Environment changes the outcome of athletic events on a regular basis. The effects of fog, rain, cold, heat, and altitude alter the performance of athletes, interfering with the body’s ability to maintain homeostasis. In this chapter, the effects of the environment on the human body are discussed. Ways of preventing environmental related illness are detailed. Treatments for these environmental illnesses are also addressed.

HEAT STRESS

Introduction
As heat stress increases, there is a reduction in physical performance (see Figure 21.1). Heat is a limiting factor during exercise, irrespective of dehydration or fuel availability status. The body must divert blood flow from muscles to the skin to regulate core temperature (1). This shunting causes a reduction in physical performance. Compounding the problem is the loss of fluid volume due to sweating causing a hypohydrated state. The diversion of blood flow from the muscles and loss of volume due to sweating leads to a decrease in cardiac function. A reduction in stroke volume and cardiac output occurs. This causes the muscles to use more carbohydrates, burning up stored glycogen and producing lactic acid (2). There is an increased perceived exertion and earlier onset of fatigue (3). All these factors bring about a decrement in physical performance.

The body’s ability to thermoregulate during exercise depends upon a myriad of factors (ambient temperature, humidity, and wind velocity, radiant heat from the sun, and the intensity and duration of exercise). To protect against the development of heat stress, exercise intensity and duration can be altered. Factors such as the weather cannot be altered, but they can be monitored. This is an important means of assessing environmental heat stress, an important factor in planning any outdoor sporting event.

Wet bulb globe temperature (WBGT) is the most efficient method (4) for determining environmental heat stress. The WBGT is determined by using several instruments: (a) a dry bulb thermometer, which measures ambient temperature (T<sub>d</sub>b), (b) a wet bulb thermometer that measures humidity, (T<sub>wb</sub>) and (c) a black globe thermometer (T<sub>g</sub>) that measures radiant heat. If the WBGT Index and wind speed are both measured, an effective temperature can be calculated. Table 21.1 correlates WBGT measurements and exercise safety. A useful website is http://www.smasa.asn.au/resources/hotweather.htm (September 2006.)

Thermoregulation
The main forms of heat loss in humans are evaporation, conduction, convection, and radiation. Evaporation is the loss of heat by evaporation of sweat from the skin. Conduction is the loss of heat passively through tissue to the periphery from the core. Convective heat loss occurs when heat is transferred from the core by the blood to the skin, warm air next to the body is displaced by cool air. The biggest factor contributing to convective heat loss is wind. Radiation is the loss of heat from the warmer body...
to the colder environment. Exercising muscles produce a large amount of heat. Roughly, 75% of the energy produced by the utilization of carbohydrates and free fatty acids is converted into heat. The human body acts as a literal furnace. At rest, humans utilize different methods of regulating core temperature compared to times of activity. At rest, the body relies on radiation over evaporative heat loss. The normal percentages are 50% and 20% for radiation and evaporation, respectively, at rest compared to 3% and 90% when exercising. The change to heat loss by way of evaporation is necessary to maintain core temperature in a narrow physiologic range.

The human body maintains a core temperature in the range of 35°C to 40°C (95°F to 104°F). The core temperature increases with exercise reaching a plateau level of homeostasis tolerated by the athlete. This level can be as high as 41°C (105.8°F) in elite athletes. The maintenance of core temperature is accomplished by the use of thermodetectors in the hypothalamus, spinal cord and limb muscles to maintain and regulate core temperature during exercise. Well-conditioned athletes can produce and dissipate over 1,000 kcals/hour safely. Radiation and convection dissipate most heat when the ambient temperature is greater than 20°C (68°F) and less than 35°C (95°F). Radiation is very important when the ambient temperature is below the body temperature. The body uses conduction and convection to transfer heat less effectively.

Evaporation is the main defense against heat stress. The body sweats to remove heat. This is accomplished by the evaporation of the sweat from the skin. Evaporative heat lost can remove a large amount of energy with 1 L of sweat removing approximately 600 Kcal of energy. An athlete will sweat varying amounts with the normal range being 0.5 to 1.5 L/hour but enormous rates of 4 L/hour have been noted. Sweat is only useful when it is in contact with the skin. If it is wiped off or rolls off the body, then the heat cannot be transferred away from the skin by this method.

The body is able to compensate for the energy produced in exercise to a certain point after which the compensatory factors fail (5). This occurs when the amount of heat generated by the human furnace is greater than the amount possible to dissipate. This varies by the intensity of the activity and the surrounding environment. The core temperature increases as the body is unable to further remove heat by evaporation. The two main reasons for the inability to shed further heat by evaporation are (a) a high WBGT in which the humidity is such that evaporation is diminished, and (b) hypohydration or dehydration in which the production of sweat is decreased. As the loss of body fluid increases, the core temperature increases faster. Compounding the problem, with dehydration the core temperature during exercise that an athlete is able to tolerate is lower. This is due to the reduction in stroke volume as well as the reduction of the volume of blood to the skin and decrease in sweating with dehydration. Hypohydration, as low as 1%, can produce an elevation in core temperature during exercise (6). The amount of increase in core temperature ranges is significant with 0.1°C to 0.25°C (32.18°F to 32.45°F)/percent body weight lost. As the amount of hypohydration increases so does the core temperature.

Predisposition

Several factors increase the susceptibility of an athlete to heat stress (7). Dehydration is the prime factor as discussed in the previous section. Preventing dehydration is important in preventing heat stress. A few facts are important to understand. Regular replacement of fluids is needed because thirst is a poor predictor of fluid status. Most athletes do not develop thirst until they have lost more than 2% body weight. One should note that sweat is hypotonic. There is very little salt in sweat, as well as the amount of salt in sweat decreases with training and acclimatization. Therefore, cold water is the best replacement fluid during exercise. When prolonged activity of greater than 1 hour is performed, a hypotonic salt solution with a 6% to 7% glucose polymer will aid in absorption of fluids.

Acclimatization is important in dealing with heat stress during exercise. This develops with 7 to 10 days of exposure. The benefits of heat acclimatization come from improved skin blood flow rates, earlier onset of sweating, an increased plasma volume, and a lower metabolic rate. Exercising in the heat for 100 minutes is the most effective method of adapting. It is important to increase the core temperature during exercise to develop the adaptive effects quickly. Even sedentary people will adapt to heat but not as quickly as exercising people. The effect is transient and is retained for 2 weeks after last exposure, which proceeds to rapid lost of acclimatization over the next 2 weeks. The more fit an athlete is, the slower the loss of the adaptive benefits.

Clothing and equipment can be significant inhibitors of heat dissipation. Dry clothes soak up sweat and prevent evaporative heat loss. Dark clothes will cause radiant heat gains instead of loss. Football equipment prevents heat loss, thereby at a lower WBGT heat related problems can occur. Adjustments in practices are needed to protect against heat stress (8). These include using less equipment (such as helmet and shorts) during high WBGT and switching...
practices to early morning or after sundown when the WBGT is lower.

There is a greater potential for heat stress during a febrile illness. These conditions can produce dehydration. Fever will cause an increase in the core temperature and decrease the body's ability to compensate to the heat. Exercising with fever is a dangerous practice especially in the heat.

There are many prescription and over-the-counter medications that can augment the heat stress. Medications such as diuretics cause dehydration. Amphetamines and stimulants can increase metabolic rate and therefore heat production. Anticholinergic agents can decrease sweat production and therefore the evaporation of sweat and removal of heat. These medications should be avoided during exercise.

Women seem to have better thermoregulation than males. Variation in heat dissipation is noted over the menstrual cycle with a slight decrease in the luteal phase. This decrease does not appear to be clinically significant. Estrogen replacement therapy is falling out of favor for numerous reasons but it appears to give a minimal benefit in thermoregulation in postmenopausal women.

**Heat Cramps**

Heat cramps occur from involuntary contractions of exercising muscles. Calf muscles are by far the most common but any muscle in the body can be affected. The cause is presumed to be an electrolyte imbalance during exercise. However, there is evidence that a spinal neural mechanism may induce cramping that is unrelated to biochemical changes either in the blood or in the affected skeletal muscle (9). The best treatment is prevention with proper hydration, sodium, and calcium supplementation. The standard treatment of heat cramps involves rest, massage, passive stretch, and rehydration. Some people swear by the use of pickle juice or yellow mustard to relieve cramps. The reason for the anecdotal success of these two treatments is unknown but both are high in sodium.

**Heat Syncope**

The syncopal episode occurs in the elderly and the poorly acclimatized individual due to volume depletion, peripheral vasodilatation, and decreased vasomotor tone. The athlete is vasodilated with large pooling of blood in the leg muscles, the heart rate and cardiac output decreases, resulting in insufficient blood supply to the brain. This is made worse by dehydration or a poor cool down after running. Treatment involves elevating the legs (to increase preload and stroke volume), rehydration, and transfer to a cool shaded location. Cardiac and neurological causes need to be considered in the evaluation.

**Heat Exhaustion**

Athletes with heat exhaustion present with fatigue, weakness, piloerection, lightheadedness, headache, and neuromuscular incoordination. They typically have elevated rectal temperatures below 104°F (40°C). The problem is due to inadequate cardiac response to heat stress. Blood is shunted to the muscles and the skin and there is a lack of cardiac preload to maintain cardiac output. Athletes may develop neurological symptoms of headache, irritability, and mild confusion. Treatment is the same as heat stroke.

**Heat Stroke**

Heat stroke is an emergency that needs to be recognized early (10). The athlete will present with an elevated rectal temperature more than 40°C (104°F) and altered mental status. The classic heatstroke patient will present with dry hot skin without sweating. The heat stroke athlete may still be sweating. Obtaining the rectal temperature of anyone with neurological symptoms is essential. The success of treatment is in early recognition. Difference in symptoms between heat cramps, heat exhaustion, and heat stroke is listed in Table 21.2.

Exertional heat stroke can cause major organ system problems. Acute renal failure, rhabdomyolysis, and disseminated intravascular coagulation are more common in victims of exertional heat stroke than in victims of classic heat stroke (elderly during heat waves). Laboratory abnormalities can include elevated white blood cell count, elevated liver function tests, and hypokalemia in the initial stages with hyperkalemia manifesting later. Sodium levels can be normal or slightly elevated, depending on the hydration status of the patient. Patients can also have

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**TABLE 21.2**

**COMPARISON BETWEEN HEAT CRAMPS, HEAT EXHAUSTION, AND HEATSTROKE**

<table>
<thead>
<tr>
<th>Heat Cramps</th>
<th>Heat Exhaustion</th>
<th>Heatstroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated body temperature</td>
<td>Heat cramps, plus</td>
<td>Heat exhaustion, plus</td>
</tr>
<tr>
<td>Thirst</td>
<td>Nausea/vomiting</td>
<td>Anhydrosis</td>
</tr>
<tr>
<td>Sweating</td>
<td>Headache</td>
<td>Hyperventilation</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Malaise</td>
<td>Renal failure</td>
</tr>
<tr>
<td>Muscle cramps</td>
<td>Myalgias</td>
<td>Hepatocellular necrosis</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td></td>
<td>Pulmonary edema</td>
</tr>
<tr>
<td>Lightheadedness</td>
<td></td>
<td>Arrhythmia</td>
</tr>
<tr>
<td>Oliguria</td>
<td></td>
<td>Rhabdomyolysis</td>
</tr>
<tr>
<td>Uncoordination</td>
<td></td>
<td>Seizure</td>
</tr>
<tr>
<td>Irritability</td>
<td></td>
<td>Disseminated</td>
</tr>
<tr>
<td>Confusion</td>
<td></td>
<td>Intravascular</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coagulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Shock/coma</td>
</tr>
</tbody>
</table>

elevated creatinine phosphokinase (CPK) secondary to rhabdomyolysis. Patients who have peak CPK levels above 10,000 IU/L are at significant risk for the development of acute renal failure.

Acute reduction of the body temperature is the key to recovery and prevention of organ damage. This can be accomplished with ice water immersion or ice packs to the groin and axillary regions. Cool mist fans or alcohol rubs can aid in cooling. Unlike fever from infection in which toxins reset the hypothalamic set point, the elevation in temperature in this case, is due to excessive energy produced. Nonsteroidal anti-inflammatory medicines are not useful in these patients and should be avoided.

In a study of exertional heat stroke cases, ice water immersion lowered core temperature to less than 39°C (102.2°F) in 10 to 40 minutes (12). There were no fatalities in this group. In another study, ice water immersion cooled twice as fast as evaporative cooling (13). The author recommends rapid cooling using ice water immersion whenever possible.

The athlete with altered mental status may have a condition other than heat stroke such as infection, midbrain cerebral vascular accident, drug use, hyponatremia, and hypoglycemia. These other diagnoses need to be remembered when considering treatment. For example, the use of intravenous hydration (half normal saline) would be inappropriate for a runner with altered mental status caused by hyponatremia so obtaining a serum sodium level is important before rehydration is started.

In endurance athletes, hyponatremia can be found secondary to over rehydration with excessive free water. There are seldom mental status changes until the serum sodium level is less than 120 mEq/L. The athlete with a history of large fluid intake in a race or with a later finishing time (usually the less fit runners) is most susceptible. The athlete will have headaches, confusion, and seizures (rare). The same is seen in heatstroke and the rectal temperature is essential in differentiating. Normal temperatures and distal swelling (rings too tight) are reasons for further evaluation of hyponatremia.

Return to activity should be restricted for anyone with syncope, core temperature of greater than 39°C, and neurological symptoms. The person should aclimatize to the heat more and avoid high-intensity activities for 48 hours. Athletes with lesser heat illnesses need to be cooled and closely monitored. There is a higher risk for reinjury in athletes with prior heat illnesses.

**Precooling**

Prolonged exercise causes body temperature to rise; the increase in core temperature occurs rapidly and reaches steady state values when heat production matches heat loss. A critical limiting temperature exists; so to reduce the risk of cellular injury or heat related illness, athletes must reduce their exercise intensity so that exercise can continue. In hot conditions, the temperature gap between the start and the critical temperature is narrowed and the situation is exacerbated, often resulting in the termination of exercise. When exercising in high temperature environments, it would therefore be sensible to start exercise with a lower body temperature. Precooling (core body temperature cooling) before exercise would be expected to widen the temperature gap and delay the time before the critical limiting temperature is reached (14).

Studies confirm that increasing body heat is a limiting factor during exercise. Precooling is probably only beneficial for endurance exercise of up to 30 to 40 minutes rather than for intermittent or short-duration exercise (15–17). Studies show precooling before an endurance event may be worthwhile if the facilities are available. Precooling takes approximately 30 minutes to complete and can be done in either a swimming pool or a cold shower (18). Ice vests have been used and are more portable than immersion techniques. Precooling could be advantageous for endurance exercise in warm conditions (18).

**COLD STRESS**

**Physiology**

During cold weather, the body tries to maintain core body temperature while heat loss continues to occur due to conduction and convection mechanisms. The wind chill index is a measurement used to evaluate the effects of increased wind speed on heat loss. Wind increases conductive heat loss, therefore increasing cooling. A difference in ambient temperature and body temperature increases conduction heat loss. The loss of body heat by conduction is 25-fold greater in water than in air (19). A person in 20°C (68°F) water will become hypothermic depending on clothes and size of person in 2 to 40 hours whereas a dry person in 20°C (68°F) temperature on dry land would not become hypothermic.

In an attempt to maintain core body temperature, basal heat production increases slightly in the cold. Yet, it is not sufficient to maintain body temperature. A layer of subcutaneous fat is helpful to act as an insulator against the cold, but again the benefit is small. Intake of calories and exercise is important to generate sufficient calories to stay warm.

Voluntary and involuntary methods of heat generation are used to maintain core temperature in humans. The body will produce 3 to 6 times the basal amount of heat by involuntary shivering. Shunting of blood from the skin to the core also occurs to prevent heat loss. Thermoreceptors in the skin signal the hypothalamus to increase heat generation both voluntarily and involuntarily and to constrict peripheral circulation.

Voluntary exercise can generate significant heat. The body acts as a furnace keeping the core temperature in the narrow physiologic range. Approximately 75% of muscle contraction energy is released as heat. The amount of energy...
the body can generate diminishes as energy stores are used up. Mild to moderate intensity exercise produces sufficient heat and helps to keep the temperature in the physiologic range. It generates less heat than severe intensity exercise but longer duration exercise is possible. Mild intensity exercise often does not produce enough heat to prevent shivering.

As the body’s core temperature starts to drop, a cold-induced diuresis occurs. This occurs because the cardiac output increases and the systemic vascular resistance is higher. The kidneys receive an increase in blood flow and an increased amount of fluid is filtered. As the body’s core temperature drops lower, the renal perfusion decreases as cardiac output becomes less and less.

Hypothermia

Hypothermia is a seen in prolonged athletic events where the temperature is below 10°C (50°F), precipitation is occurring and wind speed is elevated. Most cases, in sports, will be mild but in extreme sports during winter, the severity of hypothermia can increase. Care needs to be implemented to maintain reheating of the individual.

Mild Hypothermia

Mild hypothermia is when the body core temperature is between 32.2°C and 35°C (89.6°F and 95°F) (20). The person will have confusion, slurred speech, impaired judgment, and amnesia. Cardiac output increases with tachycardia and increases preload secondary to the peripheral constriction. Shivering still occurs at these temperatures to stimulate heat. An athlete with confusion, dysarthria and ataxia with prolonged exposure to the cold should be considered to have hypothermia. A rectal temperature is needed to evaluate core temperature. It is useful to assess consciousness and shivering. A shivering conscious athlete probably has a core temperature above 31°C (87.8°F); whereas a severely disoriented or unconscious athlete not shivering is likely to have a core temperature below 31°C (87.8°F).

Moderate/severe Hypothermia

Moderate hypothermia presents with lethargy, disorientation, and unconsciousness. As the core body temperature lowers, cardiac output decreases and respiration is decreased and shallow. Before loss of consciousness, people have been known, paradoxically, to undress. This phenomenon is secondary to the loss of peripheral vasoconstriction and sudden warming of the extremity and sense of over heating. This severe impairment in judgment does not allow them to realize that this is a poor choice. Paradoxical undressing is a poor prognostic sign.

Severe hypothermia is often confused with death. The person has fixed dilated pupils, poor pulse that could be missed on palpation, severe muscular stiffness, and areflexia, even to noxious stimuli. Electrocardiography (EKG) may show asystole, ventricular fibrillation and other findings. The person without an EKG abnormality can have an arrhythmia triggered with sudden jarring. Careful assessment of the person with hypothermia is needed to guarantee no pulse before starting cardiopulmonary resuscitation (CPR). If possible, cardiac monitoring should be done.

Treatment

Treatment for hypothermia has common components for mild, moderate and severe forms. Further heat loss should be prevented (21). The person should have any wet clothes removed. Rectal temperature should be measured and monitored if possible. Prevention of worsening orthostatic hypotension is done by placing them supine. A barrier to the cold ground such as a sleep pad, blanket, or sleeping bag is needed to prevent conductive heat loss. All of these things should be done for every hypothermic patient.

The person with mild hypothermia needs to be kept warm, supplied with warm beverages and allowed to shiver. The shivering will effectively reheat the body. Exogenous rewarming is not needed. Actually, it may slow reheating. Heated humidified air is not going to add a significant amount of heat. In most cases, the person will shiver and this is the best option/action. Active external rewarming is needed in these patients once recooling can be prevented. The use of heat packs to the groin, axilla and neck, as well as feet in hot water or force heated air device (e.g., Bair Huggers, Arizant Healthcare Inc., Eden Prairie, MN) can be useful. Care is needed to avoid thermal burns.

For severe hypothermia, active internal rewarming is recommended. Warmed intravenous solution (normal saline) at 43°C (109°F), peritoneal lavage with warmed fluids and, if possible, warmed (42°C to 46°C [108°F to 115°F]) humidified oxygen during bag-mask ventilation is to be administered. If the victim is pulseless with no detectable signs of circulation, start chest compressions immediately. Follow advance cardiac life support principals (22).

Frostnip

Frostnip is what most people get when they go out in the cold and their nose, ears, toes, fingers, cheeks, or chin get white and cold. Often ice crystals will be seen on the surface of the skin. It occurs slowly and painlessly. Reheating occurs quickly and redness, pain, and hypersensitivity will develop over the affected area. There is no permanent damage after reheating. It is important to warm the affected area and keep the area warm to prevent further freezing and damage.

Frostbite

Frostbite is a problem in athletes exposed to freezing or near freezing weather such as those participating in orienteering, cross-country skiing, hiking, kayaking, skiing, ice climbing, and skating. Any sport performed outdoors can increase the risk for frostbite if the athlete is outdoors for prolonged
periods. Length of exposure is the most important risk factor in the severity of injury.

Injury occurs by three different and simultaneous pathways: tissue freezing, inflammatory mediator release and tissue hypoxia. The tissue forms ice crystals, and then intracellular dehydration and death occur. At first, something called the "hunting response" (21) occurs with constriction and then dilation of the peripheral vessels to keep the tissue oxygenated. Once the core temperature drops with prolonged cold exposure, the body closes off the peripheral blood supply to maintain core temperature. Tissue hypoxia followed by cell death occurs. The local tissue damage causes the release of inflammatory mediators such as prostaglandin F2 (PGF2), thromboxane A2 (TA2), and oxygen radicals. The release of these mediators reaches their peak with rewarming. A "secondary" zone of injury can cause worsening injury. Table 21.3 reviews the four stages of frostbite.

Clinically it can be divided into two stages.

1. Superficial Injury (first and second degree) is when there is only damage to the skin, no deep structures are involved and with no permanent damage.
2. Deep Injury (third and fourth degree) is when the structures below the skin are frozen. They are usually permanent injuries. The muscle or bones of the extremity involved. It is not possible to identify the extent of injury initially. It takes 22 to 45 days before the definite extent of permanently injured tissue is identified.

The extent of damage is not identifiable definitively in the first 3 to 5 days. The use of magnetic resonance imaging (MRI) or bone scintigraphy to assess the extent of tissue damage should be delayed for at least 14 days. Vascular studies (i.e., Doppler ultrasound) may be useful at 1 week. Care should be the same for each stage of frostbite at the initial stage.

It is important in the care of the athlete with frostbite not to warm the affected area and then allow it to freeze. As mentioned previously this allows excessive release of inflammatory mediators and subsequent further tissue damage. It would be better to leave the tissue frozen if prompt care cannot be instituted.

Care of the athlete in the hospital setting initially involves prevention of further damage by the three pathways. The affected area is rewarmed, usually in a warm water bath of 40°C to 42°C (104°F to 107.6°F). The mediators are blocked by the administration of a nonsteroidal anti-inflammatory agent. Blisters are drained and a potent topical anti-inflammatory agent, aloe vera, is applied every 6 hours. The person is prohibited from smoking to prevent further hypoxia and if possible, hyperbaric oxygen is used to decrease hypoxia. Prevention of tetanus and bacterial infection by the administration of tetanus toxoid and immunization, I.V. penicillin, and daily hydrotherapy with hexachlorophene is done. The patient is made comfortable with narcotic pain medication, as rewarming will cause severe pain. Elevation and splinting is used to prevent swelling.

In deep injuries, amputation may be needed even with the best care. These decisions should be delayed until 22 to 45 days, when the extent of necrotic tissue is clearer (23). Debridement should be limited unless infection spread cannot be stopped. It will not be initially obvious which tissue is viable and nonviable, so patience is needed to prevent excessive amputations. Swelling and edema may cause compartment syndromes and earlier fasciotomies are done when needed.

**Prevention of Cold Injury**

Clothing Units (CLO) is a measure of the insulator ability of clothing (19). Clothes help prevent conduction heat loss. One CLO is equal to the insulator effects of normal business attire at room temperature (see Table 21.4). An athlete performing intense exercise can maintain body temperature in cold weather. As the activity level decreases, more and more insulation is needed. The dry athlete in calm winds needs different clothes than the athlete in wind and rain. As clothes get wet, the water pulls the heat from the body. It is important to have a waterproof layer to stay warm. There are many synthetic clothes that act to keep warmth close to the body. Layering is important so that overheating does not occur and subsequent excessive sweating, causing clothes to become wet and decrease insulation ability, should be avoided. Wind protection is necessary to maintain heat retention because wind helps increase conductive heat loss. The body will spare peripheral tissue to maintain the core temperature. Therefore, it is important to ensure proper protection for ears, fingers, and toes.

**LIGHTNING INJURIES**

Lightning is responsible for more deaths in the United States than any other natural phenomena. The overall
**TABLE 21.4**
THE NUMBER OF CLOTHING UNITS NEEDED FOR THE RESPECTIVE APPARENT TEMPERATURE

<table>
<thead>
<tr>
<th>Activity Levels</th>
<th>20° C (68° F)</th>
<th>0°C (32° F)</th>
<th>−20° C (−4° F)</th>
<th>−40° C (−40° F)</th>
<th>−60° C (−76° F)</th>
<th>−80° C (−112° F)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>1</td>
<td>4</td>
<td>7.5</td>
<td>10.5</td>
<td>13.75</td>
<td>17</td>
</tr>
<tr>
<td>Walking 5 Km/hr</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Running 10 Km/hr</td>
<td>1</td>
<td>1.5</td>
<td>2.25</td>
<td>2.75</td>
<td>3.4</td>
<td>4</td>
</tr>
<tr>
<td>Running 16 Km/hr</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Note how the amount of clothing units greatly increases at rest but not when running, because of the heat generated during activity.

Mortality rate in lightning strike injuries from Colorado data is 1 in 10 (24). The morbidity rate in survivors approaches 70% (25). Two paramount rules to reduce the morbidity and mortality rates from lightning strikes during sport activities are as follows: (a) have a proactive plan that has been considered before undertaking the sports activity, and (b) seek and stay in a safe shelter until the lightning risk is gone. A safe shelter has been defined as any sturdy building that has been electrically ground by metal plumbing and/or wiring (26).

**Mountain Sports**

There is a high-risk time for lightning strikes in the mountains. An analysis of lightning related injuries showed that the injuries occur in July and August, in late morning to early evening. Therefore, the recommendation is that hikers should be off the mountain by 11 a.m. in high lightning risk periods (27).

Cold weather does not eliminate danger from lightning. Skiers are not immune from lightning strikes. Thunder snowstorms may occur from October to May but most are in the spring. In addition to the normal signs of lightning, during winter the skier should watch for the appearance of convective clouds, and Graupel (a type of precipitation referred to as soft hail or snow pellets). This precipitation is associated with lightning strikes and can be seen before the visualization of any lightning (28).

Skiing and hiking are not the only sports in which lightning can cause injury. Bicyclists should be aware that rubber tires do not provide protection from lightning. Many injuries occur from being thrown from the cycle due to the force of the lightning strike. Safe structures should be found during a lightning storm while cycling.

**Golf**

At times, lightning causes more mortality in golf in 1 year than any other cause in any sport. Some problems specific to golf are the paucity of safe shelters on many courses, and the habit of golfers to stand under single tall trees for protection. Open golf carts do not provide protection from lightning. Finding safe shelter is important to live to play another round. If suitable shelter cannot be found, individuals should squat with both feet together and cover their ears with their hands ("lightning position") to minimize risk of strike and acoustic trauma.

**Ball Field**

Lightning strikes on ball fields are more common in younger athletes. Dugouts are not considered safe protective structures from lightning strikes (28). Automobiles or homes should be sought or other closed structures such as those described previously.

**Water Sports**

As unbelievable as it may sound, many people do not realize that they need to leave the water during a thunderstorm. The cause of death from lightning strikes in water is not well defined. The proposed mechanisms include the normal electrical effects seen on dry land without an entrance or exit wound due to tissue cooling from the water. Death may also be caused by temporary paralysis and subsequent drowning. Kayaks and sculls are not safe structures for prevention of lightning injuries. A safe shelter needs to be found out of the water.

**Lightning Injuries**

There are three types of injuries:

*Minor injury*—Patients are conscious but confused. Most common symptoms are muscle pain and paresthesia, but no burns or paralysis. Complete recovery can be expected.

*Moderate Injury*—Signs of disorientation are present. Mottled extremities and an absence of peripheral pulses along with first and second-degree burns that appear over several hours are common. The tympanic membrane of the ear is often ruptured. These patients may have an incomplete recovery accompanied by chronic sleep problems and coordination difficulties due to neurological problems.
**Severe Injury**—Patients may present with ventricular fibrillation or systole. Prognosis in this group is uniformly poor unless CPR is immediate. Treatment of these patients should follow the basic advanced life support guidelines. Cervical spine injuries, secondary to the initial lightning strike, are possible and should be suspected in the unconscious person so that excessive movement of the cervical spine is avoided.

**Prevention**

Prevention of lightning strike injuries is the best treatment. It is important that available safe cover is readily accessible. Monitoring of lightning is important and can be accomplished by two methods: a lightning monitor, which measures static electricity in the air and gauges distance of lightning strikes, second is the count method in which the time from visual lightning strikes to thunder sound is recorded. The time in seconds is multiplied by 0.2 mi. A safe distance is greater than 6 miles. Therefore, there will be a gap of 30 seconds between flash and sound. For a flash to bang of 30 seconds all sporting activity should be suspended and safe cover should be found. Waiting 30 minutes or longer after the last flash of lightning or sound of thunder is recommended before athletic or recreational activities are resumed (29).

**Treatment**

If you are present at a lightning strike, it is important to first assess the airway, breathing, and circulation (ABCs) and initiate CPR if appropriate. The person who is unconscious should be considered to have a cervical spine injury. Once stabilized, the person should be safely moved to a medical center for observation. A complete blood cell count and urinalysis, including a test for myoglobinuria should be performed. An electrocardiogram is essential in all patients with lightning strike due to the common presence of QT prolongation after injury. The patient with severe injury warrants further diagnostic testing including electrolyte screen, blood urea nitrogen, and serum creatinine. Serial cardiac enzymes are indicated in the patient with moderate to severe injury. Other tests should be performed as warranted.

**WATER EXPOSURE INJURY**

**Near-drowning**

Near-drowning is the ingestion of water into the lungs from submersion/immersion. Near-drowning in lakes and unchlorinated waters is associated with inhalation of bacteria and pneumonia. Prophylactic antibiotics are not recommended (30). Cervical spine injuries should be considered in divers with near-drowning. Assessment of the basic ABCs is needed. Keep the person warm and comfortable. The near-drowning victim often suffers psychological trauma from the event.

**Drowning**

Drowning is often preventable; it is estimated that 80% of all cases are preventable (31). The new uniform definition of drowning—"the process of experiencing respiratory impairment from submersion/immersion in liquid," as was agreed upon during the World Congress on Drowning in Amsterdam, Netherlands, in 2002 (32) implies that respiratory insufficiency or respiratory failure may follow soon after the incident of immersion or submersion. The brain is the most vulnerable organ for asphyxia, and cerebral impairment occurs before cardiac problems in submersion. CPR should be attempted on drowning victims. Things to consider are cervical spine injury, brain damage, cardiac arrest, respiratory arrest, and hypothermia. The exact cause of brain damage is unknown but asphyxia is the most postulated cause.

**Barotraumas**

Barotrauma is injury to tissue when there is failure of a gas-filled space to equalize the internal pressure with the pressure in the surrounding environment. Barotrauma to the ears is the most common disorder in divers during decent. Barotrauma to the sinuses causing severe headaches is another. All organs can be affected by barotrauma with the lung being one of the most dangerous due to pneumothorax. Pulmonary barotrauma can produce an air-embolism, which needs immediate attention to reduce damage to the brain. The treatment of air-embolism is hyperbaric oxygen. The increased oxygen pressure forces nitrogen out of the body and equilibrates the gaseous environment. The nearest hyperbaric oxygen chamber should be located before diving.

**Decompression Sickness**

Decompression sickness (DCS) is defined as the release of inert gas bubbles (most often nitrogen) into the bloodstream and tissues after ambient pressure is reduced (33). This can generally be divided into two broad categories: (a) those due to physical injury as a result of an expansion of gas, and (b) those due to liberation of a gas phase in tissues. The gas in a space is reduced as a diver descends (at a 10 m descent the volume is half of that at the surface). The diver breathes compressed air mixture and the volume expands. As the diver ascends to the surface the gas in the lungs expands and needs to be released or physical injury will occur. Gas in tissues will be released and gas bubbles will develop in the blood if ascent is too rapid (34). Divers with a patent foramen ovale may have these bubble transported to the brain from the venous system (34). There are two main types of DCS. Type I is a mild form that is characterized by joint pains, urticarial rash, and pruritus. The incidence of DCS among recreational scuba divers is estimated to be one case per 5,000 to 10,000 dives. The problem usually resolves in a short period without treatment but observation is warranted (35). Type II is severe
Altitude changes affect the human body because of the relative hypoxic environment and decreased atmospheric pressure. These changes can be pathological or used for enhancing training (see Table 21.5).

### Altitude Training

The 1990s were the beginning of the popularization of the “live high–train low” strategy (35). This has led to the use of a number of new altitude training devices by athletes. These include the following: (a) normobaric hypoxia through nitrogen dilution (hypoxic apartment), (b) supplemental oxygen, (c) hypoxic sleeping devices, and (d) intermittent hypoxic exposure (IHE). Each method uses alterations in the F\(_{\text{O}_2}\) to stimulate changes in the body that could be advantages to competition.

A normobaric hypoxic apartment simulates elevations equivalent to 2,000 to 3,000 m. The barometric pressure within the apartment is equivalent to sea level. The Finnish “nitrogen-houses” are actually well-furnished hotel rooms, with nitrogen gas pumped into the rooms to decrease the F\(_{\text{O}_2}\) to approximately 15%. This simulates the living at high altitude and then training at “sea level,” when the athlete goes outside to the normal “higher” F\(_{\text{O}_2}\). Long term use of a normobaric hypoxic apartment (12 to 18 hours/day for 10 to 25 days) stimulates the release of serum erythropoietin (EPO) (37) and significantly increases reticulocyte count. Not all studies demonstrate this effect (38). These differences between studies may be the result of assessment methods, simulated altitudes, and the training status of the athletes.

There is evidence that peak cardiovascular function is reduced during maximal exercise in both acute and chronic hypoxia with no evidence for any primary alterations in myocardial function. Peak skeletal muscle electromyographic activity is also reduced during hypoxia. Both support a model in which a central, neural governor constrains the cardiac output by regulating the mass of skeletal muscle that can be activated during maximal exercise in both acute and chronic hypoxia.

Published data on the use of supplemental O\(_2\) training suggest that high-intensity workouts at moderate altitude and endurance performance at sea level may be enhanced through hyperoxic training utilized at an altitude over a period of several weeks (39). Training with supplemental oxygen is a modification of the “live high–train low” philosophy. The athlete is increasing their F\(_{\text{O}_2}\) while training compared to their living conditions. A study showed the efficacy of hyperoxic training suggesting that high-intensity workouts at moderate altitude (1,860 m) and endurance performance at sea level may be enhanced when supplemental oxygen training is used (40). The full benefit from hyperoxic training is not yet known. Much more research is needed.

IHE and Intermittent Hypoxic Training (IHT) allow athletes to “live low–train high”. IHE is based on the assumption that brief exposures to hypoxia (1.5 to 2.0 hours) are sufficient to stimulate the release of EPO, bring about an increase in red blood corpuscles (RBC) concentration. Athletes typically use IHE while at rest, or in conjunction with a training session. It is unclear whether passive IHE or IHT leads to improvements in hematological measures. In addition, there are minimal data to support the claim that IHE or IHT enhance \(\text{VO2}_{\text{max}}\) and endurance performance. Anaerobic power and anaerobic capacity may be improved as a result of IHT (41).

Hypoxic sleeping devices include the Colorado Altitude Training (CAT) Hatch (Boulder CO) (hypobaric chamber) and Hypoxico Tent System (Hypoxico Inc. New York, NY) (normobaric hypoxic system), both of which are designed to allow athletes to sleep high and train low. These devices simulate altitudes up to approximately 4,575 m and 4,270 m, respectively. They are similar to the hypoxic apartments but on a smaller scale. The chambers are cheaper than the hypoxic apartments but not equivalent. RBC production, maximal oxygen uptake, and/or performance in elite athletes do not appear to be affected by the use of these systems despite theoretical advantages (42).

### Acute Mountain Sickness

#### Pathophysiology

The Lake Louise Consensus Group (43) defined Acute Mountain Sickness (AMS) as the presence of headache and
one or more of the following symptoms: gastrointestinal (anorexia, nausea, or vomiting), insomnia, dizziness, and lassitude or fatigue, in an unacclimatized person who has recently arrived at an altitude above 2,500 m. Symptoms seem to correlate with decreased urine output. Altitude related illness is rare at altitudes below 2,500 m but is common in travelers above 3,500 m. The occurrence is increased by a rapid gain in altitude and reduced by a slow ascent, allowing time for acclimatization. Most (approximately 65%) people who develop AMS become symptomatic within the first 6 to 12 hours of arrival to that altitude. Acclimatization schedules of 600 m ascent above 2,000 m/day and preexposure of 5 or more days above 3,000 m in the last 2 months help reduce AMS.

The exact mechanism of AMS is unknown. Either hypoxia-induced cerebral vasodilatation or mild cerebral edema most likely produces the headache. The headache itself can cause nausea and malaise and thereby account for mild AMS. Impaired cerebral autoregulation, the release of vasogenic mediators, and alteration of the blood-brain barrier by hypoxia may also be important (44). Similar mechanisms are thought to cause cerebral edema at high altitude, which may represent a more severe form of AMS. Differences in individual susceptibility to AMS are striking and are not fully understood.

AMS may be confused with many other ailments but it responds to restful acclimatization within 1 to 3 days. Influenza-like illness, intracerebral hemorrhage, exhaustion, migraine-vascular headaches, dehydration, alcohol hangover, substance abuse, carbon monoxide toxicity, hypothermia, and even mild stroke are in the differential diagnosis especially if symptoms develop more than 1 day after ascent. Alcohol and sedative drugs should be avoided as they alter sleep patterns, decrease ventilation, and intensify hypoxemia. The most important reason to recognize AMS is that it may represent the first stage in the progression to severe HAPE (High Altitude Pulmonary Edema) or High-altitude cerebral edema (HACE) or both. HAPE and HACE are serious conditions (discussed later below): high-altitude illness is associated with rapid ascent above 2,500 m. Alaska climbers sleep at altitude from 3,000 to 5,300 m, a high incidence of HAPE and HACE (2% to 3%) is seen on Denali. Very high altitude, 3,500 to 5,500 m, is associated with extreme hypoxemia during exercise and sleep and is the most common range for severe altitude illness. At above 5,500 m altitudes, above the highest permanent human habitation, deterioration of physiologic function eventually exceeds acclimatization ability.

The three major determinants of developing AMS are 1) a prior history of AMS (increased odds ratio (OR) of 3.0), 2) preexposure (decreased OR 3.2), and 3) slow ascent (decreased OR of 3.0). There has been no significant association of AMS prevalence with gender or smoking. Most studies find no association between training status and likelihood of developing AMS. Most studies reported determinants of risk factors for AMS such as age and obesity; which were factors with an increased risk of AMS (38).

Prevention
For the prevention of high-altitude illness, the best strategy is a gradual ascent to promote acclimatization. The suggested guidelines are an increase of 600 m/day once above 2,000 m with an extra day added for acclimatization for every increase of 1,200 m. Some people need a slower ascent of 400 m/day. Some people would choose not to climb if they had to ascend that slowly (45).

Medications that aid in reducing AMS symptoms with rapid ascent are dexamethasone, acetazolamide, and ginkgo biloba (46). Acclimatization above 4,000 m, dexamethasone (8 and 16 mg) and acetazolamide (250 mg t.i.d.) are equally efficacious in preventing AMS when ascent rates are higher than 500 m/day. With low rates of ascent, prophylaxis is shown to be worthwhile. The usefulness of acetazolamide is demonstrated by the fact that fewer than three people need to use acetazolamide for one subject not to experience AMS as compared to placebo (46). Severe rebound illness can occur when dexamethasone is discontinued at high altitude and its use is controversial. For these reasons, dexamethasone should not be used immediately before or while ascending to a higher altitude. It should be reserved for treatment with descent or treatment at an altitude when there is an inevitable delay in descent. Meta-analysis showed lower doses of dexamethasone (0.5 mg or 2 mg) and lower doses of acetazolamide (500 mg) were not effective in preventing AMS (46). Others feel that acetazolamide (500 mg) is helpful and there needs to be a randomized control trial to find the optimal dose of acetazolamide (47,48). Ginkgo biloba has been used to prevent AMS. The prophylactic ginkgo dosage of 60 mg t.i.d. for 5 days decreases the incidence of AMS during gradual ascent (49). Ginkgo biloba is a supplement and in the United States caution needs to be used because quantity and quality are not guaranteed. None of the medications will prevent AMS in all people.

Treatment
AMS usually resolves in 2 to 3 days. The person should not increase their altitude and should be monitored for signs of HAPE or HACE. Aspirin (325 mg q4 hours) has been studied and it reduces the headache associated with AMS (50). If rest and symptomatic headache treatment are not effective, descent to lower altitude is indicated. Possibly, giving the patient acetazolamide (500 mg b.i.d.) as treatment can be helpful. Dexamethasone may be used if there is delay in descent or a worsening condition. Treatment with dexamethasone in an oral dose of 8 mg initially followed by 4 mg every 6 hours results in clinical improvement. Continuous use is necessary. Nausea symptoms can be treated with antiemetics. Descent and supplementary oxygen are the treatments of choice, and for severe illness, the combination provides optimal therapy. Astonishingly, only 500 to 1,000 m descent leads to resolution of symptoms in most cases of AMS. Simulated descent with portable hyperbaric chambers is also effective. With the use of these chambers at a pressure of 2 psi
(13.8 kPa), the equivalent altitude is roughly 2,000 m lower. For example, at altitude of 5,000 m being inside the bag is like being at 3,000 m (51). If descent is not possible, combination of hyperbaric chamber and dexamethasone is very useful. AMS treatment can start as watchful waiting but close monitoring is needed and descent of 500 m for 1 day may be helpful.

High-altitude Pulmonary Edema

Patients with HAPE typically presents with dyspnea, cough, weakness, and chest tightness within 1 to 3 days of arrival at altitude (52). Resting tachypnea, tachycardia, and rales are often present. Patients frequently describe a "gurgling" in their lungs. Physical findings include tachypnea, tachycardia, rales, and cyanosis. HAPE can occur rapidly, presenting with exertional fatigue, mild dyspnea, and cough, progressing to severe hypoxic coma overnight. Fatalities related to HAPE have been reported at altitudes as low as 2,440 m (43). HAPE is the most common cause of fatality at high altitude. It is caused by high pulmonary artery pressures that lead to a protein-rich and mildly hemorrhagic edema. HAPE is a form of hydrostatic pulmonary edema with altered alveolar-capillary permeability (53).

For many years, it was misdiagnosed as pneumonia occurring within several days of ascent in healthy young men. HAPE has become increasingly recognized. Many factors have been associated with HAPE including amount of exertion, individual susceptibility, rate of ascent, maximum altitude, and sedative drug use. However, a person’s physical conditioning before the climb does not seem to decrease occurrence. Gradual acclimatization as well as avoidance of sedatives and alcohol may lower the risk of HAPE. Salmeterol 125µg inhaled b.i.d. reduced episodes of HAPE in susceptible individuals (54).

To treat HAPE, immediate descent is essential; this may require medical evacuation. Oxygen saturation of 90% or more is important, which may require 4 to 6 liters/minute or more of supplementation. Nifedipine (10 mg) is used for reduction of the pulmonary vascular resistance and pulmonary artery pressure. Acetazolamide at 250 mg orally every 6 hours has also been used in acute disease. Dexamethasone, 8 mg orally for the first dose, then 4 mg every 6 hours can be used. The use of a portable hyperbaric chamber is recommended while awaiting descent.

High-altitude Retinal Hemorrhages

High-altitude retinal hemorrhages (HARH) occur at altitudes above 4,250 m and are common 5,500 m. They are usually asymptomatic. Nonsteroidal anti-inflammatory use, previous elevated intraocular pressure, rapid ascent, and exertion with hypoxemia are thought to be precipitating stresses (55). They are probably related to increased blood flow and retinal vessel dilatation. The hemorrhages usually resolve over 7 to 10 days. They go unnoticed unless they involve the macular area (56). Fundoscopic examination is recommended if other signs of altitude illness are seen (57). If vision is impaired or hemorrhages are severe, descent is advised.

High-altitude Cerebral Edema

HACE is a potentially fatal metabolic encephalopathy associated with exposure to the hypobaric hypoxia of altitude (58). Common symptoms include headache, ataxia, and confusion progressing to stupor and coma. Often AMS worsens to HACE or even HACE and HAPE. Focal signs may be seen in severe HACE. HACE can progress within hours but can also take days. Isolated HACE without HAPE occurs at higher altitudes. The mean altitude of HACE is 4,730 m versus 3,920 m when it is coupled with HAPE (59).

Hypertension is not believed to be a major factor in HACE, although minor blood pressure elevation is seen with rapid ascent. It has been postulated that there is a predominantly vasogenic edema mechanism related to a subacute hypobaric hypoxia component in severe HACE (60). This is secondary to a beneficial response to steroids in patients with HACE. One hypothesis that could account for many of the pathophysiologic features of HACE and provide a common link with HAPE is related to capillary leakage from hypoxia-induced endothelial damage.

The treatment of HACE is similar to HAPE except one main feature. Nifedipine is known to reduce pulmonary vascular resistance and pulmonary artery pressure in suspected HAPE. The difficulty is that it can lower systemic arterial pressure, which would impair the cerebral perfusion pressure in HACE; nifedipine should be avoided. Main treatment is immediate descent or evacuation. HACE is completely reversible with expeditious treatment. The use of 100% oxygen, if possible, should be administered until oxygen saturation can be monitored. Dexamethasone, 8 mg orally then 4 mg every 6 hours should be used and a hyperbaric bag can be helpful if immediate evacuation or descent is not possible. Recovery from HACE is not as rapid as from AMS or HAPE and may be prolonged or incomplete if descent or medical therapy is delayed.

JET LAG

The internal clock (circadian rhythm) of your body gets out of step when you cross several time zones over a short period. Jet lag can cause athletes to have trouble falling asleep, early rising, fatigue, irritability, difficulty concentrating, clumsiness, memory problems, weakness, headache, loss of appetite, and an upset stomach. These symptoms may last up to several days.

There are a number of aggravating factors. These include travel across three or more time zones, age or impaired health, lack of previous travel, sleep deprivation, dehydration, stress, alcohol use, and large meals. It is best to maintain good sleep habits, eat light, avoid alcohol
and maintain hydration when traveling. Traveling west to
east is more difficult than east to west because the actual
"biological clock" is slightly longer than 24 hours.

It is best to maintain a positive and relaxed attitude.
Pretravel changes may help prevent jet lag. Starting 3 days
before departure, begin adjusting your schedule (1 hour
a day) of eating, sleeping, and training to that of your
destination time. Set your watch to destination time at start
the start of the trip. Remain as active as allowed during
travel. Sleep on the plane at the appropriate time that is
in accordance with the time at the arrival destination. Use
pillows, blankets, eyeshades, earplugs, and recline during
sleep. Get your preferred seat (aisle, window, etc.) at
the onset of the flight. Avoid boredom and stress during travel
with relaxing books, music, and so on. You should avoid
overeating. Drink plenty of water to stay hydrated. Avoid
release caffeine taken in the morning (300 mg) has been
shown to help with daytime symptoms (63). Though there
are no studies specifically in athletes, zolpidem (Ambien),
a nonbenzodiazepine hypnotic medication, appears to be
helpful in decreasing jet lag symptoms in travelers. Two
studies looking at reducing effects of jet lag with eastward
travel have shown the medication to be helpful (64,65).

Melatonin is effective in preventing and reducing jet lag
symptoms. Five mg of melatonin from a reliable source
should be recommended to adult travelers flying across five
time zones. Although research shows mixed results, studies have
shown reduced performance in both the eastward and
westward direction with worse performance found in the
westward direction (61,62).

Medications can be helpful in adjusting to travel. Slow
release caffeine taken in the morning (300 mg) has been
shown to help with daytime symptoms (63). Though there
are no studies specifically in athletes, zolpidem (Ambien),
a nonbenzodiazepine hypnotic medication, appears to be
helpful in decreasing jet lag symptoms in travelers. Two
studies looking at reducing effects of jet lag with eastward
travel have shown the medication to be helpful (64,65).

Jet lag can cause detrimental effects in athletic perfor­
ance. These effects can be limited or eliminated with
proper planning and medication. The team physician can
play a significant role in the success of athletes traveling
long distances.

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