Nurses International Presents:

An EKG Interpretation Primer

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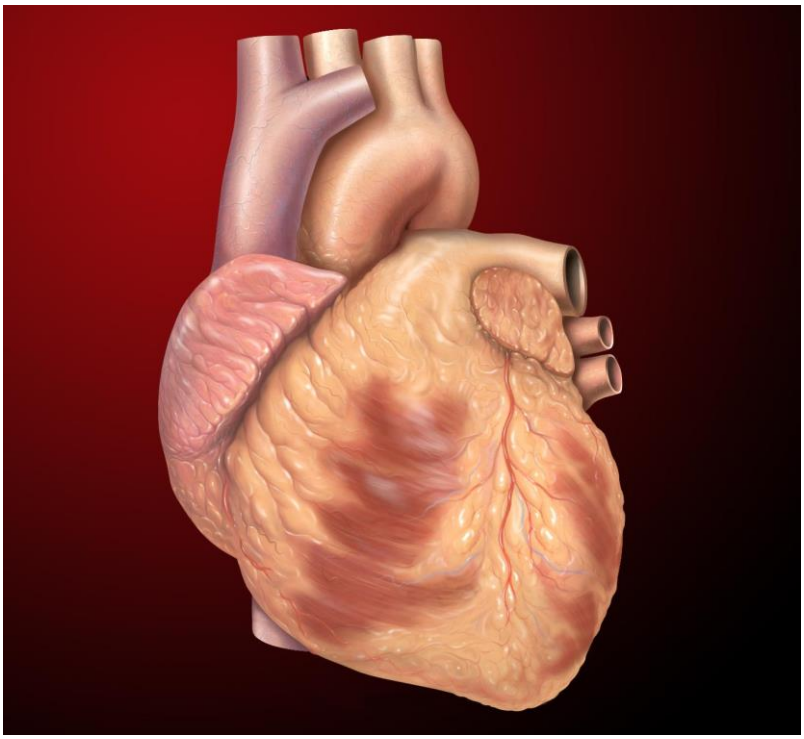
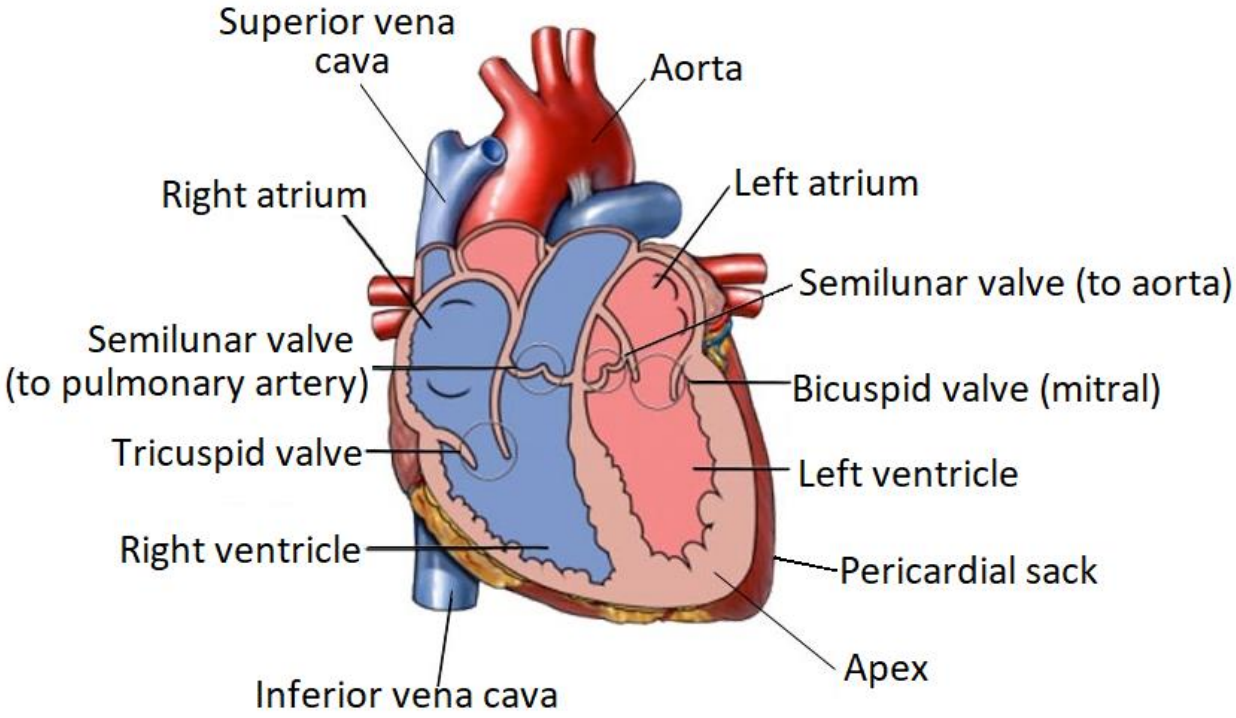


Chapter 0 - Anatomy and Physiology of the Heart

This segment is a prerequisite lecture in preparation for the EKG interpretation primer. A strong foundation in the anatomy and physiology of the heart is essential in understanding and interpreting EKGs. This segment will review that anatomy and physiology.

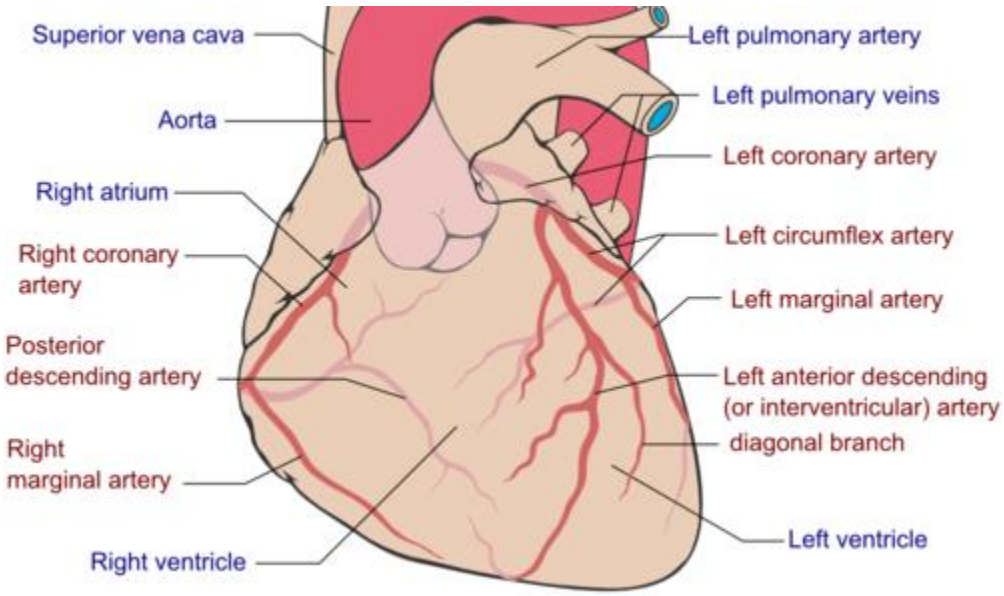


The above is a visual reminder of the structures of the heart. Of note, the tricuspid valve is also known as the right atrioventricular valve. The mitral valve is also known as the bicuspid valve or the left atrioventricular valve. Both terminology is correct. The tricuspid valve is *tri*cuspid because it has 3 flaps that seal together to form the valve, anchored along the wall of the adjacent ventricle wall. The *bi*cuspid valve only has 2 flaps.

The pulmonary and aortic valves are called semilunar valves. These valves are funnel-shaped and, while morphologically different from the cuspid valves, fulfill a similar function (preventing blood backflow). When you auscultate heart sounds with a stethoscope, the sounds you are hearing are the valves opening and closing.

Coronary Arteries

The coronary arteries provide the heart with its own dedicated blood supply. While the left ventricles do pump oxygen-rich blood to the rest of the body, the entire heart does still require their own blood supply. The coronary arteries are the first arteries that branch off the aorta.



For the time being, just be aware that there are coronary arteries, their job is to supply blood to the heart tissue, and make a mental note of where they are located. They will be revisited in a later segment.

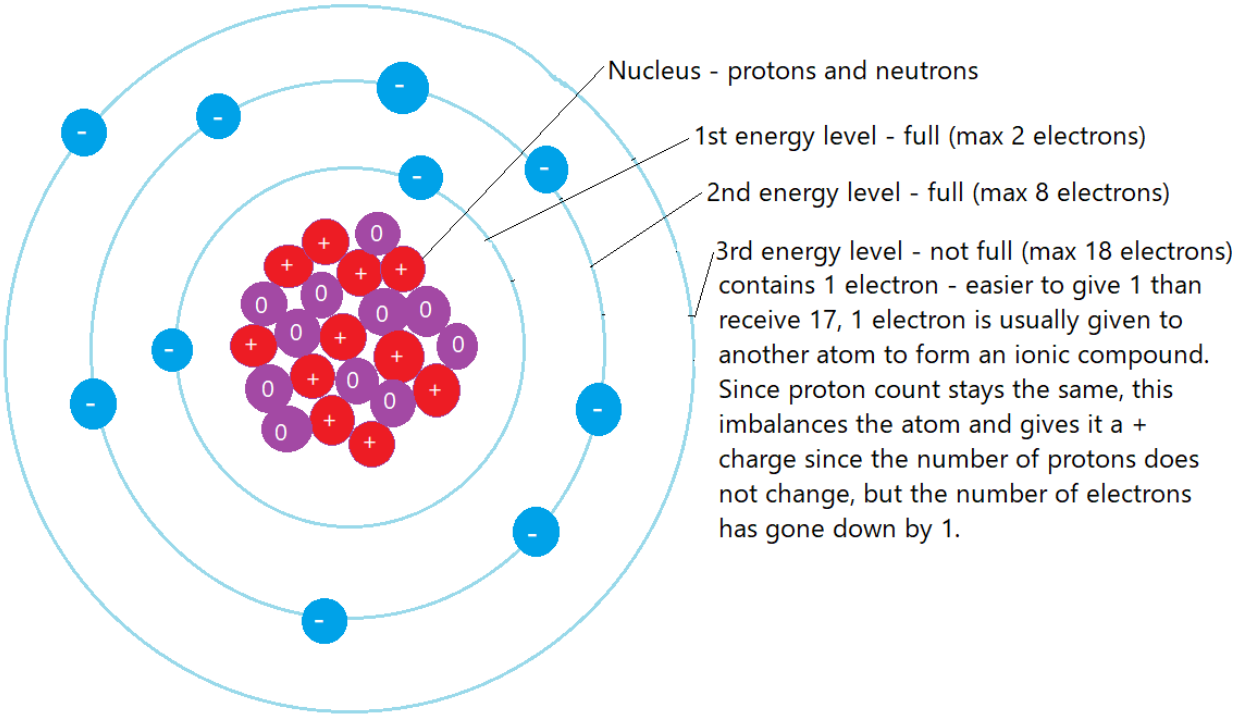
What is Electricity and Polarization?

It is first necessary to understand what electricity is. Electricity is defined as the presence and the flow of electrical charge. All forms of electricity (including the kind that is used to power devices like lights in a home) involves a flow of positively and/or negatively charged atoms from one point to another. Atoms that have a non-zero charge are called ions.

To understand what an ion is, it is helpful to understand atomic theory as it relates to the position of electrons. An atom contains a nucleus (center) made of protons and neutrons. Protons are positively charged particles, neutrons are particles that have mass but are not charged (neutral = neutron). Electrons orbit around the nucleus of the atom, much like a planet orbits around a sun. While protons and neutrons are fairly stable in their position in the atom, electrons are

constantly in motion around the outside of the atom. We can predict where they are at a given time using Bohr's theory, and use that prediction to determine how that atom is electrically charged.

In short: Each electron inhabits an area called an "energy level." Energy levels can be compared to levels in an apartment. Each level of the apartment complex has a set number of rooms, and those rooms must be filled in order to start filling the next level. The first level can contain up to 2 electrons. The second level up to 8, the third up to 18, and so on. One electron can only occupy one space, so an electron in the first energy level only counts for the first energy level; it does not get counted toward the number needed for the second energy level. We will use a sodium atom as an example:



A sodium atom contains 11 protons, 11 neutrons, and 11 electrons. According to Bohr's theory, an atom wants to have all of its energy levels completely full, and can accept electrons from other atoms or give them away so that its top-most energy level is completely full. Sodium has 2 completely full levels - the first level with 2 electrons and the second level with 8 electrons, so a total of 10 of the atom's 11 electrons have filled energy levels such that they are full. Sodium has a total of 11 electrons, so the 11th will start filling the third energy level.

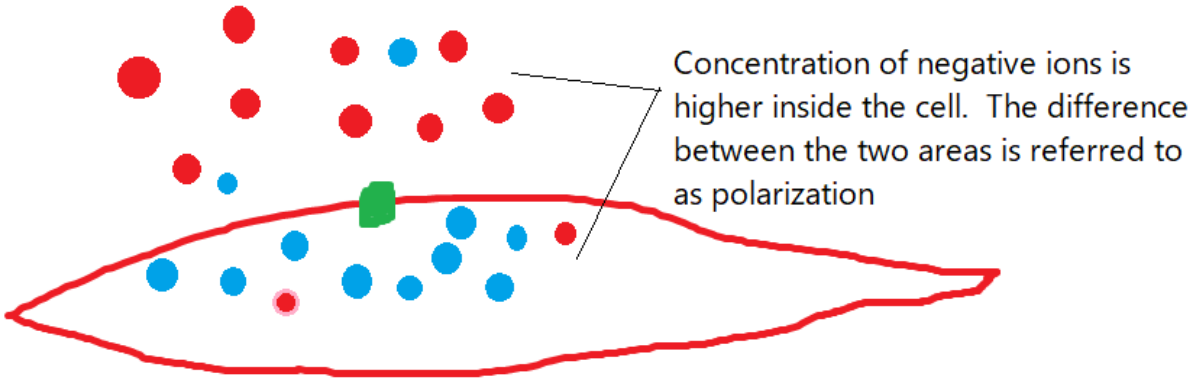
Each atom wants to have completely filled energy levels with no extra and no vacancies. The third energy level contains 18 slots, and only 1 is being occupied by electrons in the sodium atom. This electron can be "given away" and used by another atom that has an almost-filled energy level (for example, hydrogen has only 1 electron and needs 1 more to fill its first energy level). Since it is much easier for the atom to give one electron away than it is to find 17 electrons to fill its third energy level, the electron is usually given away to another atom.

Atoms normally have 0 net charge, which means the number of protons and electrons are the same. However once an electron is either given away or accepted by another atom, the balance of 11 protons to 11 electrons has changed. Now the atom only has 10 electrons. The number of protons has not changed, so that means there are 11 positive charges and only 10 negative, leading to a net positive charge of 1.

A net positive charge is written as either 1+ or just +, so a sodium ion can be written as Na+. If we examined another atom that accepts 2 electrons to fill its outer-most energy level, like sulfur, would be written as 2-, meaning it has a negative charge of 2 (because it accepted 2 electrons instead of 1). Ions that have a positive charge like sodium are called cations; ions with a negative charge like sulfur are called anions.

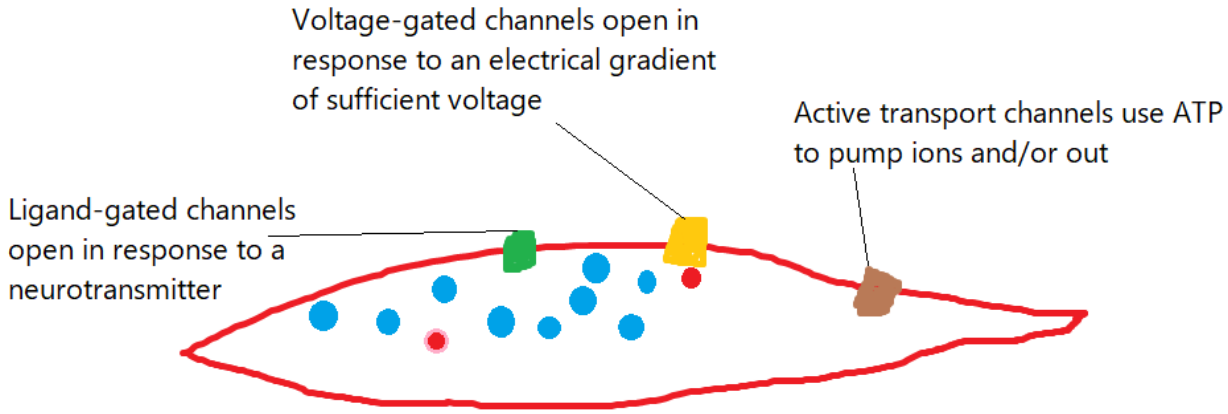
Polarization and Ion Shifts in Cardiac Muscle

When ions flow from one point to another point, it transfers energy in that direction. That transfer of energy is measurable and is referred to as electricity. Polarization refers to a concentration of positive or negative charges that are higher than other surrounding areas.



Both muscle cells and the interstitial fluid surrounding muscle cells contain cations and anions. At rest, a muscle cell is typically in a polarized state at about -75 mV. That means: there are anions whose collective charge is -75 mV within the cell, where the area outside the cell is closer to neutral or is somewhat positively charged.

Muscle cells utilize several mechanics to control the flow of cations and anions into and out of the cell which causes an electrical gradient (that is, a gradient in which there is a greater charge on one side of the cell than the other side) to form. The cell membrane contains several types of "gates" that the ions can enter and exit the cell through:



Each type of channel has its own unique purpose and function in polarizing or depolarizing the cell.

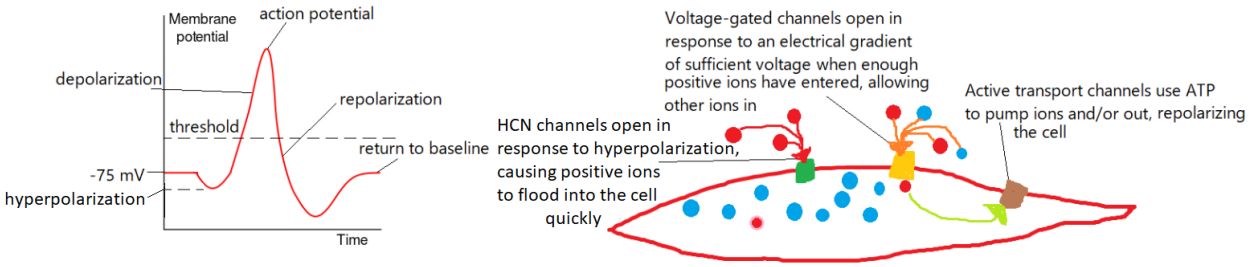
Depolarization

Depolarization is the opposite process of polarization. While polarization creates a gradient in which one area has a higher concentration of cations or anions than the other, depolarization reverses that imbalance. In muscle cells, depolarization also activates other mechanics within the cell that cause the muscle cell to contract (described later).

Depolarization occurs as a result of an action potential. An action potential is the term for a sudden change in polarization in part of the polarized muscle cell, which then spreads to the other parts of the cell. The end of an action potential is the maximum depolarization, or the furthest the cell can get from its polarized state.

In skeletal muscle cells, that outside action is a neurotransmitter opening ligand-gated channels, which increase the voltage trapped within the cell by pumping more ions into the cell. When the cell reaches a certain voltage, voltage-gated channels open in the cell membrane, allowing other ions to flow in and out, depolarizing the cell by allowing ions to flow in more quickly, reaching a near-equilibrium in charge between the outside of the cell and inside. The voltage-gated channels only stay open for a short period of time; when they close, the cell begins to repolarize, using an active transport channel like the sodium-potassium pump to move positively charged ions back out of the cell to allow the cell to return to its baseline polarized state.

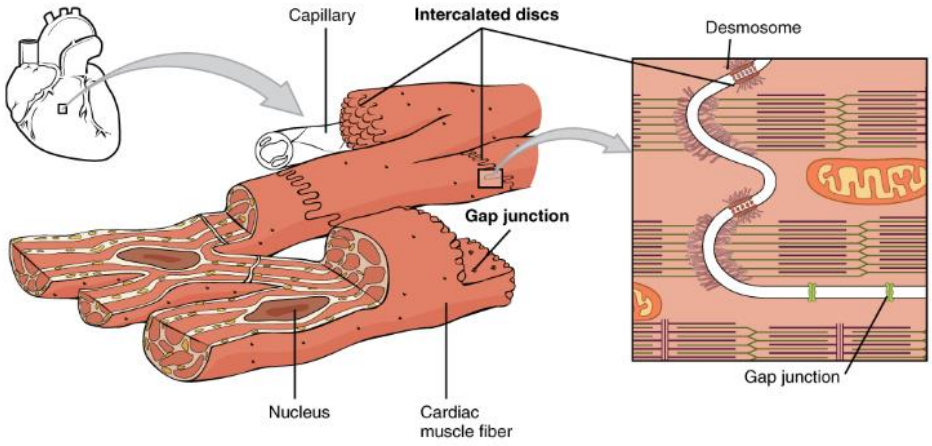
Cardiac muscle do not have ligand-gated channels, as they do not respond to neurotransmitters. Instead, cardiac muscle uses hyperpolarization activated cyclic nucleotide gated (HCN) channels, which are unique to cardiac muscle. They open in response to hyperpolarization, a state in which there is a greater-than-expected charge within the cell. In this case, it responds to negative hyperpolarization, or hyperpolarization that is measured as less than -75 mV (remember -80, for example, is a number less than -75 even though it represents a greater negative charge). They are poorly selective, which means they allow numerous types of ions in and are not very specific for which ones can flood in. Their poor ion selectivity means the depolarization process starts very quickly, as many types of cations are allowed to flood into the cell all at the same time.



Another specific type of channel that is understood to be cardiac-specific is called the fast Na+ channel. This is one type of voltage-gated channel that is selective for sodium and allows very rapid flow of sodium into the cell at a very specific voltage that occurs in the early to mid-phase of depolarization. The fast Na+ channel opens in response to a small amount of positive electrical stimulation but closes again when it exceeds a certain amount of positive electrical stimulation, well before action potential occurs. This allows rapid increase in depolarization, but slows the depolarization process immediately before action potential, allowing adjacent cardiac muscle cells to contract in conjunction. Closing the sodium channel before too much sodium flows in also prevents cell lysis: water tends to follow sodium, and too much sodium can pull too much water into the cell, stretching it and causing it to burst.

Unique Attributes of Cardiac Muscle

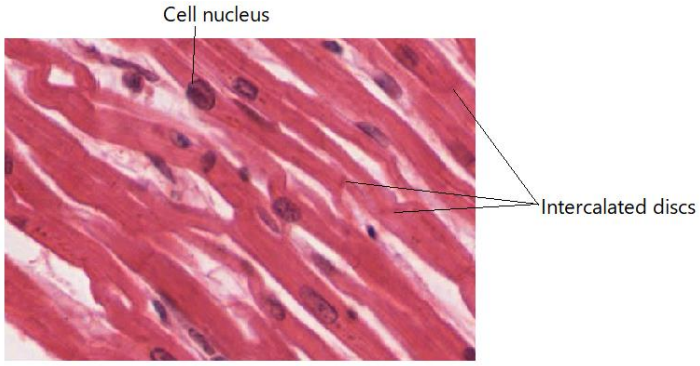
Cardiac muscle tissue has several unique attributes that allow it to function effectively specifically in the heart. The first unique attribute cardiac muscle has is its ability to excite and stimulate its own depolarization without outside intervention. Additionally, voltage that stimulates depolarization in one cell can spread to other adjacent cardiac muscle cells. Cardiac muscle can do this because it contains a physical conductor between its adjacent cells in a space called an intercalated disc:



The intercalated discs are special connections between the cardiac muscle tissue that allows it to have branch-like connections to multiple other muscle fibers. Within the intercalated discs are areas called gap junctions, which allow electrical currents to be transferred to other cells. One cardiac muscle cell can stimulate the voltage-gated channels in adjacent cells connected by intercalated discs, commencing the depolarization process of the other cells in synchrony with the originator. This allows multiple muscle fibers to go through depolarization and reach action potential together as a unit.

The branch-like pattern that almost weaves cardiac muscle cells together is important as well because it allows the muscle cells to contract together in a net-like pattern, rather than in a strictly linear pattern. This net-like network of muscle tissue is more effective at pumping and pushing blood.

Desmosomes are units that anchor the cardiac muscle tissue to one another. This prevents them from pulling apart and tearing at the junction site as they contract all at the same time.



Electrophysiologic Nodes of the Heart

The heart has two groups of specialized cells whose job is to start action potentials, though any heart muscle cell can start its own action potential (the concept of other areas of the heart starting their own action potentials will be covered in the EKG lecture series).

The specialized cells that start and regulate action potentials are located in these parts of the heart:

The purple dot represents the SA node. The SA node is normally the dominant pacemaker, which means depolarizations that cause the cardiac muscle cells to contract originate here. The SA node normally starts the entire depolarization process, and it happens in synchrony because, as discussed above, cardiac muscle cells trigger depolarization in neighboring cells when they depolarize themselves. The position of the SA node is also ideal; it starts at the top and flows in a downward fashion, causing the atria to contract from top to bottom. This top-to-bottom contraction is important because it allows blood to be efficiently pumped through the bicuspid and tricuspid valves and into the ventricles.

The green dot is the AV node. The AV node acts as a gatekeeper between the atria and the ventricles. There is an electrical barrier between the atria and ventricles; the only way for the electricity to travel to the ventricles is through the AV node. The AV node, has fewer HCN channels than the SA node, making it both less likely to be the pacemaker (though it can also take over as the dominant pacemaker, this is considered abnormal and will be covered in greater detail in a later segment) and causing it to “hold” impulses for a brief fraction of a second.

The pause between the atrial contraction and the ventricular contraction is important because it allows the blood in the atria to be pumped into the ventricles, filling them completely before they start to pump. This increases stroke volume (volume of blood pumped per ventricular contraction), which improves overall circulation compared to if the ventricles start to contract before the atria have finished pumping.

The ventricles contain a unique electrical conduction system called the bundle of His. The bundle of His is insulated, which means it protects the surrounding cells from the depolarization. This is important because, unlike the atria, the ventricles need to contract in a fashion that is from bottom to top. The bottom-to-top fashion is important because, anatomically, the semilunar valves are located at the top of the ventricles. The ventricles need to be able to push blood toward and out of the superiorly-located semilunar valves into the pulmonary vasculature and to the body via the aorta, so the insulation around the bundle of His allows electricity to travel to the bottom of the heart before it starts the depolarization of the ventricles at the apex of the heart.

Chapter 1 - EKG Basics and Terminology

EKG Terms

Depolarization: An electrical shift that takes place within muscle cells, causing the muscle to reach action potential and ultimately contract. The electrical shift is measurable via the EKG, and allows the clinician to visually predict what the heart is doing and in what pattern. All muscle cells go through depolarization when they contract.

Repolarization: A secondary, smaller electrical shift that takes place after a depolarization. This prepares the cells to be able to depolarize again. Cardiac, skeletal, and smooth muscle cells all go through repolarization.

Baseline: Also called the **isoelectric line**. The EKG graph line when there is no electrical activity happening.

Artifact: Interference from movement of the other muscles during an EKG reading, causing the EKG to look distorted. Usually caused by movement, heavy breathing, or talking during the reading. All muscles produce electrical activity when they contract (see above - depolarization and repolarization) and any muscle movement during an EKG reading will be visible on the graph even if it does not originate from the heart.

Automaticity: The ability of a cardiac muscle cell to spontaneously depolarize and reach action potential without external stimulation from a nerve or hormone. All cardiac muscle cells possess some degree of automaticity.

Irritability: The concept that individual heart muscle fibers, when deprived of a nutrient needed to function optimally (ie: oxygen, glucose, certain electrolytes), will attempt to contract without outside stimulation from another muscle cell AND without coordination from other parts of the heart. Irritability is a frequent cause of EKG abnormalities.

Pacemaker: The part of the heart from which depolarization originates. The SA node is normally the dominant pacemaker, though any part of the heart can be the pacemaker as all cardiac muscle tissue possesses automaticity.

SA Node: Sinoatrial node - a node located in the atrium from which the electrical impulse stimulating depolarization normally originates. The SA node is

the dominant pacemaker in a normally functioning heart, and depolarizes at a rate of approximately 60-100 BPM at rest.

AV Node: Atrioventricular node - a node located between the atria and ventricles that regulates electrical impulse conduction between the chambers. The AV node's primary function is to hold the electrical impulse originating from the SA node for a fraction of a second before it allows the electricity to continue into the ventricles. The AV node is also capable of functioning as the dominant pacemaker, though it is not the dominant pacemaker in a normally functioning heart.

Bundle of His: An insulated bundle of muscle fibers that extend from the AV node to the apex of the heart. The insulation around the bundle of His allows the electrical impulse to flow to the bottom of the ventricles before it stimulates depolarization in the ventricles. This enables the ventricles to pump from the bottom toward the top and is an important feature given the anatomic location of the semilunar valves near the top of the ventricles.

Rate vs Rhythm: Rate refers to the number of QRS complexes per minute (ie: a rate of 65 indicates there are 65 QRS complexes every 60 seconds). Rhythm refers to the regularity of the QRS complexes. If the QRS complexes occur at regular measurable intervals, the rhythm is called regular. If the QRS complexes occur at irregular or varying intervals but there is a pattern to the irregularity, it is referred to as "regularly irregular." If the QRS complexes are irregular without a discernible pattern, it is referred to as "irregularly irregular." Noting patterns or lack thereof in QRS irregularity can help you differentiate certain rhythms.

Electrode: The object that is adhered to the patient's chest which connects to a wire, ultimately connecting to the computer that produces the EKG graph.

Lead: The measure of electricity from a particular electrode. Leads can be positive (measuring electricity flowing toward them), negative (measuring electricity flowing away from them), unipolar (measuring both), and virtual (a "lead" that is not physically present on the patient calculated mathematically from the input from 2 other leads, meant to represent what the electrical activity looks like directly between the other 2 leads).

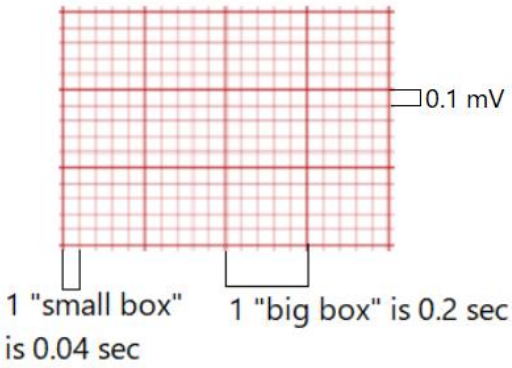
Defibrillation: An electrical intervention used to correct certain arrhythmias. Defibrillation is used when a palpable pulse is absent but a

"shockable rhythm" is present. We will review which specific rhythms are shockable during the interpretation sections. Defibrillation stimulates depolarization in the entirety of the heart at the same time, allowing it to "reset" as it repolarizes, ideally giving the SA node an opportunity to regain control as the dominant pacemaker. Defibrillation can be accomplished externally, with pads on the chest wall, or internally with a surgically implanted defibrillator device.

Cardioversion: A type of electrical intervention used to correct certain arrhythmias. Cardioversion is a type of defibrillation that is timed exactly with the heart rhythm to attempt to stop abnormal activity and allow the SA node to regain control as the dominant pacemaker. Cardioversion uses a smaller amount of electricity than defibrillation. Also called synchronized cardioversion.

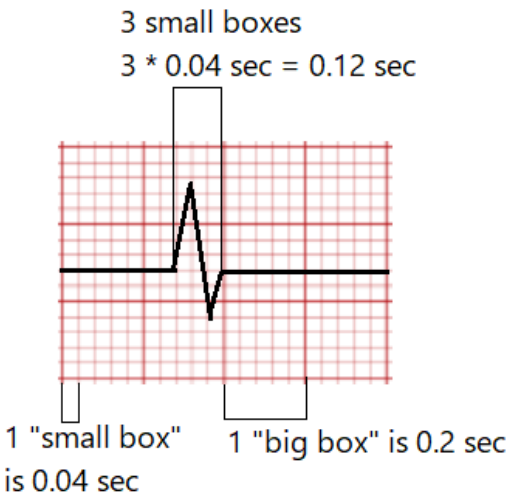
Pacing: This term refers to the regulation of cardiac electrical activity and depolarization using an external source of electricity. This can be internal, as with an implanted pacemaker, or external with percutaneous pacemakers. There are numerous types of pacemaker, including atrial pacemakers that pace only the atria, ventricular pacemakers that pace only the ventricles, and pacemakers that pace both the atria and the ventricles.

Anatomy of an EKG Graph



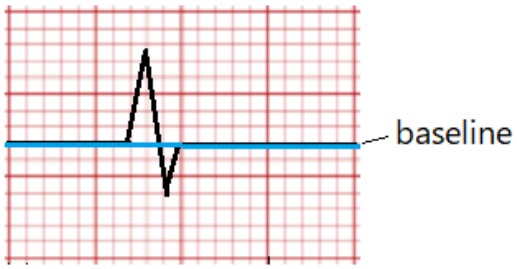
An EKG is a graph of the heart's electrical activity. The horizontal axis represents time. The vertical axis represents voltage. Our primary focus with measurements will be on time (the horizontal axis), but it is sometimes helpful to understand and measure voltages if you go into more advanced EKG interpretation.

First we will learn to measure time. This is helpful when you need to know how long it takes for a particular wave to occur. A “small box” denoted by the thinner lines represents 0.04 seconds of time. The “big box” is denoted by the bold lines and is comprised of 5 small boxes and represents 0.2 seconds of time.

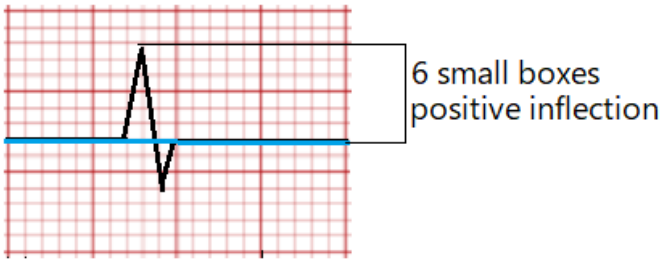


In this example, we will measure the wave complex drawn on the graph. We will start at the beginning of the complex (where it deviates upward from the baseline, in this example) and end at the end of the complex (where it returns to the baseline). It is acceptable to estimate to the nearest small box - EKG wave forms rarely line up exactly on the graph lines. This example measures the complex at approximately 3 small boxes wide. Each small box represents 0.04 seconds, so we know this wave on the EKG graph took place in 0.12 seconds.

It is a good idea to get comfortable with measuring waves in this manner and calculating the time per wave, as it will help your EKG interpretation greatly.



In order to correctly measure voltage, we have to first find the baseline of the EKG. The baseline refers to the straight horizontal "flat line" on the graph. A flat horizontal line on the EKG represents no electrical activity. The line is not always perfectly flat on a real EKG, but it should be fairly close.



For the time being, it is not important to measure how much voltage each wave has (though it can be measured in the same way as time can - by counting the small boxes and multiplying by 0.1). It is important, however, to note if the inflection is above the baseline or below the baseline. A positive inflection refers to a wave that goes upward above the baseline. A negative inflection refers to a wave that goes below the baseline.

For this example, the wave has both a positive and a negative inflection. This "wave" is actually 2 waves (also referred to as a complex), as it has 2 peaks - one positive and one negative. You can measure waves individually or you can measure them together as complexes as we did above when measuring time. An individual wave is measured from where it leaves the baseline to where it returns to the baseline - even if it continues into a negative wave as this one does.

Chapter 2 - Electrical Activity of the Heart and Basic Wave Forms on an EKG

Purple dot = Sinoatrial (SA) node

Green dot = Atrioventricular (AV) node

Blue lines: Bundle of His, purkinje fibers

Any part of the heart can act as the heart's pacemaker (pacemaker in this context refers to the part of the heart in which depolarizations begin). The normal default pacemaker is the SA node because the SA node has the fastest resting pacemaker rate. If the SA node fails, any other portion of the heart can take over as the pacemaker. This can act as a fail-safe mechanism, as it means the SA node can malfunction and not cause immediate death.

Each portion of the heart has a different pacemaker rate. This means that, if a particular part of the heart becomes the default pacemaker instead of the SA node, it will typically set the resting rate at the following heart rates:

SA node: 60-100 BPM

Atria: 60-100 BPM

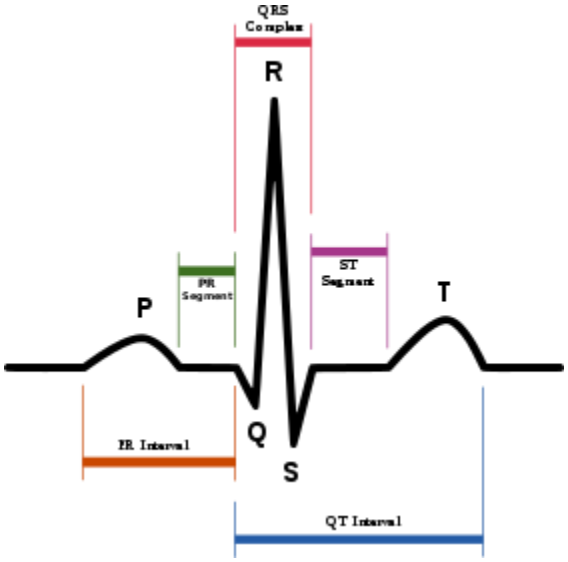
AV node: 40-60 BPM

Ventricles: 20-40 BPM

There are a variety of reasons the pacemaker of the heart may be different from the SA node, including irritability (defined above) and failure of higher nodes. The SA node is typically the default because it is the fastest - other nodes can take over and override the SA node if they contract more rapidly than the SA is going. This occurs with premature contractions (covered in the next section) and

sometimes with other rhythms like ventricular tachycardia. Other areas of the heart like the atria, AV node, and ventricles can also take over as pacemaker in the event the SA node fails.

EKGs measure electrical activity in the heart. They do NOT measure actual movement, only the electrical impulses that cause muscle contractions. In a normal heart, the electrical impulse starts in the SA node, travels from there through the atria to the AV node. The electrical impulse is held there briefly, then is allowed to continue on through the bundle of His to the purkinje fibers into the ventricles.



P wave: The electricity in the heart flowing from the SA node through the atria to the AV node; stimulates the atria to contract

PR segment: The pause in electrical activity when the AV node is holding the electrical impulse, allowing the ventricles to fill after the atria contract

PR interval: Measured from the beginning of the P wave to the beginning of the Q; should be 0.12 to 0.2 seconds (three small boxes to one big box)

QRS complex: Electricity in the heart flowing from the AV node down the bundle of His into the purkinje fibers; stimulates the ventricles to contract

T wave: The repolarization of the ventricles following contraction

QT interval: Measured from the beginning of the Q to the end of the T; should be about .45 seconds or less, can vary depending upon the overall heart rate. There is a formula that can be used to calculate an appropriate QT interval based on heart rate that will not be covered today

ST segment: Measured from the end of the S to the beginning of the T

How do I sort out all this information?

1) **EKG rate**

Tachycardia – Rate >100

Bradycardia – Rate<60

Most EKG machines will automatically provide a rate provided they are set up correctly. EKG machines usually measure the QRS complex rate as the overall rate. The easiest alternative way to measure rate is to count the number of QRS complexes in a 6 second strip, then multiply that count by 10. There are other methods that are more precise in calculating the rate (which will be reviewed in a later chapter), but the focus of this overview of EKG interpretation is in clinically relevant interpretations. The difference between a rate of 70 and 77, for example, is of minimal clinical consequence in most settings.

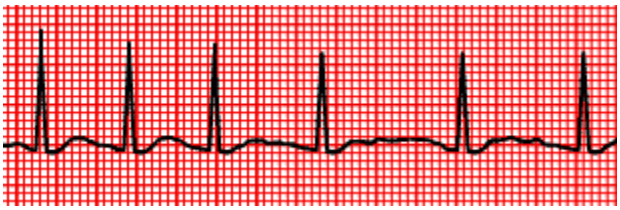
As noted above, "tachycardia" and "bradycardia" rates can vary somewhat depending upon where the electrical activity is originating from, but generally the terms refer to heart rates >100 or <60, respectively. Some patients do have a tachycardic or bradycardic rate at baseline.

2) **Is there a P wave for every QRS complex? Is there a QRS complex for every P wave?**

The ratio of P waves to QRS complexes should be 1:1 in a normal rhythm (discussed in more detail in the next segment). Noting a P to QRS ratio that is different from a 1:1 ratio will help you discern certain rhythms.

3) **Do the P waves come at consistent intervals or "march out"? The QRS complexes?**

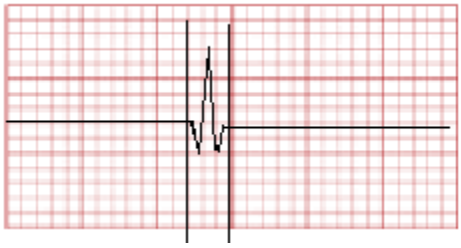
"March out" means they all occur at consistent, regular intervals. It is most accurate to use a pair of calipers to measure this, but you can often see clearly when it does not march out in regular intervals, as with the irregular QRS complexes on this EKG:



P waves may be hidden underneath the larger QRS complexes, though this is generally a consideration primarily with particular types of heart blocks (covered in next section). P waves may also be entirely absent, which should be noted and will help you differentiate the rhythm.

4) Is the QRS complex wide or narrow?

A normal QRS complex is 0.12 seconds or less, which is referred to as a narrow complex. 3 of the small EKG boxes is equal to 0.12 seconds.



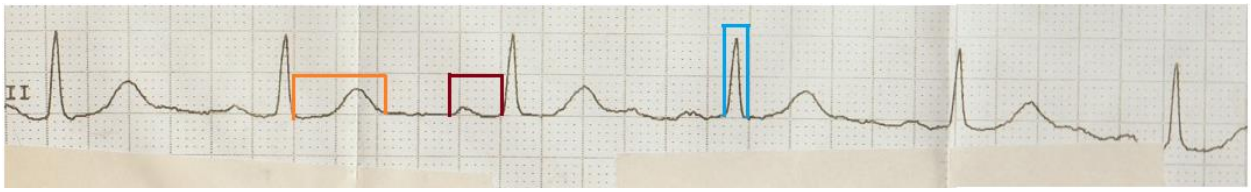
A wide QRS complex is considered anything above 0.12 seconds. You may see certain rhythms referred to as "wide complex" or "narrow complex"; these terms refer to the width of the QRS complex and will help you distinguish certain rhythms. We will discuss what these types of QRS complexes mean with regards to physiology in another segment.

Putting it all Together

Rate	
Rhythm	
P waves	
PR interval	
QRS complex	
ST segment	
T waves	

It is often helpful to have a diagram you use to measure and interpret each piece of the EKG to help you get into the habit of thinking about the parts. The above is one example of a diagram you may use that includes the most common information you will need to discern from an EKG tracing.

Example:

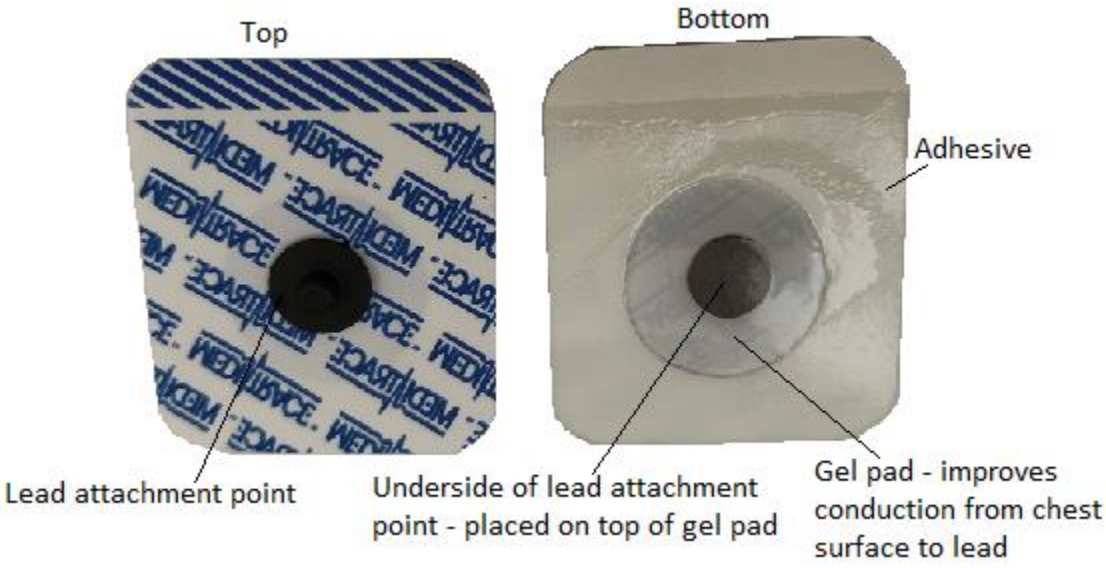


Rate	60
Rhythm	Regular
P waves	Upright, regular
PR interval	0.24 sec
QRS complex	0.12 sec
ST segment	0.44 sec
T waves	Upright

In this example, the rate is measured at 60 as there are 6 QRS complexes in this 6 second strip. The rhythm is regular because the QRS complexes occur at regular intervals. The P waves are upright and occur at the same interval every time. The PR interval, QRS complex, and ST segments are 0.24 seconds, 0.12 seconds, and 0.44 seconds in duration. The T waves are upright. In a later segment, we will discuss how to apply this information to rule sets that determine what rhythm an EKG is considered to be.

Chapter 3 - EKG Machinery and Setup

The EKG Electrodes

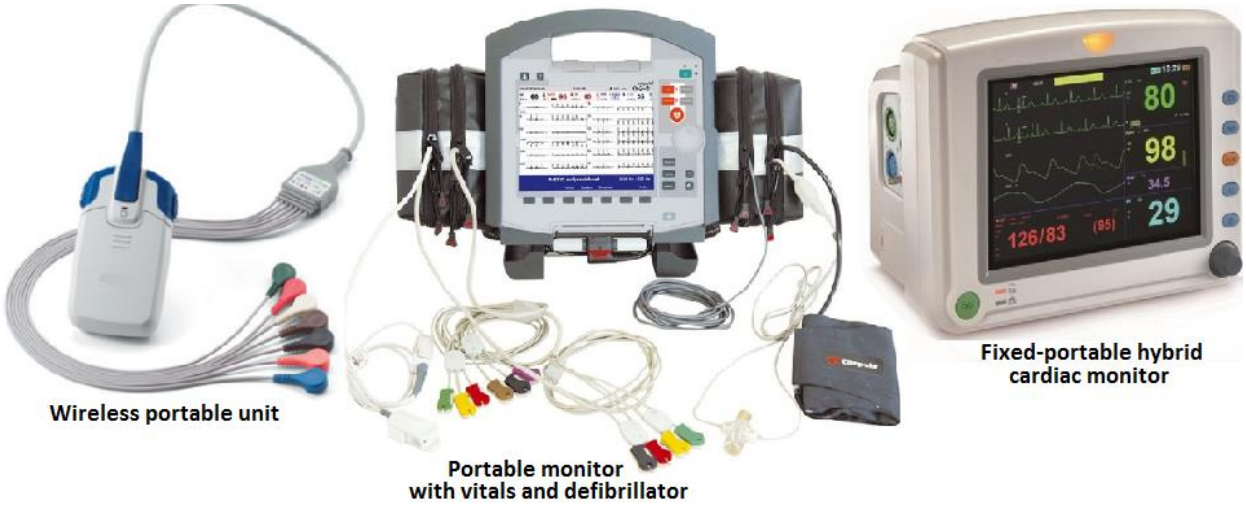


Note: There are many, many types of EKG electrode patches. Some are a simple sticker where the adhesive and gel pad are combined, some have a small metal button or clip where the wire for the leads attach. This EKG learning module does not endorse any specific brand or type of EKG electrode or patch.

The gel pad on the above EKG electrode improves conduction from the surface of the chest to the lead. Most electrodes have a similar compound and mechanism to improve the lead's ability to detect electrical activity.

The electrode also incorporates an adhesive that sticks the pad to the chest wall. The adhesive alone is usually adequate to adhere the electrode well, but a poorly adhered electrode will not detect electrical activity adequately (or at all). Common confounding factors that can make it harder for the electrode to adhere to the chest wall include excessive lotion or topical oil used on the skin, extensive sweating or diaphoresis, and hair. It is very acceptable and encouraged to shave or clip the chest of a patient who needs an EKG but has too much hair to allow the electrodes to stick. It is also acceptable to use tape or other adhesives to reinforce the electrode's adhesive.

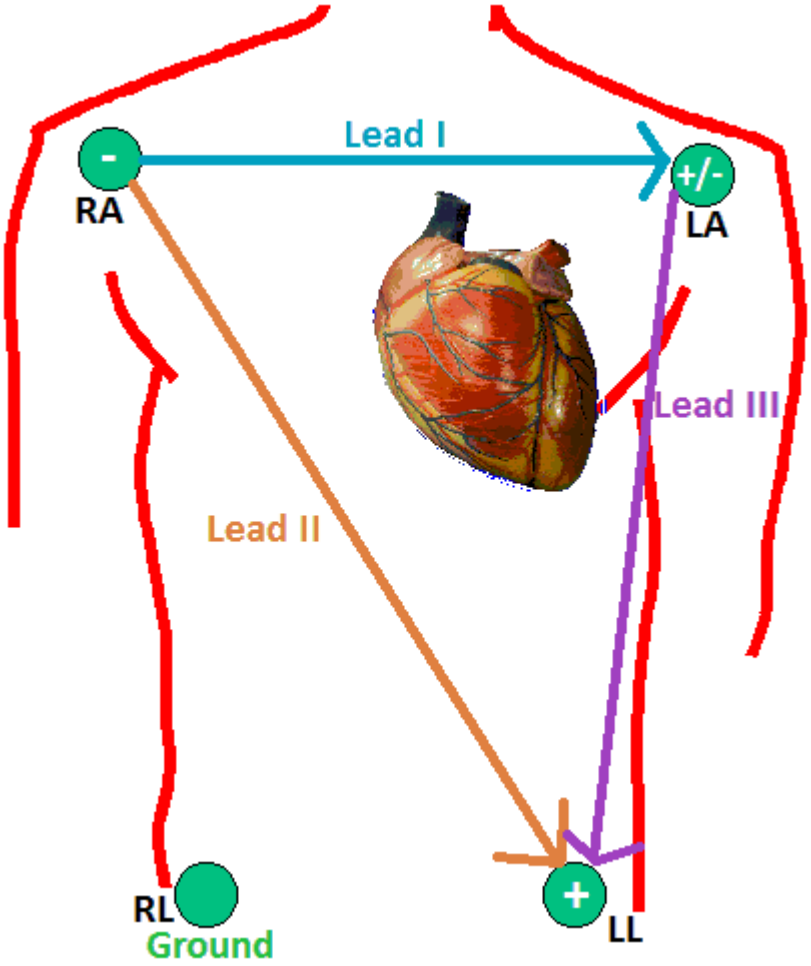
The EKG Machine



There are many, many types of monitors that serve a variety of purposes. Some units are designed for monitoring purposes only, others are multi-functional and can include many features like defibrillation, external pacing, and monitoring of other clinical data like blood pressure and end tidal CO_2. This EKG interpretation lesson series does not endorse any one EKG machine type or brand over the others.

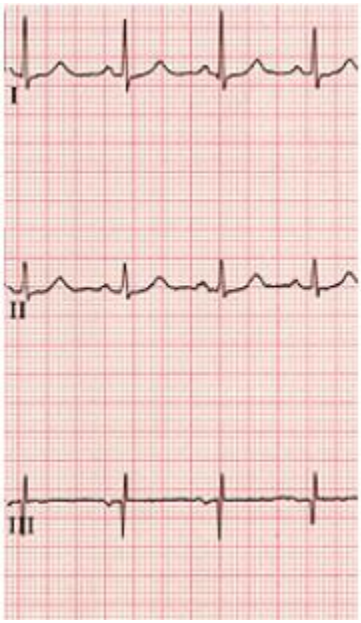
While the design of the machines are very different, their purposes as EKG monitors are all functionally the same: they all measure and interpret the flow of electrical activity. This course does not endorse or recommend any specific type of EKG monitor or machine over others.

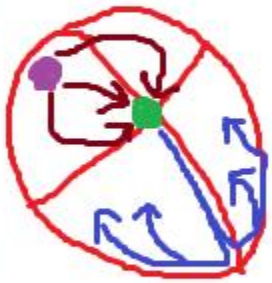
Most hospitals monitor patients via at least 3 leads simultaneously. An EKG lead is a particular electrical "view" of the heart and is representative of flow of electricity toward and away from 2 points in the heart. The setup looks like this:



Each lead is named and will be labeled: RA, LA, RL, and LL. The easiest way to remember this is RA = right arm, LA = left arm, RL = right leg, LL = left leg. The leads can be placed on the extremities or as is pictured on the corresponding corner of the trunk.

Lead II is the most commonly interpreted with regards to rhythm and is usually the first lead students learn to interpret. It goes diagonally from the right upper chest to the left lower chest in the same positions a defibrillator or AED would be in. Lead I goes laterally across the chest, measuring electricity from a positive electrode on the left upper chest and a negative electrode on the right upper chest. Lead III measures vertically across the left chest wall. Its negative electrode is the left upper chest and its positive electrode is the left lower chest. Notice that the positive electrode for lead I is the same as the negative for lead III - every physical electrode placed is capable of measuring both positively and negatively in the way the left upper chest electrode does.



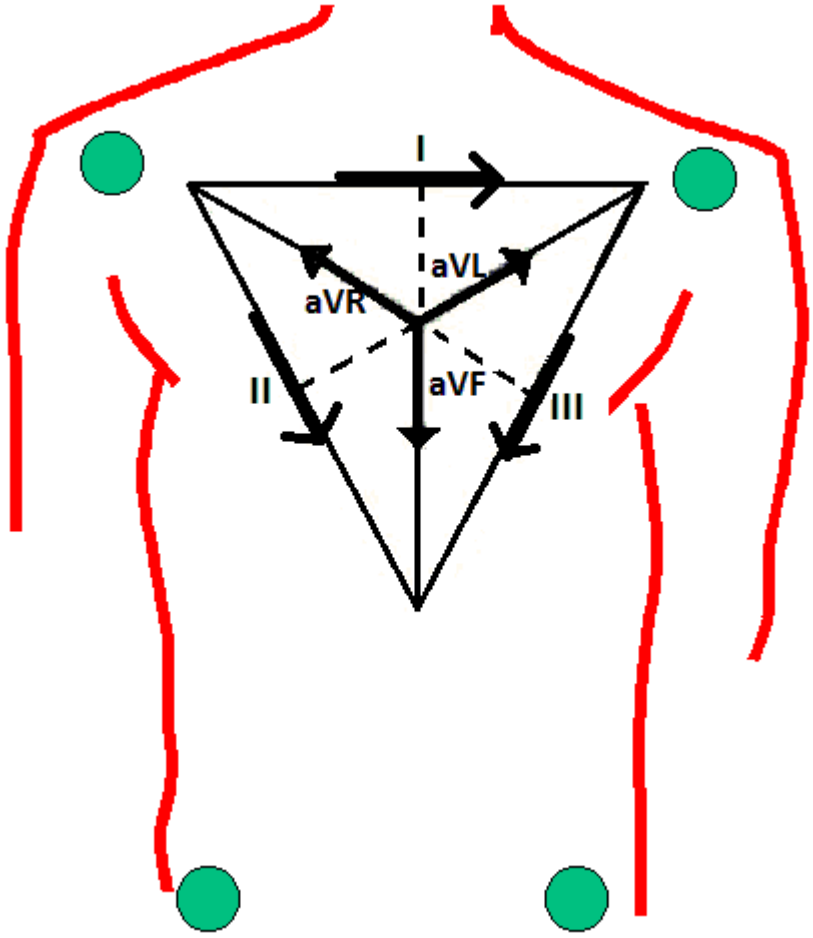


The above picture is normal sinus rhythm in leads I, II, and III. Notice the differences for each. The P waves in leads I and II are upright, as electricity in the heart flows primarily toward their positive leads. The P wave in lead III however is biphasic and somewhat flattened. That is because electricity flows toward both the positive and negative electrodes (as the electricity flows in a down-left manner, and both positive and negative electrodes are to the left of the heart). The QRS complexes have similar morphological differences due to the differences in lead positioning.

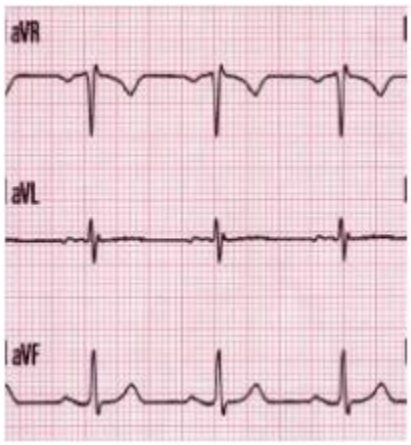
Einthoven's Triangle

A standard 12 lead EKG measures 12 "views" of the heart using 10 electrodes. 6 of the leads are bipolar, which means they have both an electrode measuring both positive and negative activity. Thanks to mathematical calculations, these 6 bipolar leads only require 4 electrodes: the 3 pictured above and a ground electrode, which helps remove interference and artifact (the above setup is possible without the ground electrode). 3 of the leads are considered to be "virtual leads," or leads that are not physically on the patient but have been

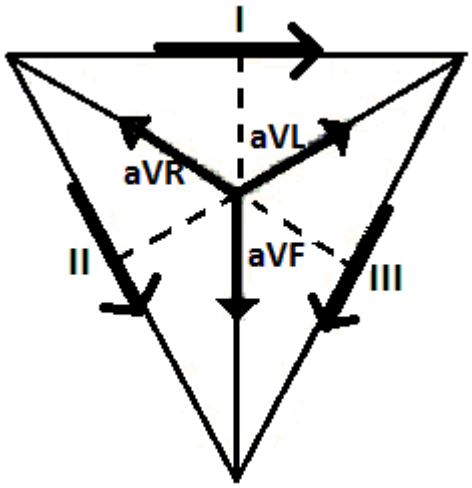
calculated mathematically based on the 3 existing electrodes. The triangular arrangement of electrodes is referred to as Einthoven's triangle.



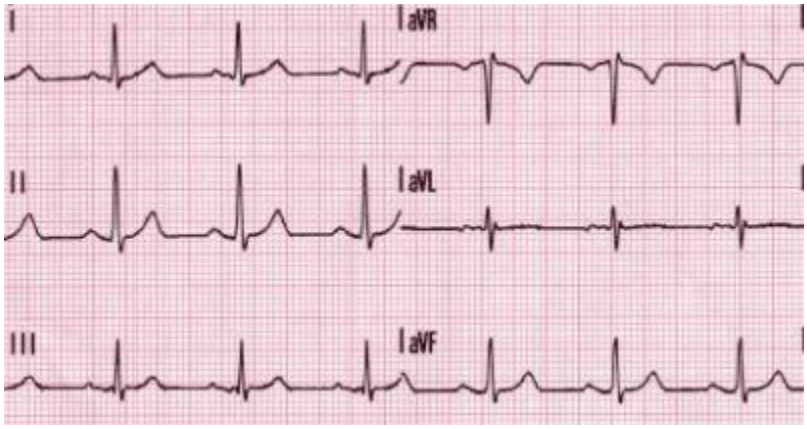
The virtual leads are aVL, aVR, and aVF. You can remember which is which by remembering that VL is the virtual left lead, VR is the virtual right lead, and VF is the virtual in**F**erior lead. It can be difficult to visualize, but the positive electrode for aVR is RA, the negative electrode is a virtual electrode that is directly between LA and LL. This lead is calculated using a formula based on the electrical activity tracked on the LA and LL electrodes. Similarly, aVL uses LA as a positive electrode and uses a virtual electrode between RA and LL that is mathematically calculated as a negative electrode. The aVF lead uses LL as a positive electrode and a virtual negative electrode between RA and LA.



Above is a normal sinus rhythm in leads aVR, aVL, and aVF. Notice that aVR is almost a perfect inversion of lead II. Refer to Einthoven's triangle:



Notice that lead II and aVR are almost (but not completely) parallel in their orientation, but their direction is opposite? The negative lead for lead II is the positive lead for aVR, and the virtual lead between LL and LA is the negative lead for aVR. Lead LL is the positive lead for lead II. Compare all 6 of the bipolar leads in this normal sinus rhythm EKG:



II and aVR are almost perfect inversions, which makes sense when you consider the lead orientation as we did above. II, III and aVF are also very similar, which also makes sense because the LL lead is the positive electrode for all 3 of these leads. The negative virtual electrode for aVF is directly between the negative electrodes for II (RA) and III (LA). Notice that the morphology of the entire aVF lead is essentially an intermediary of II and III: the P wave for aVF is taller than it is in lead III but shorter than it is in lead II. The QRS is complex has an amplitude (height) that is taller than III but shorter than II, the S in particular is not as low in amplitude (deep) as in lead II but is lower than in lead III. The T wave is also larger than in lead III, but smaller than lead II.

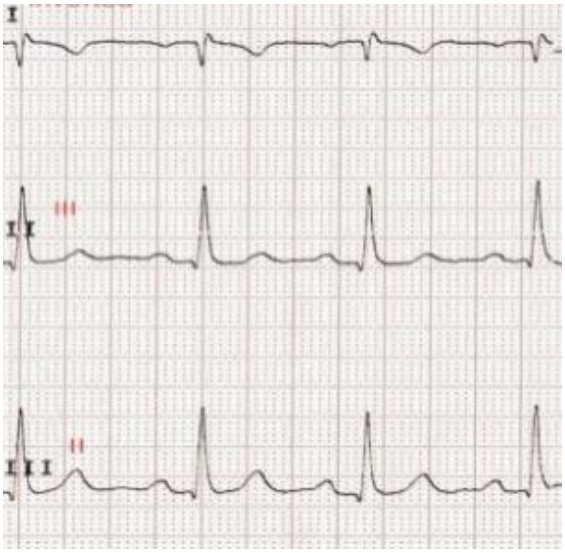
aVL is a stranger lead if directly compared to I, II, and III. While it does have some things in common with lead I, including a shared positive electrode (LA), its QRS complex is more biphasic (goes both positively and negatively) than lead I. The P wave is also flatter, and the T wave is barely visible. This has to do with the placement of the negative virtual electrode, between the RA and LL electrodes; the electricity flows simultaneously in both directions, and the amount of electricity going toward both electrodes is closer to equal than it is in lead I. This leads to flattened P and T waves because simultaneous positive and negative electrical flow registers on the EKG graph as flat. This is because the computer balances the negative against the positive, so for example electricity measuring 3 mV going toward the positive electrode would normally register as an upward inflection on the EKG that is 3 small boxes high. However, if there is also 3 mV of electricity going toward the negative electrode at the same time, the computer interprets this as 0 neg amplitude because the 3 mV flowing towards the negative electrode cancels out the 3 mV flowing towards the positive.

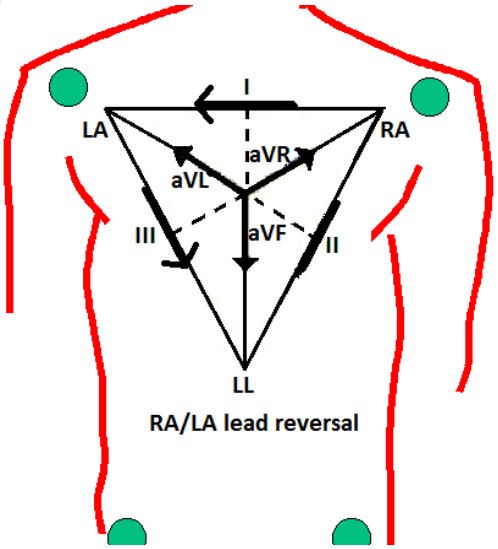
EKG interpretation in multiple leads can be more challenging because there is more to consider regarding electrical flow, directionality, and how the leads interpret the electrical flow. It is important to be able to recognize at least basic features of the EKG in each lead, as well as what is normal for each lead, because most arrhythmias need to be confirmed in multiple leads. Confirmation in more than one lead is important because EKG machines, electrodes, the wires themselves, and placement of electrodes can all cause abnormal readings, and confirming an arrhythmia in another lead is done to ensure an arrhythmia is, in fact, an electrical abnormality with the heart and not a mechanical malfunction or human error in lead placement.

Lead Reversals

Human error is common in obtaining EKGs, and while lead placement is not always a precise measurement, it is important to double check the correct wires are connected to the correct electrodes. If you accidentally mixed 2 of your leads up, there are also morphological changes to the EKG that are predictable, identifiable as a lead reversal, and can be cause for alarm if they are not correctly identified as a lead reversal.

If you reverse the LA and RA leads, for example, lead I inverts:

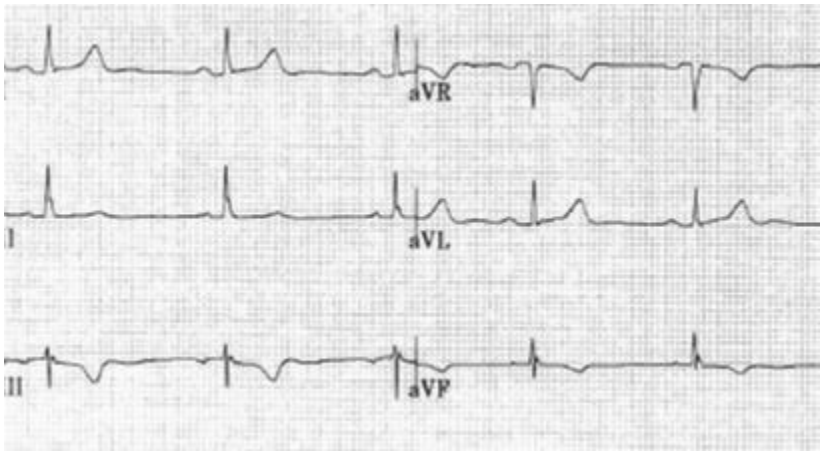




This is because the negative RA electrode is now in the LA position, and the positive LA electrode is now in the RA position in error. You will also notice that

leads II and III have switched: lead III's negative electrode, LA, is now in the RA position (normally lead II's negative electrode), and lead II's negative electrode RA is now in the LA position. The differences in leads II and III are not particularly marked in this example of lead reversal, but you will notice that the P and T waves in lead III are taller than they are in lead II which is normally opposite. The virtual leads aVL and aVR also reverse (not pictured in EKG graph), so aVL will look like aVR normally does and vice versa.

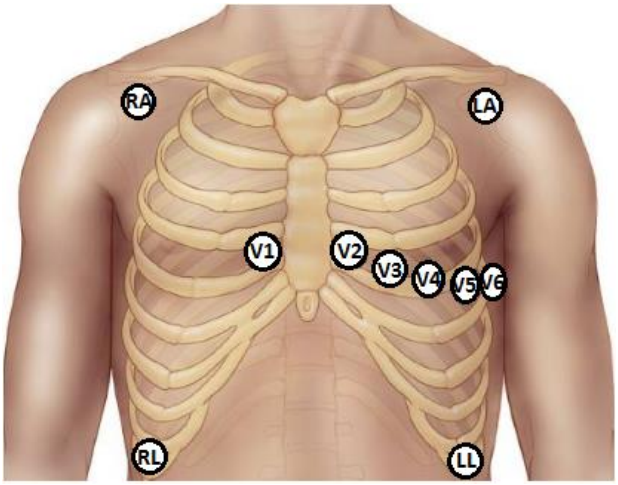
If you reverse the LA and LL leads, this is the result:



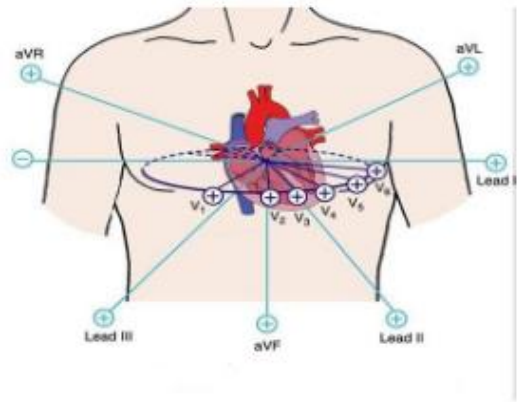
Lead III is inverted, which makes sense because LA is normally the negative electrode and LL is normally the positive electrode for this lead. When they are reversed, you will notice all of the waves are inverted compared to correct placement. You will also notice that lead I looks more like lead II with a taller P and T wave; that's because the normal leads for lead I are now in the position they should be for lead II (RA in the correct position, LA in the position of LL). Lead II is essentially now lead I, as its positive lead (LL) is incorrectly in the LA position.

All of the leads, when reversed, will show predictable changes that have to do with the physical lead placement. This primer will not go into deeper detail about lead reversal, but know that there will be at least subtle changes in most of the leads if there are lead reversals, as most modern EKG machines interpret data and use mathematical calculations based on the physical leads to predict virtual leads, like in aVR, aVL, and aVF. It is also important to consider that correct electrode placement, with or without lead reversal issues, is important for the same reason.

12 Lead Electrode Placement



The first 4 leads, including the ground lead (RL) are the same as they are for our previous readings. This time, we are adding 6 additional unipolar leads, labeled V1 through V6 on the diagram. Unipolar leads are a little different from bipolar leads. A bipolar lead measures electricity in 2 directions - toward a positive electrode and toward a negative electrode. A unipolar lead only measures toward a positive lead. You will notice that, despite not having a negative lead, leads V1 through V6 do have negative deflections on them in the same way leads I through aVF do. This is because the "negative electrode" in this case is similar to the virtual electrodes in leads aVR-aVF. The computer uses a mathematical calculation of all of the other leads to produce a virtual electrode. This time, the virtual electrode is intended to be near the center of the heart. If you compare it to an anatomic cross-section, the V leads evaluate the transverse plane while leads I-aVF evaluate the coronal plane:



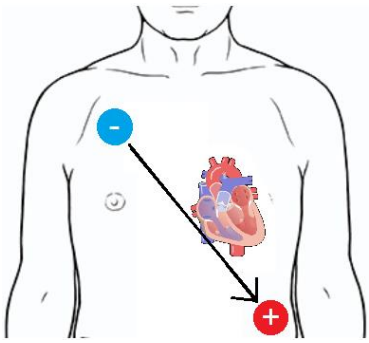
It is important to make sure leads are placed correctly for a 12 lead. V1 is the only lead that is located to the right of the sternum. V1 and V2 are placed at the 4th intercostal space to the right and left of the sternum, respectively. It is recommended to physically palpate the first 4 intercostal spaces when you place leads V1 and V2; the location is anatomically lower on the chest wall than it appears at a glance.

V3 and V4 are placed next, both in the 4th intercostal space. V4 should be placed in the 4th intercostal space directly below the nipple - this is also about the mid-clavicular line. V3 is placed in the 4th intercostal space directly, but laterally placed halfway between V2 and V4.

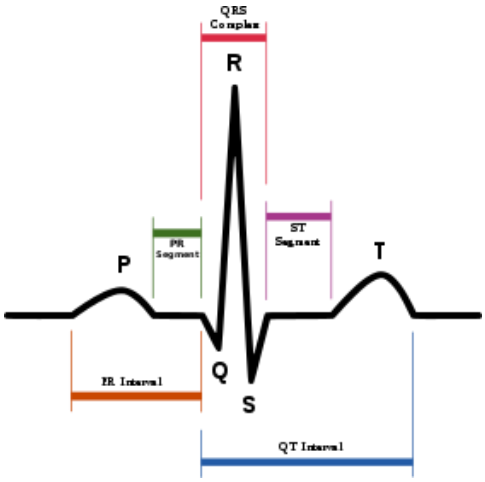
V5 and V6 are placed in the 5th intercostal space. V6 is placed horizontally along the mid-axillary line in the 5th intercostal space. V5 is placed in the 5th intercostal space, halfway between V4 and V6.

Chapter 4 - Rhythm Interpretation

Each EKG rhythm has “rules” that differentiate one rhythm from another. Rules for each rhythm include paramters for measurements like rate, rhythm, PR interval length, and ratio of P waves to QRS complexes. Today we will focus only on lead II. Each “lead” takes a different look at the heart. Because the heart is a 3 dimensional figure and electricity flows through numerous structures, multiple leads are helpful and even necessary in some instances. This segment will focus on lead II. As a reminder, lead II looks like this, anatomically:



Remember the normal flow of electricity in a heart in a normal sinus rhythm:



Electricity first flows in a down-leftward fashion, from the SA node to the AV node. This is the P wave. This is a positive inflection on the EKG graph because the electricity is flowing toward the positive electrode on the EKG and away from the negative. The QRS complex is similar - the electricity flows from the AV node down the bundle of His (and to the ventricle tissue immediately adjacent to the AV node - accounting for the negative inflection of the Q wave), which registers as a large positive wave as electricity is flowing toward the positive

electrode. The electricity eventually reaches the purkinje fibers and begins to travel back upward to the remainder of the ventricles, which is away from the positive electrode and toward the negative one, resulting in a negative S wave. The T wave then represents repolarization, which usually happens in approximately the order of the R wave (ie: in a top-down fashion) and is normally a positive wave, as the repolarization flows toward the positive electrode and away from the negative.

While lead II interpretation is generally grasp-able without fully understanding how a positive or negative electrode effects the EKG, it is important to understand this concept as we will eventually start learning to interpret EKGs in many other leads. Understanding why the waves on the EKG are positive or negative and what that means will help you understand what each wave means, how they differ in both normal and abnormal rhythms, and help you interpret more complex rhythms that require more than one lead in the future.

Lead II Rhythms and Rules

This section will focus entirely upon interpreting rhythms in lead II. Each rhythm has a set of "rules" that helps you determine what the rhythm is. It is always important to look at your PATIENT and not your MONITOR when assessing. Rhythms can easily be misinterpreted due to patient factors like artifact, poorly attached or misplaced leads, and many other factors, and it is important to verify both the veracity of the EKG reading and the patient's condition before making a clinical decision based on the patient's EKG.

The visual diagram used to display the rules for each type of rhythm will differ somewhat from the one used in the previous chapter, however the content is largely the same.

Sinus Rhythms

Normal Sinus Rhythm Rules

Rate: **60-100**
Rhythm: **Regular**
P wave: Upright Inverted Biphasic
P wave to QRS complex ratio: **1:1**
PR Interval: **0.12 to 0.2 sec**
QRS: **0.12 sec or less**

1 P wave for every QRS; 1 QRS for every P wave

Normal PR Interval (PRI) (0.12-0.2 sec), normal QRS (<0.12 sec)

Rates:

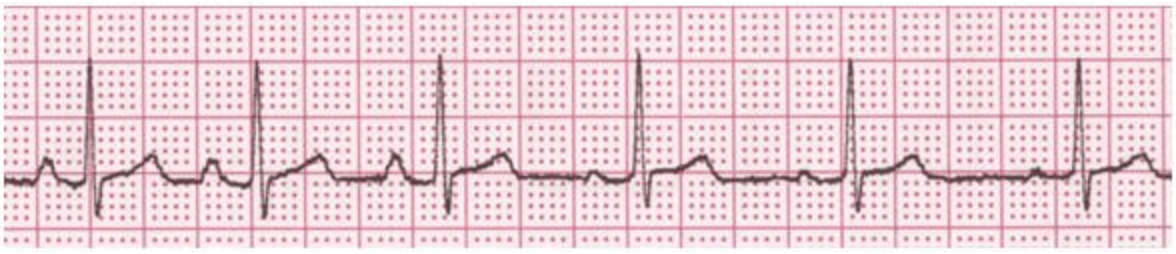
60-100 = normal sinus rhythm

>100 = sinus tachycardia

<60 = sinus bradycardia

Rhythm: Regular, except in:

Sinus arrhythmia: Follows sinus rhythm rules except: Mild fairly gradual increases and decreases in QRS spacing, usually follows pattern of patient's breathing.
Example (sinus arrhythmia):

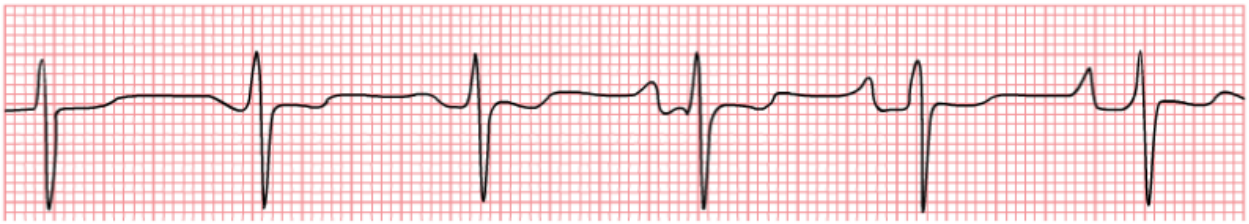


Atrial Rhythms

Atrial Pacemaker

Rate: **60-159**
Rhythm: **Usually regular**
P wave: Upright Inverted Biphasic **Can be inconsistent**
P wave to QRS complex ratio: **1:1**
PR Interval: **0.12-0.2 sec**
QRS: **<0.12 sec**

An atrial pacemaker refers to any rhythm in which an area above the AV node but NOT the SA node is the pacemaker for the heart. This usually presents as notched, sometimes inverted, or unusual looking non-rounded P waves, but has a normal PRI.



Atrial Flutter Rules

Rate: **Usually <100**
Rhythm: **Regular or regularly irregular**
P wave: ~~Upright Inverted Biphasic~~ **Flutter (F) waves replace P waves**
P wave to QRS complex ratio: **X:1**
PR Interval: **Not measurable**
QRS: **Usually 0.12 sec or less**

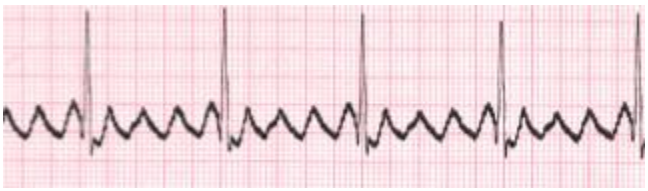
Multiple P waves for every QRS; saw tooth-like appearance of P waves, which are called “flutter” or F waves in this specific rhythm only

No measurable PRI; QRS normal lengths

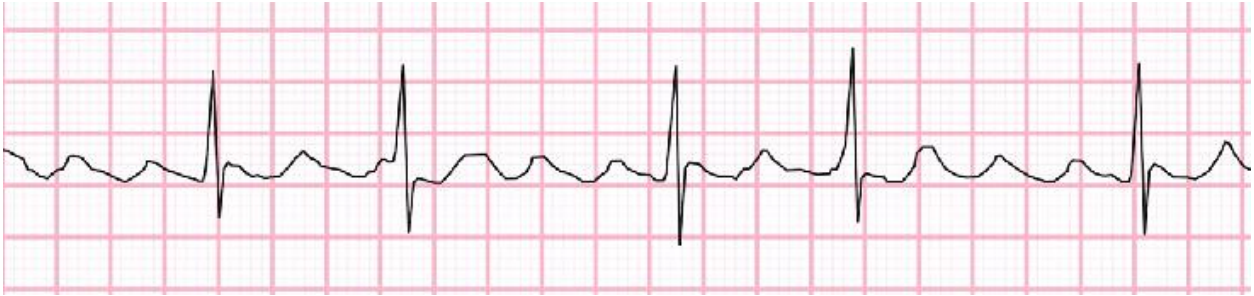
Rate: Can be regular or irregular, usually about normal rate of 60-100 but not necessarily

Rhythm: Can be regular or irregular

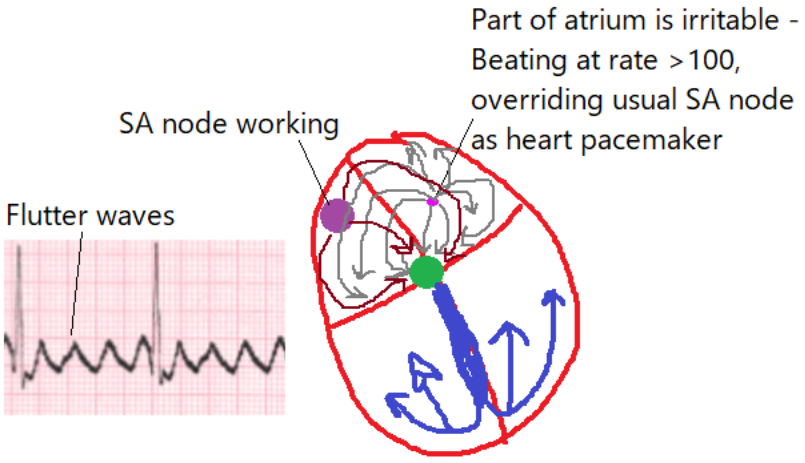
Example: Atrial flutter with regular rhythm, 4:1 conduction rate (4 flutter waves to 1 QRS complex)



Example: Atrial flutter with irregular rhythm due to variable conduction rate



Notice that some of the Flutter waves (similar to P waves, but faster and with a sawtooth shape) are buried under the QRS.



In atrial flutter, the SA node is often working as normal. The rhythm appears abnormally because another part of the heart in the atrium becomes irritable. Irritability, remember, is usually caused by deficiency of oxygen or nutrients, causing the part of the muscle to contract out-of-sync with the rest of the heart as it tries to supply itself with needed nutrients by pumping more. Cardiac muscle tissue is designed to synchronize contraction, so when the irritable part of the atrium starts to contract at a rate faster than the SA node, that portion of the atrium can override the SA node's electrical impulses and take over as pacemaker for the heart.

The flutter waves (F wave) can vary somewhat in shape depending upon the part of the atrium that is fluttering. A saw-tooth appearance is common because electricity flows both in a downward direction toward the positive electrode and upward toward the negative electrode. Additionally, electricity from the atria often flows in a more circumferential manner around the atria, rather that directly through it as it does with normal conduction originating from the SA node. These factors make the F wave look more peaked compared to a normal rounded P wave

The QRS complex appears normal because the AV node is functioning as normal. The AV node acts as a gatekeeper when the atria take over as pacemaker; it allows some electrical impulses through but not all of them. The rhythm of QRS complexes with atrial flutter is not always completely regular because the AV node is not entirely designed to be an effective gatekeeper, so it is not always a set ratio

of F waves to QRS complex (ie: 4:1 F wave to QRS ratio as the strip above). The rhythm does, however, have a pattern to its irregularity (if there is irregularity), making it regularly irregular.

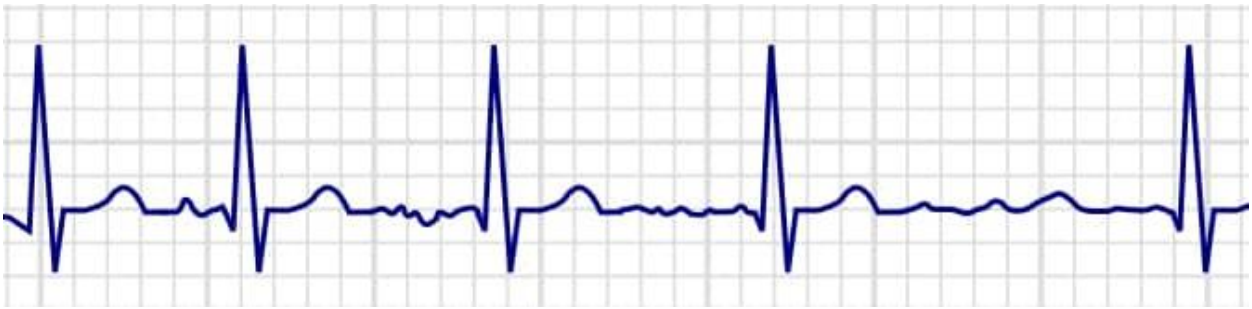
Atrial Fibrillation Rules

Rate:	**<100 - controlled, >100 - uncontrolled**
Rhythm:	**Irregularly Irregular**
P wave:	~~Upright Inverted Biphasic~~ **Not visible**
P wave to QRS complex ratio:	**No P waves**
PR Interval:	**None**
QRS:	**Usually <0.12 sec**

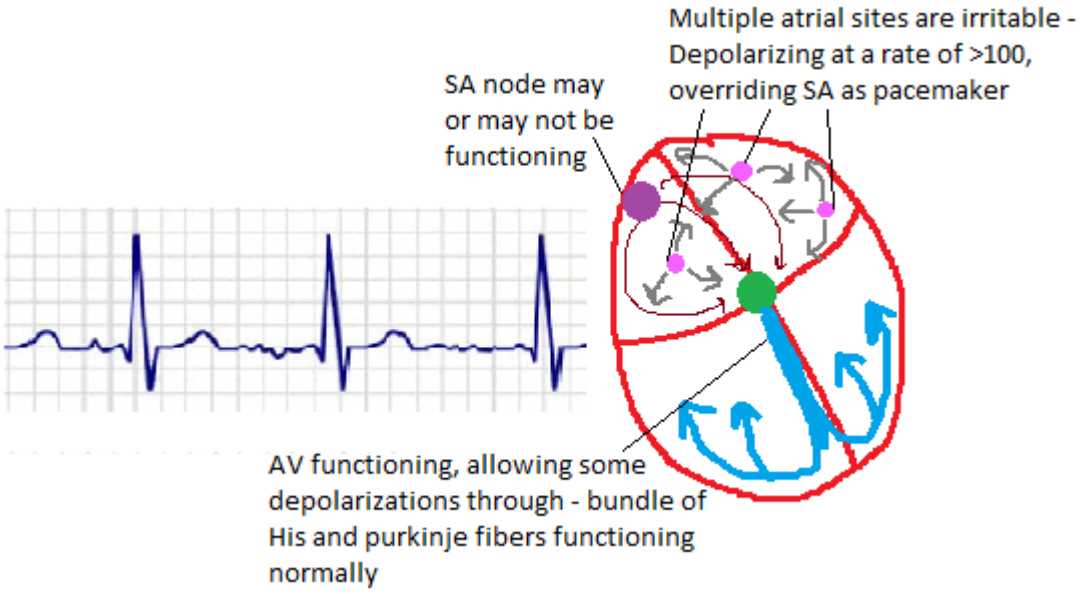
No discernible P waves; PRI not measurable, QRS usually normal length

Rate: Variable. Rates <100 are referred to as "controlled," rates >100 are referred to as "uncontrolled"

Rhythm: Irregularly irregular; no pattern to irregularity. ****This is the only irregularly irregular rhythm****



Atrial fibrillation is similar in physiology to atrial flutter. The major difference is that in atrial fibrillation there are multiple irritable sites on the atria, where there is typically only one in atrial flutter.

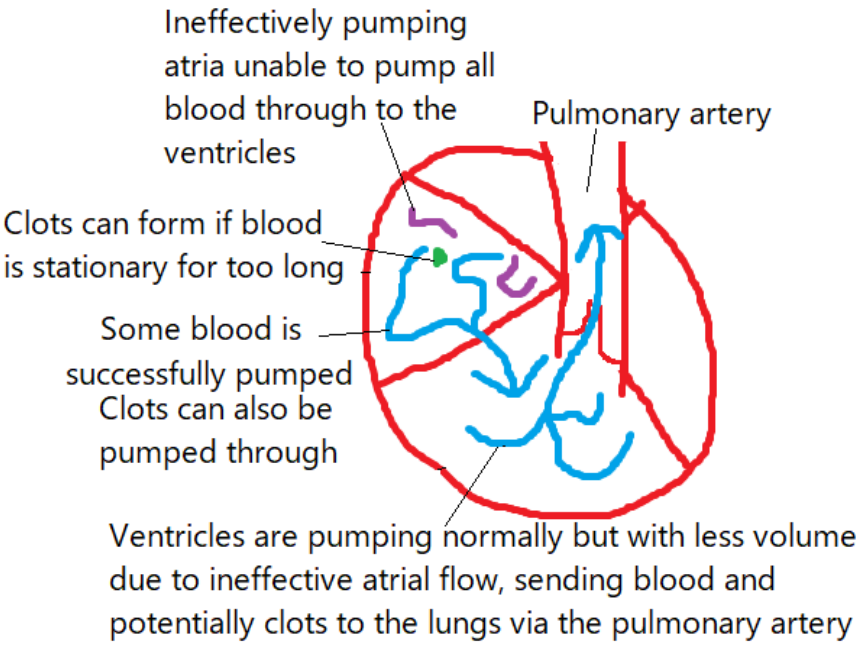


The atria are functionally "quivering" because multiple atrial sites are irritable and trying to fire out-of-sync with the rest of the heart or the SA node. The SA node, if it is functioning at all, is overridden as pacemaker. The AV node does allow some of the impulses through, but due to the irregularity of the unsynchronized atrial depolarizations, the AV node does not always allow impulses through at a regular interval. The bundle of His and purkinje fibers are functioning normally, so the QRS complex and often the T waves look normal in this rhythm despite the disorganized activity. The AV node is functionally acting as a gatekeeper, allowing the ventricles to pump effectively despite the dysfunction occurring in the atria.

Atrial fibrillation is the only consistently irregularly irregular rhythm, meaning it has no pattern to its irregularity. If you find a rhythm that is irregularly irregular, it is usually atrial fibrillation.

Both atrial fibrillation and atrial flutter decrease cardiac output. Anatomically, this is because the atria are not pumping optimally. With atrial flutter, the atria are rapidly contracting; while the contractions of the atria may be effective, the atria are not refilling between contractions, which limits their efficacy. All chambers of the heart do need time to re-fill before pumping again to maximize the pump's efficacy - a concept that will be revisited when you have CPR or BLS training/recertification. In atrial fibrillation, the atria are not contracting together in a coordinated manner, so the pump itself is not effective.

While the atria are not responsible for pumping blood to the peripheral circulatory system, the atria are responsible for receiving blood from the periphery and priming the ventricles by mechanically filling them. The ventricles do still work without priming, and they will continue to pump blood to the periphery, but they will not do so as effectively as they will with the extra fill-up from the atria.

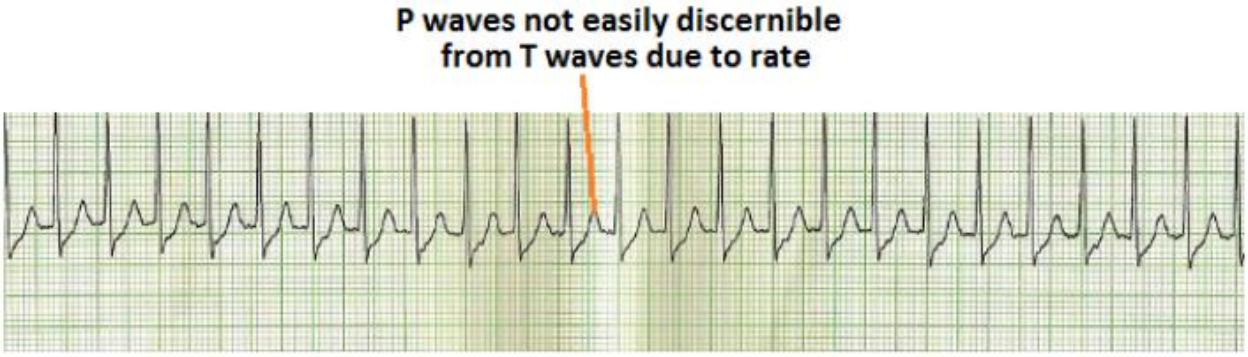


Both atrial fibrillation and atrial flutter make a patient more predisposed to thrombosis (primarily pulmonary emboli), because blood that is not being actively pumped fairly continuously starts to coagulate (clot) if it is allowed to stay stationary for long enough. Ineffective pumps allow blood to become stationary in the chamber of the heart, which starts the coagulation process.

Supraventricular Tachycardia Rules

Rate: **>160**
Rhythm: **Usually regular**
P wave: Upright Inverted Biphasic
P wave to QRS complex ratio: **1:1**
PR Interval: **<0.2 sec**
QRS: **<0.12 sec**

The primary distinguishing characteristic of supraventricular tachycardias (SVT) is a rate >160 with a normal QRS complex.



SVT happens when an area of the heart above the AV node begins to beat rapidly and the AV node allows most or all impulses through. There are some other unique circumstances, such as abnormal electrical pathways that bypass the AV node, that can also cause SVT. This type of anomaly will be discussed in a later chapter.

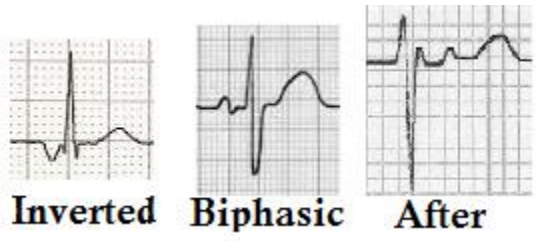
Junctional Rhythms

Junctional Rhythms
Rate: **Varied**
Rhythm: **Regular**
P wave: Upright Inverted Biphasic
P wave to QRS complex ratio: **1:1**
PR Interval: **<0.12 sec**
QRS: **Can be narrow or wide**

Same rules as sinus rhythms, EXCEPT:

PR interval <0.12

P wave may be inverted, biphasic (goes both above and below the baseline), absent entirely, or occur after the QRS complex:



Note about junctional rhythms: Cardiology may label as "accelerated junctional" or "junctional tachycardia"; focus primarily on identifying as junctional and the heart rate, don't worry about labeling it further at this point. Each originating node (ie: the SA node, AV node/junction, and ventricles) have a "rate;" the true definition of tachycardia is dependent upon the rate of the originating node. The normal rate of the AV node/junction is 40-60, so a rate of 60 or greater is technically considered to be "junctional tachycardia" even though the rate is less than 100. This concept will also be true when we discuss ventricular rhythms.

Junctional rhythms and premature junctional contractions originate from the AV node or the AV junction. The P wave in this case is inverted. The P wave is inverted because, instead of electricity flowing from the SA node downward toward the AV node (and toward the positive electrode in lead II), the electricity is originating in the AV node and flowing upward toward the negative electrode.

The PRI is short because, instead of the AV node receiving the electricity that originated elsewhere (and holding it for a fraction of a second, as the AV node normally does), the electricity is originating from the AV node, resulting in a minimized or absent pause (depending on what part of the AV node has taken over as the heart's pacemaker in place of the SA node). This is also why the P wave is sometimes absent in junctional rhythms; the P and QRS complex sometimes occur simultaneously, or the P can even take place after the QRS complex depending upon the specific location in the AV junction that has taken over as the dominant pacemaker.

The QRS complex is normal in appearance compared to a normal sinus rhythm because the bundle of His and purkinje fibers are behaving normally.

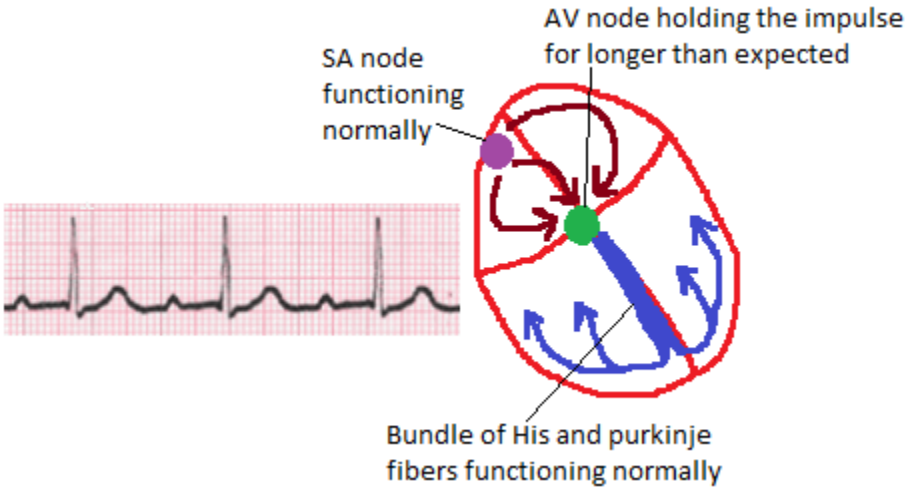
AV Blocks

AV blocks are what happen when the AV node is not functioning correctly or when the AV node and bundle of His are not communicating correctly. An AV block is an add-on to an underlying rhythm, ie: you don't have a 1st degree block, you have a sinus rhythm with a 1st degree block.

1st Degree AV Block

PR interval >0.20 every time
Rhythm otherwise identical to underlying rhythm

A first degree block is usually a minor dysfunction of the AV node. A sinus rhythm with a first degree AV block is one of the most common arrhythmias and is sometimes considered a normal variant, particularly among athletes and young adults. Most patients with a first degree AV block have no symptoms.

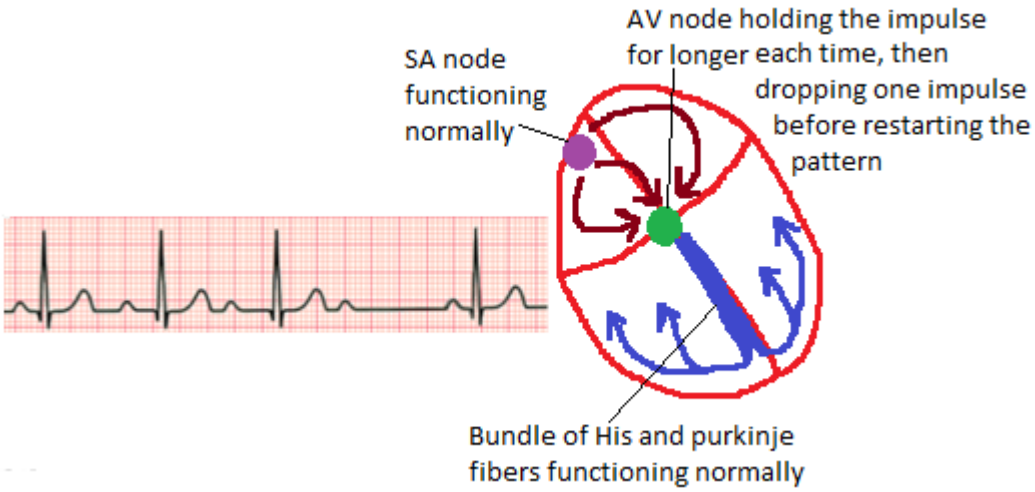


In a normal sinus rhythm, the AV node holds the depolarization impulse for a very short period of time (usually about 0.02-0.04 seconds), then releases it to continue down the bundle of His and purkinje fibers to allow the ventricles to depolarize. A first degree AV block refers to an AV abnormality in which the AV node holds the impulse for longer than is normal, causing the PR interval to be prolonged (>0.2 seconds).

2nd Degree Block – Type I

More P waves than QRS complexes: PR interval elongates over several beats, QRS complex is eventually dropped, then PR interval resets to baseline

Also referred to as Wenkebach or Mobitz I



In a 2nd degree type I block, the AV node is similarly malfunctioning. This time, the AV node is holding the impulse received from the SA node for an increasingly prolonged duration, until eventually it drops the impulse entirely for one beat. Then the cycle usually repeats, starting at a normal or nearly-normal PR interval, increasing over the process of one or more depolarizations, then dropping. When the depolarizations are allowed through, the QRS complex is normal in appearance because the bundle of His and purkinje fibers are functioning normally.

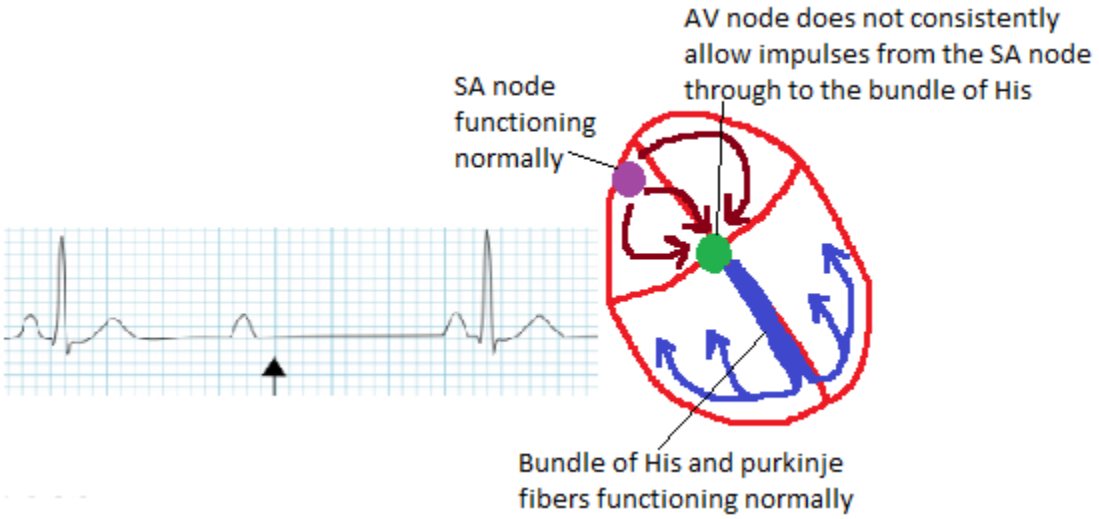
2nd Degree Block – Type II

More P waves than QRS complexes; QRS complexes occasionally dropped from P waves. PRI may or may not be >0.20 seconds.

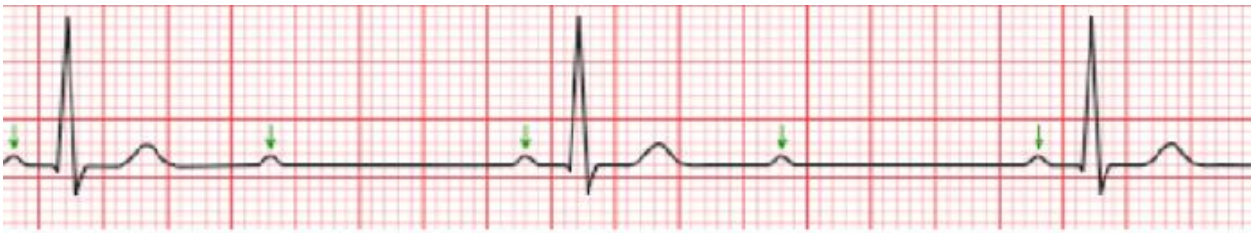
Rate of dropped QRS complexes can be regular (ex: 2:1 ratio with every other P wave generating a QRS complex) or variable (QRS complexes are dropped at random)

Also referred to as Mobitz II

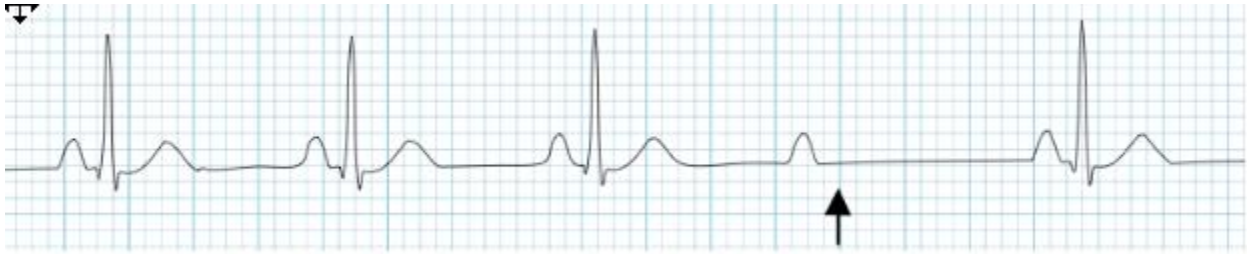
You differentiate this rhythm from Wenckebach (Mobitz I) because the PR interval do not get longer in preceding beats before the beat drops.



A 2nd degree type II block is a block in which the AV node does not consistently allow the depolarizations of the SA node through. This results in some P waves without a QRS complex, but the P waves are usually regular in rhythm and appearance. The P to QRS conduction ratio (ie: the number of Ps that are allowed through to become QRSes) is greater than 1 to 1 (ie: may be 2 Ps to 1 QRS, etc.). The conduction ratio may be regular or irregular, so you can have a second degree type II block that only allows every other QRS complex through, like this:



Or you can have 2nd degree type II blocks that do not have a particular pattern to the dropped QRS complexes, like this:



3rd Degree Block

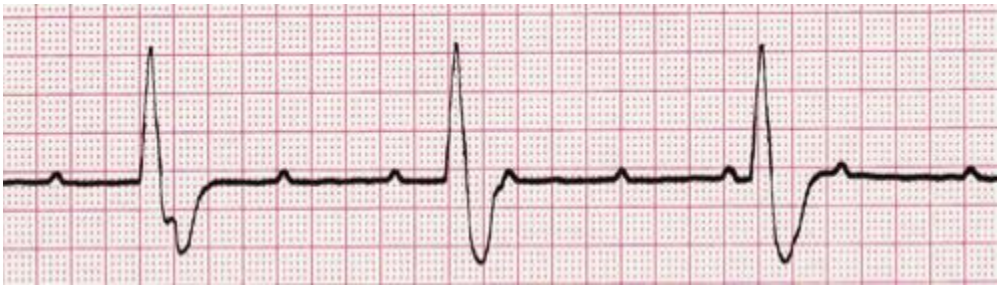
P waves all march out, QRS complexes all march out; no association between rhythm of P waves and QRS complexes

Note: P waves do sometimes get "lost" in QRS complexes; they're still there, they're just not visible when they happen at the same time a QRS is occurring

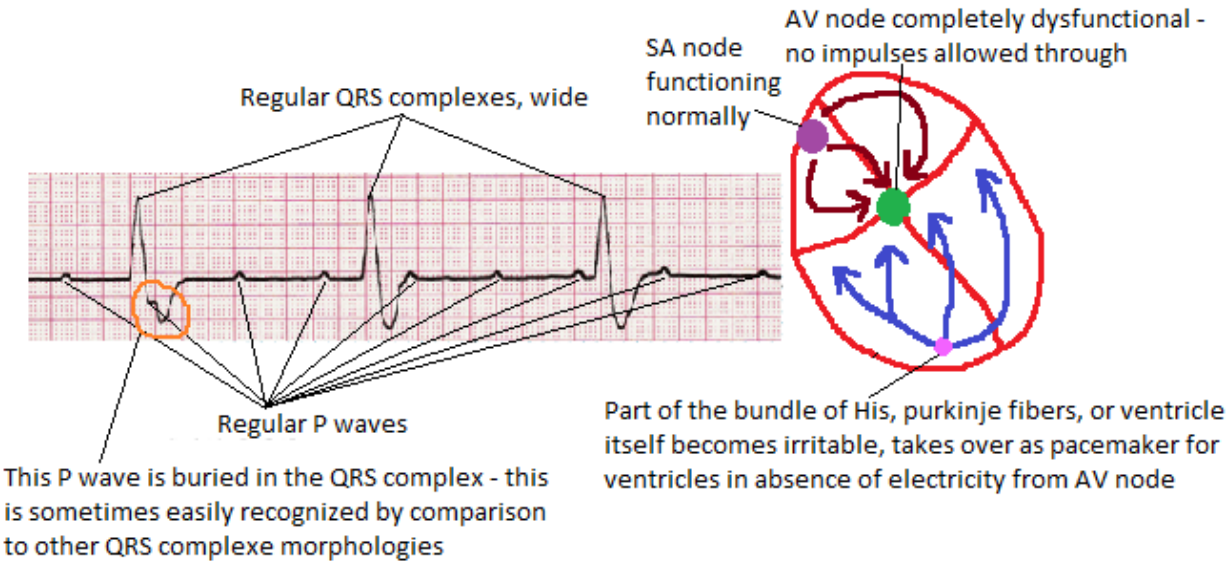
QRS complexes may be narrow or wide, wide usually indicates electrical impulse is coming from the ventricles where narrow usually indicates electricity is originating from the lower AV node or upper portion of the bundle of His (as this indicates the electricity in the ventricles is flowing semi-normally in a top-to-bottom fashion)

Also referred to as a complete heart block

These patients usually require emergent intervention



Third degree block is a complete dysfunction of the AV node. Remember that the AV node is the gatekeeper and the only route between the atria and ventricles. The AV node stops transmitting any impulses, functionally cutting off communication between the atria and ventricles. The SA node, atria, and ventricles are all are still functional, but they are unable to communicate and work in synchrony.



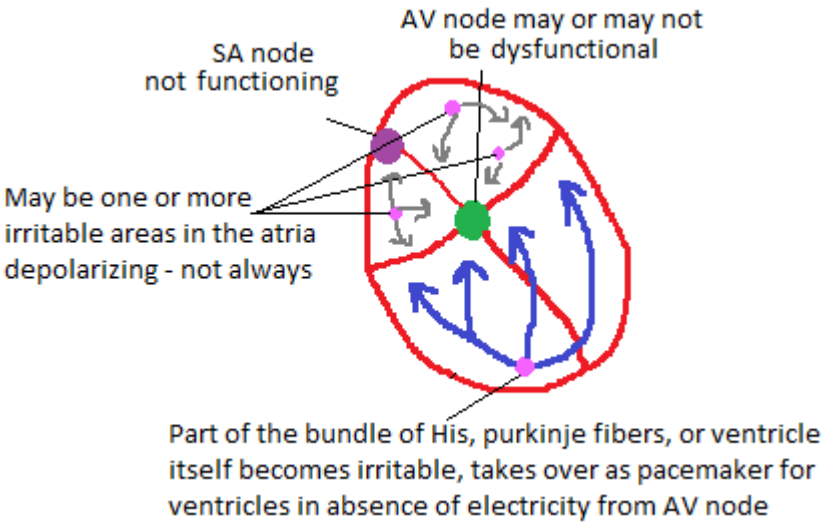
The SA node is usually functioning normally with a 3rd degree block. The AV node, which normally functions as the gatekeeper between the atria and the ventricles, is not transmitting any electricity between the two. As a result, the ventricles are not stimulated to depolarize. The ventricles become irritable, and one or more areas of the ventricles will start to depolarize and take over as the pacemaker for the ventricles.

When a ventricle takes over as their own pacemaker (because they are not receiving their usual impulses through the AV node), the QRS complexes are wide (>0.12 sec) and bizarre looking compared to a normal QRS complex. This is because the bundle of His and purkinje fibers, which is normally somewhat insulated and transfers electricity faster than the ventricles themselves, are usually not transmitting because they are not receiving electricity from the AV node. The slower transmission and the change in direction of electrical flow both prolongs the QRS complex (making it wider) and changes the shape of the QRS complex (making it look bizarre).

Of note in the particular strip above is the easy visibility of a P wave buried in the first visible QRS complex. Compare the QRS complex to the other 2 QRS complexes and note the extra bump upward toward the end of the first QRS complex - this is your P wave that is "buried." Some QRS complexes completely bury a P wave rendering it completely invisible, but this particular strip is a good example of a buried-yet-visible P wave that occurred at the same time as a QRS complex. For a better view, compare the red circled area of the QRS complex to the next QRS complex - the upward inflection in the second half of the complex that is not seen in the other complexes is the P wave. While it is impossible to know with 100% certainty when a P wave is buried in a QRS complex, you can often make an educated guess on where the P wave should be based on the rhythm of the surrounding P waves.

Ventricular Rhythms

None of the above rhythms discussed thus far have significantly affected the ventricles or caused dysfunction of the ventricular electrical conduction system. The AV node often acts as a gatekeeper or barrier, which means that atrial and junctional arrhythmias often do not cause changes to the QRS complex. This is a helpful fail-safe system for the heart, as the ventricles are responsible for circulation to the lungs and periphery (and dysfunction of ventricular conduction therefore can be far more catastrophic to patient health). The next set of arrhythmias will sometimes affect the ventricles or even be caused by the ventricles. Many (but not all) of the following arrhythmias are life-threatening and require urgent or emergent intervention.



Ventricular rhythms can occur under several circumstances. Sometimes the ventricles take over as pacemaker because all other pacemaker sites have failed. Other times, the ventricles become so irritable they begin to depolarize faster than all of the other nodes and therefore take over as pacemaker, even though the other nodes may be functioning.

Organized Ventricular Rhythms

Rate: **Varied**
Rhythm: **Regular**
P wave: ~~Upright Inverted Biphasic~~ **None**
P wave to QRS complex ratio: **N/A**
PR Interval: **N/A**
QRS: **>0.12 sec**

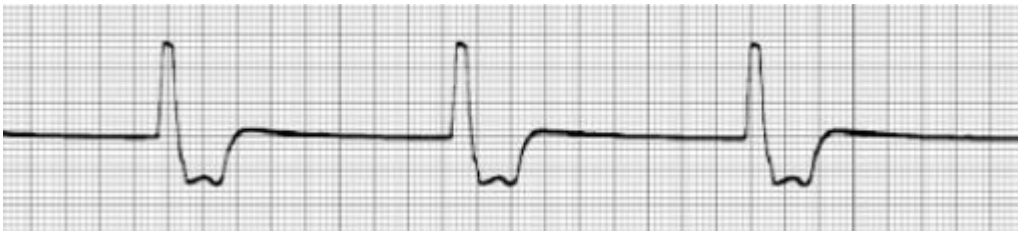
Rate determines the name for an organized ventricular rhythm. Ventricular rhythms can be distinguished by their QRS complex length of >0.12 seconds and their absence of P waves

Ventricular escape rhythm: <40 BPM

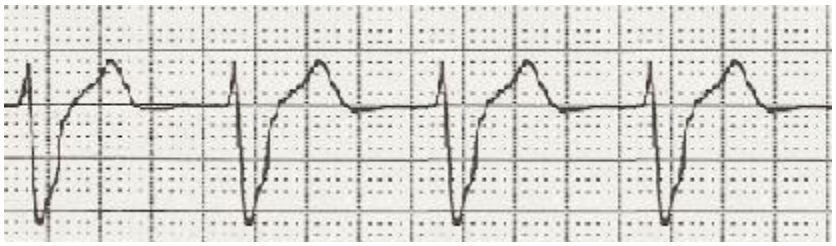
Accelerated idioventricular rhythm: 40-100 BPM

Ventricular tachycardia: >100 BPM

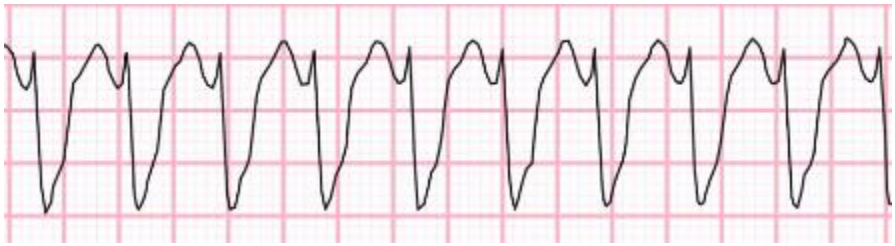
A ventricular escape rhythm typically occurs when both the SA and AV nodes have failed, so there is no transfer of electricity from the atria to the ventricles; this forces the ventricles to take over as pacemaker. In a ventricular escape rhythm, the rate is typically 20-40 BPM, there are no P waves, and the QRS complexes are wide and bizarre:



In an accelerated ventricular rhythm, the irritable ventricular site that has taken over as pacemaker is contracting at a rate >40, but is not a true ventricular tachycardia. The physiology of this rhythm is similar, but is accelerated due to increased irritability of the pacemaker site:

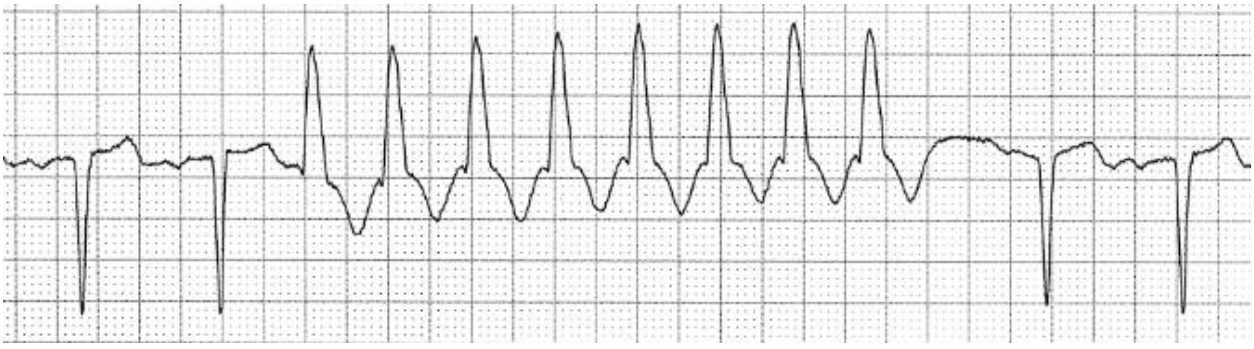


Ventricular tachycardia is essentially the ventricular version of atrial flutter. With ventricular tachycardia the irritable ventricular site has taken over as pacemaker by depolarizing rapidly.. Ventricular tachycardia is not always an indicator that the SA and AV nodes have failed, as is usually the case with ventricular escape and accelerated ventricular rhythms; instead the ventricle has taken over as pacemaker by depolarizing at a rate faster than the SA node.

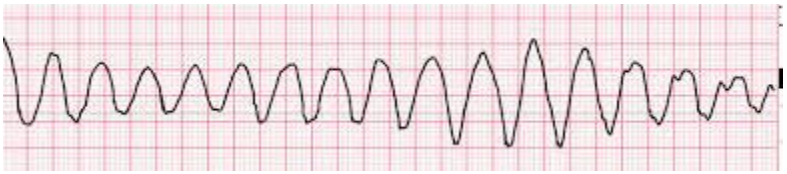


Ventricular tachycardia is sometimes pulseless, but not always. If you see or suspect ventricular tachycardia, it is important to check a pulse before intervening.

A patient can also have "runs" of ventricular tachycardia. A run of ventricular tachycardia refers to a rhythm that converts from the underlying rhythm to ventricular tachycardia for >4 QRS complexes, then reverts to the underlying rhythm. The reversion is important; it is not a "run" of ventricular tachycardia if it never converts back out of ventricular tachycardia.

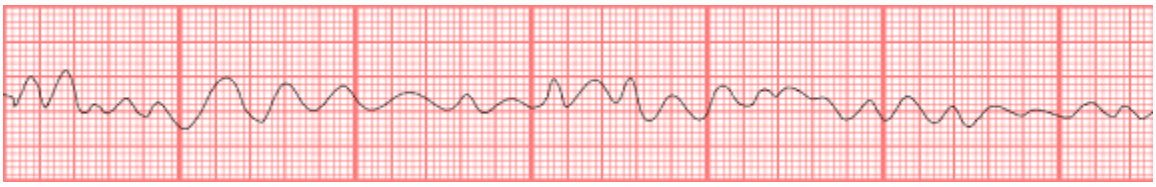


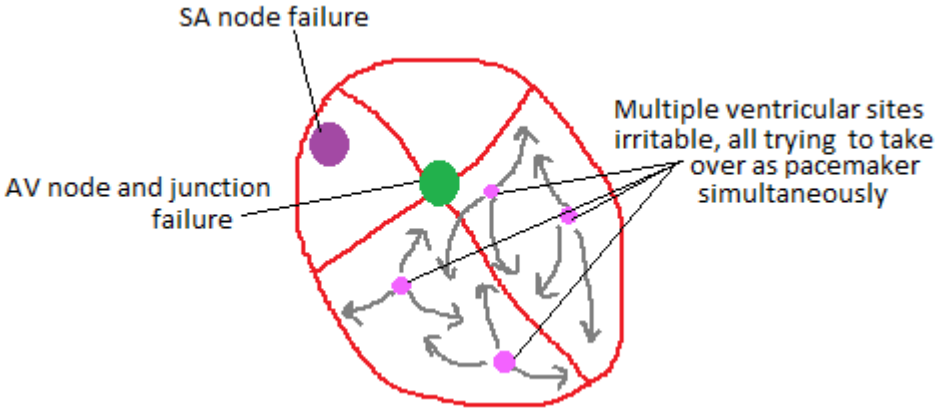
Ventricular tachycardia can also have variable QRS morphology, as in this example of a specific type of v tach called Torsades de Pointes ("turning of the points"):



Ventricular Fibrillation
Rate: **Not discernible**
Rhythm: **No organized rhythm**
P wave: ~~Upright Inverted Biphasic~~
P wave to QRS complex ratio: **N/A**
PR Interval: **N/A**
QRS: **Not discernible**

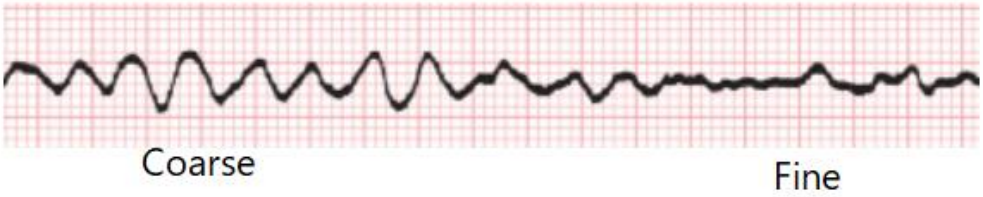
Ventricular fibrillation is a rhythm in which the ventricles are quivering. There will be no true discernible waves or QRS complexes. This is universally a life-threatening rhythm that requires both high quality CPR and defibrillation.



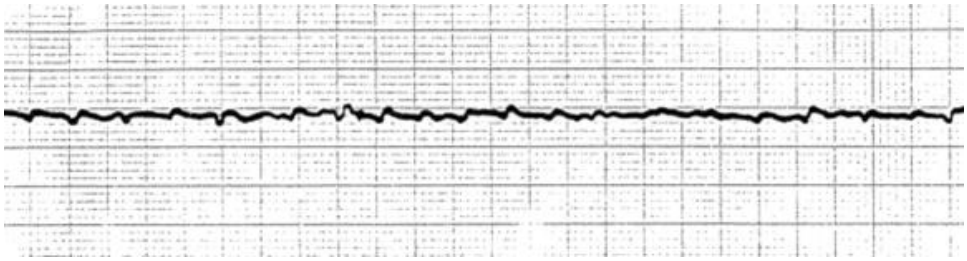


Ventricular fibrillation (VF) is what occurs when multiple sites in the ventricles become so irritable they attempt to take over as pacemaker out-of-sync. This is the same as happens in atrial fibrillation, except the problem occurs below the AV junction so the patient will not have an organized QRS complex (or indeed an organized rhythm of any kind). The SA and AV node may or may not be functioning.

Ventricular fibrillation is a lethal arrhythmia if it is not corrected via electricity and CPR. It is sometimes correctable via electricity because defibrillating the patient emulates the depolarization process in the entire heart all at the same time. The cells must all repolarize before depolarizing again, which gives the cells in the SA node a chance to become the pacemaker for the heart again while the ventricles are repolarizing and unable to spontaneously depolarize.



Ventricular fibrillation can be classified as "coarse" or "fine." This distinction is somewhat helpful because coarse VF has higher amplitudes and, therefore, larger amounts of electrical activity compared to fine VF. A patient in VF is undergoing cardiopulmonary resuscitation, so it can be helpful to differentiate coarse vs fine VF if termination of resuscitation efforts is a consideration.



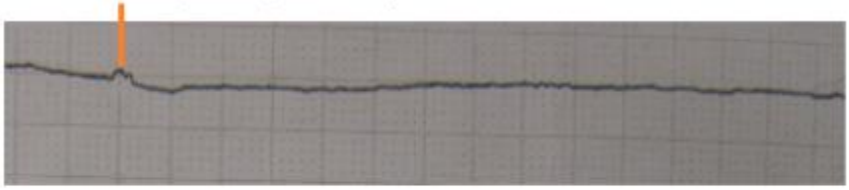
Fine VF and asystole with external artifact can be difficult to distinguish from one another. It is important to have all hands off the patient while the rhythm is being interpreted so the interpretation can be made without mistaking artifact for fine VF. Movement from external sources, such as compressions or performing other interventions while rhythm is being interpreted, can cause artifact. **While CPR interruptions do need to be minimized to improve the likelihood of resuscitation, distinguishing fine VF from asystole does impact clinical decision making**

Agonal/Asystole

Rate: **N/A**
Rhythm: **N/A**
P wave: Upright Inverted Biphasic **Usually absent**
P wave to QRS complex ratio: **N/A**
PR Interval: **N/A**
QRS: **None**

"Flat line"

P wave, no QRS complexes



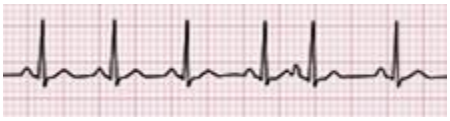
Asystole is an absence of electrical activity in the heart. An agonal rhythm is similar, but occasionally has present P waves. Asystole and agonal rhythms are distinguished by absence of any kind of QRS complex.

Premature Contractions

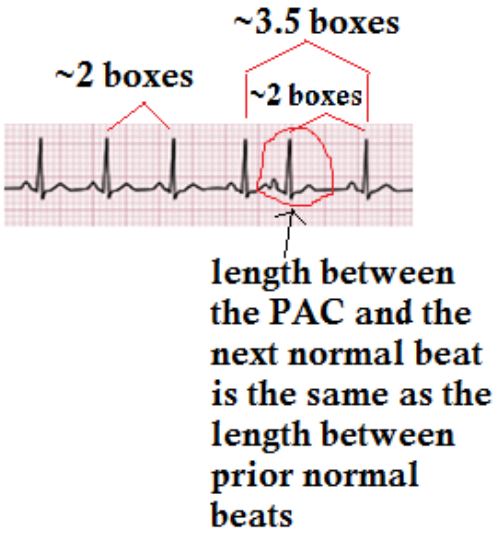
Premature contractions, whether they come from the atria, junction, or ventricles, are caused by irritability. Premature contractions are not a rhythm by themselves, they are additional beats within an underlying rhythm. An underlying sinus rhythm with a PVC should be labeled with both interpretations

Premature Atrial Contraction

Looks morphologically like a sinus or atrial beat, but is out-of-place with regards to rhythm. P wave may look different from the others but will still be upright and present and have a normal PR interval:

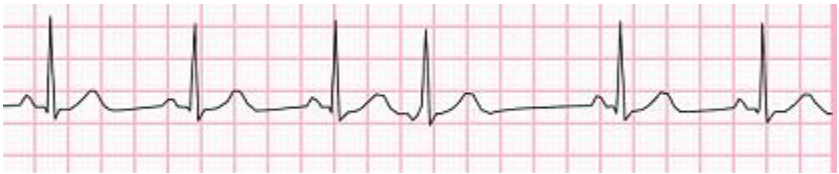


Premature atrial contractions "reset" the heart rhythm, so the rhythm starts up again from the QRS complex of the PAC:

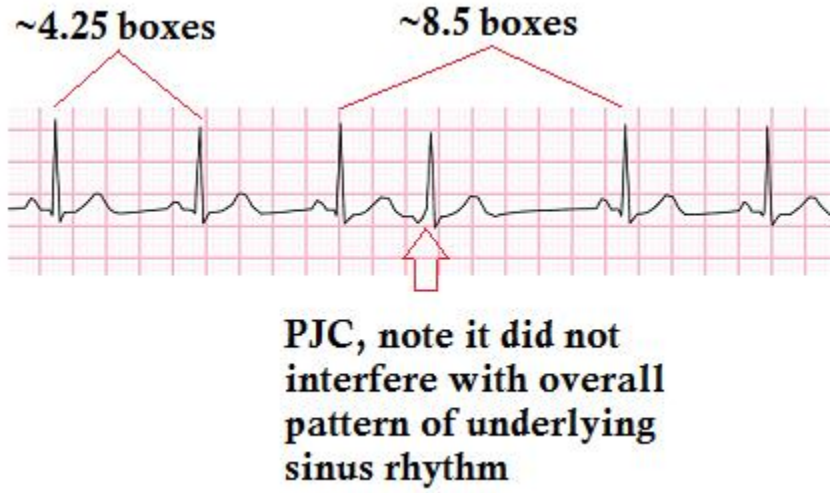


Premature Junctional Contraction

Fairly uncommon; looks similar to a sinus beat, but is out-of-place with regards to rhythm and has an inverted, absent, or biphasic P wave. It may fall after the QRS, between the QRS and the T wave. If it falls before the QRS, it will have a short PR interval similarly to the short PR interval in a junctional rhythm.

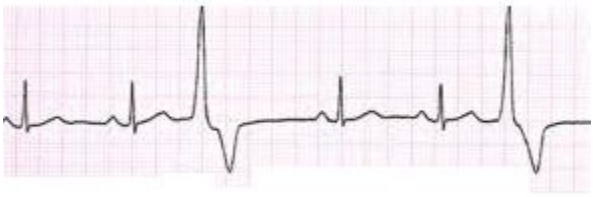


Unlike PACs, the underlying rhythm will stay at the same intervals, the PJC will just take the place of an expected beat, as here:

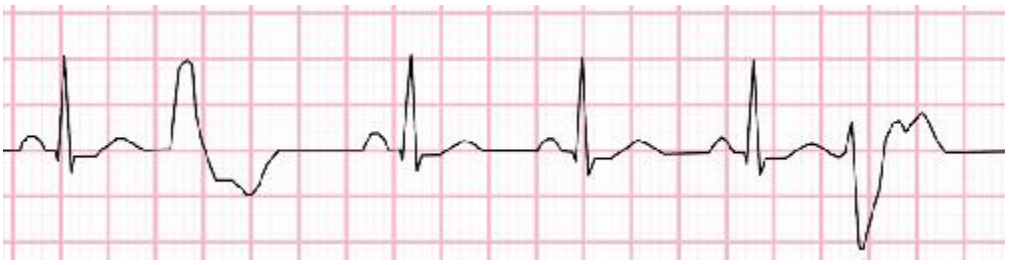


Premature Ventricular Contraction

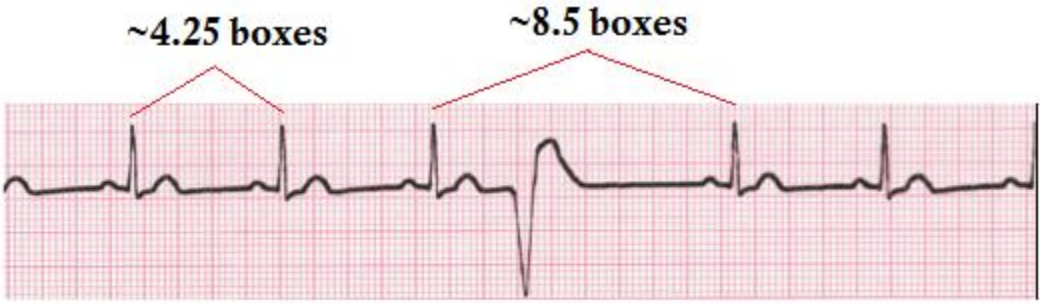
Wide, bizarre QRS, out-of-place beat:



PVCs can be unifocal, which means they come from the same part of the ventricle and are similar in appearance, or multifocal, which means they come from different parts of the ventricle and look totally different from one another:

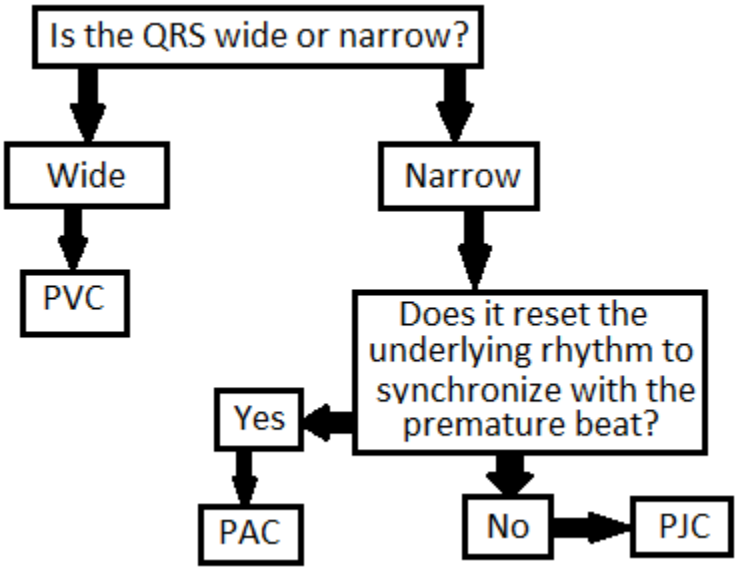


PVC will not reset the underlying rhythm:



Underlying rhythm pattern isn't altered due to the PVC

To distinguish PACs, PJCs, and PVCs, the following flow chart can be used:

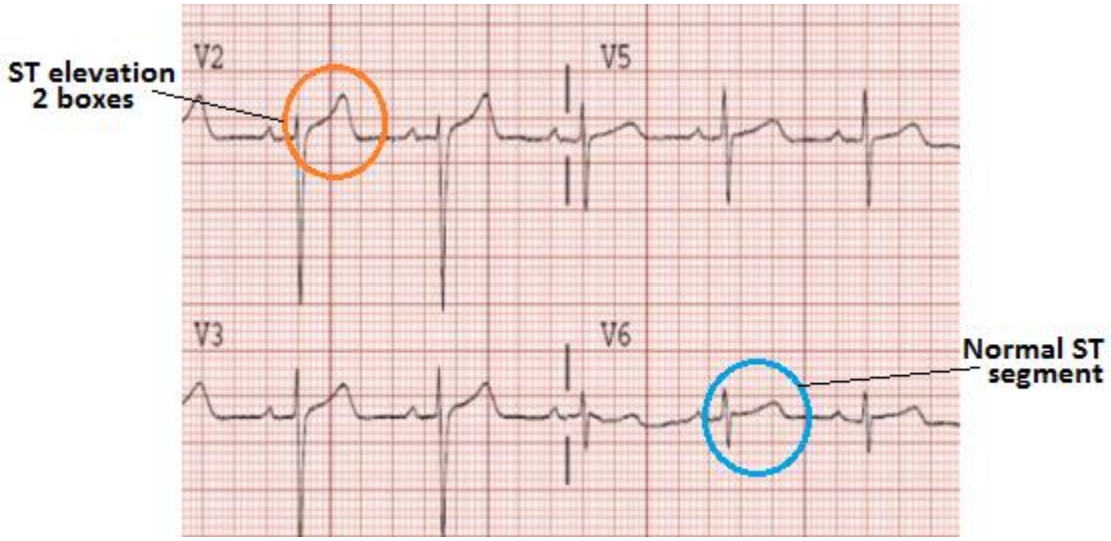


Chapter 5 - 12 Leads and Special Rhythms

This module will examine a very limited number of 12 lead interpretation methods and things to watch for in practice. This is a limited look at 12 lead EKG interpretation and should not be used as a comprehensive learning module for 12 lead interpretation.

ST Elevation

ST elevation refers to the failure of the QRS complex to return to the baseline before the T wave commences. Elevation specifically refers to this slurring of the QRS-to-T wave that occurs above the isoelectric line. It looks like this:

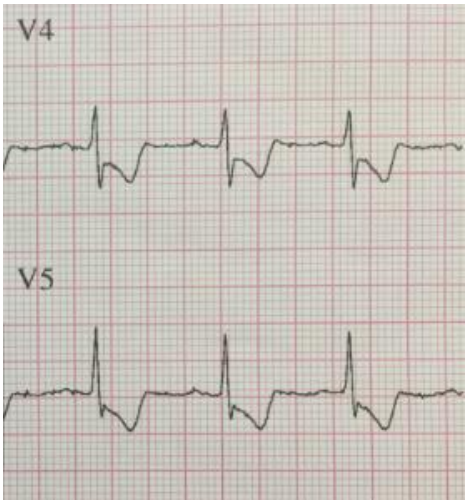


Note that the S wave in the QRS complex does not return to the baseline before the T wave starts in V2. The S wave does return to the baseline in V6.

ST elevation of 1 mV (1 small box) or larger is considered to be clinically significant, and it must be replicable in more than one full cardiac cycle (ie: P to T wave).

The reciprocal change, ST depression, is the same finding but instead of failing to return to the baseline in favor of an elevated graph above the baseline,

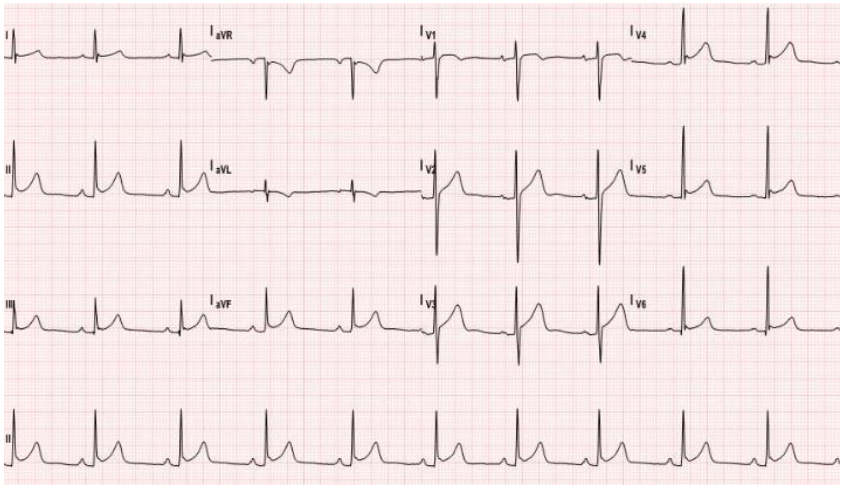
the S does not return to the baseline and remains below the baseline. ST depression looks like this:



What Does ST Elevation Mean?

It is not clearly known what causes ST elevation and depression - it is thought that it is an electrical defect caused by ventricular tissue that is ischemic and damaged or otherwise malfunctioning. One ST elevation and depression hypothesis is that it occurs because ischemic ventricular tissue stays fully polarized for longer than normal. That polarization (which is measured on the EKG as electrical activity) causes the EKG graph to measure as non-zero until after the T wave.

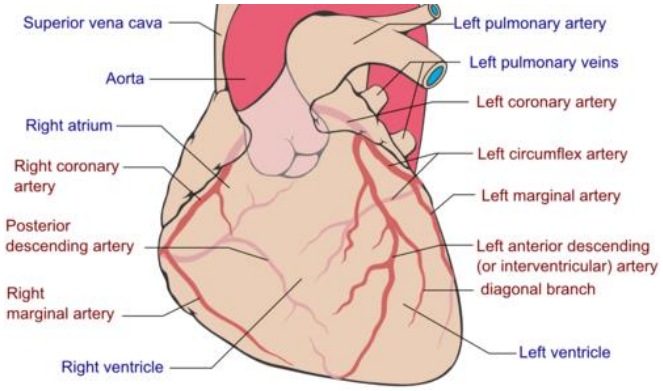
ST elevation in different leads can mean different things. A condition called pericarditis can cause mild diffuse ST elevation in most or all leads:



Pericarditis is an inflammation or infection of the sack around the heart (pericardium). It causes irritation to the overall heart tissue which presents as diffuse ST elevation.

ST Elevation Myocardial Infarction (STEMI)

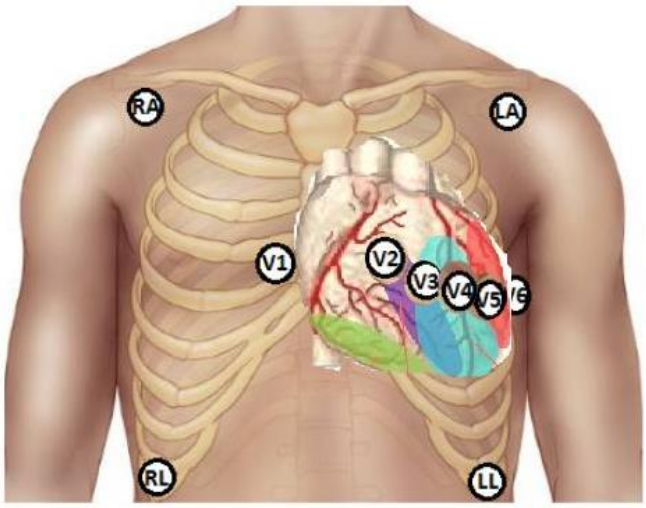
ST elevation can also be an indicator of a specific type of myocardial infarction known as STEMI. This particular type of MI is seen as emergent because a true STEMI usually indicates a blockage of 90% or more of one of the coronary arteries. Patients with STEMI require immediate intervention; lack of intervention will lead to death or debilitating chronic heart conditions like congestive heart failure. To review, the coronary arteries that deliver blood to the heart itself are located as diagrammed below:



Note that the posterior descending artery is located along the posterior wall of the heart. The left circumflex artery also wraps around to the posterior heart.

As we discussed above, ischemic heart tissue can produce ST elevation. In the case of STEMI, ischemia is caused by grossly inadequate or entirely absent blood flow due to an occlusion of one of the coronary arteries. The coronary artery affected can be approximately located using a 12 lead EKG to determine which areas are likely ischemic and therefore likely deprived of blood flow. While this method is not precise, it does give interventional cardiology a general idea of where they should start looking for coronary artery blockages.

In addition to ST elevation in specific leads, in a patient with STEMI you will often see ST depression in the reciprocal leads that are electrically opposite from the leads with elevation. This makes sense if you consider previous lessons on lead placement and what leads are designed to detect: electrical activity moving toward or away from them.



Notice that each color labeled area is supplied by a particular coronary artery.

The areas of the heart that are measured by particular leads are circled in different colors. Green represents the right ventricular region (right marginal artery) and is measured by V1. Purple represents the septal region; septal refers to the septum separating the two ventricles. The septal region is measured by V2 and V3. Light blue represents the anterior segment and is measured by V3 and V4. The red segment is the lateral and is measured by leads I, aVL, V5 and V6.

There is one remaining segment called the inferior segment that is not labeled with a color on the above diagram. The inferior segment is as the name suggests; the bottom portion of the heart that faces the diaphragm. It includes the inferior portion of the apex of the heart. This segment is measured using leads II, III, and aVF.

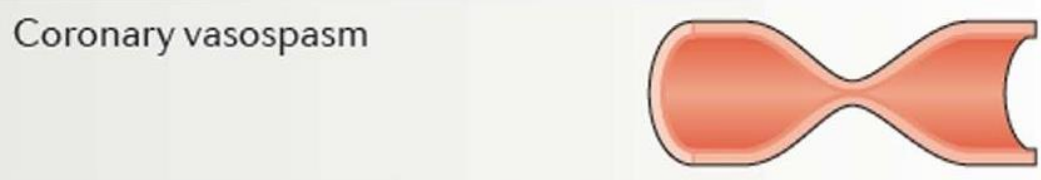
Right	V1
Septal	V2, V3
Anterior	V3, V4
Lateral	I, aVL, V5, V6
Inferior	II, III, aVF

Of note: The right ventricular area is poorly visualized by a 12 lead EKG setup. If a right STEMI is suspected, additional leads are often attached. A posterior STEMI is also generally not visible on the 12 lead EKG, as it is measured by leads V7-9 which are not applied during a standard 12 lead EKG. Much like leads V1-6 wrap around the left side of the chest, leads V7-9 continue to wrap around the posterior section of the chest wall. We will not discuss leads or placement beyond the standard 12 lead in detail during this primer.

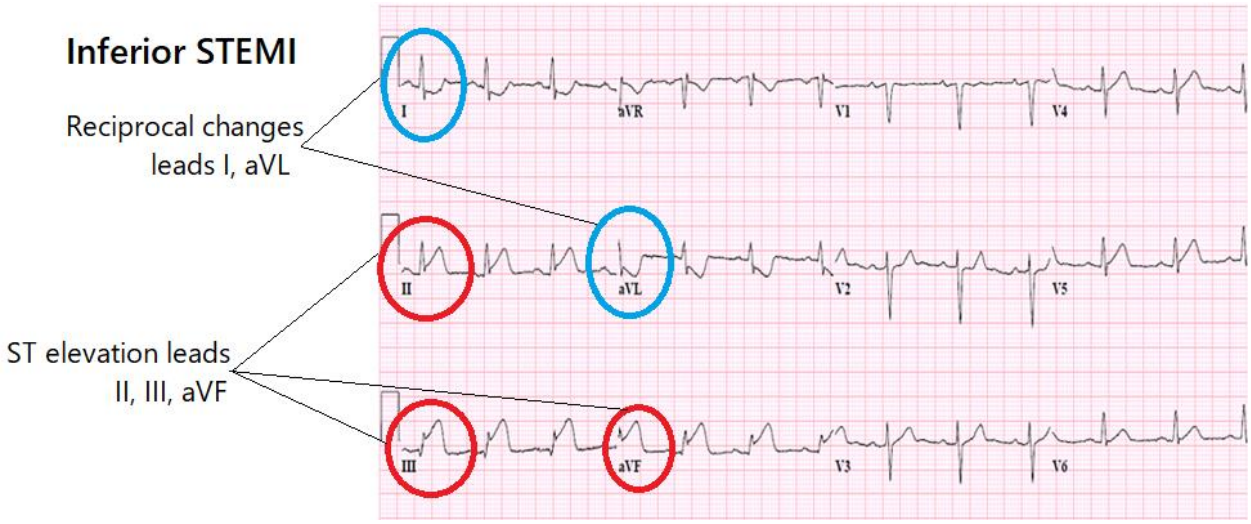
Additionally, many types of STEMI will show inverse changes in a reciprocal leads. Reciprocal leads refers to leads that show the opposite direction another lead. For example aVF and aVL are leads that go in almost perfectly opposite directions; if you have a patient with an inferior STEMI, in addition to ST elevation in aVF you will often also see ST depression in aVL. Reciprocal changes can include ST depression instead of elevation and inversions of particular waves (particularly T waves).

Prinzmetal's Angina

Printzmetal's angina, or coronary artery vasospasm, is a specific type of angina that causes EKG changes. The condition itself causes spasm of the smooth muscle around the coronary arteries. Spasm of the muscles causes a temporary occlusion of the artery, resulting in ischemia that resolves when the spasm resolves. The spasms usually resolve spontaneously, but do sometimes require intervention.

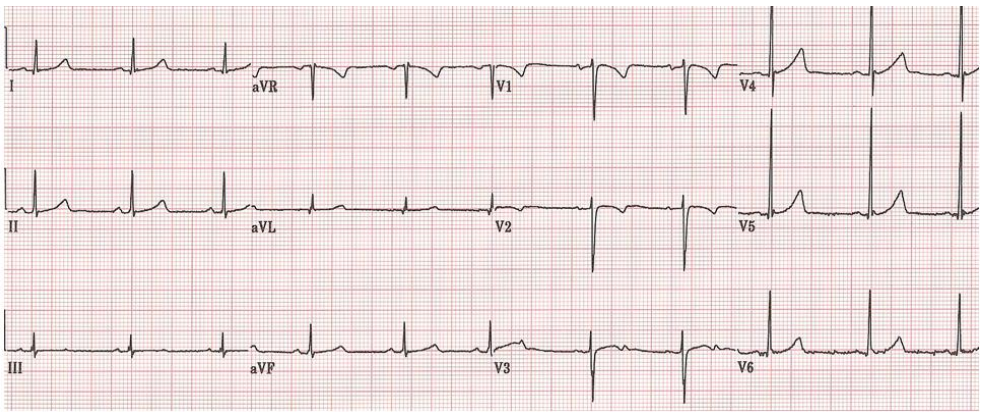


The EKG changes that occur with Printzmetal's angina depend upon the affected coronary artery, and usually present with changes that are otherwise consistent with STEMI. **Printzmetal's angina should be treated as if it were a STEMI until proven otherwise; STEMI is lethal if not treated, Printzmetal's angina is not. It is never wrong to err on the side of caution when there is concern for a condition that can impact a patient's life, limb, fertility, or vision.**



The difference between a STEMI and a patient with Printzmetal's angina is the consistency of the EKG changes. A patient with a STEMI will not have spontaneous resolution of the EKG changes; a patient with Printzmetal's angina will sometimes spontaneously self-resolve, sometimes resolve entirely with medical intervention like nitroglycerin.

Spontaneous Return to NSR



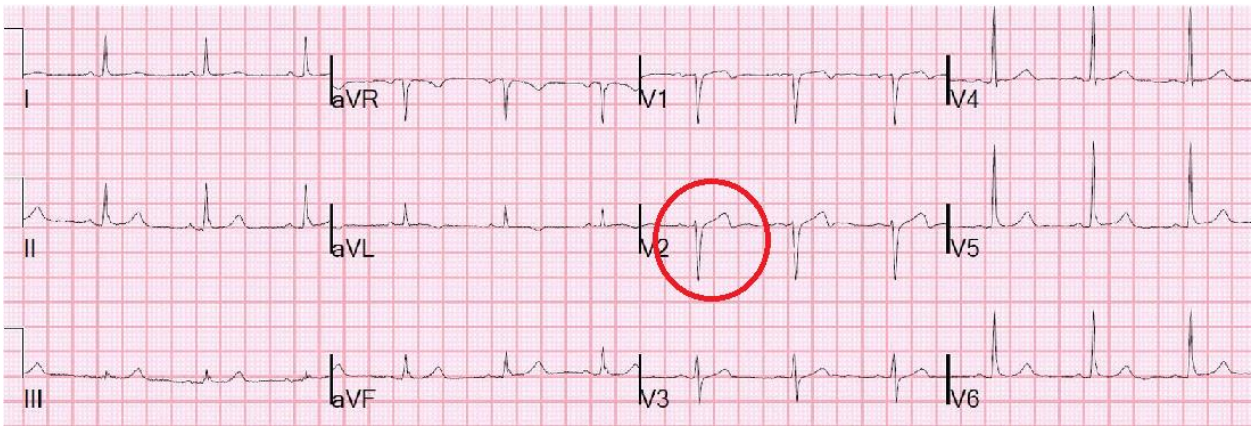
Printzmetal's angina typically presents with the same symptoms as cardiac ischemia or acute coronary syndrome: chest pain that may radiate into the left arm, jaw, neck, or left shoulder, difficulty breathing, diaphoresis, dizziness, feeling of impending doom. These symptoms spontaneously resolve when the coronary vasospasm resolves.

Common causes of Printzmetal's angina are exposure to cold, excessive stress, certain medications, and drug use (particularly cocaine). Cocaine-related Printzmetal's angina is sometimes colloquially referred to as "the cocaine chest pain" in emergency medicine as it is a fairly common and well known emergent problem in cocaine users.

Wellens Syndrome

Wellens Syndrome is an EKG change that has a strong correlation with impending anterior STEMI (approximately 75% of patients who present with Wellens Syndrome developed anterior STEMI within 1-2 weeks of noted EKG change in a study in the early 80s)[1]. It is identified as an inverted or biphasic T wave in leads V2 or V3 (sometimes both).

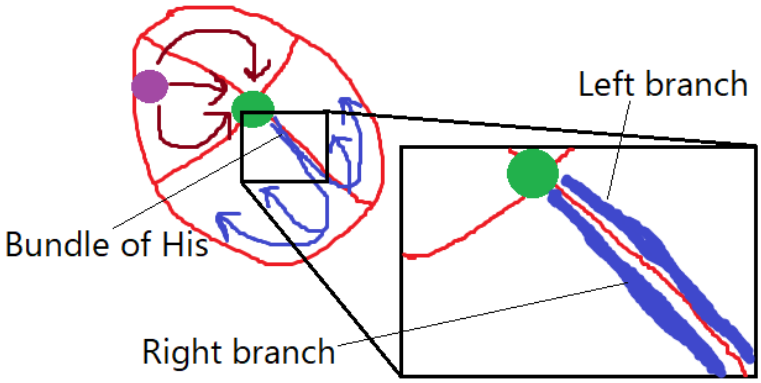
[1] Miner, B., & Hart, E. (2019). *Wellens Syndrome.* Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK482490/



It is a helpful syndrome to identify because it can flag a patient who needs cardiac intervention before they have a life-threatening event like STEMI.

Bundle Branch Blocks

A bundle branch block (BBB) is a similar issue to the AV blocks discussed in an earlier segment with regards to physiology. AV blocks refer to dysfunction of the AV node. A bundle branch block refers to a malfunction of a branch of the bundle of His (the AV node is functioning normally).

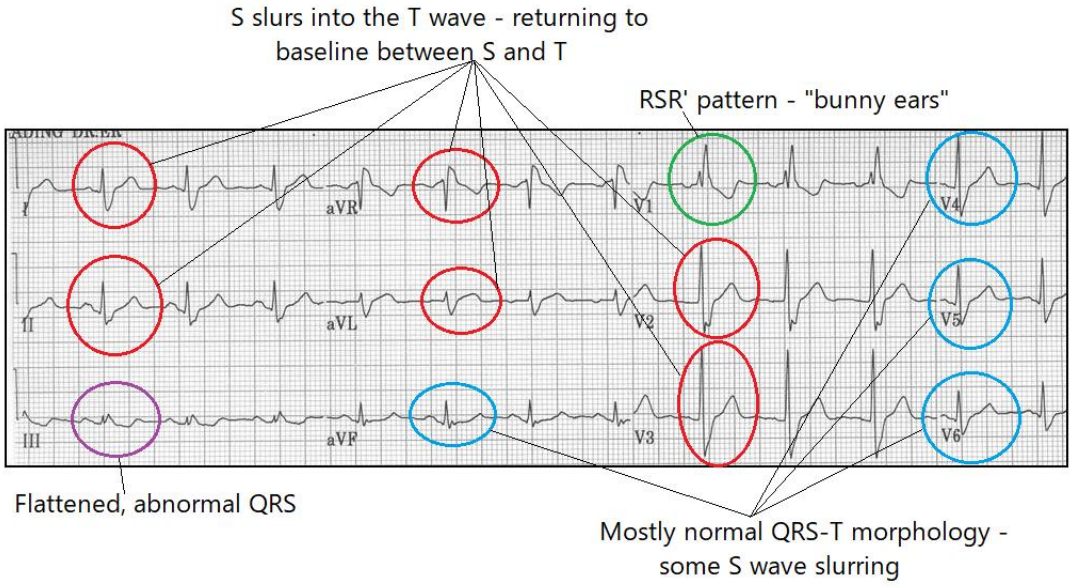


Recall the anatomy of the bundle of His and the electrophysiology of the area. The bundle of His refers to the insulated bundle of cardiac tissue that allows electricity to travel toward the bottom of the ventricles before the ventricular depolarization begins, which causes the ventricles to pump from the bottom upward toward the semilunar valves. The bundle of His is made up of 2 branches (not mentioned previously): one for the right ventricle, one for the left. These are referred to as the "bundle branches."

The bundle branches do not commonly malfunction at the same time, and a malfunction of the right bundle branch looks different from a malfunction of the left. It is also notable that, much like an AV block, a bundle branch block can be partial or complete. Bundle branch blocks can be acute or chronic, and it is important to compare them to a previous EKG if one is available. It is also important to note the patient's chief complaint and symptoms if they have a bundle branch block. A new or suspected-new bundle branch block should be treated with the same urgency as a STEMI. Conversely, many people live with a chronic bundle branch block that is asymptomatic and poses them no immediate threat.

Right Bundle Branch Block

A right bundle branch block (RBBB) occurs when the right branch of the bundle of His is not conducting correctly. This results in slowed conduction to the right ventricle, but normal conduction to the left ventricle, causing the left ventricle to pump normally but the right ventricle to pump slightly after the right ventricle with regards to time. This causes the QRS complex to look abnormally in most leads, and is most prominent in the leads that examine the right side of the heart. The first 3 precordial leads (V1-3) are the most distinct, and you will notice a progression of decreasing severity of the QRS abnormality. V1 is typically the most abnormal looking, V2 will appear somewhat better, V3 will appear better yet, etc as the precordial lead view adjusts toward the normally functioning left view.



Some of the changes can look like ST elevation. You can differentiate true ST elevation from a right bundle branch block by examining the affected leads and the morphology of the QRS complex and by examining if the S wave in the QRS complex returns to the baseline. The above example is a complete bundle branch block. Leads I, II, aVR, and and aVL show slurring as well as in the precordial leads circled red.

RSR' (pronounced R, S, R prime) is a phenomenon in which the R wave appears "interrupted" by the S wave. The R begins in the left ventricle, appears to stop abruptly during the normal S wave occurring in the left ventricle, meanwhile the R wave is occurring in the right ventricle after it occurs in the left because of the slowed conduction down the right bundle. So after the left ventricle's S wave, the R appears on the EKG to restart (the second R is referred to as R' or R prime). This is sometimes called a "bunny ears" appearance because of the two R wave peaks.

An RSR' morphology in a QRS complex will almost always appear to have ST elevation, as it does in the above example, because the T wave in the left ventricle has usually started before or as the QRS complex in the right ventricle completes, so there is no visible return to baseline on the EKG graph. Again, the key to differentiating true ST elevation from a change caused by a problem like a bundle branch block is to examine the QRS morphology and the other leads. While a right STEMI can present with ST elevation visible exclusively in V1 (due to poor depiction of the right side on a standard 12 lead), a right STEMI should not also have an RSR' pattern and may or may not have changes in any other adjacent leads.

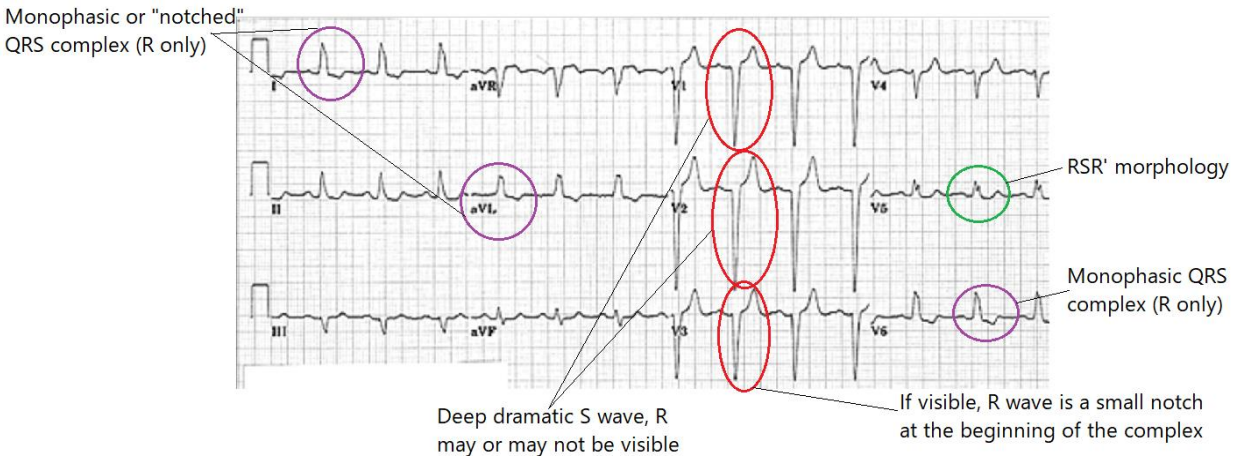
Leads III, V6, and aVF are typically the most normal appearing in a right bundle branch block because their view of the heart is least impacted by the right bundle branch - lead 6 is furthest away from the right bundle branch and aVF is a view that looks from top to bottom at an angle toward the left ventricle.

Left Bundle Branch Block

A left bundle branch block (LBBB) appears somewhat more dramatically than a right bundle branch block does. The right ventricle is smaller than the left, as the right is only responsible for pumping to the pulmonary vasculature where

the left is responsible for maintaining blood flow to the entire body; the differential in size (and thereby electrical output) causes somewhat more dramatic EKG changes compared to the right bundle branch block.

Additionally, while a right bundle branch block is best visualized in the right precordial leads V1-3 and sometimes not apparent at all in V6, a left bundle branch block is often apparent in all of the leads on a standard 12 lead and is most prominent in leads V5 and V6.



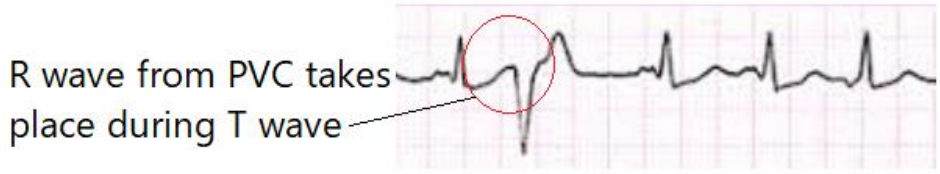
Note that in the circled leads, the QRS complexes still look somewhat abnormal and mono-directional. In a left bundle branch block, the QRS complex in the right ventricle starts before it does in the left. This is best visible in the precordial V1-3 leads as a deep, dramatic S wave with minimal other visible waves in the QRS complex. Many of the leads appear to mostly go in one direction because the right side depolarizes first, then the left; as the electricity in the right side is flowing away from the unipolar electrodes during the S wave at the end of its QRS complex, the left side is beginning its depolarization as the right is ending, resulting in electricity flowing away from the right precordial electrodes in a much larger quantity than compared to normal depolarization (remember that the height of a wave correlates to the amount of electricity detected by the electrode). The R wave may or may not be visible, depending on the exact timing of the electrical flow; if it is, it is usually a very small upward notch at the beginning of the S wave.

The same RSR' morphology is sometimes present with a left bundle branch block, but is usually in the left-facing V5 lead. The above green circled complex is an RSR' morphology; sometimes there is a similar RS morphology (which is the same except the R' is absent).

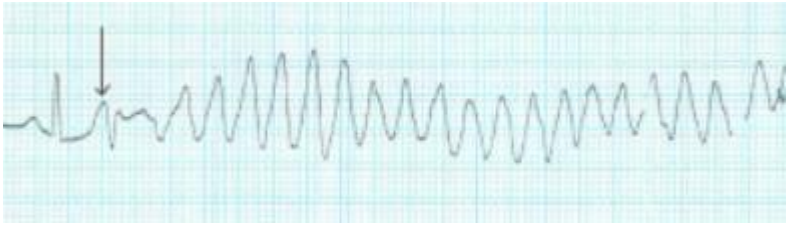
You will also notice monophasic QRS complexes in many leads, prominently in lead I, aVL, and V6. The QRS complex may be monophasic, or consist of a singular R wave, or have a small notch near the top, as aVL does in this example.

R-on-T Phenomenon and Torsades de Pointes

R-on-T phenomenon is an anomaly in which a T wave (ventricular repolarization) is interrupted by an R wave (ventricular depolarization). This is most commonly caused by a PVC or an SVT. Prolonged QT intervals can also predispose a patient to R-on-T phenomenon.

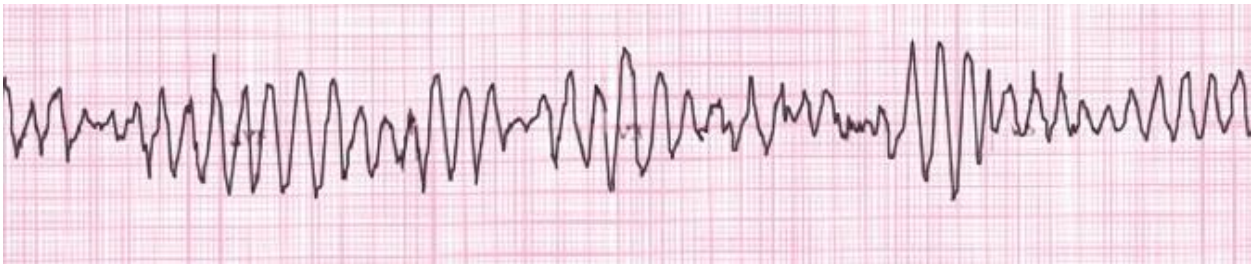


Occasionally this can cause irregularities in the depolarization-repolarization pattern in adjacent ventricles, leading to a unique type of ventricular tachycardia called torsades de pointes ("turning of the points"). The heart is most prone to this if it is already experiencing some degree of ischemia; this phenomenon is extremely rare absent any other cardiac pathology.



Torsades de pointes is a type of polymorphic ventricular tachycardia, or a ventricular tachycardia that changes in QRS morphology. Refer to our review of premature ventricular contractions: QRS morphology for ventricular rhythms is dependent on where the depolarization originates from. A polymorphic ventricular tachycardia originates from multiple different sites. In the case of torsades de pointes, it is a "rotating" of sites in a circular, gyrating pattern around the heart.

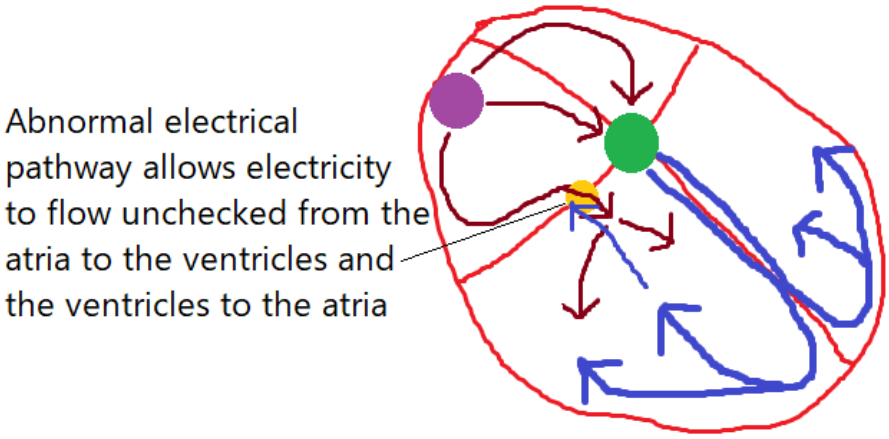
This gyrating pattern of depolarization origination causes a pattern that seems to twist around the isoelectric line.



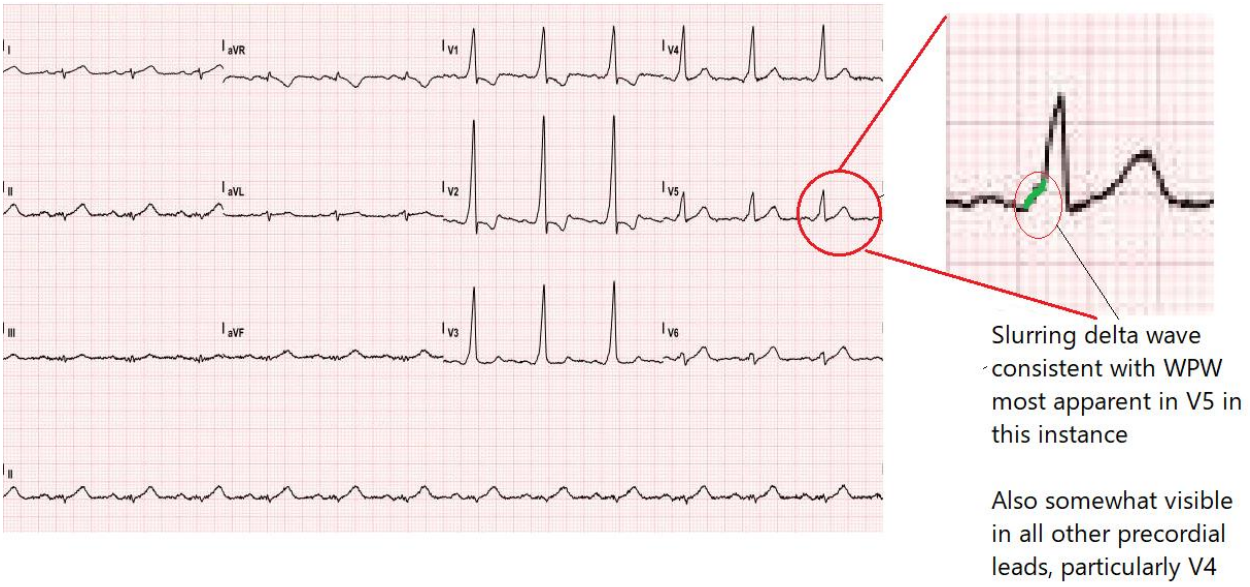
Torsades de pointes is a lethal arrhythmia and is treated with defibrillation. It can also be treated with IV magnesium, though the mechanism through which this treatment works is poorly understood. If it is not treated, it usually converts into ventricular fibrillation and ultimately asystole.

Wolff-Parkinson-White Syndrome

Wolff-Parkinson-White syndrome (WPW) is a common cause of sudden unexpected cardiac death in athletes and young adults. It is caused by an inherited abnormality in the electrophysiology pathways of the AV node. In a normal heart, the AV node is the only pathway between the atria and the ventricles and only allows electrical currents to flow in from atria to ventricles. WPW is caused by an extra pathway (called an accessory pathway) that also connects the atria to the ventricles. This means that the AV node is not a reliable gatekeeper between the atria and ventricles and electrical impulses can bypass the AV node, which causes the ventricles to begin the depolarization process in an abnormal pattern originating from the accessory pathway (referred to as pre-excitation). Additionally, the extra unregulated pathway means electricity from the ventricles can reach the atria.

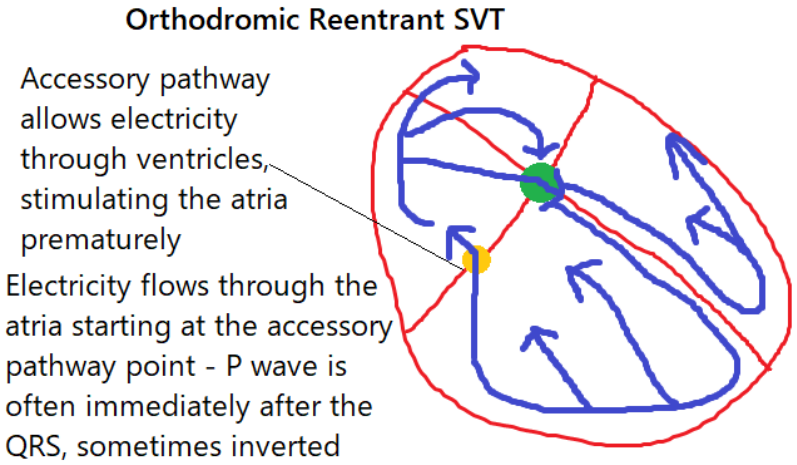


The flow of electricity from the atria to the ventricles through the accessory pathway can be seen on an EKG as an early slurring from the baseline to the QRS complex, called a delta wave. The slurring can usually be seen in most or all leads, but will be more prominent in leads viewing the part of the heart the accessory pathway is located in (ie: if the accessory pathway is on the right as in the picture, the delta wave should be most prominent in V1-2 and aVR). A WPW patient may or may not always have visible EKG changes consistent with the condition as some accessory pathways are semi-selective in the same way the AV node does not let all impulses through every time.

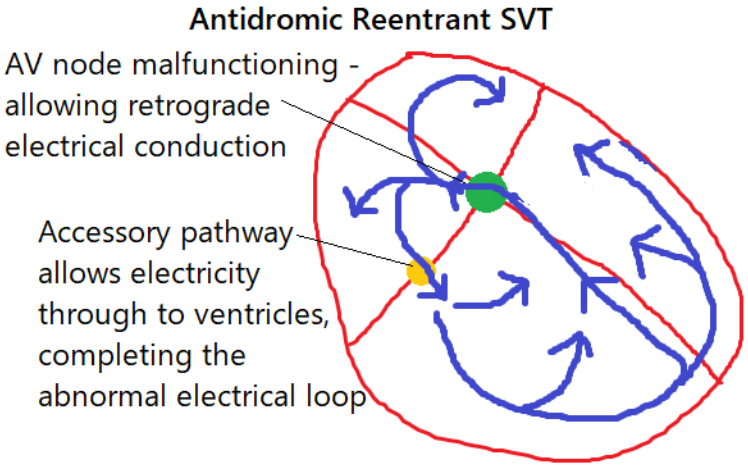


The delta wave can be subtle and difficult to notice, and generally requires a moderate to large amount of EKG interpretation experience to detect.

WPW can cause sudden cardiac death in a similar manner to R on T complex. In short: The SA node starts an impulse, the impulse travels both normally to the AV node and abnormally through the accessory pathway. The accessory pathway excites and starts ventricular depolarization early. The AV node releases its impulse after the ventricles have already started depolarization, stimulating ventricular tissue that is not necessarily repolarized and ready for another depolarization. Similarly to R-on-T complex, this can cause spontaneous ventricular fibrillation or torsades de pointes.



WPW patients are also prone to a problem called reentrant tachycardia, which is a type of supraventricular tachycardia (SVT). This occurs when the electricity from the ventricular depolarization loops back up to the atria again using the accessory pathway, stimulating them to depolarize in fast succession (as in the drawing above). This results in an electrical loop that causes a rapid heart rate. It is difficult to correctly differentiate SVT causes based on EKG, but a reentrant SVT features P waves that may or may not be visible and may be after the QRS complex (similar to a junctional rhythm). The QRS morphology is usually normal; this specific type of reentrant SVT is referred to as orthodromic.



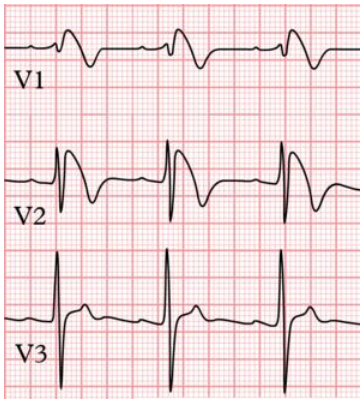
Occasionally the AV node also fails and allows retrograde electrical flow (ie: flow from the ventricles into the atria), which can cause a wide QRS complex and a circuit that loops in the opposite direction. This is referred to as an antidromic reentrant SVT. It is very difficult to differentiate both clinically and based on EKG from a ventricular tachycardia, as it is a wide complex tachycardia, and it is usually treated in the same way a ventricular tachycardia would be treated (synchronized cardioversion or defibrillation).

Wolff-Parkinson-White syndrome is, in most patients, asymptomatic and they are often unaware they have the condition. It is usually caught during an SVT event in which a patient presents for palpitations, dizziness, syncope or pre-syncope, chest pressure, or shortness of breath. It is also sometimes caught on routine EKG. If it is symptomatic or a patient is in an occupation that is considered to be high-risk, such as an athlete, the patient may undergo a cardiac ablation to remove the accessory pathway. Ablation is typically not recommended in otherwise asymptomatic patients with low occupational risk.

Brugada Syndrome

Brugada syndrome is an inherited dysfunction of the sodium channels. Recall the physiology of the heart: sodium cations (positive) are allowed to flood in via the voltage-gated fast Na+ channel, allowing the cardiac muscle cell to reach action potential and contract. Refer to the EKG Primer Part 0 segment on depolarization for more details about the fast Na+ channel's function.

Dysfunction of the fast Na+ channel alters the way the heart depolarizes, which affects both the way the muscles contract and the electrophysiology of the heart. It can be difficult to diagnose or identify correctly on EKG, but it is most commonly identified as ST elevation in any of the leads V1-3 with an inverted or biphasic T wave in the same lead.



Brugada syndrome also requires clinical criteria for diagnosis, as the EKG changes alone are not specific enough to definitively diagnose. Other criteria include sudden cardiac death or known Brugada in a member of the immediate family at a young age (generally <40), known family history of similar EKG abnormalities, personal history of life-threatening arrhythmia like ventricular fibrillation, and syncope. Brugada diagnosis is an evolving area of cardiology.

Brugada patients have a high incidence of sudden cardiac death. Brugada syndrome is thought to be a cause of some cases of SIDS, as deaths from Brugada syndrome can occur at any point in the lifespan of an affected individual. It is thought to be a possible cause (or possibly the same syndrome, named differently) of other sudden cardiac death syndromes described in Southeast Asia and Africa such as SUNDS (sudden unexpected nocturnal death syndrome), Pokkuri syndrome, and bangangut.

Numerous genes that cause functional abnormalities in the fast Na+ channels which ultimately causes Brugada syndrome have been isolated, but many of the responsible genetic defects have not yet been identified. The description as a fast Na+ channel anomaly and name Brugada syndrome are fairly new in Western medicine (described for the first time in the West in 1992), though as discussed above other non-Western medical practices may have had other names and differing descriptions of this syndrome long predating Western recognition.