THE INFLUENCE OF RESISTANCE EXERCISE ON AUTONOMIC FUNCTION
AND MOOD STATE

A thesis submitted in partial fulfillment of the requirements
For the degree of Master of Science in Kinesiology

By
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Dedication

This achievement of a Master’s degree in Kinesiology does not solely belong to me. For if there was no endless love, reassurance, emotional and financial support from my family, I would not have been able to complete this journey as a graduate student. That being said, I dedicate this thesis to my remarkable and loving family.
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ABSTRACT

THE INFLUENCE OF RESISTANCE EXERCISE ON AUTONOMIC FUNCTION AND MOOD STATE

By
Gennadiy Yeshu
Master of Science in Kinesiology

Previous research has demonstrated how acute resistance exercise training affects the autonomic nervous system (ANS). However, the effects of acute resistance exercise on the ANS and mood states have not been fully studied. Therefore, this study explored the balance of the ANS during rest, exercise, recovery, and whether it was related to mood state. Parasympathetic (PNS) and sympathetic (SNS) nervous system activity were analyzed by heart rate variability (HRV), high frequency power (HF), low frequency power (LF), standard deviation of RR intervals (SDNN), total power, pre-ejection period (PEP), respiratory sinus arrhythmia (RSA), and heart rate (HR). Participants wore the VivoMetrics™ LifeShirt to record physiologic variables at rest, during exercise, and during recovery. The Profile of Mood States (POMS) was used to assess transient mood states. The results demonstrated that there were increases in sympathetic modulation and parasympathetic withdrawal during resistance exercise. After muscular endurance exercise, ANS activity did not return to baseline values during post-exercise recovery. Indicating increased sympathetic activity and decreased parasympathetic activity throughout endurance exercise and recovery. The pre-post exercise change in cardiac
autonomic balance was a significant predictor (inversely) of the variance in tension, depression, and mental confusion.

*Keywords:* Autonomic nervous system, resistance exercise, heart rate variability, pre-ejection period, respiratory sinus arrhythmia, heart rate recovery, muscular strength, muscular endurance, profile of mood state.
Chapter 1: Introduction

Acute bouts of heavy resistance exercise can lead to fatigue (33). The plausible mechanisms underlying resistance exercise related fatigue include, changes in central nervous system control, underlying hemodynamics (44), and vagal withdrawal (PSN) (40), all of which can be manifested in alterations in mood states and ANS function (24).

During physical exertion (locomotion) the adjustments in the cardiovascular system stem from a complex neural sensory system, which receives and relays signals back to higher brain centers. The information received by the central nervous system (CNS) is processed and sent to the peripheral nervous system, more specifically, the somatic nervous system (SoNS). Efferent nerves from the SoNS are responsible for initiating muscle contraction, while the afferent nerves relay sensory information back to the CNS. Within the CNS are two branches of the nervous system: the peripheral nervous system and the autonomic nervous system (ANS). Unlike the SoNS that functions voluntarily, the ANS runs involuntarily to control blood pressure, heart rate, urination, visceral organs and other processes. Thus, we do not have conscious control over the ANS. Both the ANS and the SoNS work together in an integrative manner. When one system receives information pertaining to a sensory signal, the other system provides the appropriate autonomic response (41).

The autonomic nervous system (ANS) is responsible for regulating the heart, gastrointestinal functions, glandular secretions and many other internal organs; it has two main branches: the sympathetic and parasympathetic branches (30). On a gross physiological level, the sympathetic branch is responsible for “fight or flight” reactions, while the parasympathetic branch is responsible for “rest and digest” responses. During a sympathetic reaction, neurotransmitters are released into the circulating blood and travel to effector organs; locally, neurotransmitters are secreted into synaptic spaces around
effector cells. As the neurotransmitters bind with adrenergic receptors, homeodynamic changes occur to allow active changes in the muscle, liver, heart, and lungs (41, 42). A parasympathetic response helps bring the body back to homeostatic balance by decreasing HR, temperature, and blood pressure (29).

Heart Rate Variability (HRV) provides a useful non-invasive means to assess sympathetic and vagal tone. During activities that stress homeostatic balance, it is difficult to non-invasively and directly assess the physiological regulation of the heart. Contemporary research provides supportive evidence for the use of HRV to evaluate changes in autonomic function (41). HRV is measured by evaluating the intervals between consecutive R waves from an electrocardiograph (ECG) recording. High levels of HRV are associated with good health, whereas low HRV is associated with poor health outcomes (30,1). In order to better understand HRV, it is necessary to evaluate the frequency (e.g., LF, HF) and time domain (e.g., SDNN, RMSSD) variables associated with the ANS. However, only testing the physiological system during muscular strength and endurance exercise is one piece of the puzzle. In order to have a better understanding of the relationship between the autonomic nervous system and mood states, the POMS questionnaire was used to test the association.

**Problem statement**

An ample quantity of research exists clarifying the role of the ANS during resistance exercise. Yet, there is a relative gap in the literature examining the relationship between autonomic responses, recovery from exercise, and mood states. The purpose of this investigation was to assess ANS balance during rest, exercise, recovery, and determine the relationship to mood states. By accurately understanding the relationships
between exercise, ANS balance and mood, we can more appropriately prescribe exercise to optimize psychophysiological health and wellness.

Hypotheses

It is hypothesized that:

1. Baseline vigor will be inversely related to the restoration of autonomic function post exercise.
2. The PNS will be suppressed post exercise, and this suppression will be reflected in mood states.
3. The restoration in PNS function after exercise will be related to increased cognitive confusion and decreased vigor 24-post exercise.
4. There relative change in Cardiac Autonomic Balance (CAB) from pre to post exercise will be related to changes in mood states (tension, depression, and mental confusion) 24-hours after exercise.

Operational Definition

*Endurance exercise* is a method of sustaining muscular “work” over a given time. In the current study, it is defined as performing an exercise (e.g. leg press, seated row, chest press) at 60% 1RM until exhaustion/failure.

*HRV* is defined as the variability in beat-to-beat intervals assessed by the VivoMetrics™ LifeShirt.

*Respiratory sinus arrhythmia (RSA)* measures heart rate coupled with inspiration and expiration rates. It represents a measurement of parasympathetic activity, by taking the difference between RR HI and RR low per beat. Hence, respiratory sinus arrhythmia works in synchrony with HRV and respiration.
*PEP* gauges the onset of ventricular stimulation to contraction by the VivoMetrics™ LifeShirt. This is measured by assessing the time between ventricular depolarization and left ventricular contraction. A decrease in PEP is synonymous with SNS activity.

*RMSSD* is the root-mean square differences of successive R-R intervals. Therefore, assessing rapid short-term changes in HR, thus, representing vagal mediation. *SDNN* is the standard deviation of R-R intervals and it is looking at overall HRV. The dependent variables will be HRV, RSA, PEP, RMSSD, SDNN, and POMS.

*CAB* is defined as the measure of autonomic balance of SNS and PNS reciprocation and calculated from the difference between the normalized values of HF and PEP.

*CAR* is defined as the measure of autonomic coactivation or coinhibition and calculated as the sum of the normalized values of HF and PEP.

**Assumption**

It is necessary to assume that:

1. Participants understood how to use the resistance equipment after instructions were given.
2. Participant’s honestly/properly completed the self-report questionnaire (e.g. POMS).
3. Electrodes and physiological equipment did not malfunction.

**Delimitation**

The following are delimitations of the study:
1. Participants were healthy normotensive men and women who attend California State University, Northridge.

**Limitation**

The following are limitations of the study:

1. The subjective nature of the POMS self-report questionnaire, which is a close-ended Likert scale form.
2. Leg press maximal strength values may be underestimated, due to machine limitations.
3. Due to the sample studied, the results may not be generalizable to a different population.
Effects of exercise training

Existing research has indicated that exercise training induces physiological adaptations to the ANS by reducing sympathetic control and enhancing parasympathetic outflow. Chronic resistance exercise training promotes a reduction in resting blood pressure (BP) and heart rate (HR), and an increase in cardiac size and function (13,35). These main physiological adaptations are important in protecting the body during exercise training.

Autonomic responses to acute exercise are regulated by the following mechanisms: central command (feedforward), exercise pressor reflex (feedback), and baroreflex mediation. During an exercise bout, central command is the initial neural mechanism that is responsible for a cardiovascular and respiratory response, followed by a peripheral neural reflex (16). Central command is governed by the medulla oblongata, located in the brainstem, and assists to modulate sympathetic and parasympathetic outflow (16); consequently, controlling heart rate and blood pressure during exercise bouts. The exercise pressor reflex is triggered by mechanoreceptors within skeletal muscle or by a metabolic by-product during muscular contraction. During stimulation of these receptors, sensory signals are sent to the CNS via type III and IV nerve fibers, moreover, enhancing sympathetic outflow, while reducing parasympathetic control (23). Lastly, the arterial baroreflex mechanism originates in the carotid sinus and aortic arch; moreover, it plays a vital role in autonomic neural regulation during exercise.

Regulation of heart rate and blood pressure is due to the negative feedback system, in which baroreceptors in the aortic arch sense pressure, and augment their physiological regulatory discharge. Furthermore, when blood pressure increases, the
Baroreflex mechanism acts to increase its rate of discharge through nerve IX and X (glossopharyngeal and vagus nerve) to the nucleus tractus solitarius (NTS), which resides in the medulla (12). Additionally, the NTS is responsible for regulating autonomic control of heart and blood vessels. Thus, when the baroreceptor reflex is activated, a sympathovagal interaction occurs in order to maintain the intrinsic blood pressure set point. When an increase in blood pressure is sensed, the NTS inhibits sympathetic outflow and simultaneously increases parasympathetic control; thus, decreasing HR and BP (12). However, during an acute bout of exercise, this mechanism would be counterproductive towards autonomic balance. Therefore, the intrinsic blood pressure set point resets during exercise, which is accomplished by regulating barosensitive neurons at the NTS (8). The reduced NTS occurs because of the central command during exercise; thus, resetting the intrinsic operating point of the arterial baroreflex to a higher set point.

**Acute exercise**

In a study conducted by Rocha et al. (37), the authors tested the hypothesis that the volume and intensity of resistance exercise (RE) might influence changes in recovery HRV. Rest/recovery BP and HRV were measured in 13 normotensive male participants who were between the ages of 20 and 30 years. Participants performed two similar RE bouts with an intermission of 48-hours between the routines: routine 1 (R1) consisted of two sets of 10RM; routine 2 (R2) had three sets of 10RM. Results from this study indicated that no change in RMSSD (marker for parasympathetic modulation) was noticed after 60 minutes post-exercise by either of the two routines. However, R2 stimulated an increase in the LF/HF component of HRV 60 minutes post-exercise. These results suggest that changes or decreases in cardiovascular function after resistance
exercise are dependent on set volume. This study introduced the idea that a higher resistance exercise volume would elicit an increase in overall training intensity, followed by a decrease in hypertensive effects. It should be noted that the reduction in BP reported in this study was not associated with decrease in cardiac sympathetic modulation; however, SV and CO were related to that effect.

Anunciacao et al. (1) reported similar results, in that the LF/HF ratio increased 60-minutes post-exercise, although RMSSD (marker for parasympathetic modulation) decreased. Therefore, falling BP initiates the negative feedback loop, in which a decrease in the Baroreflex activity allows homeostasis to be maintained by causing a shift to sympathetic dominance, with a continued withdrawal of parasympathetic control post-exercise (36,1). Anunciacao et al. (2) had similar results to Rocha et al. (37), with methodological differences. Participants in his study performed RE one set in circuit, three sets in circuit, one set of conventional pattern, and three sets of conventional pattern protocol. A noteworthy finding occurred in the three sets of conventional and circuit protocol. Both routines caused a decrease in RMSSD. However, the routines with a greater volume caused an increase in LF/HF post exercise (which is indicative of higher sympathetic balance). Although the 3CIRC protocol reduced BP and RMSSD, it presented significant increase in cardiac stress as measured by HR and HRV, when compared to the control and conventional protocol. Therefore, it is imperative to use multiple sets during RE training if the goal is to reduce BP (2).

Appropriate cardiovascular health determination before implementation of RE is necessary to avoid exercise-induced ventricular arrhythmias (EIVA), specifically when dealing with cardiac patients (3). Machado et al. (26) sought to investigate autonomic effects of incremental RE in coronary artery disease (CAD) patients. To meet inclusion criteria, 10 participants were required to have CAD for at least one year and maintain
stable blood pressure. Their 1RM was gauged and after a 10-minute rest, the participants were instructed to follow a progressive discontinuous RE protocol. RE initiated at 10% of 1RM, with increases of 10% until reaching 30% 1RM. This was then followed by increments of 5% load until exhaustion. ECG and BP were recorded and analyzed; results indicated that RMSSD was reduced significantly, accompanied by an increase in HR and SBP at 30% 1RM in both groups. Similar results were found in the studies of Anunciacao et al. (1) and Anunciacao et al. (2), identifying a reduction in parasympathetic modulation (RMSSD) during 60-minutes post-exercise recovery period, coupled with an increase in sympathetic control (LF/HF). Interestingly enough, Machado et al. (26) found that an intensity of 30% of the 1RM was the tipping point at which aerobic metabolism was augmented by anaerobic metabolism. In other words, at 30% of the 1RM, sympathetic modulation will become predominantly higher after anaerobic demands become more prominent, with an inverse shift in parasympathetic control.

Previous investigators have demonstrated the central, peripheral, and neural mechanisms in response to exercise (25). As exercise intensity increases, our ANS and SNS shift towards sympathetic control, with a withdrawal of the parasympathetic system. During increased loads of RE, HR and SBP increase to restore muscle blood flow (25). This has been linked to Baroreflex activity and modulation from aerobic to anaerobic metabolism (1,25). During increased loads of RE, the peripheral arterial system becomes mechanically compressed by the working muscle, causing a decrease in blood flow. Subsequently, dorsal root (type IV) afferent fibers are stimulated, and an action potential is sent to the brainstem (ventrolateral medullary region), which elicits an increase in sympathetic modulation (25).

Pichon et al. (35) sought to investigate effects of exercise intensity and duration on HRV. Fourteen healthy male athletes performed a constant-load exercise test on a
cycle ergometer at various intensity levels (60, 70, and 80%) of the power reached at VO$_{2\text{max}}$ for duration of 3, 6, and 9 minutes. Parasympathetic modulation (expressed by HFn.u.) increased significantly with exercise load. In contrast to the Anunciacao et al. (2), LF/HF, which represents sympathovagal balance and LFn.u decreased during exercise. These findings challenged the results of other researchers (1, 2, 25, 36), which stated the post-exercise LF/HF increased and RMSSD decreased. It is plausible that the increase in HFn.u during exercise is due to a linear increase in minute ventilation ($V_e$). In other words, as respiratory rate increased, HFn.u increased. Conversely, this linear increase explains why LF/HF decreased (35).

**Changes in PEP and RSA in response to exercise**

Respiratory sinus arrhythmia (RSA) and pre-ejection period (PEP) are markers for sympathetic and parasympathetic tone. RSA is influenced by parasympathetic (vagal) tone, and measured by analyzing the frequencies of synchronized heart rate (R-R interval) with respiratory rates. During inspiration, heart rate is set to increase, and it declines with expiration (17). Therefore, a decrease in the R-R interval on an ECG recording allows for an increase in HR, and cardiac vagal efferent activity becomes withdrawn. On the contrary, efferent cardiac vagal activity reaches its highest peak during expiration, therefore, increasing the R-R interval. That being said, RSA is caused by modulation of lung inflation and mediated by preganglionic cellular inputs (43). As HR increases to facilitate more blood to working muscles; breathing rate increases linearly to enable more O$_2$ and clear CO$_2$. Individual differences will be exhibited to meet these exercise demands. Sympathetic and vagal reactivity may yield reciprocal activation response, vagal withdrawal, or sympathetic reactivity (6). Berntson, Cacioppo, and Quigley (6) outlined a detailed explanation of nine possible mechanisms that control the
ANS. They are: reciprocal sympathetic activation, reciprocal parasympathetic activation, uncoupled sympathetic activation, uncoupled sympathetic inhibition, uncoupled parasympathetic activation, uncoupled parasympathetic inhibition, coactivation, coinhibition, and nonresponse (both SNS/PNS branches are unchanged from base levels).

PEP is influenced by sympathetic tone and is derived from the electrocardiograph (ECG) and Impedance Cardiograph (ICG). Defined as the onset of septal depolarization (Q-wave) through left-ventricular ejection, it is represented by the B-point on the impedance cardiograph (ICG) (4, 5, 9, 29, 38). The left ventricular end-diastolic diameter is a factor that can regulate end-diastolic volume. The larger the ventricular chamber size, the greater amount of blood that enters. During exercise the body redistributes blood from the splanchnic region towards the heart via muscle pump (i.e., contraction on veins) and respiratory pump (i.e., ΔIntrathoracic pressure). Therefore, redistribution of blood enhances venous return, which increases stroke volume (SV). It has been identified that PEP is reliant on a robust preload, afterload, and contractility (4). During exercise, changes in β-adrenergic inotropic drive (increased ejection fraction and ventricular contractility) are related to decrease in PEP (15), therefore, assisting with SV control during increased HR.

Houtveen et al. (20) found that changes in posture (e.g., supine, sitting, standing) led to a decreased impact of beta-adrenergic action on the heart, which progressively increased PEP. Results are inconsistent with the known effects of posture and HR (e.g. HR ↑ with postural Δ from sitting-standing), due to the fact that HR increases and PEP decreases by adrenergic inotropic agents (30). Bendjelid et al. (4) mentioned that a notable change in PEP is interconnected with respiratory rates, therefore, affecting LV stroke volume. More convincingly, Houtveen et al. (20) suggests that an increase in
afterload caused an increase in PEP, which was elicited by postural change. Afterload is the pressure that the left ventricle must overcome to eject blood into the aorta. When afterload increases, end systolic volume increases, with a decrease in stroke volume. The increased resistance to flow associated with high afterload will cause PEP to lengthen until left ventricle can overcome aortic pressure (30), therefore, allowing the aortic valve to open and blood ejection from the heart. Challenging the previous statement, Houtveen et al. (20) found that when changing posture from sitting to supine, PEP decreased.

**Autonomic response during rest and post-exercise**

The majority of studies have used HR and HRV measurements to assess autonomic function before and after exercise (10, 11, 33, 34, 36). When studying the oscillatory periods during a HRV analysis, investigators are carefully measuring the R-R intervals in an electrocardiogram (ECG) recording. HRV analysis has been proven to be a reliable tool when assessing ANS mechanisms, responses and adaptation (11), assisting in the detection of overtraining symptoms, and an important marker for all cause mortality and morbidity (10). The measurement of HRV is quantitatively evaluated by spectral analysis, and is used for studying various frequencies that are associated with autonomic fluctuation. The high frequency (HF) (0.15-0.40 Hz) band constitutes parasympathetic activity (19, 34), whereas low frequency (LF) (0.04-0.15 Hz) represents parasympathetic and sympathetic activity, more specifically, baroreceptor modulation (7, 34). The baroreceptor reflex modulates both sympathetic and parasympathetic tone to the heart. Baroreceptors are located within the carotid sinus and aortic arch, which respond to pressure imposing on the vessel walls. Therefore, when BP increases, the baroreceptors sense the force on the internal wall of a blood vessel; thus, inhibiting sympathetic tone to the heart while increasing parasympathetic activity. The LF/HF ratio acts as a marker for
sympathetic to parasympathetic power, also known as, sympathovagal balance (5, 11, 34) however, reliability is questionable (17). The very low frequency (VLF) band represents peripheral vasomotor/chemoreceptor regulation, however, when analyzing a recording less than 5 minutes in length, caution should be employed in drawing conclusions from this band, due to the potential for ambiguous results (7).

According to Berntson et al. (5) an increased cardiac regulatory capacity in both autonomic branches is indexed by high HRV. Thus, low HRV can be used as a predictor for all cause mortality and morbidity (13). During resting conditions, a sympathovagal balance exists, with parasympathetic predominance. Furthermore, as exercise initiates, parasympathetic outflow beings to withdraw, yielding an increase in HR and sympathetic outflow. The SNS remains fully active during exercise, until cessation of exercise is reached. During the recovery phase, sympathetic flow is active with a gradual increase in parasympathetic tone. A number of mechanisms influence the time to return to resting, steady state levels of ANS tone post exercise, such as blood metabolites and exercise intensity.

Therefore, McDonald et al. (28) assessed HRR in aerobically and anaerobically trained cyclists. Participants consisted of 10 track cyclists (1 female) and 15 road cyclists (4 female) all highly trained. The protocol consisted of a 10-minute warm-up, followed by incremental 50-watt increases in power every 2 minutes; the test was terminated when two out of four conditions were met. When participants were unable to maintain safe HR levels, \( \text{O}_2 \) uptake plateaued, respiratory exchange ratio (RER) surpassed 1.15, or when participants requested to self-terminate test. Results indicated that HR in aerobically trained cyclist returned to pre-exercise levels sooner than non-aerobically trained. In the heart of the aerobically trained individuals, left ventricular wall diameter is larger and there is an increase in muscle mass. This allows for more optimal filling, which yields an
increase in stroke volume (18). This increase in SV results in the heart having to beat fewer times per minute to get the same overall volume of blood ejected out of the heart per minute, allowing for better overall blood redistribution.

Esco et al. (11) sought to investigate if HRV pre-exercise was related to heart rate recovery (HRR). In his study, 66 healthy men performed the “Bruce Treadmill Protocol” for 30 minutes to assess HRV. The results of this study indicated that no correlation was found resting between HRV and HRR, suggesting that pre exercise autonomic function is not related to the restoration of baseline levels of cardiovascular function post exercise. However, this study also reported significant, inverse correlations between SDNN, HFn.u, and HRR.

Heffernan et al. (19) hypothesized that RE training would not change the linear measurements of HRV (i.e., time and frequency domain) post-exercise, while HRR would improve, due to enhanced cardiac autonomic modulation. The researchers monitored fourteen male participants during a 6-week resistance training protocol. Participants carried out the 3-days/week RE protocol of five exercises that targeted all major muscle groups of the entire body. The results indicated that HRR was improved post-exercise, signifying a more rapid recovery of vagal tone. Consistent with the findings of Esco et al. (11), no improvement in the spectral measurements of HRV was found post-exercise. Esco et al. (11) also reported that a higher SDNN (overall HRV) and HFn.u. (Parasympathetic modulation) pre-exercise, could possibly correspond to a lower HR post-exercise. The results from the Heffernan et al. (19) study differed, and the authors suggested that although HRR is mediated by short-term RE, RE does not affect HRV post exercise. Thus, the re-activation of the parasympathetic nervous system, as evidenced by HF, decreases HR initially after the cessation of exercise, followed by sympathetic (LF) withdrawal post-exercise. A common pattern of the ANS response to
exercise is the withdrawal of HF at the beginning of exercise, followed by withdrawal of LF post-exercise. Within a healthy population, the ANS should adhere to these representative principles, although individual responses may differ.

In contrast to Esco et al. (11); Heffernan et al. (19), and Ng et al. (32); Gladwell et at. (15) investigated how three vigorous exercise sessions, 20 minutes in duration, affected autonomic activity results that were analyzed up to 65-minutes post-exercise.

Thirteen individuals (7 males, 6 females), who were normotensive by self-report, participated in a maximal cycle ergometer protocol that lasted 20 minutes on three separate occasions 5 days apart. HRV analyses indicated similar results to previous investigators, wherein the initiation of exercise is associated with sympathetic activation and vagal withdrawal. However, in this investigation, at 5-minutes post-exercise, vagal activity remained depressed while sympathetic input was still elevated. It is assumed that vigorous exercise intensity triggered a heightened influx of sympathetic activity, which can be associated with a slower parasympathetic reactivation. In other words, during post-exercise reactivation of the parasympathetic nervous system, an over accumulation of metabolites and catecholamine’s within the active muscle maintains the activation of sympathetic drive, while gradually increasing vagal tone (14). Conventionally, reactivation of the parasympathetic nervous system is responsible for HRR during the first minute followed by further withdrawal of the sympathetic branch (21).

**ANS effect on mood states**

A vast amount of literature has been published in order to elucidate the relationship between exercise and autonomic nervous system function. It is known that exercise training improves physiologic function and overall health (13); however, resistance exercise and mood states have not been well correlated. Current research does
demonstrate that resistance and aerobic exercise can improve mood states and mental performance (39).

Leti et al. (24) investigated the association of fatigue state in runners with markers of HRV. HRV indices were collected on 10 competitive runners (mean age 55 years) over a course of 12 weeks. HRV measurements were administered at home (during the night) after the end of the resting period, post training, post-race, and after a rest day. The Profile of Mood States (POMS), SFMS (a questionnaire regarding fatigue), and a questionnaire of quality of sleep were administered at the end of a 14-day rest (i.e. no physical activity). The resulting fatigue scores were correlated with HRV markers. The results indicated that during a competition, the sympathetic tone remained elevated more so than it did on other nights. It is conceivable to say that physical and psychological stress played a role in their findings. Therefore, a strong correlation was found between fatigue measured by the POMS test and an elevated sympathetic tone during recovery phase.

Conversely, Nuissier et al. (33) demonstrated that the POMS subscales were correlated with HRV indices. Participants underwent selective step-by-step procedure to determine who would be placed in the control or potentially overtrained (POT) group. Out of 350 participants, 28 were placed appropriately: 12 (POT group) and 16 (control group). Results revealed that during the head-up tilt (HUT) test, depression scores were significantly correlated with a decrease in HF tone and ANS activity. Thus, a high total mood disturbance (TMD) score was associated with blunted ANS activity. TMD score was inversely related to the vigor subscale of POMS. Physical exercise supported a positive effect on mood states, likewise exhibiting a significant increase in vigor (39). Therefore, vigor was used to assess overall HRV, representing a connection between increased vigor and autonomic tone. Conversely, Martins et al. (27) found that after an
aerobic and strength based exercise program did not affect mood states. Similar to other investigations (33, 39), vigor increased due to participation in a strength training program (26).

Yu et al. (44) found a relationship between mood states and hemodynamic variables. Seventy-one normotensive/ hypertensive participants spent 3 days prior to testing in a hospital setting in order to adjust to testing environment. Subsequently, participants filled out the POMS self-report, and had resting physiology measurements taken the next day. The results indicated that after 3 days rest in hospital, hypertensive participants came back to normal baseline measurements (i.e., SBP, MAP, DBP, HR, Q, SV, TPR). Therefore, an aggregate physiological measurement was used during analysis. Significant negative correlations were found between cardiac output and POMS subscales (fatigue-inertia). Significant correlations were found among log normalized stroke volume, TPR, and POMS subscales (e.g. fatigue-inertia). It was also observed that log stroke volume was negatively correlated to POMS (tension-anxiety), while log TPR was positively correlated with POMS (fatigue-anxiety). Due to the fact that hemodynamic responses are overly intricate to merely be analyzed by mood states, these results suggested that hemodynamic variables are associated with subtle changes in mood.

In summary, resistance exercise tends to benefit cardiovascular function more favorably when a program focuses on increasing the volume of resistance exercise, regardless of intensity (1, 2, 37). It is imperative to comprehensively evaluate HRV variables during muscular endurance (high volume) training, to better understand the dynamics of the ANS both during and after exercise. Understanding central cardioprotective mechanisms, exercise responses and training adaptations is important to promoting safe exercise prescription. Optimal exercise prescriptions can take into account both the physiologic and psychological changes that can occur during an acute
exercise bout, optimizing the training effect while diminishing the potential for distress. With that in mind, the purpose of this study is to investigate the ANS during and after muscular endurance exercise, to determine whether changes in the ANS are related to mood states.
Chapter 3: Methods

Participants

This study was open to all participants who met the inclusionary criteria (ability to participate in exercise protocols, between the ages of 18 to 60 years). Data from thirty-four subjects, 14 males and 20 females, aged 19-50 years old tested at California State University, Northridge (CSUN) were utilized for the study. Twenty-six additional participants were excluded due to incomplete data (the participants only participated in muscular strength exercise, instead of both strength and resistance). Exclusionary criteria consisted of (1) physical injuries, (2) untreated psychological disorders, (3) cardiovascular disease, and (4) not properly adhering to the exercise protocol. Participants were instructed to complete an informed consent, health history questionnaire, and a Profile of Mood States (POMS) form.

Table 1. Participant Descriptive Characteristics

<table>
<thead>
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<th>Minimum</th>
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<td>34</td>
<td>59.00</td>
<td>77.00</td>
<td>67.19</td>
<td>4.21</td>
</tr>
<tr>
<td>Weight (lbs.)</td>
<td>34</td>
<td>102.00</td>
<td>247.00</td>
<td>150.91</td>
<td>36.79</td>
</tr>
</tbody>
</table>

Instrumentation

The VivoMetrics™ LifeShirt system is a non-invasive, ambulatory monitoring unit that gathers and stores physiological data. The packaged system is comprised of a lightweight and machine washable vest, electrodes, a data cable, software, and an ambulatory computer. There are two respiratory sensors embedded into the LifeShirt that continuously monitor respiration and heart activity. Known as inductive plethysmography, it measures pulmonary ventilation by analyzing chest movement via
the embedded respiratory sensors. In between the two respiratory sensors is a thoracocardiograph (TCG) band. The TCG band is placed transversely across the body, at the xiphoid process level. It must be kept taut in order to achieve maximum extraction of cardiac oscillations. Respiratory movement must be suppressed and filtered for noise/artifact, in order for the data to reflect true left ventricular contraction. A three-lead ECG attaches to embedded wires in the LifeShirt; two electrodes are placed in the subclavicular region, and one is placed on the lower right abdomen. In the current investigation, all physiological data was recorded onto a compact flash card, via the portable palm pilot computer, which was attached to the LifeShirt. Following completion of data collection, information on the compact memory card was transferred onto a desktop computer for cleaning and analysis by the VivoLogic software.

**Self-Report Questionnaire**

In order to assess brief, variable feelings, and affective psychological mood state, the Profile of Mood States (POMS) questionnaire was utilized to determine how participants were feeling before each exercise session, as well as 24-hours after the resistance exercise. The form consisted of 65 adjectives rated on a 5-point Likert scale, markers ranging from “Not at all” to “Extremely”; describing feelings that participants might have had during the past week (quantifying baseline), and how they feel at the current moment (assessing 24-hours post exercise). Six subscales are formed from the combination of items when assessing changes in specific moods: tension-anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia, and confusion-bewilderment.
Protocol

Participants came to the laboratory one time during the entire study, and occupied between one and three hours of time, which varied by individual. Of the thirty-four participants, eighteen had previously performed 1RM muscular strength testing. Participants were then screened to safeguard adherence of inclusion criteria, followed by completion of consent forms. Research facilitator(s) familiarized participants regarding exercise protocol, equipment, testing instruments and exercise safety explanation/demonstration. Participants were fitted for the appropriate Vivometric LifeShirt™ size, and subsequently electrodes (3) were placed on chest and abdomen for physiological measurements. Equipment calibration and baseline measurements were obtained, followed by a baseline self-report questionnaire (Profile of Mood States).

Participants were instructed to adhere to the protocols of the three muscular strength (1RM) exercises. Initially, leg press exercises were implemented for 10 repetitions at the lowest set weight of the machine, followed by a subjective score assessing the degree of difficulty (1=easy and 10=extremely difficult). Afterwards, resistance weight incrementally increased, with five repetitions the target goal. The subjective score was assessed and resistance weight gradually increased with every single repetition until a failed attempt was observed, and 1 (RM) was determined. The preceding protocol was repeated for the seated row and chest press exercises. Following the completion of muscular strength exercises, participants were instructed to perform muscular resistance exercise at 60% of 1RM until volitional fatigue. Afterwards, recovery heart rate measurements were recorded while participants were rested for 7 minutes in the supine position. All participants were sent home with a self-report questionnaire, to be completed 24-hours after the resistance exercise workout.

Following completion of exercise procedure, the scan disc was removed from the
palm pilot computer and data was transferred to the desktop for VivoLogic cleaning and analysis. The participant’s raw data was imported into the VivoLogic system for filtering, artifact cleaning, R-wave correction, and manipulation. Subsequently, the file was cleaned for Stroke Volume and negative Pre-ejection Period values. Negative PEP values are physiological implausible; therefore, removal of artifact surrounding the negative value, or adjustment by fixing the R-waves associated with the data was necessary. Analysis Intervals were set to 00:01:00, with a step size of 00:00:30, and a Fast Fourier Transform (FFT) was used to transform data from the time to frequency domain. Heart rate variability data were calculated in order to achieve HR, RSA, PEP, VLF, Total Power, LFnu, HFnu, LF/HF, SDNN, and RMSSD values; for rest, muscular strength, muscular resistance, and recovery. SDNN and RMSSD (time domain) were derived from R-R intervals, while frequency domain were derived from the power spectrum of R-R intervals. VivoLogic software used a non-parametric power density spectrum that was based on the Fast Fourier Transformation method. The Fast Fourier Transform is a fast algorithm for one and multidimensional systems theory and signal processing, constructed by means of a given filtration or sequence of subgroups (31). Following HRV analysis, calculated VivoLogic values were entered into Excel for mean and standard deviation calculation. This was followed by entering the average value for each variable during each time period into SPSS, version 22.0. Raw numbers were used for rest to recovery and strength to endurance comparisons, and statistical tests were run with normalized data. Relative changes were calculated for rest to recovery; aggregate of strength, endurance, PEP, and RSA using the formula: [(“Exercise” – Rest)/Rest] x 100.

Mean values for cardiac autonomic balance (CAB) and cardiac autonomic reactivity (CAR) were also calculated for the study. In order to calculate CAB and CAR values, raw HF and PEP values were normalized and converted to z-scores, afterwards
CAB and CAR values were calculated in SPSS. The formula for CAB: \( \text{CAB} = \text{HFz} - (-\text{PEPz}) \), expresses that high CAB values are representative of greater parasympathetic control and lower sympathetic control. CAR expressed by: \( \text{CAR} = \text{HFz} + (-\text{PEPz}) \), CAR show a strong indication of PNS influence, coupled with SNS control; thus, demonstrating coactivation of both autonomic branches. Increased values in CAR indicate high parasympathetic and sympathetic tone (5).

**Statistical Analysis**

For the purposes of this paper, only the data from the session with muscular endurance testing was chosen for analysis. All comparisons between variables obtained at different points in time were analyzed using a repeated measures analysis of variance (rANOVA). A repeated measure ANOVA was used to analyze differences between rest and recovery for temporal and spectral data, and for all time periods of testing (rest, 1-RM, endurance, aggregate endurance, recovery), using normalized data when variables were not normally distributed. Repeated measures ANOVA was also used to analyze the differences in the relative changes from rest to 1-RM and rest to muscular endurance exercise for temporal and spectral data. Significant main effects and pairwise comparisons were distinguished, with \( p < .05 \) set as the level of significance. Statistical analyses were performed using SPSS Statistics Software, version 22.0.
Chapter 4: Results

Descriptive Statistics

Descriptive statistics for average weight lifted during muscular strength and endurance exercises, as well as the percentage of the 1RM and number of repetitions completed for each exercise are displayed in Table 2. Average weight lifted during muscular strength exercises (LP, SR, CP) are as follows: 367, 139, and 134 lbs; average weight lifted during resistance exercises were: 217, 83, and 80 lbs. The current study’s protocol for muscular endurance resistance exercise was to take 60% of the 1RM and complete consecutive lifts until failure. According to Table 2, actual percentage of the 1RM-lifted during muscular endurance exercises (LP, SR, CP) were as follows: 59.93%, 59.78%, and 60.10%, with the average number of repetitions of 81, 18, and 25 respectively.

<table>
<thead>
<tr>
<th>Table 2. Descriptive Statistics weight lifted, %1RM, and #Reps completed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>1rm LP</td>
</tr>
<tr>
<td>1rm SR</td>
</tr>
<tr>
<td>1rm CP</td>
</tr>
<tr>
<td>End LP</td>
</tr>
<tr>
<td>End SR</td>
</tr>
<tr>
<td>End CP</td>
</tr>
<tr>
<td>1RM % LP</td>
</tr>
<tr>
<td>1RM % SR</td>
</tr>
<tr>
<td>1RM % CP</td>
</tr>
<tr>
<td>Repetitions LP</td>
</tr>
<tr>
<td>Repetitions SR</td>
</tr>
<tr>
<td>Repetitions CP</td>
</tr>
</tbody>
</table>
**Rest to Recovery Comparison**

Displayed in Table 3, are the mean values for the autonomic variables rest and during recovery. Pairwise comparisons revealed statistically significant changes in HFnu, VLF, PEP, RSAIn, SDNN, RMSSD, and Total Power, indicating a decrease from Pre to Post exercise. An increase was observed in LFnu, LF/HF ratio and HR from Pre to Post exercise; CAB and CAR did not change significantly from rest to recovery. The mean and standard deviation percent change from rest to recovery CAB was -54.31 (189.47) and CAR 245.39 (1661.38).

**Table 3. Rest to Recovery Comparisons**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre-Exercise M±SD</th>
<th>Post-Exercise M±SD</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LFnu (ms²)</td>
<td>.48(.14)</td>
<td>.53(.13)*</td>
<td>.043</td>
</tr>
<tr>
<td>HFnu (ms²)</td>
<td>.43(.13)</td>
<td>.31(.17)*</td>
<td>.000</td>
</tr>
<tr>
<td>VLF (ms²)</td>
<td>463.50(345.60)</td>
<td>275.43(1060.93)*</td>
<td>.000</td>
</tr>
<tr>
<td>PEP (ms)</td>
<td>123(21.56)</td>
<td>110.62(19.79)*</td>
<td>.002</td>
</tr>
<tr>
<td>RSAIn (ms²)</td>
<td>103.10(75.04)</td>
<td>39.28(36.02)*</td>
<td>.000</td>
</tr>
<tr>
<td>LF/HF (ms²)</td>
<td>1.98(1.63)</td>
<td>3.93(3.74)*</td>
<td>.006</td>
</tr>
<tr>
<td>SDNN (ms)</td>
<td>73.46(31.63)</td>
<td>32.79(28.47)*</td>
<td>.000</td>
</tr>
<tr>
<td>RMSSD (ms)</td>
<td>70.31(43.57)</td>
<td>25.31(26.29)*</td>
<td>.000</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>66.55(10.62)</td>
<td>89.73(14.76)*</td>
<td>.000</td>
</tr>
<tr>
<td>Total Power (ms²)</td>
<td>4235.53(3736.71)</td>
<td>1503.93(3703.87)*</td>
<td>.000</td>
</tr>
</tbody>
</table>

The mean difference between rest and post-exercise recovery is significant at the *p < .05 level.

**Mean Comparison in Strength and Endurance**

Table 4a illustrated the comparisons among the HRV variables at rest, during 1RM testing, and recovery. A repeated measures ANOVA (sphericity assumed) was used to determine the effect of testing condition during the measurement of muscular strength for the time points illustrated in the table below. Pairwise comparison revealed the
changes from rest to 1RM. Significant condition related differences were found for the variables: LFnu, HFnu, VLF, RSAln, LF/HF, and HR. Sympathetic activity, expressed by LFnu, increased from rest to 1RM. RSA, representing parasympathetic modulation decreased from rest to 1RM. This relationship demonstrated reciprocal parasympathetic activation, in which an increase in sympathetic activity was coupled with a decrease in parasympathetic activity (6). Mean values from 1RM to recovery revealed significant differences in the following variables: LFnu, VLF, PEP, RSAln, SDNN, RMSSD. SDNN, representing overall HRV, showed a peak during the 1RM testing when compared to rest and recovery values. Whereas RMSSD, indicating parasympathetic modulation, was blunted. When analyzing changes from rest to recovery, all variables except LFnu changed significantly. Parasympathetic activity, as represented by HFnu and RSAln, decreased from baseline. SDNN and Total Power (total HRV modulation) were also lower after exercise. However, LFnu and PEP (markers of sympathetic activity) remained elevated during the post-exercise recovery segment.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Rest M±SD</th>
<th>Sum 1RM M±SD</th>
<th>Recovery M±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>LFnu (ms²)</td>
<td>.48(.14)</td>
<td>.62(.07)*</td>
<td>.53(.13)**</td>
</tr>
<tr>
<td>HFnu (ms²)</td>
<td>.43(.13)</td>
<td>.24(.05)*</td>
<td>.31(.17)+++</td>
</tr>
<tr>
<td>VLF (ms²)</td>
<td>463.50(345.60)</td>
<td>1938.32(3918.78)*</td>
<td>275.43(1060.93)**,+++</td>
</tr>
<tr>
<td>PEP (ms)</td>
<td>123(21.56)</td>
<td>124.27(15.84)</td>
<td>110.62(19.79)**,+++</td>
</tr>
<tr>
<td>RSAln (ms²)</td>
<td>103.10(75.04)</td>
<td>69.37(96.87)*</td>
<td>39.28(36.02)<strong>,</strong>,+++</td>
</tr>
<tr>
<td>LF/HF (ms²)</td>
<td>1.98(1.63)</td>
<td>4.01(1.42)*</td>
<td>3.93(3.74)+++</td>
</tr>
<tr>
<td>SDNN (ms)</td>
<td>73.87(32.03)</td>
<td>100.21(98.35)</td>
<td>32.82(28.92)<strong>,</strong>,+++</td>
</tr>
<tr>
<td>RMSSD (ms)</td>
<td>70.31(43.57)</td>
<td>63.02(78.68)</td>
<td>25.31(26.29)<strong>,</strong>,+++</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>66.55(10.62)</td>
<td>90(14.08)*</td>
<td>89.73(14.76)+++</td>
</tr>
<tr>
<td>Total Power (ms²)</td>
<td>4235.53(3736.71)</td>
<td>9055.52(20503.28)</td>
<td>1503.93(3703.87)+++</td>
</tr>
</tbody>
</table>

The mean difference from rest - exercise is significant at the * p < .05 level.
The mean difference from exercise - recovery is significant at the ** p < .05 level.
The mean difference from rest - recovery is significant at the +++ p < .05 level.
Table 4b demonstrated the comparisons among the HRV variables at rest, during muscular endurance testing, and recovery. A repeated measures ANOVA (sphericity assumed) was used to determine the main effect of condition during muscular endurance for the time points illustrated in the table below. Pairwise comparison revealed significant changes from endurance to recovery for the following variables: HFnu, PEP, RSAln, HR. Parasympathetic activity (HFnu, RSAln) decreased during exercise and increased slightly during recovery. However, the slight increase in HFnu and RSAln during recovery was still below baseline values. Sympathetic activation (PEP, HR) increased during endurance exercise, followed by a small decrease during recovery.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Rest M±SD</th>
<th>Sum Endurance M±SD</th>
<th>Recovery M±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>LFnu (ms²)</td>
<td>.48(.14)</td>
<td>.51(.11)</td>
<td>.53(.14)</td>
</tr>
<tr>
<td>HFnu (ms²)</td>
<td>.43(.13)</td>
<td>.17(.07)*</td>
<td>.31(.17)**,+++</td>
</tr>
<tr>
<td>VLF (ms²)</td>
<td>449.30(337.15)</td>
<td>252.81(267.98)*</td>
<td>298.53(1109.89)***</td>
</tr>
<tr>
<td>PEP (ms)</td>
<td>122.53(21.54)</td>
<td>98.13(24.40)*</td>
<td>109.56(19.24)**,+++</td>
</tr>
<tr>
<td>RSAln (ms²)</td>
<td>102.70(77.23)</td>
<td>25.70(19.98)*</td>
<td>41.10(36.36)**,+++</td>
</tr>
<tr>
<td>LF/HF (ms²)</td>
<td>1.77(1.07)</td>
<td>5.59(2.92)*</td>
<td>4.01(3.84)***</td>
</tr>
<tr>
<td>SDNN (ms)</td>
<td>72.39(31.83)</td>
<td>45.34(27.41)*</td>
<td>35.63(31.09)***</td>
</tr>
<tr>
<td>RMSSD (ms)</td>
<td>69.21(44.40)</td>
<td>22.84(20.42)*</td>
<td>26.33(26.78)***</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>66.57(11.08)</td>
<td>123.89(20.79)*</td>
<td>87.95(13.87)**,+++</td>
</tr>
<tr>
<td>Total Power (ms²)</td>
<td>4144.68(3820.47)</td>
<td>1518.23(2488.20)*</td>
<td>1589.07(3804.75)***</td>
</tr>
</tbody>
</table>

The mean difference from rest - exercise is significant at the * p < .05 level.
The mean difference from exercise - recovery is significant at the ** p < .05 level.
The mean difference from rest - recovery is significant at the +++ p < .05 level.

Relative Change CAB/CAR Correlation

A Pearson correlation coefficient was calculated for the relationship between relative change from rest to endurance (CAB/CAR) and baseline Vigor. A low inverse correlation was found between the relative change in CAB (during aggregate endurance) and Vigor (r (32) = -.361, p = .042), as well as in CAR (during aggregate endurance) and Vigor (r (32) = -.352, p = .048) depicted in Table 5. However, no
relationship was found between rest or recovery CAB/CAR and any POMS variable (tension, depression, anger, fatigue, confusion, vigor), or change from baseline to recovery with baseline POMS.

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
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<tbody>
<tr>
<td>Tension</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>Depression</td>
<td></td>
<td>.679*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anger</td>
<td></td>
<td></td>
<td>.585*</td>
<td></td>
<td>.749*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td></td>
<td></td>
<td></td>
<td>.585*</td>
<td></td>
<td>.324</td>
<td>.434*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Confusion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.501*</td>
<td></td>
<td>.634*</td>
<td>.361*</td>
<td>.246</td>
</tr>
<tr>
<td>Vigor</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.068</td>
<td>.072</td>
<td>-.176</td>
<td>-.204</td>
</tr>
<tr>
<td>%Δ CAR</td>
<td>-.187</td>
<td>-.178</td>
<td>-.050</td>
<td>.045</td>
<td>-.213</td>
<td>-.352*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>%Δ CAB</td>
<td>-.040</td>
<td>-.098</td>
<td>.014</td>
<td>-.167</td>
<td>.047</td>
<td>-.361*</td>
<td></td>
<td>.201</td>
<td></td>
</tr>
<tr>
<td>Rest CAB</td>
<td>.058</td>
<td>-.032</td>
<td>-.021</td>
<td>-.085</td>
<td>.110</td>
<td>-.119</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest CAR</td>
<td>-.162</td>
<td>-.278</td>
<td>-.013</td>
<td>-.134</td>
<td>-.223</td>
<td>-.329</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Rec CAB</td>
<td>.059</td>
<td>-.122</td>
<td>-.098</td>
<td>-.226</td>
<td>.058</td>
<td>-.082</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Rec CAR</td>
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<td>-.127</td>
<td>-.074</td>
<td>-.026</td>
<td>.189</td>
<td>-.285</td>
<td>.063</td>
<td>.319</td>
<td></td>
</tr>
</tbody>
</table>

**Baseline POMS to Relative Autonomic Change**

Table 6 demonstrates a correlation between baseline mood states (mental confusion) and autonomic function. A Pearson correlation coefficient was calculated for the relationship between baseline POMS and relative change in autonomic function. A strong positive correlation was found \( r(32) = .511, p = .002 \), between the relative change in RSA and POMS variable (mental confusion). A strong positive correlation \( r(32) = .679, p = .000 \) was found between depression and tension. Anger and tension exhibited a moderate positive correlation \( r(32) = .585, p = .000 \), while anger and depression indicated a very strong relationship \( r(32) = .749, p = .000 \). A moderate positive correlation \( r(32) = .585, p = .000 \) was found between fatigue and tension, whereas, fatigue and anger demonstrated a low to moderate relationship \( r(32) = .434, p = .010 \). Mental confusion and depression revealed a strong positive correlation \( r(32) = .634, p = .000 \); confusion and tension established a moderate positive relationship \( r = .499^* \).
(32) = .501, p = .003), while, confusion and anger showed a weak positive correlation (r
(32) = .361, p = .036).

Table 6. Correlation between % Change from baseline to recovery in Autonomic
function and baseline POMS scores

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Tension</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Depression</td>
<td>.679**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Anger</td>
<td>.585**</td>
<td>.749**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Fatigue</td>
<td>.585**</td>
<td>.324</td>
<td>.434*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Confusion</td>
<td>.501**</td>
<td>.634**</td>
<td>.361*</td>
<td>.246</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Vigor</td>
<td>-.044</td>
<td>.068</td>
<td>.072</td>
<td>-.176</td>
<td>-.204</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. %Δ HFn</td>
<td>-.067</td>
<td>-.039</td>
<td>-.003</td>
<td>-.105</td>
<td>.185</td>
<td>.023</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. %Δ RSA</td>
<td>.152</td>
<td>.029</td>
<td>-.102</td>
<td>-.137</td>
<td>.511*</td>
<td>-.044</td>
<td>.293</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>9. %Δ PEP</td>
<td>-.012</td>
<td>-.105</td>
<td>-.002</td>
<td>-.176</td>
<td>-.231</td>
<td>-.009</td>
<td>.045</td>
<td>.202</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviation: HFn - High Frequency normalized, RSA - Respiratory Sinus Arrhythmia, PEP - Pre Ejection Period
Correlation significant at the *p < .05,
**p < .01
Recovery POMS to Relative Autonomic Change

Depicted in Table 7, the relative change from rest to recovery CAB was found to be inversely related to change in POMS (tension, depression, confusion). A Pearson correlation coefficient was calculated and revealed a moderate inverse relationship ($r(32) = -0.415$, $p = .015$), between recovery tension and relative change in CAB; a moderate inverse relationship ($r(32) = -0.401$, $p = .019$) among recovery depression and relative change in CAB, and a weak inverse correlation between recovery confusion and relative change in CAB ($r(32) = -0.371$, $p = .031$).

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. %Δ CAB</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. %Δ CAR</td>
<td>.122</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Tension</td>
<td>-0.415*</td>
<td>-0.028</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Depression</td>
<td>-0.401*</td>
<td>.052</td>
<td>.812**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Anger</td>
<td>-0.207</td>
<td>-0.026</td>
<td>.541**</td>
<td>.518**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Fatigue</td>
<td>-0.064</td>
<td>.029</td>
<td>.437**</td>
<td>.385*</td>
<td>.458*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Confusion</td>
<td>-0.371*</td>
<td>.180</td>
<td>.719**</td>
<td>.720**</td>
<td>.433*</td>
<td>.459**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Vigor</td>
<td>.187</td>
<td>-0.214</td>
<td>-0.383*</td>
<td>-0.190</td>
<td>-0.353*</td>
<td>-0.415*</td>
<td>-.446**</td>
<td>1</td>
</tr>
</tbody>
</table>

**Abbreviation:** CAR - Cardiac Autonomic Reactivity, CAB - Cardiac Autonomic Balance
Correlation significant at the *$p < .05$, **$p < .01$*

A Pearson correlation coefficient was calculated for the subscales of the POMS 24-hours after recovery, and CAB and CAR immediately subsequent to activity. Post exercise autonomic function was not significantly related to any of the subscales of the POMS after recovery from exercise (Table 8).
Table 8. Correlation between recovery CAB/CAR and POMS scores

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
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<tr>
<td>2. Depression</td>
<td>.812**</td>
<td>1</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3. Anger</td>
<td>.541**</td>
<td>.518**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Fatigue</td>
<td>.437*</td>
<td>.385*</td>
<td>.458**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Confusion</td>
<td>.719**</td>
<td>.720**</td>
<td>.433*</td>
<td>.459**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Vigor</td>
<td>-.383*</td>
<td>-.190</td>
<td>-.353*</td>
<td>-.415*</td>
<td>-.446**</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Post CAB</td>
<td>.133</td>
<td>.034</td>
<td>-.012</td>
<td>-.166</td>
<td>.032</td>
<td>-.133</td>
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</tr>
<tr>
<td>8. Post CAR</td>
<td>-.155</td>
<td>-.290</td>
<td>-.066</td>
<td>.142</td>
<td>.051</td>
<td>-.289</td>
<td>.000</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviation: CAR - Cardiac Autonomic Reactivity, CAB - Cardiac Autonomic Balance
Correlation significant at the *p < .05, **p < .01

Linear regression analysis was used to assess the contribution of relative change in CAB (from rest to recovery) to each subscale of the 24-hour POMS. The three significant regression equations are reported in Table 9. The pre-post exercise change in CAB was a significant predictor (inversely) of the variance in tension (F (1,32) = 6.65, p = .015, with an R^2 = .172), depression, (F (1,32) = 6.15, p = .019, with an R^2 = .161), and confusion, (F (1,32) = 5.09, p = .031, with an R^2 = .137.)
Chapter 5: Discussion

The objective of the current study was to investigate the responses of the parasympathetic and sympathetic nervous systems to muscular endurance exercise, and to determine their influence on mood 24 hours after exercise. Our primary finding is that those subjects who reported an increase in depression, mental confusion, and tension from baseline to recovery (measured 24 hours post exercise) experienced less vagal recovery during post exercise rest (5 min), measured by (relative change in CAB).

During the 1RM, PEP decreased from rest to recovery, representing an increase in sympathetic activity, and a decrease in RSA and RMSSD represented a decrease in parasympathetic activity. Heart rate increased from rest to recovery indicating greater sympathetic activity compared to resting values. Furthermore, throughout the endurance exercise bout PEP decreased from rest to endurance, and increased during recovery. Indicating that an increase in muscular endurance (volume) caused a reciprocal increase in sympathetic activity during exercise, and a sympathetic decrease during recovery. RSA decreased from rest to endurance suggesting a vagal withdrawal during exercise, and increased slightly during recovery. However, even with the slight increase in RSA and RMSSD during recovery, parasympathetic activity remained blunted when compared to baseline values. Likewise to 1RM, heart rate increased from rest to recovery suggesting greater sympathetic dominance over parasympathetic activity. Therefore, even though RSA increased slightly during recovery, sympathetic activity remained predominant.

While baseline vigor correlated with relative change in CAB and CAR during endurance exercise, there were no significant changes statistically speaking in CAB and CAR function from rest to recovery with any mood state variables. Profile of mood states demonstrated a decrease in all variables (tension, depression, anger, fatigue, confusion, vigor) from rest to recovery.
Baseline vigor was inversely related to autonomic function post exercise. Vigor remained high during baseline measurements (M=53.62), however, demonstrated a decrease post exercise (M=52.41). These results are consistent with Nuissier et al. (33), who found that the scores on the depressive and vigor subscales of the POMS were significantly correlated with autonomic dysregulation. In the current investigation, autonomic regulation was assessed by cardiac autonomic balance (CAB) and cardiac regulatory capacity (CAR). After muscular endurance exercise, subjects demonstrated diminished CAB and CAR function. The low CAB after muscular endurance exercise indicated higher sympathetic and lower parasympathetic control subsequent to endurance exercise. It should also be mentioned that SDNN and RMSSD decreased after muscular endurance exercise (rest to recovery). Indicating a decrease in vagal mediation and global HRV, respectively, which has been linked to poor cardiovascular health (30). Suggesting that high load resistance exercise (1RM) coupled with an increased volume (repetitions) endurance exercise may lead to an acutely blunted vagal system.

Subsequently, relative change in RSA, from rest to recovery were calculated by repeated measure ANOVA and correlated to baseline POMS (r = .511, p = .002). Results indicated a moderate direct relationship of vagal activity to mental confusion; revealing a connection concerning non-healthy autonomic balance. Therefore, supporting the hypothesis that vagal change post exercise will show a direct relationship to mood states (mental confusion); strenuous muscular endurance exercise caused reciprocal sympathetic activation, blunting the PSN and effecting mental confusion. RMSSD, a recognized marker for parasympathetic modulation decreased from baseline to recovery (M=70.31 to 25.31) due to a direct change in work load/volume. Coupled with an increase in HR (M=66.55 to 97.73), this indicates a reciprocal change in sympathetic modulation with a blunted vagal tone post-exercise.
This directly supports the study by Rocha et al. (37), who found similar sympathovagal effects as a result of exercise routines with higher work volume. In their study, 13 normotensive male participants who were between the ages of 20 and 30 years participated in two similar RE bouts with an intermission of 48-hours between routines. Routine 1 (R1) consisted of two sets of 10RM; routine 2 (R2) had three sets of 10RM. Routine 2 stimulated an increase in the normalized LF and decreased normalized HF values during the 60-minutes post exercise. Therefore, suggesting that the changes or decrease in cardiovascular function are dependent on set volume. Moreover, a higher amount of repetitions could modulate post-exercise diastolic blood pressure. As heart rate increases during exercise, sympathetic modulation also increases coupled with a reduction of vagal modulation. Therefore, increased work volume will diminish cardiac vagal output, which in turn will decrease DBP post-exercise. In the current study, blood pressure was not measured, so associations between increased endurance exercise repetitions and falling DSP cannot be ascertained. Nonetheless, one can assume that an increase in LFnu (M=.48 to .53) coupled with HFnu withdrawal (M=.43 to .31) post-exercise may reflect a decreased baroreflex activity for homeostatic regulation. Pertaining to the research hypothesis, RSA decreased post exercise and the parasympathetic nervous system did not reestablish to baseline levels. Thus, inversely affecting tension, depression, and mental confusion (POMS subscale).

Cardiac autonomic balance (CAB) demonstrated an inverse relationship with tension, depression, and mental confusion (POMS subscale), conveying that the sympathetic nervous system was still active during recovery phase. As CAB decreased from pre to post exercise, autonomic control shifted from a parasympathetic drive to sympathetic control. Ideally, the physiological framework of the ANS works in a reciprocal manner. However, this is not the case for every individual. Cacioppo et al. (6)
proposed that there are nine possible methods of control, a few of which will be discussed. During baseline, participants experienced elevated RMSSD (parasympathetic) levels (M=70.31). As exercise initiated, the PNS began to slowly withdraw coupled with sympathetic stimulation, known as reciprocal sympathetic activation (6). The concept that ANS regulation functions exclusively by reciprocal sympathetic activation, and reciprocal parasympathetic activation (during recovery) is unfortunately naive and questionable. The complexity to which the body responds and recovers depends on various factors (e.g., respiratory rate, r-r variability, hemodynamics). The current study’s results found that the PNS (RMSSD, CAB, HF) did not restore to baseline levels during the 7 minutes of recovery. Gladwell et al. (14) discovered that vagal activity did not reestablish until 30-minutes post-exercise. This was due to an elevated sympathetic drive during the endurance portion, which carried over to recovery. Findings in the current study were consistent to Gladwell et al. (14), in which sympathetic drive stayed active while parasympathetic activity was blunted during 7-minutes of recovery (post endurance exercise).

A linear regression analysis was used to predict profile of mood states (POMS) by analyzing relative changes in CAB. The results confirmed the hypothesis that CAB would predict tension, depression, and mental confusion (POMS subscale). The results revealed that changes in CAB from pre to post exercise significantly explained the variance in tension (17%), depression (16%) and mental confusion (14%), respectively. Our results were somewhat similar to those of Weinstein et al. (40). These researchers discovered that baseline LF/HF ratio (HRV) predicted the development of negative mood and fatigue states (POMS). However, their study focused on exercise withdrawal, reduced parasympathetic tone (predictive of negative mood and fatigue), and subsequent effects, while the current study explored the influence of muscular endurance exercise on
mood states during recovery. Supporting the Weinstein et al. study (40), blunted PSN tone can predict negative mood; albeit, the current results focused on the effects of a recent bout of exercise, not on exercise withdrawal per se.

**Limitations**

The current study had a few methodological limitations that are important to mention. Sample size is a key determinant for strong correlations and significant results. Although our study had 34 participants, with a larger sample size, there could theoretically be greater variance, furthermore enhancing the results. During the exercise, electrodes attached to the participants began to disengage, causing missed ECG signals, which was associated with artifact in the ECG and in HRV. All artifact, however, was removed to ensure the accuracy of the remaining data. Another limitation of the study is that the leg press machine could be maxed-out by participants, which would then render the 60% 1 RM muscular endurance loads lower than 60% of their actual leg press 1RM. Participants were able to complete many sets until exhaustion, although in some cases, at an intensity lower than 60% of the 1RM. Future research should focus on attaining a true 1RM and cleaner physiological data, by making sure that electrodes have better adherence. Muscular strength exercise should be executed on a separate day from muscular endurance exercise bouts. In the current study, muscular strength and endurance bouts were performed during the same day; consequently, affecting recovery period. Future studies may also benefit by measuring recovery HRV up to 30-minutes post-exercise, which will allow enough time for the PNS to reestablish. However, a hallmark of a healthy ANS is the ability for rapid homeostatic recovery; 5-minute post-exercise recovery period will be the true test for ANS resiliency.
Conclusions

Muscular strength and endurance exercise is associated with changes in autonomic function during exercises such as the leg press, seated row, and chest press. Baseline correlations between CAB (parasympathetic/sympathetic balance) and vigor have been established. Both muscular strength and muscular endurance exercise were associated with significant changes in sympathetic and parasympathetic activity. Statistically significant responses of the ANS were found when comparing recovery to resting values. According to the elevated sympathetic tone post-exercise, it is apparent that muscular strength followed immediately by muscular endurance exercise caused a sympathetic overload, which lead to a depressed parasympathetic tone post-exercise. Correlations between changes in CAB and post-exercise mood states (tension, depression, confusion) provide us with a glimpse of how autonomic recovery after exercise could influence mood. More research should be conducted to further understand the connection between autonomic nervous system function and mood states during bouts of resistance exercise and recovery from exercise. Better understanding of this relationship can lead to better exercise prescription, physiological and psychological maintenance, as well as providing support for the use of resistance training for cardiovascular purposes. According to the current study’s results, it seems that caution should be emphasized for people with autonomic disorders when performing high repetition muscular resistance exercise. This may lead to increased autonomic stress, with dysregulated pitfalls. For now, it is suggested that potential effects from this study are taken into consideration when recommending resistance exercise, mainly muscular endurance, for any individual or population.
References


33. Nuissier, F., Chapelot, D., Vallet, C., Pichon, A. Relations between psychometric profiles and cardiovascular autonomic regulation in physical...


