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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. 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.

Keywords: Harness stress syndrome, hang syndrome, climbing, orthostatic intolerance, safety harness

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 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.

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

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

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 sequelae [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]. 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, 4]. 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 [5]. 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 [5, 6]. There are several theories that seek to explain the processes that cause death by crucifixion [7], 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 [1-4, 8-13]. Although venous pooling has been cited, venous capacitance while hanging motionless in a harness has never been directly measured [8, 11]. 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 [4]. 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 [11, 14, 15]. 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.

Compression syndrome, crush syndrome, compartment syndrome, tourniquet syndrome, and orthostatic hypotension syndrome, have been associated with HSS [1-3, 8]. 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 [14, 16-18].

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 [19]. 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 [20]. 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 neurohormonally mediated [21]. Genetic testing for copy loss of gene SHC2 that causes Shy-Drager syndrome can be isolated [22]. 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 [23]. Significant venous pooling can lead to syncope [24]. 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) [25]. 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 [26]. The logic follows that arteriole collapse occurs when capillary pressure becomes greater than arteriole pressure [27]. 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 which causes the paradoxical decline in sympathetic activity [28]. 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 [23]. 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 [29]. 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 [17, 18]. 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 [15]. 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, 8, 9, 12-16, 23, 30-31]. 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 sequelae [30]. 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 may be a neurally mediated response to HSS which 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, 32].

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 [33]. 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 [33].

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 [34]. 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 [11]. 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 [12, 13].

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. Studies on actual subjects should be performed using more advanced technology measuring physiologic variables, human biomarkers, and subjective assessments of subjects' perceived experiences.

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