# PREVENTION AND MANAGEMENT OF HEAT-RELATED ILLNESS

Federal Bureau of Prisons Clinical Guidance

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## 1. PURPOSE AND OVERVIEW

The purpose of the BOP Clinical Guidance on the Prevention and Management of Heat-Related Illness is to educate staff about:

- **RISK FACTORS** for heat-related illness (**HRI**)
- Signs and symptoms of HRI
- **PREVENTIVE STRATEGIES** to mitigate and reduce heat stress
- APPROPRIATE INTERVENTIONS for the prompt treatment of HRI

Identifying inmates with risk factors, together with implementing environmental and systemwide controls, can reduce or prevent HRI in BOP facilities. Prompt recognition of the signs and symptoms of HRI, followed by appropriate and timely intervention, may reduce the significant morbidity and mortality associated with the more severe forms of this condition. The mortality rate for heat stroke ranges from 20–60%, increasing with the severity and duration of core body temperature elevation, and the extent of organ dysfunction.

## 2. PATHOPHYSIOLOGY

**THERMOREGULATION:** The human body is able to regulate core body temperature within a fairly narrow range of  $98.6^{\circ}F + 1.8^{\circ}F$ . The hypothalamus serves as the body's control center for thermoregulation and employs a variety of physiologic mechanisms to dissipate heat.

- Sweating is controlled by the parasympathetic nervous system and is the primary and most effective method of heat loss from **EVAPORATION**.
- The sympathetic nervous system increases blood flow to the skin through vasodilation, which dissipates heat by **RADIATION**.
- **CONDUCTION** of heat occurs when a colder solid or liquid (e.g., ice pack or cold water) comes into direct contact with the skin.
- **CONVECTION** of heat is accomplished with air movement across the skin surfaces (e.g., wind from a breeze or fan).

**ACCLIMATIZATION:** In response to higher air temperatures and training, the body is able to adapt and improve its response to heat stress by expanding blood volume and blood flow to the skin, sweating at lower temperatures and in greater amounts with lower sodium content. These improved responses usually develop over a 1 to 2-week period of exposure.

**HEAT STRESS** refers to a source of heat that could potentially raise the core body temperature. When the body experiences conditions of increased temperature, the hypothalamus may reset the body's thermostat to a higher core temperature, in addition to activating the **THERMOREGULATORY** mechanisms described above.

- This may serve to improve physiologic function and is considered to be **COMPENSATED** if the body temperature remains stable at a new set point.
- However, the heat stress is classified as **UNCOMPENSATED** when the body is unable to maintain a steady state, and there is a progressive rise in core temperature.

**UNCOMPENSATED HYPERTHERMIA** may have severe and life-threatening adverse effects. This is especially true for severe hyperthermia with body temperatures exceeding 104°F. In addition to an increase in oxygen consumption and metabolic rate, cytokines are produced and endotoxins are released through damaged cell membranes, causing a systemic inflammatory response. Blood flow is directed preferentially to skin and muscle, which may result in organ ischemia, especially of the liver, kidney, and nervous system. Enzyme function becomes impaired at body temperatures exceeding 108°F.

## 3. RISK FACTORS FOR HRI

Heat dissipation and body temperature homeostasis may be impaired by a variety of factors, any one of which may increase the risk of developing HRI. These factors may be categorized as **EXTERNAL** or **INTERNAL**, based on their relationship to the human body.

### **EXTERNAL (ENVIRONMENTAL) RISK FACTORS**

- HIGHER AMBIENT AIR TEMPERATURE AND HUMIDITY is the primary underlying risk for HRI. As air temperature increases, body heat dissipation by means of convection, conduction, and radiation to the surrounding environment becomes less effective. Once the air temperature reaches or exceeds core body temperature, these mechanisms of heat loss become completely ineffective. Similarly, as humidity increases, evaporation becomes less effective in dissipating heat, becoming totally ineffective at humidity levels greater than 75%.
- **THE HEAT INDEX** is another factor. Higher humidity levels make it feel hotter than the actual measured temperature. Air temperature (measured in the shade) and humidity levels are used to calculate the heat index temperature, which has been correlated with the risk for HRI.
  - ► The National Weather Service (NWS) Heat Index, which identifies four color-coded levels of increased risk for HRI (Caution, Extreme Caution, Danger, and Extreme Danger), is discussed at: <u>http://www.nws.noaa.gov/om/heat/heat\_index.shtml</u>.
    - → See <u>Appendix 1</u> to see the NWS Heat Index Chart available at the time of this publication.
  - The NWS online calculator, which allows users to calculate the heat index based on local air temperature and relative humidity, is available at: <u>http://www.wpc.ncep.noaa.gov/html/heatindex.shtml</u>.
  - Although the Wet Bulb Globe Temperature is preferred by some for determining heat risk, the equipment and expertise for this approach are not readily available at most BOP facilities.
- **EXPOSURE TO DIRECT SUNLIGHT** also makes it feel hotter than the actual air temperature. Heat index values are based on temperatures in the shade and may increase by as much as 15°F in the direct sunlight.
- URBAN ENVIRONMENTS have been shown to increase temperatures by more than 20°F.
- NON-AIR-CONDITIONED INDOOR AREAS AND HIGHER FLOORS WITHIN BUILDINGS may magnify or trap heat.
- Excessive, HEAVY, OR DARK CLOTHING can be a risk factor for HRI.

### INTERNAL (INMATE) FACTORS

A variety of human factors increase the risk for HRI. A *Medical Duty Status of Heat Restriction* is assigned in BEMR and Sentry to all inmates with medical risk factors for heat illness as described below.

- AGE (younger than 15 years; age 65 years and older)
  - ➔ A number of changes occur with aging that impair the ability to dissipate heat, including decreased skin surface area, number of skin capillaries, and number and activity of sweat glands—in addition to vascular volume depletion and chronic vasoconstriction.
- LACK OF ADAPTATION (physical deconditioning, lack of acclimation to hotter temperatures)
  - + It may take 1 to 2 weeks for the body to adapt to a hotter climate.
- DURATION AND INTENSITY OF EXERTION
- DEHYDRATION
  - Core body temperature increases about 0.4°F for every one percent of body weight lost due to dehydration.
- MEDICAL CONDITIONS
  - Chronic diseases of the heart, lungs, liver, or kidneys; chronic degenerative neuromuscular diseases; active infections; obesity, diabetes, or hypertension; physical disabilities and impaired mobility; cognitive impairment or psychological impairment due to mental illness; pregnancy; prior history of heat stroke; large areas of scarring; and sweat gland dysfunction.
- MEDICATIONS AND OTHER SUBSTANCES
  - Medications may impair heat dissipation by decreasing cardiac output, inhibiting sweat, or causing volume depletion. The following classes of medications may increase the risk for HRI: alpha agonists, anticholinergics, antihistamines, benzodiazepines, beta blockers, calcium channel blockers, diuretics, laxatives, lithium, neuroleptics, phenothiazines, thyroid agonists, and tricyclic antidepressants.
  - Other substances are also well known for their increased risk of HRI, including alcohol, cocaine, ephedra, and stimulants.

## 4. SYMPTOMS AND SIGNS

Symptoms of HRI range in severity from mild and self-limiting, to catastrophic organ system failure and death. Experts, themselves, disagree on the classification and definitions of the various types of HRI, primarily due to the lack of data and understanding of these conditions.

The International Classification of Diseases identifies four basic types of HRI: HEAT CRAMPS, HEAT SYNCOPE, HEAT EXHAUSTION, and HEAT STROKE. The U.S. military recognizes a fifth category, HEAT INJURY, and some authors include HEAT RASH in a discussion of HRI.

• HEAT CRAMPS, RASH, and SYNCOPE: Although they occur more commonly in higher temperatures, heat cramps, rash, and syncope usually are not a direct result of hyperthermia, but may result from a lack of conditioning or acclimatization, from extreme exertion, or from the body's thermoregulatory mechanisms such as sweating, dehydration, or electrolyte abnormalities. These are considered milder forms of HRI and are not usually associated with hyperthermia.

• HEAT EXHAUSTION, INJURY, and STROKE: In contrast, heat exhaustion, injury, and stroke are directly related to elevated core body temperature, which is usually above 101°F in heat exhaustion and above 104°F in heat injury and heat stroke.

### The types of HRI are discussed below:

**HEAT RASH**, a.k.a. miliaria rubra or "prickly heat," is caused by sweating that causes blockage or inflammation of the eccrine sweat ducts. In the context of a recent relocation to a hotter climate, or heat exposure and sweating, the diagnosis is usually made based on the appearance of erythematous papules, pustules, or vesicles on the skin that are not located at the base of hair follicles. Occlusive clothing, bandages, or medication patches may also cause miliaria. It usually resolves spontaneously with acclimatization or with removal of the causative factors.

**HEAT CRAMPS** are muscle cramps that occur in association with new exercise regimens, high intensity work, or exertion to the limits of endurance. They occur more commonly in hotter weather, but can occur in colder temperatures, as well. Risk factors for heat cramps include lack of acclimatization and conditioning, excessive sweating, dehydration, and inadequate rest, hydration, or sodium intake prior to the activity.

**HEAT SYNCOPE**, also called exercise-associated collapse, is an inability to remain upright due to lightheadedness or a complete loss of consciousness that usually occurs in higher air temperatures following exercise, after prolonged standing, or quickly standing after a prolonged period of sitting. The primary cause is venous pooling or vasodilation. Syncope has a broad differential diagnosis that requires consideration, especially for cardiac etiologies.

**HEAT EXHAUSTION** occurs when cardiac output cannot meet the body's physiologic demands in the context of an elevated core body temperature between 101°F and 104°F. Extreme fatigue and weakness make further exertion difficult or impossible. Other characteristic symptoms and signs include tachycardia, hypotension, headache, muscle and abdominal cramps, nausea, vomiting and diarrhea, excessive sweating and pallor, ataxia and syncope, dehydration, and electrolyte abnormalities. If any neurologic symptoms occur, they are mild and resolve rapidly with treatment.

**HEAT INJURY** is a category of HRI characterized by a core body temperature usually  $> 104^{\circ}$ F and organ damage, but no neurologic damage or dysfunction. The organs primarily affected are the kidney, liver, and muscle, which may present as acute kidney or liver injury, or rhabdomyolysis, respectively.

**HEAT STROKE** is defined by a core body temperature > 104°F and neurologic dysfunction, usually with mental status changes, in the context of a significant heat stress. Other neurologic manifestations can include seizures, as well as abnormalities of speech, behavior, and level of consciousness. If coherent, a person with heat stroke may have symptoms and signs similar to those of heat exhaustion and heat injury. Much like with heat injury, there is usually dysfunction of other organ systems, including the lungs. Although prior descriptions of heat stroke have indicated an absence of sweating as characteristic of this condition, most patients are still sweating profusely at the time of engagement by health care workers.

• Heat stroke is classified as NON-EXERTIONAL and EXERTIONAL. Classic, non-exertional heat stroke occurs in older, sedentary individuals with chronic medical problems, while exertional heat stroke typically occurs in younger, healthy persons participating in strenuous activities.

• Included in the differential diagnosis for hyperthermia with mental status changes is NEUROLEPTIC MALIGNANT SYNDROME (NMS), which is an idiosyncratic reaction to antipsychotic medications. Although NMS most commonly develops within the first two weeks after starting a neuroleptic medication, it can occur in a person who has been taking a medication for years. Like heat stroke, NMS is a life-threatening condition that requires prompt intervention.

## 5. EVALUATION

# An important FIRST STEP for clinicians is to consider HRI in the differential diagnosis of a patient's presenting symptoms, especially during periods of high heat and humidity or strenuous exertion.

Inmates suspected of having HRI need prompt evaluation and intervention to identify the severity of illness and to reduce morbidity and mortality from severe hyperthermia. Outcomes are determined primarily by the degree and duration of temperature elevation and by the number and severity of organ systems affected.

- Initial evaluation and management starts with basic life support while relocating the inmate to a cooler environment as quickly as possible.
- VITAL SIGNS—including heart rate, respiratory rate, blood pressure, temperature, and weight may be obtained at the same time as the patient history.
  - When severe heat illness is suspected, a rectal temperature is recommended rather than oral, axillary, or tympanic temperatures because it more accurately reflects core body temperature.
  - Orthostatic blood pressure and pulse may provide evidence for dehydration in patients who can sit or stand safely.
  - If a previous weight is available, obtaining a current weight, if possible, may provide indirect evidence of dehydration.
- **PHYSICAL EXAMINATION** needs to be fairly comprehensive—including assessment of the cardiovascular, pulmonary, neurologic, muscular, gastrointestinal systems, and the skin—looking for evidence of organ system dysfunction, in particular.
- **DIAGNOSTIC STUDIES** are determined in part by the severity of symptoms and test availability in the particular setting.
  - A **blood glucose** via finger stick and **pulse oximetry** are readily available in the outpatient setting.
  - Basic labs may include 1) serum electrolytes, BUN, and creatinine (basic metabolic panel), looking for electrolyte abnormalities and renal dysfunction; 2) CBC, platelets, and INR, looking for coagulopathy and leukocytosis; and 3) liver panel, looking for hepatic enzyme elevations (enzyme elevation is common in heat stroke, may be severe, and may be delayed by 24 to 48 hours).
  - More complex testing is likely to be accomplished in an emergency department or hospital setting and may include 1) serum creatine and urine myoglobin, looking for rhabdomyolysis; 2) blood gasses, looking for metabolic acidosis and respiratory alkalosis; and 3) toxicology, if drug use is suspected.

- AN ECG, looking for signs of arrhythmia or ischemia, is appropriate for evaluation of heat syncope and for the more severe types of heat illness.
- A CHEST X-RAY, looking primarily for pulmonary edema, is indicated for cases of heat stroke and as clinically indicated for other types of heat illness.

### 6. TREATMENT

- Initial evaluation and management starts with basic life support while relocating the inmate to a cooler environment as quickly as possible. See <u>Section 5</u> above.
- RAPID COOLING and REHYDRATION are the cornerstones of HRI management in the outpatient or pre-hospital setting. See discussions below in this section.
- Treatment of organ system dysfunction is usually accomplished in the hospital setting (e.g., treatment of acute kidney or liver injury, arrhythmias or other cardiac dysfunction, neurologic deficits, pulmonary edema, and rhabdomyloysis).
- Although medications may be needed for the treatment of complications, antipyretic medication therapy has no role in the management of hyperthermia. (Antipyretic medications include fever-reducing drugs such as acetaminophen, aspirin, and non-steroidal anti-inflammatory drugs.)
- **Emergent transport for hospital management** is recommended for all inmates suspected of having heat stroke or heat injury, those with unstable vital signs, and those with heat exhaustion whose symptoms do not rapidly improve with treatment over 20 to 30 minutes.

### RAPID COOLING

- **Relocation to a cooler environment** can be accomplished by moving the inmate out of direct sunlight into a shaded area, or preferably into an air-conditioned room.
- **Rest** will reduce the amount of internal heat generated by exertion.
- **Removal of clothing** will prevent trapping of body heat and will expose the skin for other cooling measures.
- Application of cooler water or ice to exposed skin surfaces with frequent reapplication dissipates heat by conduction.
  - The axillae, neck, and groin are areas with a high volume of blood flow where wet towels or ice bags may be used to increase the effectiveness of heat dissipation. Some studies suggest that also applying these measures to the cheeks, palms, and soles leads to more rapid cooling.
  - ► In the outdoor setting, spraying cooler water from a hose can serve this purpose as well.
  - Immersion of the body in an ice water bath can achieve rapid decreases in core body temperature, but is usually reserved for treatment of severe HRI in an emergency department setting and may increase mortality in older patients.
- **Use of fans** to create air circulation across wet skin, especially in lower humidity environments like air-conditioned rooms, enhances evaporative cooling.
- **CAUTION: Rapid cooling may induce shivering,** which can generate more heat and is counterproductive to the cooling efforts. Some medical literature describes the use of a benzodiazepine (e.g., lorazepam) to temporarily stop the shivering.
- Aggressive cooling efforts may be stopped once the rectal temperature drops to about 101°F.

### REHYDRATION

- **Oral fluids**, specifically commercially available sports drinks when available, are recommended for rehydrating persons with mild HRI, especially if the HRI is related to exertion. Ice chips or chilled oral fluids, preferably not caffeinated beverages, may provide an additional source of cooling. Ice chips are a relatively simple, safe, and effective means of providing cooling and hydrating in a correctional facility.
- IV fluids with an isotonic saline solution may achieve a more rapid volume replacement in a shorter amount of time than oral fluids. IV fluids are appropriate for moderate to severe dehydration or HRI, and when persons cannot control their airway or drink sufficient fluids to correct the dehydration.

## 7. PREVENTION

The major components of a heat illness prevention program are EDUCATION, RISK FACTOR IDENTIFICATION, and MITIGATION OF HEAT RISKS. These components are discussed below.

**EDUCATION:** All staff and inmates should be educated seasonally about risk factors for and recognition of HRI, preventive strategies to reduce heat stress, and appropriate interventions.

**IDENTIFICATION OF RISK FACTORS:** It is important to assess inmates with HRI risk factors and to apply the corresponding MDS code in BEMR and Sentry when clinically appropriate.

#### MITIGATION OF RISK FACTORS:

- Acclimatization: Allow one to two weeks to adapt to a significantly hotter environment. Examples include an inmate newly arrived from a cooler climate or recently assigned to an outdoor work detail in the summer months.
- **Hydration:** Make available and encourage additional fluid intake before, during, and after periods of exertion or prolonged periods of non-exertional exposure to higher temperatures and humidity. A good rule to follow is to drink a sufficient amount of fluids (water, ice chips, sports drinks) to maintain body weight. Fluid intake should not exceed 12 quarts in any 24-hour period. (Inmates with medical conditions requiring fluid restrictions are likely to have a lower maximum fluid intake.)
- **Restrictions on exercise and work:** Reduce the duration and intensity of exertion. Allow more frequent breaks or periods of rest; limit periods of exertion to the cooler parts of the day, i.e., morning, late afternoon, or evening. During extreme heat conditions, temporarily close outdoor recreation areas and cancel non-essential outdoor work details.
- Encourage wearing of loose, light colored, and light-weight clothing in warmer temperatures, as permitted by uniform/dress requirements.
- **Environmental controls** to monitor for and lower excessively high temperatures in non-air-conditioned housing and work areas are to be determined by each institution.
- As the heat index increases, undertake additional protective measures. See the OSHA resources listed under <u>References and Resources</u>.

## **REFERENCES AND RESOURCES**

#### USING THE HEAT INDEX: A GUIDE FOR EMPLOYERS

OSHA's website on preventing and managing heat-related illness is available at: <u>https://www.osha.gov/heat/index.htmll</u>

- A printable PDF of the guide is available at: https://www.osha.gov/SLTC/heatillness/heat\_index/pdfs/protective\_measures.pdf
- Educational and training materials, both online and as printable PDFs, are available at: <u>https://www.osha.gov/pls/publications/publication.athruz?pType=Industry&pID=571</u>

## APPENDIX 1. NATIONAL WEATHER SERVICE (NWS) HEAT INDEX CHART AND HEAT ADVISORIES

Heat index temperatures are determined based on ambient outdoor air temperatures in shady conditions with light wind. Exposure to direct sunlight may increase heat index values as much as 15°F. The most recent version of the chart below is at: <u>http://www.wrh.noaa.gov/psr/general/safety/heat/heatindex.png</u>. More detailed information is available at: <u>http://www.nws.noaa.gov/om/heat/heat\_index.shtml</u>

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