Electrocardiography I Laboratory

CleveLabs Laboratory Course System Version 6.0

Introduction

The body relies on the heart to circulate blood throughout the body. The heart is responsible for pumping oxygenated blood from the lungs out to the body through the arteries and also circulating deoxygenated blood back to the lungs from the body through the veins. The heart is divided into four chambers and each chamber is responsible for a different part of the circulatory process mentioned above. Deoxygenated blood first enters the right atrium via the vena cava, where it is then pumped into the right ventricle. The right ventricle pumps this deoxygenated blood through the pulmonary artery to the lungs, where it flows through the alveoli, receives oxygen, and then is returned to the heart through the pulmonary vein and into the left atrium. The left atrium then pumps this oxygenated blood into the left ventricle, where then it is pumped out to the rest of the body through the aorta. This process of contracting the different chambers is highly coordinated and the coordination is controlled by specialized regions of the heart responsible for electrical stimulation of cardiac muscle. Like several other bioelectrical signals, the electrical impulses generated by the heart can be measured on the surface of the skin with electrodes.

Using surface electrodes the cardiac potential of the heart can be measured and correlated with regions of cardiac excitation. This measurement is called an electrocardiogram (ECG). The ECG can be used to evaluate cardiac function, heart rate, and cardiac arrhythmias. The electrical activation that creates the normal heartbeat can in some instances cause abnormal cardiac function. Disorders such as bradycardia (slow heart rate), tachycardia (fast heart rate), and electrical conduction problems such as bundle branch blocks can be all diagnosed from the ECG.

Equipment required:

- CleveLabs Laboratory Kit
- CleveLabs Software
- Four (4) Snap Electrodes and Snap Leads
- Microsoft® Excel, MATLAB® , or LabVIEW™
- Protractor

Background

Cardiac Contraction

As mentioned above, a series of events occur in a specific order during a normal heartbeat. This process is called the cardiac cycle. The cardiac cycle can be broken down into two components, systole and diastole. Diastole occurs when the heart muscle is relaxed and begins to fill with venous blood in the right atrium and oxygenated blood in the left atrium. Systole is the time when the heart contracts. During systole, the heart forces oxygenated blood out of the left ventricle and deoxygenated blood to the lungs through the right ventricle.

Deoxygenated blood first enters the heart via the superior and inferior vena cava and fills the right atrium (Fig 1). Contraction of the atria causes this blood to be pumped into the right ventricle. After blood fills the right ventricle, it contracts and the tricuspid valve closes, preventing backflow of venous blood into the right atrium. As the right ventricle contracts to pump venous blood to the lungs, the pulmonary valve opens to allow the blood to flow through the pulmonary artery to the lungs. The valve then closes to prevent backflow of the blood into the right ventricle. This blood then flows to the lungs and the red blood cells receive oxygen. This oxygenated blood then returns to the heart via the pulmonary vein and fills the left atrium. As the left atrium contracts, the mitral valve opens, sending blood into the left ventricle. Similar to the right ventricle, as the left ventricle contracts, the mitral valve closes to prevent backflow into the atria, and the aortic valve opens, sending the oxygenated blood out of the heart through the aorta. After this oxygenated blood flows through the aorta, the aortic valve closes again to prevent backflow of this blood into the left ventricle.

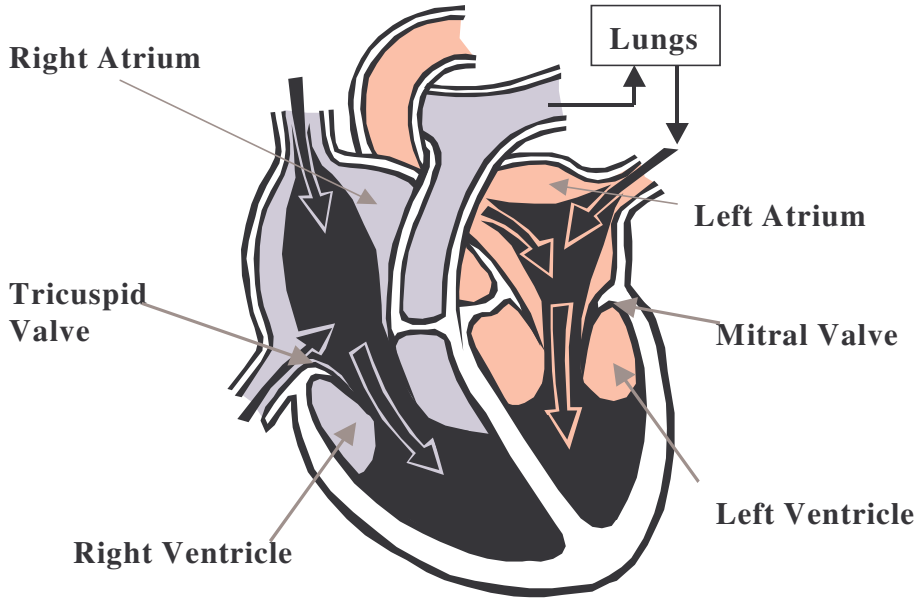


Figure 1: Blood in the heart travels from the right atrium to the right ventricle and into the lungs. After receiving oxygen in the lungs, the blood travels to the left atrium, the left ventricle and then out to the body.

Special Conductive Tissues in the Heart

There are several specialized regions within the heart to initiate electrical signals that cause cardiac contraction (Fig 2). The primary area responsible for cardiac activation is the sinus node (also known as the sinoatrial or SA node). The SA node is located at the top of the right atrium and is the major structure responsible for pacing the heart. Connecting the SA nodes to the atrioventricular (AV) nodes are the internodal pathways. These internodal pathways are located along the walls of the right atrium. The electrical signal propagates down the internodal pathways and enters the AV node. At the AV node the signal is slightly delayed. The AV node is located in the heart septum, between the right and left atrium. After the AV node, the electrical signal flows through the Bundle of His, located in the septal wall between the left and right ventricles. The Bundle of His then divides into two branches, the right branch and left branch. These branches continue along the septal wall, and then go into the Purkinje fibers, which innervate the right and left ventricular walls.

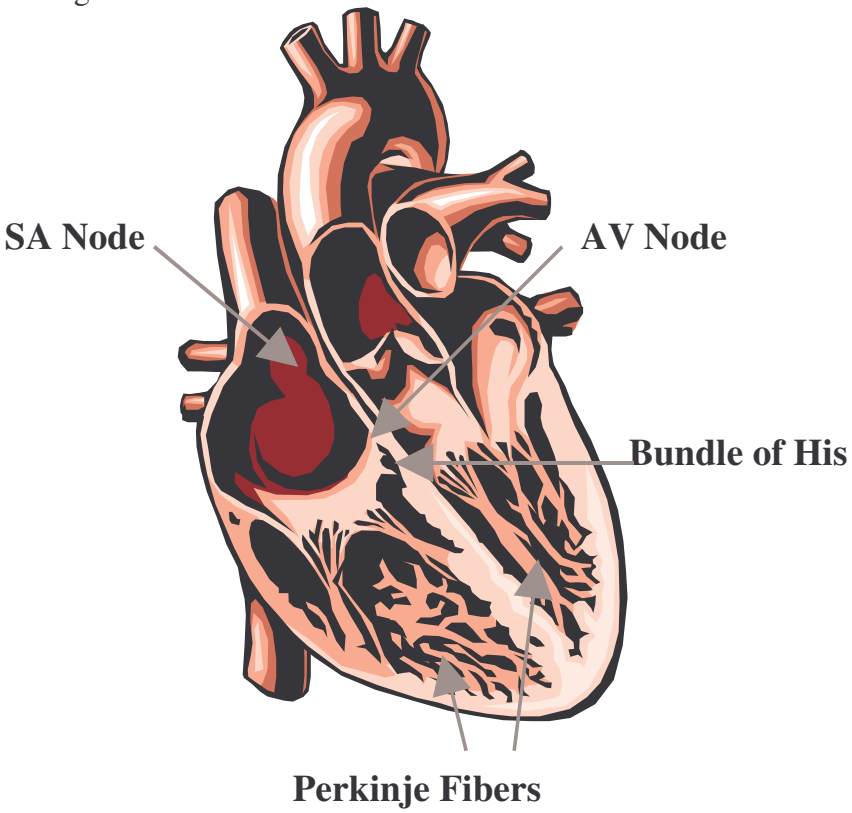


Figure 2: Conducting Pathways of the Heart

Origin of the ECG Signal

In normal cases, the SA node is the heart's natural pacemaker with the autonomic nervous system to regulate its excitation. Therefore, the electrical impulse responsible for the cardiac cycle originates at the SA node. Pulses from the SA node propagate via the internodal fibers of

the right atrium, and then to the left atrium, causing immediate atrial contraction. This electrical potential then travels to the AV node. At the AV node, the depolarization potential is then delayed, allowing the atria to fully contract. This delay allows the atrium to completely empty its contents into the ventricles before ventricular contraction. After this delay in the AV node, the potential travels down the Bundle of His, which splits into the right and left branch bundles. These bundles then innervate the ventricular walls via the Purkinje fibers. When the signal reaches the Purkinje fibers, ventricular contraction occurs, sending blood from the right ventricle into the lungs and blood from the left ventricle out the aorta. This process then repeats for the next heartbeat.

Other tissues in the heart also have natural pacing rates controlled by the autonomic nervous system. The AV node, without outside stimulation, has a natural discharge rate of 40 to 60 times a minute, while the Purkinje fibers fire between 15 and 40 times a minute. This is in contrast to the SA node that fires between 70 and 80 times a minute. The reason that neither the AV node nor the Purkinje fibers are responsible for setting the heart rate is due to the discharge rate of the SA node. The SA node fires faster than the AV node or Purkinje fibers, so these other tissues are excited from the SA impulse rather than their own rhythmic rate.

In normal conditions, the SA node is the natural pacemaker of the heart. However, sometimes the AV node or Purkinje fibers begin pacing faster than the SA node. This condition is known as an ectopic pacemaker. An ectopic pacemaker occurs when electrical activation of the heart is initiated elsewhere than the SA node.

Another condition that can lead to an ectopic pacemaker is when the signals from the SA node are prevented from conducting to the rest of the heart. This usually occurs when the signal is blocked at the AV node or the AV fibers that innervate the ventricles. In this instance, the SA node fires at its own normal rate, but these signals do not conduct down to the ventricles. Since the Purkinje fibers do not receive these impulses from the SA node, they begin to fire at their own intrinsic rate, between 15-40 times a minute. This leads to a very slow contraction rate of the ventricles, failing to pump blood. If this continues, the brain may become deprived of oxygen, and the person may faint.

A more detailed description and analysis of particular cardiac arrhythmias is presented in Laboratory session ECG II.

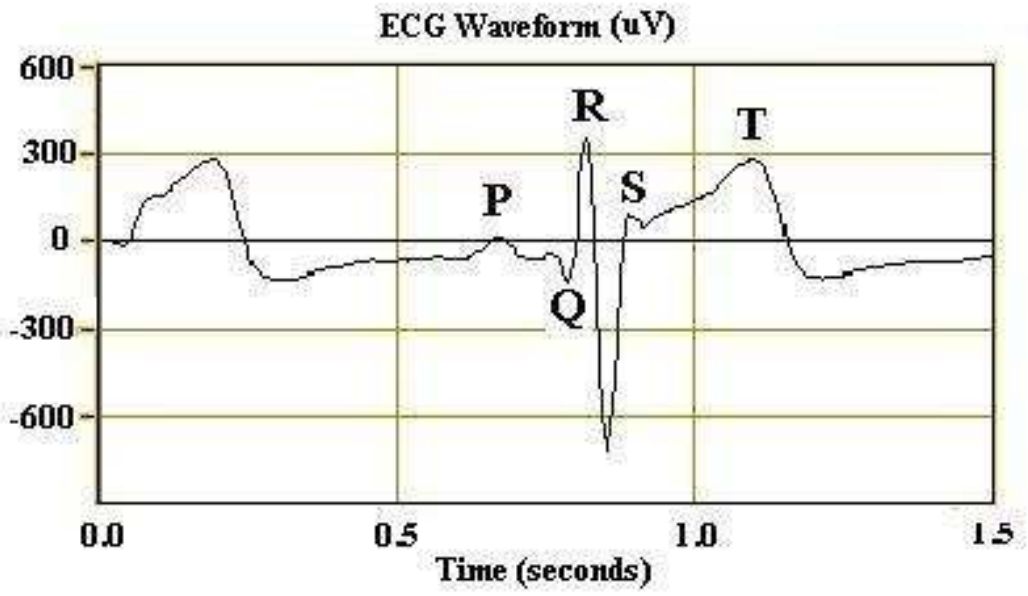


Figure 3: Typical ECG with P, QRS, and T complexes marked.

Correlation of ECG to Physiological Events

The ECG signal (Fig 3) illustrates the electrical depolarization and repolarization of the heart during a contraction. As described above, the depolarization of the cardiac muscle cells in the atrium occurs first. Therefore, the first wave in the ECG signal corresponds to the depolarization of the atrium. This is known as the P wave. Similarly, the start of ventricular contraction is the QRS wave. The ventricles stay contracted for a few milliseconds until ventricular repolarization occurs, which is seen as the T wave. Atrial repolarization typically occurs between 0.15 to 0.20 seconds after the P wave. However, this is the same time when the QRS complex occurs. The QRS complex is of much greater amplitude than atrial repolarization so it dominates the signal.

Typical Duration and Amplitudes

The voltage of the ECG signal can vary depending on the location of the electrodes placed on the body. If the electrodes are located close to the heart, the recorded potentials can be as high as 5 mV. However, if the electrodes are placed further apart, such as at the wrists, a typical value is 1mV. Both of these measurements, however, are small compared to electrodes placed directly in contact with the heart muscle membrane. Here the potential can range as high as 110 mV. Typical amplitudes are around 1mV for the top of the Q wave to the bottom of the S wave, 0.1 - 0.3 mV for the P wave, and between 0.2 - 0.3 mV for the T wave.

The PQ interval (also known as the PR interval) is the amount of time from the beginning of the P complex to the QRS complex. This represents the amount of time between the beginning of atrial contraction and the beginning of ventricular contraction. The normal duration is approximately 0.16 seconds. Similarly, the QT interval is the time between ventricular contraction and ventricular repolarization. This is measured from the beginning of the Q wave to the end of the T wave and typically lasts 0.35 seconds. The heart rate can be determined directly from the ECG. The heart rate is the inverse of the time between similar segments in the ECG recording. For example, if the time measured between two QRS complexes is 0.8 seconds, then the number of beats per second is the inverse, 1.25 beats / second. In order to obtain the heart rate per minute, you would simply multiply by 60 seconds/minute. This would yield 75 beats per minute.

Electrode Configuration

There is a standard placement of electrodes when performing ECG recordings called a standard bipolar limb lead. A lead refers to the potential difference between two electrodes. For this lab, lead placement involves three leads, which are placed on the right arm (RA), left arm (LA), and left leg (LL). The electrodes can be attached to the wrists and inner ankle, but for clinical applications, are usually attached to the chest for a more accurate signal. Leads I, II, and III constitute the standard limb lead ECG connected as follows:

Lead	+	-
I	LA	RA
II	LL	RA
III	LL	LA

Table 1. Standard bipolar limb lead ECG configuration.

In the table, the positive and negative signs denote the polarity of the leads. So, the positive end of Lead I connects to the LA, while the negative end of Lead I connects to the RA. Using these three leads, we can form what is called Einthoven's Triangle. This is a representation of vectors demonstrating the formation of the ECG signal. In interpreting these measurements, each lead is assumed to be equivalent to measurements taken across all sides of an equilateral (Einthoven's) triangle, which is superimposed over the chest, as shown below:

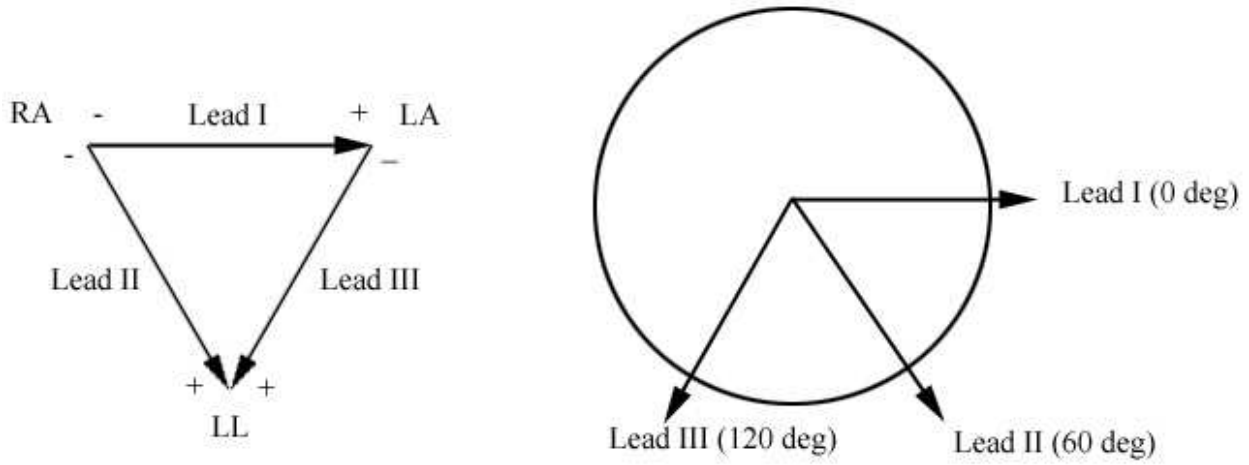


Figure 4: Einthoven's law and lead configuration.

With Einthoven's Triangle, there is an equation that relates all three vectors. Graphically, Einthoven's Law says that if the potentials of the first two leads are known, than the third lead can be found by adding the two vectors together. Mathematically, Einthoven's law states that for the potentials on each lead:

Lead I + Lead III = Lead II

Some may notice that this equation is similar to Kirchoff's Voltage law, which states that all of the voltages in a loop must equal zero. Using this equation, we only need to record two of the leads. The third lead can be determined mathematically, provided that the two leads were measured simultaneously. Einthoven's Triangle also allows us to determine the mean electrical axis of the heart. This mean electrical axis is the vector representing the summation of all the vectors that occur in a cardiac cycle. This electrical axis can be thought of as a dipole. The dipole illustrates the strength and direction of the heart's polarization during a cardiac cycle. There are two ways of determining the mean electrical axis. Lead I measures lateral voltage and the other two measure from top to bottom. One method is to measure the magnitude of the R complex along Lead I and Lead III, and to extrapolate the vector of Lead II, which would give the magnitude and angle of the vector. A more accurate way of measuring the mean electrical axis would be to add the Q, R, and S potentials for the two leads, instead of only the R wave. The QRS potentials are measured along Lead I and III, added together, and then the mean electrical axis can be computed by finding the magnitude and direction of the vector representing Lead II. If a complete measurement of the mean electrical axis is desired, twelve leads are required, since the mean electrical axis is precisely defined in three dimensions, x, y, and z. In this lab, we will only focus on the frontal plane mean electrical axis. In normal conditions, the mean electrical axis of the heart is typically around 60 degrees.

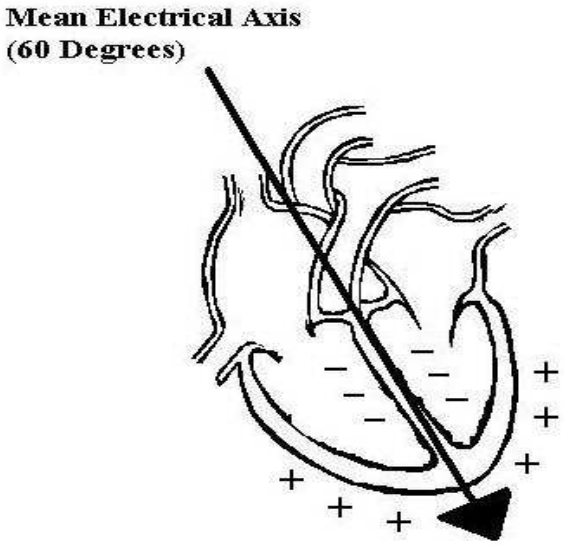


Figure 5: Mean electrical axis of the heart.

Vector Analysis of the Electrocardiogram

The ECG signal that is recorded can be derived from the Leads I-III vectors. When the ECG signal is recorded, the vector values for each of the leads changes as the atria and then ventricles contract. For example, as the QRS wave occurs, the lead I vector has a very small magnitude. This describes the slow upward growth of the lead I ECG recording. As depolarization sweeps across the atria and into the ventricles, the lead I vector begins to increase, causing the fast growth in the lead I ECG signal that is typical of the QRS complex. Then, as more of the ventricles depolarize, the lead I vector starts becoming smaller since all of the ventricular muscle has become depolarized, causing the lead I vector to have zero or slightly negative magnitude, causing the negative slope of the ECG signal in lead I. A similar analysis can be performed on the other leads and can also explain how repolarization sweeps across the heart when the T wave occurs.

Experimental Methods

Experimental Setup

During this laboratory you will record a standard three lead ECG. You should watch the setup movie included with the software prior to setting up the experiment.

1. Your BioRadio should be programmed to the “LabECGI” configuration.

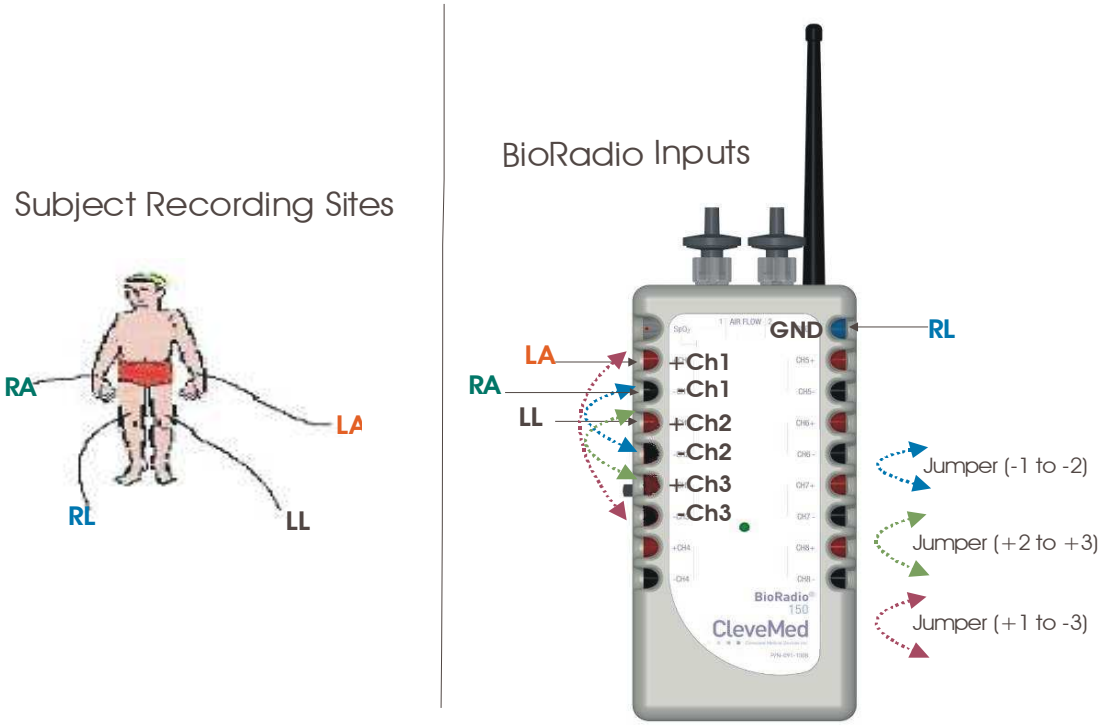


Figure 6. ECG Setup

2. For this laboratory you will need to use four snap electrodes from the CleveLabs Kit. Remember that the electrode needs to have good contact with the skin in order to get a high quality recording. The surface of the skin should be prepared and cleaned prior to electrode placement. For the best recordings, it is best to mildly abrade the surface with pumice or equivalent to minimize contact resistance by removing the outer dry skin layer. Attach one electrode on the palmar side of the right wrist, one on the palmar side of the left wrist, one on the left leg, and one on the right leg. **NOTE: The electrodes on the arms can be placed at the wrists and the electrodes on the legs can be placed near the ankles**.

3. After the electrodes have been placed on the subject, connect one snap lead to each of the four electrodes. Then, connect those snap leads and jumpers to input channels 1, 2, 3 and the ground using the picture above as a reference (Fig 6). Refer to your BioRadio User's guide for more information on setting up the system.

Procedure and Data Collection

1. Run the CleveLabs Course software. Log in and select the "Electrocardiography I" laboratory session under the Basic Physiology subheading and click on the "Begin Lab" button.

2. Turn the BioRadio ON.

3. Click on the ECG data Tab and then on the green "Start" button. Three channels of ECG should begin scrolling across the screen.

4. The first part of this lab session will record normal resting ECG with the subject sitting up and laying down. It is important that the subject is relaxed and still during this procedure in order to prevent artifacts from contaminating the ECG signal.

5. For the first test, have the subject lie down on the floor or a cot. The subject's ECG should begin scrolling across the monitor. You may need to adjust the plot scales to see the ECG clearly. Instruct the subject to relax then click on the save button and record data for approximately 10 seconds. Name the data file "layingECG". Also, click on "Report" to capture a screen shot of the data scrolling.

6. With the subject still laying relaxed, click on the Spectral Analysis tab to complete a spectral analysis. Depending on your surroundings, it is likely that there is some 60Hz noise in the signal. Note where the peak frequency of the signal occurs. Report a screen capture of the raw, unfiltered spectral analysis screen. You may need to adjust the scaling.

7. Setup filter parameters that will remove the 60Hz noise from the signal and Report a screen capture of the filtered spectral analysis to verify this.

8. Request the subject to sit up in a chair, and place his/her arm on a table or armrest. Make sure the subject is relaxed and quiet. Then save another 10-second segment. Name this data file "sitECG".

9. Next, instruct the subject to wave their left hand around in space while you are recording ECG. Save this data to file and name the file "ECGartifactleft".

10. Finally, instruct the subject to wave their left hand around in space while you are recording ECG. Save this data to file and name the file "ECGartifactright".

Data Analysis

1. Using the Post Processing Toolbox, open the data file named "sitECG".

2. Click on the spectral analysis tab and make sure you are on the time domain subtab. Change the time scale such that one beat is shown in the window. Report a screen shot of this to a new report.

3. Set the low pass filter to 20 Hz and examine the effect on the ECG signal. Report a screen shot of this to your report.

4. Using the post processing toolbox, open the data files labeled "ECGartifactright" and "ECGartifactleft". Examine each of the three leads in each file. On which leads can you detect an ECG signal? On which leads is the ECG signal distorted? Explain why.

5. Using Excel, MATLAB, or LabVIEW, import the data file "layingECG". Plot the first four beats from channel 1. Also, determine the resting heart rate of the subject based on this recording. Repeat this for the file "exercise". Label the P, Q, R, S and T segments of one beat on both plots.

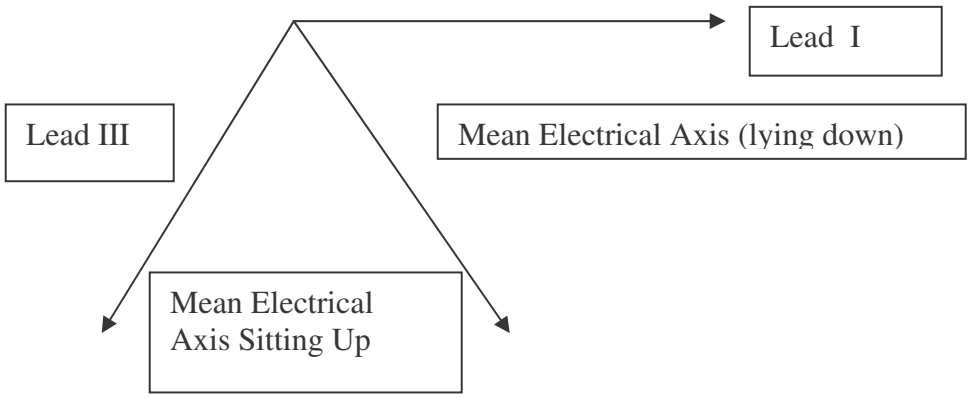
6. Using Excel, MATLAB, or LabVIEW, open the file "layingECG". Einthoven's Law stated that the sum of the potentials from all three channels should equal zero. Using this relationship, calculate what lead two should be, as if data for the first four beats was only available from leads I and III. Plot this calculated lead II, along with the measured lead II. Then, subtract the calculated lead II from the measured lead II and plot this error over time. Give a mean error between the calculated lead II and the actual lead II measurements.

7. Using Einthoven's triangle, the mean electrical axis can be approximated by adding the vectors for leads I and III, and then computing the direction and magnitude of the lead II vector, which is the mean electrical axis of the heart.

   To do this measurement, draw a figure similar to the one next to Einthoven's triangle (Fig 4), except omit the line representing lead II. Use a protractor to ensure that the angle between leads I and III is 120 degrees. For both the lead I and lead III vectors, draw twenty evenly spaced tics on the vectors, ranging from 0 to 2 mV.

8. One way of determining the mean electrical axis is to measure the amplitude of the R wave on leads I and III. Use Excel to determine the mean amplitude of the R wave for leads I and III over three or more beats. Once known, draw a line perpendicular to the lead I and a line perpendicular to lead III vectors at the value of the R wave for the respective leads. (i.e., if the mean R wave amplitude is .8 for Lead I and .6 for Lead III, find the mark corresponding to .8 mV on Lead I, and draw a perpendicular line, and do the same for Lead III) Find the intersection of these two lines, and record the magnitude and direction of this vector. Remember that zero degrees is measured from the lead I vector. This vector we computed is the lead II vector, which indicates the mean electrical axis of the heart.

9. On the same graph, repeat the above steps on the file with the subject sitting up.



A more accurate method of computing the mean electrical axis of the heart is to add the Q, R, and S potentials together. Average the total net potentials over three heartbeats, and then repeat the steps above to determine the mean electrical axis.

Discussion Questions

1. Define Einthoven's Law, explain how it relates to Einthoven's Triangle, and explain why it is useful in cardiac recordings.

2. Explain the difference, if any, in the mean electrical axis when the subject is sitting up compared to lying down. What are possible causes for the shift of the mean electrical axis? In general, why is the mean electrical axis point along the Lead II?

3. Explain the relationship between the P, Q, R, S and T potentials and heart rhythm.

4. Why isn't atrial repolarization seen in the ECG? Why is the amplitude of ventricular depolarization so much greater than the rest of the complexes in the ECG?

5. What considerations need to be taken when placing electrodes for an ECG measurement?

6. On the spectral analysis of the ECG data, where do the peaks occur and why?

7. What are some potential sources of noise in an ECG recording?

8. What type of filtering can you use to improve the quality of an ECG signal? What are the typical cutoff ranges of the filter that you should use?

References

1. Guyton and Hall. Textbook of Medical Physiology, 9th Edition, Saunders, Philadelphia, 1996.

2. Normann, Richard A. Principles of Bioinstrumentation, John Wiley and Sons, New York, 1988.

3. Rhoades, R and Pflanzer, R. Human Physiology. *Third Edition.* Saunders College Publishing, Fort Worth 1996.