

*fast*

EKG, ECG Interpretation

4.00 Contact Hours

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Outcomes

≥92% of participants will be able to recognize life-threatening ECG rhythms and understand the importance of ECG monitoring.

Objectives

After this course, the participant will be able to:

1. Describe normal cardiac anatomy and normal electrical conduction through the heart.
2. Identify and relate ECG waveforms to the cardiac cycle.
3. Characterize the different lead placements and the purpose of each lead placement.
4. Utilize a systematic process when approaching the interpretation of the ECG.
5. Differentiate normal and abnormal components on ECG.
6. Recognize sinus, atrial, junctional, and ventricular dysrhythmia on ECG and relate cause, significance, and symptoms.
7. Identify three pacemaker malfunctions.

Introduction

Electrocardiograms are used in the ambulance, emergency room, surgery, intensive and critical care, or any other hospital room to diagnose a suspected heart attack, syncope, abnormal vital signs, or pulse. An ECG test is included in routine exams for middle-aged and older adults, as they have a higher risk of heart disease than younger people. Every health professional needs to know what an ECG is and its importance in the properly and timely identification of cardiovascular system issues.

The cardiovascular system aims to supply an adequate amount of blood to all bodily tissues to meet their metabolic demands. The arterial system supplies tissues and organs throughout the body with oxygen, nutrients, hormones, and immunologic substances. The flowing blood regulates the body's temperature. Venous return removes wastes from tissues, routing deoxygenated blood through the lungs to excrete metabolic wastes (Reference.com, 2020).

The heart is a unique organ, possessing several distinctive properties. It works as a pump, expanding and contracting without adding stress to the cardiac structure and developing muscle fatigue. The heart pumps 4 to 8 liters each minute. This rate is equivalent to upwards of 6,000 liters per day. It has an inherent capability to generate electrical impulses and use them to maintain proper rhythm regardless of other factors, such as heart contraction rate. Our heart can ignore inappropriate electrical signals that might otherwise overstimulate the cardiac muscle (Miranda, 2021).

Your heart is the size of your fist, and as small as it is, it carries an impressive workload over a lifetime. It beats 60 to 100 times per minute without resting. The heart must be flexible and able to adjust to changes in the body's metabolic demands, often in a matter of seconds. Vigorous exercise can increase the metabolic requirements of muscles by as much as 20 times over their needs during rest. To meet the increased demands of the muscles, the heart accelerates its rate to increase cardiac output. Vessels must redistribute blood flow, shunting a greater proportion of blood to muscle tissues and away from internal organs.

Oh, and because it is a curious bit of data you can use to get a client's interest engaged, every minute, the body's entire volume of blood circulates three times from the heart and back again! (Miranda, 2021).

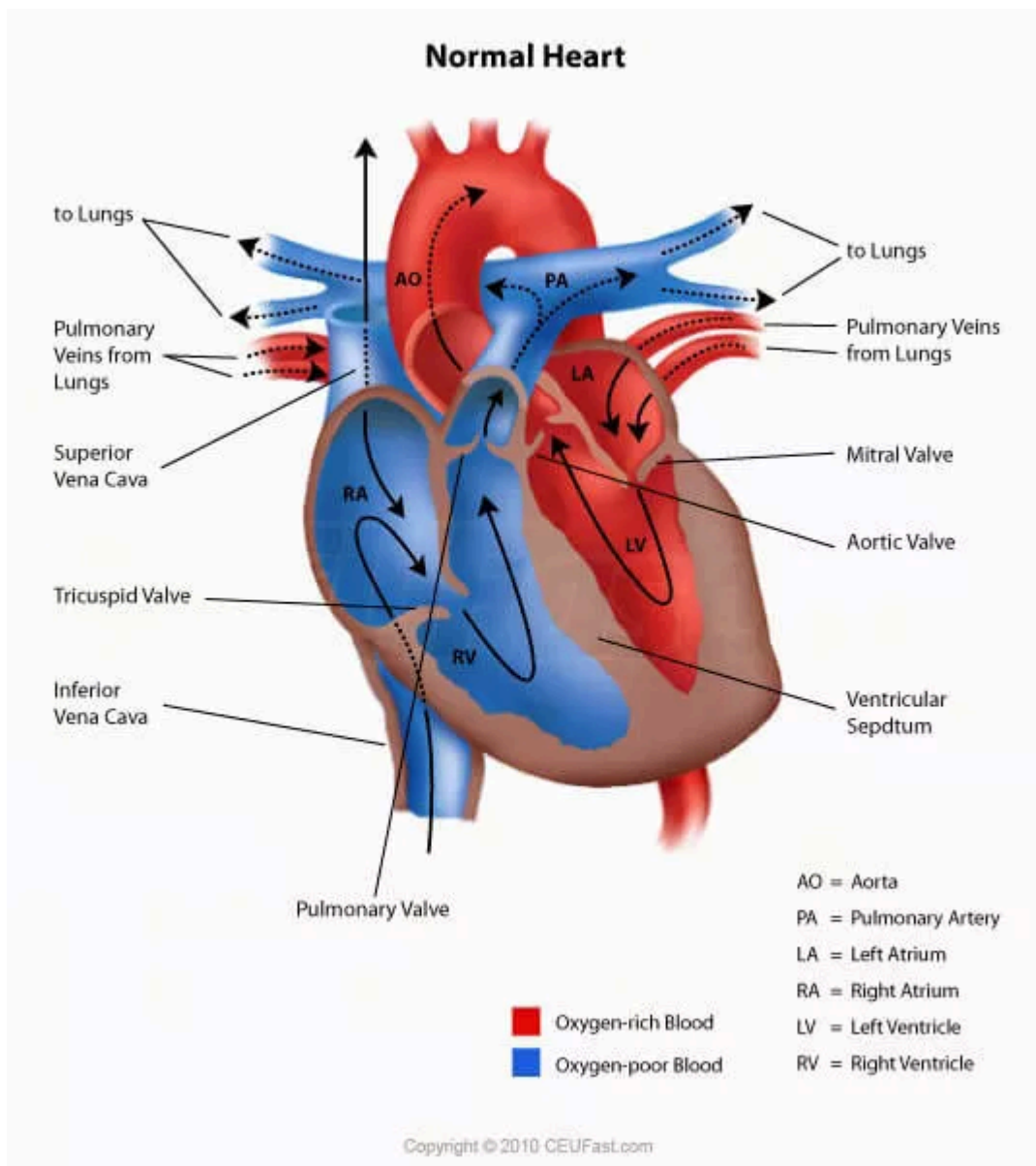
Now, something important. An Electrocardiogram (ECG) is the graphic measurement of the electrical activity within the heart. An older abbreviation stemming from the German term "electrocardiogram," EKG is used interchangeably with ECG. Yet why? There are several rationales. However, I favor the chaos speech notion from working in critical care and having led off-hours code response in a community trauma center. That is, in the heat of an emergency, calling verbally for an "EKG" is better understood than calling for an "ECG," which might be misconstrued as an "EEG," or electroencephalogram, brain wave imaging. For the sake of technical accuracy, we will be using the term ECG by preference when talking about electrocardiograms (Ferguson, 2021).

The ECG (EKG) is a valuable diagnostic tool for the healthcare provider, whether they are a doctor, nurse, emergency responder, or even a rehabilitation specialist. Understanding the ECG enables the healthcare provider to respond correctly and treat dangerous and potentially deadly arrhythmias as quickly and efficiently as possible. It is important to understand the mechanisms and know just what needs to be done to care for clients with these serious arrhythmias. New drugs and high-tech equipment which can cardiovert, defibrillate, or even serve as a pacemaker are constantly being evaluated and introduced into the healthcare system.

Anatomy and Physiology

The heart is a hollow, chambered, muscular organ located in the middle of the thoracic cavity, cradled in a cage of bone, cartilage, and muscle. It lies left of the midline of the mediastinum and just above the diaphragm. The heart is protected anteriorly by the sternum and posteriorly by the spine. Lungs are located on either side. The entire heart is enclosed in the fluid-filled pericardial sac. This sac helps to shield the heart against infection and trauma, prevents friction, and aids cardiac function by helping with the free pumping action of the heart. The heart consists of three layers: Epicardium, Myocardium, and Endocardium (Bianco, 2021).

Image 1:

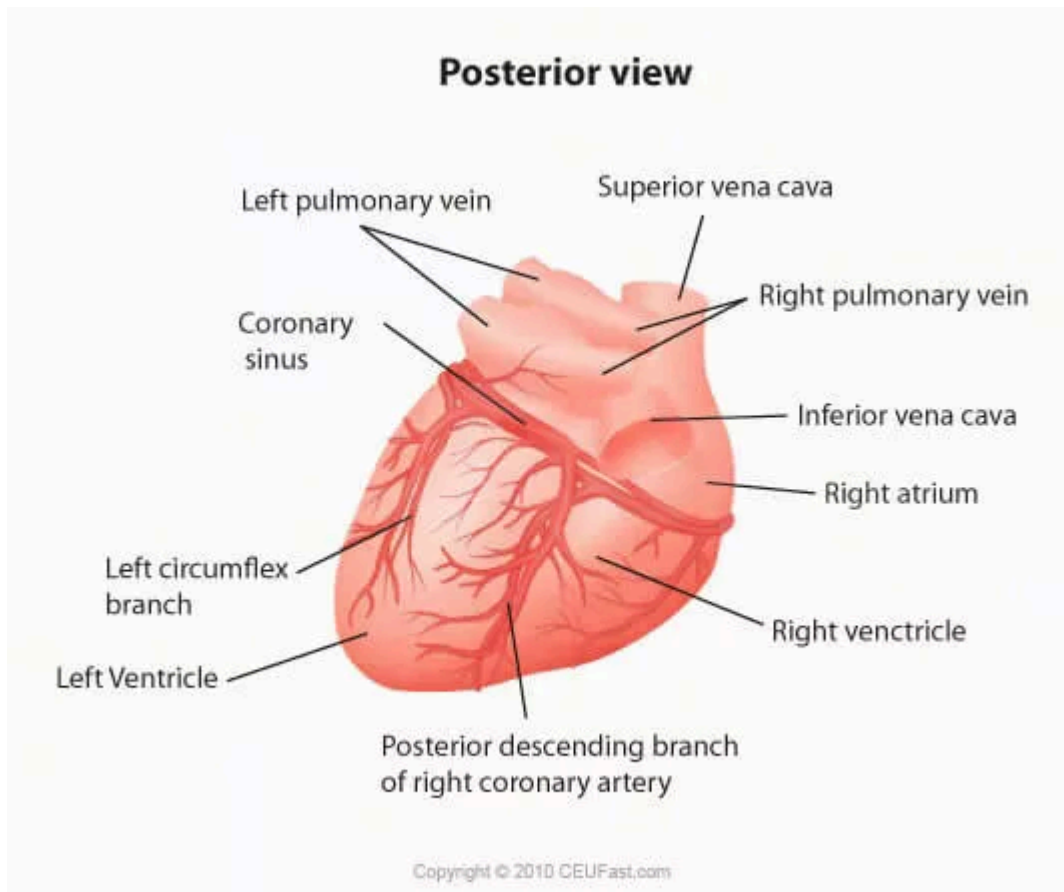


Function

Activities of the right side of the heart and the left side of the heart occur simultaneously.

The right side of the heart receives deoxygenated blood from the body via the vena cava into the right atria. Blood is ejected from the right atria into the right ventricle. Blood is pumped to the lungs from the right ventricle via the pulmonary artery. The left side of the heart receives oxygenated blood from the lungs via the pulmonary vein into the left Atria. Blood is ejected from the left atria to the left ventricle. Blood is pumped to the body from the left ventricle via the aorta. The right side of the heart pumps blood into the lungs. The left side pumps blood into the body.

Image 2:

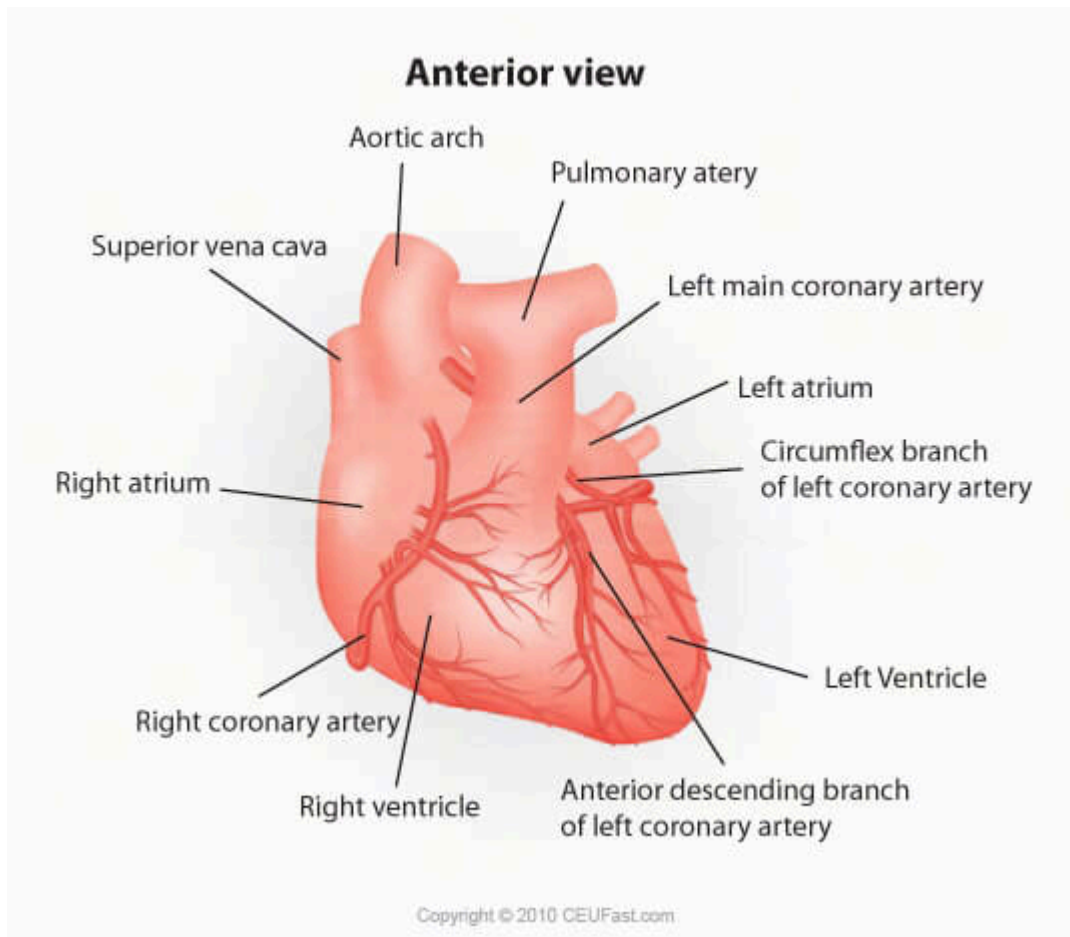


The septum and valves separate the two atria and two ventricles of the heart. Blood is pumped through the chambers, aided by four heart valves. The valves are open and close to let the blood flow in only one direction. The four heart valves are:

- The tricuspid valve located between the right atrium and the right ventricle
- The pulmonary (pulmonic) valve between the right ventricle and the pulmonary artery
- The mitral valve between the left atrium and left ventricle
- The aortic valve between the left ventricle and the aorta

Each valve has a set of "flaps" (also called leaflets or cusps). The mitral valve normally has two flaps; the others have three.

Image 3:



Coronary Arteries

Our heart supplies or pushes oxygenated blood to the cells throughout the body. It is not surprising that the heart itself also needs oxygenated blood delivered to its busy muscle cells!

Yes, Coronary arteries, the ones frequently implicated in myocardial infarctions, are the delivery arm for oxygen to myocardial cells.

Right Coronary Artery

Supplies:

- Right Atrium, Anterior Right
- Posterior and Papillary Muscle Wall Ventricle
- Posterior Aspect of Septum (90% of the population)
- Sinus and AV Nodes (80-90% of the population)
- Inferior aspect of Left Ventricle

Left Coronary Arteries

Left Anterior Descending (LAD) Supplies:

- Anterior Left Ventricular
- Anterior Interventricular Septum

Septal branches supply:

- Conduction system, Bundle of HIS, and Bundle branches
- Anterior papillary muscle

- Left ventricular apex

Circumflex Artery

Supplies:

- Left Atrium
- Posterior surfaces of Left ventricle
- Posterior aspect of the septum

(Bianco, 2021)

Electrical Activity of the Heart

The human heart is a remarkable piece of engineering. The human heart beats 80,000 to 100,000 times daily and pumps approximately 2,000 gallons every day. The heart will have beat 2-3 billion times and pumped 50-65 million gallons of blood over a 70-90-year lifespan. The human heart is made of specialized muscle capable of sustaining continuous beating. This muscle is different from the skeletal muscle that powers the arms and legs. Specialized areas of the myocardium exert electrical control over the cardiac cycle. These areas exhibit physiological differences from the rest of the myocardium, forming a pathway for electrical impulses which energize the heart muscle. The two types of cardiac cells are contractile and conductive. When myocardial cells are at rest, they are electrically more negative on the inside with respect to the outside of the cell. Charged particles (ions) of sodium and potassium move in and out of the cell, causing changes sensed by electrodes on the skin. The heart's electrical action will show as a tracing on the ECG (Kohli, 2020).

The sinoatrial (SA), or sinus node, initiates a self-generating impulse and is the primary pacemaker which sets a rate of 60 to 100 beats per minute (bpm). The SA node is located at the border or junction of Superior Vena Cava and Right Atrium. Once generated, this electrical impulse sets the rhythm of contractions and travels through both atria over a specialized conduction network to the Atrioventricular (AV) Node. The AV node is in the floor of the Right Atrium and receives the contraction impulse, which it transmits to the Bundle of His. The Bundle of His then divides the conduction pathway into a right bundle branch and two left bundle branches. These terminate in a complex network called the Purkinje Fibers, which spread throughout the ventricles. When the impulse reaches the ventricles, stimulation of the myocardium causes depolarization of the cells, and contraction occurs. The AV node serves as a gate to delay electrical conduction and, in this way, prevents an excessive number of atrial impulses from entering the ventricles (Bailey, 2020).

The SA node and AV Nodes are supplied with sympathetic and parasympathetic fibers. These nodes enable nearly instantaneous changes in the heart rate in response to physiologic changes in oxygen demand.

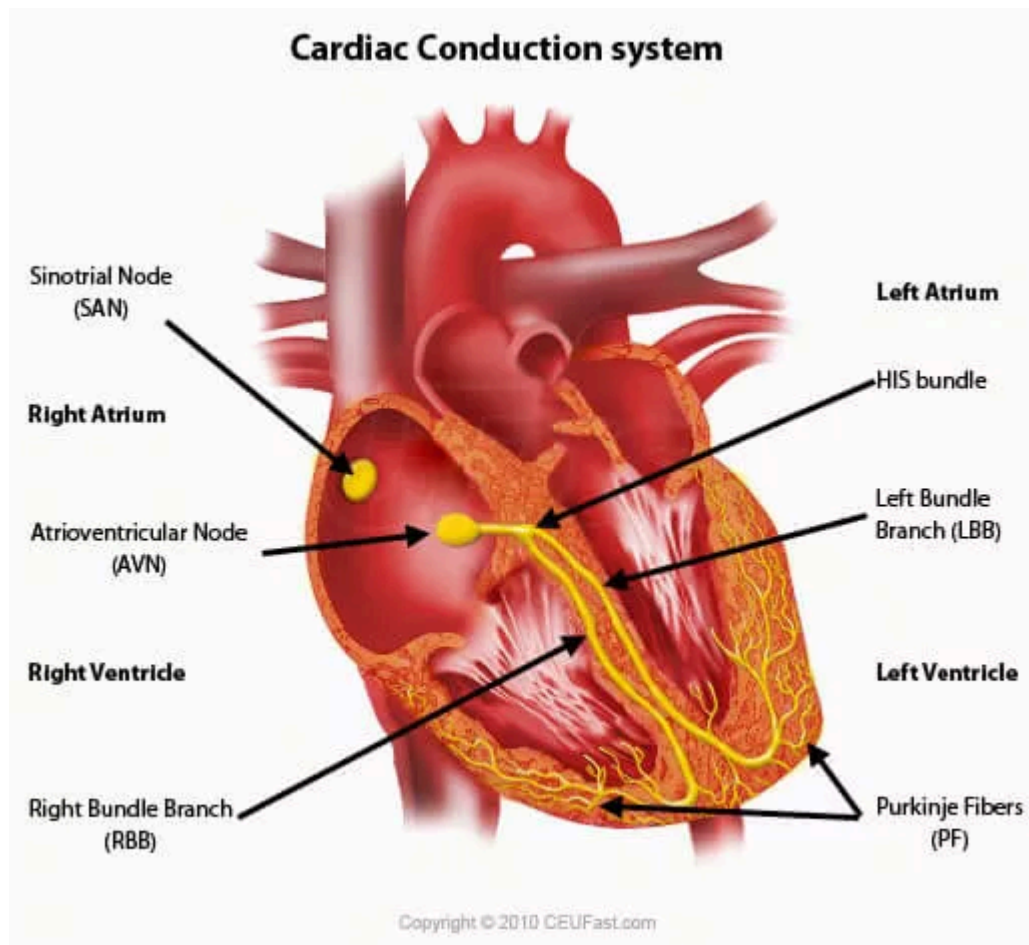
The normal cardiac conduction occurs in this sequence:

- Sinoatrial node initiates an electrical impulse and sends this impulse thru the atrium > lower section whereby an Atrial Kick occurs > AV node > Bundle of His thru ventricles via > Right Bundle & Left Bundle Branches > Purkinje fibers into the ventricular muscle.
- If the SA node falters, a hierarchy of backup pacemakers can take over. Atrial, AV node, and ventricular escape pacemakers can function as subsidiary pacemakers. However, they

generated impulses at a much slower rate. The AV node generates rates between 40 to 60 bpm and the Purkinje fibers at 20 to 40 bpm.

- Electrical impulse does not always equal the contraction of the heart. Accessory pathways play a role in re-entry tachydysrhythmias, providing a detour for unwanted electrical impulses to circle through the heart.

Image 4:



Components of the Electrical System

There are two basic cardiac cell types.

Myocardial muscle cells (mechanical cells) are the myocardium or the body of the heart. These contain contractile filaments that contract when the cells are electrically stimulated. Their primary function is contraction and relaxation. Their primary property is contractility.

Electrical cells (pacemaker cells) are electrical impulse generating cells found in the electrical conduction system. They conduct impulses very rapidly, and their primary property is automaticity and conductivity.

Electrophysiological Properties of a Cardiac Cell

Cardiac cells are surrounded by and filled with a solution that contains ions. Three key ions are sodium (Na^+), potassium (K^+), and calcium (Ca^{++}). In the resting period of the cell, the inside of the cell membrane is considered negatively charged, and the outside of the cell membrane is

positively charged. The movement of these ions inside and across the cell membrane constitutes a flow of electricity that generates the signal on an ECG (Hill, 2021).

Electrical Events of Depolarization and Repolarization

Cardiac cells that are resting have a negative polarization. Sodium ions are outside of the cell, and potassium ions are inside the cell. Both ions carry a positive charge; however, the sodium ion has a stronger charge than the potassium. Thus, the inside ion state of the cell electrically is weaker than the outside, so it is negative. The polarized state is a "ready state." When the cell is ready to accept an electrical impulse, a large amount of potassium leaks out. This leak causes a discharge of electricity, and the cell becomes positively charged. This discharge is called depolarization. The electrical wave created at this pacemaker site travels from cell to cell along the electrically sensitive conduction pathways and from there throughout the heart. Now begins cell recovery. Sodium and potassium ions are shifted back to their original place by the sodium-potassium pump. This process is called repolarization.

Action Potential of a Myocardial Working Cell

Electrical impulses result from a brief but extremely rapid flow of positively charged ions (mainly Na^+) back and forth across the cell membrane.

Cardiac action potential illustrates the changes in the membrane potential of a cardiac cell during depolarization and repolarization.

Five electrochemical phases are starting with Phase "o". Yes, that is correct, zero through four, totaling five phases. Just go with it. Clinical scientists' minds have odd quirks (Beck, 2018).

Phase o.

Rapid Depolarization ends the cell relaxation state. Thus, presumably, the "o" designation indicates coming to a readiness for the beginning of cell contraction. The electrical activity which mirrors physiological contraction is also known when considering the atrial portion of the heart as a "P" wave. Depolarization with the ventricular heart is a QRS complex.

- This phase is also called "upstroke," "overshoot," or "spike"
- Begins when a cell receives an impulse
- Sodium moves quickly into the cell through the fast sodium channels
- Potassium then leaves the cell
- Calcium moves slowly into the cell through calcium channels
- This movement produces about +20 mV or 20 micro volts of positive electrical power
- Cell depolarizes, and the process of cardiac contraction begins

Phase 1.

Early Repolarization, e.g., "partial" repolarization of the cell membrane due to sodium ion passage creating an electrical current

- The Rapid flow of sodium into the cell is stopped as the fast sodium channels close
- Potassium begins to reenter the cell, and sodium begins to leave
- This movement is about 0 mV and is therefore neutrally charged, neither positive nor negative charged
- This action is the absolute refractory period

Phase 2.

Plateau Phase (slow repolarization, part of absolute refractory period). Calcium shift causes a stasis or “holding” of the electrical charge. In the electrical mirror of heart activity, think of this as the “T” interval.

- Slowly repolarization continues
- Calcium continues to flow into the cell through slow calcium channels

Phase 3.

Final Repolarization of the cell as calcium and sodium movement is shut down. Leaving the cell at a baseline charge and raring to go.

- Rapidly the cell completes repolarization
- Calcium channels close
- Potassium rapidly flows out of the cell
- Active transport via the potassium-sodium pump begins restoring potassium to the inside of the cell and sodium to the outside of the cell
- Cell now in the negative state due to the outflow of potassium
- Gradually the cell becomes extremely sensitive to external stimuli until its original sensitivity has been restored, called the relative refractory period.

Phase 4.

Return to Resting Stage, at the resting potential of -89 millivolts (mV). Do not be fooled. Holding a negative charge means the outflow or net efflux from the cell is a positive charge (Beck, 2018).

- Corresponds to diastole
- Calcium and sodium remain outside the cell
- Potassium remains inside the cell
- During this phase, the heart is "polarized" and getting ready for discharge
- Once another stimulus occurs, the cell will reactivate

Image 5:

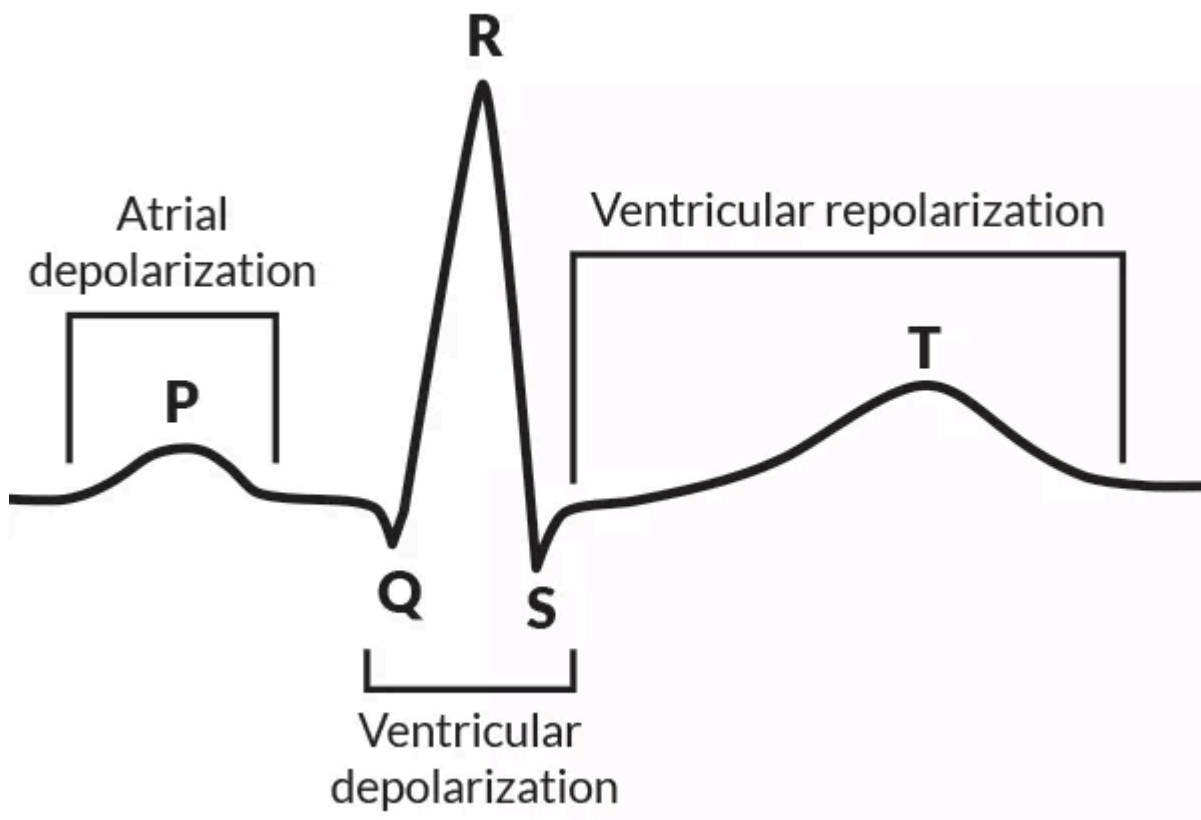




Image Source: ECG Cycle vs. Action Potentials, complements of Wikimedia Commons

Keys to Remember:

- Electrical events show as tracings on the ECG (EKG)
- Depolarization and Repolarization are Electrical Events
- Contraction and Relaxation are Mechanical Events

Properties of the Heart

Automaticity

The heart can initiate an electrical impulse. The heart can begin and maintain rhythmic activity without the aid of the nervous system. A heart removed from the body can beat on its own for a limited period. The highest degree of automaticity is found in the pacemaker cells of the sinus node. The atria, atrioventricular (AV) Node, Bundle of His, bundle branches, Purkinje Fibers, and the ventricular myocardium have less automaticity.

Excitability

The heart can respond to an electrical impulse. A cardiac cell will respond to an electrical stimulus with an abrupt change in its electrical potential. Each cardiac cell that receives an electrical impulse will change its ionic composition and its respective polarity. Once an electrical potential begins in a cardiac cell, it will continue until the entire cell is polarized.

Conductivity

The heart can conduct an electrical impulse. All areas of the heart appear to depolarize simultaneously because a cardiac cell transfers an impulse to a neighboring cell very rapidly.

The velocity of the impulse conduction transfer varies in the different cardiac tissues:

- 0.05m/s in the SA node
- 1.0m/s between the SA node and AV node
- 0.05m/s in the AV node
- 1.5m/s in the bundle of His
- 4.0m/s in the Purkinje fibers

(Greek.Doctor, 2020)

Contractility is the ability of the heart to respond by contracting.

Conduction System

The heart is all about electricity.

The normal cardiac impulse arises in the specialized pacemaker cells of the SA node, located about 1 mm beneath the right atrial epicardium at its junction with the superior vena cava. The impulse then spreads over the atrial myocardium to the left atrium via Bachmann's bundle and the AV node region via the anterior, middle, and posterior internodal tracts connecting the sinus and AV nodes. These represent the usual routes of spread but are not specialized tracts analogous to the Purkinje system. When the impulse reaches both atria, they depolarize electrically, producing a P wave on the electrocardiogram (ECG) (EKG), and then contract mechanically, producing the wave of the atrial pressure pulse and propelling blood forward into the ventricles.

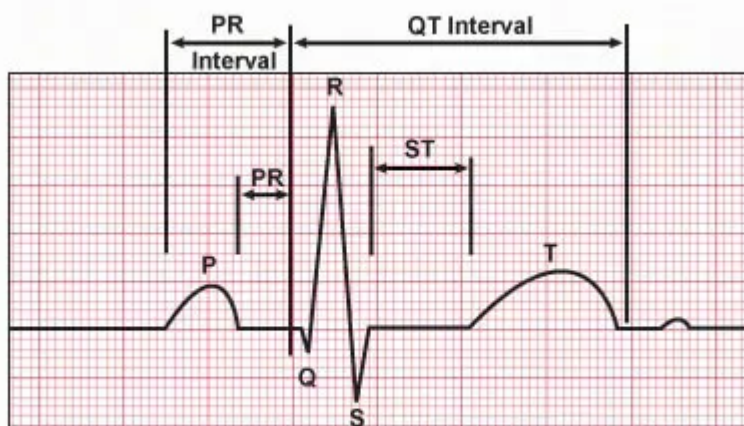
Conduction slows when the impulse reaches the AV node, allowing sufficient time for blood to flow from the atria into the ventricles. After the impulse emerges from the AV node, conduction resumes rapidly through the Bundle of HIS to the Right and Left Bundle Branches. It terminates in the Purkinje Fibers in the ventricular muscle.

Stimulation of the myocardium causes progressive contraction of the myocardial cells. Therefore, wave deflections correspond to the cardiac cycle's mechanical events, including contraction and relaxation of the cardiac chambers. Repolarization is only electrical, and during that, the heart is at rest.

Three major waves of electric signals appear on the ECG. Each one shows a different part of the heartbeat.

- The first wave is called the P wave. It records the electrical activity of the atria.
- The second and largest wave, the QRS wave, records the electrical activity of the ventricles.
- The third wave is the T wave. It records the heart's return to the resting state.

Image 6:



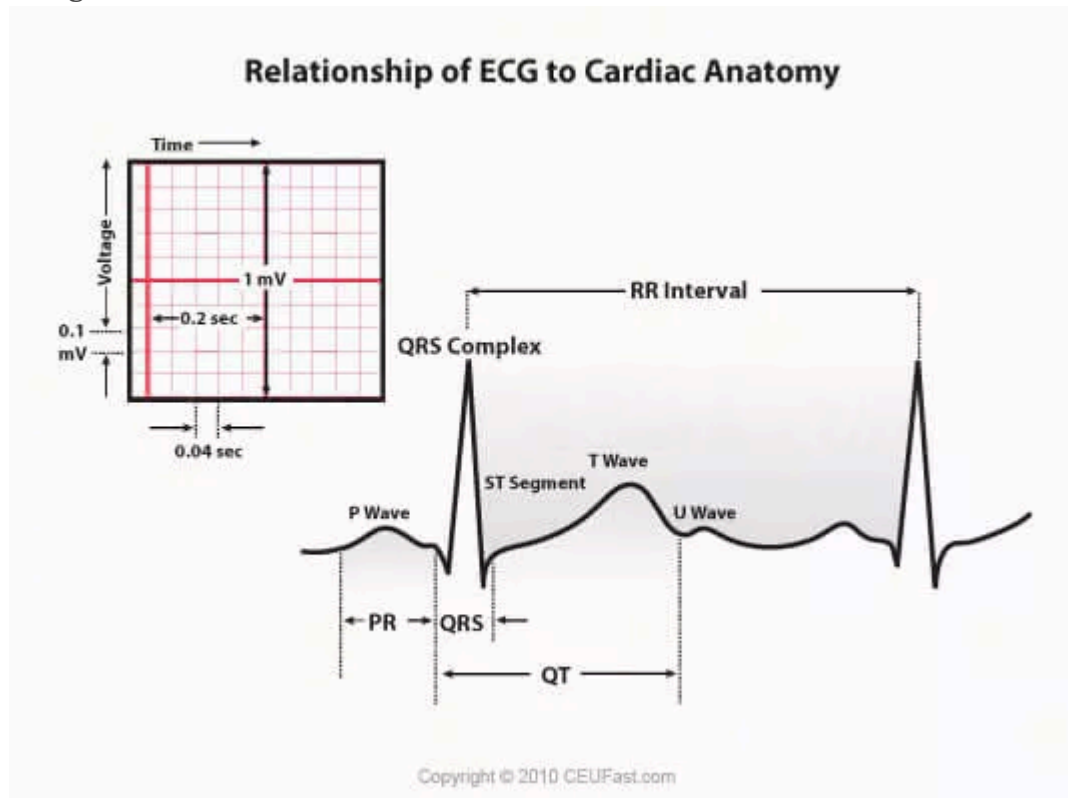
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The P wave represents atrial electrical activation. The PR interval is the time from the onset of atrial activation to the onset of ventricular electrical activation. The QRS complex represents the ventricular electrical activation spike. The QRS duration is the length of ventricular

activation. The ST-T wave represents ventricular repolarization. The QT interval is the duration of ventricular activation and recovery. The U wave, that little bump of mystery on the electrical tracing of heart activity, probably represents "after depolarization" in the ventricles (My.EKG, 2021).

Image 7:



An electrocardiogram is a recording of the heart's electrical output. This recording gives a visual index of the mechanical activity occurring with every life-giving beat. The background format of an ECG strip is a graphic field and known as electrocardiograph strip paper. An electrocardiograph tracing moves at a 25mm/sec speed, and time intervals are heat inscribed onto the special paper as the horizontal X-axis. At the same time, electrical strength (voltage) is scribed onto the Y-axis (vertically). Each large square is 0.2 seconds, so five large squares make one second. Each 0.2-second square is further divided into five small squares of 0.04 seconds each.

Voltage, or the strength of the heart's electrical impulses, is represented on the vertical Y-axis. Each micro-volt, mV, positive electrical current raises the stylus 1cm or one large square. The waveform baseline or bioelectric line is electrically neutral, usually without any deflections, e.g., a flat line.

P Wave.

A "P" wave on an ECG represents atrial depolarization. This wave represents one electrical activity associated with an impulse from the SA node and the electrical spread (conduction) through the atria. The SA node is, physiologically, located in the right atrium, so the right atrium begins constriction or depolarization. This depolarization represents the first half of the "P" wave. As depolarization spreads to the left atrium, we observe that process in the second half of the P wave. The total duration of the P wave should be, in general, three small squares, with 2.5 small squares of height.

In leads I and II, which we will get to soon, P waves should always be positive or above the baseline, while in lead aVR, again, looking ahead, in normal sinus rhythm, it should be negative. And here is a teaser to whet your interest; when you see an abnormal P wave, consider, strongly, the presence of atrial changes such as myocardial enlargement (Davey, 2018).

P-R Interval.

"P-R" Interval represents the time from the start of atrial depolarization, P wave, to the beginning of the QRS, or ventricular depolarization. The normal P-R interval is three to five small squares or 0.12 to 0.20 seconds. Long PR intervals might indicate a first-degree AV block presence, while short intervals accompany disorders of AV acceleration such as Wolf-Parkinson-White (WPW) syndrome.

QRS Complex.

"QRS" represents ventricular depolarization (phase 0 of the action potential) until the end of ventricular depolarization. Generally, the duration of the entire QRS is under 120ms or three small squares. An extended QRS might indicate the presence of a bundle branch block or hyperkalemia. Early ventricular contractions (premature ventricular contractions or PVCs) are frequently associated with a wide QRS. So, remember to count boxes in the QRS when you see those pesky PVCs.

Q Wave.

"Q" is the initial, tiny, downward, or negative deflection of ventricular depolarization, contraction. To be more exacting, Q represents depolarization of the intraventricular septum, the membranous and muscular partition separating the right from the left ventricles of the heart.

The normal Q wave is small and easily missed. It is less than 25% of the R wave amplitude and does not exceed 0.04 sec in duration, one small box. Abnormal Q waves are a strong indication of the presence of an old myocardial infarction.

R Wave.

"R" is the first upward or positive deflection after the P wave and the tallest portion of the QRS complex.

S Wave.

"S" is the first downward or negative deflection after the R wave and represents the final depolarization of the Purkinje fibers.

ST-Segment.

"ST-segment" is the electrical resting period after ventricular depolarization. It represents early repolarization of the left and right ventricles. This interval begins with the end of the QRS complex and ends with the onset of the T wave. Generally, its duration is two to three small squares, 80-120ms, and its isoelectric charge should lie at the same height as the PR interval. This height comparison is important. ST-elevation can be an indication of acute myocardial infarction (MI). Other heart conditions that are expressed in ST abnormalities include acute pericarditis, hyperkalemia, hypercalcemia, pulmonary embolism, ventricular aneurysm, left bundle branch block, Prinzmetal angina, blunt trauma to the heart, Brugada syndrome, and even subarachnoid hemorrhage.

T Wave.

"T Wave" is the process of ventricular repolarization. It should be less than two-thirds of the height of the R wave. T wave morphology changes are seen in conditions such as hyperkalemia, hypercalcemia, endocrine changes, myocarditis, pericarditis, cardiomyopathy, pulmonary embolism, fever, generalized infections, anemia, acid-base disorders, and more.

QT Interval.

"QT" interval represents the total ventricular activity from ventricular depolarization to repolarization. There is no hard and fast "normal" for this period of length, though 400 to 440ms has been suggested. It is measured from the beginning of the QRS complex to the end of the T wave, with slower heart rates having longer QTs.

Prolonged QT intervals indicate a lengthened relative refractory period, a vulnerable period for abnormal electrical mischief. Inside this vulnerable period, critical, life-threatening rhythms may attempt to establish themselves, such as Torsades de Pointe, ventricular tachycardia, or ventricular fibrillation.

U Wave.

"U" waves are small positive deflections that may be seen following the T wave. There is no clear consensus on what they represent; however, delayed repolarization of the tissue known as papillary muscle or Purkinje fibers is often seen when present.

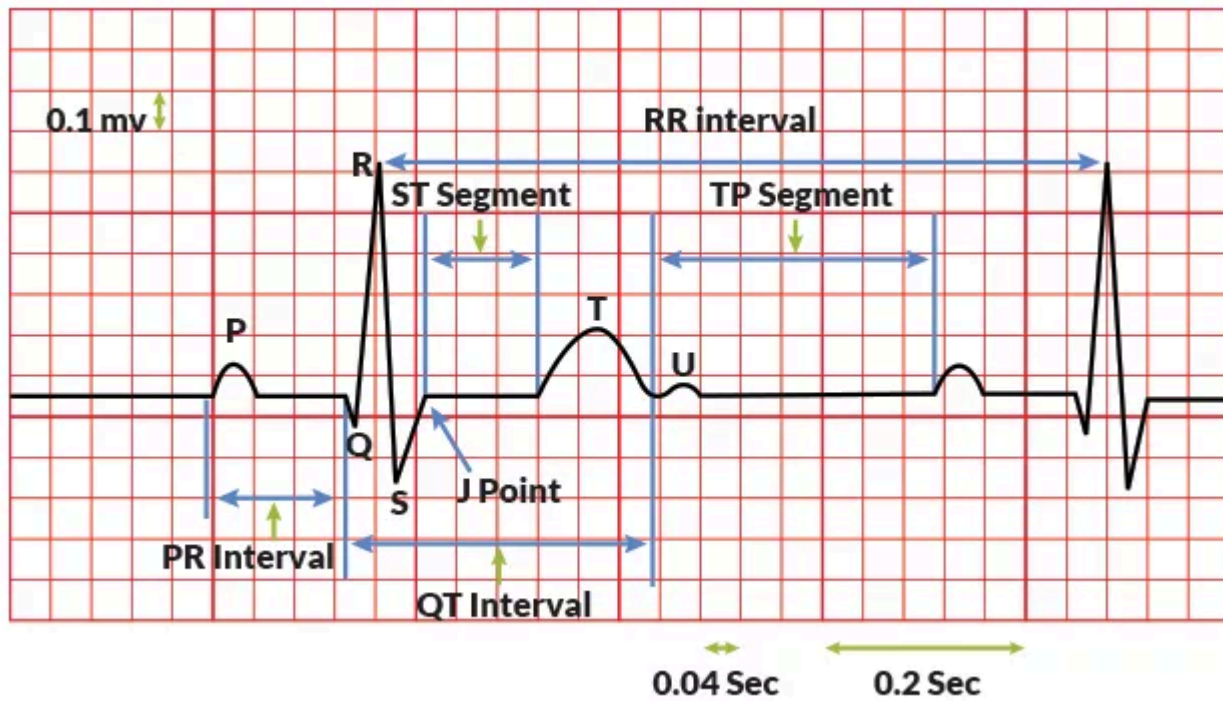
J Wave.

Osborn waves or "J" waves are seen mostly during hypothermia. This wave presents a small deflection at the junction, "J," of the QRS complex and the ST segment.

Epsilon Wave.

Should you happen upon one, the Epsilon wave is not common. If you notice a regularly occurring, small positive deflection buried on the end of the QRS complex – Congratulations! You have stumbled onto an Epsilon wave pattern, which most frequently is associated with inherited cardiomyopathy arrhythmogenic right ventricular dysplasia. First described in 1977, this condition is responsible for 22% of sudden cardiac death (SCD) in athletes and 11% of SCD in young adults (Sharma, 2020).

Image 8:



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Image Source: *Electrocardiography Components* Compliments of Wikimedia Commons

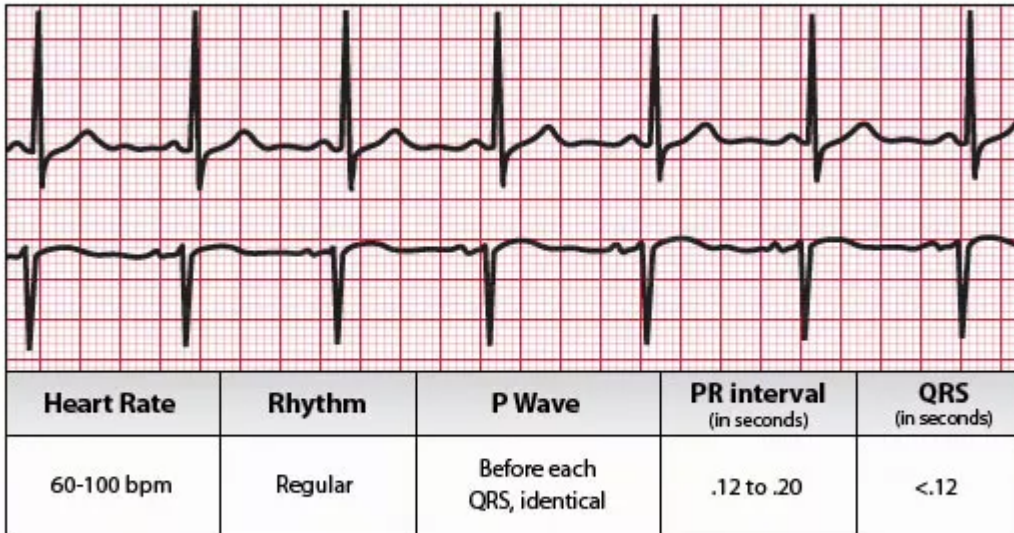
Rate and Rhythm

Utilize a Systematic Approach.

- Is it a Regular rhythm?
- Are there P waves?
- What is the QRS width?
- Does each QRS have a P wave in front?
- What is the Heart Rate?

Image 9:

Normal Sinus Rhythm



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Calculating the Heart Rate

The standard heart monitor paper speed is 25mm (five large squares)/sec. The interval between two beats (R-R) is five large squares. The HR is 60 beats/min.

Methods for calculating the heart rate:

- Rule of 300: Divide 300 by the number of the large squares between two heart beats (R-R), or, if the interval between two beats is one large square, the HR is 300 beats/min, 2 squares → 150, 3 squares → 100, 4 squares → 75, 5 squares → 60, 6 squares → 50 beats/min.

Image 10:



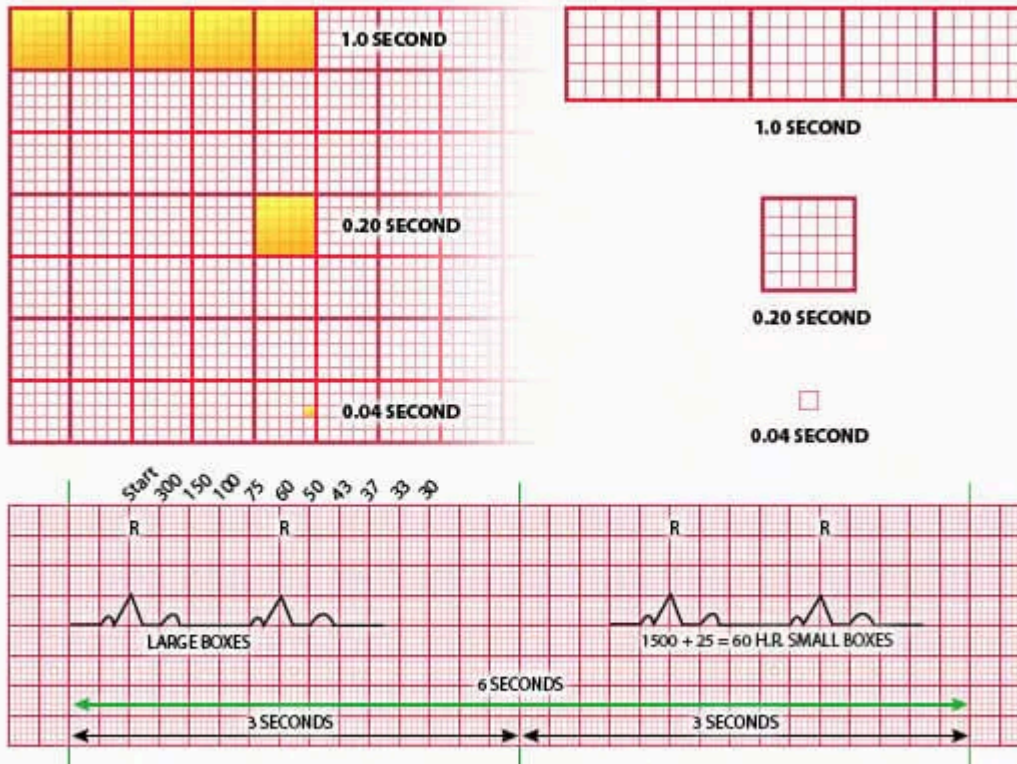
Image Source: Curtesy Wikimedia Commons

The Six-Second Method: Count the number of complete R waves within 6 seconds and multiply that number by 10. This count is the one-minute heart rate. This method can be used when the rhythm is "regular" or "irregular" (Manu, 2018).

- The Three-Second Method: Count the number of complete QRS complexes in three seconds and multiply that by twenty. This count is the one-minute heart rate.

Image 11:

Scale Detail



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Pin it

Analyzing a Rhythm Strip Using the Eight Step Approach

The eight-step system is a good starter system, and you will quickly learn what to look for in any suspect monitor strip.

Step One: Determine the Rate

- To determine the atrial rate, measure the distance between P-P and determine the rate by one of the methods listed earlier.
- What is the ventricular rate?
- To determine the ventricular rate, measure the distance between R-R.
- Note: The rate of a Normal Sinus Rhythm is 60-100 beats per minute

Step Two: Determine the Rhythm

- Is the rhythm regular or irregular?
- To determine if the atrial rate is regular or irregular, measure the distance between two consecutive P-P intervals. Use a point from one P wave to the same point on the next P wave. Then compare this with another P-P interval. If the atrial rate is regular, the P-P interval will measure the same.
- Determine if the ventricular rate is regular or irregular, measure the distance between two consecutive R-R intervals. Use a point from one R wave to the same point on the next R wave. Then compare this with another R-R interval. If the ventricular rate is regular, the R-R interval will measure the same.
- Is the rhythm regular? Regular? Regularly irregular? Irregularly irregular?

Step Three: Evaluate P Waves

- Are P waves present and uniform in appearance?
- Are P waves upright (positive) in Lead II?
- Do P waves appear regularly before each QRS complex?
- Is there more than one P wave before a QRS complex?
- If irregular, is there an associated QRS?

Step Four: Evaluate the P-R interval

- If the P-R interval is less than 0.12 or more than 0.20 seconds, conduction follows an abnormal pathway, or the electrical impulse is delayed at the AV node.
- The normal P-R interval is 0.12 to 0.20 seconds.
- Is the P-R interval consistent?

Step Five: Evaluate the QRS complex

- Do the QRS complexes occur uniformly and look the same throughout the strip?
- If the QRS measures .10 seconds or less, it is considered narrow and is presumed to be supraventricular in origin.
- If the QRS complex is greater than .12 seconds or more, it is considered wide and presumed to be ventricular in origin until proven otherwise.
- The QRS normally measures 0.04 to 0.10 seconds in duration. Determine if they are married to the P waves.

Step Six: Evaluate T Wave

- Are T waves present?
- Are T waves smooth and rounded?
- Do they have a normal amplitude of 0.5 mV or less?
- Is the deflection the same as the preceding QRS?
- Is there a relationship between any ectopy to the T wave?

Step Seven: Evaluate the QT Interval

- Is the duration from 0.36 to 0.44 seconds?

Step Eight: Evaluate other components

- Is the ST-segment elevated? Depressed? Sloping or scooped?
- Are U waves present? Prominent?
- Are there other (funny little beats) FLB's detected?

Image 12:

Normal and Pathological Electrocardiograms



Image Source: Compliments of Wikimedia Commons

Naming the Rhythm

- Origin of the Impulse plus the Cardiac Activity = rhythm name.
- Origin of the impulse. Is it sinus, atrial, junctional, or ventricular?
- Cardiac Activity: Normal (In rhythm), bradycardic (slow), accelerated (faster than normal), or tachycardic (greater than 100/min)?
- For example, sinus bradycardia, sinus tachycardia, junctional, or ventricular tachycardia

Escape Pacemakers

The normal electrical flow through the heart originates in the SA node>AV node>Bundle of His>left and right bundle branches> Purkinje fibers with mechanical cells stimulated along the way. Therefore, the primary pacemaker is the SA node and has an inherent rate of 60-100 beats/minute. The SA node has the highest level of automaticity; however, escape pacemakers exist.

Escape pacemakers are cells that will initiate a heartbeat should the faster normal pace fail to descend along the standard conduction pathway. Escape cells exist in the atrioventricular (AV) junction and the Ventricles.

The AV junction is the AV node and the nonbranching portion of the Bundle of His. The pacemaker cells in the AV junction are located near the nonbranching portion of the Bundle of His.

The AV node should only generate an impulse if the SA node does not function at its normal rate. The AV node fires electrical impulses at a slower rate of 40–60 beats/minute.

Ventricular pacemakers in the bundle branches and the Purkinje network will become the initiating pacemaker if the AV node cannot function. The inherent ventricular rate is 20–40 beats/minute.

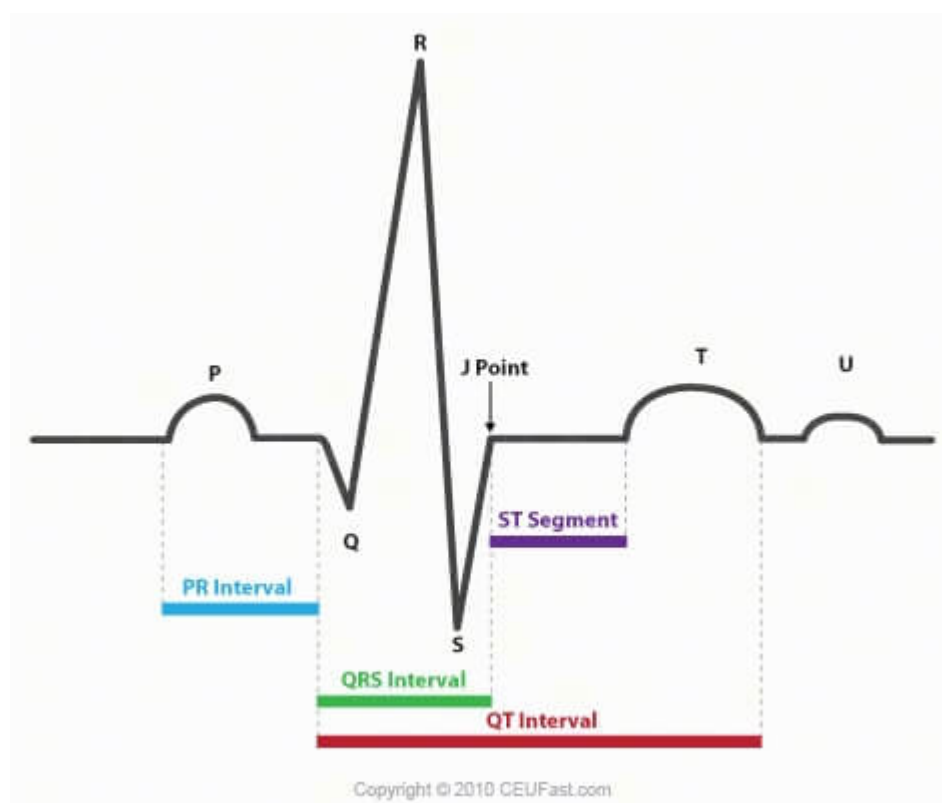
Reentry

This ventricular pacemaker occurs when an electrical impulse is delayed, blocked, or both in one or more portions of the conduction system. In contrast, the impulse is conducted normally through the rest of the conduction system. The results are a delayed impulse entering cardiac cells in which the normally conducted impulse has depolarized. If they have repolarized sufficiently, the delayed impulse will again depolarize the cardiac cells prematurely, thus producing unwanted ectopic beats and rhythms.

ECG Lead Placement

Electrocardiograms are all about depicting the energy use of the heart. To get a standardized view of electrical flow, the “angle” from which the current flow is viewed is essential. A standardized placement pattern of ECG lead wires must be used to get consistent and usable data.

Image 13: Standard Electrocardiogram Waveform Diagram

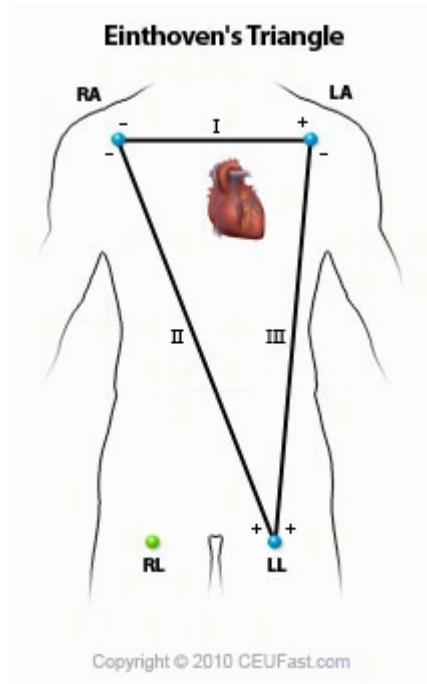




ECG Standard Leads

Let us get this clear and out of the way. The industry standard in healthcare is the 12-lead ECG. That is not what is used for monitoring a client. For ongoing client care, including pre-admission work by emergency responders, a standard three-lead configuration of foam-backed sticky electrodes is used.

Image 14:



We will start with three standard leads, add three augmented leads, then work our way to the last six precordial leads to achieve the standard 12-lead. William Einthoven refined the new principles of galvanic detection into a working ECG in 1902, for which he was later awarded a Nobel Prize in medicine. We still use his “triangle,” now referred to as “standard limb leads,” or “standard leads,” to record the electrophysiologic discharges of the heart in leads I, II, and III (Crouch, 2020).

Lead I:

- Positive electrode is placed just below the left clavicle
- Negative electrode place just below the right clavicle
- Neutral or grounding electrode placed on or close to the left leg (typically placed on the left side, below the left pectoral)
- Provides information about the left lateral wall of the chest

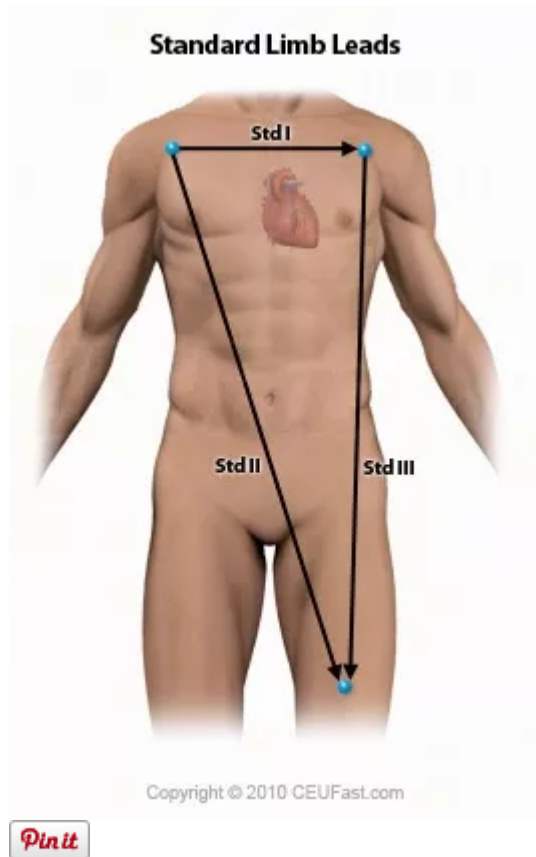
Lead II:

- Positive electrode near the left leg (typically placed below the left pectoral muscle)
- Negative electrode just below the right clavicle
- Neutral or grounding electrode just below the right clavicle
- Provides information about the inferior wall of the heart
- Common in cardiac monitoring because the position of view for this lead is close to the heart's actual conduction pathways

Lead III:

- Positive electrode is at the left pectoral muscle (instead of traditional leg left)
- Negative electrode just below the left clavicle
- Neutral or ground electrode placed just below the right clavicle
- Provides information about the inferior wall of the heart

Image 15: Standard Limb Leads



ECG Augmented Leads

Using the same placement of three-electrode pads and a little fancy math, we can get different views of the electrical activity in the heart. Known as augmented limb leads, unipolar limb leads, or just unipolar leads, an electrocardiogram can create an augmented theoretical null point in the center of Einthoven's triangle allowing a view of the absolute potential in each electrode (My.EKG, 2021).

Sound a little esoteric? Well, it is all about the angle, or vector, from which you are looking at the heart. Think about standing at the end of an extremity, right arm, left arm, or the feet, lying side by side. Now squint up at the heart along that axis. Electric waves moving away from your position will have a positive amplitude on the ECG strip. The waves moving toward you, a negative deflection. At the same time, electrical events doing neither will just be minimized or even merge with the baseline (Bernard.Health, 2021).

The augmented leads are named aVR, aVL, and aVF. "A" for "augmented," "V" for "voltage," then "Right," "Left," and "Foot." The leading "A" can be lower case or capitalized, though the lower case is correct.

Lead aVR:

- The augmented unipolar right arm lead is oriented toward the cavity of the heart.
- Electrical current from the heart is traveling towards the right arm.
- All deflections of the ECG, P, QRS, and T should be negative in this lead.

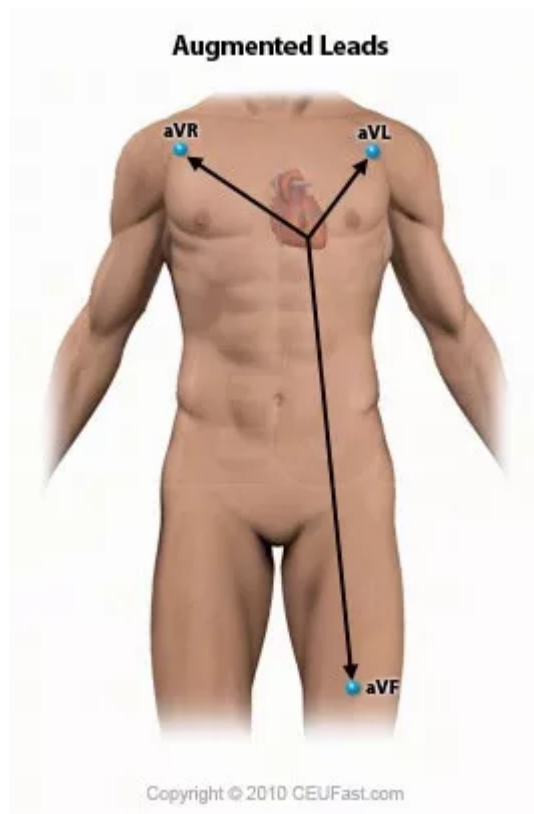
Lead aVL:

- The augmented unipolar left arm lead oriented toward the heart facing the anterolateral aspect of the left ventricle.
- Electrical current from the heart is traveling towards the left arm.

Lead aVF:

- The augmented unipolar left leg lead (feet). It is oriented toward the inferior surface of the heart.
- Electrical current from the heart is traveling toward the feet.

Image 16: Augmented Leads



Standard leads, plus augmented leads make up the first six of a 12-lead ECG. Surprise! These first six leads share one important characteristic, they all view the heart from the frontal plane, as though the client is lying prone and their heart flat on the top of a table we are looking across. Oh, if we could only see the heart's activity from the horizontal plane, as though we were looking straight through our client!

Well, we can. Welcome to the six precordial leads

Precordial Leads

The precordial, or chest leads, view the heart's electrical conduction from the straight face-to-face view. These leads, referred to as "V" leads, are the horizontal plane, unipolar leads.

Lead V1:

- It is placed in the fourth intercostal space just to the right of the sternum
- Anterior view of the right ventricle and right atrium
- Faces the heart cavity
- Provides an electrical view of the right ventricle

- QRS is mostly negative in this lead

Lead V2:

- It is placed in the fourth intercostal space just to the left of the sternum
- Anterior view
- Provides a good view of electrical activity in the right ventricle
- QRS is mostly negative

Lead V3:

- Placed exactly halfway between the positions of lead V2 and V4
- View of the hearts sternocostal surface

Lead V4:

- It is placed in the fifth intercostal space at the mid-clavicular line
- Septal view and left ventricle
- QRS is mostly positive

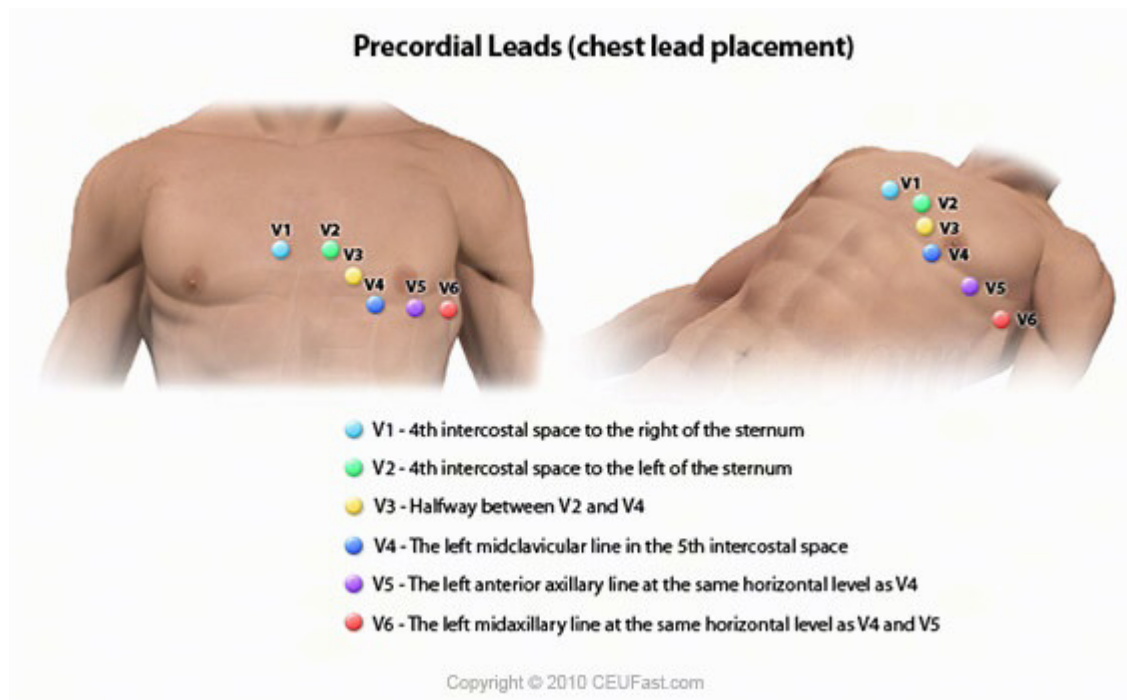
Lead V5:

- It is placed at the same horizontal level as V4 on the anterior axillary line
- Lateral view of the septum and left ventricle
- QRS is mostly positive

Lead V6:

- It is placed at the same horizontal level as V4 and V5 on the mid-axillary line
- Lateral view of the septum and left ventricle
- QRS is mostly positive

Image 17:



There are various uncommon, used for special situations, lead patterns out there. Just so you are aware, here are the most common of uncommon heart leads.

Modified Chest Leads

MCL, or modified chest leads, are different placements of electrodes used to focus on premature beats, bundle branch blocks, or supraventricular rhythms. It is sometimes difficult to discern whether a fast rhythm is supraventricular tachycardia or ventricular tachycardia; a modified lead may come into play.

MCL1:

- It is a variation of V1, where the negative electrode is situated below the left clavicle close to the left shoulder. Positive electrode in the fourth intercostal space to the right of the sternum and ground just below the right clavicle.
- Useful in assessing the anterior wall of the left ventricle and conduction through the ventricles.
- QRS appears mostly as negative deflections.
- This lead is useful in assessing the width of the QRS complex to differentiate supraventricular tachycardia (SVT) from ventricular tachycardia (VT).

MCL6:

- This variation is a deviation of chest lead V6.
- The negative electrode is placed just below the left clavicle. Positive electrode is placed in the fifth intercostal space at the left midaxillary line (like lead V6) while the ground is placed below the right shoulder.
- This lead may be used as an alternative to MCL1 for the same purposes and views the low lateral wall of the left ventricle while monitoring ventricular conduction changes.

12 Lead ECG Results

An ECG strip is the single lead readout common to client monitors. A 12-lead sheet is the standard of care for diagnostic work, as it shows the same time increment view from how many viewpoints? Correct, twelve different planes of view. What may be hidden in one view may show clearly from another point of view.

Quick trick. Although different manufacturers of ECG machines produce various-looking ECG sheets, a standard print format is employed.

Lead I	Lead aVR	Lead V1	Lead V4
Lead II	Lead aVL	Lead V2	Lead V5
Lead III	Lead aVF	Lead V3	Lead V6

Long Length Focus Lead, either designated by an operator or picked by a machine.

This trick allows you always to know which lead you are looking at on any given ECG sheet.

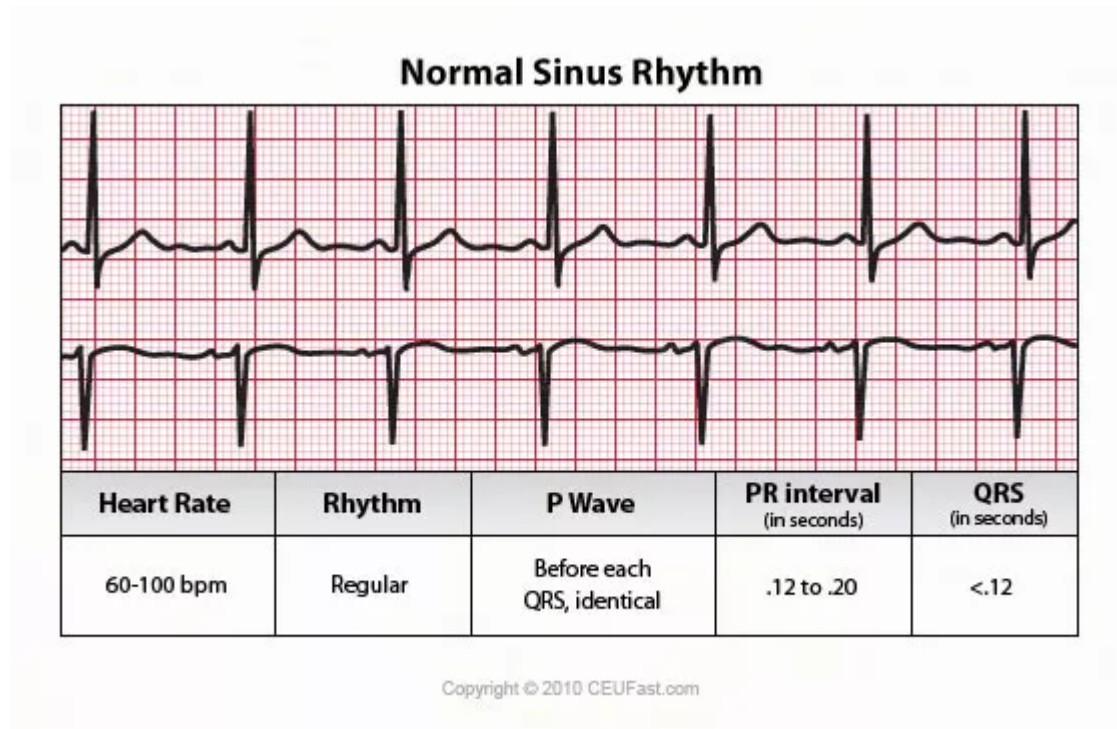
Got Rhythm?

Normal Sinus Rhythm

We consider normal a regular contracting heartbeat stemming from an electrical stimulus originating from the sinoatrial node (SA) located in the upper right atrium of our heart. An

even, regular beat, at a rate of 60 to 100 electrical waves each minute flowing through the heart myocardium's typical depolarization channels, is called a normal sinus rhythm (NSR). A normal heart rate is responsive to bodily demands such as exercise or exertion, speeding or slowing the heart rate to compensate for circulatory body demands (Sauer, 2020).

Image 18:



Sinus Bradycardia

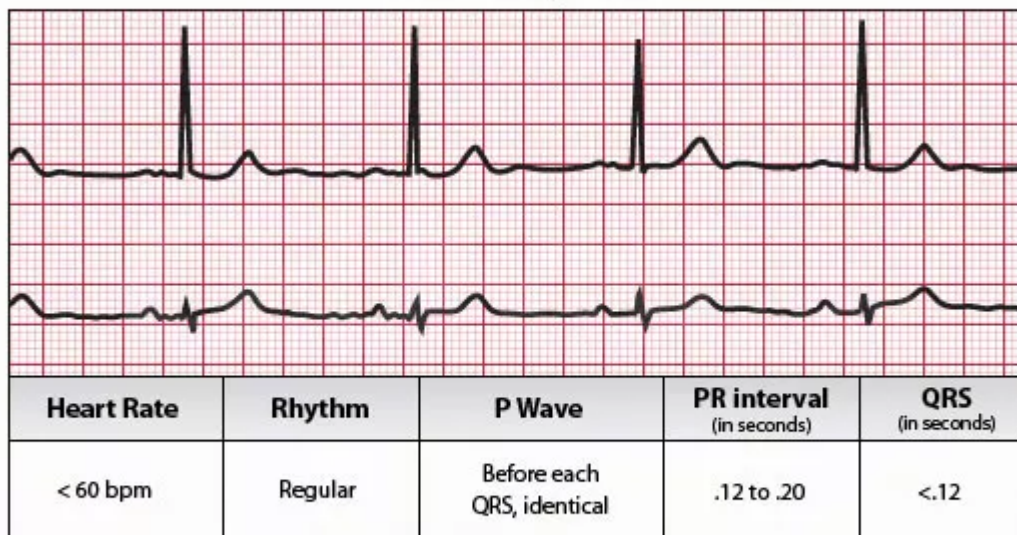
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Sinus bradycardia is a regular rhythm originating from the SA node that is slower than 60 beats per minute. In sinus bradycardia, the P vector on the ECG will be consistent with a SA node origin.

Just as a reminder, a normal SA-produced P wave will show right atrial depolarization followed rapidly by left atrial depolarization. A distinctive high to low, top to bottom, atrial polarization gives rise to the stereotypical upright P inflection in leads I, II, and aVL, with a negative P deflection in the aVR lead (Homoud, 2021)

Image 19:

Sinus Bradycardia



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The slower conduction of Sinus Bradycardia may be due to a normal response to sleep or deep breathing in a well-conditioned athlete. Abnormal drops in rate could be caused by diminished blood flow to the S-A node, vagal stimulation, hypothyroidism, increased intracranial pressure, or pharmacologic agents, such as digoxin and propranolol quinidine, or procainamide.

Hints that slow heart rate should be considered include dizziness, fatigue, syncope. Sinus bradycardia may be asymptomatic.

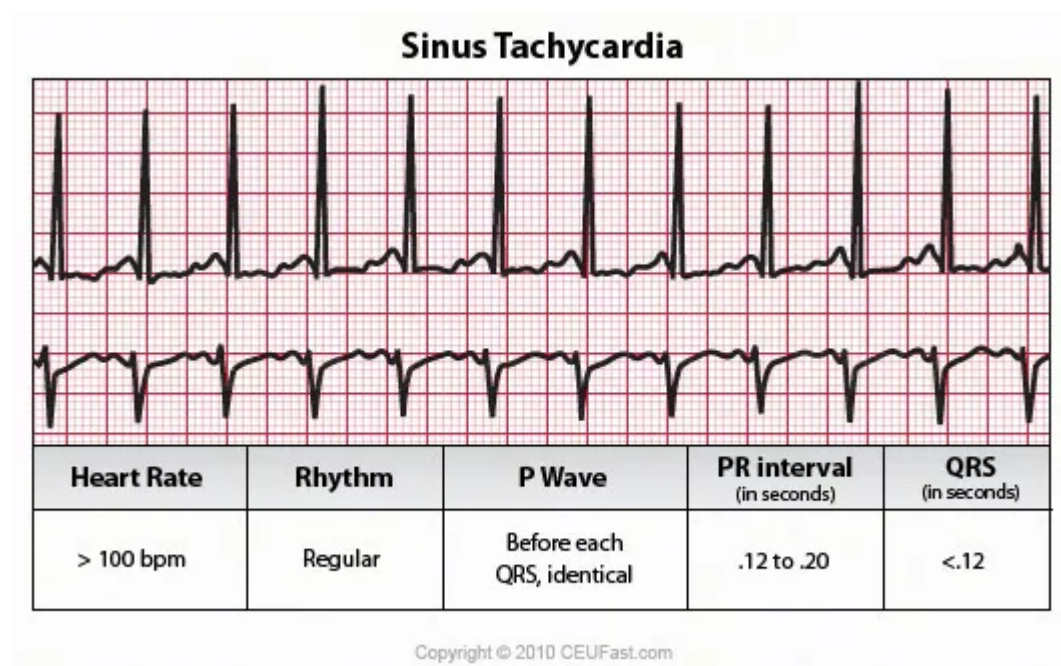
A 12-lead ECG or a wearable heart monitor is considered diagnostic of sinus bradycardia.

Sinus Tachycardia

Video licensed from **3D4MEDICAL**
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Too fast of a heart rate while at rest creates problems with heart filling. The large chambers of the heart, the ventricles, require almost a full second to fill with blood in anticipation of pushing it out on its voyage through the body. A contraction rate of greater than 100 beats per minute with the electrical stimulus arising from the SA node and the presence of evenly paired P with QRS waves qualifies as sinus tachycardia (Felman, 2020).

Image 20:





There are various types of tachycardia. Sinus tachycardia is a regular cardiac rhythm that meets normal sinus rhythm standards, apart from being too fast. Greater than 100 bpm (beats per minute) in a resting adult, faster than 150 bpm in infants to around six years old.

Do not be misled. Too quick a heart rate, even with a normal conduction mechanism, can be problematic. Strokes, heart failure, and the risk of heart attack from increased cardiac demands can accompany sinus tachycardia.

Sinus tachycardia may result from stress, exercise, pain, fever, pump failure, hyperthyroidism, caffeine, nitrates, atropine, epinephrine, and isoproterenol, nicotine, electrolyte imbalances, fatigue, blood loss, and other situations which places stress on the body.

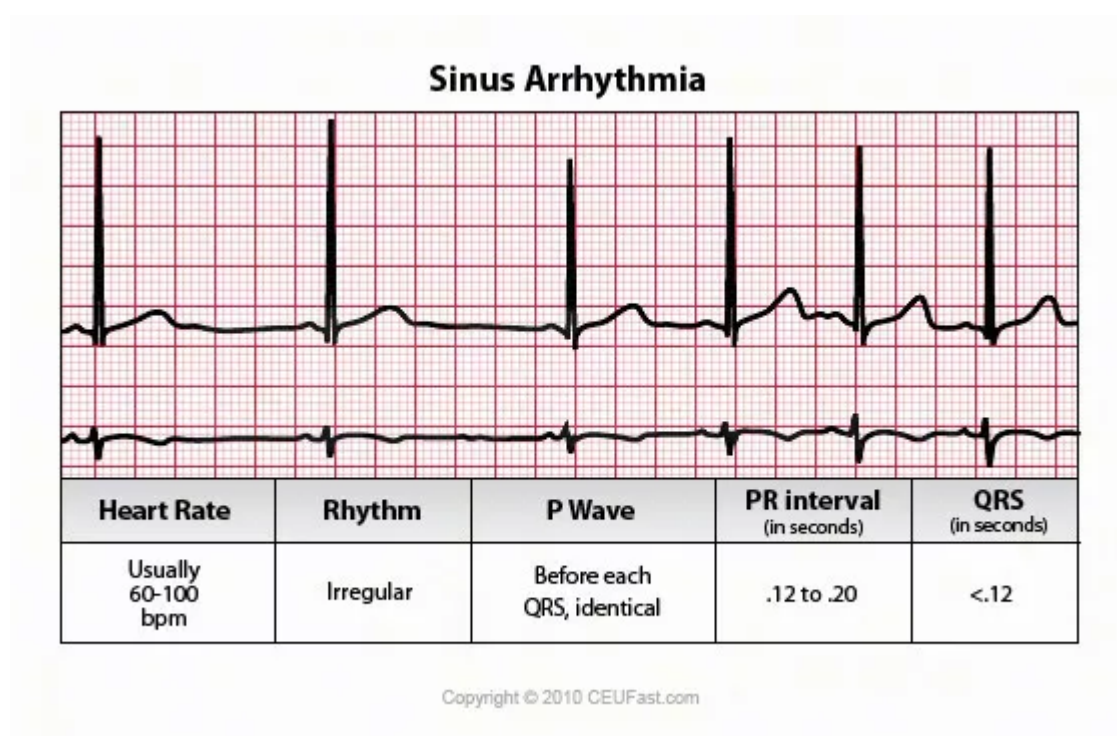
Sinus Arrhythmia

Have you ever heard that there is a healthy arrhythmia?

Well, this is it. When your cardiac system is vigorous enough that, guided by the vagal nerve, there is a slight slowing in the resting heart rate after a big breath followed by a slight speeding up during exhalation. Now that is a healthy sinus arrhythmia!

Sinus arrhythmia is most common among children and frequent among adults. It is not pathogenic and requires no treatment.

Image 21:



If there is any danger from sinus arrhythmia, it is misdiagnosed as a more serious arrhythmia. The rate is usually 60–100 beats/min but may be transiently faster or slower. Should the rate be problematic, consider a rate-specific treatment. The rhythm itself is not an issue.

A distinct P wave will be associated with each QRS complex. Be sure to look at your client's overall condition and, if in doubt, a 12-lead ECG should clarify the situation.

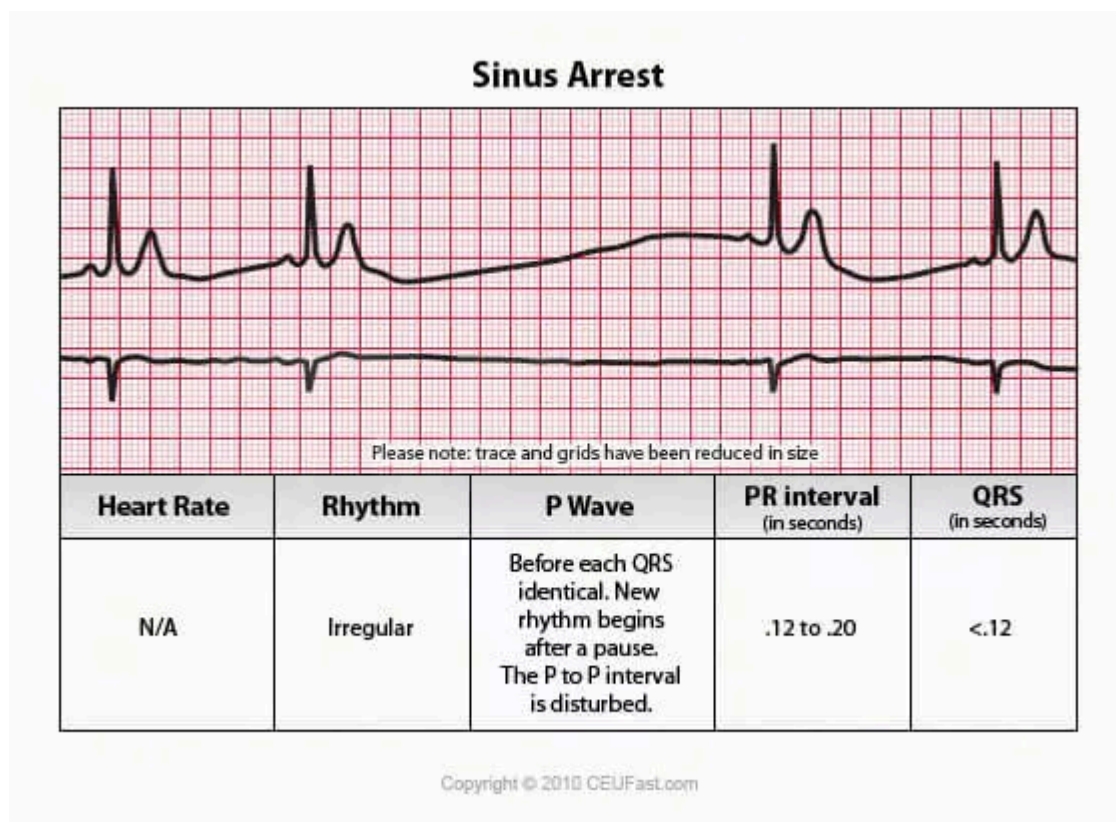
Sinus Arrest or Sinus Pause

A sinus pause is not your friendly neighborhood sinus arrhythmia.

Sinus arrest or sinus pause is an indication of a dysfunction in the SA node. This arrest or pause leads to the dropping or pausing of electrical conduction in the normal sense. The electrical depolarization cycle is initiated yet somehow blocked before the impulse can leave the SA node. These missed beats may cause little to significant symptoms yet should never be taken lightly. Whatever mechanism may be blocking a beat here and there could easily manifest into a significant and potentially deadly sinus heart block.

Interestingly our older clients are where sinus arrest is most often seen. This sinus arrest has led to the hypothesis that it is due to accumulative deterioration or damage to the SA node, which may hold, though it is difficult to do a blind study. When found in younger adults or children, sinus arrest can often be directly correlated with a specific cardiac event or a severe electrolyte imbalance. Possible symptoms accompanying sinus arrest include feeling an occasional missed beat, fatigue, dizziness, or angina.

Image 22:



Usually, the rate associated with this is 60–100 beats/min but may be faster or slower.

The rhythm on ECG will be irregular. The SA node initiates an impulse, but that impulse is blocked before leaving the node itself. This block results in an absent or dropped PQRST complex.

In sinus arrest, the pause is not a multiple of other P–P intervals. Treatment may include Atropine or a pacemaker if symptomatic.

Sinus Exit Block (Sinoatrial Block)

Sinus exit blocks are when a depolarization wave leaves the SA node yet fails to be conducted to the atria and therefore fails to stimulate the ventricles. Delays of the SA depolarization wave

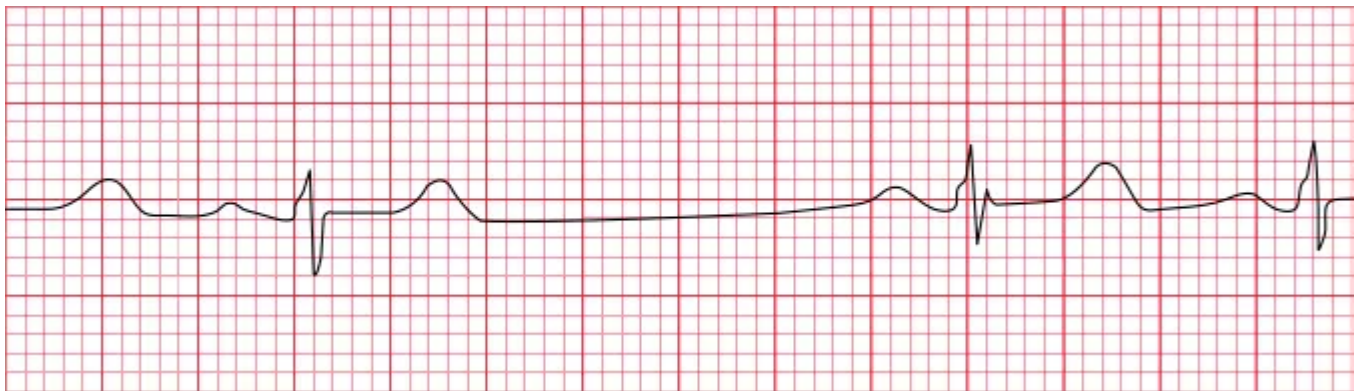
before they can kick start the muscular compression wave of the heartbeat also occur as atrioventricular (AV) blocks. There are three main types. We will get to the three major AV blocks a little later.

Failure of SA node conduction is a crisis. It may lead to the creation of an abnormal P wave and a following normal-looking QRS complex. Or a complete blocking of P wave generation leading to a condition known as sinus arrest, where secondary escape pacemaker cells are called upon to replace the missing SA conduction impulse.

Sinus arrest leads to symptoms such as bradycardia (remember, the firing of escape beats is always slower than the genuine impulse generator), dizziness, syncope, or palpitations.

Image 23:

Sinus Exit Block – Sinoatrial Block



Pin it

Heart Rate	Rhythm	P Wave	PR Interval	QRS
60–100	Irregular	P before every QRS	12–20 sec	<10

Image Source: Adapted from Wikimedia Commons

Generally, this rhythm will run around 60–100 beats/min but may be slightly faster or slower. The pattern should show as irregular as the SA node initiates each impulse, yet some impulses are blocked before leaving the node itself. This results in the occasional dropped PQRS complex. Note that the pause in these absences is the same as the distance between two P–P intervals of the underlying rhythm. Each PQRS should be uniform and upright in appearance in leads I, II, and aVL.

Premature Atrial Complexes (PACs)

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Expanding out into the heart atrium, we begin to see some common arrhythmias that present challenges to us as health professionals. Premature beats of any type are problematic, and clients who report the feeling of skipped heartbeats need to be assessed for premature heartbeats.

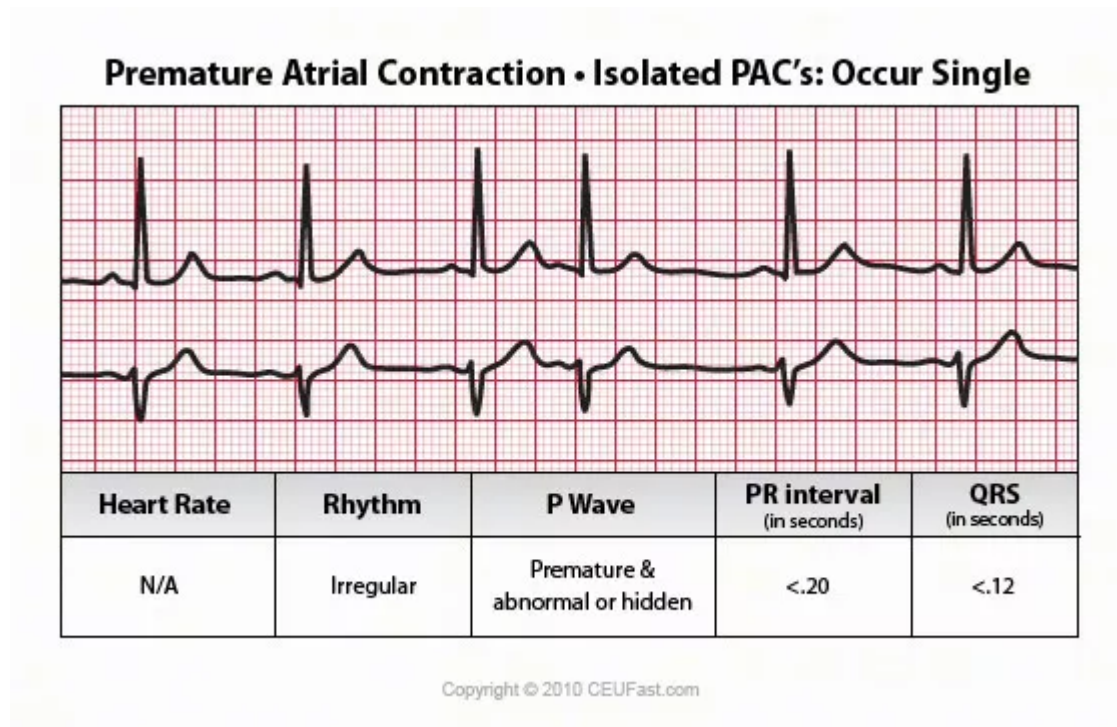
Let us start in the atrium, however, outside of the SA node, which is the official timekeeper and pacesetter of our heart. Premature atrial contractions (PACs) come from early depolarization somewhere in the atrium outside of the SA node leading to an interruption or replacement of the SA node-derived beat. Conduction initiation comes from areas of irritation, inflammation, or spots just firing off early for no discernable reason. These early depolarizations take off like wildfire and initiate an untimely contraction that flows through the atrium and then down into the ventricles, creating an early beat with a malformed P wave, though relatively normal QRS complex.

Ask about the client's clinical history. Of particular interest are any previous cardiac disorders or structural heart disease. Reviewing medication for any proarrhythmic drug use is important and for the presence of stress, fatigue, caffeine, alcohol, tobacco, and such conditions as hypertension or hyperthyroidism. An electrolyte study focusing on sodium or magnesium levels is also handy.

Symptoms of dizziness, syncope, and of course, the perception of a skipped heartbeat may accompany PACs. Be aware that when a PAC occurs, the early triggering of the ventricles will mean a contraction carrying less than a full volume of blood, so the feeling of skipping is an accurate perception by the client.

Keep in mind that premature beats are identified by their site of origin (atrial, junctional, and ventricular). PACs occur when an irritable site within the atria discharges before the next SA node is due to discharge.

Image 24:



PACs with a wide complex are referred to as aberrantly conducted PACs, acknowledging the conduction wave's eccentric route. PACs and other early beats may occur in pairs (couplet), bursts (premature atrial tachycardia) (PAT), or even every other beat (bigeminy).

In the absence of other pathology, the heart rate tends to stay normal, from 60–100 when there are only occasional premature atrial contractions. Be aware that the presence of a sinus tachycardia may serve to promote more frequent PACs, depending on the overall client condition prompting a fast heart rate.

The rhythm will be irregular due to the early PACs. The presence of the early atrial impulse will throw the rhythm into the irregular category.

The P wave of the early beat differs from SA node P waves and is early, premature. The aberrant P waves may be flattened or notched. They may even be lost in the preceding T wave, which is where a 12-lead ECG shines as it allows detection by changing up the vector of observation.

The PR interval, sometimes abbreviated to PRI, may vary from .12–.20 depending on how near the pacemaker site is to the SA node. A longer PRI means the initiation site is higher up in the atrium, e.g., closer to the SA node, while a shorter PRI indicates the pacemaker site is nearer the AV node.

The QRS complex will usually <.10 but may be prolonged.

Supraventricular Tachycardia

Tachycardia means fast. Supraventricular means the origin of the impulses is from above the cardiac ventricles. These high and fast aberrant rhythms tend to be clustered together due to their shared diagnostics.

Supraventricular tachycardia (SVT) is a group of regular fast rhythms characterized by narrow QRS complexes and high heart rates. Please note that while atrial fibrillation and atrial flutter share a high conduction origin point and fast rate, they are typically irregular rhythms and will be discussed later.

Women are twice as likely to experience SVT as men at any age. In general, about 2.25 Americans out of 1000 fit the diagnosis of SVT (Hahn, 2020).

Be aware that SVT symptoms are often misdiagnosed as a panic attack. So, when in doubt, slide on some ECG patches and get to the truth of the matter. An unusual accompanying symptom of SVT is drumroll, please, polyuria.

SVT can be a spontaneous occurrence with no known trigger. More commonly, mechanisms such as too many energy drinks, cocaine, sepsis, dehydration, or increased intracardiac pressures contribute to this arrhythmia. Cardiac structural changes may also prompt an SVT response, such as congestive heart failure, myocardial infarction, pulmonary embolus, or valvular regurgitation or stenosis.

Vagal maneuvers up to and including carotid massage, Valsalva maneuver, even cold immersion have been used as a treatment to break the SVT cycle. Pharmacologic agents that tone down AV node sensitivity and help to end the SVT reentry pattern include adenosine, verapamil, esmolol, and diltiazem (Hahn, 2020).

Definition: Frog Sign

An interesting diagnostic indicator referred to as the “frog sign” may occur during atrioventricular nodal reentry tachycardia (AVNRT). AVNRT is where the atria fire against a closed tricuspid valve causing strong rebound pressure waves referred to as “cannon waves,” which pound retrograde into the jugular vein.

This gives the appearance of a billowing neck, hence the name “frog sign.”

While this is not an ECG wave, diagnosis is a compilation of available data, and yes, cannon waves can be noted in baseline polarization deviations of a 12-lead.

Cannon waves, and to a lesser extent, the frog sign may also occur in ventricular tachycardia. However, the ECG would demonstrate a wide QRS complex which lowers the back pressure and diminishes the frog sign.

(Hahn, 2020)

Regular SVT

Supraventricular tachycardias come in two basic types, regular or irregular. SVT is referring to the interval of QRS frequency. The fast, regular rhythms originating in the atrium of the heart are AVNRT, AVRT, and Junctional tachycardia.

Of the regular SVTs, Atrioventricular Nodal Reentrant Tachycardia (AVNRT) is the most common in 60% of cases diagnosed with an SVT. Female clients compose 70% of all AVNRT. Atrioventricular Reentry Tachycardia (AVRT) is a condition most common to young people and is the next most frequent of the SVTs. Typically, both SVTs occur due to circular electrical conduction between the atria and the ventricles. In AVNRT, two separate, parallel conduction pathways present in the atrioventricular node (AV node). A slow conduction pathway and a fast

one. When two pathways occur, there is a chance of a reciprocating reentry stimulus to occur during cardiac stress. In AVRT, the AV node interplays with an accessory pathway which feeds it an untimely depolarization signal. This interplay establishes a circular trigger keeping one depolarization working its way around and around through the AV node like a hula hoop of cardiac constriction.

Definition: Accessory Pathway

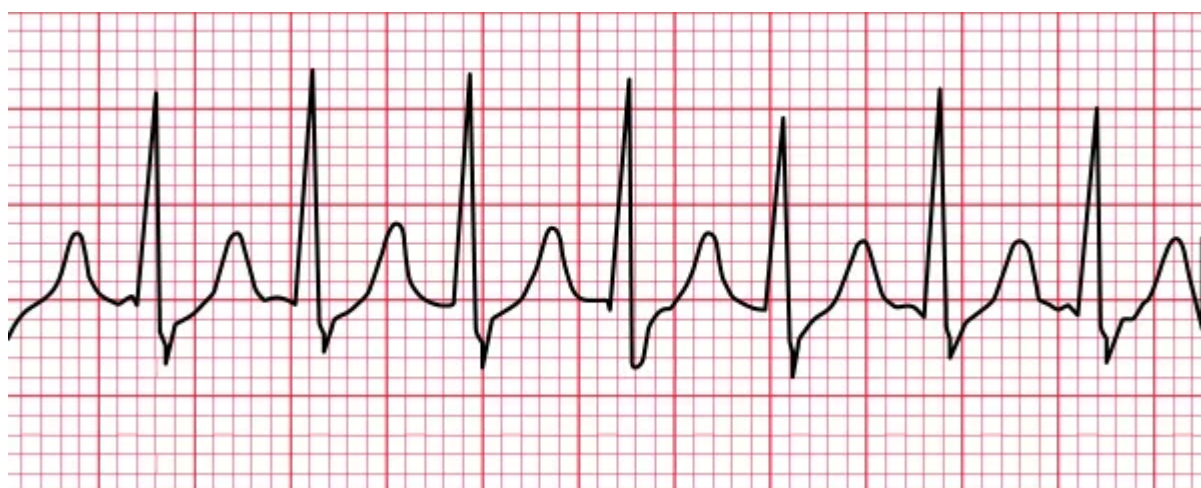
An accessory pathway is an abnormal electrical connection between the atrium and ventricle that is usually congenital. The direction of electrical impulse conduction may be anterograde (toward the ventricles), retrograde (toward the atria), or both.

Accessory pathways may lead to AVRT because of a reentry circuit with the AV node or other tachyarrhythmias where abnormal atrial impulses are shunted through the pathway (e.g., pre-excited atrial fibrillation).

(Amboss, 2021)

Image 25:

Atrioventricular Nodal Reentrant Tachycardia



[Pin it](#)

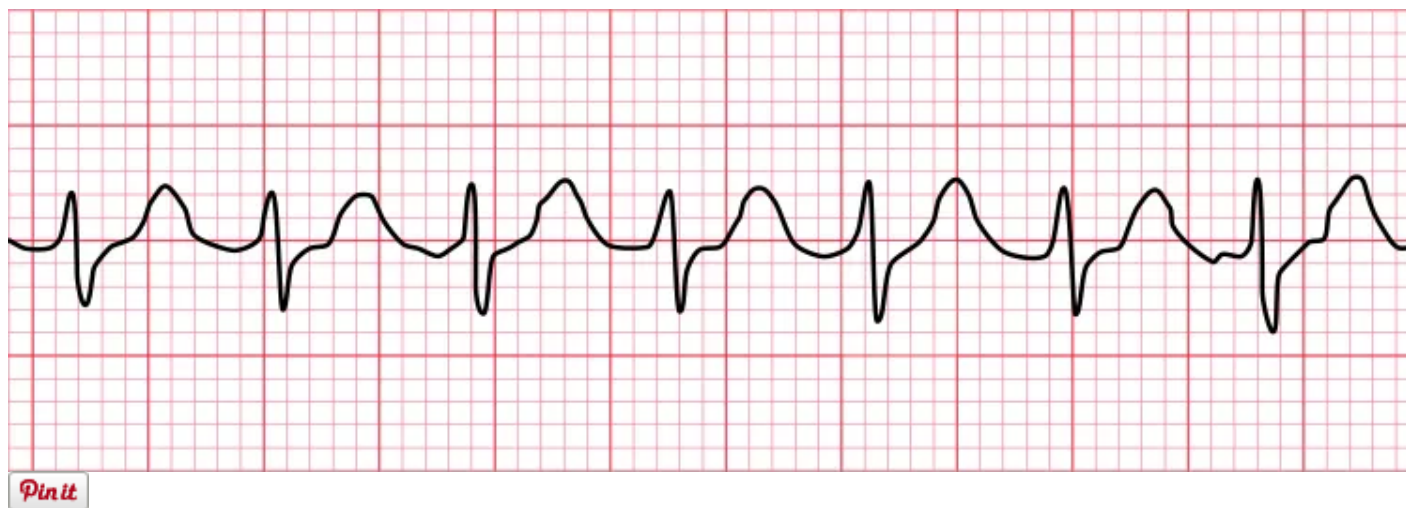
Heart Rate	Rhythm	P Wave	PR Interval	QRS
120-250	Regular	Absent hidden by preceding QRS	Hard to see .12-.20	Narrow <.12

Image Source: Adapted from Wikimedia Commons

Atrioventricular Reentry Tachycardias are the second most common of the regular SVT's and share with the SVTs what is often referred to as a pre-excitation syndrome. That is regular, rhythmic, rapid-firing stimuli originating from above the ventricles and AV nodes.

Image 26:

Atrioventricular Reentry Tachycardia

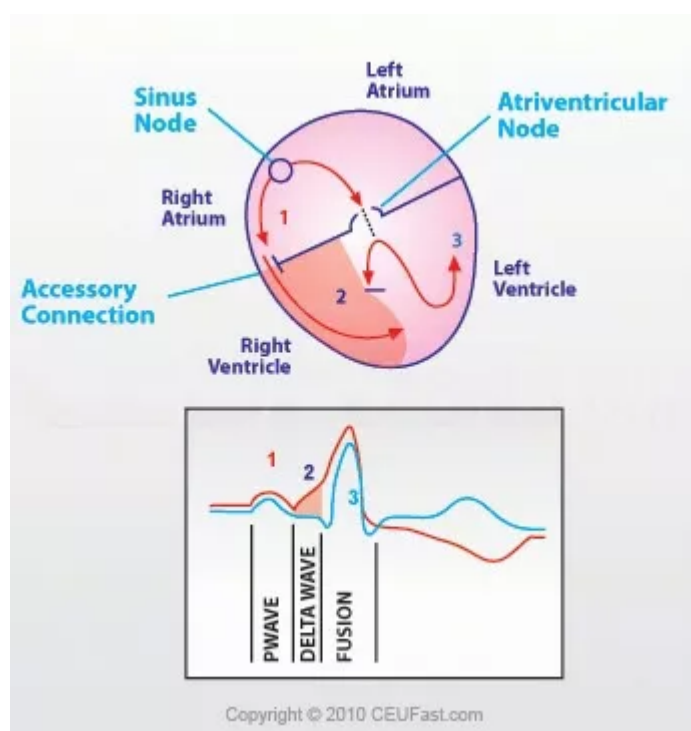


Heart Rate	Rhythm	P Wave	PR Interval	QRS
150-250	Regular	Non-sinus P waves occur after QRS Retrograde II, III, AVF	Hard to see .12-.20	Narrow <.12

Image Source: Adapted from Wikimedia Commons

Not to add confusion. However, it does make a difference in which direction the circular conduction pathway rotates. The most common circular track is an antegrade conduction pathway through the AV node is known as Orthodromic. Orthodromic has the following characteristics. RP interval < (less than) the PR interval, or RP interval > (greater than) the PR interval in a slowly conducting accessory pathway. Also, look for retrograde P waves (leads I, II, III, aVF, V1). In a slow pattern, less than 100 bpm, a delta wave might be seen. Yet only with normal sinus rhythm rate, not with tachycardia. The above image is that of the more common Orthodromic AVRT.

Image 27: Electrical Conduction Chain of Events





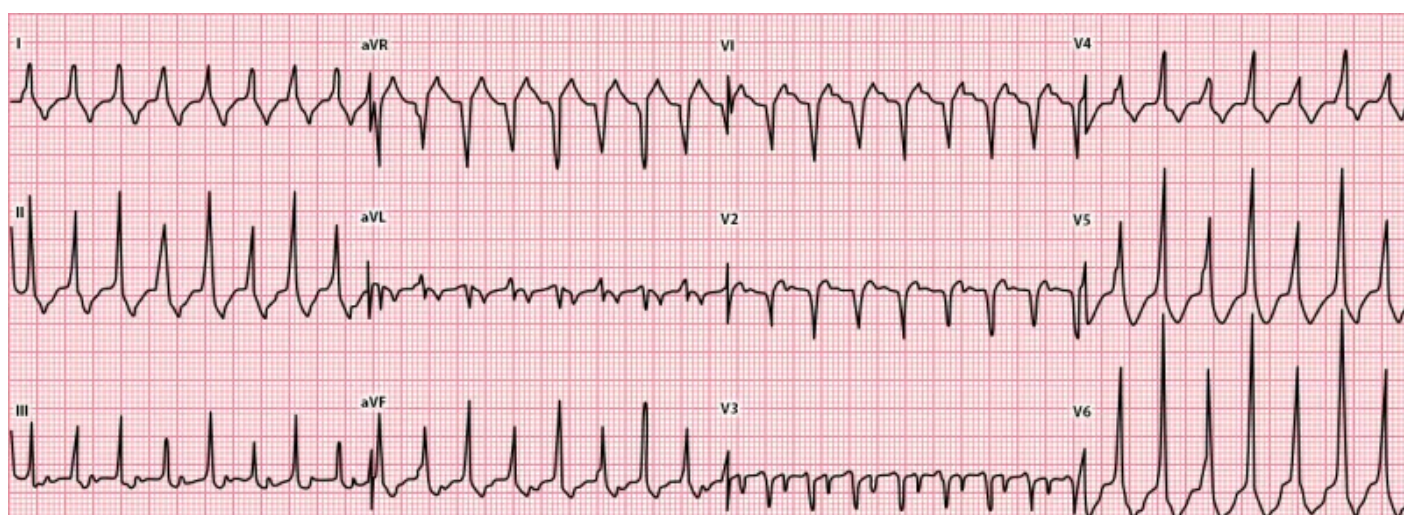
Retrograde conduction through the AV node is referred to as Antidromic AVRT.

That is, the aberrant rapid conduction flows down from the atrium through the accessory pathway. At that point, it regurgitates back up into the atrium from the depolarizing ventricles flowing in the wrong direction through the AV node. Then the atrium fires and sends another aberrant depolarization down through the accessory pathway to the ventricles, repeating the aberrant conduction. Antidromic AVRT is a much less common conduction issue and possesses a different set of characteristics, such as a short RP interval (< 100 msec). A regular, wide QRS complex (≥ 120 msec). Delta waves, the up slurring of the R wave, can be seen at rates equivalent to normal sinus rhythm and tachycardia (Patti, 2020).

Let us look at Antidromic AVRT on a 12-Lead.

Image 28:

Antidromic Atrioventricular Reentry Tachycardia



Heart Rate	Rhythm	P Wave	PR Interval	QRS
150–250	Regular	Retrograde and before QRS	Long	Wide >0.12

Image Source: Adapted from Crimelysis

Antidromic AVRT is about 5% of the cases of AVRT. Notice the delta waves easily visible in V₄, V₅, V₆. The P is crushing into the QR in leads I and II.

A quick word about another antidromic reentry pathway is Wolff–Parkinson–White (WPW) syndrome. WPW is another example of accessory pathway reentry rhythm gone wild. It has little to do with the atrium of the heart, so that we will be discussing it later. Occasionally, well, rarely really, the culprit accessory pathway in WPW acts so that the AV node is the retrograde pathway. These typically present with a wide QRS complex, regular, and extremely rapid tachycardia (Patti, 2020).

Junctional Rhythms

Junctional rhythms are protective impulses from the AV node, the junction, whenever the heart's main rhythm generator, the SA node, fails. For the sake of convenience, fast junctional

rhythms are often listed among SVTs. So, we will look at arrhythmias originating with the junction here.

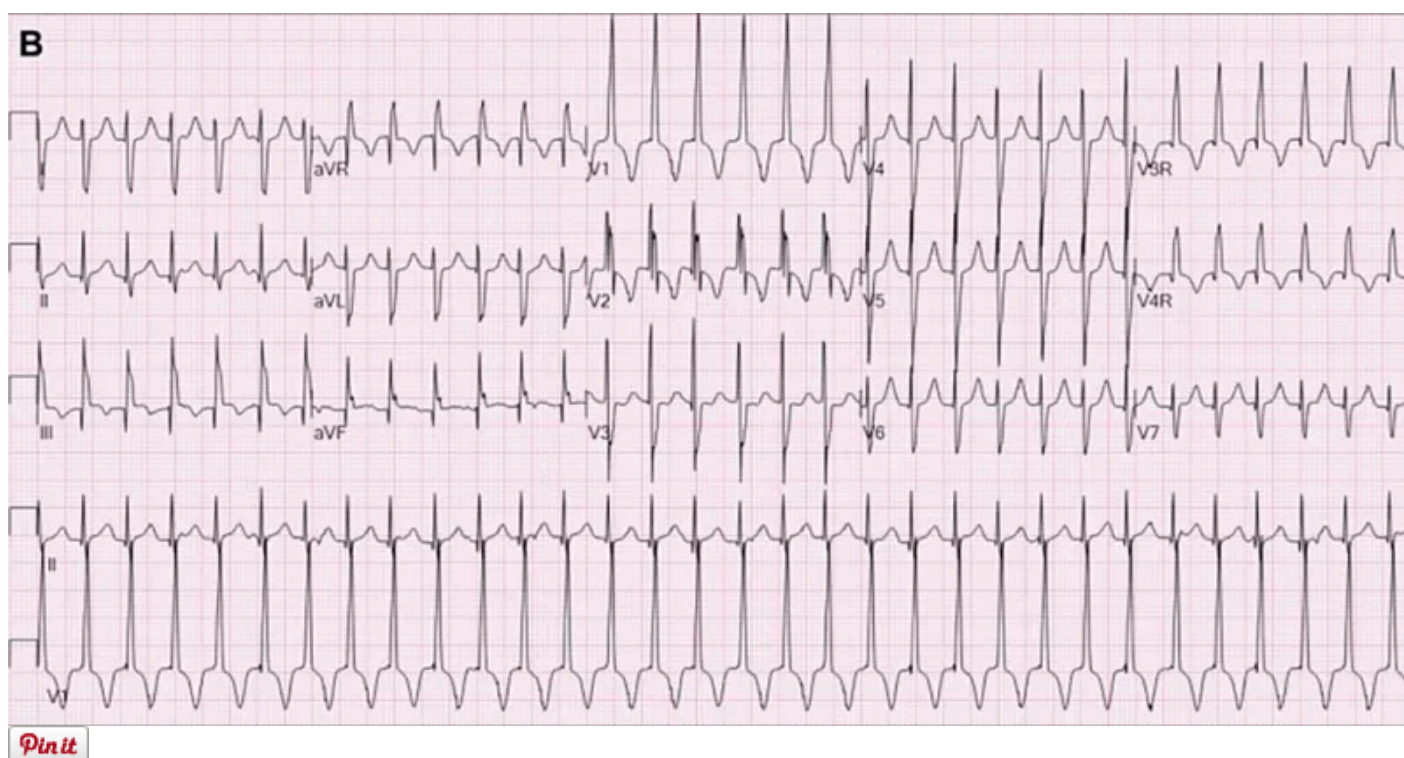
A quick review of the cardiac conduction system: The SA node in the heart's right atrium is the main cardiac pacemaker, generating an electrical burst 60–100 times per minute. This depolarizing impulse travels to both atria through the internodal pathways, creating a spreading wave of muscle contraction as they pass through the atrial myocardium, heart muscle. Atrial contraction occurs simultaneously and pushes blood into the heart ventricles. The electrical impulse keeps moving along a set conduction pathway down into the AV node, conveniently located between the atria and the ventricles. The AV node sends the impulse through the bundle of His to the right and left cardiac ventricles. To be more specific, the bundle of His sheds the depolarization wave onto the Purkinje fibers that thread through the ventricular myocardium's insides, causing the strong ventricular walls to contract and push blood through the body.

The AV node is able, on need, to serve as an emergency pacemaker should the SA node fail. The inherent rate of the AV node junction on its own is 40–60 bpm, significantly slower than the SA node.

Circumstances may arise when the junction, the AV node, accelerates faster than the primary SA pacemaker. When that happens, junctional tachycardia follows as the heart muscle follows the quickest depolarization cycle present.

Image 29:

Junctional Tachycardia



Heart Rate	Rhythm	P Wave	PR Interval	QRS
>200	Regular	P may be immediately before, follow, or be buried in QRS. May be inverted or retrograde	Short	Narrow <0.12

Image Source: Adapted from Wikimedia Commons

Note the P wave immediately before the Q as seen on lead II. This P wave is common for all junctional rhythms as the depolarization pacing point are in the AV node, and the depolarization wave must travel upwards into the atria simultaneously, or the closest thing to concurrent, as the same aberrant firing wave heads to the His bundle and ventricles (Knapp, 2020).

Junctional Rhythm Types

Rhythms sourced from the AV node are characterized by rate.

- Junctional bradycardia rate is <40 bpm.
- Junctional rhythm runs 40–60 bpm.
- Accelerated junctional rhythm runs from 60–100 bpm.
- Junctional tachycardia > 100 bpm.

Junctional rhythms will have a regular RR interval, and as a signature sign, one of the following P wave variations:

Absent P waves

A sign that the AV node is sending depolarization impulses simultaneously to the atria and ventricles

Inverted P waves

When the AV depolarization reaches the atria before the ventricles

Post QRS P waves

When the AV depolarization reaches the ventricles first

Atrial Tachycardias

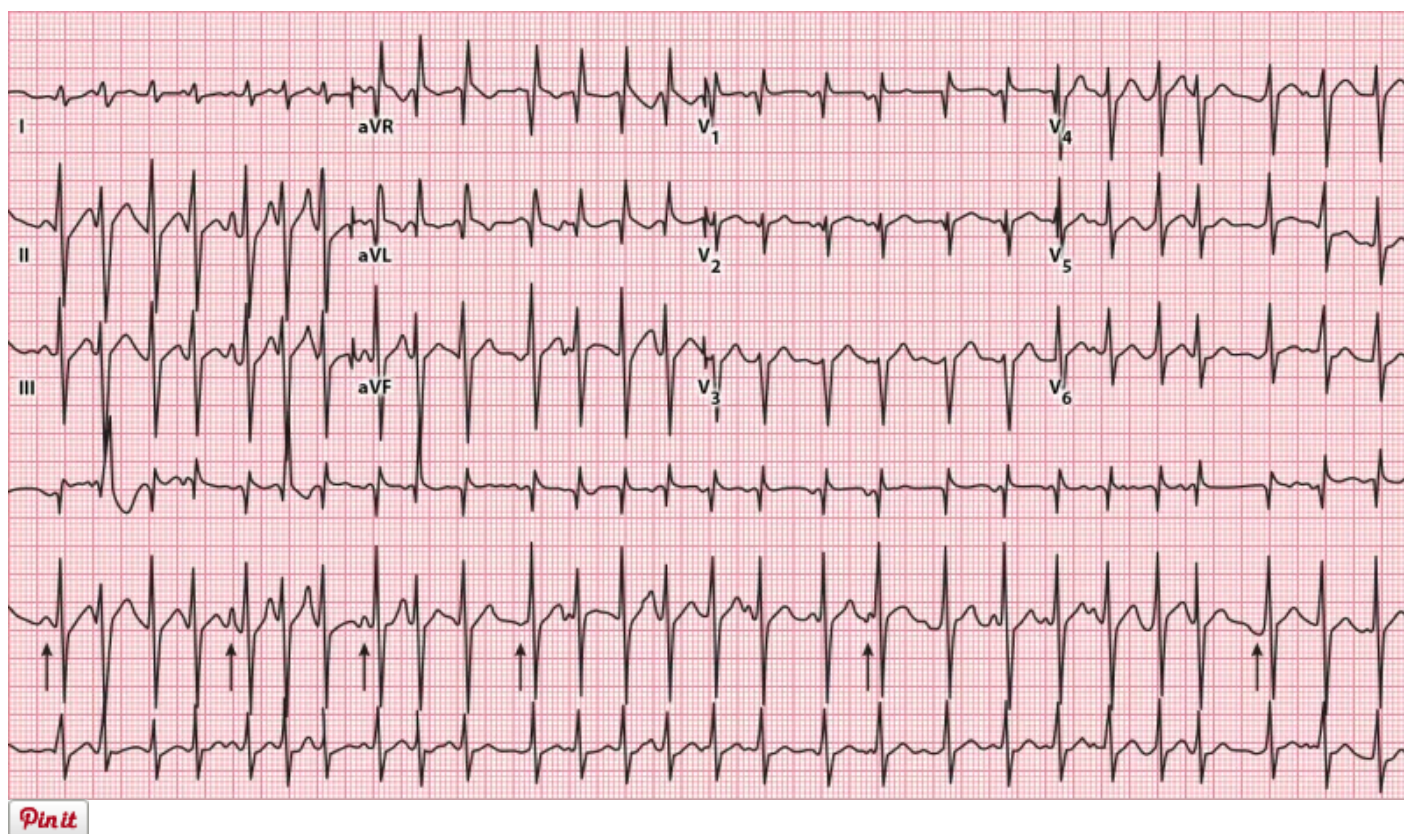
Before we leave the top of the heart, we need to discuss a few fine fast arrhythmias that can lead to a lot of mischief, the atrial tachycardias. This trio of aberrancies is often excluded from the rest of the SVT's due to their irregular rate and rhythm. They are, nevertheless, supraventricular tachycardias, even though a goodly percentage of cardiologists harumph at that designation. We talked about the fast and irregular among the SVTs, Atrial Fibrillation (AFib), Atrial Flutter, and Multifocal Atrial Tachycardia (MAT).

Multifocal Atrial Tachycardia

The name says it all. Multifocal, yes. There must be at least three different P wave morphologies to qualify. Each differing P reflects an alternate or aberrant depolarization origin within the atrium. Atrial, yes. The depolarizations are initiating from within the heart atria. Whether from areas of inflammation, irritation, scarring, or from medication or chemical toxicities. Tachycardia, yes. Faster than 100 bpm. The speed may be driven by increased cardiac demand, as in COPD, where some 60% of MAT cases come from or from the changing structure of the cardiac wall in congestive heart failure. Multifocal Atrial Tachycardia is a condition associated with up to 45% mortality (Buxton, 2020) (Tandon, 2020).

Image 30:

Multifocal Atrial Tachycardia



Heart Rate	Rhythm	P Wave	PR Interval	QRS
QRS rate usually >100	Irregular	More than 3 distinct P waves	Varies	Narrow <0.12

Image Source: Adapted from Wikimedia Commons, Courtesy of Jason E Roediger

In lead, II notice the arrows, which each point out distinctly different P wave morphologies. P-P waves will be largely irregular. Not fibrillation, not flutter, but irregular from the multiplicity of electrical stimulation points.

MAT is not in itself life-threatening. However, the comorbidities frequently associated with it are severe. A rapid irregular pulse may be the only indication of the presence of MAT. Complaints that might cause a client to possess this arrhythmia include increased shortness of breath, chest pain, palpitations, lightheadedness, or feelings of syncope.

Multifocal atrial arrhythmias can lead to some direct complications. These include myocardial infarction from unsteady oxygen supply and demand, pulmonary emboli, or atrial thrombi, especially stroke (Tandon, 2020).

Atrial Fibrillation

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Atrial Fibrillation (AFib or AF) is considered the most common type of treated cardiac arrhythmia, affecting between 2.7 to 6.1 million Americans (Thomas, 2020).

The rhythmic contraction of the atria counts for around 10% towards the overall output of the heart. When irritation, inflammation, or any other disorganizing factor gets in the way of the orderly metronome of the SA node, the chaotic firing of random atrial depolarization impulses leads to fibrillation, e.g., atrial quivering. AFib is a cardiac output disaster, especially in the elderly or those with comorbid medical conditions, as the billows-like effect from organized contractions can no longer push adequate quantities of blood into the ventricles. Despite this lack of compression, many people are asymptomatic with AFib. Those who do feel sudden fatigue, shortness of breath, dizziness, or chest pain tend to be surprised at how irregular their heart has become (Vaidya, 2018).

Image 31:

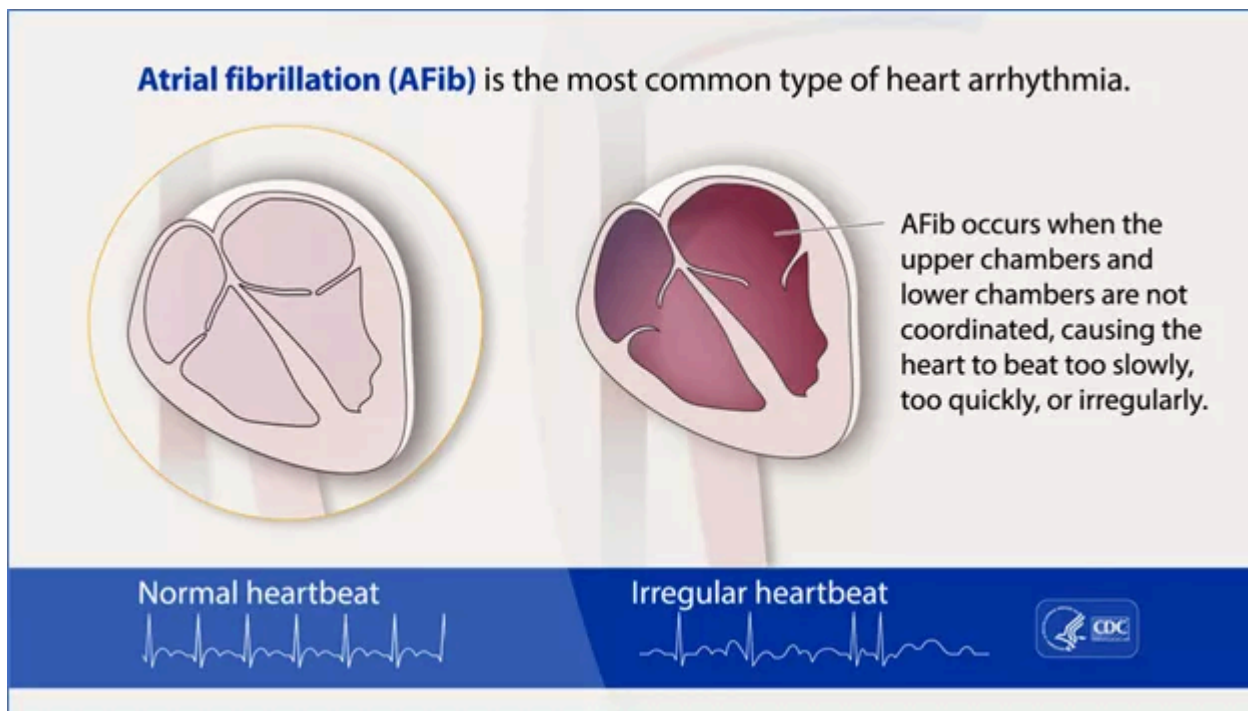


Image Source: Compliments of CDC.gov

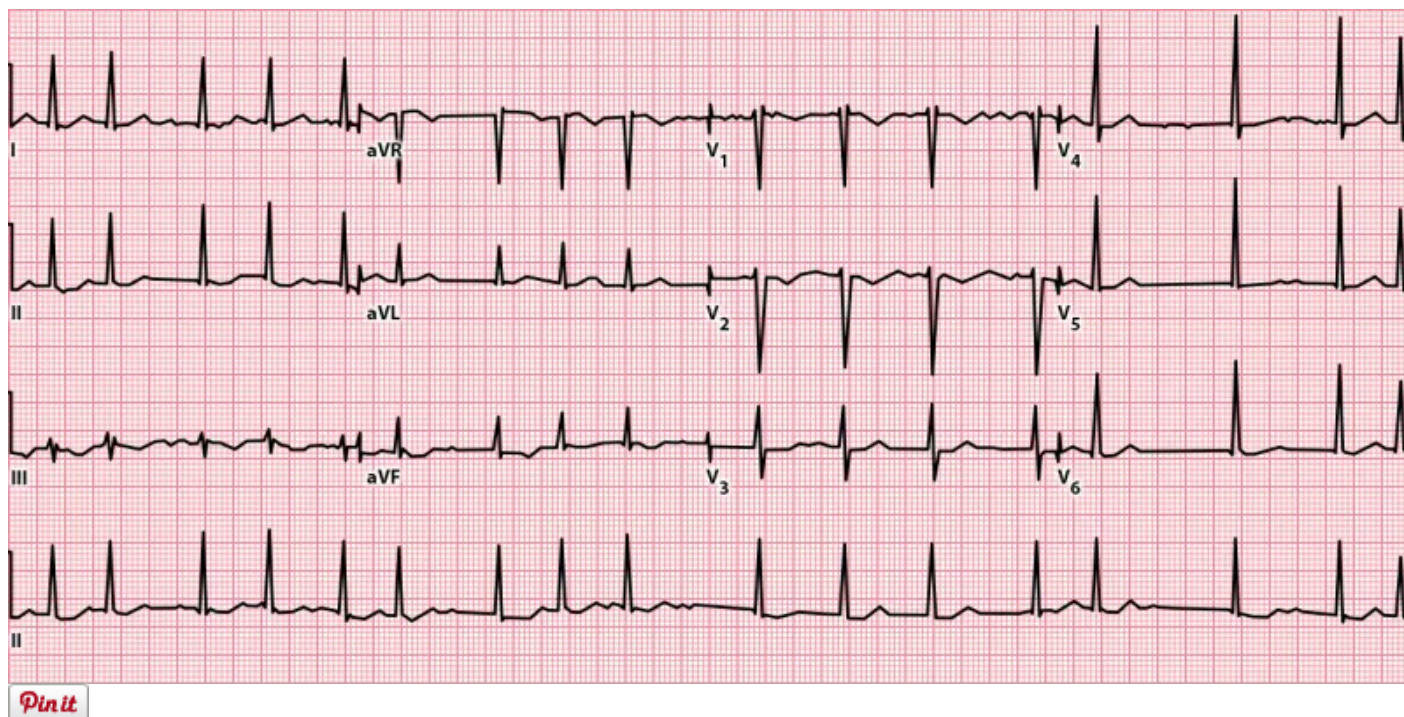
Complications of AFib can be serious (NHLBI, 2019).

- Blood Clots – Due to ineffective pumping, blood can churn and pool in the atria allowing thrombus, embolus, or clots to travel through the blood to different parts of the body.
- Stroke – Should an embolus reach the brain, a blood flow blockage or stroke may occur.
- Cognitive Impairment or Dementias – Studies support that AFib is associated with increased rates of cognitive impairment, Alzheimer's, and vascular dementia.
- Heart Attack – Women and African Americans show a high risk of heart attack associated with AFib.
- Heart Failure – The fast and uneven beating of the heart raises the risk of heart failure.
- Sudden Cardiac Death – Sudden stoppage of cardiac function and AFib sadly walk hand in hand.

So please, do not think of AFib as just a nuisance.

Image 32:

Atrial Fibrillation



Heart Rate	Rhythm	P Wave	PR Interval	QRS
Atrial rate usually >400	Irregular	No identifiable P waves	None	Narrow <0.12

Image Source: Adapted from Wikimedia Commons

The QRS rate of AFib will be all over the scale. Fast, then slower, then well, chaotic. So unpredictable that there are, in some viewpoints, three different types of atrial fibrillation.

Paroxysmal atrial fibrillation

A rhythm that switches back and forth between AFib and NSR

Permanent atrial fibrillation

The atrial fibrillation, which is present all the time and the de facto baseline rhythm

Persistent atrial fibrillation

Runs of fibrillation, when they occur, that tend to last longer than one week or until delivery of a small electric cardioversion shock or medications, reset the heart back to a normal sinus rhythm

In unstable clients, emergent, electrical cardioversion back to an NSR is needed. In clients whose condition allows more time, the consideration of rather to control the rhythm or control the heart rate must be explored. Generally, medication therapies must be individualized, and some surgical options are available. Oh, and anticipate that the client will be put on anticoagulants to minimize the occurrence of clot formation.

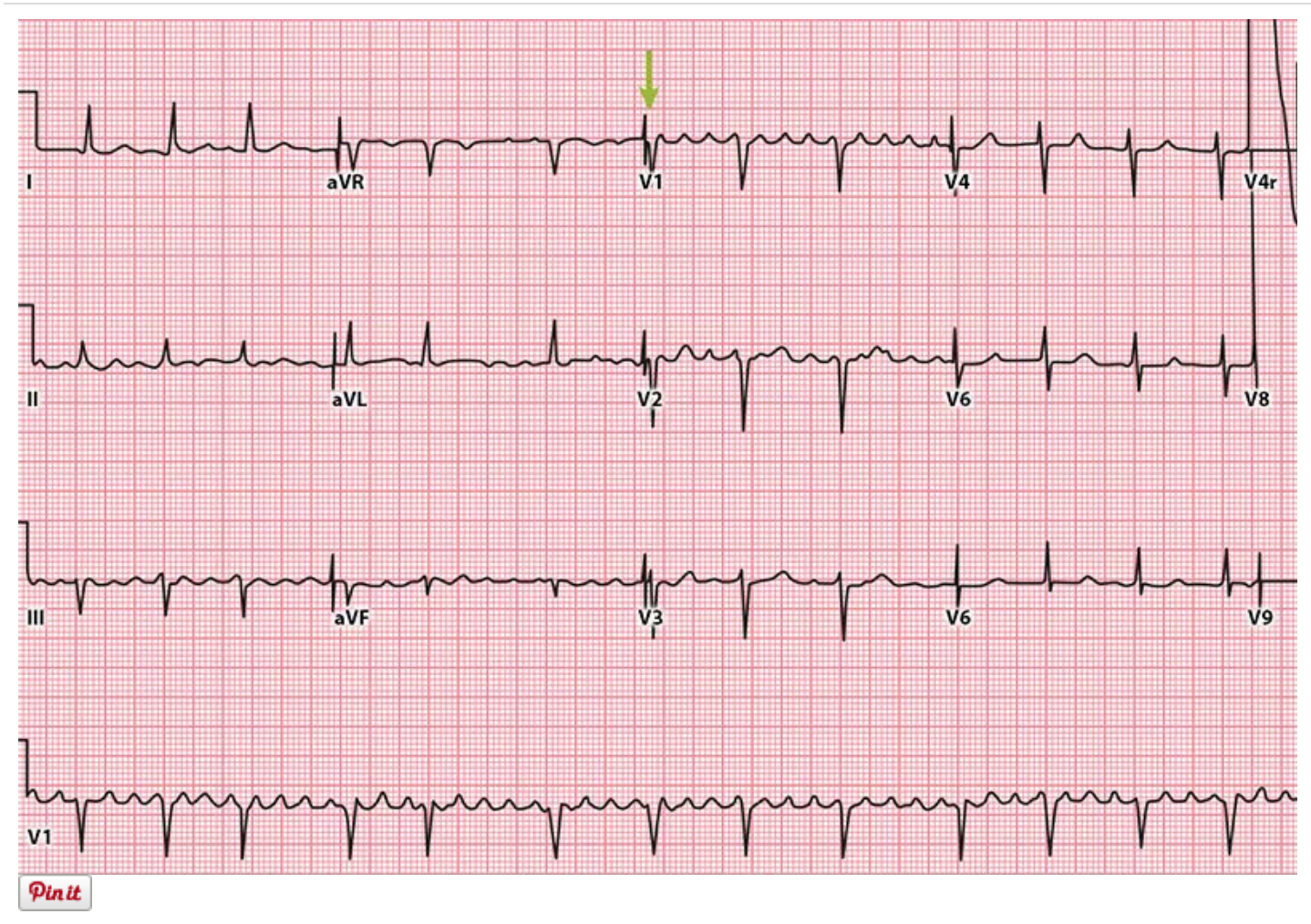
Atrial Flutter

Atrial flutter is the quintessential atrial tachyarrhythmia. Not the most common, yet it is the one a clinician tends to point out to students and state, "Now this is an atrial arrhythmia!"

The distinctive saw-tooth pattern of the atrial flutter waves is characteristic of multiple P waves successfully constricting the atria, yet only penetrating to the ventricle myocardium every second, third, or more atrial depolarizations. Pay special attention to leads II, III, aVF, and V1 to pick out the distinctive atrial flutter waves. Atrial rates will typically run 252–320 bpm. While ventricular rates will range around 120–160 bpm, with the most common ventricular rate in atrial flutter being 150 bpm, due to a phenomenon known as 2:1 atrioventricular block, which we will cover later.

Image 33:

Atrial Flutter



Heart Rate	Rhythm	P Wave	PR Interval	QRS
The atrial rate usually 250–320 Ventricular rate 120–160	Regular	Sawtooth P waves	Varies	Narrow <0.12

Image Source: Adapted from Wikimedia Commons

Here is a blowup of V1 from above. Note the choppy ocean waves of P in relation to the QRS complex. Most are 3:1, three P waves to each QRS. However, there is a variable ratio of P: QRS as we examine more of this lead. We would therefore call this Atrial Flutter with Variable Conduction (Jancin, 2020).

Image 34: V1– Atrial Flutter with variable conduction

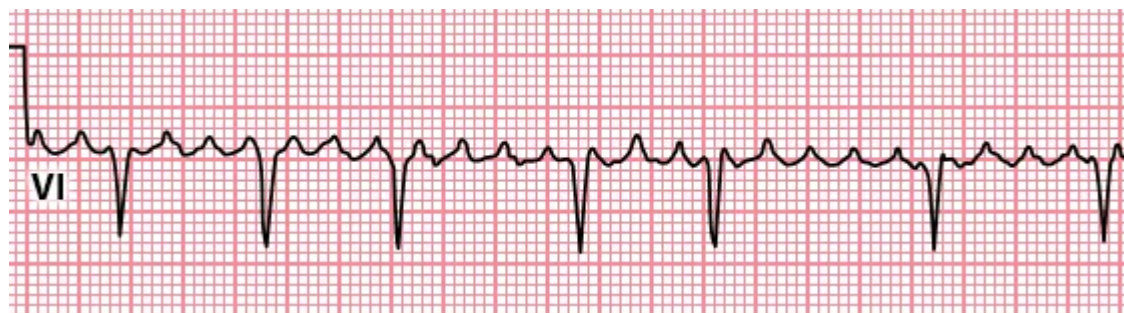


Image source: Complements of Wikimedia Commons

Atrioventricular Block

Atrial flutter tends to go hand in hand with an atrioventricular block (AV block). AV blocks are depolarization conduction delays, or at times complete barriers, to electrical conduction from the atria to the ventricles. Something as innocuous as increased vagal tone occurring during sleep, exercise, pain, or even stimulation of the carotid sinus. An AV block may also be related to cardiac fibrosis or sclerosis, ischemic heart disease, changes to the myocardial tissue, medications, or elevated plasma potassium (Medzcool, 2020).

The absent or slowed conduction from atria to ventricles, an atrioventricular block, is described by degrees. The seriousness of the conduction problem ranges from one to three. First degree AV block, second degree AV block type I (aka Wenckebach or Mobitz I} or type II (Mobitz II), and third-degree (complete) AV block (Petkar, 2021).

First Degree AV Blocks

Regular PR intervals greater than 200 ms (milliseconds) with no interruption in atrial to ventricular conduction are the signature indication of a first-degree AV block (Medzcool, 2020).

Image 35:

First Degree AV Block



Heart Rate	Rhythm	P Wave	PR Interval	QRS
60-100	Regular	Regular	>.20ms	Narrow <.12

Image Source: Adapted from Wikimedia Commons

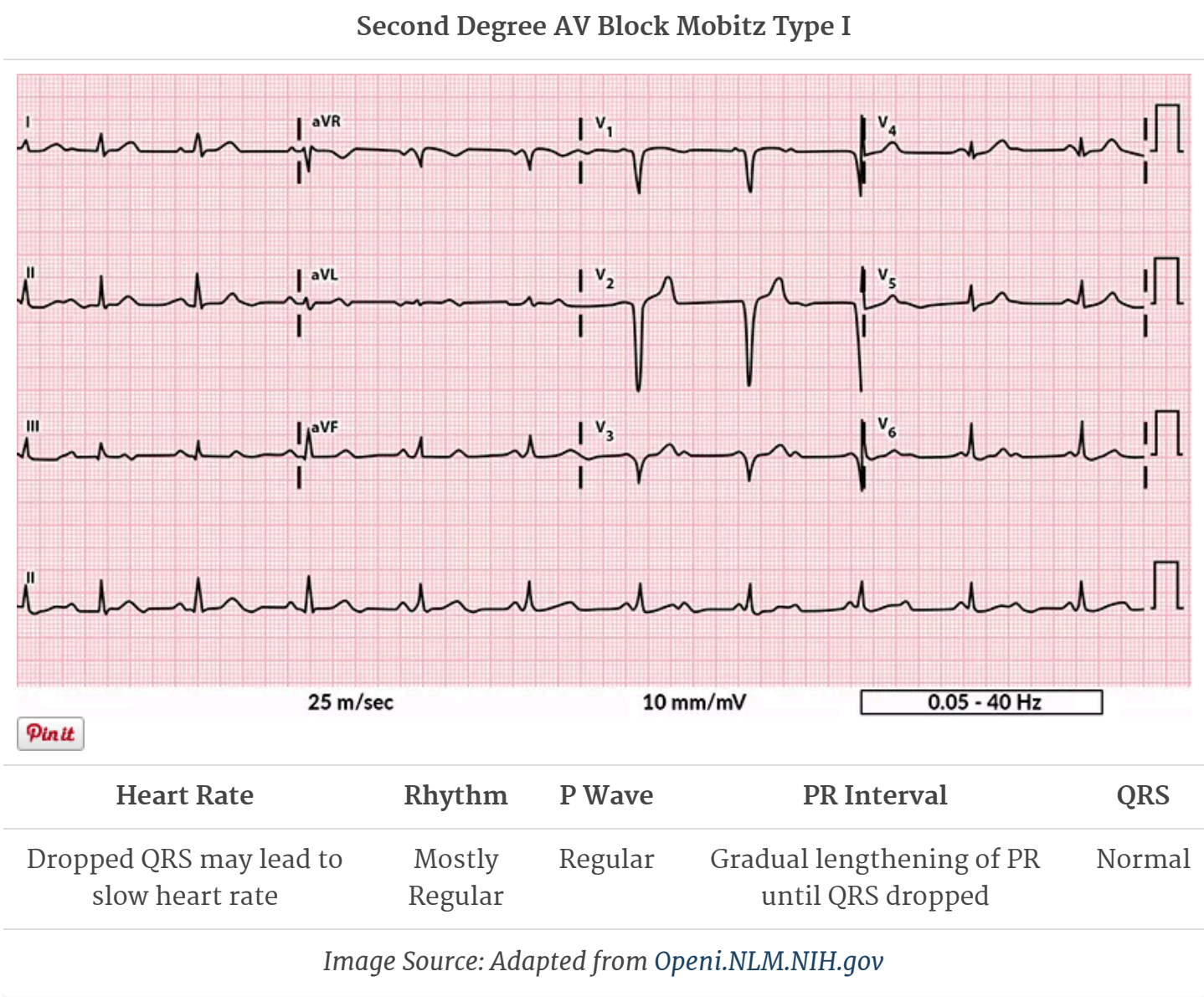
In first-degree AV block, the overall heart rate slows to approximate NSR, yet with a longer PR interval. It is remarkable in that there are no skipped beats. QRS complexes follow all P waves. In well-trained athletes or healthy younger clients with a high level of vagal tone, a first-degree

AV presentation may be non-pathologic and found only by chance. Further study is warranted for the rest of us to determine if an underlying cardiac condition may be developing, or most likely, some drug effect is manifesting (Mitchell, 2021).

Second Degree AV Block Mobitz Type I

Instead of staying static, the PR interval progressively lengthens in second-degree Mobitz type I AV block. This sequential lengthening until a QRS complex is dropped and the AV node conduction picked back up with the next beat is often referred to as the Wenckebach phenomenon, described by Karel Frederik Wenckebach in 1899.

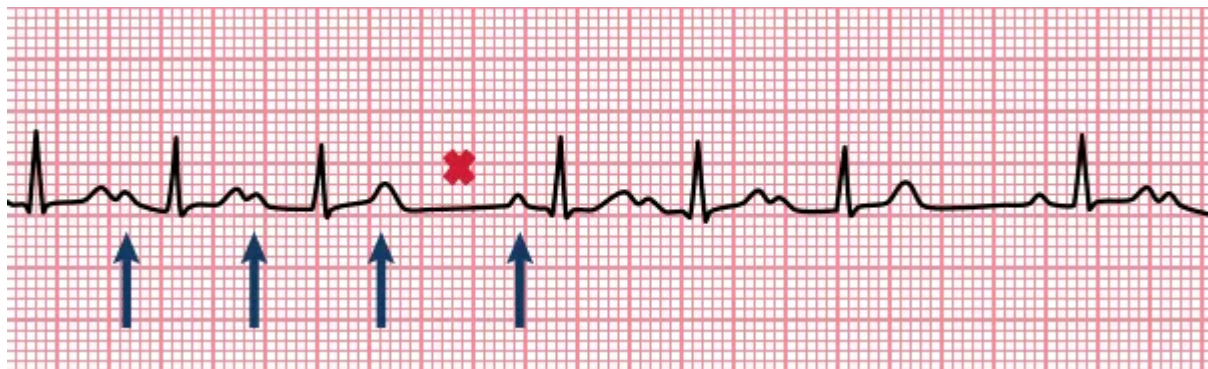
Image 36:



Second-degree AV block Mobitz type I is generally considered to be a harmless point of interest. Little risk of complete heart block exists with this arrhythmia. When it does cause, hemodynamic issues implantation of a cardiac pacemaker is the treatment by consensus.

Let us take a closer look at the strip above in lead II.

Image 37: Lead II



Pin it

P waves are regular, with receding QRS complexes until a QRS drops, then the pattern repeats.

Second Degree AV Block Mobitz Type II

Mobitz type II second-degree AV block is about the His-Purkinje system, sometimes referred to as the distal conduction system. When a block occurs at the level of the His bundle, or even slightly below at the Purkinje branches, no normal conduction will pass. There will be, in Mobitz type II, regular P waves and a regular PR interval. When QRS complexes are dropped, the P waves continue to march through the baseline until rejoined by QRS complexes holding the same PR interval as before.

This regular P wave, QRS dropped repeating pattern is where conduction P-to-QRS, written P colon QRS, or P: QRS, blocks occur. For example, a 3:2 block where three P waves accompany two successful QRS depolarizations before dropping a QRS. The P: QRS ratio can vary. However, each client should remain consistent. That is, you as a health professional may see across a spectrum of clients P: QRS ratios of 2:1, 3:1, 4:1, etc. However, in any one individual, the ratio P: QRS should remain consistent.

Type II Mobitz blocks can readily progress into a complete heart block where ventricular escape beats are too slow to maintain adequate perfusion or sudden cardiac death (SUD). Go ahead and do diagnostics, even a twenty-four-hour wearable cardiac monitor, sometimes referred to as a Holter monitor. Yet keep in mind the preferred treatment is an implanted cardiac pacemaker (Mitchell, 2021).

Image 38:

Second Degree AV Block Mobitz Type II



Pin it

Heart Rate	Rhythm	P Wave	PR Interval	QRS
Dropped QRS may slow heart rate	Regularly irregular	Regular	Regular except at dropped QRS	May be wide

Image Source: Adapted from Wikimedia Commons

Third Degree AV Block

Complete heart block, the lack of any conduction impulses making it through to the ventricles, is another way of saying third-degree AV block. No functional relationship exists between the P waves and the QRS complexes, referred to as AV dissociation.

P wave rate should always be faster than the QRS ventricular firing rate due to inherent escape rates in the atria and ventricles. The escape pacing below the atria originating from above the His bundle bifurcation (e.g., Junctional Escape rhythm) will produce narrow QRS complexes at a greater than 40 bpm heart rate. Escape pacing coming from below the bifurcation (e.g., Ventricular Escape rhythm) will have wide QRS and a slower heart rate, and with that slower heart rate, more severe symptoms such as syncope, low blood pressure, heart failure.

Junctional Escape Rhythm

Junctional escape rhythms are a sequence of electrical depolarizations that originate at, or near, the level of the AV node in the absence of a quicker, atrial, electrical depolarization event.

- Heart rate will be 40–60 bpm.
- Narrow QRS complexes.

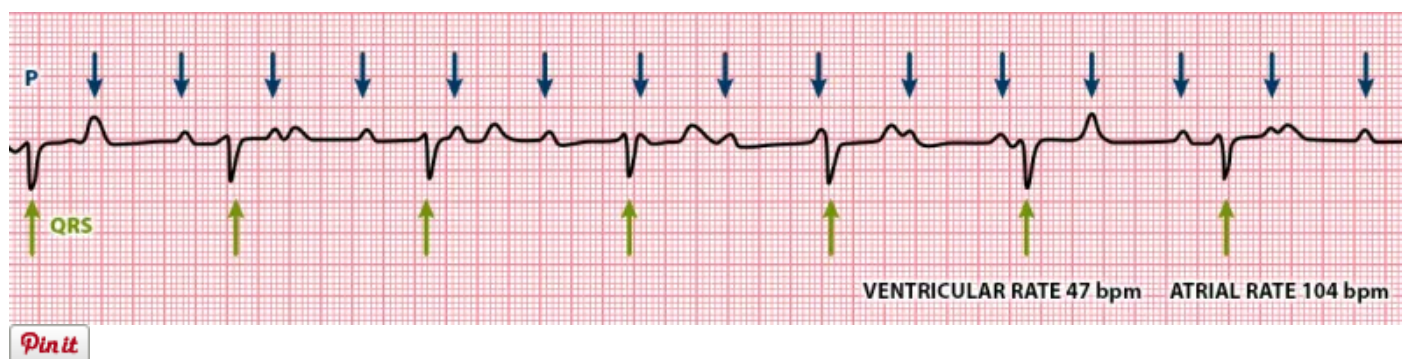
Ventricular Escape Rhythm

A rhythmic electrical depolarization that originates at, or near, the bundle of His

- Heart rate 20–40bpm.
- Wide QRS.

Image 39:

Third Degree AV Block



Heart Rate	Rhythm	P Wave	PR Interval	QRS
Different atrial and ventricular rates	Regular ventricular	Disassociated from QRS	No relationship	Widens the lower the escape source

Image Source: Wellcome Collection and Adapted from work licensed under a Creative Commons Attribute 4.0 International License

Notice in this monitor strip the atrial rate is fast, 104 bpm, and regular. The ventricular rate is regular but slow, 47 bpm. Neither atrial nor ventricular rates relate to the other. This ECG is distinctive of third-degree AV block, complete dissociative heart block.

Most clients experiencing complete heart block will require an implanted cardiac pacemaker, as their heart system is no longer able to supply this function adequately.

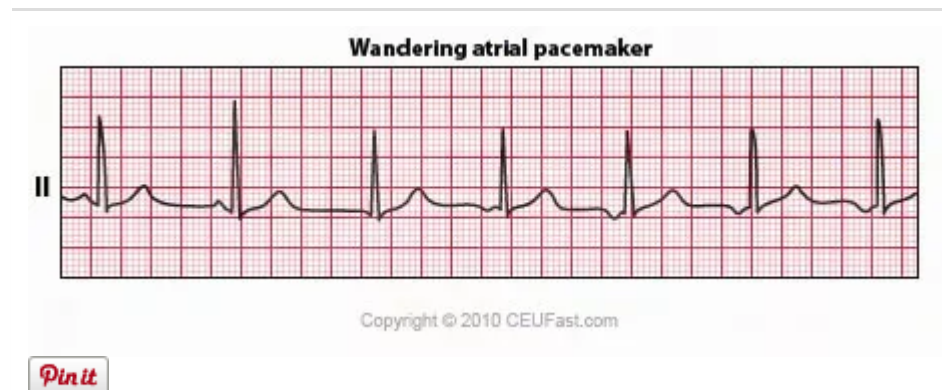
Wandering Atrial Pacemaker

No talk on heart arrhythmias would be complete unless it meandered into this, the where's Waldo of heart contractions. The Wandering Pacemaker.

A wandering atrial pacemaker is an arrhythmia originating in the atria where the pacemaker cells shift between the SA node, odd source spots within the atria themselves, and the AV node. These shifting, skipping about stimuli sites are generally best seen from lead II by looking for morphologic changes in the P waveform. It is most often seen in the young, the old, or in fit athletes. It tends not to be symptomatic and rarely requires treatment though the presence of the medication digoxin, or sometimes COPD, has been associated with it. Wandering atrial pacemaker is a favorite rhythm for instructors to give interns or learners while saying, "What's wrong here?" (Rogoff, 2021).

Image 40:

Wandering Atrial Pacemaker



Heart Rate	Rhythm	P Wave	PR Interval	QRS
60-100	Mostly Regular	From at least three sources	Mostly Regular	Narrow

Image Source: CEUfast.com

At least three different sources of atrial stimulation are present. QRS should be consistent and narrow. If this heart rate were faster, the arrhythmia would be considered multifocal atrial tachycardia.

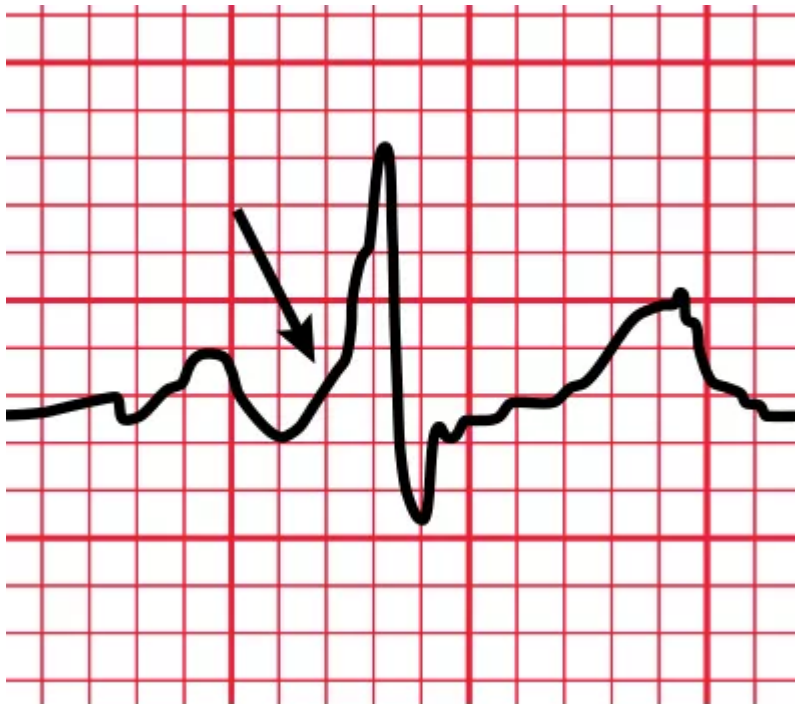
Wolff-Parkinson-White (WPW)

Video licensed from  3D4MEDICAL
Transforming Medical Learning

Wolff-Parkinson-White (WPW) displays intervals of abnormally fast heartbeats intruding on what otherwise would be a normally functioning heart rate. This rate is due to an additional abnormal electrical conduction pathway in the heart, which occasionally activates, leading to an extremely fast supraventricular tachycardia. The accessory pathways (APs) or bypass tracts connect the atrium to the ipsilateral (on the same side) ventricle allowing the ventricles a depolarization charge that pre-excites them, urging them to fire fast and often (Calkins, 2021).

This accessory pathway phenomenon, sometimes referred to as the Kent pathway, is shown by a short PR interval. By bypassing the AV node, the PR interval shortens. A delta wave becomes visible and represents early activation of the ventricles from the bypass tract. A form of fusion QRS results from two activation sequences, one from the bypass tract and one from the AV node. ST-T changes occur secondary to changes in the ventricular activation sequence.

Image 41: Delta Wave



Pin it

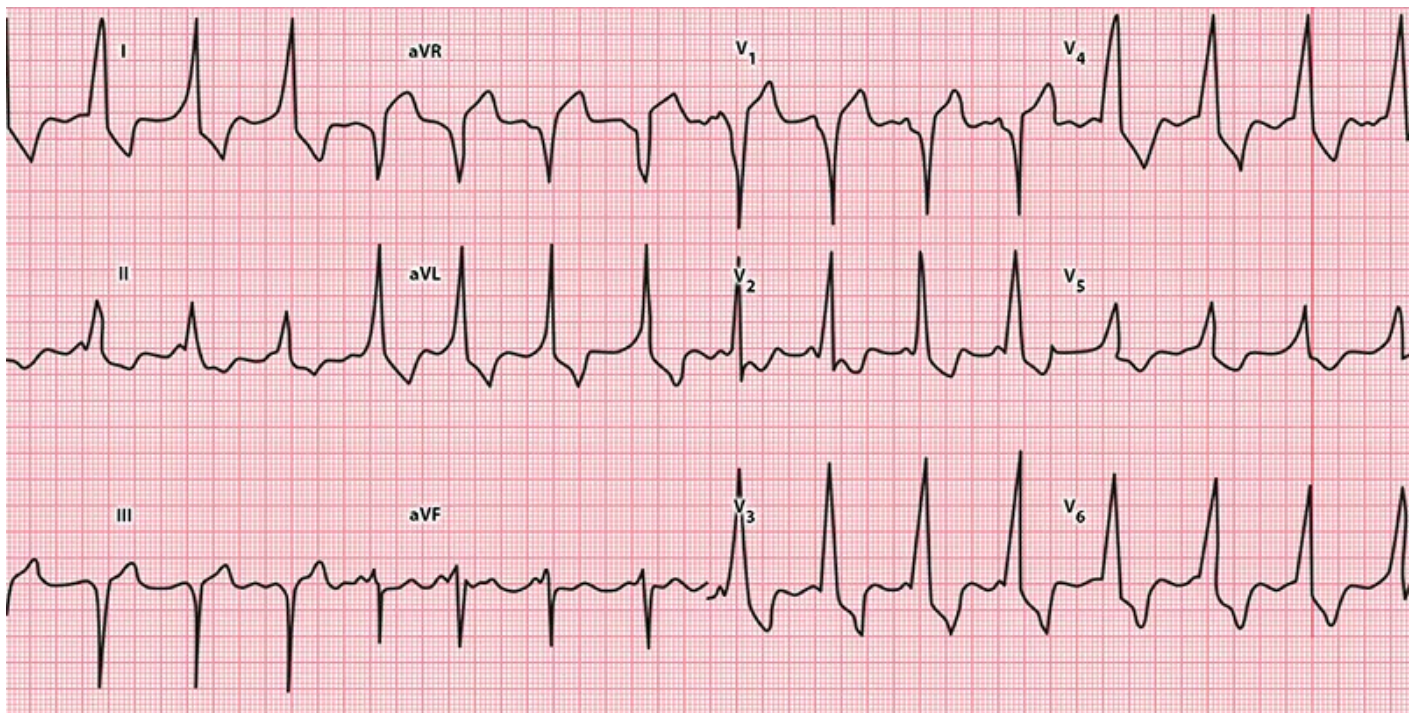
Image Source: Delta Wave, compliments of Wikimedia Commons

Short PR intervals and delta waves are best seen in leads V1-5. Pseudo-Q waves, seen in leads II, III, and aVF, are not Q waves but rather are negative delta waves. So do not be confused by the false Q's. There is no inferior MI on this ECG.

For a diagnosis, Wolff-Parkinson-White Syndrome (WPW) must be seen in more than one lead.

Image 42:

Wolff-Parkinson-White



Pin it

Heart Rate

Rhythm

P Wave

PR Interval

QRS

60-100 or more	Regular	Slurs into Q	Short when using accessory pathway	>.10
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Image Source: Adapted from Wikimedia Commons

Rate: Usually 60-100 beats/min but may be either faster or slower WPW may be due to congenital pathways that allow rapid conduction of impulses. These pathways predispose the patient to atrial tachycardia since there is no blocking of impulses at the AV node.

PRI: If this interval is short, the sinus impulse partially avoids its normal delay in the AV node by traveling rapidly down the accessory pathway.

QRS: Often greater than 0.10 seconds since there is no delay in the AV node. Subsequent activation of the ventricles depends upon intra-atrial conduction time from the sinus node to the accessory pathway plus conduction time down the accessory pathway, compared with sinus node conduction time to ventricles via orthodoxy conduction pathways.

Delta Wave: Slurring occurs at the beginning of the QRS complex.

Secondary T wave changes: Because ventricular depolarization is abnormal, repolarization will also be abnormal, causing ST and T wave changes secondary to the degree and area of pre-excitation.

Abnormal Q waves: Q waves are considered abnormal when they have an amplitude of 25% of the succeeding R wave or a duration of 0.04 seconds or greater. Such Q waves are often seen in the presence of an accessory AV pathway and may be misdiagnosed as Myocardial infarction. These are negative delta waves, not Q waves, and they reflect pre-excitation and not myocardial necrosis.

Ventricular Rhythms

Ventricular impulses come from the ventricles. The large muscular chambers of the lower heart push a pulsing stream of blood out into the body. When a command to contract signals fails to arrive from the primary pacemaker, the sinus node, escape cells within the ventricles step up to emit electrical depolarization waves that contract the ventricular myocardium.

Premature Ventricular Complexes (PVCs)

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Any early, untimely cardiac contraction arising from the ventricles is a premature ventricular complex (PVC). Many, if not most people who have the occasional PVC are completely unaware of them. Those who do perceive them tend to describe the sensation as a skipped beat or pounding heart. Both are accurate descriptions of what is brought about by the hemodynamic changes of sudden, early ventricular contractions.

Image: 43

Premature Ventricular Complexes



[Pin it](#)

Heart
Rate

Rhythm

P Wave

PR Interval

QRS

60-100	Regular atrial	Disassociated from the abnormal QRS	None on Abnormal QRS	Wide on abnormal beat >.12
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Image Source: Wellcome Collection, Adapted work is licensed under a Creative Commons Attribution 4.0 International License

Rate: Atrial and ventricular rate dependent upon the underlying rhythm.

Rhythm: Irregular due to PVC. If PVC is sandwiched between two normal beats, it is called interpolated, and the overall rhythm will be regular.

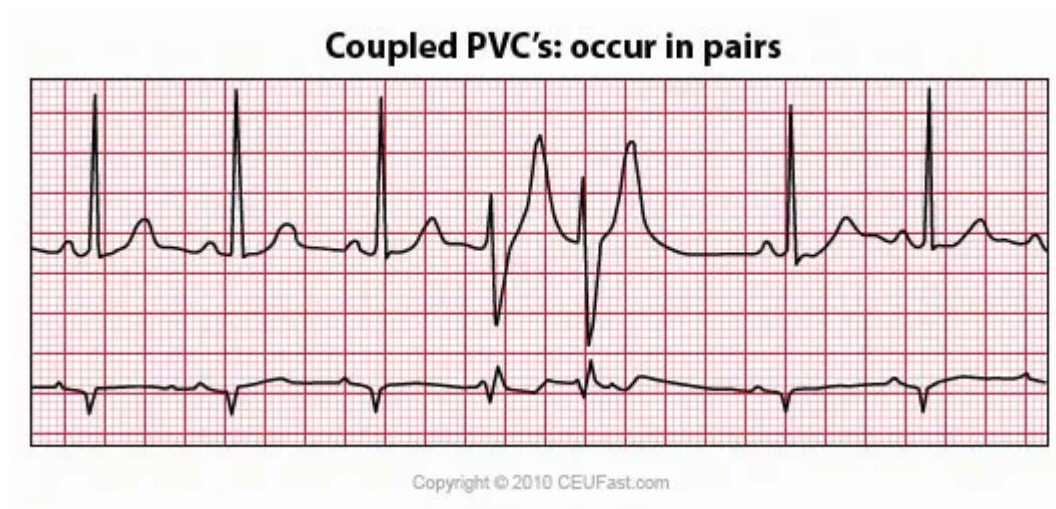
P Waves: A P wave is not associated with the PVC.

PRI: None with the PVC because the ectopic beat originates in the ventricles.

QRS: >.12 wide and bizarre. T wave frequently in the opposite direction of the QRS complex. If the QRS is negative, the T wave is usually upright; if the QRS is positive, the T wave is usually inverted.

PVCs may be due to Stress, activity, valvular disease, CAD, MI, caffeine, antihistamines, decongestants. The PVC may produce a weak pulse, and it is the client who should be treated, not the monitor.

Image 44:



Paired PVCs are referred to as couplets. Every other beat, a PVC is referred to as bigeminy PVCs.

Image 45:

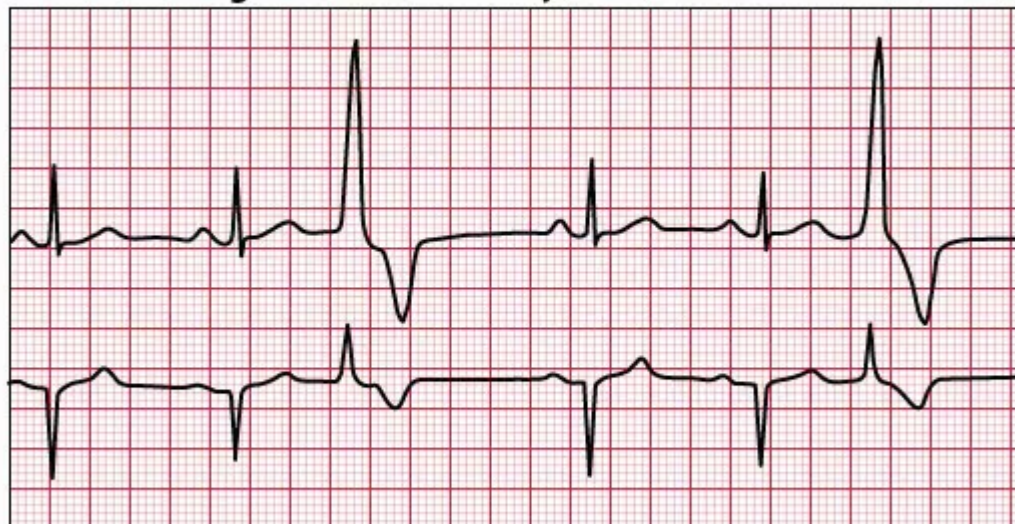
Bigeminal PVC's: every other beat is a PVC.

Copyright © 2010 CEUFast.com



Every third beat, a PVC is referred to as trigeminy.

Image 46:

Trigeminal PVC's: every third beat is a PVC

Copyright © 2010 CEUFast.com



PVCs occurring from more than one ventricular escape source during a sixty-second cycle are called multifocal PVCs.

Image 47:

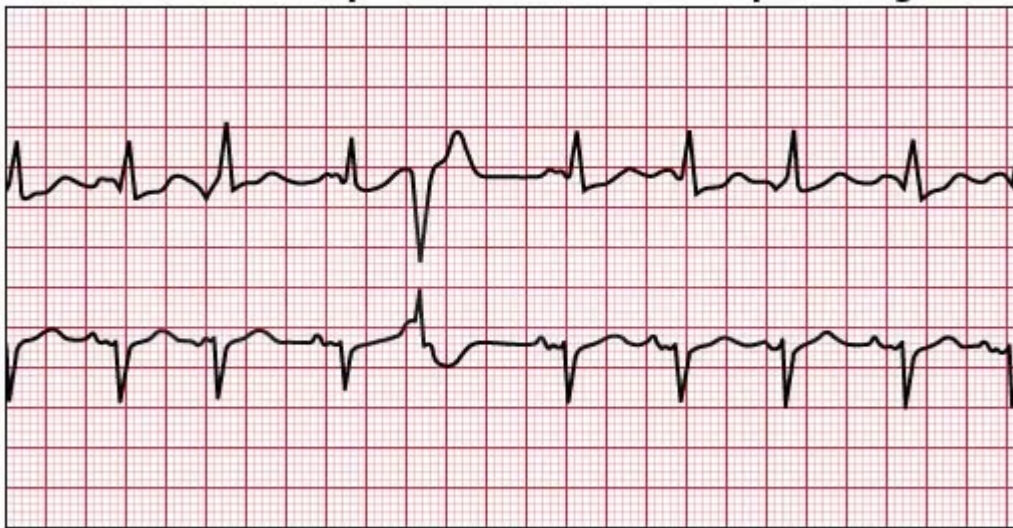
Multifocal PVC's: more than one shape

Copyright © 2010 CEUFast.com

Pin it

PVCs coming fast upon the QRS complex directly before, especially during the T wave myocardial recovery cycle, are a red alert for triggering a ventricular depolarization loop leading into ventricular tachycardia or straight into ventricular fibrillation.

Image 48:

R on T: occur on the peak of the T wave of the preceding beat

Copyright © 2010 CEUFast.com

Pin it

When considering abnormal ventricular beats, the top considerations are where the source originates and the speed. All contractions of ventricular origin tend to have wide $>.12$ QRS complexes and a lack of PRI consistency. So, let us talk speed.

Without irritation from comorbid or causation factors, a ventricular escape rhythm rate will be limited to 15-40 bpm.

Idioventricular Rhythm (IVR), aka Ventricular Escape Rhythm

Three or more ventricular origin beats in a row constitute IVR. Typically, IVR is transient, with a brisk return to a heartbeat of atrial derivation. Syncope, dizziness, and all that accompanies the rapid hemodynamic slowing which accompanies this rhythm.

Rate: Intrinsic rate is 20–40 beats per minute.

Rhythm: Atrial not discernible, ventricular essentially regular.

P waves: Absent.

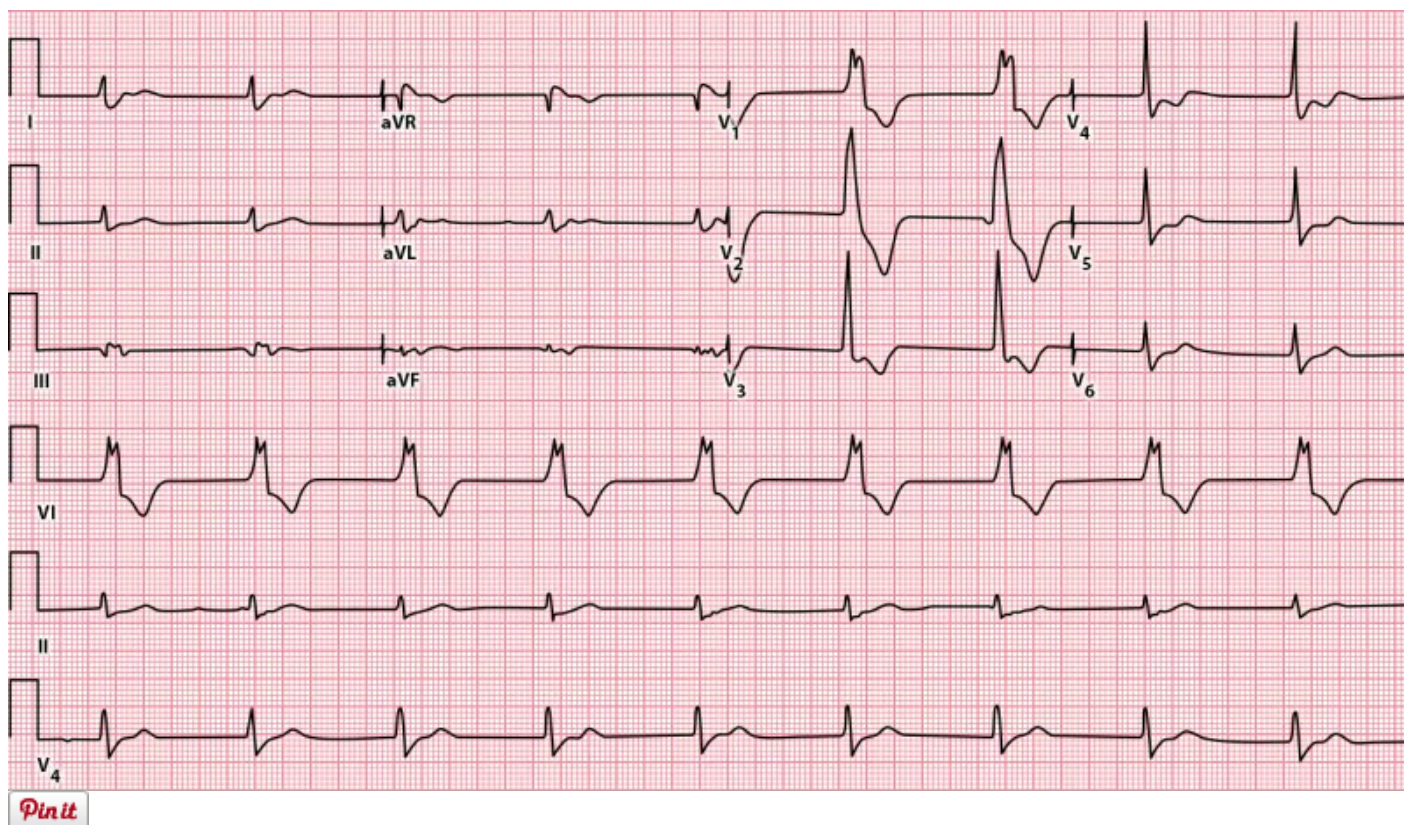
PRI: None.

QRS: $>.12$

It may be due to: MI, metabolic imbalances, or severe hypoxia. Treatment includes activation of emergency code, CPR if a client is pulseless. Lidocaine is contraindicated since it may knock out the last available pacemaker.

Image 49:

Accelerated Idioventricular Rhythm



Heart Rate	Rhythm	P Wave	PR Interval	QRS
40–100	Regular	Absent or not related to rhythm	NA	$>.12$

Image Source: Adapted from Wikimedia Commons, Courtesy of Jason E Roediger

Accelerated Idioventricular Rhythm (AIVR)

Faster than a ventricular escape rhythm, yet not fast enough to meet the blood pressure dropping ventricular tachycardia criteria. An accelerated idioventricular rhythm might, rarely, be considered a benign or asymptomatic arrhythmia. However, do not get complacent with the

lack of symptoms as AIVR is most common during cardiac tissue recovery from a myocardial injury. A time when any additional stress on the heart can tip recovery into adversity.

Rate: Atrial not discernable, ventricular 40–100 beats/minute.

Rhythm: Ventricular rate regular, an atrial rate not discernable.

P waves: Absent.

PR Interval: None.

QRS Complex: $> .12$

T Wave: NA.

QT Interval: Regular.

It may be due to Heart disease (e.g., acute myocardial infarction, digitalis toxicity, reperfusion of a previously occluded coronary artery), may occur during resuscitation, drugs (e.g., digoxin), dilated cardiomyopathy, and during outpatient procedures (especially during spinal anesthesia).

Ventricular Tachycardia (VT)

Ventricular tachycardia (V-Tach, VT) is a regular fast heart rate originating from an area of ventricular irritation. Short bursts of rapid ventricular contractions may not endanger a person. However, the less efficient circulation of blood from prolonged bouts can be life-endangering.

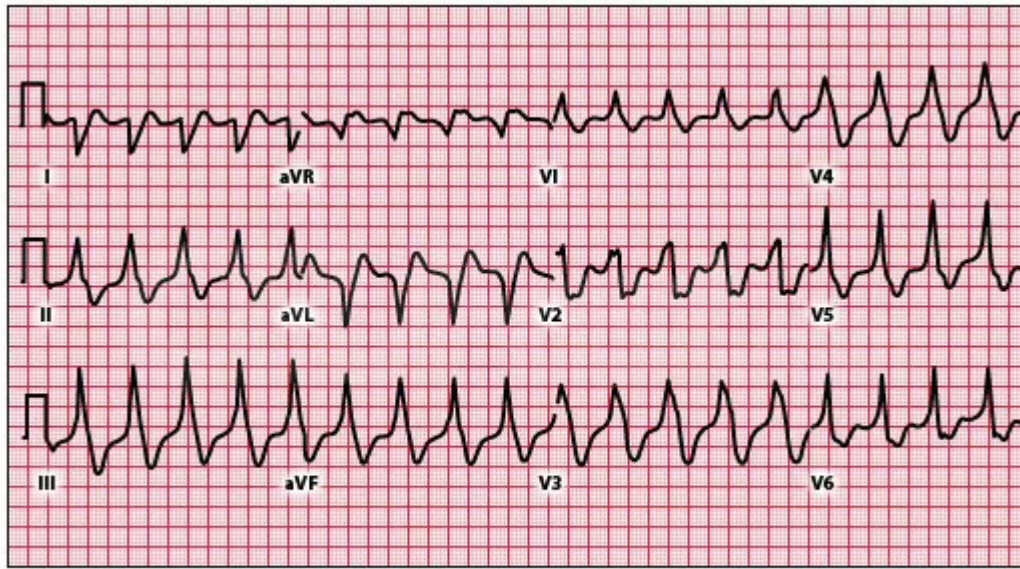
Characteristic findings indicating VT are tachycardia at >100 bpm, wide QRS complexes $> .12$, and AV dissociation. VT can be monomorphic, originating from one electrical excitation where all QRS complexes look alike. Or polymorphic, where multiple spots of electric stimulation are firing within the ventricles. Polymorphic VT is seen when each QRS shows a different morphology.

Bursts of VT lasting under 30 seconds are referred to as non-sustained V-Tach, while stretches longer than 30 seconds are referred to as sustained V-Tach.

Symptoms of ventricular tachycardia fall along the lines of reduced cardiac output and include hypotension, dizziness, syncope, cardiogenic shock, cardiac arrest.

Image 50:

Ventricular Tachycardia



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Pin it

Heart Rate	Rhythm	P Wave	PR Interval	QRS
100-250	Regular	Absent or not related to rhythm	NA	>.12

Image Source: CEUfast.com

Rate: Ventricular rate 100-250 beats/minute; atrial tends not to be discernible.

Rhythm: Atrial not discernible, ventricular essentially regular.

P Waves: May or may not be present. If present, they have no set relationship to the QRS complexes. P waves may appear between the QRS complexes at a rate different from that of the VT.

P-R Interval: None.

QRS Complex: Wide, >.12 ms (or 3 small ECG squares). Often difficult to differentiate between QRS and T wave. Three or more PVCs in a row at a rate of 100 per minute are referred to as a "run" of VT. There may be a long or a short run. A client may or may not have a pulse. If it is unclear whether a regular, wide QRS tachycardia is VT or Supraventricular Tachycardia, treat the rhythm as VT until proven otherwise.

Note: Ventricular tachycardia can occur in the absence of apparent heart disease.

T Wave: Difficult to separate from QRS.

QT Interval: Should be 390-450 ms. If longer be on alert for Torsades de Pointes.

Other Components: ≥ 3 consecutive wide QRS complexes at a frequency ≥ 100 /minute and signs of AV dissociation confirm VT diagnosis. Should the rapid QRS complexes appear identical to the client's NSR QRSs, suspect a supraventricular tachycardia is occurring.

It may be due to: An early or a late complication of a heart attack, or during cardiomyopathy, alveolar heart disease, myocarditis, electrolyte imbalance, or following heart surgery.

Torsades de Pointes

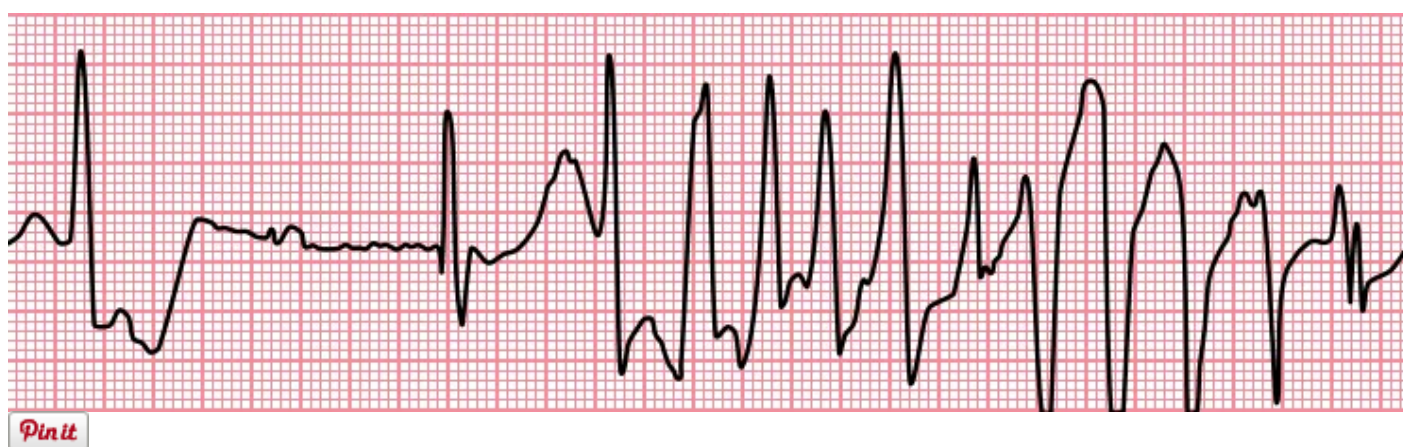
Torsades de pointes (TdP) is a type of polymorphic VT signified by a prolonged QT interval. The characteristic that makes TdP distinctive is how the QRS complexes twist around the isoelectric baseline during self-limiting bursts.

Around 50% of clients discovered with intervals of TdP are not symptomatic. However, 10% of those presenting with TdP will experience cardiac death (Cohagan, 2020).

The R on T phenomenon plays a significant role in TdP due to the prolonged QT interval that is associated with it. For example, as a PVC, stomps on the tail of an extended T wave torsades de pointes or polymorphic VT may be triggered. This occurrence magnifies the need for a thorough review of client medications as drug-induced long QT syndrome is, unfortunately, common (Cohagan, 2020).

Image 51:

Torsades de Pointes



Heart Rate	Rhythm	P Wave	PR Interval	QRS
>100	Irregular	Absent or not related to rhythm	NA	>.12

Image Source: Adapted from Wikimedia Commons

Rate: Generally, >100 bpm

Rhythm: Irregular with an oscillating or spindle looking twist around the baseline

P Waves: Absent, yet if by chance you see some, they will not be related to the QRS complexes

P-R Interval: Chaotic

QRS Complex: Each differs from its neighbor. There will be an overall effect of tall QRSs, which shorten then regain height. This complex goes with the spindle or torsade's effect.

T Wave: In the QRS complexes just before the torsades effect triggers, look for long prominent T waves. Also, keep an eye out for the R on T phenomenon, which can trigger VT or VF.

QT Interval: Prolonged QT intervals may be congenital, or more commonly, an unwanted effect of some prescription and over-the-counter medications.

Other Components: TdP is a significant adverse arrhythmia. However, the great concern when present increases in rate and degenerates into an even deadlier arrhythmia, ventricular fibrillation.

It may be due to: R on T trigger, antiarrhythmics, antipsychotics, antiemetics, antifungals, antimicrobials, basically any pharmaceutical with the adverse effect of prolonging the cardiac QT interval. Also, beware of substances that slow the hepatic metabolism. Slower liver breakdown of complex chemicals can turn a previously tolerated QT-prolonging substance into a landmine trigger, just waiting for an early R wave to activate the torsades effect (Cohagan, 2020).

Ventricular Fibrillation (VF)

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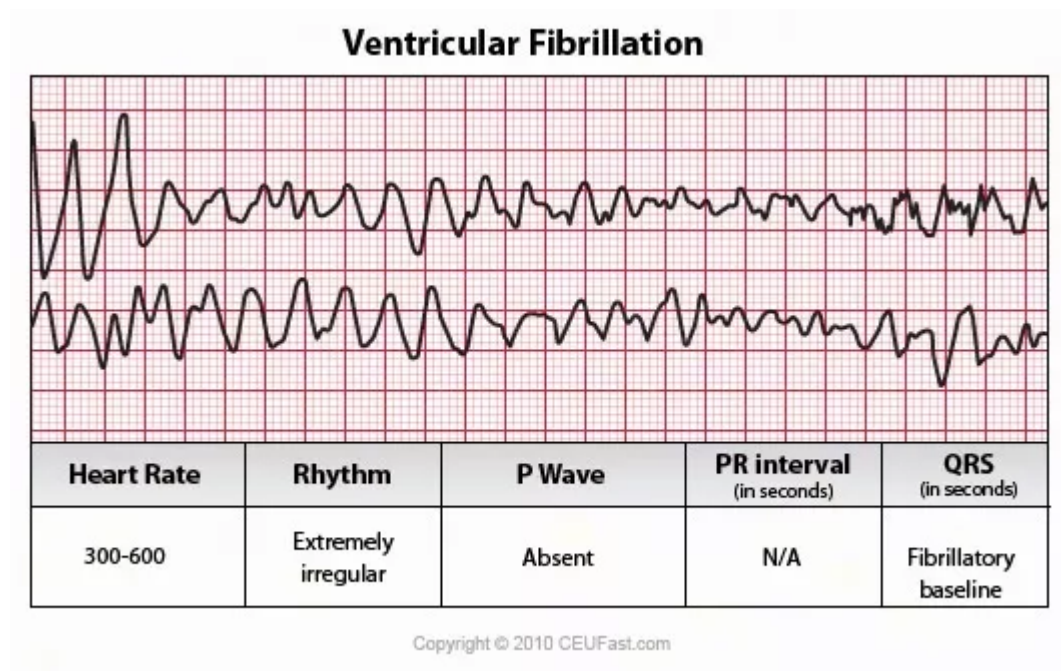
Ventricular fibrillation (V-fib or VF) is where the lower heart chambers quiver rather than constrict. Too many electrical polarization signals, arriving much too rapidly, reduce the strong rhythmic myocardial contractions to chaotic spasms. V-fib is a lethal arrhythmia resulting in rapid loss of consciousness, no pulse, and cardiac death in the absence of treatment.

Nearly 70% of cardiac arrest victims experience ventricular fibrillation. Without treatment, clinical death comes within minutes when V-fib is the prominent rhythm. Even when rescue efforts succeed, residual damage from the anoxic brain and neurologic damage requires follow-up and perhaps long-term treatment (Ludhwani, 2020).

V-Fib tends to accompany damage to the structure of the heart. Anything that can irritate or inflame the Purkinje cells of the ventricles has the potential to initiate the fast and multiple stimuli sites leading toward VF. Myocardial infarction, for example, shows V-Fib incidence of from 3 to 12% during the acute phase of myocardial damage. Many common conditions are associated with the chaotic irritability of V-Fib, including electrolyte abnormalities

(hypokalemia, hyperkalemia, hypomagnesemia), acidosis, hypothermia, hypoxia, cardiomyopathies, family history of sudden cardiac death, congenital QT abnormalities, and alcohol use (Ludhwani, 2020).

Image 52:



Rate: Rapid and disorganized

Rhythm: Irregular and chaotic

P Waves: Absent but may be recognized among the chaos

P-R Interval: Not measurable

QRS Complex: Composed of fibrillatory waves, wide irregular oscillations of the baseline

T Wave: Not measurable

QT Interval: Not measurable

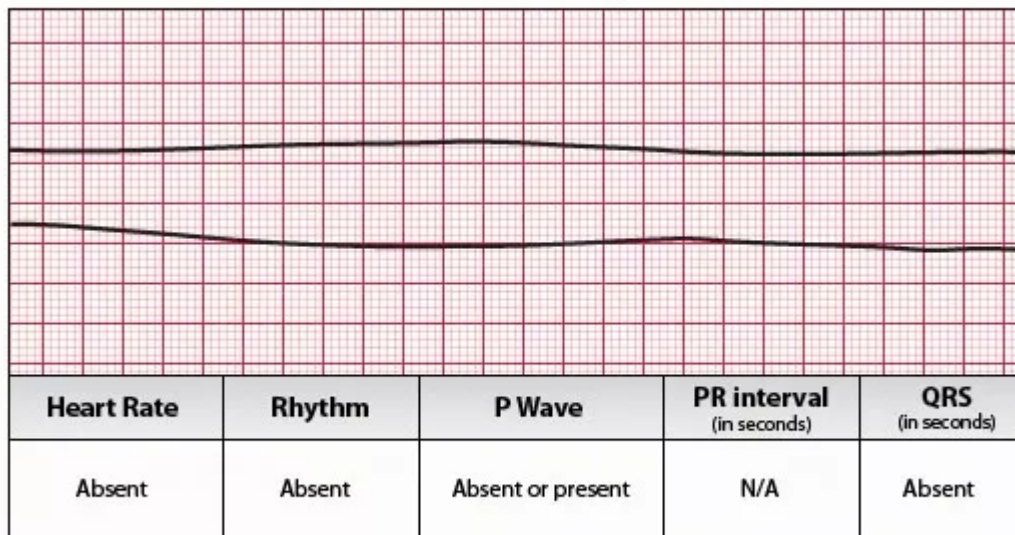
Other Components: Coarse VF is where most waveforms are 3mm or wider. Fine VF is where most waveforms are less than 3mm

Ventricular Asystole

Asystole is synonymous with Ventricular Standstill and death. Asystole is usually associated with prolonged circulatory insufficiency and cardiogenic shock. It could also be drug-related, hypothermia-related, and at times reversible.

Image 53:

Ventricular Asystole (standstill)



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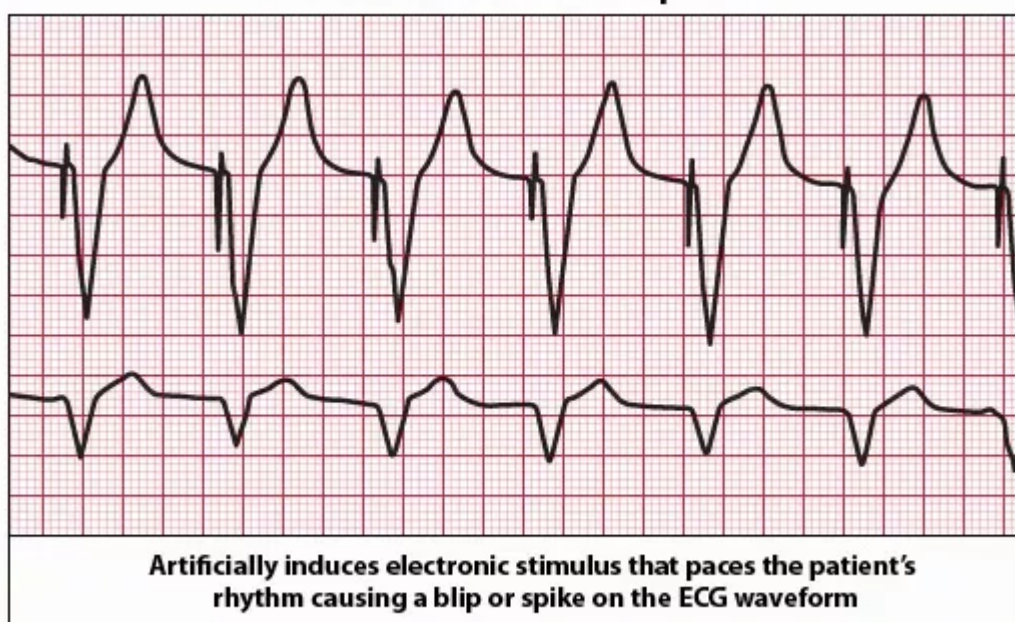
Artificial Cardiac Pacemakers

The natural electrical sources produce heart rhythms within the heart. When those natural pacemaker sites fail by producing too fast, too slow, or an absence of depolarization signals, another source of control is warranted. Enter the artificial cardiac pacemaker.

An implantable cardiac pacemaker is the most common type used. There are also external pacemakers that introduce a rhythmic electronic pulse that is used mostly during emergencies. Internal pacemakers are used in cases requiring long-term availability to override a dangerous heart rhythm or replace an absence of functional heart rhythm.

Image 54:

Electronic Pacemaker Spikes



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Implantable pacemakers can pace on-demand or continuously. They tend to stimulate just one heart chamber, or sometimes two. The small pacemaker unit is implanted under the skin with output leads connected directly to the heart muscle. Small batteries provide power to recognize the heart's electrical activity and provide needed electrical pulses to the heart muscle.

Types of Artificial Implantable Cardiac Pacemakers

Fixed-rate pacemaker

They are used primarily on clients with significant or complete heart blocks. The rate is pre-set to a rate such as 70 bpm, though rate changes can be made using external magnetic control (most commonly).

Demand pacemaker

Only fires when the R-R interval of the client's natural rhythm meets or exceeds a preset limit.

R Triggered Pacemaker

When dealing with heart blocks possessing occasional sinus rhythm, the ventricular synchronized demand-type pacemaker, the R wave triggered pacer, looks for the absence of R waves and stimulates the heart ventricles should they not appear after a short delay.

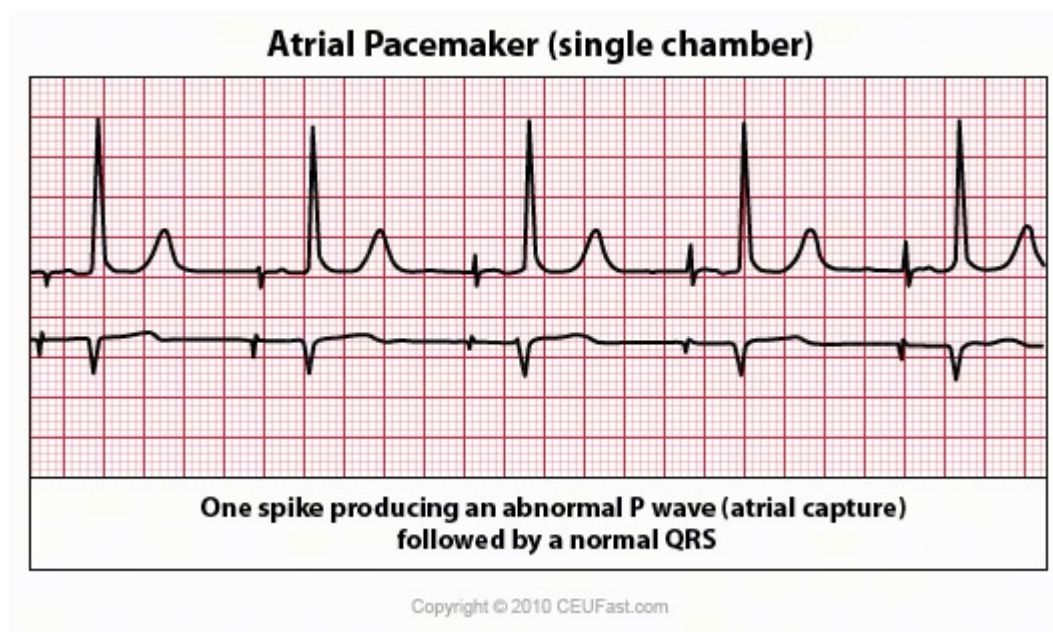
R Blocked Pacemaker

For clients with sinus rhythm and only an occasional heart block, the R wave blocked pacemaker stops firing when it detects a natural R wave produced by the client.

Atrial Triggered Pacemaker

When detecting natural atrial depolarization, the pacemaker stimulates the ventricles after a reasonable delay. This pacemaker provides the best cardiac output while following the normal atrial rate fluctuations.

Image 55:



Dual Chamber Pacemaker

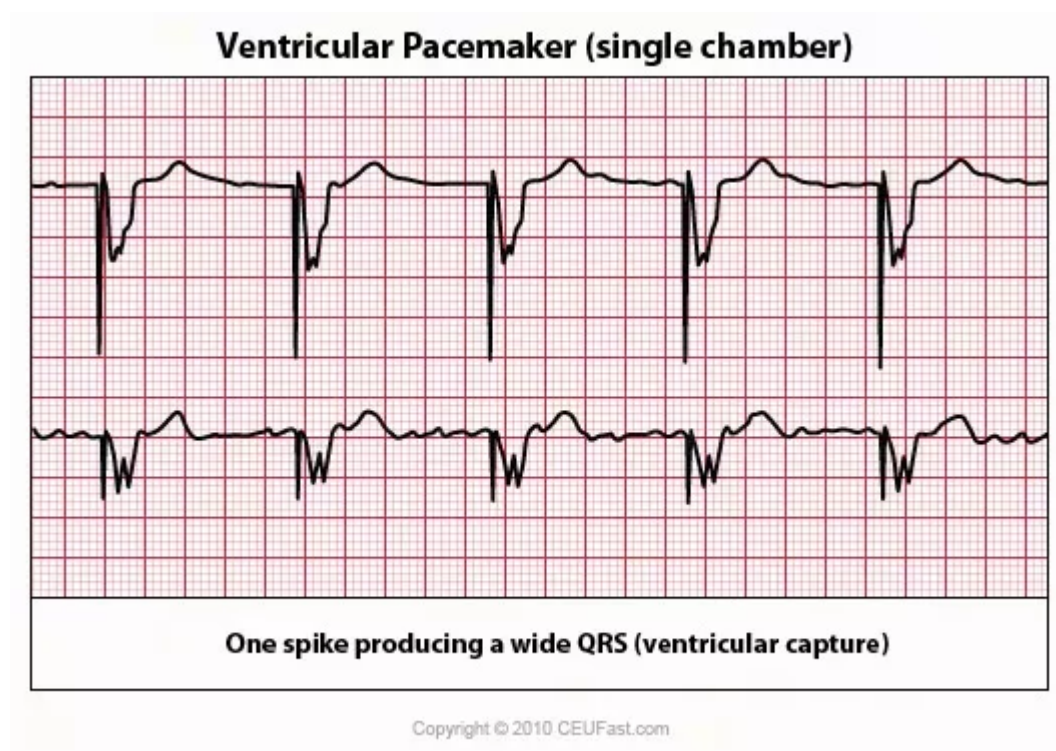
Treats most sino-atrial conditions by providing both atrial and ventricular stimulation whenever it is needed.

Pacemaker Terminology

Firing refers to the pacemaker's generation of electrical stimuli. This impulse is seen as a narrow vertical pacemaker spike on the ECG.

Capture refers to the presence of a P, a QRS, or both after a pacemaker spike. This capture indicates that the tissue in the heart chamber being paced has been depolarized. The term is that the pacemaker has "captured" the chamber being paced. Paced QRS is wide, bizarre, and resembles PVCs.

Image 56:



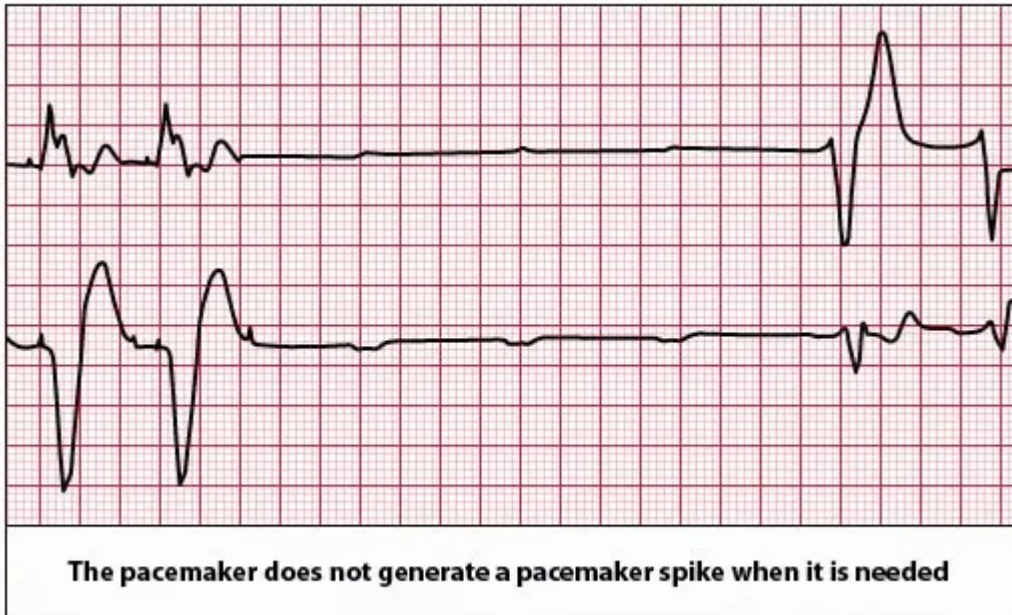
Sensing refers to the pacemaker's ability to recognize the client's intrinsic rhythm to determine if it needs to fire. Most pacemakers function in the demand mode and fire when needed.

Pacemaker Malfunctions

When a pacemaker fails to send a needed impulse, it is said to have malfunctioned, failed to fire, failed to output. Usually, this means a low battery or that one of the connecting wires anchored in the heart wall is at fault. At times, the type of electrical artifacts that crop up on ECGs can fool the pacemaker to not fire. A no output incident is revealed as the lack of a pacemaker spike where there should be one.

Image 58:

Failure to Output



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Loss of Capture:

There is no P or QRS after the pacemaker has fired when loss of capture exists, just a spike. This loss of capture usually means a pacemaker needs to be adjusted to detect the heart's need to be paced. A pacing wire may have lost contact with the chamber wall, which can occur when the heart is too damaged to respond.

Under-sensing:

Under-sensing occurs when the pacemaker fires too soon after an intrinsic beat, and there are pacer spikes where there should not be. These can appear in the T wave, on the QRS, or anywhere on the heart rhythm's tracing. Under-sensing requires adjustment with the wires or battery replacement.

Conclusion

Our heart is an exquisitely crafted pumping machine whose myocardial muscle cells move upwards of six thousand liters of blood every day. These wonderful engines are controlled by rhythmic electrical pulsations originating from natural pacemaker cells located in the apex of the heart itself. So well designed are our hearts' that redundant backup pacemaker points exist to take over should our primary pacing points fail. Ironically, this problem is one reason we need a consistent method of examining the heart's electrical activity to see what in our heart is happening.

Electrocardiography is the science of recording and examining the activity of the heart. From how the sinus node's pacemaker cells polarize then depolarize, sending a spark of life in a wave of depolarization down through electric sensitive tissue pathways. This depolarization wave creates myocardial muscle contracture of the near atrial chambers and quickly after the large muscular ventricles of the heart, creating a bellows that pushes blood into an eager body. Each step the electrical conduction wave takes through the heart creates a different waveform on the isoelectric baseline of an ECG monitor strip.

The atrial depolarization from the sinus node is the P wave. The movement of the electric pulse through the atrial tissue to the AV node and the His-Purkinje fibers causes atrial constriction. This constriction is the PR Interval. As the depolarization enters and spreads widely into the cardiac ventricles, a QRS complex is projected onto the ECG. The short period of recovery occurring between ventricular cell depolarization and repolarization is seen as the ST segment, with the T wave signaling full repolarization. Always one for a bit of mystery, our heart can throw up a wave we call U, which follows the T and precedes the P. We have no idea why, yet life contains plenty of mysteries to pique our curiosity (Amboss, 2021).

We place the positive and negative cardiac monitor to give us special angles for viewing the hearts' electrical activity. Twelve special lead placements compose a cardiac 12-lead ECG, the diagnostic standard for electrocardiograms. How we evaluate what is going on in the heart using an ECG strip requires a system. Some of the steps of a winning system include looking at the heart rhythm, heart rate, P waves, PR Intervals, QRS complexes, ST segment, T wave, QT duration, and then anything special such as Delta or U waves.

Using a systematic approach, we can determine where the rhythm originates from the atria, junction, or ventricles if it is normal, fast, or slow; If there are unusual beats; If the entire rhythm is unusual and perhaps unwanted, an arrhythmia; if unusual spots of excitement, electrical blockage, chaotic electrical fibrillation, or lack of electrical activity are present.

Not only can we see and diagnosis natural cardiac functions, but we can also look at the functioning of implanted cardiac pacemaker devices to determine if their function is appropriate or failing. A failing artificial pacemaker can show as under sense, failure to capture, or output failure.

When you want to see to the heart of health issues, remember, ECG!

Implicit Bias Statement

CEUFast, Inc. is committed to furthering diversity, equity, and inclusion (DEI). While reflecting on this course content, CEUFast, Inc. would like you to consider your individual perspective and question your own biases. Remember, implicit bias is a form of bias that impacts our practice as healthcare professionals. Implicit bias occurs when we have automatic prejudices, judgments, and/or a general attitude towards a person or a group of people based on associated stereotypes we have formed over time. These automatic thoughts occur without our conscious knowledge and without our intentional desire to discriminate. The concern with implicit bias is that this can impact our actions and decisions with our workplace leadership, colleagues, and even our patients. While it is our universal goal to treat everyone equally, our implicit biases can influence our interactions, assessments, communication, prioritization, and decision-making concerning patients, which can ultimately adversely impact health outcomes. It is important to keep this in mind in order to intentionally work to self-identify our own risk areas where our implicit biases might influence our behaviors. Together, we can cease perpetuating stereotypes and remind each other to remain mindful to help avoid reacting according to biases that are contrary to our conscious beliefs and values.

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