

BME331 Physiological Control Systems

Lab 2 Report

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Simulink Section

1-2 Chirp Input block (default parameter)

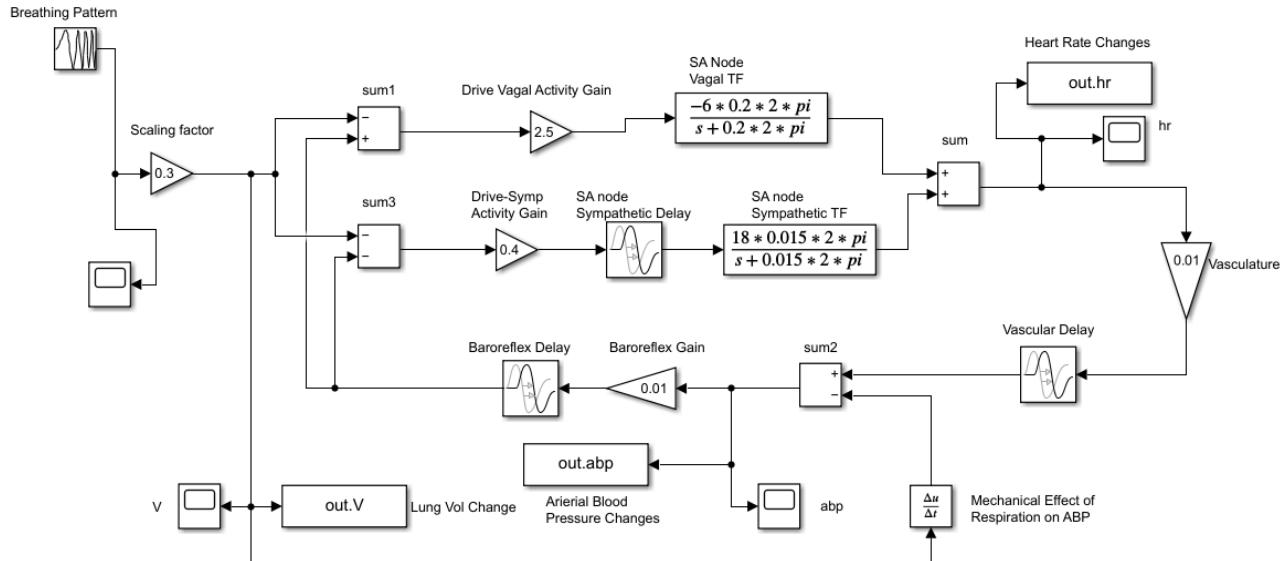


Figure 1. Simulink model with Chirp Input Block

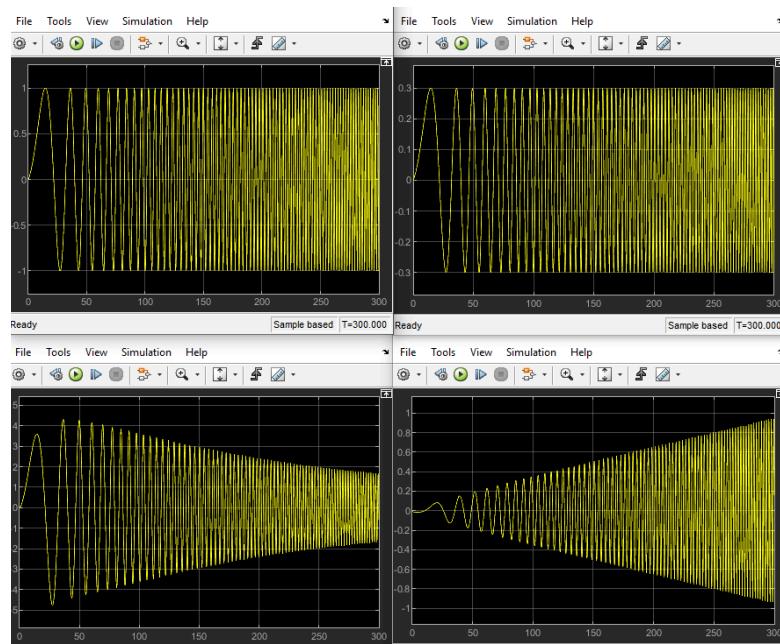


Figure 2. Plot of Outcomes vs. Time
 (left to right; top to bottom: breathing pattern, V, hr, abp)

3a) Band-Limited White Noise block

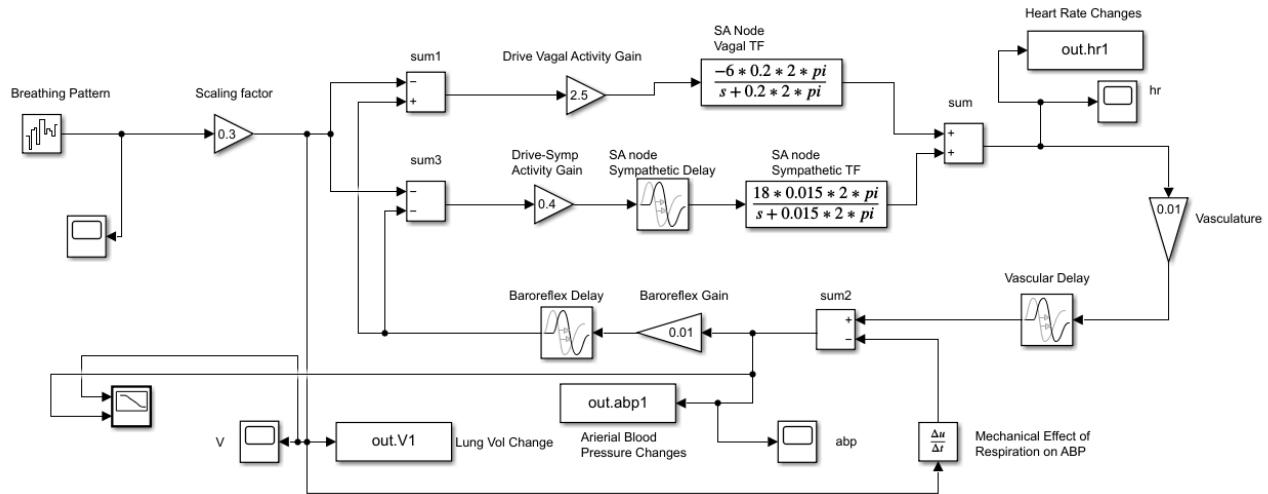


Figure 3a.1 Simulink model with Band-Limited White Noise Block

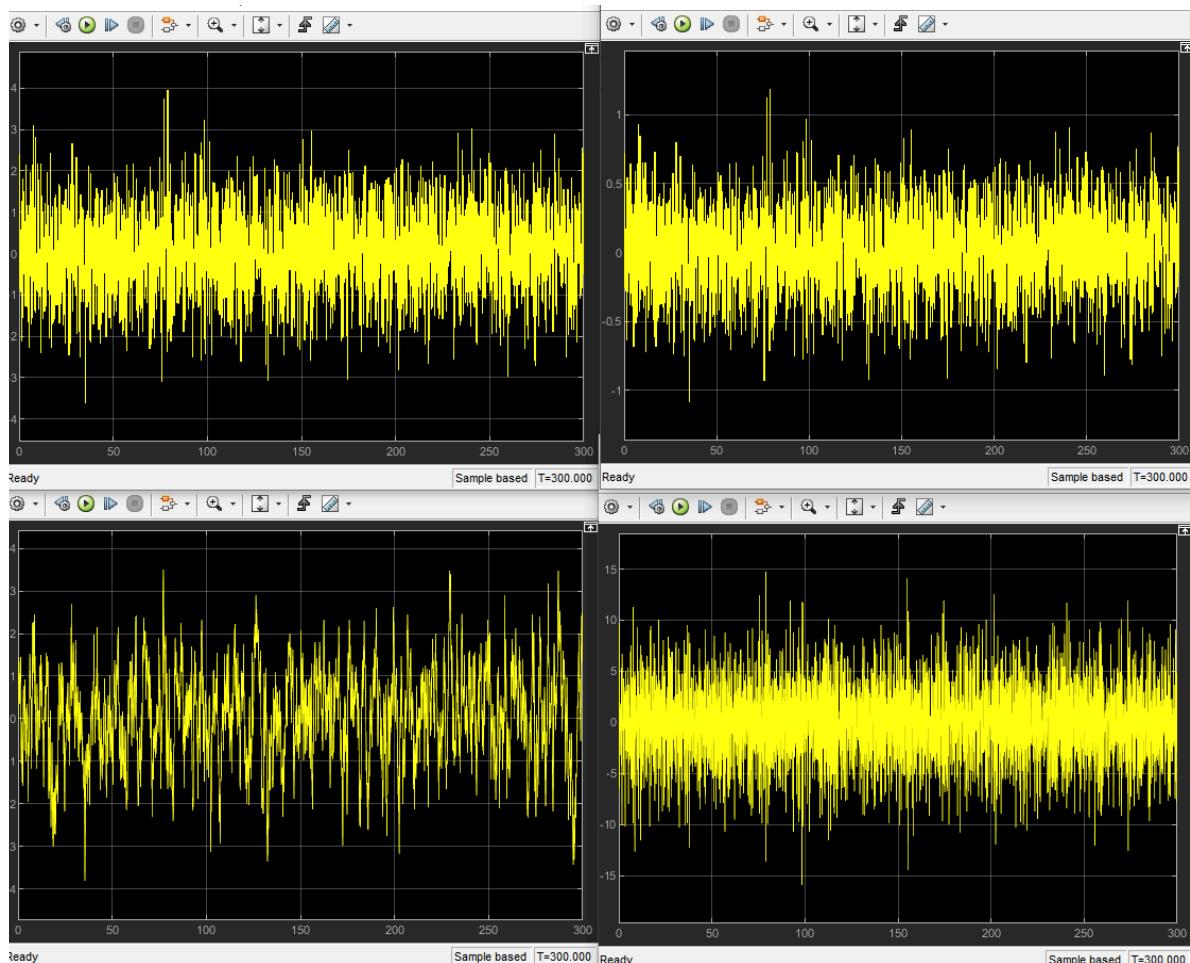
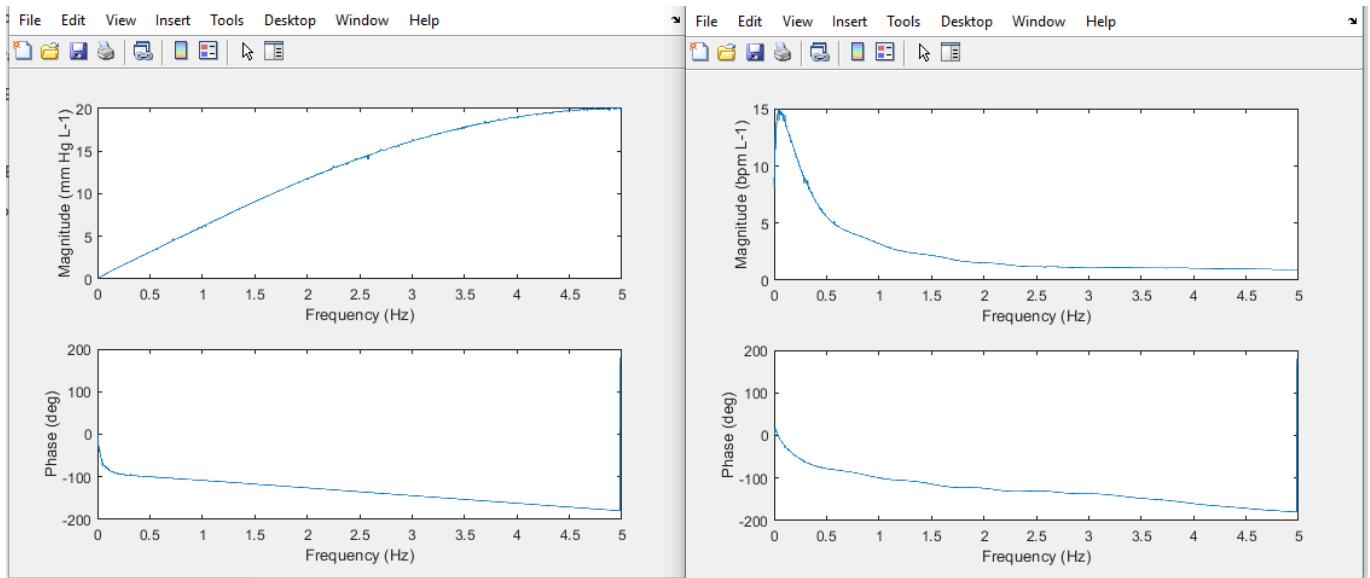
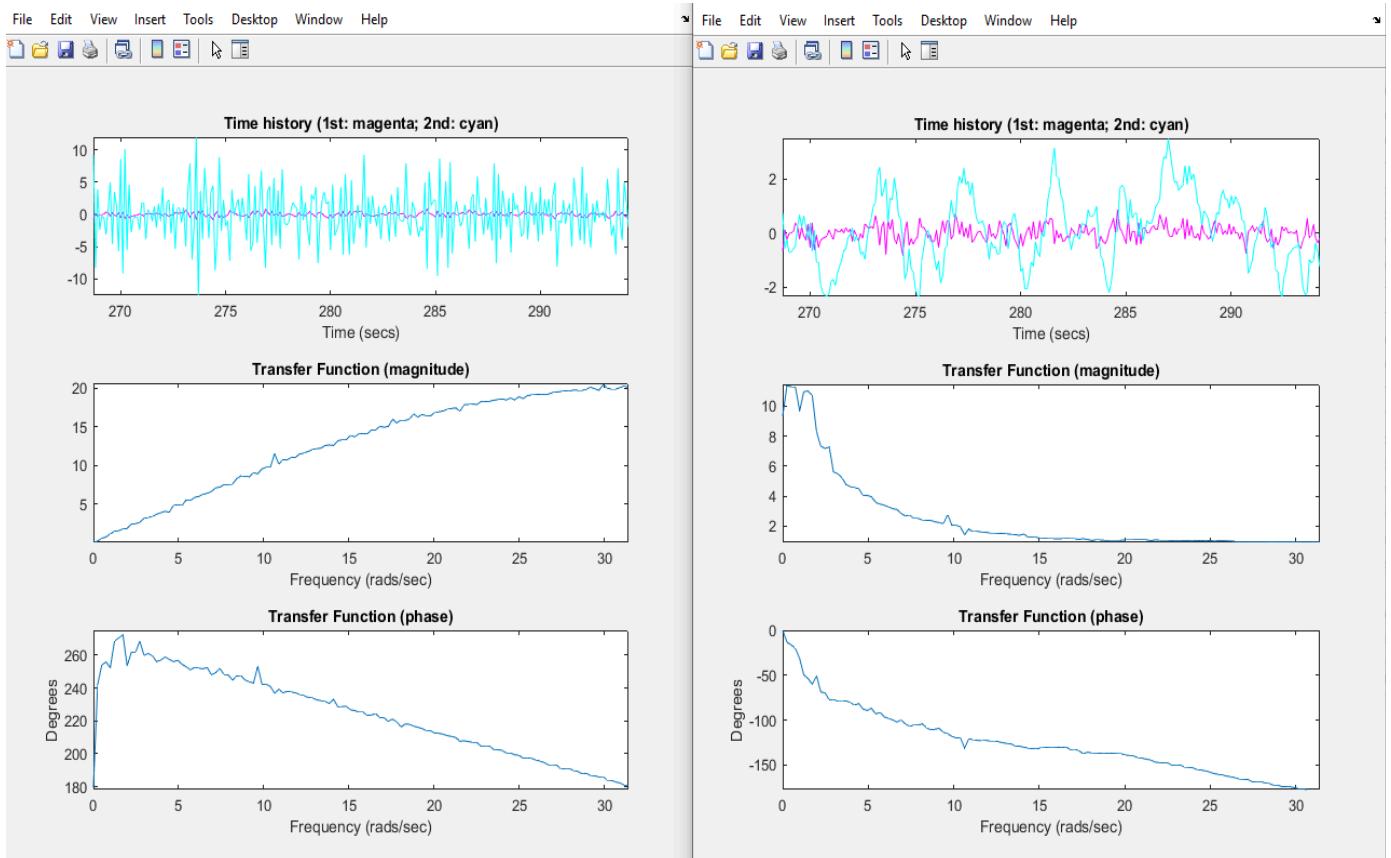


Figure 3a.2 Plot of Outcomes vs. Time
(left to right; top to bottom: breathing pattern, V, hr; abp)



*Figure 3a.3 Plot of Magnitude and Phase vs. Time
(Right: abp Left: hr)*

3b) Spectrum Analyzer



*Figure 3b. Plot of Spectrum Analyzer
(Right: abp Left: hr)*

3c) Sine Wave block

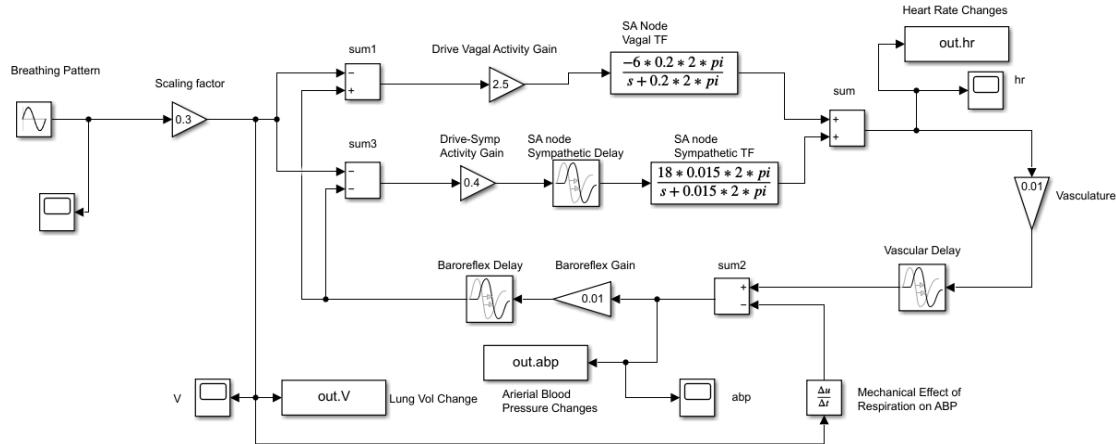


Figure 3c.1. Simulink model with Band-Limited Sine Wave Block

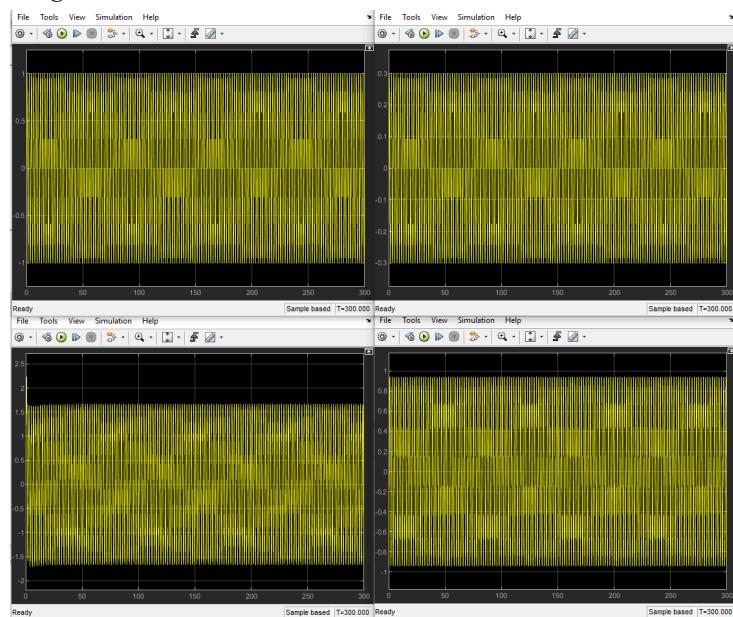


Figure 3c.2. Plot of Outcomes vs. Time (left to right; top to bottom: breathing pattern, V, hr, abp)

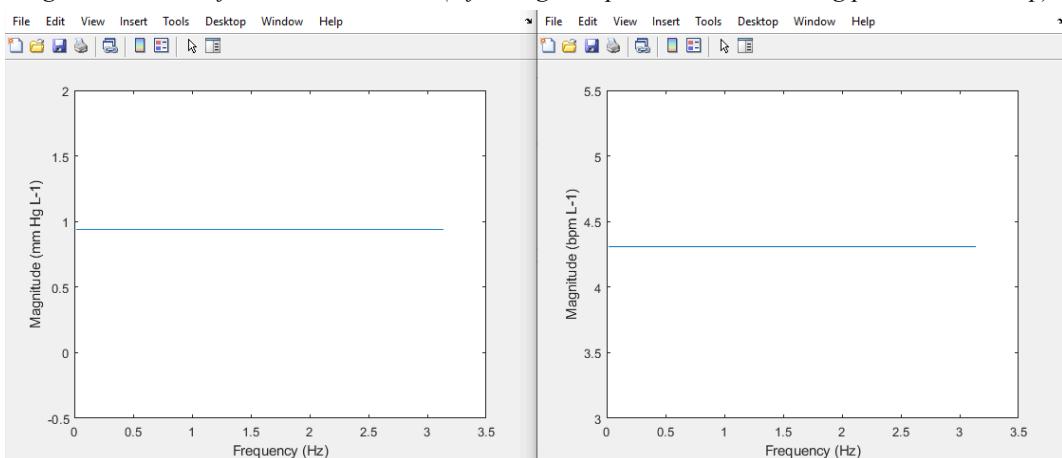


Figure 3c.3. Plot of Magnitude and Phase vs. Time (Right: abp Left: hr)

4a) Parasympathetic Nervous Modulation - Atropine

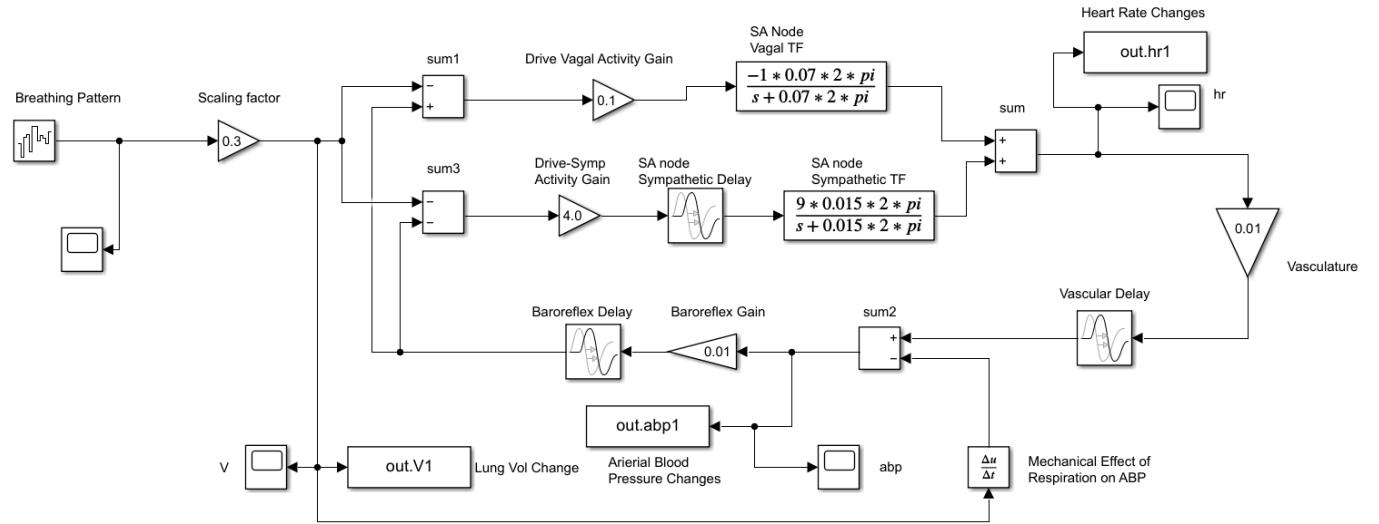


Figure 4a.1. Simulink model with Band-Limited White Noise Block - Atropine

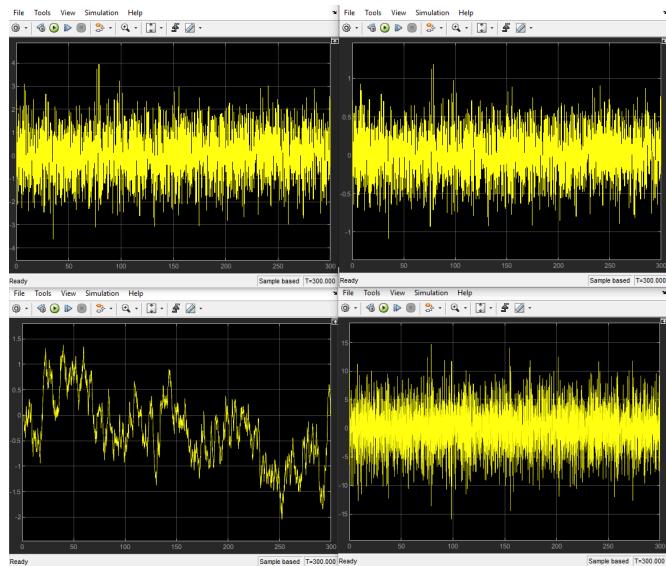


Figure 4a.2. Plot of Outcomes vs. Time (left to right; top to bottom: breathing pattern, V, hr; abp)

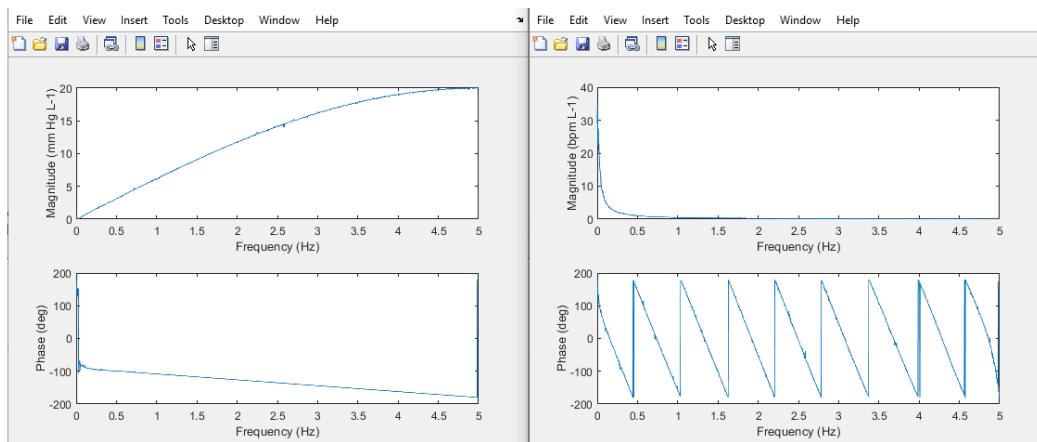


Figure 4a.3. Plot of Magnitude and Phase vs. Time (Right: abp Left: hr)

4b) Sympathetic Nervous Modulation - Propranolol

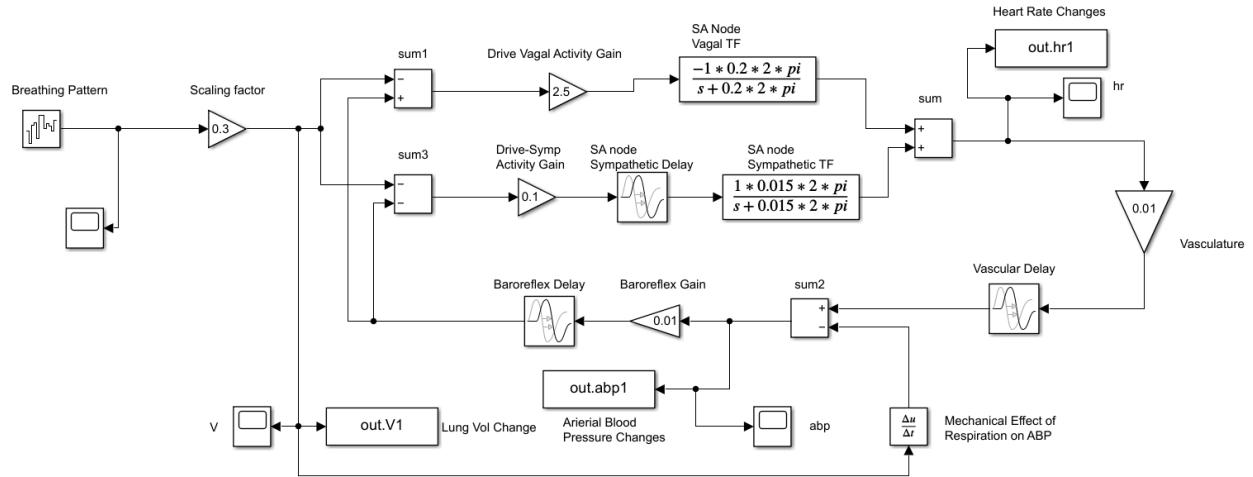


Figure 4b.1. Simulink model with Band-Limited White Noise Block - Propranolol

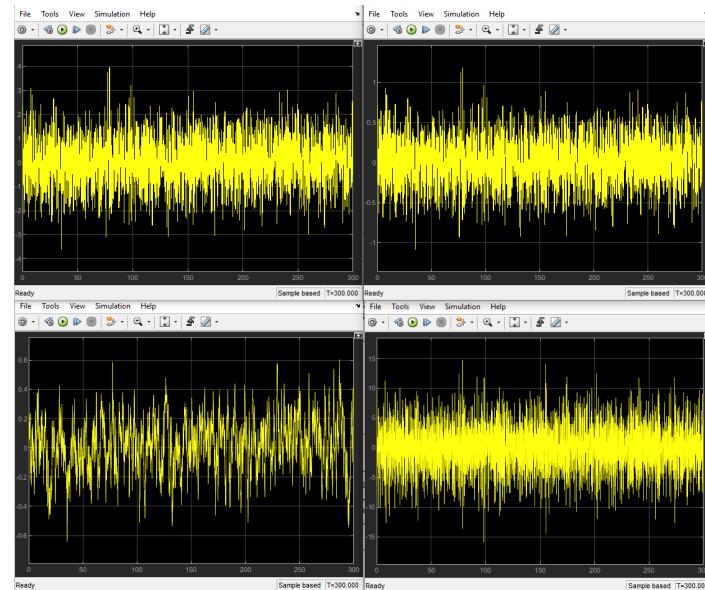


Figure 4b.2. Plot of Outcomes vs. Time (left to right; top to bottom: breathing pattern, V, hr, abp)

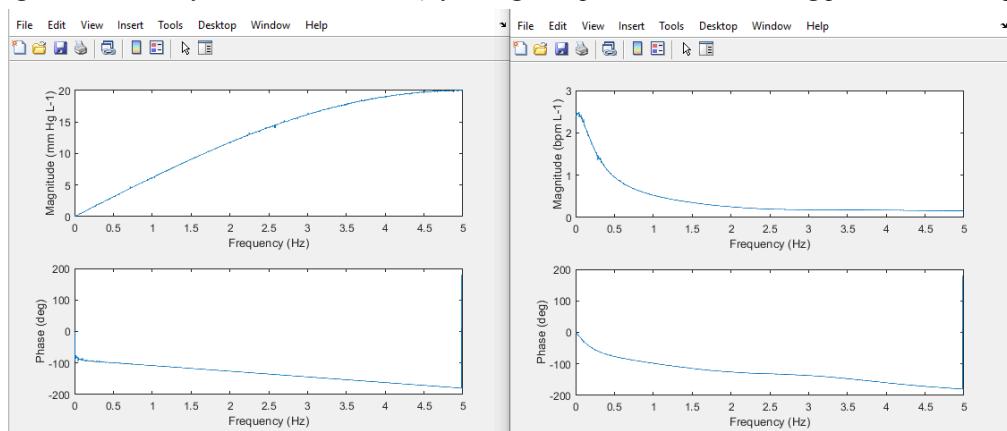


Figure 4b.3. Plot of Magnitude and Phase vs. Time (Right: abp Left: hr)

MATLAB Section

Using MATLAB, import the data file “baseECG”. 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 files “slowECG” and “fastECG”. Label the P, Q, R, S and T segments of one beat on each plot.

Base ECG

Figure 5.1 and 5.2 below are the resting heart beats plot.

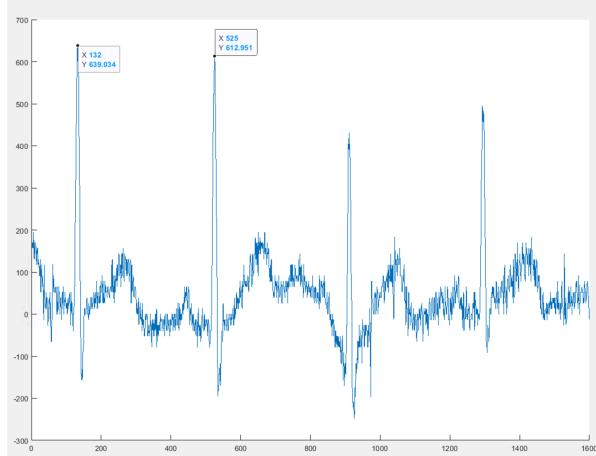


Figure 5.1. Base ECG Plot (Four beats)

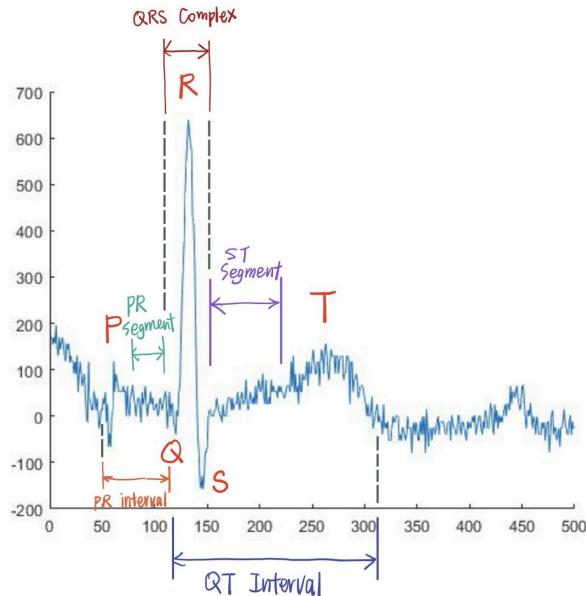


Figure 5.2. labeled Base ECG Beats Plot (One beat)

To determine the resting heart beat:

The time measured between two QRS complexes is $\frac{525-132}{600} = 0.655 \text{ seconds}$ (values obtained from the two labeled data points in figure 5.1)

The number of beats per second = $\frac{1}{0.655s} = 1.53 \text{ beats/second}$

Resting heart rate = $1.53 * 60 = 91.6 \text{ beats/min}$

Fast ECG

Figure 5.3 and 5.4 below are the fast breathing heart beats plot.

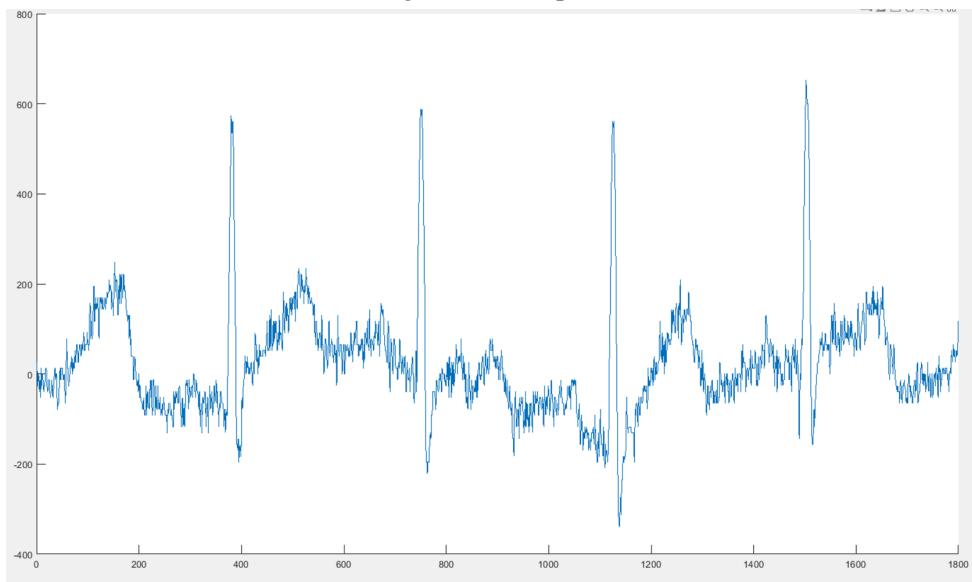


Figure 5.3. Fast ECG Plot (Four beats)

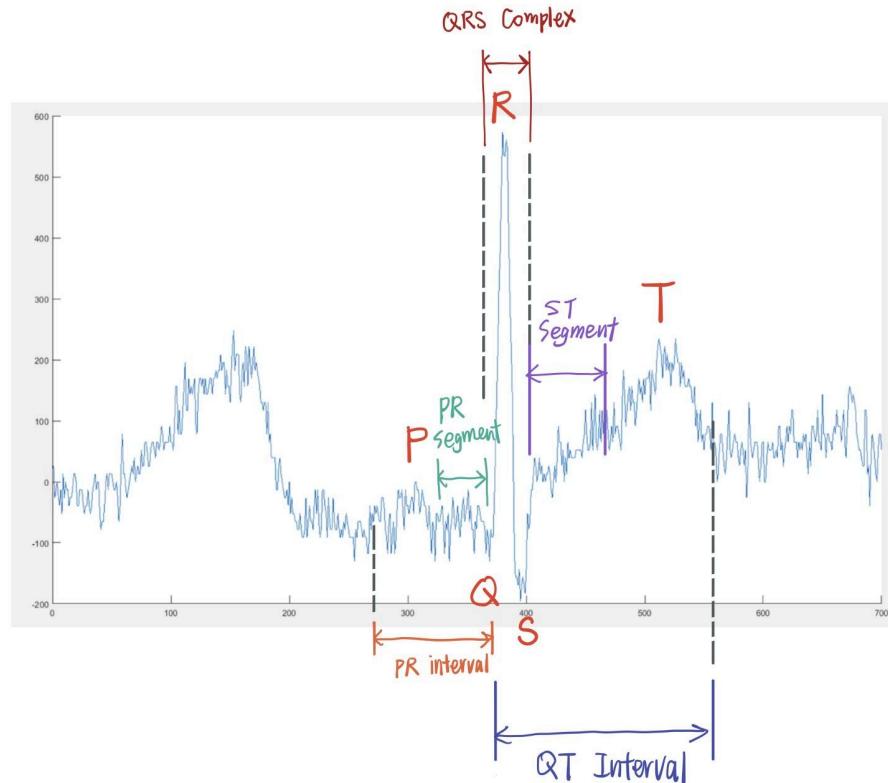


Figure 5.4. Labeled Fast ECG Plot (One beat)

Slow ECG

Figure 5.5 and 5.6 below are the slow breathing heart beats plot.

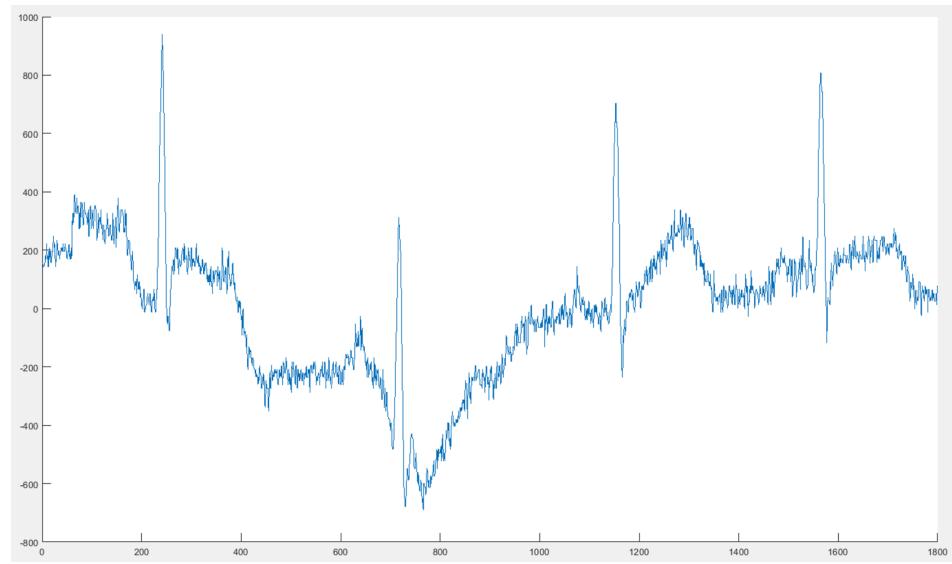


Figure 5.5. Slow ECG Plot (Four beats)

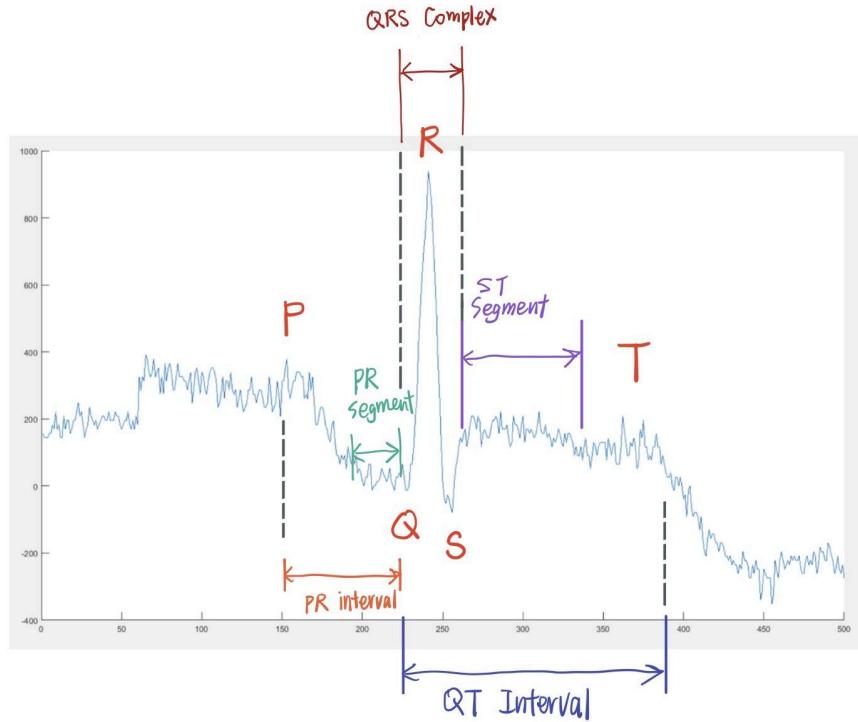


Figure 5.6. Labeled Slow ECG Plot (one beat)

Using MATLAB, plot the heart rate variability in each of the three recordings “baseECG”, “slowECG” and “fastECG”, using the method described in the Background section and the provided Matlab script. Also report the standard deviations of each of the resulting heart rate signals.

Standard deviation values:

- Baseline heart rate: 4.5698
- Slow heart rate: 8.9241
- Fast heart rate: 30.5103

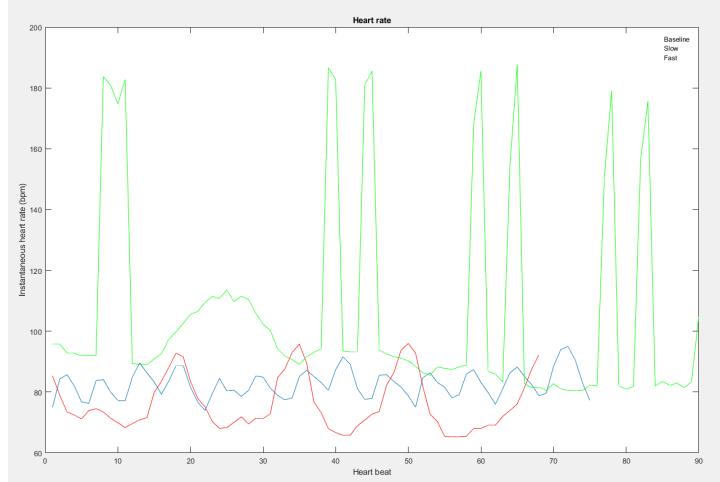


Figure 6. Heart rate variability plot in baseECG, fastECG and slowECG
Green line: fast breathing
Blue: baseline
Red: slow breathing

Using MATLAB, open the file “baseECG”. 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.

According to Einthoven’s Law, Lead I + Lead III → Lead II.

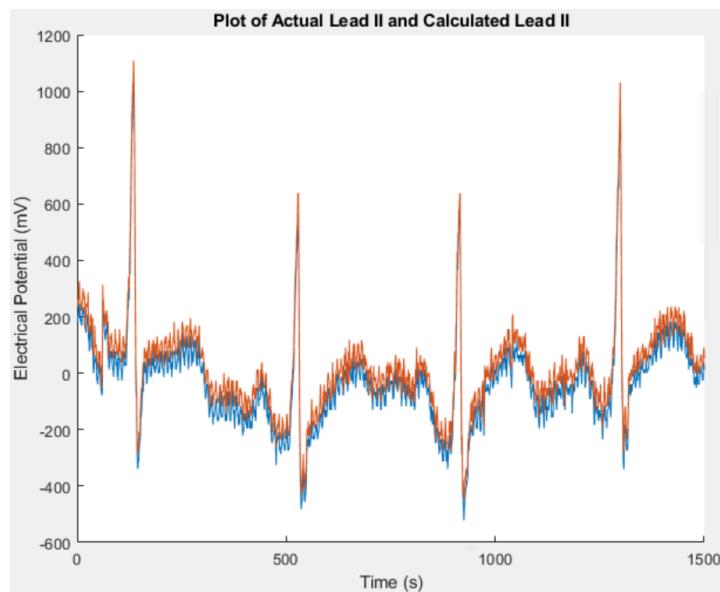


Figure 7.1. Plot of actual and calculated Lead II (red line: actual Lead II, blue line: calculated Lead II)

Error = (sum of calculated Lead II - sum of actual Lead II)/time = 51.8528

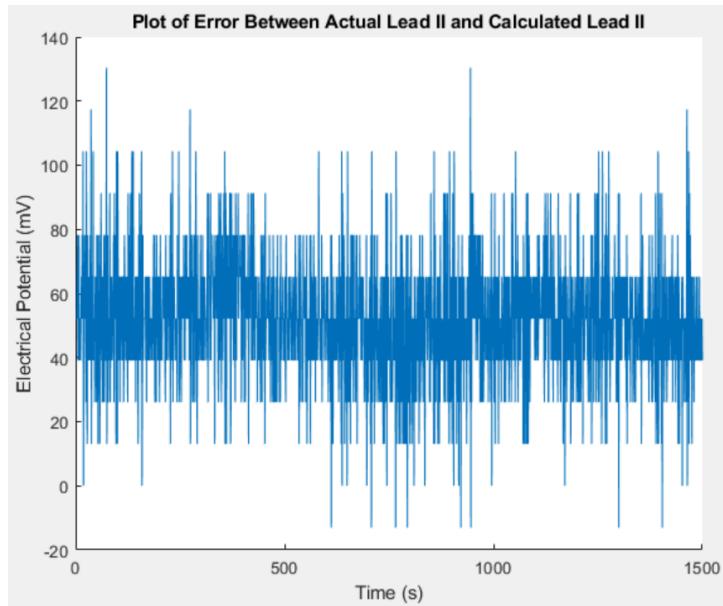


Figure 7.2. Plot of error between the calculated lead II and actual lead II

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, 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 ticks on the vectors, ranging from 0 to 2 mV.

One way of determining the mean electrical axis is to measure the amplitude of the R wave on leads I and III. 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.

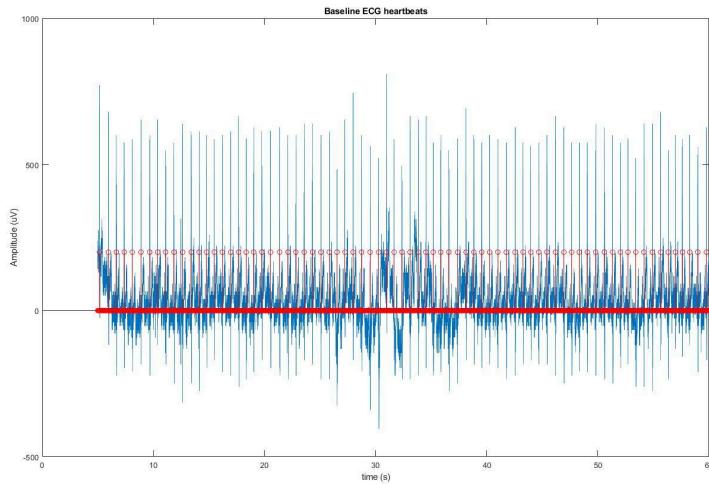


Figure 8.1. Baseline ECG heartbeats amplitude in time domain

The mean amplitude of the R wave for Lead I and Lead III is determined using figure 8.1 above.

$$\text{Lead I amplitude} = \frac{0.612+0.639+0.652+0.573}{4} = 0.619\text{mV}$$

$$\text{Lead III amplitude} = \frac{0.574+0.495+0.430+0.560}{4} = 0.515\text{mV}$$

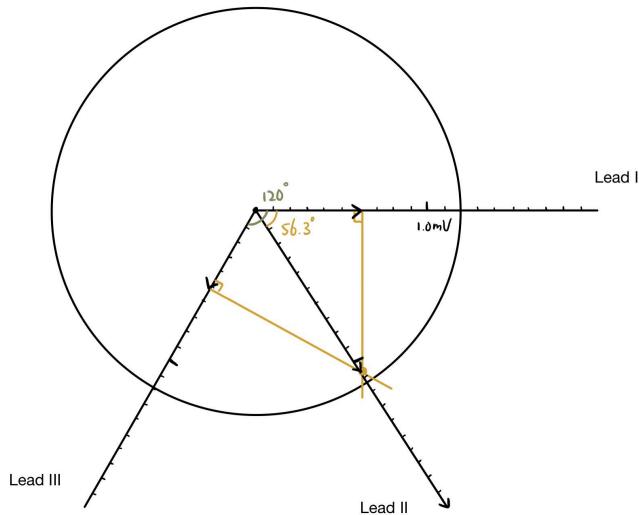


Figure 8.2. Determining the mean electrical axis

Figure 8.2 shown above is used to measure the mean electrical axis of the experimental ECG data. The magnitude of the vectors is the mean amplitude determined earlier. From this figure, it can be measured that the magnitude of Lead II is approximately 1.1mV, and the direction is 56.3 degree counterclockwise rotation from Lead I. This result makes sense as the typical mean electrical axis is around 60 degrees.

Electrocardiography Section

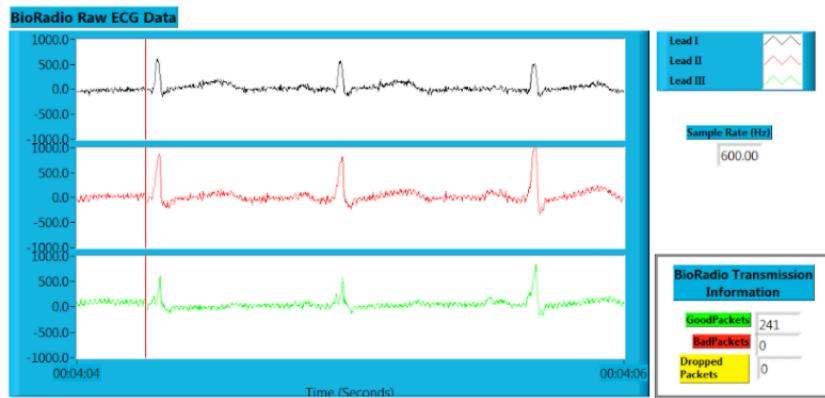


Figure 9.1. baseECG plots

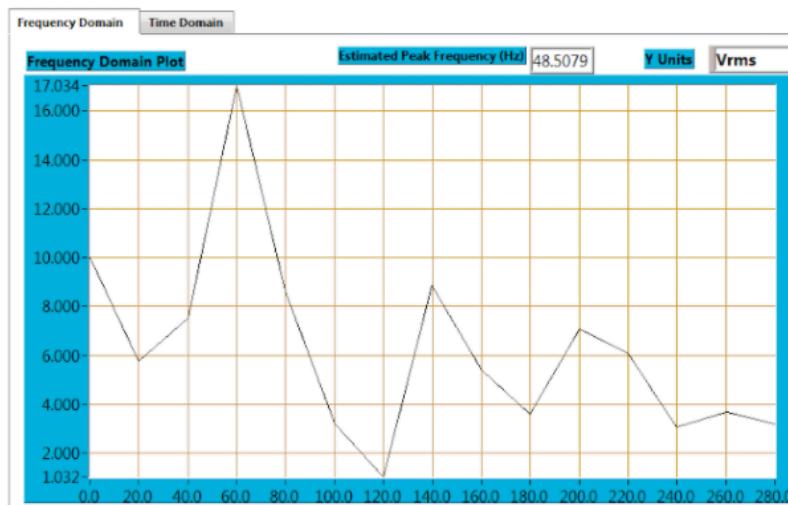


Figure 9.1.1. Raw spectral analysis of baseECG in the frequency domain.

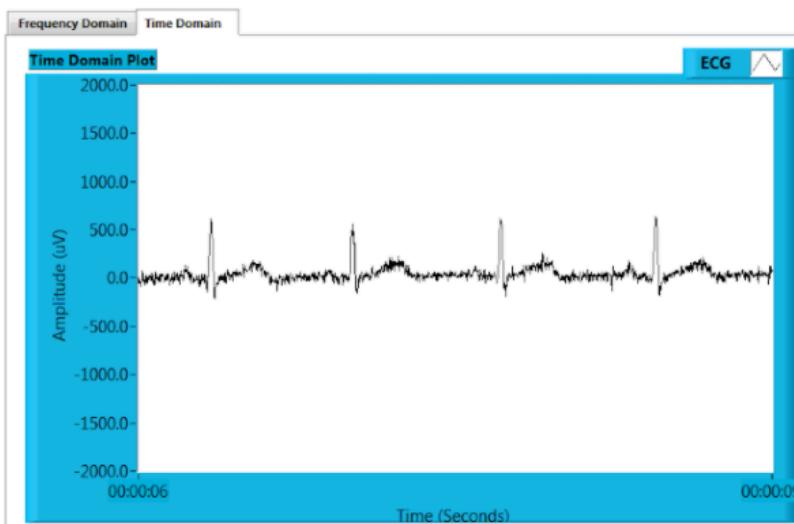


Figure 9.1.2. Raw spectral analysis of baseECG in the time domain.

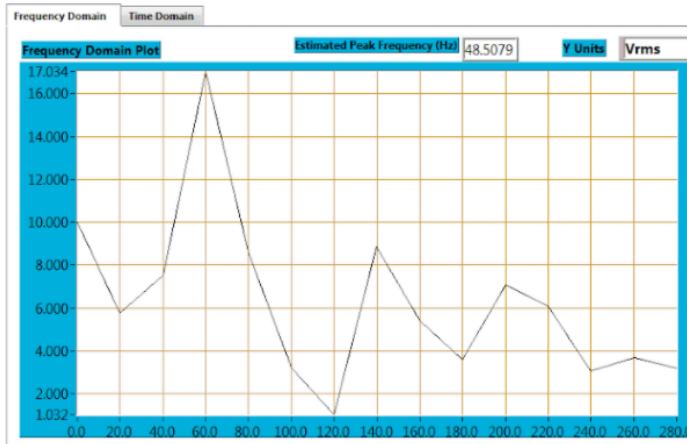


Figure 9.1.3. Spectral analysis of baseECG with 60 Hz band stop filter in the frequency domain.

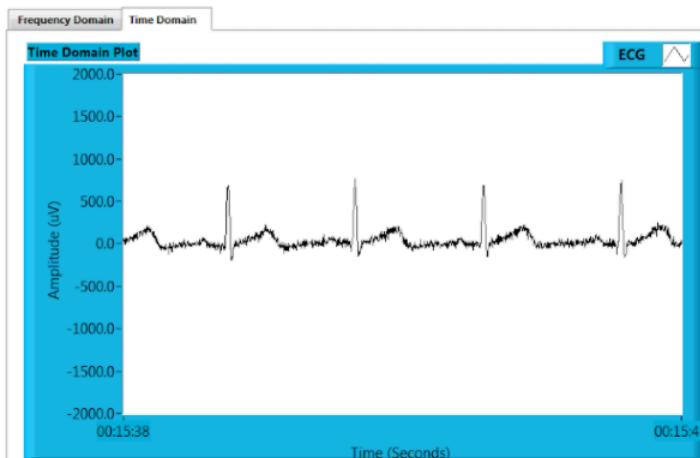


Figure 9.1.4. Spectral analysis of baseECG with 60 Hz band stop filter in the time domain.

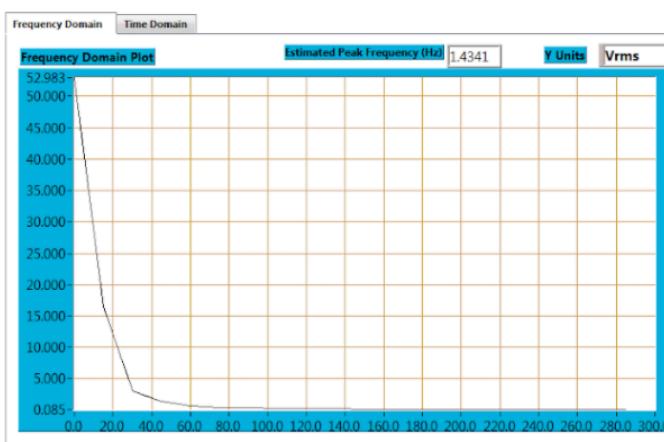


Figure 9.1.5. Spectral analysis of baseECG with a 20 Hz low-pass filter in the frequency domain.



Figure 9.1.6. Spectral analysis of baseECG with 20 Hz low-pass filter in the time domain.

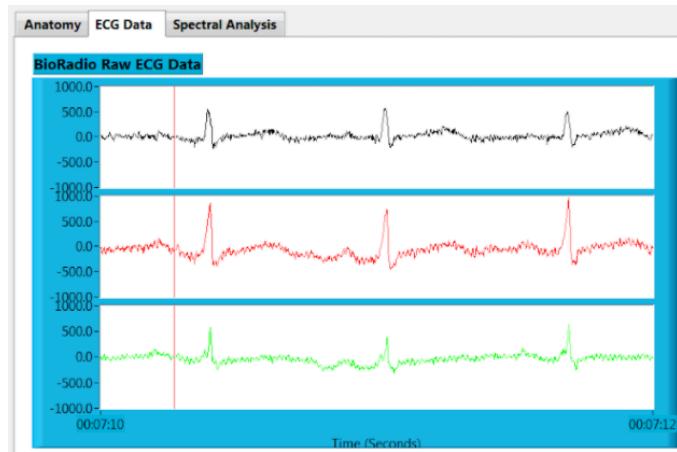


Figure 9.2. slowECG plots

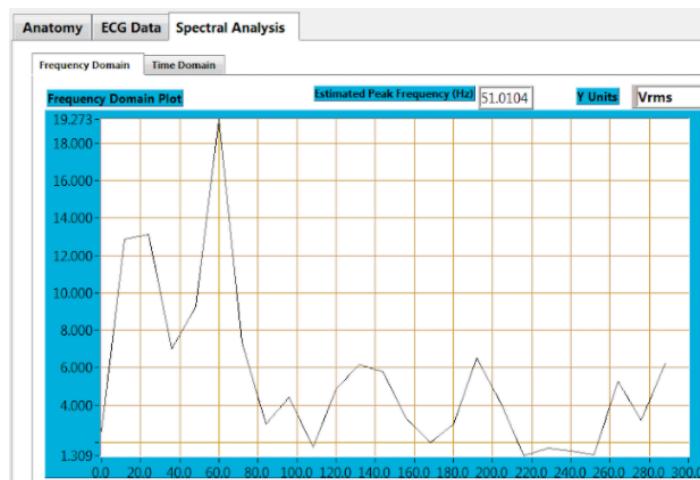


Figure 9.2.1. Raw spectral analysis of slowECG in the frequency domain.

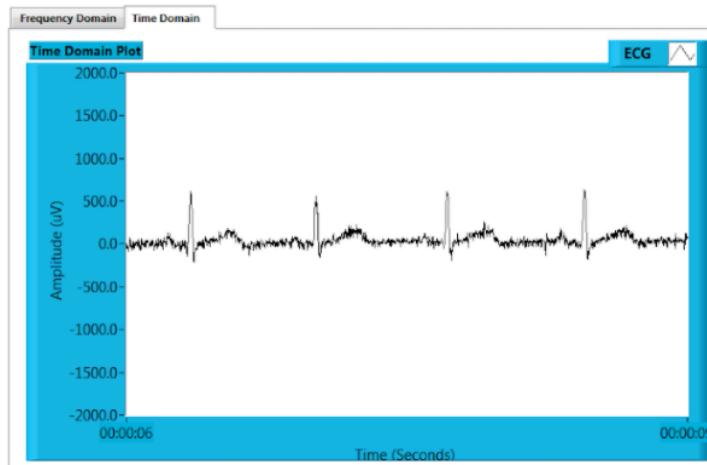


Figure 9.2.2. Raw spectral analysis of slowECG in the time domain.

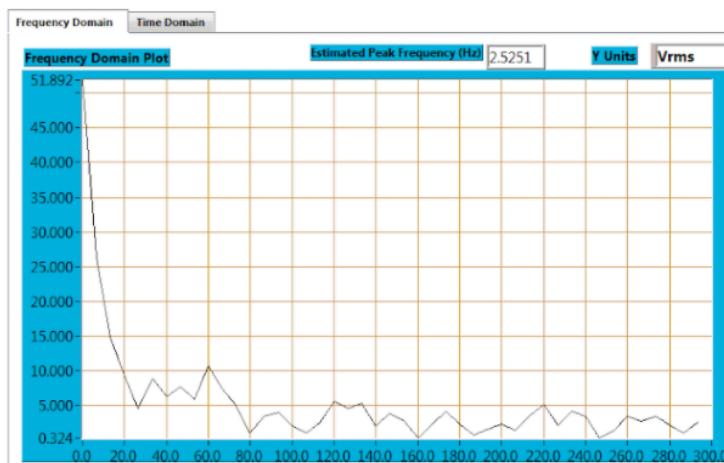


Figure 9.2.3. Spectral analysis of slowECG with 60 Hz band stop filter in the frequency domain.

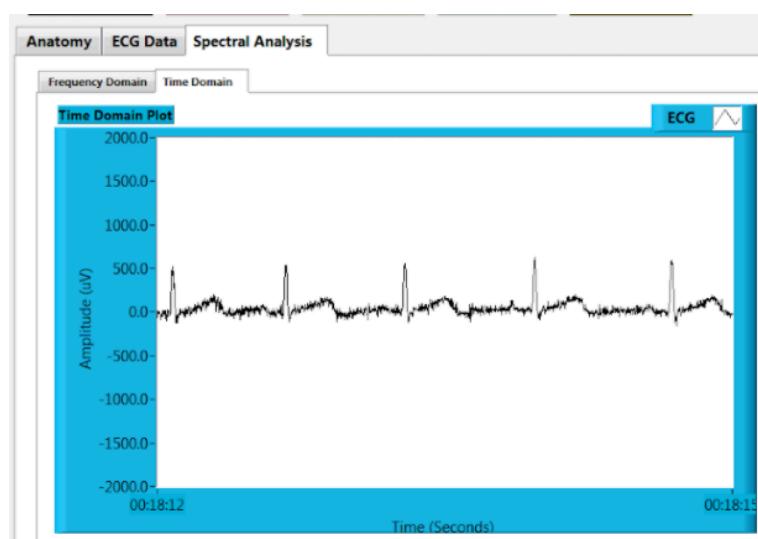


Figure 9.2.4. Spectral analysis of slowECG with 60 Hz band stop filter in the time domain.

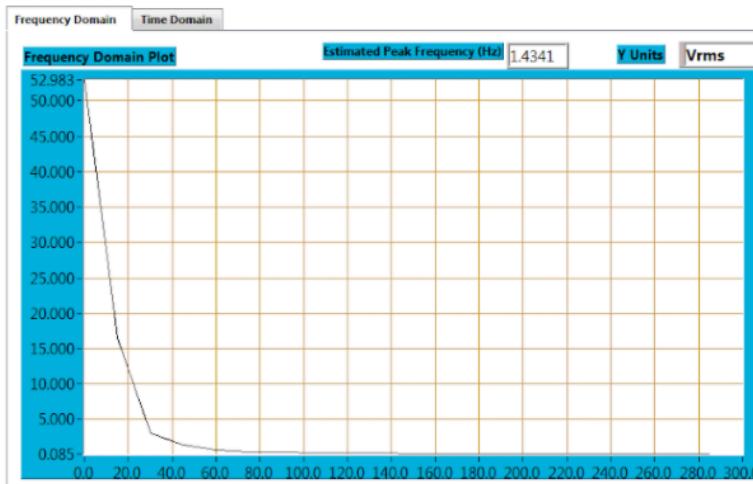


Figure 9.2.5. Spectral analysis of slowECG with 20 Hz low-pass filter in the frequency domain.



Figure 9.2.6. Spectral analysis of slowECG with 20 Hz low-pass filter in the time domain.

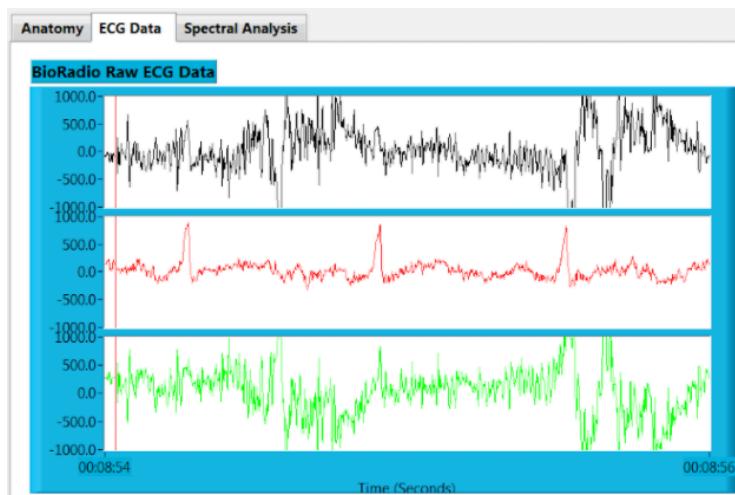


Figure 9.3. fastECG plots

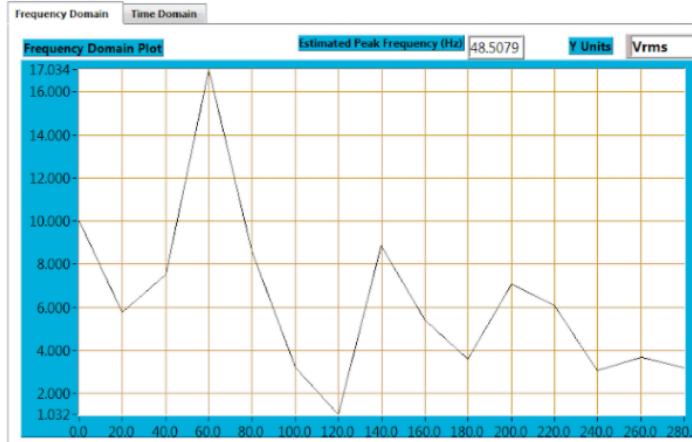


Figure 9.3.1. Raw spectral analysis of fastECG in the frequency domain.

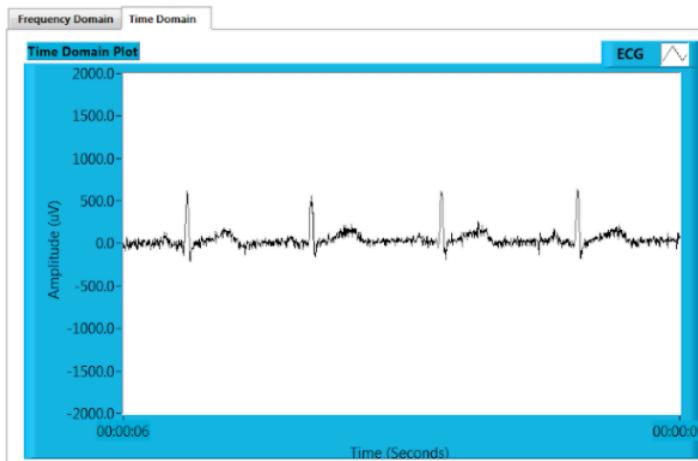


Figure 9.3.2. Raw spectral analysis of fastECG in the time domain.

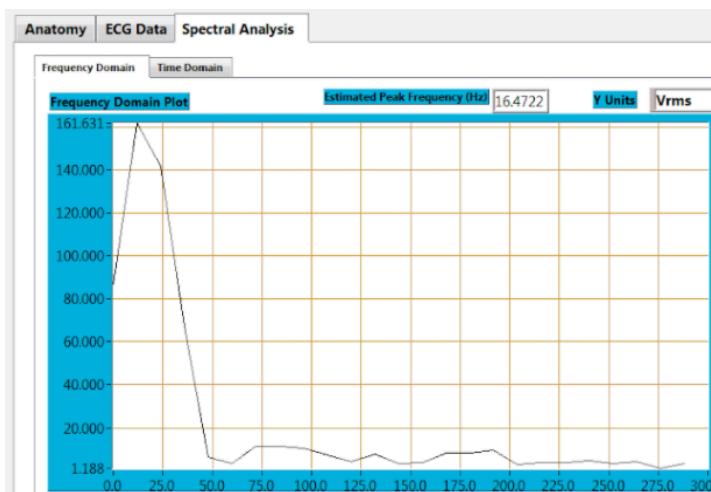


Figure 9.3.3. Spectral analysis of fastECG with 60 Hz band stop filter in the frequency domain.

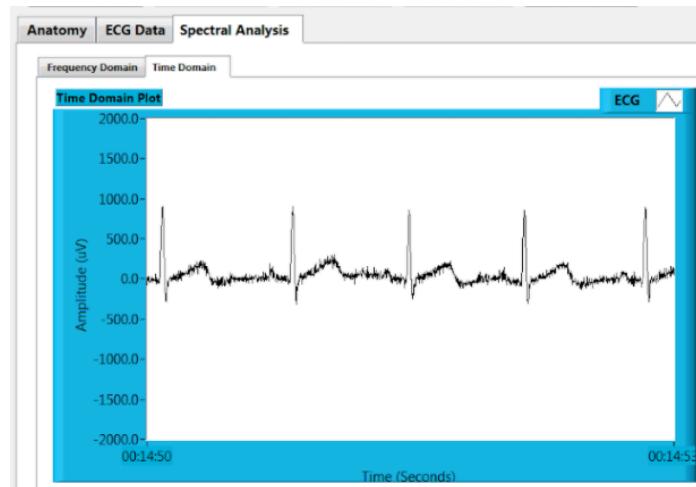


Figure 9.2.4. Spectral analysis of fastECG with 60 Hz band stop filter in the time domain.

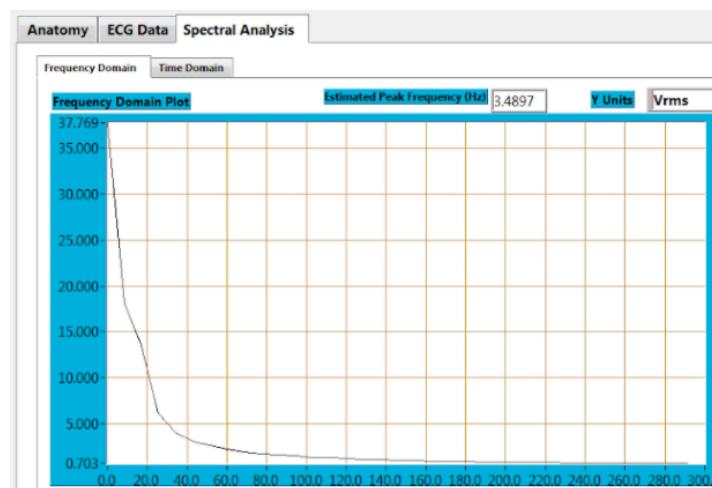


Figure 9.3.5. Spectral analysis of fastECG with 20 Hz low-pass filter in the frequency domain.



Figure 9.3.6. Spectral analysis of fastECG with 20 Hz low-pass filter in the time domain.

Discussion Section

What aspects of the experimental protocol seemed the most likely to introduce errors in the results? To what extent do you think that these issues affected the results that you are reporting, and how could the protocol be improved to address these points?

The placement of the leads and the operations for different breathing exercises have the greatest possibility to introduce errors in the results. Incorrect placement of the lead can cause deviations on the ECG, including changes to the ST-segment. When the team was collecting data for the different breathing exercises, there were inconsistencies during the data collection process that resulted in variation. An example of this error can be seen in *Figure 5.3* showing the ECG during fast breathing. Compared to the base ECG recorded for a normal breathing rate, the recording for a fast breathing rate should have shown a higher R peak frequency (higher RR interval) due to the muscles requiring more oxygen. However, as shown in the results in *Figure 5.1* and *Figure 5.3*, the R peak frequency for normal breathing and fast breathing are almost identical. Any pause or change in breathing rate will cause a minor error in the results.

With regards to the protocol, having an experienced professional checking the placement of the leads will eliminate any misplacement errors. To ensure the frequency of the breathing rate is stable throughout the entire operation, the protocol could include a metronome to guide the exercise. Having the experimenter breathe at the same rate as the metronome will ensure a constant and evenly distributed rate.

How do the heart rate variabilities analyzed from the ECG recordings (using the “IBI” script) relate to the results from Simulink model showing how *hr* varies based on the breathing pattern? Is the simulation reflective of the physiological data? If not, why not? In answering this question, make sure that you are considering what each result is telling you about the changes in heart rate as a function of the respiration rate.

Heart rate is the number of heart beats per minute, whereas heart rate variability is the fluctuation in the time domain between adjacent heartbeats. The R-R interval was determined to convert the ECG data into heart rate variability using the IBI script. Each heart rate variability is the time interval between each heartbeat. By minimizing the standard deviation of each ECG data of different breathing patterns, we obtained an accurate heart rate variability plot. The heart rate changes based on the respiratory cycle. Heart rate increases during inspiration and decreases during expiration. Therefore, when breathing rate decreases, the change in heart rate becomes less frequent. We can observe this from *Figure 6* in the MatLab section, where the plot of heart rate variability vs. heart beat for slow breathing (red line) has a longer phase compared to normal breathing (blue line). The simulation is a direct reflection of the experimental data for slow breathing and baseline model. The result from Simulink Part 1 (See *Figure 2* in the Simulink section) showed that as breathing frequency increases, phase and magnitude decrease.

In 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.

By the convention we have used, lead I has a positive electrode on the left arm and a negative electrode on the right arm, therefore it measures the potential difference between two arms. In the lead II configuration, the positive electrode is on the left leg and the negative electrode is on the right arm. Lead III has the positive electrode on the left leg and the negative electrode on the left arm.

By analyzing the data from ECGartifactright, where the subject waives their right hand around in space, we can observe that an ECG signal can be detected in the graph of lead II while ECG signal appears to be distorted in the graphs of lead I and lead III. A wave of depolarization traveling toward the right arm produces a negative deflection in both leads I and III because the negative electrode for both leads is on the right arm. Therefore, with the movement of the right arm, the signals mainly from lead I and III become distorted, whereas ECG signal from lead II remains detectable.

When we analyze the data from ECGartifactleft, where the subject waives their left hand around in space, we can observe that an ECG signal can be detected in the graph of lead II while ECG signal appears to be distorted in the graphs of lead I and lead III. A depolarization wave heading toward the left arm gives a positive deflection in lead I because the positive electrode is on the left arm. Maximal positive ECG deflection occurs in lead I when a wave of depolarization travels parallel to the axis between the right and left arms. A wave of negative depolarization traveling toward the left arm produces a deflection in lead III because the negative electrode for lead III is on the left arm. Therefore, with the movement of the left arm, ECG signals from lead I and III become distorted as the ECG signal from lead II remains detectable.

ECG artifact right

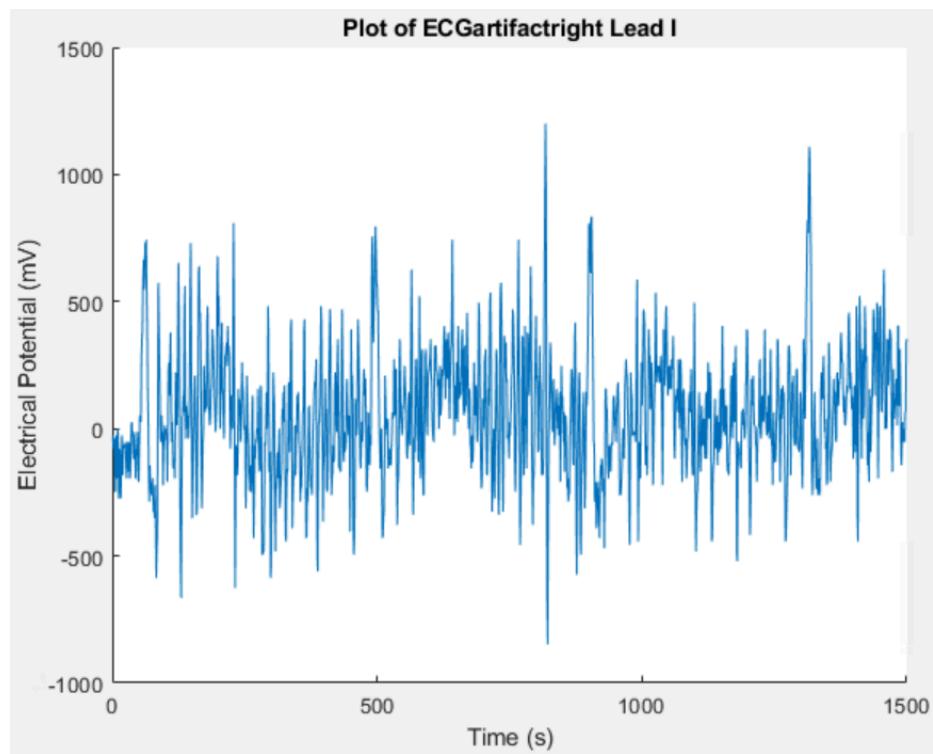


Figure 10.1.1 Lead I for ECGArtifact right

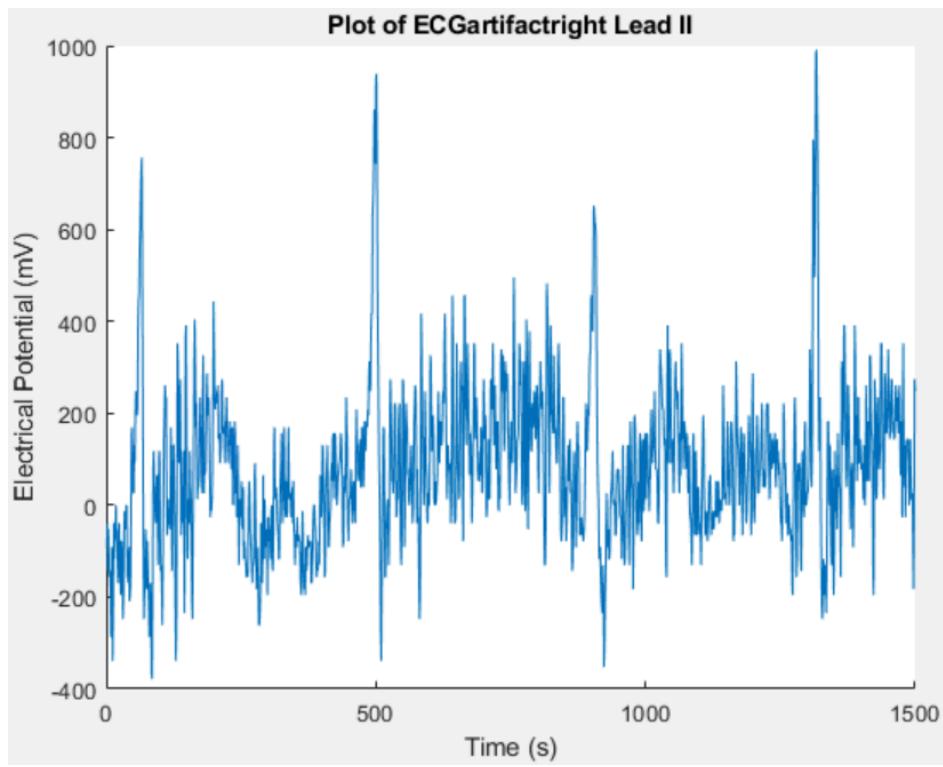


Figure 10.1.2 Lead II for ECGArtifact right

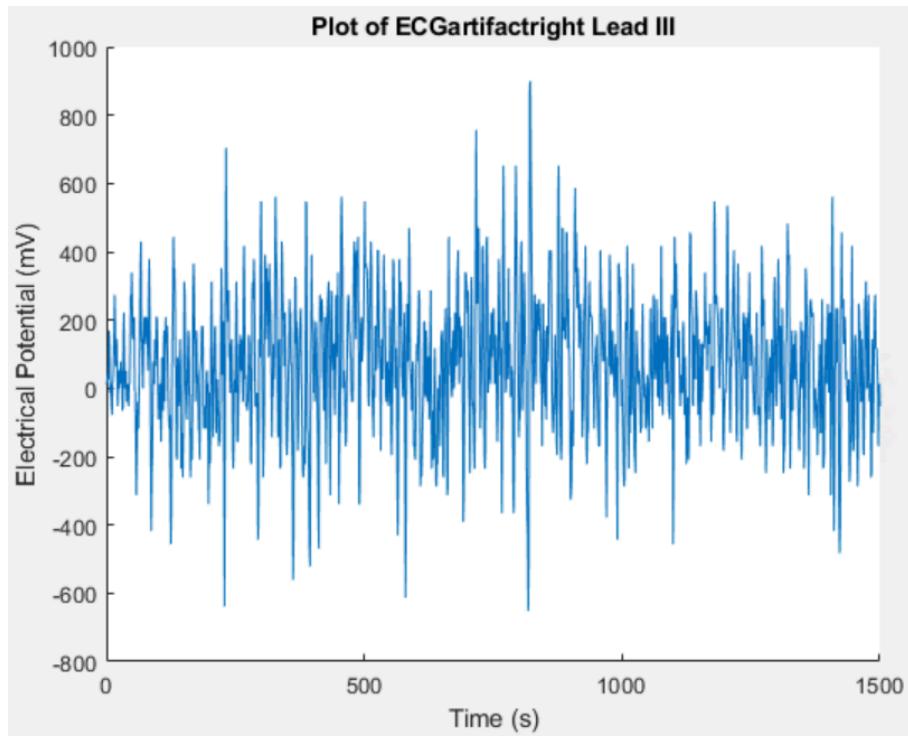


Figure 10.1.3 Lead III for ECGartifact right

ECGartifactleft

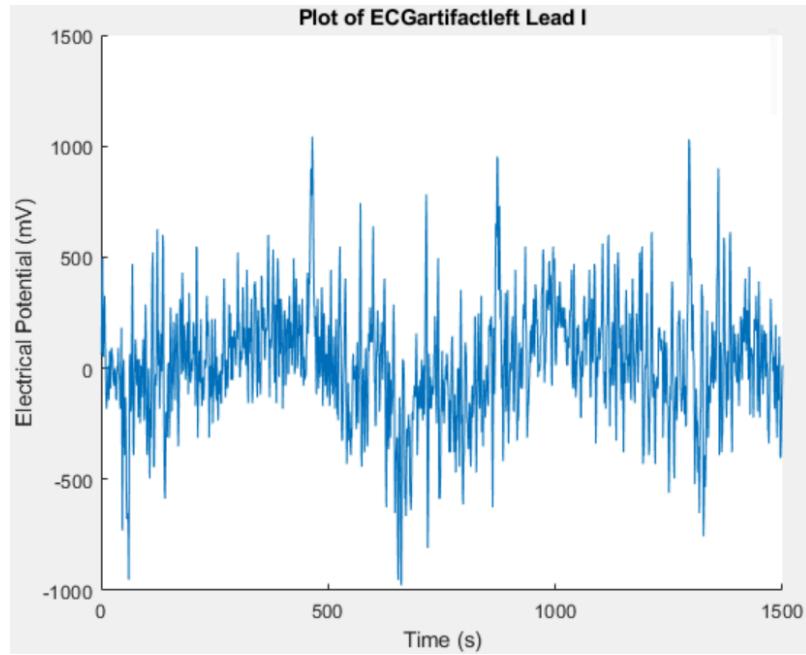


Figure 10.2.1 Lead I for ECGartifact left

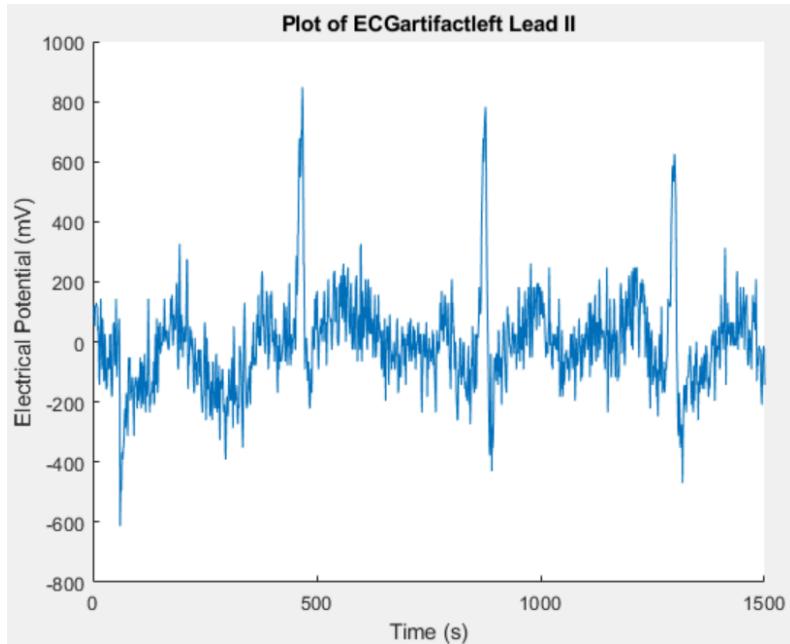


Figure 10.2.2 Lead II for ECGArtifact left

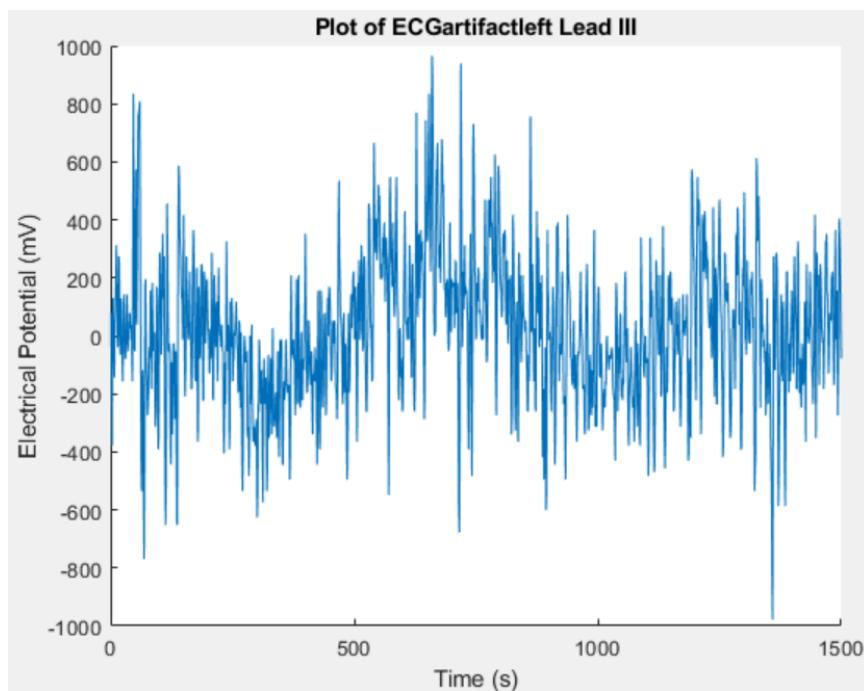


Figure 10.2.3 Lead III for ECGArtifact left

Why was a white noise input used in the Simulink model to conduct the frequency response analysis? What is the advantage of using the chirp block, as in the original model?

Band-limited white noise blocks generate normally distributed random numbers. It is a process with correlation time of 0 and constant power spectral density (PSD). Frequency response analysis in simulink

requires sinusoidal input. With a constant power spectral density, white noise input can be used to conduct the frequency response analysis. Chirp block is one of the most common sinusoidal inputs, the block generates swept-frequency cosine waves with linearly increasing frequency and amplitude. Having a simple input, like a chirp block, in the original model allows us to see how heart rate and abp changes with increasing frequency in breathing. We can observe the overall response and changing trend of the output variables by only checking the scope (output value vs. time) instead of using Matlab script to plot the magnitude and phase corresponding with different frequencies.

Consider the effects of the 60 Hz bandstop and 20 Hz lowpass filters that you used. What are the advantages or disadvantages of each of these two approaches?

The frequency of industrial American power supply is 60 Hz, which creates a noise in biomedical signals. To filter out this noise we first used a FIR filter with 60 Hz bandstop, which passes all frequencies with the exception of those within specified stop band which are greatly attenuated. An advantage of this filter is that we can get linear phase characteristics, therefore the filtered signal isn't distorted because of different spectra frequency delay. As a result our ECG becomes smoother. The disadvantage of this filter is that applying a 60 Hz bandstop filter might also remove ECG signals within the 60 Hz region. Therefore we cannot rely solely on the filtered signal when analyzing an ECG, as signals which are slightly under the stop band would be suppressed by this filter.

Low-pass filters pass signals with a frequency lower than a selected cutoff frequency and attenuates signals with frequencies higher than the cutoff frequency. The advantage of using a 20 Hz lowpass filter is that it smooths the ECG waveform and enhances its clarity. Setting the low-pass filter of the ECG signal to a low cutoff level eliminates the muscle EMG noise as well as clinically significant high-frequency components of the signals inside the QRS complex, creating a disadvantage.

Based on your simulations of the sympathetic and parasympathetic blockades, which blockade has the greatest effect on the phase of *hr*? Why? Refer to the structure of the model in your answer.
[Note: when comparing the phase results, focus on frequencies below approximately 0.5 Hz. Any "sawtooth" shape that you may observe in the plots at higher frequencies is simply an artifact due to phase wrapping. That is, imagine moving clockwise around a unit circle: when the phase reaches -180°, it will "jump" to +180°.]

Parasympathetic blockade of Atropine (See Figure 11.1), has a 360 degree phase drop within 0.45 Hz. The sympathetic blockade of Propranolol (Figure 11.2), has a 90 degree phase drop within 0.5 Hz. Therefore we can conclude that the parasympathetic blockade has the greatest effect on the phase of heart rate.

When simulating the parasympathetic blockade, we modified drive-vagal activity gain (Ap), negative gain associated with a decrease in vagal activity (Kp) and low-pass cutoff associated with a decrease in vagal activity (fp) to be lower than the sympathetic blockade case. By doing so, we suppressed the vagal activity effect on the heart rate. We also modified the drive-sympathetic activity gain (As) and gain associated with an increase in sympathetic activity (Kp) to be higher than the sympathetic blockade. These changes resulted in a drastic increase of the heart rate in the given time scale, as we did not modify

frequency associated with the increase in sympathetic activity (fs). This rapid increase in sympathetic activity, results in an increased heart rate on a faster time scale, resulting in drastic changes in the heart rate phase plot.

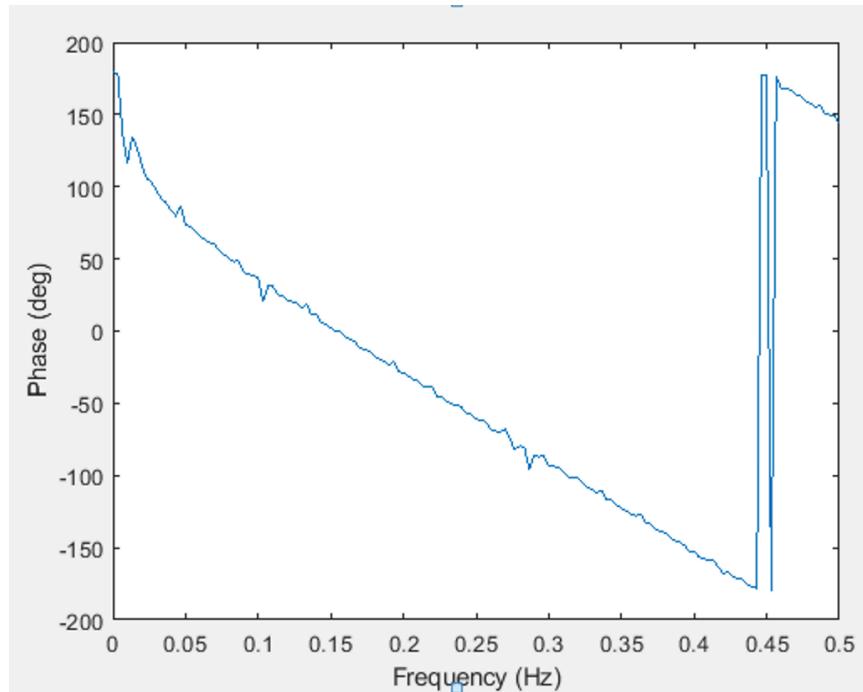


Figure 11.1. Phase of hr for the parasympathetic blockade Atropine

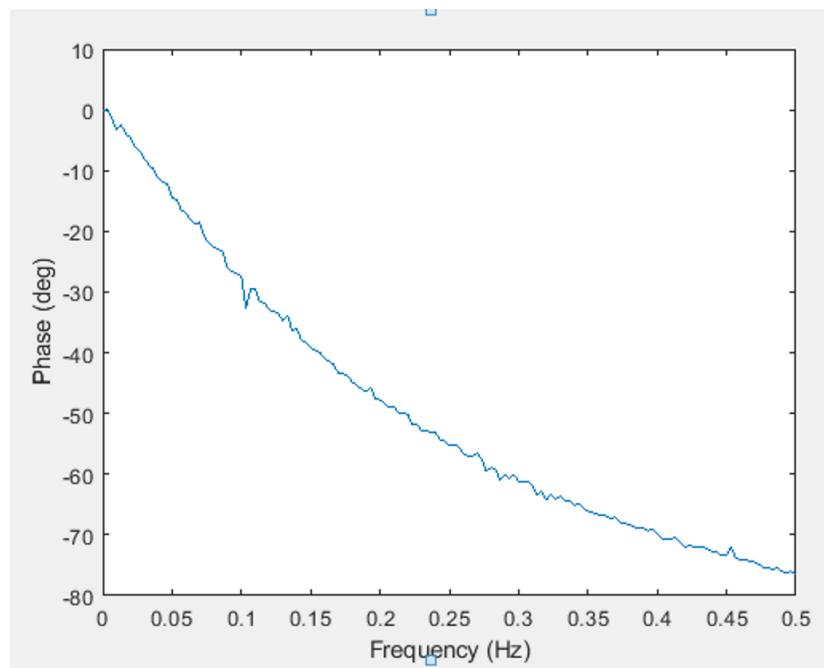


Figure 11.2. Phase of hr for the sympathetic blockade Propranolol