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AHC Media

Summer Emergencies: Heat Illness, Lightning Injuries, Drowning, and Sunburn

Introduction

The purpose of this review is to discuss some potentially serious summer emergencies — exertional heat-related illness, lightning injuries, and drowning — as well as a very common one — sunburn.

Exertional Heat-related Illness

Exertional heat-related illness is the physiologic insult to the body resulting from physical activity while exposed to elevated ambient temperatures. This stress can surpass the ability of the body to thermoregulate and may produce an elevation of the core body temperature.[1] Heat-related illness is distinguished from febrile emergencies in that the increase in temperature is caused by ambient heat rather than by a change in hypothalamic function.[1] The spectrum of heat-related illness ranges from mild — such as miliaria rubra — to severe — with a potentially fatal multi-organ dysfunction syndrome known as heat stroke.

Heat-related illness can be classified into classic and exertional. A common feature for both categories is exposure to elevated temperatures and inadequate thermoregulation; the difference is that with exertional heat illness, muscular activity, often vigorous, generates endogenous heat that contributes to the heat stress experienced by the individual, whereas in classic heat illness, muscular activity does not contribute.

Exertional heat stroke typically occurs in individuals participating in strenuous sports and those whose occupations lead to heat exposure during exertion, such as firefighters and military personnel. Classic heat stroke occurs among individuals who have an impaired physiologic mechanism of heat dissipation, stemming from comorbid metabolic or cardiac conditions, and those who lack the means to escape a hot environment due to economic, psychiatric, or social reasons.[1]

Heat-related illnesses are common in the United States and more common worldwide.[2] Heat-related illnesses are the most frequent cause of environmental injury treated in U.S. emergency departments.[3] In the past decade, the United States averaged more than 600 deaths annually associated with excessive heat exposure.[2] Heat-related illness is the leading cause of morbidity and mortality among U.S. high school athletes, with football being the most common associated sport.[2,4] The European heat wave of 2003 resulted in at least 70,000 fatalities. The annual pilgrimage to Makkah by Muslims (the Hajj) can, depending on the season, produce thousands of cases of heat exhaustion and hundreds of

EXECUTIVE SUMMARY

- The recommended treatment for exertional heat stroke is cold water immersion.
- Athletic competition should be cancelled if the WBGT is above 82.1°F.
- Sudden death from lightning strike is due to a combination of cardiac arrest (usually asystole) and respiratory arrest.
- Due to aspiration of fluid, airway maintenance and ventilation are more important in drowning victims in cardiopulmonary arrest compared to victims sustaining cardiac arrest due to coronary artery disease.
- A chemical sunscreen with an SPF of at least 30 or greater is recommended to patients with moderate to high susceptibility to sunburn.

cases of heat stroke.

Pathophysiology. Body heat is maintained by balancing internal production from metabolic processes with exchange to the external environment. Heat can be transferred between the body and the environment by four mechanisms: conduction, convection, radiation, and evaporation. As environmental heat stress and exercise intensity increase, evaporation of sweat at the skin surface becomes the predominant mechanism for body heat dissipation. The hypothalamus triggers sweating and increased cutaneous blood flow upon detection of elevation in core body temperature.[4] Hot temperatures reduce conductive heat exchange between the air and skin because of the small temperature gradient.[4] High humidity reduces the efficacy of sweating in releasing body heat.[4] Lack of air movement and wind minimizes convective heat dissipation during exercise in the sun.[4] All of these factors contribute to elevated heat stress.[4] Acclimatization takes 10 to 14 days of heat stress exposure exercise sessions that last 60-90 minutes per day.[4] This will produce the following changes: increase in plasma volume, sweat rates, cutaneous vasodilation, and aldosterone production, and decreases in urinary sodium excretion, sweating threshold, sweat electrolyte content, and heart rate at a given workload.[4]

Since evaporation is an important mechanism whereby the body transfers heat to the environment, the assessment of environmental heat stress must consider humidity as well as temperature. The heat index uses these two variables — temperature and relative humidity — to calculate an equivalent temperature perceived by an individual standing in the shade under those conditions.[5] The heat index does not consider the effect of wind, which may reduce or add to heat stress, nor the effect of solar exposure. A heat index above 90°F (32°C) is considered cautionary for strenuous activity. Another well-known and commonly used measure of heat stress is the wet bulb globe temperature (WBGT).[1] WBGT accounts for independent contributions of absolute temperature, humidity, wind, and solar radiation, and is a fairly precise predictor of human physiologic heat stress.[1,5]

Duration of exposure plays an important role in the occurrence of heat illness. Typically, heat-related mortality increases rapidly at the start of a heat wave, with some of its health impacts persisting up to three days after the maximum temperature peaks.[6]

Risk Factors for Heat Illness. The risk factors for heat illness can be divided into internal and external categories. The internal risk factors include: poor physical fitness level, lack of heat acclimatization (heat stroke is seen less among persons who live in countries where hot summers are common because of physiologic acclimatization and cultural adaptation to heat),[7] excessive or inappropriate clothing, protective equipment, very young or old age, inadequate sleep, excessive motivation, and poor education about heat illness.[4] Medical conditions that increase the possibility of environmental heat illness are obesity, recent febrile illness, hypertension, cardiovascular disease, diabetes insipidus, diabetes, and cystic fibrosis.[3,4,6] Diabetics are particularly vulnerable to extreme heat, with increased risk for adverse cardiovascular events related to heat.[6] Other patient populations at increased risk of adverse outcomes during extreme heat conditions are mental and behavioral patients.[9] Previous concussion with residual CNS dysfunction and prior history of malignant hyperthermia are also linked to an increased risk of environmental heat illness.[4] Medications and supplements associated with heat illness include stimulants, anticholinergics, antihistamines, benzodiazepines, alcohol, laxatives, cyclic anti-depressants, antipsychotics, and abused stimulant drugs.[4] Legal drugs, such as ephedra, methylphenidate, aspirin, and clopidogrel, also have been associated with an increased the risk of heat illness.[4] Caffeine may increase the risk of heat illness through diuresis, sympathetic nervous system stimulation, and an increase in the metabolic rate.[4]

External factors predisposing to heat illness include environmental conditions of high heat stress and inadequate access to fluids or shade.[4] Some studies have demonstrated that the incidence of heat illness is higher in rural areas, among low income communities, and among the uninsured.[10] Those aged 15 to 64 years had higher reported rates of ED visits with heat-related illness compared to those younger than 14 years and those older than 65 years. Conversely, excess hospitalizations occurring during a heat wave are usually among individuals older than 65 years of age.[3]

Heat Illness Syndromes. The classification of heat illness syndromes is based on the clinical signs and symptoms. In the milder syndromes of heat rash, head edema, heat-related syncope, heat cramps, and heat exhaustion, the body temperature is normal.

Heat Rash. Heat rash (miliaria rubra and/or "prickly heat") is a pruritic papulovesicular eruption over clothed areas.

Heat Edema. Heat edema produces mild edema in the dependent areas of the extremities that resolves with elevation and rest in a cool enviroment.[4]

Heat-Related Syncope. In heat-related syncope, a conscious athlete is unable to stand or walk without assistance because of lightheadedness, faintness, dizziness, or frank syncope. This condition characteristically presents at the completion

of a bout of strenuous physical activity, and accounts for up to 85% of medical visits to medical assistance tents at long-distance races.[4] There is an element of orthostatic hypotension, resulting in a decline in systolic blood pressure of at least 20 mmHg upon rising from a supine position to an upright posture.[4] Before confirming a diagnosis of heat cramps or heat syncope, heat stroke must be excluded by measuring a core temperature that is less than 40°C, absence of mental status changes, and normal sodium levels. The treatment consists of supine positioning with leg elevation 12-24 inches above the heart and oral hydration, if tolerated, or IV hydration in cases of vomiting.[4] Criteria for discharge include the ability to ambulate and normal mental status.

Heat Cramps. Heat cramps typically affect large muscles during or after exertion in the heat. Heat cramps are associated with excessive sweat loss. The individual is not necessarily overheated, and if they are, the core temperature does not exceed 40°C. The heat cramps can present initially with muscle twitches with progression to severe and widespread muscle spasms.[4] Muscle cramps are caused by excessive sodium loss through sweating, which is replaced with hypotonic fluids. Treatments for muscle cramps include rest, prolonged passive muscle stretching, salt supplementation through oral or IV fluids, salt-containing foods, and massage, with or without ice. The recommended oral hydration solution is a drink containing 3.5 g of sodium chloride in a quart of water (¼ to ½ teaspoon of table salt in water). Refractory muscle cramps may require benzodiazepines.[4]

Heat Exhaustion. Heat exhaustion is the most common form of environmental heat illness. It is characterized by cardiovascular insufficiency in persons who become dehydrated while physically active in conditions of heat stress.[4] Symptoms include nausea, vomiting, tachycardia, dizziness, muscle cramps, energy depletion, central fatigue, and syncope. Core temperature may be somewhat elevated, but is not higher than 40°C, and the mental status remains normal.[4] The initial treatment of heat exhaustion involves discontinuation of the activity, removal of clothing including sports equipment, relocation to a cool, shady area, and supine position with the lower extremities elevated.

Heat Stroke. Heat stroke is a life-threatening condition characterized by a core temperature above 40°C, central neurologic dysfunction, skeletal muscle injury, and multiple organ damage. Once initiated, the marked elevation of core temperature can become irreversible without aggressive body cooling measures because of thermoregulatory mechanism failure. Aural, oral, skin, temporal, and axillary temperature cannot confirm or exclude the diagnosis of heat stroke because these measurements do not reliably correlate with core temperature. Core temperature assessment, usually rectal, is essential to accurately confirm the diagnosis of heat stroke. Serum markers of hepatic (serum transaminases) and muscle (serum creatinine kinase and lactate dehydrogenase) injury are often elevated in heat-related illness with higher levels in heat stroke patients than in the non-heat stroke patients.[11]

Heat stroke is treated by first removing the victim from obvious sources of heat, shielding the patient from direct sunlight, removing clothing, and stopping exercise. The next step is to rapidly cool the patient. The most efficient mechanism to rapidly cool a patient with heat stroke is submersion in ice cold water accompanied by vigorous massage. This can be a challenge in the ED due to lack of tanks or tubs to perform this technique. In addition, submersion in cold water may be difficult in a severely agitated patient and contraindicated in an obtunded patient. Advanced life support monitoring (e.g., electronic cardiopulmonary monitoring) and treatment (e.g., intubation, mechanical ventilation, intravenous fluid resuscitation, and pharmaceutical administration) is likewise extremely difficult with the patient submerged in cold water. A more practical approach is to either cover the victim with ice water-soaked sheets or towels, keeping them continuously moist and cold, or, if the humidity is low and evaporative cooling is achievable, wetting the victim down followed by vigorous fanning.

Constant monitoring by medical personal is important. Don't be concerned by shivering, but recheck the temperature every 5 to 10 minutes so that when the patient has been cooled to 99.5°F to 100°F (37.5°C to 37.8°C), taper the cooling effort to avoid dropping the temperature below 98.6°F or 37°C. After the victim is cooled, recheck the temperature every 30 minutes for 3 to 4 hours. It is common to have a rebound rise in temperature.

As noted above, the elevated body temperature in heat stroke is not due to a reset in the hypothalamic "thermostat," so antipyretics (aspirin or acetaminophen) are contraindicated unless the patient also has an infection.

Treat with intravenous fluids as appropriate to restore intravascular volume status. Once the patient is alert, oral hydration can be used. Use oral fluids in which the concentration of carbohydrates or sugar in the beverage does not exceed 6% (e.g., commercially available low-sugar electrolyte drinks) to avoid inhibiting intestinal absorption. Encourage the patient to drink 1 to 2 pints of cool, sweetened liquid during the first few hours. Assume that for every pound of weight loss attributed to sweating, the patient should ingest a pint of fluid. Adequate rehydration can take up to 36 hours.[5]

Exertional Rhabdomyolysis. Exertional rhabdomyolysis is a syndrome of major muscle breakdown provoked by physical trauma of often unaccustomed physical exercise. Elevated body temperature, metabolic and electrolyte disturbances, and genetic defects contribute to its severity.[4] Serum creatinine kinase levels are dramatically increased. Clinical features include muscle pain, soreness, stiffness, and swelling, with progression to the loss of mobility and weakness.[4] Treatment is similar to that of other causes of rhabdomyolysis, with intravenous fluids to ensure strong urine flow to prevent renal damage

Hyponatremia. Exercise-associated hyponatremia is a potentially life-threatening condition characterized by a reduced serum sodium level (< 135 mEq/dL) and mental status changes.[4] It mostly affects endurance athletes who have ingested excessive quantities of hypotonic fluids during prolonged (generally more than 3 hours) physical activity. Early symptoms include vomiting, swelling of the hands and

Table 1. Activity Recommendations Based on Wet Bulb Globe Temperature (WBGT)

WBGT Temperature Range	Continuous Activity or Competition	Training or Non-continuous Competition for Non-acclimatized, Unfit, High-risk Individuals	Training or Non-continuous Competition for Acclimatized, Fit, Low-risk Individuals
≤ 65°F (18.3°C)	Generally safe	Normal activity, no precautions	Normal activity, no precautions
61.1-72°F (18.4-22.2°C)	Monitor high-risk individuals	Increase work:rest ratio, monitor fluid intake	Normal activity
72.1-82°F (22.3-27.8°C)	Monitor all competitors	Increase the rest:work ratio and decrease total duration of activity	Normal activity Monitor fluid intake
82.1-86°F (27.9-30.0°C)	Cancel activity	Increase the rest:work ratio to 1:1, decrease intensity and total duration of activity Limit intense exercise Watch at-risk individuals carefully	Plan intense or prolonged exercise with discretion Watch at-risk individuals carefully
86.1-90°F (30.1-32.2°C)		Cancel or stop practice or competition	Limit intense exercise and total daily exposure to heat and humidity Watch for early signs and symptoms
≥ 90.1°F (≥ 32.3°C)		Cancel activity	Cancel activity

feet, restlessness, confusion, wheezing, and fatigue. Symptoms may progress to seizures, pulmonary edema, cerebral edema, brainstem herniation, coma, respiratory arrest, and death. Factors that increase the severity of exercise-associated hyponatremia are a low absolute serum sodium value at the start of activity, rapid rates of sodium level decline, and longer duration of hyponatremia.[4]

Medical teams at such endurance events should be aware of the clinical manifestations of exercise-associated hyponatremia: overdrinking, nausea, vomiting, dizziness, muscle twitching, peripheral tingling or swelling, headache, disorientation, and acute mental status changes. Mild cases of hyponatremia can be treated with oral salt ingestion and rest in a cool environment, and avoiding additional water intake . More severe cases of exercise-associated hyponatremia should be managed with intravenous hypertonic saline, 100 mL of 3% saline fluid infused over a few minutes and immediate transfer to the emergency department.

Recurrence with subsequent endurance events is possible, so decisions about when it will be appropriate for the patient to return to training and competition should include establishment of individualized hydration protocols. Prevention is very important, and athletes who exercise in hot environments and sweat copiously should be counseled to consume adequate dietary sodium with meals, strive to correct salt losses accumulated during exercise, and pay attention to body weight changes, urine color, and thirst cues.

Prevention of Heat Illness. The best treatment of exertional heat illness is prevention. Several strategies are effective, such as education of coaches and conditioning staff regarding gradual heat acclimatization, slow introduction of new conditioning activities, maintenance of hydration status, and avoidance of exercise as punishment. Prompt recognition of heat illness with initiation of treatment can do much to prevent minor syndromes from developing into heat stroke. Education of the sideline staff, preparation with appropriate supplies, and creation of emergency action plans can do much to minimize the severity of exertional heat illness.

Dehydration can be avoided by drinking 1 pint of liquid 10 to 15 minutes before beginning vigorous exercise and drinking at least 1 pint to 1 quart of water during exercise associated with sweating in a hot climate. Electrolyte- and sugar-enriched drinks should be used when normal meals cannot be eaten or when sweating is excessive. Coffee, tea, and alcohol-containing beverages cause a diuretic effect and should be avoided.[5] Avoid "energy" supplements such as ephedra, which although it is purported to enhance athletic performance, increases the risk of exertional heat illness and, on occasion, deaths.

Acclimatization is attained by gradually increasing exposure to working in a hot environment for a minimum of an hour or two a day for 8 to 10 days for adults and 10 to 14 days for children.[5] Following activity recommendations based on the WBGT (*see Table 1*) is useful to prevent exertional heat illness.[5]

Season-appropriate clothing is recommended, with layers of clothing so they can be shed or added as necessary. The clothing material should be lightweight and absorbent. During activities in the sun, wear a loose-fitting, broad-brimmed hat to shield yourself from the sun. Conversely, do not wear a hat if there is no exposure to the sun during activity, as the hat reduces potential heat loss from the scalp and head from sweating.[5] Limit physical activity in the heat in individuals taking anticholinergics, antihistamines, benzodiazepines, cardiovascular drugs that lower cardiac output (beta blockers, calcium channel blockers), diuretics, cyclic antidepressants, or antipsychotics.

Exertional heat illness may first manifest with minor and nonspecific symptoms, but with continued activity, progress rapidly to heat stroke. Physicians should have a high index of suspicion when patients present after exercising or working in

Table 2. Types of Lightning Injuries

- Direct strike
- Contact
- Side flash
- Ground current (step voltage)
- Upward streamer (leader)
- Blunt trauma or barotrauma

high-temperature conditions, especially those with altered mental status. Heat stroke requires aggressive cooling, preferably at the site of first recognition. Once at the emergency department, a core temperature, usually rectal, should be measured.

Lightning Injuries

Lightning is one of the most powerful and spectacular natural phenomena.[12] The electrical current produced in a bolt of lightning can exceed 100,000 amperes (amps). It is estimated that lightning discharges are generated on the earth about 8 million times each day; of these, approximately one-fifth result in ground strikes. Estimated annual fatalities produced by lightning are about 24,000 internationally, and about 100 in the United States.[13-16] Despite the tremendous electrical current, most humans struck by lightning do not die; the number of human injuries caused by lightning is 10 times more than fatalities.[14] The chance of being struck by lightning is statistically very low; but the risk becomes much greater in those who frequently work or play outdoors. The majority of deaths reported in the United States in recent years have been associated with outdoor recreational activities.[16] Lightning fatalities are considered the second most common weather–related deaths after flooding.[12,16]

The risk of being struck by lightning is dependent on regional, seasonal, and temporal factors. Most lightning accidents occur outdoors, with occurrences in rural areas being the most common. Typical outdoor activities associated with lightning injuries are golfing, fishing, swimming, boating, camping, and hiking.[12,15] In the United States, the temporal distribution of lightning injuries is during the summer months, with the highest numbers during the months of June, July, and August. The greatest numbers of deaths occur in the afternoon between the hours of 2 pm to 6 pm. The states with the greatest number of lightning deaths are Florida, Texas, Maryland, Tennessee, and North Carolina.[12,15,16] Worldwide, the area with the highest number of lightning fatalities is Africa.[12]

Types of Lightning Injuries. Lightning can produce injury by several processes. (*See Table 2.*) A direct strike occurs when the victim is in the open, away from protection and is struck directly by the lightning discharge. This mechanism is the least common but the one with the highest mortality.[13,16,17]

A contact or indoor strike occurs when an electrical surge passes through an object to the victim in an indoor or otherwise secure location but in direct contact with that conducting object.[16] A side flash occurs outdoors when the victim seeks shelter beneath a tree or within a shelter that has not been grounded. A side flash may affect several members of the group if they are clustered together.[16] A lightning strike can produce a ground current, resulting in voltage differentials diminishing according to the distance from the strike. It is possible that the voltage differential between the feet separated by a human stride, termed step voltage, can generate an injurious electrical current from one leg to the other. In addition to a lightning bolt that descends from the clouds toward the earth, current flowing upward from the ground through the victim toward the clouds, termed an upward streamer, can be equally injurious. The sonic blast and pressure differential from the lightning strike can produce barotrauma, affecting primarily gas containing organs, such as the ears and lungs. Barotrauma is to the ears and other gas-containing organs can occur from the sonic blast and pressure differential that accompanies the lightning strike.[16]

Table 3. Cardiovascular Effects of Lightning Injuries

Asystole	Prolonged QT
Ventricular fibrillation	Atrial fibrillation
Cardiomyopathy	Elevated cardiac biomarkers
ST elevation	Pericarditis
Autonomic instability with labile blood pressure	

Pathophysiology. The effect of a lightning strike on the cardiovascular system is variable, ranging from benign electrocardiographic (ECG) changes to sudden death. (*See Table 3.*) Cardiac arrest is more commonly associated with a direct strike, whereas transient ECG changes are seen with contact strikes or step voltage ground current. Observed ECG changes include nonspecific T wave changes, ST elevation, and QT interval prolongation. Elevation of cardiac-specific biomarkers is common in survivors of direct strikes. Lightning-induced pericarditis and cardiomyopathy have been reported. Sinus tachycardia and atrial fibrillation are the two most common dysrhythmias in lightning strike survivors. Most of these findings resolve within three days, but pericarditis may recur several months after the initial injury.[14] Labile blood pressure and autonomic instability are possible after lightning strikes and may persist for weeks to months.[12,14,16,17]

The mechanism of sudden death from a lightning strike is simultaneous cardiac and respiratory arrest. The most common presentation is with asystole, but ventricular fibrillation may also be observed. In out-of-hospital cardiac arrests, asystole is an ominous sign and indicative of severe myocardial metabolic derangement with a poor chance of recovery. That is not so with lightning-induced asystolic cardiac arrests. Recovery is often possible, with return of spontaneous circulation preceding resolution of the respiratory arrest. Thus, respiratory support with airway management plays a bigger role in resuscitation of lightning-induced cardiac arrest.[14]

Neurologic injuries range from transient and incidental to life-threatening.

The transient neurologic symptoms with immediate onset include loss of consciousness, seizure, headache, paresthesia or weakness, confusion, and memory loss. Keraunoparalysis after a lightning strike is considered to be a result of overstimulation of the autonomic nervous system, leading to vascular spasm.[16] The clinical findings are lack of pulse, pallor and cyanosis, and motor and sensory loss in the affected extremities, affecting the lower extremities more than the upper extremities. It is important to check a central pulse before starting cardiopulmonary resuscitation because keraunoparalysis can mimic a pulseless victim.[12,16] The patient should be observed at the hospital even though the condition tends to resolve spontaneously.

Permanent neurologic symptoms with immediate onset include hypoxic encephalopathy resulting from cardiopulmonary arrest. Cerebral infarction, seizures, coma, cerebral edema, epidural or subdural hematoma, and intracranial hemorrhage also may be observed.[12,14,16,17] Delayed neurologic syndromes are progressive myelopathy, post-traumatic stress disorder, mood changes, insomnia, decreased libido, amyotrophic lateral sclerosis, paraplegia, quadriplegia, and regional pain syndromes.[12,14,16,17]

Dermatologic manifestations classic for lightning are transient "ferning" or "feathering," known as the Lichtenberg figures. These figures usually present within one hour of a lightning strike and resolve in less than 24 hours. Thermal or electrical burns associated with lightning injuries include linear, punctuate, or full-thickness burns. In survivors, full-thickness burns requiring grafting are uncommon.[14,16]

Ocular injuries can affect the anterior or posterior chambers. Injuries include corneal lesion, retinal detachment, loss of accommodation, hyphema, and optic nerve damage. The most common eye injury affects the lens, producing cataracts, often bilaterally. Cataracts can develop as soon as two days and as late as four years after the injury. Ophthalmology evaluation is essential for all survivors of a significant lightning strike.[14,16]

Audio-vestibular system injuries include tympanic membrane rupture, transient sensorineural deafness, and permanent hearing loss.[12,16,17]

Musculoskeletal injuries due to massive muscular contractions can produce fractures, shoulder dislocations, and rhabdomyolysis resulting in renal failure.[12,16,17] Serum creatinine kinase levels are often measured, but the level underestimates the extent of the damage.[12]

Lightning strikes in pregnancy are rare. In all the cases studied, the maternal mortality is zero, although the fetal mortality approaches 50%. The proposed reason for this mortality difference is that the fetus is surrounded by highly conductive amniotic fluid. Thus, pregnant women greater than 20 weeks of gestation with a lightning injury should undergo fetal assessment and monitoring for at least four hours.[14,17]

Treatment. The field treatment for a victim of a lightning strike should follow the "ABCs" for basic life support. The rescuer should assess the area for further hazards and transport the victim to a safe place. Resuscitation should be attempted on all lightning victims who appear lifeless because their chances of survival are better than most other victims of out-of-hospital cardiac arrest.[12,16] If the victim does not regain consciousness within 30 minutes of resuscitation, the prognosis becomes less favorable[16] even though extraordinary recoveries after prolonged resuscitation have been reported.[12] It is important that wide unreactive pupils not be interpreted as a sign of a poor prognosis or brain death.[12] In instances of multiple lightning casualties, using a reverse triage strategy is recommended, with priority given to those individuals without vital signs or spontaneous respirations.[14]

Once the patient has reached the hospital, injuries should be assessed and treated. Patients complaining of chest pain or dyspnea and should have an ECG and echocardiogram. Cardiac biomarkers have limited clinical utility.[16] The decision for hospital admission is primarily based on the injuries identified. For patients who do not have such injuries, consider admission to telemetry for 24 hours for victims who have experienced a direct strike and those with an abnormal ECG or echocardiogram, as these patients may develop delayed cardiac injury.[16] Discharged patients should be instructed to return immediately if they develop chest pain or dyspnea.[16]

Prevention. When should you seek shelter in a lightning storm? A useful tool is the 30-30 rule, which says that you should be inside a safe structure when thunder is heard within 30 seconds of seeing lightning (approximately 6 miles away), and activity should not resume for 30 minutes after the last thunder is heard or the last lightning is seen.[16] Seek shelter in the largest enclosed building available, and away from doors and windows. Another option is to be inside a metal-topped vehicle with windows and doors closed. If you are caught in a storm in the wilderness, seek shelter inside a deep cave, far into the dense forest, or in a deep ravine.[14]

If the lightning strike is imminent and you cannot reach the above mentioned shelter, assume the lightning position. This position consists of sitting or crouching with knees and feet close together, with the heels elevated to create only one point of contact with the ground.[14] When in a group, the group members should separate by more than 20 feet to prevent mass casualties.[14]

Like other aspects of modern life, technology is helping to track lightning storms and prevent lightning injuries. Numerous commercial services are available in the United States that can provide automatic notifications when nearby lightning is detected by the National Lightning Detection Network (NLDN). These services then transmit notices by e-mail, text, or cell phone to alert subscribers. There are also personal lightning detection devices sold with product names such as StormPro ® and Skyscan®. These devices are about the size of a cell phone and can detect lightning as far away as 75 miles.[14]

When in the open wilderness or hiking and/or climbing, avoid the peaks and ridgelines in the afternoon. In the water, exit the water and seek shelter. When rafting or kayaking, head to the shore and move inland away from the water's edge. When in open water, seek shelter below the deck after locking off the helm. If no below deck shelter is available, tie into a lifeline.[14]

Drowning

Throughout the ages, a traditional summertime activity — water recreation — has led to many tragedies. In temperate climates, the number of drowning deaths increases dramatically during the summertime months, especially when the temperature exceeds 30˚C.[18] There are three connected and obvious reasons that could explain this phenomenon: as the outside temperature rises, people are more likely to be exposed to water; alcohol is a large risk factor for drownings, and individuals are more likely to drink alcohol when they are outside during hot days; and the use of a floatation device may decline on days when the weather is warmer since individuals are more likely to submerge themselves in the warmer water.[18] The majority of drownings in people younger than 15 years of age occur between May and August every year.[19] For every death due to drowning, up to nine other individuals sustain injury from submersion in water.

The World Congress on Drowning in 2002 defined drowning as "the process of experiencing respiratory impairment from submersion/immersion in liquid."[20] This definition embodied a change in thinking, with the concept that drowning was a process, not an outcome. This definition replaced the previous one that defined drowning as "death resulting from suffocation within 24 hours of submersion in water." The World Congress also made recommendations to replace many terms used to describe fatal and nonfatal processes related to submersion. These definitions have been endorsed by the International Liaison Committee on Resuscitation (ILCOR) and the World Health Organization.[21] (*See Table 4.*)

According to 2011 data from the World Health Organization, drowning is the third leading cause of death from unintentional injury worldwide, with approximately 500,000 people dying from drowning yearly.[20,22] The Centers for Disease Control and Prevention reports an average of 3500 deaths yearly in the United States from unintentional drowning, which are more likely to happen on a weekend than a weekday.[20] Also, the second most common cause of death in the United States of young children (ages 1 to 4 years) is unintentional drowning.[23]

Table 4. Terminology and Definitions

Approved	
Drowning	Drowning is the process of experiencing respiratory impairment from submersion or immersion in liquid.
Non-fatal drowning	Survival after drowning
Non-fatal drowning with morbidity	Survival with physiologic impairment
Non-fatal drowning without morbidity	Survival without physiologic impairment
Fatal drowning	Death due to drowning
Accepted	
Submersion	The whole body is under water.
Immersion	Part of the body is covered in water (for drowning to occur, the face and airway would have to be immersed).
Witnessed	Drowning episode is observed from the onset of immersion or submersion. Replaced the term "active drowning"
Unwitnessed	Victim found in water, no one saw the event. Replaced the term "passive drowning"
Abandoned	
Dry and wet drowning	These terms are not useful and are considered redundant. All drownings are wet by definition, as they occur in liquid, and most drowning incidents have water aspirated into the lungs. It is impossible to tell at the scene whether water has been aspirated into the lung, and does not influence management.

Risk Factors. There are multiple factors that place individuals at a higher risk for drowning, and many are avoidable or can be ameliorated. Alcohol intake, reckless behavior, lack of close responsible adult supervision, and inadequate swimming ability all contribute to the burden caused by drowning.[18] Absent barriers around pools, failure to wear life jackets while boating, and inadequate water safety education also contribute. Although uncommon, increased risk has been associated with cerebrovascular disease and cardiac dysrhythmias, including prolonged QT syndrome.[24] Medical conditions associated with periods of unconsciousness, such as hypoglycemia (from insulin use in diabetes mellitus) or epilepsy, can lead to increased chances of drowning during episodes of unconsciousness.[23] Lastly, trauma from falls or boating accidents can lead to cervical spine injury resulting in paralysis and drowning.

Pathophysiology. The process of drowning begins with submersion or facial immersion. In general, the victim usually panics and tries to hold their breath, which is soon followed by involuntary diaphragmatic contractions.[25] Next, laryngospasm occurs, which can initially be caused by even small amounts of aspirated liquids. Eventually, laryngospasm relaxes, eventually leading to major aspiration if the victim is still submerged.[23] This period of laryngospasm prevents ventilation and leads to hypercapnia, hypoxia, and respiratory acidosis.[25] Concurrently, large amounts of fluids are often swallowed down the esophagus into the stomach. On average, for every minute that passes, the PaO_2 drops by 6 mmHg, eventually leading to cerebral hypoxia.[25]

The respiratory and cardiovascular organ systems are the predominant ones affected by the process of drowning. Aspirated fluid down the tracheo-bronchial tree impairs the lungs' ability to ventilate due to bronchospasm, atelectasis, aspirated material filling the alveolar space, and pneumonitis.[23] Ventilation

to perfusion mismatching is common, as well as intrapulmonary shunting due to blood flowing through nonventilated portions of the lungs. Similar to other systemic insults, drowning can lead to hypoxemia that has a great impact on the cardiovascular system, leading to cardiac dysfunction and arrhythmias.[23]

Multiple factors impact the prognosis of drowning victims, ranging from individual factors such as age and various other health comorbidities, to event factors such as whether the drowning accident was witnessed, the temperature of the water, whether the victim was unconscious when rescued, whether there was cyanosis upon initial presentation, and whether the patient showed any signs of life during the rescue.[25] In general, the prognosis depends on how long the victim was submerged and the resulting hypoxia. In instances of witnessed drownings, the victim tends to have been submerged for a shorter period than in unwitnessed instances of drowning.

In the past, there was thought to be a difference in morbidity and mortality depending on the chemical composition of the water in which the victim drowned. Most commonly, this difference depended on whether the drowning occurred in fresh water or salt water. However, it now is understood that the impact of the water aspirated is dependent on the quantity of water and not the electrolyte composition of the water, as previously thought. Regardless of the electrolyte content of the water, aspirating 1 to 3 mL/kg of bodyweight of the fluid will cause extensive damage to the integrity of the lungs, including alveolar collapse brought on by damage to pulmonary surfactant, intrapulmonary shunting as discussed earlier, and ventilation and perfusion mismatch.[26]

Treatment. Resuscitation methods are very similar in drowning victims as in patients experiencing cardiopulmonary arrest. However, drowning patients have often swallowed large amounts of fluid, and they are more likely to vomit and aspirate their fluids during rescue ventilation and chest compressions.[25] In 65% of individuals requiring rescue breathing alone and in 85% of individuals requiring full CPR, victims regurgitate their stomach contents. Regurgitation often leads to aspiration of the victim's own stomach contents.[22] Thus, protecting and securing the airway is important during resuscitation of a drowning victim.

Pre-hospital care is crucial to the recovery of drowning victims. Upon arriving at the scene of a drowning, remove the victim from submersion as quickly as possible, and make every effort to begin CPR immediately. If the patient is unconscious, not breathing, and pulseless, place the patient in the supine position and initiate standard CPR immediately. When the patient is unconscious but has a pulse and is breathing, place the patient in the lateral decubitus position. For individuals who are not breathing, initiate rescue breathing. For individuals who are conscious, evaluate the patient for pulmonary impairment, and if present, begin treatment with supplemental oxygen. If the victim begins to tire out or is not able to ventilate and oxygenate properly, and ALS personal are present, intubate the patient and start positive end-expiratory pressure to prevent further clinical deterioration.[22]

In victims of drowning associated with head-first diving, falls, or boating accidents, perform cervical stabilization until cervical spinal injury can be evaluated at the hospital. A cohort study found that only 0.5% of submersion victims had sustained cervical spine injuries.[27] A common feature was an associated mechanism with potential for cervical spine injury — including a fall from a great height, a diving injury, or a motorized vehicle accident — and external signs of trauma and a GCS score of less than 11. Accordingly, cervical spine immobilization is not appropriate, nor needed, just for standard submersion cases.

Upon arrival to the emergency department, place the patient on a cardiac monitor, and assess and initiate continuous pulse oximetry and establish IV access, if not previously done. Once the patient's airway, breathing, and circulation have been stabilized, place a gastric tube for decompression in unresponsive patients. Perform a more detailed physical examination, chest radiography, and consider obtaining an arterial blood gas. If the patient is still unconscious, evaluate for structural, metabolic, or toxicology causes, with ancillary tests such as a CT scan of the head and neck, a toxicology screen, glucose check, and administration of naloxone. Sometimes, an immediate chest X-ray will not reveal any signs of pulmonary injury, but several hours to days after the initiating event, further deterioration of the patient can occur, leading to acute lung injury (acute respiratory distress syndrome). More often, the patient who will suffer pulmonary damage has evidence of neurologic or respiratory injury on arrival.[25]

The outcome for individuals who experience near-drowning is very good for those who have a good neurological function after the initial resuscitation, even in the presence of increased age, acute respiratory failure, or comorbidities; the overall mortality is as low as 2.3% in those with intact neurologic function after initial resuscitation.[28]

Near-drowning and drowning can be classified into six severity grades.[29] Grade one is normal pulmonary auscultation, although the patient may be coughing. Grade two is abnormal pulmonary auscultation with rales in some pulmonary fields. Grade three is pulmonary auscultation of acute pulmonary edema without arterial hypotension. Grade four is pulmonary auscultation of acute pulmonary edema with arterial hypotension. Grade five is isolated respiratory arrest. Grade six is cardiopulmonary arrest. Severity grading of the near-drowning and drowning helps to determine the disposition of the patient. In general, those who are classified as a grade two through six should be admitted to the hospital for further evaluation. Those with a grade two can often be given noninvasive oxygen and sent home within 24 hours. Patients who are grades three through five need mechanical ventilation for support.[29]

The most important aspect of preventing the mortality and morbidity brought on by drowning is prevention. Prevention includes restricting access to water for young children and ensuring that those with access to pools or with their own pools are trained in CPR. Public education about water safety is critical to the prevention of near-drowning and drowning.

Sunburn

Sunburn is caused by overexposure to

ultraviolet radiation (UVR), which can be from multiple sources, but is most commonly caused by exposure to the sun. The ultraviolet radiation can lead to inflammation, DNA damage and cancer, premature aging, and even immunosuppression.[30] Sunburn is very common; one study from 2004 found that 69% of surveyed individuals sustained a sunburn that year.[31]

Pathophysiology. The UV spectrum is made of electromagnetic radiation that ranges from 200 to 400 nm in wavelength.[30] UV radiation is subdivided into UVA (320 to 400 nm), UVB (290 to 320 nm), and UVC (200 to 290 nm). UVC radiation is the most energetic, but fortunately most of the UVC radiation from the sun is absorbed by the ozone level, and very little reaches the earth's surface. UVB radiation undergoes some atmospheric absorption, so UVB intensity at the surface is greatest during periods when the sunlight path through the atmosphere is shortest, typically 10 am to 4 pm, depending on season and location. UVB radiation typically does not penetrate much beyond the epidermis, where it can induce inflammation and erythema. UVA radiation more readily passes through the atmosphere and penetrates to the deeper layers of the dermis. UVA has little involvement in causing the erythema, but mainly is associated with tanning, connective tissue degeneration, and immunosuppression.[32]

Clinical Features. The physical signs of sunburn include erythema and pain, often beginning three to four hours after being exposed to sunlight for an increased length of time, and reaches a peak at 12 hours.[33] These signs are the result of inflammation brought on by the UV exposure and are caused by vasodilation of the cutaneous blood vessels. Prostaglandin and leukotriene synthesis is brought about by the release of preformed mediators including histamine, serotonin, and tumor necrosis factor from mast cells.[34] DNA damage occurs within the epidermal cells, leading to apoptosis. Depending on the degree of sunburn, over a period of four to seven days, the erythematous skin begins to either fade or blister and peel.

Sunburns, like other burns, can be categorized by degrees, depending on their depth. (*See Table 5.*) First-degree sunburns are pink or red, painful to the touch, and limited to the epidermis. Second-degree burns can be categorized further depending on whether they are superficial or deep partial-thickness. The superficial second-degree burns form blisters, are erythematous, painful, and blanch upon palpation. This wound is limited to the superficial dermis. Conversely, deep second-degree partial-thickness burns range between white and erythematous areas, with areas that blanch less, and extend throughout the epidermis into the dermis. Although a third-degree sunburn occurs rarely, it appears stiff and white or tan, does not blanch at all, and is painless, compared to the first- and second-degree burns. Third-degree burns extend fully through the dermis.[24]

Table 5. Sunburn Depth Classification

Classification	Appearance	Sensation
First degree	Pink or red	Painful
Superficial second degree	Pink, clear blisters	Painful
Deep second degree	Pink, white areas, blisters	Painful
Third degree	White, tan	Painless

Table 6. Fitzpatrick Skin Typing Method

Skin Type	Susceptibility to Sunburn	Constitutive Skin Color	Facultative Tanning Ability
I	High	White	Very poor
II	High	White	Poor
III	Moderate	White	Good
IV	Low	Olive	Very good
V	Very low	Brown	Very good
VI	Very low	Black	Very good

Treatment. Treatment in the emergency department for mild sunburns that are categorized as first-degree include soaking the skin with cool compresses to bring some mild relief of the discomfort. In addition, some symptomatic treatment with NSAIDs for reduction of inflammation and pain can be helpful.[33] Aspirin, widely known to be an analgesic and anti-inflammatory, has been proposed as topical therapy to help reduce the UV radiation-induced cellular changes brought about by the sun.[35] Topical diclofenac gel may bring a significant reduction in pain and erythema arising from first-degree sunburns.[36] It is sometimes recommended that topical anesthetics should be limited to lidocaine, since benzocaine can serve as a sensitizer.[32] However, 10% benzocaine was found to be superior to control ointment for the relief of pain from mild sunburn.[37]

The use of oral or topical corticosteroids for treating sunburns has been somewhat controversial. Despite a theoretical basis for benefit, there is no good evidence about their usefulness in symptomatic treatment or recovery time. In very severe sunburns, as in other types of burns, fluids should be given to replace water loss. Antihistamines, emollients, or NSAIDs can be used for symptom reduction, but they do not reduce the recovery time for sunburns.[38]

Prevention. The first step to avoiding sunburn is to stay out of the sun. It is advised by the American Cancer Society to avoid being outside during the midday when UVR exposure is at its peak, to wear sunscreen and protective clothing to prevent exposure, and, if possible, to stay in the shade when outside.[39]

Several types of medications can increase the skin's sensitivity to the sun and cause phototoxic reactions. The most well-known medications to cause these

reactions include tetracycline, retinoids, and antifungals. Other categories of medications that are not as well-known include NSAIDs, neuroleptic medications, and certain diuretics such as furosemide and hydrochlorothiazide.[32]

Types of available sunscreens include physical sunscreen, those that scatter and reflect light, and chemical sunscreens that absorb radiation. One commonly used physical sunscreen is zinc oxide cream. Most liquid sunscreens use chemical absorbants.[32] Chemical sunscreens are graded by their Sun Protection Factor (SPF), which is primarily determined by their ability to absorb UVB radiation. As a general rule, the SPF determines how much longer a person can be exposed to the sun before burning, compared to their baseline without the sunscreen. For example, if a person would burn in 10 minutes of sun exposure, an SPF 15 sunscreen would extend that time to 150 minutes before burning. Obviously, factors related to sunscreen application may also influence the protection provided. There is no uniformly accepted measure for UVA protection. Chemical sunscreens are required to incorporate agents with UVA absorption (such as oxybenzone or avobenzone), but the SPF does not measure protection from UVA radiation. In the emergency department, these methods of protection can be discussed with the patient to help prevent future skin damage.

Those who are most at risk for damage from UV radiation can be determined using the Fitzpatrick Skin Type Method. This gives an idea about how likely an individual is to burn and experience damage from the sun. An online quiz by the Skin Cancer Foundation can help the patient determine their type and risk for sun damage (http://www.skincancer.org/prevention/are-you-at-risk/fitzpatrick-skin-quiz). For individuals with Fitzpatrick Skin Types 1 through 3, a chemical sunscreen of SPF 30 or greater is recommended.

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CME Questions

1. Which of the following methods of temperature measurement is recommended for the diagnosis of heat stroke?
   A. axillary
   B. temporal
   C. tympanic
   D. oral
   E. rectal
2. Which statement regarding cooling treatment for heat stroke is true?
   A. It should be delayed until the patient arrives to the emergency department.
   B. Cooling should be initiated immediately at the scene.
   C. The best method for rapidly cooling the patient with heat stroke is with ice packs.
   D. none of the above
3. At what WGBT temperature should competition or continuous activity be cancelled?
   A. at or above 72.1 degrees
   B. at or above 78.1 degrees
   C. at or above 82.1 degrees
   D. at or above 86.1 degrees
4. Of the following injuries from a lightning strike, which one has the worst prognosis?
   A. asystole
   B. respiratory arrest
   C. keraunoparalysis
   D. cataracts
5. In the wilderness, what is the best option for shelter?
   A. under a tall tree
   B. just under the ridge line
   C. in a deep cave
   D. in the dense forest
6. A 27-year-old female with no with a history of acne presents to the emergency department complaining of painful erythematous skin with blisters. She spent the day at the beach seven hours prior. What level of sunburn does she have?
   A. first degree
   B. superficial second degree
   C. third degree
   D. fourth degree
7. The same 27-year-old patient described above is getting ready to be discharged from the emergency department. She asks if there is anything she can do for pain relief at home. What is the best option to help relieve some of her discomfort?
   A. Nothing will help for symptomatic relief.
   B. oral corticosteroids
   C. topical corticosteroids
   D. NSAIDs
8. What is the current definition of drowning?
   A. death resulting from the suffocation within 24 hours of submersion in water
   B. aspirating fluids that leads to hypoxia causing cardiac standstill
   C. submersion of an individual into fluids that causes them to become incapacitated
   D. the process of experiencing respiratory impairment from submersion/immersion in liquid
9. In near-drowning victims, which factor has the most effect on the pulmonary morbidity caused by the aspirated fluids?
   A. the quantity of aspirated fluids
   B. the quality of aspirated fluids (salt water vs. fresh water)
   C. the temperature of the aspirated fluids
   D. the age of the victim
10. A 57-year-old male with a past history of an MI and COPD is brought to the emergency department after a near-drowning. His vitals are T 37, P 88, BP 158/92, and O2 sat 94%. On physical examination, you hear rales in some pulmonary fields. What is the severity grade classification?
    A. grade I
    B. grade II
    C. grade III
    D. grade IV

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Correction

In the Executive Summary of the March 8, 2015 issue, the phone number for the National Human Trafficking Resource Center should be 1-888-373-7888.

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EMERGENCY MEDICINE **REPORTS**

Summer Emergencies: Heat Illness, Lightning Injuries, Drowning, and Sunburn

Activity Recommendations Based on Wet Bulb Globe Temperature (WBGT)

WBGT Temperature Range	Continuous Activity or Competition	Training or Non-continuous Competition for Non-acclimatized, Unfit, High-risk Individuals	Training or Non-continuous Competition for Acclimatized, Fit, Low-risk Individuals
≤ 65°F (18.3°C)	Generally safe	Normal activity, no precautions	Normal activity, no precautions
61.1-72°F (18.4-22.2°C)	Monitor high-risk individuals	Increase work:rest ratio, monitor fluid intake	Normal activity
72.1-82°F (22.3-27.8°C)	Monitor all competitors	Increase the rest:work ratio and decrease total duration of activity	Normal activity Monitor fluid intake
82.1-86°F (27.9-30.0°C)	Cancel activity	Increase the rest:work ratio to 1:1, decrease intensity and total duration of activity Limit intense exercise Watch at-risk individuals carefully	Plan intense or prolonged exercise with discretion Watch at-risk individuals carefully
86.1-90°F (30.1-32.2°C)		Cancel or stop practice or competition	Limit intense exercise and total daily exposure to heat and humidity Watch for early signs and symptoms
≥ 90.1°F (≥ 32.3°C)		Cancel activity	Cancel activity

Cardiovascular Effects of Lightning Injuries

Asystole	Prolonged QT
Ventricular fibrillation	Atrial fibrillation
Cardiomyopathy	Elevated cardiac biomarkers
ST elevation	Pericarditis
Autonomic instability with labile blood pressure	

Types of Lightning Injuries

- Direct strike
- Contact
- Side flash
- Ground current (step voltage)
- Upward streamer (leader)
- Blunt trauma or barotrauma

Terminology and Definitions

Approved	
Drowning	Drowning is the process of experiencing respiratory impairment from submersion or immersion in liquid.
Non-fatal drowning	Survival after drowning
Non-fatal drowning with morbidity	Survival with physiologic impairment
Non-fatal drowning without morbidity	Survival without physiologic impairment
Fatal drowning	Death due to drowning
Accepted	
Submersion	The whole body is under water.
Immersion	Part of the body is covered in water (for drowning to occur, the face and airway would have to be immersed).
Witnessed	Drowning episode is observed from the onset of immersion or submersion. Replaced the term "active drowning"
Unwitnessed	Victim found in water, no one saw the event. Replaced the term "passive drowning"
Abandoned	
Dry and wet drowning	These terms are not useful and are considered redundant. All drownings are wet by definition, as they occur in liquid, and most drowning incidents have water aspirated into the lungs. It is impossible to tell at the scene whether water has been aspirated into the lung, and does not influence management.

Sunburn Depth Classification

Classification	Appearance	Sensation
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Supplement to *Emergency Medicine Reports*, July 26, 2015: "Summer Emergencies: Heat Illness, Lightning Injuries, Drowning, and Sunburn." Authors: Katia Lugo, MD, FACEP, Faculty, Florida Hospital Emergency Medicine Residency Program, Assistant Director of Simulation, Florida Hospital, Orlando, FL; and Jessica Aun, DO, Emergency Medicine Resident, Florida Hospital Emergency Medicine Residency Program, Orlando, FL.