HEART RATE VARIABILITY IN COLLEGIATE DIVISION I ATHLETES

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Part I

INTRODUCTION

Analysis of Heart Rate Variability (HRV), the variation in interbeat intervals, is a common, noninvasive method to assess autonomic activity of the heart [1-4]. During exercise there is parasympathetic withdrawal and sympathetic excitation resulting in acceleration of the HR, while these effects reverse during recovery [2, 5]. Generally, high HRV reflects a healthier and more adaptable autonomic nervous system (ANS), while diminished HRV indicates autonomic imbalance [4]. Previous studies have examined the ANS associations with HRV and exercise [5-9], exercise recovery [3, 9-16], heat and ice therapy [14, 17, 18], overtraining [19], longevity of life [4, 20], stress and chronic and acute pain [1, 21-29]. Heart rate variability has been used to determine many different health and fitness indicators such as early indications from myocardial infarction and how adaptable the heart is. While most of these HRV studies have compared the outcome to a control group to identify abnormalities, the consensus of a standard healthy HRV measurement has yet to be determined.

Assessment of HRV involves analysis of two domains: time domain and frequency domain measures [4, 30, 31]. Though there are many different measures for time-domain the four most common measurements assessed are standard deviation of all NN intervals (SDNN) and HRV triangular index both estimate the overall HRV, standard deviation of the averages of NN intervals in all five minute segments of the entire recording (SDANN) which estimates the long-term components of HRV, square root of the mean of the sum of the squares of differences between adjacent NN intervals (RMSSD) which estimates the short-term components of HRV, and the number of pairs of successive NNs that differ by more than 50ms divided by the total

number of NNs (pNN50). Time domain analysis provides a general idea of the variability of the RR interval. Frequency domain provides the sympathetic and parasympathetic activity ratio via spectral analysis, which decomposes the ECG wave into multiple bands based on its frequency. When analyzing frequency domain, three bands can be observed; very low frequency (VLF), low frequency (LF) and high frequency (HF) components [4, 30]. Very low frequency is mainly mediated by thermoregulation [4, 30, 31]. High-Frequency is mainly mediated by the parasympathetic modulation to the sinus node directly associated with respiratory activity, whereas LF reflects the mixed modulation of parasympathetic and sympathetic activities; therefore, the ratio of the high frequency power to the low frequency power (LF/HF) provides the sympathetic component [4, 30, 31].

In order to compare outcomes to other studies the data collected has to have the same units of measure, be taken during the same conditions, and define a specific population [4]. Most data collected utilizes time and frequency domain measures yet the conditions under which the data is collected may vary. When collected at rest, LF baseline scores range from as low as $170.3 \pm 202.5 ms^2$ [19] in a 10-minute data collection session to as high as $442 \pm 446 ms^2$ [32] in a 15 minute data collection session. The ranges during pre-exercise or activity vary even more, from $189 \pm 50.96 ms^2$ [27] to $2091 \pm 2177 ms^2$ [10]. The LF/HF ratio also showed differences in non-trained subjects, one study showed the ratio as high as 4.8 ± 0.5 [9] and another study showed it as low as 0.47 ± 0.35 [19]. Time domain measures reported in the previous studies also lack in consistency. The SDNN in trained subjects during pre-exercise, ranged from 77 ± 30 [10] to 218 ± 59 [11]. Though there have been several studies examining HRV, inconsistency in the protocol for data collection reflects a wide range in the outcome values.

Besides the differences in data collection protocol, HRV is affected by the fluctuation in

HR, which can be influenced by several physical and mental stressors. Changes in HR could be acute and temporary in response to short-term stressors such as illness, emotion, school work [33], respiration [34], medications [35] and body temperature [21], or could be as a result of physiological adaptations to constant stressors such as cardiovascular diseases [19] and exercise [36]. The cardiovascular response to physical activity is dependent on the intensity and the type of exercise [4, 30, 31, 37, 38]. Physiological adaptations associated with endurance exercise training will lead to a decreased heart rate, both with activity and at rest, associated with an enhanced parasympathetic modulation [11, 32, 39-44]. When comparing aerobically trained athletes to a control group, the trained athletes had higher HRV in supine and in standing condition [30, 45]. While these physiological adaptation could be viewed as positive training effects, too much training stress and too many training sessions can result in lack of adequate recovery and exhaustion, which will then lead to an autonomic imbalance, most commonly known as over-training [30]. Theoretically, HRV measures have potential to identify those who are at risk for overtraining by monitoring the autonomic response to training. However, there are no studies providing an appropriate range of HRV measures in athletes that could be used to determine if training is effective or if the athlete is at risk for overtraining.

Therefore, the purpose of this descriptive study is to analyze the characteristics of HRV in National Collegiate Athletic Association (NCAA) Division I highly trained collegiate athletes.

METHODS

Research Design

This study utilized a cross-sectional design to assess HRV in NCAA Division I athletes. All participants underwent one resting data collection session in the middle of the competitive season. Time and the fast Fourier technique of frequency domain were used as HRV outcome measures to determine the mean, SD, 95% confident interval (95%CI), and standard error of mean (SEM) for the group. These data were then compared by primary training type, aerobic or anaerobic, and by injury status, injured or non-injured.

Participants

Forty-eight (n=48) participants (average age 20 ± 1.49 y/o; M=5, F=43) that were currently training with NCAA Division I athletic teams were recruited. Participants demographics are shown in table 1. Figure 1 shows the distribution of the athletes per sport. Participants were categorized into one of the three group based on the predominant focus of the athletes' training, described below in Table 2. The team sport group was comprised of women's basketball players, women's water polo attackers, and women's soccer players. The anaerobic group was comprised of women's track and field sprinters, women's track and field throwers, swim sprinters, women's water polo goalies, baseball pitchers, women's beach volleyball and football offensive linemen who were more explosive and power athletes and the aerobic group was comprised of cross country runners and distant swimmers who spend the majority of their training in steady state. Prior to inclusion in the study, participants were asked to fill out Health History Questionnaires,

and informed consent form approved by the University of Hawaii Human Study Program. Participants that self-identified in the health history questionnaires as having an allergy to adhesives or suspected pregnancy were not eligible for inclusion in this study.

Table 1. Subject Demographics

N Males=5 Females=43	Mean ± SD	Minimum	Maximum
Age (years)	20±1.47	18	25
Height (inches)	67.7 ± 2.97	61	78
Body Mass (pounds)	157.83 ± 34.47	93	308

Table 2. Description of Groups Based on the Training Characteristics

Group #1 (n=16)	The "Team Sports" group (TS). It consists of women's basketball players, women's water polo attackers, and women's soccer players. The training often included a combination of aerobic and anaerobic training methods. Their training is also spent competing against teammates, rather than one's own time or personal record.
Group #2 (n=16)	The anaerobic group (ANA) which consist of track and field sprinters, track and field throwers, swim sprinters, water polo goalies, baseball pitcher, women's beach volleyball and football offensive linemen. They are more of the explosive/ power athletes, who often expend a lot of energy in a short period of time.
Group #3 (n=12)	The aerobic group (AE) which consist of cross country runners and a distant swimmer. The athletes in this group often perform at a slower pace for long periods of time.

Instruments

The ECG data were collected using CARDIO-CARD ver. 6.01ia software (Nasiff Associates, Inc., Brewerton, NY, USA). Anthropometric data including height (cm) measured by wall-mounted stadiometer, body mass (kg) measured by Detecto Certifier scale (Detecto, Webb City, MO, USA), and age were plugged into the CARDIO-CARD. The ECG data were

then exported to Kubios Heart Rate Variability ver. 2.2 software (Biosignal Analysis and Medical Imaging Group, Dept. of Physics, University of Kuopio, Finland) to obtain time and frequency domain data. Prior to electrode application, the skin was cleaned and prepped. The right and left arm electrodes were placed below the right and left clavicles, respectively. The right and left leg electrodes were attached to the right and left sides of the trunk, below the tenth rib on the anterior axillary line. The V5 chest electrode was placed on the left side of the fifth intercostal space on the anterior axillary line.

Experimental Procedures

Testing sessions were performed in the Human Performance Laboratory at the University of Hawaii at Manoa. Participants were asked to refrain from any vigorous activities, such as playing sports and riding bicycle as well as ingesting any caffeine for 3 hours prior to the data collection. Following the verbal explanation of the study procedure, all participants read and were asked to sign an informed consent form and fill out the Health History Questionnaire to identify the exclusionary criteria.

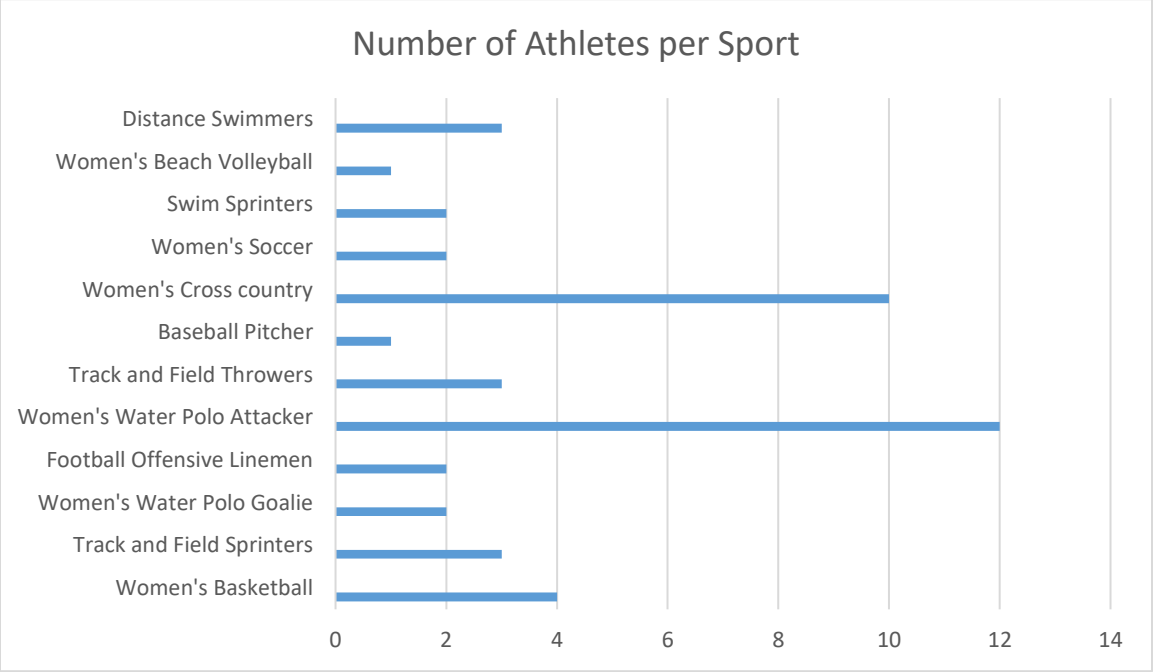
A Board of Certification Certified Athletic Trainer collected all data. Anthropometric data, including body mass, height and blood pressure, were collected and recorded prior to the testing session. Following anthropometric measurements, the participant was instructed to lie down supine or semi-reclined (less than 30 degrees) in a comfortable position in which they could remain throughout the data collection. The investigator cleaned the electrode placement sites and the electrodes were applied to designated positions. After 10 minutes of resting in comfortable position, the ECG data were recorded for 15 minutes. The participant was instructed to relax and breathe at their normal, self-determined pace, remain as steady as possible, and not to fall asleep during the data collection period.

Electrocardiography (ECG) data were collected by Cadrio-Card to obtain R-R intervals. The raw R-R data were filtered using macro excel sheet to identify the eptopic beats such as T waves higher or R waves lower than 500 millivolts. If T wave was higher than 500 millivolts, indicated as R wave, then the adjacent R-T and T-R interval were combined to obtain R-R interval. If R wave was lower than 500 millivolts, this segment was removed. [46] Following the initial data screening, R–R interval file was then converted as a .txt file to be exported into Kubios HRV analysis software (HRV Analysis Software 2.2, University of Kuopio, Finland) to assess time and frequency domain measures [46]. Data were filtered using a low-level artifact correction then the most stable five-minute data period was selected for analysis. For the frequency domain data, trend components were removed using a Smooth n Priors to obtain the normalized units, the LF and HF components without the influence of the VLF, and any artifact. Frequency bands for HRV analysis were set as follows: VLF (0-0.04 Hz), LF (0.04-0.15 Hz), and HF (0.15-0.4 Hz). Interpolation of the interbeat intervals (R-R series) was set at 4 Hz. Window width for fast Fourier transformation was set at 256 seconds with the window overlap set at 50%.[4]

Statistical Analysis

Data were assessed for distribution of normality and summarized using routine descriptive statistics using mean, SD, 95% confidence interval (95%CI), and standard error of mean (SEM). Differences in HRV by training groups and injured versus non-injured were determined using a 1-factor analysis of variance (ANOVA) and independent *t*-test, respectively. For the non-normally distributed data, the non-parametric Friedman's ANOVA was used for analysis with an alpha level set at $p<0.05$. The SPSS version 24 was used for all statistical analysis with alpha level set at $p<0.05$ (IBM Inc., Chicago, IL).

Figure 1. Subjects Divided into Sports



Results

A total of 48 athletes participated in the data collection. Following the initial data screening, four participants were excluded due to technical difficulty with Kubios data analyses. Data summary was presented in Table 3, and data distribution and outliers for each HRV measures were illustrated in the histogram and box-and-whisker plot in Figure 3. The Kolmogorov-Smirnov normality test indicated that the RMSSD, LF nu, and HF nu were normally distributed. The resting HR data were associated with 3 outliers, which were identified based on the box-and-whisker plot analysis. The outliers were defined as 1.5 times the interquartile range (IQR) of the lower (25%) or upper (75%) quartiles. Of the resting HR data,

two outliers were above the upper quartile and one was below the lower quartile. Two outliers were found in RMSSD data above the upper quartile. No outliers were indicated for the LF nu and HF nu.

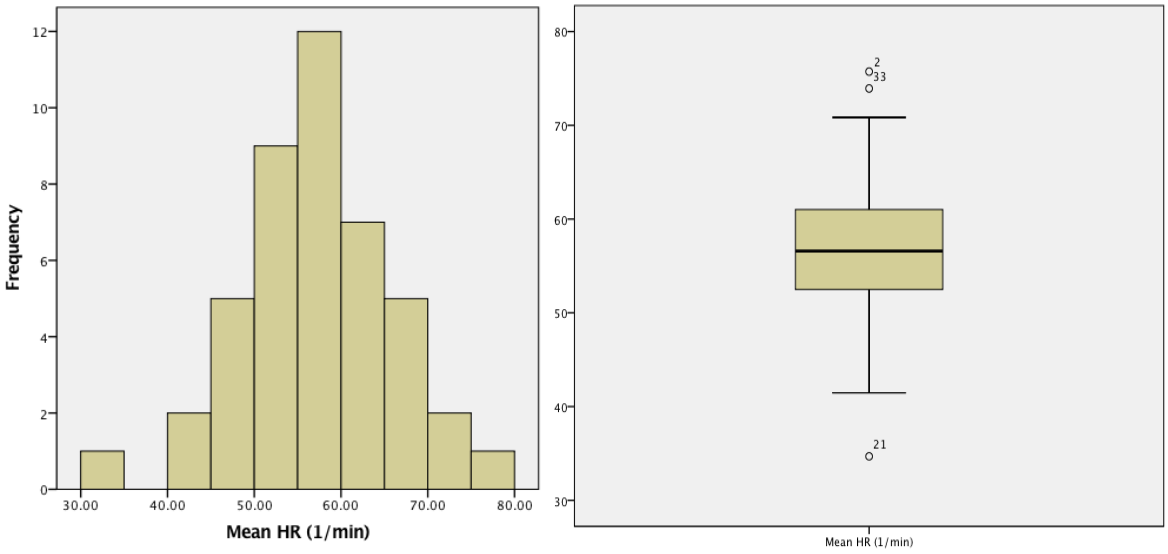
The Kolmogorov-Smirnov normality test indicated pNN50, LF ms^2, HF ms^2, and LF/HF ratio were not normally distributed. The SDNN data was positively skewed and associated with 2 outliers that were above the upper quartile; the data remained non-normally distributed after removal of the outliers. The pNN50% data did not have any outliers, and were associated with bimodal distribution. The LF ms^2 data were positively skewed and associated with 5 outliers that are above the upper quartile; the data remained non-normally distributed after removal of the outliers. The HF ms^2 data were positively skewed and associated with 3 outliers above the upper quartile; the data remained non-normally distributed after removal of the outliers. The LF/HF ration data were positively skewed with 2 outliers above the upper quartile; the data became normally distributed after removal of the outliers. Overall, the frequency power measures (LF ms^2, HF ms^2, LF/HF) were associated with high variability, whereas time domain measures (RMSSD and pNN50) as well as normalized frequency measures (LF nu and HF nu) were associated with low variability.

Table 3: Descriptive Statistics of Heart Rate Variability Time and Frequency Domain Measures for All Subjects

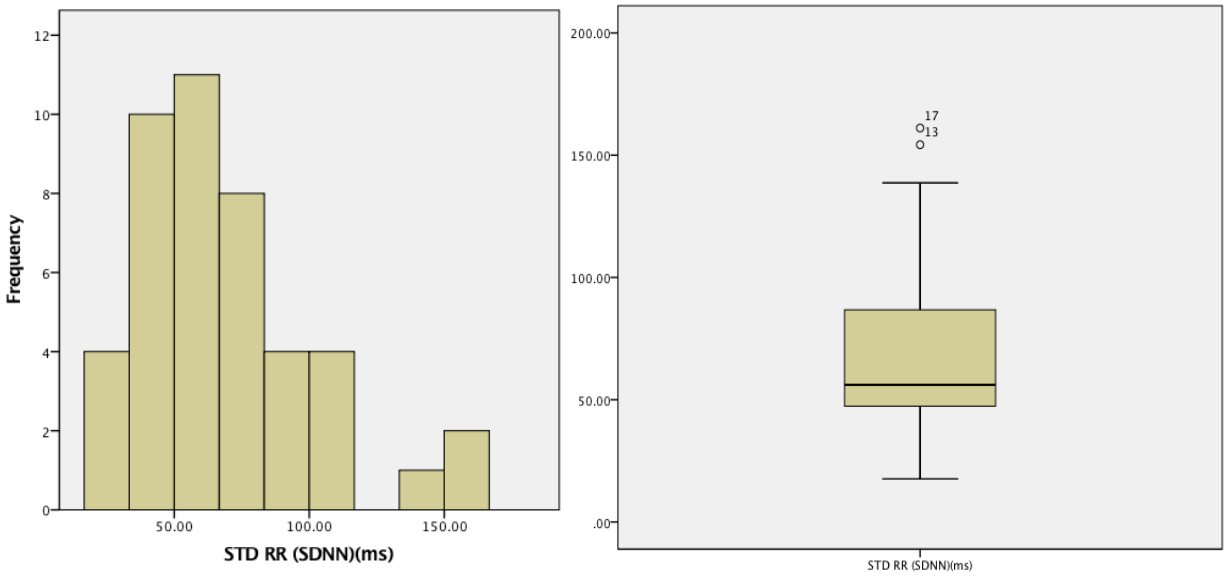
									95% Confidence Interval	
	Mean	SD	Median	SEM	Min	Max	Skewness (SEM)	Kurtosis (SEM)	Lower Bound	Upper Bound
Mean HR (1/min)	56.89	8.68	56.59	1.31	34.69	75.73	-.006 (.357)	.250 (.702)	54.25	59.53
SDNN (ms)	67.64	33.14	56.10	4.99	17.60	161.10	1.04 (.357)	1.04 (.702)	57.57	77.72
RMSSD (ms)	89.84	48.27	84.25	7.28	15.70	223.80	.901 (.357)	.720 (.702)	75.17	104.51
pNN50 (%)	50.16	22.42	56.80	3.38	.30	87.10	-.657 (.357)	-.356 (.702)	43.34	56.97
LF (ms^2)	1731.05	2600.47	927.50	392.03	89	12805	3.44 (.357)	12.452 (.702)	940.43	2521.66
LF (n.u.)	36.67	17.41	35.55	2.62	6.10	69.60	.052 (.357)	-1.041 (.702)	31.38	41.97
HF (ms^2)	3088.84	3080.79	1787.50	464.44	85	12330	1.542 (.357)	1.838 (.702)	2152.20	4025.49
HF (n.u.)	62.96	17.38	63.45	2.62	30.30	93.40	-.021 (.357)	-1.077 (.702)	57.67	68.24
LF/HF	.7211	.55	.55	.08	.07	2.30	1.142 (.357)	1.047 (.702)	.55	.88

Figure 3. Heart Rate Variability Data Distribution and outliers for NCAA Collegiate Athletes (n=44)

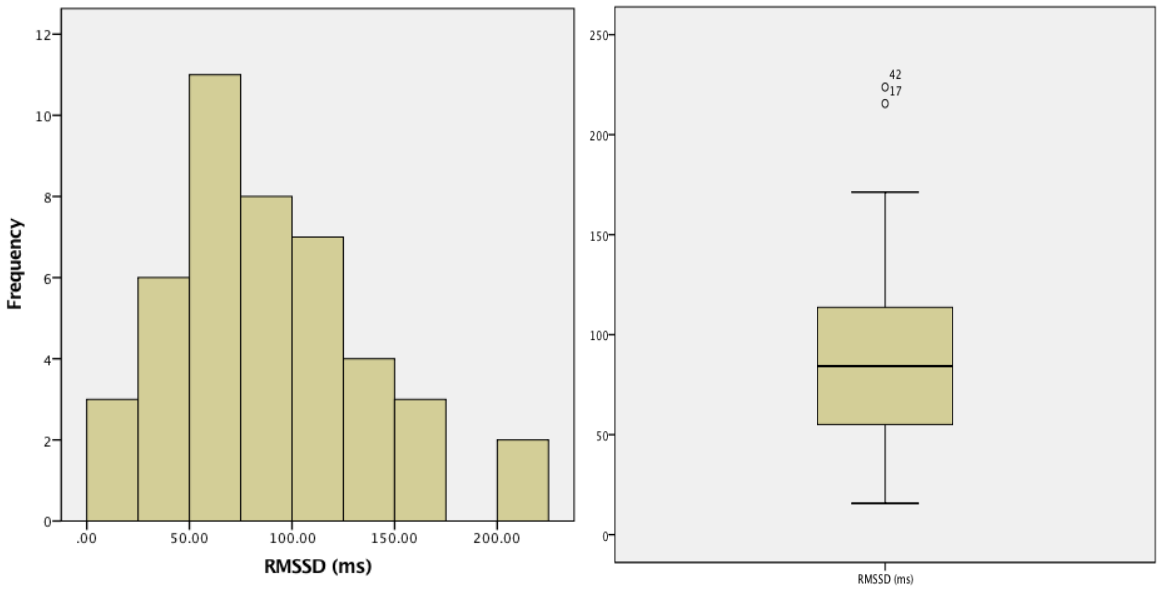
A. Resting HR (mean=56.89 ± 8.68 beats/min)



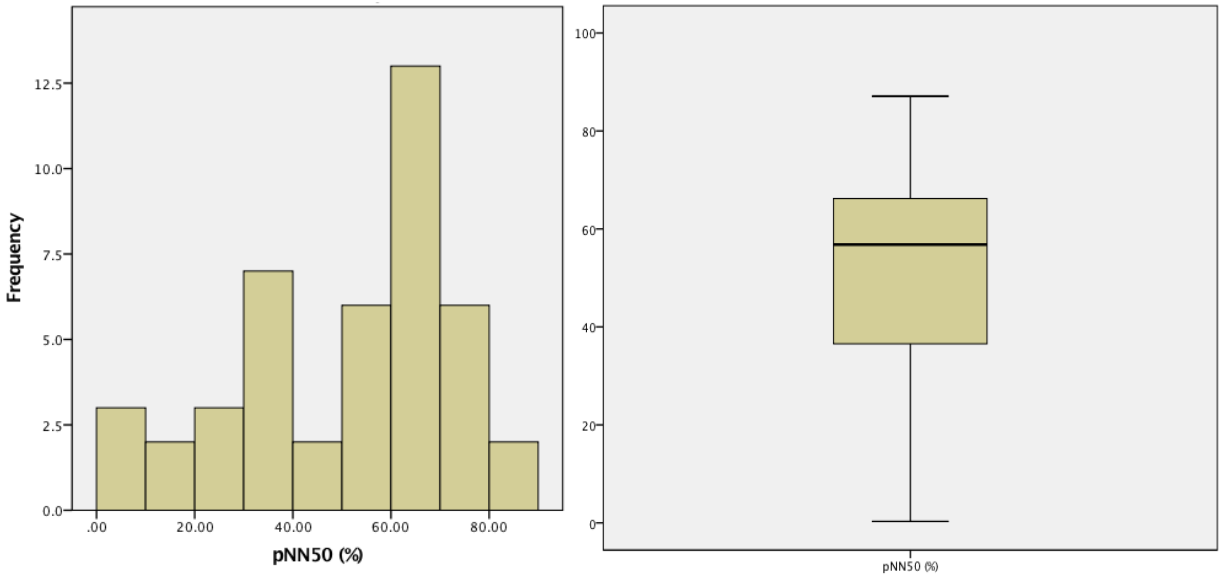
B. SDNN (mean=67.64 ± 33.14 ms)



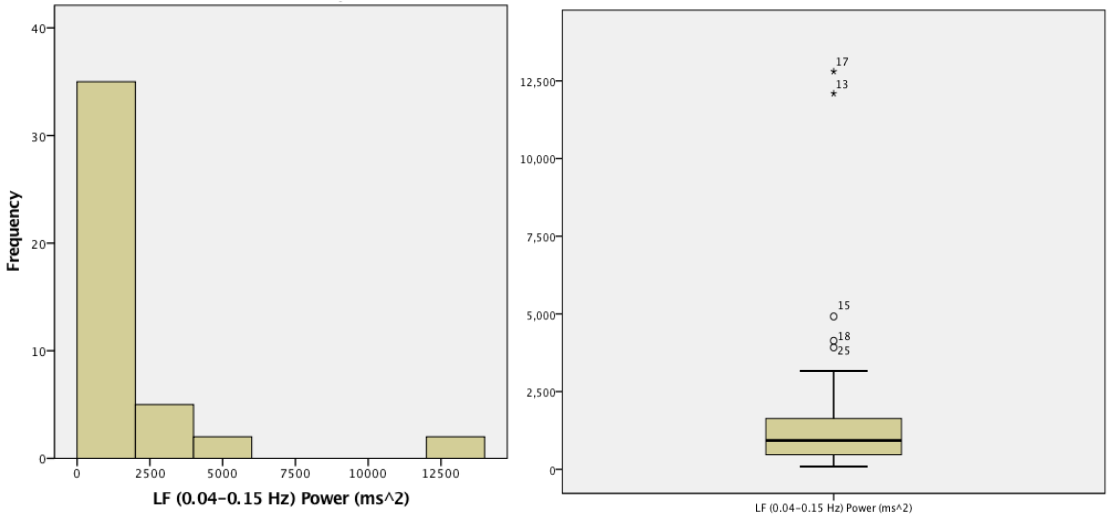
RMSSD (mean=89.84 ± 48.27 ms)



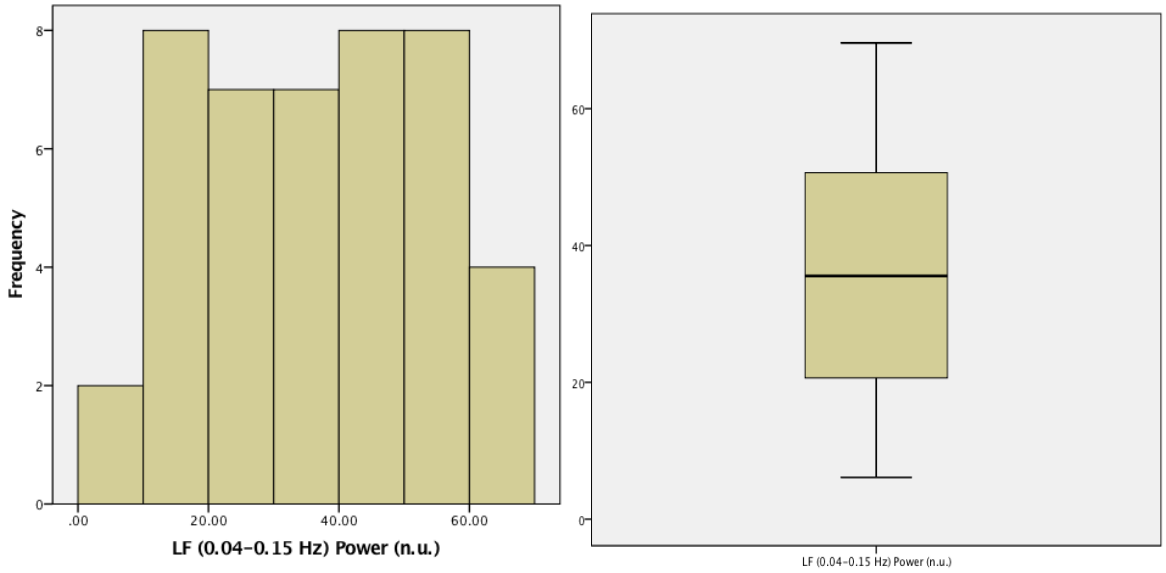
C. pNN50 (mean=50.16 ± 22.42 %)



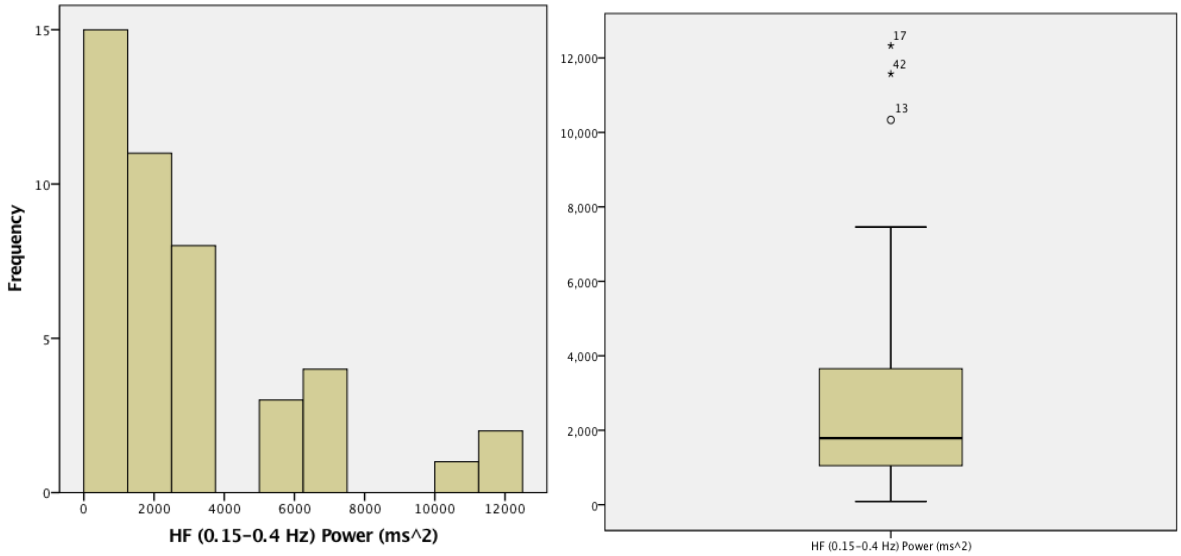
D. LF (mean=1731.05 ± 2600.47 ms^2)



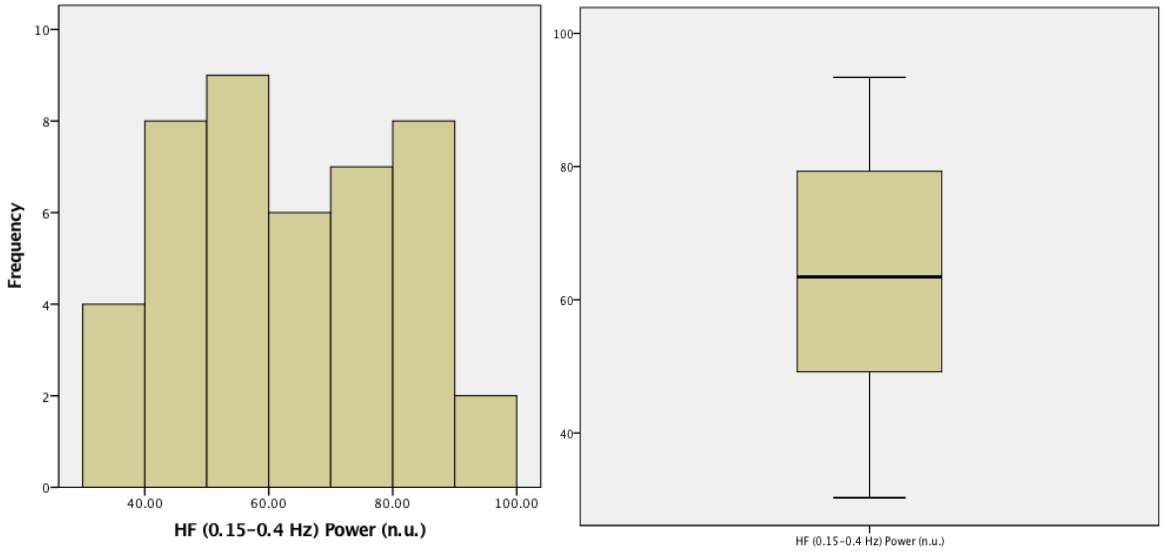
E. LF (mean=36.68 ± 17.408 nu)



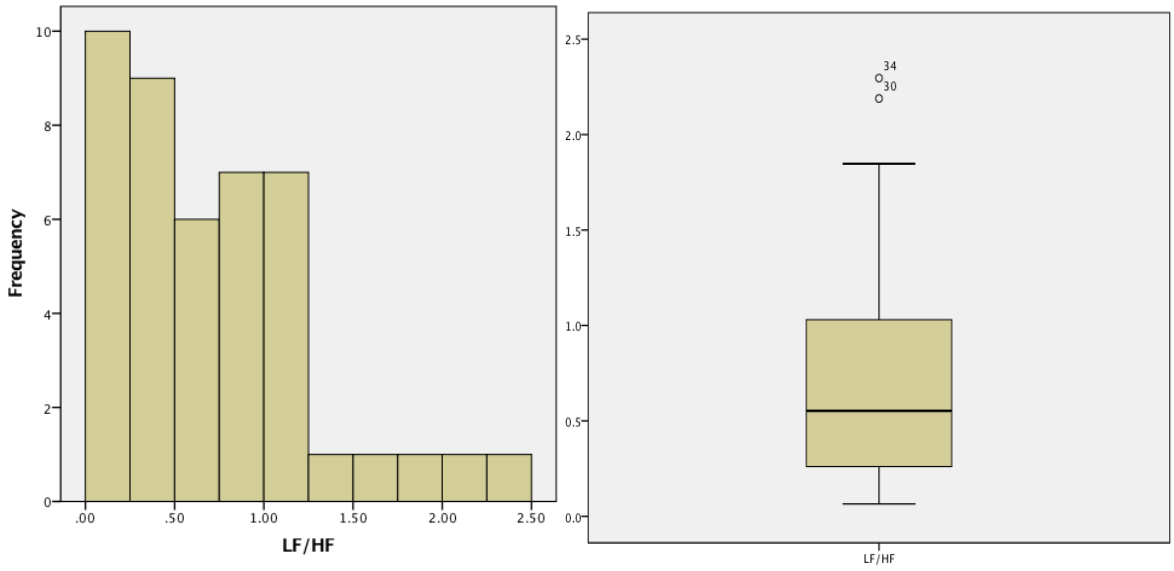
F. HF (mean=3088.84 ± 3080.788 ms^2)



G. HF (mean=62.96 ± 17.39 nu)



H. LF/HF (mean=.72 ± .551 ratio)



BY GROUP

The mean and SD of each HRV measure per group (anaerobic, aerobic, and team sport) are presented in Table 4. There was no main effect for the mean HR (p=.239), RMSSD (p=.376), LF n.u (p=.412), and HF n.u. (p=.341).

Table 4: Comparison of Average Time and Frequency Domain HRV Measures Between Training Groups. (mean ± SD)

	UNIT	Team Sports (n=16)	Anaerobic (n=16)	Aerobic (n=12)	Significance
One way ANOVA					
HR	1/min	57 .23± 6.37	59.10 ± 8.96	53.50 ± 10.47	.239
RMSSD	ms	88.475 ± 43.69	101.756 ± 57.23	75.783 ± 40.15	.376
LF	n.u.	33.43 ± 14.40	35.76 ± 21.12	42.23 ± 15.59	.412
HF	n.u.	66.46 ± 14.41	64.04 ± 20.97	56.83 ± 15.45	.341
Friedman's ANOVA					
LF/HF	ratio	.5728 ± .3494	.7574 ± .6718	.8705 ± .5884	.472
SDNN	ms	74.63 ± 34.75	76.69 ± 36.81	56.02 ± 27.61	.472
PNN50	%	52.038 ± 16.40	53.225 ± 26.23	43.567 ± 24.36	.717
LF	ms^2	1803.0 ± 3070.89	1985.25 ± 2927.02	1296.17 ± 1258.85	.779
HF	ms^2	3074.75 ± 3185.861	3786.44 ± 3455.47	2177.5 ± 2316.30	.472

INJURED VS NON-INJURED

There were six participants' self-reported as injured and remaining 38 participants were considered non-injured. Six non-injured participants were matched with each injured participant based on sport and gender for the analysis. There was no statistical significant differences in injured and non-injured groups in any HRV measures, shown in Table 5.

Table 5: Comparison of Average Time and Frequency Domain HRV Measures Between Non-Injured and Injured groups. (mean ± SD)

	UNIT	Non-Injured (n=6)	Injured (n=6)	Significance
		t-test		
Mean HR	1/min	59.67 ± 7.04	59.31 ± 5.91	.533
RMSSD	ms	83.43 ± 43.38	145.26 ± 73.84	.212
PNN50	%	47.15 ± 21.77	66.60± 26.02	.867
LF	n.u.	38.18 ± 22.93	27.33 ± 15.83	.333
HF	n.u.	61.68 ± 22.78	72.30 ± 15.63	.325
LF/HF	ratio	.84 ± .77	.44 ± .36	.195
		Friedman's Test		
LF	ms^2	1472.33 ± 1354.10	3135.33 ± 4789.33	.414
HF	ms^2	2549.67 ± 2583.26	6465.83 ± 4812.86	.102
SDNN	ms	100.07 ± 46.97	63.73 ± 26.92	.102

Discussion

The current study described the characteristics of HRV in NCAA Division I college athletes, Our data were vastly different from those reported in the meta-analysis conducted by Nunan et al [47]. This meta-analysis was aimed to describe the normative data for healthy individuals, which included all ages and genders. The mean RMSSD (89.84 ms), SDNN (67.64 ms), LF ms^2 (1731.05 ms^2), and HF ms^2 (3088.84 ms^2) were substantially higher while LF/HF (0.7211) was lower in our data compared to those reported by Nunan et al. (RMSSD: 42 ms, SDNN: 50 ms, LF ms^2: 519 ms^2, HF ms^2: 657 ms^2, and LF/HF: 2.8) [47]. The LF nu (36.68%) was lower and HF nu (62.96%) was higher in our study compared to those reported in Nunan et al. (LF nu: 52%, HF nu: 40%) [47]. With 75% of the mean HR of our subjects being less than 60, it could be assumed that the majority of our subjects had physiological parasympathetic adaptation to exercises associated with bradycardia , which could explain the higher parasympathetic measures of RMSSD and HF ms^2 and nu, and lower sympathetic measure of LF/HF in our data [7, 16, 19]. Since LF measures are influenced by both sympathetic and parasympathetic modulations, it could be interpreted that the increase in LF measures in our data are reflective of the increased parasympathetic activities. Our data are in agreement with the previous literatures reporting higher HRV in trained athletes compared to non-trained individuals [48].

The HRV measures in our data were associated with high interindividual variations, which is consistent with the previous literature [36, 47]. However, the data range of frequency power measures was substantially different from those reported by Nunan et al [47]. The ranges for LF ms^2 and HF ms^2 reported by Nunan et al. were 193-1009 and 82-3630 as compared our

data of 89-12805 and 85 – 12330, respectively. In comparison, the range for RMSSD reported by Nunan et al was 19-75ms compared to our data range of 15-223ms. While our RMSSD data range is wider than that of Nunan's, the differences in the ranges for the frequency domain measures, especially on the maximal end, were exponentially larger in our data. [47] Berkoff, et al. examined the HRV in elite aerobic and anaerobic athletes and reported high data variability in the LF ms^2 and HF ms^2 data as indicated by the SD approaching the mean value, while the SD of the RMSSD was approximately half of the mean, similar to our data [36]. While Berkoff et al did not report data range as minimum and maximum values, the large SD indicates high data variability. [36] Although interindividual variation in HRV data were reported to be high in Nunan's meta-analysis, the SD of their data were relatively smaller compared to the data presented by Berkoff et al and in our study. [36, 47] This unique characteristic could be attributed to highly trained athletes.

As training type results in different physiological adaptations, it is presumed that HRV will be different with different sports; however, there was no significant difference between training groups for any of the HRV measures. This is consistent with the findings of Berkoff et al. and Abad et al. who examined the HRV in elite track and field athletes comparing the anaerobic (sprinters and throwers) and the aerobic (distance and middle distance runners) athletes. Both studies reported no significant difference in resting HRV measures indicating type of training did not influence the HRV in highly trained individuals. [36, 48] Conversely, research that examined the influence of training type on HRV has found significant difference following endurance or strength training in participants who were not highly trained. While these non-significant differences between aerobically and anaerobically trained athletes may be unique to highly trained athletes such as Olympic qualifiers and NCAA division I athletes, it could also be

attributed to the high SD for this population. A research study with a larger sample size may be warranted to clarify the influence of the type of training on HRV measures.

Our preliminary analysis of the influence of injury on HRV indicated no significant difference between the injured and non-injured groups; however, this analysis was limited due to a small sample size. In addition, the duration, severity, and type of injury as well as the treatment received were not accounted for in this analysis. Freeman et al. reported that chronic injuries has increased the HR and decreased HRV, [6] while Appelhans et al. indicated no significant influence of acute pain on HRV using a thermal pain stimulation method. [21] Another potential confounding factor with the female population is the effect of the menstrual cycle on HRV which was not controlled for nor documented in this study. As HRV is increasingly used to examine recovery from training it is important to determine the influence of injury to be able to differentiate these conditions. Further study is warranted to examine the influence of acute and chronic injury on HRV levels.

Part II

Literature Review

Due to the lack of knowledge in the association of fatal arrhythmias and either increased sympathetic or decreased vagal activity, the development of quantitative markers of autonomic activity was first experimented. In 1965, Hon and Lee [49] showed the first relevance of heart rate variability (HRV) when they studied how fetal distress was preceded by alterations in interbeat intervals right before there was a change in the heart rate. Then in 1980, the clinical importance of HRV came to light when it was shown to be an independent predictor of mortality following an acute myocardial infraction [50-52]. Continuous Electrocadiograph (ECG) recordings are used to detect the normal-to-normal (NN) intervals between adjacent QRS complexes that result from sinus node depolarization to determine the instantaneous heart rate (HR).

There are two ways to analyze HRV, time-domain and frequency-domain measures. Time domain measures assess the amount of time the beat-to-beat or NN interval moves from one P-wave to the next. Frequency domain asses the frequency of the bands. Time-domain has several different variables: standard deviation of all NN intervals (SDNN), standard deviation of the averages of NN intervals in all five-minute segments of the entire recording (SDANN), square root of the mean of the sum of the squares of differences between adjacent NN intervals (RMSSD), number of pairs of adjacent NN intervals differing by more than 50 ms in the entire recording (NN50), NN50 count divided by the total number of all NN intervals (pNN50). The four most common time-domain HRV measurements that are assessed are SDNN and HRV

triangular index both estimate the overall HRV, SDANN which estimates the long-term components of HRV, and RMSSD which estimates the short-term components of HRV. Because of its statistical properties, the RMSSD method is usually preferred over the pNN50 and the NN50 methods. Though SDNN is among one of the easiest to calculate it is not a statistical quantity because its results are dependent on the length of the recording period. Recordings can also be derived from direct measures of NN intervals or from the instantaneous heart rate, which should be clarified when reporting data. There are three main spectral components when looking at the frequency-domain: very low frequency (VLF) with a frequency range of 0.003-0.04 Hz, low frequency (LF) with a frequency range of 0.04-0.15 Hz, and high frequency (HF) with a range of 0.15-0.4 Hz. Very low frequency is often not used because of its doubting measure when interpreting how the power is distributed among short term ECG's. Frequency domain components are usually in absolute values (ms^2), however, LF and HF are measured in normalized units (n.u.) taking the total power and subtract the VLF component. Different data collections can be compared amongst each other as long as they are of the same time length. Data recordings can be as short as two minutes or as long as 24 hours but the suggested "short-term" recording is five minutes. A recording of at least one minute is needed to assess the HF component and approximately two minutes is needed to access the LF component of HRV. Therefore, five minute recordings of the stationary system are recommended. Many studies have shown that short-term recordings quickly return to baseline after mild exercise, short acting drugs and or low strenuous activity while more intense activity, long acting drugs and more powerful stimulus activities have a prolonged effect to return to baseline. Twenty-four hour recordings allow for more time, along with more complex results when looking at a series of instantaneous heart rates or cycle intervals. Also, due to the time difference, 24-h recordings

seem to have less of a placebo effect when assessing different intervention therapies. Even though the time-domain methods, SDNN and RMSSD, are used to analyze short term recordings, the frequency-domain components usually show more easily interpretable results. The results out of the machine are usually edited manually to get rid of any data that may have been picked up as the wrong wave. Manual editing to remove outliers in the ECG data is preferred over filters which may automatically remove intervals above a specific length leading to potential error as the nature of the data is based on variability. [4]

Heart rate variability is affected by the fluctuation in HR which can be changed by many different physical and mental stressors. Some common stressors that can change HR are illness, emotional stressors such at school work, exercise, breathing, medications, body temperature and cardiovascular diseases. There is also mixed controversy stating whether gender plays a role in the change of HRV. The release of acetylcholine by the vagus nerve mediates the parasympathetic influence on the heart rate by increasing the firing rate of the SA node thereby decreasing HR. The sympathetic influence on heart rate is mediated by the release of epinephrine and norepinephrine leading to an increase in HR [4].

Many environmental and hormonal changes can affect HRV. When analyzing data, accuracy of the data and the environment for testing should be the same for every subject. For example, breathing, medications, and the position of the subject should be the same. Age and gender are factors that may influence HRV so caution should be used when comparing results of different populations. When comparing HRV between sexes, there has been mixed controversy. Some studies show no difference between genders [10, 12, 16], however, some studies showed gender differences [29] . Like most things in life, Age can play a huge role in the decline of all normal body function, including HRV. However, HRV has been shown to improve in an older

population, after different types of exercise. Position of the subject has also been shown to be different when comparing a supine position and a sitting position. Freeman et al.'s study showed a significant difference in baseline HRV in the supine position and the sitting position, however, chronic fatigue or pain could have been contributing factors [6].

In McCarty et al.'s study, it is explained that if breathing is not self-determined, and instead is increased, it can affect the parasympathetic nervous system. Activation of the parasympathetic nervous system will affect the LF/HF ratio. The HF range is from 0.15Hz and 0.4Hz which occurs between 2.5 seconds and 7 seconds. The LF range is between 0.04Hz and 0.15Hz and occurs between 7 and 25 seconds. The HF band is commonly known as the respiratory band because it correlates to the variation in HR and the respiratory cycle. The LF power primarily reflects the baroreceptor activity at rest. Slow breathing can actually cause an increase in the parasympathetic activity which in return can affect the LF:HF ratio. However, HF power can be associated with stress, panic, anxiety or worry. Unlike the FD measures, time domain can be compared between different researchers if the recordings are the exact same length of time. It was once thought that the heartbeat was monotonous rather than highly variable, which is seen better on a beat-to-beat basis. If the heart is too chaotic, such as an arrhythmia, it can affect the physiological function and energy utilization. However, if the heart is beating too slow, it can show chronic stress, pathology, or inadequate functioning throughout the bodies system. Understanding what HRV is and the components of it gives a better insight on how to use it in practical situations. [34]

Akselrod et al.'s examines the beat-to-beat analysis fluctuations of heart rate and how quickly HRV can react in conjunction with the cardiovascular control system. Seven trained, conscious, unanesthetized dogs were studied to get a clear look at the measurement of the power

spectrum fluctuation. Different cardiovascular control systems were selectively blocked by specific pharmacological agents. Prior to using the pharmacological agents, 20 minutes of baseline data was first collected. The parasympathetic blockade showed the mid- and high-frequency peaks were terminated and the low-frequency peak is decreased. The sympathetic blockade and parasympathetic blockade lead to a metronome-like heartbeat and showed no heart rate fluctuation. This indicates that the parasympathetic nervous system mediates HR fluctuation at frequencies corresponding to mid- and high-frequency peak of the power spectrum, whereas both parasympathetic and sympathetic may mediate LF fluctuation. Since pharmacological blockades show a significant outcome on a beat-to-beat level in dogs, taking medications that affect the nervous system in anyway could show a significant difference in humans HRV. [35]

Tousignant-Laflamme et al. studied the correlation between pain perception and HR. The current assumption is that when pain increases, HR will also increase, which should then show a corresponding decrease in HRV. Skin conductance was included to be able to look at the peripheral sympathetic activation and how it relates to pain perception. A total of 39 healthy volunteers, 19 men (mean age 23.1+/-4.4) and 20 women (mean age 26.5+/-7.2) were included. A two-minute baseline was first recorded, then a pretest of hand immersion was done for one minute to familiarize the subjects with the procedure. After a five-minute break the subject put their hand, up to their wrist, in hot water for two minutes. Perceived pain was rated every 15 seconds and a separate VAS was used for pain intensity and unpleasantness. Skin conductance levels (SCL) were also obtained on the middle phalanx of the first and second fingers of the opposite hand. During this hand immersion test, the subject pain perception and unpleasantness increased as the time increased. There was no significant difference in gender on pain intensity or unpleasantness. The first 15 seconds of immersion produced a 7% increase in HR over

baseline, (77.04+/-10.46 beats/min) and a second increase was seen after 120 seconds of 11%. When comparing percent increase of HR and mean pain perception, women showed a nonsignificant correlation. However, men did show a significant correlation in increased HR and mean pain perception. There is a very short time where an acute reaction to a painful stimulus will adjust the HR, which is why the athletes in the current study will need to be in a comfortable position before the data is collected. As a limitation this study was only recorded for two-minutes but did show that even though both men and women had a HR increase, men with the largest HR increase reported higher levels of pain intensity and unpleasantness. The women with the highest HR on average reported less pain. This could show that women have a higher pain tolerance than men when it comes to pain intensity and unpleasantness but this is the only study found to report this difference. [29]

Per the American Psychological Association, there are many different types of stress including acute stress, episodic acute stress, and chronic stress. Acute stress can include emotional distress, acute muscular disorders, and transient over arousals, which can increase BP. Episodic acute stress is your common "worry-warts" or the person who thinks everyone is out to get them. Chronic stress is something that wears and tears at you daily for a long period of time, day after day and/or year after year. Chronic stress can either be a childhood memory that stick with you, a long going injury or again, anything lasting for a long period of time. With cognitive stress, HRV did not show a significant difference between rets and stress but it showed a difference in LF/HF ratio. Athletes have a high pressure to succeed at the Division I level which can put added stress on the athlete. If an athlete gets hurt, that is just another thing to add to the list. One study showed that even with minimal tissue damage there was no difference in HRV when comparing an athlete to a non-athlete's HF HRV. However, athlete with chronic a chronic

injury may have a decreased HRV.

William et al. showed that subjects with higher anxiety and difficulty with emotional clarity on the self-reported difficulties in emotion regulation (ER) will be subject to lower vagal mediated HRV. When looking at the association of resting vagally mediated HRV (vmHRV) and self-reported difficulties in emotion regulation (ER), we need to take into consideration the additional variables of rumination and anxiety and how they can be a link between the ER and Inhibitory control system. A diverse population of 183 students (98F, 18-35 years old, mean 19.34) was examined. Vagally mediated HRV results were reported using RMSSD as it is highly correlated with HF power ($r=0,90$, $p<0.01$). The *Difficulties in Emotion Regulation Scale* (DERS) was used to assess perceived difficulties in ER. Significant negative correlation between resting vmHRV and self-reported difficulties in everyday ER was reported for the first time according to the DERS. Greater difficulties in ER and lower vmHRV were shown to be associated with greater anxiety and rumination. However, when psychological covariates were controlled, the subjects had lower resting vmHRV and greater difficulties with ER. The DERS breaks down the difficulties into scales to better understand the relationship. Subjects that had a lower vmHRV reported greater difficulties with emotional clarity and impulse control. Subjects that scored less on the GOAL subscale, described as a goal oriented behavior, showed more negative emotions and were more likely to experience low HRV compared to those who scored higher. Resting vmHRV is related to the individual's everyday perception of difficult and how they regulate their emotions. Subjects with higher anxiety and difficulties with emotional clarity will be subject to lower vmHRV. [53]

McDuff et al. focuses on how cognitive tasks have an impact on HRV, especially HF when compared to a control group. The gold standard for HRV measurement is the

electrocardiogram (ECG). This study uses photoplethysmography (PPG), which has been shown to be comparable to measures of HRV derived from an ECG. The PPG is a low-cost and non-invasive technique for measuring the cardiovascular blood volume pulse (BVP) through variations in transmitted or reflected light. The subjects included 10 healthy, 7 males, ages 18-30 with different skin colors. The subjects first looked into the camera for 2 minutes to get a baseline along with a chest strap to measure the breathing patterns. A costlier digital camera and computer system are used to look at the change in the 10 subjects faces when under a cognitive stressor. Then the subjects were asked to perform an arithmetic task silently. Starting with the number 4000, they were asked to count down by 7's as quickly as possible. To make this a more stressful task, they were told they were competing against the other subjects to reach the lowest number. The camera was recording the facial movement and BVP by transmitted or reflected light. They found higher LF/HF ratios when the subjects were under cognitive stress. However, HR was not significantly different between rest and stress. Cognitive stress does show a difference in LF/HF ratio but HR did not show a significant difference. This may be a limitation when looking at the HRV of athletes who score high for non-training stress, such as academic stress, in the REST-Q Sport. [54]

Appelhans et al. studied how pain can affect HRV through thermal pain sensitivity. Pain's unpleasantness is known to play an important role in maintaining homeostasis. Fifty-nine, right handed, undergraduate healthy students (37 women, 22 men) were studied. The subjects first laid down supine for seven minutes while breathing normally. For the last 5 minutes at this supine position, the HRV was recorded. The subjects then sat up and placed their non-dominate hand on a cold plate. The temperature gradually decreased and the subjects had to rate the pain they were feeling for three minutes. They could warm their hand on a heating pad for three

minutes and then their hand was to be put back on the cold plate. Greater LF was shown to be associated with lower temperatures, where participants first identified noticeable pain and moderate pain. Also, greater resting LF significantly predicted lower ratings of four degrees' Celsius pain unpleasantness and higher (colder) thresholds for noticeable and moderate thermal pain. High frequency was not a significant predictor of any measure of pain sensitivity. Since HF is known to be driven by the respiratory sinus arrhythmia, this could be the reasoning that HF was not consistently associated with the pain sensitivity. Though this study only involved minimal potential for tissue damage, this could mean that an injured athlete may show no difference when comparing a non-injured athlete's HF HRV. [21]

Freeman et al. studied how chronic fatigue syndrome (CFS) affects the ANS. Chronic fatigue was characterized as having a disabling fatigue accompanied by post exertional malaise, musculoskeletal pains, headaches, sore throat, tender cervical or axillary lymph nodes, concentration difficulties, memory impairment, and sleep disturbance. Chronic fatigue syndrome subjects were 38.9+/-2.1 years of age and the controls' age ranged from 37.9+/-1.8 years including 23 subjects total. Subjects taking any medication that would affect the ANS, discontinued the medication at least 48 hours prior to doing a battery of autonomic tests. There were significant differences between the two groups in baseline HRV in the supine position and sitting position potentially indicating that pain is the cause in the different HRV. The CFS group showed an increase in both the baseline and maximum heart rate on standing and the tilt test. Most results showed CFS could lead to cardiovascular deconditioning. Since musculoskeletal pain is a symptom of CFS, athletes with chronic injuries would be more susceptible to having an increased HR and decreased HRV. One limitation to this study was that most CFS subjects reported a viral infection preceded their CFS, which can affect the parasympathetic and

sympathetic nervous system. [6]

An increase in the sympathetic tone activity has shown to cause a rise in HR. On the other hand, the activation of the sympathetic tone will then trigger the parasympathetic tone to react as an antagonist to establish a homeostatic balance. This can be displayed when looking at an athletes HR versus their HRV. Athletes are known to have a lower HR and a higher HRV. A homeostasis imbalance such as over-training (OT) has not been shown to affect HRV, however HRV is shown to be higher in the over-trained population when compared to a non-fatigued group. This shows that fatigue rather than the metabolic stress during an intense exercise could be the reason for the delay in the heart rate recovery. There was no difference shown between sleep and awake measurements in OT and non-overtrained athletes.

Lepretre et al. questions the effects of fatigue and metabolite accumulation on post-exercise parasympathetic reactivation. Fatigue is defined as a time in which the body is unable to maintain a given muscular force at the end of a supramaximal exercise leading to a decrease in performance. It was thought that a delay in parasympathetic reactivation and longer heart rate recovery were induced by metabolic and ionic accumulation during long sprints rather than being affected by exercise intensity. Nine males and two females (mean +/-SD age; 21.6 +/-6.3 years) were tested on three outdoor track events. Five-seconds post-exercise, beat-to-beat HR was measured for a seven-minute recovery period. No significant HR amplitude was found during recovery. However, LF and RMSSD were influenced by the recovery time ($P<0.01$). Fatigue was demonstrated by the inability to maintain the same running speed. Longer heart rate recovery values were found after the 400m bouts compared to the 300m. Fatigue did not have a major effect on the RMSSD though parasympathetic reactivation seemed to be related to high intensity exercise. Both LF and HF were lower after the 400m compared to the 300m running test but

exercise has not been shown to influence HRV recovery. Even though the pace was the same for the exhaustive and the non-fatigue running exercise HRV remained lower for the exhaustive running test. The effects of metabolic status post-exercise response without exercise intensity in healthy well-trained athletes could strengthen this study. [55]

Hynynen et al. studied the autonomic imbalance in overtrained athletes during night sleep and after awakening with HRV measurements. Twelve severely overtrained (25+/-7) and 12 control subjects (24+/-5) were tested. Overtraining was diagnosed by a doctor and the athlete had to meet three different criteria: (a) had suffered from an unexplained decrement in physical performance and fatigue even after a recovery time of at least 3 weeks, which was verified by carefully interviewing the athletes and, in some cases, also their coaches; (b) had been examined to be otherwise healthy with a clinical examination and several laboratory and physiological measurements; and (c) had increased their training volume and intensity progressively for up to 6 months before the overtraining symptoms and had continued training with the symptoms without sufficient recovery. The subjects first came to the lab and got RRI's collected, and the nocturnal sample was collected. Then subjects were given take-home instructions and asked collected five minutes of resting data upon waking. Between the two groups, no differences were found in the average nocturnal RRI, HRV or urine stress hormone. However, SD of RRI and LFP after awakening were lower in the overtrained athlete then the control. Though night/sleep recordings seem to take out the environmental factors, it seems to show no difference in the measurements when sleeping. [56]

Mourot et al. found that HRV reflects cardiovascular control, exerted by both parasympathetic and sympathetic nervous system and has been used to evaluate modifications of autonomic functions due to acute exercise or training. There were seven athletes (five women

and two men; two endurance runners, four cross-country skiers, one motorcyclist) diagnosed as suffering from OT syndrome who were compared with endurance trained subjects (four women and four men) who performing regular physical training for at least three years (T) and eight sedentary control subjects (four women and four men) (C). Overtraining in this study is defined as a condition of chronic fatigue syndrome and over training syndrome. All participants, while performing their sport, stated that they felt as if they were below the acceptable level and were having difficulty maintaining their training program. Three time-domain measures were used as quantification of total variation in HR during the respiratory cycle: SDNN, rMSSD, and pNN50. In the three groups, the 60-degree upright position significantly decreased the length of mean R-R interval. An important decrease in SDNN and TP was observed during standing in T (P<0Æ05) compared with OT (NS) and C (NS). The rMSSD, pNN50, HF, SD1 and SD1n decreased significantly (P<0Æ05) during 60 upright position in C and T but not in OT and LF increased in C and T but decreased in OT (NS). Thus, in the 60-degree upright position, a decrease in HRV and parasympathetic activity was observed with the three methods of HRV analysis. The decrease was more important in T than in C and OT subjects, T exhibited higher SDNN, TP, LF, SD1, SD1n, SD2 and SD2n than the other two groups in the 60-degree upright position. During supine rest and 60-degree upright positions, HRV of OT was similar to that of C with a predominance of sympathetic activity whereas T had a higher HRV with a marked predominance of parasympathetic activity. Altered catecholamine release (reduced adrenaline, less reduced noradrenaline and dopamine excretion) has been shown in overtrained athletes indicating an adaptation of the sympathetic nervous system. The T group exhibited the greater mean R-R interval, which indicated a sympathovagal balance shift to parasympathetic predominance. The standing position was accompanied by a decrease in parasympathetic

indicators (i.e. rMSSD, pNN50, TP, HF, HF/TP) that occurred together with increased sympathetic indicators (LF/TP, LF/HF), indicating a shift in sympathovagal balance towards sympathetic predominance. However, the magnitude of these changes was higher in T suggesting a higher reactivity of the cardiovascular regulatory system to postural changes. Nevertheless, during standing, T always had higher HRV than C and OT. In standing position and in the OT state, as the parasympathetic activity decreased and sympathetic activity increased. The distinction between overreaching and overtraining (also called short-term and long-term overtraining, respectively) was not clearly established. [19]

Physical activity has a significant influence on HR, which in turn would influence the HRV and show an increase in HRV on the athletic population when compared to non-athletes. While a single day of training has not been shown to change HRV more than the smallest worthwhile change, a clinically meaningful change, an eight-week training program can show a change. Different types of training can affect the HRV differently however, different distances at the same intensity, showed no difference in HRV. In addition, exercise can also affect the older population and as decreased HRV is a sign of a cardiac event, it is possible that training can help improve cardiac function. When comparing HRV between any athletic population and "normal" population, the athletic population has a higher HRV. This could mean that they have more vagal activity. Though how much higher their HRV is unknown, comparing between sports can show which sports are more likely to have higher HRV.

Berkoff et al. studied elite track-and-field athletes and compared HRV between different events. This study looked at 145 (male=87; female=58) elite track athletes who were competing in the U.S.A. Track and Field Olympic Trials. The subjects were lying supine and a resting ECG was collected for five-minutes. The groups were first divided into two groups: power and

endurance but were later dived into five groups due to no significant differences found. The five groups included a distance group, a field group, a power group, and a strength group. Though unexpected, there was no significant difference between the greater divided group either. The only significant difference that was found in this study was between males and females. The difference was found in LFnu (men>women), HFnu (women>men), LF:HF ratio (men>women), and PNN50 (women>men). Whether an athletes sport is considered aerobic or anaerobic, training at the Division I level and the elite level can frequently include both. This could be the explanation as to why there is no difference between groups. [36]

Hautala et al. examined simultaneously measured HRV and objective physical activity (PA) during waking hours. The population was healthy (n=45) males (n=21; age 35+/-3) and female (n=24: age 34+/-4). The R-R intervals were measured over a 24-hour period with the mean PA time being 15:44+/-1:01h during waking hours. This study showed no difference in males versus females. There was a significant correlation between PA and HR (r=0.64, P=0.021) and between PA and sample entropy (r=-0.55, P=0.022). Physical activity was shown to have a significant influence on HR, SD1 and the complexity properties of HR. The outcomes showed that exercises have an evident effect on HRV which could be seen in the athletic population. [12]

Nunan et al. stated that resting HRV could be a great tool to assess the chronotropic change when you exercise. The aim of this study is to see if a short-term HRV recording could predict the HR response before, after and during a GXT. Thirty-three healthy volunteers, including 19 males, with a mean age of 41.5 (20-63 years old) participated. A 10-minute supine baseline HRV was recorded, then a GXT was performed. After the GXT was performed, a 10-minute seated HRV was recorded. Heart rate recovery was the only HR parameter that significantly correlated with resting HRV. Subjects with resting HR averages below 75 beats per

minute showed higher variability in LF (ln ms^2) (6.5+/-0.8), HF (ln ms^2) (6.6+/-1.0), and LF:HF (ln) (0.5+/-0.7) than subjects with HR higher than 75 beats per minute LF (ln ms^2) (5.7+/-0.6), HF (ln ms^2) (4.9+/-1.0), and LF:HF (ln) (0.9+/-0.6). No significance was found when looking at HRV and its ability to predict HR and cardiorespiratory response to GXT. This does however, show that the lower HR you have, the higher the HRV which is important to consider in a highly trained population that is typically bradycardic. [8]

Goldsmith (1992) et al. studied the comparison of normal R-R (NN) intervals in athletes with high levels of endurance training and untrained subjects by looking at the spectral component of heart period variability. The subjects include eight untrained and eight endurance trained healthy men ages 24 to 38. Endurance trained is classified as having a maximal oxygen consumption (VO2 max) of >/= 55 ml/kg per minute. Untrained is classified as someone who hasn't participated in any regular exercise on the past year and had a VO2 max that was </= 40 ml/kg per minute. The trained subjects showed significantly longer NN intervals ($p<0.005$) during the day and night, greater beat to beat variable, and all other measures of HRV showed to be higher in the trained population. The HF power, which shows a pure index of vagal modulation, was 4.2 times greater ($p<0.005$) in the trained men than in the untrained men. The HF increase in both groups during the night. Low frequency was 2.8 times greater in the trained men than in the untrained indicating a significant vagal influence. The bradycardia in the trained population points towards greater parasympathetic activity. While the vagal tone is higher at night for both groups higher parasympathetic activity was shown during the day and night for the trained group. The increased LF power over 24-h in the trained population indicates parasympathetic dominance, suggesting that exercise training may serve as a nonpharmacological approach to modifying the vagal activity. [11]

Imai et al. states that during exercises, the heart rate increases through a combined effect of both the sympathetic activation and a vagal withdrawal. The purpose of this study was twofold: 1) to derive a vagally mediated component from the beat-by-beat heart rate decay after exercise by using pharmacologic autonomic nerve blockade in normal volunteers; and 2) to investigate alterations of vagally mediated heart rate recovery in patients with chronic heart failure and in well trained athletes by using the derived index. Heart rate recovery after exercise is delayed in patients with chronic heart failure and these patients also have shown altered vagal activity. Whereas, well trained athletes are shown to have increased tonic vagal activity. Eight sedentary normal male volunteers, age ranging from 28 to 39 (mean 34) were subjected to pharmacological autonomic blockade challenges. To investigate changes in vagally mediated heart rate recovery in patients with chronic heart failure and in athletes, the subjects were divided into two groups, 20 men with chronic heart failure (heart failure group) and 9 male cross-country skiers trained for at least two years (athlete group). Twenty and 11 normal sedentary men formed age-matched control groups for the heart failure group and the athlete group respectively. The athlete group's end-diastolic left ventricular dimension was only slightly larger, but still statistically significant[7] compared to that of the control group. A limb-lead ECG recorded the heart rate from rest to 120 seconds (s) after cessation of exercise. Two linear components were observed at four levels without atropine, an initial rapid decrease and a subsequent slow decrease. The breakpoint of 32 decay curves obtained from the four levels in eight subjects occurred between 20 and 40 s after exercise. However, the initial rapid decrease disappeared with atropine or atropine plus propranolol administration. This study used linear regression analysis on three sections of the 32 individuals heart rate decay curve to find the best fit for the initial rapid decrease. The slope on the regression line was almost identical between the sections

from 0 to 30 s but significantly less steep in the section from 0 to 40 s (0 to 20 s, -7.8+/-1.8; 0 to 30 s, -7.6+/_1.6; 0 to 40 s, -6.0+/-1.4 x 10-3/s; 0 to 30 s vs. 0 to 40 s, $p<0.05$). Compared with the value during the baseline exercise, exercise heart rate was significantly increased by atropine administration, decreased by propranolol or concomitant administration of both drugs. Similar results were noted in systolic blood pressure. The 30 and 120s data were unaffected by propranolol, but they were markedly prolonged by atropine administration. There was also a significant correlation between 30 and 120 s that indicated both were vagally mediated. Vagally mediated heart rate recovery is accelerated in well trained athletes but blunted in patients with chronic heart failure. Vagally mediated heart rate recovery after exercise is accelerated in athletes while patients with chronic heart failure have decreased tonic vagal activity and impaired baroreflex sensitivity as compared to athletes. This may be a physiologic adaptation in the athletes which allows for rapid heart rate recovery after intense exercise. [7]

Uusitalo et al. studied the influence of a 1-year training program on HR and BP variability and the reflecting changes in the ANS. The 112 male subjects, ranging from 53-63 years of age, participated in the training program that included endurance exercise for 30-45 minutes three times a week for the first three months before the endurance and intensity of the exercises was increased to 45-60 minutes five times a week. When being tested, they only rested for two minute in the supine position before the HRV was measured. During the study, there was no significant difference in the HR or BP in the supine position. However, there was a significant change in RRI very low frequency power (VLFP) which increases in the EX group and decrease in the CO group. Depending on what parameters are used, the effect on cardiac autonomic function can be quite small (up to 15% in average) when looking at HR during exercises versus at rest. However, the training program group did show a positive change in the respiratory

compensation threshold. This study did have a very short length of recording (5 mins), which could easily be a limitation. It did show however, that the exercise group did seem to stop the declining tendency of HRV that could normally be seen. A limitation of this study is that they only did recordings for two minutes. [15]

Brown et al. studies older endurance-trained Master athletes (7M; age mean+/-SD 52.1+/-3.3; 6F; 50.5+/- 2.9) HRV post-exercise. The subjects were first put through a bike ergometer testing protocol. The protocol was stopped after the subjects' blood lactate level was above 4.5 mmol and the subject underwent a 10-minute active recovery. The pre-exercise HRV was significantly higher compared to post-exercise, with a shift towards lower r-r intervals in the post-exercise period. The post heart rates were higher than the pre-exercise (83.1+/- 8.9 vs. 57.9+/- 6.2 beats/min; $P<0.001$), which would explain the difference in HRV. There was no difference between males and females possibly due to the small sample size. In the older population, a key part in the autonomic control of tachycardia lies with the HF component recorded post-exercise, which shows parasympathetic withdrawal. There was a reduced variability in the HF following the 10-minute active recovery. A decrease in HRV during recovery was recorded for the older endurance trained Master athletes. This study shows that HRV can be influenced by exercises, even in the older population. [10]

Pluim et al. investigated how HRV correlates in cyclist with exercise induced left ventricular hypertrophy as compared with a non-athletic healthy population. Twelve highly trained male subjects mean age 41+/-10 years and 10 male control subjects mean age 46+/-9 years underwent a 24h ECG monitoring with the time domain and frequency domain measures of HRV taken from 6 hours of the sleep period to avoid daytime HRV owing to the higher activity level of the cyclist. The resting HR and the HR during atropine-dobutamine stress was lower in

the cyclist. The left ventricular mass and left ventricular mass index was higher in the cyclist when compared to the control group. Reflective of the lower HR, the HRV was much higher in the cyclist then the control group. A higher SDNN is indicative of an intrinsically higher HRV in athletes, which could also show an increased vagal control in the cyclist. This in turn could mean that the more athletic population has a decreased chance of sudden cardiac death. [57]

Levy et al. studies the effect of endurance training on HRV in younger (n=11, mean age 28, range from 24 to 32 years old) and older healthy men (n=13, mean age 68, range from 60 to 82). There were also 10 healthy young subjects with a mean age of 25 (ranging from 21-29 years old) who received atropine during rest to assure that their way of measuring HRV decreased appropriately during withdrawal of the parasympathetic tone. A total of 6 months of exercise training was completed with the training beginning at a 50%-60% of HRV based on the individual's Bruce protocol results for VO2max. Training then increased to 80%-85% of HRV by the fourth month. First, HRV was recorded for 30 minutes in the supine position before exercise testing or before the atropine injection. Atropine did show an increase of HR by 45 beats/min and the HRV decreased exponentially (r=0.93). Supine BP was higher in the older subjects while at rest for the baseline recording but both the younger and older populations had a significant reduction in HRV after the onset and throughout exercise. Even though both the younger and the older groups started training with similar resting HR levels, older subjects began with a 47% lower HRV. This decreased parasympathetic activity at rest is typically associated with a lower HR. Exercises can affect the HR and HRV in people, no matter the age, but is shown to have less of an effect once age affects the cardiovascular system. [58]

The aim of Lovallo et al. was to verify the systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MBP), HR behavior and the HRV after sessions of

aerobic exercise and resistance training combined in different orders. Though it is possible to perform both aerobic and resistance training in the same session, post-exercise hypotension (PEH) seems to be different in aerobic exercise due to the physiological mechanism. In this study nine men (24.8+/-1.1 years), physical activity, normotensive, non-smokers underwent multiple testing sessions. The first day anthropometrics were taken then the subjects were set up on a bike protocol. The second day, at least 48h after day one, one repetition maximum (1RM) were performd in various exercises. On the third and fourth day the subjects either did aerobic exercise followed by resistance training (AER) or resistance exercise followed by aerobic exercise (REA). The SBP and DBP were calculated before exercise and after exercise (60min in 10min intervals). The MBP was calculated by MBP=DBP+[(SBP-DBP)/3]. The HRV was continuously monitored before, during, and after exercise with a polar HR monitor. Although there was a HR increase after the ERA session, there were no alterations of the LF and HF components of HRV. This HR behavior may be associated with the increase in the sympathetic activity and with the decrease in the parasympathetic activity of the heart, mediated by the baroreflex control in atrial to compensate for the BP decrease after exercise. There were no differences in HRV between the rest and recovery period or between the experimental sessions. The main findings of this study were that the AER session reduced SBP, DBP and MBP in some moments. Also, the REA session promoted minimum alteration in the pressoric values. No differences were identified between the ERA and REA sessions. [14]

Kaikkonen et al. wanted to find out the HRV dynamics, mainly focusing on vagal reactivation during immediate recovery after different constant-speed endurance activities. The effects of post-exercise HRV when there is increased intensity and prolonged running distance was also examined. Metabolic functions are thought to be elevated more during high-intensity

exercises because heart rate is known to increase more during high-intensity when compared to low intensity exercises. Thirteen sedentary women (mean age =35+/-3) participated in this study. A graded maximal treadmill test was used to determine the running speeds based on the individual results. Subjects then performed two low-intensity exercises, two moderate intensity exercises, and one high-intensity exercise on the treadmill in random order. Each test started with a 5-minute sitting measurement of RRI's. After the exercises a 30-minute seated passive recovery was performed. At rest, there were no shown differences between HR, TP, or HFP between the exercise sessions. The end-exercise values between the 3 different intensities showed that the HR was lower and TP and HFP were higher at the end of the low-intensity exercises when compared to the moderate and high-intensity exercises at the same distances. There was no difference in the two low intensity exercises even though one was double the distance. This was the same for the moderate intensity exercise during the last minute of the exercise. The HR was higher after the moderate-intensity and high-intensity exercises then the low-intensity exercises throughout the entire recovery phase. The HFP was lower after the moderate-intensity and high-intensity exercises when compared to the low-intensity exercise during each minute of the 5-min recovery. The HR was higher and the TP and HFP were lower after the high-intensity and moderate-intensity exercises when compared to the low-intensity. This shows that the intensity of exercise can affect the HRV in many different ways. [13]

Vesterinen et al. used HRV to determine whether individualized training programs would show a greater adaptation in training. This study included 20 males and 20 female subjects (n=40) with at least two years of regular training in running. The subjects were put in two different training periods: four-week preparation (PREP) and an eight-week intensive training period (INT). If the subject's $RMSSD_{7D}$ fell outside of the smallest worthwhile change (SWC)

the subjects changed to low intensity or rested. Once the $RMSSD_{7D}$ was back to the mean SWC, they continued to perform moderate (MOD) or high intensity training (HIT). Mean running speed for 3000m (RS_{3000m}) improved by 2.7% +/- 2.5% over the PREP. No changes were found in endurance performance variables between sexes. The 3000m running performance improved only in the HRV-guided training group over the eight-week INT by performing less MOD and HIT sessions compared with predefined training. A single day of training does not change HRV move than SWC. Training at least for eight weeks can show a difference in HRV, which could show a difference in HRV depending on the time of the season, preseason, in-season or post-season. [16]

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Appendix A
INFORMED CONSENT FORM

I. INVESTIGATORS

Principle Investigators: Destany D. Gobin, ATC; Kaori Tamura, PhD, ATC, Portia Resnick, MA, ATC, LMT

II. TITLE

HEART RATE VARIABILITY IN COLLEGIATE DIVISION I ATHLETES

III. INFORMED CONSENT

The purpose of this consent form is to provide you with information about this research to help you decide if you would like to participate in this study. Please take your time to review this consent form. If there are any words or sections in this consent form that you do not understand or want to clarify, please do not hesitate to ask the research staff at any time.

IV. WHY IS THIS STUDY BEING DONE?

This study will try to find out more about Heart Rate Variability for Division I Collegiate athletes. It is important to have athletes from all teams and positions because there have been no studies to find normal ranges for a large, highly trainer population.

V. VOLUNTARY PARTICIPATION

A total of 150 participants will take part in this study. You are being asked to participate because you are between the ages of 18 and 25 years old and are a Division I collegiate athlete. It is important to understand that participation in this study is completely voluntary. You may decide not to participate, or withdraw at any time, and it will not affect you in any way. If you decided to participate in this study, you will be asked to sign this consent form. Upon clearance, you will be scheduled for the data collection session. We are asking 50 of the participants to return for a second data collection session, which will be exactly the same as the first. If you are willing to return for a second session, we will schedule you at the end of your first session.

VI. STUDY PROCEDURES

If you decide to participate in this study, you will be asked to attend 1 data collection session. This data collection session will take place in the Human Performance Laboratory at the University of Hawai‘i at Mānoa. You will have one ECG recording taken. We will measure your height, blood pressure, body mass and you will be asked to fill out a few forms.

The investigator will clean the electrode placement sites and then electrodes will be applied to designated positions. You will rest supine (on your back) or semi-reclined in a comfortable position for 10 minutes and no data will be collected. Then ECG data will be recorded for 15 minutes. At the same time you will place the finger of one hand over the flash of an iPhone in order to capture the same information using the Camera Heart Rate Variability smartphone application.

Should you have any redness or itching at the site of the electrodes prior to the end of data collection, report your symptoms to the investigator immediately and the electrodes will be removed.

VII. RISKS

There is minimal risk of an allergic reaction to the electrodes. Should any redness, swelling, discomfort or irritation occur while wearing the electrodes the electrodes will be removed immediately by the research team.

You will be asked to remain in on your back or semi-reclined position during 1 data collection for approximately thirty minutes. If you are not comfortable due to the position, you can ask to be re-positioned. A certified athletic trainer is available on site to deal with unexpected medical situations that may arise.

VIII. BENEFITS

You may not receive any direct or immediate benefits. However, your participation will help to further understand Heart Rate Variability and the Autonomic Nervous System in highly trained division I collegiate athletes, establishing a baseline for future studies.

IX. COSTS

All clinic and professional fees testing will be provided at no cost to you. Parking fees will be reimbursed to you if needed

X. COMPENSATION

No compensation will be given for your participation.

XI. CONFIDENTIALITY

All information about you will be held confidential to the extent allowed by state and federal law. Your personal information will not be given to anyone outside of the research team without your written permission. A code will be used as identifier instead of your name for this study. Research records that contain personal information, including code key, will be kept in a secure locked file in the Department of Kinesiology and Rehabilitation Science at the University of Hawai‘i at Mānoa. These documents will be permanently destroyed no later than 5 years after the completion of the study.

Information gathered in this research study may be published or presented in public forums, however your name and other identifying information will not be disclosed. Agencies with research oversight, such as the University of Hawai‘i Committee on Human Studies Program, have the right to review research records. You would be asked to sign an authorization form to allow the researcher to release any of your personal information obtained through the research process.

XII. INJURY RELATED TO THE STUDY

Should any injury or medical emergency occur during the data collection, first responder care (first aid and CPR) is available, and appropriate referral will be made. First responder care will be provided for free of charge, however, you will be responsible for the cost associated with referral thereafter. If your insurance will not pay for these costs, they will be your responsibility. The University of Hawai‘i has no program to pay or compensate you in any way for your injuries.

XIII. QUESTIONS

If you have any questions related to the study participation, please contact **Destany Gobin at 251-454-0968 or destany@hawaii.edu**. If you have questions or concerns about your rights as a research participant, please contact the Human Studies Program at (808) 956-5007.

XIV. STATEMENT OF CONSENT

I have read the above information, or it has been read to me. I have had the opportunity to discuss this research study with research staff, and I have had my questions answered by them in a language I understand. I take part in this study of my own free will, and I understand that I may withdraw from participation at any time and this will not affect me in any way. My consent to participate in this study does not take away any of my legal rights in the event of negligence or carelessness of anyone working on this project. A copy of this consent form has been given to me.

XV. SIGNATORIES

I agree to take part in this study.

_____

Print Name

_____

Signature **Date**

_____

Researcher Name (print)

_____

Researcher Signature **Date**

Appendix B

Data Collection

HRV Collegiate Division I Athletes

Patient Unique Number: _____ Age: _____

Date/Time: _____ Height: _____ Body Mass: _____

Blood Pressure: _____

Sport: _____ Position or Event: _____

Wave Length: I II III IV HR Segment: _____

Appendix C

Health History Questionnaire

1. Are you or is there a possibility that you may be pregnant? _____

2. Do you have any known tape allergies? _____

If you answered "YES" to any or the above question, you will not be allowed to continue this study

3. Do you have any known cardiac (heart) conditions? _____

4. If you answered yes to #4, please explain. _____

5. Are you currently injured? _____

6. If you answered yes to #5, please explain. _____

7. Do you have diabetes? _____

8. Do you have any neurological disorders? _____

9. If you answered yes to #8, please explain. _____

10. Do/did you have practice before or after data collection? If so, when? _____

11. Do you have a game before or after data collection? _____

12. Do you have an off day from normal physical activity? _____

13. Do you have any academic pressure within a 1-3 days or within 4-7 days? _____
_____

14. Are you in pre-season, post-season, or in season? _____

R E S T Q – 76 Sport

Single Code: _____ Group Code: _____

Name (Last): _____ (First): _____

Date: _____ Time: _____ Age: _____ Gender: _____

Sport/Event(s): _____

This questionnaire consists of a series of statements. These statements possibly describe your mental, emotional, or physical well-being or your activity during the past few days and nights.

Please select the answer that most accurately reflects your thoughts and activities. Indicate how often each statement was right in your case in the past days.

The statement related to performance should refer to performance during competition as well as during practice.

For each statement there are seven possible answers.

Please make your selection by marking the number corresponding to the appropriate answer.

Example:

In the past (3) days/nights

… I read a newspaper

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

In this example, the number 5 is marked. This means that you read a newspaper very often in the past three days.

Please do not leave any statements blank.

If you are unsure which answer to choose, select the one that most closely applies to you.

Please turn the page and respond to the statements in order without interruption.

In the past (3) days/nights

1) …I watched TV

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

2) …I did not get enough sleep

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

3) …I finished important tasks

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

4) …I was unable to concentrate well

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

5) …everything bothered me

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

6) …I laughed

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

7) …I felt physically bad

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

8) …I was in a bad mood

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

9) …I felt physically relaxed

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

10) …I was in good spirits

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

11) …I had difficulties in concentrating

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

12) …I worried about unresolved problems

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

13) …I felt at ease

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

14) …I had a good time with friends

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

15) …I had a headache

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

16) …I was tired from work

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

17) …I was successful at what I did

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

18) …I couldn't switch my mind off

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

19) …I fell asleep satisfied and relaxed

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

20) …I felt uncomfortable

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

21) …I was annoyed by others

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

22) …I felt down

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

23) …I visited some close friends

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

24) …I felt depressed

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

25) …I was dead tired after work

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

26) …other people got on my nerves

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

27) …I had a satisfying sleep

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

28) …I felt anxious or inhibited

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

29) …I felt physically fit

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

30) …I was fed up with everything

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

31) …I was lethargic

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

32) …I felt I had to perform well in front of others

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

33) …I had fun

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

34) …I was in a good mood

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

35) …I was overtired

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

36) …I slept restlessly

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

37) …I was annoyed

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

38) …I felt as if I could get everything done

0	1	2	3	4	5	6

never seldom sometimes often more often very often always

39) …I was upset

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

40) …I put off making decisions

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

41) …I made important decisions

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

42) …I felt physically exhausted

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

43) …I felt happy

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

44) …I felt under pressure

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

45) …everything was too much for me

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

46) …my sleep was interrupted easily

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

47) …I felt content

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

48) …I was angry with someone

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

49) …I had some good ideas

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

50) …parts of my body were aching

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

51) …I could not get rest during the breaks

0 1 2 3 4 5 6

never seldom sometimes often more often very often always

52) …I was convinced I could achieve my set goals during performance

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

53) …I recovered well physically

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

54) …I felt burned out by my sport

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

55) …I accomplished many worthwhile things in my sport

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

56) …I prepared myself mentally for performance

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

57) …my muscles felt stiff or tense during performance

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

58) …I had the impression there were too few breaks

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

59) *…I was convinced that I could achieve my performance at any time*

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

60) …I dealt very effectively with my teammates' problems

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

61) …I was in a good condition physically

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

62) …I push myself during performance

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

63) …I felt emotionally drained from performance

0 1 2 3 4 5 6
never seldom sometimes often more often very often always

64) …I had muscle pain performance

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

65) …I was convinced that I performed well

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

66) …too much was demanded of me during the breaks

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

67) …I psyched myself up before performance

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

68) …I felt that I wanted to quit my sport

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

69) …I felt very energetic

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

70) …I easily understood how my teammates felt about things

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

71) …I was convinced that I had trained well

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

72) …the brakes were not at the right times

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

73) …I felt vulnerable to injuries

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

74) …I set definite goals for myself during performance

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

75) …my body felt strong

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

76) …I felt frustrated by my sport

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

77) …I dealt with emotional problems in my sport very calmly

0	1	2	3	4	5	6
never	seldom	sometimes	often	more often	very often	always

Thank you very much!

Appendix E

Training Questionnaire

Subject: _____

Date: _____

Sport: _____

Position/event: _____

Pre-season In-season Post-season

Number of days per week you practice: _____

Number of hours per session: _____

Number of days per week you condition (weight room): _____

Number of hours per session: _____

Total number of hours spent training per week: _____