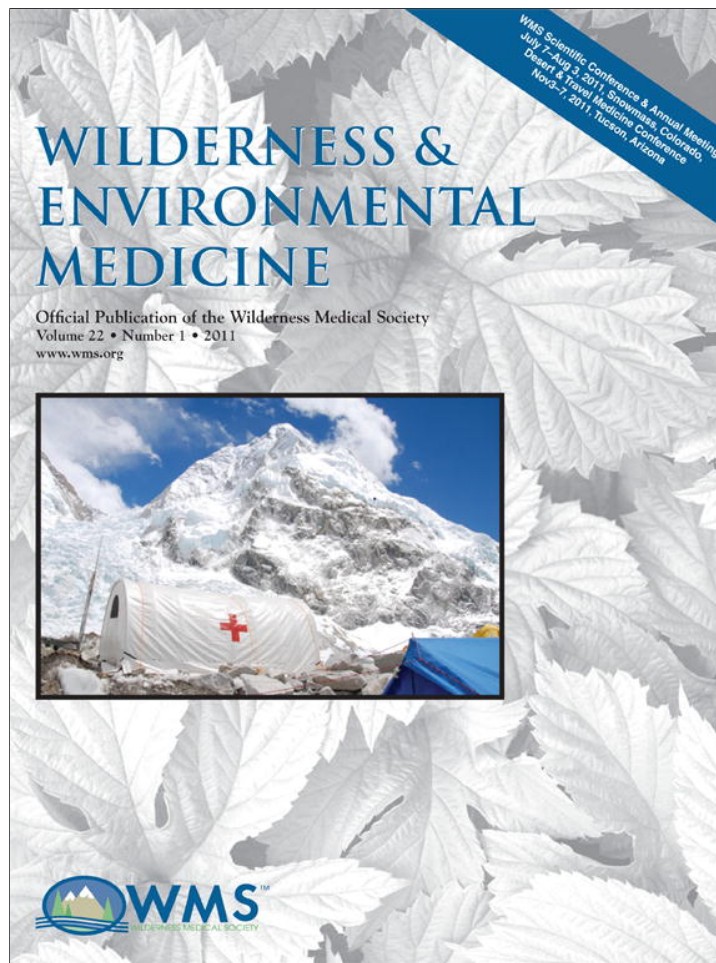


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## REVIEW ARTICLE

# Risks and Management of Prolonged Suspension in an Alpine Harness

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Suspension trauma is a state of shock induced by passive hanging. Those who survive passive suspension are at risk for rhabdomyolysis. In a wilderness setting, one can see this in cases of persons suspended on rope by their harness. In a conscious person, leg movements work the venous pump to return blood to the central circulation. In the person passively hanging, blood pools in the legs leading to hypoperfusion of vital organs. In the experimental setting, passive hanging has led to unconsciousness in a matter of minutes. Based on a previous series of deaths on rope that included 7 after rescue, many authors have recommended nonstandard treatment for shock including keeping rescued patients upright or squatting for 30 minutes prior to laying them down. This recommendation assumes that sudden death is a risk from acute volume overload or exposure to waste products in the returning blood. This suggestion is not supported by the original series that demonstrated sudden deaths after rescue nor by modern understandings of physiology. Search and rescue teams and party members assisting a colleague suspended unconscious on rope should follow standard resuscitation measures to restore circulation to vital organs immediately.

*Key words:* shock, orthostasis, vasovagal, harness, rhabdomyolysis, suspension trauma

## Introduction

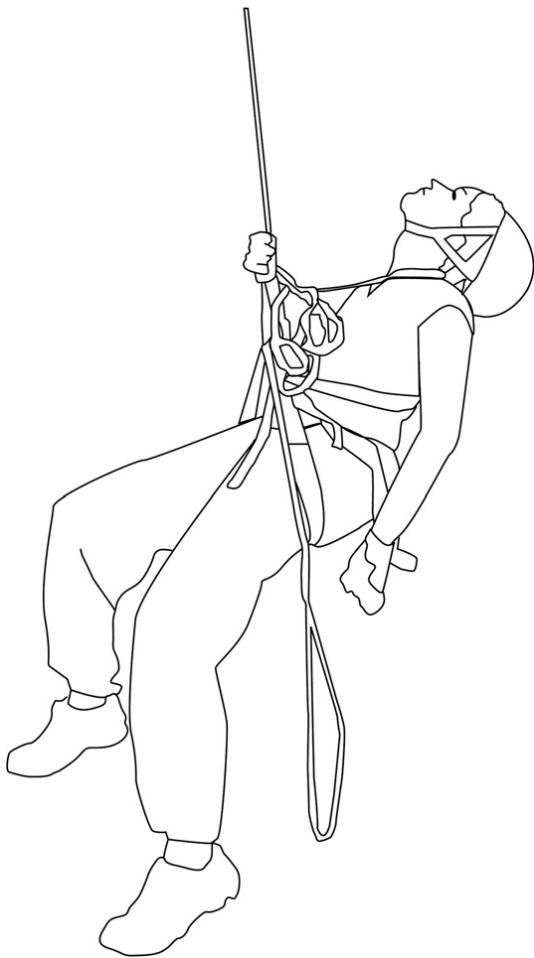
Harness suspension is an accepted and necessary part of rock climbing, ice climbing, mountaineering, canyoneering, and caving, and many industrial applications. The harness provides a soft interface with a life-supporting rope in actual or potential high-angle environments. In the event of a fall or loss of consciousness, the harness maintains attachment to the rope allowing the person to climb again or be rescued (see [Figure 1](#)). While remaining suspended and unconscious is preferable to falling, it has its own risks and management issues.

The idea of suspension trauma, shock leading potentially to death, induced by hanging passively in a harness, has been around for many years. It has been variously called orthostatic intolerance, harness pathology, and harness hang syndrome. Suspension trauma has become the most common name despite the lack of true

trauma in most cases. Terms using the word harness ought to be avoided because, as will be discussed, it is not really the harness that is at issue.

Suspension trauma has been described independently several times. Early autopsies of persons who died on rope in Austria and Spain found minimal trauma and concluded that they had died of shock.<sup>1–3</sup> The medical commission of the French Federation of Speleology also studied reports of cavers who died on rope.<sup>4</sup> Originally they felt that the cavers died of hypothermia as 10 of 12 cases were in pits with water coming down, but several lost consciousness too quickly to have been caused just by hypothermia.<sup>4</sup> American caving casualties are similar.<sup>5–11</sup> American mountaineering cases are less clear.<sup>12–14</sup> The French tried to replicate the circumstances in a lab setting by instructing the participants to act as if unconscious while suspended on rope. Their first 2 participants became unconscious in 7 and 30 minutes.<sup>4,15</sup> They stopped to reconsider their protocol. Taking it up again 2 years later in a monitored setting in a hospital, another participant lost consciousness after 6 minutes. They concluded that hypothermia was not the sole cause of death of these cavers.<sup>4,15</sup>

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**Figure 1.** Passive Hanging on Rope.

Others have approached suspension trauma from a military or occupational point of view. Orzech et al, studying parachute harnesses, had 1 individual become unconscious after 6 minutes in a body harness.<sup>16</sup> Damisch and Schauer<sup>17</sup> performed 46 suspension tests in various harnesses for up to 10 minutes. No one lost consciousness but 2 wearing chest harnesses alone had to be lowered without detectable blood pressure in 5 and 9 minutes.<sup>17</sup> Madsen et al<sup>18</sup> did a series of 69 persons, not suspended in harnesses, simply lying down on a tilt table at 50°, supported by a bicycle seat. Each was instructed to abstain from movement. These participants experienced presyncopal symptoms (nausea, lightheadedness, or feeling hot) or signs (pallor, bradycardia, or hypotension) in a median of 27 minutes. This strongly suggests that passive suspension is the risk, not the harness.

Anyone roped into a harness in a vertical environment is at risk, but some have higher risks. In general, people tolerate alpine-type sit harnesses better than full-body harnesses, which are better than chest harnesses alone, which are much better than simple waist belts.<sup>16,19–21</sup>

Intolerance of chest harnesses and waist belts is more an issue of pain and respiratory compromise than of suspension trauma per se.<sup>19,22</sup> Any condition that decreases central volume prior to suspension, such as dehydration or hypothermia, increases risk.<sup>23</sup> Support under the knees that elevates the legs seems to protect.<sup>18</sup> Gender does not affect risk, but increased weight has led to decreased mean arterial pressure in the lab setting.<sup>24</sup> Though unlikely to be used in the wilderness environment, industrial-use full-body harnesses with attachments in the back delay suspension trauma longer but do not completely prevent it.<sup>24</sup> For issues related to industrial applications of harnesses, the reader is referred to the review by Seddon.<sup>25</sup>

### Pathophysiology

At the 2nd International Conference of Mountain Rescue Doctors in 1972, the underlying physiology was first proposed.<sup>1–3,26,27</sup> Patscheider found little trauma in those who died on rope and concluded that they had died of shock.<sup>1</sup> Similarly, Toledo y Ugarte found no significant trauma but did find lower body plethora in a victim who died on rope.<sup>3</sup> In the normal person, venous return happens through muscular contractions forcing blood through the one-way valves of the lower extremity veins. This venous pump is disabled in the motionless patient while arterial flow continues. Subsequent work shows that suspension leads to decreased involuntary small muscle contractions normally used to maintain blood pressure when upright.<sup>28</sup> Failure of the venous pump leads to pooling of blood in the legs with decreasing central volume as demonstrated by enlarging thighs,<sup>24</sup> decreasing heart size,<sup>27,28</sup> decreasing stroke volume,<sup>28</sup> decreasing glomerular filtration rate,<sup>26</sup> and increasing transthoracic impedance.<sup>18</sup> Once capillary pressures rise, significant fluid can leak into interstitial spaces, decreasing total intravascular volume.<sup>29</sup> With decreasing stroke volume in a hyperautonomic state, the person becomes subject to the Bezold-Jarisch reflex, which triggers decreased heart rate and blood pressure.<sup>30</sup>

Adaptive reflexes can be pathological in the artificial situation of hanging motionless. In normal circumstances, acidosis from anaerobic metabolism decreases vascular resistance.<sup>31</sup> Decreased resistance usually leads to increased blood flow with concomitant increases in available oxygen and nutrients along with removal of waste products. In the motionless hang situation, increased flow sequesters even more blood in the periphery.

Central hypovolemia eventually leads to fainting. The vasovagal response to poor circulation normally returns one to a horizontal position, which improves blood flow

**Table 1.** Survivors after prolonged suspension on rope

Activity	Age/gender	Time on rope	Outcomes	Reference
Rappelling	26/M	1.5 hours	Nerve damage from chest harness, acute renal failure	34
Mountaineering	25/M	0.5 hour	Nerve damage from chest harness	34
Mountaineering	19/M	20 minutes	Nerve damage from chest harness	34
Mountaineering	28/M	2 hours	Nerve damage, acute renal failure	34
Mountaineering	31/NA	15 minutes	Nerve damage, acute renal failure	34
Mountaineering	43/NA	10 minutes	Minor trauma	34
Mountaineering	20/NA	5 minutes	Minor trauma	34
Mountaineering	36/NA	15 minutes	Minor trauma	34
Mountaineering	47/NA	1 hour	Nerve damage, hematuria	34
Mountaineering	28/NA	5 minutes	Minor trauma	34
Mountaineering	30/NA	3.5 hours	Shock	34
Mountaineering	24/NA	20 minutes	Minor trauma	34
Mountaineering	24/NA	20 minutes	Minor trauma	34
Caving	28/M	2 hours	Unknown	5
Caving	29/M	5–6 hours	Rhabdomyolysis, nerve damage	8
Caving	42/M	< 5 hours	None	8
Caving	18/M	4 hours	Rhabdomyolysis	9
Mountaineering	25/M	1 hour	Major trauma	12
Mountaineering	18/M	Unknown	Nerve damage	14

N/A, not available.

to the brain. Soldiers at attention fainting on the parade ground are classic examples of this—once they are down they rapidly regain consciousness. The suspended person, however, can fall no farther—decreased heart rate and blood pressure from increased vagal tone simply results in yet more catastrophic flow reduction.

In the experimental setting, one sees evidence of increasing sympathetic tone followed by a parasympathetic response. Increased sympathetic tone leads to increased heart rate to compensate for decreasing volume. Pulse pressure narrows. Finally, blood pressure decreases either as a result of the decreased available volume, or more catastrophically from a vasovagal response including bradycardia. Symptomatically, patients report nausea, lightheadedness, and flushing.<sup>4,16,17,19,21</sup> Madsen et al report that half of their 69 tilt-table patients were presyncopal within 27 minutes, with pulses between 30 and 57 beats per minute.<sup>18</sup>

While likely multifactorial, the fainting response may be partly due to the Bezold-Jarisch reflex.<sup>30</sup> This reflex is mediated by receptors in the posterior left ventricle that sense volume. At normal volume, they fire tonically to control blood pressure. As volume decreases, they fire less to allow vasoconstriction. When stroke volume decreases dramatically, they fire more resulting in bradycardia, vasodilation, and hypotension. This reflex has been blamed for bouts of hypotension and bradycardia and even asystole in patients having shoulder surgery in

a sitting position, not that far removed from the tilt-table experience.<sup>32</sup> The reflex can be demonstrated in an animal model by ligating the inferior vena cava.<sup>33</sup> Once volume is sequestered peripherally, receptor cells abruptly fire more and blood pressure and heart rate drop. When the occlusion is released and volume returns, the receptors fire less, and vital signs return towards normal.

Once off rope, several outcomes have been observed. Most recover uneventfully. Some have had sub-acute sequelae like rhabdomyolysis and renal failure.<sup>34</sup> Of 19 long-term survivors, 3 suffered from renal failure, 1 had hematuria, and 2 others had rhabdomyolysis without renal failure (see Table 1). Long-term stasis eventually leads to muscle cell necrosis with release of myoglobin, in turn leading to renal failure by a variety of mechanisms.<sup>35</sup>

More concerning are the reports of those who were alive after rescue but died soon thereafter. Flora and Holzl<sup>34</sup> accumulated a series of 10 deaths associated with prolonged suspension, of whom 7 died after rescue.<sup>34</sup> One survivor died 11 days after rescue from renal failure, diagnosed as a crush syndrome on autopsy. Six others died from a few minutes to 32 hours after being rescued. French and American caving accident reports document 3 other initial survivors, 1 of whom died immediately after rescue.

There may be some overlap of suspension trauma with compression asphyxia in which death is caused by inad-

equate ventilation from outside constriction.<sup>36</sup> Suspension in a chest harness alone does lead to decreases in forced vital capacity, heart rate, blood pressure, and cardiac output. These changes are not observed in participants wearing a sit harness.<sup>22</sup> So cases in a chest harness alone may include a degree of compression asphyxia. But as Patscheider notes, many of the victims in the Austrian series in chest harnesses alone were still able to call out while suspended.<sup>1</sup> Orzech et al<sup>16</sup> also noted breathing difficulties only in those suspended by a body belt alone and not in those suspended in a chest harness or sit harness.<sup>16</sup> In the 12 French cases reported by Bariod that launched their search for a cause of quicker than expected deaths on rope, only 1 was suspended in a chest harness after he undid his attachment with his sit harness.<sup>4</sup> Airway constriction itself is unlikely given the hyperextended position of the neck in passive hanging in a harness (see Figure 1). So restricted breathing may play a part in deaths on rope, but is unlikely to contribute much to those cases in sit harnesses.

Some have suggested that the sequestration of blood is due to a tourniquet effect from the harness.<sup>37–39</sup> This seems unlikely for several reasons. Orzech et al<sup>16</sup> found similar effects despite multiple harness types and fits, as did Nelson.<sup>19</sup> The phenomenon has been seen when no harness was involved.<sup>40–42</sup> Climbers routinely spend an entire day in a harness and can be suspended for hours at a time. Although this can be very uncomfortable, it has not proved dangerous while the climber is conscious, despite the same constriction of the harness around the legs. In the 2 cases of immediate death after rescue, neither person was using a sit harness, so removing such a harness had nothing to do with these 2 deaths. Most importantly, in alpine style harnesses with front attachments there is no compression of the anterior thighs where the femoral veins return blood to the core circulation (see Figure 2).

The pathology of suspension trauma is not absolutely clear. Other factors that can help precipitate an accident, such as drugs and alcohol, can worsen maladaptive responses. Especially in the early cases where no sit harness was used, respiratory function can also be compromised in the unconscious person on rope. What does seem clear is that passive suspension does lead to sequestering of volume in the periphery, hypotension, bradycardia, and, in the worst cases, death. Survivors are at risk for rhabdomyolysis and renal failure.

### Management

The most critical part of suspension trauma management is to get the unconscious person down from the sus-



Figure 2. Leg vessels in relation to harness straps.

pended position. This might mean lowering a belayed person down or raising someone up to anchors. The best approach is to rig in such a way that little rerigging is necessary to move the patient. A belayed person can usually be lowered immediately using the belay device itself, though in certain situations it may not be possible to lower them to within reach. A self-belay device prevents an unconscious rappeller from crashing but leaves them unconscious on rope. The more common scenario is someone climbing a fixed line who becomes injured,

cold, or exhausted from poor equipment or poor technique. It is much more difficult to deal with the person stuck on a fixed line. It is best to leave adequate rope at the top of the drop to quickly create a hauling system and bring them up, or if prepared with the skill and equipment to mitigate the situation, to go down to the patient to help. For training situations, ropes should be rigged to lower anyone experiencing difficulty immediately. In extreme cases, it may be necessary to climb or descend to the person on rope, transfer them to one's own harness or to a lowering device fixed on the rope, and lower them to the ground.<sup>43,44</sup> This type of on-rope pick-off requires training and constant practice to do efficiently.

Once off the rope, medical management is less clear. No one disputes the standard need to address airway and breathing. Restoring pooled blood into the central circulation is more controversial. In almost all cases of shock, one ought to lay patients flat if not raise their legs to encourage blood flow to the heart and brain. This was successfully done in the lab by Orzech et al,<sup>16</sup> Madsen et al,<sup>18</sup> and Stuhlinger et al.<sup>26</sup> But as early as 1972, Patschieder suggested avoiding placing a patient "abruptly" in a horizontal position and other presenters at the same conference called for them to be placed in a squatting position.<sup>1,45</sup> This call is echoed by Seddon<sup>25</sup> and others citing Seddon.<sup>38,40,46</sup>

These authors cite a concern for what has been termed "rescue death." This seems to be in response to the Flora and Holzl's series of 10 deaths on or soon after hanging on rope.<sup>34</sup> At least 3 of the 7 who were rescued had a normal mental status up to the time of death.<sup>34</sup> Table 2 summarizes known cases of persons who survived a suspension but subsequently died. The most striking is the 23-year-old who died within minutes of being rescued. Her autopsy revealed no significant trauma.<sup>2</sup> She also was suspended for 4 hours in a chest harness, longer

than many who died on rope prior to rescue (see Table 3), putting in question whether the timing of her death had anything to do with her rescue or simply the consequence of the long time she spent suspended.

A second concerning case comes from *American Caving Accidents*.<sup>11</sup> A male caver got stuck on rope. He was wearing a chest harness only. Help arrived after 4 hours; however, he died suddenly when released from his chest harness. It is unclear if he was conscious or not on rescue, or whether an autopsy was done. What is worth noting is that in both these cases of the person dying postrescue, it was after 4 hours and in both cases they wore chest harnesses only, so "releasing toxins" by removing a sit harness played no role at all.

Explaining these postrescue deaths is difficult. Pulmonary embolism fits the clinical presentation of sudden death, especially after a period of stasis, but Patschieder found no evidence of clots or other mechanical obstruction in his autopsy series.<sup>1</sup> Blaisdell has shown evidence of fibrin-platelet aggregates in the lungs after reperfusion of ischemic limbs, but this causes a more medium-term inflammatory reaction and not immediate death.<sup>47</sup> Most people have focused on cardiac dysrhythmia because of the sinus arrhythmias and premature ventricular contractions (PVCs) noted on EKG after experimental patients were laid down by Stuhlinger et al.<sup>26</sup> Others, however, note PVCs while the participants were suspended, making it hard to blame the act of laying down.<sup>16,21</sup> Cardiac arrest is a plausible cause of the sudden deaths after rescue from hanging on a rope, but the link to timing after rescue is tenuous.

Several have blamed an acute volume overload for causing cardiac arrest once someone is laid down.<sup>25,38</sup> Acute volume overload in healthy dog hearts increases T-wave alternans, a marker for sudden cardiac death, but so does increased sympathetic tone, which would be

**Table 2.** Survivors of suspension who died after rescue

Activity	Age/gender	Time suspended	Time to death after rescue	Autopsy	Reference
Caving	17/M	Unclear	5 hours	No	4
Caving	NA/M	"Rapidly"	20 hours	No	4
Mountaineering	25/M	3 hours	11 days	Rhabdomyolysis	34
Mountaineering	18/M	6.5 hours	1.5 hours	Unknown	34
Mountaineering	24/F	7 hours	32 hours	No	34
Mountaineering	21/M	4 hours	2 hours	No significant trauma	34
Mountaineering	33/M	3 hours	19 hours	Not available	34
Mountaineering	23/F	4 hours	"Few minutes"	Circulatory collapse	2, 34
Mountaineering	19/	8 hours	17 hours	Not available	34
Caving	NA/M	4 hours	Minutes	Not available	11

NA, not applicable.

**Table 3.** Deaths occurring while suspended on rope

Activity	Age/gender	Time to death	Autopsy	Reference
Training	25/M	6 minutes	No	18
Mountaineering	18/M	> ½ hour	Plethora of lower vena cava	3
Mountaineering	17/M	24 hours?	Not available	34
Mountaineering	19/M	½ hour	Not available	34
Mountaineering	25/M	2 hours	No	34
Caving	15/M	< 2 hours	No	4
Caving	NA/M	20 minutes	No	4
Caving	NA	< 1 hour	No	4
Caving	~50/M	Unclear	No	4
Caving	24/M	Unclear	No	4
Caving	NA	Unclear	No	4
Caving	NA	< 1 hour	No	4
Caving	25/M	Unclear	No	4
Caving	20/M	Unclear	No	4
Caving	23/M	Unclear	No	4
Caving	29/M	Unclear	"Hypothermia"	10
Caving	26/M	> 2 hours	"Hypothermia"	6
Caving	28/M	Unclear	No	7
Mountaineering	18/M	2–3 hours	"Asphyxia by hanging"	14
Mountaineering	16/M	35 minutes	"Suffocation caused by aspiration"	13

NA, not applicable.

present in all these patients recovering from shock.<sup>48</sup> Mechanical stretch in the context of regional ischemia increases arrhythmic potential, but not when hypoxia is global as would be the case in these young, healthy mountaineers.<sup>49</sup> Realistically, survivors of suspension will be hypovolemic or euvoletic. Laying them flat will at best reinflate shrunken ventricles back to normal size. In Oberg and Thoren's cat experiments, restoring central circulation by unoccluding the inferior and superior vena cava stopped the Bezold-Jarisch reflex; it did not provoke it.<sup>33</sup> The most likely scenario is that those who died after rescue died despite rescue, not because of it.

Some blame the nature of the returning blood for sudden cardiac changes, but this seems unlikely except in extreme cases. There is no doubt that suspension trauma can lead to rhabdomyolysis and eventual renal failure (see Tables 1 and 2). In the free-hanging mountaineer there is no crushing of muscle. Muscle damage will occur from inadequate perfusion from stasis. Muscle is tolerant of 3 to 4 hours of hypoxia prior to breaking down.<sup>47,50</sup> This is reflected in the clinical experience of earthquake victims in whom crush syndrome is rare prior to 3 hours.<sup>51</sup> Table 1 demonstrates that survival was rare after 3 hours of suspension on rope. Table 2 shows much longer suspension times in those who died.

Lab data being sparse for suspension cases, it is worthwhile looking at some crush data from which one can infer issues to expect after suspension. The most con-

cerning blood component after release from crush or suspension is potassium from muscle cell leakage. Allister reports on a case of a man with legs crushed for 8 hours who went into cardiac arrest an hour after release.<sup>52</sup> Prearrest his pH was 7.15 and during resuscitation his potassium 8.0 mEq/l with peaked T-waves. He was successfully treated with bicarbonate and insulin with glucose. Brown and Nicholls report 2 cases of crush with potassiums of 6.3 and 8.8 on arrival to hospital.<sup>53</sup> Gunal et al, however, report a series of 16 crushed patients treated with saline and bicarbonate in the field with only 1 hyperkalemic patient but 9 hypokalemic ones.<sup>54</sup>

Blood returning from a hypoxic leg will be acidotic. When a leg is tourniqueted for orthopedic surgery, venous pH changes to 6.9 after 2 hours.<sup>55</sup> While this sounds concerning, it is a common anesthetic procedure, done many times a day with no complications beyond a few minutes of hyperventilation. Moreover, while acidosis may transiently depress cardiac contractility, it has little to no effect on rhythm.<sup>31</sup> Thus, pH changes are not likely to be the cause of sudden death.

In crush cases, shock will complicate care soon after release. Once an ischemic limb has regained blood flow, it becomes edematous, removing intravascular volume.<sup>50</sup>

Studies of ischemic limbs demonstrate increasing postflow edema with worsening ischemia.<sup>47</sup> Restoring good circulation early will limit the further loss of vol-

ume into the interstitium.<sup>47</sup> This means laying the patient down as soon as possible to restore flow of oxygenated blood to damaged muscle. Keeping a suspension survivor upright will simply worsen muscle damage and worsen shock in a patient already in shock.

Seddon also recommends removing the harness slowly.<sup>25</sup> This is akin to the recommendations of some to place a tourniquet on crushed extremities prior to removing the crushing debris, then loosening it slowly. For the reasons stated above, it is not the harness itself that is the problem. Many have worried about the best way to remove a harness to mitigate any release of toxins to the rest of the body. Since it is not the harness itself preventing blood flow, it really matters little when it is loosened or removed. Comfort and transport issues should dictate this rather than any concern for resuscitation.

It seems likely that any risk of sudden cardiac death comes from the hypoxic damage to the heart itself. Risk from sudden volume overload is only theoretical. Myoglobin may cause medium-term renal failure, but neither it nor acidosis will cause sudden cardiac death. Elevated potassium does seem to be a real risk, but avoiding it, if it truly can be avoided, requires keeping a person in an upright position that will continue hypoperfusion of the brain and other vital organs and worsen subsequent shock once hypoxic limbs are reperfused.

The advice to keep upright for 30 minutes ignores the fact that most of Flora and Holzl's post-rescue deaths occurred much later than 30 minutes. Flora and Holzl's case in which death occurred within minutes was in the person who had a very long suspension time.<sup>34</sup> The caving case has fewer details but also occurred after 4 hours of suspension. The evidence of prompt resuscitation in controlled environments and a better understand-

ing of the physiology involved argue against any recommendation to keep a victim of suspension trauma upright.

All agree that immediate rescue is critical with attention to scene safety, airway protection, and breathing (see Table 4). Once on the ground, the evidence supports standard measures to restore adequate circulation to the brain and other critical organs. Assess all patients for further injuries and then protect them from the environment. Standard advanced life support (ALS) guidelines should be followed.<sup>56</sup>

The original recommendations of Patschieder, Flora, Holzl, and others to avoid laying the patient down was echoed by Seddon in his extensive review of the literature.<sup>25</sup> Following this recommendation, though, puts the patient at continuing risk of shock from pooled blood remaining in the legs. Not laying a person down is a choice of avoiding a theoretical risk of cardiac arrest in favor of an ongoing and certain risk of hypoperfusion and hypoxia. Having an unconscious patient sitting up simply replicates the head up, feet down position that caused the problem to start with, which Madsen et al have demonstrated requires no harness.<sup>18</sup> Some have started to question the evidence for the original recommendation.<sup>57</sup> More recently, Britain's Health and Safety Executive performed an evidence review after searching multiple databases for cases or studies. After their systematic review, they concluded that there was evidence for syncope after head-up passive suspension but no evidence against standard resuscitation measures. Thomassen et al did an independent review of the issue and came to the same conclusion.<sup>56</sup> Health and Safety Executive has since stated that there is insufficient evidence for their original recommendation and that standard first aid practices should be followed.<sup>58</sup> Just as no one would recommend keeping the fainted soldier upright to keep sequestered blood from returning to his heart too soon, no one should recommend keeping the unconscious suspension victim in shock.

Once measures are available, aggressive fluid resuscitation is indicated to correct volume status and prevent renal failure from rhabdomyolysis in cases of extended passive hanging. In crush situations, one can attempt IV hydration prior to rescue. In the wilderness context, the first priority is to get the patient down. Once down, the patient should be bolused with isotonic saline as soon as feasible followed by hypotonic saline with added bicarbonate.<sup>59</sup> Avoid potassium use until labs demonstrate that it is needed. Anyone losing consciousness on rope should get medical evaluation, if for no other reason than to find out why consciousness was lost. Anyone suspended for a substantial time should be transported to a facility capable of dialysis.

**Table 4.** Basics of management

- 
1. Remove the person from the rope
    - a. Be sure the scene is safe or mitigate the situation
    - b. If patients can cooperate, have them move their legs and raise them up until they can be lowered
  2. Lay the patient flat and start standard advanced life support protocols
    - a. This should not be delayed waiting for any other supplies
    - b. Airway, breathing, circulation, etc.
    - c. Hypothermia prevention
  3. Oxygen, monitoring, intravenous fluid as available (alternate saline and half-normal saline with added bicarbonate)
  4. Remove the harness if preferable for evacuation
  5. Transport. If suspended passively more than 2 hours, transport to a facility capable of dialysis
-

Flora and Holzl's series,<sup>34</sup> with 7 out of 10 dying soon after rescue, is disturbing. Sudden cardiac death seems like a real possibility, but from previous hypoxia or current hyperkalemia, not sudden volume overload. Once available, cardiac monitoring is critical and cardiology consultation appropriate for any rhythm disturbance, with possible use of beta-blockers as prophylaxis of sudden cardiac death.<sup>60</sup>

### Prevention

In the industrial setting, a hanging person can more easily self-rescue and is much more likely to have other workers present who can accomplish a rescue quickly. In the wilderness setting, rescue is often more difficult. The climber, caver, or canyoneer is much more likely to be in a small group with difficult access to someone isolated on rope. Having the personal ropework skills to resolve real or potential problems is primary. Rigging to avoid hazards like waterfalls or rock or ice fall zones is critical.

Another aspect of prevention is at the level of small party organization. This dynamic may vary widely depending on situation. What is the ratio of experienced to new persons on a trip? Who will climb first? Who will climb last? Does the first person to climb a fixed rope have the skills and gear necessary to effect a rescue from above if needed? What communal gear that might aid such a rescue is appropriate and who should carry it? How does one balance rest and rehydration versus risk of hypothermia while waiting to climb in a cold environment?

Once someone is stuck on rope, rescuers have the option of raising them, lowering them, or going to get them.<sup>61</sup> Whenever possible, one should rig in such a way that allows flexibility in response, eg, leaving extra rope at the top of a fixed line instead of lying at the bottom of a pit or canyon. This gives team members an easier way of creating a haul system to raise a stranded person or rappel down to assist. Instead of hard anchoring a fixed line, one can use a rope tied off in a rappel device so that an impaired climber can be easily lowered. Nonetheless, even with the best preparation and rescue technique, persons passively suspended on rope long enough may develop shock symptoms.

Climbers in a harness for a prolonged time should work on flexing their legs to pump venous blood.<sup>56</sup> An etrier will allow climbers to stand up in situations where they would otherwise be hanging for a long time such as during a prolonged rappel or for someone serving as a litter attendant during a prolonged extrication. Elevating the legs will decrease dependence on the venous pump. Madsen et al<sup>18</sup> found that adding a strop beneath the knees during a simulated vertical lift made suspension

tolerable to 60 minutes for 8 of 9 participants.<sup>18</sup> Someone in a prolonged suspension situation who felt at risk could improvise something similar with available cordage.

Leg elevation and leg use are also important to avoid iatrogenic suspension trauma in patients being rescued. Rescues involving a torso strop should incorporate the under-the-knees strop as well. A patient in a litter should be kept flat as much as possible and encouraged to use leg muscles as much as is safe. In an evacuation with any chance of the litter being put into a vertical or even semivertical position, a foot loop should be supplied for each nonbroken leg for patient comfort but also to give the patient something to push against to maximize the venous pump. In an unconscious person who will need to spend prolonged time in a vertical position during rescue, shock trousers may be indicated.

### Conclusions

Some have questioned the existence of suspension trauma.<sup>39</sup> It is clear though that persons suspended limp in a harness can die more quickly than expected and with no significant trauma. Suspension experiments demonstrate how this can happen. This is a shock syndrome in its early phase complicated by rhabdomyolysis in its late phase survivors. The shock is secondary to failure of the venous pump to return sufficient volume to central circulation in someone limp or inactive. The harness itself is not to blame.

Treatment starts with immediate rescue of a suspended person. Standard ALS procedures apply. Time suspended should not change the immediate response. Earlier recommendations to keep someone upright after rescue are not compatible with the acute need to restore central circulation. Oxygen and immediate intravenous fluid administration to prevent crush syndrome is appropriate but should not delay rescue.

Early work on this in the nonmedical and the non-English medical literature has kept it out of the medical eye. Despite this, suspension trauma and the recommendations on how to manage it have been prominent in occupational and search and rescue circles. Unfortunately, the lack of a modern medical perspective has led to a continuation of recommendations that are inappropriate. The concern for provoking "rescue death" has even led to purposeful delays in getting a person to ground and instituting treatment for shock.<sup>8</sup> Unless some day an appropriate animal model demonstrates the contrary, standard resuscitation measures should be used.

## References

1. Patscheider H. Pathologico-anatomical examination results in the case of death caused by hanging on the rope. Paper presented at: 2nd International Conference of Mountain Rescue Doctors [German to English translation by HSE Language Services Transl. No. 16372(1)]; November 18, 1972; Innsbruck, Austria.
2. Fodisch HJ. Morphological findings in the case of death after hanging on a rope for four hours. Paper presented at: 2nd International Conference of Mountain Rescue Doctors [German to English translation by HSE Language Services Transl. No. 16372(1)]; November 18, 1972; Innsbruck, Austria.
3. Toledo y Ugarte J-D. Death from orthostatic shock caused by hanging from the rope. Paper presented at: 2nd International Conference of Mountain Rescue Doctors [German to English translation by HSE Language Services Transl. No. 16372(1)]; November 18, 1972; Innsbruck, Austria.
4. Bariod J. Sensibilisation à la pathologie induite par l'utilisation du harnais. J. Grimbérieux and C. Ek, eds. In: *European Conference of Speleology*. Helecine, Belgium: International Union of Speleology; 1992:49-55.
5. Knutson S. American caving accidents, 1976–1979. *NSS News*. 1981;39(5, part 2):A70.
6. Putnam WO. American caving accidents, 1994–1995. *NSS News*. 1997;55(12, part 2):425–426.
7. Putnam WO. American caving accidents, 1999–2001. *NSS News*. 2003;61(6, part 2):19–20.
8. Putnam WO. American caving accidents, 2004–2005. *NSS News*. 2007;65(5, part 2):23–25.
9. Keeler R. American caving accidents, 2006. *NSS News*. 2008;66(3, part 2):16–17.
10. Knutson S. American caving accidents, 1987. *NSS News*. 1988;46(12, part 2):480–481.
11. Knutson S. American caving accidents, 1992. *NSS News*. 1993;51(12, part 2):366–367.
12. Williamson J, Plasman R. *Accidents in North American Mountaineering 1999*. Vol 52. Golden, CO: American Alpine Club; 1999.
13. Williamson J, Whalley E. *Accidents in North American Mountaineering 1977*. Vol 30. New York: American Alpine Club; 1977.
14. Ferris BG. *Accidents in North American Mountaineering 1972*. Vol 25. New York: American Alpine Club; 1972.
15. Bariod J, They B. Le point sur la pathologie induite par le harnais. *Spelunca*. 1994:39–42.
16. Orzech MA, Goodwin MD, Brinkley JW, Salerno MD, Seaworth J. *Test Program to Evaluate Human Response to Prolonged Motionless Suspension in Three Types of Fall Protection Harnesses*. Wright-Patterson Air Force Base, OH: Harry G. Armstrong Aerospace Medical Research Laboratory; 1987.
17. Damisch C, Schauer N. How safe are body harnesses? [German to English translation]. *Der Bergsteiger*. 1985: 6F–9F.
18. Madsen P, Svendsen LB, Jorgensen LG, et al. Tolerance to head-up tilt and suspension with elevated legs. *Aviat Space Environ Med*. 1998;69:781–784.
19. Nelson BA. Climbing harnesses: how long can you safely hang in your harness. *Off Belay*; 1979:10–12.
20. Amphoux M, Noel G, Archer P. La sécurité sur les chantiers du bâtiment et des travaux publics. *Ann Institut Tech Bat Travaux Pub*. 1982:78–110.
21. Noel G, Ardouin MG, Archer P, Amphoux M, Sevin A. Some aspects of fall protection equipment employed in construction and public works industries. In: Sulowski AC, ed. *Fundamentals of Fall Protection*. Toronto, Canada: International Society for Fall Protection; 1991: 1–32.
22. Roeggla M, Brunner M, Michalek A, et al. Cardiorespiratory response to free suspension simulating the situation between fall and rescue in a rock climbing accident. *Wilderness Environ Med*. 1996;7:109–114.
23. Polderman KH. Mechanisms of action, physiologic effects, and complications of hypothermia. *Crit Care Med*. 2009; 37(7[suppl]):S186–S202.
24. Turner NL, Wassell JT, Whisler R, Zwiener J. Suspension tolerance in a full-body safety harness, and a prototype harness accessory. *J Occup Environ Hyg*. 2008;5:227–231.
25. Seddon P. *Harness Suspension: Review and Evaluation of Existing Information*. Colgate, Norwich: Health and Safety Executive; 2002.
26. Stuhlinger W, Dittrich P, Flora G, Margreiter R. Circulatory and renal function changes in test subjects suspended from the upper half of the body. Paper presented at: 2nd International Conference of Mountain Rescue Doctors, November 18, 1972; Innsbruck, Austria.
27. Bernard W, Haselbach H, Scharfetter H, Aigner A, Michaeler R. Radiological, blood chemistry and lung function findings in the hanging test. Paper presented at: 2nd International Conference of Mountain Rescue Doctors; November 18, 1972; Innsbruck, Austria.
28. Shamsuzzaman ASM, Sugiyama Y, Kamiya A, Fu Q, Mano T. Head-up suspension in humans: effects on sympathetic vasomotor activity and cardiovascular responses. *J Appl Physiol*. 1998;84:1513–1519.
29. Hearon BF, Brinkley JW. Fall arrest and post-fall suspension: literature review and directions for further research. In: Sulowski AC, ed. *Fundamentals of Fall Protection*. Toronto, Canada: International Society for Fall Protection; 1991:123–137.
30. Mark AL. The Bezold-Jarisch reflex revisited: clinical implications of inhibitory reflexes originating in the heart. *J Am Coll Cardiol*. 1983;1:90–102.
31. Mitchell JH, Wildenthal K, Johnson RL. The effects of acid-base disturbances on cardiovascular and pulmonary function. *Kidney Int*. 1972;1:375–389.
32. D'Allesio JG, Weller RS, Rosenblum M. Activation of the Bezold-Jarisch reflex in the sitting position for shoulder arthroscopy using interscalene block. *Anesth Analg*. 1995; 80:1158–1162.

33. Oberg B, Thoren P. Increased activity in left ventricle receptors during hemorrhage or occlusion of caval veins in the cat—a possible cause of the vaso-vagal reaction. *Acta Physiol Scand.* 1972;85:164–173.
34. Flora G, Holzl HR. Fatal and non-fatal accidents involving falls into the rope. Paper presented at: 2nd International Conference of Mountain Rescue Doctors [German to English translation by HSE Language Services Transl. No. 16372(1)]; November 18, 1972; Innsbruck, Austria.
35. Bosch X, Poch E, Grau JM. Rhabdomyolysis and acute kidney injury. *N Engl J Med.* 2009;361:62–72.
36. Byard RW, Wick R, Gilbert JD. Conditions and circumstances predisposing to death from positional asphyxia in adults. *J Forensic Leg Med.* 2008;15:415–419.
37. Dawes R. Suspension trauma—a medical perspective. *Tech Rescue.* 2000:20.
38. Weems B, Bishop P. Will your safety harness kill you? *Occup Health Safety.* 2003;72:86–88.
39. Lee C, Porter KM. Suspension trauma. *Emerg Med J.* 2007;24:237–238.
40. Merchant D. Suspension trauma can be unrelated to harness use. *Emerg Med J, eLetters.* January 30, 2008;24:237-238.
41. Halliwell D. Suspension trauma—other places where the physiology may apply. *Emerg Med J, eLetters.* January 30, 2008;24:237-238.
42. Morgan P. Suspension trauma—case to prove. *Emerg Med J, eLetters.* November 20, 2007;24:237-238.
43. Limagne R. Le décrochage d'un equipier en difficulté sur corde. *Spelunca.* 1992:35–38.
44. Smith B, Padgett A. Vertical skills and rescue training. In: *On Rope.* Huntsville, AL: National Speleological Society; 1996:296-321.
45. Flora G, Margreiter R, Dittrich P, Stuhlinger W. Hanging tests—conclusions for the mountaineer. Paper presented at: 2nd International Conference of Mountain Rescue Doctors; November 18, 1972; Innsbruck, Austria.
46. Occupational Safety and Health Administration. Suspension trauma/orthostatic intolerance. *Safety Health Inf Bull* [serial online]. March 24, 2004. Accessed March 12, 2009.
47. Blaisdell FW. The pathophysiology of skeletal muscle ischemia and the reperfusion syndrome: a review. *Cardio-vasc Surg.* 2002;10:620–630.
48. Narayan SM, Drinan DD, Lackey RP, Edman CF. Acute volume overload elevates T-wave alternans magnitude. *J Appl Physiol.* 2006;102:1462–1468.
49. Parker KK, Lavelle JA, Taylor LK, Wang Z, Hansen DE. Stretch-induced ventricular arrhythmias during acute ischemia and reperfusion. *J Appl Physiol.* 2004; 97:377–383.
50. Michaelson M. Crush injury and crush syndrome. *World J Surg.* 1992;16:899–903.
51. Aoki N, Demsar J, Zupan B, et al. Predictive model for estimating risk of crush syndrome: a data mining approach. *J Trauma.* 2007;62:940–945.
52. Allister C. Cardiac arrest after crush injury. *Br Med J.* 1983;287:531–532.
53. Brown AA, Nicholls RJ. Crush syndrome: a report of 2 cases and a review of the literature. *Br J Surg.* 1977;64: 397–402.
54. Gunal AI, Celiker H, Dogukan A, et al. Early and vigorous fluid resuscitation prevents acute renal failure in the crush victims of catastrophic earthquakes. *J Am Soc Nephrol.* 2004;15:1862–1867.
55. Mullick S. The tourniquet in operations upon the extremities. *Surg Gynecol Obstet.* 1978;146:821–826.
56. Thomassen O, Skaiaa SC, Brattebo G, et al. Does the horizontal position increase risk of rescue death following suspension trauma? *Emerg Med J.* 2009;26: 896–898.
57. Werntz CL. Occupational Medicine Forum. *J Occup Environ Med.* 2008;50:858–859.
58. Adishes A, Robinson L, Codling A, et al. *Evidence-Based Review of the Current Guidance on First Aid Measures for Suspension Trauma.* Norwich, UK: Health and Safety Executive; 2009.
59. Sever MS, Vanholder R, Lameire N. Management of crush-related injuries after disasters. *N Engl J Med.* 2006; 354:1052–1063.
60. Josephson M, Wellens HJJ. Implantable defibrillators and sudden cardiac death. *Circulation.* 2004;109: 2685–2691.
61. Marbach G, Tourte B. Emergencies and rescue. In: Alspaugh M, trans. *Alpine Caving Techniques.* 3rd ed. Allschwil, Switzerland: SpeleoProjects; 2002:263–305.