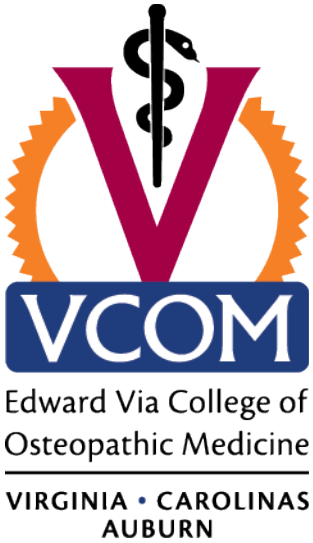
Pathophysiology of AMI and Associated EKG findings- A Case Based Presentation



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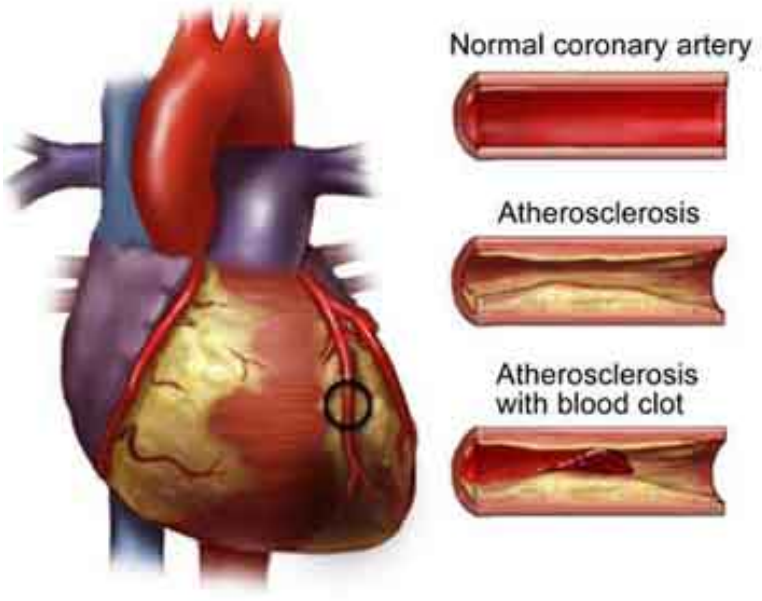
Objectives

- Providers will understand EMS precautions when treating uncommon presentation acute myocardial infarction.
- Providers will be able to describe the vascular anatomy of the heart and identify landmarks on a plastinated cardiac specimen
- Providers will be able to form a systematic method to reading EKGs
- Providers will be able to identify EKG changes in acute myocardial infarctions
- Providers will be able to identify acute vs pathological EKG changes secondary to MI
- Providers will understand the significance of nitroglycerin-induced hypotension with inferior wall acute myocardial infarction

Ischemic Heart Disease

- Ischemia – Caused by decreased blood flow to an organ
- Usually caused by atherosclerosis of coronary arteries



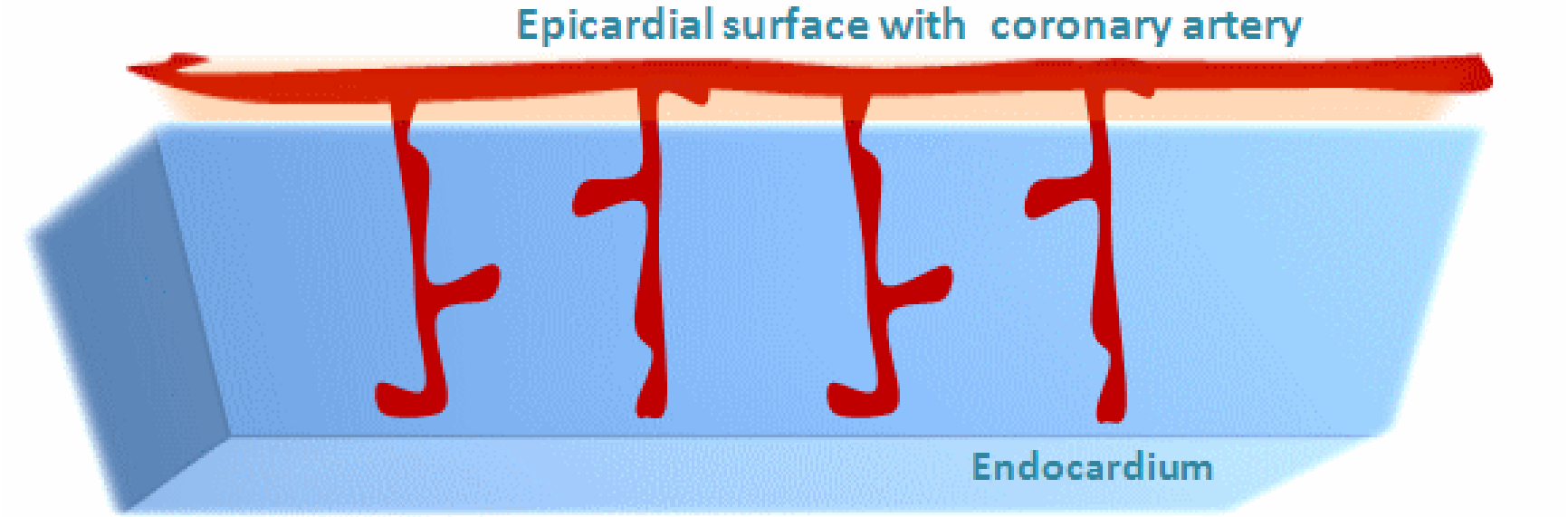
Stable Angina

- Reversible Injury to cardiac cells
- Chest Pain that occurs when the patient undergoes exertion or an emotional response to stress
- Generally occurs when a stenosis of 70% or greater is noted

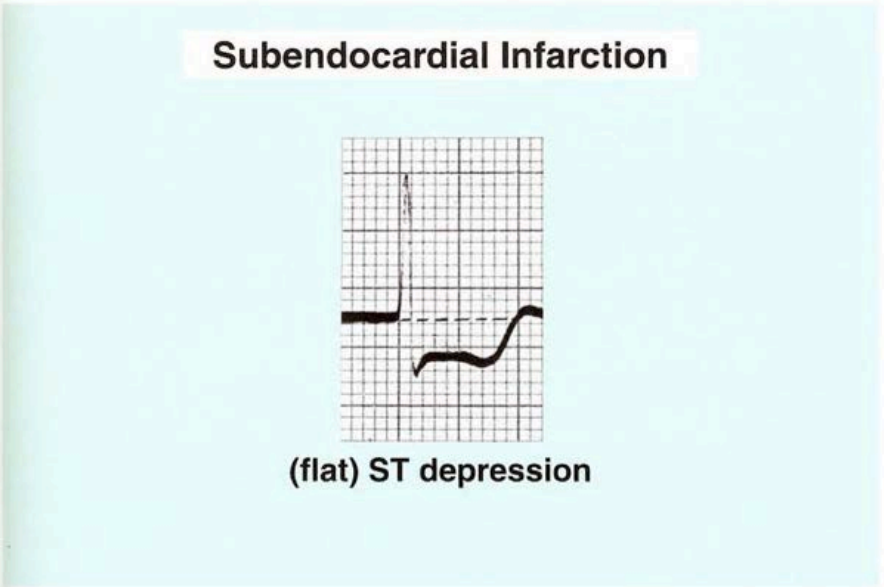
Presentation of Stable Angina

- Patients will present with classic signs and symptoms of MI including
  - Diaphoresis
  - Shortness of Breath
  - And Chest pain that radiates to the left arm or jaw
    - the key here is that it lasts less than 20 minutes

Coronary Artery Blood Flow



EKG Changes



Subendocardial infarction causes flat depression of the ST segment; however, any significant ST depression (in leads where the QRS is upright) indicates compromised coronary blood flow until proven otherwise.

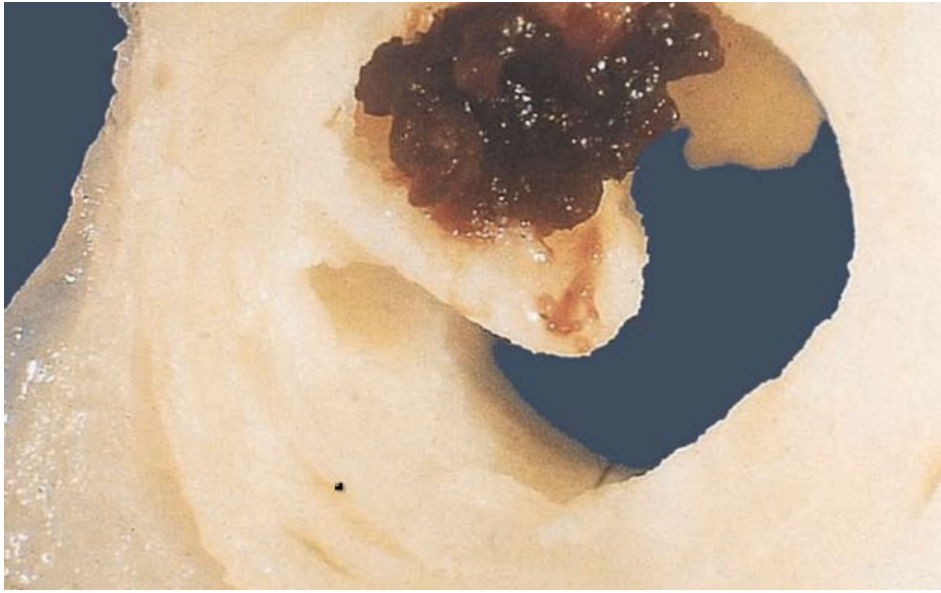
Treatment of Stable Angina

- Physical Rest or removal of emotional stimulus

- Nitroglycerin – Caused through vasodilation of veins – which decreases blood returning to the heart – decreasing the preload and demand on the heart

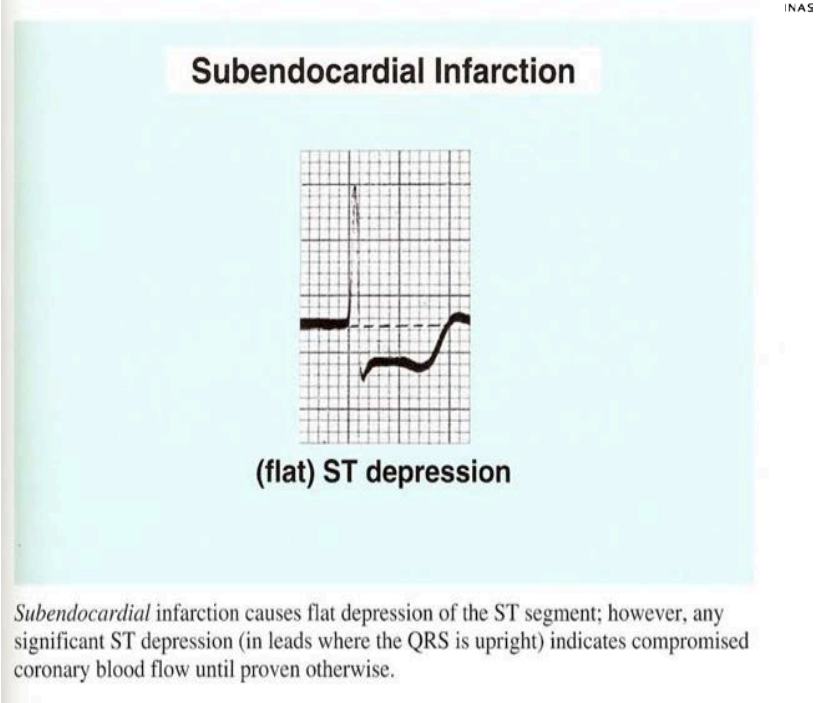
Unstable Angina

- Similar to Stable – except that this chest pain occurs at rest.
- Caused by a rupture of an atherosclerotic plaque which subsequently caused an incomplete occlusion of a coronary artery downstream



EKG

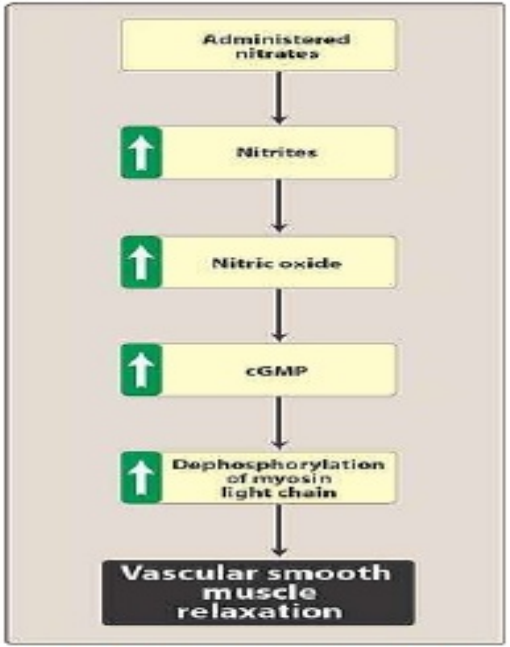
- This shows ST-Segment Depression again
- Relieved by Nitroglycerin
- This particular presentation has a high risk for progression to full myocardial ischemia



Subendocardial infarction causes flat depression of the ST segment; however, any significant ST depression (in leads where the QRS is upright) indicates compromised coronary blood flow until proven otherwise.

A. Mechanism of action (MOA)

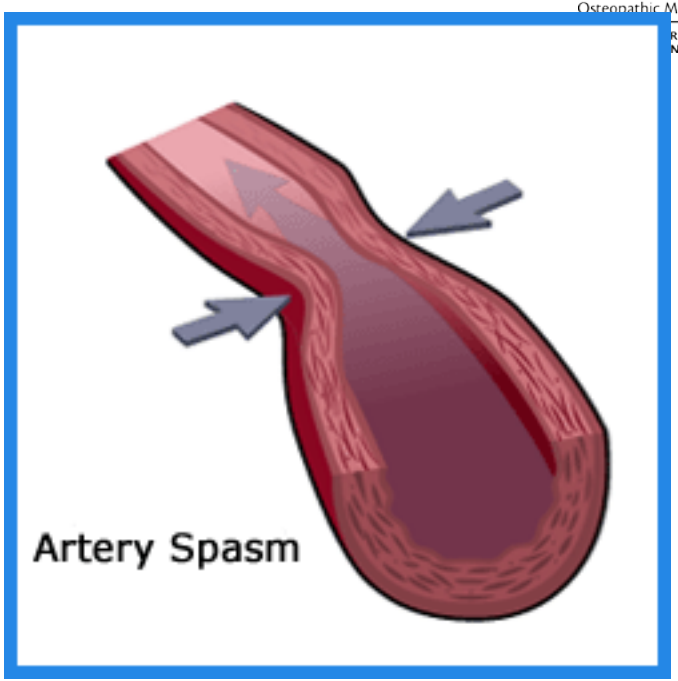
- Nitrates decrease coronary vasoconstriction or spasm and increase perfusion of the myocardium by relaxing coronary arteries.
- In addition, they relax veins, decreasing preload and myocardial oxygen consumption.
- Organic nitrates, such as nitroglycerin, which is also known as glyceryl trinitrate, are thought to relax vascular smooth muscle by their intracellular conversion to nitrite ions, and then to nitric oxide, which in turn activates guanylate cyclase and increases the cells' cyclic guanosine monophosphate (GMP). Elevated cGMP ultimately leads to dephosphorylation of the myosin light chain, resulting in vascular smooth muscle relaxation



Effects of nitrates and nitrites on smooth muscle. cGMP = cyclic guanosine 3', 5'-monophosphate.

Prinzmetal Angina

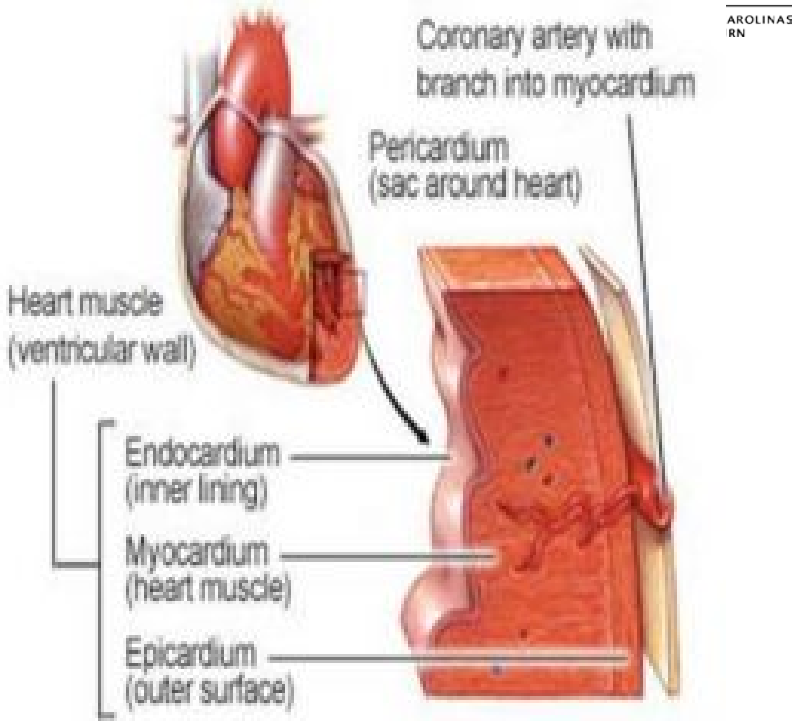
- Unique form of angina caused by spasm of the coronary arteries
- Causes episodic chest pain not related to exercise or exertion



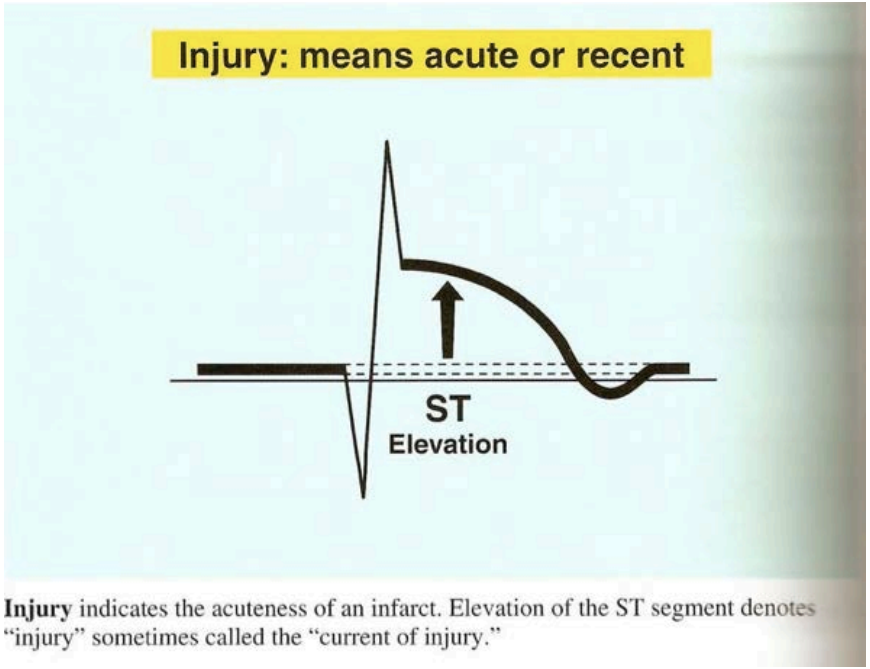
EKG Changes with Prinzmetal

- ST-Segment Elevation

- Why – The artery is clamping down at the proximal end causing transmural ischemia



Transmural Ischemia causes



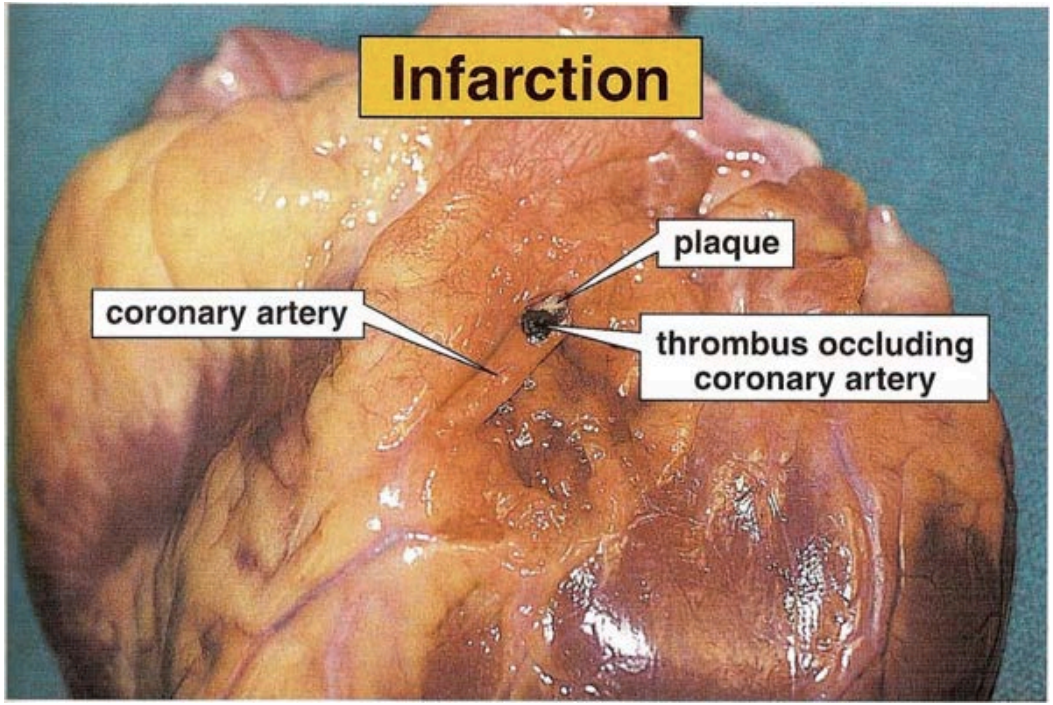
Injury indicates the acuteness of an infarct. Elevation of the ST segment denotes "injury" sometimes called the "current of injury."

Treatment for Prinzmetal angina

- Nitroglycerin – for the same reasons we mentioned above

- Calcium Channel Blockers

Myocardial Infarction



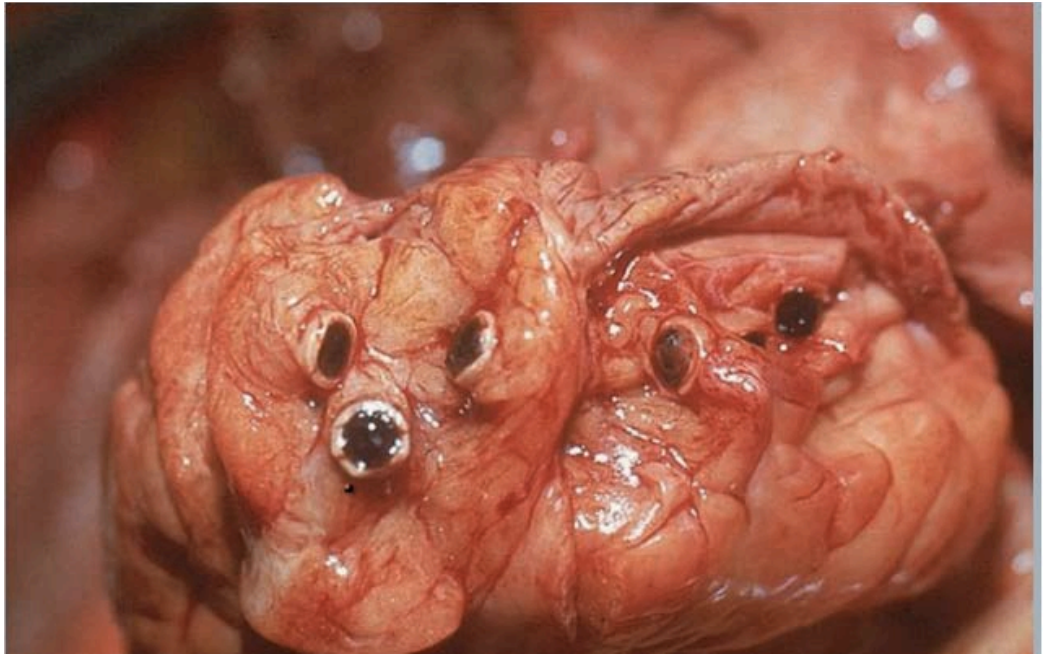
Myocardial Infarction

- Generally -a ruptured atherosclerotic plaque lodges in the coronary artery causing complete occlusion

- Any type of occlusion can cause an MI – Including
  - Vasospasm
  - Fatty emboli
  - Vasculitis
- - Drug induced MI – Cocaine or other vasoconstrictor

Myocardial Infarction

- Leads to Necrosis and irreversible cell death
- Early Diagnosis by the prehospital provider is priceless

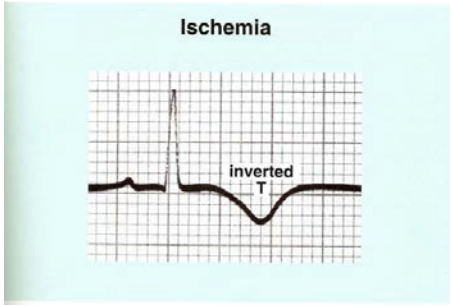


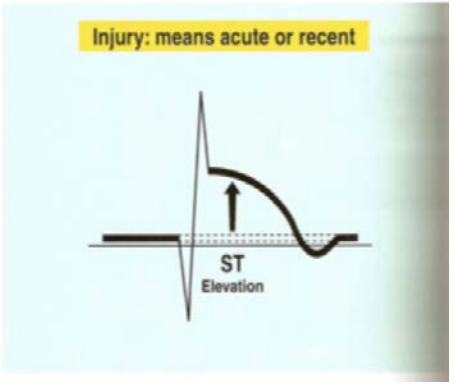
Patient Presentation of MI

- Substernal, crushing chest pain that radiates to the jaw or left arm
- Shortness of breath – **WHY**
- Diaphoresis
- Will the symptoms of a true MI be fully relieved with Nitroglycerin?

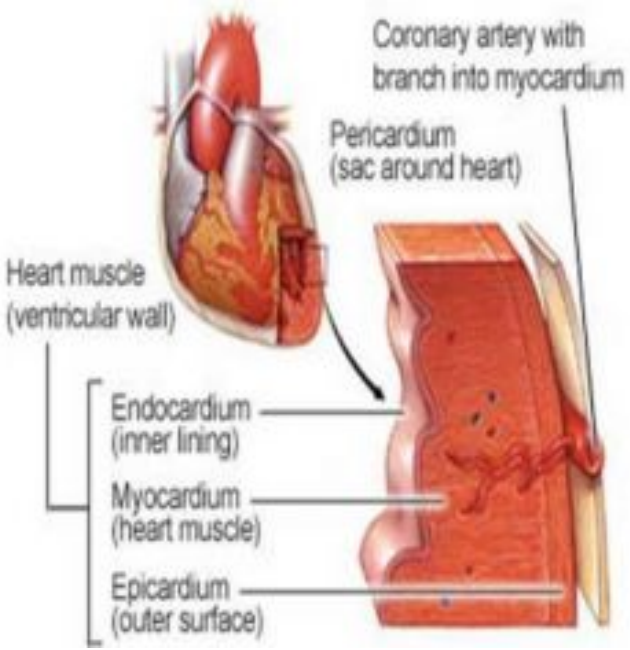
EKG Changes

Initial	Progressing to	Finally





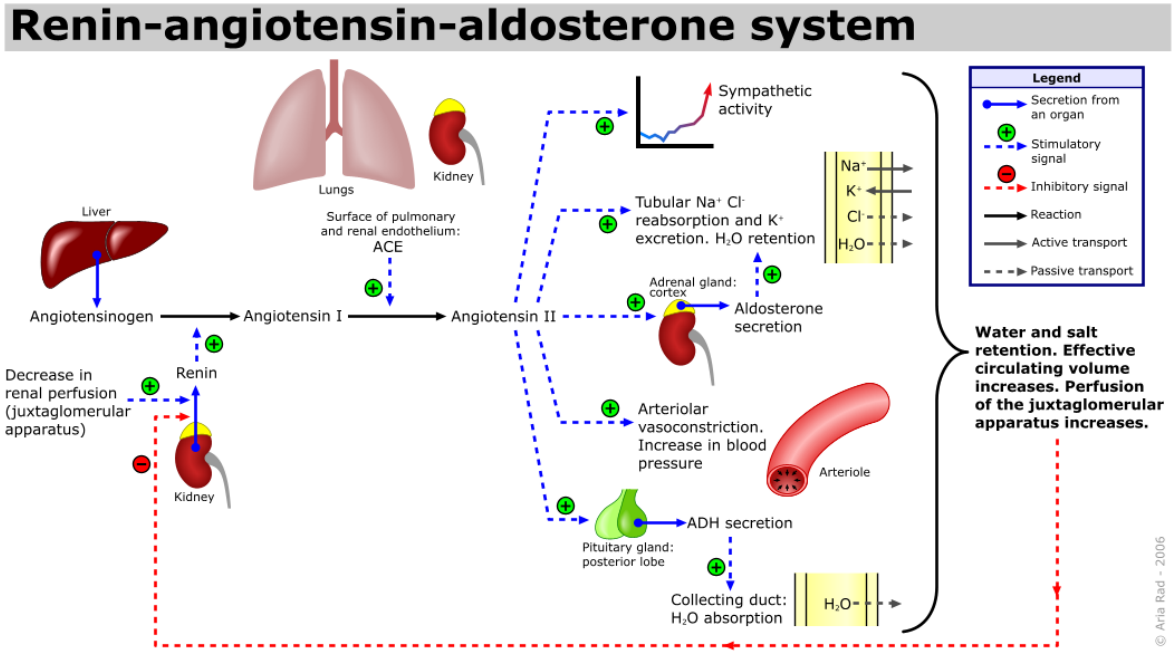
Why the change between depression and elevation?



Initial Treatment for MI

- Nitroglycerin - Why
- ASA/Heparin – Why
- Oxygen – Why
- B-Blockers – Why
- Morphine - Why
- ACE Inhibitors – Why

ACE Inhibitors



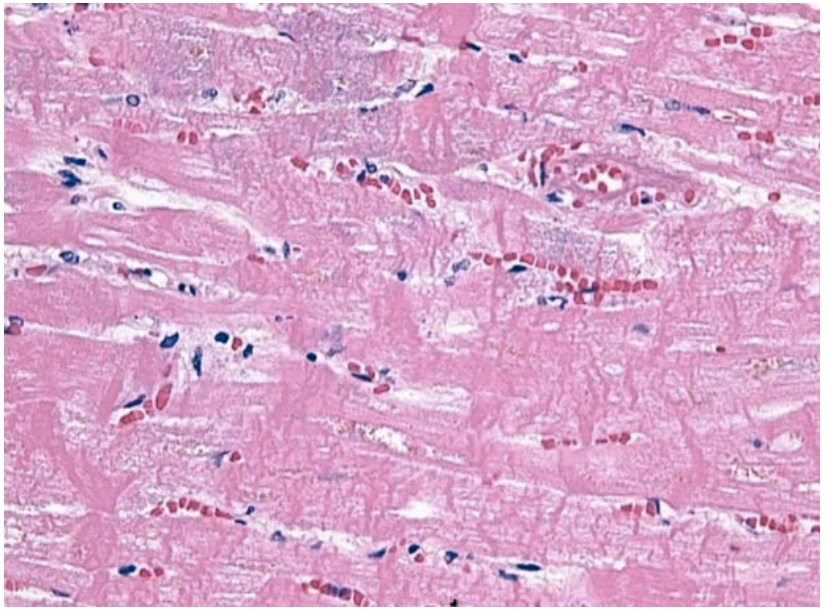
Definitive Treatment

- Fibrinolysis
- Angioplasty

- - What complications can come from these two treatments?

Contraction band necrosis

- When perfusion is restored calcium will reenter the affected cells.
- As calcium is the initiator of contraction – this will cause a unique phenomenon known as contraction band necrosis



Reperfusion Injury

- Upon restoration of the blocked blood vessels cardiac enzymes continue to rise – Why?

- We restored the blood flow back to the heart– which now contains large quantities of oxygen to damaged tissues. This causes free radical damage to the myocytes and cause often cause an additional rise in cardiac enzymes

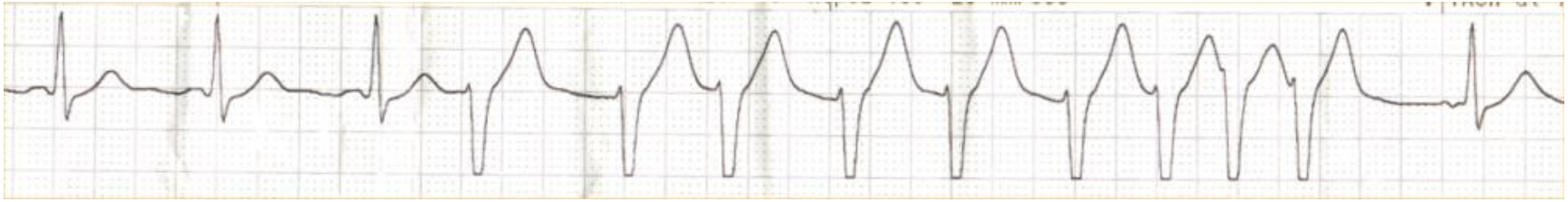
TIME FROM INFARCTION	GROSS CHANGES	MICROSCOPIC CHANGES	COMPLICATIONS
< 4 hours	None	None	Cardiogenic shock (massive infarction), congestive heart failure, and arrhythmia
4–24 hours	Dark discoloration	Coagulative necrosis (Fig. 8.4A)	Arrhythmia
1–3 days	Yellow pallor	Neutrophils (Fig. 8.4B)	Fibrinous pericarditis (Fig. 8.5A); presents as chest pain with friction rub
4–7 days	Yellow pallor	Macrophages	Rupture of ventricular free wall (Fig. 8.5B; leads to cardiac tamponade), interventricular septum (leads to shunt), or papillary muscle (Fig. 8.5C; leads to mitral insufficiency)
1–3 weeks	Red border emerges as granulation tissue enters from edge of infarct.	Granulation tissue with plump fibroblasts, collagen, and blood vessels	
Months	White scar (Fig. 8.6A)	Fibrosis (Fig. 8.6B)	Aneurysm (Fig. 8.7), mural thrombus, or Dressler syndrome

Post MI Complications

- A 47 year old male patient activates the EMS system with complaint of rapid fluttering in his chest. The onset was 24 hours prior when he was experiencing significant chest pain

- The patient did not seek medical treatment at the time

Explain the following



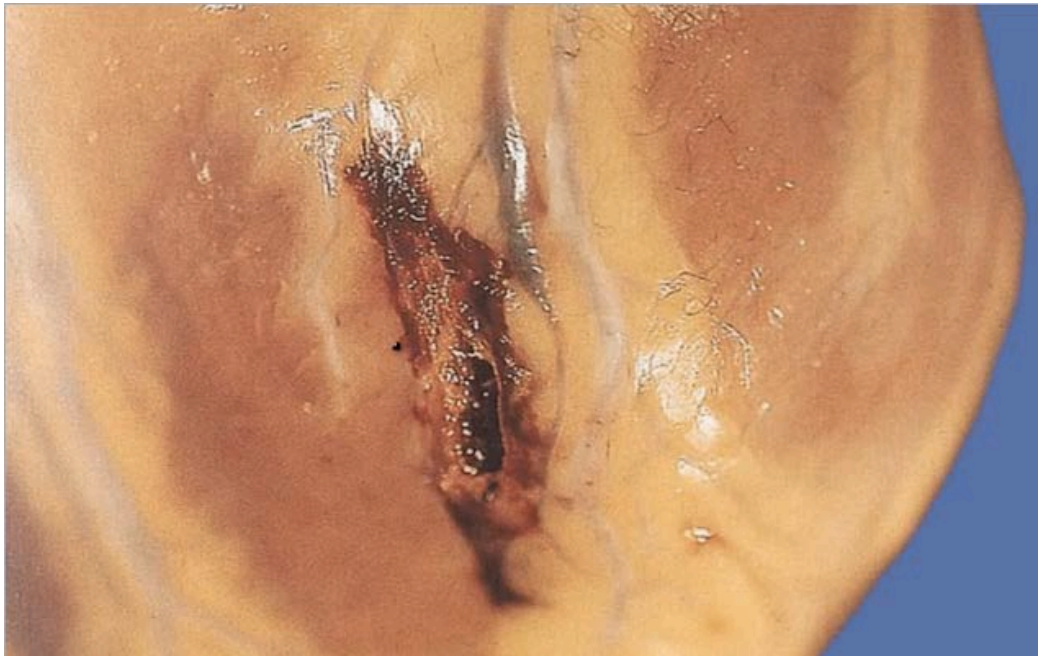
Post MI Complication

- A 52 year old male patient activated the EMS system. He states he was admitted to the hospital 7 days prior for a myocardial infarction involving the LAD. He was unsure of the treatment but knows he underwent some type of surgery.

- He now presents with hypotension, JVD, and muffled heart sounds

- Narrowed pulse pressure is observed

Rupture of the Ventricular Wall

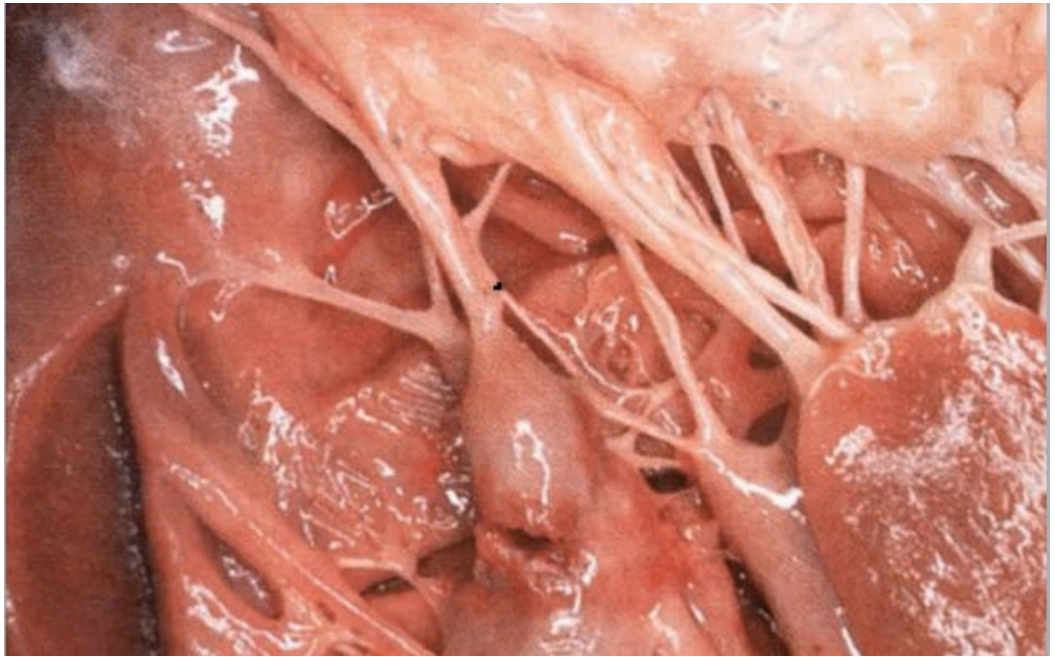


Post MI Complications

- A 62 year old male activates EMS with the chief complain of tachypnea, palpitations, dyspnea and mild chest pain. He states he was discharged from the hospital post MI 7 days prior. He stated that they had to stent his right coronary artery

- Physical exam reveals a distinctive heart murmur, pulmonary congestion, JVD, and pedal edema.

Mitral valve insufficiency - Rupture of Papillary Muscle

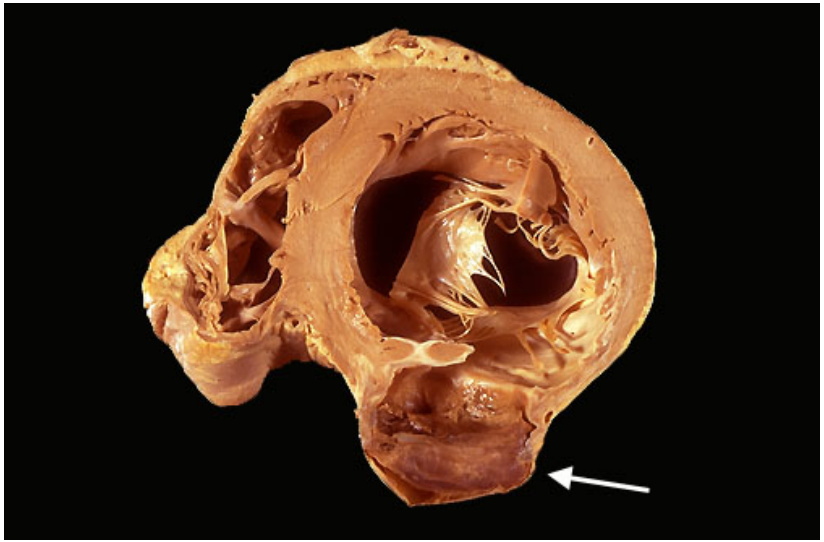


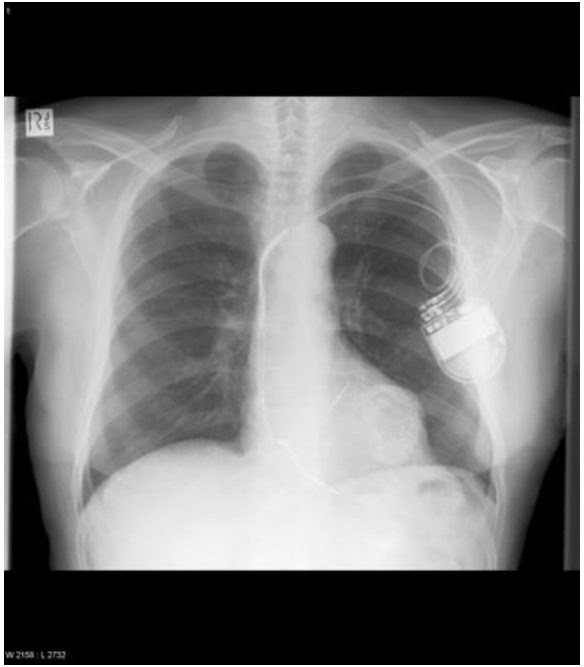
Post MI Complications

- A 60 year old female presents with the signs of symptoms consistent with a stroke. Left Hemiplegia is noted as well as slurred speech. The patient's family member state she had a myocardial infarction two months prior but had since been well.

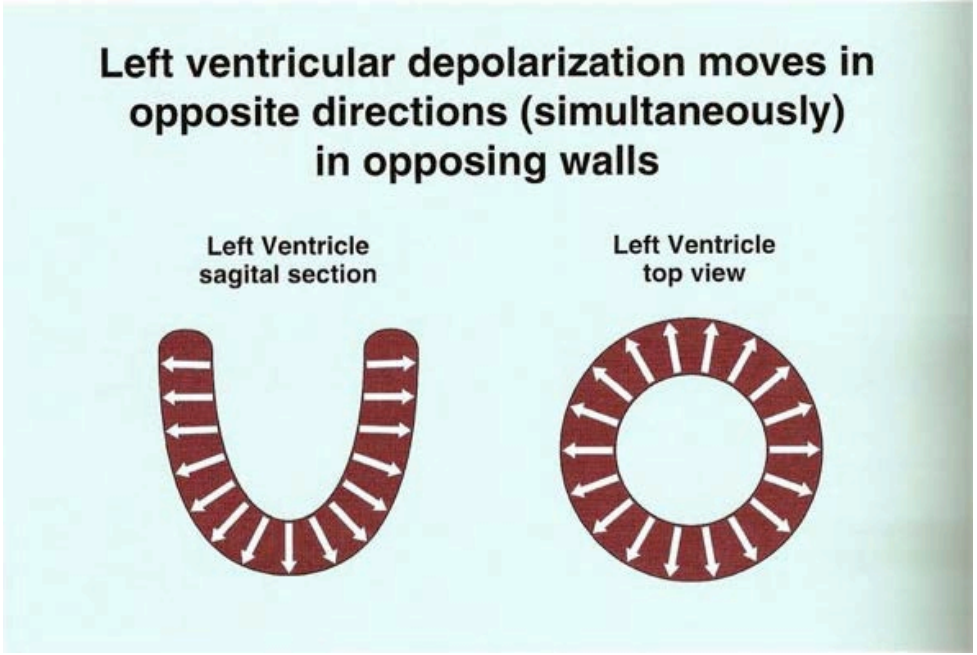
- The patient has a normal sinus rhythm. What cause her stroke?

Left Ventricular Aneurysm

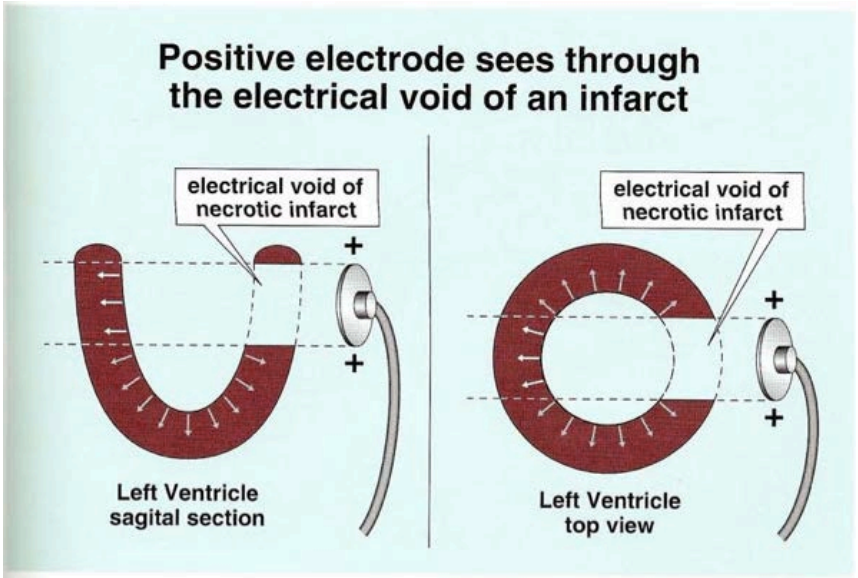




Long term EKG Changes

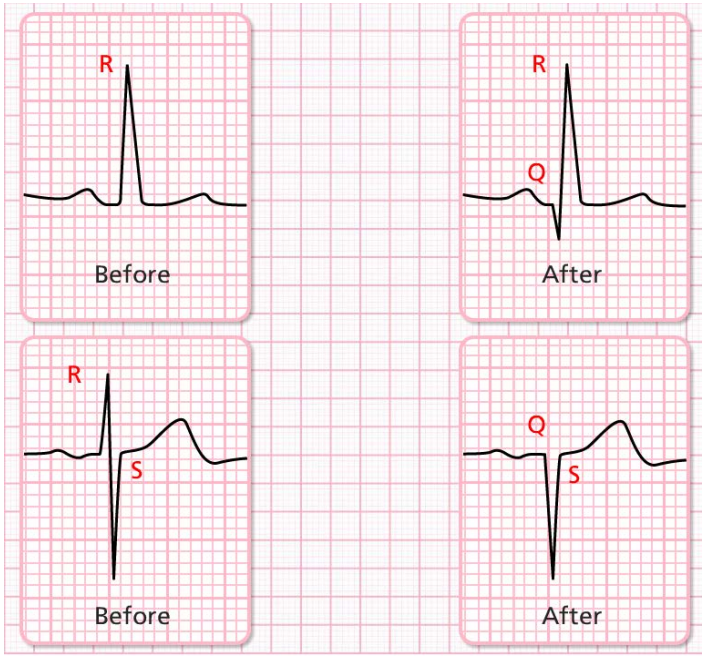


Q-Wave

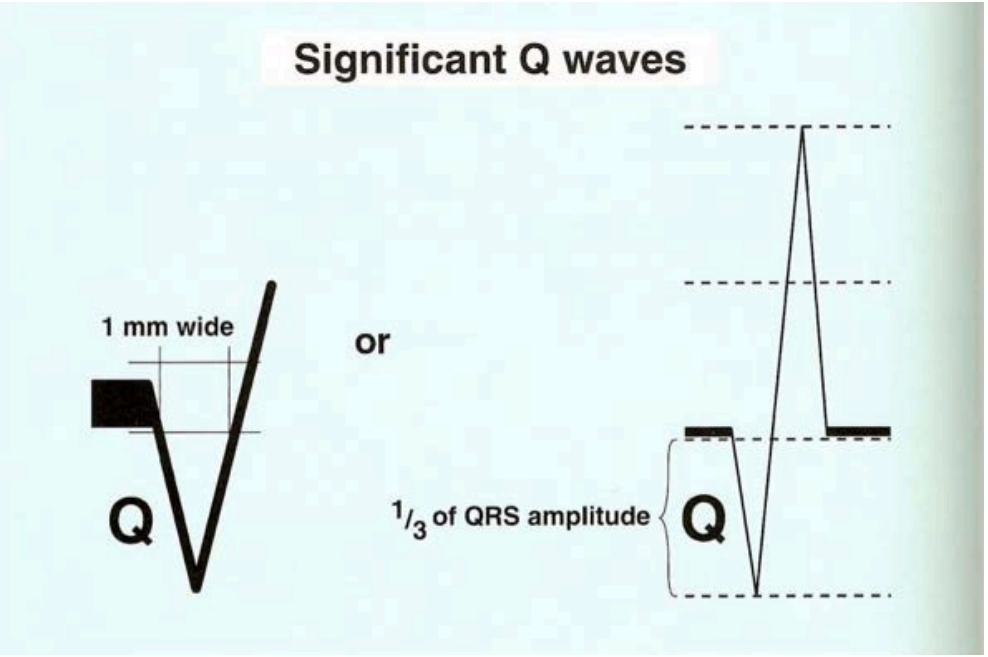


An infarct is necrotic; it cannot depolarize and has no vectors. So, the **positive** electrode nearest the infarct detects no "toward" vectors, it sees only the "away" vectors from the opposite wall (through the necrotic void). Therefore, a Q wave is inscribed on EKG in the leads which use that positive electrode for recording.

Pathologic Q-waves



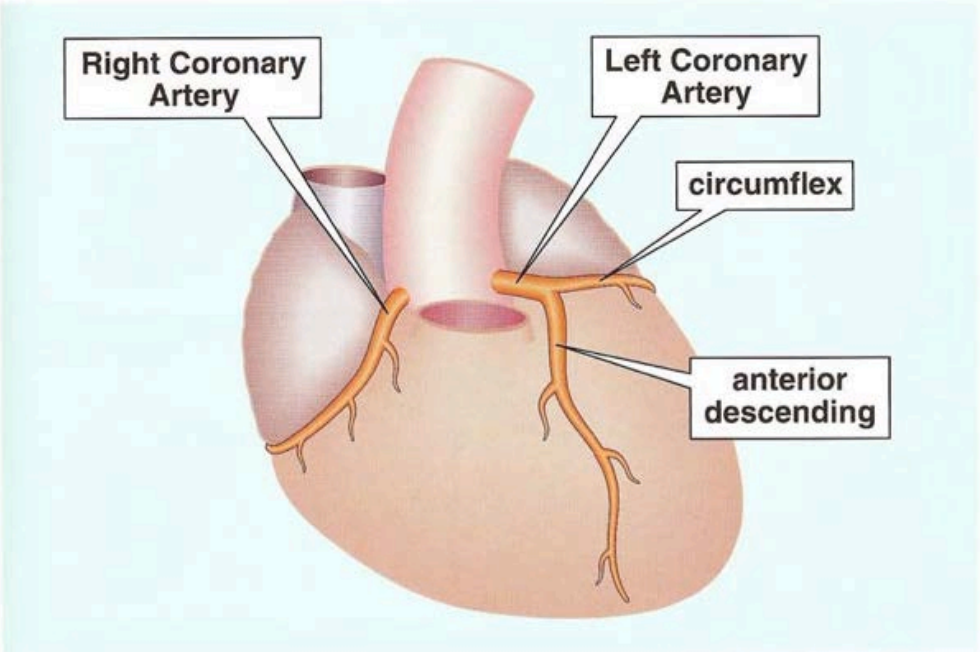
Quantifying the Q-Wave



In the absence of a prior MI

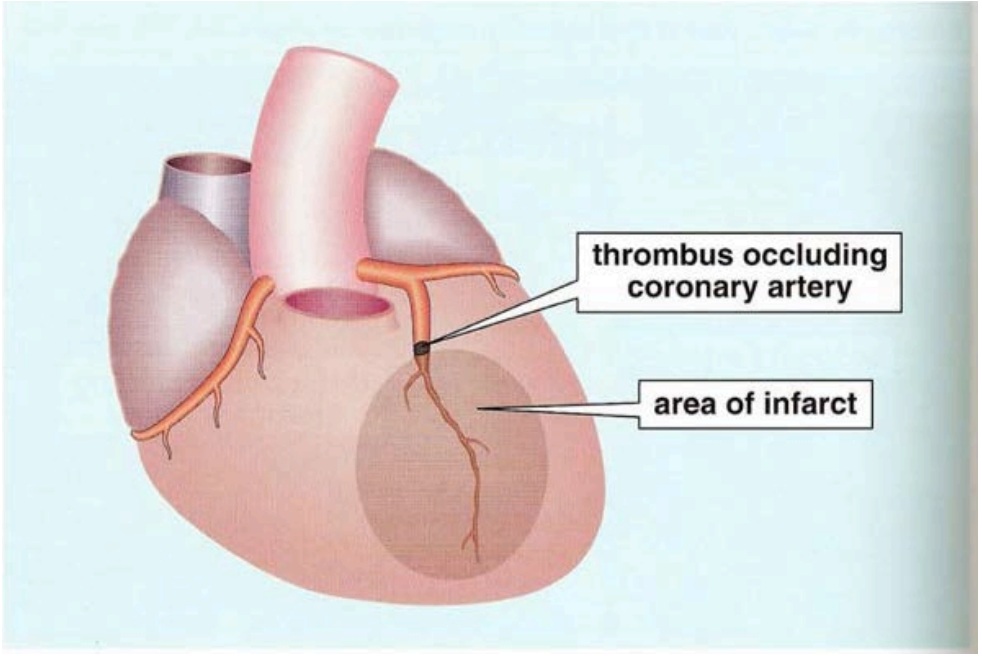
- Q-waves could mean
  - Cardiomyopathy
  - Amyloid
  - Altered Conduction (LBBB and WPW)
  - Ventricular enlargement

Coronary Vasculature

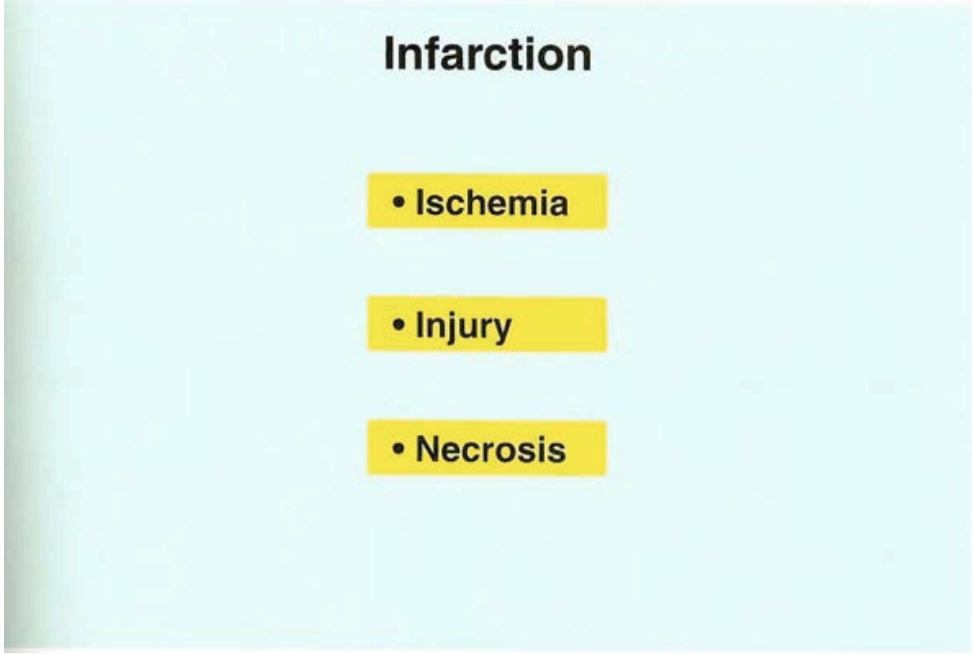


Remember, while the heart is filled with blood, it derives its blood supply solely from the coronary arteries as noted in the picture

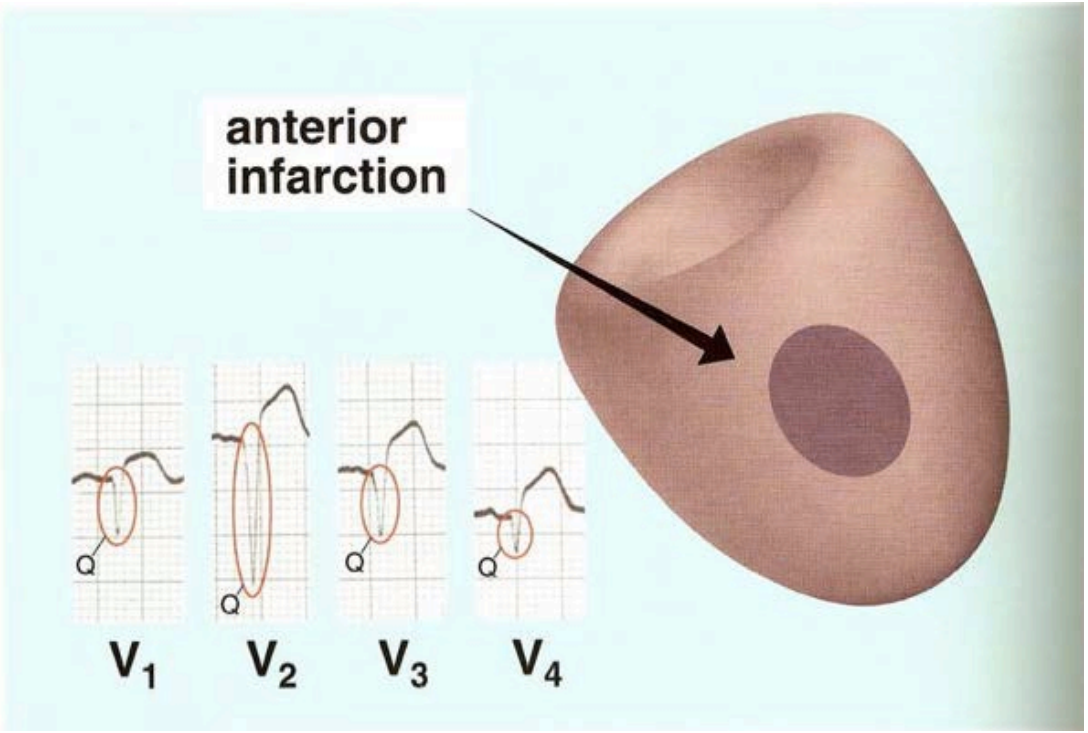
Understanding the Area of infarct



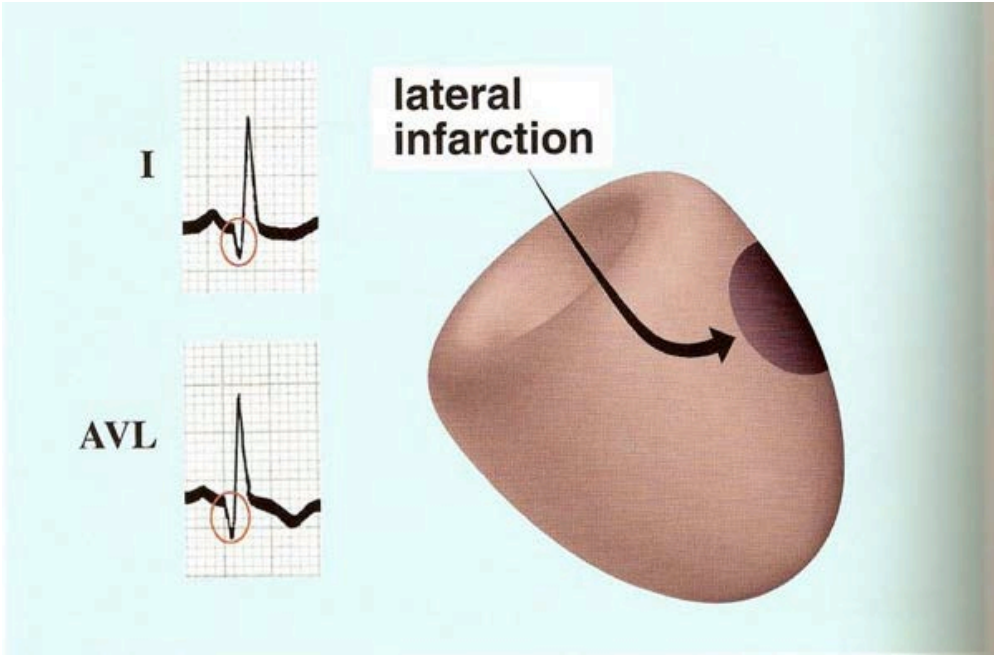
Myocardial Infarction Triad



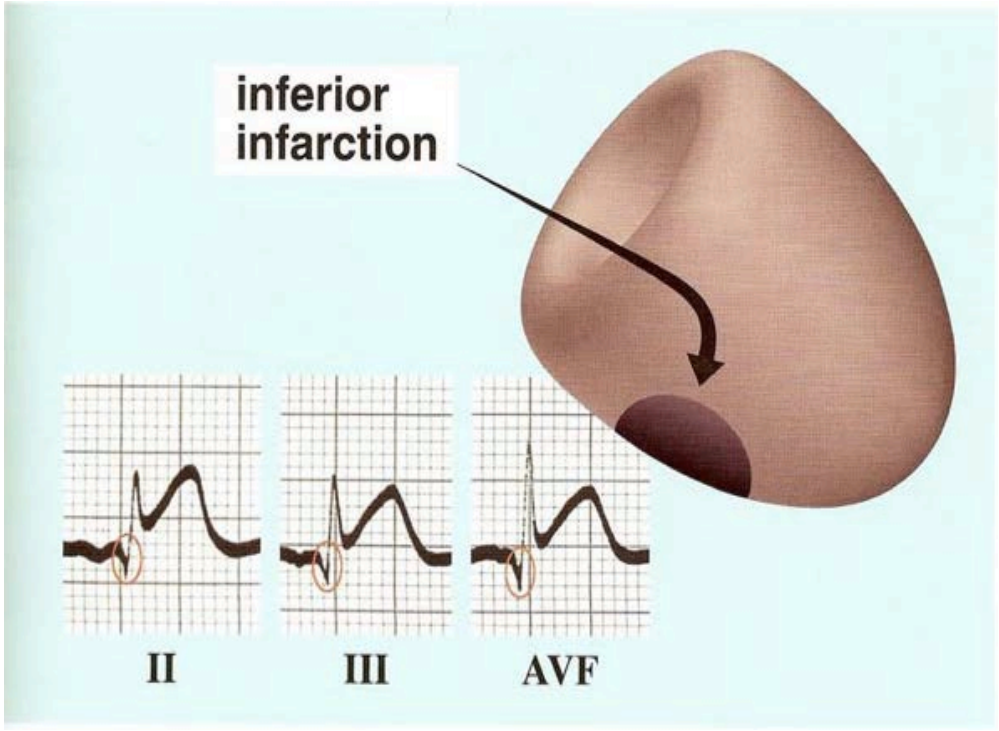
Anterior Infarction



Lateral Infarction



Inferior Infarction



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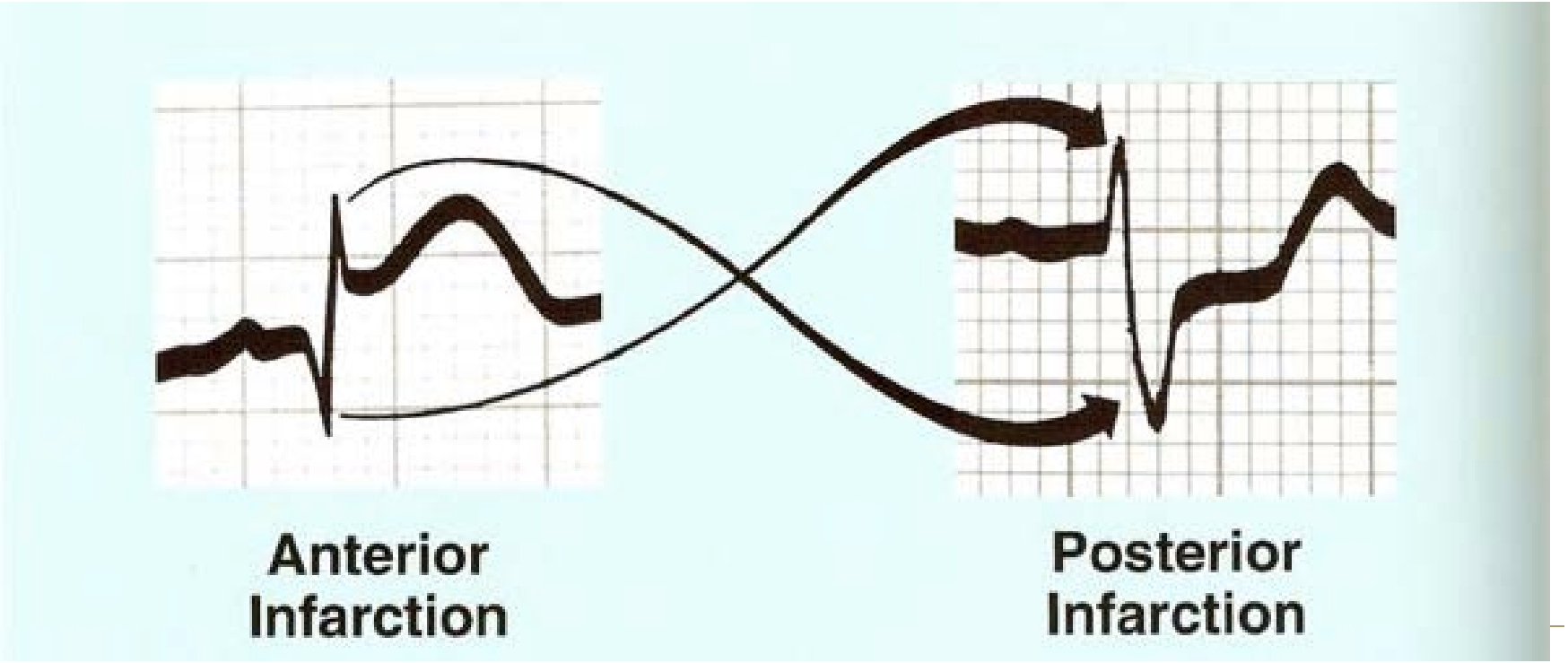
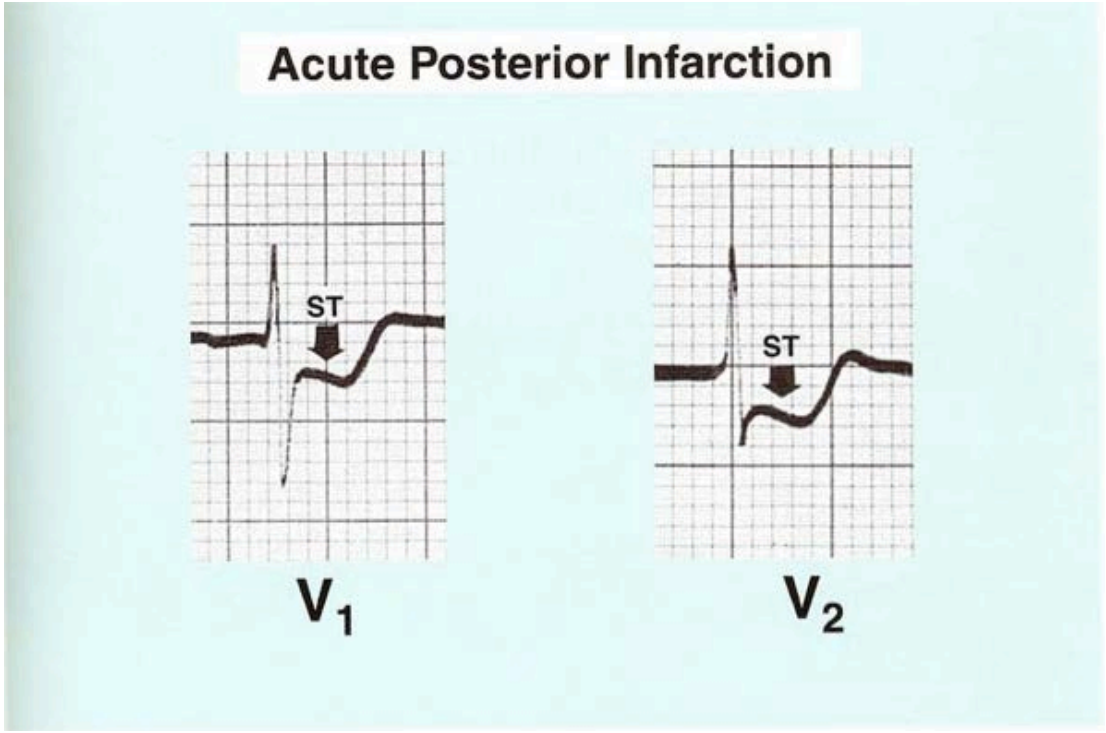
So if an anterior infarction produces Q-waves and ST Elevation – what will a Posterior infarction cause?



Acute Posterior Infarction



Ways to determine this

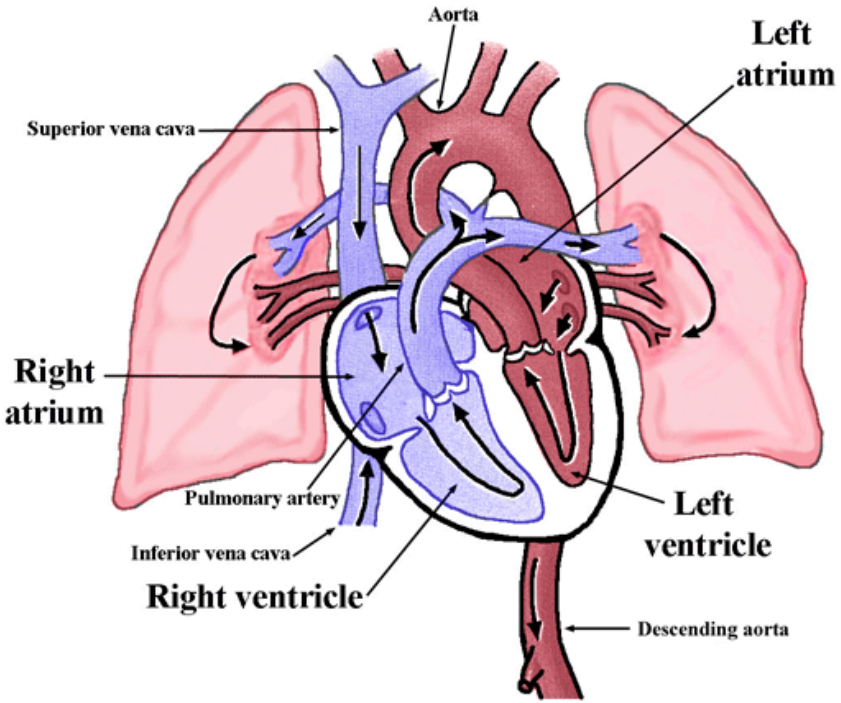
- Reversed Trans illumination
- Mirror Test
- Right sided or posterior lead ECG
  - This topic could be an entire lecture on its own so I highly suggest doing some research into EKGs for posterior myocardial infarctions

Caution with Inferior Wall MI and Nitroglycerin

- When the Right Ventricle is involved in an inferior wall MI – up to 60% of patients will develop hypotension.

- Profound hypotension can be precipitated by the administration of nitroglycerin.

Why is this?



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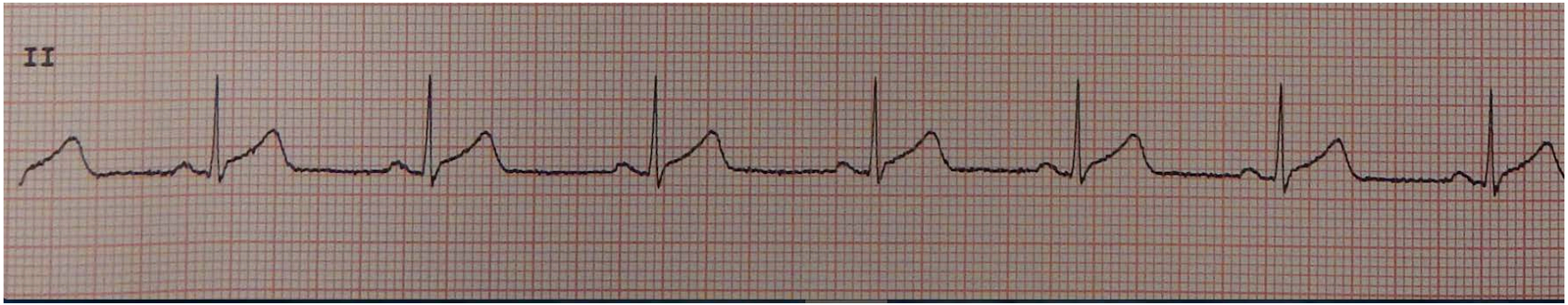
Practice

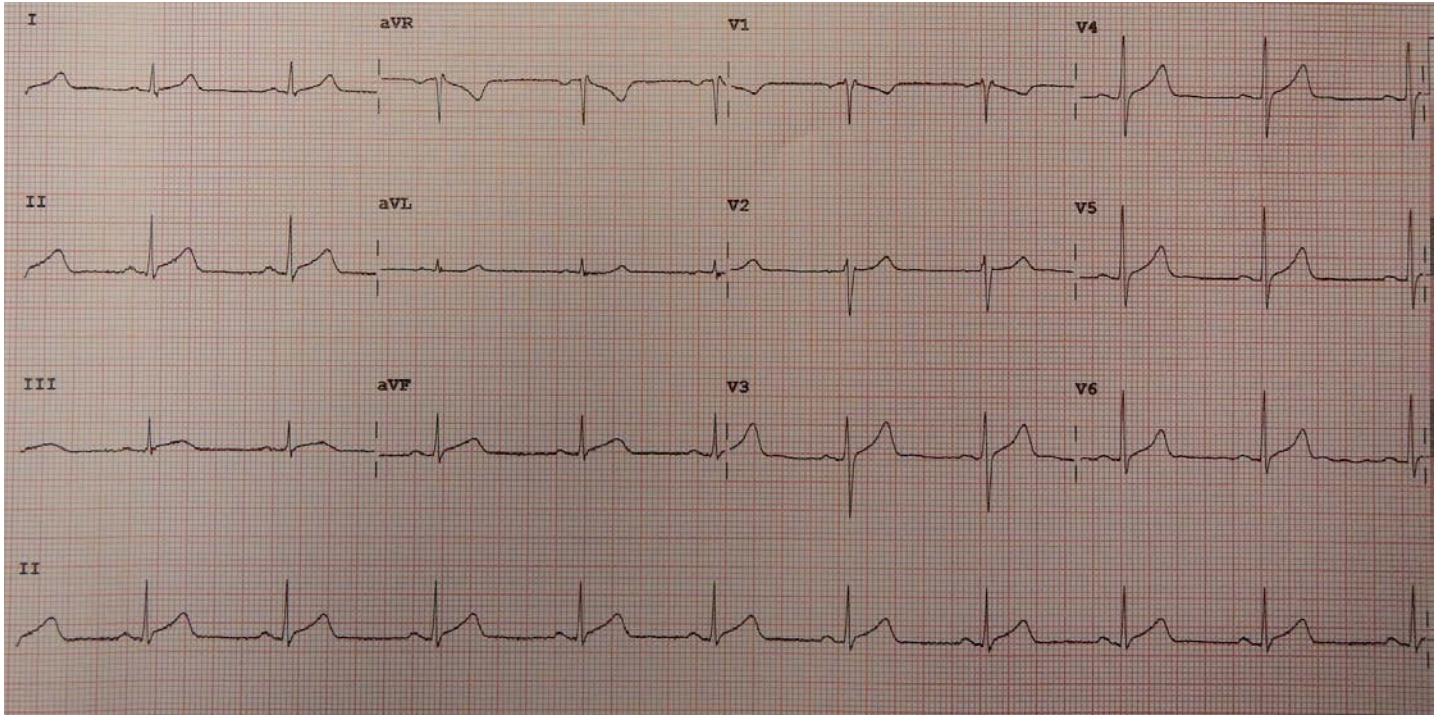
- A 42 year old man activates complaining of chest pain – is he experiencing a myocardial infarction?



Trick Question

- This was a bit of an unfair question
- Lead II is a great way to look at a cardiac rhythm but has limitations in what it can tell us.
- Obtaining a 12 lead ECG is imperative to evaluating the prehospital cardiac patient

Pericarditis



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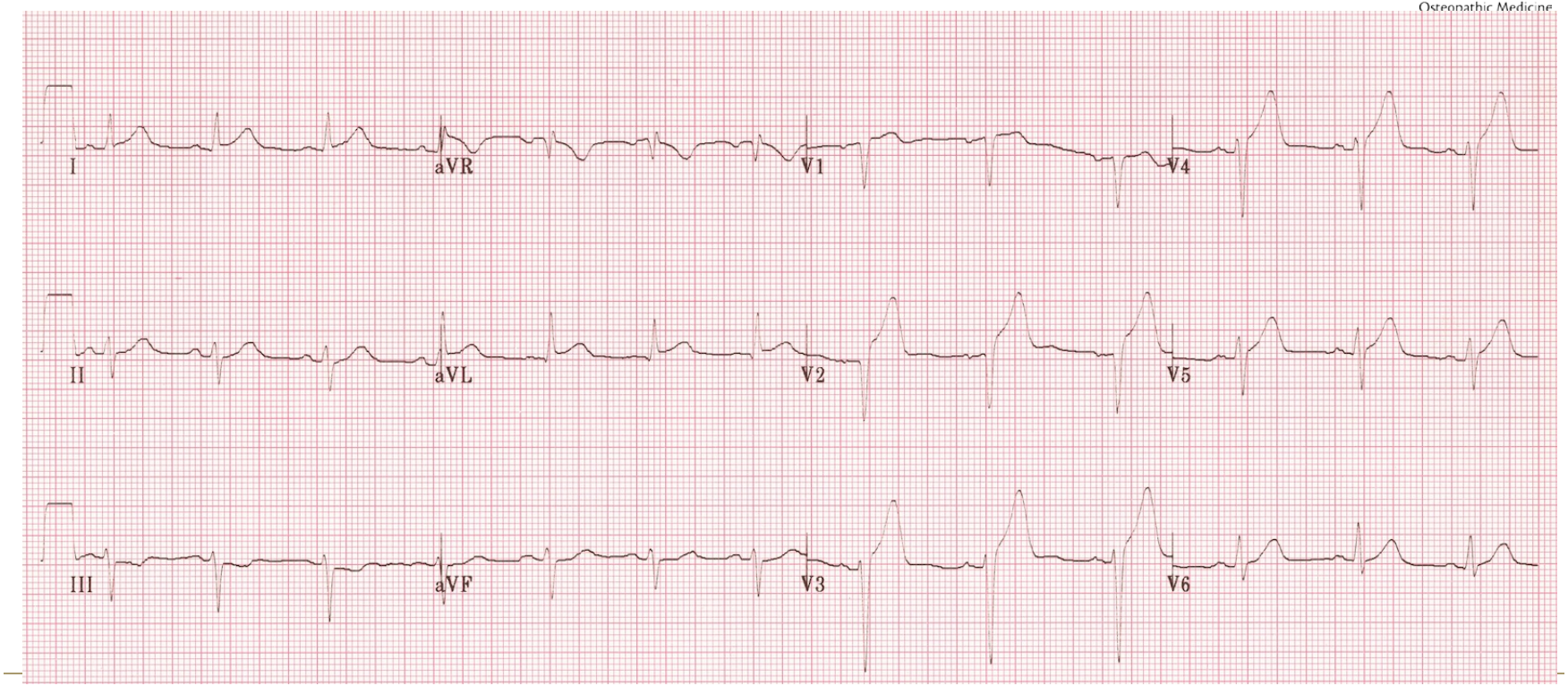
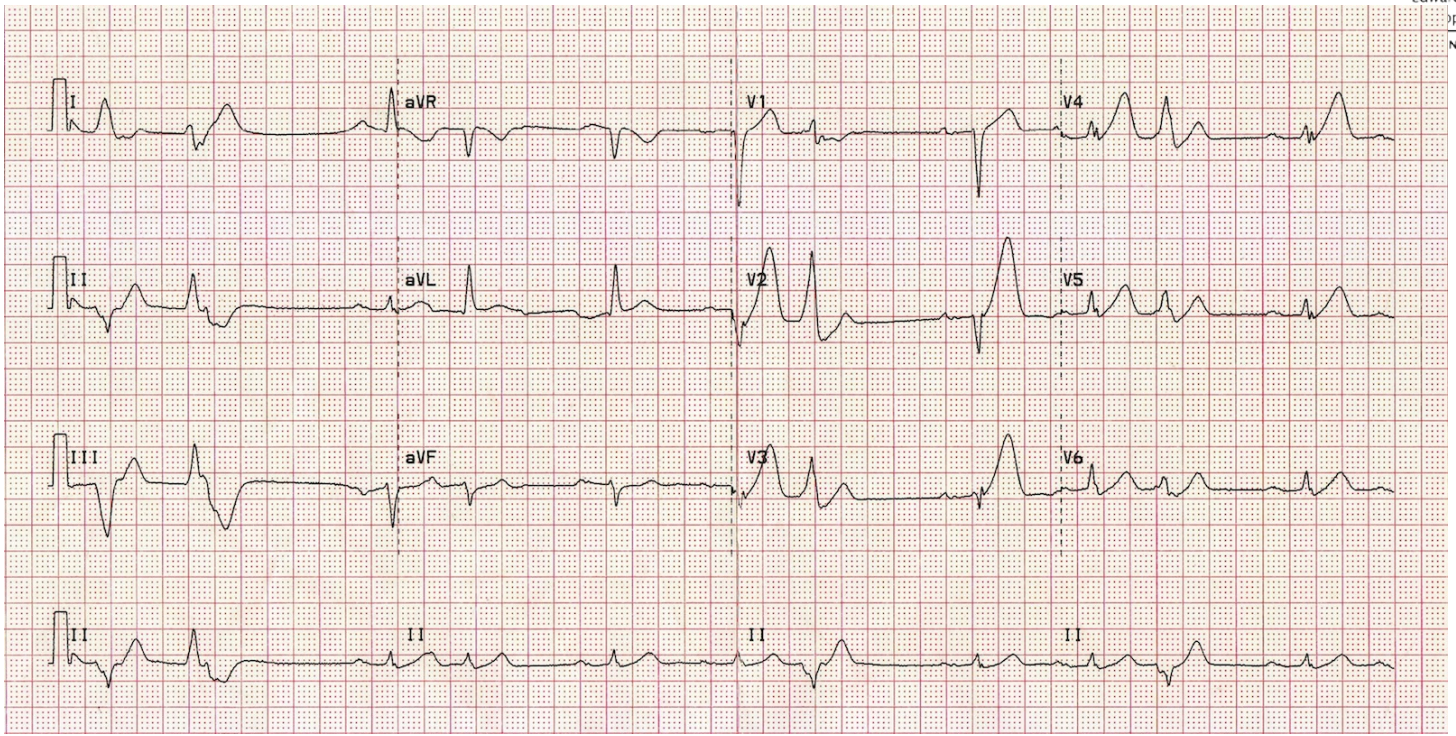
Patient EKG 1



Evaluation 1

- ST elevation is maximal in the anteroseptal leads (V1-4).
- Q waves are present in the septal leads (V1-2).
- There is also some subtle STE in I, aVL and V5, with reciprocal ST depression in lead III.
- There are hyperacute (peaked) T waves in V2-4.
- These features indicate a **hyperacute anteroseptal STEMI**

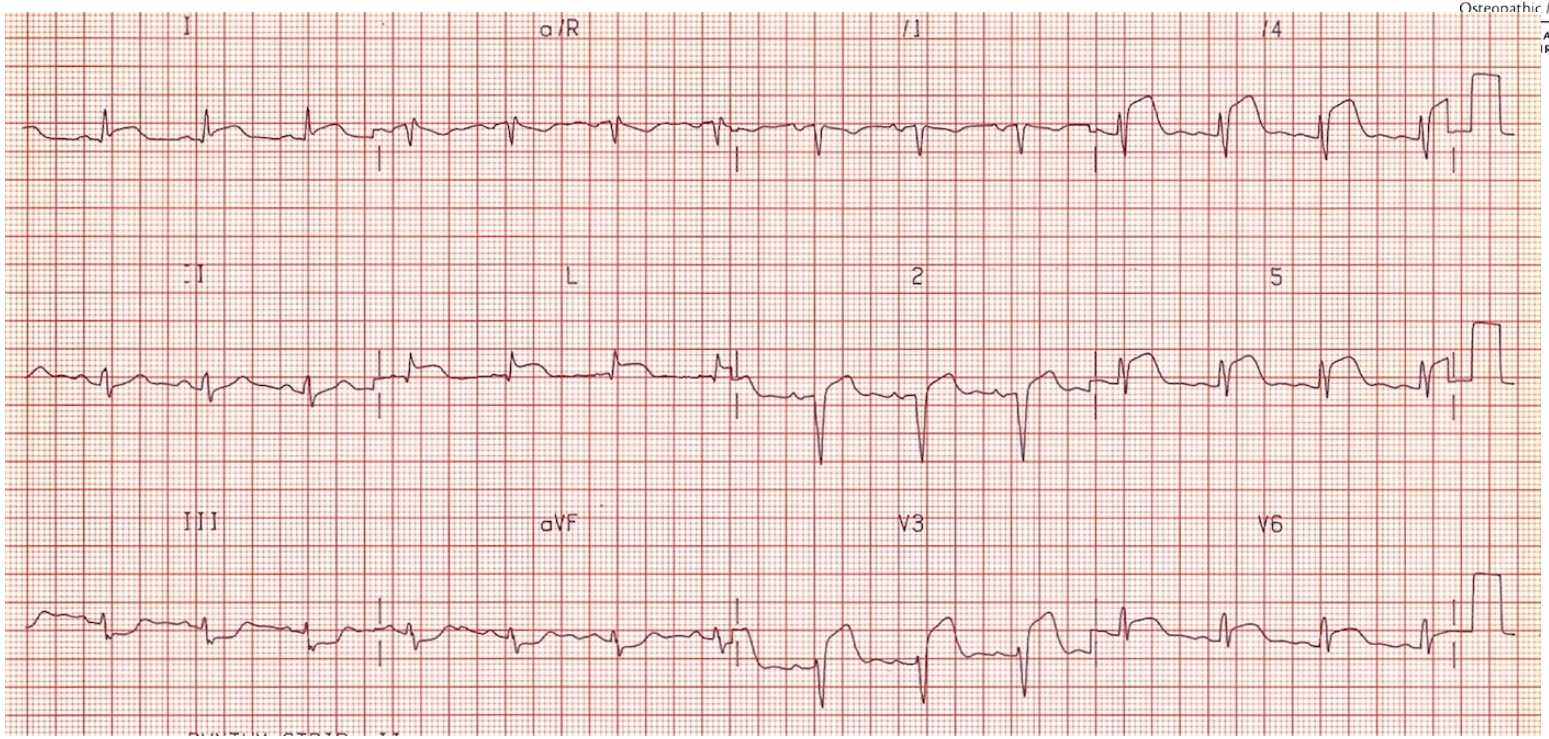
Patient EKG 2



Evaluation 2

- Hyperacute Anterior STEMI
- There are hyperacute T-waves in V2-6 (most marked in V2 and V3) with loss of R wave height.
- The rhythm is sinus with 1st degree AV block.
- There are premature atrial complexes (beat 4 on the rhythm strip) and multifocal ventricular ectopy (PVCs of two different types), indicating an “irritable” myocardium at risk of ventricular fibrillation.

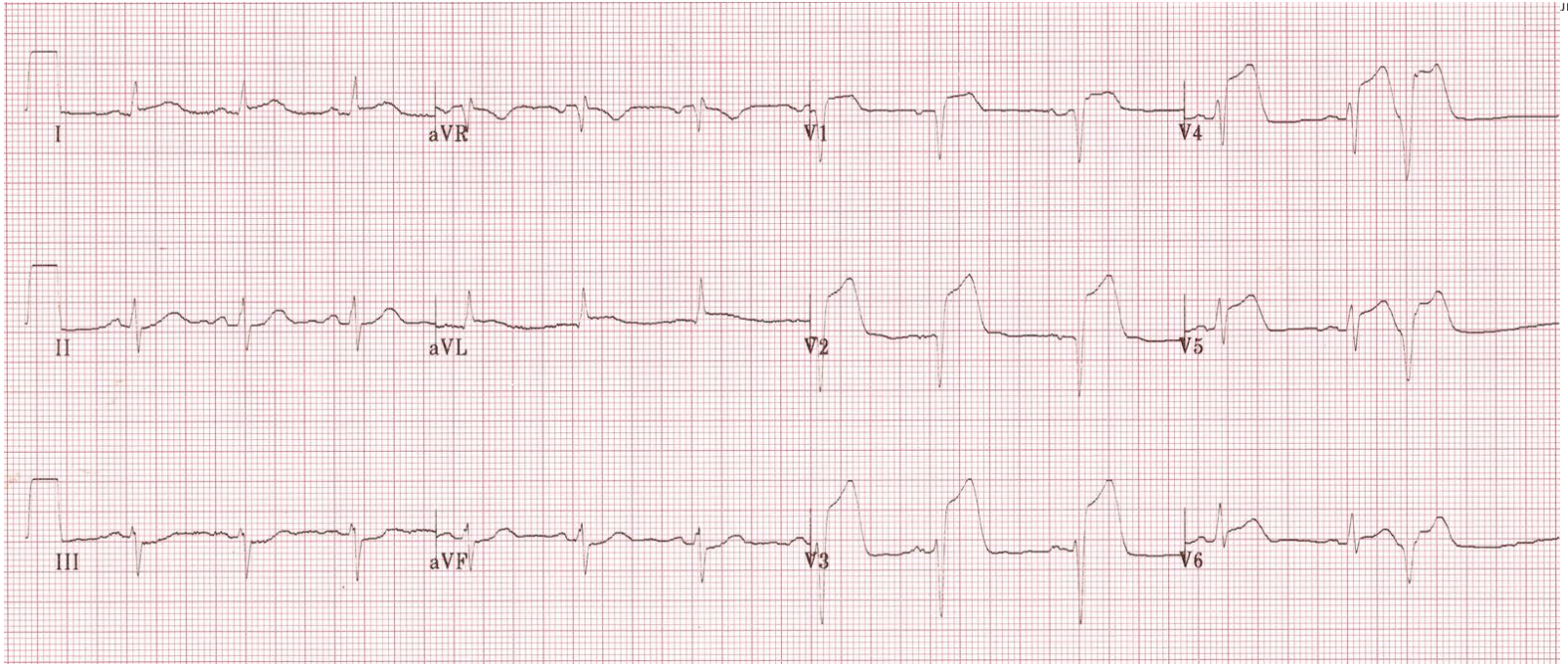
Patient EKG 3



Evaluation 3

- AnterioLateral STEMI (Acute)
- ST elevation in V2-6, I and aVL.
- Reciprocal ST depression in III and AVF.

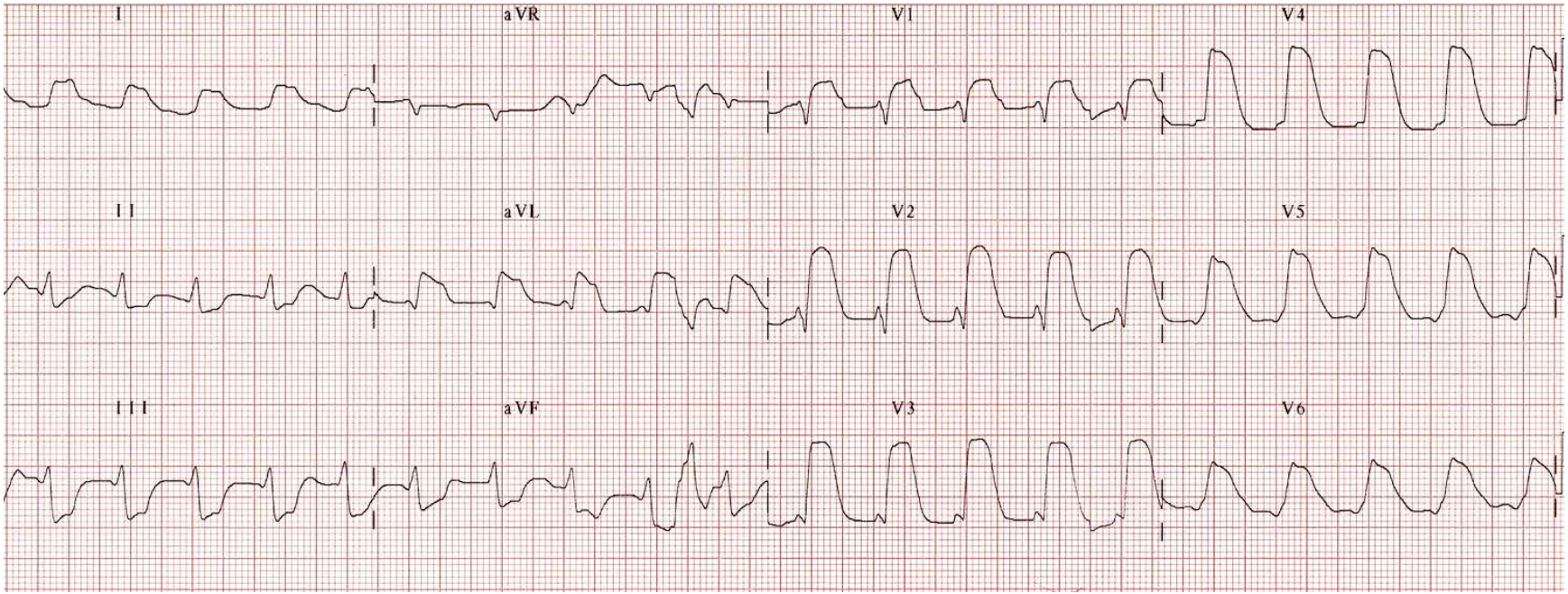
Patient EKG 4



Evaluation 4

- Extensive Anterior STEMI (acute)
- ST elevation in V1-6 plus I and aVL (most marked in V2-4).
- Minimal reciprocal ST depression in III and aVF.
- Q waves in V1-2, reduced R wave height (a Q-wave equivalent) in V3-4.
- There is a premature ventricular complex (PVC) with “R on T’ phenomenon at the end of the ECG; this puts the patient at risk for malignant ventricular arrhythmias.

Patient EKG 5



Evaluation 5

- Extensive Anterior MI
- Massive ST elevation with “tombstone” morphology is present throughout the precordial (V1-6) and high lateral leads (I, aVL).
- This pattern is seen in proximal LAD occlusion and indicates a large territory infarction with a poor LV ejection fraction and high likelihood of cardiogenic shock and death.

Patient EKG 6

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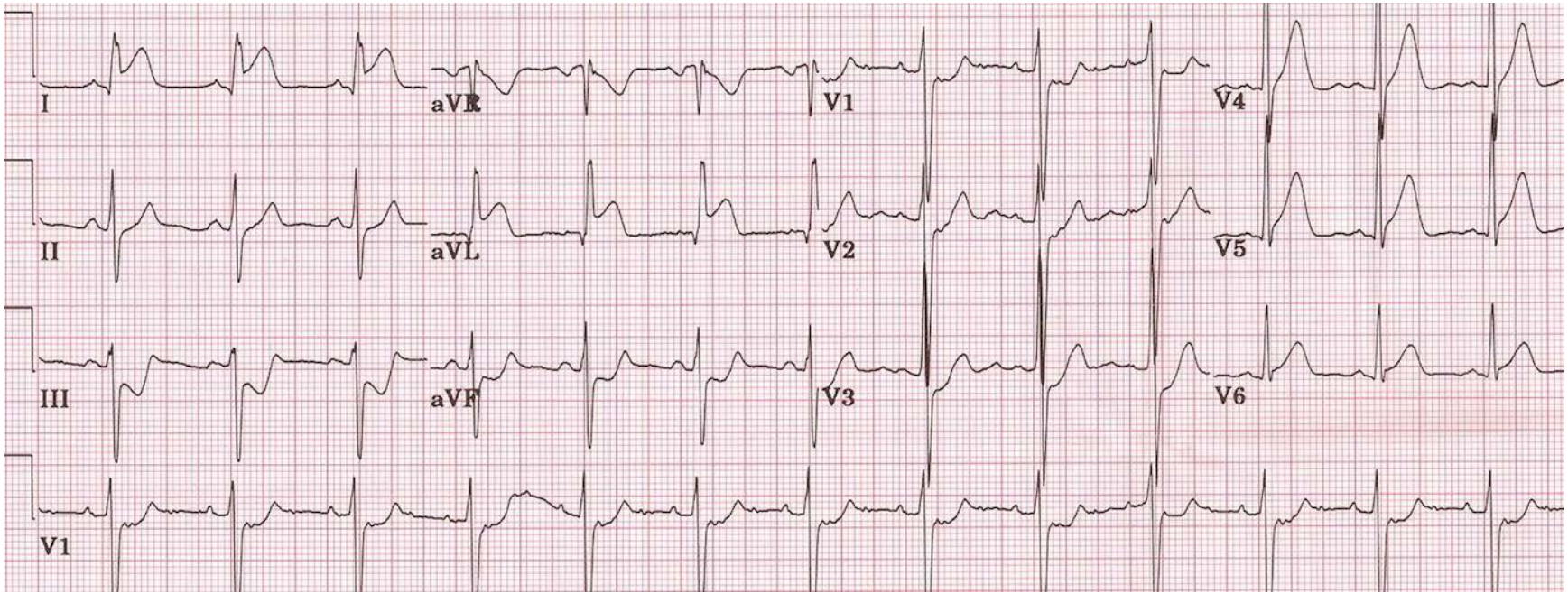
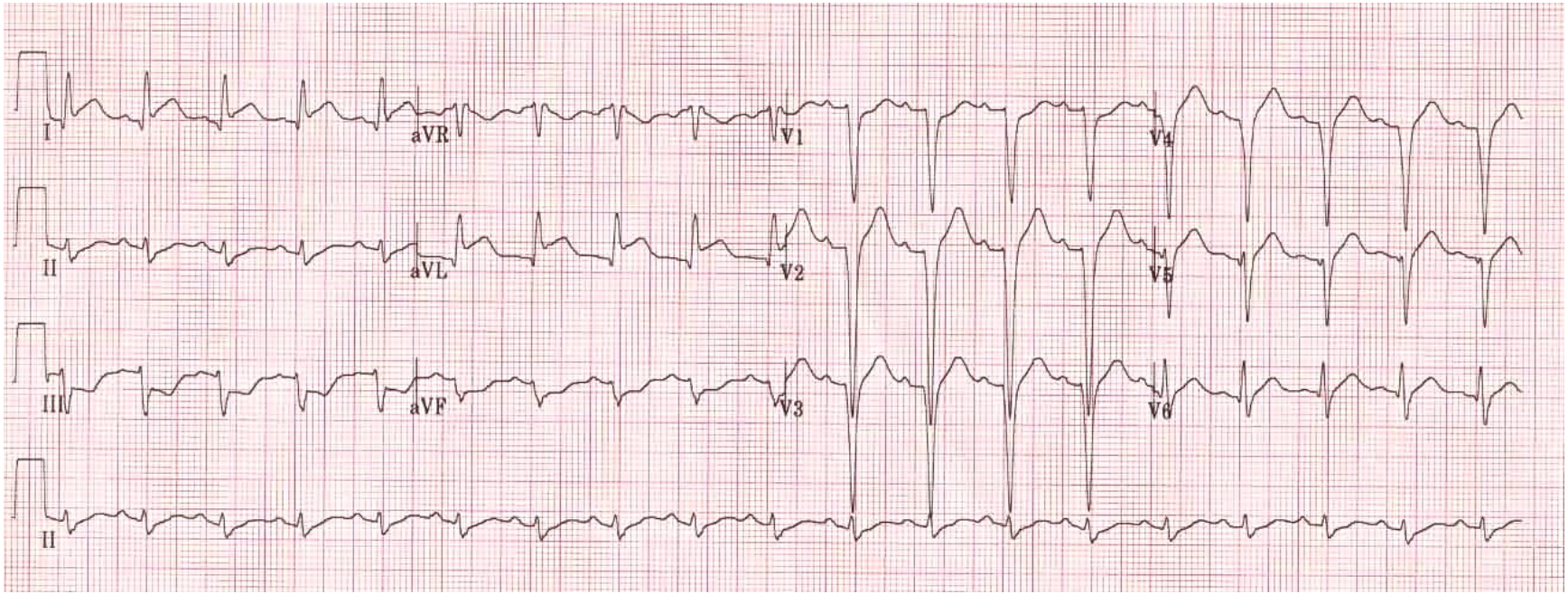


Evaluation 6

- High Lateral STEMI
- ST elevation is present in the high lateral leads (I and aVL).
- There is also subtle ST elevation with hyperacute T waves in V5-6.
- There is reciprocal ST depression in the inferior leads (III and aVF) with associated ST depression in V1-3 (which could represent anterior ischaemia or reciprocal change).
- This pattern is consistent with an acute infarction localised to the superior portion of the lateral wall of the left ventricle (high lateral STEMI).
- The culprit vessel in this case was an occluded first diagonal branch of the LAD.

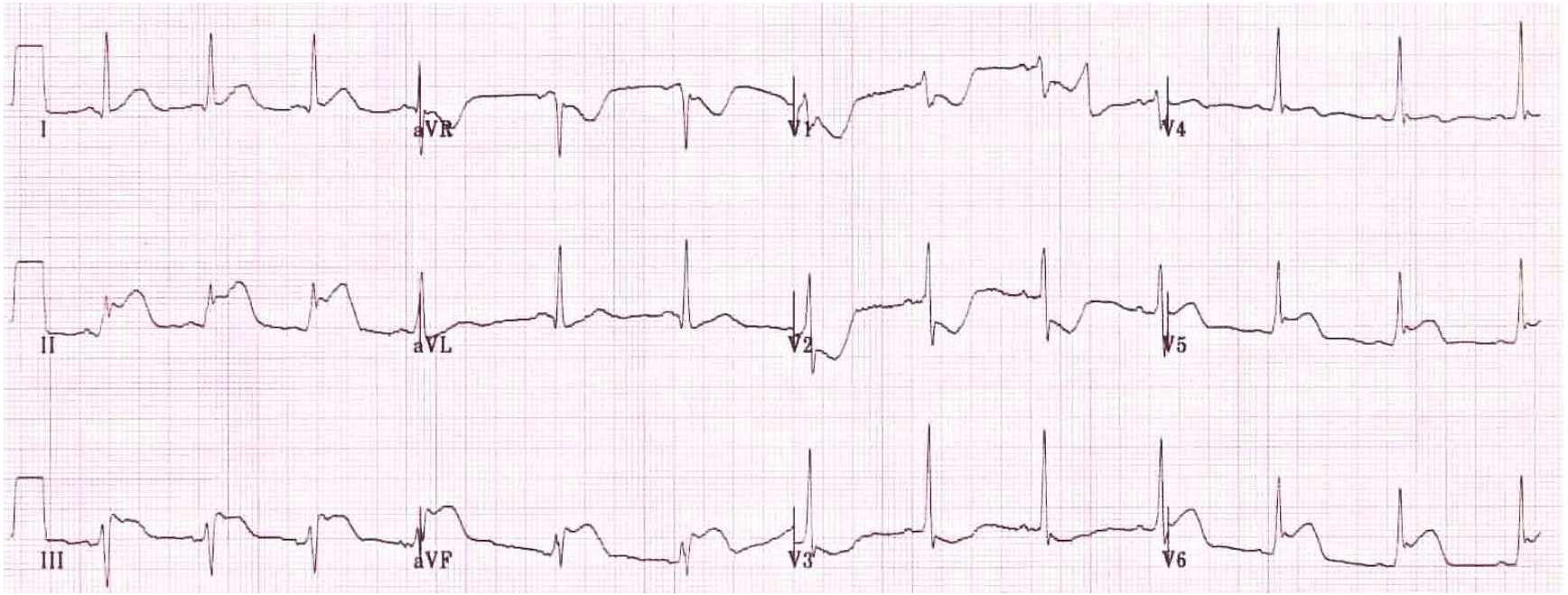
Patient EKG 7



Evaluation 7

- High Lateral STEMI:
- ST elevation is present in the high lateral leads (I and aVL).
- There is reciprocal ST depression in the inferior leads (III and aVF).
- QS waves in the anteroseptal leads (V1-4) with poor R wave progression indicate prior anteroseptal infarction.
- This pattern suggests proximal LAD disease with an acute occlusion of the first diagonal branch (D1).

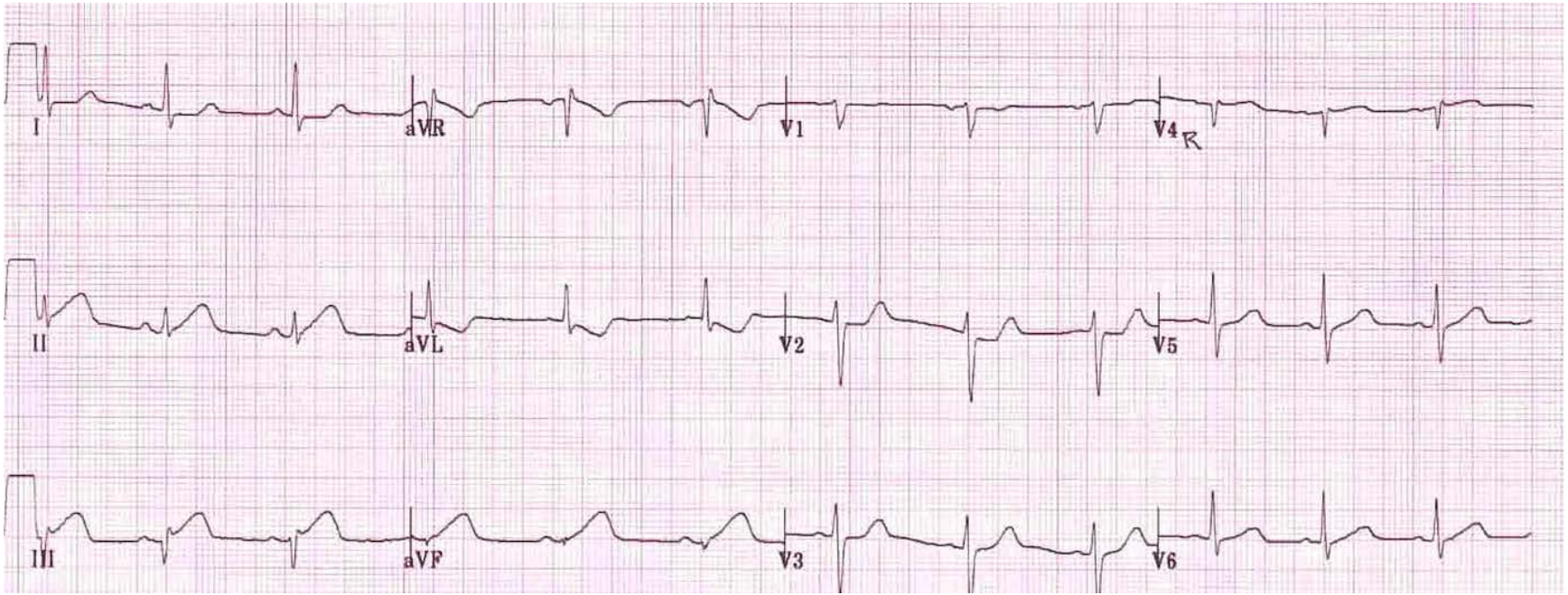
Patient EKG 8



Evaluation 8

- There is ST elevation in the inferior (II, III, aVF) and lateral (I, V5-6) leads.
- The precordial ST elevation extends out as far as V4, however the maximal STE is in V6.
- ST depression in V1-3 is suggestive of associated posterior infarction (the R/S ratio > 1 in V2 is consistent with this).
- This is an acute inferolateral STEMI with probable posterior extension.
- This constellation of ECG abnormalities is typically produced by occlusion of the proximal circumflex artery.

Patient EKG 9



Evaluation 10

- Early Inferior STEMI
- Hyperacute (peaked) T waves in II, III and aVF with relative loss of R wave height.
- Early ST elevation and Q-wave formation in lead III.
- Reciprocal ST depression and T wave inversion in aVL.
- ST elevation in lead III > lead II suggests an RCA occlusion; the subtle ST elevation in V4R would be consistent with this.

Patient EKG 10

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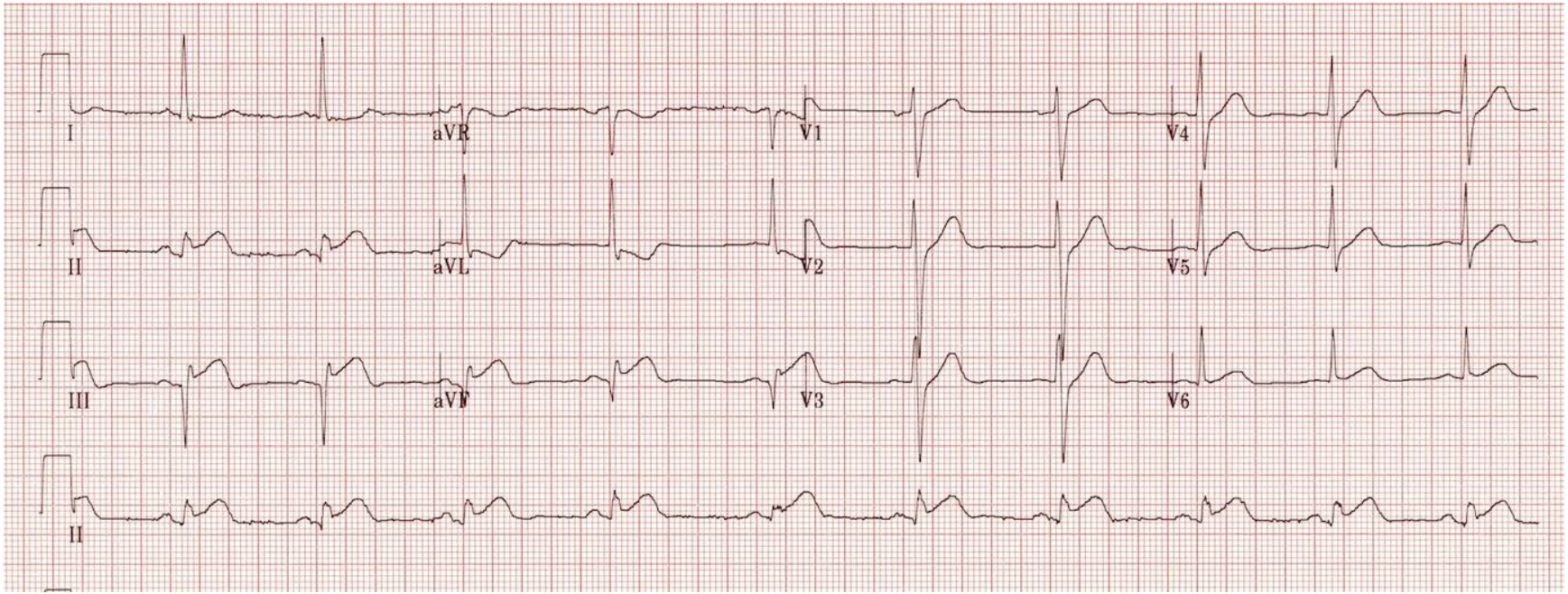

Evaluation 10

- Inferior STEMI
- ST elevation in II, III and aVF.
- Q-wave formation in III and aVF.
- Reciprocal ST depression and T wave inversion in aVL
- ST elevation in lead II = lead III and absent reciprocal change in lead I (isoelectric ST segment) suggest a circumflex artery occlusion

Patient EKG 11

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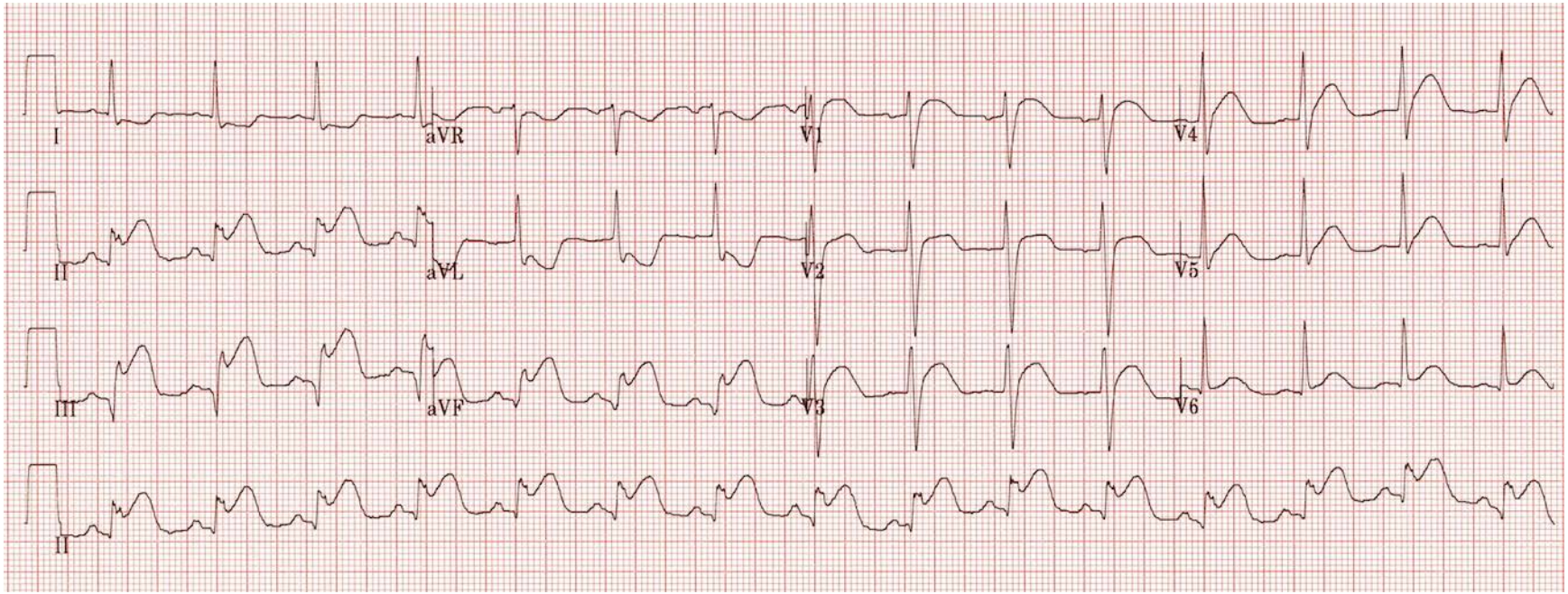
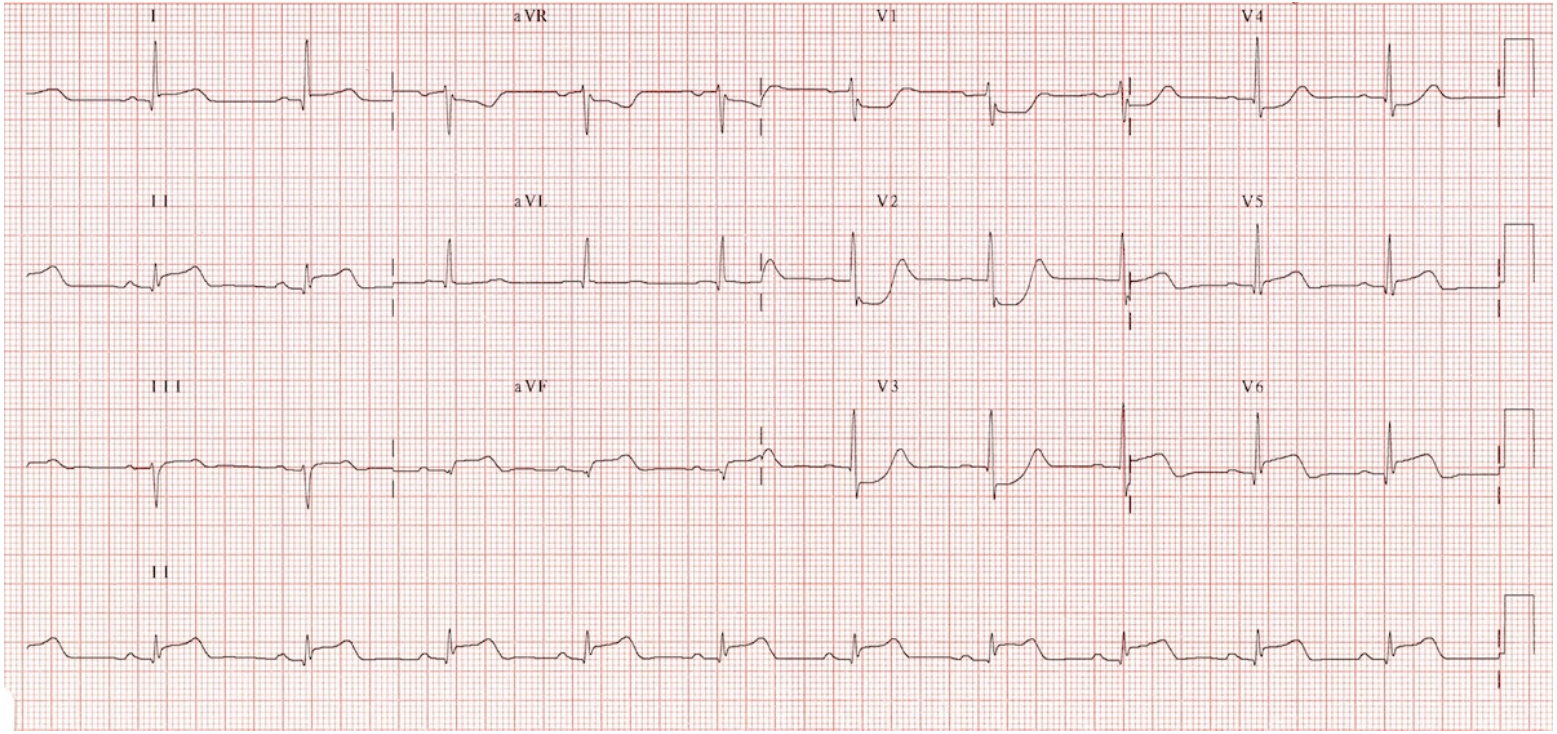


Evaluation 11

- Inferior STEMI
- Marked ST elevation in II, III and aVF with early Q-wave formation.
- Reciprocal changes in aVL.
- ST elevation in lead III > II with reciprocal change present in lead I and ST elevation in V1-2 suggests RCA occlusion with associated RV infarction: This patient should have right-sided leads to confirm this.

Patient EKG 12



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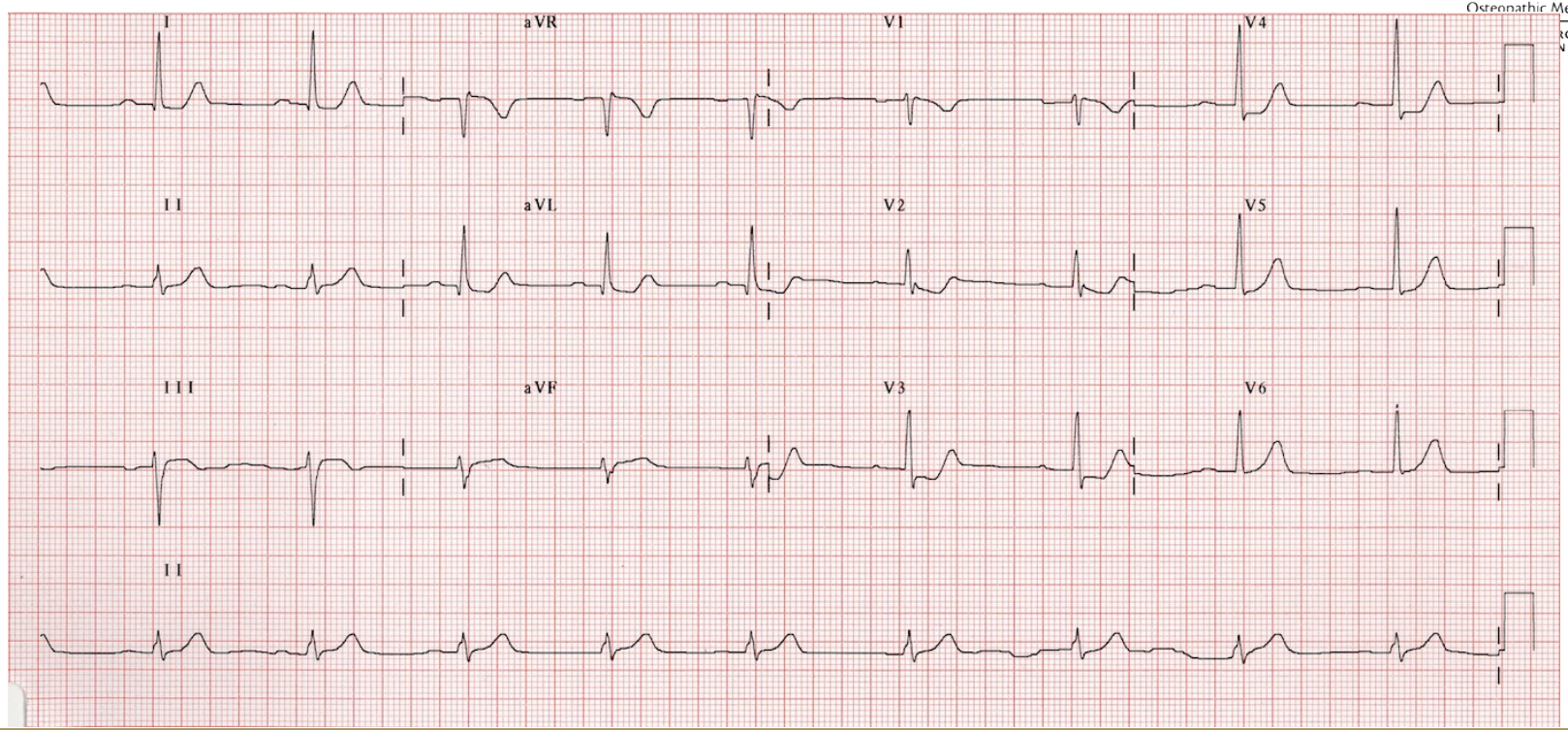
Evaluation 12

- Inferolateral STEMI with Posterior Extension.
- Horizontal ST depression in V1-3
- Tall, broad R waves (> 30ms) in V2-3
- Dominant R wave (R/S ratio > 1) in V2
- Upright T waves in V2-3

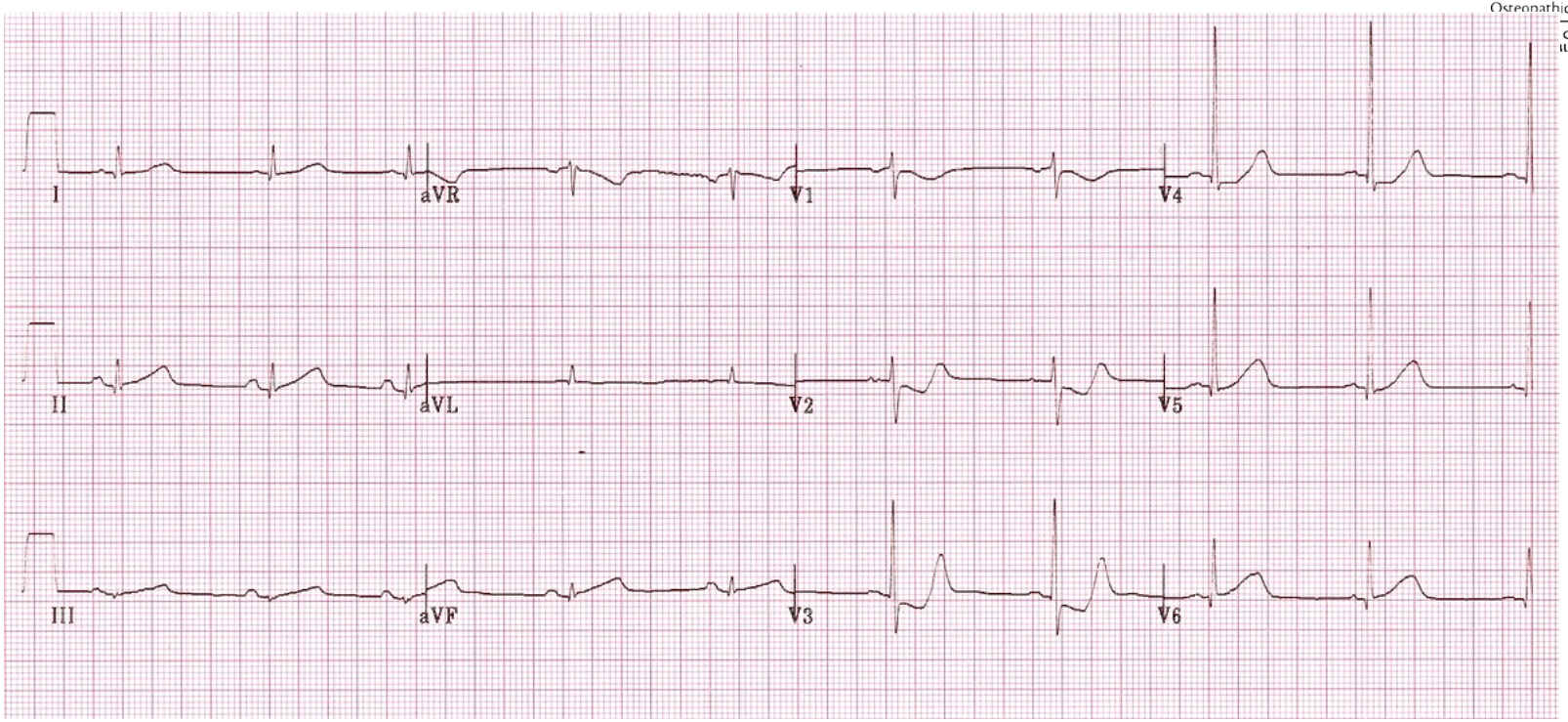
EKG 13



Evaluation 13

- Posterior MI
- ST depression in V2-3
- Tall, broad R waves (> 30ms) in V2-3
- Dominant R wave (R/S ratio > 1) in V2
- Upright terminal portions of the T waves in V2-3

EKG 14



Evaluation 14

- The ST depression and upright T waves in V2-3 suggest posterior MI.
- There are no dominant R waves in V1-2, but it is possible that this ECG was taken early in the course of the infarct, prior to pathological R-wave formation.
- There are also some features suggestive of early inferior infarction, with hyperacute T waves in II, III and aVF.

Resources and References

- Dubin, D. (2000). Rapid interpretation of EKG's: An interactive course. Tampa, Fla: Cover Pub. Co.

- Husain A Sattar - Fundamentals of Pathology - Chicago - Pathoma LLC. - 2011 - First Ed.