Chapter 2
Evaluation of Heart Rhythms

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Introduction

The electrocardiogram (ECG) is synonymous with a cardiac evaluation, just as a chest x-ray is with a pulmonary evaluation. The ECG is inexpensive, non-invasive and easy to perform. All hospitals and most offices can perform electrocardiography and because they are so ubiquitous, being able to accurately interpret an ECG is important.

The goal of this chapter is to provide a basic understanding of the principles of how to "read" an ECG, why an ECG looks the way it does, and when to order an ECG. Because the readers of this book will likely have quite varied backgrounds and exposures to ECG interpretation, this chapter will begin with more basic principles of what an ECG is and how it is generated. We will then focus on understanding the ECG waveforms and basic rhythm diagnosis. Finally, we will

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conclude with a discussion of additional ECG tests that can be performed to assess heart rhythm disturbances.

Electrocardiogram

The ECG provides a momentary picture of the electrical activity of the heart. With every beat of the heart, each heart cell depolarizes, meaning that electrolytes flow into and out of the heart cells, generating an electrical signal that provides the stimulus for the cells to contract. When enough cells depolarize together, this electrical signal becomes strong enough to be measured outside of the body. To produce an ECG, electrodes are attached to the skin and connected via wires to a machine. The machine amplifies the minute electrical signals from the heart that reach the electrodes on the skin and then filters out many unwanted signals. The machine will then display or print out a summation of the electrical activity detected by each lead. Sometimes an ECG is referred to as an EKG, which is an abbreviation of the German word *elektrokardiogram*. The standard appearance of an ECG is shown in Fig. 2.1.

When setting up an ECG, a varying number of wires can be connected to the patient. A minimum of two wires must be attached, providing a positive and negative electrode, which

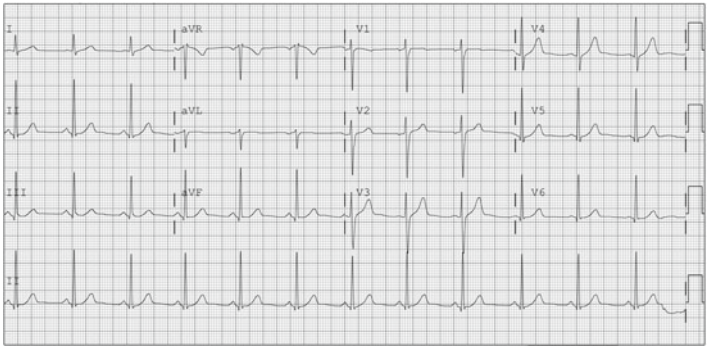


Figure 2.1 Standard 12-Lead ECG in sinus rhythm

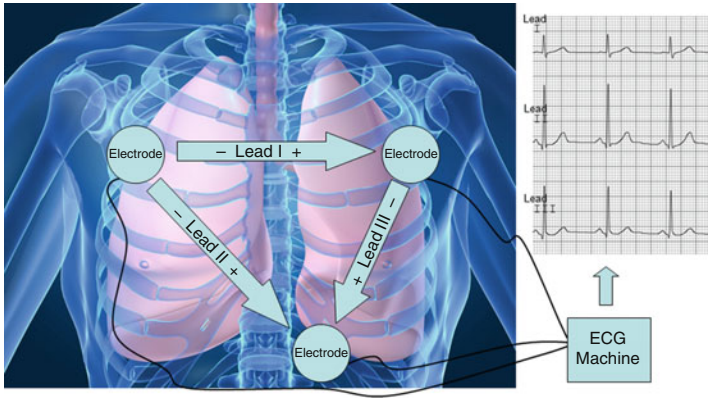


FIGURE 2.2 Einthoven's triangle. The electrode at the patient's right arm is typically always negative. The electrode towards the patient's left leg is typically always positive. The electrode at the patient's left arm can be positive or negative depending on which lead being evaluated. True bipolar leads, such as these, must always have a positive and a negative electrode

is necessary to measure changes in the electrical potential that occur with each heart beat.

The term "lead" is used to describe the electrical picture these two wires generate. While some use the term "lead" to describe the actual wire connected to the patient, technically, the correct use of "lead" is in reference to the picture that positive and negative electrodes generate together. Traditionally, three wires have been connected to the patient to form a triangle. Figure 2.2 shows what has been termed Einthoven's triangle. With these three wires, three different electrical pictures of the heart are generated, or in other words, three different "leads." These leads may show a waveform that has an upward or downward deflection depending on the direction of the electrical signal relative to that lead. By convention, a positive deflection is recorded for that lead when the electrical signal moves toward the positive electrode of that lead, and a negative deflection when electrical activity moves away from the positive electrode.

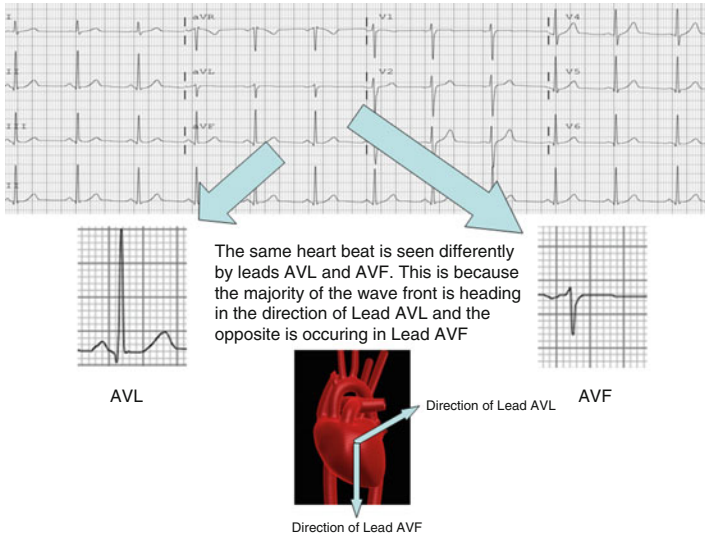


FIGURE 2.3 Different leads show a different picture of the same heart beat. Even though Lead AVL and Lead AVF are displaying the same heart beat, the waveform appears differently because in Lead AVL, the direction of electrical depolarization is away from this lead and in Lead AVF, the direction of electrical depolarization is towards this lead

Thus, it is important to realize that depending on the orientation of the electrode, the same electrical signal from the heart can be either upright or inverted as seen in Fig. 2.3. Analysis of each lead vector, therefore, provides spatial information about the electrical activity of the heart.

The heart is composed of four chambers, two upper chambers known as atria and two lower chambers know as ventricles. The sino-atrial (SA) node, which is located in the right atrium, is a group of specialized heart muscle cells whose primary function is to automatically generate an electrical signal, typically 60–100 times per minute. This signal is carried from heart cell to heart cell, thus setting the heart rate and providing the stimulus for each chamber of the heart to contract. The specialized cells of the SA node have a unique ability to

spontaneously depolarize. This electrical signal then spreads throughout the heart, first traveling through both atria to the atrio-ventricular (AV) node and then to the ventricles. The wave of depolarization through the atria can be seen on the surface ECG as a p-wave. When the signal reaches the AV node, the signal slows. In a normal heart, the AV node is the sole electrical connection between the top two chambers (atria) and the two bottom chambers (ventricles) of the heart. Thus, all of the electrical signals from the atrium must travel through the AV node to reach the ventricles.

The AV node is also composed of specialized cells with slightly different electrical properties than the SA nodal cells. As a result of these differences, AV nodal cells can slow the conduction of the electrical signal. This slowing serves two purposes. First, it allows time for atrial contraction to occur prior to activation of the ventricles. Second, the slowing is a protective mechanism, limiting rapid conduction of fast impulses from the atrium to the ventricle as occurs in atrial fibrillation.

Conduction from the SA node through the AV node is seen as the PR interval on the ECG. Once the signal exits the AV node, the signal again speeds up and travels through the bundle of His to the bundle branches and into the Purkinje fibers, which are embedded in the inner lining (endocardium) of the left and right ventricles. Depolarization of the ventricles is seen as a QRS complex on the ECG.

Repolarization, which follows depolarization, is a term used to describe the recovery of heart cells back to their electrical resting state. Just as an electrical signal is generated as the heart depolarizes, a reverse electrical signal is generated when the heart repolarizes. Atrial repolarization is not seen on the ECG because the timing is coincident with ventricular depolarization (i.e. QRS complex), and the atrial mass (and hence the electrical signal) is relatively small compared to the ventricles. Conversely, ventricular repolarization is clearly evident on the ECG and is seen after the QRS as a t-wave. Occasionally u-waves are seen and the etiology of these waves is less well understood. Some have hypothesized that these are delayed after-depolarizations of the ventricles. A detailed explanation of these activities is shown in Fig. 2.4.

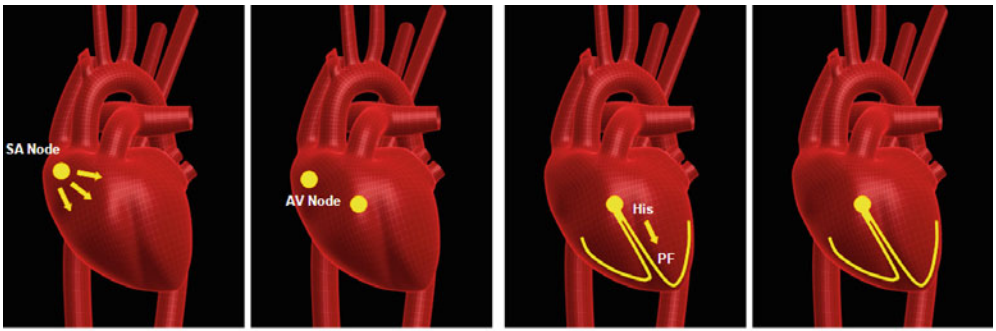
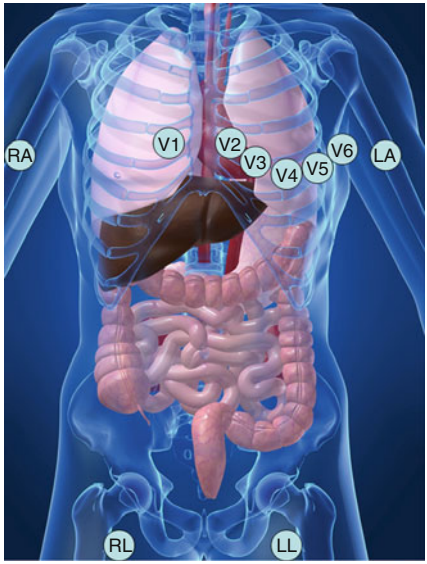


FIGURE 2.4 Simplified physical and electrical structure of the heart



The precordial electrodes are placed on the chest. V1 and V2 are placed right and left of the sternum, respectively, in the 4th intercostal space. V4 is placed in the 5th intercostal space, mid-clavicular line, and V6 is placed in the 5th intercostal spaced, mid-axillary line. V3 and V5 are placed in between these electrodes. Electrodes are then also placed on the right arm, left arm, right leg, and left leg.

FIGURE 2.5 Attaching a 12-lead ECG. Reference 1: Guidelines for electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Committee on Electrocardiography). JACC. 1992;19:473–81

Various forms of ECGs are routinely performed and differ mostly by the number of leads used and the duration of monitoring. Inpatient units, including cardiac care units, have patients on "telemetry." Telemetry typically involves five wires attached to the patient's chest that connect to a small transmitter. The transmitter sends the heart's electrical signals to a display monitor located at a nursing station, providing continuous tracings of the heart rate and rhythm. These monitors, which are observed by nurses and providers, are typically set up to alarm for abnormal heart rates or rhythms. Telemetry units are fully portable, allowing patients to be mobile as long as they remain within range of the monitoring station.

The most common form of the ECG is the "12-lead ECG." The 12-lead ECG typically involves attaching ten wires to the patient, as seen in Fig. 2.5. However, from these ten wires, the

ECG machine is capable of creating 12 electrical signals as shown in Fig. 2.1. Wires that are attached to the chest generate the precordial leads (V1-V6), while wires attached to the arms and legs create the limb leads (I, II, III, AVR, AVL, AVF). Each of these leads provides different spatial information about the orientation of the heart's electrical activity.

Leads I, II and III are also termed true bipolar leads because there is an actual single positive wire and single negative wire attached to the patient that is used to measure the electrical signal of the heart beat. The right arm wire is traditionally attached to the negative terminal of the ECG machine and the left arm to the positive terminal of the ECG machine. The electrical signal is then amplified and recorded as Lead I. In Lead II, the right arm wire is negative and the left leg wire is positive. Finally, in Lead III, the left arm wire is negative and the left foot wire is positive as shown in Fig. 2.2. Thus, each heart beat produces a slightly different electrical picture in each lead, depending on the direction of the electrical impulse relative to that lead. Different leads can therefore provide different information about the electrical activity of the heart. Waveforms that are not clearly evident in one lead may be quite obvious in another.

Another three limb leads seen on the 12-lead ECG are called the augmented limb leads, which are labeled as AVR, AVL, and AVF. These leads are called unipolar leads because the electrical potential of each individual wire is compared to a combined electrical potential created from the two remaining wires. In other words, the computer measures the electrical potential of the individual wire, which acts as the positive pole, and compares it to a composite negative pole calculated from the electrical signals of the two remaining wires. For example, the AVR lead, which stands for augmented vector right, compares the electrical potential of the right arm wire to that of a combined potential of the left arm wire and left leg wire. AVF compares the potential of the left leg to that of the left and right arm combined. AVL compares the potential of the left arm to the combined potential of the left leg and right arm. Again, the augmented limb leads are measured to produce a different

electrical snapshot of the heart which may provide additional insight into the origin of an arrhythmia.

Lastly, in a 12-lead ECG, precordial leads are recorded. Like the augmented limb leads, these are unipolar leads. The ECG machine calculates a virtual negative pole based on the different wires attached to the patient. Each lead, V1 through V6, is then a representation of the electrical potential between the individual electrode attached to the patient's chest wall and the virtual negative pole generated by combining the potential of the remaining precordial leads.

Many clinical scenarios warrant ordering an ECG. Often, an ECG is ordered to evaluate for ischemia. Other indications include following cardiac procedures, including coronary angiography or electrophysiology studies and ablations. When administering anti-arrhythmic therapy, serial electrocardiograms should be obtained. Congenital heart disease, valvular heart disease, syncope, and conditions such as stroke which have a cardiac association warrant an ECG. In general, a 12-lead ECG should be ordered anytime further evaluation of the heart beyond the physical exam is desired. Please see the referenced guidelines (ACC/AHA Guidelines for Electrocardiography. JACC 1992) for specific indications.

Reading an ECG

The key to reading ECGs well is to be consistent, using the same method each time. This can sometimes be difficult when an obvious diagnosis is present (such as a very slow heart rhythm or a myocardial infarction), but ensures that other abnormalities will not be overlooked. A common approach is *rate*, *rhythm*, *axis*, *intervals*, and *changes*, which is described below.

When you first look at an ECG, take note of the *rate*. The heart rate is considered normal if it is between 60 and 100 beats per minute (bpm). If the rate is >100 bpm, then the rate is considered fast and the term "tachycardia" is used to describe the rate. This may still reflect a normal heart rhythm, for instance, if the subject is exercising, the heart is expected to speed up to above 100 bpm. Conversely, this may reflect an

abnormal heart rhythm, which, if originating from the two top chambers (i.e. atria), would be called a supraventricular tachycardia (SVT), or if originating from the two lower chambers (i.e. ventricles) would be called a ventricular tachycardia (VT). These abnormal heart rhythms (arrhythmias) will be described further in a separate chapter of this book.

If the rate is <60 bpm, then the rate is considered slow and the term "bradycardia" is used to describe the rate. Again, this may reflect a normal heart rhythm. For instance, well-trained athletes often have slower baseline heart rates than the general population because their heart is conditioned to produce a larger stroke volume with each beat of the heart, thus allowing the heart to beat more slowly and still pump the same amount of blood. It can however reflect an abnormal heart rhythm, for which a patient may require a pacemaker. Keep in mind that the "normal" range for heart rates were decided upon in a somewhat limited manner, and many 20–30 year old people have a normal heart rate that is <60 bpm and many normal children >100 bpm. Nevertheless, these are the numbers that are typically used.

The next item to evaluate on an ECG is the *rhythm*. When looking at the ECG strips, first look to see if any atrial activity is present, as manifested by p-waves. If you do not see any p-waves specifically, that does not mean there is no atrial activity. The atria may be in atrial fibrillation, which can appear as a squiggly baseline between QRS complexes. Other causes of unapparent p-waves include sick sinus syndrome, sinus-node exit block, a junctional/ventricular rhythm or tachycardia that is obscuring the p-waves.

Once p-waves are identified, the next step is to see if each p-wave precedes the QRS and if the p-wave appears to be originating from the sinus node. Recall that the spread of atrial depolarization begins in the SA node in the right atrium and travels toward the ventricles. Thus, a p-wave that originates in the sinus node would be expected to be upright in leads I and II (i.e. signal flows toward those leads) and negative in lead AVR (i.e. signal flows away from this lead) which is shown in Fig. 2.1. The term sinus rhythm is used to describe a rhythm originating from the SA node. If the rate is

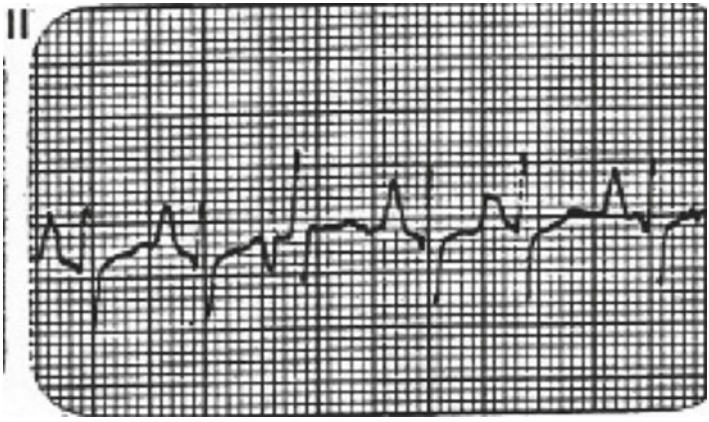


FIGURE 2.6 Multifocal atrial tachycardia. p-waves with different morphologies precede the QRS complex

>100 bpm, then the term sinus tachycardia would be applied. If the rate is <60 bpm, then the rhythm would be called sinus bradycardia.

If the p-waves are not upright in leads I and II, or negative in AVR, then it is likely that an ectopic atrial rhythm or junctional rhythm is present. An ectopic atrial focus means that the p-wave is originating from a site outside of the SA node, but still within the right or left atrium. In other words, the stimulus for the heart to beat is coming from outside the SA node but still within the atrium. If the ectopic atrial rate is >100 bpm, then the term "atrial tachycardia" is used. When three or more different p wave morphologies are seen on the ECG, meaning that three or more different sites in the atrium are causing the heart to beat, the term "wandering atrial pacemaker" is applied when the rate is <100 bpm or "multifocal atrial tachycardia" when the rate is >100 bpm (Fig. 2.6).

A junctional rhythm is considered if the PR interval is very short (i.e. <120 ms) and the p-wave appears inverted in lead II. At times, the p wave actually comes after the QRS complex. In these cases, the AV node is the source of the atrial activity. The signal that originates in the AV node travels in two directions, retrograde to the atrium resulting in atrial

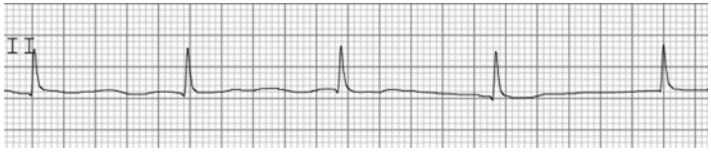


Figure 2.7 Junctional rhythm. No p-waves are present and the distance between each QRS complex (R-R interval) is fairly regular

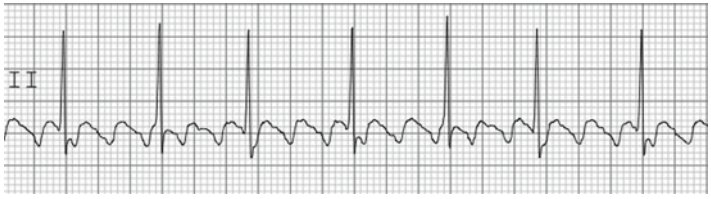


Figure 2.8 Atrial flutter

depolarization, and antegrade to the ventricles resulting in ventricular depolarization (Fig. 2.7).

Sometimes, the p-waves appear in a "saw tooth" pattern. These p-waves reflect continuous atrial activation and often occur at a frequency of around 300 bpm. These waves are called flutter waves, and the rhythm is known as atrial flutter (Fig. 2.8). Atrial flutter is a macro-reentrant arrhythmia in the atria, or in other words, an endless electrical loop that often circulates around the tricuspid valve. This type of flutter is called typical counter-clockwise atrial flutter and the ECG demonstrates inverted flutter waves in the inferior leads (leads II, III, and AVF) and upright flutter waves in V1. Clockwise flutter is characterized by positive flutter waves in the inferior leads and negative flutter waves in V1. Atypical flutters are flutters that do not involve the tricuspid annulus, and they often have slower flutter wave rates or mimic clockwise flutter.

If p-waves are not visible, then the next step is to determine if atrial fibrillation is present. This rhythm appears as a coarse, undulating baseline between QRS complexes, and the QRS complexes appear in an irregularly irregular pattern as shown in Fig. 2.9. Atrial fibrillation occurs when multiple areas of the

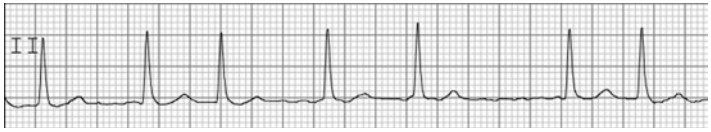


Figure 2.9 Atrial fibrillation

atrium are firing at the same time. This creates multiple electrical wavefronts and results in a lack of any organized electrical rhythm that would be recognized as a p-wave. Incidentally, it is this lack of organized electrical rhythm that results in loss of organized mechanical contraction of the atrium, which can result in stasis of blood and the formation of blood clots. Thus, patients with atrial fibrillation are at risk for strokes, which can occur if these blood clots form and embolize to the brain. Patients with atrial fibrillation must be evaluated for their risk of stroke using various risk assessment tools, such as the CHADS-2 score, as mentioned elsewhere in this book.

At times, however, p-waves are not present or are unapparent. Sinus node dysfunction, such as sick sinus syndrome or sinus exit block, junctional or ventricular rhythms are suspected in these instances, particularly when associated with a regular QRS pattern.

We already mentioned junctional rhythm above. Junctional rhythms can occur without visible p-waves on the ECG when the p-waves are buried in the QRS complex. The width of the QRS complex can help differentiate between a rhythm coming from the AV node, as in the case of junctional rhythms, or from the ventricles, which lie below the AV node. If the QRS complex is narrow (<120 ms), then this is likely a junctional rhythm. However, if the QRS is wide (≥120 ms), then a ventricular rhythm likely exists.

The QRS morphology is wide in ventricular rhythms because the origin of the beats is in the ventricles, not the conduction system. Since conduction of the electrical impulse through the ventricular muscle occurs more slowly outside of the conduction system, the QRS morphology is wide, as seen in Fig. 2.10. Exceptions to this include aberrantly conducted

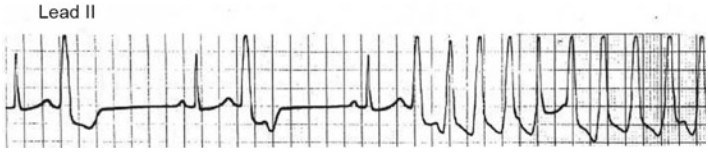


FIGURE 2.10 Ventricular tachycardia. There are two normal appearing beats, each with a wide complex early beat coming after. These single, early beats are termed premature ventricular contractions (also known as PVCs or VPCs). When these early beats sustain, this is called ventricular tachycardia

supraventricular beats. Aberrantly conducted beats mean that the rhythm is generated from the atria and conducted through the AV node; however, conduction is slowed through one of the bundle branches because of disease or drug effect, thus giving the appearance of a wide QRS complex. To differentiate ventricular rhythms from aberrantly conducted supraventricular beats, other aspects of the ECG must be examined. For instance, if the QRS is wide but the R-R intervals are irregular, atrial fibrillation may be occurring. Of note, atrial fibrillation may be suspected in Fig. 2.10 because some of the wide-complexes appear in an irregularly irregular pattern. However, p-waves can be seen in the ST/ T wave segments of the some of the beats such as the second and third wide complex beats, thus showing some organized atrial electrical activity and excluding atrial fibrillation as a diagnosis. Over the years, various criteria have been developed to distinguish ventricular tachycardia (VT) from aberrantly conducted SVT. The two most useful ones have been the Brugada criteria and the AVR (Vereckei) criteria (Fig. 2.11). It is important to learn at least one of these criteria schemes as when you are faced with a wide-complex, fast tachycardia, the treatment of VT is typically vastly different than that of an SVT as you will see later in this book.

One other very important ventricular rhythm to identify is ventricular fibrillation (Fig. 2.12). As with atrial fibrillation, ventricular fibrillation occurs when multiple areas of the ventricle fire rapidly and chaotically. This arrhythmia is life-

VT Criteria

Vereckei-Looking at Lead AVR	**Brugada**
VT if…	VT if…
• Broad monphasic R-wave in AVR	• Absence of RS complex in all precordial leads
• Initial r or q wave that is >40 msec	• R to S >100 msec in any precordial leads
• Notch on descending limb of negative QRS complex	• AV dissocaition
• Terminal electrical potential > initial electrical potential	• Morphologic VT Criteria both in $V_{1\text{-}2}$ and V_6

FIGURE 2.11 VT criteria – AVR (Vereckei) versus Brugada

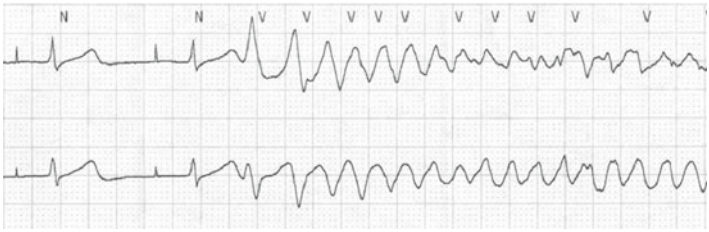
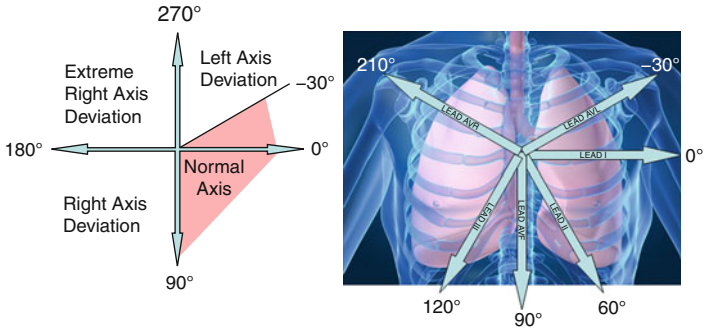


FIGURE 2.12 Ventricular fibrillation

ending if not treated. The treatment of this is direct-current, electrical defibrillation.

Axis is used to describe the overall direction of electrical current in the heart. One can evaluate the axis of p-waves, QRS complexes and t-waves. Usually, the axis of interest relates to the QRS complex, as this provides additional information including the overall orientation of the heart. For example, the electrical axis of the heart shifts leftward in the presence of increased left ventricular mass (i.e. LV hypertrophy) as occurs in chronic hypertension. Conversely, scar from a prior myocardial infarction diminishes the electrical activity in the area of scar shifting the axis in the opposite direction of the scar. If part of the conduction system is failing, such as in a hemi-block, then the axis will be shifted away from the direction of the block.

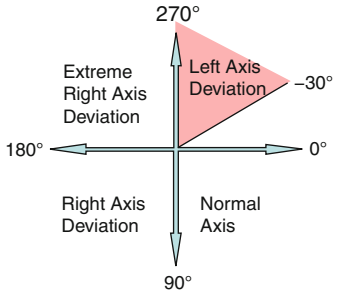


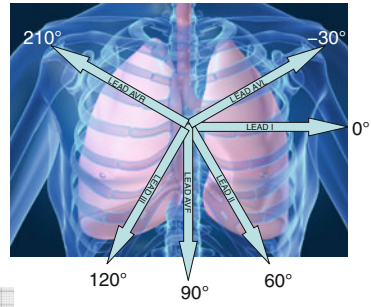
Axis of the QRS complex is determined by the direction of the QRS complex in each of the limb leads. Normal axis is between –30° to 90°, which would be a QRS complex that is positive in leads I, II, and AVF

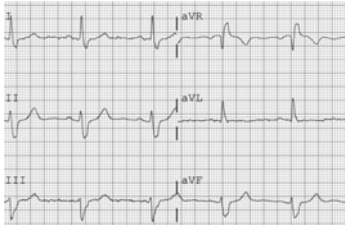
FIGURE 2.13 Determining axis and normal axis

The axis of the heart that is typically evaluated is in the frontal (i.e. coronal) plane. To do this, we look at the major deflection of the QRS in different limb leads which lie in the frontal plane as shown in Fig. 2.13. The direction of the QRS deflection will be upright in leads that the electrical signal is flowing towards and negative in leads that the electrical signal is moving away. The normal axis of the heart is –30° to 90°. Left axis deviation is present when the QRS axis is –30° to –90° (Fig. 2.14). Right axis deviation is present when the QRS axis is 90°–180°, as shown in Fig. 2.15. Extreme axis or northwest axis deviation is present when the QRS axis is 180°–270° as shown in Fig. 2.16.

The next step of the evaluation is to measure the *intervals*. Basic intervals are listed in Table 2.1. The first interval to evaluate is the P-R interval. The P-R interval, a measure of the conduction time from the sinus node to the ventricles, is typically 120–200 ms. A PR-interval that is >200 ms is abnormal and suggests delay in signal conduction from the SA node to the ventricles. The term "first degree AV block" is used to describe a PR interval that is consistently >200 ms (Fig. 2.17). A short PR interval (i.e. <120 ms) can be normal, but rhythms such as a junctional rhythm or the presence of accessory pathways, as occurs in Wolf–Parkinson–White syndrome, should be considered.

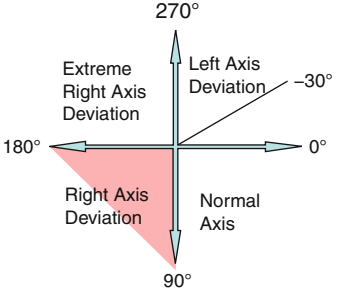


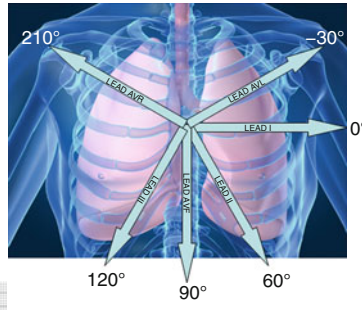


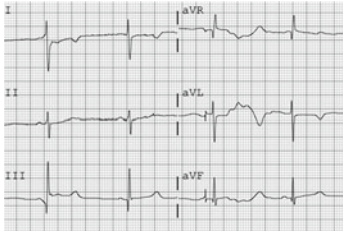


In left axis deviation, the axis of the heart is from –30° to –90° (which is equal to 270° to 330°). Here, the QRS complex is positive in Lead I, but negative in Lead II and Lead AVF.

FIGURE 2.14 Left axis deviation







In right axis deviation, the axis of the heart is from 90° to 180°. Here, the QRS complex is positive in Lead III, but negative in Lead I and Lead II

FIGURE 2.15 Right axis deviation

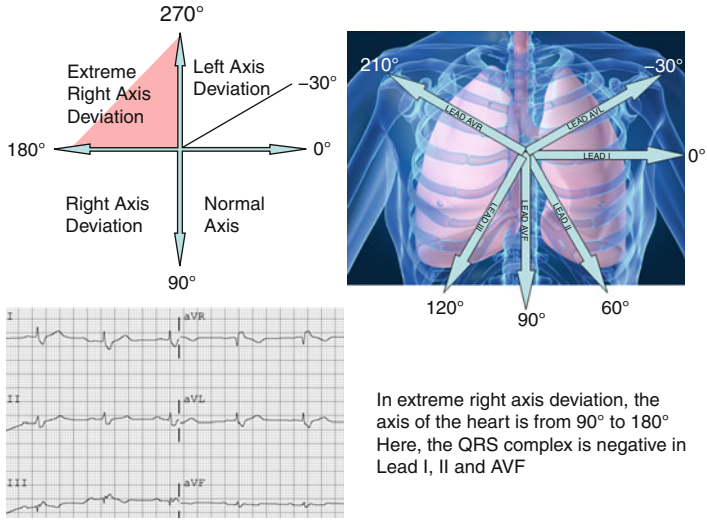
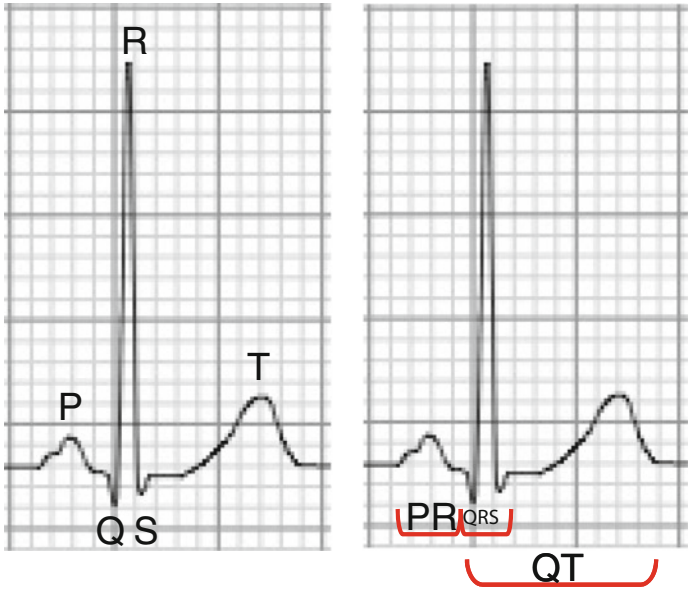


FIGURE 2.16 Extreme right axis deviation

Second degree AV block is present if there is intermittent loss of conduction between the atria and ventricles. In Type I, second degree AV block, there is progressive delay in the electrical conduction from the atrium to the ventricle. Therefore, the ECG shows a gradual increase in the PR interval until there is block in the conduction within the AV node, at which point there is absence of a QRS complex following the p-wave. Figure 2.18 shows this type of block with a missing QRS complex after a p-wave that can be seen buried in the t-wave. Type II, second degree AV block occurs when there is abrupt loss of conduction between the atrium and ventricle. In this case, the PR interval is constant both before and after a non-conducted p-wave, as shown in Fig. 2.19. If there is no association between the p-waves and the QRS complexes, then atrio-ventricular dissociation is present. If there are more p-waves than QRS complexes, then complete heart block (i.e. third AV block) is present, which is a form of AV dissociation (Fig. 2.20).

Table 2.1 Basic intervals



	Normal Interval
PR	120-200 msec
QRS	<100 msec
QT	Men 440 msec Women 460 msec

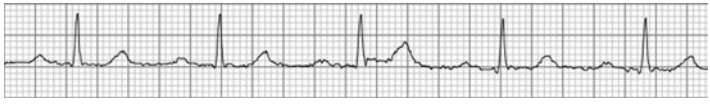


Figure 2.17 First degree AV block – the PR interval is prolonged

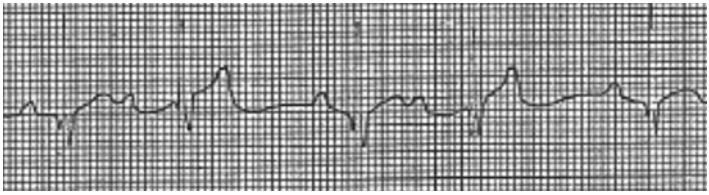


FIGURE 2.18 Second degree AV block, type I (Wenkebach) – The PR interval prolongs until the point that it no longer conducts to the ventricles and there is no QRS complex

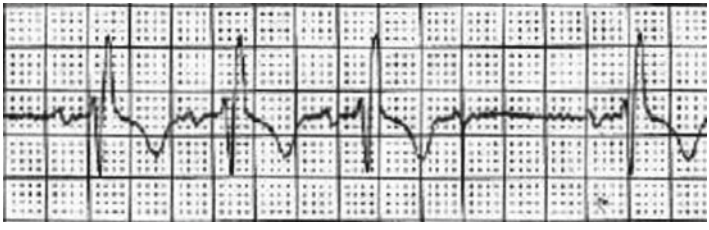


FIGURE 2.19 Second degree AV block, type II – the PR interval is fixed but intermittently the AV node does not conduct to the ventricle which results in a missing QRS complex

The next interval to evaluate is the QRS duration. This interval reflects the time it takes for electrical activation of the ventricles. Recall that when the electrical signal travels from the atrium to the ventricles, the signal travels through the AV node where it is temporarily slowed, then through the His into the left and right bundle branches to the Purkinje fibers embedded within the wall of the ventricles. The normal QRS duration is 60–100 ms. The term "incomplete" bundle branch block is used when the QRS duration is between 100 and 120 ms, suggesting conduction through one of the bundle branches is slower than normal. Complete bundle branch block is used when the QRS duration is >120 ms, suggesting absence of conduction through that bundle. Bundle branch block is further differentiated into either right or left depending on whether the right or left bundles are affected. The QRS pattern helps differentiate between right and left bundle

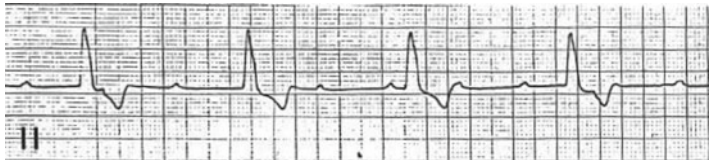


FIGURE 2.20 Third degree AV block – more p-waves than QRS complexes and no regular intervals between the p-waves and the QRS complexes

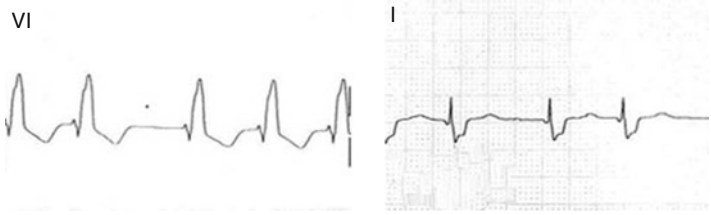


FIGURE 2.21 Right bundle branch block

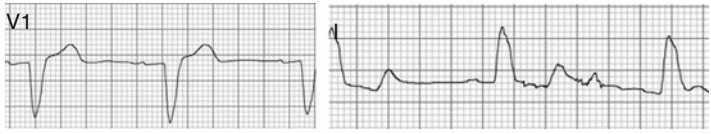


FIGURE 2.22 Left bundle branch block

branch blocks, as shown in Figs. 2.21 and 2.22. If the QRS is wide and does not appear to follow one of these two patterns, the term "non-specific inter-ventricular conduction delay" is used to describe the pattern, which suggests that there may be more than one conduction abnormality present or scar.

The easiest way to differentiate right and left bundle branch blocks is to evaluate the QRS morphology in leads V1-V2 and leads I and V6. It is important to note the position of these leads relative to the heart. V1 is positioned over the right ventricle, while V2 is positioned over the septum. Leads I and V6 are lateral leads, providing electrical information about

the left ventricle. In right bundle branch blocks, conduction down the right bundle is either slowed or absent leading to delayed activation of the right ventricle. Thus, the initial deflections of the QRS reflect left ventricular activation, while the terminal portions of the QRS reflects right ventricular activation. The typical ECG pattern of RBBB is rsr', rsR' or rSR' in leads V1-V2 (often referred to as "rabbit ears" pattern) and a terminal S wave in leads I and V6. The R' and S waves in these leads reflect the electrical activity of the right ventricle.

In left bundle branch blocks, conduction down the left bundle is either slowed or absent leading to delayed activation of the left ventricle. Thus, the initial deflections of the QRS reflect right ventricular activation, and the terminal portions of the QRS reflect left ventricular activation. The typical ECG pattern of LBBB is a broad or notched r-wave in leads I and V6 and QS or S waves in V1-V2.

The last main interval to assess is the QT segment. This measures the time it takes for the ventricles to depolarize and repolarize. If the ventricle takes extra time to repolarize, then that patient has a higher risk of having a potential life-threatening arrhythmia, such as polymorphic ventricular tachycardia. This is an often overlooked, but important interval to measure. The time it takes for the ventricle to repolarize is dependent on the heart rate. When the heart beats faster, the heart will reset faster. Thus, the QT interval is often corrected for rate. Bazett's formula is often used to calculate this and is defined as corrected QT interval (QTc) = QT/square root of the R-R interval. If the QTc is >440 ms in men or >460 ms in women, it is considered prolonged. See Fig. 2.23 for an example of a long QT interval.

In the final evaluation of the ECG, we look for any morphologic changes in each waveform, specifically, the p-waves, the PR intervals, the QRS complexes, the ST segments and the t-waves. The amplitude and duration of these waves can provide information about chamber sizes. For example, the amplitude (i.e. height) of the QRS complex increases in conditions like ventricular hypertrophy because the larger tissue mass generates greater electrical signals. This is why QRS complexes are larger than p-waves.

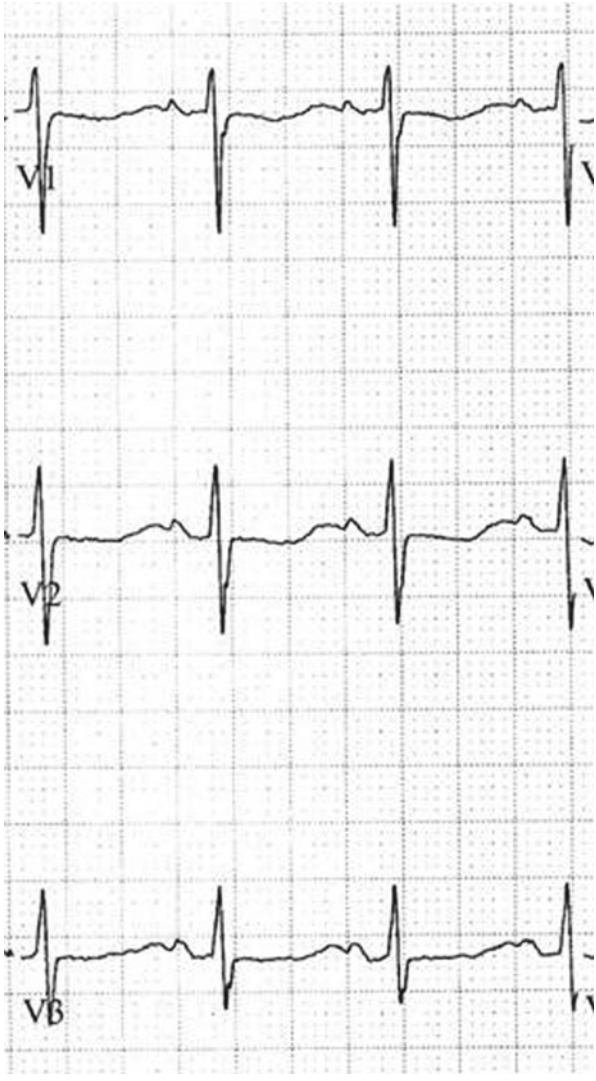


FIGURE 2.23 Long QT interval

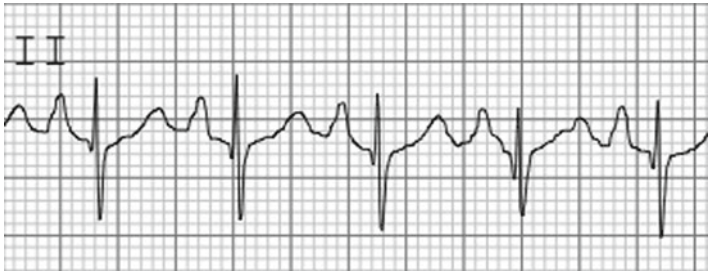


Figure 2.24 Right atrial enlargement – manifested by tall p-waves

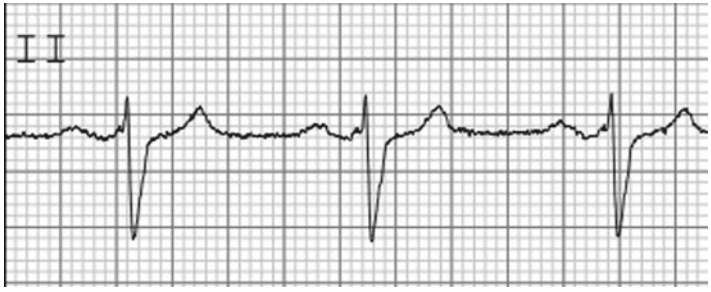


Figure 2.25 Left atrial enlargement – manifested by broad p-waves

The p-waves represent atrial electrical depolarization. Tall and/or broad p-waves suggest that there is likely increased atrial mass. Since the atria do not typically hypertrophy, this increased p-wave size represents increased volume. An important point to keep in mind is that right atrial activity is reflected in the initial portions of the p-wave, while left atrial activity is reflected in the terminal portions. Right atrial enlargement appears as p-waves that are >2.5 mm high in Lead II as shown in Fig. 2.24. Left atrial enlargement is seen as broad p-wave that are >120 ms in duration in Lead II and a negative deflection in the terminal portion of the p-wave in V1 that is >0.04 s wide and 1 mm deep (or 1 small box wide by 1 small box deep) as shown in Fig. 2.25.

To evaluate the PR segment, a baseline of the ECG must be established. By convention, this is the segment between the end

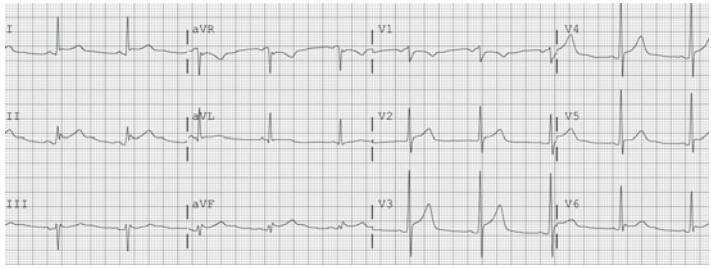


FIGURE 2.26 Pericarditis: PR depression and diffuse ST segment elevation

of the t-wave and the beginning of the p-wave. The PR segment is assessed by comparing the height of this portion to the height of the T-P segment. PR segment depression below this baseline occurs in conditions such as pericarditis as shown in Fig. 2.26.

The QRS complex is then evaluated for changes. One of the most common changes is left ventricular hypertrophy (LVH), which often occurs following years of hypertension. There are numerous ECG criteria for LVH but the most common ones to remember include the Cornell and Sokolow Lyon Criteria (Fig. 2.27). Other conditions can affect the QRS morphology including myocardial infarctions, accessory pathways in Wolff–Parkinson–White (WPW) syndrome and Brugada syndrome. WPW and Brugada syndrome will be discussed in the next chapter, Arrhythmias. As for myocardial infarctions, when a transmural (complete wall thickness) infarction occurs, q-waves can develop. Small q-waves are commonly seen, but significant and pathologic q-waves consistent with prior transmural myocardial infarctions are typically at least 0.04 s wide and 1 mm deep (i.e. a small box wide and a small box deep), or at least 1/3 the height of the QRS complex. In addition, the q-waves must be seen in at least two contiguous leads to be deemed significant. An inferior myocardial infarction manifests as q-waves in the inferior leads (Leads II, III, and AVF) as shown in Fig. 2.28. An anterior myocardial infarction will have q-waves in the anterior leads, V1-V6 (Fig. 2.29). Lateral myocardial infarctions will

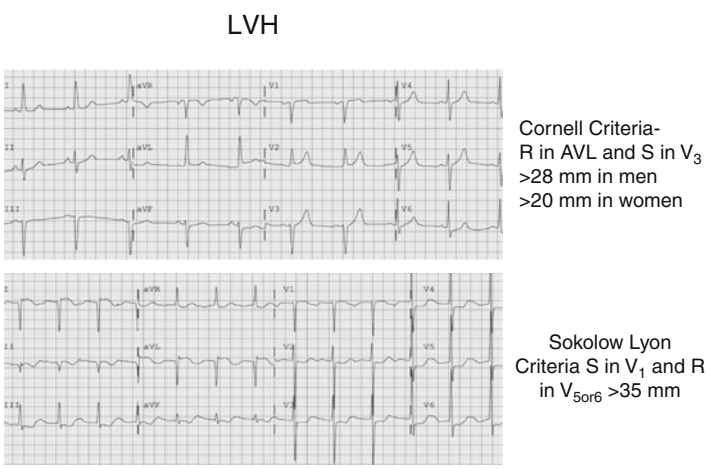


Figure 2.27 Left ventricular hypertrophy

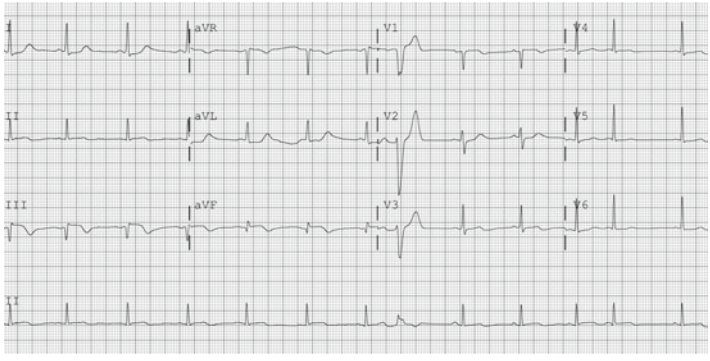


Figure 2.28 Inferior myocardial infarction

have q-waves in leads I and AVL (Fig. 2.30). A posterior infarct is more difficult to diagnose but can present with large R waves in Leads V1 and V2. Often, posterior wall infarcts are associated with inferior wall infarcts. Thus, q-waves in the inferior leads help diagnose a posterior wall infarct as opposed to other causes of large R waves in V1 and V2 (Fig. 2.31).

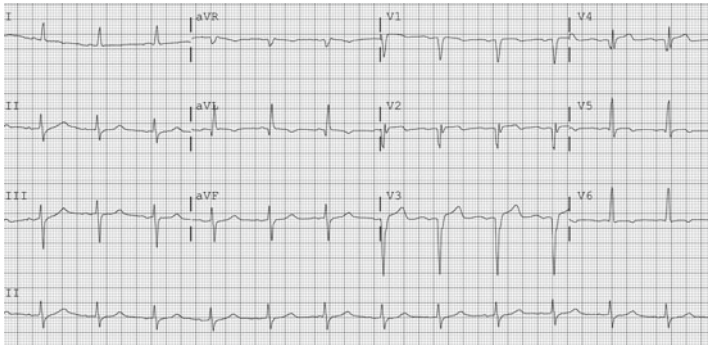


FIGURE 2.29 Anterior myocardial infarction

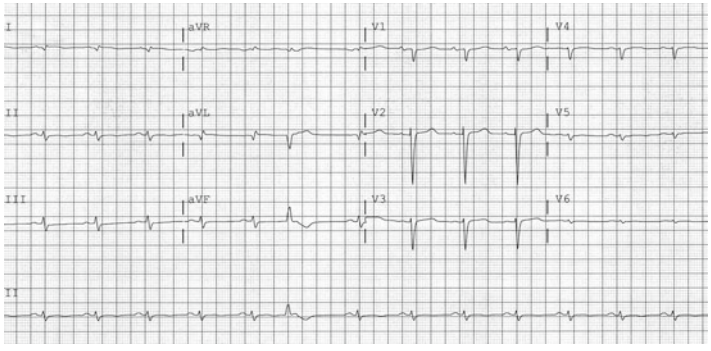


FIGURE 2.30 Lateral myocardial infarction

The next waveform to evaluate is the ST-segment. This segment is important because it shows repolarization abnormalities that can result from ischemia (inadequate blood flow to the cardiac tissue to meet the metabolic demands of the cells) or injury (inadequate blood flow to the cardiac tissue now causing cellular death), pericarditis, or electrolyte derangements. Myocardial injury is strongly suspected when there is ST-elevation in specific patterns, such as the inferior, anterior, lateral or posterior leads (Fig. 2.32). "Reciprocal changes" can be seen in leads opposite of the ST segment elevation. For example, "reciprocal changes" associated with

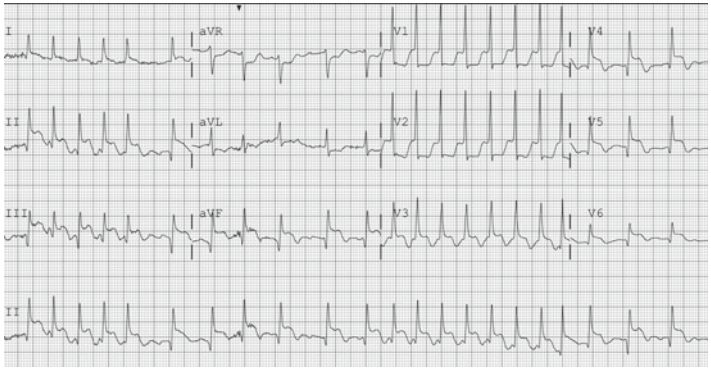


Figure 2.31 Posterior myocardial infarction

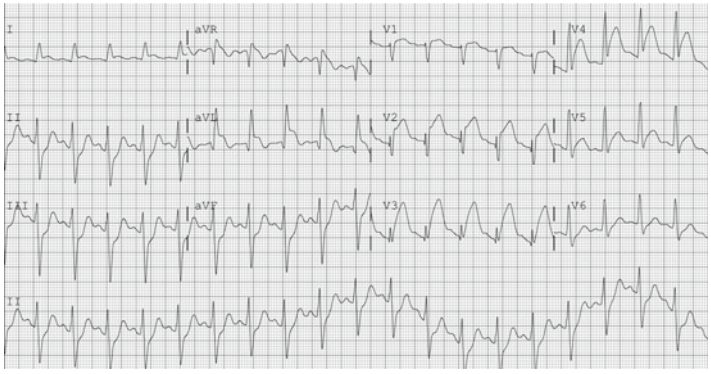


Figure 2.32 Acute anterior ST segment myocardial infarction – ST elevation in precordial leads and ST depression in the inferior leads (also known as reciprocal changes)

an anterior ST-segment elevation, can be seen as ST-depressions in the inferior leads, which is also shown in Fig. 2.32. When ST-depressions without ST-elevations in those same specific patterns occur, myocardial ischemia (and not necessarily injury yet) can be occurring (Fig. 2.33). Diffuse ST elevation is suggestive of pericarditis (Fig. 2.26) or electrolyte abnormalities in some cases.

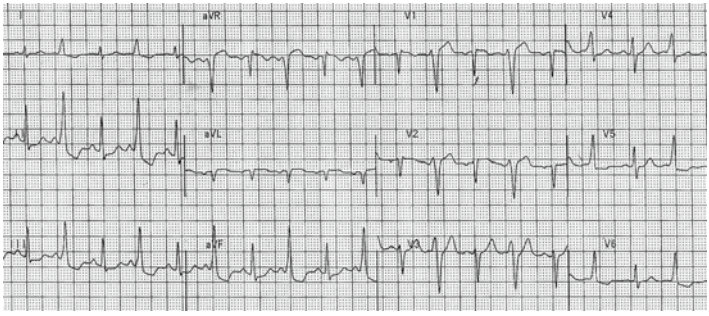


FIGURE 2.33 Inferior ST depressions

Lastly, the t-waves and u-waves can be evaluated. These tend to be less specific, but if focal t-wave inversions are seen in specific lead patterns, myocardial ischemia should be considered. Prominent u-waves can be seen in electrolyte disturbances.

Long-Term Monitoring

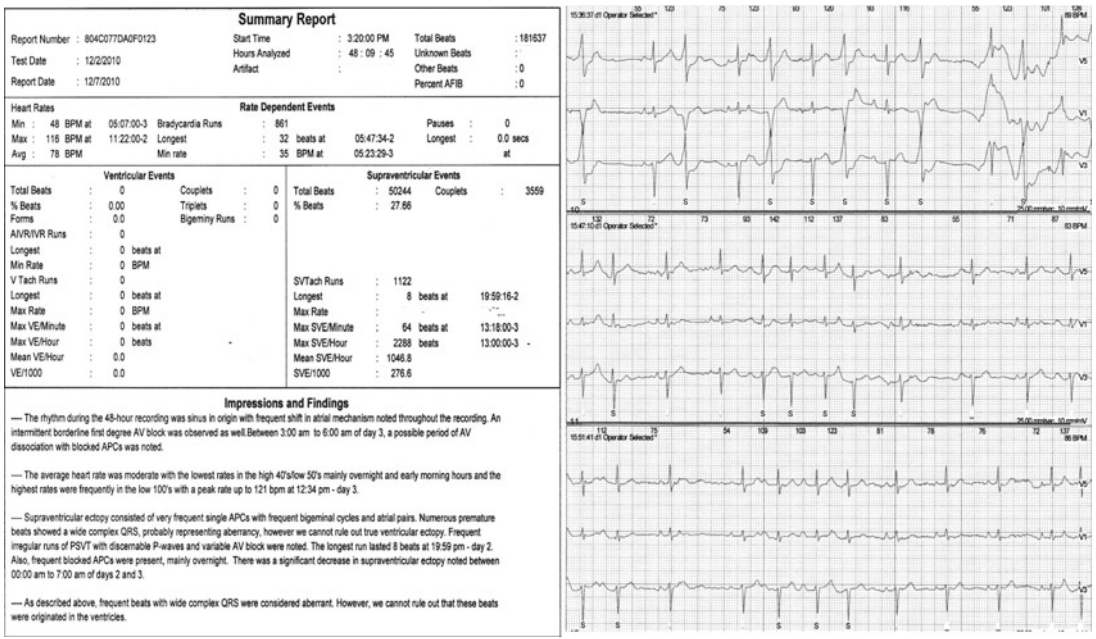
12-lead ECGs provide a momentary snapshot of the heart's electrical activity, but are not the only type of ECGs that can be obtained. Patients who have intermittent episodes of palpitations, syncope or near-syncope require longer periods of monitoring. In these scenarios, the patient is usually feeling fine during the office visit and the potential arrhythmia is not evident in the office 12-lead ECG. To address this, ambulatory monitoring ECGs have been developed. These ECGs can be worn for periods of up to 30 days, increasing the likelihood that an abnormal heart rhythm is captured. These include Holter monitors, event monitors and implantable loop recorders.

Holter monitors, which are named after the person that invented them, are designed to provide 24–48 hours of continuous ECG recording. Typically up to five wires are attached to the patient, similar to in-hospital telemetry monitoring, and these

wires insert into a recording device that is clipped to the belt or placed in a pocket. The monitor continuously records the electrical activity of the heart for the specified 24- or 48-h period. Patients are not permitted to shower during this time. At the end of the 24 or 48 h, the Holter monitor is returned where the data is downloaded. Computer software initially analyzes the data, calculating average heart beats, the fastest and slowest heart rates, and identifies potential arrhythmias that may have occurred. Technicians then review this data and confirm or deny these arrhythmias. A report is generated and sent to the physician's office. An example of a typical tracing is shown in Fig. 2.34.

If the Holter monitor does not capture the arrhythmia or the arrhythmia is occurring infrequently but at least once monthly, an event monitor can be ordered. There are two types of event monitors: continuous looping (i.e. pre-symptom) and non-looping (post-symptom) event monitors. A continuous looping event monitor is an ECG that is worn for an entire month. Typically, two wires are attached to the patient's chest and inserted into a recording device that is attached to the belt or inserted into a pocket. These monitors continuously record the electrical activity of the heart. However, storing every heart beat for an entire month would require a significant amount of memory. Thus, the event monitor is also continuously recording new data over the old data. At the onset of symptoms, the patient presses the record button and the device stores the electrical activity present immediately prior to pressing the record button (generally up to 60 s before) and records the next 30–60 s. These events are stored in the recording device and can be transmitted over standard telephone wires or wirelessly via a cell phone. Event monitors can also be programmed to auto-detect abnormal heart rhythms. Generally, these are programmed to automatically record either very fast or very slow heart rates and do not require the patient to press the record button. This feature is particularly useful when patients are unable to press the button in the event they lose consciousness.

Non-looping monitors are intended for patients that have symptoms lasting longer than 30–45 seconds and remain conscious. The greatest advantage of non-looping monitors is



Summary Report

Report Number	: 804C077DA0F0123	Start Time	: 3:20:00 PM	Total Beats	: 181637
Test Date	: 12/2/2010	Hours Analyzed	: 48 : 09 : 45	Unknown Beats	:
Report Date	: 12/7/2010	Artifact	:	Other Beats	: 0
				Percent AFIB	: 0

Heart Rates

Min	: 48 BPM at	05:07:00-3	Bradycardia Runs : 861
Max	: 116 BPM at	11:22:00-2	Longest : 32 beats at 05:47:34-2
Avg	: 78 BPM		Min rate : 35 BPM at 05:23:29-3

Rate Dependent Events

Pauses	: 0		
Longest	: 0.0 secs		
at			

Ventricular Events

Total Beats	: 0	Couplets	: 0		
% Beats	: 0.00	Triplets	: 0		
Forms	: 0.0	Bigeminy Runs	: 0		
AIVR/IVR Runs	: 0				
Longest	: 0 beats at				
Min Rate	: 0 BPM				
V Tach Runs	: 0				
Longest	: 0 beats at				
Max Rate	: 0 BPM				
Max VE/Minute	: 0 beats at				
Max VE/Hour	: 0 beats	-			
Mean VE/Hour	: 0.0				
VE/1000	: 0.0				

Supraventricular Events

Total Beats	: 50244	Couplets	: 3559		
% Beats	: 27.66				
SVTach Runs	: 1122				
Longest	: 8 beats at			19:59:16-2	
Max Rate	: -				
Max SVE/Minute	: 64 beats at			13:18:00-3	
Max SVE/Hour	: 2288 beats			13:00:00-3	-
Mean SVE/Hour	: 1046.8				
SVE/1000	: 276.6				

Impressions and Findings

--- The rhythm during the 48-hour recording was sinus in origin with frequent shift in atrial mechanism noted throughout the recording. An intermittent borderline first degree AV block was observed as well.Between 3:00 am to 6:00 am of day 3, a possible period of AV dissociation with blocked APCs was noted.

--- The average heart rate was moderate with the lowest rates in the high 40's/low 50's mainly overnight and early morning hours and the highest rates were frequently in the low 100's with a peak rate up to 121 bpm at 12:34 pm - day 3.

--- Supraventricular ectopy consisted of very frequent single APCs with frequent bigeminal cycles and atrial pairs. Numerous premature beats showed a wide complex QRS, probably representing aberrancy, however we cannot rule out true ventricular ectopy. Frequent irregular runs of PSVT with discernable P-waves and variable AV block were noted. The longest run lasted 8 beats at 19:59 pm - day 2. Also, frequent blocked APCs were present, mainly overnight. There was a significant decrease in supraventricular ectopy noted between 00:00 am to 7:00 am of days 2 and 3.

--- As described above, frequent beats with wide complex QRS were considered aberrant. However, we cannot rule out that these beats were originated in the ventricles.

FIGURE 2.34 Holter monitor report

that they do not require electrodes to be attached to the patient. Rather, the patient holds the device against the chest wall and presses the record button at the onset of symptoms. The device then records 30 s of realtime ECG. The disadvantage of this device is that it cannot go back in time and capture the onset of the abnormal heart rhythm, which can often be useful in determining the type of arrhythmia present. These events are stored and can be transmitted to the event monitoring center for analysis.

Should the potential arrhythmia not occur while wearing the event monitor for a month, an implantable loop recorder can be placed. A loop recorder is the size of a "thumb" drive that is implanted under skin of the chest overlying the heart with a minor, outpatient surgical procedure. The loop recorder then continuously monitors the electrical activity of the heart. These devices are programmed to auto-detect heart rhythms that are abnormally fast or slow and can be triggered by the patient to record when symptoms occur. The patient activates the monitor to record by placing a device the size of a garage door opener over the loop monitor on the chest and pressing the record button. The loop monitor stores an ECG of the heart's electrical activity that was present several seconds before the record button is pressed and several seconds after. These devices are periodically checked, either remotely by transmitting the data over a telephone line, or by checking the device in a doctor's office. The battery on current loop monitors lasts up to 3 years. Thus, these devices are intended to provide long-term monitoring to capture abnormal heart rhythms that occur less frequently than monthly and will record abnormal heart rhythms even when a patient loses consciousness.

Other Forms of an ECG: Signal-averaged ECG

A signal-averaged ECG is a special type of ECG which averages multiple electrical signals from the heart to look for very small changes in the QRS complex. Patients who have

signal-averaged ECGs must lie very still for up to 15 min because skeletal muscle contractions and movement can create noise. During this time, a continuous 12-lead ECG is recorded. Computer software then analyzes the rhythm strips and creates an average ECG appearance. By examining many QRS complexes, artifact from muscle movement can be filtered out and fine details on the ECG that persist throughout the entire recording become more apparent. At times, very low amplitude, high-frequency signals can be seen in the terminal portion of the QRS complexes, which are known as "late potentials." These "late potentials" reflect electrical abnormalities within the ventricles and may reflect an increased risk of developing life-threatening arrhythmias. Therefore, this type of ECG is performed in patients who are undergoing further risk assessment for sudden cardiac death. An example of a signal average ECG is shown in Fig. 2.35.

Electrophysiology Study

An electrophysiology study (EPS) is a procedure done by specialized cardiologists known as electrophysiologists, to help identify and potentially treat rhythm disturbances of the heart. The test is usually ordered by an electrophysiologist when work-up for an arrhythmia has not been clearly identified by ambulatory ECGs or when there is suspicion of an arrhythmia that can be treated by an ablation. An EPS typically involves bringing the patient into the cardiac catheterization lab where the patient is connected to external ECG machines. Then, usually two to four catheters are placed into the heart through sheaths placed in the femoral vein. These catheters are about the diameter of a coffee straw but much longer and more flexible. At the end of the catheters, there are smooth, round metal electrodes which can sense the electrical activity of the heart. Usually, various measurements are made to assess the intrinsic conduction properties of the heart. If an arrhythmia is not present initially, the heart is stimulated by pacing the heart (using the same catheters) at various rates and introducing premature beats in an attempt to induce an arrhythmia. If this

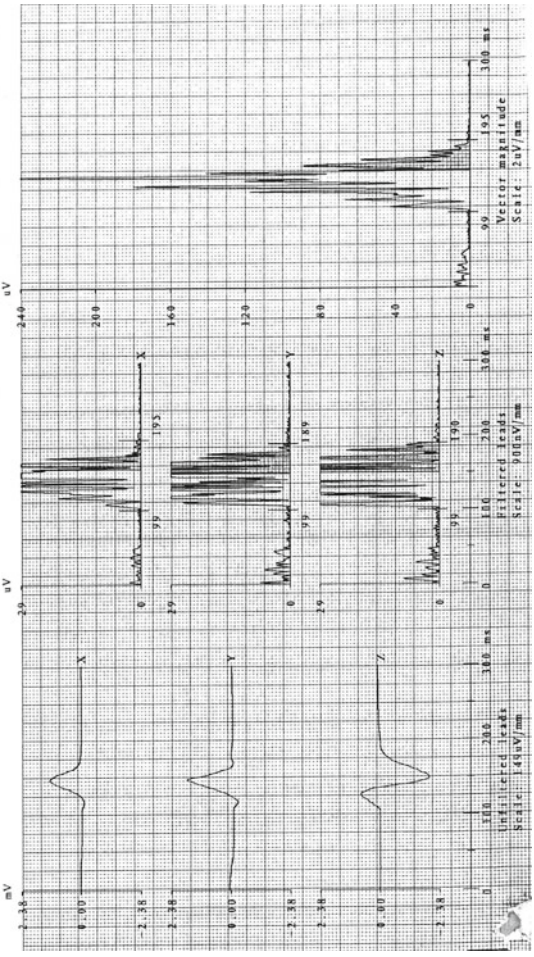


FIGURE 2.35 Signal average ECG

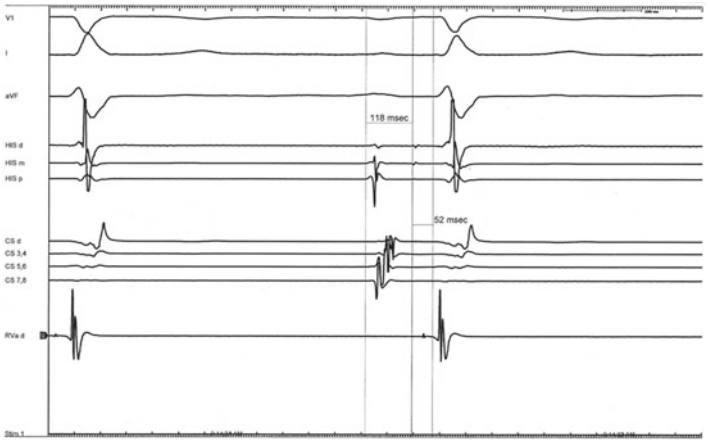


FIGURE 2.36 EPS example of sinus rhythm

does not bring out the arrhythmia, drugs that make the heart more excitable, such as isoproteronol, are administered. These procedures are usually performed with conscious sedation. Rarely, general anesthesia is implemented. Both slow and fast heart rhythm disorders can be detected by an EP study and can guide management of the arrhythmia, including the need for a pacemaker or an implantable cardioverter defibrillator (ICD). Often, an EP study identifies an abnormal heart rhythm that can be cured by ablation. An example of normal sinus rhythm seen on an EPS is shown in Fig. 2.36.

An ablation procedure is carried out by placing an ablation catheter at the site of origin of an arrhythmia. The goal of ablation is focal destruction of the offending heart tissue that is causing the arrhythmia. Ablation can occur by a variety of methods but primarily involves either heating or freezing the tissue so the heart cells will no longer conduct electrical signals. To heat the tissue, the catheter tip is designed to deliver radio frequency (RF) energy. The heart tissue absorbs this RF energy causing it to heat up and be irreversibly damaged. The damaged tissue is eventually replaced by scar, which is electrically inactive. Alternatively, cryotherapy can be applied through a specialized

catheter which freezes the heart tissue around the area of the catheter tip, thereby destroying the tissue. A scar forms at this site of ablation as well. Most commonly, RF energy is used because the ablation can penetrate more deeply and is associated with better long-term success rates. Alternatively, cryotherapy is often used in higher risk situations such as AV re-entrant tachycardia where the pathologic pathway is very close to the normal electrical system. This technique allows the creation of precise lesions safely without damaging the AV node.

Conclusion

After the physical exam, an ECG is the most common method to evaluate the heart. To accurately "read" ECGs, use the same method each time. If further evaluation or treatment of a rhythm problem is deemed necessary, longer-term ECGs can be ordered or an electrophysiology study can be performed. Refer to the texts listed below for further information regarding the interpretation of ECGs.

Further Reading

For the beginner – Dubin D. Rapid Interpretation of EKG's. 6th ed. Tampa: COVER Publishing Company; 2000.

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