Plab 6: Electrocardiogram and Pulse Wave Velocity

By: David Young

Lab Partners: Jodi Small, Jordan Nick

Abstract:

EKG recordings can provide valuable information in diagnosing patient heart function, dysfunction and disease along with function or dysfunction of the autonomic nervous system. This lab explored the setup of a simple 4 lead EKG, recording of EKG lead signals, calculation of mean QRS and creation of 2D & 3D VectorCardiograms. By varying posture, body orientation, physical strain, and oxygen supply among other maneuvers, modulations of both the morphological profile of the heart and the stimulation of the parasympathetic and sympathetic nervous systems were achievable. Resultant changes in EKG were analyzed. By using force transducers on both the finger and toe and a simultaneous EKG recording, pulse wave velocity measurements were obtained for both the path from heart to finger or toe and the path from finger to toe. Statistical analysis was performed on a small group of 23 subjects resting, max, min and equilibrium heart rates while standing, but the sample size was too small to show normal distribution. Resultant data demonstrated that VCGs are a viable method of detecting changes in heart morphology and depolarization pathways, while a simple 3 lead EKG can suffice for calculating mean QRS vectors. It was found that stimulants of the parasympathetic nervous system slowed the heart rate, including vagal stimulation and inspiration. Results on changes in PWV in response to different maneuvers were for the most part inconclusive due to small relative changes in PWV and self-defeating data.

Introduction:

Hypertension is one of the largest diagnosed cardiovascular related diseases, affecting over 67 million Americans (31% or 1 in every 3 adults), with a staggering number more in the pre-hypertensive category. Hypertension is estimated to cost the nation almost 50 billion dollars each year. **[12]** For the average case, hypertension is a problem that should be addressed in the long term by individual changes in diet exercise and lifestyle; however drug therapies are a necessary and important solution for uncontrollable cases and as a stand in when individual lifestyle change is not occurring.

To date, extensive research has been done investigating therapeutic treatments of hypertension. Beta blockers are a class of drug that targets and blocks the beta receptor on the heart. Normally the beta receptor receives signals such as epinephrine that are part of the sympathetic pathway involved in raising blood pressure. Theoretically, blocking such receptors could decrease sympathetic stimulation of the heart and decrease the risk of hypertension. In 2012, one such investigation ("Beta Blockers for HyperTension") looked into beta blockers as first-line therapy for hypertension. **[1]**

This study compared beta blockers vs. placebos and other drugs in first-line therapy for hypertension and their relative effectiveness on mortality rate and rate of disease incidence. Used data from conglomerate of studies from 2006-2011 from Cochrane Central Register of Controlled Trials, Medline, Embase with the criteria of randomized control trials of at least 1 year duration. With data from over 40,000 subjects in total, the effectiveness of beta blockers was compared to placebo, diuretics, calcium channel blockers and renin-angiotensin system inhibitors. It was found that effectiveness of beta blockers on mortality rate was not statistically different with any significance relative to the effectiveness of placebo, diuretic, or RAS inhibitors, but was significantly higher compared to calcium channel blockers. Despite the lack of significant advantage in mortality, the incidence of cardiovascular disease showed more promising advantages with beta blockers. Total incidence of cardiovascular disease was significantly lower for beta blockers compared to placebo. This was attributed to decreased incidence of stroke because there was little statistical difference between beta blockers and placebo in the incidence of coronary heart disease. However the incidence of cardiovascular disease was significantly higher in beta blockers than calcium channel blockers and RAS inhibitors. It was concluded that there were mild benefits on cardiovascular disease when using beta blockers to treat hypertension, but little effect on mortality. Additionally the effects of beta blockers

were found in whole to be inferior to other anti-hypertensive drugs. Further studies involving categorization of beta-blockers to compare their effectiveness was a logical next step.

It is clear that the nervous systems regulating the heart are complex. A simple drug therapy is often insufficient to combat more serious conditions than hypertension. Atrial Fibrillation is one such unfortunate condition. Atrial Fibrillation is one of the most common heart arrhythmias and can lead to degraded cardiac functionality and even congestive heart failure. Different methods exist to combat atrial fibrillation, such as autonomic denervation. In 2010, one study ("Ganglionated Plexi Ablation for Longstanding Persistent Atrial Fibrillation") investigated ganglionated plexi ablation as a means to prevent long term or persistent atrial fibrillation.

The autonomic nervous system is known to play a role in triggering or perpetuating Atrial Fibrillation. This paper delves into the viability of ganglionated plexi (GP) ablation as a solution to persistent Atrial Fibrillation. Ganglionated Plexi are autonomic nerves that may play roles in triggering or perpetuating Atrial Fibrillation. The removal/ablation/dissection of areas of GP in the left atrium was performed in 89 patients "symptomatic, drug-refractory, persistent Atrial Fibrillation". A second ablation was carried out for 29 of the patients, and a third for 5 of the patients. The success rate of patients that received one GP ablation procedure was 38.2% at 24 months. The paper concluded that Ganglionated Plexi ablation resulted in long term maintenance of sinus rhythm in almost 40% of cases, and with repeat procedures almost 60% of cases.

Serious procedures such as GP ablation, even if successful, are not ideal. Early detection and prevention of such conditions is key to maintaining health. Luckily there are useful & inexpensive tools at the disposal of medical professionals to assist in observing cardiac function non-invasively. The electrocardiogram (ECG or EKG) is a powerful but simple tool made up of a finite number of skin electrodes placed around the body. These electrodes can measure the electrical potentials of cardiac muscle at different viewing angles. Pattern recognition facilitates the diagnosis of arrhythmias, abnormalities, disease, and heart rate among other statistics. Intelligently combining potentials from different measurement angles can facilitate the production of vectorcardiograms. Vectorcardiograms are even more powerful tools that can show the 2 dimensional or 3 dimensional propagation of depolarization through myocardium. Without these tools, cardiologists would face a great deal more difficulty diagnosing patients and preventing heart failure. Traditional resistance and clinical roadblocks of biomedical technology aside, given the growth of modern machine learning algorithms and pattern recognition, the possibility of a pocket-sized, inexpensive and automated cardiologist is not far off. This

lab will investigate the use of the EKG as a tool in a cardiologist's arsenal, simulating various changes in the heart via morphological changes or stimulations of the sympathetic and parasympathetic nervous systems. It will walk through setting up a simple EKG, performing procedural maneuvers, recording EKG tracings and finally producing and analyzing the vectorcardiograms as well as calculating the pulse wave velocity using reference points on an EKG. The EKG is an incredibly powerful system that has yet to be combined with enough technology to push its limitations as a consumer product. With basic understanding of EKG recordings and the right implementation, more accurate and inexpensive prevention of heart failure is not only conceivable, it's feasible.

Materials:

- Shielded Bio-Amp Cable
- Shielded Lead Wires (x5)
- Adhesive Electrodes (x7)
- Pulse plethysmograph (x2)
- Measuring Tape
- Ice
- Bowl for Ice Water

Methods:

LabChart software was used in conjunction with an AD instruments Powerlab & shielded bio-amp cables to record ECG tracing from a volunteer. With the subject lying flat on a table, 30 seconds of normal and inverted data was recorded for leads I, II and III with the following electrode placements.

Lead	Location of (+) electrode	Location of (-) electrode	Loc. of ground electrode
I (normal)	Right Arm	Left Arm	Right Foot
I (inverted)	Left Arm	Right Arm	Right Foot
II (normal)	Left Foot	Right Arm	Right Foot
II (inverted)	Right Arm	Left Foot	Right Foot
III (normal)	Left Foot	Left Arm	Right Foot
III (inverted)	Left Arm	Left Foot	Right Foot

A maneuver where the subject laid down, held his breath for 30 seconds and then exhaled for 30 seconds was used to alter the QRS axis. Recordings for leads I,II, and III were made.

The ground electrode was left in place, and all other electrodes were removed, before fitting 3 new electrodes: one just below the left scalpula, one on the anterior mid-chest just to the right of the sternum and one in the middle of the left side between the armpit and the hip. A lead V1 recording was

made with the negative on the back and positive on the chest electrode, followed by a V6 recording using the side as the positive electrode.

Leaving the precordial electrodes in place, simultaneous recordings of V1 and Lead II, followed by simultaneous recording of V1 and Lead III were made for the subject in the following positions: sitting in a chair with good posture, hunched over and arching his back.

Next ECG recording were taken using different maneuvers in an attempt to modulate the ECG. A lead II ECG was recorded while:

- The subject Inspired deeply 3-4 times
- The subject's carotid arteries were massaged alternating between left right and no massaging.
- The subject was standing straight up and quickly bent over
- The subject was lying quietly for ~120seconds, and then abruptly stood up for ~60seconds

Data was recorded for the subjects resting heart rate, max and min and equilibrated after standing heart rates.

Next recording were taken to aid in the calculation of pulse wave velocity. A lead II electrode setup was used in conjunction with a pulse transducer on the tip of the right finger, and another pulse transducer on the tip of the right big toe. Data was recorded while the subject was sitting relaxed, while the subject performed Valsalva maneuvers for about 15 seconds before release and while the subject was standing straight up and then quickly bent over. Then data was recorded while the subject rested comfortably and then while the subject attempted to move the lab table with their right leg. This procedure was accomplished twice, once while the subject held their breath and again while the subject made conscious note to breath. Next data was recorded while the subject dipped their free hand into a bowl of ice water. Finally the distance from the heart to finger and toe was recorded.

Results:

Due to faulty hardware, and some poor quality recordings, the following data was borrowed. **[4,5]**

Table 1 shows the heart rate and durations/intervals of different EKG complexes during a single cardiac cycle averaged from 3-7 cardiac cycles each.

	Avg. RR interval (s)	Avg. Heart Rate (bpm)	PR interval (s)	QRS duration (s)	QT interval (s)
Lead I Normal	0.916	65.502	0.152	0.096	0.365
Lead I Inverted	0.830	72.289	0.130	0.101	0.512
Lead II Normal	1.075	55.814	0.176	0.075	0.411
Lead II Inverted	1.062	56.497	0.185	0.094	0.499
Lead III Normal	1.002	59.880	0.181	0.072	0.377
Lead III Inverted	1.014	59.172	0.179	0.088	0.382

Table 1: Heart Rate and Complex Durations or Intervals averaged over multiple cardiac cycles

Table 2 shows the measurement of amplitude for the R, Q and S waves in each of the first three leads of both a normal and an altered EKG recording. To make the altered EKG recording, the patient was laid down, inhaled for long time, and told to hold his breath for 30 seconds before exhaling for 30 seconds. This maneuver was chosen in hopes of changing the position of the diaphragm to change the angle or position of the heart. The last column in Figure 2 shows the sum of the QRS complex for each lead found by subtracting the amplitude of the negative Q and S waves from the amplitude of the positive R wave.

all in (micro-Volts)	Amplitude of R wave	Amplitude of Q wave	Amplitude of S wave	Sum/Magnitude
Lead I Normal	288.75	0.00	270.28	18.47
Lead II Normal	963.00	0.00	347.40	615.60
Lead III Normal	863.19	90.72	119.13	653.34
Lead I Altered	155.00	0.00	219.00	-64.00
Lead II Altered	843.00	0.00	274.00	569.00
Lead III Altered	779.80	73.00	151.00	555.80

Table 2: Magnitude of individual QRS waves and resultant sum for normal and altered condition leads

The magnitude and angle of the mean QRS complex was calculated for both the normal and altered EKG recording. The calculations was accomplished via vector component addition. **[10]** The procedure was done twice for each EKG, once using only leads I and II and once using all 3 leads. The results are shown in Table 3. Leads I and II together produced the most sensible results (verified) and will be considered the baseline mean QRS angle (58.53 degrees) for the subject. **[8-**verification**] [9]**

Normal	Manitude(uV)	Angle(degrees)
for all 3 leads combined	1098.93	90.02
for only leads I & II	625.04	58.53
Abnormal		
for all 3 leads combined	1071.52	93.37
for only leads I & II	539.85	65.90

Table 3: Mean QRS amplitude and Angle (measured CW in degrees from 0). Note: Lead I & II produced proper angle.

Using the set of axes defined in Figure 1 three 2D vectorcardiograms and one 3D vectorcardiogram were created. For each sample unit of time in the recording, leads II,III and v1 were decomposed into vector components in each of the 3 unit directions (x,y and z) shown in Figure 1. The components in each direction were then summed before graphing the summations in each plane as well as in 3D. Although 3 simultaneous lead recordings from the same heart beat would have been preferable, the limited number of inputs (2 leads) meant the best solution was to align two similar beats and use leads II and v1 from one recording and lead III from another.

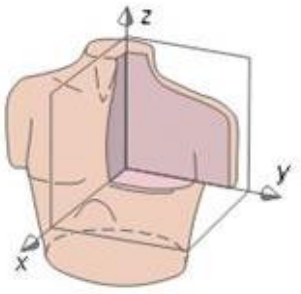


Figure 1: Definition of axes for vectorcardiograms

Figure 2 shows a set of vectorcardiograms for the subject in a normal seated position with good upright posture and a straight spine.

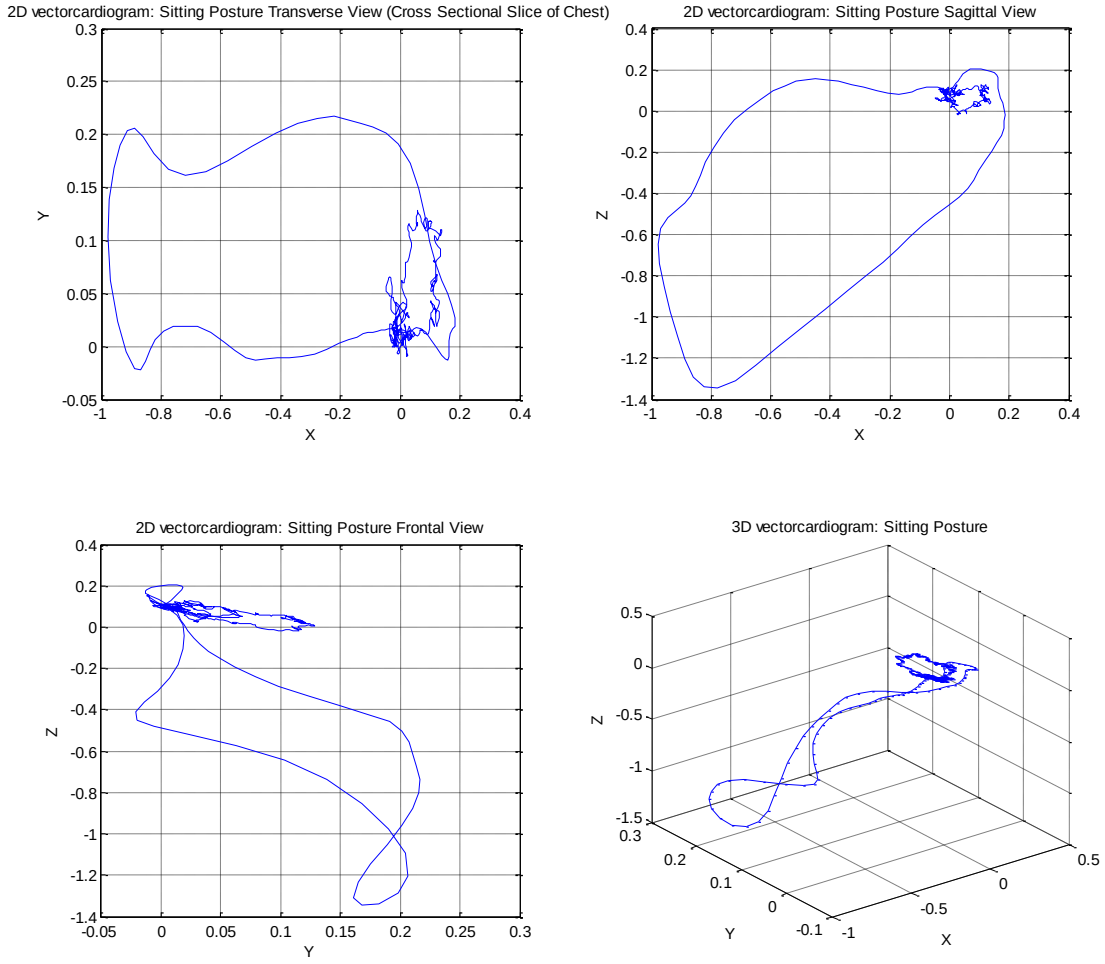


Figure 2: 2D VCGs for each plane and 3D VCG for the sitting posture.

Figure 3 shows a set of vectorcardiograms for the subject in a hunched over posture compressing the chest and elongating the back.

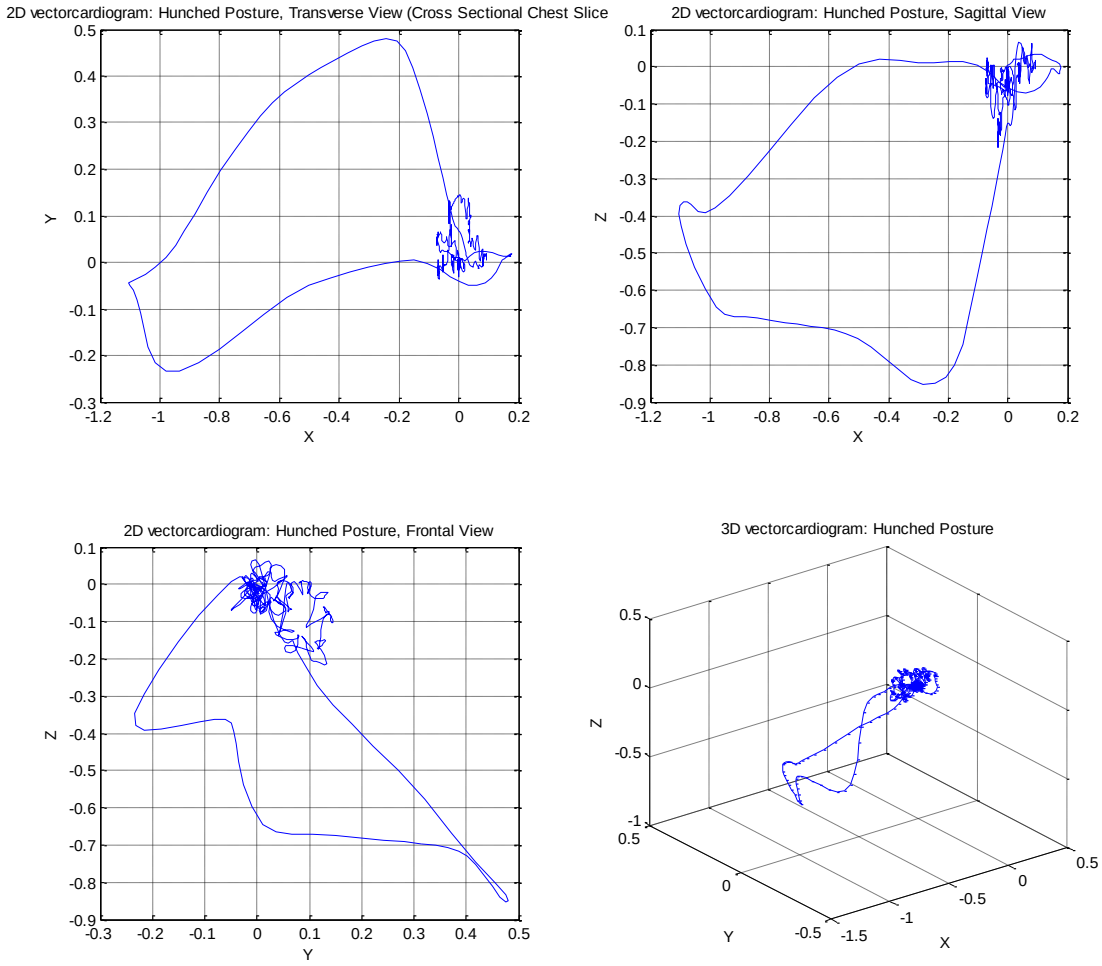


Figure 3: 2D VCGs for each plane and 3D VCG for the hunched posture.

Figure 4 shows a set of vectorcardiograms for the subject in an arched posture elongating and expanding the chest.

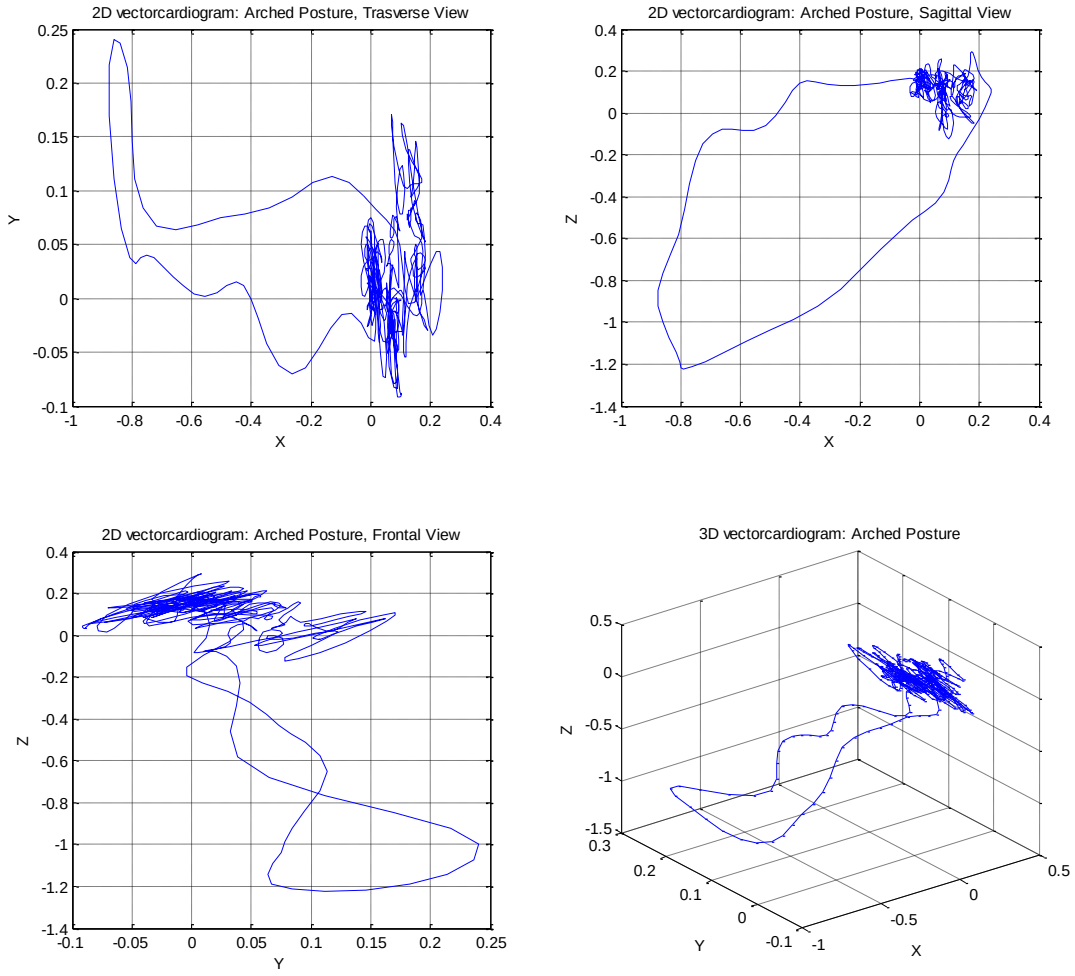


Figure 4: 2D VCGs for each plane and 3D VCG for the arched posture.

Table 4 shows the heart rate and PR interval averaged from 3 different cardiac cycles under various conditions including normal and heavy breathing, vagal stimulation, bending over and rapid elevation.

	3 x RR Interval (s)	HeartRate (bpm)	PR interval (s)
Normal Breathing	3.50	51.46	0.21
Inspiration 1	2.68	67.16	0.19
Inspiration 2	3.10	58.08	0.20
Inspiration 3	2.87	62.72	0.20
Avg. of Non baseline:	2.88	62.43	0.19
	3 x RR Interval (s)	HeartRate (bpm)	PR interval (s)
Baseline Vagal	3.30	54.56	0.17
Vagal Right	3.52	51.08	0.19
Vagal Left	3.43	52.52	0.19
Avg. of Non baseline:	3.48	51.79	0.19
	3 x RR Interval (s)	HeartRate (bpm)	PR interval (s)
Baseline Before Bend	2.95	61.08	0.19
Initially After Bend	2.52	71.46	0.22
Equilibrated After Bend	2.73	65.96	0.24
Avg. of Non baseline:	2.62	68.60	0.23
	3 x RR Interval (s)	HeartRate (bpm)	PR interval (s)
Basleine Before Standing	3.59	50.10	0.24
Initially After Standing	2.09	86.04	0.20
Equilibrated After Standing	2.42	74.26	0.19
Avg. of Non baseline:	2.26	79.72	0.20

Table 4: Heart Rate and PR interval averaged for different maneuvers including inspiration, vagal stimulation, bending over and standing

Table 5 shows the individual time and distance measurements used to calculate the average pulse wave velocity (PWV) from the heart to the finger, from the heart to the toe, and from the finger to the toe. Six different situations/maneuvers are shown to modulate the PWV including resting (which will count for both resting and pre Valsalva maneuver rest), Valsalva maneuver strain and its subsequent return phase, bending over, isometric exercise with conscious breathing and holding breath, and lastly dipping the subject's hand in ice water.

Table 5: Measurements used in and resultant calculation of PWV for resting, phases of Valsalva maneuver, bending over, isometric exercise with and without breathing, and icing the hand.

	Resting		
Finger			
	Time Between R and peak of PPG (s)	Distance (Heart to Finger) (cm)	Velocity (cm/s)
	0.257	91	
	0.252	91	
	0.262	91	
	0.268	91	
	0.268	91	
Average	0.261	91	348.13
TOE			
	Time Between R and peak of PPG (s)	Distance (Heart to Toe) (cm)	Velocity (cm/s)
	0.342	133	
	0.331	133	
	0.329	133	
	0.337	133	
	0.346	133	
Average	0.337	133	394.66
Finger & Toe			
	Time Between PPG Peaks (s)	Distance (Finger to Toe) (cm)	Velocity (cm/s)
	0.074	42	
	0.068	42	
	0.070	42	
Average:	0.071	42	594.34

	Valsalva			Valsalva Return	
Finger					
	Time Between R and peak of PPG (s)	Distance (Heart to Finger) (cm)	Velocity (cm/s)	Time Between R and peak PPG (s)	Velocity (cm/s)
	0.233	91		0.267	
	0.274	91			
	0.282	91			
	0.241	91			
	0.234	91			
Average	0.253	91	359.97		340.82397
TOE					
	Time Between R and peak of PPG (s)	Distance (Heart to Toe) (cm)	Velocity (cm/s)	Time Between R and peak PPG (s)	Velocity (cm/s)
	0.354	133		0.332	
	0.328	133			
	0.323	133			
	0.323	133			
	0.312	133			
Average	0.328	133	405.49		400.6024096
Finger & Toe					
	Time Between PPG Peaks (s)	Distance (Finger to Toe) (cm)	Velocity (cm/s)	Time Between PPG Peaks (s)	Velocity (cm/s)
	0.121	42		0.085	
	0.098	42			
	0.102	42			
Average:	0.107	42	392.52		494.1176471

	Bending Over		
Finger			
	Time Between R and peak of PPG (s)	Distance (Heart to Finger) (cm)	Velocity (cm/s)
	0.237	91	
	0.245	91	
	0.245	91	
Average	0.242	91	375.52
TOE			
	Time Between R and peak of PPG (s)	Distance (Heart to Toe) (cm)	Velocity (cm/s)
	0.086	133	
	0.331	133	
	0.331	133	
Average	0.249	133	533.42
Finger & Toe			
	Time Between PPG Peaks (s)	Distance (Finger to Toe) (cm)	Velocity (cm/s)
	0.098	42	
	0.098	42	
	0.090	42	
Average:	0.095	42	440.56

	Isometric Exercise Normal Breathing		
Finger			
	Time Between R and peak of PPG (s)	Distance (Heart to Finger) (cm)	Velocity (cm/s)
	0.260	91	
	0.271	91	
	0.256	91	
Average	0.262	91	346.89
TOE			
	Time Between R and peak of PPG (s)	Distance (Heart to Toe) (cm)	Velocity (cm/s)
	0.343	133	
	0.342	133	
	0.335	133	
Average	0.340	133	391.18
Finger & Toe			
	Time Between PPG Peaks (s)	Distance (Finger to Toe) (cm)	Velocity (cm/s)
	0.083	42	
	0.094	42	
	0.079	42	
Average:	0.085	42	492.19

Isometric Exercise Holding Breath

Finger			
	Time Between R and peak of PPG (s)	**Distance (Heart to Finger) (cm)**	**Velocity (cm/s)**
	0.241	91	
	0.248	91	
	0.271	91	
Average	0.253	91	359.21
TOE			
	Time Between R and peak of PPG (s)	**Distance (Heart to Toe) (cm)**	**Velocity (cm/s)**
	0.332	133	
	0.335	133	
	0.365	133	
Average	0.344	133	386.63
Finger & Toe			
	Time Between PPG Peaks (s)	**Distance (Finger to Toe) (cm)**	**Velocity (cm/s)**
	0.106	42	
	0.086	42	
	0.094	42	
Average:	0.095	42	440.56

Ice

Finger			
	Time Between R and peak of PPG (s)	**Distance (Heart to Finger) (cm)**	**Velocity (cm/s)**
	0.260	91	
	0.252	91	
	0.259	91	
Average	0.257	91	354.09
TOE			
	Time Between R and peak of PPG (s)	**Distance (Heart to Toe) (cm)**	**Velocity (cm/s)**
	0.323	133	
	0.316	133	
	0.331	133	
Average	0.323	133	411.34
Finger & Toe			
	Time Between PPG Peaks (s)	**Distance (Finger to Toe) (cm)**	**Velocity (cm/s)**
	0.075	42	
	0.076	42	
	0.071	42	
Average:	0.074	42	567.57

Discussion:

For leads I, II and III, switching the positive and negative electrode placements simply inverted the EKG recording. This was expected, as the electrical propagation in the heart stays constant and the placement of the electrodes is exactly the same. The only difference is the software's means of interpreting what is negative and what is positive. Since the switched leads are simply identical but inverted EKG recording relative to their normal counterpart, it is easiest to compare leads I, II and III by simply comparing their normal EKG recordings.

Lead I points horizontally from the right side of the body toward the left, at 0 degree angle on a unit circle and passes through the AV node. Lead II points from the upper right to the lower left at an angle of 60 degrees relative to lead I measured clockwise from 0. It also passes through the AV node. Lead III points from the upper left to the lower right at an angle of 120 degrees relative to lead I measured from 0. It too passes through the AV node.

The path of depolarization in the human heart is from the SA node, through the atria, through the AV node, then the bundle of HIS and its branches and through the purkinje fibers where it depolarizes the muscle tissue of the ventricles from the apex up. Because of this overall trend of upper right (location of the SA node origin) to lower left (location of the massive left ventricle) movement of depolarization in the frontal plane, the mean QRS vector is located between 0 and 90 degrees measured clockwise in a normal human heart. Typically the mean QRS is between about 60 and 90 degrees, but varies depending on size, weight, height and pathology. Because of this typical direction of propagation, it was expected that the lead II ECG recording would demonstrate the most positive overall QRS complex, followed by the lead III and lead I. This is because lead II is the closest of the 3 leads to being parallel to a normal mean QRS vector, and because lead I is pointing left (matching the horizontal component of the mean QRS) and lead III points downward (matching the more powerful vertical component of the mean QRS). Subtracting the Q and S wave amplitudes from the R wave amplitude for each lead allowed the comparison of the sum magnitude for each lead. It was found that the lead II and III were both overwhelmingly positive compared to lead I, as expected. However lead III was found to be slightly more positive than lead II in the normal heart position EKG recordings. This is most likely because the recordings are not from identical beats, but were taken in different trials.

A normal pattern of interventricular septum depolarization is a positive R wave and a negative S wave in lead v1. The small positive R wave corresponds to the initial anterior directed depolarization of

the interventricular septum and early ventricular depolarization whereas the large magnitude negative S wave corresponds to the overwhelming posterior directed ending depolarization of the left ventricle which sits slightly posterior to the AV node. Although the R wave in the normal lead v1 recording was very small relative to the S wave, it was still positive. With a positive R wave and a negative S wave, the data showed a normal pattern of interventricular septum depolarization. **[6]**

The data acquired conforms to the general layout of cardiac depolarization direction given by the Einthoven triangle, as explained before. However concerning magnitudes, conformity to the Einthoven's Law (that for any complex in the cardiac cycle, the potential of lead II equals the sum of the potentials of leads I & III) is a mixed bag in the acquired data. In the normal EKG, the lead II magnitude is within 9 percent of the sum of leads I and III. In the altered data, the lead II magnitude is within 14 percent of the sum of leads I and III.

The time intervals of different complexes (QT,PR etc.) in the different EKG recording under a specific condition should all be the same. This is because they should be recordings of the exact same depolarization movement through the heart, just from different angles. Angle does not affect timescales, simply amplitudes of the recording. Unfortunately the setup used did not permit the recording of all leads at once, so the duration of each complex is the average result from different trials. This means variation is inevitable given different beats with slightly different heart rates, conditions etc., despite an attempt to minimize conditional differences. Any discrepancies stem from this origin, as complex duration from identical beats should be identical.

For the altered scenario, the patient was laid down, inhaled for long time, and told to hold his breath for 30 seconds before exhaling for 30 seconds. This maneuver was chosen in hopes of changing the position of the diaphragm to change the angle or position of the heart. In theory, inhaling should bring the diaphragm down, making the thoracic cavity longer in the vertical direction and allowing the heart to shift to a larger angle measured clockwise from 0. It was found that the mean QRS angle shifted downward as expected. The shift was a little over 7 degrees (from 58.53 degrees to 65.9 degrees) using leads I and II to calculate the mean QRS angle.

Although raw vector component decomposition and summation was chosen to create vector cardiograms, the following derivation provides a useful formula for those looking to use angles rather than raw vector components.

$$I = V\cos\theta$$

$$II = V\cos\left(\frac{\pi}{3} - \theta\right)$$

$$Using\ Trig\ Identity: \quad II = V\left\{\cos\left(\frac{\pi}{3}\right)\cos(\theta) + \sin\left(\frac{\pi}{3}\right)\sin(\theta)\right\}$$

$$Sub\ in\ for\ I: \quad II = Icos\left(\frac{\pi}{3}\right) + Vsin\left(\frac{\pi}{3}\right)\sin(\theta)$$

$$Solve\ for\ \sin(\theta): \quad \sin(\theta) = \frac{II - Icos\left(\frac{\pi}{3}\right)}{Vsin\left(\frac{\pi}{3}\right)}$$

$$Solve\ for\ \tan(\theta): \quad \tan(\theta) = \frac{\sin(\theta)}{\cos(\theta)} = \frac{II - Icos\left(\frac{\pi}{3}\right)}{Vcos(\theta)sin\left(\frac{\pi}{3}\right)} = \frac{\frac{II}{I} - cos\left(\frac{\pi}{3}\right)}{sin\left(\frac{\pi}{3}\right)}$$

For all vectorcardiograms, a standard physical x,y,z coordinate system was used where +X points straight out of the chest, +Y points to the patients left, and +Z points directly up through the head. Figure 5 helps demonstrate the orientation. **[5]**

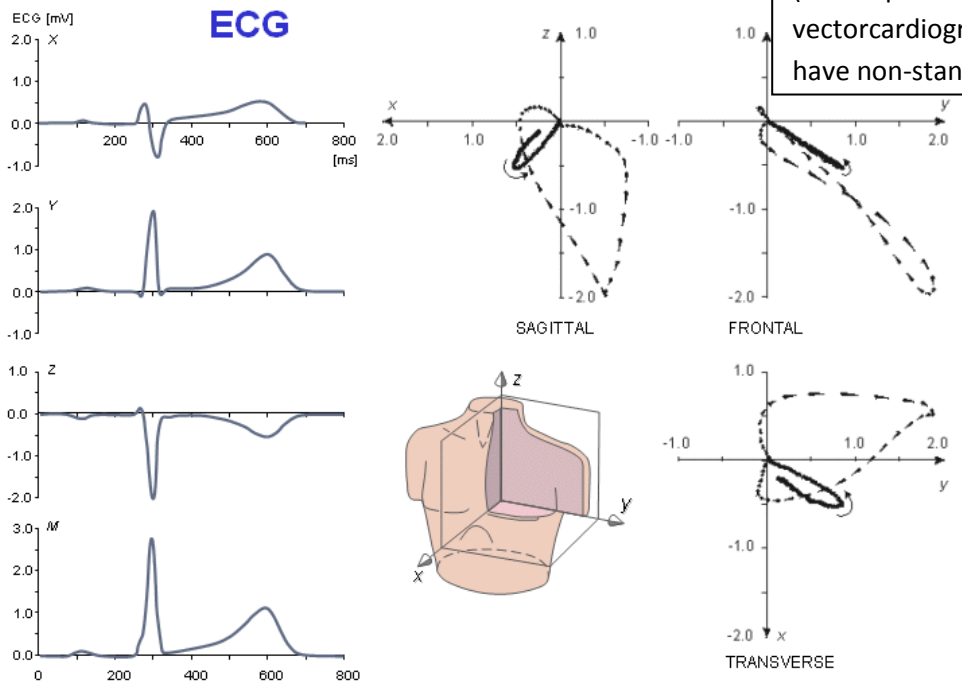


Figure 5: Reminder of Axes definition (center picture). 2D vectorcardiograms in this reference have non-standard plot rotations.

The chosen orientation of axes will drastically affect interpretation of the results, so please note the axes used here as they may differ from others. The 3 axes are zeroed at the AV node at (0,0,0). The frontal plane is the YZ plane, a cross sectional slice of the chest is the XY plane, and a side view is the XZ plane. The vectorcardiograms were formed by vector decomposition from leads II,III and v1, followed

by simple trigonometric calculations to find the projections of each lead onto each of the normal x,y,and z axes.

The vector cardiogram for the normal posture makes complete sense. Assuming the AV node to be the origin of the coordinate system, a lot of baseline noise can be seen looping around (0,0,0), mostly slightly above the origin. The depolarization moves downward and to the left (positive Y and negative Z) in the frontal plane demonstrating the depolarization moving from the AV node down toward the bottom of the heart. The depolarization moved to the left and backward (negative X and positive in Y) in the cross sectional chest plane. This corresponds to the depolarization moving from a slightly more anterior and rightward AV node toward a more posterior and leftward depolarizing mass of the heart. This is exactly as expected. Although the orientation of the heart places the apex in line or possibly anterior to the AV node, the rotated position of the heart means the larger left ventricle is actually mostly posterior to the AV node with the smaller right ventricle being slightly anterior to the AV node. **[4]**

When the subject hunched over it was expected that the compression of the chest and elongation of the back would squish the anterior side of the heart in the vertical direction and lengthen the anterior side of the heart in the horizontal direction, making the cross sectional area look more square in the frontal plane. Depending on the how serious of an angle the upper body formed bending over, it was theorized possible to simultaneously elongate the posterior side of the heart as the anterior compressed. However the latter seems relatively unlikely given that the heart is not solidly fixed to every wall of the thoracic cavity and therefore has some freedom in movement if given the room. The unavoidable morphological changes should arguably result from compression, whereas stretching requires a more dramatic change in posture given the hearts freedom to move along the inside of the pericardium. Overall it was expected that hunching would result in a shorter yet fatter heart (fatter in both horizontal directions), with a decreased mean QRS angle. It was also expected that the left ventricle would become even more posterior relative to the AV node, and therefore result in increased amplitude in the negative x-direction (toward the back). These expectations were verified by the data acquired. The range for the z coordinate (representing the heat of the heart) decreased almost 30% from normal while the range for the y coordinate increased significantly (almost by a multiple of 3). The range for the x coordinate increased slightly, but not by nearly as much as the range for y. Overall this verifies that the heart was squished in the vertical direction and elongated in the horizontal. It is easiest to see this change by comparing the areas enclosed by the 2D vectorcardiograms in the transverse plane

(XY plane). The area enclosed by the hunched posture vectorcardiogram is significantly larger than the area enclosed by the normal posture counterpart.

When the subject arched his back, it was expected that the thoracic cavity would elongate in the vertical direction and narrow in both directions of the horizontal, possibly pushing and pulling the heart along with it. It was expected that this change would yield a taller but skinner heart (specifically skinnier along the x axis from the sagittal viewpoint), and an increased mean QRS angle. It was also expected that this posture would render the left ventricle less posterior relative to the AV node compared to normal posture, resulting in a decreased amplitude in the negative x direction (toward the back). These expectations were mostly verified by the data acquired. Although the peak range of the y coordinate for the arched posture vectorcardiograms did not decrease much relative to normal posture, the area enclosed by the vectorcardiogram in the transverse plane is significantly smaller for the arched than it is for the normal posture. This is indicative of a thinning of the heat in the horizontal. The range for the z coordinate stays about the same for both normal and arched posture, indicating the vertical stretching that was expected was not so apparent. This could be due to the location of the majority of such a stretch (ie: in the upper anterior side of the heart whose depolarization is more subdued and overpowered in the recordings by the meaty left ventricle), poor recordings or simply an incorrect theory all together. Either way the heart was definitely thinned its area in the transverse plane.

The movement of the diaphragm is expected to shift the hearts orientation and morphology slightly. An elongation of the thoracic cavity in response to deep inspiration may result in the heart's mean QRS shifting towards larger angles measured clockwise from 0, ie: more toward 90 degrees (directly down). Additionally it was expected that slow powerful rhythmic respiration would decrease the heart rate slightly. The data matched the expected results. On average, the heart rate during inspiration increased from 51.46 to 62.43bpm.

It was expected that massaging the carotid arteries would stimulate the vagal nerves and induce a parasympathetic response that slowed heart rate. **[1]** This was found to be accurate with lengthened RR intervals and an average drop (between the right and left carotid artery massage) of around 3 bpm from a resting 54-55bpm.

It was expected that upon sudden standing up after resting in a horizontal position, that the patient's heart rate would increase and that the power of contraction would increase due to induced sympathetic response known as orthostatic hypotension. It was suspected that sympathetic response

would occur to compensate for changes in pressure resulting from the effects of gravity upon standing up. Since pressure is a function of height (ρgh), laying on the ground meant little pressure was required to get blood to the brain. When one quickly stands up, blood could pool in the legs unless there was some peripheral vasoconstriction and increased blood pressure to ensure blood reached the head. The data seemed to support this hypothesis, as the heart rate jumped from around 50bpm at rest to 86bpm (to compensate for reduced pressure) upon standing up and equilibrated to around 75bpm. This was a marked increase that onset immediately after standing up, implying rapid onset of sympathetic response and then retraction and balancing of sympathetic response.

Table 6 shows the raw heart rate sample data from 23 different subjects.

	Heart Rates (bpm)			
Subject #	Rest	Max while Standing	Min while Standing	Equilibrium while Standing
1	49.2	85.1	50.2	60.7
2	50.0	90.1	45.7	58.3
3	56.9	92.9	46.7	76.5
4	53.5	90.4	60.1	68.9
5	58.3	97.8	65.7	85.2
6	63.6	94.5	76.0	82.0
7	69.0	96.0	78.0	84.0
8	65.2	93.8	69.8	73.2
9	72.0	96.0	90.0	93.0
10	71.0	108.0	73.0	100.0
11	60.0	85.6	51.5	67.3
12	51.4	83.6	67.7	76.1
13	55.0	78.7	68.6	72.4
14	70.0	102.4	64.9	87.5
15	61.5	99.5	61.9	70.0
16	68.2	114.0	76.1	65.0
17	48.6	75.2	55.1	57.1
18	72.0	108.0	74.0	78.0
19	51.0	87.0	62.0	69.0
20	69.3	93.8	65.0	72.0
21	68.0	80.0	65.0	72.0
22	64.3	99.2	72.0	69.2
23	63.1	91.5	59.6	67.3

Table 6: Cumulative Data for All Lab Groups involving 23 different subjects heart rates

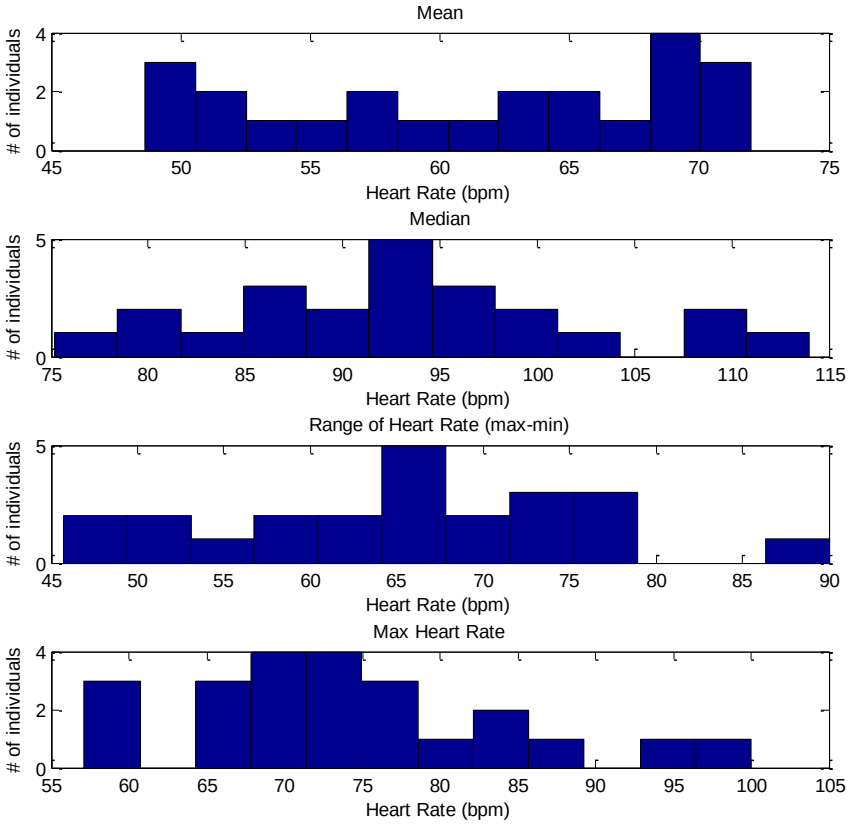
Table 7 shows some brief statistical analysis for the mean, median, range and maximum heart rates.

		Heart Rates (bpm)			
		Rest	Max while Standing	Min while Standing	Equilibrium while Standing
	Mean	61.35	93.17	65.15	74.11
	Median	63.14	93.75	65.00	72.00
	Range	23.38	38.81	44.30	42.86
	Max	72.00	114.00	90.00	100.00
Std. Deviation		8.03	9.66	10.78	10.70
	Variance	64.41	93.30	116.15	114.41

Table 7: Statistical values for each category of recorded heart rate.

A 12 bin histogram was created for the mean, median, range and maximum heart rates from a sample size of 23 subjects. See Figure 6. By visually analyzing the mean histogram, the resting heart rates do not appear to have a Gaussian distribution. However, a ztest at the MATLAB default 5% significance level was used to test if the data in each category came from a distribution with its mean. For mean, median, range and maximum heart rate, the ztest returned an h value of 0, indicating that the null hypothesis could not be rejected at the 5% significance level.

Figure 6: Histograms for resting, median, range and max heart rate



Then a sample t-test was used to test the hypothesis that the change in heart rate from resting to minimum while standing was statistically significant with a significance level of 5%. Similar tests were used to for the change in heart rate from resting to maximum while standing and to equilibrium while standing. It was found that all changes were significant. A ttest was chosen because the sample size was under 30 and a statistical evaluation of the significance of discrepancies was required.

The pulse wave velocity (PWV) to the lower extremities (measured with the right big toe) and the upper extremities (measured with the right finger) were relatively similar at 348cm/s for the former and 394cm/s for the latter. Without further understanding of the vasculature that carries blood to the two

locations, it is hard to predict which should be faster in theory, but a brief discussion presents a few theories. First it is important to note that for similar sized vasculature, all blood in the body should be circulating at nearly the same speed, as there is only one pumping source and a fixed volume of blood. However given the nature of physiology, it is rare that such a simplification and its assumptions could hold. It is possible that if the line to the toe is in its entirety a downward path through the vasculature, then the added acceleration of gravity along with the equal blood pressure could produce faster speeds to the toe relative to the upper extremities which decelerate due to gravity for the duration of the upward path to get through the shoulder before heading to the finger. This would be supported by the data which shows slightly faster speeds at the lower extremities than the upper. However significant differences were not expected. Any major discrepancies are most likely due to human error in the measurement of distances used for calculations, along with misunderstanding the route whose distance needed to be found.

The pulse wave velocity (PWV) was calculated with two methods, both measurements of distance traveled over time. When calculating PWV using the R wave of an EKG to represent the pulse starting at the heart, and the pulse from a transducer on the finger or toe to represent the pulse reaching the extremity, the results were within reason according to previous studies which found PWV varied by age among other factors and had a typical range of 300-700cm/s. **[2]** Since the subject here was young (early 20's) his vessels were compliant and not stiff, corresponding to his lower PWV in that range. However using the difference between the two extremity's pulses yielded significantly higher results with a pulse wave velocity of 594cm/s. This is most likely because the path between the two extremities is not as conforming to a simple line draw within the perimeter of the body, whereas the path from the heart to each of the extremities on its own may very well be closer to that simple. The extra hidden distance in windy path taken from upper extremity to lower extremity may explain the distorted and overly large velocity value obtained with the second method. Because of this, it is safer to stick to using an ECG and a single force transducer in calculating the PWV.

During the various stages of Valsalva maneuver, it was expected that the PWV would change. A normal PWV was expected for the initial rest, an increased PWV was expected for the strain portion due to an increase in blood pressure that is strongly correlated with increased PWV and a decreased PWV was expected for the final return phase. Using the normal initial test as the baseline for rest, the strain and return phases are poorly demonstrated in the data where there is actually a small increase in both

the PWV to the finger and toe. The pulse wave velocity from the finger to the toe however was significantly lower.

To compare two slightly different types of exercise, the subject attempted to move the table with their free leg while both holding their breath and consciously breathing. It was expected that the PWV would decrease when the subject made conscious effort to breathe relative to when the patient was holding their breath. This is because the breath holding can decrease the heart rate, raising blood pressure to compensate.**[7]** The raise in blood pressure can increase PWV. However breathing can increase the heart rate, relieving the need to increase blood pressure. A slight increase in PWV to the finger during conscious breathing countered the initial theory, but a decrease in PWV to the toe and a decrease in PWV from the finger to the toe supported the theory.

Based on previous studies and physical laboratories, it was expected that sympathetic response would result from placing the subject's hand in ice water, which would increase PWV relative to resting via an increase in heart rate and blood pressure.**[3]** The trend held true for the PWV to the finger and to the toe, which both increased. The increase was not large however and arguably insufficient to make any conclusion.

Official Sources:

(1) Wiysonge CS, Bradley HA, Volmink J, Mayosi BM, Mbewu A, Opie LH. Beta-blockers for hypertension. Cochrane Database Syst Rev. 2012;8:CD002003.
(2) Pokushalov E, Romanov A, Artyomenko S, et al. Ganglionated plexi ablation for longstanding persistent atrial fibrillation. Europace. 2010;12(3):342-6.
(3) Quantitative Physiology 2: Physical Laboratory Manual
(4) Paras Vora, Karthik Krishnan, Matt Everett – borrowed all EKG data but the vectorcardiogram data
(5) Chris Beyer – borrowed EKG data for the vectorcardiograms

Unofficial Sources:

(1) http://circres.ahajournals.org/content/5/5/562.full.pdf

(2) http://hyper.ahajournals.org/content/56/2/217/T2.expansion.html

(3)David Young "Physical Lab #3: Dive Response"

(4) Jim Miller Physics of the Heart Professor Washington University

(5) http://www.bem.fi/book/20/20.htm

(6) http://www.ncbi.nlm.nih.gov/books/NBK2214/

(7) http://www.ccjm.org/content/76/Suppl_2/S37.full

(8) Patricia Widder, Lab Author

(9) http://courses.kcumb.edu/physio/ecg%20primer/ecgaxis.htm

(10) http://hyperphysics.phy-astr.gsu.edu/hbase/vect.html#vec6

(11) http://www.bem.fi/book/20/20.htm

(12) http://www.cdc.gov/bloodpressure/facts.htm

Discussants:

-Lauren Bedell, Paras Vora, Maeve Woeltje, Karthik Krishnan,Satish, Matt Everett, Shay