

The Effect of Exercise on the Cardio-Postural Relationship

by

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ABSTRACT

Syncope is common in individuals who experience orthostatic hypotension which is often associated with cardiovascular conditions, brain injuries, and ageing. A bi-directional link between the cardiovascular and postural control systems was recently identified, and may provide insight into syncope and orthostatic hypotension. This thesis examined the inter-dependent relationship between cardiovascular and postural controls before and after light exercise to induce mild orthostatic stress. It was hypothesized that after exercise, there would be a greater reliance on the skeletal muscle pump to prevent venous pooling to maintain cardiac output and blood pressure, and that this effect would be more pronounced in men. There was an increase in skeletal muscle pump activity which maintained venous return and increased posture stability. In addition, there was a shift in the overall interaction dynamics between the two systems with a greater dependence on posture control to maintain venous return after exercise, particularly in men.

Keywords: cardiovascular control; posture control; skeletal muscle pump; electromyography; orthostatic stress; wavelet transform coherence

The more I learn, the less I know.

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LIST OF ACRONYMS

AP	Antero-Posterior
CO	Cardiac Output
COP	Centre of Pressure
COPx	Centre of Pressure in the Medio-Lateral Plane
COPy	Centre of Pressure in the Antero-Posterior Plane
DBP	Diastolic Blood Pressure
EMG	Electromyography
LF	Low Frequency
ML	Medio-Lateral
SBP	Systolic Blood Pressure
VLF	Very Low Frequency
Zo	Electrical Impedance of Blood in the Lower Leg

1 GENERAL INTRODUCTION AND LITERATURE REVIEW

1.1 Introduction

Orthostatic hypotension is a sudden reduction in blood pressure or central venous pressure commonly associated with either prolonged upright stance or a change in posture from a seated to standing position. This is of particular concern in conditions such as postural orthostatic tachycardia syndrome, mild traumatic brain injury, and ageing, among others, as orthostatic hypotension often leads to syncope (fainting) and falls. According to the Public Health Agency of Canada, the current financial cost of falls in Canada is estimated around \$3 billion annually (2005). One of the primary causes of orthostatic hypotension is thought to be the result of insufficient venous return to the heart associated with venous pooling in the lower extremities that may be the result of inefficiency in the skeletal muscle pump under orthostatic stress (Stewart et al, 2004; Stewart and Montgomery, 2004; Smith et al, 1994).

A new physiological model examining the interactions between cardiovascular and postural controls in relation to orthostatic stress has been proposed (Blaber et al, 2009; Souvestre et al, 2008). This model expands on the concepts of the cardio-locomotor model proposed by Novak et al (2007) based on the notion that forces generated by muscle contraction during locomotion act

as a pump to maintain venous return to the right atrium and on the model of space motion sickness proposed by Lackner and DiZio (2006).

Posture control involves proprioceptive awareness for the dual purpose of stability and orientation – the ability to maintain an appropriate relationship between body segments and the environment (Dichgans and Diener, 1989; Horak and Macpherson, 1996). When exposed to a perturbation, sensory information must be integrated from multiple sources including somatosensory, visual, and vestibular pathways to maintain balance. The weighted contributions of these various sensory inputs to maintain balance are unknown; however, sensory integration for posture control must be flexible to accommodate sensory and environmental changes (McCollum, 1996; Garg, 2010).

In healthy individuals, integration between the cardiovascular and postural control systems under orthostatic stress serves to maintain venous return. However, dysfunction in the mode of integration between these two systems may significantly contribute to orthostatic hypotension and increase the risk of falls.

1.2 Cardiovascular Response to Orthostatic Stress

Under normal conditions, in bipedal, upright stance, approximately 70-75% of an individual's total blood volume is below the heart, primarily in vital organs, the abdomen, and lower legs due to the effects of gravity (orthostatic stress) (Rowell, 1993). This results in increased pressure below and decreased pressure above the heart, and reduced central venous pressure; reduced central venous

pressure due to orthostatic stress may result in orthostatic hypotension (Rowell, 1993).

To compensate for reduced central venous pressure, an immediate increase in heart rate is mediated through vagal withdrawal while the maintenance of tachycardia occurs via slower sympathetic activation (Convertino, 1998; Carter et al, 2001; Gotshall et al, 1991; Jardine et al, 2002). Vasoconstriction is induced by enhanced muscle sympathetic nerve activity, which raises vascular resistance, and, in conjunction with elevating heart rate, serves to maintain blood pressure (Evans et al, 2001; Fu et al, 2004). Sympathetic discharge is transmitted to peripheral vessels via the caudal and rostral ventro-lateral medulla (Robinson and Potter, 1997). Both vagal withdrawal and sympathetic activation are critical for the maintenance of mean arterial pressure and cardiovascular regulation, particularly under conditions such as orthostatic stress (Convertino, 1998; Fu et al, 2004; Fritsch-Yelle et al, 1994; Carter et al, 2001).

Cardiovascular control under orthostatic stress is mediated via arterial baroreceptors, mechanoreceptors located in the carotid sinus, coronary arteries, and aortic arch, which are sensitive to beat-by-beat changes in mean arterial pressure and pulse pressure under conditions such as hypovolemia and orthostatic hypotension (Jacobsen et al, 1993; Rowell, 1993; Carter et al, 2001; Convertino, 1998). Orthostatic hypotension results in a rise in sympathetic nerve activity via selective baroreceptor unloading, which consequently causes a redistribution of blood from the splanchnic region (via changes in vascular

capacitance) and lower extremities (via changes in vascular resistance) (Rowell, 1993; Laszlo et al, 1998; Jezova et al, 2004). Greater levels of orthostatic stress yield further baroreceptor unloading and increased sympathetic nerve activity; this induces a rise in heart rate and peripheral resistance (Baily et al, 1990; Laszlo et al, 1998; Pawelczyk and Raven, 1989). Conversely, increased arterial pressure and arterial baroreceptor activity have the opposite effect, and cardiovascular control of heart rate shifts from sympathetic to vagal control (Carter et al, 2001; Gotshall et al, 1991; Fritsch-Yelle et al 1994). These compensatory mechanisms are necessary to maintain adequate central venous pressure and cardiac output in the presence of orthostatic stress to prevent hypotension and syncope.

1.3 Gender Differences in Cardiovascular Regulation and Orthostatic Tolerance

While men and women both exhibit similar cardiovascular regulatory reflexes (outlined in section 1.2) to maintain venous return and cardiac output under orthostatic stress, the underlying reflex mechanisms vary between genders, and women exhibit a lower tolerance to orthostatic stress than men.

It has been well documented that women exhibit a lower tolerance to orthostatic stress than men (Fu et al, 2004; Convertino, 1998; Fritsch-Yelle et al, 1994; Jardine et al, 2002; Carter et al, 2001; Gotshall et al, 1991). Studies suggest that women respond to orthostatic stress with a greater vagal withdrawal-mediated increase in heart rate, while men rely on greater sympathetic stimulation to peripheral vasculature (Evans et al, 2001; Shoemaker

et al, 2001; Frey et al, 1986; Frey et al, 1988). This was based, in part, on observations that women demonstrated greater increases in heart rate with a lower increase in total peripheral resistance than men (Convertino, 1998; Montgomery et al, 1977; White et al, 1996). However, Fu et al (2004) found no difference in peripheral vascular resistance or norepinephrine concentrations between genders at presyncope, and suggest that the elevation in heart rate in women is to counteract the greater reduction stroke volume. The reduction in stroke volume would affect blood flow through baroreceptive arteries which modulate baroreceptor activity (Fu et al, 2004; Rowell, 1993), and, therefore, sympathetic and parasympathetic cardiovascular regulation.

The effects of the menstrual cycle in women may account for some of the observed differences in cardiovascular regulation between men and women and responses to orthostatic stress.

1.3.1 Effect of the Female Menstrual Cycle on Cardiovascular Regulation and Orthostatic Tolerance

There are conflicting reports on the effect of variations in hormone levels (estrogen and progesterone) across the menstrual cycle and oral contraceptive use on cardiovascular regulation and response to orthostatic stress. Variations in hormone levels across the menstrual cycle have been shown to influence sympathetic baroreflex sensitivity (Minson et al, 2000; Saeki et al, 1997), plasma volume (Oian et al, 1987), muscle sympathetic nerve activity (Minson et al, 2000), and resting core body temperature (Hessemer and Bruck, 1985 (1 & 2)), among others. Such changes in cardiovascular regulation could contribute to

changes in orthostatic tolerance across the menstrual cycle, and the increased rate of orthostatic intolerance in women (Meendering et al, 2005). Alternatively, other studies have reported changes in cardiovascular regulation across the menstrual cycle phases, but found no differences in the net response to orthostatic stress (Hirshoren et al, 2002; Meendering et al, 2005).

1.3.2 Influence of Oral Contraceptives on Cardiovascular Regulation in Women

There are conflicting results on the influence oral contraceptives have on cardiovascular regulation. Studies have shown that women who used oral contraceptives had reduced baroreflex sensitivity during the high hormone phase of the menstrual cycle compared to women with normal cycles (Minson et al, 2000; Minson, 2004). However, other recent studies have reported that the use of oral contraceptives did not alter cardiovascular or sympathetic neural responses to orthostatic stress compared to women with normal cycles (Carter et al, 2010; Edgell et al, 2012).

1.4 Postural Control

Postural stability is the ability to maintain an upright stance through the integration of the visual, vestibular, and somatosensory systems (Horak and Macpherson, 1996; Dichgans and Diener, 1989). While the contribution of visual and vestibular cues to postural stability during quiet stance have been studied extensively (Peterka, 2002; Day et al, 1997; Dijkstra et al, 1994; Johansson et al, 1995; Fitzpatrick et al, 1994), the weighted contribution of each system to the control of postural stability has yet to be determined. However, it is clear that

there is a redundancy or re-weighting within the sensorimotor integration which can be observed when an individual is able to maintain balance with the removal of any one of these inputs, for example, when eyes are closed, in patients with loss of unilateral or bilateral vestibular function, or with reduced proprioceptive feedback by means of a body sway-referenced platform (Peterka, 2002; Winter et al, 1998; Winter et al, 2001; Mergner et al, 2003; Horak and Macpherson, 1996).

Bipedal upright stance is inherently unstable given that a slight deviation from a perfect postural stance results in a torque due to gravity that accelerates the body farther away from the original position (Peterka, 2002). This torque must be counteracted by a corrective torque generated within the body and exerted through the feet against the ground in order to maintain upright stance (Peterka, 2002).

One of the primary theories regarding postural stability is that corrective torque is generated by sensorimotor feedback control mechanisms that involve time delays due to sensory transduction, transmission, processing, and muscle activation to provide joint stabilization and control posture during quiet stance (Peterka, 2002; Horak and Macpherson, 1996; Johansson and Magnusson, 1991). Another theory suggests that feedforward mechanisms, in conjunction with feedback control, may also serve to predict and stabilize posture (Winter et al, 1998; Winter et al, 2001). While the mechanisms of postural stability remain under debate, it is evident that complex sensorimotor integration occurs among

the visual, vestibular, somatosensory, and musculoskeletal systems to provide optimal joint stability and posture control.

1.5 Cardio-Postural Relationship and Orthostatic Intolerance

The cardiovascular and postural control systems have been studied extensively as independent systems. However, until recently, little consideration was given to the integration of these two systems, the importance of the skeletal muscle pump in the maintenance of venous return and cardiac output under orthostatic stress, and how this relationship may be impaired in individuals with orthostatic hypotension or postural syncope.

A study by Claydon and Hainsworth (2005) revealed that certain individuals who displayed poor orthostatic tolerance but had no history of syncope or pre-syncope were likely able to maintain venous return due to increased postural sway in upright stance. These individuals demonstrated increased antero-posterior (AP) sway over time with a slight increase in medio-lateral (ML) sway. The calf muscles in the lower leg are primarily responsible for AP sway while the thigh muscles control ML sway (Soames and Atha, 1981; Winter et al, 1996). Since the calf muscles would be exposed to the greatest venous pressures when upright, enhanced pumping by these muscles should yield the greatest effect on venous return (Claydon and Hainsworth, 2005; Keissar et al, 2009). A conceptual model of cardio-locomotor coupling during walking was proposed by Novak et al (2007) based on the notion that forces generated by skeletal muscle contraction

during locomotion act as a pump, rhythmically propelling venous blood to the right atrium.

A physiological model examining the interactions between cardiovascular and postural controls in relation to orthostatic intolerance after long-duration spaceflight has been proposed (Blaber et al, 2009; Souvestre et al, 2008). This model was based on the cardio-locomotor model proposed by Novak et al (2007), and a model of space motion sickness proposed by Lackner and DiZio (2006). The space motion sickness model incorporated adaptation to changes in the environment, physiological stress, posture and movement, and sensorimotor integration to explain the phenomenon of space motion sickness (Lackner and DiZio, 2006). The cardio-postural model has since been modified as a ground-based model that examines cardiovascular and postural control interactions in response to orthostatic stress (Figure 1-1).

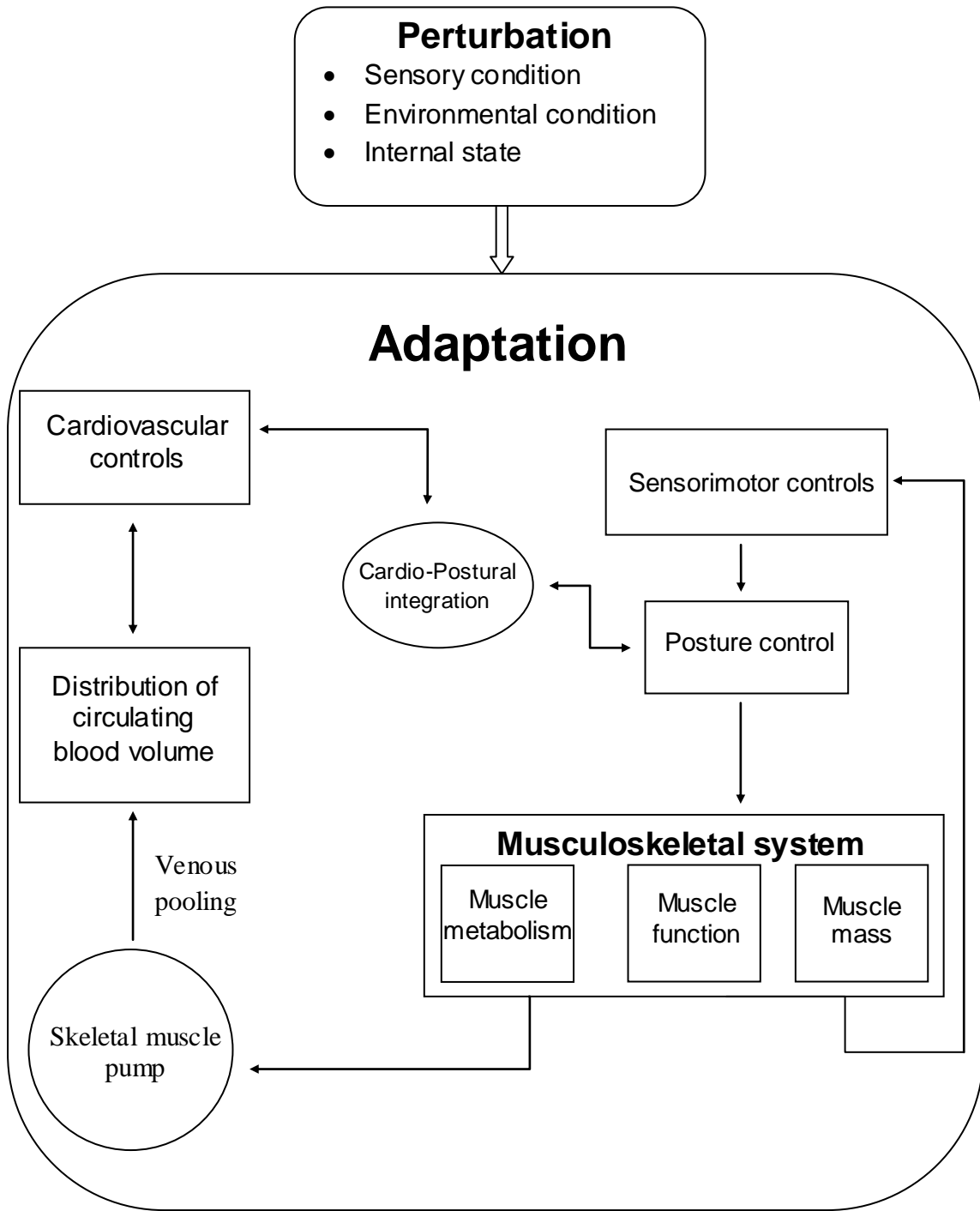


Figure 1-1: Ground-based cardio-postural model that demonstrates the inter-dependent relationship between the cardiovascular and postural control systems adapted from Souvestre et al (2008).

The cardio-postural model and the presence of a bi-directional relationship between cardiovascular and postural controls were further examined by Garg (2010). Through the comparison of the behaviour of lower leg electromyography (EMG) activity with systolic blood pressure, diastolic blood pressure, cardiac output, and postural sway centre of pressure trajectory, respectively, a frequency-dependent relationship between cardiovascular and postural control system variables was identified over the course of a 5-minute stand test (Garg, 2010). The frequency-dependent, bi-directional relationship between these two systems has yet to be fully characterized.

Data indicated a correlation between postural sway and blood pressure, and between lower leg muscle EMG and blood pressure variation (Garg, 2010). Increased EMG activity was associated with increased systolic blood pressure and decreased EMG activity with a decline in blood pressure (Garg, 2010). There was a significant association of frequency with the coupling between EMG and cardiac output, and EMG and systolic blood pressure which indicated a frequency-specific interaction between the cardiovascular and postural control systems (Garg, 2010). Further investigation into the bi-directional cardio-postural relationship is required to fully understand the interaction dynamics between the two systems and the mechanisms underlying the frequency-specific interactions.

1.6 Cardiovascular Response to Sub-Maximal Exercise

In order to further evaluate the cardio-postural relationship, a controlled perturbation must be introduced into the model to allow evaluation of the dynamic

cause and effect responses between the two systems. The cardiovascular response to sub-maximal exercise has been well documented and provides a controlled means to stress the cardiovascular system and observe the cardio-postural response.

At the onset of exercise, there is a sudden increase in the demand for oxygen by working muscle. A series of cardiovascular changes occur to redistribute blood flow in order to meet this sudden demand for oxygen. A sudden increase in skeletal muscle metabolic rate causes an increase in vascular conductance and a reduction in vascular resistance (total peripheral resistance), which leads to an increase in the arterio-venous pressure difference (Laughlin, 1999; Rowell, 1993; Hainsworth, 2004; Navare and Thompson, 2003). Contraction of working muscle serves as a pump by further increasing venous driving pressure toward the heart, thereby increasing venous return (Rowell, 1993; Carter et al, 2001; Halliwill, 2001). Increased venous return leads to increased ventricular filling pressure, a reduction in end-systolic volume, and an increase in left ventricular end diastolic volume (Laughlin, 1999; Hainsworth, 2004; Navare and Thompson, 2003; Kirkman, 2007). In accordance with the Frank-Starling law, this serves to increase stroke volume, which leads to an increase in systolic blood pressure and cardiac output. Heart rate will continue to rise as the work rate increases and contributes to the rise in cardiac output. Control of heart rate shifts from vagal withdrawal to sympathetic activation as heart rate exceeds approximately 100 beats per minute (Carter et al, 1999). Increased muscle sympathetic nerve and plasma renin activity increase vascular

resistance in the viscera, which further redirects blood flow to active skeletal muscle (Laughlin, 1999; Rowell, 1993; Carter et al, 1999). All of these mechanisms are integrated and work together to maintain blood pressure and blood flow to vital organs, in addition to meeting the demand for oxygen by working muscle.

These same mechanisms that uphold cardiovascular regulation during exercise can lead to post-exercise hypotension during inactive recovery (in which there is no cool-down following a bout of exercise) (Carter et al, 2001; Carter et al, 1999; Halliwill, 2001). Therefore, inactive recovery following sub-maximal exercise may provide a controlled method to assess cardiovascular and postural control systems interaction to prevent orthostatic hypotension.

1.7 Effect of Exercise on Postural Stability

During exercise, movement facilitates venous return through the contraction of lower extremity muscles, which is necessary to maintain adequate stroke volume and cardiac output. Upon cessation of exercise, cardiovascular variables remain above resting baseline values. In order to prevent venous pooling and orthostatic hypotension, the skeletal muscle pump may be activated. Previous research demonstrated a correlation between changes in skeletal muscle activity in the lower legs with changes in postural sway (Garg, 2010). Studies that assessed postural stability after exercise observed increased postural sway after exercise which was attributed to local (musculoskeletal) or central (cardiovascular) fatigue. These studies did not consider the effect of increased

muscle activity due to skeletal muscle pump activation on postural stability after exercise.

The consensus on the effect of local (musculoskeletal) and central (cardiovascular) fatigue on postural stability has been progressively changing. Initial reports that examined the effect of fatigue on balance indicated that significant reductions in postural stability occur immediately after exercise but do not extend beyond 5 minutes after the cessation of exercise (Nardone et al, 1998). The rate of local muscle recovery (contractility, firing rate, and force production) after fatigue is currently estimated between 1 to 3 minutes (Bigland-Ritchie et al, 1986; Hakkinen and Komi, 1983; Woods et al, 1987). On the contrary, recent studies using both traditional measures of postural stability such as the Balance Error Scoring System test and complex analyses of changes in centre of pressure trajectory, sway path, sway area, and postural control strategies have revealed significant reductions in postural stability up to 15-20 minutes after the cessation of both submaximal (aerobic and anaerobic) and maximal exercise (Corbeil et al, 2003; Nardone et al, 1997; Susco et al, 2004; Wilkins et al, 2004).

This progressive recovery of postural stability after exercise may be correlated with the period during which the cardiovascular system returns to steady state, specifically heart rate, stroke volume, and blood pressure. Only one study that examined postural stability after exercise (Nardone et al, 1998) also reported cardiovascular measures post-exercise; however, that study made no correlations between reported heart rate and postural stability outcomes.

1.8 Summary

While the responses of the cardiovascular system and posture control to various types of orthostatic stress have been studied individually, few attempts have been made to determine the interaction characteristics between these two systems. Recent data from our laboratory (Garg, 2010) confirmed the existence of a bi-directional relationship between cardiovascular and postural control systems, but additional research is required to fully characterize the dynamic interaction between these systems. Inactive recovery after aerobic exercise may be used to stress the cardiovascular system, induce mild orthostatic stress, and increase the cardio-postural interaction characteristics as these systems work together to prevent orthostatic hypotension. An increased understanding of the cardio-postural relationship will provide insight into conditions such as orthostatic hypotension, syncope, and falls, and may lead to more effective treatment or prevention of said conditions.

1.9 Purpose

Based on previous research in our laboratory, there is a frequency-dependent interaction between cardiovascular and postural controls (Garg, 2010). The primary objective of this study was to determine the effect of inactive recovery following light aerobic exercise on the established relationship between cardiovascular and postural control systems. The secondary objective of this study was to determine how the outcome characteristics of the cardio-postural model varied between men and women in response to the exercise protocol.

1.9.1 Pre- and Post-Exercise Test Hypotheses

It is hypothesized that after exercise, there will be a greater reliance on the skeletal muscle pump to maintain venous return during upright stance. Increased activation of the skeletal muscle pump will result in increased lower extremity muscle activity (measured through EMG), which will cause a corresponding increase in centre of pressure trajectory (COP) in both medio-lateral and antero-posterior postural sway. After exercise, there will also be increased coherence between individual variables representative of postural control (EMG and COP) with corresponding variables representative of the cardiovascular system (cardiac output, systolic blood pressure, diastolic blood pressure, and blood volume in the lower leg).

While there will be an alternating pattern in the driving behaviour between the variables representative of the two systems, the cardiovascular system will be the dominant driving factor within the cardio-postural model as changes in venous pooling and cardiac output will precipitate increased activation of the skeletal muscle pump.

1.9.2 Hypotheses for Male and Female Comparison

Because cardiac output and orthostatic tolerance are higher in men than women (Fu et al, 2004; Convertino, 1998; Fritsch-Yelle et al, 1994; Jardine et al, 2002; Carter et al, 2001; Gotshall et al, 1991), men should yield a higher venous return per unit increase in skeletal muscle activity, particularly after exercise. However, the overall characteristics of the cardio-postural interactions should not differ between genders.

2 METHODS

2.1 Experimental Design

This study was approved by the ethics review board at Simon Fraser University as minimal risk.

2.1.1 Participants

Twenty young male and female (10M/10F) participants aged 19-30 were recruited from the local university population, and provided written informed consent prior to participation. Participants had no history of cardiovascular, respiratory, or neurological disease, major musculoskeletal injuries, or hormone imbalance as specified in the Medical History Form in Appendix A. The use of prescription medications and naturopathic remedies were reported, and participants taking any substance that could alter cardiovascular regulation or postural stability were excluded. All participants were instructed to refrain from exercise and caffeine consumption for 24 hours prior to the experiment.

2.1.2 Experiment Protocol

Upon arrival in the lab, participants were asked to remove their socks, shoes, and all items from their pockets. Height, weight, general medical history, and present medications were recorded. Female participants were asked to report the use of prescription contraceptives and the number of days since their

last menstruation to determine the phase of the menstrual cycle at the time of testing. Women were not tested during a particular phase of the menstrual cycle.

Participants were fitted with 1) non-invasive blood pressure monitor, 2) three-lead ECG, 3) surface EMG electrodes on four bilateral lower leg muscles, 4) impedance plethysmography of the left lower leg; equipment and electrode placement are discussed in section 2.1.3. After all equipment and electrodes were in place, the participant was seated next to the force platform with their feet placed parallel, 5 cm apart on the centre of the platform, and knees and hips at a 90° angle (Figure 2-1). The signal authenticity was then verified, and any necessary adjustments were made prior to data collection.

Participants were seated quietly with arms relaxed by their sides for 5 minutes, after which assistance was provided to transition into upright stance for an additional 6 minutes. They were instructed to keep their eyes closed, maintain imaginary eye-level gaze, and not to alter foot placement. All testing took place in a sensory-minimized environment – a dark room with black drapes in front of the force platform with minimal ambient noise.

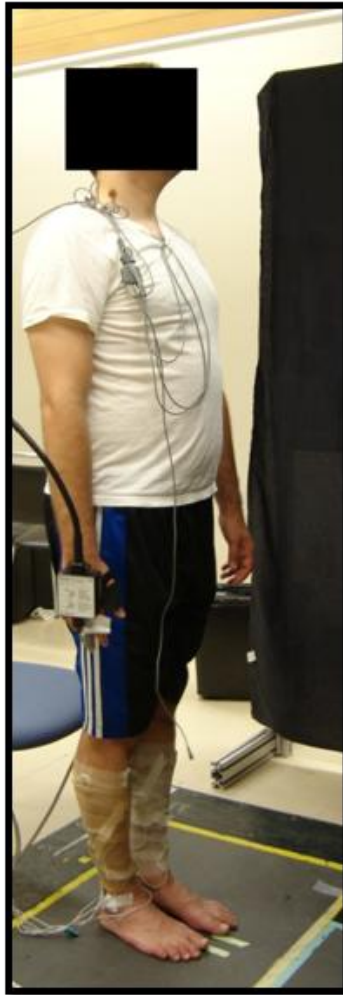
After the sit-to-stand test, participants were seated comfortably on a cycle ergometer to carry out a 12-minute, sub-maximal exercise protocol. The exercise protocol consisted of a 2-minute warm-up at 25W, followed by 10 minutes at 80W or 100W for female and male participants, respectively. Participants were instructed to maintain 70 RPM throughout the duration of the exercise protocol. This protocol was created to induce mild stress on the cardiovascular system

without crossing the aerobic threshold and limited the risk of musculoskeletal fatigue. No data were collected during the exercise period.

Immediately upon cessation of exercise, a 6-minute stand test was conducted with eyes closed, forward gaze, and identical pre-test foot placement on the force platform. It took approximately 30 seconds to transition from the cycle ergometer to the force platform and initialize data acquisition.



A



B



C



D



Figure 2-1: Instrumentation and participant positioning. (Centre) Profile view. (A, B) EMG electrode placement on the posterior (A) and lateral (B) aspects of the lower legs. (C) BP finger cuff secured to the third finger and wrist. (D) Representation of the parallel foot placement on the force platform with 5 cm between the instep of each foot.

2.1.3 Equipment and Signal Acquisition

Electromyography: Surface electromyography (EMG) was obtained from four bilateral lower leg muscles: tibialis anterior, lateral soleus, and medial and lateral gastrocnemius. Transdermal differential recording of the signals was performed using the *Bagnoli-8* (Delsys Inc, MA, USA) EMG system. Electrodes were placed distal to the origin of the muscle approximately 1/3 of the muscle length along the approximated pennation angle for the gastrocnemius and tibialis anterior; the soleus electrode was placed inferior to the gastrocnemius and lateral of the Achilles tendon. The sites for electrode placement were chosen based on recommendations from the SENIAM project (Hermens et al, 1999). For smaller participants, EMG electrode placement was adjusted as needed to accommodate the impedance plethysmography electrodes; the integrity of both EMG and impedance plethysmography signals was verified prior to data collection.

Electrocardiography: ECG signals were acquired with custom equipment from *LifePak 8* (Medtronic Inc, Minnesota, USA) in a standard Lead II electrode configuration with single Ag/AgCl electrodes.

Blood Pressure: Blood pressure (systolic (SBP) and diastolic (DBP)) and cardiac output (CO) were monitored continuously through a non-invasive photoplethysmography finger cuff placed on the left 3rd finger from *Finometer Model 1* (FMS, Amsterdam, Netherlands).

Impedance Plethysmography: Continuous changes in electrical impedance (Z_0) in the left lower leg were measured via *NCCOM 3*

Cardiodynamic Monitor (BioMed Inc, CA, USA) with Ag/AgCl electrodes. Impedance provided a representation of regional blood volume based on the electrical resistance properties of blood. Two pairs of electrodes were placed on the posteromedial and posterolateral aspect of the lower leg inferior to the knee joint, and two pairs next to the medial and lateral malleoli, along the same longitudinal axis as the superior electrode pairs. Each electrode in a pair was placed 5 cm apart along the longitudinal axis.

Postural Sway: Postural sway data, centre of pressure trajectory (COP) coordinates derived from force and moment data in the ML (COPx) and AP (COPy) planes were obtained from an *Accusway Plus Force Platform* (AMTI, MA, USA).

Cycle Ergometer: The exercise protocol was performed on a digital *Jaeger ER 800* cycle ergometer (Wuerzburg, Germany).

2.1.4 Data Acquisition and Data Collection

Data were acquired through a National Instruments data acquisition platform installed on an Intel Pentium desktop computer with Windows operating system and Labview 8.2 software (National Instruments Inc, TX, USA). This system was configured to acquire all 21 channels of data at a sampling rate of 1000Hz, 16-bit analog-to-digital conversion, and output all data into corresponding text files. Individual files were created for each trial, designated by participant number and trial number.

2.2 Analysis Methods

2.2.1 Data Pre-Processing

In accordance with previous work done in our laboratory (Garg, 2010), data pre-processing was conducted with MATLAB 2009b (Mathworks Inc, MA, USA). All data were converted and filtered with a Butterworth low-pass filter of fourth order with a cut-off frequency at 20Hz for the frequency range of interest. The R-waves of the ECG waveform were detected, and the corresponding time-mapped systolic blood pressure time series were generated. All data were re-sampled at 10Hz using interpolation prior to further analyses.

2.2.2 Validation of Wavelet Transform Coherence

In order to investigate the bi-directional behaviour between signal pairs, a common frequency range was first identified. Previous research in our lab (Blaber et al, 2009) demonstrated a relationship between medio-lateral postural sway and blood pressure; significant coherence was observed in the corresponding frequency peaks of 0.03 and 0.07 Hz when the classical coherence method was applied. Therefore, two frequency bands which encompass this region were selected in the present analyses: low frequency (LF: 0.1–0.05Hz) and very low frequency (VLF: 0.05-0.01Hz).

The coherence function is a method used to assess the existence and strength of linear coupling (in which the two signals demonstrate similar behaviour) between two signals in a specified frequency domain (Kay 1988). Classical coherence and correlation methods have been used to investigate the relationship between signals; however, signal stationarity is assumed. This

stationarity assumption removes key characteristics within the signals as physiological adjustments are made to maintain homeostasis (Garg, 2010).

Many physiological signals, namely EMG and blood pressure, have been identified as non-linear and non-stationary in nature (Padmanabhan and Puthusserypady 2004; Voss et al 2009). Furthermore, the interaction between the cardiovascular and postural control systems is both time- and pulse-dependent; thus, requiring a more sophisticated approach from the classical time series of Fourier analysis (Garg, 2010).

Wavelet transform coherence is a signal analysis tool for random-like signals created by complex mechanisms, and is used to find transient correlations between signals that are uncorrelated a majority of the time (Garg, 2010). The wavelet transform coherence method provides information on the strength of coherence as a time-frequency map, which permits identification of related signal features over specific frequency zones and time points. Desired resolution can be obtained simultaneously for each signal feature; higher temporal resolution for higher frequencies, and higher spatial resolution for lower frequencies (Garg, 2010).

Prior to utilizing the wavelet transform coherence method, it was validated as an objective tool to identify the relationship between primary signals of the cardiovascular and postural control systems (SBP and EMG). The complete mathematical procedures and validation process were performed by Garg (2010) and may be found in Appendix B. Simulated EMG and SBP signals were created that closely resembled the actual physiological signals. The coherence threshold

was established using simulated, uncoupled signal pairs. Bias, standard deviation, and false negative rate were calculated over 1000 iterations of the simulated signals to establish baseline characteristics of the method for the signals under investigation (Garg, 2010).

The theoretical coherence estimation was based on the model of a single input, single output of a linear time invariant system (Pinna and Maestri, 2001). The coherence was estimated between each input/output pair and averaged over iterations to give a coherence time series; the empirical sampling distribution (frequency histogram) was computed for each frequency band. The threshold for zero coherence was set at the $100(1-\alpha)$ percentile of the coherence sampling distribution, where α is the significance level of the statistical test kept at 95% confidence or 0.05.

Bias, standard deviation, and false negative rate of the coherence estimator were calculated for different wavelet coefficients, $\omega_0 = 6, 10, 15, 20,$ and 30, where the Morlet coefficient, ω_0 , defines the balance between frequency and time resolution. This was completed for the simulated EMG and SBP signals independently. Minimum bias and false negative rate, and low standard deviation were observed for $\omega_0 = 6$ across all coherence levels in both frequency bands. The threshold of coherence for $\omega_0 = 6$ was then determined to be 0.1894 and 0.3162 for the LF and VLF bands, respectively.

2.2.2.1 Phase angle and phase lock

Phase dependence between two signals was determined by wavelet phase estimation; phase information was derived from the cross wavelet power estimator. This provided a time-frequency map of the change in phase angle between the two signals under consideration. Averaging over scales corresponding to the LF (0.1-0.05Hz) and VLF (0.05-0.01Hz) bands yielded the phase variation in each frequency region over time (Garg, 2010).

The phase signal was differentiated to obtain the rate of change of the phase angle to determine whether two signals were phase locked; a rate of change of phase angle equal to zero indicated a constant phase angle. A Student's *t*-test was used to determine whether the differentiated phase angle was statistically different from zero. Phase difference was defined in relation to EMG or impedance (Z_o) represented as EMG-signal phase or Z_o -signal phase, respectively. A negative phase difference indicated EMG or Z_o lagged behind the secondary signal; a positive phase difference indicated EMG or Z_o led the secondary signal.

Two signals were considered phase locked when the phase difference between them remained constant, and were considered to be inter-dependent. Signal pairs were considered phase locked when the rate of change of phase angle fell within the resultant 95% confidence interval. In regions where the signal pairs were phase locked, characteristic periods of phase lead and phase lag were identified when the signals were above the threshold of significant coherence. This was performed on all signal pairs in both frequency bands.

The final step of the validation process was to apply the wavelet transform coherence method to real physiological data acquired during a 5 minute sit-to-stand test (Garg, 2010). This method identified periods of phase lock during significant coherence between signal pairs with an alternating phase lead/lag pattern across the time series, which suggested a bi-directional relationship between the cardiovascular and postural control systems (Garg, 2010).

2.2.3 Statistical Measures

The data were filtered and processed in accordance with the wavelet transform coherence method described previously in sections 2.2.1 and 2.2.2. Two- and three-way repeated measures ANOVA were performed on a) the overall mean value and variance of each signal after pre-processing, b) mean value of the variables of the transfer function for each signal pair, c) transfer function gain, d) mean phase angle when signal pairs were phase locked (phase lead and lag), e) average value of coherence, f) percent time above the threshold of coherence, and g) percent time signal pairs were phase locked above significant coherence to determine statistical differences between gender (M/F), test (pre-/post-exercise), and frequency band (LF/VLF). The significance level for all statistical analyses was fixed at $\alpha = 0.05$; all analyses were performed with JMP statistical software (SAS, Inc).

3 INTERACTION CHARACTERISTICS BETWEEN CARDIOVASUCLAR AND POSTURAL CONTROLS

3.1 Introduction

The cardiovascular and postural control systems have been studied extensively as independent systems. Evidence suggests the presence of a direct interaction between these two systems through the skeletal muscle pump which serves to maintain venous return in the presence of orthostatic stress and prevent orthostatic hypotension (Garg et al, 2010; Claydon and Hainsworth, 2005; Novak et al, 2007). This study was derived to further examine the cardio-postural interactions and the dynamic mechanisms involved to prevent orthostatic hypotension during inactive recovery immediately following light aerobic exercise in men and women.

3.2 Participant Selection

Twenty young, healthy adults (10M/10F) from the local community participated in the study. Participants confirmed that they refrained from caffeine or alcohol consumption and exercise within 24hours prior to the test. All participants completed a medical history form to screen for a) cardiovascular conditions, b) neurological conditions, c) psychosocial conditions, d) musculoskeletal conditions, e) hormone imbalance, and f) prescription medications. Female participants also provided information on their menstrual cycle to determine which phase of the cycle they were in on the day of the test.

Testing of female participants was not targeted to coincide with a particular phase of the menstrual cycle. Written, informed consent was obtained prior to data collection. All ethics documentation may be found in Appendix A.

Data from 4 participants were not used due to corruption of the data during the experiment. Therefore, only data from the remaining 16 participants (8M/8F) were analyzed (Table 3-1, Table 3-2). Of these 16, diastolic blood pressure data from one participant's (#15) post-exercise stand test was not used due to an error during interpolation of the data. The final 2 minutes of data for the post-exercise test were lost for a second participant (#13) due to a computer malfunction. The post-exercise stand test data for this participant were analyzed in accordance with the abbreviated 4-minute time scale and were included in the statistical analyses. Based on previous research in the laboratory (Garg 2010) and data reported in the literature, a sample size of 16 was sufficient to determine significant interactions (Appendix C).

Table 3-1: Anthropometric data for all participants

Participant #	Gender	Age (yrs)	Height (cm)	Weight (kg)
1	M	25	177	63
2	F	28	167	62
3	F	22	172	61
4	M	27	177	74
5	M	24	181	87
6	F	23	172	66
7	F	25	167	57
8	M	28	180	70
9	M	23	168	52
10	M	23	191	81
11	F	24	157	61
12	M	27	180	78
13	F	30	165	55
14	F	22	162	58
15	F	28	163	54
16	M	28	177	86
Mean	M	25.5±2	178.9±6.4	74±12
	F	25±3	165.6±5.1	59±4
	M + F	25±2.5	172.3±8.8	67±11

Table 3-2: Self-reported menstrual cycle data for female participants

Participant #	Cycle Phase (day #/total)	Projected Cycle Phase	Use of Oral Contraceptive
2	17/28	early luteal	No
3	25/28	late luteal	Yes
6	19/30	mid luteal	No
7	10/30	follicular	Yes
11	10/25	follicular	No
13	21/28	mid luteal	No
14	15/21	follicular	No
15	31/33	late luteal	No

All female participants self-reported normal menstrual cycle and no known hormone imbalance. Cycle Phase reported as the day in the menstrual cycle at the time of the test / total menstrual cycle duration.

3.3 Results

3.3.1 Mean and Variance of Individual Signals

The overall mean value of each variable (Table 3-3) and variance (Table 3-4) were determined prior to the wavelet transform coherence analyses. The mean value of the EMG signal was higher during the post-exercise stand test ($p < 0.001$) with a reduction in variance ($p < 0.05$); both mean and variance were higher in men than in women ($p < 0.05$). There was no effect of exercise or gender on mean Z_0 (impedance) or variance. Mean cardiac output was higher in men than in women ($p < 0.001$) both pre- and post-exercise ($p < 0.05$) with no change in variance. Mean systolic blood pressure decreased while variance increased after exercise ($p < 0.05$) with no difference between genders. There was no main effect of exercise or gender on mean diastolic blood pressure or variance. There was no effect of exercise or gender on the overall mean centre of pressure or variance in the medio-lateral or antero-posterior direction.

Table 3-3: Overall mean of cardio-postural parameters for males and females before and after sub-maximal exercise

	F	M	Pre	Post
EMG *	0.003 (0.0002)Y	0.003 (0.0002)Y	0.002 (0.0002)	0.004 (0.0002)
SBP *	110(3)	113(3)	114(2)	109(2)
DBP	69(2)	70(2)	70(2)	69(2)
CO †	4.85(0.217)	6.27(0.217)	5.40(0.173)ψ	5.72(0.173)ψ
Zo	-0.0009 (0.0004)	-0.0009 (0.0004)	-0.0010 (0.0004)	-0.0009 (0.0004)
COPx	-0.001 (0.002)	0.0008 (0.002)	-0.0009 (0.002)	0.0006 (0.002)
COPy	-0.029(0.005)	-0.036(0.005)	-0.030(0.004)	-0.034(0.004)
Reported as least square mean (SEM) Units for reported variables: EMG (V); SBP (mmHg); DBP (mmHg); CO (L/min); Zo (Ω); COP(m) * Significantly different between tests † Significantly different between genders Y Significantly different between tests within same gender ψ Significantly different between genders within same test				

Table 3-4: Variance of cardio-postural parameters for males and females before and after exercise

	F	M	Pre	Post
EMG *†	0.0013 (0.0007)	0.0035 (0.0007)	0.0013 (0.0007)	0.0036 (0.0007)
SBP *	42.30 (9.88)	55.18 (9.88)	43.16 (9.88)	54.32 (9.88)
DBP	16.15 (2.64)	19.53 (2.55)	19.00 (2.34)	16.68 (2.43)
CO	0.397 (0.100)	0.432 (0.100)	0.331 (0.100)	0.498 (0.100)
Zo	0.0007 (0.001)	0.0025 (0.001)	0.0016 (0.0007)	0.0015 (0.0007)
COPx	0.0106 (0.008)	0.0312 (0.008)	0.0269 (0.006)	0.0149 (0.006)
COPy	0.0288 (0.013)	0.0571 (0.013)	0.0507 (0.010)	0.0352 (0.010)
Reported as least square mean (SEM) Units for all reported variables: au * Significantly different between tests † Significantly different between genders				

3.3.2 Skeletal Muscle Pump

3.3.2.1 EMG-Zo

For the EMG-Zo transfer function when coherence was above threshold, mean EMG during phase lock (lead and lag) increased from pre- to post-exercise ($p < 0.001$) while mean Zo during phase lock only showed a significant decline ($p < 0.05$) post-exercise in men (Table 3-5, Table 3-6). EMG-Zo transfer gain ($\Delta Z_o / \Delta \text{EMG}$) during phase lock (lead and lag) was higher in the LF band than the VLF band ($p < 0.001$) in men ($p < 0.05$) and post-exercise ($p < 0.05$) during phase lock (lead and lag), with no difference in the main effect of exercise or gender (Table 3-7; Figure 3-1).

There was no main effect of exercise, frequency band, or gender on the average phase difference between EMG and Zo. The magnitude of the average phase angle during phase lock (lead) for EMG-Zo was higher ($p < 0.001$) in the LF band than in the VLF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$). The magnitude of the average phase angle during phase lock (lag) for EMG-Zo increased ($p < 0.05$) post-exercise, and was higher ($p < 0.05$) in the LF band than the VLF, specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-8; Figure 3-2). A plot of the coherence time series over the test duration overlaid with periods of phase lock for an individual participant demonstrated the alternating behaviour between phase lead and lag and the shift in behaviour from pre- to post-exercise (Figure 3-3).

Average coherence for EMG-Zo was lower ($p < 0.001$) in the LF band in both genders ($p < 0.05$) compared to the VLF band, and decreased after exercise

in women ($p < 0.05$) and in the LF band ($p < 0.05$; Table 3-9). The percent time above significant coherence for EMG-Zo was higher ($p < 0.05$) in the LF band than in the VLF band both pre- and post-exercise ($p < 0.05$); percent time declined after exercise in women ($p < 0.05$) and in the VLF band ($p < 0.05$; Table 3-10). The percent time EMG and Zo were in phase lock above significant coherence was lower ($p < 0.05$) in the LF band than in the VLF band in women, and decreased post-exercise in the VLF band ($p < 0.05$; Table 3-11).

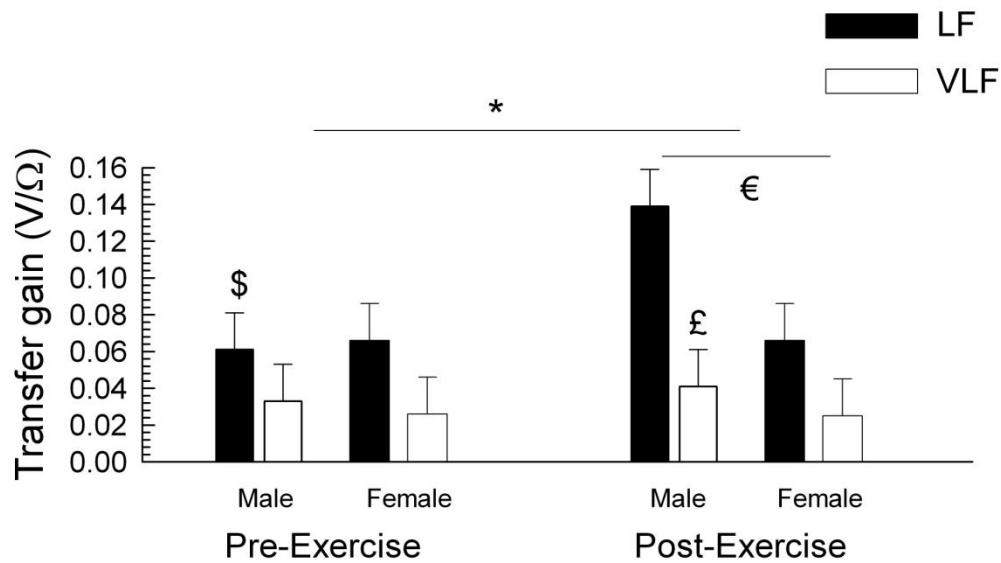


Figure 3-1: Transfer gain for EMG-Zo when the two signals were phase locked and EMG lagged behind Zo. * significant difference between tests; € significant difference between frequency bands post-exercise; \$ significant difference between tests in the LF band in men; £ significant difference between frequency bands post-exercise in men.



Figure 3-2: Mean phase angle of EMG-Zo when the two signals were phase locked and EMG lagged behind Zo. * significant difference between tests; € significant difference between frequency bands in each test; £ significant difference between frequency bands in the same test and gender.

3.3.2.2 EMG-CO

For the EMG-CO transfer function analysis during phase lock (lead and lag) when coherence was above threshold, mean EMG increased from pre- to post-exercise ($p < 0.001$); mean CO also increased ($p < 0.05$) post-exercise, and was significantly higher in men in both frequency bands ($p < 0.05$; Table 3-5). There was a decline ($p < 0.05$) in transfer gain ($\Delta CO / \Delta EMG$) after exercise for EMG-CO during phase lock (lead) with no effect of frequency band or gender (Table 3-7).

There was no main effect of exercise, frequency band, or gender on average phase difference between EMG and CO. The magnitude of the average phase angle during phase lock (lead) for EMG-CO was higher in the LF band than in the VLF band ($p < 0.001$), specifically in both genders ($p < 0.05$) and pre-

and post-exercise ($p < 0.05$). The magnitude of the average phase angle during phase lock (lag) for EMG-CO was higher ($p < 0.05$) in the LF band in than the VLF band, specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-8).

Average coherence for EMG-CO was lower in the LF band in than the VLF band ($p < 0.001$) in both genders ($p < 0.05$), but increased post-exercise in women ($p < 0.05$) and in the LF band ($p < 0.05$; Table 3-9). The percent time above significant coherence for EMG-CO was higher in the LF band than in the VLF band ($p < 0.05$) with no change post-exercise (Table 3-10). The percent time EMG and CO were in phase lock above significant coherence was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-11).

3.3.2.3 EMG-SBP

For the EMG-SBP during phase lock (lead and lag) transfer function when coherence was above threshold, mean EMG increased from pre-to post-exercise ($p < 0.001$) while mean SBP declined post-exercise in women ($p < 0.05$) and in each frequency band ($p < 0.05$; Table 3-5). EMG-SBP during phase lock (lead and lag) transfer gain ($\Delta\text{SBP}/\Delta\text{EMG}$) was higher ($p < 0.05$) in the LF band, specifically post-exercise in phase lead ($p < 0.05$; Table 3-7).

The average phase difference was significantly lower pre-exercise in the LF band in than the VLF band ($p < 0.05$); there was no main effect of exercise or gender on the average phase difference between EMG and SBP. The magnitude of the average phase angle under phase lock (lead) for EMG-SBP was higher in

the LF band ($p < 0.001$), specifically in women ($p < 0.05$) and post-exercise ($p < 0.05$). The magnitude of the average phase angle under phase lock (lag) for EMG-SBP was higher in the LF band than in the VLF ($p < 0.05$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-8). A plot of the coherence time series over the test duration overlaid with periods of phase lock for an individual participant demonstrated the alternating behaviour between phase lead and lag and the shift in behaviour from pre- to post-exercise (Figure 3-3).

Average coherence for EMG-SBP was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$), and increased after exercise in women ($p < 0.05$) and in the LF band ($p < 0.05$; Table 3-9). The percent time above significant coherence for EMG-SBP was higher in the LF band than in the VLF band both pre- and post-exercise ($p < 0.05$; Table 3-10). The percent time EMG and SBP were in phase lock above significant coherence was greater in the LF band than in the VLF band in men ($p < 0.05$) and post-exercise ($p < 0.05$; Table 3-11).

3.3.2.4 EMG-DBP

For the EMG-DBP transfer function during phase lock (lead and lag) when coherence was above threshold, mean EMG increased from pre- to post-exercise ($p < 0.001$) with no change in mean DBP; there was no difference in transfer function means between frequency bands or genders (Table 3-5). There was no change in transfer gain ($\Delta\text{DBP}/\Delta\text{EMG}$) for DBP-EMG post-exercise. Transfer gain for EMG-DBP during phase lock (lead and lag) was higher in the

LF band than in the VLF band ($p < 0.001$) with no effect of exercise or gender (Table 3-7).

There was no main effect of exercise, frequency band, or gender on the average phase difference between EMG and DBP. The magnitude of the average phase angle under phase lock (lead) for EMG-DBP was higher in the LF band than in the VLF band ($p < 0.001$) in both genders ($p < 0.05$) and pre-exercise ($p < 0.05$). The magnitude of the average phase angle under phase lock (lag) for EMG-DBP was higher in the LF band than in the VLF ($p < 0.05$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$), and decreased after exercise ($p < 0.01$; Table 3-8).

Average coherence for EMG-DBP was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$), and increased after exercise in men ($p < 0.05$; Table 3-9). There was no effect of exercise, frequency band, or gender on the percent time above significant coherence for EMG-DBP (Table 3-10). There was no effect of exercise, frequency band, or gender on the percent time EMG and DBP were in phase lock above significant coherence (Table 3-11).

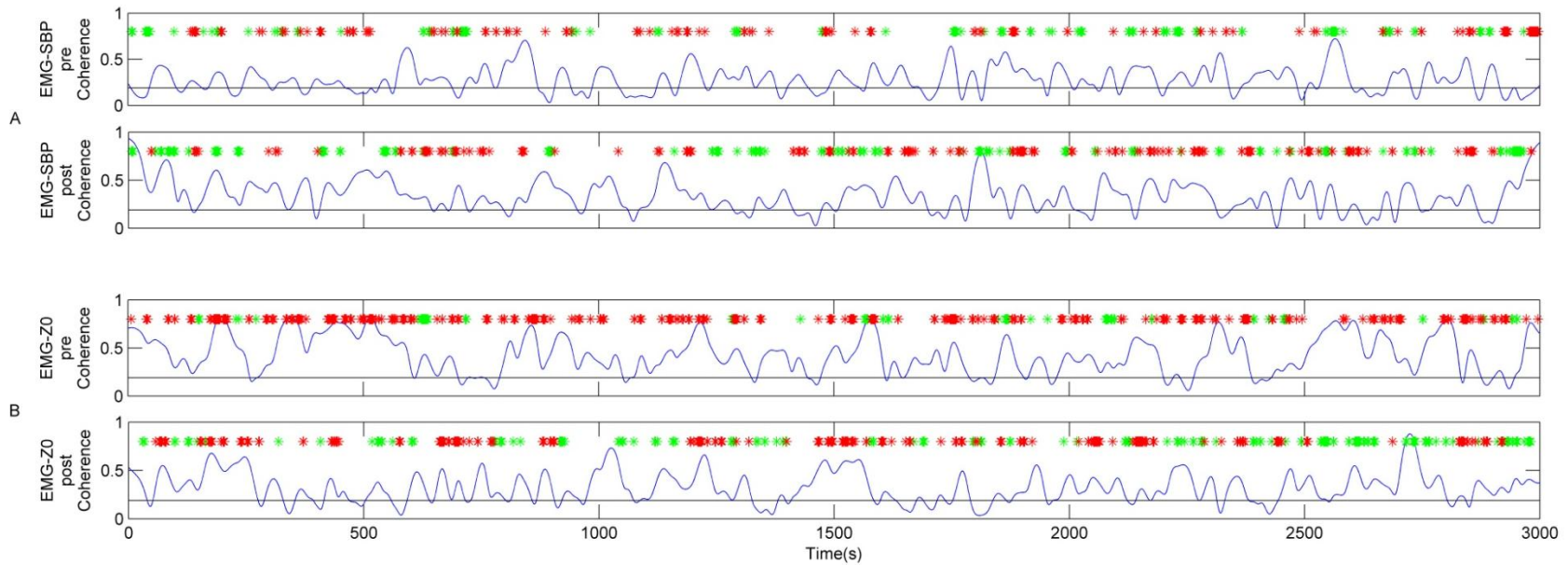


Figure 3-3: Pre- and post-exercise coherence time series for EMG-SBP (A) and EMG-Zo (B) interactions over the 5 minutes under analysis with corresponding periods of phase lock between the signal pairs in the LF band (0.1-0.05Hz) for a single participant (#15). Phase lock is depicted on each coherence plot as (*): the red (*) represents phase lead ((A) EMG is leading SBP; (B) EMG is leading Zo) and the green (*) represents phase lag ((A) EMG is lagging behind SBP; (B) EMG is lagging behind Zo). These plots demonstrate the alternating behaviour between phase lead and lag with a distinct change in this behaviour from pre- to post-exercise for both EMG-SBP and EMG-Zo. The straight line represents the significance threshold (0.1894).

Table 3-5: Skeletal muscle pump cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: mean value of the transfer function pairs during phase lock.

		F	M	Pre	Post	LF	VLF
lock	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	SBP*	110 (3) Y	113(3)	114(2)	109(2)	111(2) [^]	111(2)
lead	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	SBP*	109 (3) Y	113(3)	113(2)	109(2)	111(2) [^]	111(2)
lag	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	SBP*	110 (3) Y	114(2)	114 (2)	109 (2)	111(2) [^]	112(2)
lock	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	DBP	69(2)	71(2)	70(1)	69(1)	70(1)	70(1)
lead	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	DBP	69(2)	70(2)	70(1)	69(1)	70(1)	70(1)
lag	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	DBP	69(2)	70(2)	70(1)	69(1)	70(1)	70(1)
lock	EMG*	0.003 (0.0002) Y	0.003 (0.0002)	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	CO*†	4.87 (0.22)	6.27 (0.22) Y	5.41 (0.16) ψ	5.74 (0.16) ψ	5.58 (0.16)!	5.57 (0.16)! [^]
lead	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	CO*†	4.85 (0.23)	6.25 (0.23)	5.39 (0.17) ψ	5.71 (0.17) ψ	5.57 (0.17)!	5.53 (0.17)! [^]
lag	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	CO* †	4.89 (0.22)	6.30 (0.22)Y	5.43 (0.16)ψ	5.76 (0.16) ψ	5.60 (0.16)!	5.60 (0.16)! [^]

Reported as least square mean (SEM)
Units for reported variables: EMG (V); SBP (mmHg); DBP (mmHg); CO (L/min); Zo (Ω)
† Significantly different between genders
* Significantly different between tests
Y Significantly different between tests within same gender
[^] Significantly different between tests within same frequency band
ψ Significantly different between genders within same test
! Significantly different between genders within same frequency band

Table 3-6: Skeletal muscle pump cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: mean value of the transfer function pairs during phase lock.

		F	M	Pre	Post	LF	VLF
lock	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	Zo	-0.0011 (0.0002)	-0.0010 (0.0002) Y	-0.0013 (0.0002)	-0.0008 (0.0002)	-0.0010 (0.0002)	-0.0010 (0.0002)
lead	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	Zo	-0.0013 (0.0005)	-0.00015 (0.0005)	-0.001 (0.0004)	-0.0004 (0.0004)	-0.0010 (0.0004)	-0.0005 (0.0004)
lag	EMG*	0.003 (0.0002) Y	0.003 (0.0002) Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	Zo	-0.0010 (0.0004)	-0.0015 (0.0004)	-0.002 (0.0004)	-0.0009 (0.0004)	-0.0011 (0.0004)	-0.0013 (0.0004)
<p>Reported as least square mean (SEM) Units for reported variables: EMG (V); SBP (mmHg); DBP (mmHg); CO (L/min); Zo (Ω) * Significantly different between tests Y Significantly different between tests within same gender ^ Significantly different between tests within same frequency band ψ Significantly different between genders within same test</p>							

Table 3-7: Skeletal muscle pump cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: transfer function gain between signal pairs during phase lock

		F	M	Pre	Post	LF	VLF
EMG-SBP	Lock †	25.81 (3.74)	24.17 (3.74)	28.72 (3.43)	21.27 (3.43)	32.28 (3.43)	17.70 (3.43) [^]
	Lead †	25.79 (3.92)	23.51 (3.92)	27.95 (3.76)	21.34 (3.76)€	33.27 (3.76)	16.02 (3.76)
	Lag	29.02 (3.78)	27.5 (3.78)	32.59 (3.72)	23.93 (3.72)	32.59 (3.72)	23.93 (3.72)
EMG-DBP	Lock †	18.78 (2.35)#	15.16 (2.27)#	18.71 (2.16)	15.23 (2.24)€	23.83 (2.20)	10.11 (2.20)
	Lead †	17.64 (2.96)#	14.59 (2.87)	19.22 (2.61)	13.00 (2.71)€	22.93 (2.66)	9.29 (2.66)
	Lag †	20.65 (2.50)	18.57 (2.39)	19.52 (2.52)	19.69 (2.61)€	26.47 (2.56)	12.74 (2.56)
EMG-CO	Lock	2.16 (0.48)	1.54 (0.48)	2.14 (0.42)	1.57 (0.42)	1.92 (0.42)	1.79 (0.42)
	Lead*	2.58 (0.49)	1.39 (0.48)	2.57 (0.45)€	1.39 (0.44)€	2.36 (0.45)	1.60 (0.44)
	Lag	2.41 (0.58)	2.05 (0.58)	2.44 (0.50)	2.02 (0.50)	2.35 (0.50)	2.10 (0.50)
EMG-Zo	Lock †	0.046 (0.014)	0.065 (0.014)#	0.049 (0.012)	0.062 (0.012)€	0.081 (0.012)	0.030 (0.012)
	Lead †	0.049 (0.014)#	0.063 (0.014)#	0.055 (0.012)	0.056 (0.012)	0.083 (0.012)	0.028 (0.012)
	Lag †	0.046 (0.02)	0.069 (0.02)#	0.047 (0.01)	0.068 (0.01)€	0.083 (0.01)	0.031 (0.01)
Reported as least square mean (SEM) Units for reported variables: EMG-SBP(mmHg/V); EMG-DBP(mmHg/V); EMG-CO(L min ⁻¹ /V); EMG-Zo(Ω/V) * Significantly different between tests † Significantly different between frequency bands Y Significantly different between tests within same gender € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender ^ Significantly different between tests within same frequency band							

Table 3-8: Skeletal muscle pump cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: phase difference and average phase angle

		F	M	Pre	Post	LF	VLF
EMG-SBP	Diff	-1.20 (1.93)	0.73 (1.93)	-0.70 (1.78)€	0.23 (1.78)	-0.79 (1.78)	0.31 (1.78)
	Lead †	38.29 (1.59)#	40.77 (1.59)	38.54 (1.51)	40.52 (1.51)€	44.16 (1.51)	34.90 (1.51)
	Lag †	-38.60 (1.56)#	-38.35 (1.56)#	-37.82 (1.38)€	-39.13 (1.38)€	-44.25 (1.38)	-32.69 (1.38)
EMG-DBP	Diff	-3.28 (2.38)	1.52 (2.32)	-1.50 (2.03)	-0.26 (2.10)	-1.19 (2.06)	-0.57 (2.06)
	Lead †	38.16 (1.62)#	41.05 (1.67)#	37.25 (1.56)€	41.96 (1.62)	45.00 (1.59)	34.21 (1.59)
	Lag †	-41.35 (1.80)#	-37.44 (1.75)#	-38.02 (1.57)€	-40.77 (1.63)€	-44.63 (1.60)	-34.16 (1.60)
EMG-CO	Diff	-6.38 (2.22)	-4.81 (2.22)	-4.74 (2.10)	-6.45 (2.10)	-6.47 (2.10)	-4.71 (2.10)
	Lead †	36.57 (1.72)#	37.02 (1.72)#	36.56 (1.62)€	37.02 (1.62)€	42.50 (1.62)	31.08 (1.62)
	Lag †	-40.76 (2.14)#	-40.47 (2.14)#	-36.41 (2.07)€	-44.81 (2.07)€	-46.52 (2.07)^	-34.70 (2.07)
EMG-Zo	Diff	-6.96 (2.46)	-3.86 (2.46)	-8.01 (2.30)	-2.81 (2.30)	-4.83 (2.30)	-5.99 (2.30)
	Lead †	38.71 (1.44)#	42.43 (1.44)#	41.03 (1.37)€	40.10 (1.37)€	51.30 (1.37)	29.84 (1.38)
	Lag †	-44.55 (1.91)#	-44.63 (1.91)#	-46.59 (1.65)€	-42.58 (1.65)€	-54.10 (1.65)	-35.07 (1.65)
Reported as least square mean (SEM) Units for reported variables: degrees † Significantly different between genders * Significantly different between tests ‡ Significantly different between frequency bands Y Significantly different between tests within same gender ^ Significantly different between tests within same frequency band € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender ψ Significantly different between genders within same test							

Table 3-9: Skeletal muscle pump cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: average value of coherence when the signal pairs were above the threshold of significance

	F	M	Pre	Post	LF	VLF
EMG-SBP*†	0.3789 (0.004)#Y	0.3758 (0.004)#	0.3684 (0.003)€	0.3862 (0.003)€	0.3478 (0.003)^	0.4069 (0.003)
EMG-DBP*†	0.379 (0.003)#	0.381 (0.003)#Y	0.377 (0.003)€	0.384 (0.003)€	0.353 (0.003)	0.408 (0.003)
EMG-CO*†	0.3684 (0.004)#Y	0.3647 (0.004)#	0.3582 (0.004)	0.3748 (0.004)	0.3270 (0.004)^	0.4061 (0.004)
EMG-Zo*†	0.4173 (0.008)#Y	0.4150 (0.008)#	0.4303 (0.007)	0.4020 (0.007)	0.3931 (0.007)^	0.4392 (0.007)
Reported as least square mean (SEM) Units for reported variables: au * Significantly different between tests † Significantly different between frequency bands Y Significantly different between tests within same gender ^ Significantly different between tests within same frequency band € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender						

Table 3-10: Skeletal muscle pump cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: percent time above the threshold of significant coherence

	F	M	Pre	Post	LF	VLF
EMG-SBP†	58.59 (1.49)	56.12 (1.49)	57.97 (1.35)€	56.75 (1.35)€	69.94 (1.35)	44.78 (1.35)
EMG-DBP	57.01 (1.42)	57.28 (1.37)	57.73 (1.29)	56.56 (1.34)	70.75 (1.31)	43.54 (1.31)
EMG-CO†	51.16 (1.74)	51.17 (1.74)	50.55 (1.57)	51.79 (1.57)	60.33 (1.57)	42.01 (1.57)
EMG-Zo*	71.53 (2.29)Y	67.06 (2.29)	74.63 (1.97)	63.96 (1.97)€	80.99 (1.97)	57.60 (1.97)^
Reported as least square mean (SEM) * Significantly different between tests † Significantly different between frequency bands Y Significantly different between tests within same gender ^ Significantly different between tests within same frequency band € Significantly different between frequency bands within same test						

Table 3-11: Skeletal muscle pump cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: percent time signal pairs were phase locked above significant coherence

	F	M	Pre	Post	LF	VLF
EMG-SBP†	8.10 (0.31)	7.24 (0.31)#	7.38 (0.42)	7.96 (0.42)€	8.41 (0.42)	6.50 (0.42)
EMG-DBP	7.72 (0.036)	6.94 (0.35)	6.86 (0.42)	7.79 (0.44)	7.11 (0.43)	7.55 (0.43)
EMG-Co†	6.24 (0.55)#	5.50 (0.55)#	5.34 (0.51)€	6.40 (0.51)€	1.84 (0.51)	9.90 (0.51)
EMG-Zo*†	18.07 (0.84)#Y	17.53 (0.84)	19.91 (0.79)	15.68 (0.79)	16.33 (0.79)	19.27 (0.79)^
Reported as least square mean (SEM) * Significantly different between tests † Significantly different between frequency bands Y Significantly different between tests within same gender ^ Significantly different between tests within same frequency band € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender						

3.3.3 Vascular Responses

3.3.3.1 Zo-CO

For the Zo-CO transfer function during phase lock (lead and lag) when coherence was above threshold, there was no change in mean Zo from pre- to post-exercise; mean CO was higher in men than in women ($p < 0.05$) across test conditions and both frequency bands, and increased post-exercise ($p < 0.001$; Table 3-12). Zo-CO transfer gain ($\Delta CO / \Delta Zo$) during phase lock (lead and lag) was lower in the LF band than in the VLF band ($p < 0.05$), specifically in women ($p < 0.05$) and pre-exercise ($p < 0.05$); transfer gain during phase lock (lag) declined in the VLF band post-exercise ($p < 0.05$; Figure 3-4; Table 3-13).

There was no main effect of exercise, frequency band, or gender on the average phase difference between Zo and CO. The magnitude of the average

phase angle during phase lock (lead) for Zo-CO was higher in the LF band than in the VLF band ($p < 0.05$). The magnitude of the average phase angle during phase lock (lag) for Zo-CO (Figure 3-5) was higher in the LF band in men ($p < 0.05$) and post-exercise ($p < 0.05$; Table 3-14). There was no main effect of exercise or gender on average phase angle.

Average coherence for Zo-CO was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-15). There was no difference between frequency bands on the percent time above significant coherence for Zo-CO (Table 3-16). The percent time Zo and CO were in phase lock above significant coherence was lower in the LF band than in the VLF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-17).

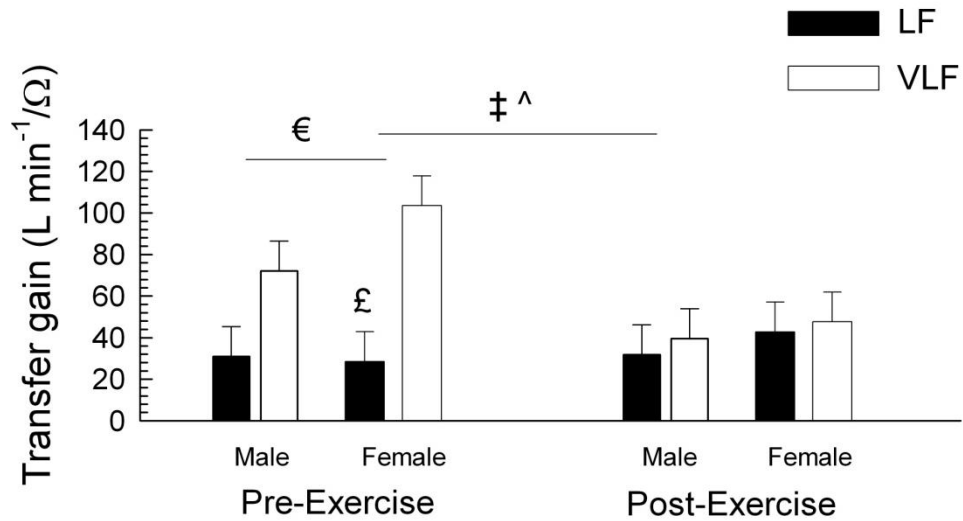


Figure 3-4: Transfer gain for Zo-CO when the two signals were phase locked and Zo lagged behind CO. ‡ significant difference between frequency bands; ^ significant difference between tests in the VLF band; € significant difference between frequency bands pre-exercise; £ significant difference between frequency bands pre-exercise in women.

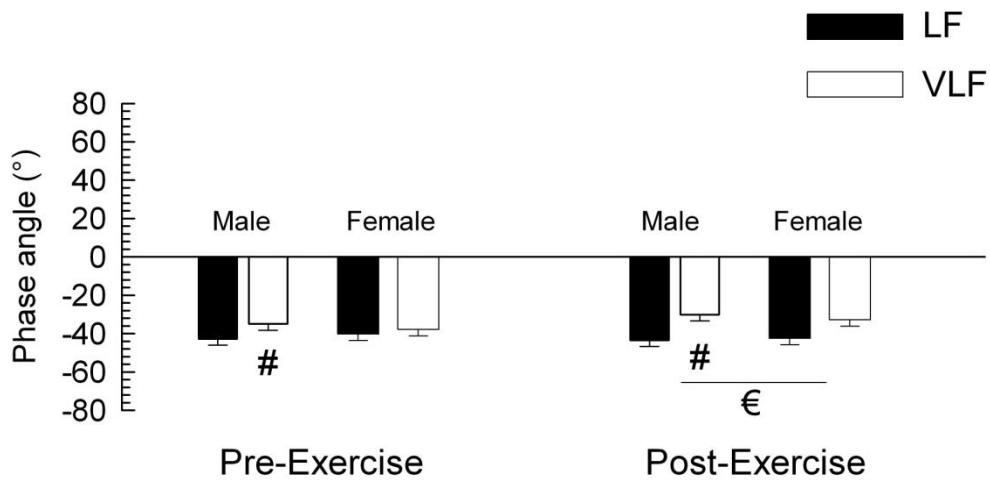


Figure 3-5: Average phase angle when Zo and CO were phase locked and Zo lagged behind CO. # significant difference between frequency bands in men; € significant difference between frequency bands in the post-exercise test.

3.3.3.2 Zo-SBP

For the Zo-SBP transfer function during phase lock (lead and lag) when coherence was above threshold, there was no change in mean Zo from pre- to post-exercise while mean SBP declined post-exercise ($p < 0.001$), specifically in women ($p < 0.05$) and in each frequency band ($p < 0.05$; Table 3-12). Zo-SBP transfer gain ($\Delta\text{SBP}/\Delta\text{Zo}$) during phase lock (lag) was lower in the LF band than in the VLF band pre-exercise ($p < 0.05$); gain during phase lock (lead and lag) significantly declined post-exercise ($p < 0.05$), specifically in the VLF band ($p < 0.05$; Figure 3-6; Table 3-13).

The average phase difference was lower in the LF band than in the VLF band ($p < 0.05$); there was no effect of exercise or gender on the average phase difference. The average phase angle under phase lock (lead) for Zo-SBP was higher in the LF band ($p < 0.001$), specifically in women ($p < 0.05$) and post-exercise ($p < 0.05$). The magnitude of the average phase angle under phase lock (lag) for Zo-SBP was higher in the LF band than in the VLF ($p < 0.05$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Figure 3-7; Table 3-14). A plot of the coherence time series over the test duration overlaid with periods of phase lock for an individual participant demonstrated the alternating behaviour between phase lead and lag and the shift in behaviour from pre- to post-exercise in the LF band (Figure 3-8).

Average coherence for Zo-SBP was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-15). The percent time above significant coherence for Zo-SBP was higher in women in the post-

exercise test compared to men ($p < 0.05$; Table 3-16). The percent time Zo and SBP were in phase lock above significant coherence was higher in the LF band than in the VLF band ($p < 0.05$), specifically in women ($p < 0.05$) and post-exercise ($p < 0.05$; Table 3-17).

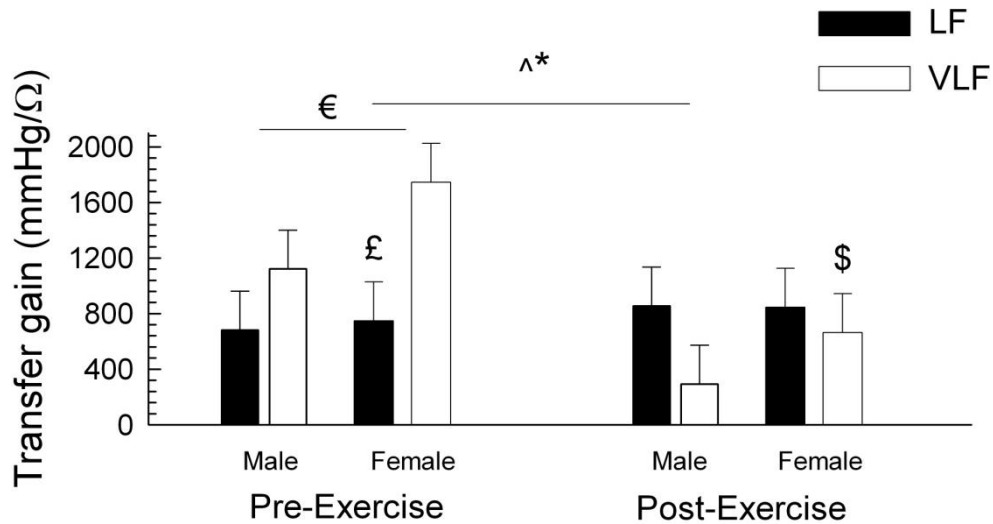


Figure 3-6: Transfer gain for Zo-SBP when the two signals were phase locked and Zo lagged behind SBP. * significant difference between tests; ^ significant difference between tests in the VLF band; € significant difference between frequency bands pre-exercise; £ significant difference between frequency bands pre-exercise in women; \$ significant difference between tests in the VLF band in women.

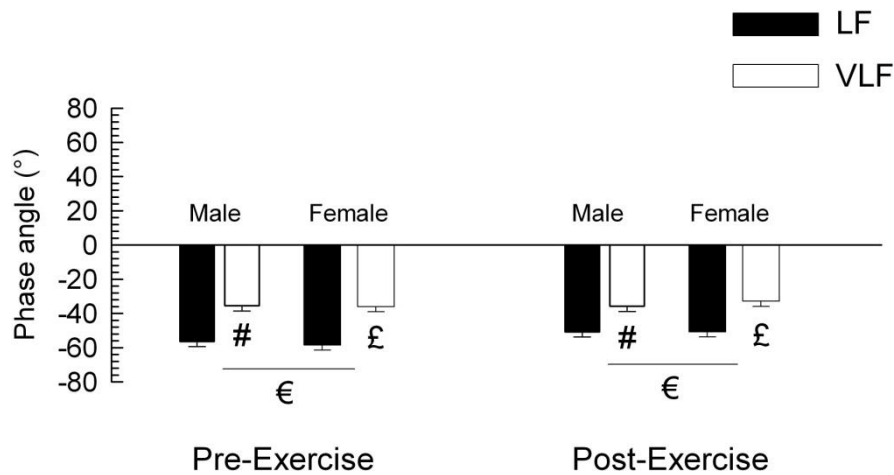


Figure 3-7: Average phase angle for Zo-SBP when the two signals were phase locked and Zo lagged behind SBP. # significant difference between frequency bands in men; € significant difference between frequency bands in each test; £ significant difference between frequency bands in the same test in women.

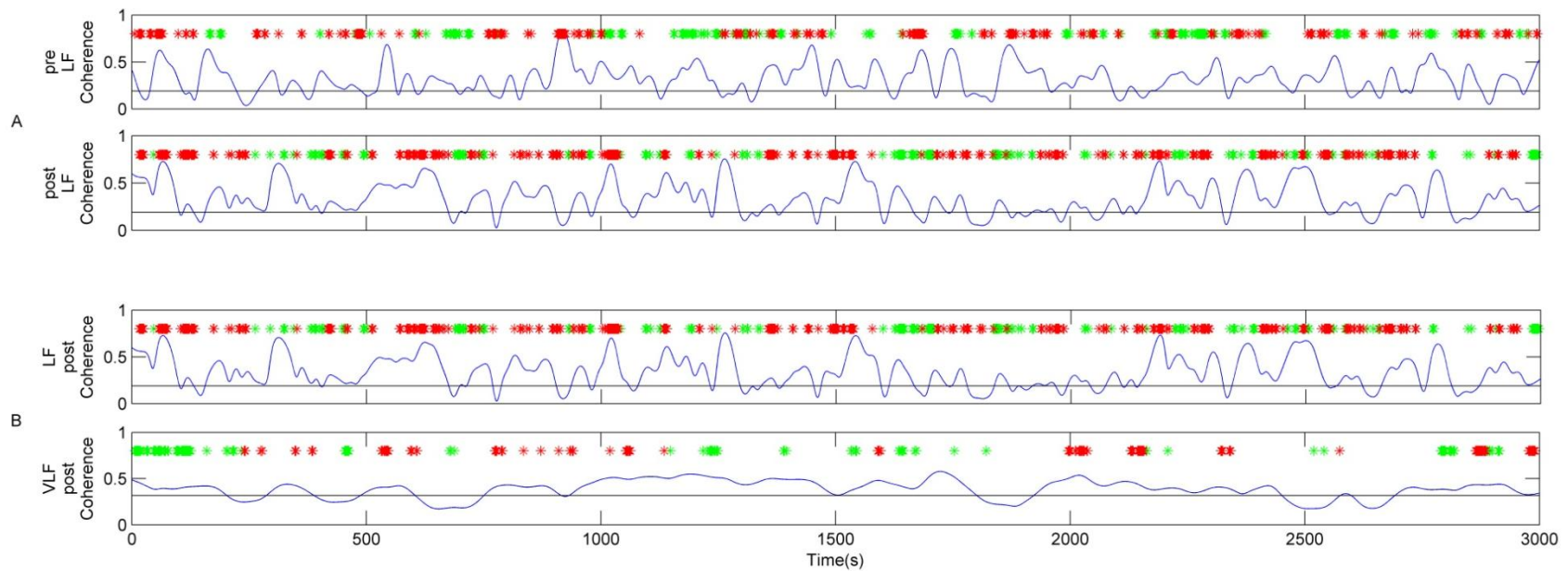


Figure 3-8: Coherence time series for Zo-SBP over the 5 minutes under analysis with corresponding periods of phase lock between the signal pairs for a single participant (#15). (A) demonstrates the differences in Zo-SBP between the pre- and post-exercise tests in the LF band (0.05-0.1Hz). (B) demonstrates differences in Zo-SBP between the LF (0.05-0.1Hz) and VLF (0.01-0.05Hz) bands in the post-exercise test. The straight line represents the corresponding significance thresholds (LF: 0.1894; VLF: 0.3162). Phase lock is depicted on each coherence plot as (*): the red (*) represents phase lead (EMG is leading Zo) and the green (*) represents phase lag (EMG is lagging behind Zo). The alternating behaviour between phase lead and lag is clearly shown with a distinct change in this behaviour from pre- to post-exercise and between frequency bands after exercise.

Table 3-12: Vascular cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: mean value of the transfer function pairs during phase lock

		F	M	Pre	Post	LF	VLf
lock	Zo	-0.0007 (0.006)	-0.0002 (0.0006)	-0.0004 (0.0005)	-0.0005 (0.0005)	-0.0008 (0.0005)	-0.00001 (0.0005)
	SBP*	109(3)Y	113(3)	114(2)	109(2)	111(2)^	111(2)^
lead	Zo	-0.0005 (0.0006)	-0.0003 (0.0006)	-0.0003 (0.0006)	0.00001 (0.0006)	-0.0007 (0.0006)	0.0004 (0.0006)
	SBP*	110(3) Y	113(3)	114(2)	109(2)	111(2)^	111(2)^
lag	Zo	-0.0011 (0.0008)	-0.0009 (0.0008)	-0.0006 (0.0007)	-0.001 (0.0007)	-0.0007 (0.0007)	-0.0012 (0.0007)
	SBP*	109(3) Y	113(3)	113(2)	109(2)	111(2)^	111(2)^
lock	Zo	-0.0008 (0.0005)	-0.0009 (0.0005)	-0.001 (0.0004)	-0.0006 (0.0004)	-0.0009 (0.0004)	-0.0008 (0.0004)
	CO*†	4.87 (0.22)	6.28 (0.22)	5.41 (0.16)ψ	5.73 (0.16)ψ	5.57 (0.16)!	5.58 (0.16)!
lead	Zo	-0.0006 (0.0006)	-0.0010 (0.0006)	-0.0003 (0.0005)	-0.001 (0.0005)	-0.0006 (0.0005)	-0.0010 (0.0005)
	CO*†	4.85 (0.23)	6.29 (0.23)Y	5.41 (0.17)ψ	5.74 (0.17)ψ	5.58 (0.17)!	5.57 (0.17)!
lag	Zo	-0.0011 (0.0004)	-0.0010 (0.0004)	-0.0009 (0.0004)	-0.001 (0.0004)	-0.0011 (0.0004)	-0.0011 (0.0004)
	CO*†	4.87 (0.23)	6.27 (0.23)	5.42 (0.17)ψ	5.72 (0.17)ψ	5.57 (0.17)!	5.57 (0.17)!

Reported as least square mean (SEM)
Units for reported variables: SBP (mmHg); CO (L/min); Zo (Ω)
† Significantly different between genders
* Significantly different between tests
Y Significantly different between tests within same gender
^ Significantly different between tests within same frequency band
ψ Significantly different between genders within same test
! Significantly different between genders within same frequency band

Table 3-13: Vascular cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: transfer gain between the signal pairs during phase lock

		F	M	Pre	Post	LF	VLF
Zo-SBP	Lock*	843.93 (185.90)	621.76 (185.90)	643.42 (144.46)€	933.46 (144.46)	643.42 (144.46)	822.28 (144.46)^
	Lead*	822.66 (176.48)	639.176 (176.48)Y	918.47 (136.45)	543.42 (136.45)	642.53 (136.45)	819.36 (136.45)^
	Lag*	1002.7 (206.8)	641.6 (206.8)	1073.9 (165.5)€	570.5 (165.5)	688.7 (165.5)	955.6 (165.5)^
Zo-CO	Lock†	49.50 (7.63)#	42.41 (7.63)	52.64 (6.52)€	39.27 (6.52)	32.77 (6.52)	59.14 (6.52)^
	Lead†	53.61 (8.19)#	44.07 (8.12)#	52.15 (6.90)€	45.54 (6.82)€	31.94 (6.90)	65.75 (6.82)
	Lag†	55.62 (8.81)#	43.62 (8.91)	58.79 (7.75)€	40.45 (7.87)	33.53 (7.87)	65.71 (7.75)^
Reported as least square mean (SEM) Units for reported variables: Zo-SBP(mmHg/Ω); Zo-CO(L min ⁻¹ /Ω) * Significantly different between tests † Significantly different between frequency bands Y Significantly different between tests within same gender € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender ^ Significantly different between tests within same frequency band							

Table 3-14: Vascular cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: phase difference and average phase angle during phase lock

		F	M	Pre	Post	LF	VLF
Zo-SBP	Diff†	0.66 (3.09)	1.60 (3.09)	-0.16 (2.60)	2.42 (2.60)	-1.82 (2.60)	4.08 (2.60)
	Lead†	40.67 (0.93)#	39.93 (0.93)	40.79 (1.18)	39.81 (1.18)€	44.04 (1.18)	36.56 (1.18)
	Lag†	-41.11 (1.63)#	-39.44 (1.63)#	-40.62 (1.50)€	-39.93 (1.50)€	-46.24 (1.50)	-34.30 (1.50)
Zo-CO	Diff	-4.54 (2.59)	-4.60 (2.59)	-6.08 (2.57)	-3.06 (2.57)	-5.22 (2.57)	-3.93 (2.57)
	Lead†	37.16 (1.53)	32.95 (1.53)	33.10 (1.56)	37.02 (1.56)	37.49 (1.56)	32.62 (1.56)
	Lag†	-38.42 (1.71)#	-37.80 (1.71)#	-38.97 (1.67)€	-42.26 (1.67)€	-42.26 (1.67)	-33.96 (1.67)
Reported as least square mean (SEM) Units for reported variables: phase difference (au), phase angle (°) † Significantly different between frequency bands € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender							

Table 3-15: Vascular cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: average coherence when the signal pairs were above the threshold of significance

	F	M	Pre	Post	LF	VLF
Zo-SBP †	0.3874 (0.004)#	0.3834 (0.004)#	0.3850 (0.004)€	0.3858 (0.004)€	0.3499 (0.004)	0.4209 (0.004)
Zo-CO †	0.3895 (0.006)#	0.3780 (0.006)#	0.3798 (0.005)€	0.3877 (0.005)€	0.3425 (0.005)	0.4249 (0.005)
Reported as least square mean (SEM) Units for reported variables: au † Significantly different between frequency bands # Significantly different between frequency bands within same gender € Significantly different between frequency bands within same test						

Table 3-16: Vascular cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: percent time above the threshold of significant coherence

	F	M	Pre	Post	LF	VLF
Zo-SBP	65.06 (1.64)	58.81 (1.64)	60.91 (1.54)	62.97 (1.54) ψ	71.92 (1.54)	51.95 (1.54)
Zo-CO	62.57 (2.71)	56.04 (2.71)	58.63 (2.31)	59.99 (2.31)	63.85 (2.31)	54.77 (2.31)
Reported as least square mean (SEM)						
ψ Significantly different between genders within same test						

Table 3-17: Vascular cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: percent time the signal pairs were phase locked and above significant coherence

	F	M	Pre	Post	LF	VLF
Zo-SBP \ddagger	10.56 (0.90) $\#$	8.84 (0.90)	9.06 (0.78)	10.34 (0.78) €	11.34 (0.78)	8.05 (0.78)
Zo-Co \ddagger	9.34 (0.89) $\#$	6.73 (0.89) $\#$	7.83 (0.76) €	8.24 (0.76) €	3.04 (0.76)	13.03 (0.76)
Reported as least square mean (SEM)						
\ddagger Significantly different between frequency bands						
$\#$ Significantly different between frequency bands within same gender						
€ Significantly different between frequency bands within same test						

3.3.4 Centre of Pressure

3.3.4.1 EMG-COPx

For the EMG-COPx transfer function during phase lock (lead and lag) when coherence was above threshold, mean EMG increased from pre-to post-exercise ($p < 0.001$) with no change in mean COPx (Table 3-18; Table 3-19). There was no effect of exercise, frequency band, or gender on EMG-COPx transfer gain ($\Delta\text{COPx}/\Delta\text{EMG}$) (Table 3-20).

The average phase difference between EMG and COPx was lower in the LF band than in the VLF band ($p < 0.05$); there was no effect of exercise or gender on average phase difference. The magnitude of the average phase angle during phase lock (lead) for EMG-COPx was higher in the LF band than in the VLF band ($p < 0.001$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$). The magnitude of the average phase angle during phase lock (lag) for EMG-COPx was higher in the LF band than in the VLF ($p < 0.05$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$); and was lower in women than in men ($p < 0.05$; Table 3-21).

Average coherence for EMG-COPx was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-22). There was no difference between frequency bands on the percent time above significant coherence for EMG-COPx (Table 3-23). The percent time EMG and COPx were in phase lock above significant coherence was lower in the LF band than in the VLF band in women ($p < 0.05$) and pre-exercise ($p < 0.05$; Table 3-24).

3.3.4.2 EMG-COPy

For the EMG-COPy transfer function during phase lock (lead and lag) when coherence was above threshold, mean EMG increased ($p < 0.001$) while mean COPy decreased ($p < 0.05$) from pre- to post-exercise (Table 3-18). EMG-COPy transfer gain ($\Delta\text{COPy}/\Delta\text{EMG}$) during phase lock (lead) was higher in the post-exercise in the LF band than in the VLF band ($p < 0.05$); gain during phase lock (lead and lag) declined post-exercise ($p < 0.05$), specifically in women ($p < 0.05$) and in the VLF band ($p < 0.05$; Figure 3-9; Table 3-20).

The average phase difference between EMG and COPy was higher women than men ($p < 0.05$), higher in the LF band than in the VLF band ($p < 0.05$), and decreased after exercise ($p < 0.05$). The magnitude of the average phase angle under phase lock (lead and lag) for EMG-COPy was lower in men than women in the pre-exercise test ($p < 0.05$), and was lower in the LF band ($p < 0.05$) in phase lag only. The average phase angle during phase lock (lead) for EMG-COPy was higher in the LF band than in the VLF band ($p < 0.001$), and decreased after exercise ($p < 0.001$; Figure 3-10). The magnitude of the average phase angle during phase lock (lag) for EMG-COPy was higher in the LF band than in the VLF ($p < 0.05$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-21). A plot of the coherence time series over the test duration overlaid with periods of phase lock for an individual participant demonstrated the alternating behaviour between phase lead and lag and the shift in behaviour from pre- to post-exercise (Figure 3-13).

Average coherence for EMG-COPy was higher in women than men ($p < 0.05$), lower in the LF band than in VLF band in women ($p < 0.05$), and declined after exercise in women ($p < 0.05$) and in both frequency bands ($p < 0.05$; Table 3-22). The percent time above significant coherence for EMG-COPy was higher in women than in men ($p < 0.05$), and declined after exercise in both genders ($p < 0.05$) and frequency bands ($p < 0.05$; Table 3-23). The percent time EMG and COPy were in phase lock above significant coherence was lower in the LF band than in the VLF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$), and declined after exercise in the VLF band ($p < 0.05$; Table 3-24).

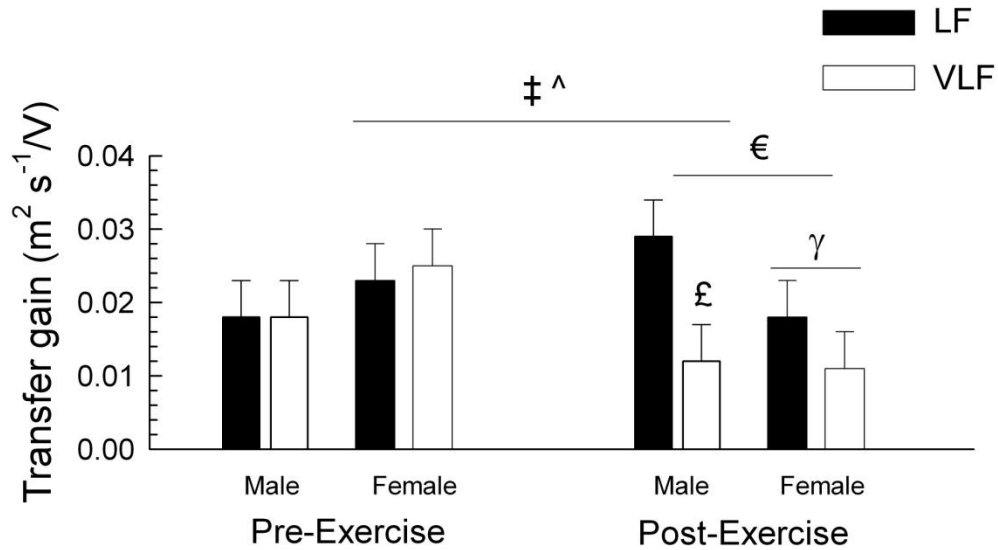


Figure 3-9: Transfer gain between EMG and COP trajectory in the AP direction when the signals were phase locked and EMG was leading COPy. ‡ significant difference between frequency bands; ^ significant difference between tests in the VLF band; € significant difference between frequency bands post-exercise; γ significant difference between tests in men; £ significant difference between frequency bands post-exercise in men.

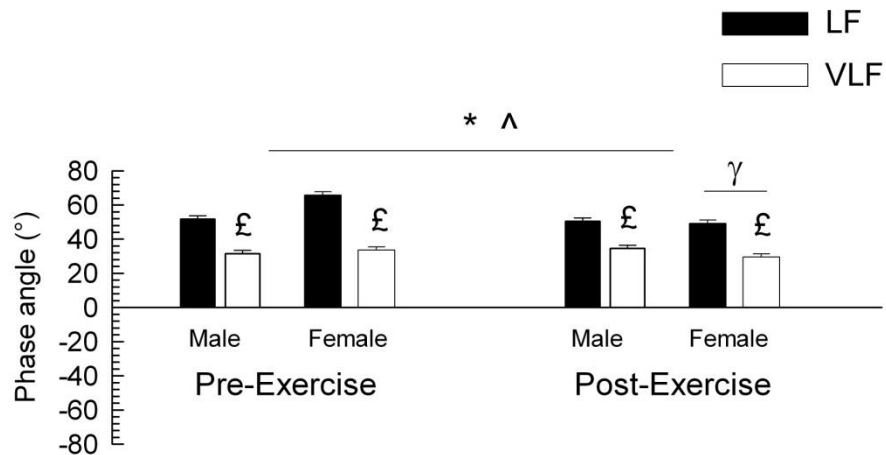


Figure 3-10: Average phase angle for EMG-COPy when the signal pair was phase locked and EMG was leading COPy. * significant difference between tests; ^ significant difference between tests in the LF band; § significant difference between tests in the same frequency band in women; γ significant difference between tests in women; £ significant difference between frequency bands in the same gender and test; § significant difference between genders pre-exercise in the LF band.

3.3.4.3 Zo-COPx

In the Zo-COPx transfer function during phase lock (lead and lag) when coherence was above threshold, there was no effect of exercise, frequency band, or gender on mean Zo or COPx (Table 3-18). Zo-COPx transfer gain ($\Delta Zo/\Delta COPx$) during phase (lag) was higher in the LF band in women ($p < 0.05$); gain during phase lock (lead) increased after exercise ($p < 0.05$; Table 3-20).

The average phase difference between Zo and COPx was lower in the LF band than in the VLF band ($p < 0.05$); there was no effect of exercise or gender on average phase difference. The average phase angle during phase lock (lead) for Zo-COPx was higher in the LF band ($p < 0.001$), specifically in women ($p < 0.05$) and post-exercise ($p < 0.05$). The magnitude of the average Zo-COPx phase angle during phase lock (lag) was higher in the LF band than in the VLF band ($p < 0.05$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-21). There was no main effect of exercise or gender on average phase angle during phase lock.

Average coherence for Zo-COPx was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-22). There was no difference between frequency bands on the percent time above significant coherence for Zo-COPx (Table 3-23). The percent time Zo and COPx were in phase lock above significant coherence was lower in the LF band than in the VLF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-24).

3.3.4.4 Zo-COPy

In the Zo-COPy transfer function during phase lock (lead and lag) when coherence was above threshold, there was no change in mean Zo while mean COPy declined significantly after exercise ($p < 0.05$; Table 3-19). Zo-COPy transfer gain ($\Delta Zo / \Delta EMG$) during phase lock (lead and lag) increased after exercise ($p < 0.05$), specifically in men ($p < 0.05$) in phase lock (lead); gain during phase lock (lag) was higher in the LF band than in VLF band ($p < 0.05$) (Figure 3-11; Table 3-20).

The average phase difference between Zo and COPy was lower in the LF band ($p < 0.05$) and increased after exercise ($p < 0.05$). There was no effect of exercise, frequency band, or gender on the average phase angle for Zo-COPy during phase lock (lead) (Figure 3-12). The magnitude of the average phase angle for Zo-COPy during phase lock (lag) was lower in the LF band than in the VLF ($p < 0.05$), specifically in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$); phase angle magnitude decreased post-exercise ($p < 0.05$), specifically in women ($p < 0.05$) and in the LF band ($p < 0.05$; Table 3-21). A plot of the coherence time series over the test duration overlaid with periods of phase lock for an individual participant demonstrated the alternating behaviour between phase lead and lag and the shift in behaviour from pre- to post-exercise (Figure 3-13).

Average coherence for Zo-COPy was lower in the LF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-22). The percent

time above significant coherence for Zo-COPy was higher in the LF band than in the VLF band in men ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-23). The percent time above significant coherence was higher in women than men in the post-exercise test ($p < 0.05$) and in the VLF band ($p < 0.05$). The percent time Zo and COPy were in phase lock above significant coherence was lower in the LF band than in the VLF band in both genders ($p < 0.05$) and pre- and post-exercise ($p < 0.05$; Table 3-24).

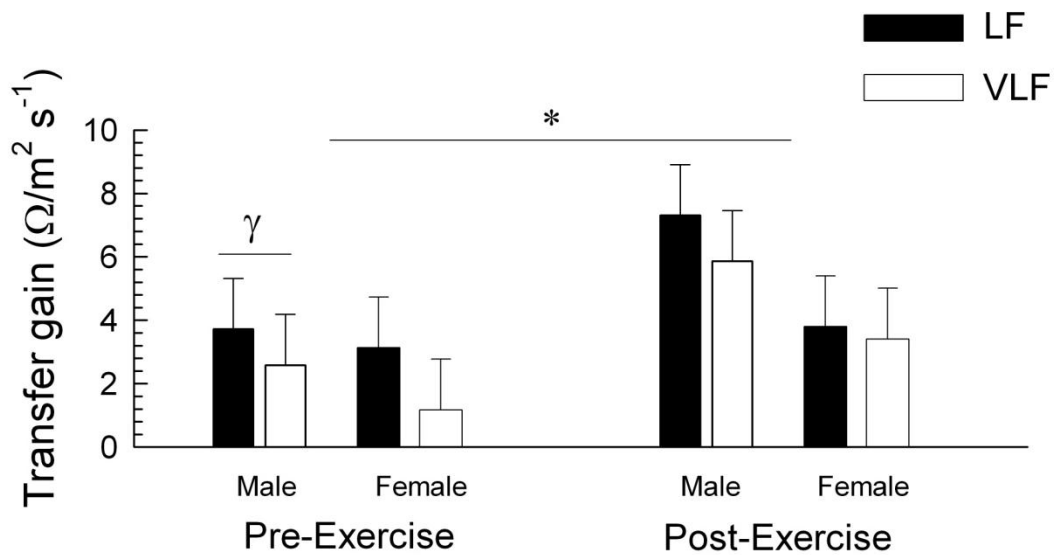


Figure 3-11: Transfer gain between Zo and COP trajectory in the AP direction when the signals were phase locked and Zo was leading COPy. * significant difference between tests; γ significant difference between tests in men.

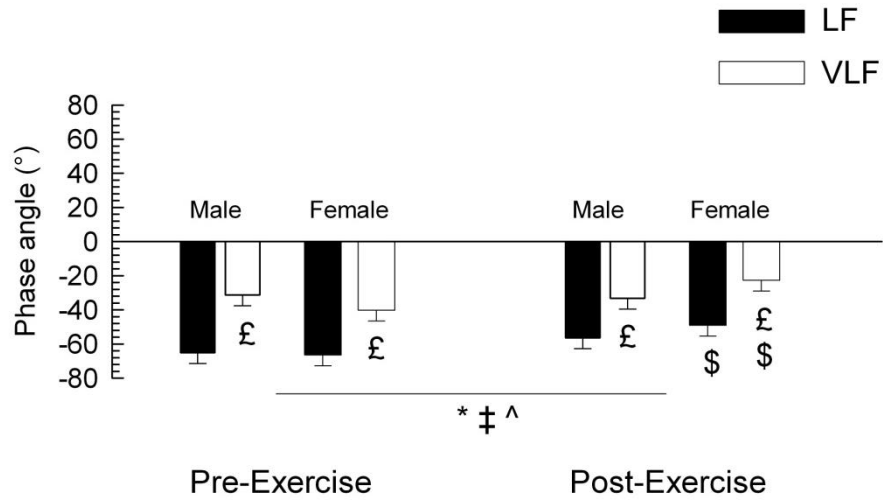


Figure 3-12: Average phase angle for Zo-COPy when the signal pair was phase locked and Zo was lagging behind COPy. * significant difference between tests; ^ significant difference between tests in the LF band; ‡ significant difference between tests in women; \$ significant difference between tests in women in each frequency band; £ significant difference between frequency bands in the same gender and test.

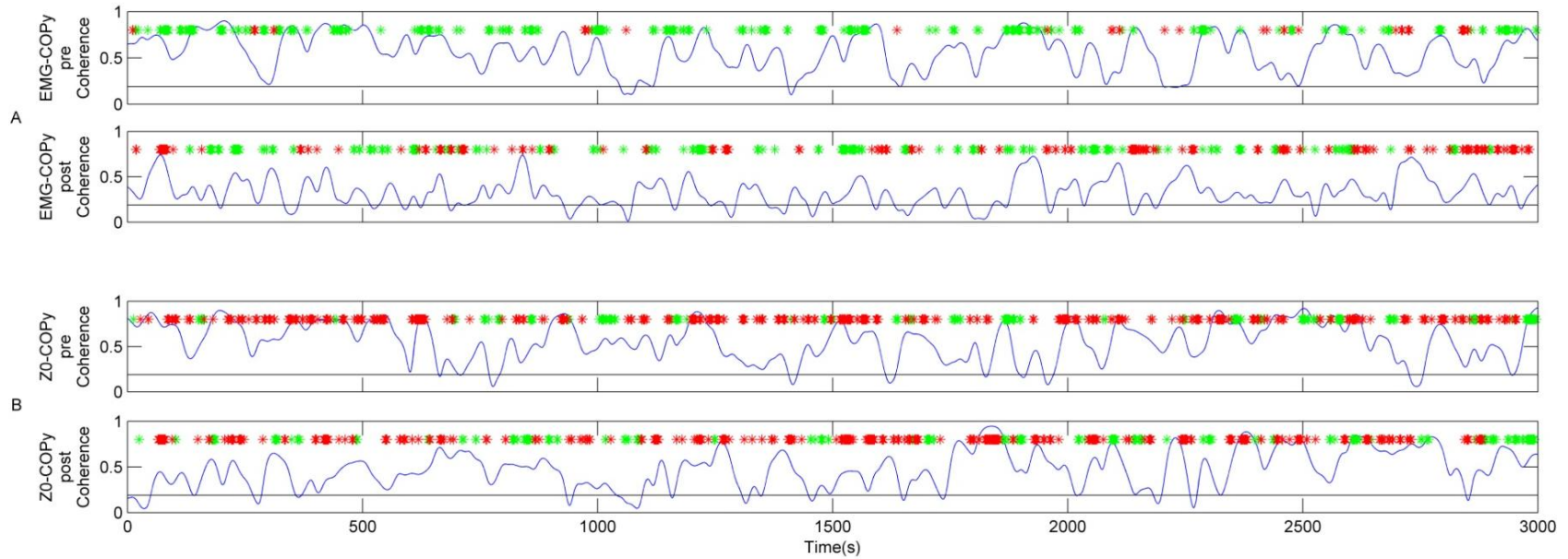


Figure 3-13: Coherence time series for EMG-COPy (A) and Zo-COPy (B) interactions over the 5 minutes under analysis with corresponding periods of phase lock between the signal pairs for a single participant (#15). The pre- and post-exercise tests are shown for each signal pair in the LF band (0.05-0.1Hz). The straight line represents the significance threshold (0.1894). Phase lock is depicted on each coherence plot as (*): the red (*) represents phase lead ((A) EMG is leading COPy; (B) Zo is leading COPy) and the green (*) represents phase lag ((A) EMG is lagging behind COPy; (B) Zo is lagging behind COPy). The alternating behaviour between phase lead and lag is clearly shown with a distinct change in this behaviour from pre- to post-exercise in both EMG-COPy and Zo-COPy.

Table 3-18: COP cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: mean value of the transfer function pairs during phase lock

		F	M	Pre	Post	LF	VLF
lock	EMG*	0.003 (0.0002)Y	0.003 (0.0002)Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	COPx	-0.0011 (0.002)	0.0007 (0.002)	-0.0009 (0.001)	0.0005 (0.001)	-0.0001 (0.001)	-0.0002 (0.001)
lead	EMG*	0.003 (0.0002)Y	0.003 (0.0002)Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	COPx	-0.0010 (0.002)	0.0008 (0.002)	-0.0008 (0.001)	0.0006 (0.001)	-0.0002 (0.001)	-0.0001 (0.001)
lag	EMG*	0.003 (0.0002)Y	0.003 (0.0002)Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	COPx	-0.0012 (0.002)	0.0006 (0.002)	-0.001 (0.001)	0.0005 (0.001)	-0.0001 (0.001)	-0.0004 (0.001)
lock	EMG*	0.003 (0.0002)Y	0.003 (0.0002)Y	0.002 (0.0002) ψ	0.005 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	COPY*	-0.029 (0.005)	-0.036 (0.005)	-0.034 (0.004)	-0.030 (0.004)	-0.032 (0.004)	-0.032 (0.004)
lead	EMG*	0.003 (0.0002)Y	0.003 (0.0002)Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	COPY*	-0.029 (0.005)	-0.036 (0.005)	-0.03 (0.004)	-0.03 (0.004)	-0.032 (0.004)	-0.032 (0.004)
lag	EMG*	0.003 (0.0002)Y	0.003 (0.0002)Y	0.002 (0.0002) ψ	0.004 (0.0002)	0.003 (0.0002) [^]	0.003 (0.0002) [^]
	COPY*	-0.029 (0.005)	-0.036 (0.005)	-0.03 (0.004)	-0.03 (0.004)	-0.032 (0.004)	-0.032 (0.004)
lock	Zo	-0.0011 (0.0001)	-0.0012 (0.0001)	-0.0013 (0.0002)	-0.0010 (0.0002)	-0.0012 (0.0002)	-0.0011 (0.0002)
	COPx	-0.0011 (0.002)	0.0007 (0.002)	-0.0009 (0.001)	0.0006 (0.001)	-0.0001 (0.001)	-0.0002 (0.001)
lead	Zo	-0.0012 (0.0003)	-0.0010 (0.0003)	-0.0009 (0.0003)	-0.001 (0.0003)	-0.0011 (0.0003)	-0.0011 (0.0003)
	COPx	-0.0010 (0.002)	0.0009 (0.002)	-0.0008 (0.001)	0.0006 (0.001)	-0.0001 (0.001)	-0.0001 (0.001)
lag	Zo	-0.0011 (0.0003)	-0.0013 (0.0003)	-0.0012 (0.0003)	-0.0011 (0.0003)	-0.0014 (0.0003)	-0.0009 (0.0003)
	COPx	-0.0011 (0.002)	0.0006 (0.002)	-0.0010 (0.001)	0.0005 (0.001)	-0.0001 (0.001)	-0.0004 (0.001)

Reported as least square mean (SEM)
Units for reported variables: EMG (V); COP(m); Zo(Ω)
* Significantly different between tests
Y Significantly different between tests within same gender
[^] Significantly different between tests within same frequency band
 ψ Significantly different between genders within same test

Table 3-19: COP cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: mean value of the transfer function pairs during phase lock (continued)

		F	M	Pre	Post	LF	VLF
lock	Zo	-0.0010 (0.0003)	-0.0011 (0.0003)	-0.0008 (0.0003)	-0.001 (0.0003)	-0.0010 (0.0003)	-0.0012 (0.0003)
	COPy*	-0.029 (0.005)	-0.036 (0.005)	-0.034 (0.004)	-0.032 (0.004)	-0.032 (0.004)	-0.032 (0.004)
lead	Zo	-0.0008 (0.0004)	-0.0018 (0.0004)	-0.0013 (0.0004)	-0.0013 (0.0004)	-0.0012 (0.0004)	-0.0014 (0.0004)
	COPy*	-0.029 (0.005)	-0.036 (0.005)	-0.030 (0.004)	-0.034 (0.004)	-0.032 (0.004)	-0.032 (0.004)
lag	Zo	-0.0017 (0.0008)	-0.0011 (0.0008)	-0.0007 (0.0007)	-0.002 (0.0007)	-0.0006 (0.0007)	-0.0022 (0.0007)
	COPy*	-0.029 (0.005)	-0.036 (0.005)	-0.03 (0.004)	-0.03 (0.004)	-0.032 (0.004)	-0.032 (0.004)
Reported as least square mean (SEM) Units for reported variables: EMG (V); COP(m); Zo(Ω) * Significantly different between tests							

Table 3-20: COP cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: transfer function gain between the signal pairs during phase lock

		F	M	Pre	Post	LF	VLF
EMG-COPx	Lock	0.010 (0.003)	0.013 (0.003)	0.013 (0.002)	0.011 (0.002)	0.013 (0.002)	0.010 (0.002)
	Lead	0.011 (0.003)	0.013 (0.003)	0.013 (0.002)	0.010 (0.002)	0.013 (0.002)	0.011 (0.002)^
	Lag	0.010 (0.004)	0.017 (0.004)	0.013 (0.003)	0.014 (0.003)	0.015 (0.003)	0.012 (0.003)
EMG-COPy	Lock†	0.020 (0.003)Y	0.017 (0.003)	0.021 (0.003)	0.016 (0.003)€	0.021 (0.003)	0.016 (0.003)^
	Lead†	0.020 (0.004)Y	0.019 (0.004)	0.021 (0.003)	0.018 (0.003)€	0.023 (0.003)	0.017 (0.003)^
	Lag*	0.020 (0.004)Y	0.021 (0.004)	0.021 (0.003)	0.015 (0.003)€	0.021 (0.003)	0.015 (0.003)^
Zo-COPx	Lock*	6.13 (1.65)	6.31 (1.65)	4.79 (1.31)	7.65 (1.31)	7.34 (1.31)	5.10 (1.31)
	Lead*	5.84 (2.17)	7.48 (2.17)	4.81 (1.74)	8.51 (1.74)	8.04 (1.74)	5.28 (1.74)
	Lag†	6.01 (0.91)#	4.58 (0.91)	4.87 (0.80)	5.72 (0.80)	6.86 (0.80)	3.73 (0.80)
Zo-COPy	Lock*	2.77 (1.22)	4.68 (1.22)Y	2.44 (0.89)	5.01 (0.89)	4.35 (0.89)	3.10 (0.89)
	Lead*	2.88 (1.21)	4.87 (1.21)Y	2.65 (0.96)	5.10 (0.96)	4.49 (0.96)	3.26 (0.96)
	Lag†	2.65 (0.74)	4.09 (0.74)	2.34 (0.68)	4.40 (0.68)	4.29 (0.68)	2.45 (0.68)
Reported as least square mean (SEM) Units for reported variables: EMG-COPx(m ² s/M); EMG-COPy(m ² s/M); Zo-COPx(Ω/ m ² s); Zo-COPy(Ω/ m ² s) * Significantly different between tests † Significantly different between frequency bands Y Significantly different between tests within same gender € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender ^ Significantly different between tests within same frequency band							

Table 3-21: COP cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: phase difference and average phase angle during phase lock

		F	M	Pre	Post	LF	VLF
EMG-COPx	Diff†	-1.80 (2.21)	1.69 (2.21)#	-0.36 (1.94)	0.25 (1.94)	-3.78 (1.94)	3.67 (1.94)
	Lead†	40.17 (1.51)#	39.74 (1.51)#	40.12 (1.35)€	39.79 (1.35)€	47.14 (1.35)	32.77 (1.35)
	Lag†	-41.44 (1.05)#	-37.96 (1.05)#	-40.60 (1.10)€	-38.80 (1.10)€	-48.80 (1.10)	-30.60 (1.10)
EMG-COPY	Diff†	26.90 (2.92)#Y	17.45 (2.92)#	31.02 (2.35)ψ	13.33 (2.35)	29.39 (2.35)^	24.96 (2.35)^
	Lead†	44.50 (1.02)#Y	41.93 (1.02)#	45.61 (0.98)ψ€	40.82 (0.98)€	54.30 (0.98)!^	32.13 (0.98)
	Lag*†	-27.71 (1.53)#	-31.75 (1.53)#	-24.88 (1.31)ψ€	-34.58 (1.31)€	-37.38 (1.31)^	-22.09 (1.31)^
Zo-COPx	Diff†	-0.14 (2.49)	1.31 (2.49)	-2.47 (2.42)	3.64 (2.42)	-3.81 (2.42)	4.97 (2.42)
	Lead†	45.09 (1.28)#	43.22 (1.28)	43.21 (1.46)€	45.10 (1.46)€	48.88 (1.46)	39.43 (1.46)
	Lag†	-45.15 (1.89)#	-40.54 (1.89)#	-43.30 (1.65)€	-42.38 (1.65)€	-51.99 (1.65)	-33.70 (1.65)
Zo-COPY	Diff†	-10.13 (6.17)#	-12.93 (6.17)#	-16.91 (4.92)	-6.16 (4.92)	-34.18 (4.92)	11.11 (4.92)
	Lead†	35.76 (2.20)	38.28 (2.20)	37.55 (1.97)	36.49 (1.97)	36.20 (1.97)	37.84 (1.97)
	Lag*†	-44.65 (5.39)#Y	-46.60 (5.39)#	-50.86 (4.04)€	-40.39 (4.04)€	-59.28 (4.04)^	-31.97 (4.04)
Reported as least square mean (SEM) Units for reported variables: phase diff (au), phase angle (°) † Significantly different between genders * Significantly different between tests ‡ Significantly different between frequency bands Y Significantly different between tests within same gender ^ Significantly different between tests within same frequency band € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender ψ Significantly different between genders within same test							

Table 3-22: COP cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: average coherence above the threshold of significance

	F	M	Pre	Post	LF	VLF
EMG-COPx†	0.3848 (0.004)#	0.3918 (0.004)#	0.3914 (0.004)€	0.3853 (0.004)€	0.3630 (0.004)	0.4137 (0.004)
EMG-COPy*†‡	0.5000 (0.016)#Y	0.4370 (0.016)#	0.5153 (0.012)	0.4216 (0.012)	0.4478 (0.012)^	0.4891 (0.012)^
Zo-COPx†	0.4188 (0.008)#	0.4204 (0.008)#	0.4129 (0.007)€	0.4262 (0.007)€	0.3934 (0.007)	0.4457 (0.007)
ZO-COPy†	0.5545 (0.025)#	0.5042 (0.025)	0.5248 (0.019)	0.5339 (0.019)€	0.5141 (0.019)	0.5446 (0.019)

Reported as least square mean (SEM)
Units for reported variables: au
† Significantly different between genders
* Significantly different between tests
‡ Significantly different between frequency bands
Significantly different between frequency bands within same gender
€ Significantly different between frequency bands within same test
^ Significantly different between tests within same frequency band
Y Significantly different between tests within same gender

Table 3-23: COP cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: percent time above the threshold of significant coherence

	F	M	Pre	Post	LF	VLF
EMG-COPx	61.90 (1.01)	62.78 (1.01)	63.86 (1.23)	60.82 (1.23)	74.80 (1.23)	49.88 (1.23)
EMG-COPy*†	80.15 (2.19)Y	73.27 (2.19)	85.25 (1.79)ψ	68.17 (1.79)	86.20 (1.79)^	67.22 (1.79)^
Zo-COPx	74.28 (1.91)	74.59 (1.91)	74.12 (1.91)	74.75 (1.91)	81.30 (1.78)	67.58 (1.78)
ZO-COPy†‡	92.41 (1.79)	84.43 (1.79)#	89.49 (1.61)€	87.35 (1.61)ψ€	92.44 (1.61)	84.40 (1.61)

Reported as least square mean (SEM)
† Significantly different between genders
* Significantly different between tests
‡ Significantly different between frequency bands
ψ Significantly different between genders within same test
Y Significantly different between tests within same gender
^ Significantly different between tests within same frequency band
€ Significantly different between frequency bands within same test
Significantly different between frequency bands within same gender

Table 3-24: COP cardio-postural interactions for males and females, pre- and post-exercise, and in each frequency band: percent time signal pairs were phase locked and above significant

	F	M	Pre	Post	LF	VLF
EMG-COPx†	12.87 (0.49)#	13.60 (0.49)	13.46 (0.56)€	13.00 (0.56)	11.58 (0.56)	14.89 (0.56)
EMG-COPy*†	17.61 (0.68)#Y	17.13 (0.68)#	18.95 (0.59)€	15.80 (0.59)€	14.57 (0.59)	20.17 (0.59)
Zo-COPx†	18.25 (1.05)#	17.14 (1.05)#	17.79 (0.94)€	17.60 (0.94)	14.74 (0.94)	20.65 (0.94)
ZO-COPy†	24.32 (1.23)#	22.05 (1.23)#	23.63 (0.98)€	22.73 (0.98)€	18.90 (0.98)	27.47 (0.98)
Reported as least square mean (SEM) * Significantly different between tests † Significantly different between frequency bands Y Significantly different between tests within same gender € Significantly different between frequency bands within same test # Significantly different between frequency bands within same gender						

3.3.5 Cardio-Postural Model

After the relationships between individual components of the cardiovascular and postural control systems were established, the effect of exercise on the interaction dynamics between the two systems was projected onto the cardio-postural model (Figure 3-14; Figure 3-15). Figure 3-14 summarizes the significant effects of inactive recovery after exercise on cardio-postural parameters (mean and variance) and interaction characteristics (transfer means and gain). Figure 3-15 summarizes the significant effects of inactive recovery after exercise on the phase difference and mean phase angle between the respective signal pairs. These figures provide graphical representation of the bi-directional interdependence between cardiovascular and posture controls.

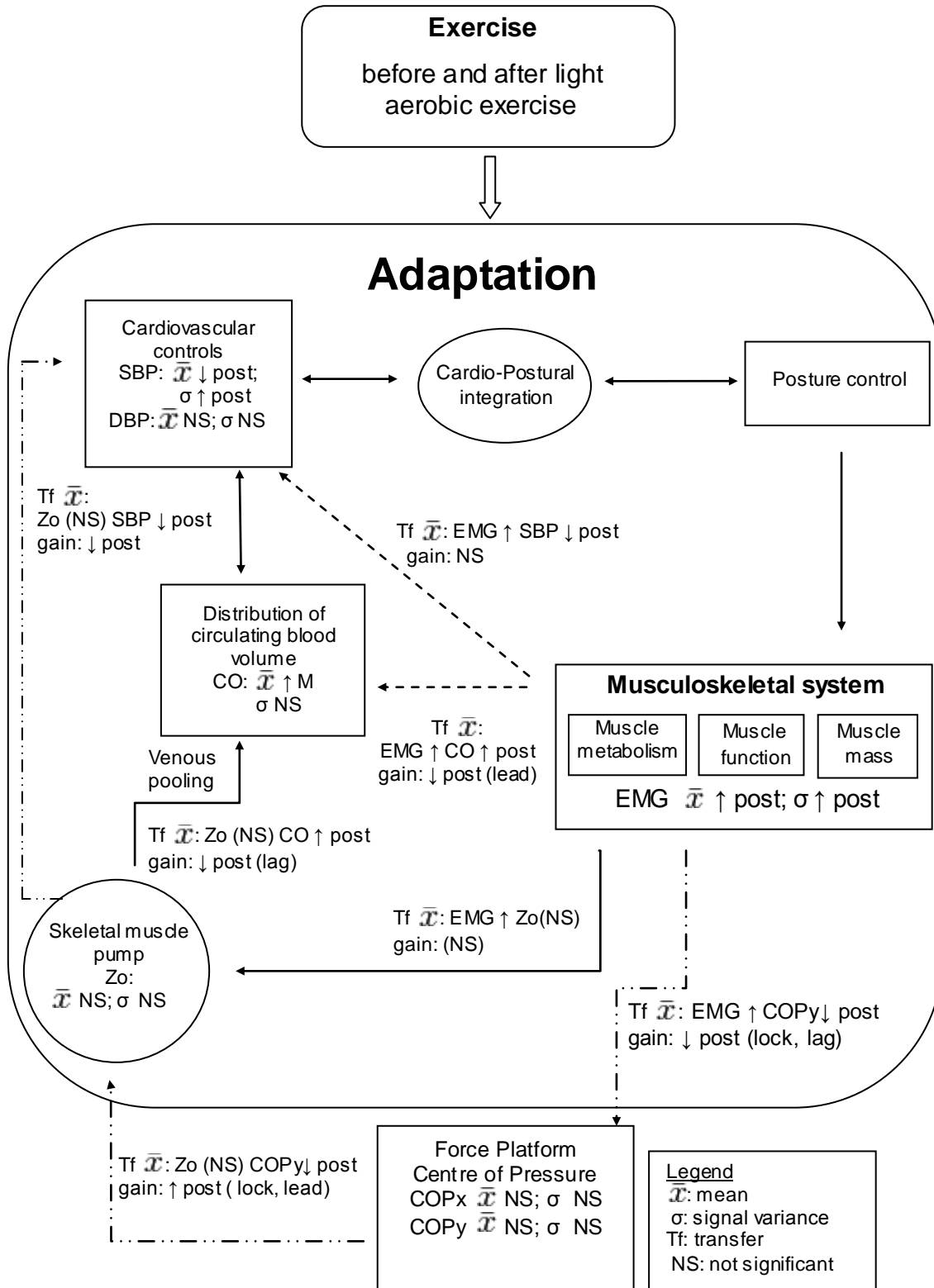


Figure 3-14: A summary of the effect of inactive recovery after exercise on the variables and transfer characteristics in the cardio-postural model. Significant effects are displayed for each variable and interaction.

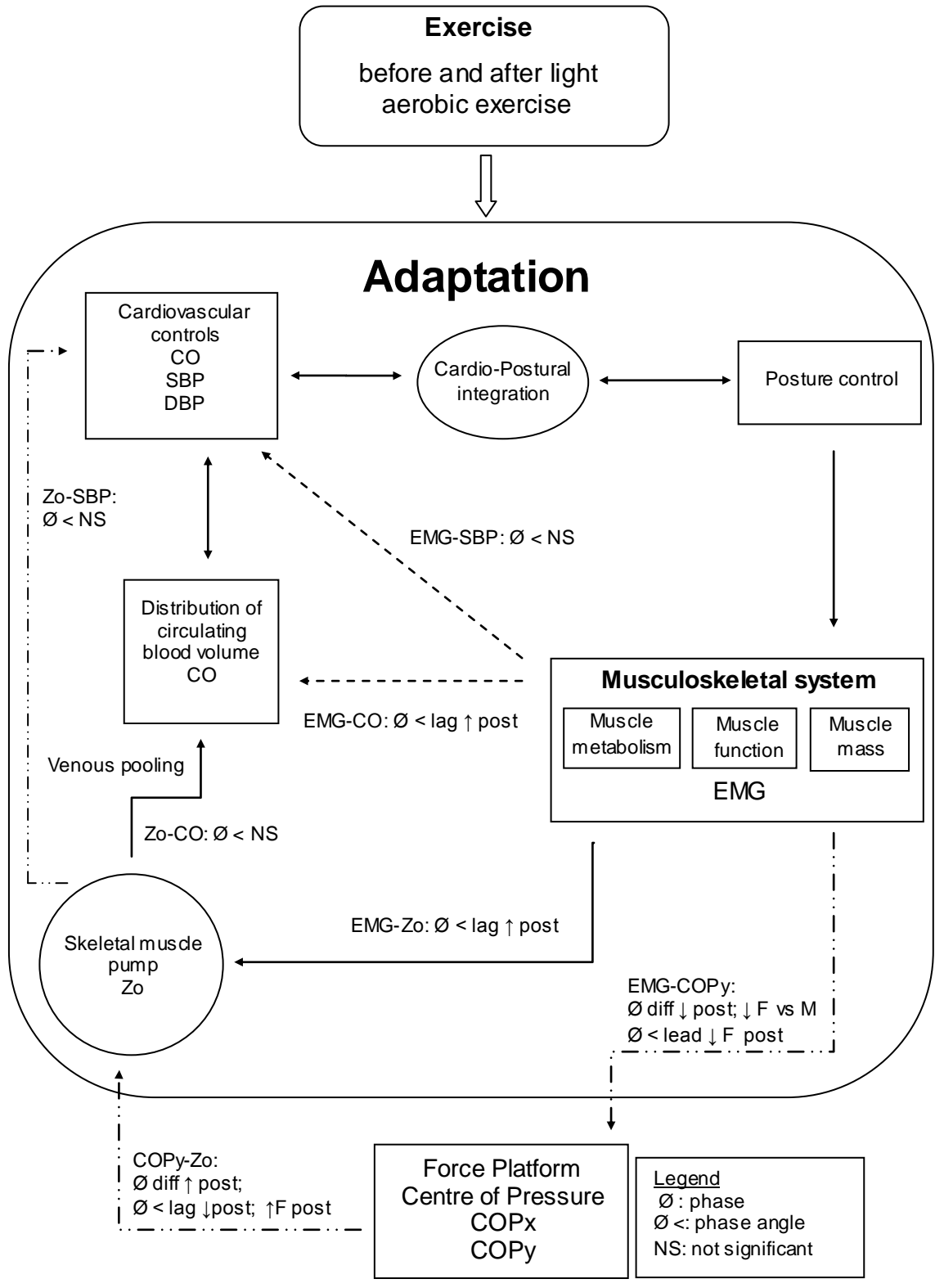


Figure 3-15: A summary of the effect of inactive recovery after exercise on average phase difference and average phase angle in the cardio-postural model. Significant effects are displayed for each interaction.

4 DISCUSSION

4.1 Cardio-Postural Model

This study examined cardiovascular and postural control interactions before exercise and during inactive recovery following light aerobic exercise to understand the dynamic, inter-dependent relationship between the two systems. The cardio-postural model (Figure 3-14; Figure 3-15) is an integration of the cardiovascular and postural control systems which can describe the adaptive responses of these systems to a given stressor such as inactive recovery following a mild exercise protocol. As one exercises, the contraction of lower-extremity musculature actively pumps blood back to the heart. After the cessation of exercise, the skeletal muscle pump must remain active to maintain adequate venous return. Activation of the skeletal muscle pump, in turn, results in changes in postural sway. Understanding this dynamic, interdependent relationship in healthy individuals will allow for increased understanding of how this relationship may be impaired in individuals with orthostatic hypotension or similar conditions which will lead to more effective treatment and prevention methods.

4.1.1 Skeletal Muscle Pump

The time delay between changes in lower leg EMG and corresponding changes in lower limb blood volume was reduced after exercise as indicated by the reduction in phase angle magnitude between lower leg EMG and lower leg impedance during phase lag (Garg, 2010). This served to minimize venous

pooling observed through the trend toward a decrease in lower leg blood volume (increase in impedance), though not statistically significant. The absence of an increase in lower leg blood volume with increased lower leg EMG variance further suggests there was greater reliance on the skeletal muscle pump to prevent venous pooling and orthostatic hypotension during inactive recovery (Carter et al, 1999; Carter et al, 2001; Halliwill, 2001; Stewart et al, 2004).

4.1.2 Vascular Response

The results indicate that cardiac output was sustained, in part, due to the increase in lower leg EMG activity that maintained lower leg blood volume (prevented additional venous pooling), thereby maintaining venous return. This suggests that the skeletal muscle pump was a primary mechanism to maintain venous return during inactive recovery (Carter et al, 1999; Carter et al, 2001; Halliwill, 2001; Stewart et al, 2004). The observed reduction in mean SBP during inactive recovery would result in a reduction in stroke volume, which suggests reduced venous return (Kirkman, 2007; Navare and Thompson, 2003; Carter et al, 1999). It may be inferred that despite a reduction in stroke volume, cardiac output was maintained due to a compensatory increase in heart rate (Halliwill, 2001; Convertino, 1998; Laszlo et al, 1998); this also indicates there may be a threshold that triggers increased activation of the skeletal muscle pump.

4.1.3 Centre of Pressure

The lower mean transfer function between lower leg EMG and AP centre of pressure indicates increased lower leg muscle activity created a stiffer system

(Winter et al, 1998) which led to a reduction in centre of pressure trajectory, particularly in the AP direction. Before exercise, changes in COP generally occurred prior to changes in lower leg EMG, and changes in lower leg EMG often occurred prior to changes in cardiovascular parameters. During inactive recovery, there was a shift in this behaviour in which changes in lower leg EMG often precipitated changes in COP, and changes in cardiovascular parameters, namely lower leg impedance, often precipitated changes in lower leg EMG. Additionally, despite a reduction in total AP sway, the sway that occurred was translated into a greater net change in lower leg blood volume. This indicates a shift in the driving mechanism in the cardio-postural relationship from postural control to cardiovascular control during inactive recovery (Garg, 2010).

The overall reduction in centre of pressure trajectory was contrary to previous research (Nardone et al, 1997; Nardone, et al 1998; Corbeil et al, 2003; Susco et al, 2004; Wilkins et al, 2004); this is likely due to variations in the duration and intensity of the exercise protocols utilized by each group. In this study, a 12-minute, sub-maximal exercise protocol was devised to provide mild stress to the cardiovascular system below the aerobic threshold with minimal fatigue of the lower-extremity muscles. Previous studies that reported increased postural sway or instability after exercise used longer exercise protocols at a higher intensity in which participants surpassed the aerobic threshold (Nardone et al, 1997; Nardone, et al 1998; Corbeil et al, 2003, Susco et al, 2004; Wilkins et al, 2004); therefore, the observed increase in COP trajectory or postural

instability in these studies was likely due to muscular fatigue, which may have impaired skeletal muscle pump efficiency.

There was a distinctive shift in the phase lead/lag behaviour of the phase locked signal pairs across all lower leg EMG and lower leg impedance/blood volume combinations during inactive recovery (Figure 3-3, Figure 3-8, Figure 3-13). This may be interpreted as an increase in skeletal muscle pump activity to maintain venous return where the alternating phase lead/lag behaviour represents the on/off timing of the pump mechanism; however this is speculative. Due to the inter-dependent relationship across all signal pairs in the cardio-postural model (Figure 3-15), a similar increase in this patterned behaviour is observed across the cardio-postural model (Figure A-1, Appendix D). When the phase lead/lag behaviour is observed for interactions in the skeletal muscle pump, vascular response, and COP at the same time (Figure A-1, Appendix D), a delayed cause/effect response may be sequentially observed throughout the cardio-postural model during inactive recovery. The exact causal time delays throughout the model have yet to be determined, and require further research.

4.2 Differences Between Men and Women

For the mean transfer function between lower leg EMG and lower leg impedance during phase lock, the value of lower leg EMG and lower leg impedance demonstrated inverse behaviour in men in which lower leg EMG increased and impedance decreased (lower leg blood volume increased) after exercise. Men demonstrated a greater change in lower leg impedance (blood volume) per change in lower leg EMG in the LF band. The higher mean cardiac

output in men for the lower leg EMG/CO and lower leg impedance/CO transfer functions during phase lock indicate a greater reliance on lower leg EMG and lower leg blood volume to maintain the same cardiac output. Therefore, men were able to produce a greater reduction in blood volume in the lower-extremities with each muscle contraction after exercise which suggests that men have a more efficient skeletal muscle pump than women. This is consistent with previous research that showed women experienced a greater decline in venous return and had a less effective compensatory vasoconstriction to correct for a reduction in cardiac output and mean arterial pressure (Carter et al, 1999; Carter et al, 2001; Halliwill, 2001; Convertino, 1998; Fu et al, 2004; Hachiya et al, 2012).

The data indicate that women rely less on the skeletal muscle pump to maintain venous return during inactive recovery. This is consistent with previous studies that suggest women tend to have a greater reliance on splanchnic redistribution to maintain venous return under orthostatic stress while men rely more on the skeletal muscle pump (White and Montgomery, 1996; Jarvis et al, 2010; Hachiya et al, 2012).

Women upheld a greater percent time above the threshold of significant coherence for the interaction between lower leg impedance and changes in AP centre of pressure; however, there was no difference in the percent time in phase lock above the threshold of significant coherence. This means that in women, the respective signals had a higher tendency to remain above the coherence threshold when not in phase lock. This is of little physiological significance at this time.

4.2.1 Effects of the female menstrual cycle

Hormonal changes during the menstrual cycle have been shown to influence components of cardiovascular regulation (such as baroreflex sensitivity), which could contribute to changes in orthostatic tolerance across the menstrual cycle, and the increased rate of orthostatic intolerance in women (Meendering et al, 2005). However, other studies have found no significant differences in the hemodynamic response to progressive orthostatic stress (head-up tilt and 20-minute stand test) (Meendering et al, 2005; Hirshoren et al, 2002). This suggests that there are compensatory changes within cardiovascular regulation that change across the menstrual cycle to maintain the same cardiovascular response.

The 8 female participants tested in this study were evenly distributed across the different phases of the menstrual cycle (Table 3-2). This sample size was insufficient to project differences in cardio-postural control across the menstrual cycle. Further research is required to understand how the interaction characteristics may be affected across the menstrual cycle.

4.3 Differences Between Frequency Bands

Spectral analysis of heart rate and blood pressure variability show peaks in power in across a range of frequency bands (primarily HFa: 0.15-0.4Hz; LFa: 0.04-0.15Hz; VLFa: 0.003-0.04Hz) (Serrador et al, 1999; Stein and Kleiger, 1999). It is known that power in the HFa band is associated with parasympathetic (vagal) modulation of heart rate and blood pressure, and reflects respiration-

mediated heart rate variability (Pumprla et al, 2002; Serrador et al, 1999; Stein and Kleiger, 1999). The LFa band is associated primarily with sympathetic and parasympathetic modulation and baroreflex activity (Pumprla et al, 2002; Serrador et al, 1999; Stein and Kleiger, 1999). The LF/HF ratio has been shown to reflect an estimate of the balance between sympathetic and parasympathetic dominance (Stein and Kleiger, 1999; Pumprla et al, 2002); however, this is beyond the scope of the present work. The VLFA band is associated with variation in neurohumoral, specifically renin-angiotensin, and peripheral vasomotor responses (Pumprla et al, 2002; Serrador et al, 1999; Stein and Kleiger, 1999), and possibly physical activity (Serrador et al, 1999).

The LF (0.1-0.05Hz) and VLF (0.05-0.01Hz) bands designated in the present analyses are subsets within the larger LFa and VLFA bands. The subset bands were selected to exclude cardiorespiratory events and effects such as Mayer waves in an attempt to isolate cardio-postural contributions as much as possible. Previous research in our laboratory (Blaber, et al, 2009) revealed peaks in power for blood pressure variability in these regions that may be associated with cardio-postural control mechanisms.

Serrador et al (1999) demonstrated that EMG activity occurs in the VLFA and LFa bands with a strong positive gain between EMG and heart rate variability in these two bands during light physical activity.

The present data further confirm the presence of lower leg EMG activity with corresponding changes in the cardiovascular system (SBP, DBP, CO, lower

leg blood volume) within these bands. While further analyses are necessary to understand the contribution of lower leg EMG or physical activity to cardiovascular regulation, the present data suggest that this plays an important role in long-term cardiovascular regulation.

4.4 Conclusion

This study provided further evidence in support of a frequency-dependent, bi-directional relationship between cardiovascular and postural controls. There was an increased dependence on the postural control system to maintain venous return after light aerobic exercise in both men and women. Although reliance on the skeletal muscle pump was greater in men than in women after exercise, this may be due to a combination of higher skeletal muscle mass and blood volume in men. Increased understanding of cardio-postural interaction dynamics in healthy individuals will provide greater insight into conditions such as orthostatic hypotension in which these cardio-postural relationships may be impaired. This may lead to improved treatment and prevention methods for individuals with such conditions.

4.5 Limitations and Future Work

4.5.1 Limitations

The work rate on the cycle ergometer was fixed based on gender, not by physiological standards such as a specified percent $\dot{V}O_2\text{max}$ or target heart rate. No physiological measures or rate of perceived exertion were obtained during the exercise period to estimate exertion.

We were unable to control certain environmental factors such as temperature, which may have affected individual participant's thermoregulatory response to exercise and, therefore, changed the cardiovascular response and cardio-postural interactions.

The sample size for this study was low and was further reduced due to loss of data as the result of computer and equipment malfunctions. The small number of female participants prevented the analysis of the effect of the menstrual cycle on the cardio-postural relationship.

4.5.2 Future Work

In the wavelet transform coherence analyses, cardiac output was used for comparison instead of the individual heart rate and stroke volume responses. To fully understand the cardio-postural interactions, heart rate and stroke must be considered independently as there could be inverse, compensatory responses that are not evident in the overall cardiac output response. Additionally, a means of quantifying the baroreflex has yet to be incorporated into the present model. This is an important component of cardiovascular regulation and must be considered in the cardio-postural relationship.

Presently, we have been unable to determine the time delays within the cardio-postural model, specifically the time delay for a change in calf venous blood volume due to increased EMG activity to yield a significant effect on blood pressure or cardiac output. These time delays must be determined to understand the interaction dynamics within the model.

In the present analyses, significance was determined based on the behaviour of signal pairs or the percent time above a threshold of coherence in each frequency band. This may not be an accurate way to determine the true cause and effect relationship between cardiovascular and postural controls. An “event” needs to be defined in terms of a given period of time in which two signals are phase locked and cross the threshold of significant coherence similar to methods for assessing baroreflex sensitivity.

Little consideration has been given to the behaviour of the signal pairs when phase locked below the threshold of coherence or how many times the signal pairs cross the coherence threshold. The overall percent time signals are phase locked and above significant coherence may be reduced from pre- to post-exercise, but the number of coupled events could be higher and indicate greater skeletal muscle pump activity. This behaviour may be useful to understand the on/off triggering characteristics of skeletal muscle pump activity within the cardio-postural model.

Additional work is required to fully understand the interaction dynamics within the cardio-postural model. Once these characteristics have been clearly defined in a healthy population, this model could serve as a useful diagnostic tool to isolate underlying causes in fall-prone individuals or those with conditions such as postural orthostatic hypotension.

5 APPENDICES

Appendix A

Informed Consent by Participants in a Research Study

The University and those conducting this research study subscribe to the ethical conduct of research and to the protection at all times of the interests, comfort, and safety of participants. This research is being conducted under permission of the Simon Fraser Research Ethics Board. The chief concern of the Board is for the health, safety and psychological well-being of research participants.

Should you wish to obtain information about your rights as a participant in research, or about the responsibilities of researchers, or if you have any questions, concerns or complaints about the manner in which you were treated in this study, please contact the Director, Office of Research Ethics by email at hal_weinberg@sfu.ca or phone at 778-782-6593.

Your signature on this form will signify that you have received this Informed Consent by Participants in a Research Study Document which describes the procedures, whether there are possible risks, and benefits of this research study, that you have received an adequate opportunity to consider the information in the documents describing the study, and that you voluntarily agree to participate in the study.

Title: The Effect of Exercise on the Cardio-Postural Relationship

**Investigator Name: Michelle Bruner
Andrew Blaber**

Investigator Department: Biomedical Physiology and Kinesiology

Purpose and goals of this study:

This study aims to improve our understanding of orthostatic hypotension which can lead to syncope and falls through the investigation of the integration of the cardiovascular and postural control systems under orthostatic stress. Additionally, this study will investigate the effect of natural hormone level variations on this cardio-postural relationship in female participants.

What the participants will be required to do:

You will first arrive at the Aerospace Physiology Lab at SFU (the Lab) to perform a sit-to-stand test. Upon arrival in the lab, you will be asked to remove your socks, shoes, and all items from your pockets, and complete the Medical History Form. Your height; weight; lower leg muscle girth, length, and skinfold will be

recorded. Additionally, female participants will be asked to report the number of days since their last menstruation, use of prescription contraceptives, and the type/dose of the prescription contraceptive, if applicable, to determine the phase of the menstrual cycle at the time of testing and determine if variations in hormone levels effect the cardio-postural relationship. You will be fitted with a 1) non-invasive blood pressure monitor, photoplethysmography, on the right index finger; 2) three-lead ECG; 3) surface electromyography (EMG) electrodes to monitor muscle activity in four muscles of each lower leg: tibialis anterior, lateral soleus, and medial and lateral gastrocnemius. Postural sway data will be obtained from a force platform; 4) impedance plethysmography of the left lower leg. Impedance plethysmography is the same non-invasive technology used in home bioelectrical impedance devices or in certain weight scales that provide body mass index and percent fat measurements. All equipment is CSA approved, and all electrodes are non-invasive, surface electrodes. You will then perform an 11 minute seated-to-quiet stance protocol to obtain a baseline control. You will be asked to sit in a straight-backed chair with their knees and hips at 90°, with feet positioned over the force platform for 5 minutes. After 5 minutes, you will then stand quietly on the force platform in an upright position with arms at your side and eyes closed with a blindfold while maintaining eye-level gaze for 6 minutes. Assistance will be provided in posture changes from seated to upright stance.

You will then perform an exercise protocol that will be carried out on a Jaeger ER 800 cycle ergometer. The exercise protocol consists of a 2 minute warm-up at 25W, followed by 80W or 100W for 10 minutes for female and male participants, respectively. You must maintain 80RPM throughout the duration of the exercise test. Immediately upon cessation of exercise, a 6 minute stand test will be conducted with eyes closed and forward gaze.

Risks to the participant:

The risks associated with this study are minimal. Though rare, the ECG electrodes may cause a mild, temporary rash that should subside in one to two days. As a participant in this study, you must be a healthy, young adult with no history of cardiovascular or respiratory disease, therefore, the risk of syncope should be minimal. However, should a you feel faint or lightheaded, an attendant will be present at all times to assist you into a seated position on a chair next to the force platform. If the test is stopped due to said symptoms, testing will resume only upon symptom resolution if you are willing to continue.

Benefits of study to the development of new knowledge:

The objective of this study is to further the development of an integrated methodology that will improve our understanding of the integration of postural and cardiovascular controls in relation to the prevention of orthostatic hypotension which can lead to syncope and falls. This is of partiucular concern in

conditions such as postural orthostatic tachycardia syndrome, mild traumatic brain injury, and ageing as the current financial cost of falls in Canada is estimated to be \$3 billion annually.

Additionally, tests of postural stability are used to assess a variety of conditions, particularly head injuries, which frequently occur during athletic events. Clinicians will often conduct such tests as soon as symptoms are reported. However, the exercise-induced stress on the cardiovascular system may decrease postural stability resulting in a false-positive test for a head injury. This study will determine how exercise-induced stress on the cardiovascular system influences postural stability. This will help clinicians determine when to administer postural stability tests to more accurately assess head injuries in athletes.

Statement of confidentiality: The data of this study will maintain confidentiality of your name and the contributions you have made to the extent allowed by the law.

All data that is collected will be recorded according to a randomized participant number, and all reference to your name or identifiers that may lead to linkage of you and your data will be removed. The experimental data will be securely stored on a CD/DVD disk in a locked cabinet in Aerospace Physiology Lab, and will be retained for a period of 3 years for reference purposes. The signed informed consent forms will be stored in a locked cabinet within the lab.

Inclusion of names of participants in reports of the study:

Names will not be included in study reports.

Contact of participants at a future time or use of the data in other studies:

You will not be contacted after completion of this study unless you consent to future contact below. Data from this study may be used in future studies; however, any link to your name and your data will have already been permanently removed.

I understand that I may withdraw my participation at any time. I also understand that I may register any complaint with the Director of the Office of Research Ethics.

Dr. Hal Weinberg, Director, Office of Research Ethics at hal_weinberg@sfu.ca or 778-782-6593

Dr. Andrew Blaber, Associate Professor, Dept. Biomedical Physiology and Kinesiology at ablaber@sfu.ca or 778-782-3276

I may obtain copies of the results of this study, upon its completion by contacting:

Michelle Bruner, Department of Biomedical Physiology and Kinesiology, Simon Fraser University, 8888 University Drive, Burnaby, BC, V5A 1S6.

I understand the risks and contributions of my participation in this study and agree to participate.

The participant shall fill in this area. Please print legibly.

Participant Last Name:

Participant First Name:

Participant Contact Information:

The researchers may contact me at a future time for related studies or to use this data in other studies:

yes

no

Participant Signature:

Date:

**Simon Fraser University
Study Detail Document**

Title: The Effect of Exercise on the Cardio-Postural Relationship

Investigator's Name: Michelle Bruner

Andrew Blaber

Investigator's Department: Biomedical Physiology and Kinesiology

Place

Shrum Science K8624 (Aerospace Physiology Laboratory), Department of Biomedical Physiology and Kinesiology, Simon Fraser University

Who are the participants in this study?

Participants will include healthy, young, male and female adults (19-30 years of age) with no history of cardiovascular, respiratory, or neurological disease, or major musculoskeletal injuries as specified in the Medical History Form. The presence of any condition listed on the Medical History Form will disqualify an individual from participation in this study.

How are the participants recruited?

Participants will be recruited by the Investigator (Michelle Bruner) from students/staff/faculty of Simon Fraser University as well as the surrounding community. Recruitment will take place through the posting of advertisements and speaking directly with prospective volunteers. Upon receiving approval from participants, he/she will be given a copy of the Informed Consent by Participants in a Research Study. Participants will receive fifteen dollars compensation for time and travel.

Overall goals of study?

This study aims to improve our understanding of orthostatic hypotension which can lead to syncope and falls through the investigation of the integration of the cardiovascular and postural control systems under orthostatic stress. Additionally, this study will investigate the effect of natural hormone level variations on this cardio-postural relationship in female participants.

What will the participants be required to do?

Participants will first arrive at the Aerospace Physiology Lab at SFU (the Lab) to perform a sit-to-stand test. Upon arrival in the lab, participants will be asked to remove their socks, shoes, and all items from their pockets. Participants' height; weight; lower leg muscle girth, length, and skinfold; and general medical history and present medications will be recorded. Additionally, female participants will be asked to report the number of days since their last menstruation, use of prescription contraceptives, and the type/dose of the prescription contraceptive if applicable to determine the phase of the menstrual cycle at the time of testing and determine if

variations in hormone levels effect the cardio-postural relationship. Participants will be fitted with a 1) non-invasive blood pressure monitor, photoplethysmography, on the right index finger; 2) three-lead ECG; 3) surface electromyography (EMG) electrodes to monitor muscle activity in four muscles of each lower leg: tibialis anterior, lateral soleus, and medial and lateral gastrocnemius. Postural sway data will be obtained from a force platform; 4) impedance plethysmography of the left lower leg. Impedance plethysmography is the same non-invasive technology used in home bioelectrical impedance devices or in certain weight scales that provide body mass index and percent fat measurements. All equipment is CSA approved, and all electrodes are non-invasive, surface electrodes. Participants will then perform an 11 minute seated-to-quiet stance protocol to obtain a baseline control. Participants will be asked to sit in a straight-backed chair with their knees and hips at 90°, with feet positioned over the force platform for 5 minutes. After 5 minutes, participants will then stand quietly on the force platform in an upright position with arms at their side and eyes closed with a blindfold while maintaining eye-level gaze for 6 minutes. Assistance will be provided in posture changes from seated to upright stance.

Participants will then perform an exercise protocol that will be carried out on a Jaeger ER 800 cycle ergometer. The exercise protocol consists of a 2 minute warm-up at 25W, followed by 80W or 100W for 10 minutes for female and male participants, respectively. Participants must maintain 80RPM throughout the duration of the exercise test. Immediately upon cessation of exercise, a 6 minute stand test will be conducted with eyes closed and forward gaze.

Equipment and devices used:

Electromyography: surface EMG will be obtained for four bilateral lower leg muscles: tibialis anterior, lateral soleus, and medial and lateral gastrocnemius. Transdermal differential recording of signals will be performed using the 8 channel EMG system, *Bengoli-8* (Delsys Inc., MA, USA).

Electrocardiography: ECG signals will be aquired using custom equipment from *LifePak 8* (Medtronic Inc, Minnesota, USA) with the Lead II configuration of ECG electrode placement.

Blood Pressure: Blood pressure will be monitored continuously through a non-invasive photoplethysmography finger cuff placed on the right 3rd finger from *Finapres* (Ohmeda, Inglewood, CO, USA).

Impedance Plethysmography: Changes in blood volume in the lower leg will be measured via *NCCOM 3 Cardiodynamic Monitor* (BioMed Inc, Irvine, CA, USA) with Ag/AgCl ECG electrodes. One electrode set will be placed inferior to the knee joint and the other electrode set will be placed at the ankle of the left leg.

Postural Sway: Postural sway data will be derived from center of pressure trajectory coordinates collected from the *Accusway plus Force Platform* (Advance Mechanical Technologies Inc, Watertown, MA).

Data Acquisition: Data will be acquired using National Instruments DAQ card with custom software on a digital computer. The data will be sampled at 1000Hz prior to storage for further analyses.

Risks to the participant, third parties, or society:

The risks associated with this study are minimal. Though rare, the ECG electrodes may cause a mild, temporary rash that should subside in one to two days. All participants in this study will be healthy, young adults with no history of cardiovascular or respiratory disease, therefore, the risk of syncope should be minimal. However, should a participant feel faint or lightheaded, an attendant will be present at all times to assist them into a seated or supine position on a chair or bed that will be next to the force platform. If the test is stopped due to said symptoms, testing will resume only upon symptom resolution if the participant is willing to continue.

How will confidentiality and anonymity be assured?

All data will be recorded according to a randomized participant number and all reference to the individuals' names or identifiers that may lead to linkage of the participants and their data will be removed. The experimental data will be securely stored on a CD/DVD in a locked cabinet in the Aerospace Physiology Lab and will be retained for 3 years for reference purposes. The signed informed consent forms will be stored in a locked cabinet within the lab.

Benefits of this study to the development of new knowledge:

The objective of this study is to further the development of an integrated methodology that will improve our understanding of the integration of postural and cardiovascular controls in relation to the prevention of orthostatic hypotension which can lead to syncope and falls. This is of particular concern in conditions such as postural orthostatic tachycardia syndrome, mild traumatic brain injury, and ageing as the current financial cost of falls in Canada is estimated to be \$3 billion annually.

Additionally, tests of postural stability are used to assess a variety of conditions, particularly head injuries, which frequently occur during athletic events. Clinicians will often conduct such tests as soon as symptoms are reported. However, the exercise-induced stress on the cardiovascular system may decrease postural stability resulting in a false-positive test for a head injury. This study will determine how exercise-induced stress on the cardiovascular system influences postural stability. This will help clinicians determine when to administer postural stability tests to more accurately assess head injuries in athletes.

This Medical History Form is to be completed by you (the participant) prior to the start of testing. Please (√) all that pertain to you to the best of your knowledge, and provide any necessary explanations below. This information will remain confidential.

No current or acute illness or disease (sinus, respiratory, infection, etc) within 2 weeks of testing	
No history of a. Congenital conditions b. Neurological disease c. Mental illness d. Moderate or severe brain injury	
No hormonal imbalance	
No severe visual impairment	
No neurological disorder (nerve damage, vertigo, tremors, migraine headaches, paresthesia, kinesthesia, etc)	
No prior lumbar or lower extremity surgery	
No other orthopedic surgery within 12 months	
No lower extremity injuries within 6 months or other orthopedic injuries within 4 weeks	
No cardiovascular conditions (high/low blood pressure, tachycardia, blood clot, arrhythmia, etc)	
No current prescription for alpha or beta blockers	

Current Prescriptions: _____

Female Participants: Date of the onset of last menstrual cycle: _____

Avg duration of menstruation: _____ Avg duration of menstrual cycle: _____

Other:

Appendix B

This section contains the mathematical description of the wavelet transform coherence method used to analyze the data collected in this study outlined in section 2.2.2. This has been adapted from Garg (2010) with permission.

Wavelet Transform Coherence

The cross wavelet transform is defined as the convolution of the scaled mother wavelet function with the analyzed function $g(t)$

$$W(s, \tau) = \int g(t) \psi_s(t - \tau) dt \quad (1)$$

The Morlet wavelet is the commonly used mother wavelet for the analysis of physiological signals. The function for the Morlet Wavelet (ψ_0), which is a plane wave modulated by a Gaussian window, is defined in equation 2.

$$\psi_0(\eta) = \pi^{-1/4} e^{i\omega_0\eta} e^{-\eta^2/2} \quad (2)$$

where ω_0 is the dimensionless frequency and η is the dimensionless time.

The discrete form of the cross wavelet transform and the mother wavelet function for a sequence x_n , is shown in equations 3 and 4.

$$W_n(s) = \sum_{n'=0}^{N-1} X_{n'} \psi^* \left[\frac{(n' - n)\delta t}{s} \right] \quad (3)$$

$$\psi \left[\frac{(n' - n)\delta t}{s} \right] = \left(\frac{\delta t}{s} \right)^{1/2} \psi_0 \left[\frac{(n' - n)\delta t}{s} \right] \quad (4)$$

The Morlet coefficient, ω_0 defines the balance between the frequency and time resolution where $\omega_0 > 6$ is the minimum requirement as per the admissibility condition (Farge, 1992). For our analysis and frequency resolution requirements, we tested the coherence estimator in $6 < \omega_0 < 30$ for its statistical acceptance.

The wavelet power density estimator of x_n is defined as:

$$W_n^{xx}(s) = W_n^x W_n^{x*} \quad (2)$$

where W_n^* is the complex conjugate of the wavelet coefficient W_n .

For the mother wavelet, the scale to frequency transformation is defined through the Fourier wavelength (equation 6) (Torrence and Compo 1998).

$$\lambda = \frac{1}{f} = \frac{4\pi s}{\omega_0 + \sqrt{2 + \omega_0^2}} \quad (6)$$

The squared wavelet coherence estimator is defined as the squared absolute value of the smoothed cross-wavelet spectrum, normalized by the smoothed power spectrum of the two signals.

$$\hat{C}_n^2(s) = \frac{\left| \langle W_n^{xy}(s) \cdot s^{-1} \rangle \right|^2}{\langle |W_n^{xx}(s)| \cdot s^{-1} \rangle \langle |W_n^{yy}(s)| \cdot s^{-1} \rangle} \quad (7)$$

The $\langle \square \rangle$ is the smoothing operator shown in equation (8) as defined by Torrence and Webster (1998), and equation (9) defines the cross wavelet transform of two time series x_n and y_n , where * denotes the complex conjugate.

$$\langle W \rangle = \left[(c_1 \omega_n(s) * e^{-n^2/2s^2})_n * c_2 \prod(\delta j_0 s) \right]_s \quad (8)$$

$$W_n^{xy} = W_n^x(s) W_n^{y*}(s) \quad (9)$$

In equation (8), c_1 and c_2 are the normalization constants, and \prod is the rectangular function (Grinsted et al, 2004). For the Morlet wavelet, the value of δj_0 is equal to 0.6 for value of ω_0 equal to 6 (Torrence and Compo, 1998).

Statistical Estimations of the Wavelet Transform Coherence Method

Theoretical Coherence

The theoretical coherence estimation was based on the model of a single input, single output of a linear time invariant system (Pinna and Maestri, 2001). The theoretical coherence between X(t) and Y(t) was given by:

$$\gamma^2(f) = \frac{1}{1 + G_{NN}(f)/G_{XX}(f)} \quad (10)$$

Where $G_{XX}(f)$ and $G_{NN}(f)$ were the spectral density functions of the input signal, X(t), and the noise that was added to get the output signal, Y(t), respectively. In order to apply the model to our time- frequency analysis, we used the wavelet power spectrum:

$$\gamma_n^2(s) = \frac{1}{1 + W_n^{NN}(s)/W_n^{XX}(S)} \quad (11)$$

Equation (11) shows that the theoretical coherence is effectively determined by the signal to noise ratio of the system. Therefore, the simplest way to obtain different levels of theoretical coherence would be to change the amount of variance of noise relative to the input signal, keeping all other parameters constant (Pinna and Maestri, 2001).

Bias and Standard Deviation Estimation

Statistical error of the wavelet transform coherence method with respect to theoretical coherence was calculated to validate the new method. Simulated signals were generated to closely resemble the real signal in the analysis. EMG and SBP signals were independently simulated.

Similar to Bonato et al. (2001), EMG signal obtained from surface electrodes was modelled as a filtered Gaussian noise signal. A shaping filter was used with the following transfer function:

$$H(f) = \frac{k^2 f h^2 f^2}{(f^2 + f_l^2)(f^2 + f_h^2)^2} \quad (12)$$

f_l = bandpass low cut off frequency

f_h = bandpass high cut off frequency

$$k = 1.699 / f_h$$

The EMG signal was synthesized using the above transfer function with the low and high cut off frequency at 0.01 and 0.5 Hz, respectively.

The SBP signal was modelled as a band-pass filtered white noise signal in the band 0.01 – 0.5 Hz to simulate an arbitrary SBP signal. All signals were generated for 2400 data points at a sampling frequency of 10Hz to create a data length equal to 4 minutes. These signals formed the input, $X(t)$, to the single input, single output model for a physiological system.

The principal input signals, $X(t)$, were obtained as described above. In accordance with the single input, single output system model for most physiological systems, the output signal, $Y(t)$, was obtained by the addition of Gaussian white noise with zero mean to $X(t)$.

The bias and the standard deviation (SD) estimates for the modulus of the transfer function for the single input, single output system have been defined by Pinna and Maestri (2001) as:

$$bias(f) = \left(\frac{1}{N} \sum_{i=1}^N |\hat{H}_i(f)| \right) - |H(f)| = \overline{|\hat{H}(f)|} - |H(f)| \quad (13)$$

$$SD(f) \cong \sqrt{\frac{1}{N-1} \sum_{i=1}^N (|\hat{H}_i(f)| - \overline{|\hat{H}(f)|})^2} \quad (14)$$

These estimates were adapted to the time-frequency estimation using the wavelet transform coherence as the transfer function.

The bias and the standard deviation for the coherence estimate were obtained as

$$bias(k) \cong \left(\frac{1}{N} \sum^N C^2(k) \right) - k = \overline{C^2(k)} - k \quad (15)$$

$$s.d.(k) \cong \sqrt{\frac{1}{N-1} \sum^N (C^2(k) - \overline{C^2(k)})^2} \quad (16)$$

where k was the theoretical coherence level (γ^2) from 0.05 to 0.95 in 0.1 steps, and $C^2(k)$ was the calculated wavelet transform coherence estimator value associated with the theoretical coherence level equal to k . This procedure was repeated for the different wavelet coefficients $\omega_0 = 6, 10, 15, 20$ and 30 .

Synthesized input/output signal pairs ($N = 1000$) were generated for EMG and SBP, respectively.

Threshold and Error Rates for the Coherence Estimator

The value of the coherence estimator for two completely uncoupled signals provides information about the significance threshold values for the particular coherence estimator. The threshold defines the value above which the coherence value will be considered significant and the two signals have linearly dependent behaviour at that time point.

To find the threshold for the wavelet transform coherence estimator, signals for the SBP and EMG were synthesized as defined previously. Using the single input, single output system model, the output signals were obtained with added white noise, but the variance was kept at a level that gave a signal to noise ratio $\ll 1$, which provided a theoretical band coherence value close to zero. The input/output pairs were then created and checked for the threshold of the wavelet transform coherence estimator for different values of ω_0 .

The coherence was estimated between each input/output pair and averaged over iterations to give a coherence time series; the empirical sampling distribution (frequency histogram) was computed for each frequency band. The threshold for zero coherence $T(f)$ was set at the $100(1-\alpha)$ percentile of the coherence sampling distribution, where α is the significance level of the statistical test kept at 95% confidence or 0.05 (Fisher and Van Belle, 1993).

The error rates were found relative to the threshold values obtained from the previous step. The false negative rate (β) was defined as the number of wavelet transform coherence estimator outputs lower than the threshold value when there was coupling between the input and output signals. The false negative rate was calculated for different variance values of the added noise in order to get theoretical coherence to range from 0.05 to 0.95 in steps of 0.1 .

Appendix C

Sample size calculations:

Source	Variable	Difference to Detect	SD or SEM	α	Power	Est. Sample Size
Garg 2010* Pg 56	Tfmean COPy	2.6 (20% Δ)	0.89	0.05	0.8	7
Garg 2010* Pg 55	Tfmean CO	0.3 (20% Δ)	0.12	0.05	0.8	8
Garg 2010 Pg 59	Coherence (EMG-COPy)	0.028 (7% Δ)	0.017	0.05	0.8	14
Garg 2010 Pg 53	Phase angle EMG-COPy (lag)	12.46 (28% Δ)	4.41	0.05	0.8	7
Garg 2010 Pg 52	Phase angle EMG-SBP (lag)	9.65 (21% Δ)	4.90	0.05	0.8	11
Carter et al 2001 (pg 1903)	Mean SBP	8 (6% Δ)	5	0.05	0.8	15
Carter et al 2001 (pg 1904)	Mean CO	1.5 (30% Δ)	0.8	0.05	0.8	12
Transfer means are with respect to EMG SD: standard deviation SEM: standard error of the mean * difference observed in the literature applied to data collected in our lab						

Across the literature, there was approximately a 20 percent reduction in postural stability parameters (sway velocity, BESS test error score, sway area, and mean COP in the ML and AP directions) (Corbiel et al, 2003; Lepers et al, 1997; Fox et al, 2008; Wilkins et al, 2004). To estimate the necessary sample size required to observe a significant change in COP trajectory, a 20 percent change in AP COP trajectory was applied to data obtained in our lab (Garg 2010).

Appendix D

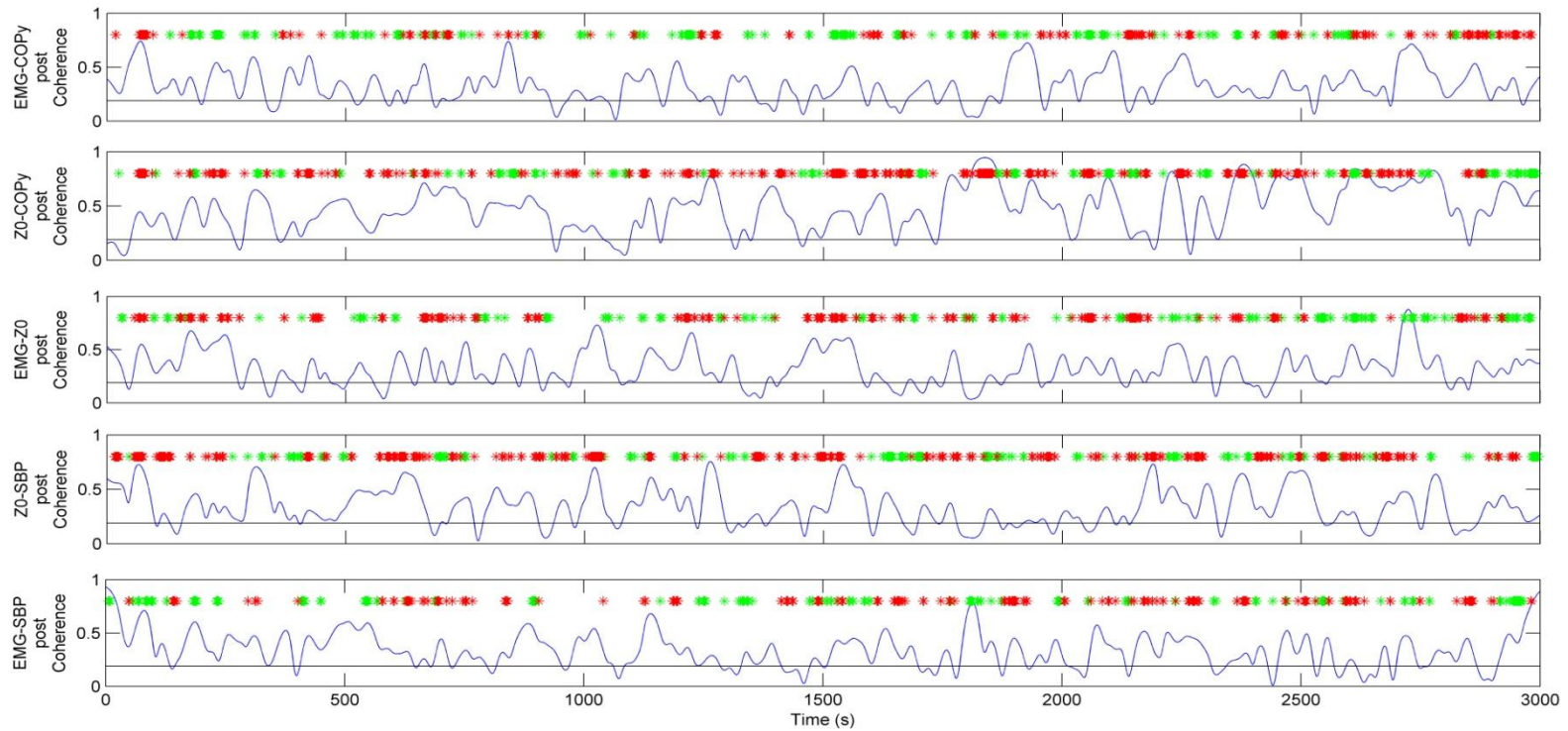


Figure A-1: Coherence time series for EMG-COPy, Zo-COPy, EMG-Zo, Zo-SBP, and EMG-SBP post-exercise interactions in the LF band over the 5 minutes under analysis with corresponding periods of phase lock between the signal pairs for a single participant (#15). The straight line represents the significance threshold (0.1894). Phase lock is depicted on each coherence plot as (*): the red (*) represents phase lead (EMG/Zo is leading the respective signal) and the green (*) represents phase lag (EMG/Zo is lagging behind the respective signal). The alternating behaviour between phase lead and lag is clearly shown. There is a sequential effect in the change between phase lead/lag across the interactions representative of the cardio-postural model.

Appendix E

Data tables from Tukey's post-hoc analyses for all interactions.

Table A-1: Mean value of the transfer function variables when the signal pairs were phase locked (lead and lag) for post-hoc analyses of skeletal muscle pump interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
lock	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	SBP	112(3)	115(3)	107(3)	111(3)	113(3)	115(3)	107(3)	111(3)
lead	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	SBP	112(3)	115(3)	107(3)	111(3)	112(3)	114(3)	106(3)	111(3)
lag	EMG	0.002 (0.0003)	0.003 (0.0003)	0.002 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	SBP	112(3)	115(3)	106(3)	112(3)	113(3)	115(3)	107(3)	112(3)
lock	EMG	0.002 (0.0003)	0.003 (0.0003)\$	0.004 (0.0003)\$	0.004 (0.0003)\$	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	DBP	70(2)	71(2)	69(2)	70(2)	70(2)	70(2)	69(2)	70(2)
lead	EMG	0.002 (0.0003)	0.003 (0.0003)\$	0.004 (0.0003)\$	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.005 (0.0003)
	DBP	70(2)	71(2)	69(2)	70(2)	69(2)	71(2)	69(2)	70(2)
lag	EMG	0.002 (0.0003)	0.003 (0.0003)	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	DBP	70(2)	71(2)	69(2)	69(2)	70(2)	70(2)	69(2)	70(2)
lock	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	CO	4.73 (0.25)	6.13 (0.25)\$	5.03 (0.25)	6.43 (0.25)\$	4.72 (0.25)	6.06 (0.25)\$	5.01 (0.25)	6.48 (0.25)\$
lead	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.005 (0.0003)
	CO	4.74 (0.25)	6.13 (0.25)\$	5.10 (0.25)	6.40 (0.25)\$	4.68 (0.25)	6.04 (0.25)\$	4.99 (0.25)	6.43 (0.25)\$
lag	EMG	0.002 (0.0003)	0.003 (0.0003)	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	CO	4.74 (0.25)	6.15 (0.25)\$	5.05 (0.25)	6.44 (0.25)\$	4.76 (0.25)	6.07 (0.25)\$	5.02 (0.25)	6.52 (0.25)\$
lock	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	Zo	-0.0010 (0.0004)	-0.0015 (0.0004)	-0.0012 (0.0004)	-0.0004 (0.0004)	-0.0009 (0.0004)	-0.0017 (0.0004)	-0.0012 (0.0004)	-0.0003 (0.0004)
lead	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	Zo	-0.002 (0.0008)	-0.0009 (0.0008)	-0.001 (0.0008)	0.00003 (0.0008)	-0.0008 (0.0008)	-0.0008 (0.0008)	-0.001 (0.0008)	0.001 (0.0008)
lag	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	Zo	-0.001 (0.001)	-0.002 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.002 (0.001)	-0.001 (0.001)	-0.001 (0.001)

Units for reported variables: EMG(V); SBP(mmHg); DBP(mmHg); CO(L/min); Zo(Ω)
 \$ Significant difference between tests in the same gender and frequency band
 § Significant difference between genders in the same test and frequency band

Table A-2: Transfer function gain between the signal pairs during phase lock (lead and lag) for post-hoc analyses of skeletal muscle pump interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
EMG-SBP	Lock	33.33 (6.51)	27.69 (6.51)	30.30 (6.51)£	38.09 (6.51)	33.09 (6.51)	20.74 (6.51)	6.84 (6.51)	10.14 (6.51)
	Lead	35.83 (7.35)	26.46 (7.35)	31.16 (7.35)	39.64 (7.35)	30.80 (7.35)	18.73 (7.35)	5.35 (7.35)	9.20 (7.35)
	Lag	30.96 (7.39)	32.77 (7.39)	28.38 (7.39)	38.24 (7.39)	40.67 (7.39)	25.95 (7.39)	16.07 (7.39)	13.04 (7.39)
EMG-DBP	Lock	26.06 (4.21)	19.34 (4.21)	26.55 (4.51)£	23.36 (4.21)	17.00 (4.21)	12.42 (4.21)	5.50 (4.51)	5.50 (4.21)
	Lead	26.54 (4.95)	22.40 (4.95)	23.96 (5.30)	18.92 (4.95)	14.77 (4.95)	13.18 (4.95)	5.39 (5.30)	3.84 (5.30)
	Lag	26.82 (5.15)	17.52 (5.15)	29.04 (5.52)	32.52 (5.15)	19.34 (5.15)	14.42 (5.15)	7.38 (5.52)	9.81 (5.15)
EMG-CO	Lock	2.11 (0.76)	1.49 (0.76)	2.02 (0.76)	2.07 (0.76)	3.42 (0.76)	1.54 (0.76)	1.10 (0.76)	1.08 (0.76)
	Lead	4.14 (0.90)	1.87 (0.84)	1.98 (0.84)	1.46 (0.84)	3.09 (0.84)	1.17 (0.84)	1.09 (0.84)	1.05 (0.84)
	Lag	2.44 (0.91)	1.56 (0.91)	2.39 (0.91)	3.03 (0.91)	3.46 (0.91)	2.29 (0.91)	1.34 (0.91)	1.32 (0.91)
EMG-Zo	Lock	0.072 (0.020)	0.066 (0.020)	0.059 (0.020)	0.128 (0.020)£	0.028 (0.020)	0.031 (0.020)	0.026 (0.020)	0.035 (0.020)
	Lead	0.086 (0.020)	0.076 (0.020)	0.055 (0.020)	0.115 (0.020)£	0.028 (0.020)	0.030 (0.020)	0.026 (0.020)	0.029 (0.020)
	Lag	0.066 (0.02)	0.061 (0.02)\$	0.066 (0.02)	0.139 (0.02)£	0.026 (0.02)	0.033 (0.02)	0.025 (0.02)	0.041 (0.02)

Units for reported variables: EMG-SBP(mmHg/V); EMG-DBP(mmHg/V); EMG-CO(L min⁻¹/V); EMG-Zo(Ω/V)
£ Significant difference between frequency bands in the same gender and test

Table A-3: Phase difference and average phase angle ($^{\circ}$) during phase lock (lead and lag) for post-hoc analyses of skeletal muscle pump interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
EMG-SBP	Diff	-3.14 (3.39)	1.63 (3.39)	-4.29 (3.39)	2.66 (3.39)	-1.00 (3.39)	-0.29 (3.39)	3.62 (3.39)	-1.08 (3.39)
	Lead	42.13 (2.94)£	41.53 (2.94)£	46.27 (2.94)£	46.73 (2.94)£	34.65 (2.94)	35.87 (2.94)	30.13 (2.94)	38.96 (2.94)
	Lag	-43.43 (2.58)	-41.65 (2.58)	-45.60 (2.58)£	-46.33 (2.58)£	-33.15 (2.58)	-33.05 (2.58)	-32.20 (2.58)	-32.37 (2.58)
EMG-DBP	Diff	-1.94 (3.73)	-2.30 (3.73)	-5.46 (3.98)	4.96 (3.73)	-4.43 (3.73)	2.67 (3.73)	1.29 (3.98)	0.77 (3.73)
	Lead	44.36 (3.01)£	44.11 (3.01)£	44.57 (3.23)	46.98 (3.01)	29.78 (3.01)	30.74 (3.01)	33.92 (3.23)	42.38 (3.01)
	Lag	-43.64 (2.95)	-45.38 (2.95)£	-48.92 (3.16)	-40.57 (2.95)	-34.61 (2.95)	-28.44 (2.95)	-38.23 (3.16)	-35.35 (2.95)
EMG-CO	Diff	-5.94 (4.09)	-4.83 (4.09)	-9.38 (4.09)	-5.74 (4.09)	-4.42 (4.09)	-3.77 (4.09)	-5.78 (4.09)	-4.89 (4.09)
	Lead	43.63 (3.15)£	41.12 (3.15)	41.08 (3.15)	44.16 (3.15)£	29.08 (3.15)	32.43 (3.15)	32.47 (3.15)	30.36 (3.15)
	Lag	-41.19 (4.07)	-39.60 (4.07)	52.20 (4.07)	-53.11 (4.07)	-31.31 (4.07)	-33.55 (4.07)	-38.36 (4.07)	-35.62 (4.07)
EMG-Zo	Diff	-13.28 (4.43)	-2.78 (4.43)	0.31 (4.43)	-3.58 (4.43)	-9.56 (4.43)	-6.44 (4.43)	-5.30 (4.43)	-2.65 (4.43)
	Lead	51.72 (2.68)£	55.11 (2.68)£	47.80 (2.68)£	50.55 (2.68)£	26.71 (2.68)	30.56 (2.68)	28.59 (2.68)	33.48 (2.68)
	Lag	-58.47 (3.00)£	-56.34 (3.00)£	-50.79 (3.00)£	-50.81 (3.00)£	-36.05 (3.00)	-35.50 (3.00)	-32.88 (3.00)	-35.85 (3.00)

£ Significant difference between frequency bands in the same gender and test

TableA- 4: Average coherence (au) above the threshold of significance for post-hoc analyses of skeletal muscle pump interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
EMG-SBP	0.333 (0.007) £	0.340 (0.007) £	0.360 (0.007) £	0.358 (0.007) £	0.404 (0.007)	0.396 (0.007)	0.418 (0.007)	0.410 (0.007)
EMG-DBP	0.351 (0.006) £	0.345 (0.006) £	0.354 (0.006) £	0.364 (0.006) £	0.409 (0.006)	0.403 (0.006)	0.410 (0.006)	0.415 (0.006)
EMG-CO	0.305 (0.007) \$£	0.318 (0.007) £	0.339 (0.007) £	0.345 (0.007) £	0.412 (0.007)	0.397 (0.007)	0.417 (0.007)	0.398 (0.007)
EMG-Zo	0.424 (0.013) \$£	0.395 (0.013)	0.364 (0.013)	0.389 (0.013)	0.468 (0.013) \$	0.434 (0.013)	0.413 (0.013)	0.442 (0.013)

\$ Significant difference between tests in the same gender and frequency band
£ Significant difference between frequency bands in the same gender and test

Table A-5: Percent time above the threshold of significant coherence for post-hoc analyses of skeletal muscle pump interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
EMG-SBP	67.66 (2.53) £	68.95 (2.53) £	73.28 (2.53) £	69.84 (2.53) £	49.03 (2.53)	46.20 (2.53)	44.37 (2.53)	39.51 (2.53)
EMG-DBP	69.63 (2.50)	69.25 (2.50)	72.07 (2.68)	72.04 (2.50)	45.40 (2.50)	46.64 (2.50)	40.93 (2.68)	41.19 (2.50)
EMG-Co	56.35 (2.94)	59.26 (2.94)	62.13 (2.94)	63.58 (2.94)	43.77 (2.94)	42.82 (2.94)	42.42 (2.94)	39.03 (2.94)
EMG-Zo	85.34 (3.57)	81.20 (3.57)	78.31 (3.57)	79.12 (3.57)	74.25 (3.57) \$	57.73 (3.57)	48.20 (3.57)	50.20 (3.57)

\$ Significant difference between tests in the same gender and frequency band
£ Significant difference between frequency bands in the same gender and test

Table A-6: Percent time signal pairs were phase locked and above significant coherence for post-hoc analyses of skeletal muscle pump interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
EMG-SBP	7.84 (0.92)	8.40 (0.92)	9.58 (0.92)	9.55 (0.92)	7.82 (0.92)	5.46 (0.92)	7.16 (0.92)	5.54 (0.92)
EMG-DBP	6.08 (0.91)	6.15 (0.91)	8.38 (0.98)	7.83 (0.91)	8.31 (0.91)	6.91 (0.91)	8.09 (0.98)	6.88 (0.91)
EMG-Co	1.34 (0.99)£	1.55 (0.99)£	2.32 (0.99)£	2.17 (0.99)£	9.41 (0.99)	9.08 (0.99)	11.90 (0.99)	9.21 (0.99)
EMG-Zo	17.09 (1.52)£	18.51 (1.52)	15.01 (1.52)	14.72 (1.52)	24.20 (1.52)\$	19.86 (1.52)	15.99 (1.52)	17.02 (1.52)
\$ Significant difference between tests in the same gender and frequency band £ Significant difference between frequency bands in the same gender and test								

Table A-7: Mean value of the transfer function variables when the signal pairs were phase locked (lead and lag) for post-hoc analyses of vascular interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
lock	Z ₀	-0.001 (0.001)	-0.0006 (0.001)	-0.0005 (0.001)	-0.001 (0.001)	-0.0006 (0.001)	0.0005 (0.001)	-0.0007 (0.001)	0.0004 (0.001)
	SBP	112(3)	115(3)	107(3)	111(3)	113(3)	114(3)	106(3)	111(3)
lead	Z ₀	-0.001 (0.001)	-0.0005 (0.001)	-0.0006 (0.001)	-0.0007 (0.001)	-0.0006 (0.001)	0.001 (0.001)	-0.00001 (0.001)	0.001 (0.001)
	SBP	112(3)	115(3)	107(3)	111(3)	113(3)	114(3)	106(3)	111(3)
lag	Z ₀	-0.001 (0.001)	-0.0004 (0.001)	-0.0003 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.003 (0.001)	-0.001 (0.001)
	SBP	112(3)	115(3)	107(3)	111(3)	112(3)	115(3)	106(3)	111(3)
lock	Z ₀	0.0001 (0.0008)	-0.001 (0.0008)	-0.001 (0.0008)	-0.001 (0.0008)	-0.0006 (0.0008)	-0.0006 (0.0008)	-0.001 (0.0008)	-0.0006 (0.0008)
	CO	4.73 (0.25)	6.09 (0.25)§	5.02 (0.25)	6.43 (0.25)§	4.71 (0.25)	6.12 (0.25)§	5.02 (0.25)	6.47 (0.25)§
lead	Z ₀	0.001 (0.001)	-0.0002 (0.001)	-0.002 (0.001)	-0.002 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.0007 (0.001)	-0.0008 (0.001)
	CO	4.73 (0.25)	6.11 (0.25)§	5.02 (0.25)	6.45 (0.25)§	4.65 (0.25)	6.13 (0.25)§	5.01 (0.25)	6.48 (0.25)§
lag	Z ₀	-0.001 (0.001)	-0.002 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.0004 (0.001)	-0.0002 (0.001)	-0.002 (0.001)	-0.001 (0.001)
	CO	4.74 (0.25)	6.09 (0.25)§	5.02 (0.25)	6.42 (0.25)§	4.75 (0.25)	6.11 (0.25)§	4.97 (0.25)	6.45 (0.25)§

Units for reported variables: Z₀ (Ω); SBP (mmHg); CO (L/min)
 § Significant difference between genders in the same test and frequency band

Table A-8: Transfer function gain between the signal pairs during phase lock (lead and lag) for post-hoc analyses of vascular interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
Zo-SBP	Lock	712.15 (236.82)	680.41 (236.82)	749.10 (236.82)	432.00 (236.82)	1346.62 (236.82)\$	994.67 (236.82)	567.87 (236.82)	379.95 (236.82)
	Lead	699.94 (222.30)	763.60 (222.30)	698.41 (222.30)	408.16 (222.30)	1258.69 (222.30)	951.65 (222.30)	633.59 (222.30)	433.49 (222.30)
	Lag	748.2 (280.9)£	681.0 (280.9)	854.7 (280.9)	471.0 (280.9)	1744.8 (280.9)	1121.3 (280.9)	663.2 (280.9)	293.0 (280.9)
Zo-CO	Lock	29.85 (11.77)\$	31.93 (11.77)	40.87 (11.77)	28.43 (11.77)	80.74 (11.77)	68.04 (11.77)	46.54 (11.77)	41.24 (11.77)
	Lead	32.01 (12.87)	33.56 (12.11)	36.17 (12.11)	26.00 (12.11)	80.57 (12.11)	62.45 (12.11)	65.70 (12.11)	54.28 (12.11)
	Lag	28.55 (14.33)£	30.95 (14.33)	42.77 (14.33)	31.85 (15.30)	103.56 (14.33)	72.11 (14.33)	47.61 (14.33)	39.56 (14.33)

Units for reported variables: Zo-SBP(mmHg/Ω); Zo-CO(L min⁻¹/Ω)
 \$ Significant difference between tests in the same gender and frequency band
 £ Significant difference between frequency bands in the same gender and test

Table A-9: Phase difference and average phase angle (°) during phase lock (lead and lag) for post-hoc analyses of vascular interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
Zo-SBP	Diff	-5.47 (4.63)	-4.03 (4.63)	0.29 (4.63)	1.92 (4.63)	2.20 (4.63)	6.67 (4.63)	5.63 (4.63)	1.82 (4.63)
	Lead	45.23 (2.57)	43.54 (2.57)	44.13 (2.57)	43.26 (2.57)	35.66 (2.57)	38.76 (2.57)	37.68 (2.57)	34.14 (2.57)
	Lag	-50.86 (2.88)£	-43.41 (2.88)	-47.35 (2.88)£	-43.35 (2.88)	-33.54 (2.88)	-34.66 (2.88)	-32.67 (2.88)	-36.35 (2.88)
Zo-CO	Diff	-1.15 (5.12)	-8.85 (5.12)	-6.68 (5.12)	-4.21 (5.12)	-10.02 (5.12)	-4.31 (5.12)	-0.31 (5.12)	-1.05 (5.12)
	Lead	38.10 (3.15)	33.55 (3.15)	40.83 (3.15)	37.48 (3.15)	30.45 (3.15)	30.29 (3.15)	39.27 (3.15)	30.47 (3.15)
	Lag	-40.29 (3.30)	-42.70 (3.30)	-42.59 (3.30)	-43.46 (3.30)	-37.90 (3.30)	-34.97 (3.30)	-32.90 (3.30)	-30.07 (3.30)

£ Significant difference between frequency bands in the same gender and test

Table A-10: Average coherence (au) above the threshold of significant coherence for post-hoc analyses of vascular interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
Zo-SBP	0.355 (0.008)£	0.343 (0.008)£	0.348 (0.008)£	0.353 (0.008)£	0.416 (0.008)	0.425 (0.008)	0.430 (0.008)	0.412 (0.008)
Zo-CO	0.340 (0.009)£	0.328 (0.009)£	0.354 (0.009)£	0.348 (0.009)£	0.430 (0.009)	0.421 (0.009)	0.434 (0.009)	0.415 (0.009)

£ Significant difference between frequency bands in the same gender and test

Table A-11: Percent time above the threshold of significant coherence for post-hoc analyses of vascular interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
Zo-SBP	72.73 (2.96)	69.22 (2.96)	75.72 (2.96)	70.01 (2.96)	51.02 (2.96)	50.66 (2.96)	60.79 (2.96)	45.34 (2.96)
Zo-Co	60.45 (4.17)	61.50 (4.17)	70.35 (4.17)	63.12 (4.17)	58.19 (4.17)	54.38 (4.17)	61.31 (4.17)	45.18 (4.17)

There were no significant differences for either interaction

Table A-12: Percent time signal pairs were phase locked and above significant coherence for post-hoc analyses of vascular interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
Zo-SBP	11.74 (1.41)	9.03 (1.41)	12.92 (1.41)	11.67 (1.41)	8.12 (1.41)	7.33 (1.41)	9.44 (1.41)	7.32 (1.41)
Zo-Co	2.27 (1.38)£	2.08 (1.68)£	4.90 (1.38)£	2.89 (1.38)£	14.93 (1.38)	12.02 (1.38)	15.26 (1.38)	9.92 (1.38)

£ Significant difference between frequency bands in the same gender and test

Table A-13: Mean value of the transfer function variables when the signal pairs were phase locked (lead and lag) for post-hoc analyses of COP interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
lock	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.003 (0.0003)
	COPx	-0.0008 (0.002)	-0.0009 (0.002)	-0.0013 (0.002)	0.0025 (0.002)	-0.0009 (0.002)	-0.0009 (0.002)	-0.0013 (0.002)	0.0023 (0.002)
lead	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	COPx	-0.0009 (0.002)	-0.0009 (0.002)	-0.001 (0.002)	0.002 (0.002)	-0.0007 (0.002)	-0.0007 (0.002)	-0.001 (0.002)	0.002 (0.002)
lag	EMG	0.002 (0.0003)	0.003 (0.0003)	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	COPx	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)	0.003 (0.002)	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)	0.002 (0.002)
lock	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	COPy	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.037 (0.005)
lead	EMG	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	COPy	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.037 (0.005)
lag	EMG	0.002 (0.0003)	0.003 (0.0003)	0.004 (0.0003)	0.004 (0.0003)	0.002 (0.0003)\$	0.003 (0.0003)\$	0.004 (0.0003)	0.004 (0.0003)
	COPy	-0.26 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.037 (0.005)
lock	Zo	-0.001 (0.0004)	-0.001 (0.0004)	-0.001 (0.0004)	-0.001 (0.0004)	-0.001 (0.0004)	-0.0007 (0.0004)	-0.0008 (0.0004)	-0.002 (0.0004)
	COPx	-0.0008 (0.002)	-0.0008 (0.002)	-0.001 (0.002)	0.003 (0.002)	-0.0008 (0.002)	-0.001 (0.002)	-0.001 (0.002)	0.002 (0.002)
lead	Zo	-0.001 (0.0007)	-0.001 (0.0007)	-0.002 (0.0007)	-0.001 (0.0007)	-0.001 (0.0007)	-0.001 (0.0007)	-0.001 (0.0007)	-0.002 (0.0007)
	COPx	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)	0.002 (0.002)	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)	0.002 (0.002)
lag	Zo	-0.001 (0.001)	-0.002 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.0001 (0.001)	-0.001 (0.001)
	COPx	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)	0.003 (0.001)	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)	0.002 (0.002)
lock	Zo	-0.001 (0.0005)	-0.0007 (0.0005)	-0.001 (0.0005)	-0.001 (0.0005)	-0.0008 (0.0005)	-0.0009 (0.0005)	-0.001 (0.0005)	-0.002 (0.0005)
	COPy	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)
lead	Zo	-0.001 (0.0009)	-0.001 (0.0009)	-0.001 (0.0009)	-0.001 (0.0009)	-0.001 (0.0009)	-0.001 (0.0009)	-0.001 (0.0009)	-0.002 (0.0009)
	COPy	-0.027 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)	-0.027 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)
lag	Zo	-0.001 (0.001)	-0.0002 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.004 (0.001)	-0.003 (0.001)
	COPy	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.038 (0.005)	-0.026 (0.005)	-0.034 (0.005)	-0.031 (0.005)	-0.037 (0.005)

Units for reported variables: EMG (V); COP(m); Zo(Ω)
 \$ Significant difference between tests in the same gender and frequency band

Table A-14: Transfer function gain between the signal pairs during phase lock (lead and lag) for post-hoc analyses for COP interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
EMG-COPx	Lock	0.011 (0.004)	0.014 (0.004)	0.010 (0.004)	0.018 (0.004)	0.014 (0.004)	0.011 (0.004)	0.005 (0.004)	0.010 (0.004)
	Lead	0.010 (0.004)	0.013 (0.004)	0.010 (0.004)	0.016 (0.004)	0.018 (0.004)\$	0.012 (0.004)	0.005 (0.004)	0.009 (0.004)
	Lag	0.011 (0.005)	0.017 (0.005)	0.010 (0.005)	0.021 (0.005)	0.012 (0.005)	0.011 (0.005)	0.006 (0.005)	0.018 (0.005)
EMG-COPy	Lock	0.025 (0.004)	0.015 (0.004)	0.019 (0.004)	0.025 (0.004)£	0.026 (0.004)\$	0.018 (0.004)	0.009 (0.004)	0.011 (0.004)
	Lead	0.023 (0.005)	0.018 (0.005)	0.018 (0.005)	0.029 (0.005)£	0.025 (0.005)	0.018 (0.005)	0.011 (0.005)	0.012 (0.005)
	Lag	0.030 (0.005)	0.012 (0.005)	0.019 (0.005)	0.022 (0.005)	0.025 (0.005)\$	0.018 (0.005)	0.007 (0.005)	0.012 (0.005)
Zo-COPx	Lock	7.71 (2.22)	5.17 (2.22)	7.68 (2.22)	8.80 (2.22)	2.85 (2.22)	3.42 (2.22)	6.27 (2.22)	7.87 (2.22)
	Lead	7.22 (2.98)	5.40 (2.98)	7.63 (2.98)	11.93 (2.98)	3.02 (2.98)	3.60 (2.98)	5.50 (2.98)	9.00 (2.98)
	Lag	8.04 (1.46)	4.88 (1.46)	8.03 (1.46)	6.51 (1.46)	2.91 (1.46)	3.67 (1.46)	2.91 (1.46)	3.28 (1.46)
Zo-COPy	Lock	2.69 (1.50)	3.61 (1.50)	3.70 (1.50)	7.41 (1.50)	1.21 (1.50)	2.26 (1.50)	3.48 (1.50)	5.45 (1.50)
	Lead	3.13 (1.60)	3.72 (1.60)	3.80 (1.60)	7.31 (1.60)	1.17 (1.60)	2.58 (1.60)	3.41 (1.60)	5.86 (1.60)
	Lag	2.44 (1.30)	3.58 (1.30)	3.75 (1.30)	7.39 (1.30)	1.33 (1.30)	2.02 (1.30)	1.33 (1.30)	3.38 (1.30)

Units for reported variables: EMG-COPx(m² s/V); EMG-COPy(m² s/V); Zo-COPx(Ω/ m² s); Zo-COPy(Ω/ m² s)
 \$ Significant difference between tests in the same gender and frequency band
 £ Significant difference between frequency bands in the same gender and test

Table A-15: Phase difference and average phase angle (°) during phase lock (lead and lag) for post-hoc analyses of COP interactions reported as least square mean (SEM).

		LF				VLF			
		Pre		Post		Pre		Post	
		F	M	F	M	F	M	F	M
EMG-COPx	Diff	-5.15 (3.59)£	-5.13 (3.59)	-3.54 (3.59)	-1.32 (3.59)	-0.46 (3.59)	9.29 (3.59)	1.95 (3.59)	3.90 (3.59)
	Lead	49.73 (2.51)£	44.46 (2.51)	49.19 (2.51)£	45.20 (2.51)£	30.78 (2.51)	35.4 9(2.51)	30.98 (2.51)	33.81 (2.51)
	Lag	-53.52 (2.26)£	-49.04 (2.26)£	-46.61 (2.26)£	-46.01 (2.26)£	-32.99 (2.26)	-26.84 (2.26)	-32.64 (2.26)	-29.95 (2.26)
EMG-COPy	Diff	55.25 (4.00)\$	29.54 (4.00)	14.21 (4.00)	18.57 (4.00)	27.81 (4.00)\$	11.47 (4.00)	10.32 (4.00)	10.23 (4.00)
	Lead	65.76 (1.93)£	51.68 (1.93)£	49.26 (1.93)£	50.48 (1.93)£	33.52 (1.93)	31.47 (1.93)	29.45 (1.93)	34.09 (1.93)
	Lag	-28.82 (2.36)	-35.52 (2.36)	-44.00 (2.36)	-41.17 (2.36)	-11.74 (2.36)	-23.45 (2.36)	-26.29 (2.36)	-26.87 (2.36)
Zo-COPx	Diff	-8.69 (4.79)	-6.69 (4.79)	-2.04 (4.79)	2.20 (4.79)	3.60 (4.79)	1.90 (4.79)	6.55 (4.79)	7.85 (4.79)
	Lead	49.93 (3.09)	45.73 (3.09)	51.06 (3.09)	48.81 (3.09)	39.31 (3.09)	37.88 (3.09)	40.08 (3.09)	40.46 (3.09)
	Lag	-54.47 (3.04)£	-48.90 (3.04)£	-54.92 (3.04)£	-49.66 (3.04)£	-38.08 (3.04)	-31.75 (3.04)	-33.11 (3.04)	-31.84 (3.04)
Zo-COPy	Diff	-38.33 (8.32)£	-39.15 (8.32)£	-30.05 (8.32)£	-29.19 (8.32)£	2.16 (8.32)	7.68 (8.32)	25.69 (8.32)	8.92 (8.32)
	Lead	37.35 (3.70)	39.15 (3.70)	29.35 (3.70)	38.96 (3.70)	36.70 (3.70)	36.99 (3.70)	39.64 (3.70)	38.02 (3.70)
	Lag	-66.49 (6.32)£	-65.09 (6.32)£	-49.14 (6.32)£	-56.40 (6.32)£	-40.25 (6.32)	-31.60 (6.32)	-22.72 (6.32)	-33.30 (6.32)
\$ Significant difference between tests in the same gender and frequency band £ Significant difference between frequency bands in the same gender and test									

Table A-16: Average coherence (au) when above the threshold of significance for post-hoc analyses of COP interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
EMG-COPx	0.368 (0.008)£	0.359 (0.008)£	0.355 (0.008)£	0.370 (0.008)£	0.416 (0.008)	0.422 (0.008)	0.401 (0.008)	0.416 (0.008)
EMG-COPy	0.540 (0.020)\$	0.443 (0.020)	0.405 (0.020)	0.405 (0.020)	0.612 (0.020)\$	0.467 (0.020)	0.444 (0.020)	0.434 (0.020)
Z ₀ -COPx	0.384 (0.012)£	0.393 (0.012)£	0.395 (0.012)£	0.401 (0.012)£	0.436 (0.012)	0.460 (0.012)	0.439 (0.012)	0.449 (0.012)
Z ₀ -COPy	0.527 (0.030)	0.516 (0.030)	0.511 (0.030)£	0.503 (0.030)	0.558 (0.030)	0.623 (0.030)	0.499 (0.030)	0.499 (0.030)

£ Significant difference between frequency bands in the same gender and test

Table A-17: Percent time above the threshold of significant coherence for post-hoc analyses of COP interactions reported as least square mean (SEM).

	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
EMG-COPx	74.48 (2.66)	75.16 (2.66)	72.68 (2.66)	76.88 (2.66)	52.42 (2.66)	53.39 (2.66)	48.01 (2.66)	45.71 (2.66)
EMG-COPy	94.80 (3.09)\$	85.51 (3.09)	82.80 (3.09)	81.68 (3.09)	92.34 (3.09)\$	68.34 (3.09)	50.64 (3.09)	57.56 (3.09)
Z ₀ -COPx	79.04 (3.43)	82.88 (3.43)	80.69 (3.43)	82.58 (3.43)	67.27 (3.43)	67.30 (3.43)	70.12 (3.43)	65.62 (3.43)
Z ₀ -COPy	92.89 (3.02)	92.65 (3.02)£	93.65 (3.02)	90.58 (3.02)	90.79 (3.02)	93.32 (3.02)	80.63 (3.02)\$	72.85 (3.02)

\$ Significant difference between tests in the same gender and frequency band
£ Significant difference between frequency bands in the same gender and test
§ Significant difference between genders in the same test and frequency band

Table A-18: Percent time signal pairs were phase locked and above significant coherence for post-hoc analyses of COP interactions reported as least square mean (SEM).

% Time Coh + Ph lock for COP interactions								
	LF				VLF			
	Pre		Post		Pre		Post	
	F	M	F	M	F	M	F	M
EMG-COPx	11.01 (1.118)	12.22 (1.18)	10.76 (1.18)	12.32 (1.18)	15.59 (1.18)	15.02 (1.18)	14.11 (1.18)	14.83 (1.18)
EMG-COPy	14.94 (1.08)£	14.75 (1.08)£	14.59 (1.08)£	14.02 (1.08)£	24.87 (1.08)	21.23 (1.08)	16.05 (1.08)	18.53 (1.08)
Z ₀ -COPx	13.76 (1.77)£	14.36 (1.77)	16.44 (1.77)	14.38 (1.77)	21.68 (1.77)	21.36 (1.77)	21.11 (1.77)	18.45 (1.77)
Z ₀ -COPy	20.00 (1.65)£	18.71 (1.65)£	19.51 (1.65)£	17.40 (1.65)£	29.19 (1.65)	26.64 (1.65)	28.59 (1.65)	25.44 (1.65)

£ Significant difference between frequency bands in the same gender and test

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