

**Multi-Lead Medics™
12 Lead ECG for Acute Care Providers**

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Learning Objectives

At the completion of this lesson, the participant should be able to:

- ♥ Describe the difference between monitoring and assessing a patient using an ECG machine.
 - ♥ Demonstrate proper lead placement for a 12 and 15 lead ECG.
 - ♥ Using a simple chart and leads I, II, III, determine electrical axis and the presence of fascicular blocks (hemiblocks).
 - ♥ Using lead V1 (MCL-1), determine bundle branch blocks.
 - ♥ Describe the clinical significance of hemiblocks and bundle branch blocks in the cardiac patient.
 - ♥ Describe a strategy for identifying V-Tach in wide complex tachycardia.
 - ♥ Describe the ECG criteria for W.P.W. syndrome
 - ♥ On a 12, 15 lead, or Multi-Lead ECG, identify ST and T wave changes relative to the triage of Acute Coronary Syndromes.
 - ♥ Describe a systematic “assessment” of a 12 and 15 lead ECG.
 - ♥ Describe possible complications of various infarct locations.
-

The Need for Multi-Lead

- **Single Lead Dependence**
- Gives only one look at the heart
- Misses vital information
- Drug contraindications
- The lead II paradigm



Lead Placement

Lead Placement really does depend on the reason that you are “monitoring” the patient: V-Fib, Asystole are two common arrhythmias that we watch out for.

The purpose of monitoring is to pick up a change in the patient’s heart rhythm. If something changes, you would immediately **assess** the situation.

If this is the purpose of the monitor, then it does not matter where you put the electrodes...be creative, stick one on the nose and one on the toes...VF and asystole will look the same!!

Assessing the Patient

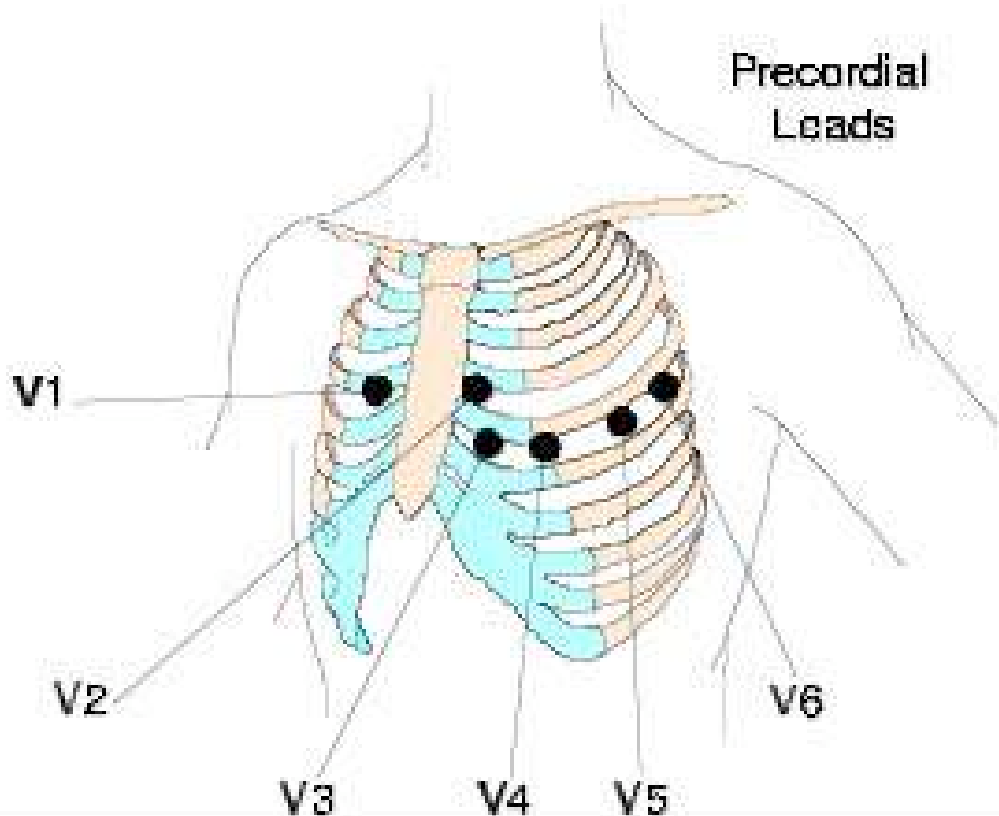
A cardiac monitor will show changes in electrical activity. These changes have been ignored because of our Lead II paradigm. That is, we have been taught that lead II will tell us all we need to know. There is much useful and potentially life saving information that can be gained from a multi-lead approach.

In the course of this session you will learn and yes, actually discover axis and hemiblocks, bundle branch blocks, VT, and MI’s. However, in order to use this information, **proper lead placement is absolutely crucial!**

**Standard
Limb Leads**



**Precordial
Leads**



Limb Leads on the limbs!

Leads I, II, III (limb leads)

Most modern 12 lead machines have the lead location stamped on the electrodes.

- ♥ RA - white electrode - means Right ARM
- ♥ LA - black electrode - means Left ARM
- ♥ LL - red electrode - means Left LEG
- ♥ RL - green electrode (LP11) means Right LEG

So why do so many people put the electrodes on the chest? This is an example of another paradigm or “the way we’ve always done it!” In all fairness, the leads were moved into the chest to reduce artifact in the hospital setting where remote monitoring is allowed. However, by placing the electrodes on the chest, you can only **monitor** the rhythm. In order to **assess** for the patient axis, hemiblocks and bundle branch blocks, you must have the leads on the LIMBS! Oh, by the way, if artifact is a problem, have the patient lie still while you get the ECG!

The 12 Lead ECG only uses 10 electrodes.

Limb leads:

Leads I, II, III (standard bipolar limb leads)

Augmented (unipolar limb leads) **AVF, AVL, and AVR.**

Precordial Leads (voltage leads)

V1, - 4th IC space, right side of sternum

V2, - 4th IC space, left side of sternum

V3, - between V4 and V2

V4, - 5th IC space, mid clavicular line

V5, - 5th IC space, anterior axillary line

V6, - 5th IC space, mid axillary line

V4, 5, 6, should be in a straight line

15 Lead ECG

V4R - 5th IC space, mid clavicular line right side

V8 - 5th IC space mid scapular **V9** - 5th IC space between V8 and spine

Run 12 Lead as usual but on the second 12 lead, Use leads V4 for V4R, V5 for V8, and V6 for V9.

Patient position and skin preparation

Supine patient is recommended.

Can run in Fowler’s position.

Clip or shave away chest hair where necessary.

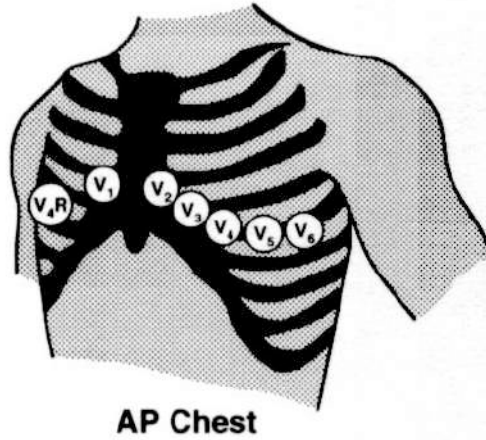
Wipe diaphoretic skin with a towel.

Skin prep tape is also very helpful

Consider benzoin spray, alcohol, or betadine prep

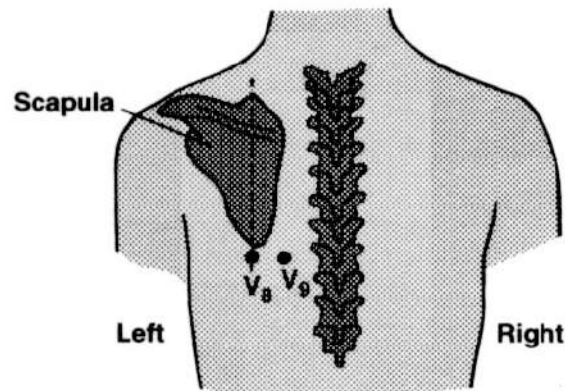
V₄R Lead Placement

Anteroposterior Chest

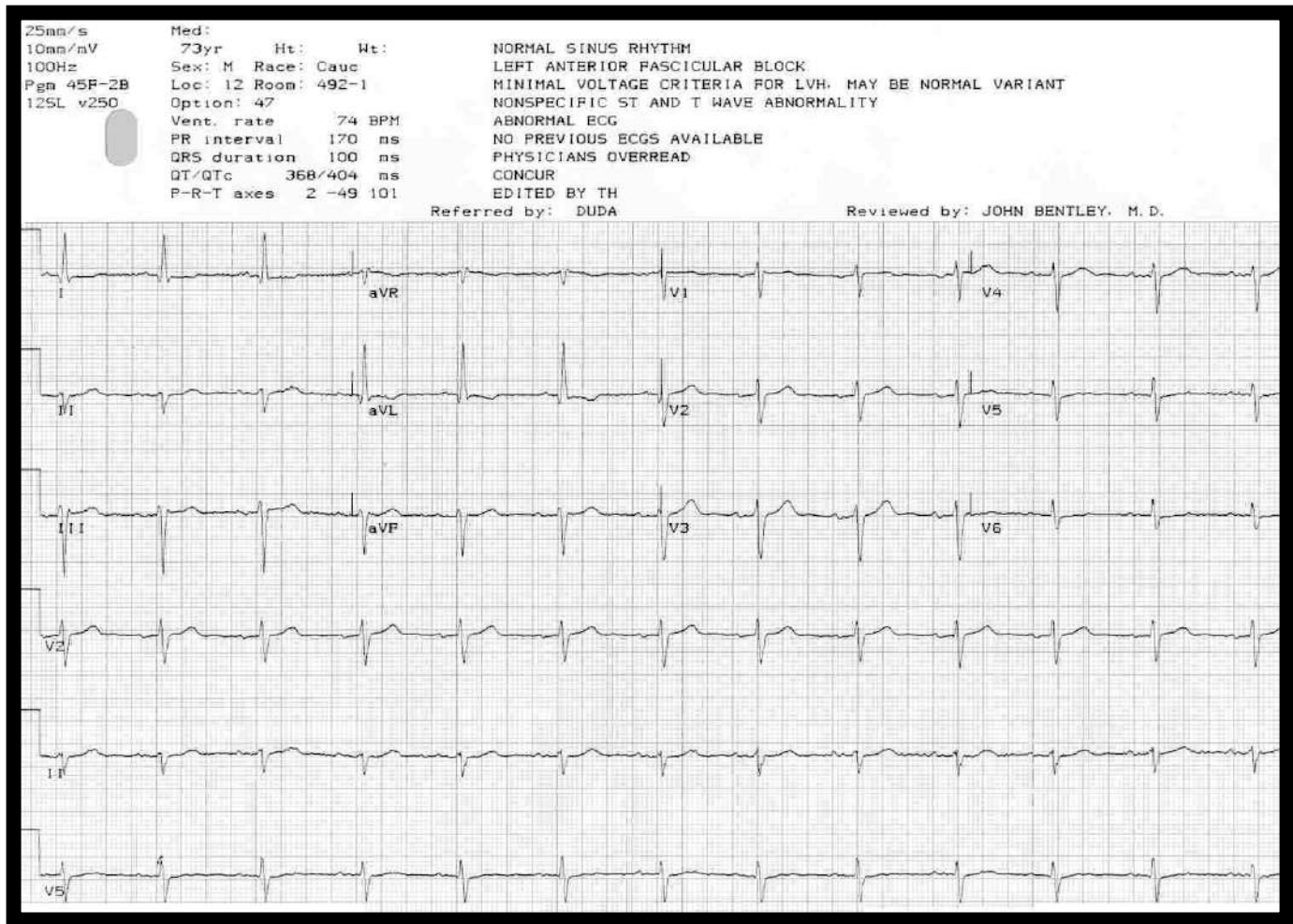


V₈ and V₉ Lead Placement

Posteroanterior Chest



A 15 Lead ECG should be performed on all patients with a normal 12 lead ECG and those with a strong suspicion of having posterior or right ventricular involvement.

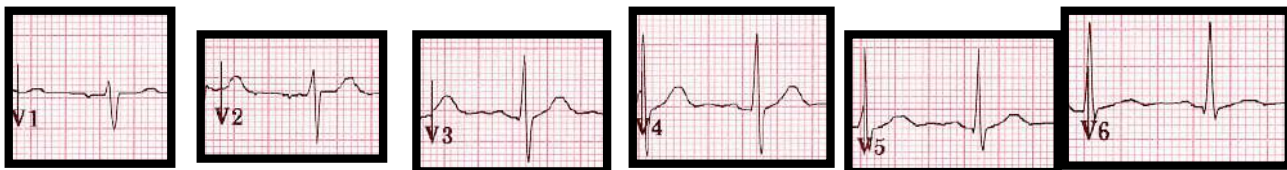


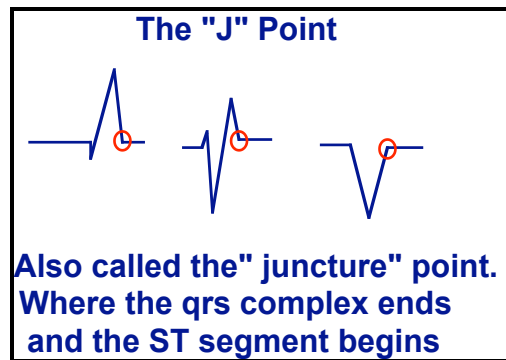
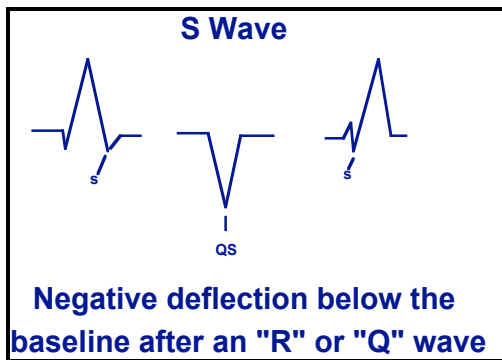
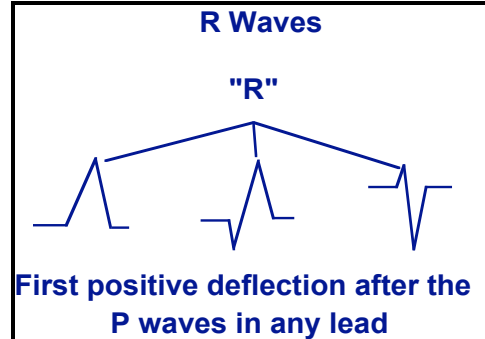
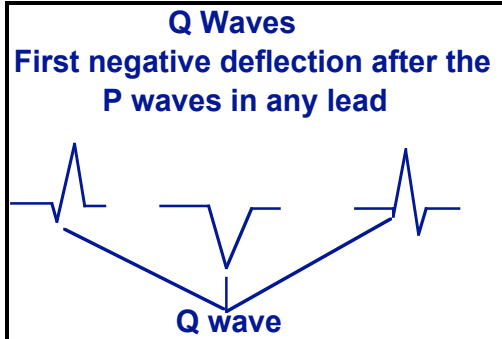
How do you know if you got the leads in the right place? 12 Lead Validation



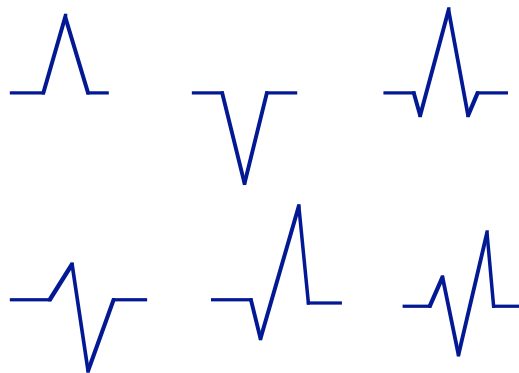
Lead I shows **Global Negativity?**
(P, QRS, and T wave inverted)
RA and LA are switched.

In the pre-cordial leads (V Leads) we look for "**R Wave Progression**" to confirm proper lead placement. Notice how the size of the r wave increases in size until between leads V3 and V4, the "QRS" goes from one that is negative to one that is positive



Review of QRS Labeling

QRS Morphologies
Can you label these complexes?

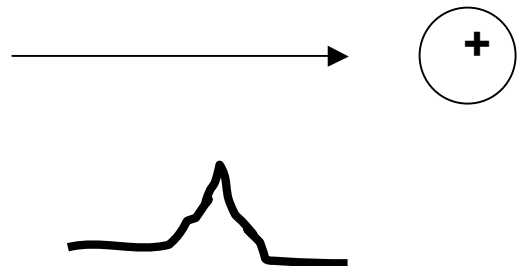
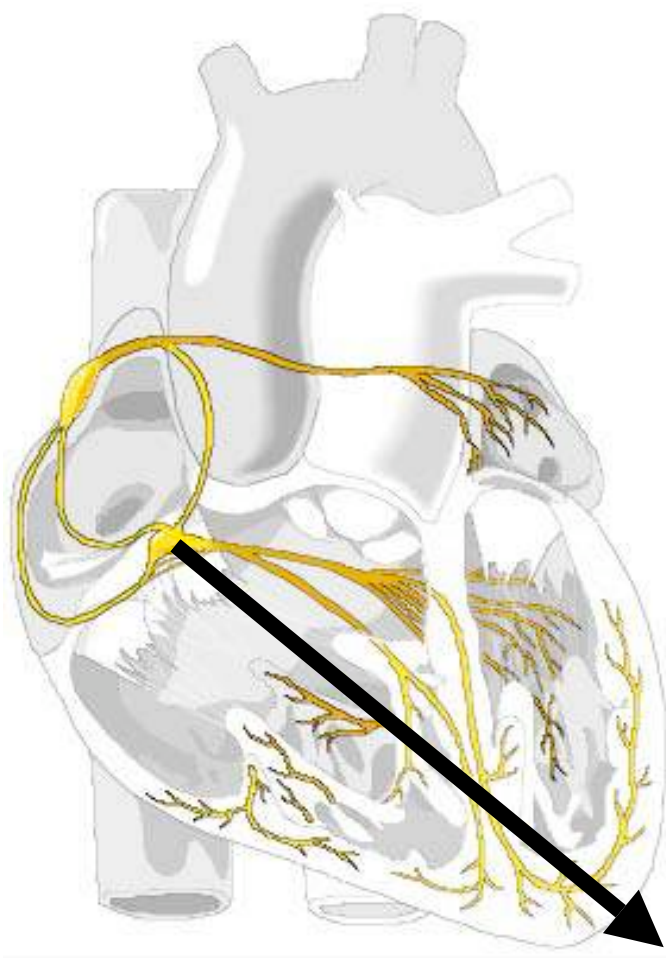


Rapid Axis and Hemiblock Determination

Axis is the predominant direction of flow of the impulses in the heart.

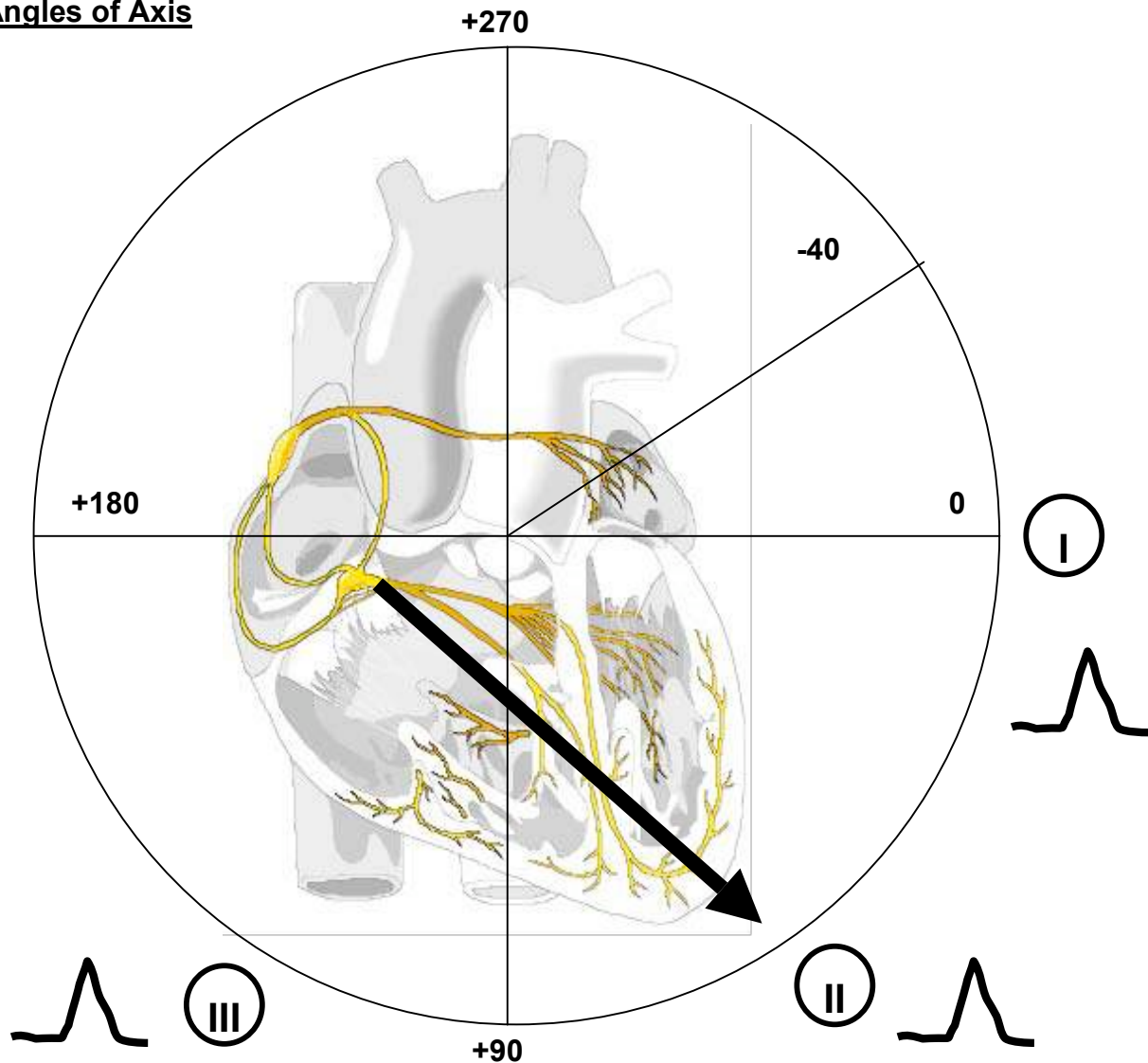
Axis can be significant:

- It can help you diagnose V-Tach
- Used to diagnose hemiblocks
- It can help you identify which patients are at high risk for complete heart block or for becoming hemodynamically unstable.

**Basic Concept of Electricity and ECG Signal**

As an impulse moves towards a positive electrode, it makes a positive deflection on the ECG

Normal direction (vector) of electricity in the heart. Although electricity moves in many different directions as it depolarizes the heart, it moves in one general direction. This is called **AXIS!**

The Angles of Axis

It would be nice to be able to “see” the “arrow” on the chest of a patient that indicated the direction of travel. Well, we may not be able to see the direction, but we can track it! Using the basic concept of electrical signals from page 8, we can place three leads around the heart. Limb leads, I, II, and III are shown here. The position represents the relative location of the positive electrode in each of those three leads. Each location will show us by the net deflection of the QRS complex whether the impulse is coming towards or going away from its respective location!
















In this example, Lead one is on the left side of the heart and looks at the entire left side, so if the impulse is traveling towards the left side, it will show a positive (UP!) deflection. Lead II see the impulse coming straight at it so it will show positive (UP!) also. Lead III still shows it coming in its general direction so we would see a positive (UP) deflection as well.

This finding, **Up** in leads I, II, and III are considered a NORMAL finding in Adults.

How to Determine Axis:

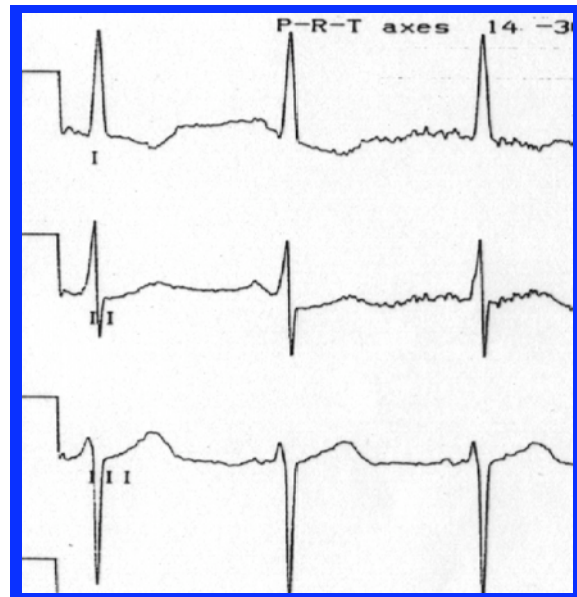
1. Run leads I, II, III. (make sure your leads are correctly placed)
2. Determine the mean QRS deflection in each lead (up or down?)
3. Compare it to the chart below and presto - instant axis!

Rapid Axis and Hemiblock Chart

Axis	Lead I	Lead II	Lead III	Comments
Normal Axis 0 - 90				
Physiologic Left Axis 0 to -40				
Pathological Left Axis -40 to -90				Anterior Hemiblock
Right Axis 90 - 180				Posterior Hemiblock
Extreme Right Axis no man's land				Ventricular in origin

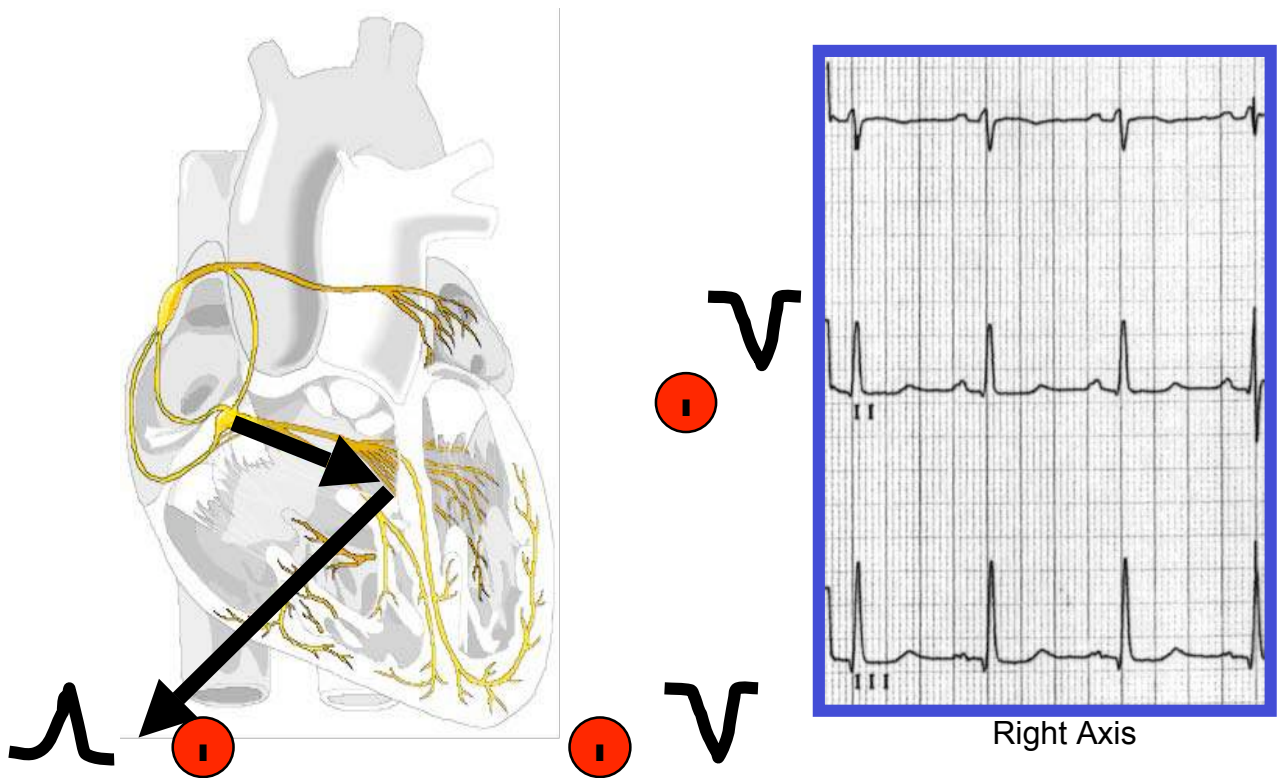
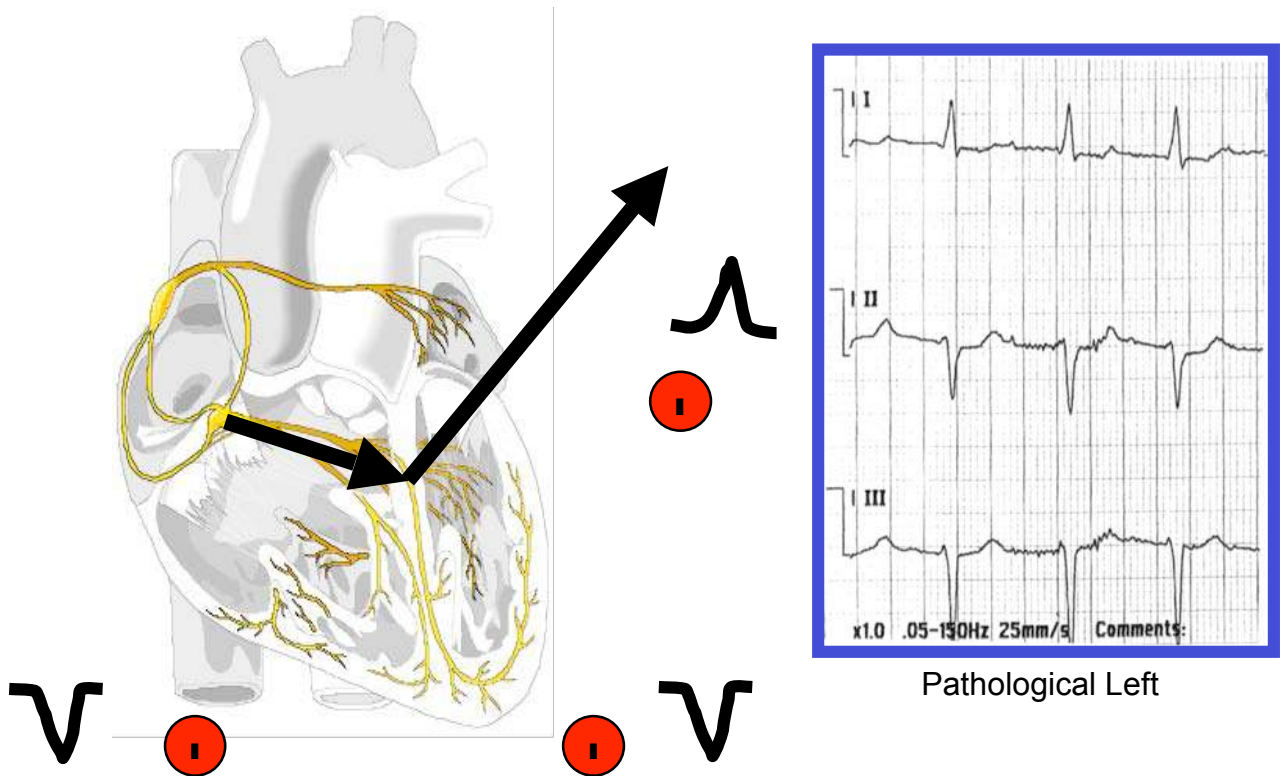
Normal Axis Variants

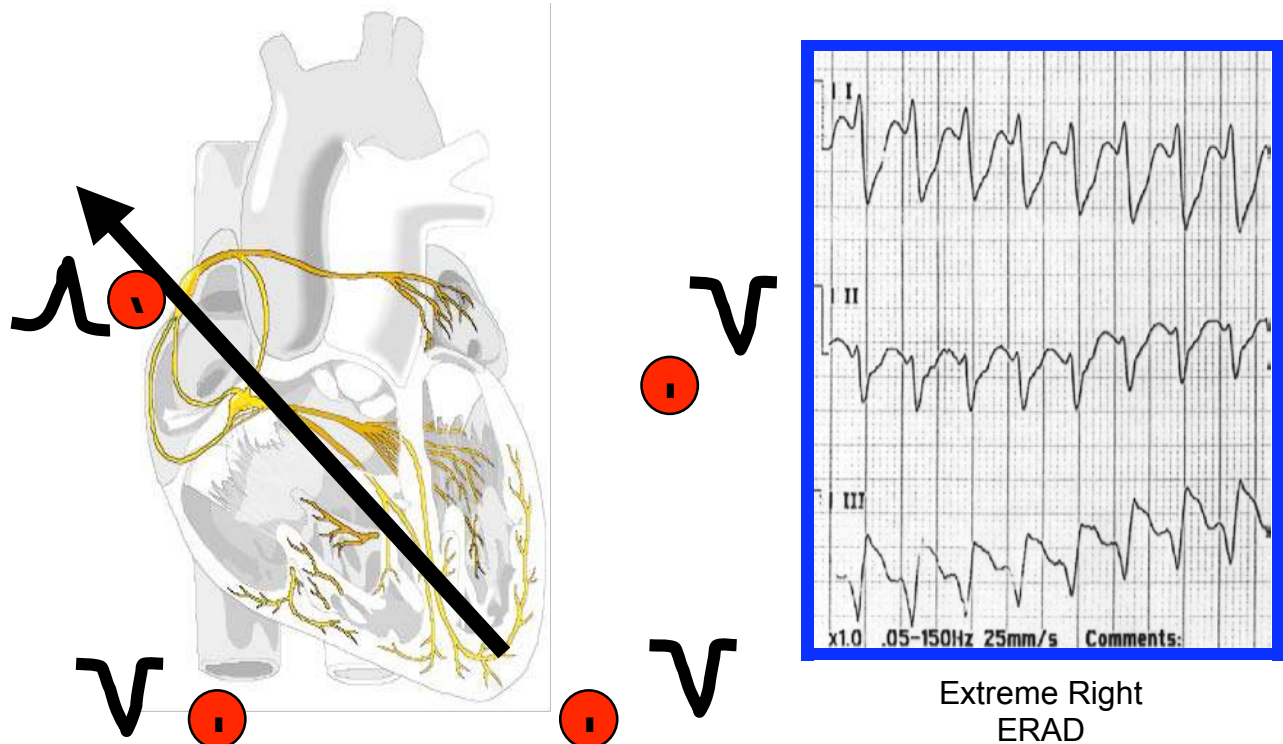
NORMAL AXIS



Physiological Left

Pathological Axis

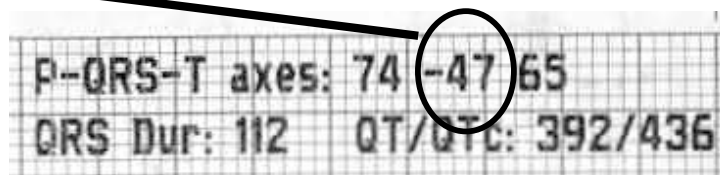


Pathological AxisAnother Easier Way

NEMO, A professional wrestler who is also a paramedic from South Carolina once quoted Jesse Ventura to me in a class by saying..."Win if you can, Lose if you must, but always, always CHEAT!" Some would think that using a chart or what is given without "working for it" as cheating. But hey, c'mon: This is life, whom are you going to cheat? The grim reaper?

Axis	
Normal Axis	0 - 90
Physiologic Left Axis	0 to -40
Pathological Left Axis	-40 to -90
Right Axis	90 - 180
Extreme Right Axis	no man's land

If you have a machine that gives you the axis information recognized as the "R" axis or QRS axis that is expressed as a number, use it! Simply look at the number and compare it to the range of axis found on your **Rapid Axis and Hemiblock Chart** and you can call the axis that way. You will find this will speed up your axis assessment, especially when there is a wide complex tachycardia when up and down are not so clear.

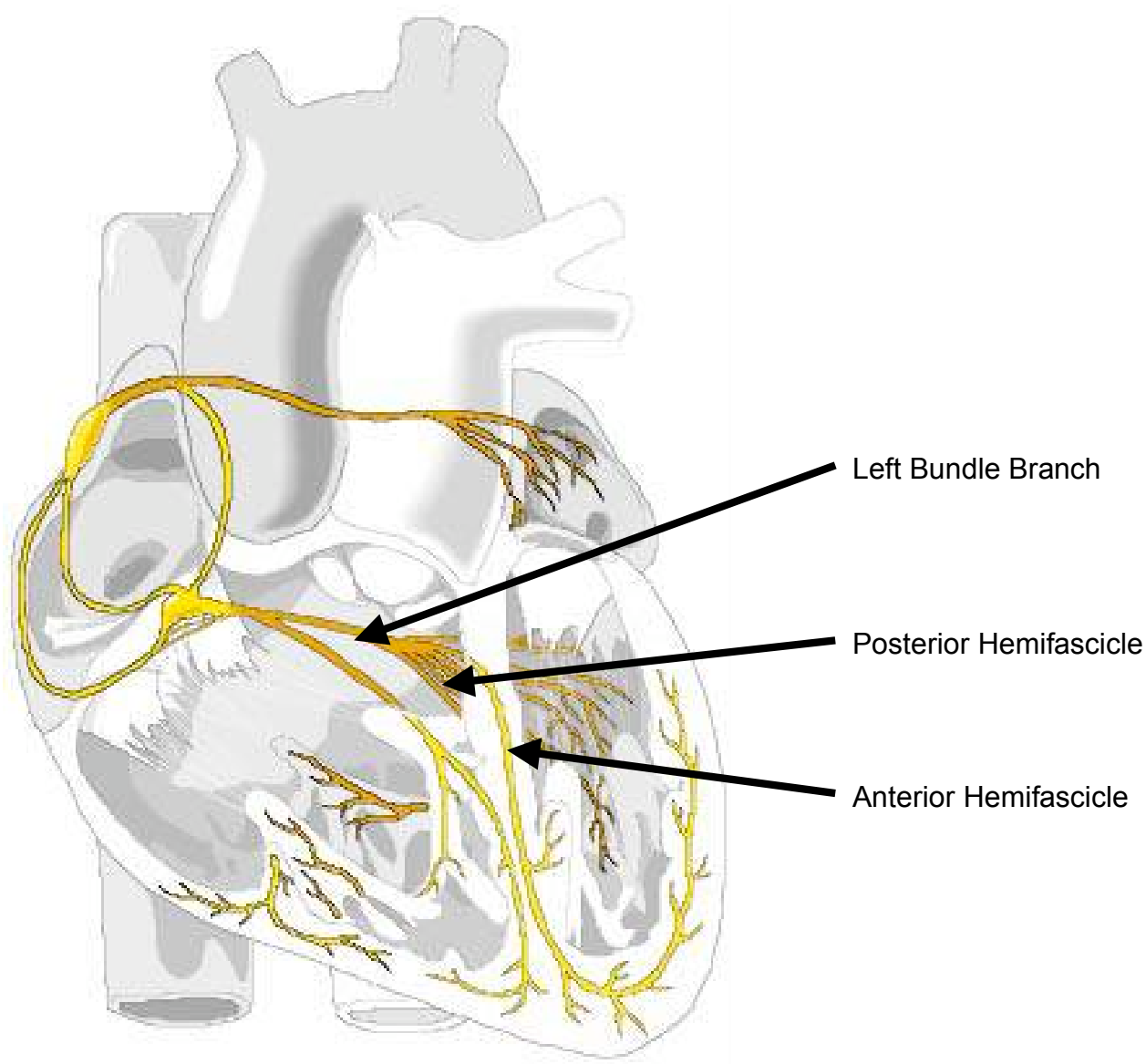


Hemiblocks:

A hemiblock is a block of one of the fascicles of the left bundle branch.

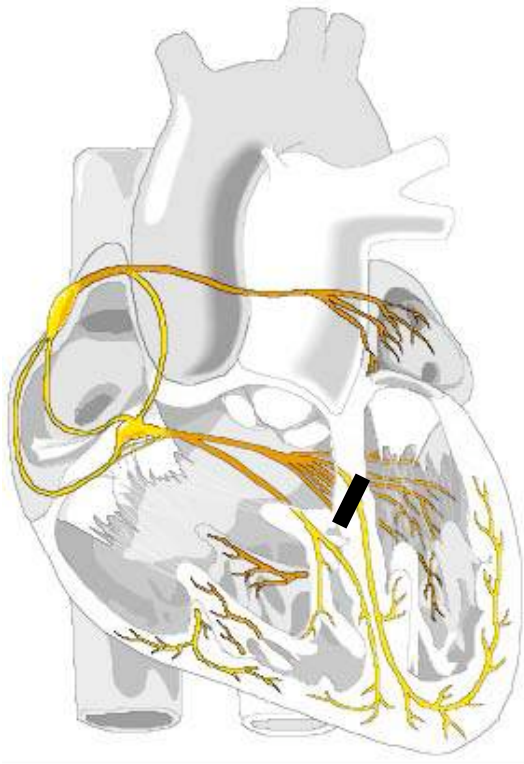
A hemiblock is a diagnosis made on ECG!

Significance: A hemiblock can be an indicator of conduction risks and severity of acute conditions such as AMI's. A hemiblock in the setting of an AMI means 4 times greater mortality



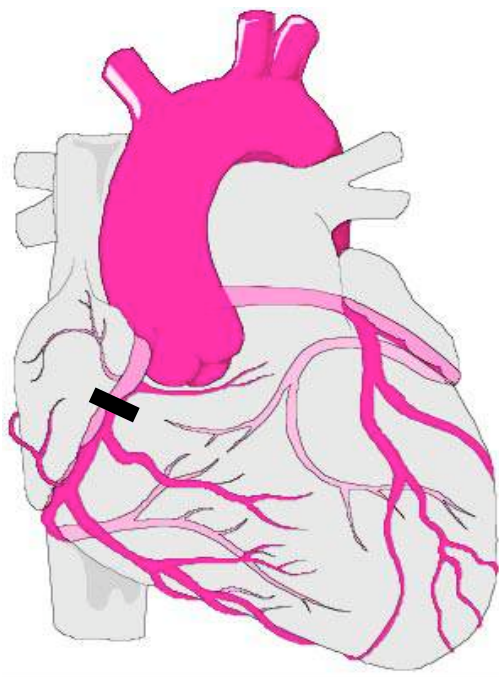
To Determine Hemiblocks:

1. Determine Axis
2. Look on the chart to determine hemiblock.



Anterior Hemiblock:

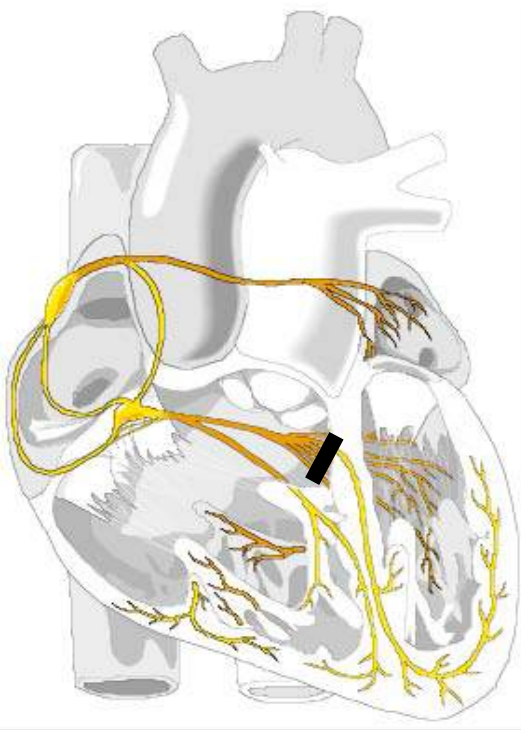
- Pathological Left Axis deviation
- Can also have small q in lead I and a small "r" in lead III.
- Common block
- 4 x higher mortality rate when associated with an AMI



Higher Mortality:

A hemiblock or other conduction system problem when associated with S/S of an AMI can indicate a proximal occlusion of a coronary artery.

The hemifascicles get their blood supply from a proximal artery branch. Therefore we assume a proximal occlusion has occurred. Mortality rate is higher because with a proximal occlusion, the risk of lethal arrhythmias is higher due to a larger area of ischemia.



Posterior Hemiblock

- ♥ Right Axis Deviation
- ♥ Can also have a small “r wave” in lead I: and a small “q wave” in lead III.
- ♥ Very high mortality rate when seen with S/S of an AMI. 70%+
- ♥ Two coronary arteries involved: Left anterior descending and Right coronary artery
- ♥ Posterior hemiblock is a rare occurrence. (Thank goodness!)

SUMMARY of AXIS and HEMIBLOCKS

- ♥ Use your chart to help you determine axis.
- ♥ People can live just fine and be asymptomatic with a hemiblock or axis deviation. It becomes relevant only when accompanied by the S/S of an AMI. Remember, **pay attention to your patient!** keep the ECG findings in the back of your mind
- ♥ A right axis deviation is a normal finding in children.
- ♥ A right axis deviation in adults is always pathological. Besides a posterior hemiblock, it can also be indicative of a pulmonary embolism, right heart failure (Cor Pulmonale).
- ♥ **You’ve always had the power to determine axis!**

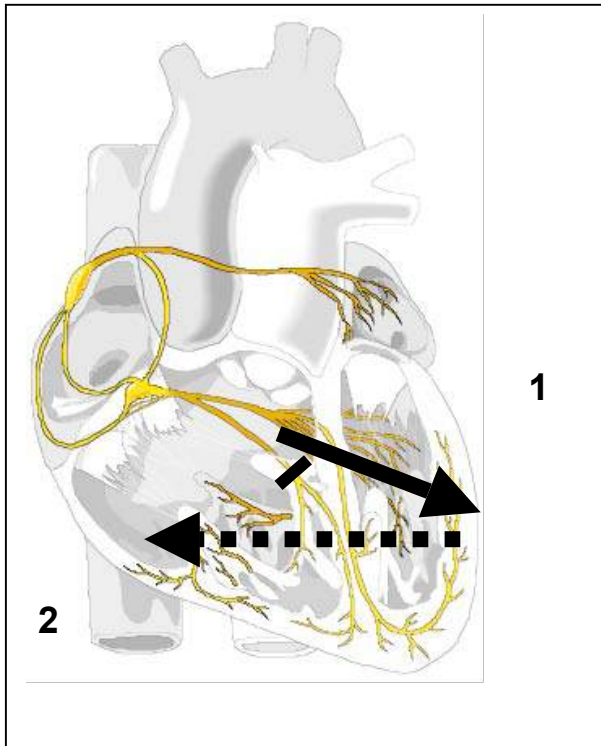
Bundle Branch Blocks

"To know bundle branch blocks is to know electrocardiography" - H.J.L. Marriott

A bundle branch block is a block of either the right or left bundle branch system.

A working knowledge of bundle branch blocks will help the paramedic determine:

1. **Who's at risk for hemodynamic compromise?**
2. **Who's at risk for complete heart block?**



The Bundle Branch System is made up of 2 main branches, right and left. The left bundle branch system is further divided into two hemi-fascicles, the anterior and posterior.

The purpose of the bundle branch system is to facilitate the entire ventricular mass to get the impulse to contract at the same time. This produces a phenomenon known as **syncytium** (both firing at the same time). Syncytium is very important to the cardiac output. In the cases of a **bundle branch block**, the ventricles are not in "sync." The preload could be reduced due to inadequate filling time. If the ventricles take a prolonged time to contract, then the force of the contraction will be reduced.

What Happens During A Bundle Branch Block?

A block of one of the fascicle of the bundle branch system can be caused by myocardial infarction (old or new), congenital defects or by ischemic tissue. In a few cases, people have acquired a BBB secondary to a procedure known as RF (radio frequency) ablation. This procedure is performed to destroy cells (which happen to be part of the bundle branch) that cause ectopy or dangerous rhythms such as VT. Some BBB's only occur during fast heart rates and can have defined "start and stop" rates. Nonetheless, a bundle branch block represents an increased severity risk for complete heart block, hemodynamic compromise, and of course sudden death when associated with an MI due to the proximal occlusion of the LAD coronary artery.

As the impulse coming from the AV node travels down the bundles, it reaches the tissue that is blocked on one side. The impulse stops on that side. The other side proceeds to depolarize as normal. **(1)** The wave of depolarization eventually works its way across to the blocked side causing a delayed depolarization. **(2)** As you might guess, the ventricles contract out of sync. The more diseased the muscle, the longer it will take to completely depolarize the heart. If the total time to depolarize the ventricles are longer than 120ms or .12 sec, then criteria is met for a bundle branch block.

So Who's at Risk for Hemodynamic Compromise?

HEMODYNAMICS 101

CO (cardiac output) = HR (heart rate) x SV (Stroke Volume)

Preload:	↙	Volume and pressure on LV
Afterload:		Vascular tone
Contractility:		Inotropic State

If the **QRS duration is > 120ms** (.12sec) (3 little boxes) then at the least a BBB exists. The ventricles, being out of sync have **reduced preload**.

If the **QRS is > 170ms** then the **Ejection Fraction is 50%** at the MOST. **Ejection fraction** is the volume percent of blood the heart can pump out. Healthy people at rest and awake have an ejection fraction between 60-75%. This results from **reduced contractility** because it takes so much time to depolarize; the contraction is also slow and weak.

What does this mean to you, the Acute Care Provider??

1. Before you give a med (other than O2) make sure you determine the presence or absence of Bundle Branch Block.
2. If the QRS is wider than 170ms and you are going to give a vasoactive drug such as Nitroglycerin;
3. REMEMBER
 - a. NITRO without an IV Line, HERE'S YOUR SIGN!
 - b. Before giving Nitro, always check ALL lung fields (especially posterior lobes)
 - c. ALWAYS have plan "D" ready
 - i. Plan D is actually the drug dopamine. (Intropin)
 - ii. The dose for contractility support (inotropic) is 5mcg/kg/min then titrated

Colorado Down and Dirty Dopamine Dose Ditty

(Thanks to Bob, Arlene, and Jill from Colorado)

Wt in lbs / 10 then subtract 2 This gives you the pump setting in ml/hr or gtts/min for dopamine at 5mcg/kg/min with in bag concentration of 1600mcg/ml.

Example the patients says they weigh 100 kg? Ha Ha yeah right! The patient weighs 220lbs. That would be 220 / 10, which is 22 minus 2 leaves 20. Set your pump at 20 ml/hr!

Since the left side has two fascicles and the right side has just one, a left bundle branch block has a higher mortality rate. It is important for the acute care provider to understand bundle branch blocks and be able to rapidly identify and differentiate left from right.

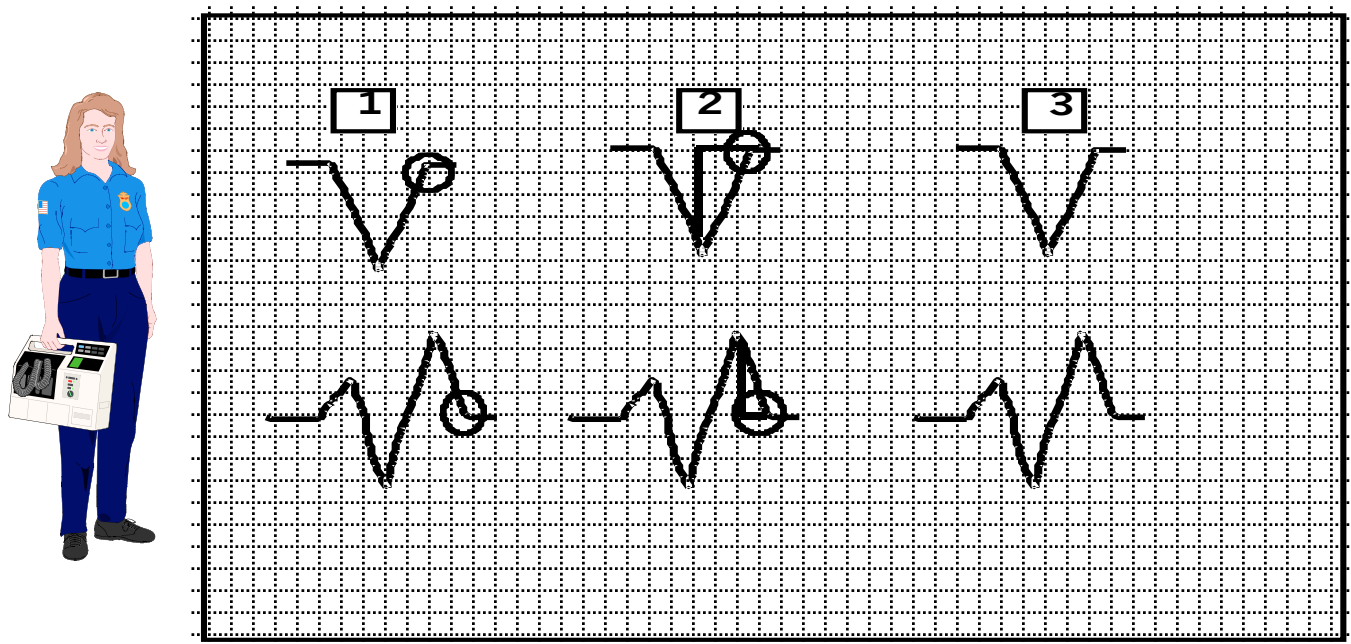
The methods presented in this session may seem ridiculously simple, but they are very accurate.

The “Turn Signal Theory” For Bundle Branch Blocks.

The turn signal theory was one of the first things I learned from Mike Taigman. This criterion is incredibly accurate and has been proven in study. Its method is non-traditional and has nothing to do with medicine.

In using this criteria there are 2 main rules:

1. It must be a supraventricular QRS complex > .12 seconds wide (120ms).
2. You must use MCL-1 (V1) for this theory to work.

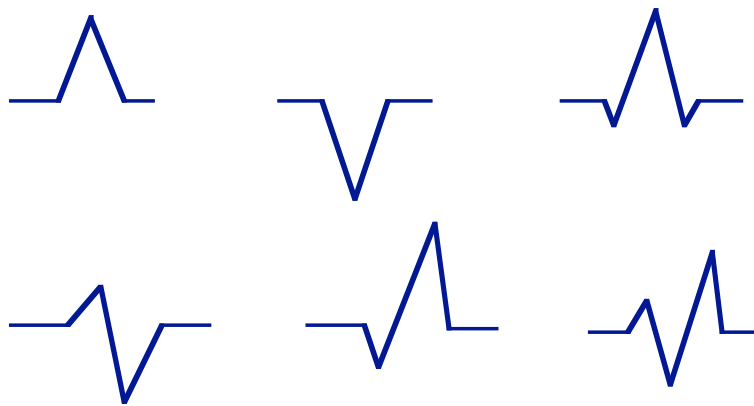


- Step 1:** Find the “J point” and circle it
- Step 2:** Draw a line into the QRS complex until halfway through the terminal wave and then down or up depending on the complex
Don't cross over any lines with yours.
- Step 3:** Shade in the arrow that this line makes.

If the arrow points up ---> Right Bundle Branch Block

If the arrow points down ---> Left Bundle Branch Block

**Can you label these bundle branch blocks?
Assume all are lead MCL-1 (V1) and are at least 120 ms wide**



Some Closing Thoughts on Bundle Branch Blocks

- ♥ Bundle branches are living breathing cells of the cardiac conduction system.
- ♥ They don't "hurt" they just "won't work" if damaged.
- ♥ Syncytium is necessary for crisp contractions and good cardiac output.
- ♥ A BBB can cause a reduction in preload and contractility.
- ♥ A very wide QRS (>170ms) means reduced ejection fraction to below 50%.
- ♥ Use your turn signals!
- ♥ You can't accurately or consistently call a LBBB or RBBB from a lead II strip!
- ♥ BBB means at least 25% greater mortality
- ♥ Like hemiblocks, people can live asymptomatic with BBB's. A BBB usually affects their lifestyle more however. Due to the hemodynamic effects mentioned earlier, the BBB will not allow much strenuous activity before putting the patient supine!
- ♥ As always, **pay attention to your patient**, keep this information in the back of your mind as you consider the treatment options and possible problems.

OF SPECIAL NOTE:

Don't look for an AMI by standard ST segment elevation with a LBBB!

Why? You may recall that in a BBB, the ventricles are firing out of sync. In a LBBB, the RBB depolarizes first followed by the left BB. By the time the LBB completes depolarization, the RBB has started repolarization. This is evidenced on the ECG as a "T" wave. This being the case, there was never a time in the cardiac cycle where there was not electrical activity going on, therefore the ECG did not have a chance to return to baseline and record an isoelectric ST segment. Thus, in a LBBB, the late depolarization (and repolarization) distorts the ST segment into appearing elevated.

Therefore most clinicians consider a LBBB to be a non-diagnostic ECG for an AMI unless of course they have a previous one to compare it to. This is always a major benefit.

Rate Dependant Bundle Branch Blocks

A major reason to monitor in MCL-1 is to spot a rate dependant bundle branch block when it occurs. Atrial fib and flutter can develop this condition as heart rate increases. Having

decreased cardiac output anyway due to lack of atrial kick, at a certain rate, the ischemia sets in and can lead to a bundle branch block. Care should be taken not to mistake this for V-Tach as therapy for VT could cause a rapid demise of the patient. Recognize the criteria for BBB (RSR', or QRS complex) rapidly. Look carefully for an irregularly, irregular rhythm. That would be a tip off for A-fib (and therefore BBB when the complex turns wide).

Who's at Risk for Complete Heart Block?

Which of your patients is at risk for complete heart block? You have all the information available by running the 4-lead baseline (I, II, III, and MCL-1). This is an important fact to know since some commonly used drugs can be contraindicated in the presence of severe degrees of *intraventricular heart block*. A severe degree is when you have two or more blocks listed at once. Below is a list of those considered to be a "severe degree" of block.

CONTRAINDICATIONS

Lidocaine is contraindicated in patients with a known hypersensitivity to local anesthetics of the amide type.

Intravenous injection is contraindicated in patients with Adams-Stokes syndrome or with severe degrees of sinoatrial, atrioventricular or intraventricular heart block.

WARNINGS

Constant monitoring with an electrocardiograph is essential for proper intravenous administration of lidocaine. Signs of excessive depression of cardiac conductivity, such as prolongation of PR interval and QRS complex and appearance or aggravation of arrhythmias, should be followed by prompt cessation of intravenous injection or infusion of this agent. It is mandatory to have emergency equipment and drugs immediately available to manage possible adverse reactions involving the cardiovascular, respiratory or central nervous systems. Evidence for proper use in children is limited.

ABBOTT HOSPITAL PRODUCTS

Lidocaine Hydrochloride Injection, USP

AQUEOUS SOLUTIONS FOR ACUTE MANAGEMENT OF CARDIAC ARRHYTHMIAS OR FOR LOCAL ANESTHESIA.

DESCRIPTION

Lidocaine hydrochloride is designated chemically as 2-(diethylamino)-2', 6'-acetoxylidide mono-hydrochloride and is freely soluble in water. Lidocaine occurs as colorless crystals. The injectable preparation is an aqueous solution of the freely soluble hydrochloride salt. pH adjusted with sodium hydroxide and hydrochloric acid.

FOR CARDIAC ARRHYTHMIAS

ACTIONS

Lidocaine reportedly exerts a cardiac antiarrhythmic effect by increasing the electrical stimulation threshold of the ventricle during diastole. At usual doses, lidocaine produces no change in myocardial contractility, in systemic arterial pressure or in absolute refractory period.

About 90% of an administered dose of the drug is metabolized in the liver. The remaining 10% is excreted unchanged in the urine.

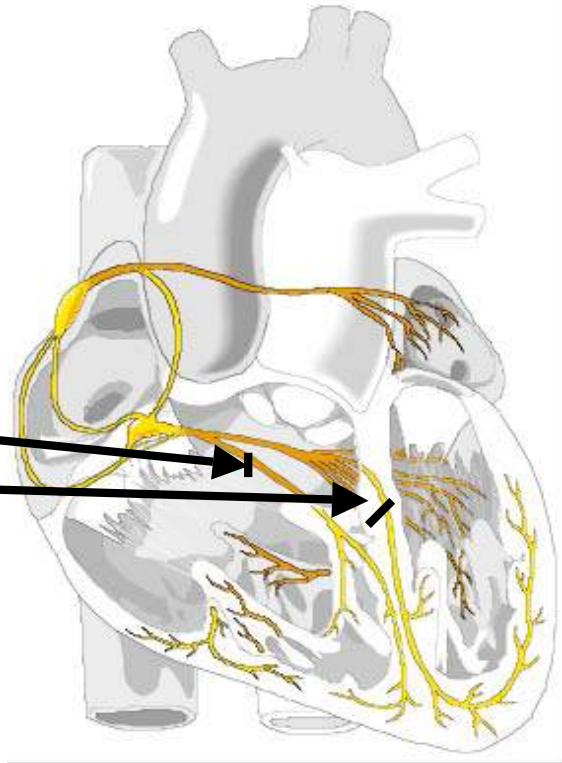
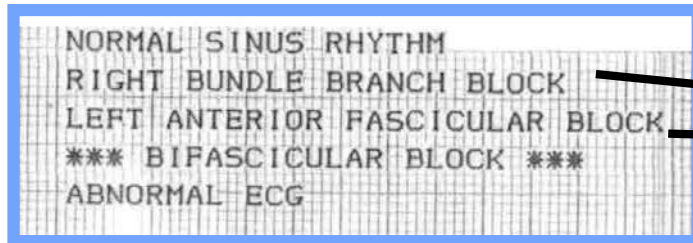
INDICATIONS

Lidocaine administered intravenously is specifically indicated in the acute management of (1) ventricular arrhythmias occurring during cardiac

SEVERE DEGREES OF BLOCK

- ♥ 2nd Degree Type 2
- ♥ 3rd Degree CHB
- ♥ Bifascicular Block
- ♥ 1st Degree + Hemiblock
- ♥ 1st Degree + BBB
- ♥ In short any time you say or imply block twice in the interpretation, the patient is at high risk for complete heart block. Anti-arrhythmia drug therapy is contraindicated for PVC's or transient arrhythmias.

A **Bifascicular block** represents a serious conduction problem for the heart. In a bifascicular block, 2 out of 3 pathways to contract the ventricles in an organized fashion have been blocked. This can compromise myocardial contractility.



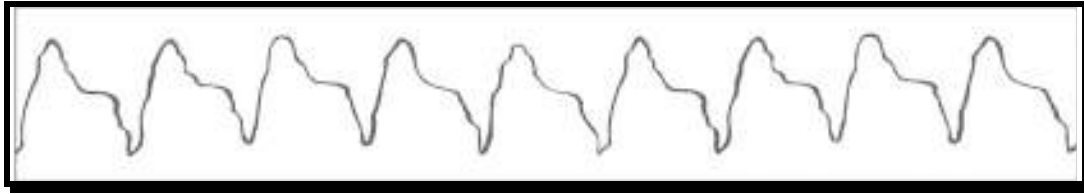
Preparing For the Symptomatic Patient with A Bifascicular Block:

- ♥ The patient is much sicker than an uncomplicated MI.
- ♥ They may go into complete heart block suddenly and without warning.
- ♥ They may **drop their blood pressure precipitously**, especially if the QRS is > 170ms. If so remember the three things to do before you give nitro and ask how much they weigh!
- ♥ They may just go into V-Fib or VT without warning.
- ♥ **Anti-arrhythmia drugs are contraindicated in cases of PVC's or transient tachyarrhythmia. Treat the cause of the arrhythmia!**
- ♥ In patients with S/S of an MI and all unstable patients, place Quick Combo or multi-function pads **on the patient**.
- ♥ Be prepared to **PACE** the patient.
- ♥ Be prepared to **DEFIBRILLATE** the patient
- ♥ Some patients may tolerate this condition well, until they get up to go do something. The heart cannot fill the request for extra output due to compromised conduction system.

Remember, as an acute care provider, your job is to make sure air goes in and out, and blood goes round and round. After that, you should triage Acute Coronary Syndromes, and either fix it yourself (with fibrinolytics) or KEEP them ALIVE and get them somewhere fast that can get it done! Knowing this information can help in that last part. The Grim Reaper usually takes acute cardiac patients one of three ways, VT or VF, Heart Block, Or Heart Failure (drop in blood pressure). Now you are ready for anything. Don't fear the Reaper!

Strategies for Differentiating Wide Complex Tachycardia and More!

Rule # 1: If the complex is very fast and wide, and your patient is unstable, in other words they would not mind if you shocked them, immediate cardioversion is indicated!



The 2000 Guidelines of the AHA put an end to unknown origin wide complex tachycardia by calling for a 12 Lead ECG to attempt to diagnose the specific rhythm. In other words, they strongly discourage a “give a drug and see what happens approach” The 12 Lead ECG is 96% diagnostic for VT identification. There have been several criteria used. In this section, I will present **“Key 3 Criteria”**. The combination of these criteria, when used for STABLE wide complex tachycardia, the following clues can help you make the right call and give the right medications, if needed.

Why even mess with a 12 lead? Can’t we see it most of the time in Lead II?

Dr. Marriott had a compelling study that demonstrated that Lead II’s diagnostic accuracy for VT as being only 34%. That means when faced with a decision about a wide complex rhythm, in lead II, you would guess wrong 66% of the time.

The most common pitfalls made in VT (or wide complex tachycardia WCT) diagnosis are as follows:

1. Reliance upon Lead II.
2. Reliance upon the machine to interpret it.
3. Thinking the vitals has to be horrible or the patient has to be symptomatic.
4. Getting in too big of a hurry.
5. Not playing the odds.
6. Forgetting about Atrial Fibrillation.
7. Thinking that VT is more common than SVT.

Besides, there is now more benefit and less risk in knowing the arrhythmia. The patient will further benefit from the 12 lead because you may be the only one to see that arrhythmia. If you have a 12 lead it will help the cardiologist determine the origin of the arrhythmia

Since ventricular tachycardia is a life threatening arrhythmia, the **KEY 3 Criteria** presented are designed to aid the acute care provider in the rapid confirmation of the presence of VT. Refer to the handy cutout of this entire sequence found at the back of this workbook

WIDE QRS TACHYCARDIAS: The KEY 3 Criteria for VT

Deciding to employ the KEY 3 Criteria

If the patient presents in a wide complex tachycardia (especially one faster than 150 bpm), then use the **KEY 3 Criteria** before you do anything else!

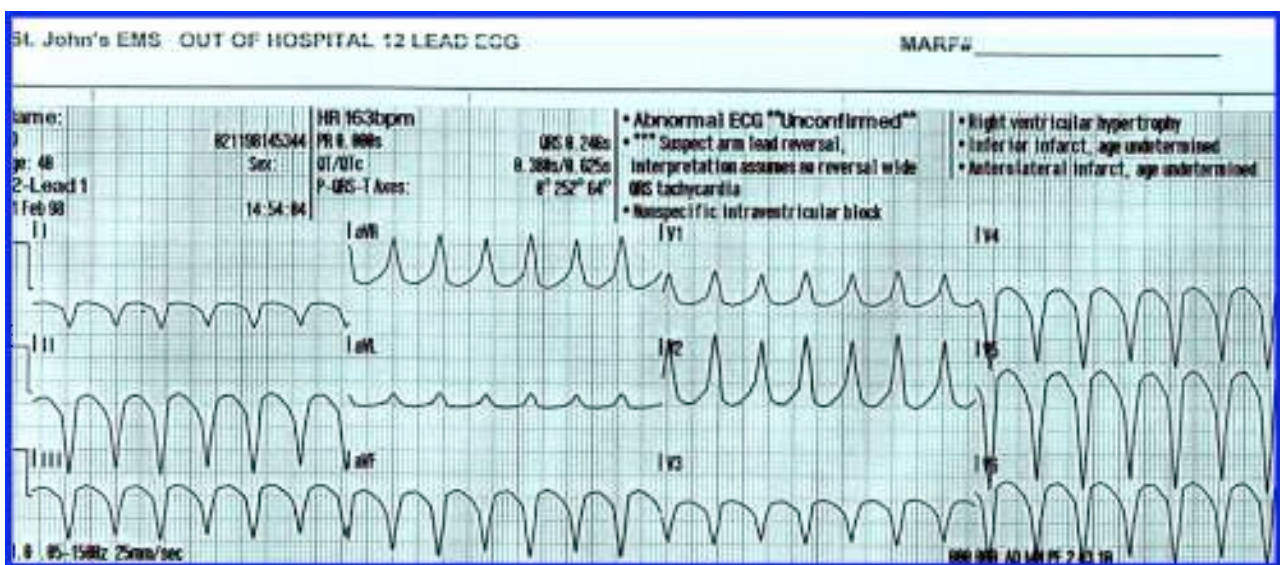
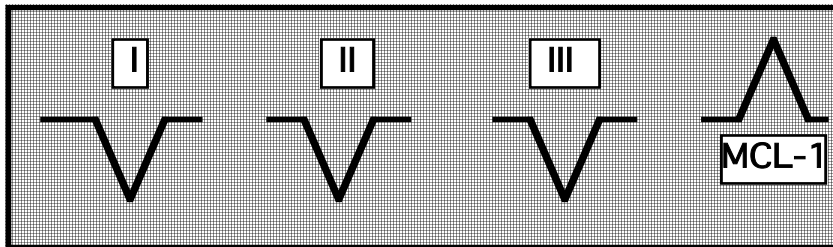
The reason is simple, if the patient is in VT, then you must treat it, now! If the patient is in a ventricular arrhythmia, then nothing else can be read on the 12 lead. No hemiblock, no BBB, no ST elevation, nothing. This also applies to the patient who has any type of ventricular based rhythms: paced ventricular, idioventricular, accelerated idioventricular, and even a third degree heart block with a ventricular escape focus.

With that Preface, lets look at the **KEY 3 Criteria for VT**

Run a 12 lead ECG or at least leads I, II, and III. MCL-1 (V1), and MCL-6 (V6)

1

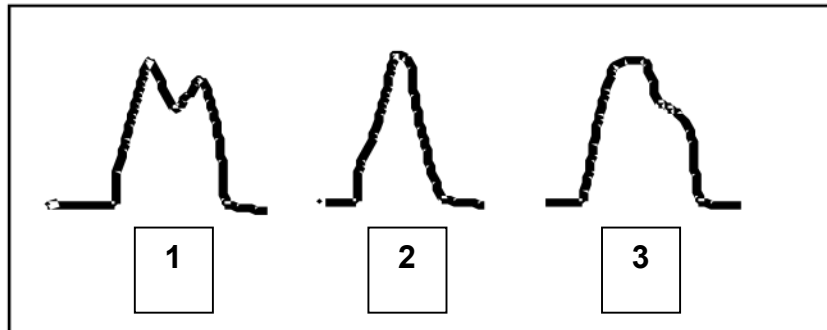
Extreme Right Axis deviation and Upright V1 (MCL-1)
If this is true, then you have VT, if not go on to #2



2

Lead MCL –1 (V1) Morphology Criteria

If MCL-1 (V1) is an upright complex....

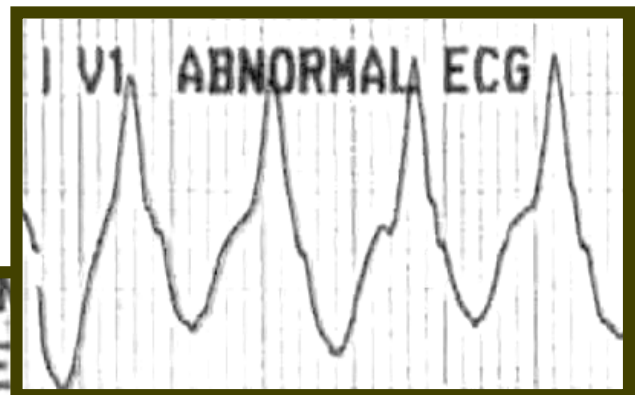
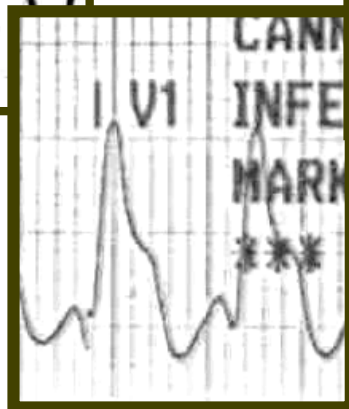
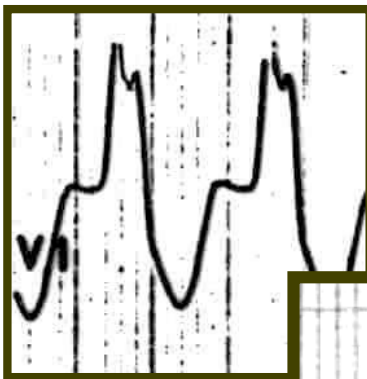


1. Taller left peak than right, “BIG mountain little mountain”
2. Single upright peak, “steeple sign”
3. Single peak with a slur, “fireman’s hat”

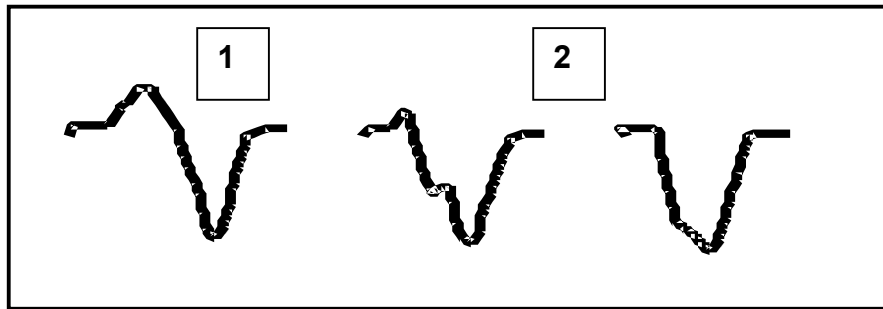
Lead MCL-1 (V1) Morphology has a 94% accuracy if it looks like one of these examples

If they look like this in a wide complex tachycardia, treat for VT!

If it does not meet these criteria, try the 3rd criteria (V6)



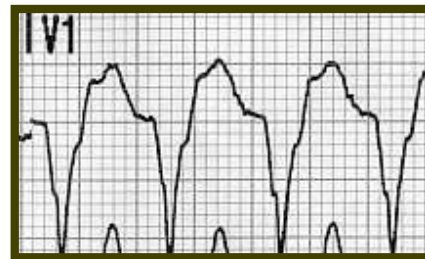
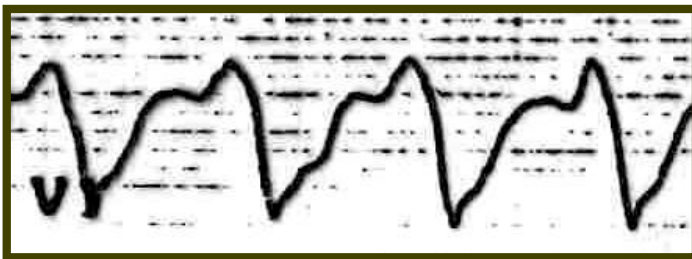
If MCL-1 (V1) is a negative deflection...



1. Fat "R" wave – the r wave is more than 40ms (one little square) wide.
2. Slurring or notching to the initial downstroke (q or s wave)

If it meets this criterion, treat as VT.

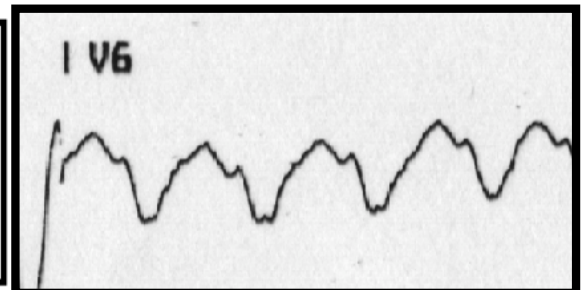
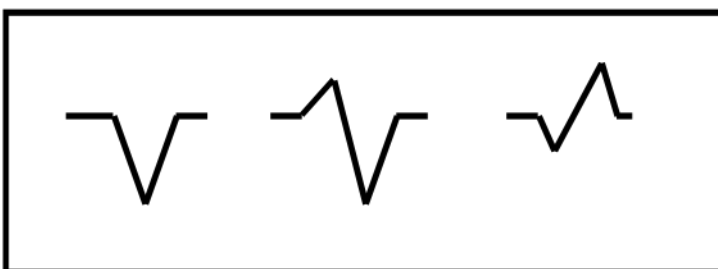
If it does not or you are unsure go to criteria # 3



3

Lead MCL-6 (V6) Criteria

1. Any negative deflection (when you get to this point) is VT
2. Rare cases show a fat q wave in a biphasic complex



What if you still don't know what it is after the Key 3 Criteria?

Here are some other interesting notes at VT that may be helpful

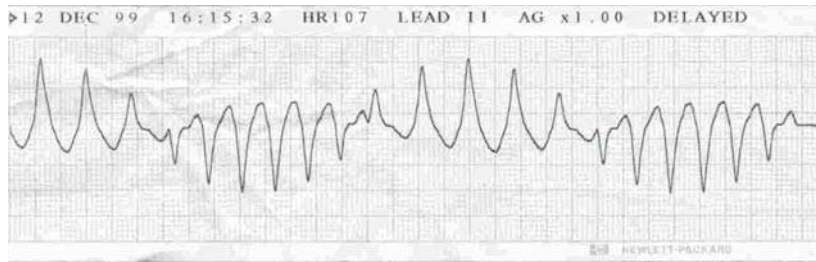
- V-TACH, unless drug induced is usually very regular
- Irregularly irregular rhythms are probably Atrial Fibrillation
- Try Adenocard
- Ask two questions: Have you had a MI before? and Did you have tachycardia after the MI?
- ***If you get a yes to both questions, the odds favor V-Tach! (86%)***

V - Tach stuff worth mentioning: BONUS INFORMATION!**1. Choose your poison (or your drug)**

Studies have shown that mixing anti-arrhythmia drugs can be “pro-arrhythmic” In other words it will actually induce other arrhythmia. Now your drug armamentarium for stable VT that is not hemodynamically impaired is as follows:

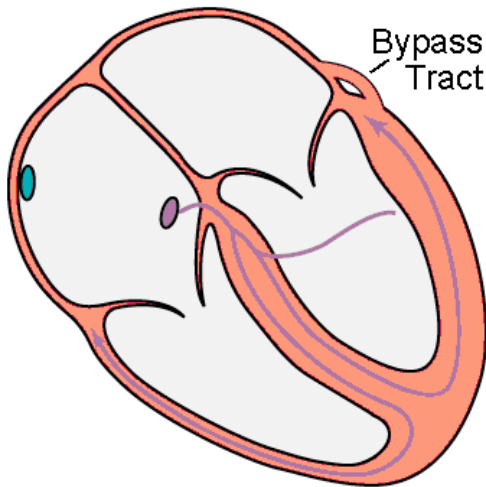
Procainamide or Amiodarone or Lidocaine

Word is if one drug does not convert it, seek cardiology consult or shock ‘em (cardioversion). To give another different drug may invite this to happen.

**2. There is another version of “Stable”.**

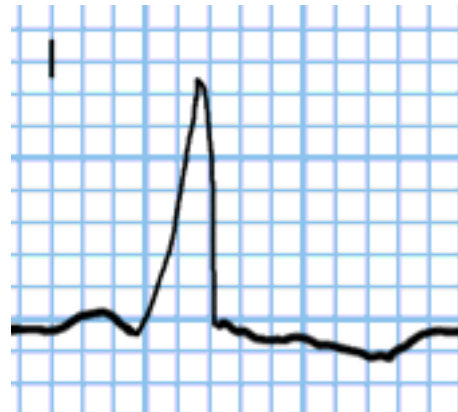
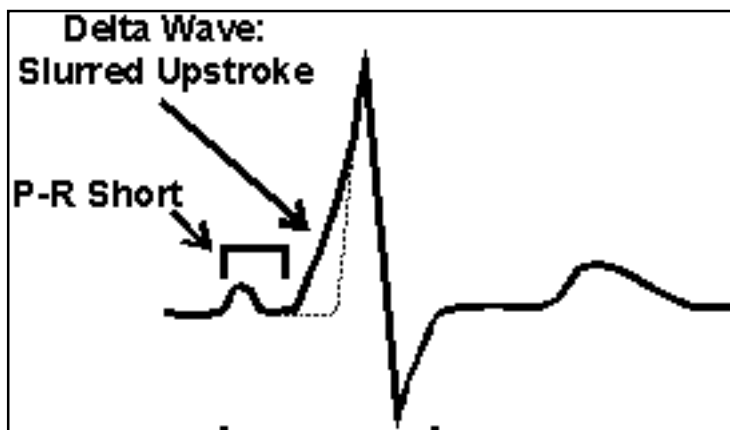
That is a person who would mind if you shock them, but they are impaired with a bad ejection fraction (less than 40%). But how do we know that? We can't measure ejection fraction. Here's the scoop: If they have crackles or present in failure (low B/P but still are awake), then Amiodarone OR Lidocaine are the drug of choice. Lay off the procainamide because it can drop the pressure and worsen the failure.

Wolff Parkinson White Syndrome

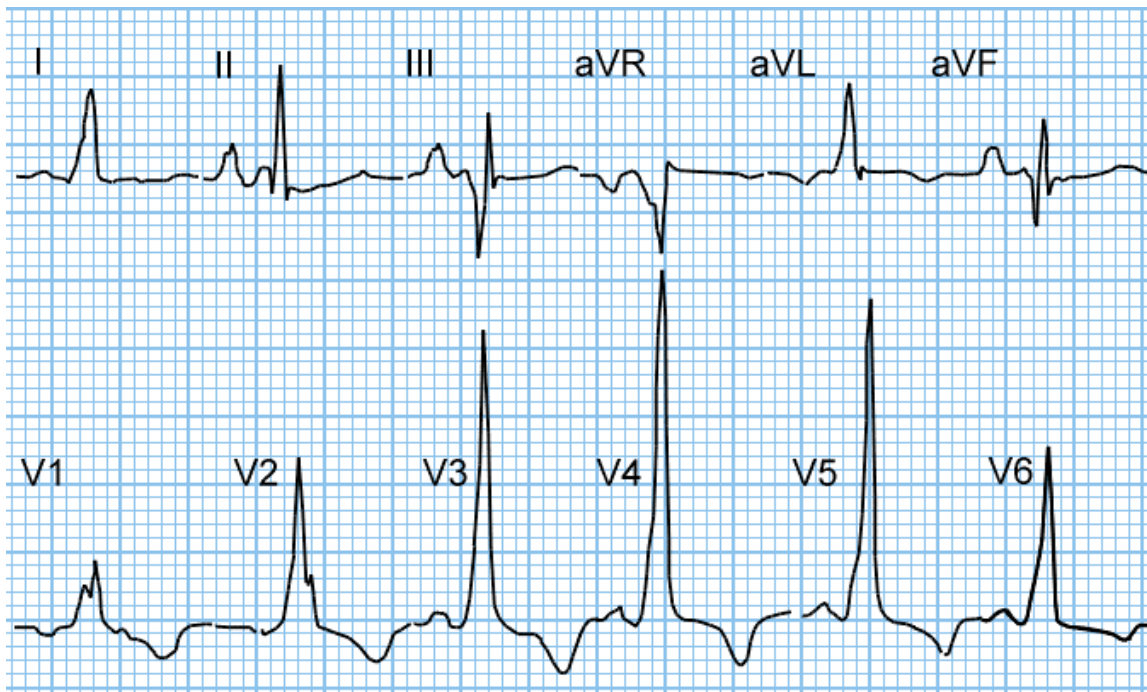


One renowned arrhythmia is caused by the presence of an accessory pathway known as WPW. In WPW, the impulse from the atria does not travel through the AV node to the ventricles. Instead it takes an accessory pathway commonly known as the Bundle of Kent. By bypassing the AV node, there is nothing to slow the impulse to the ventricles. The result is a pre-excitation of the ventricles, causing them to fire before the ventricles are through filling. This pre-excitation results in a classic ECG finding of **Delta Waves** and **short PR interval** (less than 120 ms). The accessory pathway is unreliable and will allow impulses to pass from the ventricles back to

the atria causing it to depolarize, thus completing a re-entry pathway. The result could be a very fast tachycardia that can quickly become hemodynamically unstable.



These ECG findings for WPW (Delta Waves) are diagnostic for WPW. In fact the best leads to see this in would be the leads that look at the left lateral side of the heart. Leads I, and aVL, V5 and V6, and even V3-V4. Lead II, as you might guess truly has no clue as the sensitivity for Delta Waves in lead II from one study was only 25%. That means that WPW will not show up 75% of the time in lead II. You NEED a 12 lead!



WPW. Note the slurred “delta waves” and short PR interval. In addition, a positive concordance of all of the V-leads is observed. This finding can be VT or WPW. Good thing you know the difference now, huh?

Management of WPW when the EJ is above 40%:
Procainamide or Amiodarone

Avoid Adenosine, Beta Blockers, Calcium Channel Blockers or Digoxin

The Lown-Ganong-Levine syndrome (LGL) is usually considered in the same class of pre-excitation syndromes that includes the Wolff-Parkinson-White syndrome (WPW). Investigations into WPW have revealed that an accessory pathway for conduction, called a Bundle of Kent, from the atria to the ventricles underlies the pre-excitation observed in patients with WPW. Less is known regarding the structural anomalies underlying LGL. Theories proposed to explain LGL have centered around the possible existence of intranodal or paranodal fibers that bypass all or part of the atrioventricular (AV) node. LGL is characterized by a short PR interval (less than 120ms) and normal QRS width without a delta wave. It is also believed the pre-excitation occurs as a result of enhanced AV nodal conduction. LGL usually presents with SVT but NOT A-fib or A-flutter.

Myocardial Blood Supply:**Right Coronary Artery (RCA)**

inferior wall of the left ventricle

SA Node in 50% of folks

AV node in 90 % of folks

right ventricle

Posterior Descending Artery (a branch of the RCA)

posterior fascicle of the left bundle branch

posterior wall of the left ventricle

left bundle branch

Left Anterior Descending (LAD) "widow maker"

anterior wall of the left ventricle

intraventricular septal artery (**septum**)

RBB, LBB, both fascicles of the LBB

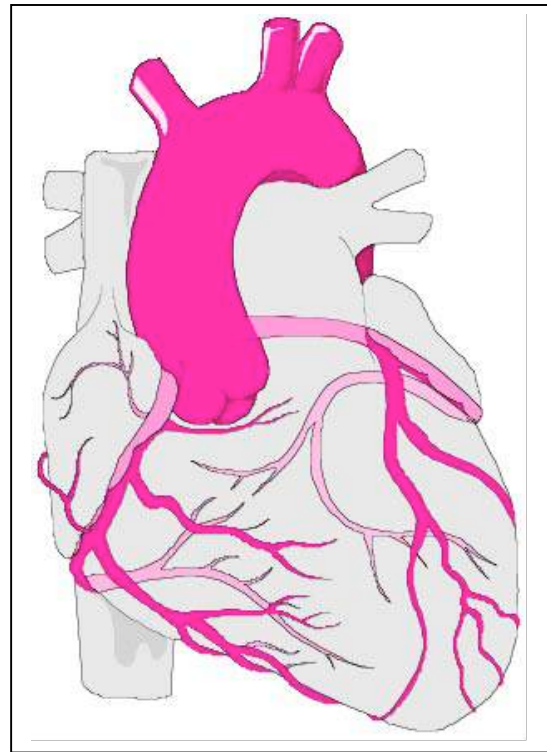
Left Circumflex Artery (LCA)

lateral wall of the left ventricle

SA node in 45% of patients

AV node in 10% of patients

posterior wall of left ventricle

Coronary Arteries

Why do you need to know this?

If you know the blood supply, then ECG changes can help you predict which areas of the heart may give you trouble either as infarct or conduction deficits. It always helps to be prepared for the worst. This preparation comes through a sound working knowledge of cardiovascular pathophysiology, and electrocardiography.

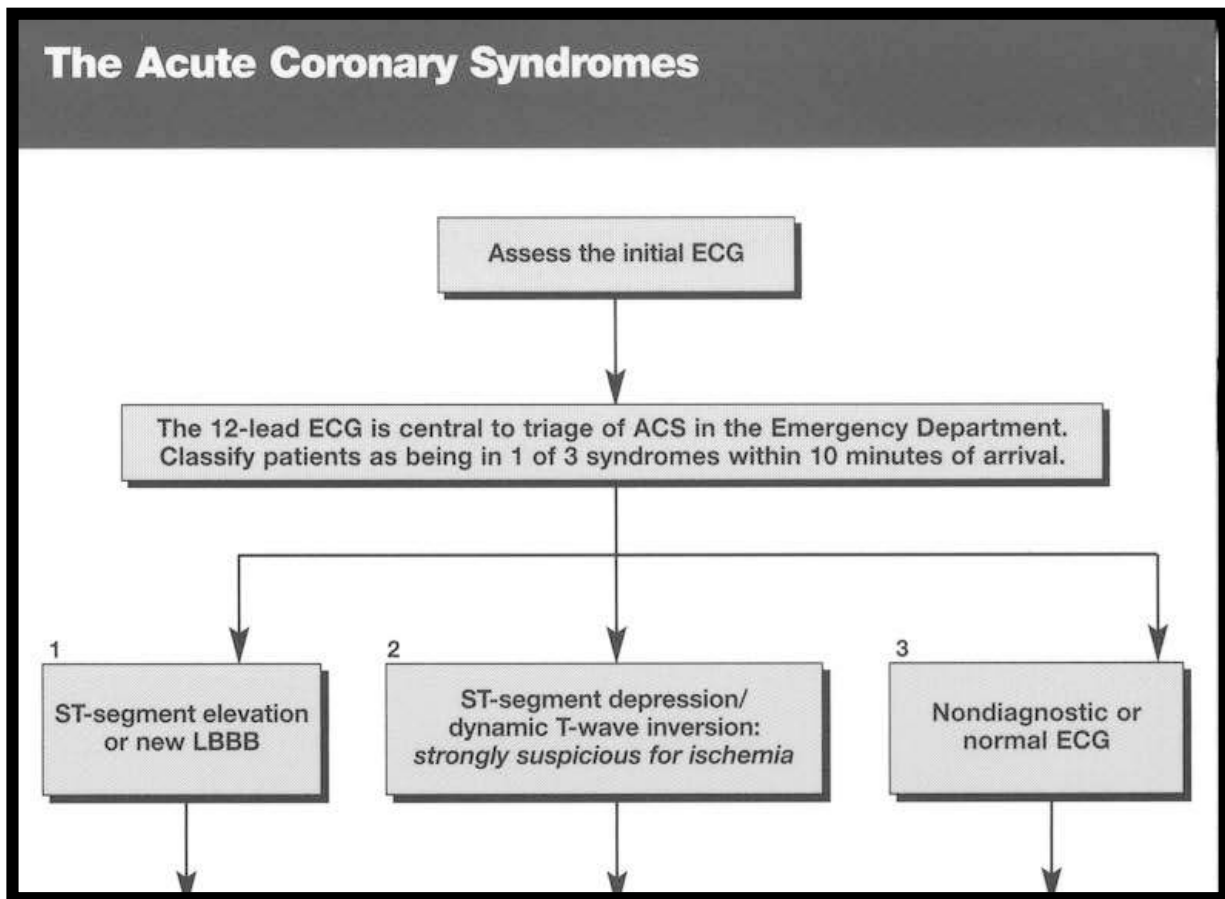
Genesis of an AMI

1. Atherosclerotic plaque formation
2. Plaque ruptures
 - Lesion sets off cascade of clot forming biochemistry
 - Clot forming is a dynamic process
 - Process can be interrupted with anti-platelets and anti-coagulants
3. Occlusion data
 - CP at exertion = 70 - 85% occlusion
 - CP at rest = 90% occlusion
 - CP that does not resolve with NTG = 100% occlusion
4. Intervention plan for EMS
 - Out of Hospital 12 lead ECG
 - Early notification of hospital
 - O2, NTG, and pain control
 - ASA, Heparin
 - Fibrinolytic checklist
 - TPA in the field?
1. Where EMS can make a difference
 - Early recognition and treatment
 - Salvage myocardium (time is muscle)
 - Reduce incidence of CHF means reduced \$\$\$
 - Saves "lifestyles"



ECG Evidence of Acute MI

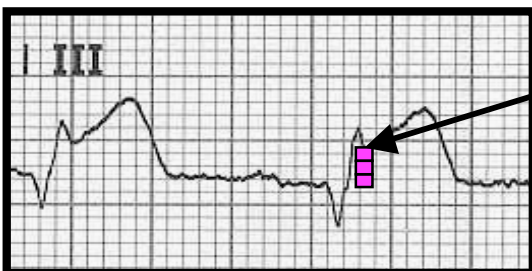
- Limitations
 - Only 46-50% sensitive (could miss 50% of MI's)
 - **Never "rules out an MI"**
 - Who is reading the 12 lead ECG?
 - ECG evidence is only one piece of a large puzzle
 - some non-MI conditions that resemble MI's
- Benefits
 - Highly specific (90% + confidence)
 - If it shows the MI, it is there
 - Rapid identification possible in early stages
 - Can commit to treat with ECG and H & P evidence
 - Complications can be identified (hemiblocks, BBB)

Triage of the 12 Lead ECG

The purpose of this triage is to rapidly identify patients that will benefit the most from early reperfusion treatment (fibrinolytic or invasive procedures such as rescue PTCA or CABG.)

1. The highest priority are those with ST segment elevation or new onset LBBB

ST segment changes must be seen in two or more related leads or those that are “anatomically contiguous”



- ST segment elevation > 1mm in two or more related leads. (> 2mm in septal leads)
- ST elevation means acute transmural injury
- Measure at J point (plus .04) to baseline.
- Look for the obvious, don't read into it!

Patients demonstrating positive ECG criteria such as seen above would reap the greatest benefit from early reperfusion strategies. Every effort should be made to expedite the process.

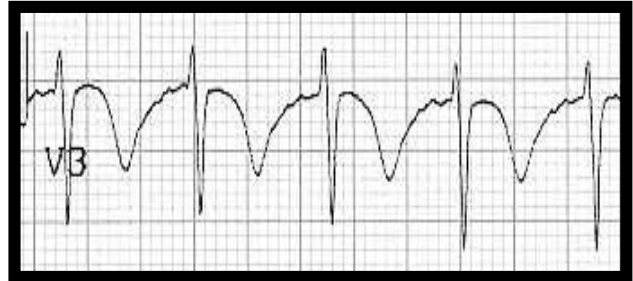
ST segment depression or “T” wave inversion: High Risk Ischemia.

ST segment changes must be seen in two or more related leads or those that are “anatomically contiguous”

These patients are not candidates for early reperfusion therapy, but should be treated aggressively with anti-platelet / anti-thrombin drugs such as Aspirin, Heparin, and newer drugs known as glycoprotein IIb/IIIa inhibitors such as Reo-Pro, Integrelin, or Aggrastat. Did you know that some of these drugs are made from snake venom?



ST segment depression
Reciprocal for elevation or acute
ischemia?



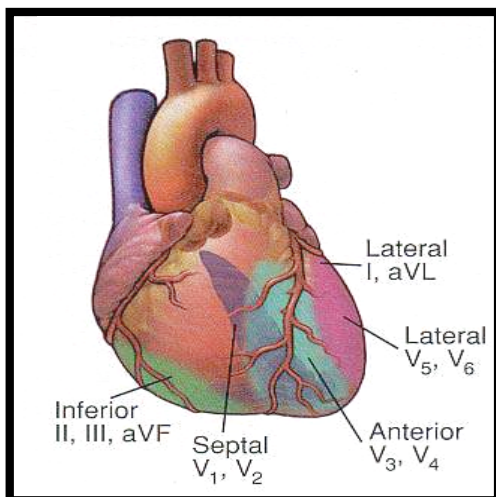
Symmetrical “T” Wave inversion
Early sign of ischemia

3. Non Diagnostic or Normal 12 Lead ECG

**Remember, a normal 12 lead does NOT rule out an infarction. It may be too early!
Run serial 12 leads every 10-15 minutes!**

Where did “Q waves” go?

Q waves did not go away, it is just that they are of little use, present or absent, in the management or diagnosis of an acute event. Q waves (wide ones or those that are deep) still indicate infarcted tissue, and in the absence of ST-T wave changes, are probably old.

A System for “Assessing a 12 lead ECG” for Infarcts**“I SEE ALL LEADS - RP”**

- **I** - Inferior leads II, III, AVF
- **S** - Septal leads V1, V2
- **A** - Anterior leads V3, V4
- **L** - Lateral leads V5, V6, I, AVL

- **R** – Right ventricle V4R
- **P** – Posterior leads V8, V9

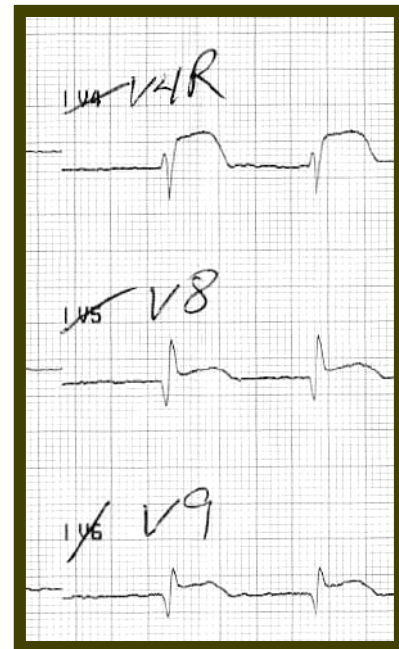
A System of Triage for Acute Coronary Syndromes: The Acute MI Locator

Acute MI Locator ST[↑]elevation		
Location	Leads	Recip.
Inferior (RCA)	II, III, F	I, AVL
Septal (LAD)	V1, V2	
Anterior (LAD)	V3, V4	II, III, F
Lateral (CIRC)	V5, V6, I, L	II, III, F
Posterior (RCA)	V8, V9 R>S in V1	ST [↓] V1 - 4
Right Vent. (RCA)	V4R	

- How to Use the System
 - Look at the lead groups in order (ISALRP)
 - Look for ST segment elevation
 - ST elevation points at the area injured
 - Write down (or circle the leads where the changes are found
 - Look up the leads on a chart to determine location
 - **ST elevation the most important thing you are looking for.**
 - **Confirm ST elevation MI with reciprocal ST depression in one of the leads listed**
 - If no ST elevation is found, use the same system to look for inverted T waves, ST depression

The Importance of a 15 Lead ECG

- 15 lead ECG's (12 leads plus V4R, V8 and V9)
 - Run on all non diagnostic 12 leads
 - Can locate isolated posterior MI
 - Use leads V8 and V9
 - Increases sensitivity by 23%
 - Can't see posterior wall directly on a 12 lead
 - Inferior MI on 12 lead
 - 50% have **Posterior and/or Right Ventricular involvement**
 - Right ventricular infarction is a preload problem and could cause dramatic hypotension with nitrates. Manage hypotension with fluids in this case.
 - Use lead V4R to look for right ventricle infarction
 - Clinical signs of RVI include JVD, dry lungs, and hypotension



Other ACS Information of Note: Things that could have ST elevation but aren't an AMI.

- **Infarct Imitators**

- **LBBB** - late depolarization of left ventricle makes ST elevation nearly impossible to distinguish.
- LBBB considered to be a non diagnostic ECG
- Left Ventricular Hypertrophy (Huge QRS complexes)
- LVH won't have reciprocal ST depression



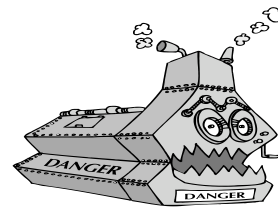
- **Pericarditis**



- Concave ST elevation in all leads
- Patient feels better when He/she leans forward
- Pericarditis will not have reciprocal ST depression

- **Dissecting Thoracic Aortic Aneurysm**

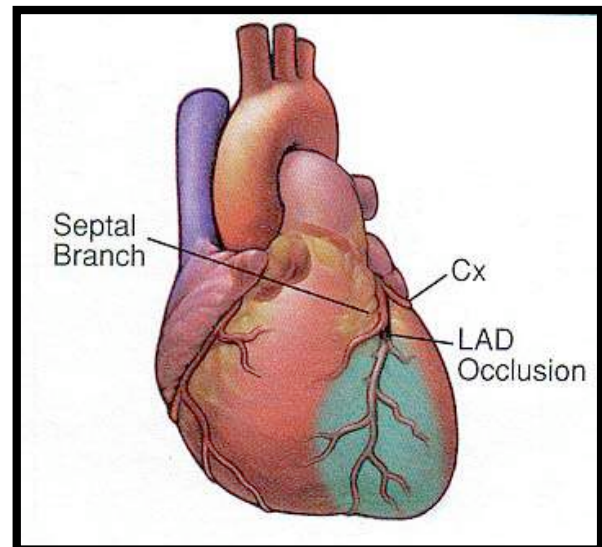
- Dangerous if misdiagnosed as MI
- Heparin and NTG could be fatal
- Does not have reciprocal changes (ST depression)
- Ripping, tearing pain
- Assess B/P in both arms

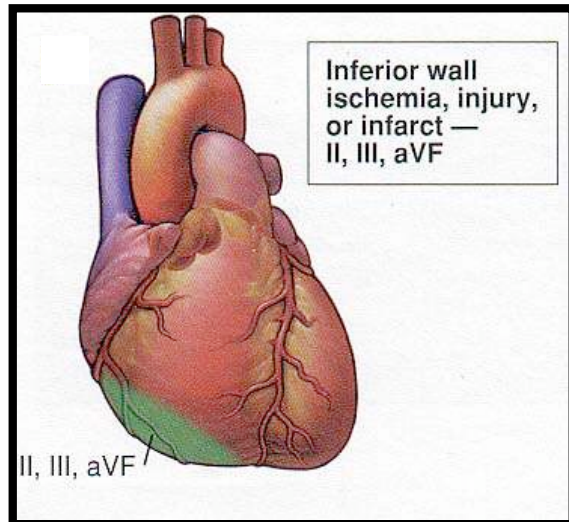


- **Infarct Caveats (What to expect from various infarct locations)**

- **Anterior MI**

- Most lethal (highest mortality)
- Can suddenly develop, CHB or VF, VT
- If presents with hemiblocks or BBB, apply quick combo pads to the patient and prepare for the worst
- Can extend to septum (anteroseptal) and / or lateral (anterolateral) walls
- Nitrates are great, fluids are spared





Inferior MI

- Most common seen
- Can be very lethal
- 50% have posterior and/or right ventricle and hypotension
- Could also have 1 degree AV delays or 2nd degree 1
- Nausea is a common finding: Anti-emetics are in order here!
- Look for RVI with V4R, nitrates with caution, fluids may be needed (a liter or more) to support B/P.
- Frequently extend to lateral (inferolateral) wall

- Posterior wall
 - Seen as ST depression in leads V1 - V4 (reciprocal leads)
 - Can present with R > S in V1 and V2
 - A 15 lead ECG (V8 and V9) will show the ST Elevation
- Lateral and Septal Wall
 - Rarely occur alone, usually an extension of anterior or inferior MI

Final Thoughts on **Multi-Lead Medics™**

12 lead ECG's are fast becoming NOW a standard of care!

The need for multi-lead is very plain.

There are patient's everyday that can benefit from your "extra lead knowledge."

At first, doing multi-lead seems a bit awkward.

There may also be those who have never heard of, or others who give you a hard time about multi-lead things such a lead placement and running extra leads. Remember however, that it is *your* patient. You always want the best for your patient! Time does not pass as fast as you might think! Also realize that the methods taught in this course are non-traditional and that there are many ways to get to the correct interpretation. Don't be surprised if others get the same answer, in a different manner! Also, don't be surprised if some don't believe what you have learned! Lead by example.

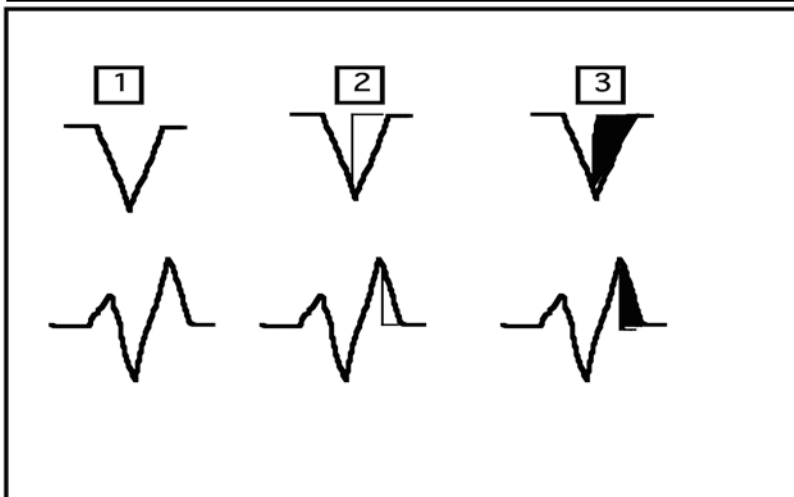
Practice, practice, practice!

If a patient has a "pulse and a problem", you're going to run a 12/15 lead ECG. Any time you hook up the cardiac monitor, run at least the 4 lead baseline. Any look at the heart is worth at least 4 looks! Run it on your partners at work, or during training sessions. Remember when you first learned how to read an ECG strip? I'll bet it took you a little time to get comfortable in reading them. This skill is no different! With a little practice, it will come naturally. Check on the web at www.multileadmedics.com for practice and links to more practice.

Handy Cutouts:

Clip these charts and post in field guides or note pages

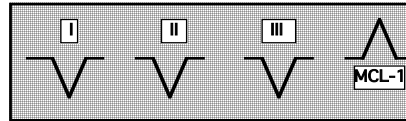
Axis	Lead I	Lead II	Lead III	Comments
Normal Axis 0 - 90				
Physiologic Left Axis 0 to -40				
Pathological Left Axis -40 to -90				Anterior Hemiblock
Right Axis 90 - 180				Posterior Hemiblock
Extreme Right Axis no man's land				Ventricular in origin



Acute MI Locator ST [↑] elevation		
Location	Leads	Recip.
Inferior (RCA)	II, III, F	I, AVL
Septal (LAD)	V1, V2	
Anterior (LAD)	V3, V4	II, III, F
Lateral (CIRC)	V5, V6, I, L	II, III, F
Posterior (RCA)	V8, V9 R>S in V1	ST [↓] V1 - 4
Right Vent. (RCA)	V4R	

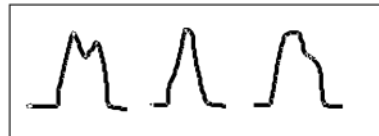
KEY 3 CRITERIA FOR VT

ERAD and Upright V1 (MCL-1)



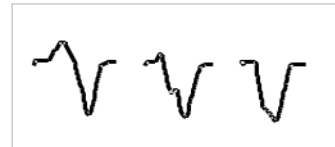
Upright V1 (MCL-1)

"BIG mountain, little mountain"
"steeple" or "fireman's hat"



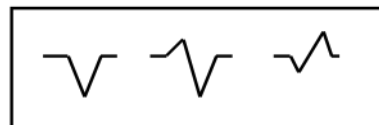
Negative V1 (MCL-1)

Fat "R" wave
Notch or slur to downstroke



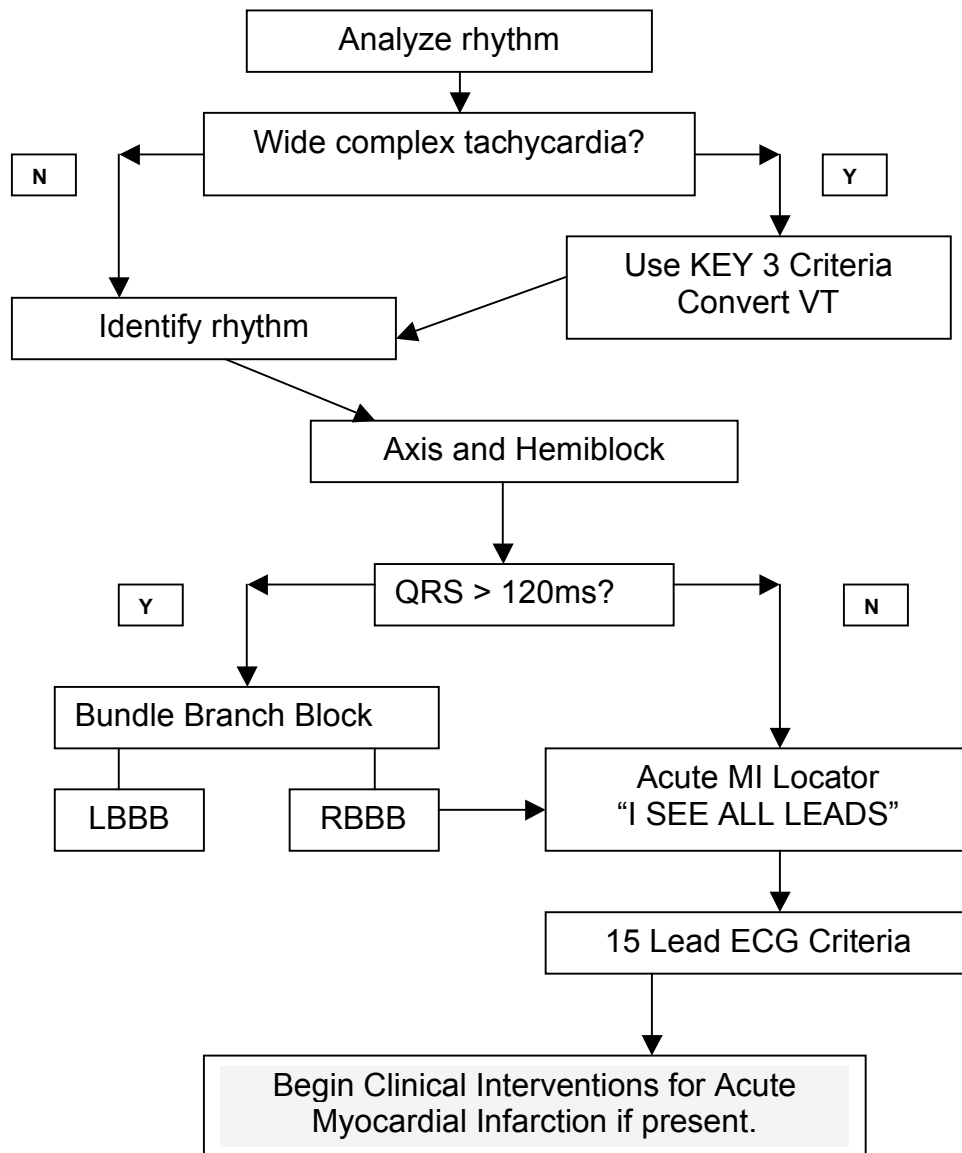
V-6 (MCL-6)

Any negative complex



Putting it All Together

When journeying to a new land, everybody needs a road map. Even if you know the area, changes make a map essential. In order to facilitate your journey through the 12 lead, I have prepared this algorithm to lead you step by step along the way.



Want to Learn More or Need to Practice?

Visit Bob's website at www.multileadmedics.com or
<http://homepage.mac.com/edutainment>

Get Bob's Book:

12 Lead ECG for Acute and Critical Care Providers: Page, B 2005 Brady/Prentice Hall

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