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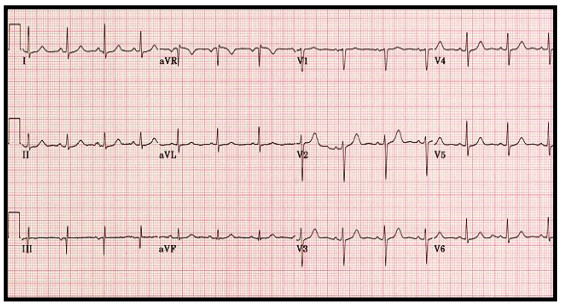
CARDIOCOMP™

Electrocardiography Primer



INTELITOOL®
Intelligent Tools for Physiology™
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CARDIOCOMP 1
Version 8
Electrocardiography Primer

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CHAPTER 1 INTRODUCTION TO ELECTROCARDIOGRAPHY

An electrocardiogram, abbreviated ECG (or EKG - German), is a graphical representation of the electrical activity of the heart as measured at the surface of the body. The electrocardiogram allows us to observe the sequential events of excitation and recovery of the pumping chambers of the heart. The electrocardiogram therefore allows us to identify normal and abnormal excitation and rhythms. By interpreting the ECG the location of damaged tissue, poor cardiac circulation, atrial or ventricular hypertrophy (enlargement), the presence of systemic diseases that affect the heart, the effects of drugs, and many other cardiac problems can be found. The interpreter of the ECG needs not only a good accurate recording device, but also a good understanding of cardiac electrophysiology. Armed with an understanding of cardiac function, each bump on the trace becomes a clue which combined with an understanding of the way the signal was gathered, tells the interpreter something about the actual cardiac tissues. For all of its diagnostic strength, the ECG is only one tool which must be combined with others in real clinical applications. In other words, sometimes "normal" patients will have an abnormal ECG, while some "abnormal" patients will display a normal ECG.

CHAPTER 2 THE PHYSIOLOGY BEHIND ELECTROCARDIOGRAPHY

The ECG read at the surface of the body, while due to the change in the transmembrane potential in individual myocardial cells acting together, is nothing like that change in potential at all. To explain the ECG signal, we must start with individual myocardial fibers, and progress to an understanding of the way the cardiac contraction signal spreads throughout the myocardium (cardiac muscle tissue).

2.1 The Myocardial Cell Membrane Potential

When myocardial cells are in a resting situation, we say the transmembrane potential is a resting potential. This potential is positive on the outside and negative on the inside of the membrane and is maintained by the active (requires metabolic processes) pumping of sodium ions to the outside of the membrane, while pumping an unequal amount of potassium ions to the inside of the membrane. This sodium-potassium pump establishes a diffusion gradient for sodium with a higher concentration on the outside and lower on the inside of the membrane, and a diffusion gradient in the opposite direction for potassium ions. While the degree of potential difference varies depending on where the fiber being measured is located in the heart, the resting potential is usually about -90 mv on the inside of the membrane, relative to the outside of the membrane. When the cell experiences an action potential, the polarity of the membrane is weakly reversed, and the myocardial cell contracts. This occurs either spontaneously or in response to an outside stimulation, and is due to a transitory state in the membrane's ion permeability properties.

First, the membrane becomes very permeable to sodium ions and extracellular sodium rushes in, reversing the charge. Next the membrane's state of less permeability to sodium ions rapidly returns. (This beginning of the myocardial action potential is very similar to the beginning of an action potential of a skeletal muscle fiber. The next stage of the action potential is very different from a skeletal muscle fiber.) For the next 200 milliseconds a slow inward diffusion of calcium ions does not allow the re-establishment of the resting potential, causing a "plateau phase" in the action potential which in turn causes a sustained contraction period. Finally the cell membrane becomes momentarily permeable to potassium ions, which diffuse outward, and the resting potential is reestablished. The following figure shows the voltage fluctuations over time of the inside of a myocardial cell membrane during an action potential.

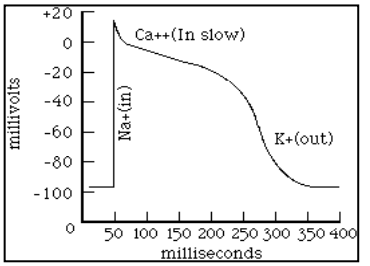

Figure 2.1 Movement of Ions During the Myocardial Action Potential

Myocardial cells are not stable during the resting potential stage. That is, myocardial cells undergo the above described transmembrane excursions, or depolarizations, spontaneously. Their instability is due to a slow leaking inward of sodium ions which gradually raises the resting potential until a threshold is crossed at which time the cell membrane opens to sodium ions and the ensuing rapid influx of sodium ions initiates an action potential. After the action potential has been returned to a resting potential the myocardial cells are the farthest from depolarizing and are said to be hyperpolarized. The least stable of all myocardial cells (the leakiest to sodium ions) are the myocardial cells in sinoatrial node (S-A node), a patch of tissue on the posterior wall of the right atrium near the entrance of the superior vena cava. Because the cells in the S-A node are the least stable, they are the cells which first cross a threshold causing them to depolarize, starting a depolarization sweep (the cardiac impulse) that depolarizes all of the myocardial cells. When they all repolarize, they are again the farthest from experiencing an action potential spontaneously. Since the cells of the S-A node are the least stable, they are again the first to cross a threshold which precipitates depolarization. This sets the stage for the cells of the S-A node to start a depolarization wave again, and again, and again.... For this reason the S-A node is often referred to as the pacemaker of the heart.

2.2 Propagation of the Cardiac Impulse

The ECG is not the reading of a transmembrane potential, but it is rather a gross potential between patches of myocardium which are depolarized, and patches of myocardium which are polarized, as read at the surface of the skin. It takes time for the cardiac impulse to travel through the myocardium. Because of this time lag, some of the myocardium will be depolarized while some are not (before the impulse gets there), creating an electrical potential. After the impulse has passed, some of the myocardium will repolarize before the rest of the myocardium, creating another electrical potential.

The myocardial fibers of the atria and ventricles are considered to be two separate interconnecting branched networks, separated from each other by fibrous connective tissue. They are connected to each other functionally by only the conduction system. When any part of these networks are stimulated, the stimulus is propagated throughout the entire muscle mass. Because the cells are so intimately associated functionally, they are called the atrial syncytium and ventricular syncytium.

The conduction system begins with the sinoatrial node. When this patch of tissue depolarizes, the entire atrial syncytium depolarizes. Special myocardial cells which form the internodal pathways spread the impulse faster than the normal myocardial cells of the atria, bringing the impulse quickly to the atrioventricular node (A-V node). The A-V node, located in the floor of the right atrium, provides the only conduction pathway to the ventricular syncytium. The internodal pathways are connected to the A-V node by very short, small diameter, junctional fibers which delay the impulse so that the ventricles will have time to fill with blood as the atria contract. The cardiac impulse passes from the A-V node into a bundle of fibers called the A-V bundle (bundle of HIS), in which the signal spreads very rapidly. The A-V bundle then enters the upper ventricular septum where it divides into left and right branches that lie just under the endocardium (the tissue which lines the inside of the cardiac chambers). Halfway down the septum the branches become enlarged Purkinje fibers which are the fastest cells of the conduction system. The Purkinje fibers continue down into the apex of the ventricles and then back upward over the lateral walls of the ventricles, causing a very rapid depolarizing of the ventricular myocardium.

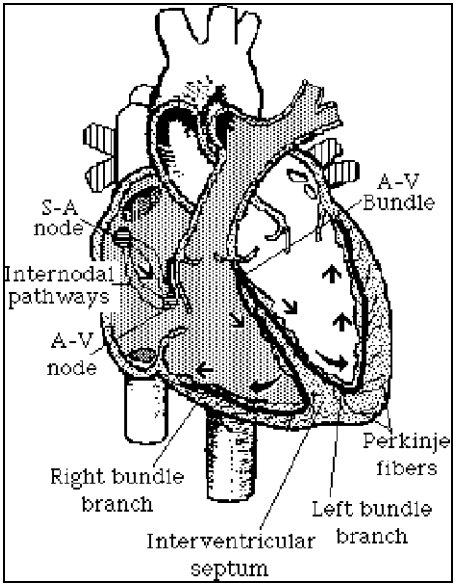

Figure 2.2 The Cardiac Conduction System

The ECG shows a distinct pattern of deflections which are a consequence of the above described movement of the cardiac signal around the heart. This movement causes potential differences between depolarized and polarized tissues. The ECG pattern that you are probably the most familiar with is the pattern seen on a LEAD II ECG.

The line between cardiac events, when all of the myocardium is in the resting condition (polarized), is called the isoelectric line.

The first deflection is the relatively slow and weak depolarization of the atria, called a P wave, which occurs just before the atria contract. As the atrial myocardium depolarizes, the potential between the depolarized and polarized atrial fibers increases to a rounded peak. Then as all of the cells of the atria become depolarized, the potential falls back to zero (the isoelectric line). The next series of deflections are called the QRS complex. These rapid and strong deflections occur just before the strong muscles of the ventricles contract. As the impulse spreads through the ventricles, the potential rapidly rises to a sharp peak, again falling to zero as all of the ventricular myocardial cells depolarize. The next wave seen on the ECG is the large, slow T wave. This wave is caused by the repolarization of the ventricles. The T wave rises to a rounded peak as the ventricular cells repolarize, falling back to zero when all of the cells are repolarized. The atrial repolarization would also cause a similar, much smaller, wave, but atrial repolarization coincides with ventricular depolarization and is totally obscured by the QRS complex.

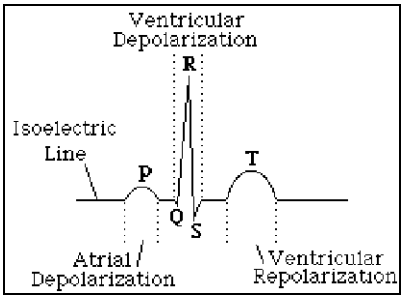

Figure 2.3 Example Of A Normal LEAD II QRS Complex

The impulse propagation travels at normal speeds in myocardial tissues that are in good condition and have a normal blood supply. The expected time intervals for the ECG events are shown below.

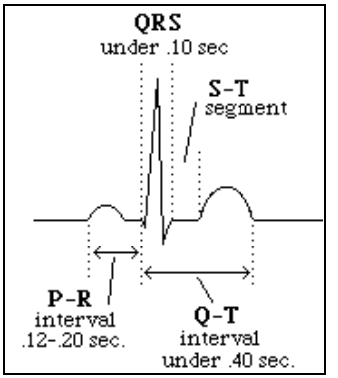

Figure 2.4 Normal LEAD II QRS Complex and Intervals. The “normal” Q-T interval varies greatly depending on ventricular rate, gender and age.

Now try to integrate your knowledge of the impulse propagation with your powers of logic and try to determine what the problem might be at the tissue level in the next three LEAD II ECGs.

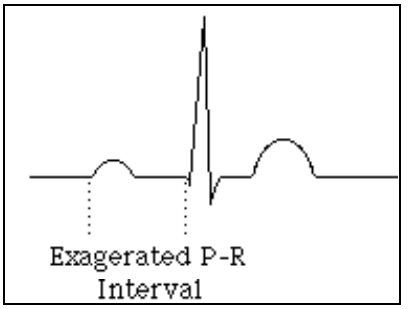

Figure 2.5 A LEAD II QRS Complex With An Exaggerated P-R Interval

The ECG shown in the figure above has a P-R interval that is longer than usual.

Question to ask: What does the P-R interval represent?

Answer: The P-Q interval (the major portion of the P-R interval) represents the time it takes for the impulse to travel from the atria to the ventricles, in particular most of this interval is the time used as the impulse is slowed at the A-V node.

Possible meaning: An exaggerated P-R interval might indicate that something is wrong with the A-V node or the A-V bundle.

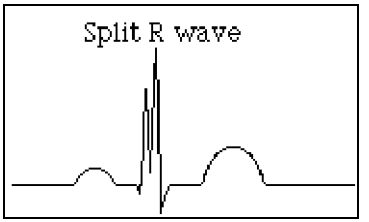

Figure 2.6 A LEAD II QRS Complex With A Split R-Wave

In the ECG above we have a split R-wave.

Question to ask: What does the R-wave represent?

Answer: The simultaneous depolarization of the right and left ventricles.

Possible meaning: One of the bundle branches is damaged or undernourished perhaps due to poor blood supply. Because of this, one ventricle is depolarizing later than the other.

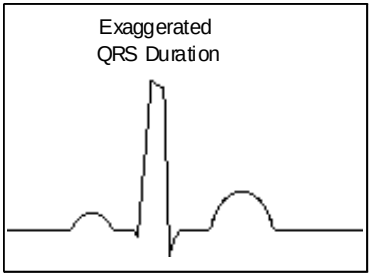

Figure 2.7 Example Of A LEAD II QRS Complex
With An Exaggerated QRS Duration

The above ECG shows an exaggerated QRS duration:

Question to ask: What does the QRS wave represent?

Answer: The normally rapid depolarization of the ventricles.

Possible meaning: The Purkinje fibers might be damaged, or poorly supplied with blood. Therefore the impulse is taking a long time to get to all of the ventricular myocardium.

CHAPTER 3 THE FRONTAL PLANE ECG LEADS

An ECG LEAD is a potential difference between two or more points on the surface of the body. An ECG LEAD is usually viewed as the graphing of the electrical potential between those two or more points over time. In other words an ECG is a graph with two axes. The magnitude of the electrical potential, expressed in millivolts, is displayed on the vertical axis, and the time over which the electrical events take place is displayed on the horizontal axis. The graph will be centered about zero so that if the potential is greater toward the positive electrode, the plot will deflect upward, and if the potential is greater toward the negative electrode, the plot will deflect downward. Which electrode is positive, and which electrode is negative is not important for any other reason than it is easier to compare ECGs if they are always monitored the same way. On all of the LEAD diagrams the right leg has a ground electrode on it. This electrode does not have a function other than to help the machine (Cardiocomp™) eliminate noise.

3.1 The Six Frontal LEADs

Standard LEADs (LEADs I, II, III)

The standard LEADs I, II and III are called bipolar LEADs because they are the potential between two electrodes, one which is defined as the positive electrode, the other as the negative electrode. The electrodes for the standard LEADs I, II, and III are usually placed on the right arm, left arm and the left leg, hence they are also called the appendicular electrodes. Electrically the signal is virtually the same as if the electrodes were placed at the shoulders and the groin (except weaker). Therefore these LEADs look at the potential around the heart in a three sided fashion which describes a triangle. This view of the heart was proposed by Einthoven, who produced the first clinical Electrocardiograph at about the turn of the century; hence this triangle is called the Einthoven triangle.

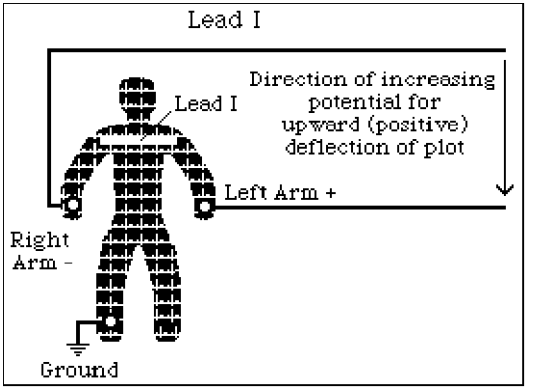

Figure 3.1 LEAD I Vector

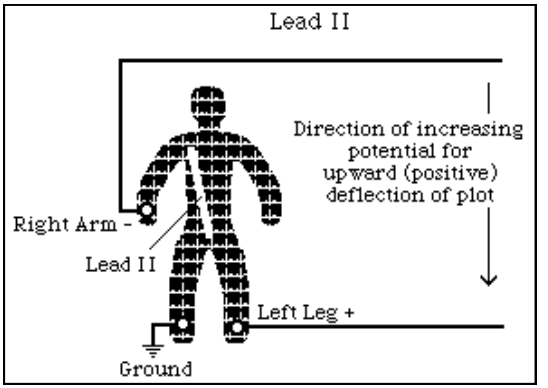

Figure 3.2 LEAD II Vector

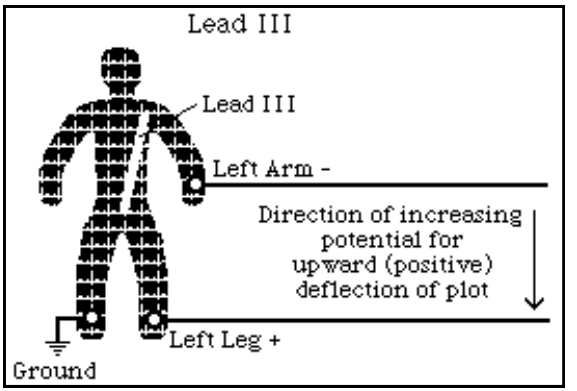

Figure 3.3 LEAD III Vector

When we put LEADs I, II, and III together on one diagram, we see that the three LEADs form a triangle around the heart, the Einthoven triangle.

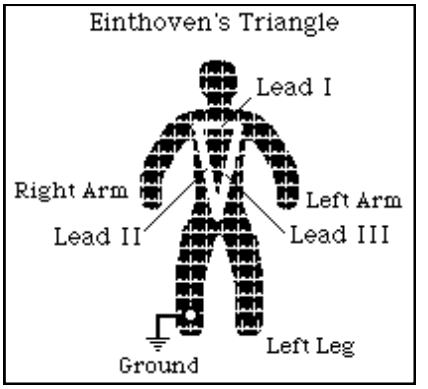

Figure 3.4 Einthoven's Triangle

A synopsis of all of the preceding figures is pictured below:

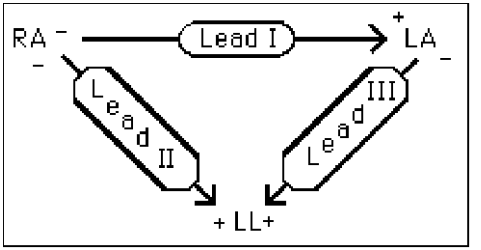

Figure 3.5 Einthoven's Triangle

In this figure the arrows represent the potential gradient direction for a positive deflection on the screen.

Augmented Unipolar LEADs (aVR, aVL, aVF)

An augmented unipolar LEAD is called unipolar because it is the potential between one appendicular electrode, which is defined as positive, and the average of the other two appendicular electrodes, which are negative. In other words one electrode is selected to be compared to the other two. The reason these LEADs are called augmented is because many years ago they were read in a very clumsy way so that the signal strength on these LEADs was thought to be weak. Because they were thought to be weak, these LEADs were "augmented" or exaggerated so that the would look similar to the other frontal LEADs in strength. Long ago it was realized that this was a mistake (the signal is just as strong on these LEADs), and we now read them correctly but the name has stuck. The augmented unipolar LEADs are not "augmented". The usefulness of these LEADs is to create three more LEADs perpendicular to the three standard LEADs from the same appendicular electrodes. These LEADs could be imagined as moving the electrodes to the center of each of the segments of the Einthoven triangle so that we now are reading "new" potentials, as illustrated in the following figures.

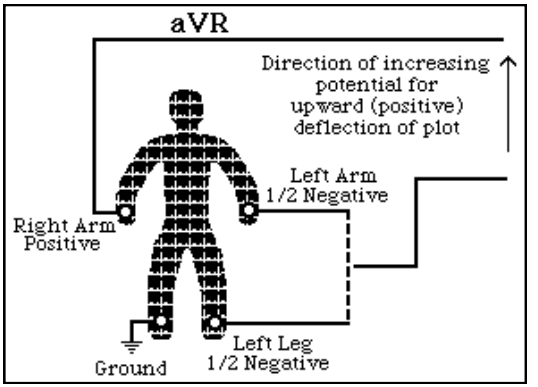

Figure 3.6 LEAD aVR Vector

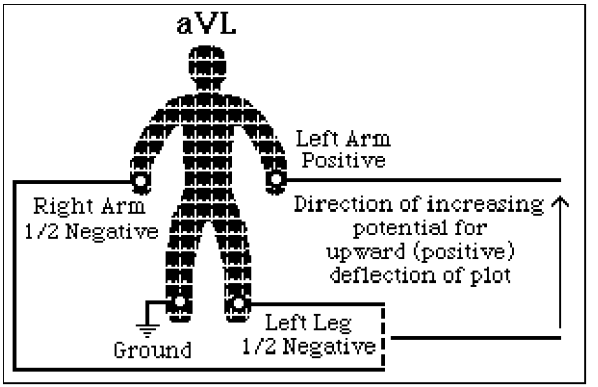

Figure 3.7 LEAD aVL Vector

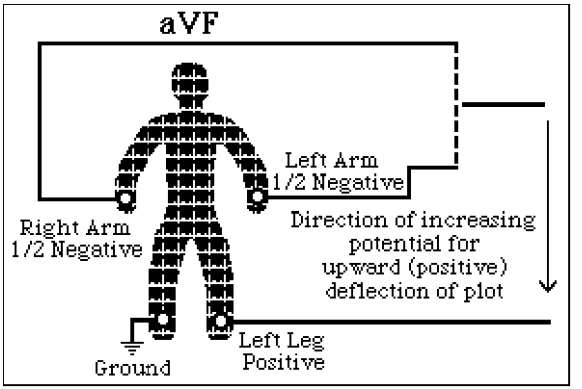

Figure 3.8 LEAD aVF Vector

In the three previous figures, LEADs, aVR, aVL and aVF are shown below as they are interpreted. These diagrams of the augmented unipolar LEADs are superimposed over the standard LEADs I, II, and III (Einthoven triangle).

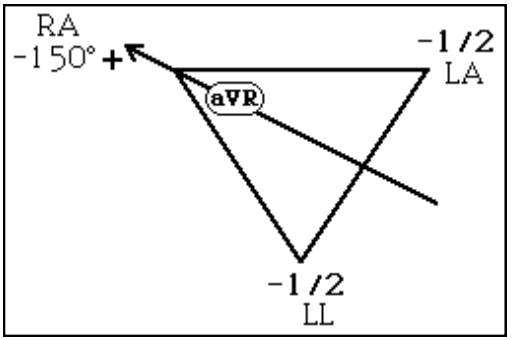

Figure 3.9 LEAD aVR Vector

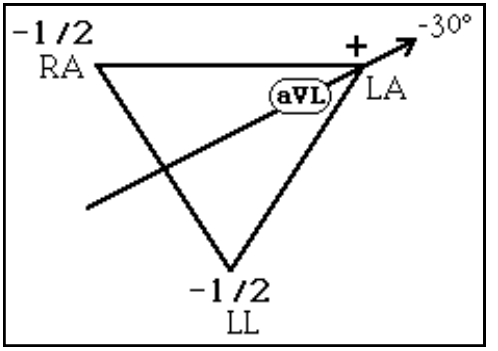

Figure 3.10 LEAD aVL Vector

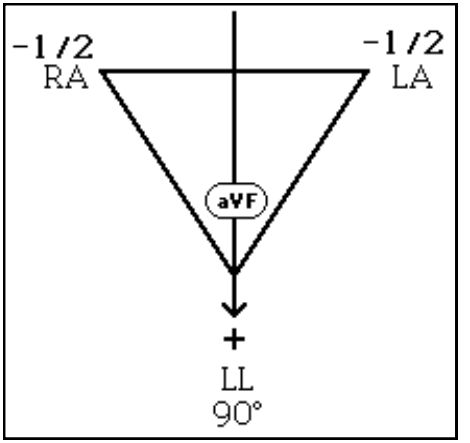

Figure 3.11 LEAD aVF Vector

The below diagram shows a synopsis of all of the six frontal LEADs.

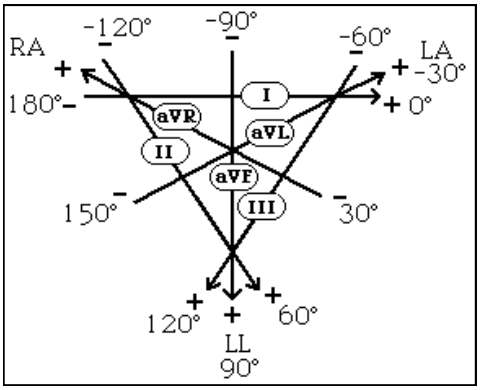

Figure 3.12 Frontal LEAD Vectors

A much clearer representation of the above is found in the figure below. Notice that both diagrams are really the same thing. From this diagram we can finally clearly see that each LEAD is just the potential difference found between two actual electrode locations which define a direction because of their placement.

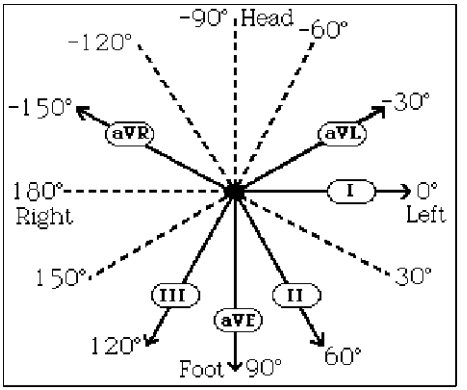

Figure 3.13 Frontal Plane LEAD Vectors

The following table is a review of the electrode setup for all six of the frontal LEADs:

LEAD	Direction	(+) Electrode	(-) Electrode(s)
I	0°	LA	RA
II	60°	LL	RA
III	120°	LL	LA
aVR	-150°	RA	1/2LA + 1/2LL
aVL	-30°	LA	1/2RA + 1/2LL
aVF	90°	LL	1/2RA + 1/2LA

Figure 3.14

A simple explanation as to why these six LEADs are useful, is that each of the LEADs is nothing more than the potential difference between two points in a particular direction. It is important to look at the potential change along different directions because if you did not it would be possible to completely miss some of the information. For example, figure 3.19 illustrates that if the actual potential gradient of the cardiac event under study was perpendicular to the axis of the LEAD, it would not matter what the actual gradient strength was, the potential difference between the electrodes for that LEAD would always be zero. From this it can be seen that two LEADs, each perpendicular to each other would always contain all of the information traditionally viewed in the six frontal LEADs. So, you may ask, why bother with six different LEADs? The reason for this is that the six different LEADs actually are very useful for diagnostic work. An experienced person can scan the six frontal LEADs and rapidly determine the direction of the potential of any electrical event. The direction of the electrical gradient of the R-wave, for instance, can be determined by simply noting in which LEAD the R-wave has the strongest deflection. If it was in LEAD II (it usually is), the doctor knows that the potential is in the direction from the right shoulder to the left foot, or about 60 degrees.

3.2 Interpreting the LEADs as Projections

The following diagrams are all representations of the exact same potential gradient as it would be seen if viewed by the six different frontal LEADs. The direction of the actual potential gradient is 60°. The magnitude as it shows up on any one LEAD is proportional to both the projection of the actual potential gradient onto the angle of the LEAD we are viewing and the distance between the two points of the LEAD. Notice that the closer the angle of the view (LEAD) is to being parallel to the actual potential gradient, the greater the deflection is. LEAD II is exactly parallel, and there we see the maximum deflection. Also notice that in LEAD aVL, which is a view perpendicular to LEAD II, the same potential causes no deflection at all. In LEADs aVR and aVF the viewed magnitude is the same, since both LEADs are 30° from the angle of the actual potential. However the direction of the deflection is opposite, since the LEADs are read in opposite directions, and in LEAD aVR the gradient is in the direction of the negative electrode(s). Finally notice that the magnitude in LEADs I and III is the same, since they are both 60° from

the angle of the actual potential. With these two LEADs the direction of the deflection is also the same, since the direction of the potential and the LEADs are the same.

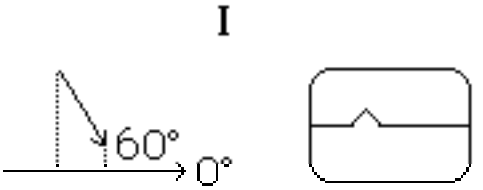

Figure 3.15 Projection of LEAD I

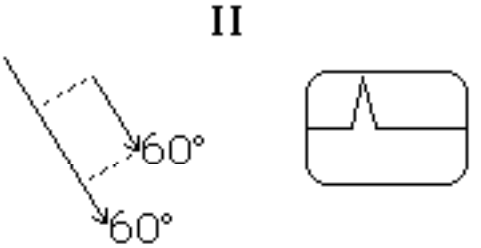

Figure 3.16 Projection of LEAD II

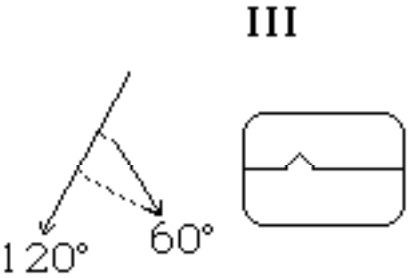

Figure 3.17 Projection of LEAD III

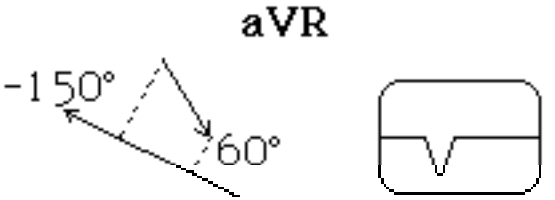

Figure 3.18 Projection of LEAD aVR

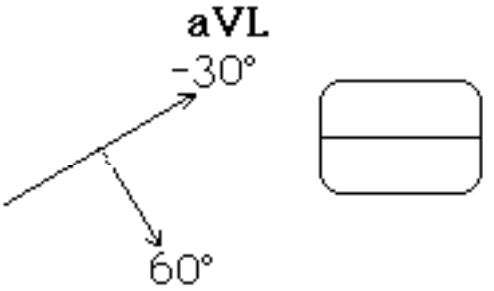

Figure 3.19 Projection of LEAD aVL

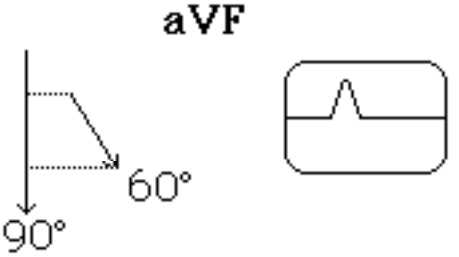

Figure 3.20 Projection of LEAD aVF

The order in which the six frontal LEADs are read, and the direction of the LEADs, have a historical basis rather that a practical one. Because of the time over which they were developed, and also because the interpretation of some of the LEADs was at times in the past very incorrect, the LEADs are what they are today; somewhat jumbled. However, it is very useful to keep the frontal LEADs just as they are because as long as we always view each patient the same way, a system for comparing normal to abnormal can be developed, allowing the frontal LEADs to become a strong diagnostic tool.

CHAPTER 4 THE CHEST LEADS

The chest LEADs were introduced when it was realized that using some electrodes closer to the heart would allow a more accurate study of the heart's electrical activity. There are six standardized chest LEADs. Since the chest LEADs were introduced at a later date than the frontal LEADs, their construction follows a logical order. The names of these LEADs are V1, V2, V3, V4, V5, and V6. While the frontal LEADs help us determine electrical events in one spatial plane, the frontal, the chest LEADs are designed to help us study electrical events in a transverse plane. Another advantage to the chest LEADs is that their very close position to the ventricles provides us with a highly localized look at the ventricular myocardium. The chest LEADs are unipolar LEADs in that each chest electrode is defined as a positive electrode, referenced against the average of the three negative appendicular electrodes. The direction of the electrical difference measured by each of the chest electrodes is toward the selected electrode, with the angle rotating counterclockwise, as viewed from above, for each LEAD starting with V1 at +120° and ending with V6 at 0°. The following figure shows the direction each of the chest LEADs represents.

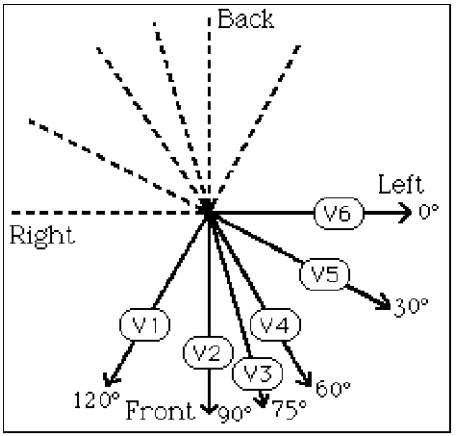

Figure 4.1 Transverse Plane Vectors

Keep in mind the fact that with the chest LEADs we would be looking at potential gradients in the transverse plane, not the frontal plane.

Because the chest LEADs are so close to the heart, the magnitude of the potential on these LEADs tends to be very strong. Because of this, these LEADs are often read with a less sensitive scale than are the frontal plane LEADs.

CHAPTER 5 VECTORCARDIOGRAPHY

5.1 The Vectorgram

A vector is a physical quantity which has both magnitude and direction. The magnitude and direction of the cardiac electrical potential gradient can be described with vectors.

If we take the knowledge gained in sections 3, 4 and 5 one step further, we can generate a picture of the direction and magnitude of the ECG potential gradient throughout the entire cardiac cycle. Such a picture would be called a vectorcardiogram. If the vectorcardiogram was generated with only the frontal LEADs (frontal plane), as is currently the case with Cardiocomp™, the vectorcardiogram would be a two-dimensional vectorcardiogram. If data from the chest LEADs (transverse plane) were added, a three dimensional vectorcardiogram could be developed.

An electrical potential gradient has a direction (angle) and magnitude (field strength/meter). Looking at an ECG of any LEAD, the information displayed tells us the gradient as projected on a fixed angle and distance view. In order to talk about direction we must have a reference frame. The reference frame we use in cardiology is one where the heart is in the center of a cartesian system. The horizontal axis is drawn from right to left, with the heart at the center, the left side is at 0°, and the right side is ±180°. The vertical axis is drawn through the heart with the head at the top at -90° and the foot at the bottom at 90°. Remember that the direction of the view is defined by the electrode placement for that LEAD. We could determine the actual direction and magnitude of the cardiac impulse potential gradient with just the data from two different LEADs which were read simultaneously.

For example, if the voltage on LEAD I, at one point in time, was -0.2 mv and the voltage on LEAD III was 0.8mv for the same point in time, the actual voltage and direction of the cardiac impulse electrical potential for that point in time could be estimated as follows:

First of all, at a glance we can see that the angle must be greater than 90°, and less then -150°. Why? Because that is the only area where the direction of the potential would show up both as negative in LEAD I (which is negative only between -90° and +90°, through ±180°) and positive in LEAD III (which is positive only between +30° and -150°, through +120°). Also because the signal is so much stronger on LEAD III, we know that the direction is quite close to LEAD III, probably a little less than 120° with a magnitude only a little more than 0.8mv.

We could calculate the vector of the actual electrical potential gradient using trigonometry. Calculating manually the magnitude and direction for each point on an ECG would be very laborious to say the least, so, why not let a computer do it?

That is exactly what the computer does to generate the vector-cardiogram seen in Cardiocomp™. The vectorcardiogram is shown as a cartesian plot where the heart lies at the center of the horizontal and vertical axes. The magnitude of the potential is

represented by the distance from the center to the point plotted. The direction is shown by the angle from the center to the point plotted.

As the cardiac impulse is propagated throughout the heart, the direction and magnitude of the electrical potential gradient varies greatly from moment to moment. The potential gradient will form loops as it sweeps out and around for each of the various events of the cardiac cycle. A typical vectorcardiogram might look like the figure below. The isoelectric line seen in an ECG is now an isoelectric point in a vectorcardiogram.

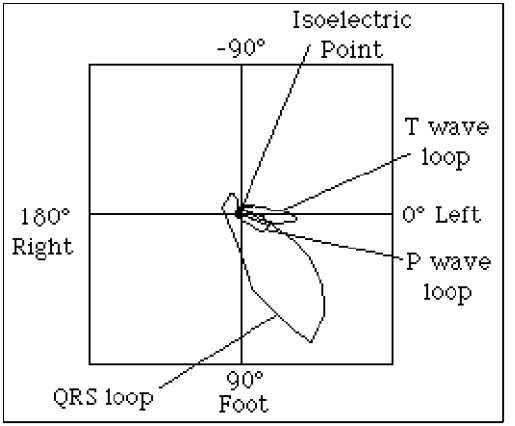

Figure 5.1 Typical Vectorcardiogram of a Single Complete Cardiac Cycle

To better understand just what the vectorcardiogram represents, lets look at two randomly chosen points on the QRS loop.

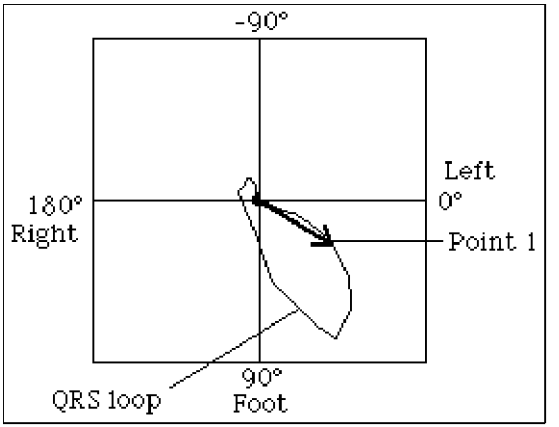

Figure 5.2 Typical Vectorcardiogram of a Single QRS Complex

Point 1 represents an instant in the QRS Loop when the magnitude of the cardiac potential was 0.42mv and the direction was 36°.

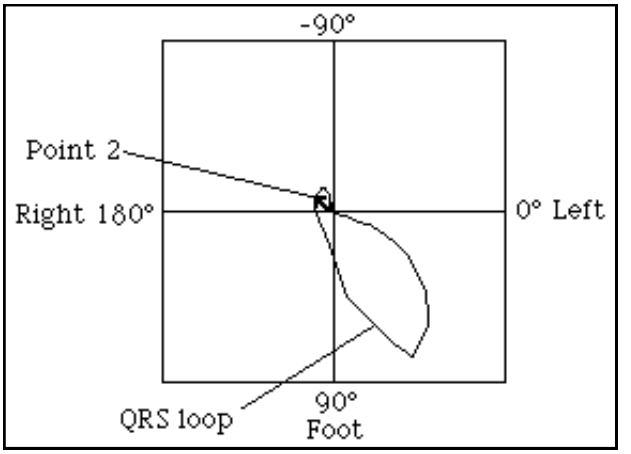

Figure 5.3 Typical Vectorcardiogram of a Single QRS Complex

Point 2 represents an instant in the QRS Loop when the magnitude of the cardiac potential was 0.11mv and the direction was -133°.

5.2 The R-Wave Axis (QRS Axis)

The R-wave axis is a single vector, which represents the potential direction of ventricular depolarization. The R-wave axis tells us about the degree of coincidence between the two ventricles. If there is a conduction problem where the signal is slowed in getting to one ventricle and not slowed in getting to the other, the axis will be rotated. This type of rotation is an electrical rotation of the axis and is often an important diagnostic. If the axis is electrically rotated to the right (clockwise), then the signal is getting to the right ventricle faster than it is getting to the left. If the axis is rotated to the left (counter-clockwise) then it is getting to the left ventricle faster.

The R-wave axis is also affected by the anatomical position of the heart. Shorter people often have a rotation to the left, for example. The position of the person, such as sitting versus laying down, can affect the anatomical position of the heart. Sitting, versus lying, will often rotate the heart to the left. If the axis (laying down) is between 0° and 90° it is said to be normal.

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