



Heat Illness

Physiological Adaptation To Heat Stress

U.S. Army Research Institute of Environmental Medicine

Peer Review Status: Internally Peer Reviewed

Humans have well defined physiologic mechanisms to counteract rises in body temperature from either internal heat production or environmental heat stress. These mechanisms support our need to maintain a narrow range of body temperature for optimal function. In response to a rise in body temperature from an internal or external heat source, we increase both cutaneous blood flow and sweating. Heat energy is then dissipated to the environment either directly from the warmed skin surface by conduction/convection and radiation or by evaporation of sweat.

The direct transfer of heat energy to the environment occurs by radiation and conduction. Radiative transfer occurs between the body surface and all other sources of radiant energy. Conductive transfer occurs by direct contact between the body surface and other materials including air, water or ground. Both radiative and conductive heat transfer move heat energy between the body surface and some other material. Convection augments conductive heat transfer. Conduction occurs between two materials in direct contact. Conduction will stop when the two materials in contact reach thermal equilibrium. Convection prevents thermal equilibrium developing by constantly replacing at least one of the materials. For example, convection replaces air warmed by conduction while in contact with the skin surface with air not yet warmed so heat transfer can continue.

The rate of heat transfer by conduction/convection and radiation is dependent on the difference in temperature between the body surface and the materials or radiating surfaces in the environment. Furthermore, the two routes of direct energy exchange (radiation and conduction/convection) between the body surface and the environment are two-way streets. If the body surface is warmer than the environment, the body will lose energy to the environment. However, very warm air or surfaces will transfer heat to the body by conduction/convection; and sunlit surfaces or sky will transfer heat to the body by radiation.

Maintenance of body temperature requires that the amount of heat energy in the body remains constant. Under circumstances when internal heat production exceeds the capacity of direct routes of heat transfer to dissipate it, an additional means of heat transfer, evaporation of sweat, comes into play. Furthermore, when the environment is sufficiently hot to cause heat gain by the direct transfer routes, evaporative cooling is the only thermoregulatory mechanism available to control body temperature.

Sweating is primarily controlled by the central nervous system. Core temperature increments detected by thermosensitive neurons in the hypothalamus stimulate increases in skin blood flow and sweating. The process of sweating is triggered primarily by sympathetic cholinergic stimulation of the eccrine sweat glands, which are distributed ubiquitously over the body surface. Sweat production rates can reach 2 liters per hour for short periods and up to 15 liters per day. Each liter of sweat evaporated from the body

surface removes approximately 580 Kcal of heat energy. Under conditions that allow rapid evaporation (e.g., deserts), the daily cooling capacity of the sweating mechanism is several thousand kcal, adequate to maintain body temperature even during vigorous work in the heat.

Sweat is a hypotonic solution of sodium chloride. The concentration of sodium chloride in sweat depends on acclimatization state and sweating rate. Higher sweating rates reduce the opportunity for the eccrine secretory epithelium to conserve salt, so, at higher sweat rates, sweat salt concentration rises. Acclimatized sweat glands conserve salt more effectively and produce sweat with reduced salt concentration for any given flow rate. This conservative phenomenon is an important protection from salt depletion in hot environments.

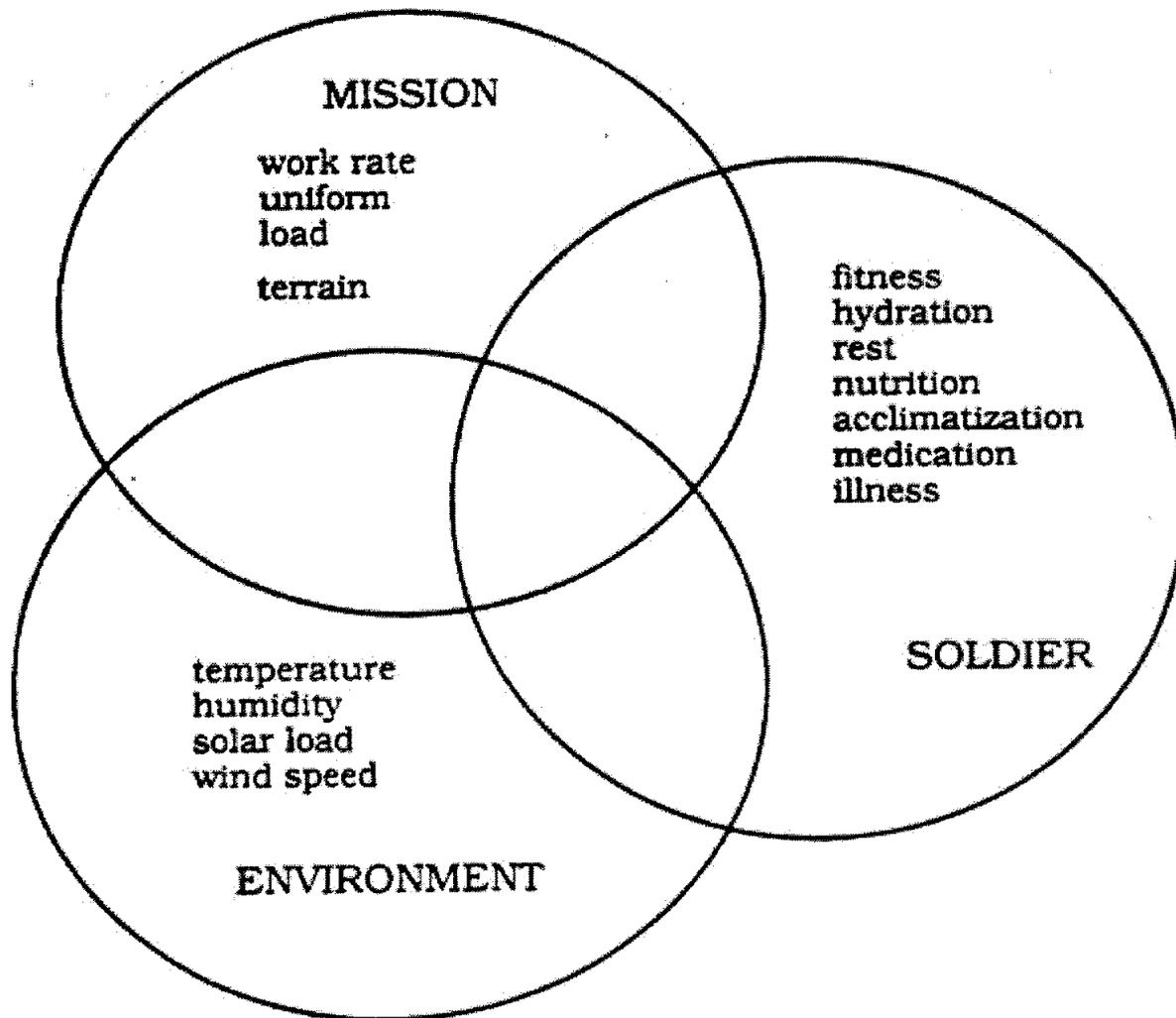
Furthermore, reducing the salt content of sweat increases the proportion of intracellular water contributing to sweat formation. Consequently, for any given amount of body water lost as sweat, less will be taken from the extracellular fluid, conserving, to a degree, plasma volume.

In addition to conserving salt, humans acclimatized to heat initiate sweating and evaporative cooling at lower body temperatures. In environments where sweating contributes to cooling acclimatized individuals can maintain lower body temperatures for any amount of heat stress.

Physical work causes an increase in cardiac output and the redistribution of blood flow toward the working muscles and away from the viscera. If an elevation in core temperature also occurs, then an additional portion of the cardiac output is directed to skin for thermoregulation and visceral flow is further reduced. Maintenance of effective circulating volume is essential to permit adequate muscular, visceral and thermoregulatory blood flow. High sweat rates will quickly compromise blood volume. Therefore, work in the heat requires constant fluid replenishment. Since net water absorption in the gut is about 20 cc per minute or 1200 cc per hour, compensation for high sweat rates requires rest periods with reduced sweat rates and time for hydration.

The degree of acclimatization is dependent on the degree of thermal stress to which the individual has been exposed. Regular vigorous exercise, even in temperate climates, produces sufficient elevation of core temperature to induce a small degree of heat acclimatization. However, any exposure to environment with significant additional heat stress will require an additional period of acclimatization.

Acclimatization develops at a rate that depends on the degree of heat stress imposed. Achieving the maximum rate seems to require about 2 hours of continuous exercise exposure per day.



HEAT STRESS IS THE PRODUCT OF AN INTERACTION OF MISSION, ENVIRONMENTAL AND HUMAN FACTORS.

ANALYSIS OF HEAT INJURY RISK BY THE UNIT SURGEON MUST CONSIDER ALL OF THESE FACTORS.

[Next Page](#) | [Previous Page](#) | [Section Top](#) | [Title Page](#)

[Virtual Naval Hospital Home](#) | [Help](#) | [Search](#) | [Site Map](#) | [Disclaimer](#) | [Comments](#)

cartographer@vnh.org

Collective copyright © 1997-2002 The University of Iowa. All rights reserved.

URL: <http://www.vnh.org/>



Heat Illness

Prevention of Heat Illness

U.S. Army Research Institute of Environmental Medicine

Peer Review Status: Internally Peer Reviewed

Introduction

Minimizing Heat Casualties in Basic Training

Minimizing Heat Casualties in Operations

Medical Planning and Readiness

Introduction

This section is intended to be a practical guide to preventing heat casualties in military training and operations. The first part introduces the three interacting factors that influence the risk of heat casualties: the **soldier**, the **environment** and the **mission**. The second part discusses preventing heat casualties among recruits in basic training and introduces the Appendices in the back of this handbook that can be used to estimate safe limits for heat stress exposure. The third part discusses operations planning to minimize heat casualties. The fourth section discusses medical planning and readiness to manage heat casualties should they occur.

Heat casualties are the result of the interaction of three factors: the condition of the **soldier**, the external heat stress from the **environment** and the internal heat stress required by the **mission**. Medical officers should assess each component in their preparation of plans for primary prevention of heat casualties.

The Soldier

A soldier is optimally capable to manage heat stress when he is fully hydrated, physically fit, acclimatized, well nourished and well rested.

Hydration is the most important element in a plan to prevent heat casualties. Full hydration is critical to the prevention of heat casualties because it is essential to maintain both blood volume for thermoregulatory blood flow and sweating. Both are reduced by dehydration. Consequently, the dehydrated soldier has less ability to maintain body temperature in the heat.

Water requirements are not reduced by any form of training or acclimatization. Exercises to teach soldiers to work or fight with less water are fruitless and dangerous.

!REMEMBER!

Soldiers cannot reduce thermoregulatory water requirements by water deprivation during training. Acclimatization does not reduce water requirements. Commanders must understand this principle and recognize its logistic and operational implications.

Avoiding heat casualties requires that soldiers drink enough water to replace what they lose. In hot environments, soldiers do not drink enough water to voluntarily maintain hydration. This phenomenon has been called "**voluntary dehydration**", although there is nothing willful about it. In hot environments, thirst is not stimulated until plasma osmolarity rises 1-2% above the level customarily found in temperate climates. Consequently, if thirst is used as the guide to drinking, soldiers will maintain themselves at a level that is 1-2% dehydrated relative to their usual state. If soldiers are to fully replace the water they lose in their daily activities and eliminate voluntary dehydration, they must understand the need to drink even though they are not thirsty and leaders must enforce water drinking discipline.

Even in the face of a clear understanding of the importance of water and hydration, soldiers may decide that water drinking creates problems that outweigh its importance. For example, soldiers may not drink before going to sleep to avoid having to wake up and dress to urinate or they may not drink before convoys if no rest stops are planned.

Units which have soldiers who do not drink because they do not have opportunities to urinate have a leadership problem. Unit leaders must reinforce hydration by planning for all aspects of adequate hydration: elimination as well as consumption.

The medical officer must be aware that soldiers may not follow drinking discipline. Be sure operations are planned so that drinking does not become a problem. Be aware of the soldiers hydration status. Urine color, body weight change and orthostatic blood pressure change can all be used as guides to hydration.

CONSEQUENCES OF DEHYDRATION

Acutely, mild dehydration (2-3% of body weight) reduces physical capacity and heat tolerance. As dehydration progresses, cognitive function deteriorates and both thermoregulation and physical capacity become seriously compromised. 5-6% dehydration is incompatible with further functioning.

Chronic mild dehydration is associated with renal stones and urinary infection, severe constipation, rectal afflictions and cutaneous drying.

In hot environments, water losses can reach **15 liters per day per soldier**. Complete replacement requires realistic estimates of potable water requirements, an adequate water logistic system and soldiers who understand and act on their water requirement. Water for hygiene will be needed in addition to water for drinking.

There is no advantage to carbohydrate/electrolyte beverages beyond their palatability which may encourage drinking. They should not be the sole source of water as they can be mildly hypertonic.

Aerobic fitness provides the cardiovascular reserve to maintain the extra cardiac output required to sustain thermoregulation, muscular work and vital organs in the face of heat stress. In addition, regular

strenuous aerobic physical training will provide a small degree of heat acclimatization.

Regardless of their physical condition, however, soldiers who are required to deploy on short notice to hot environments, will arrive incompletely acclimatized. Adequate **acclimatization** will require several days to achieve. During this initial acclimatization period, soldiers must be provided copious quantities of water and carefully supervised to prevent excessive heat exposure. If possible, work tasks should be regulated using work-rest cycles tailored to the soldiers' physical capacity by direct medical oversight.

In the first few days of acclimatizing, sweat salt conservation will not be fully developed. Salt depletion is a risk if soldiers are exposed during this time to sufficient heat or work stress to induce high sweating rates (>several liters per day), particularly if ration consumption is reduced. Salt depletion can be avoided by providing a salt supplement in the form of salted water (0.05 to 0.1%). A 0.1% salt solution can be prepared using the directions outlined in Appendix A to this handbook. Acclimatization should eventually eliminate the need for salt supplementation.

The requirements of military operations frequently mean lack of sleep and missed meals. All these factors reduce thermoregulatory capacity and increase the risk of heat injury. Recommendations to planning staffs should emphasize the importance of adequate sleep and food to reduce the likelihood of heat casualties.

Coincidental illnesses increase heat casualty risk through fever and dehydration. The consequences of dehydration are discussed above. Fever, whether due to immunization or illness, reduces thermoregulatory capacity by resetting the hypothalamus toward heat conservation rather than heat dissipation. This phenomenon eliminates the beneficial effect of acclimatization. Soldiers recovering from fever will have increased susceptibility to heat illness even after all clinical evidence of illness has disappeared. Until clearly able to manage normal work rates in the heat they will require increased command supervision and moderated work schedules.

The skin is a vital thermoregulatory organ. Sunburn and the other skin diseases of hot environments reduce the ability of the skin to thermoregulate. Sunburn must be prevented by adequate clothing, shade and sunscreens. Skin diseases are best prevented by adequate hygiene. Commanders and logisticians must understand the importance of a functioning skin and provide adequate water for washing.

Some medications will effect thermoregulatory adaptations and can increase the risk of heat illness. A list of such medications is in Appendix H.

The Mission

The physical exertion required to accomplish a mission is an important determinant of heat stress. Marching speed and route, load carried, work tasks required and terrain covered all will affect internal heat production. Appendix B categorizes various military tasks into four different work intensities. These four categories of work intensity are used throughout the remainder of the Appendices.

The level of Mission Oriented Protective Posture (MOPP) required to accomplish the mission affects heat strain in two ways. First, MOPP gear, particularly the chemical protective overboots, adds to the work of movement and increases internal heat production during the performance of a task. Second, the BDO and hood encapsulate and effectively isolate the soldier from the environment. Consequently, the soldier creates his own microenvironment within the chemical protective uniform. The air trapped in the uniform is warmed by the skin and saturated with water vapor from sweat, so that the soldier's immediate environment becomes extremely hot and humid. The only opportunity to moderate the heat

and humidity inside the uniform is to transfer water vapor and heat through the fabric, just the transfers the uniforms are designed to prevent.

Each alternative plan to accomplish the mission has its own particular constraints on the availability of water, shade and rest.

The Environment

The environmental heat stress to which soldiers will be exposed must be known if effective preventive measures are to be taken. It should be measured in circumstances as close as possible to those in which the troops will be operating. It can vary tremendously over short periods of time and space and in unpredictable ways. For example, on a sunny, calm day an open field may have the greater heat stress than an adjacent forest, but, on a windy, cloudy day the forest may have the greater heat stress. Heat stress indices calculated for a whole post or region are only general guides. Particularly when conditions seem extreme, on site measurements are essential. There is no substitute for knowledge of local conditions.

The U.S. Army has adapted the WBGT Index as the standard metric for environmental heat stress. Appendix C describes the calculation of the WBGT Index and summarizes general guidance for regulating physical training according to the WBGT value. A WBGT apparatus is available in the federal supply system (NSN 6665-00-159-2218). Appendix C contains directions for making a field expedient apparatus for measuring WBGT.

There are four environmental characteristics that influence heat stress:

the air temperature, the solar (or radiant heat) load, the dew point temperature and the wind speed.

Absolute air temperature (ambient temperature or "drv-bulb" temperature) is measured by a shaded thermometer to avoid any effect of radiant heat. By itself, it is a relatively small contributor to heat stress. It is weighted 10% in the calculation of the WBGT Index.

Solar load can be an important contributor to heat stress. Under severe conditions, full sun on bare skin can add up to 400 watts to an individuals heat load. Solar load is measured by the black globe thermometer. The black globe temperature is weighted 20% in the WBGT Index calculation.

Humidity determines the rate at which sweat can evaporate and is the principal component of the WBGT Index. The rate of sweat evaporation is determined by the difference between water vapor pressure at the skin surface and in the ambient air, which are measures of absolute humidity. The relative humidity, which is a measure of the saturation of ambient air with water vapor, does not determine the rate of sweat evaporation. The important point to remember is that a change in air temperature alone does not change the absolute humidity of the air; that is, the rate of sweat evaporation and cooling will not change just because air gets warmer or cooler.

The temperature recorded by a wet bulb thermometer provides the measure of absolute humidity for the WBGT Index. A wet bulb thermometer is a shaded standard thermometer whose bulb is surrounded by a wet cotton wick and exposed to moving air. The cotton wick cools as water evaporates from it. The rate of evaporation depends on the absolute humidity of the ambient air and is speeded by air movement. cooling effect of evaporation brings the temperature reported by the thermometer below that of the ambient air. Drier air means greater cooling effect and lower wet bulb temperature. And, in contrast, if the air is completely saturated with water vapor so that no evaporation is possible, then the wet bulb

thermometer will not be cooled below the temperature of the ambient air. In this case, the dry bulb and wet bulb temperatures will be the same.

The wet bulb temperature is the most important component of the WBGT Index, befitting the thermoregulatory importance of evaporation in hot environments. The wet bulb temperature is weighted 70% in the WBGT Index.

Wind speed is an important factor in determining environmental heat stress. Air movement increases convective heat transfer; cool winds reduce heat stress, hot winds increase it. Air movement will assist evaporation. The WBGT does not include any direct measure of wind speed.

Minimizing Heat Casualties in Basic Training

Recruits are particularly susceptible to heat illness during basic training in hot weather. A number of reasons for their susceptibility are related to their rapid transition from civilian life to a demanding schedule of physical and military training. First, they are neither acclimatized to heat on entry nor as physically fit as fully trained soldiers. They need to become fit in a short time and, so, quickly begin strenuous exercise. Second, they commonly suffer sleep loss and dehydration. Third, contagious febrile illnesses are common. Fourth, they are unfamiliar with heat illness and don't recognize early signs of heat illness or understand the importance of early treatment.

Heat illness can occur in any component of basic training. Certain activities, though, are associated with the highest risk. Those activities are road marches, unit runs (including morning PT), evening parades and rifle range marksmanship training.

Recruits doing **road marches** and **unit runs** have very high sustained rates of endogenous heat production and muscular work. They usually develop temperature elevations and, after 30-60 minutes, significant dehydration. Both temperature elevation and dehydration are aggravated if they begin their exercise dehydrated (as, for example, starting just after waking without directed rehydration) or if they are wearing a heavy uniform that prevents loss of heat to the environment (for example, chemical protective equipment) or if environmental conditions retard heat loss. The combined elevated temperature, muscular work and dehydration lead to high risk of heat exhaustion and heat stroke.

Heat casualties encountered at **evening parades** usually result from dehydration developed during a day of vigorous physical training.

At first glance, one would not ordinarily associate a significant risk of heat casualties with **rifle marksmanship training**. However, the association exists because, rifle range training is often done during extreme heat which prohibits other outdoor training. Recruits are exposed for long periods to intense solar and ground contact heat loads without consideration to the heat induced water requirement. Under these conditions, recruits develop hyperpyrexia and dehydration.

In recruit training, primary prevention of heat illness is instituted by using the following steps:

1. Assess the **recruits** who will be training. Consider their acclimatization, physical fitness and state of rest, nutrition and hydration.

Identify individuals or units at particular risk, for example, individual recruits recovering from a febrile

illness or units just beginning training. Provide safe alternative training for those identified at increased risk.

2. Measure the **environmental conditions** in which the training will take place. Remember that conditions can vary substantially even in a short distance and in unpredictable ways. A shaded forest may seem to have less heat stress because of the lower solar load, but may, in fact, have a HIGHER heat stress because of high humidity and lack of wind.

Have the environmental conditions become more stressful recently? Sudden increases in environmental heat stress are particularly risky. Recruits who have acclimatized to a moderate degree of heat stress will not be tolerant of sudden more severe heat stress.

3. Assess the work load of the proposed **training**. What work rate and duration is planned?

What uniform will the training be conducted in? Recruits will experience much greater heat strain in uniforms, such as the BDU, that restrict heat exchange with the environment.

Will the recruits have the opportunity to remove or loosen portions of the uniform during training? Unblousing trousers, removing jackets or helmets can reduce heat stress considerably.

Will the recruits be protected from solar heat load? Although loosening clothing can permit better evaporative and conductive/convective cooling, the skin and head should be protected from direct sun by shade or light clothing.

4. From the condition of the recruits, the environmental conditions, the work rate and uniform to be worn, use the Appendices in the back of this handbook to estimate water requirements and safe work times.

Use **APPENDIX B** to judge the work rate imposed by the training. Appendix B divides military work into four categories from **very light** to **heavy**. These categories are used throughout the handbook.

Running is much more demanding than even "heavy" military work. The Appendices that use the Appendix B military work categories **DO NOT APPLY TO RUNNING**. A separate Appendix D has been calculated for running.

The two figures in Appendix B (Figures B-1 and B-2) show the estimated rate of heat casualties in a unit for each work rate for WBGT Indices between 60° F and 100° F. Figure B-1 is calculated for soldiers wearing the BDU. The left hand edge of each shaded area represents the number of minutes until 5% of a unit would be disabled. The right hand edge of each shaded area represents the number of minutes until 20% of a unit would be disabled. For example, a unit of soldiers in optimal condition in MOPPO, performing moderate work at a WBGT Index of 90° F would be estimated to experience 5% heat casualties after 70 minutes and 20% casualties after 120 minutes if work continues uninterrupted.

It is **ESSENTIAL** to note that the estimates are calculations that assume the soldiers are fully rested, acclimatized, and hydrated and that the soldiers are euthermic at the start of work. In circumstances when those optimal conditions are not met, the estimates of tolerance time must be revised downward. For example, if the unit in MOPPO performing moderate work in a WBGT Index of 90° F were suffering from dehydration, the time to 5% casualties would be expected to be shorter.



Heat Illness

Management of Heat Illness

U.S. Army Research Institute of Environmental Medicine

Peer Review Status: Internally Peer Reviewed

Heat Exhaustion

Heat Cramps

Heat Stroke

Minor Heat Illnesses

Heat Exhaustion

Heat exhaustion is the most commonly encountered form of heat illness. It occurs when the cardiac output is insufficient to meet the competing demands of thermoregulatory skin blood flow, skeletal muscle and vital organs. Heat exhaustion is usually due to the combination of increased circulatory load due to thermoregulatory and muscular demand and reduced "effective" plasma volume and venous return due to vasodilation in skin and muscle and sweating-induced depletion of salt and water. Heat exhaustion, by definition, is a "functional" illness and is not associated with evidence of organ damage.

Classically, heat exhaustion has been divided into two forms: salt depletion heat exhaustion and water depletion heat exhaustion. In practical clinical terms, neither entity is encountered in a "pure" form; rather, classic heat exhaustion always includes elements of both water and electrolyte depletion which are present in variable proportions.

1. Pathogenesis

The proximate cause to heat exhaustion is the inability of the cardiovascular system to meet the demands of thermoregulatory, muscular and visceral blood flow. The fluid and electrolyte depletion associated with exposure to hot environments acts synergistically with the increased demand for cardiac output by reducing the volume of extracellular fluid available for the maintenance of plasma volume and venous return.

Salt depletion in hot environments develops from increased salt loss in sweat (particularly among the unacclimatized) and reduced salt intake due to anorexia. Salt depletion usually develops over several days, so the contraction of extracellular fluid is gradual and symptoms develop slowly. The reduced extracellular fluid volume produces symptoms of fatigue and orthostatic dizziness. Because salt depletion does not produce intracellular hypertonicity, thirst is not prominent until the extracellular fluid volume (ECF) has contracted enough to cause volumetric stimulation of thirst. Nausea and vomiting are common but of unknown mechanism. Hemoconcentration occurs due to the contraction of ECF. Muscle

cramps are a common accompaniment of salt depletion (see "Heat Cramps" below). Potassium depletion commonly accompanies salt depletion due to diminished intake and mineralocorticoid driven kaliuresis. Frank hypokalemia is uncommon.

Water depletion in hot environments develops from sweat rates sufficiently in excess of water replacement rates to produce hypertonic dehydration. Even though the loss of water occurs from both intracellular and extracellular compartments, the rate of dehydration is usually quite rapid and symptoms evolve quickly. Thirst is prominent due to hypertonicity. Oliguria, clinical dehydration, tachycardia and tachypnea with symptomatic hyperventilation are all prominent clinical features.

2. Diagnosis

Presenting complaints in heat exhaustion include: thirst, syncope, profound physical fatigue, nausea, vomiting, symptomatic hyperventilation with acroparesthesia and carpopedal spasm, dyspnea, muscle cramps, confusion, anxiety and agitation, mood change, orthostatic dizziness, ataxia, hyperthermia and frontal headache. The symptoms of heat exhaustion are non-specific and no combination of presenting symptoms and signs is pathognomonic. Each patient requires careful clinical evaluation addressed to the presenting complaint.

Heat exhaustion is frequently superimposed on other conditions that increase circulatory load, such as febrile illness, or produce fluid-electrolyte losses, such as gastroenteritis.

At the first opportunity the following objective data should be obtained to support the clinical analysis and management of potential heat exhaustion: careful vital signs including orthostatic blood pressure and rectal temperature, CBC (including platelet count), serum electrolytes, BUN, creatinine, glucose and U/A. If heat stroke is suspected, FT, APTT, Fibrin split products or analog, liver enzymes, CK isoenzymes, LOG and CXR should be obtained as soon as possible. Other data are obtained as needed to complete the differential diagnosis of the presenting complaint. Rectal temperature should be frequently monitored to ensure that core temperature is falling to normothermic levels.

3. Management

The management of heat exhaustion is directed to correcting the two pathogenic components of the illness: excessive cardiovascular demand and water-electrolyte depletion. The load on the heart is reduced by rest and cooling. Water-electrolyte depletion is corrected by administering oral or parenteral fluids.

Heat exhausted patients do not REQUIRE active cooling measures: removal of heavy clothing and rest in a shaded and ventilated space provides an adequate opportunity for spontaneous cooling. However, if available, cool water can be used to cool the skin. The consequent cutaneous vasoconstriction will rapidly reduce circulatory demand and improve venous return.

Heat exhaustion casualties retain the ability to cool spontaneously if removed from the stressful circumstances. However, spontaneous cooling is necessarily observed only **AFTER** cooling has occurred. Since casualties with heat stroke and heat exhaustion are hard to distinguish initially, medical personnel who elect to delay active cooling to see if a casualty can spontaneously cool, will occasionally fail to provide immediate active cooling for a casualty with heat stroke. The **SAFEST COURSE** is to provide active cooling for all casualties

who are at risk for heat stroke.

Intravenous fluids replenish the extracellular volume quickly. Oral fluids suffice for those patients who can take fluids without risk of vomiting. However, clinical observation suggests parenteral fluids produce more rapid recovery than oral fluids, probably because of the slower absorption of oral fluids. Patients with evidence of clinically significant plasma volume depletion (tachycardia at rest or orthostatic signs) should initially receive normal saline in 200-250 cc boluses in an amount sufficient to restore normal circulatory function. No more than 2 liters of NS should be administered without laboratory surveillance. Subsequent parenteral fluid replacement should be D5/O.5 NS or D5/0.2 NS. Individuals with significant salt depletion have coincident potassium depletion, often amounting to 300-400 meq of KCl. To begin the restoration of the potassium deficit, inclusion of potassium in parenteral fluids after volume resuscitation is appropriate if there is no evidence of renal insufficiency or rhabdomyolysis. Oral fluids should not be given until all risk of vomiting has abated. Significant hypernatremia should be corrected slowly to avoid cerebral edema.

4. Recovery and Profiling

Patients with heat exhaustion experience rapid clinical recovery. However, they all need at least 24 hours of rest and rehydration under first echelon or unit level medical supervision to reverse their water-electrolyte depletion.

Any patient in whom the diagnosis of heat stroke is possible will need at least 72 hours to complete an adequate period of observation, rest and rehydration at a second or third echelon MTF. Patients who are clinically well but still being observed can be assigned supervised light duty at the MTF if shade and water are plentiful. Under no circumstances should they be reexposed to significant heat stress during this period.

A single episode of heat exhaustion does not imply any predisposition to heat injury. No profile is required. An attempt should be made to determine the reason for the heat exhaustion. e.g., insufficient work-rest or water discipline, coincident illness or medication, etc. The individual should return to his unit with advice, both to the soldier and the chain-of-command, about how the incident happened and how to avoid similar episodes in the future.

Repeated episodes of heat exhaustion require thorough evaluation. Soldiers should not be returned to duty. They should be evacuated to a referral facility with a temporary profile against heat exposure.

Heat Cramps

1. Pathogenesis

The etiology of heat cramps is not known. Generally, heat cramps occur in salt-depleted patients generally during a period of recovery after a period of work in the heat. Whole body salt depletion is thought to be associated with the cause of the muscle contraction of heat cramps. Salt supplementation has been found to reduce the incidence of heat cramps in industrial populations at risk.

2. Diagnosis

Patients with heat cramps present with painful tonic contractions of skeletal muscle. The cramp in an individual muscle is usually preceded by palpable or visible fasciculation and lasts 2-3 minutes. Cramps are recurrent and may be precipitated by manipulation of muscle. The cramps involve the voluntary muscles of the trunk and extremities. Smooth muscle, cardiac muscle, the diaphragm and bulbar muscles are not involved. Pain in cramping muscle is severe. There are no systemic manifestations except those attributable to pain. Despite the salt-depletion associated with heat cramps, frank signs and symptoms of heat exhaustion are unusual. The cramps can occur during work or many hours after work.

The diagnosis of heat cramps is usually straightforward. The differential diagnosis includes tetany due to alkalosis (hyperventilation, severe gastroenteritis, cholera) or hypocalcemia, strychnine poisoning, black widow spider envenomation or abdominal colic. These entities should be distinguishable on clinical examination.

3. Management

Replenishment of salt orally or parenterally resolves heat cramps rapidly. The response to therapy is sufficiently dramatic to be valuable in the differential diagnosis. The route of administration should be determined by the urgency of symptom relief. Salt tablets should not be used as an oral salt source. If oral salt replenishment is to be used to treat heat cramps, use 0.1% salt solution. (SEE APPENDIX A)

No significant complications have been reported from heat cramps except muscle soreness.

4. Recovery and Profiling

Patients with heat cramps usually have substantial salt deficits (15-30 grams, 2-3 days usual dietary intake). These individuals should be allowed 2-3 days to replenish salt and water deficits before resuming work in the heat.

An episode of heat cramps does not imply any predisposition to heat injury. No profile is needed except to assure an adequate period of recovery.

As with heat exhaustion, an attempt should be made to determine the reason for the episode so that appropriate advice can be given to the soldier and chain-of-command to avoid future episodes.

Heat Stroke

HEAT EXHAUSTION VS. HEAT STROKE

At presentation, the distinction between heat exhaustion and heat stroke, in all but the most extreme cases, is impossible. Individuals who do not respond dramatically to rest and fluid-electrolyte repletion should be observed for 24 hours with laboratory surveillance for the delayed complications of heat stroke. Encephalopathy, coagulopathy or persistent elevation of body temperature suggest the probability of severe heat stroke. Immediate institution of active cooling and evacuation to a rear echelon hospital is required. Active cooling should be continued throughout evacuation.

Since the renal and hepatic complications of heat stroke can be delayed for 48-72 hours, any evidence of renal or hepatic injury during the initial 24 hours of observation should lead to the presumptive diagnosis of heat stroke. These patients should be evacuated to rear echelons for further evaluation, medical care and rehabilitation.

I. Pathogenesis

Heat stroke is distinguished from heat exhaustion by the presence of clinically significant tissue injury. The degree of injury appears to relate to both the degree of temperature elevation and duration of exposure. Since the degree of illness in patients with heat stroke is not entirely predicted by the magnitude of temperature elevation and duration, other pathogenic factors including tissue ischemia, hypokalemia, exercise induced lactic acidosis, endotoxemia, and activation of intravascular coagulation probably have a role in the evolution of heat stroke.

Heat stroke occurs in two settings sufficiently different to produce different clinical pictures and management. "Classical" heat stroke occurs in individuals, frequently with impaired thermoregulation due to illness or medication, exposed passively to heat and dehydration. It is principally an epidemic affliction of young children and elderly occurring during urban heat waves. "Exertional" heat stroke occurs in physically active individuals experiencing substantial endogenous heat loads. The primary clinical difference between the two is that exertional heat stroke is complicated by acute rhabdomyolysis with consequent renal failure.

Five organ systems, the brain, hemostatic, liver, kidneys and muscle, are the principal foci of injury in heat stroke.

Encephalopathy is a sine-qua-non of heat stroke. Its presentation ranges from syncope and confusion to seizures or coma with decerebrate rigidity. The etiology of encephalopathy is not known.

Coagulopathy due to DIC is common. The principal causes of DIC seem to be thermal damage to endothelium, rhabdomyolysis and direct thermal platelet activation causing intravascular microthrombi. Fibrinolysis is secondarily activated. Hepatic dysfunction and thermal injury to megakaryocytes slows the repletion of clotting factors.

Hepatic injury is common. Transaminase enzyme elevation, clotting factor deficiencies and jaundice can be seen in the course of heat stroke.

Renal failure following heat stroke can be caused by several factors: myoglobinuria from rhabdomyolysis in exertional heat stroke, acute tubular necrosis due to hypoperfusion, glomerulopathy due to DIC, direct thermal injury and hyperuricemia.

Rhabdomyolysis is a frequent acute complication of exertional heat stroke. Acute muscular necrosis releases large quantities of potassium, myoglobin, phosphate and uric acid and sequesters calcium in the exposed contractile proteins.

If heat stroke is suspected and temperature is elevated, cooling should not be delayed to accomplish a diagnostic evaluation. Cooling and evaluation should proceed simultaneously

2. Diagnosis

Heat stroke presents as collapse with variably severe encephalopathy and hyperthermia. There may be clinical evidence of dehydration, coagulopathy or shock.

The differential diagnosis includes infection (particularly meningococemia and *P. falciparum* malaria), pontine or hypothalamic hemorrhage, drug intoxication (cocaine, amphetamines, phencyclidine, theophylline, tricyclic antidepressants), alcohol or sedative withdrawal, severe hypertonic dehydration and thyroid storm.

Laboratory evaluation should be directed by the differential diagnosis appropriate for the clinical circumstances. Patients with heat stroke require serial monitoring of platelets and plasma clotting factors, renal and hepatic function and electrolyte and acid-base status.

The patient with heat stroke requires early evacuation to medical facilities with intensive care capabilities. Active cooling should be started immediately and continued during evacuation.

3. Management

a. Emergency Care

i. The clinical outcome of patients with heat stroke is primarily a function of the magnitude and duration of temperature elevation. Therefore, the most important therapeutic measure is rapid reduction of body temperature. Any effective means of cooling is acceptable. A variety of techniques have been used. No particular technique has been unequivocally demonstrated to be superior.

Immersion in cool or iced water with skin massage is a classic technique for cooling heat stroke patients. Both have demonstrated effectiveness in lowering body temperature. Ice water probably produces the most rapid rate of cooling. However, ice water is an uncomfortable environment in which to work and, in the field, is very difficult to obtain.

Conscious patients will occasionally fight ice water immersion complicating management. Cool water is less demanding logistically and less uncomfortable for the medical attendants. In hot dry environments, field expedient immersion baths which will keep water cool can be constructed by digging plastic-lined shaded pits (The water is cooled by contact with cool subsurface sand and surface evaporation) or by rigging shallow canvas tubs in elevated frames in ventilated shade (The water is cooled by evaporation from the wetted canvas surface. In the case of canvas tubs the water can cool to nearly the atmospheric dew point temperature, often as low as 50 F in deserts.) If immersion devices cannot be prepared in advance, cool water can be kept available in Lyster bags. Heat stroke patients frequently have diarrhea and vomiting. The water immersion baths should be disinfected between cases.

Although not as effective as immersion, cooling can also be accomplished by wetting the body surface and accelerating evaporation by fanning. The water can be applied by spraying or by application of thin conforming cloth wraps (sheets, cotton underwear).

Circulating cooling blankets (unlikely to be available in the field situation) will also lower body temperature. Although cooling blankets have the advantage of maintaining a dry working environment, their limited contact surface provide slower cooling than immersion or surface wetting techniques. Their

best use is probably maintaining normal body temperature in the period after resuscitation and rapid cooling where temperature instability is characteristic.

Invasive cooling techniques have been tried including ice water lavage or enemas and peritoneal lavage with cool fluids. These techniques do not provide faster cooling and have the additional disadvantages potential complications and substantial inappropriate fluid loads. These techniques are not recommended.

While cooling is underway, rectal temperature should be closely monitored. Active cooling should be discontinued when the rectal temperature reaches 39° C to avoid hypothermia.

ii. Heat stroke patients usually do not require aggressive fluid resuscitation. Fluid requirements of 1 to 1.5 liters in the first few hours are typical. Over-replacement carries the risk of congestive heart failure, cerebral edema and pulmonary edema. Since heat stroke patients are frequently hypoglycemic, the initial fluid should include dextrose.

Hypotensive patients who do not respond to saline should receive inotropic support. Isoproterenol has been reported anecdotally to be helpful. Careful titrated use of dopamine or dobutamine is also reasonable and has the potential added advantage of improving renal perfusion.

Pulmonary artery wedge pressure monitoring should be used in patients with persistent hemodynamic instability.

iii. Airway control is essential. Vomiting is common and endotracheal intubation should be used in any patient with a reduced level of consciousness. Supplemental oxygen should be provided when available.

iv. Patients are frequently agitated, combative or seizing. Valium is effective for control and can be administered iv, endotracheally or rectally. The sedated heat stroke patient should be intubated. Nasogastric intubation to control vomiting should be done as soon as practicable.

v. Hyperkalemia is the most life threatening early clinical problem. Measurement of plasma [K] is an early priority. Tall T-waves on the surface electrocardiogram are consistent with hyperkalemia but not definitive. The interpretation of plasma [K] early in the clinical course of heat stroke is difficult due to confounding electrolyte and acid-base disturbances. Clinically significant hyperkalemia is manifested by electrocardiographic changes including increased T wave amplitude, slowed A-V conduction with widening of the P-R interval, diminishing P wave amplitude and "sine wave" ventricular rhythms. Hyperkalemia greater than 6.5 meq/l or with electrocardiographic changes should be treated. Glucose (50 gms slow iv), insulin (20 units of regular insulin iv) and sodium bicarbonate (1-2 amps iv) will lower plasma [K] within minutes. Serious ventricular dysrhythmia should be treated with iv calcium gluconate (1-2 amps). Cardiac monitoring and electrocardiography can be used to supplement laboratory monitoring for changes in plasma potassium (T wave amplitude) and calcium (QT interval).

vi. Acute renal injury is common in exertional heat stroke. Urinary catheterization to monitor urine output and obtain urine for [Na] should be done early. The oliguric patient with a casts, pigmenturia or red cells and a urine [Na] greater than 30 meq/l (before diuretics) has a high likelihood of acute renal failure. Early management of suspected acute renal failure should include assuring adequate renal perfusion and mannitol (12.5-25 grams iv).

b. Continuing Care

After cooling and hemodynamic stabilization, continuing care is supportive and is directed at the complications of heat stroke as they appear.

i. Patients with heat stroke frequently have impaired temperature regulation for several days with alternate periods of hyperthermia and hypothermia. Constant monitoring is essential and clinically significant deviations in temperature may require either cooling or warming measures. It is important to remember that changes in temperature may be due to reasons OTHER than hypothalamic instability, such as infection.

ii. The effects of rhabdomyolysis that require management are renal injury due to myoglobinuria and hyperuricemia, hyperkalemia, hypocalcemia and compartment syndromes due to muscle swelling. Assurance of adequate renal perfusion and urine flow will moderate the nephrotoxic effects of myoglobin and uric acid. Hyperkalemia can be managed by kayexalate or dialysis. The hypocalcemia does not usually require treatment. Increasing tenderness or tension in a muscle compartment may represent increasing intracompartmental pressures. Direct measurement of intramuscular pressure or fasciotomy should be considered at this point. Pain and paresthesia may not signal the compartment syndrome until permanent damage has occurred.

iii. Prognosis is worse in patients with more severe degrees of encephalopathy. Permanent neurologic sequelae can develop after heat stroke including cerebellar ataxia, paresis seizure disorder and cognitive dysfunction.

Management of encephalopathy is supportive, directed at minimizing cerebral edema by avoiding fluid overreplacement and assuring hemodynamic, thermal and metabolic stability. Intravenous mannitol has been used to treat life threatening cerebral edema if renal function is adequate. The efficacy of dexamethasone for treating heat stroke induced cerebral edema is not known.

Neurologic deterioration after initial recovery may represent intracranial hemorrhage related to DIC or hematoma related to trauma unrecognized at the time of initial presentation.

iv. Subclinical coagulopathy does not require active management. Clinically significant bleeding is an ominous sign. Treatment is directed at reducing the rate of coagulation and replacement of depleted clotting factors. Intravascular coagulation can be slowed by heparin infusion (5-7 units/kg per hour), followed in 2-3 hours by fresh frozen plasma and platelets. The administration of heparin will interfere with the usual laboratory measures of coagulation. However, successful management leads to a decline in indices of fibrinolysis (for example, fibrin split products). Heparin is tapered gradually over 2-3 days as directed by laboratory evidence of control.

v. Management of acute renal failure requires exquisite attention to fluid and electrolyte balance. Uremic metabolic acidosis and hyperkalemia require dialysis for control.

vi. Other complications include gastrointestinal bleeding, jaundice due to hepatic injury, pneumonia, noncardiogenic pulmonary edema and myocardial infarction. Immunoincompetence and infection are late complications, particularly in patients with severe renal failure.

4. Recovery and Profiling

Patients with heat stroke will require prolonged convalescence. They should receive profiles restricting heat exposure until clinical recovery is complete and their heat tolerance has been evaluated.

Heat intolerant individuals are considered to have either limited thermoregulatory response to heat stress or limited capacity for heat acclimatization and, therefore, be predisposed to heat injury. Certain diseases are well known to cause heat intolerance (a classic example is congenital ectodermal dysplasia in which sweat glands are absent). Heat stroke has been considered to be evidence for heat intolerance. However, a recent study was able to demonstrate measurable heat intolerance in only 1 of 10 individuals after recovery from heat stroke.

Minor Heat Illnesses

1. Miliaria Rubra, Miliaria Profunda and Anhidrotic Heat Exhaustion

Miliaria rubra is a subacute pruritic inflamed papulovesicular skin eruption which appears in actively sweating skin exposed to high humidity. In dry climates, miliaria is confined to skin sufficiently occluded by clothing to produce local high humidity. Each miliarial papulovesicle represents an eccrine sweat gland whose duct is occluded at the level of the epidermal stratum granulosum by inspissated organic debris. Eccrine secretions accumulate in the glandular portion of the gland and infiltrate into the surrounding dermis. Pruritus is increased with increased sweating. Miliarial skin cannot fully participate in thermoregulatory sweating, and therefore, the risk of heat illness is increased in proportion to the amount of skin surface involved. Sleeplessness due to pruritus and secondary infection of occluded glands have systemic effects that further degrade optimal thermoregulation.

Miliaria is treated by cooling and drying affected skin, avoiding conditions that induce sweating, controlling infection and relieving pruritus. Eccrine gland function recovers with desquamation of the effected epidermis, which takes 7 to 10 days.

Miliaria that becomes generalized and prolonged (miliaria profunda) can cause an uncommon but disabling disorder: anhidrotic heat exhaustion (or tropical anhidrotic asthenia). The lesions of miliaria profunda are presumed to develop from persistent miliarial lesions by superimposing inflammatory obstruction of the eccrine duct below the epidermal level of the inciting obstruction. The lesions are truncal, noninflamed, papular, with less evidence of vesiculation than the lesions of miliaria rubra. They may only be evident during conditions of active sweat production. Sweat does not appear on the surface of affected skin. The lesions are asymptomatic, which may explain why the patient does not seek medical evaluation early in the course.

Miliaria profunda causes a marked inhibition of thermoregulatory sweating and heat intolerance similar to that of ectodermal dysplasia. Symptoms of heat exhaustion and high risk of heat stroke occur under conditions well tolerated by other individuals. Management of miliaria profunda requires evacuation to a cooler environment for several weeks to allow restoration of normal eccrine gland function.

2. Heat Syncope

Syncope occurring on standing in a hot environment has been called "heat syncope". Heat syncope is probably not a discrete clinical entity. Rather, thermal stress increases the risk of classic neurally-mediated (vasovagal) syncope by aggravating peripheral pooling of blood in dilated cutaneous vessels. No special heat-related significance should be assigned to syncope occurring in these circumstances. Clinical evaluation and management should be directed toward the syncopal episode, not potential heat illness.

However, syncope occurring during or after work in the heat or after more than 5 days of heat exposure should be considered evidence of heat exhaustion.

3. Heat Edema

Mild dependent edema ("deck legs") is occasionally seen during the early stages of heat exposure while plasma volume is expanding to compensate for the increased need for thermoregulatory blood flow. In the absence of other disease, the condition is of no clinical significance and will resolve spontaneously. Diuretic therapy is not appropriate and may increase the risk of heat illness.

4. Sunburn

Sunburn reduces the thermoregulatory capacity of skin and, as any injury, has systemic effects, including fever, that influence central thermoregulation. Sunburn should be prevented by insisting on the use of adequate sun protection. When it does occur, effected individuals should be kept from significant heat strain until the burn has healed.

5. Heat tetany

Heat tetany is a rare condition which occurs in individuals acutely exposed to overwhelming heat stress. Extremely severe heat stress induces hyperventilation which appears to be the principal pathophysiologic etiology. The manifestations of heat tetany are characteristic of hyperventilation. They include respiratory alkalosis, carpopedal spasm and syncope. Management required removal from heat and control of hyperventilation. Dehydration and salt depletion are not prominent features.

[Next Page](#) | [Previous Page](#) | [Section Top](#) | [Title Page](#)

[Virtual Naval Hospital Home](#) | [Help](#) | [Search](#) | [Site Map](#) | [Disclaimer](#) | [Comments](#)

cartographer@vnh.org

Collective copyright © 1997-2002 The University of Iowa. All rights reserved.

URL: <http://www.vnh.org/>

<http://www.vnh.org/HeatIllness/manageht.html>

Modified: Wed Dec 19 15:30:49 2001

Displayed: Fri Apr 12 07:38:51 2002