

Lab #10: Cardiovascular Physiology

Background

The heart serves as a pump to drive the flow of blood through the body. It does so by undergoing a cycle of contraction and relaxation called the *cardiac cycle*. During the initial portion of the cardiac cycle, an electrical signal is generated in so-called “pacemaker cells” that is distributed through the heart through an electrical conduction system. In response to electrical stimulation, the myocardium of first the atria and then the ventricles undergoes contraction (*systole*), followed by sequential relaxation (*diastole*) of the two sets of chambers a fraction of a second later. This cycle of compressing on the blood in the ventricles during systole followed by the filling of the ventricles during diastole induces pressure changes in the ventricles that cause one-way valves in the heart to close audibly at different intervals of the cardiac cycle. The result of the injection of blood into the arteries by the ventricles undergoing systole is the generation of blood pressure, the primary driving force for the flow of blood through the body. In this exercise we will examine both electrical and mechanical events that take place during the cardiac cycle as well as measure the resultant blood pressure generated through this contractile activity.

Electrical stimulation of the heart and electrocardiograms.

The heart is auto-excitatory. Action potentials are formed spontaneously at regular intervals in specialized cells called *pacemaker cells*. These cells are arranged in a network that enables signals to be conducted throughout the myocardium from the point of origin. Four major structures are found within the conduction network (Fig 10.1). The *sinoatrial node* (SA node), which is located in the right atrial wall near the junction for the superior vena cava, contains pacemaker cells that undergo spontaneous depolarizations at a higher rate than any of the other pacemaker cells in the heart. As a result, the SA node sets the basic tempo for

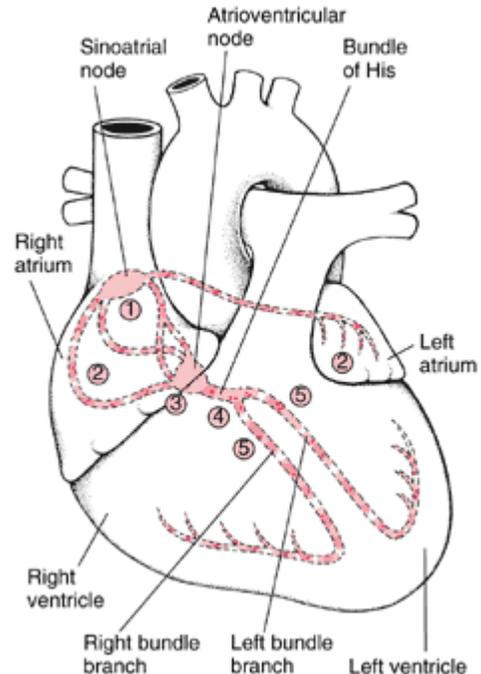


Fig 10.1. The conduction system of the heart and the path of cardiac excitation. Typically, action potentials originate in the sinoatrial node (1) and are conducted rapidly through the atria (2) to the atrioventricular node (3). Once having passed through the AV node, the action potential propagates through the Bundle of His (4) and the lateral branches that arise from it (5). Once the signal has reached the apex of the heart, Purkinje fibers distribute the depolarization to the ventricular myocardium. Image from <http://www.merck.com/mmhe/>

heart contraction (the *sinus rhythm*), and thus is often referred to as the pacemaker of the heart. Action potentials originating in the SA node are conducted rapidly through both atria through tracts of pacemaker cells. Located in the medial wall of the right atrium, near its junction with the right ventricle, is the *atrioventricular node* (AV node). The AV node contains the only pacemaker cells that lead out of the ventricles, thus normally electrical signals originating in the SA node and passing through the atria can only be conducted to the ventricles through this structure. The pacemaker cells in the AV node have very low conduction velocities, thus electrical signals pass through this region very slowly. Once the signal passes through the AV node, it is transferred to a structure called the

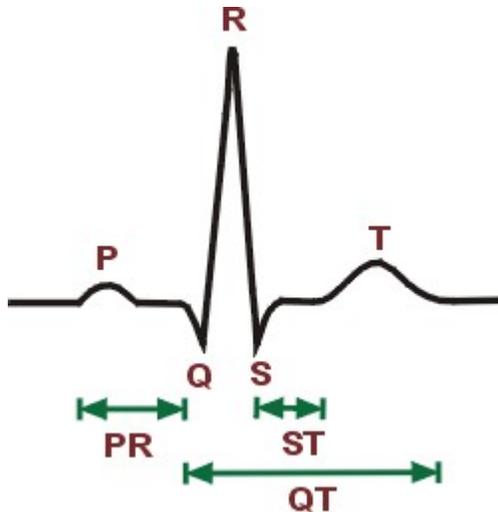


Fig 10.2 An electrocardiogram for a single cardiac cycle. Note the three distinctive waveforms: the P wave, QRS wave, and the T wave. Three important diagnostic intervals (P-R, R-T, and S-T) are also noted.

atrioventricular bundle (AV bundle) or *Bundle of His*, which conducts the signal through the interventricular septum towards the apex of the heart. Soon after entering the interventricular septum the AV bundle bifurcates into two separate branches. The conduction of the electrical signal through the interventricular septum, coupled with the slow conduction velocity of the AV node, causes a delay from when action potentials form in atrial myocardium and when they form in the ventricular myocardium (and subsequently in when the two sets of chambers contract) called the *atrioventricular delay*. This delay ensures that atrial systole is complete at the onset of ventricular systole. Once the signal reaches the apex of the heart it is conducted up the lateral walls of the ventricle through branched tracts of pacemaker cells called *Purkinje fibers*, which distribute the electrical signal to the ventricular myocardium.

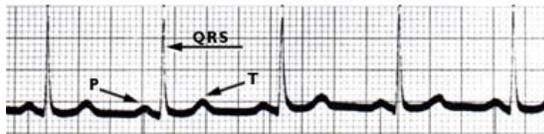
Electrical changes occurring during the cardiac cycle can be monitored from the surface of the body in a recording called an *electrocardiogram* (ECG, or EKG). A normal ECG recording associated with a single cardiac cycle contains three distinctive waveforms (Fig 10.2). The *P wave* is generated when the atria depolarize as the action potential wave spreads out from the sinoatrial node. The *QRS complex* (which consists of the Q, R, and S waves) is

triggered by the depolarization of the ventricles just before ventricular systole. During the QRS wave the atria are repolarizing, but the small electrical disturbance caused by this is masked by the massive change in extracellular charge caused by the ventricles depolarizing. The last waveform, the *T wave*, is triggered by the repolarization of the ventricles at the end of ventricular systole.

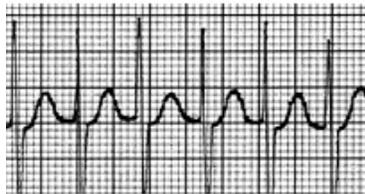
A number of important intervals can be measured from an ECG recording. A simple measure of the duration of the cardiac cycle can be measured simply as the time that elapses between a particular point in one cardiac to that same point in the next cardiac cycle (e.g., from R wave to R wave). The *P-R interval*, (which here we will measure as from the start of the P wave to the peak of the R wave), indicates the duration of time that the atria are depolarized, which is roughly equal to the duration of atrial systole. In addition, the P-R interval indicates how long it takes electrical signals to travel from the atria to the ventricles (i.e., the AV delay). During the *R-T interval* (here measured as the duration from the peak of the R wave to the start of the T wave), the ventricles remain in a depolarized state. The duration of this interval is roughly the duration of ventricular systole, thus the amount of time that blood is being forced out of the heart and into the arteries. Conversely, the *T-R interval* (here measured as the duration between the start of the T wave of one cardiac cycle to the peak of the R wave of the next cycle) indicates how long the ventricles remain in a polarized state between depolarizations, corresponding to the duration of ventricular diastole and thus how long the ventricles refill with blood following contraction. Finally, the *S-T interval* (the segment of baseline between the end of the S wave and the start of the T wave) is an important diagnostic interval, in that this section may become elevated as a result of a recent myocardial infarction (“heart attack”) or depressed in individuals with coronary ischemia.

ECGs are important diagnostic tools for evaluating cardiac abnormalities. Some examples of ECGs associated with particular pathologies are presented in Fig 10.3.

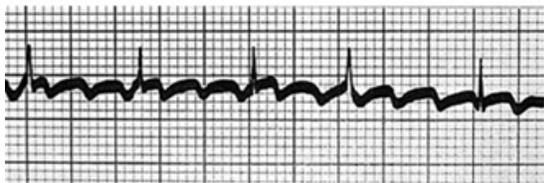
ECG: Normal Heartbeat



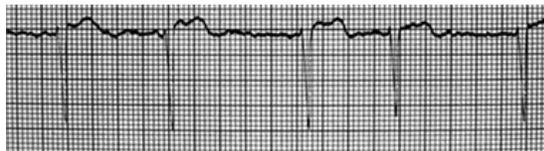
ECG: Paroxysmal Atrial Tachycardia



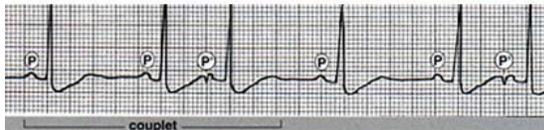
ECG: Atrial Flutter



ECG: Atrial Fibrillation



ECG: Atrial Ectopic Beats



ECG: Heart Block



ECG: Ventricular Tachycardia



ECG: Ventricular Fibrillation

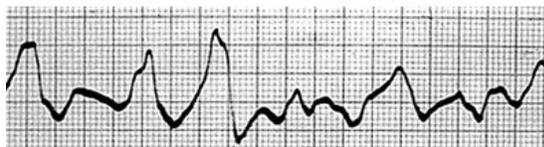
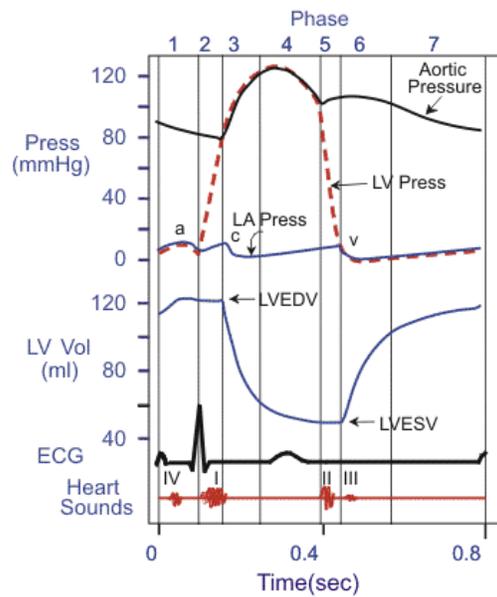


Fig 10.3 Examples of Abnormal ECGs. Images (as well as information on the pathologies that cause these abnormal ECGs) are from <http://www.merck.com/mmhe/>



Abbreviations:

- LV Press, left ventricular pressure
- a, a-wave; c, c-wave; v, v-wave
- ECG, electrocardiogram
- LVEDV, left ventricular end-diastolic volume
- LVESV, left ventricular end-systolic volume

Fig 10.4 Events associated with the cardiac cycle. The top of the figure represents changes in blood pressure in the aorta, left ventricle, and left atrium. The mid portion of the figure displays changes in the volume of the left ventricle. The bottom indicates associated events in an ECG as well as the generation of sounds associated with the cardiac cycle. Ventricular systole consists of phases 2-4 in the cardiac cycle. The two normal sounds generated by the heart are I and II
Illustration from www.cvphysiology.com/Heart%20Disease/HD002.htm

The Cardiac Cycle and Heart Sounds

The electrical signals recorded on an ECG are caused by intermittent periods where the myocardium of the heart undergoes action potentials. These action potentials trigger the myocardium of the ventricles to contract for a period of time and then relax. The resultant cycle of contraction and relaxation of the heart is called the cardiac cycle. During the contraction phase of the cardiac cycle (*systole*), the walls of the ventricles contract on the blood within these chambers. This elevates the pressure of this blood above that of the blood in the arteries, thus forcing blood out of the ventricles and into the

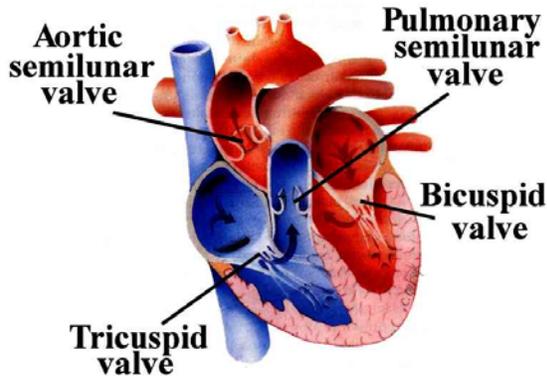


Fig 10.5. One-way valves of the heart. Image from <http://www.starsandseas.com/SAS%20Physiology/Cardiovascular/Cardiovascular.htm>

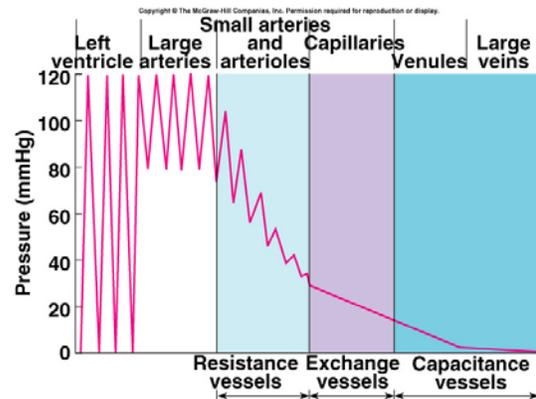


Fig 10.6. Changes in blood pressure in different regions of the systemic circuit. From Fox, S.I. Human Physiology, 8th ed. McGraw Hill.

arteries (Fig 10.4). During the relaxation phase (diastole), the blood pressure in the ventricles falls below venous pressure. Thus blood drawn from the veins fills the ventricles, and the volume of the ventricles expands.

A series of one-way valves prevents backflow of blood from the ventricles into the atria during systole and from the arteries into the ventricles during diastole (Fig 10.5). The closure of these valves can be heard during the cardiac cycle (Fig 10.4). The first sound produced, the “lub” sound, is caused by the closure of the atrioventricular valves at the beginning of ventricular systole when pressure in the ventricles exceeds atrial pressure. The second sound, the “dub” sound, is generated at the beginning of ventricular diastole when ventricular pressure falls below arterial blood pressure.

The Cardiac Cycle and Arterial Blood Pressure

The flow of blood through the cardiovascular system is driven by pressure differences between one segment of a blood vessel circuit and the next. Blood pressure drops sequentially throughout the circuit (Fig 10.6), and thus the blood at one point will flow to the next where the pressure is lower. The contractions of the heart elevate blood pressure high enough so that it can be propelled through the entire circuit.

Arteries have particularly important roles in ensuring adequate blood flow through the cardiovascular system. Arteries serve as pressure reservoirs—their elastic walls expand during ventricular systole to accommodate the influx of fresh blood, and then compress back on the blood during ventricular diastole, maintaining relatively high blood pressure even when ventricular blood pressure has dropped to near 0 (Fig 10.6). This ensures that blood flows constantly through the cardiovascular system throughout the cardiac cycle.

Blood pressure in the arteries oscillates during the cardiac cycle. *Systolic blood pressure* (i.e., the pressure in the arteries during ventricular systole) is ~120 mmHg, similar to that of blood in the ventricles during this period. *Diastolic blood pressure* (pressure in the arteries during ventricular diastole) is somewhat lower at ~80 mmHg, although not nearly as low as the pressure in the ventricles at this time. The difference in pressure between systole and diastole is called the *pulse pressure*, which is a useful diagnostic measure for cardiovascular health. Another derived measurement is the *mean arterial pressure*, which is the average blood pressure in the arteries throughout the cardiac cycle. Mean arterial pressure is calculated as follows:

$$\text{MAP (mmHg)} = \text{Diastolic pr} + 1/3 (\text{Pulse pr})$$

Mean arterial pressure is an important diagnostic measurement in identifying chronic hypertension.

Blood pressure can change based on activity levels and on body position. For example, when a person is standing, blood will tend to be drawn into the extremities (particularly the legs) with the force of gravity. Thus the heart will need to pump harder in order to recover blood and to deliver blood to the brain against the force of gravity, thus blood pressure will become elevated. In contrast, if a person is reclining, blood tends to pool in the abdomen and thorax, and the effects of gravity become less, thus the heart does not need to pump blood as rigorously to ensure adequate circulation, thus blood pressure will tend to become lower.

Cardiovascular Fitness

Blood flow through the cardiovascular system is adjusted in order to meet the demands of the tissues. During high levels of activity, *cardiac output* (the rate that blood is pumped into circulation by the heart) becomes elevated. This

is typically due to an increase in both components of cardiac output: *heart rate* (how frequently the heart beats) and *stroke volume* (how much blood is ejected from the ventricles with each beat). Although both normally elevate during exercise, the relative contributions of each can differ substantially based on cardiovascular fitness. If an individual exercises regularly, they tend to increase the number of myofibrils in their cardiac muscle cells, and thus the ventricles can contract more forcefully during systole and increasing the stroke volume. As a result, heart rate does not need to increase as much during exercise to generate the same degree of cardiac output. This enables individuals who exercise regularly to sustain the same level of exercise for longer periods, to recover more quickly from exercise, and to be able to compensate for changes in circulation (e.g., positional changes) more effectively.

Experiment I. Electrocardiogram.

You will be recording two different ECG tracings. First, record the ECG of the subject in your group per the instructions below. Then unsnap the cable leads from the adhesive electrodes. Have the subject exercise (run around in the hallway or outside, up / down staircases, do jumping jacks, etc.) to get their heart rate elevated. Then quickly reconnect the cable leads to the electrodes and record a second tracing.

For the Subject

1. Removal any metal jewelry from wrists and left ankle.
2. Apply an adhesive disk electrode to the skin on the inside of each wrist and to the left ankle.
3. Attach the ECG electrode cables to the electrode disks in the following manner:
 - White (-) – left arm
 - Red (+)– right arm
 - Black (ground) – left ankle

The disks snap on to the cables

4. Attach the clip on the ECG cable to a belt loop of the edge of your pocket to help keep the cables from moving.
5. Attach the pulse plethysmograph transducer (Fig 10.7) to the tip of your index or middle finger so that the white top of the electrode is in contact with the pad of the finger. It should be secured firmly (but not so hard that it cuts off circulation) with the Velcro strap.
6. Sit down on a stool and rest your arms on top of your legs. Be sure that the ECG cables are slack, and that you are relaxed. Sit quietly during the recording, and avoid moving, talking, etc.— electrical signals from your skeletal muscles will distort the recording.



Fig 10.7. Pulse plethysmograph transducer.

For the Operator

1. Once the subject is ready, left-click on START in the upper right corner of the screen.
2. You should see the ECG being traced on the upper part of the screen, and the pressure pulse of the finger being recorded on the lower part of the screen (See Figure 7A). *If you do not see a distinctive ECG pattern or a regular pulse pattern, ask the instructor for help.*
3. Record for ~15-20 seconds, then left-click on STOP in the upper right corner of the screen.

Data Analysis

Click on the two marker button  (the second to last button in the top row of buttons in your window) to bring up the measuring tools. The two measuring markers (blue vertical lines) will appear over your tracings. You can move the position of either blue line by moving the arrow over a line, holding down on the left mouse button, and dragging the line. Notice that as you move the lines a couple of sets of numbers change:

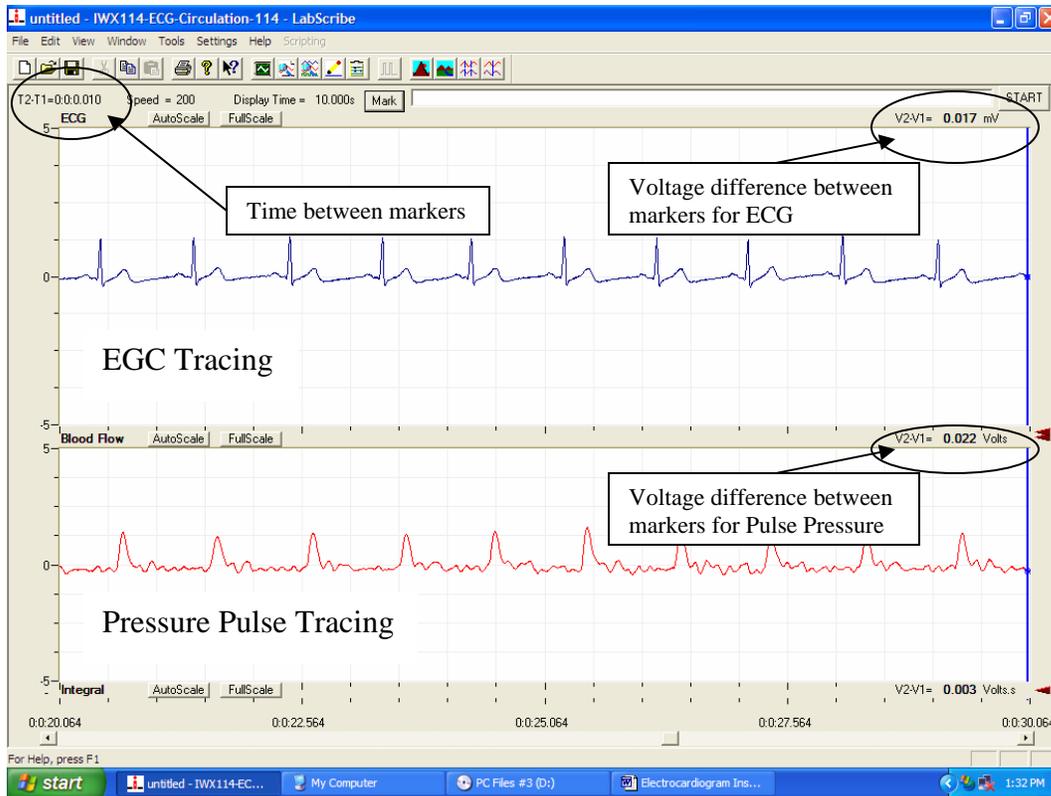


Fig. 10.8 ECG tracing with pressure pulse recording.

- **T2-T1** (top left corner of the window, Fig 10.8). This number is the difference in time measured between the two blue lines on the recording. The number is expressed as HOURS:MINUTES:SECONDS, with seconds given in fractions down to 0.001 sec (or 1 msec)
- **V2 - V1** (top right hand corner of each tracing, Fig 10.8). This number is the difference in voltage recorded between the positions demarcated by the two blue lines. You can use these to measure changes in signal strength to make precision estimates of when particular events occur (e.g., when the R wave reaches its peak, etc.)

Using the blue lines and their associated values, determine the following:

1. **Duration of a single cardiac cycle.** Measure from the peak of one R wave of the QRS complex to the peak of the next R wave (See Fig. 10.9). Do this for 4-5 intervals, and note any differences that may occur in the duration of these ECG cycles.
 - Using this value, calculate the **heart rate (bpm)** by dividing 60 by the cardiac cycle duration.



Fig. 10.9. Position of reference lines for determination of cardiac cycle duration.

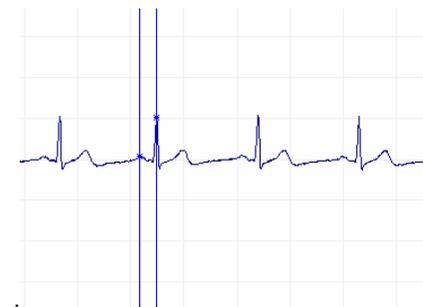


Fig. 10.10. Position of reference lines for determination of the P-R interval

$$\text{Heart Rate (beats/min)} = \frac{60 \text{ sec/min}}{\text{cardiac cycle (sec)}}$$

2. **Duration of the P-R interval.** Measure from the peak of the P wave to the peak of the R wave in the QRS complex (See Fig. 10.10). This is an indication of the time it takes for an electrical signal to travel from the SA node to the ventricles.
3. **Duration of the R-T interval.** Measure from the peak of the R wave to the peak of the T wave in the same ECG cycle (See Fig. 10.11). This is a measure of how long the ventricles are depolarized when they undergo an action potential, and is roughly the same duration as *ventricular systole*.
4. **Duration of the T-R interval.** Measure from the peak of the T wave of one cardiac cycle to the peak of the R wave in the same ECG cycle (See Fig. 10.12). This is a measure of how long the ventricles remain repolarized after undergoing an action potential, and is roughly the same duration as *ventricular diastole*.
5. **Duration of a single heart beat.** Measure the amount of time that elapses between one heartbeat on the pulse pressure recording and the next (See Fig. 10.13). You can use either the start of the heartbeat or the peak or the heartbeat as your reference point. Compare this value with that of the duration of a single cardiac cycle.
6. **Blood conduction time.** Measure the duration between the R wave of a cardiac cycle and the peak of the corresponding pressure wave in the pulse recording (See Fig. 10.14). This is roughly how long it takes blood to travel from your heart to the tip of your finger through arteries.
 - If you assume that the distance from your heart to your finger is ~1 meter, calculate the velocity that blood is traveling by dividing 1m by the blood conduction time.

$$\text{Blood conduction velocity (m/s)} = \frac{1 \text{ m}}{\text{conduction time (sec)}}$$



Fig. 10.11. Position of reference lines for determination of the R-T interval.

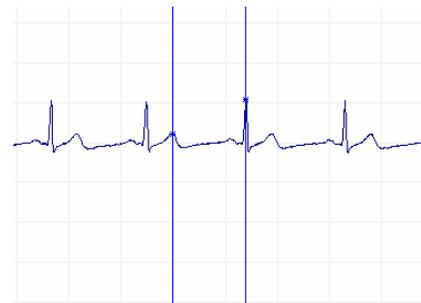


Fig. 10.12. Position of reference lines for determination of the T-R interval.

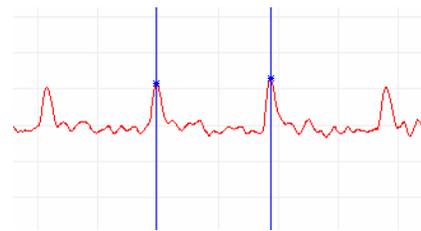


Fig. 10.13. Position of reference lines for determination of heartbeat duration based on pressure pulse.

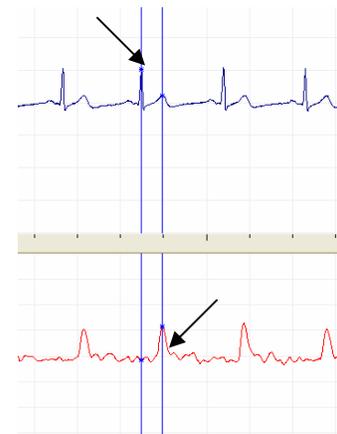


Fig. 10.14. Position of reference lines for determination of blood conduction time. Note the first reference line is positioned on the ECG tracing, but the second is on the pulse tracing.

Experiment II. Heart Auscultation and Blood Pressure Measurement

Heart Auscultation

Place the earpieces of the stethoscope into both ears and position the bell of the stethoscope at the various positions indicated in Fig 10.15 to hear the closures of the different valves.

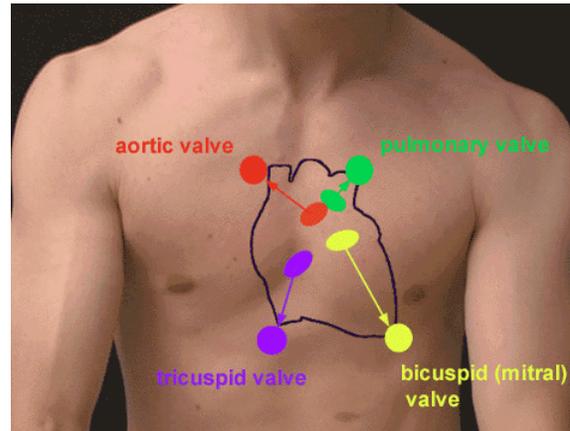


Fig 10.15. Approximate positions where the closure of specific heart valves are best heard. Image is from anatomy.med.umich.edu/surface/thorax/hsounds.html

Arterial Blood Pressure Measurement

- With the subject seated, apply the cuff of the sphygmomanometer around the upper arm of the subject so that the hosing for the cuff is positioned over the cubital fossa (Fig 10.16).
- Apply the bell of the stethoscope to the skin over the brachial artery in the cubital fossa.
- Close the screw valve on the hand pump and pump the cuff to a pressure of ~160 mmHg. Do not exceed 180 mmHg.
- Open the screw valve on the pump to slowly release the pressure, listening to the brachial artery through the stethoscope and noting at what pressure the sounds of Korotkoff (the sounds generated by blood turbulence in a partially occluded artery) begin (systolic pressure) and end (diastolic pressure). Record these values.
- Calculate the pulse pressure for the subject as follows:

$$\text{Pulse Pressure} = \text{Systolic BP} - \text{Diastolic BP}$$

- Calculate the mean arterial pressure for the subject as follows:

$$\text{Mean Arterial Pressure} = \text{Diastolic BP} + 1/3 (\text{Pulse Pressure})$$

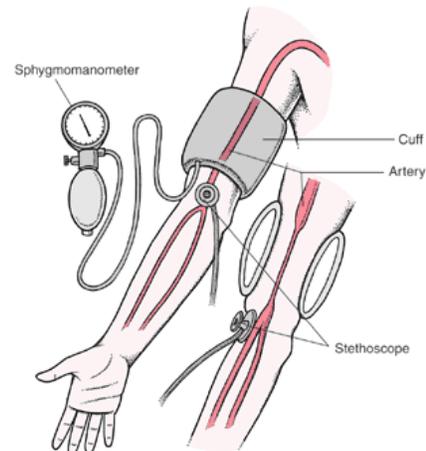


Fig 10.16. Measurement of blood pressure with a sphygmomanometer and stethoscope. Image from <http://www.merck.com/mmhe/>

Experiment III. Cardiovascular Fitness.

In this exercise, we are going to evaluate your cardiovascular fitness using an old but very reliable index called the Schneider index (developed by E.C. Schneider and published in the Journal of the American Medical Association in 1920). The activities in this exercise evaluate the ability of the cardiovascular system to compensate for changes in body position (which alter the effects of gravity on circulation) and changes in activity (a brief amount of exercise).

This is a low intensity exercise. However, if you have any potentially serious cardiovascular conditions (e.g., chronic severe hypertension, heart disease, etc.) that could be aggravated by these activities, please have someone else in your group serve as the subject for this activity.



Fig 10.17. Two techniques for monitoring pulse. Images from <http://www.nlm.nih.gov/medlineplus>

Activity 1. Reclining Heart Rate

1. Have the subject recline on the lab table for a period of 5 min
2. Record reclining heart rate by measuring either radial or carotid pulse (See Fig 10.17) for 30 seconds and multiplying that value by two. Record this value
3. Measure the subject's blood pressure with a sphygmomanometer and record this value.
4. Score points for the individual based upon their reclining heart rate:

Reclining Pulse (bpm)	
Rate	Points
50-60	3
61-70	3
71-80	2
81-90	1
91-100	0
101-110	-1
SCORE	

Activity 2. Standing (Normal) Heart Rate,

1. The subject should stand up and their pulse should be immediately measured for 30 seconds then multiplied by 2.
2. Two minutes after standing up, measure and record the subject's blood pressure.
3. Score points for the individual based upon a) how much systolic blood pressure changed upon standing b) standing pulse rate, and c) how much pulse rate increased upon standing.

Change in Systolic Pr from Reclining to Standing	
Change in Pr	Points
Increase 8+ mmHg	3
Increase 2-7 mmHg	2
No change (± 1 mmHg)	1
Fall of 2-5 mmHg	0
Fall of 6+ mmHg	-1
SCORE	

Standing Pulse (bpm)	
Rate	Points
61-70	3
71-80	3
81-90	2
91-100	1
101-110	1
111-120	0
121-130	0
131-140	-1
SCORE	

Difference in Pulse btw Standing and Reclining					
Reclining Rate (bpm)	Increase in heart rate				
	0-10 bpm	11-18 bpm	19-26 bpm	27-34 bpm	35-43 bpm
50-60	3	3	2	1	0
61-70	3	2	1	0	-1
71-80	3	2	0	-1	-2
81-90	2	1	-1	-2	-3
91-100	1	0	-2	-3	-3
101-110	0	-1	-3	-3	-3
SCORE					

Activity 3: Changes in Heart Rate with Exercise

1. Have the subject step up onto an 18” stool, right foot first, then bring up the left foot and place it next to your right. Step down with the left foot and then bring the right foot down to the floor next to it. Repeat this exercise five times, allowing three seconds total time for each repetition.
2. Immediately after completion of the fifth repetition, measure the subject’s heart rate for 15 second and multiply the number of pulses for 15 seconds. Record this value.
3. Repeat the 15-second pulse measurements at 30, 60, 90, and 120 seconds post-exercise. Record the time it takes the pulse to return back to normal rate.
4. Score the subject based upon a) the difference between heart rate immediately post-exercise and normal standing heart rate and b) The time needed for heart rate to return to normal standing rate after cessation of exercise.

Difference btw normal and immediate post-exercise heart rates					
<i>Standing Pulse (bpm)</i>	<i>Elevation in heart rate over normal rate</i>				
	<i>0-10 bpm</i>	<i>11-20 bpm</i>	<i>21-30 bpm</i>	<i>31-40 bpm</i>	<i>>40 bpm</i>
61-70	3	3	2	1	0
71-80	3	2	1	0	-1
81-90	3	2	1	-1	-2
91-100	2	1	0	-2	-3
101-110	1	0	-1	-3	-3
111-120	1	-1	-2	-3	-3
121-130	0	-2	-3	-3	-3
131-140	0	-3	-3	-3	-3
SCORE					

Time for pulse to return to normal	
<i>Time (sec)</i>	<i>Points</i>
0-30	3
31-60	2
61-90	1
91-120	0
> 120	-1
SCORE	

TOTAL SCORE (sum of all scores): _____

Cardiovascular Fitness Rating	
<i>Total Score</i>	<i>Rating</i>
17-18	Excellent
14-16	Good
8-13	Fair
0-7	Poor
Negative value	Please check yourself into the hospital after class