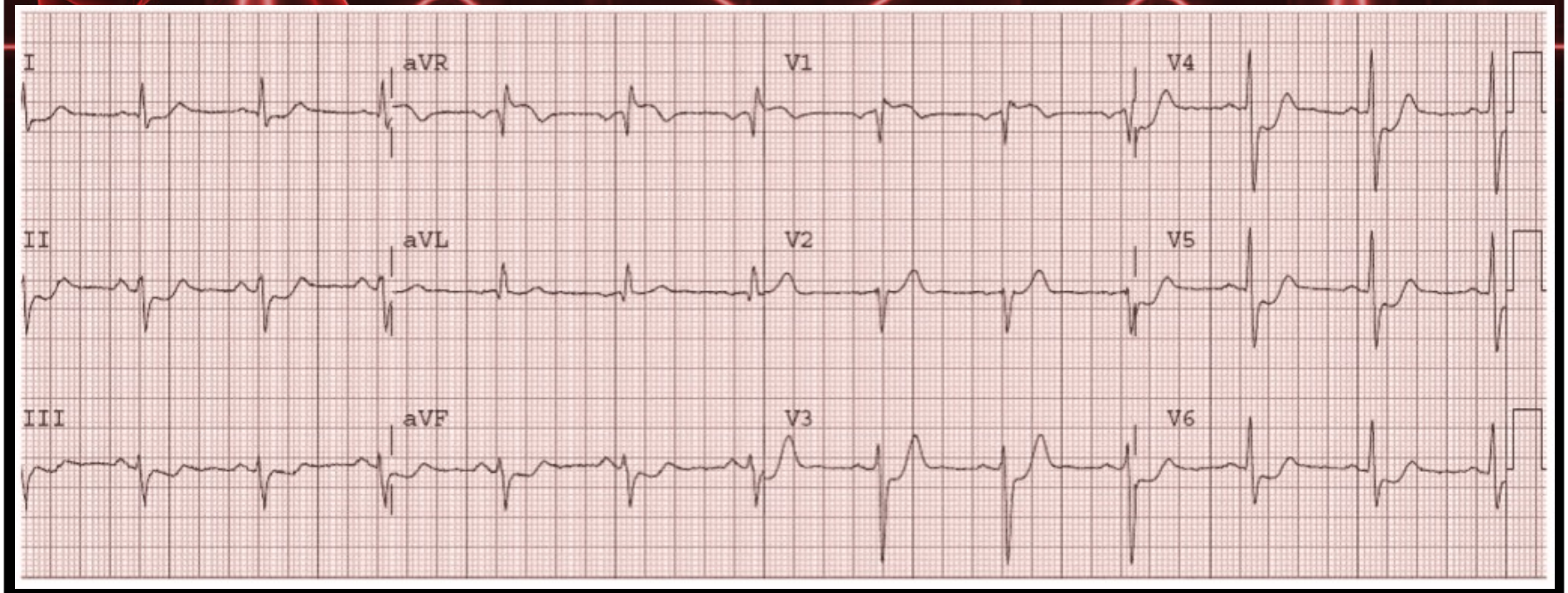
Transport to a facility capable of:

- ✓ Prompt percutaneous transluminal angioplasty
- ✓ Cardiac catheterization or
- ✓ CABG surgery

Would Have Never Guessed

- A 59-year-old man with a history of hypertension, smoking 1 ppd for 40 years, and body mass index (BMI) of 43 developed severe persistent central chest pain at rest that started this AM. His temperature is 100.0, blood pressure is 140/90 mm Hg, respirations are 22 per minute, heart rate is 70 beats per minute and O2 Sat is 92% on room air. His EKG from EMS while in route is presented to you upon arrival. What do you do?

His EKG - What Do You See?



Left Main STEMI

- Lead aVR has often been called the “forgotten lead”, but it is worth paying attention to because ST-segment elevation in aVR portents a worse prognosis in ACS.
- ST elevation in aVR ≥ 1mm is the strongest independent predictor of either severe LMCA or triple-vessel disease requiring CABG in patients with NSTEMI.
- Elevation in aVR of ≥ 0.5 mm is an independent predictor of mortality in patients with STEMI.

Left Main Coronary Artery Disease

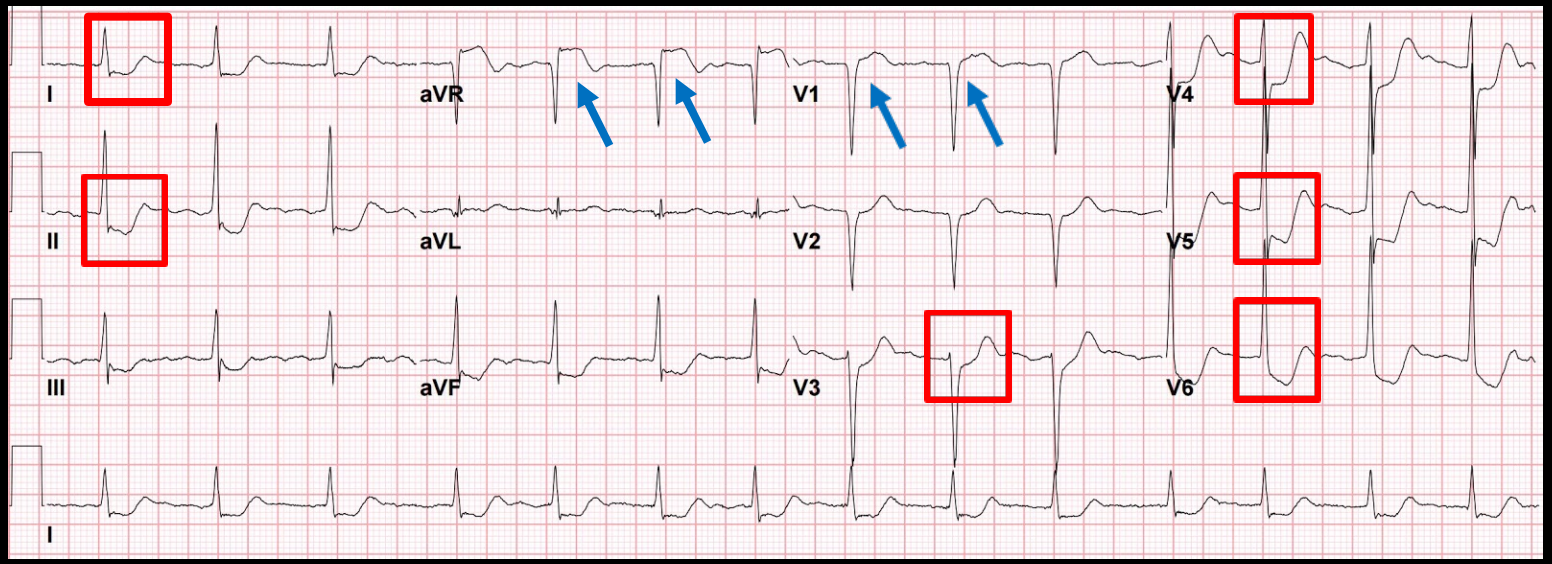
- Early identification of LMCA disease is critical because acute occlusion can cause rapid hemodynamic and electrical deterioration.
- LMCA insufficiency due to critical stenosis of the left main artery is important to recognize because these patients can progress to complete occlusion and are likely to require surgical intervention (such as CABG).

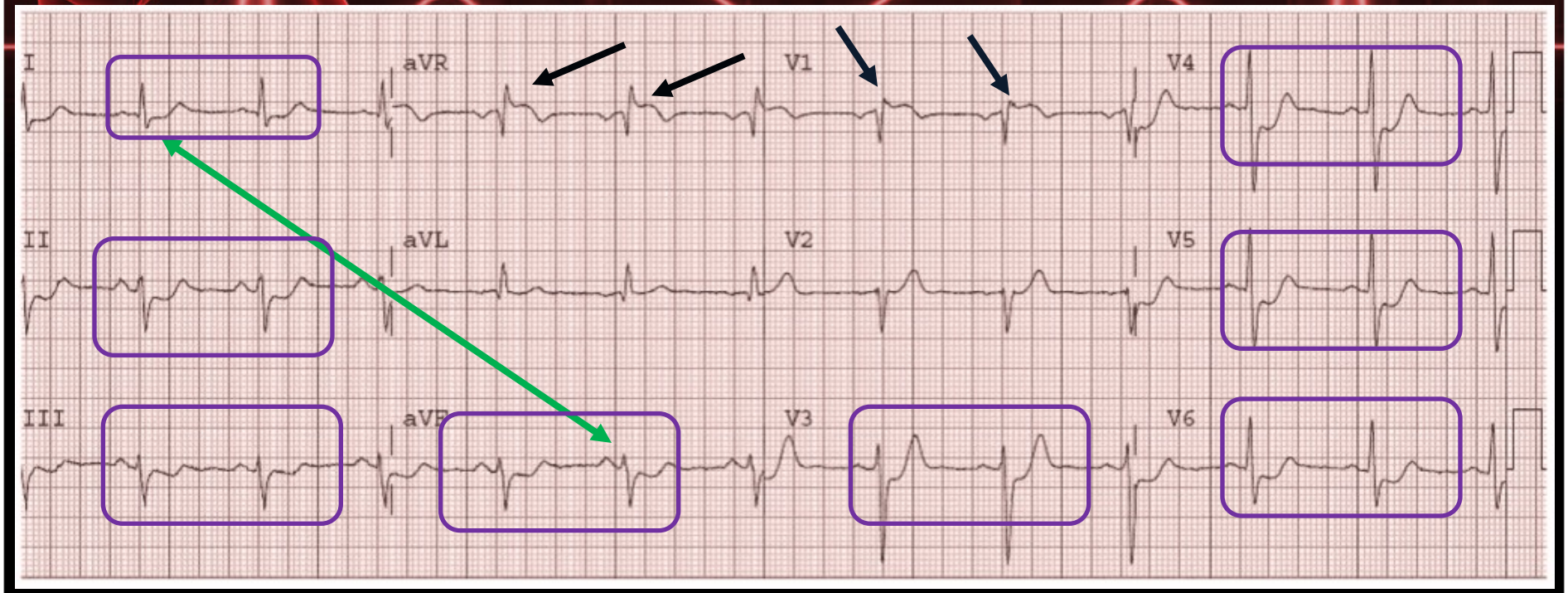
How it Occurs

- ST-elevation in aVR occurs by the following mechanisms:
  - Critical narrowing of the LMCA causing subendocardial ischemia due to insufficient blood flow.
  - Transmural infarction of the basal septum due to a very proximal LAD occlusion or complete LMCA occlusion.
  - Severe multi-vessel coronary artery disease. **
  - Diffuse subendocardial ischemia from oxygen supply/demand mismatch.

Classic Findings on EKG

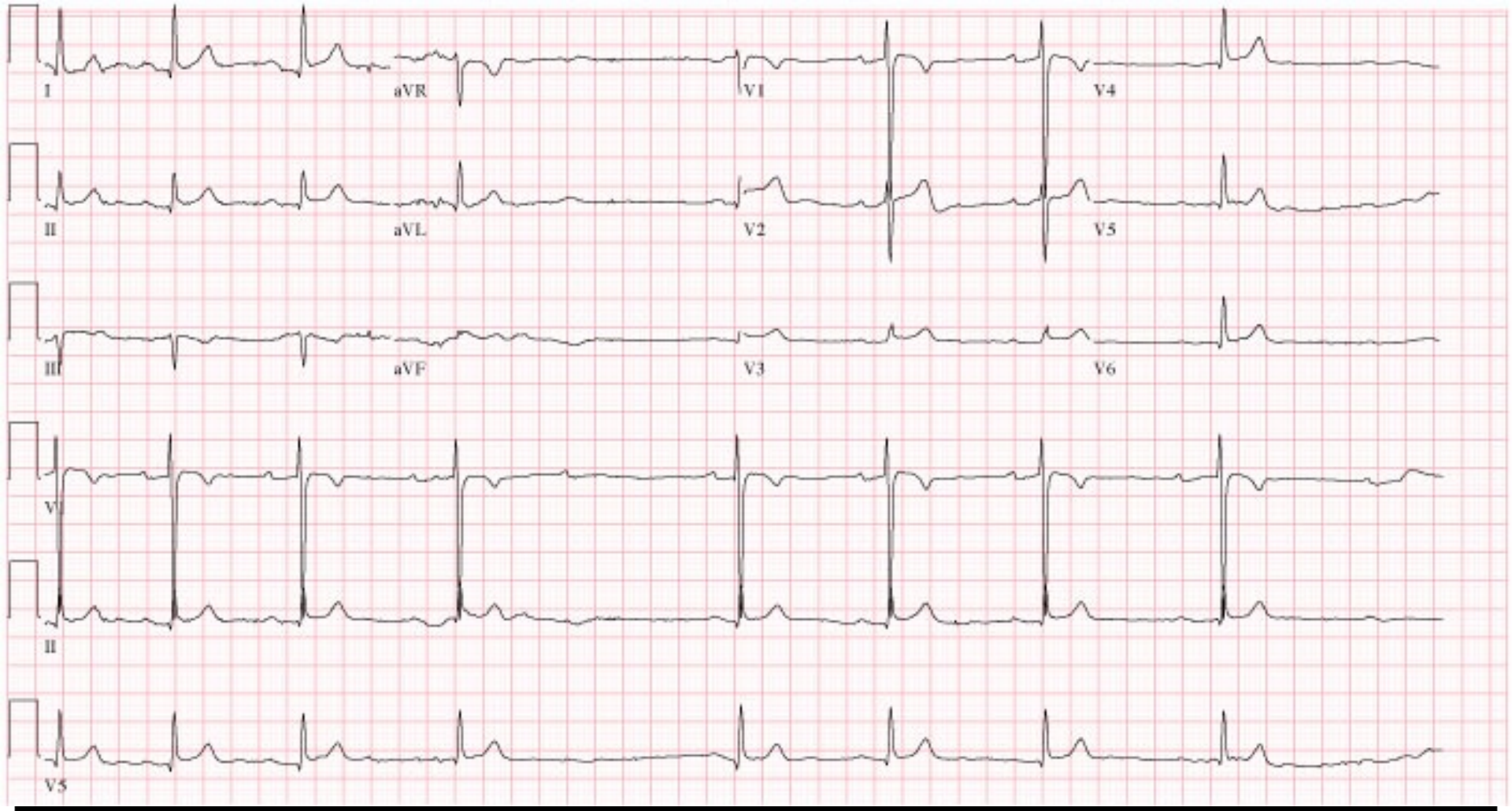
- ST depression in leads I, II, aVL and V4-6
- ST elevation in aVR ≥ 1mm
- ST elevation in aVR ≥ V1




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

A Few Tips on Left Main Presentation

- STE ≥ 1 mm in aVR or V1 with STD ≥ 1 mm in ≥ 6 leads can suggest left main coronary artery insufficiency, proximal LAD insufficiency, or triple vessel disease, especially if accompanied by pathologic Q-waves, hemodynamic compromise, and/or refractory symptoms.
- STD are most prominent in the inferior and lateral leads and thought to represent subendocardial ischemia.
- This EKG pattern is not specific to LMCA/proximal LAD insufficiency and can be seen in other conditions (eg, pulmonary embolism, aortic dissection, LVH with strain pattern).



Rate_Underlying Rhythm 75 Rhythm Regular? _No P-Waves Upright? _Yes_

Q-Waves__No_ Any ectopic beats? _Yes – lateral and anterior ST elevation__

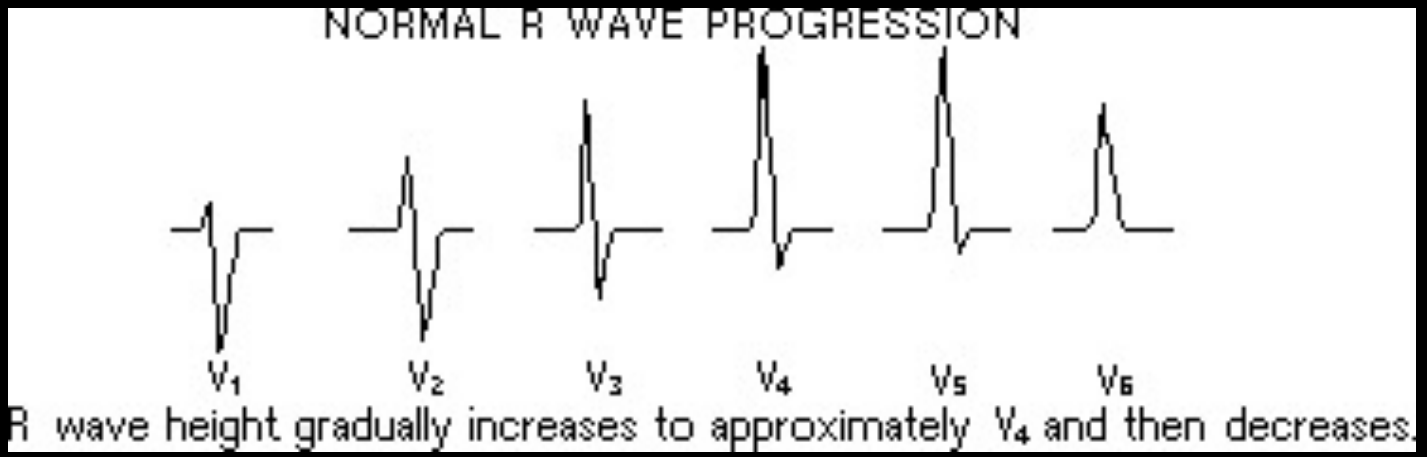
Rhythm Diagnosis is Sinus Rhythm with sinus pause and ST Elevation____

Now we search for everything else of importance.

R-Wave Progression

R Wave Progression

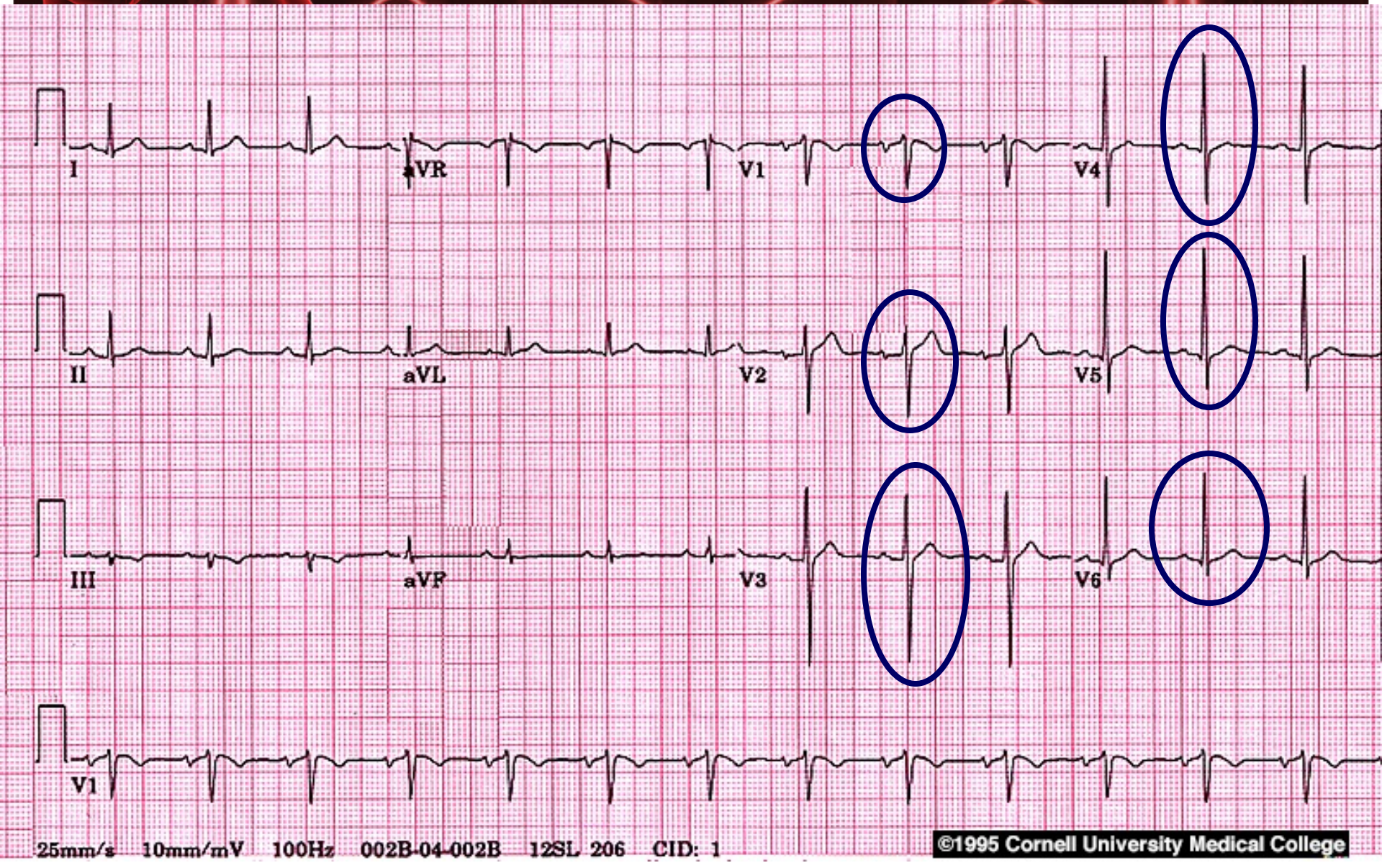
- Normally the R wave becomes progressively taller as one moves across the precordial leads.
- A number of conditions may be associated with **"poor" R wave progression**, in which the R wave in leads V1 through V3-V4 either does not become bigger, or only increases very slowly in size.



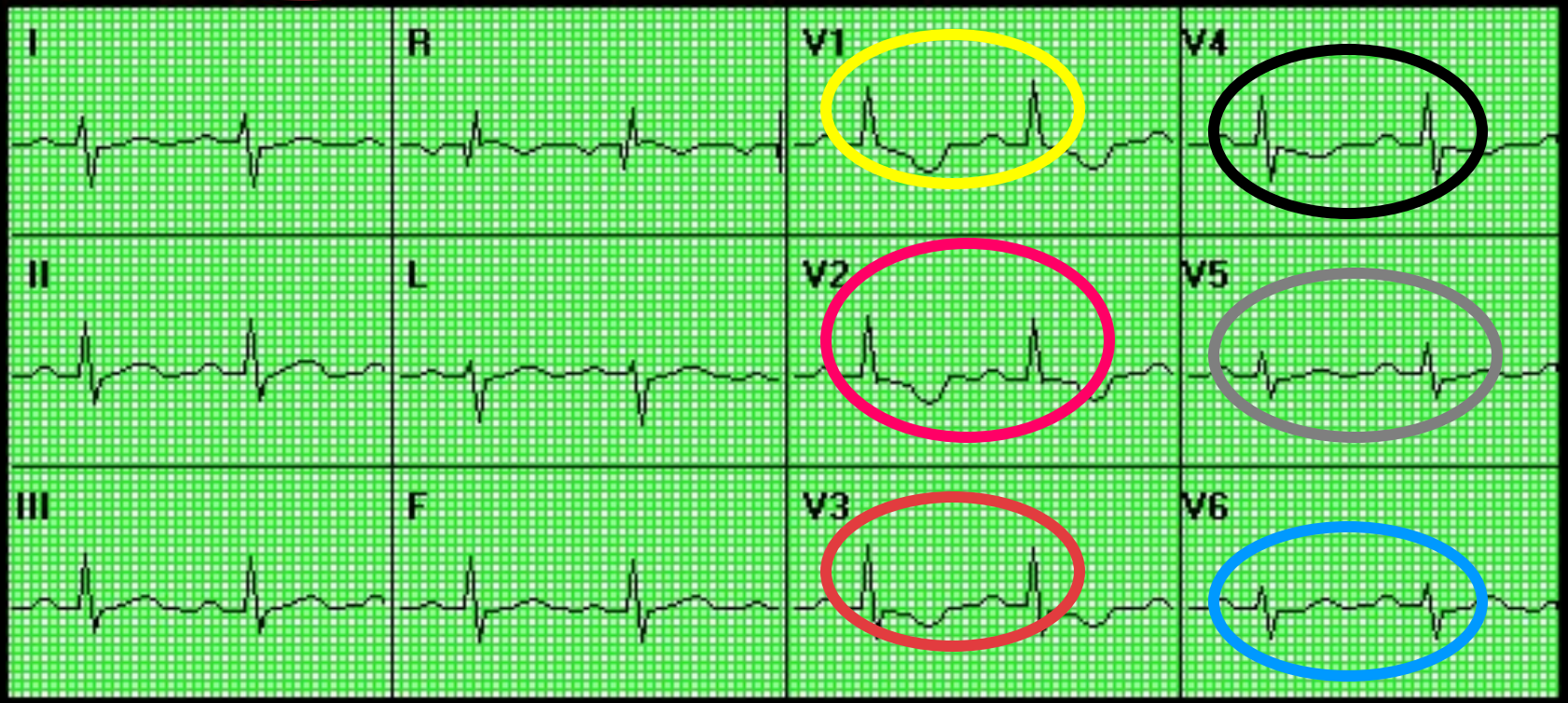
Causes of Poor R Wave Progression

LVH
RVH
Pulmonary disease (i.e., COPD, chronic asthma)
Anterior or anteroseptal infarction
Conduction defects (i.e., LBBB, LAHB)
Cardiomyopathy
Chest wall deformity
Normal variant
Lead misplacement

Good R-Wave Progression

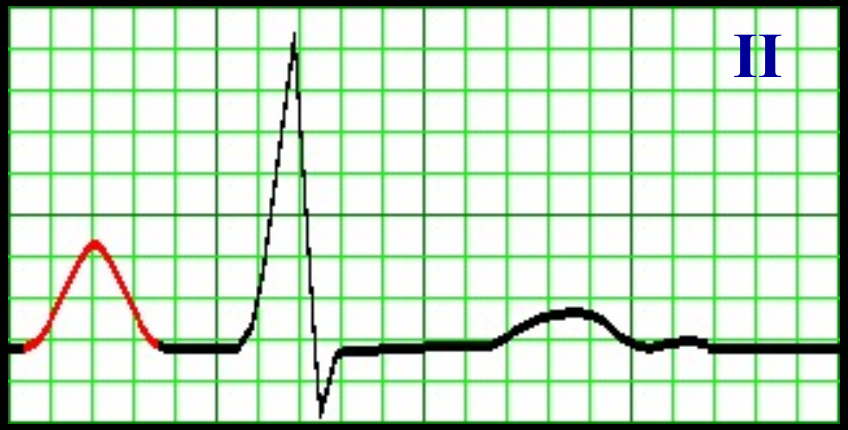


Poor R-wave Progression



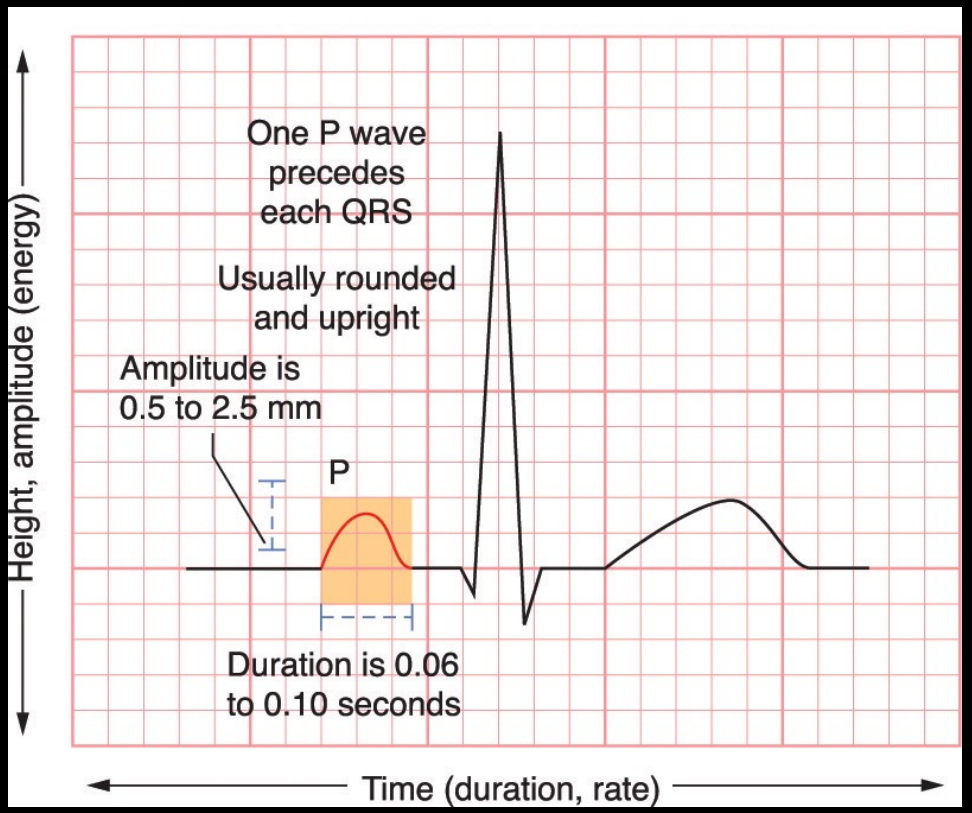
Right Atrial Enlargement

- Diagnosed by looking for a:
  - Biphasic P-wave in lead V1 &
  - P wave 2.5 millimeters or greater in height in lead II
- Causes of right atrial enlargement include COPD, mitral stenosis, mitral regurgitation, or pulmonary emboli.
- Because RAE is so frequently seen in chronic pulmonary disease, the peaked P wave is often called “P pulmonale.”

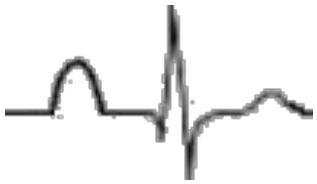
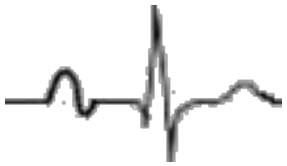


Normal P Wave

- Duration 0.06 – 0.10 seconds
- Amplitude 0.5 – 2.5 mm
- First portion represents right atrial depolarization
- Terminal portion represents left atrial depolarization

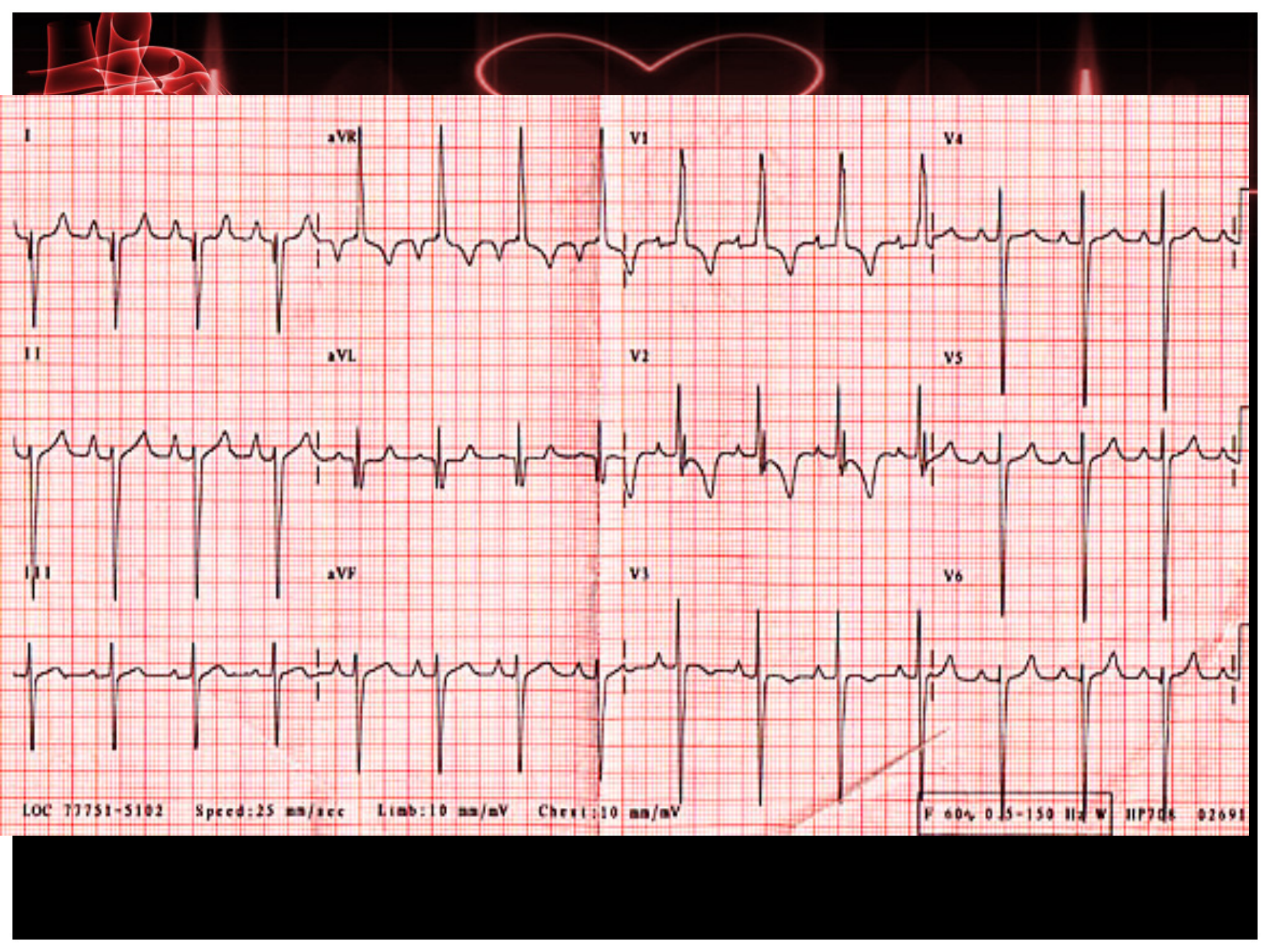


P-Pulmonale Pattern

P pulmonale in II, III, aVF	P pulmonale in V1
	

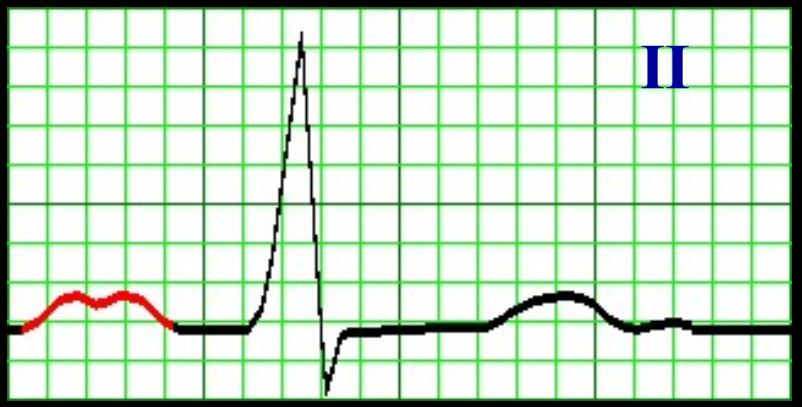
Classic finding in Severe Right Atrial Enlargement (RAE)

Tall ***P***eaked and ***P***ointed ***P*** waves in the ***P***ulmonary leads (II, III, aVF). If the P wave looks "uncomfortable to sit on", think ***RAE!!!***


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
LOC 77751-5102
Speed:25 mm/sec
Limb:10 mm/mV
Chest:10 mm/mV

Left Atrial Enlargement

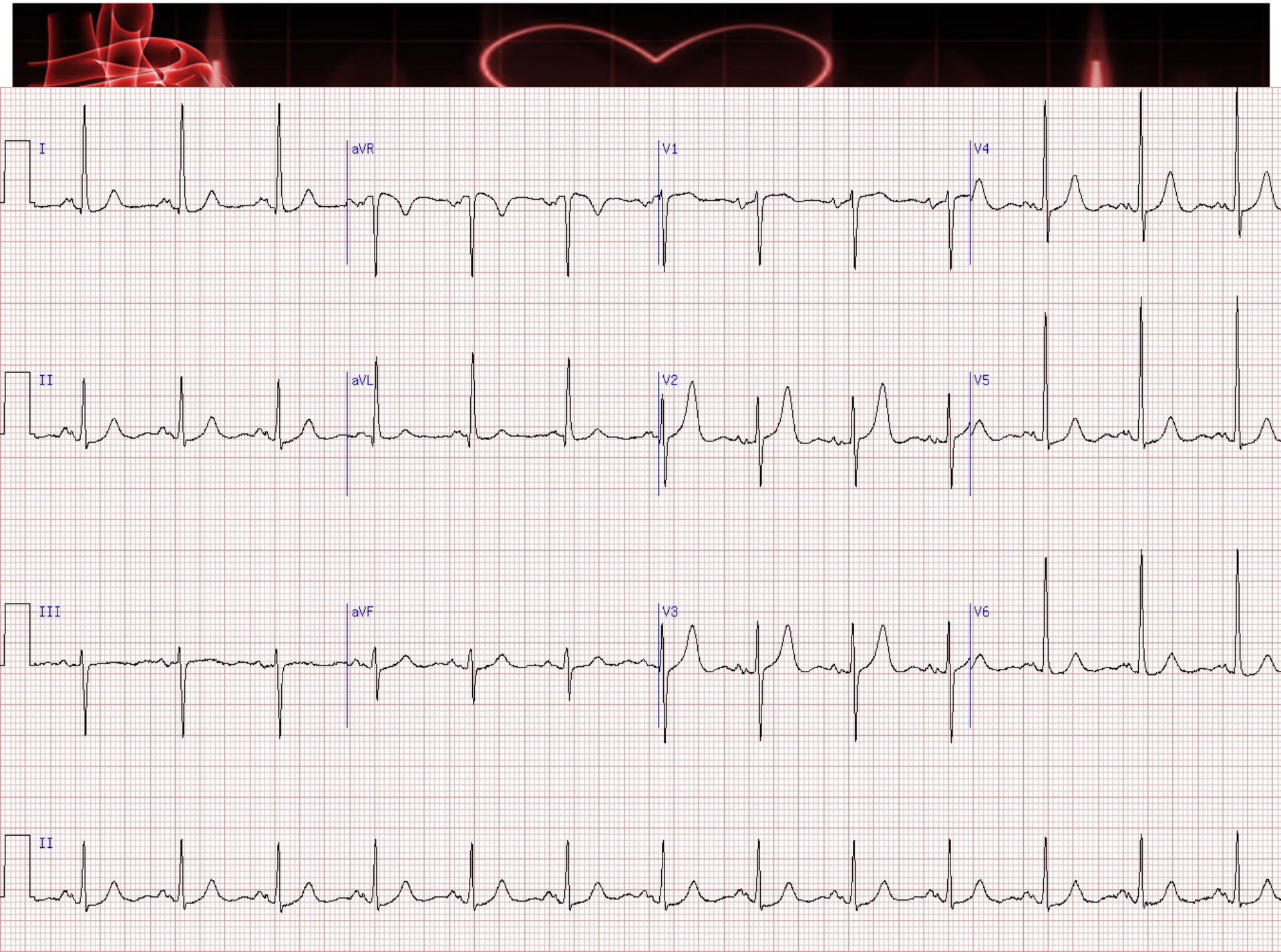
- Dilation or hypertrophy of the left atrium may increase the DURATION of the P wave (> 0.11msec). (Recall that right atrial enlargement causes an increase in the HEIGHT or amplitude of the P wave.)



P-Mitrale Pattern

P mitrale in II, III, aVF	P mitrale in V1
	

- Diagnosed by finding an *m*-shaped (notched) and widened P wave (≥ 0.12 second) in a "*m*itral" leads (I, II, aVL) and/or a deep negative component to the P in lead V1.
- Caused by conditions that increase either pressure or volume loading on the atria leading to enlargement and/or hypertrophy.
  - Longstanding hypertension
  - Obstructive cardiomyopathy
  - Aortic stenosis
  - Aortic regurgitation


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

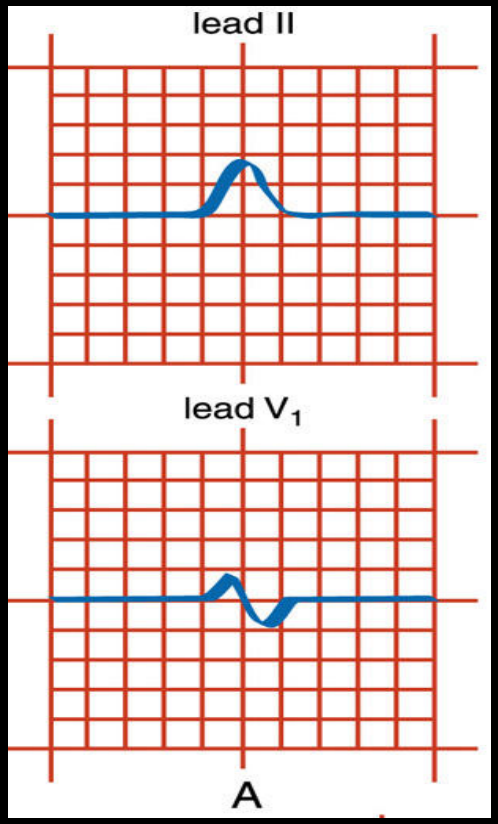
Summary

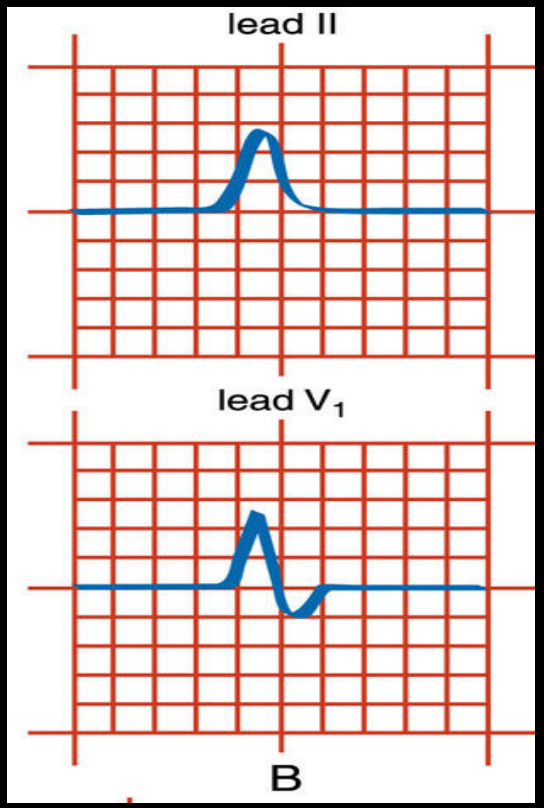
- **Atrial Enlargement Criteria**

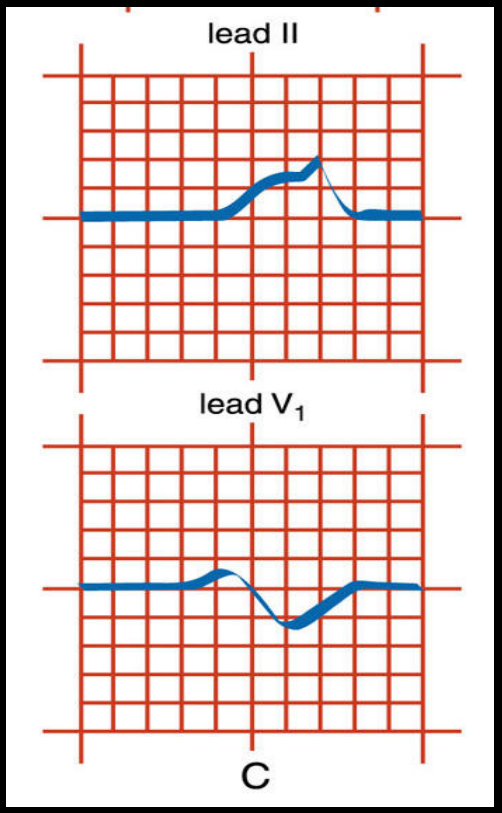
  P > 2.5mm height = RAE

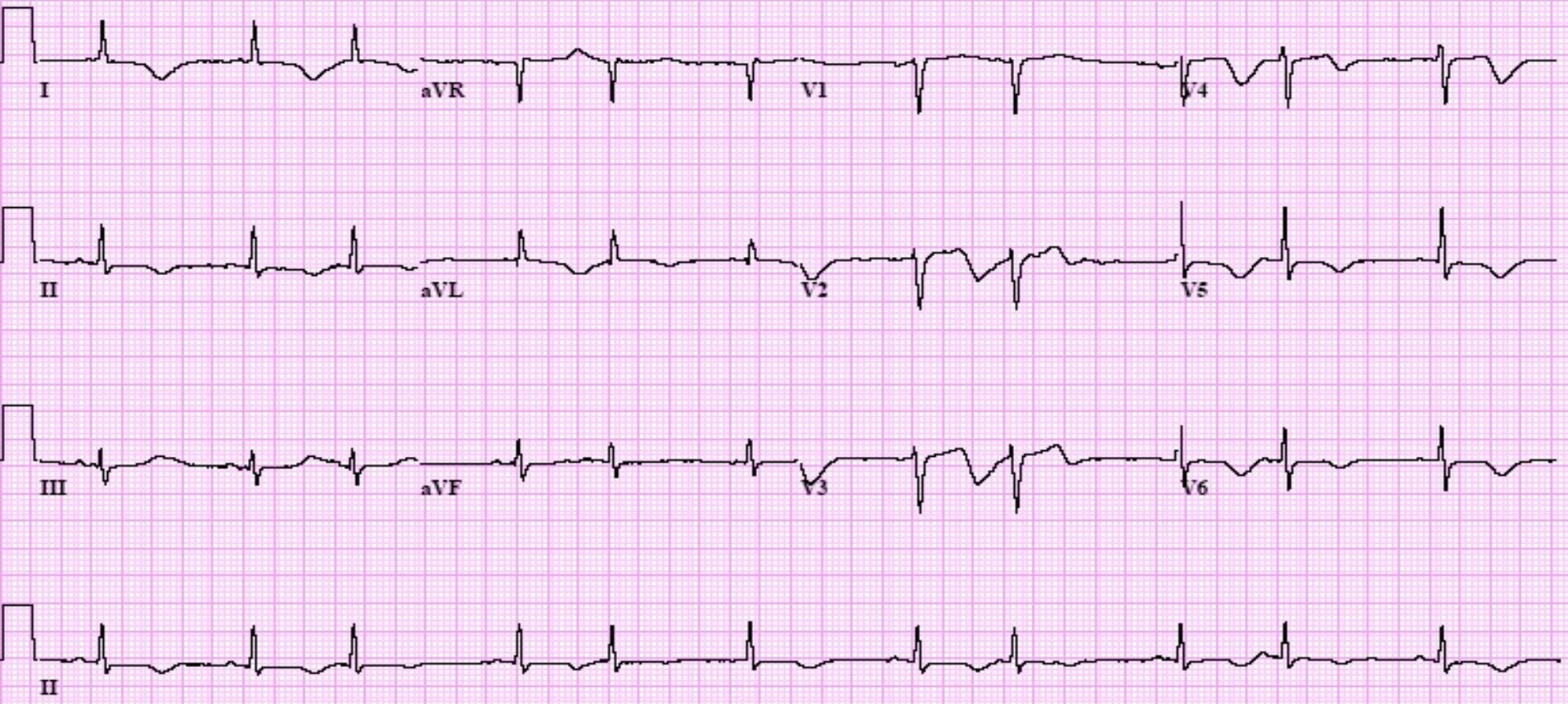
  P > 0.11 sec or P notch > 1 box width

  or P biphasic > 1 box square = LAE









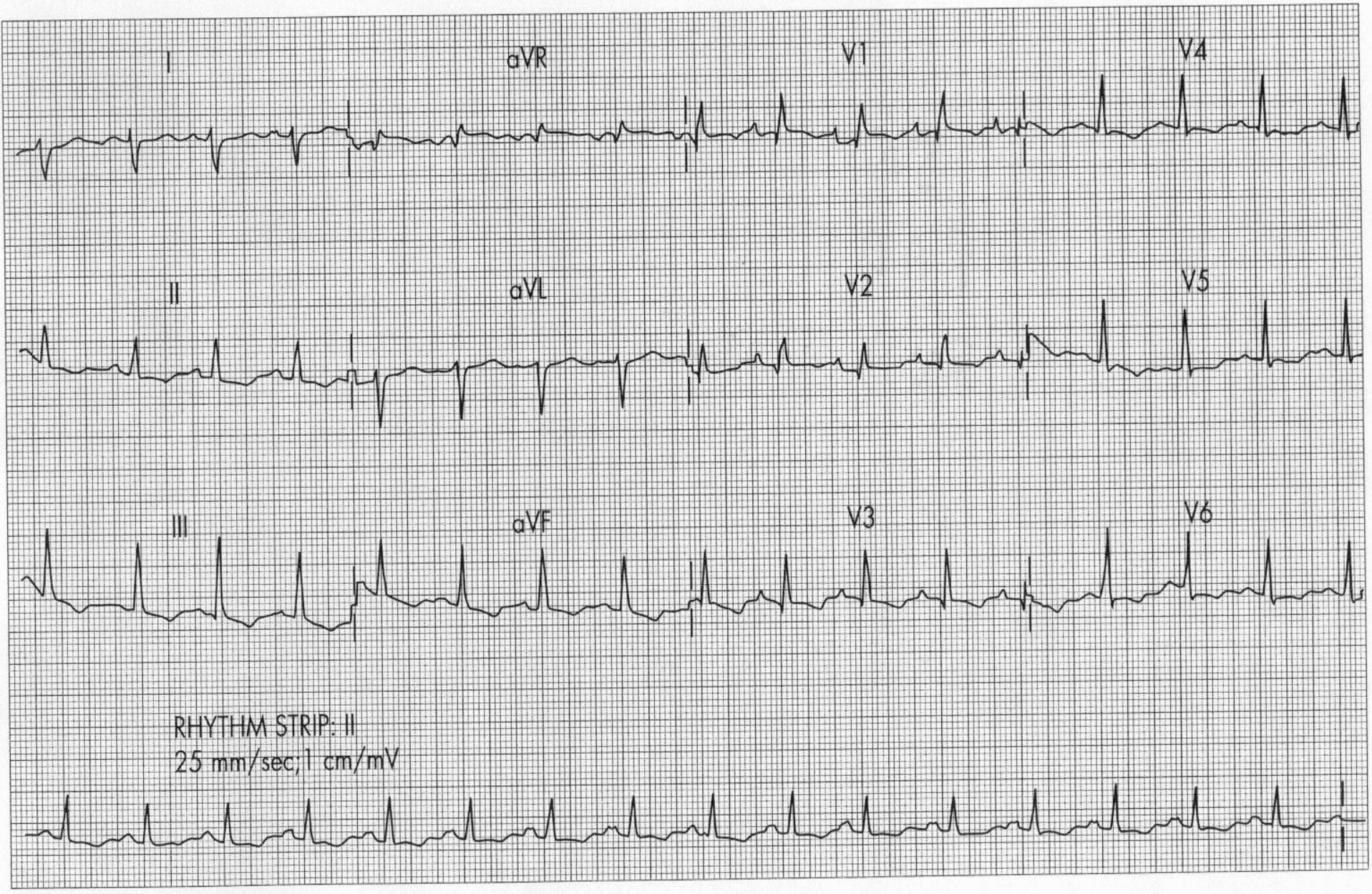
Rate_Variable_ Rhythm Regular? _No___ P-Waves Upright? __Yes_____

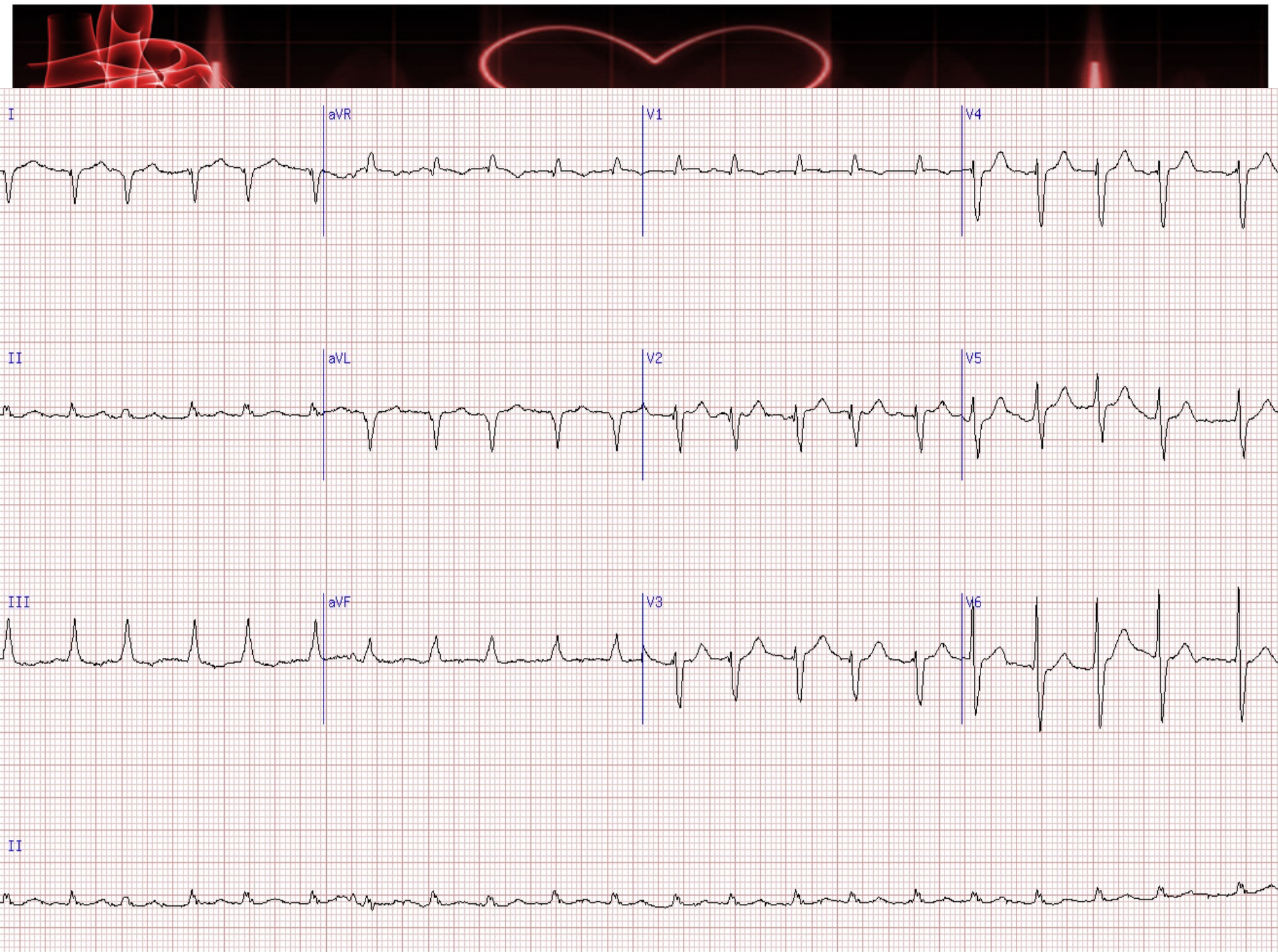
Q-Waves_No_ Any ectopic beats? __Yes_____

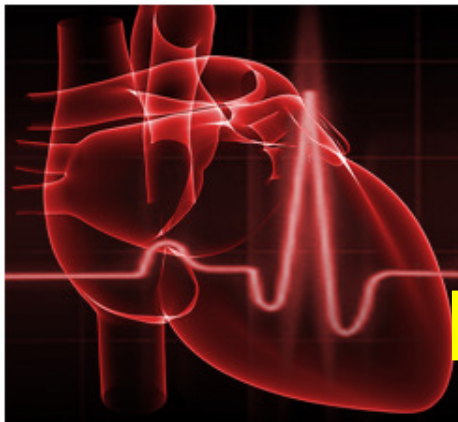
Rhythm Diagnosis is _Sinus Rhythm with bigeminal pattern of PJC/PAC's____

Right ventricular hypertrophy

- RVH increases the height of the R wave in V1. An R wave in V1 that is greater than 7 boxes in height, or larger than the S wave, is suspicious for RVH. Other findings are necessary to confirm the EKG diagnosis.
  - Other findings include right axis deviation, taller R waves in the right precordial leads (V1-V3), and deeper S waves in the left precordials (V4-V6). The T wave is inverted in V1 (and often in V2).
- True posterior infarction may also cause a tall R wave in V1, but the T wave is usually upright, and there is usually some evidence of inferior infarction (ST-T changes or Qs in II, III, and AvF).


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec;1 cm/mV


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



Left Ventricular Hypertrophy

- Caused by increased loads on the left ventricle.
- Etiologies include:
  - hypertension, aortic stenosis or regurgitation, mitral regurgitation
- Left ventricular hypertrophy (LVH) may be difficult to diagnose with certainty from the EKG.
  - Different scoring criteria have been recommended.
- One of the simplest uses five criteria, with the certainty of diagnosis based on the number of criteria present. If one is present, diagnose “possible LVH”; if two, “probable LVH”; if three are found, “definite LVH.”

Multiple LVH Criteria

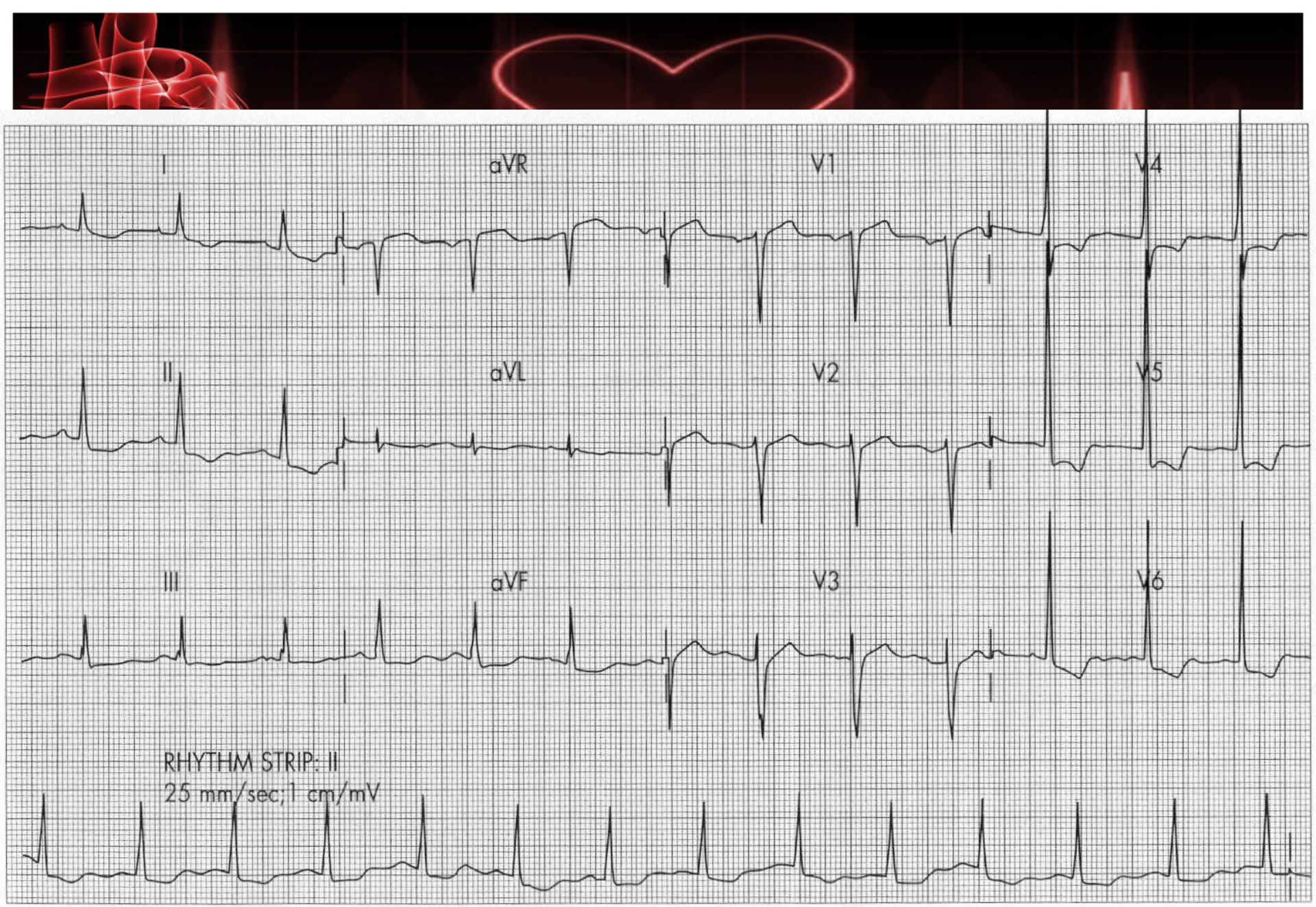
LVH Criteria #1:
Increased limb lead QRS voltage: R in lead I plus S in lead III greater than 25 mm.

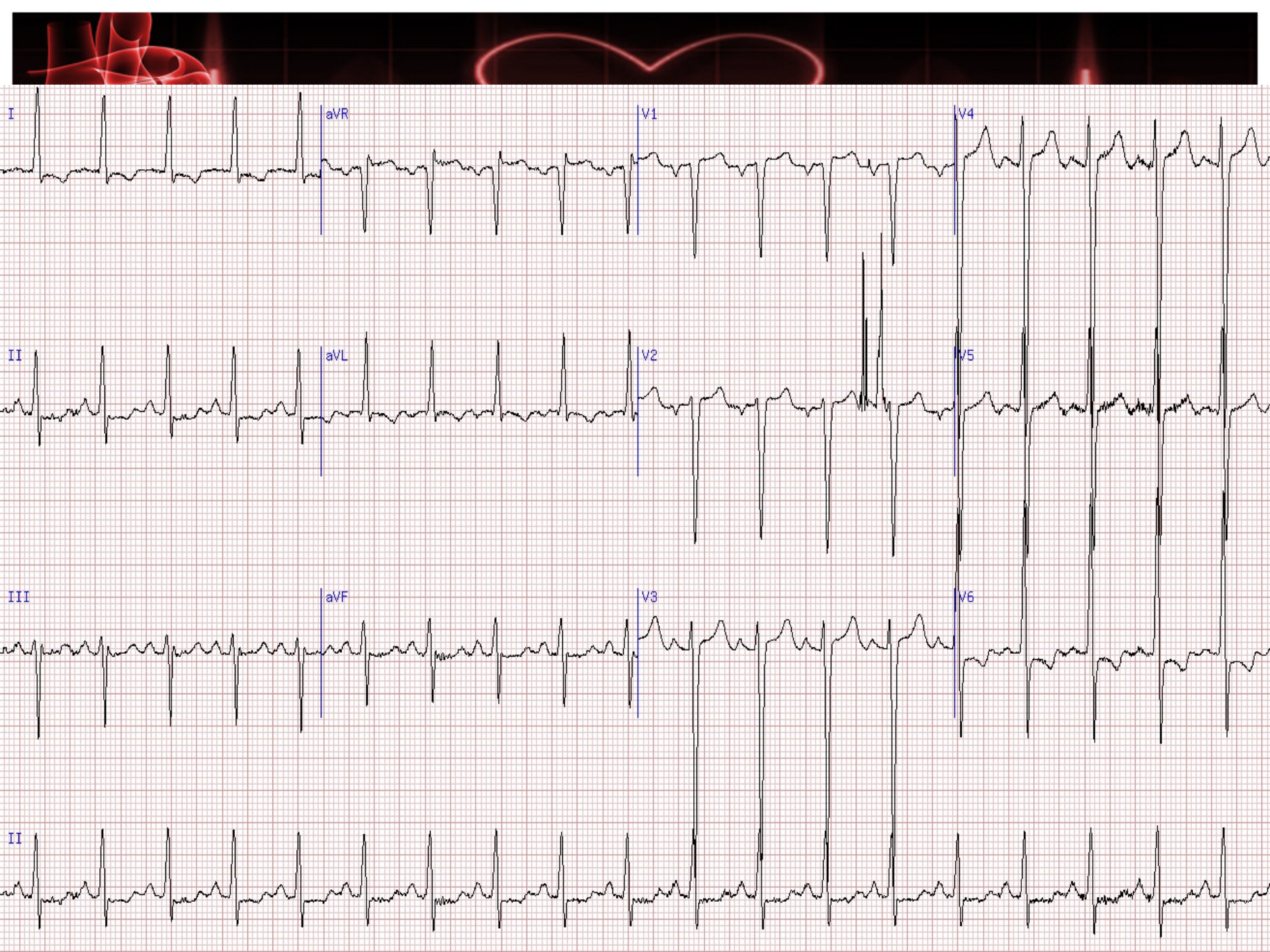
LVH Criteria #2:
Increased precordial QRS voltage: S in lead V1 plus R in either V5 or V6 greater than 35 mm.

LVH Criteria #3:
Typical ST and T abnormalities: ST depression or T wave inversion (or both) in the “lateral” leads (I, aVL, V4-V6)

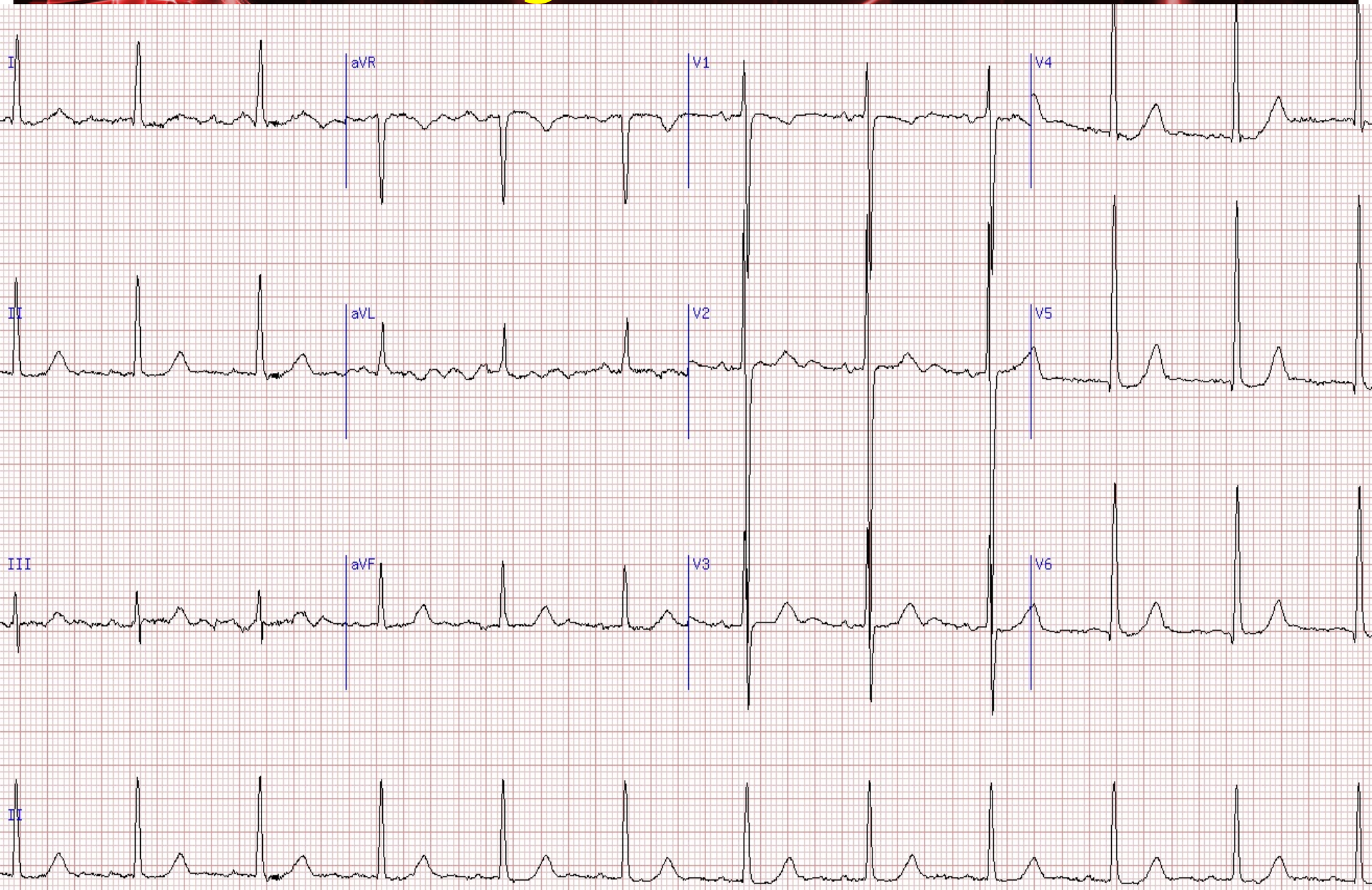
LVH Criteria #4:
Large leftward voltage: R wave in lead AVL greater than 11 mm.

LVH Criteria #5:
Left atrial enlargement: Wide (greater than 0.11 msec) P wave. This criterion is used IN SUPPORT of the diagnosis, not alone.

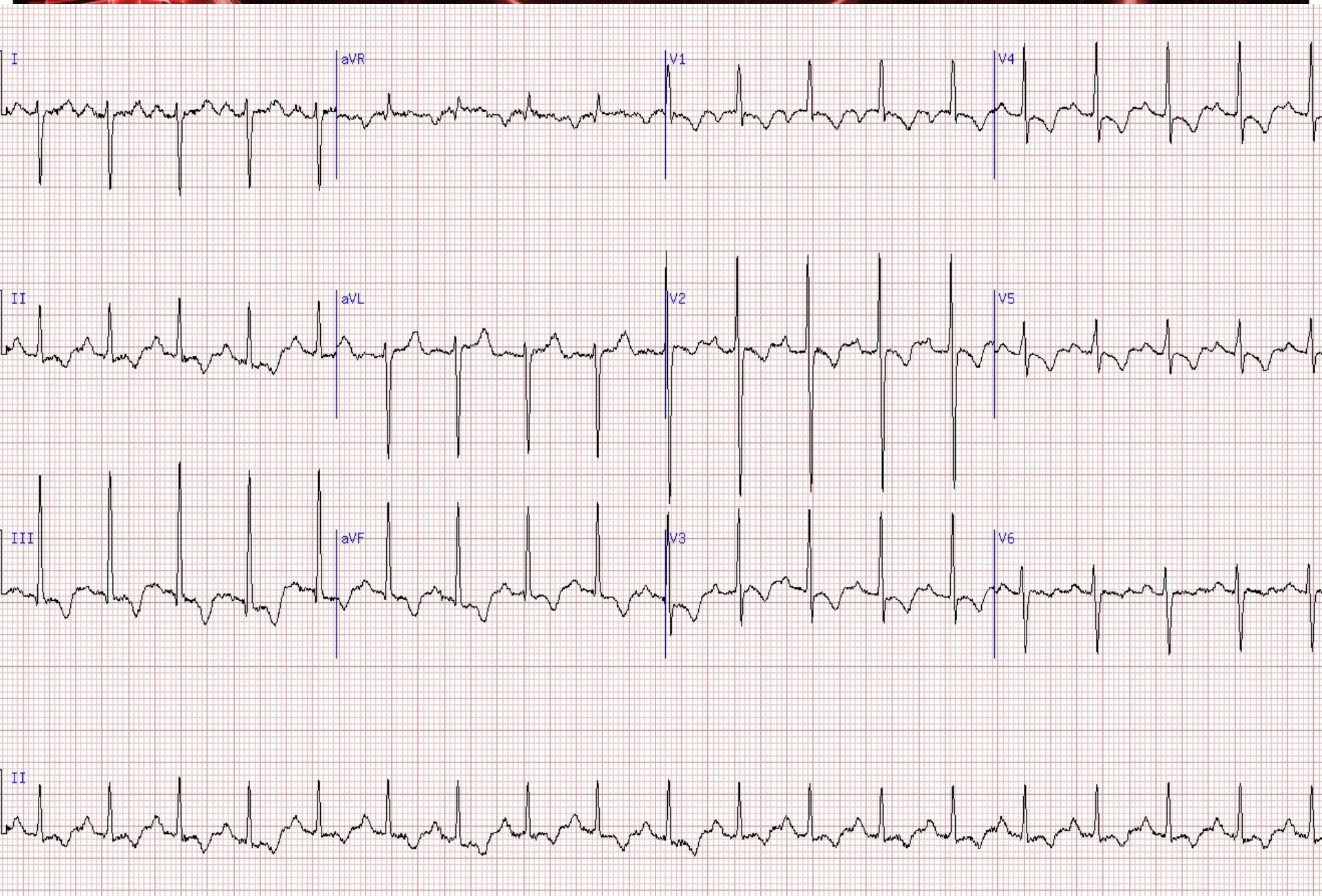

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec;1 cm/mV

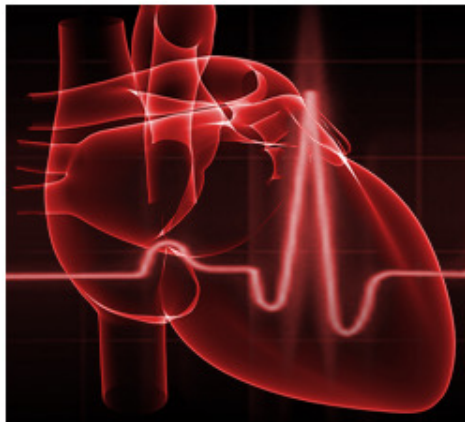

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Right or Left?



Right or Left?



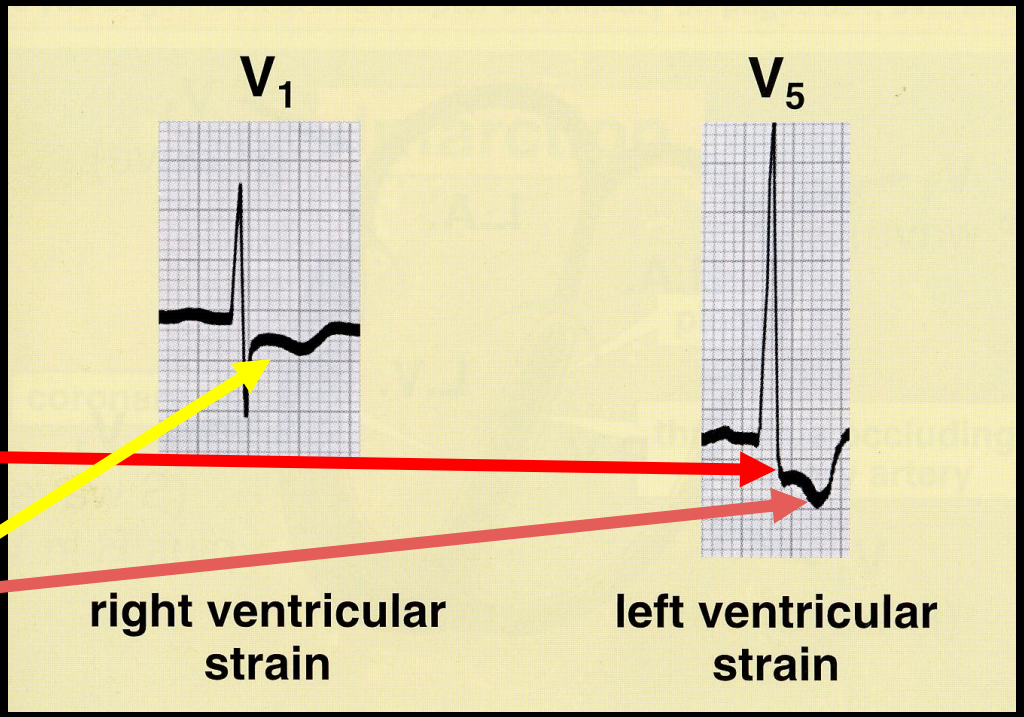


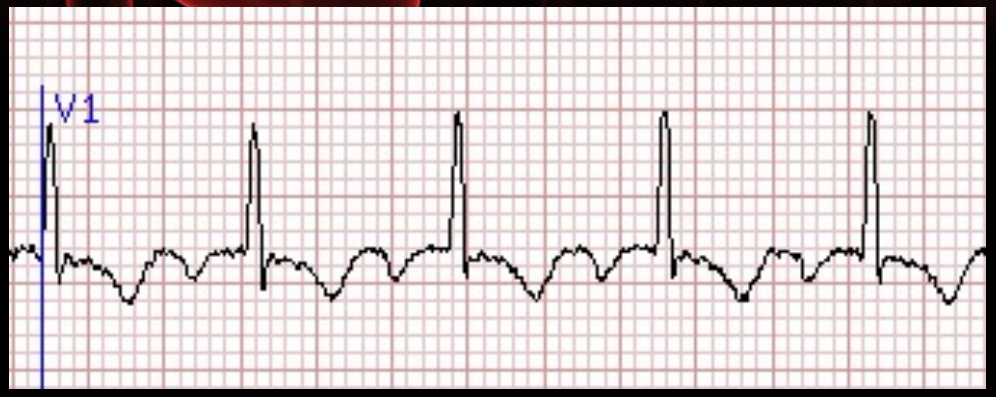
Strain in Hypertrophy

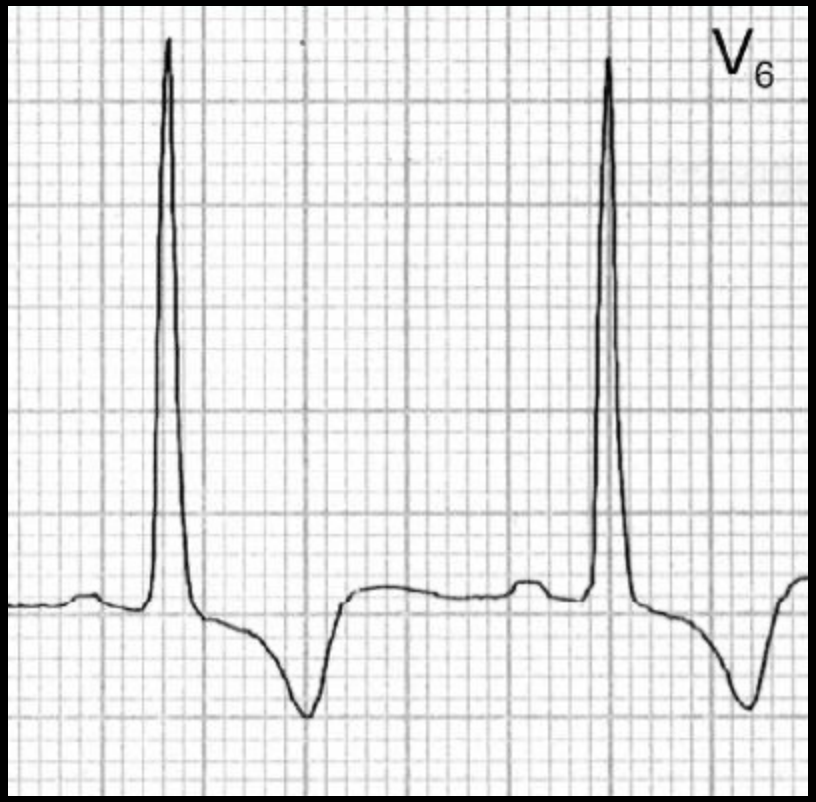
- Strain is usually associated with ventricular hypertrophy since a ventricle that is straining against some kind of resistance will become hypertrophied in its attempt to compensate.
- Ventricular strain depresses the ST segment, which generally humps upward in the middle of the segment.

Strain

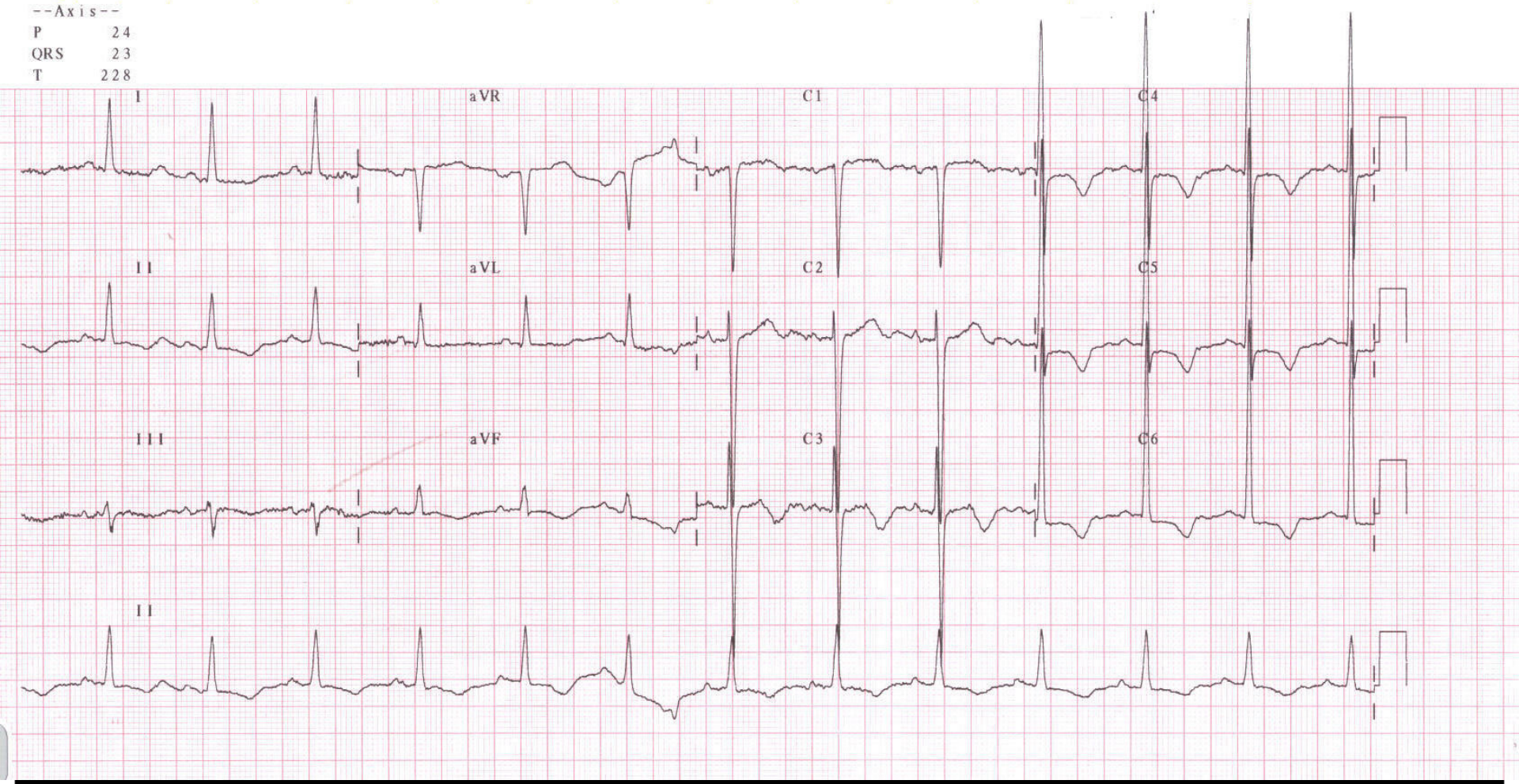
- Look for these changes when hypertrophy is present to determine if there is strain present:
  - Downsloping ST segment & ST depression
  - T-wave inversion




V1


V6

LVH with Strain



Summary

- **RVH Criteria**
  R in V1 > 7 mm or > S wave
  T in V1 inverted
  Right axis deviation
  S waves in V5-V6

- **LVH Criteria**
  1) R-I + S-III >25 mm
  2) S-V1-2 + R-V5-6 >35 mm
  3) ST-Ts in left leads
  4) R-L >11 mm
  5) LAE + other criteria
- Positive Criteria:
  1=possible 2=probable 3=definite

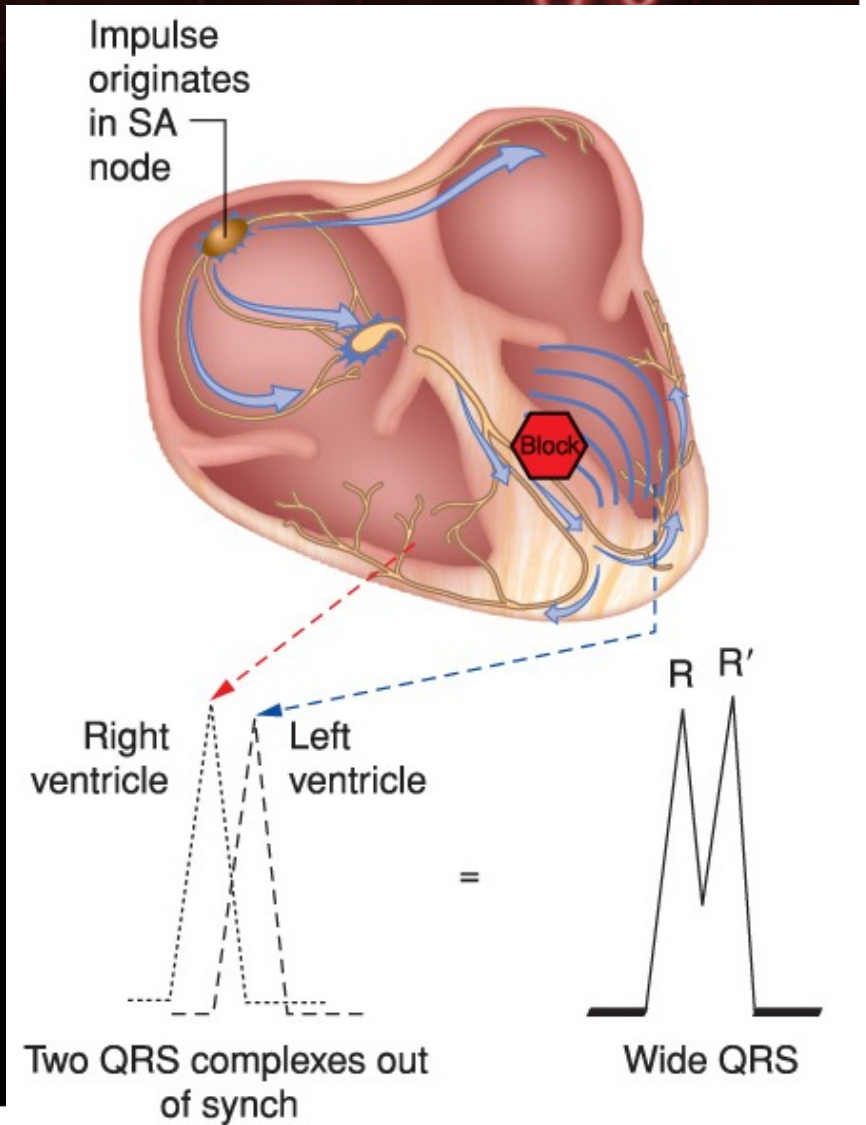
Bundle Branch Blocks

- All forms of bundle branch block involve delays or electrical blockages within the ventricles, they are all types of *intraventricular conduction deficits (IVCD)*. Bundle branch blocks may also be categorized as either partial (incomplete) or complete, depending on which patterns of electrical activity are detected on an EKG.

- When the criteria for BBB are partially met (e.g. < 0.12 sec), this is termed “incomplete bundle branch block”

Bundle Branch Block

- Widened QRS complex
- RR’ configuration in chest leads



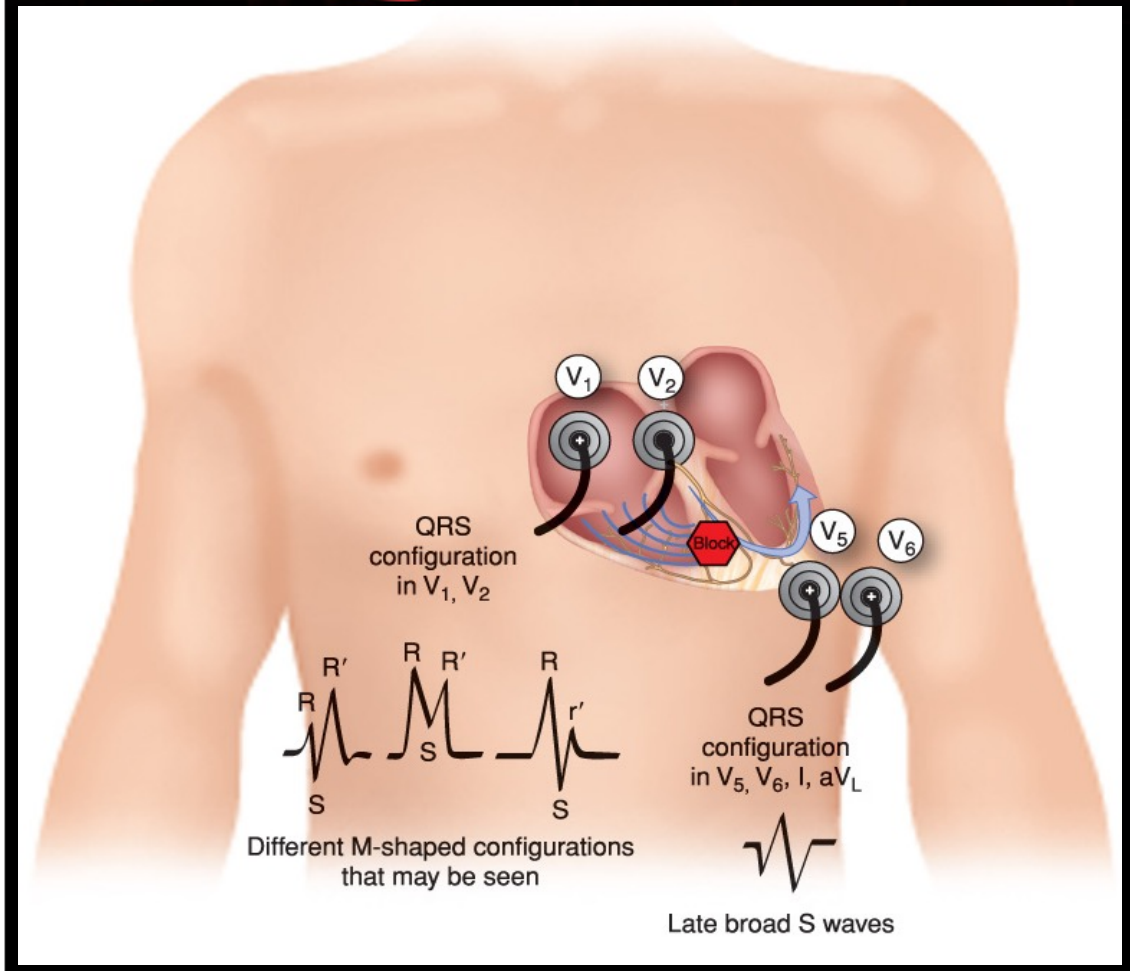
Risk factors and Etiologies of BBB

- Degenerative effects of aging
- Hypertension
- Past heart attack that damaged the heart muscle
- Past viral infection
- Valvular heart disease, particularly calcific aortic stenosis
- One of several heart or lung conditions that could have affected the ventricles (e.g., heart failure or COPD)
- Congenital condition
- Past injury to the chest

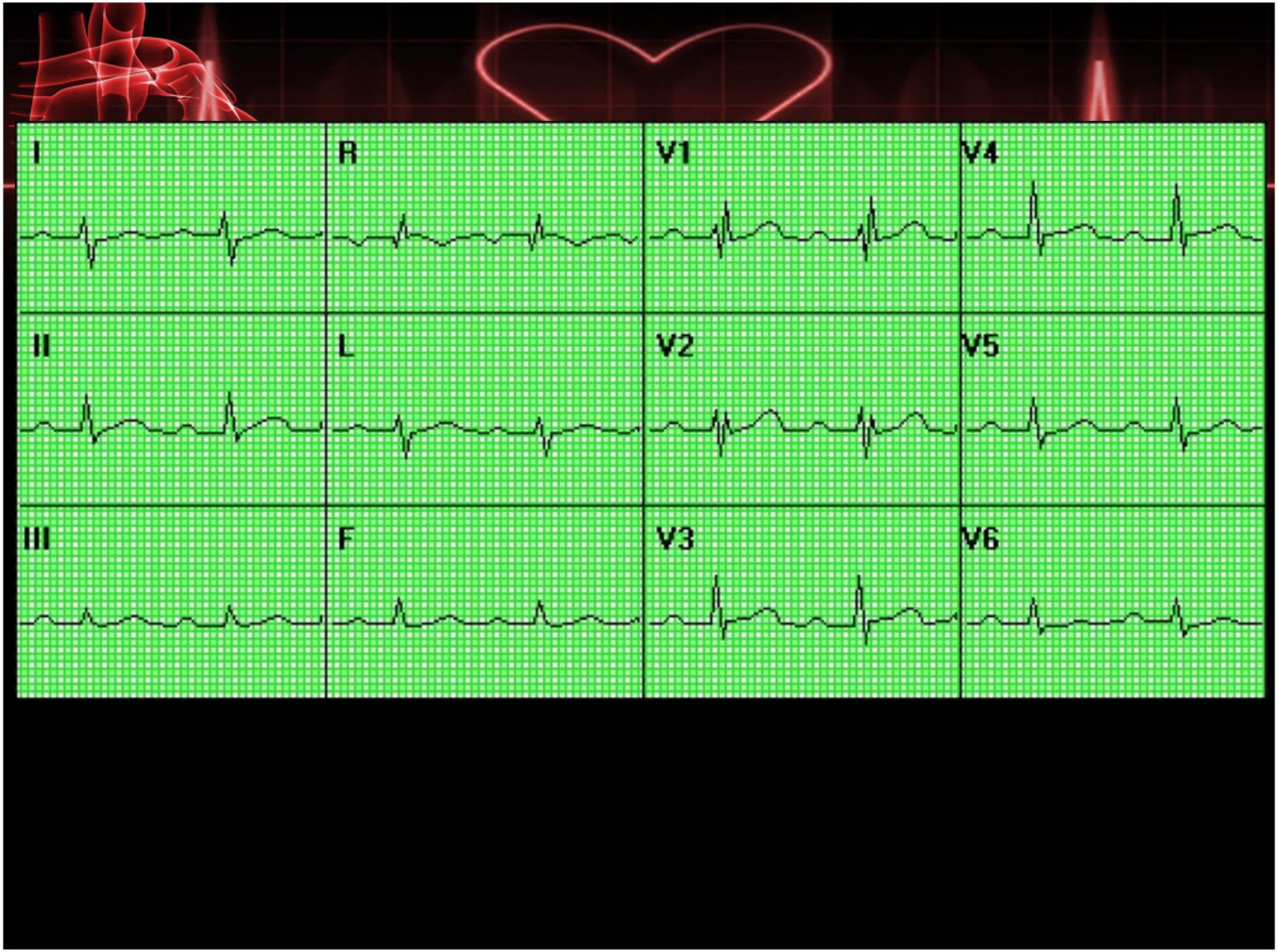
Right Bundle Branch Block

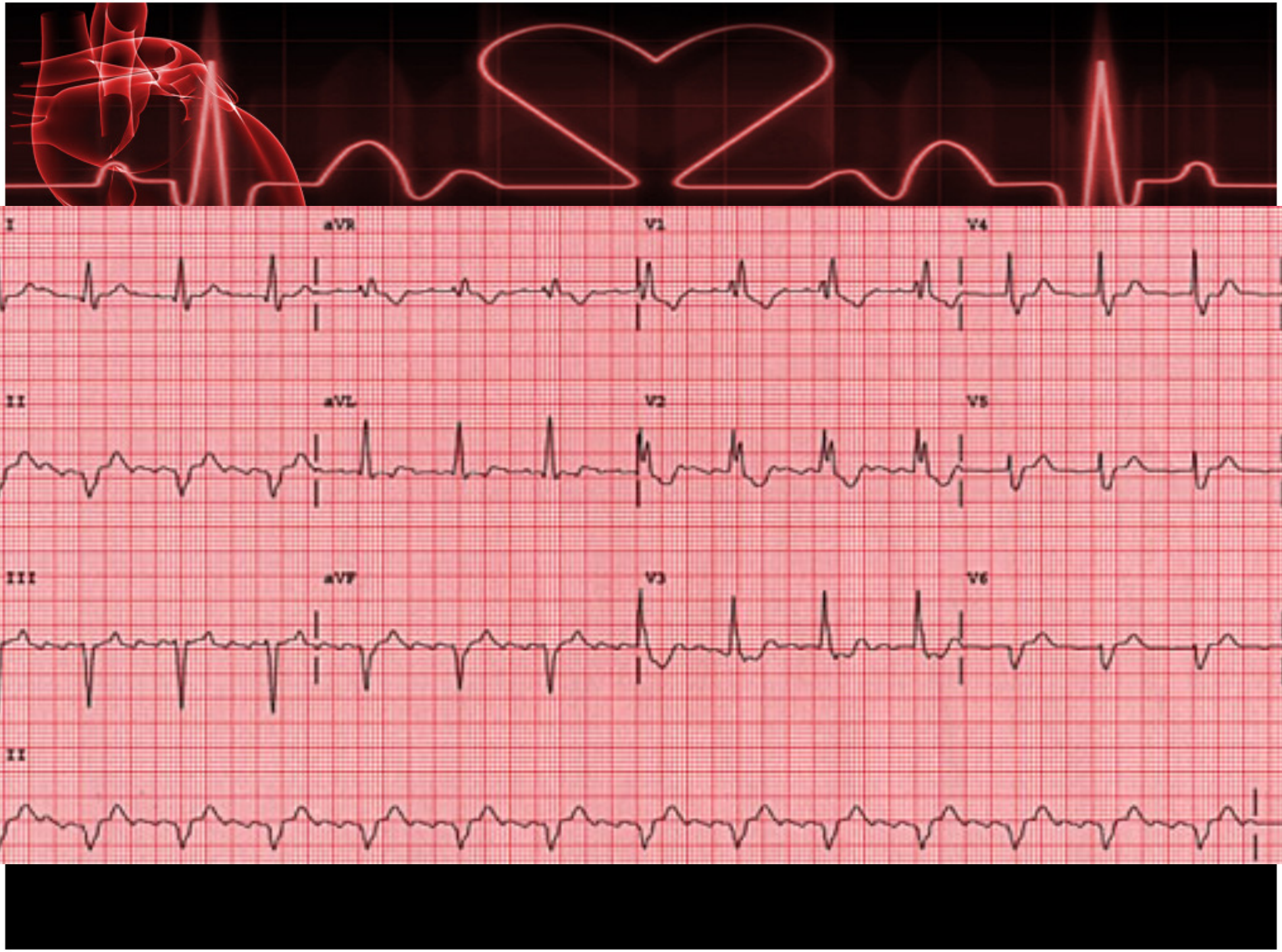
- When the right bundle branch is blocked, activation of the right ventricle begins when electrical activity “spills over” from the left ventricle. Depolarization of the right ventricle is delayed.
- The QRS is prolonged (over 0.10 sec) in right bundle branch block (RBBB). This extra length of the QRS is caused by late activation of the right ventricle, which is then seen after the left ventricle activity. Normally, right ventricle activity is not seen, as it is overshadowed by the larger left ventricle.
- In RBBB, a typical RsR’ wave occurs in lead V1. Also, a wide S wave is seen in leads I, V5, and V6, along with a broad R in lead R. When RBBB occurs in a patient with old or new septal infarction, the initial septal R wave may not be seen in lead V1. Instead, a wide QR complex is seen.

Right Bundle Branch Block



Look for RR’ in leads V_1 or V_2


I
R
V1
V4
II
L
V2
V5
III
F
V3
V6

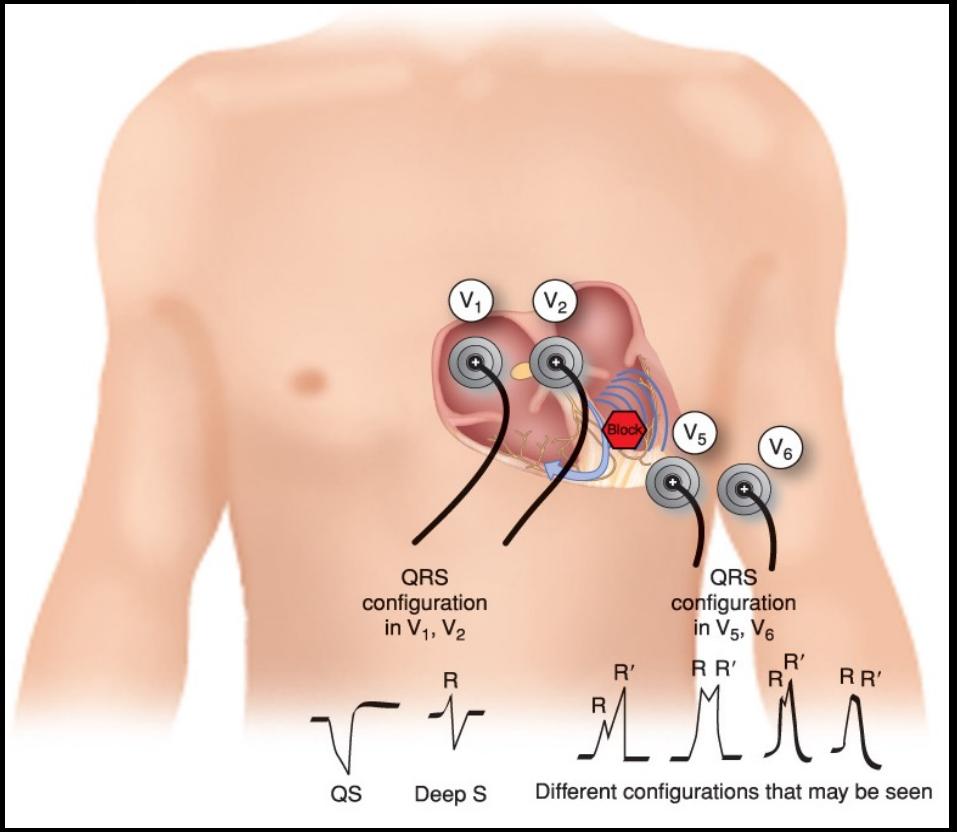

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

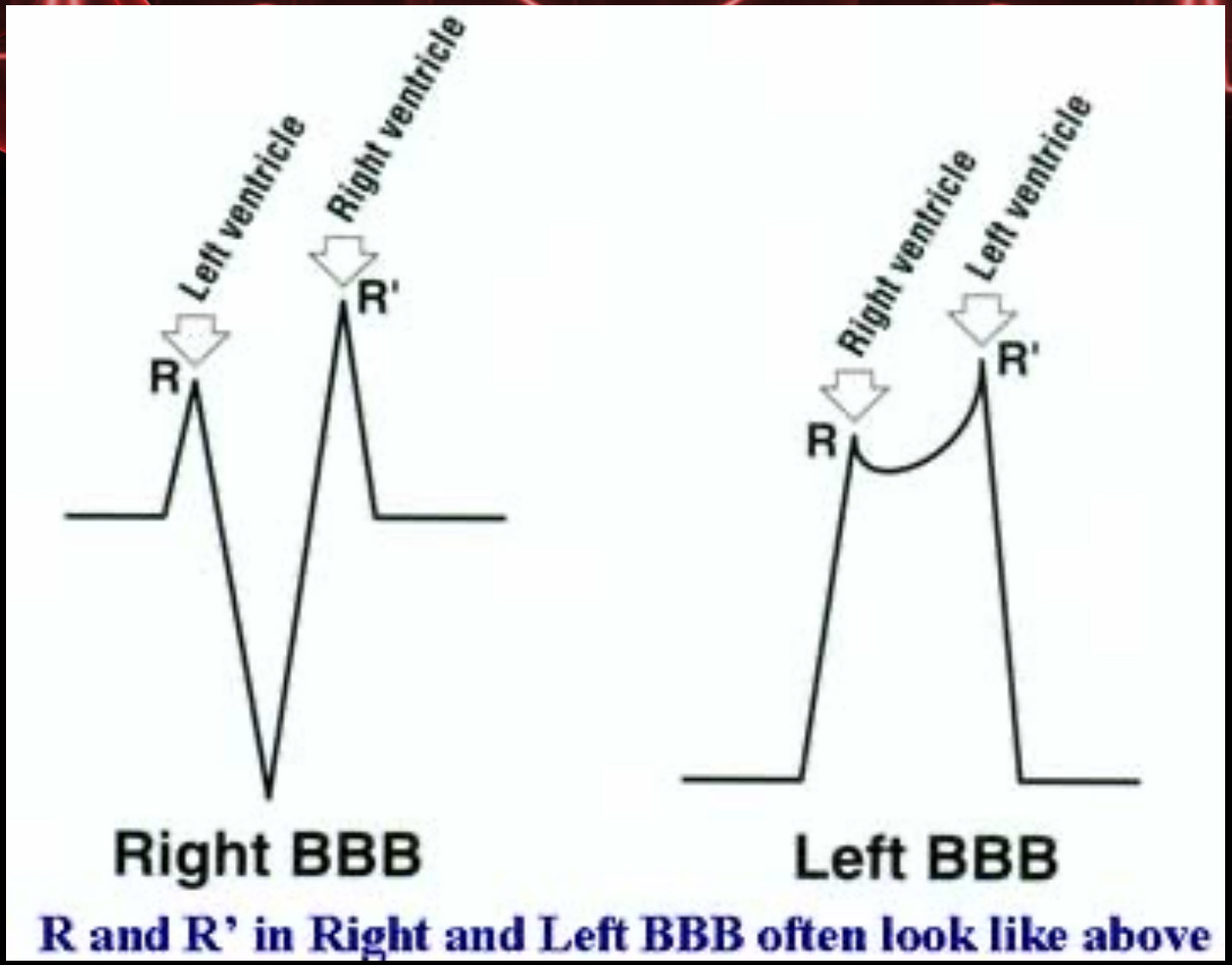
Left Bundle Branch Block

- LBBB usually indicates widespread cardiac disease. When the left bundle is blocked, activation of the left ventricle proceeds through the muscle tissue, resulting in a wide (>.12 msec) QRS complex.
- In left bundle branch blockage (LBBB), the QRS usually has the same general shape as the normal QRS, but is much wider and may be notched or deformed. Voltage (height of the QRS complex) may be higher.
- In LBBB, look for wide (possibly notched) R waves in I, L, or V5-V6, or deep broad S waves in V1-V3. There is left axis deviation. “Septal Q waves” sometimes seen in I, L, and V5-V6 disappear in LBBB.
- T waves in LBBB are usually oriented opposite the largest QRS deflection. That is, where large R waves are seen, T waves will be inverted. ST segment depression may occur.

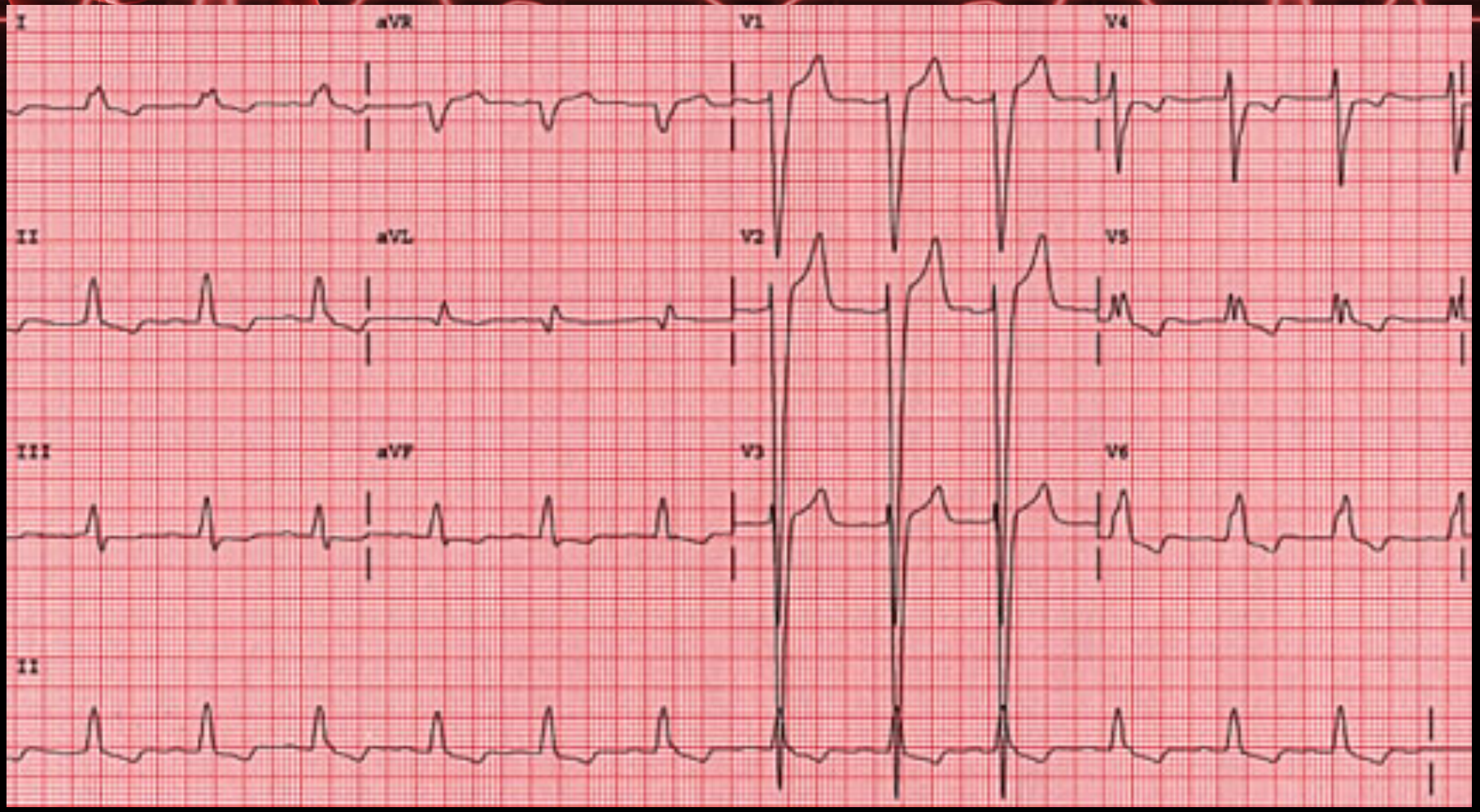
Left Bundle Branch Block

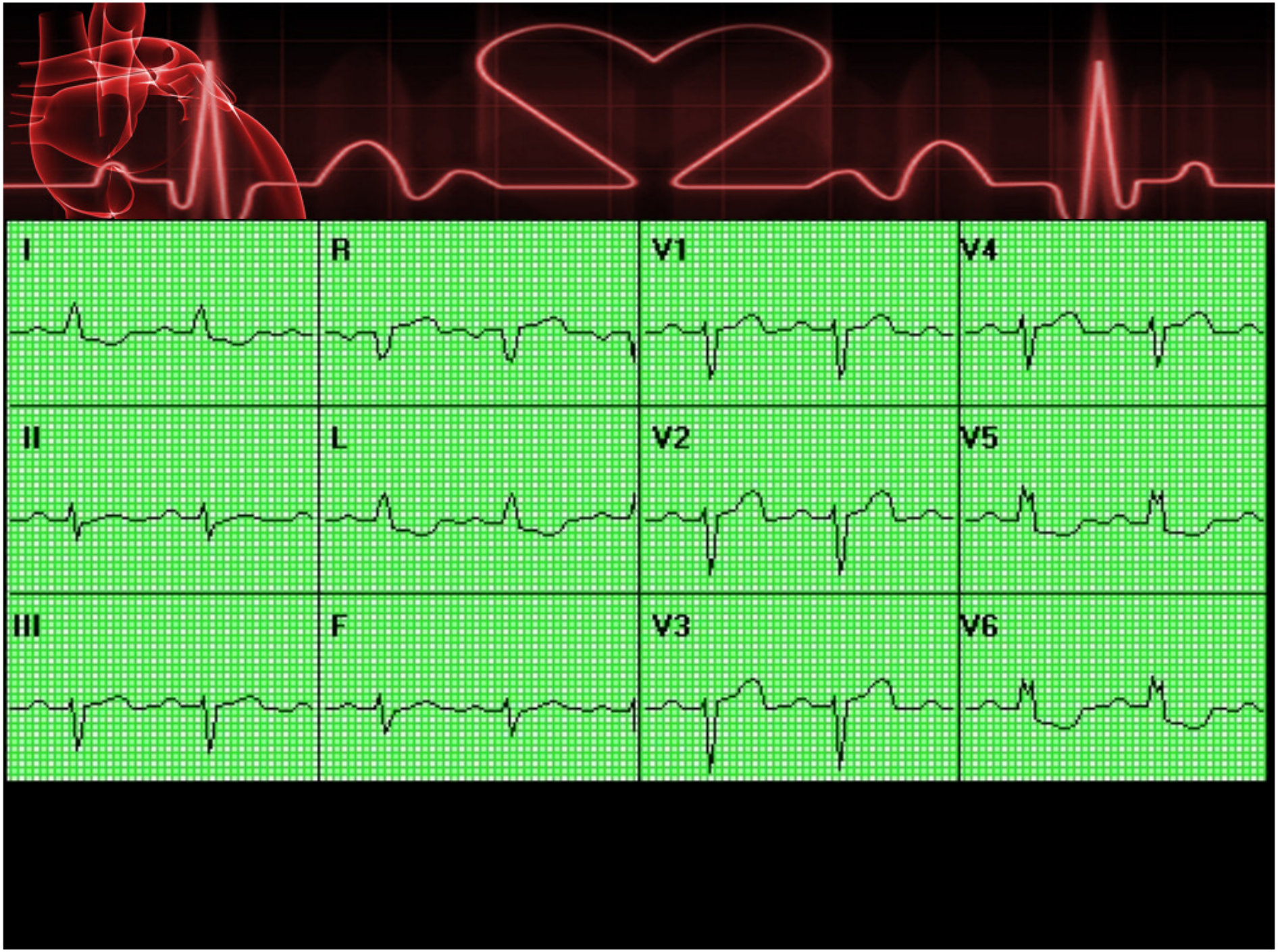
Look for RR’ in leads V_5 or V_6




Left ventricle
Right ventricle
R
R'
Right BBB
Right ventricle
Left ventricle
R
R'
Left BBB
R and R' in Right and Left BBB often look like above

LBBB




I
R
V1
V4
II
L
V2
V5
III
F
V3
V6

Diagnosis of Infarction with BBB

- This is difficult, but not necessarily impossible. Look for ST-T wave changes or new Q waves in left-sided leads (I, aVL, V6) with LBBB.
- New onset of LBBB is typically representative of an MI which must be R/O.
- Evidence of infarction (Q waves, ST-T changes) is easier to see with RBBB.

EKG PEARLS

- When you see a “normal” looking EKG on a test, start looking for:
  - Hyperkalemia :Peaked T waves
  - Hypokalemia : U waves
  - Hypomagnesimia : Prolonged QT
  - Hypercalcemia: Shortened QT
  - WPW : short PR, slurring of upstroke qrs
  - Hypothermia : Osborne J waves (notched downstroke QRS; reversed delta waves)
  - TCA overdose : S-tach, widening QRS, slurring of the terminal rS in aVR
  - Axis deviation & Hemiblocks : LAFB, LPFB

EKG PEARLS

- Usefulness of aVR & V1
  - Tall R wave in V1
    - Left main stem MI
    - RBBB
    - WPW
    - Posterior wall MI
    - Severe RV strain: PE, pneumothorax, severe COPD
  - aVR is normally flipped/negative polarity
    - Slurring terminal rS in TCA OD
    - PR elevation in pericarditis
- Diffuse ST elevation: think pericarditis

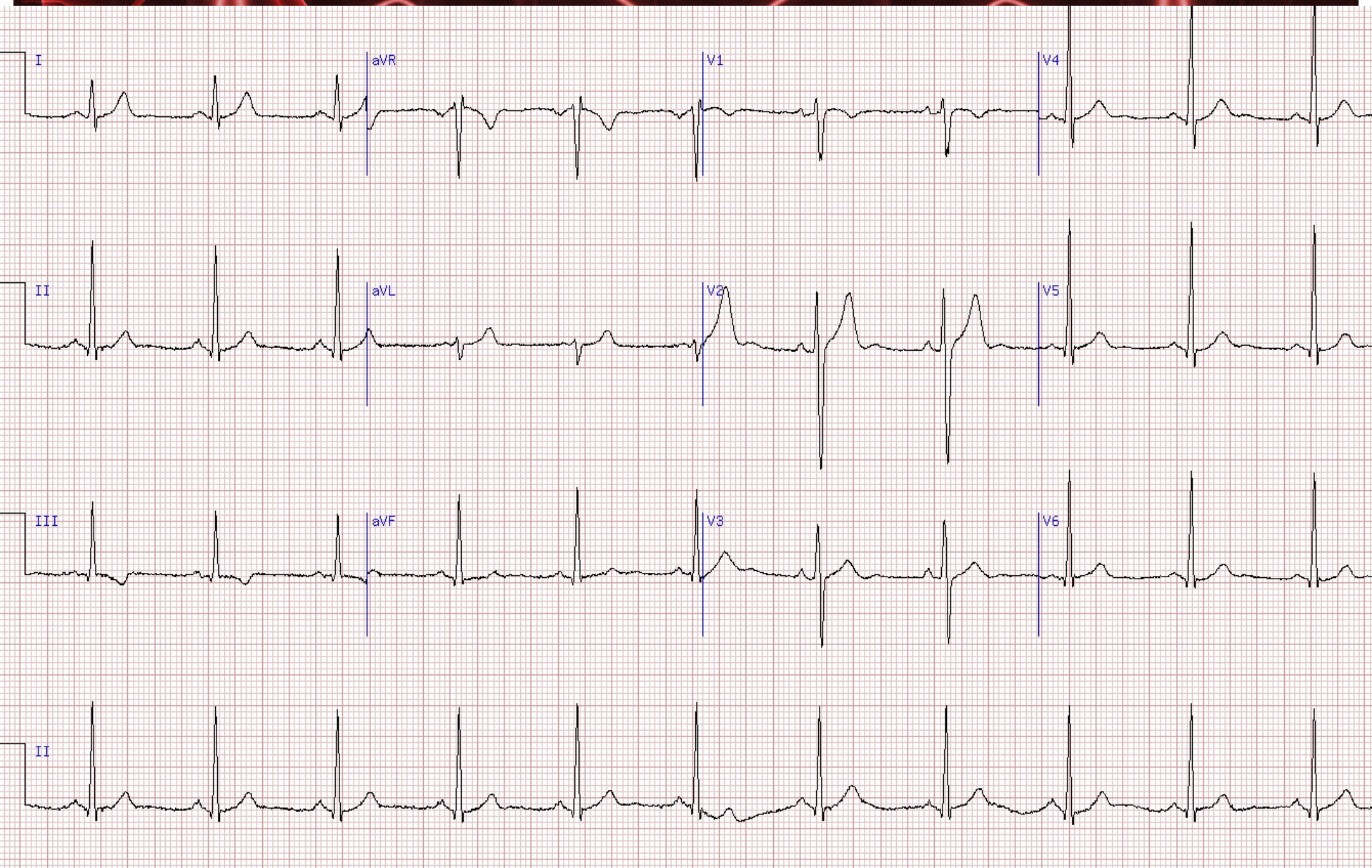
More Practice Time

Take Home Pointers

1. Use a Systematic Approach
   a. Approach your analysis to the 12 lead EKG the same way every time.
2. Identify Lethal Rhythms first
3. Cover up the computer interpretation
4. Determine if the rhythm regular or irregular
5. Don’t take forever trying to read the EKG
6. Look at every EKG you can to get more comfortable reading them

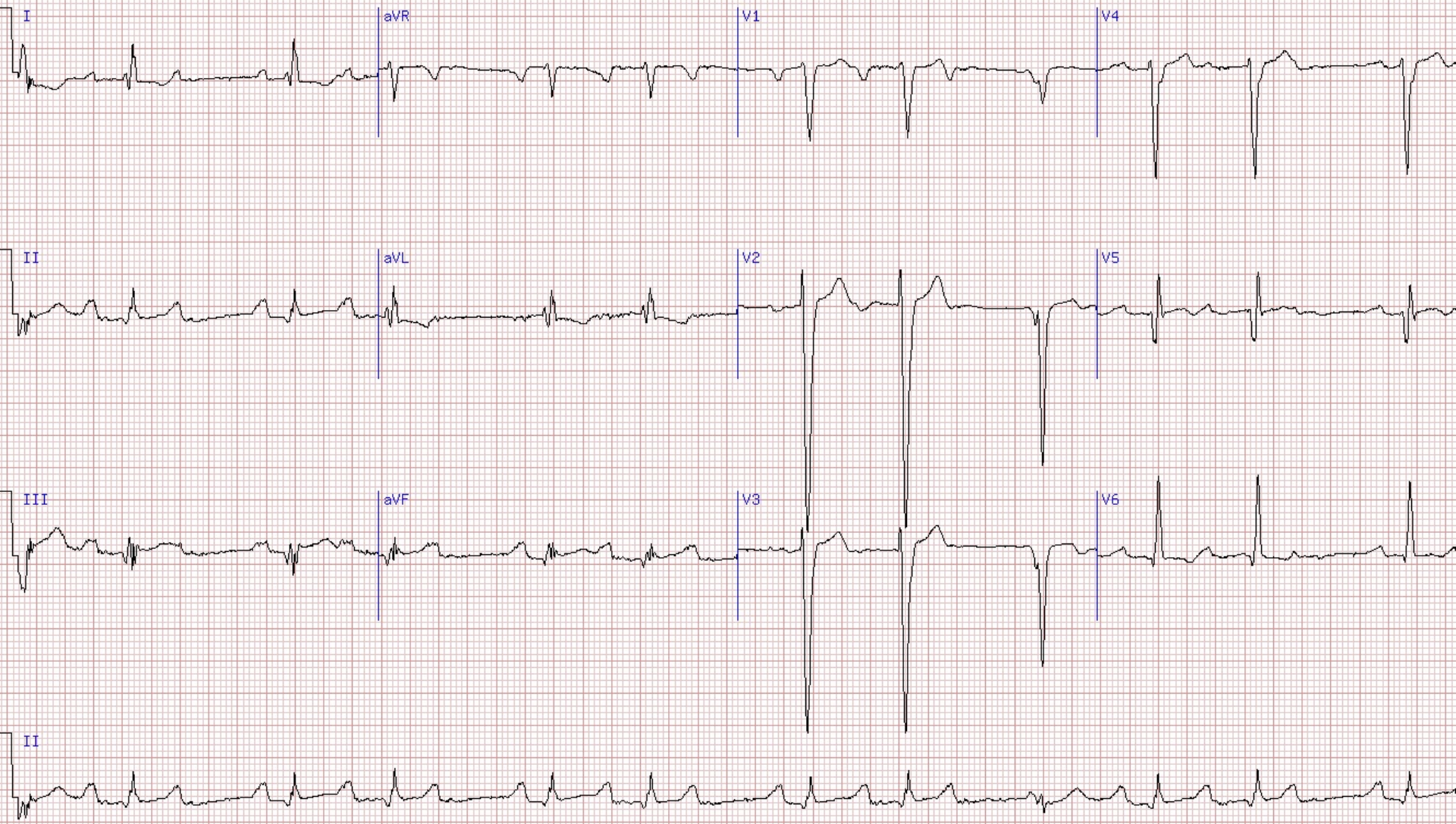
What Do you See?

Practice EKG 1



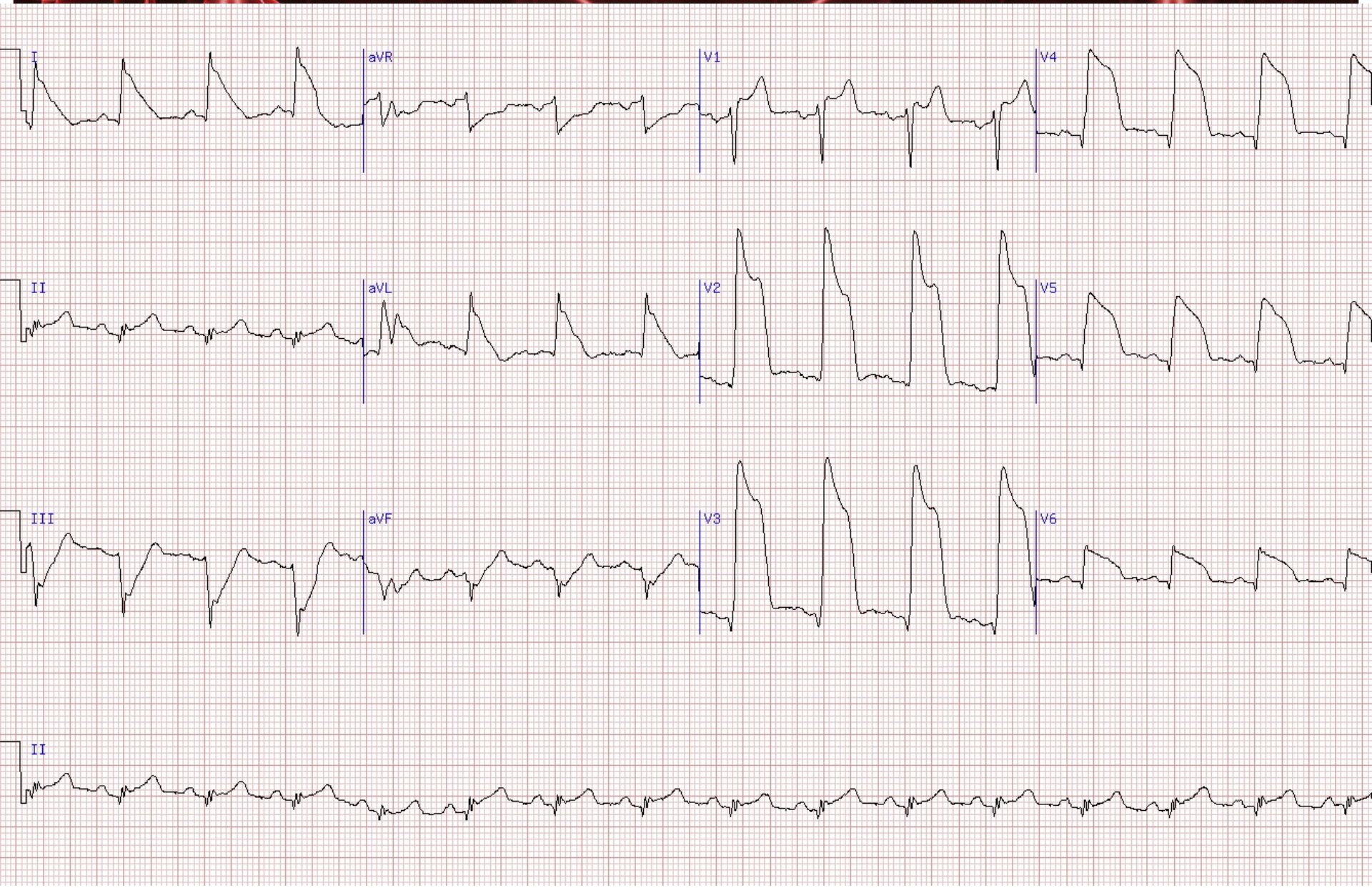
Practice EKG 2

What Do you See?



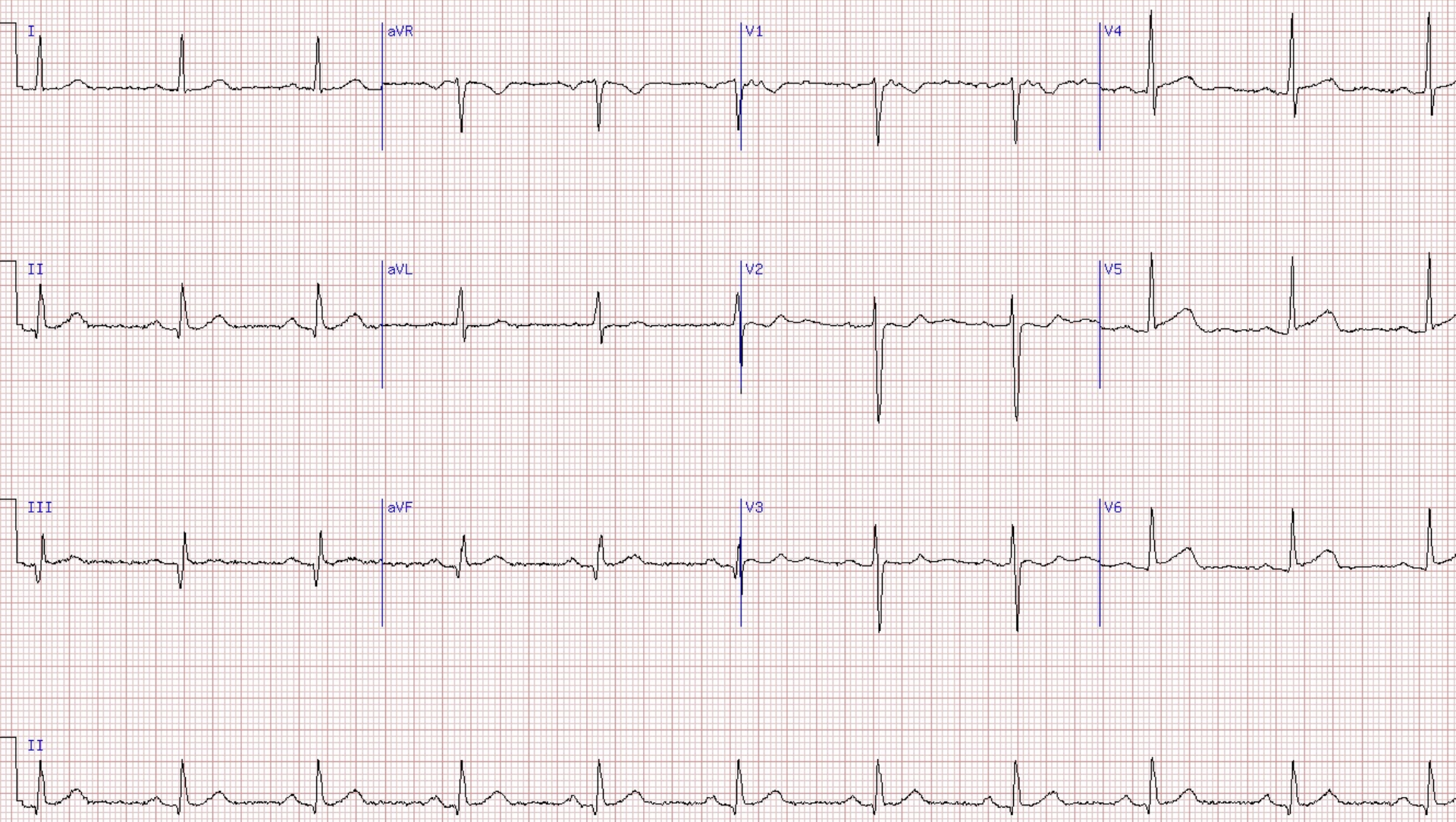
Practice EKG 3

What Do you See?



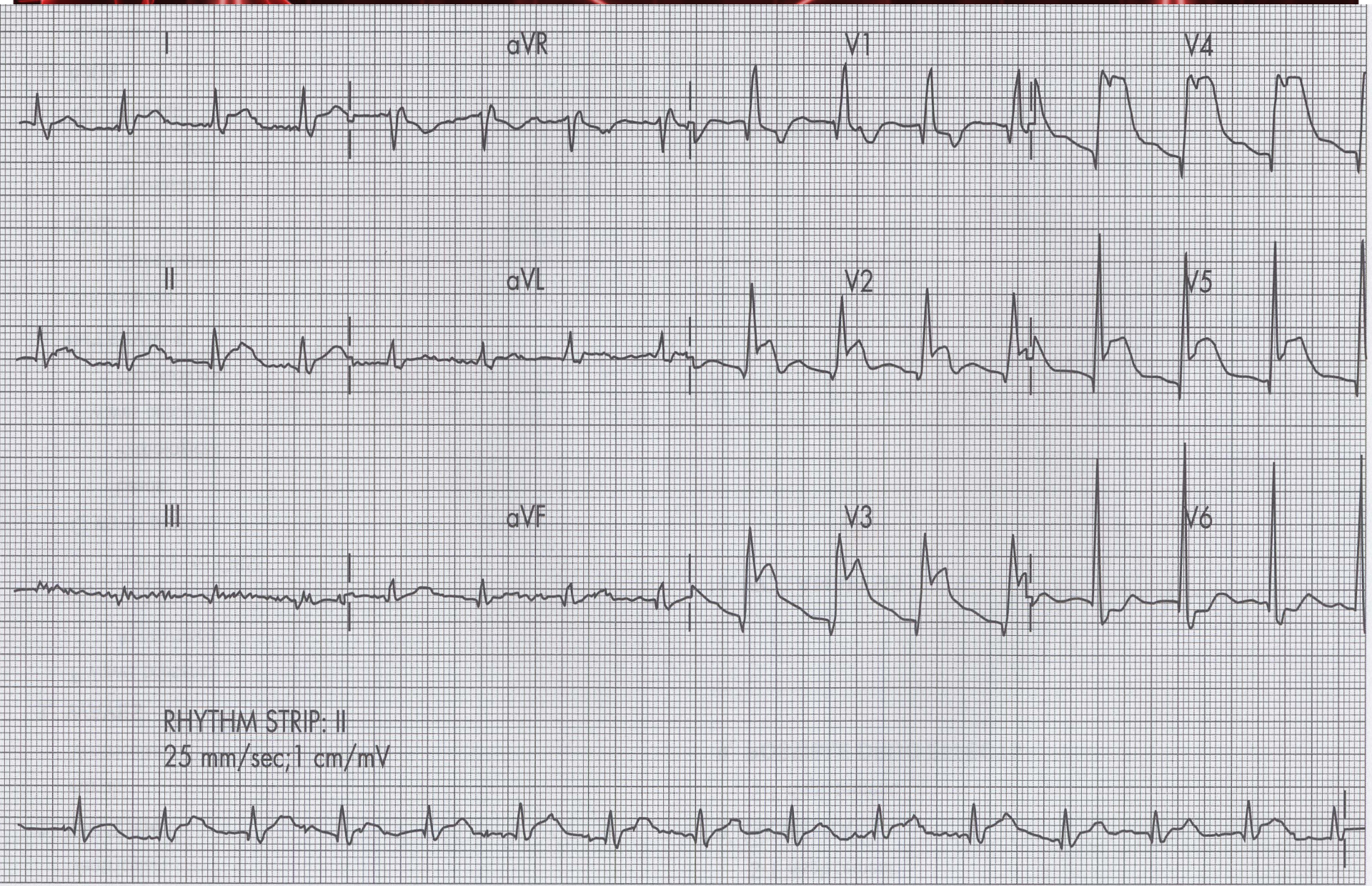
What Do you See?

Practice EKG 4



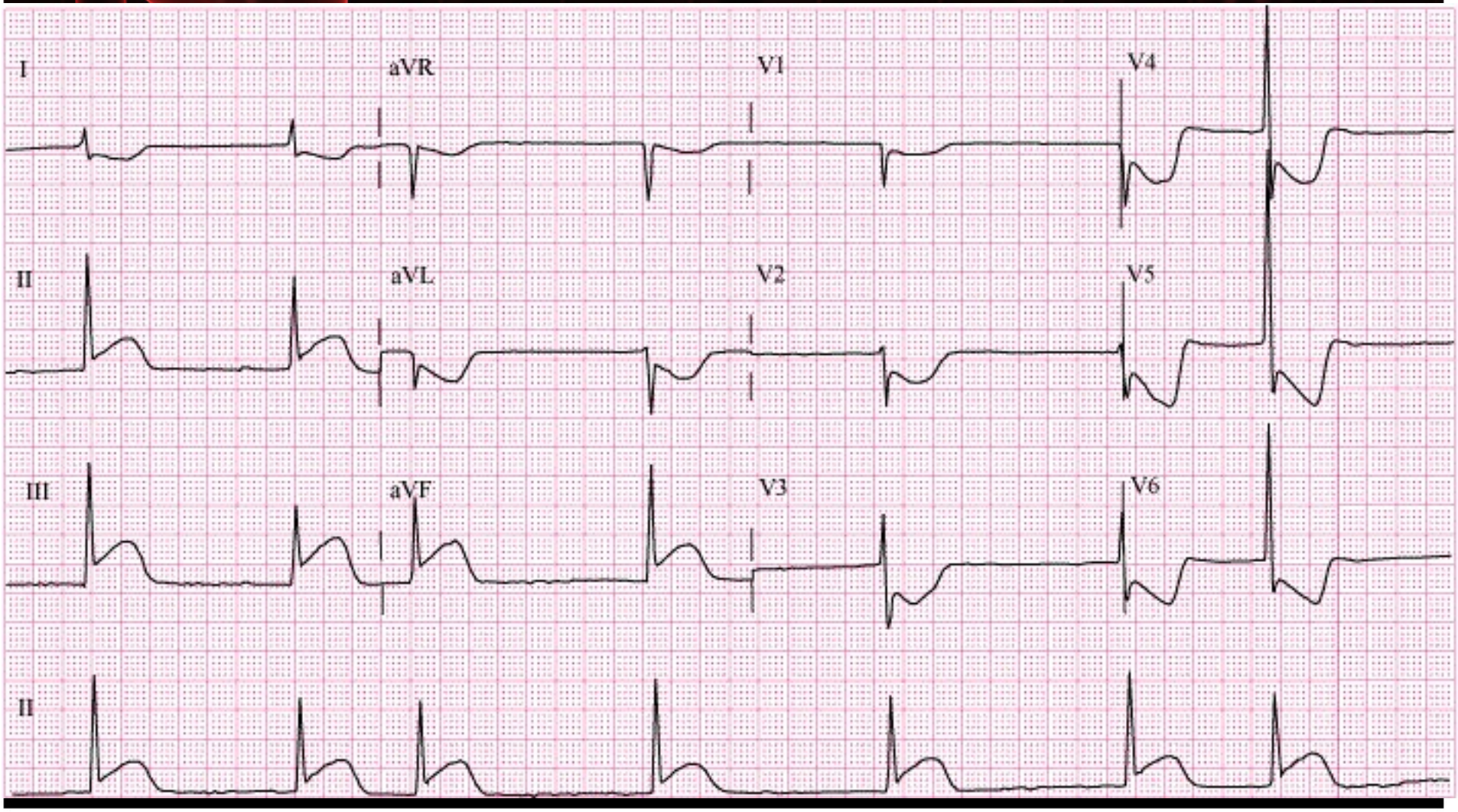
What Do you See?

Practice EKG 5



What Do you See?

Practice EKG 6



Questions????

References

- https://litfl.com/ecg-library/ecg-references/
- https://www.uptodate.com/contents/ecg-tutorial-basic-principles-of-ecg-analysis
- https://www.healio.com/cardiology/learn-the-heart/ecg-review/ecg-interpretation-tutorial/stemi-mi-ecg-pattern

If you would like further EKG information, send me an email at:
benhwa@hotmail.com