

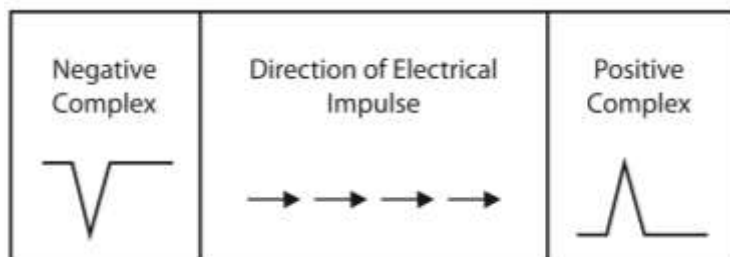
12 Lead ECG Basics

Objective

- Verbalize the ability to look at a 12-lead ECG and recognize normal waves and conduction.

I. Components of the 12 lead ECG and lead systems

- A. Purpose of the 12 lead is to record electrical activity as it flows through the heart. Also looks at multiple views of the heart.

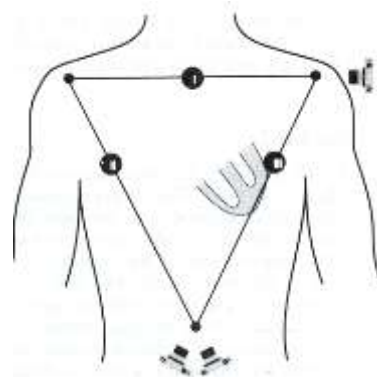


B. Types of leads

1. Bipolar leads
2. Unipolar leads
3. Limb leads
4. Precordial leads
5. See upright waves when the electricity travels toward the positive pole

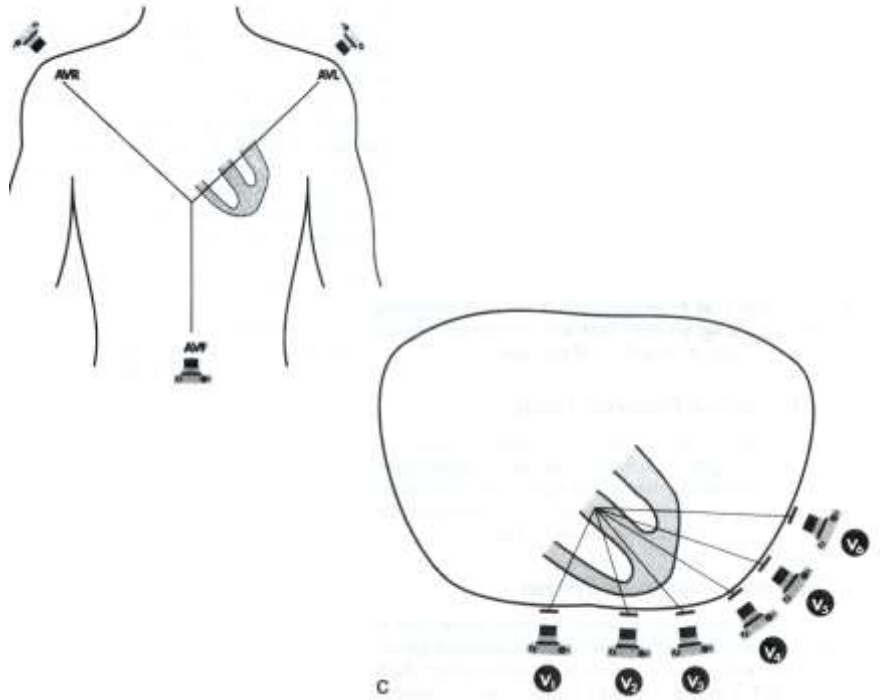
C. Bipolar leads

1. Lead I
2. Lead II
3. Lead III

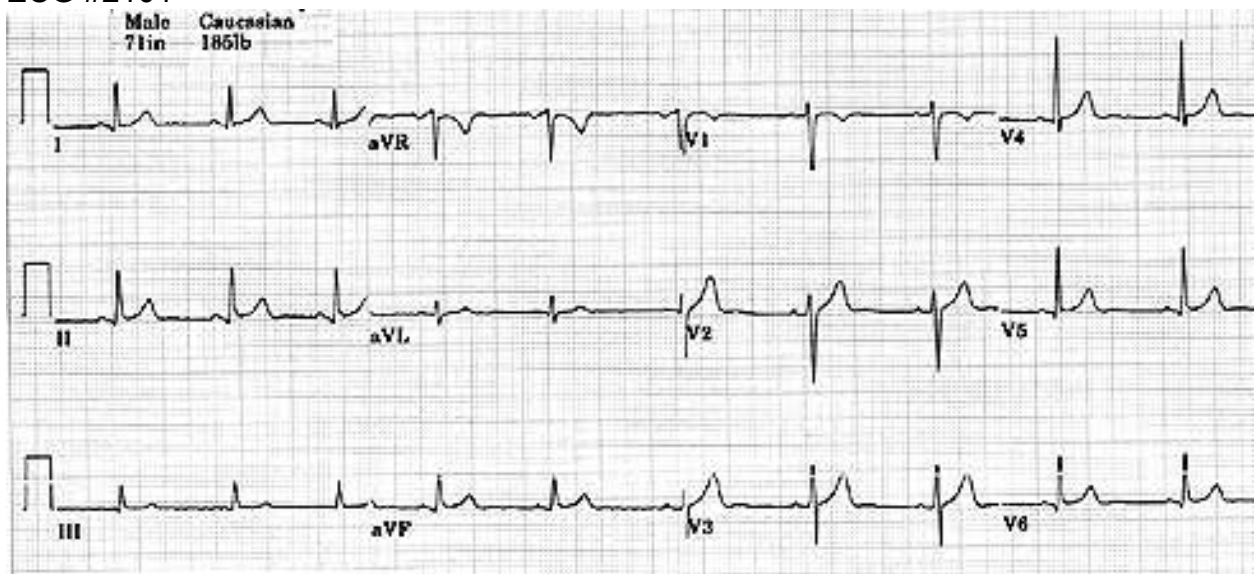


D. Unipolar leads

1. aVR
2. aVL
3. aVF
4. V1
5. V2
6. V3
7. V4
8. V5
9. V6
10. Right chest leads are V1R-V6R
11. Posterior leads are V7-V9 and V7R-V9R



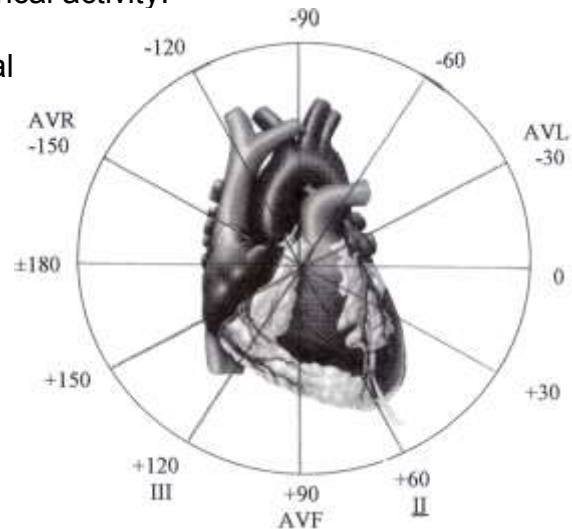
ECG #2101



II. The hexaxial reference system

A. Used to determine the axis of the electrical activity.

1. Where the majority of the electrical activity is flowing.
2. Divided into four quadrants by degrees.



B. Normal quadrant

C. Shift to the left

D. Shift to the right

E. No man's land (extreme right shift)

III. Normal sequence of conduction and depolarization and seeing it on the 12-lead

A. SA node

B. AV node

C. Bundle of His

D. Right bundle branch

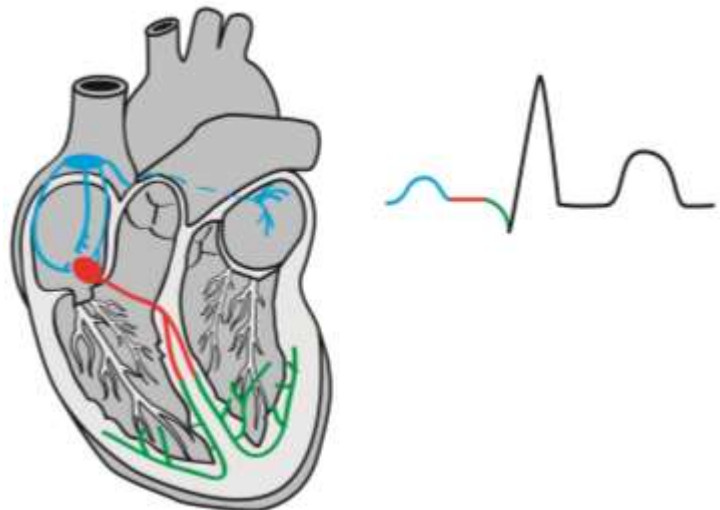
E. Left bundle branch

1. Anterior fascicle
2. Posterior fascicle

F. Purkinje system

G. P waves

1. First waveform on the ECG tracing.
2. Represents atrial depolarization.
3. Normal duration is not over 0.11 seconds.
4. Because the atria are thin walled structures, the deflection is small and not normally more than 2.5 mm in height.
5. P wave variables.



H. PR interval

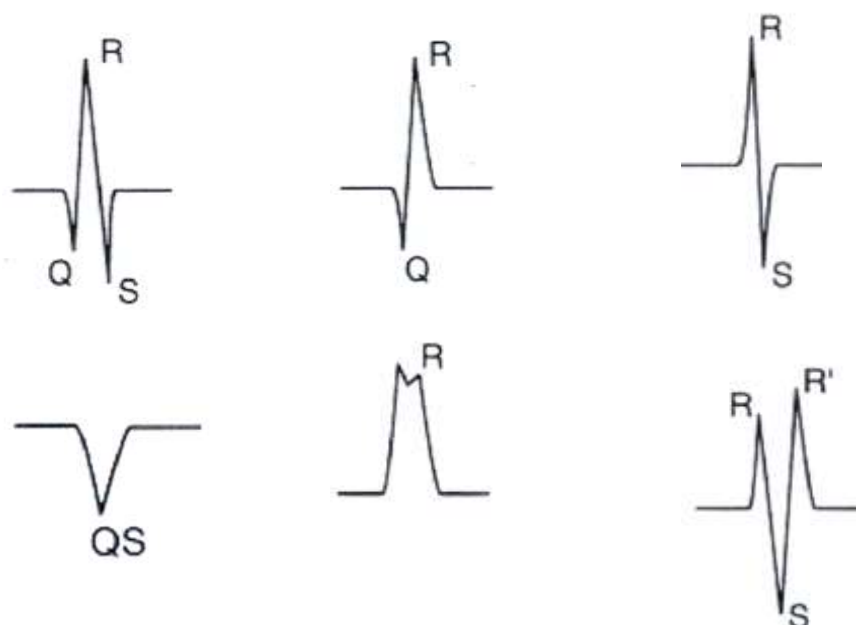
1. The period from the beginning of the P wave to the beginning of the QRS complex.
2. Normally it is 0.12 to 0.20 seconds.
3. Represents the time taken for the original impulse to leave the SA node and travel through the atria, the AV node, and until the onset of ventricular depolarization.
4. A shorter than normal PR interval implies accelerated conduction of the impulse to the ventricles either than a shorter than normal pathway or through a pacemaker site located close to the ventricles.
5. PR intervals longer than 0.20 seconds imply delayed conduction of the impulse to the ventricles. Long PR intervals are seen in heart block.

I. PR segment

1. Part of the PR interval.
2. End of the P wave to the beginning of the QRS complex.
3. Normally isoelectric.

J. QRS complex

1. Represents ventricular depolarization.
2. The QRS complex is composed of three waveforms. The Q, R, and S waves. Many variations exist in the shape of the QRS. Whatever the variation, it is still called a QRS complex in analyzing rhythms.



3. The Q wave is the first deflection when it is negative.
4. The R wave is the first positive deflection.
5. The S wave is the first positive inflection after the R wave that goes below the isoelectric line.
6. The normal QRS duration is less than 0.12 seconds. The QRS is measured from the beginning of the QRS as it leaves the isoelectric line until the end of the QRS when it returns to the isoelectric line. If the ST segment is elevated or depressed, the end of the QRS is when the ST segment begins.
7. The QRS may be prolonged with ventricular hypertrophy since the electrical impulse needs more time to get through the thickened myocardium. May also be prolonged with a defect in conduction such as bundle branch block, intraventricular conduction defect, or WPW.
8. May have low voltage QRS complexes.

K. ST segment

1. The ST segment is the straight line between the QRS and the T wave.
2. Represents the time between the completion of ventricular depolarization and the beginning of repolarization (T wave) and is sometimes referred to as the resting phase of the cardiac cycle.
3. The beginning of the P wave is the baseline from which ST segment deviation is judged.
4. ST segment elevation indicates myocardial injury. The ST segments associated with injury are elevated above the baseline, usually in an upward curving manner.
5. ST segment depression indicates myocardial ischemia. The ST segment associated with ischemia is depressed below the baseline in a horizontal line, or they sag downward like a sagging clothesline.
6. Scooped ST segments are most often seen with digoxin toxicity.

L. T wave

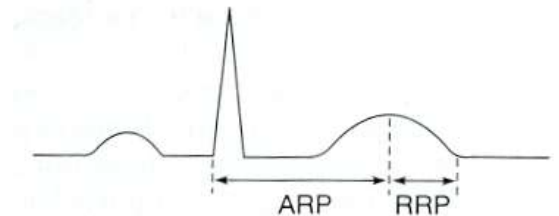
1. Represents ventricular repolarization.
2. The T wave is a rounded waveform, taller and wider than the P wave. It follows the QRS complex and the ST segment.
3. Normal T waves are upright in the most common monitoring leads and may be upright or inverted in V1.
4. Myocardial ischemia produces T wave changes. Typically the T waves of ischemia are inverted and sharply pointed.
5. Tall, peaked T waves can be indicative of hyperkalemia.

M. U wave

1. Thought to represent the terminal phase of ventricular repolarization.
2. The U wave is a small, rounded, low-amplitude deflection occurring just after the T wave and before the next P wave.

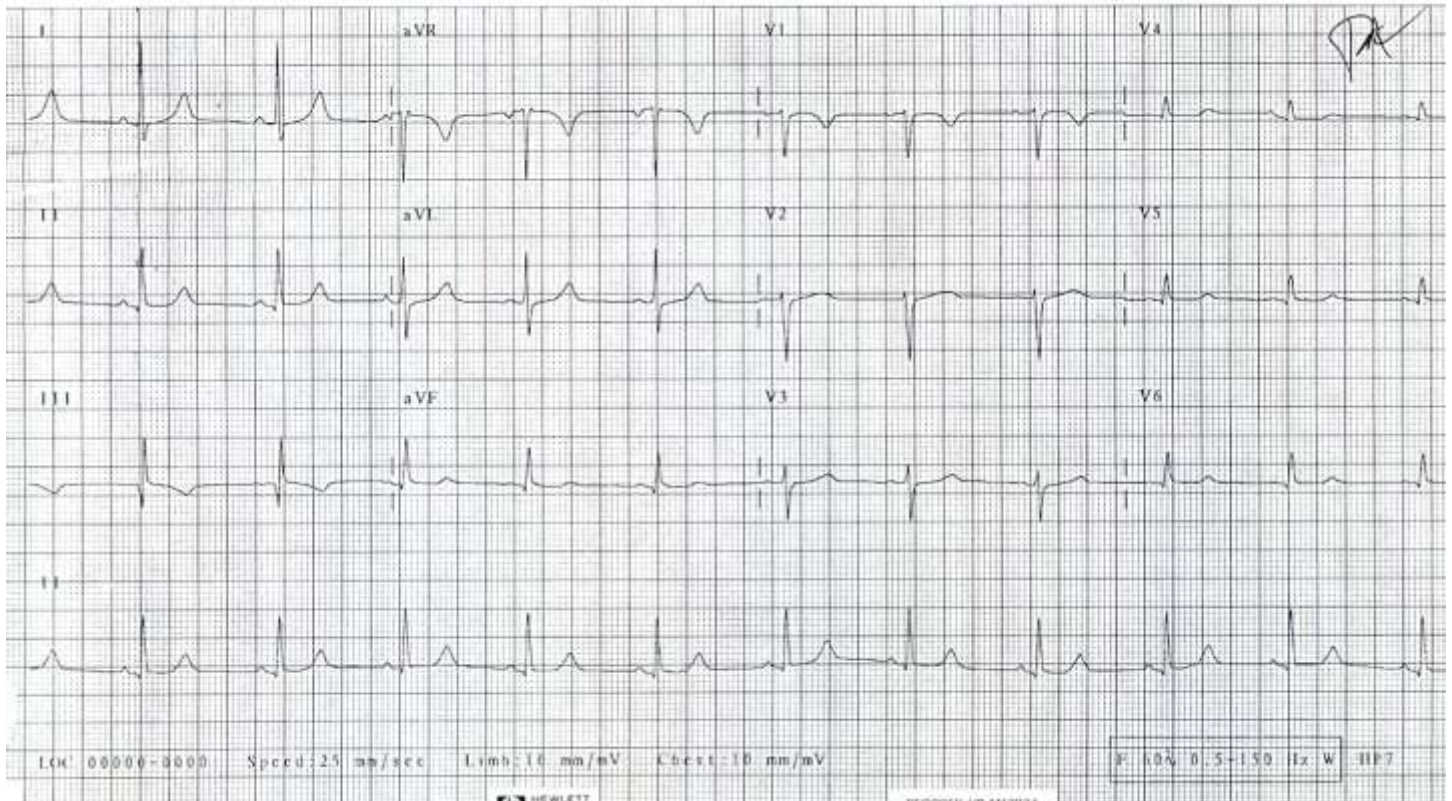
N. QT interval

1. Period from the beginning of ventricular depolarization until the end of ventricular repolarization. The QT interval is measured from the onset of the QRS complex until the end of the T wave.
2. During the initial portion of this interval the heart is completely refractory to all premature stimuli.
3. Partial refractoriness of the conduction system is seen during the latter portion of this interval.



O. The normal 12 lead: R wave progression

ECG #2101



IV. Axis determination

A. Movement toward the most muscle mass.

B. Can actually determine axis for P waves, QRS complexes and T waves.

C. Usually axis refers to the QRS complex.

D. Normal axis.

E. Left axis deviation

- Left ventricular hypertrophy (LVH)
- Left bundle branch block (BBB)
- Left anterior hemiblock (LAH)
- Ventricular tachycardia
- Inferior MI
- Wolf-Parkinson-White (WPW)

F. Right axis deviation

- Right ventricular hypertrophy (RVH)
- Right bundle branch block (BBB)
- Left posterior hemiblock (LPH)
- Ventricular tachycardia
- Dextrocardia
- Wolf-Parkinson-White (WPW)

G. Indeterminate

- Ventricular tachycardia
- Bifascicular block

H. Determining axis

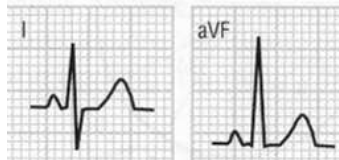
1. Look at the computer readout
2. Forces on the frontal plane
3. “Quick and dirty”
4. Equiphasic limb lead

I. Forces on the frontal plane

1. Most accurate which averages the forces moving right and left with those moving up and down.
2. Uses lead I which is the most direct right and left lead. Use aVF which is the most direct up and down lead.
3. Counts the blocks and intersects the lines to get the most accurate axis.
 1. Look at the QRS in aVF. Count the positive and negative boxes and get a sum. Mark this on the graph.
 2. Draw a perpendicular line
 3. Then draw a line for the degrees

J. "Quick and dirty"

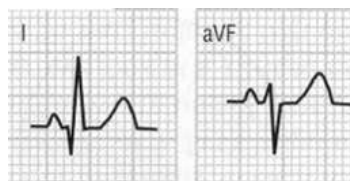
1. Practice time: Which way are the complexes?
2. "Quick and dirty": Look at leads I and aVF. Usually they are both positive. When both positive, axis is normal. When they are both positive, they both point upward.



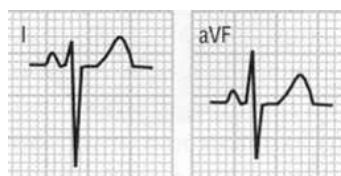
- a. Now, remember, "right together, left apart".
- b. When they point toward each other, lead I points down and lead aVF points up, they are together and thus a right axis deviation.



- c. When they point opposite from each other, lead I points up and lead aVF points down, they are apart and thus a left axis deviation.

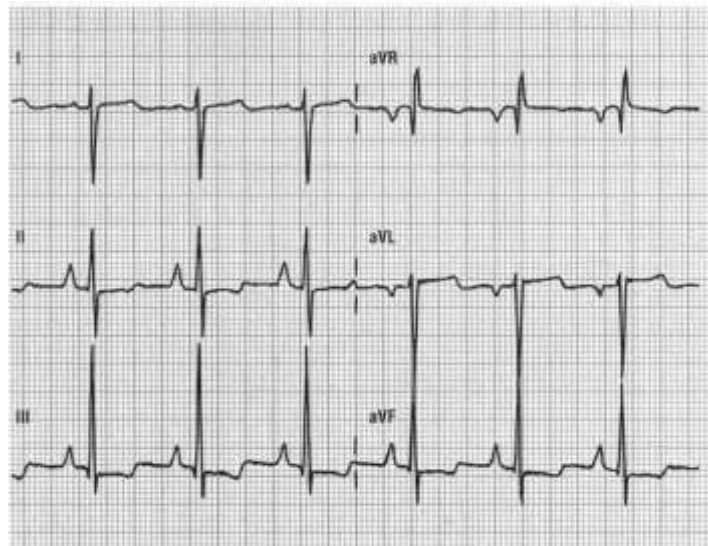
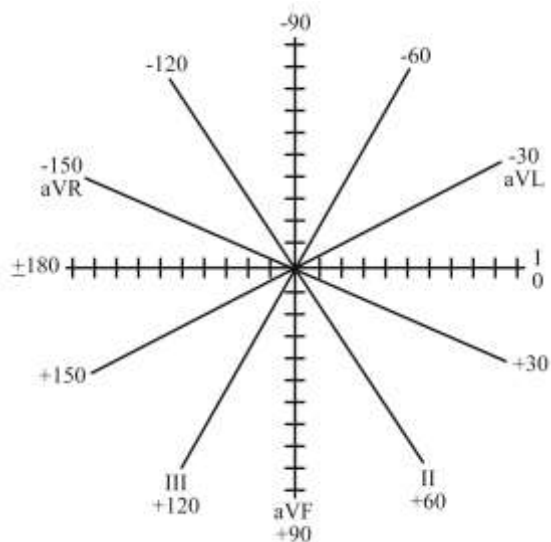
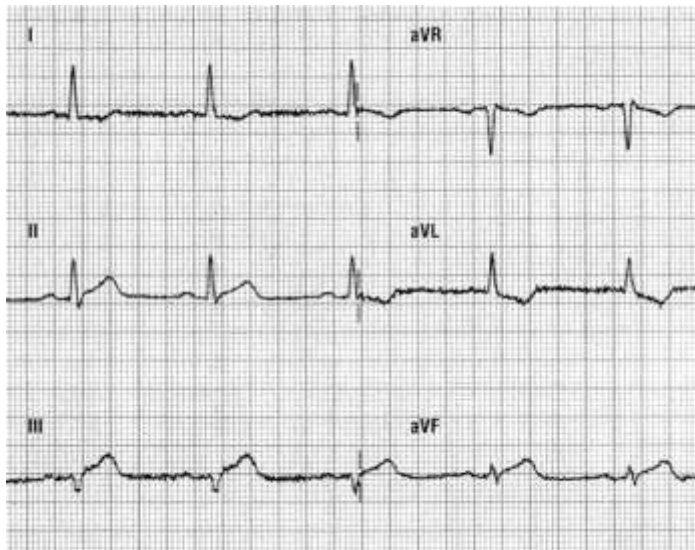
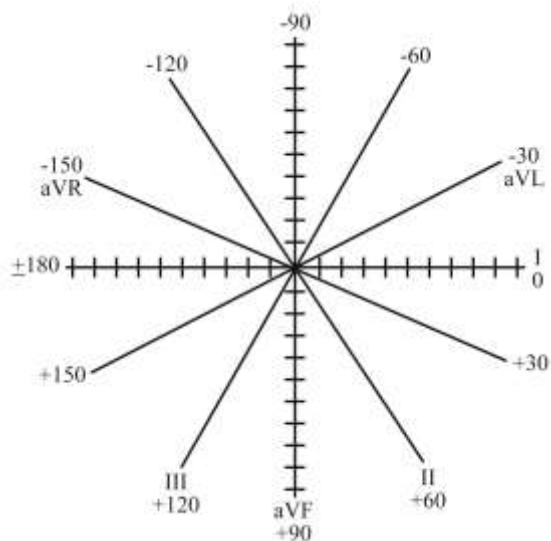


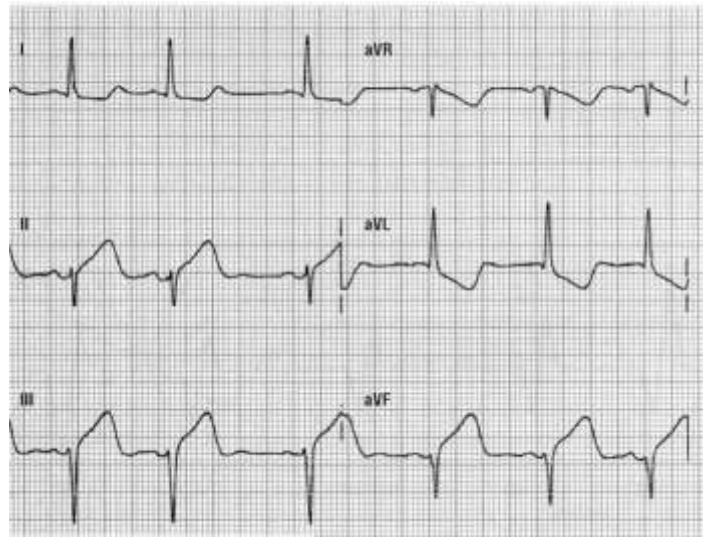
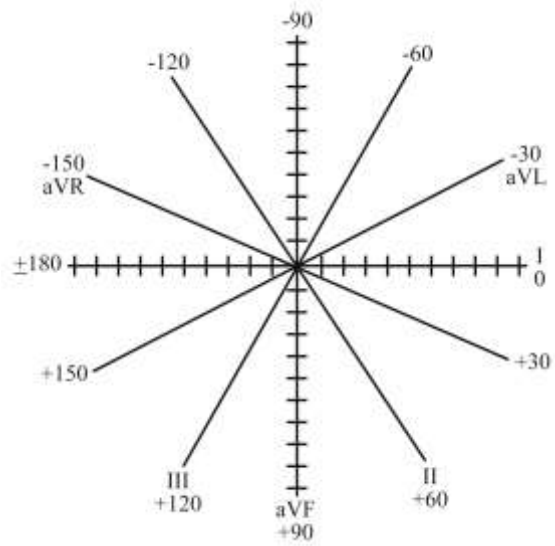
- d. When they both point down, they are "indeterminate" or in "no man's land"



K. Equiphase limb lead

1. Do "quick and dirty"
2. Of the limb leads, determine which is the most equiphase (or the smallest).
3. Then determine which lead is perpendicular to the equiphase lead.





Intraventricular Conduction Delays

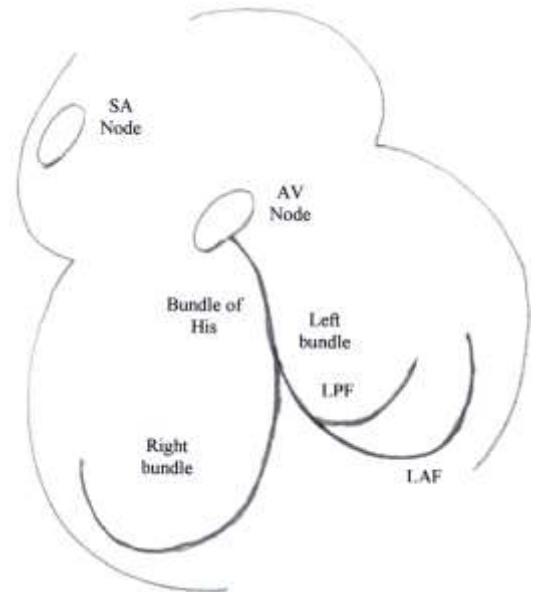
Objective

- Identify intraventricular conduction delays which may be present on the 12 lead ECG.

I. Intraventricular conduction delays

A. Conduction review

1. Bundle of His
2. Left main bundle
3. Left anterior fascicle
4. Left posterior fascicle
5. Right bundle
6. When conduction occurs down other sides of the conduction system, an upright pattern results in lead I and lead III.



B. Types of blocks

1. Monofasicular is when one part is blocked
2. Bifasicular is when two parts are blocked.
 - RBBB combined with LAH
 - RBBB combined with LPH

C. Bundle branch blocks

1. Can be either bundle.
2. QRS widens to 0.12 or more seconds.
3. Asynchronous depolarization of the ventricles.
4. To determine BBB, look at leads I, V6 and V1.

D. Right bundle branch block

1. Septal activation occurs from left to right.
2. Left ventricle depolarizes.
3. Right ventricle depolarizes.
4. S wave in I and V6. Slurred in V6.
5. Confirm in lead V1 with a triphasic and upright complex.

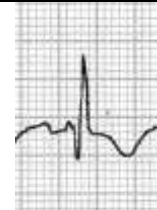
Normal lead I	RBBB in lead I
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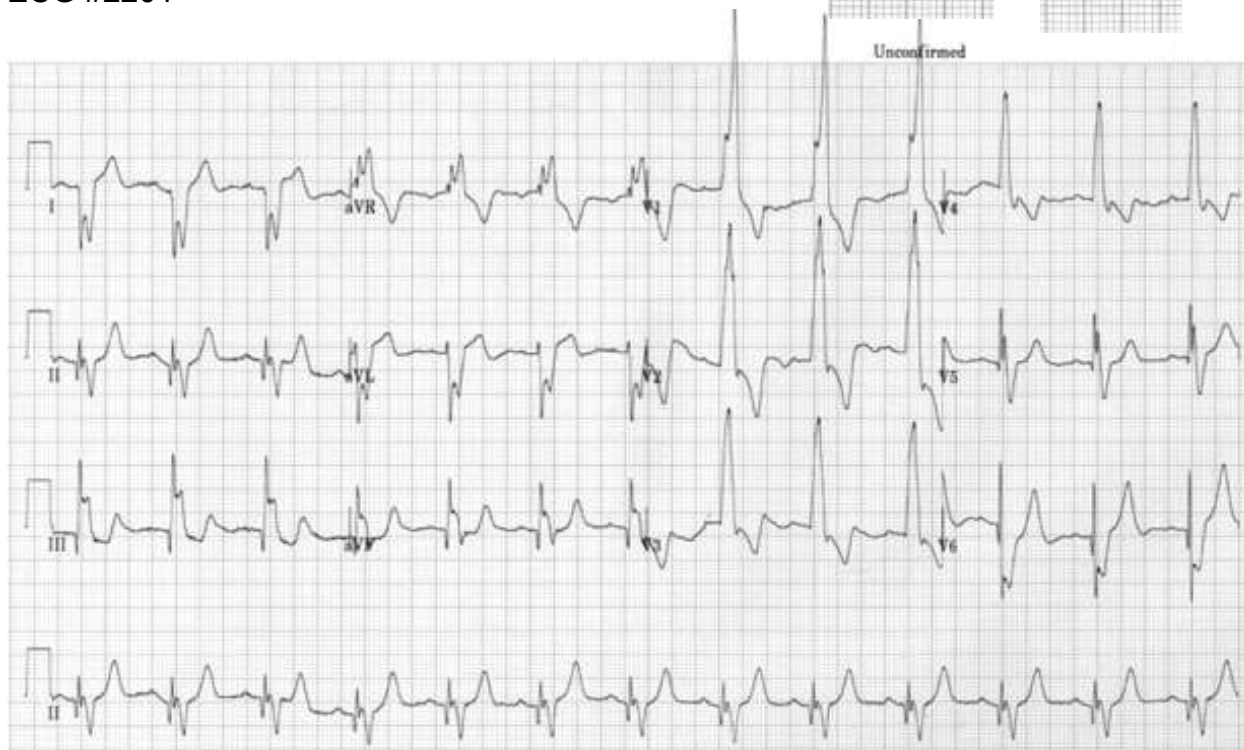
Normal V6	RBBB in V6
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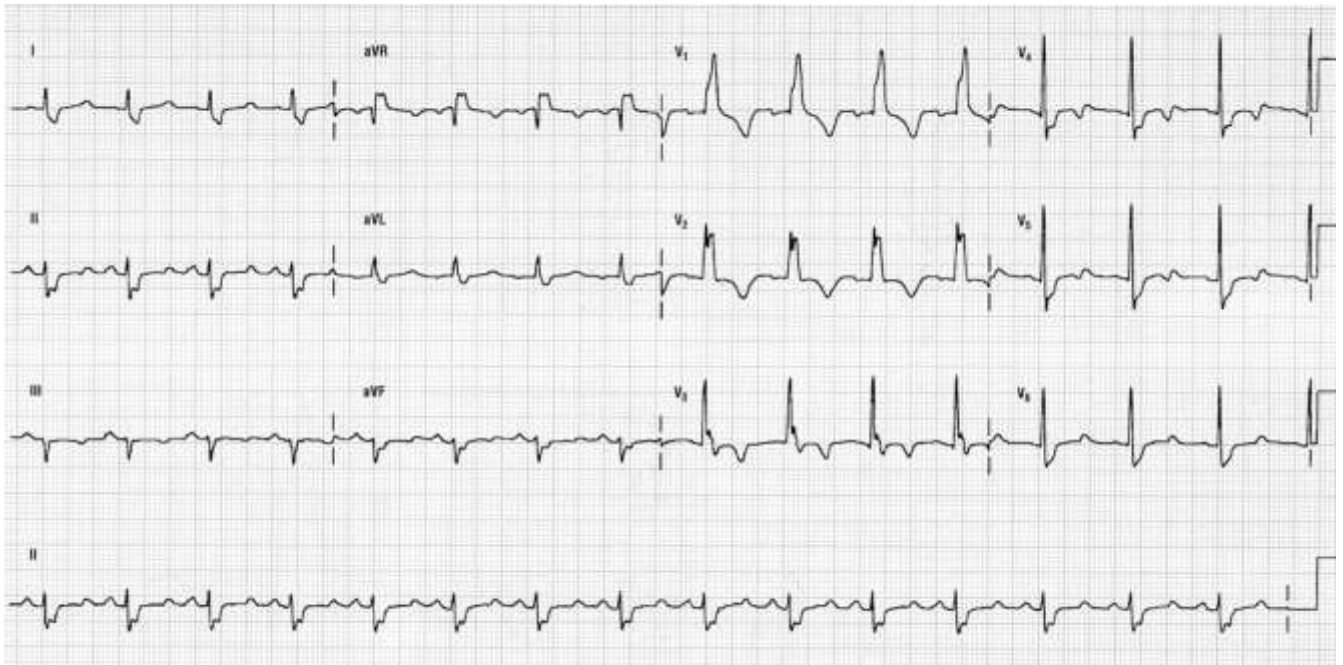
Normal V1	RBBB in V1
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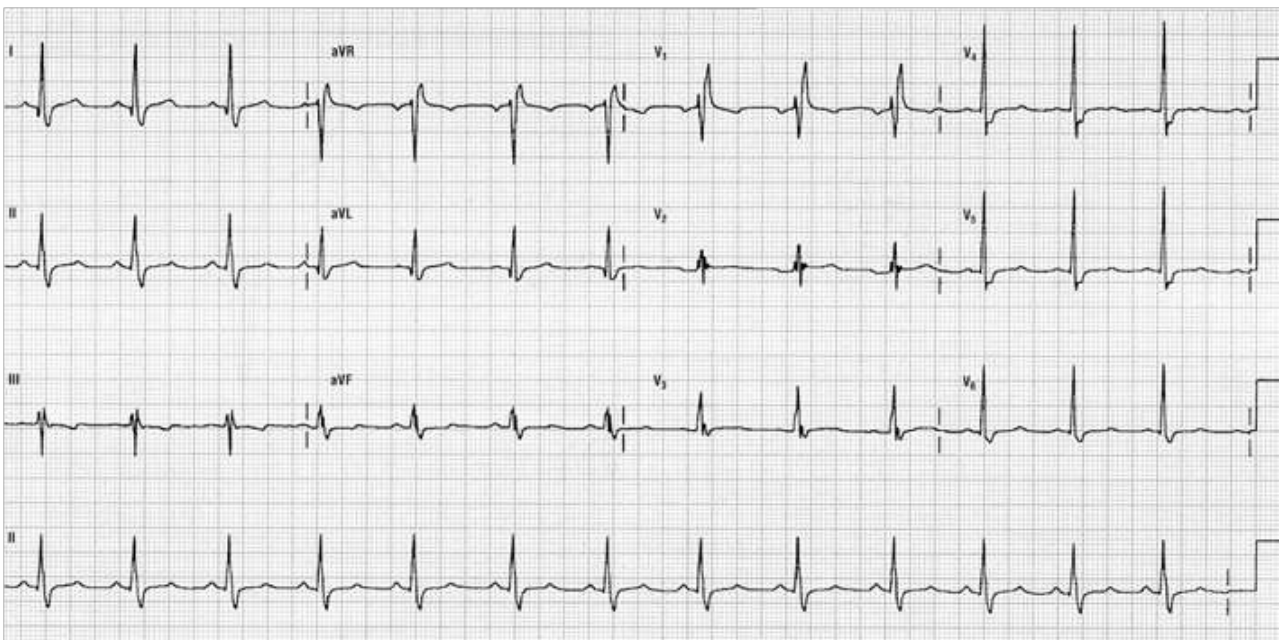
ECG #2201



ECG #2202

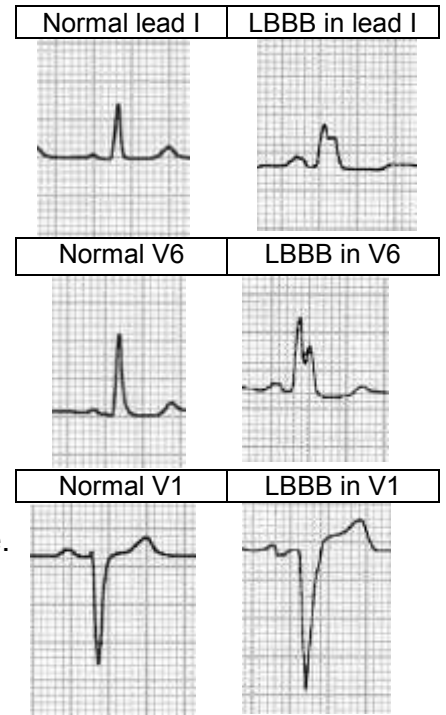


ECG #2203



E. Left bundle branch block

1. Septum now unable to depolarize in the normal left to right direction.
2. Because of this, identification of AMI difficult in the setting of LBBB.
3. Need to carefully evaluate the symptoms.
4. See a monophasic pattern.
5. Look in lead I, V6, and then V1.
6. Best seen in lead V1. Monophasic and negative.



F. ST and T wave changes with bundle branch blocks

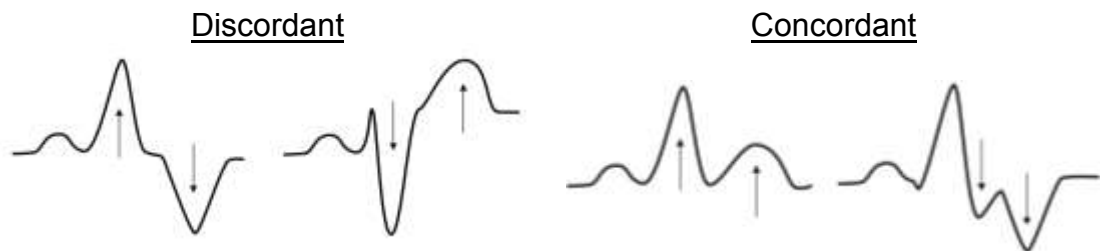
1. Discordance is expected.

“Dismiss it, it is not dangerous.”

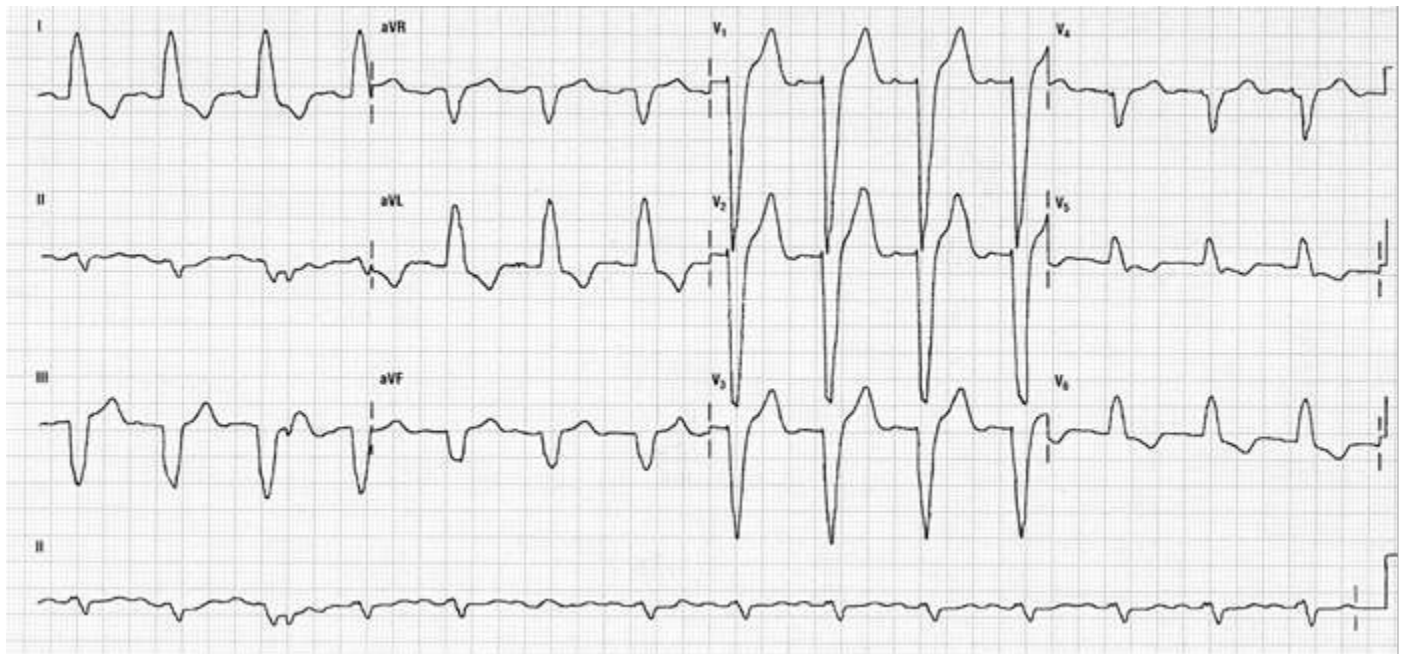
2. In a BBB, the T wave should be opposite of the QRS complex.
3. Concordance is pathological and not normal.

“Consider an MI”

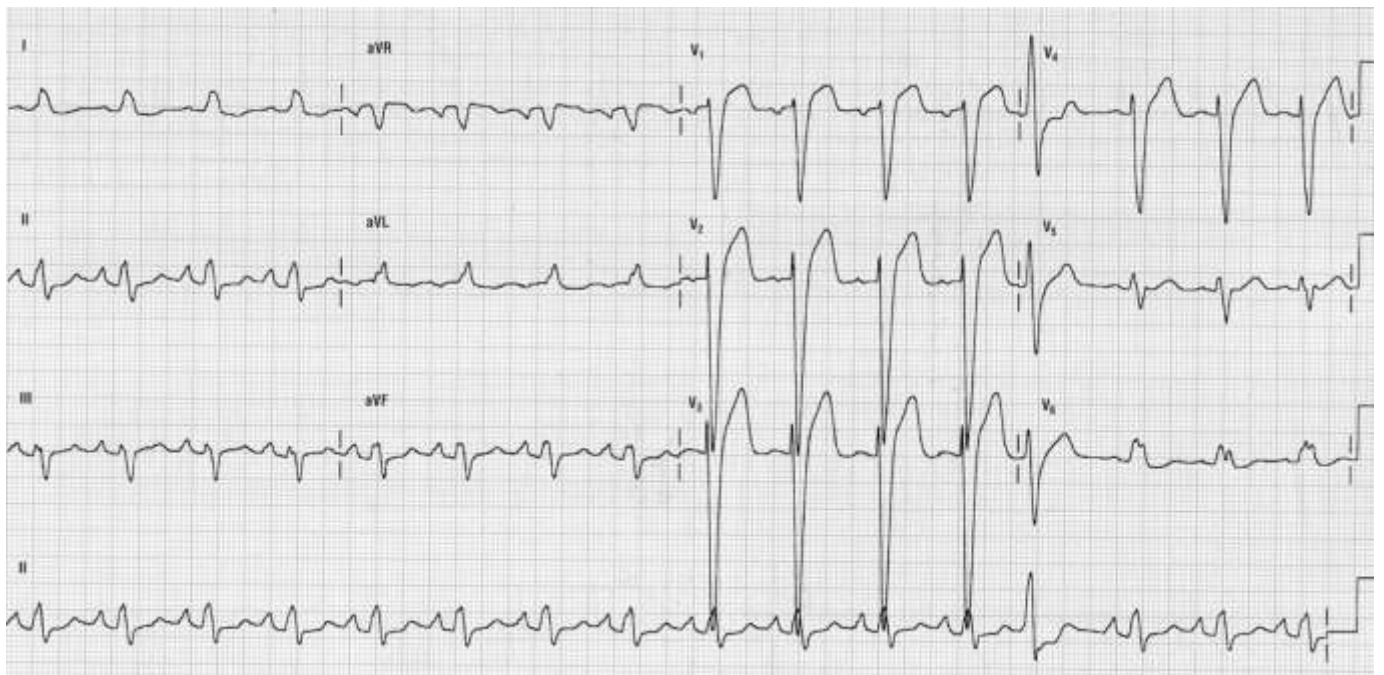
4. This is when the T wave is the same direction of the QRS complex. The only time it is normal is when it is present in old ECGs.



ECG #2204

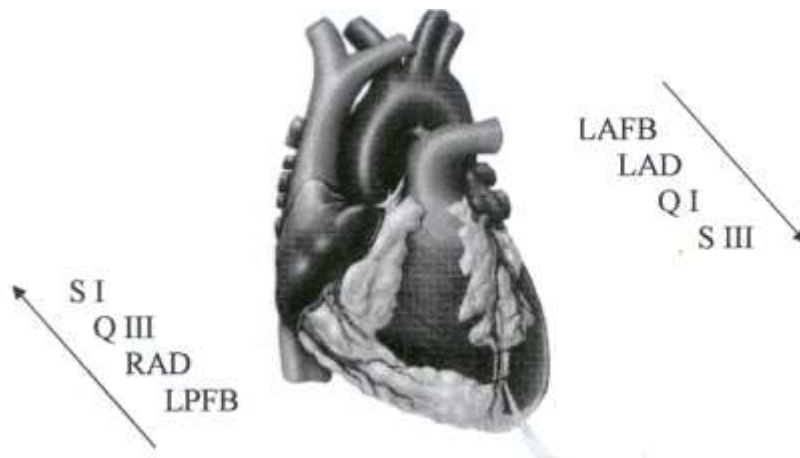


ECG #2205



G. Fascicular blocks

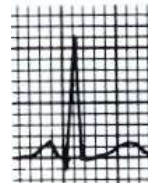
1. Block occurs in one of the fascicles of the left bundle.
2. Can be a left anterior fascicular block or a left posterior fascicular block.
3. QRS normal. Because the ventricles will both still depolarize, the QRS doesn't widen.
4. Axis deviation will occur with a fascicular block. The diagnosis of a fascicular block is a diagnosis of exclusion.



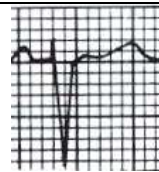
H. Left anterior fascicular block

1. Left anterior fascicle is organized into a tight bundle and easy to damage.
2. Left axis deviation occurs and a Q wave is seen in I and an S wave is seen in III.

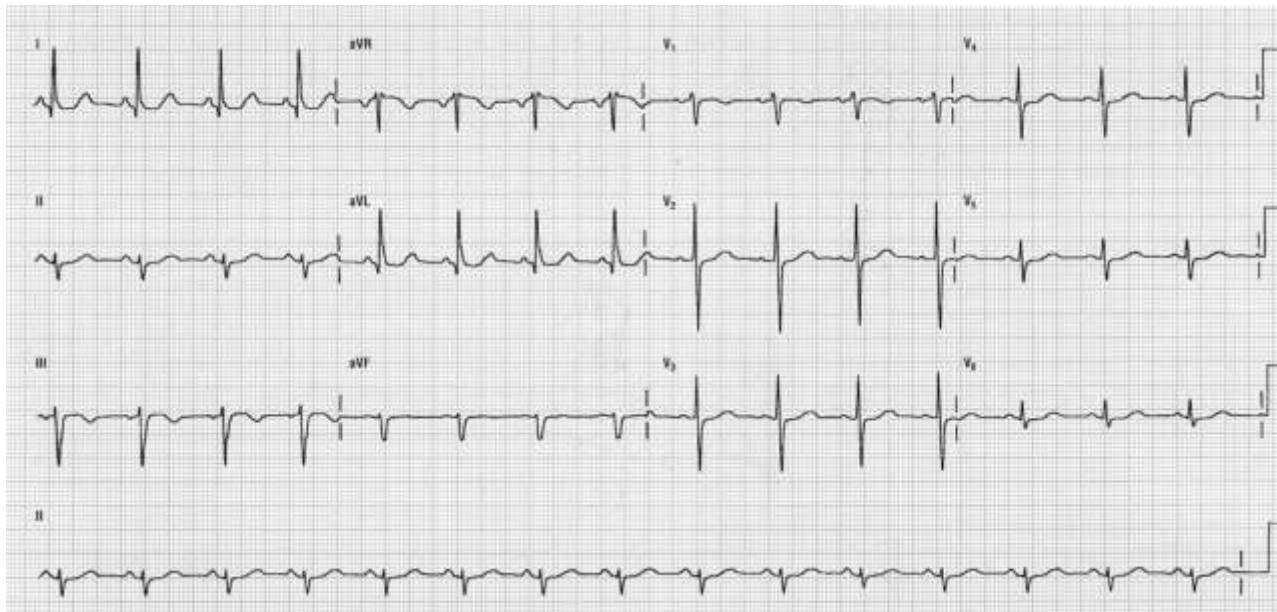
LAFB in lead I

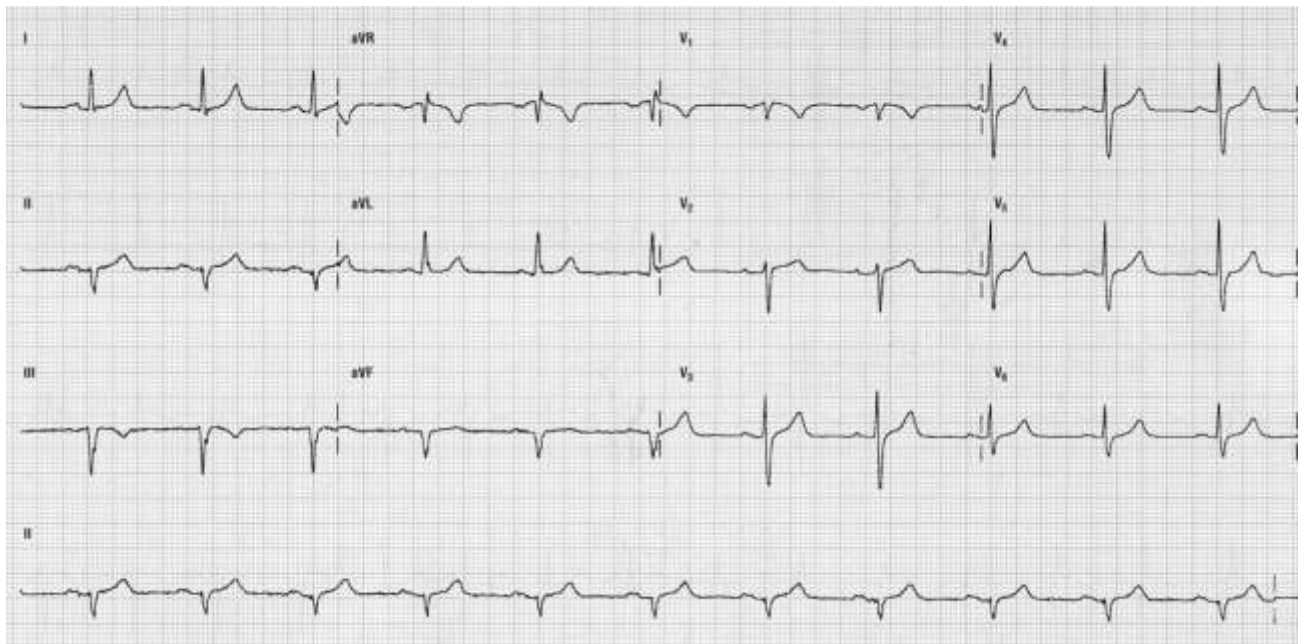


LAFB in III



ECG #2206

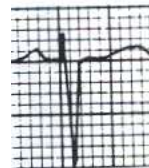




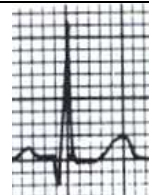
I. Left posterior fascicular block

1. The left posterior fascicle is loosely dispersed and fans out. It carries a dual blood supply.
2. Left posterior fascicle is difficult to damage. More commonly found in patients with CAD, HTN, or VHD.
3. Right axis deviation occurs and a Q wave is seen in III and an S wave in I.

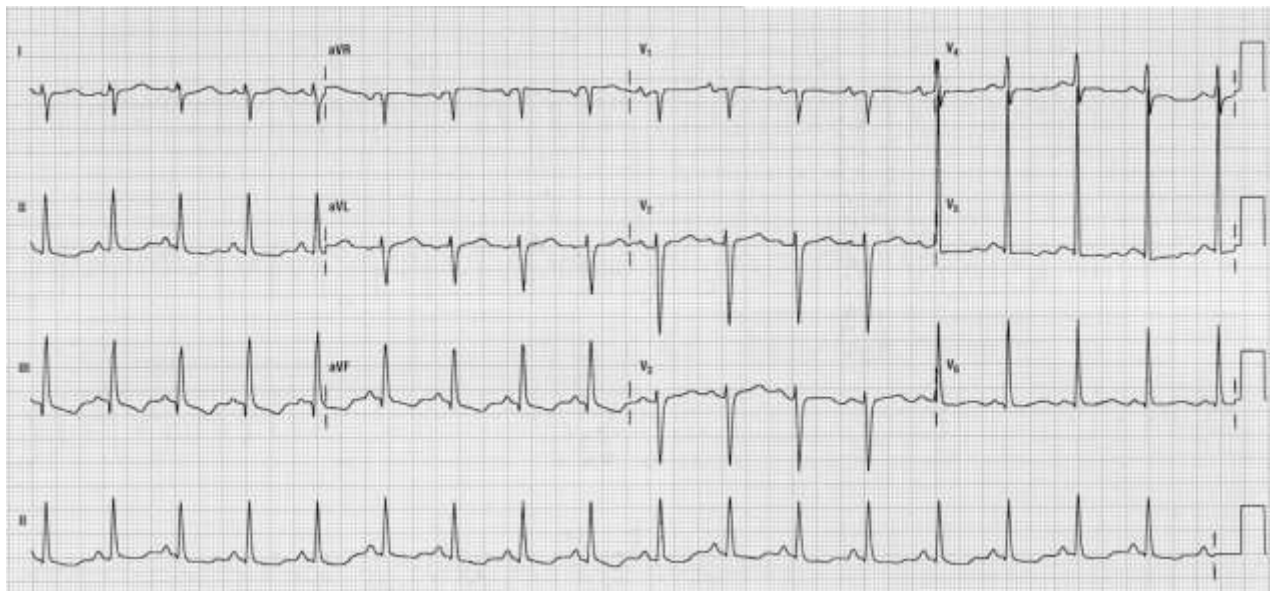
LPFB in lead I



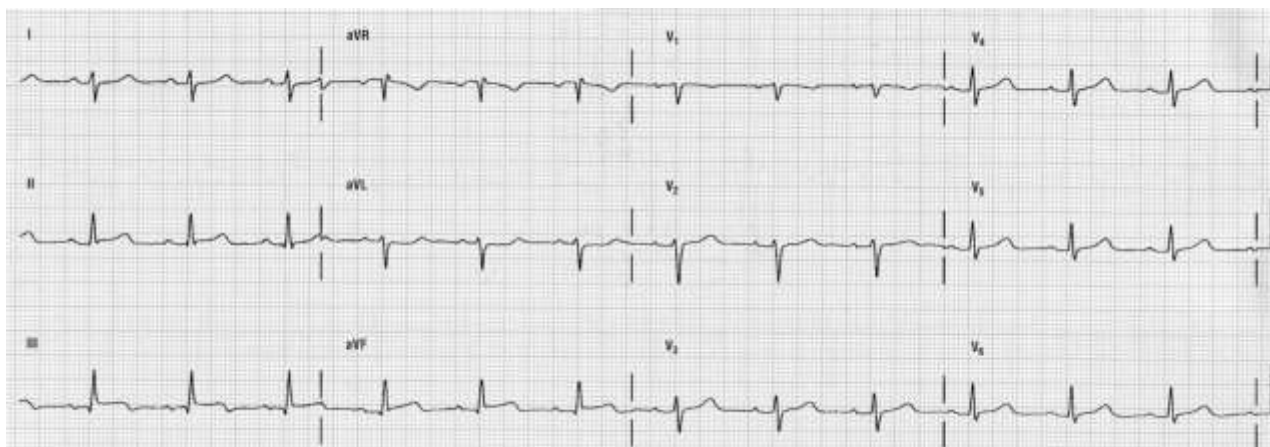
LPFB in III



ECG #2208



ECG #2209



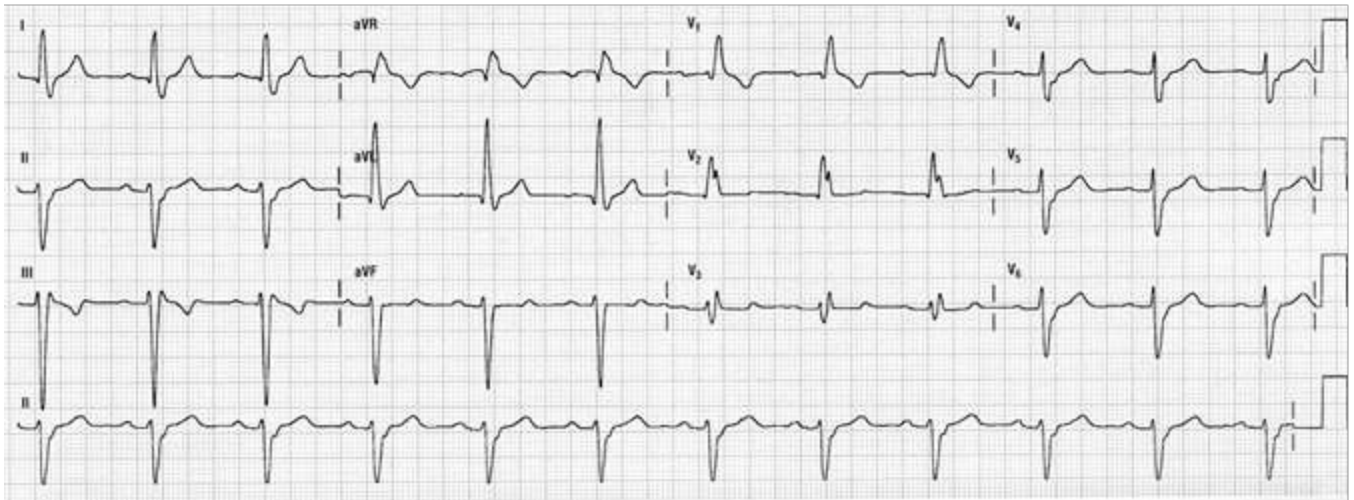
J. Right bundle branch block and a fascicular block

1. Two of the three fascicles which depolarize the ventricles are now blocked.
2. See a right BBB pattern plus axis deviation and evidence of a hemiblock by looking at leads I and III.

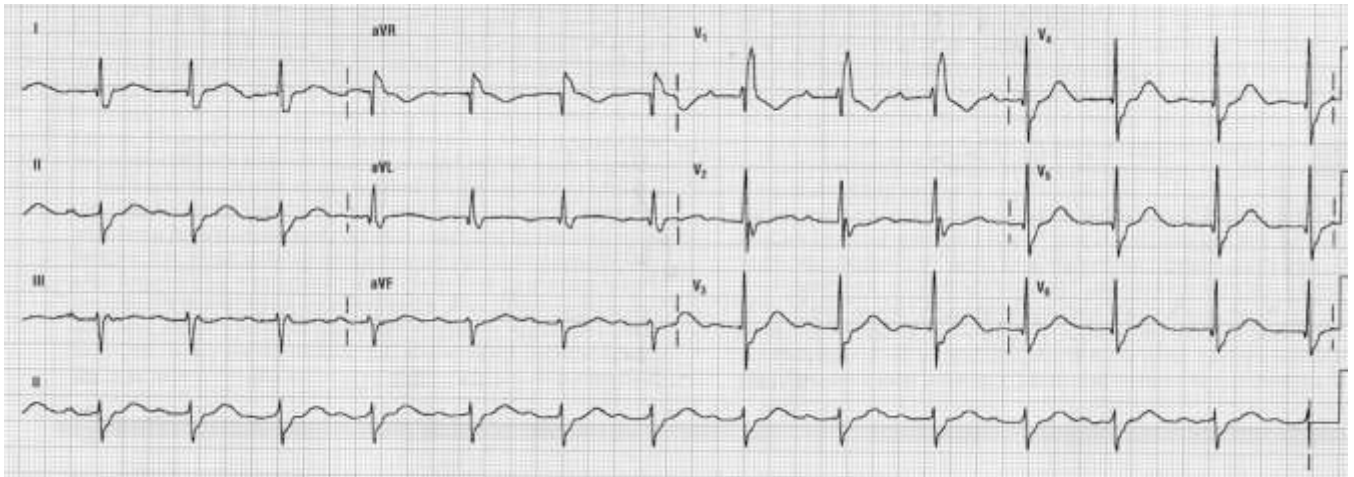
K. Right BBB with LAFB

1. More common together than LPFB by itself.
2. Very unstable pattern in patients with AMI. Deteriorates into complete heart block.
3. RBBB
4. Right axis deviation
5. Q in III and S in I

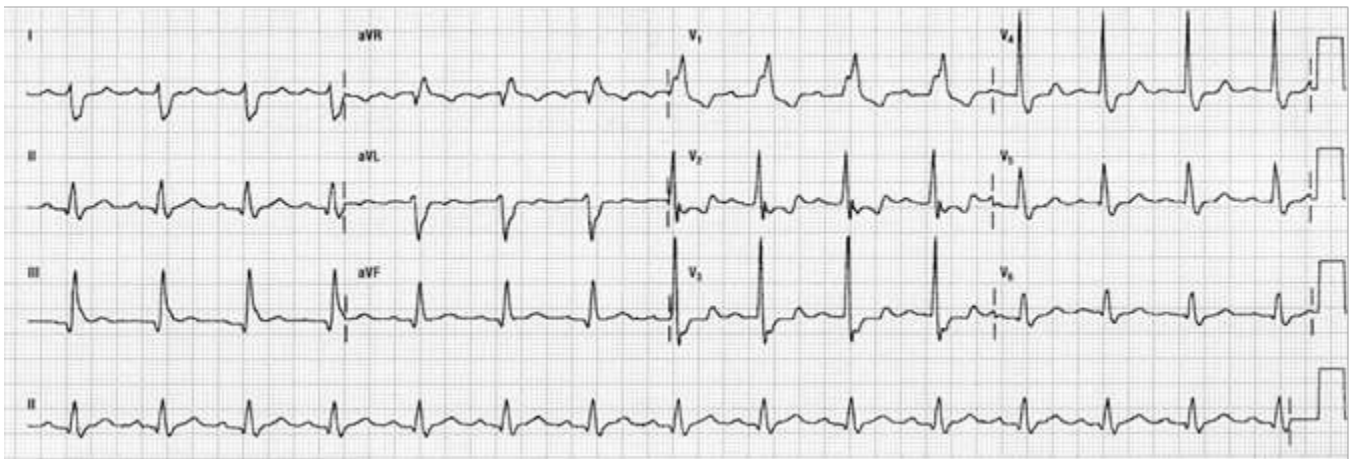
ECG #2210



ECG #2211



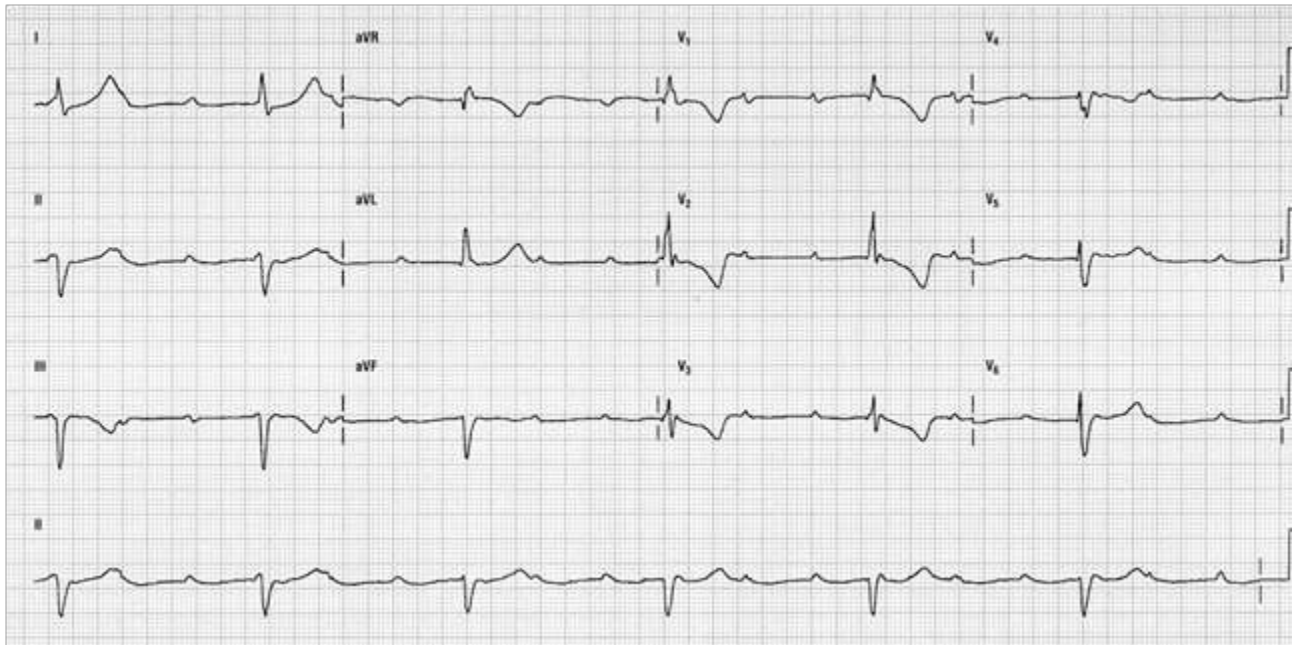
ECG #2212



ECG #2701 (G10-21)

Clinical correlation

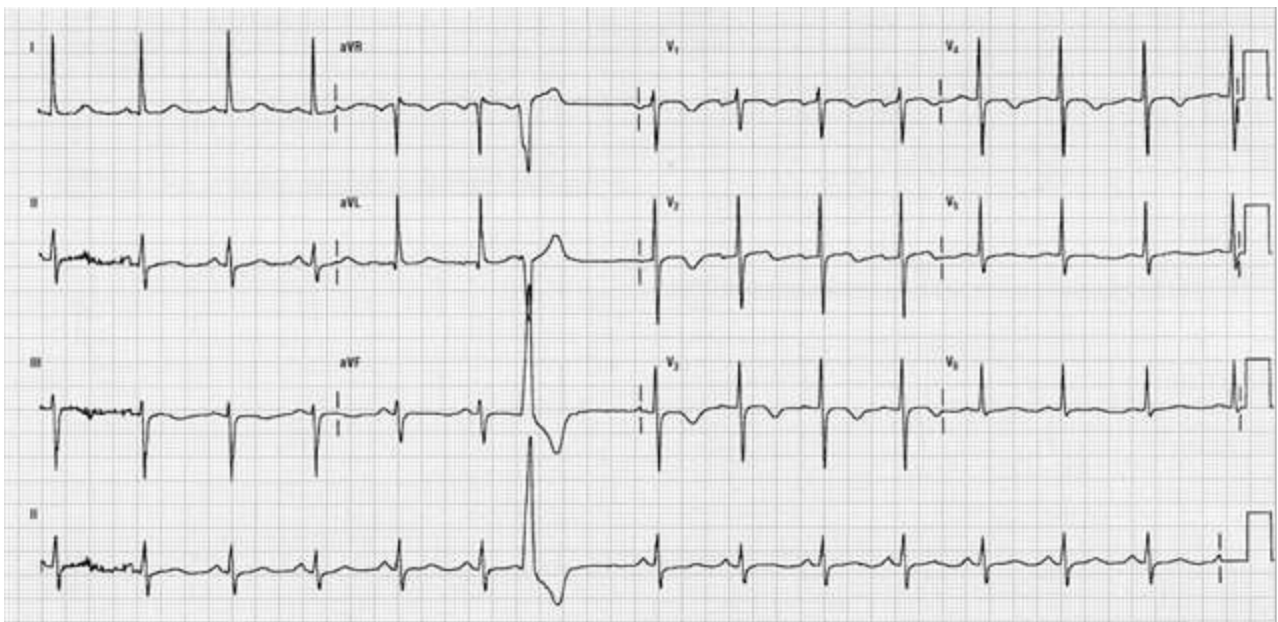
- Patient is a 72-year-old male presenting with chest pain and feeling light-headed.
- HR is 35 BPM.



ECG #2702 (G11-13)

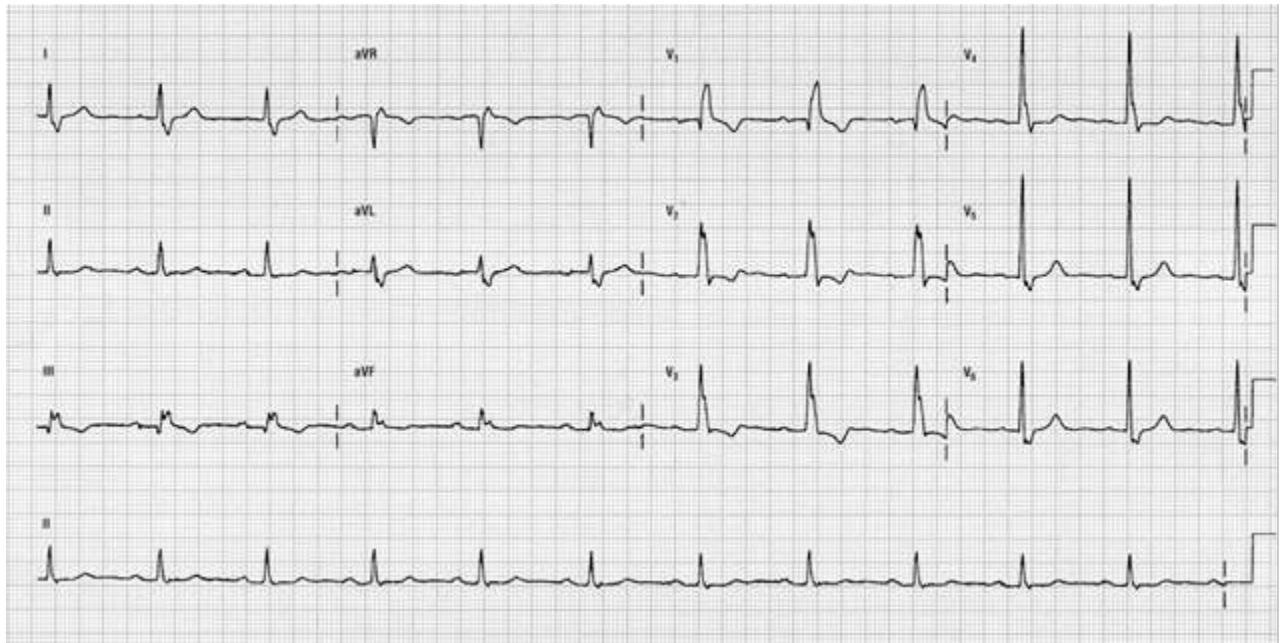
Clinical correlation

- Patient is a 72-year-old male admitted to the hospital with pneumonia. He has a history of coronary artery disease and heart failure.
- He is not complaining of any chest pain or dyspnea.



Clinical correlation

- Patient is a 62-year-old male admitted to the hospital with acute systolic heart failure.
- HR is 70. He is not currently complaining of chest pain but is dyspneic on exertion.



Ischemia, Injury and Infarction

Objective

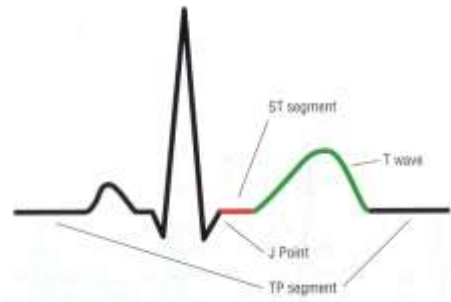
- Describe the ECG changes and patterns seen in myocardial ischemia and infarction.

I. ST segment and J point

A. The ST segment is the time when the ventricles are between the completion of depolarization and repolarization.

B. ST segment is measured from the J point.

1. Isoelectric
2. Elevated
3. Depressed



C. Concave up indicates early repolarization or pericarditis.

D. Flat and depressed ST segment indicates a non-STEMI.

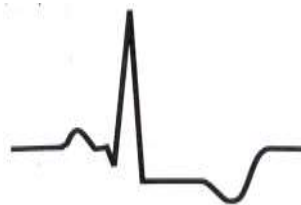
E. Flat and elevated considered an injury pattern.

F. Tombstone appearance considered an injury pattern but may also be a ventricular aneurysm.

Concave up



Flat and depressed



Flat and elevated



Tombstone

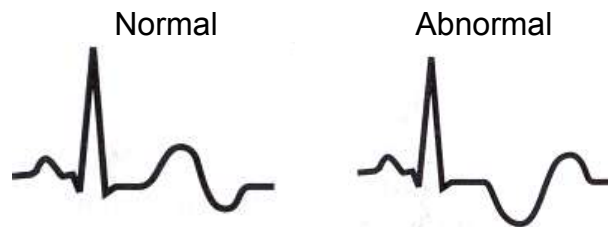


G. Inverted U waves indicate ischemia but are rarely seen.

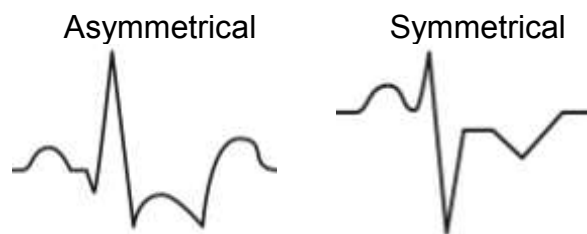


II. T waves

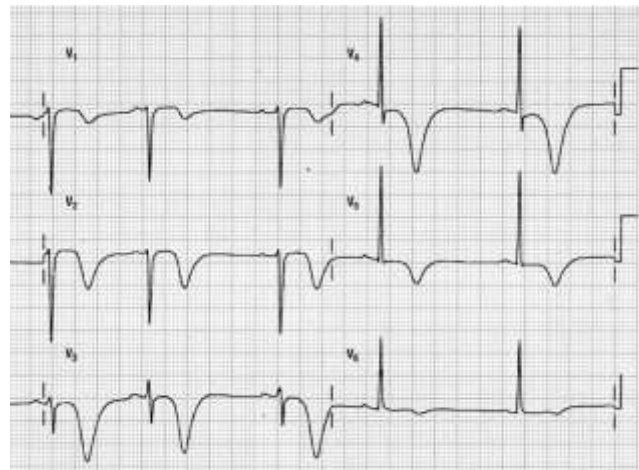
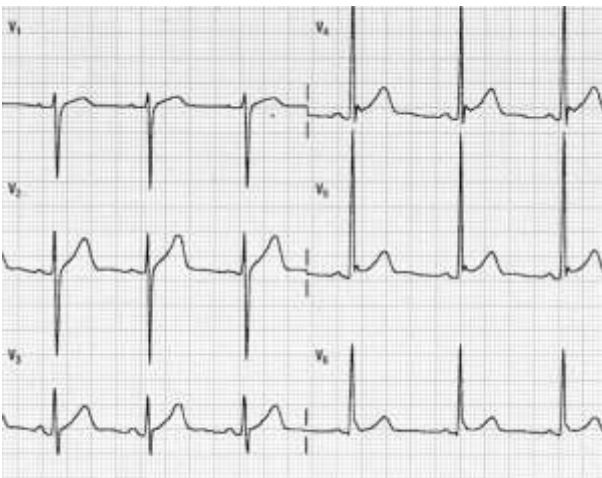
- A. T waves are usually upright in leads I, II and V3 to V6. They are always negative in aVR.
- B. T waves can be biphasic. Biphasic pathological is negative and then positive.



- C. Symmetrical T waves indicate problems, asymmetrical usually do not.



- D. In general, T waves should not be more than 6 mm high in the limb leads and 12 mm high in the precordial leads. A good rule is: If the T wave is more than $\frac{2}{3}$ the height of the R wave, it is abnormal.
- E. Flipped T waves concerning



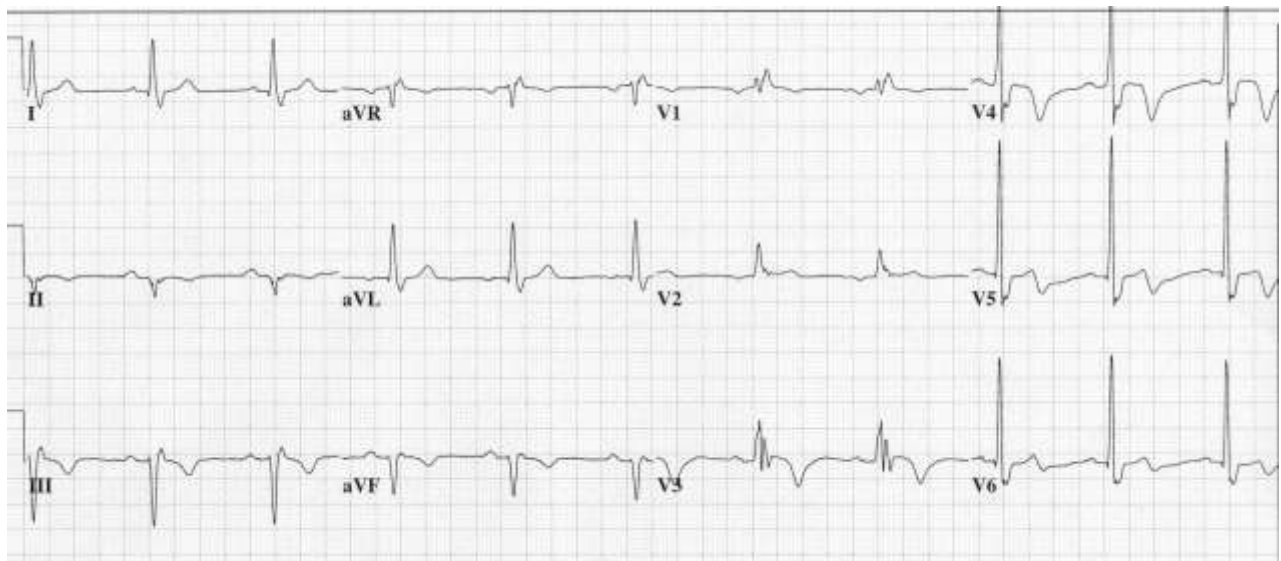
F. Wellen's syndrome

1. Critical stenosis of the LAD.
2. History of angina or atypical symptoms.
3. Normal or minimal cardiac enzyme elevation.
4. Deep, symmetrical T wave inversion in the precordial leads or biphasic T waves.
5. Possible minimal ST segment elevation of < 1 mm
6. No Q waves

ECG #2301

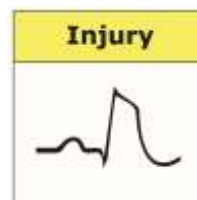
Clinical correlation

- 56-year-old male presented to the emergency department with #8/10 chest pain.
- Nitroglycerine is administered and chest pain resolves.



III. Ischemia, injury and infarction

- A. Ischemia: Imbalance between oxygen supply and demand.
- B. Injury: Occurs when ischemia is not reversed.



- C. Infarction: Tissue dies
- D. Other causes of ST segment and T wave changes
- Aberrant conduction
 - Amyloidosis
 - Bundle branch block
 - Cardiomyopathy
 - Vasospasm
 - Drugs
 - Early repolarization
 - Hemiblock
 - Hypercalcemia
 - Hyperkalemia
 - Hyperventilation
 - Hypocalcemia
 - Hypoglycemia
 - Hypothermia
 - Intracranial hemorrhage
 - Myocardial metastases
 - Myocarditis
 - Paced rhythm
 - Pancreatitis or acute abdomen
 - Pericarditis
 - Physical training
 - Pulmonary embolism
 - Tachycardia
 - Vasospastic angina
 - Ventricular aneurysm
 - Ventricular hypertrophy
 - Ventricular rhythms
 - Wolf-Parkinson-White syndrome

E. ECG changes with current of injury

1. J point elevation may be sharp or diffuse. Both concerning for injury and infarction.
2. ST segment elevation of 1 mm or more above the baseline.
3. Straightening of the ST that slopes up to the peak of the T wave without spending anytime at the baseline.
4. Will typically see ST segment elevation over injured tissue.
5. Will typically see ST segment depression over non-injured tissue.
6. During the very early phase of an acute MI, it is possible to only see T wave changes before ST segment elevation begins. During this early period, very tall, peaked T waves may occur as potassium leaks from the injured tissues. T waves will then invert as the MI evolves.

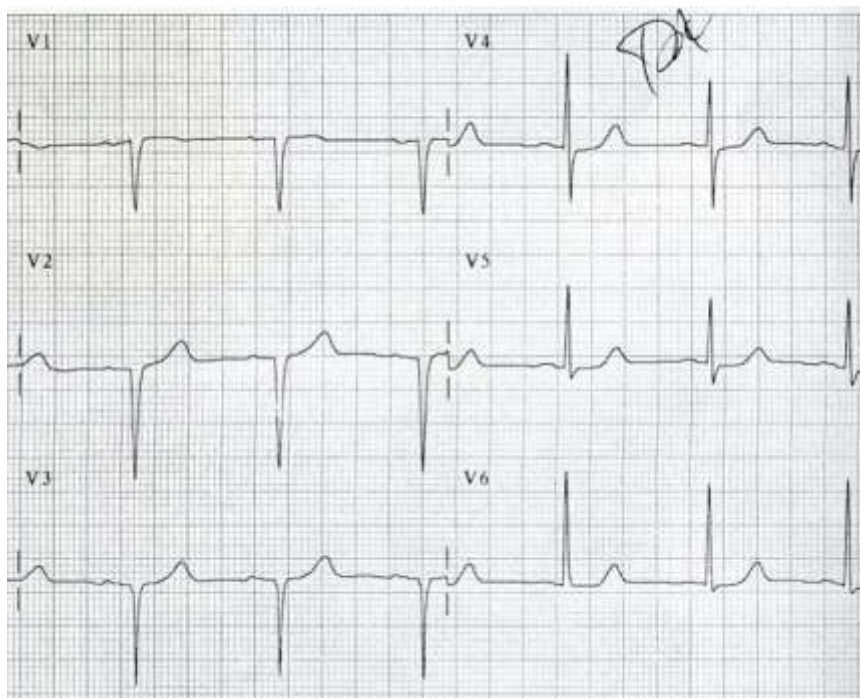


F. ECG changes with infarction

1. Development of new Q waves in the upright leads of I, II, III, aVL, aVF, V5 and V6.
2. Pathological Q waves
 - More than 1/3 the height of the R wave.
 - Development of new Q waves.
 - Deepening of previous Q waves.
3. STEMI (Q wave infarction)
 - Total occlusion.
 - Higher incidence of acute mortality.
 - Early, aggressive treatment is recommended.
4. Non-STEMI (Non-Q wave infarction)
 - Partial occlusion.
 - Higher incidence of long term mortality if not treated.
 - More in the elderly and those with prior MI.
5. Causes of Q waves on the ECG which are not associated with myocardial infarction.
 - Anterior and posterior hemiblock
 - Cardiac amyloidosis
 - Hypertrophic cardiomyopathy
 - Pulmonary embolism
 - Ventricular hypertrophy
 - Ventricular pre-excitation (WPW)



6. Loss of R wave progression in the negative leads V1, V2, V3 and V4 is seen in anterior wall infarction.



IV. ECG changes in the evolution of an acute myocardial infarction

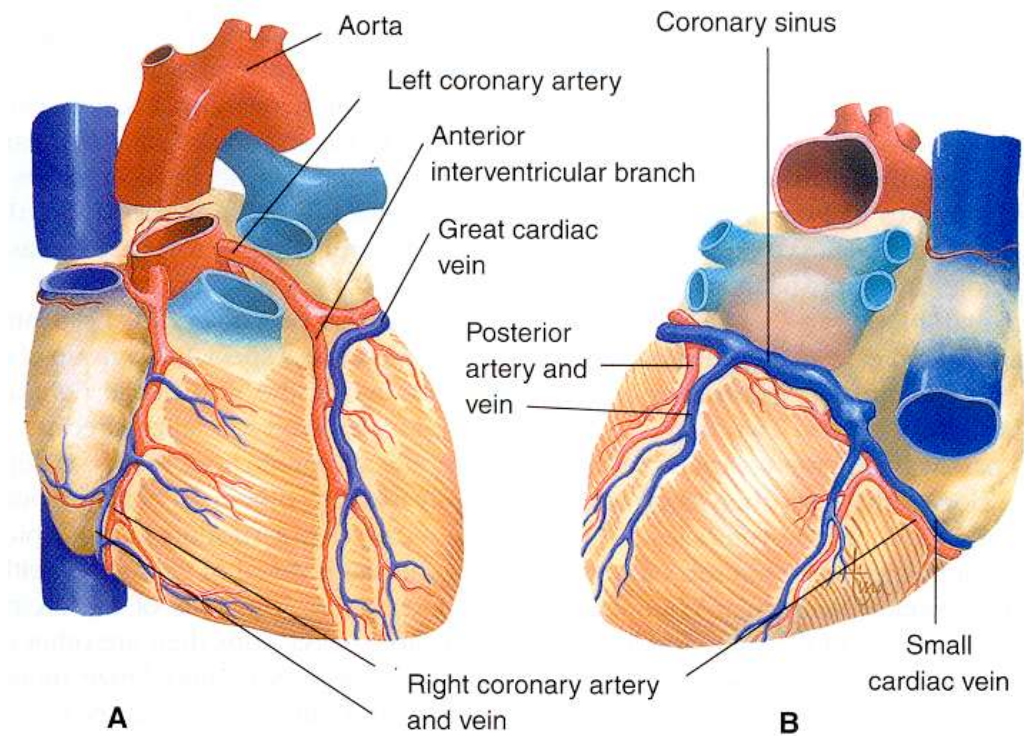
- A. Peaked, widening T waves.
- B. ST segment elevation.
- C. Q waves develop.
- D. ST segment returns to normal with T wave inversion.
- E. Total loss of the R wave.
- F. Return of the T wave to normal.

- G. Evolving myocardial infarction: ST segment coming back down and Q waves have begun to develop. T waves are still inverted.
- H. Old myocardial infarction: Q waves are present with ST segment back to baseline. T waves normal or inverted.

V. Locating the infarction on the 12 lead ECG

- A. Indicative changes
- B. Reciprocal changes
- C. ECG changes associated with AMI

<u>Location</u>	<u>Indicative</u>	<u>Reciprocal</u>	<u>Blood Supply</u>
Anterior	V3 to V4	I, aVL, II, III, aVF	LAD
Anteroseptal	V1 to V4	Right posterior	LAD
Anterolateral	V3 to V6 Can involve I, aVL	II, III, aVF	LAD
Inferior	II, III, aVF	I, aVL, V1 to V4	Right coronary, left circumflex
Lateral	I, aVL, V5, V6	II, III, aVF, V1, V2	Left circumflex
Inferolateral	II, III, aVF, V5, V6 Can involve I, aVL	V1, V2	Left circumflex
Apical	I, II, III, aVF, aVL, V2 to V6		Right coronary
Posterior	"Mirror changes" V1 and V2	V1 to V4	RCA or Left circumflex
Right ventricle	V1 and V2 V3R to V6R, V4R most sensitive		Left circumflex supplies part. Right coronary supplies most.



D. Clinical manifestations

1. Chest pain
2. Pallor
3. Cool, clammy
4. Hypertension
5. Dyspnea
6. Change in heart tones
7. Heart failure
8. Fatigue
9. Weakness
10. Nausea and vomiting
11. Decreased level of consciousness
12. Temperature elevation

E. Treatment options

1. M _____

O _____

N _____

A _____

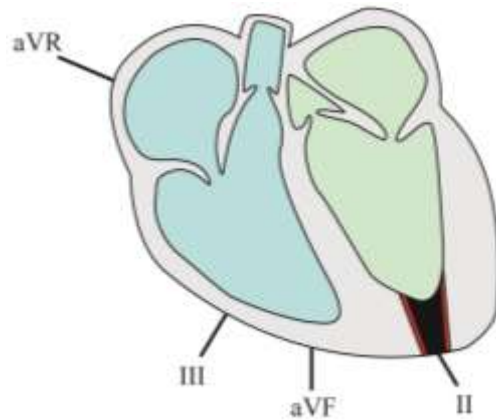
B _____

2. Antiplatelet agents such as eptifibatide (Integrilin) or abciximab (ReoPro).
3. Anticoagulants such as enoxaparin (Lovenox).
4. Thrombolytics in some situations.
5. Definitive therapy such as percutaneous coronary angioplasty or coronary bypass surgery.
6. Long term drug therapy is usually balanced therapy.
 - Beta blockers
 - ACE inhibitors or angiotensin receptor blockers
 - Nitrates
 - Anticoagulants and/or antiplatelet agents
 - HMG CoA reductase inhibitors
 - Calcium channel blockers in some patients

VI. Inferior wall myocardial infarction

A. Pathology and ECG changes

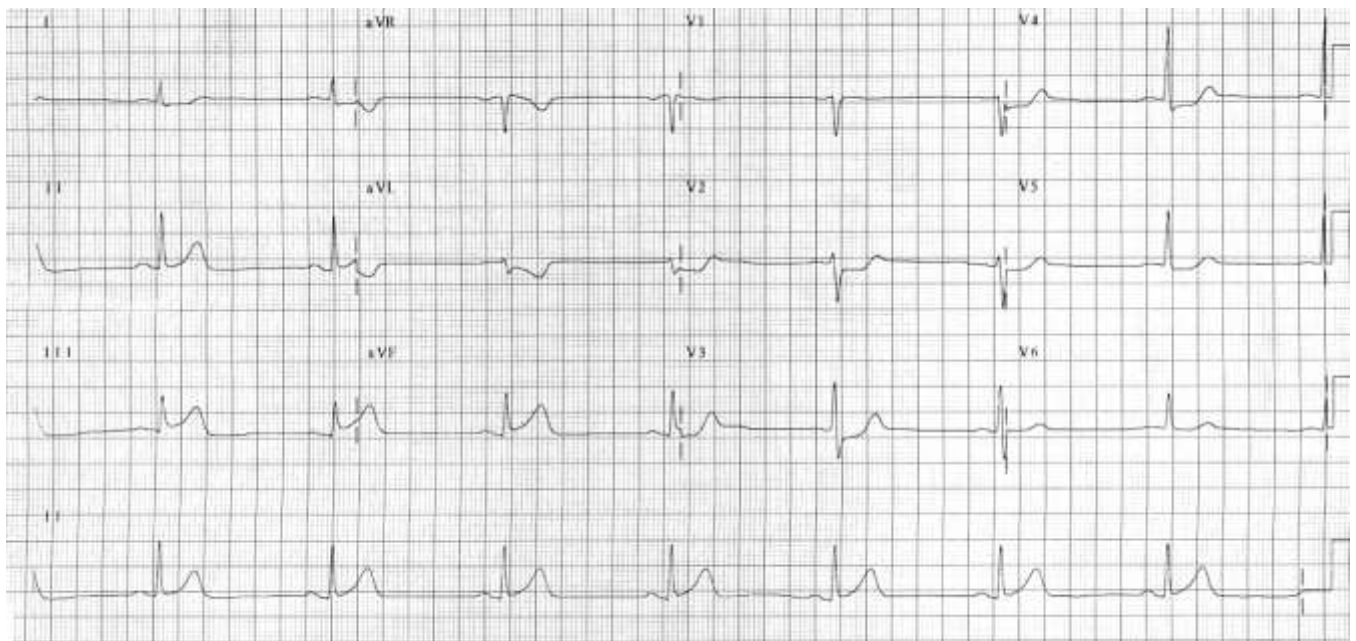
1. Occluded right coronary artery. Circumflex occlusion in those with left dominant circulation
2. Indicative changes seen in II, III, and aVF.
3. Reciprocal changes seen in the lateral or V leads.
4. Lead II can have a Q wave present under normal situations.
5. Up to 40-45% of acute inferior wall MIs may also involve the right ventricular muscle mass.



EC #2302

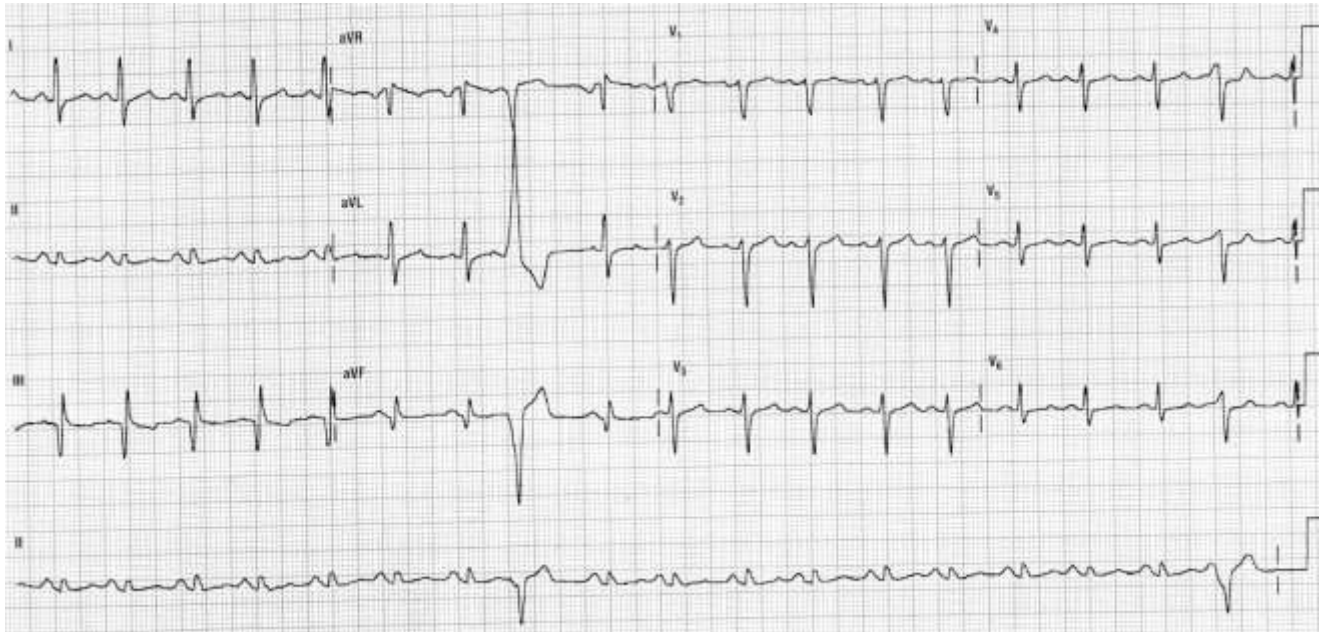
Clinical correlation

- 56-year-old presenting to the ED having chest pain for the past 20 minutes while at work. Complains of feeling lightheaded and is nauseated. BP 160/100.
- PR=0.20 QRS=0.08



Clinical correlation

- Patient presents to the ED after having left anterior chest pain a week ago.
- He has been more fatigued the past week and presents to the ED. Crackles are heard bilaterally although he denies dyspnea. He is pain free at the present time.
- PR=0.16 QRS=0.10



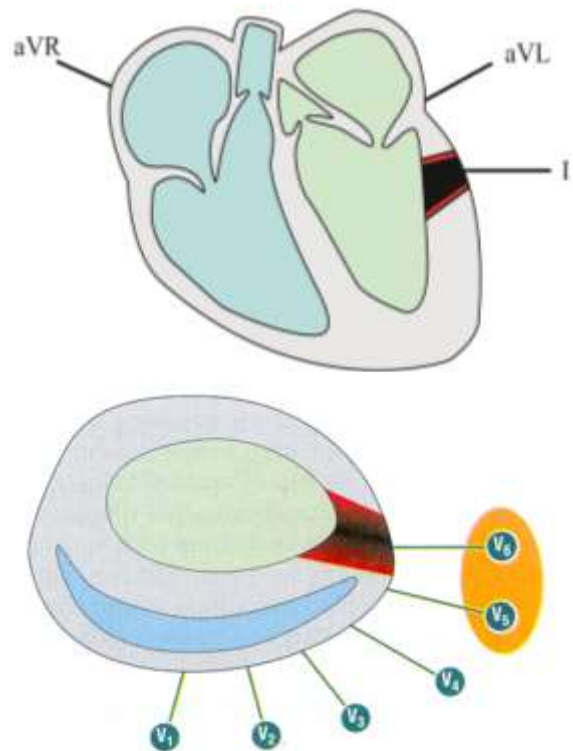
B. Clinical implications

1. Sinus node and atrial dysrhythmias may develop.
2. Various AV blocks, especially second degree AV block, type I are common and usually are transient and resolve without intervention.
3. Hypotension and bradycardia are common due to increased parasympathetic activity.
4. Hiccups, nausea and vomiting are also frequent due to parasympathetic stimulation.

VII. Lateral wall myocardial infarction

A. Pathology and ECG changes

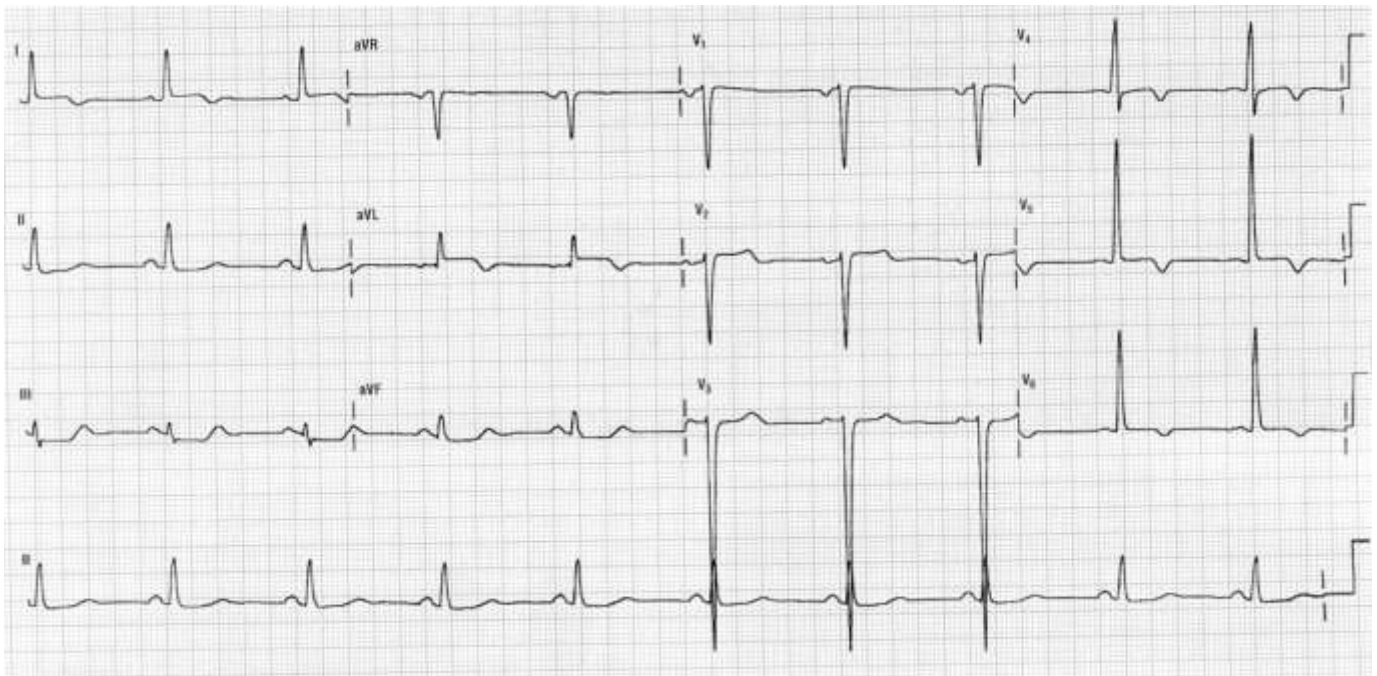
1. Occluded circumflex artery.
2. Indicative changes seen in I and aVL. V5 and V6 may also have changes.
3. Reciprocal changes in the inferior or anterior leads.
4. May occur alone, but usually occurs in conjunction with an anterior or inferior wall MI.



EC #2304

Clinical correlation

- 72-year-old female complaining of vague chest discomfort.
- She has a 20-year history of type II diabetes mellitus.
- PR=0.18 QRS=0.10



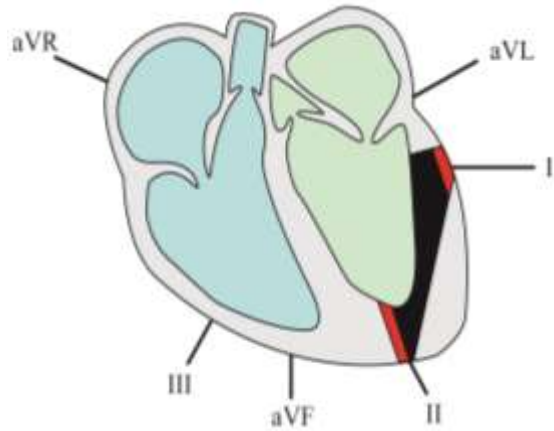
B. Clinical implications

1. Usually occurs with other walls of the heart and then significant myocardial damage can occur.
2. Because the left circumflex artery also supplies the AV node, Bundle of His and the papillary muscles in 10% of the population, occlusion may result in conduction abnormalities and mitral valve dysfunction.

VIII. Inferolateral wall myocardial infarction

A. Pathology and ECG changes

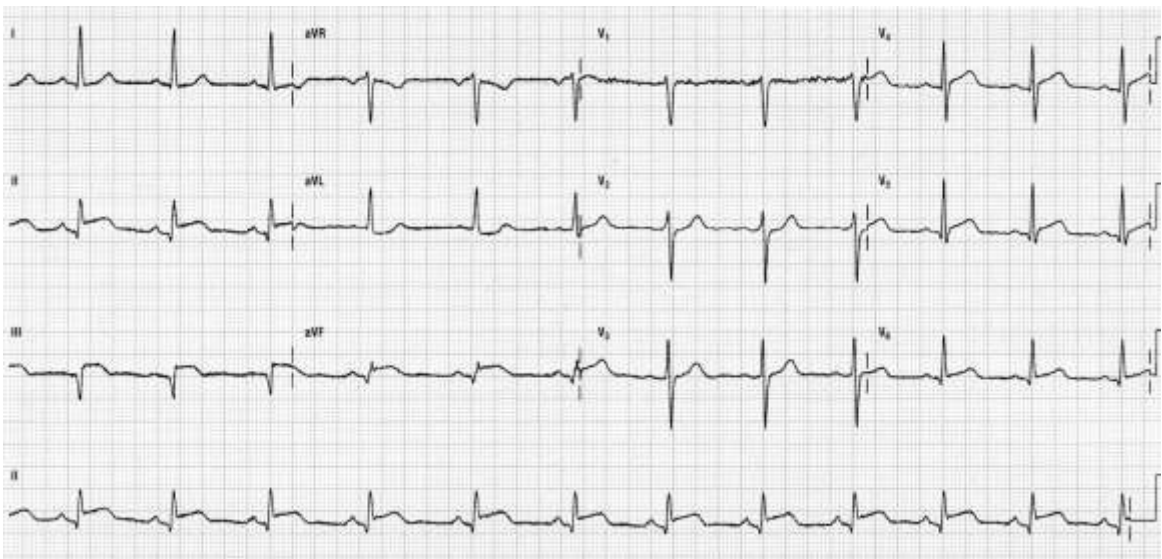
1. Occlusion of the left circumflex.
2. Changes in II, III, aVF, V5 and V6, and I and aVL if high lateral involvement.
3. ST changes can also occur in V2 to V4 as well depending on how far the infarction extends.



EC #2305

Clinical correlation

- Patient is complaining of vague chest discomfort and shortness of breath. He had not been feeling well for the past few hours but wanted to wait until his wife got home before he went to the hospital.
- On assessment, an S4 gallop is auscultated.
- PR=0.18 QRS=0.10



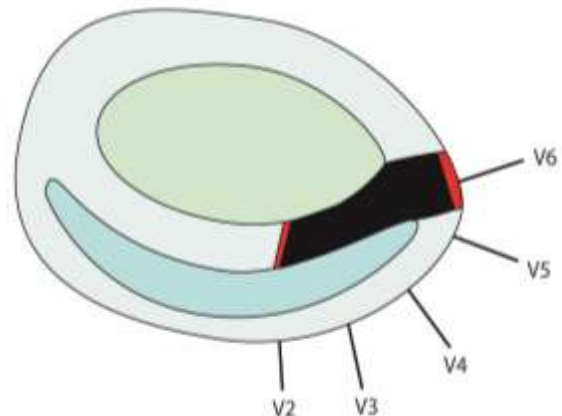
B. Clinical implications

1. Significant muscle damage.
2. Frequently occurring MI.
3. Could present with some heart blocks such as second degree type I or bradycardia.
4. Sinus dysrhythmia common in this type of MI.

IX. Anterolateral wall myocardial infarction

A. Pathology and ECG changes

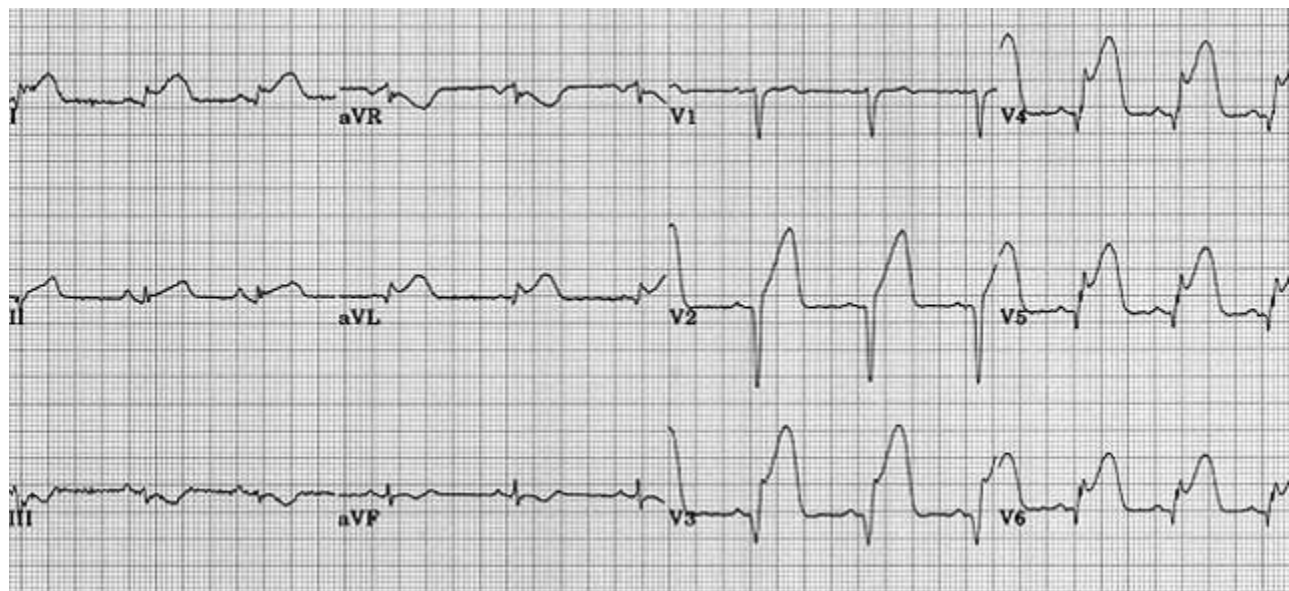
1. Occlusion of the left anterior descending.
2. Indicative changes seen in V3-V6. Lead I and aVL if high lateral.
3. Reciprocal changes seen in the lateral and inferior leads.
4. May be an extensive anterior MI if the lateral and/or septal walls are both involved.



ECG #2306

Clinical correlation

- 65-year-old having severe crushing chest pain for the past 45 minutes.
- PR=0.16 QRS=0.08



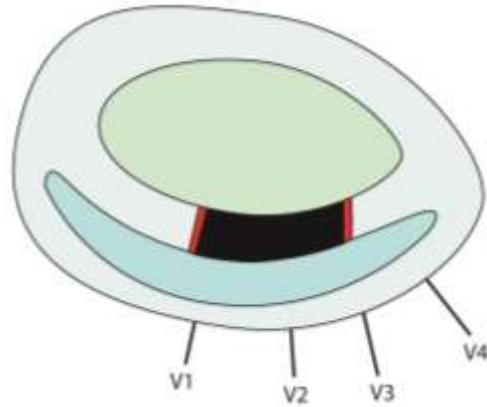
B. Clinical implications

1. Anterior wall rarely occurs alone. When lateral or septal involvement with the anterior wall, complications may include severe left ventricular dysfunction, resulting in heart failure and cardiogenic shock.
2. Fascicular blocks or AV blocks may be present.
3. Sinus tachycardia is a common finding in the setting of acute anterior MI.

X. Anteroseptal wall MI

A. Pathology and ECG changes

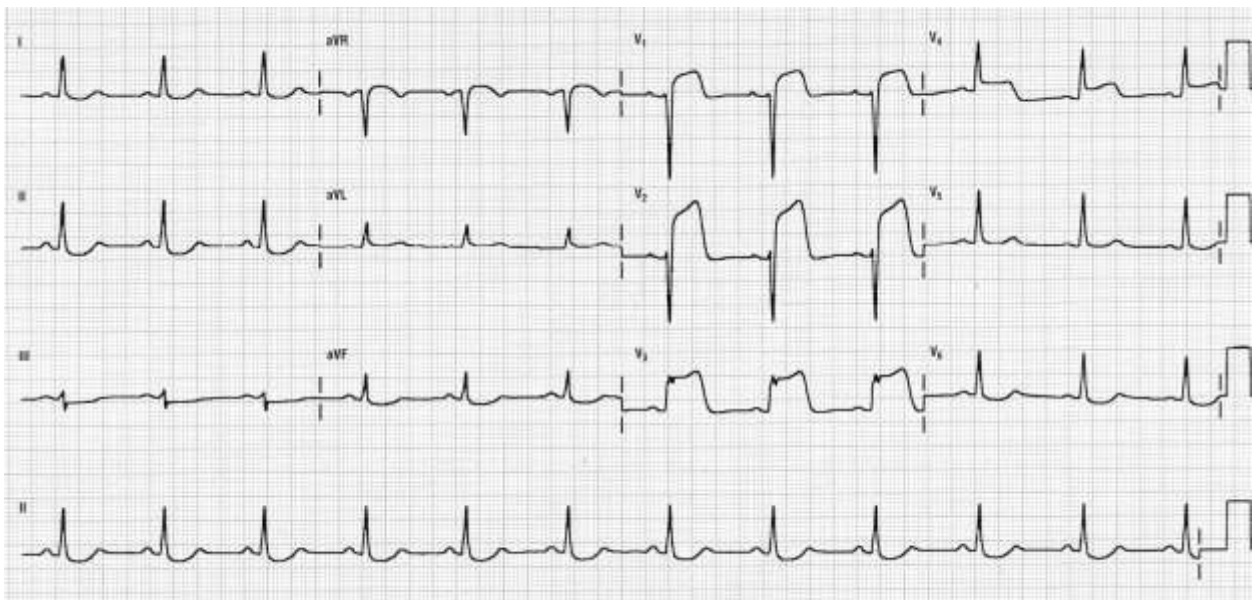
1. Occlusion of the LAD.
2. ST segment elevation in V1 through V4.
3. Reciprocal changes in the right posterior leads.
4. If changes in the limb leads seen, there is involvement of other walls of the heart.



ECG #2307

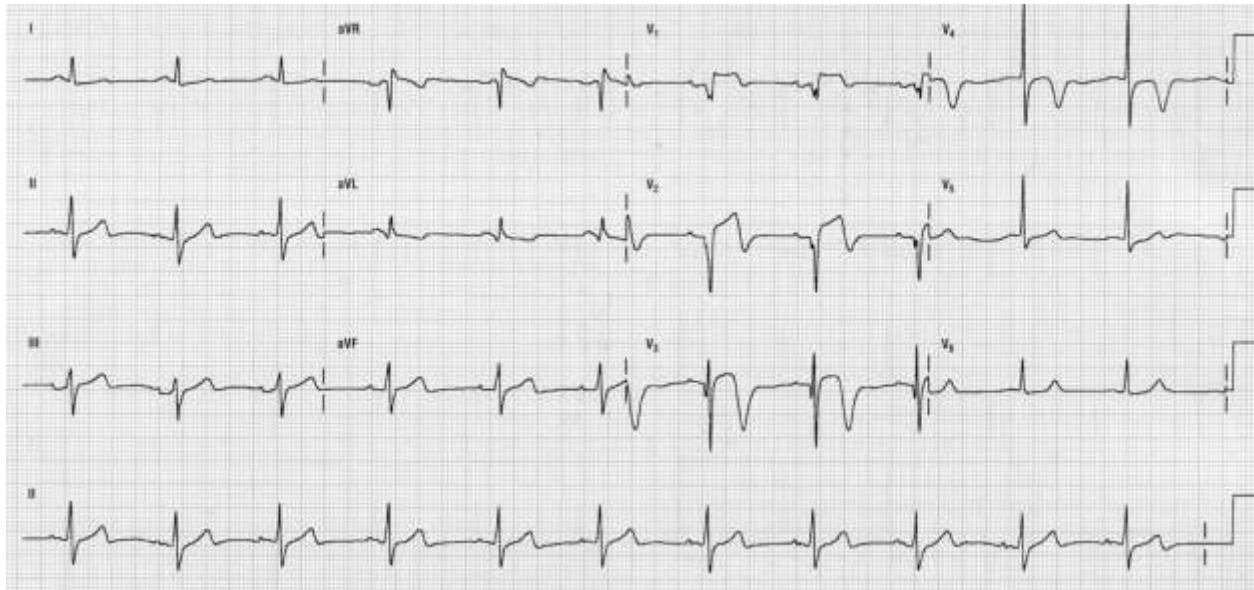
Clinical correlation

- Patient presents to the emergency room having chest pain.
- He feels like his heart rate is slightly irregular and he has been “skipping beats”.
- PR=0.18 QRS=0.08



Clinical correlation

- Patient presents to the ED with a history of intermittent chest pain for the past few hours which would lessen with rest.
- She is pale, anxious, cool and clammy. BP 105/60. Crackles are heard bilaterally. An S3 gallop is present.
- PR=0.18 QRS=0.10



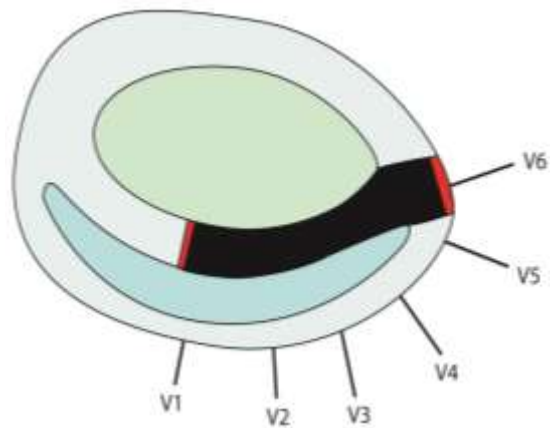
B. Clinical implications

1. Common type of MI.
2. Hemodynamic instability and cardiogenic shock.
3. May see fascicular blocks and bundle branch blocks.

XI. Anteroseptal wall MI with lateral extension

A. Pathology and ECG changes

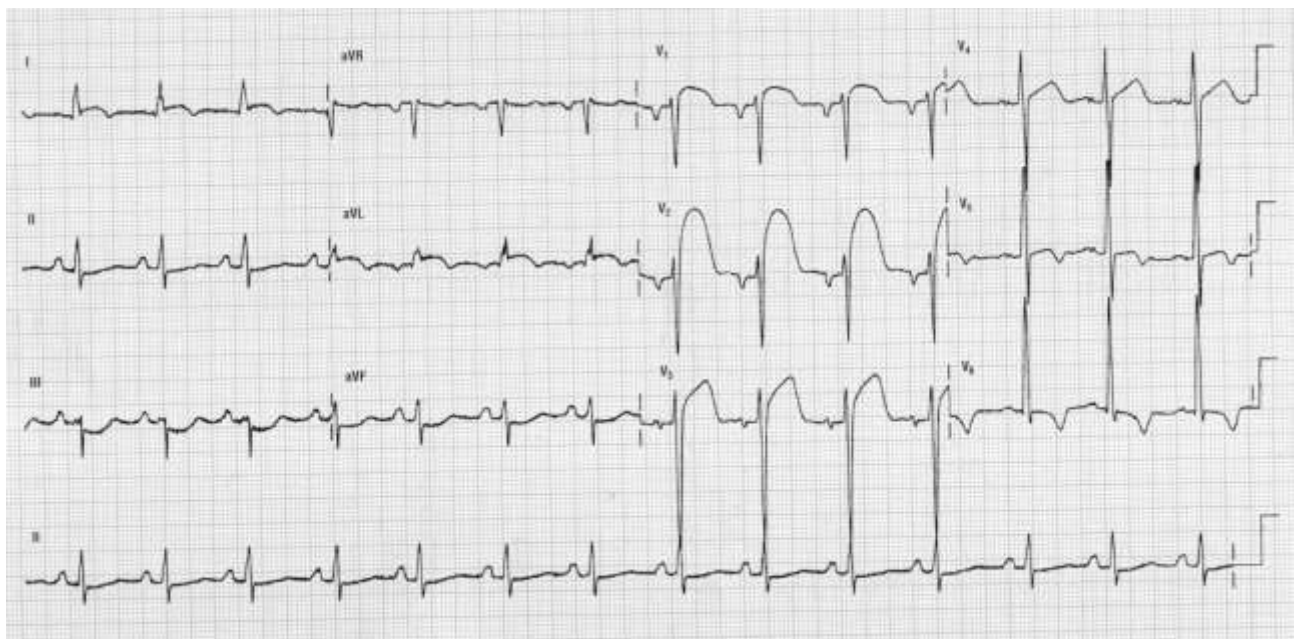
1. Caused by obstruction of the proximal left main or the proximal LAD.
2. ST segment elevation in V1 through V6. Can also involve leads I and aVL.
3. Reciprocal changes in II, III, and aVF.



ECG #2309

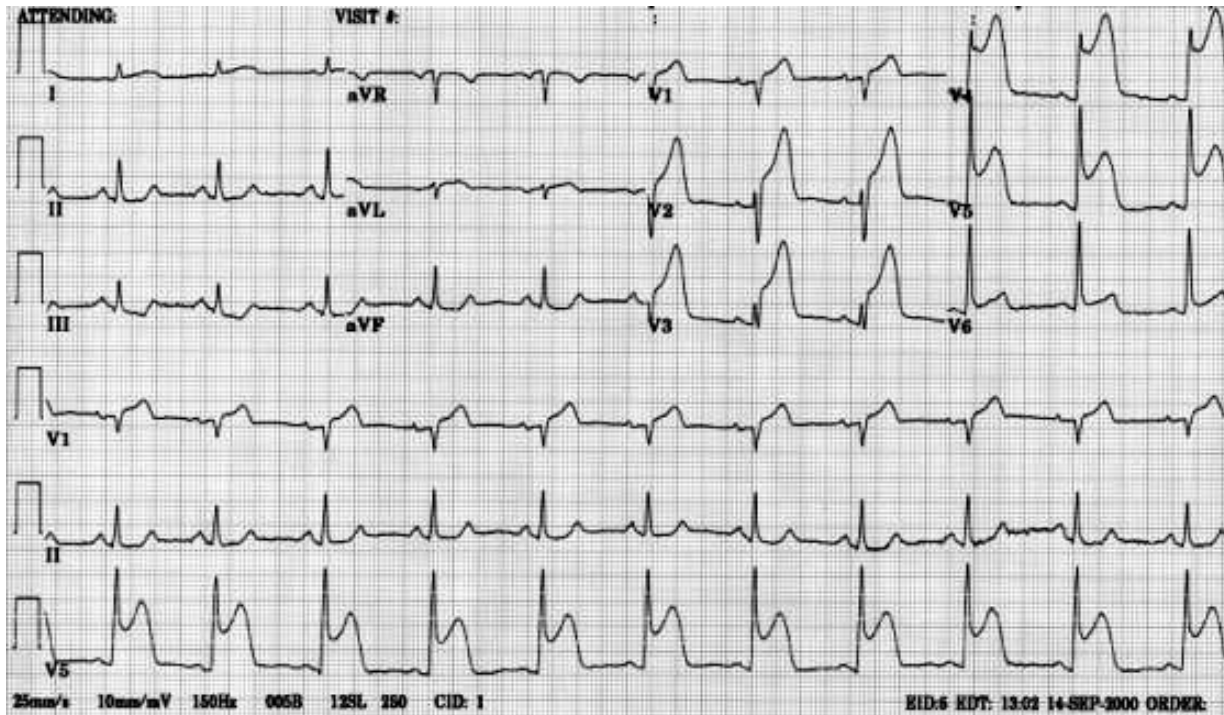
Clinical correlation

- Patient admitted two days ago with pneumonia.
- Calls out complaining of a sudden onset of chest pain.
- PR=0.18 QRS=0.08



Clinical correlation

- 64-year-old female presenting with crushing chest pain. She is pale, anxious, and cool and clammy. BP is 160/105. Pulse is 75 per minute.
- Past history of COPD and diabetes mellitus, type II. She has a family history of CAD. Her mother died of sudden death at the age of 50 years.
- PR=0.20 QRS=0.10



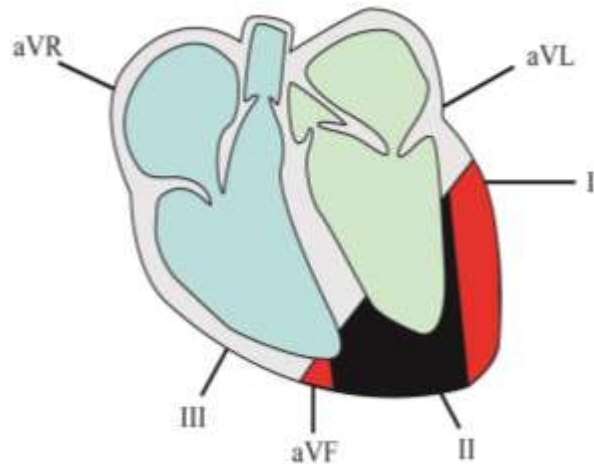
B. Clinical implications

1. Common infarction.
2. Large amount of myocardium is damaged.
3. Early, aggressive intervention is vital.
4. Fairly common and represent extensive damage.
5. May also be called “extensive anterior MI.
6. Important to do serial ECGs to evaluate for any new blocks or extensions of the infarction.

XII. Apical wall myocardial infarction

A. Pathology and ECG changes

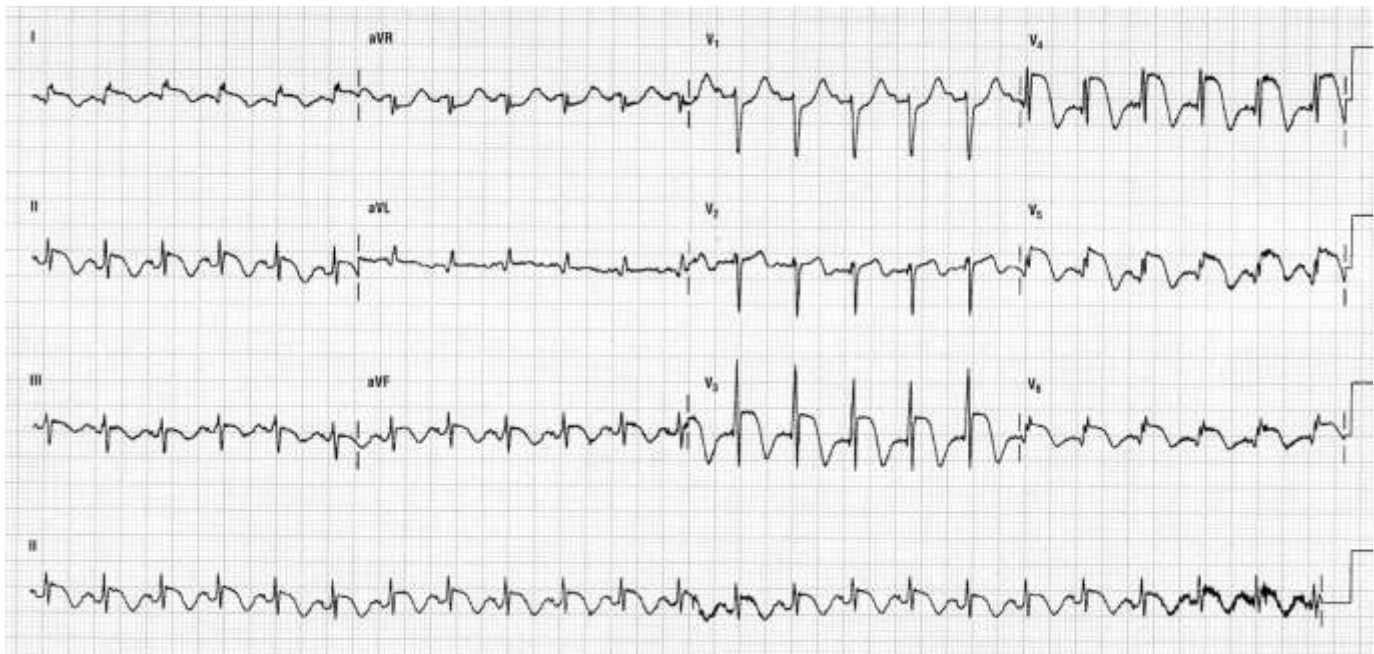
1. Occlusion of the right coronary artery in a client who is right dominant.
2. Changes in I, II, III, aVF, aVL, and V2 to V6.



ECG #2311

Clinical correlation

- 46-year-old male with complaint of severe chest pain for the past 20 minutes while at work at a factory.
- EMS was called. ECG was performed in the squad and transmitted to the hospital.
- Patient is overweight and is a 1-ppd smoker. He has a family history of CAD.
- PR=0.18 QRS=0.08



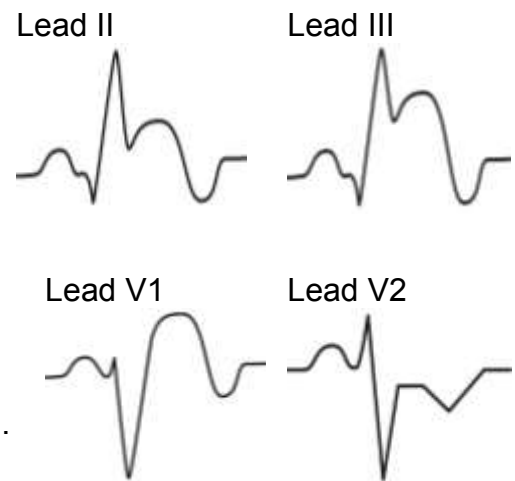
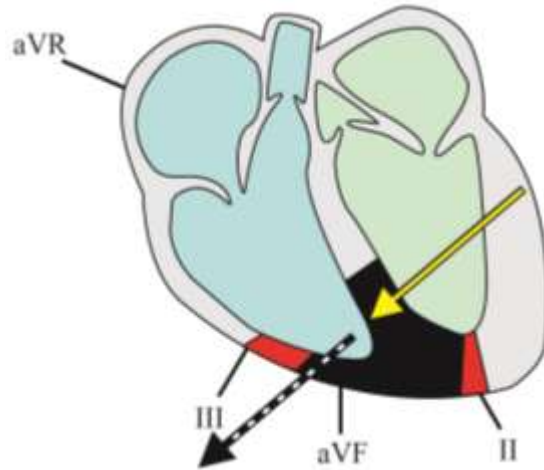
B. Clinical implications

1. Large amount of muscle damage with an apical MI. Often unstable with hemodynamic compromise.
2. Treatment of choice is revascularization due to a large amount of muscle at risk.
3. Can be mistaken for pericarditis.

XIII. Right ventricular myocardial infarction

A. Pathology and ECG changes

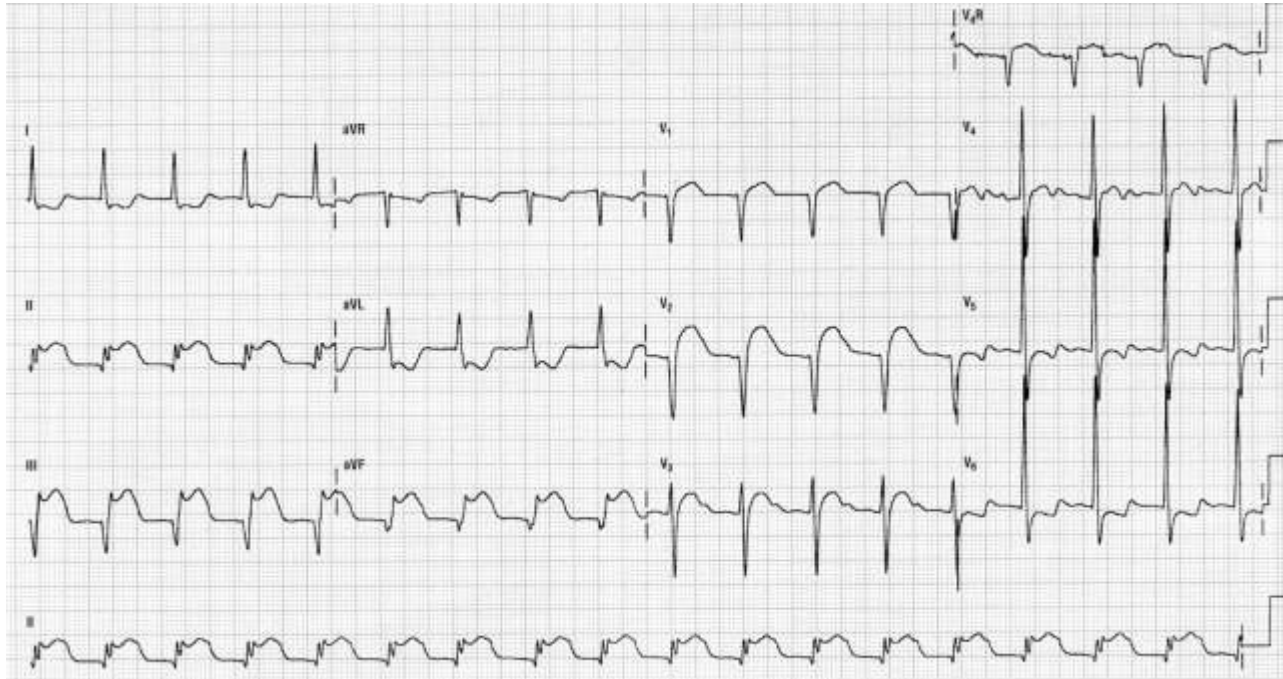
1. May occur in 40-45% of inferior wall MI.
2. If isolated RV MI, occlusion of the left circumflex.
3. If inferior with RV, occlusion of the proximal RCA.
4. May see ST segment elevation in conjunction with an inferior MI. Suspect right ventricular involvement.
5. ST segment elevation will be higher in III than in II in right ventricular.
6. May see discordance between ST segment changes in V1 and V2.
 - ST segment elevation in V1.
 - ST segment depression in V2.
7. May see ST segment elevation in V1-V2.
8. Do right precordial chest leads.
 - See changes in V3R through V6R.
 - V4R tends to be the most sensitive and one in which changes are most often seen.



ECG #2312

Clinical correlation

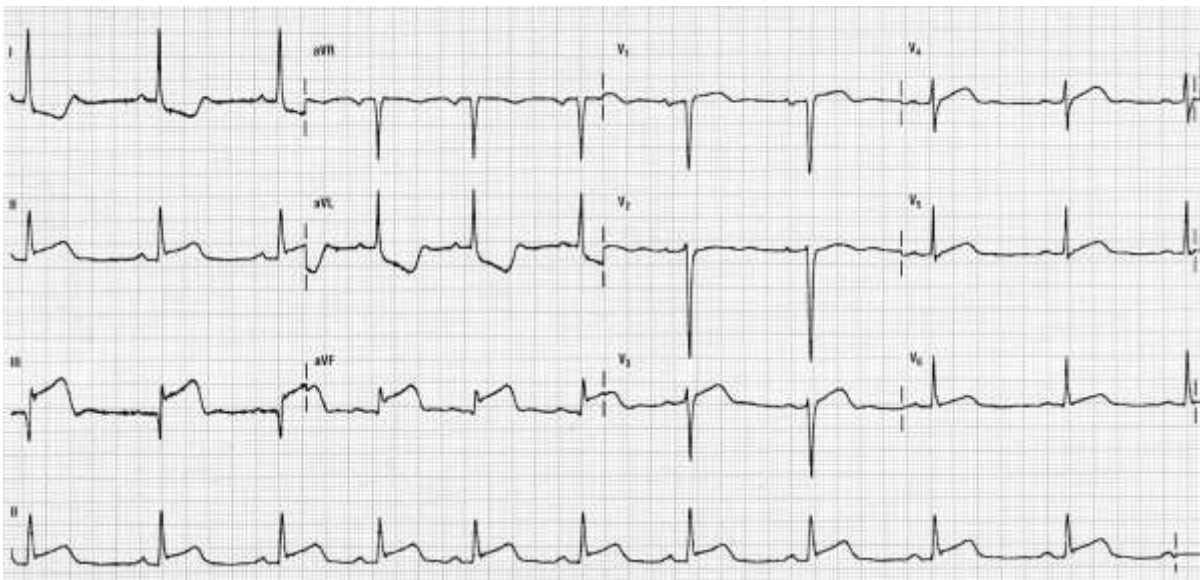
- 52-year-old male who presents to the ED via the EMS having chest pain.
- After the initial ECG, the paramedic gave NTG sublingual and the blood pressure fell to 80/40. HR is 100 and strong. He is pale, cool and clammy.
- PR=0.32 QRS=0.10



ECG #2313

Clinical correlation

- Patient is in recovery room after a TURP and begins to have chest pain.
- Pulse is slightly irregular. BP 152/94.
- PR=0.24 QRS=0.08



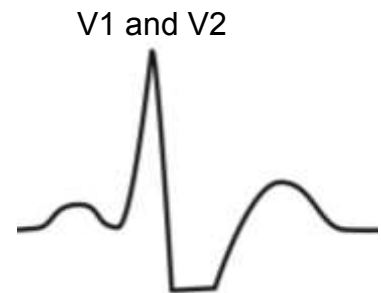
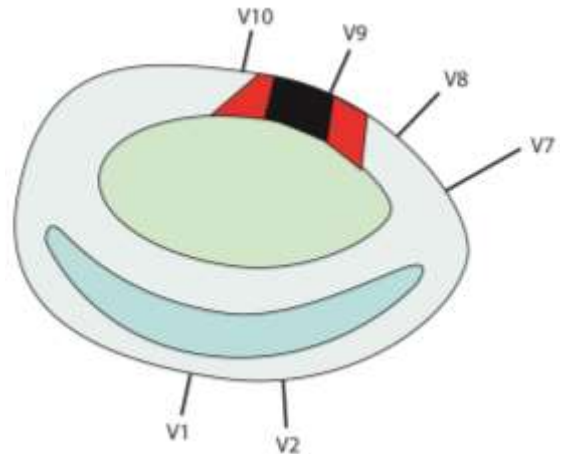
B. Clinical implications

1. Large amount of muscle mass.
2. Treatment protocols are different with acute right ventricular damage. Administration of NTG for chest pain will result in decrease preload and will compromise coronary filling and cardiac output.

XIV. Posterior wall MI

A. Pathology and ECG changes

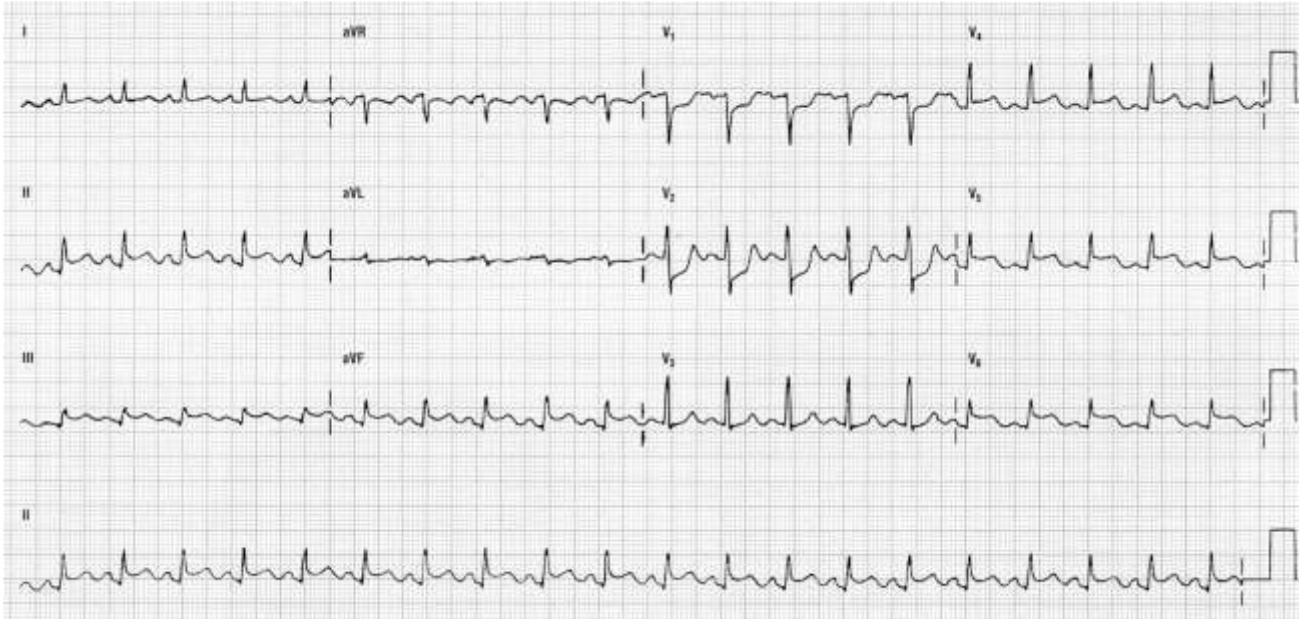
1. Occlusion of the right coronary or circumflex in left dominant people.
2. Usually occurs in conjunction with an inferior MI.
3. Less obvious on the 12 lead ECG. No real "indicative changes".
4. "Mirror changes" will be present. V1 and V2 are used to recognize the infarction.
 - Taller than normal R wave.
 - ST segment depression.
 - Taller than normal, and upright, T wave in V1 and V2.



EC #2314

Clinical correlation

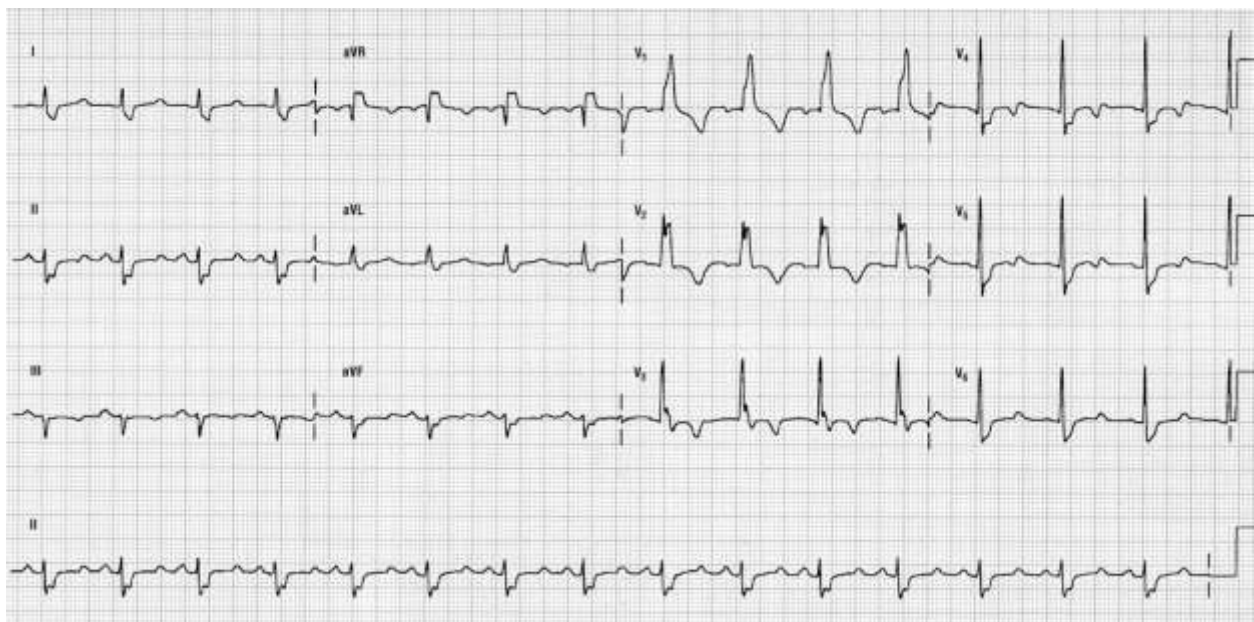
- Patient presents to the ED having epigastric and back pain. Also complains of nausea.
- He is pale, cool and extremely anxious. BP 174/90. Pulse 115/min.
- PR=0.18 QRS=0.08



ECG #2704

Clinical correlation

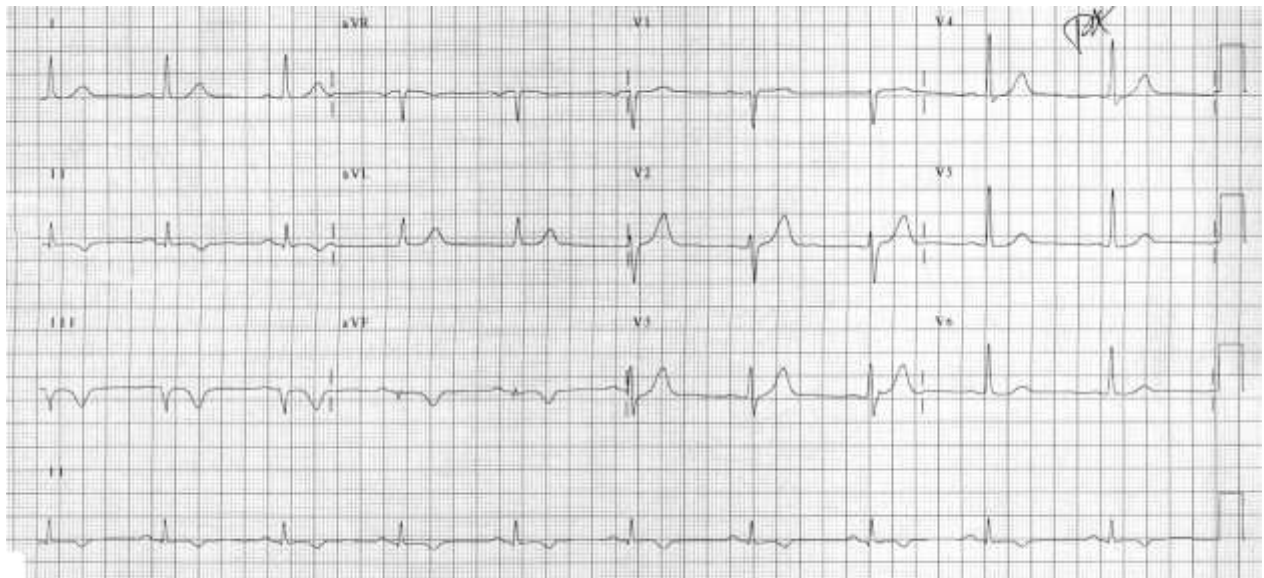
- 68-year-old female admitted for a cholecystectomy.
- The ECG was completed as part of her pre-admission testing.
- PR=0.18 QRS=0.14



ECG #2705

Clinical correlation

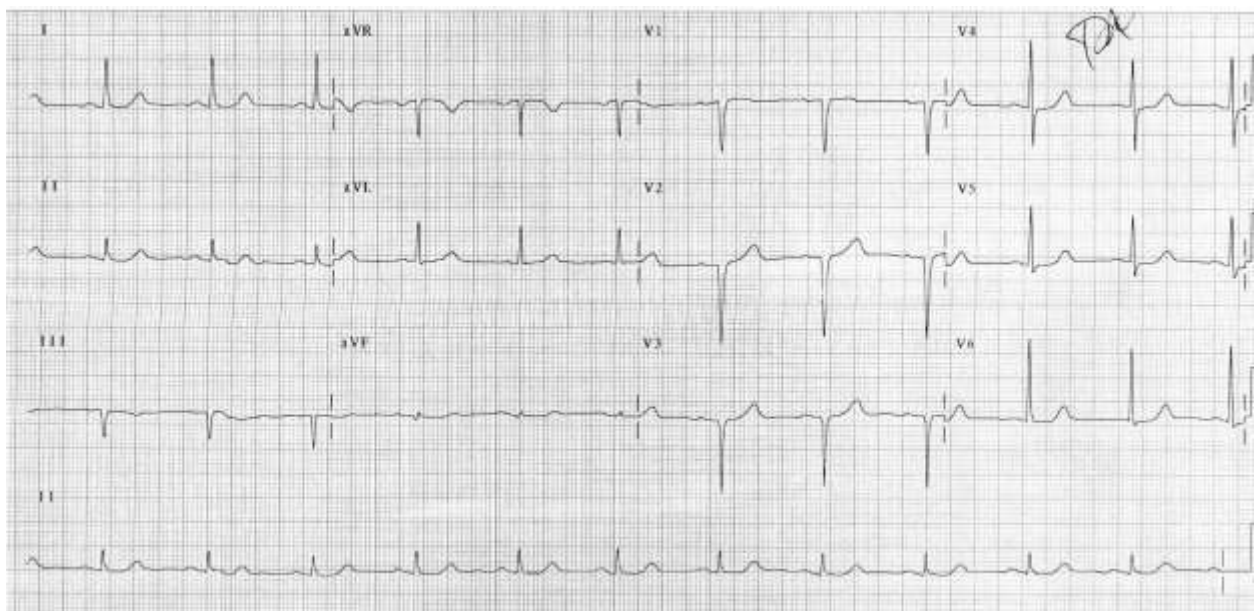
- 57-year-old male presents with complaints of chest pain for the past 12 hours.
- ECG one month ago was normal.
- PR=0.18 QRS=0.08



ECG #2706

Clinical correlation

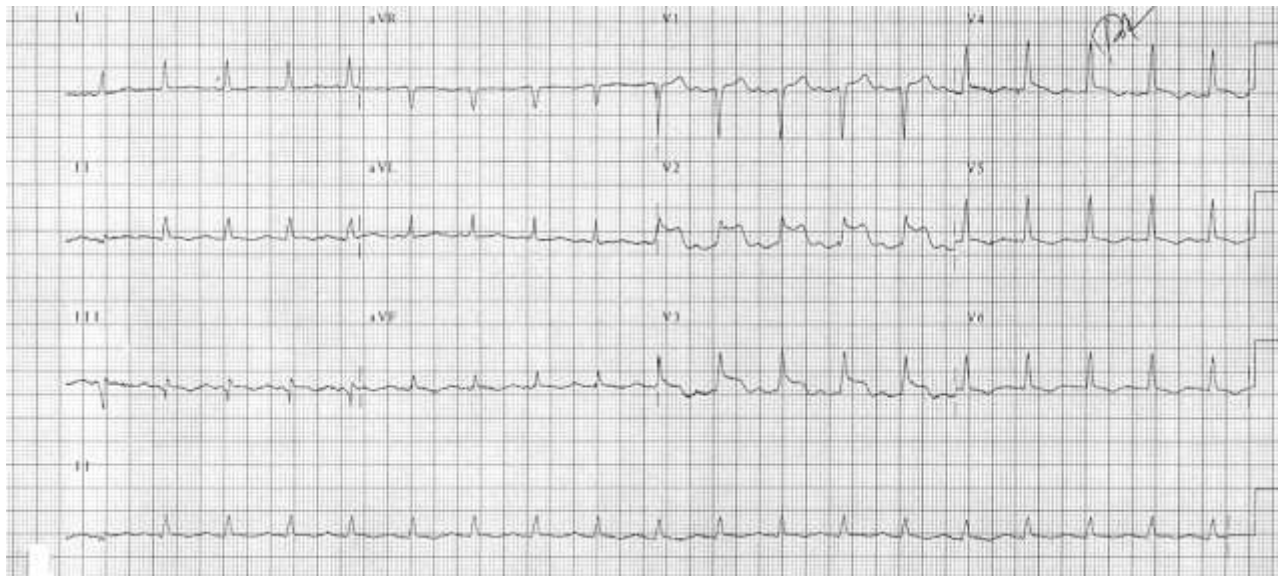
- 78-year-old female admitted with vaginal bleeding.
- ECG completed prior to a hysterectomy.
- PR=0.18 QRS=0.08



ECG #2707

Clinical correlation

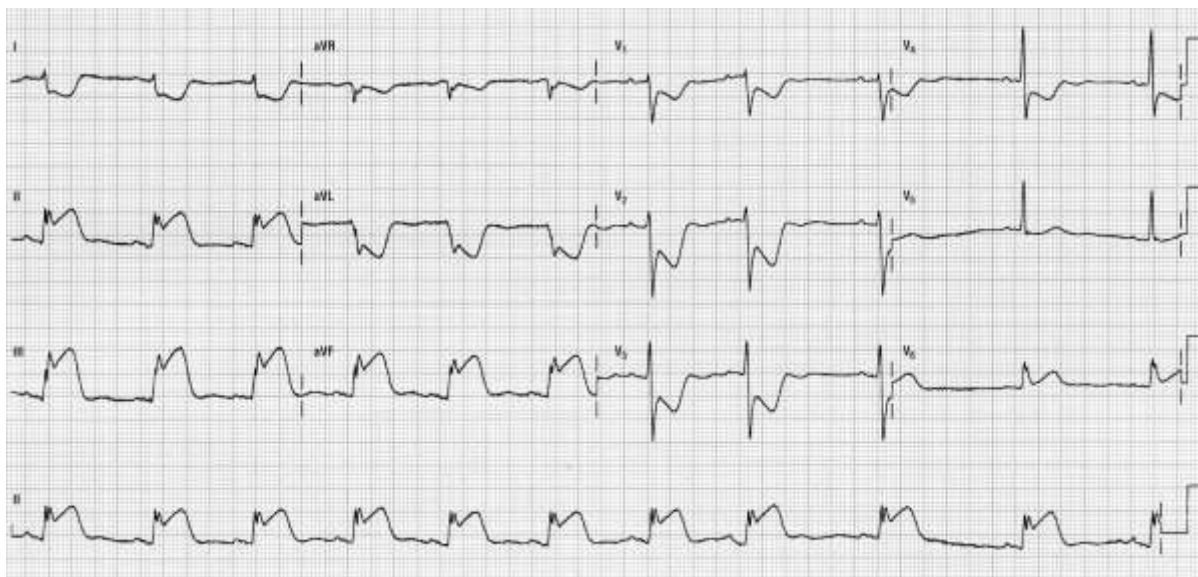
- 85-year-old female presenting with complaints of chest pain.
- She is pale and anxious. HR is 114 and regular. BP slightly elevated.
- PR=0.18 QRS=0.08



ECG #2708

Clinical correlation

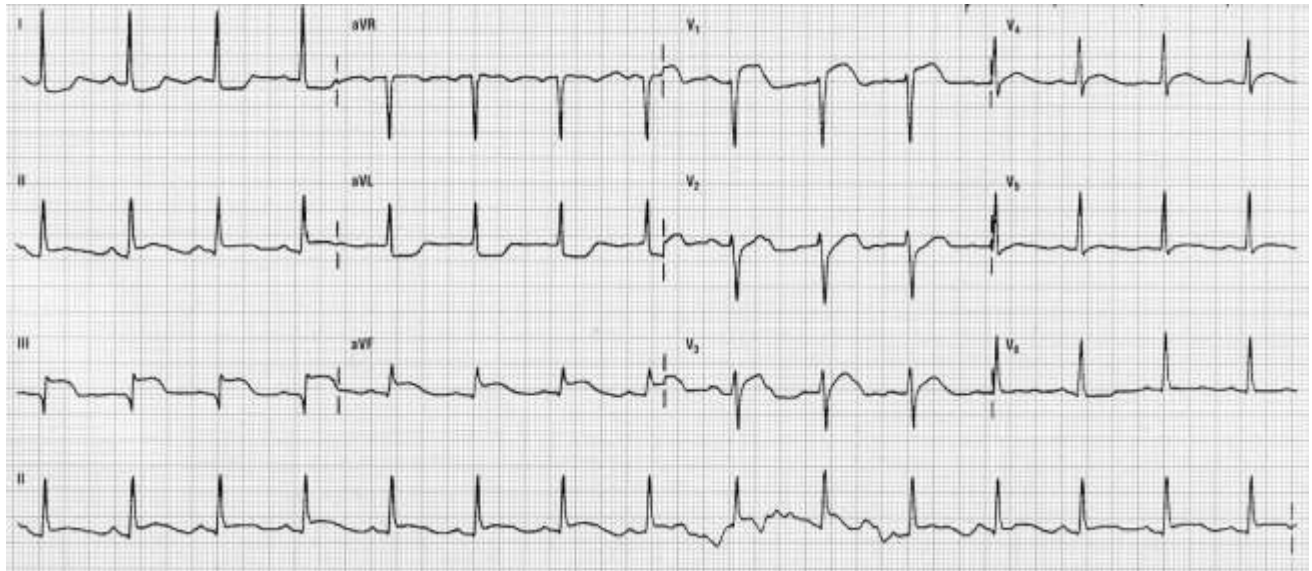
- 52-year-old male presenting via rescue squad with a history of crushing chest pain for the past 30 minutes.
- He is pale, diaphoretic and extremely anxious. NTG sublingual was given in the EMS and hypotension resulted. Fluid bolus given with improvement of the blood pressure.
- PR=0.18 QRS=0.10



ECG #2709

Clinical correlation

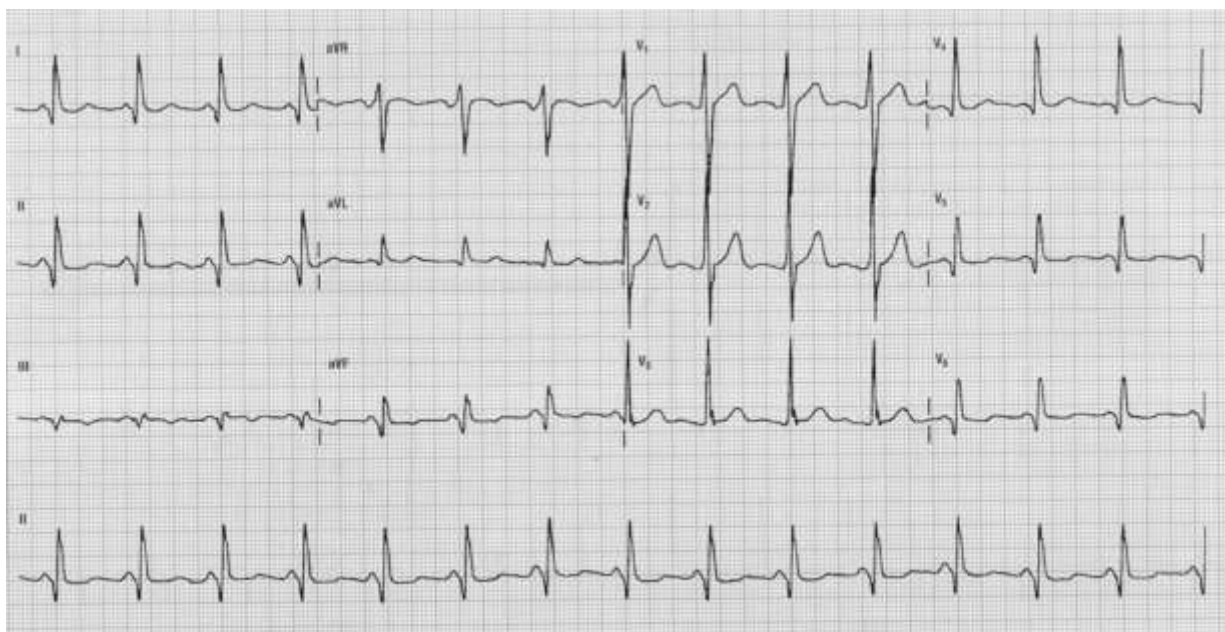
- 48-year-old male admitted with acute calcifying pancreatitis. He has a long history of alcohol abuse and has been admitted multiple times for pancreatitis.
- On the second day of his hospitalization, he complains of chest discomfort. He has no previous cardiac history. HR is 88 per minute.
- PR=0.18 QRS=0.08



ECG #2710

Clinical correlation

- 77-year-old female with a history of CAD and an MI two years ago.
- She is currently in the hospital with pneumonia.
- PR=0.16 QRS=0.10



Chamber Hypertrophy

Objective

- Verbalize the ability to recognize ECG changes associated with chamber hypertrophy.

I. Atrial enlargement

A. Normal P waves

1. Maximum of 0.11 seconds wide.
2. Maximum height of 2.5 mm.
3. Upright in II, III, V4-6.
4. May be diphasic or inverted in V1.

B. Left atrial enlargement (P-mitrale)

1. Causes

- Mitral valve disease
- Hypertension
- Left heart failure

2. P wave morphology in left atrial enlargement

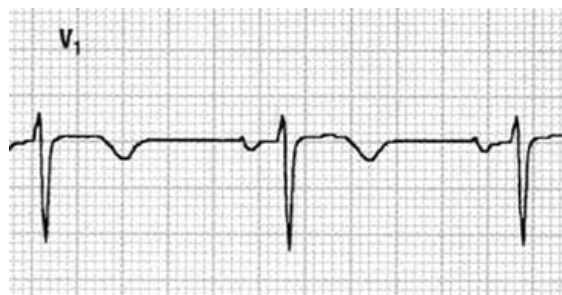
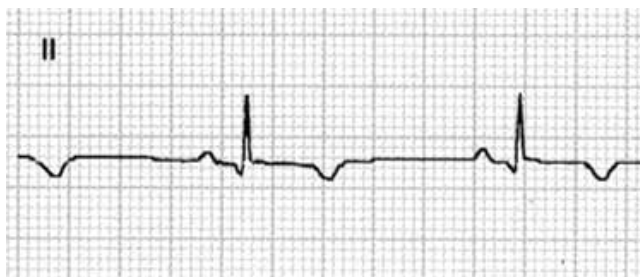
- Wider than 0.11 seconds.
- Often notched in I, II, aVL, V4-6.
- Biphasic in V1 with a deeper negative component.

Lead II

Notched in LAE
Tall and peaked in RAE
Wide and tall in bi-atrial

Lead V1

↑ then deep ↓ in LAE
Tall and peaked in RAE
(May be inverted)
Biphasic in bi-atrial



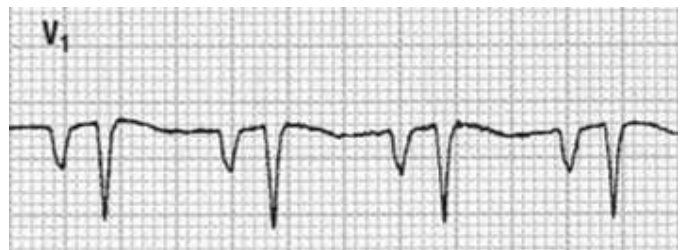
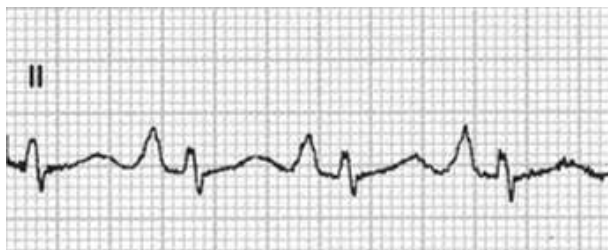
C. Right atrial enlargement (P-pulmonale)

1. Causes

- Pulmonary hypertension
- Pulmonic valve disease
- Tricuspid valve disease

2. P wave morphology in right atrial enlargement

- Taller than 2.5 mm and peaked.
- See best in leads II and III.
- Can look for tall and peaked in V1.

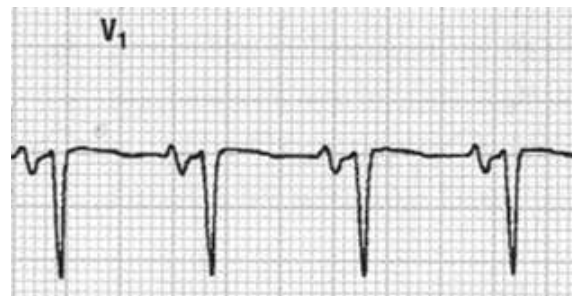
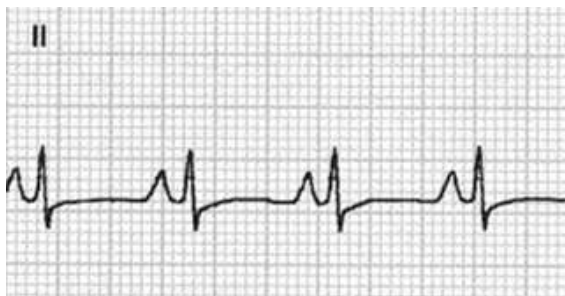


D. Batrial enlargement

1. Both atria are enlarged.

2. P wave morphology.

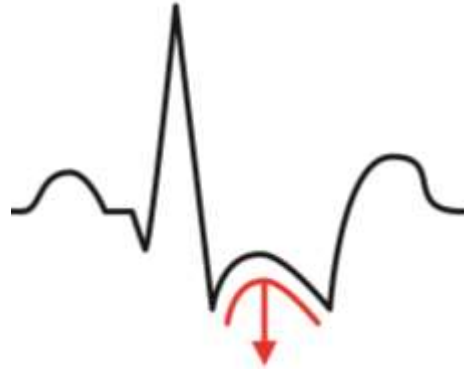
- Look for evidence of both types of P wave configurations on the ECG.
- Product of width times height greater than or equal to 0.3 seconds.
- Wider than 0.11 seconds, taller than 2.5 mm in lead II.
- Biphasic in V1.



II. Ventricular hypertrophy

A. ST and T wave changes in hypertrophy

1. Often called “strain” pattern.
2. Reflects repolarization abnormalities due to the increased muscle mass.
3. ST segment depression with down sloping and inverted T waves.
4. Changes are seen in inferior leads with hypertrophy.
 - When left ventricular hypertrophy, will also see strain in the lateral leads.
 - When right ventricular hypertrophy, will also see strain in the septal leads.



B. Left ventricular hypertrophy

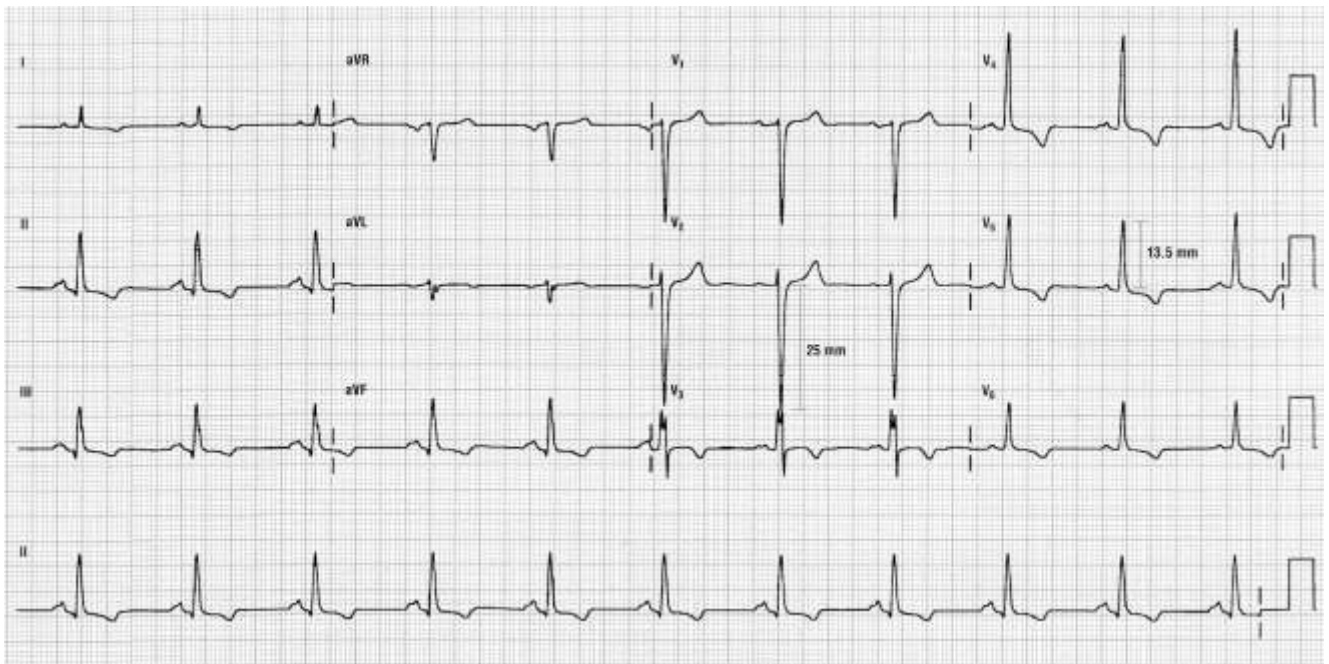
1. Causes

- Increased volume
- Diastolic overload
- High preload
- High systolic pressures and afterload

2. ECG changes

- QRS changes in leads V1 and V2: Find the deepest S wave.
- QRS changes in leads V5 and V6: Find the tallest R wave.
- Strain pattern in inferior and lateral leads.

ECG #2405



C. Right ventricular hypertrophy

1. Causes

- Pulmonary disease
- Left to right shunt

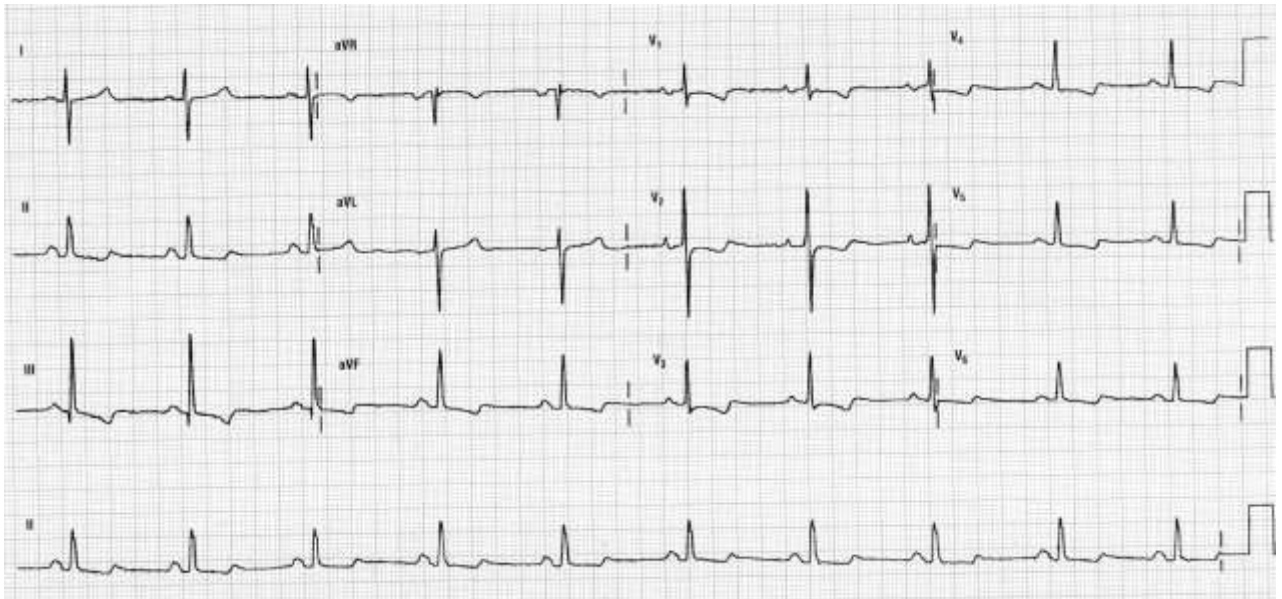
2. Depolarization is altered.

3. ECG changes

- I, aVL, V1-2: Very tall R waves.
- V5-6: Smaller R waves.

4. Strain pattern in V1-2 and II, III, and aVF.

ECG #2406



Select Electrolyte Imbalances and Drug Effects

Objective

- Identify ECG changes which may occur with electrolyte imbalances and select medications.

I. Electrolyte imbalances

A. Hypokalemia

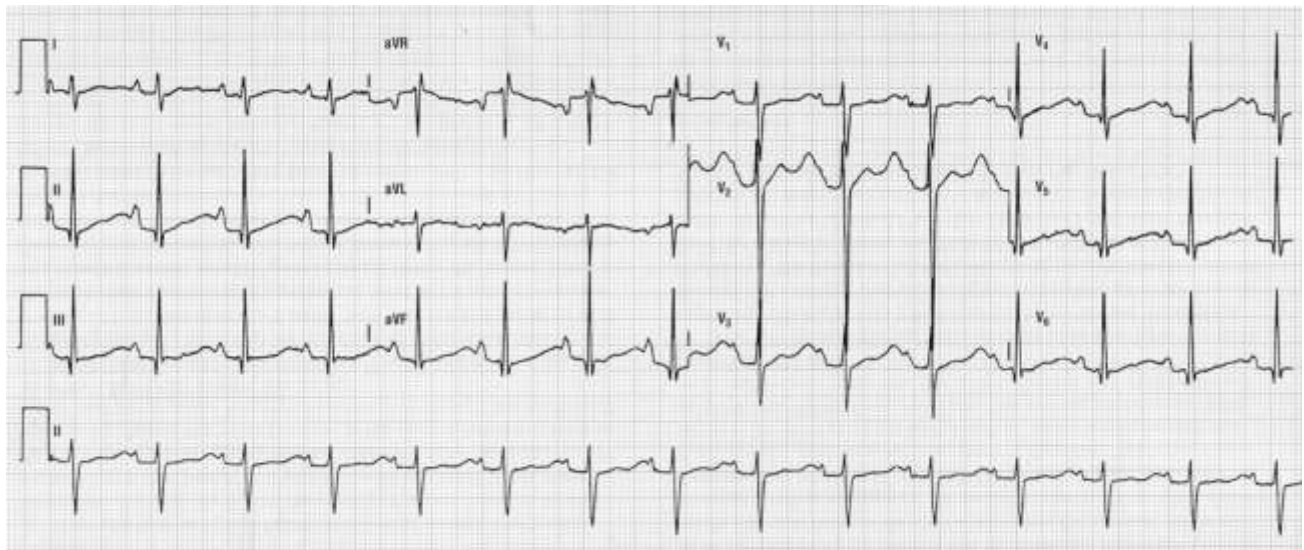
1. Positive U wave
2. ST segment depression
3. Flattened T wave
4. Longer PR
5. PVCs common



ECG #2501

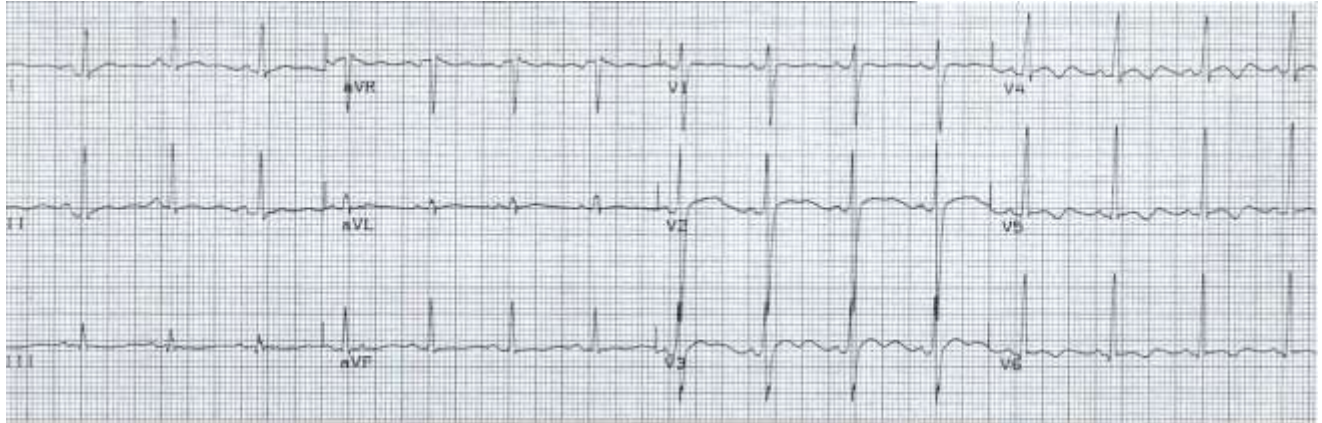
Clinical correlation

- 64-year-old male with severe vomiting and diarrhea for the past 2 days.
- Has a history of heart failure and COPD.
- K^+ on admission was 2.6 mEq/L.



Clinical correlation

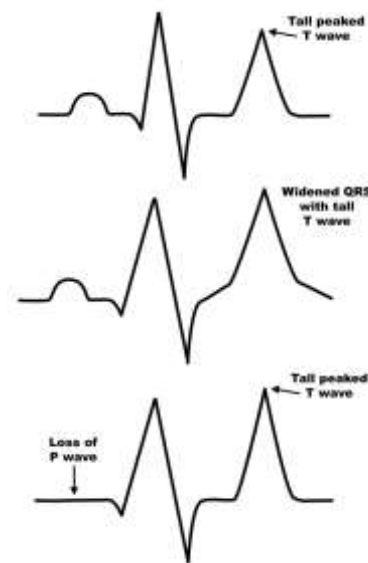
- 24-year-old male with an aspirin overdose and metabolic acidosis. Heart rate of 93 BPM.
- K^+ on admission was 2.5 mEq/L.



B. Hyperkalemia

1. T wave abnormalities, especially tall and peaked T waves when potassium level greater than 5.5 mEq/L.

- Tall, peaked, and narrow ONLY occur 22% of the time.
- The other 78% of the time, the T waves are any combination of tall, peaked, narrow, or wide. The most common is tall and wide.



2. Intraventricular conduction delays

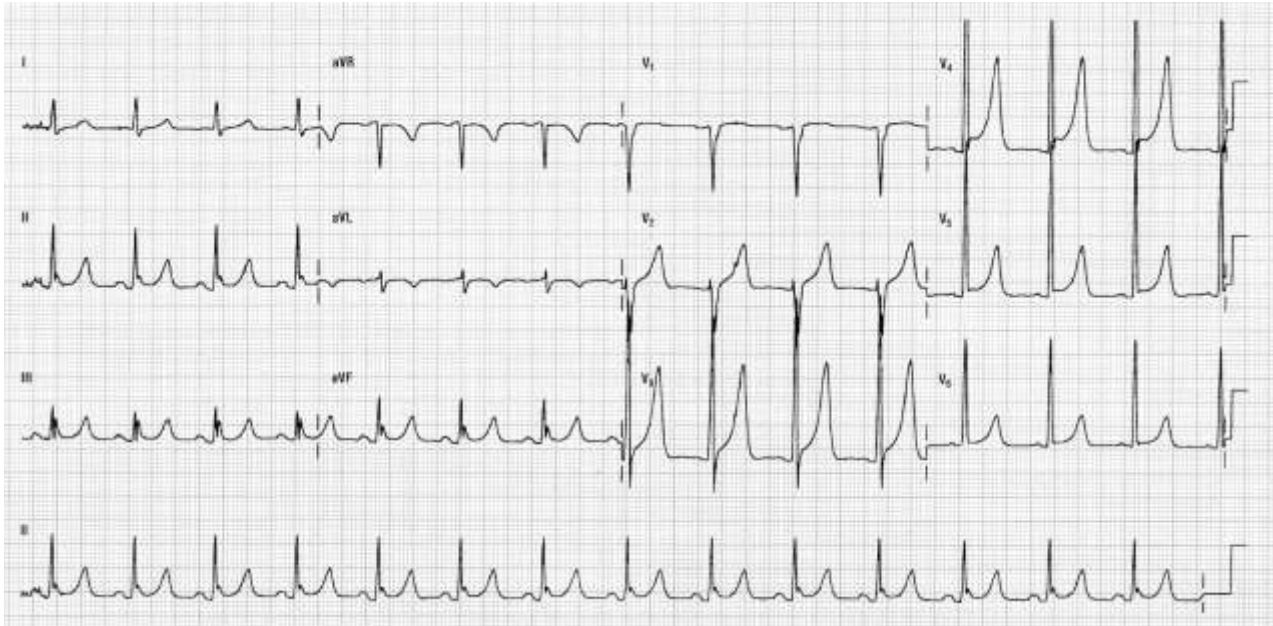
- Widening of the complexes usually begins when the potassium concentration is at 6.5 mEq/L or greater.
- ANY time you diagnose an IVCD, think hyperkalemia.

3. P waves missing or of decreased amplitude when potassium > 7.0 mEq/L or greater.

4. ST segment changes simulating an injury pattern.

5. Cardiac dysrhythmias of any type.

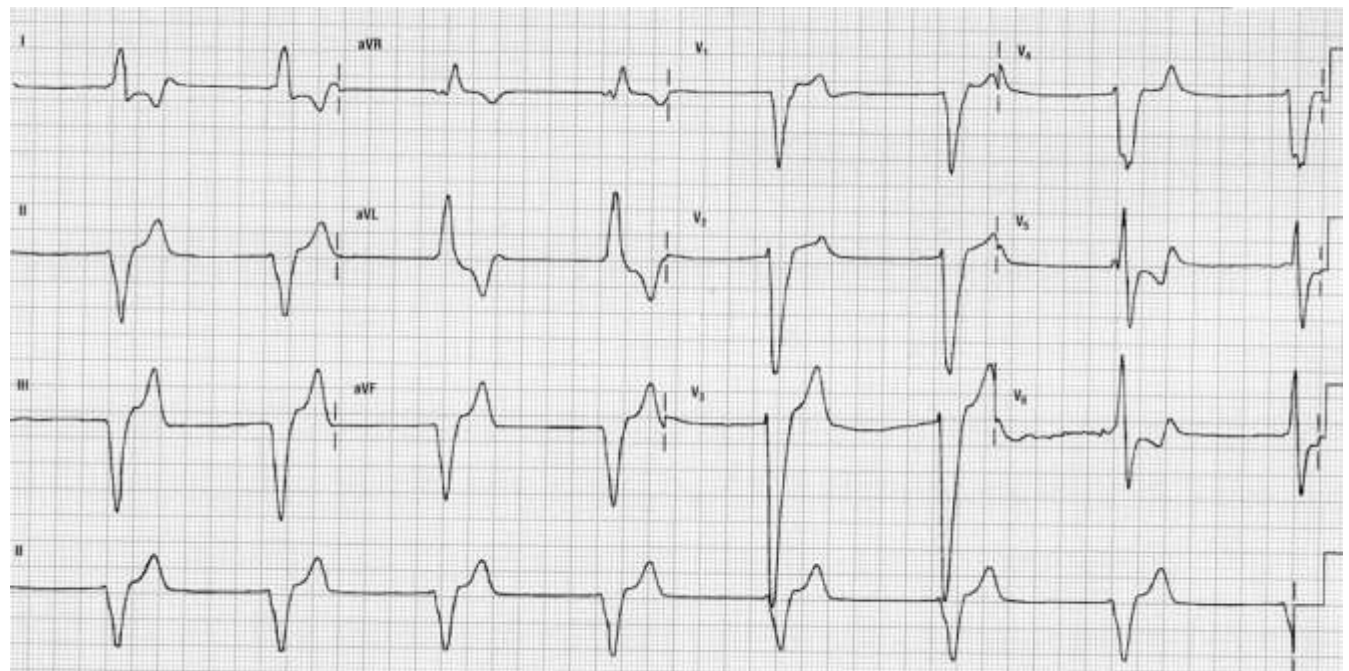
ECG #2503



ECG #2504

Clinical correlation

- 28-year-old male presents to the emergency room after being ill for 3 days with dyspnea, congestion and a productive cough of green sputum. His level of consciousness is diminished. No additional history is available.
- Placed on the monitor. Heart rate 40 BPM. ECG obtained.



6. Treatment of hyperkalemia

<u>Drug</u>	<u>Onset of action</u>
10% calcium gluconate over 2 minutes. (Duration of one hour)	2-3 minutes
Sodium bicarbonate 1 mEq/kg	30-60 minutes
D ₅₀ and 10 units of regular insulin IV	30-60 minutes
Sodium polystyrene (Kayexalate) 30-60 grams	2-3 hours

Dialysis

Peritoneal dialysis to remove 10-15 mEq of potassium per hour.

Hemodialysis to remove 25-35 mEq of potassium per hour.

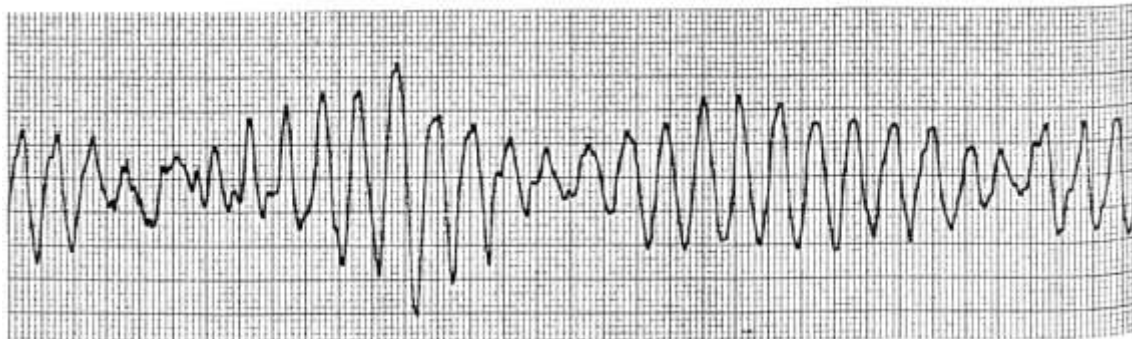
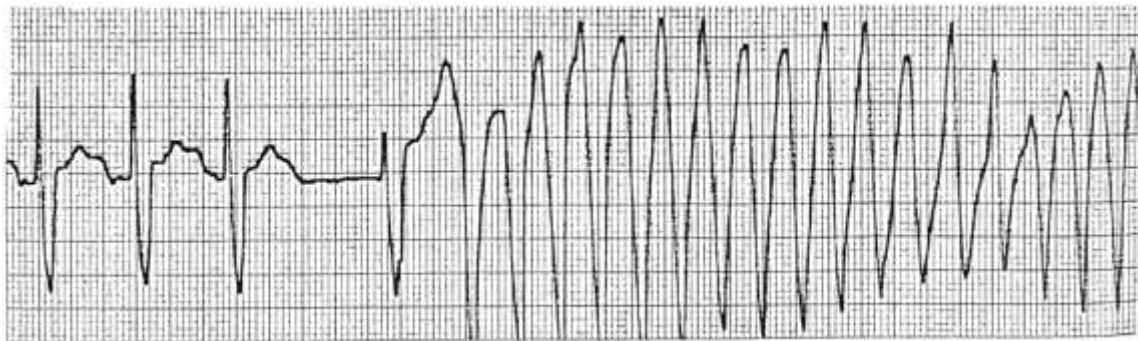
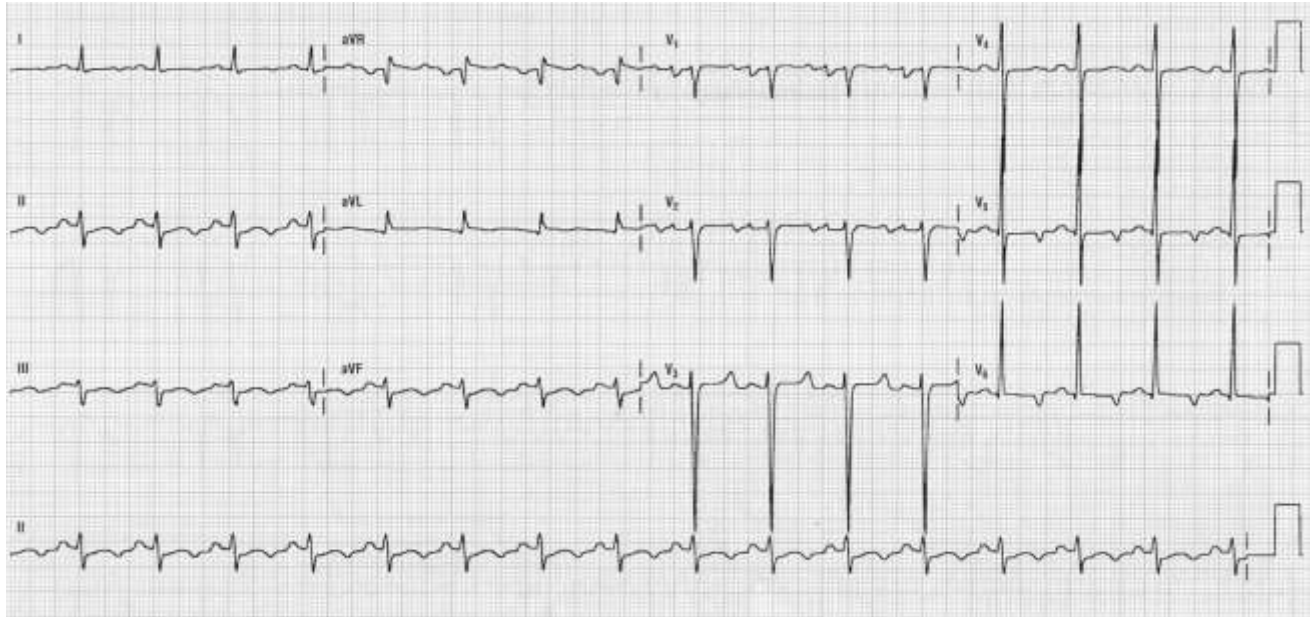
C. Hypocalcemia / Hypomagnesemia

1. Slightly prolonged QRS complex.
2. Prolonged QT interval.
3. ST segment depression.
4. Premature ventricular contractions.
5. As the level drops, myocardial sensitivity increases.
6. Lethal rhythms such as torsades de pointes.

ECG #2505

Clinical correlation

- 30-year-old female with a history of anorexia.
- Complaining of profound weakness and muscle twitching. Ca^{+} level 5.7 mg/dL



D. Hypercalcemia / hypermagnesemia

1. Widened QRS complex.
2. Tall T waves.
3. Shortening of the QT interval. ST segment may disappear.
4. Various types of AV block.
5. Complete heart block and cardiac arrest.

II. Drug effects

A. Sodium channel blockers

1. Lengthened QRS and QT intervals.
2. Possible AV blocks.
3. Slowed SA node conduction and possible blocks.
4. Dysrhythmias.
5. Select class I antidysrhythmics
 - Disopyramide (Norpace)
 - Flecainide (Tambocar)
 - Lidocaine (Xylocaine)
 - Mexiletine HCl (Mexitil)
 - Moricizine (Ethmozine)
 - Procainamide (Pronestyl)
 - Propafenone HCl (Rhythmol)
 - Quinidine gluconate (Quinaglute)
 - Quinidine sulfate (Quinidex)
 - Tocainide (Tonocard)

B. Calcium channel blockers

1. AV conduction delay
2. Varies depending on the drug
3. Select calcium channel blockers used for dysrhythmias
 - Adenosine (Adenocard)
 - Diltiazem (Cardizem)
 - Verapamil (Calan, Isoptin, Verelan)

C. Beta blockers

1. Decreases the automaticity of the SA node and the Purkinje fibers.
2. Slowing of the sinus rate.
3. May see AV blocks
4. Select beta blockers used for dysrhythmias
 - Acebutolol (Sectral, Monitan)
 - Esmolol (Brevibloc)
 - Metoprolol (Lopressor)
 - Propranolol (Inderal)
 - Sotalol (Betapace)

D. Potassium channel blockers

1. Prolongs the entire action potential.
2. Slowed SA conduction.
3. Slowed AV conduction.
4. Prolonged QT interval.
5. Suppression of the Purkinje system.
6. Select potassium channel blockers used for dysrhythmias.
 - Amiodarone (Cordarone, Pacerone)
 - Dofetilide (Tikosyn)
 - Ibutilide fumarate (Corvert)
 - Sotalol (Betapace)

E. Phenothiazines and cyclic antidepressants

1. Wide QRS.
2. Prolonged QT interval.
3. Prolonged PR interval.
4. T wave abnormalities.
5. Dysrhythmias are common in overdose.
6. Select phenothiazines
 - Chlorpromazine (Thorazine)
 - Fluphenazine (Prolixin, Permitil)
 - Trifluoperazine (Stelazine, Suprazine)
 - Perphenazine (Trilafon)
 - Prochlorperazine (Compazine)

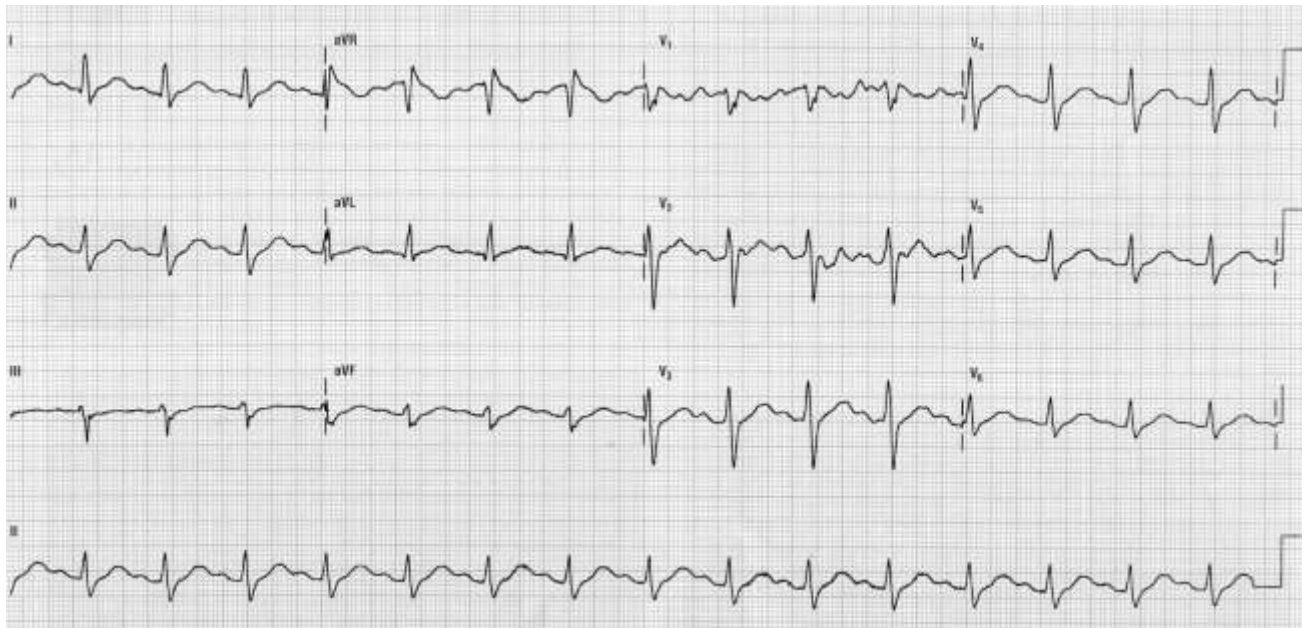
7. Select cyclic antidepressants

- Amitriptyline (Elavil)
- Amoxapine (Ascendin)
- Clomipramine (Anafranil)
- Desipramine (Norpramin)
- Doxepin (Adapin, Sinequan)
- Imipramine (Tofranil)
- Maprotiline (Ludiomil)
- Mirtazapine (Remeron)
- Nortriptyline (Aventyl, Pamelor)
- Protriptyline (Vivactil)
- Trimipramine maleate (Surmontil)

ECG #2507

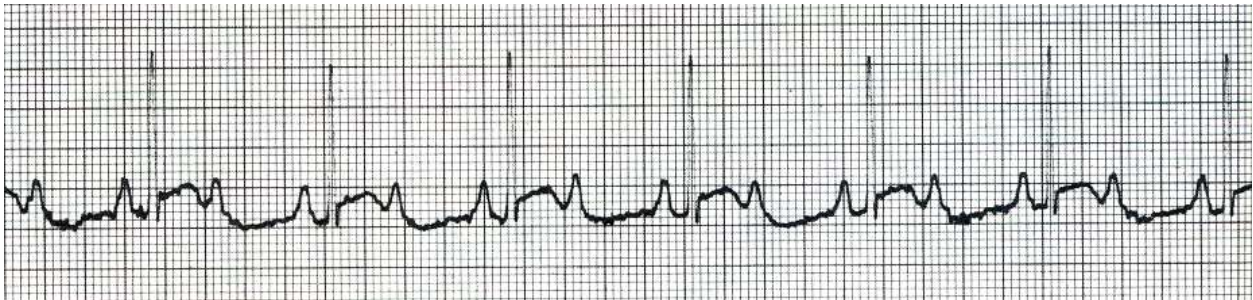
Clinical correlation

- 18-year-old who had a fight with his girlfriend and decided to take his mother's amitriptyline (Elavil).



F. Digoxin (Lanoxin)

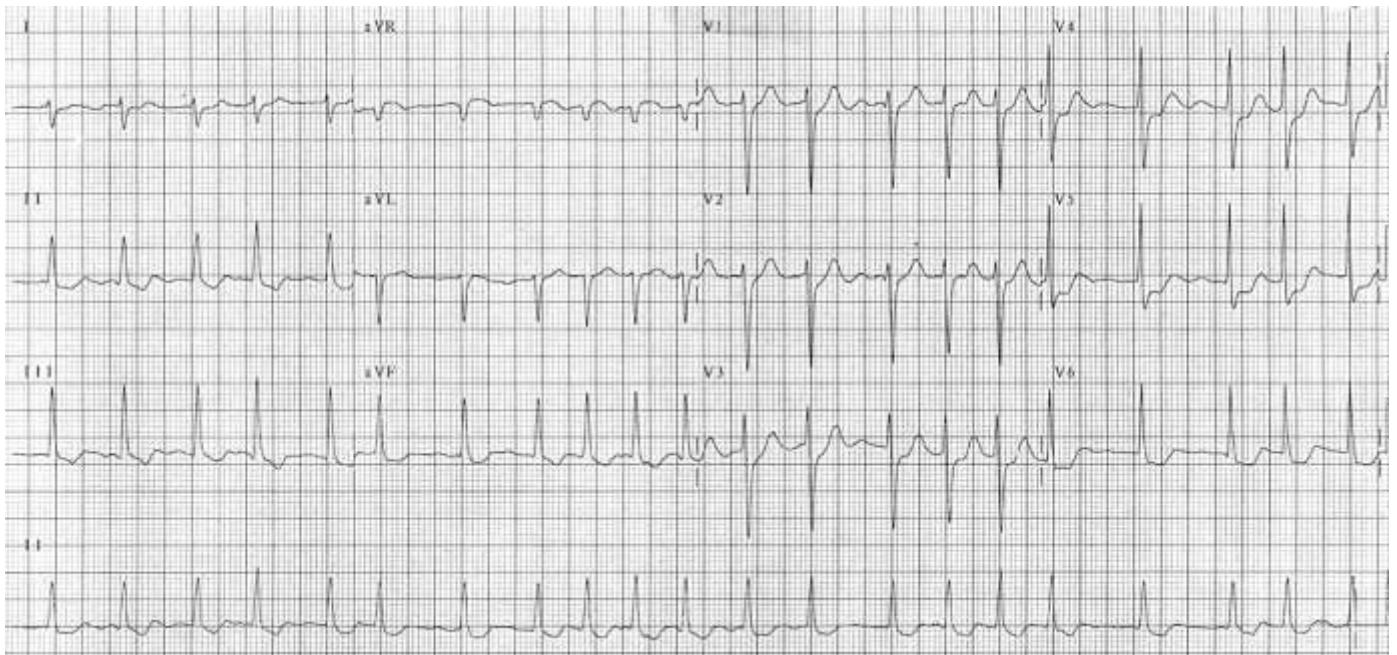
1. Scooped ST segment depression.
2. T waves are shorter and can be biphasic.
3. U waves more prominent.
4. In toxicity, see a wide variety of dysrhythmias.
5. Bigeminal PVCs and atrial tachycardia with 2:1 block.
6. Atrial fibrillation with a regular ventricular rate.
7. Ventricular tachycardia.



ECG #2508

Clinical correlation

- 79-year-old female with a history of atrial fibrillation and on digoxin.
- Heart rate is 130 BPM. QRS=0.10



Select Medical Conditions

Objective

- Correlate the clinical manifestations seen in select pathological conditions with the ECG changes which may occur.

I. Pericarditis: Inflammation of the pericardium which is usually a complication of a viral illness. Complication is pericardial effusion with greater than 50 ml of fluid in the pericardial sac. Chronic pericarditis causes fibrous thickening of the sac.

A. Causes of acute pericarditis

- Viral infection
- Post-MI (Dressler's syndrome)
- Trauma
- Uremia
- Connective tissue disorders
- Endocrine disorders

B. Clinical manifestations

- Chest pain
- Dyspnea when supine
- Low grade fever
- Weakness and fatigue
- Dry cough
- Dependent edema
- Pericardial friction rub
- Elevated sedimentation rate

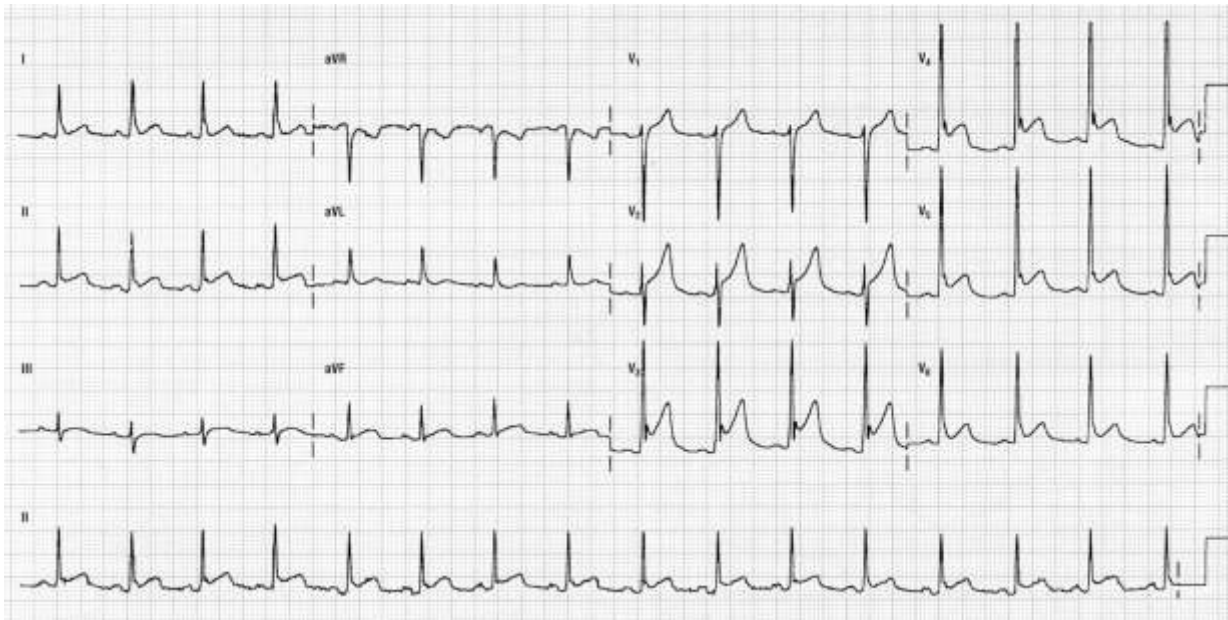
C. ECG changes

- PR depression.
- Diffuse ST elevation.
- Scooping, upwardly concave ST segments.
- Notching at the end of the QRS.



Clinical correlation

- 32-year-old presents with a history of low grade fever for the last week.
- Began having chest pain about two hours ago. Took a couple of Motrin and tried to lay down and the pain got worse.



- II. Pericardial effusion and tamponade: Accumulation of fluid or blood in the pericardial sac. If blunt or penetrating trauma to the pericardium or heart, blood will accumulate rapidly and be life threatening. The larger the effusion, and the faster it develops, the more symptomatic the patient will be.

A. Clinical manifestations

- Clear lung sounds
- Tachycardia
- Pulsus paradoxus
- Pulsus alternans
- Elevated CVP

Beck's triad

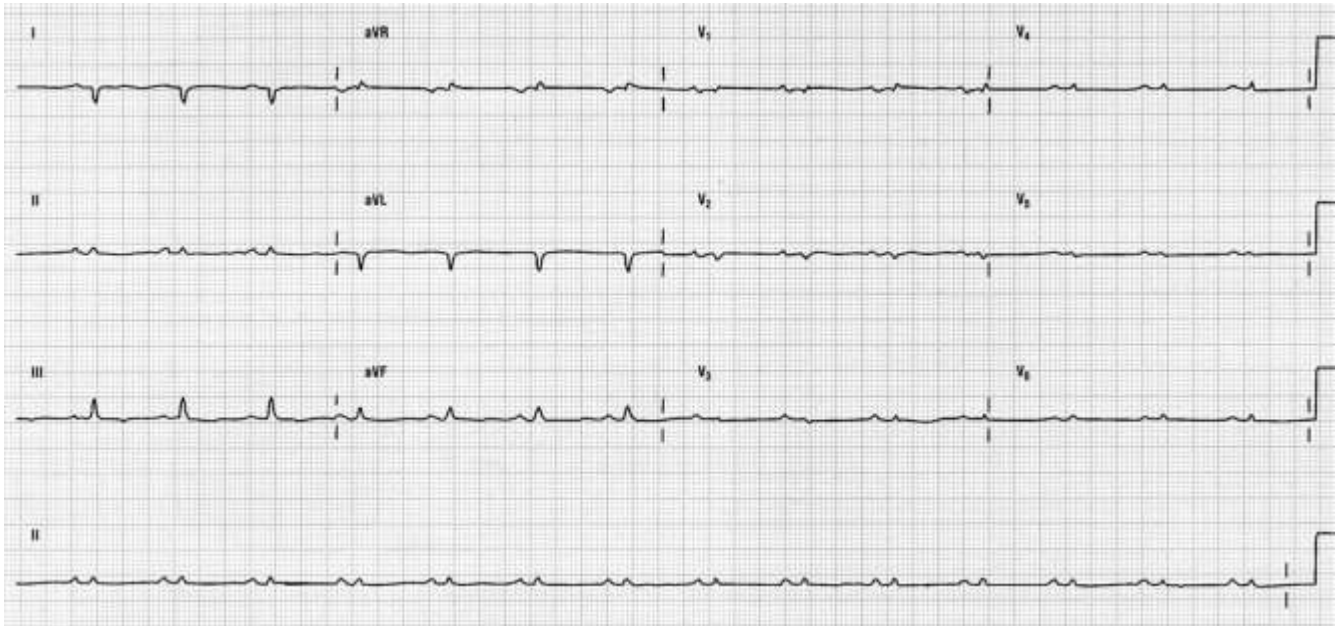
- Narrowing pulse pressure
- Jugular vein distention
- Muffled heart tones

B. ECG changes

- Small QRS complexes. The larger the effusion, the smaller the complexes.
- Electrical alternans may be present.

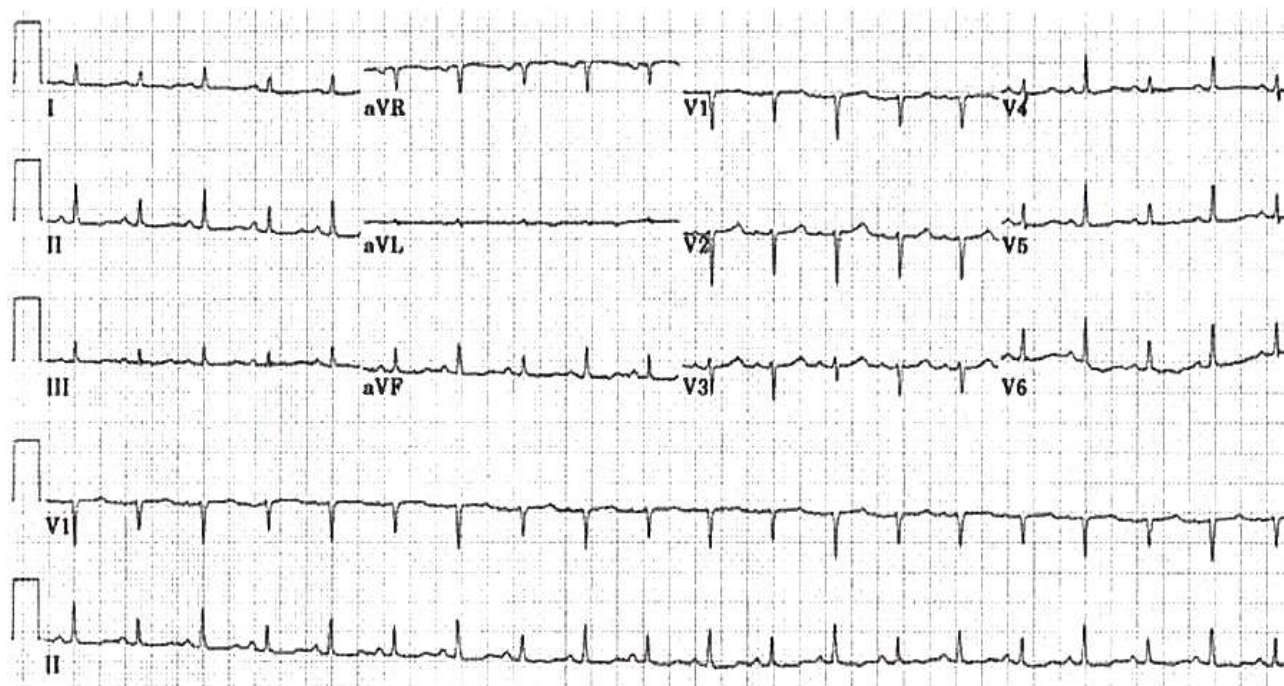
Clinical correlation

- 55-year-old male with a history of coronary artery disease and esophageal cancer.
- On beta blockers.



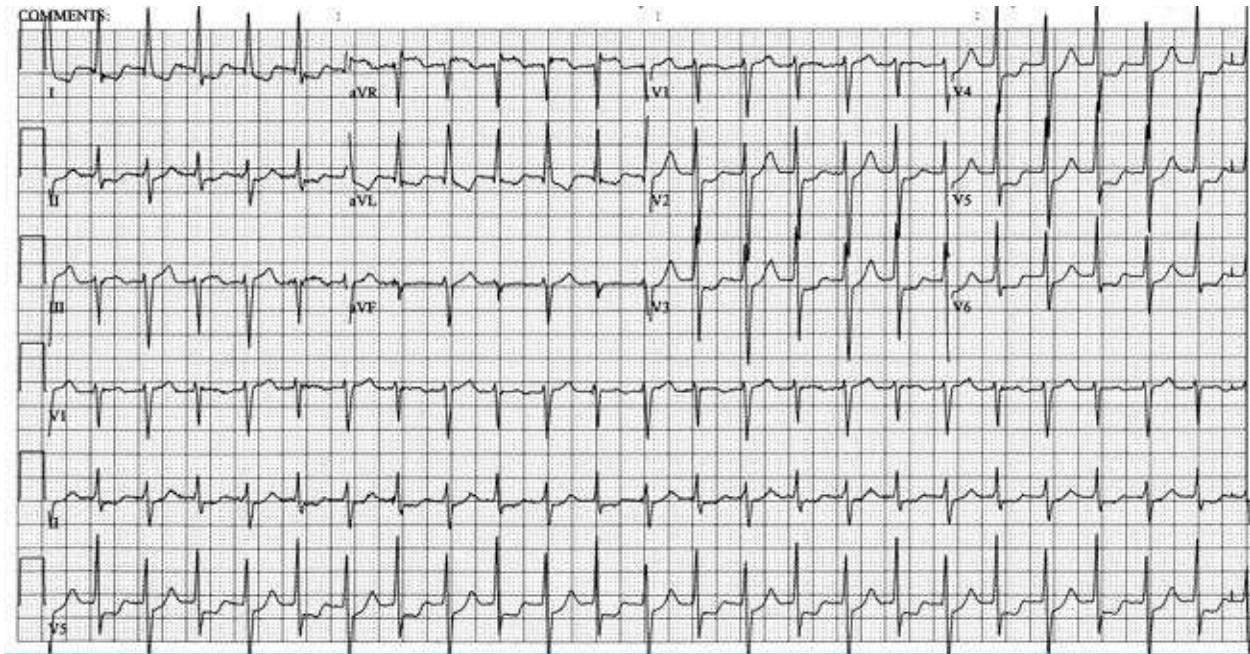
Clinical correlation

- 62-year-old male who had been involved in a motor vehicle crash with a front end collision and significant damage to the steering wheel. He was the driver of the car and has bruising from the seat belt injury.
- He is on the cardiac monitor when his monitor pattern changes.



Clinical correlation

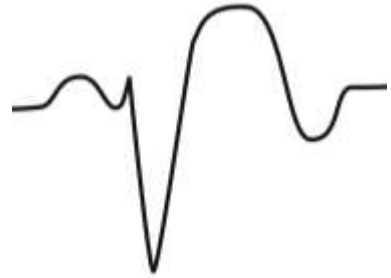
- Electrical alternans in atrial fibrillation may be a normal variant. When seen in sinus rhythm, associated with pericardial effusion and tamponade.
- 82-year-old female with a history of dementia who resides at the nursing home.
- Recent dismissed after a hospitalization for atrial fibrillation with RVR that was successfully converted with diltiazem (Cardizem).



III. Ventricular aneurysm: Localized dilation of the left ventricle. Often occur in the apex and may calcify over time and accumulate thrombus. True aneurysms are not prone to rupture but cause increased morbidity and mortality because of recurrent ventricular dysrhythmias, thought to be incited at the junction of the aneurysm with adjacent, normal myocardial tissue.

A. Clinical manifestations

- Fatigue
- Heart failure
- Dysrhythmias
- Stroke symptoms
- Sudden death if a pseudo aneurysm ruptures.

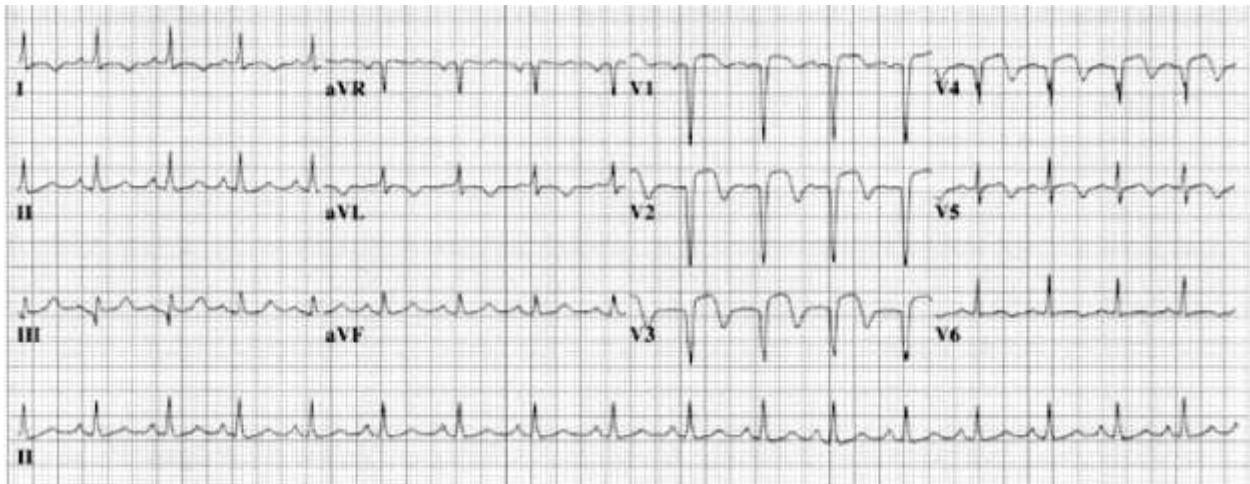


B. ECG changes: Persistent ST segment elevation in the anterior leads.

ECG #2605

Clinical correlation

- 72-year-old female with shortness of breath, fever, and a productive cough.
- Chest x-ray demonstrates pneumonia. She has a history of CAD.
- ECG completed due to age and her history.

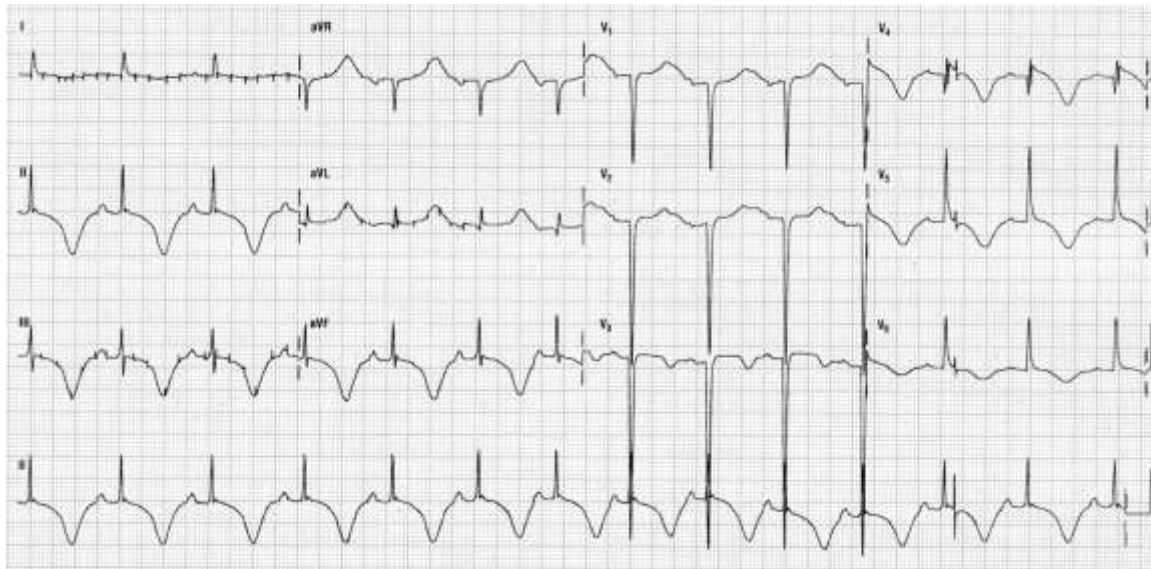


- IV. Cerebrovascular accident: Reduction of blood flow to a part of the brain structure due to occlusion of a blood vessel by a clot or the rupture of a vessel.
- A. Clinical manifestations depend on the lobe of the brain involved and the cerebral artery occluded.
- B. ECG changes include very broad T waves.

ECG #2606

Clinical correlation

- 78 year-old brought to the ED with a severe headache and neurological deficits. CT demonstrated a large intracranial bleed.



V. Hypothermia: Core temperature of less than 95 degrees F. Profound hypothermia is a core temperature of 86 degrees F or less.

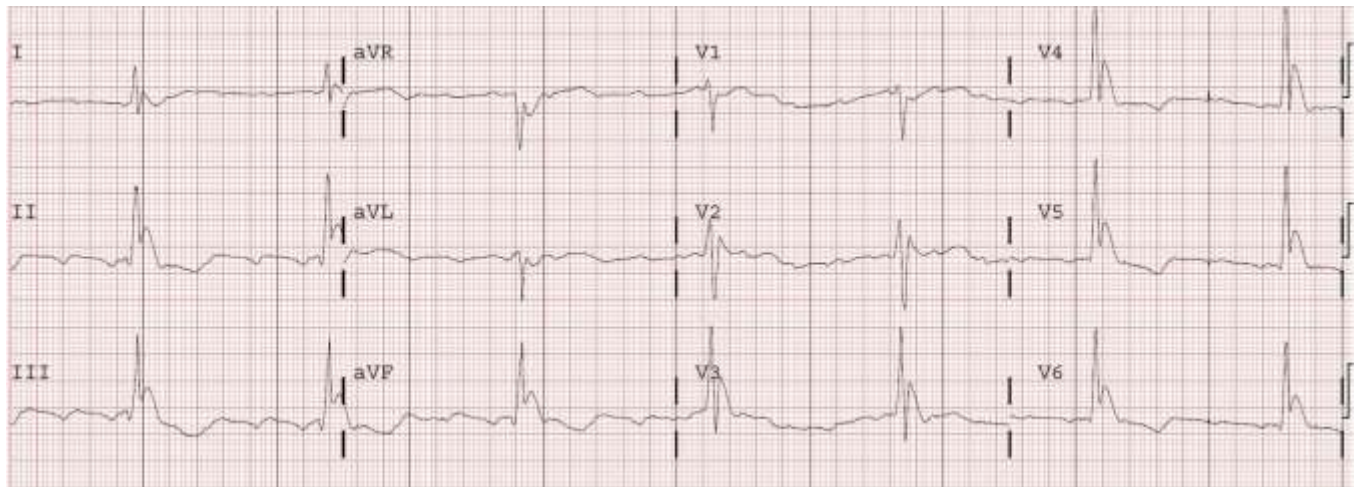
A. Osborne waves seen on the terminal portion of the QRS and a long QT interval.

B. Treatment

- Careful handling to prevent ventricular fibrillation.
- Maximize oxygenation.
- Environmental measures to rewarm.
- External rewarming.
- Active rewarming.
- Accurate temperature monitoring.
- Cardiac pacing for bradycardia.

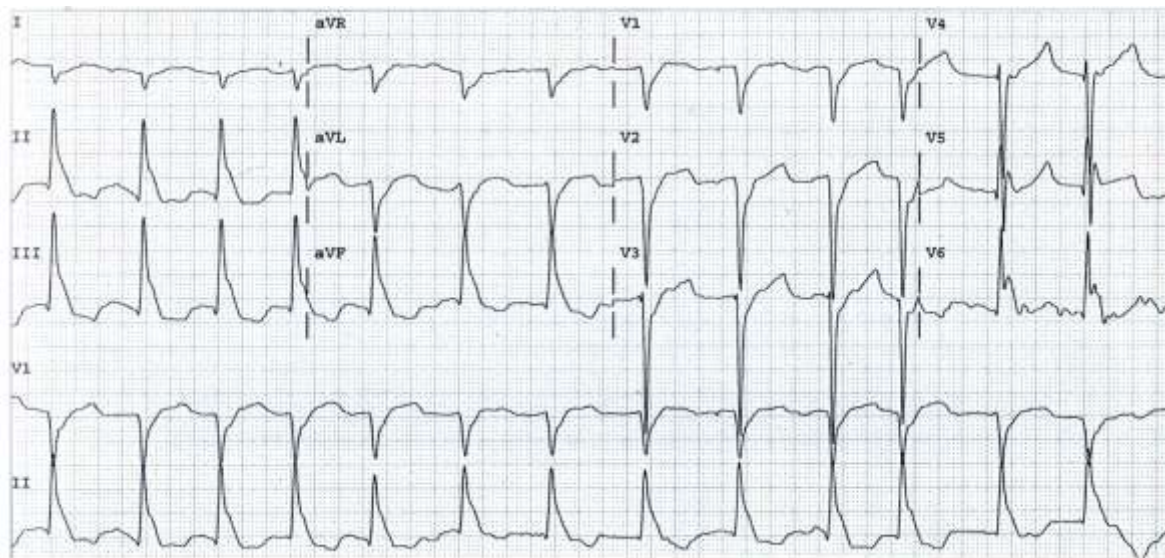


ECG #2607



Clinical correlation

- 61-year-old man found unconscious with BP 60/40 and temperature 84 degrees.



VI. Hypothyroidism: Condition where the thyroid gland does not make enough hormones. May be caused by inadequate iodine intake, stress or aging.

A. Clinical manifestations

- Intolerance to cold
- Lethargy
- Apathy
- Dry skin
- Brittle nails and hair
- Receding hairline and hair loss
- Facial and eyelid edema
- Thick tongue, slow speech
- Blank expression
- Enlarged thyroid
- Muscle aches and weakness
- Extreme fatigue
- Anorexia with weight gain
- Constipation
- Menstrual disturbances

B. Late clinical signs in myxedema

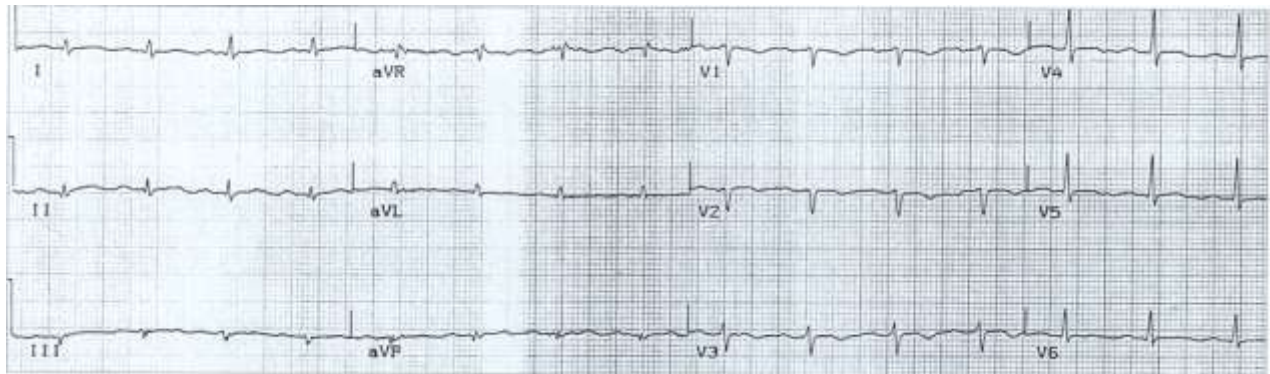
- Low temperature
- Bradycardia
- Lethargy
- Thick skin
- Cardiac complications

C. Myxedema coma can be precipitated by acute illness, anesthesia, withdrawal of thyroid medication and use of medications such as sedatives, narcotics, lithium or amiodarone (Cordarone). Treatment is intubate and IV thyroid hormone.

D. ECG changes include bradycardia, low voltage complexes and prolonged QT interval.

Clinical correlation

- 45-year-old man with myxedema and sepsis.
- Temperature of 101 degrees. Heart rate 100 beats per minute.



VII. Pulmonary embolism: Obstruction of blood flow to the pulmonary vasculature caused by an embolus. If pulmonary infarction occurs, lung tissue is destroyed. In massive pulmonary embolus, pulmonary hypertension and right-sided heart failure will be seen.

A. Virchow's triad is the predisposing factors to DVT and pulmonary embolus along with patient and family history of prior embolus.

Venous stasis

- | | |
|--------------------------------|-----------------------|
| • Bedrest | • Varicose veins |
| • Standing in one place | • Atrial fibrillation |
| • Sitting for prolonged period | • Heart failure |
| • Age > 55 years | • Burns |
| • Obesity | • Pregnancy |

Hypercoagulation

- | | |
|---------------|-----------------------|
| • Dehydration | • Birth control pills |
| • Cancer | • Smoking |
| • COPD | |

Vascular injury

- Abdominal, pelvic, or thoracic surgery.
- Leg or pelvic trauma or surgery.

B. Clinical manifestations

- Hypoxia and confusion
- Chest pain
- Dyspnea
- Hemoptysis
- Tachycardia
- Fever
- Hypotension
- Crackles

C. ECG changes

- S 1, QT 3 in 15-30% of cases.
- T wave inversion in leads V1 - V3.
- Right Bundle Branch Block or incomplete RBBB.
- Low amplitude waveforms.

D. Treatment

- Prevention and recognition of risk factors.
- Anticoagulants because of the positive feedback of clotting.
- Embolectomy if very large.
- Inferior vena cava filter for prevention if high risk factor or multiple clots and resultant high pulmonary pressures.

Lead I
S wave



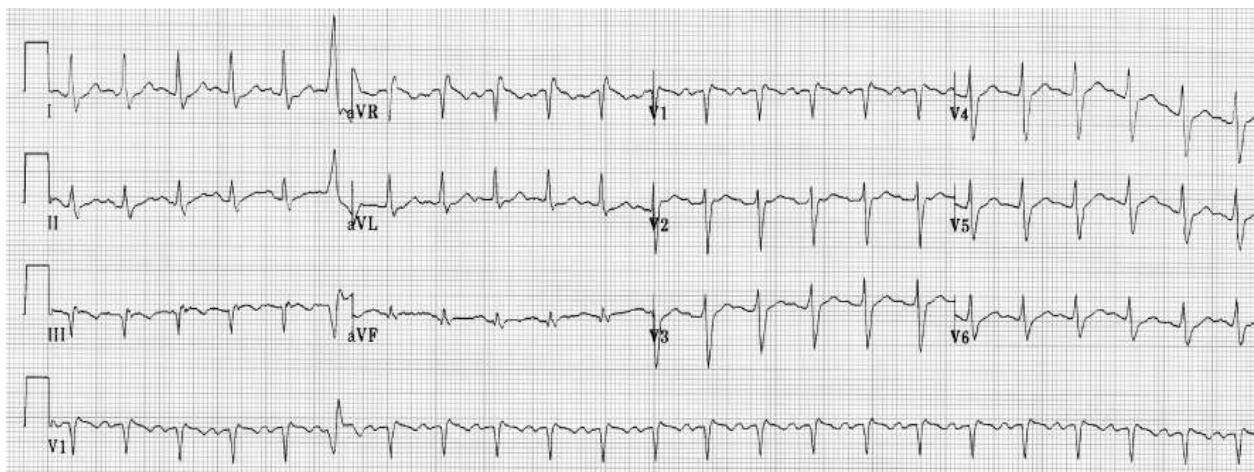
Lead III
Q wave and
↓ T wave



ECG #2610

Clinical correlation

- 62-year-old female presents from the provider's office with a history of dyspnea for the past week. Had been placed on antibiotics by the provider with no improvement in clinical status.
- CXR completed and shows bilateral lower lobe pneumonia.



Clinical correlation

- 66-year-old female. On evaluation she tells you she is having pain the right chest and had a sudden onset of shortness of breath 2 days ago. She has been feeling poorly since then and came to the ED today for evaluation.
- HR is 140 BPM. BP 150/92.

