

EKG INTERPRETATION

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INTRODUCTION

Medicine complicates. We take simple concepts and cloud them over with a plethora of information which **certainly for EMS** providers, is not necessary and only adds confusion to a typically confounding topic. EKG interpretation falls into that category. The goal of this paper is to streamline the interpretation down to a level that is practical and inclusive for your level of practice.

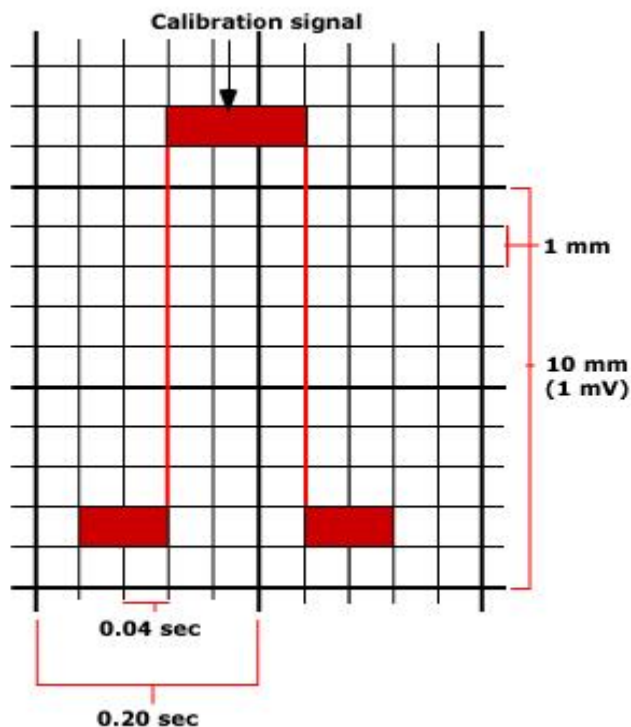
The EKG is a way of interpreting information about the heart. It is more than just rhythms and conduction disturbances or assessing for heart attacks. The EKG can also tell us information about toxicities from medications and overdoses, metabolic abnormalities and infections to the heart such as pericarditis and pericardial effusions.

I. EKG FUNDAMENTALS

Interpretation in any form requires an understanding of the fundamental parts that create the structure. If you were to read, you need to know all the letters and use that understanding to form words. Words turn into sentences, which become paragraphs which then becomes a book. EKG's are similar in that each part has to be understood so as to interpret the meaning of the EKG.

The EKG is mapped out on graph paper which is referred to as the grid. The grid allows you to measure the speed of the heart along with the duration of different parts of the EKG. For example, the rate is determined by measuring the distance between complexes.

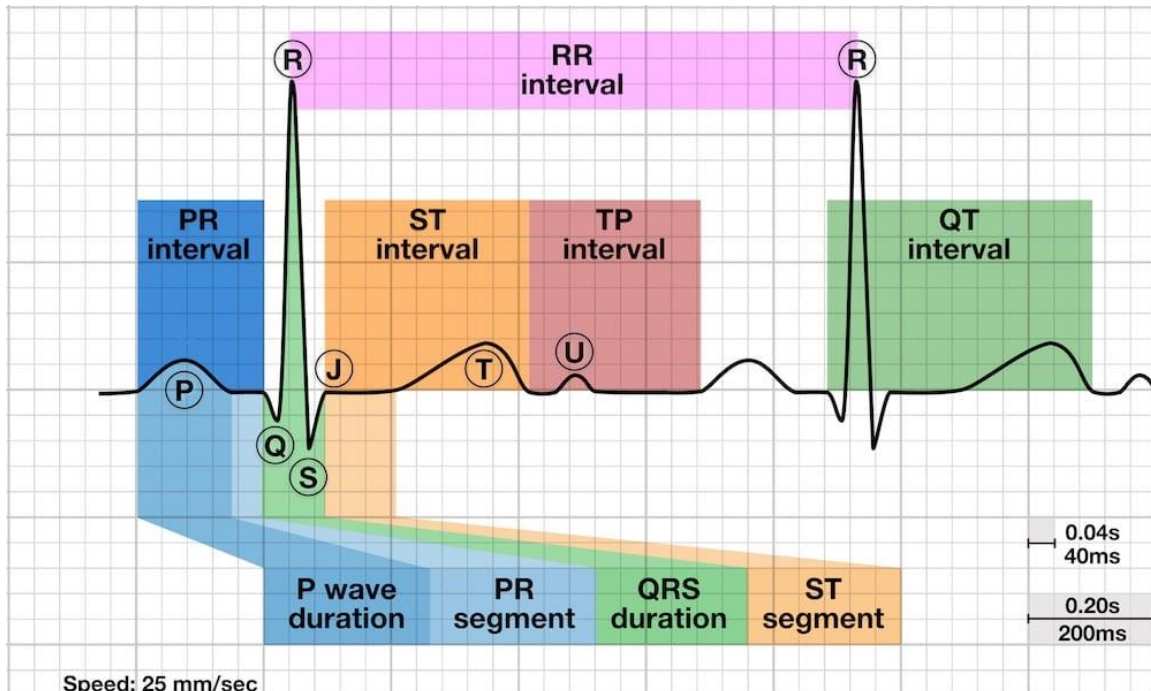
A Left Bundle has to have a QRS duration greater than 0.12 seconds. Without graph paper, these measurements would become very tedious.



The horizontal measurements are 0.04 seconds for each small box and 0.2 seconds for the darker boxes which is equal to 5 small boxes. The vertical boxes measure the amplitude and are in millivolts. Their function is important in determining processes such as MI's and Left Ventricular Hypertrophy. One small box is equal to 1 millimeter and 10 small boxes or 2 large boxes is equal to 1 millivolt.

The fundamental parts of the EKG are the complexes; P wave, QRS complex and T wave. Each reflects the conduction of a different part of the heart and the morphology can give you insight into injury patterns such as heart attacks, conduction disturbances, electrolyte abnormalities etc. Segments and intervals are also referred to. Remember that segments have no “waves” and intervals include “waves”.

P WAVE: The P wave represents the depolarization or contraction of the atrium. Normally, the impulse fires off from the Sinoatrial node (SA Node) which is located in the right atrium. The left atrium then fires off and the impulses combine and travel down the atria to the Atrioventricular node (AV Node). A normal duration of this impulse is 0.12 seconds or 3 small boxes.



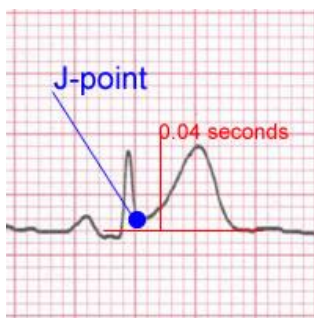
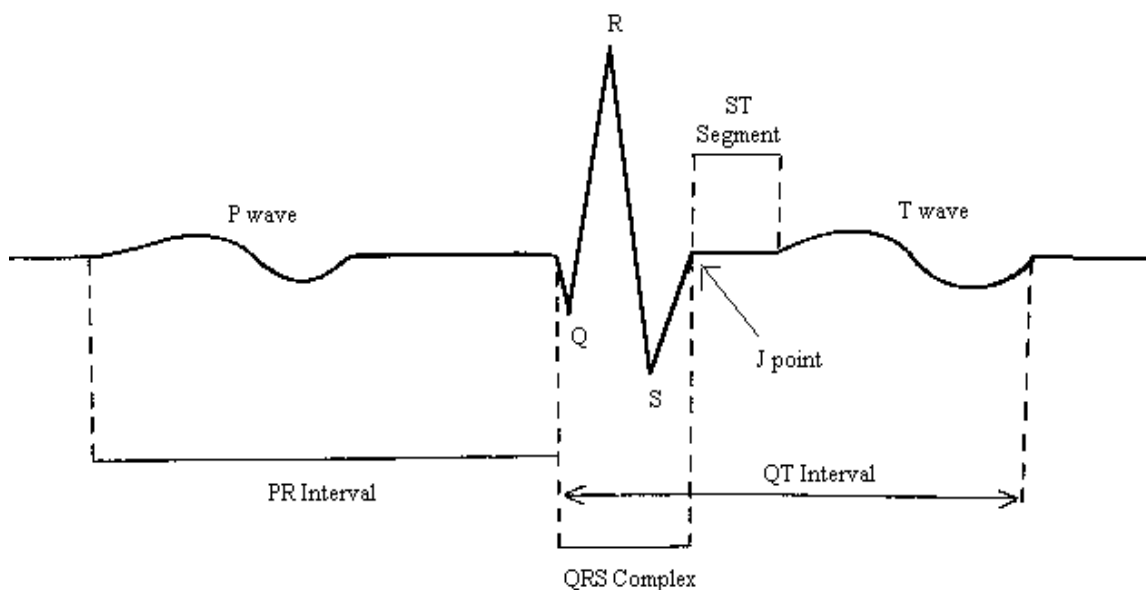
PR INTERVAL: The PR Interval is measured from the beginning of the P wave to the beginning of the R wave (or Q wave). It measures the time it takes for the atria to depolarize or contract, travel through the atrioventricular node and reach the His-Purkinje fibers where the QRS then contracts. The PR Interval shortens when the heart rate increases and lengthens out when the heart rate slows down. Normal duration is 0.14 to 0.20 seconds.

QRS COMPLEXES: The QRS complex represents the depolarization of the ventricles. Here you will examine the heart for injury patterns such as myocardial infarctions and metabolic abnormalities. The Q wave is a negative or downward deflection on the EKG and represents the septal depolarization or contraction. It is seen in V1 and V2. Small Q waves can be seen in other leads but typically are less than 25% of the R wave. When large Q waves are seen in other leads, this represents previous or evolving heart attacks. R waves are positive or upward deflections that represent the depolarization or contraction of the left ventricle. S waves are negative or downward deflections and represent the depolarization or contraction of the high lateral wall.

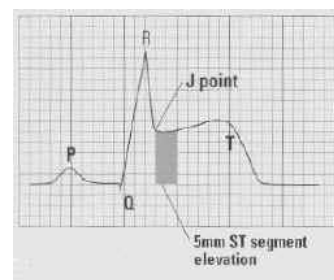
T WAVE represents ventricular repolarization. During this period, the cells equilibrate and prepare to depolarize or contract again. The heart is refractory during most of this period and no impulses or activity can occur. The morphology is slower than the contraction of the ventricle and the T wave has a broad slow upstroke.

ST SEGMENT: The ST Segment represents a “silent” period in the conduction activity of the heart. During this period which begins at the end of the S wave and ends at the beginning of the T wave, the heart is resting and in between depolarization (think heart contracting) and repolarizing (think heart filling) when the muscle gets prepared for the next contraction. Normally the ST Segment is a flat or “isoelectric” line. This is also the area where injuries to the heart are seen. In cardiac ischemia and pericarditis, the ST Segment can have an upward, downward, convex or concave pattern.

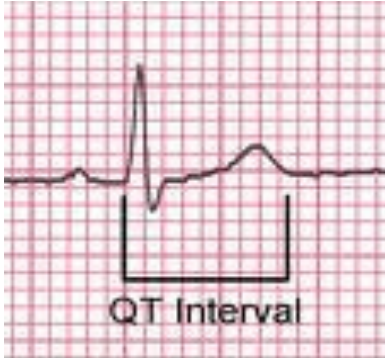
J POINT: In order to determine an injury pattern to the heart, one must first determine if there is true elevation or depression to the ST Segment. The **J POINT** is the point where depolarization ends, and repolarization begins. It is right at the end of the S Wave and beginning of the ST Segment. Typically, it is slightly below the baseline in a normal EKG. It will rapidly move upward and is normally at baseline or isoelectric within two small boxes or 0.08 seconds. The PR Segment is best to look at in determining the isoelectric line



The injury pattern is determined by looking for **J POINT ELEVATION** which is seen by going over horizontally one small box or 0.04 seconds. When there is elevation or depression of 0.1 milivolts or more, (one or more small boxes an injury pattern exists).



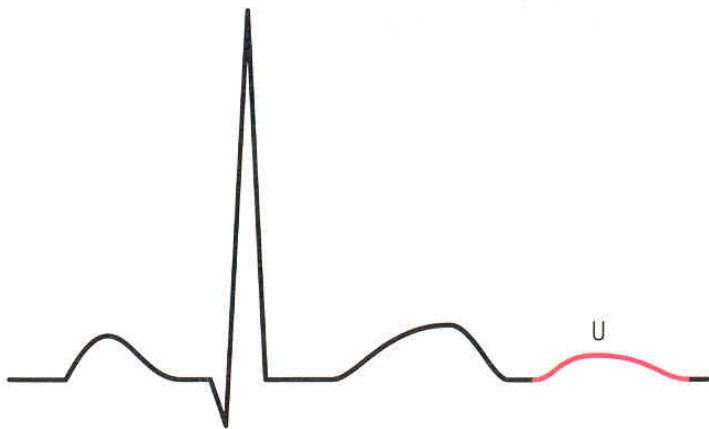
The repolarization phase is measured by the **QT INTERVAL**. The interval is measured from the beginning of the QRS and ends at the termination of the T Wave. The size changes with the heart rate. A rapid rate shortens the QT Interval, and a slow rate lengthens it. Because of this variability, QT Interval is corrected to account for the rate.



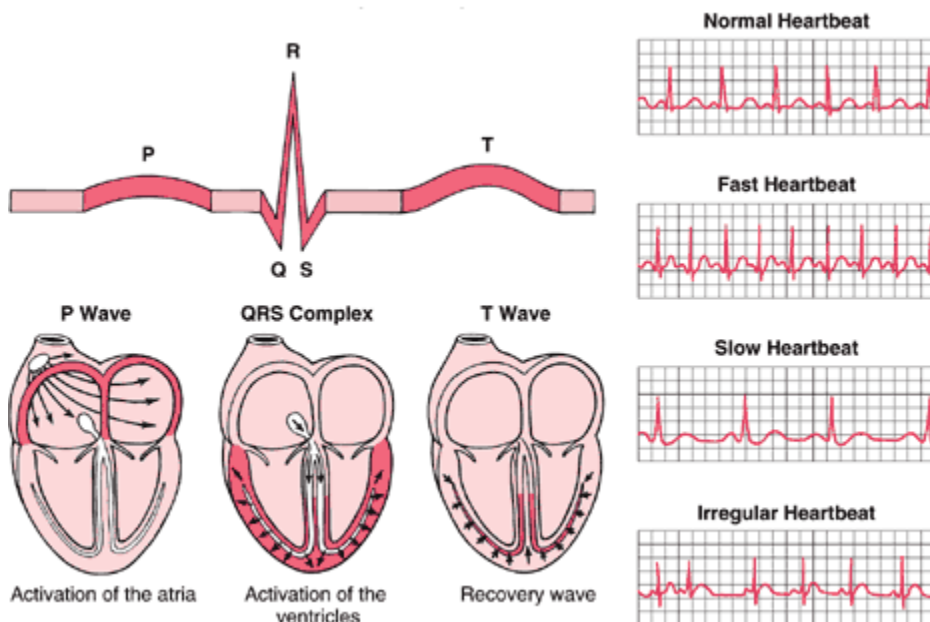
Normally it is less than or equal to 0.44 seconds. Anything longer can be indicative of underlying pathology which could lead to arrhythmias.

For practical purposes the QT interval should be less than the distance from one R wave to the next R wave. For those interested, the **QT Interval corrected** is: $QT_c = QT \text{ Interval} \div \sqrt{\text{RR interval in seconds}}$.

U WAVES occur after the T Waves. The mechanism is not completely understood but can indicate metabolic abnormalities such as **hypokalemia**. The presence of a U Wave is not always indicative of hypokalemia.



EKG COMPLEXES SUMMARY

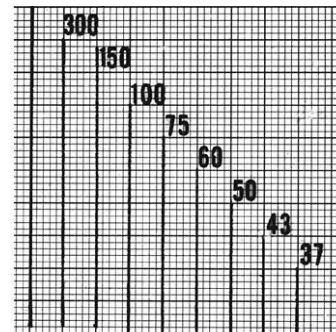


II. EKG INTERPRETATION

Knowing the fundamental parts of the EKG is similar to learning the alphabet. Once you know all the letters, you can form words and sentences which can be interpreted. EKG interpretation requires that you can determine the rate, recognize different rhythms, and interpret different morphologies that can represent disease states and myocardial infarctions.

HEART RATE: The heart rate is measured by determining the distance between the QRS complexes. The exact rate is not necessary and with irregular rhythms is almost impossible to determine. The easiest way to determine the rate is to divide 60 seconds by the number of large blocks separating the QRS complexes or counting out the number of blocks separating the QRS complexes.

- 60 seconds / 0.2 (1 large block) = 300
- 60 seconds / 0.4 (2 large blocks) = 150
- 60 seconds / 0.6 (3 large blocks) = 100
- Normal heart rate: 60-100
- Bradycardia: less than 60
- Tachycardia: greater than 100



AXIS: Axis or axis deviation provides information about the magnitude and the direction of the electrical impulse. The axis, primarily, can be normal, left or right axis deviation. While information can be gleaned from the axis, it really has very little practical utility in the pre-hospital and emergency setting.

AXIS IS DETERMINED BY LOOKING AT THE QRS COMPLEX IN LEADS 1 and aVf.

	QRS LEAD 1	QRS LEAD aVf
Normal Axis	Upright	Upright
Left Axis	Upright	Downward
Right Axis	Downward	Upright
Pathologic Left Axis	Downward	Downward

CAUSES OF LEFT AXIS DEVIATION

- Normal variation (physiologic, often with age)
- Mechanical shifts, such as expiration, high diaphragm (pregnancy, ascites, abdominal tumor)
- Left ventricular hypertrophy
- Left bundle branch block
- Left anterior fascicular block
- Congenital heart disease (atrial septal defect, endocardial cushion defect)
- Emphysema
- Hyperkalemia

- Ventricular ectopic rhythms
- Preexcitation syndromes
- Inferior wall myocardial infarction.

CAUSES OF RIGHT AXIS DEVIATION

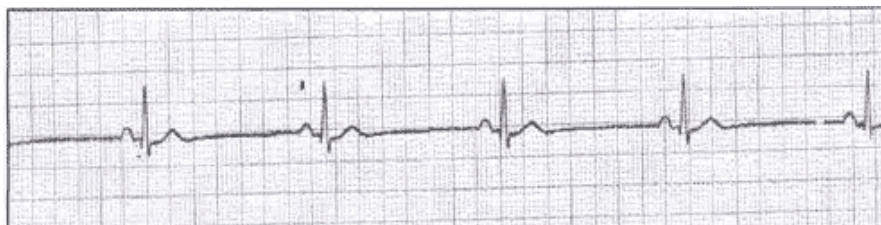
- Normal variation (vertical heart with an axis of 90°)
- Mechanical shifts, such as inspiration and emphysema
- Right ventricular hypertrophy
- Right bundle branch block
- Left posterior fascicular block
- Dextrocardia
- Ventricular ectopic rhythms
- Preexcitation syndrome
- Lateral wall myocardial infarction

RHYTHM ANALYSIS is performed every time an EKG is interpreted. Rhythms can be regular and irregular. The expectation in a normal rhythm is that for every p wave there is an associated QRS complex. When this occurs, the EKG is considered a **SINUS RHYTHM**. Within this context, the sinus rhythm can be a **SINUS BRADYCARDIA**, less than 60 beats per minute: **NORMAL SINUS RHYTHM**, 60 to 100 beats per minute and **SINUS TACHYCARDIA**, 100 to 180 beats per minute. Rhythms can be regular but not sinus. In this case, the P wave is not followed by a QRS complex. P waves can hide in the QRS complex in **JUNCTIONAL or AV NODAL REENTRANT TACHYCARDIA**. With **AV REENTRANT OR VENTRICULAR TACHYCARDIA**, the P wave may be hidden in the ST Segment. These rhythms are typically faster than a sinus tachycardia with rates between 140 and 220 beats per minute. Typically, reentrant tachycardias have a normal QRS with the complex less than 0.12 seconds. They on occasion do present as a wide tachycardia and a QRS greater than 0.12 seconds. In these cases, the rhythm could be a **SUPRAVENTRICULAR RHYTHM WITH ABERRANCY OR A VENTRICULAR TACHYCARDIA**

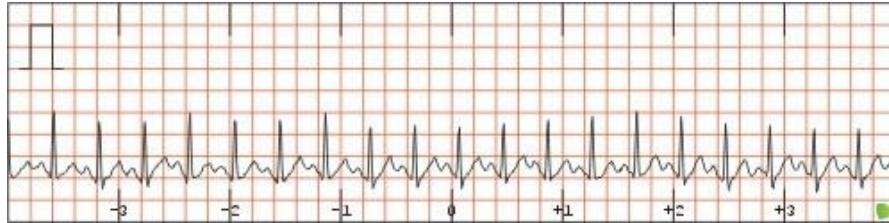
REGULAR RHYTHMS

	MORPHOLOGY	RATE BEAT PER MINUTE
Normal Sinus Rhythm	P Wave Followed By QRS	60-100
Sinus Bradycardia	P Wave Followed By QRS	Less than 60
Sinus Tachycardia	P Wave Followed By QRS	Between 100 & 180
AV Nodal/Junctional/SVT	P Wave Hidden in the QRS	140 - 220
AV Reentrant/Ventricular	P Wave May Hide in the ST Segment	140 - 220

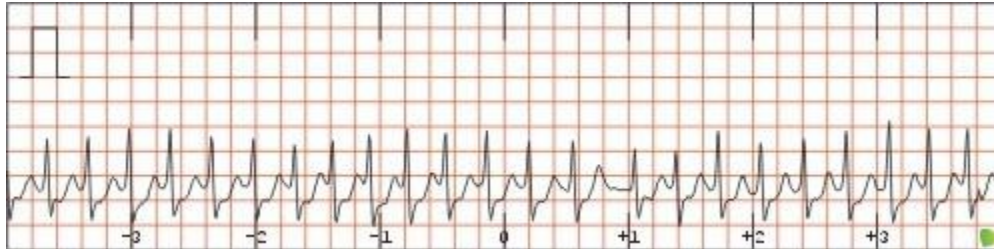
SINUS BRADYCARDIA RATE 30



SINUS TACHYCARDIA RATE 150

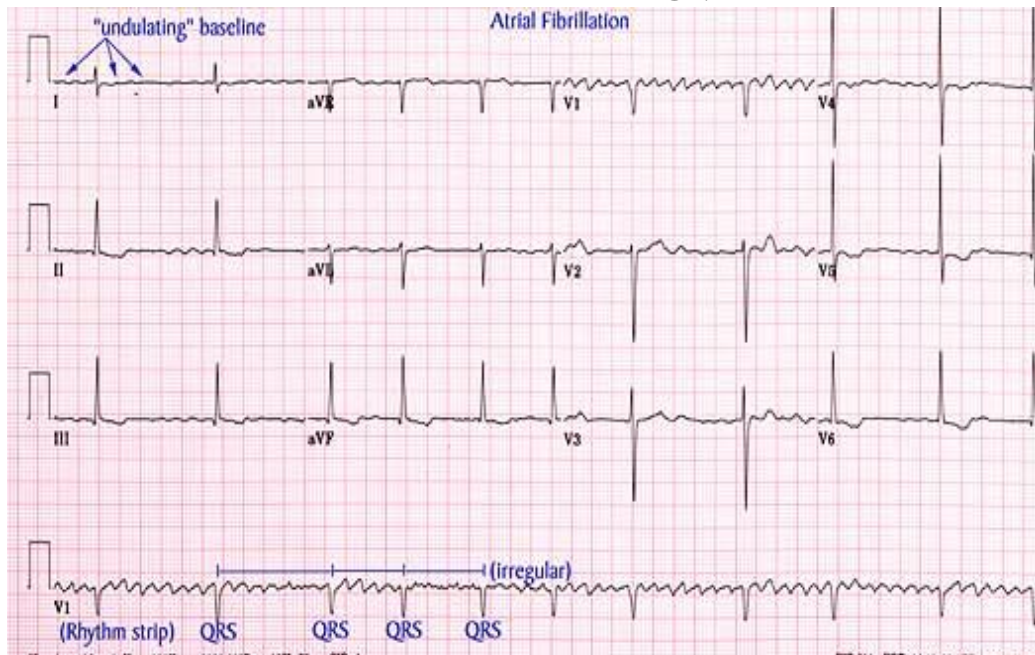


ATRIOVENTRICULAR REENTRANT TACHYCARDIA RATE 196



The primary **IRREGULAR RHYTHM** you will see is **ATRIAL FIBRILLATION**. It has two precursor rhythms in **ATRIAL FLUTTER AND MULTIFOCAL ATRIAL TACHYCARDIA** that while appearing regular, are oftentimes antecedent rhythms to atrial fibrillation. **ATRIAL FIBRILLATION** is characterized by being “irregularly irregular”. Multiple foci of p waves fire off but only one can go through the AV node. It can only pass after the node has repolarized and is ready to release a charge. For that reason, the depolarization of the ventricle is random and “irregular”. Because of all the P waves firing off at once, the P wave is typically not seen. You can visualize what is happening by thinking of the AV node as a door. People line up to get into a building and enter one at a time when the attendant says to pass. The process is orderly, just as a P wave passes through in a sinus rhythm. Now presume a fire breaks out in the room. Everyone panics and crowds the door trying to push their way through. Randomly and “irregularly” the people squeeze their way through when there is enough space, just as the P waves in atrial fibrillation move through the AV node.

ATRIAL FIBRILLATION

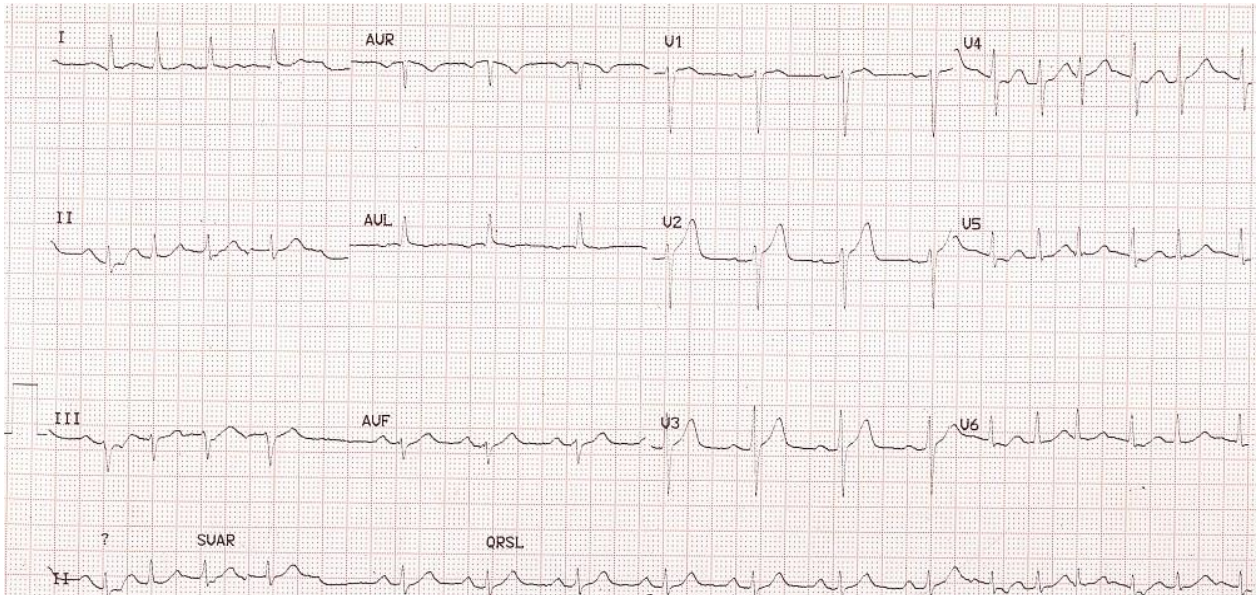


ATRIAL FLUTTER has a regular pattern of P waves and QRS complexes. It typically will have 2 to 4 P waves to every QRS complex. The ratio of P waves to QRS complexes is referred to as atrial flutter with 2:1, 3:1, 4:1 etc. block. This block gives atrial flutter the characteristic saw tooth or picket fence look to the P waves. Uncontrolled atrial flutter typically presents 2:1 block and will have a rate of 150 beats per minute. Whenever the heart rate is 150, always consider atrial flutter. Oftentimes the P waves are buried in other complexes.

Atrial Flutter				
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

MULTIFOCAL ATRIAL TACHYCARDIA is seen commonly with COPD patients. It too is considered a precursor to atrial fibrillation. Multifocal Atrial Tachycardia typically has 3 or more different P waves. The heart rate is also greater than 100. The rhythm is typically irregular because there are several different but distinct P waves firing off. The p-p interval is irregular but there is a distinct P wave followed by a QRS complex.

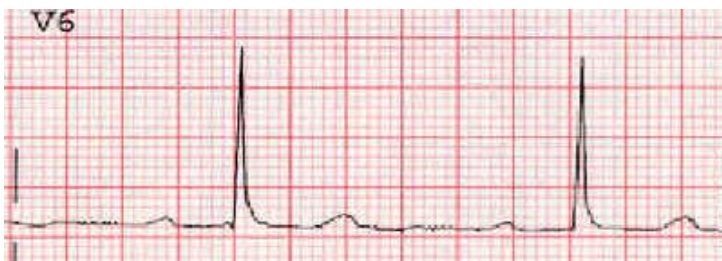
MULTIFOCAL ATRIAL TACHYCARDIA



ATRIAL VENTRICULAR BLOCKS are problems with the AV NODE. There is a variable level of blockage with the P wave passing through the AV node. The degree and variability of blockage determines whether it is a first-, second- or third-degree block.

FIRST DEGREE AV BLOCK results in P wave taking longer than normal to get through the AV node. The PR interval is 0.2 seconds or greater. The interval never changes and there are no clinical or pathologic conditions associated with it.

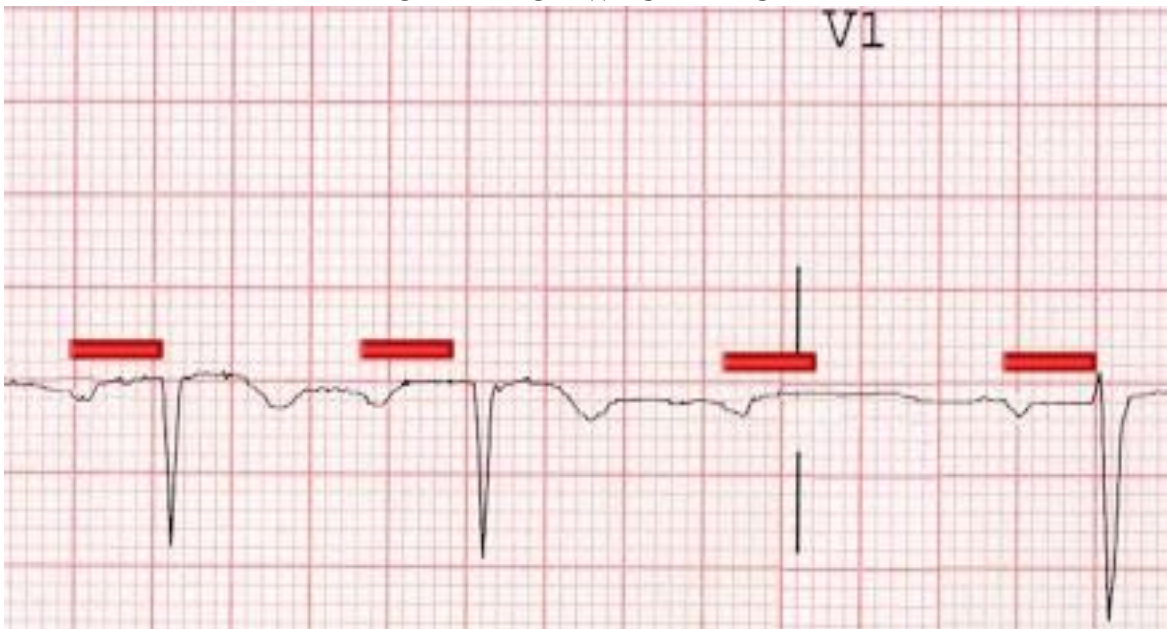
FIRST DEGREE AV BLOCK



SECOND DEGREE AV BLOCK has two forms: **MOBITZ 1 OR WECKEBACH** and **MOBITZ 2**. Both of these blocks, but especially Mobitz 2 are considered prodromal to a third-degree block and will end up in the patient getting a pacemaker. Drugs such as beta blockers, calcium channel blockers and digoxin as well as structural damage cause second and third AV blocks. In second degree blocks, there is a dropped QRS complex. With third degree blocks, the P waves and QRS complexes beat independently of each other.

With both these blocks, think of the AV node as being a door with an irritating older brother standing behind it. You are the P wave, and your job is to get through the door and turn on the switch also known as the ventricle. In **MOBITZ 1 OR WECKEBACH** the older brother continually closes the door little by little, making it more and more difficult for you, the P wave to get through. On the first pass, the door is still open enough for you to zip right through. The next time the older brother has slightly closed the door and you have to slow down some to maneuver through. The next time you approach, the older brother has just about closed the door. It's a struggle, but you the P wave squeeze out to the other side and flip the switch to the ventricle. Now you approach the door again, but this time the door is shut. You can't get through and just like the P wave you stand there completely blocked and unable to get through to flip the switch.

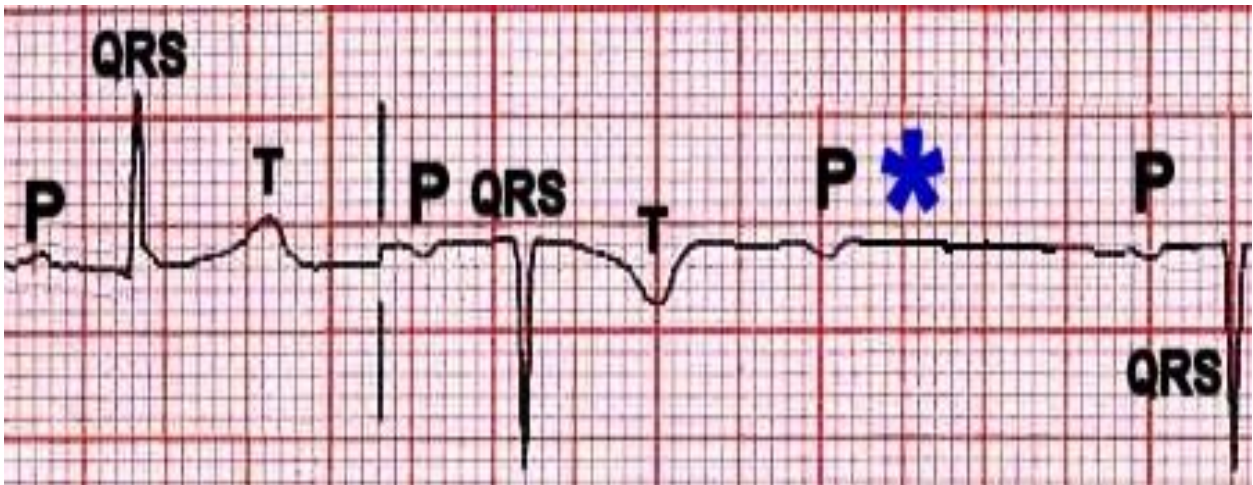
MOBITZ 1 OR WECKEBACH



- THE P WAVE MEETS RESISTANCE GOING THROUGH THE AV NODE.
- THIS CAUSES A PROLONGED PR INTERVAL WITH EACH CONDUCTION
- RESISTANCE INCREASES RESULTING IN A CONTINUED PROLONGED PR INTERVAL WITH EACH BEAT.
- AT SOME POINT THE RESISTANCE BECOMES TOO GREAT AND THE P WAVE CANNOT CONDUCT THROUGH THE AV NODE
- AT THIS POINT THERE IS A DROPPED QRS.

MOBITZ 2 is characterized by regular PR intervals followed by a blocked P wave and a missed QRS beat. Let's go back to the pain in the ass older brother. He just tortured you by obstructing you from getting through. The last insult was to prevent you from getting through altogether. Just like Lucy with Charlie Brown, he convinces you to do it again. After some promises, you agree. Everything seems fine at the beginning. You go through the first time without any problem. Again you approach the door and much to your surprise; you breeze right through and flip the switch. Off you go again! You have your rhythm down and a smile on your face. You're ready to fly through the door when BAM.... THE SON OF A BITCH SLAMMED THE DOOR SHUT IN YOUR FACE.

MOBITZ 2

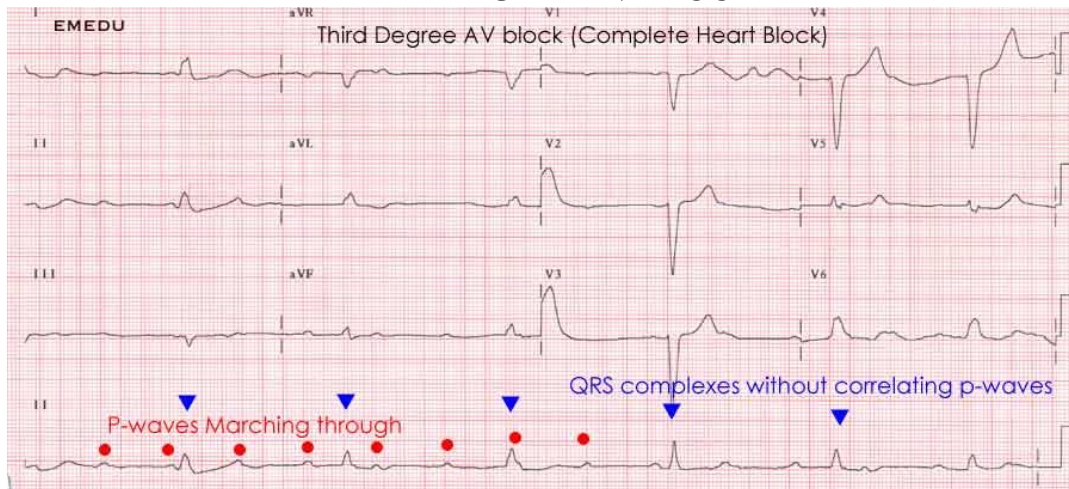


- RESISTANCE THROUGH THE AV NODE IS INTERMITTENT AND CAUSES COMPLETE BLOCKAGE WHEN IT OCCURS.
- PATTERN RESULTS IN NORMAL PR INTERVALS FOLLOWED BY DROPPED QRS COMPLEX AT THE TIME THE AV NODE CAUSE A COMPLETE BLOCKAGE OF THE CONDUCTION

THIRD DEGREE AV BLOCK is indicative of a complete blockage of the atrioventricular node. P waves initiate from the Sinoatrial node but cannot get through the AV node. The door is completely shut! Because of the complete blockage, the ventricle which has the ability to contract on its own will do so but at a slow rate. Third Degree AV Block will have P waves that march out in an orderly fashion with interspersed QRS complexes that also march in an orderly fashion.

Medications such as beta blockers and calcium channel blockers can cause the blockage. Damage due to scarring and heart attacks may result in damage as well. Septal myocardial infarctions can result in the blood supply to the node being permanently cut off. Inferior myocardial infarctions can cause swelling to the AV node that result in a temporary Third-Degree AV Block.

THIRD DEGREE AV BLOCK



VENTRICULAR ARRHYTHMIAS are characterized by atypical beats originating from the ventricle. The most common is a **PREMATURE VENTRICULAR CONTRACTION** while the others are life threatening and potentially lethal. Inclusive in these are **VENTRICULAR TACHYCARDIA, TORSADES DE POINTE AND VENTRICULAR FIBRILLATION.**

PREMATURE VENTRICULAR CONTRACTIONS are typically seen as a singular type or a multifocal variety. In most all the cases, they are a nuisance and not life threatening. The characteristic finding of a PVC is that there is a compensatory pause associated with it. The following beat will take longer to occur. When the beat before the PVC along with the PVC and the beat after are measured against three beats without a PVC, the distance is the same.

PREMATURE VENTRICULAR CONTRACTION



Note how the distance between the three normal beats equals the same distance of the three beats with the PVC in the middle and the “compensatory pause”.

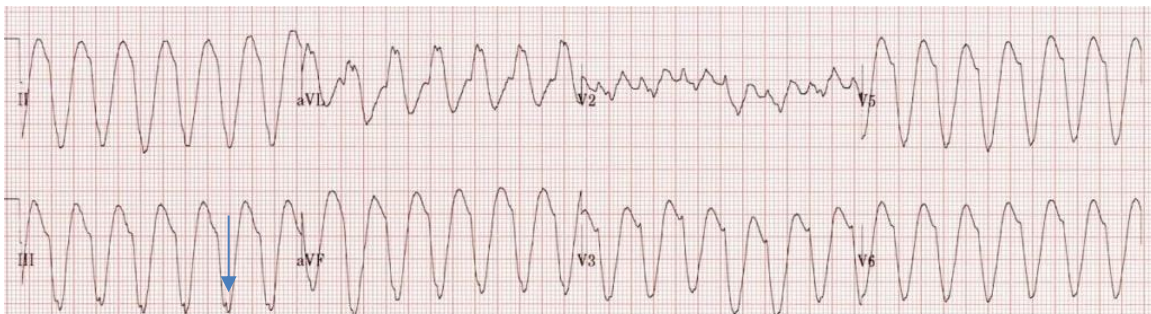
VENTRICULAR TACHYCARDIA is a wide complex tachycardia that originates in the ventricle and is regular, with a rate that is usually greater than 180 beats per minute. P waves are not typically seen. The complexes are wide and greater than 0.12 seconds. The ventricular arrhythmia results in poor perfusion of the heart and low pump pressure. Ultimately this rhythm can not be sustained and will digress to a **ventricular fibrillation.** Ventricular tachycardia is best diagnosed on a 12 lead EKG. Monitors can pick up motion and artifact that mimics Ventricular Tachycardia. However, complexes that are

sustained for greater than 30 seconds or result in hypoperfusion manifested as hypotension and/or altered mental status are going to be Ventricular Tachycardia.

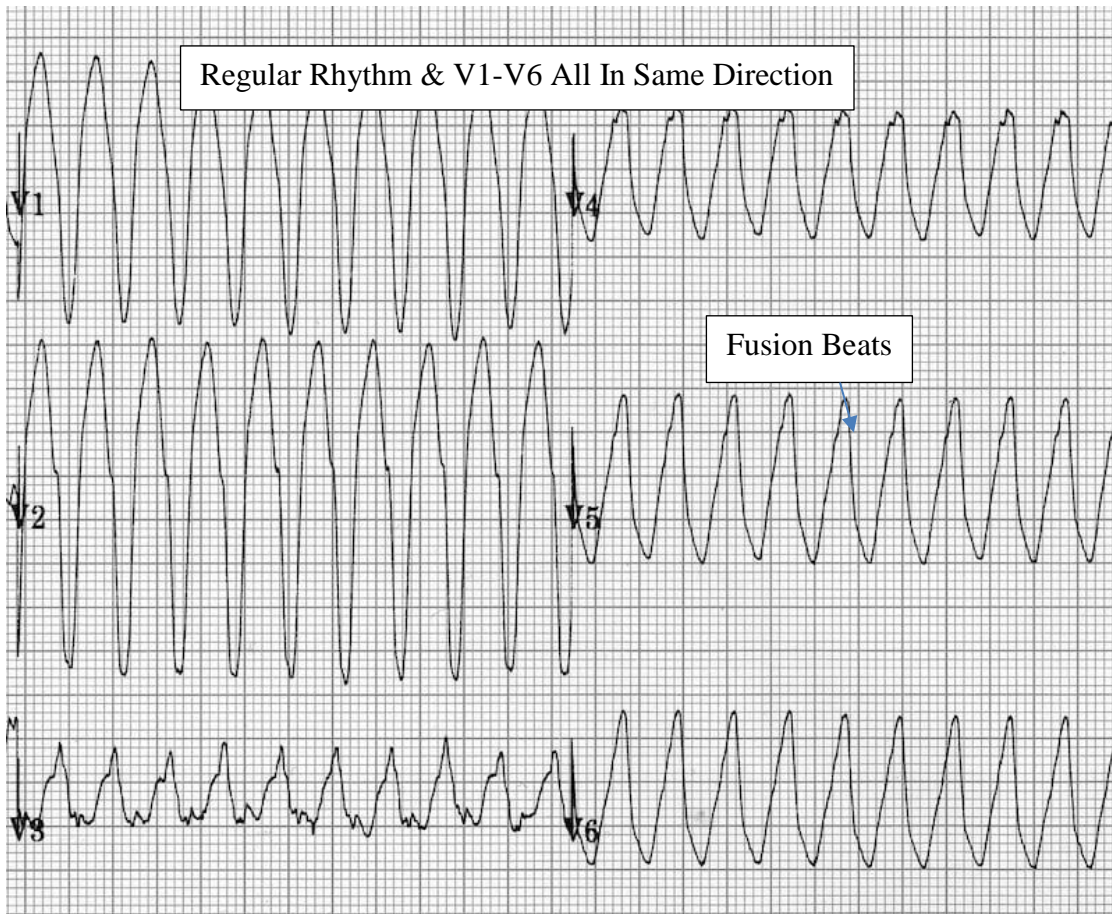
So, what are characteristics of Ventricular Tachycardia?

1. A wide complex greater than 0.12 milliseconds or 120 milliseconds
2. The rhythm is REGULAR
3. There is usually AV dissociation but occasionally a sinus complex breaks through.
4. There are fusion beats where the P wave and the QRS complex fuse (blend) into one.
5. Positive or negative concordance in leads V1-6, where the complexes are all negative or all positive
6. Josephson's sign or notching in the inferior leads, II, III or aVF, at the bottom of the S wave.

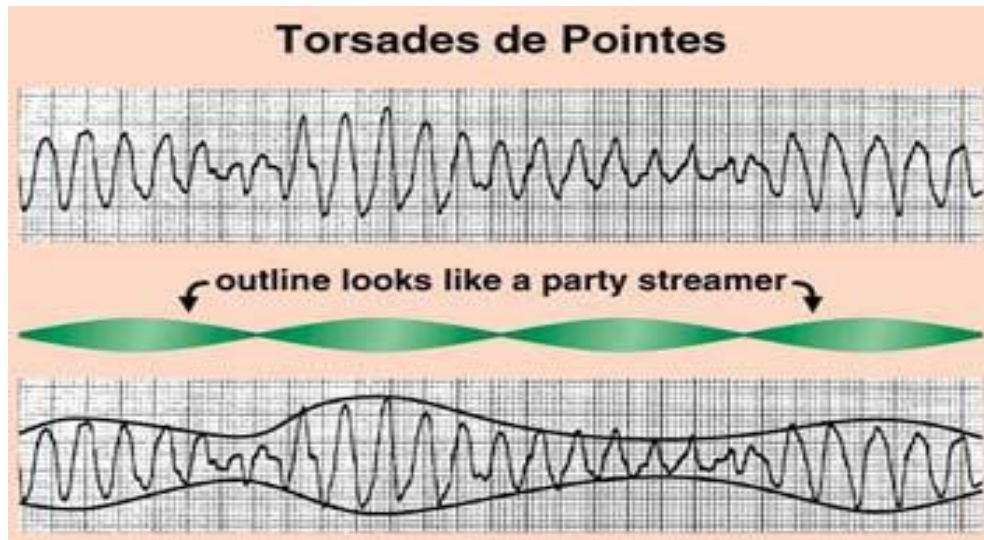
VENTRICULAR TACHYCARDIA



Josephson's sign – Notching near the nadir (bottom) of the S-wave in II, III or aVF.

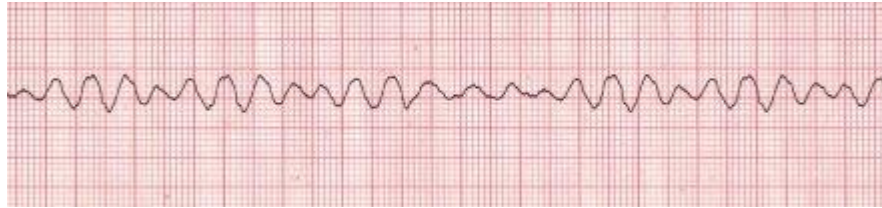


TORSADES de POINTES is a **VENTRICULAR TACHYCARDIA** that appears to twist on itself as if rotating on its axis. Torsades means twists in French. The identification of Torsades is necessary because the first line treatment is magnesium and not the other antiarrhythmic drugs.



VENTRICULAR FIBRILLATION is an irregular disorganized ventricular rhythm. The complexes do not allow for perfusion and the complexes are small, rapid and random. It is the final rhythm prior to asystole!

VENTRICULAR FILBILLATION



ACUTE MYOCARDIAL INFARCTIONS occur when the coronary arteries get obstructed or clotted off. Risk factors include age, gender, history of heart disease and co-morbid conditions such as diabetes. However, heart attacks do occur in younger people because of congenital anomalies and substance abuse especially with stimulants such as cocaine.

Two types of myocardial infarctions can be diagnosed with the assistance of an EKG; subendocardial and endocardial myocardial infarctions. The subendocardial infarction does not typically involve the whole wall of the heart but only a smaller partial segment. It usually represents occlusion of small branches of a coronary artery. The typical EKG finding is **J POINT ST DEPRESSION** in contiguous leads.

SUBENDOCARDIAL INFARCTION

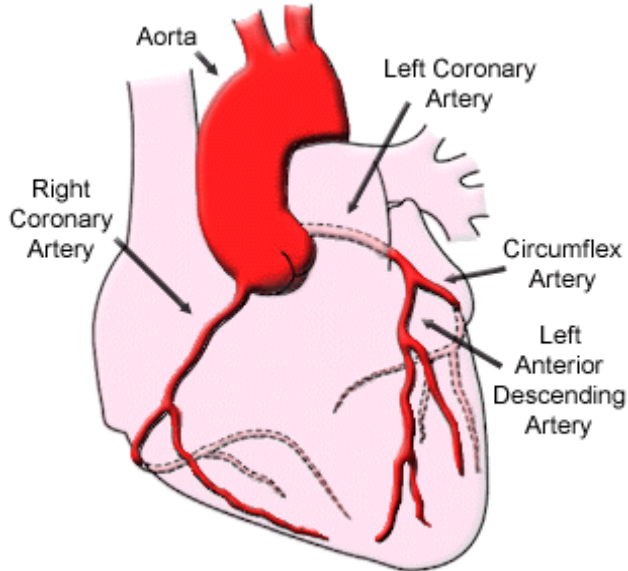


These occlusions are not amendable to cardiac catheterization and are not considered STEMI's.

Endocardial myocardial infarctions result in occlusion of one of the main arteries perfusing the heart. Blockage results in ischemia or lack of oxygen to the whole wall of the heart beyond the point of the blockage. EKG's reflect the ischemia by having ST elevation at the J point of at least two contiguous leads. The EKG can further delineate what part of the heart is involved by which leads are elevated. From this, it can be determined what artery is most likely occluded.

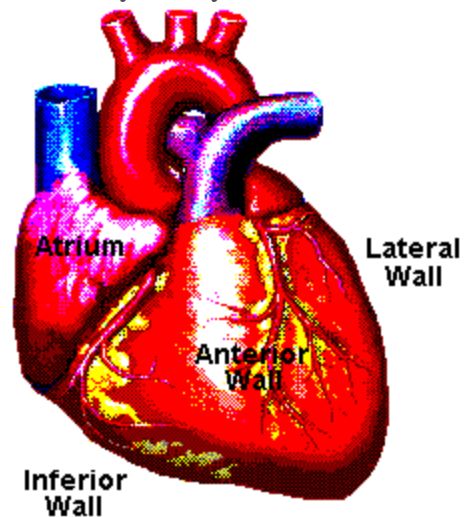
CARDIAC ANATOMY

The heart has several distinct vessels that are each associated with supplying different



parts of the heart. These vessels, also known as the coronary arteries, come off the aorta right at the beginning of the ascending aorta. The three main vessels are the Left Anterior Descending Artery and the Circumflex Artery which come off the Left Coronary Artery. The other main vessel is the Right Coronary Artery. Each of these

arteries supplies a different part of the heart. Depending on which artery is occluded, and where the occlusion occurs in the artery will determine the location and the extent of the heart attack. The pattern of ST Elevation on the EKG will allow you to determine the location of the heart attack and the vessels involved.

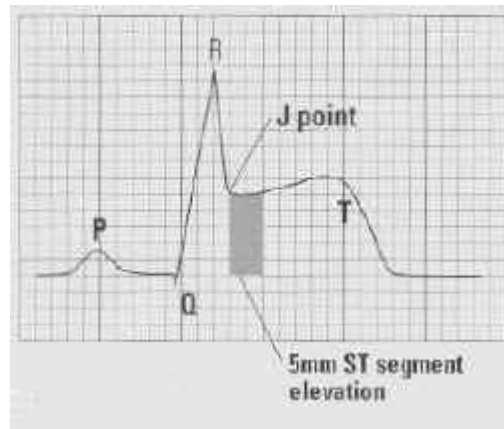
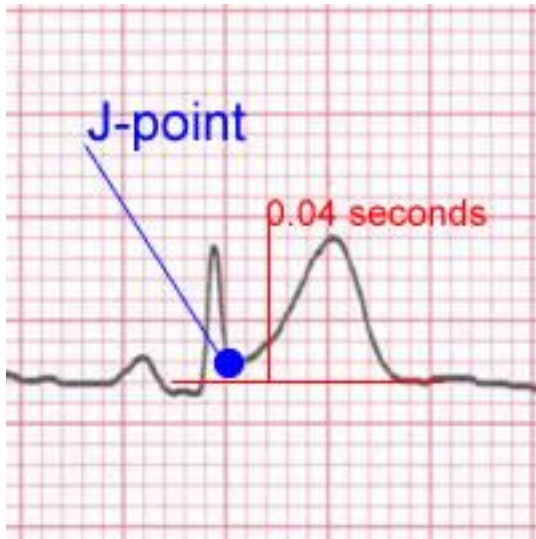


ISCHEMIA	LEADS	ARTERY	LOCATION
SEPTAL	V1	LEFT ANTERIOR DESCENDING	SEPTUM
ANTERIOR-SEPTAL	V1-V4	LEFT ANTERIOR DESCENDING	ANTERIOR WALL
ANTERIOR	V2-V4	LEFT ANTERIOR DESCENDING	ANTERIOR WALL
LATERAL	V5, V6, I, aVL	CIRCUMFLEX	LATERAL WALL
INFERIOR	II, III, aVF	RIGHT CORONARY 90% CIRCUMFLEX 10%	INFERIOR WALL
POSTERIOR	V2-V4	RIGHT CORONARY	POSTERIOR WALL

INTERPRETING AN ACUTE MYOCARDIAL INFARCTION

Determining a heart attack is based on injury pattern at the ST Segment. **ELEVATION OF MORE THEN ONE MILLIMETER OR ONE SMALL BOX IN TWO**

CONTIGUOUS LEADS IS CONSISTENT WITH ST ELEVATION AND AN ACUTE MYOCARDIAL INFARCTION aka HEART ATTACK. Elevation is measured at the J Point which is where the S wave ends, and the ST Segment begins. The injury pattern is determined by looking for **J POINT ELEVATION** which is seen by going over horizontally one small box or 0.04 seconds. When there is elevation or depression of 0.1 milivolts or more, (one or more small boxes) an injury pattern exists.



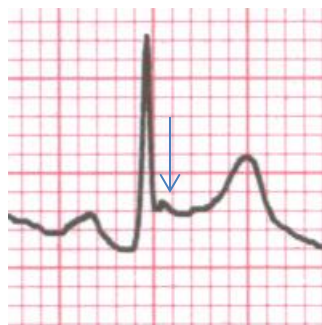
Attached is a video link for understanding and diagnosing Acute MI's

<https://www.youtube.com/watch?v=uqNQvKxAiuo>

MIMICKERS: There are EKG changes which can mimic an acute myocardial infarction; in particular, pericarditis and early repolarization.



ACTUE MI: At the J point, going over one small box the ST Segment is upward sloping indicative of an Acute Myocardial Infarction.



PERICARDITIS: At the J point, ST Segment is upward sloping and appears to be indicative of an Acute MI. However, note how the PR segment is downward sloping which is seen in pericarditis. There is also the fishhook sign (arrow)

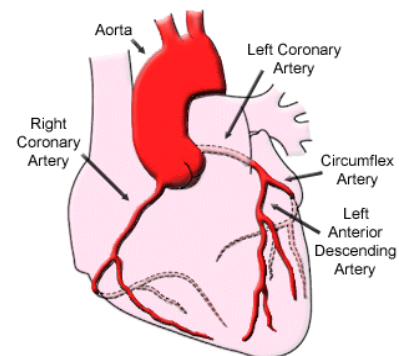


EARLY REPOLOARIZATION: At the J point, going over one small box the ST Segment is downward sloping indicative of early repolarization. DON'T FORGET THE "HAPPY FACE"

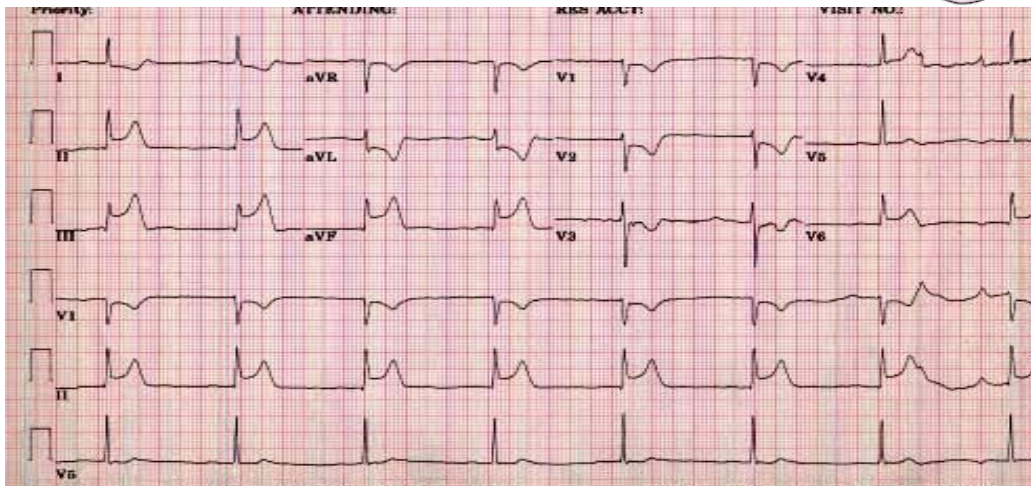


INFERIOR WALL MYOCARDIAL INFARCTION

- ST elevation in II, III, AND aVF
- 90% due to blockage of right coronary artery and 10% due to blockage in the circumflex artery.
- Reciprocal changes seen anteriorly

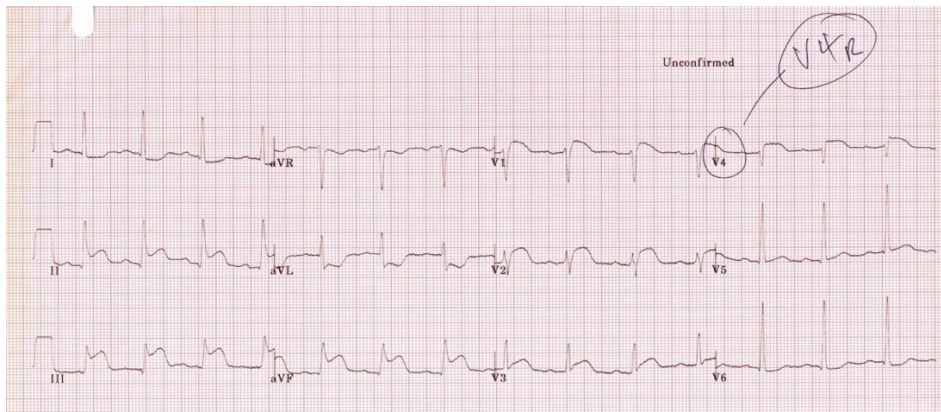


INFERIOR WALL MI



Inferior wall MI's may result in secondary complications. Swelling can occur around the AV node and cause a 3rd degree block which is usually temporary but can result in the need for a temporary pacemaker. Another complication is an extension of the heart attack to the right ventricle. The right ventricle is a low flow system and very susceptible to vasodilatation caused by medications such as nitroglycerine. The **RIGHT VENTRICULAR INFARCTION** is diagnosed by using right sided leads and looking for ST elevation in V4.

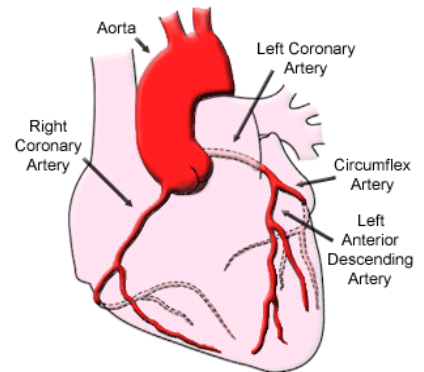
RIGHT VENTRICULAR INFARCTION



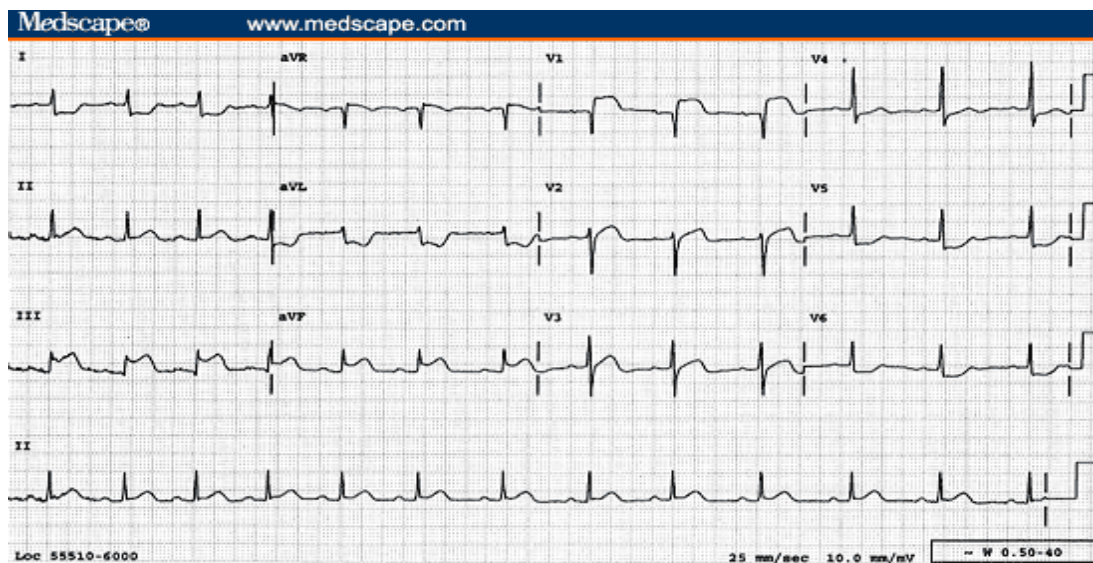
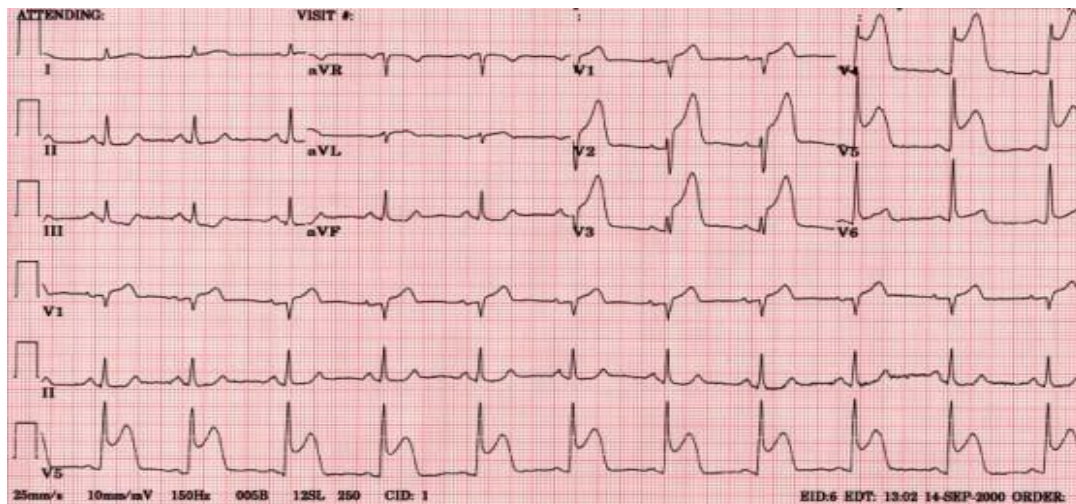
ANTERIOR WALL MYOCARDIAL INFARCTION

- ST elevation in V2-V4
- V1 elevation reflects septal involvement.
- V5-V6 with I and aVL indicates lateral involvement
- Blockage occurs in the left anterior descending artery
- Reciprocal changes seen inferiorly

Septal involvement can result to loss of blood supply to the AV node. In this case a third-degree block may occur that is permanent. Typically, there is not a pure Septal, Anterior or Lateral MI. There is usually an overlap between the regions due to the shared blood supply prior to the bifurcation of the Left Coronary Artery.

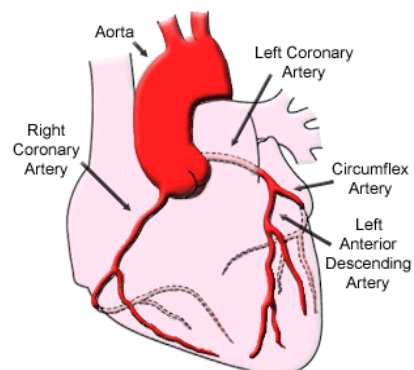


ANTERIOR SEPTAL MYOCARDIAL INFARCTION



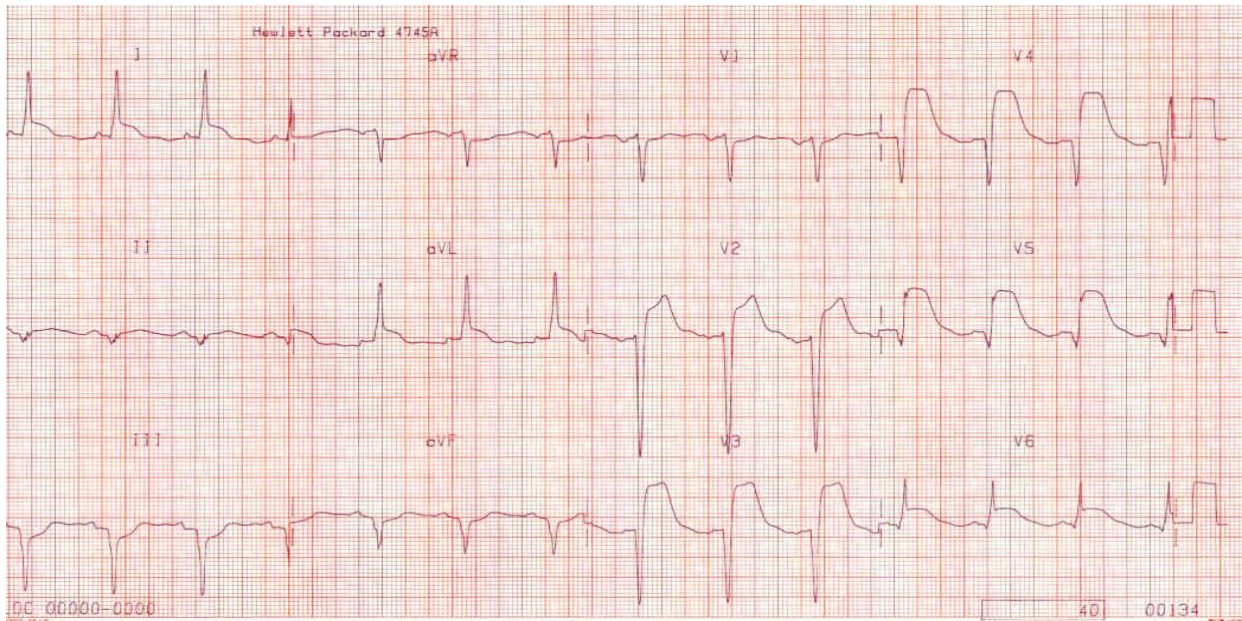
ANTERIOR-LATERAL WALL MYOCARDIAL INFARCTION

- Occlusion of lad or circumflex
- St elevation in V2-V6, I AND aVL
- Reciprocal changes inferiorly



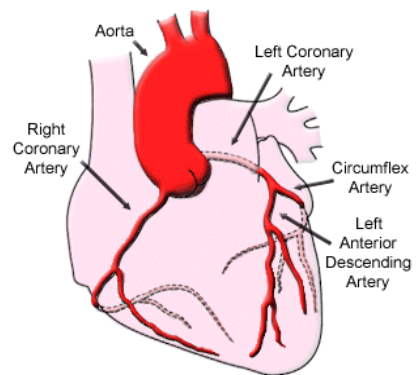
arteries.

ANTERIOR-LATERAL WALL MYOCARDIAL INFARCTION



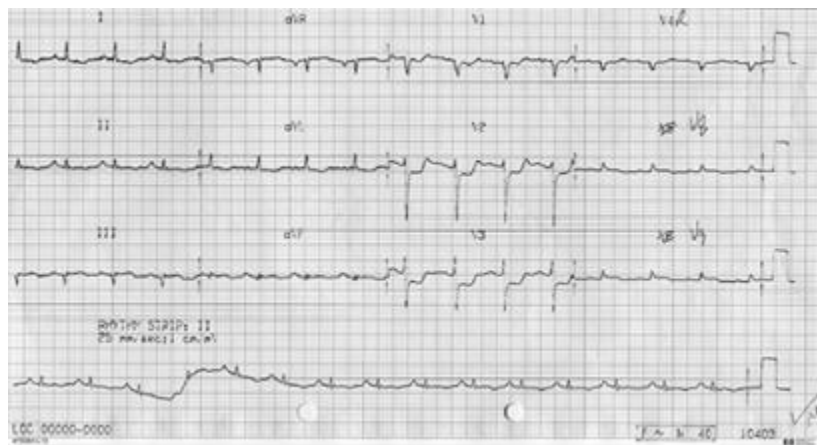
LATERAL MYOCARDIAL INFARCTION

- Occlusion of circumflex artery
- ST elevation in V5, V6, I and aVL
- Reciprocal changes inferiorly

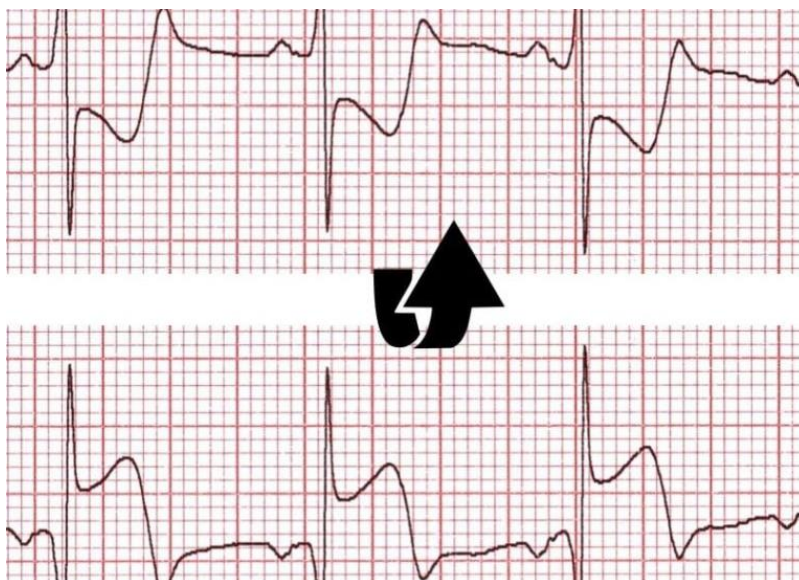


POSTERIOR WALL MYOCARDIAL INFARCTION

- Occlusion of the Right Coronary Artery
- Involves V2-V4
- Since it is on the opposite side of an anterior myocardial infarction, the leads are a mirror image of what is found with an acute anterior myocardial infarction.
- There is a large R wave associated with ST depression.
- Oftentimes associated with inferior and lateral myocardial infarctions since the occlusion can share the same blood supply.



If you were to flip the EKG upside down and backward, you would see ST elevation.



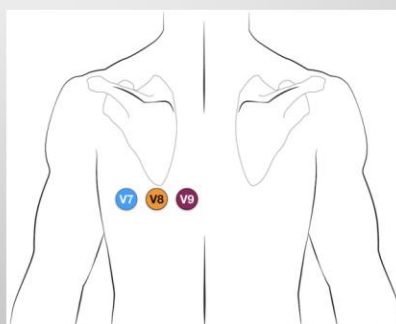
This technique is effective for a quick “look see” but is not as sensitive as checking posterior leads. V7-9 are the posterior leads but require placing the leads on the back which can potentially be cumbersome and time consuming. Checking these leads should not delay transport. If you suspect a posterior MI, call the STEMI! REMEMBER that posterior MI’s only require 0.5 mm of ST elevation.

POSTERIOR LEADS

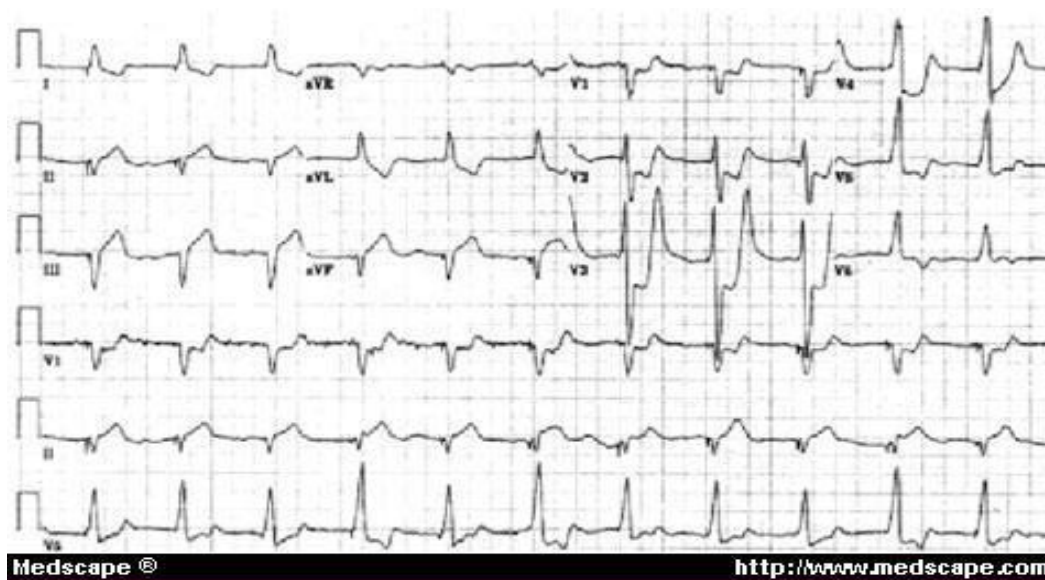
Leads V7-9 are placed on the posterior chest wall in the following positions (see diagram below):

- V7 – Left posterior axillary line, in the same horizontal plane as V6.
- V8 – Tip of the left scapula, in the same horizontal plane as V6.
- V9 – Left paraspinal region, in the same horizontal plane as V6.

*The degree of ST elevation seen in V7-9 is typically modest – note that only **0.5 mm of ST elevation** is required to make the diagnosis of posterior MI!*



INFERIOR-POSTERIOR MYOCARDIAL INFARCTION

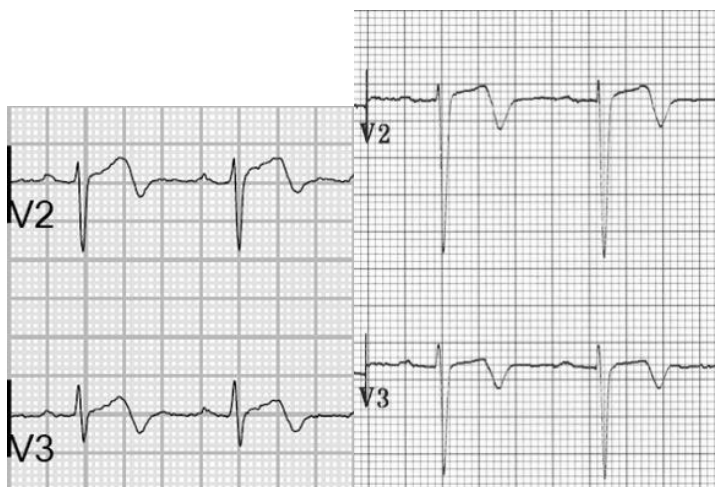


SPECIAL CONSIDERATIONS

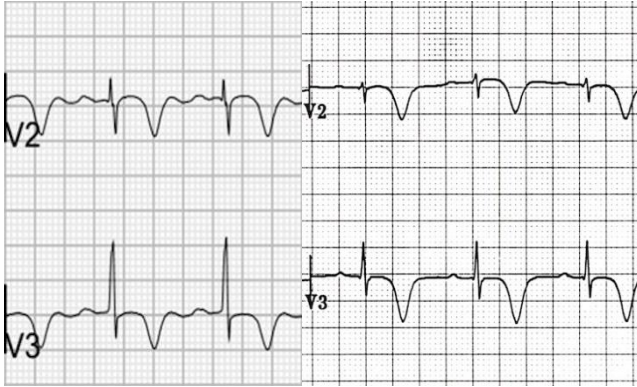
WELLEN'S SYNDROME: This is an electrical manifestation seen in leads V2 & V3 where there are deeply inverted or biphasic T-waves. This finding is highly specific for a critical stenosis or narrowing of the Left Anterior Descending Artery.

Typically, the patient will be pain free. However, if still actively having chest pain this finding should be treated like a STEMI!

WELLEN'S TYPE A: BIPHASIC T-WAVES



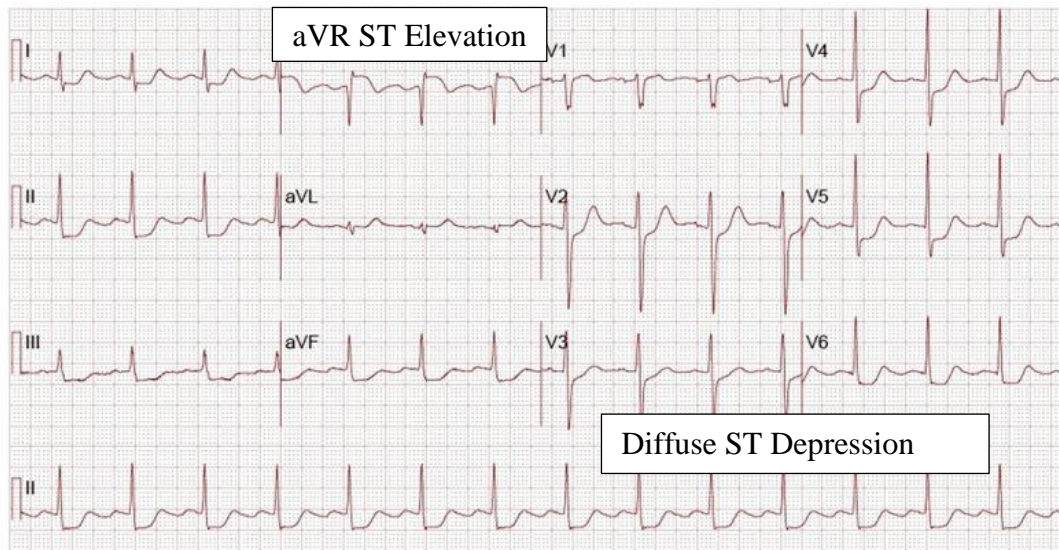
WELLEN'S TYPE B: INVERTED T-WAVES



LEFT MAIN CORONARY ARTERY OCCLUSION

LMCA

Left Main Coronary Artery Occlusion is obviously a lethal obstruction if left untreated. It essentially would take out the whole anterior and lateral sides of the heart and should quickly lead to cardiogenic shock and death in most cases. **At times narrowing or stenosis of the left main can be ascertained when there is greater than one millimeter of ST elevation in aVR along with ST depression in the anterior lateral leads.** There is depression of the vessels due to partial flow through the left main preventing ST elevation. An alternative diagnosis to this is 3 vessel disease resulting in the diffuse ST depression



LEFT BUNDLE BRANCH BLOCKS AND PACED RHYTHMS

For years the teaching has been that a LBBB and paced rhythm prevented the interpretation of an acute MI. Studies also showed that those who had these findings had worse outcomes in the setting of an acute MI.

National Registry of Myocardial Infarction 2

- Right bundle branch block was present in approximately 6 percent.
- Left bundle branch block in 7 percent of infarctions

Outcomes and treatment were also affected.

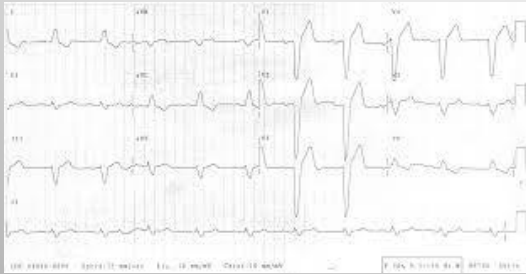
- Patients with bundle branch block were significantly less likely to receive
 - Aspirin
 - Beta blockers.
- Had more co-morbid disease.
- Had a significant increase of in-hospital mortality.

Studies of EKG's did find that in the setting of an acute MI, there were potential changes that could reflect acute changes that would warrant the activation of the cardiac catheterization lab. SGARBOSSA criteria are changes seen in LBBB's and VENTRICULAR paced rhythms that reflect acute finding and blockage of the coronary vessels.

In both LBBB and Ventricular Paced Rhythm, there is a natural discordance. This means if the R wave is up the S wave is down and vice versa. In the setting of an acute MI, this discordance changes and the ST segment will move causing some concordance, which means the R wave or the S wave moves toward the other side. This follows in two of the three criteria while the third criteria is seen when there is ST segment of 5 mm or more in the discordant

LBBS & MI

LBBS

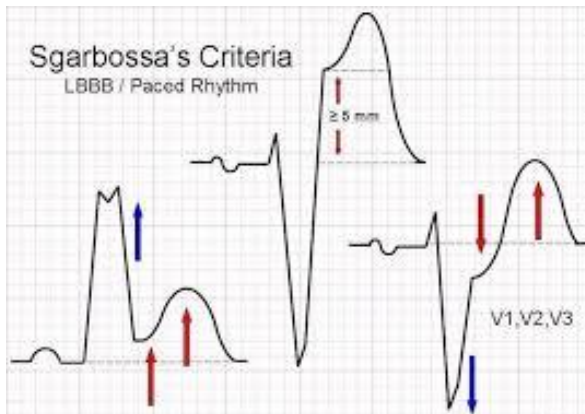


SGARBOSSA'S CRITERIA



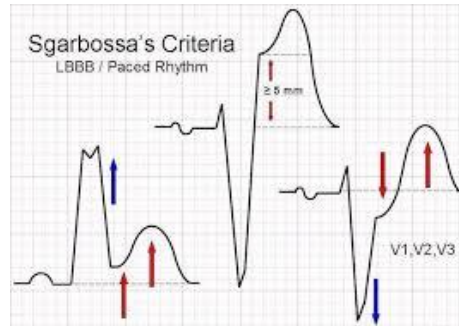
The criteria actually give a score depending on the type of change with a score of 3 or more having a high probability of an acute MI. For our purposes, any of the changes should be called in as a STEMI.

SGARBOSSA CRITERIA

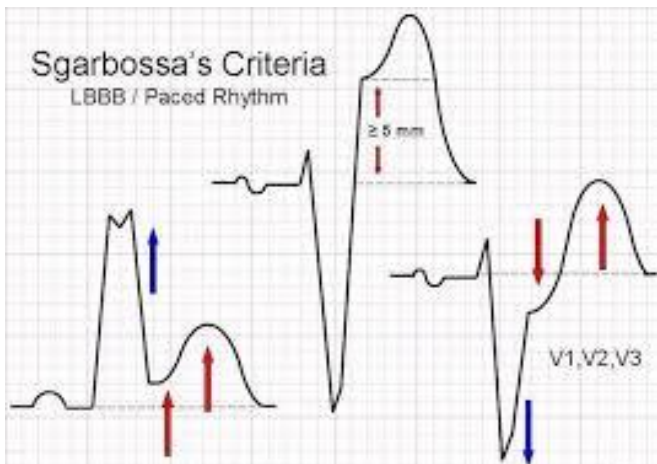


- ST segment elevation of 1 mm or more that is in the same direction (concordant) as the QRS complex in any lead: score 5.

ST segment depression of 1 mm or more in any lead from V1 to V3: score 3



SGARBOSSA CRITERIA



- ST segment elevation of 5 mm or more that is discordant with the QRS complex (ie, associated with a QS or rS complex): score 2.
- 3 or greater suggestive of MI

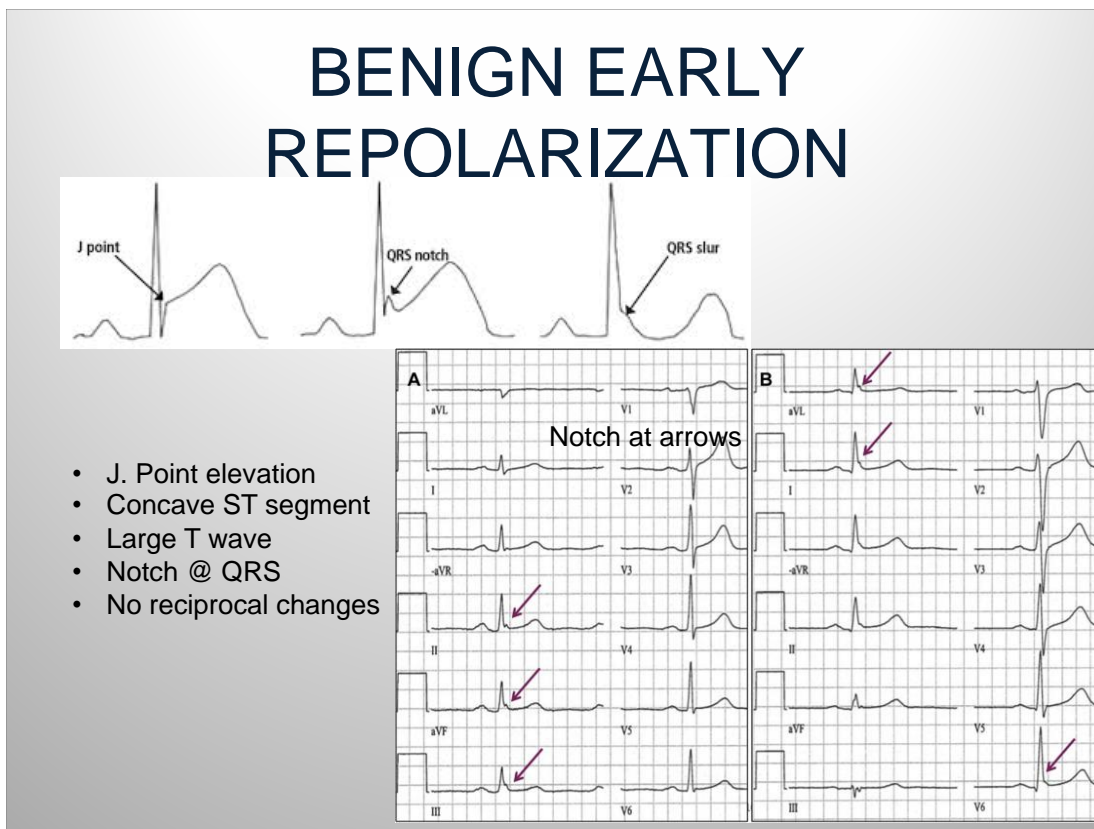
MI MIMICKERS

There are two common mimickers of acute MI's: Early Repolarization and Pericarditis. Both can have changes that look like MI's but also have distinct characteristics that help exclude them.

Benign Early Repolarization

Early Repolarization presents as J point elevation that at first glance can look like an acute MI. However it has several characteristic that help exclude it.

- Appears as ST elevation since the ST interval does not fall to baseline
- Has a concave shape AKA “happy face”.
- Usually in young people and does not typically occur in those over 50 years old.
- Won't see reciprocal changes.
- They don't look like they are having a heart attack.
- May have a notch at the QRS.



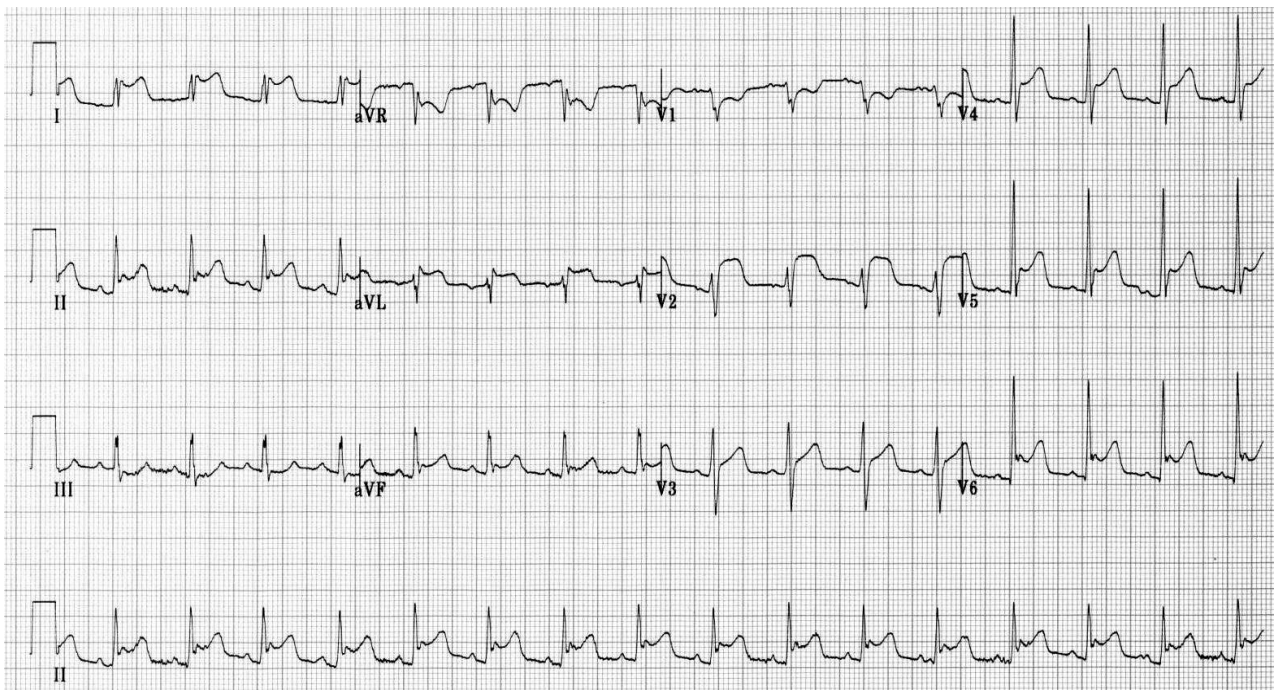
Video explaining repolarization: <https://www.youtube.com/watch?v=fLbcp3jF764>

PERICARDITIS

Pericarditis is also an imitator of an acute myocardial infarction. Because there is inflammation to the heart lining; the pericardium, the person will complain of chest pain. It often improves when leaning forward and may have a rub. The EKG also may have diffuse elevation which does not follow the anatomy of the coronary arteries and the associated findings on the EKG. PR depression is oftentimes seen as well.

- Chest pain is often retrosternal in nature, pleuritic, and positional (relieved by sitting forward, worse lying flat)
- There may be an associated pericardial friction rub, or [ECG evidence of a pericardial effusion](#)
- Widespread ST segment changes occur due to involvement of the underlying epicardium (i.e. myopericarditis)
- Widespread concave ST elevation and PR depression throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6)
- Reciprocal ST depression and PR elevation in lead aVR (\pm V1)
- Sinus tachycardia is also common in acute pericarditis due to pain and/or pericardial effusion

PERICARDITIS



Note the diffuse ST elevation that encompasses nearly every lead as well as the down sloping PR intervals along with the ST depression in aVR

MANAGING THE ACUTE MYOCARDIAL INFARCTION

The management of MI's begins in the field. Making the diagnosis and alerting the hospital allows for rapid deployment of the Cardiac Catheterization Team. The quicker

the clot is removed; the more heart muscle is saved. Oftentimes, rapid deployment results in virtually no damage to the heart muscle.

Medications are also available which can benefit the patient. Early administration of aspirin has proven to aid in the advantageous outcome of heart attacks. The administration of nitroglycerin along with analgesics is within the scope of a paramedics practice. With the advent of emergent catheterizations, the utility of nitroglycerin has changed. In the past, it was used to vasodilate and potentially release the clot or allow blood flow around the narrowing. It vasodilates all vessels and has the potential of causing hypotension. With a heart that cannot pump efficiently, the hypotension could become catastrophic. Morphine as well can cause hypotension. It definitely reduces pain which in turn can help reduce the overall oxygen demands on the heart. Fentanyl on the other hand does not cause hypotension but does reduce the pain associated with a heart attack. In the end, with the ability of emergently catheterizing the patient and removing the blockage in a timely fashion, the use of any medication that causes hypotension should be administered with the appropriate caution. Always remember the first rule of medicine..."do no harm"