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THE LONG TIME SUSPENSION TRAUMA: A REVIEW

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Introduction. Suspension trauma, also referred to as a harness-induced pathology or suspension syndrome, occurs when an individual is suspended motionless in a harness. Potentially fatal outcomes of such a condition consist in venous pooling, cerebral hypoperfusion, and rhabdomyolysis.

Objective. To review literature sources on the key mechanisms of suspension injury and potential methods for improving the safety of people at risk.

Results. This condition, recognized since the 1970s, affects individuals involved in activities requiring harness use, such as climbing and industrial work. Recent studies have emphasized the need for immediate horizontal positioning during rescue to restore blood flow and prevent complications. Proper management of hyperkalemia and rhabdomyolysis has become a crucial focus in treatment protocols. Additionally, recognition of the role of the Bezold–Jarisch reflex in cardiovascular collapse highlights the importance of comprehensive rescue strategies. Advances in harness design are also noted as significant preventive measures.

Discussion. The findings indicate that while early management strategies focused on preventing sudden blood return to the heart by maintaining an upright position, more recent insights emphasize the importance of prompt horizontal repositioning. The role of neurocardiogenic factors, such as the Bezold–Jarisch reflex and the influence of rhabdomyolysis-related hyperkalemia, on outcomes has been recognized. This shift reflects an increased awareness of comprehensive rescue protocols that might mitigate risks associated with reflow syndrome and cardiovascular instability.

Conclusions. The progress in understanding suspension injury has significantly improved prevention and treatment protocols. Immediate adjustment of the victim to a horizontal position, proper treatment of complications (for example, hyperkalemia), and improved design of safety systems — all play a key role in minimizing deaths. Further studies should be aimed at investigating the main pathogenetic mechanisms of suspension syndrome and development of advanced rescue methods for improving the safety of people at risk.

Keywords: harness-induced pathology; harness use; industrial safety; prolonged suspension; recreational accidents; suspension shock; suspension syndrome; suspension trauma

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ТРАВМА ПОДВЕШИВАНИЯ В ТЕЧЕНИЕ ПРОДОЛЖИТЕЛЬНОГО ВРЕМЕНИ: ОБЗОР

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Введение. Травма подвешивания, также известная как синдром зависания в обвязке или синдром подвешивания, возникает, когда человек подвешивается неподвижно в страховочных стропах, что приводит к потенциально фатальным последствиям, таким как венозный застой, церебральная гипоперфузия и рабдомиолиз.

Цель. Изучить по данным литературы основные механизмы возникновения травмы подвешивания и потенциальные методы повышения безопасности лиц в группе риска.

Результаты. Это патологическое состояние, впервые упоминаемое еще в 1970-х годах, возникает у людей, занимающихся видами деятельности, требующими использования страховочных систем, такими как альпинизм и промышленные работы. Согласно последним исследованиям в ходе оказания помощи для восстановления кровотока и предотвращения осложнений необходимо немедленно перевести пострадавшего в горизонтальное положение. Надлежащая коррекция гиперкалиемии и рабдомиолиза стала ключевым направлением в протоколах лечения. Кроме того, признание роли рефлекса Бецольда — Яриша в развитии сердечно-сосудистого коллапса подчеркивает важность комплексных стратегий спасения. В качестве важных превентивных мер также отмечаются достижения в разработке более совершенных и безопасных страховочных систем.

Обсуждение. В то время как первоначальные стратегии лечения были направлены на предотвращение внезапного возврата крови к сердцу путем поддержания вертикального положения, последние исследования подчеркивают важность своевременного горизонтального положения. Роль нейрокардиогенных факторов, таких как рефлекс Бецольда — Яриша и влияние гиперкалиемии, связанной с рабдомиолизом, на исход подчеркивают эволюционирующее понимание патофизиологии. Этот сдвиг отражает возросшую осведомленность о комплексных протоколах спасения, которые снижают риски, связанные с синдромом восстановленного кровотока и сердечно-сосудистой нестабильностью.

Выводы. Прогресс в понимании травмы подвешивания значительно улучшил протоколы профилактики и лечения. Немедленное переведение пострадавшего в горизонтальное положение, надлежащее лечение осложнений (например, гиперкалиемии) и усовершенствованная конструкция страховочных систем играют ключевую роль в минимизации летальных исходов. Продолжение изучения основных механизмов патогенеза синдрома подвешивания и разработка новых методов спасения имеют большое значение для дальнейшего повышения безопасности лиц в группе риска.

Ключевые слова: патология страховочной системы; использование страховочных систем; охрана труда; длительное подвешивание; несчастные случаи на отдыхе; шок подвешивания; синдром подвешивания; травма подвешивания

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INTRODUCTION

Suspension trauma, also referred to as harness-induced pathology or suspension syndrome, is a potentially fatal condition that occurs when an individual remains suspended in a harness for a prolonged period of time. Suspension trauma occurs relatively rare in well-regulated industrial settings due to effective prevention strategies and proper equipment.

Over an 11-year span of observation, 5.8 million hours of using a harness by qualified personnel in different fields were recorded. During this period, no episodes of syncope or injuries associated with prolonged suspension were registered [1]. This could be related to efforts from employers to ensure the safety of their staff members. Only a few cases of this complication during the mentioned time span were reported from healthy individuals in sporting accidents and recreational activities in cases of poor training and improper use of harness [2, 3]. The mortality rate associated with suspension trauma varies widely depending on several factors, including the duration of suspension, the condition and comorbidities of the patient, and the promptness of rescue. Although accurate statistical data is lacking, some studies suggest that the risk of severe injury or death increases significantly following approximately 15 minutes of suspension. If the patient is not rescued within half an hour, the mortality rate can increase up to 50%. Timely rescue and pertinent management are crucial for improving patient outcomes [4].

In this review, we carry out an analysis of the evolving understanding of suspension trauma, focusing on key scientific advancements, treatment protocols, and the pathophysiological mechanisms involved. Importantly, no cases of suspension trauma from Poland have been documented so far.

Historical Development

The first accounts of suspension trauma were reported during the Second International Conference of Mountain Rescue Doctors in 1972. This pivotal moment raised awareness about the dangers associated with immobile suspension in a harness, particularly for climbers and cave explorers. One report at that conference informed that out of the 10 individuals who had been trapped in a suspended position, two died before rescue, three died immediately afterward, and five were pronounced dead over the next several days [2, 4]. Early hypotheses attributed these fatal cases to circulatory collapse, encouraging researchers to investigate the underlying causes and outcomes of suspension trauma.

The term “suspension trauma” has gained widespread recognition, particularly in high-risk industries and activities involving harness use. It was initially thought that the harness itself induced a “tourniquet effect,” compressing major veins and arteries, which in turn led to circulatory shock [3]. However, this initial assumption was challenged by later research indicating that venous pooling, rather than mechanical compression from the harness, was a significant factor in the development of suspension trauma [1, 5].

Pathophysiology

The physiological response to prolonged vertical suspension is complex, involving several mechanisms. When a person is suspended motionless in a vertical position, gravity causes blood to pool in the lower extremities. This pooling leads to a decreased venous return to the heart, resulting in a significant reduction in cardiac output and cerebral perfusion. During movement, venous pressure in the foot typically measures approximately 25 mmHg; however, in the state of immobility, it can exceed 90 mmHg [6]. This pooling can result in up to 20% of circulating blood volume

being retained in the lower limbs, leading to a reduction in venous return to the heart, which decreases preload and, consequently, cardiac output. As cardiac output declines, there is a reduction in systemic blood pressure, which severely compromises cerebral perfusion [7]. The body attempts to compensate for this situation by increasing the sympathetic tone, which causes the heart rate to accelerate. However, these compensatory mechanisms are often insufficient to counteract the effects of sustained venous pooling. As cerebral blood flow continues to drop, the brain experiences reduced oxygen delivery, leading to presyncopal symptoms such as dizziness, light-headedness, blurred vision, nausea, and sweating. Given that the individual remains suspended and corrective actions are not taken, these symptoms can quickly progress to syncope (fainting) due to critical hypoperfusion. If unconsciousness ensues, there is a significant risk of death, particularly if the suspension position compromises airway patency or worsens cardiovascular instability [1, 5].

The Bezold–Jarisch reflex, which triggers bradycardia and hypotension in response to reduced blood flow to the heart, is thought to play a key role in the rapid collapse experienced by those affected by suspension trauma [6, 7]. Once the individual loses consciousness, they cannot move into a horizontal position, exacerbating the problem and leading to further reductions in cerebral perfusion and oxygenation [3, 1].

However, the hypothesis that a reduction in cardiac preload, and consequently a decrease in cardiac output, is the primary cause of loss of consciousness and other injuries has not been confirmed so far. Recent reviews highlight the importance of neurocardiogenic mechanisms leading to reduced cerebral perfusion and loss of consciousness. In the suspension trauma syndrome, significant effects on systemic hemodynamic parameters, such as compensatory tachycardia and reduced stroke volume — typically associated with low cardiac preload — are not observed [8].

While the Bezold–Jarisch reflex may be linked to suspension syndrome, experimental studies have provided sufficient evidence. The mechanoreceptors responsible for this reflex, located in the left ventricle, respond to poor ventricular filling [9]

The mechanism of post-rescue death remains to be unclear. The available literature indicates that the sudden return of acidotic blood, which accumulates in the veins of the lower body, to the heart is capable of temporarily depressing cardiac contractility. However, this is not associated with heart rhythm abnormalities. Published review papers highlight rhabdomyolysis as a key concern in the suspension trauma syndrome, primarily resulting from reduced blood flow and muscle damage, leading to the release of substances such as myoglobin and potassium. The higher risk of death due to suspension trauma can relate to the suspension duration of more than 30 minutes, height of more than five feet, and older age.

Clinical Observations and Case Reports

Several case studies have provided information on the clinical manifestations of suspension trauma. For example, a notable case from 2011 involved a climber found unresponsive in his harness after being suspended for several hours. The autopsy results revealed the death to result from mechanical asphyxia, a consequence of immobility and venous pooling [10]. Another series of incidents reported climbers who experienced “rescue death” shortly after being rescued, highlighting the delayed effects of suspension trauma [12].

In addition to venous pooling and circulatory collapse, rhabdomyolysis — the breakdown of muscle tissue due to immobility — has been frequently associated with suspension trauma. This condition releases myoglobin into the bloodstream, which can lead to acute renal failure and complicate treatment [2, 13]. Elevated potassium levels (hyperkalemia) caused by rhabdomyolysis can also lead to fatal cardiac arrhythmias [13].

Management and Treatment

For decades, the standard treatment protocol for suspension trauma has emphasized the importance of keeping victims in an upright position after rescue. This approach was based on the belief that lying the victim down would cause a sudden return of pooled blood to the heart, leading to cardiac overload and rescue death [5, 9]. The protocol was formulated in 1970s on the basis of observational studies and opinions of experts largely from nonmedical fields [2, 3, 9]

The study by Pasquier et al. (2010) demonstrated the significance of airway management and fluid resuscitation, consistent with ALS guidelines, over other interventions in rescuing injured individuals. However, there is a lack of scientific evidence to support the claim that positioning a patient horizontally during assistance increases the risk of death [14].

The guidelines of the International Commission for Mountain Emergency Medicine (ICAR MedCom) have also been updated. In the latest version, one of the key recommendations is to position victims horizontally in a proper manner. This measure restores cardiac output and prevents the effects of venous stasis. Additionally, initiating resuscitation procedures at the earliest sign of cardiac arrest is a top priority, including treatment for hyperkalemia and potential rhabdomyolysis [15].

Prevention and Harness Design

An essential component in preventing suspension trauma is the use of appropriate harnesses with multiple attachment points and adjustable straps, as well as devices in the type of leg loops. Such equipment allows for greater mobility, reducing the risk of venous blood pooling.

Equally important is the proper education and training of both harness users and rescue teams. Users should be informed of early symptoms of suspension trauma, such as hot flashes, sweating, and dizziness, and be reminded to move their legs while suspended [3, 1]. Rescue teams should be trained to quickly reposition the injured individual and minimize the duration of immobility [1, 5].

Research Trends and Future Directions

The current understanding of suspension trauma has improved significantly. However, treatment and prevention measures are constantly under discussion. One of the key areas of debate is whether there are specific variables, such as duration of suspension, type of harness, or pre-existing medical conditions, that increase the risk of severe outcomes, including rescue death [1, 9, 16]

A promising area of research focuses on the role of reflow syndrome, also known as rescue death. This syndrome describes the potential for sudden cardiac failure following the return of pooled, deoxygenated blood to the heart [8, 17, 18]. Earlier studies hypothesized that rescue death could result from placing the victim in a horizontal position too quickly. More recent findings, however, suggest that this phenomenon is more likely related to hyperkalemia and acidosis, both of which can arise from the release of muscle breakdown products during rhabdomyolysis [2, 13, 19]. Continued research into these mechanisms, including studies of human physiology during suspension, is needed to further refine rescue protocols. Understanding of the key mechanisms, such as the role of inflammatory cytokines and oxidative stress in reperfusion injuries, and the effects of prolonged suspension on electrolyte imbalances and muscle degradation are promising research directions for optimizing rescue techniques and improving patient outcomes.

According to current studies, including randomized controlled trials, rapid recovery of blood flow to the brain and heart can be safely achieved by laying the victim flat, countering earlier concerns about rapid reperfusion injury [16]. However, challenges in conducting long-term human studies due to ethical concerns limit the analytical data about severe cases and long-term consequences.

The evolving prevention strategies include improved harness designs that allow suspended individuals to acquire semi-horizontal positions or activate leg muscles to prevent venous pooling. Research into the ergonomic design of harnesses continues, with efforts focused on improving comfort, reducing pressure on the femoral veins, and facilitating faster self-rescue or external rescue. These advances will likely reduce the number of fatal and severe cases of suspension trauma in the future [1, 19].

CONCLUSION

Suspension trauma, a condition once poorly understood, is now recognized as a complex medical phenomenon that requires timely and informed intervention to prevent fatal outcomes. Over the years, the pathophysiological processes occurring in suspension trauma, such as venous pooling, cerebral hypoperfusion, and the Bezold–Jarisch reflex, have been elucidated. Modern harness designs, improved rescue techniques, and updated medical protocols have significantly reduced the risk of death associated with suspension trauma, although further research is required to optimize treatment and prevention strategies.

While earlier practices focused on keeping victims upright after rescue, contemporary guidelines recommend immediate horizontal positioning and followup care to manage conditions such as hyperkalemia and rhabdomyolysis. As research continues and harness technology advances, the risks posed by suspension trauma are expected to diminish. At the same time, awareness and preparedness remain critical in minimizing the dangers faced by workers and adventurers using harness systems.

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