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Harness Suspension Stress Physiological and Safety Assessment

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DISSERTATION

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Abstract

Background: No robust trials including imaging or biomarkers have been performed to evaluate the possible stress of hanging motionless in a harness (harness suspension stress - HSS). Untoward effects are from case reports and sparsely documented data, leaving the topic open to debate. No cause-effect relationship has been established, therefore providing no guide to diagnostic, prognostic, or therapeutic evaluation or treatment for possible detrimental effects of HSS. Expert opinion provides the majority of the literature concerning HSS. Many of the effects of HSS have been correlated to other syndromes or causes, but none have been excluded, and none have been established. **Methods:** Human biomarkers from blood, fitness levels, subjective discomfort levels, and sonographic measurements were obtained to help gain a better understanding of the effects of hanging motionless in a full body harness for thirty minutes by both front and dorsal points of attachment. **Results:** Full body harnesses are not well tolerated. Hanging in a harness for

extended periods leads to pain, discomfort, and anxiety. No changes in blood laboratory data, fitness level, or gender, accounts for potential bradycardic events or vasovagal syncope.

Conclusions: Industrial full body harnesses designs appear to be generally poor and cause enough discomfort to augment human homeostasis in some subjects. We find no evidence to support more than standard recommendations for Advanced Cardiac Life Support guidelines. Further research is needed to help guide understanding of what may or may not constitute a credible etiology during HSS, but also to guide medical response, if any is indicated.

Dedication

In memoriam of Steve Hudson, who continually probed for better answers to hard questions that addressed real problems for those involved in the technical rope rescue discipline. This is also dedicated to those who fight against the odds, on whatever scale, but choose to make a difference. I would like to thank Dr. Mermier for her support and encouragement to continue to follow my course of study, and for being, in large part, the reason that I came to the Exercise Science program. Her guidance and generous mentorship through the coursework over the years has been the backbone of my success. I appreciate her love for the outdoors, her fervor to answer difficult questions, and her dedication to not only the student, but also the person, that are one and the same. Thank You for always being there, even through difficult times. I would like to thank Steve Attaway, PhD, whom, without his support and mentorship I would never have chosen the path of continual learning. I have walked the line of peril with Dr. Attaway while serving on rescues in New Mexico, helping those in the wilderness and austere environments. We have performed multiple studies on ice anchors and technical rope rescue issues to help increase safety and efficiency among the technical rope rescue community. Certainly, I could not have had this opportunity without you. It's an honor to have this opportunity to work closely with you. Thank You!

We stand on the shoulders of the giants who helped mentor us and make us who we are. I am fortunate enough to have many of those mentors in my climbing and guiding career, my medical career, and my doctoral pursuit.

Thank You.

Acknowledgments

I would like to thank the University of New Mexico Department of Emergency Medicine

for their assistance in this work, as their insight and contribution is significant. I would

also like to thank the human test subject volunteers for their time and willingness to

participate in our study. Countless hours were spent by the co-authors and subjects to put

the research together to help give insight into a field not supported otherwise for intense

research such as this. All authors and contributors declare no conflict of interests.

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SYMBOLS / ABBREVIATIONS

$\geq$: greater than or equal to

$>$: greater than

$\leq$: less than or equal to

$<$: less than

$\pm$: plus or minus

$\sim$: approximately

$°C$: degrees Celsius

ml: milliliter

α: alpha

ANOVA: analysis of variance

AG: ankle girth

ALT: alanine transaminase

ALP: alkaline phosphatase

AST: aspartate amino transferase

ATP: adenosine triphosphate

ACLS: Advanced Cardiac Life Support

% fat: body fat percentage

bpm: beats per minute

BUN: blood, urine, nitrogen

BW: body weight

C++: Calcium

CBC: complete blood count

CInd: cardiac index

CK: creatine kinase

CO: cardiac output

Cr: creatinine

cm: centimeters

CMP: complete metabolic panel

D: dorsal

DBP: diastolic blood pressure

DP: lower extremity pulse quality

DpcMAPmax: Dorsal percent change mean arterial pressure maximum

ECHO: echocardiogram: myocardial sonogram

EKG: electrocardiogram

EF: ejection fraction

F: frontal

g: gram

Glu: glucose

HR: heart rate

HSS: harness suspension stress

ICG: impedance cardiography

IVCi: inferior vena cava upon inspiration

IVCexp: inferior vena cava upon expiration

K+: postassium

kg: kilogram

LA: lactate

LAL: limulus amebocyte assay

LPS: lipopolysaccharide

MAP: mean arterial pressure

mg: milligram

MV: mitral valve

n: number of subjects

Na+: sodium

NaCl: sodium chloride

PLA: placebo

RR: respiratory rate

PP: pulse pressure

plt: platelet

r^2: coefficient of determination

RM ANOVA: repeated measures analysis of variance

RPE: rating of perceived exertion or discomfort

SBP: systolic blood pressure

SD: standard deviation

SE: standard error

SpO2: percentage of oxygen

SV: stroke volume

SVR: systemic vascular resistance

USG: urine specific gravity

VO_{2max}: maximal oxygen consumption

VO_2 peak: peak oxygen consumption

CHAPTER 1

Introduction

Harness Suspension Stress (HSS) is defined by what is also known as suspension trauma, harness hang syndrome, harness induced trauma, harness induced pathology, or other terms. The idea that free hanging in a climbing or industrial harness can have an untoward outcome is a relatively new concept that has come into the literature through limited testing, case reports, and expert opinion. [1, 2, 3, 4, 5] Anyone using a harness, whether it is a recreational climbing harness with a frontal waist point of attachment, such as those used in rock and ice climbers, or an industrial-style harness with a dorsal or chest point of attachment used by construction workers, have been thought to expose users to undue risk by simply hanging in their harness. However, after nearly 15 years after the Seddon report which described a warning about the possibility of the adverse effects of hanging in a harness, no deaths have been directly attributed to hanging in a harness. [6] Although articles have introduced the possible existence of the existence of HSS, more robust studies have been called for in order to examine real or theoretical risk. [3, 2, 7]

Two pilot studies have been performed in an effort to gain not only better insight and information, but also to refine techniques for a more complete yet baseline study, into the effects of hanging motionless in a harness and what possible physiological effects may occur. The insights will help with future investigations.

Study Purpose and Hypotheses

The purpose of this research is to determine if suspending subjects in a harness for thirty minutes shows any difference in biomarkers and perception of comfort from

baseline. Our pilot research showed no indication that hanging in a harness causes any more than irritation and slight discomfort.

Aims of the Study

1)   Gain insight into potential factors that cause biomarkers to shift from baseline as a function of hanging in a harness.

2)   Investigate any possible focus for further testing and evaluation that may create exclusion criteria for those who may be exposed to hanging in a harness.

3)   Develop a more definitive definition for harness suspension stress.

4)   Exclude other pathologic etiologies that have been associated with HSS.

5)   Assess the possibility that hanging in a harness is not a cause for an emergent response in and of itself, but that other extraneous factors should be considered first.

Hypothesis

We hypothesize that:

1)   There will be no shifts from baseline biomarkers that are measurable by clinicians after hanging in a harness for thirty minutes.

2)   Laboratory markers of physiological stress will differ for those experiencing HSS depending on the type of suspension harness.

3)   Laboratory markers of physiological stress will differ for those experiencing HSS depending on the degree of individual fitness.

4)   There will be an interaction between degree of fitness and type of harness in physiologic responses to HSS

Scope of the Study

This research will be the first study to measure biomarkers during human trials while hanging in a harness. It has the potential to drive further research, if indicated, in the rope rescue and rope access industries relating to anyone who hangs in a harness for any purpose.

This study will also help guide emergency responders and clinicians in understanding what modalities may or may not be warranted in regards to possible medical or emergent therapies for those hanging in a harness for thirty minutes.

Limitations

This research cannot address every instance, environmental situation, body habitus, race, genetic trait, medical ailment, or a multitude of other circumstantial issues. The age group and demographic with which we were able to perform this study is somewhat limited. We hope that the limitations of our research will point towards other areas that may need to be evaluated, or may be the source of an end-investigation. Non-invasive studies such as these only present a window of insight.

Environmental conditions for climbers and workers vary around the world in temperature, humidity, ambient air pressure, and a host of other factors. The human body behaves differently under the myriad of conditions that exist. Blood viscosity, evaporative sweating, and heart rate are only a few of the body functions that are constantly changing and adjusting in response to a given environment. Controlling the environment for the sake of the study was of paramount concern, so that the data are comparable. Although it would be ideal to test subjects under multiple conditions, this was not feasible.

The mapping and diagnosing of subjects by genetic testing is becoming more commonplace in the medical world, potentially picking up cardiac disease markers, Parkinsonian markers, and many others. Therefore, it may be that some subjects are more prone to unforeseen complications when hanging in a harness than others, simply based on their genetics. Body habitus may be attributed to genetic traits to a certain degree, but we cannot account for all body types in our sample. Race sometimes places a role in physical abilities or limitations for certain sports or tasks. Although our sample was from a diverse background, the sample will not be able to be extrapolated to all populations.

Known previous medical ailments that could potentially restrict someone from physical activities, such as working in a harness, were excluded from this study.

Age is also of importance. However, the Institutional Review Board, due to increased risk of cardiovascular disease, restricts maximal exercise testing on subjects forty-five years of age or greater. However, we tested subjects between 18-40 years of age, and are somewhat representative of the population who would be the most expected to be exposed to HSS. There are many people greater than forty years old, who are actively using full body harnesses in an occupation that mandates the use of a harness either as fall protection or for rope access,

Overall, despite the limitations of the research study design, it is clear that the subjects available will represent a majority of the population at large who uses harnesses, and are exposed to potential HSS.

Assumptions

The effect of randomization in the trials assumes that our samples represent a normal distribution of those who utilize full body harnesses. With the small sample size

and the limitations of not testing all age groups, it is reasonable to say that our research can only represent a smaller population. However, this is generally the population who is exposed to fall hazard while working at height and with a potential to be suspended in a full body harness for any appreciable period of time.

We also assume that our techniques follow standards and normal practices in obtaining and analyzing our data, as well as the practical configurations for which we are testing. The harnesses that we use are from actual industry manufacturers and are tested in the configuration recommended by those manufacturers.

Significance of the Study

This research protocol has the potential to reveal further investigative leads to the effects of HSS by excluding popular syndromes or other reported manifestations that have been closely correlated only by expert opinion. The study will pertain to all industry manufacturers and vendors providing goods or services such as harnesses, or training, respectively, and has a direct effect to anyone who hangs in a harness for any purpose.

This research may guide emergency responders and clinicians in understanding what the modalities may or may not be in regards to possible medical or emergent therapies for those hanging in a harness for thirty minutes.

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CHAPTER 2

Harness Suspension Stress, Narrowing the Focus

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Abstract

Harness Suspension Stress (HSS) is defined as the physiological stress resulting from hanging motionless in a harness for a length of time. HSS may produce pain in the legs, numbness, syncope, and has been the subject of debate without much clinical data to support the physiologic explanation for these clinical features. HSS has been reported loosely in peer-reviewed literature. Further, one's predisposition of developing HSS, or subsequent medical ramifications requiring therapy has not been well evaluated. Our knowledge of HSS to this point has been derived mostly from expert opinion and case reports over the last 50 years. A rise in manufacturer development of fall protection equipment, including the use of harnesses, has resulted in increased regulative preventative measures, rescue techniques, and postulations for medical care. Other syndromes have been associated with the effects of HSS, but the constellation of symptoms reported for HSS are inconsistent with any other set of well established existing medical syndromes, leaving a gap in understanding of the overall etiology and pathogenesis of HSS. Treatment of HSS should only be based on sound, evidence-based medicine and high level of experimental research. This review aims to examine possible factors that may help qualify or quantify a series of measurable signs or symptoms that may establish HSS as its own syndrome, or if pre-dispositional factors may play a role that could be of clinical or practical use.

Introduction

Harness Suspension Stress (HSS) is also known as suspension trauma, harness hang syndrome, harness induced trauma, or harness induced pathology. The theory that free hanging in a climbing or industrial harness can have an adverse outcome is a concept

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that has been described through case reports and expert opinion [1-3]. Simply hanging in a harness, whether it is a recreational climbing harness (waist point of attachment), such as those used by rock and ice climbers, or an industrial-style harness which may use a dorsal (between the scapula) (Figure 1), or frontal chest point of attachment (Figure 2) as used by construction, industrial, or rescue workers has been thought to expose users to undue risk.

The first reports of HSS were documented during parachute testing in 1968 where four out of five observed subjects experienced only minor discomfort within thirty minutes. One subject experienced loss of consciousness for unknown reasons, but was revived quickly and without sequellae. [1] Limited studies on harness hanging were performed using various harnesses in France during the early 1980s, and researchers established that ventral and thoracic belts that only utilized the thorax should not be applied because of medically adverse effects such as restriction of the lungs. [1,4] Other tests were performed using mountaineering-style harnesses and techniques, such as the use of a rope to create a crude waist harness, to more extensive modern full-body harness designs. These studies concluded that symptoms occurred within twenty minutes regardless of harness type.[1] Of the research conducted in the past, many harness types have been shown to be potentially harmful. [1,5] The purpose of this review is to examine previous reports of HSS, discuss the possible mechanisms of adverse events during harness suspension, and to consider current guidelines in treatments for HSS.

The physiological response to harness suspension stress (HSS)

Being suspended in an upright position for a prolonged period of time spans centuries, specifically in reference to crucifixion for which we can find reports previous

to the crucifixion of Christ. [6] The act of crucifixion leading to death may take anywhere from 4 hours to many days, where shorter time is attributed to hemorrhage, exposure, and primary trauma rather than to hanging. [6,7] There are several theories that seek to explain the processes that cause death by crucifixion [8], however, it is unknown whether any of these theories can be used to explain symptoms reported while hanging in a harness.

Although reports of HSS agree that symptoms may or may not develop, there is no agreement on the pathophysiology of these changes. Several possible mechanisms have been postulated and will be discussed. The best approach for treatment for a subject who may be suffering from HSS is a topic of debate, but the evidence is lacking on the actual mechanism(s) to be treated. [1]

Most accounts of HSS report assumptions of venous pooling. It is the venous pooling effect that is thought to lead to subsequent decreased cardiac blood return, a diminished cellular perfusion, and possible syncope. [1-5,9,10] Although venous pooling has been cited, venous capacitance while hanging motionless in a harness has never been directly measured. [4] Only vital signs, subjective reports, and electrocardiograms (EKG) performed in previous studies. The one study using an EKG reported that the tracings were un-interpretable for undisclosed reasons. [5] Variable heart rates have been reported with and without symptoms, further complicating the clinical picture.

Several physiological mechanisms that seek to explain changes that occur while hanging in a harness have been proposed and will be discussed. [4,11,12] These mechanisms have been presented in review articles, magazines, journal articles, and as opinion on the Internet, with no actual quantitative measurements reported during testing. This leads to a hypothetical basis from which to center further investigation or treatment modalities.

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Compression syndrome, crush syndrome, compartment syndrome, tourniquet syndrome, and orthostatic hypotension syndrome, have been associated with HSS. [1-4] These syndromes have their own set of criteria, and perhaps some degree of crossover with HSS, but this has yet to be evaluated or established. The association of other syndromes has been inferential in an attempt to lump HSS into the same physiological processes as well as to justify potential treatment algorithms for emergency responders and medical providers. The inference of a cause-effect relationship has established potential therapeutic regimens. [11]

Compression syndrome generally refers to ischemia-reperfusion injury after prolonged limb compression during surgery, or excessive pressure applied to the limb for an extended period of time. Compartment syndrome is defined as increased tissue pressure buildup within a non-expandable fascia, which may impede circulation and nerve impulse transmission. [13] Those who sustain long periods of pressure to a limb may develop compartment syndrome in the extremity, especially following alcohol or drug intoxication, or during surgery. [14] The proposed mechanism of HSS exacerbation was postulated to stem from the idea that the harness leg loops caused a tourniquet effect, thereby cutting off circulation to the lower extremities. Neither compression nor compartment syndromes have ever been shown to exist in any of the studies or literature reviewed in regards to HSS.

Shy-Drager syndrome is a form of autonomic dysfunction and may be a neurohormonally mediated. [15,16] Genetic testing for copy loss of gene SHC2 that causes Shy-Drager syndrome can be isolated. [17] Although Shy-Drager syndrome may appear to be related to the pathophysiology postulated of HSS, there is no evidence of HSS and

Shy-Drager being related structurally as a neurohormonally mediated process. The symptoms and demographic onset of Shy-Drager are quite different than those found with previous small studies of HSS, however, it cannot be ruled out that a neurohormonal etiology is at play.

Venous pooling is a favored cause of HSS, though never directly measured during testing. Venous pooling may also be contrasted to a diminished arterial or venous flow. [18] Significant venous pooling can lead to syncope. [19] There are multiple forms of reflex syncope, including neurocardiogenic syncope (NCS), carotid sinus hypersensitivity (CSH), postural orthostatic tachycardia syncope (POTS), and joint hypermobility syncope (JHS). [20] Both NCS and CSH are considered vasovagal in origin. NCS is found to occur in the younger population and has similar symptoms as HSS, including a similar profile of a prodrome of lightheadedness, diaphoresis, and nausea with a sudden onset of syncope that is easily reversed. [21] The logic follows that arteriole collapse occurs when capillary pressure becomes greater than arteriole pressure. [22] The resultant poor venous return may be secondary to venous pooling, leading to an exaggerated increase in cardiac response, and overload of neurological stimulation to the brain that causes the paradoxical decline in sympathetic activity. [23] None of these neural pathways have been studied in association with HSS.

Crush syndrome has also been associated to HSS through the weight of the body on the harness leg straps. [18] Crush syndrome usually occurs when a relatively heavy mass or large pressure is exerted on localized tissue and subsequently released. This may result in release of destroyed tissue that returns toxins into the blood stream that may render systemic ramifications such as kidney dysfunction or rhabdomyolysis. [24] No laboratory

testing has been performed demonstrating potassium release or creatinine kinase increase, markers for crush syndrome, after HSS exposure.

Maneuvers Proposed to Prevent HSS

Public fear of the possible consequences of hanging in a harness has launched a manufacturing HSS prevention market and has created speculative expertise on pharmacological therapies to treat suspected effects of HSS. Harness suspension loops and straps have been marketed and sold, reporting that they can treat or offset the effects of HSS. However, incomplete investigation of how these devices work on a biochemical or physiological level remains.

Pharmacotherapy for treating HSS has been suggested to emergency medical technicians and hospital providers to provide for IV bicarbonate, calcium chloride, albuterol, dextrose 50%, insulin, or IV fluid therapy, as they might for suspected crush injury, even though there is no evidence to support any of these treatments as they have merely been extrapolated from the treatment of rhabdomyolysis. [12] Furthermore, there is no direct evidence that HSS has any relationship to electrolyte imbalances or fluid shifts including diminished cardiac return or venous pooling. Yet, treatment regimens have postulated a direct cause-effect relationship that must be treated as referenced somewhere within the literature or on-line information. [1,4,9,11,25-28] This causes confusion for medical practitioners and rescue personnel.

The most recent literature review on HSS found no rationale to support previous treatment therapies for treating HSS or its sequellae. [27,29] The authors' position, and the current standard, is summed up in their stance that there is little information in the literature to cite a cause-effect etiology for symptoms. They support the notion that there

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may be a neurally mediated response to HSS that may be due to harness type and configuration, and that there may be some degree of redistributive hypovolemia, but that published data is inconclusive. [10,30]

Surprisingly, no confirmed cases have been reported of "death by harness" since the first formalized literature was published, even given over seven million man hours of work on-rope with use of a harness annually world-wide, so little empirical evidence exists in regards to treatment of HSS. Only four reportable injuries, one of which was a fatality not attributed to hanging in a harness, occurred among all levels of professionals working on-rope, according to the Industrial Rope Access Trade Association in 2013. [31] The reported harness accident was caused because the victim fell without being attached to an anchor and never had the opportunity to hang in his harness. [31]

Markers of HSS

The only markers of prodromal HSS that are initially detected are the signs and symptoms of a subject that precede such an event, but up to this point are only subjective clinically. It would be of great benefit to understand the possible contributing factors that may lead to HSS symptoms, but none have been identified or correlated to any body habitus, fed or hydrated state, specific anatomy, sex, race, altitude, or any other possible contributing factor. There is no consistency in the original research from which to draw pre-defining markers for which could be associative or confounding to HSS.

Hypovolemia, vasovagal stimulation, embolism, rhabdomyolysis, hyperkalemia, lactic acidosis, and lysed red blood cells are among the other plethora of etiologies that are thought to be attributable to HSS. [25] No direct cause-effect marker has been associated with hanging in a harness. Autopsies performed on individuals who died while

hanging in a harness have not clearly shown any of these conditions to be the direct cause of death.

The Undue Stress of HSS

There is no certainty about how long can one safely hang in a harness. Some are able to hang in a harness for many hours, while others are unable to tolerate the discomfort of hanging in a harness for brief moments. The variability of pain threshold, anxiety level, and other un-quantifiable measurable markers such as potential pre-existing medical conditions, harness fit and design, or other subtle factors may be of paramount influence or concern for those participating in activities with potential to HSS exposure.

The human body's performance while hanging in a harness is inextricably linked to present harness technology available as harnesses are created for the masses, but may not be anthropometrically sensitive. It is not only the ability to find the human susceptibilities that may be a predisposition to symptoms, but also the responsiveness of the manufacturers of harnesses to understand these frailties and address them.

Conclusion

The pathophysiology of HSS remains unclear. It appears to be a distinct clinical syndrome, perhaps with some overlapping features as those seen in orthostatic hypotension syndrome, crush syndrome, compression syndrome, and compartment syndrome.

Treatment of HSS should only be based on sound, evidence-based medicine and high level of experimental research. It is not surprising that there is confusion as to what the actual pathophysiological insult may be, if any, which complicates further what to do if this situation is indeed more than theoretical. Without a basis from which to treat,

treatment would be little more than guessing. There remains no solid rationale for treatment of those thought to be experiencing HSS, and further research should be performed. [4] Clinical treatment should be based on findings rather than speculation.

No deaths have been directly attributed to hanging in a harness, although articles have introduced the possibility of HSS as a contributing factor in the demise. More robust studies have been called for in order to examine real risk or theoretical risk. [25,26] Further non-research based articles and information on the Internet will continue to obstruct any progress to a realized understanding of the issue. We call for further formal investigation before hanging in a harness receives a label of a syndrome, suggest treatment modalities based on actual experimentation, and are against extrapolation of other conditions to be applied in practice. Testing should be performed using more advanced technology to measure physiologic variables, human biomarkers, and subjective assessments of those while hanging in a harness.

Figure Legend

Figure 1 showing a front, chest, or anterior, point of attachment.

Figure 2 demonstrates free-hanging in a dorsal point of attachment

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CHAPTER 3

Harness Suspension Stress (HSS) Physiological and Safety Assessment

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Abstract

Background: Harness Suspension Stress (HSS) is commonly known as suspension trauma, harness hang syndrome, harness induced trauma, harness-induced pathology. Hanging motionless in a harness has been thought to mimic other medical conditions such as compartment syndrome, compression syndrome, and crush syndrome, or syncope secondary to venous pooling, by hanging in a harness for an extended time. The prevalence or likelihood of metabolic or physiologic changes while hanging in a harness has not been evaluated. **Methods:** Eighteen healthy volunteers were recruited (males = 9, female = 9), and underwent a prescreening questionnaire, tilt-table test, skinfold for estimation of body composition, and treadmill maximum consumption of oxygen test (VO_{2max},) prior to undergoing HSS testing. Subjects were randomized to hang motionless and freely by frontal or dorsal point of attachment for thirty minutes, volitional end-test, or becoming clinically symptomatic. Ultrasonography was performed to measure IVC, MV, and EF before during and after hanging testing. Vital signs, SpO2, and ECG, and subjective comfort were monitored throughout testing. **Results**: Discomfort and maximum mean arterial pressure was significantly ($p<0.05$) and clinically related to symptomatic end-test. **Conclusion**: Many variables were evaluated to isolate the etiology and guide management of HSS. However, further research should be performed and caution is advised for aggressive treatments of HSS without direction.

Introduction

Hanging motionless in a full body harness may induce physiological effects on the body. Early research documented a near syncopal episode that occurred while testing military parachute harness in 1968.[1] Since then, some testing of HSS has been reported to produce

symptoms of dizziness, diaphoresis, nausea, cardiac dysrhythmias, and syncope. [2,3] The

full body harness was found to be a superior design compared to a chest-only harness or

body belt design harness, as it did not increase intrabdominal or intrathoracic pressures

which caused asphyxiation. The full-body harness was still associated with

symptomatology leading to end testing in less than thirty minutes. [4,5] The

pathophysiology has been postulated to be secondary to venous pooling, leading to

decreased cardiac return as a focus of symptoms, but only vital signs and subjective data

have been reported. [6,7] Vasovagal syncope, as a function of parasympathetic tone, has

also been cited as a possible cause. [4] The exact mechanisms of HSS have not been

understood or explained, warranting further investigation into the pathophysiology. [8,9]

Dynamic fall testing has been suggested as future research. [4,10] However, no

investigations have looked deeper into the possible contributing factors prior to moving

into more complex testing, because there has not been an established consensus of a

cause-effect relationship.

To maintain simplicity, only motionless HSS was evaluated, not abrupt velocity

declarations, such as in parachute deployment, rock and ice climbing, or industrial rope

work situations. It was hypothesized that there will be no significant change in

biomarkers, cardiac measurements, or discomfort for human subjects hanging motionless

in a harness, and there will be no correlation between gender, fitness level, or body fat

percentage and becoming symptomatic while hanging motionless in a harness.

Methods

A G-power analysis was performed with a power set at 0.80 ($\alpha = 0.05$) to

determine a sample size of 18 needed to detect symptoms in suspension duration of 30

minutes. Twenty-two healthy volunteer subjects were initially recruited. Four females and one male dropped out of the testing due to time constraints, and one female was released because of aversion to intravenous needles, all prior to harness testing. Subsequently, nine females and nine males (n=18) were retained and enrolled into a prospective randomized crossover trial. For Internal Review Board approval, subjects were between 18-40 years old, and informed consent was obtained prior to testing. Exclusion criteria were defined as: current illness, chronic illness, and history of cardiac dysfunction, orthostatic hypotension, pregnancy, and inability to properly fit into a harness. The University of New Mexico Institutional Review Board approved the protocol as outlined in Figure 3.

Prior to harness testing, all subjects performed a tilt table test to evaluate for orthostatic hypotension syndrome predilection. No subjects were disqualified due to tilt testing results. Body fat percentage was estimated using skinfold calipers and a treadmill maximum oxygen consumption test (VO_{2max}) was performed and recorded on a separate day from HSS testing. On the day of the first harness test, subjects were randomized to hang in the frontal (**Figure 8**) or dorsal (**Figure 9**) point of attachment. All subjects performed urine analysis within fifteen minutes prior to hanging in a harness, and for females, a negative urine pregnancy test was needed to proceed. A 12-lead ECG (Zoll, Chelmsford, MA, U.S.A.) was performed before testing to rule out abnormalities, and a 4-lead ECG was left on the subject for the remainder of the HSS testing for continuous rhythm monitoring.

Because of large variations in anthropometry of the subjects, a harness was chosen by the subject to accommodate the most comfortable and proper fit. [10] All

harnesses were of the same general design by multiple manufacturers (**Figure 7**).
Subjects were suspended from two bolts in the ceiling and a climbing rope clipped to the
attachment point of the harness. Subjects stepped up on a stool, and when the step was
removed, subjects remained suspended approximately eight inches off the padded floor,
and then the timer was started. The subject remained still during testing to achieve a
simulated situation resembling an unconscious victim hanging from a single front or
dorsal point of the harness.

Systolic (SBP) and diastolic blood pressure (DBP), heart rate (HR), respiratory
rate (RR), blood oxygen saturation percent (SpO_2), lower extremity pulse quality (DP),
and ankle girth (AG), were measured before and every five minutes during the test until
thirty minutes, volitional end test, or indication for end test by researchers. Using the
NRS-11 pain scale, subjective discomfort (Discom) level was assessed every five minutes
on a 0-10 scale, least to most discomfort, respectively. [11]

A peripheral venous catheter was placed for the duration of the harness testing.
Venous blood was taken from the catheter before testing and after testing. Venous blood
samples included complete blood count (CBC), complete metabolic panel (CMP), and
creatine kinase (CK). This blood was sent to a Quest laboratory for testing.

A physician performed ultrasonography while the subject was supine during pre-
test, standing, hanging at fifteen minutes, and at end test in supine position to monitor
changes in inferior vena cava, mitral valve e-point, and left ventricular ejection fraction
(EF).

For continuous tracking of cardiac performance, a BioZ noninvasive impedance
cardiography (ICG) monitor (Sonosite, San Diego, California, 92121 USA) was used to

measure and monitor SBP, DBP, HR, cardiac output (CO), and systemic vascular resistance (SVR), Stroke Volume (SV), cardiac index (CI) throughout the HSS testing.

Subjects were placed in the harness and then suspended for thirty minutes or less if they became clinically symptomatic, or elected to end the testing. Symptomatic conditions disallowing the subject to continue with the study included severe or intolerable pain, dizziness, light-headedness, pre-syncope or syncope, cardiac dysrhythmias, reported visual disturbances, decreased cardiac output, systemic vascular resistance changes, or abnormal ECG, or subject asking to discontinue with testing.

Statistical Analysis:

Differences in cardiovascular variables and discomfort ratings were analyzed using a two-way repeated measures analysis of variance (ANOVA), with trial (frontal vs. dorsal) being one factor and time (0, 5, 10, and 15 min.) being the other factor. Differences in inferior vena cava between inspiration and expiration (IVCi and IVCex) were analyzed using a two-way repeated measures ANOVA, with trial (frontal vs. dorsal) being one factor and position during trial (begin supine, stand, hang, and end supine) being the other factor. Differences in blood markers were analyzed using a two-way repeated-measures ANOVA, with trial (frontal vs. dorsal) being one factor and time (pre vs. post) being the other factor. Main effects for trial and time, in addition to the trial-by-time interaction were accepted as significant if $p < .05$. Post-hoc paired samples t-tests were conducted when there were significant main effects or interactions, and comparisons were accepted as significant if $p < .05$. Differences in specific gravity between trials were analyzed using a paired-samples t-test. Differences were accepted as significant if $p < .05$. The relationships between those who ended early during the dorsal

trial and HR, SBP, DBP, MAP, Pulse Pressure, and discomfort rating at 0, 5, 10, 15, 20, 25, and 30 minutes were investigated using linear regression analysis. Pearson correlations were accepted as significant if $p < .05$. All values are presented as means ± SD. Data were analyzed using IBM SPSS Statistics version 19.0, Chicago, IL.

Results

Heart Rate

During the dorsal trial, HR was significantly greater at 5 min (86±14 bpm) and 15 min (87±17 bpm) when compared to baseline (75±13 bpm) (CI = 95%; $p = 0.003$). Heart rate was significantly higher at 15 min during the dorsal trial (87±17 bpm) when compared to 15 min during the front trial (76±15 bpm) (CI = 95%; $p = 0.01$).

Blood Pressure

SBP (front trial) was significantly higher compared to baseline after 5 min (118±15 mmHg vs. 125±17 mmHg) (CI = 95%, $p = 0.009$), but change thereafter for front SBP was statistically insignificant ($p = 0.225$). There were not any significant differences in SBP during the dorsal trial (CI = 95%, $p \geq 0.05$). Diastolic blood pressure (front trial) was significantly higher after 5 min (80±9 mmHg vs. 85±8 mmHg), 10 min (80±9 mmHg vs. 84±9 mmHg), and 15 min (80±9 mmHg vs. 85±11 mmHg) when compared to baseline (CI = 95%, $p < 0.05$). There were not any significant differences in diastolic blood pressure during the dorsal trial (CI = 95%, $p \geq 0.05$). Mean arterial pressure (front trial) was significantly higher after 5 min (92±10 mmHg vs. 98±10 mmHg), 10 min (92±10 mmHg vs. 96±10 mmHg), and 15 min (92±10 mmHg vs. 97±11 mmHg) when compared to baseline (CI = 95%, $p < 0.05$). There were not any significant differences in mean arterial pressure during the dorsal trial (CI = 95%, $p \geq 0.05$). The percent change in

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the MAP of all subjects combined in both trials was greater than the percent change in SBP, DBP, and Pulse Pressure (PP) for all subjects combined in both trials (**Figure 5**). A weak rise in dorsal percent change pulse pressure (DpcPP) and dorsal percent change MAP maximum (DpcMAPmax) showed no direct cause-effect relationship ($p \geq 0.05$) over time **(**Figure 6).

Blood laboratory values

Change in the major blood laboratory markers of concern during this testing, Potassium (K+), Creatine Kinase (CK), and Calcium (Ca++), were insignificant both statistically ($p \geq 0.05$) and clinically as seen in Table 1. An additional post-test lab for CK was drawn when subjects returned 24 – 48 hours after each HSS test was finished. Labs generally used to help diagnose pathology are seen in Table 2.

Fitness

A wide range of VO_{2max} test results were recorded ($34.2 - 65.4$ mL*kg^{-1}*min^{-1}). There was no significant difference for those completing HSS testing at thirty minutes (48.97 ± 8.89 mL*kg^{-1}*min^{-1}) (CI = 95%; p = 0.91, n = 11), and those who had enough symptoms either clinically or volitionally, to end the test (46.95 ± 8.81 mL*kg^{-1}*min^{-1}) (CI = 95%; p = 0.90, n = 7). There was also a wide range of percent body fat (% fat) (4.6% - 39.01%). There was no significant difference in body fat percent for those completing HSS testing at thirty minutes (16.97 % ± 8.86%) (CI = 95%; p = 0.894, n = 11), and those who had enough symptoms either clinically or volitionally, to end the test (16.30% ± 12.16%) (CI = 95%; p = 0.902, n = 7).

Hydration:

Hydration status was evaluated by urine specific gravity analysis prior to both HSS tests. No significant difference between front or dorsal (1.05 ± 0.32; 1.02 ± 0.01, respectively) (CI = 95%; $p = 0.3$) was found between groups. Paired samples correlations showed no significant difference for subjects who had symptoms and those who did not in either front or dorsal (CI = 95%; $p = 0.9$, $p = 0.28$, respectively) points of attachment.

Discomfort:

The discomfort rating during the front trial significantly increased from 1 ± 1 at baseline to 3 ± 2 at 15 min (CI = 95%; $p = .000$). The discomfort rating during the dorsal trial significantly increased from 2 ± 2 at baseline to 4 ± 2 at 15 min (CI = 95%; $p = 0.007$). The subjective reasons for ending testing are listed in Table 3.

There were no significant changes to biomarkers, oxygen saturation, or calf girth in any subject (CI = 95%; $p \geq 0.05$).

One heart dysrhythmia, a junctional escape rhythm, was observed in a male subject during both frontal and dorsal trials, and return to sinus rhythm occurred within 3 minutes after end testing. Sonography and ECG of another male subject, prior to end testing, detected a single sinus pause without further ectopy or dysrhythmia. No other ectopy or rhythm disturbances occurred for any other subject. The sonographic data was not robust enough to perform statistical analysis. No tests were discontinued due to clinical observation of sonographic findings.

Discussion

The purpose of this study was to measure and analyze biomarkers, cardiac measurements, and discomfort amongst both male and female subjects of varying fitness levels and body fat percentages in an effort to gain insight into the possible etiology of HSS. Our results conflict with prior research, as anthropometric measurements, gender, and level of fitness, appear to be a poor tool to for prediction of symptoms during harness suspension.

Changes were noted in HR, BP, MAP, PP and discomfort. Only a cause and effect relationship to becoming symptomatic was found with elevated HR within groups, and elevated DBP within and between groups. Volitional ending of testing due to discomfort, rather than syncope, could account for insignificant findings in vital signs. Beat-by-beat BP measuring may help gain better data.

Pressures exerted on the body while hanging in a harness are markedly different than when standing, the forces are redistributed to the groin region and are remarkably uncomfortable. It is possible that continued forces exerted on pain receptors at harness pressure sites increase overall pain area under the curve. Pain may lead to anxiety that can induce a hypothalamus-pituitary-adrenal catecholamine response by activating cytokines, and releasing cortisol that inhibits cognition, driving misperception of external stimuli. [12] Acute spikes in MAP that are sustained over time may resemble Cushing's reflex: MAP increase, irregular RR, and bradycardia. The combination of the harness pressures, increased MAP, and adrenaline, could lead to a Cushing's-type response. [13] Both of these reflexes appear to be somehow related, so it follows that the induced cardiovascular effects of hanging in a full body harness may also apply with these concepts.

Some subjects had discomfort immediately upon being suspended in the harness, while others may have had a gradual increase. No subjects failed the tilt table test prior to HSS testing, no ankle girths increased throughout testing, and MAPs were elevated one harness testing commenced. The culmination of the results from this research does not support previous theories regarding venous pooling as a possible mechanism for HSS.

There were no statistically or clinically significant changes in biomarkers or hydration status in any subject throughout this study. The notion that HSS is directly related to increases in K+, Cr, CK, or other blood assays is unfounded. No correlation was found to exist between becoming symptomatic and gender, fitness level as measured by VO_{2max}, or body fat percentage.

Possible explanations for these findings include: lack of a pathology that would cause a change in biomarkers, lack of electrolyte shifts causing symptoms, or variable cardiac changes that are not attributable to a single pathology. These data may be pointing instead towards modulated central fatigue. [14] No assays of neurotransmitters were performed, but initial catecholamine release and subsequent withdrawal may mimic these symptoms. Neurohormonal regulation of cardiovascular system is complex and was beyond the scope of this study.

The use of ultrasound was technically difficult as the full body harness hinders sonographic access and windows. Moreover, no standards have been set for subjects in the HSS configuration a baseline for comparison.

Improvements to this study should include a reliable method to fully evaluate global perfusion status. Methods should be improved to closely monitor observations

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such as continuous central venous cardiac pressures in conjunction with peripheral arterial pressures to account for any shunting effect and possible volume displacement.

Beat-by-beat blood pressure measurement may have provided a more precise view of changes in MAP, SBP and DBP peripherally. The lack of having good access for sonography due to the full body harness hindered the ability to assess blood volume flow and cardiac function uniformly.

It is concluded that the ability to predict who will be susceptible to HSS is not clear. Medical treatment modalities for HSS should be carefully considered, administered with caution, and should be based on current ACLS guidelines. Discomfort shows a direct relationship to symptoms within groups. Current harness design should be evaluated in light of these findings.

Future testing should involve continuous peripheral and central vascular monitoring as well as beat-to-beat measurement of blood pressure. Quantitative laboratory assays of cortisol, epinephrine, and choline-acetylcholine, should be considered. Vasovagal etiology may also be of interest as one subject displayed a junctional escape rhythm in both trials. Strain gauges sensitive to pressure should be evaluated not only at the groin region, but also around the carotid bodies of the neck.

Competing Interests

All authors and contributors declare no conflict of interests. All models have been

released. Photographs are used by permission from the Beverly Collection.

Table of Figures

Table 1 Statistical chart by biomarkers

Biomarkers				
Variable	**Pre-test**	**Post-test**	**n**	**p-value**
Creatine Kinase (CK)				
Front	154.8 ± 102	146.7 ± 91.4	17	0.451
Dorsal	162.3 ± 137.7	157.1 ± 113.4	17	0.451
Potassium (K+)				
Front	4.2 ± 0.3	4.3 ±.3	17	0.61
Dorsal	4.2 ± 0.4	4.3 ± 0.5	17	0.61
Calcium (Ca++)				
Front	9.6 ± 0.3	9.6 ± 0.4	18	0.67
Dorsal	9.5 ± 0.3	9.6 ± 0.4	18	0.67
Creatinine (Cr)				
Front	1.01 ± 0.2	1.00 ± 0.2	18	0.458
Dorsal	0.9 ± 0.2	0.9 ± 0.2	18	0.458
Glucose (Glu)				
Front	75.5 ± 17.7	76.35 ± 14.8	17	0.51
Dorsal	77.3 ± 14.0	80.9 ± 16.3	17	0.51
Platelet (plt)				
Front	229.4 ± 45.0	237.4 ± 47.9	17	0.544
Dorsal	240.1 ± 66.6	238.3 ± 56.3	17	0.544
Urine Specific Gravity (USG)				
Front				
No symptoms	1.05 ± 0.12		16	0.902
Symptoms	1.04 ± 0.02		2	0.902
Dorsal				
No symptoms	1.02 ± 0.01		11	0.278
Symptoms	1.02 ± 0.01		7	0.278

Values are Mean ± SD, n = number, p-value = statistical significance. There was no statistically significant treatment effect for CK, K+, Ca++, Cr. Glu, plt, or USG. Therefore, *post-hoc* analyses were not performed. Tests were performed before and after HSS testing.

Table 2 describes general blood indices that are used for help to diagnose pathology

Blood indices by pathology	
	p-value
CK = renal injury,crush theory, rhabdomyolysis	>0.05
K+ = dysrhythmias, crush theory, toxic venous return, afterdrop	>0.05
Ca++ = reperfusion injury, dysrhythmias	>0.05
Cr = dehydration theory (Bezold-Jarsch), rhabdo	>0.05
Glucose = hypoglycemia	>0.05
Platelet = clotting factors/DVT/PE/MI/CVA/hemorrhage	>0.05
USG = dehydration theory	>0.05

Table 3 Subject by configuration and reasons at end test.

Subject	Symptoms	Position
15	"Nausea, light headedness", Sinus Brady @ 52 BPM	D
5	"feels like I'm going to pass out"	D
1	"Light headedness"	D
6	"Nausea", junctional escape rhythm	D/F
16	"Light headedness"	D
17	"Light headedness", near syncope	F
21	"Nausea, pain in groin", bradycardia	D

Graphs and Charts
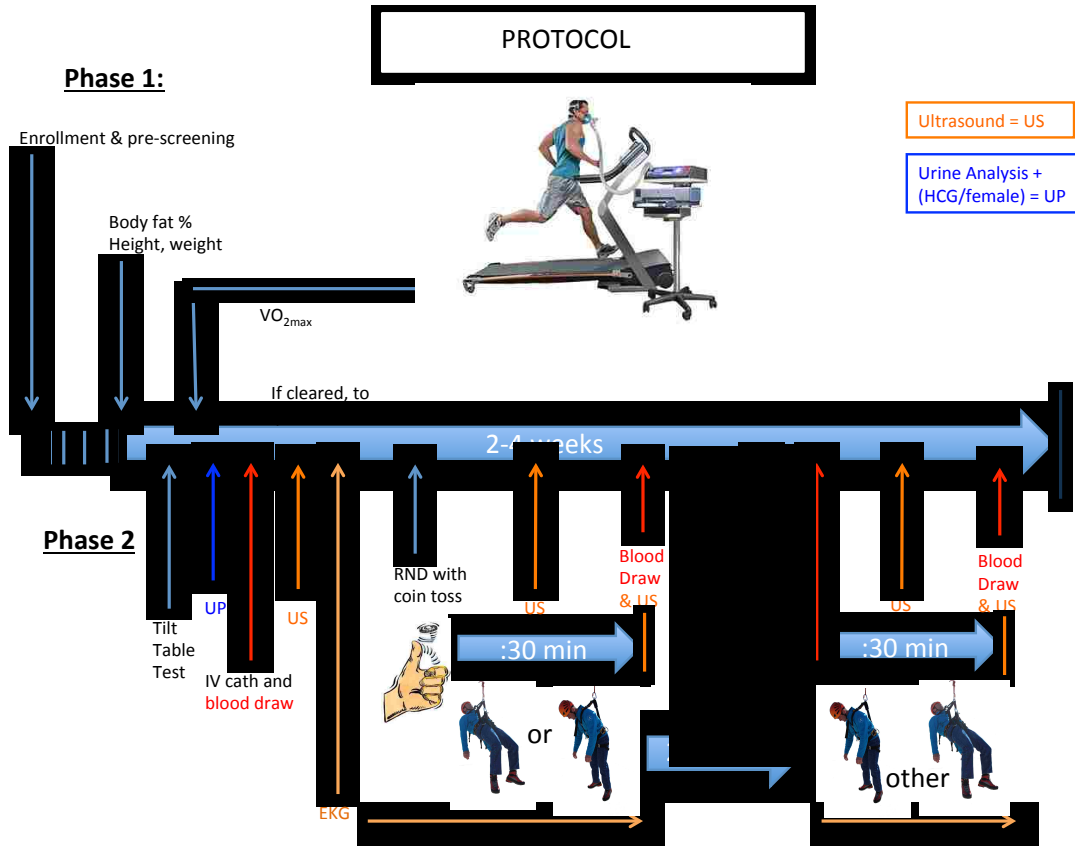
Figure 3 the entire protocol is shown with timeline**.**
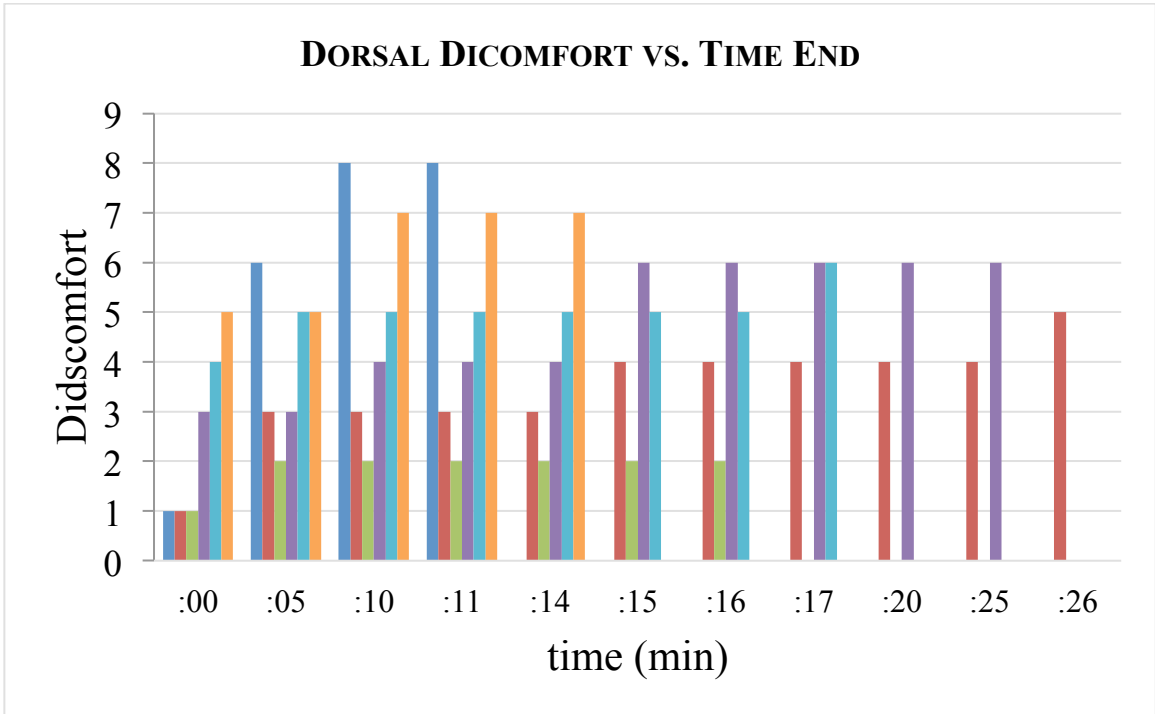
DORSAL DICOMFORT VS. TIME END

Figure 4 shows the six subjects with dorsal position hangs had end early tests vs. discomfort. Discomfort was the main reason to stop tests overall.

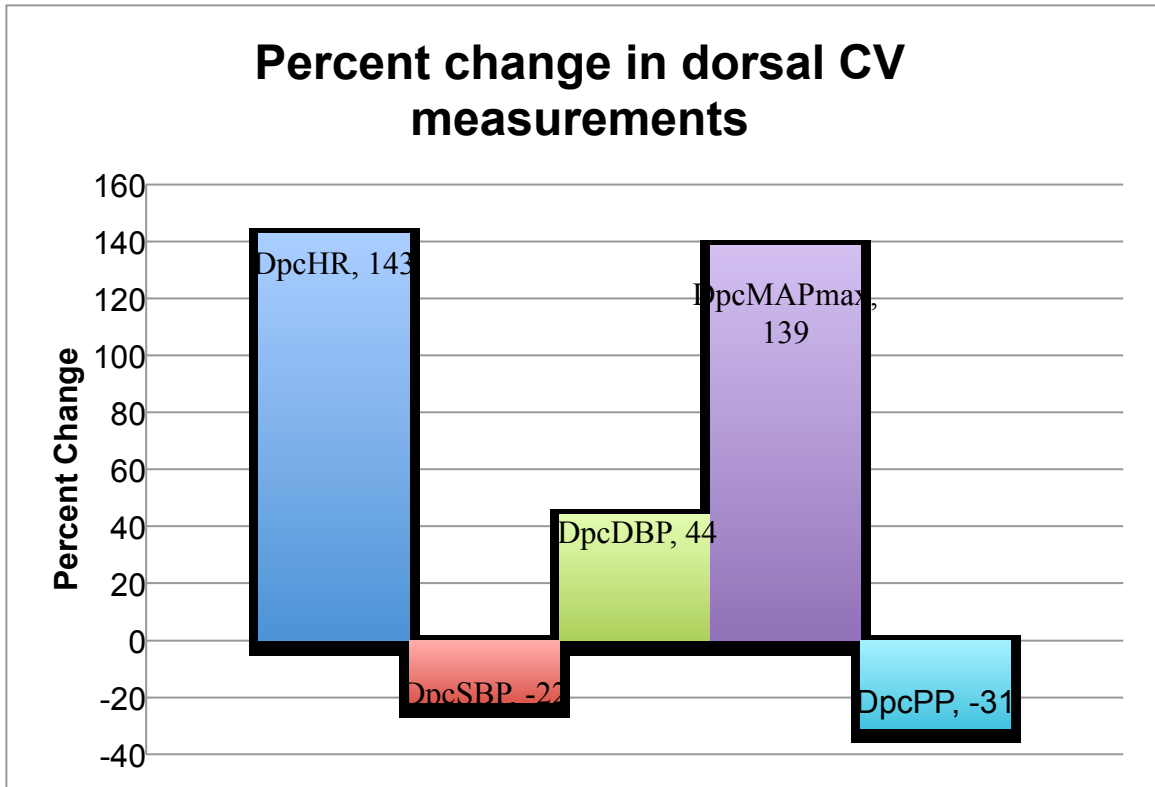**Percent change in dorsal CV measurements**

Figure 5 showing the total sum of the averages in percent change from baseline (0) to end test, for all subjects in the dorsal hanging position: (from left to right) heart rate, diastolic blood pressure, pulse pressure, systolic blood pressure, and mean arterial maximum. Overall, spikes in MAP increased are 139% from baseline.
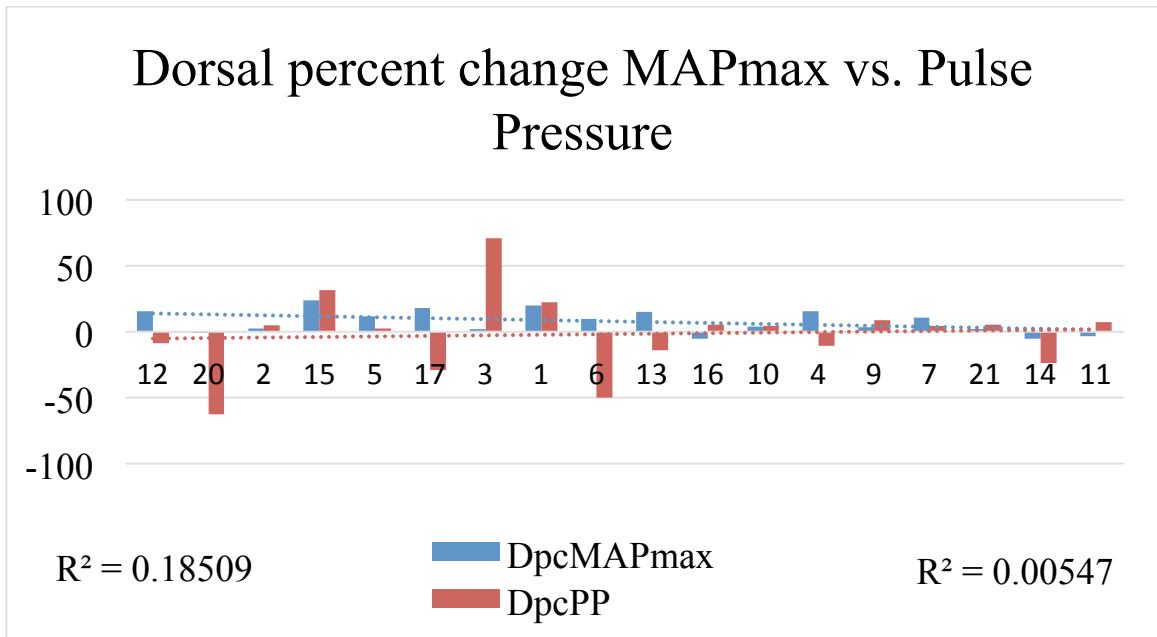
Figure 6 shows dorsal mean arterial pressure percent change at maximum during the trial vs. pulse pressure percent change. No direct relationship is made in these trials.
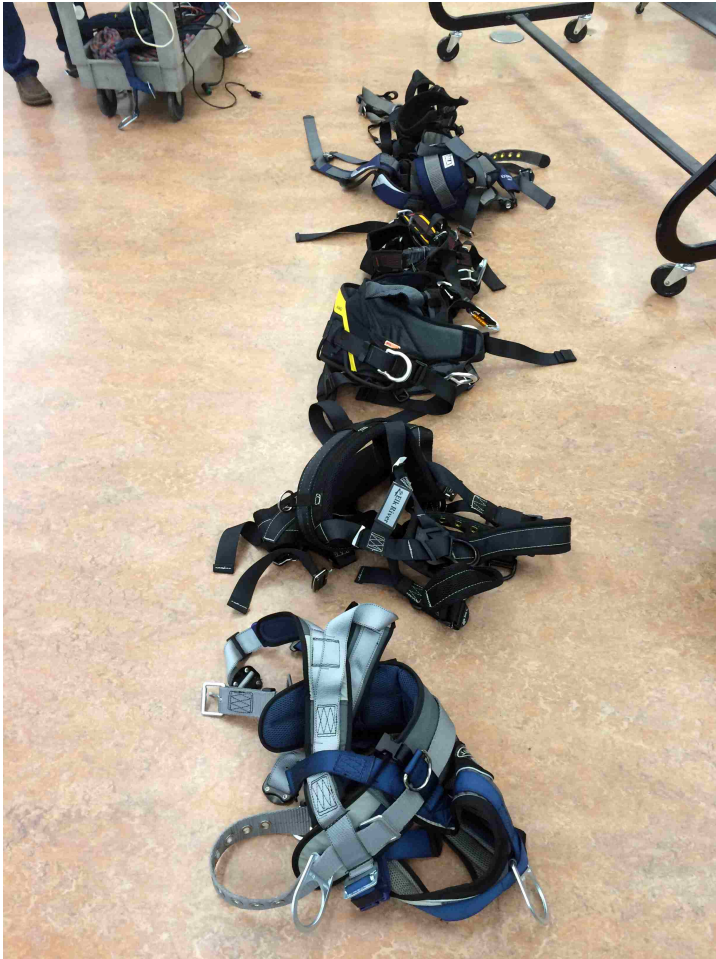
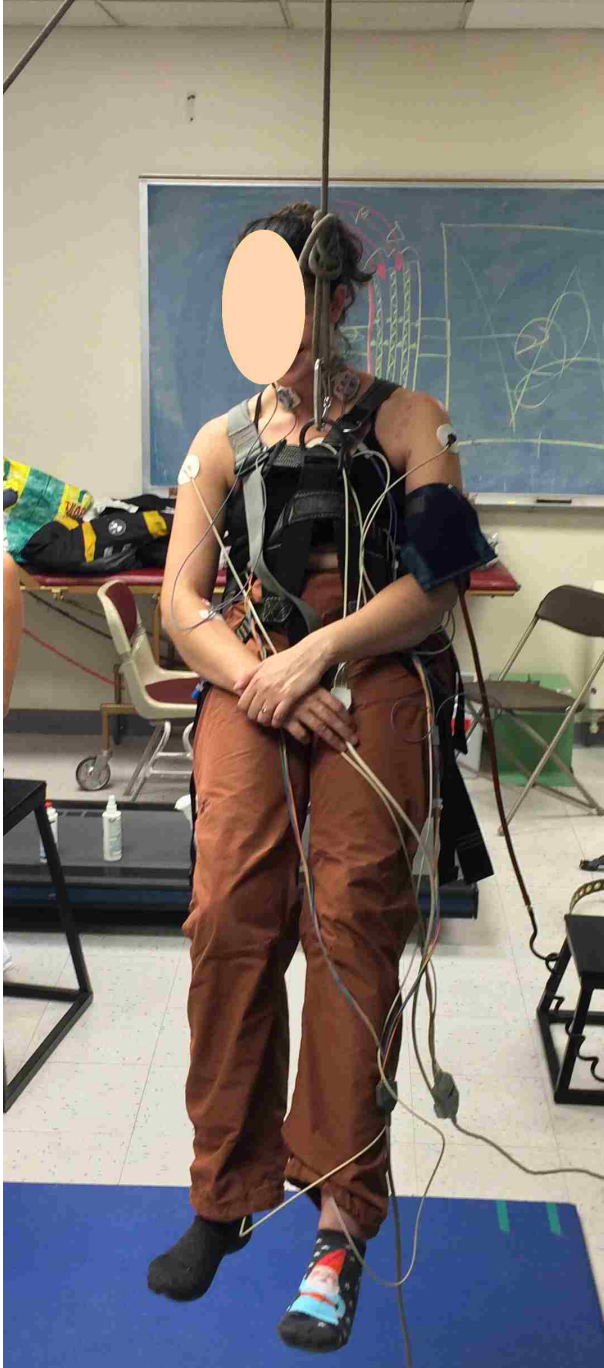**Figure 7** multiple harnesses were made available to assure best fit for the subjects.

Figure 8 the instrumented frontal point of attachment test configuration.

Figure 9 the dorsal point of attachment used while instrumented during testing with close monitoring.

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Chapter 4

Summary

Articles have been published on the topic of HSS, many of which have low levels of evidence or only written as expert opinion. A formal review of literature has not been recently performed to evaluate the pathology and treatment recommendations regarding HSS. Rather, the literature suggests further investigation into the pathophysiology of HSS, and against any recent documented approaches to rescue or medical therapies by expert opinions. There are minimal bodies of data to draw information from if one were to investigate the etiology of HSS. Lack of consensus on the likely etiology and proposed treatment of HSS is made evident by performing this formal literature review and research. This research manuscript sought to compile the varying theories and suggested treatment methods for HSS in an effort to guide formalized research on the topic of HSS.

Current literature does not include studies on HSS that measure biomarkers such as clinically relevant laboratory blood markers, multiple cardiac measures, neurohormonal regulation, autonomic nervous system regulation, discomfort, nor consideration of gender, fitness level, or body fat percentage to assess for the possible pathology of, or predisposition to, HSS. Performing an experiment that focuses on these measures provided a data set that is able to be statistically and clinically reviewed to present evidence regarding the development of, and predisposition to, HSS. These findings will help guide future research on this topic, which has yet to be fully understood.

The research manuscript includes a prospective randomized cross-over study with statistical analysis on the correlation, cause and effect, and clinical significance of biomarkers, multiple cardiac measures, discomfort, gender, fitness level, and body fat

percentage to becoming symptomatic while hanging motionless in a harness. The clinical and statistical analysis of the data provides information that differs from the body of literature available and sheds new light onto the lack of clinical pathology that was observed in subjects who became symptomatic during one or both trials.

Conclusions

There were no changes to biomarkers including K+, Cr, CK in any subject, symptomatic or asymptomatic, of clinical or statistical significance to account for onset of symptoms in either trial. There is no statistically significant evidence of HR, BP, MAP, or PP having any relationship to becoming symptomatic in either trial, but these measures should be further investigated and understood prior to proceeding on with dynamic testing as suggested in the literature. Clinical changes to HR, heart rhythm, BP, PP, and MAP were observed but were not consistent amongst subjects There is currently no better method to investigating cardiac function in real time than with invasive techniques. It may be that in order to fully appreciate the nuances of HSS, invasive monitoring of subjects may need to be a consideration. There is a direct relationship between discomfort and subjects ending the trial before the maximum time of thirty minutes, which complicates observations more and clouds the evidence needed to progress towards a more concrete pathophysiology.

Gender, body fat percentage, age, and fitness level did not predict who became symptomatic. There was no correlation between hydration status and becoming symptomatic. Lower leg measurements of DP quality and of ankle girth did not change at any point in the trial for asymptomatic or symptomatic subjects. Sonographic

measurements were too difficult to obtain while subjects were suspended in the harness since the harness obstructed the window. Therefore, if there was a statistically significant change to IVCi:IVCexp or other cardiac function, we were unable to appreciate it using manual sonography. Based on the physician's observations, there was no notable difference in ejection fraction, but real-time measurements were difficult to obtain during testing. Moreover, there are no current standards for measurements in these configurations, making comparison to baseline parameters difficult.

Recommendations

The findings of this experiment do not point directly to any one specific etiology of HSS. They do, however, reveal that there are no electrolyte shifts statistically or clinically of significance, and the clinically observed cardiac changes varied with no consistent pattern. Current medical and maneuver treatment methods for pre and post rescue from hanging in a harness that are currently proposed should be administered with caution, as the data gathered during this study does not clearly support any single treatment algorithm that was found in the review of literature. The most up-to-date advanced cardiac life support algorithm should be administered at the discretion of the provider caring for the patient.

Further research is needed to investigate the etiology of HSS so that evidence-based treatment methods can be developed. More precise measurements of peripheral and central perfusion trends should be obtained to more carefully evaluate cardiologic pathology that could possibly develop while hanging in a harness.

The usefulness of the dorsal point of attachment becomes in question in light of the increased symptoms seen in our subjects. Harness construction design needs to be reevaluated not only for comfort and fall protection effectiveness, but also for preventing adverse effects of HSS. Although the prevalence of HSS is exquisitely low (0 out of 7 million man hours*year^{-1} over the past several years, per Industrial Rope Access Trade Association) the potential still exists. Without a better understanding of the basic mechanisms that predispose and evoke symptoms, it will be difficult to find a solution to drive harness design and increase confidence that symptoms will not develop.