

NFHS Sports Medicine Handbook: HEAT-RELATED ILLNESS

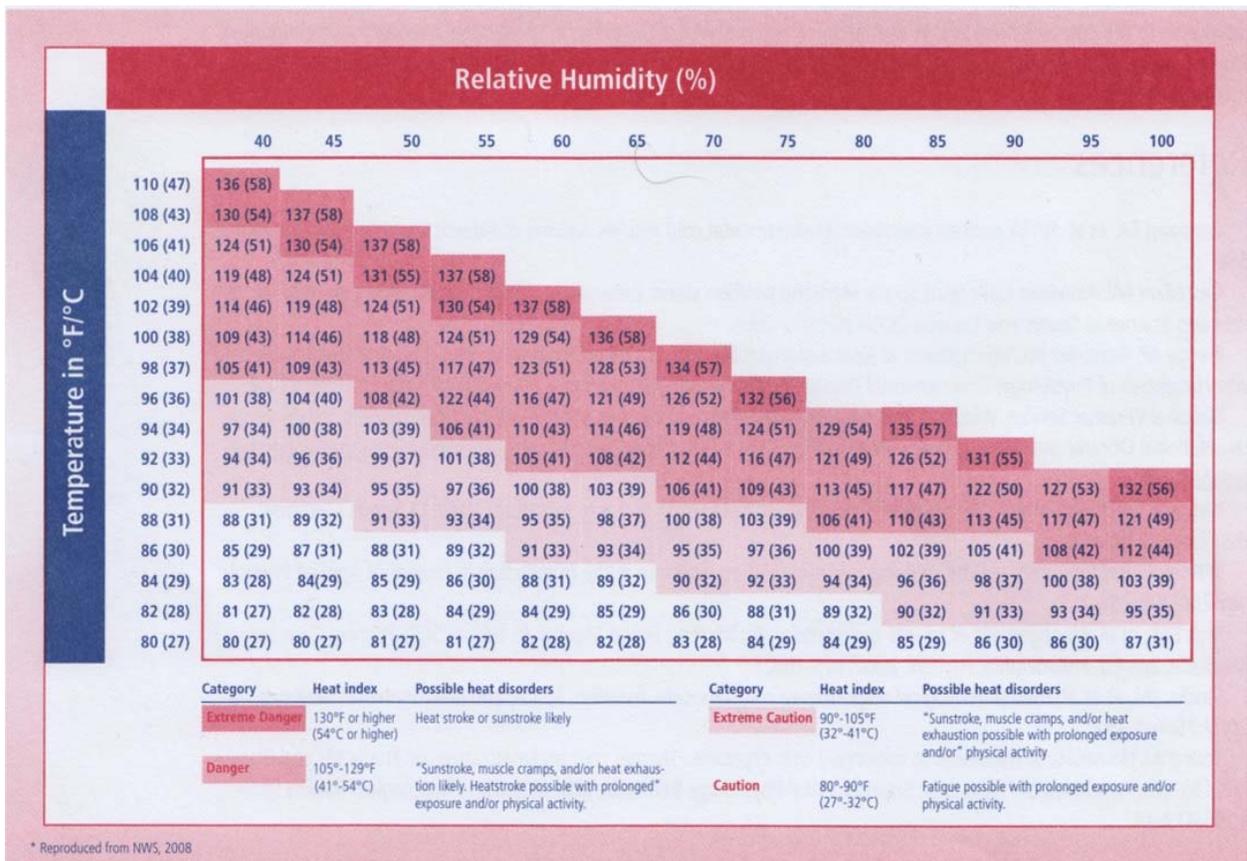
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- Thermoregulation depends primarily on the evaporation of sweat to dissipate the heat produced by exercise.
- Predisposing factors that increase an athlete's risk for heat illness include: dehydration, heat acclimatization, clothing/equipment, fitness level, recent or current illness, medication use, obesity, age and prior heat illness.
- Prevention of heat illness includes designing an environmental action plan, modifying activity time (including intensity and duration) and increasing frequency and length of rest periods, providing and monitoring adequate hydration, minimizing clothing and equipment, ensuring adequate heat acclimatization, early recognition of signs and symptoms and appropriate sports medicine care.

SIGNIFICANCE

Heat illness is the leading cause of preventable death in high school athletes. These heat stroke deaths mainly occur in the summer months, at the beginning of conditioning for fall sports. Heat production during intense exercise is 15 to 20 times greater than at rest and can raise body core temperature one to two degrees Fahrenheit every five minutes unless heat is dissipated.

Figure 10. Heat Index Chart.



BACKGROUND

Thermoregulation

Athletes lose heat by evaporation, conduction, convection and radiation. Heat is lost from the skin by evaporation of sweat. *Conduction* is passive transfer of heat from warmer to cooler objects by direct contact. Heat transfer from the core to the peripheral muscles and skin and from skin to an ice bag is by conduction. *Convection* is the warming of air next to the body and the displacement of that warm air by cool air. Wind accelerates convection. *Radiation* is the loss of heat from the warmer body to the cooler environment by electromagnetic waves. At rest, 20 percent of body heat loss is by evaporation and 50 percent by radiation. With exercise, up to 90 percent of heat loss is by evaporation. Thus, thermoregulation during exercise relies primarily on evaporation. Radiation becomes a more important source of heat loss during exercise as the air temperature falls significantly below body temperature.

The body normally maintains core temperature within the range of 95 to 104 degrees Fahrenheit. Brain temperature is always slightly higher than body temperature. The removal of body heat is controlled centrally by the hypothalamus and spinal cord and peripherally by centers in the skin and organs. The body compensates for the increased heat produced during exercise by increasing blood flow to the skin and increasing sweat production so as to increase heat loss by evaporation. Importantly, evaporation is less effective at high humidity and when sweat production decreases due to dehydration. When heat production exceeds the ability to dissipate the heat, then core temperature, along with brain temperature, rises excessively. The result is further decompensation of normal thermoregulation, decreased heat dissipation, decreased cerebral blood flow and decreased muscular strength. This sets the stage for heat illness.

Acclimatization

An effective protection against heat illness is acclimatization. Proper acclimatization requires progressively increasing the duration and intensity of exercise during the first 10 to 14 days of heat exposure. However, full heat acclimatization may require up to 12 weeks of exposure. With repeated exposure to heat, there is an increase in skin blood flow rate, more rapid onset of sweating, an increase in plasma volume and a decrease in metabolic rate. Equipment and clothing should be minimized during acclimatization. Heat acclimatization can be lost over two weeks without ongoing heat exposure, but the loss may be slower in better-conditioned athletes.

Measuring Environmental Risk of Heat Illness

As humidity increases, perspiration evaporates less readily. Heat loss by sweating can be dramatically impaired when the humidity is greater than 50 percent. The Heat Index is a calculation of the danger of heat illness based on ambient temperature and humidity. The Heat Index can be determined by entering the zip code at your location at this Web site: <http://www.osaa.org/heatindexdefault.asp>. The Heat Index rises, so does the risk of heat illness (Figure 10).

Wet bulb globe temperature (WBGT) is the most effective method for determining environmental heat risk, because it takes into account not only ambient temperature and humidity, but also solar radiation. WBGT employs a dry bulb thermometer that measures ambient temperature, a wet bulb thermometer that measures humidity and a black globe thermometer that measures radiant heat.

As WBGT increases, the risk for heat illness increases (Table 11). WBGT less than 65 is low risk. WBGT 65 to 73 is moderate risk, WBGT 73 to 82 is high risk, and WBGT greater than 82 is extreme risk of heat

illness. Experts recommend that distance races should be cancelled if WBGT is 80 or above. Only acclimatized, fit, low-risk athletes should undertake limited exercise at WBGT 86 to 90. Exercise should absolutely be cancelled for everyone when WBGT is 90 or more. The WBGT Risk Indices were developed for athletes wearing only a T-shirt and light pants. Therefore, safe values should be adjusted downwards in the presence of equipment and clothing that inhibit evaporation.

MANAGEMENT AND PREVENTION

Practices and Contests

The greater the risk of heat illness, the more steps should be taken to safeguard the athletes, and the greater consideration should be given to cancellation or postponement of a practice or contest. An Environmental Action Plan should be in effect, covering every athletic practice and competition, and it must delegate responsibility for decision-making (see Emergency Action Planning chapter).

1. Measure the WBGT when possible. If not, then determine the heat index. Re-measure several times throughout the event or practice. Infrared thermometers can be used to measure playing surface temperature. The greater the intensity and duration of an event, the greater the risk of heat illness. Long-distance endurance events place athletes at more risk than sports that have frequent breaks during play. Consideration should be given to reducing playing time, extending rest periods and creating regular stoppage of play for rest and hydration. Practices and contests should not be scheduled during the hottest part of the day (commonly 11 a.m. to 6 p.m.).
2. Minimize clothing and equipment (football or lacrosse practice without shoulder pads and helmets).
3. Provide unlimited opportunities for hydration (see Fluid Replacement and Dehydration chapter). Provide extra water for wetting clothes, hair and face. Hydration should never be withheld as a punishment!
4. In multi-session or multi-day events, monitor for cumulative dehydration by repeated measurement of body weight.
5. Allow a minimum of three, and preferably six, hours for recovery and rehydration between exercise sessions during "daily doubles."
6. Assure acclimatization prior to high endurance/intensity exercise in heat.
7. Consider providing shade, air conditioning or fans on sidelines during contests and practices.
8. If at all possible, practices should be attended by an athletic trainer or team physician who is prepared to manage heat-related emergencies.
9. Identify athletes whose medical history places them at increased risk (see Risk Factors below).

Table 11. Wet Bulb Globe Temperature and Risk of Heat Illness.

<65°	Low Risk
65-73°	Moderate Risk
73-82°	High Risk
>82°	Very High Risk
>90°	Cancel Activity

RISK FACTORS FOR HEAT ILLNESS

1. **Dehydration.** Fluid loss during exercise occurs primarily by perspiration and respiration. Dehydration during exercise occurs more rapidly in hot environments, when perspiration exceeds oral fluid replacement. Moderate dehydration (three to five percent body weight) reduces exercise performance and makes the athlete more susceptible to fatigue and muscle cramps. With severe dehydration, sweat production and cutaneous blood flow decrease and the athlete is less able to dissipate the heat produced by exercise. Water deficits of six to 10 percent can occur with exercise in hot environments, reducing exercise tolerance and heat dissipation by decreasing cardiac output, sweat production, and skin and muscle perfusion.

In addition to losing fluid with sweating, electrolytes (salt or sodium and chloride) are also lost. The percentage of salt lost in sweat usually decreases with an improving level of heat acclimatization. Salt depletion can be a significant factor in muscle cramps. While cold water is a good fluid replacement during short duration exercise, a sports drink with six to eight percent carbohydrate is preferable during continuous activity lasting 45 minutes or more. Regular, scheduled fluid replacement is important because athletes typically do not become thirsty until they have already lost two percent of body weight in fluid. (See Fluid Replacement and Dehydration chapter).

An athlete may begin an activity in a dehydrated state due to inadequate rehydration following previous exercise, attempts to lose weight rapidly, diuretic medication, febrile illness, or gastrointestinal illness with vomiting or diarrhea. Measurement of body weight before and after activity is a good estimate of hydration status changes. Rehydration should be with a fluid volume that meets the weight lost with activity, ideally not exceeding 48 ounces per hour. Urine volume and color are another means by which to estimate hydration with lower volume and darker color representing greater dehydration.

2. **Clothing and Equipment.** Clothing and equipment inhibit heat loss from the body and increase the risk for heat illness. Dry clothing and equipment absorb sweat and prevent evaporative heat loss. Dark clothing or equipment produces radiant heat gain. Clothing and equipment decrease convective heat loss by interfering with air contact with the body. During periods of high WBGT or Heat Index, the risk of heat illnesses increases when clothing and equipment are worn. Thus, risk may be minimized through removing equipment and participating in drills wearing shirts and shorts only. Given that a great deal of heat is radiated from the head, helmets should be removed early on in hot and humid conditions.
3. **Fitness.** Physical training and improved cardiovascular fitness reduce the risk of heat illness.
4. **Febrile illness.** A fever increases core temperature and decreases the ability of the body to compensate. It is dangerous to exercise with a fever, especially when WBGT is high. Athletes with a fever, respiratory illness, vomiting or diarrhea should not exercise, especially in a hot environment.
5. **Medications.** Amphetamines (including ADHD medications), ephedrine, synephrine, ma huang and other stimulants increase heat production. Some medications have anti-cholinergic actions (amitriptyline, Atrovent) resulting in decreased sweat production. Diuretics can produce dehydration. Athletes taking medication for ADHD should be monitored closely for signs and symptoms of heat illness.
6. **Obesity.** Athletes with a high percentage of body fat are at increased risk for heat illness, as fat acts to insulate the body and decreases the body's ability to dissipate heat.
7. **Sickle Cell Trait.** Athletes with sickle cell trait (SCT) are at increased risk for a sickling crisis with exercise during hot weather. Special precautions should be taken in hot and humid conditions for athletes with SCT (see Sickle Cell Trait chapter).

8. **A prior episode of heat illness** is a risk factor for a subsequent heat illness. After an episode of heat stroke, most athletes demonstrate normal thermoregulation within two months, but the rate of recovery is highly variable and may require up to a year or more. Decreased heat tolerance may affect 15 percent of athletes with a history of previous heat illness.

STAGES OF HEAT ILLNESS

1. **Exercise-associated Muscle Cramps (EAMC).** Painful muscle spasms following prolonged exercise, often, but not always, in a hot environment. These are sometimes called "heat cramps."
Recognition: The cramps can occur without warning, can be excruciatingly painful, and may last several minutes or longer. They may be replaced by the onset of a cramp in another location. Severe episodes can last up to six to eight hours. Commonly, heat cramps affect the calf, but the thighs, hamstrings, abdomen and arms may be involved. Core temperature may be normal or increased and signs and symptoms of dehydration such as thirst, sweating and tachycardia may occur.

EAMC are usually associated with exercise-induced muscular fatigue, dehydration and a large loss of sodium through sweat. Sweat sodium losses that are incompletely replaced result in a total body sodium deficit. Low extracellular (outside of the cells in our body) sodium concentration is thought to alter nerve and muscle resting potential, resulting in EAMC. EAMC is more likely in athletes with high salt sweat content. Athletes with high salt sweat content or "salty sweaters" may be noticeable by salt staining on hats and clothing.

Management: EAMC usually responds to rest, prolonged stretching of involved muscle groups, and sodium replacement in fluid or food (e.g., one quarter teaspoon of table salt or one to two salt tablets in 500 ml of water or sports drink, tomato juice or salty snacks). In the case of severe full body cramps, the athlete should be transported by EMS to a hospital to receive intravenous fluids. Protracted cramping in the absence of signs of dehydration suggests dilutional hyponatremia (low sodium) and serum sodium levels should be measured prior to administering intravenous fluids.

2. **Heat Exhaustion.** Heat exhaustion is the inability to continue to exercise and can occur at any temperature and is not necessarily associated with collapse. Heat exhaustion associated with dehydration is more common in a hot, humid environment.

During high intensity exercise, blood flow to organs and skin decreases as blood flow to exercising muscle increases. When exercise, dehydration and humidity combine to make evaporative heat loss ineffective, the core body temperature increases. As core temperature rises, central controls of blood flow distribution begin to fail, and the body attempts to increase blood flow to the skin in an effort to increase radiant and convective heat loss. The result is a loss of the original decrease in blood flow to the internal organs and to the skin. Through a series of complex physiological events, the pooled blood in the skin and extremities is unable to transport heat from the core to the skin. Muscular fatigue, decreased urine output, decreased cerebral flow, increased core temperature and fainting (syncope) can result.

Recognition: Signs and symptoms of heat exhaustion include tachycardia, fatigue, weakness, piloerection (goose bumps), muscle cramps, nausea, vomiting, dizziness, syncope, headache, poor coordination and confusion. Rectal temperature is elevated, but below 104 degrees Fahrenheit (40 C). The skin may still be cool and sweating or may be hot and dry. Decreased cerebral perfusion may produce confusion or syncope. Heat exhaustion can be confused with other causes of depressed mental status in the athlete, including concussion, cardiac causes, infection, drug use, hypoglycemia and hyponatremia. Heat exhaustion is characterized by an elevated core body temperature. Any athlete with altered mental state of unknown etiology must be removed from activity and further evaluated.

Management: While heat exhaustion may present similarly to other conditions, heat exhaustion should be assumed if any of the signs and symptoms are present. Elevate the legs to increase venous return and cardiac preload, rehydrate to correct volume depletion, and transfer to a cool, shaded location. Aggressive decrease in core temperature is indicated to prevent progression to heat stroke. If a team physician or athletic trainer is unavailable to assess the athlete, EMS should be activated so the athlete can be transported to an emergency facility. There should be no same-day return to activity for athletes with syncope, altered mental status, neurologic symptoms or core temperature greater than 104 degrees Fahrenheit. Adequate time for full recovery is necessary prior to returning to play.

3. **Exertional Heat Stroke (EHS)** is defined by the presence of a rectal temperature greater than 104 degrees Fahrenheit (40C) combined with altered mental status. As heat production continues to exceed the body's capacity to dissipate the heat, then core temperature rises to a level that disrupts organ function.

Recognition: There is usually sweat-soaked, pale skin. Hyperventilation, tachycardia, vomiting, diarrhea and shock frequently progress to arrhythmia, acute renal failure, rhabdomyolysis (the release of muscle potassium, acid and enzymes into the blood as muscle cells break open and die), pulmonary edema, disseminated intravascular coagulopathy (coagulation of blood throughout the vessels) and cardiac arrest. Often, central nervous system signs are the first to appear: altered mental status, confusion, seizures and coma.

Management: EHS is a medical emergency and EMS must be activated. Successful treatment requires early recognition. Rapid reduction in core temperature is the key to prevention of organ failure. This is best accomplished by immersion in ice water. Less effective substitutes include ice packs to the groin and armpits, cool mist fans and alcohol rubs. If optimal cooling can be provided in the field, if there are no other life-threatening complications and if there is the ability to monitor the athlete during cooling, then cooling may be completed prior to transport. Otherwise, while efforts at cooling may be initiated in the field, they should not delay "load and go" EMS transport to a facility capable of comprehensive care.

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