

## ENVIRONMENTAL THERMAL STRESS

Samuel M. Keim<sup>1</sup>, John A. Guisto<sup>1</sup>, John B. Sullivan, Jr<sup>2</sup><sup>1</sup>Department of Emergency Medicine, Arizona Health Sciences Center, Tucson, Arizona, USA<sup>2</sup>Division of Clinical Toxicology, Arizona Poison and Drug Information Center, Arizona Health Sciences Center, Tucson, Arizona, USAKeim SM, Guisto JA, Sullivan JB, Jr: Environmental Thermal Stress. *Ann Agric Environ Med* 2002, 9, 1–15.

**Abstract:** Thermal stress from cold and heat can affect health and productivity in a wide range of environmental and workload conditions. Health risks typically occur in the outer zones of heat and cold stress, but are also related to workload. Environmental factors related to thermal stress are reviewed. Individuals undergo thermoregulatory physiologic changes to adapt and these changes are reviewed. Heat and cold related illnesses are reviewed as well as their appropriate therapy. Published standards, thresholds and recommendations regarding work practices, personal protection and types of thermal loads are reviewed.

**Address for correspondence:** Samuel M. Keim, MD, Department of Emergency Medicine, PO Box 245057, Tucson Arizona 85724-5057. E-mail: sam@aemrc.arizona.edu

**Key words:** heat, cold, body temperature regulation, heat stress disorders, environmental disorders, occupational health, occupational diseases, occupational exposure, hypothermia, heat exhaustion, heat stroke.

## INTRODUCTION

Thermal stress can be broadly divided into cold stress and heat stress. Cold stress is classified as hypothermia from cold water immersion, cold water submersion, exposure to environmental elements such as cold weather, rain or snow, ice, and frostbite. Heat stress includes heat exhaustion and heat stroke from hyperthermia.

Health risks occur in the outer zones of heat and cold stress across the continuum of thermal stress. The individual under environmental thermal stress, either hot or cold, undergoes thermoregulatory physiologic changes to adapt. According to the laws of thermodynamics, heat is transferred from a high temperature to a lower temperature. The body loses heat when environmental temperatures are lower and gains heat when environmental temperatures are higher. Heat transfer in either direction occurs by 4 mechanisms:

- Conduction,
- Convection,
- Radiation,
- Evaporation.

Environmental factors relating to heat transfer are a combination of air temperature, wind speed, relative humidity and radiation. Added to these are human factors of age, sex, workload upon the individual, pre-existing health problems, medications which can interfere with physiologic thermoregulation, clothing and metabolic rate.

Thermal stress is a risk factor in many different environmental situations and occupations. For example, outdoor temperatures vary between -50°C and 55°C depending on the geographic and site meteorological conditions.

## HEAT STRESS

Humans, as homeothermic creatures, attempt to maintain constant core body temperature. Obviously, this requires balancing heat production and absorption with heat loss. The balance is achieved by a combination of physiologic and behavioral mechanisms.

Heat stress results in physiologic responses of increased temperature, increased heart rate and increased sweating. Individuals can acclimatize to heat stress to some degree

by acclimatizing introduction to such environments over a period of days.

The skin performs a major function of heat exchange with the environment through blood flow. The temperature difference between the skin and the environment affects heat exchange. There can be either heat loss or heat gain. Sweating helps remove heat through evaporation, increasing the loss of heat energy from the skin and cooling the skin. Normally, the body is able to balance heat gain with heat losses so heat storage rate is zero.

Heart rate and rate of sweating are two physiologic measures relating to degree of heat stress. As individuals acclimatize to heat they are able to sweat more and therefore lose more heat. As cooling increases, the heart rate and core temperature will drop.

**Heat production.** The normal human internal temperature ranges from 36–38°C (37°C = 98.6°F). Limits for efficient thermoregulation are 35–40°C. In a resting adult, the normal heat production is approximately 60–70 kcal/hr/m<sup>2</sup> of body surface or 100 kcal/hr [18, 31, 51]. In the absence of heat loss mechanisms, this could result in a temperature rise of about 1°C per hour.

Walking produces 250–300 kcal/hr. With exercise, 70–100% of the metabolic rate is released as heat that must be dissipated to maintain thermoregulatory balance. In times of maximal exertion, heat production can exceed 1,000 kcal/hour, which could produce core temperatures consistent with heatstroke within a matter of minutes. Thus, endogenous heat production could overwhelm heat loss mechanisms and produce heat illness even in the absence of an excessively hot environment [18, 51]. Cellular metabolism increases 13% for each 1°C rise in body temperature until the body reaches heatstroke [18]. If there is no mechanism to dissipate heat, the addition of 70 cal to a 70 kg person would be able to theoretically raise core temperature 0.8°C (1.4°F) [18]. Heat stress or exercise can increase the core temperature to 40–42°C (104–107.6°F) [18]. At such temperature extremes, thermoregulatory mechanisms must adapt or heat stroke can occur.

**Environmental Heat Exchange.** In addition to endogenous heat load, an environment of high ambient temperature and/or humidity will contribute to the body's storage of excess heat, with resultant rise in core temperature. Transfer of heat energy between the body and the environment occurs via the 4 basic thermodynamic mechanisms: (1) radiation, (2) conduction, (3) convection, and (4) evaporation. Each of these mechanisms can be affected by combination of physiologic and behavioral responses.

**Radiation.** Radiation refers to the movement of heat between body and environment via electromagnetic waves. The body absorbs and emits thermal radiation as does all matter. Solar radiation in hot climates may present a significant thermal stress. Clothing will significantly

alter the radiant heat loss or gain, but clothing can reduce the effectiveness of cooling by evaporation [22, 18].

Solar load is a major source of heat gain in hot climates and ranges up to 250 kcal/hr in a partially clothed human and up to 100 kcal/hr in a fully clothed person [18]. Pigmented skin absorbs 20% more heat than non-pigmented skin. Increased skin blood flow maximizes heat loss from the body by delivering heat to the skin so it can evaporate sweat [18].

**Conduction.** Conduction refers to energy transfer between surfaces in direct contact. Most of the time an individual will compensate appropriately to avoid conductive heat loss. This would include vasomotor control of blood flow to the exposed body part, contacting or moving away from a heat source or heat sink, and interposing insulation [18].

**Convection.** Convection is the transfer of heat energy between a surface and a gas or liquid. In addition to simple temperature gradient, the rate of motion of the gas (air movement) or liquid (water) and its relative heat storage capacity will affect the rate of energy transfer. However, the compensatory mechanisms remain essentially the same as for conduction [18]. Convective heat exchange varies with the velocity of air movement. Heat loss following cold water immersion is rapid because of the high thermal conductivity of water, and the greater surface area of the body exposed to cold water, which is 32 times that of air [18].

**Evaporation.** The most important mode of cooling during times of environmental heat stress is evaporative. Approximately 580 kcal are lost for each liter of sweat evaporated from the body surface. The head and upper body are responsible for the majority of evaporative loss, thus wearing a hat and shirt or coat can interfere with cooling significantly [52]. Humidity and motion of either air or the body also significantly affect sweating efficiency. Perspiration which drips from the body before it can evaporate contributes essentially no cooling, and evaporation of sweat which has soaked into overlying clothing will cool the body much less than evaporation directly from the skin [16].

The maximum rate of sweat vaporization from the skin's surface depends on the dryness of the air and air movement. The environment's capacity to vaporize sweat depends on the relative humidity and wind velocity. As humidity approaches 100%, sweat vaporization becomes minimal [18].

The risk of hyperthermia increases with increasing humidity and increasing air temperature. But while exertion related heat stroke is prone to occur in hot, humid environments when individuals are not acclimatized, it can also occur when the temperature is not very hot. This is because exertion induced heat stroke can occur when the rate of heat production exceeds the rate of heat loss [18].

**Physiologic Control and Thermoregulation.** Thermo-regulatory physiologic mechanisms include autonomic nervous system and behavioral processes that alter or modify heat production and heat loss. Psychologic factors are:

- Appropriate personal protection and choice of clothing
- Rate of skin blood flow
- Vasodilation and vasoconstriction
- Sweating
- Shivering
- Changes in basal metabolism
- Acclimatization

These physiologic factors interact to maintain a narrow range of internal body temperature. The anterior hypothalamus controls physiologic temperature regulation mechanisms through the autonomic nervous system [9]. The hypothalamus may not actually function as a central control center, translating temperature sensing input into response. However, for practical purposes, it can be thought of as thermostat-like and having a “set point” toward which body responses will be directed [18]. The “set point” may be altered, for example, in febrile illness or by drugs such as aspirin. In the circumstance of overheating, peripheral vasodilatation is the initial response, resulting in increased heat loss through conduction, convection, and radiation. Sweating follows, and vaporization is aided by the already increased skin temperature and air movement.

Sweat glands are eccrine glands that respond to parasympathetic and sympathetic stimulation. Sweating and sweat rate are controlled by the hypothalamus. Sweat is hypotonic, being 99.5% water by weight with 0.41% sodium content [18]. Sweat glands are distributed over palms, soles, head, extremities and trunk with an average density of 100-200/cm<sup>2</sup> [31]. Sweating can cause a significant loss of water and sodium. Although, sweat loss rates of 1 liter/hr are sustainable without significant dehydration [18]. Intensive sweat can result in dehydration and hyponatremia. In a hot occupational environment, as much as 6 liters per day can be lost from sweat.

Acclimatization is the process of change in thermoregulation involving sweating, skin blood flow, and thermoregulatory set point. Acclimatization after training in a hot environment can result in improved ability to handle thermal stress within 7–10 days. Sweat glands become more efficient, losing less salt per milliliter of perspiration, and peak output increases. Also, plasma volume increases and vasodilatation and sweating begin at lower temperatures [3, 10, 18, 35].

**Susceptible Populations.** Some people are at increased risk from heat stress [32]. Those at extremes of age, for a variety of reasons, may be susceptible. Infants with smaller body mass and immature physiology will experience more rapid rise in body temperature than a healthy adult in the same hot environment. Also, they may not be able to respond by removing themselves from the stress environment or pursuing other behavioral defenses.

**Table 1.** Modification of sports activity using wet bulb globe temperature.

Index (°F)	Limitation
<50	Low risk for hyperthermia but possible risk for hypothermia
<65	Low risk for heat illness
65–73	Moderate risk toward end of workout
73–82	Those at high risk for heat injury should not continue to train; practice in shorts and T-shirts during the first week of training
82–84	Care should be taken by all athletes to maintain adequate Hydration
85–87.9	Unacclimated persons should stop training; all outdoor drills in heavy uniforms should be canceled
88–89.9	Acclimated athletes should exercise caution and continue workouts only at a reduced intensity; light clothing only
90 or above	Stop all training

Reprinted with permission: Hubbard R, Gaffin S, Squire: Heat-Related Illnesses. In: Auerbach P (Ed): *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. McGraw-Hill, St. Louis, Missouri 1995.

The elderly may have similar behavior response problems. These factors may also apply to individuals with chronic disease, those who are socially isolated [46]. Drugs such as cocaine, methamphetamines, and psychostimulants can increase the risk of hyperthermia. Persons taking some prescription medications which interfere with heat loss mechanisms, for example anticholinergic medications and some cardiac drugs, are more susceptible to heat illnesses.

A subgroup of individuals at risk are those who pursue heavy exercise or exertion in warm environments. The highly motivated athlete may be at increased risk, especially if not acclimated. Individuals who work under conditions that inhibit heat shedding, such as wearing sweat impermeable clothing, or in very humid circumstances are also susceptible. Interestingly, risk of heat illness in at least one study appeared to be increased by environmental exposure the previous day, suggesting that heat stress can predispose patients to developing illness for at least a day [18].

## WET BULB GLOBE TEMPERATURE

Environmental temperature, humidity and solar radiation are factors contributing to heat stress. The wet bulb globe temperature (WBGT) is an index of heat stress that incorporates these 3 factors [18]. The WBGT can be calculated or obtained from heat stress monitors and is used to help prevent heat stress related illness (Tab. 1).

Clothing, physical conditioning, acclimation to the environment, and state of hydration all contribute to preventing heat stress related illness. Activity should be based on the WBGT. Acclimation to heat stress environments should occur with moderate intensity over 8-10 days. Children require 10–14 days for similar conditions [18].

**Table 2.** Comparison of classic and exertional heatstroke.

	Classic	Exertional
Age group	Elderly	Med (15-45 yr)
Health status	Chronically ill	Healthy
Concurrent activity	Sedentary	Strenuous exercise
Drug use	Diuretics, antidepressants, antihypertensives, anticholinergics, antipsychotics	Usually none
Sweating	May be absent	Usually present
Lactic acidosis	Usually absent; poor prognosis if present	Common
Hyperkalemia	Usually absent	Often present
Hypocalcemia	Uncommon	Frequent
Hypoglycemia	Uncommon	Common
Creatine phosphokinase/aldolase	Mildly elevated	Markedly elevated
Rhabdomyolysis	Unusual	Frequently severe
Hyperuricemia	Mild	Severe
Acute renal failure	<5% of patients	25% to 30% of patients
Disseminated intravascular Coagulation	Mild	Marked; poor prognosis
Mechanism	Poor dissipation of environmental heat	Excessive endogenous heat production and overwhelming of heat loss mechanisms

Reprinted with permission: Hubbard R, Gaffin S, Squire: Heat-Related Illnesses. In: Auerbach P (Ed): *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. McGraw-Hill, St. Louis, Missouri 1995.

Adequate hydration is essential if heat stress environments are to be encountered.

### HEAT STRESS SYNDROMES

**Heat Stroke.** The classical clinical description of heat stroke is the triad of hyperpyrexia, central nervous system (CNS) dysfunction, and anhydrosis. Anhydrosis is not a diagnostic requirement, however, and may appear as a later finding when volume depletion is severe. Classical heat stroke, as opposed to exertional, will often develop over days of heat stress (Tab. 2).

Heat stroke is part of the differential in any patient who presents with high core body temperature and altered mental status or CNS symptoms. As there is a high potential for permanent organ damage or death, immediate intervention should follow when this diagnosis is suspected.

The CNS manifestations are multiple, including syncope, agitation or combative behavior, hallucination, delirium or coma. Physical examination findings include ataxia, abnormal plantar reflexes, posturing, hemiplegia and seizures. Pupils may be constricted, and oculogyric crisis can occur [28, 48].

Multiple organ systems damage may occur, complicating the course. Cerebral edema with petechial hemorrhage and neuronal degeneration have been noted at autopsy. In severe cases, hypotension and shock occur. Hyperventilation is common, and alkalosis producing tetany can occur. Other potential respiratory complications are pulmonary edema, ARDS and aspiration in the comatose or seizing

patient. Disseminated intravascular coagulopathy (DIC) can develop, possibly resulting from the release of thromboplastic substances from endothelium damaged by high temperature and hypotension [18, 48].

Hyponatremia develops when sweat loss exceeds 5 liters/day. Hyperthermia leads to hypocalcemia due to shifting of calcium to injured skeletal muscles [18]. However, if acute renal failure intervenes or rhabdomyolysis occurs, hypercalcemia can result. Potassium concentration in sweat is 4–5 mmol/liter. Magnesium concentration in sweat varies from 0.02–5 mmol/L [18]. Up to 1% of total body magnesium can be lost with extreme exercise. Hypomagnesemia can be seen in heat stroke but probably results from magnesium shifts into muscles and red blood cells as opposed to losses in sweat.

Hypophosphatemia is often seen in exertional heat stroke which may be secondary to excessive renal clearance combined with increased muscle uptake [18].

Heat stroke results in increased cortisol and catecholamines which can cause elevated glucose through breakdown of liver glycogen stores. However, some patients with heat stroke may be hypoglycemic.

The combination of hypotension, DIC, myoglobinuria, and direct thermal damage jeopardizes the kidneys in particular. Up to 25% of heatstroke patients will suffer acute renal failure. Hepatocellular injury can also result, with elevation of liver function enzymes, cholestasis, and hepatocellular necrosis [18, 30].

Nausea and vomiting are frequently symptoms of heat stroke, and contribute to dehydration. In this setting,

**Table 3.** Signs and Symptoms of Salt and Water Depletion Heat Exhaustion.

Signs and symptoms	Salt Depletion Heat Exhaustion	Water Depletion Heat Exhaustion	Dilutional Hyponatremia
Recent weight gain	No	No	Yes
Thirst	Not prominent	Yes	Sometimes
Muscle cramps	In most cases	No	Sometimes
Nausea	Yes	Yes	Usually
Vomiting	In most cases	No	Usually
Muscle fatigue or weakness	Yes	Yes	No
Loss of skin turgor	Yes	Yes	No
Mental dullness, apathy	Yes	Yes	Yes
Orthostatic rise in pulse rate	Yes	Yes	No
Tachycardia	Yes	Yes	No
Dry mucous membranes	Yes	Yes	No
Increased rectal temperature	Yes	In most cases	No
Urine Na <sup>+</sup> /Cl <sup>-</sup>	Negligible	Normal	Low
Plasma Na <sup>+</sup> /Cl <sup>-</sup>	Below average	Above average	Below average

Reprinted with permission: Hubbard R, Gaffin S, Squire: Heat-Related Illnesses. In: Auerbach P (Ed): *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. McGraw-Hill, St. Louis, Missouri 1995.

especially with associated renal impairment, electrolyte and acid-base abnormalities may emerge. With the hyperventilation often seen early in heat illness, a respiratory alkalosis may result. As hypotension worsens and anaerobic metabolism becomes important, rising serum lactate will produce a metabolic acidosis. Hyponatremia from water loss is possible, but if dietary sodium intake is inadequate and large amounts of sodium are lost in sweating, serum sodium may be normal or even low. Potassium also may be either low, as potassium is lost in perspiration, or high, with exertional heat stroke victims possibly at greater risk for hyperkalemia. This greater risk may be a result of intracellular potassium release from contracting muscle. Other mechanisms for hyperkalemia include impaired renal function, release from damaged cells, and failure of the sodium-potassium pump as ATP availability falls in the face of hypotension and cellular hypoxia. The effect of heat stroke on serum potassium concentrations therefore is variable. While classic heatstroke patients appear to have a normal serum potassium concentration, exertional heat stroke victims often manifest hypokalemia and rhabdomyolysis [18].

Heat stroke patients may be hypovolemic with normal serum potassium or hypokalemia. When the hypovolemia is corrected, the serum potassium tends to fall. Finally, hypocalcemia, hypomagnesemia, and hypophosphatemia may also be present [18, 48, 51].

Some of the manifestations of heat stroke resemble septic shock, and may result from a similar mechanism. As intestinal hypoperfusion develops, permeability increases.

As a part of normal gram negative gastrointestinal bacterial activity, large amounts of cell wall lipopolysaccharide are produced. Theoretically, increased absorption of this endotoxin will activate macrophages, resulting in increased secretion of cytokines, including interleukins and Tumor Necrosis Factor. These substances mediate responses which worsen the symptoms of heat stroke, including fever, malaise, and vasodilation with hypotension. Whether some form of anti-lipopolysaccharide therapy will help patients with heat stroke remains to be clarified [7, 8, 18].

In patients presenting with fever and altered mental status, heat stroke is a diagnosis of exclusion. Other potentially life-threatening conditions which present similarly include meningitis, stimulant overdose such as cocaine, and malignant hyperthermia.

**Heat Exhaustion.** Heat exhaustion is used to describe patients suffering any of a wide variety of symptoms from exposure to heat stress (Tab. 3). Usually the syndrome develops over a several day period of exposure, as water and/or electrolyte loss occur. In contrast to heat stroke mental status is normal, although heat exhaustion syndrome may be a point along the continuum on the way to heat stroke.

Symptoms of heat exhaustion include headache, dizziness, lightheadedness or even syncope. Non-specific complaints such as fatigue, malaise, nausea, vomiting and myalgias are common. Examination can reveal tachycardia, orthostatic hypotension, diaphoresis or tachypnea. At the time of examination, patients' body temperatures may be elevated or normal.

This syndrome is thought to result from depletion of body resources in attempting to maintain normal temperature in the face of heat stress. Serum electrolyte assays and complete blood counts may be useful to assist in the choice of replenishment. At the least, values will generally be consistent with water loss, for example increased hematocrit. Electrolyte abnormalities will depend on the circumstances of loss, and content of patient intake during the stress period. Sodium and chloride levels may be elevated from water loss, low from hypotonic fluid replacement, or normal. Potassium and other electrolytes are similarly variable.

Heat exhaustion has three clinical presentations:

- Classic heat exhaustion
- Salt-depletion exhaustion
- Water depletion exhaustion

Salt depletion heat exhaustion is associated with hyponatremia and hypochloremia which require 3–5 days to develop. Muscle cramps, nausea and vomiting are more prominent symptoms, while thirst is less prominent [18].

Water depletion heat exhaustion is accompanied by thirst, nausea, muscle weakness, mental dullness, orthostatic vital sign changes and increases in serum sodium and potassium due to the relative water loss.

Classic heat exhaustion is a result of cardiovascular stress of maintaining normothermia [18]. Symptoms include headache, dizziness, fatigue, irritability, nausea, vomiting, muscle cramps, anxiety, piloerection, chills and heat sensation in upper torso and head [18]. Tachycardia, hyperventilation, hypotension and syncope are frequent. Internal temperature ranges from 39–40°C. Profuse sweating also occurs with classic heat exhaustion.

**Heat Cramps and Heat Tetany.** Heat cramps may occur following heavy muscular exertion in a hot environment, with heavy sweat production and associated intake of hypotonic replacement. Muscular spasm and cramping, usually of the muscles that have been in use, may follow. Lower extremity and shoulder muscle groups are commonly affected. These symptoms are the result of electrolyte deficiencies at a cellular level. Hyponatremia and other electrolyte abnormalities may represent an absolute deficiency, or in part a relative deficiency created by fluid replacement with hypotonic solution [31]. Symptoms usually improve quickly with appropriate fluids. Most patients can be replenished orally with commercial electrolyte solutions, or a 0.1–0.2% salt solution. Severe cases, or cases with nausea and vomiting may benefit from replacement with intravenous isotonic saline, or even hypertonic saline in small amounts.

Heat tetany is a hyperventilation syndrome that can result from a short period of excessive heat stress. Symptoms are typical for hyperventilation, with carpopedal spasm and paresthesias. Arterial blood gases may reveal a respiratory alkalosis. Symptoms will usually resolve with the patients' removal from the heat stress and slowing of their respirations.

**Heat Syncope.** The combination of intravascular volume depletion, peripheral vasodilation, and decreased vasomotor tone can occur with heat stress syndromes and lead to syncope. The greatest difficulty facing the clinician in these patients will be the exclusion of other more serious medical problems, such as cardiac dysrhythmias and neurologic disorders. Heat syncope alone will respond in most cases to rehydration, after removal from the stress environment.

**Prickly Heat and Heat Edema.** Heat edema, which affects mostly the elderly, is the result of peripheral vasodilation and extravascular fluid pooling. The hands and feet are usually affected, and the edema may be pitting. This condition will usually resolve over days to a few weeks; elevation and/or compression of the affected limbs may hasten recovery. Since this condition does not represent a relative intravascular overload, diuretics are ineffective. As always, other medical causes of edema should be considered.

If sweat pores become blocked by macerated corneal skin material, an erythematous, pruritic maculopapular rash known as heat rash or prickly heat may result. This condition, which is also sometimes referred to as lichen tropicus, is the result of rupture of the sweat ducts, creating superficial vesicles which are intensely pruritic. The condition may be treated or prevented by the wearing of clean, loose-fitting and lightweight clothing. Itching should respond to antihistamines, and secondary infection, if it remains superficial, can be treated with a chlorhexidine cream or lotion. If the condition is allowed to continue, vesicles may rupture deeper into the skin, producing a profound stage of prickly heat that can progress to a chronic dermatitis. Desquamation of the skin to relieve plugging may be necessary using 1% salicylic acid 3 times a day. Also in this stage, secondary infection with *Staphylococcus aureus* is common, and may require oral anti-staphylococcal antibiotic use.

## TREATMENT FOR HEAT STROKE AND HEAT EXHAUSTION

Treatment is directed at immediate lowering of core body temperature by the most rapid and expedient available methods. Other therapies are guided by clinical and laboratory assessment.

**Cooling Measures.** In the true emergency of heat stroke, rapid reduction of the core body temperature is crucial. The ideal is to cool the patient rapidly with minimal side effects.

The most effective modality in lowering core temperature is immersion in a cold or iced water bath [11, 18]. In this method, the patient is undressed and placed in a bath of sufficient depth to cover the trunk and extremities. This will in most cases lower core temperature below 39°C in less than an hour [11]. Cold water may result in similar rates of cooling. It is difficult

to maintain monitoring and perform therapeutic interventions on patients in an immersion bath. The arguments are made that immersion will produce shivering and shunting of blood centrally, with associated rise in core temperature. However, this is contradicted somewhat in the investigative literature [18, 38], and shivering might be controlled with intravenous chlorpromazine at 25–50 mg [24].

Evaporative cooling has been argued as the modality of choice, as it is simpler than immersion, may be nearly as effective, and is non-invasive [29, 51, 53]. In this method, the undressed patient is sprayed with cool water on all exposed surfaces, while warm air is blown over them. Additional exposure can be achieved by suspending the patient in a net sling [29]. Monitoring equipment can remain attached to the patient, and interventional access by health care personnel is essentially unchanged.

Additional cooling can be accomplished through the use of ice packs placed at the groin and axilla. Extremity placement is undesirable as peripheral vasoconstriction may result, and reduce the effectiveness not only of the ice packs, but also of evaporative cooling on the extremities.

More invasive methods include iced gastric lavage, peritoneal lavage, and intubation with cooled, humidified air. In addition to being more invasive, these methods appear less effective in lowering body temperature than immersion or evaporative cooling [18, 51]. If these methods are used, common sense caveats should be observed. Gastric lavage should not be performed on unintubated patients with altered mental status. Peritoneal lavage in patients with prior abdominal surgery, or pregnant patients should be administered with caution if at all. Also, one of the above more effective methods should be added or used simultaneously, unless there is an immediate favorable response.

The use of antipyretic medications is generally not of beneficial in heat illness. This is because drugs such as acetaminophen and aspirin act on fever by reducing the thermoregulatory “set point” back toward normal. In heat illness, the set point is not changed, but rather there is a failure or overwhelming of compensatory cooling mechanisms in the face of heat stress [18, 51].

**Rehydration.** Heat stroke and cases of heat exhaustion, heat syncope, or heat cramps generally require intravenous fluid replacement [18]. Normal saline or Ringer’s lactate are initial fluids of choice, with the addition of other electrolyte replacements such as potassium guided by laboratory testing. Care should be taken, especially in elderly patients, that aggressive fluid replacement does not precipitate pulmonary edema. Monitor such indicators as urine output, serum values, and breath sounds to gauge resuscitation.

Most heat illness patients can be rehydrated orally, with water, salt solutions, or electrolyte solutions as appropriate. Patients with vomiting may need initial anti-emetic treatment before they can be given oral replacements.

**Prevention.** As mentioned above, most if not all heat is preventable. Modifications of the environment with proper shelter, air conditioning, and proper clothing can rectify or at least improve the effects of having to exist in a heat stressed environment [32, 46].

Where exposure to heat stress is necessary or unavoidable, care should be taken to ensure adequate replacement of fluid and electrolytes. For most persons dietary intake of electrolytes is adequate, whereas free water loss is the more significant problem. Adequate hydration before beginning activity or entering a heat stress environment should be obvious. Active individuals should adhere to a regimen of 8–12 ounces intake every 20–30 minutes regardless of thirst. Thirst is not a sufficiently sensitive sentinel of dehydration. Alternatively, urine output and color, or body weights can be monitored [18].

Prophylactic electrolyte replacement with special glucose-electrolyte solutions is usually not necessary and provides no clearly proven benefit beyond taste [18, 25, 58]. The intake of salt tablets as a preventative measure is unnecessary, and may produce gastric irritation. If an individual chooses to use a glucose-electrolyte solution for fluid maintenance, it is important to be aware that solute concentrations that are too high may inhibit absorption or even draw water out into the gastrointestinal tract. Glucose polymer solutions can provide significant carbohydrate replacement in addition to water, without the high osmolality of a straight glucose solution with the same caloric value. The inclusion of sodium salt and other electrolytes might theoretically enhance absorption, but have not been unequivocally shown superior to water in this regard [37, 56].

## COLD STRESS

Cold stress includes hypothermia and frostbite. Hypothermia can result from cold water immersion, cold water submersion and environmental weather exposure. Hypothermia is defined as a core temperature less than 35°C as measured by rectal, tympanic or esophageal probe. Moderate hypothermia is defined as a core temperature less than 32°C, and severe as being less than 26°C. Accidental hypothermia is further defined as a decrease in temperature without a lesion of the thermal regulatory mechanism in the central nervous system [17]. Moderate and severe hypothermia causes nearly 600 deaths each year in the United States [4, 13, 23].

Exposure to a cold environment is the most important factor leading to hypothermia. Protective clothing characteristics are important to protect against hypothermia, especially in an occupational arena. Ethanol ingestion raises the risk of hypothermia by preventing vasoconstriction, impairing the shivering mechanism, causing hypothalamic dysfunction, and promoting high-risk behaviors [45, 55, 57]. Although hypothermia frequently occurs with concomitant ethanol ingestion, mortality does not appear to be affected in patients with core temperatures above

**Table 4.** TLVs for Heat Exposure [Values are given in °C and (°F) WBGT].

Hourly Activity	Work Rates		
	Light	Moderate	Heavy
100% Work	30.0 (86.0)	26.5 (80.0)	25.0 (77.0)
75% Work; 25% Rest	30.5 (87.0)	28.0 (82.5)	26.0 (79.0)
50% Work; 50% Rest	31.5 (89.0)	29.5 (85.0)	28.0 (82.5)
25% Work; 75% Rest	32.0 (89.5)	31.0 (88.0)	30.0 (86.0)

Reprinted with permission: *Thermal Stress in 1997 TLVs and BEIs: Threshold Limit Values for Chemical Substances and Physical Agents, Biological Exposure Indices*, ACGIH Worldwide, 1997.

26°C. Signs and symptoms of hypothermia are presented in Table 4.

The very young, very old, and chronically ill are at higher risk of hypothermia in a susceptible environment. Infants have greater heat loss and more rapid core cooling secondary to their larger body surface area to total mass ratio compared to average-sized adults. The elderly have a lower metabolic rate and may have a diminished ability to perceive ambient temperature [10, 26]. Illness and disease in the elderly, such as hypothyroidism, hypoadrenalism, diabetes mellitus and cardiac disease are risk factors for hypothermia and independent of their effect of promoting exposure [45].

Outdoor accidents and injury are associated with a risk of hypothermia. Injured hypothermic patients also have an increased risk of disseminated intravascular coagulation. Furthermore, hemorrhagic shock promotes worsening hypothermia [6, 34].

**Weather and Exposure Hypothermia.** Outdoor and weather related episodes of hypothermia are probably the most common causes of death outdoors for hikers, climbers, campers, hunters and those lost outdoors. Most cases of hypothermia do not occur in extreme cold environments. The combination of cold air, wind and wet conditions such as snow or rain has a profound effect on heat loss from a body, and increases the risk of severe hypothermia and death [50]. Studies demonstrate that windy, wet conditions decrease clothing insulation by 90% and increased metabolism by 50% compared to dry, non-windy conditions [41, 54]. Drastic declines in strength, comfort, performance and dexterity can occur in subjects exposed to windy and wet conditions if they are not properly protected. Such cold stress responses are associated with an accelerated core cooling rate, and a 40–50% decline in strength and dexterity in studies. Thus, outdoor windy and wet conditions create an increased risk for hypothermia which can accelerate heat loss, sap strength, performance and dexterity causing inability to escape the elements.

Exertional fatigue, sleep deprivation and a negative energy balance also increase susceptibility to hypothermia

[49, 59]. Such conditions blunt the person's thermogenic response to cold weather.

**Cold Water Immersion and Submersion.** Cold water varies in temperature from glacial melt lakes and rivers and arctic waters to warmer lakes and pools. Water temperatures near 0°C (32°F) are not uncommon in some high country rivers and alpine lakes. Cold water submersion refers to near-drowning.

Cold water immersion is subdivided into 3 phases [19]:

1. **Cold Shock:** The initial shock-effect of cold water on a person's face, head or body causes a gasp reflex, acute peripheral vasoconstriction, hyperventilation and tachycardia. These reflexes can produce sudden death, syncope, vagal nerve response with bradycardia and cardiac arrest, convulsions, and ventricular ectopy [19, 33, 36]. The dive reflex is activated by cold water on the face and causes breath holding, peripheral vasoconstriction, bradycardia, and increased arterial blood pressure.

2. **Rapid Cooling of Peripheral Tissues:** The cold shock response can prevent a person from rapidly responding to an initial cold water immersion. Once the cold shock has passed, if the person cannot escape, rapid peripheral cooling occurs with a consequent loss of motor performance, loss of strength, and loss of coordination, making survival and rescue efforts difficult [19, 33, 36]. Loss of grip strength and movement can result in drowning.

3. **Loss of Core Heat:** Individuals surviving the cold shock and peripheral cooling phases of cold water immersion may experience hypothermia from a rapid loss of core temperature due to cold water. Survival time depends on factors of body composition, thermoregulatory response, clothing and insulation, water temperature, water condition (river, lake, sea).

In moderately cold water (18–26°C) immersion core cooling occurs at the same rate in most individuals no matter what their body composition [21]. But if water is extremely cold (8°C), loss of core temperature is attenuated by greater amounts of fat and muscle. Exercise and struggling in cold water will increase surface heat loss and result in faster core cooling [43]. Treading water thus can increase cooling rate in spite of an increased metabolic heat production.

The lowest water temperature in which a person can maintain normal core temperature by generating heat through muscular activity is estimated to be 25°C [43]. A combination of shivering thermogenesis and core insulation by peripheral vasoconstriction may allow maintenance of a steady core temperature even this it is below the normal core temperature, thus preventing death from severe hypothermia [19]. In extremely cold water (8–18°C), core temperature will continue to fall despite shivering and peripheral vasoconstriction, and this core temperature decline will accelerate when shivering induced thermogenesis stops due to severe hypothermia as the core body temperature falls to 30°C and below.

Cold water near-drowning or cold water submersion can occur when the head is submerged. The dive reflex is

activated when the face contacts cold water. This causes breath holding, bradycardia, peripheral vasoconstriction and an increase in mean arterial blood pressure [19]. Whether the diving reflex in humans can actually protect against drowning is unsure. Research has confirmed bradycardia produced by cold water immersion is proportional to water temperature [44].

**Cold Stress Pathophysiology.** Heat is lost by conduction, radiation, convection, and evaporation. Heat loss may be compensated for by the thermogenic shivering response and increased muscular activity.

Cooling primarily decreases metabolism and gradually decreases and inhibits neural transmission and muscular control. In the conscious individual, initial physiologic responses to cooling are shivering as a thermogenic response and increased metabolism, increased ventilation, increased heart rate, increased cardiac output and increased mean arterial pressure. As core temperature falls, the effects of hypothermia occur. Non-cognitive thermoregulation is coordinated by the autonomic and neuro-endocrine systems [42]. The body can be thought of as possessing 3 thermoregulatory zones: superficial, intermediate, and core. The superficial zone is composed of the skin, the subcutaneous tissues, and their thermoreceptors. The temperature of this zone varies with blood flow and the temperature of the environment. Cooling temperatures typically result in a reflex vasoconstriction within this zone until the tissue reaches approximately 10°C. The reflexes are then lost, resulting in paralysis of the vasomotor response and vasodilation ensues [27]. The intermediate zone consists of the skeletal muscle mass. Heat is generated by muscle activity and can be productive or nonproductive with regard to its affect on core temperature. An example of nonproductive muscle activity would be when the core temperature of a subject decreases even after removal from a cold environment secondary to shivering producing peripheral vasodilation. The core temperature then experiences an “after-drop” as cold pooled blood in the superficial zone is shifted centrally. This phenomena can also occur with the application of external heat to severely hypothermic patients.

On a cellular level, hypothermia produces an overall reduction in enzymatic activity, the degree of which varies from organ to organ. Oxygen consumption decreases by a factor of 0.5 for every 10°C change from normal. Decreased metabolic activity resulting in less CO<sub>2</sub> production somewhat counters the effects of decreased respiratory drive. Evaluation of blood gases in hypothermia has therefore been controversial. As the temperature in the superficial zone decreases, peripheral vasoconstriction results in a local metabolic acidosis (shifts oxygen dissociation curve to the right). With further temperature decreases, however, the oxygen dissociation curve is shifted to the left, decreasing the release of bound oxygen from the hemoglobin molecule. The progressive changes in pH do not completely compensate for the changes

induced independently by temperature. Other confusing variables include lactic acidosis from poor perfusion, partial resuscitation efforts including bicarbonate administration and hyperventilation. It is therefore appropriate to utilize the “uncorrected” blood gas values during resuscitation. However, excessive hyperventilation or bicarbonate administration will shift the curve even further to the left.

The initial response of the cardiovascular system is an elevation of heart rate (HR), cardiac output (CO), and mean arterial pressure (MAP). These are secondary to elevated catecholamines, peripheral vasoconstriction, and the resultant increase in core blood volume. As cardiac function decreases and vasomotor paralysis ensues during severe hypothermia, heart rate and cardiac output will decrease. The electrocardiogram will demonstrate a generalized conduction slowing and a pathognomonic J wave (Osborn wave) may be seen as the temperature falls below 33°C [2, 12, 20, 39]. The J wave has also been observed in sepsis and central nervous system lesions [12]. The initial tachycardia evolves into bradycardia with lengthening of the PR, QRS, and QTC intervals. Atrial and ventricular fibrillation may also occur. Successful cardioversion from ventricular fibrillation becomes increasingly difficult as hypothermia worsens. Anti-dysrhythmia medication also appears to become ineffective [15, 20].

Early respiratory changes include central stimulation of rate. As the core temperature drops, however, there is central and reflex depression with decreased rate and tidal volume. Anatomic and physiologic dead space increases and ventilation becomes progressively worse. Cold induced bronchorrhea compounds the loss of cough and gag reflexes, thus making early airway management critical.

Cerebral blood flow will fall approximately 6–7% for each 1°C decrease in core temperature. At 34°C patients usually experience amnesia and below 30°C most victims are confused and disoriented. Pupil reactivity and deep tendon reflexes usually disappear by 25°C [12, 20, 26, 42].

Renal function decreases as cardiac output drops. The initial cold diuresis, secondary to “third-spacing” and a defect in renal concentrating abilities, may give the clinician false security in the patient’s volume status. Electrolyte disturbances including hyponatremia and hyperkalemia, are common. Large losses in intravascular volume can occur while the hematocrit increases. Extravascular fluid shifts occur with diuresis and decrease in perfusion [14]. The clinical progression of hypothermia is as follows:

- 37.6°C (98.6°F) - “Normal” oral temperature;
- 35–32°C - Mild hypothermia; Thermoregulatory mechanisms operate fully; Ataxia, shivering, dysarthria, apathy, difficulty concentrating;
- 32–38°C - Moderate hypothermia;
- 35°C (95°F) - Maximum hypothermia;
- 32°C (89.6°F) - Consciousness clouded; blood pressure; becomes difficult to obtain; pupils dilated but react to light; shivering ceases as thermoregulation and thermogenesis cease;

- 30°C (86°F) - Progressive loss of consciousness; muscular rigidity increases; pulse and blood pressure difficult to obtain; respiratory rate decreases;
- 28°C (82.4°F) - Severe hypothermia; Atrial fibrillation and ventricular fibrillation possible with myocardial irritability;
- 27°C (80.6°F) - Voluntary motion ceases; pupils nonreactive to light; deep tendon and superficial reflexes absent;
- 25°C (77°F) - Ventricular fibrillation may occur spontaneously;
- 24°C (75.2°F) - Pulmonary edema;
- 22°C (71.6°F) - Maximum risk of ventricular fibrillation;
- 20°C (68°F) - Cardiac standstill.

**Rewarming therapy for hypothermia.** Rewarming techniques are categorized as either passive, active external, or active core rewarming. Each technique has advantages and disadvantages.

Passive rewarming is simple endogenous heat production by the patient without external application of heat. For this to be effective, the patient must be only mildly hypothermic, capable of sensing and responding to the hypothermic state, and tolerant of the relative delay in reverting to normothermia.

Active external rewarming involves the application of an external heat source directly to the body. In mildly hypothermic patients this technique is usually effective in raising body temperature. The potential danger results from the rapid peripheral vasodilation causing the core temperature and pH to drop as the peripheral blood is shunted toward the core. After-drop may lead to cardiac dysrhythmias. Active external rewarming should not be applied to patients with core temperatures less than 30°C.

Active core rewarming is the approach of choice in the moderate to severely hypothermic patient. Airway rewarming involves the use of warmed, humidified air or oxygen through either a face mask or endotracheal tube. This technique provides modest heat gains but is useful as it is readily available and minimizes heat loss from the respiratory tract. Intravenous fluids should be warmed to 37°C and is recommended in all patients mild to severe. Warm saline lavage, gastric or colonic, is practically simple. Upper GI tract lavage patients should obviously have airway protection before institution.

Peritoneal irrigation utilizes potassium free dialysate warmed to between 40.5–42.5°C. The use of at least 2 catheters, 1 for inflow and 1 for outflow, has been advocated. Pleural irrigation via thoracostomy tubes has been studied and carries the potential advantage of placing warm fluid in direct proximity to the heart. Obvious care should be exercised in avoiding the complications of chest tube insertion, tension hydrothorax, and irritating the heart with the thoracostomy tube. Cardiopulmonary bypass, although most invasive, has the theoretic advantage of rapid rewarming, equilibrium of fluid composition, oxygenation, and hemodynamic support during resuscitation.

**Table 5.** Occupational Heat Stress Controls.

1. General Controls	
•	Appropriate training for occupational environment
•	Understanding work demands
•	Recognizing heat-related disorders
•	First aid for heat-related disorders
•	Heat stress hygiene practices and heat stress alert programs
•	Company policy regarding heat stress
•	Medical surveillance and risk evaluation
2. Specific Controls	
•	Engineering controls:
	Reduce work demand
	Reduce air temperature
	Reduce air humidity
	Change clothing
	Reduce radiant heat
	Increase air movement
•	Administrative controls:
	Change way work is performed
	Acclimation
	Change pace of work
	Work share
	Scheduling of work
	Personal monitoring
•	Personal protection:
	Circulating air systems
	Circulating water systems
	Ice garments
	Reflective clothing

More recently, continuous arteriovenous rewarming which utilizes a counter current heat exchanger has been reported. This technique would, however, not be useful in hypotensive or cardiac arrest patients [12, 13, 20, 26, 45].

Early recognition and institution of therapy is paramount to minimize morbidity and maximize survival of hypothermic patients. Prehospital and emergency personnel must maintain a high index of suspicion [20]. The sometimes forgotten “fourth vital sign” should be obtained in all patients. Emergency departments should have access to thermometers accurately measuring temperatures below 30°C. Appropriate rewarming equipment should also be readily accessible to emergency departments.

Victims of hypothermia should be removed rapidly from the cold environment and transported to a facility where definitive treatment can be provided. In addition to an accurate temperature measurement, the evaluation should also include electrolyte analysis, ABG, and appropriate x-rays would include a chest x-ray and those necessary to rule out traumatic injuries. A 12-lead EKG and continuous monitoring should also occur. Rewarming techniques, in addition to warmed inspired oxygen and intravenous fluids, should be chosen according to the severity of illness. In the setting of severe hypothermia and cardiac arrest, aggressive rewarming should be continued until the patient is hemodynamically stable or the core temperature has risen to greater than 32°C, signaling the futility of further resuscitation.

**Table 6.** Assessment of work load. Average values of metabolic rate during different activities.

A. Body position and movement		kcal/min	
Sitting		0.3	
Standing		0.6	
Walking		2.0-3.0	
Walking up hill		add 0.8 per meter (yard) rise	
B. Type of Work		Average kcal/min	Range kcal/min
Hand work	light	0.4	0.2-1.2
	heavy	0.9	
Work with one arm	light	1.0	0.7-2.5
	heavy	1.7	
Work with both arms	light	1.5	1.0-2.5
	heavy	2.5	
Work with body	light	3.5	2.5-15.0
	moderate	5.0	
	heavy	7.0	
	very heavy	9.0	

Reprinted with permission: *Thermal Stress in 1997 TLVs and BEIs: Threshold Limit Values for Chemical Substances and Physical Agents, Biological Exposure Indices*. ACGIH Worldwide, 1997.

## OCCUPATIONAL THERMAL STRESS REGULATIONS

The National Institute for Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) recommend specific controls for heat stress based on work practices, personal protection and types of heat loads. ACGIH has threshold limit values (TLVs) for both cold stress and heat stress. Their heat stress TLVs refer to heat stress conditions under which it is believed that nearly all workers may be repeatedly exposed without adverse effects. The ACGIH TLVs assume acclimatized workers that are fully clothed and who receive adequate hydration. Such workers should be able to function effectively under their work condition without exceeding a core temperature of 38°C (100.4°F). When there is a requirement for additional personal protective clothing, a correction to the Wet Bulb Globe Temperature (WBGT) must be applied [1] (Tab. 4).

Heat stress and heat strain must be assessed in evaluating worker safety and health. Heat stress is the net heat load on the body with contributions from heat production and environmental factors including relative humidity, clothing, radiant heat and air movement. Heat strain is the net physiological load resulting from heat stress [1]. Control of heat stress and heat strain involves understanding the causes and then taking action to control those causes based on constraints of the occupational environment (Tab. 5).

**Table 7.** Guidelines for heat exposure limits.

Always monitor signs and symptoms of heat-stressed workers. When WBGT-TLV criteria are exceeded or water vapor impermeable clothing is worn, discontinue any environmentally induced or activity-induced heat stress for a person when:

1. Sustained heart rate is greater than 160 beats per minute for those under 35 years of age; 140 for 35 years or older, or
2. Deep body temperature is greater than 38°C (100°F), or
3. Blood pressure falls more than 40 torr in about 3.5 minutes, or
4. There are complaints of sudden and severe fatigue, nausea, dizziness, lightheadedness, or fainting, or
5. There are periods of inexplicable irritability, malaise, or flu-like symptoms, or
6. Sweating stops and the skin becomes hot and dry, or
7. Daily urinary sodium ion excretion is less than 50 mmoles.

Reprinted with permission: *Thermal Stress in 1997 TLVs and BEIs: Threshold Limit Values for Chemical Substances and Physical Agents, Biological Exposure Indices*. ACGIH Worldwide, 1997.

**Table 8.** TLV WBGT Correction Factors in °C for Clothing.

Clothing Type	Clo Value	WBGT Correction
Summer work uniform	0.6	0
Cotton coveralls	1.0	-2
Winter work uniform	1.4	-4
Water barrier, permeable	1.2	-6

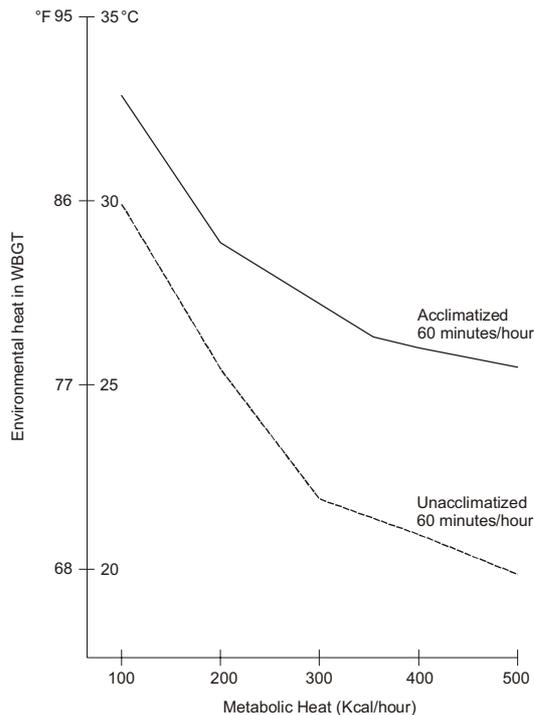
Reprinted with permission: *Thermal Stress in 1997 TLVs and BEIs: Threshold Limit Values for Chemical Substances and Physical Agents, Biological Exposure Indices*. ACGIH Worldwide, 1997.

**Environmental Evaluation.** The goal of an occupational or industrial heat stress program is to assure that core body temperature does not exceed 38°C (100.4°F) by using a combination of general and specific controls. For general thermal comfort in office environments, the American Society for Heating, Refrigeration and Air Conditioning Engineers (ASHRAE) has developed comfort zone standards (ASHRAE 55-1981). To assure that heat stress is not becoming a problem, the environment and the worker must be evaluated:

- Environmental evaluation
- Measuring WBGT index
- Work load evaluation
  - Light - up to 200 kcal/hr
  - Moderate - 200-350 kcal/hr
  - Heavy - 350-500 kcal/hr
- Worker evaluation
  - Acclimatized
  - Unacclimatized
  - State of hydration
  - Medical condition and fitness level
  - Clothing

The wet bulb globe temperature (WBGT) is an index used to evaluate occupational heat stress, outdoors and indoors. The WBGT is calculated by the following 2 equations:

1.  $WBGT \text{ (index)} = 0.7 T_{nwb} + 0.3 T_g$   
(indoors or outdoors on cloudy day)



**Figure 1.** Threshold Standards for Work-related Heat Production in Acclimatized and Non-acclimatized individuals.

2. WBGT (index) =  $0.7 T_{nwb} + 0.2 T_g + 0.1 T_{db}$   
(outdoors in direct sunlight)

$T_{nwb}$  = natural wet bulb temperature as measured by a specific instrument sensitive to humidity and air movement

$T_g$  = globe temperature, a measure of radiant heat and convective heat exchange with ambient air

$T_{db}$  = dry bulb temperature, a direct measurement of air temperature.

Instruments are available that measure direct wet bulb globe temperatures.

**Work Load Evaluation.** Total heat load is a combination of heat produced by the body and total environmental heat load. For work performed in hot environments, the work load category of the job and the heat exposure limit should be compared to the acceptable standards in order to protect the worker.

Work load is divided into light, moderate and heavy from which the permissible heat exposure TLV can be estimated (Tab. 4). Estimating the worker's metabolic rate from work load can also be performed (Tab. 6) and compared to standards (Fig. 1).

**Worker Evaluation.** Guidelines for monitoring workers under heat stress are listed in Table 7. Proper training and fitness are essential to prevent heat stress related health problems. Training should include pre-placement training for new employees hired for thermal stress environments and annual updates for regular workers. Workers should be taught to understand the basics of heat stress and physiological responses to thermal stress. They should have instruction in heat stress hygiene practices, recognition of

heat-related disorders and first-aid knowledge. Company policy and guidelines should be reviewed as part of the safety program.

Heat stress hygiene practices are those taken by the individual worker to prevent or reduce the risk of heat-related health problems. These include fluid replacement, general fitness and healthiness, self-limiting of exposure, and acclimatization.

Fluid replacement is essential because fluid losses under heat stress can reach or exceed 6 liters a day or 13 pounds of weight [40]. Thirst is not an efficient motivator for complete fluid replacement; therefore, workers should drink small quantities of fluid or cool water frequently. Drinking fluid prior to starting work can help reduce dehydration also. Beverages containing 0.1% salt should be available to unacclimatized workers. Fluid breaks should be taken every 15–20 minutes in heat stress environments [1, 40].

Acclimatization to heat stress environments requires physiological and psychological adjustments during the first 7–10 days of heat stress exposure. As acclimatization progresses, the level of work ability increases and risk for heat-related problems declines. Loss of acclimatization occurs during any period of illness, and workers who are off the job need to be re-acclimatized before resuming full work loads in heat stress occupations.

Clothing influences heat stress risks. The heat exposure TLVs are valid for light clothing. If workers must wear heavier clothing or protective gear that interferes with sweat evaporation or has a high insulating capacity, then heat tolerance is significantly reduced (Tab. 8). When personal protective equipment must be utilized, special training and expertise are required to judge heat stress permissible exposure limits.

Overall health and fitness contribute to reduction of heat stress related illness. Medical surveillance of worker health and fitness is essential in occupations requiring thermal stress exposure. Workers with cardiovascular disease, respiratory disease and renal disease are at higher risk for heat stress related illness. Certain medications also increase the risk, especially those that interfere with heat loss mechanisms such as anticholinergic drugs:

- Tricyclic antidepressants,
- Thyroxine,
- Pherothiazines,
- Antihistamines,
- Buterophenones,
- Benztropine mesylate,
- Atropine.

Medication review should include both prescription and over-the-counter drugs.

Environmental controls such as reducing temperature, reducing humidity, increasing air flow, can be helpful in reducing heat stress risk. But these measures may not be possible to effect in some environments. In this case, administrative controls should be instituted to limit worker exposure (pacing of work, limiting work time, changing hours).

**Table 9.** Windchill equivalent temperatures (°C).

Estimated Wind Speed (in mph)	Actual Reading Temperature (°C)											
	10	4	-1	-7	-12	-18	-23	-29	-34	-40	-46	-51
Equivalent Chill Temperature (°C)												
Calm	10	4	-1	-7	-12	-18	-23	-29	-34	-40	-46	-51
5	9	3	-3	-9	-14	-21	-26	-32	-38	-44	-49	-56
10	4	-2	-9	-16	-23	-30	-37	-43	-50	-57	-64	-71
15	2	-6	-13	-21	-28	-35	-43	-50	-58	-65	-73	-80
20	0	-8	-16	-23	-32	-39	-47	-55	-63	-70	-79	-85
25	-1	-9	-18	-26	-34	-42	-51	-59	-67	-76	-83	-92
30	-2	-11	-19	-28	-36	-44	-53	-62	-70	-78	-87	-96
35	-3	-12	-20	-29	-37	-46	-55	-63	-72	-81	-89	-98
>35	-3	-12	-21	-29	-38	-47	-56	-65	-73	-82	-91	-100
Wind speeds greater than 40 mpg have little additional effect.	LITTLE DANGER In <hr with dry skin. Maximum danger of false sense of security.				INCREASING DANGER Danger from freezing of exposed flesh within one minute.				GREAT DANGER Flesh may freeze within 30 seconds.			

Trenchfoot and immersion foot may occur at any point on this chart.

Developed by U.S. Army Research Institute of Environmental Medicine, Natick, MA.

Increased air motion increases evaporative cooling and convective cooling if the air temperature is less than 35°C (95°F) [40]. Between temperatures of 35–40°C (95–104°F) increased air movement actually increases heat gain by convection, but more than offsets this gain by the increase in evaporation [40]. For temperatures above 40°C (104°F), increased air movement increases overall heat stress. The greatest reduction in heat stress occurs when air movement increases from less than 1 meter/second to 2 meters/second. No gain is achieved in evaporative cooling at rates higher than 3 meters/second [40].

Air temperatures greater than 40°C (104°F) cause heat gain to exposed workers. Air temperatures below 32°C (90°F) cause heat loss to exposed workers. Thus, simply lowering the temperature, if possible, reduces heat stress. Lowering humidity adds to this reduction in heat stress risk.

Some workers may require special clothing to protect them from heat stress such as reflective clothing that deflects heat. Reflective clothing reduces radiant heat stress, but it also can reduce sweat evaporation and increase the risk of heat stress related problems. Personal cooling clothing such as circulating water garments and ice garments should be selected by knowledgeable experts in occupational safety.

### ENVIRONMENTAL AND OCCUPATIONAL COLD STRESS

Cold stress TLVs exist to protect workers from hypothermia and frostbite [1]. The objective of cold stress TLVs is to prevent core temperature from falling below

36°C (96.8°F), and to prevent cold injury. To combat cold stress, the body tries to conserve heat loss. Heat loss increases with wind speed and widening temperature between skin and the environment. The equivalent chill temperature (ECT), also called the chill factor, reflects the effect of wind on cold stress risk (Tab. 9, 10).

**Recognizing Cold Stress.** Workers in cold stress environments require dry clothing with adequate insulation when working in temperatures below 4°C (40°F). Wind chill factors must also be taken into account. Gloves, head protection and eye protection may also be necessary.

Cold stress symptoms begin with feeling cold. Other early signs may be loss of manual dexterity, unsafe behaviors, shivering or accidents. The person's core temperature may be below 36°C (96.8°F) if these signs and symptoms are present [40]. A core temperature of 35°C (95°F) is associated with maximum shivering. Hypothermia can occur with ambient temperature up to 10°C (50°F). Skin does not freeze until air temperature is less than -1°C (30°F) or wind chill (ECTs) is greater than -30°C (-22°F) [40]. Cold injury to tissues is unlikely to occur without initial signs and symptoms of hypothermia. Cardiovascular disease, arterial and venous insufficiency may exacerbate cold stress.

**Control of Cold Stress.** Evaluation and control of cold stress should take place in the following circumstances:

- Continuous exposure of skin should not be permitted when air speed and temperature results in a wind chill temperature of -32°C (-25.6°F).

**Table 10.** Windchill equivalent temperatures (°F).

Estimated Wind Speed (in mph)	Actual Reading Temperature (°F)											
	50	40	30	20	10	0	-10	-20	-30	-40	-50	-60
	Equivalent Chill Temperature (°F)											
Calm	50	40	30	20	10	0	-10	-20	-30	-40	-50	-60
5	48	37	27	16	6	-5	-15	-26	-36	-47	-57	-68
10	40	28	16	4	-9	-24	-33	-46	-58	-70	-83	-95
15	36	22	9	-5	-18	-32	-45	-58	-72	-85	-99	-112
20	32	18	4	-10	-25	-39	-53	-67	-82	-96	-110	-121
25	30	16	0	-15	-29	-44	-59	-74	-88	-104	-118	-133
30	28	13	-2	-18	-33	-48	-63	-79	-94	-109	-125	-140
35	27	11	-4	-20	-35	-51	-67	-82	-98	-113	-129	-145
>35	26	10	-6	-21	-37	-53	-69	-85	-100	-116	-132	-148
Wind speeds greater than 40 mpg have little additional effect.	LITTLE DANGER In <hr with dry skin. Maximum danger of false sense of security.			INCREASING DANGER Danger from freezing of exposed flesh within one minute.				GREAT DANGER Flesh may freeze within 30 seconds.				

Trenchfoot and immersion foot may occur at any point on this chart.

Developed by U.S. Army Research Institute of Environmental Medicine, Natick, MA.

- For workers immersed in water or with wet clothes, immediate change to dry clothes is critical at air temperatures of less than 2°C (35°F).
- TLVs are recommended for properly clothed workers for period of work at temperatures below freezing.
- Special precautions and hand warming methods are required if fine dexterity work is performed bare-handed for more than 10-20 minutes in temperatures below 16°C (60.8°F).
- Gloves should be worn when temperatures are less than 16°C (60.8°F) for light work, -7°C (19°F) for moderate and heavy work.
- Air temperatures equal to or below -17.5°C (0°F) require mittens.
- For air temperature below -7°C (19°F) are within reach, a warning should be given to avoid bare skin contact with cold surfaces such as metal.
- Eye protection should be available for ice and snow conditions.

Administrative controls, personal protection and engineering controls should be implemented to reduce cold stress and cold related injury [40]:

- Engineering Controls
  - General warming
  - Hand warming
  - Minimizing air movement
  - Reducing conductive heat loss
  - Warming shelters
- Administrative Controls
  - Work rest cycles
  - Work schedule changes

- Additional workers
- Observation of workers – “buddy” system
- Avoid long sedentary periods
- Acclimatization period for new workers
- Medical surveillance
- Personal Protection Controls
  - Appropriate clothing
  - Attention to exposed body parts
  - Avoid getting wet
- For handling evaporative organic solvents, avoid skin contact and soaking of clothing due to increased evaporative cooling effect.
- Wear layers of clothing made of insulating materials. The outer layer should be water repellent and breathable.
- Special medical certification is necessary for workers whose job exposes them routinely to temperatures below -24°C (-11°F).

**REFERENCES**

1. ACHIG: *Threshold Limit Values and Biogocial Exposrue Indicies*. Cincinnati, Ohio, 1997.
2. Alhaddad IA, Khalil M, Brown EJ, Jr: Osborn waves of hypothermia. *Circulation* 2000, **101**, E233-E244.
3. Allan JR, Wilson CG. Influence of acclimatization on sweat sodium concentration. *J Appl Physiol* 1971, **30**, 708-712.
4. Anonymous. Hypothermia-related deaths-suffolk County, New York, January 1999-March 2000, and United States, 1979-1988. *JAMA* 2001, **285**, 1009-1010.
5. ASHARE: *Zone Standards*, 1981. www.ashrae.org/
6. Bergstein JM, Slakey DP, Wallace JR, Gottlieb M: Traumatic hypothermia is related to hypotension, not resuscitation. *Ann Emerg Med* 1996, **27**, 39-42.

7. Bouchama A, al-Sedairy S, Siddiqui S, Shail E, Rezeig M: Elevated pyrogenic cytokines in heatstroke. *Chest* 1993, **104**, 1498-1502.
8. Bouchama A, Parhar RS, el-Yazigi A, Sheth K, al-Sedairy S: Endotoxemia and release of tumor necrosis factor and interleukin 1  $\alpha$  in acute heatstroke. *J Appl Physiol* 1991, **70**, 2640-2644.
9. Boulant JA: Hypothalamic control of thermoregulation. **In:** Morgane PJ, Panksepp J (Eds): *Handbook of the hypothalamus*, vol 3, Part A. *Behavioral studies of the hypothalamus*, 1-70. Marcel Dekker, New York 1981.
10. Collins KJ, Exton-Smith AN, Doré C: Urban hypothermia. Preferred temperature and thermal perception in old age. *Br Med J* 1981, **282**, 175.
11. Costrini AM: Emergency treatment of exertional heatstroke and comparison of whole body cooling techniques. *Med Sci Sports Exerc* 1990, **22**, 15-18.
12. Danzl DF: Accidental hypothermia. **In:** Auerbach RS: *Management of Wilderness Emergencies*, ed 3. Mosby, St. Louis, MO 1995.
13. Danzl DF: Accidental hypothermia. **In:** Marx: *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 5th Edition. Mosby, St. Louis 2002.
14. Danzl DF, Pozos RS, Auerbach PS, Glazer S, Goetz W, Johnson E, Jui J, Lilja P, Marx JA, Miller J, Mills W, Nowak R, Shields R, Vicario S, Wayne M: Multicenter hypothermia survey. *Ann Emerg Med* 1987, **16**, 1042-1055.
15. Elenbaas RM, Mattson K, Cole H, Steele M, Ryan J, Robinson W: Bretylium in hypothermia-induced ventricular fibrillation in dogs. *Ann Emerg Med* 1984, **13**, 994-999.
16. Epstein Y, Sohar E: Fluid balance in hot climates: sweating, water intake and prevention of dehydration. *Public Health Rev* 1985, **13**, 115-137.
17. Ferguson J, Epstein F, Van de Leuv J: Accidental hypothermia. *Emerg Med Clin North Am* 1983, **1**, 619-637.
18. Gaffin SL, Moran DS: Heat-related illnesses. **In:** Auerbach PS (Ed): *Wilderness medicine management of wilderness and environmental emergencies*, 240-316. Mosby-Year Book, St. Louis 2001.
19. Giesbrecht GG: Cold stress, near-drowning and accidental hypothermia: a review. *Aviat Space Environ Med* 2000, **71**, 733-752.
20. Giesbrecht GG: Prehospital treatment of hypothermia. *Wilderness Environ Med* 2001, **12**, 24-31.
21. Giesbrecht GG, Bristow GK: Influence of body composition on rewarming from immersion hypothermia. *Aviat Space Environ Med* 1995, **66**, 1144-1150.
22. Gilat R, Shibolet S, Sohar E: The mechanism of heatstroke. *J Trop Med Hyg* 1963, **66**, 204-212.
23. Harnett RM, Pruitt JR, Sias FR: A review of the literature concerning resuscitation from hypothermia: 1. The problem and general approaches. *Aviat Space Environ Med* 1983, **54**, 425-434.
24. Jesati RM: Management of severe hyperthermia with Chlorpromazine and refrigeration. *NEJM* 1956, **254**, 426-428.
25. Johnson HL, Nelson RA, Consolazio CF: Effects of electrolyte and nutrient solutions on performance and metabolic balance. *Med Sci Sports Exerc* 1988, **20**, 26-33.
26. Jolly BT, Ghezzi KT: Accidental hypothermia. *Emerg Med Clin North Am* 1992, **10**, 311-327.
27. Keatinge WP: Direct effects of temperature on blood vessels: Their role in cold vasodilation. **In:** *Physiological and Behavioral Temperature Regulation*, 231-236. Charles Thomas, Springfield, IL, 1970.
28. Khogali M: Heatstroke and heat exhaustion. *Travel Traffic Med Int* 1983, **1**, 166-169.
29. Khogali M: The Makkah body-cooling unit. **In:** Khogali M, Hales JRS (Eds): *Heat stroke and temperature regulation*. Academic Press, Sydney 1983.
30. Knochel JP: Environmental heat illness. *Arch Int Med* 1974, **133**, 841-864.
31. Knochel JP, Reed G: Disorders of heat regulation. **In:** Narin RG (Ed): *Maxwell and Kleeman's Clinical disorders of fluid and electrolyte metabolism*, 1549-1590. McGraw-Hill, New York 1994.
32. Krueger-Kalinski MA, Schriger DL, Friedman L, Votey SR: Identification of risk factors for exertional heat-related illnesses in long-distance cyclists: Experience from the California AIDS Ride. *Wilderness Environ Med* 2001, **12**, 81-85.
33. Lazar HL: The treatment of hypothermia. *NEJM* 1997, **337**, 1545-1547.
34. Leben J, Tryba M, Bading B, Heuer L: Clinical consequences of hypothermia in trauma patients. *Acta Anaesthesiol Scand* 1996, **109 (Suppl.)**, 39-41.
35. Lind AR, Bass DE: Optimal exposure time for development of acclimatization to heat. *Fed Proc* 1963, **22**, 704-708.
36. Miller JW, Danzl DF, Thomas DM: Urban accidental hypothermia. *Ann Emerg Med* 1980, **9**, 456-461.
37. Murray R: The effects of consuming carbohydrate-electrolyte beverages on gastric emptying and fluid absorption during and following exercise. *Sports Med* 1987, **4**, 322-351.
38. O'Donnell TF, Clowes GHA: The circulatory abnormalities of heatstroke. *N Engl J Med* 1972, **287**, 734-737.
39. Osborn JJ: Experimental hypothermia; respiratory and blood pH changes in relationship to cardiac function. *Am J Physiology* 1953, **75**, 389.
40. Plog B, Niland J, Quinlan P: *Fundamentals of Industrial Hygiene*. National Safety Council, Itasca, Illinois, 1996.
41. Pugh L: Cold stress and muscular exercise with special reference to accidental hypothermia. *Br Med J* 1967, **2**, 333-337.
42. Reuler JB: Hypothermia. Pathophysiology, clinical settings and management. *Ann Int Med* 1978, **89**, 519.
43. Sagawa S, Shiraki K, Yousef M, Konda N: Water temperature and intensity of exercise in maintenance of thermal equilibrium. *J Appl Physiol* 1988, **65**, 2413-2419.
44. Schagatay E, Hold B: Effects of water and ambient air temperatures on human diving bradycardia. *Eur J Appl Physiol* 1996, **173**, 1-6.
45. Schneider SM: Hypothermia: From recognition to rewarming. *Emerg Med Rep* 1992, **13**, 1-20.
46. Semenza JC, Rubin CH, Falter KH, Selanikio JD, Flanders WD, Howe HL, Wilhelm JL: Heat-related deaths during the July 1995 heat wave in Chicago. *N Engl J Med* 1996, **335**, 84-90.
47. Senay LC, Mitchell D, Wyndham CH: Acclimatization in a hot, humid environment: body fluid adjustments. *J Appl Physiol* 1976, **40**, 786-796.
48. Shibolet S, Lancaster MC, Danon Y: Heatstroke: a review. *Aviat Space Environ Med* 1976, **47**, 280-301.
49. Thompson R, Hayward J: Wet-cold exposure and hypothermia: thermal and metabolic response to prolonged exercise in rain. *J Appl Physiol* 1996, **81**, 1128-1137.
50. Vanggaard L: Physiological Reactions to Wet-Cold. *Aviat Space Environ Med* 1975, **46**, 33-36.
51. Walker JS, Barnes SB: Heat emergencies. **In:** Tintinalli JE, Kelen GD, Stapczynski JS (Eds): *Emergency Medicine a comprehensive study guide*, 1235-1242. McGraw-Hill, New York 2000.
52. Weiner JS: The regional distribution of sweating. *J Physiol* 1945, **104**, 32-40.
53. Weiner JS, Khogali M: A physiological body-cooling unit for treatment of heatstroke. *Lancet* 1980, **1**, 507-509.
54. Weller AS, Millard CE, Stroud MA, Greenhaff PL, Macdonald IA: Physiological responses to cold stress during prolonged intermittent low- and high-intensity walking. *Am J Physiol* 1997, **272**, R2025-R2033.
55. Weyman AE, Greenbaum DM, Grace WJ: Accidental hypothermia in an alcoholic population. *Am J Med* 1974, **56**, 13-21.
56. Wheeler KB, Banwell JG: Intestinal water and electrolyte flux of glucose-polymer electrolyte solutions. *Med Sci Sports Exerc* 1986, **18**, 436-439.
57. White JD: Hypothermia: The Bellevue experience. *Ann Emerg Med* 1982, **11**, 417-424.
58. White J, Ford MA: The hydration and electrolyte maintenance properties of an experimental sports drink. *Br J Sports Med* 1983, **17**, 51-58.
59. Young AJ, Castellani JW, O'Brien C, Shippee RL, Tikuisis P, Meyer LG, Blanchard LA, Kain JE, Cadarette BS, Sawka MN: Exertional fatigue, sleep loss, and negative energy balance increase susceptibility to hypothermia. *J Appl Physiol* 1998, **85**, 1210-1217.