

**An Evidenced Based Review of Suspension Trauma Pathophysiology  
and Medical Management**

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## ABSTRACT

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Fatal falls continue to be one of the leading causes of traumatic occupational death in the US, with approximately four construction workers killed each day and a third of those cases due to falls<sup>1</sup>. Despite advances in safety and prevention, workers and climbers who are suspended in their harness post-fall are at serious risk of imminent disability and death.

Suspension Trauma, (also referred to as Orthostatic Intolerance, or Harness Hang Syndrome) continues to be a common concern for rescuers, climbers, and workers alike. However, an accurate and in-depth understanding of the physiological mechanisms at play in a suspended patient is often lacking. Similarly, readily available information online does not always capture the full picture or reflect the latest scientific evidence regarding pathophysiology and medical treatment.

This paper provides a detailed examination of the physiologic impact of Suspension Trauma on the human body, as well as a thorough review of the latest evidence for appropriate medical management. The body employs four methods to ensure adequate blood return from the lower extremities, especially during orthostatic challenges: one-way venous valves, the skeletal-muscle pump, smooth muscle tone, and the thoracic pump mechanism. However, in the context of Suspension Trauma, the patient's upright position, limited movement, and venous compression overwhelm the body's compensatory mechanisms and result in syncope, circulatory failure, and eventually death. Symptoms of Suspension Trauma set in rapidly and research has found a minimum onset of 5 minutes and mean of 14 minutes<sup>2,3</sup>. Reflow Syndrome is often touted as an additional complication of Suspension Trauma, whereby there is a build-up of metabolic toxins in the legs due to poor tissue perfusion, which when returned to normal circulation, result in vascular toxicity, impaired body and brain function, and possible death. However, various scientific studies have found no evidence for Reflow Syndrome in Suspension Trauma<sup>3,4,5</sup>. As a result, evidence-based treatment of Suspension Trauma has also begun to shift. Original treatment methods invoked the Reflow Syndrome concept and suggested that patients should be kept at a 30° incline to slow toxin return. After reviewing all of the available literature in this area, it is safe to conclude that all post-suspension patients should be placed in a fully horizontal position for treatment and recovery<sup>3,4,5</sup>.

## **1. INTRODUCTION**

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Fatal falls continue to be one of the leading causes of traumatic occupational death in the US, with approximately 4 construction workers killed each day and a third of those cases due to falls<sup>1</sup>. Despite advances in safety and prevention, workers and climbers who are suspended in their harness post-fall are at serious risk of imminent disability and death.

Suspension Trauma, (also referred to as Orthostatic Intolerance, or Harness Hang Syndrome) continues to be a common concern topic for rescuers, climbers, and workers alike. However, an accurate and in-depth understanding of the physiological mechanisms at play in a suspended patient is often lacking. Similarly, readily available information online does not always capture the full picture or reflect the latest scientific evidence regarding pathophysiology and medical treatment.

Suspension Trauma is the loss of consciousness due to a patient being held upright in a harness with limited movement for a period of time. Without prompt recognition and intervention, Suspension Trauma can rapidly become fatal. As a result of impaired motion, blood begins to pool in the venous system of the legs, leading to an overall decrease in the volume of arterial blood effectively perfusing tissue, called effective circulating blood volume, by up to 20%<sup>2</sup>. Lying in a horizontal position, the average person has 12-15% of their blood volume in their legs, which increases to 19-22% when standing<sup>3</sup>. As a consequence of this relative hypovolemia (low blood volume), brain perfusion and oxygenation are compromised, resulting in syncope (loss of consciousness) and eventually death.

## **2. PHYSIOLOGY OF VENOUS BLOOD FLOW**

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In a healthy state, there are four separate mechanisms involved in returning blood from the venous circulation back to the heart.

### **2.1 One-Way Valves:**

The venous system uses frequent one-way valves to prevent backflow of blood due to gravity or increases in internal compartment pressures. These bicuspid leaflets are found throughout the extremities and predominantly in vessels inferior to the heart<sup>4</sup>. For a healthy individual with a mean arterial pressure of 95 mm Hg, venous pressures will be between 3-15 mm Hg<sup>5</sup>. This pressure is only sufficient to increase the height of blood up the legs by approximately 20 cm when standing. Naturally, these valves are necessary to ensure appropriate flow direction.

### **2.2. Skeletal-Muscle Pump:**

Active skeletal muscle contraction directly increases the pressure surrounding venous vessels in the extremities and mechanically propels venous blood forward. The space or compartment into which a muscle must contract is relatively fixed due to the internal connective tissue fascia and exterior skin epithelium. As a result of this fixed volume, intra-compartmental pressures inevitably increase during muscle contraction. This, in turn, causes an increase in driving blood pressure, which forces venous blood from the

legs towards the heart (aided by the directionality of the one-way valves discussed above).

### **2.3 Vascular Smooth Muscle Cells:**

Vascular smooth muscle cells (VSMCs) found in the middle layer, or tunica media, of veins and the larger vena cava are capable of adjusting the internal resistance. These VSMCs are arranged circumferentially around the vessels, and when in a contracted state, reduce the internal radius of the vessels, thereby raising internal resistance and pressure<sup>5</sup>. These VSMCs are not responsible for increasing driving blood pressure, which is accomplished specifically by the skeletal-muscle pump, but rather for responding to hypo- and hypertensive states by adjusting the venous blood flow. These VSMCs are controlled primarily by the sympathetic nervous system via the agonistic actions of norepinephrine on  $\alpha$ -1,  $\alpha$ -2, and  $\beta$ -2 adrenergic receptors<sup>5</sup>.

### **2.4 Thoracic-Pump:**

Lastly, the thoracic-pump mechanism aids in enhancing venous return to the heart. During inspiration, intrapleural pressure becomes more negative, leading to expansion of the lungs, increased blood flow into the pulmonary vasculature, and reduced pressure in the right atrium, ventricle, and major veins entering the heart. This pressure drop results in a higher volume of blood entering the right side of the heart, thereby increasing right preload, raising right-sided stroke volume, and increasing the pressure gradient for venous return<sup>6</sup>.

## **3. PATHOPHYSIOLOGY**

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### **3.1 Relative Hypovolemia:**

Suspension Trauma is, at its foundation, a manifestation of shock; relative hypovolemia and syncope due to increased blood pooling in the lower extremities. This is in contrast to absolute hypovolemia as a result of haemorrhage or inadequate fluid replacement/increased insensible losses. As discussed above, the intact skeletal-muscle pump in the healthy state is a key component to ensuring appropriate venous blood return to the heart. Without the periodic increases in local venous pressure caused by muscle contraction, sufficient volumes of blood are not able to return from the legs, regardless of overall blood pressure, vasoconstrictive effects of VSMCs, or the thoracic-pump mechanism. Indeed, limited movement and muscle contraction is the primary driver of Suspension Trauma and may result from any number of factors including tangled equipment, a poorly fitted harness, physical injury, hypothermia, or fatigue. As blood return is reduced, overall effective circulating volume is diminished resulting in decreased heart filling and stroke volume, as well as decreased kidney glomerular filtration rate<sup>7</sup>. This can eventually lead to a decrease in vital organ perfusion, most notably a decrease in brain oxygenation and perfusion.

Given the increase in blood volume in the legs, capillary pressure will also begin to rise. This increased capillary hydrostatic pressure raises the flow of fluid across the capillary wall, as established by the famous Starling equation<sup>5</sup>, resulting in leakage of fluid into the interstitial space, between the blood vessels and cells, further decreasing intravascular volume. Compounding this fluid shift and lower extremity oedema is the

decreased vascular resistance in the periphery. As the effective circulating volume drops, and hypoperfusion develops, cells are forced to meet their energy needs via anaerobic metabolism. This process produces lactic acid as a by-product, causing an acidic environment and a fall in blood pH. The normal physiological response to acidosis is a decrease in vascular resistance to improve perfusion<sup>8</sup>. Paradoxically in Suspension Trauma, as perfusion to the lower limbs is enhanced via decreased resistance, more blood pools in the legs, further reducing the effective circulating volume and exacerbating the hypovolemia.

One of the last lines of defense that the body employs to protect against brain hypoperfusion is the vasovagal response of syncope, or fainting. This effectively renders the person horizontal, which aids blood return, thereby increasing heart filling and stroke volume, and delivering more oxygenated blood to the brain. However, the suspended patient is, by nature of the situation, unable to move and remains in a relatively vertical position. The person becomes unconscious; blood pressure, oxygenation and perfusion continue to decrease; and eventually the patient dies from hypoxemia.

### **3.2 Venous Compression:**

A potential complicating factor in Suspension Trauma is the pressure exerted by the full body harness leg straps around the femoral vessels. We know from experiments that hypotension and pre-syncope symptoms are reproducible in most healthy individuals subjected to a passive table tilt test in under an hour<sup>2</sup>. Similarly, the example of a soldier fainting while standing at attention is a well-known phenomenon. Yet neither of these examples places any additional physical pressure on blood vessels to impede return. Experiments by Orzech<sup>9</sup> tested several different types of harnesses but found no statistically significant different results. Similarly, the review by Mortimer<sup>7</sup> suggested this was unlikely to be a factor in Suspension Trauma.

However, some evidence does exist to suggest that venous compression may be a contributing factor in some, but not all situations. Animal models have shown that ligation of the caval veins can produce syncopal symptoms, which are reversed when the pressure is relieved<sup>10</sup>. Increased body weight is known to increase the risks of Suspension Trauma, potentially due to increased vessel compression<sup>11</sup>, and these same experiments also showed improvements in tolerable hang time with different full-body harness types. Given the superficial nature of the femoral veins and their highly compressible nature, it seems plausible that the occluding effect of the leg straps could aggravate the already impaired venous blood flow. However, given the limited and conflicting available scientific research it is difficult to draw firm conclusions at this time. As a point of interest and somewhat related note, the review by Mortimer<sup>7</sup> highlighted that some amount of respiratory compromise from compression asphyxia may occur in climbers suspended from a chest harness only, representing yet another potential compounding factor.

## **4. RESCUE DEATH CONTROVERSY**

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The possibility of post-rescue death has been a topic of debate for many years and is often referred to as Rescue Death or Reflow Syndrome. Originally postulated by Patscheider<sup>14</sup> in 1972, there were some early investigation and publications which supported this pathophysiology. Different mechanisms were proposed to cause Rescue

Death including a flood of toxins or acidic blood reinterring circulation in overwhelming concentrations. A thorough review by Seddon<sup>15</sup> for the British Health and Safety Executive in 2002 of all available research into Suspension Trauma at the time highlights several of these publications and hypotheses. One notable investigation was done by Flora and Holzl<sup>16</sup> who reviewed a number of post-rescue deaths, including one of a 23-year-old climber. She died within minutes of rescue and autopsy revealed no signs of major trauma. Understandably, given this evidence, recommendations were made to alter post-rescue management to prevent Rescue Death from occurring. The consensus was that placing post-suspension patients at a 30° elevation instead of fully horizontal would slow the return of toxic blood, giving the body more time to adapt<sup>15</sup>.

Others have suggested mechanisms such as pulmonary embolism, cardiac arrhythmias, or acute volume overload as being responsible or involved in precipitating Rescue Death. Unfortunately, the evidence supporting these hypotheses is lacking. Patscheider<sup>14</sup> found no evidence of PE on autopsy of post-suspension patients. Mortimer<sup>7</sup> highlights several investigations which evaluated cardiac functioning via ECG in suspension and could not produce a robust link. He also points out that patients who are post-suspension are likely to be euvolemic if not hypovolemic, making volume overload an unlikely answer.

There is evidence to suggest that patients suspended for extended periods of time may develop rhabdomyolysis, potentially leading to acute kidney injuries, hyperkalemia, and acidemia post-rescue. The process of rhabdomyolysis is a result of prolonged hypoxemia and nutrient deprivation in muscle tissue. As cellular metabolic requirements become scarce, cellular acidosis and ionic imbalances begin to develop, which eventually destabilize the cellular membrane and result in cell necrosis and apoptosis causing intracellular contents to spill out<sup>12</sup>. Myoglobin, an iron and oxygen binding protein, is one of the intracellular components released by these cells and is toxic to kidney cells. Myoglobin's presence outside of myocytes is detectable in lab tests by elevated creatine phosphokinase (CPK) levels. If large quantities of myoglobin are released, this can overwhelm the kidneys' capacity causing irreversible damage<sup>13</sup>. The intracellular environment is also rich in Potassium, and with enough myocyte destruction, this can significantly increase blood potassium concentrations and produce cardiac dysrhythmias<sup>13</sup>. Similarly, the hypoxemia and concurrent lactic acidosis can significantly depress blood pH, which can be fatal. It should be cautioned that limb hypoxemia and acidosis are common side effects in routine anaesthesia technique, and beyond causing brief cardiac contractility depression, do not affect rhythm<sup>8</sup>.

Skeletal muscle is far more robust than cardiomyocytes and requires 3-4 hours of impaired perfusion before showing signs of destruction<sup>12</sup>. There are certainly case reports of patients being suspended well beyond this threshold or who died post-rescue<sup>16</sup>. However, the question becomes, what roll, if any, does rhabdomyolysis play in causing or exacerbating any Rescue Death? Put differently, was rescue from Suspension Trauma the culprit for developing rhabdomyolysis, or was this process already developing despite rescue efforts? While potentially fatal, rhabdomyolysis and the concurrent AKI are not an immediately acute process, occurring instead over hours and days. Some post-rescue survivors have ultimately died many days later of kidney failure in hospital, but this would not account for those cases in which the patient died within minutes or hours of rescue<sup>16</sup>. The pathophysiology of Suspension Trauma is undoubtedly complex with many potential exacerbating factors. However,

rhabdomyolysis and any concomitant AKI, hyperkalemia, or acidemia appear to be risk factors of Suspension Trauma, rather than possible causes of Rescue Death.

Perhaps the most important consideration at this time is the arrival of more recent evidence and guidelines on the topic, which are refuting the original existence of Rescue Death entirely. Publications by Thomassen et al.<sup>17</sup> as well as by Adisesh, Lee, and Porter<sup>18</sup> found no evidence to support the hypothesis that a flood of toxins were responsible for any Rescue Death. Indeed, the review by Mortimer<sup>7</sup> also calls this concept into question, doubting its existence beyond theoretical possibility. Perhaps most importantly, the latest review by the British Health and Safety Executive in 2009 found insufficient evidence to continue with their originally published recommendation, and states that all post-suspension patients should be treated in the same manner as any other patient suffering from shock, that is, in a fully supine and horizontal position<sup>19</sup>. The debate is likely far from over, but current scientific evidence suggests that while Rescue Death may be a theoretical risk, in reality, patients dying post-rescue are succumbing to their prolonged hypovolemia and hypoxia despite their rescue, not as a result of it<sup>15</sup>.

## **5. PREVENTION OF SUSPENSION TRAUMA**

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As with any incident, prevention is easier than a cure. Whether in recreational or industrial settings, proper pre-planning, equipment, training, and safety awareness are crucial. One study found that presyncopal symptoms developed in a median time frame of 27 minutes<sup>2</sup>, while another study using parachute harnesses produced syncopal symptoms in an average of 14 minutes, and as rapidly as 6 minutes<sup>9</sup>. Clearly, rescue efforts face a significant time constraint, and depending on outside factors such as weather, personnel, training, or accessibility, can be greatly impeded.

Despite these challenges, many practical and potentially life-saving techniques exist. Simple foot loops, either pre-sewn into a full body harness, or created using prusiks provide suspended patients a hold to step into, thus relieving the pressure on their legs and allowing their skeletal muscle pump to function appropriately. Reclining into a semi-recumbent position if possible while suspended also helps limit the amount of blood stasis in the legs, thus improving blood flow<sup>20</sup>.

Rescue teams should also be well versed in the equipment available to them and their proper functioning. Training to simulate a post-fall suspension rescue not only develops safe and effective rescue skills for team members, but helps ensure they have the necessary technique and understanding to perform self-rescue if needed. Repeated exposure to these situations also improves a rescue team's ability to adapt to different situations, which in both industrial and recreational settings can be a constant challenge. In circumstances where a rescuer is lowered to the patient to assist, helping position the patient into a horizontal position or giving them a foothold may be beneficial, however this should not delay prompt rescue.

## **6. MANAGEMENT OF SUSPENSION TRAUMA: BEST PRACTICES**

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Once rescue has been safely performed, ALS protocols should be followed to ensure proper ABC protection, and patients should be placed in a fully horizontal position. Treat for hypothermia and provide oxygen and IV fluids so long as this does not interfere with

or delay rescue. If lab values show signs of hyperkalemia or acidemia, IV fluids may need to be adjusted appropriately. Transport to facilities with dialysis should be considered for patients who have been suspended for extended periods of time<sup>7</sup>.

Finally, it cannot be stressed enough, that at no point should rescue or treatment be delayed in order to prevent Rescue Death or Reflow Syndrome. Until proven otherwise, the most recent scientific evidence suggests that these are theoretical risks and that the complications of delaying rescue to accommodate for them far outweigh any small hypothetical gains.

## **7. CONCLUSION**

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Suspension Trauma is a manifestation of shock resulting in relative hypovolemia and syncope due to increased blood pooling in the lower extremities. As a result of prolonged immobility, the body's natural homeostatic mechanisms are unable to appropriately regulate blood distribution, leading to organ hypoperfusion, syncope, and death. Additional external factors such as tangled equipment, physical injury, hypothermia, patient training level, haemorrhage, or fatigue can exacerbate the situation, causing syncope in a matter of minutes<sup>2,9</sup>. While the pathophysiology of Suspension Trauma is complex, the possibility of Rescue Death has been a matter of debate for over 40 years<sup>14</sup>. Original evidence, hypotheses, and recommendations as recent as 2002, suggested that post-suspension patients should be placed on a 30° incline rather than fully horizontal for treatment. However, more evidence continues to surface calling this into question, with new guidelines stating that there is insufficient evidence to support the concept of Rescue Death. Patients should be treated with standard shock protocol, in a fully horizontal position, and treatment should never be delayed to account for any theoretical risks of Rescue Death.

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## GLOSSARY OF TERMS

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- Acidemia:** An abnormally high amount of acid in the blood resulting in low pH.
- Artery:** Any of the muscular-walled tubes forming part of the circulation system by which blood (mainly that which has been oxygenated) is conveyed from the heart to all parts of the body.
- Capillary:** Any of the fine branching blood vessels that form a network between the arteries and veins.
- Cardiac Arrhythmias:** Irregular heart beats due to improperly coordinated electrical impulses.
- Caval Veins:** Referring to both the Superior Vena Cava and Inferior Vena Cava, the two largest veins returning blood from the body to the heart.
- Creatine Phosphokinase:** An enzyme found in the heart, brain, and skeletal muscles.
- Effective circulating volume (ECV):** The volume of arterial blood that is effectively perfusing tissue.
- Fascia:** A band or sheet of connective tissue, primarily collagen, beneath the skin that attaches, stabilizes, encloses, and separates muscles and other internal organs.
- Glomerular filtration:** The first step in the production of urine whereby the kidneys filter excess fluid and waste products out of the blood into the urine collecting tubules for excretion.
- Haemorrhage:** An escape of blood from a ruptured blood vessel, especially when profuse.
- Hyperkalemia:** Abnormally high level of Potassium in the blood.
- Hypertension:** Abnormally high blood pressure.
- Hypoperfusion:** Inadequate blood flow to body tissues, resulting in an inadequate supply of oxygen and nutrients to these tissues.
- Hypotension:** Abnormally low blood pressure.
- Hypovolemia:** A decreased volume of circulating blood in the body.
- Hypoxemia:** Abnormally low concentration of oxygen in the blood.
- Intrapleural:** The potential space between the outer lining of the lungs, called the visceral pleura, and the inner lining of the chest wall, called the parietal pleura.
- Lactic Acidosis:** An abnormally high level of lactic acid in the blood resulting in low pH.
- Myoglobin:** An iron- and oxygen-binding protein found in muscles.
- Oedema:** A condition characterized by an excess of watery fluid collecting in the cavities or tissues of the body.
- Perfusion:** The circulation of blood through the capillary vascular bed of tissue.
- Pulmonary Embolism (PE):** A sudden blockage of a lung artery, usually caused by blood clots.
- Pulmonary vasculature:** The blood supply network to the lungs.
- Rhabdomyolysis:** The destruction of striated muscle cells.
- Syncope:** A temporary loss of consciousness caused by a fall in blood pressure.
- Tunica media:** New Latin for "middle coat." The middle layer of an artery or vein between the tunica intima on the inside and the tunica externa on the outside.
- Vascular:** Of, relating to, affecting, or consisting of a vessel, especially those that carry blood.
- Vasoconstriction:** The constriction of blood vessels, which increases blood pressure.
- Vein:** Any of the tubes forming part of the circulation system by which blood (mainly that which has been deoxygenated) is conveyed from the body back towards the heart.

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