EFFECTS OF SUPINE AND INVERTED SEATED POSITIONS ON NEUROMUSCULAR AND CARDIOVASCULAR PARAMETERS

by

ERICA JOCELYN PAUL CASEY

A thesis submitted to the

School of Graduate Studies

in partial fulfillment of the

requirements for the degree of

Master of Science (Kinesiology)

School of Human Kinetics and Recreation

Memorial University of Newfoundland

October, 2014

St. John's, Newfoundland and Labrador

Abstract

Purpose: The inverted and supine body positions are not common postures humans adopt. During unusual situations (e.g. overturned helicopters, motor vehicle accidents, gymnastics) when body position is altered, neuromuscular and cardiovascular (CV) responses can change. To optimally manage these situations, it is necessary to examine the changes in muscle force output, activation and CV parameters. In previous research, impairments during posture change were believed to be secondary to inhibition of sympathetic nervous system activity in recreational subjects. The purpose of this research was to evaluate the neuromuscular and CV changes in supine and inverted body positions on a group of aerobically trained athletes before and following physical fatigue.
Methods: Twelve male athletes completed three trials in the upright, supine and inverted seated positions. At baseline (upright), leg extension (LE) and elbow flexion (EF) evoked contractile properties and MVCs were performed. The participant was then positioned for 150s in each posture, followed by a 30s MVC (MVC30). During each trial, stroke volume (SV), cardiac output (Q), heart rate (HR), time and frequency domain HR variability measures and mean arterial blood pressure (MAP) measurements were recorded. As well, force-fatigue and EMG relationships were evaluated. **Results: An** ANOVA showed no statistical differences in EF MVC force, but a tendency ($p=0.12$) for LE MVC decline across positions with moderate effect size. EF evoked resting twitch ($p=0.1$) had a decline in force that was not significant from upright to inverted and no change from upright to supine. Potentiated peak twitch ($p=0.06$) force displayed a tendency towards an increase from upright to supine and decline in force from upright to inverted postures ($p=0.1$). LE

evoked resting twitch had a non-significant ($p=0.1$) but large magnitude increase in force across the three positions and there was a significant increase in potentiated twitch force ($p=0.03$). Force-fatigue, electromyography-fatigue relationships and HR variability during MVC30 fatigue across positions were not affected. HR and Q were significantly ($p<0.01$) lower with inversion following both LE and EF fatigue. **Conclusions:** The lack of significant postural changes in resting force and CV measures may demonstrate that highly trained individuals may adapt better to the supine and inverted positions.

KEYWORDS: cardiac output, heart rate, EMG, fatigue, force, inversion, supine, tilt

ACKNOWLEDGEMENTS

First and foremost, I would like to sincerely thank Dr. David Behm. This project, from its inception, has taken me a very long time to complete. You have consistently, over the years, been supportive of me finishing my thesis. You have undoubtedly been the most influential person in ensuring that I achieved this, and for that, I am truly grateful. I am fortunate to have been able to work under your supervision. A big thanks to my fellow graduate students who also supported me throughout this process, Kevin Parfrey, Geoff Power, Grant Handrigan, Deanne Smith and Liz Coady.

Thanks to Dr. Patrick Neary and Danielle Salmon for their work on this project, as well, Dr. Fabien Basset for his time and energy during the two years completing my academics for graduate school at Memorial University. I would also like to thank my husband, Andrew Casey, for supporting me to see this project through to completion.

Finally, a special thanks to all the subjects who volunteered their time to be a part of this research project because without them, we would not have been able to acquire this scientific knowledge. Lastly, thanks to all those whose names are not mentioned here, for your kind works of encouragement seeing me through completing this project and supporting me over the years.

Funding for this project was provided by the Natural Sciences and Engineering Research Council (NSERC) of Canada.

TABLE OF CONTENTS

List of Figures

List of Tables

List of Abbreviations

ANOVA: analysis of variance

bpm: beats per minute

CV: cardiovascular

EF: elbow flexion

EMG: electromyography

ES: effect sizes

FFT: Fast Fourier transform

HDT: head down tilt

HDBT: head down body tilt

HF: high frequency

HR: heart rate

Hz: Hertz

iEMG: integrated electromyography

LBNP: lower body negative pressure

LBPP: lower body positive pressure

LE: leg extension

LF: low frequency

MAP: mean arterial pressure

MSNA: muscle sympathetic nervous activity

MVC: maximum voluntary contraction

MVC30: 30 second isometric maximum voluntary contraction fatiguing protocol

N: Newton

NTS: solitary tract

Par-Q: Physical Activity Readiness Questionnaire

Q: cardiac output

SPSS: statistical package for the social sciences

SV: stroke volume

VO_2: alveolar oxygen uptake

VLF: very low frequency

VSR: vestibulosympathetic reflex

Co-Authorship Statement

E. Casey was involved in study development, collection of data, analysis of results and preparation of the manuscript

D.G. Behm developed the study, assisted in data collection, results analysis and writing the manuscript

J.P Neary collected the cardiovascular data, analyzed this component of the results and assisted in preparation of this component of the manuscript

D.M. Salmon was involved in collection of the data and analysis of the cardiovascular results

1. Introduction

1.1 Overview of the Thesis

This thesis entitled “Effects of supine and inverted seated positions on neuromuscular and cardiovascular parameters” is presented in manuscript format.

Chapter 2 includes a review of the literature on the effects of change in body positions on cardiovascular (CV) and neuromuscular parameters. The review examines oxygen kinetics, hydrostatic pressure, neuromuscular parameters, the vestibular system, baroreceptor function and respiratory changes and attempts to integrate these factors with adaptations of aerobically trained individuals.

Chapter 3 reports the findings of the research study of the effects of supine and inverted body positions on voluntary contractile properties, evoked contractile properties, fatigue-force relationship, fatigue-electromyography relationship and CV parameters including stroke volume, heart rate, cardiac output and mean arterial blood pressure. It also discusses limitations of this research and concluding remarks.

1.2 Background of Study

To date, there has been a relatively small body of research published on the effects of complete body inversion. Investigation and new knowledge into this area could be of use in numerous fields. On a daily basis, humans rarely subject themselves to situations whereby they are in an inverted or supine body position whilst exerting physical force. There are however, situations, ranging from recreational pursuits to professional ventures where people may find themselves in a variety of body positions. Examples of these situations include motor vehicle accidents, flight accidents and simulations, first responder situations, rollover in a kayak, mountaineering, rock climbing, gymnastics and air sport activities such as zip lining, paragliding and hang gliding. In each of these scenarios humans may be required to perform forceful and coordinated maneuvers whilst not in the upright position, where our body is most accustomed to performing. Operational efficiency and more importantly, at times, survival is dependent upon the ability to complete tasks under these difficult conditions. These individuals may be in situations where the body is taxed both physically and psychologically, and they are forced to perform at their optimum.

We maintain homeostasis through a complex network of efferent and afferent signals within the sensorimotor system. Changes in heart rate (HR) (Schneider and Chandler, 1973), the vestibulosympathetic reflex (VSR) response and hydrostatic pressure are central factors thought to be involved in postural adjustments, while peripheral factors include changes in blood pressure (BP) (Bosone et al. 2004), total peripheral resistance

(Goodman and LeSage, 2002) and peripheral perfusion (Thomas and Segal, 2004).

There is a small body of research that has been done on inversion in recreationally active individuals. These results consistently demonstrate that there are physiologic responses associated with body inversion that result in a reduction in maximal voluntary contraction (MVC) force, electromyography (EMG) activity, HR and BP (Behm and Paddock, 1999; Hearn, Cahill and Behm, 1999 and Johar et al. 2013) both in the upper and lower extremities and under a variety of circumstances. It has been hypothesized that this may alter sympathetic nervous system stimulation.

It has been proven that physically trained individuals have a larger cardiovascular (CV) reserve, biochemical adaptations, differences in neuromuscular function (Kenney, Wilmore and Castill, 2012) and reduced baseline sympathetic nervous system activity (Iellamo et al., 2000; Niemela et al., 2008; Pagani et al., 1988; Somers et al., 1991). It is logical to assume that many of the population who find themselves in these positions will be aerobically trained and physically fit (e.g.. fighter pilot, military personnel, athletes); it can be hypothesized that these adaptations may translate into less stress on the body during changes in posture and position in this population.

1.3 Purpose of Study

Whereas the implications of the previous research can be associated with athletic performance and the ability to escape from simulated life threatening situations,

examining the impact of inversion can also reveal basic physiological responses. Since only three studies have investigated the responses to full seated inversion, further work is necessary to elucidate a greater range of physiological responses. Are the previously reported reductions in HR and BP related to changes in stroke volume (SV), cardiac output (Q), heart rate variability, and mean arterial pressure (MAP)? Are these parameters changed to a greater or lesser degree such as when muscle fatigue is combined with whole-body inversion?

The purpose of this study was to evaluate neuromuscular and CV responses in the upper and lower extremities prior and following physical fatigue in the supine and inverted positions in trained individuals. If these subjects already have training-induced changes in sympathetic activity, can their response to change in body posture differ from that of the recreational population? To evaluate this, each subject was brought through a series of tests to evaluate neuromuscular function and a fatigue protocol whilst simultaneously measuring HR, Q, SV and MAP.

1.4 Significance of Study

A change in body postures places unfamiliar stresses on both the neuromuscular and CV systems. Results from this study should provide information regarding the possibility of reduced capabilities of the aforementioned individuals when faced with challenges of performance in an inverted position. An example of this could be an inverted helicopter in

water, another could be a military flight pilot while actively performing his or her job. This could further extend into sports such as hang gliding, ziplining, mountaineering, gymnastics and kayaking. Finally, the laboratory testing can illustrate the mechanisms contributing to inversion impairments. Based on previous research (Behm and Paddock, 1999; Hearn, Cahill and Behm, 1999 and Johar et al. 2013) it was hypothesized that an inverted seated position would decrease muscle force, activation and accelerate fatigue. Furthermore, if an inverted position inhibits sympathetic stimulation then it was hypothesized that CV measures such as the time and frequency domains of heart variability would be affected.

1.5 References

Goodman, L.S. and LeSage, S. (2002). Impairment of cardiovascular and vasomotor responses during tilt table simulation of 'push-pull' maneuvers. *Aviation Space and Environmental Medicine,* 73, 971-979.

Hearn J., Cahill F., and Behm D.G. (2009). An inverted seated posture decreases elbow flexion force and muscle activation. *European Journal of Applied Physiology,* 106, 139-147.

Iellamo, F., Legramante, J.M., Massaro, M., Raimondi, G. and Galante, A. (2000). Effects of a residential exercise training on baroreflex sensitivity and heart rate variability in patients with coronary artery disease: a randomized, controlled study. *Circulation,* 102, 2588–2592.

Johar P., Grover V., Disanto M.C., Button D.C. and Behm D.G. (2013). A rapid rotation to an inverted seated posture inhibits muscle force, activation, heart rate and blood pressure. *European Journal of Applied Physiology,* 113, 2005-2013.

Kenney, W.L., Milmore, J.H. and Costill, D.L. (2012). *Physiology of Sport and Exercise (5th Edition).* USA: Human Kinetics.

Niemela, T.H., Kiviniemi, A.M., Hautala, A.J., Salmi, J.A., Linnamo, V. and Tulppo, M.P. (2008). Recovery pattern of baroreflex sensitivity after exercise. *Medicine and Science in Sports and Exercise,* 40, 864–870.

Pagani, M., Somers, V., Furlan, R., Dell'Orto, S., Conway, J., Baselli, G., Cerutti, S., Sleight, P. and Malliana, A. (1988). Changes in autonomic regulation induced by physical training in mild hypertension. *Hypertension,* 12, 600-610.

Paddock N. and Behm D.G. (2009). The effect of an inverted body position on lower limb muscle force and activation. *Applied Physiology Nutrition and Metabolism,* 34**,** 673-680.

Schneider, M.F. and Chandler, W.K. (1973).Voltage dependent charge movement of skeletal muscle: a possible step in excitation-contraction coupling. *Nature,* 242, 244-246.

Somers, V.K., Conway, J., Johnston, J. and Sleight, P. (1991). Effects of endurance training on baroreflex sensitivity and blood pressure in borderline hypertension. *Lancet* 337, 1363–1368.

Thomas, G.D. and Segal, S.S. (2004). Neural control of muscle blood flow during exercise. *Journal of Applied Physiology,* 97, 731–738.

2. Review of Literature
2.1 Introduction

On a daily basis, humans rarely subject themselves to situations whereby they are exerting themselves in an inverted or supine position. There are however, scenarios, ranging from recreational pursuits, to professional ventures where people may find themselves in inverted or supine postures. Examples of these situations include motor vehicle accidents, flight accidents and simulations. Occupations or recreational pursuits that are associated with sudden changes in body position include first responders, gymnasts, zip liners mountaineers, hang gliders and paragliders, among others. These individuals may find themselves in situations where they are required to respond either voluntarily or involuntarily with forceful, coordinated movements whilst not in the upright position, where the body is most accustomed to performing. The purpose of this research is to examine the effect that these positions have on the neuromuscular and cardiovascular (CV) response in humans.

Postural control is a complicated process that combines peripheral and central processing factors (Ivanenko et al. 2000). Humans maintain homeostasis through a complex network and efferent and afferent signals within the sensorimotor system, with constant feedback from somatosensory, vestibular and visual inputs (Kollmitzer et al. 2000) accompanied by consistent anticipatory postural adjustments (Slijper and Latash, 2000).

Efferent output is activated during adjustments in posture to produce and maintain muscle

contraction and provide autonomic outflow to contracting muscles and the brain. Input from the brain's central command acts in a feed-forward manner and has an effect on both autonomic and somatic outflow. In contrast, input from somatic and visceral afferents act in a feedback manner, such that decreases in blood volume, pressure and muscle contractions result in adjustments in sympathetic nervous system output (Kerman, McAllen and Yates, 2000).

An afferent system that is activated during changes in posture and movement is the vestibular system. Changes in physical orientation of the body in space are quickly and accurately detected by vestibular input. The vestibular system has been investigated in the literature and proven to assist in autonomic adjustments required in postural changes. This is in an effort to maintain body homeostasis to ensure that blood flow is adequately distributed throughout the body (Kerman, McAllen and Yates, 2000). The adequate distribution of blood flow would have implications on the ability to exert force, power and counteract fatigue with changes in posture, such as the supine and inverted positions.

Head-down tilt (HDT), synonymously referred to as head-down body tilt (HDBT) is a method often used in research to simulate weightlessness. It is applicable to research on changes in body orientation, as the participant is placed in a position whereby the feet are higher than the head by a number of degrees. This is comparable to the supine and inverted body positions, as there is little published research on total body inversion, and the most similar protocols to inversion in the literature utilized HDBT. In this study protocol, the feet and head are parallel when in the supine posture, and the feet are higher

than the head whilst inverted.

HDT or HDBT studies have evaluated the effects of changes in body posture on outcomes in the cardiovascular (CV) system, such as heart rate (HR) (Schneider and Chandler, 1973), blood pressure (BP) (Bosone et al. 2004), total peripheral resistance (Goodman and LeSage, 2002) and peripheral perfusion (Thomas and Segal, 2004). Each of these changes is believed to be secondary to inhibition of sympathetic activity during change in body position (Bosone et al. 2004; Cooke and Dowlyn, 2000; Cooke et al. 2004; Fu et al. 2000).

Related to this, oxygen kinetics and blood flow have also been examined during changes in posture and body positioning with varying intensities of exercise. It has been demonstrated that there are differences in the rate of alveolar oxygen uptake, perfusion and blood flow distribution (Cerretelli et al. 1977; Convertino, Goldwater and Sandler, 1984 and Hughson, Cochrane and Butler, 1993), time to task failure (Rochette et al. 2003) and changes in endurance and fatigue (Egnana and Green, 2004) with changes in body orientation.

This review will investigate reported literature and elaborate on research related to body posture. It will evaluate the effects on oxygen uptake and distribution, the effects of changes in hydrostatic pressure, neuromuscular changes, changes in the vestibular and sympathetic nervous systems, baroreceptor function and respiratory changes. It will then attempt to link these changes and apply this to CV measures and force generation, power

and fatigue in the supine and inverted positions. This review will also discuss the sympathetic adaptations of trained athletes and the implications that these adaptations may have on their ability to tolerate supine and inverted postures as compared to the recreational population.

2.2 Oxygen uptake kinetics and blood flow distribution during postural changes

Oxygen uptake kinetics and blood flow have been examined during changes in posture and positioning. It has been demonstrated that there are significant changes in alveolar oxygen uptake, perfusion and blood flow distribution (Cerretelli et al. 1977; Convertino, Goldwater and Sandler, 1984 and Hughson, Cochrane and Butler, 1993), time to task failure (Rochette et al. 2003) and changes in endurance and fatigue (Egana and Green, 2004) with changes in body posture.

In an effort to achieve homeostasis and mean arterial blood pressure (MAP), the peripheral muscles vasoconstrict (Wallin and Sundlof, 1982). There is no net gravitational effect on circulating blood flow in the supine position as arterial and venous reservoirs are in the same plane when the body is supine. Secondary to this, during a supine body position, peripheral vasoconstriction, venoconstriction and vasoactive factors (e.g. epinephrine, angiotensin-II, vasopressin, nitric oxide) are also at a resting state (Stewart and Clarke, 2011). Blood flow and distribution, hence, oxygen delivery are directly affected by this, and oxygenation of the muscular system directly affects the fatigability of the muscular system. This, in turn, affects the ability to exert force and

power while in the supine position.

Ceretelli et al. (1977) demonstrated that during submaximal arm cranking and leg peddling in the supine position, alveolar oxygen uptake (VO_2) is adjusted and slows when compared with equivocal work in the upright position. This has been further supported in the literature. Convertino, Goldwater and Sandler (1984) evaluated constant load exercise in the supine and upright positions. Their research found that steady state VO_2 was similar across both protocols and total VO_2 was greater in the upright position while oxygen deficit and recovery VO_2 was decreased in the supine position. Due to this effect, supine exercise was concluded to result in a reduction in VO_2 capacity and increased oxygen deficit.

MacDonald et al. (1998) studied blood flow delivery at the onset of large muscle mass exercise in supine and upright positions. They found that MAP and HR decreased during exercise in the supine position. There was a 35% VO_2 reduction and 60% leg blood flow reduction in supine versus upright positions. They hypothesized from this that a reduced perfusion pressure in the supine body position was responsible for slower increase in leg blood flow and subsequent oxygen delivery and suggested that alterations in metabolic control by availability of oxygen at commencement of exercise in supine body position limits increase in oxygen uptake.

Fitzpatrick, Taylor and McCloskey (1994) reported that fatigue and endurance are both affected by the limb's orientation in space. It was speculated that changes in perfusion

pressure acting on the skeletal muscle elicited these responses. Koga et al. (1999) concurred with this, demonstrating that despite increased cardiac output (Q) in the supine position, blood flow availability to the working muscles was decreased. Supine posture subsequently had significant reductions in peak work rate, peak oxygen uptake, estimated lactate threshold and peak HR when compared with upright posture kinetics during incremental exercise.

Regarding complete inversion, there have been several studies conducted to evaluate the effect of inversion as compared to upright posture on CV outcomes. Hearn, Cahill and Behm (2009) and Paddock and Behm (2009) both demonstrated a consistent reduction in HR and BP in the inverted posture, indicating that central outcomes decreased in the inverted position. Johar et al. (2013) also demonstrated that irrespective of rotation speed, there is inhibition of CV responses in the inverted posture. Neary et al. (2011b) published an abstract reporting that there was a significant decrease in HR and Q with complete seated inversion. Research in this field has also been conducted on the effect of hemodynamics and anti-orthostasis. An anti-orthostasic posturing study (Balueva and Sergeev, 2010), found a reduction in BP and Q with 45 degrees of HDT.

Raffai et al. (2009) and Egana and Green (2005) provided some evidence to the contrary. The former research found that 45 degrees HDT resulted in an increase in BP in rats over 7 days. Egana and Green (2005) explored the effects of postural changes and peripheral circulation by studying the effects of body tilt on calf muscle strength, fatigue and endurance in a head up position in humans. The research (2005) investigated the body's

response to the horizontal position via three experiments. The first tested body tilt in three different angles on muscle strength and graded exercise to the point of failure; the second examined the effects of body tilt on muscle fatigue and endurance during constant exercise to the point of failure in the horizontal plane and three differing inclined positions; the third evaluated the effects of body tilt angle on leg blood flow at two different intensities. Results found that strength was not significantly affected by tilt angle in this study. Time to task failure during the second and third experiments were higher during tilt. Time to failure was increased by up to 100% and there was a decrease in the rate of fatigue by >50% when tilt angle was increased from the horizontal. Intensity was close to maximum VO_2 in these studies, and therefore, leg blood flow in the supine position would be at a resistance that is highly dependent upon blood flow to the area. They found overall, that endurance of the calf muscle improved as the body was tilted from the horizontal to inclined position, dependent on intact peripheral circulation and mediated by blood flow to the muscle, with no effect on strength. Based on these findings, there was a tilt induced increase in fatigue resistance and reduction of the fatigue effect. The authors suggest that these data support that the range of tilt angles and their effects on muscular performance may vary widely, and warrants further research. It is also important to consider, when applying these results to the current study on supine and inverted postures, that this study was performed during head-up tilt. The current research study is with HDT in the supine and inverted position.

There are several hypotheses in the literature to explain the gravity-induced changes in arterial pressure observed in the aforementioned studies. The first suggestion is that

perfusion during exercise is increased during body tilting and attributed to the muscle pump (Folkow et al. 1971). It has been suggested that during an inclined body position, hydrostatic pressure, acting on the arterial and venous peripheral system at rest is removed from the venous system during relaxation. In the supine position, we would not observe this increase in perfusion pressure; the key concept of this mechanism is that any change in venous pressure directly affects perfusion pressure (Laughlin and Schrage, 1999).

The second proposed mechanism behind the gravity induced change in pressure in the upright position describes collapsible arteriolar blood vessel flow and is coined the 'vascular waterfall' model of circulation (Permutt and Riley, 1963). It states that downstream pressure affecting perfusion pressure is influenced by vascular tone; there is a reduction in perfusion pressure in the supine position with a change in local vascular regulatory factors (MacDonald et al. 1998). Force output is directly affected by blood flow in the contracting muscles (Hogan, Richardson and Kurdak, 1994), and subsequently, muscle fatigue during submaximal and moderately intense work is sensitive to changes in blood flow to the working muscle.

Although there are conflicting findings in the above studies regarding the effect of body posture on oxygen availability and blood flow to working muscles, and it is important to consider that our research is performed during HDT in supine and inverted positions. We speculate that there will be a reduction in blood perfusion to the active limbs as blood is shunted centrally with posture adjustments. The remainder of this chapter will elaborate

on other integral factors in this process and our research will help explore this further.

2.3 Hydrostatic pressure and positional changes

Studying the effect of gravity on a limb in space, and its postural relationship to the heart, is valuable when evaluating force output, CV and neuromuscular responses to changes in body positioning. There is a gravity dependent hydrostatic component to all intravascular changes within the human body.

Perfusion pressures are dependent on the distance of the activated muscle from the heart. Limbs that are elevated above heart level will have a reduction in perfusion pressure (Nielson, 1983). A reduction in perfusion has a direct implication on blood flow and hence, oxygen carrying capacity to the peripheral limb (Fitzpatrick et al. 1996). This concept can be applied inversely, with an increase in perfusion pressure and force production when the peripheral limb is below the level of the heart. During orthostasis, the body works to offset the effect of increased hydrostatic pressures and blood pooling in the periphery. All efforts are made to shunt the blood to the thorax. Mechanisms that are involved in this process include the respiratory muscle pump (Miller et al. 2005), skeletal muscle pump (Delp and Laughlin, 1998), and veno-vasoconstriction (Vissing, Secher and Victor, 1997).

In a normally functioning, circulatory system, the Frank-Starling mechanism of the heart ensures the translocation of blood and fluid from the venous to the arterial side of the

circulation. The concept of the Trendelendberg maneuver, used in medicine to restore perfusion to the heart and brain, is based upon this principle. This protocol is similar to the mechanism of HDBT, resulting in an increase in venous return via left ventricular end diastolic volume or increased preload, with subsequent augment of SV and Q with HDT. This would, in turn, divert blood flow from the non-essential organs, such as contracting muscles (Summers et al. 2009).

Work by Cattell and Edwards (1928) and Brown (1958) found that high pressures applied directly to resting muscle increased isometric twitch tension with a reduced rate of contraction and relaxation. This was concurred in 1991 by Ranatunga and Geeves, who also reported that peak tension, time to peak and twitch contraction time to half relaxation increased with increasing exposures to hydrostatic pressure in mammals. They also found a reduction in tetanic force under high hydrostatic pressures in rat muscle, along with a 15% reduction in isometric active tension in psoas muscles (Geeves and Ranatunga, 1987). Fortune et al. (1994) reported a similar effect in rabbit psoas muscles; a force depressing effect with increased hydrostatic pressure. Studies on the application of positive pressure, increasing hydrostatic pressure, to peripheral limbs, such as that by Sundberg and Kaisjer (1992) have the same results with a reduction in perfusion resulting in a reduction in force output.

On a neuromuscular level, it has been hypothesized that with changes in the hydrostatic pressure gradient, induced by HDT, there is a lower number of active crossbridges with maximum calcium activation plus/minus a reduction in the force per crossbridge (Vawda

et al. 1996) responsible for the impairment in force of fatigued muscle. Research by Heinman, Stuhmer and Conti (1987) documented decreased muscle firing frequency accompanied by decreased enzymatic activity of markers indicating tissue damage with increased hydrostatic pressure. We can assume, from this research, that an increase in hydrostatic pressure has a negative effect on the fatiguing muscle.

The purpose of our research was to further evaluate the effects of hydrostatic pressure changes on neuromuscular outcomes. We can hypothesize, based on the previous studies published in the area, that changes in posture (e.g.. upright to supine), will have a negative impact on neuromuscular outcomes. Based on Frank Starling's law we may be able to infer that HDT will increase preload, SV and Q at the level of the heart, hence diverting it from the muscles.

2.4 The neuromuscular system and postural changes

It has been demonstrated in numerous studies that acute HDBT inhibits sympathetic nervous system activity (Bosone et al. 2004, Cooke and Dowlyn, 2000, Cooke et al. 2004, Fu et al. 2000). Depression in the sympathetic nervous system is associated with a reduction in HR (Schneider and Chandler, 1973), BP (Bosone et al. 2004), total peripheral resistance (Goodman and LeSage, 2002) and muscle blood flow (Thomas and Segal, 2004) which has an effect on muscle fatigability.

Bosone et al. (2004), in an effort to clarify which aspects of the response to HDBT might

be attributed to changes in sympathetic outflow versus those secondary to increased intracranial arterial pressure, studied acute intracranial hypertension by inducing 10 minutes of -30 degree HDT and comparing hemodynamic responses to other sympathetic activation tests. The study aimed to evaluate the hydrostatic pressure gradient between the brain and heart. HDT produces an increase in the hydrostatic loading of blood from the extremities towards the chest and head. The research suggested that during HDT, the cerebrovascular bed has increased compliance, which was likely the result of a decrease in sympathetic tone.

In contrast, Heckmann et al. (1999) found that with sixty seconds of -80 degrees of HDT there was a significant reduction in HR in the first 20 seconds and a significant increase in pulsatility index, with no significant change in BP. They felt that this could suggest no activation of the sympathetic nervous system and attributed their findings to the effect of increased cerebrovascular resistance, possibly mediated by the myogenic mechanism of cerebrovascular autoregulation.

Whichever the effect, cerebrospinal fluid is impacted by the same hydrostatic gradient that affects the arterial and venous bloodstreams, resulting in a change in intracranial pressure. Bosone et al. (2004) reported that there were hemodynamic transients at the beginning and end of maneuvers, suggesting a lack of feed forward control of central circulation. The increase in intracranial pressure and reduction in sympathetic outflow may not allow all motor neurons to activate maximally via reduction of neural outflow,

reducing the ability to sustain maximal or submaximal contractions. Goldsmith (1998) and Nagaya et al. (1995) felt that this phenomenon may be due to the fact that HDT produces a sympathetic reflex inhibition secondary to loading and increased firing of the cardiopulmonary and baroreceptors reducing vascular tone in the periphery. Whatever the mechanism, it can be concluded that HDT and changes in neuromuscular measures is a complex stimulus, likely involving metabolic and myogenic mechanisms in the cerebral hemodynamic response.

Cooke et al. (2004) also demonstrated that acute HDBT affects sympathetic nervous system activity, stating that otolith activation results in an increase in muscle sympathetic nerve activity (MSNA). The purpose of this research was to investigate vestibulosympathetic reflex (VSR) activation and study the response of the CV, cerebrovascular and autonomic rhythms. They concluded that vestibular stimulation by head down rotation stimulates the efferents of the sympathetic nervous system with no obvious effect on the parasympathetic cardiac system. Cooke and Dowlyn (2000) and Cooke et al. (1999) also found a significant reduction in MSNA during HDT, the latter research finding no effect on cerebral blood flow velocity, suggesting that peripheral sympathetic activity impacts cerebral vascular responses minimally. We will further discuss the VSR and effect of vestibular adjustments during changes in body posture in the 'vestibular input and the vestibulosympathetic reflex' section of this review.

To further support the decline in sympathetic activity with changes in body positioning, Fu et al. (2000) found that with six degrees of HDT, MSNA decreased by 27%, and to a

similar extent at 10 and 20 mm Hg of lower body positive pressure (LBPP) and HDT. LBPP is used in gravitational studies to simulate the CV and renal effects of decreased gravitational stress. They did find that MAP remained unchanged at low levels of LBPP and HDT and HR was not significantly changed during the procedures. Total peripheral resistance decreased by 9% with HDT. Both SV and Q tended to decrease at 30 mm Hg LBPP, but increased with HDT. Results of this study concluded that autonomic responses differ with LBPP as compared to HDT, particularly with increasing LBPP, activating both the cardiopulmonary and arterial baroreflexes and mechanoreflexes in comparison to isolated activation of the cardiopulmonary baroreflexes with HDT.

In contrast to this, Yao et al. (1999) studied healthy subjects over increments of 24 hours of -6 degree HDT and found that HR, Q and index was significantly reduced with HDT, while SV and index, along with total peripheral resistance increased. Research performed on newborns by Fifer et al. (1999) on HDT also documented reductions in HR. This could suggest that there are other mechanisms at work in regulation of homeostasis during alterations in body position.

The nervous system contributes to control of time to task failure of a submaximal sustained contraction (Akima et al. 2002) in varying body positions. There are numerous physiological mechanisms at play during submaximal contractions and subsequent fatigue resulting in performance decrements. A reduction in perfusion pressures at the level of the muscle may result in increased motor recruitment in an effort to maintain a constant force output from the muscle. As reported by Behm (2004), the ability to maintain a

submaximal contraction is an intricate interplay of facilitation and inhibitory responses. Peripheral afferents play a role in muscle fatigability and this includes factors such as metabolites, acidity, pressure, pain, tension and stretch, among others.

To evaluate neuromuscular physiology and the effect of body posture, Rouchette, Hunter, Place and Lepers (2003) found that time to task failure with sustained submaximal contractions for the knee extensor muscles did not change in the seated versus supine position. There were no changes in EMG activities or torque fluctuations. In this study, there was, however, a 10% reduction in MVC torque, attributed to decreased activation of knee extensor muscles after the fatiguing contraction in the supine posture. This was in contrast to previous research by Hunter and Enoka (2003) evaluating submaximal isometric contractions and muscle activation in the elbow flexor muscles. The effects of reductions in sustained contractions in both postures were felt to be secondary to processes distal to the neuromuscular junction, related to structural organization of the muscle group and felt to involve both neural and peripheral mechanisms.

Maffiuletti and Lepers (2003) compared voluntary and electrically evoked knee extensor torque, EMG and activation of the rectus femoris muscle in the seated versus supine positions (increasing rectus femoris length). Results were a 10.6% increase in knee extensor MVC in the seated position; m-wave amplitude was significantly lower in the supine position along with a 4% activation reduction in the supine position. These changes were attributed to increased neural activation and motor recruitment in the seated position as compared to the supine.

Shifts within internal compartments, specifically, volume expansion relieves the effects of orthostatic intolerance. Charkoudian et al. (2004) studied the effects of 10-degree HDT and its relation to increases in central venous pressure. The study evaluated HR, arterial pressure, central venous pressure and perineal MSNA. Results found that an upright to tilted posture change increases central venous pressure, reducing sensitivity of baroreflex control of the sympathetic nerve activity and thus reducing sympathetic outflow. This reduction in sympathetic outflow is felt to have direct implications on the ability of neurons to activate fully. Sensitivity of baroreflex control of MSNA was decreased in the HDT condition. This reflex will be further discussed throughout this review.

The literature published on inversion supports the findings of reduction of sympathetic responses to changes in body posture. Three published studies (Hearn, Cahill and Behm, 2009; Paddock and Behm, 2009; Johar et al. 2013) have evaluated muscle activation, force output, and CV changes during complete inversion versus an upright body posture.

Paddock and Behm (2009) evaluated lower limb muscle activation and demonstrated significant reduction in maximal and submaximal voluntary contraction force, rate of force development and EMG muscle activity during inversion in recreational human subjects. Hearn, Cahill and Behm (2009) demonstrated a reduction in upper limb neuromuscular response with significant decreases in elbow flexor MVC force, rate of force development and biceps EMG in recreationally active participants, concluding that maximal force, instantaneous strength and muscle activation were decreased in inverted

postures with an increase in muscular co-contractions. They felt that this was as a result of impaired neuromuscular function and sympathetic response in the inverted position.

Johar et al. (2013) evaluated bicep and quadricep MVC forces, EMG activity, HR and BP with rapid and slow transitions from the upright to inverted posture and found a reduction in force output. They concluded that there was an inhibition of the sympathetic nervous system in both the rapid and slower transitions from upright to seated postures.

The studies by Paddock and Behm (2009), Hearn, Cahill and Behm, (2009) and Johar et al. (2013) have all reported inversion-induced reduction in force production, muscle activation, evoked contractile properties, HR and BP when postural stress is placed on the body. It was felt that an inhibition of sympathetic nervous system activity was the rational for reduction in this response; HDBT studies support this rationale.

The sympathetic response is the fight or flight response of the body. The above research (Bosone et al. 2004; Cooke and Dowlyn, 2000; Cooke et al. 2004; Fu et al. 2000; Paddock and Behm, 2009; Hearn, Cahill and Behm, 2009 and Johar et al. 2013) consistently demonstrates that putting the body in differing orientations affects this response mechanism, and therefore, the body organs and systems downstream from it. Sympathetic nervous system activation is linked to increases in force production (Seals and Enoka, 1989) as it may affect motor unit rate coding (Miller et al. 1981), fluctuations in cerebral blood or intracranial hydrostatic pressure and changes in BP, peripheral resistance and HR, among others (Cerretelli et al. 1997; Convertino et al. 1984;

McDonald et al. 1998). While there is some evidence to the contrary, as previously described (Heckmann et al., 1999; Yao et al., 1999 and Rouchette et al., 2003) we have hypothesized with this research that the inverted posture will reduce all sympathetic responses and contribute to subsequent reduction in MVC, force production and other neuromuscular and CV parameters.

2.5 Lower body negative pressure and baroreceptors

Research to evaluate the implications of tilt and inversion (orthostatic stress) on CV and neuromuscular response is sometimes performed with the implementation of lower body negative pressure (LBNP). LBNP increases the pressure gradient from the heart to the distal extremities, simulating what could be expected with change in body position. This review will further elaborate on some of the research in this area, as it applies to changes in body orientation.

Research performed by Hughson, Cochrane and Butler (1993) placed subjects on a cycle ergometer with submaximal exercise, positioned in upright and supine positions while investigators applied -40 mm Hg LBNP in the supine trial position. They found that LBNP resulted in increased pooling of blood in the distal lower extremities activating the baroreflex. The baroreflex is a negative feedback loop that helps maintain a constant BP; it will be discussed further in the next paragraph. Exercising in the supine position with LBNP also resulted in a faster rate of increase in VO_2 compared to supine exercise without LBNP demonstrating that oxygen transport was rate-limiting with increased

orthostatic stress.

Baroreceptors throughout the body attempt to maintain adequate cerebral perfusion. They are mechanoreceptors located in the blood vessels of all vertebrates that are activated by stretch of the blood vessel in response to stimuli, providing information about the body's position in space to the central nervous system. The nucleus tractus solitarii (NTS) and medulla oblongata recognize the response of the baroreceptor system and change the response of the sympathetic nervous system in turn. The receptor of these sensory neurons is primarily via autonomic reflexes that affect Q and total peripheral resistance. This negative feedback system is called the baroreflex. The simplest way to think about this mechanism is through the role that it plays when going from lying to standing. To maintain perfusion to the brain and distal extremities, the baroreflex counteracts the decrease in venous return and Q (Ponte and Purves, 1974). When reviewing the literature on changes in body postures, it is important to review the effect of baroreceptors and understand its role in maintaining homeostasis with changes in orientation.

Charkoudian (2004) studied baroreflex control of the sympathetic nervous system and found that small increases in central venous pressure, resulting from a change in posture from the upright to the tilted position decreased the sensitivity of baroreflex control of sympathetic nerve activity. In contrast, Cooper and Hainsworth (2001) suggested that an increase in the response of the baroreflex might help achieve maintenance of perfusion, via blood pressure, with orthostatic stress. They applied -40 mm Hg of LBNP, which resulted in a significant increase in vascular resistance with a simultaneous increase in

baroreceptor response, but did not have a significant effect on cardiac response. The ability of the body to respond to changes in posture and pump blood against gravity in healthy subjects was found within seconds of head down tilt in 2005 by Jauregui-Renaud et al. with lowering of pulse rates secondary to activation of the baroreceptors in the carotid artery and aorta.

There has been some research on the resetting of the arterial baroreflex from supine to upright body tilt; Kamiya et al. (2010) found that baroreflex reset affected control of sympathetic nerve activity in rabbits. As previously described, Hearn, Cahill and Behm (2009) and Paddock and Behm (2009) have demonstrated that during complete head down inversion, there is a significant reduction in BP that may be attributable to the baroreflex and decreased sympathetic activity.

In summary, we can hypothesize that the increase in hydrostatic pressure during changes in body orientation results in activation of the sympathetic nervous system, at least partially attributable to the response of baroreceptors. An increase in central venous pressure will activate the baroreflex, with the proceeding cascade of events previously described. The objective of this research was to study CV changes with inversion and supine postures in athletes, when interpreting these data, it is important to understand the role that the baroreflex may play.

2.6 Respiratory system

The effect of HDT has also been studied on the respiratory system (Doberentz and Madea, 2009; Henderson, 2006). It has been found that with a change in body position there is a compensatory increase in inspiratory muscle contraction force (Aleksandrova et al. 2005), impaired gaseous exchange (Prisk et al. 2002), decreased lung compliance (Donina et al. 2009), changes in pulmonary blood flow (Hillebrecht et al. 1992) and an increase in airflow impedance of the respiratory system (Donina et al. 2009). It can be speculated that the culmination of these factors may result in a reduction of oxygen supply to the working muscles and an increased work of breathing during supine body position. HDT contributes to redistribution of blood from caudal to cephalic; shunting blood into the pulmonary vasculature and activating arterial baroreceptors, as previously discussed. The results of research by Henderson et al. (2006), suggested that during tilt, pulmonary vascular pressures increase, with greater heterogeneity on pulmonary functional MRI scan. Lying supine, also causes an increase in blood flow dispersion throughout the lungs, however, a much smaller effect in comparison to HDT.

Prisk et al. (2002) reported that pulmonary function during HDT and supine postures differ from changes that are modulated by CV function. They concluded that pulmonary ventilation is a result of mechanical changes within the lungs, chest and abdomen, whereas CV changes are controlled by fluid shifts. The effects of postural change on pulmonary gas exchange investigated by Hillebrecht et al. (1992) over 10 days of 6

degrees HDT and LBNP reported decreased pulmonary blood flow and diffusing capacity with 24% reduction of pulmonary blood flow with LBNP and 7% reduction in diffusing capacity with minimal change in lung tissue volume. The researchers concluded that LBNP caused significant changes in pulmonary blood-flow and minor effects on pulmonary gas exchange. Aleksandrova et al. (2005) reported that -30 degrees HDT results in reduction of minute ventilation and functional reserve of the respiratory system, resulting in failure of the diaphragm during prolonged exposure (21 days) in rats. Donina et al. (2009) reported an increase in negative intrathoracic pressure during HDT with increased inspiratory muscle effort as a result of decreased lung compliance and increased airflow impedance in the inverted position. The increased central venous pressure with HDT is felt to play a role in this; causing a swelling of bronchial mucosa and increase in lung elastic recoil.

Oxygen is essential for adequate function of skeletal muscles (Hepple et al. 2002), hence, decreased oxygen supply may reduce force production and impair excitation-contraction coupling. Inverted postures decrease orthostatic stability causing an increase in inspiratory contraction force, along with contractile failure of the diaphragm musculature due to alteration in excitation-contraction coupling. There are significant changes associated with posture that affect the ventilatory system. We can postulate, based on these findings, that oxygen supply to the working muscle may be negatively affected secondary to a reduction in lung compliance, increased airflow impedance and fatigue of the diaphragmatic muscle, all associated with changes in body orientation.

2.7 Vestibular input and the vestibulosympathetic reflex

There are many inputs and outputs involved in maintaining body homeostasis while undergoing the stress of postural change. Adjustments are accomplished through the cooperation of various systems. These include somatic motoneurons to induce muscle contraction and complementary autonomic outflows to ensure adequate blood supply to contracting muscles and the brain. There are several purposes of these adjustments. Firstly, it assists in autonomic changes required during posture change; secondly, it ensures maintenance of homeostasis by ensuring adequate blood flow distribution. There are two types of input involved in the coordination of motor and autonomic responses. The central command is believed to affect efferent and afferent outflow simultaneously (Waldrop et al. 1996), while the sensory input from somatic efferents (working muscles) and visceral afferents (stretch and baroreceptors) exert autonomic control during changes in posture and exercise (Kaufman and Forster, 1996) in a complementary feedforward and feedback manner, respectively.

It has been studied, that there is a close connection between the vestibular system and autonomic control (Costa et al. 1995). As the vestibular system senses changes in body posture, there is evidence that the system responds to these changes to achieve homeostasis via activation of the sympathetic and parasympathetic nervous systems (Kaufmann et al. 2002). The autonomic control of this system is also affected by thermal stress, mental stress and arterial chemoreceptor responses (Mathias and Bannister, 2013).

Changes in the head's position in space are quickly and accurately detected by afferent inputs from vestibular end organs that participate in regulation of sympathetic activity. A change in posture engages the otoliths (which respond to linear motion) and increases sympathetic activity (Cobbold et al. 1968) in humans (Shortt and Ray, 1997). Ray and Carter (2003) examined the effect of sympathetic activity, by inducing head down rotation in participants to physically alter the vestibular system. The head was rotated downwards while the subject was in the prone position, activating the otolith organs, while simultaneously measuring MSNA and CV outcomes. It was concluded that alterations in MSNA during head down rotation are a direct result of alterations in the otolith organs as outside factors were controlled. Ray (2000) also concluded that the VSR increases sympathetic outflow to maintain orthostasis in humans and during upright standing, assists in BP regulation in humans.

The VSR plays a large role in controlling this interplay of numerous mechanisms; acting synergistically with sympathetic reflexes. The NTS contributes to vestibular input of the VSR as it receives third-order afferent inputs from the vestibular system, which in turn synapse with the carotid sinus baroreflex (Spyer, 1981) and exert an inhibitory response on the rostral ventrolateral medulla; influencing CV control. Subjects with a reduced vestibular response have a lower CV response to stress on the system, supporting the hypothesis that the vestibular system impacts CV responses (Yates et al. 1999).

Early evidence suggesting the presence of the VSR was through caloric and electrical labyrinthe stimulation that elicited inhibitory and excitatory alterations in CV function

and sympathetic nerve activity (Cobbold, Megirian and Sherrey, 1968; Ishikawa and Miyazawa, 1980 and Megirian and Manning, 1967). Spiegel (1946) reported a reduction in BP in response to labyrinth stimulation, which was early evidence of the interaction between the vestibular and autonomic pathways.

Kerman and Yates (1999), in an effort to narrow the influence of the vestibular system on sympathetic outflow, recorded activity of multiple sympathetic nerves, whist simultaneously activating vestibular afferents. The findings of this research suggest that inner ear inputs have potential to produce changes in autonomic functions throughout the body. It was found that the renal nerve, which may be directly implicated in CV endpoints, was particularly sensitive to vestibular stimulation. Due to the fact that the VSR contributes directly to CV function during postural adjustments (Yates, 1996), it can be deduced that the regional differences in VSR activity effects innervation of vascular smooth muscle. The neurons of the rostral ventrolateral medulla have been shown to receive inputs from the vestibular neurons, with the primary function of regulating BP (Ceriello, Caverson and Polosa, 1986). This has implications on regional hemodynamics and vasoconstrictor efferent activity. Overall, the VSR serves a complex role in ensuring adequate blood supply to muscle involved in postural control while maintaining perfusion to the brain (Kerman, McAllen and Yates, 2000).

There have been mixed results in the literature on MSNA and vestibular stimulation. Research by Costa et al. (1995) designed to compare vestibular stimulation and sympathetic response found no effect on HR, BP, plasma norepinephrine levels and

MSNA. They did elicit however, that the limitation of this research was the localized measurement of activity to the perineal nerve. While there were no acute changes in the vascular beds in this region, it could be postulated that there were unmeasured changes elsewhere in the body. However, the lack of significant change of systemic indicators of perfusion may indicate otherwise. To explore this further, future research is warranted.

In contrast to these findings, research by Cui et al. (1997) found changes in HR after caloric stimulation of the labyrinthe system, with an enhancement of sympathetic stimulation proportional to the magnitude of vestibular excitement. Tanaka et al. (2006) concurred with this as vestibular-deficient rats, were less able to regulate BP as compared to their vestibular-intact equivalents. They also found that galvanic vestibular stimulation, which is the process of sending electrical signals to the vestibular nerve, obscured the vestibulo-cardiovascular reflex in rats during gravitational change, reducing the above effect. To also examine the response of MSNA on stimulation of the otolith organs, Kowanokuchi et al. (2001) applied 10 mm Hg of LBNP and 6 to 8.5 degrees of HDT. They found that VSR rather than cardiopulmonary baroreceptors suppress sympathetic outflow with 6 to 8 degree HDBT with LBNP in humans.

Other mechanisms that are active in an effort to maintain homeostasis with changes in posture include the interaction of baroreceptors and skeletal muscle reflexes. Ray and Carter (2003) found that there was an additive interaction between baroreceptor unloading and skeletal muscle afferent feedback with MSNA in humans. They found a similar additive interaction for skeletal muscles and the VSR. The authors concluded that

MSNA activation by the vestibular system is a robust reflex that is capable of activation, simultaneously with other reflexes.

We can conclude, that the VSR is vital to maintain homeostasis during gravity related changes, which would apply to complete inversion and the supine body postures. The above research seems to indicate that reduction in sympathetic nervous system activation is associated with stimulation of the vestibular system and VSR. This may play a direct role on CV and neuromuscular changes during changes in body orientation.

2.8 Aerobic physiologic adaptations

Previous complete inversion studies have been performed on recreationally active individuals (Paddock and Behm, 2009; Hearn, Cahill and Behm, 2009 and Johar et al. 2013) there is, however, little research on the effects of changes in position on aerobically trained individuals. There is little research, as far as we are aware, on the effect of acute changes in posture and adjustment in the aerobically trained athlete.

Aerobic exercise increases CV endurance. The skeletal muscle becomes more efficient in the delivery of oxygen and substrates. There is greater recruitment and changes in discharge rates of the motor unit at the neuromuscular junction, there is also counteraction of autogenic inhibition, a reduction of co-activation of agonist-antagonist muscle response and an increased number of muscular crossbridges, thought to be secondary to muscle fiber hypertrophy, and possibly hyperplasia in endurance trained individuals.

There are numerous biochemical adaptations with endurance training, including an increased number of mitochondria, increased myoglobin stores transporting oxygen to the muscle, as well as increased capillary density and availability of substrates that produce ATP aerobically such as triglycerides and glycogen. CV changes associated with aerobic exercise include an increase in SV, reduced resting and submaximal HR and increase in the effective redistribution of blood, along with increase in minute volume, vital capacity, improved strength of the respiratory muscles and increased oxygen diffusion (Kenney, Wilmore and Costill, 2012). These adaptations may be able to counteract some of the previously described physiologic changes with HDT and inversion.

We know that aerobic training is associated with a reduction in BP in normotensive trained individuals and this would be indicative of an effect on the baroreflex via the sympathetic nervous system (Iellamo et al., 2000; Niemela et al., 2008; Pagani et al., 1988; Somers et al., 1991). A review by Martins-Pinge in 2011 evaluated literature on the CV adaptations associated with chronic exercise that may be the result of central nervous system plasticity. Research focused on the NTS and caudal ventrolateral medulla, involved in regulating cardiac and vascular autonomic function. The results of these studies indicate that there is a beneficial effect of aerobic training due to reductions in sympathetic nervous system activity associated with physical fitness. A review article by Hautala et al. (2009) commented on numerous studies linking aerobic fitness to improvement in autonomic regulation. Their review stated that regular (chronic) aerobic training is associated with increased cardiac vagal modulation of HR and reciprocal reduction of sympathetic activity.

Literature published on prolonged HDT and exercise includes data on 120 days of 5 degrees HDT in humans. The effects of physical training were evaluated with absolute reductions in MVC; electrically evoked tetanic tension and isometric twitch contraction. There was a force deficiency of 40% after 120 days of bedrest. However, overall conclusions from this study were that physical training provided a reserve of neuromuscular function in these subjects with neural and muscular adaptations in comparison to no physical training (Koriak, 2013).

Abad et al. (2014) examined cardiac sympathetic nervous system control in a comparison of high endurance and power track and field athletes, hypothesizing that these athletes would have significantly different cardiac autonomic control at rest. Cardiac autonomic activity was measured in HR variability time and frequency domains and symbolic analysis. HR variability is used to analyze sympathetic activity as it corresponds to R-R interval fluctuations. The low frequency (LF) and high frequency (HF) oscillations are an index of sympathetic activity (Hautala et al. 2009). There were no differences found between the groups, in time and frequency domains; LF/HF ratio had high effect sizes, and symbolic analysis showed a significant increase in parasympathetic modulation and reduction in sympathetic modulation in endurance, as compared to power track and field athletes.

The effect of aerobic training on autonomic control of HR during exercise in a group of

middle-aged men was studied by Chacon-Mikahil (1998). Half of the group underwent an exercise-training program three times a week for ten months at 70-85% max HR, while the other half maintained a sedentary lifestyle. The investigators examined the effect of sympathetic and parasympathetic exercise tachycardia based on HR response with increasing and steady state wattage on a cycle ergometer. There was a significantly lower resting HR during exercise in the trained group; there was also a significant decrease in HR during exercise at the middle and high wattage powers. They found a significant decrease in slow tachycardia (1-4 minutes) and a small increase in fast tachycardia (0 - 10 seconds) in the trained group. These changes were thought to be related to the sympathetic-dependent mechanism. Oxygen uptake and peak were also higher in the trained group. The authors concluded that aerobic training resulted in physiologic adaptations expressed as a reduction in sympathetic effects on HR and an increase in oxygen transport during exercise.

To summarize, the above articles demonstrate a reduction of sympathetic nervous system activity in trained subjects. As discussed throughout this review, changes in body posture have been proven to have an effect on the autonomic nervous system, namely, a reduction in sympathetic activity leading to changes in CV and neuromuscular outcomes. The purpose of this study is to evaluate changes in endurance-trained individuals in supine and head-down inverted positions. If these subjects already have training induced changes in sympathetic activity, we can postulate that their response to change in body posture may differ from that of the recreational population.

2.9 Conclusion

This review has identified and discussed numerous mechanisms that may alter CV and neuromuscular parameters during changes in body position. There is a complex interaction between efferent and afferent responses and central and peripheral factors to maintain homeostasis under physiologic stress. Changes in the CV and respiratory system may affect oxygen availability to the working muscle, this, in combination with increased hydrostatic pressure, changes in the baroreflex and the contribution of the vestibular system all play a role in the responses of the body expected with postural change.

Previous research by Paddock and Behm (2009), Hearn, Cahill and Behm (2009) and Johar et al. (2013) on recreationally active subjects have found a significant reduction in CV and neuromuscular outcomes in upper and lower extremities and rapid versus slow inversion. As stated above, sympathetic responses in trained athletes have been shown to be different from the recreational population, and the effects of acute outcomes with supine and inverted positions has not been evaluated in this population. Will these changes be equivocal to the sympathetic response seen in previous studies with reductions in force output, maximal voluntary contraction, EMG, HR, BP and other CV and neuromuscular parameters? This research study was designed to investigate this further and possibly provide insight into the physiological mechanisms behind these questions.

2.10 References

Abad, C.C., do Nascimento, A.M., Gil, S., Kobal, R., Loturco, I., Nakamura, F.Y., Mostarda, C.T. and Irigoyen, M.C. (2014). Cardiac autonomic control in high level brazilian power and endurance track-and-field athletes. *International Journal of Sports Medicine,* 35, 772-778.

Akima, H., Foley, J.M., Prior, B.M., Dudley, G.A., and Meyer, R.A. (2002). Vastus lateralis fatigue alters recruitment of musculus quadriceps femoris in humans. *Journal of Applied Physiology,* 92, 679-684.

Aleksandrova, N.P., Baranov, V.M., Tikhonov, M.A., Kolesnikov, V.I., Kotov, A.N., and Kochanov, V.S. (2005) The effect of head-down hypokinesia on functional state of diaphragm in rats. Rossiiskii fiziologicheskii zhurnal imeni I. M. Sechenova, 91, 1312-1319.

Behm, D.G. (2004) Force Maintenance with Submaximal Fatiguing Contractions. *Canadian Journal of Applied Physiology,* 29, 274-290.

Bosone D., Ozturk V., Roatta S., Cavallini A., Tosi P., and Micieli G. (2004) Cerebral haemodynamic response to acute intracranial hypertension induced by head- down tilt. *Functional Neurology,* 19**,** 31-35.

Brown, D.E., Guthe K.F., Lawlor, H.C. and Carpenter, M.P. (1958) The pressure, temperature and ion relations of myosin ATP-ase. *Journal Cell Physiology,* 52, 59-77.

Cattell, M. and Edwards, D.J. (1928b). The energy changes of skeletal muscle accompanying contraction under high pressure. *American Journal of Physiology,* 86, 371-381.

Cerretelli, P., Shindell, D., Pendergast, D.P., Di Prampero, P.E., and Rennie, D.W. (1977) Oxygen uptake transients at the onset and offset of arm and leg work. *Respiratory Physiology* 30, 81–97.

Ciriello, J., Caverson, M.M. and Polosa, C. (1986). Function of the ventrolateral medulla in the control of circulation. *Brain Research Review,* 11, 259-391.

Chacon-Mikahil, M.P.T., Forti, V.A.M, Catai, A.M., Charkoudian N., Szrajer, J.S., Golfetti, R., Martins, L.E.B., Lima-Filho, E.C., Wanderley, J.S., Marin-Neto, J.A., Maciel, B.C. and Gallo-Jr, L. (1998). Cardiorespiratory adaptations induced by aerobic training in middle-aged men: the importance of a decrease in sympathetic stimulation for the contribution of dynamic exercise tachycardia. *Brazilian Journal of Medical and Biological Research,* 31, 705-712.

Charkoudian N, Martin E.A., Dinenno F.A., Eisenach J.H., Dietz N.M., and Joyner M.J. (2004) Influence of increased central venous pressure on baroreflex control of sympathetic activity in humans. *American Journal of Physiology and Heart Circulatory Physiology,* 287**,** H1658-H1662.

Cobbold, A.F., Megirian, D., and Sherrey, J.H. (1968). Vestibular evoked activity in autonomic motor outflows. *Archives of Italian Biology,* 106, 113-123.

Convertino, V.A., Goldwater, D.J. and Sandler, H. (1984). Oxygen uptake kinetics of constant-load work: upright vs. supine exercise. *Aviation, Space and Environment Medicine,* 55, 501-506.

Cooke, W.H., Carter, J.R., and Kuusela, T.A. (2004) Human cerebrovascular and autonomic rhythms during vestibular activation. *American Journal of Physiology Regulatory, Integrative and Comparative Physiology,* 286, R838–R843.

Cooke, W.H. and Dowlyn, M.M. (2000) Power spectral analysis imperfectly informs changes in sympathetic traffic during acute simulated microgravity. *Aviation, Space and Environmental Medicine,* 71, 1232–1238.

Cooke, W.H., Hoag, J.B., Crossman, A.A., Kuusela, T.A., Tahvanainen, K.U.O., and Eckberg, D.L. (1999). Human responses to upright tilt: a window on central autonomic integration. *Journal of Physiology,* 517, 617–628.

Cooper, V.L and Hainsworth, R. (2001). Carotid baroreceptor reflexes in humans during orthostatic stress. *Experimental Physiology,* 86, 677–681.

Costa, F., Lavin, P., Robertson, D., and Biaggioni, I. (1995). Effect of neurovestibular stimulation on autonomic regulation. *Clinical Autonomic Research,* 5, 289-293.

Cui, J., Mukai, C., Iwase, S. Sawasaki, N., Kitazawa, H., Mano, T., et al. (1997). Response to vestibular stimulation of sympathetic outflow to muscles in humans. *Journal of Autonomic Nervous System,* 66, 154-162.

Doberentz, E. and Madea, B. (2009). Drowning in a head-down position - report of an unusual accident. *Achives of Kriminology,* 224, 108-115.

Donina, Zh.A., Danilova, G.A., and Aleksandrova, N.P. (2009). Effects of body position on the ventilatory response to hypercapnia. *European Journal of Medical Research,* 7 Suppl 4:63-66.

Delp M.D., and Laughlin M.H. (1998) Regulation of skeletal muscle perfusion during exercise. *Acta Physiologic Scandinavica,* 162**,** 411-419.

Egana, M. and Green, S. (2005) Effect of body tilt on calf muscle performance and blood flow in humans. *Journal of Applied Physiology,* 98, 2249-2258.

Essandoh, L., Duprez, D., and Shepherd, J. (1988). Reflex constriction of human resistance vessels to head down neck flexion. *Journal of Applied Physiology,* 64, 767-770.

Fifer, W.P., Greene, M., Hurtado, A., and Myers, M.M. (1999) Cardiorespiratory responses to bidirectional tilts in infants. *Early Human Development,* 55, 265-279.

Fitzpatrick, R., Taylor, J.L. and McCloskey, D.I. (1996). Effects of arterial perfusion pressure on force production in working human hand muscles. *Journal of Physiology,* 495, 885-891.

Folkow, B., Haglund, U., Jodal, M., and Lundgren, O. (1971). Blood flow in the calf muscle of man during heavy rhythmic exercise. *Acta Physiologica Scandinavica,* 81, 157-163.

Fortune, N.S., Geeves, M.A. and Ranatunga, K.W. (1994). Contractile activation and force generation in skinned rabbit muscle fibers: effects of hydrostatic pressure. *Journal of Physiology,* 424, 283-290.

Fu, Q., Sugiyama, Y., Kamiya, A., and Mano, T. (2000) A comparison of autonomic responses in humans induced by two simulation models of weightlessness: lower body positive pressure and 6 degrees head-down tilt. *Journal of the Autonomic Nervous System,* 80, 101-107.

Geeves, M.A. and Rantunga, K.W. (1987) Tension responses to increased hydrostatic pressure in glycerinated rabbit psoas muscle fibres. *Proceedings of the Royal Society,* B232, 217-226.

Goldsmith, S.R. (1991). Impaired suppression of plasma norepinephrine during head-down tilt in patients with congestive heart failure. *American Heart Journal,* 122, 104-107.

Goodman, L.S. and LeSage, S. (2002) Impairment of cardiovascular and vasomotor responses during tilt table simulation of 'push-pull' maneuvers. *Aviation Space and Environmental Medicine,* 73, 971-979.

Hautala, A.J., Kiviniemi, A.M., and Tulppo, M.P. (2009) Individual responses to aerobic exercise: The role of the autonomic nervous system. *Neuroscience and Biobehavioral Reviews,* 33, 107-115.

Hearn J., Cahill F., and Behm D.G. (2009) An inverted seated posture decreases elbow flexion force and muscle activation. *European Journal of Applied Physiology* 106**,** 139-147.

Heckmann, J.G., Hilz, M.J., Hagler, H., Mueck W.M. and Neundoerfer, B. (1999). Transcranial Doppler sonography during acute 80 degree head-down tilt for the assessment of cerebral autoregulation in humans. *Neurological Research*, 21, 457-462.

Heinemann, S.H., Conti, F. Stuhmer, W. and Neher, E. (1987). Effects of hydrostatic pressure on membrane processes. Sodium channels, calcium channels, and exocytosis. *Journal of General Physiology,* 90, 765-778.

Henderson, A.C., Levin, D.L., Hopkins, S.R., Olfert, I.M., Buxton, R.B., and Prisk, G.K. (2006). Steep head-down tilt has persisting effects on the distribution of pulmonary blood flow. *Journal of Applied Physiology,* 101, 583-589.

Hepple, R.T. (2002) The role of O2 supply in muscle fatigue. *Canadian Journal of Applied Physiology,* 27, 56-69.

Hillbrecht, A., Schulz, H., and Meyer, M, Baisch, F., Beck, L., and Blomgvist, C.G. (1992). Pulmonary responses to lower body negative pressure and fluid loading during head-down tilt bedrest. *Acta Physiologica Scandinavica Suppl.,* 604, 35-42.

Hogan, M.C., Richardson, R.S., and Kurdak, S.S. (1994). Initial fall in skeletal muscle force development during ischemia is related to oxygen availability. *Journal of Applied Physiology,* 77, 2380-2384.

Hughson, R.L., Cochrane, J.E., and Butler, G.C. (1993). Faster O2 uptake kinetics at onset of supine exercise with than without lower body negative pressure. *Journal of Applied Physiology,* 72, 1962-1967.

Hunter, S.K. and Enoka, R.M. (2003). Changes in muscle activation can prolong the endurance time of a sub maximal isometric contraction in humans. *Journal of Applied Physiology,* 94, 108-118.

Iellamo, F., Legramante, J.M., Massaro, M., Raimondi, G., and Galante, A. (2000). Effects of a residential exercise training on baroreflex sensitivity and heart rate variability in patients with coronary artery disease: a randomized, controlled study. *Circulation*,102, 2588–2592.

Ishikawa, T. and Miyazawa, T. (1980). Sympathetic responses evoked by vestibular stimulation and their interactions with somato-sympathetic reflexes. *Journal of Autonomic Nervous System,* 1, 243-254.

Ivanenko, Y., Solopova, I., and Levik, Y. (2000) The direction of postural instability affects postural reactions to ankle muscle vibration in humans. *Neuroscience Letters,* 292, 103-106.

Johar P., Grover V., Disanto M.C., Button D.C., and Behm D.G. (2013) A rapid rotation to An inverted seated posture inhibits muscle force, activation, heart rate and blood

pressure. *European Journal of Applied Physiology,* 113, 2005-2013.

Jaufman, M.P. and Forster, H.V. (1996). Reflexes controlling circulatory, ventilatory and airway responses to exercise. In Oxford University Press (Ed.), *Roswell, LB., Shepherd, J.T., eds. Handbook of Physiology,* (pp. 381-447), New York.

Jauregui-Renaud, K., Villanueva, P.L., and del Castillo, M.S. (2005). Influence of acute unilateral vestibular lesions on the respiratory rhythm after active change of posture in humans subjects. *Journal of Vestibular Research,* 15, 41-8.

Kaufman, M.P., Biaggioni, A., Voustianiouk, A., Diedrich, F., Costa, R., Clarke, M., Gizzi, T., and Raphan, B. (2002). Vestibular control of sympathetic activity. *Experimental Brain Research*, 143, 463-469.

Kaufman, M.P. and Forster, H.V. (1996). Reflexes controlling circulatory, ventilatory and airway responses to exercise. In Oxford University Press (Ed.), *Rowell, L.B.; Shepherd, J.T., eds. Handbook of Physiology* (pp. 381-447). New York.

Kawanokuchi, J., Fu, Q., Cui, J., Niimi, Y., Kamiya, A., Michikami, D., Iwase, S., Mano, T., and Suzumura, A. (2001). Influence of vestibulo-sympathetic reflex on muscle sympathetic outflow during head-down tilt. *Environmental Medicine,* 45, 66-68.

Kamiya A., Kawada T., Yamamoto K., Michikami D., Ariumi H., Uemura K., Zheng C., Shimizu S., Aiba T., Miyamoto T., Sugimachi M., and Sunagawa K. (2005b). Resetting of the arterial baroreflex increases orthostatic sympathetic activation and prevents postural hypotension in rabbits. *Journal of Physiology,* 566, 237-246.

Kenney, W.L., Milmore, J.H., and Costill, D.L. (2012). *Physiology of Sport and Exercise (5th Edition).* USA: Human Kinetics.

Kerman I.A. and Yates, B.J. (1999). Patterning of sympathetic nerve activity in response to vestibular stimulation. *Brain Research Bulletin,* 53, 11-16.

Kerman, I.A., McAllen, R.M., and Yates, B.J. (2000). Patterning of sympathetic nerve activity in response to vestibular stimulation. *Brain Research Bulletin,* 53*,* 11-16.

Kerman, I.A., Yates, B.J., and McAllen, R.M. (2000). Anatomic patterning in the expression of vestibulosympathetic reflexs. *American Journal of Physiology Regulation Integrative and Comparative Physiology,* 279*,* R109-R117.

Koga, I.A., McAllen, R.M., and Yates, B.J. (2000). Patterning of sympathetic nerve activity in response to vestibule stimulation. *Brain Research Bulletin,* 53, 11-16.

Kollmitzer, J., Ebenbichler, G.R., Sabo, A., Kerschan, K., and Bochdansky, T. (2000). Effects of back extensor strength training versus balance training on postural control.

Medicine and Science in Sports and Exercise, 32, 1770-1776.

Koriak, I.A. (2013). Influence of physical training under conditions of 120-day simulated microgravity on contractile properties and musculo-tendinous stiffness of the triceps sure muscle. *Fiziol Zh.* 59,71-84.

Laughlin, M.H. and Schrage, W.G. (1999). Effects of muscle contraction on skeletal muscle blood flow: when is there a muscle pump? *Medicine and Science in Sports and Exercise,* 31, 1027-1035.

Lu, L.L., Zhong, C.F., Yang, J.S., Tao, Y., and Zhao, G.X. (2000). Effects of -30 degrees head down tilt on lung function. *Space Medicine and Medical Engineering (Beijing),* 13, 187-190.

MacDonald, M.J., Shoemaker, J.K. Tschakovsky, M.E. and Hughson, R.L. (1998). Alveolar oxygen uptake and femoral artery blood flow dynamics in upright and supine leg exercise in humans. *Journal of Applied Physiology,* 85, 1622-1628.

Maffiuletti, N.A. and Lepers, R. (2003). Quadriceps femoris torque and EMG activity in seated versus supine position. *Medicine and Science in Sports and Exericse* 35, 1511-1516.

Mathias, C.J. and Bannister R. (2013). *Autonomic Failure: A Textbook of Clinical Disorders of the Autonomic Nervous System (5th Edition)*. Oxford, United Kingdom: Oxford University Press.

Martins-Pinge, M.C. (2011). Cardiovascular and autonomic modulation by the central nervous system after aerobic exercise training. *Brazilian Journal of Medical and Biological Research,* 44, 848-854.

Megirian, D. and Manning, J.W. (1967). Input-output relations of the vestibular system. *Archives Italiennes de Biologie*, 105, 15-30.

Midgley A.W., McNaughton L.R., Polman R., and Marchant D. (2007). Criteria for determination of maximal oxygen uptake: a brief critique and recommendations for future research. *Sports Medicine,* 37**,** 1019-1028.

Miller, R.G., Mirka, A., and Maxfield, M. (1981). Rate of tension development in isometric contractions of a human hand muscle. *Experimental Neurology,* 73, 267-285.

Nagaya, K., Wada, F., Nakamitsu, S., Sagawa, S., and Shiraki, K. (1995). Responses of the circulatory system and muscle sympathetic nerve activity to head-down tilt in humans. *American Journal of Physiology* ,268, R1289-R1294.

Neary, J.P., Salmon, D.M. Pritchett, E., and Behm, D.G. (2011b). Effects of an inverted body position on muscle force and cardiovascular parameters. *European College of*

Sports Science, 11:S234

Nielson, H.V. (1983). Arterial Pressure - blood flow relations during limb elevation. *Acta Physiologica Scandinavica,* 118, 405-413.

Niemela, T.H., Kiviniemi, A.M., Hautala, A.J., Salmi, J.A., Linnamo, V., and Tulppo, M.P. (2008). Recovery pattern of baroreflex sensitivity after exercise. *Medicine and Science in Sports and Exercise,* 40, 864–870.

Paddock N. and Behm D.G. (2009). The effect of an inverted body position on lower limb muscle force and activation. *Applied Physiology Nutrition and Metabolism,* 34**,** 673-680.

Pagani, M., Somers, V., Furlan, R., Dell'Orto, S., Conway, J., Baselli, G., Cerutti, S., Sleight, P., and Malliani, A. (1988). Changes in autonomic regulation induced by physical training in mild hypertension. *Hypertension,* 12, 600–610.

Permutt, S. and Riley, R.L. (1963). Hemodynamics of collapsible vessels with tone the vascular waterfall. *Journal of Applied Physiology,* 18, 924-932.

Ponte J., and Purves M.J. (1974). The role of the carotid body chemoreceptors and carotid sinus baroreceptors in the control of cerebral blood vessels. *Journal of Physiology,* 237**,** 315-340.

Prisk G.K., Fine J.M., Elliott A.R., and West J.B. (2002). Effect of 6 degrees head-down tilt on cardiopulmonary function: comparison with microgravity. *Aviation Space and Environmental Medicine,* 73**,** 8-16.

Raffai, G., Cseko, C., Kocsis, L., Dezsi, L. and Monos, E. (2009). Does long term experimental antiorthostasis lead to cardiovascular deconditioning in the rat? *Physiology Research,* 58, *57-67.*

Ranatunga, K.W., and Geeves, M.A. (1991). Changes produced by increased hydrostatic pressure in isometric contractions of rat fast muscle. *Journal of Physiology,* 441, 423–431

Ray, C.A. (2000). Interaction of the vestibular system and baroreflexes on sympathetic nerve activity in humans. *American Journal of Physiology - Heart and Circulatory Physiology,* 279, H2399-2404.

Ray, C.A. and Carter, I.R. (2003). Vestibular activation of sympathetic nerve activity. *Acta Physiologica Scandinavica* 177, 313-49.

Rochette, L., Hunter, S.K., Place, N., and Lepers, R. (2003). Activation varies among the knee extensor muscles during a submaximal fatiguing contraction in the seated and supine postures. *Journal of Applied Physiology,* 95, 1515-1522.

Schneider, M.F. and Chandler, W.K. (1973).Voltage dependent charge movement of

skeletal muscle: a possible step in excitation-contraction coupling. *Nature,* 242, 244-246.

Seals, D.R. and Enoka, R.M. (1989). Sympathetic activation is associated with increases in EMG during fatiguing exercise. *Journal of Applied Physiology,* 66, 88-95.

Shortt, T.L. and Ray, C.A. (1997). Sympathetic and vascular responses to head-down neck flexion in humans. *American Journal of Physiology,* 272, H1780-H1784.

Slijper, H. and Latash, M. (2000). The effects of instability and additional hand support on anticipatory postural adjustments in leg, trunk, and arm muscles during standing. *Experimental Brain Research,* 135, 81-93.

Somers, V.K., Conway, J., Johnston, J., and Sleight, P. (1991). Effects of endurance training on baroreflex sensitivity and blood pressure in borderline hypertension. *Lancet* 337, 1363–1368.

Spiegel, E.A. (1946). Effect of labyrinthe reflexes on the vegetative nervous system. *Archives of Otolaryngology*, 44, 31-72.

Spyer, K.M. (1981). Neural organization and control of the baroreceptor reflex. *Reviews of Physiology, Biochemistry and Pharmacology,* 88, 24-124.

Stewart, J.M. and Clarke, D. (2011). "He's Dizzy when he Stands Up." An introduction to initial orthostatic hypotension. *Journal of Pediatrics,* 158, 499-504.

Summers, R.L., Thompson, J.R., Woodward, L.H. and Martin, D.S. (2009). Physiologic Mechanisms Associated with the Trendelenburg Position. *American Journal of Clinical Medicine,* 6, 24-27.

Tanaka K., Gotoh T.M., Awazu C., and Morita H. (2006). Roles of the vestibular system in controlling arterial pressure in conscious rats during a short period of microgravity. *Neuroscience Letters,* 397, 40–43.

Thomas, G.D. and Segal, S.S. (2004). Neural control of muscle blood flow during exercise. *Journal of Applied Physiology,* 97, 731–738.

Vawda, F., Ranatunga, K.W. and Geeves, M.A. (1996). Effects of hydrostatic pressure on fatiguing frog muscle fibers. *Journal of Muscle Research and Cell Mobility* 17(6), 631-636

Vissing, S.F., Secher, N.H., and Victor, R.G. (1997). Mechanisms of cutaneous vasoconstriction during upright posture. *Acta Physiologica Scandinavica,* 159, 131-138.

Waldrop, T.G., Eldridge, F.L., Iwamoto, G.L., and Mitchell, J.H. (1996). Central neural control of respiration and circulation during exercise. In Oxford University Press (Ed.), *Rowell, L.B.; Shepherd, J.T., eds. Handbook of Physiology* (pp. 333-380). New York.

Wallin, B.G. and Sundlof, G. (1982). Sympathetic outflow to muscles during vasovagal syncope. *Journal of the Autonomic Nervous System,* 6, 287-291.

Yao, Y.J., Wu, X.Y., Sun, X.Q., Hao, W.Y., Wei, Y.B., and Cao, X.S. (1999). Effects of 24 h -6 degrees head-down tilt bed-rest on cardiovascular function and response to orthostatic stress. *Space Medicine and Medical Engineering (Beijing),* 12, 401-405.

Yates, B.J. (1996). Vestibular influences on the autonomic nervous system. *Annals of the New York Academy of Science,* 781, 458-473.

Yates B.J., Aoki M., Burchill P., Bronstein A.M., and Gresty M.A. (1999). Cardiovascular responses elicited by linear acceleration in humans. *Experimental Brain Research,* 125, 476–484.

EFFECTS OF SUPINE AND INVERTED SEATED POSITIONS ON NEUROMUSCULAR AND CARDIOVASCULAR PARAMETERS

E. Casey, D.G Behm, J. P Neary, D.M Salmon

School of Human Kinetics and Recreation, Memorial University of Newfoundland, St. John's, Newfoundland and Labrador, Canada

Faculty of Kinesiology and Health Studies, University of Regina, Regina, Saskatchewan, Canada

RUNNING TITLE: Responses to Inversion

3.1 Introduction

Humans involuntarily and voluntarily adopt a variety of postures. In addition to the predominant upright posture, humans may voluntarily become inverted during sporting (gymnastics), recreation (zip line) and vocational (e.g. military pilot, circus trapeze artists) activities or involuntarily with vehicle accidents (e.g. automobiles, buses, helicopters). Previous research has demonstrated that an inverted seated position can lead to impairments of maximal voluntary contraction (MVC) force, electromyography (EMG) activity, heart rate (HR) and blood pressure (BP) (Hearn et al., 2009, Paddock and Behm, 2009, Johar et al., 2013). Collectively these authors suggest that these responses may be related to altered sympathetic nervous stimulation. Whereas the implications of the previous research can be associated with athletic performance and the ability to escape from life threatening situations, examining the impact of inversion can also reveal basic physiological responses. Since only three studies have investigated the responses to full seated inversion on recreational persons, further work is necessary to elucidate a greater range of physiological responses. Are the previously reported reductions in HR and BP related to changes in stroke volume (SV), cardiac output (Q), heart rate variability, and mean arterial pressure (MAP)? Are these parameters changed to a greater or lesser degree when an additional stress such as muscle fatigue is combined with inversion? Does this change when the population being studied are trained athletes in comparison to the recreational population?

A change in body postures places unfamiliar stresses on both the neuromuscular and cardiovascular (CV) systems. It has been proven that physically trained individuals have a larger CV reserve, a change in biochemical adaptations, neuromuscular function (Kenney, Wilmore and Costill, 2012), increased nervous system plasticity, reduced baseline sympathetic nervous system activity and improved autonomic regulation (Iellamo et al., 2000; Niemela et al., 2008; Pagani et al., 1988; Somers et al., 1991; Martins-Pinge, 2011; Hautala et al., 2009 and Abad et al., 2014). Can these adaptations translate into less stress on the body during head down tilt (HDT)? If true, a prior background of general aerobic training might alleviate some of the impairments reported with inversion (e.g. decreased muscle activation and force) and may be applicable to prepare those who may find themselves in these positions.

The purpose of this study was to determine whether CV (HR, SV, Q, time and frequency domains of heart rate variability, MAP) and neuromuscular (leg extension and elbow flexion muscle force, activation and fatigue) changes occur when trained individuals are inverted. Based on previous studies (Hearn et al., 2009, Paddock and Behm, 2009, Johar et al., 2013) it was hypothesized that an inverted seated position would decrease muscle force and activation and accelerate fatigue. Furthermore, if an inverted position inhibits sympathetic stimulation then it was hypothesized that CV measures such as the time and frequency domains of HR variability would also be affected.

3.2 Materials and Methods

3.2.l Participants

Twelve well-trained male athletes (mean stature = 183.4 +/- 9.7 cm, mean mass = 82.2 +/- 17.1 kg, mean age = 22.5 +/- 1.6 years) volunteered to participate in this study. Participants were recruited from predominantly endurance sport backgrounds that included the Memorial University of Newfoundland cross country running team, local soccer and track and field teams. All participants were free of physical injuries, had no previous history of hypertension or cerebral-related conditions or joint and bone problems as determined by completion of the Physical Activity Readiness Questionnaire (PAR – Q) (Health Canada, Canadian Society for Exercise Physiology, 2004). All testing was performed in the School of Human Kinetics and Recreation Applied Physiology research laboratory and participants were fully informed of the procedures, gave written informed consent and in accordance with Memorial University's Human Investigation Committee, ethical approval was obtained.

3.2.2 Experimental Design

Muscle activity was measured using surface EMG and force was measured using a strain gauge that was calibrated each morning prior to testing, this will be explained below. Initially, each participant underwent the experimental procedure in an upright position as a baseline protocol. The testing procedure was subsequently performed in upright, supine and inverted positions. In each of these positions, voluntary and evoked contractile

muscle properties of the dominant elbow flexors and knee extensors as well as CV properties (HR, HR variability, BP and predicted SV and Q) were measured. Voluntary force and activation were monitored with MVC and EMG of the elbow flexors and leg extensors. Evoked contractile properties were measured with evoked (electrical stimulation) resting and potentiated twitches. The response to fatigue was measured with a 30s MVC and compared across testing conditions.

3.2.2.1 Protocol

Participants were given an orientation session within one month prior to the first testing session. Orientation allowed the participants to become familiar with the protocol under the upright, supine and inverted positions where they performed all procedures. Participants were instructed to refrain from exercise at least twenty-four hours prior to testing and were asked not to drink caffeinated beverages, smoke or drink alcohol for at least six hours prior to testing and not eat food at least two hours prior to testing (Health Canada, Canadian Society for Exercise Physiology, 2004).

The general testing protocol is described in detail elsewhere (Hearn et al., 2009, Paddock and Behm, 2009). Briefly, all subjects were warmed up prior to testing by cycling on a cycle ergometer set at 1 kp and 70 repetitions·min^{-1} (70 Watts) for five minutes. The order of unilateral dominant arm and leg testing was randomly allocated. Both limbs were tested during each session. Each testing session began with an initial evoked twitch elicited from the rectus femoris or the biceps brachii. The maximal evoked force response

was sequentially increased in the upright position with a high–voltage stimulator (Stimulator Model DS7H+; Digitimer, Welwyn Garden City, Hertfordshire, UK) until a force plateau was achieved. Each evoked stimulation had a duration of fifty microseconds. The amplitude and duration of the muscle action potential (m-wave) were concurrently monitored with EMG electrodes. The maximum twitch force was determined when the maximum m-wave amplitude and twitch force were both achieved and this stimulation parameter was utilized for subsequent stimulation.

Each participant was instructed to perform five submaximal voluntary contractions at an effort of approximately 50% maximum to warm-up. Following a one-minute break, the subject performed two isometric MVC of the quadriceps or biceps for five seconds with a one-minute rest interval and this value was recorded. If the second MVC measurement was not within five percent of the previous MVC, a third five second MVC was performed. Following the contraction, the participant was then told to relax while a potentiated twitch was administered one second later.

The previously described protocol served as a baseline (pre-test) measurement in the upright position during each testing session. At this time, the participant would be positioned in one of the three conditions that were being tested in the inversion chair. The upright, supine and the inverted positions were assigned on a randomized basis. In any of the three positions, the participant would receive an evoked muscle twitch at the voltage and amperage previously determined. This would be followed by a one minute break; they would then perform a five second MVC of the knee extensors or elbow flexors. This

was followed one second later by a maximum potentiated evoked twitch to determine the relative potentiation. The evoked contraction was followed by a one minute rest interval, at which time the participant would be returned to a supine position if inverted or remain in a supine position if tested in supine. The participant spent approximately two-and-a-half minutes in the inverted position during the evoked and voluntary contraction protocol before returned to a supine position. After inversion, the participant returned to the supine position during the one minute break due to complaints and concerns regarding intraocular and sinus pressure which made the inverted position uncomfortable and difficult to maintain for extended periods of time. Following the one minute rest interval at the supine position, the participant was then returned to the inverted position and performed a thirty second MVC fatigue protocol (MVC 30). With the supine and upright positions the participant remained in the assigned posture for the duration of the testing. Each study participant was verbally encouraged to maximally contract against the strain gauge. The force, measured in newtons, and electromyography (EMG) readings were recorded for each of the measurements described.

The protocol was performed to determine muscle activation of the rectus femoris and the biceps brachii in each condition. The participants were provided a ten minute break to transfer equipment to the limb that had not been tested (leg extensor or elbow flexor) and performed the baseline and testing protocol under the same condition. Each session lasted approximately one hour and testing sessions were separated by twenty four to forty eight hours. Every effort was made to test each study participant at a similar time of the day to eliminate changes that may be attributable to diurnal rhythms (Figure 1).

3.2.2.2 Apparatus

Each participant underwent the testing protocol in the inversion chair (custom designed by Technical Services of Memorial University of Newfoundland, St. John's, NL) in the upright, supine and inverted position (180° from upright or fully inverted; Figures 1 and 2) (Paddock and Behm, 2009, Hearn, Cahill and Behm, 2009). Hips and knees were positioned at 90°. Each individual was securely fastened into the chair via a five-point strap with an apparatus similar to a pilot's seatbelt arrangement. Belts were securely fastened over the two shoulders and around the waist. There was a padded bar secured across the legs to maintain a 90° position of the hips and knees during the maximal contractions. While seated in the inversion chair and performing the lower limb protocol, each study participant had their right lower leg in a strap positioned across the medial and lateral malleolus and a high tension wire located perpendicular to the limb and attached to a Wheatstone Bridge configuration strain gauge (Omega Engineering Inc., LCCA 250, Don Mills, Ontario, Canada) (Figure 2). While testing the elbow flexors, the upper limb was positioned in a convex padded cuff with velcro straps that were added to the inversion chair to maintain stability and eliminate extraneous movement. There was a strap positioned to keep the arm isolated. The right forearm had a strap across the ulnar and radial styloid processes with a high-tension wire located perpendicular to the limb and attached to a Wheatstone bridge configuration strain gauge (Omega Engineering Inc., LCCA 250, Don Mills, Ontario, Canada) (Figure 3).

3.2.3. Dependent Variables

3.2.3.1 Electromyography

For the quadriceps protocol, paired EMG electrodes (Kendall Medi-Trace 100 series, Chikopee, MA) were placed on the midline of the rectus femoris. Electrodes were placed halfway between the inguinal ligament and patella tendon to the midline of the quadriceps parallel with corresponding muscle fibers on the belly of the palpable muscle, with a contact diameter of 2 cm and a center-to-center distance of approximately 3-4 cm (Behm and St-Pierre, 1997). The ground electrode was placed on the head of the tibia. Stimulating electrodes were constructed that fit around the muscle belly for each subject from aluminum foil and paper towel immersed in water and covered in conduction gel (EcoGel 200, Eco-Med Pharmaceutical Inc., Mississauga, Ontario, Canada) that measured 4-5 cm in width and were placed on each study participant in approximately the same position. Preparation of the skin for the electrodes involved removal of hair from the surface of the skin using a razor, removal of dead epithelial cells with abrasive (sand) paper and a cleansing of the skin using isopropyl alcohol swabs (Behm and St-Pierre, 1997).

For the biceps protocol, paired EMG electrodes (Kendall Medi-Trace 100 series, Chikopee, MA) were placed on the bicep brachii muscle. Electrodes were placed approximately halfway between the midline of the biceps parallel with corresponding muscle fibers on the belly of the palpable muscle, with a contact diameter of 2 cm and a center-to-center distance of approximately 3-4 cm (Behm and St-Pierre, 1997). The

ground electrode was placed on the ulnar styloid process. A stimulating pad electrode covered in conduction gel was fitted around the muscle belly of each subject (EcoGel 200, Eco-Med Pharmaceutical Inc., Mississauga, Ontario, Canada) that measured 3–4 cm in width. It was placed on each participant in approximately the same position.

EMG activity was collected during the MVCs and the fatigue protocol at 2000 Hz, amplified 1000x, with an input impedance of 2 MΩ and common mode rejection ratio of >110 dB minimum (50/60 Hz), and filtered (10–1000 Hz). During the fatigue protocol, the EMG was collected in 5-second intervals (0–5s, 5–10s, 10–15s, 15–20s, 20–25s and 25–30s) (Biopac System MEC 100 amplifier, Santa Barbara, CA) and stored on a computer (MacBook, St. John's, Newfoundland, Canada). The EMG data were processed using Fast Fourier transform (FFT) filter (10–1000 Hz) and reported as median frequencies. The EMG signal was also rectified and integrated (iEMG) by the software package over 500 milliseconds during an MVC (Paddock and Behm, 2009; Hearn, Cahill and Behm, 2009; Johar et al., 2013).

3.2.3.2 MVC force

All forces detected by the strain gauge was amplified (DA 100 and analog to digital converter via analog to digital converter MP199WSW, Biopac Systems Inc., Holliston, MA) and monitored on a computer. Data were sampled at a rate of 2000 Hz and recorded and analyzed with AcqKnowledge software (AcqKnowledge III, Biopac Systems Inc., Holliston, MA).

3.2.4 Cardiovascular measures

HR variability metrics were calculated using Kubios HRV software (v2.1) from the Department of Applied Physics (University of Eastern Finland, Kuopio, Finland) as mean variables over the 30s fatigue protocol for both postures. HR variability measures for the time domain and frequency domain variables included the distance between R-R peaks of the electrocardiogram (ECG)(ms), HR (beats/min), root mean squared of SD (RMSSD)(ms), very low frequency (VLF)(Hz), low frequency (LF)(Hz), high frequency (HF)(Hz), LF/HF ratio and the total power of the frequency domain (ms^2).

3.2.5 Statistical analysis

Data were analyzed using one- and two-way repeated measures ANOVAs (SPSS 16.0 for Windows, 2007 SPSS Inc.). Fatigue data were analyzed using a 2-way ANOVA (3x6) with factors including the three postures (upright, supine and inverted) and six measurement times (0–5s, 5–10s, 10–15s, 15–20s, 20–25s and 25–30s). A one–way ANOVA determined whether significant differences existed between pre-fatigue force measures (separate ANOVAs for evoked and voluntary measurements) in the upright and inverted positions. Differences were considered significant when the *p* values were below an alpha level of 0.05. If significant differences were detected, individual paired t-tests identified individual differences among groups. To calculate the magnitude or the clinical importance of the outcomes (Drinkwater, 2008), effect sizes (ES) Cohen d's were calculated. Cohen (Cohen, 1988) classified an ES of less than 0.2 as trivial, 0.2 - 0.41 as

small, greater than 0.41 - 0.70 as moderate and greater than 0.70 as large. Data were reported as mean ± standard deviation (SD).

3.3 Results

3.3.1 Voluntary Contractile Properties

The 5-second MVC elbow flexion force showed no significant main effect change across positions. The 5-second MVC leg extension force showed a trivial and moderate magnitude (p = 0.125; ES = 0.1 upright to inverted; 0.7 upright to supine) drop of 1.6% in mean force from the upright (342.5 ± 63. 7 N) to the inverted (336.7 ± 41. 7 N) positions as well as 18.5% decrease from the upright to the supine position (279.05 +/- 111.53 N). There were no significant differences between inverted and supine positions.

3.3.2 Evoked contractile properties

Elbow Flexion

There was a non-significant tendency with a large magnitude observed (p = .10; ES = 0.74) for an 18.6% decline in the mean elbow flexors' twitch force from the upright (26. 3 ± 6.6 N) to the inverted (21.4 ± 3.1 N) position. There were no significant changes between upright and supine positions or between inverted and supine positions.

Elbow flexor potentiated twitch values displayed a strong tendency towards significance for changes in force (p = .069) from the upright to supine and inverted positions. There

was a 35.2% (ES = 0.6) increase in the mean potentiated twitch values from upright (31.2 +/- 8.4 N) to supine (42.2 +/- 23.2 N) positions, and 11.9% decline in the mean potentiated twitch value from the upright to inverted (27.5 +/- 6.6 N) positions (ES = 0.44). There was a significant ($p<0.05$, ES = 0.98) 34.8% decrease in inverted potentiated twitch forces compared to supine forces.

Leg Extension

There was a non-significant ($p = .17$; ES = 0.76) but considerable magnitude increase of 17.5% in the mean resting twitch force in the quadriceps from the upright (75.8 ± 17.4 N) to inverted (89.1 ± 25.9 N) positions as well as a 25.4% (ES = 0.84) increase from the upright to supine (95.1 +/- 28.3 N). Potentiated twitch forces showed a significant 27.3% increase ($p = .039$; ES = 1.27) with a large magnitude from the upright (83.4 ± 17.9 N) to the inverted (106.2 ± 24.6 N) position and an even larger increase of 34.1% (ES = 1.11) from the upright to the supine (111.8 +/- 33.6 N) position. There were no significant differences between inverted and supine positions.

3.3.3 Fatigue - force relationship

Elbow flexors

The force output for the 30-second fatigue protocol significantly decreased 14.3% (moderate magnitude) over time (186.4 ± 65.8 N to 159.8 ± 56.6 N; $p<0.001$; ES = 0.4) and did not show any significant changes across the three positions.

The force output at the 20–25s interval (171.8 ± 59.3 N; $p = 0.05$;) and the 25–30s interval (159.8 ± 56.6 N; $p<0.001$) were significantly lower than the 0–5s interval (186.4± 65.8 N). There was a decrease of 7.8% (ES = 0.24) and 14.2% (ES = 0.46) from the 0–5s interval, respectively.

The ANOVA results showed statistical ($p<0.01$) differences of 19.2% and 10.4% for elbow flexion MVC force from baseline (pre-test) to post MVC30 for both postures respectively (Upright pre: 190.9 ± 55.7 N vs. post: 154.2 ± 57.5 N; Inverted pre: 176.7 ± 42.1 N vs. post: 158.2 ± 37.5 N). No MVC30 differences occurred between postures.

Knee extensors

The force output for the 30-second fatigue protocol significantly ($p = .005$) decreased 4.7% (ES = 0.16; trivial magnitude) over time (273.9 ± 78.3 N to 260.8 ± 78.9 N) and did not show significant changes across the three positions.

The force output at the 5–10s interval (283.6 ± 73.9 N) was 3.5% and 8.7% higher than the 0–5s interval (273.9 ± 78.3 N; $p = 0.018$; ES = 0.13), and the 25–30s interval (260.8 ± 78.9 N; $p = 0.067$; ES = 0.28) respectively. The 25-30s interval showed a tendency towards being lower than the 0-5s interval with a reduction in force of 8.3% between the two (ES = 0.16)

The ANOVA results showed 10.5% and 8.1% statistically significant ($p<0.05$) decreases in leg extension MVC force from baseline (pre-test) to post- MCV30 for the upright and

inverted postures (Upright 296.7 ± 78.1 N vs. 265.5 ± 62.6 N; Inverted 295.0 ± 60.5 N vs. 271.0 ± 59.7 N). Although there was a trend (p=0.10) there were trivial, 2.1% magnitude changes (ES = 0.1) for post-MVC30 force differences between postures (Upright 265.5 ± 62.6 N; Inverted 271.0 ± 59.7 N)

3.3.4 Fatigue – EMG relationship

Biceps Brachii

There were significant ($p < 0.001$) 15.1% decrements (ES = 0.9; large magnitude) with the mean median frequency over the 30s elbow flexion fatigue protocol (123.2 ± 20.4 Hz to 104.7 ± 32.3 Hz). Significant decreases were found ($p < 0.0001$) from 0–5s (123.2 ± 20.4 Hz) when compared to all time intervals: 5–10s, 10–15s, 15–20s, 20–25s and 25–30s. Decrements were 4.3%, 7.5%, 10.9%, 13.3% and 15.1%, respectively (Figure 4). There were no significant differences between the three positions over time.

Rectus femoris

There were significant ($p < 0.001$; ES = 0.8) 10.1% decrements of a large magnitude found with the rectus femoris mean median frequency over the 30s leg extension fatigue protocol (123.5 ± 15.4 N to 111. 1 ± 19.4 Hz). Significant decreases were found ($p < 0.0001$) from 0–5s (123.5 ± 15.4 Hz) when compared to all time intervals: 5–10s, 10–15s, 15–20s, 20–25s and 25–30s. Decrements were 3.6%, 5.8%, 7.8%, 7.8% and 10.1%, respectively (Figure 5). There were no significant differences between the three positions over time.

3.3.5 Cardiovascular Measures

There were no significant main effects for position for the HR during MVC elbow flexion or leg extension across the three positions. There were significant main effects for time. There were increases for HR of 38.2% and 41.5% ($p < 0.001$) with a large magnitude from baseline to MVC30 for elbow flexion (72.3 ± 18.6 to 117.0 ± 21.8 bpm; ES = 2.4) and leg extension (70.3 +/- 17.8 to 120.3 ± 22.4 bpm; ES = 2.8) protocols, respectively. There was a significant position by time interaction (large magnitudes) with the elbow extension protocol. HR showed differences ($p<0.001$) between postural conditions post-MVC30, with upright (110 ± 9.8 bpm) being 16% (ES = 1.16) higher than the inverted position (92.6±14.8 bpm), respectively. Similarly, following the leg extension fatigue protocol, HR and Q were significantly ($p<0.01$) higher for upright vs. inverted (HR=15.6%; Q=7.6%) (Figure 6). There were no significant differences between inverted and supine.

Statistical differences were also found for SV and MAP from baseline to MVC30 (Figure 7). While HR showed significant increases from baseline to MVC30 for all postural positions (Upright=33%; Inverted=25%; Supine =27%), SV showed a corresponding significant decrease (Upright=30%; Inverted=33%; Supine =31%), resulting in no statistical changes in Q. However, increases in MAP (11%, p=0.08) and Q (18%, p=0.08) approached statistical significance post-MVC30 with all positions. There were no significant changes to any time or frequency domain HR variability measures during the 30s fatigue protocols (Tables 1 and 2).

3.4 Discussion

In this study, the most important finding was that well-trained varsity athletes experienced non-significant reductions in force, fatigue and CV measures with the supine and inverted positions. However, the additional stress of a fatigue protocol revealed post-fatigue CV reductions in HR (17%), MAP (11%) and Q (18%). The previously posited rationale of an inversion-induced inhibition of sympathetic stimulation (Bosone et al., 2004, Hearn et al., 2009; Paddock and Behm, 2009; Johar et al., 2013) was not fully substantiated in this study in either the supine or inversion positions, as there were no significant changes to any time or frequency domain HR variability measures.

While previous research (Hearn, Cahill and Behm, 2009; Paddock and Behm, 2009; Johar et al., 2013) reports supine and inversion induced reductions of resting HR, this study did not reproduce those findings, hence the results do not fully support the hypothesis. The present study only found significant HR decreases after the fatigue protocol. The present study did however provide additional CV measures demonstrating that Q and MAP were diminished with inversion and supine postures, but also only after fatigue.

Q changes mirrored the fatigue-related reductions seen for HR, with differences between the inverted, supine and upright postures. These hemodynamic changes suggest that blood pooling was evident during inversion (Bosone et al., 2004) but may only have had a trivial consequence on the ability of the brain to activate the associated motorneurons before, during and following fatigue.

As the participants in the present study were trained cross country runners, soccer and track and field athletes it may be feasible that their greater aerobic capacities, improved skeletal muscle efficiency, increased minute volume, improved strength of the respiratory muscles and increased oxygen diffusion (Kenney, Wilier and Costill, 2012) may counteract and better cope with HDT induced hydrostatic and hemodynamic challenges when compared to the recreationally trained subjects considered in previous studies (Paddock and Behm, 2009; Hearn, Cahill and Behm, 2009; MacDonald et al.,1998; Koga et al., 1999).

Physiological mechanisms associated with changes in body posture include changes in hydrostatic pressure. An inability to adapt to changes in hydrostatic pressure is speculated to be related to the decline in muscle force output during inversion with the previous three studies (Hearn, Cahill and Behm, 2009; Paddock and Behm, 2009; Johar et al., 2013). During orthostasis, the body works to offset the effect of increased hydrostatic pressures and blood pooling in the periphery. Mechanisms that are involved in this process include the respiratory muscle pump (Miller et al. 2005), skeletal muscle pump (Delp and Laughlin, 1998), vestibulosympathetic reflex *(*Spyer, 1981) and veno-vasoconstriction (Vissing, Secher and Victor, 1997); ensuring adequate venous return to the heart. It is not clear if these mechanisms act efficiently or in the same manner when in the inverted or supine postures.

On a neuromuscular level, it has been hypothesized that with changes in the hydrostatic

pressure gradient, induced by HDT, there is a lower number of active cross bridges with maximum calcium activation plus or minus a reduction in the force per crossbridge (Vawda et al. 1996) responsible for the impairment in force of fatigued muscle. Research by Heinman, Stuhmer and Conti (1987) documented decreased muscle firing frequency accompanied by decreased enzymatic activity. Unfortunately there is no directly applicable human research; we therefore draw our conclusions from animal research suggesting that increased hydrostatic pressure could have adverse effects on evoked contractile properties.

Bosone et al. (2004) studied acute intracranial hypertension by inducing 10 minutes of -30 degree HDT and comparing hemodynamic responses to other sympathetic activation tests finding that HDT produces an increase in the hydrostatic loading of blood from the extremities towards the chest and head. The research suggested that during HDT, the cerebrovascular bed has increased compliance; likely the result of a decrease in sympathetic tone. Activation of the sympathetic nervous system can have an influence on neuromuscular outcomes by affecting muscle contractility (Roatta, Arendt-Nielsen and Farina, 2008), the reticular activating system, glycogen utilization and muscle tone (Martini and Nath, 2008). As previously described by Hearn, Cahill and Behm (2009) and Paddock and Behm (2009), complete head down inversion resulted in a significant reduction in BP that may be attributable to the baroreflex and decreased sympathetic activity. In this research, the lack of significant changes in time or frequency domains of HR variability suggest that sympathetic inhibition was not evident here (Kim et al., 1997; Lombardi et al., 1987). It could be argued that a longer time series of HR data are needed

for the accurate assessment of heart rate variability (Malik, 1996) and thus additional research is warranted.

Baroreceptors maintain adequate cerebral perfusion by counteracting the decrease in venous return and Q (Ponte and Purves, 1974). Charkoudian et al. (2004) found that an upright to tilted posture change increases central venous pressure, reducing sensitivity of baroreflex control and reducing sympathetic outflow. Aerobic training is associated with a reduction in BP in trained individuals and this would be indicative of an effect on the baroreflex via the autonomic nervous system (Iellamo et al., 2000; Niemela et al., 2008; Pagani et al., 1988; Somers et al., 1991). The lack of significant change in CV and neuromuscular measures prior to fatigue in the present study may indicate that trained individuals' physiological capacities (e.g. baroreceptor and vestibulosympathetic response) may be able to overcome the increased peripheral hydrostatic pressure that untrained subjects cannot.

Athletes with the previously described CV and respiratory adaptations may also be able to counteract the compensatory increase in inspiratory muscle contraction force (Aleksandrova et al. 2005), gaseous exchange (Prisk et al. 2002), lung compliance (Donina et al. 2009), pulmonary blood flow (Hillebrecht et al. 1992) and airflow impedance (Donina et al. 2009) that accompany changes in posture, resulting in no significant changes across positions in this study. If these factors were adversely at play in this study, their effect was only evident following the fatiguing protocol.

Evoked contractile properties demonstrated differing responses between the leg extensors and elbow flexors with a change in body position. Leg extensor peak and potentiated twitch forces showed a non-significant increase with a large magnitude and significant increase with a large magnitude, respectively. This is in contrast to a non-significant tendency for a reduction of the elbow flexors' peak twitch and potentiated twitch forces from the upright to inverted positions, respectively. While elbow flexors potentiated twitch forces decreased when inverted, they were elevated when changing from upright to supine.

Why the differences between the upper and lower extremities? It can be postulated that the differences in these two muscle groups may be related to changes in hydrostatic pressure, as there is a gravity dependent hydrostatic component to all intravascular changes within the human body. Limbs that are elevated above heart level will have a reduction in perfusion pressure (Nielson, 1983). Ranatunga and Geeves (1991), reported that peak tension, time to peak and twitch contraction time to half relaxation increased when the muscle fiber bundles isolated from the rat extensor digitorum longus were exposed to increased hydrostatic pressure. Work by Cattell and Edwards (1928) and Brown (1958) also found that high pressures applied directly to resting muscle increased isometric twitch tension with a reduced rate of contraction and relaxation. Unfortunately there is no directly applicable human research studying this. In an inverted position, the leg extensors would experience lower hydrostatic pressures; while the upper extremity, given its proximity to the heart, may be experiencing high hydrostatic pressures. The significantly higher supine elbow flexor potentiated twitch forces compared to inversion

may be related to lower hydrostatic pressures when in the supine position compared to inverted. Conversely, higher hydrostatic pressure would be present in the leg extensors when in a resting upright-seated posture (with legs dangling). Peripheral mechanisms such as the skeletal muscle pump (Delp and Laughlin, 1998) would not be engaged to help redistribute the venous blood volume; an increased hydrostatic pressure of the leg extensors in the upright position could have contributed to reduced evoked contractile forces when upright compared to supine and inverted.

The adaptations of the trained athlete are plentiful, as elaborated previously (Kenney, Wilmore and Costill, 2012). The literature has some evidence that chronic exercise may result in central nervous system plasticity, reduction in sympathetic activity, improvements in autonomic regulation, increased cardiac vagal modulation of HR and subsequent increased parasympathetic modulation (Martins-Pinge, 2011; Hautala et al., 2009; Abad et al., 2014). When applying these findings to inversion research, it can be speculated that the athlete population already experiences, as an effect of aerobic training, a reduction in sympathetic nervous system activity with exercise. The lack of significant findings in CV and neuromuscular measures prior to the fatigue protocol may be attributable to the fact that the baseline sympathetic response was depressed and thus, less likely to be significantly reduced with force output prior to fatigue. Essentially, the reduced autonomic response found in the athlete population may have enabled them to better counteract the sympathetic changes associated with inversion.

3.5 Limitations

Potential limitations of the present study would include the relatively modest number of solely male participants. The differences in training background and training regime could have contributed to different aerobic capabilities of the participants. We were unable to quantify this in the study. As well, considering training backgrounds and participant personalities and motivators, some participants may have been more amenable to the verbal feedback provided whilst performing a MVC. Regarding the experimental design, there was a single but extensive orientation session. It is not known if multiple and/or prolonged exposures to an inverted position would significantly alter the physiologic responses documented. As well, all experimental testing of both the upper and lower limbs was performed on the same day. Although a ten minute rest interval was allocated between upper and lower limb testing, there is conflicting research documenting non-local muscle fatigue effects in upper and lower limbs (Kennedy et al., 2013, Takahashi et al., 2011). As previously mentioned, all efforts were made to test participants at the same time of the day to account for diurnal variations, however this was not always possible. As previous elucidated, regarding the lack of significant findings in time or frequency domains of HR variability in the pre-fatigue protocol, it could be argued that a longer time series of data for accurate assessment of heart rate variability (Malik, 1996) is warranted; this may be an avenue for future research.

3.6 Conclusions

The present study demonstrated that highly trained individuals may be more impervious to the detrimental effects of physical exertion associated with supine and inverted seated position when not fatigued. The reduction in sympathetic nervous system activity in the aerobic trained population may counteract some of the pre-fatigue sympathetic changes found in previous studies. However, after a 30s MVC fatigue protocol, the trained individuals present many similar responses as untrained individuals including decreases in HR and MAP, indicating sympathetic nervous system activation. The inversion-induced decreased HR was also associated with a decreased Q but no significant change in SV. Evoked muscle contractile properties were adversely affected possibly due to blood pooling and changes in hydrostatic pressure.

3.7 References

Abad, C.C., do Nascimento, A.M., Gil, S., Kobal, R., Loturco, I., Nakamura, F.Y., Mostarda, C.T. and Irigoyen, M.C. (2014). Cardiac autonomic control in high level brazilian power and endurance track-and-field athletes. *International Journal of Sports Medicine,* 35, 772-778.

Aleksandrova, N.P., Baranov, V.M., Tikhonov, M.A., Kolesnikov, V.I., Kotov, A.N., and Kochanov, V.S. (2005) The effect of head-down hypokinesia on functional state of diaphragm in rats. Rossiiskii fiziologicheskii zhurnal imeni I. M. Sechenova, 91, 1312-1319.

Behm, D.G. and St-Pierre, D.M. (1997). Effects of fatigue duration and muscle type on voluntary and evoked contractile properties. *Journal of Applied Physiology,* 82**,** 1654-1661.

Bosone D., Ozturk V., Roatta S., Cavallini A., Tosi P., and Micieli G. (2004) Cerebral haemodynamic response to acute intracranial hypertension induced by head-down tilt. *Functional Neurology,* 19**,** 31-35.

Brown, D.E., Guthe K.F., Lawlor, H.C. and Carpenter, M.P. (1958). The pressure, temperature and ion relations of myosin ATP-ase. *Journal Cell Physiology,* 52, 59-77.

Canadian Society for Exercise Physiology (2003). *Professional Fitness and Lifestyle Consultant Resource Manual*, Ottawa: Health Canada Publishers 45-66.

Cattell, M. and Edwards, D.J. (1928b). The energy changes of skeletal muscle accompanying contraction under high pressure. *American Journal of Physiology,* 86, 371-381.

Chacon-Mikahil, M.P.T., Forti, V.A.M, Catai, A.M., Charkoudian N., Szrajer, J.S., Golfetti, R., Martins, L.E.B., Lima-Filho, E.C., Wanderley, J.S., Marin-Neto, J.A., Maciel, B.C. and Gallo-Jr, L. (1998). Cardiorespiratory adaptations induced by aerobic training in middle-aged men: the importance of a decrease in sympathetic stimulation for the contribution of dynamic exercise tachycardia. *Brazilian Journal of Medical and Biological Research,* 31, 705-712.

Charkoudian N, Martin E.A., Dinenno F.A., Eisenach J.H., Dietz N.M., and Joyner M.J. (2004). Influence of increased central venous pressure on baroreflex control of sympathetic activity in humans. *American Journal of Physiology and Heart Circulatory Physiology,* 287**,** H1658-H1662.

Cohen, J. 1988. *Statistical power analysis for the behavioral sciences,* Hillside N.J.: L. Erbraum Associates.

Delp M.D. and Laughlin M.H. (1998). Regulation of skeletal muscle perfusion during exercise. *Acta Physiologic Scandinavica,* 162**,** 411-419.

Doberentz, E. and Madea, B. (2009). Drowning in a head-down position - report of an unusual accident. *Achives of Kriminology,* 224, 108-115.

Donina, Zh.A., Danilova, G.A., and Aleksandrova, N.P. (2009). Effects of body position on the ventilatory response to hypercapnia. *European Journal of Medical Research,* 7 Suppl 4:63-66.

Drinkwater, E. J. 2008. Application of confidence limits and effect sizes in sport research. *The Open Sports Sciences Journal,* 1**,** 3-4.

Hautala, A.J., Kiviniemi, A.M., and Tulppo, M.P. (2009). Individual responses to aerobic exercise: The role of the autonomic nervous system. *Neuroscience and Biobehavioral Reviews,* 33, 107-115.

Hearn J., Cahill F., and Behm D.G. (2009). An inverted seated posture decreases elbow flexion force and muscle activation. *European Journal of Applied Physiology,* 106**,** 139-147.

Heinemann, S.H., Conti, F. Stuhmer, W. and Neher, E. (1987). Effects of hydrostatic pressure on membrane processes. Sodium channels, calcium channels, and exocytosis. *Journal of General Physiology,* 90, 765-778.

Hillbrecht, A., Schulz, H., Meyer, M, Baisch, F., Beck, L., and Blomgvist, C.G. (1992). Pulmonary responses to lower body negative pressure and fluid loading during head-down tilt bedrest. *Acta Physiologica Scandinavica Suppl.,* 604, 35-42.

Iellamo, F., Legramante, J.M., Massaro, M., Raimondi, G., and Galante, A. (2000). Effects of a residential exercise training on baroreflex sensitivity and heart rate variability in patients with coronary artery disease: a randomized, controlled study. *Circulation,* 102, 2588–2592.

Johar P., Grover V., Disanto M.C., Button D.C., and Behm D.G. (2013) A rapid rotation to An inverted seated posture inhibits muscle force, activation, heart rate and blood pressure. *European Journal of Applied Physiology,* 113, 2005-2013.

Kenney, W.L., Milmore, J.H., and Costill, D.L. (2012). *Physiology of Sport and Exercise (5th Edition).* USA: Human Kinetics.

Kennedy, A., Hug, F., Sveistrup, H. and Guevel, A. (2013). Fatiguing handgrip exercise alters maximal force-generating capacity of plantar-flexors. *European Journal of Applied Physiology,* 113**,** 559-566.

Kim, Y.H., Ahmed, M.W., Kadish, A.H. and Goldberger, J.J. (1997). Characterization of

the factors that determine the effect of sympathetic stimulation on heart rate variability. *Pace Pacing and Clinical Electrophysiology,* 20**,** 1936-1946.

Koga, I.A., McAllen, R.M., and Yates, B.J. (2000). Patterning of sympathetic nerve activity in response to vestibule stimulation. *Brain Research Bulletin,* 53, 11-16.

Lombardi, F., Sandrome, G., Pernpruner, S., Sala, R., Garimoldi, M., Cerutti, S., Baselli, G., Pagani, M. and Malliani, A. (1987). Heart-Rate-Variability as an Index of Sympathovagal Interaction after Acute Myocardial-Infarction. *American Journal of Cardiology,* 60**,** 1239-1245.

MacDonald, M.J., Shoemaker, J.K. Tschakovsky, M.E. and Hughson, R.L. (1998). Alveolar oxygen uptake and femoral artery blood flow dynamics in upright and supine leg exercise in humans. *Journal of Applied Physiology,* 85, 1622-1628.

Malik, M. (1996). Heart Rate Variability: Standards of Measurement, Physiological Interpretation, and Clinical Use. *Circulation,* 93**,** 1043-1065.

Martini, F.H. and Nath, J.L. (2008). *Fundamentals of Anatomy and Physiology,* New Jersey: Pearson Benjamin Cummings.

Martins-Pinge, M.C. (2011). Cardiovascular and autonomic modulation by the central nervous system after aerobic exercise training. *Brazilian Journal of Medical and Biological Research,* 44, 848-854.

Midgley A.W., McNaughton L.R., Polman R., and Marchant D. (2007). Criteria for determination of maximal oxygen uptake: a brief critique and recommendations for future research. *Sports Medicine,* 37**,** 1019-1028.

Miller J.D., Pegelow D.F., Jacques A.J., and Dempsey J.A. (2005). Skeletal muscle pump versus respiratory muscle pump: modulation of venous return from the locomotor limb in humans. *Journal of Physiology,* 563**,** 925-943.

Nielson, H.V. (1983). Arterial Pressure - blood flow relations during limb elevation. *Acta Physiologica Scandinavica,* 118, 405-413.

Niemela, T.H., Kiviniemi, A.M., Hautala, A.J., Salmi, J.A., Linnamo, V., and Tulppo, M.P. (2008). Recovery pattern of baroreflex sensitivity after exercise. *Medicine and Science in Sports and Exercise,* 40, 864–870.

Pagani, M., Somers, V., Furlan, R., Dell'Orto, S., Conway, J., Baselli, G., Cerutti, S., Sleight, P. and Malliana, A. (1988). Changes in autonomic regulation induced by physical training in mild hypertension. *Hypertension*, 12, 600-610.

Paddock N. and Behm D.G. (2009). The effect of an inverted body position on lower limb

muscle force and activation. *Applied Physiology Nutrition and Metabolism,* 34**,** 673-680.

Ponte J. and Purves M.J. (1974) The role of the carotid body chemoreceptors and carotid sinus baroreceptors in the control of cerebral blood vessels. *Journal of Physiology,* 237**,** 315-340.

Prisk G.K., Fine J.M., Elliott A.R., and West J.B. (2002). Effect of 6 degrees head-down tilt on cardiopulmonary function: comparison with microgravity. *Aviation Space and Environmental Medicine,* 73**,** 8-16.

Ranatunga, K.W. and Geeves, M.A. (1991). Changes produced by increased hydrostatic pressure in isometric contractions of rat fast muscle. *Journal of Physiology,* 441, 423–431.

Roatta S., Rendt-Nielsen L., and Farina D. (2008). Sympathetic-induced changes in discharge rate and spike-triggered average twitch torque of low-threshold motor units in humans. *Journal of Physiology,* 586, 5561-5574.

Somers, V.K., Conway, J., Johnston, J., and Sleight, P. (1991). Effects of endurance training on baroreflex sensitivity and blood pressure in borderline hypertension. *Lancet,* 337, 1363–1368.

Spyer, K.M. (1981). Neural organization and control of the baroreceptor reflex. *Reviews of Physiology, Biochemistry and Pharmacology,* 88, 24-124.

Takahashi, L., Maruyama, A., Hirakoba, K., Maeda, M., Etoh, S., Kawahira, K. and Rothwell. J.C. (2011). Fatiguing intermittent lower limb exercise influences corticospinal and corticocortical excitability in the nonexercised upper limb. *Brain Stimulation,* 4**,** 90-96.

Vawda, F., Ranatunga, K.W. and Geeves, M.A. (1996). Effects of hydrostatic pressure on fatiguing frog muscle fibers. *Journal of Muscle Research and Cell Mobility,* 17, 631-636

Vissing, S.F., Secher, N.H., and Victor, R.G. (1997). Mechanisms of cutaneous vasoconstriction during upright posture. *Acta Physiologica Scandinavica* 159, 131-138.

3.8 Appendices

FIGURE 1 - Experimental Protocol

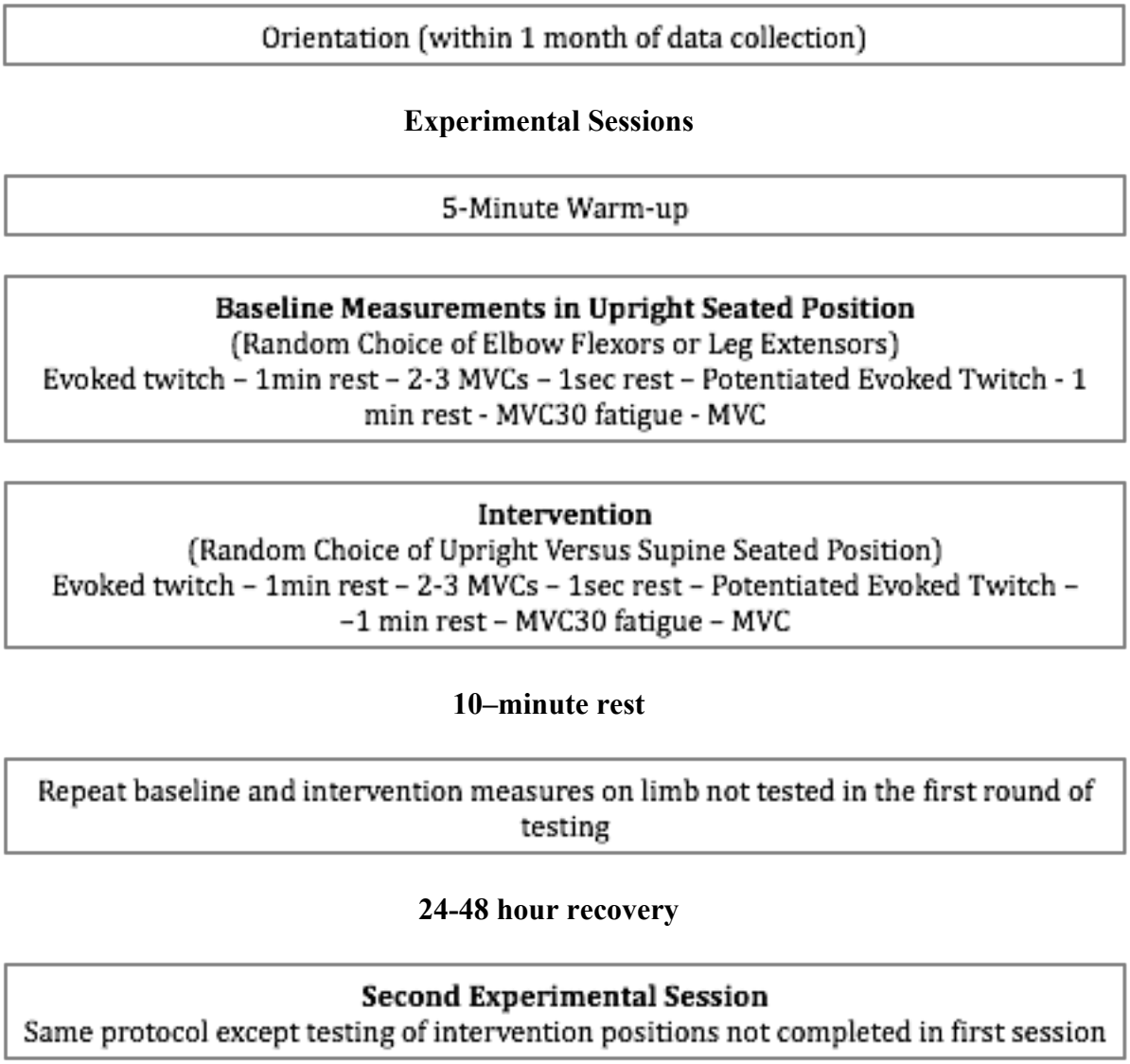
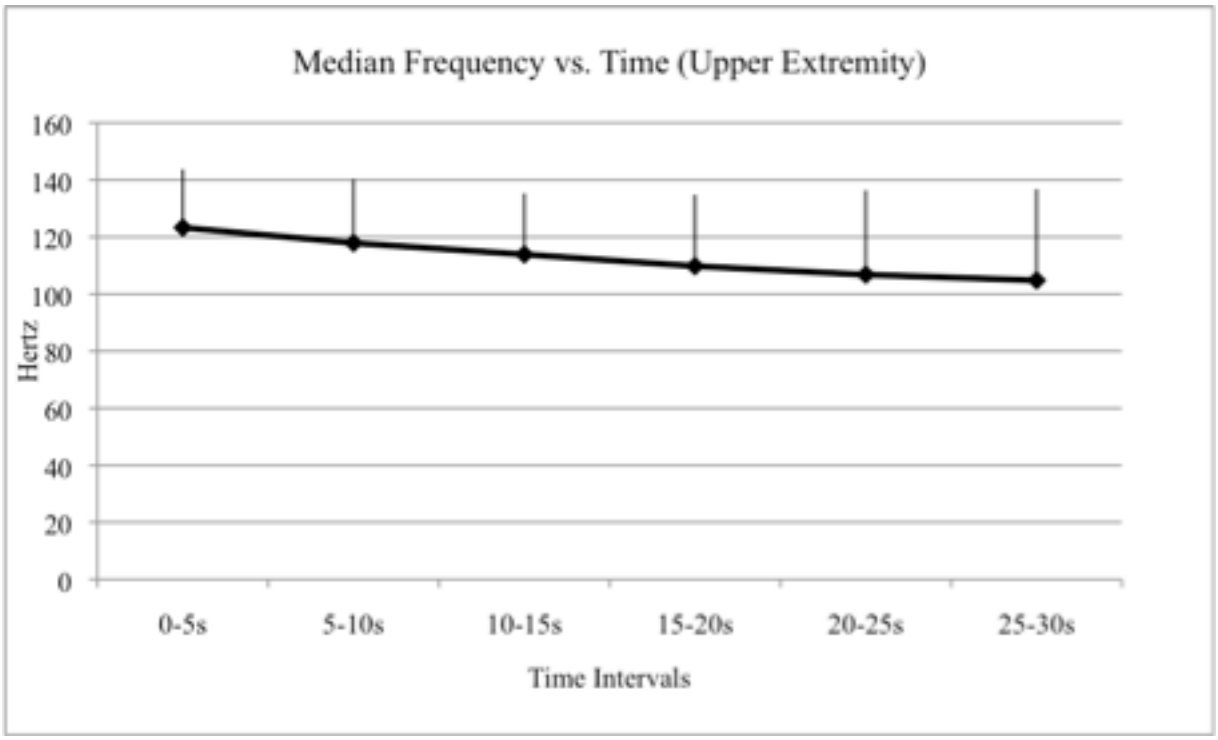


FIGURE 2 – Inversion chair upright position with subject prepared for lower limb protocol

FIGURE 3 – Inversion chair inverted position with subject performing upper limb protocol

FIGURE 4 - Force - EMG relationship in the biceps brachii

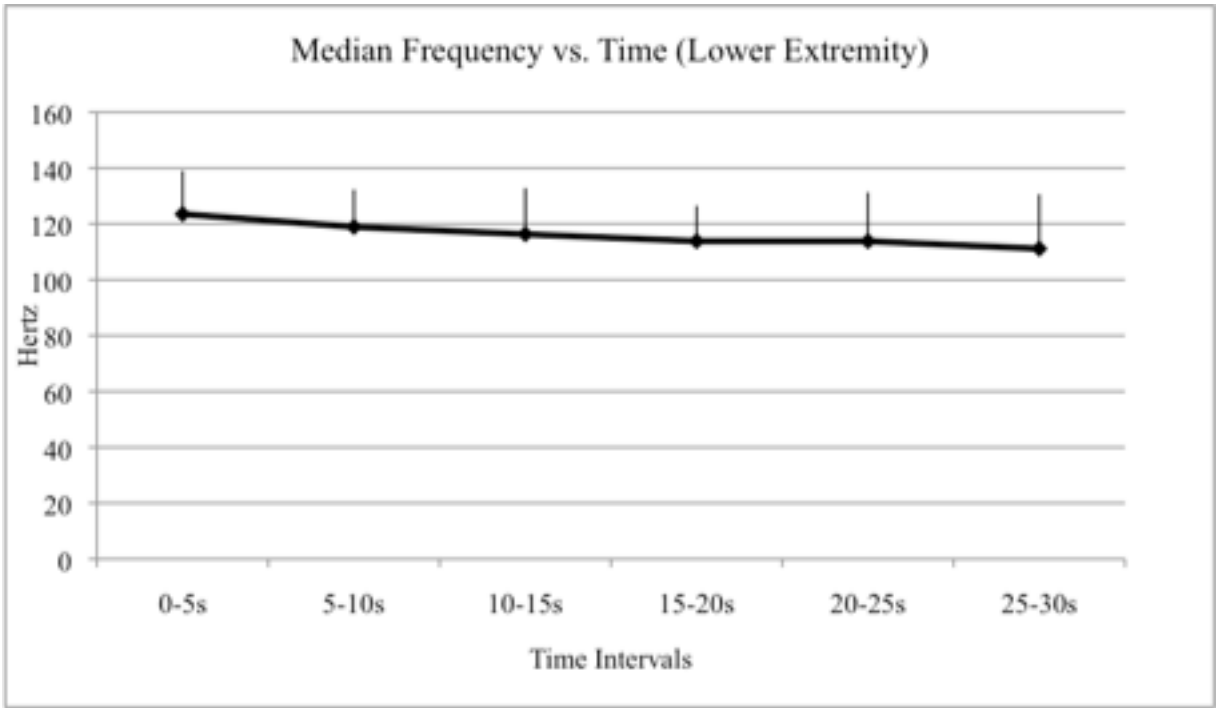
Mean median frequency (Hz) EMG and standard deviations for the biceps brachii/upper extremity (UE) during 5-second epochs of the 30 second fatigue protocol (MVC30) provided in mean Hz for the positions.



Significant decrements in mean median frequency ($p < 0.0001$) were found from 0–5s (123.2 ± 20.4 Hz) across all time intervals

FIGURE 5 - Force - EMG relationship in the rectus femoris

Mean median frequency (Hz) EMG and standard deviations for the rectus femoris/lower extremity (LE) during 5-second epochs of the 30 second fatigue protocol (MVC30) provided in mean Hz for the positions.



Significant decrements in mean median frequency ($p < 0.0001$) were found from 0–5s (123.5 ± 15.5 Hz) across all time intervals

FIGURE 6 - Cardiac output (Q) for each postural condition illustrating changes in Baseline vs. MVC30. Paired letters (a,a; b,b) indicate significant differences between postures for MVC30. Asterisks (*) indicates significant differences between postures at p=0.08.

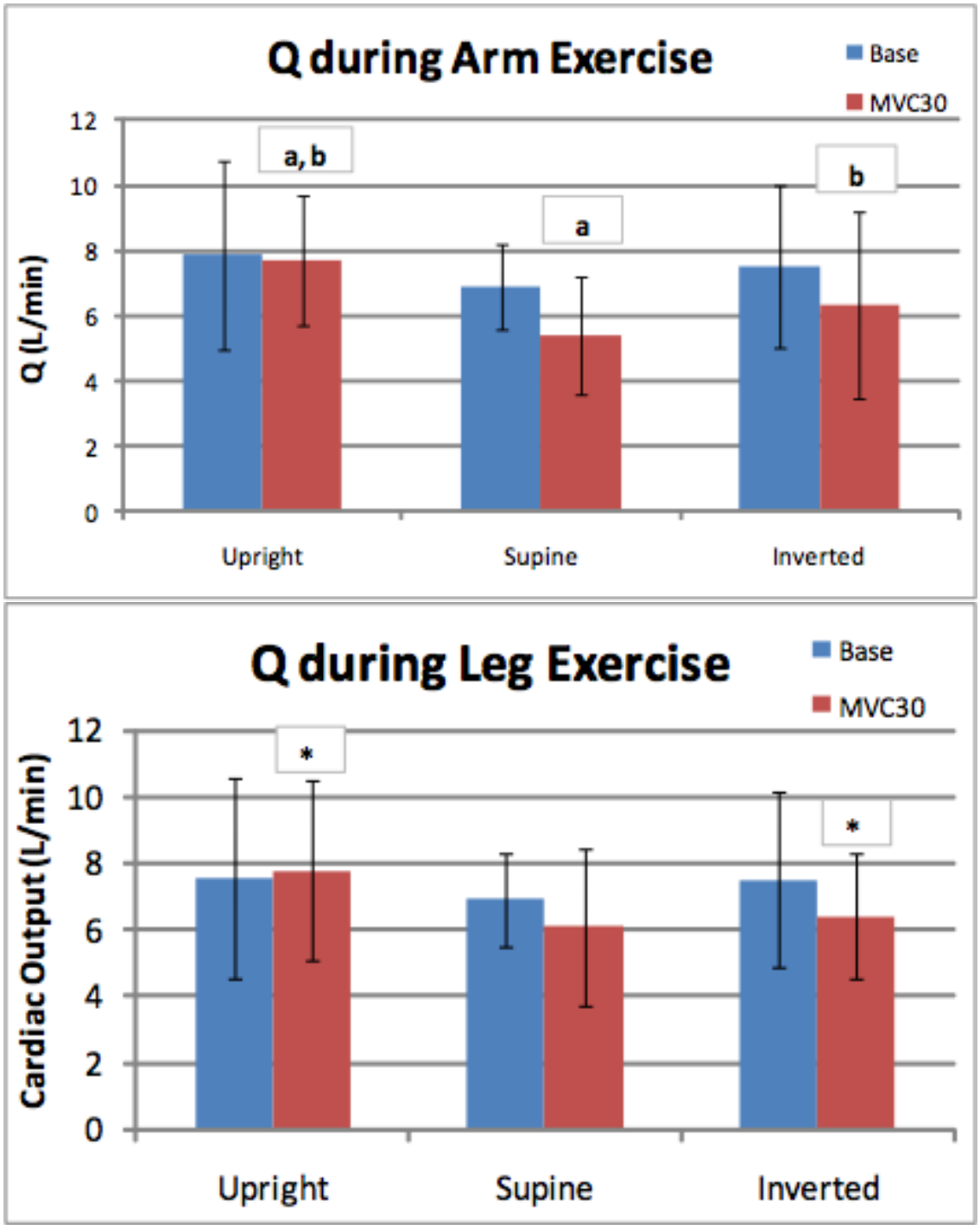


FIGURE 7 - Mean arterial pressure (MAP) for each postural condition illustrating changes in Baseline vs. MVC30. Asterisks (*) indicates significant differences between postures at p=0.08.

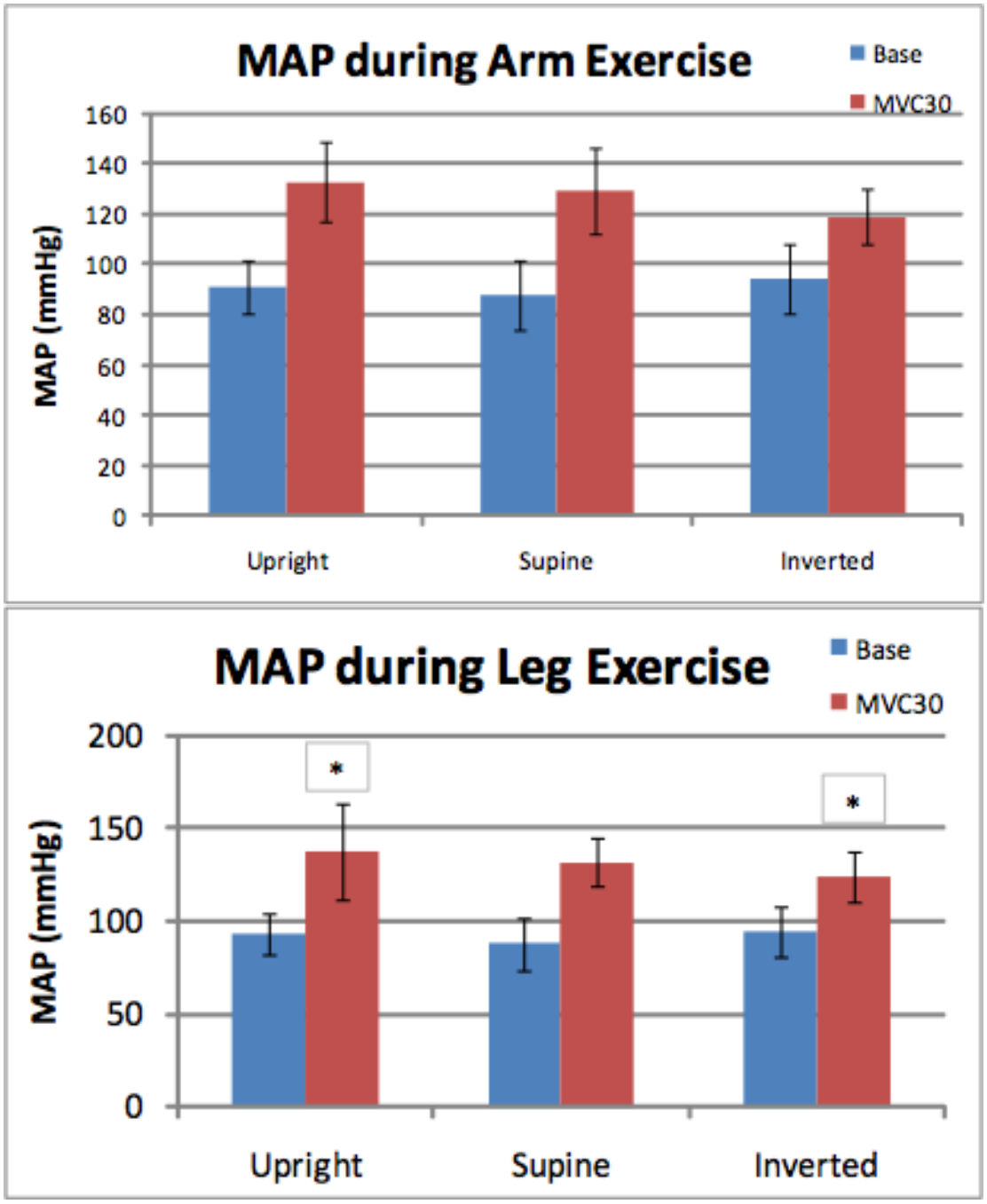


TABLE 1 - Heart rate variability results for the time domain and frequency domain variables. R-R = distance between R-R peaks of ECG (ms), HR= heart rate (beats/min), RMSSD = root mean squared of SD (ms), VLF = very low frequency (Hz), LF= low frequency (Hz), HF (Hz), LF/HF = ratio between LF and HF, and Total Power of the frequency domain (ms^2). Values are mean ± SD. No significant differences between conditions at $p<0.05$.

Time Domain	**R-R (ms)**	**HR (bpm)**	**RMS**
Upright	855.34±170.3	72.7±5.5	40.7±26.5
Inverted	912.49±152.9	67.9±4.8	58.7±37.2

Frequency Domain

(Peak Hz)	**VLF**	**LF**	**HF**	**LF/HF**
Upright	0.0223±0.0138	0.067 ± 0.022	0.201±0.054	7.76±7.5
Inverted	0.0296±0.01	0.092±0.036	0.23±0.055	2.7±2.2

TABLE 2 - Cardiovascular measures from baseline to post-MVC30

Heart Rate (HR: bpm)	HR Elbow Flexion Upright	HR Elbow Flexion Inverted	HR Leg Extension Upright	HR Leg Extension Inverted
Pre-MVC30	74.6 ± 19.3	71.0 ± 17.9	72.3 ± 19.4	68.3 ± 15.4
Post-MVC30	120.4 ± 22.8	113.6 ± 20.8	110.5 ± 15.3	95.6 ± 13.7
Cardiac Output (Q: $l.min^{-1}$)	Q Elbow Flexion Upright	Q Elbow Flexion Inverted	Q Leg Extension Upright	Q Leg Extension Inverted
Pre-MVC30	7.8 ± 2.1	7.7 ± 3.4	7.8 ± 3.1	7.75 ± 3.4
Post-MVC30	7.9 ± 2.9	6.1 ± 2.7	7.9 ± 3.3	6.2 ± 2.8
Stroke Volume (SV: ml)	SV Elbow Flexion Upright	SV Elbow Flexion Inverted	SV Leg Extension Upright	SV Leg Extension Inverted
Pre-MVC30	101.3 ± 39.8	106.8 ± 43.2	101.9 ± 35.4	107.6 ± 40.8
Post-MVC30	67.4 ± 24.3	69.7 ± 29.4	73.6 ± 31.1	72.9 ± 33.4
Mean Arterial Pressure (MAP: mmHg)	MAP Elbow Flexion Upright	MAP Elbow Flexion Inverted	MAP Leg Extension Upright	MAP Leg Extension Inverted
Pre-MVC30	94.2 ± 27.6	96.1 ± 32.4	92.3 ± 29.9	96.4 ± 26.5
Post-MVC30	137.9 ± 41.3	120.5 ± 39.6	139.6 ± 37.8	121.7 ± 38.1