Osteopathic Family Physician (2013) 5, 200–207

ELSEVIER

acofp Osteopathic Family Physician

Exposure injury: Examining heat- and cold-related illnesses and injuries

Bryan D. Grooms, DO, Daryn Straley, DO

From O'Bleness Memorial Hospital, 75 Hospital Dr, Athens, OH.

KEYWORDS:
Exposure injury;
Cold-related illness;
Heat-related illness;
Heat exhaustion;
Heat stroke;
Hypothermia;
Frostbite;
Miliaria;
Pernio;
Chilblains

Abstract Heat- and cold-related illnesses represent a broad spectrum of preventable common conditions affecting diverse populations every year. Clinicians treating patients near areas of outdoor recreation and nursing homes or who potentially see critical pediatric, indigent, or intoxicated patients can benefit from a better understanding of exposure injuries. Heat-related illnesses include the continuum of heat rash, heat cramps, heat exhaustion, and the potentially deadly heat stroke. Cold-related illnesses encompass a broad array of conditions ranging from soft tissue injuries like pernio, trench foot, and frostnip to the more severe effects of frostbite and hypothermia. The majority of patients with severe environmental exposure would usually present to an emergency or urgent care center; however, milder acute cases and more subtle chronic presentations would generally first be seen by a primary care physician. With any temperature-related illness, the family physician's role should most importantly include identification of at-risk populations and promotion of proper preventative strategies allowing early symptom recognition and rapid treatment.

Thermoregulation

Heat exchange between the body and the environment is accomplished in 5 ways—evaporation, conduction, radiation, convection, and respiration. Ambient features such as humidity and temperature, as well as heat index and wind chill, create a contrast with the body temperature, thus greatly influencing to what extent and velocity heat is lost or gained. Additionally, medications may predispose a patient to significant temperature alterations (Table 1). Evaporation is the most effective method by which humans dissipate heat via perspiration. In optimal conditions, the average individual can dissipate 600 kcal/h and produce up to 1-2 L of sweat per hour.[1,2] Conduction relates to the direct transfer of heat from a warmer surface to a cooler surface. The amount of heat exchanged is directly proportional to the temperature gradient and surface area contacted. For example, the larger the surface area submerged during cold-water immersion, the more the heat dissipated. Similarly, radiation is the direct transfer of heat into the environment. It is the second most effective method of dissipating heat; however, it is largely dependent on the ambient temperature being below body temperature. Because children have a greater relative surface area-to-mass ratio, more heat is absorbed via radiation than any other source.[1] This becomes particularly important in preventing heat-related illness, especially in children and adolescents, as light-colored clothing reflects more radiant energy thus absorbing less heat. Convection depends on air flow around the body to produce a heat gradient. This can be generated from movement through the environment as with cycling or running or can be dependent on wind current. Air-conditioning, fans, and loose-fitting clothing contribute to increased heat loss via convection.[3] In a better-protected

Corresponding author: Bryan D. Grooms, O'Bleness Memorial Hospital, 75 Hospital Drive, 9065 Echo Lane, Athens, OH 45701.
E-mail address: bg115206@ohio.edu

1877-573X/$ - see front matter © 2013 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.osfp.2013.04.002

Table 1 Medications that interfere with thermoregulation

Increase heat production
α-Adrenergic agents
Amphetamines
Beta blockers
Calcium channel blockers
Dietary supplements (ie, ephedra and diet pills)
Diuretics
Illicit drugs (ie, cocaine, heroin, LSD, and PCP)
Monoamine oxidase inhibitors
Thyroid Agonists
Tricyclic Antidepressants
Decrease sweating
Anticholinergics
Antihistamines
Benztropine mesylate
Typical antipsychotics (phenothiazines, thioxanthines, and butyrophenones)
Decrease thirst
Haloperidol

LSD = lysergic acid diethylamide; PCP = phencyclidine.
Adapted from Howe and Boden,[2] Becker and Stewart, and [3] Moran and Gaffin.[9]

individual, such as those in a well-planned mountain expedition, the effects of respiratory heat loss initially dominate, in most cases, because of high altitude air that is typically colder, more windy, and dry. By understanding the various methods through which heat is lost or gained, the family physician can work with athletes, coaches, and trainers, as well as specific populations like environmental workers, caregivers, and the elderly, to provide better education on proper strategies, for example appropriate attire, hydration, and weather conditions, to prevent exposure injury.

The physiological regulation of body temperature involves a complex array of mechanisms controlled by the hypothalamus in response to factors such as heat production, absorption, and dissipation. Increases in the core temperature of less than 1°C (33.8°F) can activate the hypothalamic thermoregulatory response between the spinal cord and peripheral centers in the skin and organs to help maintain core body temperature between 36°C (96.8°F) and 37.5°C (99.5°F).[1,3,4] The primary response to the increased core temperature is peripheral vasodilation, which promotes heat loss and reduces environmental heat gain. The increased epidermal blood flow causes a compensatory drop in splanchnic blood flow to maintain blood pressure.[5] When the core body temperature approaches 40°C (104°F), this physiological response creates an ischemic environment that promotes the release of gut and renal endotoxins, thereby leading to systemic inflammatory cytokine release, coagulopathies, and multiorgan system dysfunction. If not promptly treated, this inflammatory cascade can result in death.[6]

Similarly, yet on the opposite end of the temperature spectrum, prolonged cold exposure causes peripheral vasoconstriction and direct tissue-freeze injury. Under ischemic conditions induced by vasoconstriction, ice crystals form intracellularly (in rapid freezing) or extracellularly (in gradual freezing), causing derangement of cellular proteins, lipids, electrolytes, and hydration, which leads to cytolysis and tissue necrosis.[7] If thawing and refreezing occurs, tissue integrity rapidly deteriorates from ischemia, extravasation of fluids, inflammatory mediator cascade spikes, and thrombi showers.[7] As shivering proceeds, with tissue hypoxia and freeze injury, lactic acid production and muscle-breakdown products develop a metabolic acidosis with impending renal failure with associated hyperkalemia. Furthermore, as the core temperature decreases along the continuum of hypothermia, a hypercoagulable state ensues secondary to direct inhibition of enzymes of the clotting cascade and the temperature-dependent production of thromboxane B_2, thus impairing platelet function and leading to thromboembolism.[8] To further complicate the situation, there is a sequestration of platelets in the splanchnic vasculature, liver, and spleen. As the body temperature falls, blood buffering systems also lose effectiveness. Eventually, muscle fatigue and metabolic dysfunction along with glycogen depletion lead to multiorgan decompensation and extreme temperature drop. Without proper treatment, neurologic decline, cardiopulmonary arrest, and mortality soon occur.

Acclimatization to extreme heat or cold can prevent such reactions from occurring, but typically take several weeks. A gradual increase in physical activity over 8-10 days should provide optimal acclimatization. Children need a longer period of 10-14 days to achieve a similar response.[9] Physiological adaptations to higher ambient temperatures include improved renal function—increased sodium retention and glomerular filtration rate—and enhanced cardiovascular performance.[10] Because of these changes, persons can work, live, and play in more extreme environments than what would normally be tolerated.

Heat-related illness

Heat-related illness is a spectrum of common conditions caused by tissue and physiological changes to higher temperatures, which is largely preventable by simple measures. These conditions range from relatively minor ailments like heat rash or heat cramps, to more serious illnesses like heat exhaustion and life-threatening heat stroke. Currently, heat is the leading cause of weather-related deaths in the United States.[11] Because of physiological differences, access to hydration or air-conditioning, or prolonged environmental stress, at-risk populations for heat-related illness include the elderly, children, athletes, military recruits, and environmental workers. Individuals with heart and lung conditions, mental illness, and those who take medications that may blunt cardiovascular compensation are also at risk. Of the deaths that occur because of heat-related illness, the largest percentage occurred amongst environmental workers, particularly

Table 2 Summary of risk factors

Internal factors	External factors
Age (<15 or >65)	Ambient temperature
Alcohol consumption	Excessive clothing wear
Comorbidities (cardiovascular, respiratory, hematologic, and psychiatric conditions)	Humidity
Dehydration	Lack of water or access to cooling
Medications	Occupation
Obesity	Wet bulb globe temperature
Physical disability or impaired mobility	
Poor acclimatization	
Poor exercise tolerance or cardiovascular fitness	
Skin conditions (eczema, psoriasis, sunburn, and burns)	

Adapted from Howe and Boden,[2] Becker and Stewart,[3] Glazer,[4] and Ewald and Baum,[16]

agricultural workers.[12] A survey amongst summer high school athletes demonstrated that football players have a 10-fold increased risk of developing heat-related illness.[13] A summary of risk factors is presented in Table 2. Prompt recognition of symptoms and early treatment are crucial to prevent further progression to heat stroke.[3]

Heat rash

Miliaria rubra, also known as heat rash or prickly heat, is an erythematous, pinpoint papulovesicular rash commonly found in areas covered by clothing, such as the waist, trunk, or groin (Figure 1).[14] It usually occurs after an individual is exposed to high heat and humidity causing profuse sweating, which results in eccrine sweat leakage into the dermis or epidermis secondary to obstruction of sweat glands. It is intensely pruritic in nature, and is often described as having a stinging or "prickling" quality to the rash. Rapid onset and history of heat exposure and excessive sweating help distinguish heat rash from other causes such as viral exanthema, urticaria, or contact dermatitis.[2] Treatment is mostly conservative, involving cooling the affected areas, wearing loose-fitting clothing where possible, and avoiding oil-based topical lubricants.[1,2] The rash is generally benign and can take up to a week to resolve; however, secondary staphylococcal infections can occur and delay resolution. Preventative strategies include limited heat exposure, and wearing loose-fitting, light-colored clothing preferably with an open weave design to allow sweat to dissipate.[2]

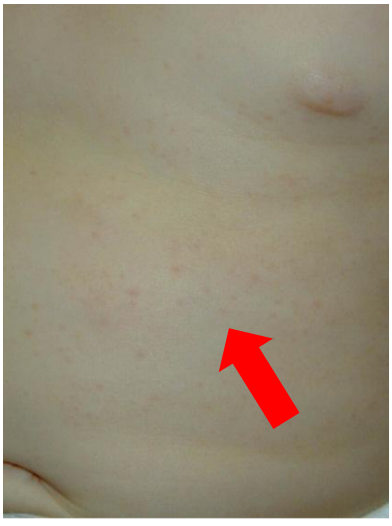

Figure 1 Miliaria rubra.

Heat cramps

Heat cramps or exercise-associated muscle cramps are painful, sporadic abrupt contractions commonly involving the quadriceps, hamstrings, gastrocnemius, and abdominal musculature during rest after vigorous exercise.[1-3] These cramps are believed to be caused by sodium loss as the result of excessive heat exposure, profuse sweating, and water intake without adequate electrolyte replacement.[2] Athletes engaging in strenuous exercise for more than 2 hours, steel mill workers, and military personnel deployed to hot, humid environments have a high incidence of heat cramps.[15] The development of heat cramps does not predispose an individual to further heat illness; however, it is an early symptom.[2] Treatment generally consists of rest and electrolyte replacement. For most cases, oral electrolyte solutions are effective, although in situations where conservative measures fail, intravenous normal saline at 5-10 mL/kg over 20 minutes is recommended.[16] Other treatment measures include cooling, massaging, and stretching the affected muscles.[17] Proper hydration, including electrolyte replacement, before and during periods of activity, along with proper loose-fitting attire is key to preventing heat cramps.[2]

Heat exhaustion

Heat exhaustion continues along the same physiological basis for heat-related illness and is generally divided into 2 etiologies—water depletion or sodium depletion. Water depletion leads to a more rapid onset and occurs secondary to inadequate fluid intake combined with increased fluid losses. Salt depletion has a more gradual course and results from free-water replacement without electrolyte intake.[1] Either cause generally produces a myriad of nonspecific symptoms, such as heat cramps, nausea or vomiting, headache, malaise, and myalgias. Persons can also show signs of circulatory collapse, like hypotension and lightheadedness.[18] Typically, core temperature, accurately measured with a rectal thermometer, is normal or shows slight

hyperthermia—less than 40°C (104°F).[1] Initial management includes removing excess clothing, moving the victim to a cooler environment and placing them in a supine position with the legs elevated. Oral hydration is preferred over intravenous if the patient can tolerate it. Continuous monitoring of vital signs should be performed with prompt transport to emergency facilities if there is no improvement after 20-30 minutes. Heat exhaustion represents a moderate compromise to afflicted individuals, however, without timely treatment; their condition can deteriorate into life-threatening heat stroke.[3]

Heat stroke

Heat stroke represents the most severe and life-threatening condition in the continuum of heat-related illness and is a true medical emergency. Most episodes occur during summer heat waves with several days of high ambient temperatures and humidity. The elderly have the highest risk for mortality, but outdoor laborers, children, and athletes are also at-risk populations. Diagnosis of heat stroke relies on elevated core temperature of 40°C (104°F) or greater and profound central nervous system symptoms, which may include seizures, delirium, hallucinations, irritability, ataxia, loss of sweating, and hypotension, along with symptoms of heat exhaustion.[1,3,4] As the body tries to maximize heat dissipation, tachycardia ensues. Hypotension is a late finding in the condition and represents impending circulatory collapse.[1] After recognizing the symptoms of heat stroke, treatment should be initiated immediately at the scene with focus on addressing airway, breathing, and circulation, as well as activation of the emergency response system. Once these are addressed, on-site rapid cooling before transfer to an emergency facility should be initiated. Cold-water immersion is the treatment of choice and has been shown to greatly reduce morbidity and mortality, however, if this is not possible, applying ice packs or cold wet towels to the head, neck, axilla, and groin is a viable option.[3] Evaporative cooling with water mist and fanning is another alternative and is likely to be better tolerated for small children.[1] Regardless of method, treatment goals are aimed to achieve a core body temperature of less than 38.9°C (102°F). Controversy exists about the best treatment approach for heat stroke; however with morbidity and mortality being directly related to the duration and intensity of elevated core temperature, cooling modality should be based on first availability.[9]

Cold-related illnesses

Cold-related illnesses encompass hypothermia and soft tissue injuries ranging from various forms of nonfreezing cold injuries (NFCI), such as pernio and trench foot, to frostnip and frostbite. Although cold-related illnesses and injuries are a major problem for recreationalists in the wilderness, they are also frequently encountered in the setting of family medicine—particularly in the pediatric, geriatric, and homeless populations. Beyond the extremes of age, higher risk is associated with chronic comorbid conditions and intoxication. During the years of 1999-2003, an average of 1536 persons per year in the United States died of hypothermia-related diagnoses[19]—600 of whom attributed to primary hypothermia—approximately half of whom were elderly, over 65 years of age.[20] Multitudes more suffered permanent sequelae such as limb loss and end-organ damage.

Hypothermia

Hypothermia is defined as a core body temperature less than 35°C (95°F). Once this diagnosis is determined, continuous monitoring should be performed to assess for core temperature and myocardial electrical activity as an ongoing evaluation for oxygenation, metabolic derangement, hematocrit changes, renal function, and neurologic decline. More indolent, subtle hypothermia may present in the elderly or young pediatric patients whereas more severe presentations are more commonly associated with outdoor enthusiasts, homeless population, immersion accidents, and in more remotely populated regions. Additional medical or behavioral comorbidities further worsen the adaptation mechanisms to cold and thus the prognosis. Patients presenting from seemingly acclimatized environments should raise the suspicion of intoxication, medication effects (especially sedative-hypnotics and phenothiazines), neurologic insult, severe infection, and endocrine failure involving the adrenals, thyroid, or central pituitary axis. Early hypothermia is typically associated with hypocapnia and alkalosis, shifting the oxygen dissociation curve to the left, thereby leading to impaired tissue oxygen absorption from hemoglobin. This process is aggravated by colder, more viscous blood that also gradually increases in hematocrit by 2% per 1°C (33.8°F) drop in core body temperature[8] and reactive peripheral vasoconstriction designed to reduce further heat loss from the body surface.

The clinical continuum of depressed core temperature provides a useful assessment and prognostic tool for anticipated pathologic events (Table 3). Clinical manifestations can range from mild variations in vital signs and shivering to severe central nervous system suppression, cardiac arrest, and death.

In addition to maintaining normal core temperature and hydration, the presence of peripheral tissue-freeze injuries (pernio or chilblains, frostnip, trench foot, and frostbite) mandates additional measures to improve distal perfusion while preventing further heat loss. In any environment—typically in the field—where tissue refreeze after thawing is likely, warming techniques are contraindicated because of the resulting severe, subsequent injury augmentation. In addition, the risks of ambulation on a frozen extremity with expansion of tissue damage needs to be weighed against the risks of attempting alternative evacuation options.

Table 3 Stages of hypothermia and clinical features

Mild, 32.2°C-35°C (90°F-95°F)
Initial cold exposure agitation
Hypertension
Shivering
Tachycardia
Tachypnea
Vasoconstriction
Subsequent fatigue onset
Apathy
Ataxia
Renal dysfunction—cold diuresis
Hypovolemia
Impaired judgment
Moderate, 28°C-32.1°C (82.4°F-89.9°F)
Atrial dysrhythmias
Bradycardia
Declining consciousness
Bradypnea
Pupillary dilation
Diminished GAG reflex
Cessation of shivering
Hyporeflexia
Hypotension
J Waves
Severe, <28°C (82.4°F)
Apnea
Coma
Electroencephalographic
Senescence
Pupillary areflexia
Oliguria
Pulmonary edema
Ventricular dysrhythmia or asystole

Adapted from Hypothermia-Related Deaths—United States, 1999-2002 and 2005.[19]

If the core temperature is below 32°C (89.6°F), in addition to hospitalization and passive rewarming, active core rewarming is necessary. Although mild hypothermia and frostbite could be simultaneously treated, moderate and severe hypothermia should be treated before frostbite.[7] This may involve the use of external hot water bottles, heating pads or blankets, forced warm air, humidified oxygen at 40°C (104°F), or limb portion immersion in water at 45°C (113°F). Concurrently, an algorithm of gradual escalation of internal warming techniques can be employed. This should begin with warmed (40°C-45°C) intravenous fluids—ideally with 5% dextrose and normal saline to restore depleted glycogen stores—and carefully escalated. Advancement of active rewarming should be performed in a hospital setting with sophisticated monitoring capabilities and could involve warm-fluid gastric, bladder, colonic, and even intrathoracic or peritoneal lavage. Medical management should carefully consider the risks and benefits of each intervention and carefully monitor for cardiac dysrhythmia, rewarming acidosis, and core temperature afterdrop—a paradoxical core temperature drop early in rewarming as cold peripheral blood reenters the systemic circulation. With the available services in extenuating circumstances, the most effective approach is extracorporeal blood rewarming via cardiopulmonary bypass, arteriovenous rewarming, venovenous rewarming, or hemodialysis.[21] The potential for core temperature increase is 1.0°C-2.0°C (33.8°F-35.6°F) every 3-5 minutes.[22] Although arterial blood gas, coagulation, and electrolyte evaluations may be remarkably abnormal, years of research have shown that correction of these abnormalities is generally detrimental because of their self-correction tendency during rewarming.

NFCI

Commonly mistaken for frostbite, it is important to first consider NFCI, such as pernio (chilblains) and trench foot (immersion foot), to provide the best treatment and prevention techniques. Although sometimes heralding a connective tissue disease, pernio is more typically an idiopathic, localized, bluish red lesion pattern of recurring, acral ulcerations that are painful and itchy after cold exposure, which respond well to rewarming (Figure 2).[23] Repeated, seasonal exposures typically lead to increasing severity of subcutaneous arterial vasoconstriction and pain. Precipitating cold cryoglobulinemia may present similarly and respond equally well to warming and cold avoidance. Becoming infamous during World War I and again in World War II, immersion foot injuries combine the effects of vasoconstrictive perfusion loss in the setting of opportunistic infection (usually fungal) and tissue decay after prolonged wet and cold conditions. Pain, fear, physically constrictive footwear, and immobility maintain persistent vasoconstriction during a heightened sympathetic nervous system response with subsequent peripheral nerve ischemia.[24] Free radical formation leads to further endothelial damage, interstitial edema, and tissue hypoxia thereby leading to necrosis. After the initial blanching appearance, rewarming can involve significant hyperemia, pain, blistering, hemorrhage, edema, and necrosis.

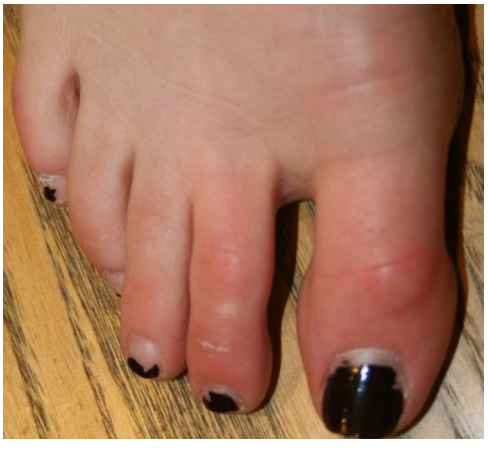

Figure 2 Pernio.

Pernio and cryoglobulinemia are best treated by prevention of cold exposure and tissue rewarming, and intensively with calcium channel blocker vasodilation to facilitate healing. In the event of significant, prolonged freezing with or without thawing in an ambient temperature ranging from −15° to −6°C, (5°F-21.2°F) cellular damage and microvascular insults collectively would likely lead to partial (superficial) or full-thickness (deep) soft tissue frostbite and necrosis.[25] Upon clinical stability of core hypothermia management, concurrent NFCI or frostbite therapy may be initiated. Paradoxically, immersion foot should begin with core warming while actually keeping the affected extremity cool, thus lowering metabolic demands, until hyperemia subsides.[24] As with all soft tissue cold injuries, rubbing should always be avoided.

Frostnip and frostbite

Most patients presenting with soft tissue injury complain of localized numbness following prolonged cold exposure; however, the exact extent of permanent tissue damage cannot be clearly predicted until a reevaluation of tissue changes occurring during and after rewarming. Although more specific definitions exist for tissue cold-injury classification, for the primary care physician it is sufficient to categorize them as superficial or deep-freeze injury. Superficial frostbite may be preceded by frostnip, distinctly a nonfreezing injury involving intense vasoconstriction. By definition, ice crystal formation is limited to the surface and results in moderate numbness, possible paresthesia, and pallor without permanent tissue damage.

As frostnip proceeds to superficial frostbite, there is an intensification of numbness, erythema, white or yellow plaques with mild tissue sloughing, and edema—sometimes advancing to clear or milky fluid vesiculation (Figure 3).[26] Tissue loss is expected to be superficial and minimal. Deeper injuries extending into or through the dermis, into the subcutaneous tissue, or into muscle and bone result in hemorrhagic blisters and full-thickness tissue necrosis, which is expected to result in considerable tissue loss.[7]

Although initial management of frostbite and inflammatory tissue damage varies significantly, whether beginning in the field or in hospital, Table 4 describes an adaptation of the recommended first-line approaches that apply best to a primary care setting transitioning to the hospital. Although there is mixed evidence regarding the routine use of antibiotics in otherwise clean, necrotic tissue, the incidence of opportunistic infection in trench foot or other contaminated areas is quite high. In the examination of the recommendations listed in Table 4, evidence guidelines from the American College of Chest Physicians regarding management are given grade recommendation strength based upon the quality of clinical evidence. Grade 1 is given strong recommendation with Category A having a high level of evidence, Category B with moderate evidence, and Category C with low evidence.[27,28]

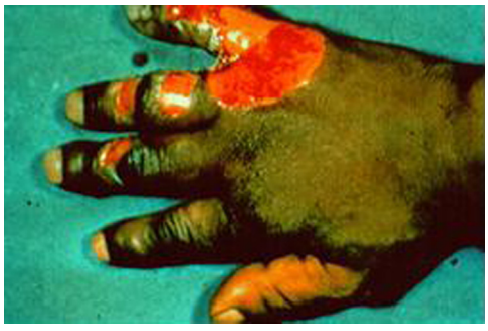

Figure 3 Frostbitten hand.

Table 4 Summary of prehospital and initial hospital frostbite management

1	Basic life support (BLS) and advanced cardiac life support (ACLS/PALS) as appropriate
2	Remove jewelry and extraneous materials from affected body part
3	Rapid, active rewarming of core and involved limb (s) per above
4	Ibuprofen (12 mg/kg/d divided twice daily) (evidence 2C)
5	Analgesia medication and tetanus prophylaxis (evidence 1C)
6	Air dry
7	Protect from refreezing and trauma; avoid unnecessary ambulation
8	Debridement
9	Topical *Aloe vera* (evidence 2C) and dry, bulky dressing change every 6 h
10	Elevate affected body part
11	Systemic hydration (evidence 1C)
12	Thrombolytic therapy consideration for deep frostbite if <24 h after thawing; utilize angiography for prethrombolytic intervention and progress monitoring (evidence 1C)
13	Clinical interval examination (plus angiography or technietium-99 bone scan or both if necessary) to determine surgical margins as appropriate
14	Surgical evaluation as appropriate for suspected further debridement or amputation or both

PALS = pediatric advanced life support. Adapted from McIntosh et al.[7]

In localized hypercoagulation, such as in the distal extremities, however, many studies have revealed limb-saving benefits of intra-arterial-directed tissue plasminogen activator (tPA) when provided within 24 hours of thawing following initial hospital management techniques.[7] During therapeutic augmentation, the risks and benefits of each escalation should be measured for the specific clinical scenario. In severe soft tissue-freeze injuries, such aggressive interventions have shown considerable preservation of viable tissue.

Never to be used in the field, the use of these thrombolytics should be carefully analyzed for benefits vs risks, such as catheter-site bleeding, compartment syndrome, and failure of tissue salvage, thereby necessitating intensive monitoring capabilities.[7] Owing to the altered

pharmacodynamics, liver metabolism, and protein binding of core hypothermia, the potential toxic drug effects upon warming generally deems pharmacotherapy of any kind to be overly hazardous until a more normal physiological temperature range is reached. Early successes of limb salvage using combined thrombolytics and vasodilators, however, have led to ongoing evaluation for efficacy of aspirin plus buflomedil, iloprost, and intravenous tPA plus iloprost.[7] Currently, there are insufficient data to support heparin-derived and vasodilation therapies unless used as adjuncts to tPA. Although antibiotics are not routinely recommended, it is important to ensure tetanus prophylaxis and is beneficial to administer topical *Aloe vera* to thawed tissues along with oral ibuprofen (12 mg/kg divided twice daily) until the wound heals or is surgically debrided.[7]

Osteopathic manipulative treatment (OMT) considerations

Studies evaluating the efficacy of OMT for exposure injuries are lacking in the literature; however, from what is known about the pathophysiology of heat-related illness, it is intuitive that OMT would be a beneficial adjunct to current therapies. The catecholamine release leading to cardiovascular compensation and sweat gland activation is driven through the sympathetic ganglion. Rib or thoracic dysfunction and fascial restrictions along the paravertebral ganglion could alter this sympathetic drive and thus diminish proper physiological response to core temperature elevation. Functional methods, rib raising, or myofascial release could be used to restore balance along the sympathetic chain and promote balanced response. Impaired lymphatic flow can cause congestion of inflammatory cytokines and heat stress proteins potentially exacerbating the condition. Release of the lymphatic diaphragms can facilitate restoration of flow and removal of harmful toxins. Further study is needed to determine the efficacy of OMT techniques and should not delay standard treatments.

Although no official recommendation has been developed, owing to the risks of further injury to frozen tissues or mobilization of freeze-induced emboli, manipulative therapy should be reserved for postnormalization of core hypothermia and milder soft tissue injury such as frostnip. Although there are intuitive benefits to lymphatic and circulatory mobilization, these must be carefully weighed against the potential for further tissue damage and the release of elevated levels of the products of catabolism and thrombogenesis. During later recovery stages, a more comprehensive evaluation can better reveal the safety of the integration of tissue and lymphatic mobilization therapeutics.

Discussion

Prevention of temperature-related illnesses consists of identifying at-risk populations and altering behavior and physical activity depending on environmental conditions, appropriate hydration, and access to cooling methods. Family physicians should assert preventative strategies, including community-sponsored cooling shelters, as part of their anticipatory guidance to the indigent, elderly, and pediatric populations as well as their caregivers.[9] Physicians working with sports programs should educate players, coaches, and trainers on signs and symptoms of exposure injury, proper hydration—before, during, and after activity—and encourage the use of lightweight, reflective clothing during periods of high ambient temperatures.[3] Rapid access to air-conditioning has been shown to be the most effective method at reducing heat-related illness. Although many communities distribute fans among at-risk populations, these are inadequate during times of extreme heat and humidity.[4] Beginning with discussions about the prevention of cold-induced injuries, the primary care physician is well poised to assist in reducing the potentially permanent or lethal sequelae of hypothermia. Specific at-risk populations, as identified previously, need to be educated on the proper attire to prevent heat loss during cooler weather. This includes personal protective strategies such as layering attire preferably with moisture-wicking undergarments, and the use of gloves, socks, hats, and scarves. Exercise during winter months should be encouraged, as inactivity has shown to reduce cold tolerance. More advanced preventative strategies include properly insulating and weatherizing homes.[29] Although most cold injuries presenting to the primary care setting are relatively mild, failure to identify and correctly treat them can lead to significant long-term morbidity and mortality. Additionally, most clinical settings are not completely isolated from potentially more severe environmental exposure pathology. For the practitioner working within urgent or emergent care delivery systems or personally involved in an outdoor activity, a thorough understanding of the presentation and management of exposure injuries is essential to effectively reduce disability and death. Although no one is immune to extreme heat- or cold-related illness, by implementing these strategies, the number of cases can greatly be reduced.

References

1. Grubenoff JA, Ford K, Roosevelt GE. Heat-related illness. *Clin Pediatr Emerg Med.* 2007;8(1):59–64
2. Howe AS, Boden BP. Heat-related illness in athletes. *Am J Sports Med.* 2007;35(8):1384–1395
3. Becker JA, Stewart LK. Heat-related illness. *Am Fam Physician.* 2011;83(11):1325–1330
4. Glazer JL. Management of heatstroke and heat exhaustion. *Am Fam Physician.* 2005;71(11):2133–2140
5. Leon LR, Helwig BG. Heat stroke: role of the systemic inflammatory response. *J Appl Physiol.* 2010;109:1980–1988
6. Lim CL, Mackinnon LT. The roles of exercise-induced immune system disturbances in the pathology of heat stroke. *Sport Med.* 2006;36(1):39–64
7. McIntosh SE, Hamonko M, Freer L, et al: Wilderness medical society practice guidelines for the prevention and treatment of frostbite. *Wilderness Environ Med.* 2011;22:156–166

8. Danzl DF, Pozo RS. Accidental hypothermia. *N Engl J Med.* 1994;331:1756–1760
9. Moran DS, Gaffin SL. Clinical management of heat-related illnesses. In: Auerbach PS, ed. *Wilderness Medicine*, 5th ed, St Louis, MO: Mosby; 2007. [Chapter 2]
10. Bouchama A, Knochel JP. Heat stroke. *N Engl J Med.* 2002;346(25):1978–1988
11. Davis RE, Knappenberger PC, Michaels PJ, Novicoff WM. Changing heat-related mortality in the United States. *Environ Health Perspect.* 2003;111(14):1712–1718
12. Centers for Disease Control and Prevention (CDC). Heat-related deaths among crop workers—United States, 1992-2006. *MMWR Morb Mortal Wkly Rep.* 2008;57(24):649–653
13. Centers for Disease Control and Prevention (CDC). Heat illness among high school athletes – United States, 2005-2009. *MMWR Morb Mortal Wkly Rep.* 2010;59(32):1009–1013
14. Bill Abbottsmith (Physician). (2002). Miliaria rubra [Photograph], Retrieved on September 7, 2012, from Dermatlas; ⟨http://www.dermatlas.org⟩.
15. Ganio MS, Casa DJ, Armstrong LE, Maresh CM. Evidence-based approach to lingering hydration questions. *Clin Sports Med.* 2007;26:1–16
16. Ewald MB, Baum CR. Environmental emergencies. In: Fleisher G, Ludwig S, eds. *Textbook of Pediatric Emergency Medicine*, 5th ed, Philadelphia, PA: Lippincott Williams & Wilkins; 2006:1017–1021
17. Seto CK. Environmental illness in athletes. *Clin Sports Med.* 2005;24:695–718
18. Wexler RK. Evaluation and treatment of heat-related illnesses. *Am Fam Physician.* 2002;65(11):2307–2313
19. Centers for Disease Control and Prevention (CDC). Hypothermia-Related Deaths—United States, 1999–2002 and 2005. *MMWR Morb Mortal Wkly Rep.* 2006;55(10):282–284
20. Danzl DF. Accidental Hypothermia. In: Auerbach, et al., eds. *Wilderness Medicine*, 5th ed, Philadelphia: Mosby Elsevier; 2007:125–160
21. Mccullough L, Arora S. Diagnosis and treatment of hypothermia. *Am Fam Physician.* 2004;70(12):2325–2332
22. Splittgerber FH, Talbert JG, Sweezer WP, Wilson R. Partial cardiopulmonary bypass for core rewarming in profound accidental hypothermia. *Am Surg.* 1986;52:407–412
23. Cohen, Bernard (Physician). 2011. Pernio. [Photograph], Retrieved on September 7, 2012, from Dermatlas; ⟨http://www.dermatlas.org⟩.
24. Lipman GS, Castellani JW. Nonfreezing Cold-Induced Injuries. In: Auerbach, ed. *Wilderness Medicine*, 5th ed, Philadelphia: Mosby Elsevier; 2007:188–195
25. McCauley RL, Killyon GW, Smith Jr DJ, Robson MC, Heggers JP. Frostbite. In: Auerbach, et al., eds. *Wilderness Medicine*, 5th ed, Philadelphia: Mosby Elsevier; 2007:195–210
26. Workplace and Safety Health Topics: Frostbite. Centers for Disease Control. Available at ⟨http://www.cdc.gov/niosh/topics/coldstress/⟩. Accessed September 3, 2012.
27. ECC Committee, Subcommittees and Task Forces of the American Heart Association. 2005 American Heart Association Guidelines for Cardiopulmnary Resuscitation and Emergency Cardiovascular Care, Part 10.4: Hypothermia. *Circulation.* 2005;112:IV136–IV138
28. Guyatt G, Guttermann D, Baumann MH, et al: Grading strength of recommendation and quality of evidence in clinical guidelines: report from an American College of Chest Physicians Task Force. *Chest.* 2006;129(1):174–181
29. Conlon K, Rajkovich N, White-Newsome J, Larsen L, O'Neill M. Preventing cold-related morbidity and mortality in a changing climate. *Maturitas.* 2011;69(3):197–202