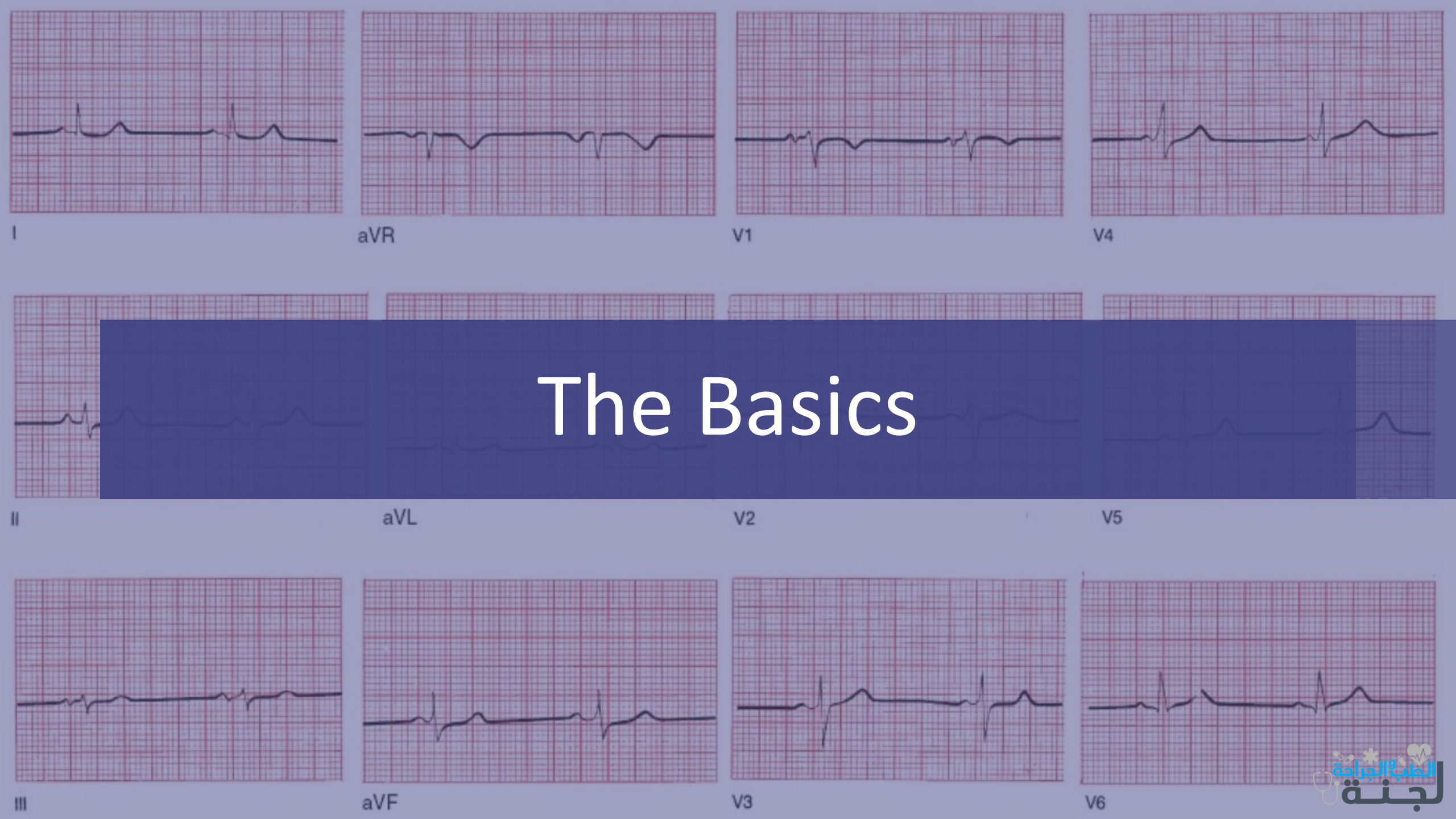
EKG Hacks

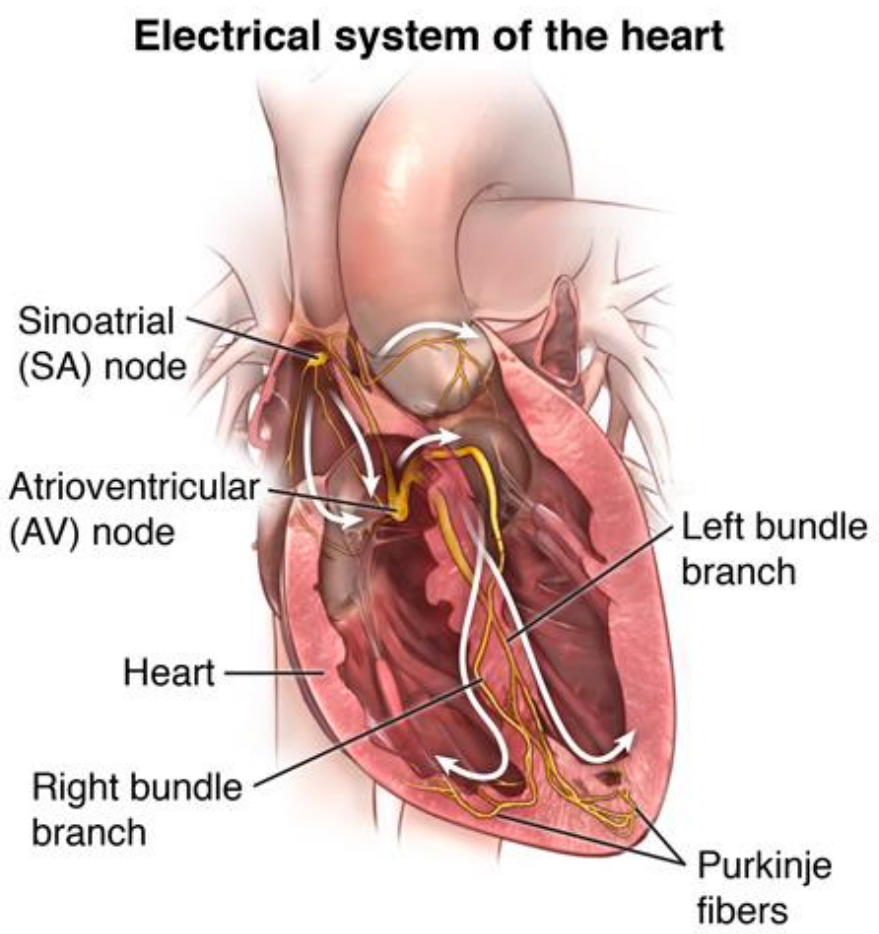
2024 Edition

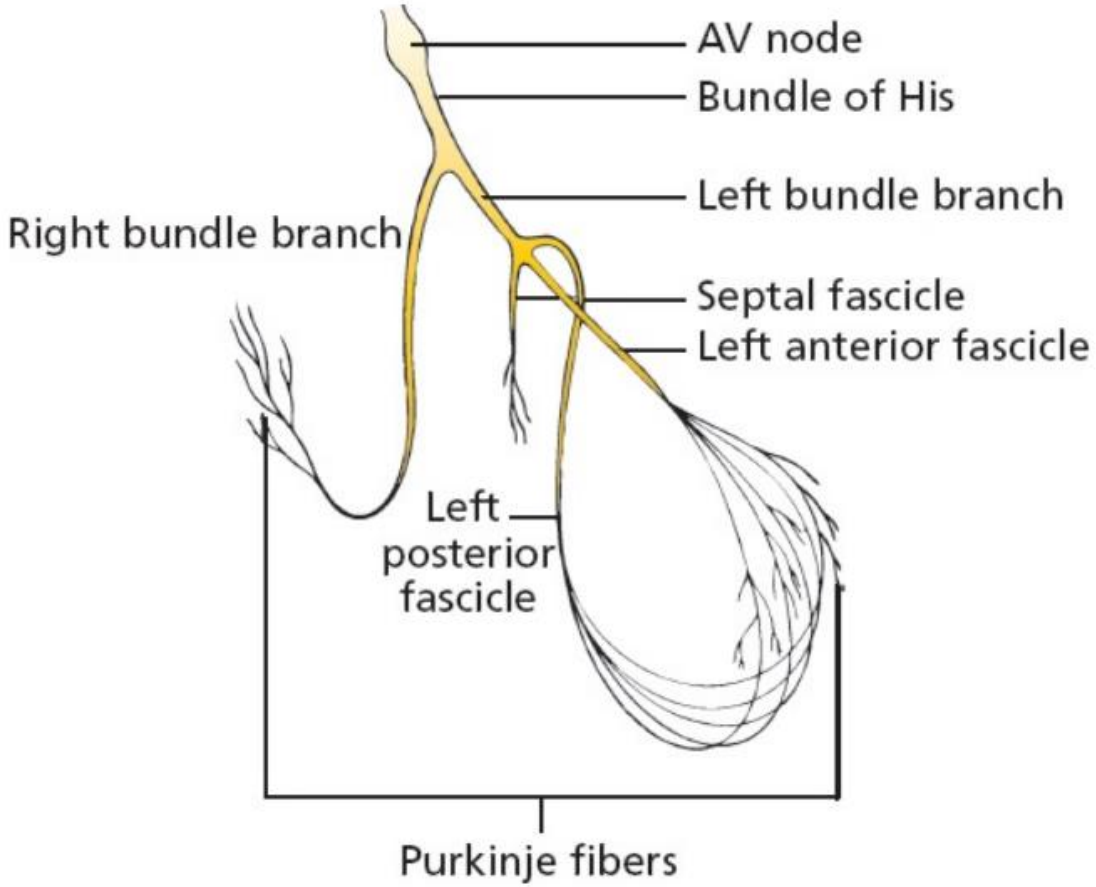
إعداد محمود بركات



The Basics

Anatomy of the heart conductive system

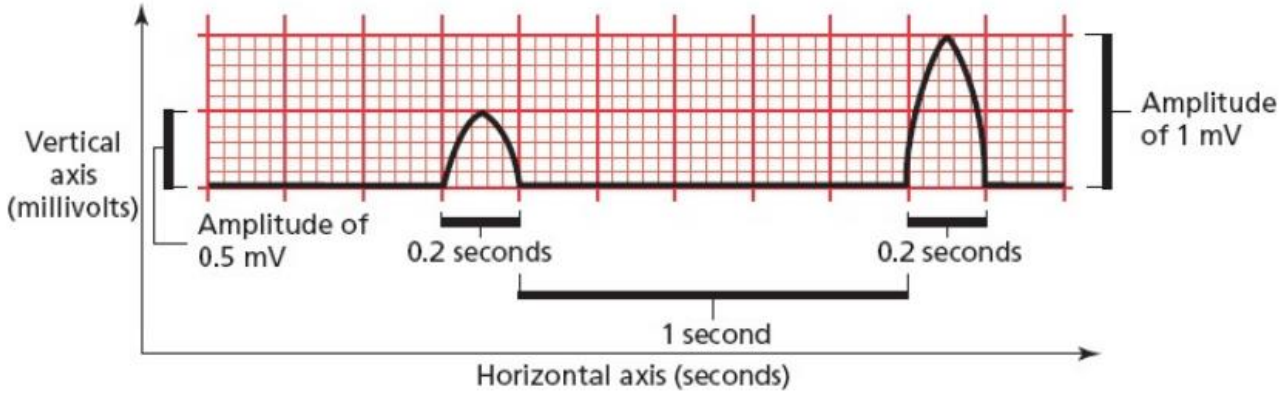




The ventricular conduction system, shown in detail. Below the bundle of His, the conduction system divides into right and left bundle branches. The right bundle branch remains intact, whereas the left divides into three separate fascicles.

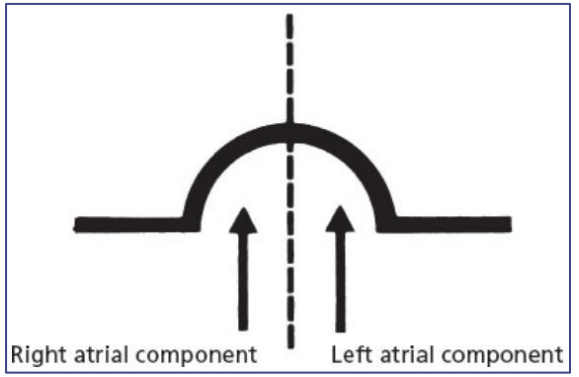
EKG Paper

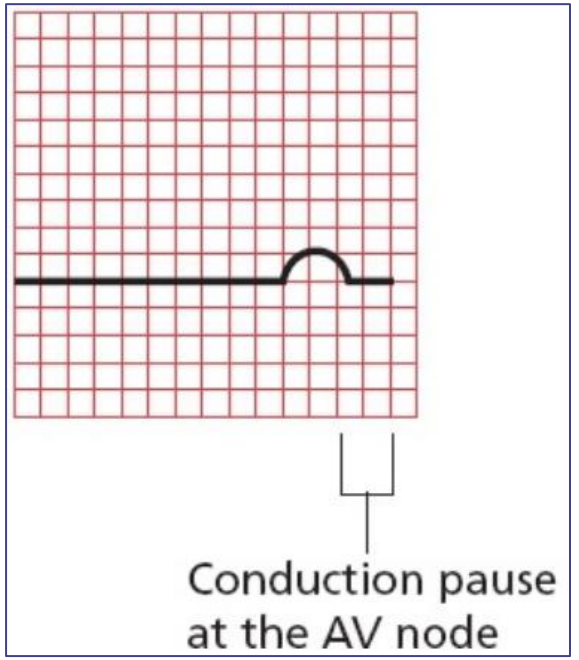
- EKG paper is a long, continuous roll of graph paper, with light and dark lines running vertically and horizontally. The light lines circumscribe small squares of 1 × 1 mm; the dark lines delineate large squares of 5 × 5 mm.
- **The horizontal axis measures time**.
  - The distance across one small square represents 0.04 seconds.
  - The distance across one large square is five times greater, or 0.2 seconds.
- **The vertical axis measures voltage**.
  - The distance along one small square represents 0.1 mV.
  - The distance across one large square is five times greater, or 0.5 mV.



The basic waves and lines of the standard EKG

- **P wave**
  - Represents atrial depolarization
  - The **first part of the P wave** predominantly represents right atrial depolarization, and **the second part** left atrial depolarization, because the sinus node is located in the right atrium, the right atrium begins to depolarize before the left atrium and finishes earlier as well. [First picture]
  - Once atrial depolarization is complete, the EKG again becomes electrically silent
- **PR interval** [Second picture]
  - A Pause Separates Conduction From the Atria to the Ventricles as the AV node slows conduction to a crawl.
  - This physiologic delay in conduction is essential to allow the atria to finish contracting before the ventricles begin to contract.

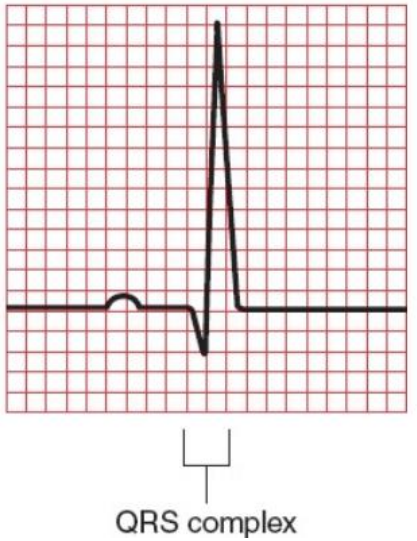


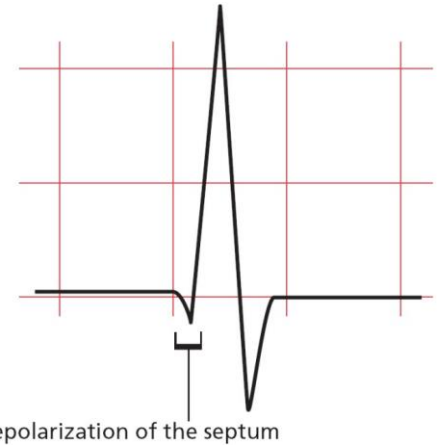


The basic waves and lines of the standard EKG

❖QRS complex

- Ventricular myocardial depolarization causes ventricular contraction.
- The amplitude of the QRS complex is much greater than that of the atrial P wave because the ventricles have so much more muscle mass than the atria.
- The earliest part of the QRS complex represents depolarization of the interventricular septum by the septal fascicle of the left bundle branch. The right and left ventricles then depolarize at about the same time, but most of what we see on the EKG represents left ventricular activation because the muscle mass of the left ventricle is about three times that of the right ventricle
- The QRS complex is also more complicated and variable in shape than the P wave, reflecting the greater intricacy of the pathway of ventricular depolarization. (See next slide)

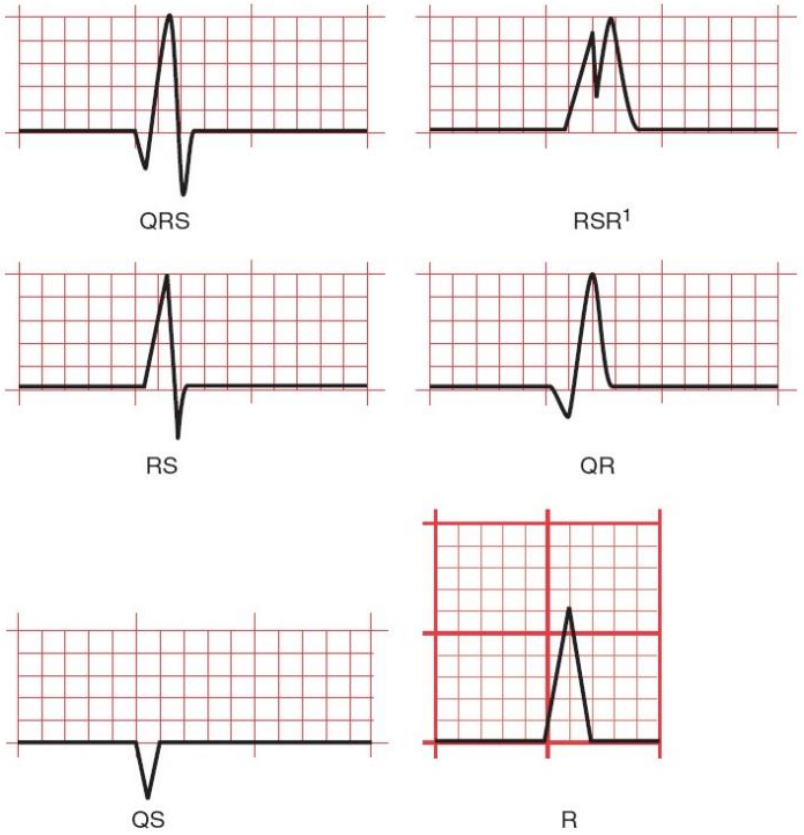




QRS complex configurations

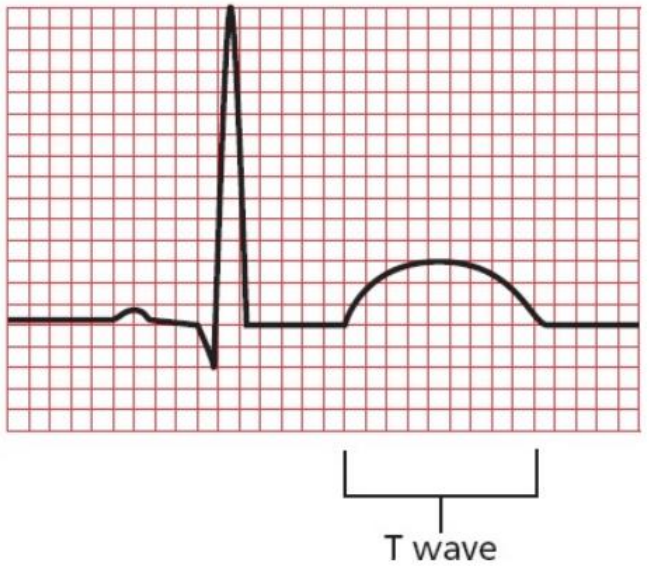
Because the precise configuration of the QRS complex can vary so greatly, a standard format for naming each component has been devised

1. If the first deflection is downward, that part of the QRS complex is called a Q wave
2. The first upward deflection is called an R wave
3. If there is a second upward deflection, it is called R′ (“R-prime”)
4. The first downward deflection following an upward deflection is called an S wave. Therefore, if the first wave of the complex is an R wave, the ensuing downward deflection is called an S wave, not a Q wave. A downward deflection can only be called a Q wave if it is the first wave of the complex. Any other downward deflection is called an S wave
5. If the entire configuration consists solely of one downward deflection, the wave is called a QS wave



The basic waves and lines of the standard EKG

- T wave (Repolarization)
  - After myocardial cells depolarize, they pass through a brief refractory period during which they are resistant to further stimulation. [QT segment]
  - They then repolarize; that is, they restore the electronegativity of their interiors so that they can be restimulated. Just as there is a wave of depolarization, there is also a wave of repolarization. This, too, can be seen on the EKG. Ventricular repolarization inscribes a third wave on the EKG, the T wave.
  - Ventricular repolarization is a much slower process than ventricular depolarization. Therefore, the T wave is broader than the QRS complex. Its configuration is also simpler and more rounded, like the silhouette of a gentle hill compared to the sharp, jagged, and often intricate contour of the QRS complex.



Note: There is a wave of atrial repolarization as well, but it coincides with ventricular depolarization and is hidden by the much more prominent QRS complex.

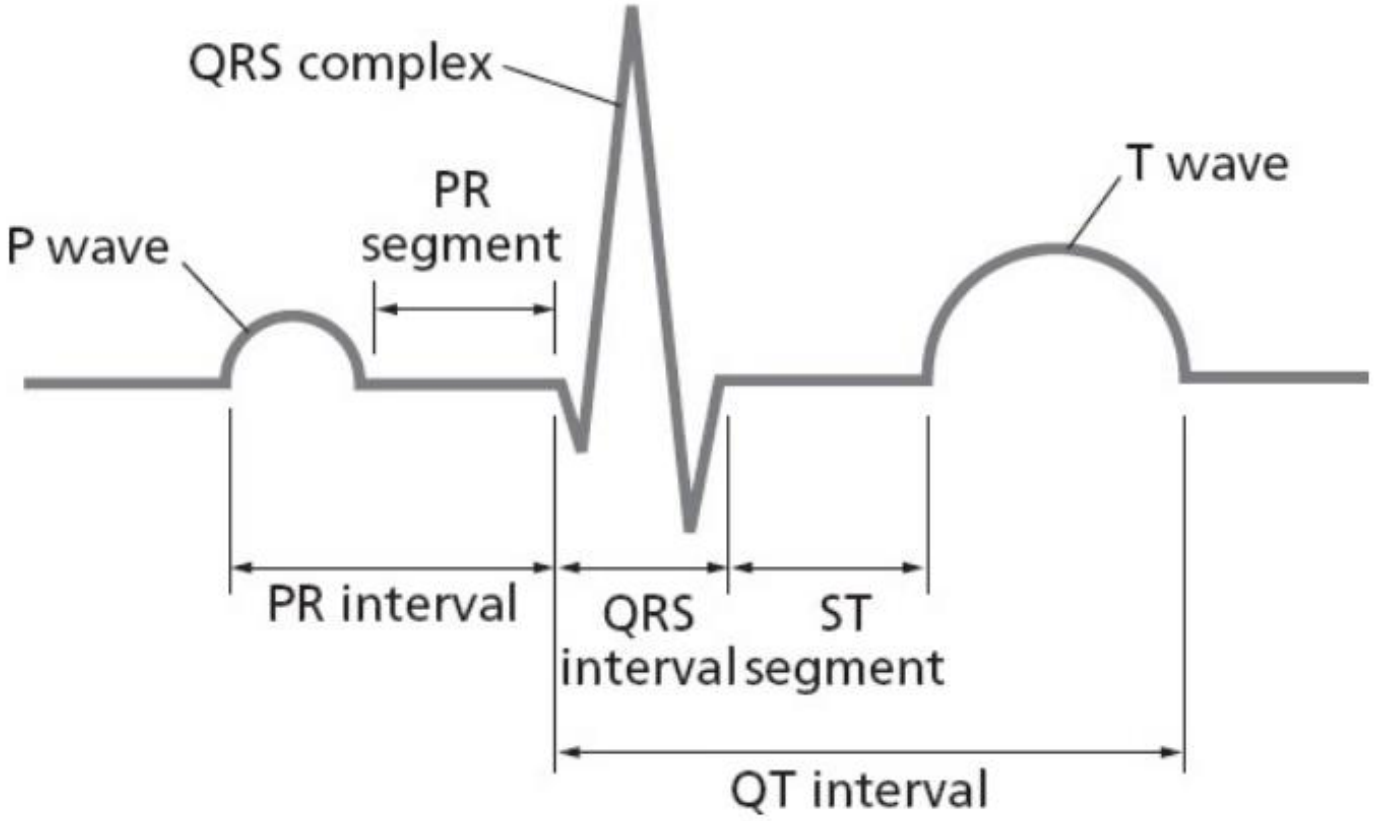
The basic waves and lines of the standard EKG

Various segments and intervals describe the time between these events:

a. The PR interval measures the time from the start of atrial depolarization to the start of ventricular depolarization
b. The PR segment measures the time from the end of atrial depolarization to the start of ventricular depolarization
c. The ST segment records the time from the end of ventricular depolarization to the start of ventricular repolarization
d. The QT interval measures the time from the start of ventricular depolarization to the end of ventricular repolarization
e. The QRS interval measures the time of ventricular depolarization

What differentiates a segment from an interval?

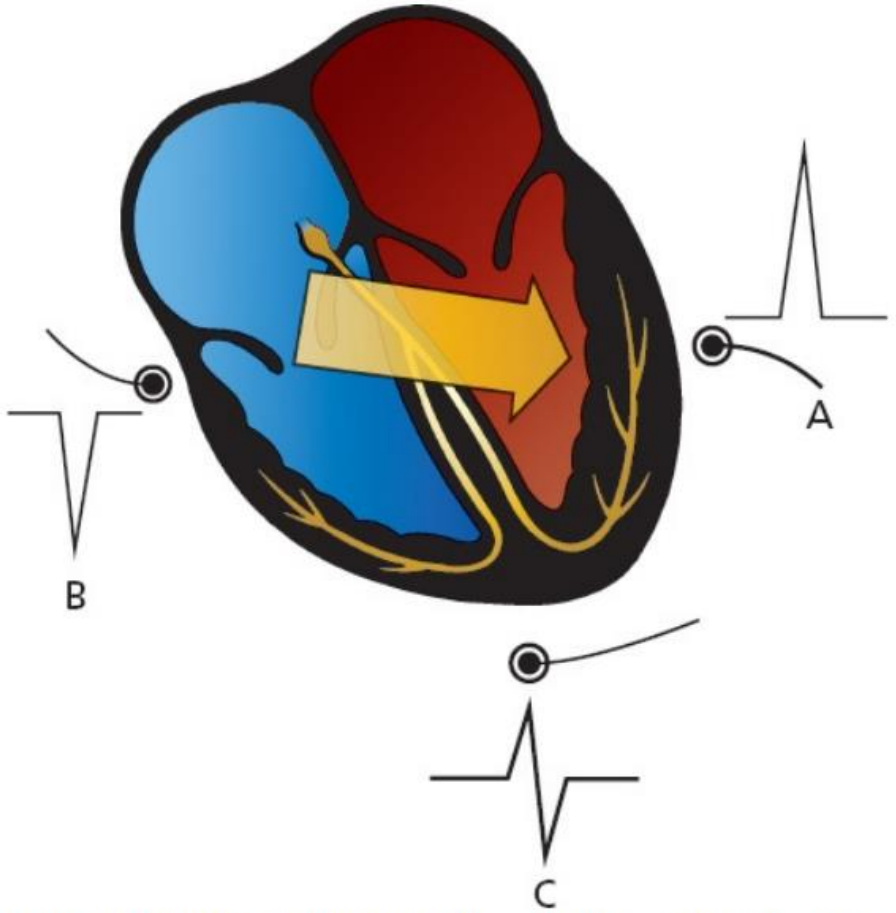
- A **segment** is a straight line connecting two waves
- An **interval** encompasses at least one wave plus, in most instances, the connecting straight line.



Making Waves

Electrodes can be placed anywhere on the surface of the body to record the heart's electrical activity.

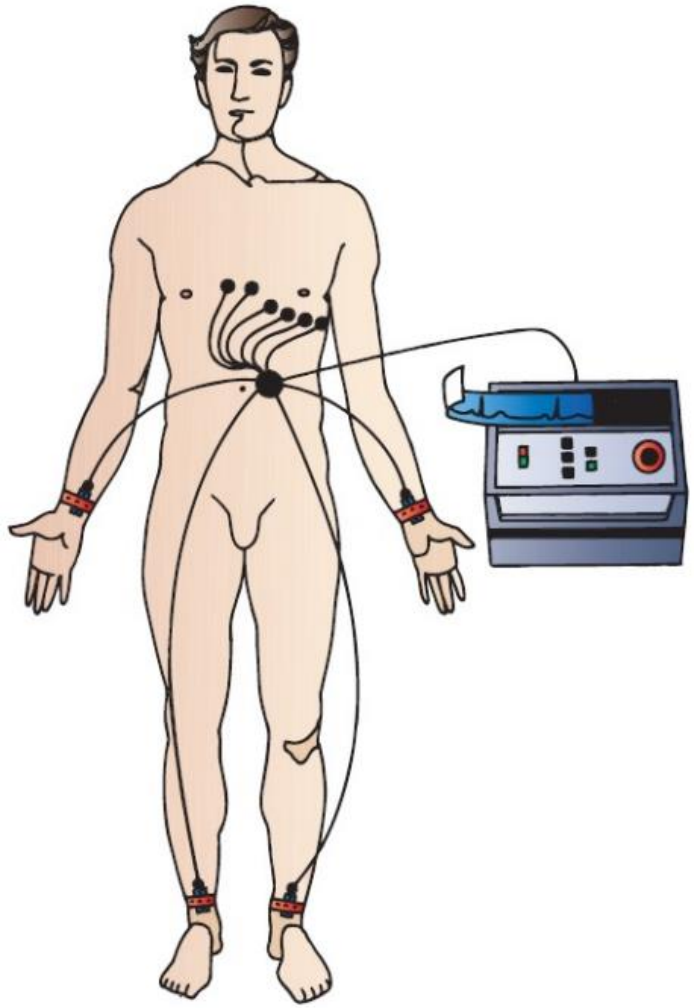
- If a wave of depolarization passing through the heart is moving toward a surface electrode, that electrode will record a positive deflection (electrode A)
- If the wave of depolarization is moving away from the electrode, the electrode will record a negative deflection (electrode B)
- If the wave of depolarization is moving perpendicularly to the electrode, the electrode will record a biphasic wave (electrode C)
- The effects of repolarization are precisely the opposite of those of depolarization, as you would expect



A wave of depolarization moving through the heart (*large arrow*). Electrode *A* records a positive deflection, electrode *B* records a negative deflection, and electrode *C* records a biphasic wave.

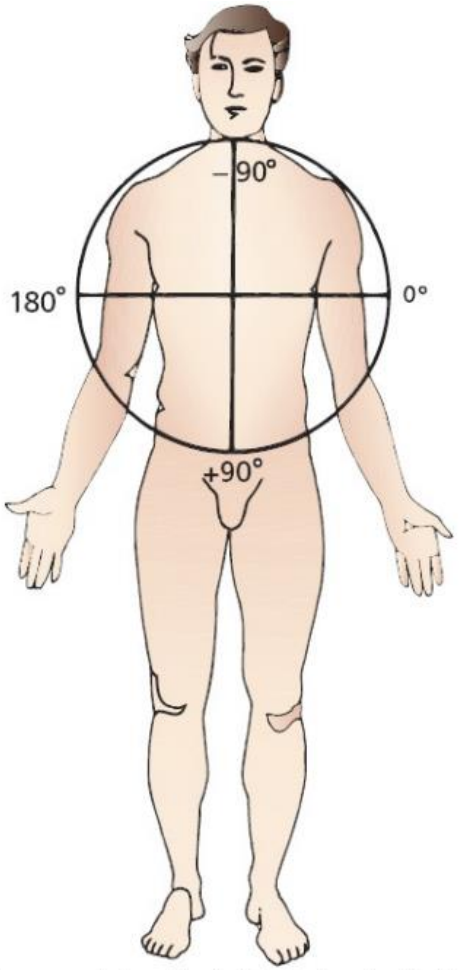
The 12 Views of the Heart

- To prepare a patient for a 12-lead EKG, two electrodes are placed on the arms and two on the legs. These provide the basis for the six limb leads, which include the three standard leads and the three augmented leads.
- Six electrodes are also placed across the chest, forming the six precordial leads.
- The electrical recordings will vary depending on the precise placement of the electrodes. Therefore, adherence to standard positioning protocols is very important to allow comparison between EKGs taken at different times in different settings.



The Six Limb Leads

- The limb leads are created by putting electrodes on all four extremities.
- They view the heart in a vertical plane called the frontal plane.
- The frontal plane can be envisioned as a giant circle superimposed on the patient's body. This circle is then marked off in degrees.
- Each lead has its own specific view of the heart, or angle of orientation. The angle of each lead can be determined by drawing a line from the negative electrode(s) to the positive electrode(s). The resultant angle is then expressed in degrees by superimposing it on the 360° circle of the frontal plane.

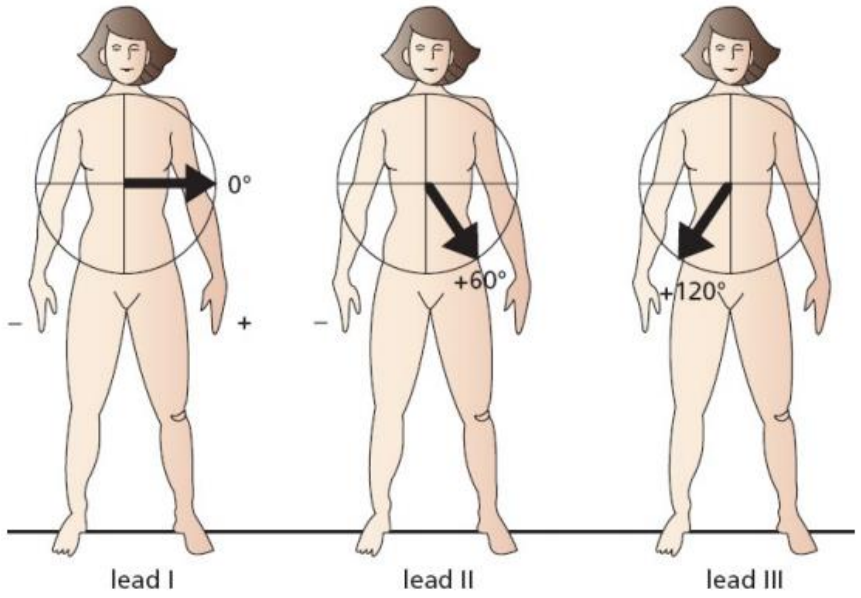


The frontal plane is a coronal plane. The limb leads view electrical forces moving up and down and left and right on the frontal plane.

The Six Limb Leads

The three standard limb leads are defined as follows:

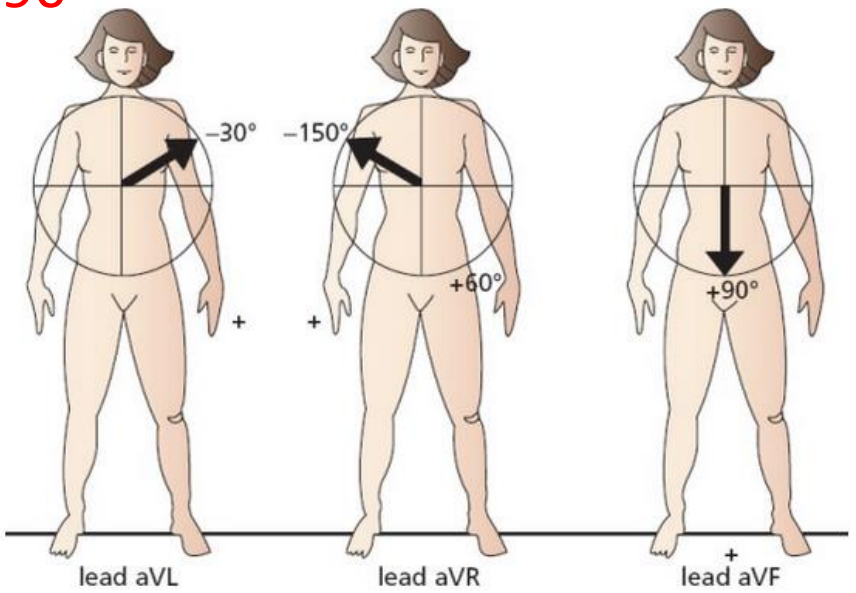
- **Lead I** is created by making the left arm positive and the right arm negative. Its angle of orientation is 0°
- **Lead II** is created by making the legs positive and the right arm negative. Its angle of orientation is 60°
- **Lead III** is created by making the legs positive and the left arm negative. Its angle of orientation is 120°



The Six Limb Leads

The three augmented limb leads are defined as follows:

- **Lead aVL** is created by making the left arm positive and the other limbs negative. Its angle of orientation is −30°
- **Lead aVR** is created by making the right arm positive and the other limbs negative. Its angle of orientation is −150°
- **Lead aVF** is created by making the legs positive and the other limbs negative. Its angle of orientation is +90°

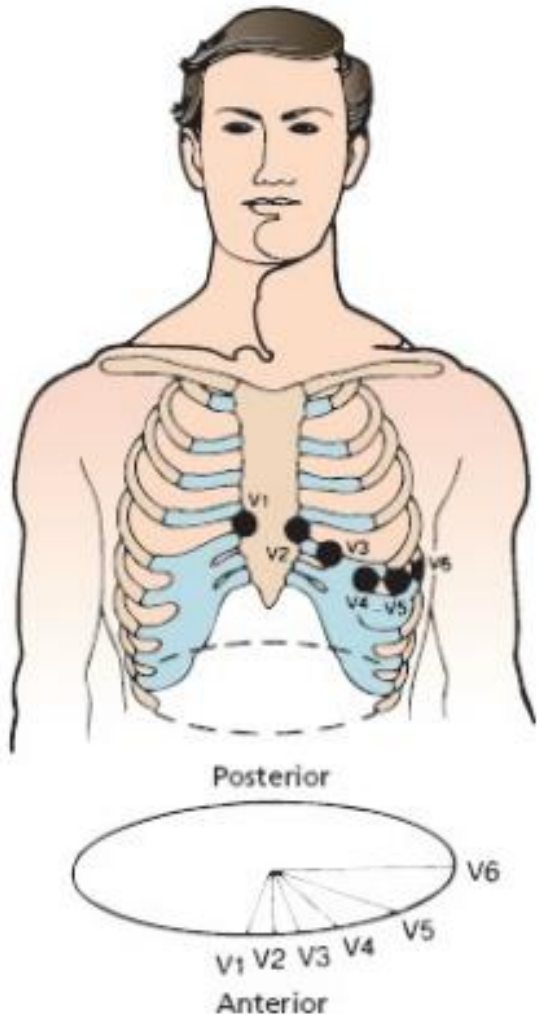


Note: They are called augmented leads because the EKG machinery must amplify the tracings to get an adequate recording.

The Six Precordial Leads

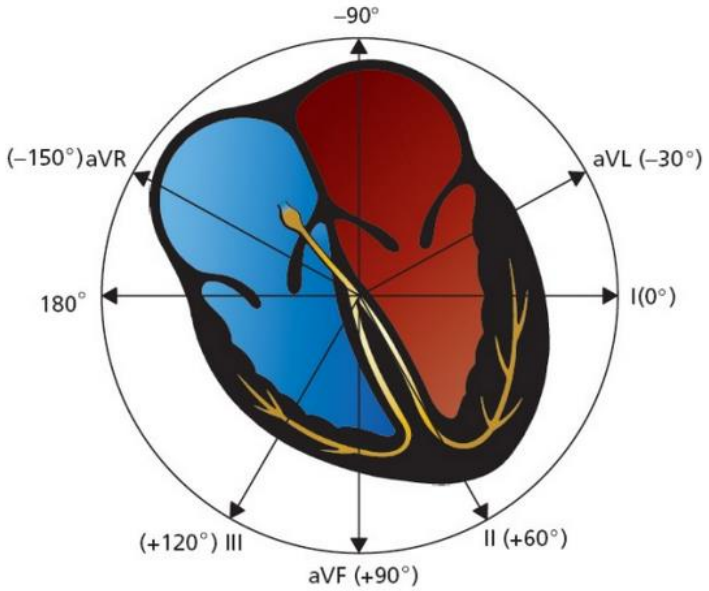
The six positive electrodes are positioned as follows:

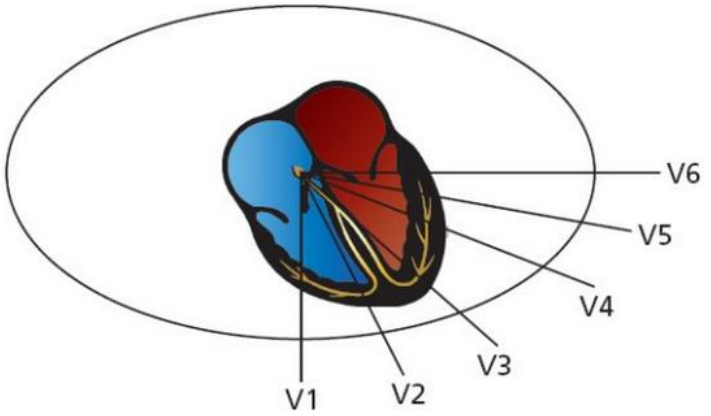
- V1 is placed in the fourth intercostal space to the right of the sternum.
- V2 is placed in the fourth intercostal space to the left of the sternum.
- V3 is placed between V2 and V4.
- V4 is placed in the fifth intercostal space in the midclavicular line.
- V5 is placed between V4 and V6.
- V6 is placed in the fifth intercostal space in the midaxillary line



Leads Grouping

Group	Leads
Anterior	V2, V3, V4
Left lateral	I, aVL, V5, V6
Inferior	II, III, aVF
Right ventricular	aVR, V1





Keep in mind

❖What Happens If You Misplace the Electrodes?

- Correct placement of electrodes during an EKG is crucial for accurate results. Reversing limb electrodes or misplacing precordial leads can lead to distorted readings, potentially misdiagnosing a healthy patient. Careful electrode placement is essential, especially in challenging situations such as dealing with obese patients or those with large breasts. Neglecting proper adherence of electrodes due to hair can be a common mistake, and it's emphasized to shave the area if needed. Up to 4% of EKGs may have incorrect lead placement due to carelessness or time constraints in fast-paced healthcare settings.

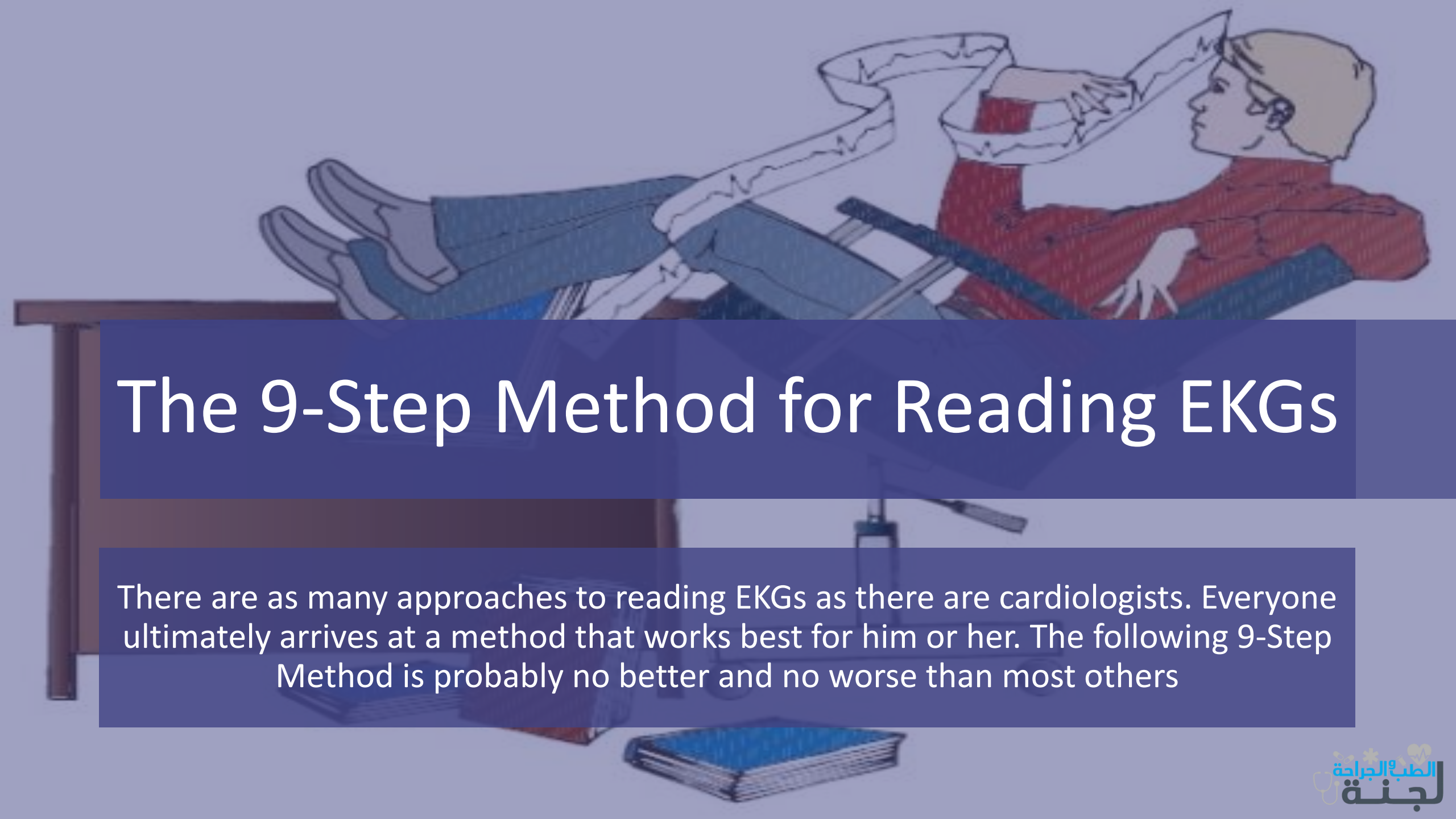
❖Does My Patient Have To Be Lying Down?

- Patients undergoing an EKG should ideally lie flat for accurate results. While exceptions may exist, such as patients with heart failure or neck pain, efforts should be made to keep them as flat as possible. Using a pillow or slightly elevating the head is acceptable, but excessive elevation should be avoided. Body posture changes can impact heart position and affect voltage measurements on the EKG. Subtle changes like variations in ST segments and new Q waves can be crucial in diagnosing a myocardial infarction.

The Normal 12-Lead EKG

Orientation of the Waves of the Normal EKG

- The P wave is small and usually positive in the left lateral and inferior leads. It is often biphasic in leads III and V1. It is usually most positive in lead II and most negative in lead aVR
- The QRS complex is large, and tall R waves (positive deflections) are usually seen in most left lateral and inferior leads. R-wave progression refers to the sequential enlargement of R waves as one proceeds across the precordial leads from V1 to V5. A small initial Q wave, representing septal depolarization, can often be seen in one or several of the left lateral leads, and sometimes in the inferior leads
- The T wave is variable, but it is usually positive in leads with tall R waves



The 9-Step Method for Reading EKGs

There are as many approaches to reading EKGs as there are cardiologists. Everyone ultimately arrives at a method that works best for him or her. The following 9-Step Method is probably no better and no worse than most others

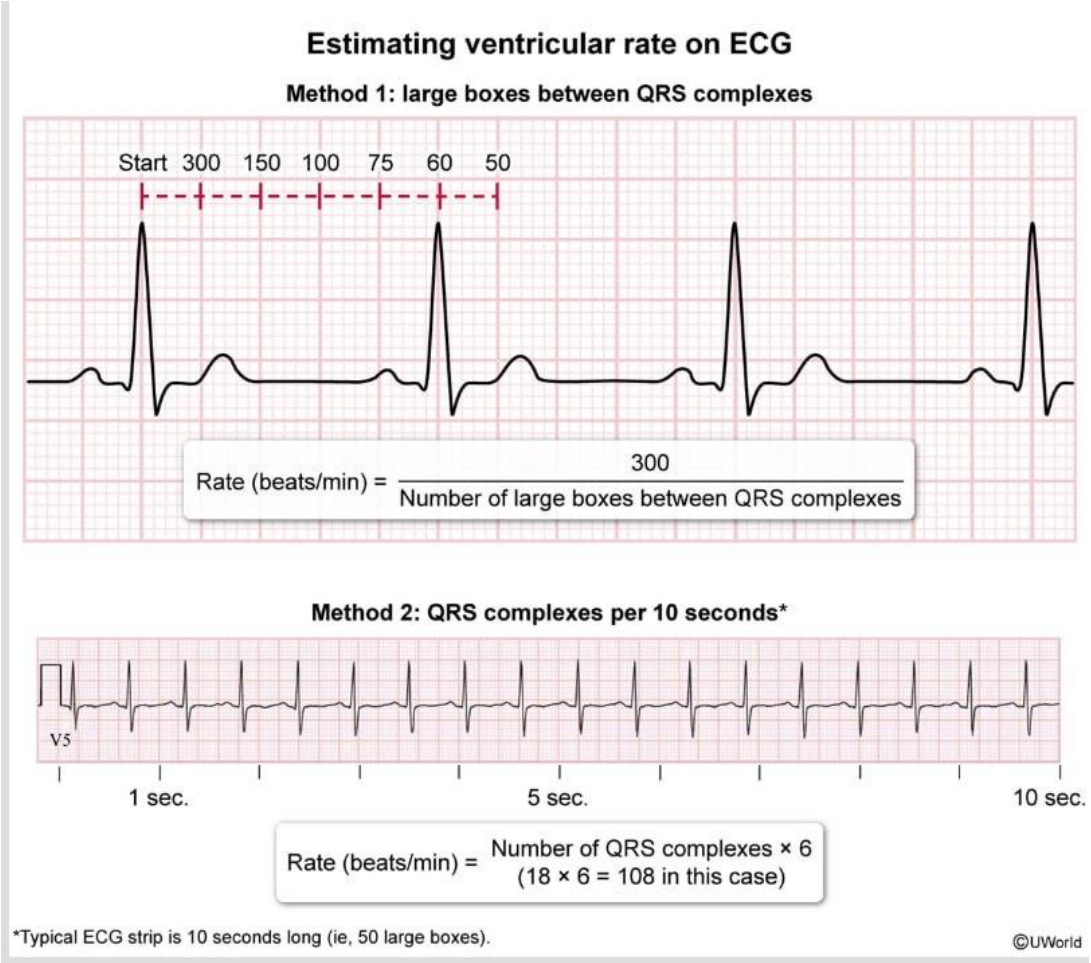
The 9-Step Method for Reading EKGs

We will first list down the steps and then start to discuss them one by one

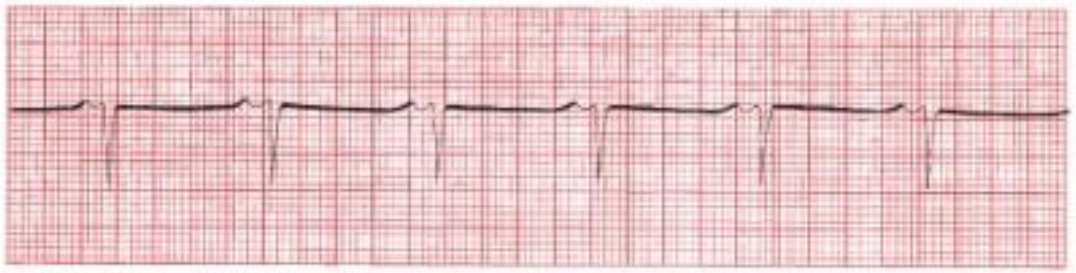
1. Determine the heart rate
2. Measure the length of the PR and QT intervals and the width of the QRS complexes
3. Is the axis of the P waves, QRS complexes, and T waves normal, or is there axis deviation?
4. Rhythm. The Four Questions (Discussed in arrythmia section)
5. Apply the criteria for conduction blocks (AV blocks → BBB → hemiblocks)
6. Apply the criteria for preexcitation
7. Apply the criteria for enlargement and hypertrophy
8. Apply the criteria for coronary artery disease
9. Is there anything on the EKG that suggests one of the other cardiac or noncardiac conditions

Determine the heart rate

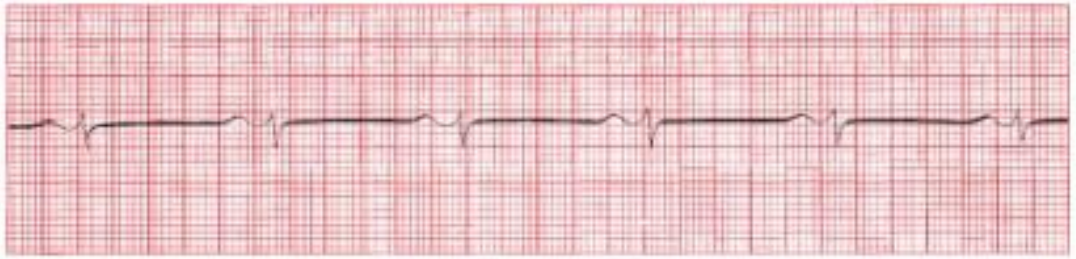
- If the rhythm is regular, we can use the fast method of dividing 300 by the number of large squares between two subsequence R waves or more precisely by dividing 1500 by the number of small squares
- Another way to calculate the heart rate that can also be used for irregular rhythms is the number of R waves in 30 large squares × 10 or more precisely by calculating the number of R waves in 50 large squares × 6



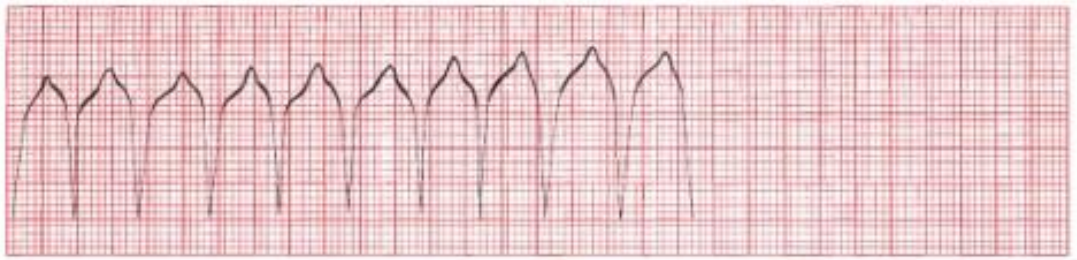
What is the heart rate of the following strips?



A



B



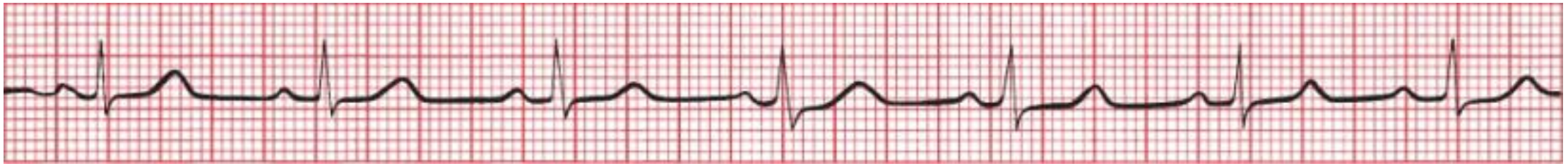
C

(A) About 75 beats per minute

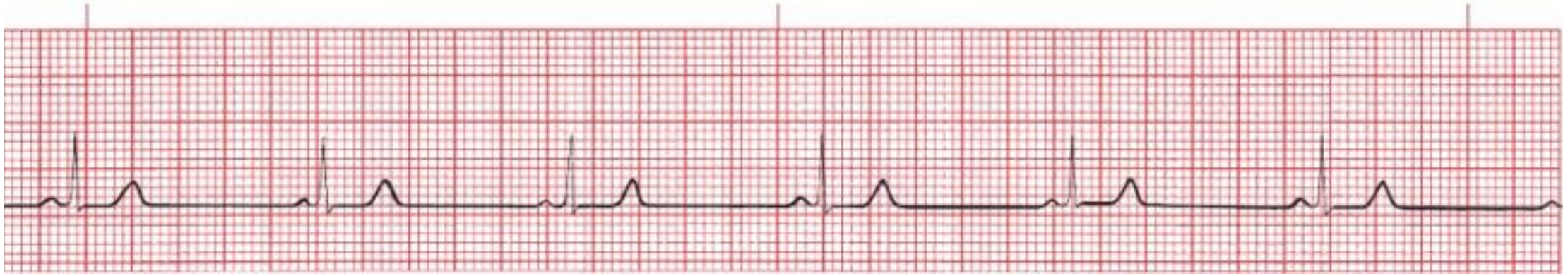
(B) About 60 beats per minute

(C) About 150 beats per minute

What is the heart rate of the following strips?



The R waves are slightly more than four squares apart—let's say four and one-quarter. The rate must therefore be between 60 and 75 beats per minute. If you guess 70, you'll be close. Alternatively, divide 300 by four and one-quarter and get 70.6 beats per minute.



Note the small pink slashes at the top of the rhythm strip marking off 3-second intervals. There are about five and one-half cycles within two of the 3-second intervals. The rate is therefore about 55 beats per minute.

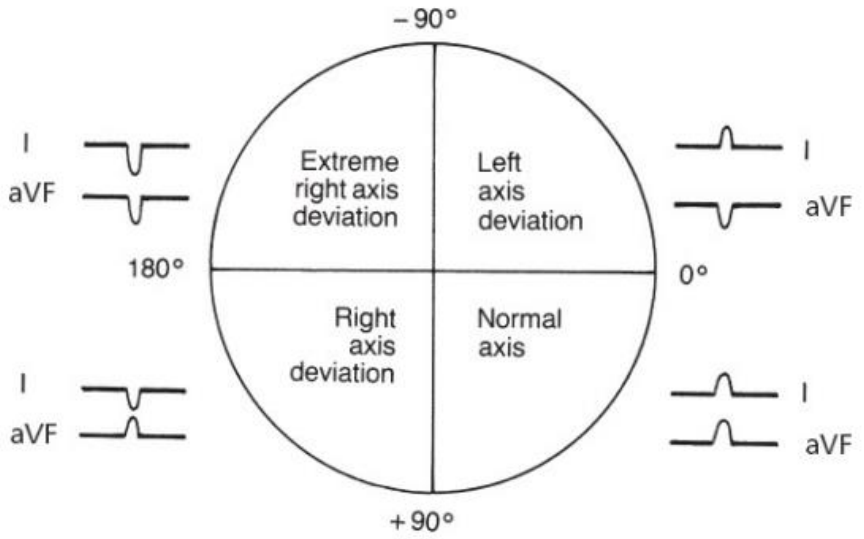
Measure the length of

- **PR interval length**
  - The PR interval normally lasts from 0.12 to 0.2 seconds (3 to 5 mm on the EKG paper).
- **QT interval length**
  - The QT interval constitutes about 40% of each cardiac cycle (R–R interval).
  - The faster the heart beats, the shorter the QT interval.
- **QRS complex width**
  - A normal QRS interval, representing the duration of the QRS complex, is 0.06 to 0.1 seconds in duration (1.5 to 2.5 mm on the EKG paper).

- The DDx of abnormalities is discussed in Miscellaneous Conditions

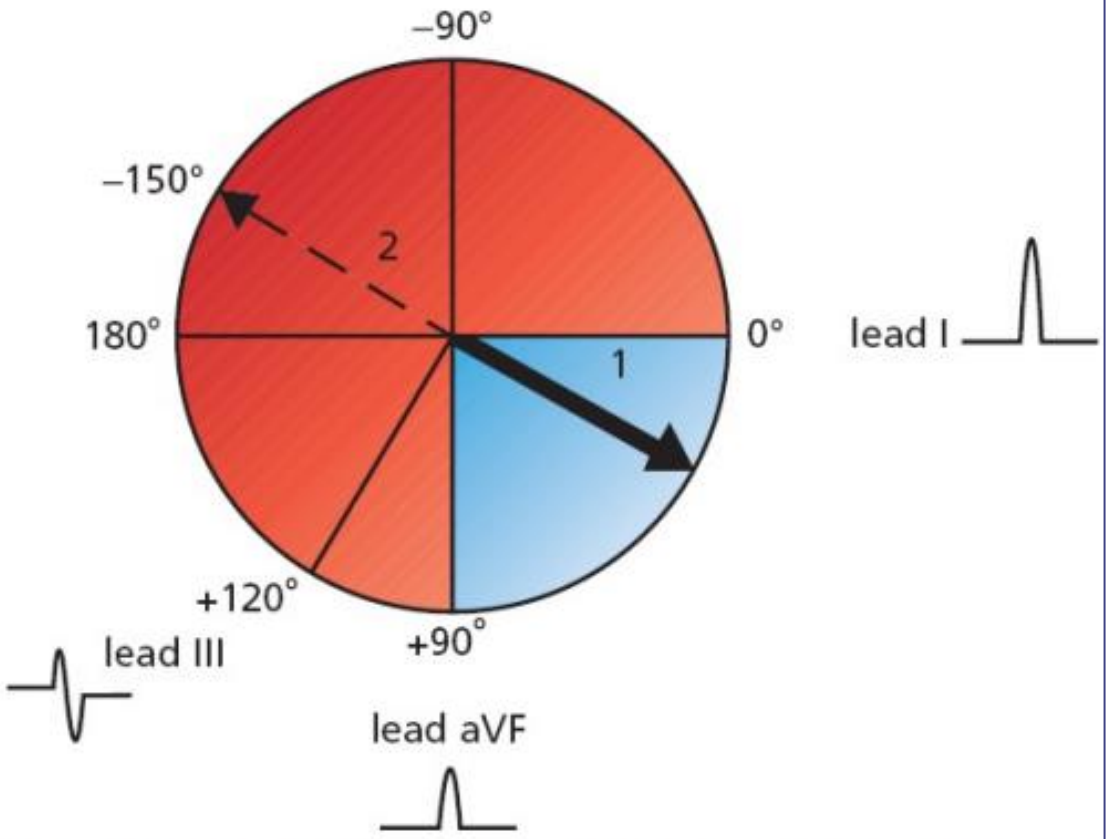
Axis summary

- The term axis refers to the direction of the mean electrical vector, representing the average direction of current flow. It is defined in the frontal plane only
- To determine the axis of any wave, find the lead in which the wave is most nearly biphasic. The axis must lie approximately perpendicular to that lead
- A quick estimate of the axis can be made by looking at leads I and aVF: → →
- Things might get clearer with the examples in the next 2 slides

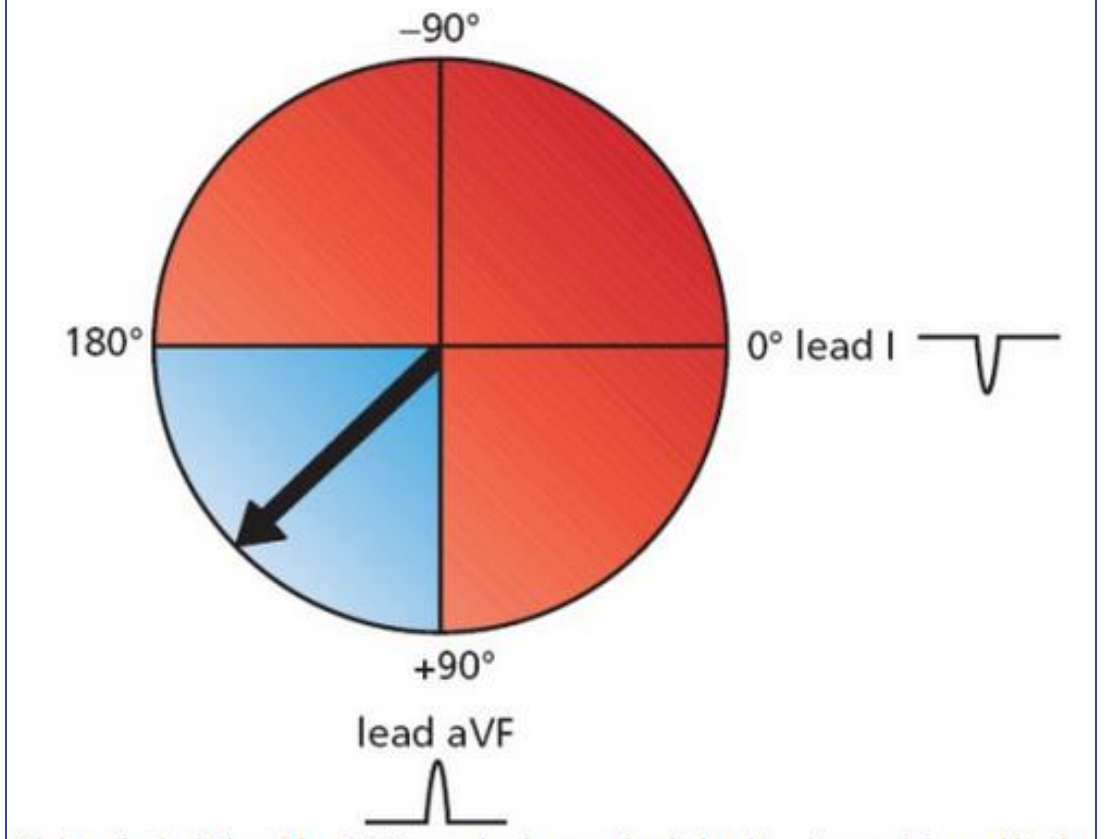


Axis	Lead I	Lead aVF
Normal axis	Positive	Positive
Left axis deviation	Positive	Negative
Right axis deviation	Negative	Positive
Extreme right axis deviation	Negative	Negative

Axis summary

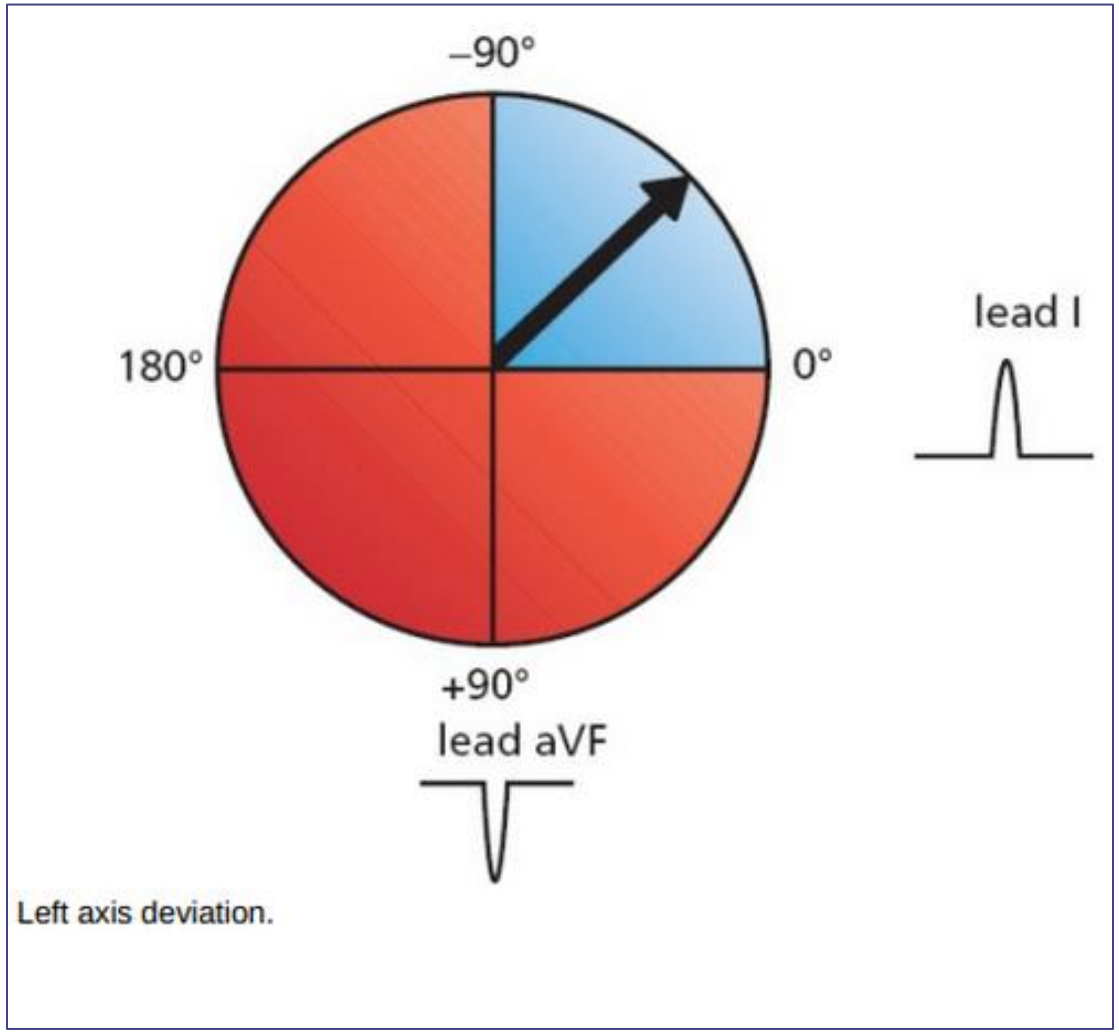


QRS complexes are shown for leads I, III, and aVF. Determining the axis is easy. The QRS complex in lead III is biphasic. The axis therefore must be either +30° or −150°. However, because the QRS complex is positive in both leads I and aVF, the axis must be normal; that is, it must lie within the shaded quadrant. The axis therefore can only be +30°.

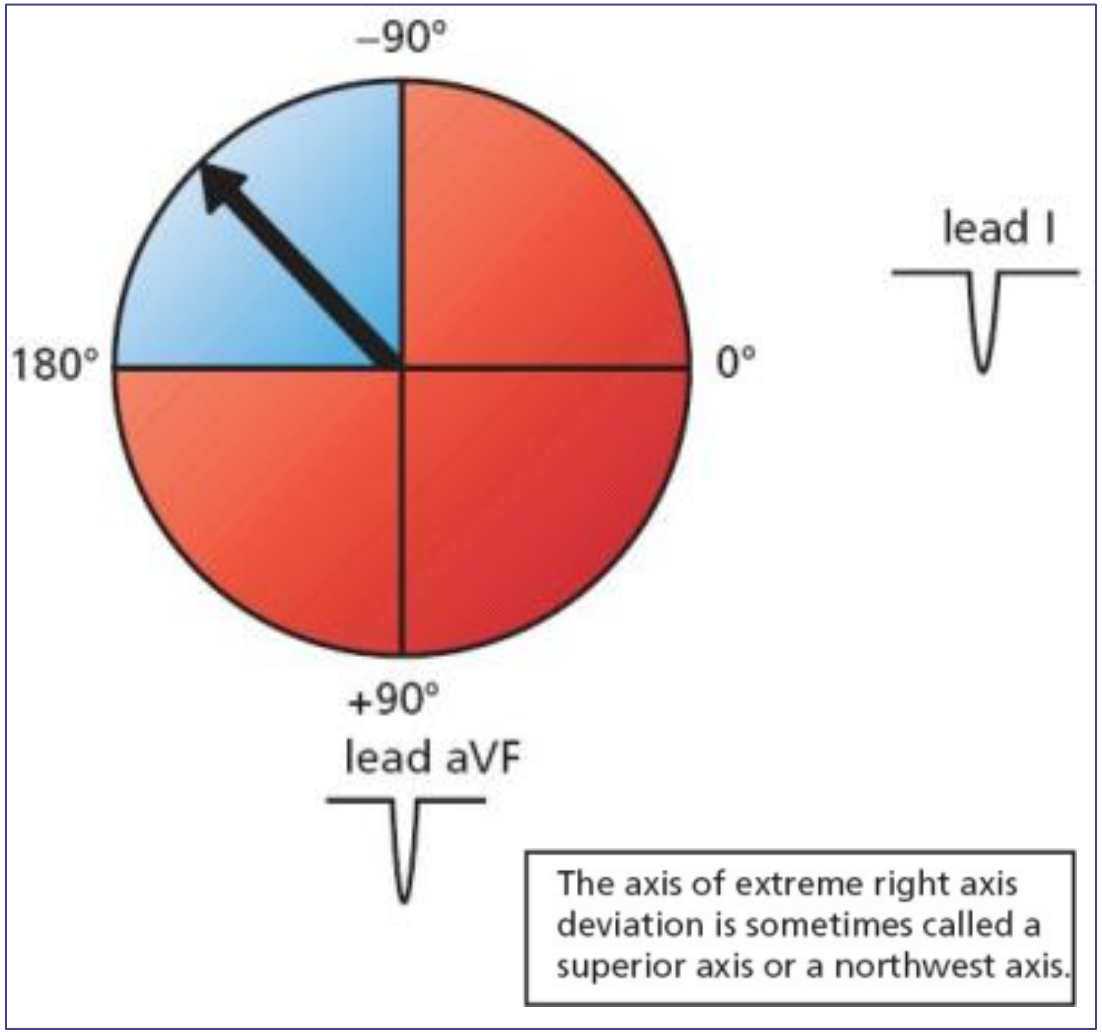


Right axis deviation. The QRS complex is negative in lead I, whereas it is positive in aVF.

Axis summary



Left axis deviation.



The axis of extreme right axis deviation is sometimes called a superior axis or a northwest axis.

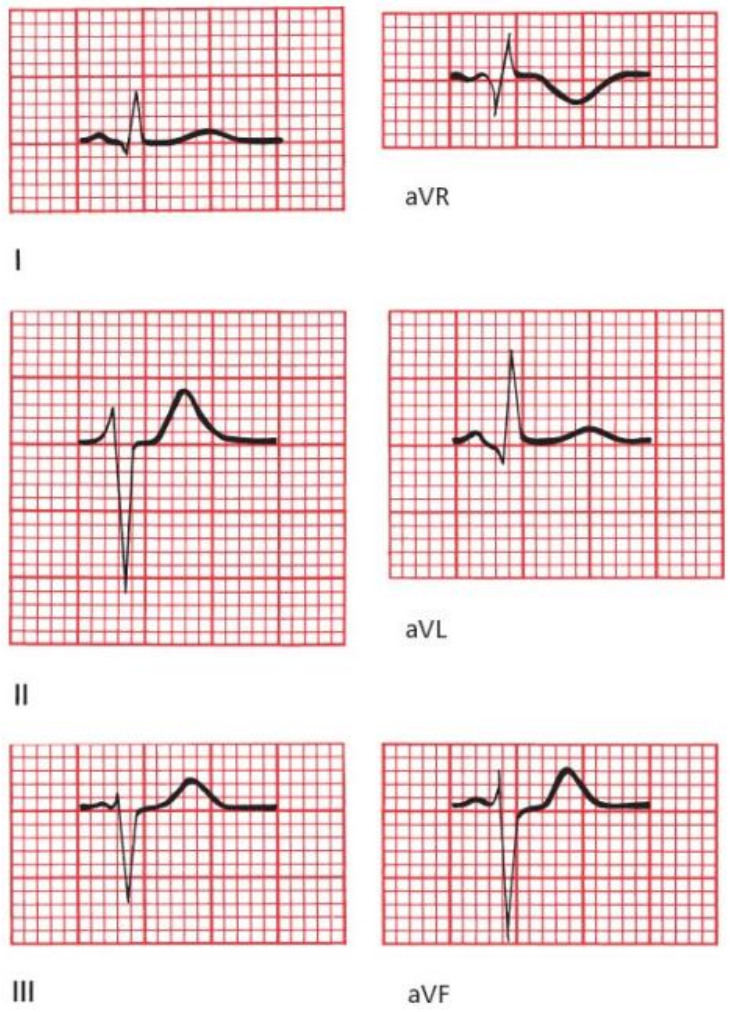
Exercise 1

Is the QRS axis normal, or is there axis deviation?

- This patient has left axis deviation; the QRS complex is predominantly positive in lead I and negative in lead aVF

Now, can you define the axis more precisely by finding the lead with a biphasic QRS complex?

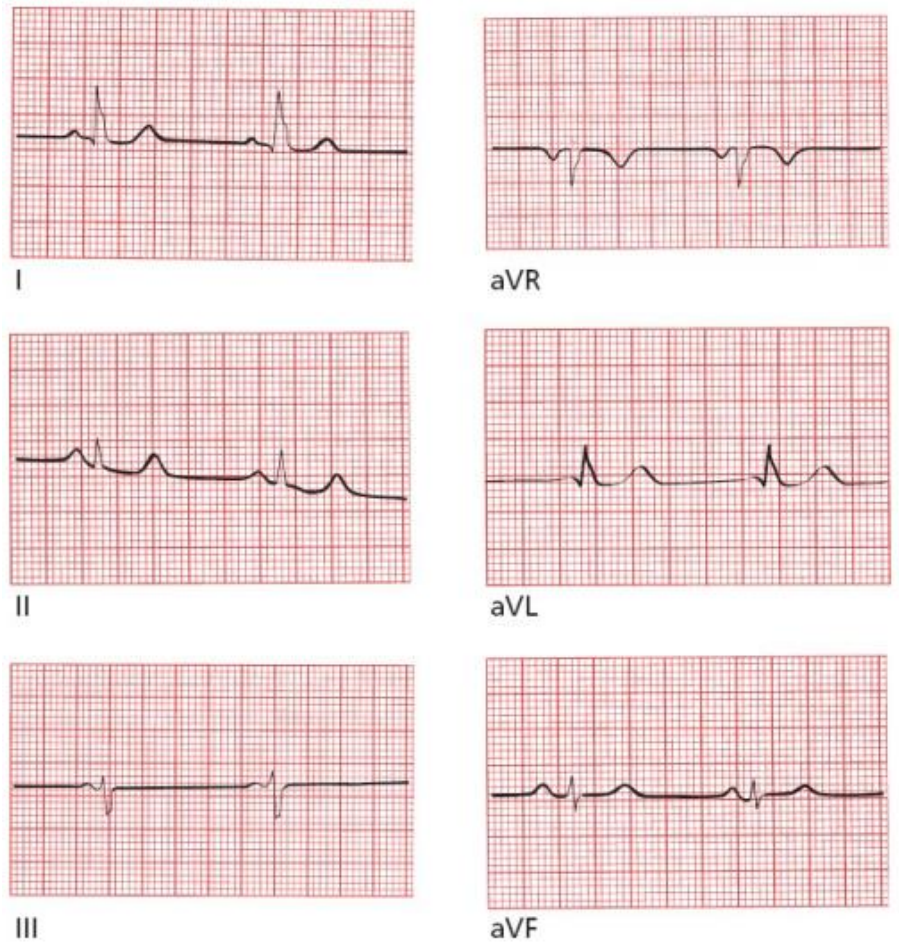
- The QRS complex in lead aVR is approximately biphasic; therefore, the electrical axis must lie nearly perpendicular to it, that is, at either −60° or +120°. Because we already know that the axis falls within the zone of left axis deviation (i.e., between 0° and −90°), the correct axis must be −60°.



Exercise 2

Can you identify the axis of the QRS complex, P wave, and T wave on the following EKG?

- **The QRS complex**: the QRS axis is about 0°. It is nearly biphasic in aVF, implying an axis of 0° or 180°. Because the QRS complex in lead I has a tall R wave, the axis must be 0°.
- **The P wave**: in lead aVL, the P wave is virtually invisible (isoelectric), so the P-wave axis must lie perpendicular to this lead and is either 60° or −120°. Since the P wave is positive in leads I and aVF, the axis must be 60°.
- **The T wave**: all of the leads with tall R waves have positive T waves. The T waves are flat in lead III, indicating an axis perpendicular to lead III (either +30° or −150°). Because there is a tall T wave in lead I, the axis must be about +30°.



Rhythm and Arrythmias

Rhythm

Sinus Rhythm

1. Normal Sinus Rhythm
2. Arrhythmias of sinus origin (The electrical activity follows the usual conduction pathways we have already outlined, starting with depolarization of the sinus node, but it is too fast, too slow, or irregular)

Tachyarrhythmia

- Are either ectopic rhythms or reentrant arrhythmias
- Subdivided into supraventricular (atrial and junctional arrythmias) and ventricular arrythmias

Bradyarrhythmia

- The electrical activity originates in the sinus node and follows the usual pathways but encounters unexpected blocks and delays.
- Heart blocks are subdivided to
  1. AV block
  2. Bundle branch block
  3. Hemiblock

Preexcitation

- The electrical activity follows anomalous accessory conduction pathways that bypass the normal ones, providing an electrical shortcut, or short circuit.
- The most important syndrome being Wolf-Parkinson-White syndrome

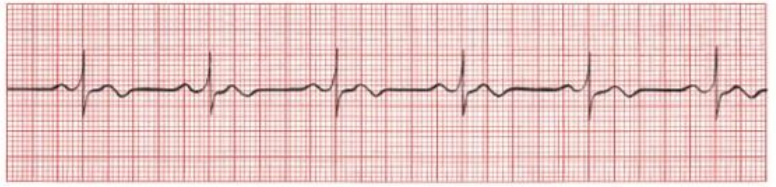
Arrhythmias of Sinus Origin

❖Normal sinus rhythm

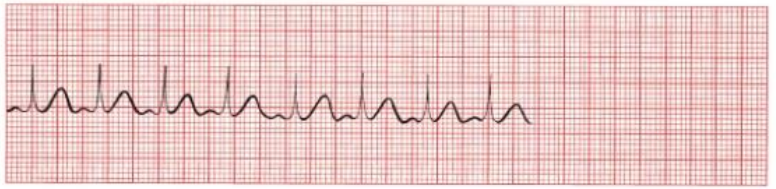
- Depolarization originates spontaneously within the sinus node. The rate is regular and between 60 and 100 beats per minute.
- **Sinus tachycardia**: Rhythm speeds up beyond 100
- **Sinus bradycardia**: It slows down below 60

❖Sinus Arrhythmia

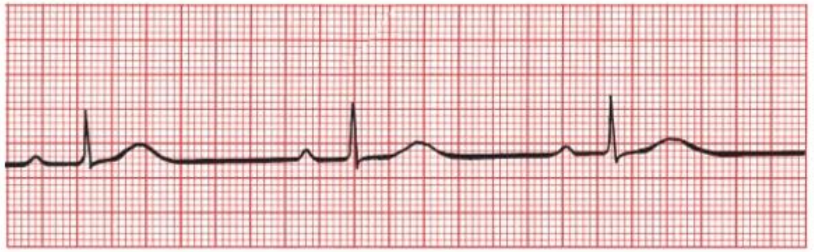
- Sometimes, the EKG shows a rhythm that seems like normal sinus rhythm but is slightly irregular, known as sinus arrhythmia. It's a normal occurrence linked to the natural variation in heart rate during breathing. This effect can be subtle or, in rare cases, pronounced enough to mimic more concerning irregular heartbeats. The heart rate speeds up during inspiration and slows down during expiration.



Normal sinus rhythm

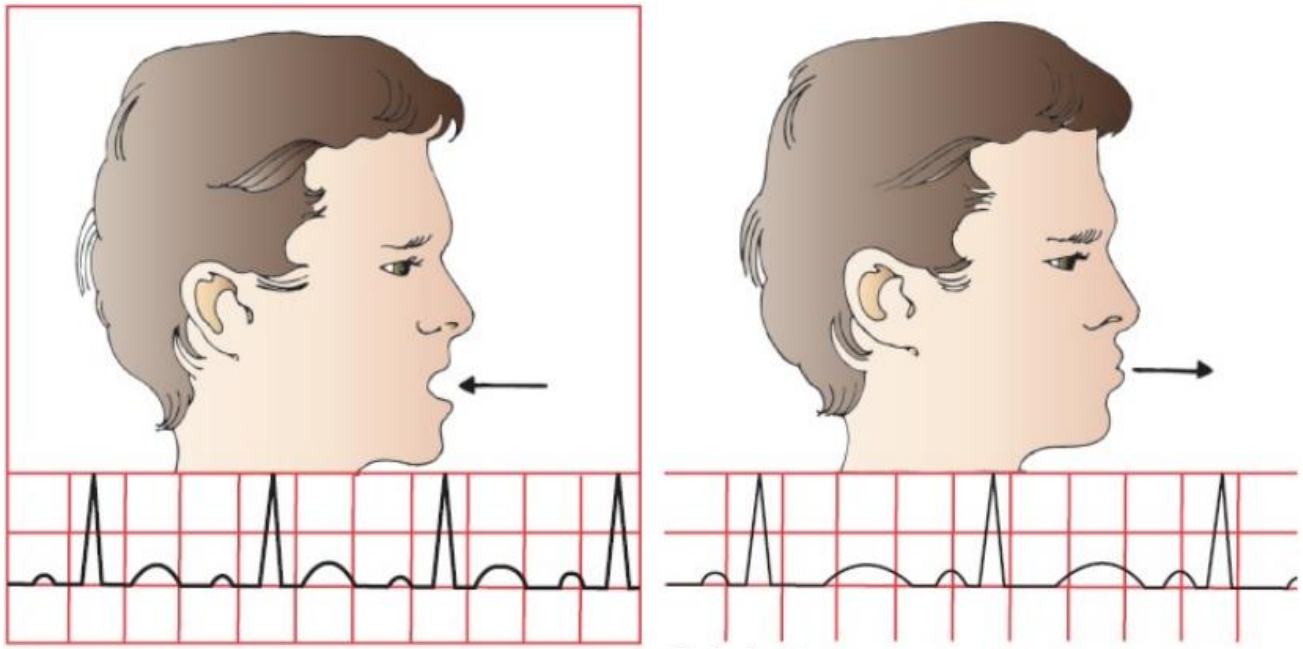


Sinus tachycardia

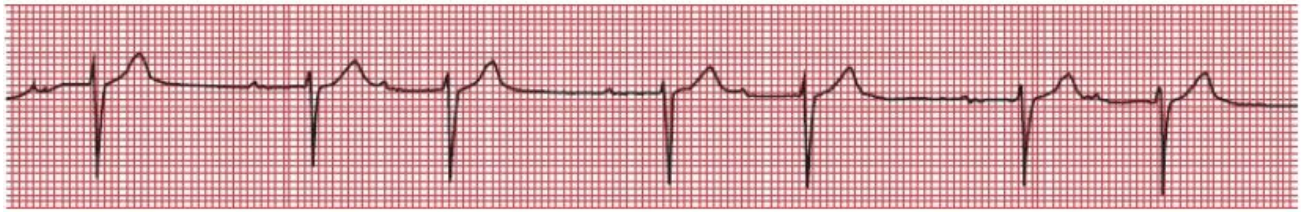


Sinus bradycardia

Sinus Arrhythmia



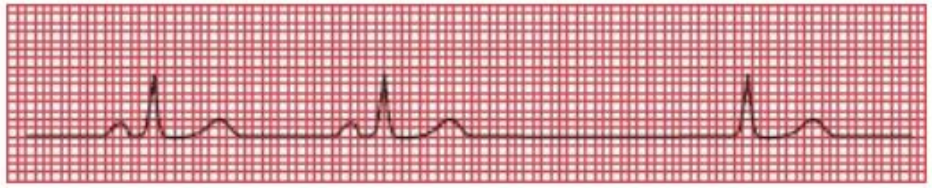
Sinus arrhythmia. The heart rate accelerates with inspiration and slows with expiration.



Note: A loss of sinus arrhythmia may be caused by diminished autonomic feedback to the sinus node. It is therefore often seen in patients with diabetes mellitus, which over time can cause an autonomic neuropathy. Sinus arrhythmia can also be diminished with aging, with obesity, and in patients with long-standing hypertension.

Sinus Arrest, Asystole, and Escape Beats

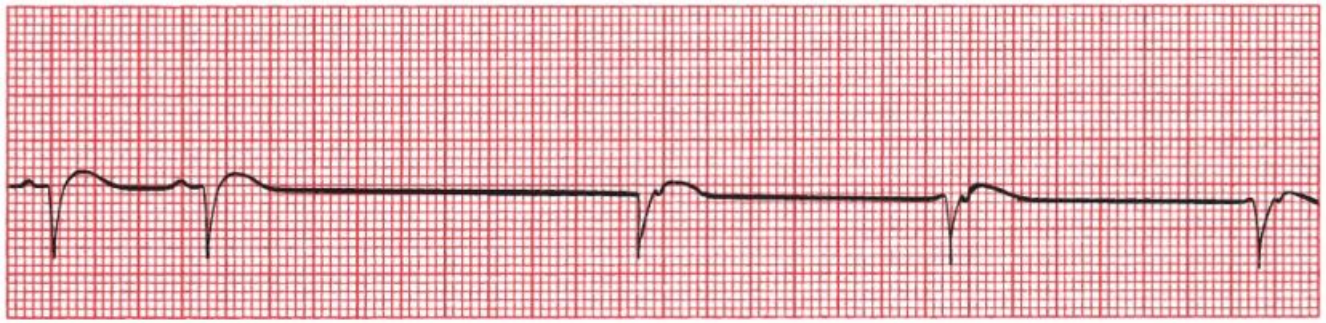
- Sinus arrest happens when the sinus node stops firing, leading to a flat EKG line and potential death. Prolonged electrical inactivity is known as asystole. Fortunately, most heart cells can act as pacemakers. Normally, the sinus node is the fastest pacemaker, but in sinus arrest, other pacemakers (escape beats) can kick in to rescue the heart
- Like the sinus node, which typically fires between 60 and 100 times each minute, the other potential pacemaker cells of the heart have their own intrinsic rhythm. Atrial pacemakers usually discharge at a rate of 60 to 75 beats per minute. Pacemaker cells located near the AV node, called junctional pacemakers, typically discharge at 40 to 60 beats per minute. Ventricular pacemaker cells usually discharge at 30 to 45 beats per minute.



Sinus arrest occurs after the second beat—note the long pause. The third beat, restoring electrical activity, has no P wave. This beat is called a junctional escape beat, which we will explain in the very next section.

Sinus Arrest, Asystole, and Escape Beats

- Of all of the available escape mechanisms, junctional escape is by far the most common.
- Junctional escape involves depolarization near the AV node, leading to an absence of the usual atrial depolarization pattern. Typically, there's no P wave, but occasionally a retrograde P wave may appear, indicating atrial depolarization moving backward from the AV node into the atria. The retrograde P wave has a reversed mean electrical axis compared to the normal P wave.



Junctional escape. The first two beats are normal sinus beats with a normal P wave preceding each QRS complex. There is then a long pause followed by a series of three junctional escape beats occurring at a rate of 40 to 45 beats per minute. Retrograde P waves can be seen buried in the early portion of the T waves (can you

The four questions to assess any rhythm disturbance on the EKG

❖Are Normal P Waves Present?

- If there are normal-appearing P waves with a positive axis in lead II and a negative axis in lead aVR, the arrhythmia likely originates within the atria. If no P waves are present, it probably originated below the atria in the AV node or ventricles. Abnormal P wave axes may indicate activation from a non-sinus atrial focus or retrograde activation from the AV node or ventricles, such as through an accessory pathway connecting the atria and ventricles.

❖Are the QRS Complexes Narrow (0.12 Seconds in Duration)?

- A narrow QRS complex suggests efficient ventricular depolarization through the usual pathways (AV node to His bundle to bundle branches to Purkinje cells). It indicates the rhythm originates at or above the AV node. Conversely, a wide QRS complex suggests ventricular depolarization originates within the ventricles, spreading more slowly. However, the distinction between wide and narrow QRS complexes may not always reliably determine the arrhythmia's origin. We'll see why shortly.

The four questions to assess any rhythm disturbance on the EKG

❖What Is the Relationship Between the P Waves and the QRS Complexes?

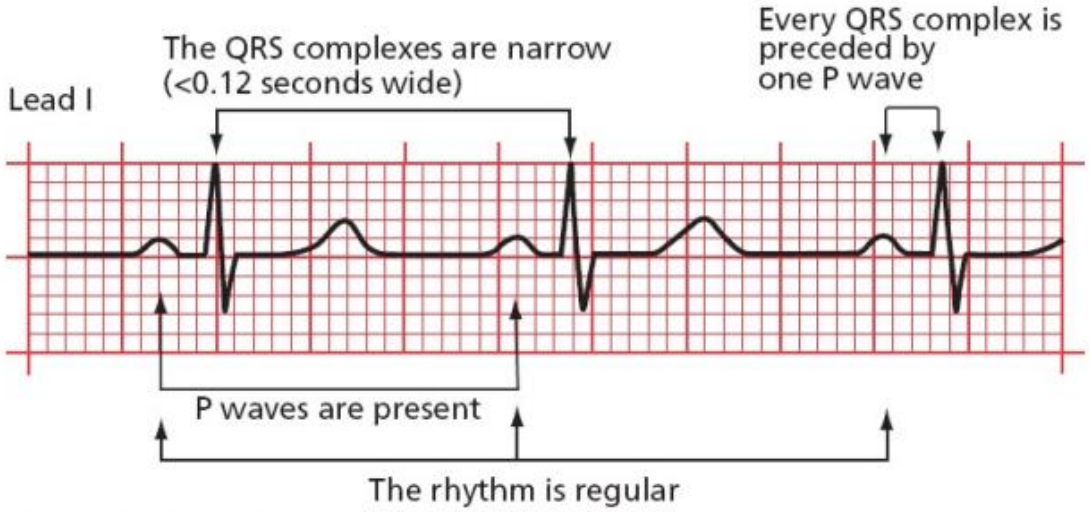
- If each P wave consistently precedes a QRS complex in a one-to-one fashion, the rhythm likely originates from the sinus or another atrial source. However, when the atria and ventricles depolarize independently, leading to a lack of correlation between P waves and QRS complexes, it is termed AV dissociation—a potentially dangerous situation, as the two chambers are not synchronizing properly.

❖Is the Rhythm Regular or Irregular?

- This is often the most immediately obvious characteristic of a particular rhythm and is sometimes the most critical.

The four questions to assess any rhythm disturbance on the EKG

- For the sinus rhythm (normal sinus rhythm and arrythmias of sinus origin), the answers are easy:
  1. Yes, there are normal P waves.
  2. The QRS complexes are narrow.
  3. There is one P wave for every QRS complex.
  4. The rhythm is essentially regular.



Normal sinus rhythm and "The Four Questions" answered.

Tachyarrhythmia

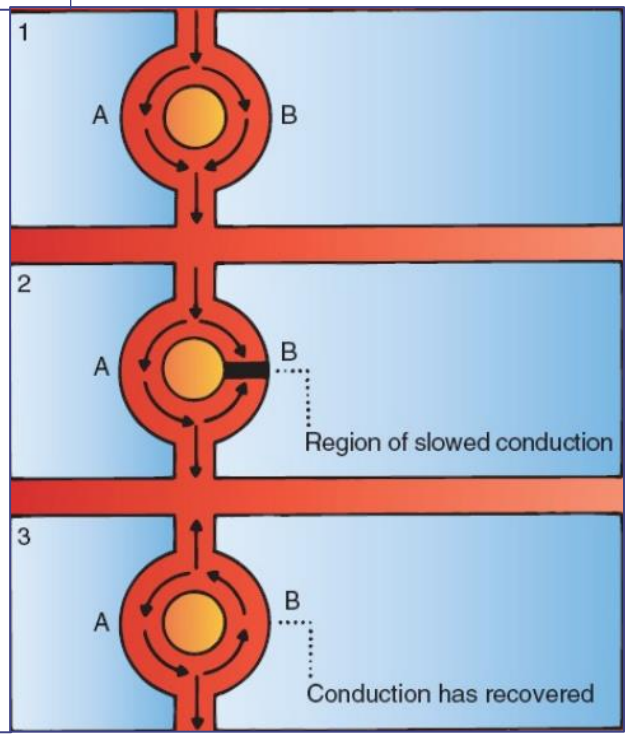
The two major causes of non-sinus arrhythmias are ectopic rhythms and reentrant rhythms

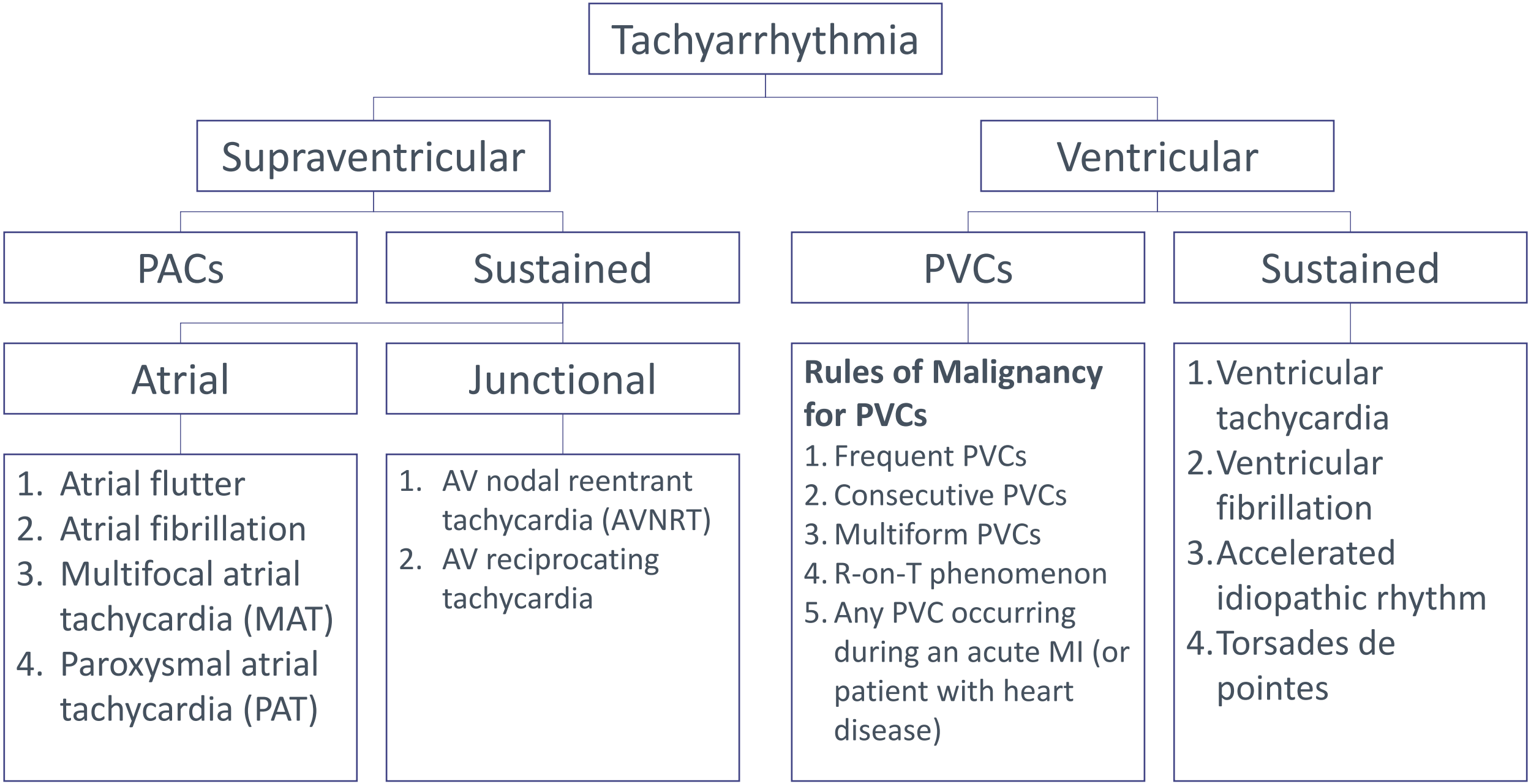
Ectopic rhythms

- Ectopic rhythms are abnormal heart rhythms originating outside the sinus node. They can be single isolated beats or sustained arrhythmias. At the cellular level, they result from increased automaticity (intrinsic pacemaker activity) in a non-sinus node site.
- Normally, the sinus node is the fastest pacemaker, but in abnormal situations, other scattered pacemakers in the heart can accelerate and establish their own ectopic rhythm.
- Causes include digitalis toxicity, beta-adrenergic stimulation from inhalers, caffeine, alcohol, and stimulant drugs like cocaine and amphetamines.

Reentrant rhythms

1. Normally, pathways A and B conduct current equally well.
2. Here, however, conduction through pathway B is temporarily slowed. Current passing down A can then turn back and conduct in a retrograde fashion through B.
3. The reentry loop is established.

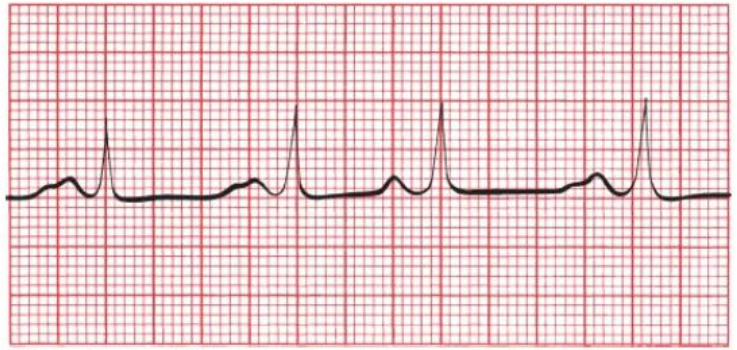




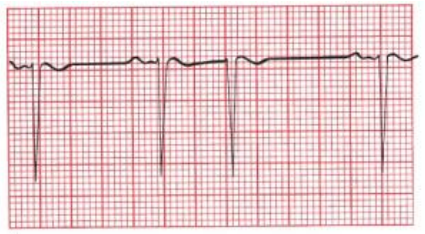
Note: The key to diagnosing a supraventricular tachyarrhythmia is to look for P waves. They are most likely to be prominent in leads II and V1.

Atrial Premature Beats (PACs) and Junctional Premature Beats

- Single ectopic supraventricular beats can originate in the atria or in the vicinity of the AV node
- These are common phenomena, neither indicating underlying cardiac disease nor requiring treatment. They can, however, initiate more sustained arrhythmias
- An atrial premature beat can be distinguished from a normal sinus beat by the contour of the P wave and by the timing of the beat
  - **The contour** of the P wave differs because the atrial premature beat originates from a site distant from the sinus node. If the origin is far from the sinus node, the axis of the P wave will also differ from normal
  - In terms of **timing**, an atrial premature beat occurs too early, interrupting before the expected sinus wave
- With junctional premature beats, there is usually no visible P wave, but sometimes, a retrograde P wave may be seen



The third beat is an atrial premature beat. The P wave is shaped differently from the other, somewhat unusual-looking P waves, and the beat is clearly premature.

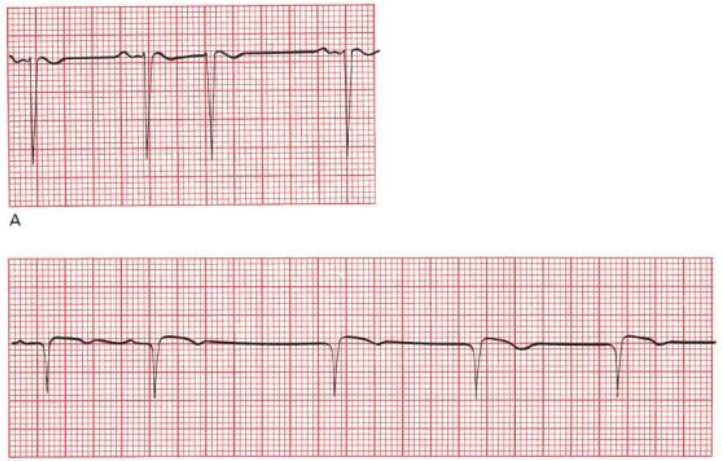


A

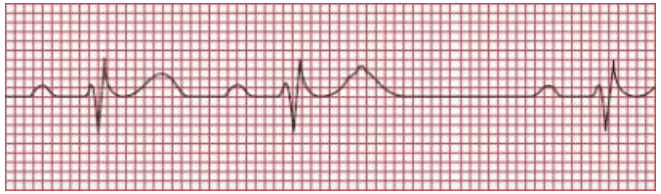
(*A*) A junctional premature beat. The third beat is obviously premature, and there is no P wave preceding the QRS complex.

Atrial Premature Beats (PACs) and Junctional Premature Beats

- What is the difference between a junctional premature beat and a junctional escape beat?
  - They look exactly alike, but the junctional premature beat occurs early, prematurely, interposing itself into the normal sinus rhythm. An escape beat occurs late, following a pause when the sinus node has failed to fire.
- Both atrial and junctional premature beats are usually conducted normally to the ventricles, and the resultant QRS complex is therefore narrow.
- An atrial premature beat may occur so early that the AV node hasn't fully recovered from the previous beat and can't conduct the atrial premature beat into the ventricles. In such cases, the ECG may display only a P wave without a subsequent QRS complex, referred to as a blocked atrial premature contraction.



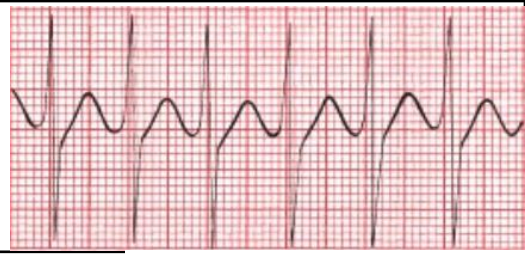
(*A*) A junctional premature beat. The third beat is obviously premature, and there is no P wave preceding the QRS complex. (*B*) The third beat is a junctional escape beat, establishing a sustained junctional rhythm. It looks just like a junctional premature beat, but it occurs late, following a prolonged pause, rather than prematurely.

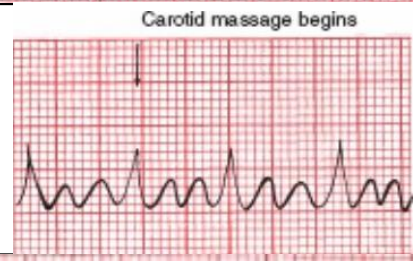


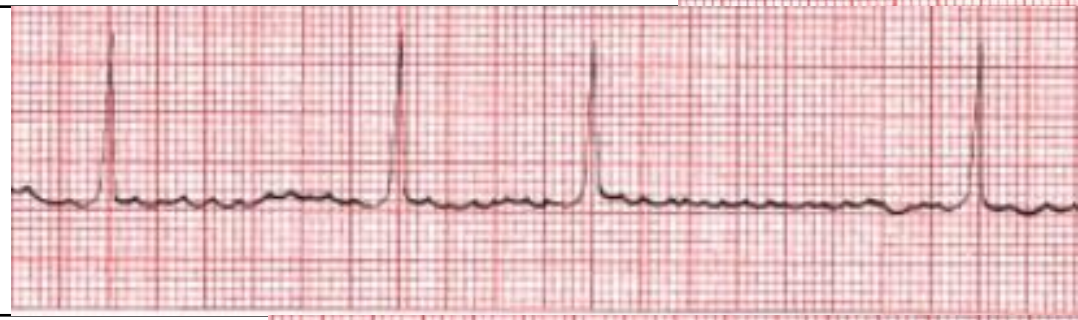
Look at the second beat. The T wave looks deformed, clearly different from the preceding T wave. Why? There is a PAC buried within it. At the time of the PAC, the AV node is still repolarizing and is therefore unable to conduct the PAC into the ventricles. This kind of PAC is called a blocked PAC. There is therefore a pause—no QRS complex or T wave can be generated before the next normal P wave at last comes along and reestablishes normal conduction.

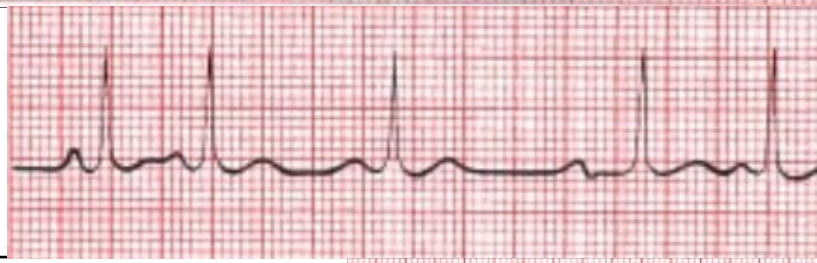
Sustained Supraventricular Arrhythmias

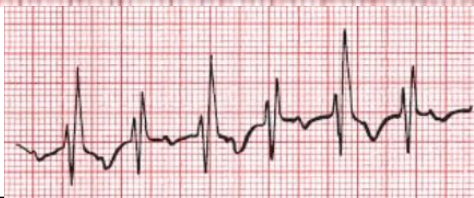
Arrhythmia	Characteristics
AV nodal reentrant tachycardia	• Regular • P waves are retrograde if visible • Rate: 150–250 bpm • Carotid massage: slows or terminates
Atrial Flutter	• Regular, saw-toothed 2:1, 3:1, 4:1, etc., block • Atrial rate: 250–350 bpm • Ventricular rate: one-half, one-third, one-quarter, etc., of atrial rate • Carotid massage: increases block
Atrial Fibrillation	• Irregular • Undulating baseline • Atrial rate: 350–500 bpm • Ventricular rate: variable • Carotid massage: may slow ventricular rate
Multifocal atrial tachycardia	• Irregular • At least three different P-wave morphologies • Rate: 100–200 bpm; sometimes • Carotid massage: no effect
Paroxysmal atrial tachycardia	• Regular Rate: 100–200 bpm • Characteristic warm-up period in the automatic form • Carotid massage: no effect, or only mild slowing





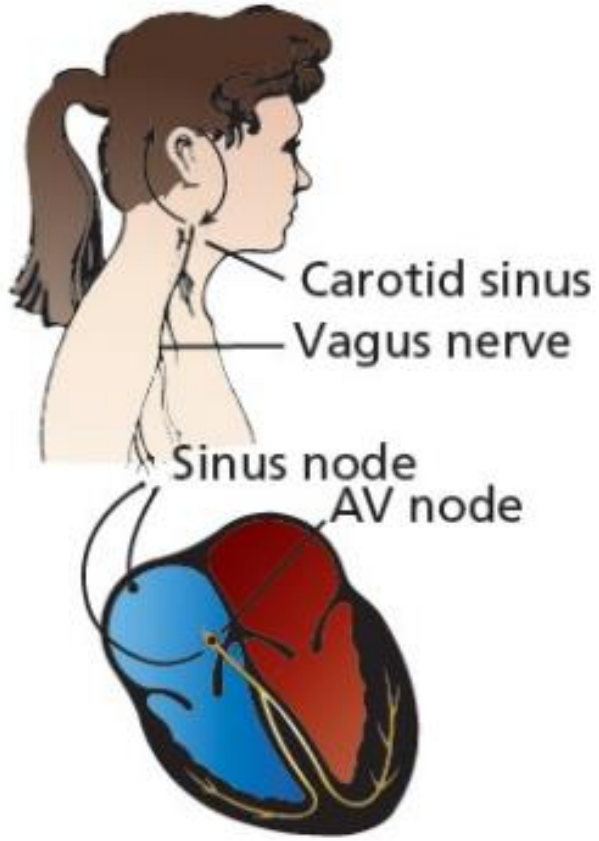






Carotid Massage

- Massaging the carotid artery can aid in diagnosing and stopping episodes of AVNRT (atrioventricular nodal reentrant tachycardia).
- Baroreceptors, located at the carotid artery's bifurcation, detect blood pressure changes. When pressure rises, these receptors trigger vagal responses, slowing the sinus node firing and AV node conduction.
- Gentle external pressure on the carotid artery can trick these baroreceptors into perceiving a blood pressure increase.
- Carotid massage, a widely used method, stimulates vagal input, potentially interrupting the reentrant circuit causing AVNRT. This may terminate the arrhythmia or, at least, slow it down, facilitating easier diagnosis by revealing P waves.



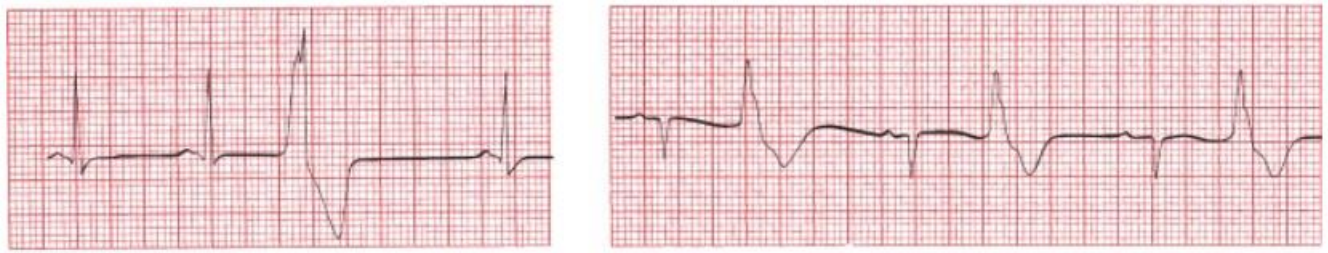
How to Do Carotid Massage

Carotid massage must be done with great care.

1. Auscultate for carotid bruits. You do not want to cut off the last remaining trickle of blood to the brain nor dislodge an atherosclerotic plaque. If there is evidence of significant carotid disease, do not perform carotid massage.
2. With the patient lying flat, extend the neck and rotate the head slightly away from you.
3. Palpate the carotid artery at the angle of the jaw and apply gentle pressure for 10 to 15 seconds. Press as firmly as would be required to compress a tennis ball.
4. Never compress both carotid arteries simultaneously!
5. Try the right carotid first because the rate of success is somewhat better on this side. If it fails, however, go ahead and try the left carotid next.
6. Have a rhythm strip running during the entire procedure so that you can see what is happening. Always have equipment for resuscitation available; in rare instances, carotid massage may induce sinus arrest.

Premature Ventricular Contractions

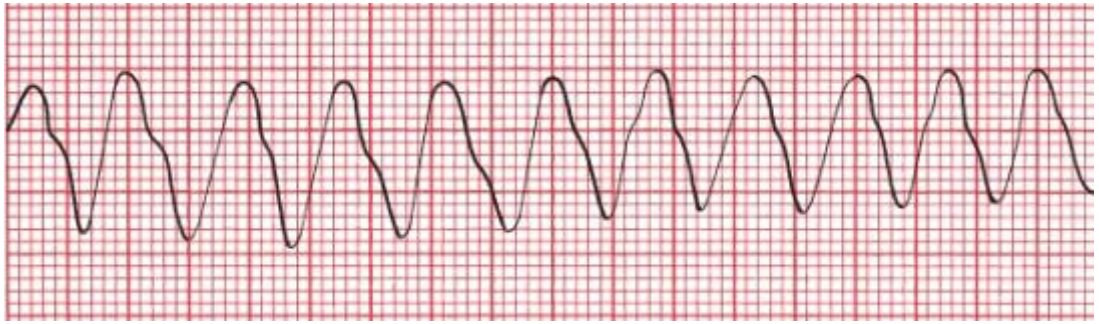
- Premature ventricular contractions (PVCs) are the most common ventricular arrhythmias. The QRS complex appears wide and unusual because ventricular depolarization deviates from normal conduction pathways. However, QRS width may vary in different leads, so examine the entire 12-lead EKG for an accurate diagnosis. A QRS duration of at least 0.12 seconds in most leads is required to diagnose a PVC. Retrograde P waves may occasionally be seen, but often there is no P wave. A compensatory pause usually follows a PVC before the next beat, but occasionally, PVCs may occur between normally conducted beats without a pause, known as interpolated PVCs.
- Isolated PVCs are common in normal hearts and typically don't need treatment. However, in the context of an acute myocardial infarction, they can be more concerning as they may trigger life-threatening ventricular arrhythmias like ventricular tachycardia or fibrillation.
- PVCs usually occur randomly but may alternate with normal sinus beats in a regular pattern. One normal beat followed by one PVC is called bigeminy, two normal beats for every PVC is trigeminy, and so on.



(*A*) A PVC. Note the compensatory pause before the next beat. (*B*) Bigeminy. PVCs and sinus beats alternate in a 1:1 fashion.

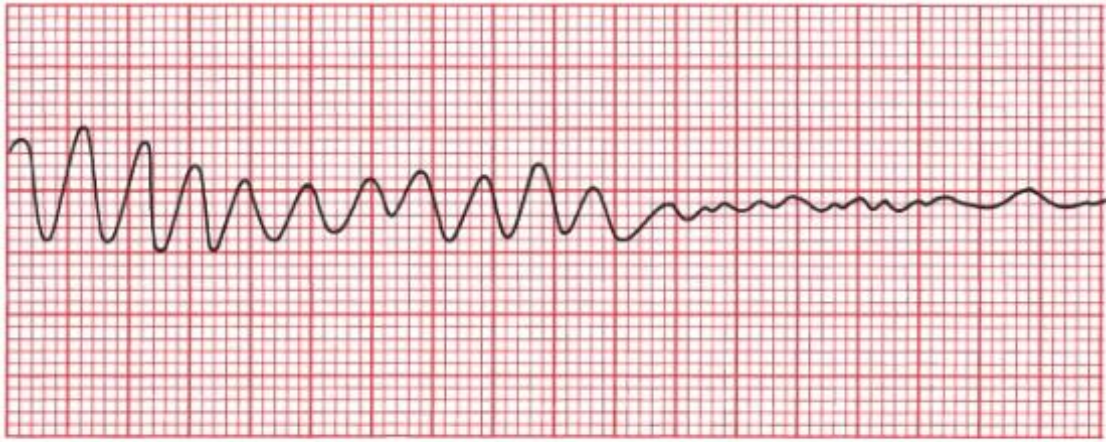
Ventricular Tachycardia

- A sequence of three or more consecutive premature ventricular contractions (PVCs) is termed ventricular tachycardia. The rate typically ranges from 120 to 200 beats per minute and may show slight irregularity. Sustained ventricular tachycardia, lasting over 30 seconds, or tachycardia linked to hemodynamic instability is considered an emergency, signaling the potential for cardiac arrest and necessitating immediate treatment.
- Ventricular tachycardia can have a uniform morphology, where each complex looks similar, or a polymorphic morphology, changing appearance from beat to beat. Polymorphic ventricular tachycardia is often linked to acute coronary ischemia, infarction, electrolyte imbalances, and conditions causing prolonged QT intervals. Uniform ventricular tachycardia is more common with healed infarctions, as the scarred myocardium provides the substrate for reentrant ventricular tachycardia.



Ventricular Fibrillation

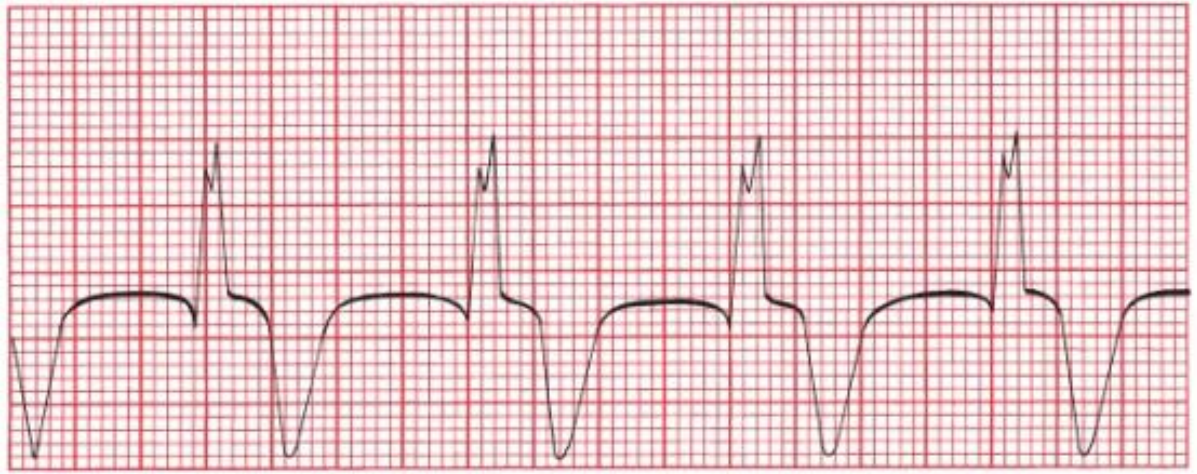
- Ventricular fibrillation is a critical event, typically observed in failing hearts and is the most common arrhythmia leading to sudden death in adults. The EKG tracing shows spasmodic jerking (coarse ventricular fibrillation) or gentle undulating (fine ventricular fibrillation) movements with no true QRS complexes.
- In ventricular fibrillation, the heart produces no cardiac output, requiring immediate cardiopulmonary resuscitation and electrical defibrillation.
- In many cases, ventricular fibrillation is preceded by ventricular tachycardia
- Common precipitants of ventricular fibrillation include MI, Heart failure, Hypoxemia or hypercapnia, Hypotension or shock, Electrolyte imbalances, Overdoses of stimulants, especially when used in combination



Ventricular tachycardia degenerates into ventricular fibrillation.

Accelerated Idioventricular Rhythm

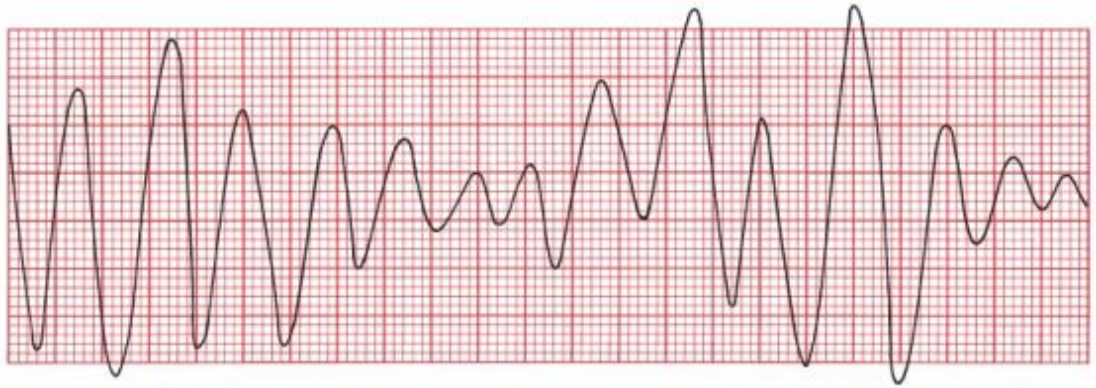
Accelerated idioventricular rhythm, often observed during acute infarction or in the early hours post-reperfusion, is a benign rhythm. It occurs regularly at 50 to 100 beats per minute, possibly arising from a ventricular escape focus that has accelerated enough to drive the heart. It is typically short-lived, does not advance to ventricular fibrillation, and seldom necessitates treatment. When the rate drops below 50 beats per minute, it is referred to as an idioventricular rhythm without the term "accelerated."



Accelerated idioventricular rhythm. There are no P waves, the QRS complexes are wide, and the rate is about 75 beats per minute.

Torsade de Pointes

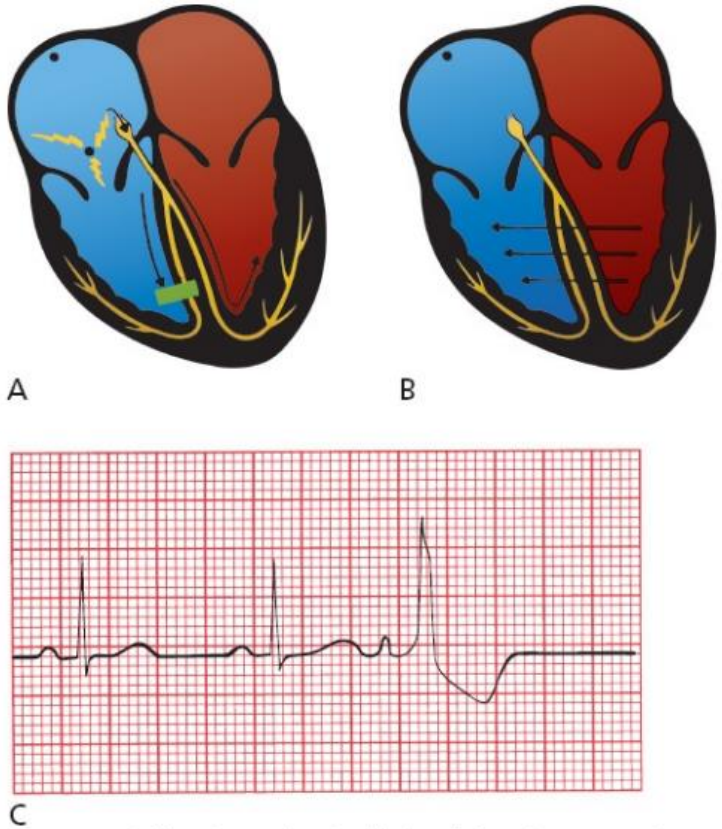
- Torsade de pointes, meaning "twisting of the points," is a unique form of ventricular tachycardia often observed in individuals with prolonged QT intervals. The QT interval covers the duration from ventricular depolarization to repolarization, typically constituting 40% of the cardiac cycle.
- Prolonged QT intervals can be congenital, result from electrolyte imbalances, or occur during acute myocardial infarction. Several drugs, including antiarrhythmics, tricyclic antidepressants, phenothiazines, and certain antibiotics with antifungal medications or antihistamines, can also lengthen the QT interval. Prolonged ventricular repolarization, indicated by a lengthened T wave, is a common cause.
- Torsade de pointes, resembling standard ventricular tachycardia, is distinguished by QRS complexes spiraling around the baseline, altering their axis and amplitude. Differentiating torsade de pointes from typical ventricular tachycardia is crucial for appropriate treatment.



Torsade de pointes. The QRS complexes seem to spin around the baseline, changing their axis and amplitude.

Supraventricular Tachycardia With Aberrancy

An atrial premature beat may occur very early in the cardiac cycle, catching the Purkinje fibers in the ventricles before they have fully repolarized. The right bundle branch, known for its slower recovery, can be particularly sluggish. As a result, when the premature atrial impulse reaches the ventricles, the right bundle branch is still in a refractory state. Consequently, the impulse can freely pass down the left bundle branch, causing the areas typically supplied by the right bundle branch to receive electrical activation from the already depolarized areas by the left bundle branch. This leads to an unusually prolonged ventricular depolarization, distorting the vector of current flow and resulting in a wide, abnormal QRS complex resembling a premature ventricular contraction (PVC).



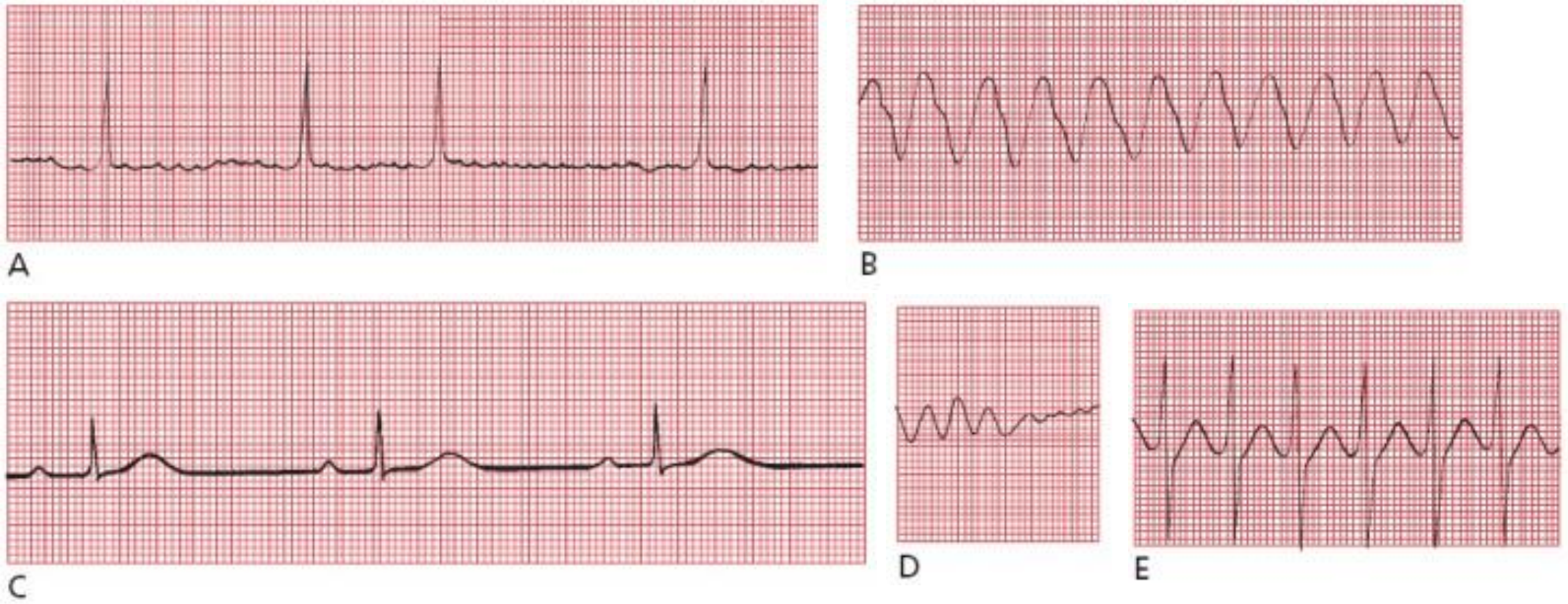
(*A*) A premature atrial impulse catches the right bundle branch unprepared. Conduction down the right bundle is blocked but proceeds smoothly down the left bundle. (*B*) Right ventricular depolarization occurs only when the electrical forces can make their way over from the left ventricle—a slow, tedious process. This mode of transmission is very inefficient and results in a wide, bizarre QRS complex. (*C*) The third P wave is a premature atrial contraction. It is conducted aberrantly through the ventricles, generating a wide, bizarre QRS complex.

V Tach Vs Supraventricular Tach With Aberrancy

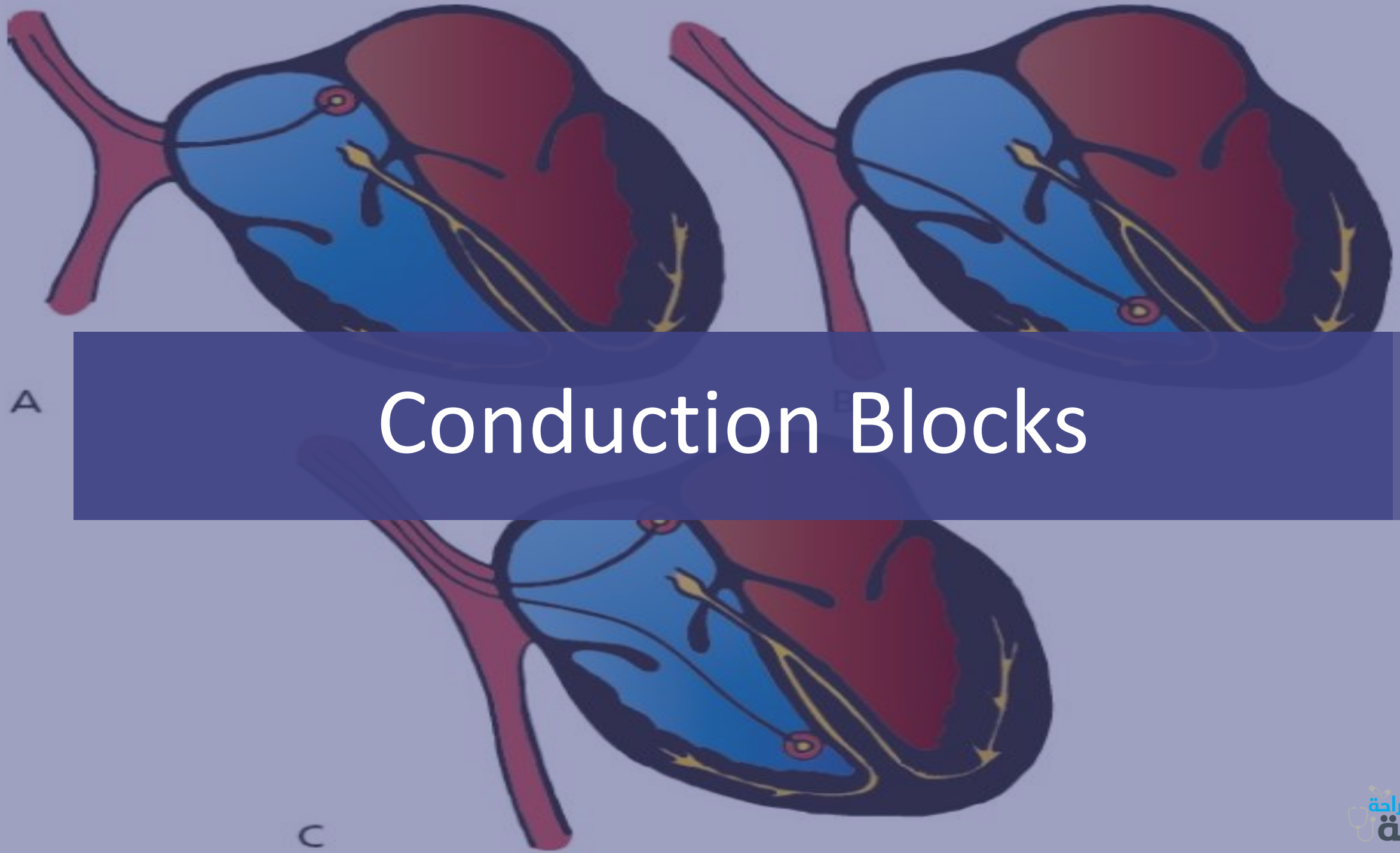
How do you tell the two apart?

		Ventricular Tachycardia	Supraventricular Tachycardias
Clinical Clues	Clinical history	Diseased heart	Usually, healthy heart
	Carotid massage	No response	May terminate
	Cannon A waves	May be present	Not seen
EKG Clues	AV dissociation	May be seen	Not seen
	Fusion beats	May be seen	Not seen
	Initial QRS deflection	May differ from normal QRS complex	Same as normal QRS complex

What are the arrythmias seen bellow ?



A	Atrial fibrillation
B	Ventricular tachycardia
C	Sinus bradycardia
D	Ventricular tachycardia degenerating into ventricular fibrillation
E	AVNRT



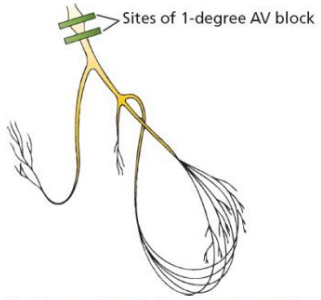
Conduction Blocks

AV Blocks

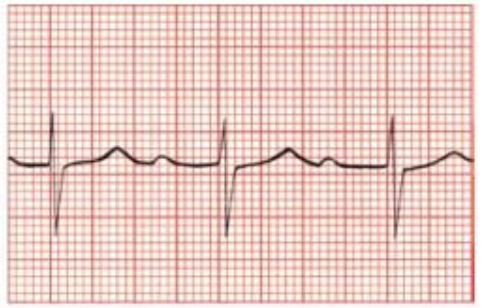
Note: Different degrees of AV block can coexist in the same patient (for example, a patient can have both first-degree and Mobitz type II heart blocks). Blocks also can be transient—a patient with Lyme carditis can bounce back and forth between different degrees of AV block within seconds!

First degree

- The PR interval is greater than 0.2 seconds; all beats are conducted through to the ventricles.
- It is a common finding in normal hearts

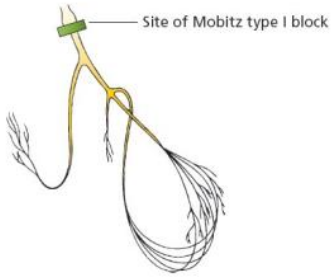


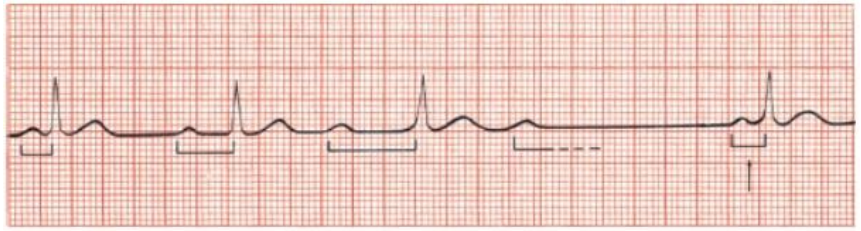
First-degree AV block. Note the prolonged PR interval.



Second degree Mobitz type I

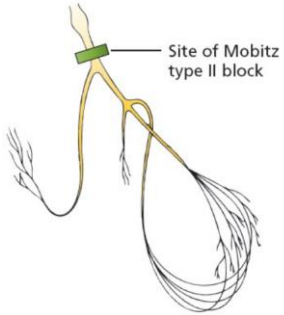
Progressive lengthening of each successive PR interval until one P wave fails to conduct through the AV node and is therefore not followed by a QRS complex

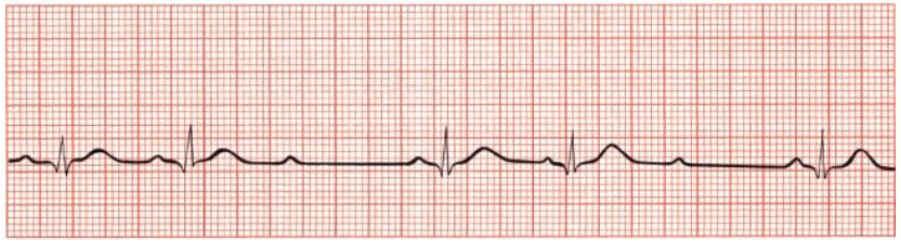




Second degree Mobitz type II

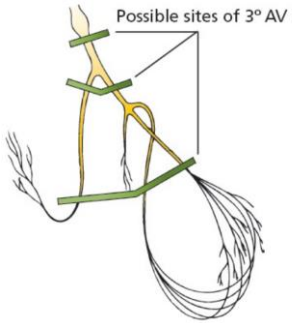
- All-or-nothing conduction, in which QRS complexes are periodically dropped without prolongation of the PR interval
- Usually due to a block below the AV node in the His bundle

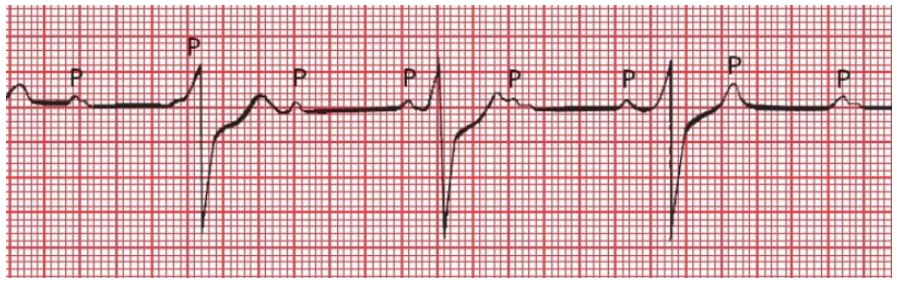




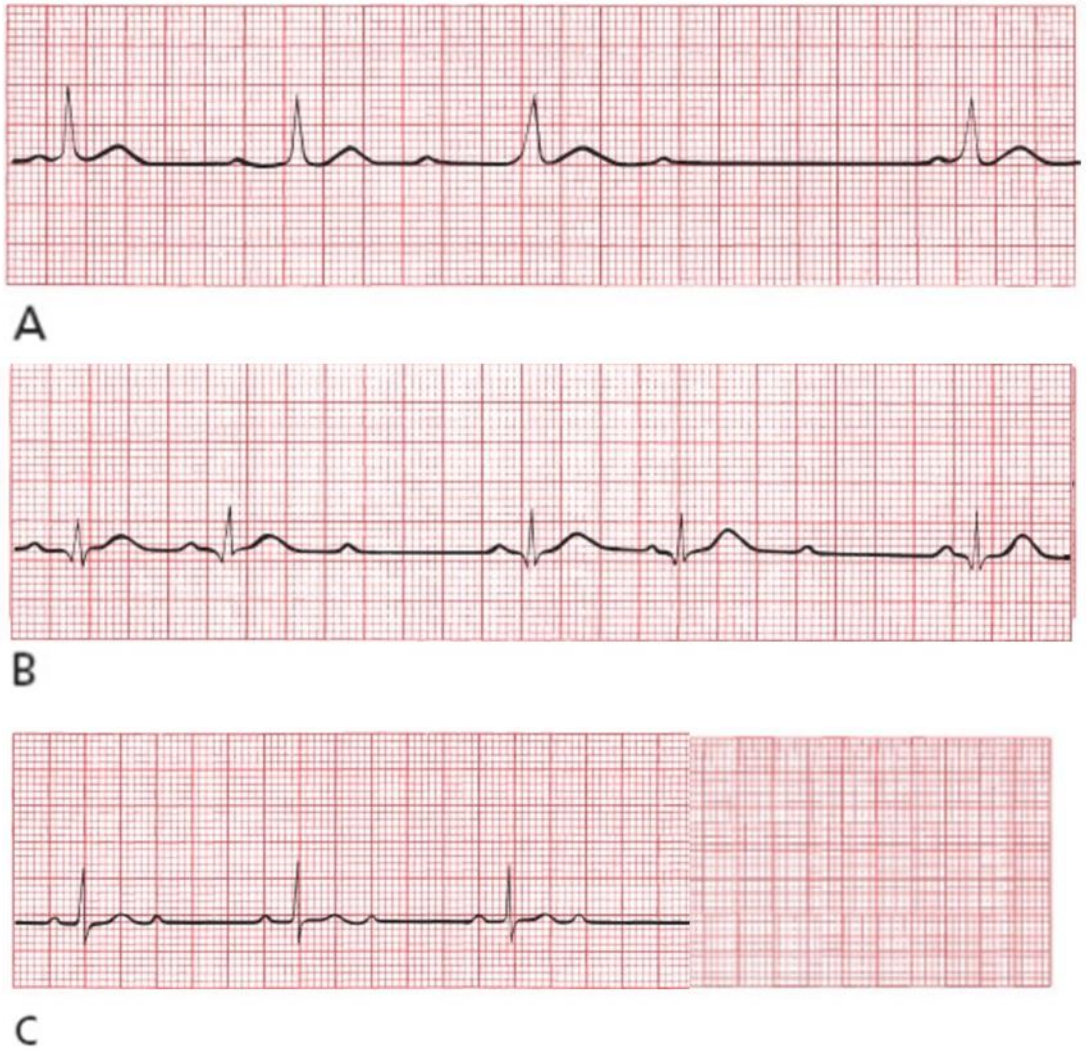
Third degree

No beats are conducted through to the ventricles. There is complete heart block with AV dissociation. No impulses reach the ventricles from above, and the ventricles are driven by a ventricular escape rhythm





Is It a Wenckebach Block or a Mobitz Type II Block?



(A) Wenckebach block, with progressive lengthening of the PR interval

(B) Mobitz type II block, in which the PR interval is constant

(C) Indeed, it's a classic example of second-degree heart block with a P wave–to–QRS complex ratio of 2:1. However, determining whether it's Wenckebach block or Mobitz type II block is impossible in this case due to the 2:1 ratio without clear PR lengthening. It's more accurate to label it as 2:1 AV block

Bundle Branch Block

Bundle branch block is diagnosed by looking at the width and configuration of the QRS complexes

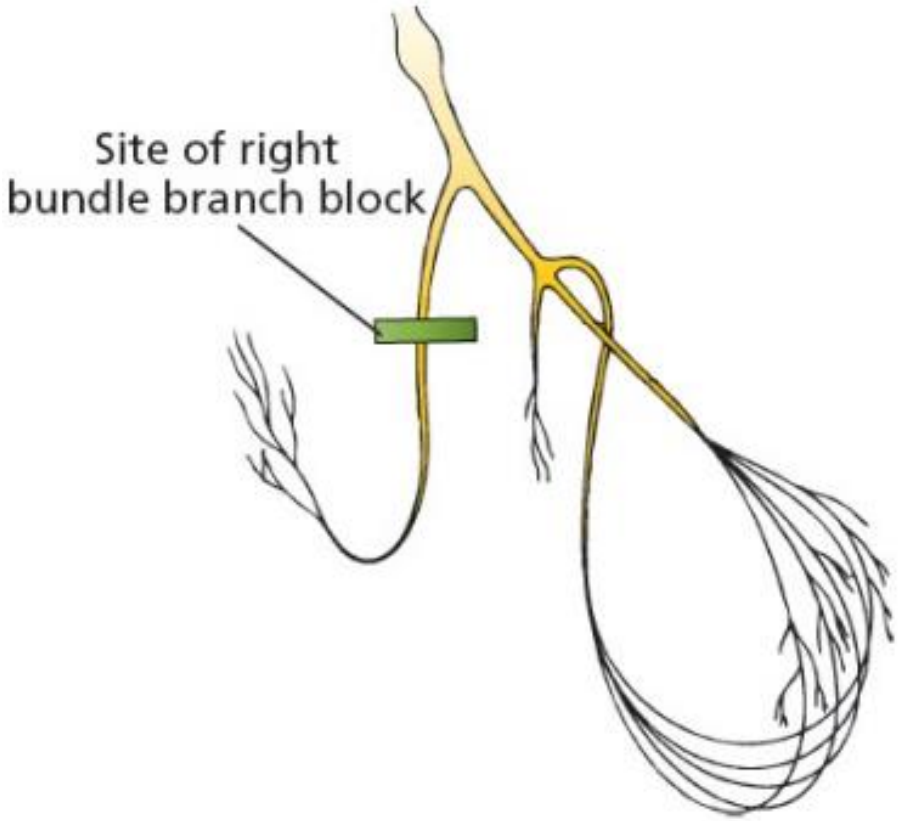
❖Criteria for Right Bundle Branch Block

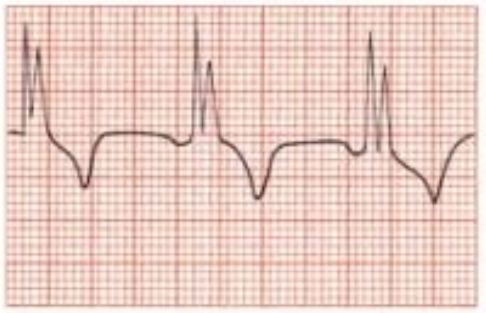
1. QRS complex widened to greater than 0.12 seconds
2. RSR' (rabbit ears) or a tall R wave in V1 and V2 with ST-segment depression and T-wave inversion [you will not always see a beautiful pair of rabbit ears with RBBB]
3. Reciprocal changes in V5, V6, I, and aVL
   - **Note**: Right bundle branch block doesn't cause right axis deviation

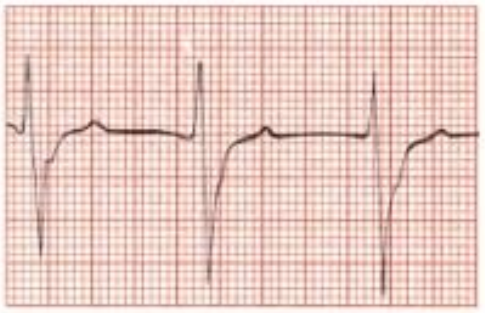
❖Criteria for Left Bundle Branch Block

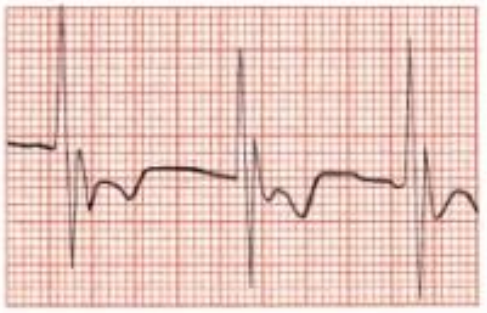
1. QRS complex widened to greater than 0.12 seconds
2. Broad or notched R wave with prolonged upstroke in leads V5, V6, I, and aVL, with ST-segment depression and T-wave inversion
3. Reciprocal changes in V1 and V2
4. Left axis deviation may be present

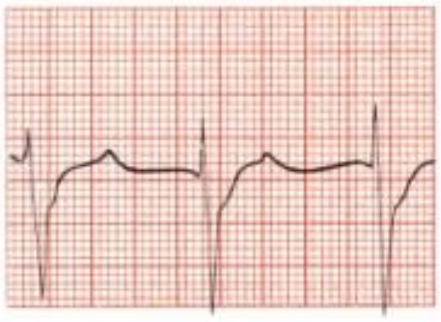
Right Bundle Branch Block

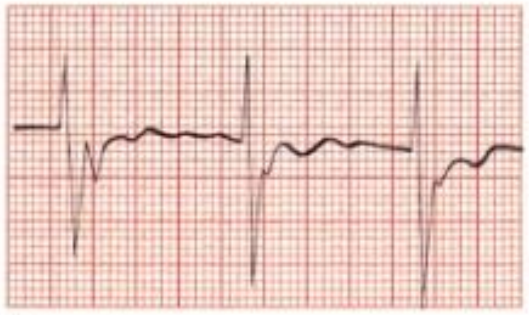


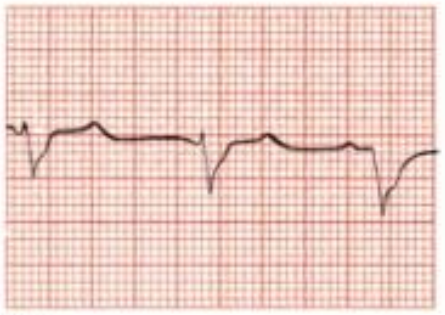

V1


V4

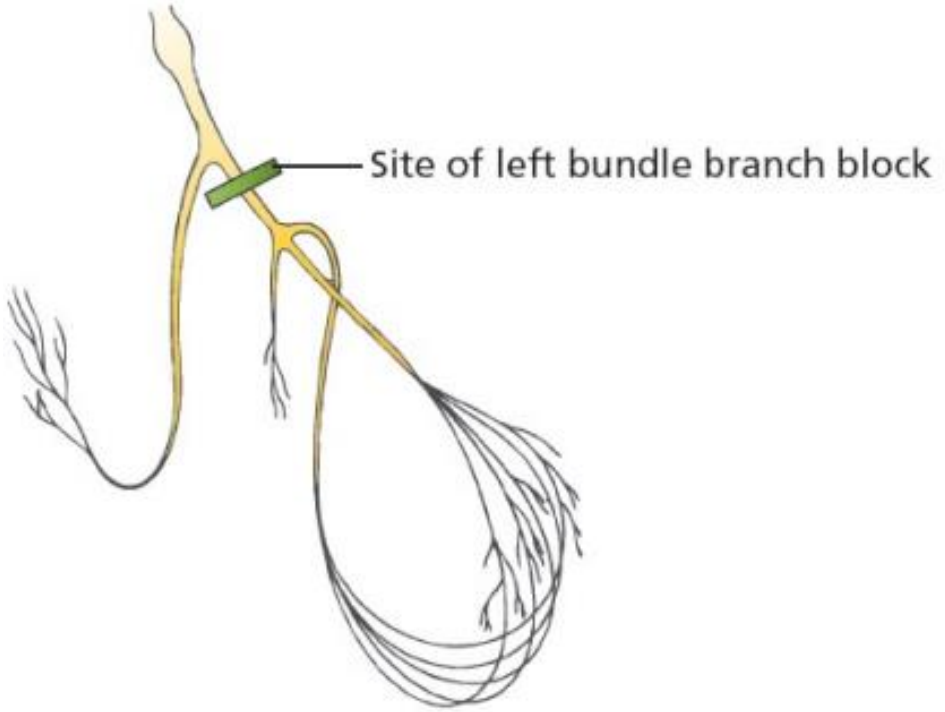

V2

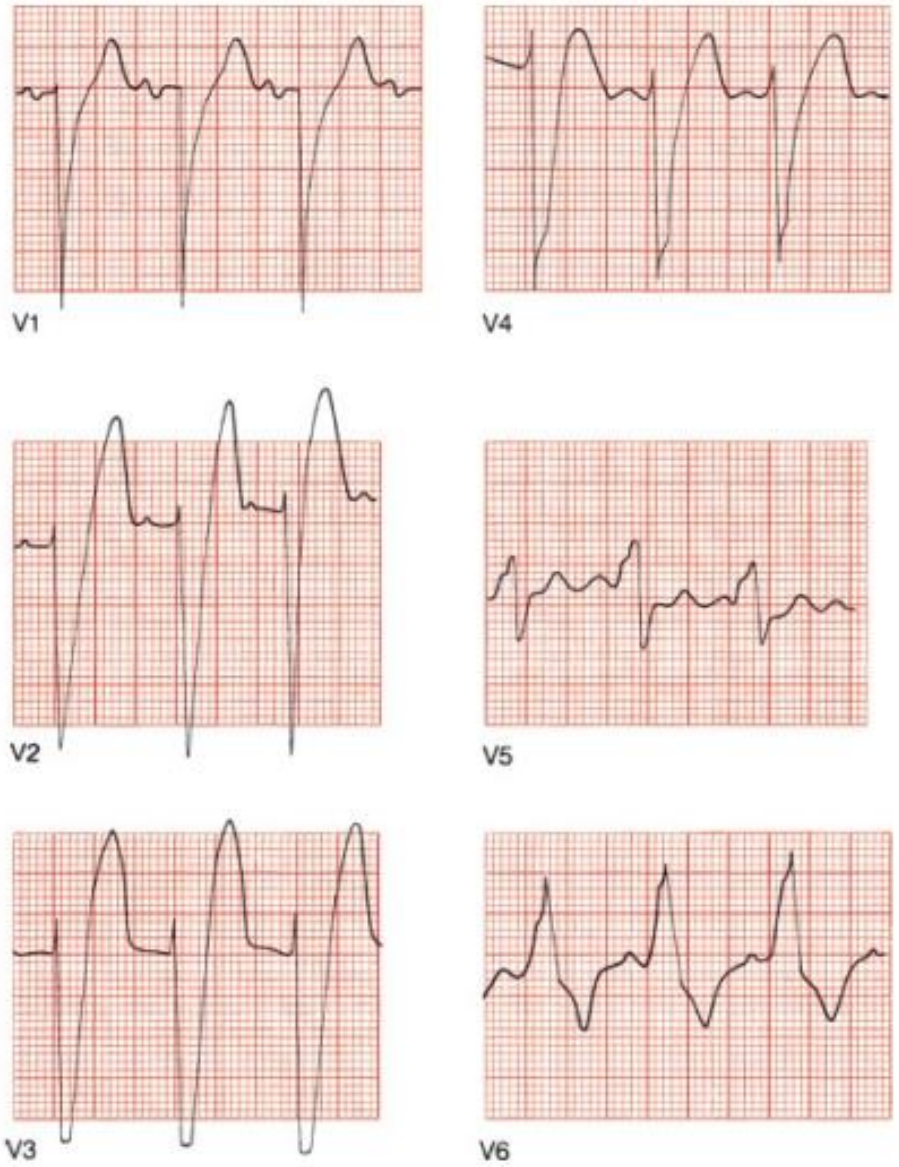

V5


V3


V6

Left Bundle Branch Block





Bundle Branch Block

- **Note 1**: Because bundle branch block affects the size and appearance of R waves, the criteria for ventricular hypertrophy (discussed later) cannot be used if bundle branch block is present. Specifically, right bundle branch block precludes the diagnosis of right ventricular hypertrophy, and left bundle branch block precludes the diagnosis of left ventricular hypertrophy. In addition, the diagnosis of a myocardial infarction can be extremely difficult in the presence of left bundle branch block.
- **Note 2**: While right bundle branch block can result from conducting system diseases, it's also quite common in hearts that are otherwise normal. In contrast, left bundle branch block is infrequent in normal hearts and typically indicates substantial underlying cardiac issues, like degenerative conduction system disease or ischemic coronary artery disease.
- **Note 3**: Both right and left bundle branch blocks can be either intermittent or fixed. For some individuals, bundle branch block only manifests at a specific heart rate, known as the critical rate. In simpler terms, electrical impulses are conducted normally at slow heart rates, but above a certain rate, bundle branch block occurs. The occurrence of rate-related bundle branch block is directly linked to the time it takes for a specific bundle branch to repolarize and prepare for the next electrical impulse. If the heart rate is too rapid for a bundle branch to repolarize in time, a temporary block to conduction occurs, presenting the characteristic EKG appearance of rate-related bundle branch block.

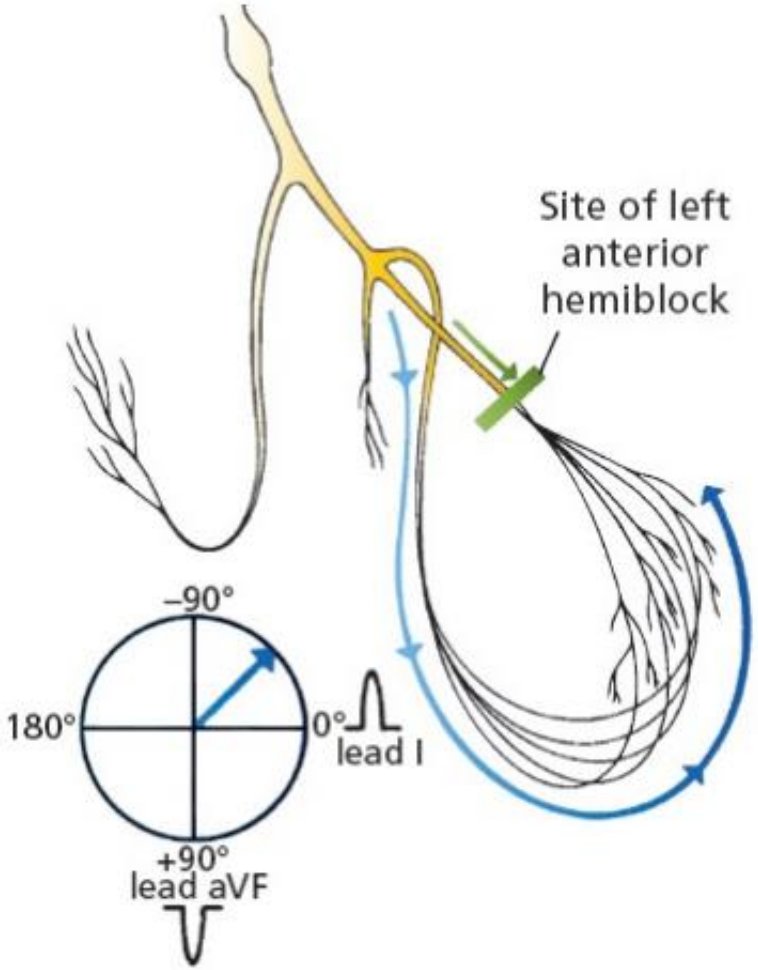
Hemiblocks

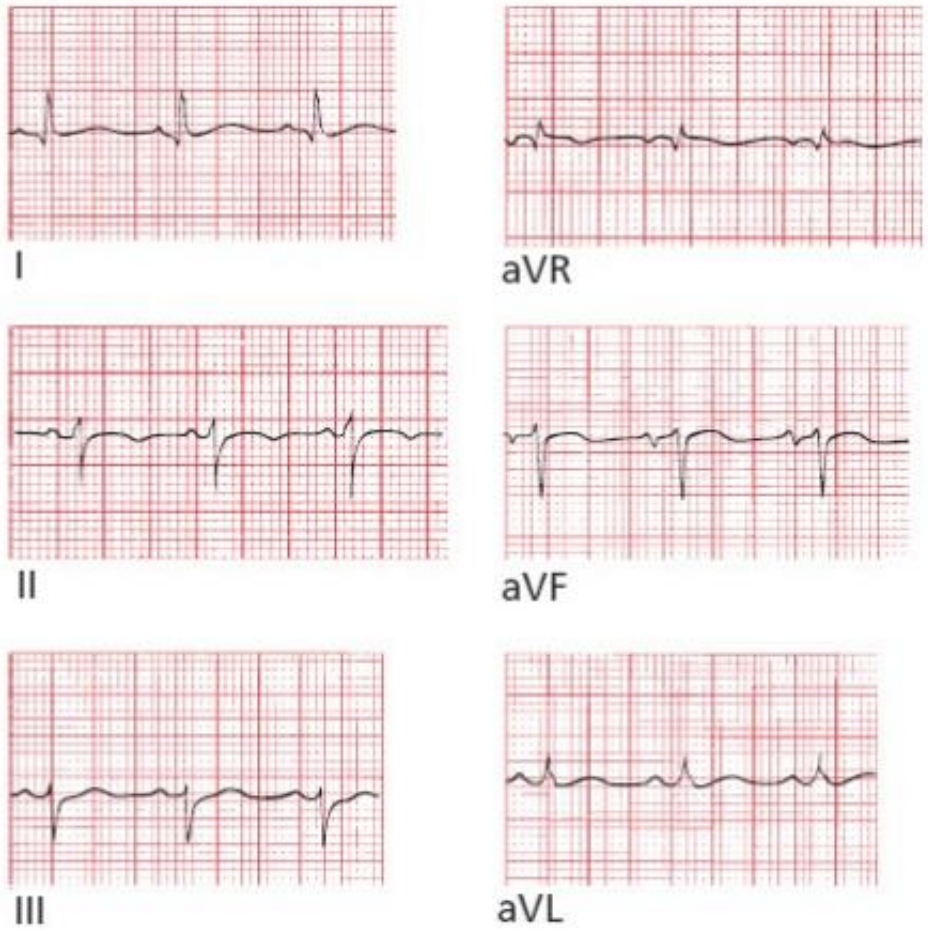
Hemiblocks Do Not Prolong the QRS Complex

Hemiblocks Cause Axis Deviation and is diagnosed by looking for left or right axis deviation:

- **Left Anterior Hemiblock** (More common & can be seen in normal hearts)
  1. Normal QRS duration and no ST-segment or T-wave changes.
  2. Left axis deviation between −30° and −90°.
  3. No other cause of left axis deviation is present.
- **Left Posterior Hemiblock** (Less common & only seen in diseased hearts)
  1. Normal QRS duration and no ST-segment or T-wave changes.
  2. Right axis deviation.
  3. No other cause of right axis deviation is present.

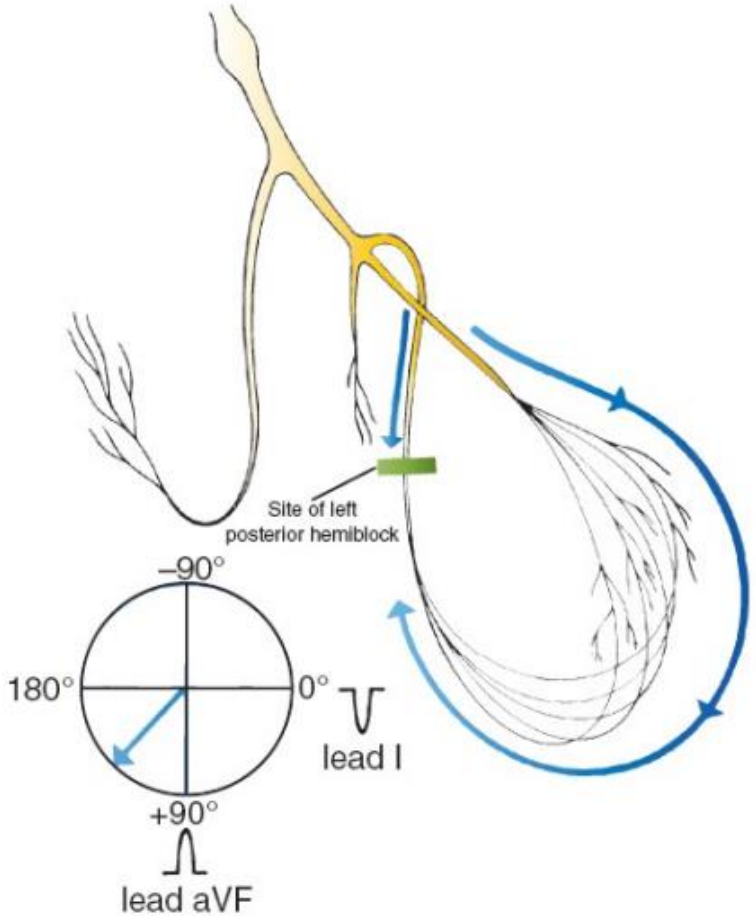
Left Anterior Hemiblock

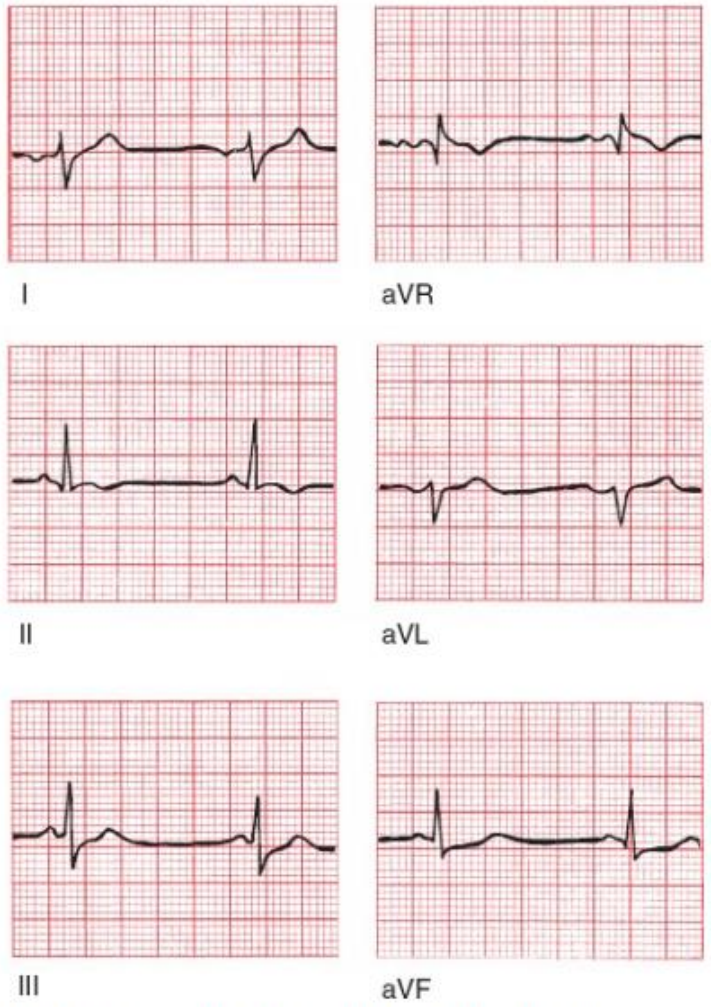




Left anterior hemiblock. Current flow down the left anterior fascicle is blocked; hence, all the current must pass down the posterior fascicle. The resultant axis is redirected upward and leftward (left axis deviation).

Left Posterior Hemiblock

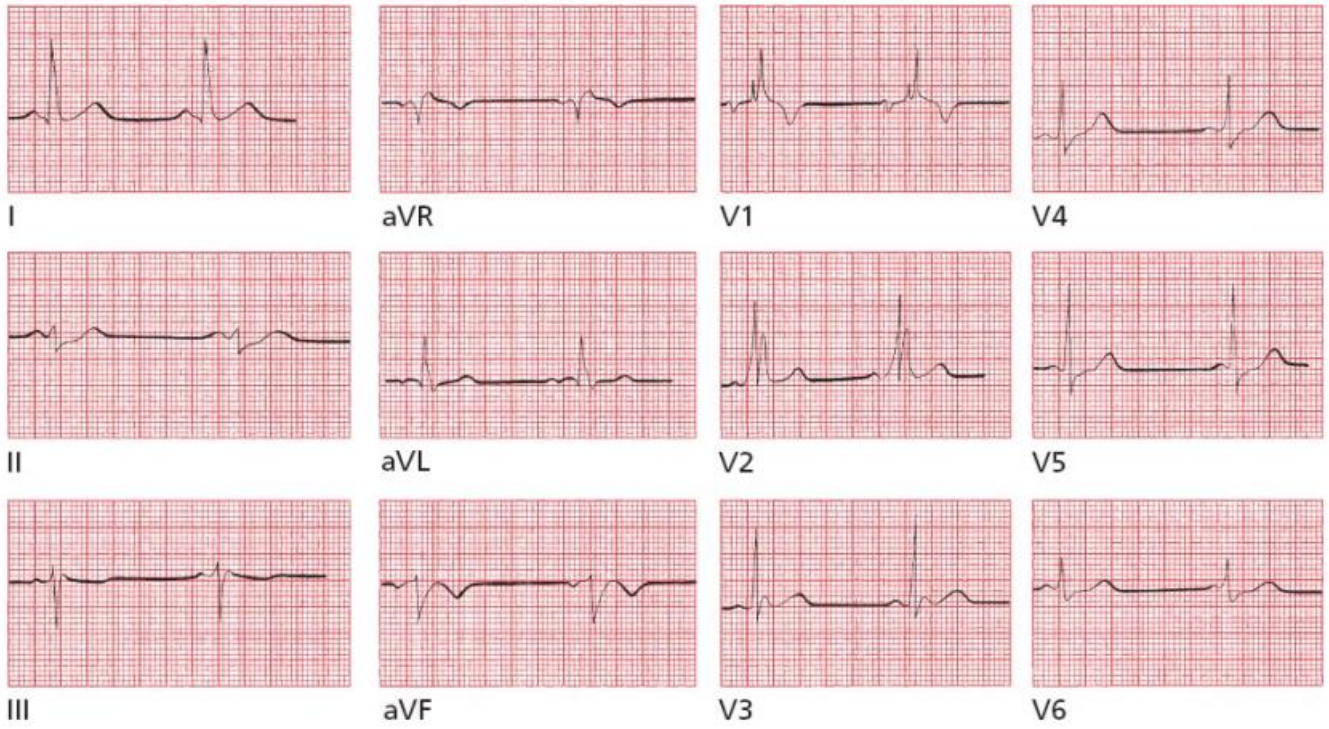




Left posterior hemiblock. Current flow down the left posterior fascicle is blocked; hence, all the current must pass down the right anterior fascicle. The resultant axis is redirected downward and rightward (right axis deviation).

Bifascicular block

- Right bundle branch block and hemiblocks can occur together. The term bifascicular block refers to the combination of right bundle branch block with either left anterior or left posterior hemiblock
- The EKG findings include a combination of features of both hemiblock and right bundle branch block

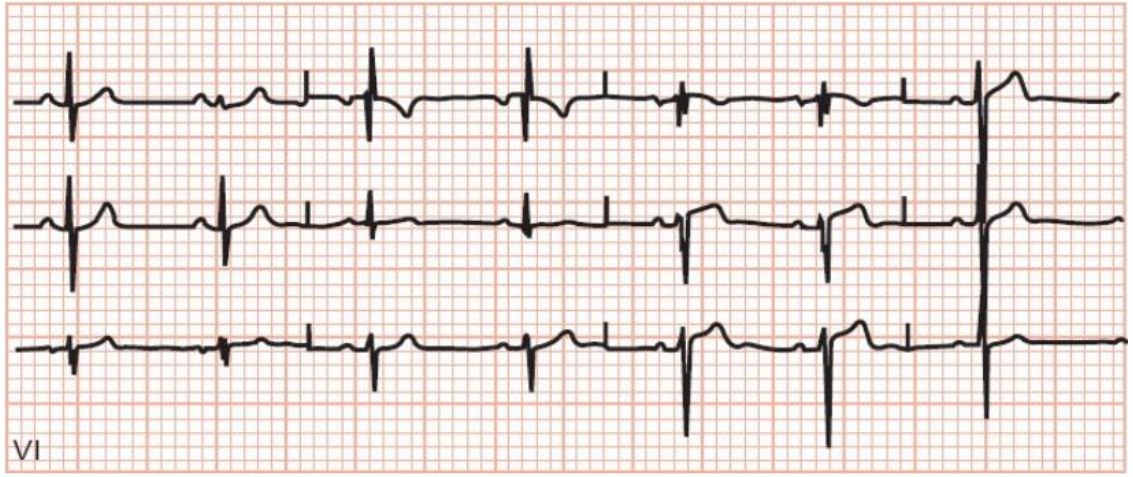


This is an example of right bundle branch block combined with left anterior hemiblock. Note the widened QRS complex and rabbit ears in leads V1 and V2, characteristic of right bundle branch block, and the left axis deviation in the limb leads (the QRS complex is predominantly positive in lead I and negative in leads aVF and II) that suggests left anterior hemiblock

Blocks That Underachieve

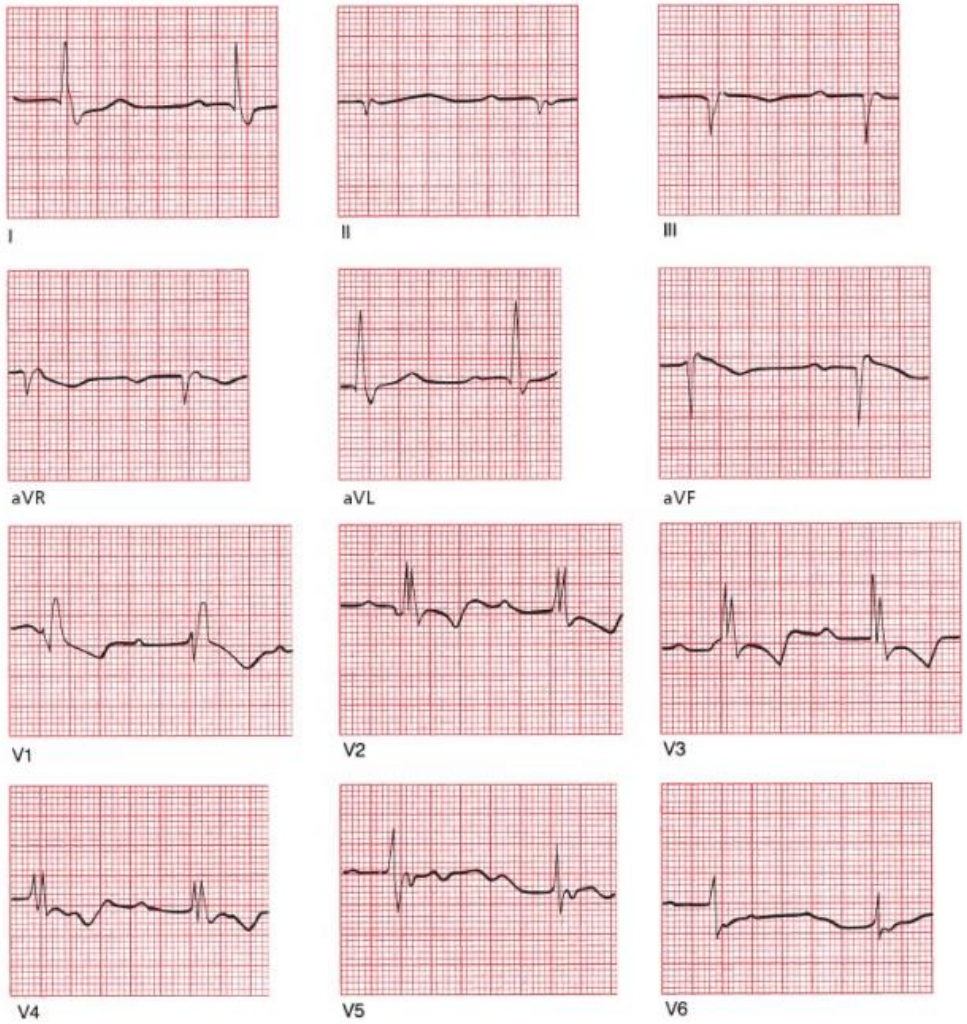
Not every conduction block meets all the criteria for a bundle branch block or bifascicular block. These are extremely common and generally fall into two types:

- **A nonspecific intraventricular conduction delay** occurs when there is QRS widening greater than 0.10 seconds without the other criteria for either bundle branch block or bifascicular block.
- **An incomplete bundle branch block** occurs when the EKG tracing shows a left or right bundle branch appearance (e.g., rabbit ears in V1 in right bundle branch block), but the QRS duration is between 0.10 and 0.12 seconds.
- These conduction blocks are caused by the same disease processes that cause the other conduction blocks.



Incomplete right bundle branch block; the QRS complex is not widened, but note the classic rabbit ears configuration in V1.

Can you identify the different conduction blocks that are present ?



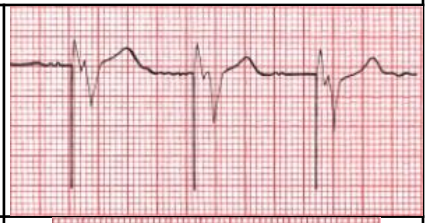
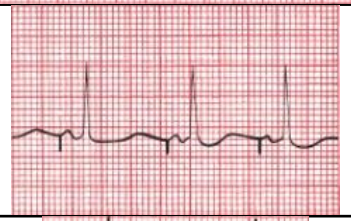
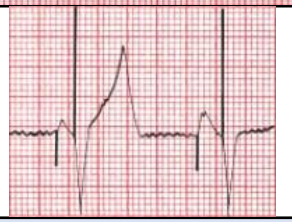
An orderly approach is essential

1. Is there an AV block?
2. Is there a bundle branch block?
3. Is there a hemiblock?

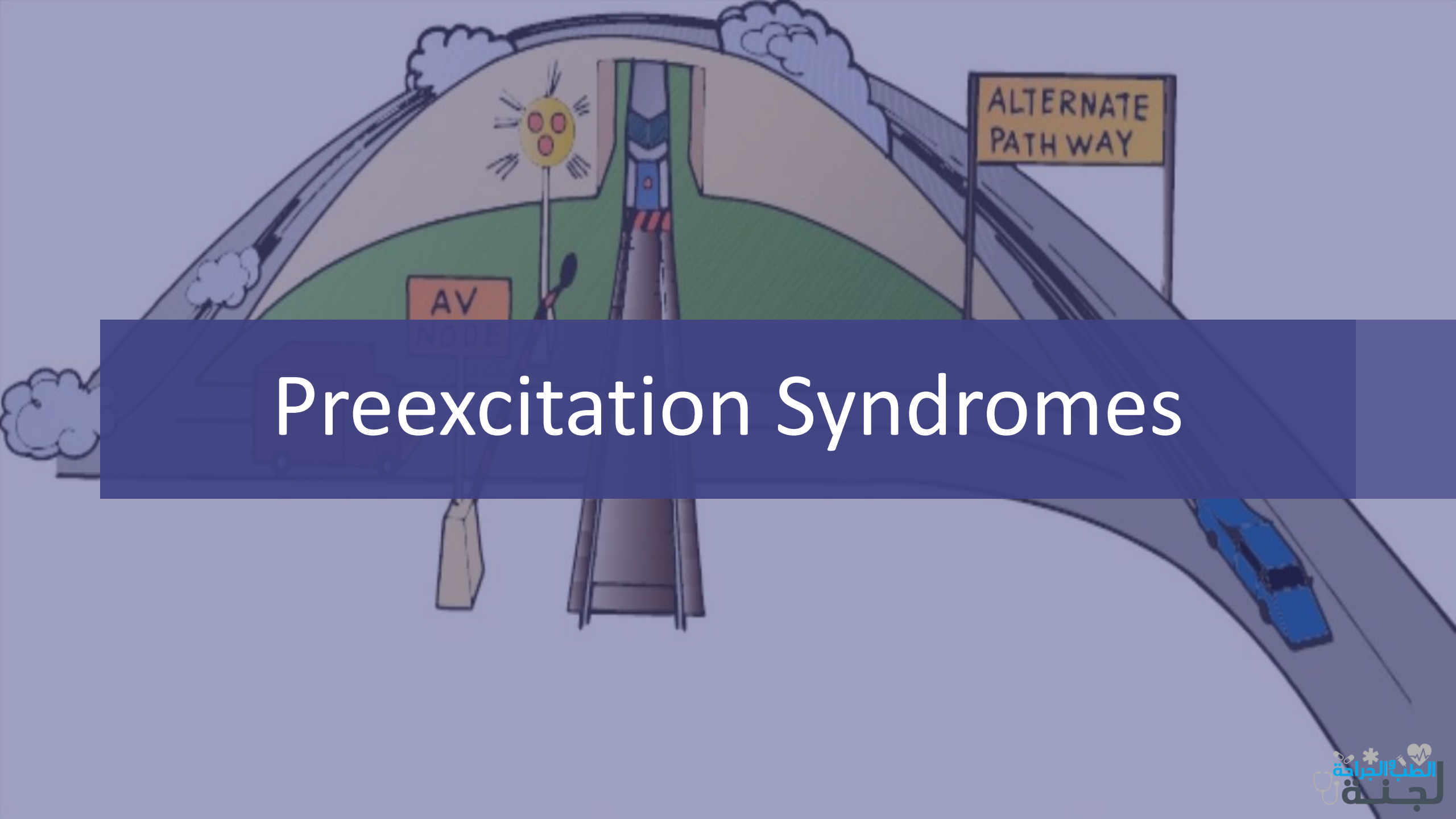
This EKG shows:

1. first-degree AV block (the PR interval exceeds 0.20 seconds)
2. right bundle branch block (there are wide QRS complexes with rabbit ears in leads V1 through V4)
3. left anterior hemiblock (left axis deviation is present)

Pacemakers

Pacemaker	When a pacemaker fires, a small spike can be seen on the EKG.	
Ventricular Pacemaker	The ensuing QRS complex will be wide and bizarre, just like a PVC	
Atrial Pacemaker	Generate a spike followed by a P wave, a normal PR Interval, and a normal QRS complex	
Sequential Pacemaker	Two spikes will be seen, one preceding a P wave and one preceding a wide, bizarre QRS complex	

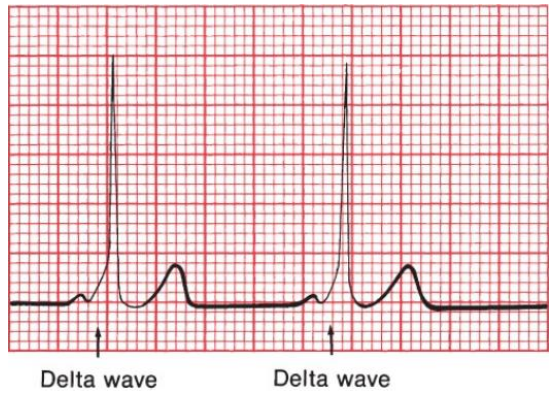
Note: In certain cases, pacemaker spikes may be challenging to discern on a standard EKG due to their low amplitude, sometimes less than 1 mV. When analyzing an EKG with wide QRS complexes and left axis deviation from an unfamiliar patient, suspicion of a pacemaker is warranted even if the small pacemaker spikes are not visible. Obviously, examination of the patient or—if the patient is lucid—a simple question or two will reveal the presence or absence of an electrical pacemaker



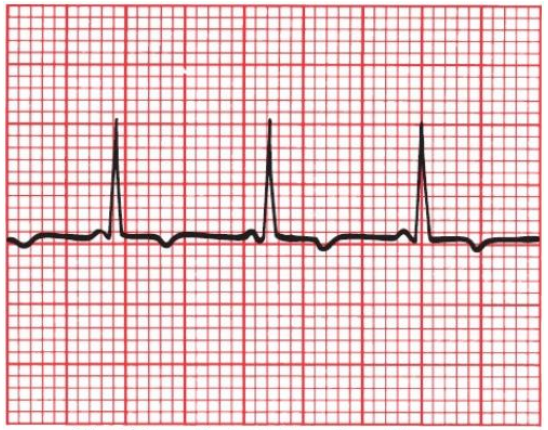
Preexcitation Syndromes

Preexcitation Syndromes

- The diagnosis of preexcitation is made by looking for a short PR interval.
- The most important preexcitation syndrome is Wolf – Parkinson–White
- **Criteria for WPW**
  1. PR interval less than 0.12 seconds
  2. Wide QRS complexes
  3. Delta wave seen in some leads
- Even more common than WPW is the presence of a short PR interval without an accompanying delta wave.
  - This finding, lacking a consistently identified anatomic pathway, likely results from various structural abnormalities. Some individuals may possess a small bypass pathway within or very close to the AV node, while others may have an AV node that conducts more rapidly than normal.

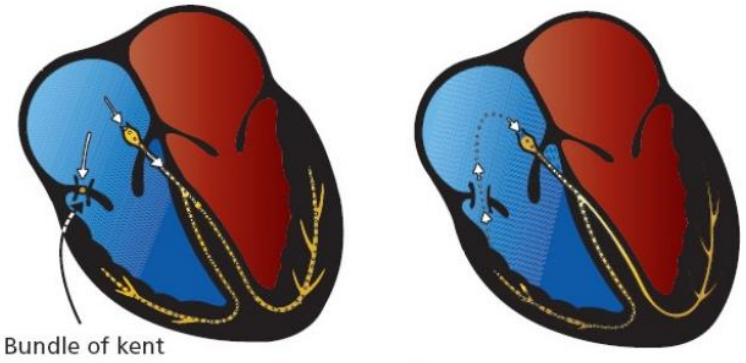


Wolff–Parkinson–White (WPW). Current is held up by the normal delay at the AV node but races unimpeded down the accessory pathway. The EKG shows the short PR interval and delta wave.

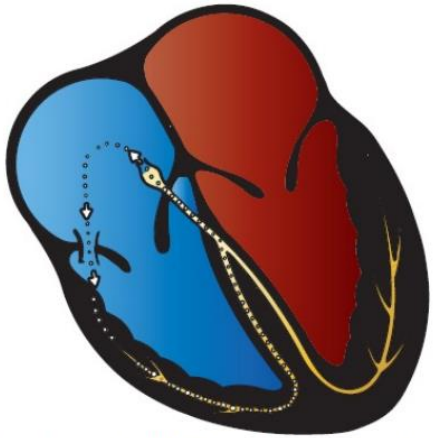

The PR interval is short, but there is no delta wave.

Why Do We Care About Preexcitation?

- In many individuals with WPW, preexcitation poses few, if any, clinical problems. However, preexcitation does predispose to various tachyarrhythmias.
- Arrhythmias commonly seen include the following:
  1. **AV reciprocating tachycardia**:
     - When the tachycardia activates the ventricles in an antegrade manner through the AV node, generating a narrow QRS complex, the arrhythmia is further subcategorized as an **orthodromic tachycardia**. (more common)
     - Reciprocating tachycardias that activate the ventricles through the accessory pathway, generating a wide QRS complex, are subcategorized as **antidromic tachycardia**.
  2. **Atrial fibrillation**:
     - Can be very rapid and rarely can lead to ventricular fibrillation (it must be considered a diagnostic possibility in patients who have been resuscitated from an episode of sudden death or syncope and are found to have preexcitation on their cardiograms)



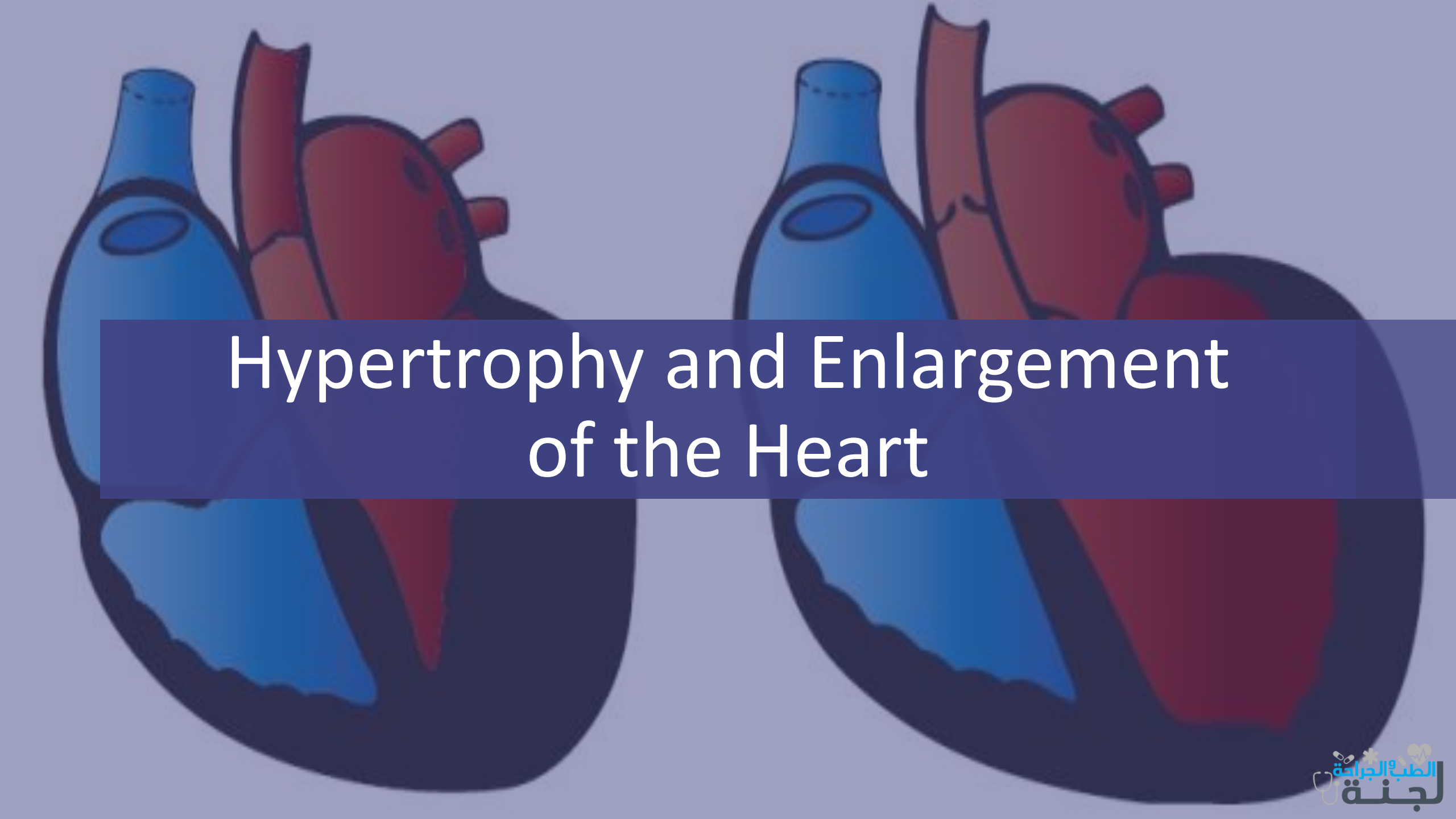
The formation of a reentry circuit in WPW. (*A*) A premature atrial beat sends current down the normal conduction pathways but not through the refractory accessory pathway. (*B*) Current then circles back through the accessory pathway, which is no longer refractory to conduction, forming a complete reentrant circuit.



A second type of reentry circuit in WPW. Current moves antegrade down the accessory pathway and then retrograde through the AV node, establishing an independent revolving circuit.

Notes

- **Note 1**: Because the presence of an accessory pathway in WPW alters the vectors of current flow to at least some degree, you cannot assess axis or amplitude with any precision, and hence, any attempt to determine the presence of ventricular hypertrophy or bundle branch block is bound to be unreliable.
- **Note 2**: **DDx of Wide Complex Tachycardias**: Ventricular tachycardia, Supraventricular tachycardia with aberrant conduction, AV reciprocating tachycardia (antidromic tachycardia) in a patient with preexcitation, Paced rhythms
- **Note 3**: When you see what appears to be a wide complex tachycardia and you did not run the EKG yourself—for example, if you are looking at the tracing on a hospital monitor—make sure you are not seeing an artifact caused by the patient's activity



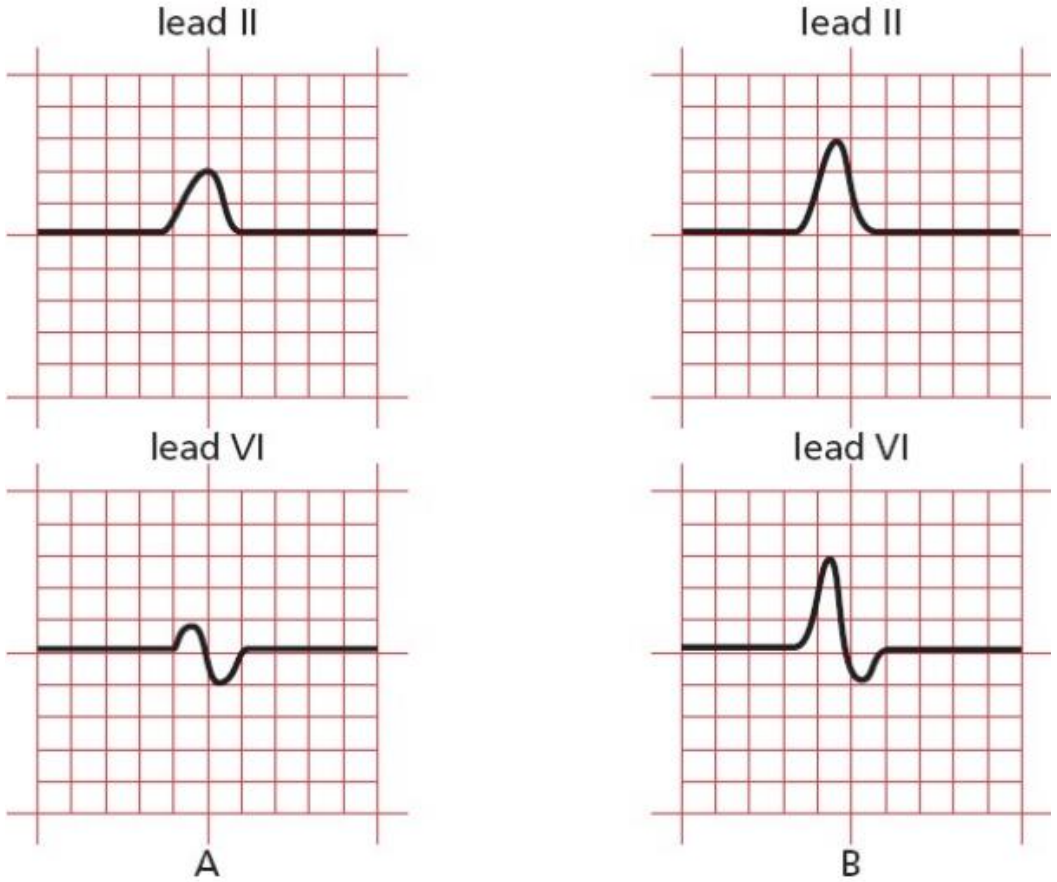
Hypertrophy and Enlargement of the Heart

Atrial Enlargement

- To diagnose atrial enlargement, look at leads II and V1.
- **Right atrial enlargement is characterized by the following:**
  1. P waves with an amplitude exceeding 2.5 mm in the inferior leads
  2. No change in the duration of the P wave
  3. Possible right axis deviation of the P wave
- **Left atrial enlargement is characterized by the following:**
  1. The amplitude of the terminal (negative) component of the P wave may be increased and must descend at least 1 mm below the isoelectric line in lead V1.
  2. The duration of the P wave is increased, and the terminal (negative) portion of the P wave must be at least 1 small block (0.04 second) in width.
  3. No significant axis deviation is seen because the left atrium is normally electrically dominant.

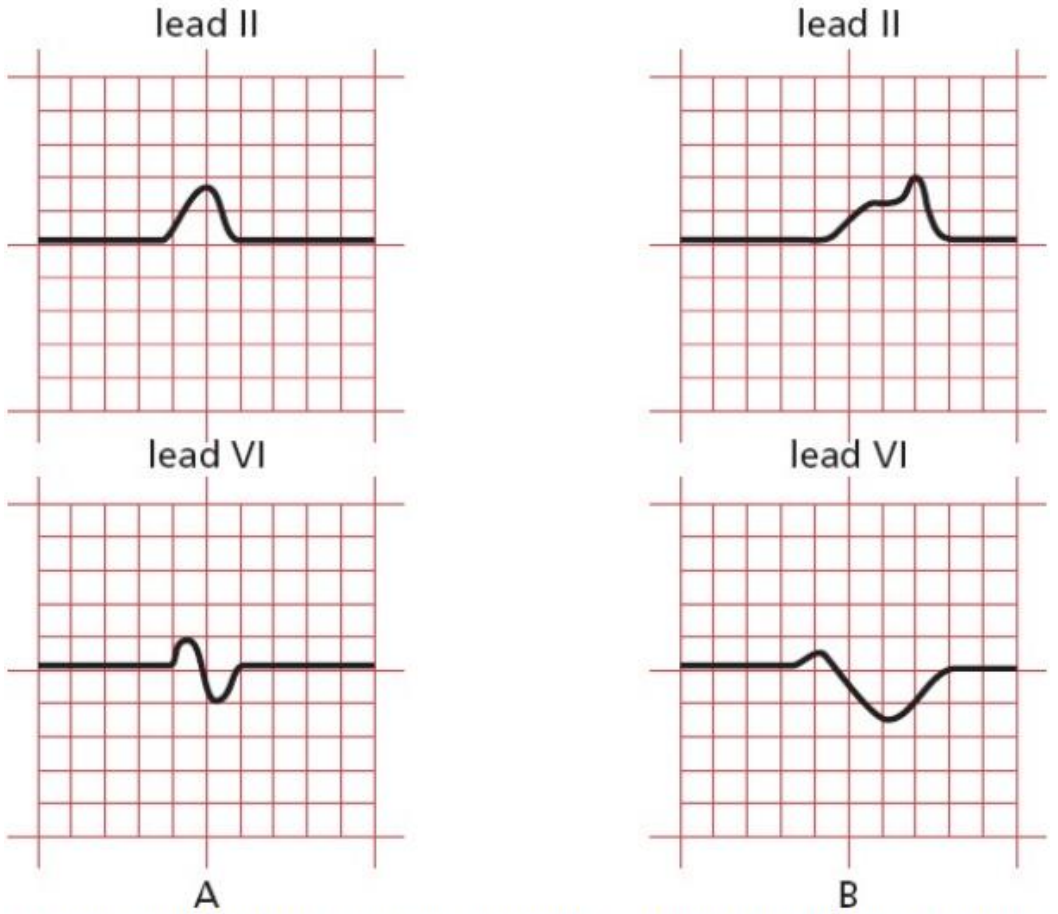
Atrial Enlargement

Right Atrial Enlargement



(*A*) The normal P wave in leads II and V1. (*B*) Right atrial enlargement. Note the increased amplitude of the early, right atrial component of the P wave. The terminal left atrial component, and hence the overall duration of the P wave, is essentially unchanged.

Left Atrial Enlargement



(*A*) Again, the normal P wave in leads II and V1. (*B*) Left atrial enlargement. Note the increased amplitude and duration of the terminal, left atrial component of the P wave.

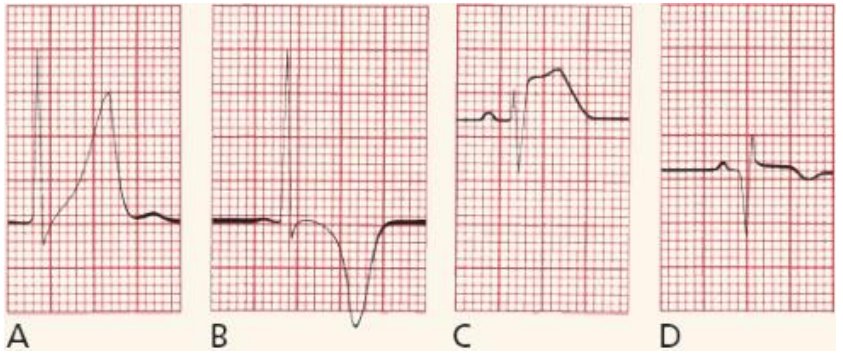
Ventricular Hypertrophy

- **Right ventricular hypertrophy is characterized by the following:**
  1. Right axis deviation is present, with the QRS axis exceeding +100°.
  2. The R wave is larger than the S wave in V1, whereas the S wave is larger than the R wave in V6.
- **Left ventricular hypertrophy is characterized by** voltage criteria and, not infrequently, secondary repolarization abnormalities. The most useful criteria are the following:
  1. The R wave in V5 or V6 plus the S wave in V1 or V2 exceeds 35 mm.
  2. The R wave in aVL is 11 mm.
  3. The R wave in aVL plus the S wave in V3 exceeds 20 in women and 28 in men.
  4. Left axis deviation exceeding −15° is also often present.
- Secondary repolarization abnormalities include asymmetric, T-wave inversion and down-sloping ST-segment depression.

Myocardial Ischemia and Infarction

During an acute STEMI, the EKG may evolve through 3 stages

- The T wave peaks (A) and then inverts (B)
  - Inverted T waves can be normal in leads V1 through V3 in children and can persist into adulthood in some patients; an isolated inverted T wave in lead III is also a common normal variant
- The ST segment elevates (C)
  - Distinguishing ST Elevation of Ischemia from J Point Elevation
- Q waves appear (D)
  - Ischemic Q waves are almost never isolated to a single lead
  - **Note**: Because lead aVR occupies a unique position on the frontal plane, it normally has a very deep Q wave. Lead aVR should not be considered when using Q waves to look for possible infarction.



Criteria for Ischemia:

Leads With ST Elevation	Men < 40	Men > 40	Women of All Ages
Leads V2 or V3	>2.5 mm	>2.0 mm	>1.5 mm
All other leads	>1 mm	>1 mm	>1 mm

The ST elevation must be present in at least two contiguous leads

Localizing the Infarct

The characteristic electrocardiographic changes of infarction occur only in those leads overlying or near the site of infarction:

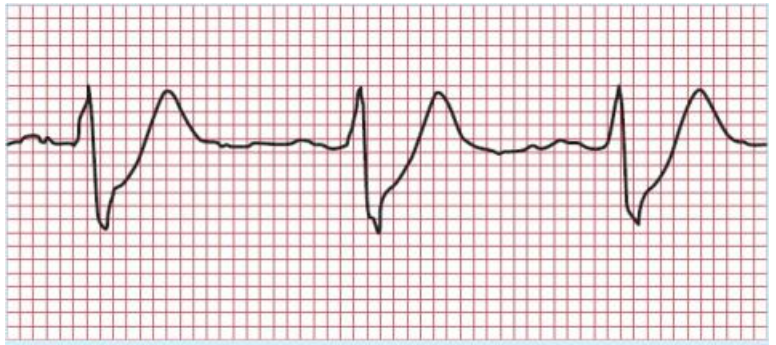
1. Inferior infarction involves the diaphragmatic surface of the heart. It is often caused by occlusion of the right coronary artery or its descending branch. The characteristic electrocardiographic changes of infarction can be seen in the inferior leads II, III, and aVF.
2. Lateral infarction involves the left lateral wall of the heart. It is most often due to occlusion of the left circumflex artery. Changes will occur in the left lateral leads I, aVL, V5, and V6.
3. Anterior infarction involves the anterior surface of the left ventricle and is usually caused by occlusion of the left anterior descending (LAD) artery. Any of the precordial leads (V1 through V6) may show changes. Occlusion of the left main artery will characteristically cause an extensive anterolateral infarction with changes in the precordial leads plus leads I and aVL.
4. Posterior infarction involves the posterior surface of the heart and is usually caused by occlusion of the right coronary artery. Posterior infarctions rarely occur in isolation, but usually accompany an inferior infarction or, less commonly, a lateral infarction. There are no leads directly overlying the posterior wall. The diagnosis must therefore be made by looking for reciprocal changes in the anterior leads, for example, a tall R wave in leads V1, V2, or V3.

Anterior STEMIs

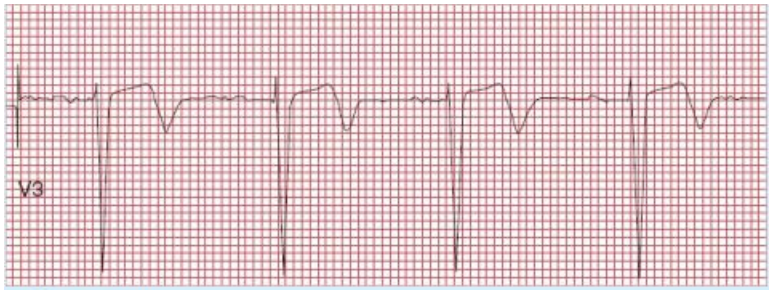
Anterior STEMIs have serious consequences, affecting much of the left ventricular myocardium. Early recognition is vital.

Two T-wave abnormalities signal left anterior descending artery occlusion and an impending anterior myocardial infarction:

1. **deWinter's T waves**: In a patient with chest pain, upsloping ST depression leading into tall, symmetric, hyperacute T waves in the precordial leads can be the first sign of an anterior infarction.
2. **Wellens' waves**: Deeply inverted or biphasic T waves in leads V2, V3 and sometimes V4 predict a proximal occlusion of the left anterior descending artery and are cause for concern. When you see biphasic T waves in these leads, a tip-off that these may be Wellen's waves is that the upright portion of the T wave occurs first, followed by inversion of the terminal portion. There may or may not be ST-segment elevation.



deWinter's T waves



Wellen's waves

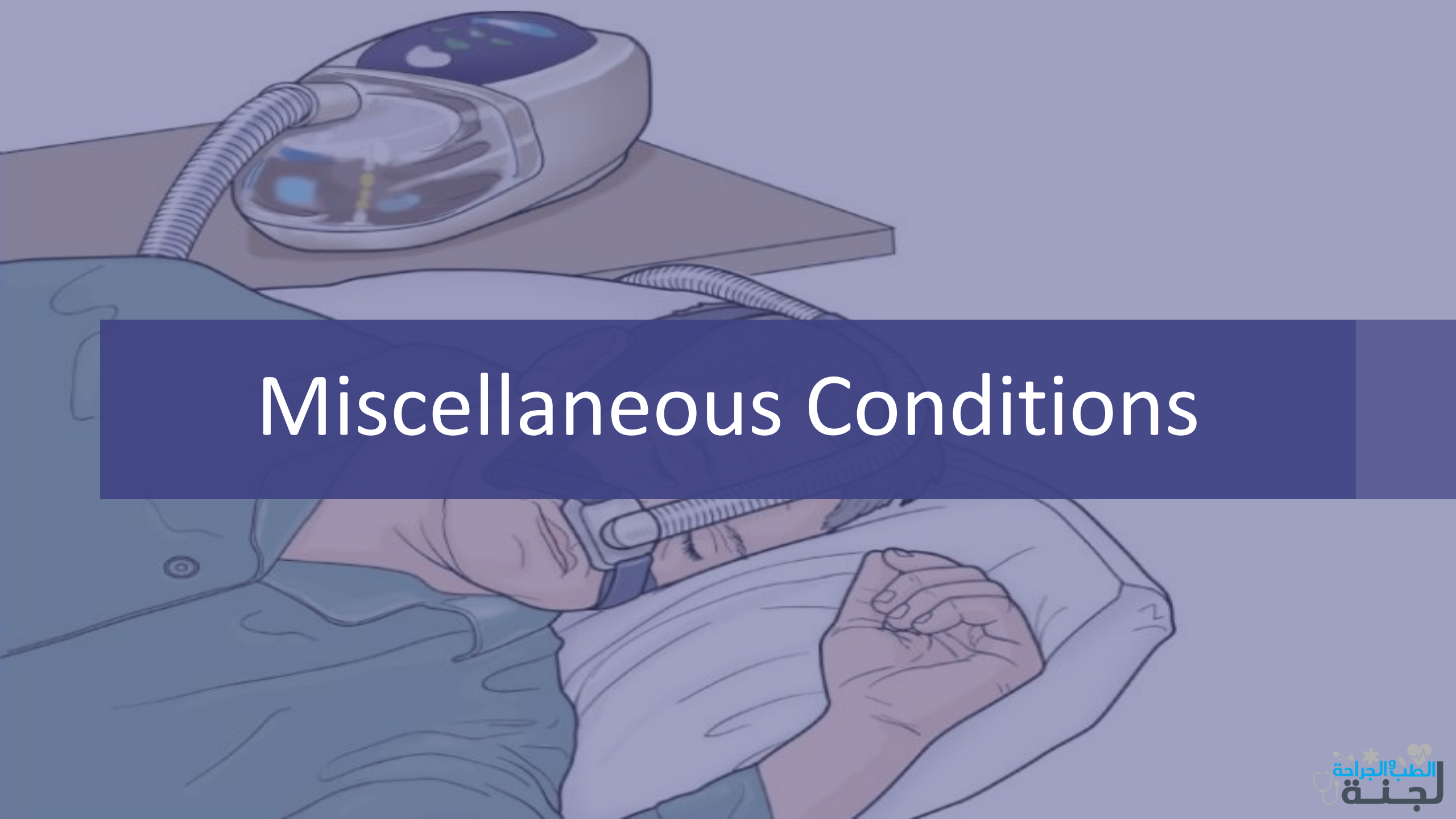
Right Ventricular Infarctions

- Right ventricular infarctions typically accompany inferior infarctions. Expect changes in leads II, III, and aVF indicating an inferior infarction, along with T-wave alterations and ST-segment elevation in the rightward anterior lead, V1. If ST elevation appears in lead V2, it's usually of smaller magnitude than in V1, with possible ST depression in V2. In limb leads, a clue to a right ventricular STEMI accompanying an inferior infarction is greater ST elevation in lead III than in lead II, given the far-right positioning of lead III.
- Does it make any difference clinically whether or not an infarction of the inferior wall of the left ventricle is accompanied by right ventricular infarction? Yes, indeed it does. Patients with right ventricular infarctions are “preload sensitive,” that is, they require high fluid volumes to maintain an adequate cardiac output and blood pressure, and they can become extremely hypotensive if they are treated with nitrates

Sorting Out the Different Ischemic Syndromes

Symptom or Syndrome	ST-Segment Changes	Cardiac Enzymes
Stable angina without infarction	ST depression	Normal*
Unstable angina without infarction	ST depression	Normal*
STEMI	ST elevation	Elevated
Non-STEMI	ST depression	Elevated
Takotsubo cardiomyopathy	ST elevation	Elevated**
Prinzmetal's angina	ST elevation	Normal

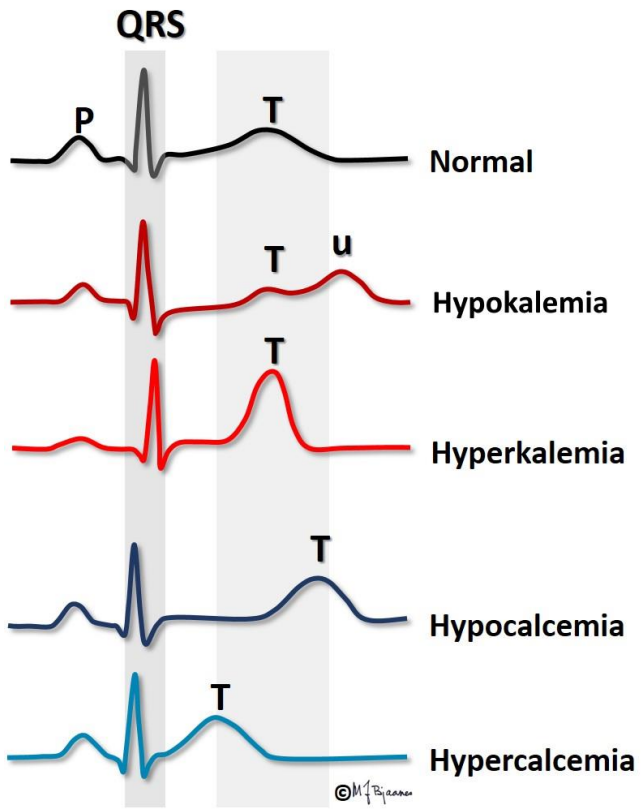
*Stable and unstable angina are distinguished by the clinical history as described earlier.
**Patients often have to undergo cardiac catheterization to distinguish this from infarction.

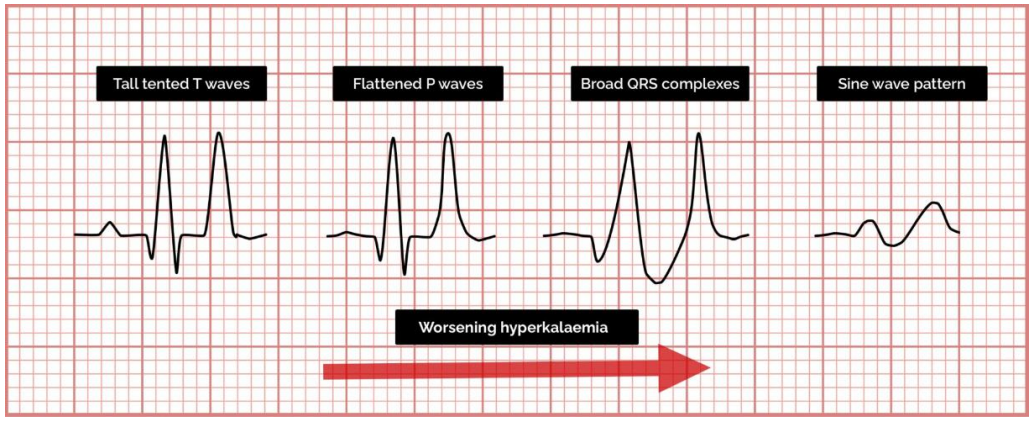


Miscellaneous Conditions

Electrolyte Disturbances

- **Hypokalemia**: ST depression, T-wave flattening, U waves. When severe, prolonged QT interval
- **Hyperkalemia**:
  - Evolution of (1) peaked T waves, (2) PR prolongation and P-wave flattening, and (3) QRS widening
  - Ultimately, the QRS complexes and T waves merge to form a sine wave, and ventricular fibrillation may develop
- **Hypocalcemia**: Prolonged QT interval
- **Hypercalcemia**: Shortened QT interval

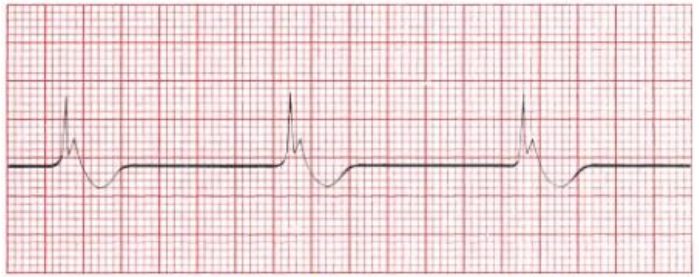




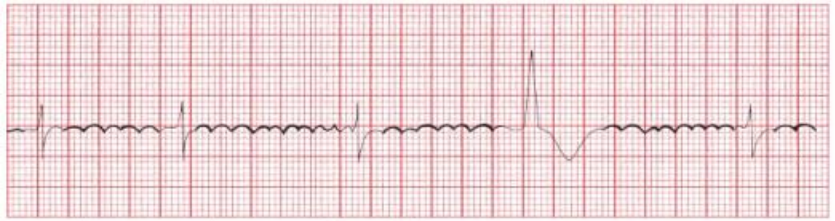
Other Conditions

❖Hypothermia

- Osborn waves (It consists of an abrupt ascent right at the J point and then an equally sudden plunge back to baseline), prolonged intervals, sinus bradycardia, slow junctional rhythms, and slow atrial fibrillation.
- Beware of muscle tremor artifact.



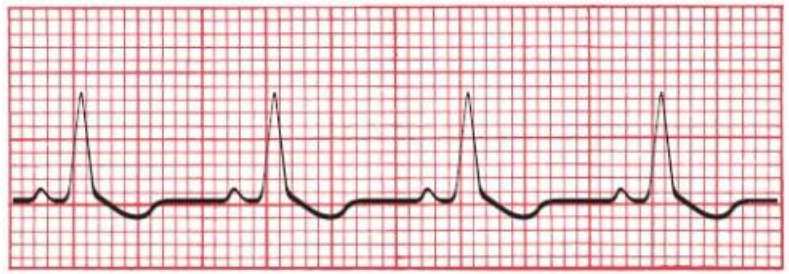
Hypothermia. The Osborn waves are very prominent.



A muscle tremor artifact resembles atrial flutter.

❖Digitalis

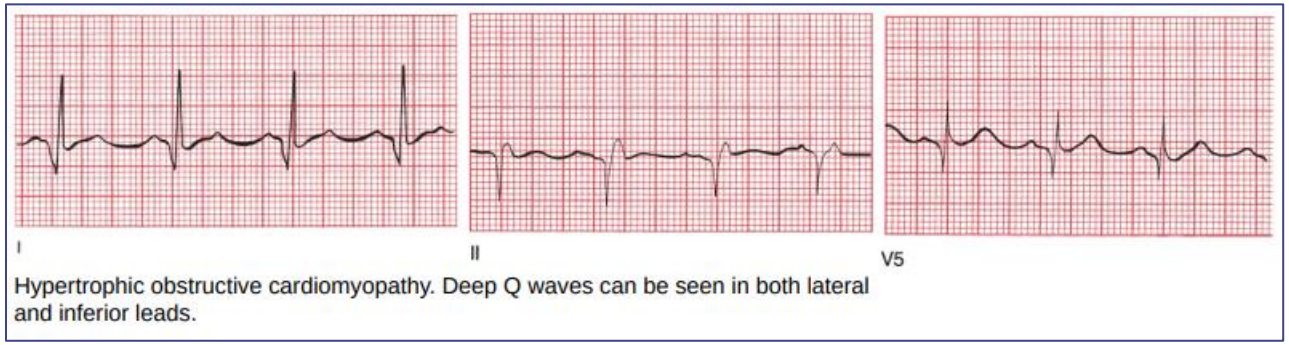
- Therapeutic levels are associated with ST-segment and T-wave changes in leads with tall R waves; toxic levels are associated with tachyarrhythmias and conduction blocks; PAT with block is most characteristic.



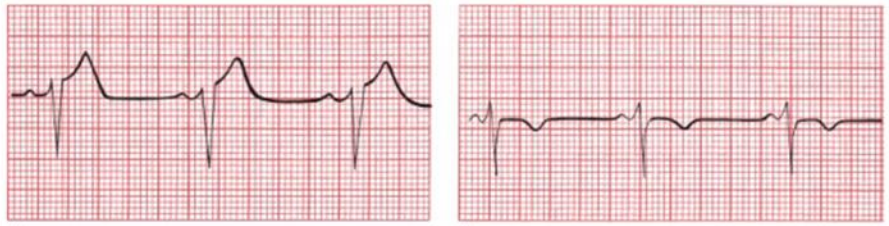
The digitalis effect, with asymmetric ST-segment depression.

Other Disorders

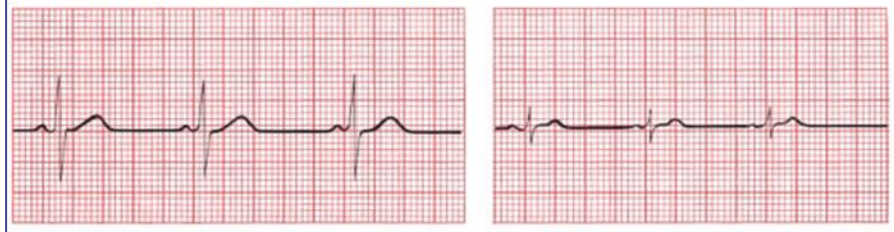
- **Pericarditis**: Diffuse ST-segment and T-wave changes, PR depression. A large effusion can cause low voltage and electrical alternans.
- **Myocarditis**: Conduction blocks.
- **Hypertrophic cardiomyopathy**: Ventricular hypertrophy, left axis deviation, inferior and lateral Q waves



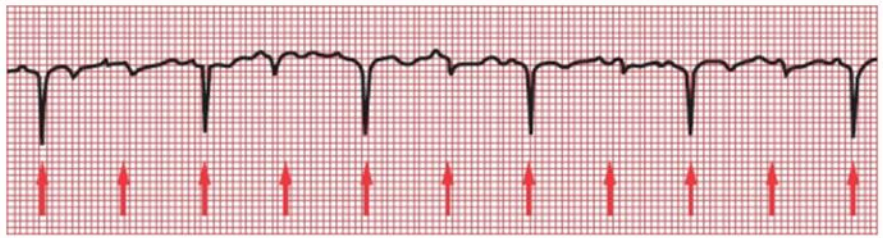
Hypertrophic obstructive cardiomyopathy. Deep Q waves can be seen in both lateral and inferior leads.



(*A*) Lead V3 shows the ST-segment elevation of acute pericarditis. (*B*) The same lead several days later shows that the ST segments have returned to baseline and the T waves have inverted. There are no Q waves.

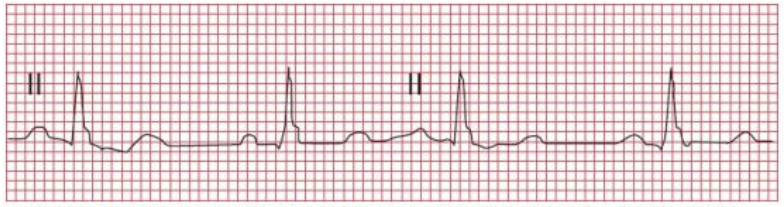


Lead I before (*A*) and after (*B*) the development of a pericardial effusion. Decreased voltage is the only significant change.

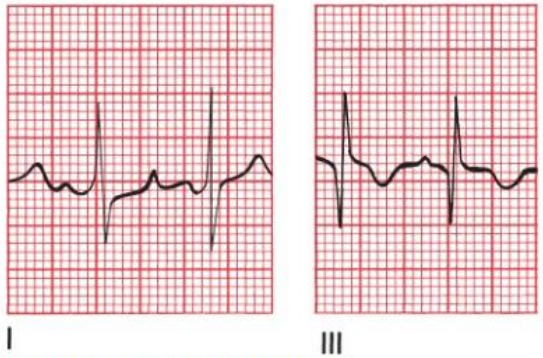

Electrical alternans. The *arrows* point to each QRS complex.

Other Disorders

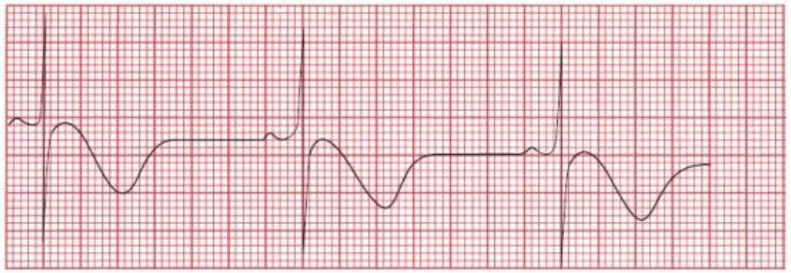
- **Atrial septal defect**:
  - First-degree AV block, atrial tachyarrhythmias, incomplete right bundle branch block, right axis deviation, QRS complex crochetage.
- **Pulmonary Disorders**
  - **COPD**: Low voltage, right axis deviation, and poor R-wave progression. Chronic cor pulmonale can produce P pulmonale and right ventricular hypertrophy with repolarization abnormalities.
  - **Acute pulmonary embolism**: Right ventricular hypertrophy with repolarization abnormalities, right bundle branch block, S1Q3 or S1Q3T3. Sinus tachycardia and atrial fibrillation are the most common arrhythmias.
- **CNS Disease**
  - Diffuse T-wave inversion, with T waves typically wide and deep; U waves



The small notch in the terminal portion of these QRS complexes is the crochetage pattern of an atrial septal defect.



The S1Q3T3 pattern of a massive pulmonary embolus.


V4
Deeply inverted, wide T waves in lead V4 in a patient with a central nervous system bleed.

The Athlete's Heart & Preparticipation Screening

- **The Athlete's Heart**
  - Nonpathologic findings can include sinus bradycardia, junctional rhythms and a wandering atrial pacemaker, nonspecific ST-segment and T-wave changes, left and right ventricular hypertrophy, incomplete right bundle branch block, first-degree or Wenckebach AV block, and a notched QRS complex in lead V1.
- Preparticipation Screening for Athletes, Findings that require further evaluation:
  - T-wave inversion beyond lead V2 in white athletes or beyond V4 in African American or Caribbean athletes
  - T-wave inversion in the lateral leads
  - ST-segment depression in any lead
  - Evidence of a congenital heart condition such as hypertrophic cardiomyopathy, long QT syndrome, Wolff–Parkinson–White syndrome, Brugada syndrome, and arrhythmogenic right ventricular cardiomyopathy

DDx of Wide Complex Tachycardias

1. Ventricular tachycardia
2. A supraventricular tachycardia with aberrant conduction (e.g., supraventricular tachycardia with underlying bundle branch block); often rate-related, appearing only with fast heart rates
3. AV reciprocating tachycardia (antidromic tachycardia) in a patient with preexcitation
4. Paced rhythms

DDx of QT Interval abnormalities

- DDx of a Prolonged QT Interval
  1. Hypocalcemia
  2. Hypomagnesemia
  3. Severe hypokalemia
  4. Congenital heart disorders
  5. Many medications
  6. Hypothermia
- DDx of a Shortened QT Interval
  1. Hypercalcemia
  2. Hyperkalemia
  3. Congenital heart disorders

How to Measure the QT Interval Accurately

Because the QT interval varies with the heart rate, a *corrected QT interval*, or QTc, is used to assess absolute QT prolongation. The QTc adjusts for differences in the heart rate by dividing the QT interval by the square root of the R-R interval—that is, the square root of one cardiac cycle:

$$QTc = \frac{QT}{\sqrt{RR}}$$

The QTc should not exceed 500 ms during therapy with any medication that can prolong the QT interval (550 ms if there is an underlying bundle branch block); adhering to this rule will reduce the risk for ventricular arrhythmias. This simple formula for determining the QTc is most accurate at heart rates between 50 and 120 beats per minute; at the extremes of heart rate, its usefulness is limited.

Causes of a Prolonged QTc Interval

- Hypocalcemia
- Hypomagnesemia
- Hypokalemia (severe)
- Congenital disorders
- Medications
- Hypothermia

ST-Segment abnormalities

Causes of ST-Segment Elevation

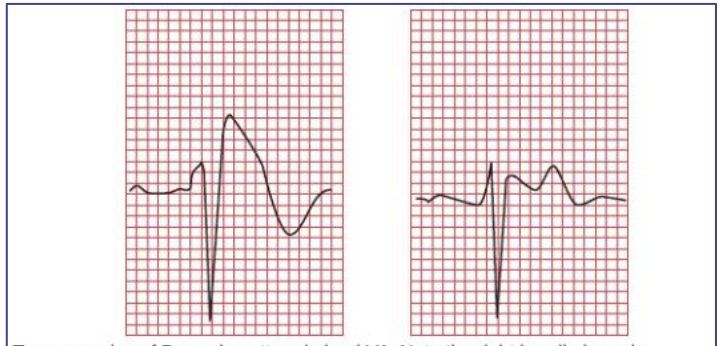
1. An evolving STEMI
2. Prinzmetal's angina
3. J point elevation/early repolarization
4. Takotsubo cardiomyopathy
5. Acute pericarditis
6. Acute myocarditis
7. Pulmonary embolism
8. Brugada pattern
9. Hypothermia (Osborne waves)
10. Ventricular aneurysm
11. CNS catastrophes
12. Postcardioversion
13. Left bundle branch block
14. Left ventricular hypertrophy
15. Paced rhythms

Causes of ST-Segment Depression

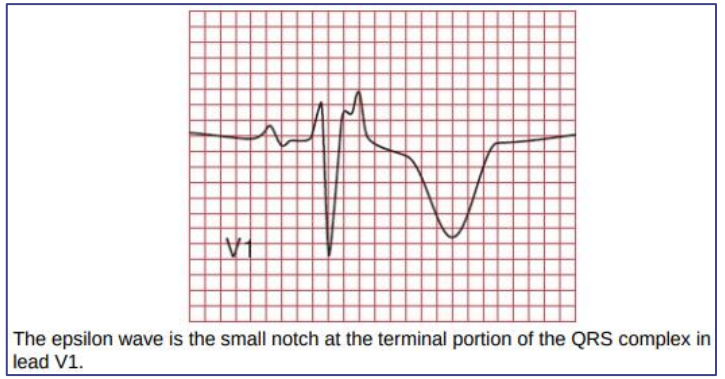
1. Stable and unstable angina without infarction
2. Non-STEMI
3. Supraventricular tachycardias—ST depression in this setting does not imply coexisting ischemic disease
4. Typically seen in leads V1–V3 with right bundle branch block
5. Hypokalemia

Causes of Sudden Cardiac Death

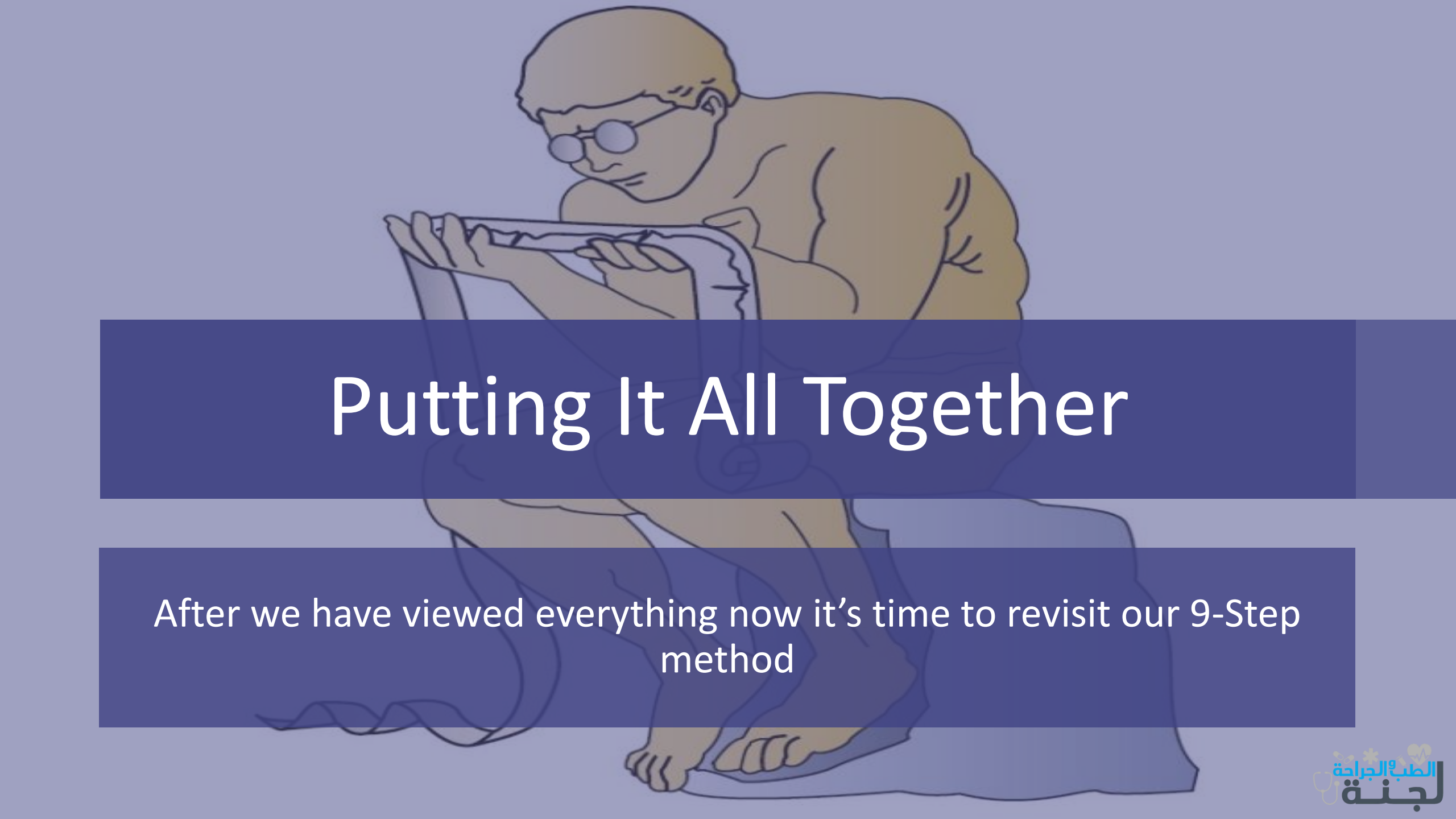
1. Coronary artery disease
2. Hypertrophic cardiomyopathy
3. Long QT syndrome
4. Wolff–Parkinson–White syndrome
5. Viral pericarditis/myocarditis
6. Infiltrative diseases of the myocardium
7. Valvular heart disease
8. Drug abuse (especially stimulants)
9. Trauma (commotio cordis)
10. Anomalous origin of the coronary arteries
11. Brugada syndrome: right bundle branch pattern with ST elevation in V1-V3
12. Arrhythmogenic right ventricular cardiomyopathy—may see an epsilon wave in terminal portion of QRS



Two examples of Brugada pattern in lead V1. Note the right bundle branch appearance and the inverted T wave in V1. The ST-segment elevation can appear coved (first figure) or saddle-backed (second figure).



The epsilon wave is the small notch at the terminal portion of the QRS complex in lead V1.



Putting It All Together

After we have viewed everything now it’s time to revisit our 9-Step method

The 9-Step Method for Reading EKGs

Before you start, make sure the standardization mark on the EKG paper is 10 mm high so that 10 mm = 1 mV. Also make sure that the paper speed is correct.

1. **Determine the heart rate**. (300/# of large squares)
2. Measure the length of the **PR** and **QT intervals** and the width of the **QRS complexes**.
3. Is the **axis** of the P waves, QRS complexes, and T waves normal, or is there axis deviation? (look at lead I & aVF)
4. **Rhythm**. Always ask The Four Questions:
    a. Are there normal P waves present?
    b. Are the QRS complexes wide or narrow?
    c. What is the relationship between the P waves and QRS complexes?
    d. Is the rhythm regular or irregular?

The 9-Step Method for Reading EKGs

5. **Conduction blocks**
   a. Atrioventricular (AV) block.
   b. Bundle branch block (QRS complex widened) or hemiblock (Axis Deviation).
6. **Preexcitation**, Criteria for WPW (PR interval less than 0.12 seconds, Wide QRS complexes, Delta wave seen in some leads).
7. Apply the criteria for both **atrial enlargement** and **ventricular hypertrophy**.
8. **Coronary artery disease**: Look for Q waves and the ST-segment and T-wave changes. Remember that not all such changes reflect coronary artery disease; know your differential diagnoses.
9. Other conditions. Is there anything on the EKG that suggests one of the other cardiac or noncardiac conditions discussed in the miscellaneous conditions? Are you totally lost? Never hesitate to ask for assistance.

And if you are still thinking, “Is this really all I need to know?” The answer—reminding you that information only becomes knowledge with wisdom and experience—is, “Yes!”