

# BE 172 SPRING 2018

## WEEK VIII: HUMAN ELECTROCARDIOGRAM (ECG)

### OBJECTIVE

This lab will consist of 2 parts: First will be an electronic simulation of an ECG and examination of some properties of the differential amplifier, the same type used in the ECG amplifier. Second, groups will record the 12 Lead ECG on a fellow student to obtain & interpret a normal electrocardiogram, and observe and interpret some physiologic changes to the normal ECG.

### LABORATORY REPORT

A 2-page abstract in the usual format which includes: introduction, data, and discussion (be sure to consider the questions posed in the lab protocol). Included in this 2-page limit should be a composite 12 lead ECG of your experimental subject.

### PRELAB QUESTIONS

- Give 2 ways of determining the mean frontal QRS axis from a 12-lead ECG recording.
- What is respiratory sinus arrhythmia?
- Why might a condition such as cardiac hypertrophy (growth of the heart) change the mean frontal axis of the heart?
- What is the valsalva maneuver, and how will it be done in the lab?

### I. ELECTROCARDIOGRAM: AN AMPLIFIED RECORDING OF ELECTRICAL POTENTIALS GENERATED BY THE HEART

- Most commonly obtained using non-invasive, indirect methods -- surface electrodes applied to skin
- On some occasions ECG recorded directly by either applying electrodes to the surface of the heart or by using a catheter placed inside the cardiac chambers.

Extremely useful in clinical medicine to examine heart orientation, relative sizes of chambers, disturbances in rate and rhythm, extent and location of disease, etc.

- The ECG amplifier is basically a high-gain differential amplifier with some filtering circuitry.

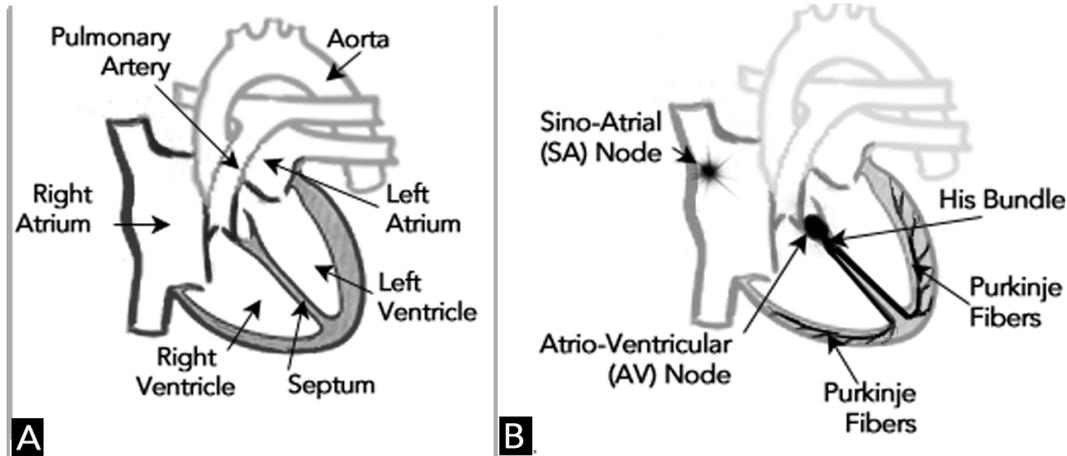
### II. KNOWLEDGE OF BASIC CARDIAC ANATOMY AND PHYSIOLOGY RELEVANT TO UNDERSTANDING OF THE ECG

Two types of cardiac muscle cells (myocardium):

1. "Working " myocardium
  - Cells have stable resting membrane potentials (RMP), contain large amounts of contractile proteins (actin and myosin), and are electrically connected to each other via gap junctions. When depolarized past threshold produce action potentials (AP) which are slowly conducted from cell to cell [via the gap junctions]
  - Makes up the majority of heart muscle mass (both atria and ventricles),
2. "Specialized" myocardium
  - Cells are all capable of becoming a pacemaker, but in normal situations never do so. Pacemaker mechanism related to slow depolarization during phase 4 of AP. Each specialized cell type has characteristic phase 4 slope, so that approx. pacemaker rates for each location are known.

Fastest = dominant pacemaker (typically Sino Atrial Node). Remaining pacemakers normally suppressed

- Specialized cells have much less contractile protein (actin & myosin) and fewer mitochondria, thus internal resistance is lower. Some of these cells (e.g., Purkinje fibers have extremely fast conduction velocities
- Locations include: Sino Atrial Node, Atrio-Ventricular Node, Bundle of His (Right Bundle Branch & Left Bundle Branch ), & Purkinje Fibers.

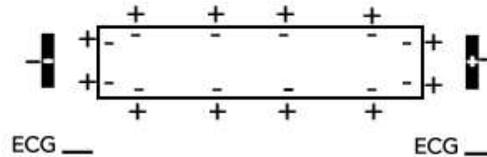


MAJOR STRUCTURES OF HUMAN HEART

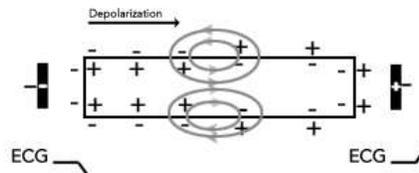
PACEMAKERS & CONDUCTING SYSTEM OF HEART

### III. BIOPHYSICAL BASIS OF THE ECG DESCRIBED USING CARDIAC DIPOLE MODEL

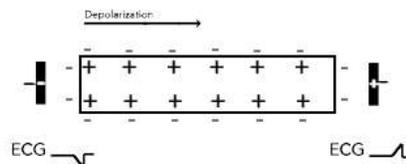
Consider a strip of isolated cardiac muscle placed in a volume conductor and arranged so that it can be electrically stimulated. The muscle can depolarize, contract, and repolarize. Electrical fields of depolarization and repolarization can be recorded using two unipolar electrodes (one at either end).



At rest, heart muscle cell is polarized (outside positive, inside negative). There is no potential difference between the two ends and no electrical activity detected. The ECG is isoelectric.

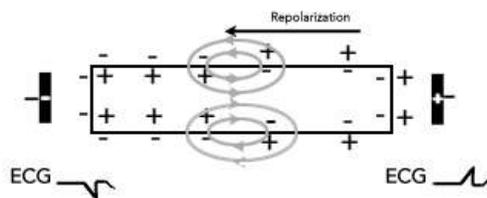


The muscle is stimulated. Propagation of the AP (depolarization wave) creates current flow in the surrounding medium due to the potential difference at the boundary between the depolarized and polarized tissue. These local currents are responsible for depolarizing additional membrane ahead of the action potential, causing it to propagate down the cell. By convention, a positive electrode deflection is recorded when the positive recording pole facing the positive pole of an approaching dipole.

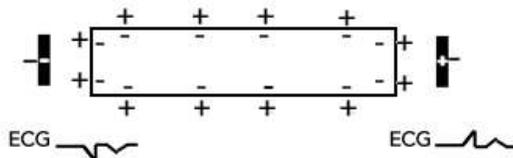


When depolarization complete, there is no electrical difference between the two ends of the muscle strip and the recording returns to baseline.

In hearts depolarization is rapid and displays a sharp positive spike.



In normal cardiac muscle repolarization starts in the last area to be depolarized and travels in a direction opposite that of depolarization.



Repolarization is a slower process, fewer cells being repolarized at any moment. Thus the repolarization wave is smaller and wider than the depolarization wave.

#### IV. RECORDING AND UNDERSTANDING THE BASIC ECG

##### A. ECG Overview

1. The millions of cardiac cells depolarize nearly simultaneously. First atria [P wave], then after a short delay, ventricles depolarize [QRS complex] with left ventricle depolarizing slightly ahead of right. Ventricular repolarization [T wave] occurs more slowly.
2. The analysis of typical ECG assumes heart is:
  - in a homogenous, isotropic conducting medium
  - observed from a distance large compared to the size of the heart, such that all individual current dipoles would appear to originate at the same point in space.

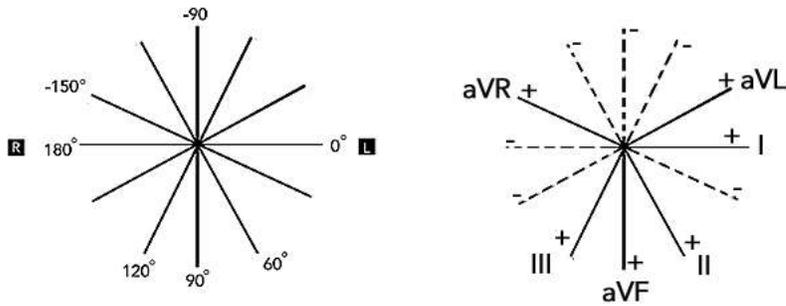
As a consequence, the heart vector can be approximated as a single dipole with magnitude & direction resulting from the vector summation of all individual dipoles.

##### B. ECG Recordings:

Uses electrodes on left leg [LL], left arm [LA], right leg [RL], right arm [RA]. RL is always ground, thus there are really 3 electrodes, 2 of which form a differential amplifier with the RL as ground. The following are the frontal plane combinations:

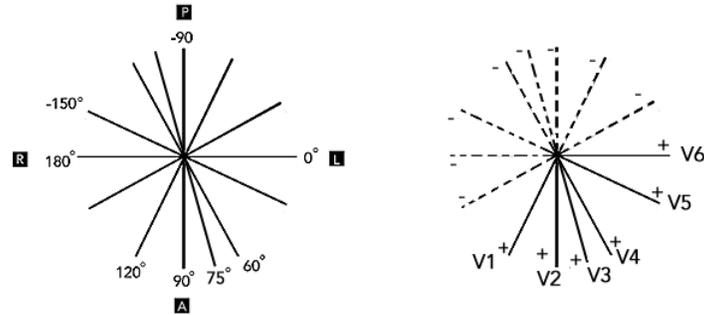
##### 1. Frontal plane electrocardiogram

- Standard limb leads - Bipolar, measuring potential difference across two sites
  - Lead I: +LA - RA
  - Lead II: +LL - RA
  - Lead III: +LL - LA
- Augmented limb leads- Bipolar, with one active (exploring) electrode and one reference electrode. Reference electrode is the average of 2 non-exploring electrodes.
  - Lead aVR (+ at right arm)
  - Lead aVL (+ at left arm)
  - Lead aVF (+ at left leg – they used F for foot since L (leg) was already taken by left!)
- Hexaxial Reference System includes the 3 standard and 3 augmented limb leads



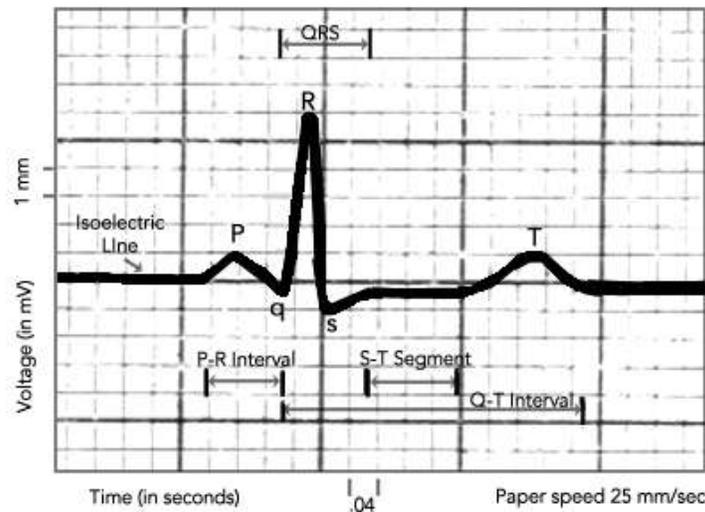
## 2. Transverse (Horizontal) Plane recordings

- Transverse plane leads [ designated V1- V6 ] provide Anterior-Posterior view of heart, i.e. components of the cardiac vector in the transverse plane
  - V1 4th intercostal space at R sternal border
  - V2 4th intercostal space at L sternal border
  - V3 Equidistant between V2 and V4
  - V4 5th intercostal space at left midclavicular line
  - V5 Anterior axillary line at 5th intercostal space
  - V6 Midaxillary line at 5th intercostal space
- Also called precordial leads (electrodes arranged on front of chest over heart)



## C. ECG Terminology

Consider the electrical depolarization & repolarization:



### 1. Sino Atrial Node depolarization

- Isoelectric. The number of cells is too small to be detected.

### 2. Atrial Depolarization

- Wave carried by specific conducting fibers toward Atrio-Ventricular Node
- Depolarization spreads through the working atrial muscle right -> left & superior-> inferior

- On ECG (typically lead II) small upward deflection - P Wave
3. Atrio-Ventricular Node & His-Purkinje Depolarization
    - Isoelectric. The number of cells is too small to be detected.
  4. Ventricular Depolarization
    - Multiphasic wave form, generically called- QRS Complex
    - Depolarization spreads from endocardium (inner wall) to epicardium (outer wall)
  5. Atrial Repolarization
    - Not visible in the recording. Occurs during QRS complex and is masked
  6. Ventricular repolarization
    - Produces upward deflection - T Wave
    - Typically "concordant" with most QRS complexes, but not mirror image of QRS
    - Repolarization is from epicardium to endocardium

Waves are regular deflections from baseline. For example: P wave, QRS Complex, T wave. As described above, the convention is to record a positive, upward deflection when an electrical impulse flow towards the (+) electrode and to record a negative, or downward deflection when electrical impulses are moving away from the (+) electrode.

Segments = periods between waves. For example: ST segment

Intervals = waves and segments (or complex waves). For example:

- PR interval (includes atrial, Atrio-Ventricular Node, his bundle, purkinje depolarization)
- QRS interval (ventricular depolarization); Duration related to time elapsed from 1st ventricular cell depolarized to last cell depolarized. Faster the spread through ventricle, narrower the QRS complex
- RR interval (=heart rate)

## V. BASIC ANALYSIS OF THE BASIC 12 LEAD ECG

### A. Rate and Rhythm

#### 1. Normal Sinus Rhythm

- Sinus P wave preceding each QRS complex
- Rate should be regular (60-100 beats per minute)

#### 2. Variations in sinus rate

- Sinus bradycardia - rate <60/min
- Sinus tachycardia - rate >100/min
- Sinus arrhythmia - variation in RR interval exceeds 0.16s

### B. Intervals

1. RR interval (1/heart rate) should be between 1 and 0.6 seconds, heart rate=60-100 bpm.
2. PR interval: 0.12-0.20 seconds (changed by Autonomic Nervous System & disease)
3. QRS interval: Usually <0.09sec. If > 0.12, then abnormal

### C. Mean Frontal QRS Axis [Normal -30° to +120°]

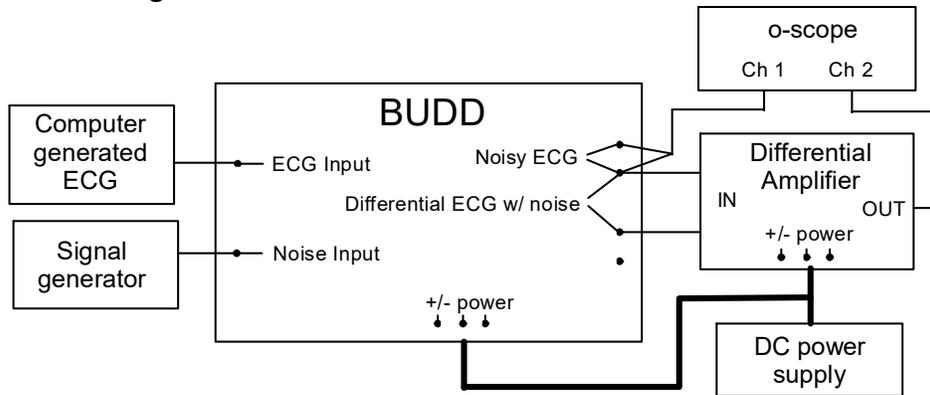
1. Average orientation of the time-varying 3D vector of ventricular depolarization in frontal plane
2. Alternative methods of determination
  - Maximum Amplitude method: look for largest R wave in frontal plane
  - Mean Net Amplitude method:
    - find lead with zero net amplitude (equi-phasic); vector perpendicular to this axis
    - inspection of another lead will show on which side mean vector located

# Human Electrocardiogram: Laboratory Protocol

## NOISE REJECTION BY A DIFFERENTIAL AMPLIFIER

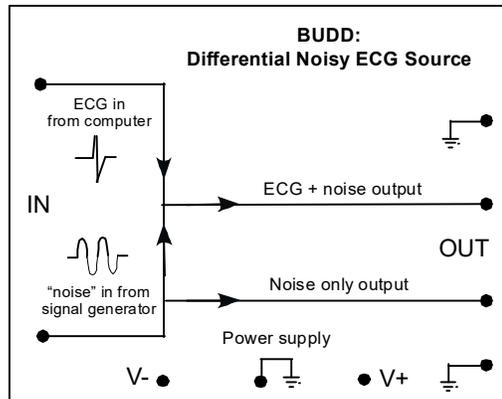
The ECG amplifier used in this lab is based on the AD620 differential amplifier. We will first examine the noise-rejection capability of this amplifier.

We will set up a differential voltage source which will be a computer-generated ECG with added sinusoidal "noise". Remember this is a differential signal, there is an ECG on one output and common noise on both. Thus, if we amplify the difference, we should see only the ECG and reject the noise. The set-up for this is shown in the figure below.



**Differential Signal Source Setup**

Set up the differential voltage source and amplifier as shown. We will use the box shown in the figure below, and call it "Budd", although you are welcome to think of other descriptive names for it. Budd has the ability to create a differential signal from 2 single-ended inputs. We will create an artificial ECG signal using the computer D/A converter, then add common "noise" to the ECG and see how the AD620 deals with the noise.



**Your Friendly Differential Voltage Source: Budd**

Create a differential source, ECG only: First Budd will be used to create an ECG signal. Before we subject Budd to the ECG, make sure the computer program is working properly by connecting the D/A Output 0 directly to the o-scope. Use the Labview program "ECG Waveform Generator". When you hit the start button a repeating ECG (taken from a real experiment) will be available at D/A Output #0. Look at it on the scope using a 200ms time scale and 500mV voltage scale; it should look like an ECG.

Connect the signal generator to the sine input of Budd. Turn the amplitude of the signal generator to the minimum position so it does not supply any voltage. Connect the output from the D/A panel of the computer interface as the other input to Budd. Budd also needs +/- power, which are the same lines as used by the differential amp. Look at Budd's output (ECG+noise) directly on the o-scope; it should be the ECG with some amplitude attenuation due to Budd's internal components. Remember that Budd's output is floating, hence never connect Budd's differential output directly to a grounded device like the scope.

Differential amplifier and a "clean" ECG: Now connect Budd's differential output (white terminals) to your differential amplifier (the same one used in the Nerve lab) and look at the amplifier's output on the o-scope. You should again see an ECG but amplified through the AD620. Check to see that the gain is close to that found previously.

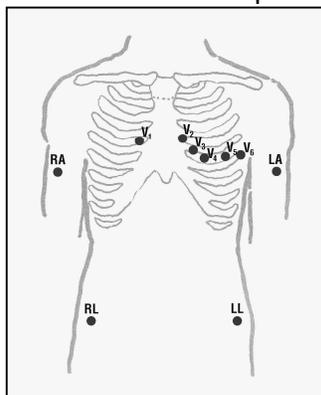
Differential amplifier and a "noisy" ECG: Finally, add "noise" to Budd's ECG by turning up the amplitude of the signal generator. This adds noise to both of Budd's differential outputs. Start with 60 Hz noise: you should see a sine-wave (a common signal) and maybe a hint of an ECG (the differential signal). But the output of the differential amplifier should only be an ECG. Notice as you turn the 60Hz "noise" amplitude way up, you will start to see some 60 Hz on the ECG (no amplifier is perfect!). By varying the amplitude of the sine wave common signal, produce a plot of ECG "noise" amplitude vs. input noise amplitude. Do this for one other frequency besides 60 Hz. The best place to measure the amplitude of the noise on the ECG is during systole after the QRS complex when the ECG is essentially "flat". Also, record the amplitude of the QRS complex; we will use this measure of "noise rejection" on the real ECG signal. Comment on any difference in noise rejection as a function of frequency (for your 2 frequencies).

## RECORDING THE 12-LEAD ELECTROCARDIOGRAM ON A FELLOW STUDENT

Take apart the Budd setup and put away all of the equipment except the o-scope and the blue/white ECG amplifier box and prepare to record a 12 lead ECG on at least one student volunteer from your group. ECGs can be recorded on other members of the group if time permits. You will use the ECG amplifier provided (also based on the AD620 amp) and record the output on the computer with the Labview software. There may be substantial 60Hz noise on the ECG (the noise varies with leads) which may mask parts of the ECG waveform.

### A. LEAD PLACEMENT

1. The subject should be sitting. Place all 10 electrode pads on the subject in the proper locations. Electrodes have a small amount of conductive jelly, and are made for single-time use only. Do not peel them off and move them! Limb leads are placed on the wrists and ankles. Connect the 4 limb lead wires according to color (Green=LA, White=LL, Black=RA, Brown=RL, Red=Chest). These wires will remain attached for the duration of the lab. The six precordial leads are positioned as follows:



- V1- Right parasternally, 4th intercostal space
- V2 - Left parasternally, 4th intercostal space
- V3 - Directly between V2 and V4
- V4 - 5th intercostal space, left mid clavicular line
- V5 - Directly between V4 and V6
- V6 - Left midaxillary line, same plane as V4

2. To measure the ECG in the transverse plane, the Chest wire is moved from to one of the 6 "V" electrodes, and the amplifier set on "V".

## B. ECG Recording

1. Connect the output of the amplifier to the oscilloscope and the A/D patch panel input. Start the Labview acquisition program, and use the "Generic 2-Channel A/D Module" setup from the top menu of the vi. Turn on the battery-operated ECG amplifier, and select one of the 6 frontal plane leads. Adjust the scope so that an ECG waveform is seen; you will have to use a "slow" time setting, around 0.5 seconds. Confirm that the baseline is free of interference. If there appears to excessive 60Hz noise, try moving the wires, the subject and possibly the electrodes. It is important to see the QRS complex as clearly as possible.
2. Record the ECG signal for a few seconds from each of the 12 leads, at 1000 Hz. Keep track of each lead you record on the computer. Remember you will include a 12-lead ECG as part of your lab report. Each person at the station will need the raw ECG data for further analysis.

## C. ECG ANALYSIS (ANSWER THESE IN YOUR REPORT, YOU DO NOT NEED TO DO THEM IN THE LAB)

1. What is the basic (underlying) cardiac rhythm? What are the criteria for normal sinus rhythm?
2. What is the heart rate? Is this value within normal limits?
3. Measure the PR, QRS, and QT intervals using lead II. Are they normal?
4. Examine the P waves. Do they appear normal? What leads are best for seeing them and why?
5. Are the QRS complexes normal? What criteria are you using?
6. What are the orientations of the T waves?
7. Are the PR, ST, and TP segments isoelectric? What does this imply?
8. Determine the mean frontal QRS axis. Is this value within normal limits?
9. Determine the mean transverse QRS axis. From the frontal and transverse axis vectors, find the mean 3D cardiac vector (in component form is fine).

## D. PHYSIOLOGICAL VARIATIONS IN THE ECG

For the remainder of the lab, select the lead with the "best" signal (least noise), and keep that configuration for all of these tests. You may need to adjust the acquisition duration to acquire data for different durations during the tests. Answer the questions in your lab report.

1. Determine the effect of normal expiration and inspiration. Try normal breaths first, then deep, sustained breaths.
  - What happens to the heart rate and rhythm? This is called respiratory sinus arrhythmia.
  - Is the result different with a deep sustained inspiration and expiration?
  - What is the mechanism of this change (look up respiratory sinus arrhythmia on the internet)? Hint: vagal nerve signals are involved.
2. Determine the effect of breath hold.

After a normal expiration, stop breathing (the longer the better!). Don't hyperventilate beforehand or take a deep breath. Record the ECG at the beginning and near the end of the breath hold.

  - What happens to the heart rate?
  - What might be the mechanism? Think chemoreceptors.

3. Determine the effect of a Valsalva maneuver (a forced expiration against a closed glottis).
  - How does HR change during this maneuver?
  - How do the PR, QRS, and QT intervals change with changes in heart rate?
  - What is the mechanism of this change? (again consult a friendly web site, this one is mechanical)
4. Determine the effect of body position.

Obtain a baseline ECG 'rhythm strip' while the subject is sitting quietly. Repeat the ECG immediately & two minutes after subject stands up. While standing, subject should remain perfectly still without moving the legs.

  - What are the changes and why do they occur?
5. Determine effects of hyperventilation.

Obtain control ECG recording with subject breathing quietly. After subject hyperventilates once for approximately 15-20 sec. [until "light-headed"] repeat measurement immediately & again every 30 seconds for 2-3 min.

  - How does the ECG change? How does this compare with the breath hold experiment?
6. Determine the effect of cold pressor.

Obtain control ECG again. Then have the subject immerse his/her hand in a bucket of ice water while heart rate is measured every 30 seconds for 2 min. Try to keep the hand in the water for the full 2 minutes even though it may be a little painful (this is your chance to get back at your lab partner for all of those experiments they ruined during the quarter!). Continue HR measurements every minute for an additional 3 minutes after the hand is removed from the ice.

  - How does the ECG change?
  - Can you explain the causes of these changes? This is a more complicated response via the central nervous system, part of the pain response.

#### E. ESTIMATION OF 60HZ NOISE ON THE REAL ECG

Based on your analysis of the AD620 differential amplifier with added "noise", determine the relative magnitude of noise on the real ECG signal recorded from the subject (assume 60 Hz noise). You should estimate the amplitude of the noise on the real signal, and amplitude of the QRS complex, as you did with BUDD. Based on the results from "noise rejection" measure obtained earlier, you should be able to estimate the amplitude of noise coming into the system on your actual ECG signal. Comment on this value and give some possible sources of the noise on an ECG.

**Make sure to turn off the battery-powered amplifier when finished!**