# Monitoring Heat Injuries in a Hazmat Environment

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A tool that identifies heat injuries early can avoid the progression of symptoms from heat stress to heat exhaustion and heat stroke.

Heat injuries are a major problem worldwide. In a study chronicling heat deaths in the U.S. from 1979 to 1999, a total of 8,015 deaths were associated with excessive heat exposure.[1] Weather conditions caused 3,829 (48%) deaths, and manmade conditions (kitchens, vehicles, boiler rooms, etc) caused 377 (5%) deaths, particularly for those wearing protective clothing.[1]

Military members who wear combat gear are especially vulnerable to heat injuries, but none more so than members who wear personal protective equipment (PPE). In this review, PPE is defined as self-contained breathing apparatus protective equipment (SCBA) levels B or C. The challenge of PPE is the inability of the individual to dispel heat through radiation, convection, and evaporation. The only close approximation of the PPE environment is combat and football protective equipment. In 2011, CDC reported that football players in uniforms, which resemble PPE for the purpose of this discussion, experienced heat injury at a rate 10 times higher than the average rate for other sports.[2] These heat injuries in football players occurred most often during August.[2] The injuries could be due to the application of protective clothing and the lack of the participants' acclimatization. Protective clothing impedes the wearer's ability to balance heat production with heat dissipation.

In 2010, Armstrong and colleagues suggested that the weight of a football uniform increases heat production.[3] And the insulation provided by a football uniform reduces heat dissipation to the surrounding air, decreasing heat loss.[3] Additionally, this same study indicated that the more protective gear the subject used, the greater the heat stress.[3] The most challenging environment for heat injury is PPE due to the inability to facilitate any heat loss. In 2011, Caldwell and colleagues observed that wearing torso armor increased body temperature 10.8% faster than that of the control group, and those wearing full armor increased body temperature 38% faster than that of the control group.[4] And it was proposed that 60% of this heat effect was from wearing the combat helmet.[4]

The inability to dissipate heat, particularly in protective gear, results in degradation of the effectiveness of the individual and, if left unchecked, may lead to death. Methods exist for health care providers to assess, intervene, and treat populations with heat injuries. These methods include but are not limited to vital signs (blood pressure [BP], body temperature, respiration rate), history of previous heat injury, medications (over-the-counter and prescription), and mental status.

## HEAT INJURIES

Heat injuries are generally divided into 3 categories defined by their severity: heat stress, heat exhaustion, and heat stroke. Heat injuries are due to the individual's inability to dissipate heat. As the severity of the heat exposure continues, the individual will experience heat stress, and if decompensation continues, the individual will progress to heat exhaustion and finally heat stroke.

If the individual's physiology is limited or if compensatory mechanisms are compromised, heat stress may occur. Heat compensation can be retarded by any number of the following (including but not limited to): humidity, previous heat injury, lack of sleep, medications, sedentary lifestyle, obesity, caffeinated energy drinks, and dehydration.

In the early phases of heat stress, an individual's vital signs will increase to compensate for the increase in body heat. Heat exchange is dependent on gradients

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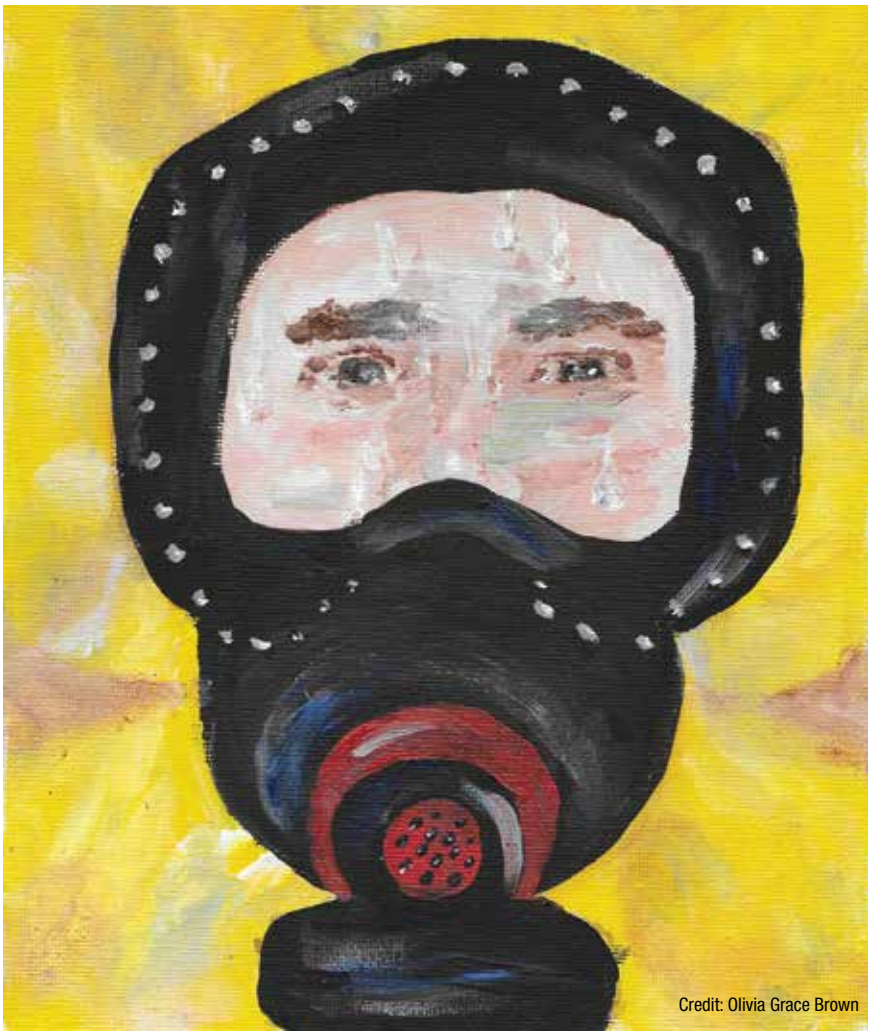
of temperature and humidity, and as temperature and humidity increase, the ability to transfer heat decreases and becomes less efficient. Failure to accommodate for the increased heat generated and transferred will inevitably result in heat injury.

Working in a hazmat environment in PPE is the worst possible heat transfer scenario due to the inability to use evaporation, the primary means by which heat is released from the body. In this scenario, heat injuries can become dangerous and even fatal if monitoring of vital signs and uncompensated heat production is allowed to continue. As the heat insult progresses from heat stress to heat exhaustion and heat stroke, the core temperature, heart rate, and BP continue to increase. Also, during the progression of heat injury, mental status changes often begin to occur. In 2012, Morley and colleagues found that firefighters wearing protective clothing demonstrated a neurocognitive decline after 50 minutes of treadmill exercise, but these performance declines were not noted until 1 hour or more following the exercise.[5]

Mental status change is a key diagnostic factor that indicates the progression of the patient from heat stress to heat exhaustion and from heat exhaustion to heat stroke. As the hyperthermia progresses, vital signs increase, and the patient's mental status will begin to deteriorate. If the hyperthermia advances from heat exhaustion to heat stroke, hospitalization is required to reverse the condition. If homeostasis is not restored, the patient may die.

Mental status changes are usually described as fatigue, lethargy, disorientation, headache, seizure and coma. Indeed, mental status changes may be one of the most important factors that can assist the clinician in the identification, mitigation, and treatment of heat injury before it reaches a critical stage. Clinical familiarity with and diagnosis of delirium resulting from heat injury could prove beneficial in protecting an individual exposed to severe heat environments.


Credit: Olivia Grace Brown

In 2011, Becker and Stewart suggested that in the absence of hyperthermia, the presence of central nervous system (CNS) symptoms should prompt the clinician to pursue another diagnosis.[6] However, a core temperature of 104°F with associated CNS changes and anhydrosis should be defined as heat stroke and is a medical emergency.[6]

Death rates from excessive heat are documented as high as 31%.[7] Signs of CNS dysfunction such as irritability, ataxia, headache, nausea, vomiting, anhydrosis, confusion, and decreased cognitive function are essential to the diagnosis of heat stroke. Classic heat stroke will present as a triad of hyperpyrexia, anhydrosis, and mental status changes.[8] However, making the diagnosis of heat stroke based on anhydrosis could be dangerous, because in exertional heat stroke, many patients continue to sweat. Overlooking the diagnosis of heat stroke based on anhydrosis could lead to a delay in treatment and severe complications.[8] These complications may include hyperkalemia, hyperphosphatemia, hypocalcemia, and myoglobinuria.

Once heat stroke has occurred, coagulopathies may manifest as epistaxis, and endothelial damage may present as peripheral or pulmonary edema. Additionally, a core temperature of above 104°F may trigger a cascade of events that may include systemic inflammatory response resulting in increased cell wall

**Table. Micro-Mental Status Test**

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| **Appearance** | Gestalt based on prescreening assessment |
| **Task** | Behavior—Gestalt based on prescreening assessment<br>Speech—Self-identifying questions<br>Mood—Gestalt based on prescreening assessment<br>Affect—Gestalt based on prescreening assessment |
| **Thought** | Process—Finding personal record<br>Content—"Where were you working?" "What was your specific job?" |
| **Cognition** | Finding personal record<br>Insight—"Anything troubling about the job?" "Do we need to change our approach to the emergency?" "Tell me the content of your day—with 3 ups and 3 downs"<br>Judgment—"Would you tell me the truth if we needed to change our approach to the emergency?" |

permeability and release of endotoxins. These events can lead to tissue hypoxia, metabolic acidosis, and organ failure. Sequalae from heat stroke can result in multisystem failure.

A 1998 study of Chicago heat wave victims reported that the degree of functional disability predicted survival at 1 year.[9] Although hospital mortality was 21%, severe functional impairment at discharge was 33%, with an additional 28% mortality at 1 year.[9] And the 1-year mortality from heat stroke is similar to that of cerebral vascular accidents.[10] Within 24 hours, heat stroke victims often will display evidence of muscle, kidney, and cardiac dysfunction. Delay in intervention raises the risk of fatalities associated with hyperthermia.[11,12] Tissue destruction due to uncompensated heat may lead to rhabdomyolysis and subsequent myoglobinuria and renal injury. Damaged hepatocytes may lead to coagulopathies and hepatitis. Injured heart muscle may lead to arrhythmias and cardiac arrest.

The CNS symptoms may be difficult to ascertain in an intense working environment. The CNS system dysfunction is indicative of progression from heat injury to heat stroke and thus a medical emergency. It is imperative that the clinician be able to assess the individual quickly and accurately.

## DELIRIUM

Along with physical problems associated with it, heat injury can also lead to relatively abrupt mental status changes. In 2005, Glazer reported that even with minimally elevated core temperatures, CNS system changes can present with altered mental status, convulsions, and coma.[13] This qualifies as a medical emergency known as delirium. Patients with delirium may present with a history of abrupt and fluctuating levels of consciousness. This fluctuation in symptoms that resemble sepsis could confuse medical providers.[13] Thus, it is imperative that there be continuity of care of the patient with the ability to compare states of consciousness longitudinally over time.

In 1984, Pérez reported that nurses, perhaps because of their familiarity with and proximity to the patient, recorded delirium in 93% of patients, whereas psychiatric consultants recorded delirium only 34% of the time.[14] Delirium manifests with several neurologic signs and symptoms; these include but are not limited to tremor, myoclonus, difficulty reading and writing, and visuoconstructive deficits, such as copying designs and problem solving.[15] No matter the method to discover the delirium, the definitive treatment is to identify and treat the underlying medical condition.[15] The CNS system dysfunction consistent with delirium such as irritability, ataxia, and confusion are essential to the diagnosis of heat stroke.[13] Coma and seizures may occur, and retarded recovery of functional ability is an indication of a poor prognosis.[9]

## OBJECTIVE

The authors propose that in addition to vital signs, an assessment of a patient's mental status through the use of a mental status exam could be a tool that identifies the problem early and avoids the progression of symptoms from heat stress to heat exhaustion and heat stroke. Early intervention in the progression of symptoms of hyperthermia can save lives, decrease suffering, and maintain a more robust mission-ready posture for the individual and the unit.

## STUDY

During the fall of 2014, the Chemical, Biological, Radiological, Nuclear, and Explosive (CBRNE) unit of the Utah Air and Army National Guard participated in exercises using 2,159 patient encounters that were in PPE (full hazmat and SCBA) also known as level C protective clothing. Temperatures ranged from a minimum of 29°F to a high of 56°F. A mock disaster was practiced for 5 days, and of those 2,159 iterations, 43 were disqualified (2%) for any reason. Two individuals presented

with altered mental status and disrupted vital signs and were disqualified for heat injuries with cognitive symptoms (0.00092%). These members were excused from duty, monitored in the medical work/rest tent until mental status and vital signs returned to baseline.

The tool used in the study was the Micro-Mental Test. This is a mental status exam that is more than a simple gestalt of how the patient is performing cognitively but less than a full Mini-Mental State Examination (MMSE). This abbreviated mental status exam provides a field expedient measurement of the individual's ability to function cognitively. It is important to realize that this exam is most effective when repeated over time to assess the patients' mental status longitudinally. It would be cavalier to propose that an abbreviated mental status exam would be sufficient to diagnose heat stroke, but a mental status exam—however brief—along with symptoms of hyperpyrexia, abnormal vital signs, and anhydrosis can be a useful tool to make the diagnostic transition from heat exhaustion to heat stroke.

### Micro-Mental Status Exam

The traditional mental status measures are appearance, behavior, speech, mood, affect, thought process, thought content, cognition, insight, and judgment. Rapidly assessing mental status is crucial for the assessment of heat injuries, because increased vital signs coupled with neurologic changes indicate a medical emergency. The MMSE is painstaking and a somewhat cumbersome tool to use in the field. Therefore, the authors suggest a micro-mental status exam (Table). This abbreviated mental status exam is performed before the individual is placed in the PPE and enters the working environment.

The individual is then assessed after every rotation exiting the PPE and allowed to rest under supervision. Assessing the individual with vital signs and mental status longitudinally allows the provider to rapidly assess and intervene if the patient begins to exhibit mental status changes along with increased vital signs. The patient is assessed for ataxia, confusion, irritability, and lack of coordination. Patients are asked to find from a file drawer their individual prescreen checklist. This test assesses fine motor skills and cognition. Following this, self-identifying personal information from a precheck sheet is verified, and finally, simple questions regarding orientation to person, place, date, and time are posed.

### Assessing Executive Function

Examples of measures of thought processes include assessing executive function by having participants find their paperwork, identifying their platoon leader, and correctly responding to questions, such as, "Where exactly were you working in the emergency area and what exactly were you doing?" This assesses executive function and thought process. Thought content could be assessed with inquiries such as, "Anything troubling about your work?" or "Would you tell me honestly if there were anything troubling or unsafe about the work you have performed?" Cognition could be assessed by questions regarding chain of command (both officer and enlisted), 3 suggestions to improve, 3 suggestions to maintain, and knowledge of the rotation schedule for the rest of the day.

The abbreviated mental status exam should in no way replace the robust and accurate mental status exam. However, in a rapidly changing, austere, or asymmetrical environment, a simple gestalt of the patient is ineffective, and the full mental status evaluation may be too time consuming. The authors propose the Micro-Mental Exam as an alternative. It is imperative that the exam be compared with the baseline assessment of the individual during the prescreening of vital signs before the individual enters the exercise.

This Micro-Mental Exam provides a quick, easy, nonintrusive, and stress-free assessment of the patient. The clarity of cognition and ability to perform simple mental tasks could serve to reassure the provider that the patient has not progressed into the dangerous area of delirium secondary to heat exposure.

Use of this simple tool during the CBRNE exercise resulted in the disqualification of 2 individuals for probable heat injury; additionally, it gave the providers a rapid assessment tool to quickly identify and treat individuals with progressive heat stress to heat stroke.

## DISCUSSION

Compared with studies of heat injuries in military and football equipment, the expected heat injury in PPE gear is very low.[2-4] The low number of disqualifications during the CBRNE exercise could be due to the extensive measures in place to assist individuals under heat stress. These measures include strict adherence to the work/rest cycles mandated by the DoD, competent leadership in evaluating and treating individuals participating in the exercise, and paying close attention not only to the vital signs, but also participants' mental status.

A study in 2002 suggested that spending time in an air-conditioned area is the strongest factor in preventing heat-related deaths.[16] The

study also recommended prevention measures if heat exposure cannot be avoided: working in the cooler part of the day, plenty of water or nonalcoholic drinks, cool showers, lightweight light-colored clothing, and avoiding direct sunshine.[16]

A study in 2013 suggested that heat injuries are a significant threat to the effectiveness of military operations in general and to the youngest (the most inexperienced soldiers) specifically.[17] The study further suggested that it is imperative that leaders be aware of adequate hydration on the one hand and excessive water intake on the other and enforce effective countermeasures against all types of heat injuries.[17]

## HYPONATREMIA

Hyponatremia is a possible complication of heat exposure and can be divided into categories according to volume: hypovolemia, euvolemia, and hypervolemia.[18] Hyponatremia is associated with excessive water consumption and excessive sodium losses via sweat during prolonged physical exertion. Symptoms of hyponatremia are related to the severity of sodium deficit and the rate of sodium decline.[18] These symptoms include but are not limited to polydipsia, muscle cramps, headache, altered mental status, coma, and status epilepticus.

Hypovolemic hyponatremia usually will have signs of volume depletion, and sodium levels < 20 mEq/L. Treatment typically consists of volume replenishment with isotonic saline (0.9%), treatment of the underlying condition, and correction of the factors causing hypovolemia.

Euvolemic hyponatremia is typically due to the syndrome of inappropriate antidiuretic hormone (SIADH) and spot urinary sodium is > 20 mEq/L. Correction consists of fluid restriction and correction of the underlying cause.[18]

Hypervolemic hyponatremia occurs when the kidneys are overwhelmed and cannot excrete water effectively. It is commonly caused by heart failure, cirrhosis, or renal injury. Treatment consists of correction of the underlying cause, sodium and fluid restriction, and diuretic therapy.[18] In severe cases of hyponatremia, sodium levels usually have decreased rapidly—typically in less than 24 hours.

Hyponatremia is defined as plasma sodium levels < 135 mEq/L, and severe symptoms often occur when the sodium level reaches 120 mEq/L. Treatment must be initiated quickly to avoid cerebral edema, respiratory failure, brain stem herniation, and death. Correction includes hypertonic 3% saline infusion at a rate of 0.5 to 2 mL/kg per hour until symptoms resolve. Two separate studies in 2014 and 2013 suggested that the rate of sodium correction should be 6 to 12 mEq/L in the first 24 hours and 18 mEq/L or less in 48 hours.[19,20]

In 2009, Sterns and colleagues suggested that for the treatment of hyponatremia the therapeutic goals for serum sodium concentrations should be 6 to 8 mmol/L in 24 hours, 12 to 14 mmol/L in 48 hours, and 14 to 16 mmol/L in 72 hours.[21] To exceed these parameters in the correction of hyponatremia risks overcorrection and iatrogenic brain damage.[21]

Care must be taken not to overcorrect sodium levels. In 2013, Sood and colleagues reported that in severe hyponatremia, a combination of 3% saline and 1 to 2 µg of desmopressin every 6 to 8 hours achieved a predictable correction of 3 to 7 mEq/L per hour with no overcorrection.[22]

In the spring of 1998, U.S. Army guidelines were revised not only to protect service members from heat injury, but also from hyponatremia caused by excessive sodium loss due to exertion combined with excessive water consumption. There were fewer hospitalizations of soldiers for hyponatremia due to excessive water consumption after the guidelines were implemented.[23] Potential hyponatremia in PPE is even greater due to the strenuous environment. The potential injury due to heat injury on the one hand and hyponatremia on the other demands tailored scrutiny by experienced providers and commanders who can make appropriate changes to the work-rest cycle as needed.

Quick recognition and treatment of exercise-induced hyponatremia is essential to avoid altered mental status, seizures, coma, and death. Current guidelines for the correction of exercise-induced hyponatremia suggest rapid correction of hyponatremia with up to three 100 mL boluses of 3% NaCl in 10-minute intervals. A 2012 case study by Elsaesser and colleagues reported that a severely dehydrated marathon runner with exercise-induced hyponatremic encephalopathy achieved a resolution of symptoms with rapid correction with 100 mL boluses of 3% NaCl spaced in 10-minute intervals. An additional volume of 650 mL of 3% NaCl given over 2 hours for a total volume of 950 mL was needed to resolve the exercise-induced hyponatremia.[24] It seems that a 4- to 6-mmol/L increase in serum (Na+) is adequate to reverse most serious clinical manifestations of acute hyponatremia.[21]

When hyponatremia is corrected too rapidly, the brain's ability to absorb the metabolites is overwhelmed, resulting in osmotic demyelination.[21] Demyelination was produced in animal models by the rapid induction of hypernatremia and can occur in

patients who are overcorrected to a hypernatremic state.[20] When individuals with chronic hyponatremia are corrected to normal sodium levels, an initial improvement may occur followed by new and often progressive neurologic deficits.[20]

In 2012, Elsaesser and colleagues suggested that concern regarding overcorrection of hyponatremia might be exaggerated in the setting of exercise-induced hyponatremia. Indeed, the only cases of death associated with exercise-induced hyponatremia have been in the setting of no treatment or slow correction of hyponatremia with normal saline.[24]

## CONCLUSIONS

The issue of heat injury in athletic and military environments plagues participants and leaders alike. This article has sought to shed light on mechanisms that are helpful in mitigating heat injury. Football equipment and military protective gear that diminishes that ability to dissipate heat through the retardation of evaporation, convection, and radiation is a key factor in the development of heat injury.

Personal protective equipment is the most hazardous environment for the development of heat injury. This protective gear along with increased environmental humidity, elevated temperature, and increased workload create a dangerous environment for the individuals involved. Careful monitoring of vital signs is an important factor in avoiding heat injuries.

This article proposes that vital signs along with strict monitoring of mental status through (1) orientation; (2) simple task completion; (3) thought processes; and (4) cognitive ability over time combine to be a powerful deterrent to heat injury in an austere and dangerous working environment. It would be cavalier to propose that all heat injuries in any environment could be avoided by following these guidelines, and more tools to avoid heat injury will be developed. But medical providers trained not only to use vital signs, but also monitor and respond to mental status changes in the patient can mitigate heat injuries more effectively. Finally, careful attention should be placed on correcting hypo- and hypernatremia when rehydrating individuals in this challenging environment. ●

### *Acknowledgements*

*The authors wish to thank the following for their contribution to this manuscript: Sarah M. Paulsen, REB Smith, and the entire CERF-P leadership of the Utah National Guard.*

### *Author disclosures*

*The authors report no actual or potential conflicts of interest with regard to this article.*

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