1 Introduction

This lab exercise has two parts. In the first part we will measure blood pressure and heart rate using a sphygmomanometer and stethoscope. The measurements can be made before and after physical work to observe how heart rate and blood pressure change with exertion.

In the second part of the lab, we will use the Vernier computer data acquisition system to record electrocardiograms (ECGs or EKGs). The interval between ECGs is also a measure of heart rate, and we can compare this result for heart rate with that obtained using the manual stethoscope method, with the following caveat. There is a practical issue related to “sampling rate”. There are only so many data points that can be stored in the LabPro memory. If you record at high speed to get good resolution on a single ECG, the memory is exceeded before very many multiple ECGs can be logged. You need to average over multiple ECGs to get a proper measure of rate, i.e., the reciprocal of the interval between ECGs. So there is a trade-off between temporal resolution and length of data collection. If you record for many beats, to get good rate data, the number of sample points per unit time must be less. Thus the exact time at which a beat occurs is less well defined. Is the interval between two beats 2.05 seconds or 2.0 seconds or somewhere between 1.5 and 2.5 seconds? Suppose you have a heart rate of 60 beats per minute, or 1/second. If you collect data at 1000 samples per second for two minutes you would be able to get an average rate over 120 interval values and your time precision would be 1/1000th of a second (one millisecond). This would require storing 1000 points per second x 120 seconds or 120,000 time values. This is no problem, but now suppose you want to watch heart rate change over 15 minutes? At some point you will run out of memory for this task.

2 Blood Pressure

Each time the heart beats it forces a volume of blood into the aorta. This periodic entry of blood into the arterial system causes the pressure within the arteries to rise and fall. The peak pressure obtained during each cardiac cycle is called systolic pressure; the lowest pressure reached just before the next heart contraction is called diastolic pressure. These pulsations are transmitted throughout the arterial system.

The strength of the arterial pulse transmitted to the arteries depends upon both the amount of blood injected into the aorta by contraction of the ventricles and the distensibility of the arterial vascular system. Thus, if the arteries are dilated and allow rapid circulation of the blood, diastolic pressure may be low, while systolic pressure remains relatively unchanged.

This produces a wide pulse pressure (difference between systolic and diastolic pressures). When palpated
at the radial artery, such a wide pulse pressure yields
a pounding sensation to the palpitating fingers. On the
other hand, if the arteries are tightly constricted, the
systolic pressure may rise higher than normal; but di-
astolic pressure will also remain higher than normal,
and pulse pressure may even be narrowed. The pulse
in this instance yields a sensation that is short and sud-
den.

Mean arterial pressure (usually considered to be
one-third of pulse pressure plus diastolic pressure)
provides the perfusion pressure head which causes the
flow of blood through the arteries, arterioles, capil-
laries, venules, and veins. Arterial pressure depends
upon the amount of blood forced into the aorta by the
heart (cardiac output) and the resistance to flow of-
fered by the peripheral vessels (total peripheral resis-
tance). Cardiac output liters/minute is determined by
the stroke volume (amount of blood pumped with each
beat of the heart) and the heart rate (number of beats
per minute). Stroke volume depends, to a large extent,
upon the amount of blood available to fill the ventri-
cles during diastole. The force that causes ventricular
filling is the difference between the peripheral venous
pressure and the pressure in the ventricle during dias-
tole. The heart, within a very wide variation, is capa-
ble of pumping out all the blood that flows into it. If
the central venous pressure rises, there is more filling
of the heart, and the heart pumps more blood. Con-
versely, if venous pressure is low, the heart does not
fill as much and less blood is pumped with each stroke.
If you change position from lying to standing, there is
an immediate increase in arterial pressure in the ves-
sels of your feet. This is described as an increase in
the hydrostatic column. The same hydrostatic column
will impede the flow of venous blood from the feet,
and venous pressure must rise in order to force blood
upward to the right atrium of the heart. The action of
muscles in the legs squeezing the arteries and veins, in
addition to pressoreceptor and other reflexes that cause
vasoconstriction, combine to prevent pooling of blood
in the legs and feet when you stand up. Venous return
is restored and the decrease in cardiac output is not no-
ticeable. These mechanisms are absent or weak in per-
sions with postural hypotension. As a result their car-
diac output is decreased sufficiently to jeopardize the
flow of blood to the brain, and they can faint when they
move from a lying to a standing position. After stand-
ing, if one begins to exercise, the increased metabolic
rate of the contracting muscles requires more oxygen
and nutrients. Vasodilation increases the local blood
flow, and the cardiovascular system both increases car-
diac output and decreases the blood flow to areas not
involved in exercise.

2.1 Measurement of blood pressure with the
sphygmomanometer and stethoscope

Read this part carefully before taking any measure-
ments. Note these sphygmomanometer components:
the rubber bag of the pressure cuff (Figure 1), the
bulb by which the bag can be inflated, and the release
d valve for slow deflation. The rubber bag is placed on
the upper arm just above the elbow; the strip of cloth
is wound snugly, but without much pressure, around
the arm and the end tucked under. The bell of the
stethoscope is placed lightly over the bifurcation of
the brachial into the radial and ulnar arteries, approx-
imately at the bend of the elbow. The bell should be
held very close to the armlet but not in contact with it.

Here is how it works: When you pump up the pres-
sure cuff it will constrict the blood vessels of your arm.
As pressure builds you first prevent blood flow during
the low pressure (diastolic) portion of the cardiac cy-
cle. With further pressure increase even the higher sys-
tolic pressure cannot overcome the pressure applied by
the cuff, and no blood flows. So you pump up the cuff
until there is no flow (about 180 mm Hg), then allow the pressure in the cuff to fall slowly by opening the release valve. The pressure gauge on the cuff will glide down as the air pressure declines but as soon as the systolic pressure can get through, the gauge will wiggle for each heart beat. The wiggle represents the difference in pressure between systole and the cuff’s air pressure. Now you are also listening with the stethoscope to the sound of blood flow under the cuff. When blood can get through you will hear a systolic flow impulse: record the pressure at this point (the systolic pressure). As the air pressure bleeds down further, the sound gets louder (more and more blood gets through) until the diastolic pressure also overcomes the cuff pressure. At this point you don’t hear any sound in the stethoscope, because the blood is no longer crashing into cuff-constricted vessels trying to force them open. The pressure at this point is the diastolic pressure, which you will record.

2.1.1 Clinical Protocol

For clinical blood pressure measurement, there is a standard routine used. The idea is to have comparable data across individuals and for repeated measurements on the same individual. First blood pressure is measured with the patient laying down, then sitting, then standing, and finally after exercise. Here is a typical protocol:

1. Have the patient lie on his/her back for two minutes. Ensure that the head and toes are completely level.

2. Place sphygmomanometer two to three finger-widths above the anticubital space.

3. Make sure the cuff is tight enough (do this by gently tugging) if the cuff moves down it is not snug enough.

4. Adjust the height of the arm at the anticubital space to be at the same elevation as the heart (about half way between the horizontal surface the patient is on and the top of their rib cage).

5. Take reading.

6. Move patient to a sitting position and wait two minutes.

7. Allow the arm to hang loose; this will allow the anticubital space to naturally line-up with the heart.

Figure 1: How to make blood pressure measurements.
8. Take reading.

9. Move patient to a standing position and wait two minutes.

The steps above should be repeated for each of the conditions given in section 2.3.

Make a table to record your measurements. See the example below:

<table>
<thead>
<tr>
<th>Conditions</th>
<th>HR (bpm)</th>
<th>BP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### 2.2 The effects of exercise

Exercise involves muscle activity, and the more active any tissue, the greater is its oxygen demand. That in turn requires more blood perfusion per unit time and so an increase in cardiac output. The main control of cardiac output is heart rate, thus an immediate effect of exercise is an increase in heart rate. For athletic individuals there is long-term conditioning such that resting heart rate can be lower, and the increase in rate with exercise is less. Average individuals will have a much greater increase in rate with exercise.

Now that you are experts at measuring blood pressure and heart rate (via pulse rate), see if one or more of your lab partners is willing to do a little exercise to measure the change in their circulatory parameters. A handy vigorous exercise is running stairs, but do be careful. A rapid walk is safer! Measure resting rate and pressure as above, then have the volunteer engage in stair running/walking until the person can sense an increase in breathing and probably sense an increase in heart rate. Now immediately measure blood pressure and heart rate. Do this again after five minutes and continue to measure every five minutes until the resting rate is restored. Note that an important clinical consideration of cardiovascular fitness is the time to recovery. Everyone who exercises will experience an increase in pressure and rate, but very fit individuals will return to baseline levels very quickly.

### 2.3 Investigation of Venous Circulation

In the following tests you will observe changes in venous filling under different circumstances.

1. Hydrostatic pressure. Hold your hand down and note the veins on the back of the hand. Raise the hand high above the head and notice the almost instant collapse of the veins. Why?

2. Muscle exercise and gravity. Notice the prominence of the veins in your feet after you have been standing still for about two minutes. Now take 5
or 6 steps and immediately notice the veins. Explain why the veins are so large before, and empty immediately after, the exercise. Upon what part of the circulatory system has gravity its greatest effect? Why? How do position and activity of the body influence this?

3. Valves and unidirectional flow. Let one member of the group serve as a subject. Apply a fairly tight sphygmomanometer around the arm above the elbow. Note the great filling and distention of the veins of the hand and forearm. Also note the swellings found at intervals on the veins. What are these due to?

With one finger firmly press down upon a distal (part farthest from the body) part of the vein and with another finger press the blood forward beyond the next swelling. Notice that the vein is mechanically emptied. Remove finger 1. From which end did the refilling of the vein start? Why? Is there any evidence of valves?

With a finger moving down over a vein try to force the blood toward the hand. What is the result? How do you explain this?

3 The Mammalian Electrocardiogram

Heart contraction is triggered by excitation of the cardiac pacemaker (SA node). An electrical trigger signal is sent first to the atria and then, after a short delay, to the ventricles.

Purkinje fibers conduct the trigger signal quickly to all regions of the ventricle to ensure synchronous contraction of all the muscle cells in it. Just prior to the contraction of muscle, including cardiac muscle, a muscle action potential is generated. This potential is an electrical reversal of the normal polarization of the muscle cells. Because of the synchronized electrical activity in heart muscle cells, a large voltage or potential difference sums over the entire atrium or ventricle and can be recorded even some distance from the heart. The geometry of chambered hearts is such that these potentials have a complex form (see Figure 3). The electrocardiogram, (ECG or EKG) is an electrical record or artifact of muscle contraction detected at a distance from the heart. The signal is weakest on the skin surface because, unlike the internal body tissues which are relatively salty and good conductors of electricity, the skin is a relatively good insulator. Never-
theless, with sensitive electronic instruments and careful placement of electrodes, the heart electrical signal can be recorded. Note that it is possible to record signals this way from any muscle when it contracts, but electrical instruments designed to detect heart muscle contraction are specialized to pick up only the characteristic waveform of the heart-generated signals, and ignore the signals from skeletal muscle activity.

Typically the EKG consists of a series of upward deflections (Figure 3), arbitrarily designated the P, R, and T waves, and two downward deflection, the Q and S waves. The waves occur in alphabetical order. The EKG has a complex interpretation owing to (1) the inhomogeneities of the conduction pathways from the heart to the body surface, (2) the fact that the body is not of infinite size, and (3) the complex origin of the various waves with definite events in the excitation cycle of the heart, as discussed subsequently.

![Figure 3: The EKG](image)

3.1 Determination of the Electrocardiogram in Humans

In taking EKGs, electrodes are applied to the right arm, left arm and left leg. The green electrode is placed on the left arm. The red electrode is placed on the right arm. Finally the black (ground) electrode is placed on the left leg. This arrangement of three electrodes is a type of “differential” recording which greatly minimizes the pick-up of room electrical noise. The electrodes, when arranged this way will give you the most common type ECG output known as Lead II. Note that sometimes to get the ECG shape as you expect it, the green and red electrodes must be switched.

![Figure 4: Here are the optimal locations for electrodes to get an electrocardiogram](image)

**Detailed Electrical Pathway**

The sinoatrial node of the heart initiates the electrical activity which ultimately results in the contraction of the cardiac musculature. This electrical activity spreads over the atria at the rate of about 1 meter/second. The P wave of the EKG is due to this spread; its duration is roughly 80 ms.

The excitation wave reaches the atrioventricular (AV) node just before its arrival at the interventricular septum. There is a delay of approximately 100 ms at the AV node. After this delay, excitation spreads rapidly (1.5 to .4 m/sec) down the Purkinje fibers of the common bundle and the bundle branches. Then excitation spreads over the Purkinje fiber network on the endocardial surface. Finally the excitation travels from the endocardial to the epicardial surface at the rate of about 0.4 to 0.5 m/sec. The P-R interval represents the time between the start of the spread of excitation though the atria and the beginning of its spread.
through the ventricles. It is measured from the begin-
ing of the P wave to the onset of the QRS com-
plex (see below). The Q, R, and S waves are due to
the spread of the excitation wave through the ventricu-
lar musculature. These deflections comprise the QRS
complex. Frequently these complexes occur with one
or another of the three waves absent. The term QRS
complex is applied despite this. By definition, if the
first rapid deflection is below the base line, it is called
a Q wave. The first upward deflection is the R wave.
The first downward deflection immediately following
the R wave is called the S wave. The QRS complex
has a duration of about 80 ms.

The T wave is due to repolarization of the ventric-
ular musculature. Whereas the QRS complex, repre-
senting depolarization of the ventricles is a sharp, sud-
den set of deflection, the T wave is of longer duration
and lower amplitude. This has been interpreted as in-
dicating that the ventricular repolarization process is a
less integrated event than that concerned with depolar-
ization. The duration of the Q-T interval, representing
the time from the beginning of ventricular depolariza-
tion to the end of ventricular depolarization is strongly
dependent on heart rate. An average value is 360 ms.

3.1.1 Experimental Procedure

You will collect EKG data from a subject both before
and after exercise. Note if the subject was exercising
for the blood pressure activity be sure to allow at min-
imum 15 minutes for their heart rate to return to base-
line.

1. Have the subject apply the electrode stickers as
shown in figure 4. These stickers should not need
to be moved, you can change leads by clipping
the electrode clips onto different stickers. Con-
nect green (negative clip) to the sticker labeled 1,
the black clip should be connected to the sticker
labeled 2 and the red clip to sticker 3 (Fig. 4).

2. Once your subject is all connected have them
sit or stand calmly without touching their hands.
Launch logger-pro and hit the green collect but-
ton. You should see a EKG trace appear. If you
don't check all your connections make sure your
subject is in a relaxed position and try again. If it
still doesn't work ask your TA for help.

3. Once you have a good EKG trace your subject
may disconnect the clips, but keep the stickers in
place. Zoom in on one of the heart beats and iden-
tify the P wave the QRS complex and the T wave.
Also identify the PR interval and the QT interval.
Table 5 is helpful for organizing your data.

4. Zoom out and measure and record the time re-
quired for 3 complete beats. Also, measure and
record the duration of the PR and QT intervals on
three different heart beats.

5. Once you have collected all of your data have
your subject go into the stairwell and do enough
exercise to become winded. Running down to the
first floor and back up should do it for most peo-
ples. When they return re-attach the electrodes and
collect another EKG. Heavy breathing may cause
intercostal muscles and the diaphragm to dis-
rupt the EKG so have the subject try and breathe
calmly.

3.2 Additional Experiments

Once your subject has had a moment to recover from
exercise, decide as a group to investigate one or more
of the variables listed below by analyzing the resultant EKG. Before starting a test you should design a mini experiment complete with hypothesis and then test your prediction.

- Breath holding
- Posture sitting, standing, lying down
- Submerging the subjects face in cold water
- Strength training exercises rather than cardiovascular exercises
- Other EKG leads (I and II)

You will want to include a figure showing your EKG in your lab report. The correct way to do this is to copy the data from the Vernier X,Y columns (time points and voltage values) and paste the numbers into Excel. Then you should have Excel make a scatter plot, and adjust the plot parameters until you get a waveform that looks like the EKG plotted by the Vernier software. The wrong way to produce a figure for your lab report is to cut and past the Vernier graphic into a MS Word document!

Here are two items to consider in your lab report:

- What change in blood pressure was observed between lying down and standing? What is the reason for this?
- Did PR and QT intervals get shorter at higher heart rates? If not how does heart rate increase?

**Discussion Questions**

1. Why does the needle of the sphygmomanometer twitch only when the device is between the subjects systolic and diastolic blood pressure?

2. Based on what you know about the cardiac cycle what is the maximum possible heart rate? Why?

3. Why is blood flow pulsatile in some vessels and smooth in others?
<table>
<thead>
<tr>
<th></th>
<th>PR interval (ms)</th>
<th>QT interval (ms)</th>
<th>Time for 3 beats (s)</th>
<th>Heart rate in BPM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Before Activity</strong></td>
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<tr>
<td>Beat 1</td>
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<tr>
<td>Beat 2</td>
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</tr>
<tr>
<td>Beat 3</td>
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<tr>
<td>Mean ± SD</td>
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<tr>
<td><strong>Post Activity</strong></td>
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<tr>
<td>Beat 1</td>
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<td>Beat 2</td>
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<td>Beat 3</td>
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<tr>
<td>Mean ± SD</td>
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</tbody>
</table>

Figure 5: A good way to record the parameters you obtain from your EKG traces.